Helmet Protection against Traumatic Brain Injury: A Physics Perspective

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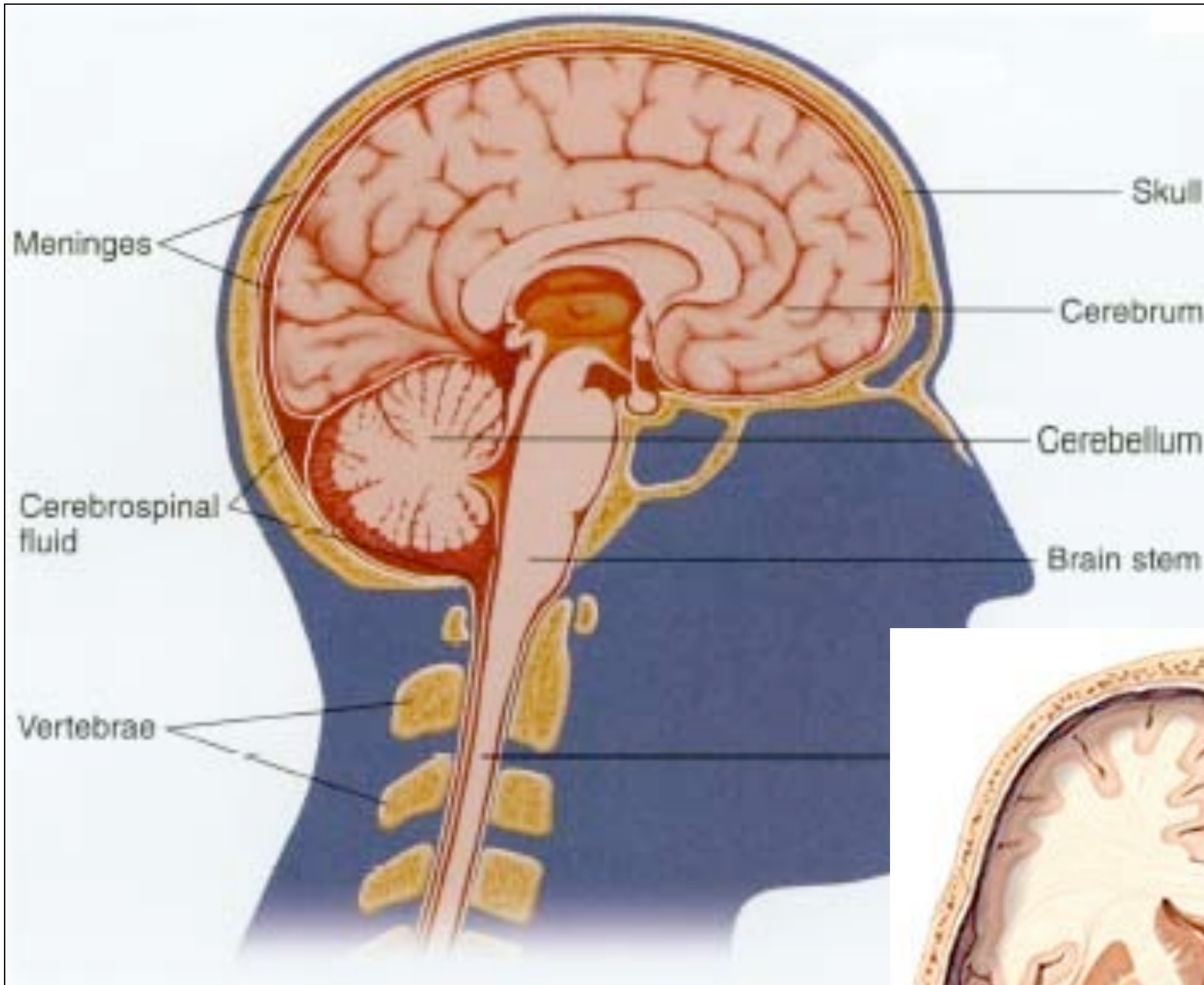
