MA621L-Mathematical Modelling

Model-4 : Navier-Stokes Equation - Blood Flow Model

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Blood Flow Dynamics

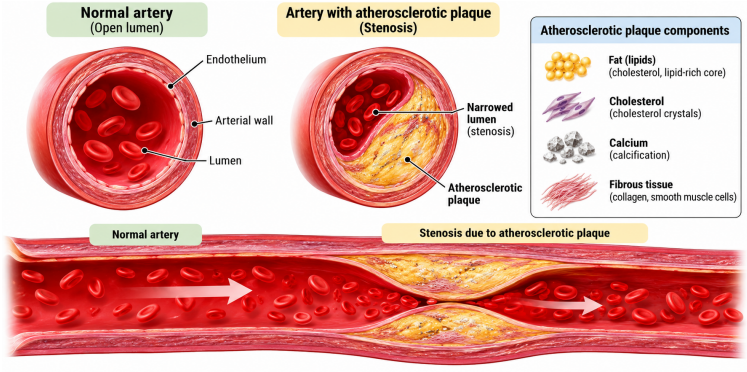
What Is a Stenosis?

A **stenosis** is a local narrowing of the lumen, usually caused by an atherosclerotic plaque (fat, cholesterol, calcium, fibrous tissue) growing into the vessel wall.

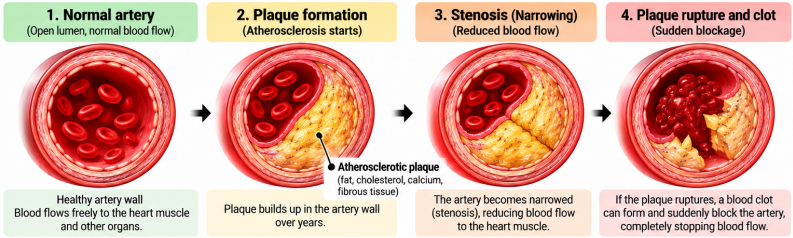


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- Severity is reported through the minimum lumen diameter d and the healthy reference diameter D:

$$\%\,\text{diameter} = \left(1 - \frac{d}{D}\right) \times 100, \qquad \%\,\text{area} = \left(1 - \frac{d^2}{D^2}\right) \times 100$$

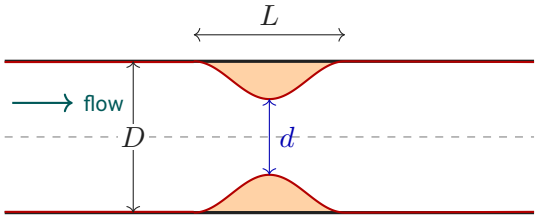
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% diameter	% area	Throat velocity	Resistance
30%	51%	$\times 2$	$\times 4$
50%	75%	$\times 4$	$\times 16$
70%	91%	$\times 11$	$\times 123$
90%	99%	$\times 100$	$\times 10^4$

Stenosis Geometry: What We Must Describe



A common idealised (axisymmetric, cosine) profile with peak plaque height $\delta = (D - d)/2$:

$$R(x) = \begin{cases} \dfrac{D}{2} - \dfrac{\delta}{2}\left(1 + \cos\dfrac{2\pi x}{L}\right), & |x| \le L/2, \\ \dfrac{D}{2}, & \text{otherwise.} \end{cases}$$

Other shape descriptors: eccentricity (off-axis plaque), entry and exit angles, lesions in series.

The Modelling Route for Today

#	Key word	Dym's question	Blood flow
1	Why?	What are we looking for?	Which stenoses matter
2	Find?	What do we want to know?	$\mathbf{u}$, p, ρ and FFR
3	Given?	What do we know?	Physiology, data, laws
4	Assume?	What can we assume?	Assumption ledger A1–A10
5	How?	How should we look at this model?	Moving parcel $\rightarrow$ Navier–Stokes
6	Predict?	What will our model predict?	Regimes, jet, resistance
7	Valid?	Are the predictions valid?	Units, limits, ledger check
8	Verified?	Are the predictions good?	Benchmarks, patient data
9	Improve?	Can we improve the model?	Lift assumptions one at a time
10	Use?	How will we exercise the model?	Diagnosis, surgery, devices



Principle 1 — Why? What are we looking for?

Why Simulate Blood Flow



- Cardiovascular disease is the leading cause of death worldwide, accounting for approximately 17.9 million deaths per year (WHO, 2023).
- A central challenge in cardiology is identifying which arterial narrowings — stenoses — are haemodynamically significant enough to cause ischaemia and require intervention such as stenting or bypass surgery.

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- A central challenge in cardiology is identifying which arterial narrowings — stenoses — are haemodynamically significant enough to cause ischaemia and require intervention such as stenting or bypass surgery.

The modelling question. Given the geometry of an artery with a stenosis, is the narrowing severe enough to starve the tissue downstream of blood?

Why Simulate Blood Flow

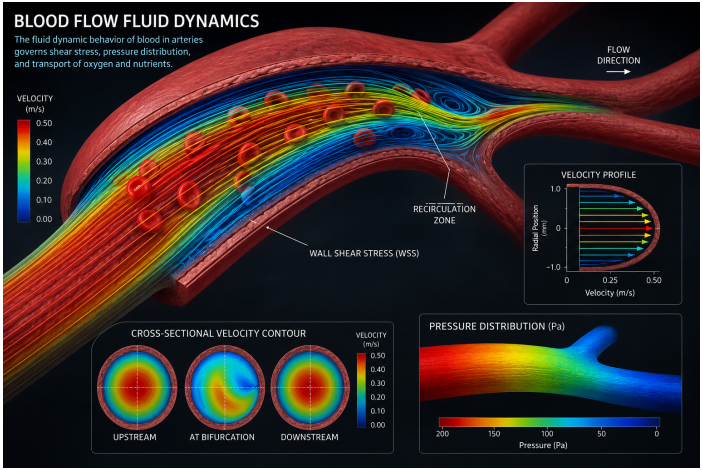


Figure 1: Blood Flow Fluid Dynamics



Principle 2 — Find? What do we want to know?

What we want to compute:

$\mathbf{u}(\mathbf{x}, \mathbf{t})$

Velocity field — how fast and in which direction blood moves

$\mathbf{p}(\mathbf{x}, \mathbf{t})$

Pressure field — the driving force; FFR comes from this

$\boldsymbol{\rho}(\boldsymbol{x}, \boldsymbol{t})$

Density field — mass per unit volume; captures compressibility and variation in blood properties

From Fields to a Clinical Number

- The fields $\mathbf{u}$, p, ρ are what the model computes.



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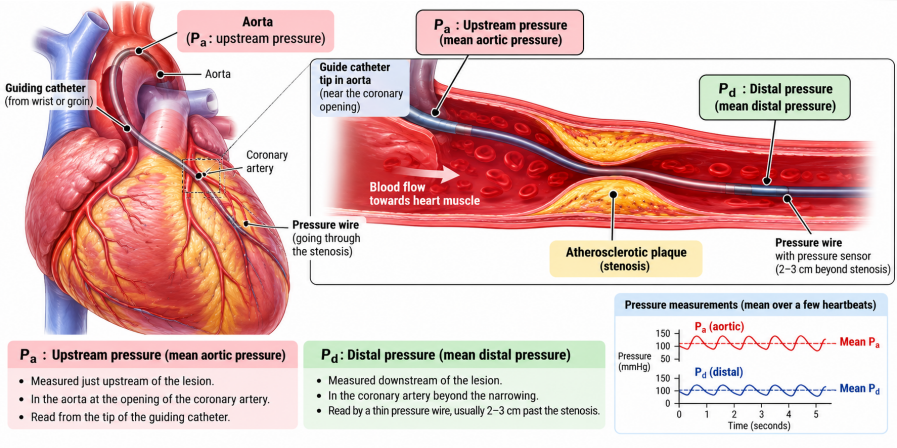
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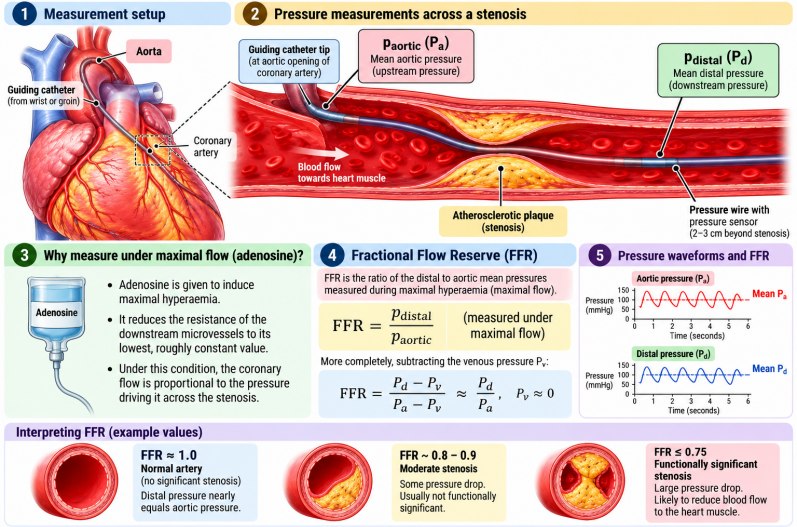
- $\mathrm{FFR} \le 0.80$ is the usual threshold for a significant stenosis.
- A second output, the wall shear stress $\tau_w = \mu\, \partial u/\partial n \big|_{\text{wall}}$, is used for plaque and aneurysm risk.

Find: $\mathbf{u}(\mathbf{x}, t)$ and $p(\mathbf{x}, t)$ in the artery, and from them FFR and τ_w.

