# Blood Flow in the Human Circulatory System

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Report of modern techniques for modeling the motion of blood within a Human's Macrocirculatory System.

**Keywords:** *computational hemodynamics, 0D blood-flow, 1D blood-flow, 2D-blood-flow, PINN's, finite element methods, discontinuous galerkin, Lax-Wendroff, fluid-structure ineraction (FSI)*

## Contents

# 1 Preliminaries

## 1.1 Notation

| | |
|---|---|
| $\mathbb{R}$ | set of real numbers |
| $\mathbb{R}^+$ | set of positive real numbers |
| $\mathbb{R}^-$ | set of negative real numbers |
| $\mathbb{R}^n$ | n-dimensional real vector space |
| $\Omega \subset \mathbb{R}^n$ | a connected open subset of $\mathbb{R}^n$ |
| $\overline{\Omega}$ | the closure of $\Omega$ |
| $\partial\Omega$ | the boundary of $\Omega$ |
| $dx$ | Lebesgue measure on $\mathbb{R}^n$ |
| $dS$ | surface measure on $\partial\Omega$ |
| $dV$ | volume measure on $\Omega$ |
| $\nabla$ | gradient operator |
| $\Delta = \nabla^2 = \nabla \cdot \nabla(\cdot)$ | Laplace operator |
| div | divergence of a vector field |
| **div** | divergence of a tensor |
| $\boldsymbol{v}_i$ | $i$-th component of vector $\boldsymbol{v}$ |
| $\langle \cdot, \cdot \rangle_X$ | inner product on vector space $X$ |
| $\langle \boldsymbol{u}, \boldsymbol{v} \rangle$ | inner product of vectors $\boldsymbol{u}, \boldsymbol{v} \in \mathbb{R}^n$ |
| $\dfrac{\partial}{\partial \hat{\boldsymbol{n}}} = \langle \nabla, \hat{\boldsymbol{n}} \rangle$ | normal derivative on $\partial\Omega$ |
| $\| \cdot \|$ | $L^2$-norm |
| $C^k(\Omega)$ | space of $k$ times continuously differentiable functions on $\Omega$ |
| $C_0^k(\Omega)$ | space of $k$ times continuously differentiable functions with compact support in $\Omega$ |
| $C_0^k(\overline{\Omega})$ | space of $k$ times continuously differentiable functions which have bounded and uniformly continuous derivatives up to order $k$ with compact support in $\Omega$ |
| $C_0^\infty(\Omega)$ | space of smooth functions with compact support in $\Omega$ |
| $L^p(\Omega)$ | Lebesgue space of $p$-integrable functions on $\Omega$ |

## 1.2 Symbols and Abbreviations

| | |
|---|---|
| $\therefore$ | consequently |
| $\because$ | because |
| $\Longrightarrow$ | implies |
| $\Longleftrightarrow$ | if and only if |
| $:=$ | defines |
| $\equiv$ | equivalence |
| a.e. | almost everywhere |
| e.g. | "exempli gratia" (for example) |
| i.e. | "id est" (that means) |
| s.t. | such that |
| m.b.s. | m.b.s. |
| w.r.t. | with respect to |
| wlog | without loss of generality |
| ODE | Ordinary Differential Equation |
| PDE | Partial Differential Equation |
| PDES | System of Partial Differential Equations |
| IC | Initial Condition |
| BC | Boundary Condition |
| 0D | Zero dimensional |
| 1D | One dimensional |
| 2D | Two dimensional |
| 3D | Three dimensional |
| FSI | Fluid-Structure Interaction |
| WHO | World-Health Organization |
| SB | Stenotic Blockage |
| bpm | beats per minute |
| RBC | Red Blood Cell |

