THE GEOMETRICAL MULTISCALE MODELING IN HEMODYNAMICS

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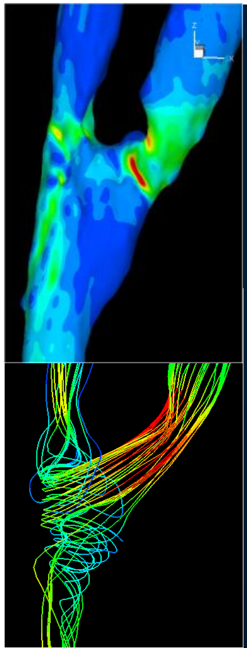


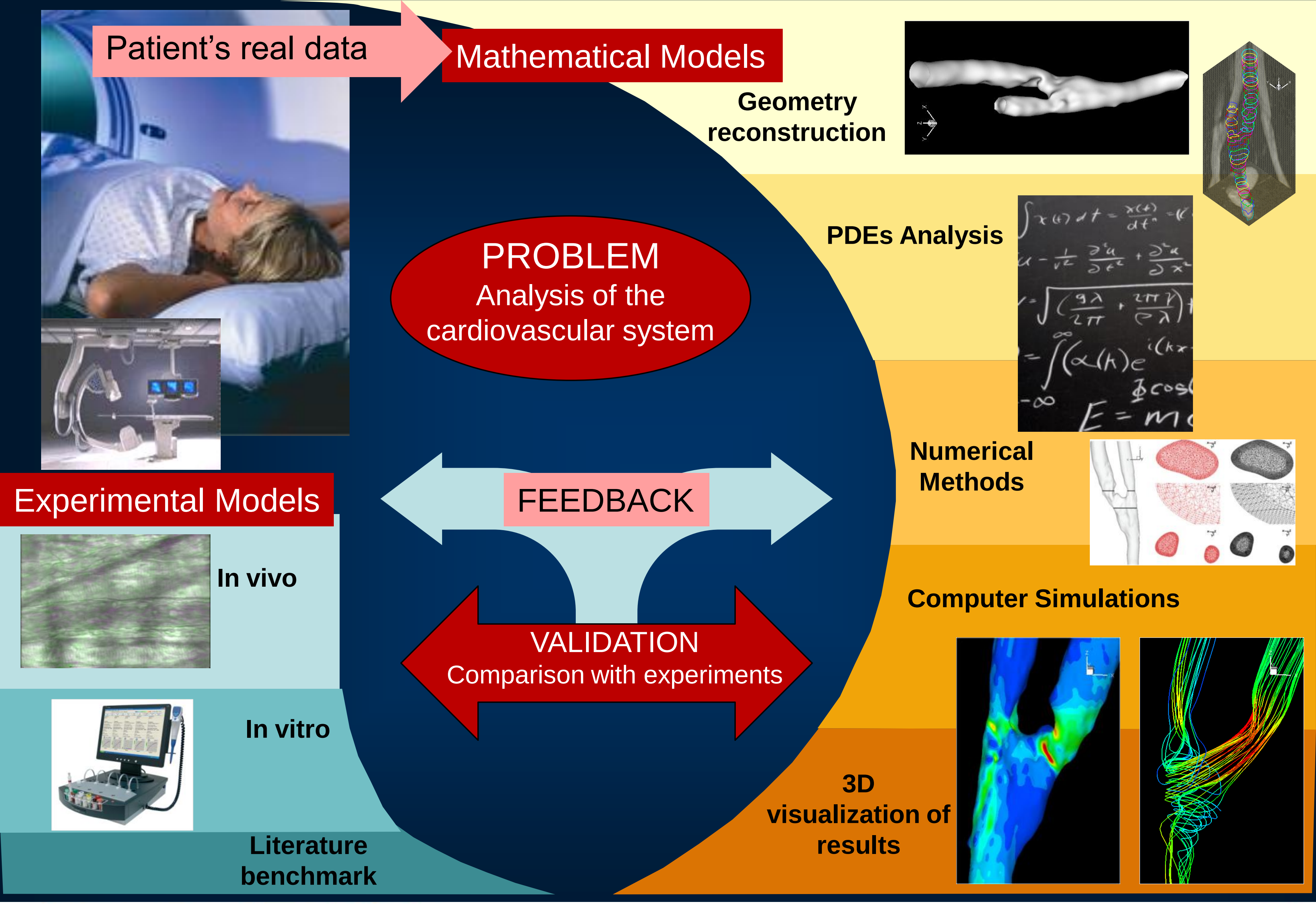
ADÉLIA SEQUEIRA

Workshop on Nonlinear PDE's

Dedicated to 80th anniversary of birth of Prof. Jindřich Nečas

December 13-15, 2009

Czech Academy of Sciences, Prague (Czech Republic)



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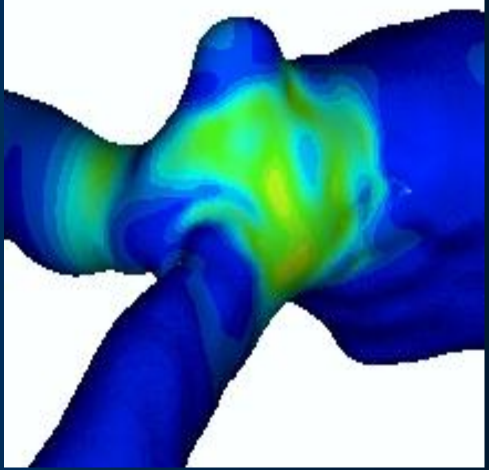
MOTIVATION

Hemodynamics vs cardiovascular diseases: local fluid patterns and **wall shear stress** are strictly related to the development of cardiovascular diseases (indicator of atherosclerosis)

$$WSS = \mu\left(\frac{\partial \mathbf{u}}{\partial \mathbf{n}} \cdot \boldsymbol{\tau}\right)\Big|_{wall}$$



WSS pulmonary artery (congenital heart disease)

➢ Difficulties in modeling blood flow

- **Blood Rheology**
- **Complex geometry** → 3D flow simulations are restricted to specific regions of interest
- **Closed system** → Local flow dynamics has an important role in the systemic circulation (and vice-versa)

MATHEMATICAL MODEL

1. Blood rheology

2. Fluid-structure interaction

3. Geometrical multiscale approach

CIRCULATORY SYSTEM: FLUID DYNAMIC VALUES

Relationship between arterial size, number of vessels and average Reynolds numbers

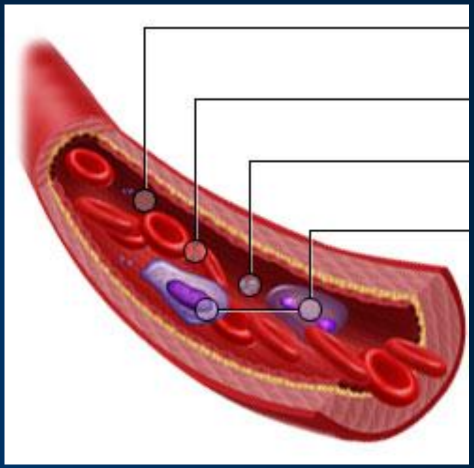
Vessel	Radius (cm)	Number	Wall thickness (cm)	Average Re number
Aorta	1.25	1	0.2	3400
Arteries	0.2	159	0.1	500
Arterioles	1.5×10^{-3}	5.7×10^{7}	2×10^{-3}	0.7
Capillaries	3×10^{-4}	1.6×10^{10}	1×10^{-4}	0.002
Venules	1×10^{-3}	1.3×10^{9}	2×10^{-4}	0.01
Veins	0.25	200	0.05	140
Vena cava	1.5	1	0.15	3300

Turbulence can develop in a few cases:

High cardiac output (exercise); Stenoses; Low blood density (for example: anemia)

BLOOD COMPOSITION

- Blood is a suspension of
  - cells
    - erythrocytes (RBCs)
    - leukocytes (WBCs)
    - platelets
  - plasma (90-92% water + proteins, organic salts)



	Number/ mm^3	Shape (unstressed)	Size µm (unstressed)	Volume Conc.
erythrocytes	$4\text{-}6\times10^6$	Biconcave discs with no nuclei	8×1-3	45%
leukocytes	$4\text{-}11\times10^3$	roughly spherical	7-22	1%
platelets	$2.5\text{-}5\times10^5$	Rounded or oval discs	2-4	

BLOOD RHEOLOGY

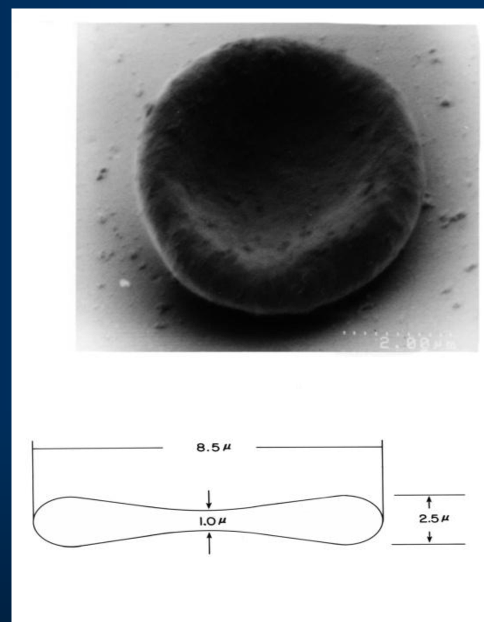
- Why is blood a non-Newtonian fluid ?

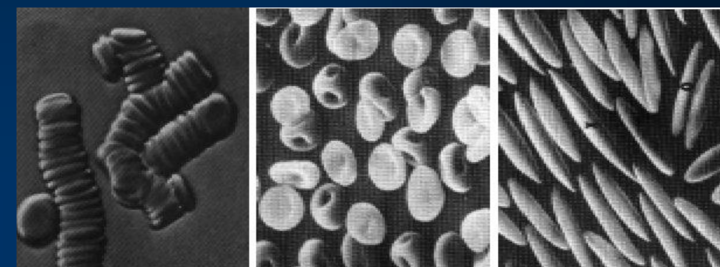
▶ Non-Constant Viscosity ◀

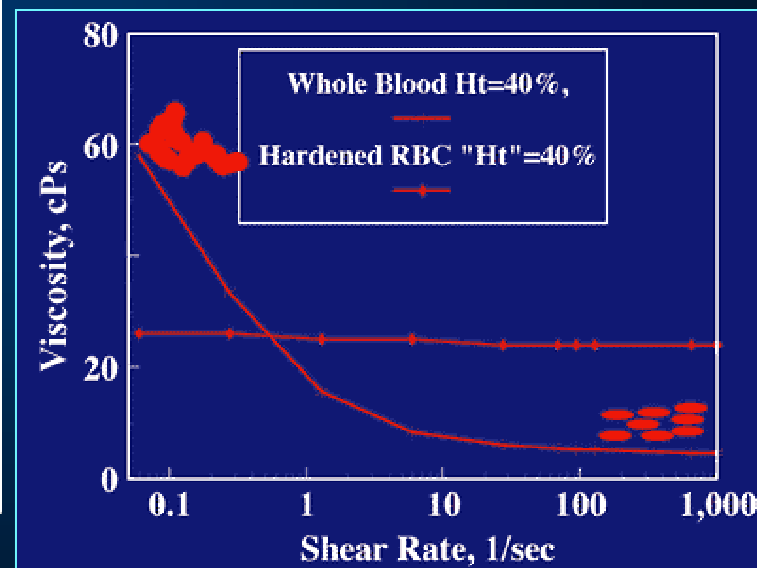
Main Factors:

RBC aggregation and deformability

Shear Thinning







Other Factors:

Hematocrit
Osmotic Pressure
Plasma Composition
…………………..

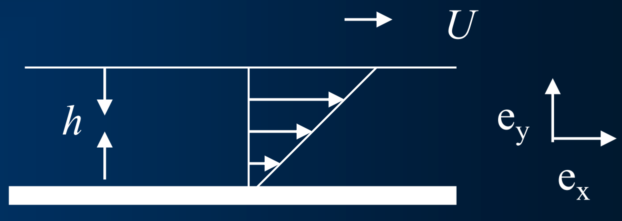
Courtesy of Prof. K.B. Chandran, University of Iowa.

