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SIMULATION OF EARLY-TIME HEAD IMPACT LEADING TO TRAUMATIC BRAIN INJURY

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INTRODUCTION

Traumatic brain injury, or TBI, occurs as a consequence of many civilian accident and military combat scenarios. Examples include head impact sustained in automobile accidents as well as blast wave loading from detonated improvised explosive devices. TBI is associated with a loss of functional capability of the brain to perform cognitive and memory tasks, process information, and a variety of motor and coordination deficits. In many instances, the person involved in the event will not experience the full loss of brain function until days or weeks after the event has occurred. This suggests the existence of threshold levels and/or conditions of mechanical stress experienced by the brain that, if exceeded, lead to neural injury and evolving symptoms of TBI in the days or weeks following an accident.

We are developing numerical simulation models of the human head to study a spectrum of impact and blast wave conditions that lead to the onset of TBI. Specifically, we have established collaboration between the University of New Mexico Health Sciences Center and Sandia National Laboratories in order to create accurate models of the various tissue and geometries of the human head. With these models, we are conducting simulations of head impact that will allow us to establish a correlation between the incipient levels, rates, and duration of stress experienced by the brain and the onset of TBI.

This article presents the results of an initial scoping study to simulate the early-time wave interactions within the human head as a result of impact of an unrestrained automobile passenger with the windshield in a 34 mph collision into a stationary barrier. Our head model was created by importing a digitally processed, computed tomography (CT) scan of a healthy female head into the material definition package of the shock physics hydrocode CTH (Hertel, et al., 1993). The CT scan was digitally processed to segment all soft tissue and bone into three distinct materials: skull, brain, and cerebral spinal fluid (CSF). Preliminary constitutive models were formulated for the skull, brain, CSF, and windshield. The simulations were run on a parallel architecture computer employing 64 processors for each simulation. The results of our simulations demonstrate the complexities of the wave interactions that occur between the skull, brain, and CSF as a result of the frontal impact with the glass windshield. These wave interactions result in the formation of localized regions within the brain that experience significant levels of pressure and deviatoric (shearing) stress.

Previous work focusing on the simulation of head impact leading to TBI includes that of Horgan, Gilchrist, and O'Donoghue (2000, 2001, 2003, 2004), Nishimoto and Murakami (2000), Suh, et al. (2005), Willinger, et al. (1999, 2003), Zhang, et al. (2001), Park and Yoon (1997), Kleiven and von Holst (2002), and Bandak, et al. (1995). The present work distinguishes itself by employing digitized CT scan data for head definition, sophisticated constitutive models for the biological materials, and a wave physics hydrocode with which to conduct the head impact simulations. Furthermore, the present work focuses on the early time (~1 msec) wave action within the skull, brain, and CSF in order to investigate the connection between stress wave focusing and ensuing brain injury due to localized tissue damage.

METHODOLOGY

To conduct simulations of head impact, the CTH hydrocode requires material geometry definition and constitutive properties for each material. The material geometry for the human head model was generated by digitally processing a CT scan such that the various biological materials comprising the head were segmented into skull, brain, and cerebral spinal fluid (CSF). Although the brain consists of a variety of tissue types (e.g., gray and white matter), delineated into compartments by the falx and tentorium, it was our intention to start with a simplified representation to conduct scoping calculations. We expect to refine these models at a later date. Consequently, our head model consists of homogeneous brain tissue possessing ventricular space, filled with CSF, and encapsulated by the skull.

The CT scan data consisted of a stack of two-dimensional image planes perpendicular to the longitudinal axis of the head and body. The resolution of the CT scan was such that it provided high resolution within each axial plane but low resolution between planes. Consequently, the material geometry for our head model occupies a three-dimensional mesh consisting of a stack of 51 planes possessing 512 cells per side leading to a total of roughly 13.4 million computational cells per calculation. The simulations also contained a glass plate, representing the windshield, positioned for frontal head impact.

Constitutive property definition for the various materials required the provision of an equation-of-state to describe pressure-volume response, a strength model for the elastic & inelastic deviatoric (shear) response, and a fracture model representing failure behavior. Specifically, we assigned model representations for the skull (bone), brain, CSF, and windshield glass. Details on this aspect of the work can be found in Taylor and Ford (2006).

SIMULATION

In various discussions on the nature of brain trauma, it became evident to us that distinct cell damage mechanisms can occur, depending on whether the cells are subjected to pressure or shearing stress. Specifically, pressure imposes a volumetric change in the cell that can damage its internal structure. Shearing stress, on the other hand, tends to act as a

tearing mechanism damaging cell walls. Both of these mechanisms are at play in most incidents leading to TBI. As such, we present the results of our simulations by inspecting the pressure and the shear stress distributions separately.

