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Correlation of Simulation with Clinical Assessments of Blast-Induced Traumatic Brain Injury

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Blast-Induced Traumatic Brain Injury (TBI)

Background

- Closed-Head Blast Injuries are leading cause of traumatic brain injury (TBI) in military personnel returning from combat [1-3]
  - Recent statistics show 267,000 US warfighters sustained TBI
  - 69% as a result of IED blast exposure in Iraq & Afghanistan
- Our Focus: Primary Blast Injury (caused by direct blast exposure)
  - Investigate early-time wave mechanics leading to localized brain injury
- Research Approach:
  - Develop high fidelity digital head-neck model
  - Conduct simulations of blast exposure from various directions
    - Identify specific brain regions experiencing concentrated deposition of wave energy
  - Conduct Clinical Assessment of Blast Victims displaying mild TBI (mTBI)
    - Neuropsychological Testing
    - Magnetic Resonance Image (MRI) assessments of localized brain injury
  - Attempt correlation of simulation predictions of wave physics variables with localized regions of brain injury identified in clinical assessments

[1] Defense & Veterans Brain Injury Center. DoD Worldwide Numbers for TBI | DVBIC.
[2] Fischer, H., 2007, United States Military Casualty Statistics: Operation Iraqi Freedom and Operation Enduring Freedom, Congressional Research Service Report RS22452.
[3] Warden, D., 2006, TBI during the Iraq and Afghanistan Wars, J. Head Trauma Rehab. **21**, 398-402.

TBI Modeling & Simulation
Head-Neck Model

- Finite volume & finite element models developed from Visible Human Project [4] data
  - Constructed from 256 1mm-thick, axial anatomical slices of human male from the VHP
  - Anatomically correct distributions of white & gray brain matter, cerebral spinal fluid, bone, falx & tentorium membranes, muscle/scalp

Full Model Images:

Model Size: 5.9M Cells

Coronal, Axial, & Sagittal Cuts:

[4] National Institutes of Health, 2007, “The Visible Human Project,” National Library of Medicine http://www.nlm.nih.gov/research/visible/visible_human.html

TBI Modeling & Simulation
Constitutive Models

- Biological Materials:
  - White, Gray Matter – Mie-Gruneisen EOS[1], Viscoelastic models [5]
    - M-G EOS being replaced by Tillotson-Brundage Cavitation EOS [6]
  - Cerebral Spinal Fluid (CSF) – Mie-Gruneisen EOS
    - Being replaced by Tillotson-Brundage Cavitation EOS
  - Bone - Linear Elastic model w/ Fracture [5,7]
  - Falx & Tentorium (membranes) –Elastic models [5]
  - Muscle & Scalp - Elastic models [5,8]
  - Sinus Air (and surrounding air) - Non-linear Compressible EOS

[1]EOS – Equation of State: describes volumetric thermomechanical response

[5] Zhang, L., Yang, K.H., & King, A.I., 2001, "Comparison of Brain Responses between Frontal and Lateral Impacts by Finite Element Modeling," J. Neurotrauma **18**(1), pp. 21-30.
[6] Brundage, A. L., 2013, "Prediction of Shock-Induced Cavitation in Water," Proc. 2013 APS Shock Compression of Condensed Matter, Seattle, WA.
[7] Carter, D.R., 1985, "Biomechanics of Bone," Biomechanics of Trauma, Appleton-Century-Crofts, Norwalk, CT, pp. 135-165.
[8] Mak, A.F.T. & Zhang, M., 1998, "Skin and Muscle," in Handbook of Biomaterial Properties, ed. J. Black & G. Hastings, Chapman & Hall, London, pp. 66-69.

Modeling & Simulation

Methodology & Validation

- Simulation Methods
  - Eulerian methods using CTH (w/ finite volume model)
    - Blast, Projectile Penetration
  - Lagrangian methods using Presto (w/ finite element model)
    - Blunt Impact, Imposed kinematic conditions (e.g. acceleration)
  - Lagrangian-Eulerian coupled methods using Presto/CTH (w/ finite element model)
    - Blast (more accurate fluid-structure interactions than Eulerian)
- Head/Neck Model Validation
  - Compared Simulation predictions with laboratory data
    - Magnetic Resonance Tagging & Elastography data on the human head (in vivo) courtesy of Prof. Philip Bayly research team, Washington University at St. Louis, MO USA [9,10]

[9] Sabet A.A., Christoforou E., Zatlin B., Genin, G.M., Bayly, P.V., 2008, "Deformation of the Human Brain by Mild Angular Head Acceleration," J. Biomech., **41**, pp. 307-315.
[10] Feng Y., Abney T.M., Okamoto R.J., Pless R.B., Genin G.M., Bayly P.V., 2010, "Relative Brain Displacement and Deformation during Constrained Mild Frontal Head Impact," J. Roy. Soc. Interface, **7**(53), pp. 1677-1688.

