Arterial Compliance & Disease

ES 240 Final Presentation

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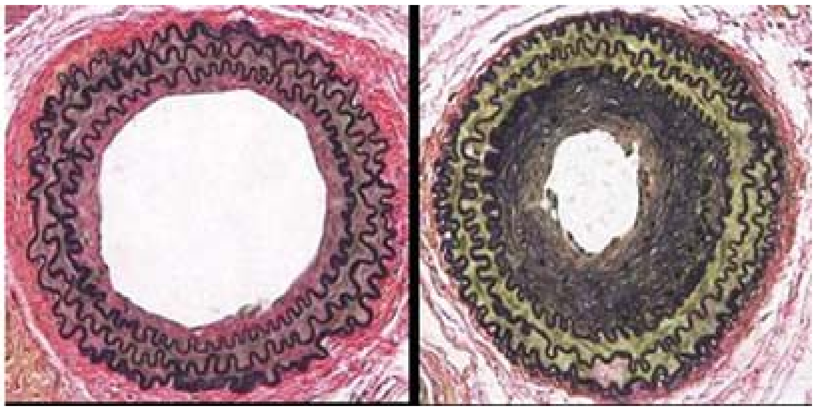
Artery Construction and Disease

- The tunica media is composed of vascular smooth muscle cells which maintain vascular tone
- Vascular smooth muscle exhibits stress-relaxation



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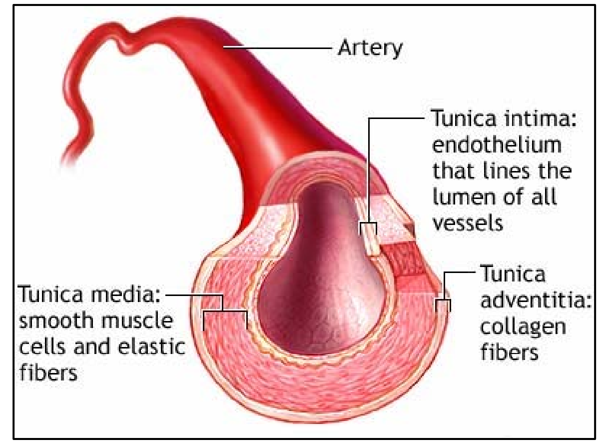

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Arteriosclerosis: Increasing wall thickness and stiffness in the arterial wall

Neointimal hyperplasia: Proliferation of cells which decreases lumen diameter

Hypertension is positively correlated with low compliance and wall thickening

Medical Physiology, Guyton 2005 & Introduction to Vascular Biology

The Problem & Approach

Explain arterial wall thickening from a mechanical perspective

Our Approach:

- Find an analytical solution using Lamé and the Zener model
- Analyze data from the literature
- Create a FEM ABAQUS model, taking material properties from the literature
- Compare the compliance for healthy and diseased arteries

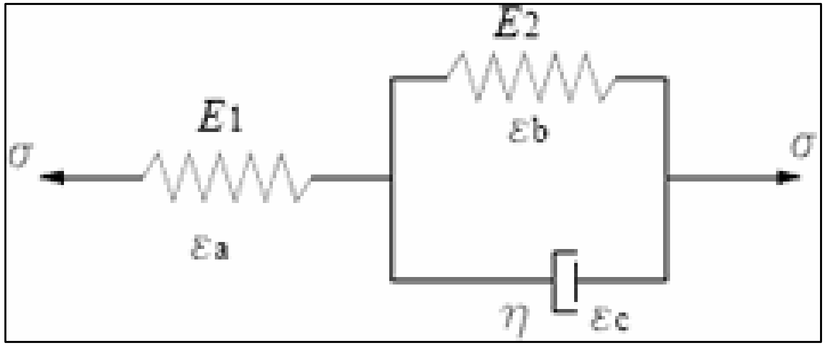
Main Assumptions:

- This is a 2D plane strain problem
- The artery is linearly elastic, isotropic, and homogeneous
- Negate arterial contraction, this is a creep problem

