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Abstract

The objective of this paper is to present the mixed velocity-pressure (v-p) finite element method that solves the pulsatile blood flow in arteries. The solution exploits the Galerkin method and the fully implicit incremental-iterative procedure for the three-dimensional nonlinear finite element equations. This methodology is applied to model biological flows that are important in predicting growth and rupture risks in abdominal aortic aneurysms (AAA). The numerical technique was validated with the analytical solution of the Womersley model. Next, a physiologically realistic pulsatile blood flow waveform was imposed onto the idealized cylindrical arterial model and solved as a benchmark problem. The model represents a healthy abdominal aorta. This pulsatile condition simulates an in vivo aorta at rest. The numerical results were used to quantify clinically relevant flow dynamics that play a significant role in today’s field of medical treatment planning and development of predictive methods via computational modelling for assessing common clinical problems such as AAAs.

Keywords: Three-dimensional finite element methods; Pulsatile flow dynamics; Abdominal aorta.

1. Introduction

Abdominal aortic aneurysm (AAA) is a common clinical problem that requires determination of hemodynamic conditions and subsequent rupture prediction. AAAs, are aneurysms occurring in the abdominal part of the aorta, more specifically, between the renal bifurcation and the iliac branches. The main causes of aneurysm are arteriosclerosis and cystic medial degeneration, but also genetic disorder, malfunction of the aorta (i.e. biomechanical phenomenon), mycotic infections or arthritis can be a cause of aneurysm [1]. Another cause of aneurismatic disorders mentioned by scientists is the loss of distensibility of the vessels.
This pathologic condition has been found to affect 8.8% of the population over the age of 65 and if left untreated it may lead to rupture [2]. Although the size of the aneurysm and its rate of expansion are parameters widely associated with the risk of rupture, it is important to understand the flow dynamics of pulsatile flow in a healthy aorta and its flow implications under an aneurismal condition [3].

A fundamentally new approach in medical treatment planning and development of predictive methods in clinical applications is computational modelling. Mathematical models can help to interpret non-invasive monitoring techniques. The numerical methods used to study AAA development vary from one researcher to another. However, the finite element method is commonly used these days [4,5,6]. The key factor that determines the reliability of the finite element scheme is its stability. Generally, the finite element computation of incompressible flows involves two main sources of potential numerical instabilities associated with the Galerkin formulation of a problem.

One source is due to the presence of advection terms in the governing equations, and can result in spurious node-to-node oscillations primarily in the velocity field. Such oscillations become more apparent for advection-dominated (i.e. high Reynolds number) flows and flows with sharp layers in the solution.

The other source of instability is due to using inappropriate combinations of interpolation functions to represent the velocity and pressure fields. These instabilities usually appear as oscillations primarily in the pressure field. In last decades a number of stabilization procedures have been developed to prevent potential numerical instabilities described above [7,8,9,10,11]. In order to guarantee the stability of the scheme, the finite elements for velocity and pressure need to be selected in a proper way to satisfy the inf-sup condition of Ladyzhenskaya-Babuska-Brezzi (LBB) condition [12].

On the finite element level, we consider solving a system of equations in a finite element space $V_h \times P_h$, where $V$ and $P$ are velocity and pressure respectively. Here the parameter $h$ usually refers to the mesh size. The stability properties of the finite element method based on $V_h \times P_h$ is determined by the inf-sup condition such that the inf-sup constant is:

$$\gamma_h := \inf_{p \in P_h} \sup_{v \in V_h} \frac{\int_{\Omega} pd{\bf v} \cdot {\bf v}}{\|v\|_{1,\Omega} \|p\|_{0,\Omega}} > 0$$

where $\Omega$ is a polygonal domain. A finite element is called stable if there exists $\gamma > 0$ independent of $h$ such that $\gamma_h \geq \gamma$ holds for any mesh of the domain $\Omega$ and for any mesh size $h > 0$. Similarly, a finite element is said to be stable for a mesh family if $\lambda_h$ can be bounded by a positive number for any mesh of the mesh family. Therefore, with good approximation properties and finite element method can be stable.

