Modeling Blood Flow in Macrocirculatory Systems

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Notes follow pertaining to mathematical models of blood flow in the macrocirculatory systems. We develop mathematical models describing the hemodynamics of arterial and large venous segments. We then 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)*

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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 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
$\Delta = \nabla^2 = \nabla \cdot \nabla(\cdot)$	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 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 $\left[-\right]$

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2 Introduction

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 coronary artery stenosis are rudimentary; and often prediction doesn't mean coronary artery stenosis obstruction ([1]). 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 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 ω of the pulse wave to the blood's dynamic viscosity η 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) ≈ 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).

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 η: as H increases, η typically increases (cf. S6.5.1 [2]).

2.2 Modeling Blood Flow as a Continuum Fluid

A *domain* in an open, nontrivial, bounded, path-connected subset of $\mathbb{R}^N$ for $N \in \{1, 2, 3\}$. W.a.t. simulate the hemodynamics in a 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 $t \in [0, T]$ (s.t. $T > 0$ is the final time of interest). On occasion, we refer to the spatial-temporal domain $\Omega := \Omega(t) \times [0, T] \subset \mathbb{R}^{N+1}$ as our *computational domain*. We use the following field terminology on $\Omega(t)$:

$\phi : \Omega(t) \to \mathbb{R}$ 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*.

Continuum modeling framework. In *continuum mechanics*, fluids are modeled by continuous fields. At microscopic scales the continuum hypothesis breaks down, but at macroscopic scales such models are

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

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

We adopt the continuum hypothesis and treat blood (at large-vessel scales) as a single-constituent, homogeneous, isotropic fluid in $\Omega(t)$; ensuring that $\boldsymbol{u},\ p,$ and ρ are sufficiently smooth in space.

Definition 1. The material derivative of a physical quantity ϕ in a material element moving with velocity field $\boldsymbol{u}$ is

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

The literature also refers to D_t as the *substantial derivative*, *advective derivative*, *lagrangian derivative*, or *convective derivative*.

By our assumption that blood is homogenous, the material property ρ does not depend on position in $\Omega(t)$, i.e., $\rho \equiv \rho(t)$. Applying Newton's Laws, mass conservation and linear momentum balance read respectively:

$$\begin{cases} \partial_t\rho + \mathrm{div}(\rho\boldsymbol{u}) \ =\ 0, \\ \rho(\partial_t\boldsymbol{u} + (\boldsymbol{u}\cdot\nabla)\boldsymbol{u}) \ =\ \mathbf{div}(\boldsymbol{T}) + \rho\boldsymbol{f_b}, \end{cases} \qquad \text{in } \Omega(t)$$

where $\boldsymbol{T}$ is the Cauchy stress tensor, $\boldsymbol{f_b}$ is body force per unit mass $\left[\frac{m}{s^2}\right]$, and **div** acts rowwise on $\boldsymbol{T}$. Let the rate-of-deformation tensor be 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 continuum of fluid deforms locally under the velocity field $\boldsymbol{u}$.

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 λ the bulk viscosity.

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

Definition 5 (Incompressibile fluid)**.** A fluid is *incompressible* if its density is constant (in space and time), i.e. $\rho \equiv \rho_0 > 0$.

Definition 6 (Incompressible flow)**.** A flow is *incompressible* if density is materially constant (in space and time), i.e. $D_t\rho = 0$ in $\Omega(t)$ for all t.

Remark (Divergence-free condition under incompressibility). By assumption that blood is an incompressible fluid, it's density $\rho \equiv \rho_0 \in \mathbb{R}^+$. By the conservation of mass in an incompressible fluid volume, it may be shown that the fluid's flow is incompressible in the sense of Definition 6. Assuming incompressibility of the fluid and flow, we have the equivalences:

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

So under incompressibility of the fluid and flow, we obtain the *divergence-free condition* $\mathrm{div}(\boldsymbol{u}) = 0$ 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). We model blood as Newtonian and incompressibile. Throughout we assume the constanst *dynamic viscosity* $\eta \in \mathbb{R}^+$ and blood with constant denisity $\rho \equiv \rho_0 \in \mathbb{R}^+$. The

kinematic viscosity follows, defined as:

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

Here η quantifies the internal resistance of blood to shear deformation, i.e., $\eta := \frac{\tau}{\dot{\gamma}}$, with units $[Pa \cdot s]$. Moreover 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.

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 η:

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

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, η_r is often constant, justifying the Newtonian assumption. [[3], sec. 3.1]

2.3 Navier-Stokes System

Let $\boldsymbol{f}$ an external force acting on a continumm of blood fluid. When modeling non-Newtonian effects (when $\eta \neq$ constant), the kinematic viscosity $\mu(\cdot)$ is often chosen by Careau model [2]

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

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 [[3], pg. 38], 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 the Newtonian case, we choose $\eta = \eta_\infty$ which allows us to determine μ as $\mu(|\boldsymbol{D}|^2) = \eta$. When coupling our momentum balance equation with the divergence-free condition of $\boldsymbol{u}$, we obtain the Navier-Stokes (NS) equations.