## 1.3 Parameters and Units

| | |
|---|---|
| $\rho$ | density of blood $\left[\frac{kg}{m^3}\right]$ |
| $\eta$ | dynamic viscosity $\left[Pa \cdot s\right]$ |
| $\mu$ | kinematic viscosity $\left[\frac{m^2}{s}\right]$ |
| $\tau$ | shear stress |
| $\dot{\gamma}$ | shear rate |
| $R$ | radius of vessel with diameter $2R$ |
| $\boldsymbol{u}$ | velocity field |
| $p$ | pressure field |
| $W_0$ | Womersley number $\left[-\right]$ |
| $Re$ | Reynolds number $\left[-\right]$ |
| $Pe$ | Péclet number $\left[-\right]$ |
| $c$ | concentration of a material element |
| $D$ | diffusion coefficient $\left[\frac{m^2}{s}\right]$ |
| $t$ | time $\left[s\right]$ |
| $T$ | terminal time $\left[s\right], t > 0$ |
| $\omega$ | angular frequency $\left[\frac{rad}{s}\right]$ |
| $\boldsymbol{f_b}$ | body force per unit volume $\left[\frac{N}{m^3}\right]$ |

## 1.4 Mathematical Foundations

| | |
|---|---|
| $\rho$ | density of blood $\left[\frac{kg}{m^3}\right]$ |
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# 2 Introduction

Hemodynamics studies the kinematics of blood. Our interest is the kinematic motion of blood within the human macrocirculatory system, i.e. the flow of blood in large vessels such as arteries and veins. Blood is observed as a complex fluid of formed elements suspended in plasma, thus, the rheological behavior of blood is non-trivial. We report techniques and methodologies for modeling blood's motion in large vessels.

Our report is organized as follows.

We start our introduction by stating the report's motivation, then Sec. 2.1 provides a brief physiological review of the human circulatory system. We follow with Sec. 2.2, which reviews the continuum hypothesis, (a necessary postulate in fluid mechanics), which treats blood as a continuous medium. This framing reduces the hemodynamic problem to describing blood's motion in a continuum. Then Sec. 2.3 discusses various rheological assumptions and constitutive relations for modeling blood as an incompressible fluid. And finally, Sec. 2.4 derives the kinematic-viscosity Navier–Stokes (NS) equations, a system of nonlinear partial differential equations (PDEs) that herein serves as the foundation of our modeling efforts.

**Motivation** Coronary artery stenosis (CAS) is the narrowing of the coronary arteries due to the buildup of plaque. Such narrowing can restrict blood flow to the heart muscle, which may lead to various cardiovascular problems. Current methods for predicting a stenotic blockage (SB) in a coronary artery are rudimentary, and often SB predicition doesn't mean obstruction [[1]]. Additionally, current clinical methods for assessing the severity of a SB rely on imaging techniques such as angiography, intravascular ultrasound (IVUS), and optical coherence tomography (OCT) to visualize the arteries and identify areas of narrowing. Such methods provide valuable information about the anatomy of the arteries, but they do not provide direct information about the functional significance of the CAS. Functional assessment of CAS typically involves measuring the fractional flow reserve (FFR), which is the ratio of the blood pressure downstream of the stenosis to the blood pressure upstream of the stenosis during maximum blood flow. However, measuring FFR requires the use of a pressure wire, which can be invasive and carries some risks. Therefore, there is a need for non-invasive methods to assess the functional significance of CAS.

## 2.1 Physiology

The circulatory system consists of a human's heart, vascular network, lungs, and organs. The heart is the source, transporting Oxygen-rich blood to the organs and deoxygenated (and carbon dioxide-enriched) blood back to the lungs. Lungs discharge $CO2$ and enrich the blood with Oxygen. We refer to these respective processes as the *pulmonary circulation* and the *systemic circulation* (resp.). The *macrocirculatory system* consists of the heart and the large vessels in the systemic circulation. Particularly, the arteries of the macrocirculatory system transport oxygenated blood from the heart, driving the return of deoxygenated blood in large vessels back to the heart. Hemodynamics refers to the study of blood flow in the circulatory system.

