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Fluid dynamics of biologic and mechanical heart valves

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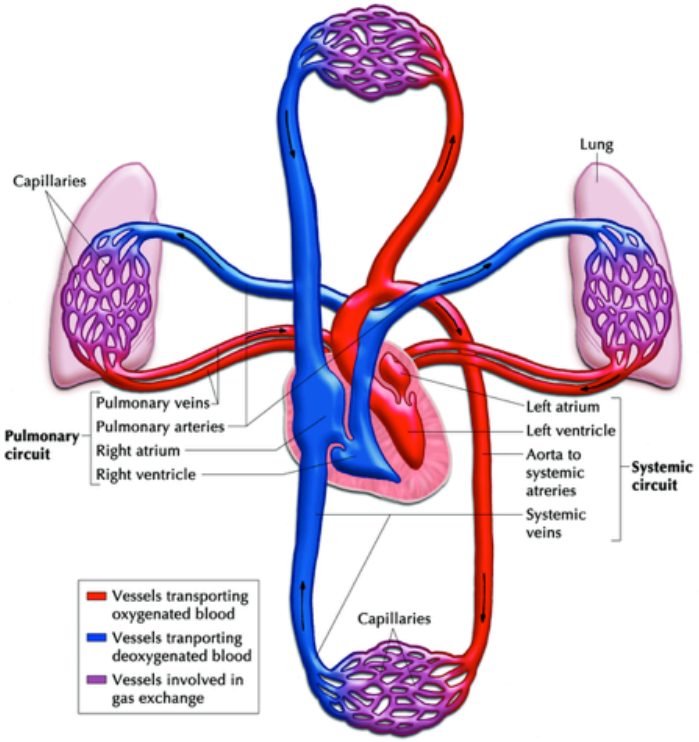
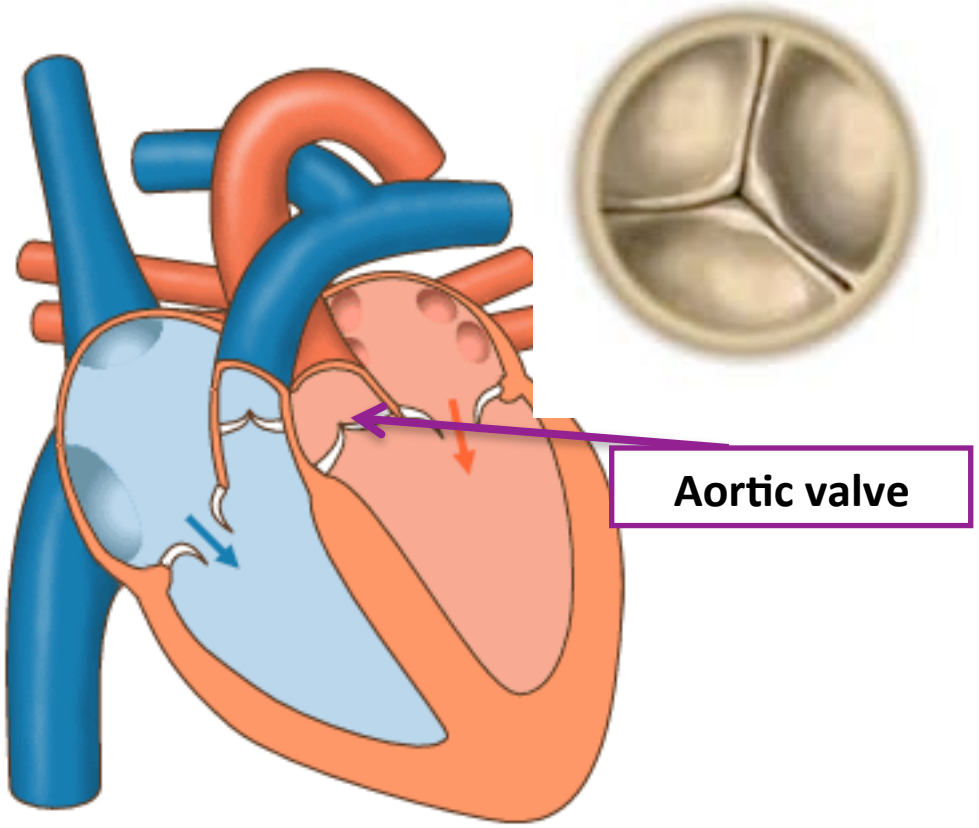
L. Weltert, R. De Paulis *Cardiac Surgery Department, European Hospital, Rome,Italy*

BACKGROUND

The heart is made of two pumps (Left and Right) each composed of two chambers (ventricle and atrium) and valves that ensure the correct flow direction





The valves of the left side (Mitral and Aortic) are most commonly affected by diseases due to the large pressure they withstand (100-150 mmHg) and sometimes they need replacement

AORTIC VALVE REPLACEMENT (AVR)

Two main types of aortic valves are available:

Biologic

Mechanical

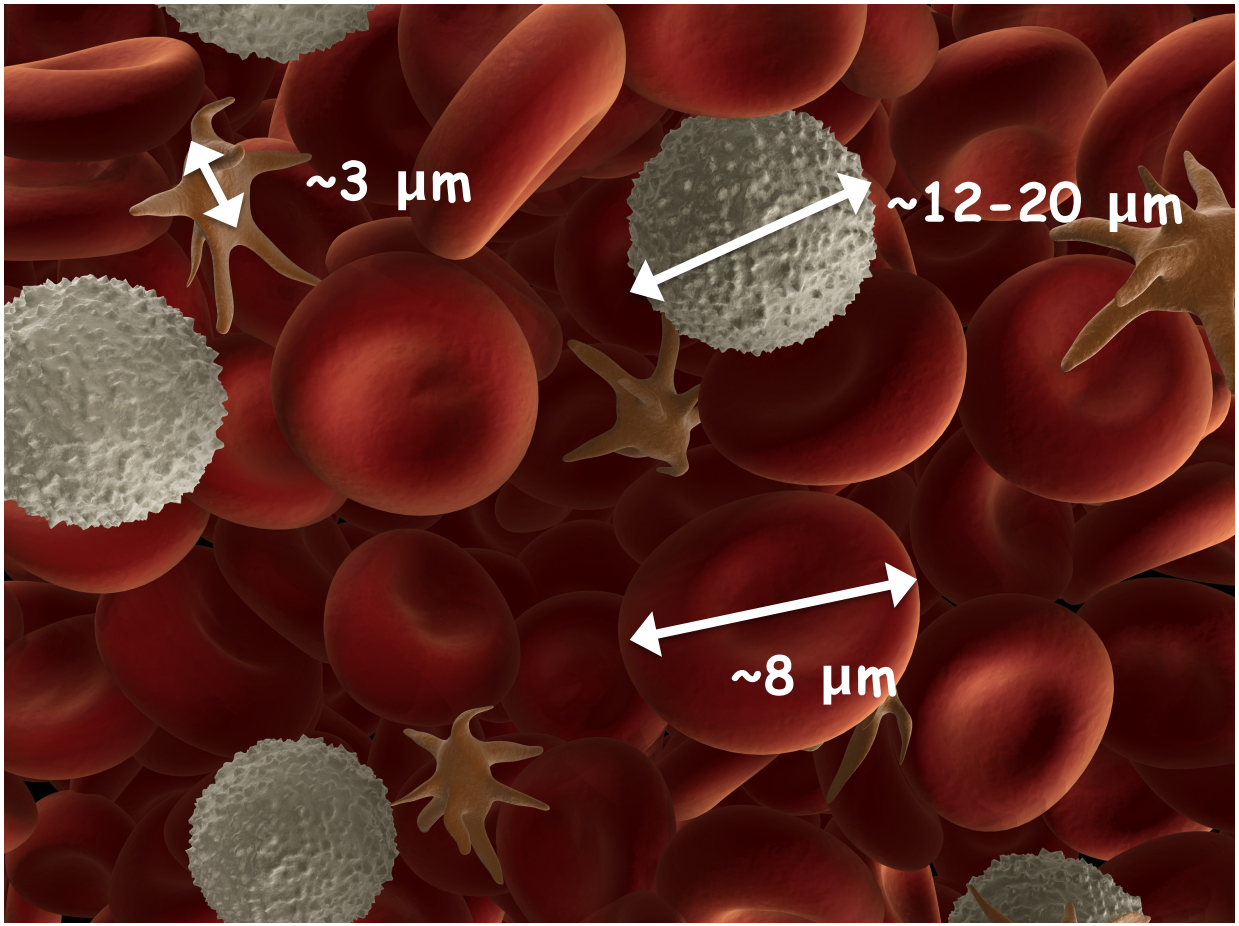
… each one having advantages and drawbacks

