



MODELLING AND SIMULATION OF MYOCARDIAL INFARCTION IN THE HUMAN CARDIOVASCULAR SYSTEM

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ABSTRACT

Modelling the physiological processes leading to myocardial infarction can help ameliorate the severity of the condition by improving early detection. Thus, the aim of this study was to model the cardiovascular system and simulate its response to myocardial infarction. Two methods were deployed for this simulation. The first method is the Computational Fluid Mechanics approach, simulated using Mathematica and the solutions of the resulting equations were obtained using Differential Transform Method. The second method is the Lumped Parameter method, simulated using MATLAB/Simulink. With Computational Fluid Mechanics, at 0% blockage within the arteries, no significant stress on the arterial wall was observed. At 10% and 50% blockage levels, a gradual increase in stress from the inlet through the entire arteries' length was observed. 100% blockage resulted in an exponential increase in the stress. A similar output was seen with the Lumped Parameter approach. The blood flow decreases rapidly and reaches zero at a pressure of about 170mmHg. The responses of the different arteries to myocardial infarction as simulated can be applied in the early detection of heart diseases.

Keywords: myocardial infarction, stress, pressure, blood flow, arterial blockage.

1. INTRODUCTION

Myocardial infarction, commonly known as a heart attack, occurs when blood flow decreases or stops to a part of the heart, causing damage to the heart muscle. Most myocardial infarctions occur due to coronary artery disease with underlying risk factors such as high blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, and excessive alcohol intake [1, 2].

In a previous study [3], the authors developed a four compartment model of the cardiovascular system with an algorithm that determines the most important model parameters and used these as a basis for a four stage process which accurately determines all parameter values. The model accounts for aortic insufficiency and pressure changes in the vascular tree which results from changes in vascular compliance and vascular resistance. However, the simplicity of the model allows for further refinements in order to model different conditions including blood loss and haemorrhagic shock. A similar study [4]

presented a two-dimensional modelling and simulation of blood vessel using *COMSOL Multiphysics*. The rationale behind the study is that mathematical modelling and numerical simulation can lead to better understanding of the phenomena involved in vascular diseases. The study assumed that the fluid is laminar, non-Newtonian, viscous and compressible and the arterial wall is elastic. Parameters of blood vessel were assigned to the wall and parameters for fat deposits to the blockages.

In another study, the pulsatility of coronary circulation based on vascular geometry, branching pattern and material properties of the coronary vasculature has also been simulated [5]. A Womersley-type mathematical model was developed to analyse within the entire coronary tree, the pulsatile blood flow in diastole, in the absence of vessel tone. The authors were able to predict the pressure distribution, impedance and the pulsatile flow distribution. The results were validated by experimental measurements in six diastolic-arrested,

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vaso-dilated porcine hearts. In addition, the assumption that blood is a two-layer fluid (which can be considered as the suspension of erythrocytes that behave like a non-Newtonian fluid in the core region and as a Newtonian fluid in the peripheral region of plasma) was used to study the pulsatile flow of blood through a catheterized artery [6]. The result is a mathematical model of nonlinear implicit system of partial differential equations. By solving the system of nonlinear implicit partial differential equations, the authors established that the longitudinal impedance and wall shear stress decrease while the flow rate and velocity increase with the increase in the thickness of peripheral layer.

The Lumped Parameter method has also been used to represent blood circulation in the cardiovascular system [7]. The pumping of the heart, the systemic circulation and pulmonary circulation were represented through eight compartments of the body. The pumping heart was modelled by the time-dependent linear curves of compliances in the heart. The numerical results are consistent with those published in literature for normal hearts. In a similar study, the Lumped Parameter model was used to simulate the blood flow in systemic arteries [8]. This is a review of different approaches to arterial tree modelling and discussion on the applications of such models. Lumped parameter method has also been used for simulating a model which describes the hydrodynamic parameters of the human cardiovascular system using 36-vessel model [9]. The result shows that the calculated pressure for aorta from the complex circuit is similar to measured pressure obtained with the use of advanced medical instruments. One study [10] proposed an analog electrical model of the coronary circulation for patients with obstructive disease undergoing revascularization. The proposed model permits an original biomechanical analysis of pre-operative hemodynamic data for quantitative evaluation of pressure and flow within stenosed arteries, collateral network and bypass grafts. The model thus provides a computational tool to evaluate therapeutic strategies for a patient.

In addition to all of the above studies, a mathematical model of the human cardiovascular system using Lumped Parameter method has also been developed [11]. The model is divided into three parts: systemic circulation, pulmonary circulation and the heart. The established mathematical model was simulated with *MATLAB* software. The innovation of

the study is in describing the system based on the vessel diameters and simulating mathematical equations with active electrical elements. The developed model is useful to understand the anatomy of the human cardiovascular system and related syndromes.