Cardiovascular disease (CVD) is the leading cause of death in developed nations. According to the World Health Organization (WHO), CVD accounts for approximately 30% of all global deaths in 2012. Understanding the hemodynamics of the macrocirculatory system is crucial for diagnosing, treating, and preventing CVD. Consequently, our motivation is to simulate and analyze areterial stenosis with the aim to better enable patient-provider outcomes.

A single beat of the heart propels blood through the macrocirculatory system, the "lub-dub" sound. We refer to the beat and the following sequence of events until the successive beat as the *cardiac cycle*. The cardiac cycle consists of two main phases: systole and diastole, during which the heart chamber is accumulating blood and releasing blood (resp.). The beat can be recognized as a pulse wave in large vessels, characterized by the Wormersley number

$$W_0 := 2R \cdot \sqrt{\frac{\omega\rho}{\eta}}, \quad : \ \rho \text{ is fixed blood density}$$

a dimensionless parameter comparing the frequency $\omega$ of the pulse wave to the blood's dynamic viscosity $\eta$ and the vessel diameter $2R$. The Reynolds number $Re$ characterizes flow in blood vessels

$$Re := 2R \cdot \frac{\rho U}{\eta}, \quad : \ \rho \text{ is fixed blood density}$$

Low $Re$ indicates laminar flow, while high $Re$ suggests turbulent flow.

### Observed Cardiac Cycle Characteristics

Normal resting heart rate is considered to be $\omega = 70$ bpm, so the cardiac cycle is approximately $0.86s.$, consisting of:

1. Systole (ventricular contraction) $\approx 0.3$ seconds.
2. Diastole (ventricular relaxation) $\approx 0.7$ seconds.

The blood volume of a human is approximately 5.7-6.0 liters of blood, flowing a full cycle roughly every

minute. The energy driving the flow comes from oxygen and nutrients absorbed from food, creating waste products that must be removed; the *coronary artery*'s responsibility. The buildup of waste products results in Arteriosclerosis, a narrowing of the coronary artery, leading to reduced and turbulent blood flow. (Add citations here of turbulence in the presence of stenotic arteries).

Note [3, Table 1.1, p. 10, §1.1] shows $W_0 \propto 2R$ and $Re \propto (2R)^{-1}$; we observe large pulses and turbulent flow in large vessels and small pulses and laminar flow in small vessels.

**Constituents and hematocrit.** Blood consists of plasma and formed elements which we call cells. Red blood cells (RBCs) comprise $\approx 97\%$ of the cellular volume, and cellular volume is approximately $\approx 45\%$ of the blood volume. The remaining $\approx 55\%$ of blood volume is plasma, which is $\approx 90\%$ water. The ratio of RBC volume to total blood volume is the *hematocrit value* $H$, a key metric governing apparent viscosity $\eta$: as $H$ increases, $\eta$ typically increases (cf. S6.5.1 [2]). The formed elements suspended in plasma include white blood cells (WBCs) and platelets.

## 2.2 Continuum

A *domain* in an open, nontrivial, bounded, path-connected subset of $\mathbb{R}^N$ for $N \in \{1, 2, 3\}$. Our aim is to simulate the hemodynamics of a time-dependent spatial fluid domain $\Omega(t) \subset \mathbb{R}^N$, where

$$\Omega(t) := \{x \in \mathbb{R}^N \ : \ x \text{ lies inside the blood vessel at time } t\}$$

for all $t \in I_T$ s.t.

$$I_T \ := \ [0, T], \quad T > 0.$$

The spatial-temporal domain is

$$\Omega \ := \ \{(x, t) \in \mathbb{R}^{N+1} \ : \ x \in \Omega(t), \ t \in I_T\},$$

On $\Omega(t)$, we say that

$\phi : \Omega(t) \to \mathbb{R}$ is a *scalar field*,
$\boldsymbol{f} : \Omega(t) \to \mathbb{R}^N$ is a *vector field*,
$\boldsymbol{T} : \Omega(t) \to \mathbb{R}^{N \times N}$ is a *(second-order) tensor field*.