We aim to derive a complete set of three-dimensional finite element equations arising from the mixed velocity-pressure (v-p) finite element method by implementing the Galerkin method and the fully implicit incremental-iterative procedure for solving the nonlinear Navier Stokes and continuity equations that represent blood as a viscous incompressible Newtonian fluid, that can be applied to solve pulsatile flow problems in arteries.

2. Methods

2.1. Governing equations

Except in tiny capillaries, the blood flow can be assumed to behave as a continuum, as well as incompressible, apart from severe pathological situations [13]. Although, in reality blood is a non-Newtonian suspension of cells in plasma, but it is reasonable to model it as a Newtonian fluid in vessels greater than approximately 0.5mm in diameter [14]. The
three-dimensional governing equations (momentum and continuity equations) of a viscous incompressible Newtonian fluid (blood) flow, using the indicial notations with the usual summation convention are, respectively,

$$\rho \left( \frac{\partial v_i}{\partial t} + v_i v_{i,j} \right) = \sigma_{y,j} + f^B_i$$

(1)

$$v_{i,j} = 0$$

(2)

where \(v_i\) is velocity of blood flow in direction \(x_i\), and summation is assumed on the repeated (dummy) indices, \(i, j = 1, 2, 3\). \(\rho\), \(\sigma_{y,j}\) and \(f^B_i\) are the fluid density, fluid stress and body force, respectively. Its prescribed velocity on the surface \(S_1\) and boundary force on the surface \(S_2\) as the governing boundary conditions imposed onto equations (1) and (2) are, respectively,

$$v_i = \bar{v}_i \bigg|_{S_1}$$

(3)

$$\sigma_{y,j} n_j = f^S_i \bigg|_{S_2}$$

(4)

where the fluid domain of interest \(S\) with its boundaries are given as;

\[ S_2 \cup S_1 = S \quad \text{and} \quad S_2 \cap S_1 = \emptyset \]

(5)

Next, a constitutive equation for the fluid stress for Newtonian fluid is introduced as;

$$\sigma_{y,j} = -p \delta_{y,j} + 2\mu \dot{e}_{y,j}$$

(6)

where, \(p\) is the fluid pressure, \(\mu\) is the dynamic viscosity of the fluid, \(\dot{e}_{y,j}\) is the tensor of velocity deformation, \(\dot{e}_{y,j} = \frac{1}{2} \left( \frac{\partial v_i}{\partial x_j} + \frac{\partial v_j}{\partial x_i} \right)\) and \(\delta_{y,j}\) is the Kronecker delta. Substituting equation (6) into equation (1) and subsequently applying equation (2) gives rise to the final form of the flow equation;

$$\rho \left( \frac{\partial v_i}{\partial t} + v_i v_{i,j} \right) = -p_j + \mu \dot{e}_{y,j} + f^B_i$$

(7)

2.2. Pulsatile flow and boundary conditions

A physiologically realistic pulsatile blood flow simulating an in vivo cardiac cycle of the abdominal aorta section at rest was imposed at the inlet of the non-dilated aorta entry, \(d_{AA}\), as shown in Figure 1 [15]. The mean Reynolds number, \(Re_{\text{mean}} = 4Q_{\text{mean}}/\pi \nu d_{AA}\) and peak Reynolds Number, \(Re_{\text{peak}} = 4Q_{\text{peak}}/\pi \nu d_{AA}\) of the pulsatile flow were 525 and 2325, respectively. The mean Reynolds number is based on the mean flow rate, \(Q_{\text{mean}}\) of a full cardiac cycle whereas the peak Reynolds number is based on the peak systolic flow rate, \(Q_{\text{peak}}\) of the pulsatile flow. The pulsatile flow consists of a pulse frequency, \(\omega\) of 60beats/min, so that the Womersley number, \(\alpha = 0.5d_{AA} \sqrt{\omega/\nu} \approx 12\), where \(\nu\) is the kinematic viscosity of blood which was taken to be 3.5mm²/s.