Definition 8 (Conservative-Momentum Balance Form)**.**

$$\begin{cases} \partial_t(\rho \boldsymbol{u}) \;+\; (\rho \boldsymbol{u}\cdot\nabla)\boldsymbol{u} \;=\; -\nabla p \;+\; \mathbf{div}\big(2\,\eta\,\boldsymbol{D}(\boldsymbol{u})\big) \;+\; \rho\,\boldsymbol{f}, \\ \operatorname{div}(\boldsymbol{u}) \;=\; 0, \qquad \rho \equiv \rho_0 > 0 \text{ (constant)}. \end{cases}$$

Since $\rho \equiv \rho_0$ and $\eta = \mu|\boldsymbol{D}|^2$, the advective form follows from 8.

Definition 9 (Generalized-Newtonian Navier-Stokes (NS))**.**

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

We divide ρ and obtain the kinematic-viscosity form from 9.

Definition 10 (Kinematic-Viscosity Navier-Stokes (NS))**.**

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

Finally, we write the NS system in operator form.

Definition 11. We write Eq. 9 in standard form

$$\begin{cases} F\big(\partial_t \boldsymbol{u},\, \nabla \boldsymbol{u},\, \nabla p,\, \boldsymbol{u},\, p;\, \rho, \mu\big) \;=\; \boldsymbol{f}, \\ \operatorname{div}(\boldsymbol{u}) \;=\; 0, \end{cases}$$

where $\quad F\big(\partial_t \boldsymbol{u},\, \nabla \boldsymbol{u},\, \nabla p,\, \boldsymbol{u},\, p;\, \rho, \mu\big) \;:=\; \partial_t \boldsymbol{u} \;+\; (\boldsymbol{u}\cdot\nabla)\boldsymbol{u} \;+\; \dfrac{\nabla p}{\rho} \;-\; \mathbf{div}\Big(\dfrac{2}{\rho}\,\mu\,(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big)$

Remark. The NS equations are a non-linear coupled system of PDEs. The first equation follows from the balance of linear momentum, where the terms:

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

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

If pressure p and the velocity $\boldsymbol{u}$ are given, the Caucy stress $\boldsymbol{T}$ is computed from Eq. 7. 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 $\partial\Omega = [0,T] \times \Omega(t)$ be a surface in $\mathbb{R}^4$ 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

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

where $R(z,t)$ is the vessel radius at axial position z and time t.

Ref. notes for further details on deriving the transformation rules for vector calculus operators, or, notes where I do the derivation directly. Then discuss simplifications for axisymmetric flow.

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 extending our approach to bifurcations and arterial networks. Our strategy involves a *domain decomposition approach.*

We seek solutions to inital and boundary value problems of Eq. 9.

Definition 12. Let $\boldsymbol{u_0} : \Omega(0) \to R^3,\ \boldsymbol{x} \mapsto \boldsymbol{u_0}(x)\ :\ \boldsymbol{u}(0,\boldsymbol{x}) = \boldsymbol{u_0}$. We refer to $\boldsymbol{u_0}$ as the initial condition of velocity feild $\boldsymbol{u}$

Definition 13. Let $\boldsymbol{u^0}(t)$ and $\boldsymbol{u^1}(t)$ be the velocity feilds at S_0 and S_1.

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

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 9 by imposing further simplifying assumptions. Namely, w.a.t. compute average pressures and velocities after a relatively short simulation time by solving the 1D NS system in a compliant vessel with suitable side conditions. Because our model averages pressure and velocity over a surface-area, we obtain a uniform distribution of WSS on the vessel.

One may start by introducing a rigid-vessel assumption, which leads to a no slip condition that $\boldsymbol{u}\big|_{\partial\Omega} = \boldsymbol{0}$. Instead we seek to model the link between blood flow and the deformation of the vessel wall. We begin our derivation of Dimension-Reduced models by assuming the following transformation exists of our fluid domain boundary $\Omega(t)$ to a simplified geometry.