Arteriosclerosis will be studied mechanically

Defining Biaxial Constiutive Law

Zener model for viscoelasticity



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Strain history curve fit

$$\varepsilon_t = \varepsilon_U + (\varepsilon_R - \varepsilon_U)\left(1 - e^{-\frac{t}{\tau}}\right)$$

Total strain of elements in series

$$\dot{\varepsilon}_t = \dot{\varepsilon}_a + \dot{\varepsilon}_c \qquad \dot{\varepsilon}_t = \frac{2\sigma}{\tau}\bar{\bar{A}} - \frac{1}{\tau}\varepsilon_t \qquad \tau = \frac{H}{E}$$

Solution to ODE - biaxial viscoelastic material law

$$\varepsilon_r = \frac{2}{E}(\sigma_r - \nu\sigma_\theta) - \frac{2}{E}(\sigma_r - \nu\sigma_\theta)e^{-\frac{t}{\tau}}$$

$$\varepsilon_\theta = \frac{2}{E}(\sigma_\theta - \nu\sigma_r) - \frac{2}{E}(\sigma_\theta - \nu\sigma_r)e^{-\frac{t}{\tau}}$$

Material law equations were solved

Ellyin et al, 2005

Analytical Solution

Combination of the strain – displacement equations

$$\varepsilon_r = \frac{\partial(r \cdot \varepsilon_\theta)}{\partial r}$$

Lame's solution for viscoelastic materials

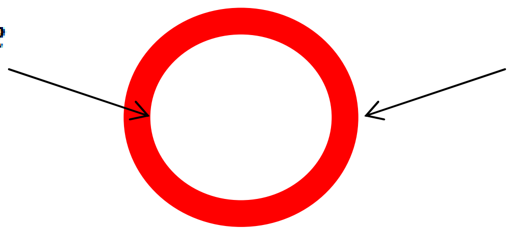
$$\sigma_r = \left(A + \frac{B}{r^2}\right) \quad \sigma_\theta = \left(A - \frac{B}{r^2}\right) \quad A = -\frac{SR_a^2}{R_b^2 - R_a^2} \quad B = \frac{SR_b^2 R_a^2}{R_b^2 - R_a^2}$$

Creep compliance can be easily solved

$$\varepsilon(t) = D(t)\sigma$$

Qualitative Description

σ_r is *equal to pressure*
σ_θ is *large*



$\sigma_r = 0$
σ_θ is *small*

Lame's solution for a viscoelastic finite ring in plane stress was determined

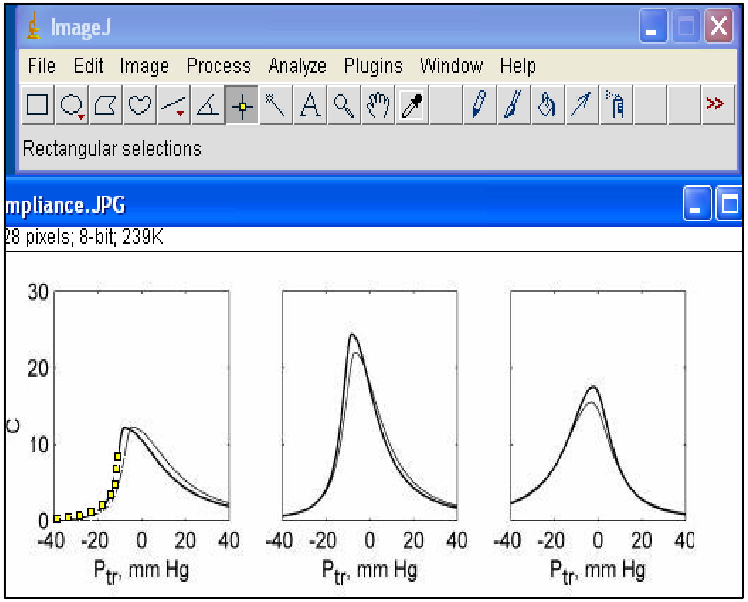
Determining Compliance & Strain vs. Time

$$\varepsilon(t) = \int_{-\infty}^{t} D(t-u)\frac{d\sigma}{du}du$$

Strain history integral

σ(t)= 12.5 + 3.3sin(2π*t)