The aim of the present study therefore is to model the cardiovascular system and simulate its response to myocardial infarction using the Computational Fluid Mechanics approach as well as the Lumped Parameter approach and to compare the outcomes of both methods.

2. METHOD

Two methods were used in this study to model the human cardiovascular system with application to myocardial infarction (MI):

- i. Computational Fluid Dynamics (Fluid Mechanics) approach
- ii. Lumped Parameter (Electronic Components) approach

2.1 Computational Fluid Dynamics using Differential Transform Method

This computation is based on the Navier-Stokes algorithm as given in equation 1.

$$\frac{\partial u}{\partial t} + (u \cdot \nabla)u = \frac{1}{\rho} \nabla \cdot \sigma \tag{1}$$

Continuity

equation: $\nabla \cdot u = 0.$ (2)

where: ρ is the plaque density; u is the blood velocity σ is the stress applied on the arterial wall

In order to make the model consistent, a minimum set of parameters with meaningful values was determined in such a way that the rest of the undetermined values can be calculated with new determined parameters.

The model was developed using *Mathematica* and the solutions of the resulting equations were obtained using Differential Transform Method (DTM). The modelling was also based on the physiological assumptions that blood is incompressible, flow in the vessels is laminar, blood flow in and out is constant, flow is non-Newtonian, artery walls are elastic, and that there is no reaction of blood with vessel walls. The algorithms for the computational fluid dynamics approach are summarized as:

1. Boundary conditions: $\sigma(1) = \alpha 1; u(0) = 0; U(1) = 1; \rho = 1050 \text{ kg/m}^3$ (plaque deposit)

2. The momentum equation was solved to give the governing equation described in equations (1) and (2) above.

Noting that

$$\nabla \cdot \sigma = \frac{\partial}{\partial x} \tag{3}$$

Equations (1) and (2) becomes

$$\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} = \frac{1}{\rho} \frac{\partial \sigma}{\partial x} \tag{4}$$

$$\frac{\partial u}{\partial x} = 0 \tag{5}$$

Substituting equation 5 into 4, we obtain that:

$$\frac{\partial u}{\partial t} = \frac{1}{\rho} \frac{\partial \sigma}{\partial x} = 0 \tag{6}$$

Transforming (6) with DTM, the equation can be written as:

$$(h + 1)U(k, h + 1) - \frac{1}{\rho} (k + 1) \sigma (k + 1, h) = 0 \tag{7}$$

where h and k represents the variables of the transformed function, $U(h, k)$

3. Equation (7) was solved using the following values of σ :

$$\sigma[2, 0] = \rho/2; \sigma[2, 1] = \rho; \sigma[2, 2] = 3\rho/2; \sigma[2, 3] = 2\rho; \sigma[2, 4] = 5\rho/2; \sigma[2, 5] = 3\rho; \sigma[2, 6] = 7\rho/2.$$

The blockages were modelled with variation in height from 10%, 50% and 100% of their respective diameters.

2.2 Lumped Parameter Model (Electrical Components Approach)

Equivalent electronic circuit (lumped model) is frequently utilized to signify blood flow and pressure in the cardiovascular system. These lumped models can be achieved from electronic circuit equivalences [11] where current denotes arterial blood flow and voltage characterizes arterial pressure. Resistances

signify arterial and peripheral resistance that occur as a result of viscous dissipation inside the vessels; capacitors characterize volume compliance of the vessels that lets them accumulate blood and inductors represent inertia of the blood.

The correlations between electrical characteristics of the system and equivalent mechanical analogous components are as follow: 0.01ml/Pa \cong 1 μ F (compliance \cong capacitance); 1 Pa.s²/ml \cong 1 μ H (inertia \cong inductor); 1 Pa.s/ml \cong 1k Ω (resistance \cong resistance); 1 mmHg \cong 1 volt (pressure \cong voltage); 133416 ml \cong 1A (volume \cong charge).

The following equations were generated for simulation:

Blood vessel resistance (R), depending on blood viscosity and vessel diameter, is simulated by resistors

$$R = \frac{128\mu \ell}{\pi D^4} \tag{8}$$

where: μ is blood viscosity; ℓ and D are respectively length and diameter of each artery segment.

The blood inertia (L) is simulated by inductors:

$$L = \frac{4\rho \ell}{\pi D^2} \tag{9}$$

where ρ is blood density. The vessel compliance (C) is modelled as a capacitor.

$$C = \frac{\pi \ell D^3}{4Eh} \tag{10}$$

where: D is artery diameter; E is modulus of elasticity of the artery, and h is the thickness of artery.