From Fields to a Clinical Number



From Fields to a Clinical Number



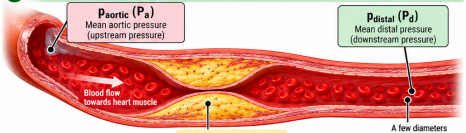
From Fields to a Clinical Number

1 Concept: FFR as a flow ratio

$$\text{FFR} \approx \frac{\text{max flow with stenosis}}{\text{max flow if healthy}}$$

- FFR estimates flow, not just pressure.
- Measured under maximal hyperaemia (e.g., adenosine).
- An FFR of 0.75 means the lesion allows only 75% of the flow the heart muscle could get.
- FFR ≤ 0.80 is usually considered functionally significant and treated.

2 Pressure measurement in a coronary artery



3 Why measure under maximal flow?



- Adenosine induces maximal hyperaemia.
- It reduces the resistance of the downstream microvessels to its lowest, roughly constant value.
- Under this condition, flow is proportional to the pressure driving it across the stenosis.

4 Definition of FFR

$$\text{FFR} = \frac{P_{distal}}{P_{aortic}} \quad \text{(measured under maximal flow)}$$

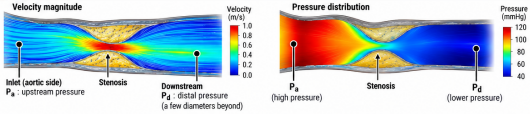
Including venous pressure P_v:

$$\text{FFR} = \frac{P_d - P_v}{P_a - P_v} \approx \frac{P_d}{P_a}, \quad P_v \approx 0$$

5 Interpreting FFR

FFR value	Interpretation
≈ 1.0	**Normal artery** (no significant stenosis) Flow with stenosis ≈ flow if healthy
0.80 – 0.90	**Mild to moderate stenosis** Usually not functionally significant
≤ 0.80	**Functionally significant stenosis** Reduced flow to heart muscle Usually considered for treatment
0.75 (example)	Only 75% of the maximal flow the heart muscle could get

6 Example from CFD / CT-FFR simulation



Computed FFR (CT-FFR)

P_a = 100 mmHg
P_d = 75 mmHg

$$\text{FFR} = \frac{P_d}{P_a} = \frac{75}{100} = 0.75$$

- FFR = 0.75 means the lesion allows only 75% of the maximal flow.
- This is below the 0.80 threshold for treatment.
- This is exactly how CT-FFR software reports it, using computed pressures just before and after the narrowing.



Principle 3 — Given? What do we know?

How Blood Flows?

Assume that you are an Ant-Man.



Blood Flow 1
Blood Flow 2

How Blood Flows?

Imagine you are standing inside a human artery, roughly the size of a garden hose, watching blood rush past at nearly a metre per second.



How Blood Flows?

- Red blood cells surge past me like spinning discs—millions of them—tumbling, colliding, drifting in organized chaos.



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- Do they flow quietly?



How Blood Flows?

- Red blood cells surge past me like spinning discs—millions of them—tumbling, colliding, drifting in organized chaos.
- Do they flow quietly?
- No, they rush, a living river moving at nearly a metre per second.
- Brace yourself as the current threatens to pull me along.



How Blood Flows?

- What is your blood pressure?



How Blood Flows?

- What is your blood pressure?
- How does the doctor measure it?



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- What is Systolic and What is Diastolic?

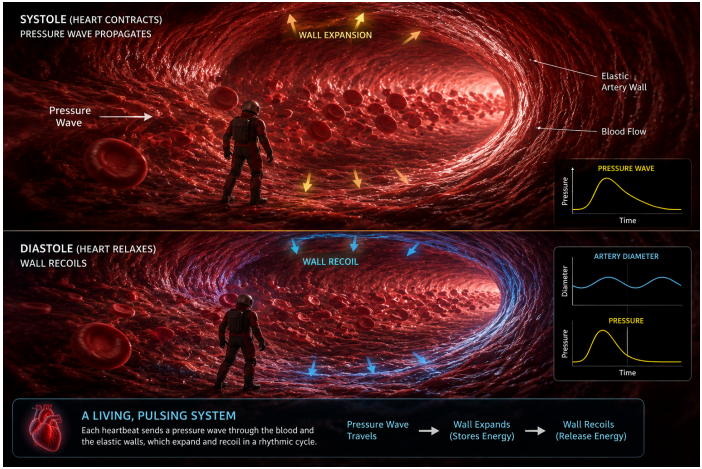


How Blood Flows?



- What is your blood pressure?
- How does the doctor measure it?
- 120/80 mmHg, what is 120 and what is 80?
- What is Systolic and What is Diastolic?
- 120 - Systolic (maximum force against artery walls when the heart beats) measures the pressure when the heart contracts and pushes blood out
- 80 - Diastolic (pressure in the arteries while the heart rests between beats) measures the pressure when the heart relaxes and refills with blood

How Blood Flows?



How Blood Flows?

Let another pulse hit. What happens now?



How Blood Flows?



Let another pulse hit. What happens now?

- The entire artery wall expands outward, then recoils.
- It's not rigid—it breathes.
- You can feel the pressure wave from the heartbeat traveling through the fluid and the elastic walls, like standing inside a rhythmic, biological engine.

How Blood Flows?

What is happening now?



How Blood Flows?



What is happening now?

- The wide passage suddenly tightens

How Blood Flows?



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- A fatty deposit has narrowed the artery, cutting the channel almost in half
- As you move closer, the flow transforms
- The blood accelerates sharply, squeezing through the constriction like a river forced through a canyon.
- The smooth motion breaks, eddies begin to form, and subtle instabilities creep into the stream.

Blood Physical Properties

$$\rho = 1060\ \mathrm{kg/m^3}, \quad \mu = 3.5 \times 10^{-3}\ \mathrm{Pa{\cdot}s}, \quad \nu = \mu/\rho = 3.3 \times 10^{-6}\ \mathrm{m^2/s}$$



Given: What the Ant-Man Has Told Us



Observations

- Flow is pulsatile: 120 mmHg (≈ 16 kPa) in systole, 80 mmHg in diastole
- Pressure falls from $\sim$120 mmHg at the aortic root to $\sim$10 mmHg in capillaries
- Walls are elastic; they expand and recoil with every beat
- A stenosis narrows the lumen; flow accelerates and eddies form behind it
- Blood is a suspension of red cells in plasma

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Laws we trust

- Newton's second law: $F = ma$
- Conservation of mass



Principle 4 — Assume? What can we assume?

Every Observation: Keep It or Drop It

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Every Observation: Keep It or Drop It



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- We cannot carry all of it into the first model.
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An unstated assumption costs more than an arithmetic error.
Every assumption must be liftable on its own.

How Blood Flows?

- Let us assume ourselves as an imaginary cube in place of Ant-Man now, for simplification.



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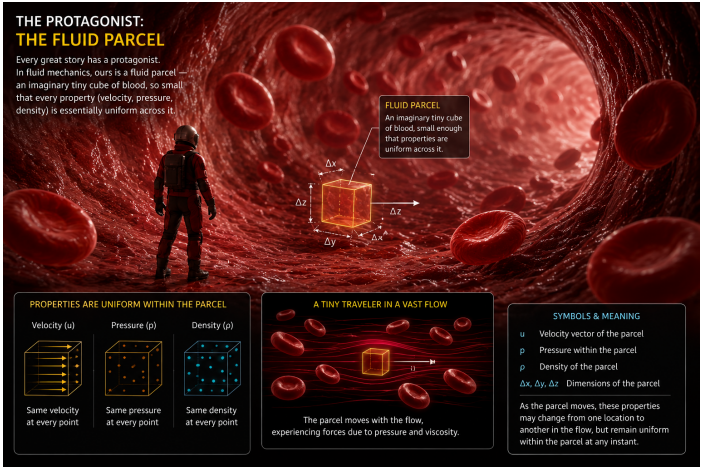
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- This small cube carries all the properties such as velocity, pressure, and density
- They are uniform across this cube.
- We will follow this parcel as it travels through the artery: squeezing through the stenosis, spinning in the recirculation zone, scraping along the vessel wall

How Blood Flows?



Assumption Ledger (1/2)

No.	Assumption	What we drop
A1	Continuum: the tiny cube carries uniform $\mathbf{u}$, p, ρ	Individual molecules and cells
A2	Single-phase, homogeneous fluid	Red cells as separate particles
A3	Newtonian: $\tau = \mu\,\partial u/\partial y$, μ constant	Shear-thinning of blood
A4	Incompressible: ρ constant	Density variation
A5	Rigid wall	The breathing wall we just felt

Assumption Ledger (2/2)

No.	Assumption	What we drop
A6	No-slip: blood at the wall is at rest	Slip at the endothelium
A7	Laminar flow	Turbulence
A8	Body forces negligible, $\mathbf{f} \approx 0$	Gravity, posture
A9	Isothermal	Temperature dependence of μ, ρ
A10	Derivation in 1D: $u(x,t)$ along x, shear across y; generalised to 3D at the end	Full 3D geometry during the derivation

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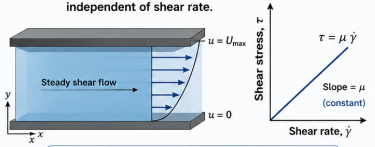
A5 contradicts what we observed in Principle 3. That is allowed — as long as we say so, and come back to it in Principle 9.

A3: Is Blood Newtonian?

NEWTONIAN FLUID vs NON-NEWTONIAN FLUID

NEWTONIAN FLUID

Viscosity is **constant** independent of shear rate.

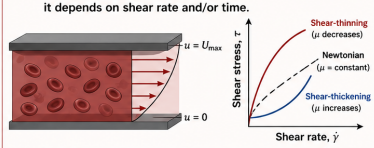


Newton's law of viscosity: $\tau = \mu\,\dot{\gamma}$ where μ = constant

Examples: Water, Air, Glycerin, many simple oils

NON-NEWTONIAN FLUID

Viscosity is **not constant** it depends on shear rate and/or time.