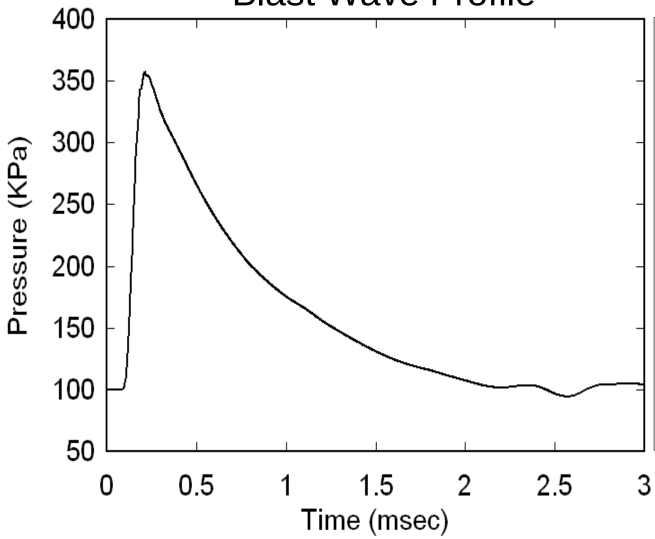
TBI Modeling & Simulation

Example: 3.6 bar (360 KPa) Blast

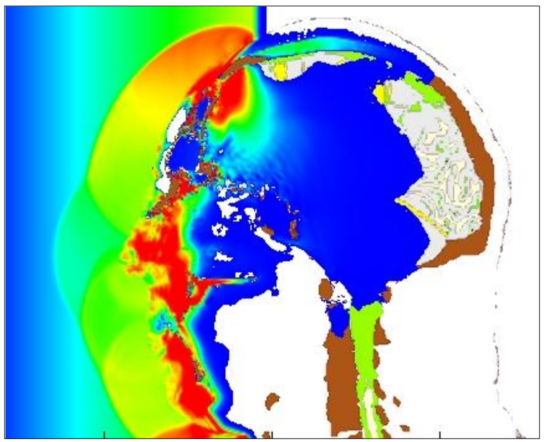
Snap-Shot Images of Blast-Induced Pressure Wave Propagating through Head

Time ~ 130 μs after blast wave encounters head

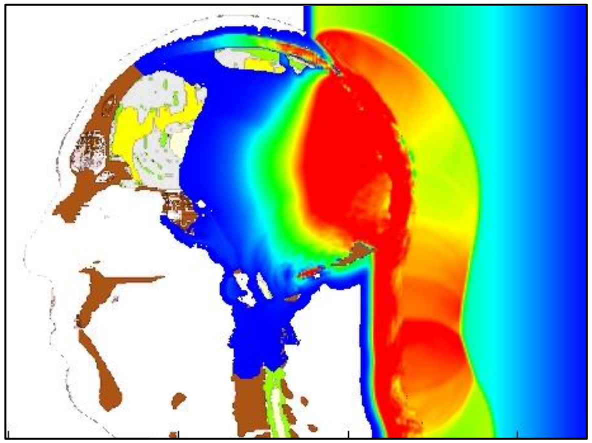
Blast Wave Profile



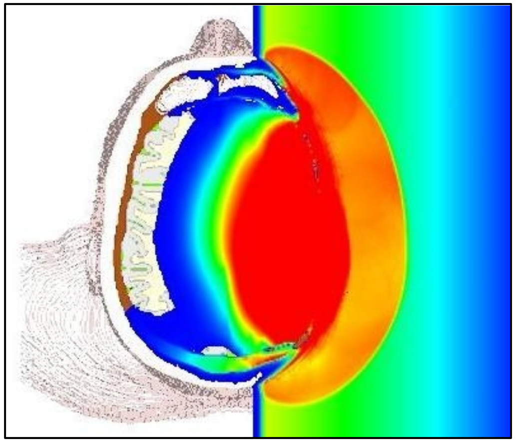
Frontal Blast

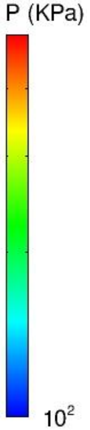


Rear Blast



Side Blast





The Big Question

- Can we correlate predicted wave physics variable(s) with clinically observed indicators of localized brain injury?
  - Stress magnitude extrema
  - Strain magnitude extrema
  - Strain Energy extrema
  - Stress Power
- Our Approach:
- Start by attempting correlation of wave energy extrema with localized injury
  - Energy takes into account both stress magnitude and its associated strain
    - Isotropic Compressive Energy (ICE): associated w/ Crush
    - Isotropic Tensile Energy (ITE): associated w/ Dilatation
    - Deviatoric Shear Energy (DSE): associated with Shear and Tearing