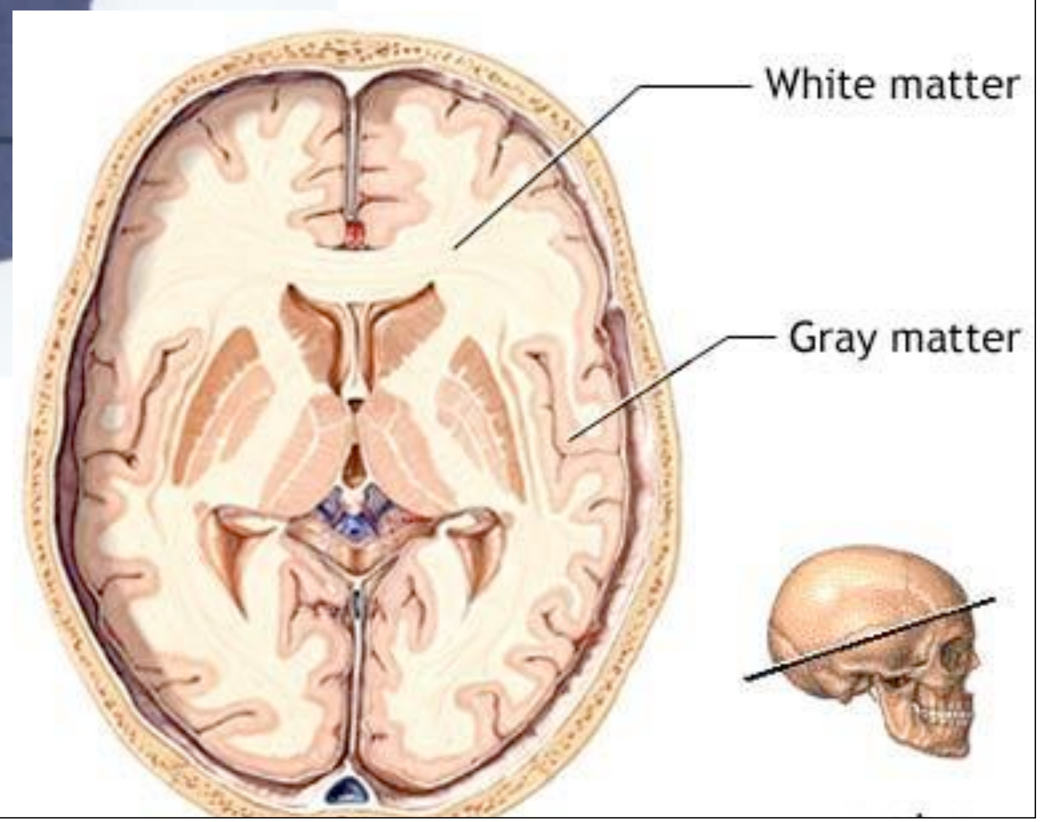
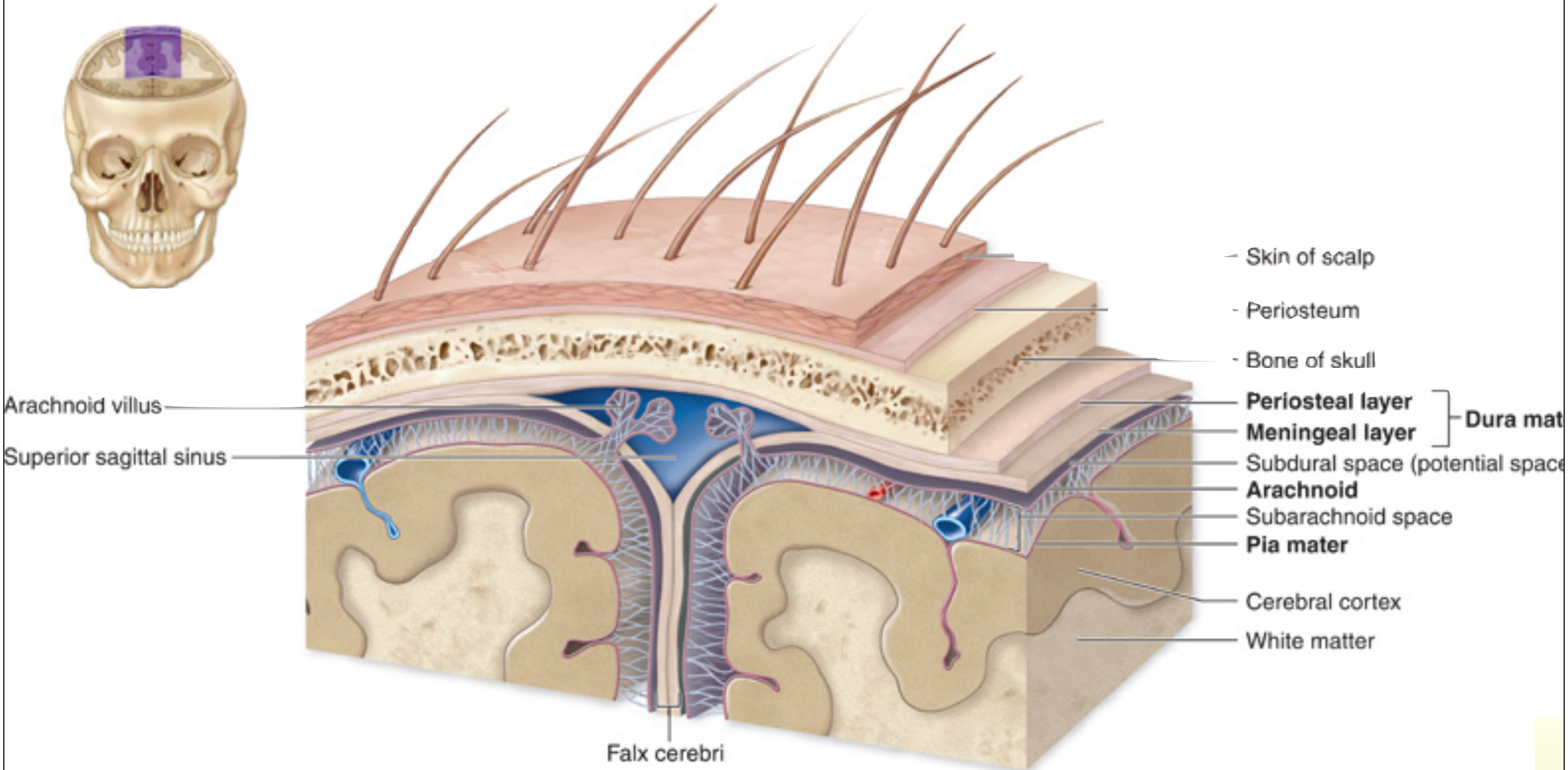
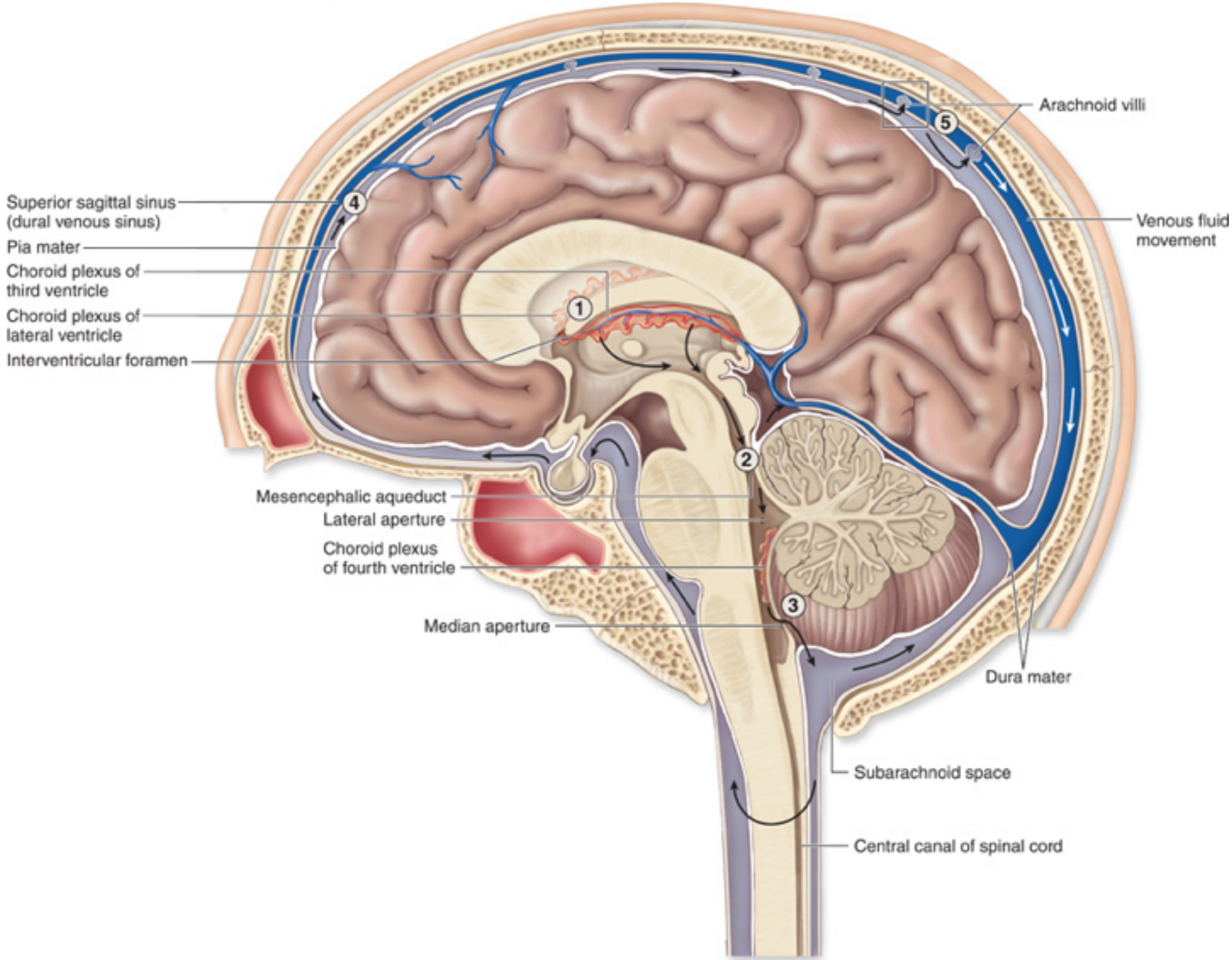
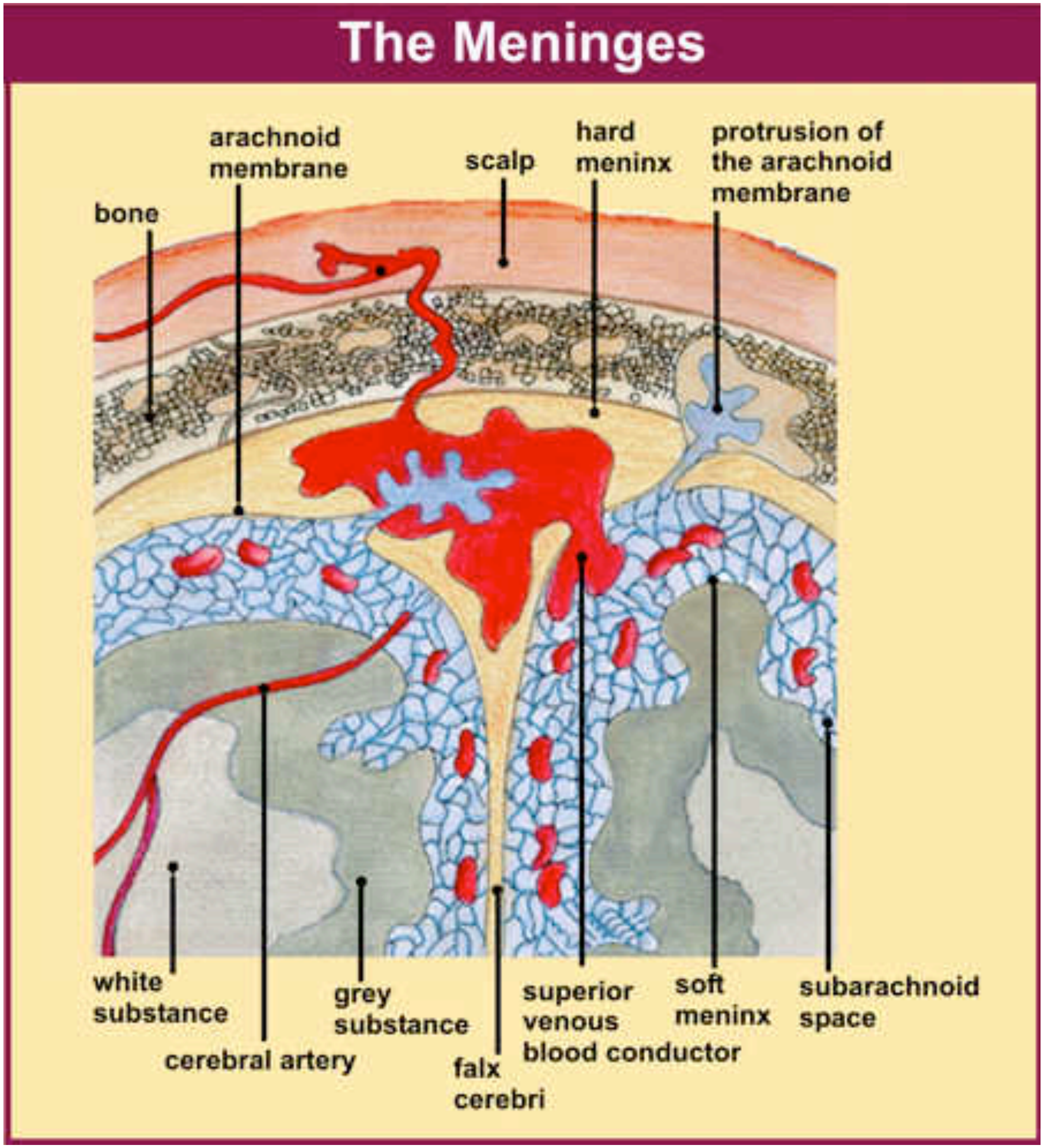