Given $\mu = 0.04\text{gcm}^{-1}$; $\rho = 1.0\text{gcm}^{-3}$; $E = 2 \times 10^6\text{gcm}^{-1}\text{s}^2$; $h = 0.08D$, each artery is modelled by R, L, C element to obtain equivalent electrical circuit of an artery in *Simulink (MATLAB)* as shown in Figure 1. The computed values of the elements are shown in Table 1.

Table 1: The resistance, inductance and capacitance (R, L, C) values for each artery at different percentage blockages.

Coronary Artery	D (cm)	L (cm)	% Blockage	R	L	C
Right Coronary Artery	0.31	2.7	10	588.3	39.8	1.08 x 10 ⁻⁶
			50	1906	71.6	4.49 x 10 ⁻⁷
			90	47650	358	4.02 x 10 ⁻⁸
Mid left descending Coronary Artery	0.49	4.2	10	146.6	24.8	4.23 x 10 ⁻⁶
			50	474.96	44.6	1.75 x 10 ⁻⁶
			90	118.74	223	1.57 x 10 ⁻⁷
Mid left ascending Coronary Artery	0.46	1.9	10	85.4	12.7	1.68 x 10 ⁻⁶
			50	276.8	22.86	6.97 x 10 ⁻⁷
			90	6920	114.3	6.23 x 10 ⁻⁸

All values are in standard CGS units.

D = diameter; l = length; R = arterial resistance L = arterial inductance; C = arterial capacitance.

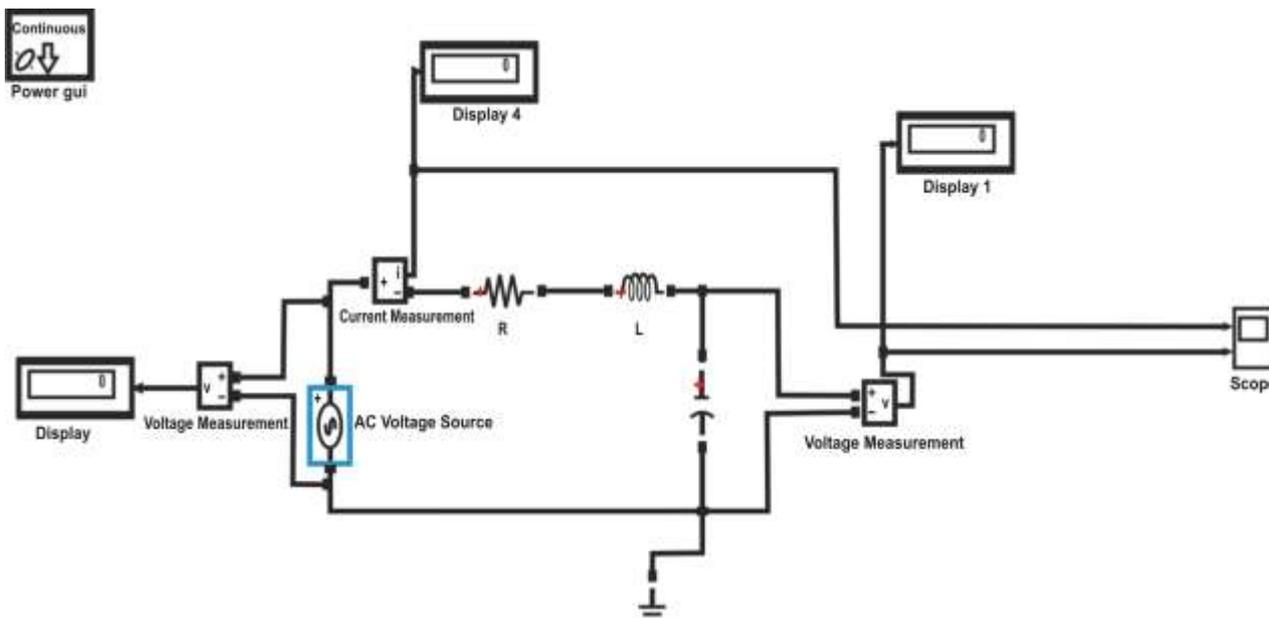


Figure 1: Equivalent circuit model from Simulink (MATLAB)

Each artery was simulated for 0.8 second using *ODE45* (a function in *Matlab/Simulink* for solving differential equations) and blood flow and pressure curves were obtained. In this simulation, pressure of 120mmHg was used.

3. RESULTS AND DISCUSSION

3.1 Results for Computational Fluid Mechanics Approach

The stress on the artery increases as the thickness of the blockage increases. The results show the variation of the stress and length of each artery for 0%, 10%, 50% and 100% blockages. The blockage size of 0% is seen to mount very little or no significant stress on the arterial wall as seen in Figure 2.

Blockages with size 10% and 50% showed gradual increase in stress from the inlet through the arteries as seen in Figures 4-8. This does not result in a sudden occurrence of myocardial infarction; it is a slow reaction as the stress increases gradually. If detected at this stage, it can be treated and a severe reaction avoided.