NEWTONIAN vs NON-NEWTONIAN FLUID BEHAVIOR

♦ Non - Constant Viscosity

$$T = -pI + \tau = -pI + 2\mu(\dot{\gamma})D$$

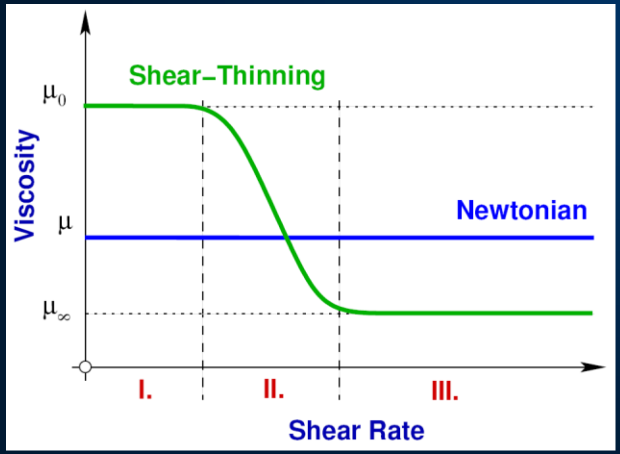
Simple Shear



$\dot{\gamma} = U / h$ → shear rate

$v = \dot{\gamma}\, y\, e_x$ → velocity field

- Shear thinning (or pseudoplastic) fluids
- Shear thickening (or dilatant) fluids
- Yield stress (Bingham plastic) fluids



$$\mu(\dot{\gamma}) = \frac{\tau(\dot{\gamma})}{\dot{\gamma}}, \qquad \mu(\dot{\gamma}) > 0$$

shear-viscosity function
(apparent viscosity)

at constant shear rate:

- Thixotropic fluids (apparent viscosity decreasing in time)
- Rheopectic fluids (apparent viscosity increasing in time)

SHEAR-THINNING BLOOD FLOW MODELS

$$\mu_0 = \lim_{\dot\gamma \to 0} \mu(\dot\gamma) = 0.056 Pas \qquad \mu_\infty = \lim_{\dot\gamma \to \infty} \mu(\dot\gamma) = 0.00345 Pas$$

Model	$\dfrac{\mu(\dot\gamma) - \mu_\infty}{\mu_0 - \mu_\infty}$	Material constants for blood
Powell-Eyring	$\dfrac{\sinh^{-1}(\lambda\dot\gamma)}{\lambda\dot\gamma}$	$\lambda = 5.383s$
Cross	$(1 + (\lambda\dot\gamma)^m)^{-1}$	$\lambda = 1.007s, m = 1.028$
Modified Cross	$(1 + (\lambda\dot\gamma)^m)^{-a}$	$\lambda = 3.736s, m = 2.406, a = 0.254$
Carreau	$(1 + (\lambda\dot\gamma)^2)^{(n-1)/2}$	$\lambda = 3.313s, n = 0.3568$
Carreau-Yasuda	$(1 + (\lambda\dot\gamma)^a)^{(n-1)/a}$	$\lambda = 1.902s, n = 0.22, a = 1.25$

(Y.I.Cho and K.R.Kensey, *Biorheology*, 1991)

BLOOD RHEOLOGY

- Anne M. Robertson, Adélia Sequeira and Marina V. Kameneva. **Hemorheology.** In: *Hemodynamical Flows: Modeling, Analysis and Simulation*, G. P. Galdi, R. Rannacher, A. M. Robertson, S. Turek, Oberwolfach Seminars, Vol. 37, pp.63-120, 2008.

- Anne M. Robertson, Adélia Sequeira and Robert Owens. **Rheological models for blood.** In: *Cardiovascular Mathematics,* A. Quarteroni, L. Formaggia and A. Veneziani (eds.), Springer-Verlag, 2009.

BLOOD FLOW DYNAMICS

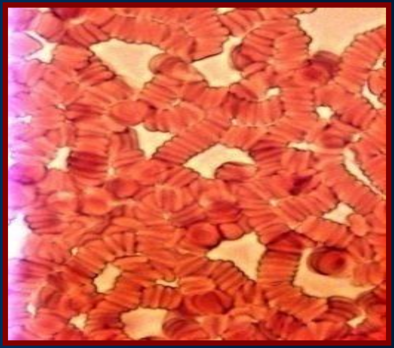
Blood flow: Generalized Newtonian fluid equations

$$\rho\left(\frac{\partial \boldsymbol{u}}{\partial t} + \boldsymbol{u}\cdot\nabla\boldsymbol{u}\right) + \nabla p - \nabla\cdot\boldsymbol{\tau}(\boldsymbol{u}) = 0 \quad \text{in } \Omega$$

$$\nabla\cdot\boldsymbol{u} = 0 \quad \text{in } \Omega$$

$$\boldsymbol{\tau}(\boldsymbol{u}) = 2\mu(\dot{\gamma})\boldsymbol{D}(\boldsymbol{u}) \quad \text{in } \Omega$$

$$\boldsymbol{D}(\boldsymbol{u}) = \frac{1}{2}(\nabla\boldsymbol{u} + (\nabla\boldsymbol{u})^T), \qquad \dot{\gamma} = \sqrt{2\boldsymbol{D}(\boldsymbol{u}) : \boldsymbol{D}(\boldsymbol{u})}$$



Rouleaux aggregation

Shear-thinning viscosity Carreau model

$$\frac{\mu(\dot{\gamma}) - \mu_\infty}{\mu_0 - \mu_\infty} = \left[1 + (\lambda\dot{\gamma})^2\right]^{(n-1)/2}, \qquad n \leq 1$$

$$\mu_0 = \lim_{\dot{\gamma}\to 0}\mu(\dot{\gamma}) = 0.056 Pa\, s$$

$$\mu_\infty = \lim_{\dot{\gamma}\to\infty}\mu(\dot{\gamma}) = 0.00345 Pa\, s$$

$$\lambda = 3.313\, s$$

$$n = 0.3568$$

MATHEMATICAL RESULTS

If the correspondence $\boldsymbol{T} \to \mu(|\boldsymbol{T}|^2)\boldsymbol{T}$ satisfies, for some $p > 1$, and for every symetric second order tensors $\boldsymbol{A}, \boldsymbol{B}$, the relations

$$\mu(|\boldsymbol{A}|^2)\boldsymbol{A} : \boldsymbol{A} \geq C|\boldsymbol{A}|^p$$
$$|\mu(|\boldsymbol{A}|^2)\boldsymbol{A}| \leq C(1 + |\boldsymbol{A}|)^{p-1}$$
$$\big(\mu(|\boldsymbol{A}|^2)\boldsymbol{A} - \mu(|\boldsymbol{B}|^2)\boldsymbol{B}\big) : (\boldsymbol{A} - \boldsymbol{B}) > 0$$

then for $f \in L^{p'}(\Omega)$ and $p > \dfrac{2d}{d+1}$, there exists a weak solution $\boldsymbol{u} \in W^{1,p}(\Omega)$,

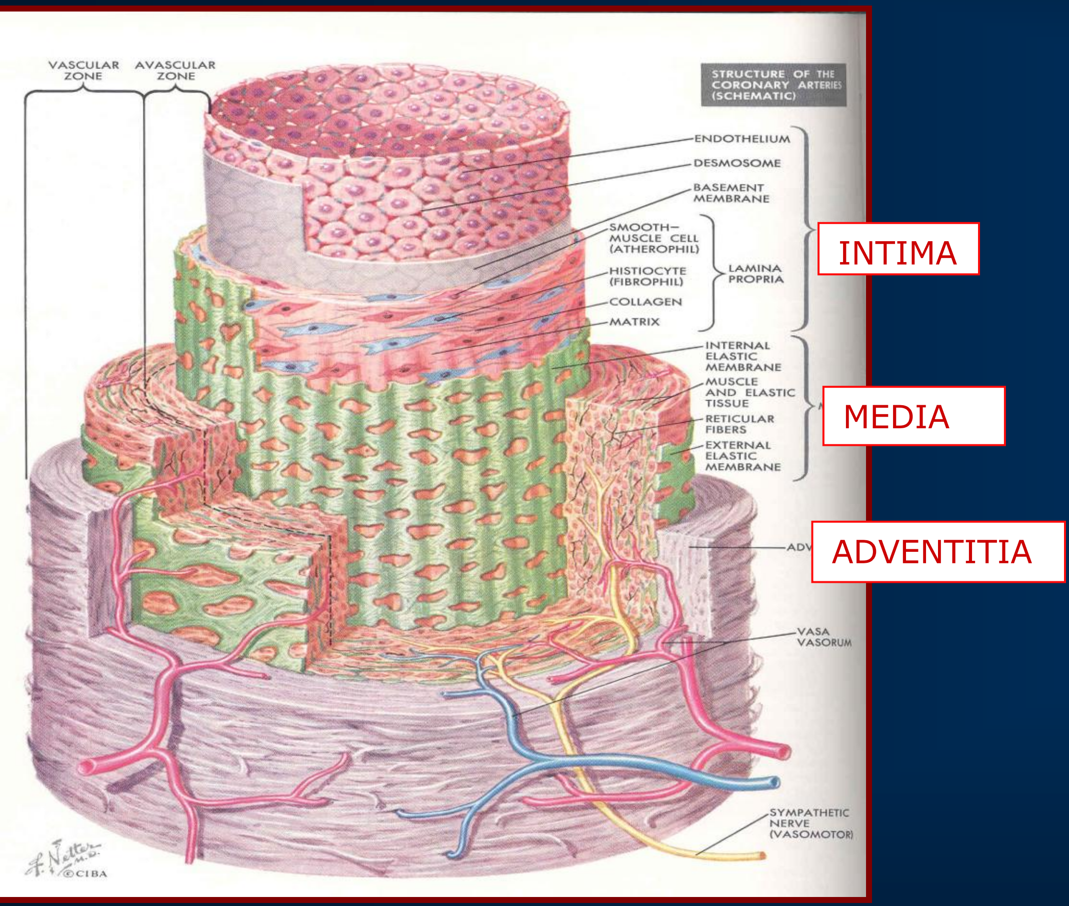
$$\int_\Omega \mu(\dot\gamma)\boldsymbol{D}\boldsymbol{u} : \boldsymbol{D}\boldsymbol{v} + \int_\Omega (\boldsymbol{u} \cdot \nabla \boldsymbol{u}) \cdot \boldsymbol{v} = \int_\Omega \boldsymbol{f} \cdot \boldsymbol{v}, \quad \forall \boldsymbol{v} \in \mathcal{V}.$$

$p > \dfrac{3d+2}{d+2}$ (unsteady, Ladyzhenskaya (1968-70), Lions(1969))

$p > \dfrac{3d}{d+2}$ (unsteady, space periodic, Bellout(1994), Málek(1996))

$p \geq \dfrac{3d}{d+2}$ (steady, Lions(1969)) $p > \dfrac{2d}{d+1}$ (steady, Frehse(1997), Ruzicka(1997), Arada and Sequeira(2005))

MORPHOLOGY OF THE BLOOD VESSELS



The vessel wall is formed by many layers made of tissues with different mechanical characteristics

Mechanical model of the arterial vessel: linear or non-linear elasticity in Lagrangian formulation

↓

Mechanical interaction
(Fluid-wall coupling)

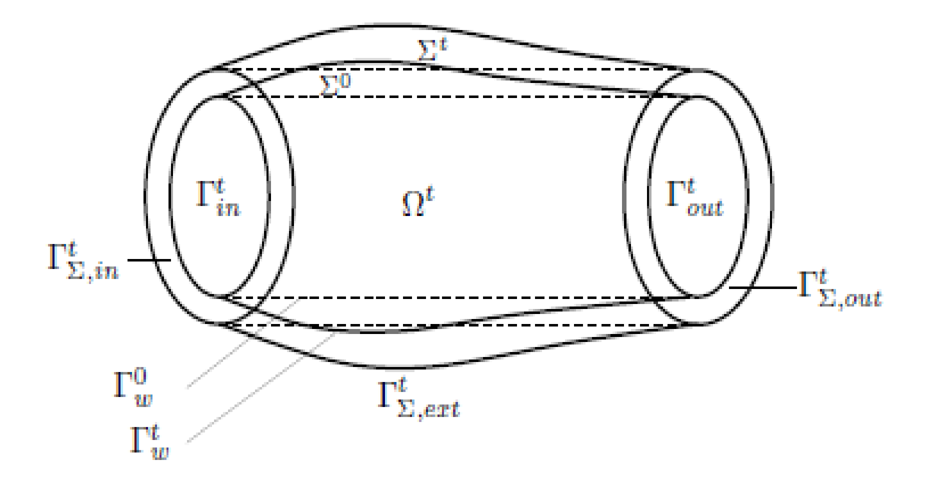
MECHANICAL FLUID-STRUCTURE INTERACTION

Equations for the deformation of the vessel wall

3D nonlinear hyperelasticity (Lagrangian formulation)

$\Sigma^t \subset \mathbb{R}^3$ → structure domain

Σ^0 → reference configuration



$$\partial\Sigma^t = \Gamma^t_\omega \cup \Gamma^t_{\Sigma,ext} \cup \Gamma^t_{\Sigma,in} \cup \Gamma^t_{\Sigma,out}$$

$$\partial\Sigma^0 = \Gamma^0_\omega \cup \Gamma^0_{\Sigma,ext} \cup \Gamma^0_{\Sigma,in} \cup \Gamma^0_{\Sigma,out}$$ → reference boundaries

MECHANICAL FLUID-STRUCTURE INTERACTION

3D nonlinear hyperelasticity (Lagrangian formulation)

$$\rho_w \frac{\partial^2 \eta}{\partial t^2} - \nabla_0 . \sigma(\eta) = 0 \quad \text{in } \Sigma^0, \forall t \in I$$

η → displacement vector

ρ_w → wall density

$$\sigma(\eta) = F(\eta) S(\eta) = (I + \nabla_0 \eta) S(\eta)$$ → first Piola-Kirchhoff tensor

$F(\eta)$: deformation gradient tensor

$S(\eta)$: second Piola-Kirchhoff tensor

3D nonlinear hyperelasticity (Lagrangian formulation)

Green-St Venant strain tensor

$$E = E(\eta) = \frac{1}{2}(F^T F - I) = \frac{1}{2}\left((\nabla_0 \eta)^T + \nabla_0 \eta + (\nabla_0 \eta)^T \nabla_0 \eta\right)$$

St Venant – Kirchhoff material $S(\eta) = \lambda tr(E) I + 2\nu E$ (linear response)

with

$\lambda(\bar{E}, \xi), \nu(\bar{E}, \xi)$ → Lamé constants (functions of Young modulus, Poisson ratio)