Skull
Meninges
Cerebrum
Cerebellum
Cerebrospinal fluid
Brain stem
Vertebrae


White matter
Gray matter


Skin of scalp
Periosteum
Bone of skull
Arachnoid villus
Periosteal layer
Meningeal layer
Dura mat
Superior sagittal sinus
Subdural space (potential spac
Arachnoid
Subarachnoid space
Pia mater
Cerebral cortex
White matter
Falx cerebri



(a) Midsagittal section


The Meninges
arachnoid membrane
scalp
hard meninx
protrusion of the arachnoid membrane
bone
white substance
cerebral artery
grey substance
falx cerebri
superior venous blood conductor
soft meninx
subarachnoid space

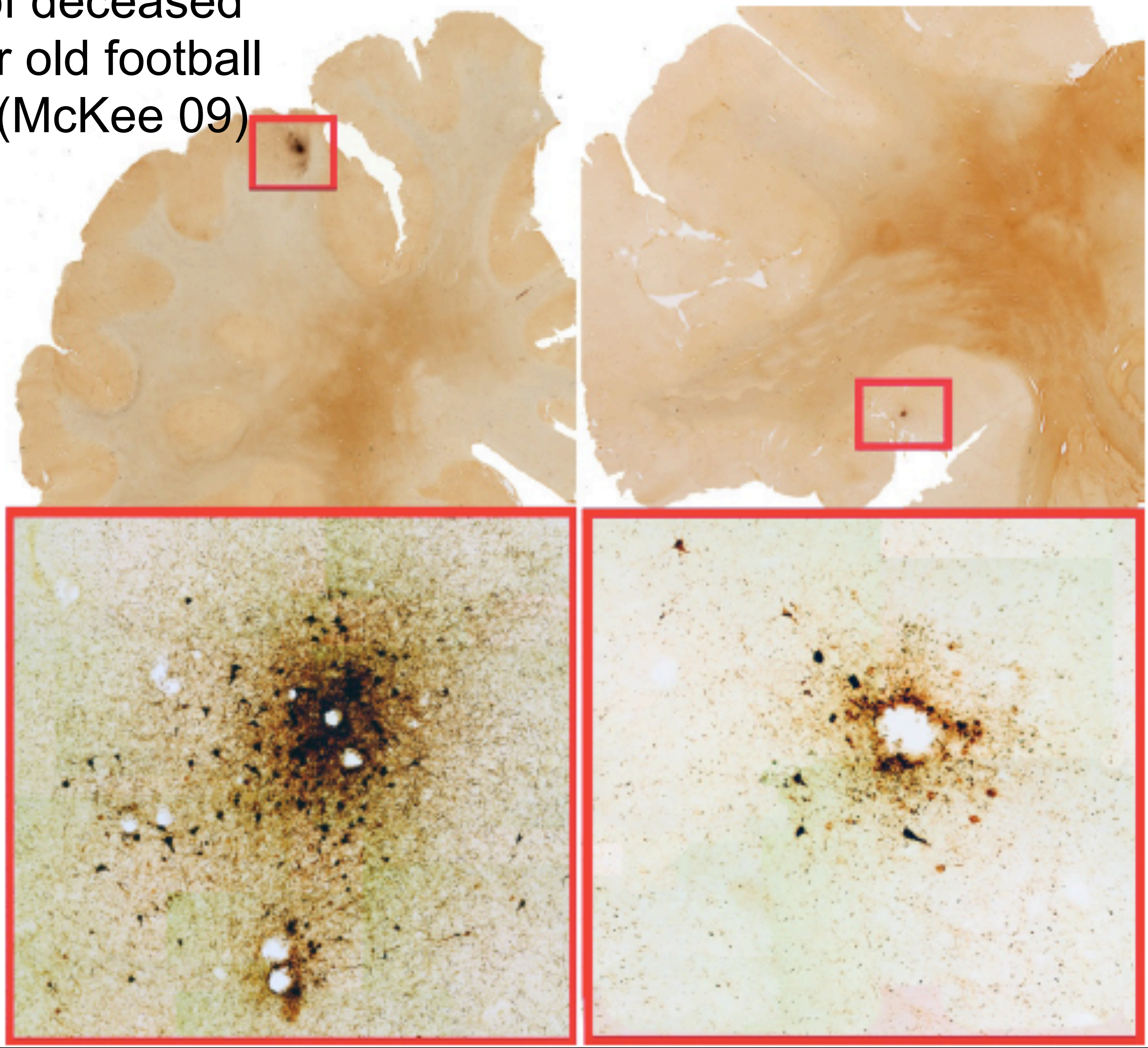
Closed Traumatic Brain Injury (TBI)

- physical injury to the brain without skull fracture
  - **concussions** (non-local; midbrain, brainstem, frontal lobe)
  - **diffuse axonal injury** (shear damage of axons; white matter grey matter linkage)
  - **contusions** (general bruising)
  - **subdural hematoma** (bridging vein damage)
  - **chronic traumatic encephalopathy** (CTE) (degenerative brain injury from repetitive head trauma)

Chronic Traumatic Encephalopathy (e.g. McKee 2009)

- CTE: toxic "tau protein" builds up in brain cells, preventing normal connections to other cells; cells die
- tau protein shows up as neurofibrillary tangles (NFTs) and glial tangles
- Tangles are formed by hyperphosphorylation of tau proteins in microtubules, causing tau to aggregate
- accompanies dementia though not itself a signature of Alzheimers (no beta amyloid)
- Prevalent in brain tissue of deceased football players and boxers, some even without clinical history of excessive concussions.
- Role of many low level impacts vs. few extreme impacts on CTE/ ITBI requires more work

Brain of deceased 18 year old football player (McKee 09)



Sources of TBI without skull fracture

- (1) head impact (ITBI)
- (2) blast overpressure (OTBI)
- (3) blast + impact: ITBI + OTBI combination must be common

Cost of TBI (in USA)

- **Human costs**

- **Civilian**: $2x10^6$ cases/yr; 50% auto; 25% sports (McArthur 04)
  - 20 deaths per 100,000: $20 billion/yr treatment

- **Military**:
  - before 2006; estimated 3% of soldiers have TBI (60% of hospital injured soldiers)
  - 0.6% of all soldiers serious TBI
  - New screenings: 2006-2009 ~20% of all troops have TBI; 1.5% of all troops unfit to return by current military standard
  - cost $2.7 million (Blimes 07) per 25 yr post-TBI life of soldier >$2 billion/year just for treatment of soldiers

- **Workforce / mission / security costs**

TBI is an Interdisciplinary Frontier

-Timely TBI: military, NFL

-Modern protection equipment has reduced fatalities, leaving previously hidden secondary injuries.

-NFL: 60% incur at least 1 concussion; retired players 19 times more likely to show symptoms of CTE (McKee 09)

-Many aspects of TBI science are nascent

- medical screening and correlation with trauma
- "macho" culture: TBI not always understood as physical
- PTSD vs TBI diagnosis and treatment
- physiology and biology of injury
- connecting external force to specific injury (impact vs. blast)
- basic physics of protection/ engineering protective equipment
- understanding deficiencies in protective equipment
- data collection

-Business, Politics, vs. Science

- **Impact TBI (ITBI) protection suffers from:**
  - -inadequate measures and standards
  - -insufficient data
  - -lack of first-principles modeling
  - -insufficient interdisciplinary research

