Injury Assessment Tools

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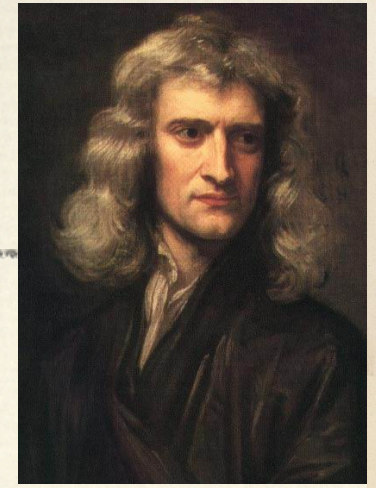
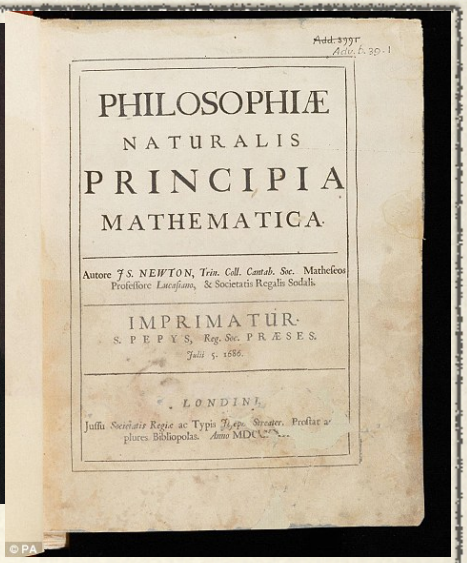
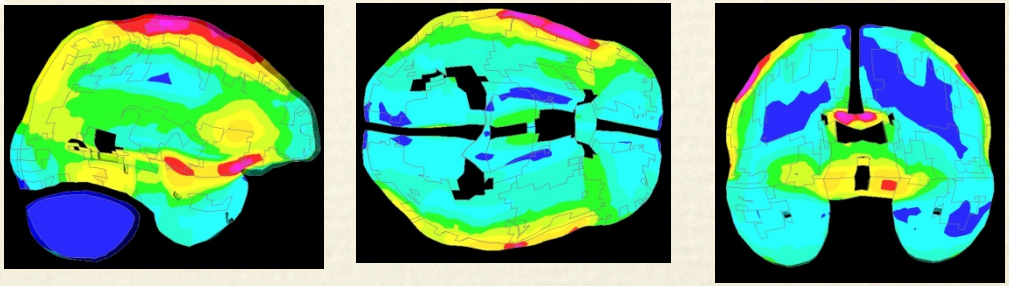
The views expressed here are my own and not necessarily those of DOT

TOOLS





- Newton's laws of motion
- Continuum mechanics
- Uniqueness of the skull-brain-blood-CSF mechanical system
- Material properties of the skull, brain, blood, and CSF
- FE models

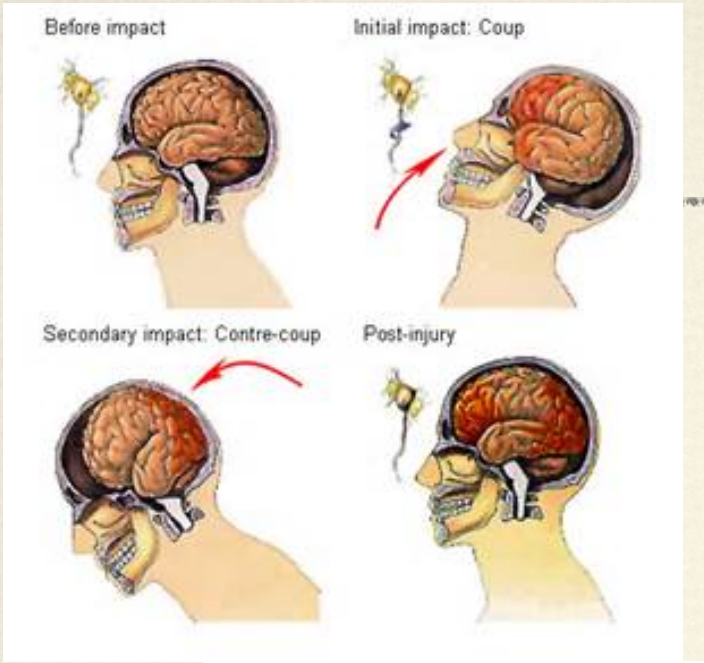


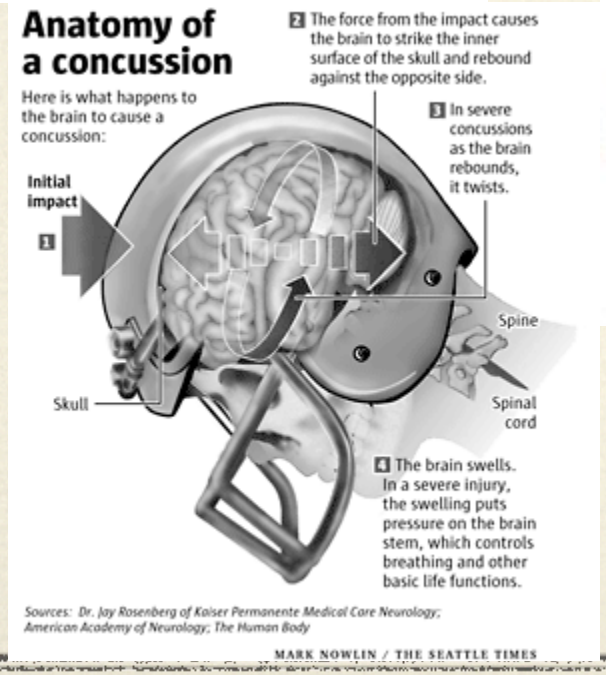
- Common sense (whenever appropriate)

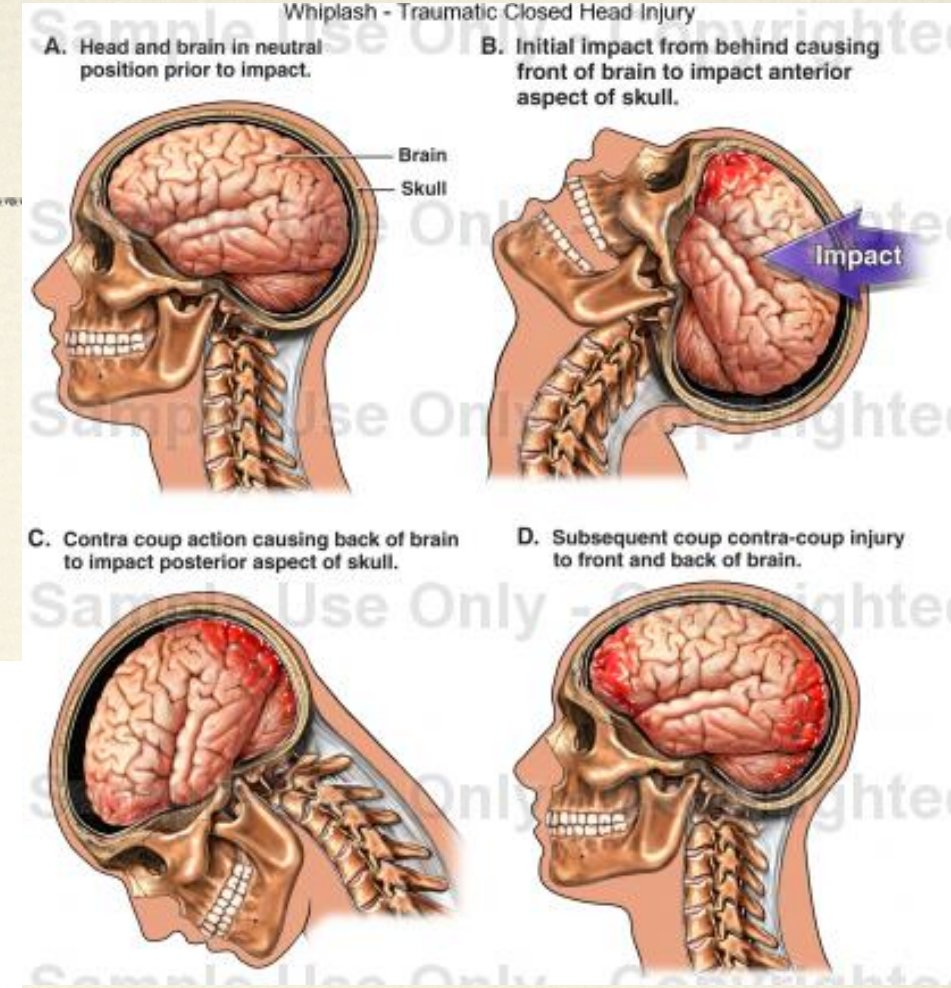
Uniqueness of the skull-brain-blood-CSF mechanical system

Holbourn (1943)

1. Extreme incompressibility of the brain (and blood, CSF), e.g. the brain doesn't change its size when subjected to hydrostatic pressure (bulk modulus ~ 2.07 GPa)
2. Very small shear modulus, e.g. it offers very small resistance to changes in shape compared to resistance to changes in size (shear modulus ~ 1 KPa)
3. Uniform density of the brain, blood and CSF are approximately the same and equal to that of water
4. The stiffness of the skull is much greater than that of the brain
5. The shape of the skull (inner table) and brain are important in deciding the location of injuries


Before impact
Initial impact: Coup
Secondary impact: Contre-coup
Post-injury


Anatomy of a concussion
Here is what happens to the brain to cause a concussion:
Initial impact
1
2 The force from the impact causes the brain to strike the inner surface of the skull and rebound against the opposite side.
3 In severe concussions as the brain rebounds, it twists.
Spine
Skull
Spinal cord
4 The brain swells. In a severe injury, the swelling puts pressure on the brain stem, which controls breathing and other basic life functions.
Sources: Dr. Jay Rosenberg of Kaiser Permanente Medical Care Neurology; American Academy of Neurology; The Human Body
MARK NOWLIN / THE SEATTLE TIMES


Whiplash - Traumatic Closed Head Injury
A. Head and brain in neutral position prior to impact.
B. Initial impact from behind causing front of brain to impact anterior aspect of skull.
Brain
Skull
Impact
C. Contra coup action causing back of brain to impact posterior aspect of skull.
D. Subsequent coup contra-coup injury to front and back of brain.

Where did these come from?

Concussion

- According to Adams (The genuine work of Hippocrates), the term concussion like symptoms could be traced back to Hippocrates. One of the precepts in Hippocrates' *Aphorisms* (circa 415 B.C.) is translated as "*shaking or concussion of the brain produced by any cause inevitably leaves the patient with an instantaneous loss of voice (i.e. unconscious)*".