MECHANICAL FLUID-STRUCTURE INTERACTION

Equations for the deformation of the vessel wall (Lagrangian formulation)

$$\rho_w \frac{\partial^2 \eta}{\partial t^2} - \nabla_0 . \sigma(\eta) = 0 \quad \text{in } \Sigma^0$$

initial

&

boundary conditions

$$\eta = \eta_0 \quad \text{for } t = 0, \quad \text{in } \Sigma^0$$

$$\frac{\partial \eta}{\partial t} = \frac{\partial \eta_0}{\partial t} \quad \text{for } t = 0, \quad \text{in } \Sigma^0$$

$$\sigma(\eta).n_0 = \hat{\phi} \quad \text{on } \Gamma^0_\omega$$

$$\sigma(\eta).n_0 = 0 \quad \text{on } \Gamma^0_{\Sigma,ext}$$

$$\eta = 0 \quad \text{on } \Gamma^0_{\Sigma,out}$$

$$\eta = 0 \quad \text{on } \Gamma^0_{\Sigma,in}$$

\+

compatibility conditions

$$\frac{\partial \eta_0}{\partial t} = u_0 \quad \text{on } \Gamma^0_\omega$$

&

interface conditions

(clamped structure)

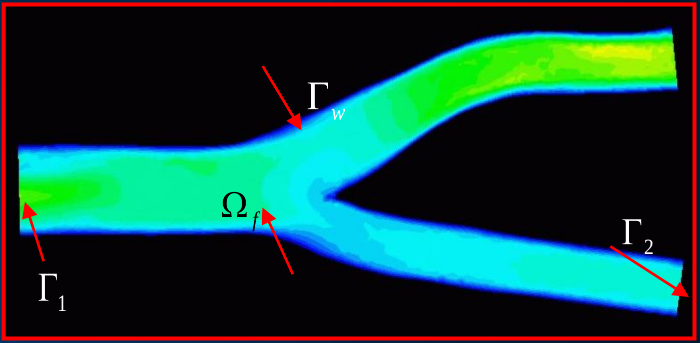
MECHANICAL FLUID-STRUCTURE INTERACTION

- Blood flow: Generalized Newtonian flow (ALE frame)

$$\rho\left(\frac{\partial \boldsymbol{u}}{\partial t} + (\boldsymbol{u}-\boldsymbol{w})\cdot\nabla\boldsymbol{u}\right) + \nabla p - \nabla\cdot\boldsymbol{\tau}(\boldsymbol{u}) = 0 \quad \text{in } \Omega_f$$

$$\nabla\cdot\boldsymbol{u} = 0 \quad \text{in } \Omega_f$$

$$\boldsymbol{\tau}(\boldsymbol{u}) = 2\mu(\dot\gamma)\boldsymbol{D}(\boldsymbol{u}) \quad \text{in } \Omega_f$$



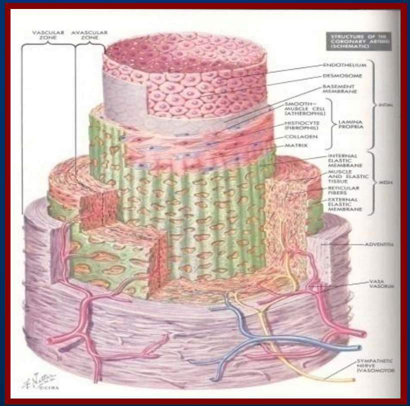
- Deformation of the vessel wall

$$\rho_w \frac{\partial^2\eta}{\partial t^2} - \nabla_0.\sigma(\eta) = 0 \quad \text{in } \Sigma^0$$

- Interface conditions

$$\boldsymbol{\sigma}(\eta)\cdot\boldsymbol{n} = -p\boldsymbol{n} + \boldsymbol{\tau}(\boldsymbol{u})\cdot\boldsymbol{n} \quad \text{at } \Gamma_w$$

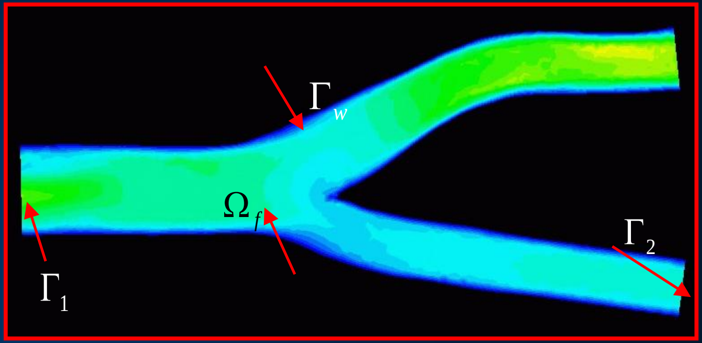
$$\boldsymbol{u} = \frac{\partial\eta}{\partial t} \quad \text{at } \Gamma_w$$



u = blood velocity
w = domain velocity
p = pressure
ρ_f = density
μ = viscosity
η = wall displacement

+ initial and boundary conditions at Γ_i (i=0,1,2)

MECHANICAL FLUID-STRUCTURE INTERACTION



- Interface conditions

$$u = \frac{\partial \eta}{\partial t}, \quad \forall t \in I, \ \text{at } \Gamma_\omega^t$$

$$\sigma(\eta) \cdot n = -pn + \tau(u) \cdot n, \quad \forall t \in I, \ \text{at } \Gamma_\omega^t$$

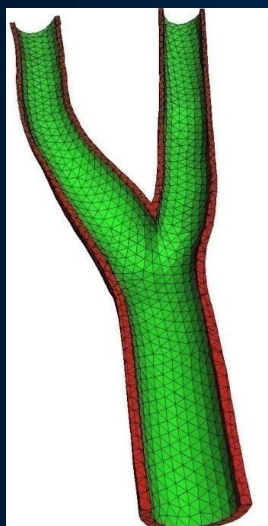
(using the Piola transform)

$$-(\det \nabla_0 \eta)\tau(u, p)(\nabla_0^{-T} \eta) \cdot n_0 = \sigma(\eta) \cdot n_0, \quad \forall t \in I, \ \text{on } \Gamma_\omega^t$$

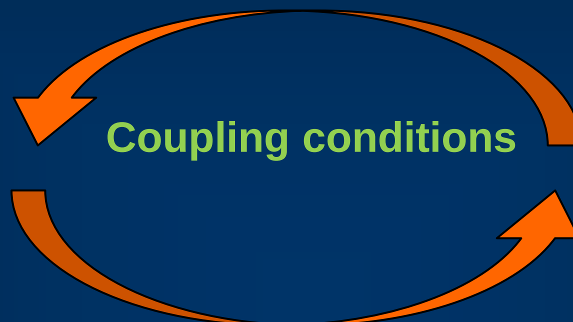
MECHANICAL FLUID-STRUCTURE INTERACTION

Blood:
Newtonian or non-Newtonian fluid

Deformation of the Vessel Wall:
3D (nonlinear) elasticity or 2D shell type models

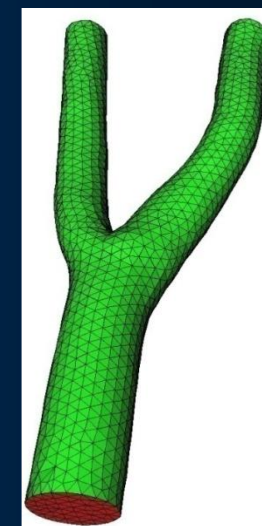


Displacement (new domain)



Normal stress

implicit coupling
(iterative procedure)



Open problems:

Well posedness of the FSI problem

Contributions given by e.g. : D.Coutand, S. Shkoller, Y.Maday, C.Grandmont, B.Desjardins, M.Esteban, G.P. Galdi, H.Beirão da Veiga, among others

Devise efficient numerical algorithms

Contributions given by e.g. : P. le Tallec, F.Nobile, M.A.Fernandéz,M.Moubachir,J-F.Gerbeau, S.Deparis, W.A.Wall, among others

MECHANICAL FLUID-STRUCTURE INTERACTION

Regularity Assumptions:

$\Omega^t \subset \mathbb{R}^3$ is open and connected

$\partial\Omega^t = \Gamma^t_\omega \cup \Gamma^t_{in} \cup \Gamma^t_{out}$ Is locally Lipschitz $(\partial\Omega^t \in C^{1,1})$

$$\frac{\partial \eta}{\partial t} \in H^{1/2}(\Gamma^t_\omega) \Longrightarrow u(t) \in H^1(\Omega^t), \forall t$$

MECHANICAL FLUID-STRUCTURE INTERACTION

An Energy Estimate for the coupled FSI problem

[J. Janela, A. Moura, A. S, 2009 – generalization of L. Formaggia, A. Moura, F. Nobile, 2007]

$$\mathrm{E}(t) = \frac{\rho}{2}\|u\|^2_{L^2(\Omega^t)} + \frac{\rho_w}{2}\left\|\frac{\partial \eta}{\partial t}\right\|^2_{L^2(\Sigma^0)} + \mu(\dot{\gamma})\|E(\eta)\|^2_{L^2(\Sigma^0)} + \frac{\lambda}{2}\|trE(\eta)\|^2_{L^2(\Sigma^0)}$$

THEOREM: The coupled FSI problem, with homogeneous Dirichlet boundary conditions $u = 0$ at Γ^t_{in} and Γ^t_{out} satisfies the following energy inequality

$$\frac{d}{dt}(\mathrm{E}(t)) + 2\mu_\infty \|D(u)\|^2_{L^2(\Omega^t)} \leq 0$$

and, consequently, the energy decay property

$$\mathrm{E}(t) + 2\mu_\infty \int_0^t \|D(u)\|^2_{L^2(\Omega^t)}\, dt \leq \mathrm{E}(0)$$

where E(0) is a constant depending only on the initial data $u_0, \eta_0, \dot{\eta}_0$

REMARK: $\int_{\Gamma^t_{in}} |u|^2\, u.n > 0,\ \int_{\Gamma^t_{out}} |u|^2\, u.n > 0$ for homogeneous Neumann conditions

MECHANICAL FLUID-STRUCTURE INTERACTION

Sketch of the PROOF:

1. Multiply the structure equation by $\frac{\partial \eta}{\partial t}$, integrate over the reference domain, use the boundary and matching conditions

2. Multiply the fluid equation by u , integrate over the fluid domain, ...

3. $\mu_\infty \le \mu(\dot{\gamma}) \le \mu_0$ (shear-thinning viscosity fluid)

$$\Rightarrow \int_{\Omega^t} 2\mu(\dot{\gamma}) D(u) : \nabla u \, d\omega \ge 2\mu_\infty \|D(u)\|^2_{L^2(\Omega^t)}$$

Finally $$\frac{d}{dt}(\mathrm{E}(t)) + 2\mu_\infty \|D(u)\|^2_{L^2(\Omega^t)} + \frac{\rho}{2} \int_{\Gamma^t_{in} \cup \Gamma^t_{out}} |u|^2 \, u.n d\gamma \le \int_{\Gamma^t_{in} \cup \Gamma^t_{out}} (\tau(u).n).u d\gamma$$

MECHANICAL FLUID-STRUCTURE INTERACTION

MECHANICAL FLUID-STRUCTURE INTERACTION

FSI Algorithm: (adapted from Fernandéz & Moubachir, 2005)

ALE formulation to account for the evolution of the computational domain

Efficient solvers for each fluid and structure subproblems to ensure accurate and fast convergence of the FSI nonlinear coupled system

Fluid equations:
Discretization in time: **implicit Euler scheme**
Discretization in space: **stabilized P1 bubble / P1 FE**

Structure equations:
Discretization in time: **mid-point Newmark method**
Discretization in space: **P1 FE**

Coupling strategy: fully implicit coupling based on a Newton algorithm with the exact computation of the Jacobian

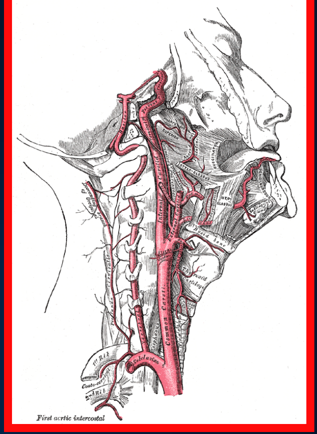
BLOOD FLOW SIMULATIONS

Newtonian vs non-Newtonian behavior

Main objectives:

