

What can we learn from computational model studies of the eye?

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Understanding the mechanisms of retinal hemorrhages can lead to a better understanding of the externally applied forces and accelerations necessary to cause retinal hemorrhages in infants. Finite element model analysis is one of many computational tools that biomechanical engineers use to help understand how the structural components of the eye may respond to these types of externally applied loads. Because model predictions of tissue deformations and stresses are influenced by the geometry, anatomy, tissue material properties, tissue-tissue interactions, and applied loads used in the model, one should be aware that inaccuracy of these inputs will yield fallacious outputs, and the results of the computer model will have little or no similarity to a real-life response. Because the material properties and tissue-tissue interactions of pediatric ocular tissues have not been measured or published, it is currently impossible for a finite element model to mimic the response of the pediatric eye. Furthermore, it is unknown what values of retinal and ocular stresses produce injury, so these models cannot be used to predict the occurrence of retinal hemorrhages or other ocular injuries. In these circumstances when biomechanical data are limited, engineers use parametric studies—sets of simulations varying some feature of the model over a relevant range—as an important first step to identify tissue components that strongly influence model predictions.

Using adult eye property data in a child-sized eye model as a starting point, Rangarajan and colleagues¹ present a parametric study investigating the effect of vitreous and fat material properties on predictions of retinal stress during cyclic rotations of the eye. The authors report significant differences in retinal stress values and distribution when the stiffness of the vitreous is increased 10-fold and when the tissue-tissue interaction is changed (ie, uniform vitreoretinal adhesion in the fluid model versus vitreoretinal adhesion only at the vitreous base and macula in the solid model). Unfortunately, the authors do not report specific references for each of the material properties used in

the parametric simulations; thus it is difficult to evaluate whether properties are realistic. For example, it is unclear why the authors selected a value of 3.18 MPa for sclera stiffness in their model when Uchio and colleagues² measured adult sclera to be 310 MPa. Additionally, it is unknown whether the 10-fold increase in vitreous stiffness investigated in the parametric simulations (such that vitreous would be approximately 10 times stiffer than the extra-ocular fat) is in the physiologic range. Finally, as with any other current-day ocular finite element model analysis, the lack of pediatric ocular property data precludes Rangarajan and colleagues from predicting actual stress in the eye, and lack of failure stress data prevents them from making any predictions of retinal hemorrhage.

Despite these limitations, the strength of this parametric study is that it demonstrates that the representation of fat and vitreous is important to the prediction of retinal stress in the model. Appropriately, the authors do not conclude which of the adhesion conditions is most true to real life, but they underscore the need for experiments to measure the properties of pediatric vitreous and its regional adhesion of the vitreous.

Given the limited pediatric ocular data, no model at this time can prove or disprove that retinal hemorrhages occur from vitreoretinal traction during repetitive rotational motions of the eye. However, this preliminary study by Rangarajan and colleagues plays an important role in laying the foundation for future biomechanical studies that will increase our understanding of mechanisms of retinal hemorrhages from abusive head trauma. We look forward to the continued development and validation of their model.

References

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See accompanying report on pages 364-369.

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