Image-based modeling of the cardiovascular system

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Outline

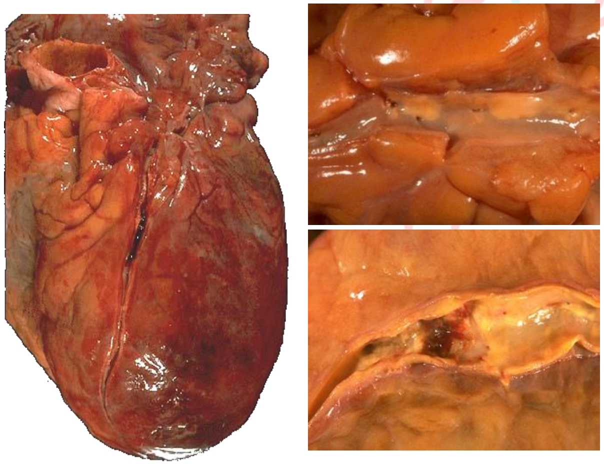
- Lecture 1: Introduction to function and modeling of the CV system
- Lecture 2: Techniques for Parameter Estimation in the CV system
- Lecture 3: Simulation of Transitional Physiology
- Lecture 4: Advanced Topics, Clinical Applications and Challenges

Lecture 1:
Introduction to function and modeling of the CV system

Introduction

- Cardiovascular disease (CV) is the leading cause of death in the Western World.

Coronary Artery Disease



(C) 1994 Pathology Department, Univ. of Utah

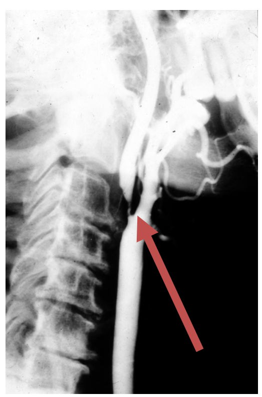
Aneurysm Disease

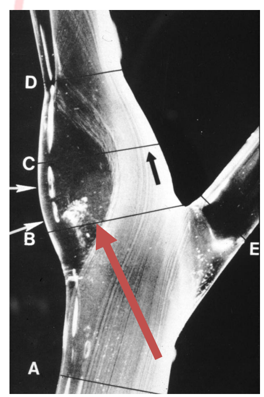
Carotid Artery Disease

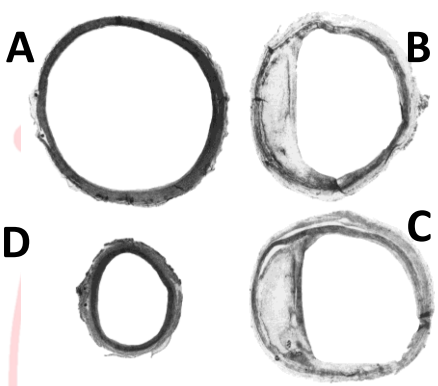
Images courtesy of C.K. Zarins

- The role of hemodynamic conditions in the pathogenesis of CV disease is now widely accepted

Images courtesy of C.K. Zarins



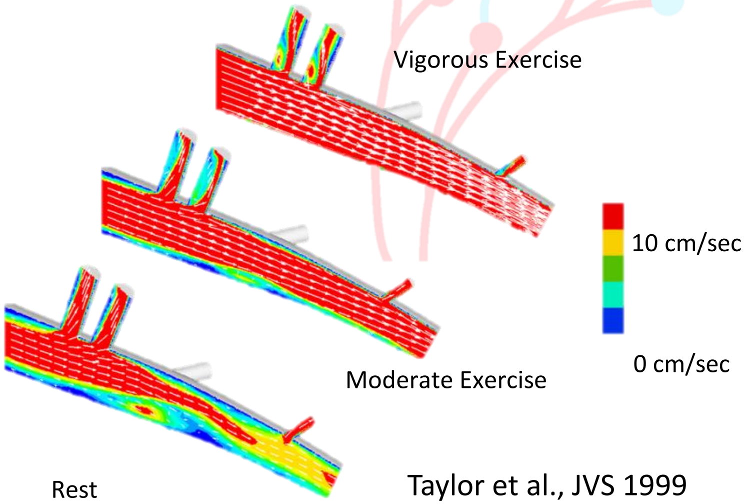




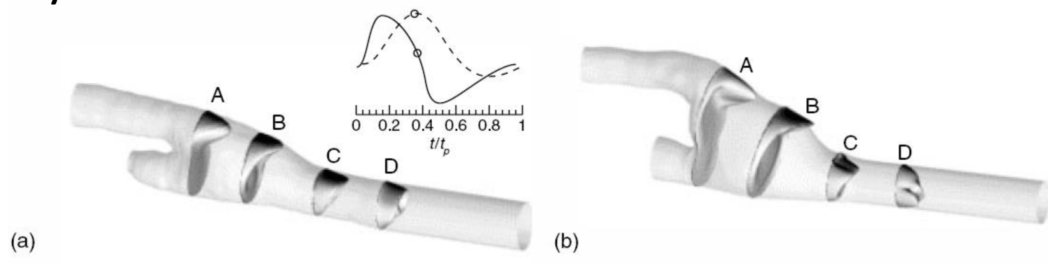
Regions of low shear stress correlate with locations of atherosclerotic lesions

Introduction

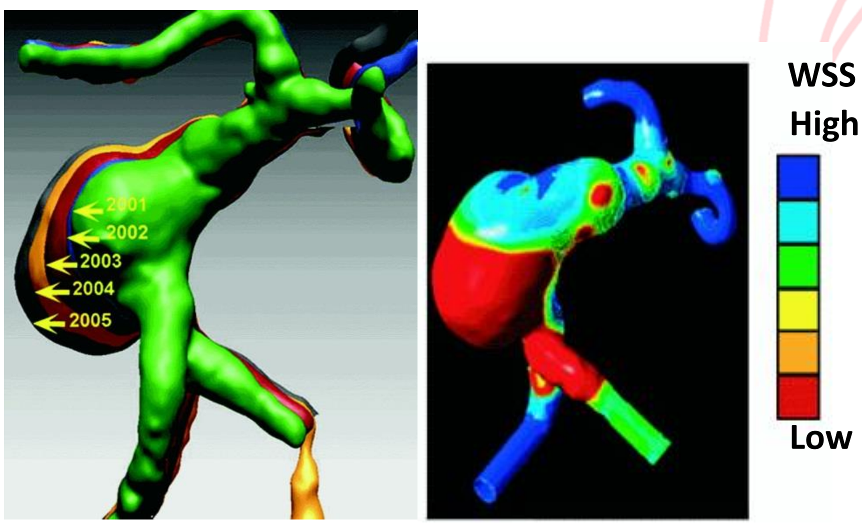
- Mathematical and computational tools have been extensively used since the mid 1990’s to model the cardiovascular system in health and disease:



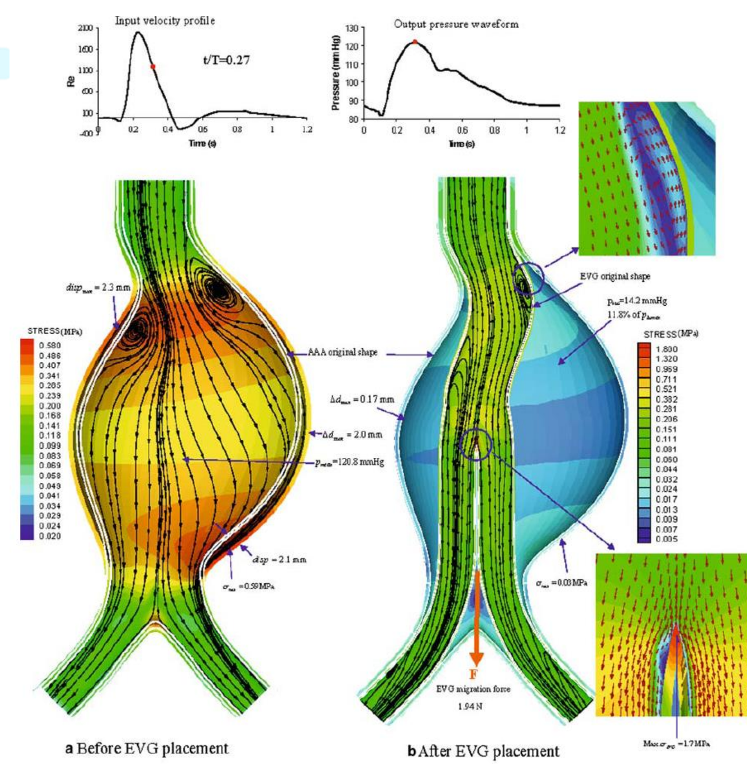
Taylor et al., JVS 1999



Leuprecht et al., J Biomech 2002



Acevedo-Bolton et al., Neurosurgery 2006



Li et al., Biomech Model Mechanobiol 2005

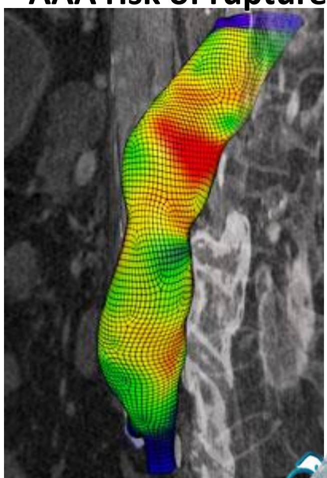
Introduction

Time-scales in Cardiovascular Modeling

seconds → **minutes** → **months & years**

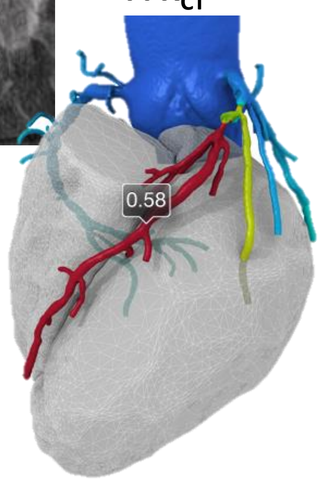
Single Cardiac Cycle

AAA risk of rupture



VASCOPS

FFR$_{CT}$

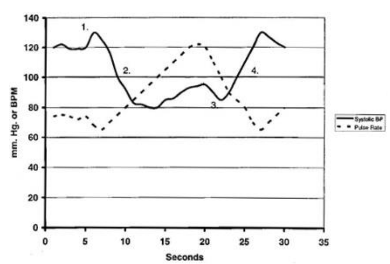


HEARTFLOW

Transitional Stages

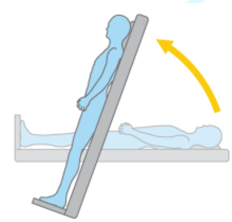
Arterial adaptations during surgery

Valsalva



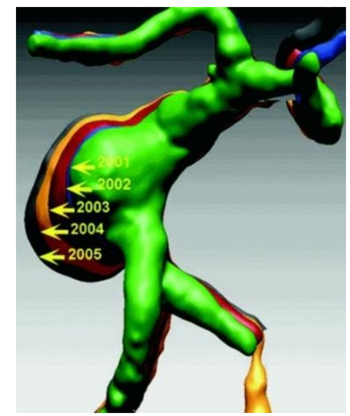
Micro-gravity

Tilt tests

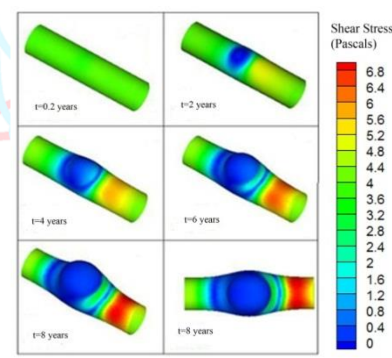


Exsanguination (trauma)

Tissue Growth & Remodeling



Acevedo-Bolton et al.



Watton et al.

The circulatory system

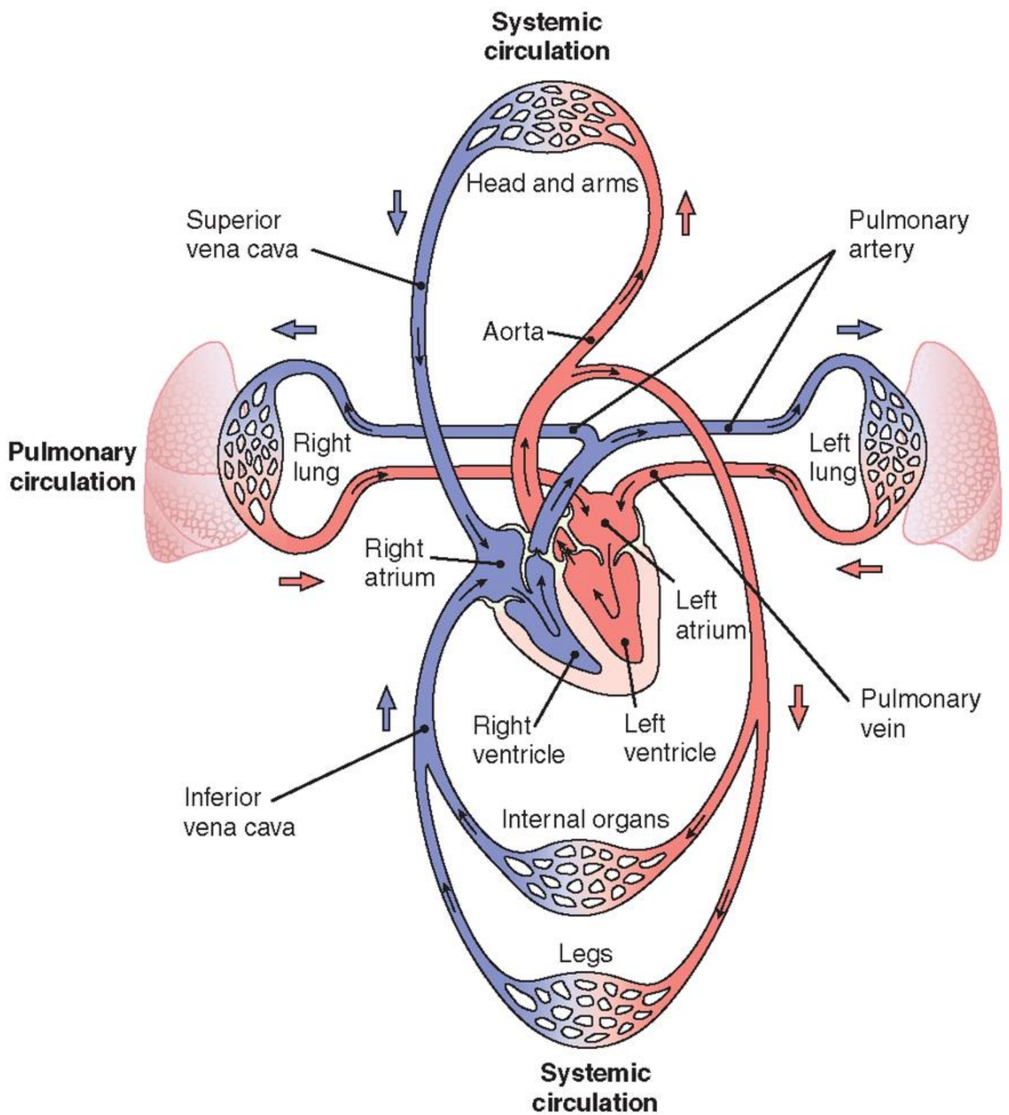
The circulatory system

- Contain & transport blood - Transport O_2, nutrients and hormones to and waste products from each of 100 trillion cells in the body
- Control heat and mass transfer
- Redistribute blood depending on end-organ demands (auto-regulation)

The circulatory system

Key Concepts:

- Closed Loop System
- Double Circulation
  - Systemic & Pulmonary
- Pumps in Series

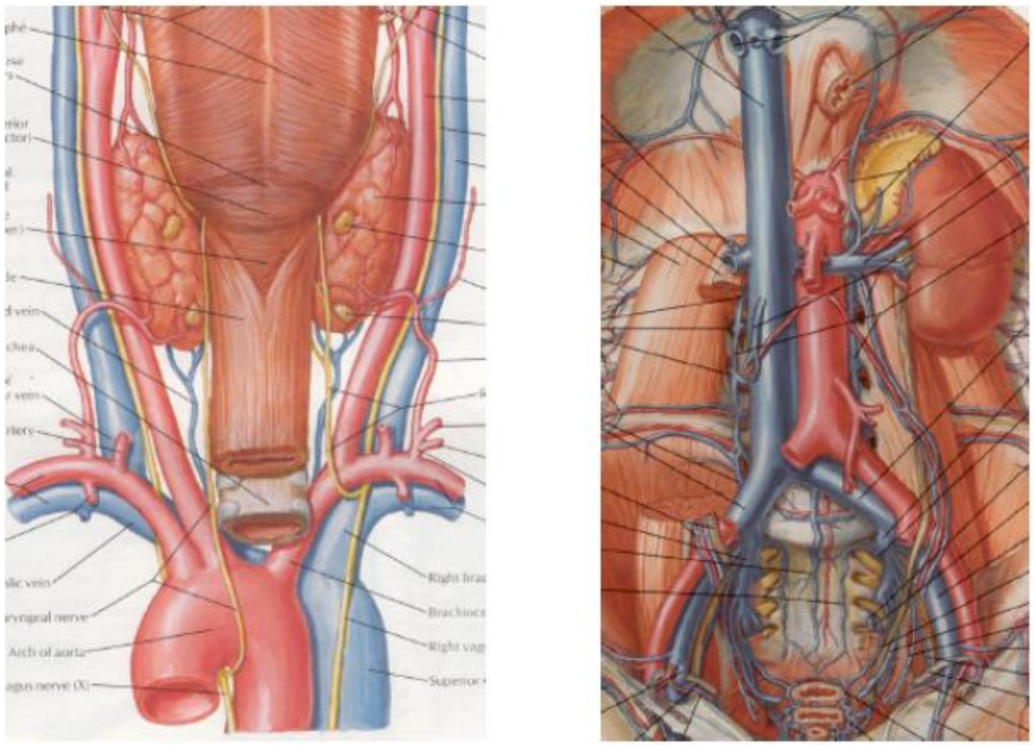


genius.com

The circulatory system

Key Concepts:

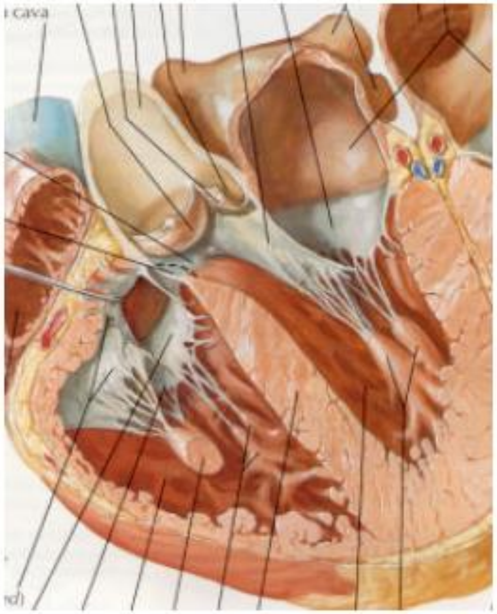
- Arteries and Veins run in parallel

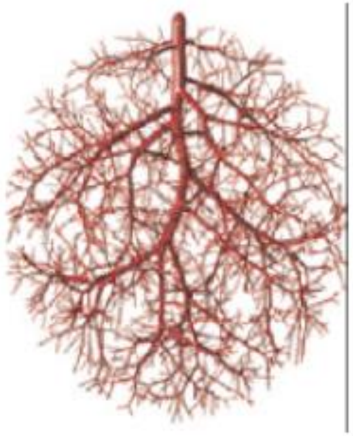


Atlas of Human Anatomy. Frank Netter

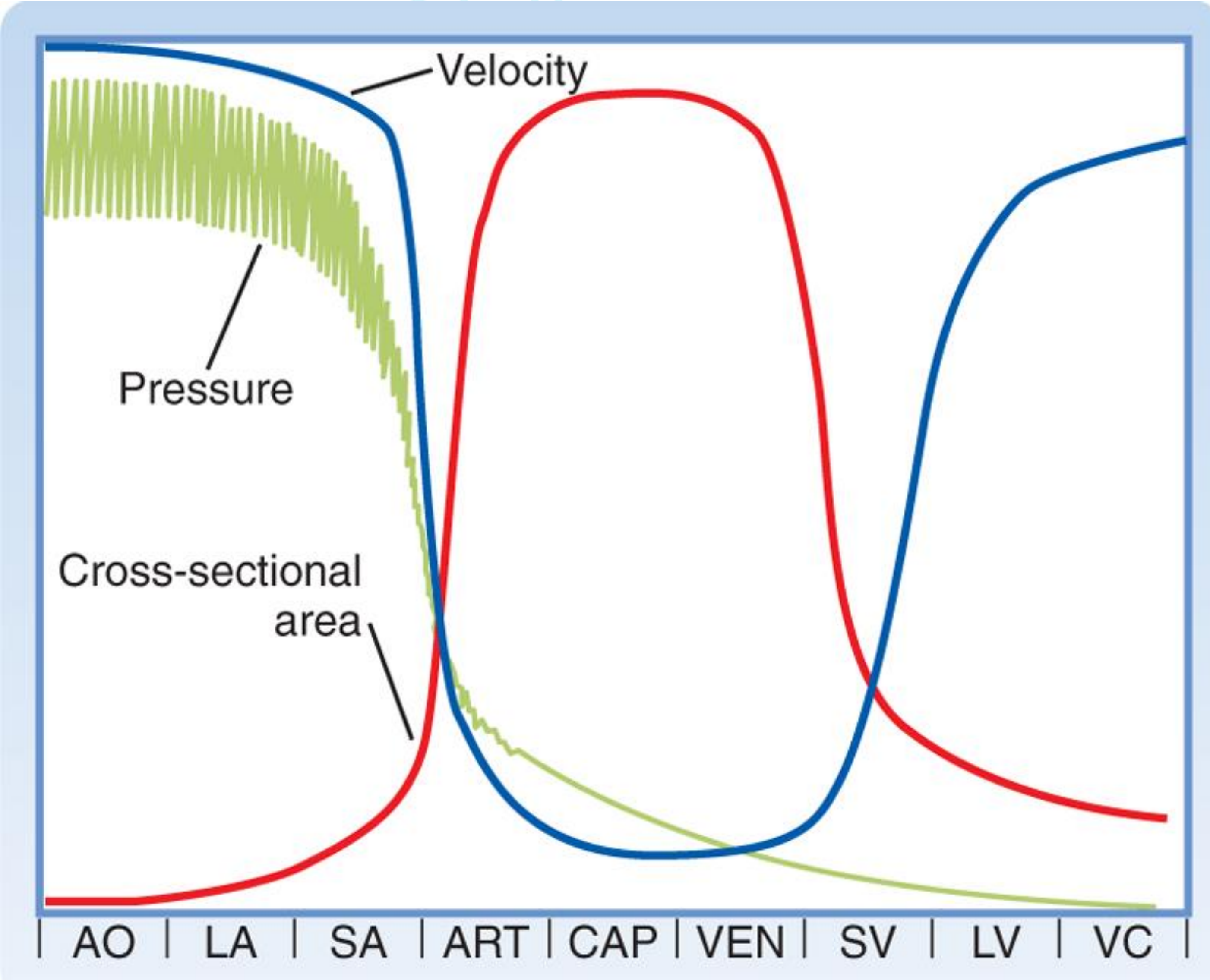
The circulatory system

- Form follows function
  - Arteries thicker than veins since pressure is higher
  - Left ventricle more muscular than right
  - Vessel luminal size depends on volume flow rate
  - Vascular networks distribute blood from single source and “fill space” by fractal-like geometry





