Blood Flow in the Human Circulatory System

Daniel Henderson

Michigan Technological University

November 15, 2025

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 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

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

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 $\left[-\right]$
c	concentration of a material element
D	diffusion coefficient $\left[\frac{m^2}{s}\right]$
t	time $\left[s\right]$
T	terminal time $\left[s\right], t > 0$
ω	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

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 $\left[-\right]$
c	concentration of a material element
D	diffusion coefficient $\left[\frac{m^2}{s}\right]$
t	time $\left[s\right]$
T	terminal time $\left[s\right], t > 0$
ω	angular frequency $\left[\frac{rad}{s}\right]$
$\boldsymbol{f_b}$	body force per unit volume $\left[\frac{N}{m^3}\right]$

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. 2.5 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 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]). 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 ρ is sufficiently smooth in Ω.

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 ϕ 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 ϕ 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 ϕ 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 Ω 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 λ 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 Ω.

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 η quantifies the internal resistance of blood to shear deformation, i.e., $\eta := \dfrac{\tau}{\dot{\gamma}}$, 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 Blood Model

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. 15.

Definition 8. 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 9. 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.

2.4 Dimension-Reduced Models of Blood Flow

We start our disscusion with 1D and 0D models, reducing the d.o.f. in the NS system 15 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.

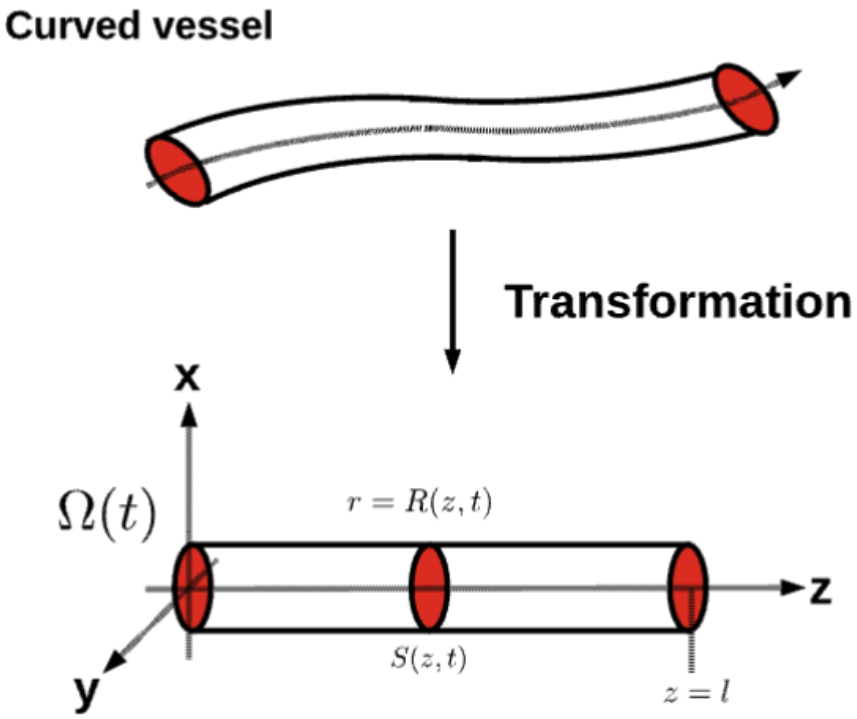
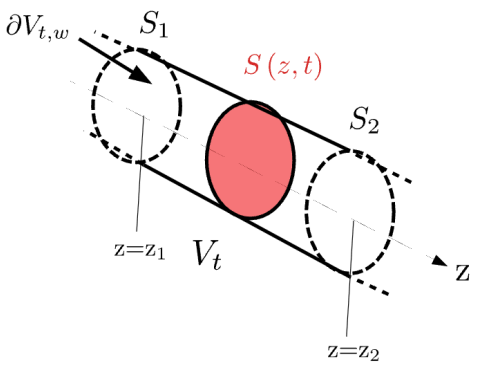


Figure 1: From [3] [Fig. 3.2, pg. 37]

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



‘’

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 10. 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 10, 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 10 as

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

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

complete, pg. 45

Remark. By taking $f = u_3$ in 10, momentum conservation follows directly. Also, by our assumption that blood is incompressible, we simplify 10 as

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

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

Remark (Tube law from a thin elastic cylindrical wall). We briefly justify the pressure–area relation used in aq_1d_compliant.jl

Definition 11.

$$p(A) - p_{ext} = \beta\big(\sqrt{A} - \sqrt{A_0}\big)$$

used in the 1D (A, Q) model. Consider a straight cylindrical vessel with (local) lumen radius $R(x,t)$, reference radius R_0, wall thickness $h \ll R$, and internal pressure $p(x,t)$ relative to an external pressure p_{ext} (assumed constant in space and time for simplicity). The corresponding lumen area is

$$A(x,t) = \pi R(x,t)^2, \qquad A_0 = \pi R_0^2.$$

Under the thin–wall assumption, balance of forces in the circumferential direction (Young–Laplace law) yields

