

# Modeling Blood Flow in Macrocirculatory System

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Notes pertaining to mathematical models of blood flow in the macrocirculatory system.

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# 1 Preliminaries

## Mathematical 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
$\Delta$	Laplace operator
$\text{div}$	divergence of a vector field
$\mathbf{div}$	divergence of a tensor
$v_i$	$i$ -th component of vector $\mathbf{v}$
$\langle \cdot, \cdot \rangle_X$	inner product on vector space $X$
$\langle \mathbf{u}, \mathbf{v} \rangle$	inner product of vectors $\mathbf{u}, \mathbf{v} \in \mathbb{R}^n$
$\frac{\partial}{\partial \hat{\mathbf{n}}} = \langle \nabla, \hat{\mathbf{n}} \rangle$	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 $\Omega$

## Symbols and Abbreviations

$\therefore$	consequently
$\because$	because
$\implies$	implies
$\iff$	if and only if
$:=$	defines
$\equiv$	equivalent
s.t.	such that
w.r.t.	with respect to
r.t.a.	referred to as
a.e.	almost everywhere
wlog	without loss of generality
i.e.	"id est" (that means)
e.g.	"exempli gratia" (for example)
ODE	Ordinary Differential Equation
PDE	Partial Differential Equation
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 Bloodflow
bpm	beats per minute

## Parameters and Units

$\rho$	density of blood	$\left[\frac{kg}{m^3}\right]$
$\eta$	dynamic viscosity	$[Pa \cdot s]$
$\mu$	kinematic viscosity	$\left[\frac{m^2}{s}\right]$
$\tau$	shear stress	
$\dot{\gamma}$	shear rate	
$W_0$	Wormsley number	$[-]$
$Re$	Reynolds number	$[-]$
$Pe$	Peclet number	$[-]$

## 2 Introduction

### 2.1 Physiological Background

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; which discharge  $CO_2$  and enrich 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. We aim to develop mathematical models that accurately describe blood flow in the macrocirculatory system. Our motivation is to use these models to simulate and analyze various cardiovascular conditions, such as arterial stenosis, aneurysms, and heart valve disorders.

A single beat of the heart propels blood through the macrocirculatory system, the "lub-dub" sound. We refer to the beat and the 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 Wormsley number  $W_0$ :

$$W_0 := D \cdot \sqrt{\frac{\omega \rho}{\eta}},$$

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

$$Re := \frac{\rho U D}{\eta},$$

where  $U$  is the mean flow velocity. 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 human is approximately 5.7-6.0 liters of blood, flowing a full cycle approximately 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 [1, Table 1.1, p. 10, §1.1] shows  $W_0 \propto D$  and  $Re \propto D^{-1}$ ; we observe large pulses and turbulent flow in large vessels and small pulses and laminar flow in small vessels.

## Properties of Blood

Blood consists of plasma and cells. The red blood cells comprise  $\approx 97\%$  of the cell volume, which is  $\approx 45\%$  of blood volume. The remaining blood volume, plasma, is  $\approx 90\%$  water. The ratio of red blood cells and the total blood volume is called the *hematocrit value*, a metric describing the influence of red blood cells on the flow.

**Definition 1.** Let  $V$  be a fluid volume  $V$  with viscosity  $\eta = \frac{\tau}{\dot{\gamma}} \equiv \text{const.}$ , where *shear stress*  $\tau := \frac{F}{A}$  (s.t.  $F$  is parallel to it's surface  $S$  with area  $A$ ) and *shear rate*  $\dot{\gamma}$ . Such fluids are *Newtonian fluids*.