The circulatory system



AO: Aorta

LA: Large Arteries

SA: Small Arteries

ART: Arterioles

CAP: Capillaries

VEN: Venules

SV: Small Veins

LV: Large Veins

VC: Venae Cavae

Berne and Levy, 6th edition

The circulatory system

Table 15-1. Vascular Dimensions in a 20-kg Dog

Vessels	Number	Total Cross-Sectional Area (cm^2)	Total Blood Volume (%)
Systemic			
Aorta	1	2.8	
Arteries	40 to 110,000	40	11
Arterioles	2.8×10^6	55	
Capillaries	2.7×10^9	1357	5
Venules	1×10^7	785	
Veins	110 to 660,000	631	67
Venae cavae	2	3.1	
Pulmonary			
Arteries and arterioles	$1\text{-}1.5 \times 10^6$	137	3
Capillaries	2.7×10^9	1357	4
Venules and veins	2×10^6 to 4	210	5
Heart			
Atria	2		5
Ventricles	2		

Blood

Blood transports:

- Oxygen and carbon dioxide between lungs and cells
- Nutrients, enzymes, and hormones to cells
- Waste to kidneys
- Heat to and from the cells

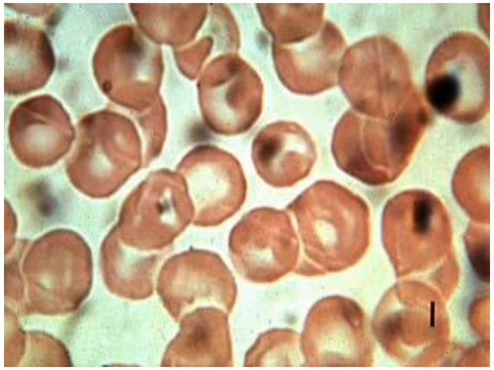
Composition of Blood

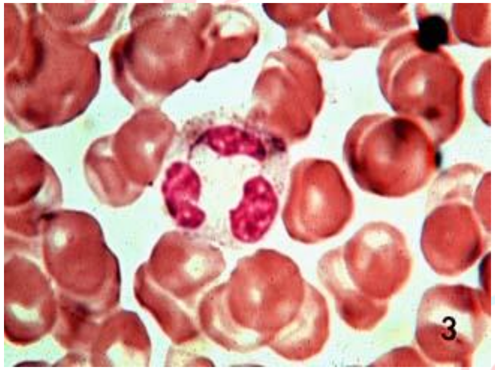
Approximately 5 liters of blood consisting of cells and plasma

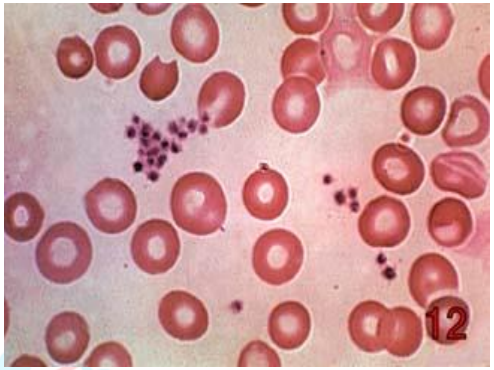
- Blood Cells (50% by volume)
- Hematocytes (Greek)

“Haima” = “blood”

“kytes” = “cell”



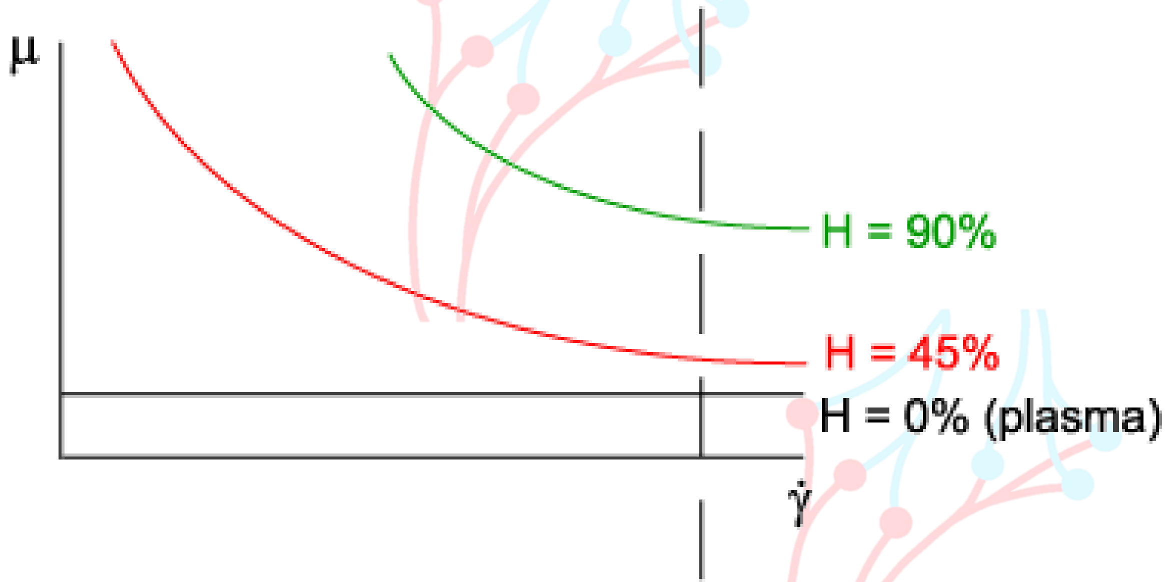




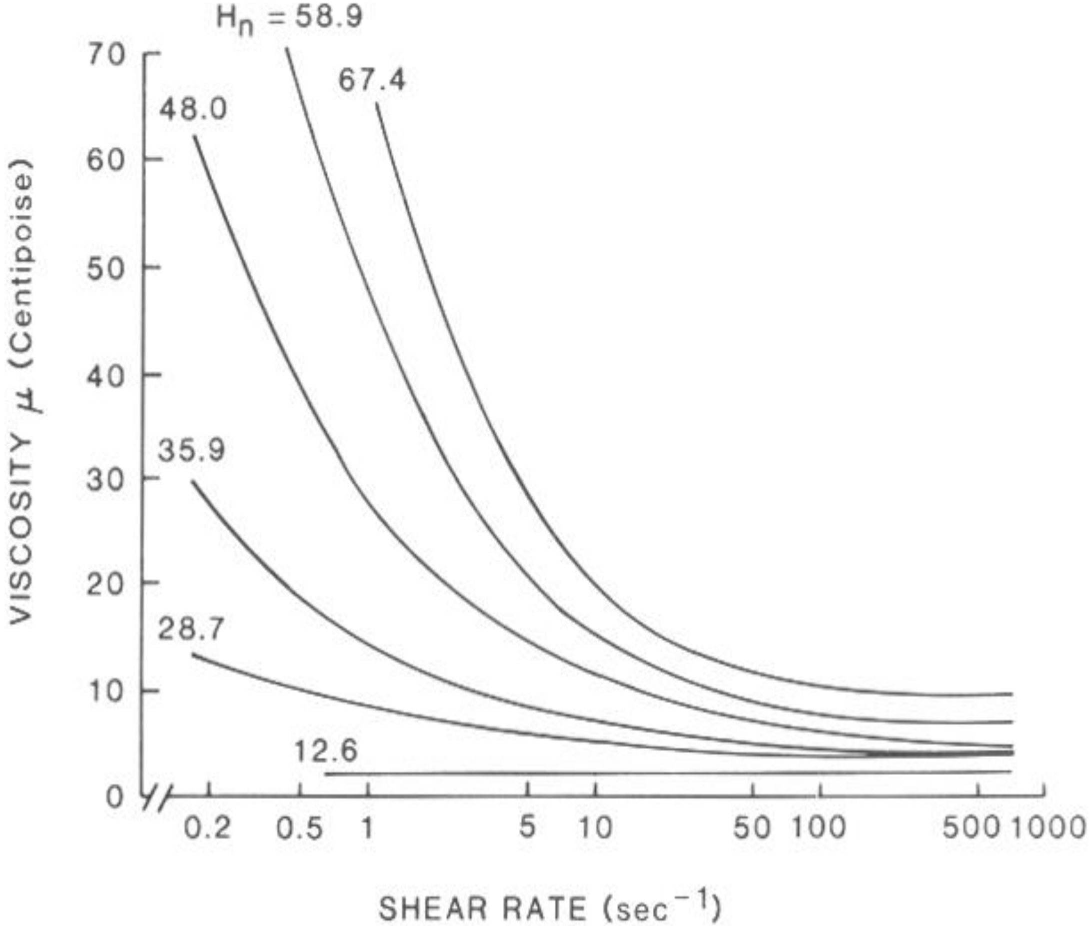
Blood contains red blood cells (erythrocytes), white blood cells (leukocytes) and platelets (monocytes)

Behavior of Blood

- The blood viscosity $\mu=\mu(H)$ where H is **hematocrit** (% of volume occupied by cells)
- At a given shear rate, ↑ H will result in ↑ μ
- Blood is **non-Newtonian** and viscosity also depends on hematocrit



Behavior of Blood

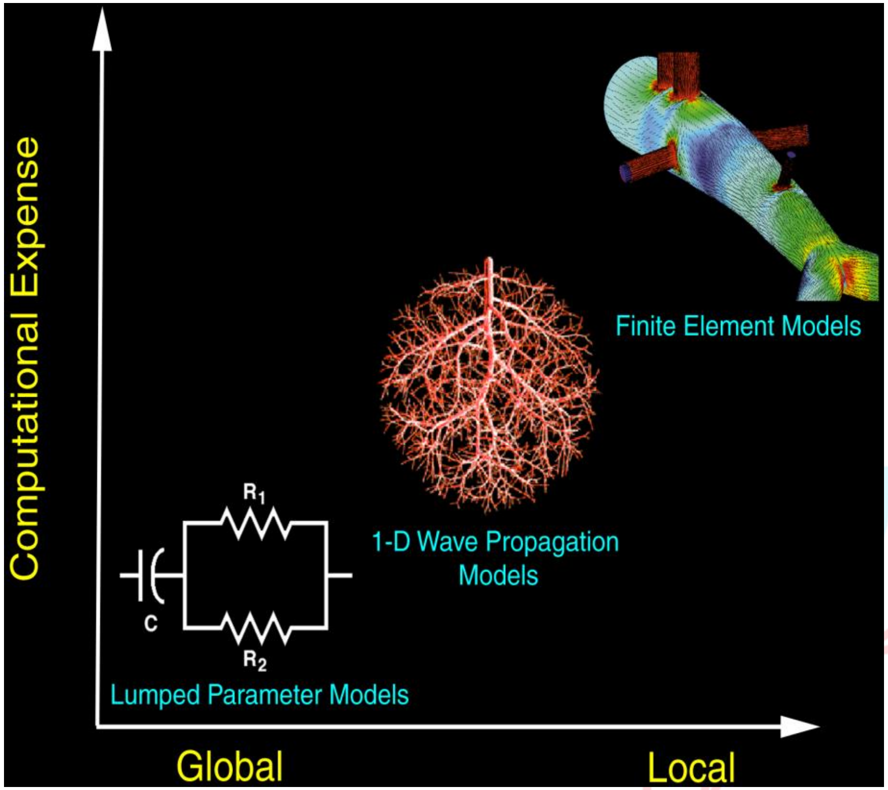


Blood exhibits "shear thinning" behavior. Viscosity increases with increased percentage of red blood cells (Hematocrit)

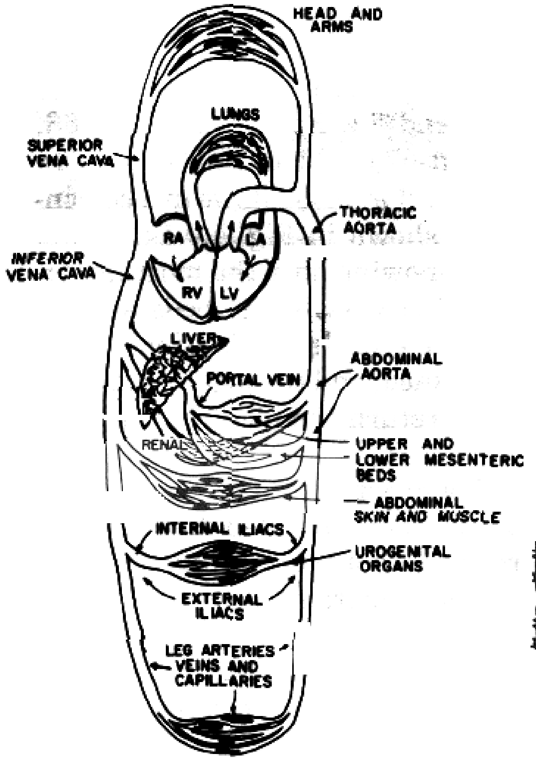
Modeling approaches

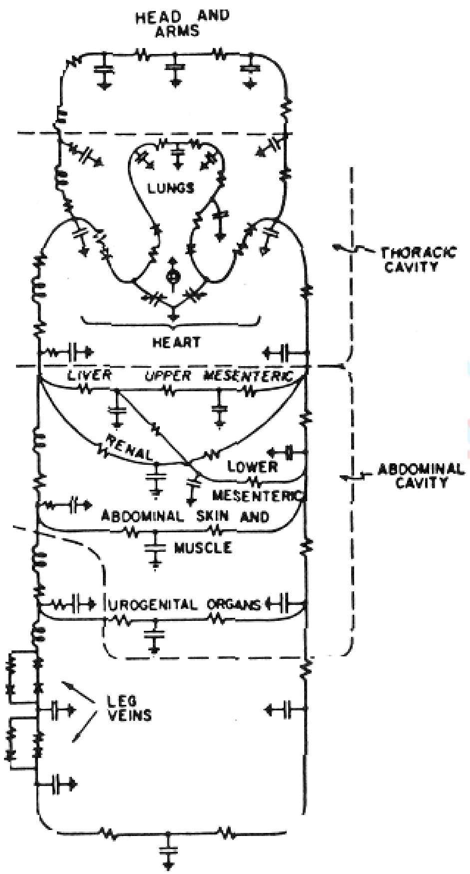
Modeling of the CV system

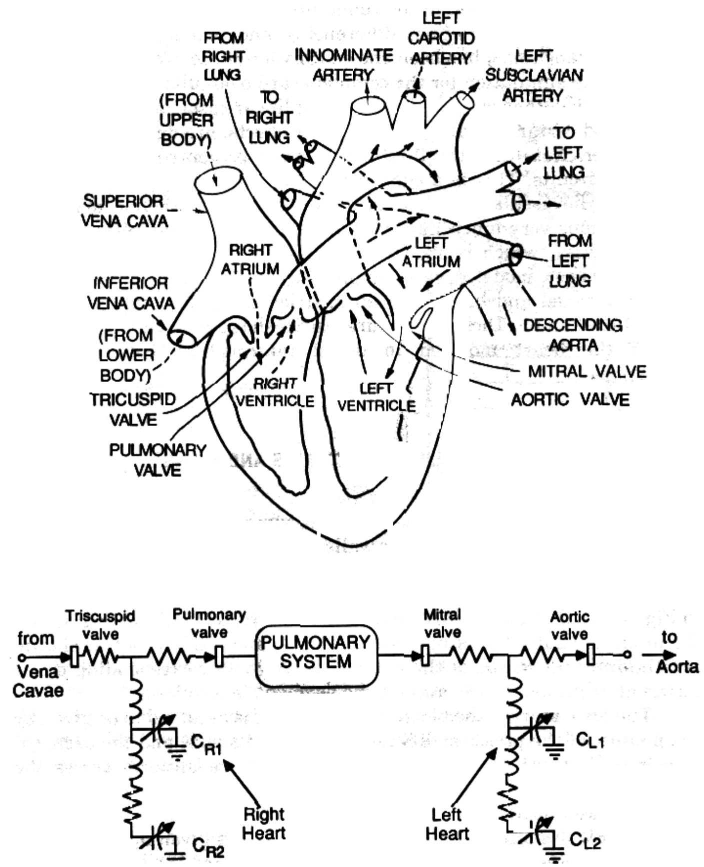
The cardiovascular system is extraordinarily complex and vast! We need tools ranging from simple lumped parameter methods to sophisticated 3D numerical techniques.



Lumped parameter methods



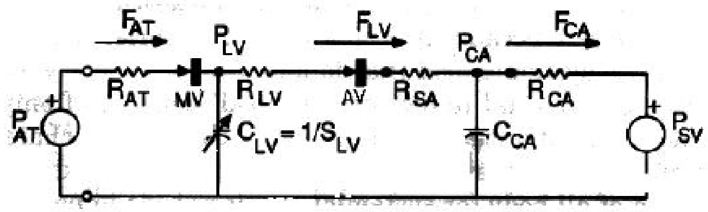




Lumped parameter methods

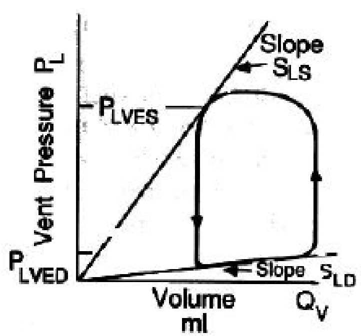
- Cardiac mechanics simulated using time varying capacitors and diodes.

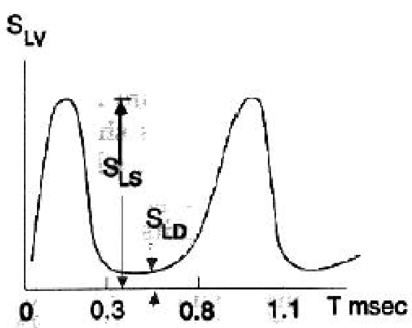
Atrial pressure (assumed constant)



Systemic Venous pressure (assumed constant)

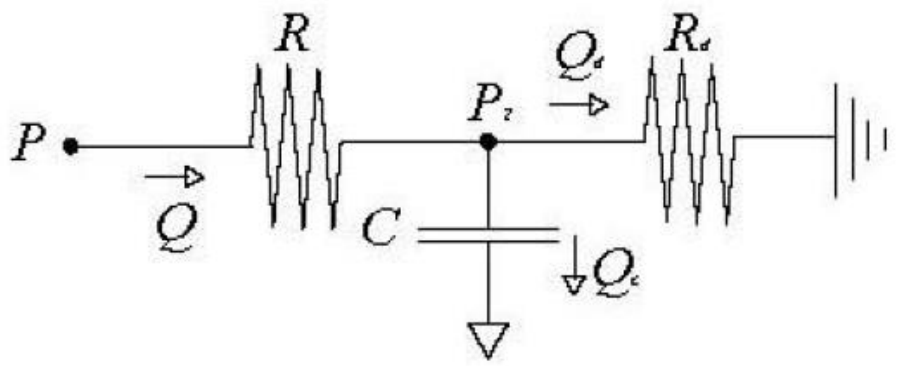
(a)





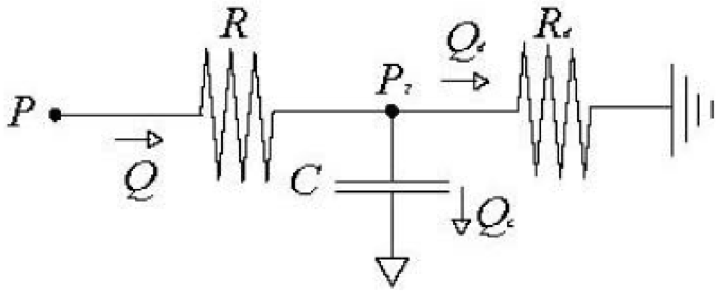
The 3-element Windkessel

- We can now assemble a simple model of an arterial tree, the RCR circuit.



The 3-element Windkessel

- The following equations that govern pressure and flow can be obtained by circuit analysis on the RCR circuit model.



$$Q = Q_c + Q_d$$

$$Q_c = C\frac{dP_2}{dt}$$

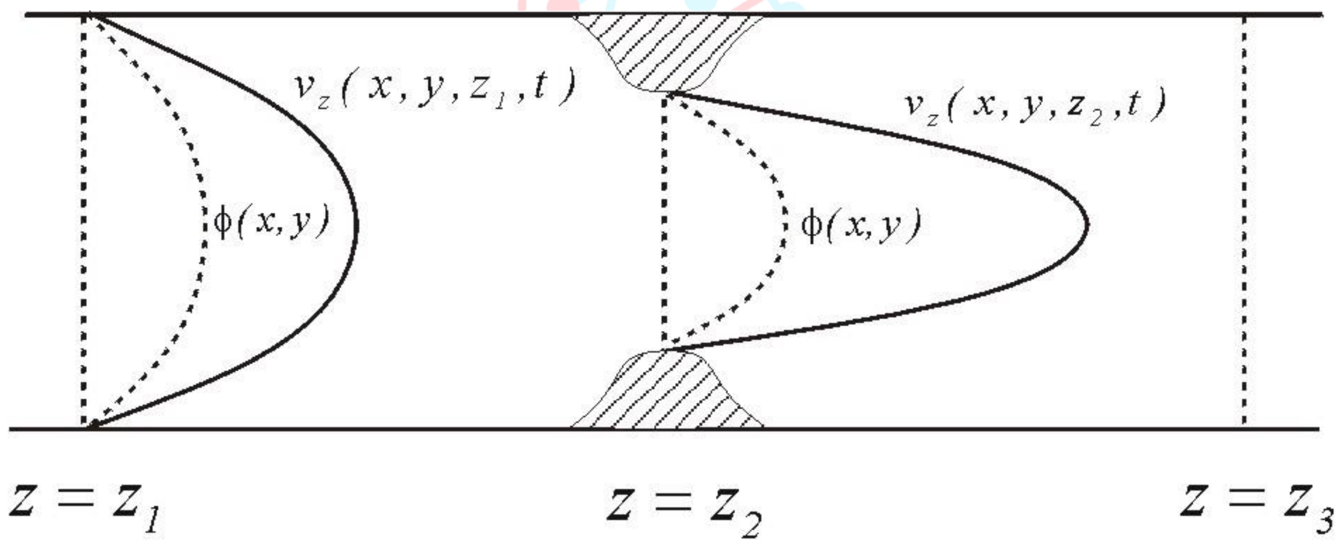
$$P - QR - P_2 = 0$$

$$P_2 - Q_d R_d = 0$$

$$\frac{dP}{dt} + \frac{1}{\tau}P = R\frac{dQ}{dt} + \frac{1}{\tau}(R + R_d)Q$$

One-dimensional theories for blood flow

- Velocity: Only consider the axial component of velocity and assume it can be written as $v_z = \phi(x,y)v(z,t)$
  - $\phi(x,y)$ is a **given** profile function, often chosen to be parabolic.
  - $v(z,t)$ is the unknown **mean** velocity.



