

Modeling Blood Flow in the Gastrointestinal System

T. D. S. Mabotuwana, L. K. Cheng, N. P. Smith and A. J. Pullan

Abstract—Compared to cardiac ischemia, relatively little is known about ischemia that develops within the gastrointestinal system. The work presented here is a first step towards developing a detailed anatomically and biophysically based model of the mesenteric arterial system of the human intestine to be used to simulate normal and compromised blood flows. Data from the Visible Human project were used to develop an initial model of the mesenteric arterial tree. Using this tree, equations that govern blood flow within extensible vessels were set up and solved for pressure, radius and velocity. Results were analyzed for the four distinct phases of cardiac contraction - diastole, isovolumic contraction, ejection and isovolumic relaxation and the profiles showing the temporally varying pressure and velocity within the network for a periodic input varying between 10.29 kPa (77 mmHg) and 14.63 kPa (110 mmHg) at the abdominal aorta are presented in this paper.

I. INTRODUCTION

When blood flow through a blood vessel becomes compromised, for example as a result of a partial blockage, the tissues that rely on this blood flow become deprived of oxygen. This reduction in blood flow is commonly known as ischemia. When this happens in the heart, the results can be severe – part of the heart muscle can die, leading to a full scale heart attack and potential death. Fortunately there are some early clinical indicators of cardiac ischemia, most notably a change in a person's electrocardiogram. Less well understood, but potentially just as dangerous, is ischemia that develops in the gastrointestinal system. Such ischemia is difficult to diagnose without surgery, and most sufferers complain of "gut pain" but discriminating ischemia from the many other gut pains (which are more common and less severe) is difficult, even for the trained specialist. Very little is known about the development of gastrointestinal ischemia and if such a condition is left for too long, the only treatment is the removal of large sections of the intestine.

The purpose of this research is to develop an extensible anatomically and biophysically based generic model of the mesenteric arterial system, which is the main blood supply of the human intestine, and to use this model to carefully examine intestinal blood flow and ischemia. As an initial step towards this goal, a model of the human intestine has been developed based on the Visible Human (VH) dataset,

and blood flow and pressure distributions through this network have been simulated.

II. METHODOLOGY

A. Data Digitization

The data required to develop the model were obtained from the male VH dataset. We used the 2D axial slices on which the centre-line of the mesenteric arterial system was visually identified and traced. By stacking these images as shown in Fig. 1(b), an initial 3D model was constructed. In some regions it was difficult to determine the actual boundaries of the mesenteric network, and anatomical text [1] was used to augment the digitized data. A vertical segment of 251 mm of the body was traced (Fig. 1(a)).

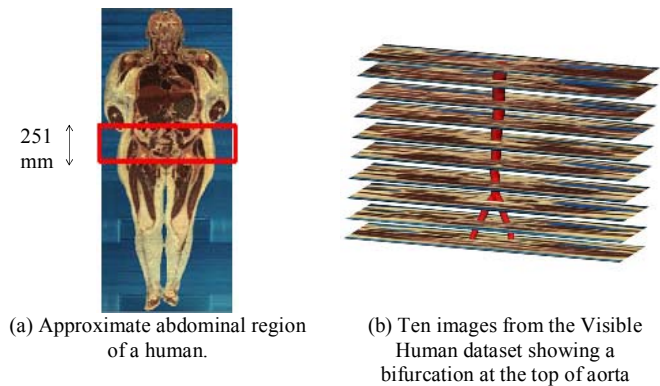


Fig. 1. Digitization of the male Visible Human images.

B. Finite Element Model

Points were selected at regular intervals from the set of raw data points obtained after digitization and used as nodes (darker points in Fig 2(a)) in the construction of the finite element mesh. The selected nodes were then connected linearly to form the initial, linear finite element model. The created linear elements were then fitted to the entire digitized dataset using a 1D cubic Hermite interpolation scheme. The result of this fitting process is the smooth network shown in Fig. 2(b).

