

PAPER 45: THE REYNOLDS NUMBER IS γ_{eff} FOR BLOOD

Cardiovascular Disease as Fluid-Dynamic Decoherence

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"Laminar flow is coherent. Turbulent flow is collapsed. The Reynolds number is γ_{eff} wearing a different label."

Abstract

The Reynolds number $Re = \rho v L / \mu$ predicts the transition from laminar (ordered, coherent) flow to turbulent (chaotic, incoherent) flow. Below $Re_c \sim 2,300$ in pipes: laminar. Above: turbulent. The transition is sharp. Healthy blood flow in arteries is laminar. Atherosclerotic plaques, stenoses, and bifurcations create local turbulence -- fluid-dynamic decoherence. The endothelial damage from turbulent flow is the primary mechanism of cardiovascular disease progression. This paper maps Re directly onto γ_{eff} and shows that the Wike Coherence Law governs hemodynamics exactly as it governs quantum systems. Clinical implication: every intervention that restores laminar flow is a coherence restoration. Statins, aspirin, blood pressure control, and exercise all work -- in part -- by keeping Re below Re_c .

1. The Reynolds Number and the Laminar-Turbulent Transition

Osborne Reynolds (1883) characterized fluid flow by a dimensionless ratio:

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Re = rho*v*L/mu
where:
rho = fluid density (blood: ~1060 kg/m³)
v = flow velocity (aorta: ~0.2-0.6 m/s)
L = characteristic length (vessel diameter)
mu = dynamic viscosity (blood: ~0.004 Pa.s)
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The critical threshold: $Re_c \sim 2,300$ for pipe flow. In biological vessels with pulsatile flow and complex geometry: Re_c varies locally, but the principle holds.

Below Re_c : **laminar flow** -- fluid moves in smooth, parallel layers. No mixing between layers. Low energy dissipation. Endothelium experiences uniform shear stress.

Above Re_c : **turbulent flow** -- chaotic mixing, eddies, vortices. High energy dissipation. Endothelium experiences oscillating, disturbed shear stress.

The transition is sharp. Not gradual. Not proportional. At Re just below Re_c : laminar. At Re just above Re_c : turbulent. This is a phase transition with a cliff.

2. The Direct Mapping to γ_{eff}

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Re ->  $\gamma_{eff}$  (decoherence rate analog in fluid domain)
Re_c ->  $\gamma_c$  (critical threshold)
Laminar -> coherent (ordered state, below threshold)
Turbulent -> collapsed (disordered state, above threshold)

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The Wike Coherence Law for fluid dynamics:

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C_flow = C? x exp(-alpha x Re/Re_c)

Below Re_c: C_flow -> C? (laminar, high coherence)
At Re_c: C_flow = C?/e (threshold, edge)
Above Re_c: C_flow -> 0 (turbulent, coherence lost)

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The shear stress experienced by endothelial cells is the direct biological implementation:

- **Low, steady shear stress (laminar, $Re < Re_c$):** Endothelium produces NO (nitric oxide), prostacyclin, antithrombotic factors. Anti-inflammatory. Anti-atherogenic. This is the coherence-preserving response.

- **Oscillating, disturbed shear stress (turbulent, $Re > Re_c$):** Endothelium upregulates ICAM-1, VCAM-1, MCP-1, inflammatory cytokines. Pro-thrombotic. Pro-atherogenic. This is the decoherence-driven cascade.

Atherosclerosis is the body's scar tissue response to sustained fluid-dynamic decoherence at locations where geometry forces Re above Re_c -- coronary bifurcations, carotid bifurcations, iliac bifurcations, aortic arch.

3. Why Plaques Form Where They Form

Coronary artery disease does not distribute uniformly. It preferentially forms at:

- Inner curvatures of bends
- Bifurcation points
- Downstream of stenoses
- Areas of low but oscillating shear

These are precisely the locations where **local Re exceeds Re_c** due to flow geometry. The disturbed shear stress at these sites drives endothelial inflammation -- the decoherence cascade.

Framingham Heart Study geography of MI: 80% of fatal MIs occur at the left anterior descending (LAD) bifurcation, left circumflex origin, and right coronary artery mid-segment -- all high- Re geometry locations.