At the inlet of the aorta, the pulsatile flow imposed is considered to be a fully developed flow in an infinitely long straight cylindrical tube model. All velocity components at the fixed vessel wall of the aorta model are prescribed as zero. The no-slip condition at the inner vessel wall was adopted. Along the symmetric plane of the model, the velocity components normal to this plane and the tangential stresses are imposed to be zero. At the outlet of the aorta, the normal and tangential stresses are set to be zero as well respectively (i.e. stress-free condition). Therefore, near the outlet of the aorta the solution differs from the solution in an infinitely long straight cylindrical tube model.
2.3. Finite element formulation

The advantage of the mixed velocity-pressure (v-p) formulation is that the pressure, velocity, velocity gradient and stress boundary conditions can be directly incorporated into the finite element matrix equations [12]. A 21/8 node 3D brick element was used for this three-dimensional analysis. 21 nodes were employed to interpolate the velocities and 8 nodes were employed to interpolate the pressure, hence, providing stable elements expressed by the inf-sup condition of Brezzi-Babuska. The finite element mesh of the model consists of 7,200 elements and 8,450 nodes, as shown in Figure 2.

Applying the Galerkin method in equations (2) and (7) yields;

\[ \int_V G_s v_i dV = 0 \]  
\[ \int_V \rho \frac{\partial v_i}{\partial t} dV + \int_V H_s v_i v_j dV = -\int_V H_s p_{ij} dV + \int_V \mu H_s v_{ij} dV + \int_V H_s f_i dV \]  

Integrating by parts and transforming the volume to surface integral of equation (9) yields;
\[
\rho \int_V \frac{\partial \text{conserved variables}}{\partial t} dV + \rho \int_V \text{body forces} dV - \int_V \text{boundary forces} dS = \int_V \nabla \cdot \text{divergence term} dV
\]

The chosen interpolation functions for the velocity and pressure are, respectively;

\[
v_i = H_u v_{ia}
\]

\[
p = G_s p_s
\]

Next, equations (11) and (12) are substituted into equations (10) and (8) to arrive at;

\[
\left[ \rho \int_V H_a H_{i\beta} dV \right] v_{i\beta} + \left[ \rho \int_V H_a H_{\gamma\beta} H_{\gamma i} dV \right] v_{\gamma i} + \left[ \int_V \mu H_a H_{\beta i} dV \right] v_{i\beta} - \left[ \int_V H_a H_{\gamma i} dV \right] p_\gamma = \int_V H_a f_\beta dV + \int_S H_a (-p n + \nu \cdot n) dS
\]

\[
\left[ \left\{ \int G_s H_{a,i} dV \right\} v_{i\alpha} = 0 \right.
\]

Finally, the finite element matrix equation takes the form of;

\[
\begin{bmatrix}
M_v & 0 \\
0 & 0
\end{bmatrix}
\begin{bmatrix}
v \\
p
\end{bmatrix}
+ \begin{bmatrix}
K_{vv} + K_{\nu \gamma} & K_{vp} \\
K_{pv} & 0
\end{bmatrix}
\begin{bmatrix}
v \\
p
\end{bmatrix}
= \begin{bmatrix}
R_n + R_s \\
0
\end{bmatrix}
\]

where, the mass, convective, viscosity, pressure gradient, volume force and surface force terms are, respectively;

\[
(M_v)_{i\alpha\beta} = \rho \int_V H_a H_{i\beta} dV = \rho \int_V H^T H dV
\]

\[
(K_{vv})_{i\alpha\beta} = \rho \int_V H_a H_{i\beta} H_{\gamma i} dV = \rho \int_V H^T \left( H v_i H_{x_i} + H v_2 H_{x_2} + H v_3 H_{x_3} \right) dV
\]

\[
(K_{\mu \nu})_{i\alpha\beta} = \int_V \mu H_a H_{i\beta} dV = \int_V \left( H^T H_{i\beta_1} + H^T H_{i\beta_2} + H^T H_{i\beta_3} \right) dV
\]

\[
(K_{wp})_{i\alpha\beta} = -\int V H_a G_s dV = -\int V H^T G dV
\]

\[
(R_n)_{i\alpha} = \int V H_a f_\beta dV = \int V H^T f^\beta dV
\]

\[
(R_s)_{i\alpha} = \int S H_a (-p n + \nu \cdot n) dS = \int S H^T (-p n + \nu \cdot n) dS
\]