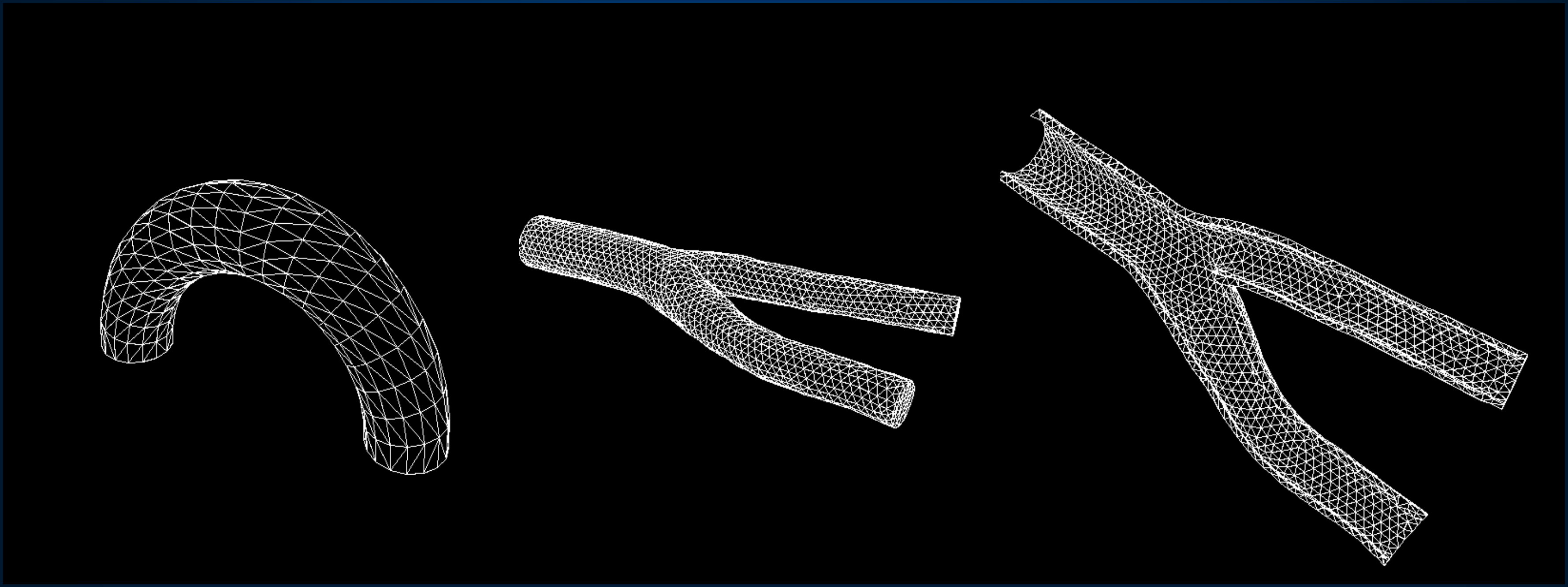
- 3D Non-Newtonian models for blood flow
- 3D Fluid-Structure Interaction algorithms for pressure wave propagation in arteries and detailed flow patterns using Newtonian and non-Newtonian blood flow models
- Geometrical multiscale simulation of the cardiovascular system using non-Newtonian models

BLOOD FLOW SIMULATIONS



carotid
bifurcation

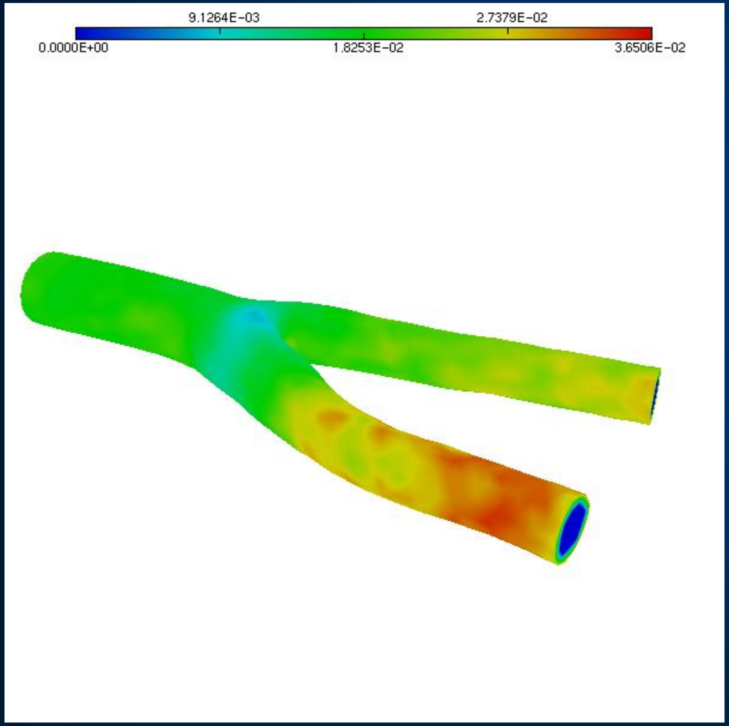
Idealized vs reconstructed geometries
&
computational grids



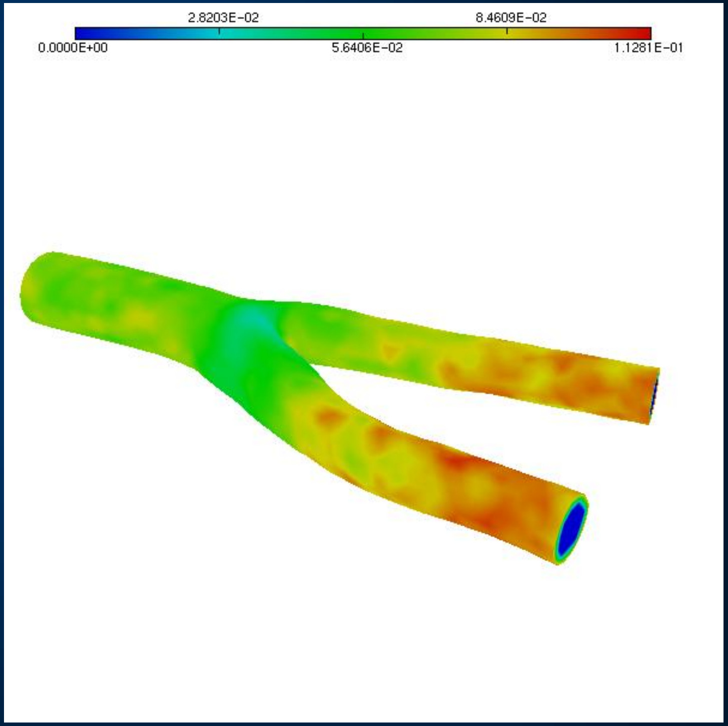
BLOOD FLOW SIMULATIONS

Newtonian vs non-Newtonian

Carotid Bifurcation: Wall Shear Stress (WSS)



Carreau model



Newtonian model

$\mu_\infty = 0.0035 Pas$

A. Moura & J. Janela

BLOOD FLOW SIMULATIONS

Carreau model

Curved vessel: Pressure

Fluid:

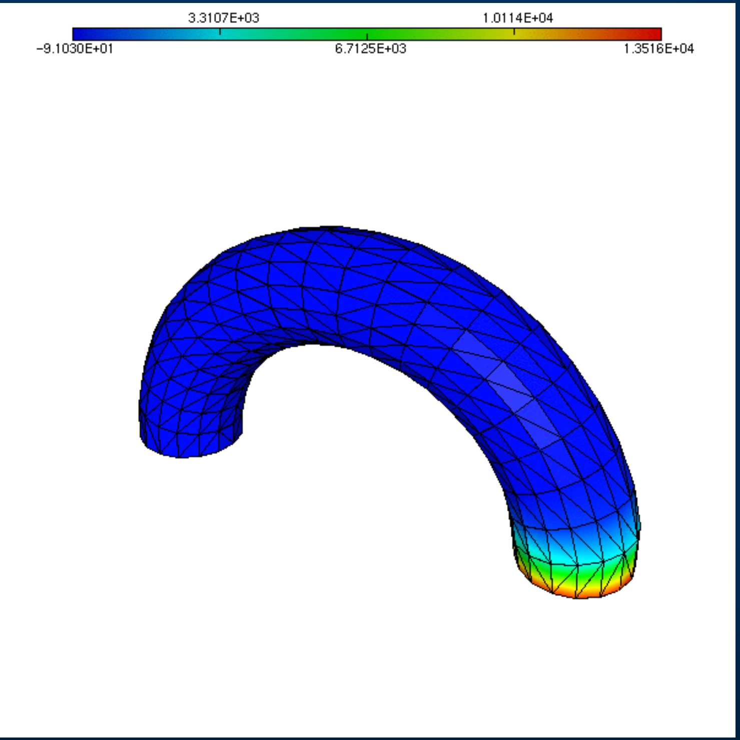
$$\mu_0 = \lim_{\dot{\gamma}\to 0} \mu(\dot{\gamma}) = 0.056 Pas$$

$$\mu_\infty = \lim_{\dot{\gamma}\to \infty} \mu(\dot{\gamma}) = 0.00345\, Pas$$

$$\lambda = 3.313\, s$$

$$n = 0.3568$$

$$\rho = 1g / cm^3$$



Structure:

$$\lambda(E,\xi) = 3x10^6\, dynes / cm^2$$

$$\nu(E,\xi) = 0.3$$

$$\rho_\omega = 1.2g / cm^2$$

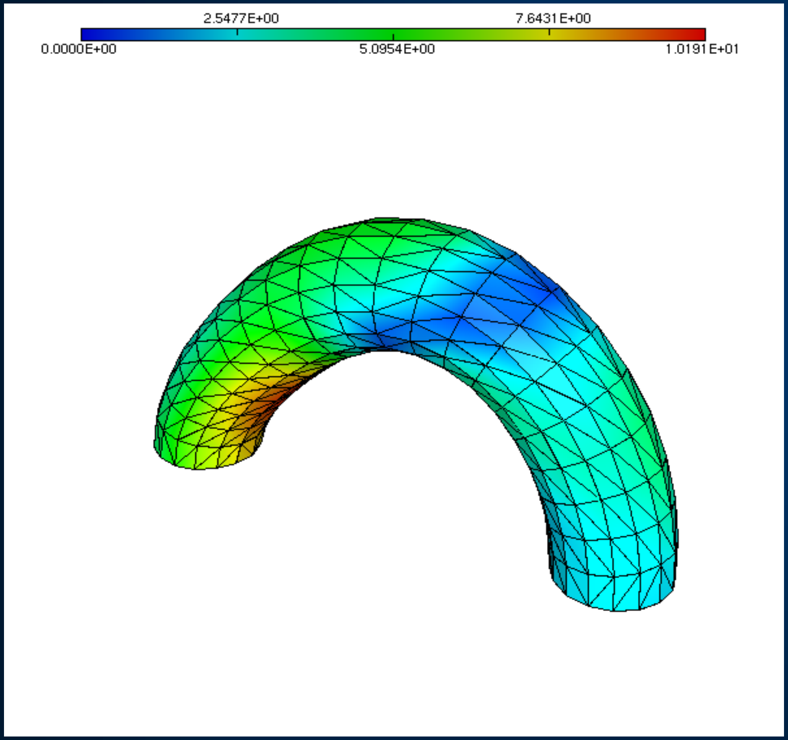
$$h = 0.1cm$$

A. Moura & J. Janela

BLOOD FLOW SIMULATIONS

Newtonian vs non-Newtonian

Curved vessel: Wall Shear Stress (WSS)



Carreau model

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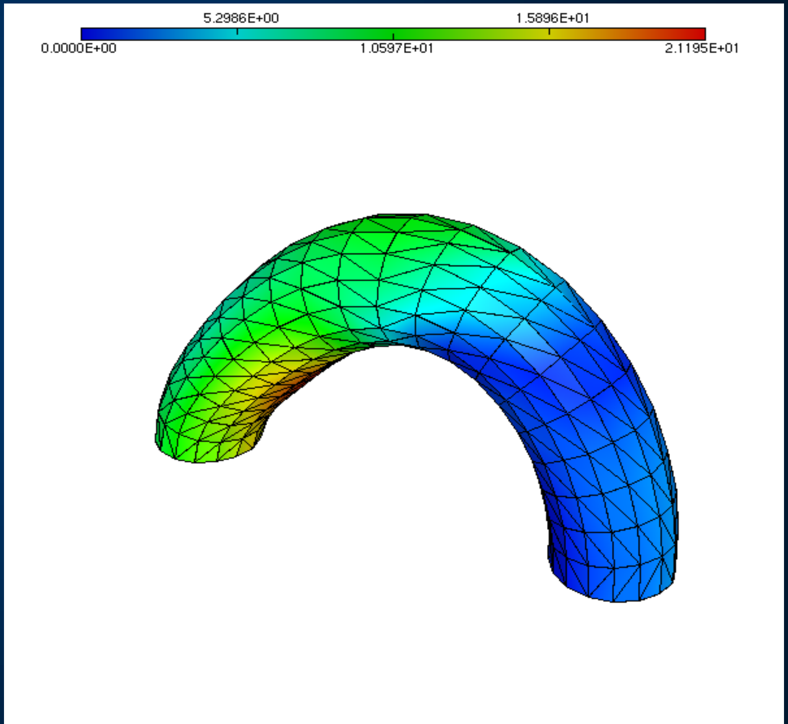
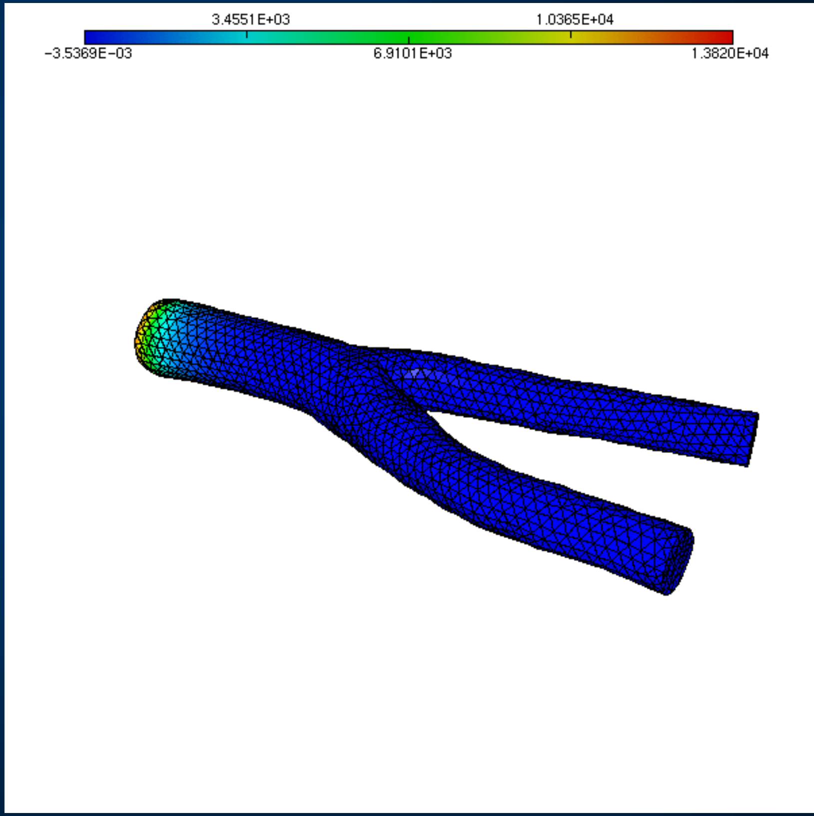


