Biomechanics of Brain and Brain Stem Trauma – Protective Measures and MVAs

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Expert Opinions

- Biomechanics and Medical Analysis in regard to motor vehicle accidents
  a) Cause of injury or death – biomechanics and medical perspectives (loading trauma {forces}) v. a medical unrelated event- or simply naturally progressing pre-existing pathology

  b) Could the serious injury or death have been mitigated by protective equipment; motor cycle helmet, motor vehicle seat belt restraints.
FUNDAMENTALS OF MVAs  A quick review

- Contrast between a braking vehicle and a crashing vehicle- Measures of delta velocity and negative accelerations. It is the accelerations that produce the trauma to the occupants (including CNS Injury).
Hannon, 2006

(a) Braking car

\[ KE_i = \frac{1}{2} m V_i^2 \]

\[ KE = 0 \]
\[ Work = Force \times Distance = KE_i \]
\[ Remaining \ Energy = 0 \]

(b) Crashing car

\[ KE_i = \frac{1}{2} m V_i^2 \]

\[ KE = 0 \]
\[ Work = KE_i \]
\[ Remaining \ Energy = 0 \]
Hannon, 2006

A braking vehicle braking in a little over 3 seconds
Restrained vs. unrestrained occupants

- One issue many times addressed by the biomechanist in civil motor vehicle cases.
- Restraint non-use. A comparison is made usually between the actual (non-use) and the affect of using a two or more commonly a three point restraint.
- Usually a restraint is beneficial for the occupant- but each case must be judged individually
Approximate 159 gs applied to the occupant

Vehicle acceleration 19.8 g

Occupant acceleration 158.7 g over 10 ms

3.0 inches

25 inches

Car

Occupant

80 ms of veh crush

10 ms of occupant decel.
Hannon, 2006-08

Restrained Occupant - much lower g levels

Approximate 23 gs average

End of occupant deceleration

Still 80ms of veh. Crush

Occupant begins deceleration

35.0 mph (51.1 fps)

Time

Velocity

0 0.04 0.08 0.110 sec

25 inches

11 in

Still 80ms of veh. Crush

Occupant deceleration 22.7 g over 70 ms

8.0 inches of additional occupant deceleration after the vehicle stops

33 inches over ground

End of occupant deceleration
Seat Belts

- Use of a two or three point restraint during a motor vehicle accident - 2 questions
  1) Was the restraint in use?
  2) Would the restraint have been protective and reduced or eliminated injury?

- Physical belt evidence
- Biomechanical evidence
- Medical evidence
Steering wheel pattern injury
– No restraint in use
Less Evident Injury of a Driver Wearing a Heavy Coat
Seatbelt Abrasions of Driver-three point was in use
Brain and Brain Stem Biomechanics
Infant skull

- Parietal bone
- Anterior fontanel
- Coronal suture
- Frontal bone
- Posterior fontanel
- Occipital bone
- Mastoid (posterolateral) fontanel
- Temporal bone
- Nasal bone
- Sphenoid bone
- Zygomatic bone
- Maxilla bone
- Mandible bone
The Adult Skull

- The skull bones each consist of a thick outer layer or table of bone, the spongy *diploe* and the thinner *inner table*.

- The base of the skull only contains a very thin (1-2 mm) *inner table*. 
Protective Factors Against Impact

1. Scalp
2. Compact bone $\frac{1}{8}$ to 3/8 inch thick
3. Duramater
4. Cerebral spinal fluid
5. Brain may be viewed as an incompressible, deformable liquid which has viscoelastic properties
Scalp layers

- Skin
- Connective tissue
- Aponeurosis
- Loose connective tissue
- Periosteum (pericranium)
MRI of head enhancing the appearance of the scalp

The scalp functions as a “soft helmet”
Membrane Coverings

- The brain and spinal cord are protected by three membranes:
  - **duramater** (thick, adherent to skull bone)
  - **arachnoid**
  - **piamater** (thin, in direct contact with the brain)

- The piamater and arachnoid are designated as pia-arachnoid

- **Subdural space**: the small space between the inner surface of the duramater and the thin membranous arachnoid

- **Subarachnoid space**: lies between arachnoid and pia and contains cerebrospinal fluid
Mechanisms of Brain and Brain Stem Injury
Mechanisms of Brain Injury II

Another approach but compatible with the previous flow chart

- Penetration
- Acceleration - due to impact or inertial loading
  - Linear
  - Angular
- Energy wave propagation
- Examples
Penetration mechanism

- Penetration is affected by load distribution.
- With increased load distribution and a consequent reduction in compressive stress, penetration becomes less likely or the displacement magnitude of the penetration is decreased.
- This is the function of the skull, a construction worker’s hard hat and one of the functions of a helmet.
Penetration: Phineas Gage

