Finite Element Modeling Of VIIP Syndrome

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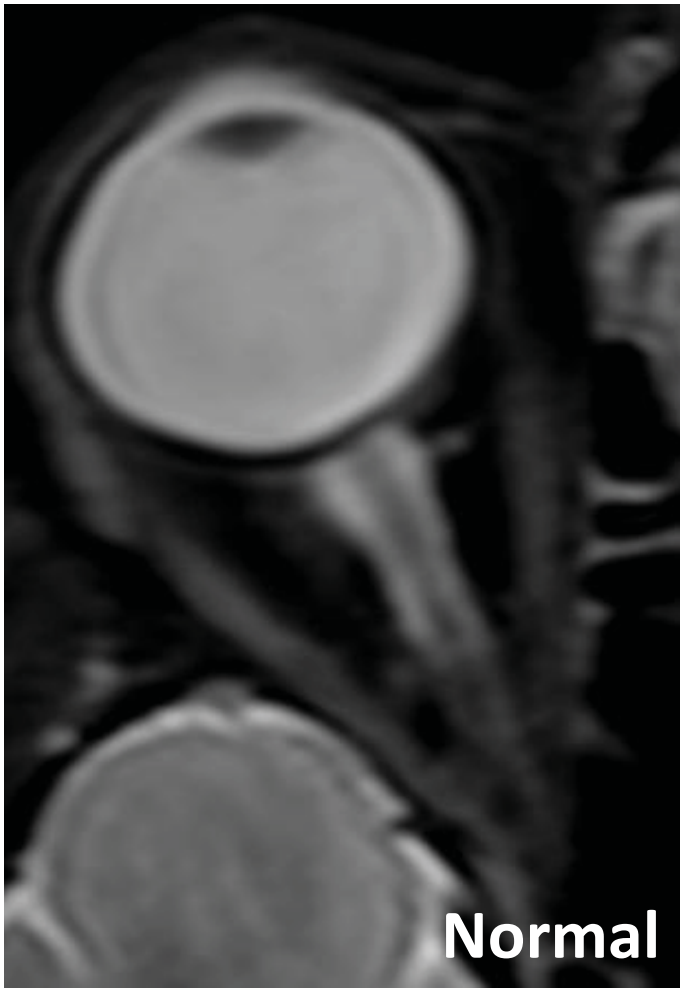
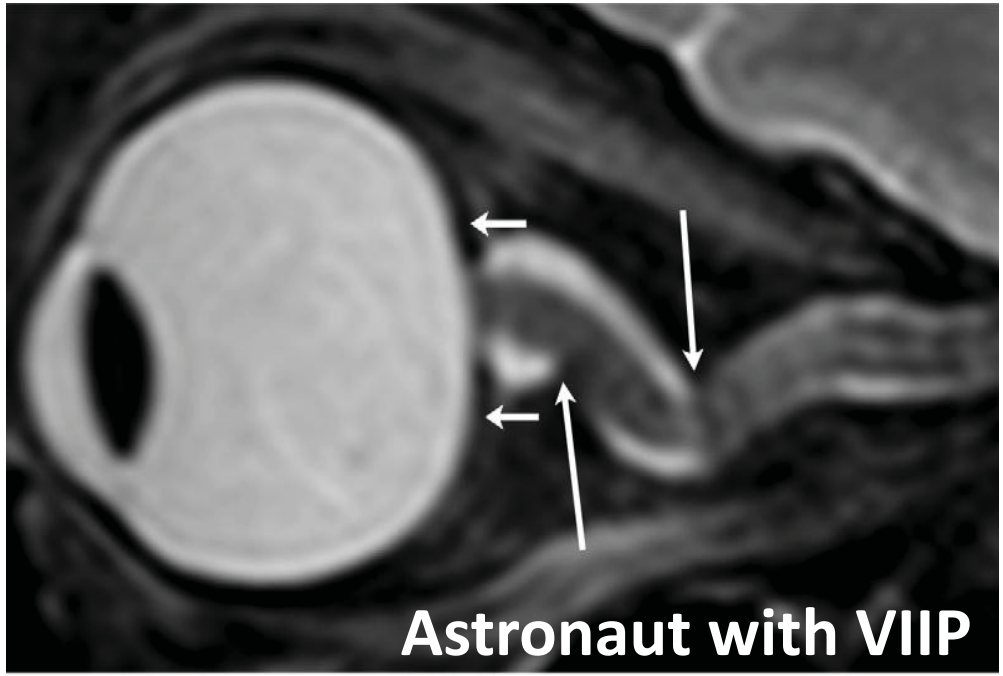
Structural Changes in the Optic Nerve





Kramer et al. Radiology, 2012.

Hypothesis

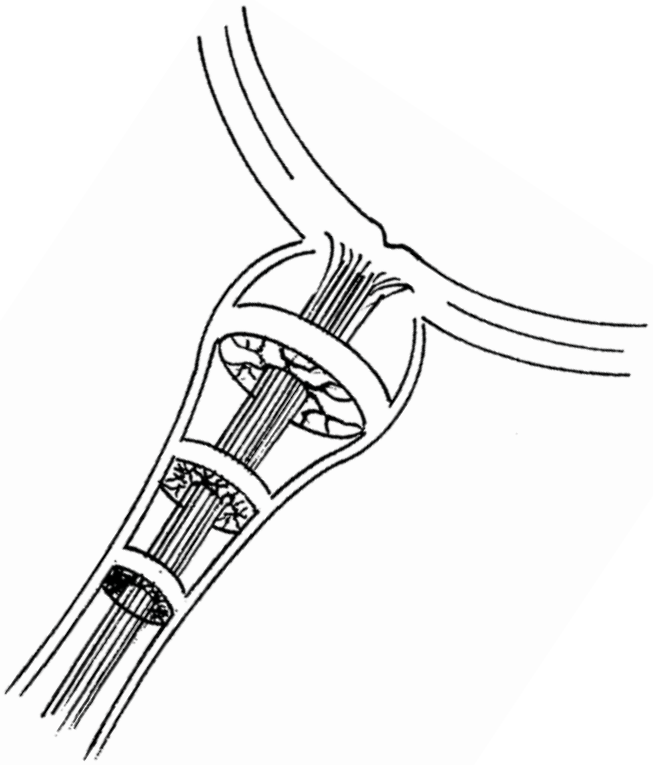
Increased CSF pressure drives remodeling of connective tissues in the posterior eye and optic nerve sheath