- Circa 16th century (Capri and Pare) introduced the term *"Commotio Cerebri"* to describe the effects of injuries to the brain without skull fracture.

- 1705. French surgeon Alexis Littre reported to the Royal Academy of Surgery the case of criminal sentenced to be broken on the wheel. To escape the torture, the man has killed himself by rushing across the dungeon (15 feet across) and striking his head against the wall. Examination of the head revealed "no external marks of violence": no fracture, no lesions, etc. This has become one of the most quoted paper and stimulus for more research.

- 1828. Brodie noted that little is known about the motion of the brain during concussive injury, and it (concussion) was regarded as a "phenomenon beyond comprehension".

Concussion

- 1874. Koch and Filehne produced concussion by means of repeated light blows to the head instead of a single severe blow and performed autopsies on all their animals – they did not find any gross lesions. Proposed a mechanisms of concussion to be molecular disturbance inside the cells.

- 1878. Ferrier wrote: "It is supposed that contrecoup occurs by actual concussion of the cerebral mass against the skull". This mechanism can still be found in the literature and some internet sites along with the animations
http://www.bing.com/videos/search?q=concussion+animation+video&adlt=strict&view=detail&mid=B33E9DF1B43675A70303B33E9DF1B43675A70303&rvsmid=A2C422E493545079B464A2C422E493545079B464&fsscr=0&FORM=VDQVAP

- 1892. Miles presents the point of view that symptoms of concussion are due to a profound disturbance of the circulation of the brain, and that it is due to anemia. Based on animal impact tests he observed that concussive symptoms happen so rapidly after the blow (first few seconds) that they "cannot be attributed to lesions, which, from their nature must take some time to be produced"

Concussion

- 1927. Miller. Quote: "In spite of a considerable body of experimental work, a great deal of indefiniteness, not to say confusion, admittedly exists in the actual knowledge of concussion, particularly as to its physiology and pathology". Using impact tests on dogs and cats he concluded that anemia is not a factor in the causation of concussion, but rather is due to direct mechanical action on the brain cells

- 1941. Denny-Brown and Russell defined the word "concussion" as the state of coma, stupor, and confusion following injury to the head, and that it can occur without cerebral lesions. They used pendulum impacts on cats, dogs, and monkeys. Many observations were made: (1) the head has to move to produce concussion (as opposed to being supported on a hard surface), called it acceleration concussion; (2) impact velocity was about 28 fps to produce concussion; (3) sub-threshold blows sometimes resulted in depression of cardiac, vasomotor, and respiratory function for 10-30 seconds, occasionally longer – possible explanation of the knock-out effect in boxing; (4) death from acceleration concussion is due to failure of blood pressure; etc. Overall 21 conclusions.

Back to the basics

What is/are THE Mechanism/s of Brain Injuries?

Pressure? Strain?

Stress Tensor

$$\boldsymbol{\sigma} = \begin{bmatrix} \sigma_{11} & \sigma_{12} & \sigma_{13} \\ \sigma_{21} & \sigma_{22} & \sigma_{23} \\ \sigma_{31} & \sigma_{32} & \sigma_{33} \end{bmatrix}$$

$$\boldsymbol{\sigma} = \underbrace{\boldsymbol{\sigma}_{HYD}}_{\text{Dilatational or Hydrostatic Component}} + \underbrace{\boldsymbol{\sigma}'}_{\text{Deviatoric Component}}$$

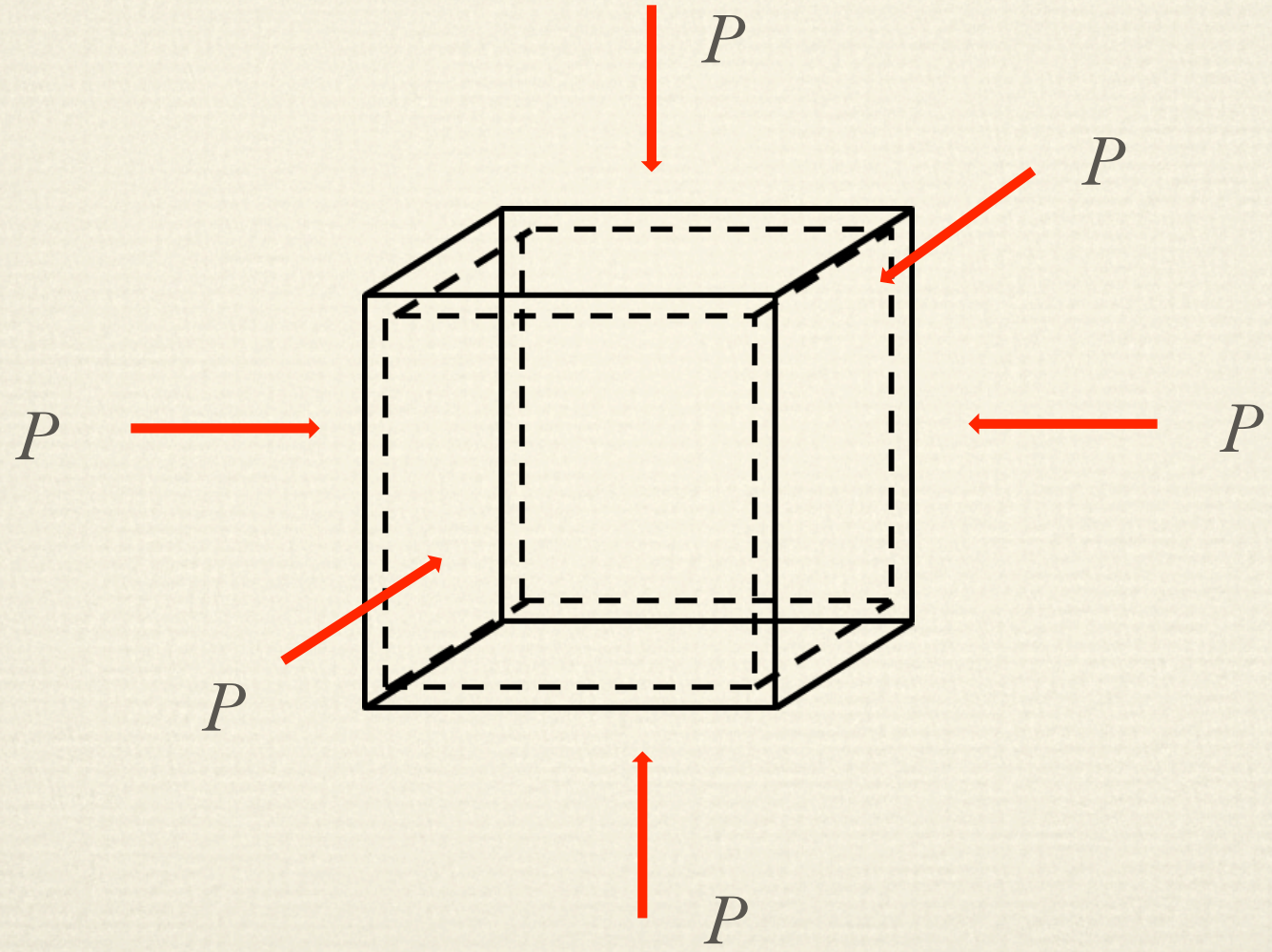
Dilatational/Hydrostatic Component of Stress Tensor: Pressure

$$\sigma_{HYD} = \left(\sigma_{11} + \sigma_{22} + \sigma_{33}\right)/3$$

$$\sigma_{HYD} = \begin{bmatrix} \sigma_{HYD} & 0 & 0 \\ 0 & \sigma_{HYD} & 0 \\ 0 & 0 & \sigma_{HYD} \end{bmatrix}$$

$$P = -\sigma_{HYD} = -\left(\sigma_{11} + \sigma_{22} + \sigma_{33}\right)/3$$

Dilatational/Hydrostatic Component of Stress Tensor: Pressure



Hydrostatic stress/pressure are equal in all directions

They do NOT change under coordinate transformation – invariant

There are NO shear stresses and each direction is a principal direction

Hydrostatic stress/pressure tries to change the Volume of a material and is proportional to the BULK Modulus

The Bulk Modulus of the brain tissue is ~ 2.07 Gpa (McElhaney et al., 1976)

The Bulk Modulus for CSF and Blood is in the same order of magnitude

What if there is pressure gradient?

In this case, the deviatoric component of the stress tensor exists, i.e. it is not equal to zero

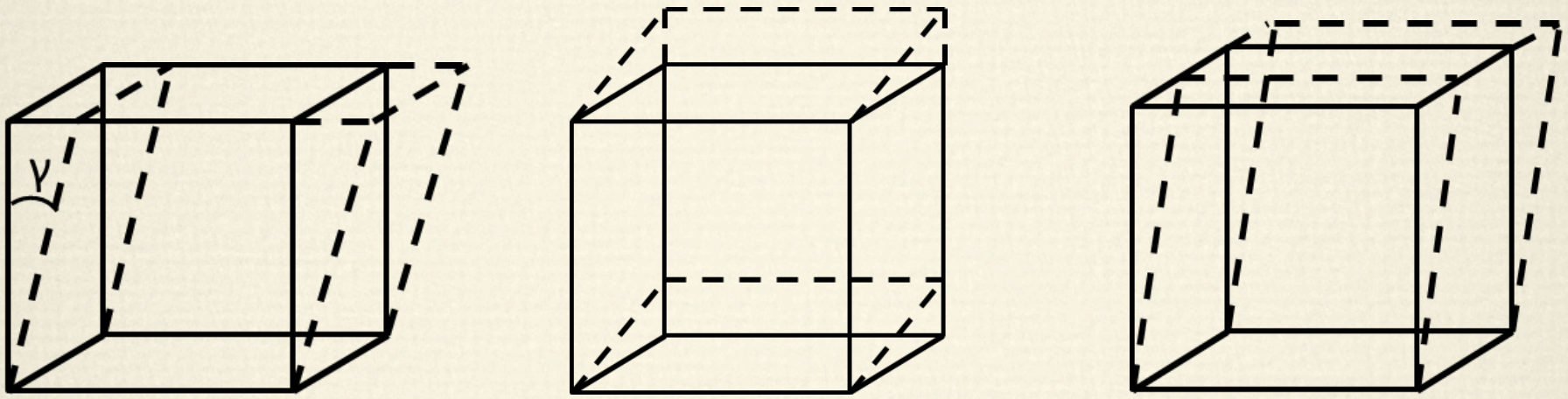
Deviatoric Component of the Stress Tensor

$$\boldsymbol{\sigma}' = \boldsymbol{\sigma} - \boldsymbol{\sigma}_{HYD}$$

$$\boldsymbol{\sigma}' = \begin{bmatrix} \sigma_{11} - \sigma_{HYD} & \sigma_{12} & \sigma_{13} \\ \sigma_{21} & \sigma_{22} - \sigma_{HYD} & \sigma_{23} \\ \sigma_{31} & \sigma_{32} & \sigma_{33} - \sigma_{HYD} \end{bmatrix}$$

$$\boldsymbol{\sigma}' = \begin{bmatrix} \sigma_{11} + P & \sigma_{12} & \sigma_{13} \\ \sigma_{21} & \sigma_{22} + P & \sigma_{23} \\ \sigma_{31} & \sigma_{32} & \sigma_{33} + P \end{bmatrix}$$

