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# Finite Element Modeling and simulation of arteries in the human arm to study the aortic pulse wave propagation

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**Abstract**

Finite modelling and simulation of the arterial network in the human arm has been presented in this paper with an objective to study the aortic pulse wave propagation. In the biomedical domain, it becomes extremely essential to understand the propagation of the aortic pulse along the arterial network, to get a better insight about the functioning of the cardiovascular system. This would assist in haemodynamic measurements, diagnosing disorders and visualizing the effect of medical treatment. The fluid structure interaction has been simulated using COMSOL Multiphysics 4.4 with an objective to obtain the pressure, velocity profile of the aortic pulse and wall shear stresses at the ascending aorta, carotid, brachial, interosseous, ulnar and radial artery. The arterial walls are considered flexible and pulsatile pressure pulse has been used as boundary condition. The validity of the finite element simulation has been supported by comparing the numerical results to the standard published results.



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*Keywords:* Finite Element Method; blood flow rate; velocity; pressure; human arm; wave propagation

## 1. Introduction

The bio-medical domain is a very challenging domain to understand since it deals with the factors that cause various ailments in the human body. The mechanics of blood flow have a considerable impact on health of individuals. The condition of the arterial wall may cause disturbances in the blood flow leading to clinical complications. Even though blood flow is usually laminar, periodically unsteady nature of flow may lead to

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turbulence in the narrower arteries at higher velocities. The concept of fluid dynamics can be readily applied in understanding of environment, influence of wall modifications, local pressures and velocities on flow patterns thereby providing better guidelines in diagnosis and finding faster and improved remedial measures. Computational analysis is very rare and has picked up pace only in the last decade. Besides, the pulsatile nature of blood flow which represents an actual cardiac waveform has been scarcely studied. In most cases, the artery is modelled as a simple cylindrical tube resulting in approximate solutions. A mathematical model for studying the interaction of blood flow with the arterial walls surrounded by cerebral spinal fluid was developed by Venuti [1]. Sankar and Lee [2] studied the effects of pulsatility, stenosis and non-Newtonian behaviour of blood, treating the blood as Herschel–Bulkley fluid. Siddiqui et al.[3], treated the blood as Casson's fluid and investigated the effects of non-Newtonian nature of pulsatile blood flow through a stenosed artery. In 2011, Razavi et al. [4], performed the simulation using Newtonian as well as non-Newtonian viscosity models. This work has been carried out for stenosed carotid artery with a pulsatile nature of blood flow. Nadeem et al. [5] in 2011, analyzed blood flow through a tapered artery with a stenosis. In 2013, Tian et al. [6] used a simplified model to simulate a pulsatile non-Newtonian blood flow past a stenosed 2D artery caused by atherosclerotic plaques of different severity. In 2012, Mortazavinia et al. [7] simulated the pulsatile blood flow to study renal artery stenosis and explored the its effect on the blood flow and vessel walls. The attempt was made to develop a realistic model of arteries from CT- scan images. Rabby et al. [8], in 2013 used finite volume method to perform numerical analysis of a pulsatile flow through a two-dimensional (2D) axisymmetric pipe with an idealized stenosis. Fojas and De Leon [9], in their paper presented in 2013, have demonstrated the use of computational software for hemodynamic analysis of two-dimensional model of the carotid artery and its bifurcation.

This paper aims at modelling the arterial network in the entire human arm and investigates variations in the aortic pulse wave parameters as it propagates from the ascending aorta towards the radial artery. In the biomedical domain, it becomes extremely essential to monitor these changes as they are indicators of proper functioning of the cardiovascular system. Disturbed blood flow and wall shear stress may lead to Atherosclerosis (Vascular Disorders). The numerical simulation of blood flow within the arteries not only helps in monitoring the significant parameters but also helps in obtaining the variations in these parameters at the aorta and the other nodes of the arterial network. Certain hemodynamic parameters such as cardiac output, stroke volume are measured either using invasive techniques or with techniques which are costlier and require a large setup. The results of this simulations can also be effectively utilized in devising mathematical relations between the aortic and radial parameters, thereby making bedside measurements of these parameters simpler.

