A boundary layer model for wall shear stress in arterial stenosis

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Abstract. This paper proposes a model for wall shear stress in arterial stenosis based on boundary layer theory. Wall shear stress estimates are obtained by solving the momentum integral equation using the method proposed by Walz and applying this method to various stenosis geometries for Reynolds numbers (Re) of Re = 59–1000. Elevated wall shear stress may be of importance when considering thrombosis and vascular erosion in stenosis, as well as the potential for debris from the stenotic area to ‘break away’ and cause further pathology. The values of shear stress obtained using the model in this study agree well with published values of wall shear stress. When compared to a previously published boundary layer model utilizing the Thwaites method (Reese and Thompson, 1998), the model proposed herein performs better at higher Re while the model utilizing the Thwaites method performs better at lower Re. Wall shear stresses are shown to increase with increasing stenosis (increased area reduction) for a given stenosis length, increase with increasing Re for a given stenosis geometry, and increase for steeper stenosis of the same constriction. The boundary layer model proposed can be easily implemented by clinical researchers to provide in vivo estimates of wall shear stress through arterial stenoses.

Keywords: Wall shear stress, boundary layer theory, arterial stenosis

1. Introduction

Arterial stenosis, localized narrowing of the artery, is often the result of vascular disease. This atherosclerosis alters the flow pattern of blood, which can induce further pathology [26]. Wall shear stresses on the vessel wall are created by the velocity difference between flowing blood and the stationary artery wall. In an arterial constriction blood velocity increases due to the conservation of mass, producing increased wall shear stress in the region of blood acceleration ([2,9,14,18,29,37,38] and others). Increased wall shear stress near the throat of the stenosis can aid in plaque cap rupture resulting in thrombosis (a blood clotting reaction of the coagulation cascade) [7,17,36]. Wootton et al. [36] examined thrombogenic stenoses experimentally and computationally. Their data and analysis revealed the tallest thrombi were present in the stenosis throat and in the converging region of the stenosis. The converging region and the stenosis throat are known to be regions of elevated shear stresses. Both high and low shear stresses have been proposed as mechanisms for stenosis formation, however, shear stress variation

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in space and time appear to be more important than magnitude [3]. In addition, according to Berger and Jou [3], atherosclerotic disease may not be solely a function of the stenosis but a function of the tendency for parts of the thrombus or plaque to break off the vessel wall and occlude smaller vessels downstream. Therefore, it appears that high and low shear stress each has a role in vascular disease. Since, many features of fluid mechanics (i.e., shear stress, turbulence, head loss, choked flow in collapsing tubes) [6,17,31] play a role in the generation, detection, and treatment of arterial disease [17], fluid mechanics models which describe blood flow behavior and the fluid–vessel interaction in the vascular system are of importance.

Several studies have investigated the flow of blood through constricted tubes. Since, wall shear stresses are difficult to determine experimentally [21] and are only accurate to 20–50% [17], researchers have relied heavily on analytical and computational models to calculate shear stress in arterial stenosis, i.e., [2,14,16,18,20,24,25,29]. Lee and Fung [18] solved the Navier–Stokes (NS) equations for steady flow through arterial stenosis numerically, but were only able to solve these equations for Reynolds numbers (Re) 0–25 due to numerical instability. Morgan and Young [25] applied momentum and internal-energy equations in integral form to obtain approximate solutions for velocity distribution, pressure drop, wall shear stress, and separation in axisymmetric constrictions. Boundary layer theory for laminar steady flow through a conical constriction was used to estimate wall shear stress using a local similarity method [2]. Wall shear stress estimates agreed fairly well with the magnitude of shear stress levels obtained from solving the NS equations for steady and pulsatile flow, but underestimate the wall shear stress. Boundary layer theory utilizing the Thwaites method [32] to obtain approximate solutions for wall shear stresses was employed by Reese and Thompson [29] and compared to published data by Back and Crawford [2] and Huang et al. [14]. Huang et al. [14] applied a finite difference scheme to calculate wall shear stress for an incompressible Newtonian fluid through an axisymmetric smooth rigid tube in five stenosis geometries. In addition, Huang et al. [14] examined steady flow in stenoses experimentally and reported good agreement with model data. The results of the model proposed herein will be compared to results obtained by Back and Crawford [2], Huang et al. [14], and Reese and Thompson [29].