A shear stress  $\tau$  deforms  $V$  by an angle  $\gamma \implies \dot{\gamma}$  is an angular velocity (const. for Newtonian fluids). Our models assume blood is Newtonian. We use a relative viscosity  $\eta_r$ , which depends on the absolute viscosity  $\eta$ , scaled vessel diameter  $D := D/(1.0\mu m)$ , and hematocrit value  $H$ . In particular, an empirical model from [3] gives:

$$\eta_r = 1 + (\eta_{0.45} - 1) \frac{(1 - H)^C - 1}{(1 - 0.45)^C - 1} : \begin{cases} \eta_{0.45} = 6e^{-0.085D} + 3.2 - 2.44e^{-0.06D^{0.645}} \\ C = (0.8 + e^{-0.075D}) \cdot (-1 + \frac{1}{1+10^{-11}D^{12}}) + \frac{1}{1+10^{-11}D^{12}} \end{cases} . \quad (1)$$

In large vessels,  $\eta_r$  is observed constant ( $\implies$  Newtonian behavior).

Our concern is the flow of blood in the macrocirculatory system. We develop mathematical models describing the hemodynamics of arterial and large venous segments, then we extend our models to macrocirculation networks using a domain decomposition approach.

## 2.2 Navier Stokes System

In *continuum mechanics*, matter is modeled by *continuous fields*. For instance, to model the behavior of an *incompressible* fluid in time, one could use a **pressure field** and a **velocity field**. At *atomic scales*, this model breaks down—fluids as fluids are discrete collections of molecules, not continuous fields. However, at the macroscopic scale, one can hope that such models remain accurate.

The fundamental mathematical model for incompressible fluids (e.g., water) is the **viscous incompressible Navier-Stokes equations** (NS):

$$\begin{cases} \frac{\partial}{\partial t} \mathbf{u} + (\mathbf{u} \cdot \nabla) \mathbf{u} = \nu \Delta \mathbf{u} - \nabla p, & \text{(Momentum equation: Newton's law } F = ma) \\ \nabla \cdot \mathbf{u} = 0, & \text{(Incompressibility condition)} \\ \mathbf{u}(0, x) = \mathbf{u}_0(x), & \text{(Initial conditions)} \end{cases}$$

where  $\mathbf{u} : [0, +\infty) \times \mathbb{R}^3 \rightarrow \mathbb{R}^3$  is the velocity field and  $p : [0, +\infty) \times \mathbb{R}^3 \rightarrow \mathbb{R}$  is the pressure field. The initial velocity field is given by:

$$\mathbf{u}_0 : \mathbb{R}^3 \rightarrow \mathbb{R}^3, \quad \nabla \cdot \mathbf{u}_0 = 0.$$

The *global regularity problem* for (NS) asks:

*For any smooth, spatially localized initial data  $\mathbf{u}_0$ , does there exist a global smooth solution  $(\mathbf{u}, p)$  to (NS)?*

This problem is one of the seven Millennium Prize Problems posed by the Clay Mathematics Institute in 2000, with a prize of one million dollars for a correct solution.

It is known that (NS) has a *local existence and uniqueness theorem*:

**theorem.** For any smooth, spatially localized initial data  $\mathbf{u}_0$ , there exists a **maximal time of existence**  $0 < T_* \leq +\infty$  and a solution.

If  $T_* \neq +\infty$ , then **blowup** occurs in the sense that

$$\sup_{x \in \mathbb{R}^3} |\mathbf{u}(t, x)| \rightarrow +\infty \quad \text{as } t \rightarrow T_*.$$

If  $T_* = +\infty$ , no blowup occurs, and  $|\mathbf{u}| \rightarrow 0$  as  $t \rightarrow +\infty$ .

Numerical simulations suggest that global regularity is generally **true**—for *most* choices of initial data  $\mathbf{u}_0$ , one has a global smooth solution to (NS).