Frontal view

Anterior

Posterior
Penetration Injuries

The *Duramater* is a gray membrane of connective tissue attached to the inner surface of the skull. Arteries run along inner surface. Contusion-hemorrhages along these arteries (on top of duramater) are called epidural hemorrhages.
Fracture of the skull (left side of picture) ruptures small arteries which pushes away duramater from the bone. The epidural hematoma is formed.
Epidural Hemorrhages

- Epidural Hematomas are rare in general.
  - Due to trauma to skull especially when followed by fractures of the skull.
  - Most common in falls and traffic accidents.
  - Infrequent in elderly and young because their duramater is strongly adhered to the skull.
Epidural Hemorrhages

- Usually unilateral and in the temporal region.
- Symptoms occur 4-8 hours after injury.
- Death due to displacement of brain with compression of the brain stem.
- Usually have thick, disk-shaped appearance.
2009 rock climbing case-epidural bleed
Direct Impact- Middle men. artery

No helmet
Worn
Rock climbing incident 2009
Seventeen-year-old male patient status post-motor vehicle accident and seizure. CT (A) reveals an epidural hematoma in the left frontal region. Companion xenon-enhanced CT (B) shows a focal perfusion defect corresponding to the hematoma. Diffuse globally decreased perfusion is clearly present on the slice immediately above the hematoma (C). In the uppermost slice (D), blood flow is globally somewhat decreased with a more severe deficit on the side of the hematoma.

Source. Case contributed by Dr. Susan Weathers, Baylor College of Medicine.
Insults to the Brain Resulting from Linear Acceleration

1. Contusion- coup injury
2. Contrecoup- cavitation
   - Resulting in subdural hematoma
     - Artery bleed
     - Venous bleed
     - Subdural and/or subarachnoid bleed
     - Midline shift- pineal gland
     - Extrusion of the brainstem through the foramen magnum termed brain herniation (patient herniates)
Contracoup contusions most common when a moving head link is brought to a stop.

Arrows indicate contact point and direction of applied force. Shaded areas show location of contusion.
Contrecoup Contusions

Primate model-experimental loading
Monroe-Kellie doctrine
Result of increased intra-Cranial pressure

Figure 7.4. This frontal view of a monkey brain demonstrates the massive amount of acute brain swelling that can occur after the removal of an acute subdural hematoma. The "mushroom" over the frontal lobe (left) is swollen brain that protruded through the craniotomy defect moments after.
Tolerance Limits for Linear Acceleration
Head Injury Criterion

- Only used for head impacts to 35 msecs-predominant loading
- HIC of 1000 ≈ 60Gs over a time period of 35 milliseconds
- Rear impacts- head to headrest will not cause this injury
  - Rear impacts required $\Delta$ velocity
  - Stiff seat 54 mph $\Delta$ vel-Holloman AFB
  - Soft seat 39 mph $\Delta$ vel
- Both estimates above assume elastic rebound and no failure point for the seat back
- Revised HIC over 15 milliseconds time epoch
Mean Strain Criterion

Further development of the MSC approach has led to the Translational Head Injury Model (Stalnaker 1987)- However, this energy-displacement approach to brain injury at present has not gained wide acceptance.
Mean Strain Criterion

- Stalnaker 1971 - A lumped parameter model approach -
- Looks at the relative displacement of two masses divided by the diameter of the cranium -
- Strains resulting from stresses can be compared in the experimental model and in actual injuries - then run the correlation!
Hookes' Law applied to springs

Two dashpots as per the revised MSC
Insults to the Brain resulting from Angular Acceleration
Figure 25.1. (A) Schematic representation of the effect of rotational acceleration of the head upon the brain. (B) Due to inertia, the brain stays initially behind the rotational momentum of the skull. Thus a strain is placed on the bridging veins (V) that may disrupt them. (From F. Unterharnscheidt and K. Sellier.)
Figure 25.5. (A) The boxer is shown before the fight. (B) The right side of the face is hit in this lightweight boxing match. (Associated Press.)
Figure 25.6. A boxer is seen whose face is totally distorted by the enormous force of the blow. (United Press International.)
Figure 25.8. In a lightweight fight a stiff right from the right fighter milliseconds after connecting with the left side of the other’s face shows the effect. (United Press International.)
Figure 25.7. This welterweight connected with a sharp right to the opponent’s face, distorting his features in the seventh round of the fight. The force of the blow can be imagined from the sweat spraying from the fighter's head. (Associated Press.)
Figure 25.9. In an international middleweight fight the boxer on the left won the fight in the sixth round by a knockout. The photograph shows the extensive rotational acceleration of the head and hyperextension of the victim’s cervical spine due to a hard blow by the opponent.
Note the affect of padded gloves—both linear and angular acc. are increased.