Results obtained using computational fluid dynamics (CFD) are often considered to be highly accurate. However, implementation of CFD models for every particular geometry can require extensive time and resources. For this reason analytical models are a reasonable alternative to CFD if they obtain results within acceptable limits of error. Large variations in physiology, anatomy and stenosis parameters are seen among patients, therefore, extremely accurate values of shear stress would require clinical analysis of the patient’s blood, complex imaging techniques, and intensive computational models. Thus, analytical models, which can be easily implemented, could provide researchers and clinical engineers with a tool for an initial estimation of wall shear stresses and aid in identifying when these values are reaching potentially problematic levels. The purposes of this paper are (1) to study the hypothesis that wall shear stress values obtained from boundary layer models will improve when solved with a velocity profile family more suited to wedge type flows, and (2) to present a model for obtaining wall shear stresses in arterial stenosis within reasonable error which could easily be implemented by clinical researchers.

2. Approximate method

2.1. Basic assumptions

The stenosis geometry used in this study is a smooth, rigid, axisymmetric conical constriction in a tube (Fig. 1). This geometry is similar to the geometry employed in previous analytical models.
Fig. 1. Diagram of the assumed stenosis geometry used in this study. Radius $r_1$ is associated with the $x_1$ (entrance to stenosis) location, $r_2$ is associated with the $x_2$ (stenosis throat) location, and the hatch marks indicate that $x_0$ is assumed to be well upstream.

[2,14,20,24,25,29]. The above assumed geometry is commonly employed for arterial stenosis [16] and is reasonable since the thickened intima at the stenosis reduces vessel flexibility making it effectively rigid [2]. Blood is considered an incompressible Newtonian fluid. Although blood behaves in a non-Newtonian fashion at low shear rates it can be treated as an incompressible Newtonian fluid for the shear rates encountered in medium and larger arteries [11]. Physiologically blood flow is pulsatile, however to make the mathematical problem more manageable steady flow is assumed. As discussed by Misra and Kar [24], assuming steady flow may not be valid in general but is meaningful in certain situations, such as, arteries where the Womersley number [35] is sufficiently low [23]. In addition, quasi-steady flow can exist in some larger arteries due to acquired constrictions [4,22]. Since this model is investigating maximum shear stresses, which are expected to occur at the maximum fluid velocity [29] and some vessels have a reasonably low Womersley number, it appears a reasonable assumption. Blood is known to be laminar except in areas of the heart or under pathological conditions (i.e., recirculation zones distal to the stenosis throat [1,12]). The entrance flow is assumed to be laminar flow [1] in this model and the Reynolds numbers examined range from 59–1000, which is reasonable for medium vessels where $Re$ is typically on the order of 100–1000 [17]. Fluid flow is considered to have started from a stagnation point upstream of the stenosis and become fully developed at the entrance of the stenosis [1]. This assumption has been applied to boundary layer models for blood flow [15,20,29].

2.2. Boundary layer model

In flows where a frictional layer forms at the wall, due to velocity gradient between the fluid and the wall, boundary layer equations apply. The model proposed herein examines an approximate solution to the Von Karman momentum integral equation for incompressible flow (derived in Panton [27], Schlichting and Gersten [30], and others)

$$\frac{d}{dx}(U^2\theta) + \delta^* U \frac{dU}{dx} = \frac{\tau_w}{\rho},$$

(1)

In Eq. (1), $\theta$ and $\delta^*$ are respectively the momentum and displacement thickness, which are defined by

$$\theta = \int_0^\delta \frac{u}{U} \left(1 - \frac{u}{U}\right) dy,$$

(2)

$$\delta^* = \int_0^\delta \left(1 - \frac{u}{U}\right) dy.$$

(3)
In Eqs (2) and (3), $\delta$ is the boundary layer thickness, $u$ is the velocity in the boundary layer, and $U$ is the outer flow velocity.

