Finite Element Analysis of Traumatic Brain Injury

Chu, YH, Bottlang, M
Legacy Clinical Research & Technology Center, Portland, OR. 503-413-5489, mbottlan@lhs.org

Introduction

In the United States, traumatic brain injury (TBI) has an incidence rate of 95 per 100,000 population, and a mortality rate of 23%[1]. In 1941, Denny-Brown and Russell[2] proposed that the change in momentum from acceleration to deceleration is critical in the genesis of diffuse axonal injury (DAI) during TBI. Despite extensive efforts in computational modeling of DAI[3], this acceleration-deceleration scenario has not been explored by finite element analysis to date.

The objective of the present study is to gain a better understanding of brain injury caused by a sequence of acceleration-deceleration induced shear strains, generated by rotation of the skull. In this study, a finite element model of the brain was created, taking into account the distinct material properties of specific brain region, as well as cerebrospinal fluid (CSF), which surrounds the brain and spinal cord, and is believed to serve as a neuro-protective layer that absorbs shear energy. We hypothesized that an acceleration-deceleration loading regime will induce shear strain concentration in areas, which closely correlate to clinically observed region of DAI.

Materials and Methods

The geometry of a para-sagittal plane of the head was reconstructed from the Visible Human Male data set, and the finite element analysis was proceeded with ANSYS™ (Fig. 1). The nonlinear viscoelastic material properties of the cerebral and cerebellar white matter were assigned with exponential decay shear modulus G_0=39.47KPa, G_∞=17.24KPa and a decay constant t=0.01s[3]. A constant high bulk stiffness of K=1.86GPa was implemented to simulate nearly incompressibility of brain tissue[3]. The material properties of white matter were assumed four times stiffer than those of the cerebral and cerebellar cortex (gray matter) to account for their fibrous structure[4]. Cerebrospinal fluid was modeled as a viscoelastic solid that was assumed 10 times softer than the cerebral white matter. The skull and the cervical vertebrae were modeled as homogeneous linear elastic solids with E=6.5GPa and ν=0.2[3]. To simulate a sever brain injury over an acceleration-deceleration sequence over 40 ms, a 10,000N frontal step load was applied for 10 ms, followed by a dorsal step load of −10,000N, which was applied for 30 ms. Boundary conditions enforced angular rotation of the skull around a hinge joint at the second cervical vertebra (C2). Nonlinear time-dependent transient dynamic analysis was performed to allow for large deformation, while accounting for inertial loading effects.

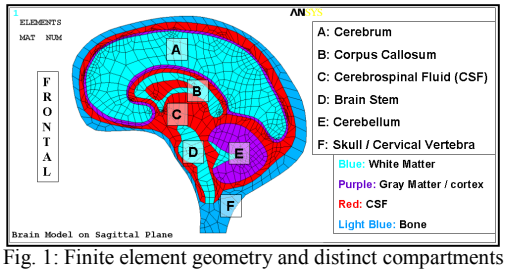

Fig. 1: Finite element geometry and distinct compartments of the brain.

Results

Throughout the loading regime, CSF acted as a protective layer for brain tissue and sustained maximum shear strains of up to 500%. During acceleration (*Phase 1*), among brain compartments, the corpus callosum exhibited the highest nodal shear strain of up to 66% between 7 to 10 ms (Fig. 2a). The inner frontal cerebral white matter and the dorsal cerebrum had high focal shear strain. During the first 10 ms of deceleration (*phase 2*), shear strain distributions changed dramatically, indicating high shear strain rates due to inertial effects (Fig. 2b). Maximum shear strains reached peak values around t = 20 ms for all brain compartments. At t = 20ms, a contrecoup effect was noticeable in both, the cerebral cortex and the white matter of the frontal lobe, reaching a maximum nodal shear strain of up to 126%. Between t = 20 to 30 ms (*phase 3*), shear strain decreased in all brain compartments by up to 57% and in the surrounding CSF by up to 65%(Fig. 2c) as compared to peak values during *Phase 2*. Focalized shear strain in CSF at the mid-brain persisted. With continuous increase of inertial energy during the final 10 ms (*phase 4*), the resulting shear strain distributions were comparable to those observed during *phase 2*. The entire acceleration-deceleration scenarios ended at t = 40ms (Fig. 2d).