- **Overpressure TBI**: an even newer frontier

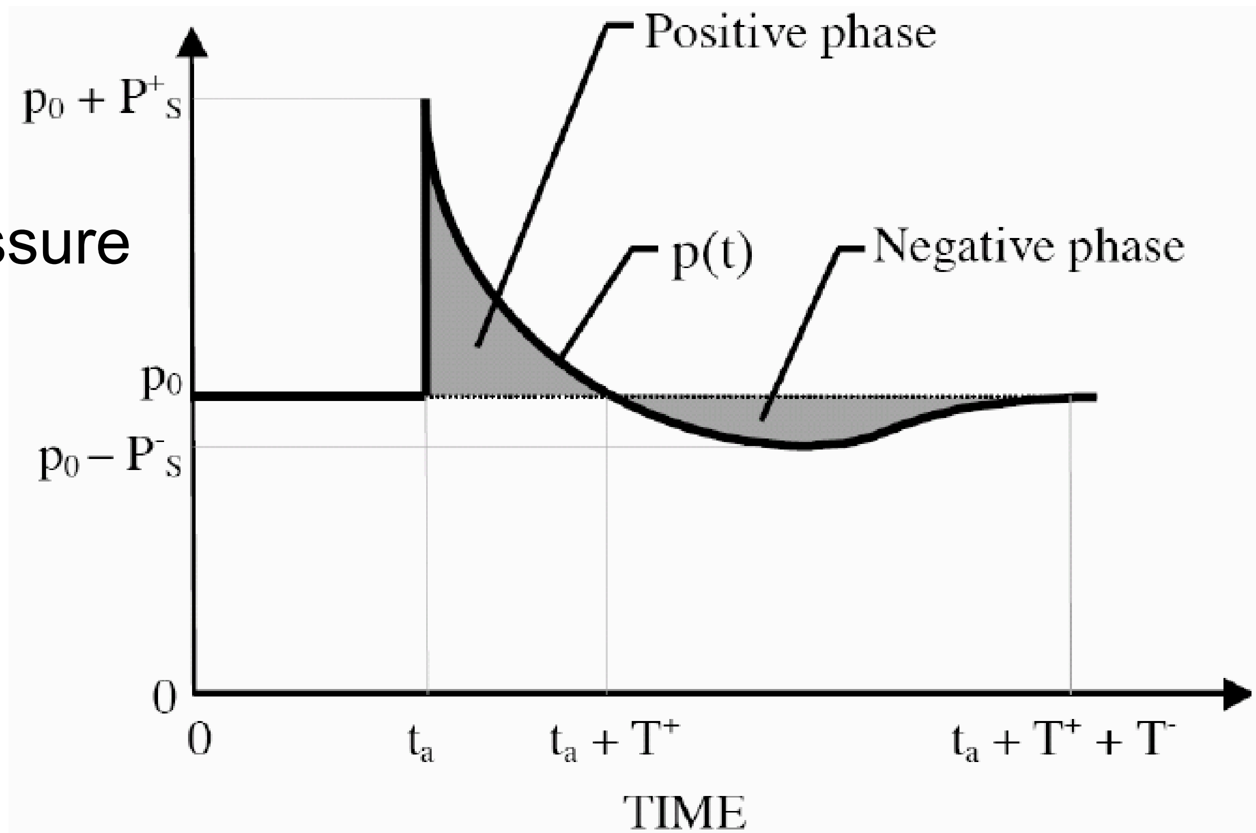
- **Blast produces pressure + impact injury**

Blast Injury: OTBI vs. ITBI

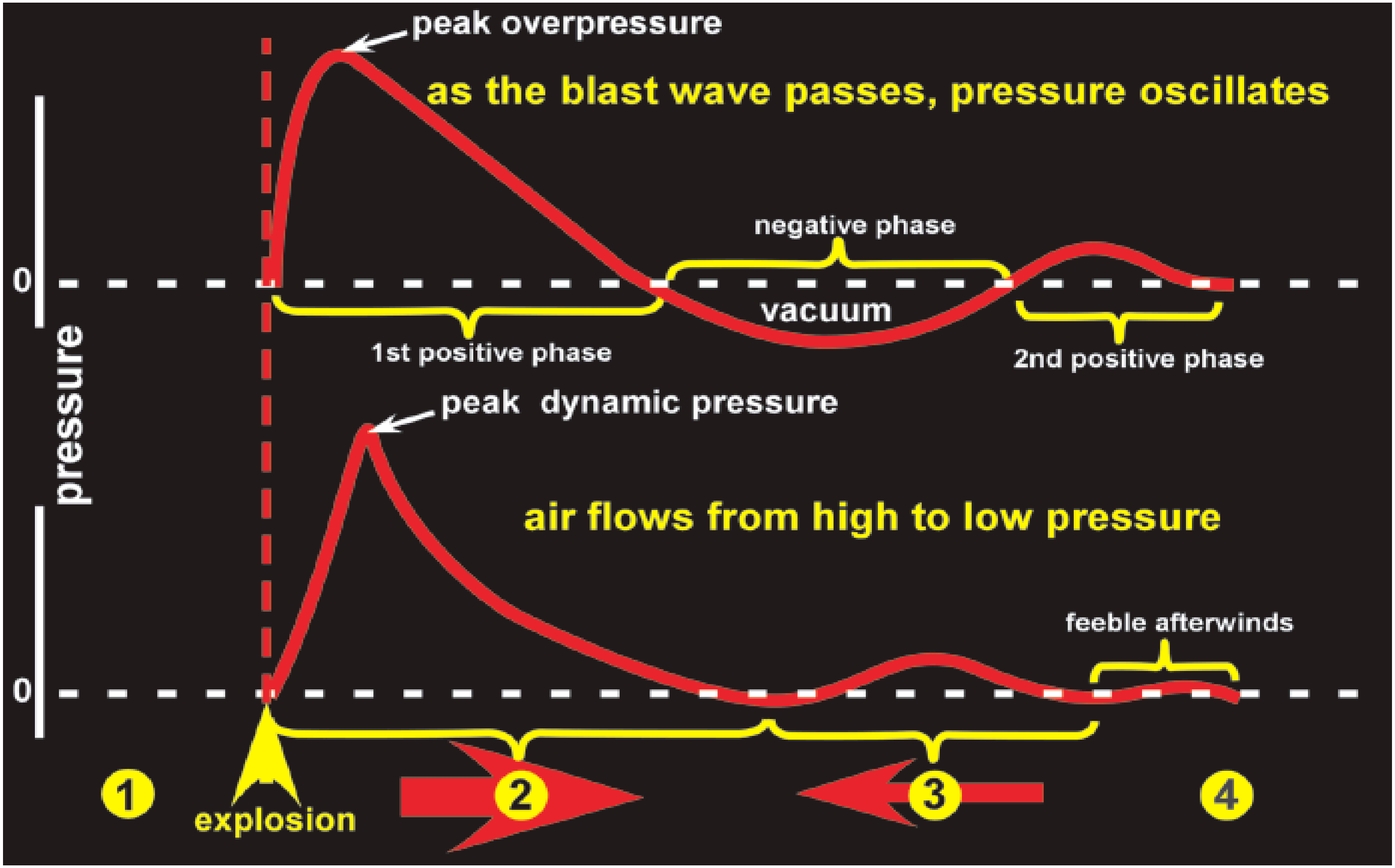
- new frontier of helmet design
- Sources of Injury
  - Primary (overpressure)
  - Secondary (shrapnel)
  - Tertiary (impact)

Basic Blast Physics

Static over-pressure
at fixed position



Basic Blast Physics



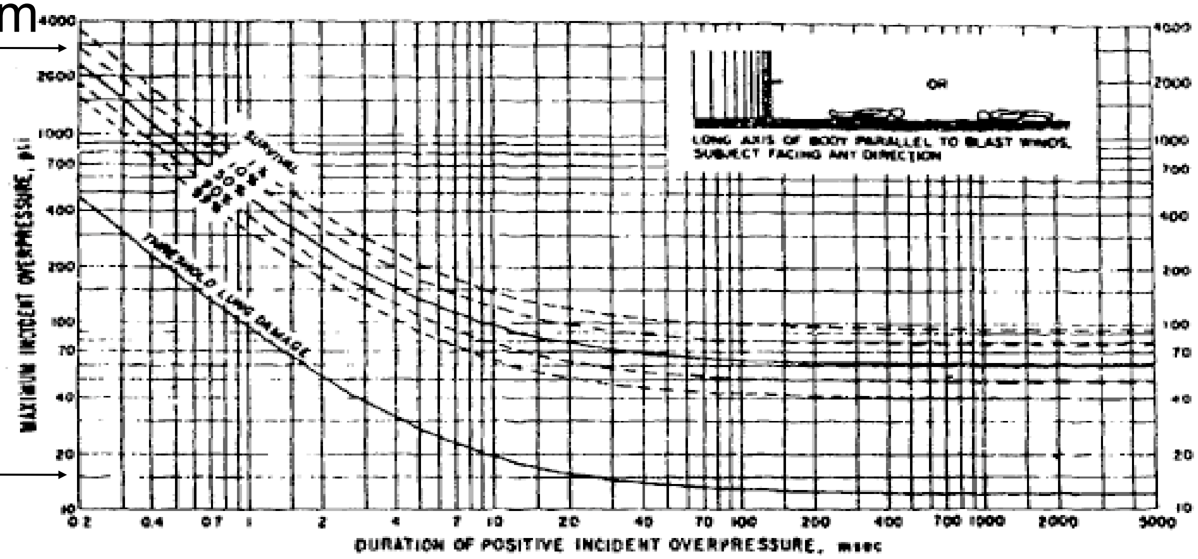
Overpressure Injury (empirical)

Blast Overpressure	Physiological Effect
0.2 psi (~0.01 atm)	Minor Ear Damage
1 psi (~0.1 atm)	Knock a Person Over
5 psi (~0.34 atm)	Eardrum Damage
15 psi (~1 atm)	Lung Damage
35 psi (~2.4 atm)	Fatalities Possible
65 psi (~4.4 atm)	Fatality Almost Certain

(Moss & King, personal comm.)