Next, in order to implement the incremental-iterative procedure, the velocity and pressure at the end of each time step is defined, respectively;

\[
\begin{align*}
\text{\textsuperscript{\textit{m+1}\Delta\text{t}}} v_{i\alpha} &= \text{\textsuperscript{\textit{m\Delta\text{t}}} v_{i\alpha}^{(m)}} + \Delta \text{\textsuperscript{\textit{m\Delta\text{t}}} v_{i\alpha}^{(m)}} \\
\text{\textsuperscript{\textit{m+1}\Delta\text{t}}} p_s &= \text{\textsuperscript{\textit{m\Delta\text{t}}} p_s^{(m)}} + \Delta \text{\textsuperscript{\textit{m\Delta\text{t}}} p_s^{(m)}}
\end{align*}
\]

where \((m)\) is iteration. Next, by substituting equations (17) and (18) into equations (13) and (14) we obtain the incremental-iterative equations as;

\[
\begin{align*}
\rho \int_{V} \left[ H_{\alpha} \dot{H}_{\beta} dV \right] (\Delta_{s})^{(1)} + \rho \left[ H_{\alpha} \dot{H}_{\beta} \right] (\Delta_{s})^{(1)} + \rho \left[ H_{\alpha} \dot{H}_{\beta} \right] (\Delta_{s})^{(2)} + \\
\int_{\Gamma_{v}} \left[ H_{\alpha} G_{\beta} dV \right] (\Delta_{s})^{(2)} = \left[ \int_{\Gamma_{v}} \left( \dot{H}_{\alpha} \right) \right] dV + \int_{S} \left( -p \eta + v \right) dS
\end{align*}
\]

\[
\frac{1}{\Delta t} \int_{V} \left[ H_{\alpha} G_{\beta} dV \right] (\Delta_{s})^{(1)} - \rho \left[ H_{\alpha} \dot{H}_{\beta} \right] (\Delta_{s})^{(1)} - \left[ \int_{\Gamma_{v}} \left( \dot{H}_{\alpha} \right) \right] dV + \int_{S} \left( -p \eta + v \right) dS
\]

\[
\frac{1}{\Delta t} \int_{V} \left[ H_{\alpha} \dot{H}_{\beta} dV \right] (\Delta_{s})^{(2)} - \rho \left[ H_{\alpha} \dot{H}_{\beta} \right] (\Delta_{s})^{(2)} - \left[ \int_{\Gamma_{v}} \left( \dot{H}_{\alpha} \right) \right] dV + \int_{S} \left( -p \eta + v \right) dS
\]

\[
\left[ \begin{array}{c}
\Delta_{v}^{(1)} \\
\Delta_{p}^{(1)} \\
\Delta_{p}^{(2)} \\
\end{array} \right] = \left( \begin{array}{c}
\frac{1}{\Delta t} M_{v} + t + \Delta \mu K_{wv} v_{y}^{(m-1)} + t + \Delta \mu J_{wv} - K_{vp} \\
0 \\
0 \\
\end{array} \right) \left( \begin{array}{c}
\Delta_{v}^{(1)} \\
\Delta_{p}^{(1)} \\
\Delta_{p}^{(2)} \\
\end{array} \right) = \left( \begin{array}{c}
\frac{1}{\Delta t} F_{v}^{(m-1)} \\
0 \\
0 \\
\end{array} \right)
\]

where the matrices and vectors are:

\[
(M_{v})_{i,j} = \rho \int_{V} H_{\alpha} H_{\beta} dV = \rho \int_{V} H^{T} H dV
\]

\[
K_{vp}^{(m-1)} = \int_{V} \left[ H_{\alpha} \dot{H}_{\beta} \right] (\Delta_{s})^{(1)} - \rho \left[ H_{\alpha} \dot{H}_{\beta} \right] (\Delta_{s})^{(1)} - \left[ \int_{\Gamma_{v}} \left( \dot{H}_{\alpha} \right) \right] dV + \int_{S} \left( -p \eta + v \right) dS
\]

