An Experimental and Finite Element Model for Injury in Aorta

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Abstract—An experimental model of porcine aorta was developed to simulate the pinching phenomenon, i.e., dynamic large deformations due to direct contact which is considered to be one of the mechanisms of TAR. Traumatic descending aortas, instead of a clear urethane box were presented to 3.8 psi (11 kPa). The specimens were fixed at the two ends and allowed to bend in the middle upon impact. A 5 mm flat indenter was placed in front of the specimen approximately at the mid-point and the setup was decelerated at 37g to cause bending. A finite element model of the experiment was developed in LS-Dyna (LSTC, CA) using the Arbitrary Lagrangian Eulerian (ALE) formulation for the fluid and a hyper-viscoelastic-Lagrangian formulation for the aorta material. The aorta wall was assumed to be single-layer, homogeneous and isotropic in the FE model. To address the inhomogeneity of the material properties of aorta, micro-indentation tests were performed on the wall along the radial direction and the variation in the viscoelasticity of the shear modulus was determined.

Materials and Methods

Experimental Model simulating pinching phenomena: 200mm of porcine thoracic aorta incased in a urethane box filled with PBS.

Pressurized up to 1.6 psi or 11 kPa (physiological pressure) at 2 ends and allowed to bend in the middle by 5mm indenter placed approximately at mid-point.

Setup was decelerated at 55g using a highly repeatable impact system.

Finite Element Model:

- Developed in LS-Dyna (LSTC, CA) using the Arbitrary Lagrangian-Eulerian (ALE) formulation for the fluid and hyper-viscoelastic-Lagrangian formulation for the aorta material. The aorta wall was assumed to be single-layer, homogeneous and isotropic in the FE model. To address the inhomogeneity of the material properties of aorta, micro-indentation tests were performed on the wall along the radial direction and the variation in the viscoelasticity of the shear modulus was determined.

- Significant W-shaped deformations were observed in the specimen upon impact and deformation sections of aorta near the indenter showed laceration in inner media. The FE model successfully simulated the experiment in terms of global kinematics and internal pressure and strain history. The strain predicted by the FE model near the indenter was about 30% which agreed with the expected threshold of partial failure for a young human aorta. The results of indentation tests showed that the shear moduli of the aorta wall increased radially toward the media layers which explains why more strain was sustained in inner layers.

- The effective strain predicted by the FE model near the indenter was about 30% (Figure 7).

- The peak deflections in the FE model appear about 5 ms after the experimental results. Also the difference between the maximum distal and proximal deflections was almost double in the FE model.

- These differences were attributed to idealization in the modeling of aorta (homogeneous, isotropic, and constant thickness) approximations in the numerical interface (contact) algorithms.

- The effective strain predicted by the FE model near the indenter was about 30% (Figure 7).

Figure 4. The Instantaneous shear modulus of aorta wall determined from indentation tests.

Finite Element results:

- The FE model was able to successfully simulate the experiment in terms of global kinematics (Figure 5) and simulated large deformations and contacts in the aorta wall. Effective strain in the indentation site reaches the failure limit.

Figure 5. FE model (LSDYNA) of the aorta impact model with local pinching. The model uses ALE formulation and simulates large deformations and contacts in the aorta wall. Effective strain in the indentation site reaches the failure limit.

Figure 6. Comparison between the maximum deflections measured in one experiment and its FE simulation.

Conclusion

The models used in this study showed that dynamic local pinching that created about 30% local effective strain caused laceration in the media layer of aorta. This result confirms the role of pinching (direct contact), as a contributing factor in traumatic aortic injury. However this mechanism, alone is unlikely to cause fatal injury.

Acknowledgement

The support for this study was provided by the NHLBI under Grant Numbers K23HL0881201 and R21 HL08815901 and Temple University College of Engineering.

References