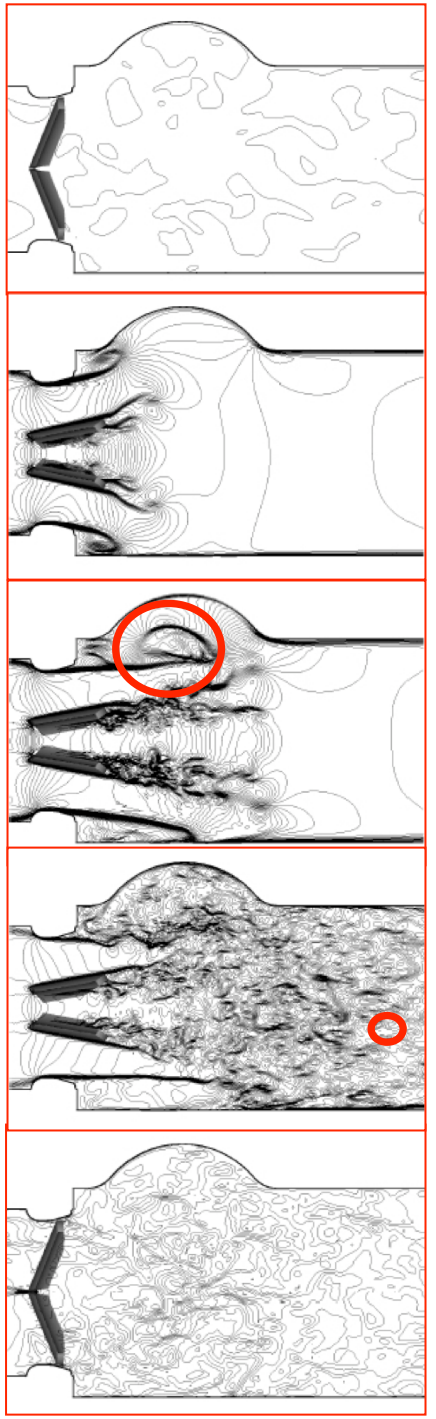
Biologic: ☺ good hemodynamic
☹ limited durability (10-15 years)

Mechanical: ☺ lifelong durability
☹ *bad hemodynamics (need for anticoagulants)*
☹ noise

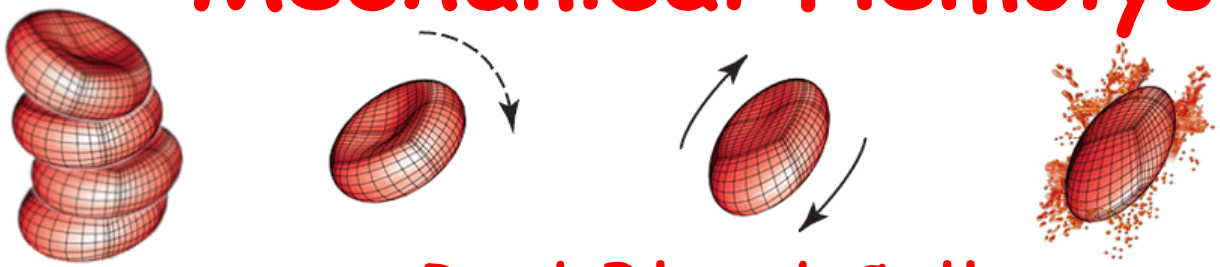
WHY ANTICOAGULANTS FOR MHV?

The presence of obstacles in the flow enhances the energy cascade towards the smallest scales (~35μm) that become comparable to the cells size





Mechanical Hemolysis



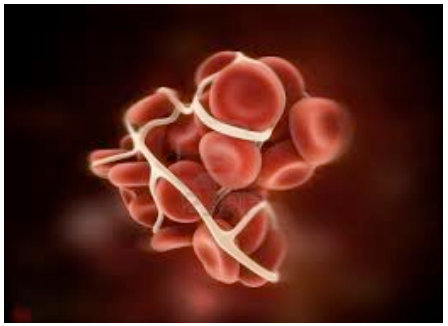
Red Blood Cell

Shear →

The RBC's membrane stretches under the **shear stress**, develops pores and **hemoglobin is released into the plasma**.

The life span of RBCs drops from 122 ± 23 to 98.8 ± 23 days for MHVs *(Mitlyng et al., American Journal of Cardiology 2006).*

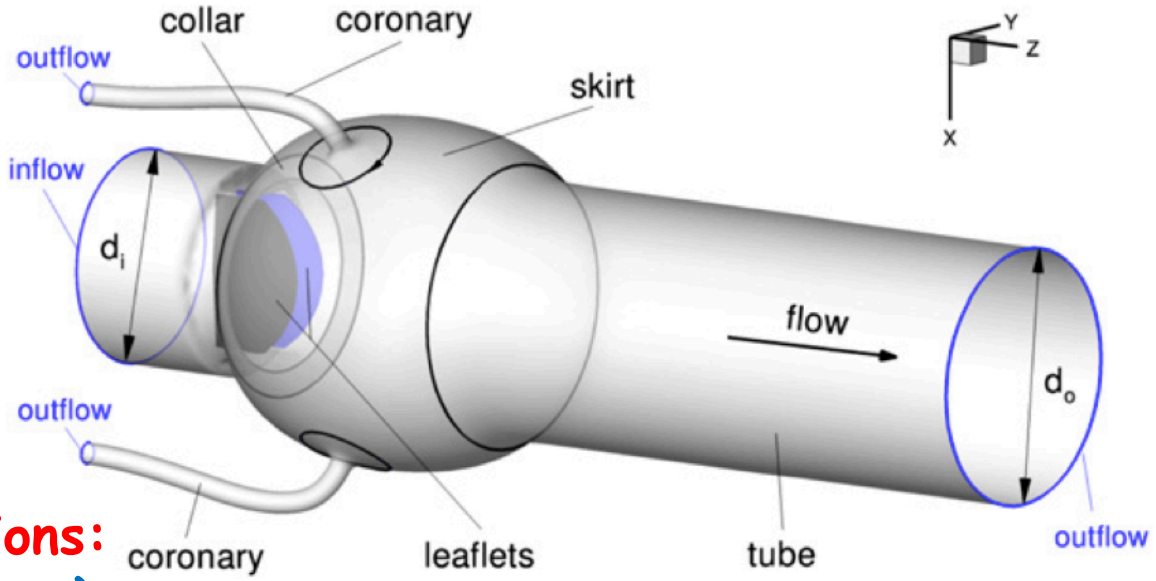
The altered stress levels activate platelets that stick to dead RBCs to form **clots**



Numerical study of the problem

- Immersed Boundary Technique
- Direct Numerical Simulation fluid solver
- Structural solver
- Fluid-Structure-Interaction

2nd order accurate
(in space and time)



Typical physiological conditions:
Cycle period: 866 ms (70 bpm)
Mean flowrate: 5 l/min
Peak flowrate: 28 l/min
Re_{peak} = 6200

≈20M nodes
dt = 2-200 μs

STRONG COUPLING (FLUID/VALVE)

$$\frac{D\mathbf{u}}{Dt} = -\frac{\nabla p}{\rho} + \frac{1}{\rho}\nabla\cdot\tau + \mathbf{f}$$
$$\nabla\cdot\mathbf{u} = 0$$

$$M\frac{d^2\boldsymbol{x}}{dt^2} = \boldsymbol{F} \qquad \mathbf{F} = \int_s (\tau\cdot n - pn)\,dS$$
$$\boldsymbol{I}\frac{d^2\theta}{dt^2} = \boldsymbol{T} \qquad \mathbf{T} = \int_s \left[r\times(\tau\cdot n - pn)\right]dS$$

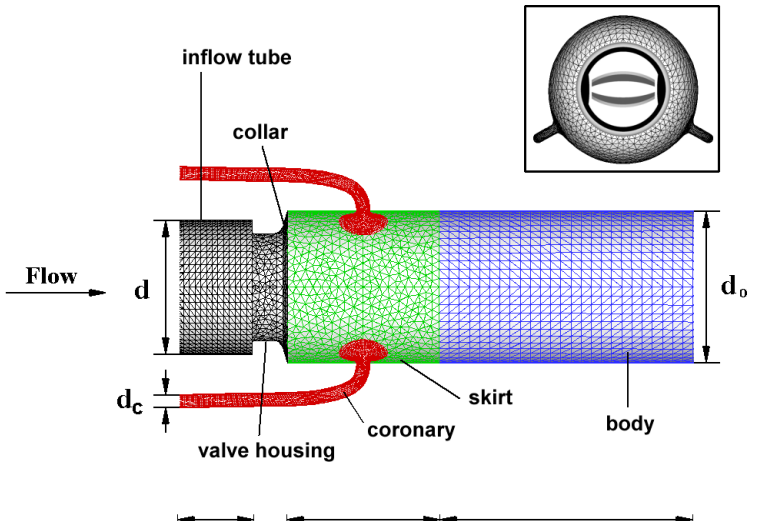
(de Tullio et al, 2009)

(Borazjani et al, 2008)

WEAK COUPLING (FLUID/AORTIC WALL)