## 2. Mathematical Model

The essential parameters which characterize the blood flow are velocity u and pressure p. These field parameters help in computing the stresses developed in the arterial wall subjected to the blood movement. This basically aids in monitoring the condition of the arterial wall. The blood flow being pulsatile in nature causes radial deformations in the arterial walls. Thus, in addition to the flow parameters, structural deformations also need to be considered, thereby leading to a fluid structure interaction problem. Arterial blood flow is a three dimensional flow, where the blood is considered to be incompressible Newtonian fluid. The Navier –Stokes equations thus can be applied [10]. Eq. 1 expresses the conservation of mass:

$$\frac{\partial u}{\partial x}+\frac{\partial v}{\partial y}+\frac{\partial w}{\partial z}=0 \tag{1}$$

where u, v and w denote the velocities in x, y and z directions respectively. Eq. 2 expresses the conservation of momentum in x, y and z directions.

$$\left.\begin{aligned}
\frac{\partial u}{\partial t}+u\frac{\partial u}{\partial x}+v\frac{\partial u}{\partial y}+w\frac{\partial u}{\partial z}&=-\frac{\partial p}{\partial x}+\boldsymbol{\nu}\left(\frac{\partial^2 u}{\partial x^2}+\frac{\partial^2 u}{\partial y^2}+\frac{\partial^2 u}{\partial z^2}\right)+F_x\\
\frac{\partial v}{\partial t}+u\frac{\partial v}{\partial x}+v\frac{\partial v}{\partial y}+w\frac{\partial v}{\partial z}&=-\frac{\partial p}{\partial y}+\boldsymbol{\nu}\left(\frac{\partial^2 v}{\partial x^2}+\frac{\partial^2 v}{\partial y^2}+\frac{\partial^2 v}{\partial z^2}\right)+F_y\\
\frac{\partial w}{\partial t}+u\frac{\partial w}{\partial x}+v\frac{\partial w}{\partial y}+w\frac{\partial w}{\partial z}&=-\frac{\partial p}{\partial z}+\boldsymbol{\nu}\left(\frac{\partial^2 w}{\partial x^2}+\frac{\partial^2 w}{\partial y^2}+\frac{\partial^2 w}{\partial z^2}\right)+F_z
\end{aligned}\right\} \quad (2)$$

Where t denotes time, p the pressure scaled by density, ν the kinematic viscosity and, the external body force, which is omitted in the simulation. No-slip boundary condition has been assumed over the aortic walls, as blood velocity adjacent to the wall in larger arteries changes with same velocity as the wall [11]. With $\mathbf{u}=(u,v,w)^T$ , Eq.1 and Eq.2 in condensed form can also be denoted as Eq. 3 and Eq. 4:

$$\nabla.u=0 \quad (3)$$

$$\frac{\partial u}{\partial t}+(u.\nabla)u=-\frac{1}{\boldsymbol{\rho}}\nabla p+\frac{\boldsymbol{\mu}}{\boldsymbol{\rho}}\boldsymbol{\nu}\,\nabla^2 u+g \quad (4)$$

Where ρ is the density. These equations need boundary conditions. The blood cannot flow through the wall and due to its viscosity it sticks to the wall. Thus at the wall:

$$u=0; v=0 \; and \; w=0$$

The pulsatile flow of the blood is simulated at the inlet of the artery by pushing it in. The blood volume pushed in can be controlled by the area of the inflow cross section and the blood velocity at the inlet. Therefore inflow condition becomes :

$$u(x,y,z,t)=u_{in}(t)$$

At the outlet, the fluid is assumed to be unidirectional and the pressure to be equal to pressure outside the fluid.

$$\frac{\partial u}{\partial n}=0 \; and \; p=p_0$$

where $n=(n_x,n_y,n_z)$ is the outward pointing normal to the outflow boundary.

The simulation approach involving Ansys FLUENT, ICEM CFD etc., though validated successfully with numerical results is still inconclusive as to how the actual arterial anatomy with its characteristic material properties will influence the blood flow dynamics. With a view to explore this scope, an attempt is made to utilize the advanced simulation software – COMSOL Multiphysics 4.4. The following section shows the detailed simulation steps using the software.

### *2.1. Geometrical Modelling*

The simulation process is carried out by utilizing two physics modules – the Fluid Flow>Single-Phase Flow>Laminar Flow(spf) module, and; the Structural Mechanics>Solid Mechanics (solid) module. The 3D geometry for the artery was modelled using CAD software with the dimensions presented in the Table 1, sourced from literature [12]. Fig. 1 shows the arterial structure of the right human arm that needs to be modelled.