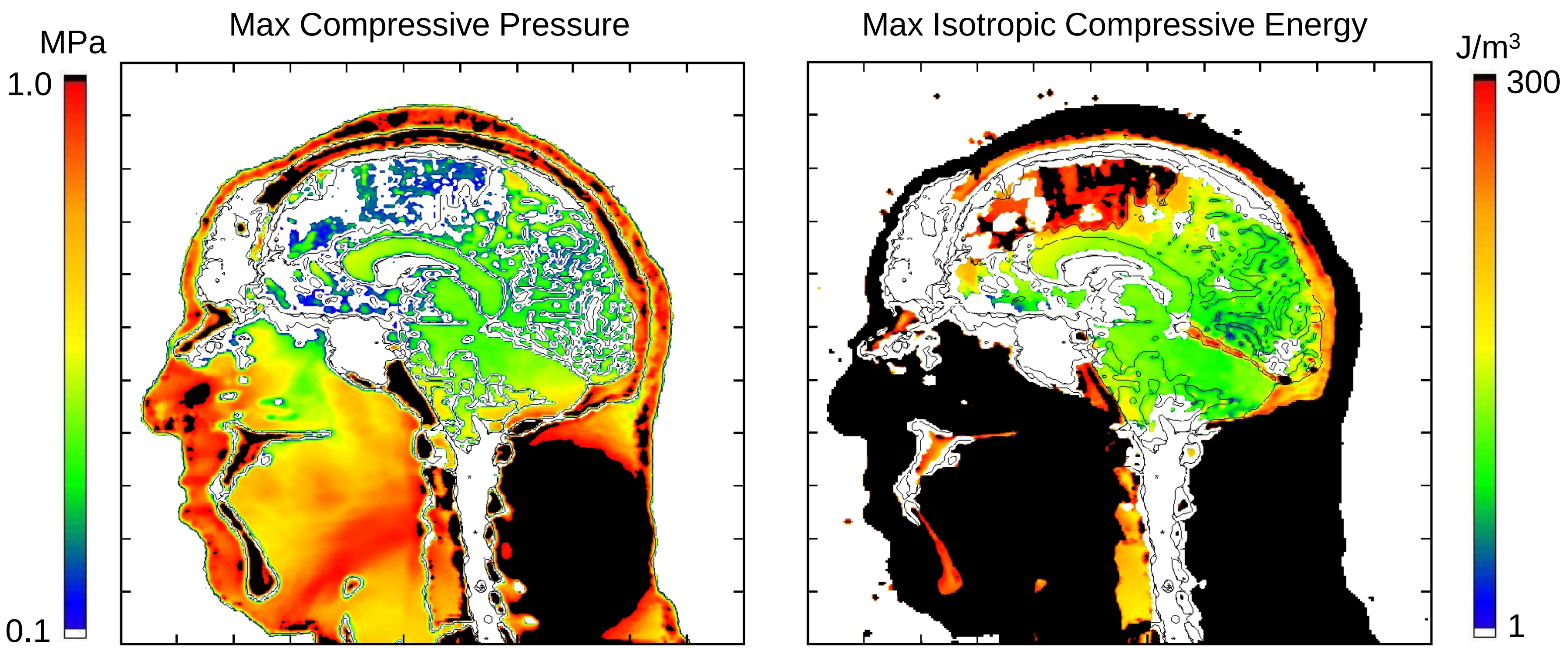
TBI Modeling & Simulation

3.6 bar Frontal Blast Exposure: Compressive Pressure & Energy

Max Pressure & Isotropic Compressive Energy (ICE) associated with Crush

- Dependent on blast direction [11]
- No known correlation with local tissue injury

$$ICE = Pos[\int P \frac{d\rho}{\rho}]$$



[11] Taylor, P., Vakhtin, A., Ford, C., 2013, "Investigation of Blast-Induced Traumatic Brain Injury," submitted to Brain Injury.