Non-linear one-dimensional theories

- We have three variables, v, p, and S, all functions of time and position, namely the axial coordinate, z.
- We need three equations, namely…
  - Conservation of Mass: $\dfrac{\partial S}{\partial t} + \dfrac{\partial Q}{\partial z} = 0$
  - Balance of Momentum:

$$\frac{\partial Q}{\partial t} + \frac{\partial}{\partial z}\left[(1+\delta)\frac{Q^2}{S}\right] + \frac{S}{\rho}\frac{\partial p}{\partial z} = Sf + N\frac{Q}{S} + \nu\frac{\partial^2 Q}{\partial z^2}$$

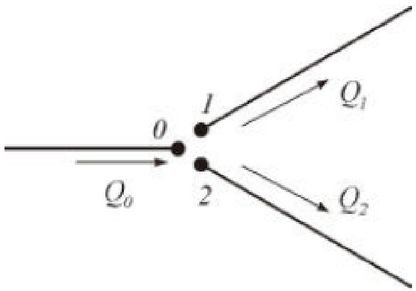
  - Constitutive Equation:

$$S = \hat{S}(p, z, t)$$

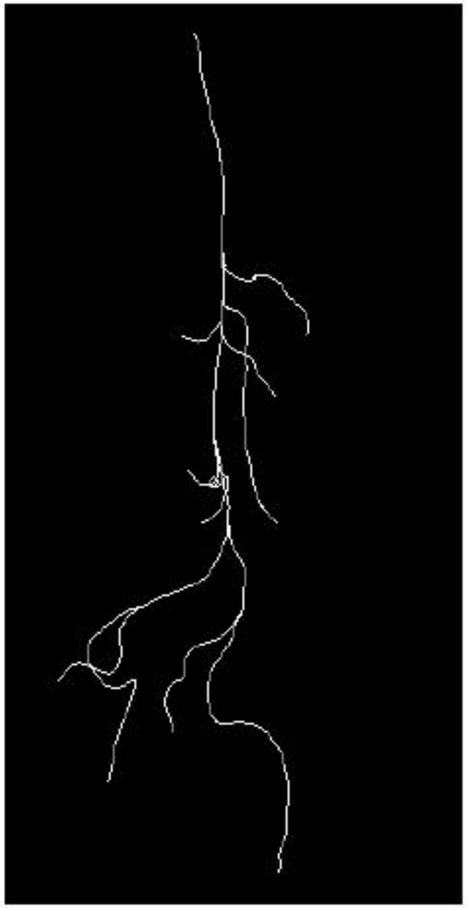
Non-linear one-dimensional theories

- δ and N depend on the shape of the velocity profile. For a parabolic profile function, $\delta = \frac{1}{3}$ and $N = -8\pi\nu$.
- f is a body force, e.g. gravity.
- Equations are **nonlinear** and must be solved numerically.
- Branches can be included by enforcing mass conservation and pressure continuity across a branch.

$$Q_0 = Q_1 + Q_2$$
$$p_0 = p_1 = p_2$$



Non-linear one-dimensional theories



1-D Model

3-D model

Non-linear one-dimensional theories

- Minor loss coefficient K describes the pressure loss in viscous flow caused by constrictions, branching, curvature, etc.

$$\Delta p = K \frac{1}{2} \rho V^2$$

- Incorporate additional pressure losses into momentum balance equation by assuming constant flux, no external forces, and integrating over the length

$$\Delta p = N \rho \frac{Q}{S^2} L$$

Linear one-dimensional theories

- A linear version of the one-dimensional wave theory of blood flow is found by simplifying nonlinear terms, and using a constitutive equation to obtain partial differential equations in *p* and *v*.

$$\frac{\partial p}{\partial t} + \rho c_o^{\ 2} \frac{\partial v}{\partial z} = 0$$

$$\rho \frac{\partial v}{\partial t} + \frac{\partial p}{\partial z} + f_o v = 0$$

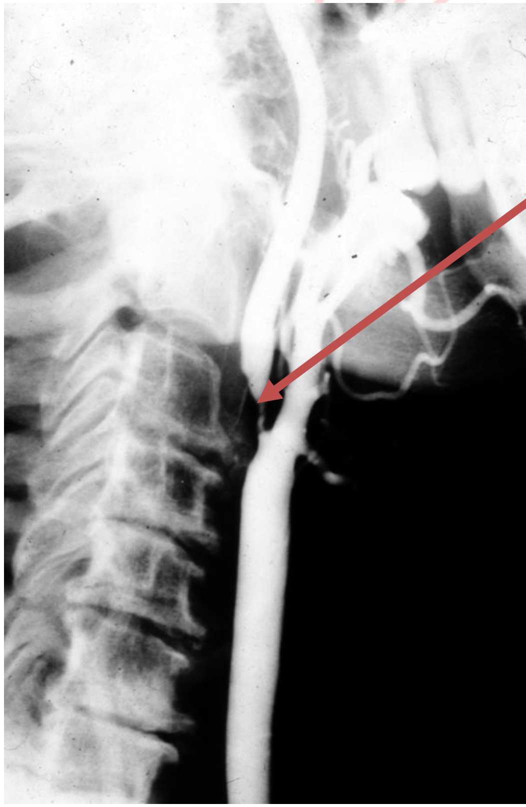
- These give the usual 'wave equation'

Computational (3D) methods: BCs, FSI, etc

Computational Fluid Dynamics

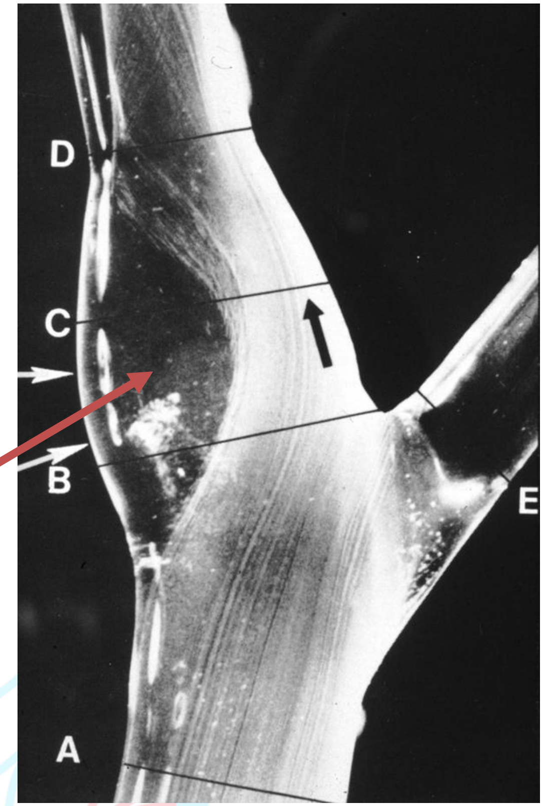
Blood flow in major arteries (where disease predominantly occurs and devices go) is three-dimensional in nature. While simpler lumped parameter and one-dimensional models may be suitable for predicting flow rate and pressure these methods yield no insight into three-dimensional flow features.

Blood Flow Affects Progression of Disease



Focal plaque in human carotid artery (can lead to strokes)

Flow recirculation demonstrated in glass model of human carotid artery



Zarins et al, 1983

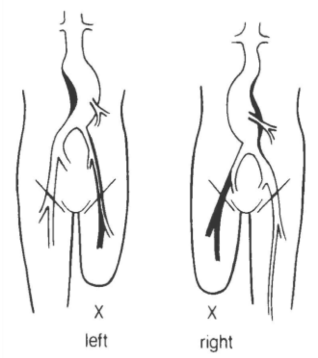
Blood Flow Affects Progression of Disease

Blood flow forces impact the progression and rupture of aneurysms by degrading extracellular matrix in vessels[1]

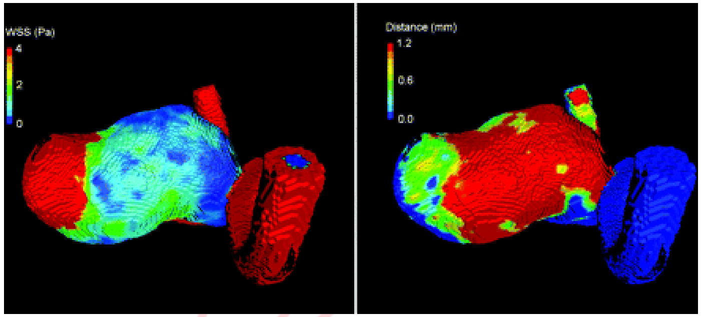
M.K. O'Connell, S. Murthy, S. Phan, C. Xu, J. Buchanan, R. Spilker, R.L. Dalman, C.K. Zarins, W. Denk, C.A. Taylor (2008) The Three-Dimensional Micro- and Nanostructure of the Aortic Medial Lamellar Unit Measured Using 3D Confocal & Electron Microscopy Imaging. Matrix Biology. Vol. 27, No. 3, pp. 171–181.

Lamellar Structure of Elastic Vessels

Human AAA



AAA in amputees

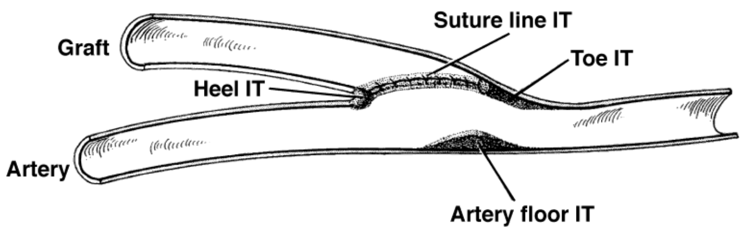


Low shear correlates with regions of cerebral aneurysm growth

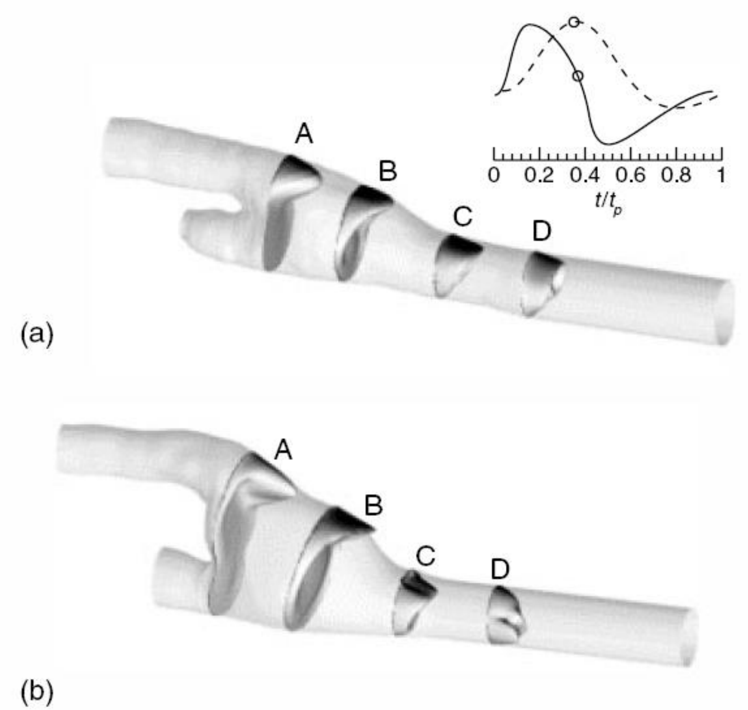
Boussel, L. et al. Stroke 2008;39:2997-3002

1. J.D. Humphrey, C.A. Taylor (2008) Intracranial and Abdominal Aortic Aneurysms: Similarities, Differences and Need for a New Class of Computational Models. Annual Review of Biomedical Engineering, Vol. 10, pp. 221-246.

Blood Flow Affects Graft Patency

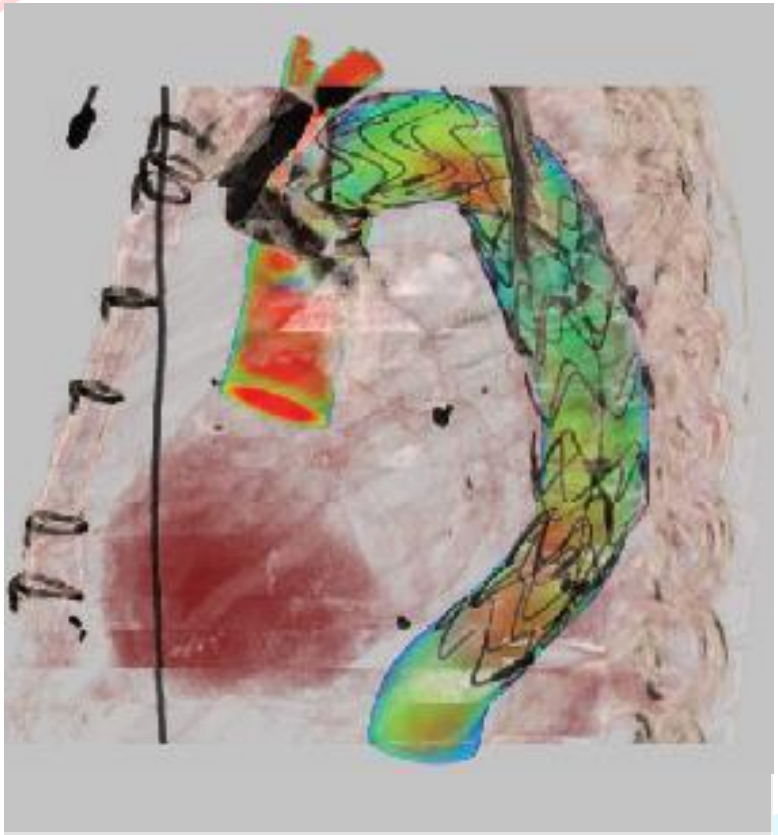


Patterns of Intimal Hyperplasia in end-to-side graft-artery anastomosis



Computed velocity patterns in two models of graft-artery bypass junctions. Solid line represents flow rate, dashed line represents pressure pulse. (Reprinted from *Journal of Biomechanics, 35, Leuprecht et al., Numerical study of* hemodynamics and wall mechanics in distal end-to-side anastomosis of bypass grafts, 225–236, Copyright 2002, with permission from Elsevier.)

Blood Flow Affects Device Performance



C.A. Figueroa, C.A. Taylor, A.J. Chiou, V. Yeh, C.K. Zarins (2009) Magnitude and Direction of Pulsatile Displacement Forces Acting on Thoracic Aortic Endografts. To appear in Journal of Endovascular Therapy.

3-D Incompressible Navier-Stokes Eqns.

Strong Form is:

$$\rho\vec{v}_{,t} + \rho\vec{v}\cdot\nabla\vec{v} = -\nabla p + \mu\Delta\vec{v}$$

$$\nabla\cdot\vec{v} = 0 \qquad (\vec{x},t)\in\Omega\times(0,T)$$

$$\vec{v} = \vec{g} \qquad (\vec{x},t)\in\Gamma_g\times(0,T)$$

$$\vec{t}_{\vec{n}} = \underset{\sim}{\sigma}\vec{n} = \vec{h} \qquad (\vec{x},t)\in\Gamma_h\times(0,T)$$

We have 3 momentum balance equations and 1 incompressibility equation

Our weak form is:

$$\boxed{\int_\Omega \left\{ \vec{w}\cdot\left(\rho\vec{v}_{,t} + \rho\vec{v}\cdot\nabla\vec{v} - \vec{f}\right) + \nabla\vec{w} : \left(-p\underset{\sim}{I} + \mu\Delta\vec{v}\right) - \nabla q\cdot\vec{v} \right\} d\vec{x} - \int_{\Gamma_h} \vec{w}\cdot\vec{h}\,ds + \int_{\Gamma_s} q v_n\,ds = 0}$$

Our unknowns are v_i and p. We have weighting functions w_i for the momentum balance equation and another weighting function, q, for the incompressibility constraint. This is once again a “mixed” problem”.

Finite Element Method

Remarks:

1. Dramatic spatial oscillations in velocity and pressure fields arise with Galerkin's formulation for fluid mechanics, due to nature of flow equations

2. Stabilized methods that accomodate equal order interpolations are found in many commercial FEA codes. These methods are often called "SUPG" (Streamline Upwind Petrov Galerkin) methods.

Finite Element Method

Stabilized Finite Element Methods involve adding "Residual-Based" terms to Galerkin's method to ameliorate stabilization and convergence issues due to coarse meshes

$$\int_{\Omega}\left\{\vec{w}\cdot\left(\rho\vec{v}_{,t}-\vec{f}\right)+\nabla\vec{w}:\left(-p\underset{\sim}{I}+\mu\Delta\vec{v}-\rho\vec{v}\otimes\vec{v}\right)\right\}d\vec{x}-\int_{\Gamma_h}\vec{w}\cdot\left(-p\underset{\sim}{I}+\underset{\sim}{\tau}\right)\cdot\vec{n}ds$$

$$+\sum_{e=1}^{n_{el}}\int_{\bar{\Omega}_e}\left\{\nabla\vec{w}:\left(\tau_M\vec{L}^{-}\otimes\vec{v}\right)+\nabla\cdot\vec{w}\tau_C\nabla\cdot\vec{v}\right\}d\vec{x}$$

Residual-based stabilization term

$$-\int_{\Omega}\nabla q\cdot\vec{\vec{v}}d\vec{x}+\int_{\Gamma_g}q\vec{v}\cdot\vec{n}ds+\int_{\Gamma_h}q\vec{v}\cdot\vec{n}ds=0$$

Finite Element Method

Remarks:

3. Primary variables computed are velocity and pressure fields. Derived quantities: flow rate, shear stress and shear rate are also of interest. If velocity field is accurately represented, volume flow rate will be as well – not necessarily true for shear stress or shear rate.

4. Velocity profiles can be well represented in simple and moderately complex flows *in vitro* (not true for any method or program, but have been demonstrated by a few groups).

Finite Element Method

Remarks:

5. Pressure gradients are accurate if velocities are accurate, but pressure fields can still be "dead wrong". How can this be? Let's look at governing equations ...

$$\rho \vec{v}_{,t} + \rho \vec{v} \cdot \nabla \vec{v} = -\nabla p + \mu \Delta \vec{v}$$

We only see the pressure gradient term in this equation.

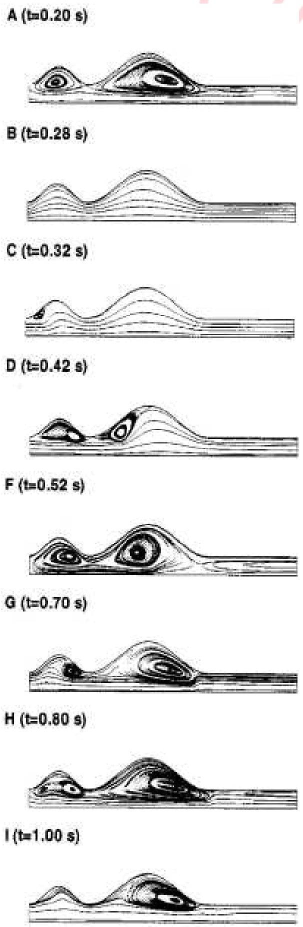
Q: How do we get absolute pressures?

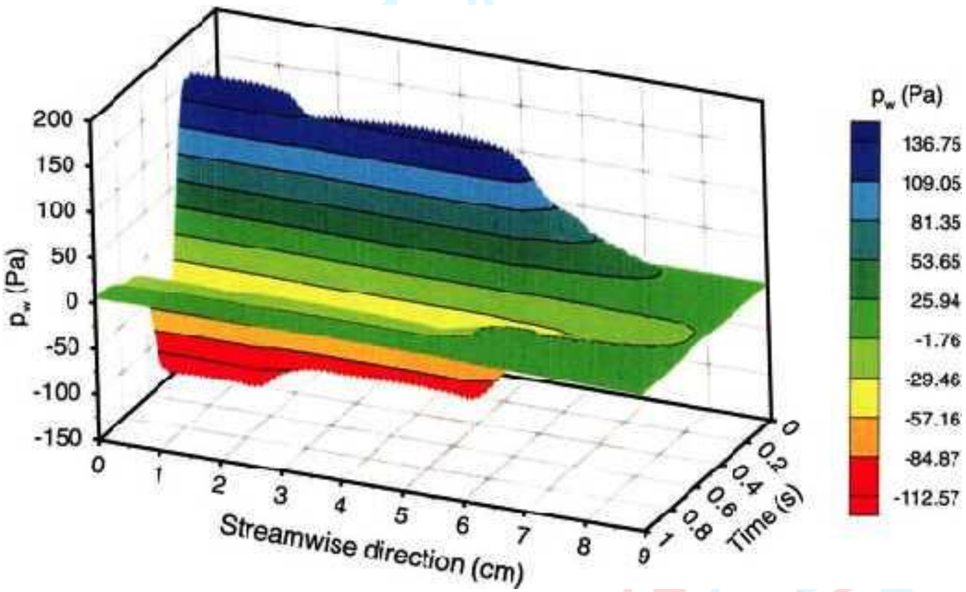
A: The boundary conditions.

The problem with computing pressures emanates from the fact that pressure is usually set to be zero at one (or more) boundaries.

What's wrong with these pictures?