However, for large initial data, turbulence can occur. Energy moves from coarse to fine scales in a complex way before dissipating due to viscous effects. The NS equations contain two competing effects:

- The **transport term**:  $\mathbf{u} \cdot \nabla \mathbf{u}$
- The **dissipation term**:  $\nu \Delta \mathbf{u}$

Heuristically, if  $\nu\Delta\mathbf{u} \gg (\mathbf{u} \cdot \nabla)\mathbf{u}$ , one expects linear, non-turbulent behavior (*global regularity*). If  $(\mathbf{u} \cdot \nabla)\mathbf{u} \gg \nu\Delta\mathbf{u}$ , one expects **nonlinear turbulence** (and possibly **blowup**).

### Kinetic Energy Considerations

The kinetic energy is given by:

$$E(t) = \frac{1}{2} \int_{\mathbb{R}^3} |\mathbf{u}(t, x)|^2 dx.$$

This quantity is *decreasing in time* due to dissipation. Heuristically:

$$E(t) \gtrsim V^2 L^3.$$

This leads to a bound:

$$V = O(L^{-3/2}).$$

### Blowup Scenarios

This suggests a **possible blowup scenario**. If the velocity field concentrates in a ball of radius  $L_1$ , we estimate:

$$|\mathbf{u}| \approx L_1^{-3/2}.$$

At a later time  $t_2$ , the energy has concentrated further into a ball of radius  $L_2 = L_1/2$ , so:

$$|\mathbf{u}| \approx L_2^{-3/2}.$$

This leads to **potential infinite velocity** at a single spatial point:

$$\sum_n L_n^{r_2} < \infty.$$

At each stage, the dissipative forces are negligible compared to the transport effects. This results in an approximately self-similar solution.



### 3 Mathematical Models of Blood Flow

Mathematical models of blood flow in the macrocirculatory system can be classified into different categories based on their dimensionality and complexity. The choice of model depends on the specific application, computational resources, and desired accuracy.

### 4 Numerical Methods for Blood Flow Simulation

Numerical methods play a crucial role in simulating blood flow in various vascular geometries. These methods allow for the approximation of complex fluid dynamics equations and enable the study of hemodynamics in realistic scenarios.

### 5 Misc

Coronary artery stenosis (CAS) is the narrowing of the coronary arteries due to the buildup of plaque. This narrowing can restrict blood flow to the heart muscle, leading to various cardiovascular problems, including chest pain (angina), heart attacks, and other serious complications. Current methods for predicting coronary artery stenosis are rudimentary; and often prediction does mean coronary artery stenosis obstruction ([2]). . There is a need for more accurate and reliable methods to predict and assess the severity of CAS. narrowing can restrict blood flow to the heart muscle, leading to various cardiovascular problems, including chest pain (angina), heart attacks, and other serious complications. Current methods for predicting coronary artery stenosis are rudimentary; and often prediction does mean coronary artery stenosis obstruction. . There is a need for more accurate and reliable methods to predict and assess the severity of CAS.

We perform a literature survey of arterial blood flow using known methods from the literature, with the hope of understanding the computational challenges and tradeoffs of various *mathematical models*.

Provide an overview of the different numerical methods used for blood flow simulation, including finite element methods (FEM), finite volume methods (FVM), and computational fluid dynamics (CFD) approaches. Discuss the advantages and limitations of each method.

Make some comment about "correct termi-

## 6 Appendix

### References

- [1] Luca Formaggia, Alfio Quarteroni, and Alessandro Veneziani. *Cardiovascular Mathematics. Modeling and Simulation of the Circulatory System*. Springer, 2009.
- [2] Add authors here. “Add exact title here”. In: *European Heart Journal* (2021). DOI: 10.1093/eurheartj/ehab332. URL: <https://watermark.silverchair.com/ehab332.pdf>.
- [3] Gaehtgens P Pries AR Neuhaus D. “Blood viscosity in tube flow: dependence on diameter and hematocrit”. In: *Am J Physiol.* (6 Pt 2).263 (1992 Dec). DOI: 10.1152/ajpheart.1992.263.6.H1770.

### References

### Code Listings

Optional Space for supplementary code listings of computations done while investigating

#### Code 1: Algorithm 16.5

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