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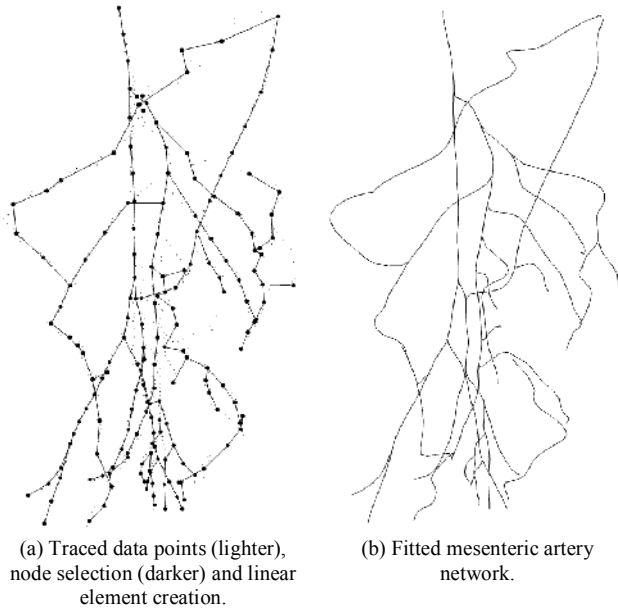


Fig. 2. Finite element creation and fitting of mesenteric arteries.

C. Initial Radius Assignment

The initial undilated radius of the mesenteric arterial mesh was determined via a visual inspection of VH images. Where possible, these radii were checked against other published data to ensure their accuracy. The initial diameters of the abdominal aorta, superior mesenteric artery (SMA) and inferior mesenteric artery (IMA) were considered to be 20.2 mm, 8.8 mm and 7.3 mm respectively with a maximum difference of only 1.3 mm compared to those in published material [2].

Shown in Fig. 3 is the model constructed after the initial radii were assigned.

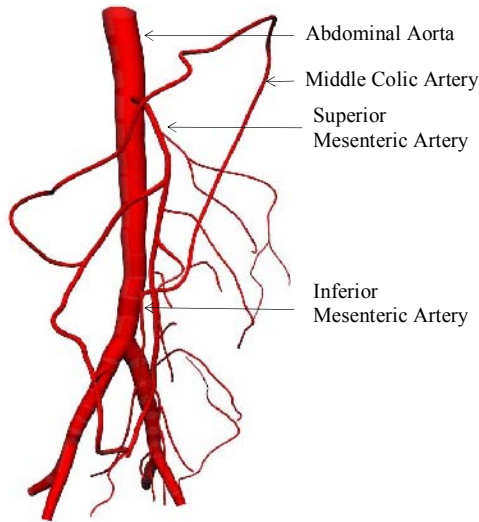


Fig. 3. Anterior view of the 3D anatomical model of a segment of abdominal aorta with the superior and inferior mesenteric arteries and their sub-divisional branches.

D. Modeling Blood Flow

To simulate blood flow through the mesenteric arterial model, we use the same approach as presented in [3]. In that work, blood flow through the coronary arterial network was simulated assuming that the blood was an incompressible, homogeneous, Newtonian fluid with an axisymmetric laminar flow and constant viscosity. Under these assumptions, the complete 3-dimensional Navier-Stokes equations can be reduced to a set of 1-dimensional flow equations. Using a cylindrical coordinate system (r, θ, x) with the x axis aligned with the local vessel axial direction, the following equations can be derived for the 3 unknowns: average velocity (V), pressure (p) and inner vessel radius (R) [3];

$$\frac{\partial R}{\partial t} + V \frac{\partial R}{\partial x} + \frac{R}{2} \frac{\partial V}{\partial x} = 0 \quad (1)$$

$$\frac{\partial V}{\partial t} + (2\alpha - 1)V \frac{\partial V}{\partial x} + 2(\alpha - 1) \frac{V^2}{R} \frac{\partial R}{\partial x} + \frac{1}{\rho} \frac{\partial p}{\partial x} = -2 \frac{v\alpha}{\alpha - 1} \frac{V}{R^2} \quad (2)$$

and

$$p(R) = G_0 \left[\left(\frac{R}{R_0} \right)^\beta - 1 \right] \quad (3)$$

where α is a parameter used to define the axial velocity profile, G_0 and β are constants defining a particular wall behavior and R_0 is the initial vessel radius. ρ and v represent blood density and viscosity respectively.

III. RESULTS

Following the studies of [3], the selected material values used in flow analyses were $G_0 = 21.2$ kPa (158 mmHg), $\beta = 2.0$ and $\alpha = 1.1$ due to the nature of the arterial walls. Blood density was assumed to be 1.05 gm^{-3} and viscosity to be $3.2 \text{ cm}^2\text{s}^{-1}$.