Generalized relation: $\tau = \mu(\dot{\gamma})\,\dot{\gamma}$ where μ depends on shear rate $\dot{\gamma}$

Examples: Blood, Paints, Ketchup, Toothpaste, Polymer solutions

NEWTONIAN vs NON-NEWTONIAN FLUID – AT A GLANCE

Feature	Newtonian Fluid	Non-Newtonian Fluid
Viscosity (μ)	Constant (independent of shear rate and time)	Variable (depends on shear rate and/or time)
Shear Stress–Shear Rate Relationship	Linear ($\tau \propto \dot{\gamma}$)	Non-linear (τ not directly proportional to $\dot{\gamma}$)
$f(x)$ Constitutive Equation	$\tau = \mu\,\dot{\gamma}$ (μ = constant)	$\tau = \mu(\dot{\gamma})\,\dot{\gamma}$ (μ = variable)
Flow Behavior	Predictable and uniform	Complex (shear-thinning, shear-thickening, yield stress, thixotropy, etc.)
Examples	Water, Air, Glycerin, Many oils	Blood, Paints, Ketchup, Yogurt, Toothpaste, Slurries, Polymer solutions
Typical Occurrence	Simple molecular fluids	Complex fluids with particles, polymers, or microstructure
Modeling in Navier–Stokes	Use $\mu\nabla^2 u$	Use $\nabla \cdot \left[\mu(\dot{\gamma})\,(\nabla u + \nabla u^T)\right]$



Principle 5 — How? How should we look at this model?

How We Will Look at It



- Follow one fluid parcel (A1) and apply Newton’s second law to it.
- Identify every force on the parcel: pressure, viscous, body.
- Add conservation of mass.
- Close the system with initial and boundary conditions.
- Rewrite in dimensionless form to see which ratios control the flow.

Newton's Second Law



$$F = ma$$

Newton's Second Law



$$F = ma$$

- F: Force
- m: mass
- a: Acceleration

How Blood Flows?

- What is our velocity field?



How Blood Flows?

- What is our velocity field? $u(x,t)$



How Blood Flows?

- What is our velocity field? $u(x,t)$
- Then what is our acceleration?



How Blood Flows?



- What is our velocity field? $u(x,t)$
- Then what is our acceleration? u_x or u_t or both?

How Blood Flows?



- What is our velocity field? $u(x,t)$
- Then what is our acceleration? u_x or u_t or both?
- Let's assume the fluid parcel stays at the same location

Therefore,

$$F = m\frac{\partial u}{\partial t}$$

How Blood Flows?



- What is mass?

$$m = \rho \, dV$$

  where ρ denotes the density of the fluid and dV is the tiny volume of the imaginary cube.

- Also, let us calculate the Force per unit volume, then

Therefore,

$$F = \rho \, dV \frac{\partial u}{\partial t} \implies \frac{F}{dV} = \rho \frac{\partial u}{\partial t}$$

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- How does velocity change along the path of the particle?

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$$dx = u\,dt \implies \frac{dx}{dt} = u$$

- How does velocity change at a point?

$$u = u(x(t), t)$$

- How does velocity change along the path of the particle? Using the chain rule

$$\frac{du(x(t),t)}{dt} = \frac{\partial u}{\partial t} + \frac{dx}{dt}\frac{\partial u}{\partial x} = \frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x}$$

How Blood Flows?

$$\frac{du(x(t),t)}{dt} = \frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x}$$

- Flow itself changes with time $\frac{\partial u}{\partial t}$
- You are moving into a new region where the velocity is different. Your speed u
- Spatial gradient $\frac{\partial u}{\partial x}$
- Rate of change due to motion = speed $\times$ spatial change = $u\frac{\partial u}{\partial x}$

How Blood Flows?

$$\frac{F}{dV} = \rho \left(\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} \right)$$

How Blood Flows?



$$\frac{F}{dV} = \rho \left(\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} \right)$$

- Where does the force come from?

How Blood Flows?



$$\frac{F}{dV} = \rho \left(\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} \right)$$

- Where does the force come from?
- For a fluid parcel, forces don’t come from inside—they act on the surface

Heart's Gift: Pressure Forces



- The heart creates high pressure at the aortic root ($\sim$120 mmHg) and lower pressure in the capillaries ($\sim$10 mmHg).

Heart's Gift: Pressure Forces



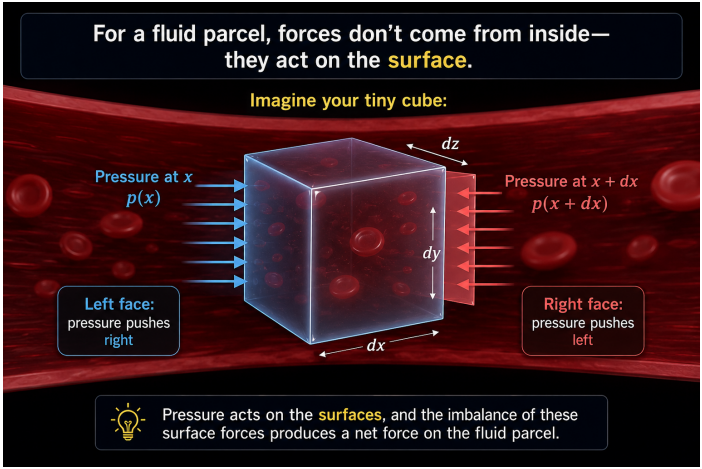
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- This pressure difference pushes blood from high to low, just as water flows downhill.
- On our tiny cube, pressure acts on all six faces.
- The net force is the difference between opposite faces — NOT the pressure itself, but its GRADIENT.

Heart's Gift: Pressure Forces



Heart's Gift: Pressure Forces



At position x

- Left Face Force

Heart's Gift: Pressure Forces



At position x

- Left Face Force $p(x)A$

Heart's Gift: Pressure Forces



At position x

- Left Face Force $p(x)A$
- Right Face Force

Heart's Gift: Pressure Forces



At position x

- Left Face Force $p(x)A$
- Right Face Force $p(x+dx)A$

Heart's Gift: Pressure Forces



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- Net Force

$$F = p(x)A - p(x+dx)A$$

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$$p(x+dx) \approx p(x) + \frac{\partial p}{\partial x} dx$$

$$F = -\frac{\partial p}{\partial x} A\, dx \implies F = -\frac{\partial p}{\partial x} dV$$

Heart's Gift: Pressure Forces

$$\frac{F}{dV} = \rho \left(\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} \right)$$

Heart's Gift: Pressure Forces



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Heart's Gift: Pressure Forces

$$\rho\left(\frac{\partial u}{\partial t}+u\frac{\partial u}{\partial x}\right)=-\frac{\partial p}{\partial x}$$

- Standing inside the artery, I feel pushes from all sides.
- But they are not equal.
- The pressure behind me is slightly higher than the pressure ahead, and that tiny imbalance is enough to accelerate the flow.

Flow moves from high pressure to low pressure

Blood's Resistance to Shear

- Pour honey onto a table, then pour water. Which spreads slowly? Why?



Blood's Resistance to Shear



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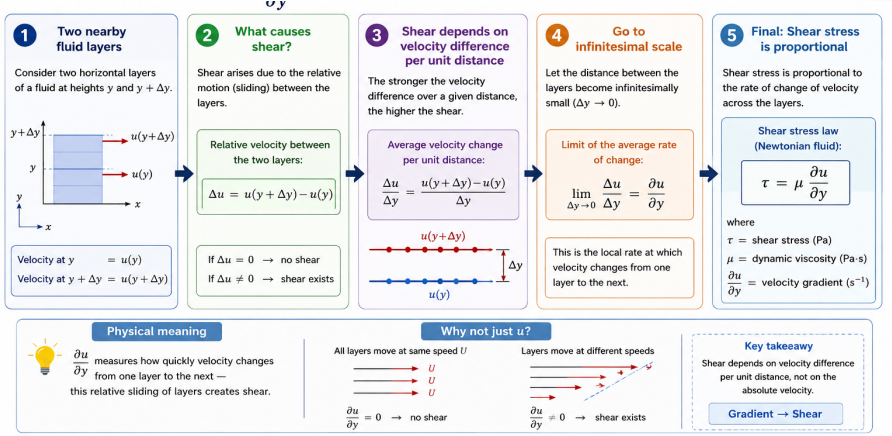
Blood's Resistance to Shear



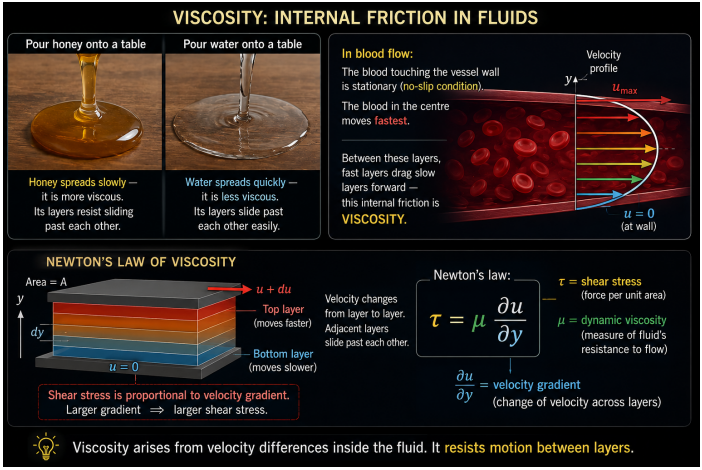
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- Between these layers, fast layers drag slow layers forward — this internal friction is VISCOSITY.

