Modeling Blood Flow in Macrocirculatory System

Names Here

November 5, 2025

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

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

Contents

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 Ω
$\partial\Omega$	the boundary of Ω
dx	Lebesgue measure on $\mathbb{R}^n$
dS	surface measure on $\partial\Omega$
dV	volume measure on Ω
∇	gradient operator
Δ	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 Ω
$C_0^k(\Omega)$	space of k times continuously differentiable functions with compact support in Ω
$C_0^k(\overline{\Omega})$	space of k times continuously differentiable functions which have bounded and uniformly continuous derivatives up to order k with compact support in Ω
$C_0^\infty(\Omega)$	space of smooth functions with compact support in Ω
$L^p(\Omega)$	Lebesgue space of p-integrable functions on Ω

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
m.b.s.	m.b.s.
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
RBC	Red Blood Cell

Parameters and Units

ρ	density of blood $\left[\frac{kg}{m^3}\right]$
η	dynamic viscosity $\left[Pa \cdot s\right]$
μ	kinematic viscosity $\left[\frac{m^2}{s}\right]$
τ	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 $\left[-\right]$

Mathematical Notation

ρ	density of blood $\left[\frac{kg}{m^3}\right]$
η	dynamic viscosity $\left[Pa \cdot s\right]$
μ	kinematic viscosity $\left[\frac{m^2}{s}\right]$
τ	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 $\left[-\right]$

2 Introduction

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 ([3]). 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.

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. 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 various cardiovascular conditions, including arterial stenosis, aneurysms, and heart valve disorders, with the aim to better enable provider treatment.

SEO for insurance companies

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 Wormersley number W_0:

$$W_0 := 2R \cdot \sqrt{\frac{\omega\rho}{\eta}}$$

a dimensionless parameter comparing the frequency ω of the pulse wave to the blood's kinematic viscosity η and the vessel diameter $2R$.

Definition 1. The Reynolds number Re characterizes flow in blood vessels:

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

where U is the mean flow velocity.

Low Re indicates laminar flow, while high Re suggests turbulent flow. what is ρ?

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) ≈ 0.3 seconds.
2. Diastole (ventricular relaxation) ≈ 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).

Explain Anuerysms and their hemodynamic characteristics

Explain Heart valve conditions and there hemodynamic Characteristics

Note [1, 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.

2.2 Fluid Model of Blood

We define the term *domain* as an open, nontrival, bounded, and path-connected subset of $\mathbb{R}^N : N = 1, 2, 3$. Our aim is to model the kinematics of blood on the time interval $[0, T]$ in the lumen $\Omega(t) \subset \mathbb{R}^3$ of a blood vessel:

$$\Omega(t) := \{x \in \mathbb{R}^3 \ : \ x \text{ is inside the blood vessel at time } t\},$$

Here we say $\Omega(t)$ is the *fluid domain*.

Definition 2. Let Ω be a domain, then if:

$$
\begin{aligned}
f : \Omega \to \mathbb{R}, &\quad \text{then } f \text{ is a scalar feild.} \\
\boldsymbol{f} : \Omega \to \mathbb{R}^N, &\quad \text{then } f \text{ defines a vector feild.} \\
\boldsymbol{T} : \Omega \to \mathbb{R}^{N\times N}, &\quad \text{then } T \text{ defines a tensor feild,} \\
&\quad \text{assuming } T \text{ defines a second-order tensor on } \Omega.
\end{aligned}
$$

Constituents and hematocrit. Blood consists of plasma and formed elements (cells). Red blood cells (RBCs) comprise $\approx 97\%$ of the cellular volume, and cells occupy $\approx 45\%$ of the blood volume. The remaining $\approx 55\%$ is plasma, which is $\approx 90\%$ water. The ratio of RBC volume to total blood volume is the *hematocrit value* H, an important metric describing the influence of red blood cells on the flow. Blood viscosity η increases dramatically as H increases (see S6.5.1 [2]).