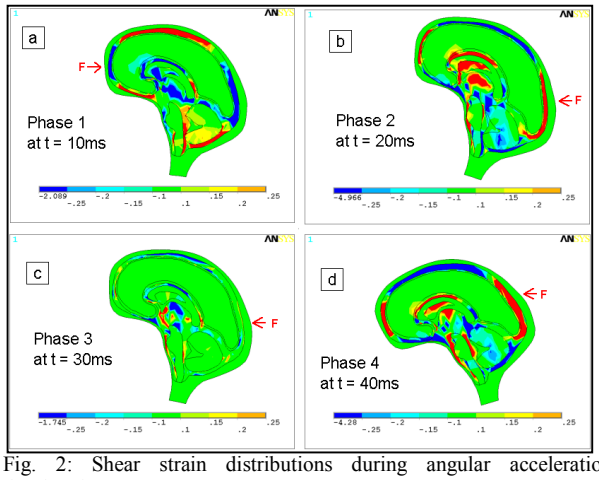

Fig. 2: Shear strain distributions during angular acceleration-deceleration sequence.

Discussion

The results of this study demonstrated that CSF reduced the shear strain to the brain by absorbing shear strain energy. In 1989, Adams[5] introduced a grading system for DAI, indicating *Grade 1* DAI as axonal damage limited to white matter; *Grade 2* DAI as focal lesion in the corpus callosum in addition to *Grade 1*; and *Grade 3* DAI as focal lesion in the brain stem in addition to *Grade 2*. In this study, high shear strain in the white matter of the frontal and dorsal cerebrum during *phase 1* correlated with *Grade 1* DAI. In *phase 2*, the focal shear strain in the anterior corpus callosum and inner frontal lobe of cerebrum were indicative for *Grade 2* axonal injury. High shear strain in the peripheral brain stem correlates with *Grade 3* DAI. Results in this study confirmed the hypothesis, that an acceleration-deceleration sequence can effectively simulate clinically observed coup/contrecoup and *Grade 1* through *Grade 3* axonal injury phenomena.

The 10,000 N focal impact load and time history realistically resembled conditions during vehicle accidents which are associated with mild to sever DAI[6]. Several factors deserve consideration when interpreting the results contained in this study. This finite element analysis did not simulate a sliding interface between the CSF and the brain. Furthermore, this 2D brain injury model can not realistically represent the 3D scenario. Despite these limitations, the results of this study closely resembled clinically observed shear strain distributions during an angular acceleration-deceleration sequence.

Acknowledgement

This study was supported by the Foundation of Legacy Health System.

References

1. TBI incidence rate, risk factors, and causes from 12 states in the US during 1995-1996. In: National Center for Injury Prevention and Control.
2. Denny-Brown, D., Russel, W.R., 1941. Experimental Cerebral Concussion. *Brain*, 64, 93-164.
3. Bandak, F.A., 1995. On the Mechanics of Impact Neurotrauma: A Review and Critical Synthesis. *J. Neurotrauma*, 12, 635-649.
4. Cotter, C.S., Smilarkiewicz, P.K; Szczyrba, I.N. On Mechanisms of Diffuse Axonal Injuries. 2001 Bioengineering Conference, ASME 2001. 315-316.
5. Adams, J.H., Doyle, D., Ford, I., 1989. Diffuse axonal injury: Definition, diagnosis and grading. *Histopathology*, 15, 49.
6. Varney, R.N., Roberts, R.J., 1999. Forces and Accelerations in Car Accidents and Resultant Brain Injuries. In: The Evaluation and Treatment of Mild Traumatic Brain Injury. Varney, N.R., Roberts, R.J.(ed.) Lawrence Erlbaum Assoc., London.