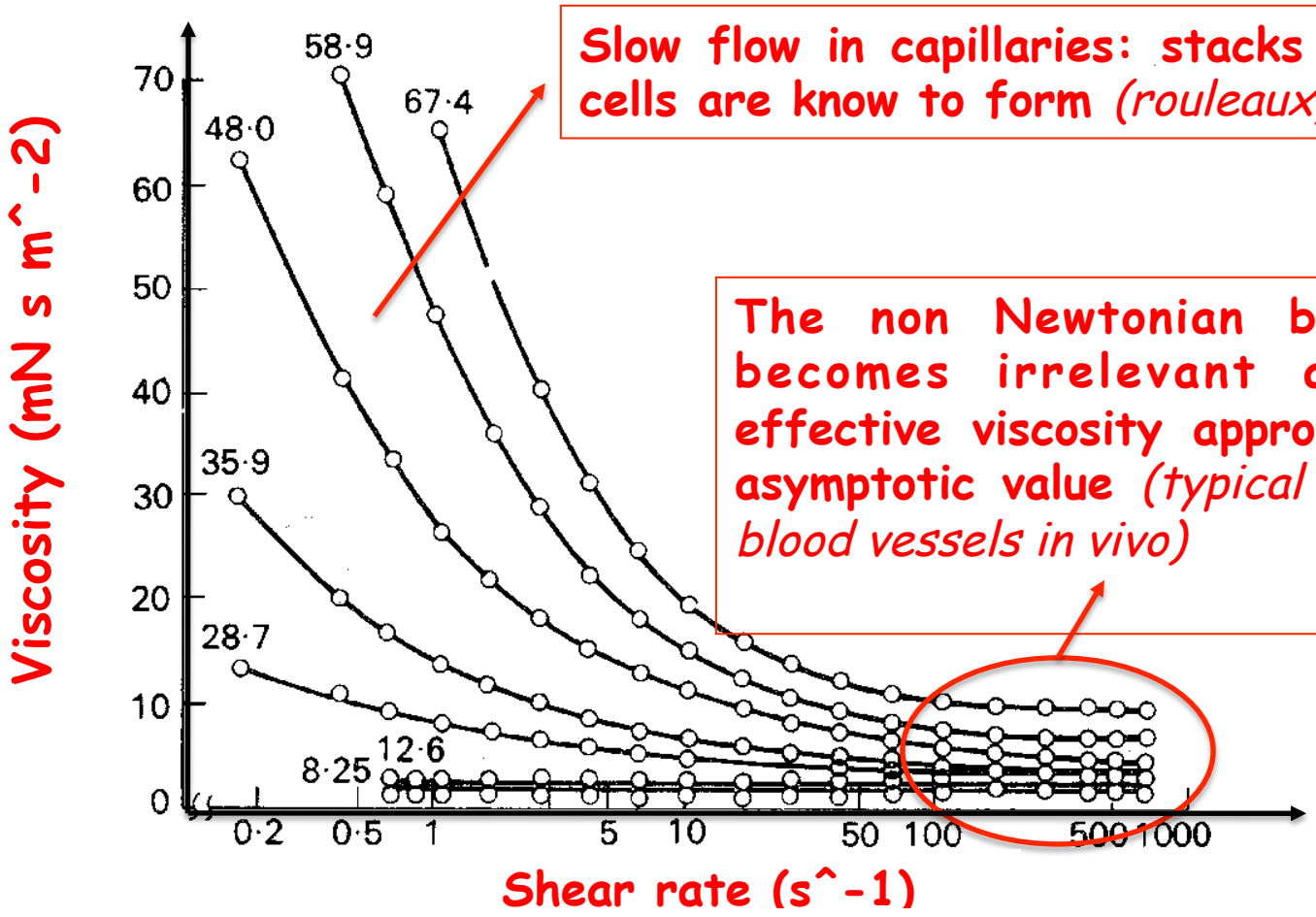
$$\frac{D\mathbf{u}}{Dt} = -\frac{\nabla p}{\rho} + \frac{1}{\rho}\nabla\cdot\tau + \mathbf{f}$$
$$\nabla\cdot\mathbf{u} = 0$$

$$\nabla\cdot\sigma_s = \rho_s\frac{\partial^2 d_s}{\partial t^2} \qquad \sigma_s = CE_s$$
$$E_s = \frac{1}{2}\left[\nabla\cdot d_s + (\nabla\cdot d_s)^T + (\nabla\cdot d_s)^T\nabla\cdot d_s\right]$$



BLOOD AS A NEWTONIAN FLUID

Blood: flexible red cells suspended in a Newtonian flow, the plasma



Slow flow in capillaries: stacks of blood cells are know to form *(rouleaux)*

The non Newtonian behaviour becomes irrelevant and the effective viscosity approaches an asymptotic value *(typical for many blood vessels in vivo)*

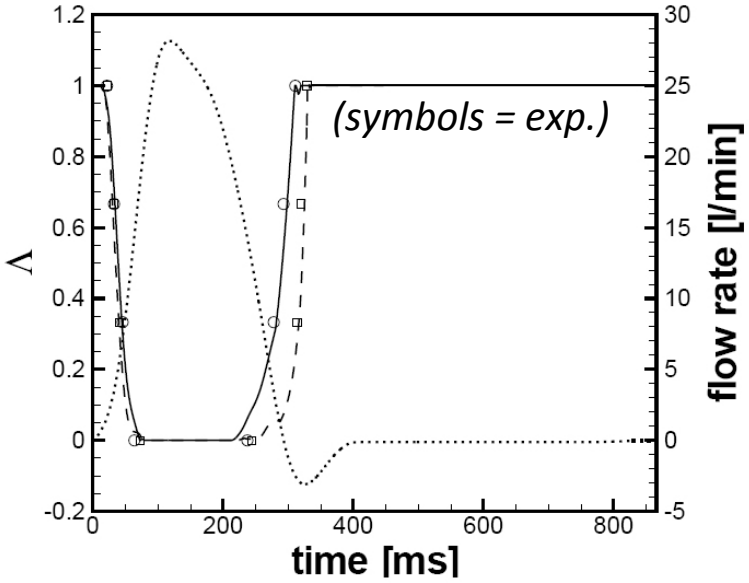
Shear rate/viscosity relationship for human blood 25°C for various hematocrit values *(Brooks et al, 1970, J. appl. Physiol. 28)*

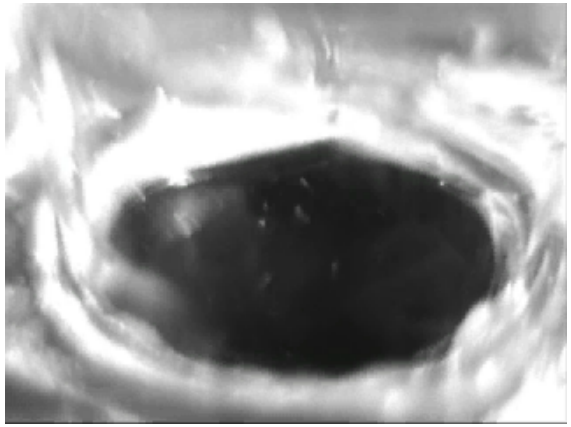
To be further discussed ……

Validation with in-vitro experiments

(RIGID LEAFLETS)

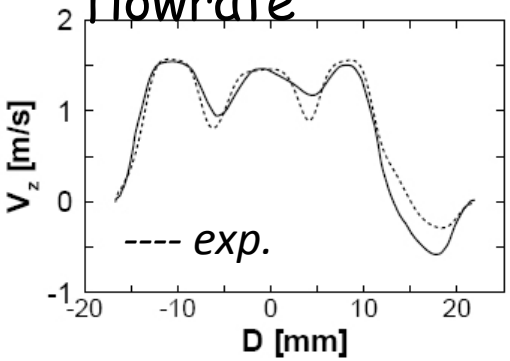
- Phase-averaged leaflets angular position