Using these parameter values, equations 1, 2 and 3 were solved for pressure, radius and velocity using the two-step Lax-Wendroff finite differencing scheme. At the distal ends of the network, a constant pressure of 9.6 kPa (72 mmHg) was imposed. This value was just below the diastolic pressure imposed at the inlet. At the proximal end of the network, the blood inflow was set to have a periodic pressure pulse varying between 10.29 kPa (77 mmHg) and 14.63 kPa (110 mmHg). This pressure profile (Fig 4) was based on data from [4] and represents the four distinct cardiac contraction phases – diastole, isovolumic contraction, ejection and isovolumic relaxation.

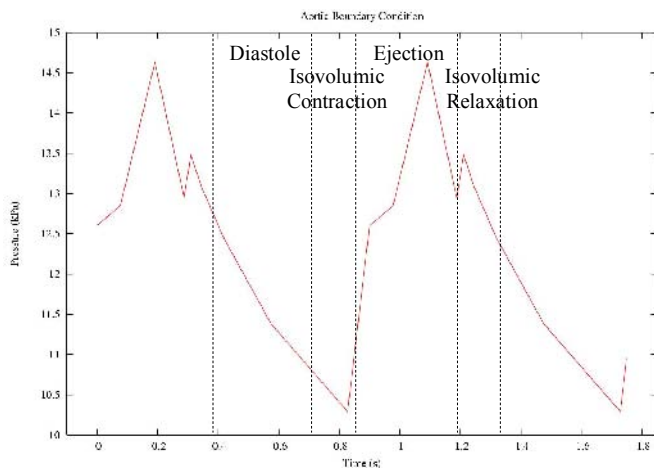


Fig 4. Aortic inflow pressure boundary condition.

For our initial computations, the middle colic artery was removed from the network and flow was simulated using the network shown in Fig. 5. The middle colic artery connects the SMA back onto the IMA, and this loop allows complicated retrograde flows to be generated in our simulations, which can lead to instabilities. For these reasons, this loop was excluded in our initial simulations.

Although the flow equations were solved for pressure, radius and velocity, only pressure and velocity results are presented herein as the radii changes over time were less than $\pm 5\%$ of initial radius.

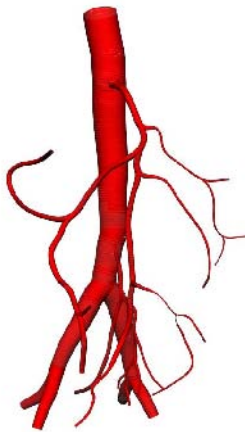


Fig. 5. Simplified mesenteric artery network, obtained from that shown in Fig 3 through the removal of the middle colic artery.

Pressure profiles were examined at different times for the pressure input shown in Fig. 4, and Fig. 6(a) – 6(d) show the pressure distribution of the network during the four distinct cardiac contraction phases.

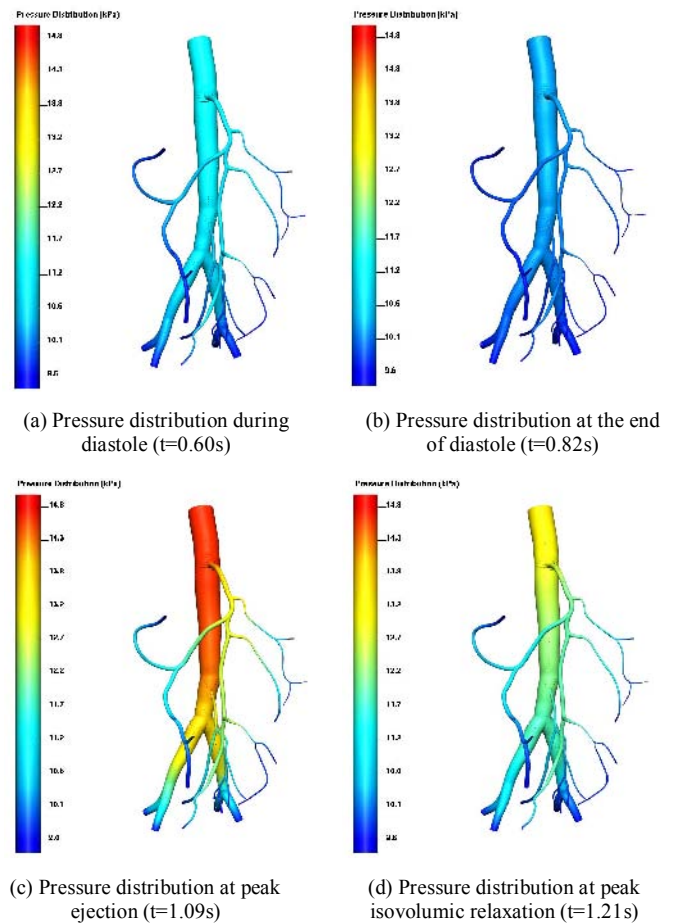


Fig. 6. Pressure distribution of the simplified mesenteric artery network at selected times.