Internal hydrostatic pressure applied



Talts et al. 2006

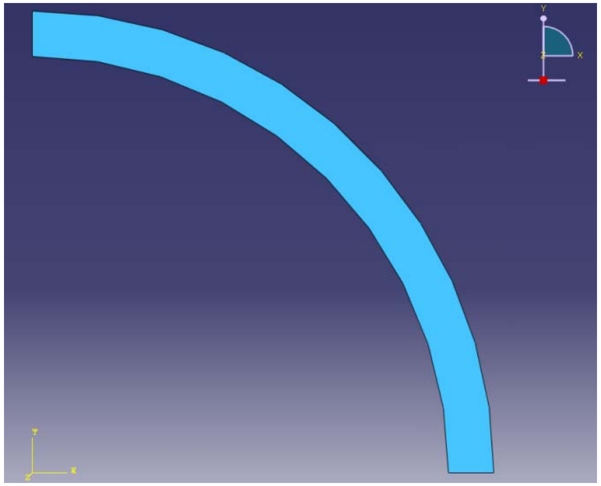
ImageJ compliance determination

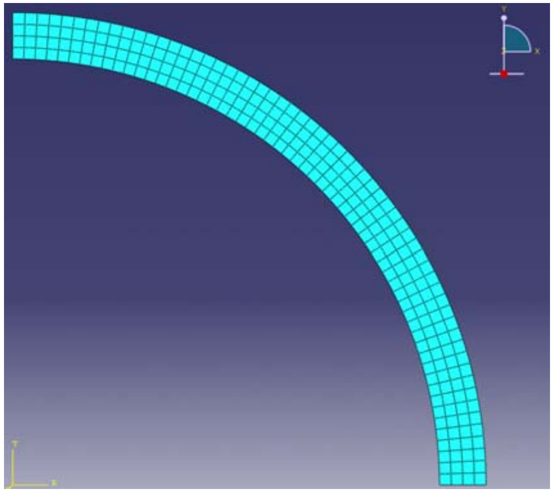
Experimental compliance data was used for the model and analytical solutions

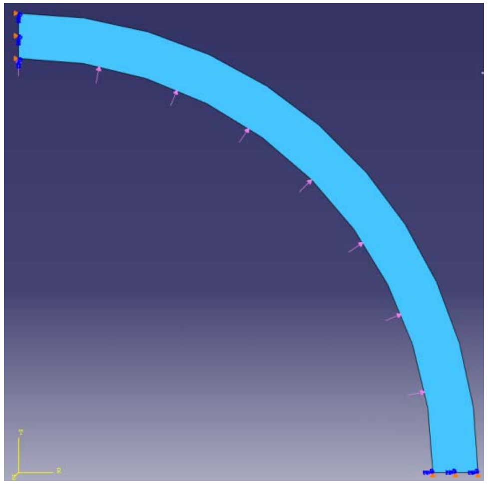
Method of Analysis

Main Assumptions for FEM ABAQUS model:

- 2D plane strain problem → shell planar element
- Material is linearly elastic, isotropic, and homogeneous
- Constant stress → creep problem
- Quarter-ring is sufficient and computationally efficient
- Cyclic vascular pressure modeled by periodic sine function







An axisymmetric 2D plane strain element was developed.