Walz [33] proposed an approximate method for solving the momentum integral equation (Eq. (1)). This method is well described in Schlichting and Gersten [30]. Although this method was first proposed by Alfred Walz in 1966 [33] the Walz 1969 English translation [34] was examined for this study and is slightly changed from the original German text. Walz [34] assumed a single parameter velocity profile corresponding locally (local similarity) to the Hartree [13] velocity profiles, which are solutions to the single parameter Falkner–Skan equation [30]. In solving Eq. (1) Walz [34] introduced two functions as independent variables,

$$Z(x) = \frac{\theta^2 U}{\nu}, \quad (4)$$

$$\Gamma(x) = \frac{-\theta^2 \left( \frac{\partial^2 u}{\partial y^2} \right)_w}{U}, \quad (5)$$

where $Z(x)$ is the thickness parameter, $\nu$ is the kinematic viscosity, $U$ is the free stream velocity, $x$ is the axial distance, and $\Gamma(x)$ is the shape parameter defined as the nondimensional second derivative of the velocity profile at the wall. By applying wall compatibility conditions for the Prandtl boundary layer equation [30] and substituting Eq. (4) into Eq. (5), gamma (Eq. (5)) becomes,

$$\Gamma = \frac{Z \, dU}{U \, dx}, \quad (6)$$

Using Eqs (4) and (6), Eq. (1) can now be transformed into,

$$\frac{dZ}{dx} + (3 + 2H_{12}) \frac{Z \, dU}{U \, dx} = F_1(\Gamma), \quad (7)$$

where

$$H_{12}(\Gamma) = \frac{\delta^*}{\theta}, \quad (8)$$

$$F_1(\Gamma) = \frac{2\theta \tau_w}{\mu U}. \quad (9)$$

The symbols $\mu$ and $\tau_w$ represent the dynamic viscosity and wall shear stress, respectively. Functions $H_{12}$ and $F_1$ for the Hartree profiles [13] are tabulated as functions of gamma ($\Gamma$) and can be found in table form in Walz [34] and Gersten and Schlichting [30]. Hence, once gamma is known, wall shear stress can be obtained. At this point it should be noted that $H_{12}$ is not necessary in the initial estimation of shear stress, however, it is necessary to calculate the displacement thickness and increases the accuracy of calculated shear stress values as described below.

In order to solve for gamma to obtain the shear stress, the parameter $Z$ must be determined. To solve for $Z$, Eq. (7) can be expressed as an ordinary differential equation. This is acceptable since $dZ/dx$ is
nearly linear for wedge flow and can be approximated by the linear function \( \frac{dZ}{dx} = a - b\Gamma \) [30]. Hence, Eq. (7) can be expressed as an ordinary differential equation of the form

\[
\frac{dZ}{dx} + bZ \frac{dU}{dx} - a = 0,
\]

where, \( a = 0.441 \) and \( b = 4.165 \) for \( \Gamma > 0 \) (accelerating flow) and \( a = 0.441 \) and \( b = 5.165 \) for \( \Gamma < 0 \) (retarded flow). Accelerating flow parameters are used in this study. Solving Eq. (10) for \( Z \) from the upstream stagnation point \( (x = 0) \) to the stenosis throat \( (x = x_2) \) yields,

\[
Z(x) = \frac{a}{U(x)^b} \int_0^{x_2} U(x)^b \, dx.
\]

Applying additivity, Eq. (11) can be separated into two parts and simplified [29,30]. The first integral considers the stagnation point \( (x_0) \) to the stenosis inlet \( (x = x_1) \) and a second integral considers the stenosis inlet \( (x = x_1) \) to the stenosis throat \( (x = x_2) \),

\[
Z(x) = \frac{a}{U(x)^b} \left[ \int_0^{x_1} U(x)^b \, dx + \int_{x_1}^{x_2} U(x)^b \, dx \right].
\]

The first integral (upstream to stenosis inlet) can be solved for Poiseuille flow in a pipe using Eq. (2) for the momentum thickness. Poiseuille flow has been shown experimentally [1,19] and analytically [14] to be a reasonable assumption for entry flow. Hence, Eq. (12) can be simplified using the exact upstream momentum thickness for parabolic Poiseuille flow, \( \theta_1 = 0.133 \) [8]. Using \( \theta_1 \) and Eq. (4), the first integral then becomes,

\[
\frac{\theta_1^2 U_1^{b+1}}{a\nu} = \int_0^{x_1} U(x)^b \, dx.
\]

Applying Eqs (4) and (13), Eq. (12) becomes,

\[
\theta_2^2 = \theta_1^2 \left( \frac{U_1}{U_2} \right)^{b+1} + \frac{a\nu}{U(x)^{b+1}} \int_{x_1}^{x_2} U(x)^b \, dx.
\]