Pressure distribution in axisymmetric Abdominal Aortic Aneurysm





≈ -0.84 mmHg

Common inlet boundary conditions

- Prescribed velocity field

$$v(\vec{x},t) = g(\vec{x},t) \quad \vec{x} \in \Gamma_g, t \in (0,T) \quad \text{"Dirichlet" boundary condition}$$

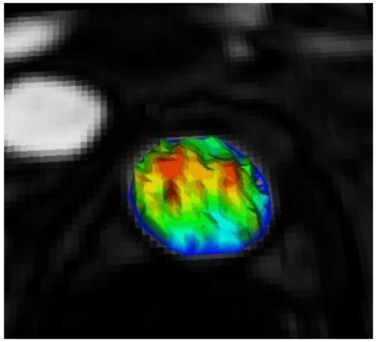
- Functional space requirement:

$$\boldsymbol{S} = \left\{ \vec{v} \,\middle|\, \vec{v}(\cdot,t) \in H^1(\Omega)^{n_{sd}}, t \in [0,T], \vec{v}(\cdot,t) = \vec{g} \text{ on } \Gamma_{in} \right\}$$

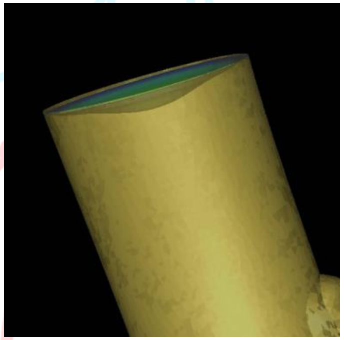
$$\boldsymbol{W} = \left\{ \vec{w} \,\middle|\, \vec{w}(\cdot,t) \in H^1(\Omega)^{n_{sd}}, t \in [0,T], \vec{w}(\cdot,t) = \vec{0} \text{ on } \Gamma_{in} \right\}$$

- In practice velocities often come from MRI:

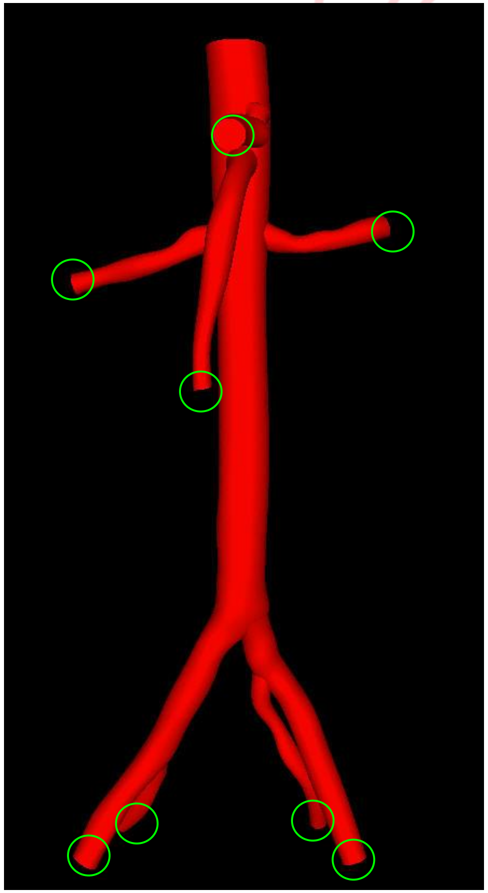
As they are measured (usually filtered)



The measured mean flow rate is mapped onto a velocity profile



Simplest outflow boundary conditions



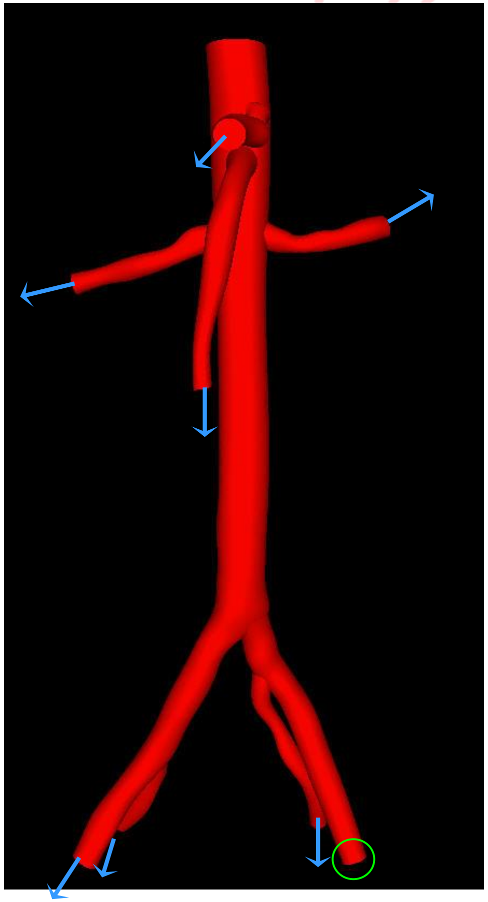
The "traction-free" approach: (Neumann BC)
Zero mean traction or pressure is prescribed at every outlet

$$\int_{\Gamma_h} \vec{w} \cdot \vec{h} ds = 0$$

Proposed Advantage:
Very easy to prescribe

Issues:
- Non physiologic level of pressure
- Problematic for multiple outlets
- Not applicable to deformable wall simulations

Simplest outflow boundary conditions



The "velocity approach":

- Velocity prescribed at all outlets but one

$$v(\vec{x},t) = g(\vec{x},t)$$

- Zero traction/pressure at the last one

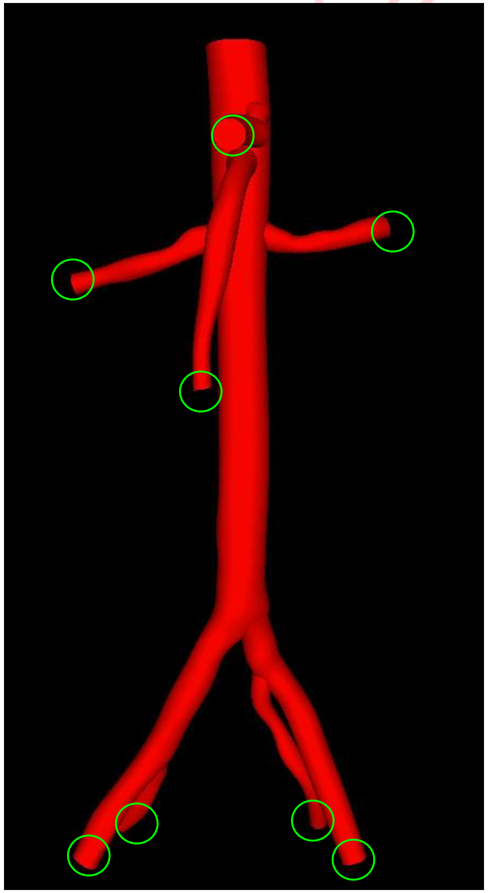
$$\int_{\Gamma_h} \vec{w} \cdot \vec{h} ds = 0$$

Proposed Advantage:
Control over flow splits

Issues:

- Non physiologic level of pressure
- Rigid walls: not easy to align measurements
- Deformable walls: can't account for wave propagation

Simplest outflow boundary conditions



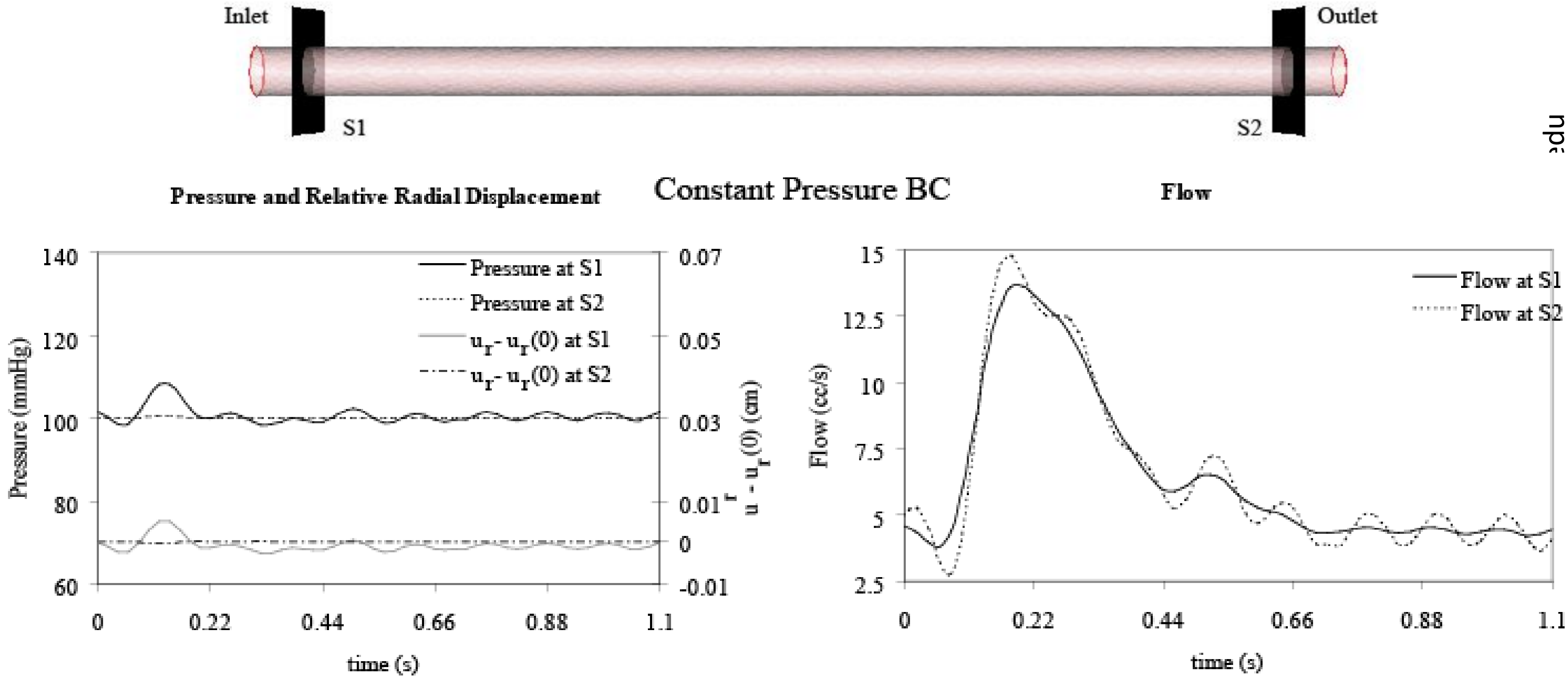
The "time varying pressure approach":
Time varying pressure prescribed at all outlets

- $$\int_{\Gamma_h} \vec{w} \cdot p\vec{n}ds = \int_{\Gamma_h} \vec{w} \cdot p(t)\vec{n}ds$$

Proposed Advantage:
If different pressures prescribed, one can control flow splits…

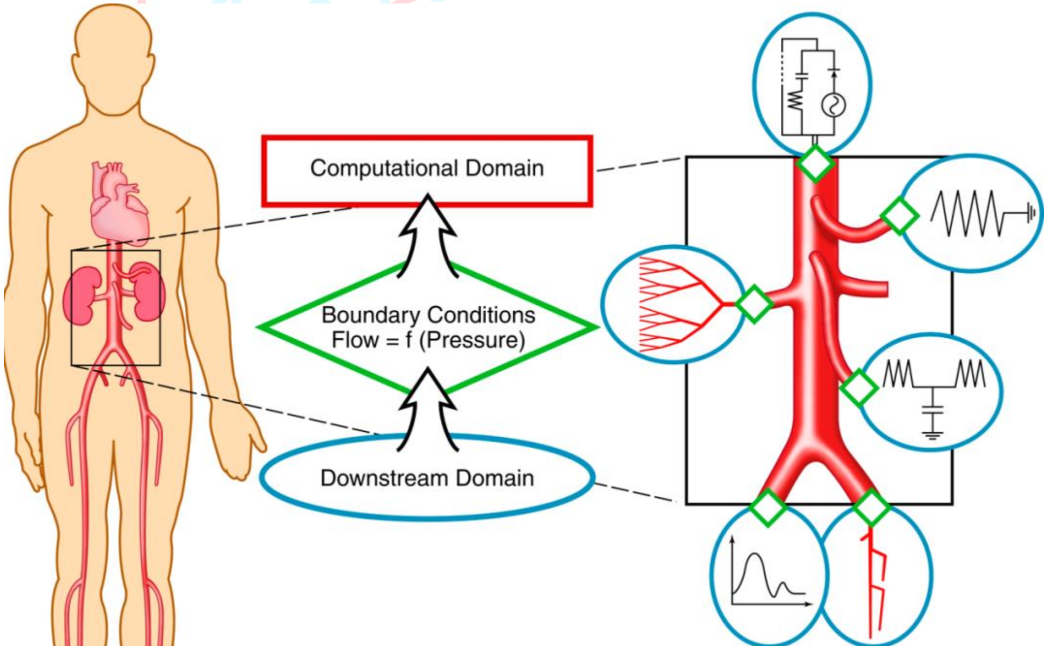
Issues:
- In practice, impossible to get measurements and to adjust them for rigid walls
- Deformable walls: can't account for wave propagation

Constant pressure outflow boundary condition



Simulations (whether physical or numerical) of blood flow in the cardiovascular system require careful consideration of the “boundary conditions”

A multi-scale (multi-resolution) approach for BCs



$$\int_{\hat\Omega} \hat{\vec{w}}\cdot\left(\rho\hat{\vec{v}}_{,t}+\rho\hat{\vec{v}}\cdot\nabla\hat{\vec{v}}-\vec{f}\right)+\nabla\hat{\vec{w}}:\left(-\hat{p}\underset{\sim}{I}+\underset{\sim}{\hat{\tau}}\right)d\vec{x}-\int_{\hat\Gamma_h}\hat{\vec{w}}\cdot\left(-\hat{p}\underset{\sim}{I}+\underset{\sim}{\hat{\tau}}\right)\cdot\hat{\vec{n}}ds$$

$$\boxed{-\int_{\Gamma_B}\hat{\vec{w}}\cdot\left(\underset{\sim}{M}_m(\hat{\vec{v}},\hat{p})+\underset{\sim}{H}_m\right)\cdot\hat{\vec{n}}ds}-\int_{\hat\Omega}\nabla\hat{q}\cdot\hat{\vec{v}}d\vec{x}+\int_{\hat\Gamma}\hat{q}\hat{\vec{v}}\cdot\hat{\vec{n}}ds$$

$$+\boxed{\int_{\Gamma_B}\hat{q}\left(\vec{M}_c(\hat{\vec{v}},\hat{p})+\vec{H}_c\right)\cdot\hat{\vec{n}}ds}=0$$

Vignon-Clementel, Figueroa, Jansen & Taylor, CMAME 2006

Changes to Finite Element Method

Add integrals to weak form (shown in green boxes below) to incorporate more general boundary conditions that are functional relationships between flow rate and pressure.

$$
\begin{aligned}
&\int_{\Omega} \left\{ \vec{w} \cdot \left(\rho \vec{v}_{,t} - \vec{f} \right) + \nabla \vec{w} : \left(-p \underset{\sim}{I} + \underset{\sim}{\tau} - \rho \vec{v} \otimes \vec{v} \right) \right\} d\vec{x} \\
&\quad - \int_{\Gamma_h} \vec{w} \cdot \left(-p \underset{\sim}{I} + \underset{\sim}{\tau} \right) \cdot \vec{n} ds \boxed{ - \int_{\Gamma_B} \vec{w} \left(\underset{\sim}{M}_m (\vec{v}, p) + \underset{\sim}{H}_m \right) \vec{n} d\Gamma } \\
&\quad + \sum_{e=1}^{n_{el}} \int_{\bar{\Omega}_e} \left\{ \nabla \vec{w} : \left(\tau_M \vec{L} \otimes \vec{v} \right) + \nabla \cdot \vec{w} \tau_C \nabla \cdot \vec{v} \right\} d\vec{x} = 0
\end{aligned}
$$

$$
-\int_{\Omega} \nabla q \cdot \vec{\vec{v}} d\vec{x} + \int_{\Gamma_g} q \vec{v} \cdot \vec{n} ds + \int_{\Gamma_h} q \vec{v} \cdot \vec{n} ds \boxed{ + \int_{\Gamma_B} q \left(\vec{M}_c (\vec{v}, p) + \vec{H}_c \right) \vec{n} d\Gamma } = 0
$$

Operators for different downstream domains

- Resistance model $p = QR$

$$\left[\underset{\sim}{M}_m(\vec{v},p)+\underset{\sim}{H}_m\right]_{\Gamma_B} = \left(-R\int_{\Gamma_B}\vec{v}(\tau)\cdot\vec{n}\,d\Gamma\;\underset{\sim}{I} + \underset{\sim}{\tau} - \rho\vec{v}\otimes\vec{v}\right)\Big|_{\Gamma_B}$$

$$\left[\vec{M}_c(\vec{v},p)+\vec{H}_c\right]_{\Gamma_B} = \vec{v}\Big|_{\Gamma_B}$$

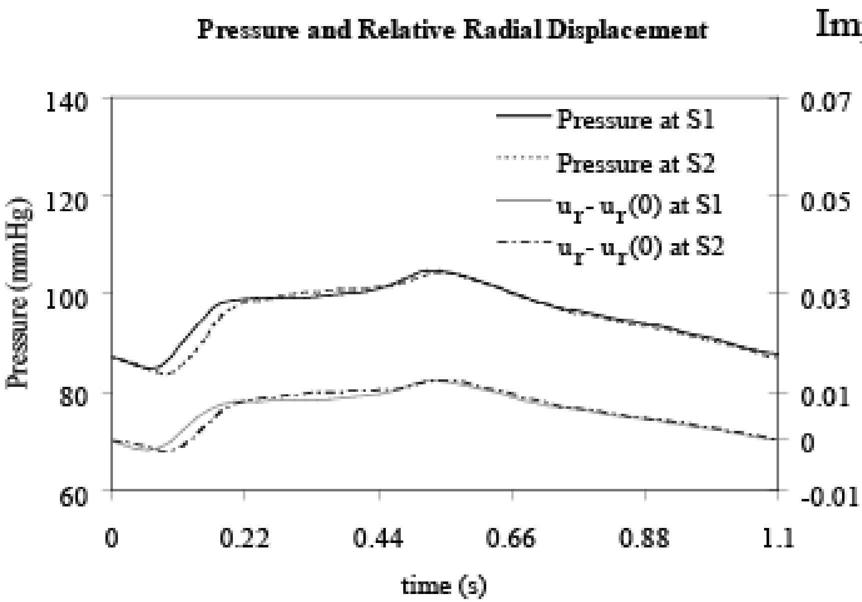
- 1D – impedance $p(t)=\dfrac{1}{T}\int_{t-T}^{t} Z(t-\tau)Q(\tau)\,d\tau$

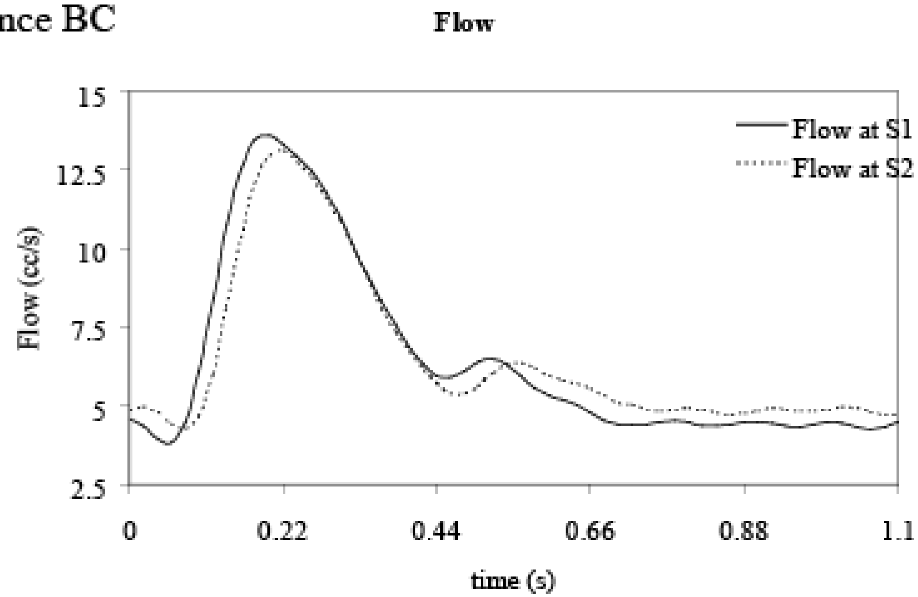
$$\left[\underset{\sim}{M}_m(\vec{v},p)+\underset{\sim}{H}_m\right]_{\Gamma_B} = \left(-\frac{1}{T}\int_{t-T}^{t} Z(t-\tau)\int_{\Gamma_B}\vec{v}(\tau)\cdot\vec{n}\,d\Gamma\,d\tau\;\underset{\sim}{I} + \underset{\sim}{\tau} - \rho\vec{v}\otimes\vec{v}\right)\Big|_{\Gamma_B}$$

$$\left[\vec{M}_c(\vec{v},p)+\vec{H}_c\right]_{\Gamma_B} = \vec{v}\Big|_{\Gamma_B}$$

Multidomain impedance boundary condition

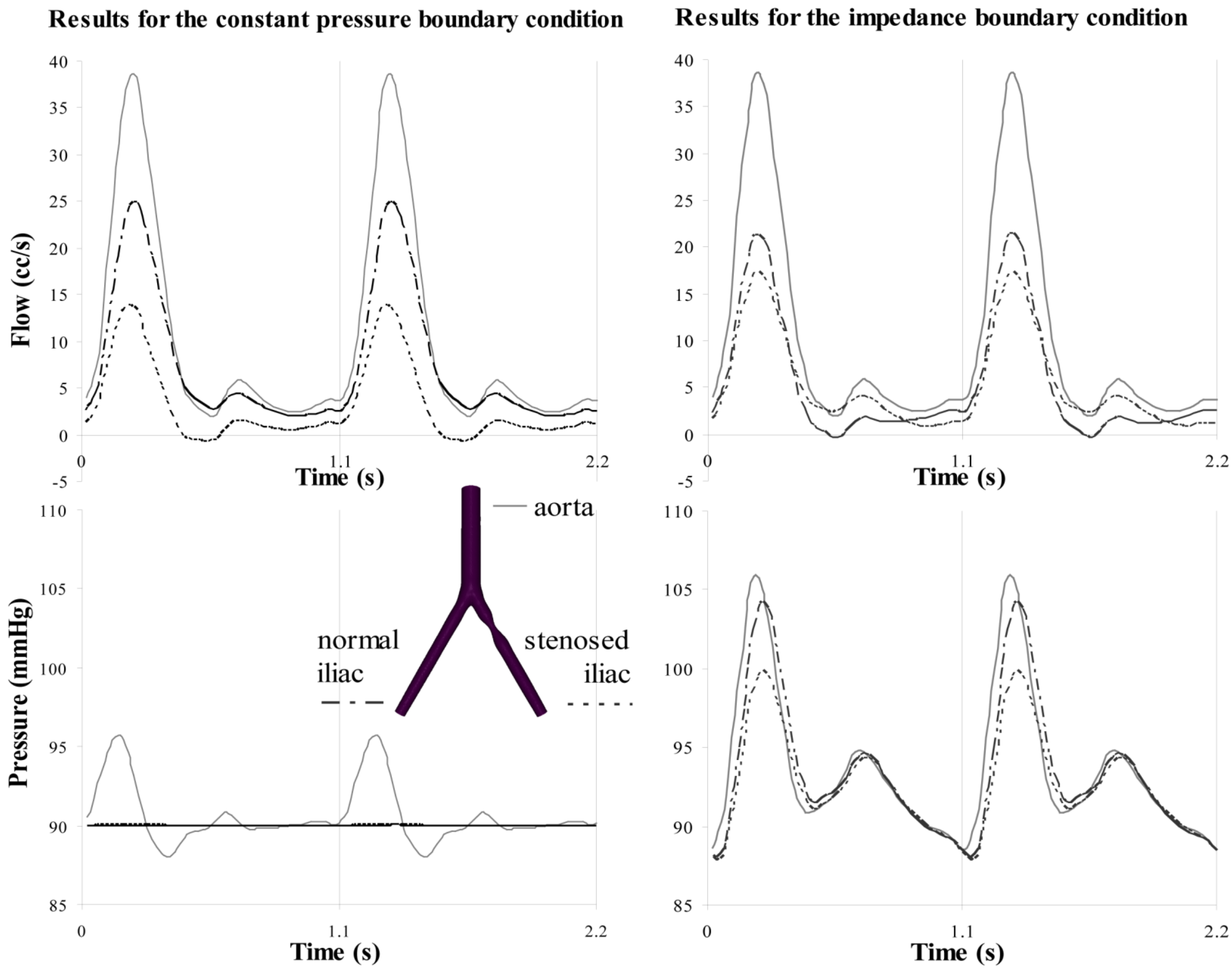
Impedance BC





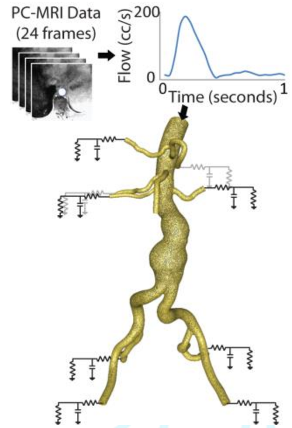
Boundary conditions represent the portions of the body not included in the numerical domain.

