Effects of CSF Cavitation and Skull Flexure on Blast Induced Traumatic Brain Injury

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Mechanisms of blast induced traumatic brain injury (TBI), particularly the role of primary pressure wave, are still not fully understood. Recent neuropathological analyses of brain tissue from animal tests and post-mortem cases of blast TBI indicate that the brain tissue close to ventricles sustains damage, in contrast to impact TBI [1]. These damages are likely to be produced by large forces concentrated in these regions. The brain tissue and CSF have a very similar volumetric response under positive pressure, but CSF can only bear negative pressures up to its vapour pressure (-100 kPa) [2]. CSF cavitation can create a discontinuous pressure distribution, which may lead to strain concentration at its boundary with brain. Blast pressure wave can also generate skull flexure, causing brain deformations. Here we investigate whether CSF cavitation and skull flexure can produce large strain and strain rate concentrations at the CSF/brain interface.

To test this hypothesis, we developed a two-dimensional human head FE model of blast TBI (Fig 1), composed of skin, skull, CSF, brain and ventricles. The skin and skull were modelled by linear elastic models. Brain was modelled with a viscoelastic material model. To model CSF cavitation, a cut-off pressure was defined for the volumetric response. Once the CSF elements reached the cavitation state, the pressure was kept at the vapour pressure. To investigate the effects of CSF cavitation on brain tissue deformation, CSF material models with and without cut-off pressure were employed. The effect of skull flexure was studied by increasing skull stiffness. We simulated detonation of a TNT charge generating a 12.5 bar incident pressure (Fig 2).

Fig 3 shows that CSF cavitation pressure leads to strain discontinuity at the CSF/brain interface and elevated strain levels in brain. Similar effects can be seen for strain rate distribution (Fig 4), though the discontinuity is not as pronounced as for strain distribution. Fig 5a shows that increasing skull stiffness reduces the strain level within cranium, but it does not have a significant effect on strain distribution as shown in Fig 5b, which has a lower limit for strain contour. Interestingly, increasing skull stiffness did not have a significant influence on the level and distribution of strain rate (Fig 6).

In this study we used a 2D brain model, an approach adopted in previous work [3], which is one of the limitations. We will extend the work by using 3D models. Our results suggest that blast pressure wave can cause CSF cavitation leading to strain concentration in the brain near ventricles and that skull flexure increase the level of strain. These results may have implications for novel ways to better prevent bTBI.

References
[1] Shively S., et al., Lancet Neurology, 2016.
[2] Goeller, J., et al., J neurotrauma, 2012.
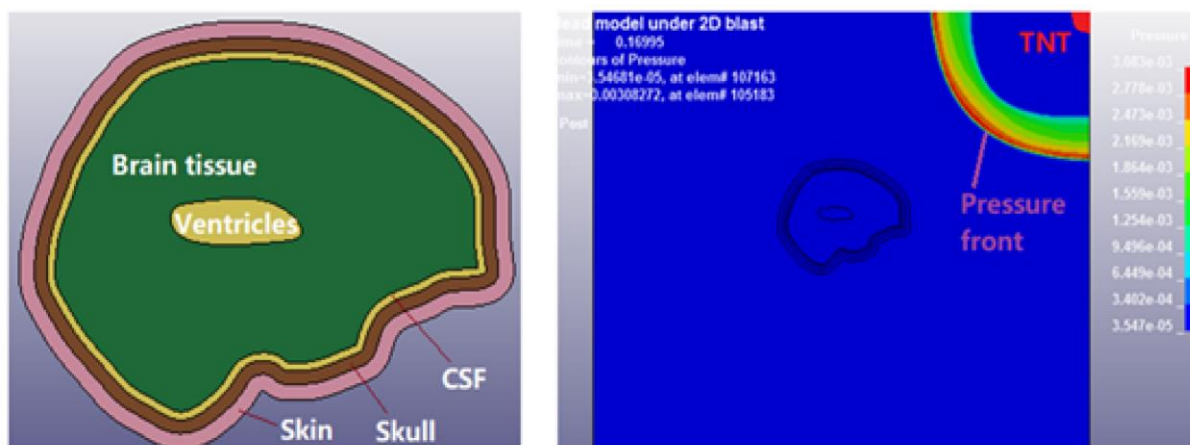[3] Panzer, M.B., et al., Ann of Biomed Eng, 2012.

Fig 2 2D human head FE model and blast loading generation. (In all simulations, the positive-phase duration of the blast wave is 0.2 ms and the incident overpressures are 12.5 atm)
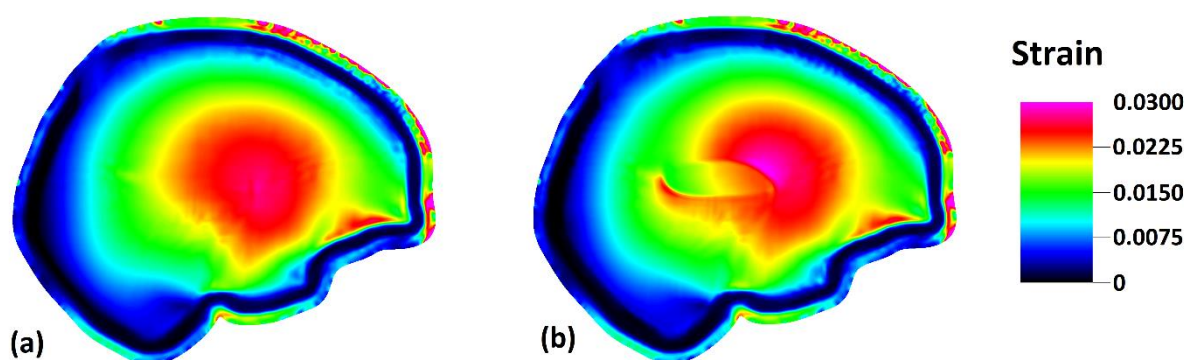