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TBI Modeling & Simulation

3.6 bar Frontal Blast Exposure: Tensile Pressure & Energy

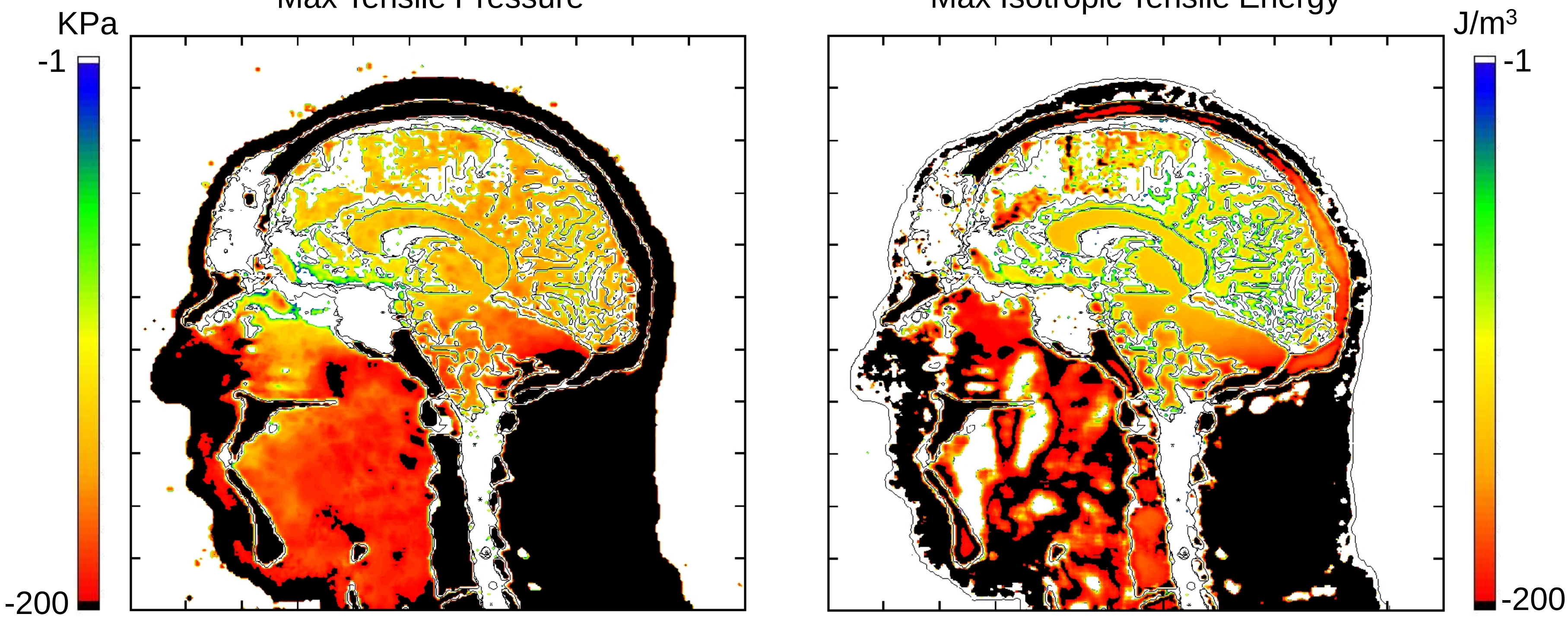
Max Tensile Pressure & Isotropic Tensile Energy (ITE) associated with volumetric Dilatation & possibly Cavitation

- Independent of blast direction [11]
- Suspected tissue injury mechanism

$$ITE = Neg[\int P \frac{d\rho}{\rho}]$$

Max Tensile Pressure

Max Isotropic Tensile Energy



[11] Taylor, P., Vakhtin, A., Ford, C., 2013, "Investigation of Blast-Induced Traumatic Brain Injury," submitted to Brain Injury.