For blockage of size 100%, the stress generation builds up really high at the inlet and spans through the entire artery length. This indicates that, for larger thickness of blockage, stress generation profile is increased exponentially in the artery. The stress-length profile for 100% blockage in mid-left

ascending and descending coronary arteries are shown in Figures 10 and 11 respectively.

Stress, σ

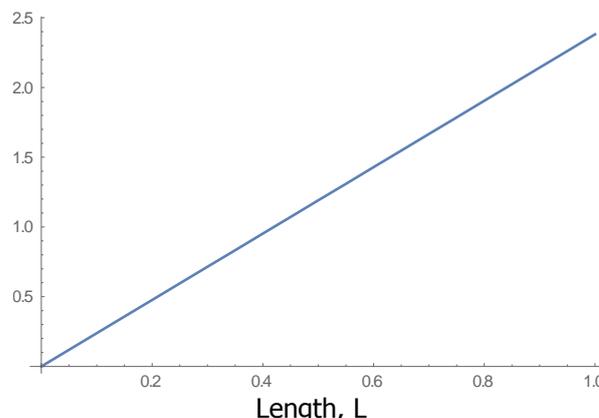


Figure 2: Stress-length profile for 0% blockage

In this simulation, the results obtained indicate that the blockage can be predicted at very early stages. Most importantly, it is possible to detect in which artery the blockage exists, and accurately predict that blockage is near or far away from the heart. Incorporating the actual percentage blockage by observing the pressure contour and stress profile detection of blockages can be done.

The nature of this model allows many phenomena to be observed or incorporated, providing for more accurate simulations of arteries and techniques used to treat associated disease/conditions.

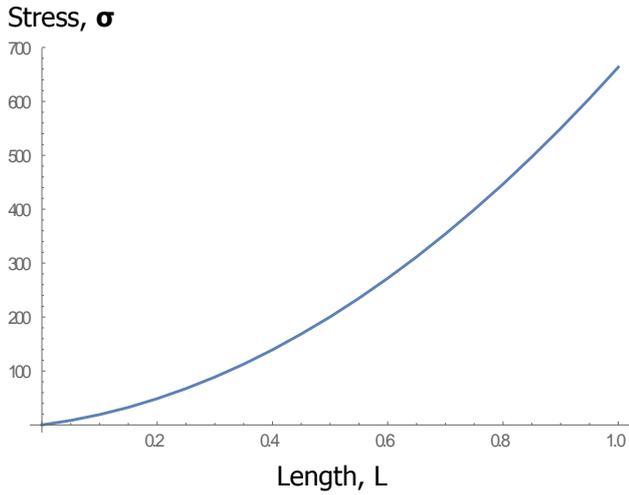


Figure 3: stress-length profile for 10% blockage in right coronary artery

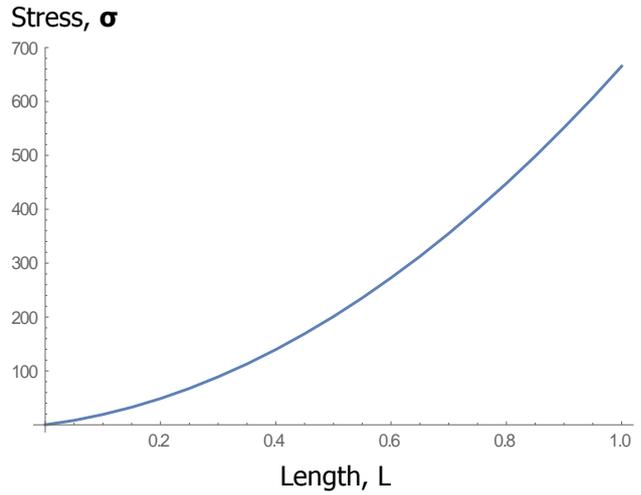


Figure 4: stress-length profile for 50% blockage in right coronary artery

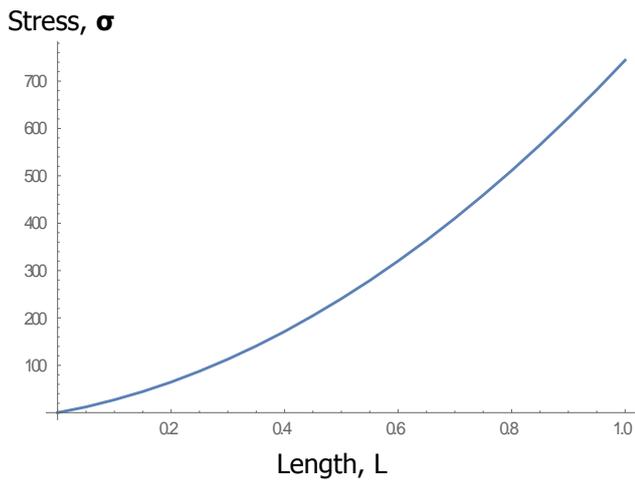


Figure 5: stress-length profile for 10% blockage in mid-left descending coronary artery