Newton's law: shear stress = viscosity $\times$ velocity gradient (A3)

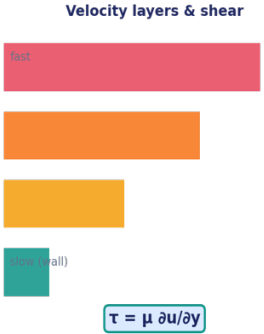
Viscosity

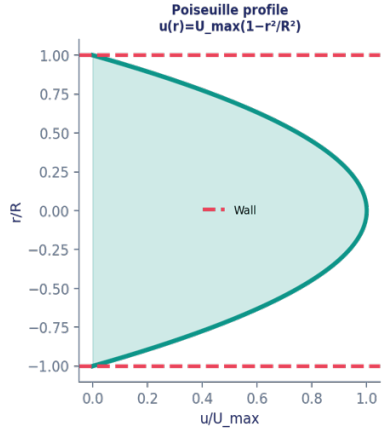

1 Two nearby fluid layers
Consider two horizontal layers of a fluid at heights y and $y + \Delta y$.
$y+\Delta y$
y
$u(y+\Delta y)$
$u(y)$
Velocity at y $= u(y)$
Velocity at $y + \Delta y$ $= u(y+\Delta y)$
2 What causes shear?
Shear arises due to the relative motion (sliding) between the layers.
Relative velocity between the two layers:
$\Delta u = u(y+\Delta y) - u(y)$
If $\Delta u = 0$ → no shear
If $\Delta u \neq 0$ → shear exists
3 Shear depends on velocity difference per unit distance
The stronger the velocity difference over a given distance, the higher the shear.
Average velocity change per unit distance:
$\frac{\Delta u}{\Delta y} = \frac{u(y+\Delta y) - u(y)}{\Delta y}$
$u(y+\Delta y)$
Δy
$u(y)$
4 Go to infinitesimal scale
Let the distance between the layers become infinitesimally small ($\Delta y \to 0$).
Limit of the average rate of change:
$\lim_{\Delta y \to 0} \frac{\Delta u}{\Delta y} = \frac{\partial u}{\partial y}$
This is the local rate at which velocity changes from one layer to the next.
5 Final: Shear stress is proportional
Shear stress is proportional to the rate of change of velocity across the layers.
Shear stress law (Newtonian fluid):
$\tau = \mu \frac{\partial u}{\partial y}$
where
τ = shear stress (Pa)
μ = dynamic viscosity (Pa·s)
$\frac{\partial u}{\partial y}$ = velocity gradient (s^{-1})
Physical meaning
$\frac{\partial u}{\partial y}$ measures how quickly velocity changes from one layer to the next — this relative sliding of layers creates shear.
Why not just u?
All layers move at same speed U
U
U
U
$\frac{\partial u}{\partial y} = 0$ → no shear
Layers move at different speeds
$\frac{\partial u}{\partial y} \neq 0$ → shear exists
Key takeaway
Shear depends on velocity difference per unit distance, not on the absolute velocity.
Gradient → Shear

Viscosity


VISCOSITY: INTERNAL FRICTION IN FLUIDS
Pour honey onto a table
Pour water onto a table
Honey spreads slowly — it is more viscous. Its layers resist sliding past each other.
Water spreads quickly — it is less viscous. Its layers slide past each other easily.
In blood flow:
The blood touching the vessel wall is stationary (no-slip condition).
The blood in the centre moves fastest.
Between these layers, fast layers drag slow layers forward — this internal friction is VISCOSITY.
Velocity profile
y
u_{max}
$u = 0$ (at wall)
NEWTON'S LAW OF VISCOSITY
Area = A
y
dy
$u + du$
Top layer (moves faster)
Bottom layer (moves slower)
$u = 0$
Velocity changes from layer to layer. Adjacent layers slide past each other.
Newton's law:
$\tau = \mu \frac{\partial u}{\partial y}$
τ = shear stress (force per unit area)
μ = dynamic viscosity (measure of fluid's resistance to flow)
$\frac{\partial u}{\partial y}$ = velocity gradient (change of velocity across layers)
Shear stress is proportional to velocity gradient.
Larger gradient ⇒ larger shear stress.
Viscosity arises from velocity differences inside the fluid. It resists motion between layers.

Viscosity


Velocity layers & shear
fast
slow (wall)
τ = μ ∂u/∂y


Poiseuille profile
u(r)=U_max(1−r²/R²)
1.00
0.75
0.50
0.25
0.00
−0.25
−0.50
−0.75
−1.00
r/R
Wall
0.0
0.2
0.4
0.6
0.8
1.0
u/U_max

Blood's Resistance to Shear



- Unlike pressure, viscosity comes from velocity differences inside the fluid

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- Top layer of fluid moves faster

Blood's Resistance to Shear



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Newton's law: shear stress = viscosity $\times$ velocity gradient. As per the shear stress law

$$\tau = \mu \frac{\partial u}{\partial y}$$

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- Top layer of fluid moves faster $\tau(y + dy)$
- Bottom layer moves slower

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$$\text{Net Force} = \tau(y + dy)A - \tau(y)A$$

As per Taylor Series expansion

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$$F = \tau(y + dy)A - \tau(y)A = \frac{\partial \tau}{\partial y} A\, dy = \mu \frac{\partial^2 u}{\partial y^2} A\, dy = \mu \frac{\partial^2 u}{\partial y^2} dV$$

Blood's Resistance to Shear



$$\frac{F_2}{dV} = \mu \frac{\partial^2 u}{\partial y^2}$$

Already, we have a force due to pressure and now another viscous force, so we have

$$\frac{F}{dV} = \rho \left(\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} \right)$$

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$$\rho \left(\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} \right) = -\frac{\partial p}{\partial x} + \mu \frac{\partial^2 u}{\partial y^2}$$

Navier–Stokes Equation

If any other external forces such as gravity, electromagnetic forces, rotation etc. act, then we get them as

$$\rho\left(\frac{\partial u}{\partial t}+u\frac{\partial u}{\partial x}\right)=-\frac{\partial p}{\partial x}+\mu\frac{\partial^2 u}{\partial y^2}+\rho g$$

Navier–Stokes Equation

Lifting A10 (1D $\to$ 3D), with A3 and A4 in force:

$$\rho\left(\frac{\partial \mathbf{u}}{\partial t} + (\mathbf{u}\cdot\nabla)\mathbf{u}\right) = -\nabla p + \mu\nabla^2\mathbf{u} + \mathbf{f}$$

$$\rho\frac{D\mathbf{u}}{Dt} = -\nabla p + \mu\nabla^2\mathbf{u} + \mathbf{f}$$

Navier–Stokes Terms (Blood Flow)

$\rho \frac{\partial \mathbf{u}}{\partial t}$	**Unsteady inertia** How fast is velocity changing at a fixed point? Zero for steady flow.
$\rho(\mathbf{u} \cdot \nabla)\mathbf{u}$	**Convective inertia** Is the parcel moving into a faster/slower region? Nonlinear term.
$-\nabla p$	**Pressure gradient**: Drives blood from high to low pressure.
$\mu \nabla^2 \mathbf{u}$	**Viscous diffusion**: Viscosity smooths velocity differences.
$\mathbf{f}$	**Body force**: Gravity, magnetic effects. Often negligible in arteries (A8).



How? (continued)
Mass Conservation

Mass In = Mass Out

- Blood rushes past me from the left.



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- I pick an imaginary gate—just a thin slice of space—and start counting.

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- “How much leaves?”

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- For a moment, I wonder—could more blood leave than enter?

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Impossible

Mass In = Mass Out

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- Then blood would start piling up here—the artery would swell.

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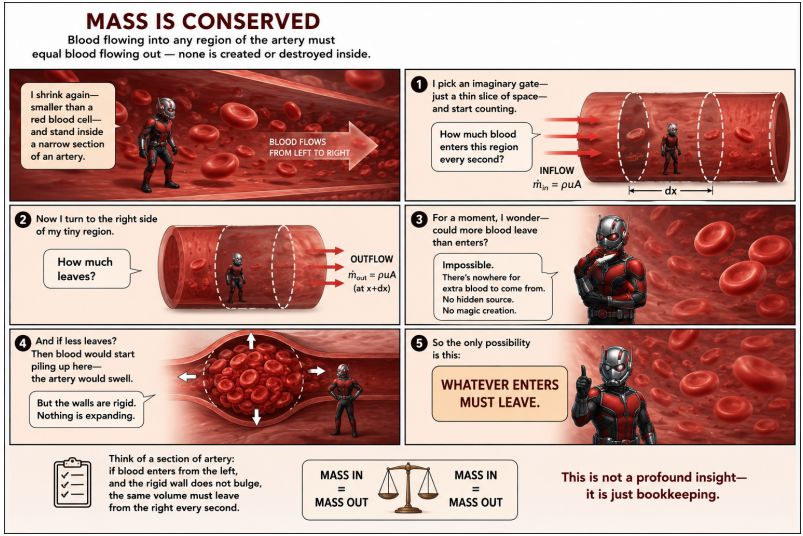
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- But the walls are rigid (A5). Nothing is expanding.
- So the only possibility is this:

Whatever enters must leave.