Deviatoric Component of the Stress Tensor



Deviatoric stress, when applied to a material, tries to change its shape

Deviatoric stress is traceless (it's first invariant or trace is zero) or hydrostatic stress of deviatoric stress tensor is zero

Deviatoric stress can be formed entirely from shear components, i.e. a coordinate system can be transformed such that only shear components exist

Deviatoric (or shear) stress is proportional to Shear Modulus

Shear Modulus of Brain Tissue is ~ 1 kPa

Mini Summary I

- Bulk Modulus of Brain Tissue is high (brain tissue is virtually incompressible) ~ 2.07 Gpa
- Shear Modulus of Brain Tissue is low ~ 1kPa
- There is ~ 2 Million times difference between the bulk and shear moduli for brain tissue (it is even greater for blood and CSF)
- It is ~ 2 Million times easier to change the shape of the brain than to change its size
- If stress is not hydrostatic, then deviatoric/shear component of stress tensor is non-zero

Stress Tensor

$$\boldsymbol{\sigma} = \begin{bmatrix} \sigma_{11} & \sigma_{12} & \sigma_{13} \\ \sigma_{21} & \sigma_{22} & \sigma_{23} \\ \sigma_{31} & \sigma_{32} & \sigma_{33} \end{bmatrix}$$

$$\boldsymbol{\sigma} = \underbrace{\boldsymbol{\sigma}_{HYD}}_{\text{Dilatational or Hydrostatic Component}} + \underbrace{\boldsymbol{\sigma}'}_{\text{Deviatoric Component}}$$

Does dilatational component of the stress tensor or hydrostatic stress/ pressure cause damage to brain tissue?

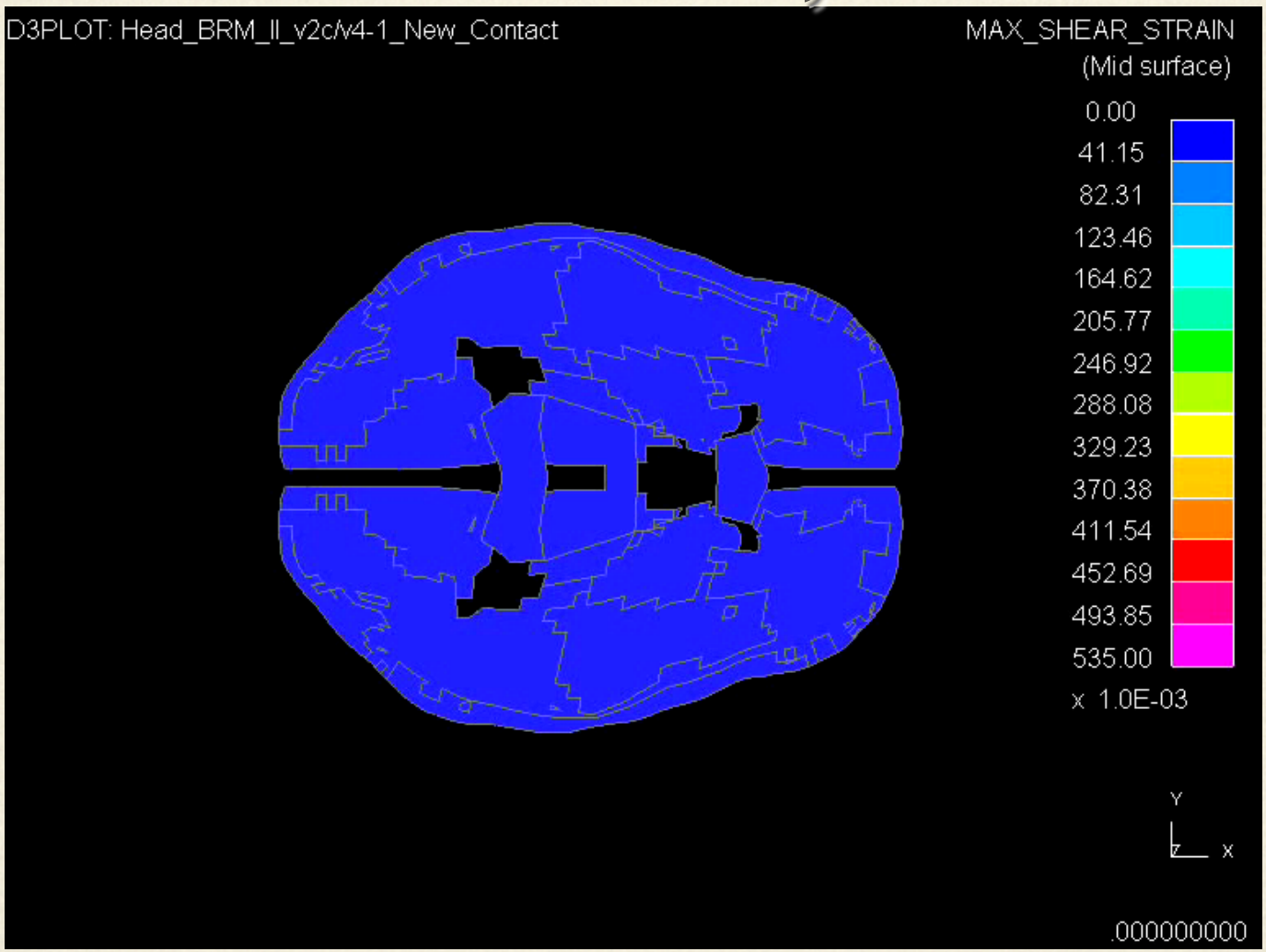
In 1936 Grundfest presented a study on the effects of different hydrostatic pressures upon the threshold of the frog sciatic nerve. There were minimal effects on nerve function for pressures up to 5,000 psi with only 10% decrease in the magnitude of the action potential and immediate recovery upon release of the pressure. When higher pressures up to 15,000 psi were applied, the potential reduced further, but was reversible even after being loaded for periods up to 20-30 minutes.

Wait a Minute. What about Linear Acceleration?

Logic:

Linear Acceleration causes
Pressure Gradient causes
Deviatoric Stresses cause
Shear Strains

GHBMC 50th Male FE Model
HIC = ~1,500



Okay, but what about Negative Pressure and Cavitation?

Logic:

Linear Acceleration causes Pressure Gradient causes
Negative Pressure at Contrecoup causing CAVITATION

Back to the basics: Part II

Contrecoup Brain Injuries

- Fallopius (1523-62) was first to describe cerebral damage on the side of the brain opposite/other the site of impact
- 1766. The French Royal Academy of Surgery sponsored research and even set prizes for elucidation of problems concerning brain injuries. Contrecoup injuries were declared a subject of particular interest.
- 1892. Miles – impact tests on animal heads (rabbits, pigs, birds) in different directions. Gave a theory of contrecoup based on a "cone of depression" forming at the site of impact that propagates to the opposite side and forming a "cone of bulging", which causes contrecoup lesions

Contrecoup Brain Injuries

- 1940. Goggio introduced the pressure gradient theory based on a simple hydrostatic theory, where negative pressure at the side opposite to impact was proposed as the mechanism of contrecoup injuries
- 1958. Gross experimented with partially fluid filled flask (simulating human brain) and attempted to explain various brain injury mechanisms including contrecoup injuries due to cavitation. He concluded that “it is violent collapse of the cavities that produces the tissue damage rather than effect of the negative pressure”; “coup cavitation occurs at the site of the impact because of the snap-back of the locally deformed skull”, etc.

Did anyone actually find/measure cavitation in brain tissue?

Nusholtz et al., 1984

- Used live anesthetized and post-mortem Rhesus monkeys and repressurized cadavers in impactor tests. Measured 3D skull kinematics and epidural pressure
- Concluded that skull deformation and angular acceleration of the head are potentially important parameters on brain injury
- "For live Rhesus subjects, negative pressure peaks during an impact event equal to or greater than ***one atmosphere*** do NOT appear to produce injury"

Takhounts et al., 2003

- DDM – dilatational damage measure
- "The DDM monitors the volume of the brain experiencing specified negative pressure levels... For the purposes here, this pressure threshold is set at ***–14.7 psi (~100 Kpa)***, the vapor pressure of water."

Note: 14.7 psi = ~ 1 atmosphere

What really is the “vapor pressure of water” or cavitation pressure of water?