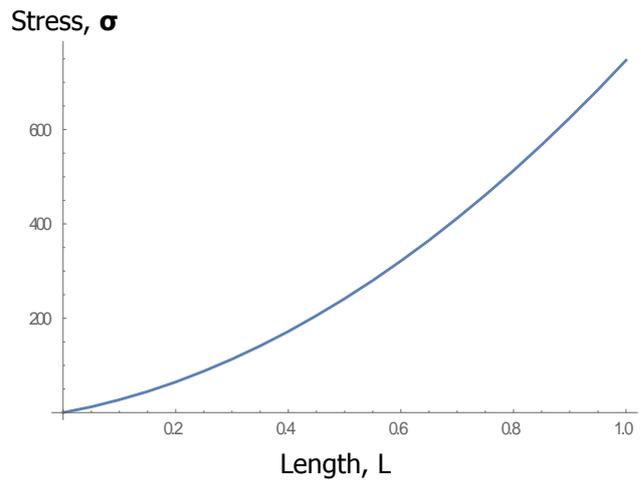


Figure 6: stress-length profile for 50% blockage in mid-left descending coronary artery

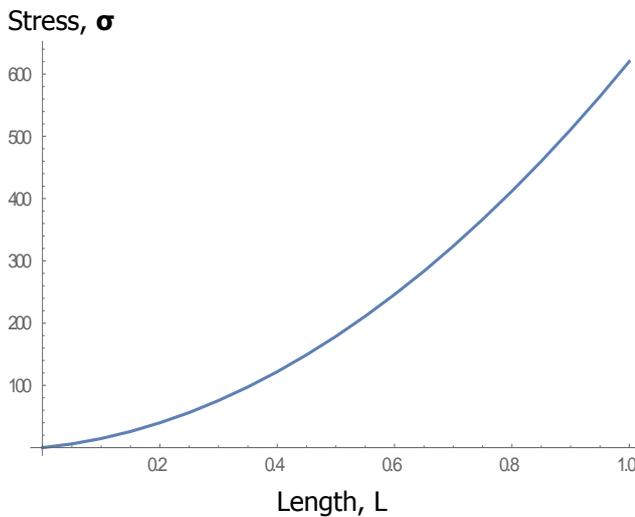


Figure 7: stress-length profile for 10% blockage in mid-left ascending coronary artery

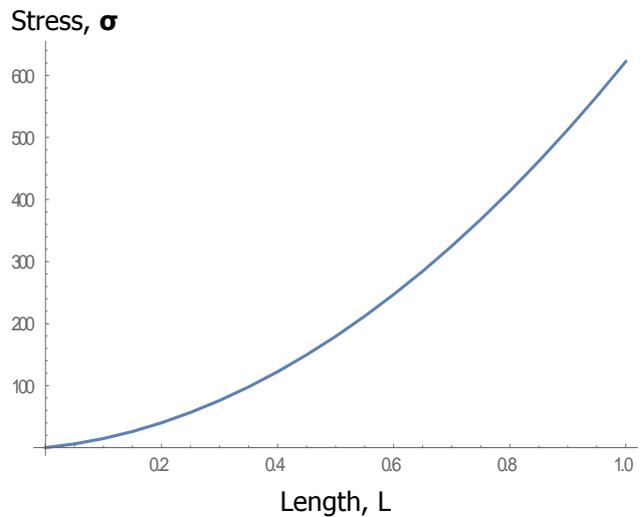


Figure 8: stress-length profile for 50% blockage in mid-left ascending coronary artery

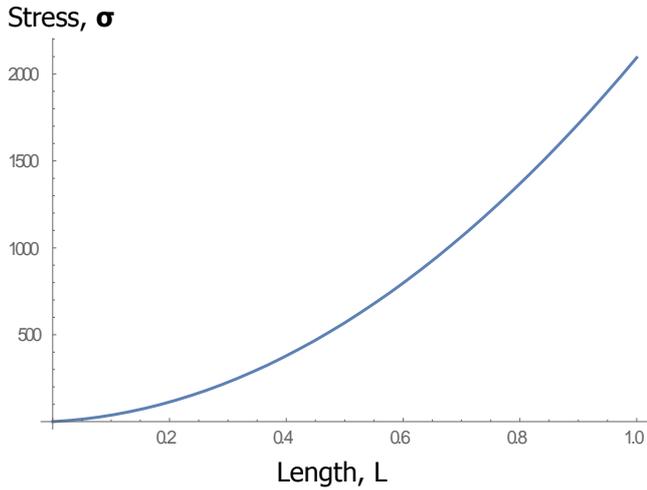


Figure 9: stress-length profile for 100% blockage in mid-left ascending coronary artery