Goal

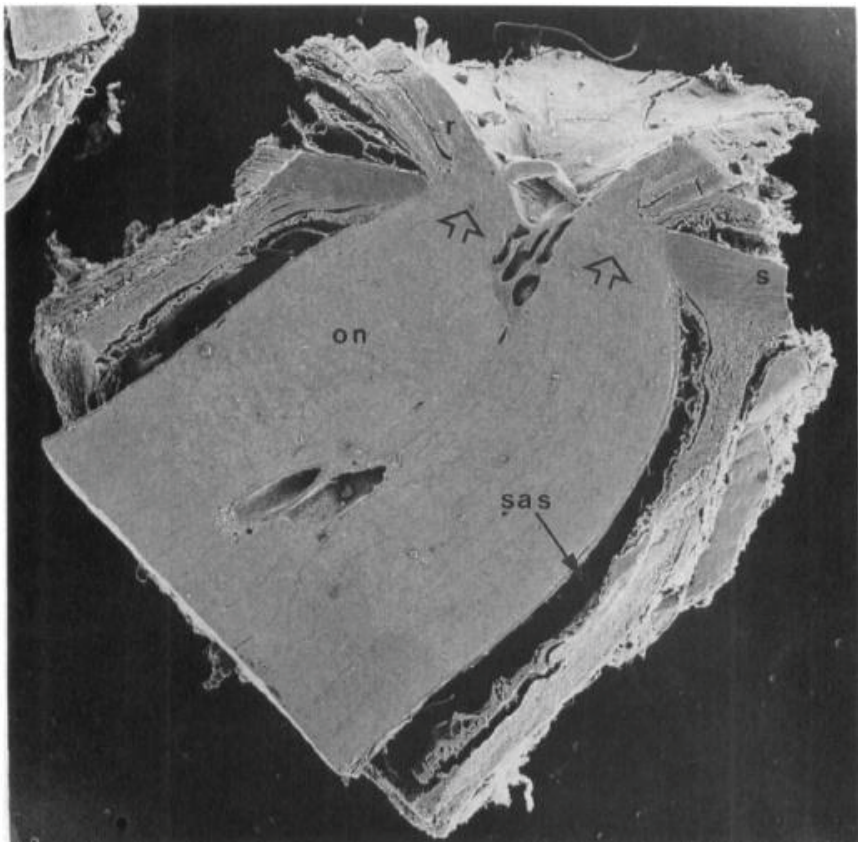
Study the biomechanical response of the optic nerve sheath and posterior eye to elevated CSF pressures

Eventually, understand visual disturbances that occur during long-duration space travel

Basic Modeled Geometry



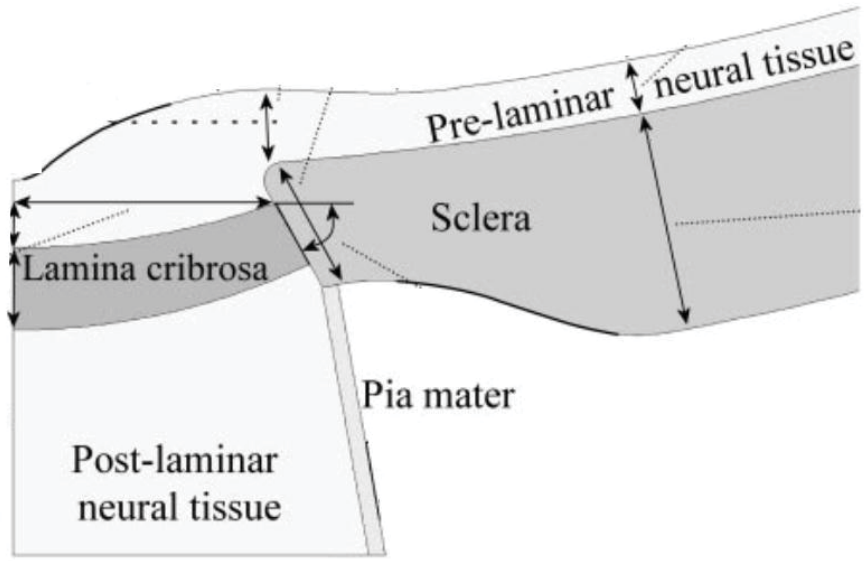
Hansen et al. Acta Ophthalmologica, 2011.