Caupin and Herbert, 2006
"Cavitation in water: A review"

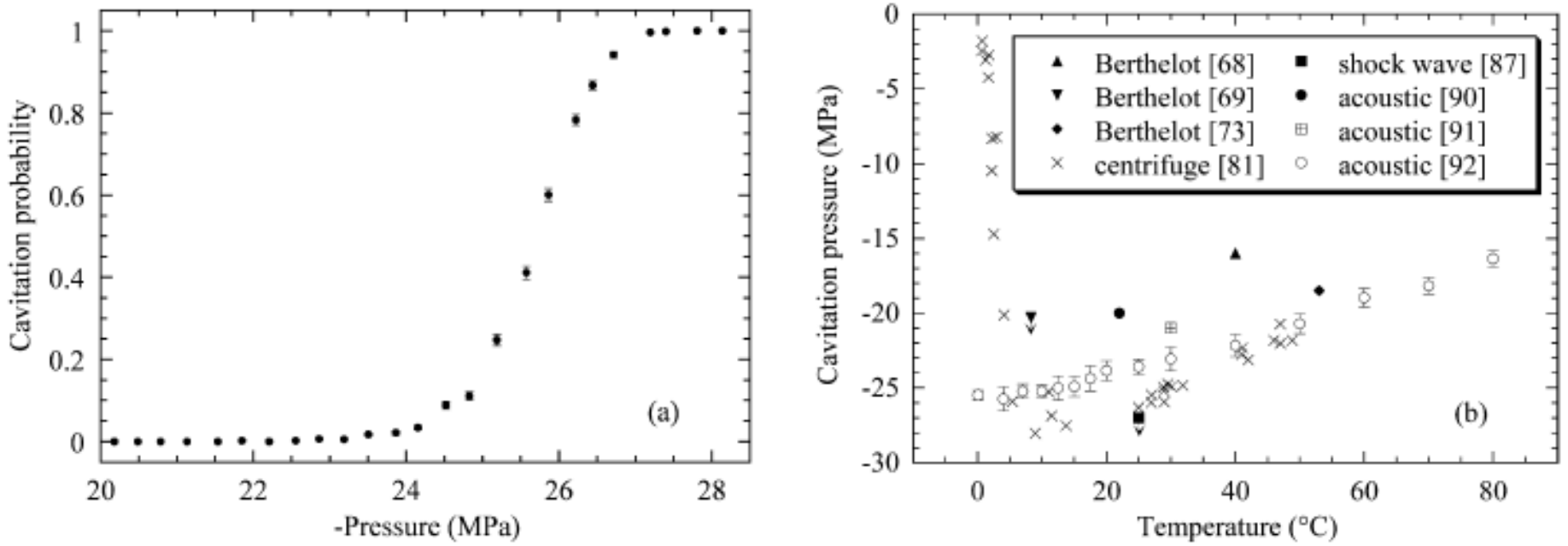


Fig. 3. (a) Cavitation probability as a function of pressure at 4 °C. Each data point was obtained by repeating 1000 acoustic bursts. The error bars are calculated from the binomial law. (b) Cavitation pressure as a function of temperature for different experiments: the corresponding method and reference are given in the legend. Only the experiments with the most negative cavitation pressures were selected, except the inclusion work, for sake of clarity. An arrow means that cavitation was not observed. The error bars on the empty circles represent the uncertainty on the pressure calibration.

What are the magnitudes of negative pressure usually measured in impact tests?

Nahum et al., 1977

1 mmHg = 133.3 Pa

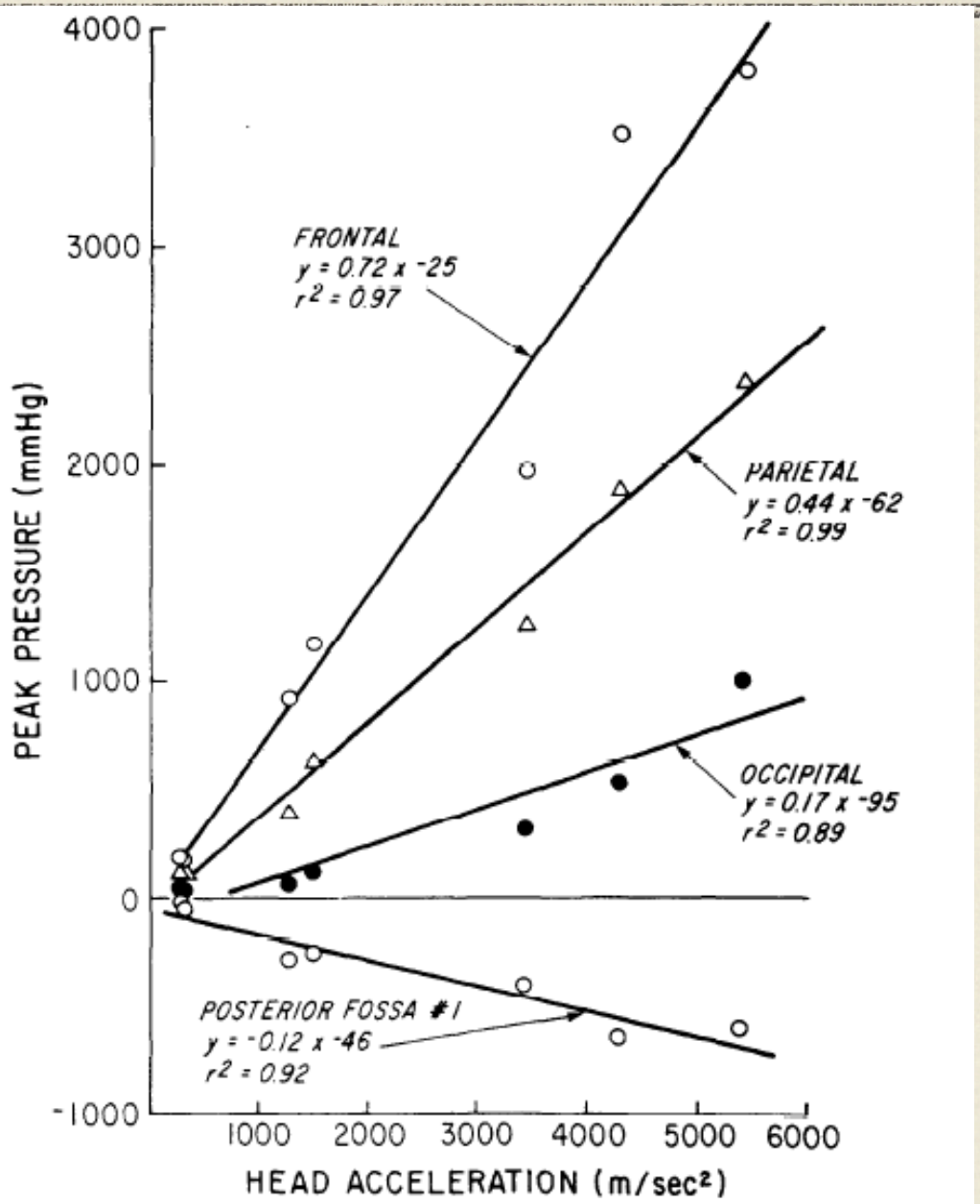


Fig. 7 - Regression analysis of peak pressure-head acceleration relationship

Mini Summary II

- Hydrostatic stress/pressure does NOT appear to cause nervous tissue damage
- Linear acceleration does cause shear strains, but they are small
- Brain injuries due to negative pressure have never been demonstrated
- Negative pressure does cause cavitation, but these negative pressures are in order of -25 Mpa at room and body temperature, much lower than -150 kPa that are seen in impact tests
- In order to cause brain injury due to cavitation two conditions have to be satisfied: (1) cavitation has to be present at the body temperature, and (2) sufficient time has to pass for the vapor nuclei to cause tissue damage – neither has been demonstrated experimentally
- Cavitation as a mechanism of brain injury is NOT supported by the existing experimental data

Stress Tensor

$$\boldsymbol{\sigma} = \begin{bmatrix} \sigma_{11} & \sigma_{12} & \sigma_{13} \\ \sigma_{21} & \sigma_{22} & \sigma_{23} \\ \sigma_{31} & \sigma_{32} & \sigma_{33} \end{bmatrix}$$

$$\boldsymbol{\sigma} = \underbrace{\boldsymbol{\sigma}_{HYD}}_{\text{Dilatational or Hydrostatic Component}} + \underbrace{\boldsymbol{\sigma}'}_{\text{Deviatoric Component}}$$

Does deviatoric or shear stress component of the stress tensor cause damage to brain tissue?

Recall:

Deviatoric or shear stress component of the stress tensor tries to change the shape of a material and is proportional to shear strain

How to change the shape of the brain?

Or

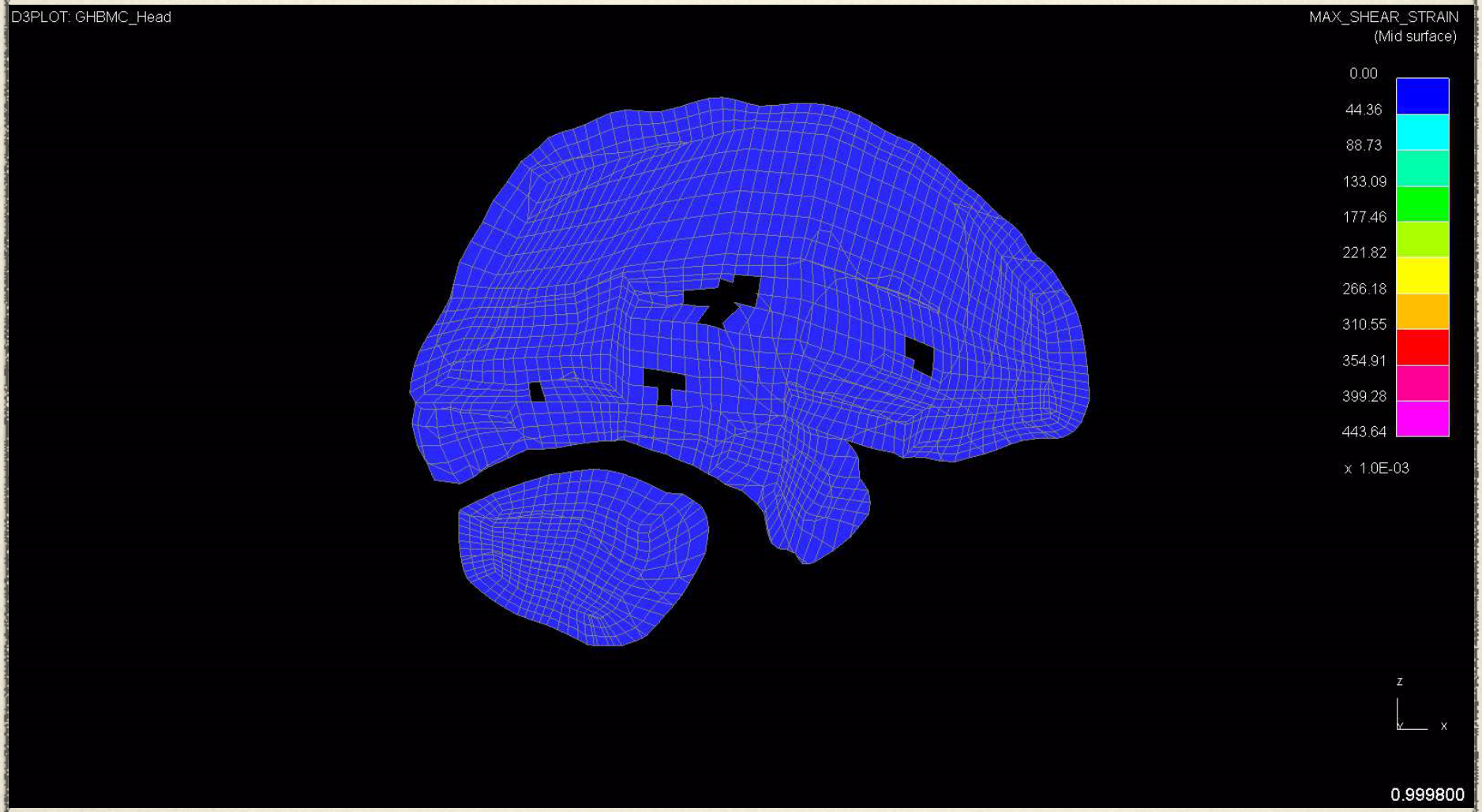
What is the easiest way to change the shape of the brain?