Modeling framework. In *continuum mechanics*, fluids are modeled by *continuous fields*. At *microscopic scales*, our models break down—fluids as fluids are discrete collections of molecules, not continuous fields. However, at macroscopic scales we can hope that such models remain accurate. We proceed with an overview of the continum hypothesis in the context of blood flow, explaining the consequences of assuming that blood is a single-constituent, homoegenous, isotropic fluid in domain $\Omega(t)$.

cite references that our asssump. holds in the scale of large artiers

explain how isotropic implies Adiabatic

Let the blood's velocity, thermodynamic pressure, and density fields be

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

Consider a fluid element V, explain how newton's second law implies Euler equations for invcisid and incompressible. Then assuming the flow is incompresible, we obatin the Cauchy equations for conservation of mass and balance of momentum.

Then the rate-of-deformation tensor is defined as the symmetric part of the velocity gradient,

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

measuring how a continuum of fluid deforms locally under the velocity field $\boldsymbol{u}$.

Definition 3. A fluid is *Newtonian* if its Cauchy stress $\boldsymbol{T}$ depends linearly on the rate-of-deformation tensor $\boldsymbol{D}(\boldsymbol{u})$.

Definition 4. 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 $\boldsymbol{Q} \in \mathrm{SO}(3)$,

remove group ref to SO(3)

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

Definition 5 (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 λ the bulk viscosity.

Note, an isotropic fluid at rest (quiescent state $\boldsymbol{u} \equiv \boldsymbol{0}$) sustains only hydrostatic stress: $\boldsymbol{T} = -p\,\mathrm{I}$.

Definition 6 (Incompressibile fluid)**.** A fluid is *incompressible* if its density is constant in space and time.

Definition 7 (Incompressible flow)**.** A flow is *incompressible* if density is materially constant in space and time. Let $D_t := \partial_t + \mathrm{div}\big((\cdot)\boldsymbol{u}\big)$ be the material derivative, then incompressible flow means $D_t(\rho) = 0$ in $\Omega(t)$ for all time t.

Remark. By assumption that blood is an incompressible fluid, $\rho \equiv \rho_0 > 0$ constant. By mass conservation of an incompressible fluid volume, it may be shown that the fluid's flow is incompressible in the sense of Definition 7. Assuming incompressibility of the fluid and flow, we have the equivalences:

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

So under incompressibility of the fluid and flow, the divergence-free condition $\mathrm{div}(\boldsymbol{u}) = 0$ of our velocity field holds in $\Omega(t)$. Consequently, the Cauchy stress simplifies to

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

Remark (Dynamic vs. Kinematic Viscosity). Throughout assume $\eta \in \mathbb{R}^+$ denote the *dynamic viscosity* of blood, and let $\mu \in \mathbb{R}^+$ denote the *kinematic viscosity*. Then:

$$\mu := \frac{\eta}{\rho}$$

where ρ is the blood density $\left[\frac{kg}{m^3}\right]$ (assumed constant by incompressibility). Thus:

- η quantifies the internal resistance of blood to shear deformation, i.e., $\eta := \frac{\tau}{\dot{\gamma}}$, with units $[Pa \cdot s]$;
- μ adjusts η by the density ρ, capturing the viscous diffusion of momentum per unit mass, with units $\left[\frac{m^2}{s}\right]$.

Intuitively, η measures how "thick" or "sticky" the fluid is, while μ measures how quickly momentum diffuses through the fluid due to viscosity.

We will model blood as Newtonian in large arteries by taking η constant (so $\mu = \eta/\rho$ constant). When diameter d and hematocrit effects are needed, we use a relative viscosity $\eta_r(H, d)$ that scales an absolute baseline η:

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

How do we conclude now that η is constant, so μ is constant? Confirm this follows from Newtonian assumption.

An empirical fit (e.g. [5]) is

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

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

2.3 Navier Stokes System

The Navier-Stokes (NS) system in conservative form:

Definition 8 (Incompressible-Newtonian NS (Conservative Form))**.**

$$\begin{cases} \rho\big(\partial_t \boldsymbol{u} + (\boldsymbol{u}\cdot\nabla)\boldsymbol{u}\big) = -\nabla p \;+\; \mathbf{div}\Big(2\,\mu(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big) \;+\; \rho\,\boldsymbol{f} \\ \operatorname{div}(\boldsymbol{u}) = 0, \end{cases}$$

where $\boldsymbol{f}$ is an external force field (e.g., gravity),

We rewrite Eq. 8 in standard form (as found in [4], [1].)