Example: Effect of boundary condition

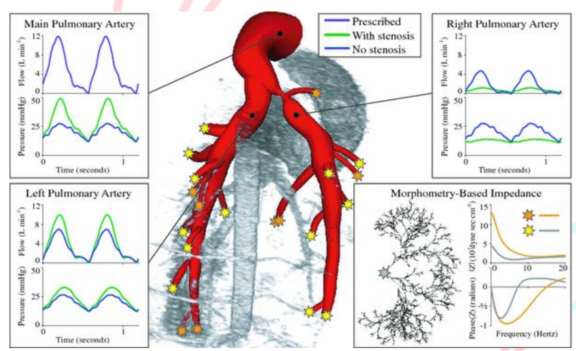


Towards more realistic boundary conditions

1. Lumped models



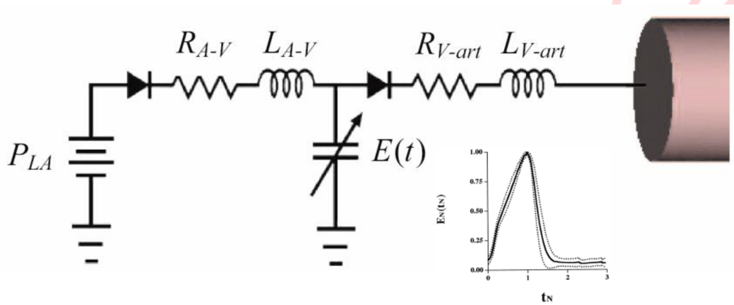
2. Impedance constructed from 1-D linear wave theory with data varying with organ / patient specific / state

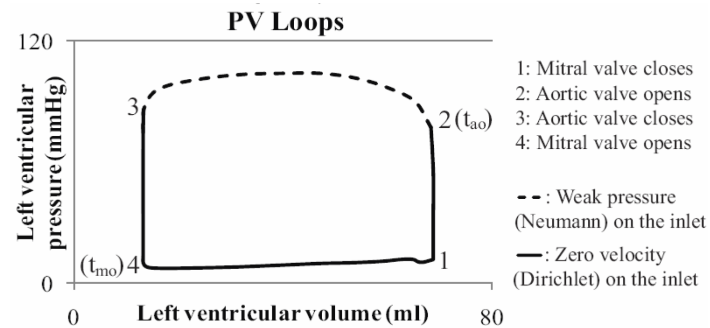


3. Closed loop models with feedback control

Lumped Parameter Heart Model

- Aortic pressure and flow result from the interactions between the left ventricle and the arterial system.
- The Coupled Multidomain Method is used to couple a lumped parameter heart model to the inlet of a three-dimensional finite element aortic model.
- To represent the left or right ventricular pressure, normalized elastance function is used after scaling the function to match subject-specific cardiac output and pulse pressure.





P H. Senzaki et al., Single-beat estimation of end-systolic pressure-volume relation in humans. A new method with the potential for noninvasive application, Circulation, 94 (10) (1996) 2497-506.

H.J. Kim, I. E. Vignon-Clementel, C.A. Figueroa, J.F. LaDisa, K.E. Jansen, J.A. Feinstein, C.A. Taylor (2009) On Coupling a Lumped Parameter Heart Model and a Three-dimensional Finite Element Aorta Model. Submitted to Annals of Biomedical Engineering.

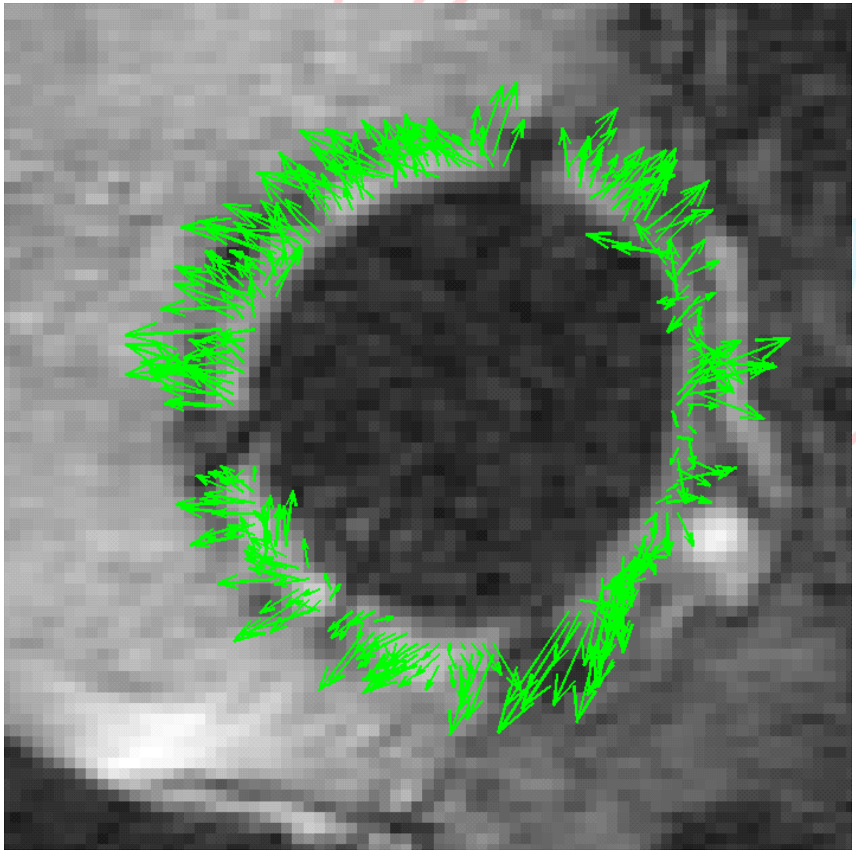
Coupled Flow-Vessel Motion

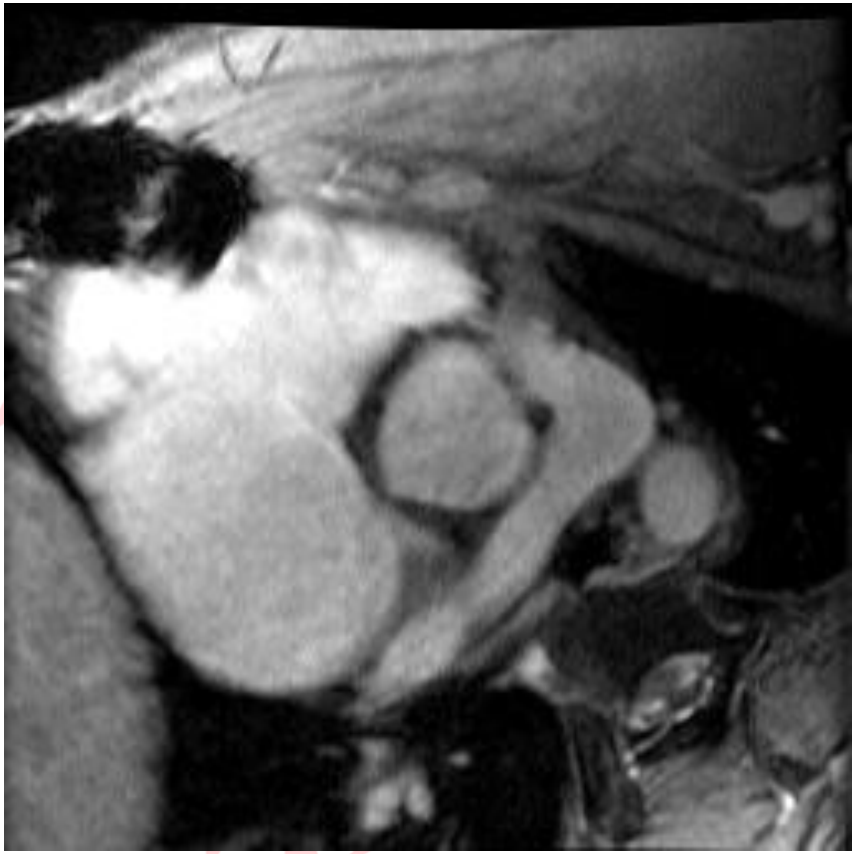
- Blood Flow and Pressure depend dramatically on the elasticity of the arteries.

- If the deformability of the vessel wall is neglected and an incompressible constitutive model is adopted for the blood (both assumptions are fairly common when simulating blood flow), then **wave propagation is completely absent** from the model.

- Stress and strain in arteries are greatly influenced by the blood flow velocity and pressure fields.

Coupled Flow-Vessel Motion



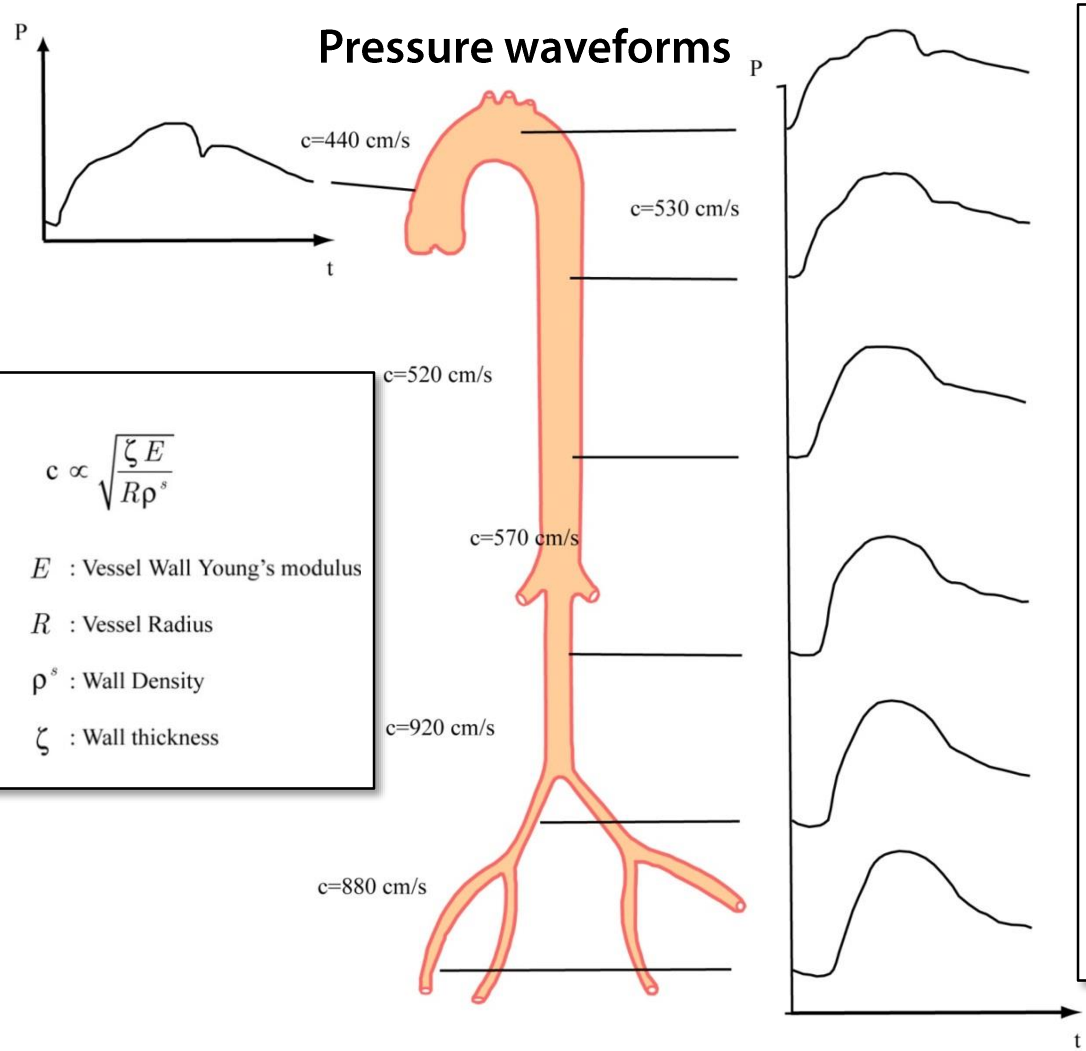


© C. Alberto Figueroa – figueroc@med.umich.edu

Blood vessels are not rigid!

Hemodynamics and Arterial Stiffness

Pressure waveforms



Latham et al. *Circulation*. 1985

Pulse wave velocity (PWV or c) depends on:

vessel geometry, vessel stiffness

$$c \propto \sqrt{\frac{\zeta E}{R \rho^s}}$$

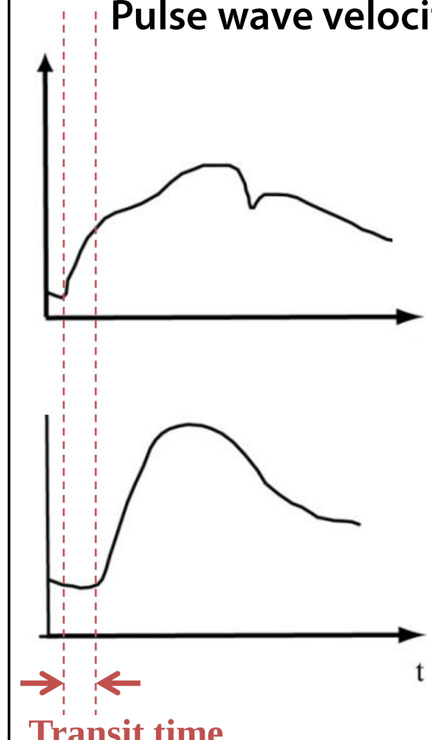
E : Vessel Wall Young's modulus

R : Vessel Radius

ρ^s : Wall Density

ζ : Wall thickness

Pulse wave velocity



$$c = \frac{\text{distance}}{\text{transit time}}$$

Hemodynamics and Arterial Stiffness

Increased Arterial Stiffness

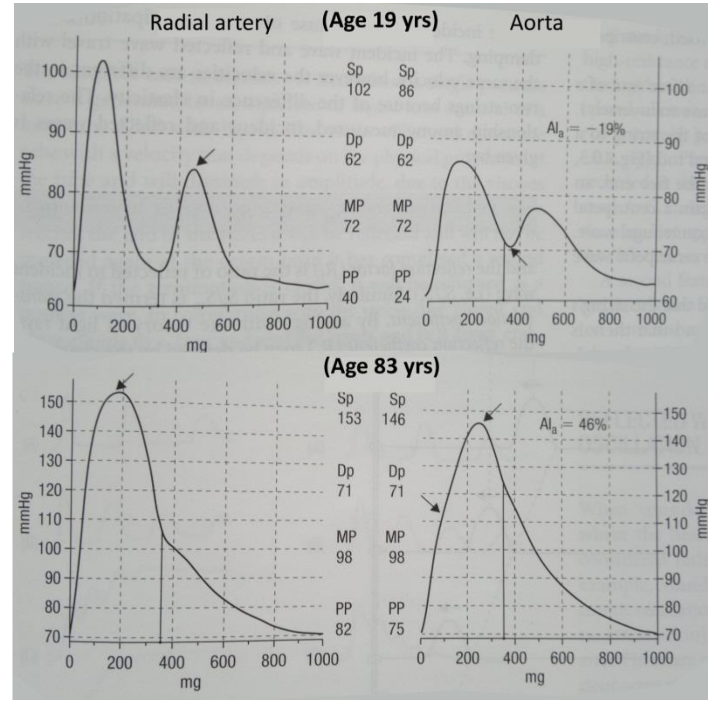
Elastin ↓

Collagen ↑

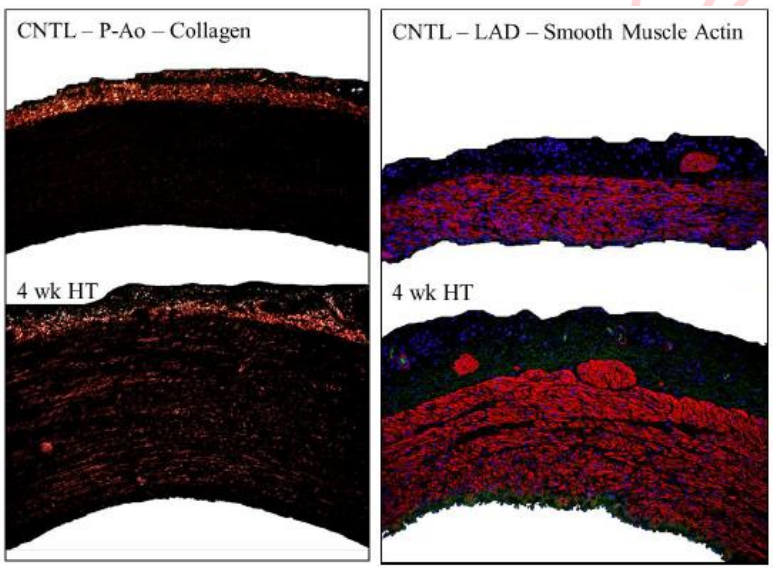
ECM structural organization ↓

SMC, endothelial cell function ↓

Nichols & Singh, Curr Opin Cardiol 2002



Hayenga et al. 2012



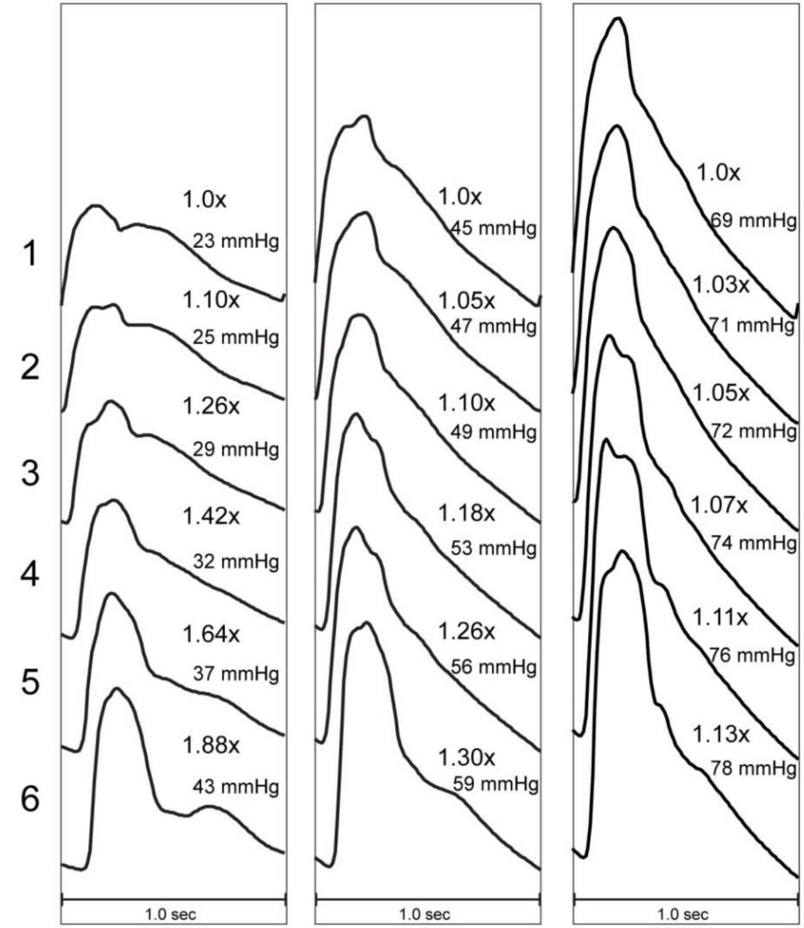
Adverse Arterial Hemodynamics

- ↑ pulse wave velocity (PWV)
- ↑ augmentation index (AIx)
- ↑ central pulse pressure (cPP)
- ↑ flow pulsatility

Arterial Stiffness, Pulse Pressure, and PWV in Humans

PWV (m/s)

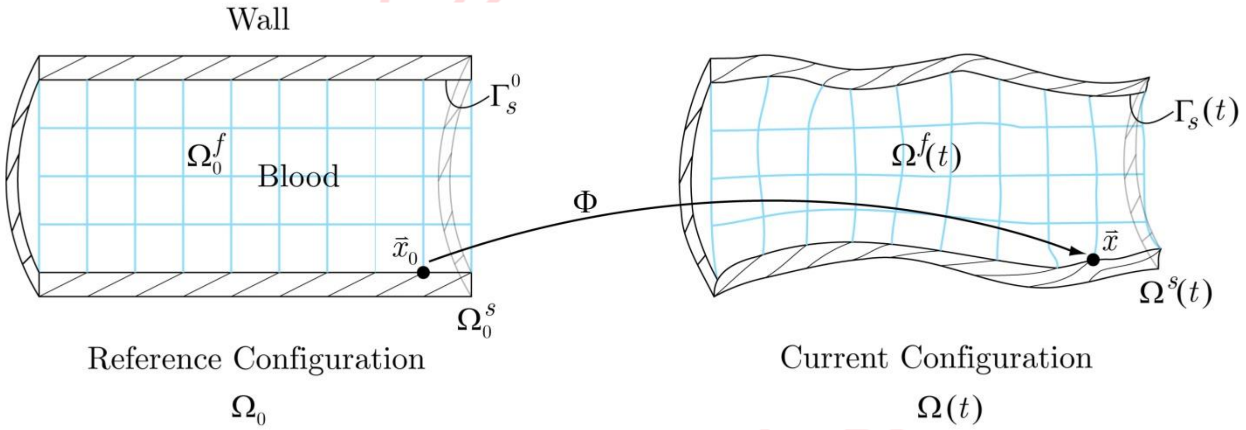
Interval	Young	Middle-Aged	Elderly
1 to 2	3.01	3.36	3.57
2 to 3	3.43	5.61	6.15
3 to 4	3.52	5.32	8.11
4 to 5	4.30	6.38	8.00
5 to 6	3.56	5.06	6.24
Aortic to Iliac (1 to 6)	3.74	5.08	6.19



Demonstrated changes in pressure amplification and pulse pressure due to stiffening (Xiao et al., 2013)

How to model these fluid-structure interactions?