Table 1. Dimensions of arteries for simulation

| Name of segment | Length (cm) | Radius (cm) | Thickness (cm) |
|---|---|---|---|
| Ascending aorta | 4.0 | 1.470 | 0.163 |
| R. carotid | 17.7 | 0.473 | 0.063 |
| Brachial | 42.2 | 0.515 | 0.067 |
| R. radial | 23.5 | 0.367 | 0.043 |
| R. interosseous | 7.9 | 0.194 | 0.028 |
| R. ulnar II | 17.1 | 0.433 | 0.046 |

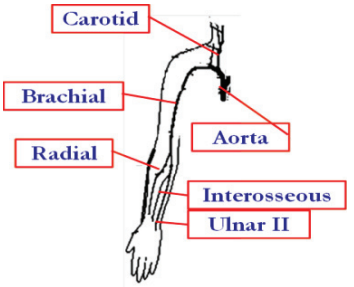


Fig. 1. Arterial geometry of the right human arm

*2.2. Pre-processing*

The pre-processing starts with globally defining parameters for the time continuation parameter (t) as 0(s) and relative velocity amplitude (alpha) as 1/5. A piecewise function f(t) is defined as in Eq. 5.

$$f(t)=\begin{cases}(1-\alpha) \quad \sin\ (2.857\ \pi\ t) & 0\le t\le 0.35\\ (1-2\alpha)\ \sin\ \left(2.22\ \pi\ (t-0.325)\right) & 0.325\le t\le 0.8\end{cases} \tag{5}$$

The domains such as Blood and Artery walls are explicitly defined as well as boundaries such as Inlet, Outlet, Roller boundaries (boundaries not in contact with blood) and Loaded boundaries (interface between blood and artery inner wall surface). The density for blood was taken as 1050 kg/cu.m and dynamic viscosity of 0.005 Pa.s. Fig. 2. shows the geometrical model of the arteries used for the simulation.

The material properties made available through literature [12] were selected as shown in Table 2:

Table 2. Material Properties for the arterial wall

| Material Property | Artery wall |
|---|---|
| Young's modulus (kPa) | 300 |
| Density (cu.m) | 1000 |
| Poisson's ratio | 0.27 |

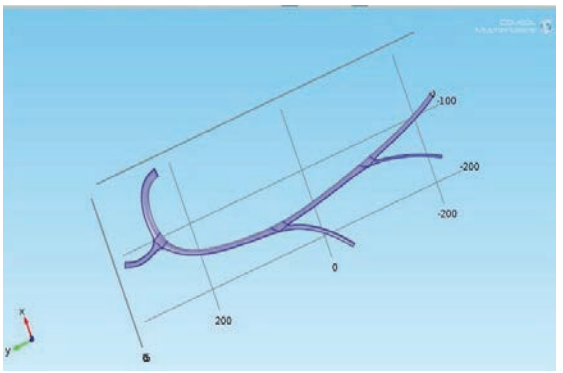


Fig. 2. Geometrical model of arterial network in the human arm

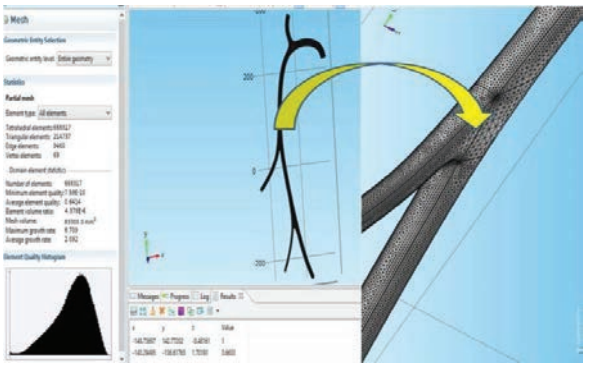

Fig. 3. Mesh View of the geometry

The inlet velocity for the artery simulation was set as 0.3[m/s] * f(t). The outlet pressure was set to 125[mm Hg] * f(t). The geometry was meshed in Free Tetrahedral mode giving a very fine mesh as shown in Fig. 3. The outer

wall, wall thickness and blood within the artery have been modelled using the 668317 tetrahedral elements, while the lumen has been modelled using 214737 triangular elements. The default element type in COMSOL for most physics is tetrahedral element, as any three dimensional shape can be effectively meshed with these elements. Mesh refinement can also be easily done. The Solid mechanics(solid) domain was computed as a time dependent study whereas the study was stationary for the Laminar flow domain with the time range of (0,0.05,0.8)s.

### *2.3. Post-processing*

In the post-processing, various parameters such as distributions of velocity, pressure and wall shear stress are obtained in the form of slices, contours, iso-surfaces, etc. and these parameters are obtained at equal intervals of 0.05 seconds starting from 0 s and going the entire range till 0.8 s. The velocity and pressure are plotted at peak amplitude of the pulsatile flow which occurs at 0.175 sec and are depicted in Fig. 4 and Fig. 5 respectively.