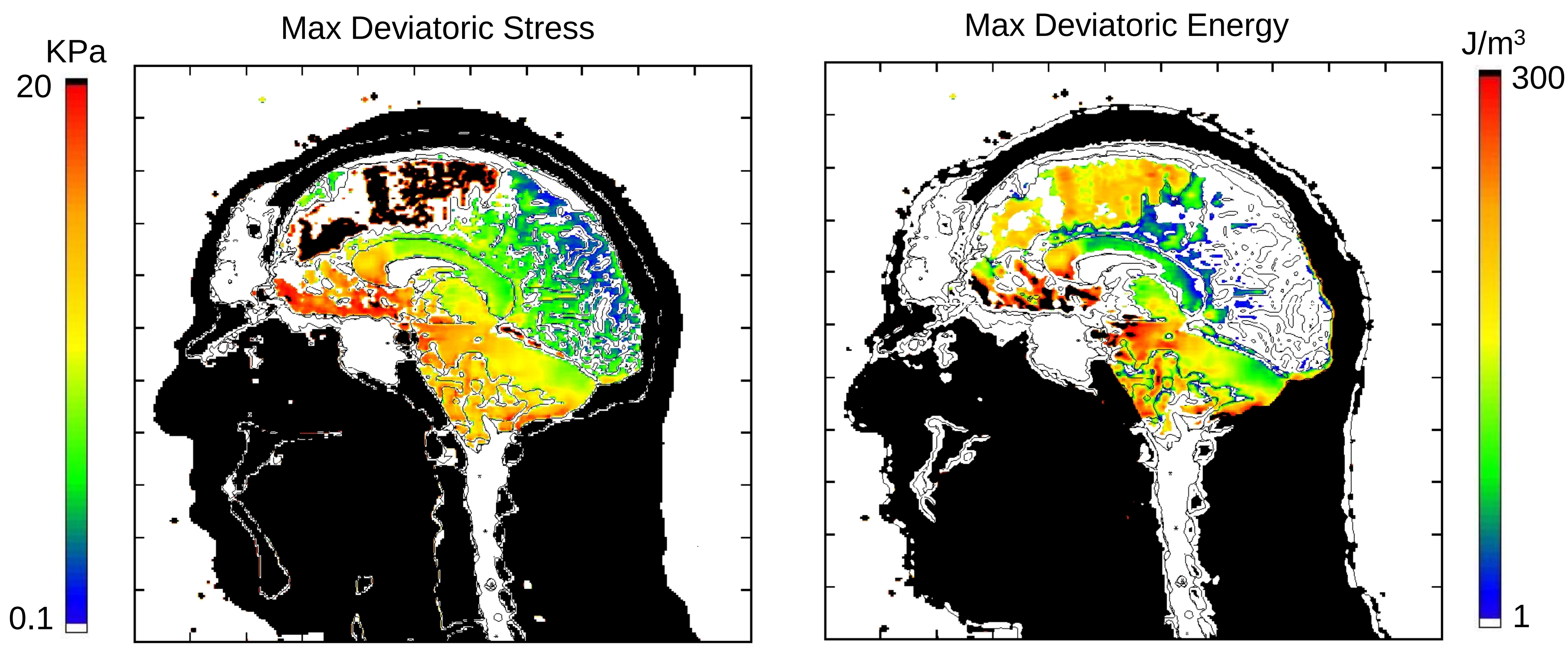
TBI Modeling & Simulation

3.6 bar Frontal Blast Exposure: Deviatoric (Shear) Stress & Energy

Max Deviatoric Stress & Energy (DSE) associated with Shear & Tearing

- Independent of blast direction [11]
- Suspected tissue injury mechanism
  - Cytoskeleton disruption & membrane rupture

$$DSE = \int tr(\mathbf{Sd})\,dt$$



[11] Taylor, P., Vakhtin, A., Ford, C., 2013, “Investigation of Blast-Induced Traumatic Brain Injury,” submitted to Brain Injury.

TBI Clinical Assessment Strategy

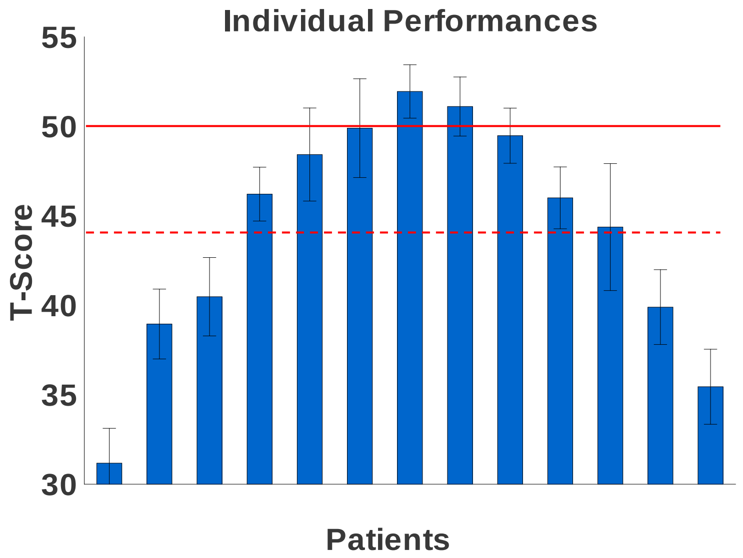
- We recruited & studied a group of 13 blast-injured veterans
- Assessments Conducted [12]:

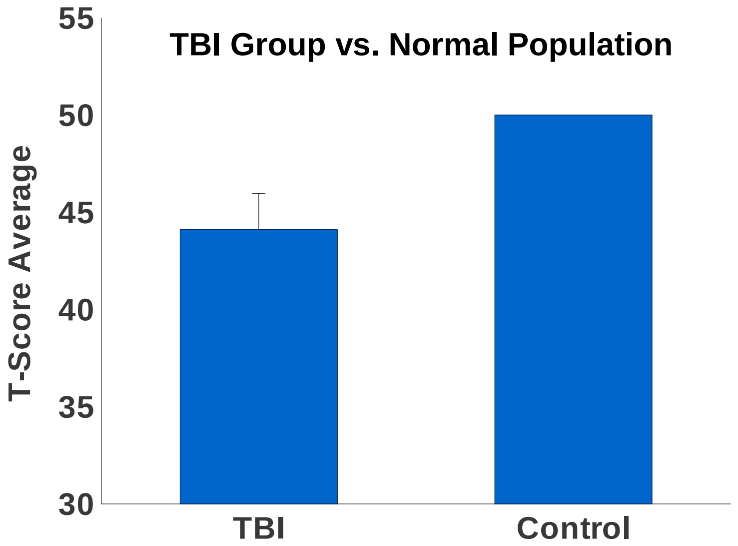
1. Neuropsychological testing (12 tests) to confirm injury and identify domains of impairment – informative
2. High resolution anatomic imaging for macroscopic tissue damage – no tissue damage detected
3. Diffusion Tensor Imaging (DTI) to assess injury to axonal fiber tracts – no detected fiber tract degradation
4. Functional MRI (fMRI) studies of resting state networks for evidence of altered brain activity & functional connectivity – informative

[12] Vakhtin, A., Calhoun, V., Jung, R., Prestopnik, J., Taylor, P., Ford, C., 2013, “Changes in Intrinsic Functional Brain Networks following Blast-Induced Mild Traumatic Brain Injury,” Brain Injury **27**(11), 1304-1310.

Neuropsychological Testing Results

- T-scores averaged across 12 tests for 13 individual TBI subjects (left)
- Gaussian distribution observed (mean score 44)
- Average TBI subjects' T-scores were lower than control population p<0.003
  – Subject group labeled as mild TBI (mTBI)