Equation (14) can be solved numerically for an arbitrary free stream flow \( U(x) \). In this application the free stream velocity will be taken as the maximum velocity varying linearly through the stenosis. This assumption has been shown to be reasonable for boundary layer solutions of this type [29]. The peak velocity at position 1 is twice the mean velocity for Poiseuille flow. Based on reported Reynolds number the maximum velocity \( U_1 \) becomes,

\[
U_1 = \frac{Re \nu}{r_1},
\]
Table 1

<table>
<thead>
<tr>
<th></th>
<th>M1</th>
<th>M2</th>
<th>M3</th>
<th>M4</th>
<th>M5</th>
<th>M6</th>
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<tbody>
<tr>
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<td>1/4</td>
<td>1/3</td>
<td>1/2</td>
<td>1/2</td>
<td>1/2</td>
<td>3/10</td>
</tr>
<tr>
<td>$L$</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>8</td>
<td>2</td>
<td>14</td>
</tr>
<tr>
<td>Area reduction (%)</td>
<td>44</td>
<td>56</td>
<td>75</td>
<td>75</td>
<td>75</td>
<td>51</td>
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</table>

$R$ is the radial stenosis height and $L$ is the stenosis length in units of $r_1$. M1–M5 are geometries studied by Huang et al. [14]. M6 is the geometry examined in Back and Crawford [2]. Note, Back and Crawford [2] did not designate this geometry M6. This geometry is identified as M6 in this study for continuity with the designation given in Reese and Thompson [29].

but more closely resembles a plug like profile as shown numerically [14] and experimentally [1,14]. For this reason the peak velocity at the throat is calculated by the mean velocity,

$$U_2 = \frac{U_1 r_1^2}{2 r_2^2}. \quad (16)$$

The linearly varying velocity distribution through the stenosis is

$$U(x) = U_1 + \frac{U_2 - U_1}{x_2 - x_1} (x - x_1). \quad (17)$$

At this point $\theta$ can be solved numerically, allowing $Z$ and hence $\Gamma$ to be solved. Once $\Gamma$ is known, values of $F_1$ and $H_{12}$ can be determined. $F_1$ and $H_{12}$ can be plotted as functions of $\Gamma$ from table values given in Schlichting and Gersten [30]. For this model $F_1$ and $H_{12}$ were plotted for the positive values of $\Gamma$ present in this problem and curve fit ($R^2 > 0.98$). An initial value of wall shear stress ($\tau_w$) can now be determined.

Reese and Thompson [29] showed that the estimate of $U_2$ (and hence shear) can be significantly improved if the displacement thickness is subtracted from the radius at the stenosis throat to more accurately predict the mean velocity as the peak velocity. This is valid since the flow at the stenosis is not a perfect plug flow. To implement the above correction, the displacement thickness calculated from Eq. (8) is subtracted from the actual throat radius to give a new effective throat radius ($r_2^1 + \delta^* = r_2^2 - \delta^*$) and $\theta, \Gamma$, and $\tau_w$ (wall shear) are re-determined. This process is repeated until there is no change in the effective radius (no change was assumed when the difference is within 1E-6). As stated by Reese and Thompson [29], for moderate stenosis under-relaxation is required when iterating the effective radius so that the full displacement thickness is not initially implemented. This prevents values larger than the throat radius. Under-relaxation was required when this model was applied to geometry M1 (described below). This model was solved using Engineering Equation Solver (EES) (http://www.fChart.com) and Microsoft Excel® for stenosis geometries reported in the literature (Table 1) and compared to reported results.

In summary, the model is implemented for specified geometry (stenosis height and length), physiologic parameters, and constants $a$ and $b$ for accelerating flow. The Reynolds number is input resulting in $U_1$, which leads to $U_2$ and $U(x)$ for the given geometry. Momentum thickness (Eq. (14)) in the stenosis is then output for the calculated velocities. From the momentum thickness ($\theta$) the thickness parameter ($Z$) and shape parameter ($\Gamma$) can be found, allowing $F_1$ and $H_{12}$ to be attained. The shear stress is now
Table 2

Peak wall shear stress (dyn/cm$^2$) in the arterial stenosis geometries given in Table 1