Pressure Results

Examination of the pressure results of our simulations suggests the classic coup-contrecoup insult to the head. That is, the frontal lobes of the brain experience significant compressive pressure as the shock wave propagates into the brain from the impact with the glass. This short-duration (0.2 msec) compressive wave propagates through the brain and reflects off of the skull at the rear of the head generating a tensile pressure wave which then propagates back into the brain. In particular, the frontal lobes sustain 25-30 bars of compressive pressure over a period of 0.1 msec early in the impact. The wave reflection off the rear portion of skull results in a tensile pressure of 3-4 bars in the occipital region of the brain over a time period of approximately 0.1 msec. These results can be seen in Figures 1-4 which display the sagittal and axial views of the compressive pressure in the frontal region (Figures 1 & 2) and tensile pressure in the occipital region (Figures 3 & 4). These figures also display the ventricles containing CSF in the central brain region and the skull, appearing as a shell surrounding the brain. Our simulations also predict the presence of elevated pressure at the brain/skull and brain/ventricle interfaces. This phenomenon is due to the shock impedance mismatch existing between different materials within the head, i.e., brain, skull, and CSF. This effect is more apparent in the plots of deviatoric stress, presented next.

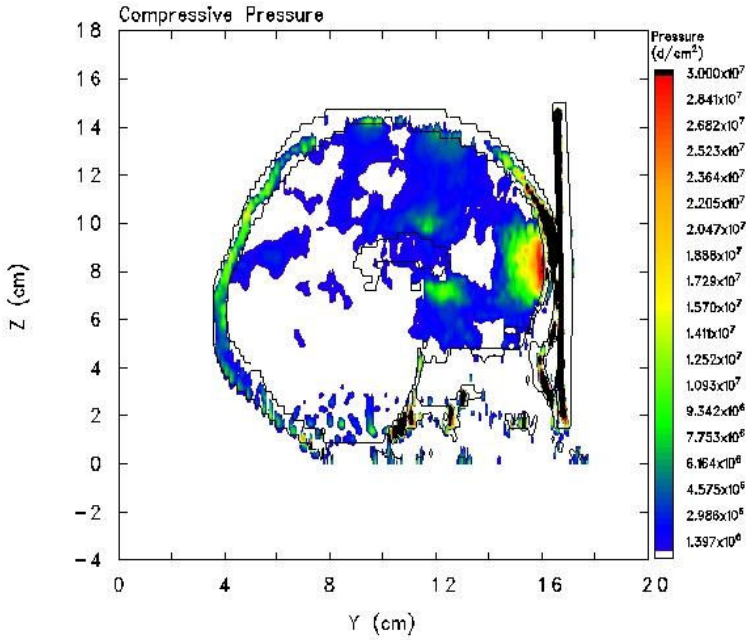

Figure 1. Compressive pressure; sagittal view (glass at right); pressure scale: red: 30 bars, blue: 1 bar.

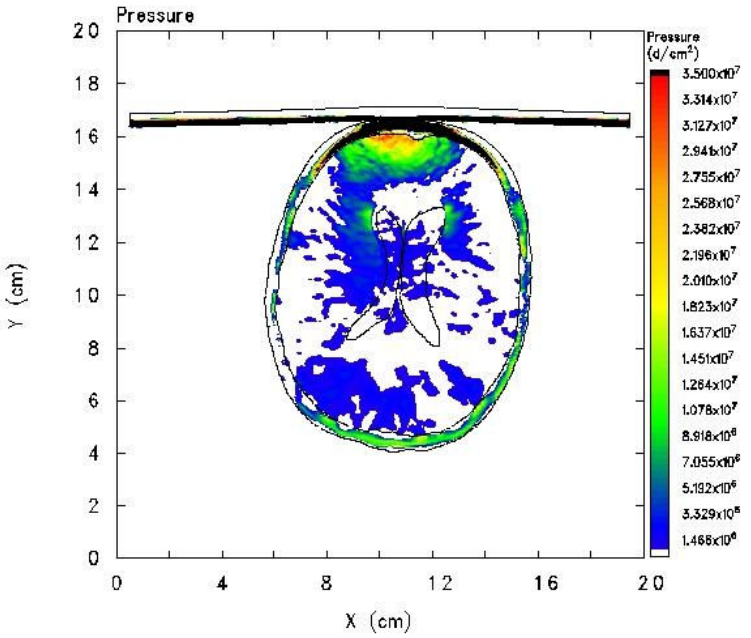

Figure 2. Compressive pressure; axial view (glass at top); pressure scale: red: 35 bars, blue: 1 bar.