Definition 9.

$$
\begin{aligned}
&\rho\big(\partial_t \boldsymbol{u} + (\boldsymbol{u}\cdot\nabla)\boldsymbol{u}\big) = -\nabla p \;+\; \mathbf{div}\Big(2\,\mu(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big) \;+\; \rho\,\boldsymbol{f} \\
&\iff \rho\big(\partial_t \boldsymbol{u} \;+\; (\boldsymbol{u}\cdot\nabla)\boldsymbol{u}\big) \;=\; -\nabla p \;+\; \mathbf{div}\Big(2\,\frac{\eta}{\rho}(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big) \;+\; \rho\,\boldsymbol{f} \\
&\iff \rho\big(\partial_t \boldsymbol{u} \;+\; (\boldsymbol{u}\cdot\nabla)\boldsymbol{u}\big) \;=\; -\nabla p \;+\; \eta\;\mathbf{div}\Big(2\,\frac{1}{\rho}(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big) \;+\; \rho\,\boldsymbol{f} \quad (\because\ \eta = \frac{\mu}{\rho} = \text{const.}) \\
&\iff \rho\big(\partial_t \boldsymbol{u} \;+\; (\boldsymbol{u}\cdot\nabla)\boldsymbol{u}\big) \;=\; -\nabla p \;+\; \mu\,\rho\;\mathbf{div}\Big(2\,\frac{1}{\rho}(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big) \;+\; \rho\,\boldsymbol{f} \\
&\iff \partial_t \boldsymbol{u} \;+\; (\boldsymbol{u}\cdot\nabla)\boldsymbol{u} \;=\; -\frac{\nabla p}{\rho} \;+\; \mu\;\mathbf{div}\Big(2\,\frac{1}{\rho}(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big) \;+\; \boldsymbol{f} \\
&\iff \partial_t \boldsymbol{u} \;+\; (\boldsymbol{u}\cdot\nabla)\boldsymbol{u} \;+\; \frac{\nabla p}{\rho} \;-\; \mathbf{div}\Big(\frac{2}{\rho}\,\mu\,(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big) \;=\; \boldsymbol{f} \\
&\iff F\big(\partial_t \boldsymbol{u},\, \nabla\boldsymbol{u},\, \nabla p,\, \boldsymbol{u},\, p;\, \rho, \mu\big) \;=\; \boldsymbol{f}
\end{aligned}
$$

Remark. The NS system 8 is a non-linear coupled system of PDEs. The first equation expresses the conservation of linear momentum, as derived from Newton's second law, where

- $\rho(\boldsymbol{u}\cdot\nabla)\boldsymbol{u} = \rho\begin{bmatrix}\langle \boldsymbol{u}, \nabla \boldsymbol{u}_1\rangle \\ \langle \boldsymbol{u}, \nabla \boldsymbol{u}_2\rangle \\ \langle \boldsymbol{u}, \nabla \boldsymbol{u}_3\rangle\end{bmatrix}$ is the convective acceleration term (non-linear),
- $\mathbf{div}\Big(2\,\mu(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big)$ is the viscouselastic and diffusive term (linear if μ constant).

The second equation is the continuity equation, a consequence of the assumed fluid properties of blood. The total system comprises of four equations in four unknowns: the three components of the velocity field $\boldsymbol{u}$ and the pressure field p.

We will seek solutions of Eq. 8 to an initial value problem.

Definition 10. Let $\boldsymbol{u_0} : \Omega(0) \to R^3,\ \boldsymbol{x} \mapsto \boldsymbol{u_0}(x)\ :\ u(0,(x)) = \boldsymbol{u_0}$. We refer to $\boldsymbol{u_0}$ as the initial condition of velocity feild $\boldsymbol{u}$

In practice, $\boldsymbol{u_0}$ may be prescribed or determined from sensor data.