<table>
<thead>
<tr>
<th>$Re$</th>
<th>M1</th>
<th>M2</th>
<th>M3</th>
<th>M4</th>
<th>M5</th>
<th>M6</th>
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<td>59</td>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>B (19.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>R (26.2)</td>
</tr>
<tr>
<td>83</td>
<td></td>
<td></td>
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<td>37.4</td>
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<td></td>
<td></td>
<td></td>
<td>B (28.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>R (37.4)</td>
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<td>100</td>
<td>8.4</td>
<td>12.8</td>
<td>33.3</td>
<td>30.8</td>
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</tr>
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<td></td>
<td>H (8.3)</td>
<td>H (13.2)</td>
<td>H (36.7)</td>
<td>H (28.6)</td>
<td>H (49.9)</td>
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</tr>
<tr>
<td></td>
<td>R (8.9)</td>
<td>R (13.1)</td>
<td>R (33.2)</td>
<td>R (28.5)</td>
<td>R (40.2)</td>
<td></td>
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<tr>
<td>207</td>
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<td></td>
<td></td>
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<td>B (90.0)</td>
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<td>R (102.7)</td>
</tr>
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<tr>
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<td>H (67.1)</td>
<td>H (110.6)</td>
<td>H (316.9)</td>
<td>H (240.3)</td>
<td>H (423.2)</td>
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<tr>
<td></td>
<td>R (73.3)</td>
<td>R (105.8)</td>
<td>R (269.6)</td>
<td>R (215.2)</td>
<td>R (345.1)</td>
<td></td>
</tr>
<tr>
<td>1000</td>
<td>201.9</td>
<td>274.7</td>
<td>757.7</td>
<td>552.2</td>
<td>1021.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>H (168.3)</td>
<td>H (279.2)</td>
<td>H (788.1)</td>
<td>H (607.7)</td>
<td>H (1022.7)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>R (201.7)</td>
<td>R (285.2)</td>
<td>R (690.2)</td>
<td>R (539.1)</td>
<td>R (910.2)</td>
<td></td>
</tr>
</tbody>
</table>

Values in bold are the stresses determined with the model given in this study. Values in parenthesis are previously published values by (H) Huang et al. [14] using CFD, (B) Back and Crawford [2], using an analytical model, and (R) Reese and Thompson [29], using a boundary layer model.

Output from Eq. (9) and the displacement thickness (for the iteration steps) is output from Eq. (8). Iteration steps are performed until the effective radius shows no significant change, and a final value of shear stress is obtained.

2.3. Physiologic parameters

In order to compare the results of this model to previously reported values of shear stress, the same physiologic parameters as those used in the comparing study where applied. The physiologic parameters used by Huang et al. [14] for models M1–M5 (Table 1) are $r_1 = 3.5$ mm, $\rho = 1060$ kg/m$^3$, $\mu = 3.71 \times 10^{-3}$ N s/m$^2$, and $Re = 100, 500, 1000$. Back and Crawford [2] used parameters $r_1 = 1.54$ mm, $\rho = 1050$ kg/m$^3$, $\mu = 3.68 \times 10^{-3}$ N s/m$^2$, and $Re = 59, 83, 207, 353$ for model geometry M6 (Table 1). Reese and Thompson [29] compared the results of their model to reported values of Back and Crawford [2] and Huang et al. [14]. Therefore, Reese and Thompson [29], used the parameters of each study for the respective geometries.
3. Results

The boundary layer model presented in this study was solved numerically for $Re$ and stenosis geometries previously reported in the literature. Values of shear stress are shown in Table 2. Results produced by this model demonstrate that wall shear stresses increase with increasing percent stenosis for a given stenosis length, increase with increasing $Re$ for a given stenosis geometry, and increase for steeper stenosis of the same constriction (Table 2). These trends agree with previously published results by Back and Crawford [2], Huang et al. [14], Morgan and Young [25], and Reese and Thompson [29]. Shear stresses reached a maximum immediately preceding the throat, which agrees with previously reported results [2, 14, 20, 25, 29]. This result is expected due to the idealized stenosis geometry used in this model. The maximum shear stress must occur where the velocity gradient is finite. At the throat the derivative of the velocity is undefined and therefore the maximum shear stress occurs immediately preceding the throat.

