ON THE ROLE OF THE BRAIN'S GEOMETRY IN CLOSED HEAD INJURIES

Martin Burtscher (1), Igor Szczyrba (2)

(1) School of Electrical and Computer Engineering
Cornell University
Ithaca, NY

(2) Department of Mathematical Sciences
University of Northern Colorado
Greeley, CO

INTRODUCTION

The two most frequently used predictors for developing tolerance criteria for Closed Head Injuries (CHI) are (i) the distribution of the strain field in the brain matter [1] and (ii) the distribution of the intracranial pressure [2]. Although it is widely accepted that the fluidity of the brain tissue should be taken into account when modeling brain injuries (e.g., [3]), recent experiments with piglets show pressure to be an inferior predictor of brain injuries in comparison to strain [4].

Our study on the role of the brain's geometry in CHI provides an explanation why pressure might be an inadequate predictor. The results are based on a nonlinear, viscoelastic, (incompressible) fluid model that generalizes the linear, viscoelastic, solid Kelvin-Voigt model. To describe the fluidity of the brain, we replace the linear, temporal, partial derivative with the nonlinear, temporal (material) derivative and add pressure and hydrostatic compression terms [5]. The basic tenet of our approach is that nonlinear wave phenomena can explain the origin and features of CHI. Such phenomena arise when the material velocity V of the tissue is comparable to or exceeds the brain shear-wave velocity c. The nonlinear effects can induce large values of strain and consequently damage veins and/or neurons.

We showed previously (see [5,6] and references therein) that when a head rotation induces V that exceeds c, a noncircular brain cross-section suffices for nonlinear phenomena to appear. We also showed that even if V induced in the gray matter is smaller than c in the gray matter, a deviation from circular symmetry combined with the differences in the physical properties of the gray and the white matter (or of the white matter and the cerebral fluid in the ventricles) can explain why Diffuse Axonal Injuries (DAI) are created in the white matter near the border with the gray matter (or the ventricles).

NUMERICAL SIMULATION SETUP AND MAIN HYPOTHESIS

We improved our numerical solver to allow us to model a realistic 3D brain geometry and investigate its role in leading to nonlinear phenomena in the 'flow' of the brain tissue. For the purpose of this study, we simulated rotations of a human brain about an axis through the brain's 'center'. To exclude possible effects due to differences in the physical properties of the various brain substructures, we assumed the brain tissue to uniformly posses the properties of a mixture of the gray and the white matter with the shear-wave velocity c=1.5m/s and viscosity v=0.01m^2/s. Since such properties imply the length of the shortest shear waves to be 0.02m [5], we used a 0.001m grid resolution combined with a 0.00002s time step. This amounted to more than 20,000 calculation nodes within a typical cross-section of a brain.

Our hypothesis is that even if the initial flow of the homogenous tissue (forced by a brain rotation) is slower than c, a realistic brain shape could speed it up so that the material velocity would (at least locally) exceed c at a later time. Consequently, high values of strain could appear leading to vein and/or neuron damage in the gray and/or the white matter (not necessarily near the borders mentioned above). At the same time, following simple physical reasoning, one would expect the amplified material velocity to be accompanied by *a drop and not an increase* in the pressure. Therefore, the intracranial pressure might not be a good predictor for brain injuries.

To verify our hypothesis, we simulated impulsive rotations of a human brain with the maximal rotational velocity m (of any point) smaller than c lasting for 0.04s, i.e., by a 38^o or smaller angle, which is representative for traumatic situations. We decided to keep the angle small since our (as well as the Kelvin-Voigt) model assumes a linear relation between strain and stress, which is adequate for modeling small deformations. To obtain more accurate predictions for large deformations, a nonlinear relation should be used instead [7]. On the other hand, experimental data regarding the 'free interfacial conditions between the brain and skull' [8] imply that brain rotations by relatively small angles can be caused by larger rotations of the head.

As an indicator of potential injuries, we use the norm N of the displacement's Jacoby matrix evaluated relative to the rotation, i.e., the strain matrix norm [5]. We monitored the evolution of the solutions for some time after the forced rotation has stopped to see if the appearing high values of N tend to 'spread' at a later time (as has been observed in some of our previous simulations). Extensions of our model suitable for dealing with large deformations as well as the effects of rotations about an arbitrary axis are being investigated.

RESULTS

Figures 1–3 show the norm values at time t=0.05s over sagittal and horizontal cross-sections of a human brain. The localized lighter spots correspond to high values of N. In each case, the brain is rotated for 0.04s with m=1.3m/s, i.e., by a 33° angle around its axis perpendicular to the cross-section. The high values of N begin to appear at a much earlier time than shown – near the locations where the material velocity exceeds the assumed value c=1.5m/s.