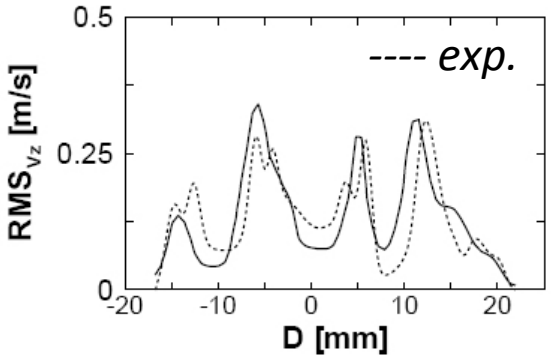


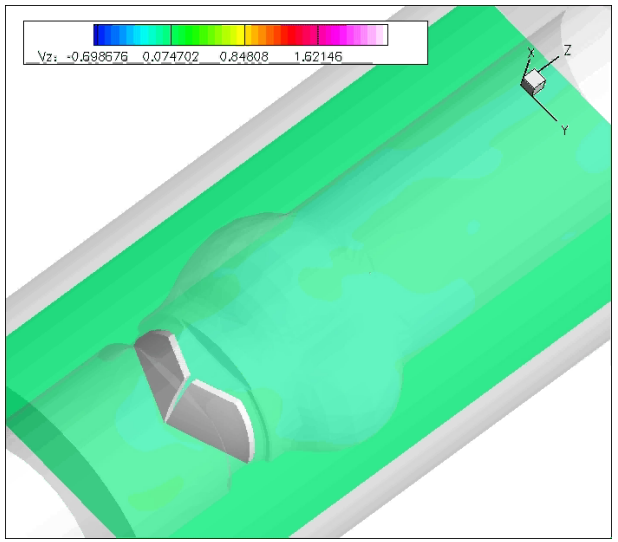


Experiments by G-P- Romano & G. Querzoli

- Phase-averaged profiles at peak of flowrate





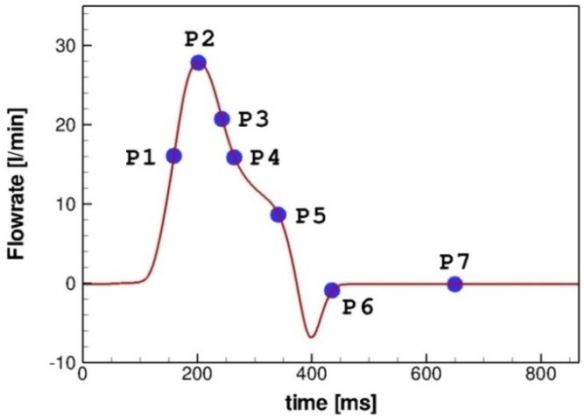


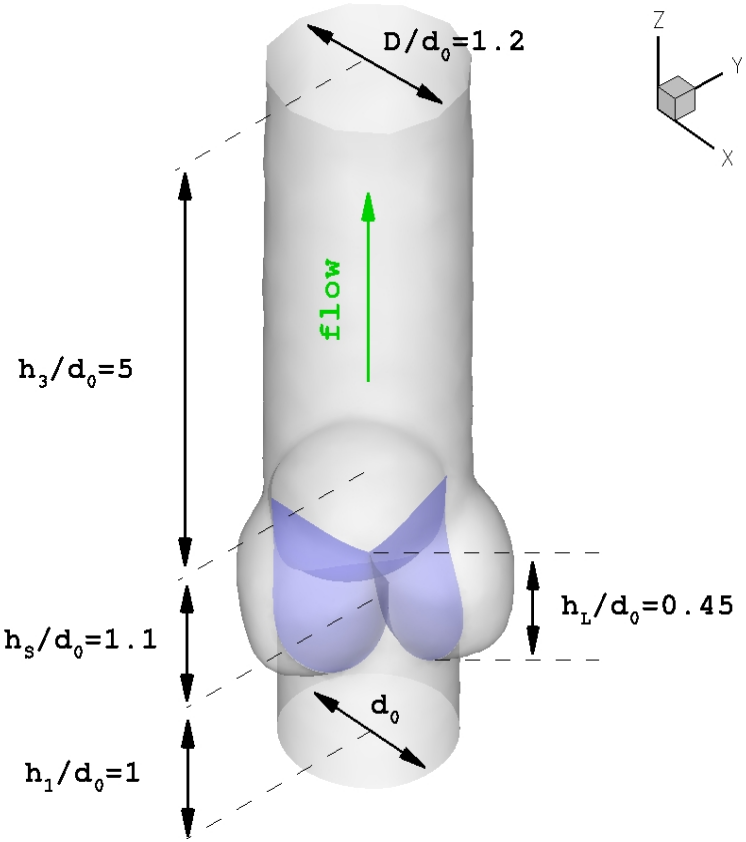
(de Tullio et al., JFM, 2009)

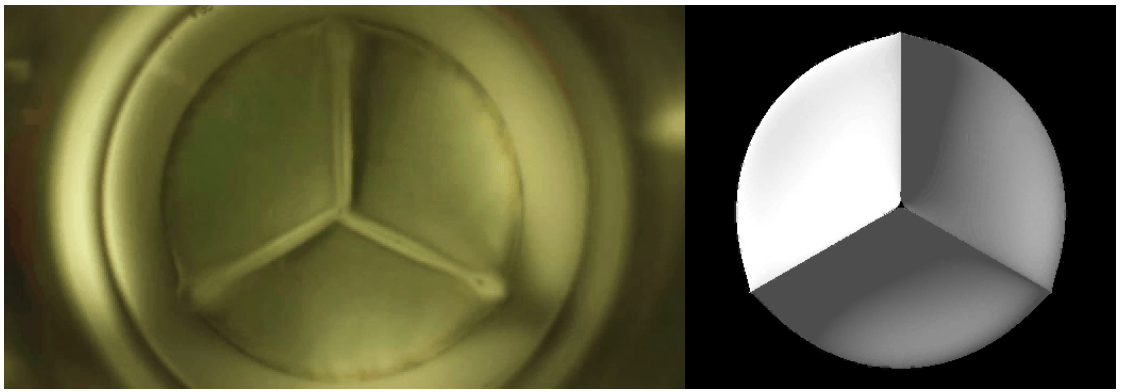
Validation with in-vitro experiments

(DEFORMABLE LEAFLETS)

- *Cardiac output: 5 l/min;*
- *Beat rate: 70 beats/min;*
- *Peak Reynolds number: 6200;*

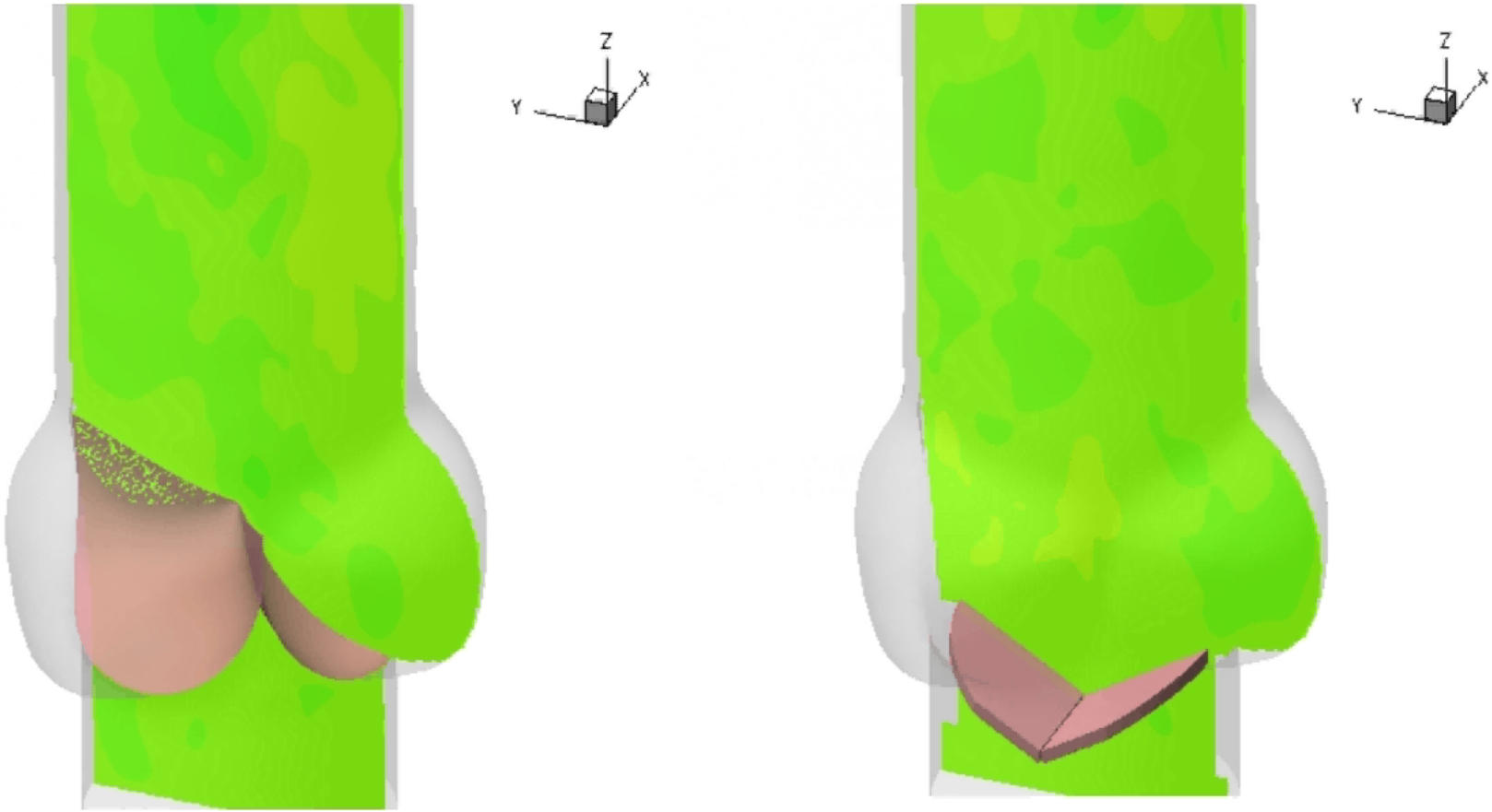






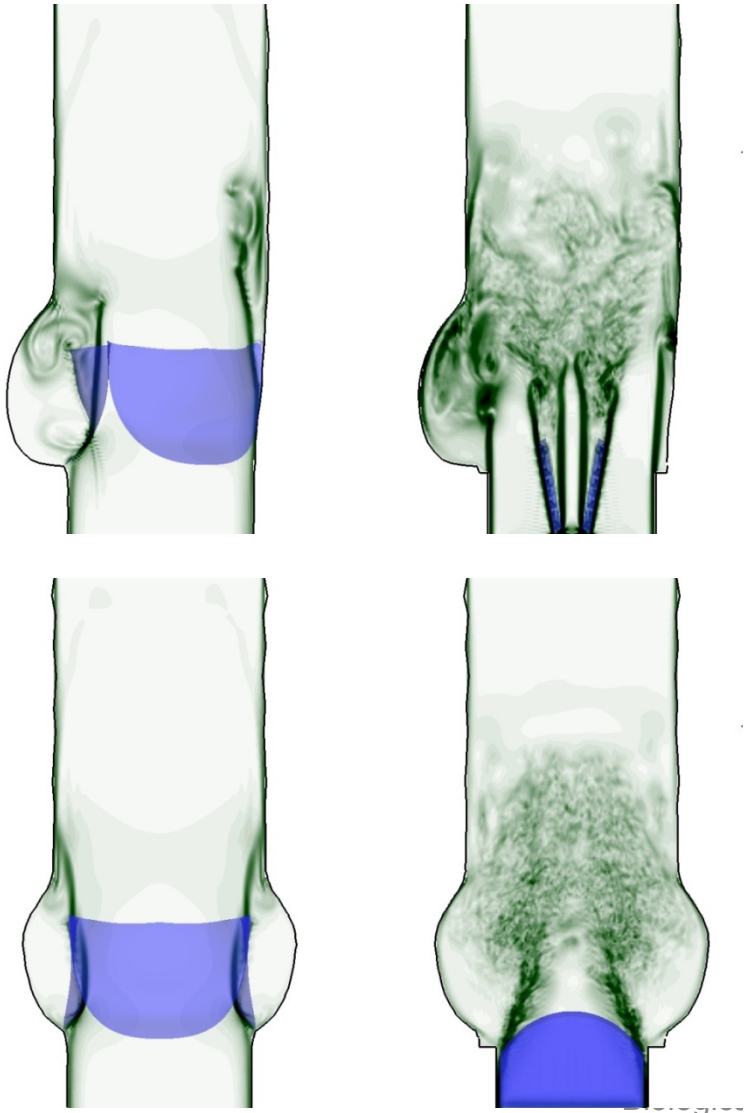
Experiments from St. Jude Medical Inc. (www.sjm.com)