To provide an indication of how well the model works, values of shear stress are compared to previously published values obtained computationally by Huang et al. [14] (Fig. 2), analytically by Back and Crawford [2] (Fig. 2) and Reese and Thompson [29] (Fig. 3) by examining the ratio of shear stress determined in this study to published values. When comparing the data to the data of Huang et al. [14], the maximum and minimum ratios are 1.2 and 0.81 with a mean value of 0.96 ± 0.1 (mean ± s.d.). With the exception of M1, the ratio is closer to unity at higher $Re$ (Fig. 2). In addition, the model has an overall average ratio for all three $Re$ closer to unity for geometry M4 (0.97) than M2, M3 and M5. When comparing the data to the data of Back and Crawford [2], the maximum and minimum ratios are 1.35 and 1.02 with a mean value of 1.22 ± 0.16 (mean ± s.d.). Again these values occur at low $Re$, as $Re$ increases the ratio approaches unity (Fig. 2). The data agreed well with the data of Reese and Thompson [29]. The maximum and minimum ratios are 1.12 and 0.88 with a mean value of 1.01 ± 0.07 (Fig. 3).
4. Discussion

The values of shear stress obtained using a model based on boundary layer theory are in good agreement with previously reported values of shear stress. To determine the percent difference between CFD results by Huang et al. [14] and the results of this study, the equation \(100 \times \frac{\tau_{\text{CFD}} - \tau}{\tau_{\text{CFD}}}\) was applied. In this equation \(\tau\) is the value of shear stress obtained in this work and \(\tau_{\text{CFD}}\) is the value obtained by Huang et al. [14] using CFD. From Table 3 it can be seen that the minimum and maximum differences are 0.1% and 19.9%, respectively. In addition to examining the percent difference between results of this study and reported CFD values, results of this study are compared to the results of Back and Crawford [2] (geometry M6). Differences were evaluated using the percent difference equation discussed above \([100 \times \frac{\tau_{\text{B}} - \tau}{\tau_{\text{B}}}\]). The percent differences are 35.3, 33.6, 15.1, and 2.9% for \(Re = 59, 83, 207,\) and 353, respectively. Back and Crawford [2] state the accuracy of their analytical model to be approximately 15% and to underestimate the shear stress values, therefore the results of this study agree well with data from Back and Crawford [2]. Although the values of shear stress obtained in this study do not exactly match CFD values, which are assumed to be highly accurate, they agree within reason and could provide a tool for estimating shear stress in arterial stenosis. Due to variations in physiology, anatomy and stenosis parameters in different patients, extremely accurate values of shear stress would require clinical analysis of the patient’s blood, complex imaging techniques, and intensive computational models. For this reason analytical models such as the model proposed in this study that estimate wall shear stresses in arterial stenosis with reasonable error that can be implemented with minimal model inputs and computing hardware could provide researchers with an initial diagnostic tool.

The percent difference between the shear stress values obtained using the model proposed by Reese and Thompson [29] using the Thwaites [32] method to solve the momentum integral equation and CFD
Table 3
Percent difference between the values obtained using model described in this work and results published in Huang et al. [14]

<table>
<thead>
<tr>
<th>Re</th>
<th>M1</th>
<th>M2</th>
<th>M3</th>
<th>M4</th>
<th>M5</th>
</tr>
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<tbody>
<tr>
<td>100</td>
<td>1.2</td>
<td>3.0</td>
<td>9.3</td>
<td>7.7</td>
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<td>500</td>
<td>2.5</td>
<td>16.1</td>
<td>11.3</td>
<td>6.5</td>
<td>10.4</td>
</tr>
<tr>
<td>1000</td>
<td>19.9</td>
<td>1.6</td>
<td>3.9</td>
<td>9.1</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Table 4
Percent difference between the Thwaites method used in Reese and Thompson [29] and CFD results published in Huang et al. [14]

<table>
<thead>
<tr>
<th>Re</th>
<th>M1</th>
<th>M2</th>
<th>M3</th>
<th>M4</th>
<th>M5</th>
</tr>
</thead>
<tbody>
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<td>7.2</td>
<td>0.8</td>
<td>9.5</td>
<td>0.3</td>
<td>19.4</td>
</tr>
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<td>4.3</td>
<td>14.9</td>
<td>10.4</td>
<td>18.5</td>
</tr>
<tr>
<td>1000</td>
<td>19.8</td>
<td>2.2</td>
<td>12.4</td>
<td>11.3</td>
<td>11.0</td>
</tr>
</tbody>
</table>