Figure 1 depicts the norm values over a sagittal cross-section after rotating the brain forward. The analogous backward rotation leads to quite different locations of the injury-prone sites (Figure 2).

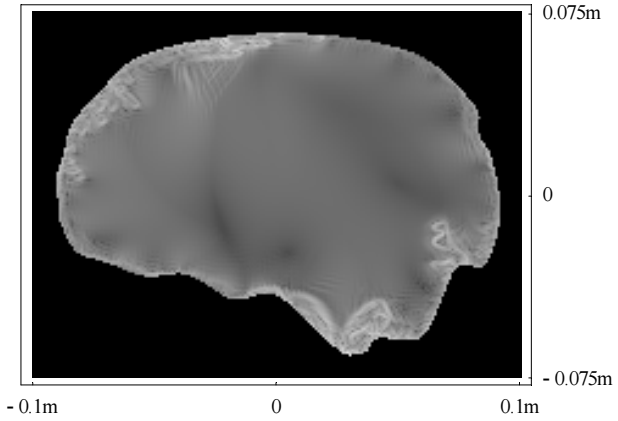

Figure1:
Strain matrix norm 0.01s after a forward rotation.

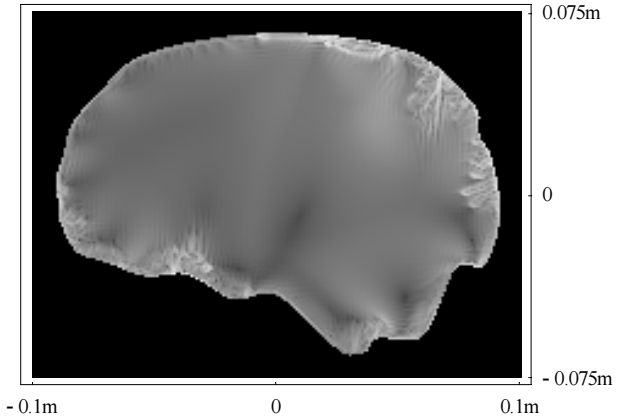

Figure 2:
Strain matrix norm 0.01s after a backward rotation.

Figure 3 shows the values of the norm over horizontal cross-sections of the brain's hemispheres separated (left panel) or not (right panel) by *falx cerebri.* The brain is rotated clockwise. Surprisingly, high values of N appear in very similar locations in the cross-sections. This is due to the fact that in both cases the velocity distributions, while very different initially, become almost identical at time t=0.03s despite the presence or absence of the *falx cerebri*. Consequently, at later times the locations of high values of N are almost identical.

In all our simulations, the high strain values are not accompanied by high pressure. In fact, the pressure is rather uniform in the entire brain during and after the forced rotation.

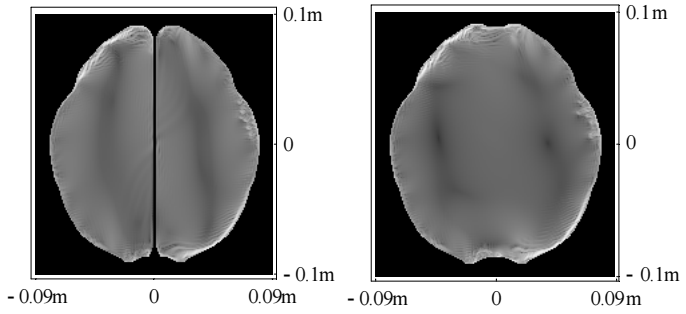

Figure 3:
Strain matrix norm 0.01s after a clockwise rotation; with *falx cerebri* (left), without *falx cerebri* (right).

CONCLUSIONS

Our simulations confirm that the shape of the brain is an important factor for CHI to occur. The brain's geometry is able to amplify the material velocity so that even a relatively slow rotation of the head can lead to nonlinear shear-wave phenomena resulting in high values of the material velocity and the strain in some localized areas.

In general, a 'flattening' of the spherical symmetry or a 'corner' area can cause an increase in the material velocity above the shear-wave velocity in a nearby region, so that high values of strain appear locally. However, a complicated brain shape does not necessarily lead to high strain values. Furthermore, the locations of the injury-prone sites depend strongly on the direction of the rotation.

Our model's predictions, which are based on the distribution of the strain, are in general agreement with clinical and experimental data regarding the locations of vein ruptures and DAI occurrences. Since in our simulations the increased values of strain are not accompanied by an increase in pressure, the latter appears not to be a good predictor for Closed Head Injuries.

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