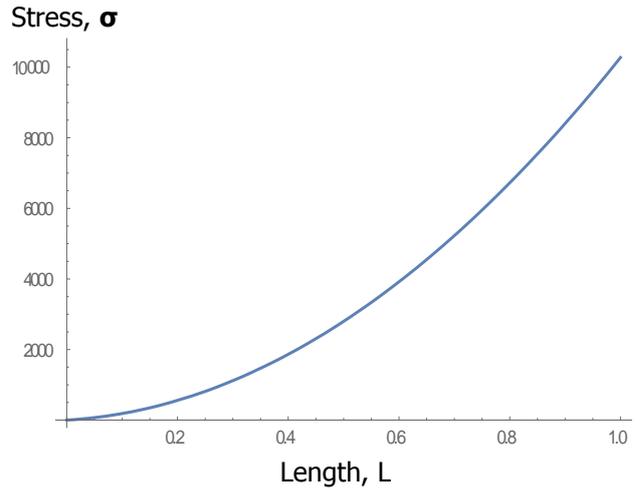


Figure 10: stress-length profile for 100% blockage in mid-left descending coronary artery

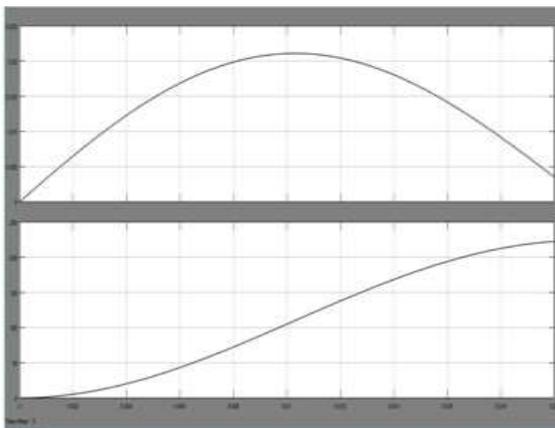


Figure 11: Blood flow and pressure profile at 0% blockage in right coronary artery

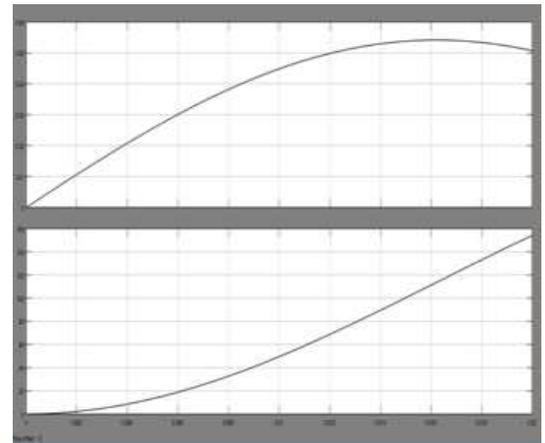


Figure 12: Blood flow and pressure profile at 0% blockage in mid-left descending coronary artery

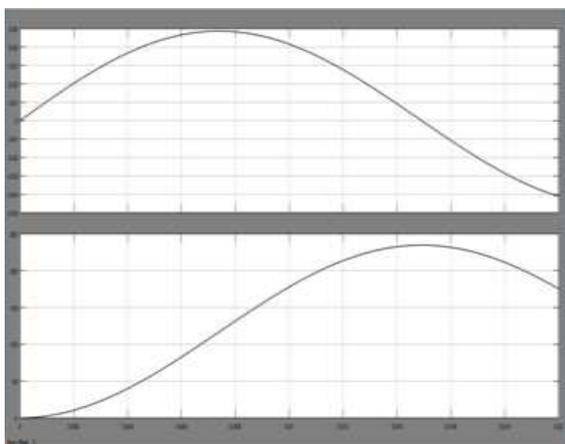


Figure 13: Blood flow and pressure profile at 0% blockage in mid-left ascending coronary artery.