Head

ROTATION

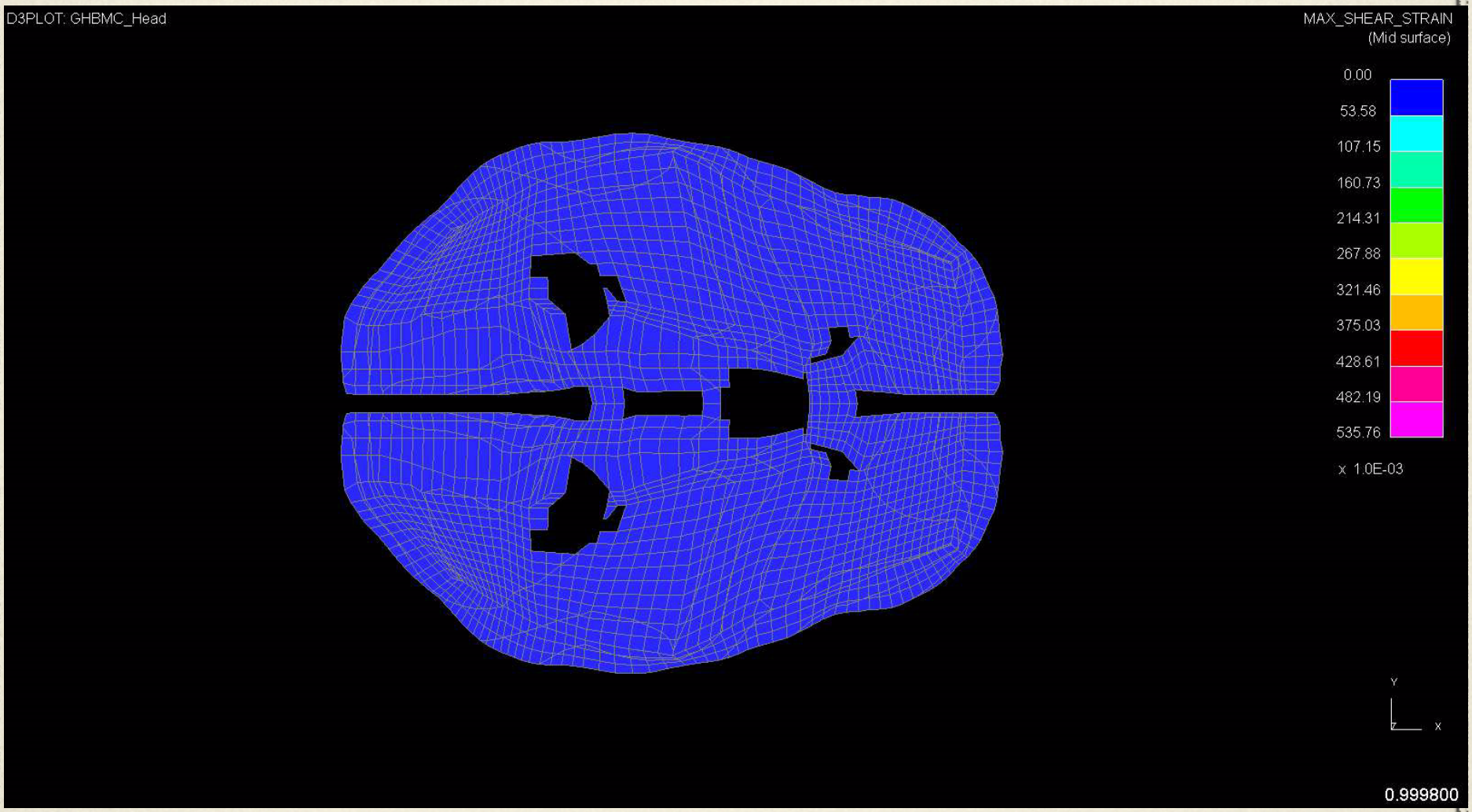
GHBMC 50th Male FE Model

Angular Velocity = 40 rad/s; Sagittal Plane



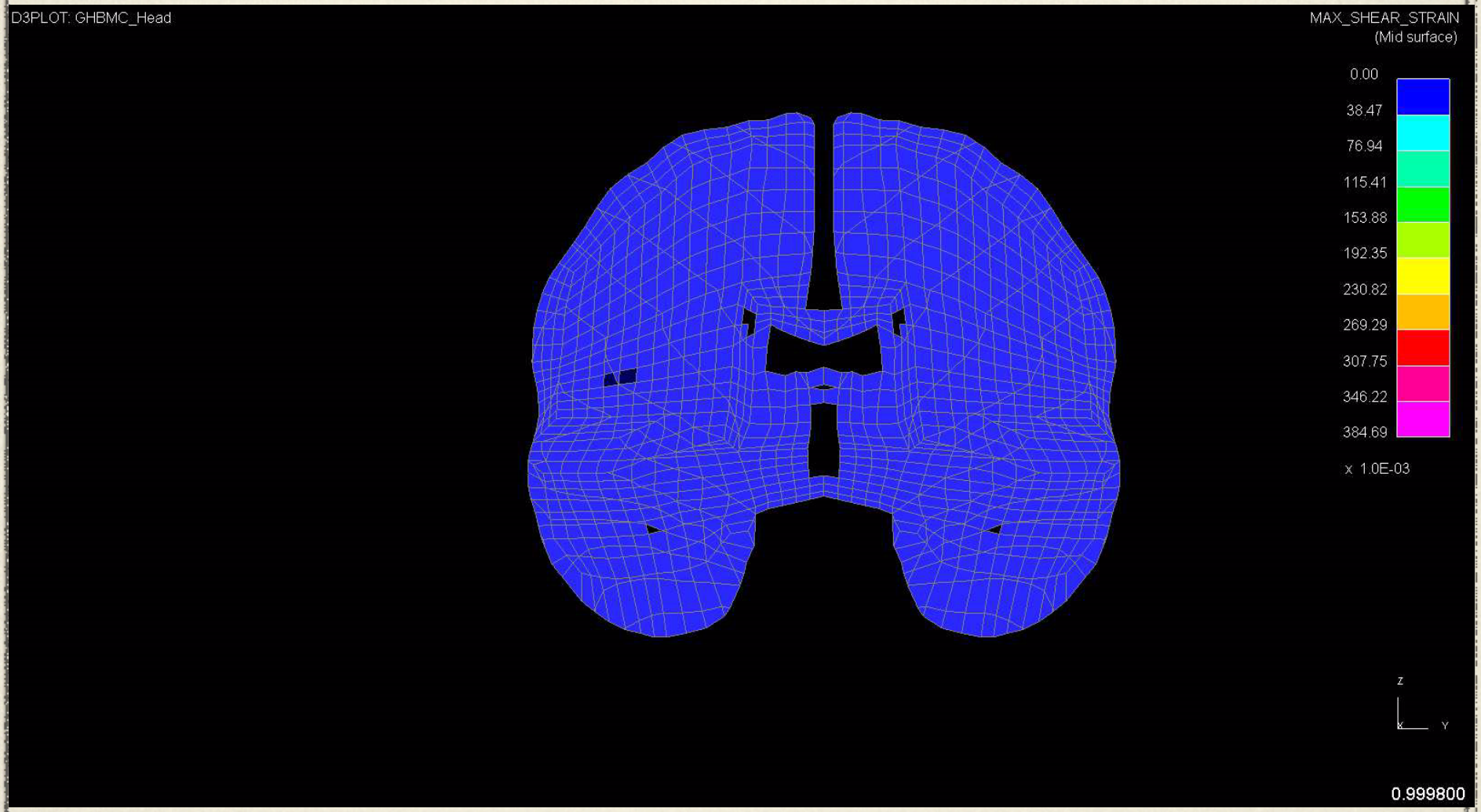
GHBMC 50th Male FE Model

Angular Velocity = 40 rad/s; Horizontal Plane



GHBMC 50th Male FE Model

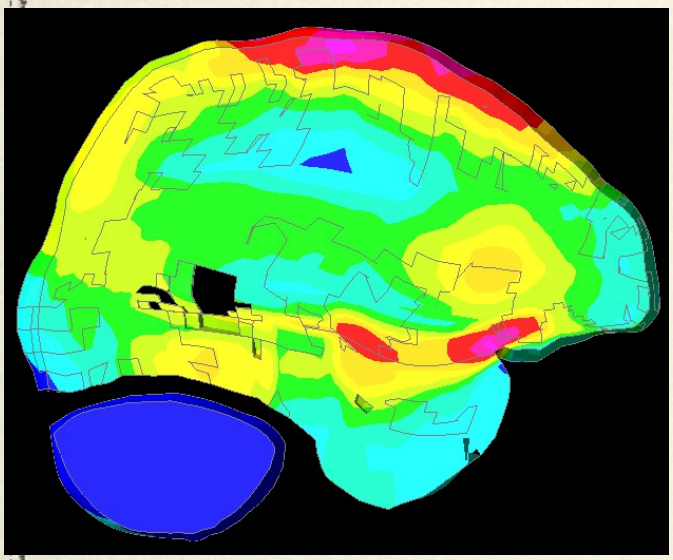
Angular Velocity = 40 rad/s; Coronal Plane

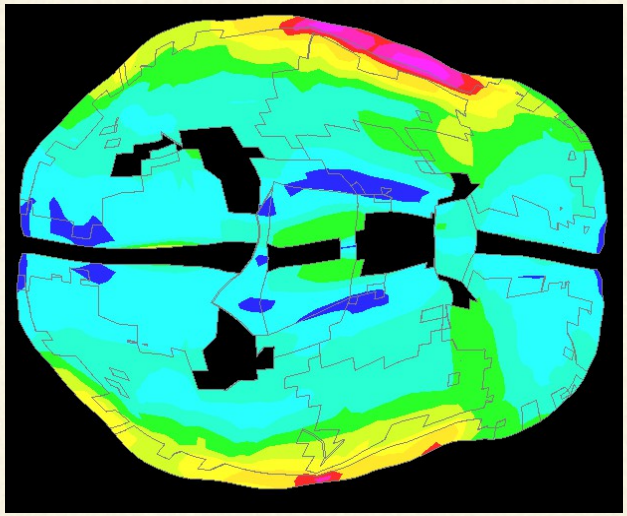


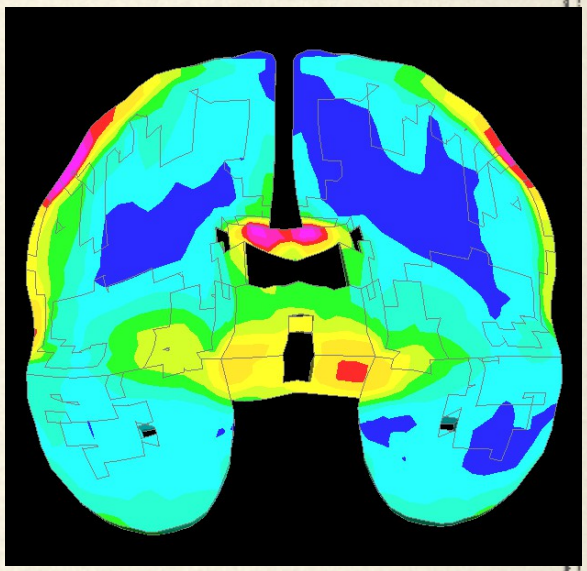
Can Contrecoup injuries be explained with the shear strain/ rotation theory?