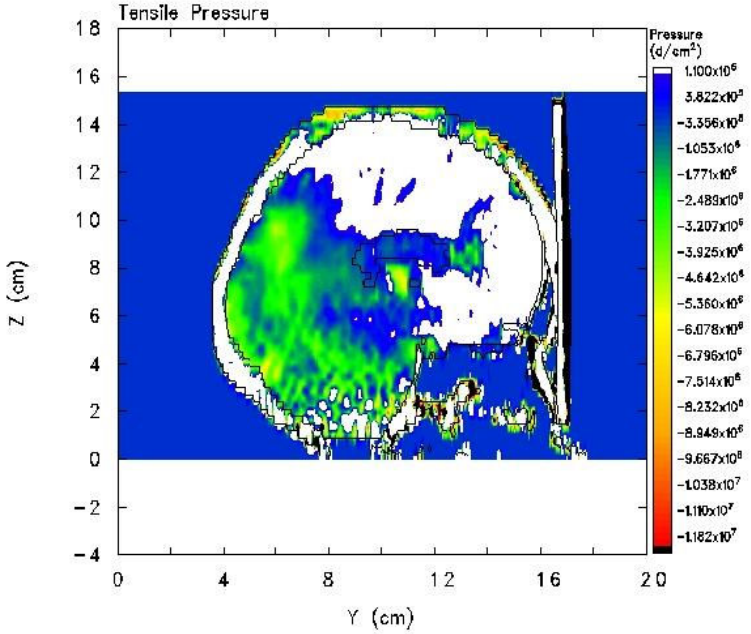

Figure 3. Tensile pressure; sagittal view (glass at right); pressure scale: red: 12 bars tensile, blue: 1 bar compressive.

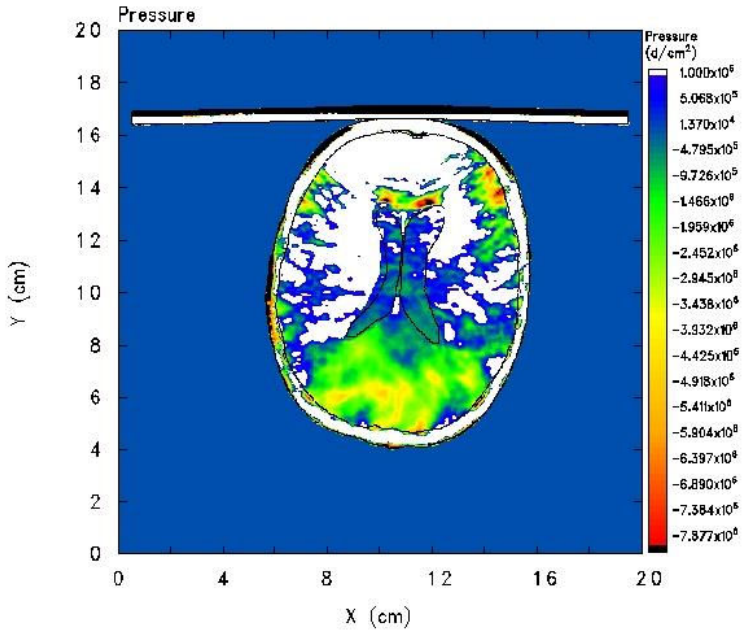

Figure 4. Tensile pressure; axial view (glass at top); pressure scale: red: 8 bars tensile, blue: 1 bar compressive.

Deviatoric (Shearing) Stress Results

Regions of significantly elevated deviatoric stress were also observed at various times in the simulations. These results are displayed in Figures 5 & 6 containing plots of the von Mises stress magnitude, a quantity related to the distortional (shearing) strain experienced by the material. This type of stress results in a tearing action that, for biological materials, could lead to cell membrane damage.

Figure 5 shows the von Mises stress distribution in the sagittal plane with the presence of a large frontal region of the brain experiencing stresses up to 30 bars. Figure 6 provides an axial view of the von Mises stress distribution for the same time (i.e., 0.4 msec) as that depicted in Figure 5. Here, however, one can see the concentration of von Mises stress in the brain in proximity to the forward portions of the ventricles.