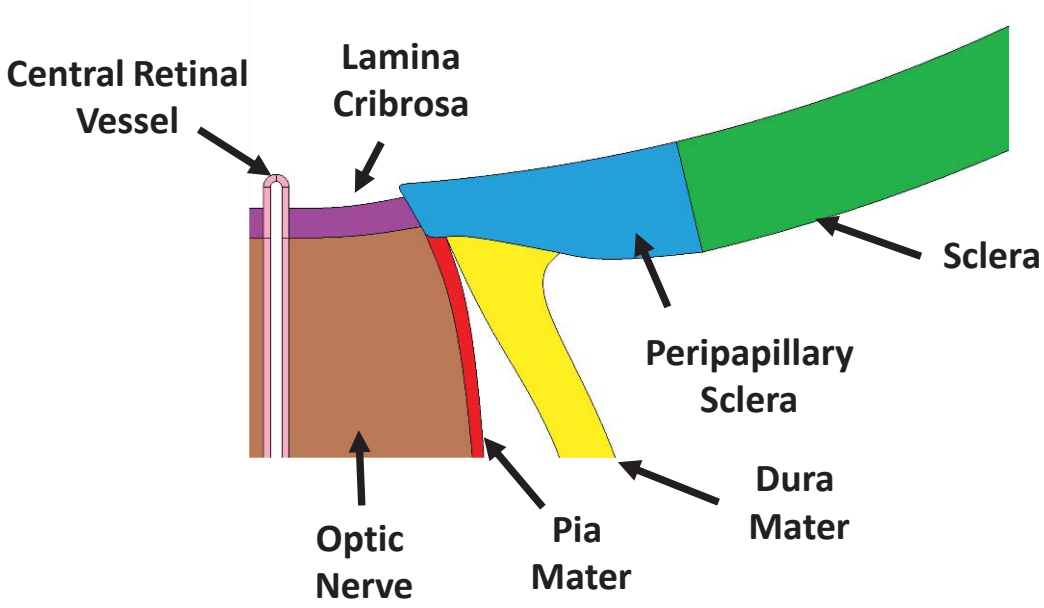


Adopted from Ekington et al. 1990

Optic Nerve Head (ONH) Geometry

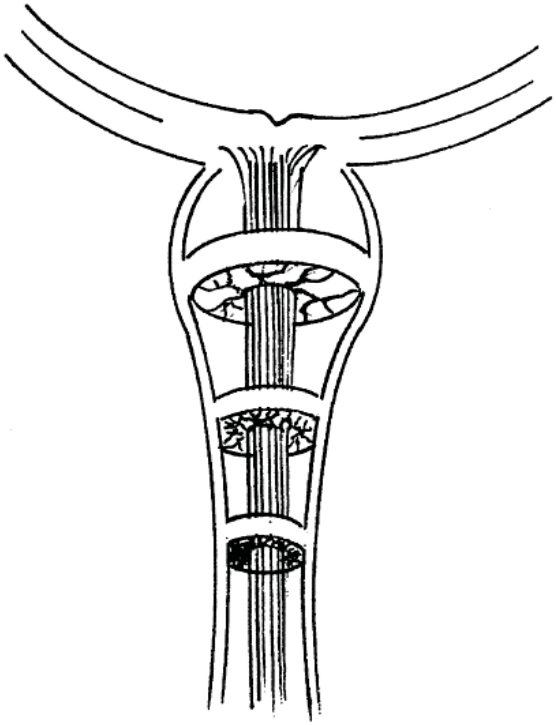
- Based on models of Sigal et al., 2005

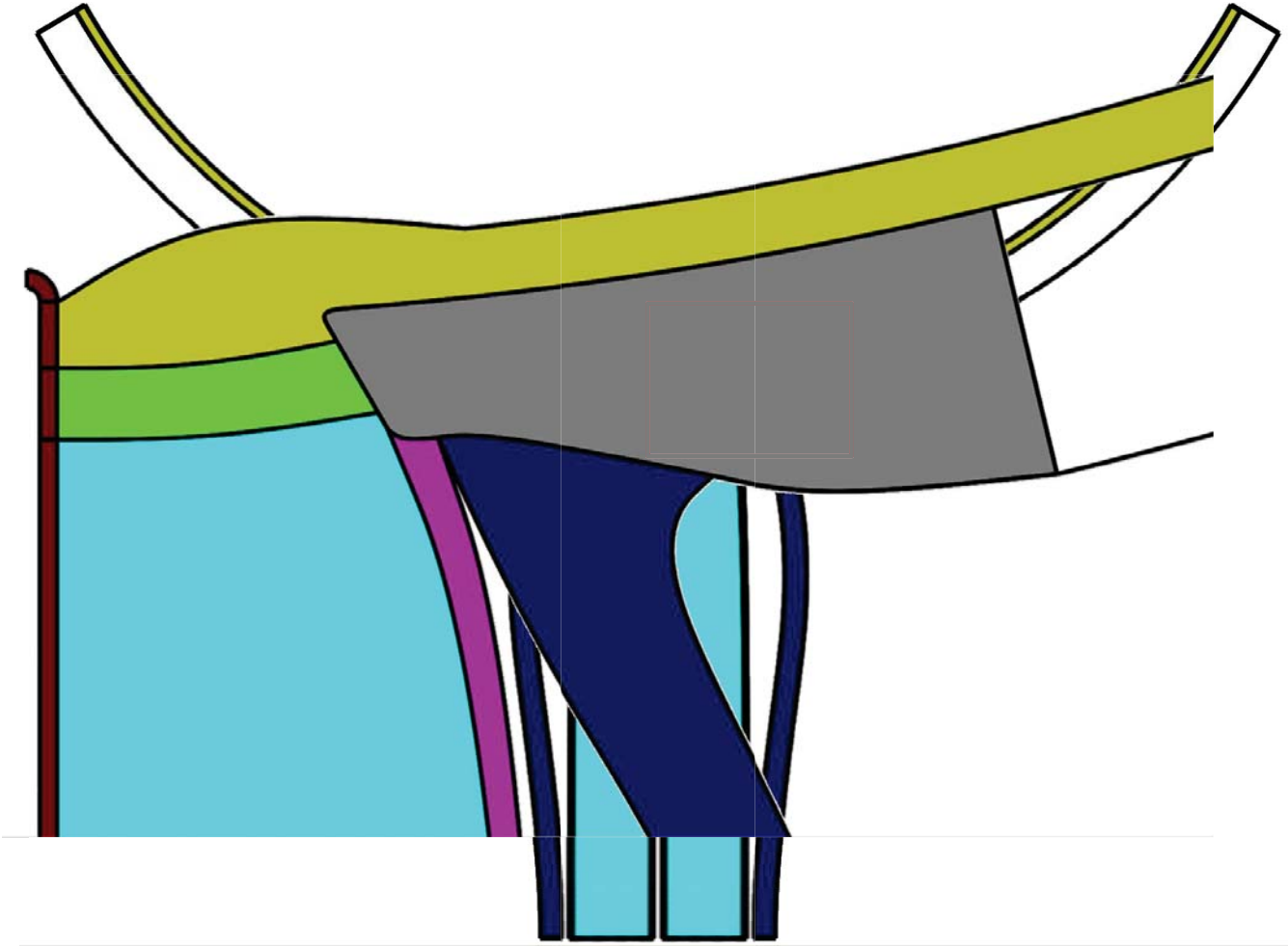




Sigal et al. 2005

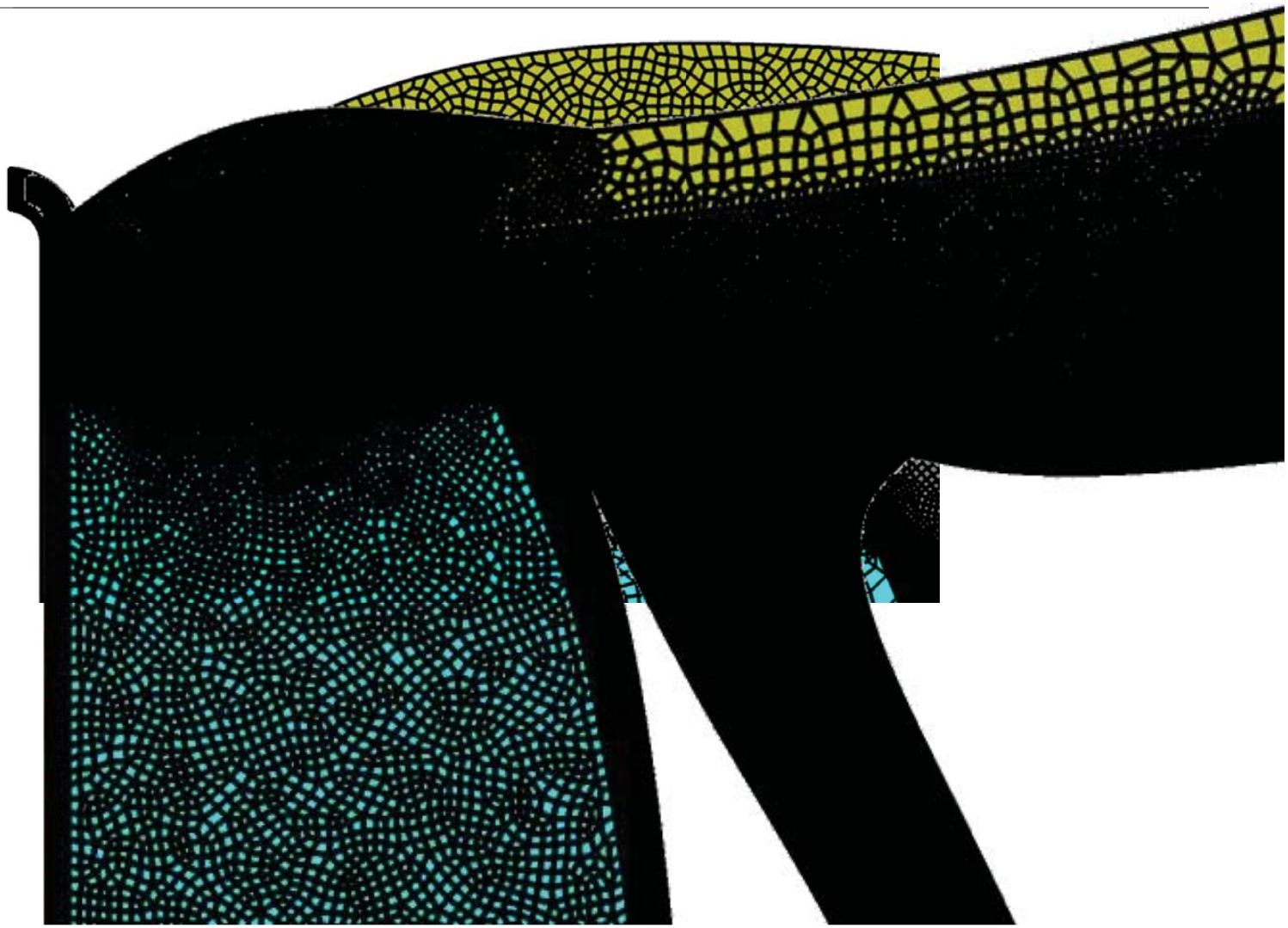
Model Overview





Finite Element Mesh

Gmsh – (version 2.8) was used to create the geometry and mesh for our finite element model



Finite Element Framework

Simulations were run using FEBio (V2.0) assuming all tissues were isotropic, linear-elastic and incompressible.

Component	Modulus (MPa)	Number of Elements (Hexahedral)
Sclera	3.0	4139
Peripapillary sclera	3.0	7304
Retina	0.03	3608
Lamina cribrosa	0.3	4415
Optic nerve	0.03	25308
Pia mater	3.0	19662
Dura mater	1.0	17935
Central retinal vessel	0.3	27944 (of which, 51 prism elements)

Loading conditions

1. Baseline (Standing or walking)

   IOP – 15 mmHg **ICP – 0 mmHg** RVP – 55 mmHg

2. Supine

   IOP – 15 mmHg **ICP – 12 mmHg** RVP – 55 mmHg

3. Elevated ICP

   IOP – 15 mmHg **ICP – 30 mmHg** RVP – 55 mmHg

Outcome measures

- Strain (fractional tissue elongation) in all tissue regions
  - Strain is a tensor and can be decomposed into 3 primary components
    - First principal strain (stretch)
    - Second principal strain
    - Third principal strain (compression)
- Why do we care about strain?
  - Cells are mechanosensitive and alter their phenotype in response to mechanical strain