To model non-Newtonian effects (when $\eta \neq$ constant), the kinematic viscosity $\mu(\cdot)$ is often chosen by Careau model [2]

$$2\,\mu(|\boldsymbol{D}|^2) = \eta_\infty + (\eta_0 - \eta_\infty)\cdot\big(1 + \kappa|\boldsymbol{D}|^2\big).$$

Where η_0 and η_∞ are chosen to be the viscosity for very small and very large shear rates, resp., and $\kappa \in \mathbb{R}^+$ and $n \in (-0.5, 0)$ are model parameters. According to [1], we often set

$$\eta_0 = 65.7\cdot 10^{-3}\ Pa\cdot s,\ \eta_\infty = 4.45\cdot 10^{-3}\ Pa\cdot s, \kappa = 212.2\ s^2,\ \text{ and } n = -0.325$$

In a Newtonian case, one often chooses $\mu(|\boldsymbol{D}|^2) = \eta_\infty$ ([1], pg. 38).

If pressure P and the velocity $\boldsymbol{u}$ are given, the Caucy stress $\boldsymbol{T}$ is computed from Eq. . The wall shear stress (WSS) at the vessel wall is:

$$\text{WSS} := \langle \boldsymbol{t_{blood}},\ \boldsymbol{T}\,\hat{n}\rangle \quad : \quad \begin{cases} t_{blood} \text{ is tangent of a flow line through a cross-sectional area} \\ \hat{n} \text{ is outer normal of the cross-sectional area} \end{cases}$$

Forgoing the rigid-wall assumption allows us to model the relationship between the vessel wall and blood flow. Applicable models are reffered to as fluid-structure interaction (FSI) models.

discuss in a later section

2.3.1 NS in Cylindrical Coordinates

Let our vessel wall $\partial\Omega$ be a surface in $\mathbb{R}^3$ that evolves in time which we refer to as the interface. Let $\overline{\Omega} = \partial\Omega \cup \Omega$ be the closed and compact region enclosed by our interface. So the region enclosed by our interface is Ω, and we aim to model the velocity and pressure fields on Ω.

The relationship between cartesian and cylidrical coordinates is

$$(x,\ y,\ z)\ \mapsto (r\sin(\theta),\ r\cos(\theta),\ z), \quad r = \sqrt{x^2+y^2}.$$

Assume a vessel of length L is aligned with the z-axis whose cross-section is circular with radius $R(z,t)$ at axial position z and time t. Our fluid domain becomes

$$\Omega(t)\ =\ \{(r,\ ,\theta\ ,z) \in \mathbb{R}^3\ :\ r \in [0, R(z,t)],\ \theta \in [0, 2\pi),\ z \in [0, l)\}$$

3 Mathematical Models of Blood Flow

One chooses a model based upon the specific application, computational resources, and desired accuracy. We construct models of blood flow in various geometries, starting from a single vessel, then extendind our approach to bifurcations and arterial networks. Our strategy involves a *domain decomposition approach*.

3.1 Dimension-Reduced Models of Blood Flow

We start our disscusion with 1D and 0D models, reducing the d.o.f. in the NS system 8 by imposing further simplifying assumptions.

3.1.1 1D Models

By averaging pressure and blood velocity on slice of a vessel, we consider the flow through each cross-section, leading to a model of the flow along the length of the vessel. Because our model averages pressure and velocity over a surface-area, we obtain a uniform distibution of WSS on the vessel. There are various approaches to deriving a 1D model:

1. Assume $R << \ell$, e.g., (ref dimension reduced paper)
2. Integrating the NS system 8 in a compliant vessel.
3. A simplification of the reynolds equation (**??**, Chap. 10, **??**, **??**)