\[
J_{vp}^{(m-1)} = \int_{V} \mu H_{\alpha,j} H_{\beta,j} dV = \int_{V} \mu \nabla H^{T} \nabla dV
\]

\[
K_{vp}^{(m-1)} = \int_{V} \left[ H_{\alpha} \dot{H}_{\beta} \right] (\Delta_{s})^{(2)} - \rho \left[ H_{\alpha} \dot{H}_{\beta} \right] (\Delta_{s})^{(2)} - \left[ \int_{\Gamma_{v}} \left( \dot{H}_{\alpha} \right) \right] dV + \int_{S} \left( -p \eta + v \right) dS
\]

\[
F_{v}^{(m-1)} = F_{v}^{(m-1)} R_{R} + t + \Delta \mu R_{S}^{(m-1)} - t + \Delta \mu K_{wv} v_{y}^{(m-1)} + t + \Delta \mu J_{wv} - K_{vp}^{(m-1)} - F_{v}^{(m-1)}
\]

\[
F_{p}^{(m-1)} = -K_{vp}^{(m-1)} v_{y}^{(m-1)}
\]

\[
R_{R}^{(m-1)} = \int_{V} H^{T} f^{R} dV = \int_{V} H^{T} f^{R} dV
\]

\[
R_{S}^{(m-1)} = \int_{S} H \left( -p^{(m-1)} n + \nabla v^{(m-1)} \cdot n \right) dS
\]

The left upper index \( t + \Delta t \) denotes that the quantities are evaluated at the end of time step. \( V \) and \( S \) are the volume and the surface of the finite element, respectively. The matrix \( H \) and \( \nabla \) contains the interpolation functions for the velocities and the pressure, respectively. The matrix \( M_{v} \) is mass matrix, \( K_{wv} \) and \( J_{wv} \) are convective matrices, \( \mu \) is viscous matrix, \( K_{vp} \) is pressure matrix and \( F_{v} \) and \( F_{p} \) are forcing vectors. \( R_{R} \) and \( R_{S} \) are volume and surface forces, respectively.
3. Results and discussion

In order to assess the accuracy of the finite element methods employed, the pulsatile flow in a typical cylindrical artery is computed and compared with the well known analytical Womersley solution [16,17]. An idealized long, straight, rigid-walled cylindrical artery with length $L$, and radius $r$, is subjected to an inflow velocity that was uniform in space and periodic in time. The time variation is described by a sinusoidal function $V(t) = \overrightarrow{V}(1 + \sin(2\pi(t/T)))$ with mean velocity, $\overrightarrow{V} = 135 \text{mm/s}$ and period, $T = 0.2s$ as shown in Figure 3.

A sufficient distance from the inlet, the radial and circumferential components of velocity and pressure vanish. The axial velocity becomes a function of radius only and the pressure varies linearly with axial position. Figures 4 and 5 illustrate the numerically computed axial velocities against the well known analytical solution and the velocity profiles at 4 different phases within one cardiac cycle for the flow in a long, straight, cylindrical tube i.e. $t/T = 0.125$, $t/T = 0.375$, $t/T = 0.625$ and $t/T = 0.875$, respectively. In these figures and thereafter, the blood flow direction is referred to the arrow and the various phases of the pulsatile flow are referred to the flow waveform icon within each figure. The maximum error observed between the analytical and numerical results is below 15%. Further improvements can be made by adopting a much finer mesh.
Next, as a benchmark problem for flow simulation in abdominal aortic aneurysm models, we characterized the flow in a healthy infrarenal aorta. The infrarenal aorta is idealized as a straight tube of diameter $d_{AA}=18\text{mm}$. The model is supplied with a physiologically realistic pulsatile inflow waveform measured in a male subject at rest, as shown in Figure 1. The flow evolution was studied at six different phases of the pulsatile flow: (i) acceleration to systole, $t/T=0.035$ (ii) peak systole,
During flow acceleration, the flow develops into the characteristic top-hat velocity profile. When the Womersley number is small, viscous forces dominate and the velocity profiles are parabolic in shape. However, for Womersley number above 10, which is the case in abdominal aorta, the unsteady inertial forces dominate, and the flow is nearly top-hat with thin boundary layers. At the peak systole, the thickness of the boundary layer scales as $d_{at}/\alpha$. After the peak systole, that is, during flow deceleration, the flow slows down along the walls and quickly reverses, while the central region bulk of the fluid in the healthy aorta moves forward with a blunt velocity profile. At peak retrograde flow, this central region bulk of fluid only reverses fully. As the flow comes out from the retrograde flow region into the early diastolic flow region, the net flow decelerates back to zero and ends up moving forward again. At late diastole, the flow relaxes to near rest before being accelerated again at the beginning of the next cardiac cycle. It is important to point out that although the flow develops an inflexional velocity profile during diastole, it remains entirely laminar during the whole cardiac cycle. The axial velocity distributions at each key step along the pulsatile flow are as illustrated in Figure 8.