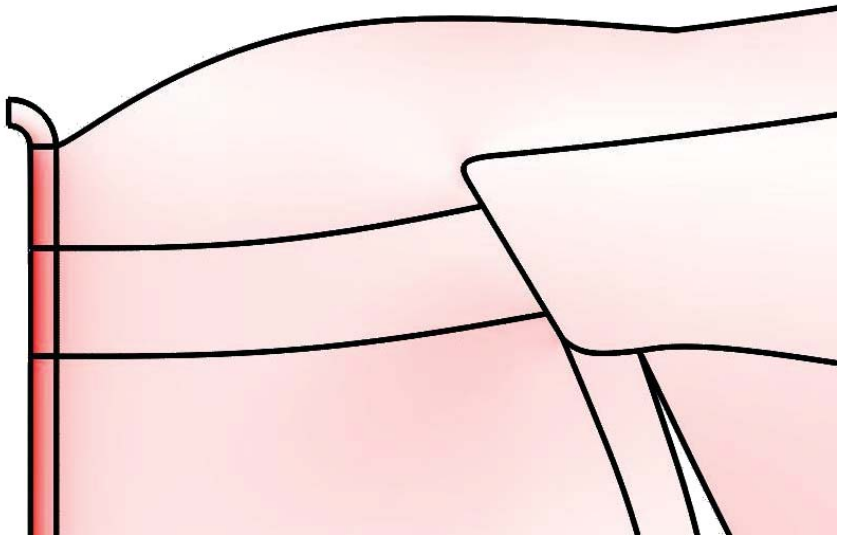
First Principal Strain

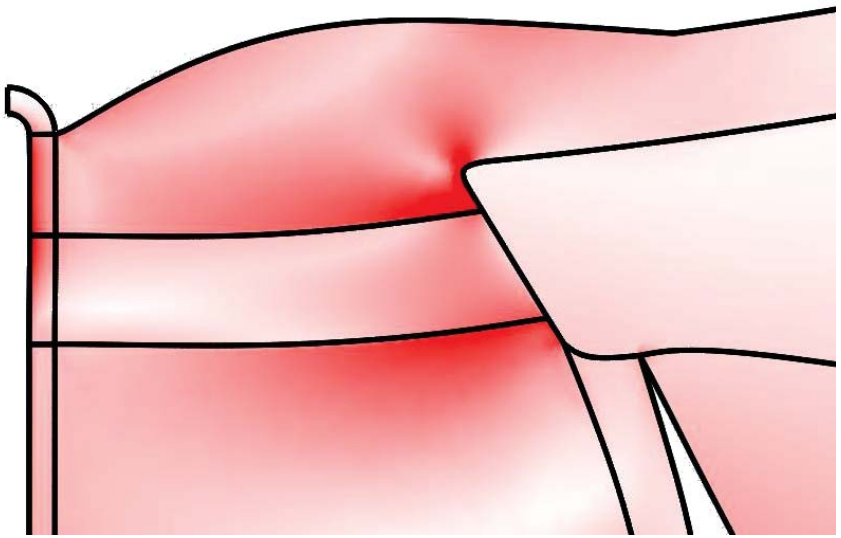
ICP: 0 mmHg

IOP: 15 mmHg

ICP: 30 mmHg

IOP: 15 mmHg





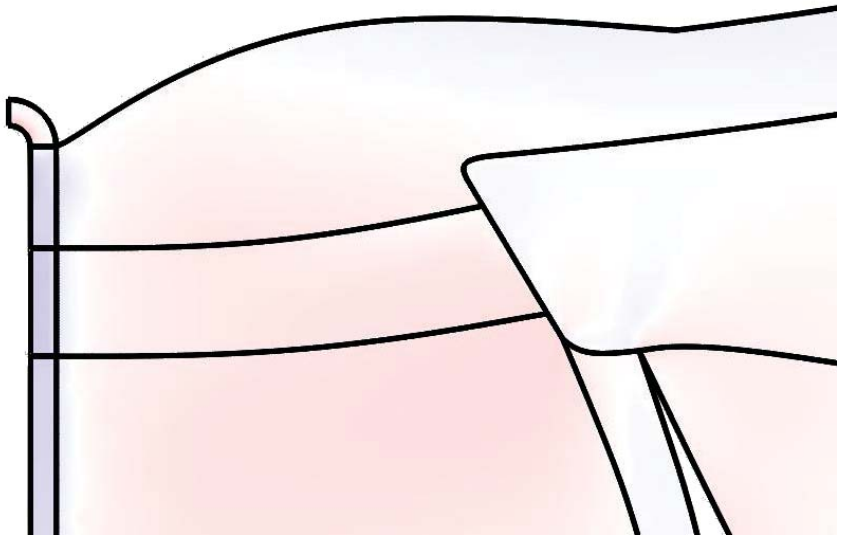
Second Principal Strain

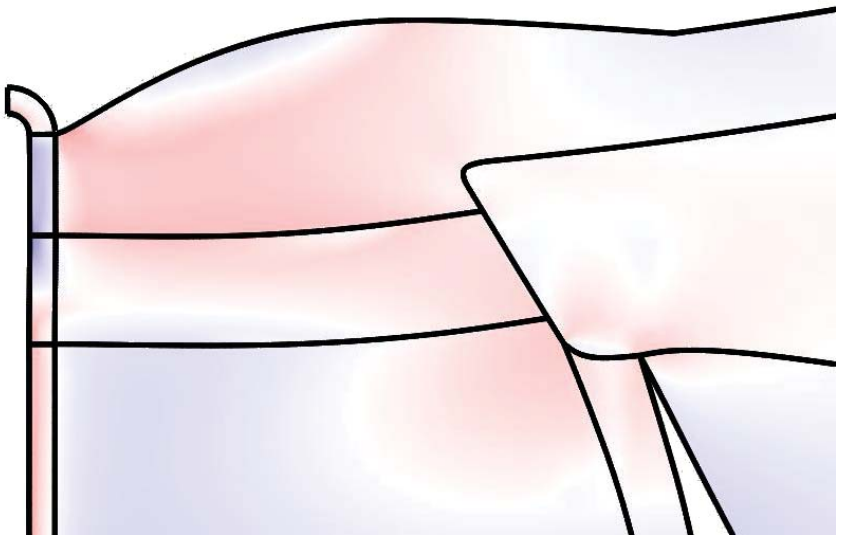
ICP: 0 mmHg

IOP: 15 mmHg

ICP: 30 mmHg

IOP: 15 mmHg





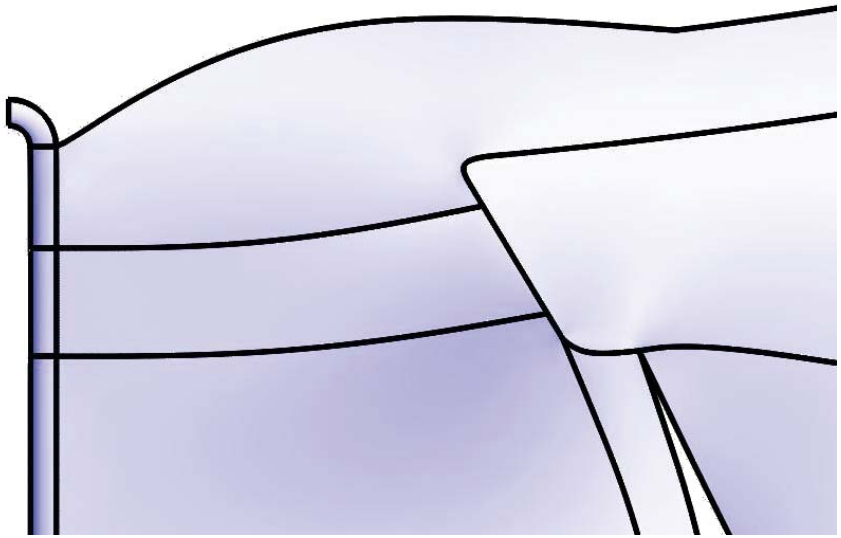
Third Principal Strain

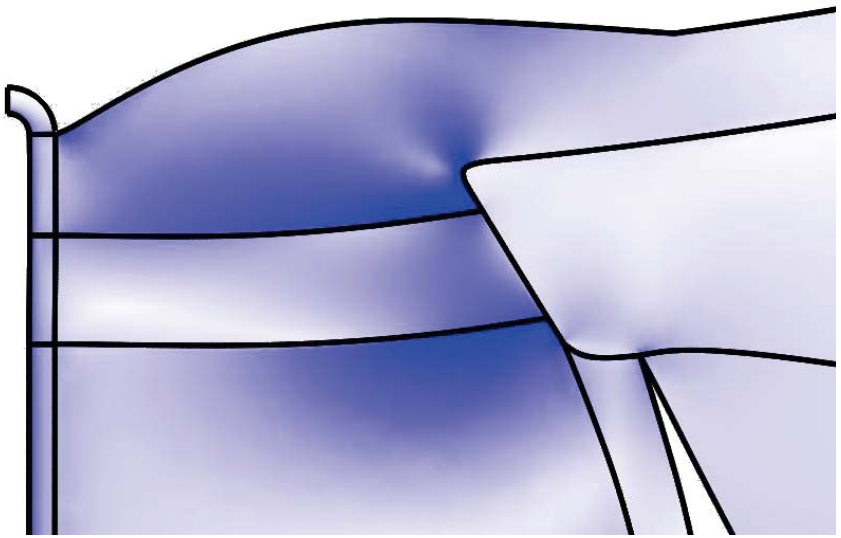
ICP: 0 mmHg

IOP: 15 mmHg

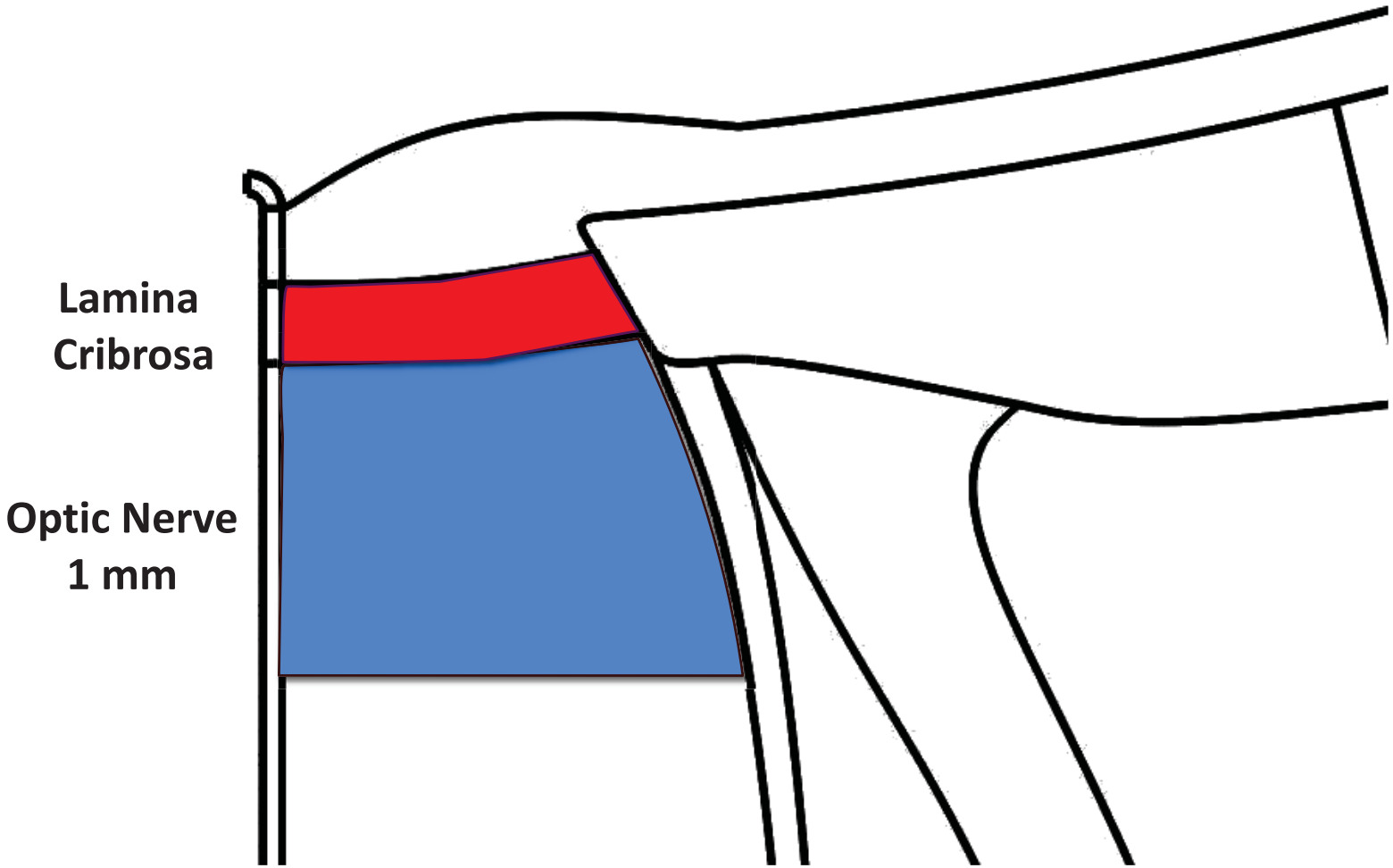
ICP: 30 mmHg

IOP: 15 mmHg

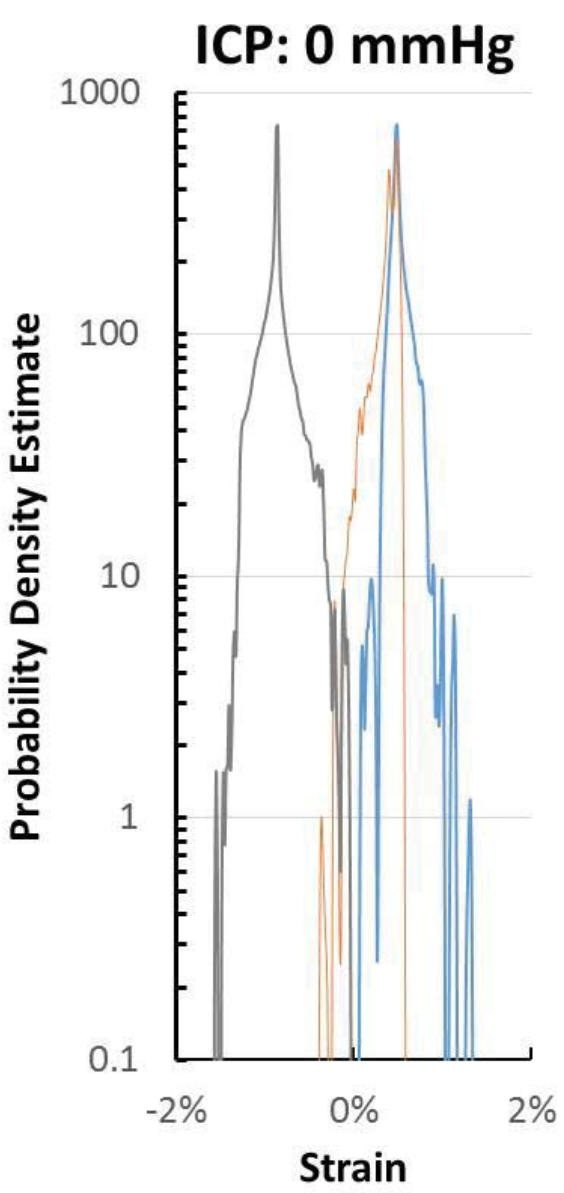