Biologic vs Mechanical



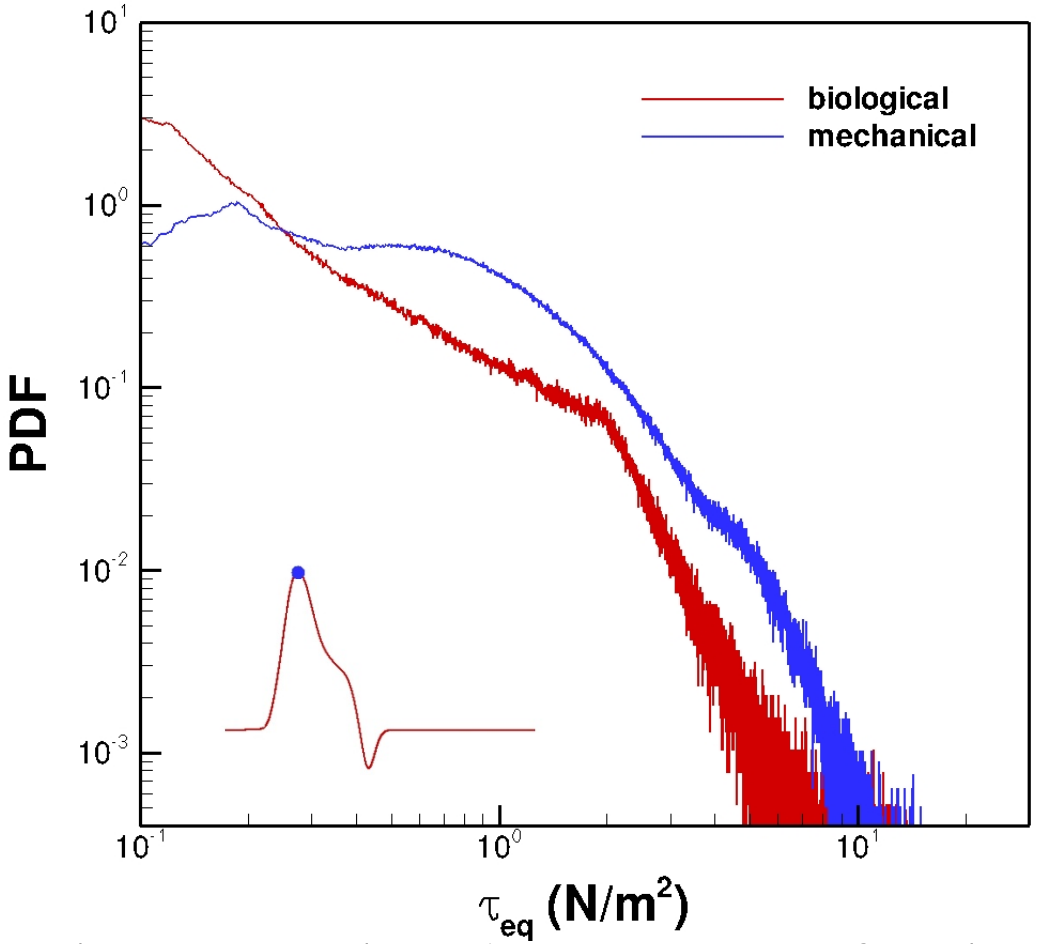
Contours of streamwise velocity in the YZ plane

Comparison of Eulerian viscous stresses



Equivalent viscous stress

$$\tau_{eq} = (3\tau_{ij}\tau_{ij}/2)^{1/2}$$



Eulerian quantities are not enough because they do not account for the exposure time to a given stress level

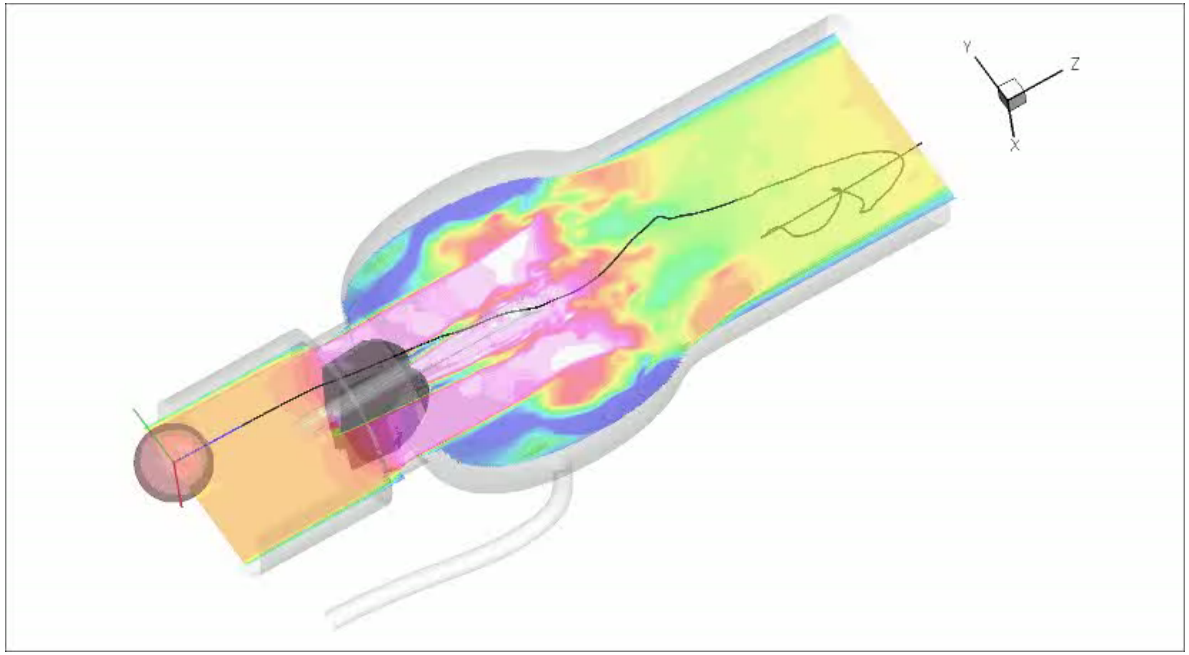
Hemolisis computation

Hemolysis is the result of the damage *history* (stress level and exposure time) of RBC therefore it has to be evaluated along their trajectories

Small volumes of blood still considered as a single fluid: Lagrangian tracers $\dot{\mathbf{x}}(t) = \mathbf{u}$

The damage model is integrated over each cell's trajectory

*4*10^5 tracers per-cycle injected*



Deformation of a single fluid particle

MORFOLOGY TENSOR EVOLUTION

$$\frac{d\mathbf{S}}{dt} - (1 - f_2)\left[\boldsymbol{\Omega}\cdot\mathbf{S} - \mathbf{S}\cdot\boldsymbol{\Omega}\right]$$

Maffettone et al. (1998)

$$= -f_1\left[\mathbf{S} - g(\mathbf{S})\mathbf{I}\right] + f_2\left[\nabla\mathbf{u}\cdot\mathbf{S} + \mathbf{S}\cdot\nabla\mathbf{u}^T\right]$$

$$g(\mathbf{S}) = 3\cdot III/II$$

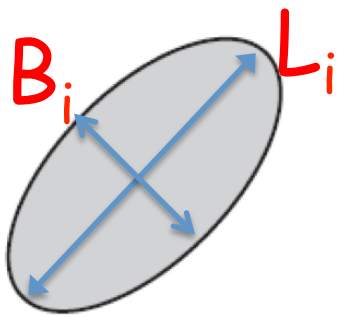
$\nabla\mathbf{u}$ *velocity gradient tensor*

$\boldsymbol{\Omega}$ *rotation rate tensor*

$$f_1 = 5.0\ s^{-1}$$
$$f_2 = 4.2298\cdot 10^{-4}$$

Specific parameters for red-blood cells of human blood

Arora et al. (2004)