Mass is Conserved



Mass is Conserved

$$\frac{d}{dt}(\text{mass inside}) = \text{mass inflow} - \text{mass outflow}$$

$$\frac{d}{dt}(\text{mass inside}) = -\text{Net outflow}$$

Mass Conservation



Step 1: Consider a small fluid element

$$\text{Volume} = A\,dx, \quad \text{Mass} = \rho A\,dx$$

Mass Conservation



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$$\text{Outflow at } x + dx: \quad (\rho u A)\big|_{x+dx}$$

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Step 3: Net mass outflow

$$(\rho u A)_{x+dx} - (\rho u A)_x \approx \frac{\partial(\rho u A)}{\partial x}\,dx$$

Mass Conservation

Step 4: Rate of change of mass inside



Mass Conservation



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- At this same location

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- As time changes

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$$\frac{\partial}{\partial t}(\rho A\, dx) = A\, dx \frac{\partial \rho}{\partial t}$$

Mass Conservation



Step 4: Rate of change of mass inside

- At this same location
- As time changes
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Step 5: Apply conservation of mass (constant A, by A5)

$$A\, dx \frac{\partial \rho}{\partial t} + \frac{\partial(\rho u A)}{\partial x}\, dx = 0 \quad \Rightarrow \quad \frac{\partial \rho}{\partial t} + \frac{\partial(\rho u)}{\partial x} = 0$$

Mass Conservation



$$\frac{\partial \rho}{\partial t} + \frac{\partial (\rho u)}{\partial x} = 0$$

In 3D,

$$\frac{\partial \rho}{\partial t} + \nabla \cdot (\rho \mathbf{u}) = 0$$

If blood is incompressible (A4: ρ constant, “Blood changes speed—but not density.”),

$$\nabla \cdot (\rho \mathbf{u}) = \rho \, \nabla \cdot \mathbf{u} = 0 \quad \Rightarrow \quad \nabla \cdot \mathbf{u} = 0$$

Mass and Momentum Conservation

In 3D,

$$\rho \frac{D\mathbf{u}}{Dt} = -\nabla p + \mu \nabla^2 \mathbf{u} + \mathbf{f}$$

$$\frac{\partial \rho}{\partial t} + \nabla \cdot (\rho \mathbf{u}) = 0$$

Four unknowns (u_1, u_2, u_3, p) with ρ fixed by A4; four equations. The model is not yet complete.

Initial and Boundary Conditions in Blood Flow



$$\rho \frac{D\mathbf{u}}{Dt} = -\nabla p + \mu \nabla^2 \mathbf{u}$$

To solve this, we need:

- Initial Condition (IC)
- Boundary Conditions (BCs)

Initial Condition

Definition:

$$\mathbf{u}(\mathbf{x}, 0) = \mathbf{u}_0(\mathbf{x})$$



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Common choices in blood flow:

- Rest start: $\mathbf{u}(\mathbf{x}, 0) = 0$
- Developed flow profile (Poiseuille)
- Previous simulation state



Initial Condition



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Common choices in blood flow:

- Rest start: $\mathbf{u}(\mathbf{x}, 0) = 0$
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Physical Meaning: How is blood moving at the start?

Boundary Condition: Vessel Wall

$$\mathbf{u} = 0$$



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No-slip condition (A6, together with the rigid wall A5)

- Blood sticks to the vessel wall
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Boundary Condition: Vessel Wall



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Interpretation: Fluid in contact with the wall does not move

Boundary Condition: Inlet



Velocity-driven inlet:

$$\mathbf{u} = \mathbf{u}_{in}(t)$$

Boundary Condition: Inlet

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Example (pulsatile flow):

$$u(t) = U_0(1 + \sin \omega t)$$



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Flow-rate condition:

$$\int_A \mathbf{u} \cdot \mathbf{n} \, dA = Q(t)$$



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$$u(t) = U_0(1 + \sin \omega t)$$

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Interpretation: Flow is driven by the heart

Boundary Condition: Outlet

Pressure condition:

$$p = p_{out}$$



Boundary Condition: Outlet

Pressure condition:

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Traction-free condition:

$$-p\mathbf{n} + \mu \nabla \mathbf{u} \cdot \mathbf{n} = 0$$



Boundary Condition: Outlet



Pressure condition:

$$p = p_{out}$$

Traction-free condition:

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Interpretation: Blood exits into a region of known pressure

Boundary Condition: Symmetry

$$\frac{\partial \mathbf{u}}{\partial n} = 0$$



Boundary Condition: Symmetry

$$\frac{\partial \mathbf{u}}{\partial n} = 0$$

- Used in symmetric geometries
- Reduces computational domain



Summary of Conditions

Location	Condition	Meaning
Wall	$\mathbf{u} = 0$	No-slip
Inlet	$\mathbf{u}(t)$ or $Q(t)$	Driven by heart
Outlet	$p =$ const	Downstream pressure
Initial	$\mathbf{u}(\mathbf{x}, 0)$	Starting state



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Key Insight: Equations describe motion, boundary conditions define reality

Dimensionless View of Blood Flow



Key Idea:

- The Navier–Stokes equations involve parameters: ρ, μ, D, U
- But nature does not respond to absolute values
- **It responds only to ratios**

Dimensionless View of Blood Flow



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Non-dimensionalization: scale length by D, velocity by U, time by D/U, pressure by ρU^2:

$$\frac{\partial \mathbf{u}^*}{\partial t^*} + (\mathbf{u}^* \cdot \nabla^*)\mathbf{u}^* = -\nabla^* p^* + \frac{1}{\mathrm{Re}} \nabla^{*2} \mathbf{u}^*, \qquad \nabla^* \cdot \mathbf{u}^* = 0$$

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Flows with the same dimensionless numbers are physically equivalent

A lab artery model can replicate real blood flow if these ratios match

Dimensionless View of Blood Flow

Each variable is written as a typical size times a pure number. Starred quantities carry no units and are of order one.



Dimensionless View of Blood Flow

Each variable is written as a typical size times a pure number. Starred quantities carry no units and are of order one.

Variable	Scale	Substitution
Length	D, vessel diameter	$\mathbf{x} = D\,\mathbf{x}^*$
Velocity	U, mean speed	$\mathbf{u} = U\,\mathbf{u}^*$
Time	D/U, time to travel one diameter	$t = \dfrac{D}{U}\,t^*$
Pressure	ρU^2, dynamic pressure	$p = \rho U^2\,p^*$

Dimensionless View of Blood Flow



Since $x = D\,x^*$, a change in x is D times a change in x^*:

$$\frac{\partial}{\partial x} = \frac{1}{D}\frac{\partial}{\partial x^*} \quad \Longrightarrow \quad \nabla = \frac{1}{D}\nabla^*, \qquad \nabla^2 = \frac{1}{D^2}\nabla^{*2}.$$

Similarly, since $t = (D/U)\,t^*$:

$$\frac{\partial}{\partial t} = \frac{U}{D}\frac{\partial}{\partial t^*}.$$

Dimensionless View of Blood Flow

Term	After substitution	Result
$\rho \dfrac{\partial \mathbf{u}}{\partial t}$	$\rho \cdot \dfrac{U}{D} \cdot U \dfrac{\partial \mathbf{u}^*}{\partial t^*}$	$\dfrac{\rho U^2}{D} \dfrac{\partial \mathbf{u}^*}{\partial t^*}$
$\rho (\mathbf{u} \cdot \nabla)\mathbf{u}$	$\rho \cdot U\mathbf{u}^* \cdot \dfrac{1}{D}\nabla^* U\mathbf{u}^*$	$\dfrac{\rho U^2}{D} (\mathbf{u}^* \cdot \nabla^*)\mathbf{u}^*$
$-\nabla p$	$-\dfrac{1}{D}\nabla^* \rho U^2 p^*$	$\dfrac{\rho U^2}{D} (-\nabla^* p^*)$
$\mu \nabla^2 \mathbf{u}$	$\mu \cdot \dfrac{1}{D^2}\nabla^{*2} U\mathbf{u}^*$	$\dfrac{\mu U}{D^2} \nabla^{*2}\mathbf{u}^*$

Dimensionless View of Blood Flow



The equation becomes

$$\frac{\rho U^2}{D}\left(\frac{\partial \mathbf{u}^*}{\partial t^*} + (\mathbf{u}^* \cdot \nabla^*)\mathbf{u}^*\right) = \frac{\rho U^2}{D}\left(-\nabla^* p^*\right) + \frac{\mu U}{D^2}\nabla^{*2}\mathbf{u}^*.$$

The coefficient of the viscous term becomes

$$\frac{\mu U/D^2}{\rho U^2/D} = \frac{\mu}{\rho U D} = \frac{1}{\mathrm{Re}}, \qquad \mathrm{Re} = \frac{\rho U D}{\mu} = \frac{U D}{\nu}.$$

$$\frac{\partial \mathbf{u}^*}{\partial t^*} + (\mathbf{u}^* \cdot \nabla^*)\mathbf{u}^* = -\nabla^* p^* + \frac{1}{\mathrm{Re}}\nabla^{*2}\mathbf{u}^*$$

Dimensionless View of Blood Flow

$$\nabla \cdot \mathbf{u} = \frac{1}{D}\nabla^* \cdot (U\mathbf{u}^*) = \frac{U}{D}\,\nabla^* \cdot \mathbf{u}^* = 0.$$

Since $U/D \neq 0$,

$$\nabla^* \cdot \mathbf{u}^* = 0$$

With $\nu = 3.3 \times 10^{-6}\ \mathrm{m}^2/\mathrm{s}$:

Vessel	D	U	$\mathrm{Re} = UD/\nu$
Aorta	2 cm	0.2 m/s	≈ 1200
Coronary artery	3 mm	0.2 m/s	≈ 180



Principle 6 — Predict? What will our model predict?