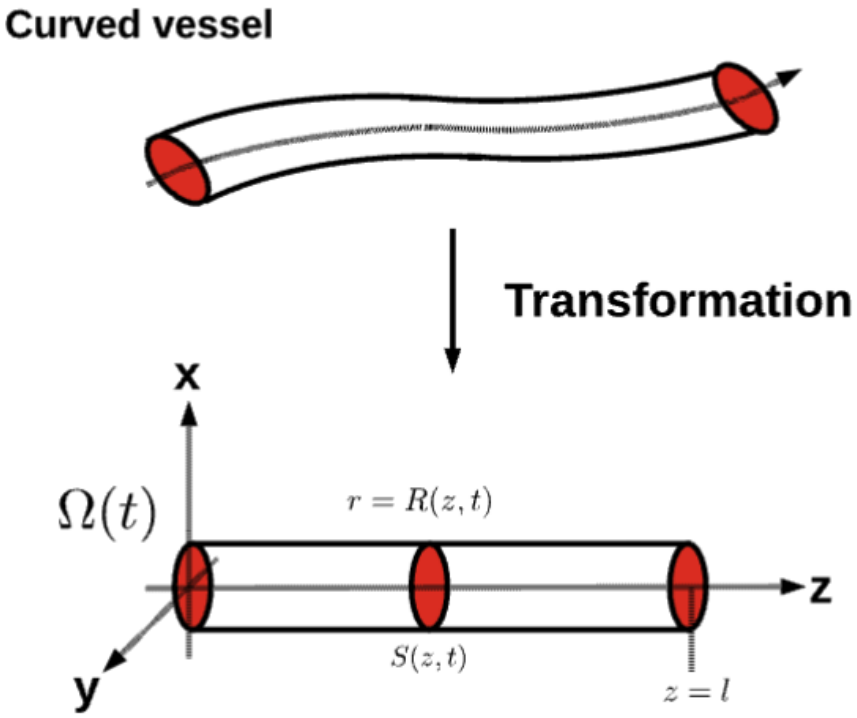

Figure 1: From [3] [Fig. 3.2, pg. 37]

We consider the fluid dynamics of the following fluid element contained in a portion of the lumen $\Omega(t)$ Let

Fig. 3.3 Notation describing the different parts of the vessel portion

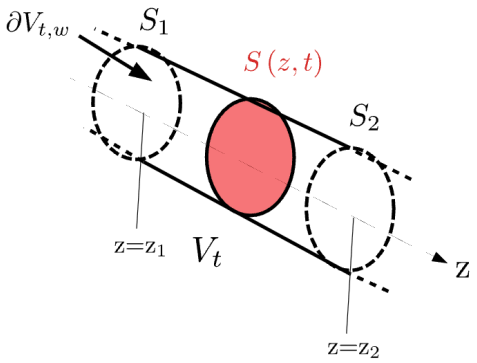

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Figure 2: From [3] [Fig. 3.3, pg. XX]

$S_1(t),\ S_2(t)$ be the time-dependent shaded boundaries at $z = z_1$ and $z = z_2$ s.t. $0 < z_1 < z_2 < \ell$. Let V_t be the fluid element of blood. The boundary of the fluid element is $\partial V_t = S_1(t) \cup S_2(t) \cup \partial V_{t,w}$ such that $\partial V_{t,w}$ is the vessel wall in contact with the fluid element. According to the Reynold's transport theorem for scalar feild $\phi \in L^1(\Omega(t))$.

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

where $\boldsymbol{u_b}$ is the velocity feild deforming the boundary ∂V_t (Pf. see wiki). If we assume the normal component of $\boldsymbol{u_b} = \boldsymbol{0}$ 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 ∂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

$$\begin{aligned}\int_{\partial V_t} (\boldsymbol{u_b}\cdot\hat{\boldsymbol{n}})\phi\, dS \;&=\; \int_{\partial V_t} (\boldsymbol{u_w}\cdot\hat{\boldsymbol{n}})\phi\, dS \\ &=\; \int_{\partial V_t} (\boldsymbol{w}\cdot\hat{\boldsymbol{n}})\phi\, dS \;+\; \int_{\partial V_t} (\boldsymbol{u}\cdot\hat{\boldsymbol{n}})\phi\, dS\end{aligned}$$

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

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

Now we may rewrite the volume integral in the LHS of RT theorem

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

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

$$\int_{V_t} \frac{\partial \phi}{\partial t} dV \;=\; \int_{z_1}^{z_2} \frac{\partial}{\partial t}\left[A \cdot \bar{\phi}\right] dz,$$

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

$$\int_{\partial V_t} (\boldsymbol{u_b} \cdot \hat{\boldsymbol{n}}) \phi dS = \int_{\partial V_t} (\boldsymbol{u_b} \cdot \hat{\boldsymbol{n}}) \phi dS....$$

With a little more work, one may obtain:

cleanup, ref. pg. 43

Definition 14. The 1D Reynolds Transport theorem for both compressible and incompressible fluids:

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

Remark. By taking $f = \rho$ in 14, 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 14 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 14, momentum conservation follows directly. Also, by our assumption that blood is incompressible, we simplify 14 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.

3.1.1 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

W.r.t. numerical methods for PDEs as *schemes*. To ensure converge of our schemes, we choose an initial condition by solving the homogenous-stationary NS problem in form 10 with $\boldsymbol{f} \equiv \boldsymbol{0}$:

Definition 15 (Stationary Stokes Problem)**.**

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

Let

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

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

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 9 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.

Heuristic Considerations and Energy Balance

Starting from the incompressibility condition:

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

Understand Dr. Zhou's statement: Regarding the assumption about the absence of a vortex, I cannot definitively say whether it is correct or not. Please review the references provided. If needed, we can discuss

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] Francois Derimay, Gerard Finet, and Gilles Rioufol. "Coronary Artery Stenosis Prediction Does Not Mean Coronary Artery Stenosis Obstruction". In: *European Heart Journal* (2021). DOI: 10.1093/eurheartj/ehab332. URL: https://watermark.silverchair.com/ehab332.pdf.

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

[3] Tobias Köppl and Rainer Helmig. *Dimension Reduced Modeling of Blood Flow in Large Arteries. An Introduction for Master Students and First Year Doctoral Students*. Springer Nature Switzerland, 2023.

[4] 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). 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
```