Newtonian model

$\mu_{\infty} = 0.0035 Pas$

BLOOD FLOW SIMULATIONS

Carotid Bifurcation: Pressure pulse



Carreau model

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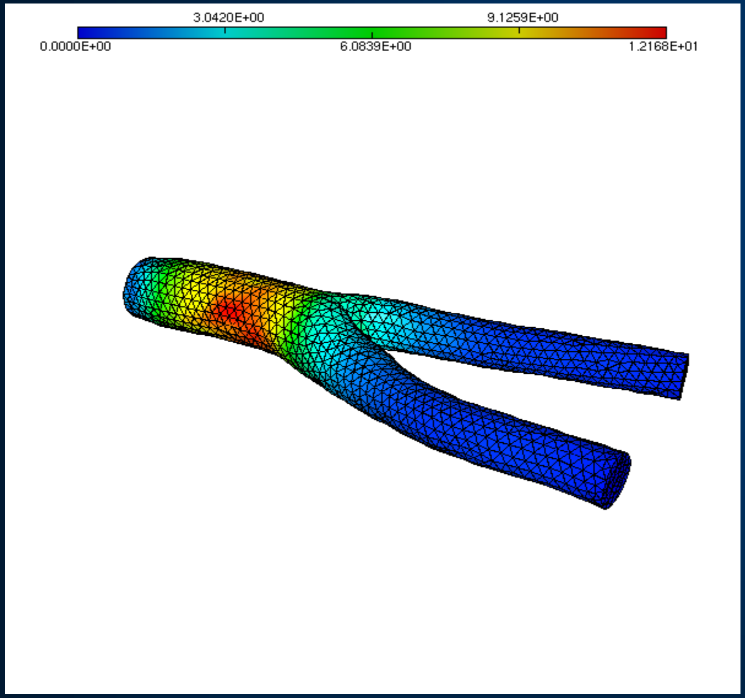
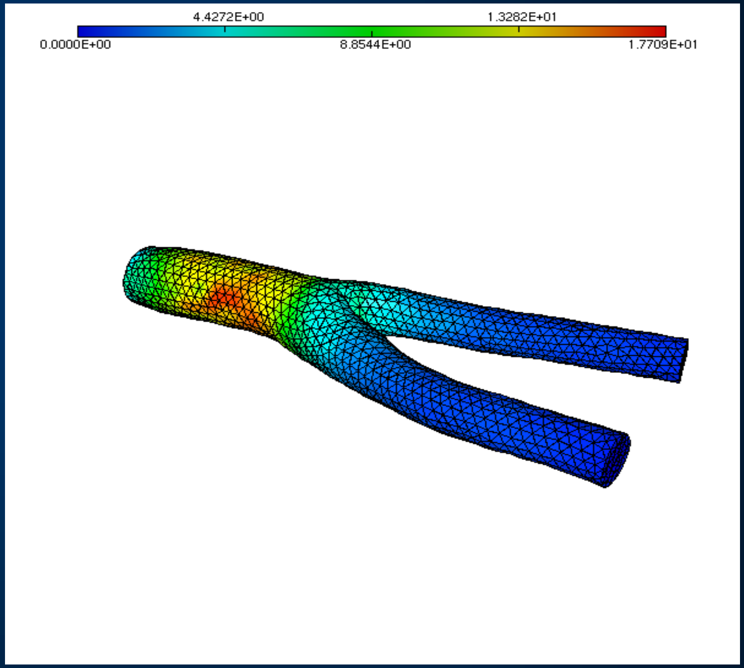
BLOOD FLOW SIMULATIONS

Newtonian vs non-Newtonian

Carotid Bifurcation: Wall Shear Stress (WSS)



Carreau model



Newtonian model

$\mu_\infty = 0.0035 Pas$

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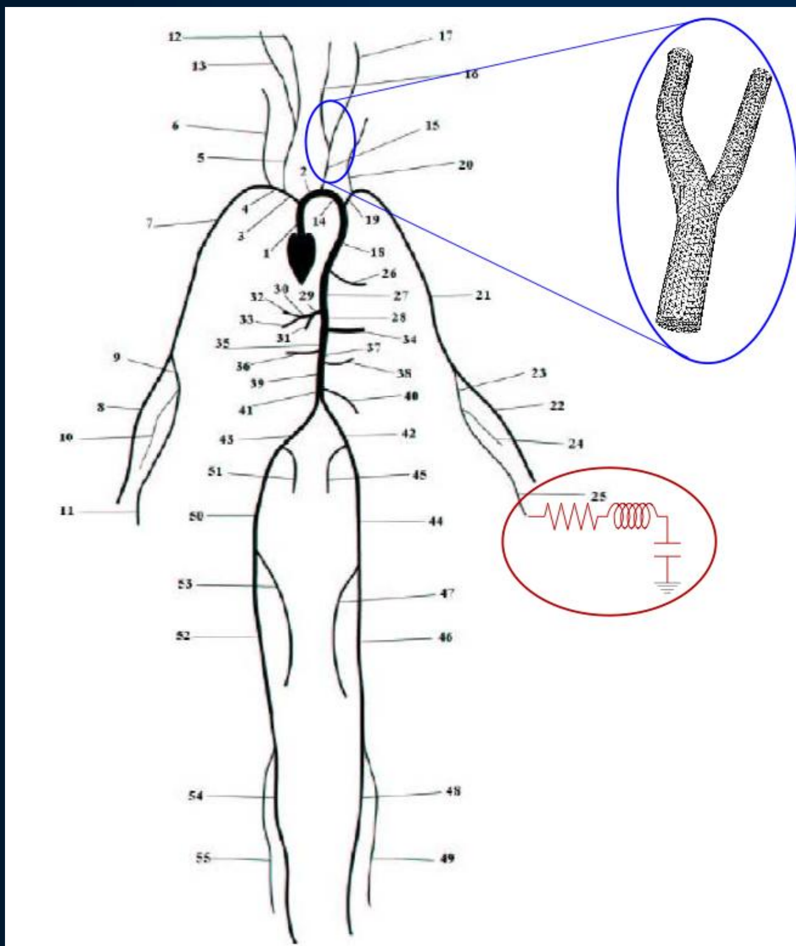
GEOMETRICAL MULTISCALE

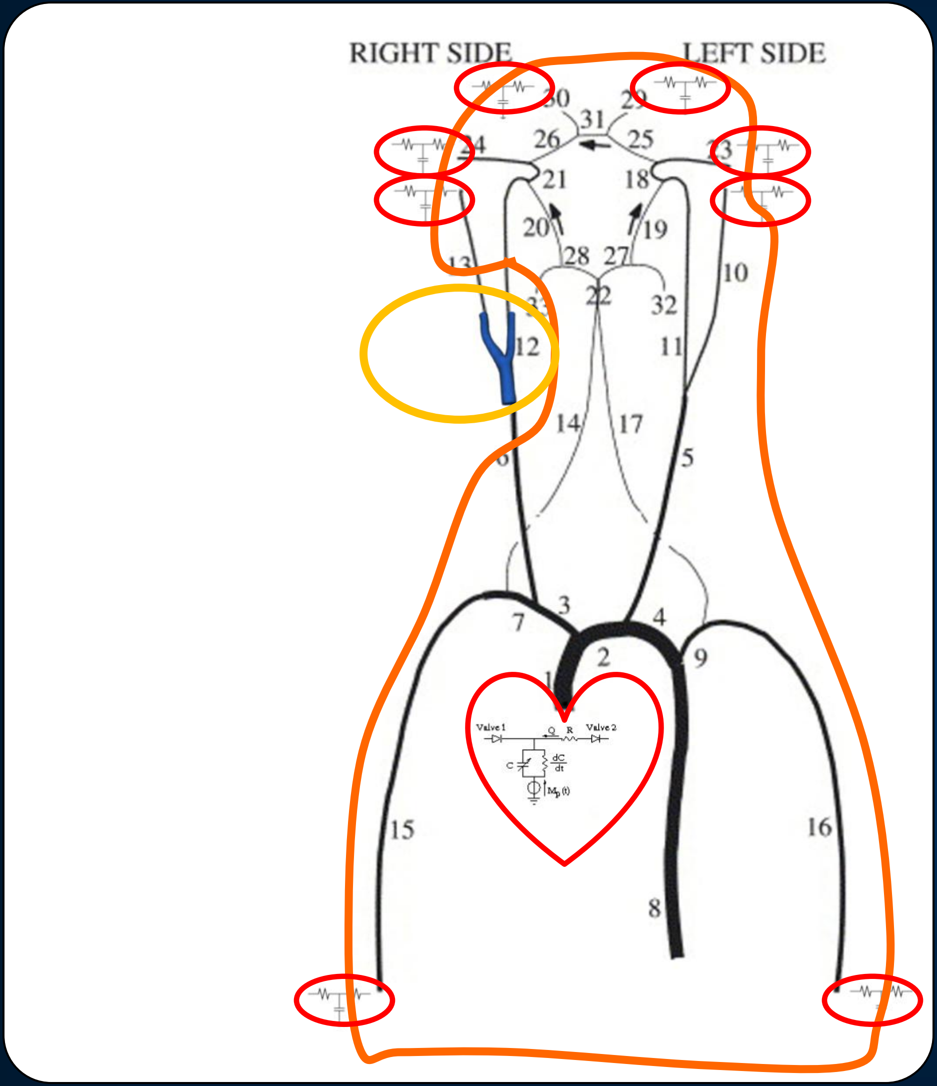


- Global features have influence on the local fluid dynamics
- Local changes in geometry or material properties (e.g. due to surgery, aging, stenosis, …) may induce pressure waves reflections ➔ global effects

Modeling strategy

- use the expensive 3D model only in the region of interest
- couple with network models that include peripheral impedances to account for global effects

GEOMETRICAL MULTISCALE



Allows to take into account the global circulation in localized simulations and set proper boundary conditions

3D

- Very detailed simulations
- Very complex
- Computationally very costly

1D

- Evolution of mean pressure and flux in arteries
- System of hyperbolic equations
- Low computational cost

0D

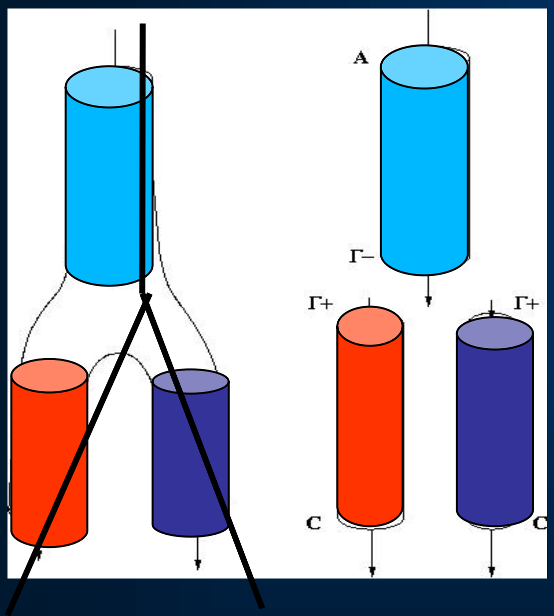
- Evolution in time of mean pressure and flux in wide compartments
- System of ODEs
- Very low computational cost

GEOMETRICAL MULTISCALE

1D Model

Allows for the simulation of complex arterial networks!