This boundary layer of reversed flow close to the wall is an important characteristic of pulsatile flow. This exhibits that even for a straight section of an artery with positive volume flow, there is always some time within the cardiac cycle where the viscous traction forces are opposite the dominant flow direction. This has implications in the design of in vitro devices designed to replicate the shear forces on the inner wall of the artery. Namely, in vivo, the luminal surface is exposed to shear forces which reverse in direction for some portion of the cardiac cycle.
Figure 6. 3D Velocity flow contours in a healthy aorta under physiologically realistic pulsatile flow at six phases of the pulsatile flow cycle: (i) flow acceleration, $t/T=0.035$; (ii) peak systole, $t/T=0.16$; (iii) flow deceleration, $t/T=0.35$; (iv) peak retrograde flow, $t/T=0.55$; (v) early diastole, $t/T=0.77$; (vi) late diastole, $t/T=0.87$.
(i) $t/T = 0.035$

(ii) $t/T = 0.16$

(iii) $t/T = 0.35$

(iv) $t/T = 0.55$

Figure 7. 3D Velocity vector profiles in a healthy aorta under physiologically realistic pulsatile flow at six phases of the pulsatile flow cycle: (i) flow acceleration, t/T=0.035; (ii) peak systole, t/T=0.16; (iii) flow deceleration, t/T=0.35; (iv) peak retrograde flow, t/T=0.55; (v) early diastole, t/T=0.77; (vi) late diastole, t/T=0.87

Figure 8. Axial velocity distribution of pulsatile blood flow in a healthy aorta at six different phases within one complete pulsatile flow cycle

4. Conclusion

A complete set of three-dimensional mixed velocity-pressure (v-p) finite element method was derived to solve the pulsatile nature of blood flow in a rigid-walled healthy aorta. The numerical scheme exploits the Galerkin method and the implicit incremental-iterative procedure.
The presented computational technique was used to analyze the flow dynamics in a healthy abdominal aorta under a physiologically realistic pulsatile flow at rest that can be considered as a benchmark problem. It was observed that there exists a boundary layer of reversed flow close to the wall that is considered an important characteristic of pulsatile flow. This exhibits that even for a straight section of an artery with positive volume flow, there is always some time within the cardiac cycle where the viscous traction forces are opposite the dominant flow direction. This has implications in the design of in vitro devices designed to replicate the shear forces on the inner wall of the artery. Namely, in vivo, the luminal surface is exposed to shear forces which reverse in direction for some portion of the cardiac cycle. This leads to the ability to further co-relate flow dynamics in an aneurismal aorta and subsequently allow biomedical engineers and physicians to diagnose and design patient-specific treatment plans that can improve their care. Having said this, clinical application of numerical modelling and computer-aided surgical planning is the key for the future of medicine.

**Nomenclature**

<table>
<thead>
<tr>
<th>Symbol</th>
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<tr>
<td>$\rho$</td>
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<tr>
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<td>$f$</td>
<td>Body force</td>
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<td>Velocity</td>
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**References**