A second-order (rank-2) tensor $\boldsymbol{T}$ defines a linear map from $\mathbb{R}^N \to \mathbb{R}^N$, and once a basis is fixed, $\exists\ A \in \mathbb{R}^{N \times N}$ s.t. $\boldsymbol{T}(\boldsymbol{x}) = A\,\boldsymbol{x}$ for all $\boldsymbol{x} \in \Omega(t)$.

**Modeling Framework** At microscopic scales the continuum hypothesis breaks down as matter is a discrete collection of molecules, but at macroscopic scales empirical evidence suggests our models are accurate. Let the blood's velocity, thermodynamic pressure, and density fields be

$$\boldsymbol{u} : \Omega(t) \to \mathbb{R}^3, \ (x, y, z) \mapsto (u_1(x, y, z), \ u_2(x, y, z), \ u_3(x, y, z))^\top, \quad \left[\ \frac{m}{s^2}\ \right]$$
$$p : \Omega(t) \to \mathbb{R}, \ (x, y, z) \mapsto p(x, y, z), \quad \left[\ Pa \equiv \frac{N}{m^2}\ \right]$$
$$\rho : \Omega(t) \to \mathbb{R}^+, \ (x, y, z) \mapsto \rho(x, y, z). \quad \left[\ \frac{kg}{m^3}\ \right]$$

Our assumption that blood is a simple fluid ensures $\boldsymbol{u}$, $p$, and $\rho$ is sufficiently smooth in $\Omega$.

*Remark* (Simple Fluid). A *simple fluid* is a fluid where the Cauchy stress tensor depends only on the current state of deformation and rate of deformation at each point in the fluid, and not on the history of deformation. The contrary are *complex fluids*, where stress may depend on past deformations r.t.a. viscoelastic fluids, which exhibit both viscous and elastic characteristics. Here, we assume blood is a simple fluid, one that isn't

subject to memory effects.

**Definition 1.** Let

$$D_t\phi \;:=\; \partial_t\phi + \boldsymbol{u}\cdot\mathrm{div}(\phi),$$

be the material derivative $D_t\phi$ measuring the instantanous rate of change of quantity $\phi$ moving with velocity field $\boldsymbol{u}$. The literature also refers to $D_t$ as the *substantial derivative*, *advective derivative*, *lagrangian derivative*, or *convective derivative*.

For $\phi$ subject to the flow $\boldsymbol{u}$, note that

$$D_t\phi(\boldsymbol{x},t) \;=\; \lim_{\Delta t\to 0}\frac{\phi(\boldsymbol{x}+\boldsymbol{u}(\boldsymbol{x},t)\,\Delta t, t+\Delta t)-\phi(\boldsymbol{x},t)}{\Delta t},$$

i.e., $D_t\phi$ is the instantanous rate of change of quantity $\phi$ if a material fluid element $V(t)_{\boldsymbol{x}}$ positioned at $\boldsymbol{x}\in\Omega(t)$ and moving with velocity $\boldsymbol{u}(x,t)$. By the incompressibile assumption of blood, the material density of $V(t)_{\boldsymbol{x}}$ is $\rho(\boldsymbol{x},t)\equiv\rho_0$ for all $\boldsymbol{x}\in\Omega(t)$ and $t\in I_T$. Implying $D_t\rho=0$, so mass conservation in $\Omega(t)$ reads

$$\partial_t\rho + \mathrm{div}(\rho\boldsymbol{u}) = 0 \quad \Longleftarrow \text{ incompressibility assumption}$$