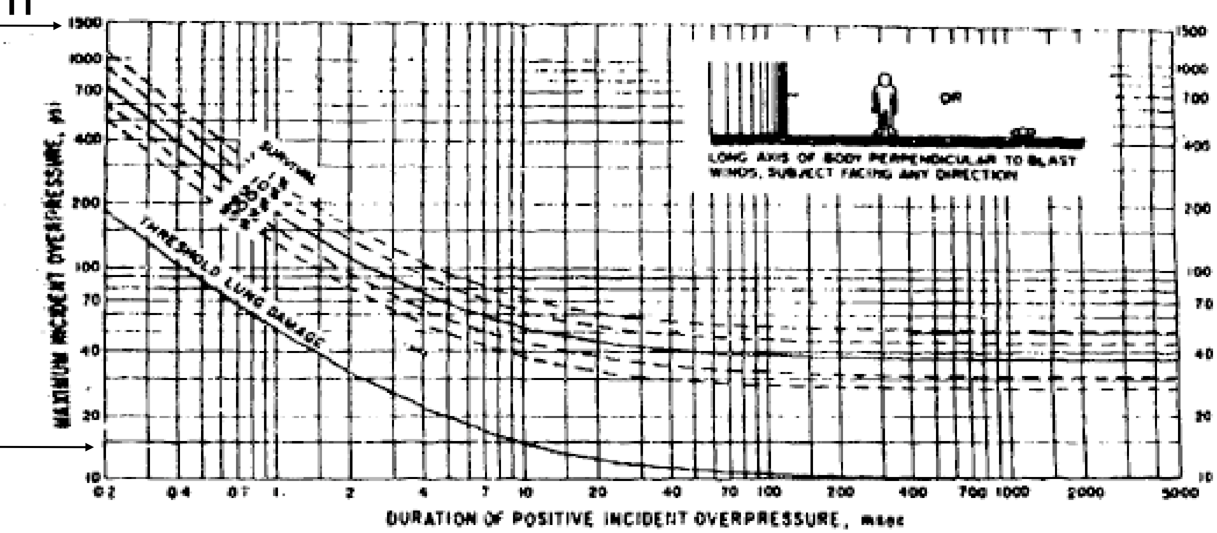
Outdated Bowen Curves: No TBI threshold

200 atm



1 atm

100 atm



1 atm

Bowen et al 68

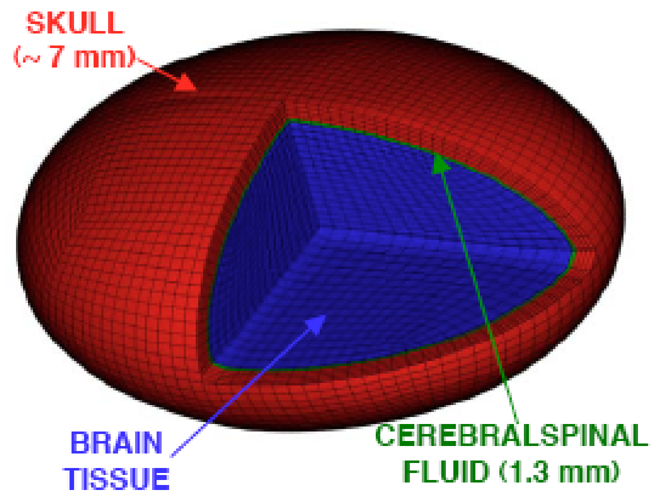
Simulations of Blast vs. Impact:

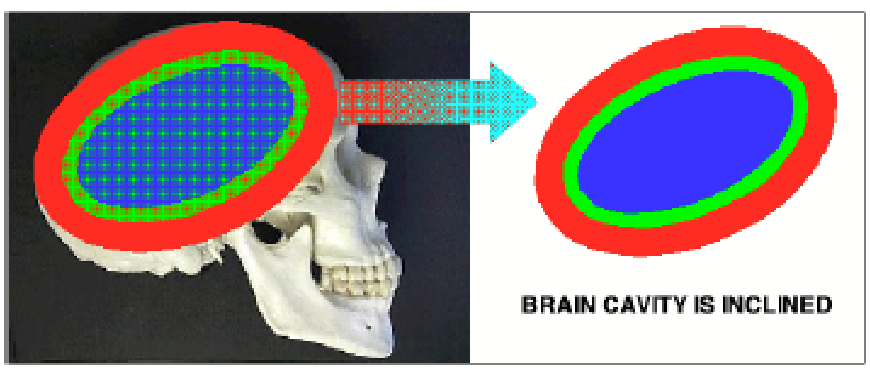
Moss, King, Blackman (2009)

ALE3D: LLNL's blast analysis code

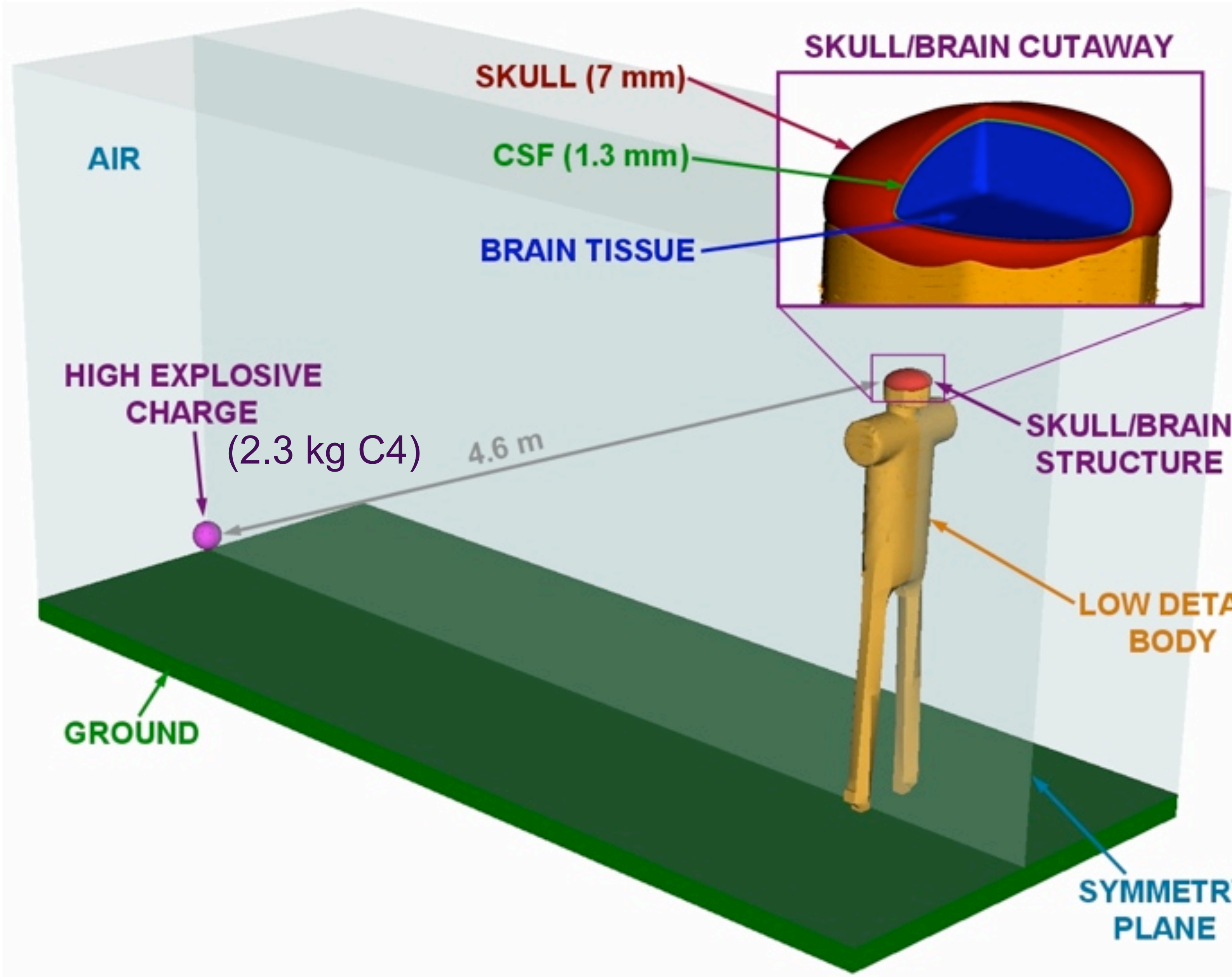
- Originally developed to support the nuclear weapons complex
- 3D Arbitrary Lagrangian-Eulerian Hydrocode
  - Advection capabilities
  - Built in methods for coupling fluid and structural interactions
  - Complex geometries
  - Massively parallel capabilities ← analysis with supercomputers
  - Rich material library
  - Thoroughly tested
- ALE3D is specially designed for studying the response of complex structures to blast