Domain decomposition



$$
\begin{cases}
\dfrac{\partial A}{\partial t} + \dfrac{\partial Q}{\partial z} = 0 \\
\dfrac{\partial Q}{\partial t} + \alpha \dfrac{\partial}{\partial z}\left(\dfrac{Q^2}{A}\right) + \dfrac{A}{\rho}\dfrac{\partial P}{\partial z} + K\dfrac{Q}{A} = 0 \\
P - P_0 = \Psi(A)
\end{cases}
$$

Area → $A(z,t) = \int_{\Omega \cap \Sigma(z)} d\gamma$

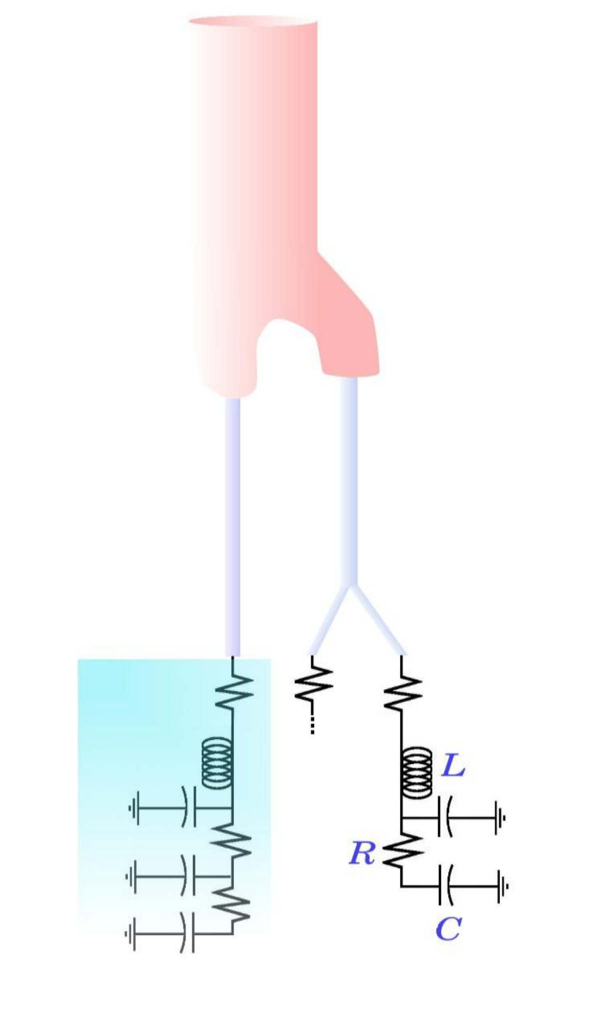
Flux → $Q(z,t) = \int_{\Omega \cap \Sigma(z)} u_z(x,t)\, d\gamma$

Mean Pressure → $P(z,t) = \dfrac{1}{|\Sigma(z)|} \int_{\Omega \cap \Sigma(z)} p(x,t)\, d\gamma$

- ►describes de wave propagation nature of blood flow
- ► acts as absorbing boundary condition for the 3D model
- ► simulation of complex arterial trees by coupling 1D models

GEOMETRICAL MULTISCALE

0D Model



0D Lumped parameters (system of linear ODE's)

$$C\frac{dP_i}{dt} = -(Q_{i+1} - Q_i),$$

$$L\frac{dQ_i}{dt} = -(P_i - P_{i-1}) - RQ_i$$

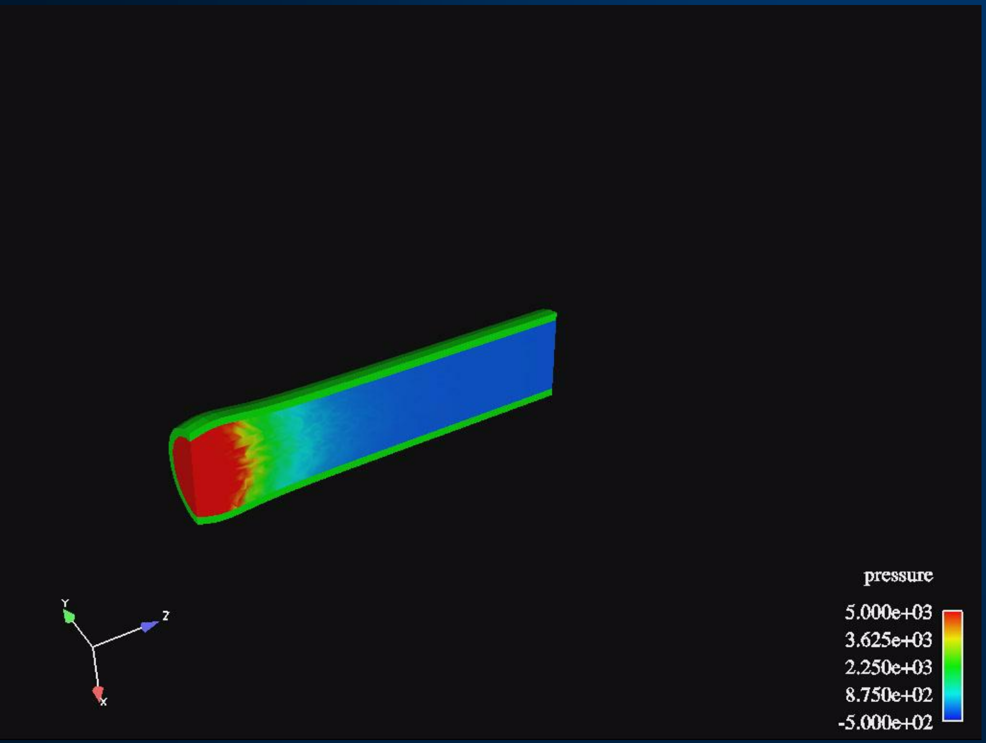
The analogy

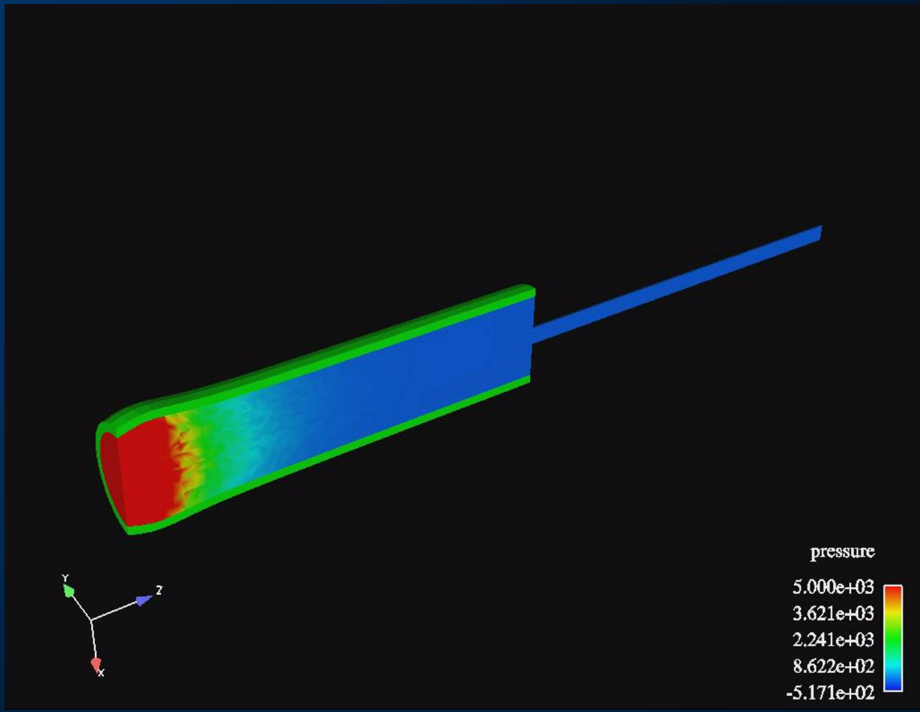
Fluid dynamics	Electrical circuits
Pressure	Voltage
Flow rate	Current
Blood viscosity	Resistance R
Blood inertia	Inductance L
Wall compliance	Capacitance C

- RLC circuits model "large" arteries
- RC circuits account for capillary bed
- Can describe compartments (such as peripheral circulation)

GEOMETRICAL MULTISCALE

3D and 1D for a cylindrical artery: pressure pulse





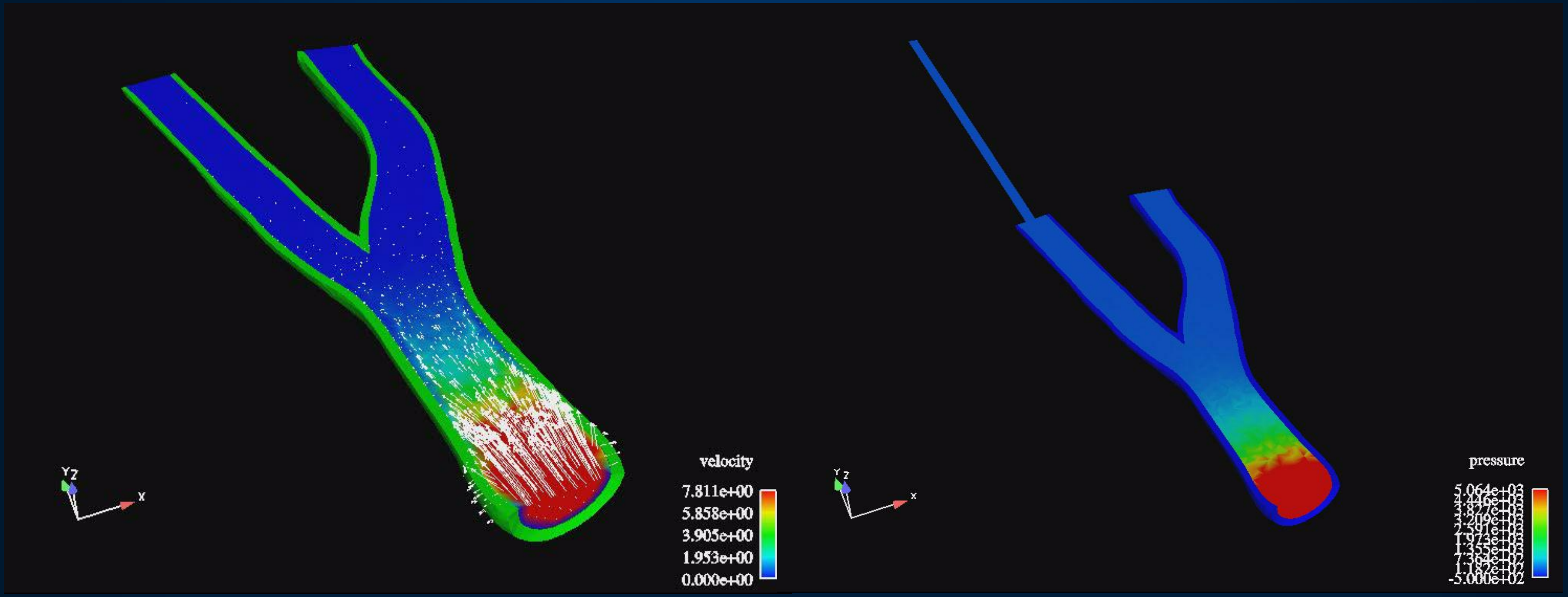
(A. Moura)

3D model (spurious reflections)

3D-1D coupled model

GEOMETRICAL MULTISCALE

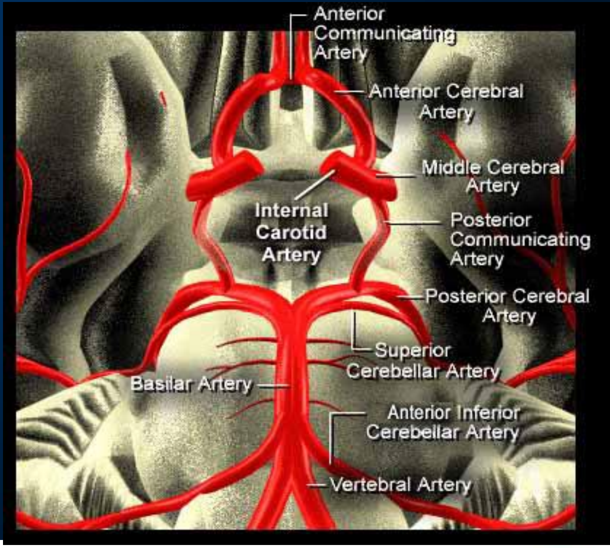
3D-1D for the carotid bifurcation: velocity field & pressure pulse



(A. Moura)

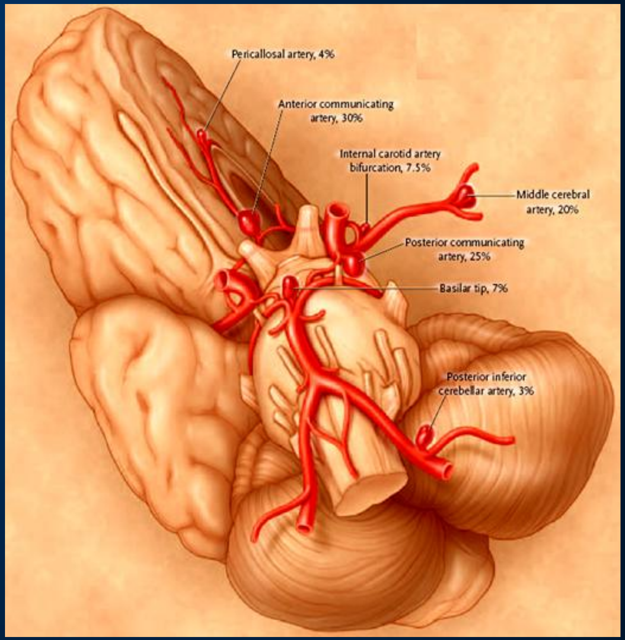
MODELING CEREBRAL ANEURYSMS

- **Cerebral Aneurysms:**
  - Most common cause of hemorrhagic strokes
  - Tend to be silent until rupture
  - High prevalence, low risk
- **Main Goal:**
  - Help improve the evaluation & treatment of cerebral aneurysms
- **Our Approach:**
  - Patient-specific image-based CFD modeling to link hemodynamics & clinical observations



MECHANISMS

- The mechanisms responsible for the development, growth and rupture of intracranial aneurysms are not well understood



- Better understanding of these processes can lead to better patient evaluation and improved treatments