GHBMC 50th Male FE Model

Angular Velocity = 40 rad/s

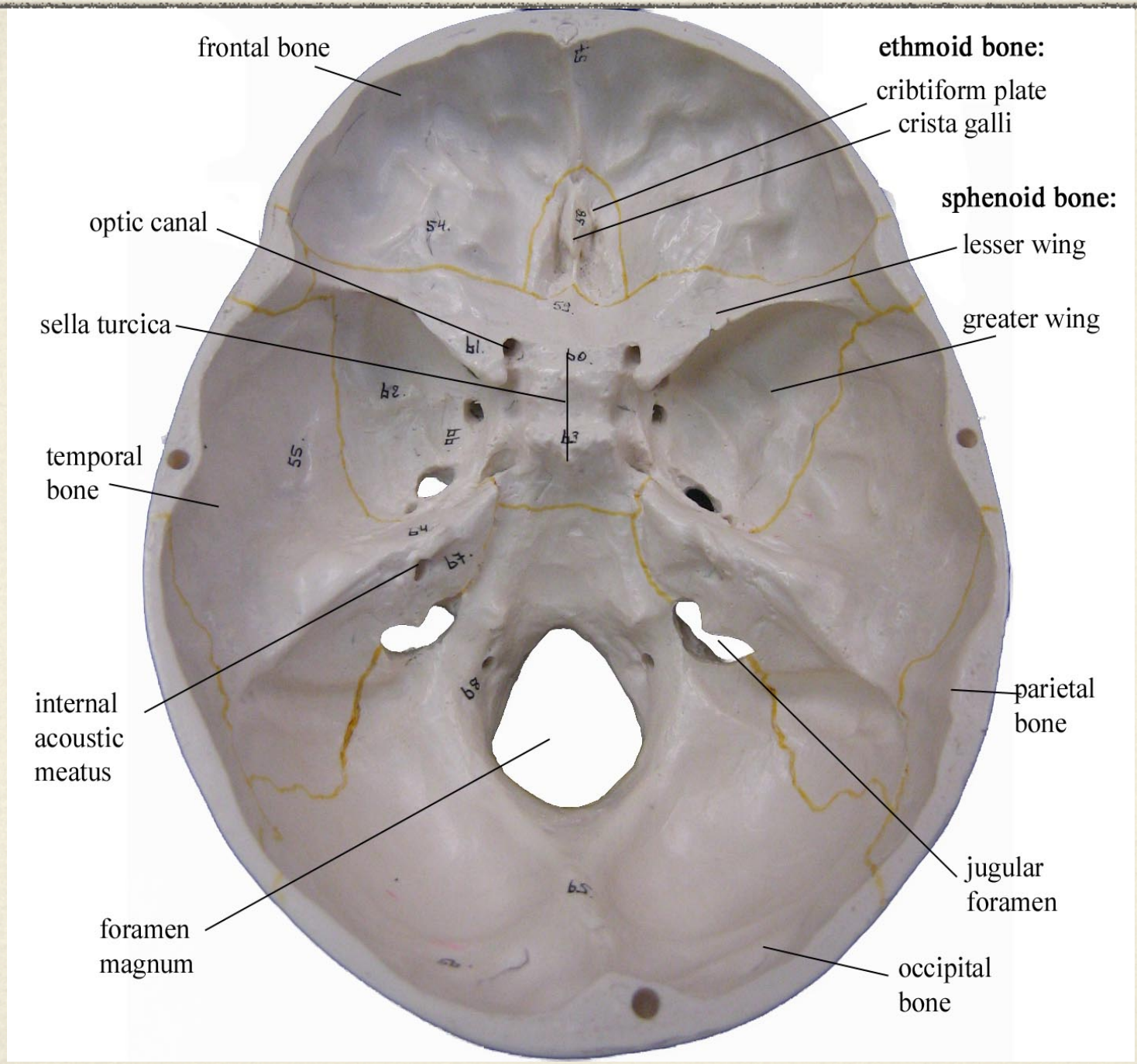






Courville, 1942

Reviewed 206 cases of fatal brain injuries that were results of automotive collisions and falls. Made a few valuable observations: (1) frontal impacts – only coup contusions occurred on the basilar surface of the frontal lobe; (2) occipital impacts – only contrecoup contusions occurred at the same site as frontal impacts; (3) in side impact to the temporal or parietal regions the major contusion is the contrecoup one (smaller coup lesions are also found in some cases). Observed that the more irregular the bony walls of the skull are the more likely it is that that part will sustain coup or contrecoup contusions


frontal bone
ethmoid bone:
cribtiform plate
crista galli
sphenoid bone:
optic canal
lesser wing
sella turcica
greater wing
temporal bone
internal acoustic meatus
parietal bone
jugular foramen
foramen magnum
occipital bone

Head rotation causes high shear strains inside the brain.

Are these shear strains proportional to head rotational angle, velocity, or acceleration?

Holbourn, 1943

Takhounts et al., 2013

The best correlate to max principal or shear strain in the brain is the head rotational/angular velocity

Mini Summary III

- Deviatoric component of shear stress tensor is proportional to shear strains
- The easiest way to create high shear strains inside the brain is via head rotation
- Contrecoup (and coup) injuries can be explained with the shear strain injury mechanism
- Max shear and principal strains inside the brain are proportional to the magnitude of rotational/angular velocity (for the vast majority of impacts)

Takhounts et al., 2013

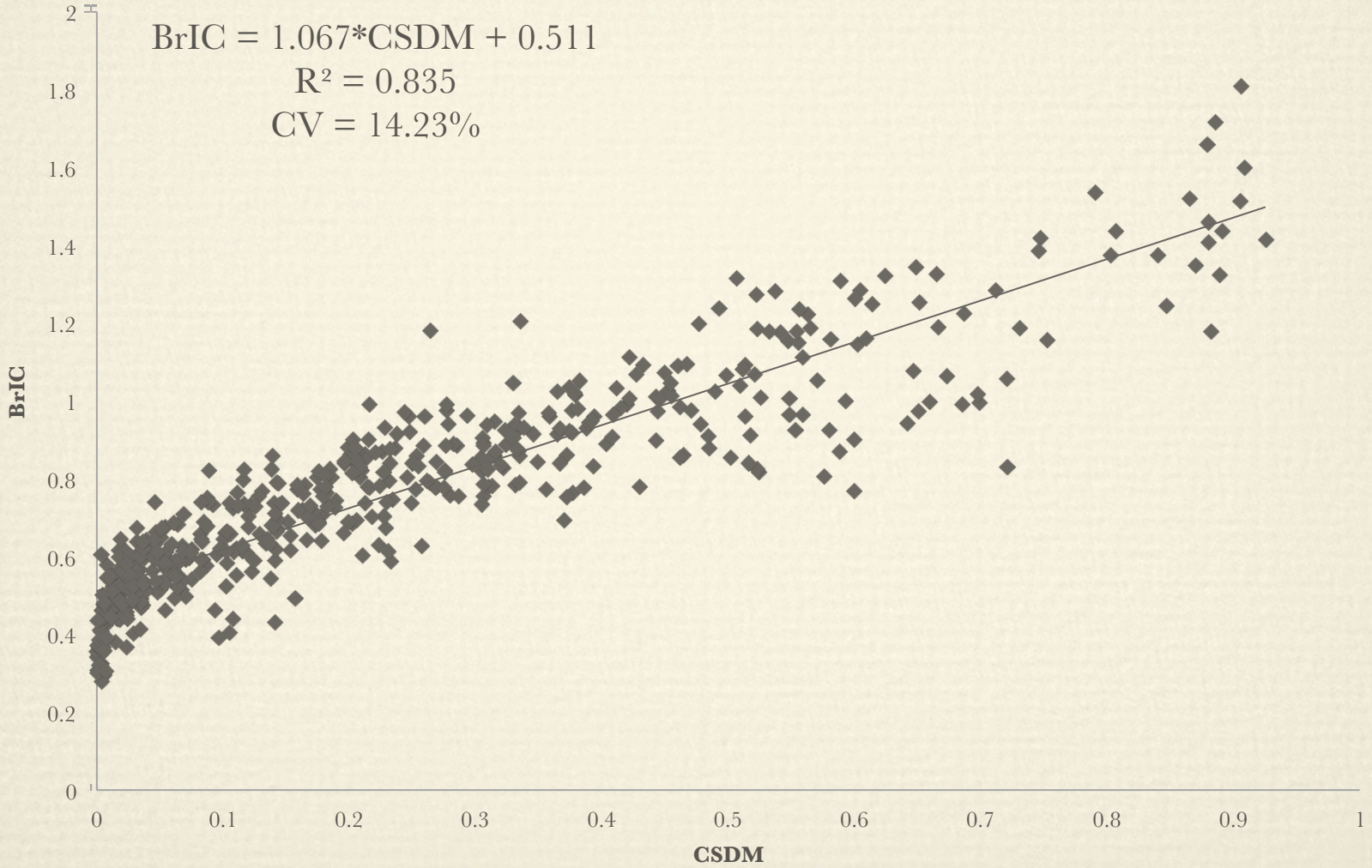
Even better correlate to max principal strain is BrIC

BrIC Formulation

$$BrIC=\sqrt{((\omega_x/\omega_{xC})^2+((\omega_y/\omega_{yC})^2+((\omega_z/\omega_{zC})^2}$$