Reynolds Number (Flow Regime)

$$\boxed{\mathrm{Re} = \frac{\rho U D}{\mu} = \frac{U D}{\nu}}$$



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- $\mathrm{Re} < 2300 \quad \rightarrow$ Laminar flow
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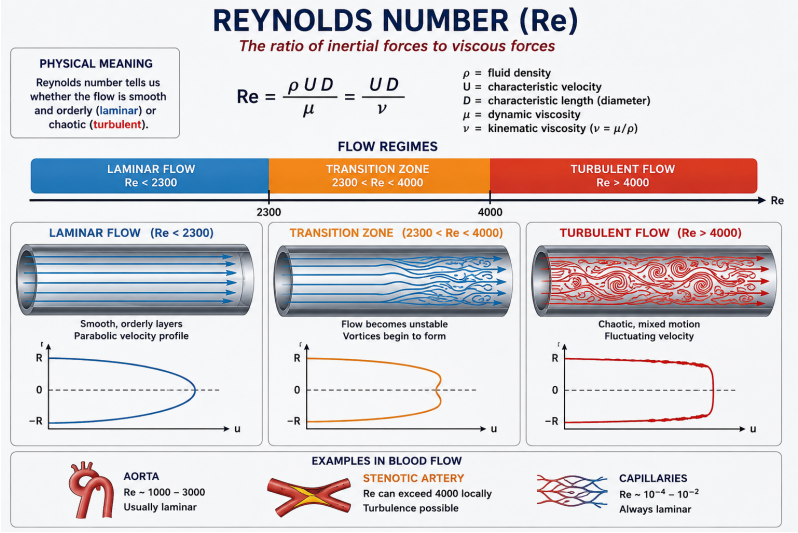
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In blood flow:

- Arteries: moderate Re (mostly laminar)
- Disturbed regions (stenosis): possible transition

Reynolds Number



Pulsatile Flow



- We consider incompressible flow of a Newtonian fluid with density ρ and dynamic viscosity μ (kinematic viscosity $\nu = \mu/\rho$) in a straight, rigid tube of radius R.
- The tube axis is the z-axis, so the cross-section is $x^2 + y^2 \leq R^2$.
- In Cartesian coordinates, with velocity $\boldsymbol{u} = (u, v, w)$, the Navier–Stokes equations read

$$\nabla \cdot \boldsymbol{u} = 0, \tag{1}$$

$$\rho \left(\frac{\partial \boldsymbol{u}}{\partial t} + (\boldsymbol{u} \cdot \nabla)\boldsymbol{u} \right) = -\nabla p + \mu \nabla^2 \boldsymbol{u}. \tag{2}$$

Pulsatile Flow



We assume the flow is purely axial:

$$u = v = 0, \qquad w = w(x, y, z, t). \tag{3}$$

The continuity equation (1) then reduces to

$$\frac{\partial w}{\partial z} = 0 \quad \Longrightarrow \quad w = w(x, y, t), \tag{4}$$

so the flow is fully developed.

Pulsatile Flow

With $u = v = 0$, the x- and y-components of (2) contain no velocity terms and give

$$\frac{\partial p}{\partial x} = 0, \qquad \frac{\partial p}{\partial y} = 0 \quad \Longrightarrow \quad p = p(z, t). \tag{5}$$

In the z-component, the convective term is

$$(\boldsymbol{u} \cdot \nabla) w = u \frac{\partial w}{\partial x} + v \frac{\partial w}{\partial y} + w \frac{\partial w}{\partial z} = 0, \tag{6}$$

and $\partial^2 w / \partial z^2 = 0$. The nonlinear term therefore drops out, leaving

$$\rho \frac{\partial w}{\partial t} = -\frac{\partial p}{\partial z} + \mu \left(\frac{\partial^2 w}{\partial x^2} + \frac{\partial^2 w}{\partial y^2} \right). \tag{7}$$

Pulsatile Flow

In (7), every term except $\partial p/\partial z$ depends on (x, y, t) only, while $\partial p/\partial z$ depends on (z, t) only. Both must therefore equal a function of time alone:

$$-\frac{\partial p}{\partial z} = G(t). \tag{8}$$

At each instant, the pressure varies linearly along the tube.

Pulsatile Flow

Let $r = \sqrt{x^2 + y^2}$. By axisymmetry, $w = w(r, t)$. Since

$$\frac{\partial r}{\partial x} = \frac{x}{r}, \qquad \frac{\partial r}{\partial y} = \frac{y}{r}, \tag{9}$$

the chain rule gives

$$\frac{\partial w}{\partial x} = \frac{\partial w}{\partial r}\,\frac{x}{r}. \tag{10}$$

Differentiating again with the product rule, and using $\partial(x/r)/\partial x = 1/r - x^2/r^3$,

$$\frac{\partial^2 w}{\partial x^2} = \frac{\partial^2 w}{\partial r^2}\,\frac{x^2}{r^2} + \frac{\partial w}{\partial r}\left(\frac{1}{r} - \frac{x^2}{r^3}\right). \tag{11}$$

Pulsatile Flow

By symmetry in y,

$$\frac{\partial^2 w}{\partial y^2} = \frac{\partial^2 w}{\partial r^2}\frac{y^2}{r^2} + \frac{\partial w}{\partial r}\left(\frac{1}{r} - \frac{y^2}{r^3}\right). \tag{12}$$

Adding the two and using $x^2 + y^2 = r^2$,

$$\frac{\partial^2 w}{\partial x^2} + \frac{\partial^2 w}{\partial y^2} = \frac{\partial^2 w}{\partial r^2} + \frac{1}{r}\frac{\partial w}{\partial r} = \frac{1}{r}\frac{\partial}{\partial r}\left(r\frac{\partial w}{\partial r}\right). \tag{13}$$

Pulsatile Flow

Substituting into (7),

$$\boxed{\rho \frac{\partial w}{\partial t} = G(t) + \frac{\mu}{r} \frac{\partial}{\partial r} \left(r \frac{\partial w}{\partial r} \right), \qquad 0 \leq r \leq R,} \tag{14}$$

subject to

$$w(R,t) = 0 \quad \text{(no slip)}, \qquad \left. \frac{\partial w}{\partial r} \right|_{r=0} = 0 \quad \text{(symmetry)}. \tag{15}$$

Pulsatile Flow

For a pulsatile flow driven by one Fourier mode of the pressure gradient, $G(t) = A\,e^{i\omega t}$, introduce

$$r^* = \frac{r}{R}, \qquad t^* = \omega t, \qquad w^* = \frac{w}{U}. \tag{16}$$

The unsteady inertia term scales as $\rho\omega U$ and the viscous term as $\mu U/R^2$. Their ratio is

$$\frac{\rho\omega U}{\mu U/R^2} = \frac{\omega R^2}{\nu} \equiv \alpha^2, \qquad \boxed{\mathrm{Wo} = R\sqrt{\frac{\omega}{\nu}}} \tag{17}$$

and (14) becomes

$$\mathrm{Wo}^2\,\frac{\partial w^*}{\partial t^*} = \hat{A}\,e^{it^*} + \frac{1}{r^*}\frac{\partial}{\partial r^*}\left(r^*\frac{\partial w^*}{\partial r^*}\right), \qquad \hat{A} = \frac{AR^2}{\mu U}. \tag{18}$$

Equivalently, $\mathrm{Wo} = \sqrt{2}\,R/\delta$, where $\delta = \sqrt{2\nu/\omega}$ is the Stokes layer thickness.

Pulsatile Flow

Seeking $w = W(r)\, e^{i\omega t}$ in (14) gives

$$W'' + \frac{1}{r} W' - \frac{i\omega}{\nu} W = -\frac{A}{\mu}. \tag{19}$$

The particular solution is $A/(i\rho\omega)$; the homogeneous solution regular at $r = 0$ is $J_0\big(i^{3/2}\alpha\, r/R\big)$. Imposing $W(R) = 0$,

$$w(r,t) = \mathrm{Re}\left\{ \frac{A}{i\rho\omega} \left[1 - \frac{J_0\big(i^{3/2}\mathrm{Wo}\, r/R\big)}{J_0\big(i^{3/2}\mathrm{Wo}\big)} \right] e^{i\omega t} \right\}. \tag{20}$$

For $\mathrm{Wo} \ll 1$ this tends to quasi-steady Poiseuille flow in phase with the pressure gradient; for $\mathrm{Wo} \gg 1$ the core moves as a plug lagging the pressure gradient by about 90°, with viscous effects confined to a wall layer of thickness $O(R/\mathrm{Wo})$.

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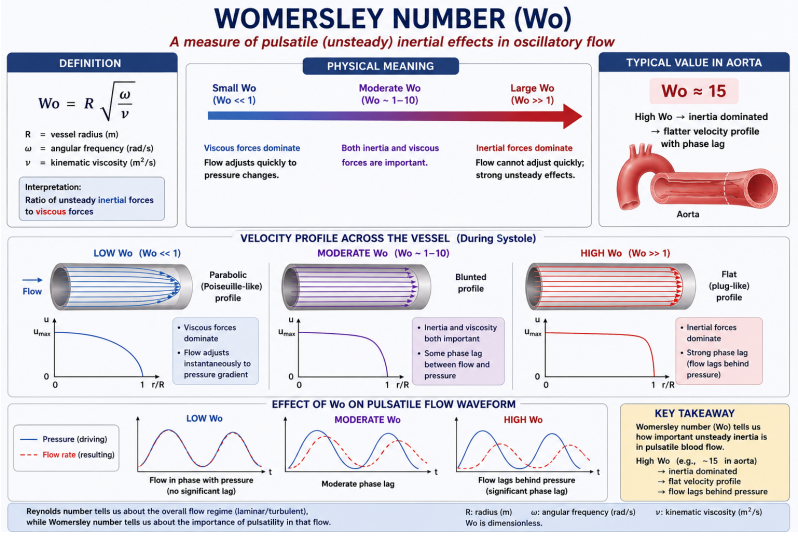
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In blood flow:

- Aorta: $\mathrm{Wo} \approx 15$
- Flow is strongly pulsatile and inertia-dominated

Womersley Number (Wo)



Prediction 1: Which Regime Are We In?