The **Arbitrary Lagrangian-Eulerian (ALE)** formulation couples the solid mechanics problem (in Lagrangian frame) with the fluid mechanics problem (in a mixed Eulerian-Lagrangian frame)



Definition of grid velocity for the fluid mesh:

$$\vec{v}_G \equiv \left(\partial \vec{x}/\partial t\right)\Big|_{\vec{x}_0}$$

Motion of the "interior" of the fluid mesh is given by an arbitrary mapping Φ that matches the structure motion at the interface $\Gamma_s(t)$

$$\Phi : \Omega_0 \times I \rightarrow \Omega(t)$$
$$(\vec{x}_0, t) \rightarrow \vec{x} = \Phi(\vec{x}_0, t)$$
$$\Phi(\vec{x}_0, t) \in \Gamma_s(t) \quad \forall \vec{x}_0 \in \Gamma_0^s$$

Navier-Stokes equations in a moving domain:

$$\nabla_{\vec{x}} \cdot \vec{v} = 0$$

$$\text{in } \Omega^f(t)$$

$$\rho \frac{\partial \vec{v}}{\partial t}\bigg|_{\vec{x}_0} + \nabla_{\vec{x}} \cdot \left(\rho \vec{v} \otimes (\vec{v} - \vec{v}_G)\right) = \nabla_{\vec{x}} \cdot (\underset{\sim}{\sigma}) + \vec{f}$$

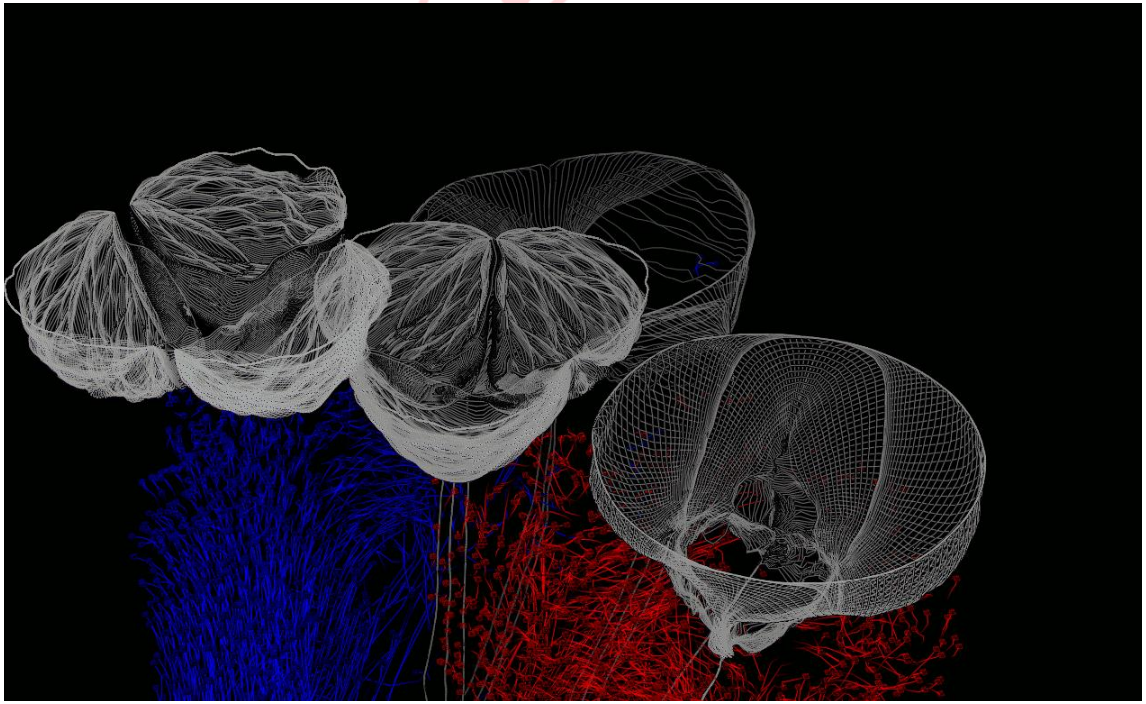
Elastodynamics equations in Lagrangian frame:

$$\rho_0 \frac{\partial \vec{v}}{\partial t} + \nabla_0 \cdot \left(\underset{\sim}{\sigma} F^{-T}\right) = \vec{f}_0 \qquad \text{in } \Omega_0^s$$

Hughes et al., Comp Meth Appl Mech Eng, 1981

How to model these fluid-structure interactions?

The **Immersed Boundary Domain Method (IBM)**



Griffith, McQueen, Peskin.

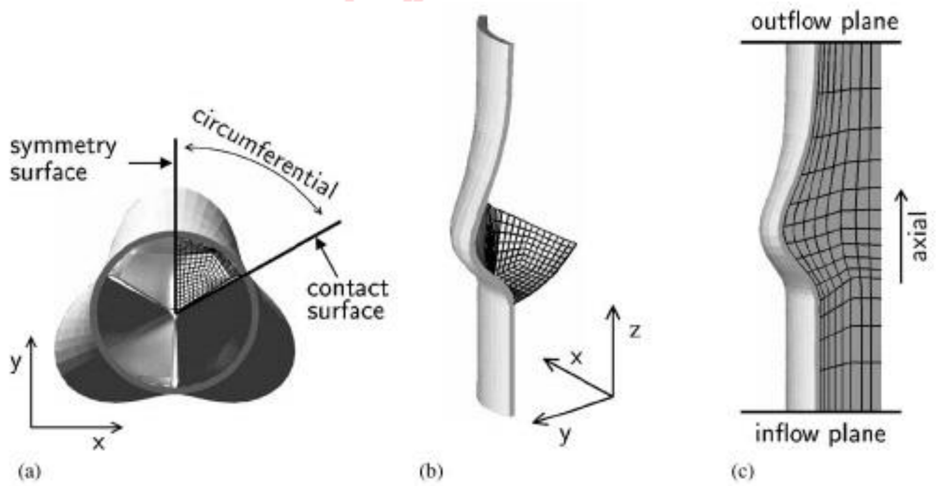
- Developed by C. Peskin, it uses a purely Eulerian description for the fluid, and introduces a fictitious body force that drives the motion of a thin structure embedded in the fluid.
- Lack of contact mechanics formulation.
- Used on Cartesian grids.

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

$$\vec{f} = \int_{\Omega} \vec{F}\,\delta\left(\vec{x} - \vec{X}\right) d\Omega$$

How to model these fluid-structure interactions?

The **Fictitious Domain Method (FDM)**



De Hart, Peters, Schreurs, Baaijens, 2004

- Developed by Glowinski.
- Closely related to IBM, but developed in a finite-element context, introducing Lagrange multipliers to constrain the motion of the fluid an the solid at the interface.
- Baaijens developed an extension of the method suitable for slender structures and applied to aortic valve simulations.
- Has been used in combination with ALE formulations

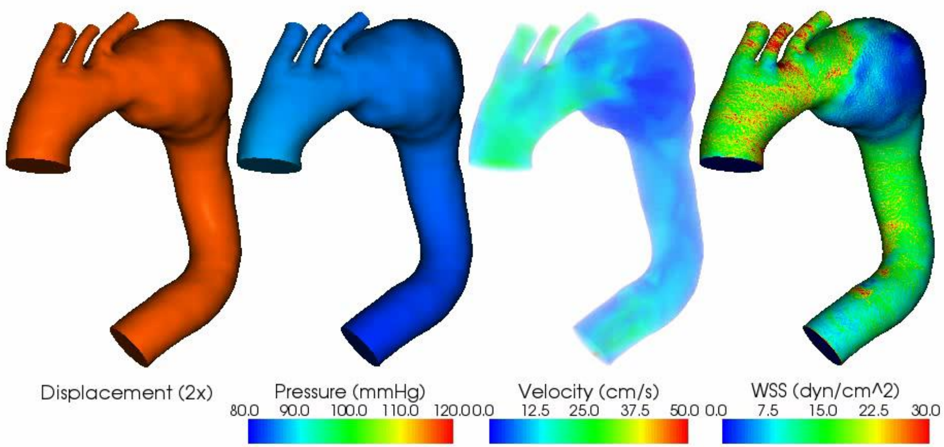
How to model these fluid-structure interactions?

The **Coupled Momentum Method**

$$
\begin{aligned}
B(\vec{w}, q; \vec{v}, p) = \int_{\Omega} \left\{ \vec{w} \cdot \left(\rho \vec{v}_{,t} + \rho \vec{v} \cdot \nabla \vec{v} - \vec{f} \right) + \nabla \vec{w} : \left(-p \underset{\sim}{I} + \underset{\sim}{\tau} \right) - \nabla q \cdot \vec{v} \right\} d\vec{x} \\
- \int_{\Gamma_h} \vec{w} \cdot \vec{h} ds + \int_{\Gamma_h} q v_n ds + \int_{\Gamma_g} q v_n ds + \text{stabilization terms} \\
+ \zeta \int_{\Gamma_s} \left\{ \vec{w} \cdot \rho^s \vec{v}_{,t} + \nabla \vec{w} : \underset{\sim}{\sigma}^s (\vec{u}) \right\} ds - \zeta \int_{\partial \Gamma_s} \vec{w} \cdot \vec{h}^s dl + \int_{\Gamma_s} q v_n ds
\end{aligned}
$$

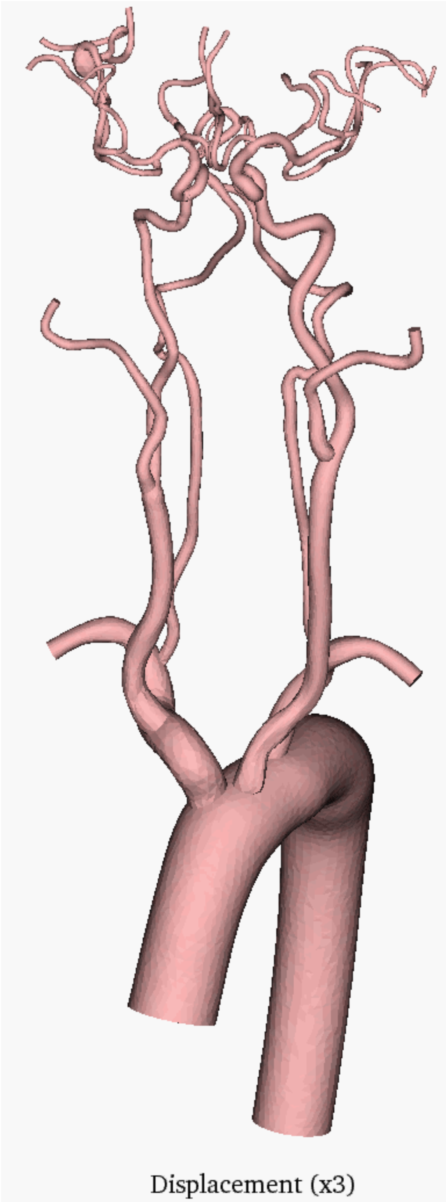
Figueroa, Vignon-Clementel, Jansen, Hughes & Taylor CMAME 2006

Blood flow simulation in a thoracic aneurysm model

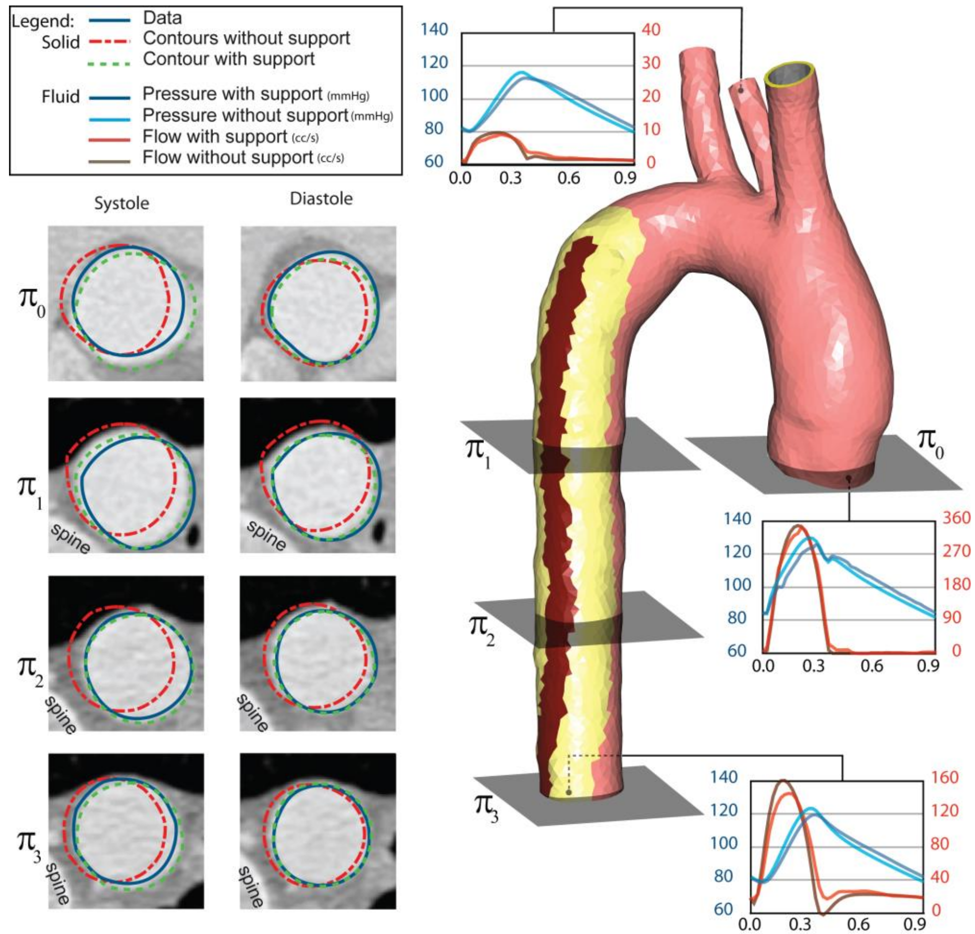


Xiong, Figueroa, Xiao & Taylor IJNMBE 2010

A Model for External Tissue Support



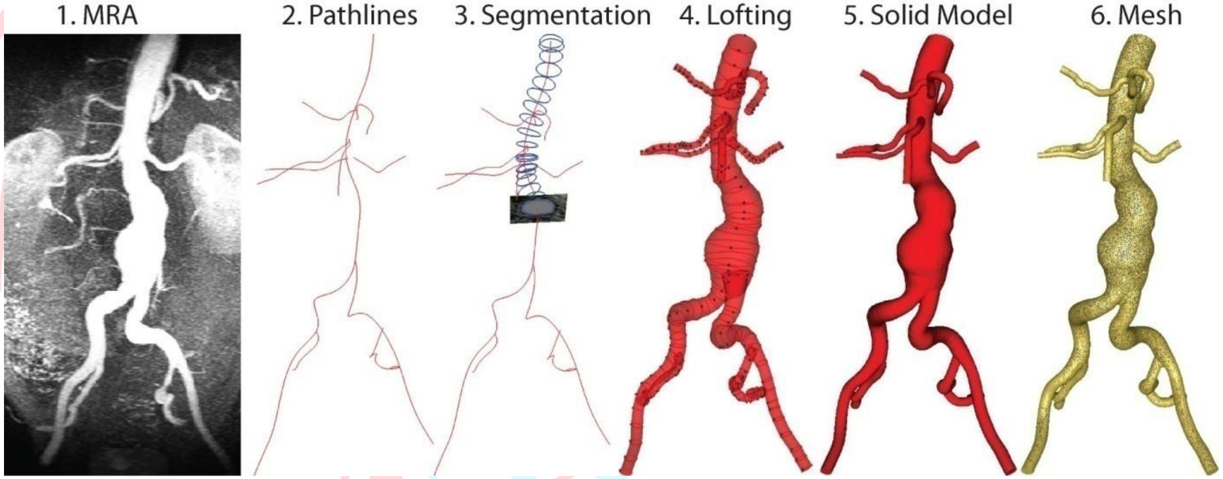
$$\boldsymbol{\sigma}_s \cdot \boldsymbol{n} = -k_s(\boldsymbol{d}_s - \boldsymbol{d}_b) - c_s(\boldsymbol{u}_s - \boldsymbol{u}_b)$$



Moireau, Xiao, Astorino, Figueroa, Chapelle, Taylor & Gerbeau, BMMB 2012

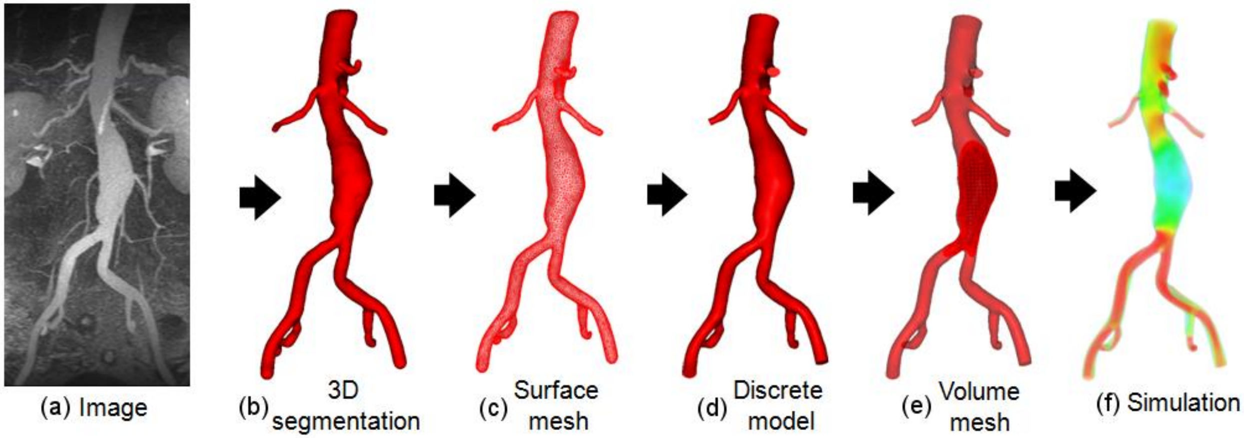
2D / 3D Geometric modeling techniques

2D-based modeling



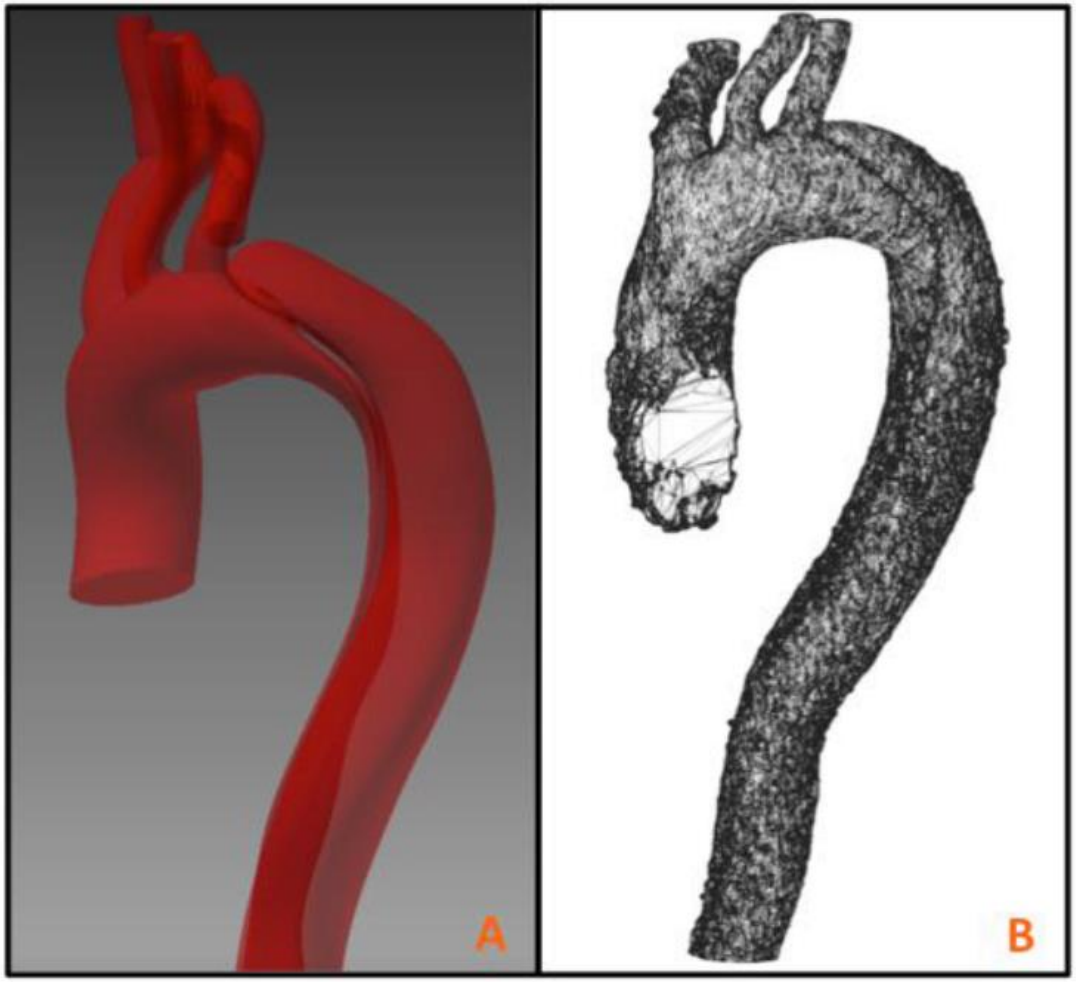
Les, Shadden, Figueroa et al., ABME 2010