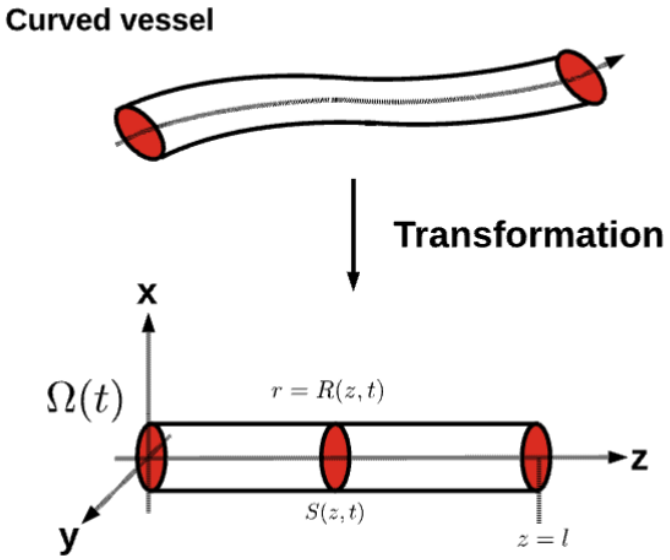

Figure 1: From [1] [Fig. 3.2, pg. 37]

Assume the vessel under study may be transformed to a simplified geometry as displayed in Figure **??**. One may start by introducing a rigid-vessel assumption, which leads to a no slip condition that $\boldsymbol{u}\big|_{\partial\Omega} = \mathbf{0}$. Instead we seek to model the link between blood flow and the deformation of the vessel wall.

Let $V(t) \subset \Omega(t)$ be a material fluid element of the vessel $\overline{\Omega}(t)$ such that $\partial V(t) \subset \partial\Omega(t)$, i.e., $V(t)$ is on the the vessel wall. According to the Reynold's transport theorem for $f \in L^1(\Omega(t))$

$$\frac{d}{dt}\int_{V_t} f dV \;=\; \int_{V(t)} \frac{\partial f}{\partial t} dV \;+\; \int_{\partial V(t)} (\boldsymbol{u_b}\cdot\hat{\boldsymbol{n}}) f dS$$

where $\boldsymbol{u_b}$ is the velocity feild deforming the boundary $\partial V(t)$ (Pf. see wiki). If we assume the normal component of $\boldsymbol{u_b} = \mathbf{0}$ near the inlet and outlet boundaries S_1 and S_2 (resp.) of Ω, then the motion of the vessel wall is coupled to the blood flow through the fluid element $V(t)$. The velocity $\boldsymbol{u_b}$ is equivalent to the velocity of the vessel wall $\partial\Omega(t)$ in contact with the boundary element $\partial V(t)$. I.e., the vessel wall velocity $\boldsymbol{u_w} = \boldsymbol{u_b}$. Now let $\boldsymbol{w} = \boldsymbol{u_w} - \boldsymbol{u}$ be the relative velocity of the vessel wall w.r.t. the velocity $\boldsymbol{u} = (u_1, u_2, u_3)^\top$ of the blood element $V(t)$. Then it follows that

$$\begin{aligned}\int_{\partial V(t)} \big(\boldsymbol{u_b}\cdot\hat{\boldsymbol{n}}\big) f \; dS \;&=\; \int_{\partial V(t)} \big(\boldsymbol{u_w}\cdot\hat{\boldsymbol{n}}\big) f \; dS \\ &=\; \int_{\partial V(t)} \big(\boldsymbol{w}\cdot\hat{\boldsymbol{n}}\big) f \; dS \;+\; \int_{\partial V(t)} \big(\boldsymbol{u}\cdot\hat{\boldsymbol{n}}\big) f \; dS\end{aligned}$$

Let $\bar{f}$ denote the average value of f defined over a surface S

$$\bar{f} \;:=\; \frac{1}{A}\int_{S(z,t)} f \; dS \quad : \quad A(z,t) \;:=\; \int_{S(z,t)} dS$$