During diastole, there is relatively a smaller pressure gradient in the entire tree since the distal ends of the network are held at a constant 9.6 kPa. The reduction in this pressure gradient becomes more evident in Fig. 6(b) when the proximal end pressure reaches 10.29 kPa.

As vessels bifurcate giving rise to daughter vessels whose area sum is greater than the area of the parent, the pressure drops and these pressure changes can be observed clearly in Fig. 6(c) where the pressure gradient is steepest.

During isovolumic relaxation, the ventricles relax and pressures gradually return to the rest state. Peak isovolumic relaxation (Fig. 6(d)) was determined to be 13.48 kPa (101 mmHg).

Velocity profiles were also examined and Fig. 7(a) – 7(d) show the corresponding velocity distribution in the network for the above times.

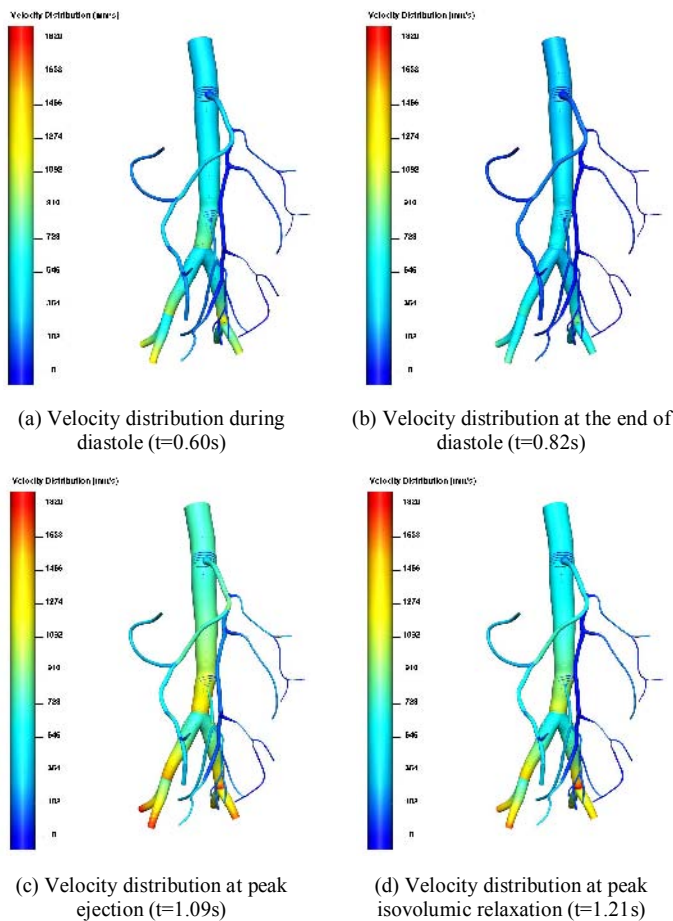


Fig. 7. Velocity distribution of the simplified mesenteric artery network at selected times.

Similar to the pressure profiles, a drop in velocity can be observed at each bifurcation as the area sum of daughter vessels is greater than the area of the parent.

During the diastole phase, arterial velocity decreases as the pressure boundary condition at descending aorta drops, while venous velocity rises as flow begins to be transmitted through vessels with smaller radii.

With the onset of ejection, the rapid rise in aortic pressure begins to dominate velocities. The rising pressure produces a drop in radii in the low pressure, venous vessel network, resulting in an increase in velocity as blood is squeezed out of the vessels.

IV. DISCUSSION

Modeling mesenteric arterial blood flow will hopefully increase our understanding of blood supply to the intestine. The work presented here is our first step towards our goal of developing an extensible, anatomically and biophysically based model of the mesenteric arterial system to be used to carefully examine intestinal blood flow and ischemia. Our initial model has been based on images from the male VH dataset which represent the geometry of a deceased person. Therefore, we now intend to develop a new model based on MRI images of a healthy volunteer, which should lead to a

better anatomical description of the network of a living human. We also plan to carefully examine the numerical procedures we are using to simulate blood flow with the aim of including the middle colic artery currently excluded from our model. Ultimately, we plan to use these models to simulate ischemic conditions as occur in those suffering from mesenteric ischemia.

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