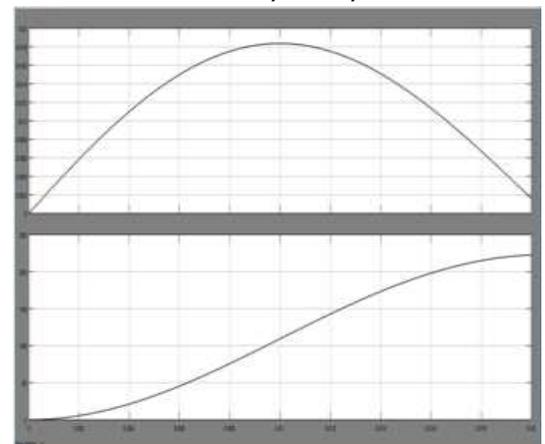


Figure 14: Blood flow and pressure profile at 10% blockage in right coronary artery

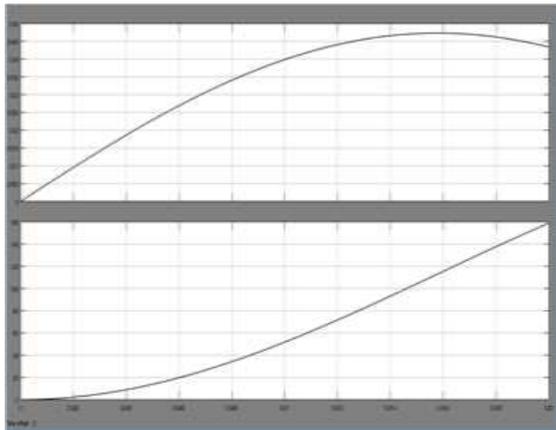


Figure 15: Blood flow and pressure profile at 10% blockage in mid-left descending coronary artery.

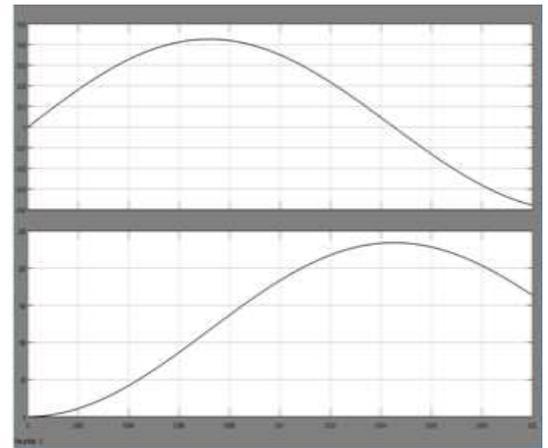


Figure 16: Blood flow and pressure profile at 10% blockage in mid-left ascending coronary artery

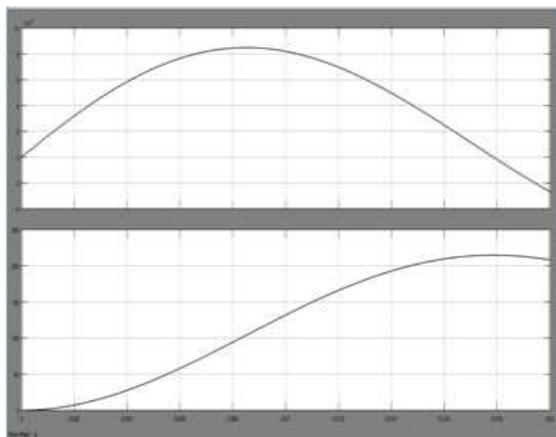


Figure 17: Blood flow and pressure profile at 50% blockage in right coronary artery.

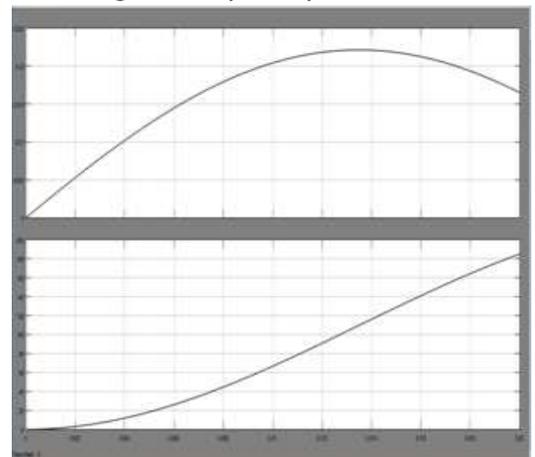


Figure 18: Blood flow and pressure profile at 50% blockage in mid-left descending coronary artery.

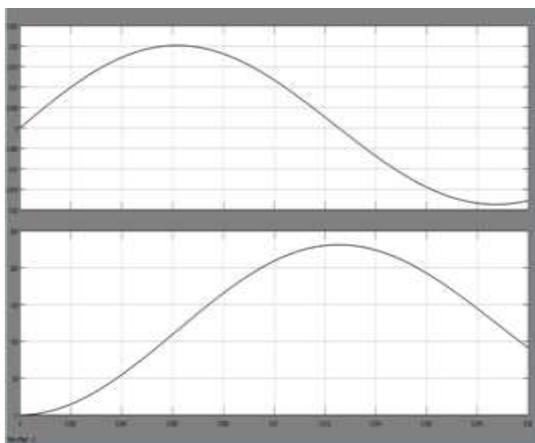


Figure 19: Blood flow and pressure profile at 50% blockage in mid-left ascending coronary artery

