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Wall shear stress variations and unsteadiness of pulsatile blood-like flows in 90-degree bifurcations

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ABSTRACT

Complex and slow interaction of different mechanical and biochemical processes in hemodynamics is believed to govern atherogenesis. Over the last decades studies have shown that fluid mechanical factors such as the Wall Shear Stress (WSS) and WSS gradients can play an important role in the pathological changes of the endothelium. This study provides further indications that the effects of fluid mechanical aspects are correlated with the diseased regions of the larger arteries. Unsteady high temporal WSS gradients (TWSSG), a function of the shear-thinning property of the non-Newtonian viscosity, move with the separation bubble. Red Blood Cell (RBC) dilution due to the secondary flows determines the magnitudes of the WSS and TWSSG. The results indicate that the focal nature of the TWSSG may have implications on the response of the endothelium.

1. Introduction

Atherosclerosis is a common cardiovascular disease that develops rather slowly. It may lead to partial or complete occlusion of the coronary and possibly also to aortic arterial branches causing disruption of blood flow to important regions of the body. The formation or development of atherosclerosis is initially localized to regions of sharp curvature or bifurcations of the larger arteries. In these regions, the flow is characterized by flow separation, reversed flow and unsteadiness and has been strongly attributed to the development process of atherosclerosis over the last sixty years [7,11,19,23,35].

The flow behavior is believed to affect biological processes through increased residence times or biochemical release processes [2,4,8–10,27]. Direct mechanical forces on the endothelial layer, on the other hand, have been related to the dysfunction or damage to the cells [23]. In recent years, both processes have been studied through variations in wall shear stresses (WSS). One of the most common correlations to atheroma location is regions of low WSS, thought to be related to the modified transport of certain substances and cells into the arterial wall [9,10]. More recently, both experimental and numerical, steady and pulsatile flow studies have correlated time-averaged and oscillatory WSS to regions of reversed flow and sites prone to disease, i.e. locations of arterial plaque [3,4,25,27,52]. It is often mentioned that time-averaged and oscillatory WSS is believed to affect the mass transport of atherogenic materials in the lumen, increasing the residence times near the arterial walls [4,8,27]. Moreover, there are also suggestions from studies linking high WSSG to the disruption of the endothelial cell layer, causing dis-orientation or damage [18,22,38]. Intimal hyperplasia, believed to be an initiator to atherogenesis and a consequence of relevant WSSG components [17,26,51], is related to widening and stretching of endothelial cell junctions [31]. Thus, there is no definitive reason or process that can account for atherogenesis, other than being a complex interaction of many processes.

The aim of this study is to investigate temporal and spatial variations of the WSS for a non-Newtonian, blood like fluid, in regions where plaques are commonly found. The flow is affected by local viscosity variations due to variations in local RBC volume fraction in the region of the bifurcation. The computational results will be related to current hypotheses relating WSS variations to known possible risk sites in arterial bifurcations. Three different, RBC concentration dependent, non-Newtonian viscosity models are investigated, considering the effect of an increase in the peak flow rate and heart pulsation frequency rate. To the knowledge of the authors, this is the first numerical study of its kind considering coupling of the local RBC volume fraction variation affecting the viscosity of the blood flow in large arteries. This study is based upon a geometry used in a previous Newtonian flow study to which comparison can directly be made [21].

2. Theoretical background

2.1. Viscosity models

Blood is a multiphase mixture of water with molecules of widely different sizes and different cells. The average volume fraction of RBCs