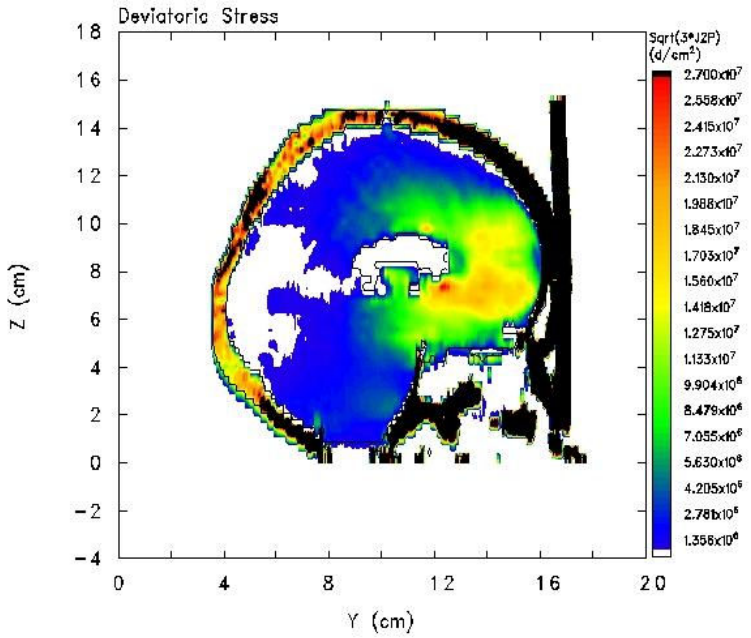

Figure 5. Deviatoric stress; sagittal view (glass at right); von Mises stress scale: red: 27 bars, blue: 1 bar.

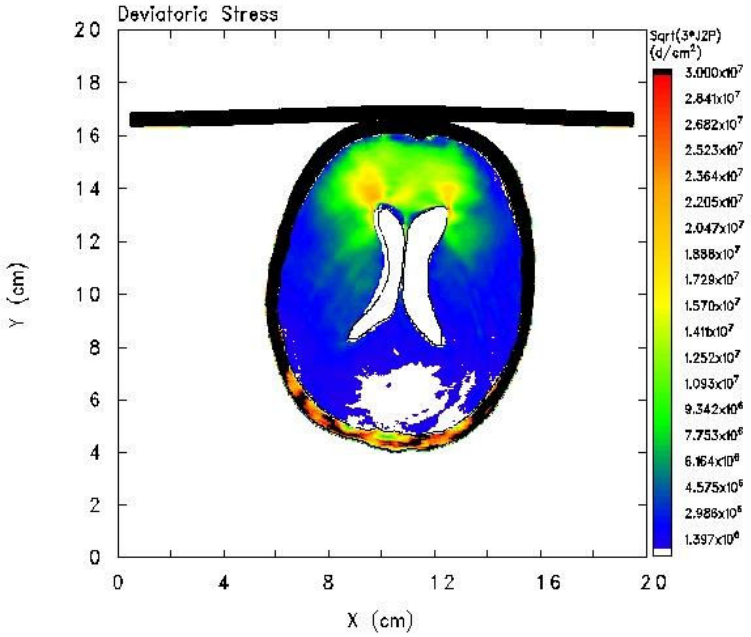

Figure 6. Deviatoric stress; axial view (glass at top); von Mises stress scale: red: 30 bars, blue: 1 bar.

CONCLUSION

To summarize, our head impact simulations predict the classic coup-contrecoup insult when analyzing the pressure history results. This suggests that cell damage associated with this phenomenon is due to changes in cell volume. Furthermore, since the frontal

region of the brain experiences compression whereas the occipital region sustains both compression and tension, it is conceivable that different degrees of damage will occur at these sites depending on the cell's tolerance of compressive versus tensile stress. The simulations also predict focusing of both compressive and tensile pressures at localized sites adjacent to the brain/skull and brain/ventricle interfaces.

The prediction of localized regions of elevated deviatoric stress around the ventricles suggests yet another mechanism and location for damage to occur. As mentioned earlier, deviatoric stresses shear material. In this case, this type of stress could result in the tearing of cellular membranes. Since the brain is principally composed of neurons which conduct electrical impulses along their outer membrane, a tear in this membrane would be synonymous with the loss of electrical conductivity and hence, functionality. Axonal and glial cell injury could also trigger apoptotic mechanisms of axon self-transection, in addition to the direct physical transection by shearing forces. In cases of mild to moderate head injury, the location of elevated deviatoric stresses would depend on the direction and conditions of the impact and might explain why permanent cognitive and behavioral problems occur in one subject but not in another experiencing an injury of similar intensity.

Perhaps the most important conclusion one can draw from these results is that significant levels of stress are generated in the head during the impact event *before* any overall motion of the head occurs in response to the impact. Consequently, researchers studying automobile accident scenarios need to consider the early time wave action occurring within the head in addition to the late time torsional and translational head motion that has been the traditional focus of such studies.

Although we have demonstrated the ability to predict the focusing of stress within the brain due to an impact event, we cannot, as yet, state what specific levels of stress will necessarily lead to TBI. This is a goal of future work, the establishment of a quantitative correlation between the onset of TBI and the level, loading rate, and duration of pressure and deviatoric stress within the brain. This would, in turn, lead to a threshold criterion that could be used in the study of other insult scenarios to the head and the mitigation of critical conditions leading to the onset of TBI (e.g., protective headgear).

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