Also—note—numerous knockouts in MMA fighting—reason being!!

**Figure 25.2.** This table shows the measurements of acceleration using boxing gloves of different weights (ordinate, acceleration expressed in g; abscissa; ounces of gloves). In serial testing, the gloves’ material showed “fatigue” or “fading” of its elastic qualities, and consequently the acceleration measurements rose. (From F. Unterharnscheidt and K. Sel-lier.)
Human Tolerance: Head Link
Angular Accelerations

Tolerances:
- Ommaya ≈ 1,700 rads/s²
- Gennarelli ≈ 1,800 – 2,800 rads/s²
  - Concussion at 500 – 600 rads/s²
- Muzzy 1976 ≈ 1,800 rads/s² not uncommon-yet without injury
- Picemalle et al.- boxers reach 5 -6K rads/s² for a very short duration- over 3-5 msec- no injury!!!-
  Linear accelerations were short duration but in the range of 130-159 gs!

Newer data- to discuss
Results of high levels of Angular Acceleration
Subdural Hematomas (SDH)

- Sudden acceleration of the head (during impact) causes movement of the brain relative to the duramater. Linear or angular acceleration!

- Contusions will produce bleeds

- Angular acceleration shears and tears the small veins which bridge across the gap between the duramater and the cortical surface of the skull.

- The leaking blood accumulates over several hours and usually tracks extensively as a film over the surface of the brain.
Ommaya – Bridging veins tolerance @ 4500 rads/sec² ???

Duramater and bridging veins
Subdural Hematomas

SDH is especially common in:

- the elderly (brain atrophy widens the gap),
- children (“shake and strike” injury as part of the child abuse syndrome); and
- alcoholics (frequent unprotected falls and prolonged bleeding times).

- A small, self-limiting SDH may remain asymptomatic and be an incidental finding at autopsy.
Subdural Hematomas

- The most common LETHAL injury associated with head trauma.
- Can also be caused by cerebral aneurysm or intra-cerebral hemorrhage.
- Often includes countercoup contusions.
Subdural Hematomas

- Often, NOT associated with skull fractures
- Onset of symptoms is usually rapid - Arterial bleed or the large bridging veins
Subdural Hematomas

Three categories of Subdural Hematomas:

- **Acute** - Manifest within 72 hours of injury.
- **Subacute** - Manifest within 3-4 days to 2-3 weeks of injury.
- **Chronic** - Manifest over 3 weeks after injury.
Subdural Hematoma - the skull is opened, exposing the hematoma underneath the thick membrane called dura (arrow)
Subarachnoid Hemorrhage

- Most common type of trauma to the head.
- May be:
  - focal or diffuse,
  - minor or severe.
- A Subarachnoid hemorrhage in itself can cause death. May be due to linear or angular acceleration applied to the brain.
Subarachnoid Hemorrhage

- May be natural, due to rupture of a dilated blood vessel (an aneurysm), or traumatic.
- SAH is highly irritant to the brain-stem and is usually rapidly fatal.
Subarachnoid Hemorrhage

- The most rapid deaths are *multifocal*, but most cases are diffuse injuries with minimal pooling on the cortical surface of the brain.
- Large collections of blood in Subarachnoid space are more common in natural diseases than in trauma.
Subarachnoid Hemorrhage

- Most Subarachnoid hemorrhages have origins in the veins.
- May be produced postmortem, secondary to decomposition.
- May also be produced while the coroner or medical examiner is removing the brain during autopsy.
Energy Wave Propagation
head injury mechanism

- Not common in motor vehicle accidents
- Golfing- Injury-1996

- A miss hit with the bottom edge of an iron club

- Head Impact with a line drive impact- high parietal bone resulting in a fatality within one week of the accident
Case Study: Energy wave propagation

- The neurosurgeon was told on the phone about the existence of a film of subdural hematoma—not able to transmit the image to the surgeon.
- In the first 24 h after injury the simple CAT scan did not show the areas with low blood flow in the brain.
- Provided that the surgeon would have repeated the scans, it would have shown the area of energy wave propagation injury damage.
Notice the very small scalp contusion initially
Very small subdural hematoma of the occipital region
This area of the brain looked almost normal in this coronal plane slice.
Note the deep damage (KE) at autopsy
Energy Wave propagation injuries may be invisible during the initial exam.

- In this case the wave propagation injury did not manifest grossly until more than 4 days after the trauma occurred.
END OF PRESENTATION