3D-based modeling

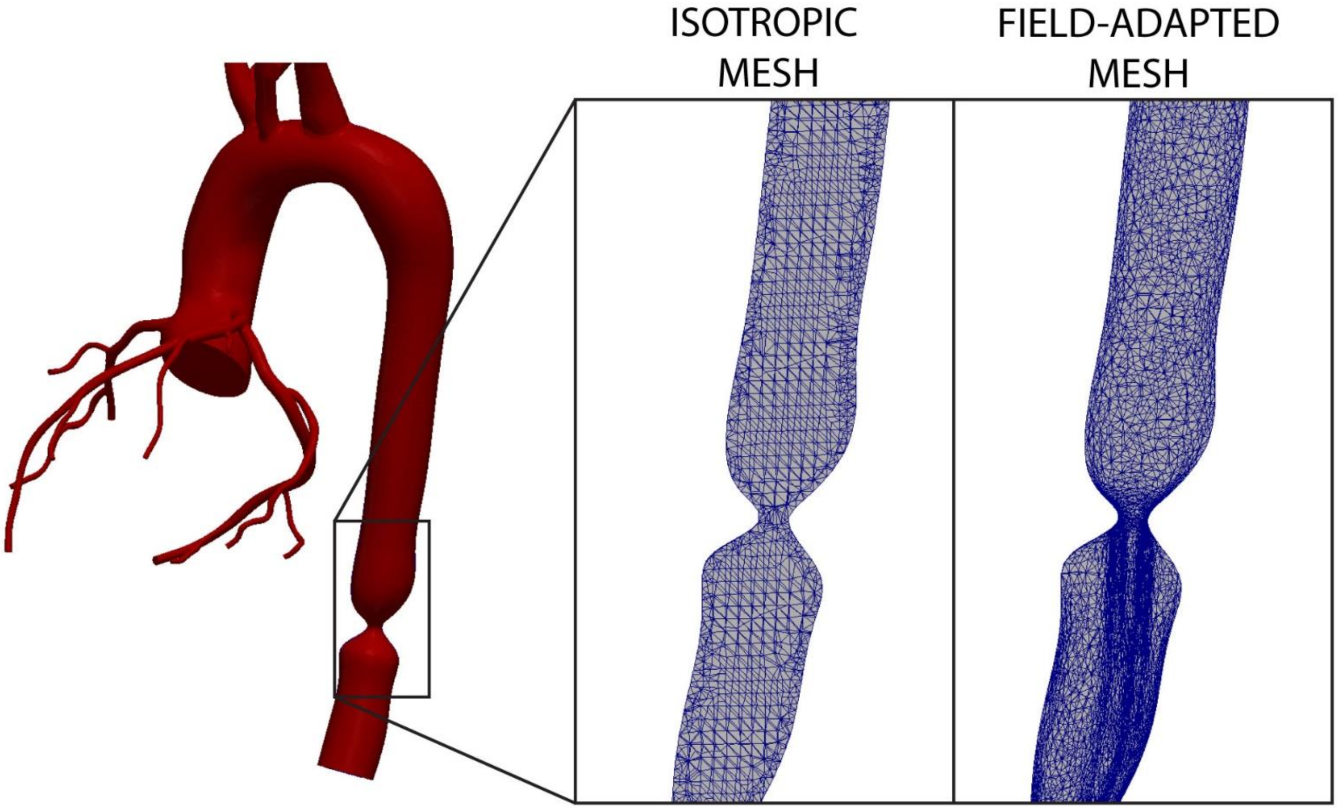


Xiong, Figueroa, Xiao & Taylor, IJNMBE 2010

Discrete vs Analytical (NURBS) Geometry Definitions

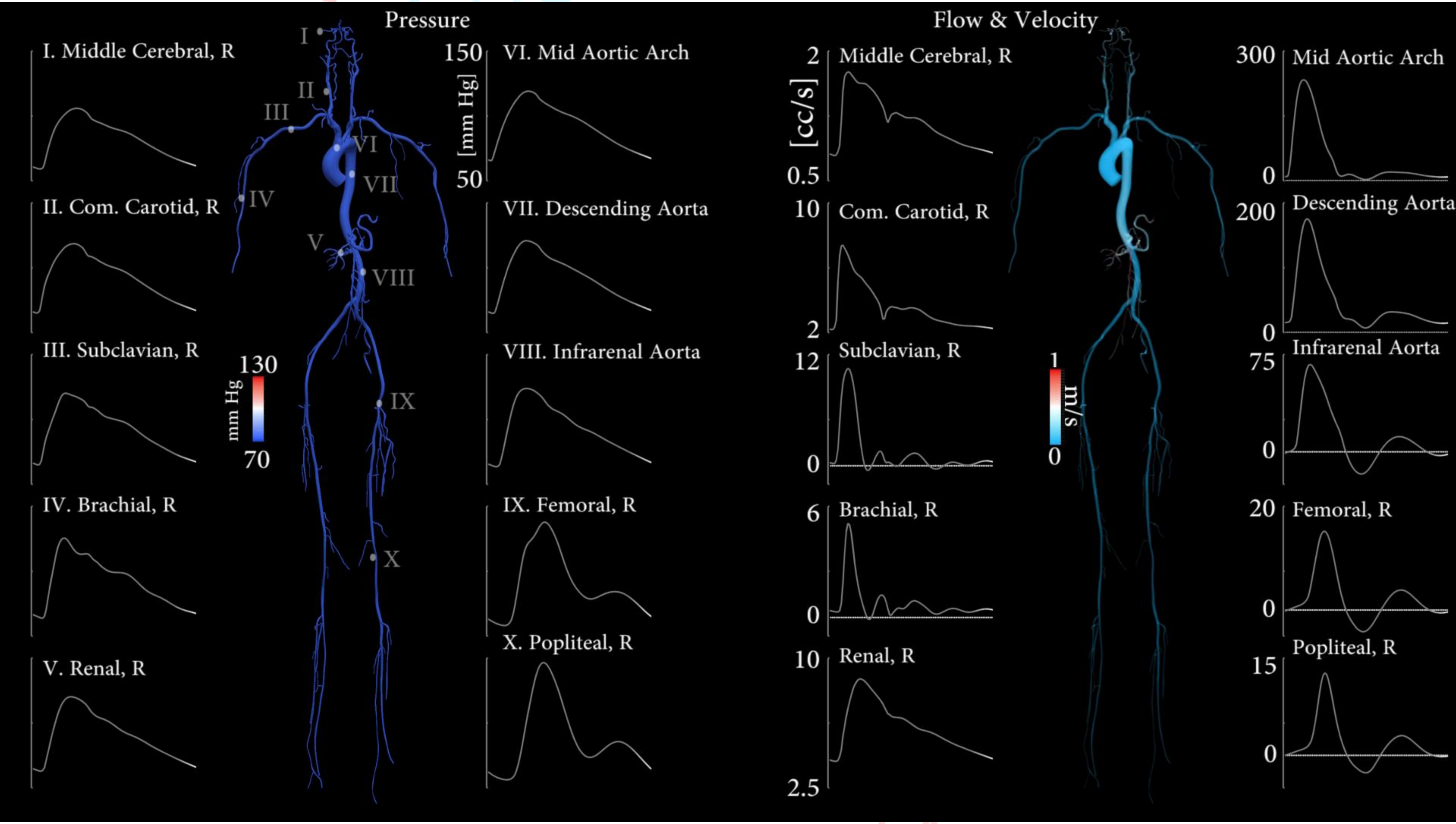


Mesh adaptation



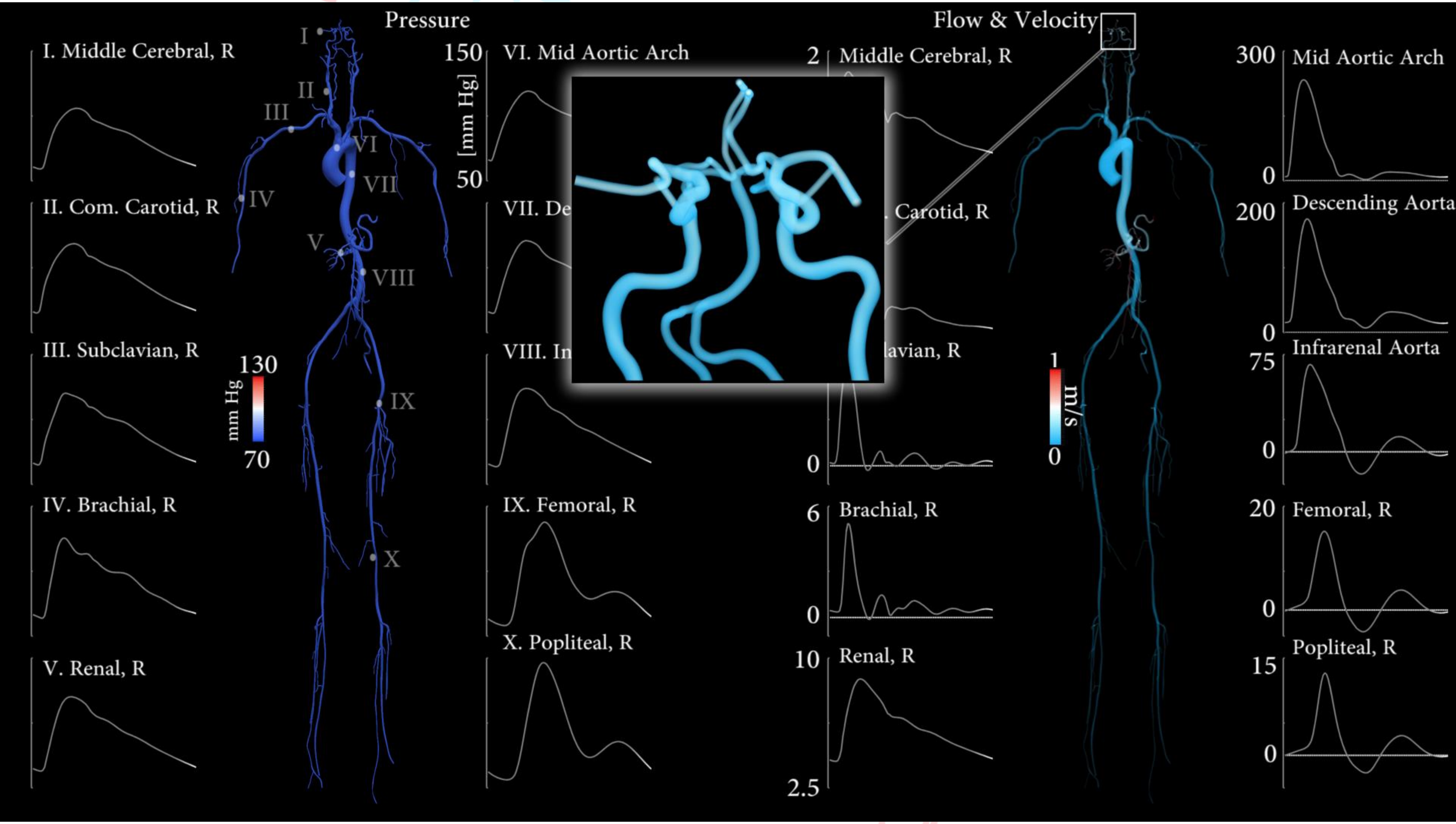
Sahni, et al, CMAME 2006

Hemodynamics in Full-Body Scale Models



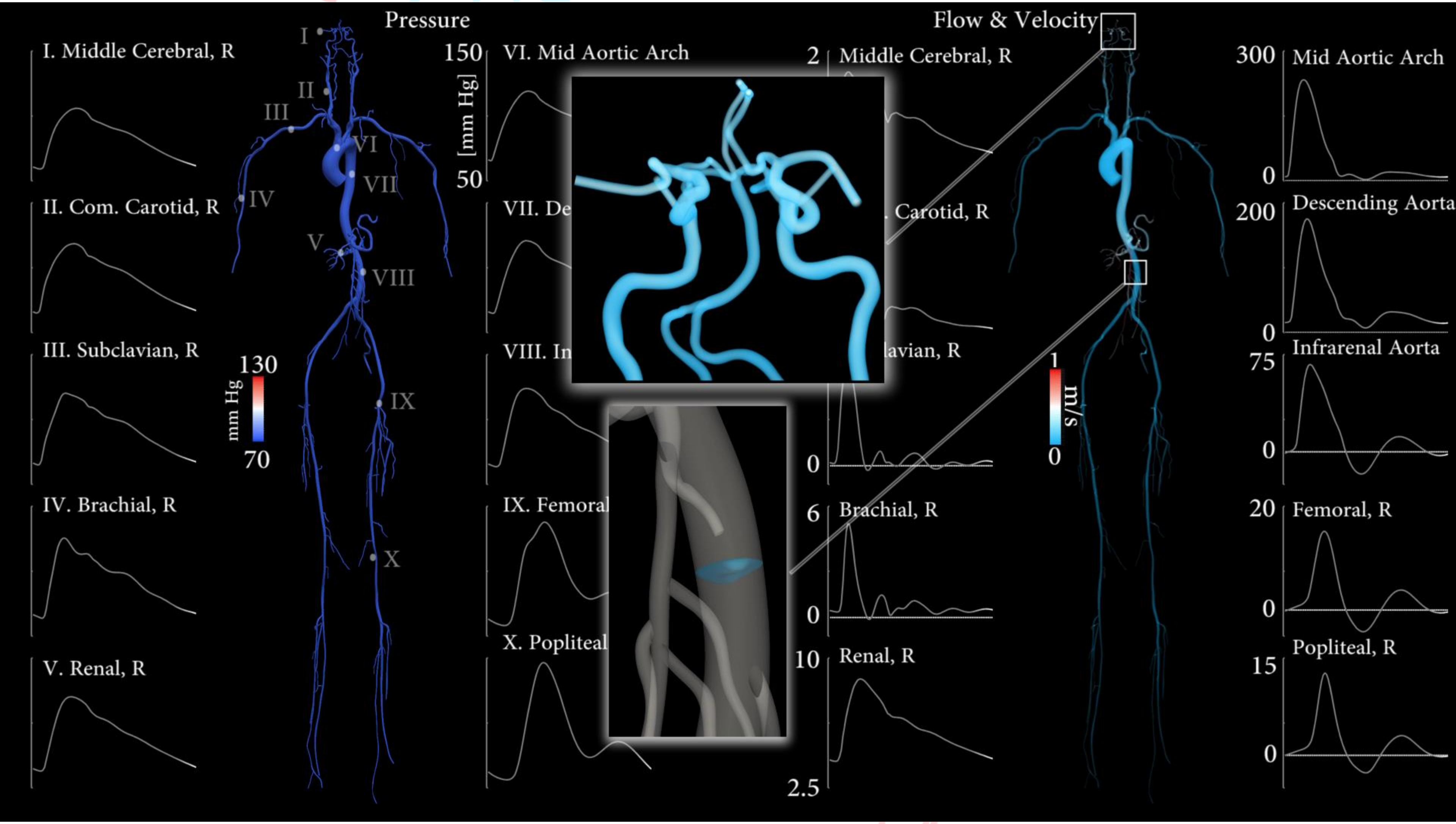
Xiao, Humphrey, Figueroa, JCP, 2013

Hemodynamics in Full-Body Scale Models



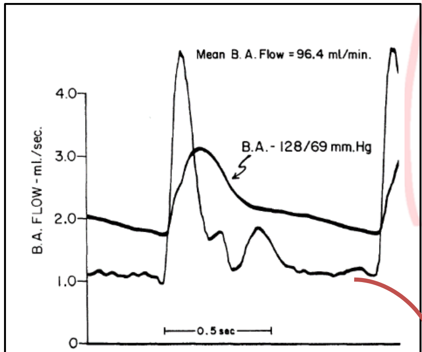
Xiao, Humphrey, Figueroa, JCP, 2013

Hemodynamics in Full-Body Scale Models

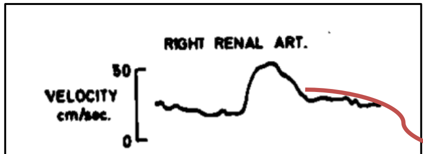


Xiao, Humphrey, Figueroa, JCP, 2013

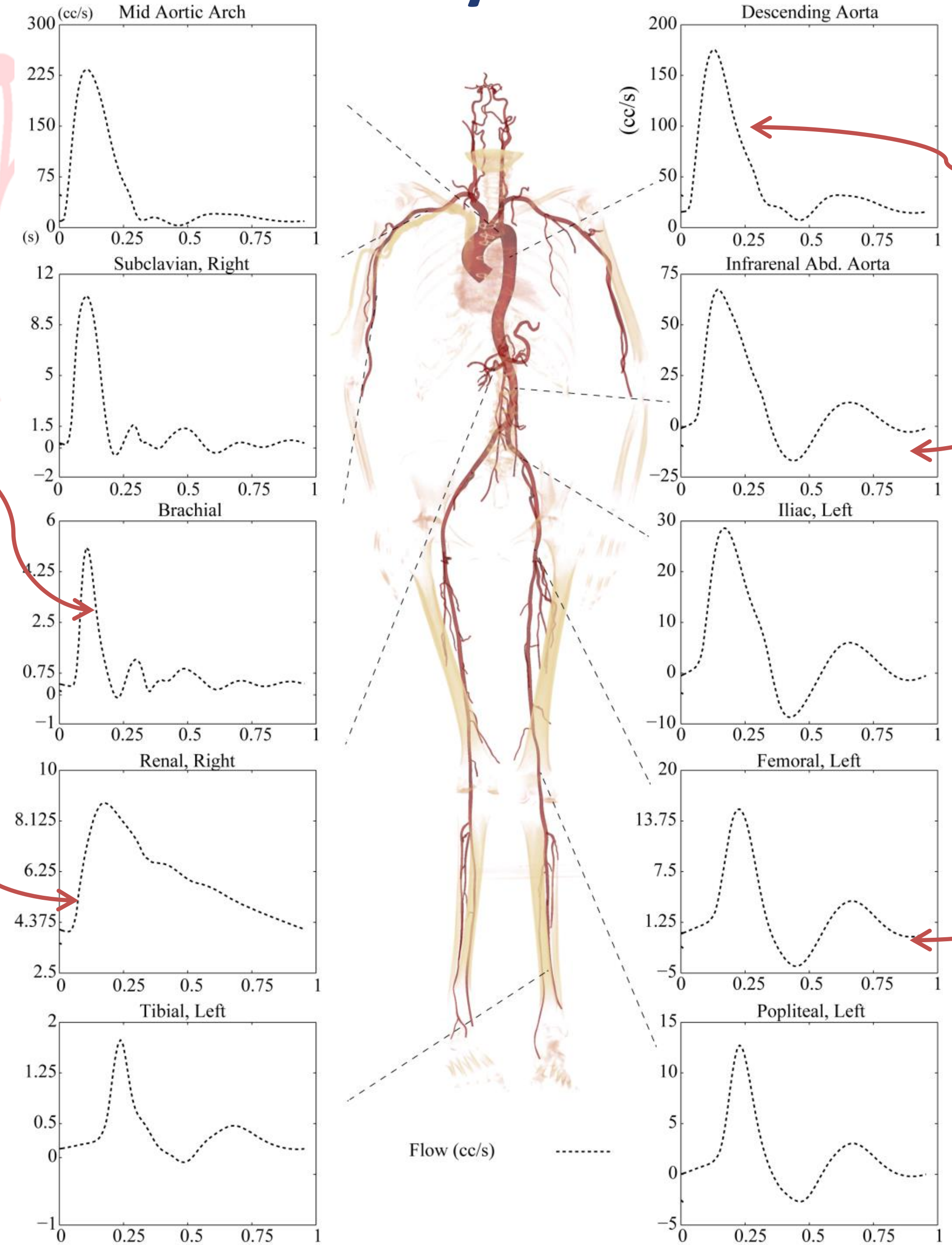
Hemodynamics in Full-Body Scale Models

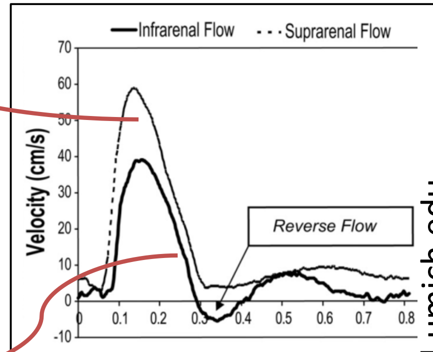


Brachial artery flow
Gault et al, Circulation, 1966



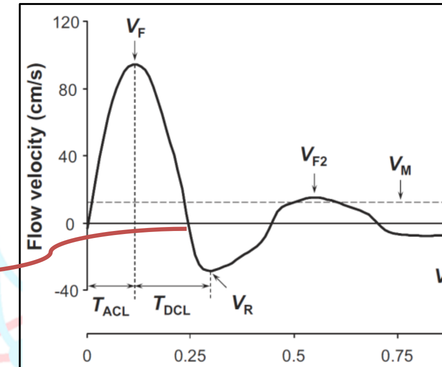
Renal artery velocity
Mills et al, Cardiovas Res, 1970





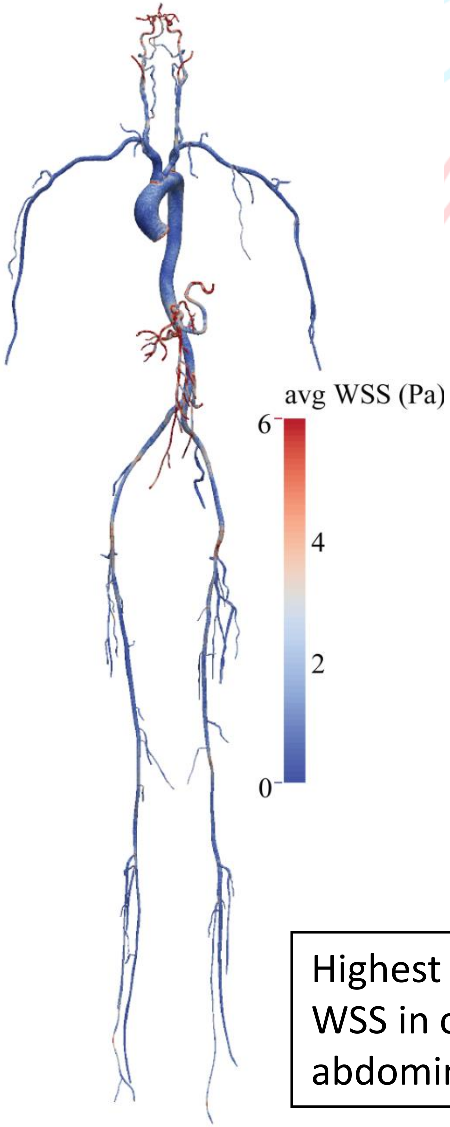
Suprarenal, infrarenal velocity
Amirbekian et al, Heart Circ Phys, 2010

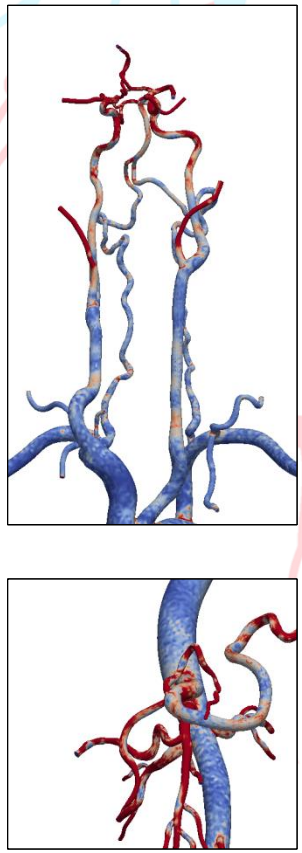
– figueroc@med.umich.edu



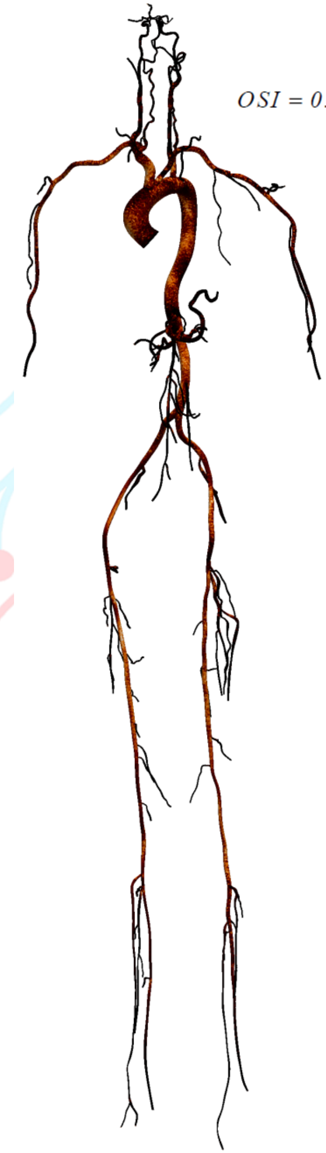
Femoral artery velocity
Hashimoto et al, Hypertension, 2010

Hemodynamics in Full-Body Scale Models

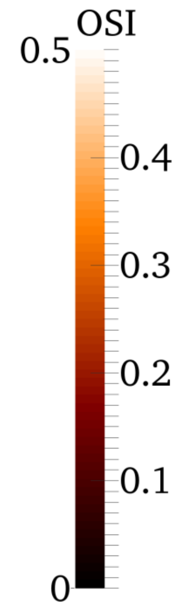


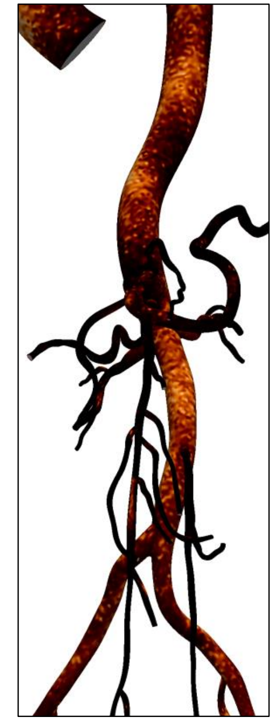


Highest time-averaged WSS in cerebral arteries, abdominal branches



$$OSI = 0.5 \times \left(1.0 - \frac{\left| \int_0^T \vec{WSS}\, dt \right|}{\int_0^T \left| \vec{WSS} \right| dt} \right)$$





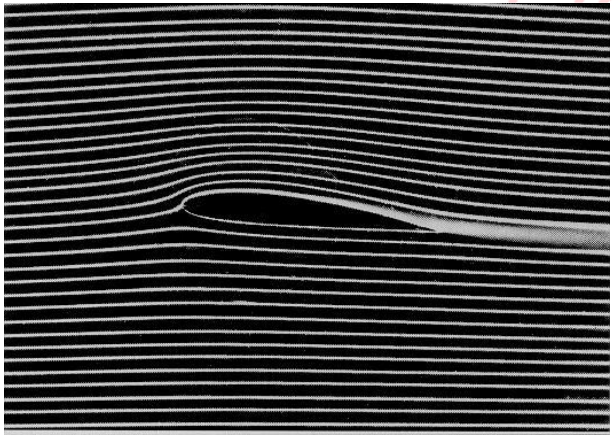
Uniformly high OSI in infrarenal abdominal aorta

The issue of turbulence

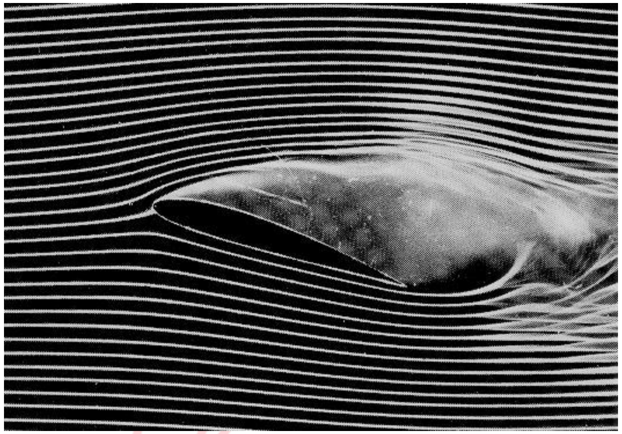
A *laminar* flow is a flow in which fluid particles move in smooth layers, or laminae, sliding over adjacent layers without mixing.