Fig 3 Strain distribution of brain, using CSF models (a) without cut-off pressure and (b) with cut-off pressure
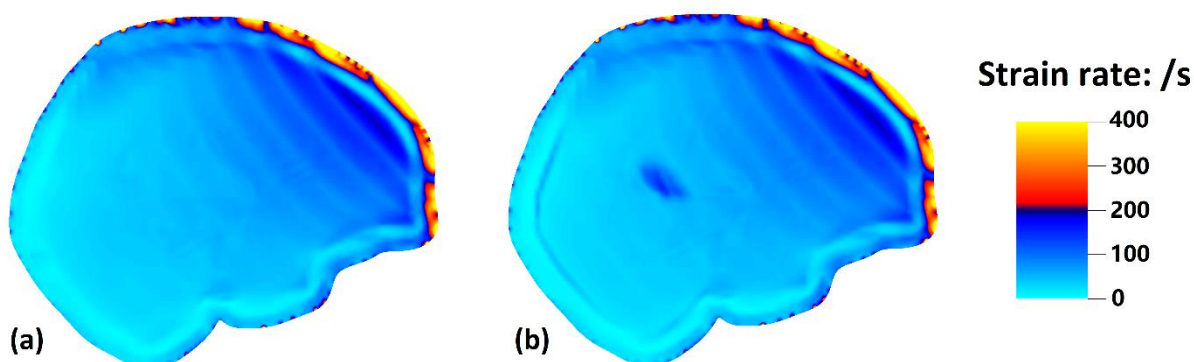

Fig 4 Strain rate distribution of brain, using CSF models (a) without cut-off pressure and (b) with cut-off pressure
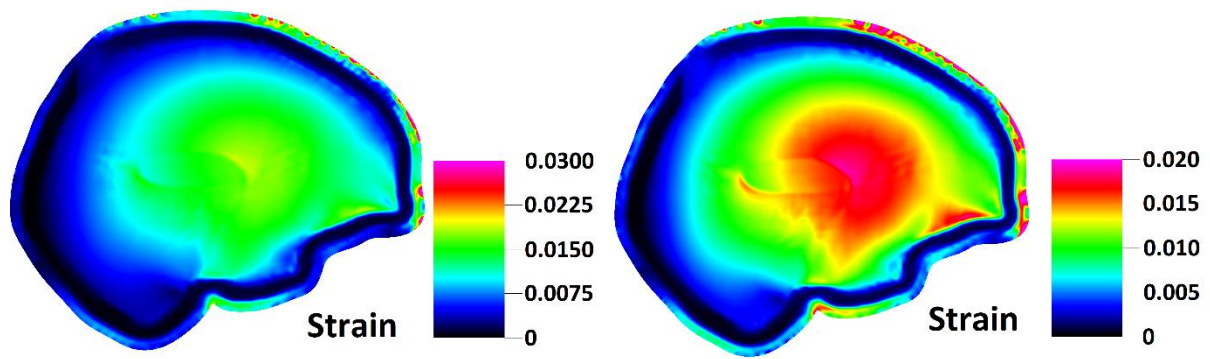
Fig 5 Strain distribution of brain with a 5 times stiffer skull (a) strain contour range: 0-0.03 and (b) strain contour range: 0-0.02
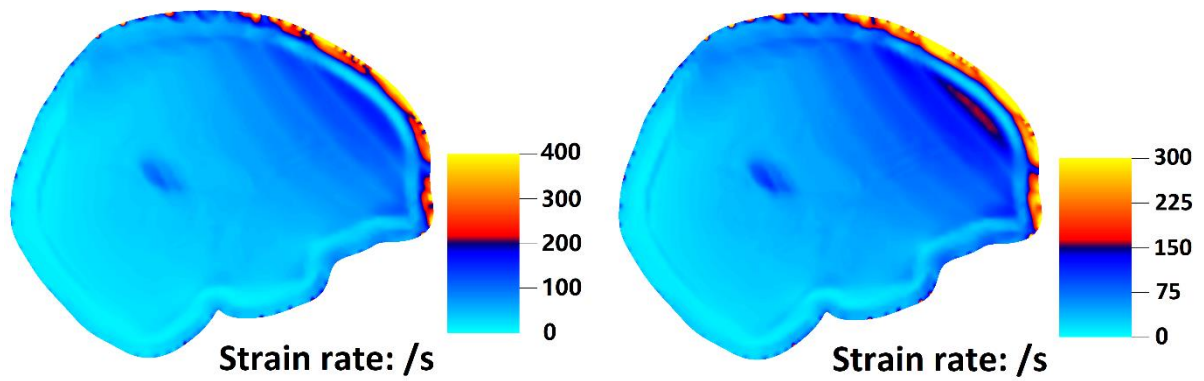

Fig 6 Strain rate distribution with a 5 times stiffer skull (a) strain rate contour range: 0-400/s and (b) strain rate contour range: 0-300/s