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Nomenclature

α	The Womersley number (–)
a, β, α_C	Casson empirical constants (–)
$\dot{\gamma}_C$	Quemada critical shear rate (s^{-1})
$\dot{\gamma}$	shear rate magnitude (s^{-1})
$\tilde{H}, m, \delta$	RBC distribution equation constants (–)
μ	effective dynamic viscosity (Pa s)
μ_p	plasma dynamic viscosity (Pa s)
μ_w	water dynamic viscosity (Pa s)
ω	angular frequency (rad/s)
ϕ	sample property (–)
ρ	density (kg/m^3)
ρ_m	blood mixture density (kg/m^3)
ρ_p	blood plasma density (kg/m^3)
ρ_w	water density (kg/m^3)
τ	fluid shear stress (Pa)
τ_y	yield shear stress (Pa)
τ_{xr}	axial WSS component (Pa)
$\tau_{\theta r}$	azimuthal WSS component (Pa)
$\lvert\tau\rvert_{PM}$	mean value of coupled model peak WSS magnitudes ($\overline{\lvert\tau\rvert_P}$) (Pa)
$\lvert d\tau/dt\rvert_P$	peak TWSSG magnitude (Pa/s)
$\lvert d\tau/dt\rvert_{PM}$	mean value of coupled model peak TWSSG magnitudes (Pa/s)
θ	azimuthal position (°)
A_D	main branch cross-sectional area (m^2)
BPM	heart beats per minute (min^{-1})
c	exponential variable (–)
C	Casson model (–)
C_1	Walburn–Schneck empirical viscosity constant (Pa s)
$C_{2,3,4}$	Walburn–Schneck empirical constants (–)
d	diameter of daughter branch (m)
D	diameter of main branch (m)
D_H	RBC diffusivity (m^2/s)
H	RBC volume fraction (V_{RBC}/V_{Total}) (–)
H_{wall}	RBC inlet wall value (–)
IFC	inflow case (–)
k_C	Casson viscosity (Pa s)
k_0	Quemada zero shear viscosity (–)
k_∞	Quemada infinite shear viscosity (–)
nonN–N	non-Newtonian minus Newtonian models (–)
n	number of pulsation periods (–)
N	number of samples (–)
p	static pressure (Pa)
$P1$	inlet RBC profile 1 (–)
$P2$	inlet RBC profile 2 (–)
Q	Quemada model (–)
Q_{IN}	inlet flow rate (m^3/s)
r	radial co-ordinate (m)
RBC	red blood cell (–)
Re	The Reynolds number (–)
S1	near wall data point (–)
S2	flow field data point (–)
Sc	The Schmidt number (–)
t	pulsation time (s)
T	pulsation period time (s)
TPMA	total proteins minus albumin (g/l)
TWSSG	temporal wall shear stress gradient (Pa/s)
u	velocity vector (m/s)
U_0	peak mean inlet velocity (m/s)
V_{RBC}	volume of RBCs (m^3)
V_{Total}	total fluid volume (m^3)
WS	Walburn–Schneck model (–)
WSS	wall shear stress (Pa)
WSSG	wall shear stress gradient (Pa/m)
x	axial position (m)

is about 45% of the total blood volume [5,16,34]. The local RBC concentration cannot be assumed to be constant as the cells are subjected to different forces, contributing to the non-Newtonian viscous behavior. Thus, a realistic model of blood viscosity requires that the temporal and spatial RBC distribution is accounted for and integrated into the viscosity models. In this study, three different viscosity models have been implemented. The chosen models are comprehensively developed and widely used concerning RBC volume fraction (H) and shear rate ($\dot{\gamma}$).

The *Casson model*, initially derived to describe the flow behavior of printing ink, was adapted in order to describe blood viscosity [12,13]. In general, the local mixture viscosity coefficient (μ) is defined by the ratio of fluid shear stresses (τ) to the fluid shear rate ($\dot{\gamma}$), (Eq. (1)). The Casson model hypothesizes the stresses in the fluid mixture to be influenced by a yield stress, $\tau_y(H)$, i.e. the stress required to initiate flow [12,13]. The viscosity is defined according to this model as follows:

$$\mu = \frac{\tau}{\dot{\gamma}} \tag{1}$$

$$\mu = \frac{[\sqrt{k_C(H)\dot{\gamma}} + \sqrt{\tau_y(H)}]^2}{\dot{\gamma}} \quad \text{for} \quad \tau > \tau_y(H), \tag{2}$$

$$\dot{\gamma} = 0 \quad \text{for} \quad \tau <= \tau_y(H)$$

The terms $k_C(H)$ (Casson viscosity) and $\tau_y(H)$ (shear strength) take the form

$$k_C(H) = \frac{\mu_p}{(1-H)^{a\beta}} \tag{3}$$

$$\tau_y(H) = \left[\frac{a\alpha_C - 1}{a\beta}((1-H)^{a\beta/2} - 1)\right]^2 \tag{4}$$

where the grouped empirical constants, $a\beta$ and $a\alpha_C - 1$, are evaluated according to experimental values for $k_C(H) = 0.003$ Pa s and $\tau_y(H) = 0.0053$ Pa, determined for human blood at H~45% [15,36,40]. The Casson model is valid over a wide range of shear rates, $\dot{\gamma} > 1\ s^{-1}$ [13,54].

One of the most recently developed models is that of the *Quemada constitutive equation*, representing one of the broadest range of shear rates for blood, greater than approximately $0.01\ s^{-1}$ [54]. It was developed to describe the shear thinning viscosity dependency of concentrated particle suspensions through the following equation [42,43]:

$$\mu = \mu_p \left(1 - \frac{1}{2} H \left[\frac{k_0(H) + k_\infty(H)\sqrt{\dot{\gamma}/\dot{\gamma}_C(H)}}{1 + \sqrt{\dot{\gamma}/\dot{\gamma}_C(H)}}\right]\right)^{-2} \tag{5}$$

Parameters $\dot{\gamma}_C(H)$, $k_0(H)$ and $k_\infty(H)$ are the critical shear rate and non-dimensional intrinsic viscosities related to low and high shear rates, respectively. Empirical correlations for the RBC volume fraction (H) dependency on each of these parameters are