Investigate the Effect of Angular Acceleration-Deceleration Pulse Shapes on Brain Response using a Validated Human Head Model

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Objective

During the recent decade, the role of the rotational acceleration of head on resulting brain injury has been pursued rigorously in an effort to develop new brain injury criteria. Previous studies used animal, experimental and physical models as test specimens to show focal and diffuse brain injury association with angular loadings of the head. The reported loadings were however, inconsistent in terms of the magnitude of acceleration-deceleration, angular velocity and associated pulse durations. Recently, sophisticated finite element models of the human head have been developed and subjected to rigorous validations. These models have been applied to understand the local tissue response and correlate to pathological injury. However, the role of the acceleration-deceleration pulse shapes on regional specific and tissue specific strain response in an anatomical realistic head model is not understood. The aim of this study was to investigate effects of different angular acceleration-deceleration loading profiles on global and local brain responses using a detailed validated GHBMC (the Global Human Body Models Consortium) human head model.

Method

A set of different angular motion loading time histories were constructed as a simulation matrix. The loading histories consist of acceleration-only, deceleration-only, acceleration followed by deceleration as well as deceleration followed by acceleration with varying separation time intervals of 0, 10 and 20ms. The angular loading of various forms was applied to the GHBMC human head FE model to produce coronal plane of rotation about the y axis of the head. Maximum principal strain (MPS) magnitude, distribution and cumulative strain damage measure (CSDM) predicted by the model were analyzed for various structures, regions and tissues including cerebral white matter, grey matter, corpus callosum, and brainstem.

Results and discussion

For single phased acceleration or deceleration loading, MPS magnitude experienced in all regions was lower from deceleration-only pulse. The peaking time was much later than the acceleration-only pulse. For acceleration followed by deceleration pulse at various dwell time in between, the two peak MPS were observed in the grey matter and corpus callosum and the peak values did not change with the various duration of dwell time. Whereas MPS in the white matter and brainstem from the loading with 0 dwell time had much lower first peak value than the second peak value. Further, the second peak strain increased with increasing dwell time. For deceleration followed by acceleration loading profile, the first peak MPS was similar between all cases at all brain locations. The second peak MPS increased from 0 to 10 ms dwell time but did not increase for 20ms time interval case. In general, the second peak MPS values were found to be higher than the first peak at all regions. The CSDM results showed that 10ms dwell time led to more volumetric failure followed by 20ms and 0 separation time for all brain regions from all loadings.

Conclusion

Under the same velocity but differing peak and duration of the acceleration or deceleration, the brain strained differently. The acceleration pulse of longer duration and lower magnitude followed by shorter duration and higher magnitude produced greater strains in all regions than other forms of pulse. Corpus callosum was the highly strained structure irrespective of type of loading. MPS increased with increasing dwell time only till some extent after that increased in separation time will reduce strain. The brain strain was sensitive to the shape of the angular acceleration pulse of same angular velocities.