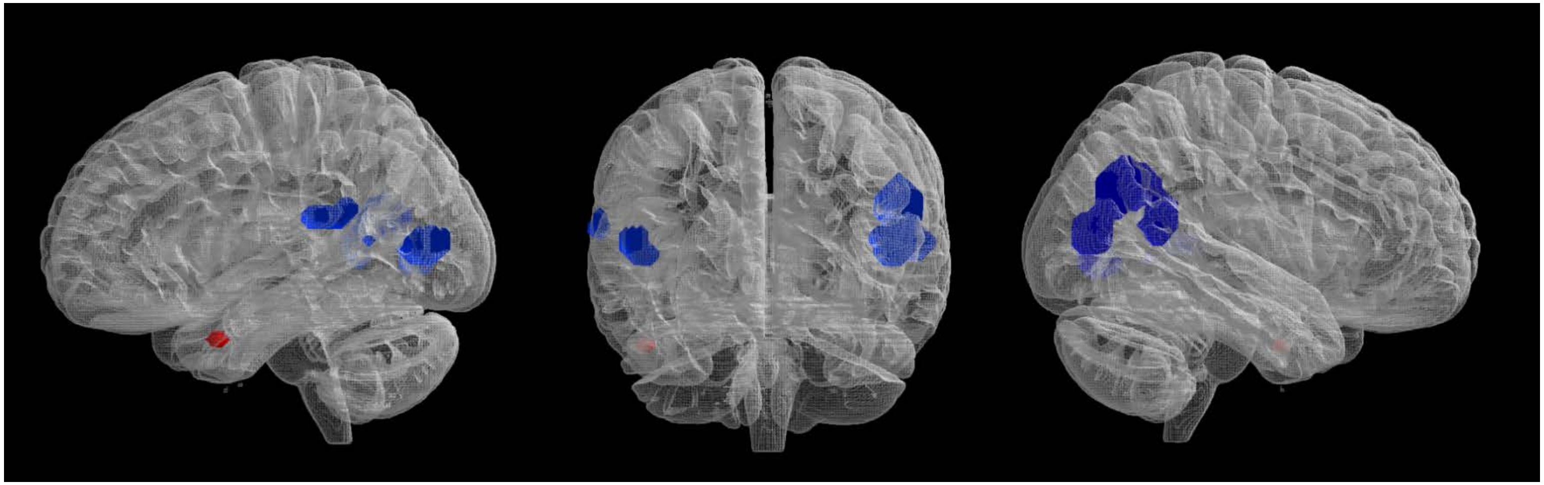




Functional MRI Results

Independent Component Analysis of TBI vs Normal Controls

- Blue & Red areas show regions of TBI brain functioning statistically different ($p<0.05$) from normal controls
  - TBI subject group displayed higher activations in bilateral temporo-parietal regions & lower activation in left inferior temporal lobe
  - Blue == Hyperactive Regions:
    - Visual Network & Attentional Network
  - Red == Hypoactive Region
    - Frontal Network (associated with executive function)
- 6 Functional Network Connections (FNC) impaired vs. Normal Controls:
  - Attentional-Sensorimotor, Attentional-Frontal, Frontal-Default Mode, Default Mode-Basal Ganglia, & Sensorimotor-Sensorimotor (2)