results reported in Huang et al. [14] are calculated in the same fashion as above and shown in Table 4. From Table 4 it can be seen that the minimum and maximum differences are 0.3% and 19.8%, respectively. Comparison with the results of Back and Crawford [2] results in percent errors of 37.9, 33.6, 14.1, and 1.5% for Re = 59, 83, 207, and 353, respectively. Overall, values of shear stress obtained using the method herein produce results which agree more closely to values obtained using CFD than values obtained using are method based of Thwaites [32] method for solving the momentum integral equation (11 out of 15 shear stress values have smaller error). However, applying the Thwaites [32] method appears to result in slightly smaller differences at lower Reynolds number and weaker stenosis while applying the Walz [33] method appears more accurate at higher Reynolds number and larger stenosis (Tables 2–4). This may be due to the velocity profile used in each method. The parameters used by Thwaites [32] are obtained by taking an average of all the known experimental and analytical results available at the time the method was proposed, while parameters used by Walz [33] are obtained from the Hartree [13] profiles. Hartree [13] numerically solved the Falkner–Skan equation, which is suited to wedge type accelerating flow and are similar to the stenosis geometry used in this model. For this reason the parameters used in this study may be more suited for stenosis analysis, while a model using the method proposed by Thwaites [32] is more generally suited. Overall the model proposed herein seems superior at higher Reynolds number, whereas Reese and Thompson’s model performs better at lower Reynolds number. However, both models appear to perform adequately for the required purpose.

Examining values of shear stress obtained if Poiseuille flow is assumed through the entire stenosis demonstrates the need for more intensive analytical and computational models. Huang et al. [14] reported shear stress values ranging from approximately 2–20 dyn/cm² for Poiseuille flow through geometries M1–M5. This value drastically underestimates the shear stress in arterial stenosis and does not provide biologically critical values such as the approximately 400 dyn/cm² at which damage to vascular endothelium is believed to exist [9,10]. Since, non-Poiseuille like profile have been reported experimentally and numerically for flow through arterial stenosis, it is not unexpected that Poiseuille flow does not accurately predict shear stress in arterial stenosis.

The model proposed in this study utilizes several assumptions. Hence, the problem solved in this manuscript is less complex than the pathology existing in vivo. The principle assumptions introduced into the model are a rigid walled axisymmetric stenosis, blood as an incompressible Newtonian fluid, and steady flow. Under pathologic conditions the vessel wall undergoes intimal thickening resulting in an
effectively rigid vessel [2,17,20]. Blood behaves in a Newtonian fashion in medium and large arteries [11]. Yet, conditions do exist in vivo under which blood is non-Newtonian, however, Brookshier and Tarbell [5] demonstrated that non-Newtonian components do not significantly effect the magnitude wall shear stress. Hence, although vessel wall elasticity and non-Newtonian viscosity can be physiologically relevant in vivo, for many biologic flows these parameters may be considered as secondary in importance [17]. The third principle assumption, steady flow, presents a larger deviation from physiologic conditions. In many vessels the Womersley number (Wo; ratio of unsteady forces to viscous forces) can be sufficiently high to warrant unsteady flow analysis. However, in many vessels such as the femoral artery the Womersley number is sufficiently low (2.5 < Wo < 3.5) to allow steady flow analysis [23]. For Wo larger than 10 unsteady inertial forces dominate and the flow displays piston like behavior [17], at much lower Wo viscous forces dominate and velocity profiles are parabolic and the centerline velocity oscillates in phase with driving pressure [17,23,31,35]. A range of flow behavior occurs between low and high Womersley number, hence each vessel must be examined in a case by case basis to determine when the steady assumption is tolerable. As discussed by Lorthois et al. [20], error between assuming steady and unsteady behavior, determined by dimensional analysis [28], can range from 15 to 5% for 30 to 85% stenosis. Hence, it is not appropriate to make exact statements between the present results and complex conditions of pathologic vessels. However, the model presented in this paper reveals differences in boundary layer model behavior depending upon the chosen velocity profile and presents a model which can provide reasonable initial estimates of wall shear stress in stenosed vessels at low Womersley number.

5. Conclusions

In conclusion, this model gives reasonable estimates of wall shear stress in arterial constrictions. Elevated wall shear stress may be of importance when considering thrombosis and vascular erosion in stenosis, as well as the potential for debris from the stenotic area to ‘break away’ and cause further pathology. Results of this study agree well with published results regarding variations in shear stress with the amount of stenosis, length of stenosis, and Reynolds number. In addition, the model proposed herein performs better at higher Re while boundary layer models utilizing the Thwaites method perform better at lower Re. Estimates of shear stress may be of importance in thrombogenic stenosis and vascular damage. The model presented in this study provides reasonably accurate values of wall shear stress in stenosis using minimal time and resources and may be of use to clinical researchers for initial estimates of wall shear stress.

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References