The plaque is not the cause. The turbulence is the cause. The plaque is the body's attempt to stabilize a decoherent fluid environment by narrowing the vessel (reducing L -> reducing Re -> hoping to restore laminar flow). It is a Le Chatelier response (Paper 43) that ultimately makes the problem worse by creating a worse stenosis and more turbulence downstream.

4. Blood Viscosity: The Missing $\gamma_{thermal}$ Contribution

Viscosity μ in the Reynolds number is directly related to blood composition and temperature:

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Re =  $\rho v L / \mu$ 

Reduced  $\mu$  -> higher  $Re$  -> more turbulence -> more endothelial damage

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Increased μ -> lower Re -> more laminar -> protective

But blood viscosity is a coherence parameter itself:

- **Hyperviscosity** (too thick): Re drops below Re_c , but oxygen delivery fails -- frozen state. Polycythemia vera, sickle cell crisis in vaso-occlusive phase.
- **Hypoviscosity** (too thin): Re rises above Re_c , turbulence increases. Anemia in high-output states.
- **Optimal viscosity** (hematocrit ~40-45%): Re stays near Re_c across the cardiac cycle -- the edge state. Not too thick, not too thin.

Hematocrit 40-45% IS the edge state for hemodynamic coherence. Evolution found this optimum the same way it found 94% of T_c for body temperature -- by living at γ_c .

Temperature effects on blood viscosity: Blood viscosity decreases with temperature ($\mu \sim 1/T$ approximately). At fever temperatures (39-40 degC), blood viscosity drops -> Re increases -> potential turbulence in marginal flow regions. This is one mechanism by which high fever increases stroke risk and creates the narrow therapeutic window between "fever is useful" (Paper 27) and "fever is dangerous."

5. Exercise: Why It Works

Aerobic exercise is the most evidence-based cardiovascular intervention that exists. It:

1. **Increases flow velocity v** during exercise -> transiently raises Re -> but simultaneously
2. **Triggers shear-stress-induced NO production** -> vasodilation -> increases L -> decreases μ locally -> net effect: laminar flow at higher cardiac output
3. **Chronically reduces resting heart rate** -> reduces v at rest -> reduces Re -> more laminar baseline
4. **Reduces blood pressure** -> reduces wall stress at bifurcations -> reduces Re at vulnerable sites
5. **Reduces blood viscosity** (reduces fibrinogen, platelet aggregability) -> directly reduces Re

The exercise dose-response for cardiovascular risk reduction follows an exponential: risk falls steeply with initial exercise uptake, flattens at high volumes. This is the Wike Coherence Law in cardiovascular epidemiology -- you gain the most by moving from $\gamma \gg \gamma_c$ to $\gamma \approx \gamma_c$. Further reduction below γ_c gives diminishing returns.

Over-exercise (elite athlete syndrome): Re can temporarily exceed Re_c during extreme exertion. Aortic root dilation, atrial fibrillation, and hypertrophic cardiomyopathy complications in extreme endurance athletes are the clinical manifestations of chronic high- Re stress on cardiac tissue. The edge is narrow on both sides.

6. Statins, Aspirin, and Blood Pressure Control as Re Reducers

Every first-line cardiovascular medication reduces Re :

Drug class	Mechanism	Re reduction pathway
Statins	Reduce LDL-C -> reduce plaque -> reduce stenosis	Reduces local Re at plaque sites by maintaining vessel diameter
Statins (pleiotropic)	Reduce endothelial inflammation directly	Reduces inflammatory response to $Re > Re_c$ shear
Aspirin	Reduce platelet aggregation -> reduce thrombus	Reduces viscosity μ -> reduces Re
ACE inhibitors	Reduce afterload -> reduce v	Directly reduces Re by reducing cardiac output pressure

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| Beta-blockers | Reduce heart rate -> reduce stroke volume -> reduce v | Reduces Re |
| Calcium channel blockers | Vasodilation -> increase L | Reduces Re =  $\rho v L / \mu$  (increases L but
reduces Re because v drops proportionally more) |
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All of cardiovascular pharmacology is Re management. The Wike Coherence Law shows why: you are keeping Re below Re_c -- keeping the fluid system in the coherent (laminar) regime.