The “Head” in the simulations

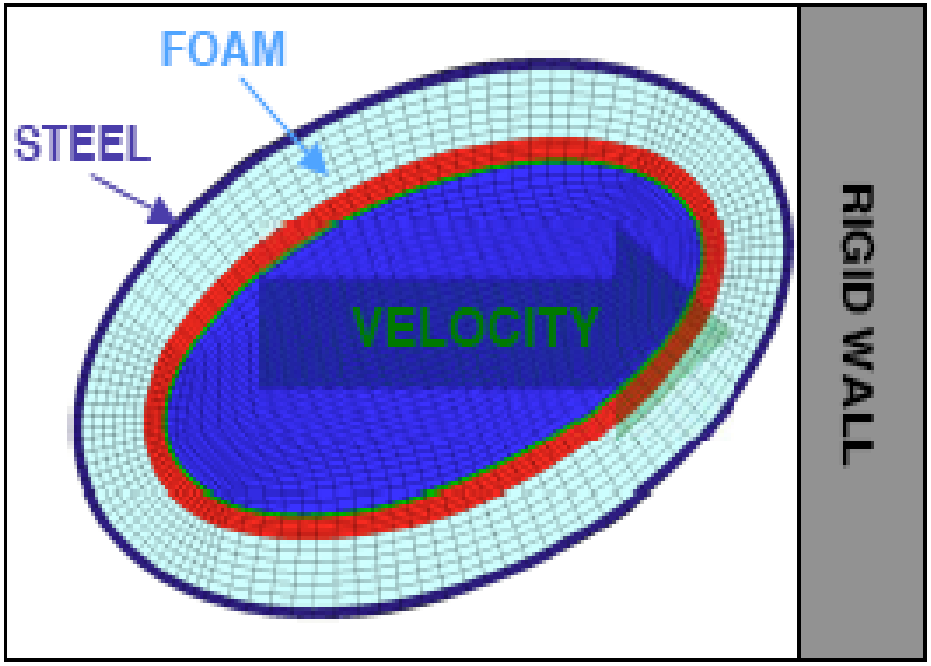




Brain cavity model


SKULL/BRAIN CUTAWAY
SKULL (7 mm)
CSF (1.3 mm)
BRAIN TISSUE
AIR
HIGH EXPLOSIVE CHARGE
(2.3 kg C4)
4.6 m
SKULL/BRAIN STRUCTURE
LOW DETAIL BODY
GROUND
SYMMETRY PLANE

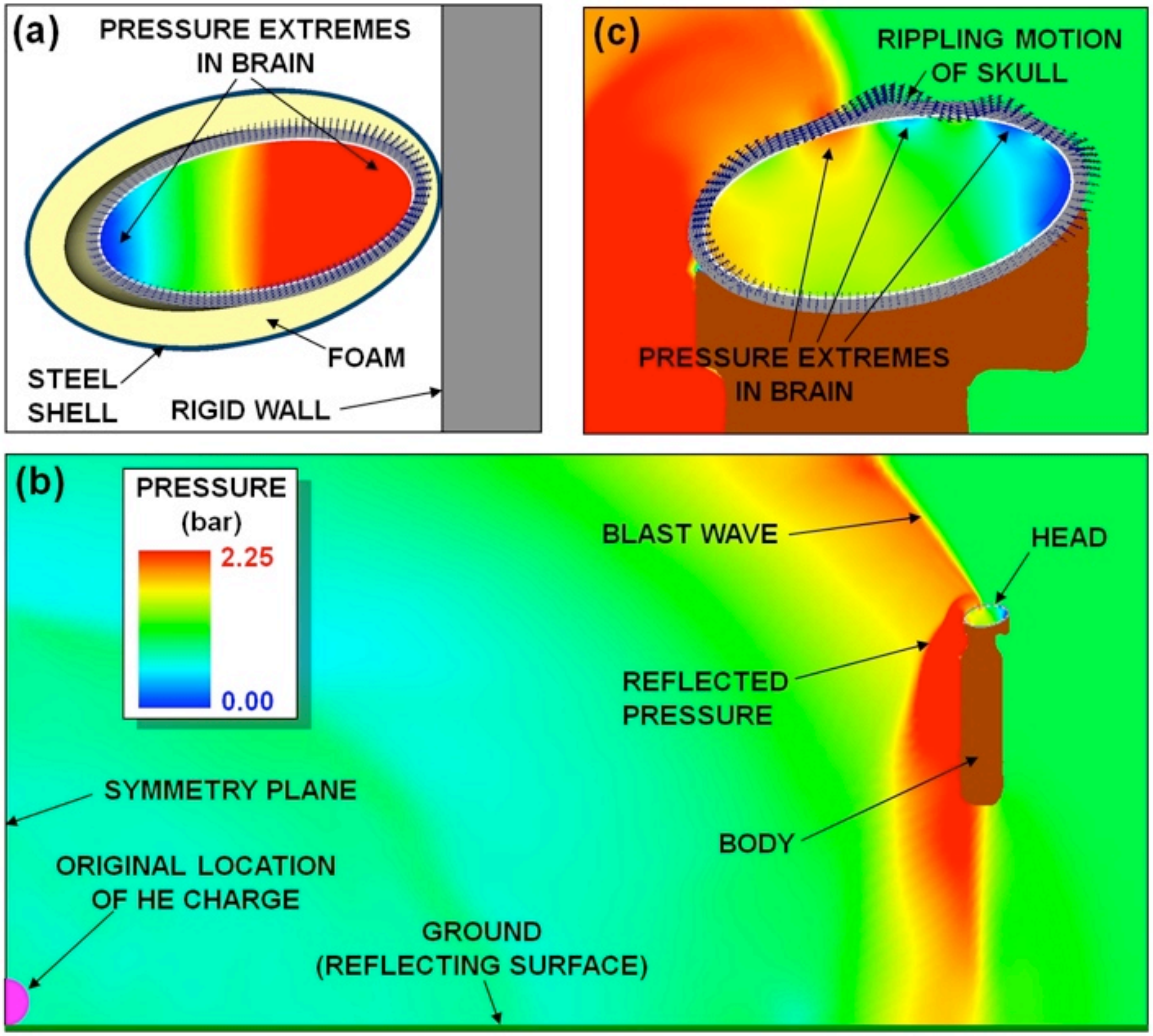
Model for Impact



Impact model with padded helmet (angled)

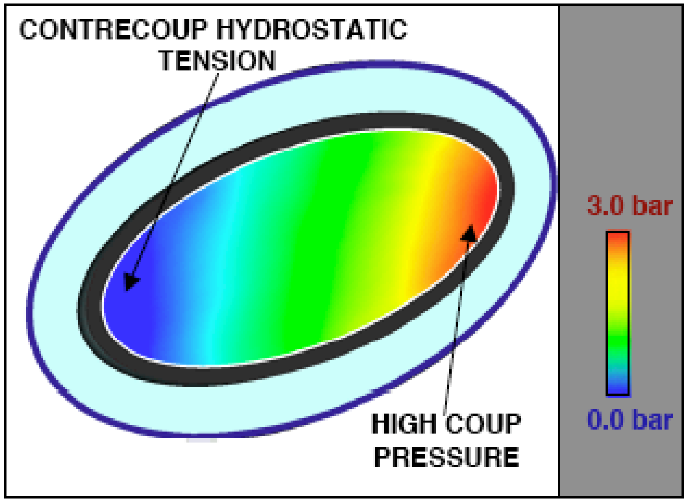
- HIC = 1090
- peak g 194 g
- impact duration 2.1 ms