ω_{xC}	66.25 rad/s
ω_{yC}	56.45 rad/s
ω_{zC}	42.87 rad/s

"Original BrIC" is a Correlate for CSDM (543 tests)



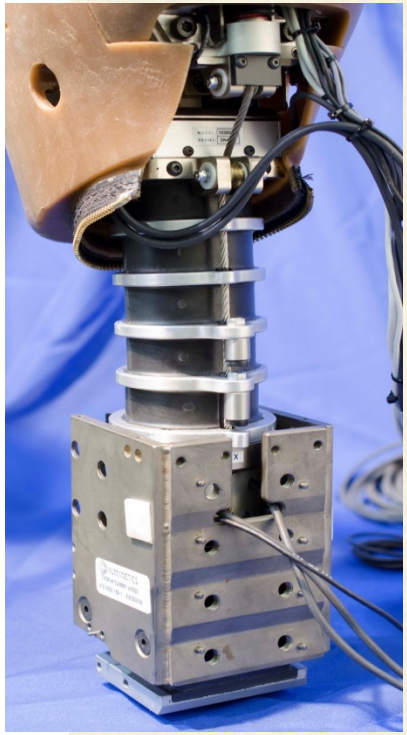
CSDM -> Cumulative Strain Damage Measure

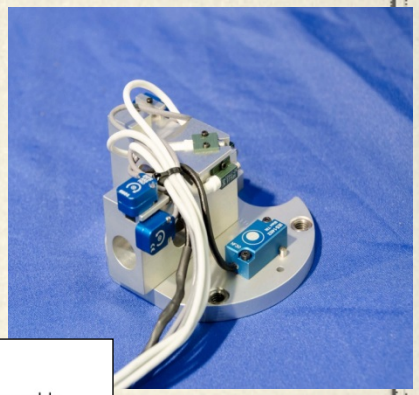
The volume of the brain exceeding prescribed level of max principal strain

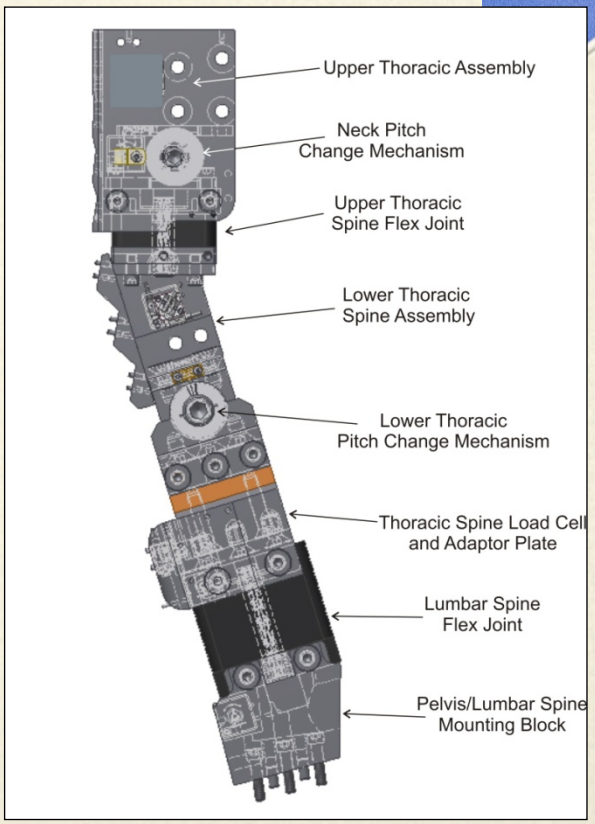
TOOLS to Measure Correct Head Angular Kinematics

- Measuring 3D head motion is not a trivial task
- Much easier to measure only linear head kinematics
- Angular rate sensors (ARS) for measuring angular velocity
- Measuring angular accelerations is still a challenge
- Biofidelic human models (physical and/or mathematical)

Biofidelic Human Physical Model – THOR ATD



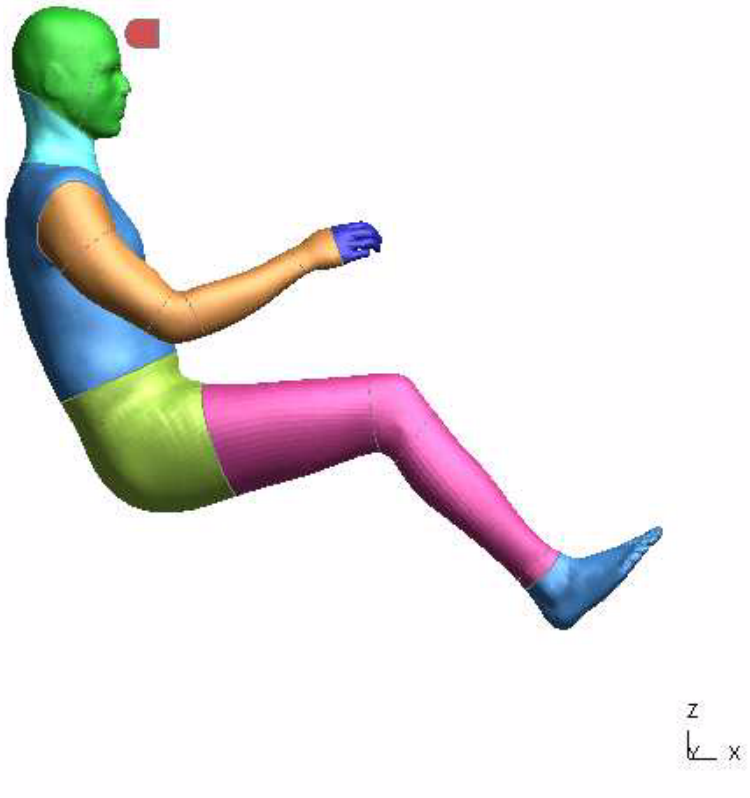




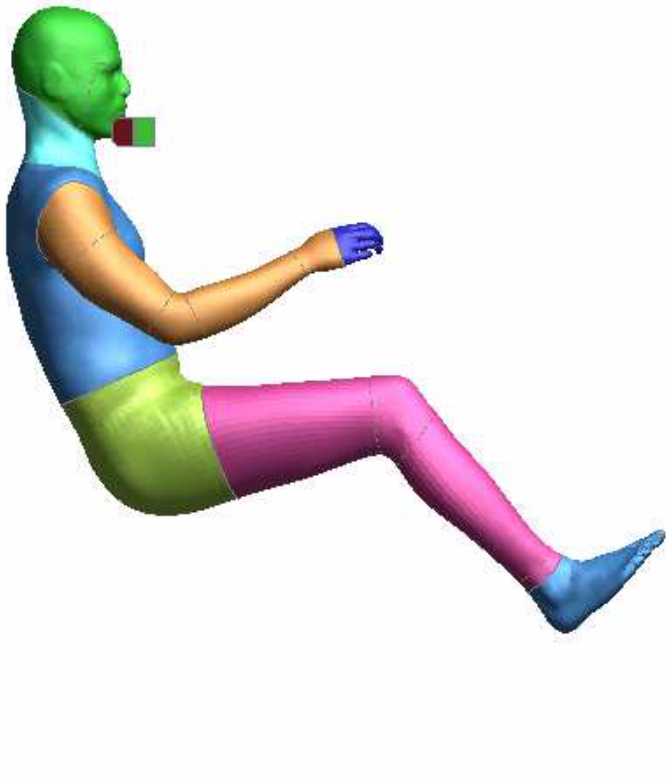
Is there any particular impact direction that causes highest strains inside the brain?

Biofidelic Human Mathematical Model – GHBMC FE Model

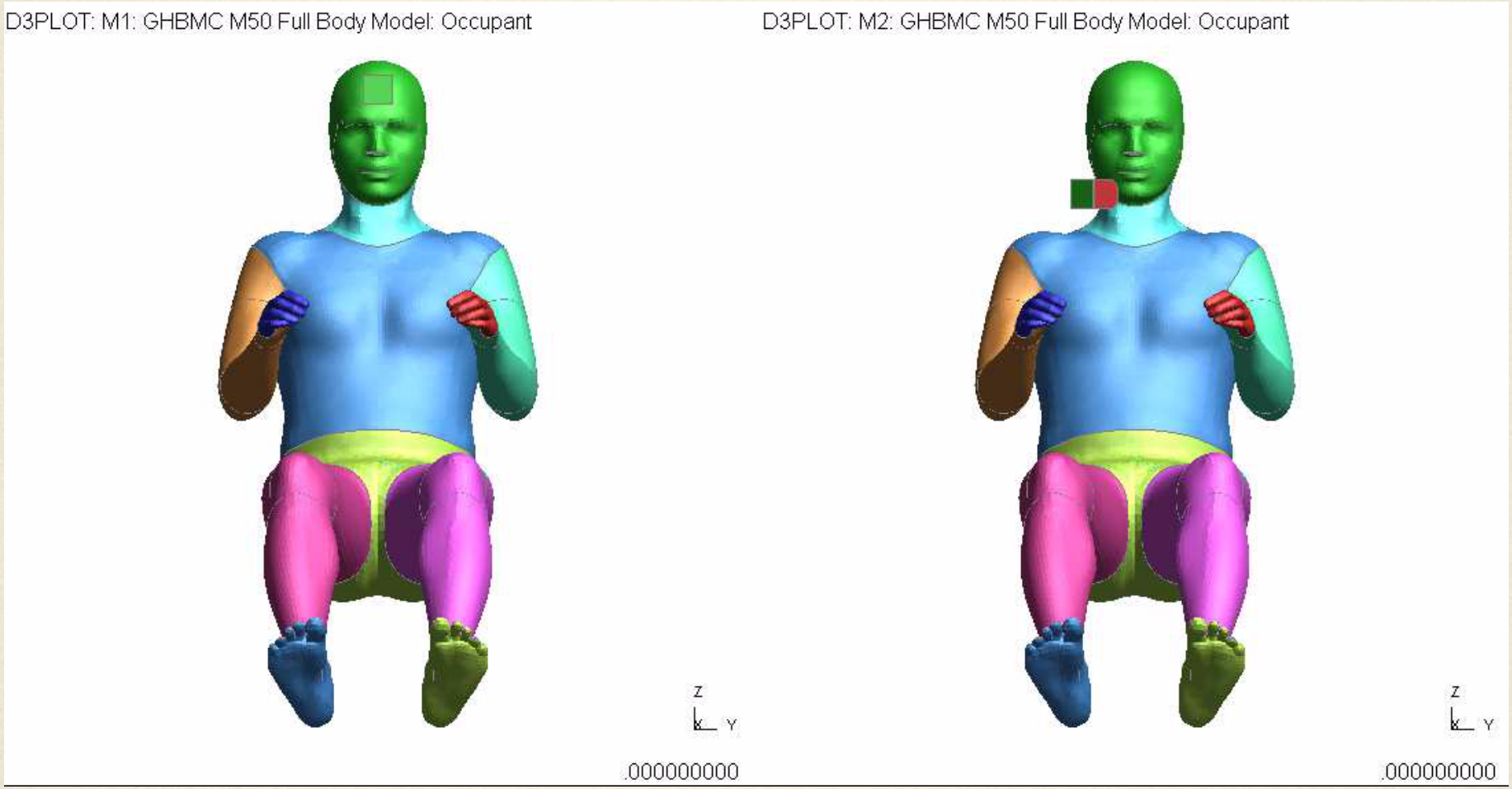
D3PLOT: M1: GHBMC M50 Full Body Model: Occupant