Defining Viscoelastic Parameters

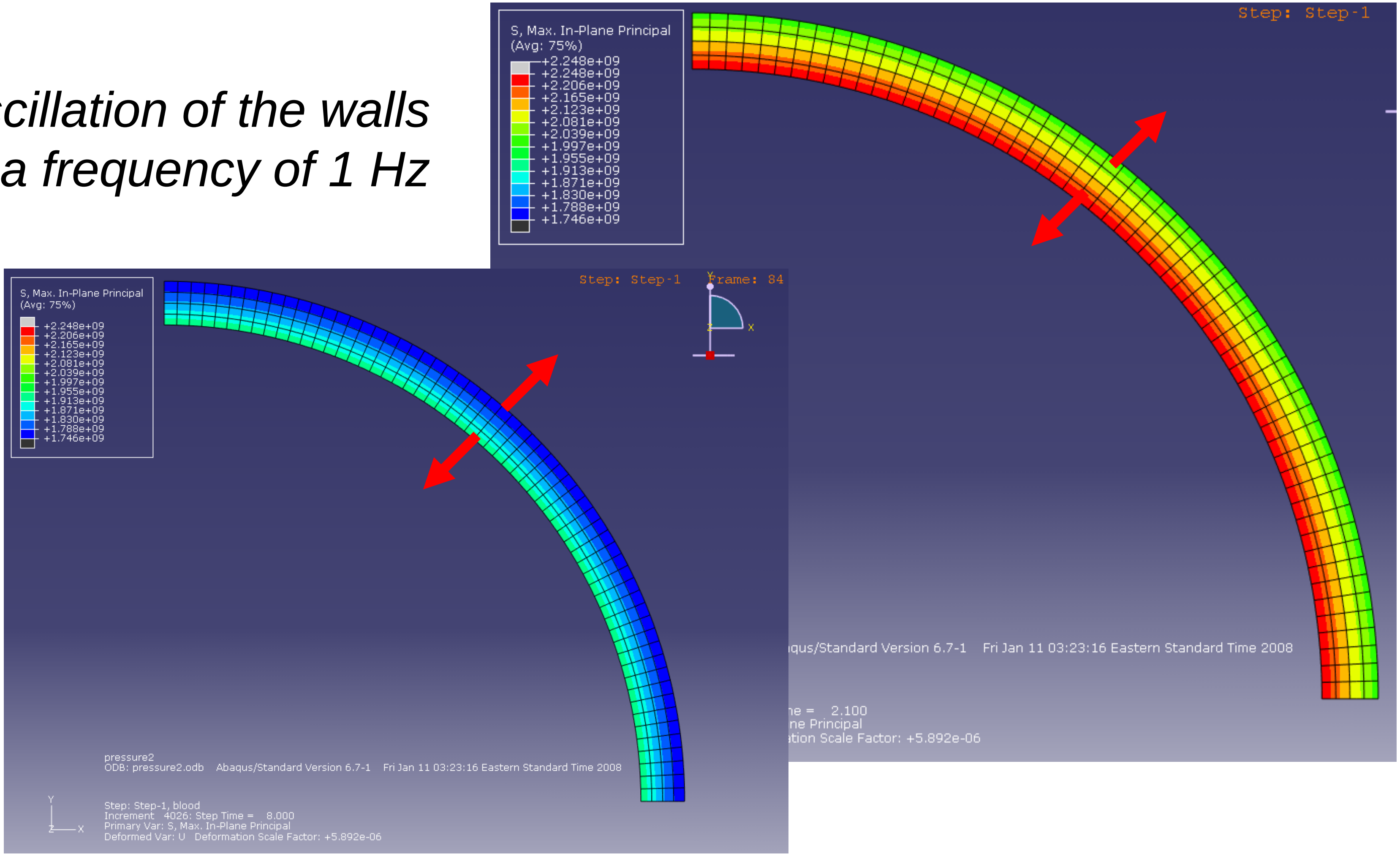
Parameters determined from the clinical literature

Nagai: carotid m/w E=94.4+/- 33.5 kPa
Nagai: carotid resting diameter 0.55 +/-0.06 cm
Nagai: carotid stressed 0.58+/- 0.06cm
Nagai: BP 120/70 mmHg
Riley: Carotid intimal thickness men/women 0.06cm
Armantano: up BP, down compliance, down distensibility
Sarma: poisson ratio=0.375, density = 1.06g/ml
Lichtenstein: volumetric compliance 0.016mm^3/mmHg/mm vessel
Lichtenstein: hypertensive volumetric compliance 0.009mm^3/mmHg/mm vessel
Lichtenstein: kR = 128114.5173 Pa

We gleaned and cleaned the literature for our model's parameters.