Snapshot of Impact vs Blast Pressures

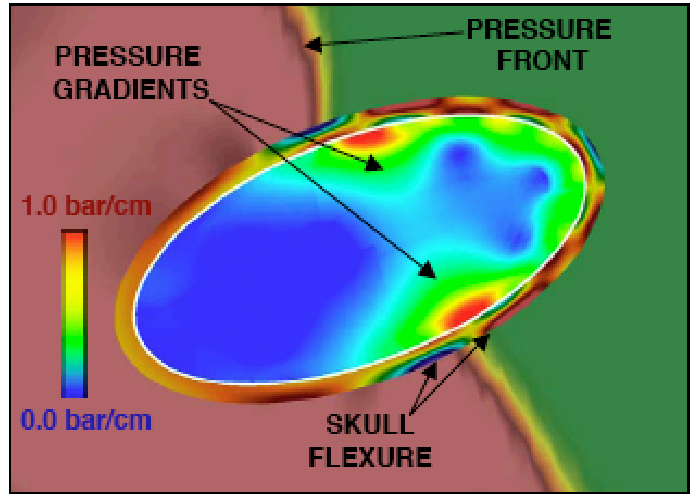


Blast wave at 5.6 ms after detonation

Impact versus blast



PRESSURES DURING IMPACT



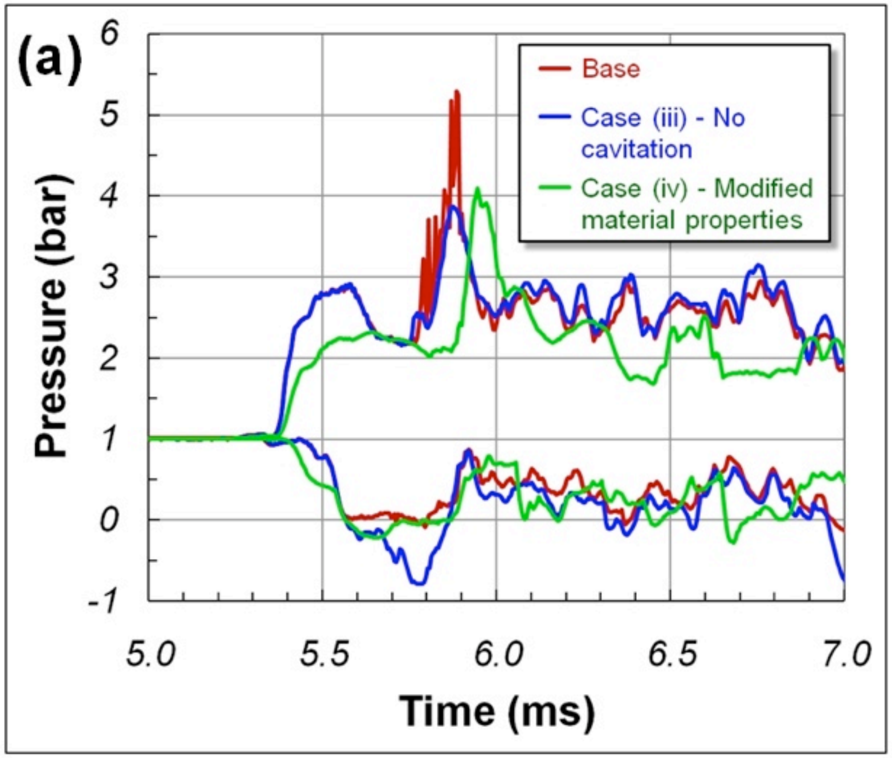
PRESSURE GRADIENTS DURING BLAST

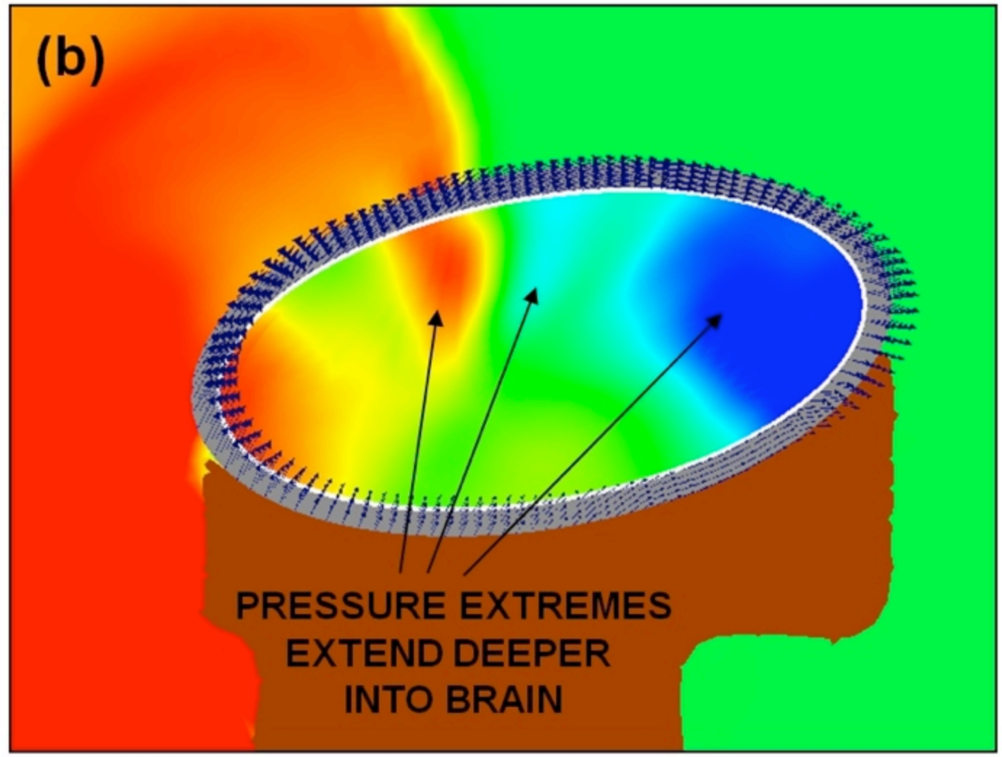
Impact

- Large linear accelerations
- Angled→large rotations
- Moderate skull flexure at ends
- High coup pressure
- Contrecoup tension → cavitation
- Small pressure gradients
- Rotation→large shear strains, bridging vein stretching

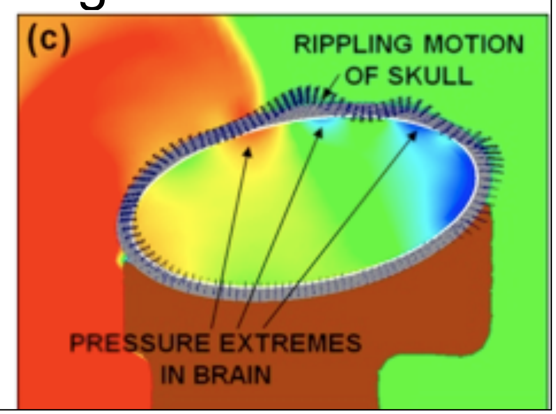
Blast

- Negligible linear acceleration
- Small rotations (more with whiplash?)
- Pressure wave → large lateral skull flexure
- Moderate coup/contrecoup pressure
- Hydrostatic tension → cavitation
- Skull flexure → large pressure gradients
- Rotation, pressure gradients → moderate shear strains



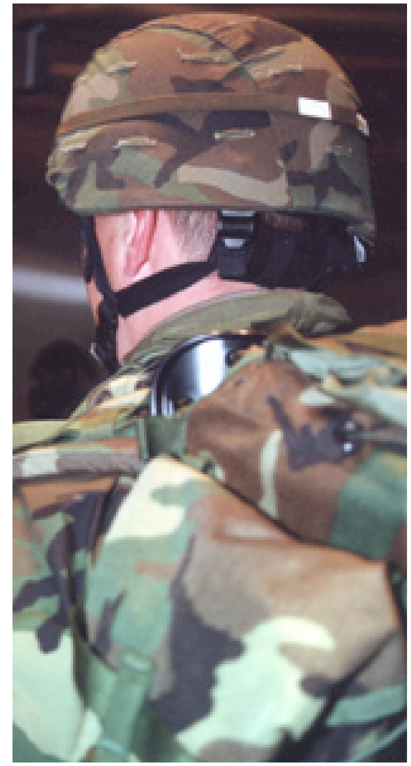


- Brain pressure peaks then followed by “after shocks” long after blast front passes
- Reducing wave speed in brain by reducing bulk modulus produces deeper penetration of pressure extremes as stress gradients are slower to relax



Advanced Combat Helmet (ACH) is based on the MICH design and prov
PASGT. In addition to providing the Soldier ballistic and impact protection,
communication devices, and Night Vision devices. It provides increased 9m
although reducing area of coverage, will improve the field of vision and hea
components: The helmet shell, the pad suspension system, the retention sy

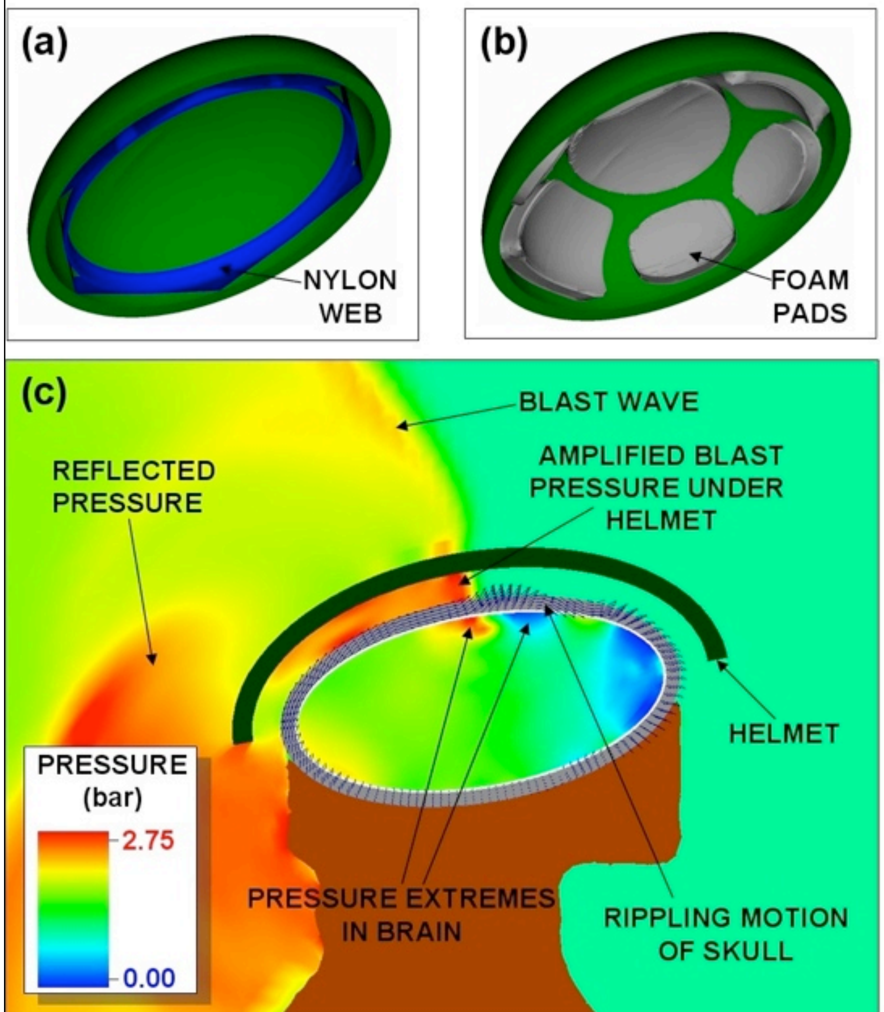
Modular Integrated Communications Helmet (MICH) is a lightwe
interface with most tactical communications headsets and microphon