Heart rate 72 beats/min $\Rightarrow \omega = 2\pi(1.2) \approx 7.5$ rad/s; $\nu = 3.3 \times 10^{-6}$ m^2/s.

Vessel	D	U (mean)	Re	Wo
Aorta	2 cm	0.2 m/s	≈ 1200	≈ 15
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- Mean flow is laminar everywhere in this table: A7 survives the check.
- Aorta: flat, pulsatile profile. Coronary: nearly parabolic, quasi-steady.

Prediction 2: The Stenotic Jet

From $\nabla \cdot \mathbf{u} = 0$ and rigid walls, the volume flow rate is the same at every cross-section:

$$Q = U_1 A_1 = U_2 A_2 \quad \Rightarrow \quad \frac{U_2}{U_1} = \frac{A_1}{A_2} = \left(\frac{D_1}{D_2}\right)^2$$

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This is exactly what the Ant-Man saw: the blood “accelerates sharply, squeezing through the constriction”.

Prediction 3: Why a Narrowing Steals Pressure



Steady, fully developed flow in a straight rigid tube of radius R and length L ($\partial_t = 0$, convective term vanishes, $\Delta p = p_{\text{in}} - p_{\text{out}}$). Navier–Stokes reduces to

$$0 = \frac{\Delta p}{L} + \mu \frac{1}{r}\frac{d}{dr}\left(r\frac{du}{dr}\right) \quad \Rightarrow \quad u(r) = \frac{\Delta p}{4\mu L}\left(R^2 - r^2\right)$$

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- Halve the radius $\Rightarrow$ resistance $\times 16$.
- For the same Q, the pressure drop across the stenosis rises sharply, so p_{distal} falls and FFR falls.

What Controls Blood Flow?

- Reynolds number $\rightarrow$ flow regime (laminar vs turbulent)
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Key Insight: Two flows with the same Reynolds and Womersley numbers are dynamically identical

Physics is scale-free when expressed in dimensionless form



Principle 7 — Valid? Are the predictions valid?

Check 1: Dimensional Consistency

Every term in Navier–Stokes must carry the units of force per volume, $\mathrm{N/m^3} = \mathrm{kg\,m^{-2}\,s^{-2}}$.

$\rho\,\partial\mathbf{u}/\partial t$	$\mathrm{kg/m^3 \cdot m/s^2}$
$\rho\,(\mathbf{u}\cdot\nabla)\mathbf{u}$	$\mathrm{kg/m^3 \cdot (m/s)(m/s)/m}$
∇p	$\mathrm{Pa/m = N/m^3}$
$\mu\nabla^2\mathbf{u}$	$\mathrm{Pa\,s \cdot (m/s)/m^2 = Pa/m}$

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Limit checks

- $\mu \to 0$: Euler equations of inviscid flow.
- $\mathbf{u} \to 0$: $\nabla p = \mathbf{f}$, hydrostatics.

Check 2: Revisit the Ledger

No.	Check	Verdict
A1	Red cell $\approx 8\,\mu$m vs coronary lumen 3 mm	Holds; fails in capillaries
A3	Blood shear-thins at low shear rates; large-artery shear is mostly high	Holds; doubtful in the recirculation zone
A4	$\Delta p \sim 16$ kPa vs bulk modulus of water ~ 2 GPa $\Rightarrow \Delta\rho/\rho \sim 10^{-5}$	Holds
A5	Aortic diameter changes by several percent per beat	Questionable in the aorta
A7	Mean Re < 2300 in the table	Holds; post-stenotic jet may transition
A8	Patient lying flat	Holds; posture adds hydrostatic offsets

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A model is valid only within the region of the ledger that survives this table.



Principle 8 — Verified? Are the predictions good?

Comparing with Reality

- **Analytical benchmarks**: a solver must reproduce the Poiseuille profile (steady) and the Womersley profile (pulsatile) before it is trusted on a patient.



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Fitting $\neq$ truth $\neq$ usefulness.



Principle 9 — Improve? Can we improve the model?

Lift One Assumption at a Time

Lift	Improved model
A3	Non-Newtonian viscosity $\mu(\dot{\gamma})$: Carreau–Yasuda, Casson
A5	Fluid–structure interaction: the wall breathes again
Outlet	Lumped Windkessel (RCR) models for the downstream vessels
A7	Transition and turbulence models behind severe stenoses
A1, A2	Particle or multiphase models for red cells in small vessels
A10	Patient-specific 3D geometry from CT or MRI



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Because each assumption was numbered and independent, each improvement has a clear address in the ledger.

Why Geometry Controls the Pressure Loss

A classical lumped model of the pressure drop across a stenosis (Young–Tsai, Gould):

$$\Delta p = \underbrace{\frac{8\pi\mu L}{A_s^2}\,Q}_{\text{viscous loss in the throat}} + \underbrace{\frac{\rho}{2}\left(\frac{1}{A_s}-\frac{1}{A_0}\right)^2 Q^2}_{\text{expansion (separation) loss}}$$

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- Length L enters *linearly*; the throat area A_s enters as A_s^{-2}. The minimum lumen dominates.
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- The quadratic term grows fastest when flow rises. This is why FFR is measured at maximal flow.

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Two lesions with the same % diameter but different length or shape give different Δp, so different FFR. Percent narrowing alone is not enough to decide treatment.

Why Geometry Matters for the Model

Geometric feature	Flow consequence
Converging section	Flow accelerates, pressure falls
Throat (d)	High-speed jet; peak wall shear stress, linked to plaque rupture
Diverging section	Adverse pressure gradient, flow separation
Post-stenotic region	Recirculation; low and oscillating wall shear, linked to further plaque growth
Eccentric plaque	Asymmetric jet that bends toward one wall
Lesions in series	Pressure losses add; one lesion changes the flow seen by the next



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In the model, geometry enters only through the boundary of the domain, yet it decides almost everything downstream. In patient-specific CT-FFR, segmentation of the lumen is a leading source of error.

Research Direction

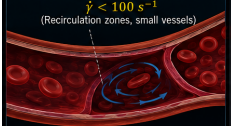
NON-NEWTONIAN BLOOD (DIRECTION 3 RESEARCH)

Blood viscosity depends on shear rate ($\dot{\gamma}$) – it is **NOT** constant.

WHERE SHEAR RATE IS LOW

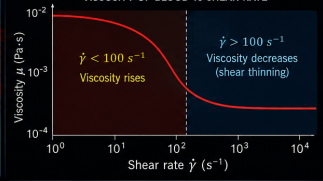
$\dot{\gamma} < 100\ s^{-1}$

(Recirculation zones, small vessels)

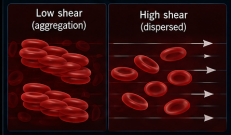


Low shear ⇒ RBCs aggregate (rouleaux)
⇒ higher resistance to flow
⇒ viscosity rises

VISCOSITY OF BLOOD vs SHEAR RATE



WHY?



At low shear, RBCs form aggregates
⇒ more resistance ⇒ higher viscosity.

THEREFORE, BLOOD IS NON-NEWTONIAN: viscosity μ depends on shear rate $\dot{\gamma} \rightarrow \mu = \mu(\dot{\gamma})$

OLD (NEWTONIAN) MODEL – INCORRECT HERE

Assumes μ = constant

$$\mu \nabla^2 u$$

Ignores spatial variation of viscosity.
Missing a major term!

CORRECT MODEL FOR NON-NEWTONIAN BLOOD

Viscosity varies in space through $\dot{\gamma}(x)$

$$\nabla \cdot \left[\mu(\dot{\gamma}) \left(\nabla u + \nabla u^T \right) \right]$$

Divergence of stress tensor

Accounts for viscosity gradients in space

THE MISSING VISCOSITY-GRADIENT TERM

When $\mu = \mu(x)$, expand:

$$\underbrace{\nabla \cdot \left[\mu(\dot{\gamma}) \left(\nabla u + \nabla u^T \right) \right]}_{\mu \nabla^2 u \ + \ (\nabla u) \cdot (\nabla u + \nabla u^T)}$$

This viscosity-gradient term is $O(1)$ in recirculation zones and small vessels!
It cannot be neglected.

In low-shear regions ($\dot{\gamma} < 100\ s^{-1}$), viscosity rises and varies strongly in space.

You **MUST** use $\nabla \cdot \left[\mu(\dot{\gamma}) \left(\nabla u + \nabla u^T \right) \right]$ — the extra term is *not* small; it is **O (1)** !

Typical units

$\dot{\gamma}$: s^{-1}

μ : $\mathrm{Pa} \cdot s$

Research Direction

RESEARCH DIRECTION 3 — NON-NEWTONIAN BLOOD & NUMERICAL ACCURACY

1 BLOOD IS NOT NEWTONIAN

- Blood is a suspension of cells — ~45% red blood cells by volume.
- **High shear** (fast flow, large arteries): cells align and move smoothly → Newtonian behaviour ($\mu \approx 3.5$ mPa·s)
- **Low shear** (slow flow, recirculation): cells aggregate into stacks (rouleaux) → effective **viscosity RISES** dramatically.

Shear-thinning: Viscosity decreases with increasing shear rate.