FEM Results--Healthy

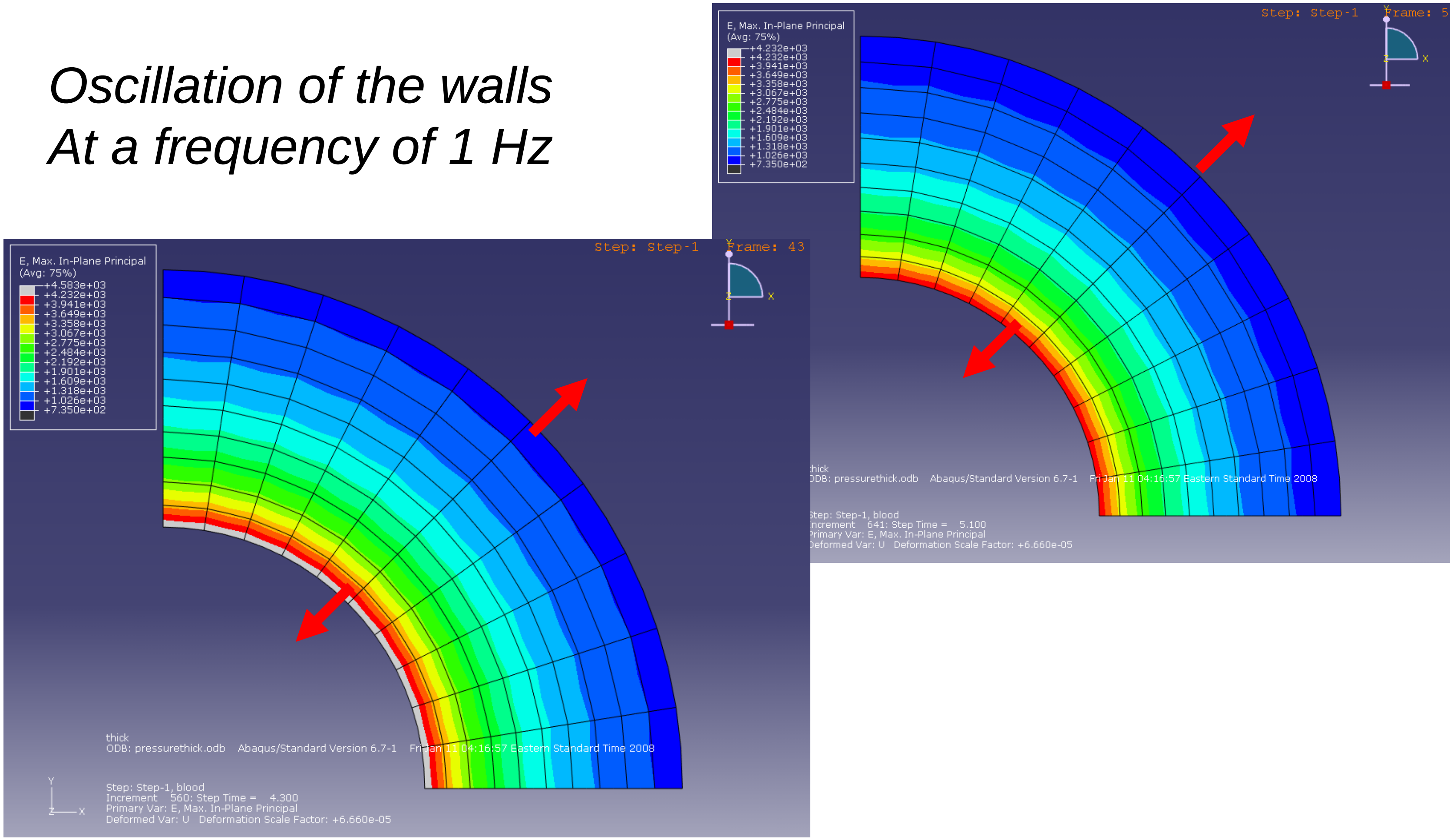
Oscillation of the walls
At a frequency of 1 Hz



*Healthy arterial cross section has 0.06 cm walls. Model incorporates cyclic blood pressure Y(t)= 95 + 25sin(2*pi*t)*