No we may rewrite the volume integral in the LHS of RT theorem

$$\int_{V_t} f dV \;=\; \int_{z_1}^{z_2}\int_{S(z,t)} f \; dS \; dz \;=\; \int_{z_1}^{z_2} A\cdot\bar{f} \; dz$$

where $z_1 < z_2$ are fixed z-coordinates for S_1 and S_2. Then we differentiate the integrands in the above equation w.r.t. t

$$\int_{V(t)} \frac{\partial f}{\partial t} dV \;=\; \int_{z_1}^{z_2} \frac{\partial}{\partial t}\Big[A\cdot\bar{f}\Big] \, dz,$$

and we've rewritten the first term in the RHS of the reynolds system. The surface integral in the RHS may be written as

$$\int_{\partial V(t)} (\boldsymbol{u_b}\cdot\hat{\boldsymbol{n}}) f dS = \int_{\partial V(t)} (\boldsymbol{u_b}\cdot\hat{\boldsymbol{n}}) f dS....$$

With a little more work, one may obtain:

cleanup, ref. pg. 43

Definition 11. The 1D Reynolds Transport theorem for both compressible and incompressible fluids:

$$\frac{\partial}{\partial t}\left(A\bar{f}\right) + \frac{\partial}{\partial z}\left(A(\overline{f \cdot u_3})\right) = \int_S \left(\frac{\partial f}{\partial t} + \nabla \cdot (f\boldsymbol{u})\right) dS + \int_{\partial S} f\boldsymbol{w} \cdot \hat{\boldsymbol{n}}\, d\gamma$$

Remark. By taking $f = \rho$ in 11, mass conservation follows directly. Also, by our assumption that blood is incompressible, we have $\begin{cases} \operatorname{div}(\boldsymbol{u}) = 0 \\ \rho = \text{const.} \end{cases}$ and we simplify 11 as

$$\frac{\partial A}{\partial t} + \frac{\partial}{\partial z}\left(A(\overline{u_3})\right) = \int_{\partial S} \boldsymbol{w} \cdot \hat{\boldsymbol{n}}\, d\gamma$$

The RHS term above describing the transport process across the vessel wall.

complete, pg. 45

Remark. By taking $f = u_3$ in 11, momentum conservation follows directly. Also, by our assumption that blood is incompressible, we simplify 11 as

$$\frac{\partial}{\partial t}\left(Au_3\right) + \frac{\partial}{\partial z}\left(A(\overline{u_3^2})\right) = \int_S \left(\frac{\partial u_3}{\partial t} + \nabla u_3 \cdot \boldsymbol{u}\right) dS + \int_{\partial S} u_3 \boldsymbol{w} \cdot \hat{\boldsymbol{n}}\, d\gamma$$

The RHS term above describing the transport process across the vessel wall.

3.1.2 0D Models

The 0D model, on the other hand, treats the vessel as a lumped parameter system, focusing on overall pressure and flow relationships without spatial resolution.

4 Numerical Methods for Blood Flow Simulation

Numerical methods for PDEs, or *schemes*, are generally of the form:
To ensure converge of our schemes, we choose an initial condition by solving the homogenous-stationary NS problem in standard form 8:

Definition 12 (Stationary Stokes Problem)**.**

$$\begin{cases} -\mathbf{div}(\mu \boldsymbol{D}(\boldsymbol{u_0})) + \nabla p = \boldsymbol{f}, \\ \operatorname{div}(\boldsymbol{u_0}) = 0 \end{cases} \quad \text{in } \Omega(t)$$

and we seek a strong solution . We take the $\boldsymbol{f} \equiv \boldsymbol{0}$ with

Let

$$\begin{cases} S_0 := S(0,t) & \text{be the inlet} \\ S_T := S(\ell,t) & \text{be the outlet} \end{cases} \quad \forall\, t \in [0,T]$$

Suitable boundary conditions are, e.g., S_0 m.b. a time-dependent velocity or pressure profile and the outlet S_T m.b. WSS stress values, as determined from a pressure and velocity profile.

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.

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 ([3]). 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*.