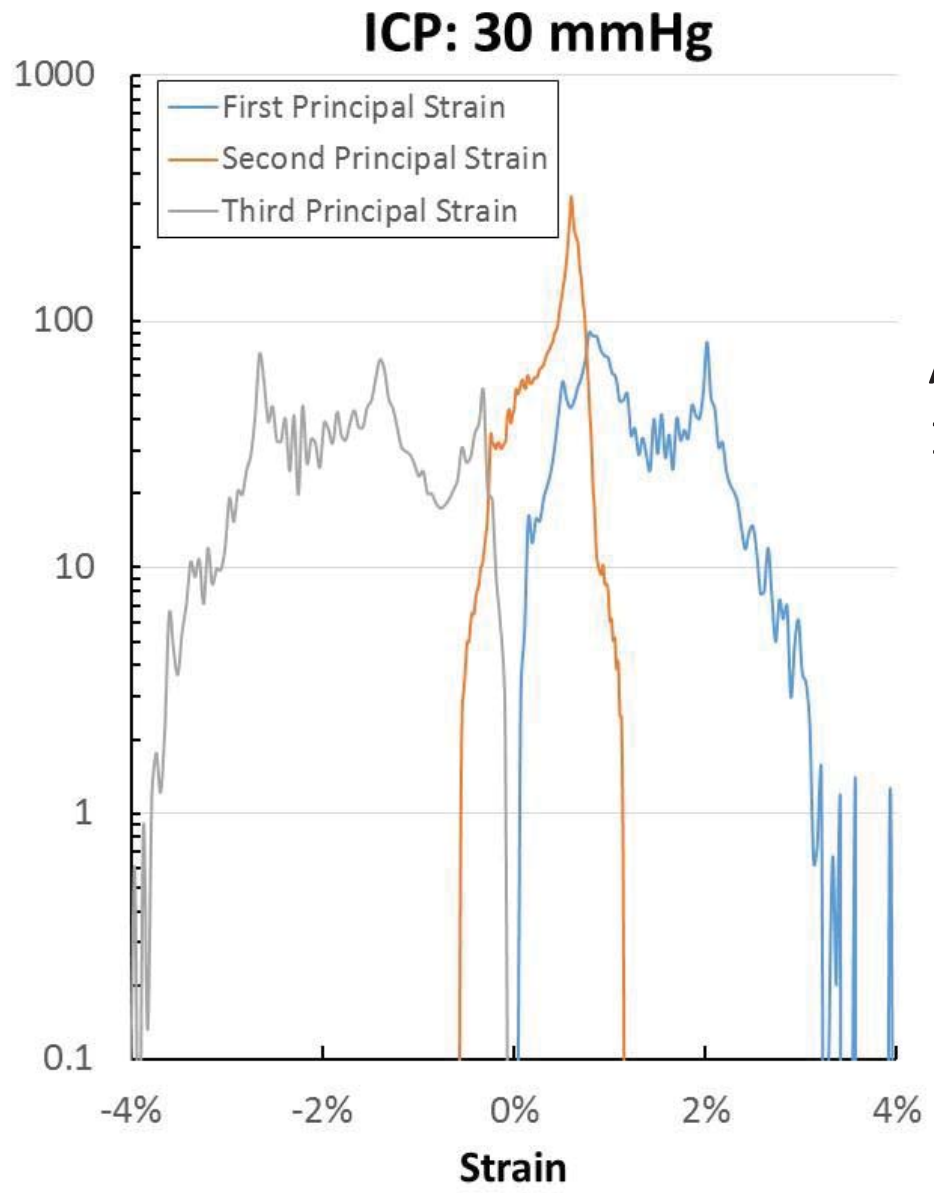


Regions of Interest



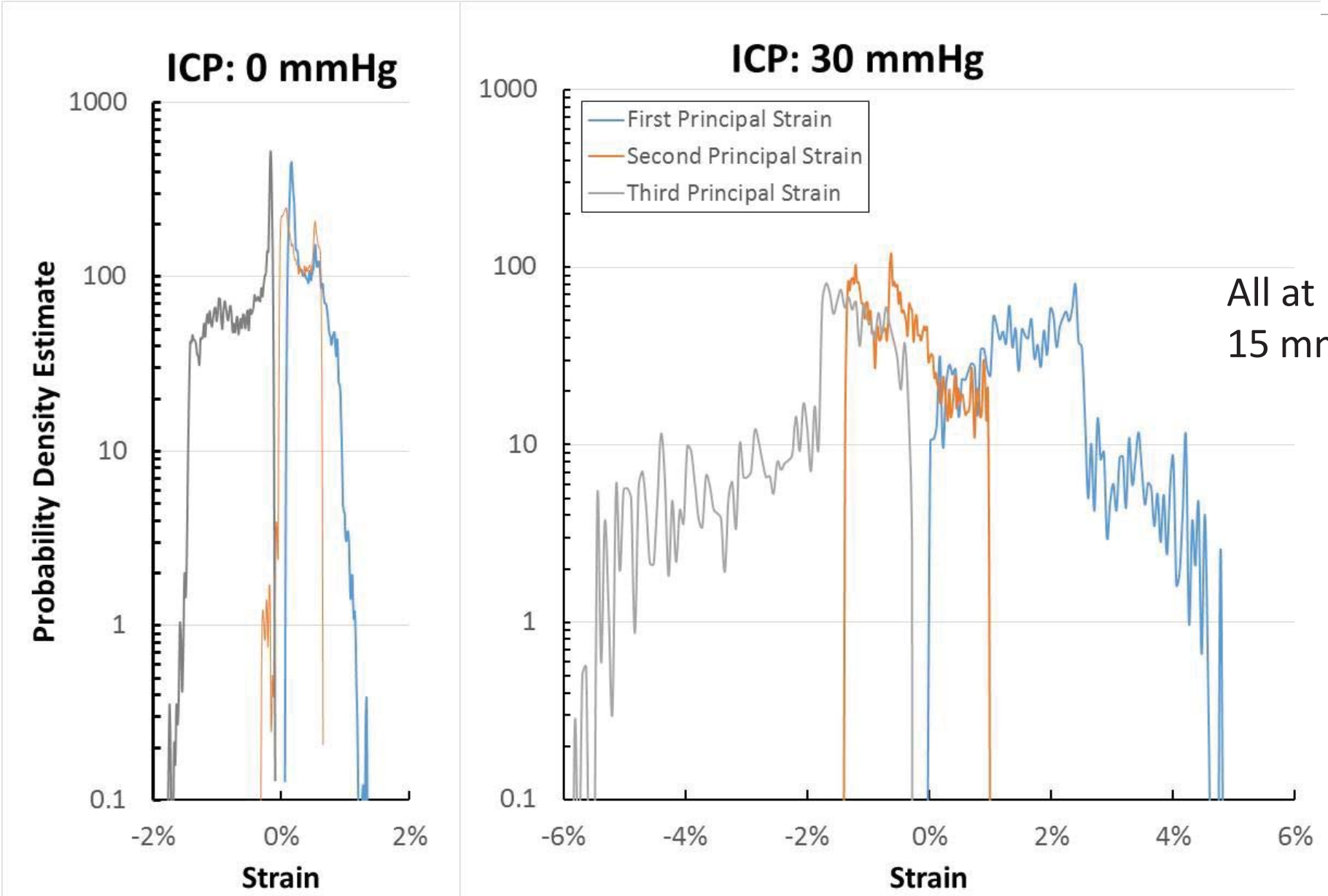
Lamina cribrosa strain histograms





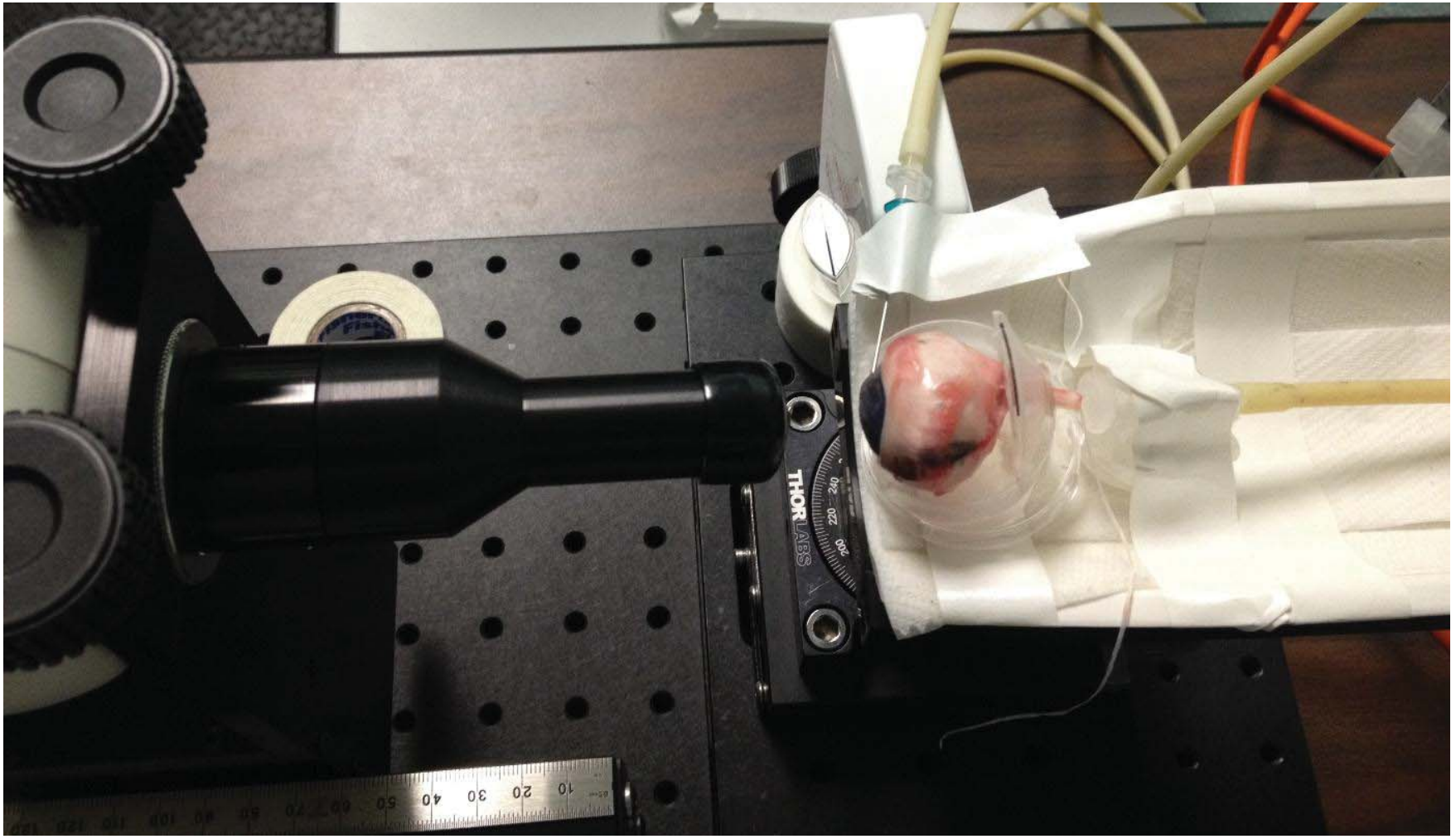
All at IOP:
15 mmHg

Optic nerve strain histograms



All at IOP:
15 mmHg

Bioptigen OCT Imaging



Conclusions

- At a fixed IOP, increasing ICP from 0 to 30 mmHg significantly changed strains within the posterior eye and ONS (more extreme strains).
  - Elevated ICP strongly affects ocular connective tissue biomechanics.
- Little/no anterior motion of the prelaminar neural tissue predicted
  - Optic nerve swelling/papilledema/axoplasmic stasis is typically seen with elevated ICP.
  - Need specialized FE models to capture axoplasmic stasis to study papilledema.
- Mechanical deformations of connective tissues computed by these FE models can inform the design of cell culture and other laboratory models, designed to bridge the gap between biomechanics and pathophysiological function in VIIP.

Related Presentations at IWS

- Poster “Numerical Modeling of Ophthalmic Response to Space” by Dr. Emily Nelson Need times
- Talk “Finite Element Modeling Techniques for Analysis of VIIP” by Dr. Andrew Feola (Time: Thursday at 8:00 am Session: Computational Modeling and Simulation 1)
- Poster “An Integrated Model of the Cardiovascular and Central Nervous Systems for Analysis of Microgravity Induced Fluid Redistribution” by R. Price et al. (K. Heinemann at the poster).
- Talk “Lumped Parameter Models of the Central Nervous System for VIIP Research” by Dr. Jerry Vera.

Acknowledgements

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BME at Georgia Tech/Emory