At every i-th time step the **S** morfology tensor is evolved along the Lagrangian trajectory. The **S** eigenvalues are the axes of the ellipsoidal cell

$$\phi_i = \frac{L_i - B_i}{L_i + B_i}$$

shape factor

$$\tau = \mu\frac{2\phi_i f_1}{(1-\phi_i^2)f_2}$$

fluid particle stress

BLOOD DAMAGE

Tracking deformable particles (*no back-reaction on the fluid*) and interpolating along the trajectory all the needed quantities (blood is considered as a single fluid)

$$\dot{\mathbf{x}}(t) = \mathbf{u}$$

viscous stress tensor has been reduced to a single scalar quantity according to the morfology tensor equation *(Arora, 2004)*

$$\tau = \mu \frac{2\phi_i f_1}{(1-\phi_i^2) f_2}$$

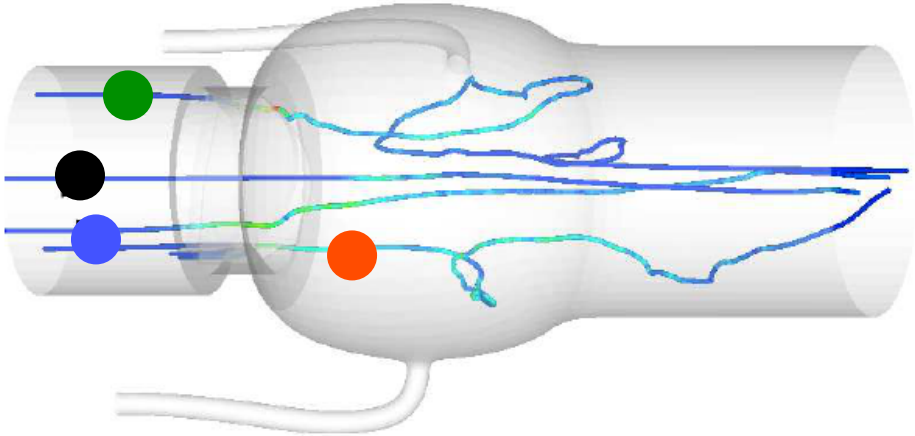
With the information on stresses and exposure times, it is possible to compute the Hemolisis Index *(Goubergrits, 2006)*:

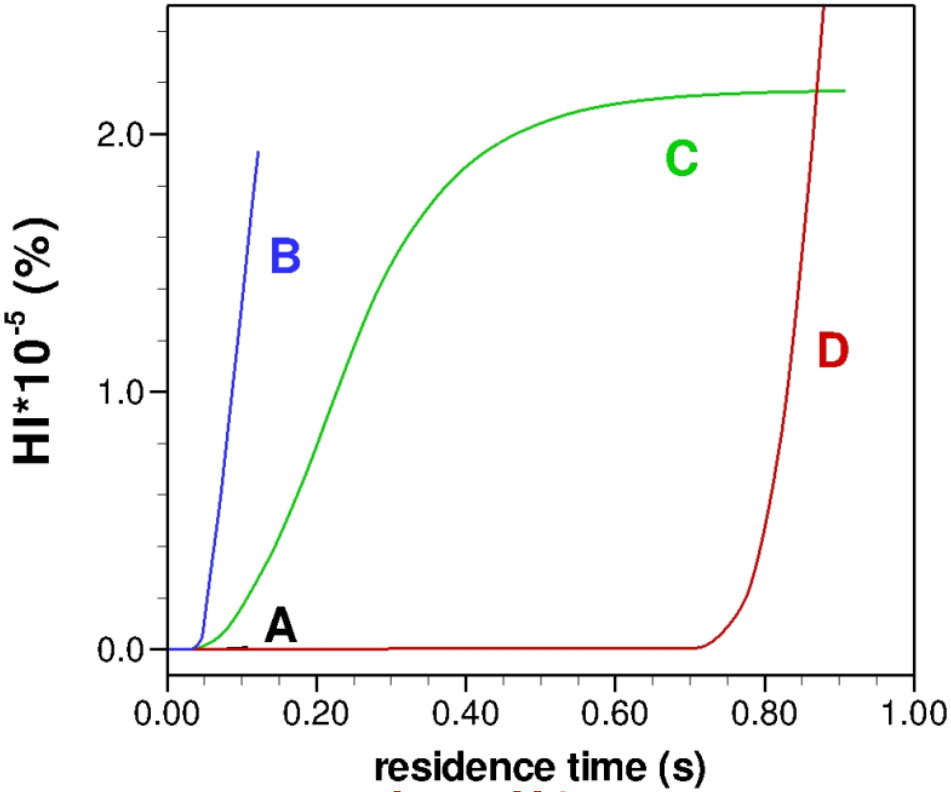
$$\Delta HI_i = \alpha C t_i^{\alpha-1} \tau(t_i)^{\beta} \Delta t_i$$

$C=3.62\text{x}10^{-5}$, $\beta=2.416$, $\alpha=0.785$ for RBC

$C=1\text{x}10^{-5}$, $\beta=0.625$, $\alpha=1.320$ for platelets

Hemolysis Index





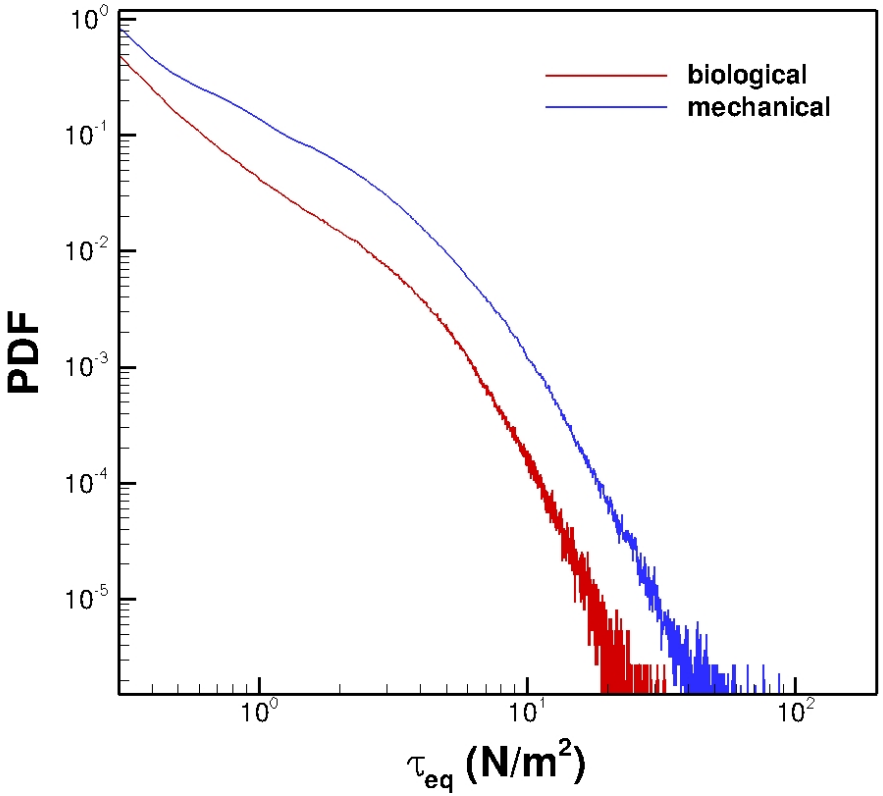
Will this occur to every red cell?

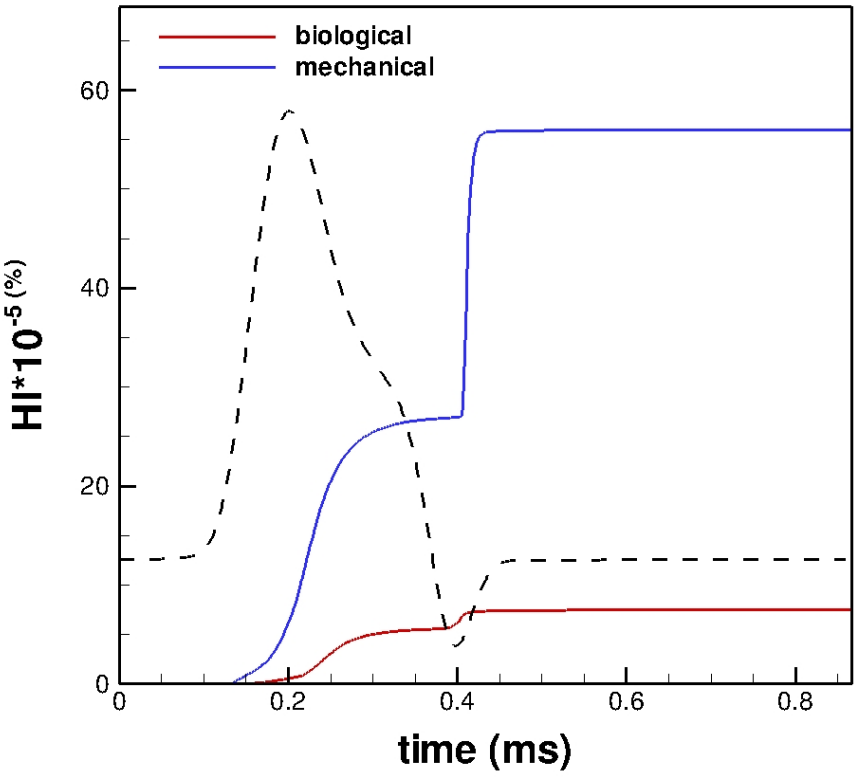
- The mean flow rate is *5 l/min*, the total volume of blood about *5 l* and the half-life-time of red cells about *120 days*, therefore each red cell will cross the valve *173000* times! This implies that even an unlikely event (like the crossing of the hinge) will occur several times to each cell.