Make some comment about "correct terminology for describing the types of coronary arterial stenosis is "coronary artery stenosis morphology." and ref figures in DOI: 10.1056

5.0.1 Existence and Uniqueness of NS

Remark (Global Regularity Problem for (NS)). *For any smooth, spatially localized initial data* $\mathbf{u}_0$*, does there exist a global smooth solution* $(\mathbf{u}, p)$ *to NS 8 Such question is one of the Millennium Prize Problems posed by the Clay Mathematics Institute in 2000, with a prize of one million dollars for a correct solution.*

theorem (Local Existence and Uniqueness)**.** Given smooth, localized initial data $\boldsymbol{u}_0$, there exists a maximal time $0 < T_* \leq \infty$ for which a unique solution exists.

If $T_* < \infty$, a **blow-up** occurs:

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

Otherwise, if $T_* = \infty$, then $|\boldsymbol{u}| \to 0$ as $t \to \infty$. Numerical evidence suggests global regularity holds in most practical cases, but turbulent behavior can emerge for large initial data.

Understand Dr. Zhou's statement: Regarding the assumption about the absence of a vortex, I cannot defini-

Heuristic Considerations and Energy Balance

Starting from the incompressibility condition:

$$
\begin{aligned}
&\operatorname{div}(\boldsymbol{u}) = 0 \\
&\iff \quad \rho \text{ is constant in } \Omega(t) \\
&\iff \quad \text{chain rule applies to } \mathbf{div}\big(\frac{2}{\rho}\mu(|\boldsymbol{D}|^2)\boldsymbol{D}\big) \\
\implies &\mathbf{div}\big(\frac{2}{\rho}\mu(|\boldsymbol{D}|^2)\boldsymbol{D}\big) = \nabla \cdot \big(\frac{2}{\rho}\mu(|\boldsymbol{D}|^2)\boldsymbol{D}\big) \\
&= \langle \frac{2}{\rho}\mu(|\boldsymbol{D}|^2)\boldsymbol{D}, \nabla \rangle \\
&= \frac{2}{\rho}\langle \mu(|\boldsymbol{D}|^2)\boldsymbol{D}, \nabla \rangle \\
&= \frac{2}{\rho}\nabla \cdot \big(\mu(|\boldsymbol{D}|^2)\boldsymbol{D}\big) \\
&\therefore \quad \frac{2}{\rho}\,\mathbf{div}\big(\mu(|\boldsymbol{D}|^2)\boldsymbol{D}\big) = 0.
\end{aligned}
$$

This vanishes if μ is constant (Newtonian fluid) and ρ is constant (incompressibility). So the diffusive term becomes:

$$\frac{2\eta}{\rho}\Delta \boldsymbol{u}, \quad \text{with } \eta = \mu\rho.$$

We heuristically compare dominant terms:

show simplification of diffusive term to laplacian

1. If $\eta\Delta \boldsymbol{u} \gg (\boldsymbol{u} \cdot \nabla)\boldsymbol{u}$, viscous dissipation dominates $\Rightarrow$ smooth, regular behavior.
2. If $(\boldsymbol{u} \cdot \nabla)\boldsymbol{u} \gg \eta\Delta \boldsymbol{u}$, nonlinearity dominates $\Rightarrow$ turbulence, potential blow-up.

We construct rigorous energy estimates in Section 3.

6 Appendix

References

[1] Luca Formaggia, Alfio Quarteroni, and Alessandro Veneziani. *Cardiovascular Mathematics. Modeling and Simulation of the Circulatory System*. Springer, 2009.

[2] Giovanni P. Galdi et al. *Hemodynamical Flows: Modeling, Analysis and Simulation*. Ed. by Unknown. Birkhäuser Basel, 2008. DOI: https://doi.org/10.1007/978-3-7643-7806-6.

[3] Add authors here. "Add exact title here". In: *European Heart Journal* (2021). DOI: 10.1093/eurheartj/ehab332. URL: https://watermark.silverchair.com/ehab332.pdf.

[4] L. John, P. Pustějovská, and O. Steinbach. "On the influence of the wall shear stress vector form on hemodynamic indicators". In: *Computing and Visualization in Science* 4 (2017), pp. 113–122. DOI: 10.1007/s00791-017-0277-7.

[5] 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 sumplementary code listings of computations done while investigating

Code 1: Algorithm 16.5

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