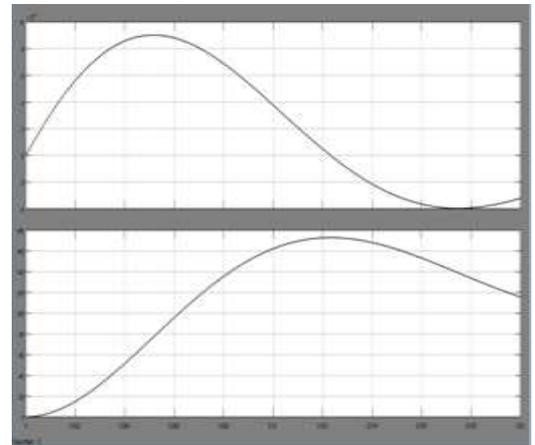


Figure 20: Blood flow and pressure profile at 90% blockage in right coronary artery

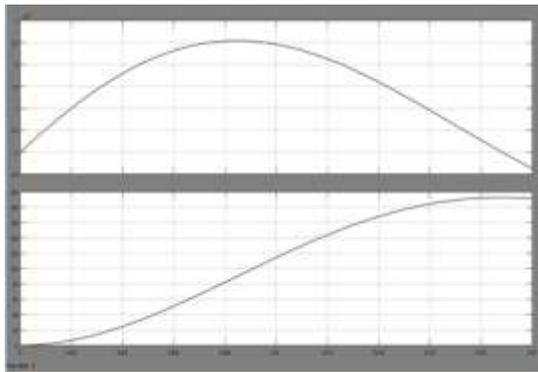


Figure 21: Blood flow and pressure profile at 90% blockage in mid-left descending coronary artery.

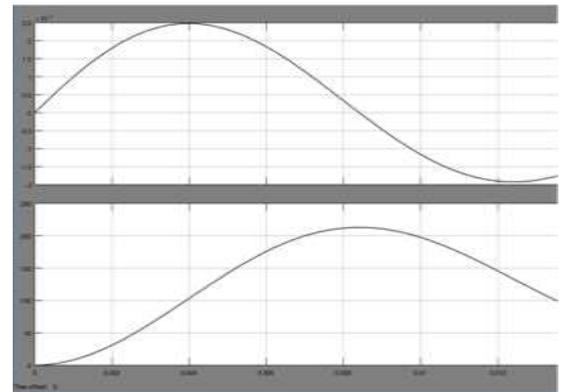


Figure 22: Blood flow and pressure profile at 90% blockage in mid-left ascending coronary artery.

3.2 Results for Lumped Model

It can be seen from Figures 11-13, that there is a steady flow with response to increase or decrease in pressure in an artery with no blockage. The flow rises with pressure, but on getting to 120mmHg which is the average standard pressure, the blood flow begins to drop with a continuous increase in pressure.

For a 10% blockage, the pressure still appears normal as seen in 0% blockage but there is a slight change at the point of decrease in blood flow compared to 0% blockage.

The decrease in blood flow in 50% blockage begins at a pressure of 90mmHg, which means the blood supplied to the heart by the artery is more reduced in 50% blockage with a continuous rise in pressure.

As seen in the graphs of the arteries having 90% blockage (Figure 20 – 22), the decrease in blood flow starts at a pressure of about 100mmHg. The blood flow decreases rapidly and drastically such that at a pressure of about 170mmHg, blood flow reaches zero (0). At this point, the patient is most likely to have a heart attack and if proper measures are not taken, might result to death.

3.3 Comparative analysis of the outputs of the two methods – Computational Fluid Dynamics using DTM and Lumped Parameter Method

The two models used in this study showed similar results when simulated. For the fluid model using differential transform method (DTM), the results as seen in Figures 2 to 10 show the effects of different percentage blockage on the pressure and subsequently stress on the arterial wall. The stress increases with increase in blockage, leading to a

corresponding decrease in the blood flow in the arteries. For the lumped model, the results as seen in Figures 11 to 22 show the blood flow to pressure relation of each artery for different percentage blockage. For pressure increase beyond normal blood pressure of 120mmHg, a decrease in blood flow is observed and continuous decrease occurs with subsequent increase in pressure to such a point that myocardial infarction and eventually death is possible.

4. CONCLUSION

This study presents models for the study of the human cardiovascular system in response to myocardial infarction. It was observed that the increase in stress and pressure and decrease in blood flow are more pronounced in arteries with smaller diameter compared to those of larger diameter. At a young age, the arteries are more elastic, hence the effect of similar blockage is not pronounced. As the arteries harden in adults, it affects the blood flow and blood pumping function of the heart. This model thus provides an insight for future studies on arterial blood flow and coronary artery disease models with lifesaving applications.

Conflict of Interest

The authors declare that they have NO competing interests.

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