IMAGE-BASED MODELING OF BLOOD FLOWS

blood vessel imaging

$$\rho\left(\frac{\partial \boldsymbol{u}}{\partial t}+(\boldsymbol{u}-\boldsymbol{w})\cdot\nabla\boldsymbol{u}\right)+\nabla p-\nabla\cdot\boldsymbol{\tau}(\boldsymbol{u})=0 \quad \text{in } \Omega_f$$

$$\nabla\cdot\boldsymbol{u}=0 \quad \text{in } \Omega_f$$

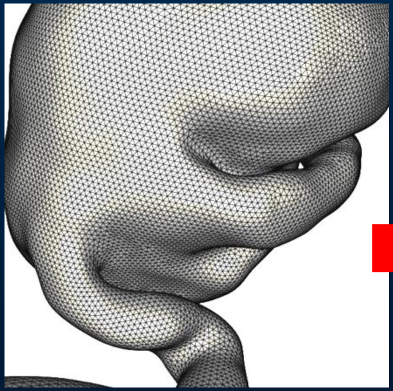
$$\boldsymbol{\tau}(\boldsymbol{u})=2\mu(\dot{\gamma})\boldsymbol{D}(\boldsymbol{u}) \quad \text{in } \Omega_f$$

$$\rho_w\frac{\partial^2\eta}{\partial t^2}-\nabla_0.\sigma(\eta)=0 \quad \text{in } \Sigma^0$$