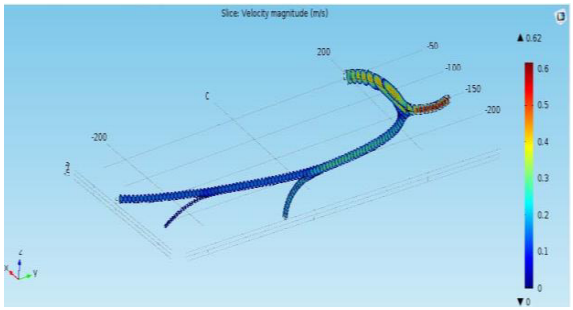


Fig. 4. Velocity plot for arterial geometry at t=0.175 sec

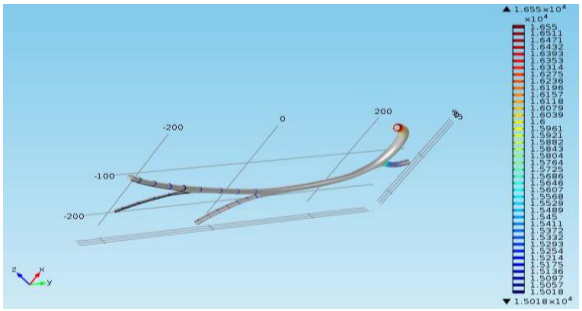


Fig. 5. Pressure plot for arterial geometry at t=0.175 sec

Fig 6 shows the maximum and minimum wall shear stress developed in different sections of the arteries during the cardiac cycle. Velocity at different locations of the arterial network is shown in Fig. 7. Maximum velocity of 0.6 m/sec is obtained in the carotid artery during the systole. Velocity of the aortic pulse reduces to 0.19 m/sec in the ulnar II artery due to the pulsatile nature of the blood flow. Flow rate helps in determining the maximum volume of blood in different sections during the cardiac cycle. The maximum blood volume pumped out of the ascending aorta (at peak systole) per beat is called the stroke volume. This is the most important parameter for consideration in the hemodynamic assessment of a subject. The variation of this blood volume along the arterial network up to the wrist has been depicted in Fig. 8. Due to the pressure exerted on the walls of the deformable artery, there is change in the radii of the various sections of the artery model. Fig. 9 shows the radial deformation in the artery.

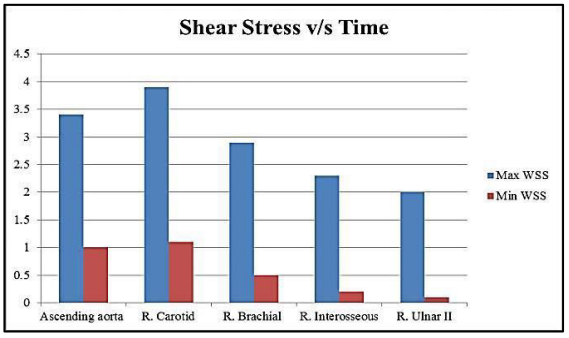


Fig. 6 Maximum and minimum Wall Shear stress (WSS) v/s Time plot for inlet and various Outlet arteries