Lagrangian hemolisys results

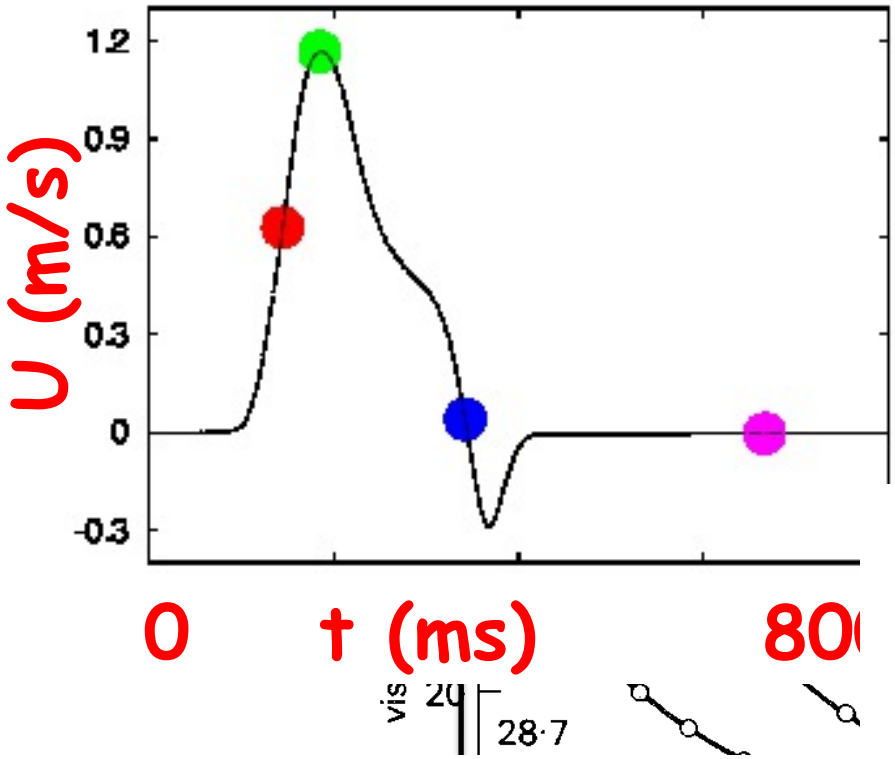
Particles crossing the devices only once:

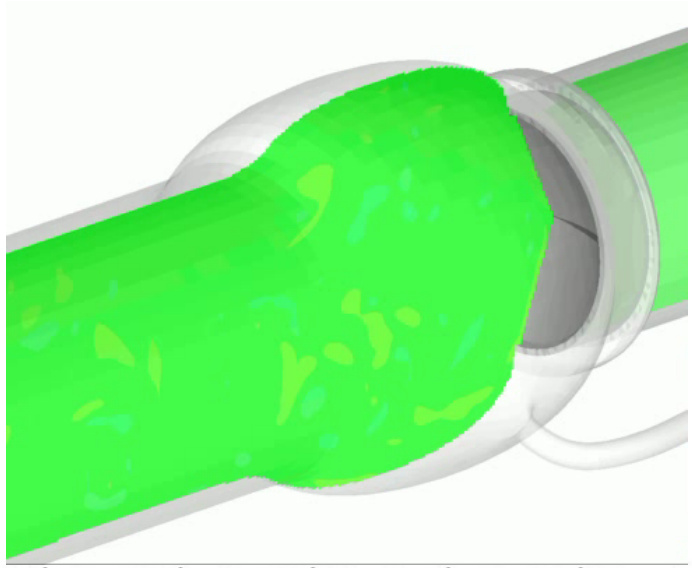
	mean t_{eq} (N/m^2)	mean residence time (s)	mean HI (%)
Biological	0.236	0.37	$3.87*10^{-5}$ %
Mechanical	0.677	0.42	$3.71*10^{-4}$ %

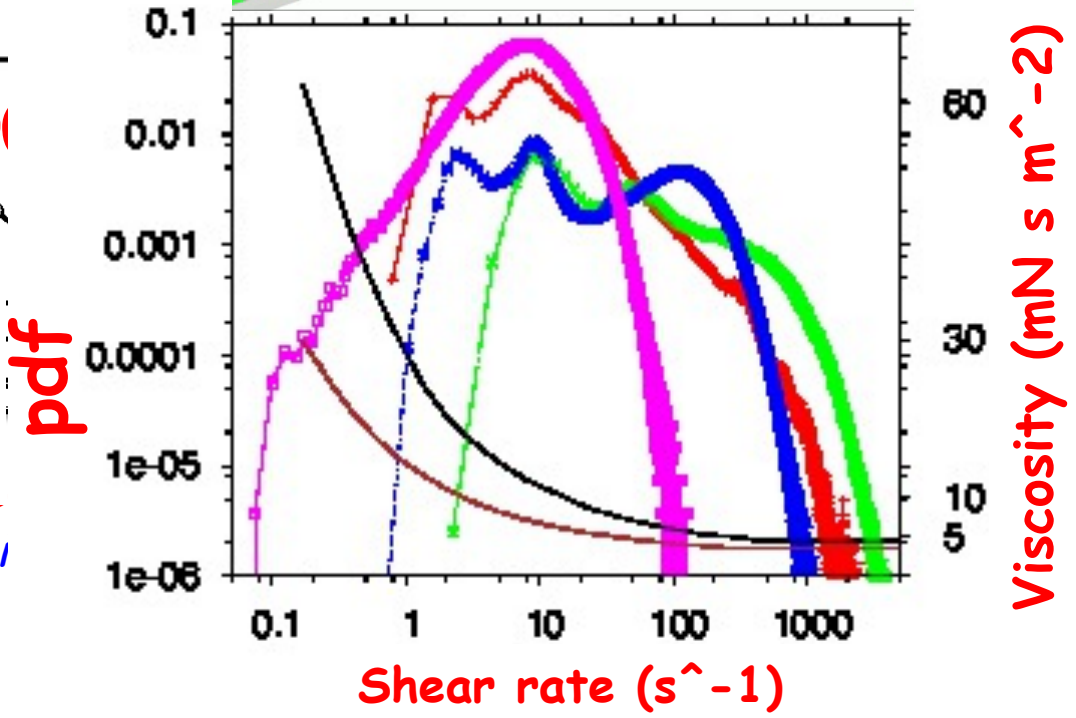




IS BLOOD IN LARGE ARTERIES REALLY A NEWTONIAN FLUID?