A *turbulent* flow is characterized by irregular, erratic intermingling of fluid particles.

The transition from a laminar to turbulent flow is dependent upon a non-dimensional parameter known as the Reynold's number, *Re*.



Laminar flow



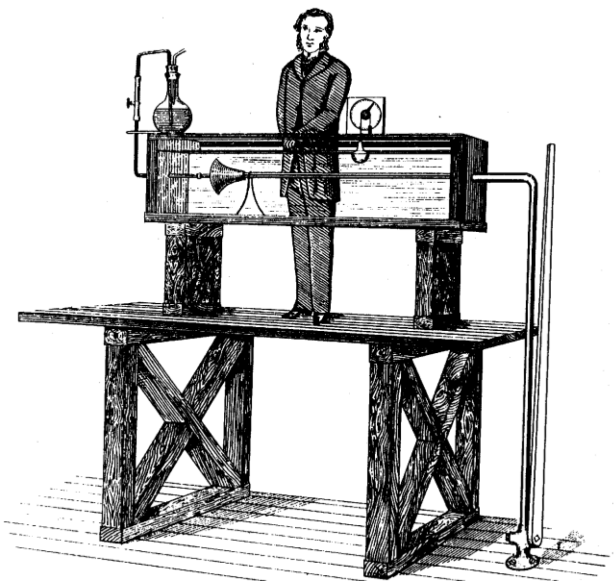
Turbulent flow

http://www.tn.utwente.nl/wsl/research/turbulence/wing.html

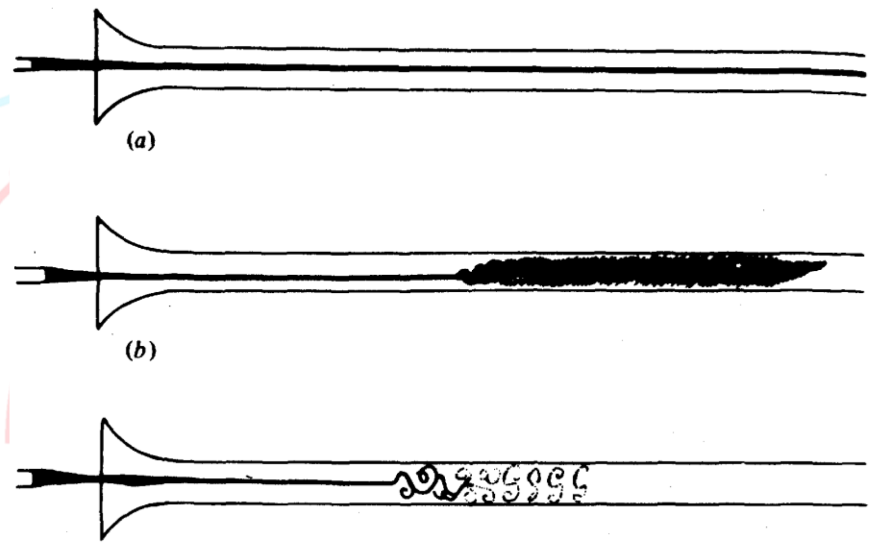
The issue of turbulence

For steady flow in a pipe, $\mathrm{Re} = \dfrac{\bar{v}\rho d}{\mu}$

where $\bar{v}$ is mean velocity, and *d* is diameter. Critical threshold is between 2000 and 3000 for most cases of pipe flow.



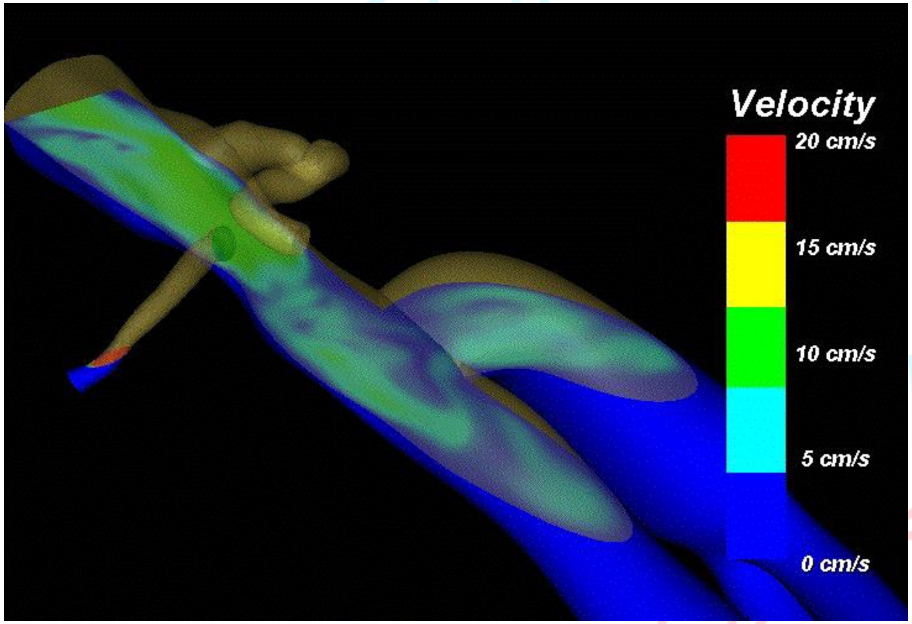
Osborne Reynolds and his experiment releasing dye in steady flow in circular tube



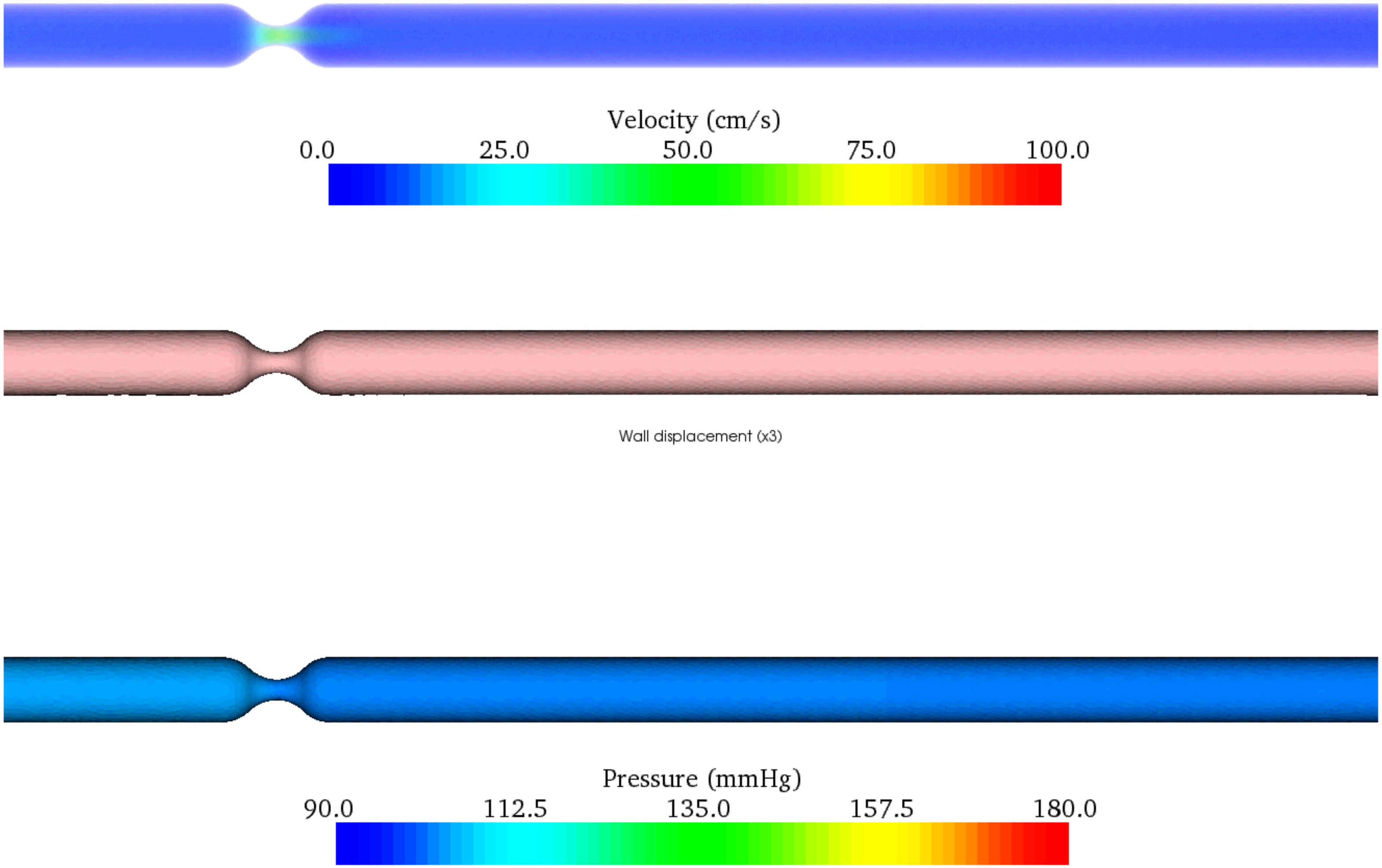
Osborne Reynolds' sketches of from his experiment showing laminar flow then transition to turbulence. (a) laminar flow, (b) transition to turbulence induced by motion of water in surrounding tank, (c) transition to turbulence induced by increased flow through tube w/ stationary fluid in exterior tank.

The issue of turbulence

In general, we approximate blood by an incompressible, homogeneous viscous fluid. In large arteries a Newtonian approximation is reasonable. In most cases, for healthy vessels, blood flow is laminar (or transitional). Not necessarily true for diseased vessels or increased flow rates (e.g. during exercise).

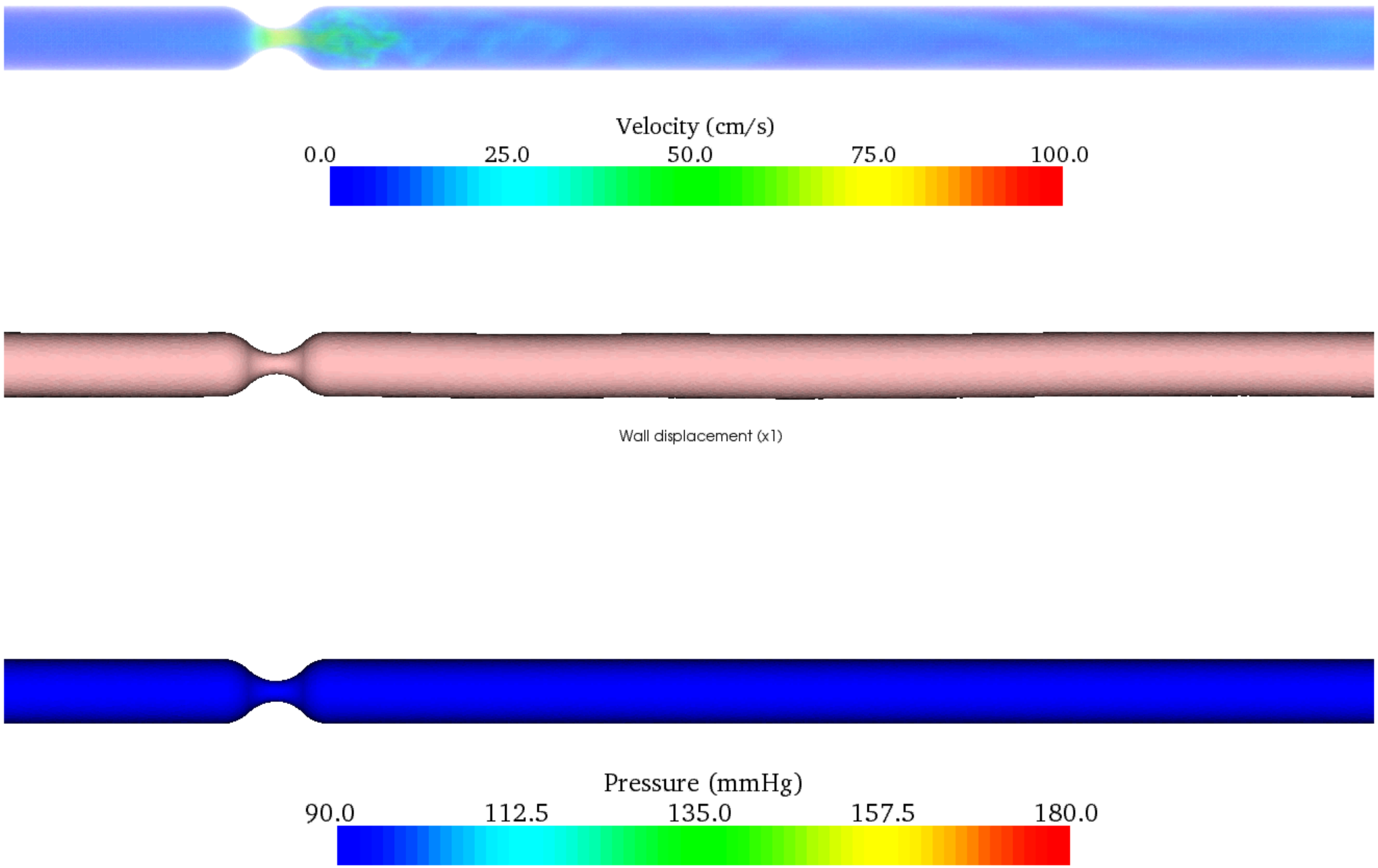


The issue of turbulence



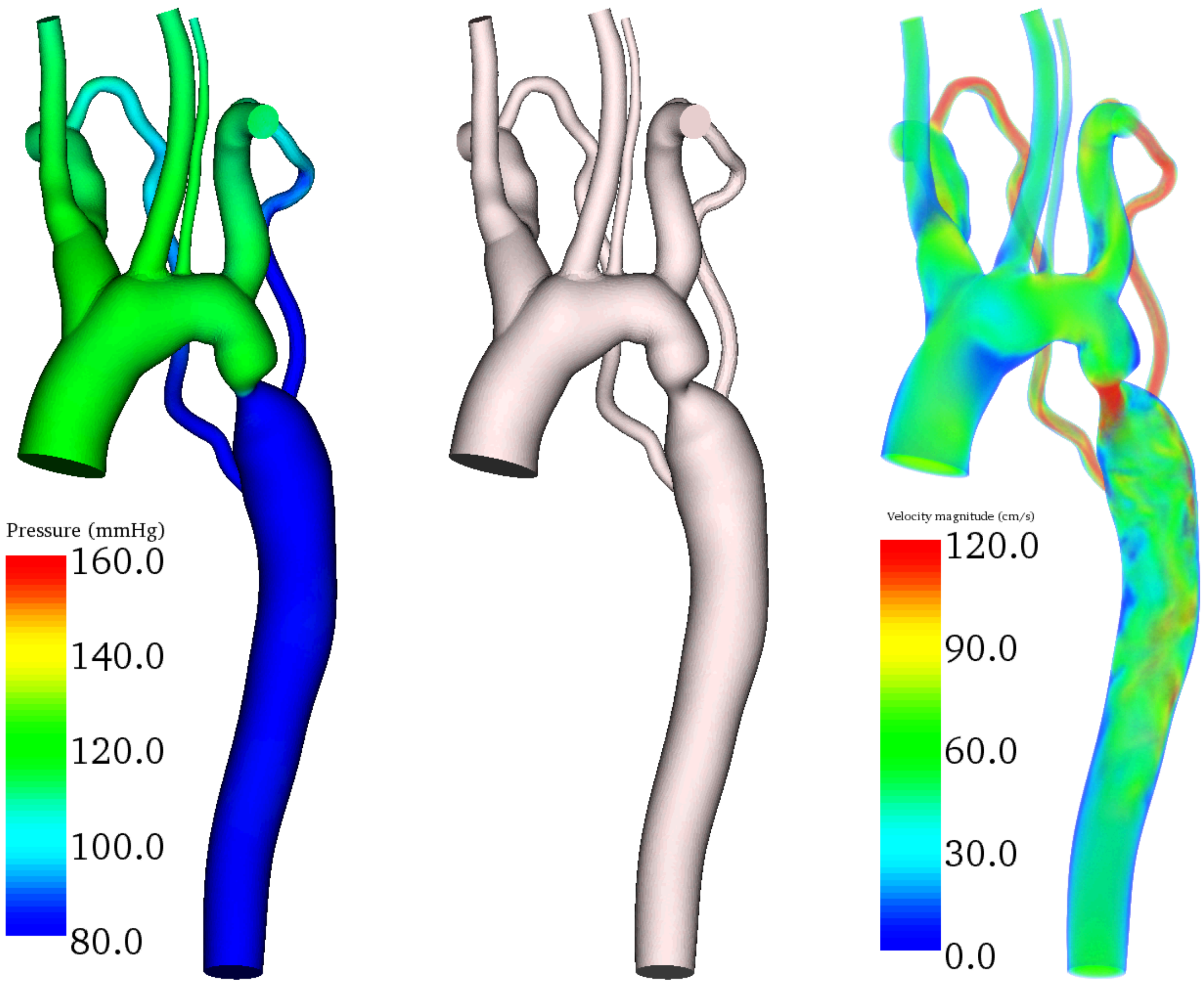
Velocity magnitude, wall displacement, and pressure in rat with aortic coarctation during resting conditions. Laminar or turbulent?

The issue of turbulence



Velocity magnitude, wall displacement, and pressure in rat with aortic coarctation during resting conditions. Laminar or turbulent?

The issue of turbulence



Pressure, wall displacement, velocity magnitude in patient with aortic coarctation during resting conditions. Laminar or turbulent?

Turbulent Kinetic Energy

$$TKE = \frac{3}{2}\rho\langle\tilde{u}(x,t)\rangle^2$$

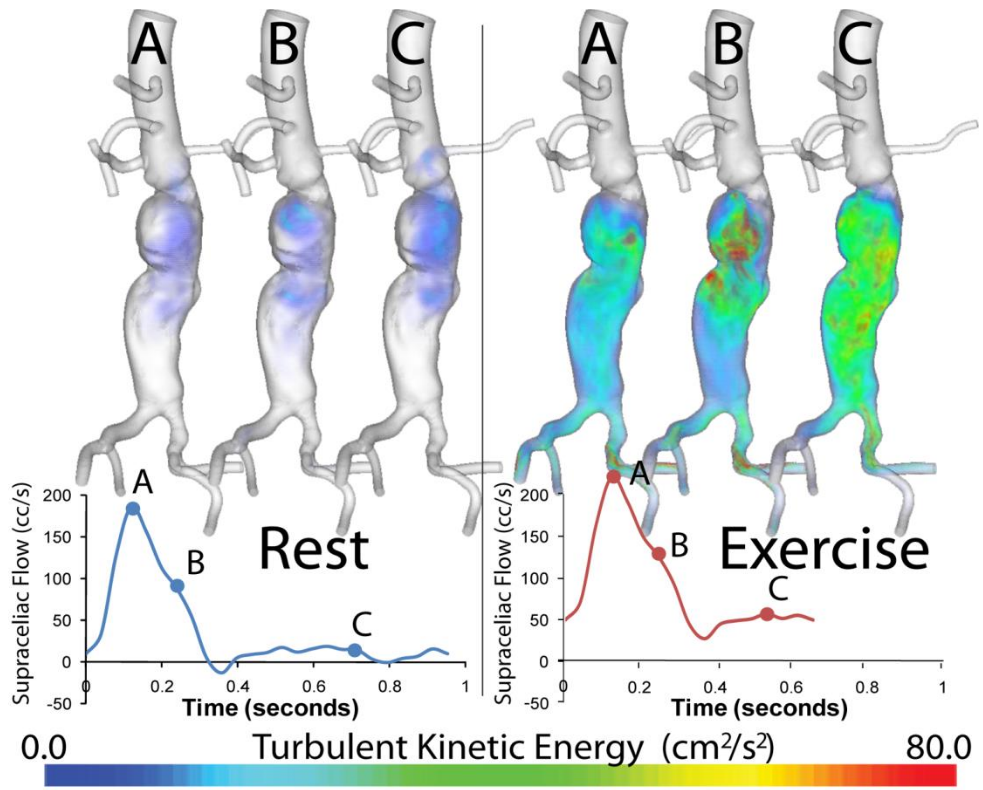
$$u(x,t) = \tilde{u}(x,t) + U(x,t)$$

$u(x,t)$ is the fluid velocity

$U(x,t)$ is the fluid average velocity

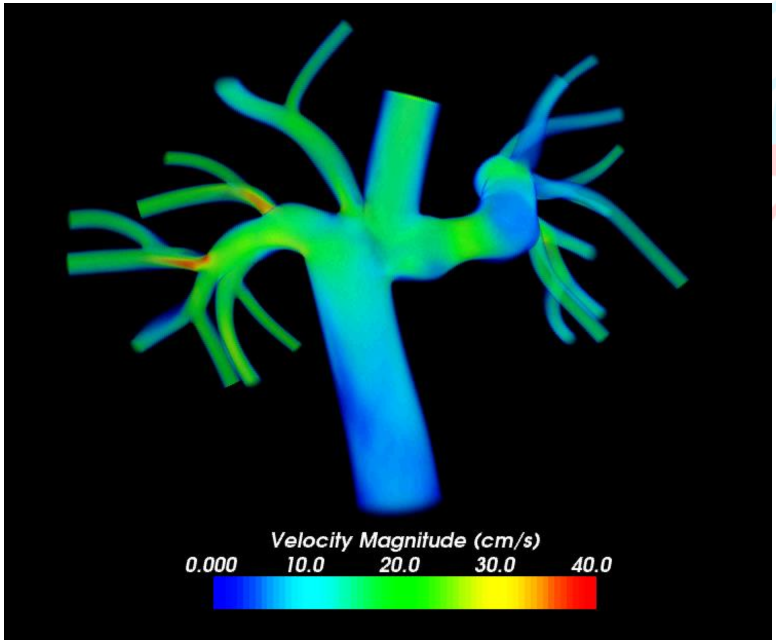
$\tilde{u}(x,t)$ is the fluid deviation from this average velocity

$\langle\tilde{u}(x,t)\rangle$ is the r.m.s $= \sqrt{\frac{1}{n}\sum_{i=1}^{n}\tilde{u}_i^2} = \sqrt{\frac{1}{3}\left[\tilde{u}_1^2 + \tilde{u}_2^2 + \tilde{u}_3^2\right]}$



Eulerian vs Lagrangian description of the flow

Eulerian description of flow in total cavopulmonary connection (TCPC)



Lagrangian description of flow in total cavopulmonary connection (TCPC)