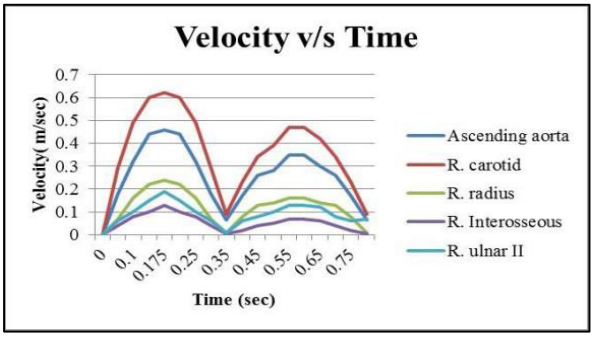


Fig. 7 Velocity v/s Time plot for inlet and various Outlet arteries

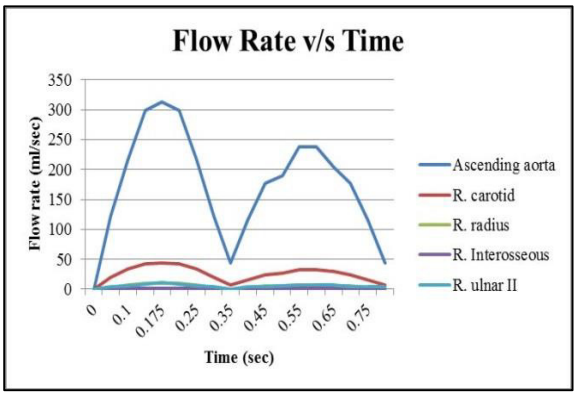


Fig. 8 Flow Rate v/s Time plot for inlet and various Outlet arteries

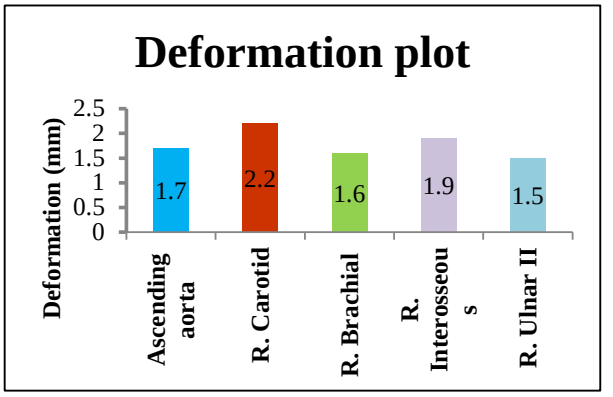


Fig. 9 Radial Deformation in the arteries due to the pulsatile flow

## 3. Validation of the Finite Element Simulation

The results obtained using the finite element simulations were found to be in agreement with the earlier published literature. The simulations carried out by the other researchers were limited to modelling single sections of the arterial network in order to study the effects of stenoses[13], aneurysms, anatomical variations [14] and vessel occlusions[15,16]. The simulation study carried out in this research has modelled the arteries in the entire human arm right from the ascending aorta up to the ulnar II. Besides, the pulsatile nature of blood flow has been scarcely studied with the actual cross-section of the artery which is made up of layers of different densities. The blood velocity obtained in the simulation at the ascending aorta is 0.46 m/sec, which in agreement with 40-50 cm/sec [17], 40 cm/sec[18, 19]. Flow rate at the ascending aorta cited by Pieles et al[20] is 415 ± 83 ml/sec, while Frans et. al[21] have stated it in the range of 300 to 550 ml/sec which very well validates the flow rate obtained in this simulation.

The flow rate (ml/sec) has been calculated by multiplying the peak systolic velocity (m/sec) of the vessel with the cross sectional area ($m^2$). Maximum flow rate of 312 ml/sec has been obtained at the ascending aorta which reduces to 11.19 ml/sec at the ulnar II artery. The flow rate in the simulation reaches its maximum in 0.175 sec. Thus multiplying this duration ($\Delta t$) by flow rate ($\Delta Q/\Delta t$) will yield the stroke volume (ml) i.e. flow at the ascending aorta per beat. The simulation has been carried out for a cardiac cycle of 0.8 sec, thus the heart rate would be 60/0.8 i.e. 75 beats per minute, where the range of heart rate for a normal beating heart is 60 to 100 beats /min. The cardiac output obtained from simulation is 4095 ml/min ( the range of cardiac output for normal human being is 4000 -6000 ml/min). Cardiac output has been caclulated using Eq. 6 [22].

$$Stroke\ volume(ml) = Flowrate(ml/sec) * max\ rise\ time(sec)$$
$$CardiacOutput(L/\min) = Stroke\ Volume(ml/beat) * Heart\ Rate(beats/min) \quad (6)$$

The normal range of wall shear stress in a human artery for blood flow is between 0.5 to 4 Pa[23]. The simulation yielded a maximum wall shear stress of 2.6 Pa the in the ascending aorta which is also well within the maximum limits of the stress, thereby making the simulation more realistic. The simulation results exhibit zero velocity at the walls and maximum at the centre of the vessel, portraying a laminar blood flow.

## 4. Conclusion

The finite element modelling and simulation of the arteries within the human arm has been presented in this paper. Most researchers have modelled either small sections of the arteries or stenosed arteries. The work presented

in this paper considers the arterial network in the entire human arm and analyses the various parameters related to the hemodynamics of the blood flow right from the ascending aorta to the ulnar artery. The wall shear stress obtained in the simulation is 4 Pa which is well within the standard limits. The max velocity 0.46 m/sec is obtained for ascending aorta and in the entire arterial tree the velocity profile obtained is laminar. The reduction in pressure by 3.6036 %, velocity by 71.7391 % and flow rate by 96.4162 % has been observed from ascending aorta to the ulnar II artery of the human arm. These variations can be used to devise relations between the aortic and the peripheral blood flow parameters after incorporating the effects of height and weight of the individual. The selection of proper inlet pulse equations and the boundary conditions has helped in obtaining the valid results for the arterial model. This simulation can be further applied to actual arterial geometry obtained from the CT scan images to help the clinical investigations.

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