High shear (large arteries)
Cells align with flow



Smooth flow, Newtonian behaviour
$\mu \approx 3.5$ mPa·s

Low shear (recirculation zone, stenosis)
Cells aggregate (rouleaux formation)



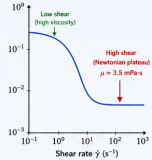
Increased effective viscosity

2 CARREAU–YASUDA MODEL

Shear-dependent viscosity model for blood:

$$\mu(\dot{\gamma}) = \mu_\infty + (\mu_0 - \mu_\infty)\left[1 + (\lambda\dot{\gamma})^a\right]^{\frac{n-1}{a}}$$

μ_0	—	Zero-shear viscosity (Pa·s)
μ_∞	—	Infinite-shear viscosity (Pa·s)
λ	—	Time constant (s)
a	—	Dimensionless parameter (shape)
n	—	Power-law index (0<n<1 for shear-thinning)
$\dot{\gamma}$	—	Shear rate (s^{-1}) $= \sqrt{2\,\mathbf{S} : \mathbf{S}}$ where $\mathbf{S} = \frac{1}{2}(\nabla\mathbf{u} + \nabla\mathbf{u}^T)$ is the strain-rate tensor



3 THE CRITICAL ERROR IN EXISTING GFDM SOLVERS

WHAT IS COMMONLY DONE (INCORRECT)

Viscous term approximated as:

$$\mu\nabla^2 u$$

This **DROPS** the viscosity-gradient term.

$\neq$

WHAT IS CORRECT

The viscous term should be:

$$\nabla \cdot \left[\mu(\dot{\gamma})\,(\nabla u + \nabla u^T)\right]$$

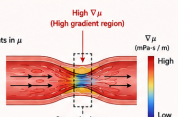
Expanding the correct term:

$$\nabla \cdot \left[\mu(\nabla u + \nabla u^T)\right] = \mu\nabla^2 u + (\nabla\mu)\cdot(\nabla u + \nabla u^T)$$
$$= \mu\nabla^2 u + 2\mathbf{S}\cdot\nabla\mu$$

MISSING TERM:
$2\mathbf{S}\cdot\nabla\mu$
(viscosity-gradient term)

WHY THIS MATTERS (AT STENOSIS)

- Near the stenosis throat: Large gradients in shear rate → large gradients in μ
- The missing term $2\mathbf{S}\cdot\nabla\mu$ is $\mathcal{O}(1)$
- Neglecting it introduces $\mathcal{O}(h)$ error in **Wall Shear Stress (WSS)**
- Including it restores $\mathcal{O}(h^2)$ accuracy



4 IMPACT AND CONTRIBUTION

PHYSICAL IMPACT
Captures true non-Newtonian behaviour of blood, especially in low-shear, recirculating regions behind stenosis.

NUMERICAL IMPACT
Correct treatment of viscosity gradients eliminates leading-order error in WSS predictions.

CLINICAL IMPACT
More accurate WSS → better assessment of plaque progression risk and treatment planning.

OUR CONTRIBUTION
Develop and implement a GFDM stencil that includes the viscosity-gradient term, restoring second-order accuracy for non-Newtonian blood flow.

★ ACCURATE PHYSICS + CORRECT NUMERICS = RELIABLE PREDICTIONS WHERE IT MATTERS MOST.



Principle 10 — Use? How will we exercise the model?

Clinical Applications of Blood Flow Simulation



- **Surgical planning**: Stent placement and sizing for coronary disease, Bypass graft haemodynamics
- **Device design**: Artificial heart valves — FSI simulations, Ventricular assist devices (VADs)
- **Diagnosis**: Fractional flow reserve (FFR) from CT angiography, Aneurysm rupture risk assessment
- **Drug delivery**: targeted nanoparticle transport

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Back to Principle 1. Image the artery, solve for $\mathbf{u}$ and p, read off FFR: if $\mathrm{FFR} \leq 0.80$, the stenosis is treated; otherwise the patient is spared a procedure.

Summary

BLOOD FLOW DYNAMICS — A COMPLETE SUMMARY

From Fundamentals to Dimensionless Insight

1. MASS CONSERVATION (Continuity Equation)

Consider a small fluid element of length dx and area A

Mass inside $= \rho A\, dx$

Inflow at x: $\rho u A$

Outflow at $x + dx$: $[\rho u A]_{x+dx}$

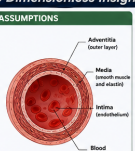
ρ, u, A, dx, x

Step 1	Step 2	Step 3	Step 4	Step 5
Mass inside	Mass flow rate	Net outflow	Rate of increase of mass inside	Conservation of mass (Rate of increase + Net outflow = 0)
$m = \rho A\, dx$	Inflow $= \rho u A$ Outflow $= [\rho u A]_{x+dx}$	$[\rho u A]_{x+dx} - [\rho u A]_x$ $\approx \frac{\partial(\rho u A)}{\partial x} dx$	$\frac{\partial m}{\partial t} = \frac{\partial(\rho A\, dx)}{\partial t}$ $= A\, dx \frac{\partial \rho}{\partial t}$	$A\, dx \frac{\partial \rho}{\partial t} + \frac{\partial(\rho u A)}{\partial x} dx = 0$ $\Rightarrow \frac{\partial \rho}{\partial t} + \frac{\partial(\rho u)}{\partial x} = 0$

For incompressible blood (ρ = constant): $\frac{\partial \rho}{\partial t} = 0 \Rightarrow \frac{\partial u}{\partial x} = 0$ (Volume flow in = Volume flow out)

2. BLOOD FLOW: KEY ASSUMPTIONS

- Blood is treated as a Newtonian fluid ($\mu \approx 3$–4 mPa·s for large arteries)
- Incompressible: density ρ = constant (~1060 kg/m³)
- Arteries are compliant (walls deform), but locally we analyze flow in a small segment
- Pulsatile (unsteady) flow driven by heartbeat
- Axisymmetric flow often assumed in healthy straight arteries



3. NAVIER–STOKES EQUATIONS FOR BLOOD

Incompressible Newtonian fluid

$$\rho\left(\frac{\partial u}{\partial t} + (u \cdot \nabla)u\right) = -\nabla p + \mu \nabla^2 u + f$$

Unsteady inertia | Convective inertia | Pressure gradient | Viscous effects | Body forces

Continuity equation (incompressible):

$$\nabla \cdot u = 0$$

Parameters:

- ρ = density (kg/m³)
- μ = dynamic viscosity (Pa·s)
- D = characteristic length (e.g., diameter)
- U = characteristic velocity (e.g., mean velocity)

Nature does not see all combinations equally — it sees only RATIOS. That is why we use DIMENSIONLESS NUMBERS.

4. REYNOLDS NUMBER (Re) — FLOW REGIME

$$Re = \frac{\rho U D}{\mu} = \frac{U D}{\nu}$$

$\nu = \mu/\rho$ (kinematic viscosity)

LAMINAR FLOW Re < 2300 | TRANSITION ZONE 2300 < Re < 4000 | TURBULENT FLOW Re > 4000

10^{-3} 10^{-2} 2300 10^{3} 4000 10^{4} 10^{5} Re

PHYSICAL MEANING: Ratio of inertia forces to viscous forces.

TYPICAL VALUES IN CARDIOVASCULAR SYSTEM

Aorta: Re ~ 1000 – 3000 (laminar)
Carotid: Re ~ 500 – 2000 (laminar)
Coronary: Re ~ 200 – 1000 (laminar)
Arterioles: Re << 1 (creeping flow)
Stenosis/aneurysm: local Re can exceed 4000 → turbulence

Laminar (Re < 2300)	Transition (2300 < Re < 4000)	Turbulent (Re > 4000)
Smooth, orderly layers	Flow becomes unstable	Chaotic, eddies, mixing



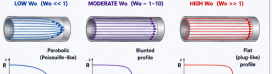
5. WOMERSLEY NUMBER (Wo) — PULSATILITY

$$Wo = R\sqrt{\frac{\omega}{\nu}}$$

R = vessel radius (m)
ω = angular frequency (rad/s)
ν = kinematic viscosity (m²/s)

PHYSICAL MEANING: Ratio of unsteady inertial forces to viscous forces.

LOW Wo (Wo << 1)	MODERATE Wo (Wo ~ 1–10)	HIGH Wo (Wo >> 1)
Parabolic (Poiseuille-like)	Blunted profile	Flat (plug-like) profile



TYPICAL VALUE: Aorta at rest: Wo ≈ 15 (High Wo → inertia dominated, flat velocity profile with phase lag)

EFFECT ON PULSATILE WAVEFORM

Pressure (driving) — Flow rate (resulting)



6. DIMENSIONLESS VIEW — THE BIG PICTURE

By non-dimensionalizing the Navier–Stokes equations (using D and U), the flow is governed only by dimensionless numbers (ratios), not by absolute values.

Key dimensionless numbers in blood flow

- $Re = \rho U D/\mu$ → flow regime
- $Wo = R\sqrt{\omega/\nu}$ → pulsatility
- Other important groups (Womersley, Reynolds–Carpenter, etc.) in advanced analyses

SIMILARITY PRINCIPLE

Two flows with the same Re and Wo are dynamically similar — even if their sizes, velocities, or fluids are different.



7. TAKE-HOME MESSAGE

- Mass is conserved: In = Out (for incompressible blood).
- Flow behavior depends on inertia vs viscosity (Re).
- Pulsatile nature introduces unsteady effects (Wo).
- Dimensionless numbers reveal the true physics.

Understand the ratios, and you understand the flow.

ANT-MAN PERSPECTIVE



Inside a tiny slice of artery, I see ordered layers in calm regions, disturbance in stenosis, and strong pulses with each heartbeat.

But the rules that govern my ride are written in ratios — Re and Wo!

Exercises



1. The common carotid artery has $D \approx 6$ mm and mean velocity ≈ 0.3 m/s. Compute Re and Wo at 72 beats/min. Which assumptions in the ledger are affected?
2. Starting from the 1D momentum equation, state the extra assumptions needed to reach the Poiseuille profile. Add them to the ledger as A11, A12, …
3. In a capillary ($D \approx 6\,\mu$m), which ledger entries fail? Propose one way to lift each.
4. A 70% diameter stenosis: by what factor do the throat velocity and Poiseuille resistance change?
5. Write Dym's ten questions for blood flow in a compliant aorta (A5 lifted). Which answers change?



Thank You