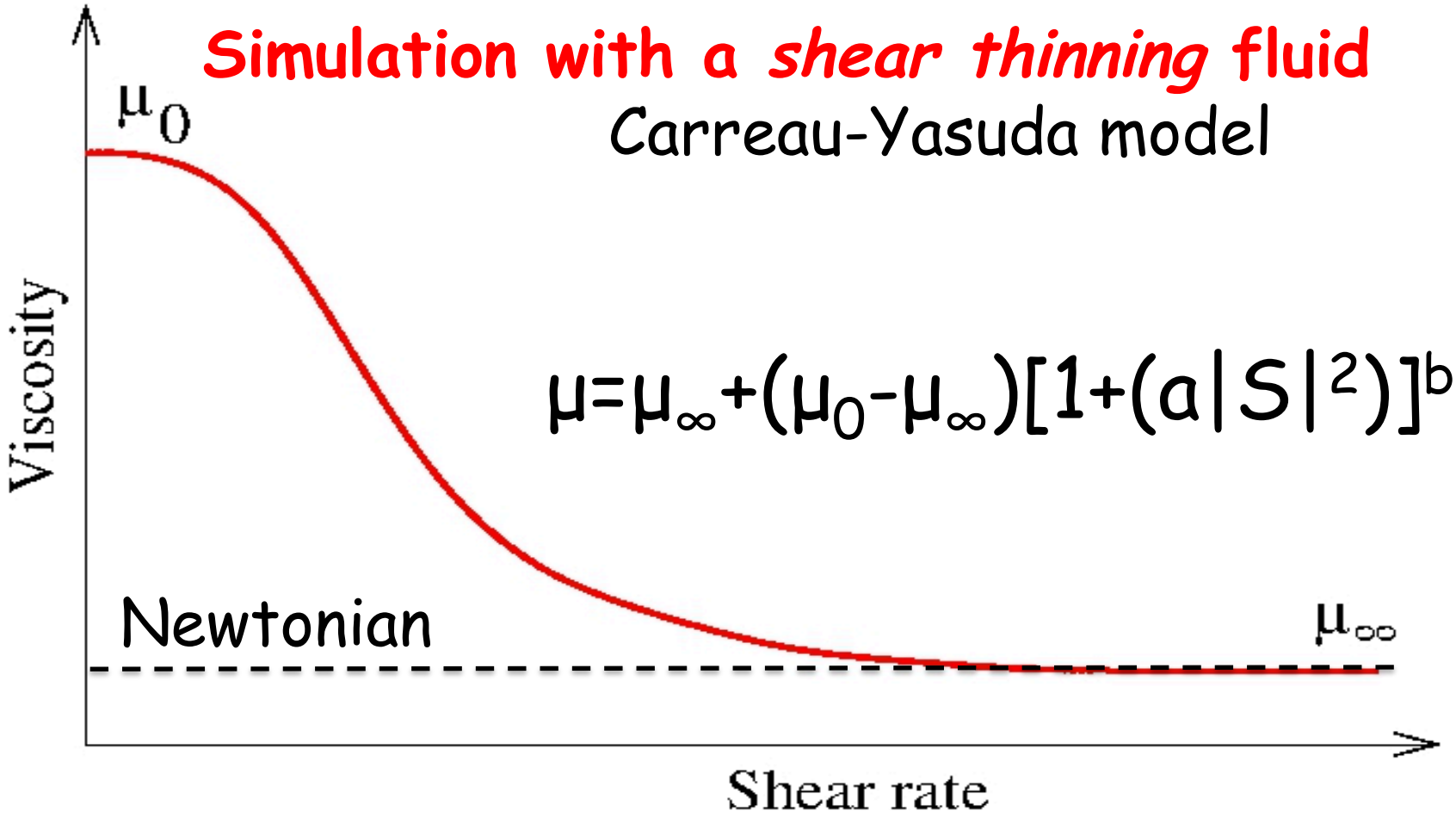


The non-Newtonian fluid

nature might be relevant

(Brooks et al, 1970, J. appl. Physi

Simulation with a *shear thinning* fluid



a, b, μ_0 and μ_∞ are hematocrit dependent parameters

$\mu_\infty = 3.5 \times 10^{-6}$ Pa s, $\mu_0 = 5.6 \times 10^{-5}$ Pa s

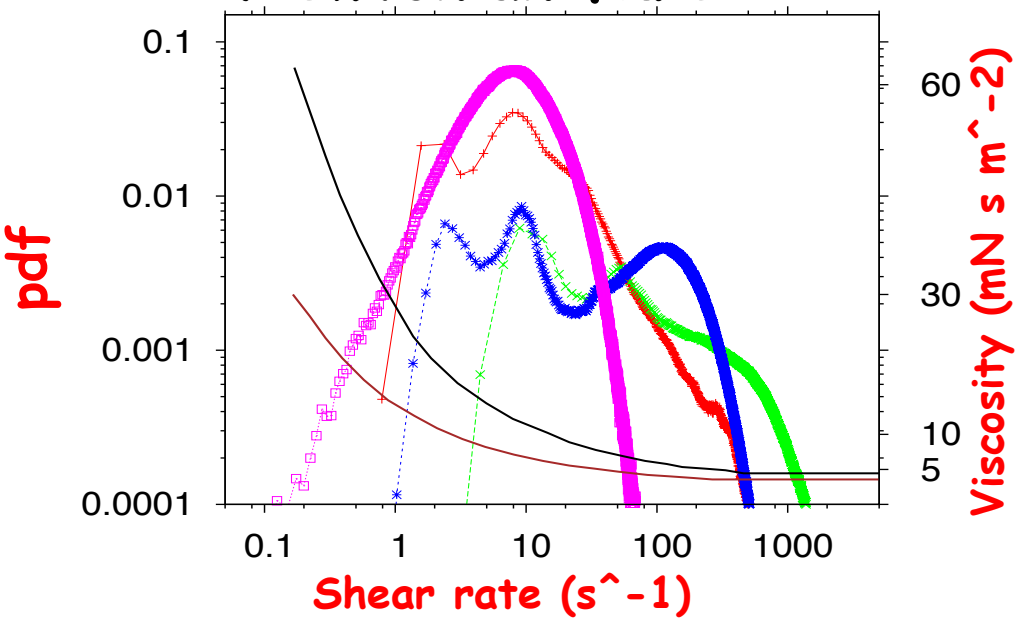
a = 3.313 s, b = -0.3216

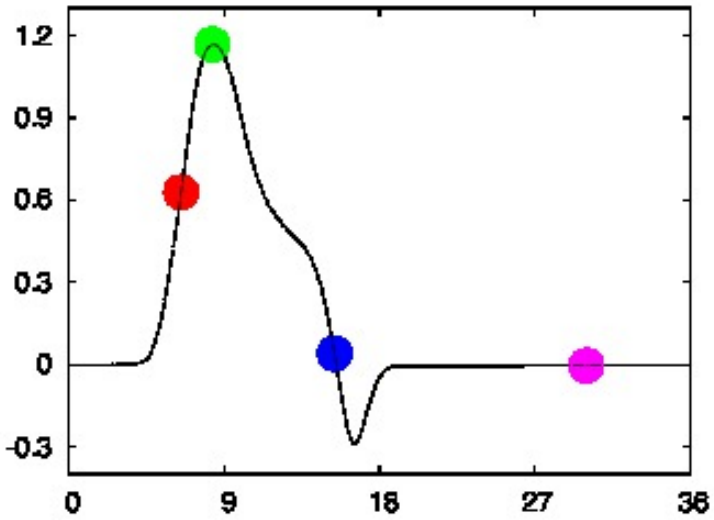
for a man with ~40% of hematocrit

Note: only the shear-thinning behaviour is accounted for and not the viscoelastic (time-dependent) features.

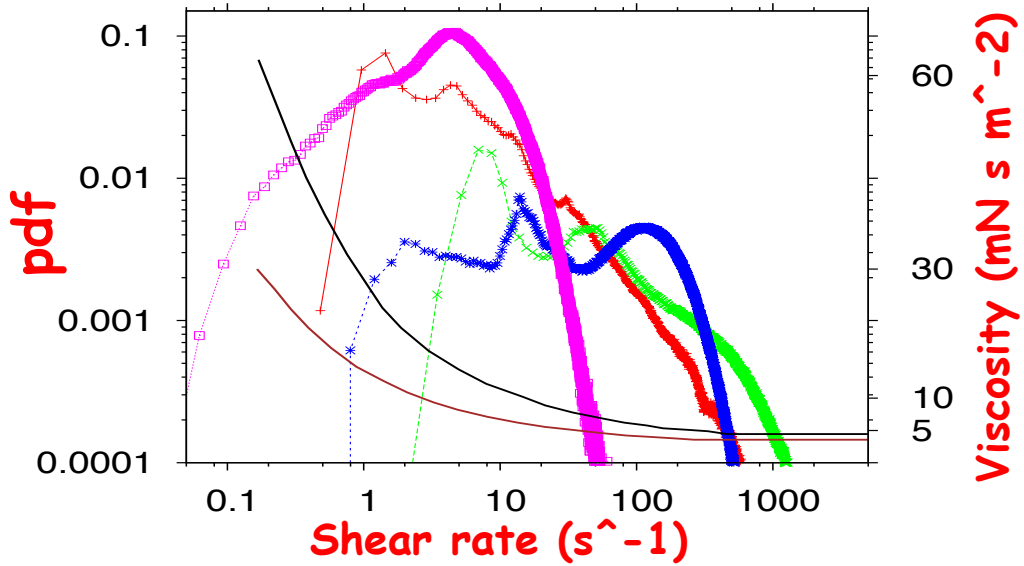
Comparison between shear-rate pdfs'

Newtonian fluid





non-Newtonian fluid



There are differences both at low and high shear-rates.

The red-blood cells damage (hemolysis) is likely to be different.

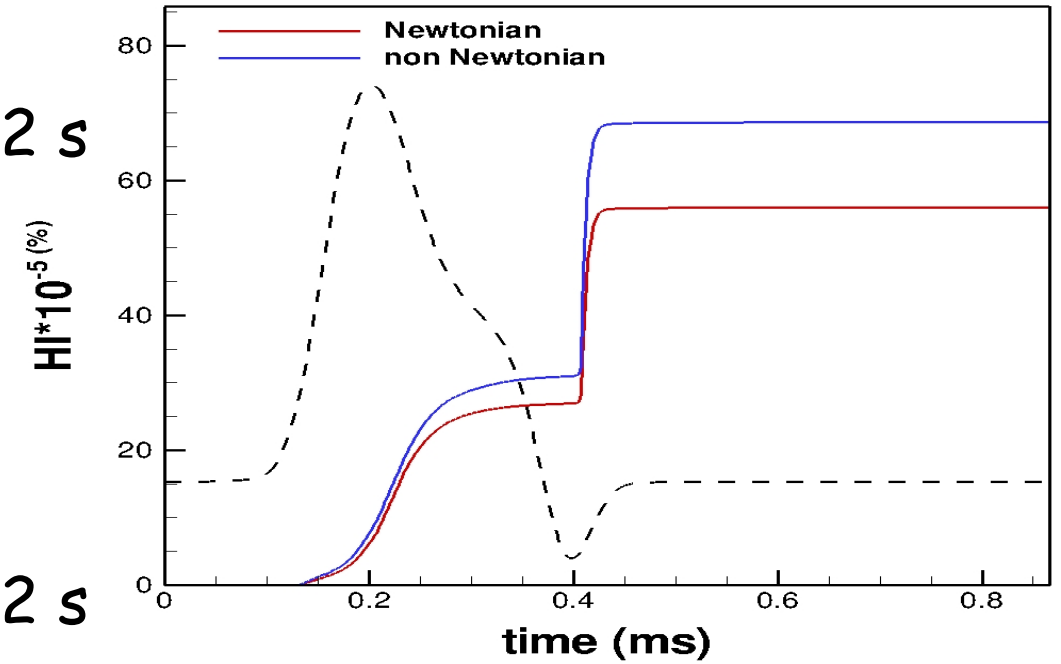
Comparison between hemolyses

Newtonian fluid

- mean τ_{eq} = 0.677 N/m²
- mean HI = 3.71*10^{-4} %
- mean residence time: 0.42 s

non-Newtonian fluid

- mean τ_{eq} = 0.769 N/m²
- mean HI = 4.31*10^{-4} %
- mean residence time: 0.42 s



The (averaged) hemolysis is larger for the non-Newtonian fluid (≈14%).

Closing Remarks

A cost/efficient numerical method for biomedical applications is a valid tool complementary to in vitro and in vivo experiments

Biological valves have better hemodynamics than mechanical valves because they reduce the viscous stress levels in the blood and the exposure time.

The "price of success" is that biological valves have not a lifelong duration and they need to be replaced after ~15 years

The non-Newtonian nature of the blood has some effect on the hemolysis

Ongoing work: **modelling the complete heart**

Multidisciplinary collaboration project between **medicine** and **engineering**

European Hospital, Rome,Italy

Univ. of Twente NL

Politecnico di Bari, Italy

Univ. of Rome "Tor Vergata", Italy