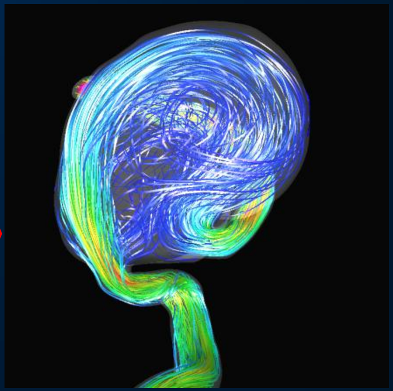
image processing

geometry modeling

meshing

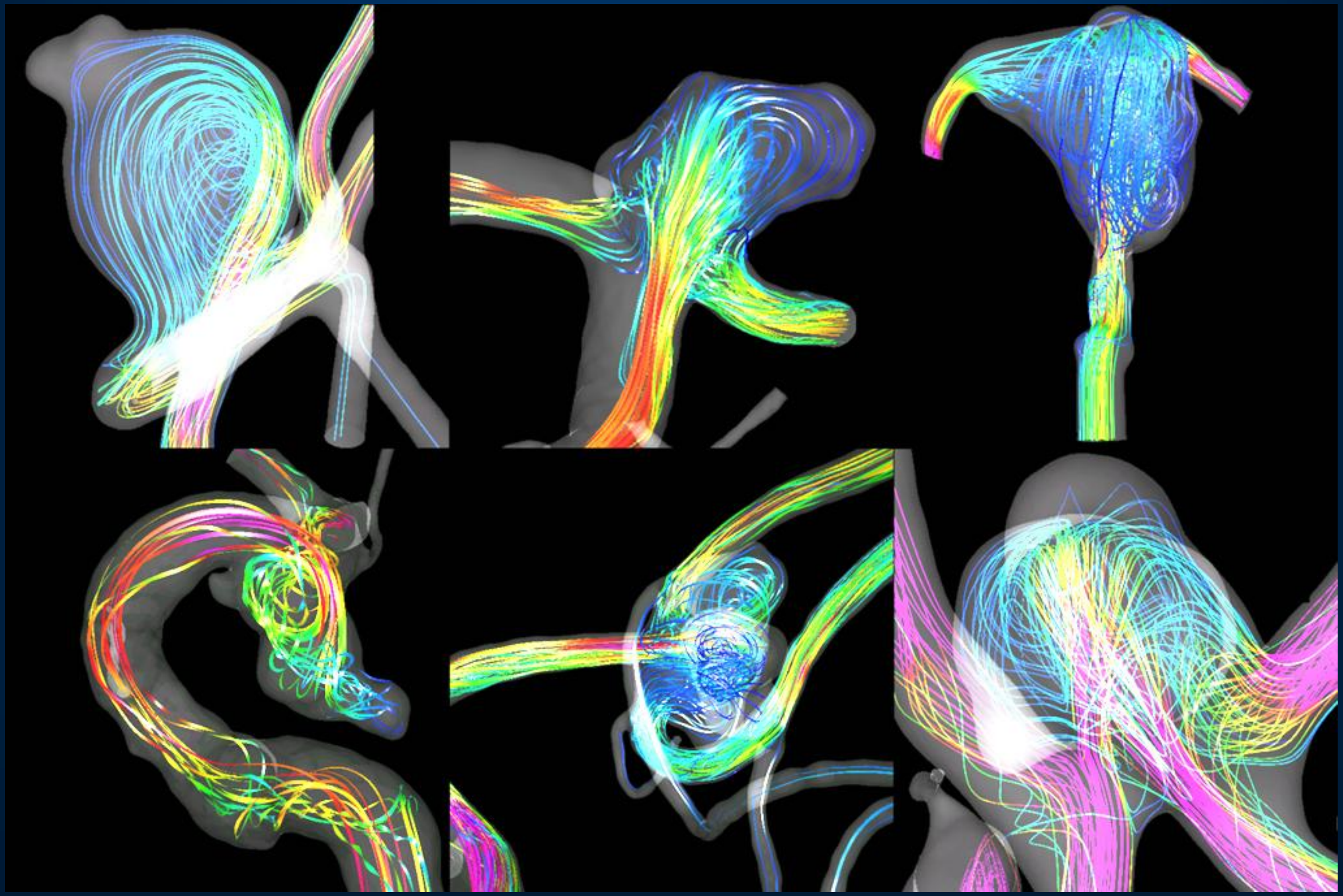


flow solution & visualization



FLOW COMPLEXITY & STABILITY

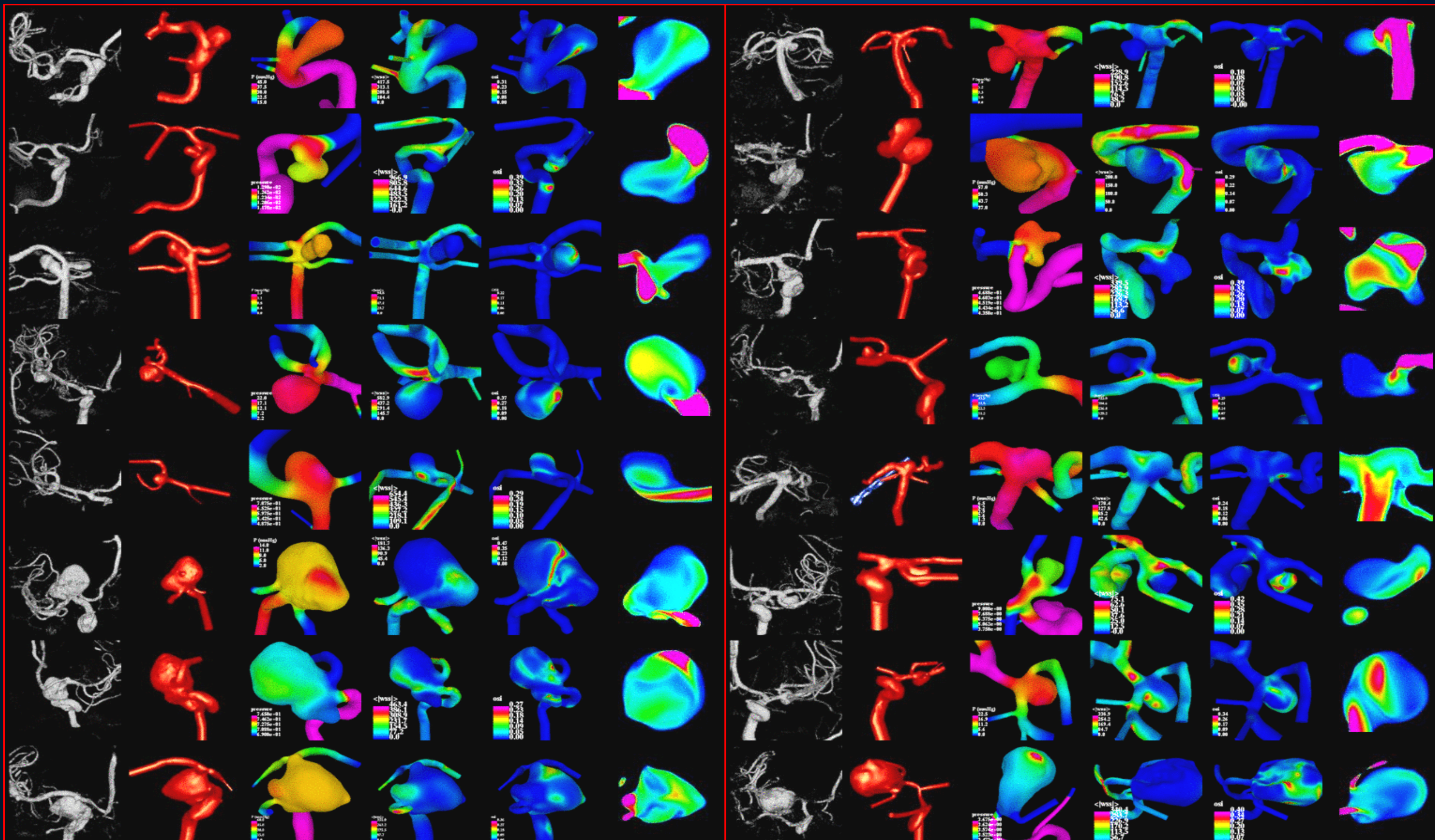
simple



J. Cebral, George Mason Univ

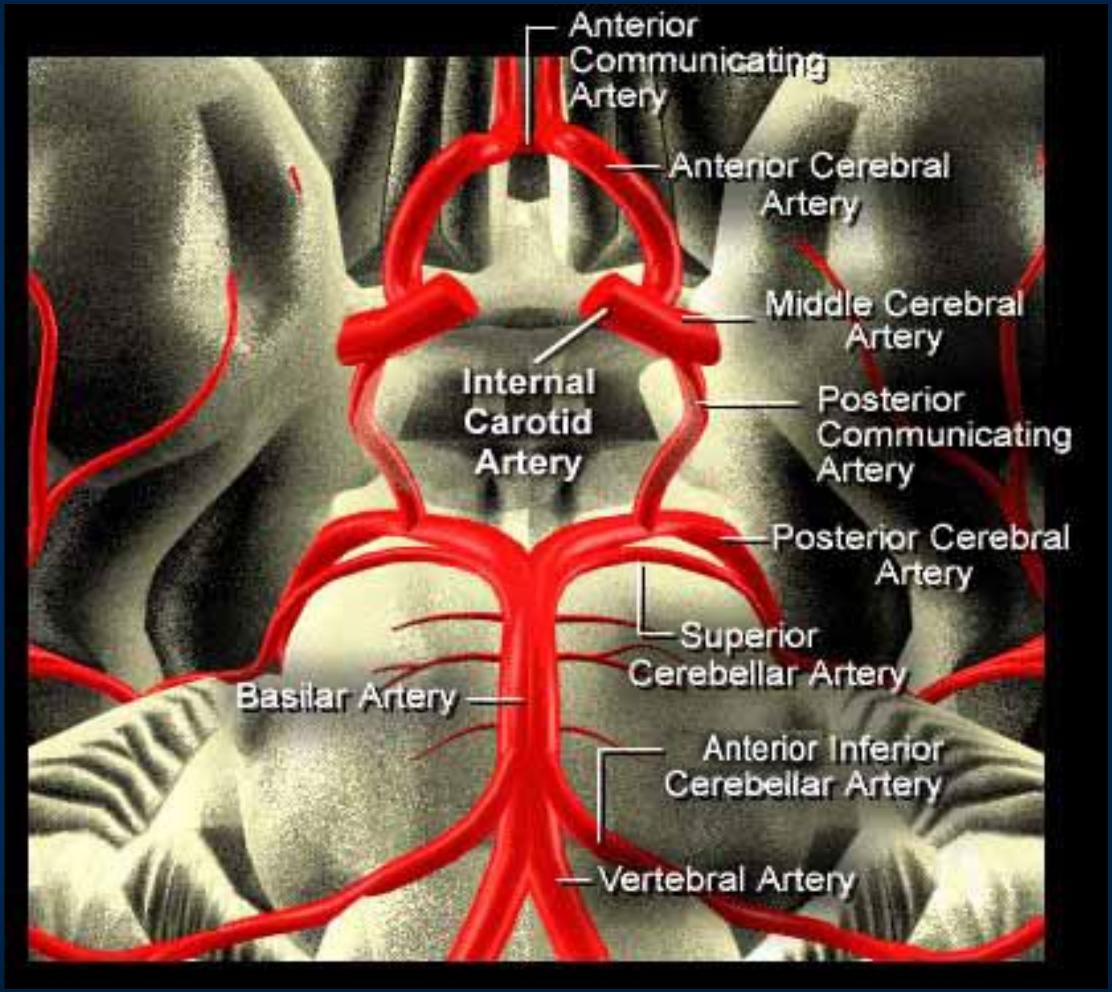
complex

DATABASE: ANEURYSM MODELS & CLINICAL INFO

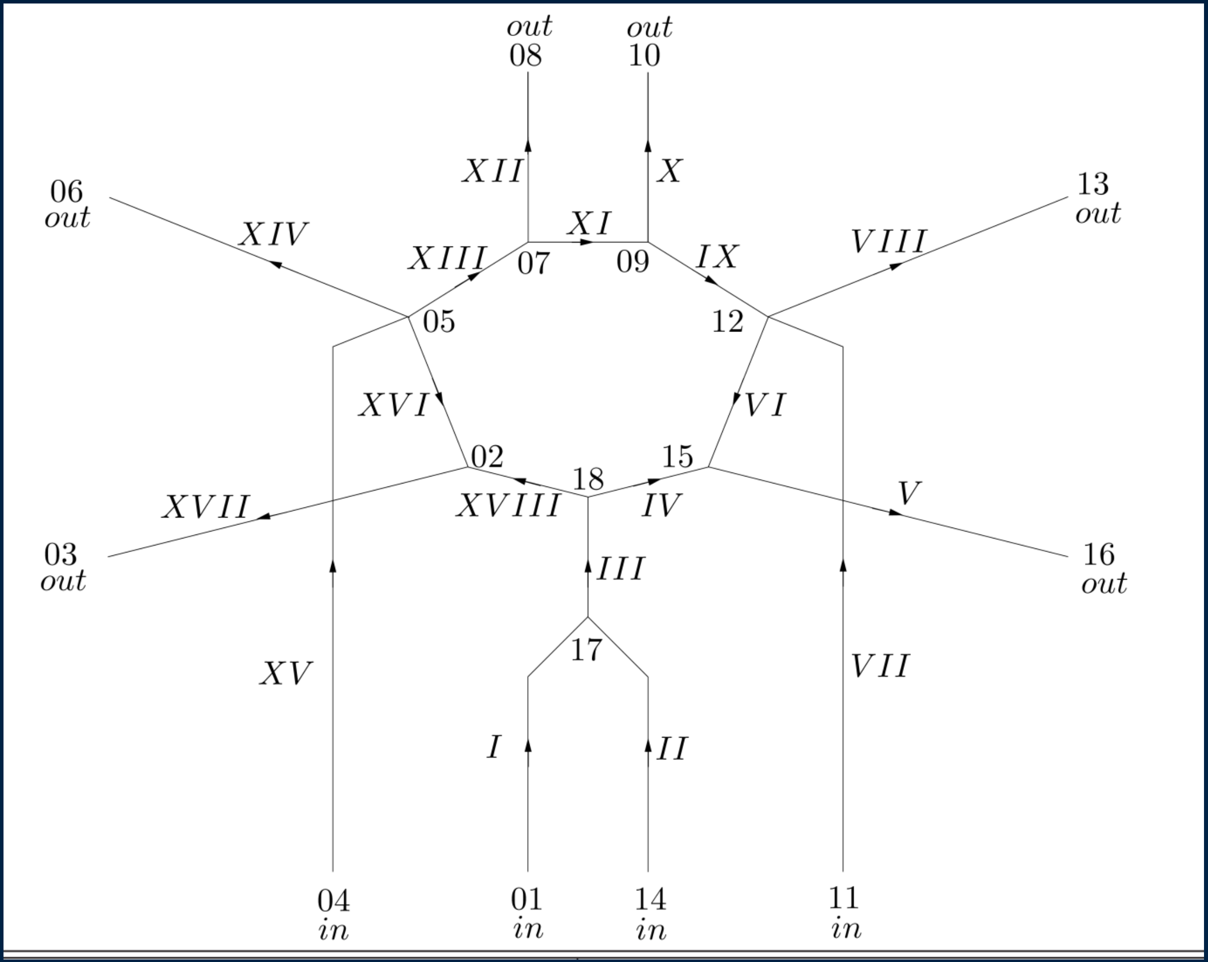


J. Cebral, George Mason Univ

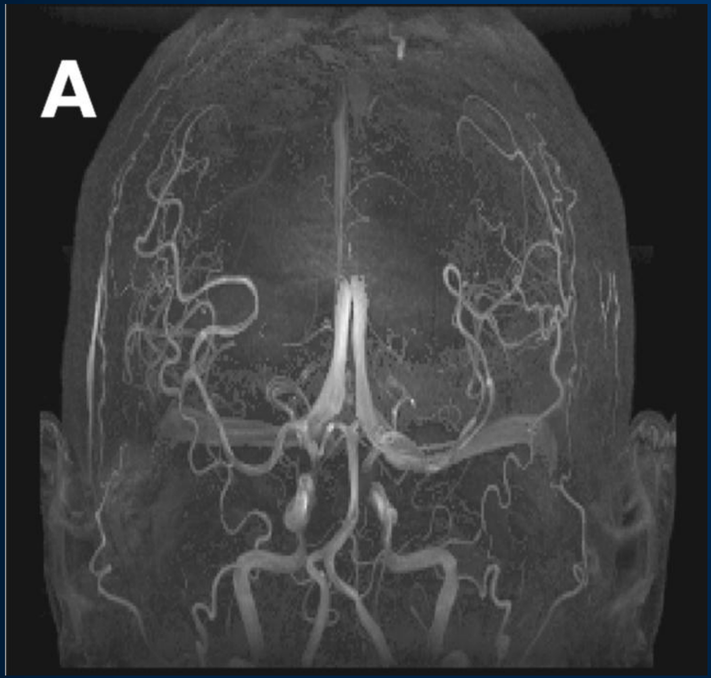
CIRCLE of WILLIS: 1D – NETWORK ?

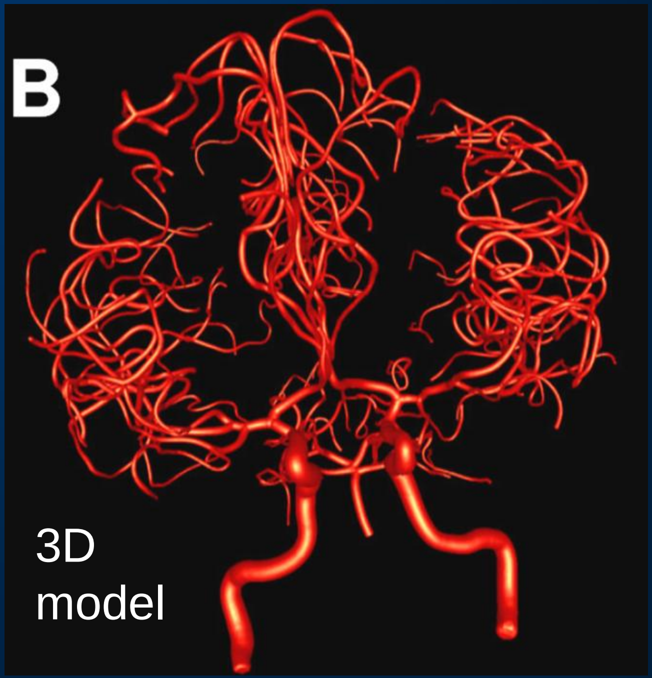


CIRCLE of WILLIS: 1D - NETWORK

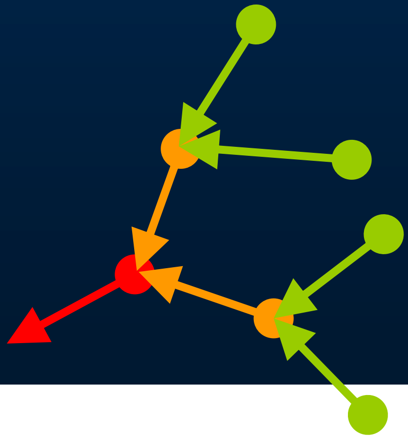


MRA-BASED SUBJECT- SPECIFIC MODELING



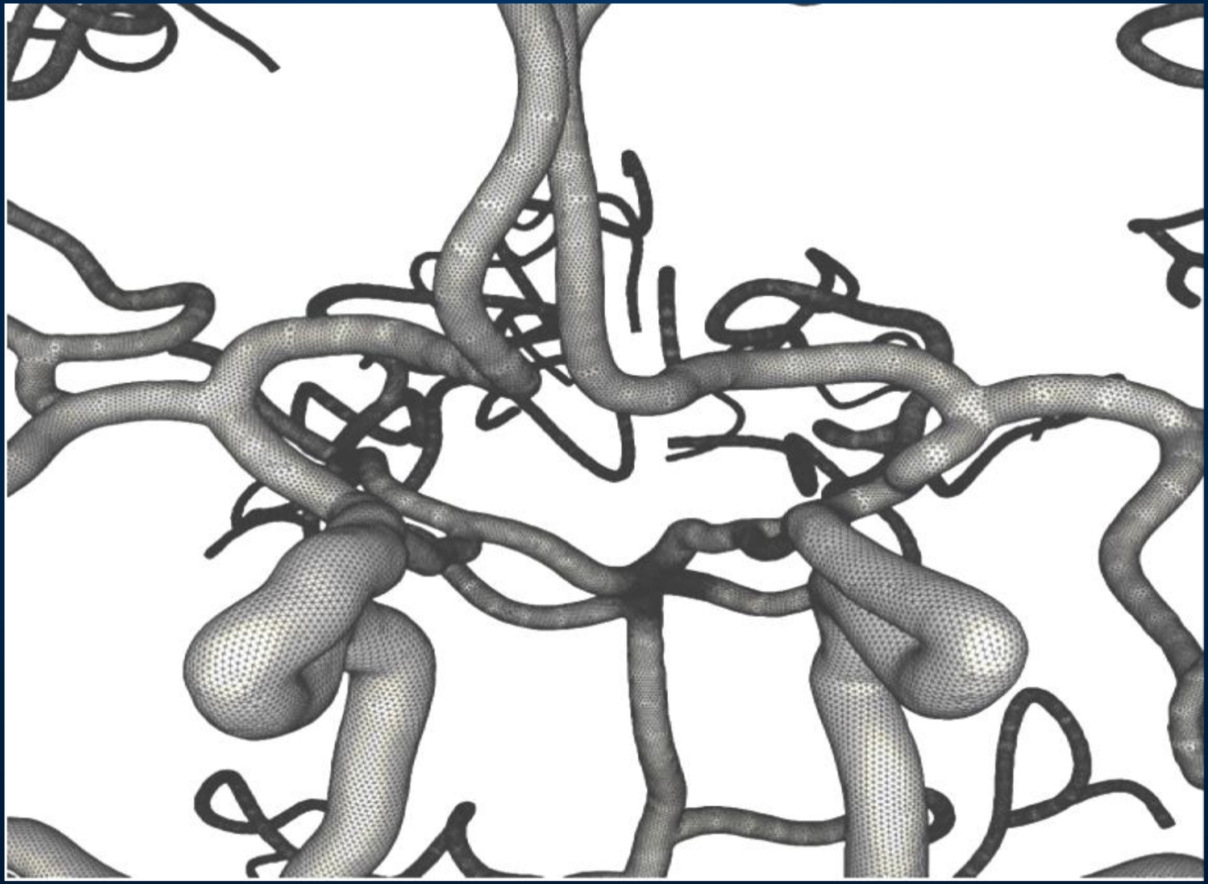


Semi-manual reconstruction
Vector representation of
arterial network



FINITE ELEMENT MESH

Advancing front method
>20 million tetrahedra



CONCLUSIONS/ OUTCOME

Patient-specific CFD models are capable of realistically representing the *in vivo* hemodynamic characteristics

These models can be used to better understand the mechanisms of aneurysm growth and rupture

They can also be used to answer specific clinical questions and to improve aneurysm risk assessment

Simulation-assisted treatment planning and patient evaluation tools are becoming a reality

RESEARCH TEAM

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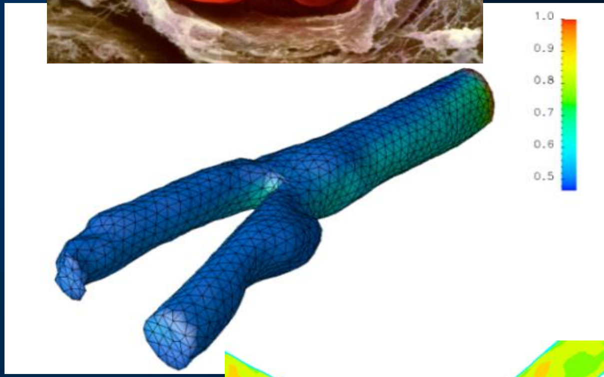

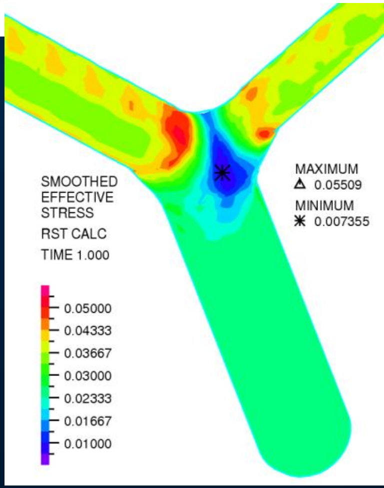

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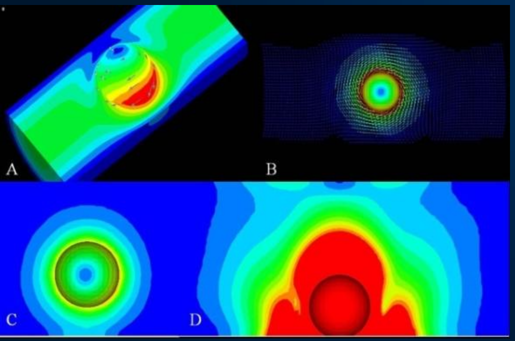

CURRENT PROJECTS

- ***Multiscale Mathematical Modelling in Biomedicine***
  PTDC/ MAT / 68166/ 2006 [2007– 2010]

- ***Cardiovascular Imaging Modeling and Simulation* – SIMCARD**
  UTAustin/CA/0047/2008 [2009 - 2012]

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A Network for Market Oriented R&D

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European Partnership: EPFL – Switzerland

Alfio Quarteroni
CMCS - Modelling and Scientific Computing Group

THANK YOU !!!