Definition 12.

$$(p - p_{ext})\, 2\pi R = \sigma_\theta\, 2h\pi$$

where σ_θ is the circumferential (hoop) Cauchy stress in the vessel wall. We model the wall as linearly elastic in the hoop direction, so that

Definition 13.

$$\sigma_\theta = E_{eff}\, \varepsilon_\theta$$

with an effective circumferential modulus $E_{eff} > 0$ and circumferential strain

$$\varepsilon_\theta \;=\; \frac{\text{change in circumference} - \text{reference circumference}}{\text{reference circumference}} \qquad = \; \frac{2\pi R - 2\pi R_0}{2\pi R_0} \qquad = \; \frac{R - R_0}{R_0}.$$

Equating (12) and (13) gives

$$\begin{aligned}
(p - p_{ext})\, 2\pi R \;&=\; E_{eff}\, \frac{R - R_0}{R_0}\, 2h\pi \\
\iff (p - p_{ext})\, R \;&=\; E_{eff}\, \frac{R - R_0}{R_0}\, h
\end{aligned}$$

Express R and R_0 in terms of the areas A and A_0:

$$\begin{aligned}
R = \sqrt{\frac{A}{\pi}} \\
&= \frac{\sqrt{A}}{\sqrt{\pi}}, \\
R_0 = \sqrt{\frac{A_0}{\pi}} \quad &= \frac{\sqrt{A_0}}{\sqrt{\pi}},
\end{aligned}$$

so that

$$\begin{aligned}
R - R_0 \;&=\; \frac{\sqrt{A}}{\sqrt{\pi}} - \frac{\sqrt{A_0}}{\sqrt{\pi}} \\
&=\; \frac{1}{\sqrt{\pi}}\big(\sqrt{A} - \sqrt{A_0}\big).
\end{aligned}$$

Substituting into the expression for $p - p_{ext}$, we obtain

$$\begin{aligned}
(p - p_{ext})\, \frac{\sqrt{A}}{\sqrt{\pi}} \;&=\; E_{eff} h\, \frac{\frac{1}{\sqrt{\pi}}\big(\sqrt{A} - \sqrt{A_0}\big)}{R_0} \\
\iff (p - p_{ext})\, \frac{\sqrt{A}}{\sqrt{\pi}} \;&=\; E_{eff} h\, \frac{\frac{1}{\sqrt{\pi}}\big(\sqrt{A} - \sqrt{A_0}\big)}{R_0}.
\end{aligned}$$

For moderate deformations where A remains close to A_0, we approximate the factor $1/\sqrt{A}$ by its reference value $1/\sqrt{A_0}$, which yields

$$\begin{aligned}
(p - p_{ext})\, \frac{\sqrt{A_0}}{\sqrt{\pi}} \;&=\; E_{eff} h\, \frac{\frac{1}{\sqrt{\pi}}\big(\sqrt{A} - \sqrt{A_0}\big)}{R_0} \\
\iff (p - p_{ext}) \;&=\; \frac{E_{eff} h}{R_0 \sqrt{A_0}}\, \big(\sqrt{A} - \sqrt{A_0}\big).
\end{aligned}$$

Defining the lumped stiffness parameter

$$\beta \;:=\; \frac{E_{eff}h}{R_0\sqrt{A_0}}.$$

we arrive at the tube law (**??**) used in the 1D model:

$$p(A) - p_{ext} \;=\; \beta\big(\sqrt{A} - \sqrt{A_0}\big).$$

In the numerical experiments below, we take β and A_0 to be constant along the vessel, so that p can be written as a function of A alone.

2.4.1 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.

2.5 Navier-Stokes

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]

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

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 [[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 14 (Conservative-Momentum Balance Form)**.**

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

Since $\rho \equiv \rho_0$ and $\eta = \mu|\boldsymbol{D}|^2$, the advective form follows from 14.

Definition 15 (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 15.

Definition 16 (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 17. We write Eq. 15 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 $F\big(\partial_t \boldsymbol{u},\, \nabla\boldsymbol{u},\, \nabla p,\, \boldsymbol{u},\, p;\, \rho, \mu\big) \;:=\; \partial_t \boldsymbol{u} \;+\; (\boldsymbol{u}\cdot\nabla)\boldsymbol{u} \;+\; \dfrac{\nabla p}{\rho} \;-\; \mathbf{div}\Big(\dfrac{2}{\rho}\,\mu\,(|\boldsymbol{D}|^2)\,\boldsymbol{D}\Big)$

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 Cauchy stress $\boldsymbol{T}$ is computed from Eq. 7. It follows that 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 referred to as fluid-structure interaction (FSI) models.

discuss in a later section

2.5.1 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 Appendix

Bibliography

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.

Code Listings

Code listings

Code 1: Algorithm 16.5

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