Personal Armor System for Ground Troops (PASGT) helmet (also called the Kevlar, K-Pot and Fritz) wa: helmet, available in five sizes, provides ballistic protection for the head from fragmenting munitions. It is a c ballistic fiber and phenolic PVB resin. For a complete story, go to olive-drab.com

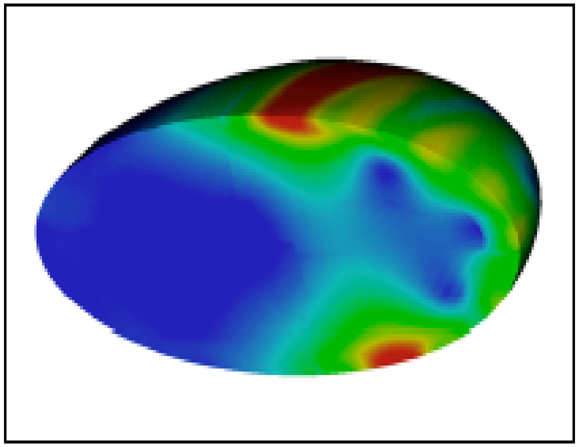
Ý This is the standard helmet above

Role of Current Helmets for Blast

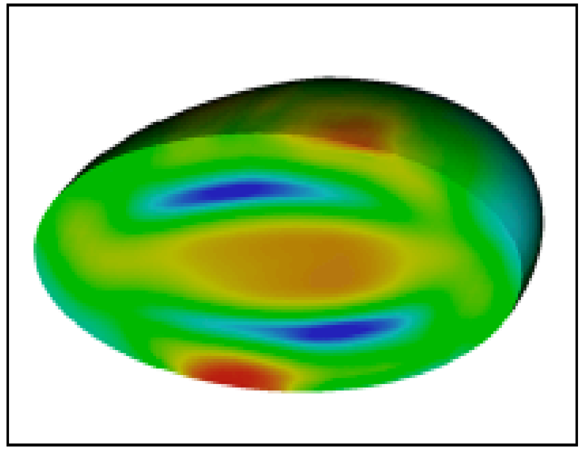


- without pads, “underwash” amplfies pressure under helmet: helmet without pads is WORSE than no helmet
- but, with overly stiff pads, head is more strongly coupled to skull and energy is not dissipated in the pads
- need to optimize pad and shell stiffness for both blast + impact

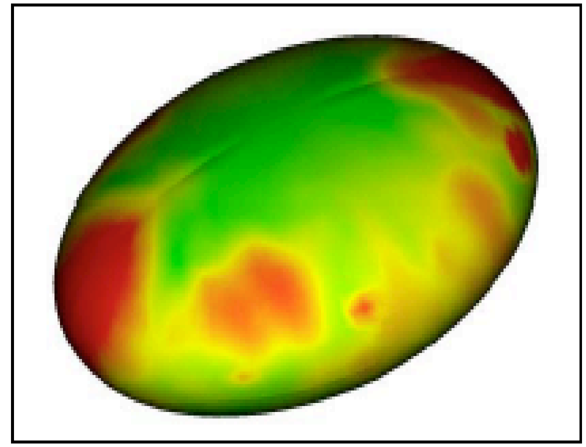
POTENTIAL DAMAGE METRICS FROM VARIOUS SIMULATIONS



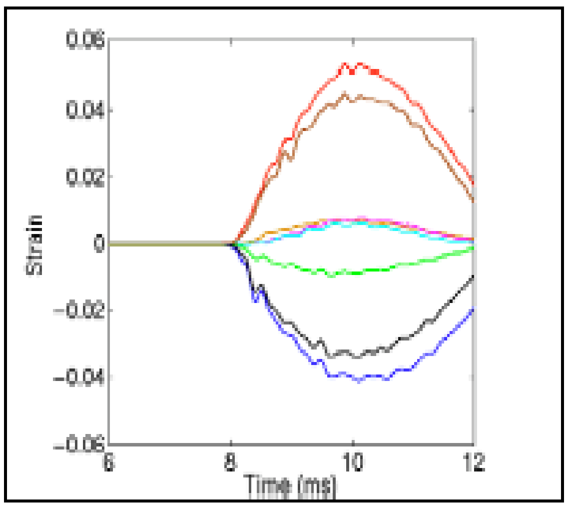
Pressure gradients in brain
HEMORRHAGING, DIFFUSE AXONAL INJURY



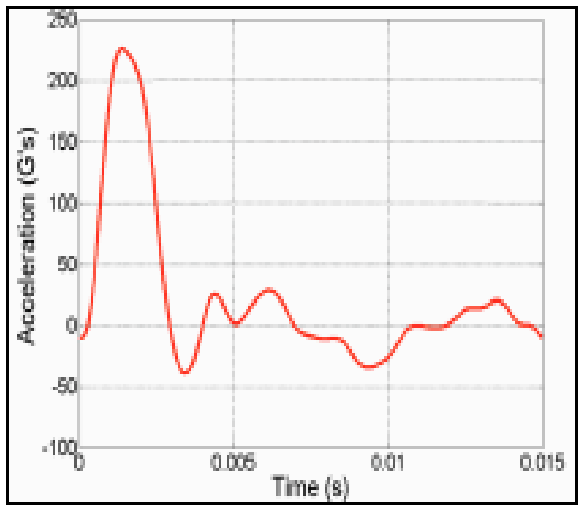
Shear strain in brain tissue
DIFFUSE AXONAL INJURY



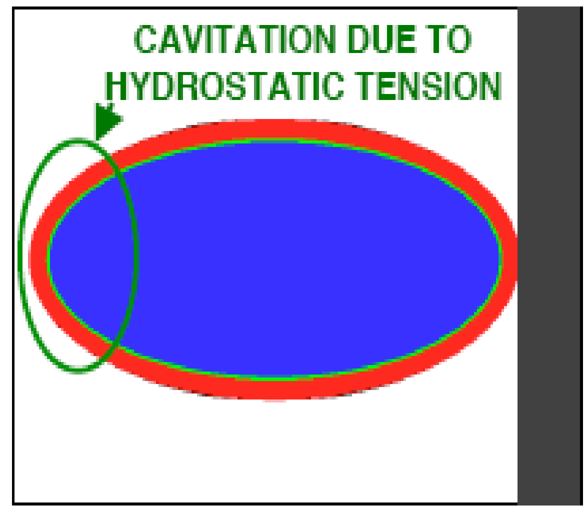
Peak pressures at brain surface
CONTUSIONS



Strain in bridging veins between brain and skull
SUBDURAL HEMATOMA



Acceleration of skull during impact
INDUSTRY DAMAGE METRIC



Hydrostatic tension, cavitation
CONTUSIONS/HEMORRHAGING

Moss et al. 09 .

Initial Lessons From Blast-Head Simulations

- Skull flexure NOT acceleration is primary mechanism of OTBI through skull
- need helmet that prevents skull flexure: e.g. a rigid shell + cushioning that damps the stress waves away from head
- under-wash in current helmets exacerbates injury
- current cushioning in use blocks under-wash but does not damp skull flexure effectively: not elastic enough
- need to optimize OTBI and ITBI protection
  - too much rigidity leads to more residual bulk acceleration
  - impacts are likely to follow non-fatal blasts

Future Studies

- BLAST over-pressure (OTBI)
  - Add more realistic head model
  - compare to other pathways that couple blast to brain (e.g. Cernak 01,05)
- IMPACT (ITBI)
  - consider impacts of different durations
  - include effect of body attached to head for the impact and vary impact with angle extract effective mass
- For BOTH:
  - correlate specific external forces with specific internal stresses
  - simulate helmet shells and cushioning to develop "intuition" and "principles" that guide material design to mitigate the internal stresses
  - run impact simulations for pre-injured brain from overpressure
  - correlate specific blast vs. impact history with with medical symptoms
  - correlate stresses with biological/biochemical changescorrelate stresses with biological/biochemical changes
  - integrate/test simulations with clinical studies where injury history, symptoms, and pressure acceleration data are available

Need Interdisciplinary Effort

- Pinning down quantitative thresholds for injury requires better in vivo measurements of tissue properties and correlation with clinical data
- Also need better material measurements
- BUT: let us not confuse “principles” with “parameters”: e.g. simulations are powerful tools and its easy to change the parameters
- Need iterative interplay between simulations and experiment to “benchmark” simulations

END