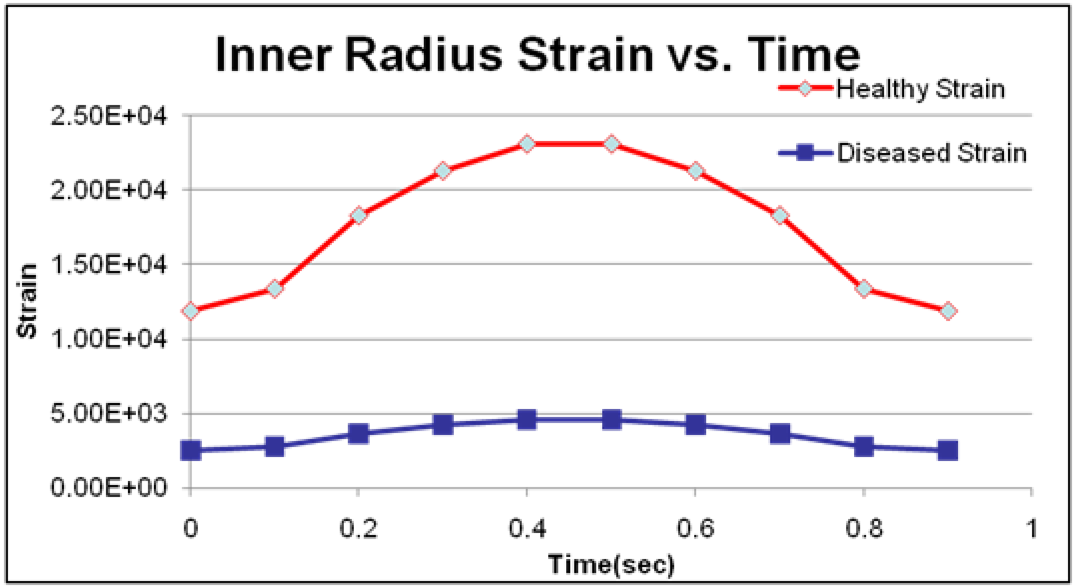
FEM Results--Diseased

Oscillation of the walls
At a frequency of 1 Hz



We modeled 6-fold inward wall growth for the diseased condition.