By assumption blood is Newtonian, linear momentum balance follows from Newton's 2nd Law ($F=ma$) as

$$\rho(\partial_t\boldsymbol{u} + (\boldsymbol{u}\cdot\nabla)\boldsymbol{u}) \;=\; \mathbf{div}(\boldsymbol{T}) + \rho\boldsymbol{f_b}, \qquad \text{in } \Omega(t)$$

where $\boldsymbol{T}$ is the Cauchy stress tensor, $\boldsymbol{f_b}$ may be some body force per unit mass $[\frac{m}{s^2}]$. Note that **div** acts rowise on $\boldsymbol{T}$, implying $\exists$ matrix $A \;:\; \boldsymbol{T}\boldsymbol{u}=\boldsymbol{T}$ $\because$ blood is a simple fluid. Let the rate-of-deformation tensor be defined as the symmetric part of the velocity gradient, i.e.,

$$\boldsymbol{D}(\boldsymbol{u}) := \tfrac{1}{2}\big(\nabla\boldsymbol{u} + (\nabla\boldsymbol{u})^\top\big) \quad \text{s.t.} \quad \nabla\boldsymbol{u} := \begin{bmatrix} \frac{\partial u_1}{\partial x} & \frac{\partial u_1}{\partial y} & \frac{\partial u_1}{\partial z} \\ \frac{\partial u_2}{\partial x} & \frac{\partial u_2}{\partial y} & \frac{\partial u_2}{\partial z} \\ \frac{\partial u_3}{\partial x} & \frac{\partial u_3}{\partial y} & \frac{\partial u_3}{\partial z} \end{bmatrix}.$$

Note that $\boldsymbol{u}\mapsto\boldsymbol{D}(\boldsymbol{u})$ captures spatial deformations of element $V_{\boldsymbol{x}}$ in $\Omega$ under flow $\boldsymbol{u}$.

**Definition 2.** A fluid is *Newtonian* if its Cauchy stress tensor $\boldsymbol{T}$ depends linearly on the rate-of-deformation tensor $\boldsymbol{D}(\boldsymbol{u})$.

**Definition 3.** A fluid is *isotropic* if its constitutive response is independent of the coordinate system.

Writing the Cauchy stress as $\boldsymbol{T} = \boldsymbol{T}(\boldsymbol{D})$, isotropy means that for every orthogonal rotator $\boldsymbol{Q} \in \mathrm{SO}(3)$,

$$\boldsymbol{Q}\,\boldsymbol{T}(\boldsymbol{D})\,\boldsymbol{Q}^{\top} \;=\; \boldsymbol{T}\big(\boldsymbol{Q}\,\boldsymbol{D}\,\boldsymbol{Q}^{\top}\big).$$

**Definition 4** (Newtonian, isotropic constitutive law)**.** For a Newtonian, isotropic fluid the Cauchy stress is

$$\boldsymbol{T} = -\,p\,\mathrm{I} + 2\,\eta\,\boldsymbol{D}(\boldsymbol{u}) + \lambda\,\mathrm{div}(\boldsymbol{u})\,\mathrm{I},$$

where $\eta > 0$ is the dynamic (shear) viscosity and $\lambda$ the bulk viscosity.

So an isotropic fluid at rest (quiescent state $\boldsymbol{u} \equiv \boldsymbol{0}$) sustains only hydrostatic stress:

$$\Longrightarrow\; \boldsymbol{T} \;=\; -\,p\,\mathrm{I} \quad \text{when } \boldsymbol{u} \equiv \boldsymbol{0}.$$

One may make a distinction between incompressible fluids and incompressible flows, and by assuming the former w.a.t.s. the divergence-free (senodial) condition on velocity feild $\boldsymbol{u}$.

**Definition 5** (Incompressibile fluid)**.** An element $V \in \Omega$ with constant density $\rho(\boldsymbol{x}, t)$ for all $\boldsymbol{x} \in V(t), t \in I_T$ is an *incompressible fluid*.

**Definition 6** (Incompressible flow)**.** An element $V \in \Omega$ subject to $\boldsymbol{u}$ s.t. $V$ with constant rate of material density change (in both space and time so that $D_t\rho = 0$ for all $t \in I_T$) undergoes an *incompressible flow* in $\Omega$.