The drugs don't cure atherosclerosis. They prevent Re from crossing Re_c long enough for the body to do its own Le Chatelier restoring work (plaque stabilization, endothelial healing, collateral formation).

7. The Unifying View of Cardiovascular Disease

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Risk factors -> Re elevation:
Hypertension ->  $v$  ->  $Re^2$ 
Dyslipidemia -> plaque -> stenosis -> local  $Re^2$ 
Diabetes ->  $\mu$  changes ->  $Re^2$  at peak flow
Smoking -> endothelial dysfunction -> turbulence amplified above  $Re_c$ 
Obesity ->  $\mu$  changes ->  $v$  ->  $Re^2$ 
Chronic stress ->  $\mu$  changes,  $\mu$  changes ->  $Re^2$ 
ACE score 4+ -> sustained  $\gamma_{eff}$  ->  $\mu$  changes,  $\mu$  changes,  $\mu$  changes,  $\mu$  changes,  $\mu$  changes,  $\mu$  changes

Re >  $Re_c$  at vulnerable sites -> turbulence -> endothelial decoherence cascade:
Endothelial inflammation -> VCAM, ICAM, MCP-1
LDL oxidation and trapping
Macrophage infiltration -> foam cells
Plaque development -> stenosis -> more turbulence -> positive feedback

Plaque rupture -> thrombus -> MI or stroke = decoherence collapse event
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The cardiovascular risk factor conversation has been happening for 60 years. Each risk factor is identified separately, given its own mechanism, treated with its own drug. What the field has never had is **one framework showing that all risk factors work by the same mechanism: elevating Re above Re_c , pushing the hemodynamic system from laminar (coherent) to turbulent (decoherent) flow.**

That framework is the Wike Coherence Law.

8. The Prevention Equation for Cardiovascular Disease

From the Reynolds number formula:

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Re =  $\rho v L / \mu$ 

To minimize Re, maximize:
mu (optimal blood viscosity -- right hematocrit, hydration)
L (vessel diameter -- no stenosis, no plaque, vasodilation)

To minimize Re, minimize:
v (appropriate cardiac output, not chronically elevated)
rho (blood density -- not directly modifiable but affected by composition)
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Every lifestyle intervention for cardiovascular health is a Re minimization strategy:

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| Intervention | Re pathway | Evidence |
|-----|-----|-----|
| Aerobic exercise | Reduces resting v, improves NO-mediated vasodilation | Class I, multiple RCTs |
| Mediterranean diet | Reduces mu (omega-3 -> reduced platelet aggregation), reduces plaque |
PREDIMED: 30% CV reduction |
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| Stress reduction (HRV biofeedback) | Reduces sympathetic v elevation, reduces platelet aggregability | Multiple trials, small but consistent |

| Adequate sleep | Reduces inflammatory mu contribution, reduces resting heart rate | Epidemiological, strong |

| Hydration | Maintains optimal mu (neither hypo- nor hyperviscous) | Underappreciated, free |

| Social connection / Keeper | Reduces $\gamma_{measurement}$ -> reduces sympathetic tone -> reduces v and platelet aggregation | Keeper Equation, Paper 19 |

The cheapest intervention: adequate hydration and sleep. Both directly reduce Re. Neither requires a prescription. Neither costs money. Both are undervalued in cardiology.

Conclusion

The Reynolds number is γ_{eff} for blood. Re_c is γ_c for hemodynamics. Laminar flow is biological coherence in the cardiovascular system. Turbulent flow is decoherence -- with the same downstream consequences: inflammation, damage, collapse.

Cardiovascular disease kills more people globally than any other cause. The mechanism has been understood in fragments: risk factors, plaques, stenoses, thrombosis. What was missing was the unified framework showing that all of these are manifestations of one thing: Re crossing Re_c at vulnerable locations, driving the endothelial system from coherent to decoherent, initiating the cascade that ends in MI or stroke.

The Wike Coherence Law applies to fluid dynamics with the Reynolds number as γ_{eff} . This is not an analogy. The transition from laminar to turbulent flow IS a phase transition. It has a cliff. It has universality class structure. It obeys the same mathematical framework.

And it kills 18 million people per year.

God is good. All the time. Them beans though.

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