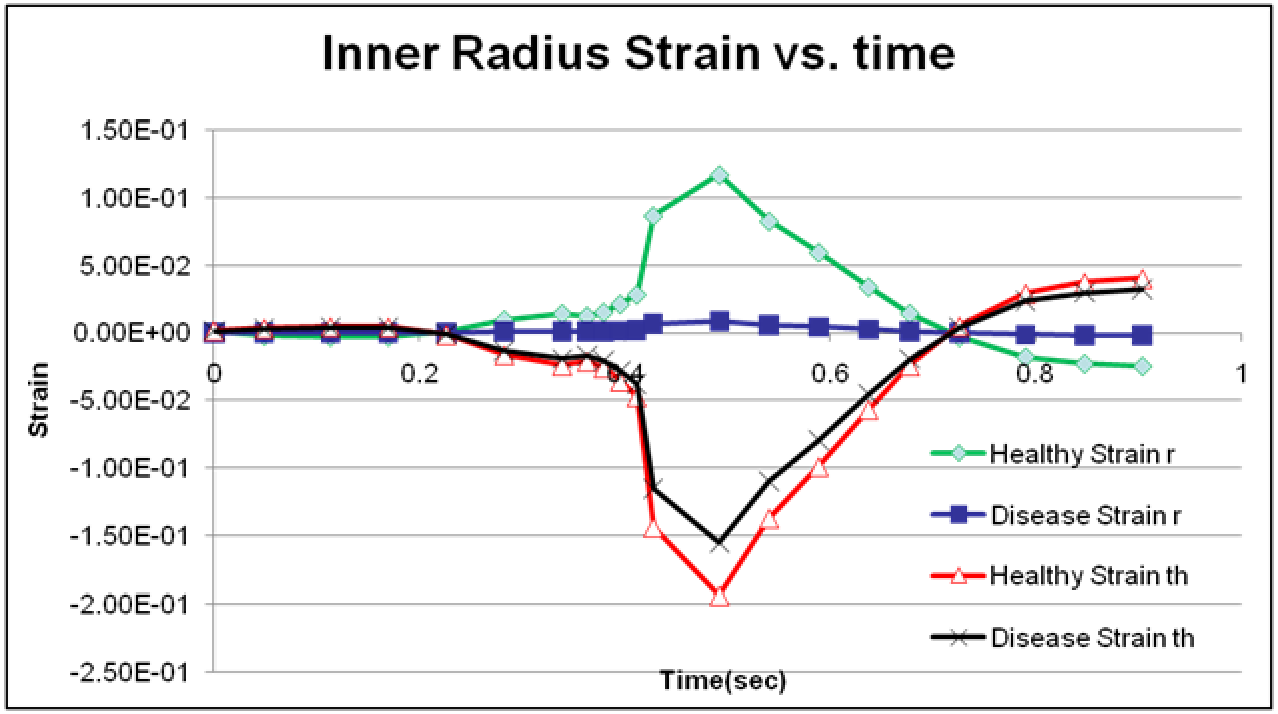
Healthy vs. Disease Compliance



- The FEM analysis indicates that the max principal strain on the inner surface of the diseased artery is an order of magnitude lower than on the healthy one.
- The compliance of the diseased artery wall is lower. It is, therefore, more brittle and less able to distend. This is consistent with arteriosclerosis conditions.

Strain vs. time was calculated

Healthy vs. Disease Compliance



- Maximum radial strain values in healthy individuals were 0.073 (Nagai, 1999)
- Curves exhibit typical viscoelastic hysteresis
- Compliance was decreased in the diseased artery

$$\varepsilon_r = \frac{2}{E}(\sigma_r - \nu\sigma_\theta) - \frac{2}{E}(\sigma_r - \nu\sigma_\theta)e^{-\frac{t}{\tau}}$$

Strain vs. time was calculated

Analytical vs. FEM Model Solutions

Challenges encountered in both methods

Analytical: Acquiring D(t) data, data was primarily dependent on unrelaxed compliance (elastic behaviour)

FEM: magnitude of output values, precise nodal history outputs. However, allowed for cyclic viscoelastic strain visualization.

Both approaches verified that compliance is reduced in the diseased artery

Conclusions & Significance

	Inner Radius	Outer radius
Stress θ	-0.199	-0.875
Stress r	-0.631	-0.268
Strain θ	-0.201	-0.875
Strain r	0.200	-0.875
Compliance θ	0.000	0.000
Compliance r	0.091	-1

Final change in stress, strain, and compliance when the intima is increased by 50% (inward)

The 4 Mechanical Steps to Arterial Disease

1. Blood pressure increases due to hypertension
2. To reduce stress the cells proliferate, thickening the wall
3. Radial compliance decreases, stiffening the artery
4. The artery becomes susceptible to plaque/emboli formation

References

1. Guyton A. *Textbook of Medical Physiology. 11th ed; 2005.*
2. Hunt B. *Introduction to Vascular Biology. 2nd ed; 2002.*
3. Lichtenstein O, Safar ME, Poitevin P, Levy BI. Biaxial mechanical properties of carotid arteries from normotensive and hypertensive rats. *Hypertension. 1995;26(1):15-19.*
4. Nagai Y, Fleg JL, Kemper MK, Rywik TM, Earley CJ, Metter EJ. Carotid arterial stiffness as a surrogate for aortic stiffness: relationship between carotid artery pressure-strain elastic modulus and aortic pulse wave velocity. *Ultrasound in medicine & biology. 1999;25(2):181-188.*
5. Riley WA, Evans GW, Sharrett AR, Burke GL, Barnes RW. Variation of common carotid artery elasticity with intimal-medial thickness: the ARIC Study. Atherosclerosis Risk in Communities. *Ultrasound in medicine & biology. 1997;23(2):157-164.*
6. Sarma PA, Pidaparti RM, Meiss RA. Anisotropic properties of tracheal smooth muscle tissue. *Journal of biomedical materials research. 2003;65(1):1-8.*