For all material elements $V \in \Omega$, 5 implies 6. Note, the converse is not generally true: an incompressible flow ($D_t\rho = 0$) only preserves density along particle paths and allows $\rho = \rho(\boldsymbol{x})$ to vary spatially; but when the initial density is distributed uniformly in space, incompressible flow implies incompressible fluid.

*Remark* (Divergence-free condition)*.* By assuming blood is incompressible fluid and flow, we have

$$\begin{aligned}
&D_t\rho = 0 \\
&\iff \partial_t\rho + \mathrm{div}(\rho\boldsymbol{u}) = 0 \\
&\iff \mathrm{div}(\rho_0\boldsymbol{u}) = 0 \quad (\because\ \rho = \rho_0) \\
&\iff \nabla\cdot(\rho_0\boldsymbol{u}) = 0 \\
&\iff \langle\rho_0\boldsymbol{u}, \nabla\rangle = 0 \\
&\iff \rho_0\langle\boldsymbol{u}, \nabla\rangle = 0 \\
&\iff \langle\boldsymbol{u}, \nabla\rangle = 0 \\
&\iff \nabla\cdot\boldsymbol{u} = 0,
\end{aligned}$$

and we r.t. $\nabla\cdot\boldsymbol{u} = \mathrm{div}(\boldsymbol{u}) = 0$ as the *divergence-free condition* of $\boldsymbol{u}$ in $\Omega(t)$.

Consequently, the constitutive law simplifies as follows.

**Definition 7** (Incompressible stress tensor)**.** The Cauchy stress 4 simplifies to

$$\boldsymbol{T} \;=\; -p\,\mathrm{I} + 2\,\eta\,\boldsymbol{D}(\boldsymbol{u}).$$

*Remark* (Dynamic vs. Kinematic Viscosity). In our model assumptions, *dynamic viscosity* $\eta \in \mathbb{R}^+$ and *kinematic viscosity* $\mu \in \mathbb{R}^+$ relate as

$$\mu \;:=\; \frac{\eta}{\rho} \;=\; \frac{\eta}{\rho_0} \;\in\; \mathbb{R}^+.$$

Here $\eta$ quantifies the internal resistance of blood to shear deformation, i.e., $\eta := \dfrac{\tau}{\dot{\gamma}}$, with units $[Pa \cdot s]$. Moreover $\mu$ adjusts $\eta$ by the density $\rho$, capturing the viscous diffusion of momentum per unit mass, with units $\left[\frac{m^2}{s}\right]$. Intuitively, $\eta$ measures how "thick" or "sticky" the fluid is, while $\mu$ measures how quickly momentum diffuses through the fluid due to viscosity.

*Remark* (Newtonian Blood Justification). When diameter $d$ and hematocrit effects are needed, one may use a Non-Newtonian model with relative viscosity $\eta_r(H, d)$ that scales an absolute baseline $\eta$:

$$\eta_{\text{eff}} \;=\; \eta_r(H,d)\,\eta \quad \text{(effective viscosity)}$$

An empirical fit from [4]

$$\eta_r \;=\; 1 + (\eta_{0.45} - 1)\,\frac{(1-H)^C - 1}{(1-0.45)^C - 1} \text{ s.t. } \begin{cases} \eta_{0.45} = 6\,e^{-0.085\,d} + 3.2 - 2.44\,e^{-0.06\,d^{0.645}}, \\ C = \left(0.8 + e^{-0.075\,d}\right)\left(\dfrac{1}{1+10^{-11}\,d^{12}} - 1\right) + \dfrac{1}{1+10^{-11}\,d^{12}}, \end{cases}$$

where $d := 2R/(1.0\mu m)$ is the (scaled) vessel diameter. In large vessels, $\eta_r$ is often constant, justifying the Newtonian assumption. [[3], sec. 3.1]

## 2.3 Blood Model

## 2.4 Navier-Stokes

# 3 Appendix

## Bibliography

# References


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## Code Listings

Code listings

**Code 1:** Algorithm 16.5

```
1        function foo()
2            println("Hello World)
3        end
```