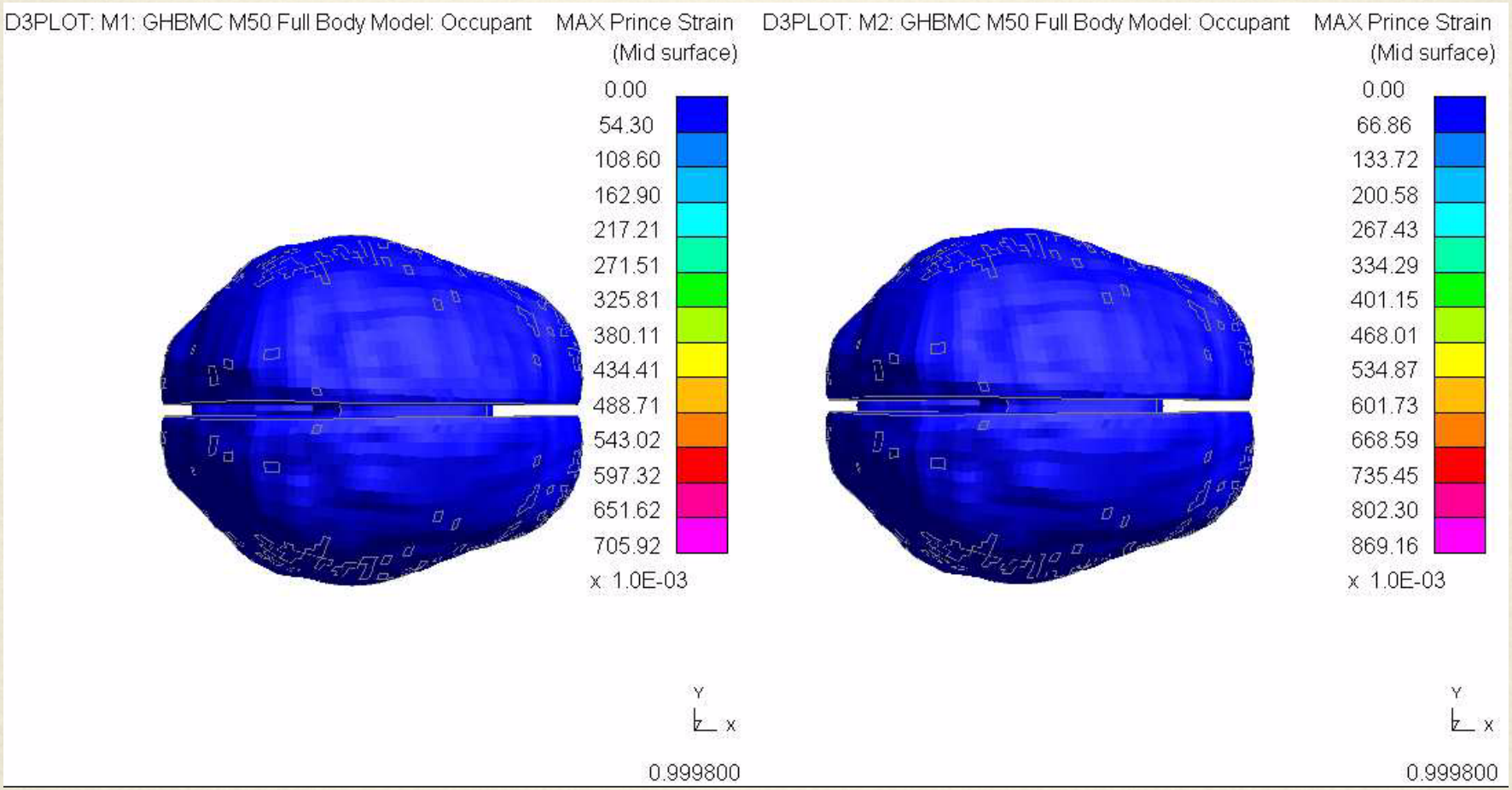


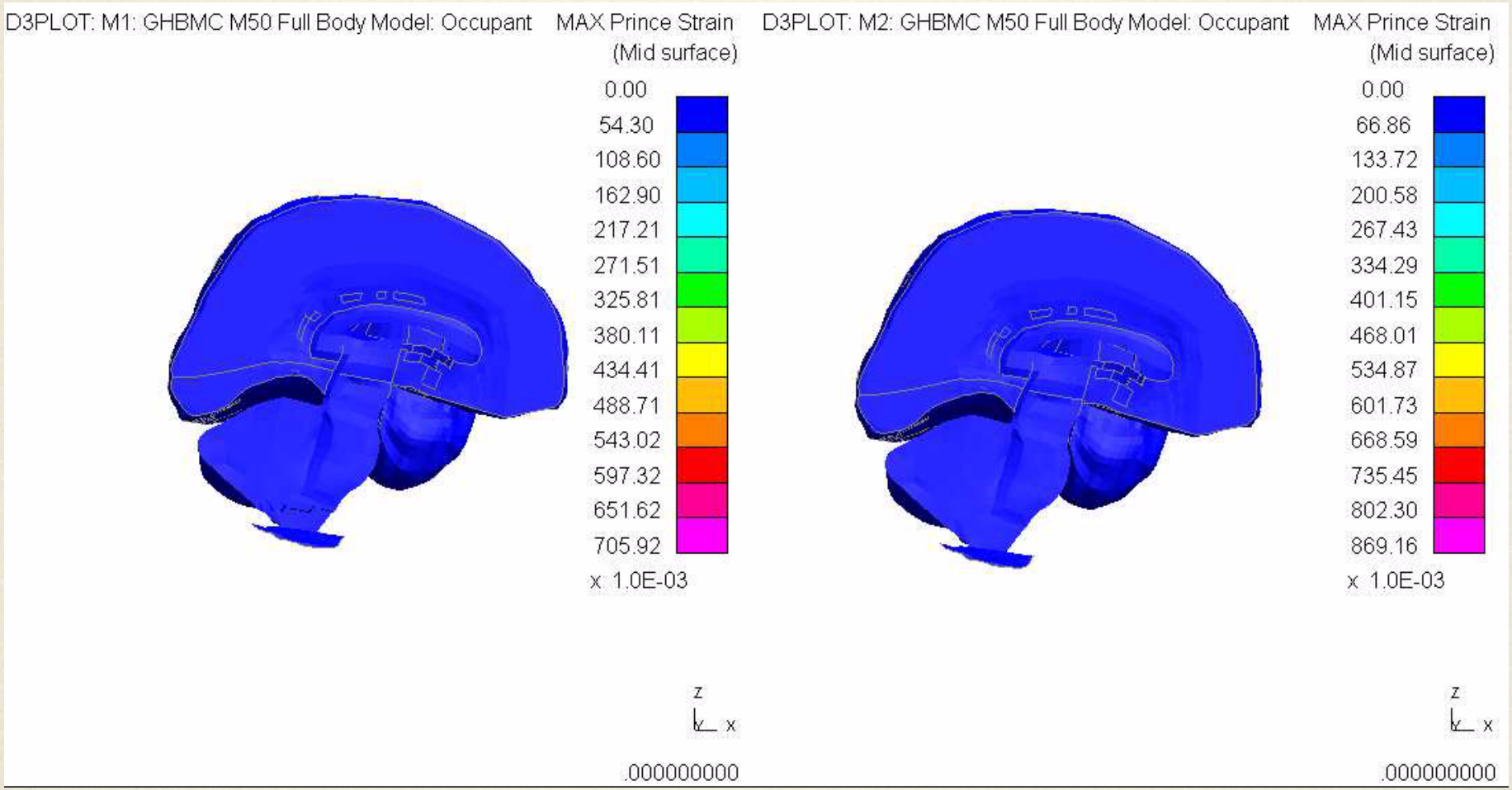
D3PLOT: M2: GHBMC M50 Full Body Model: Occupant



Biofidelic Human Mathematical Model – GHBMC FE Model




D3PLOT: M1: GHBMC M50 Full Body Model: Occupant
MAX Prince Strain
(Mid surface)
0.00
54.30
108.60
162.90
217.21
271.51
325.81
380.11
434.41
488.71
543.02
597.32
651.62
705.92
x 1.0E-03
Y
x
0.999800
D3PLOT: M2: GHBMC M50 Full Body Model: Occupant
MAX Prince Strain
(Mid surface)
0.00
66.86
133.72
200.58
267.43
334.29
401.15
468.01
534.87
601.73
668.59
735.45
802.30
869.16
x 1.0E-03
Y
x
0.999800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Impact	Contact Force	CSDM	MPS
Forehead	28KN	0.42	0.71
Chin	4KN	0.43	0.87

Conclusions

- Dilatational component of the stress tensor (pressure) does NOT appear to cause brain injuries
- Cavitational theory of brain injury is without experimental foundation
- Linear acceleration doesn't results in high strains inside the brain
- The easiest way to create high strains inside the brain is via head rotation – rotational velocity
- Rotational velocity is proportional to the strains inside the brain
- BrIC correlates better to the strains inside the brain than any other kinematic parameter
- Chin impact requires much lower force than the forehead impact to generate similar levels of strain and CSDM

Tools

- GHBMC FE human models
  - http://www.ghbmc.com/ghbmc-wp-site
  - http://www.elemance.com
- NHTSA website (free stuff)
  - http://www.nhtsa.gov/Research/Biomechanics-&-Trauma/Brain-Injury-Research
    - FE models (SIMon keyword, SIMon stand alone, presentations, papers, etc.)
  - http://www.nhtsa.gov/Research/Biomechanics-&-Trauma/THOR-50th-Male-ATD
    - THOR ATD

The End

Questions?

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