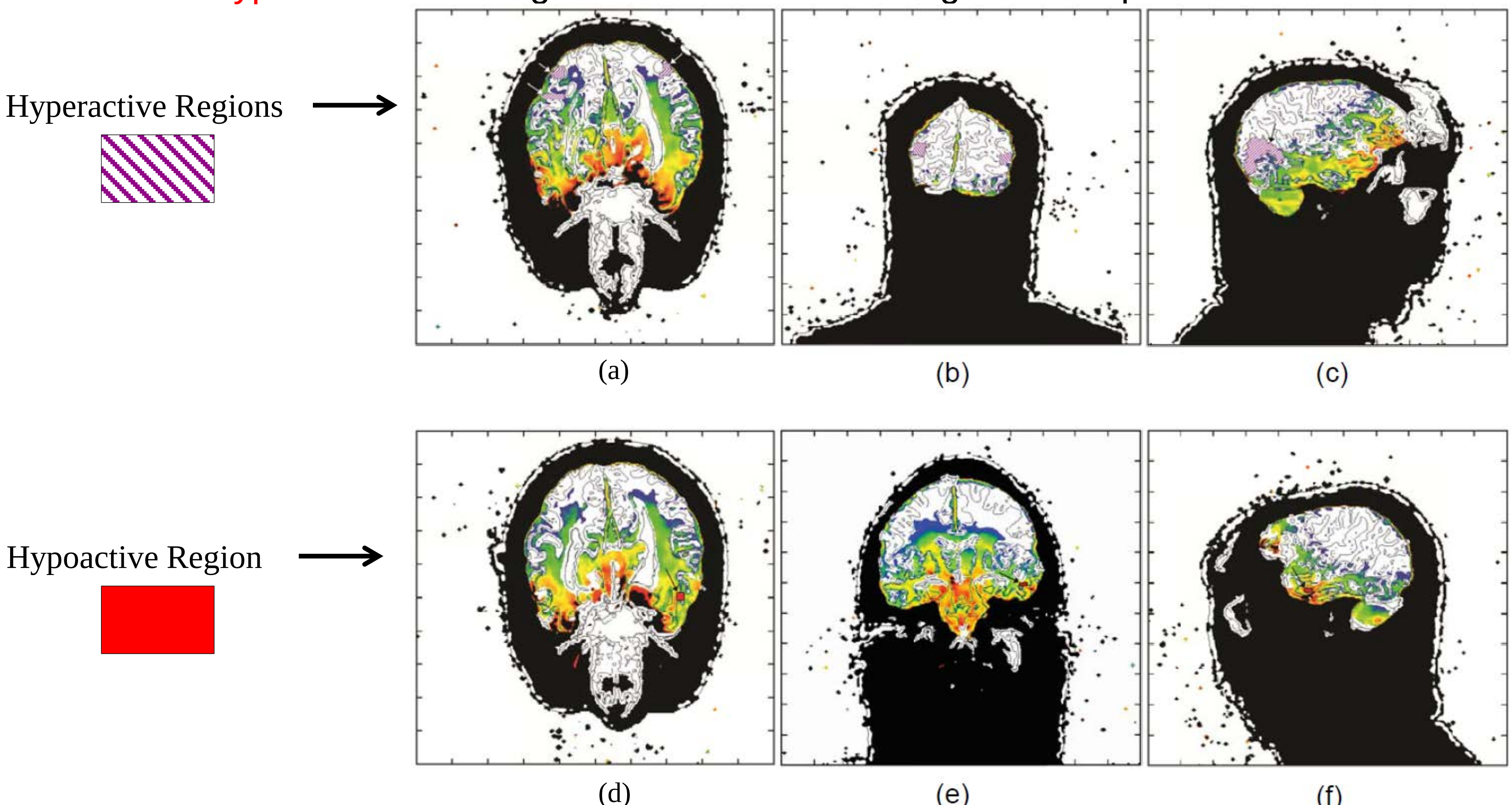


Comparison of Simulation w/ Clinical TBI Data

Blast-Generated Deviatoric (Shear) Energy & fMRI Data

Deviatoric Shear Energy (DSE) deposition correlates with fMRI Results from clinical study of blast-injured veterans displaying mTBI

- fMRI Hyperactive brain regions located in areas of low DSE deposition
- fMRI Hypoactive brain region located in area of high DSE deposition



Summary of Current Results

- Comparison of simulation predictions w/ clinical data on mTBI blast subjects suggests possible correlation between DSE & fMRI data
  - Focused regions of deviatoric shear energy (DSE) overlap with local region of brain hypoactivity in mTBI subjects
    - Left inferior temporal lobe (frontal network; assoc. w/executive function)
  - Hyperactive brain regions reside in locations experiencing low DSE deposition
    - Bilateral temporo-parietal junctions (Visual & Attentional networks)
    - Hyperactive regions compensating for damaged regions

  → DSE deposition appears to correlate with local regions of altered brain activity from blast injury

  - Simulation predictions also show localized regions in brain experiencing elevated levels of tensile pressure and energy

    → Cavitation – a suspected but unconfirmed injury mechanism

Where Do We Go to from Here? Part I

- Extend Present Work
  - Recruit extended sample of subjects displaying symptoms of mild- *and* moderate-TBI from impulsive loading (blast, impact)
    - Expect greater number of Hypoactive Regions in fMRI assessment
    - Axonal injury may also be detectable by Diffusion Tensor Imaging (DTI)
    - Map out and quantify spatial extent of local brain injury
  - Conduct wider spectrum of blast & impact simulations to capture injury scenarios experienced by TBI subjects
  - Attempt further correlation of simulation with clinically identified brain injury
    - Identify complete set of wave physics variables that correlate with clinical DTI & fMRI measures of brain injury
      - Candidate: Isotropic Tensile Energy (ITE) → Dilatation → Cavitation
    - Attempt qualitative and, if possible, quantitative correlation
  - Ideal Goal:
    - Establish a Brain Injury Threshold Criterion
      - Based on threshold values of select wave physics variables that correlate with the onset of localized brain injury

Where Do We Go to from Here? Part II

- Expand M&S Toolset
  - Investigate brain injury from dilatation
    - Recall: simulation predictions showed localized regions experiencing elevated levels of tensile pressure and energy
    - This dilatation may portend the onset of cavitation
  - Cavitation hypothesized to cause local injury leading to TBI [13-16]
    - Collapse of bubbles formed in fluid cause local shock wavelets that damage surrounding tissue
  - Investigate the effects of cavitation on brain tissue injury
    - Verify existence of intracranial cavitation
      - If it exists, model it & attempt to correlate w/ Clinical measures (fMRI, DTI)

[13] Lubock P., Goldsmith W., 1980, "Experimental Cavitation Studies in a Model Head-Neck System, J. Biomech. **13**, pp. 1041-1052.
[14] Brennen C.E., 2003, "Cavitation in Biological and Bioengineering Contexts," Proc. 5th Int. Symp. Cavitation, Osaka, Japan.
[15] Nakagawa A., Fujimura M., Kato K., Okuyama H., Hashimoto T., Takayama K., Tominaga T., 2008, "Shock Wave-Induced Brain Injury in Rat: Novel Traumatic Brain Injury Animal Model, Acta Neurochir. Supp. **102**, pp.421-424.
[16] Taylor P.A., Ludwigsen J.S., Vakhtin A.A., Ford C.C., 2013, "Simulation and Clinical Assessment of Blast-Induced Traumatic Brain Injury," Neurotrauma Letter, submitted.

Questions?