TREATMENT CRANIOCEREBRAL TRAUMA

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OBJECTIVES

• Review management of mild, moderate, severe head injuries
• Review current recommendations for treatment, based on AANS Brain Trauma Foundation Guidelines
• Understand treatment recommendations based best evidence
• Understand the physiologic events leading to secondary neuronal injury after traumatic brain injury (TBI) as well as the advances in the care of critically ill patients
TRAUMA

- Leading cause death to age 44 years of age
- Ages 5-34 years more deaths than all other diseases combined
- MVA 31%, Suicide 21%, Homicide 14%
- MVA cause 50,000 deaths/yr
- 66% bicycle deaths occur in Age group 5-14 years
TRAUMA TRIVIA

- 2/3 fatal injuries are accidental
- 70% trauma fatalities occur in rural areas
- 20-30% decrease in mortality due to EMT, pre-hospital system, helicopter.
EMERGENCY MEDICINE ISSUE

- Head injury occurs every 7 seconds in USA
- Patient dies of head injury every 5 minutes
- 60% all trauma deaths have head injury
- >70% vehicular trauma deaths due to head injury
TRAUMATIC BRAIN INJURY

- Each year ~ 2 million people suffer TBI
- ~100,000 die and 90,000 left with long term disabilities
- Over 500,000 require hospital admission/yr
- 20% will exhibit some level of disability following recovery
- 10-20% graded as severe
- Annual cost of caring for TBI - $25 billion
Worldwide >500,000/yr die

Equivalent jet crash killing 1,350 people every day!

USA - MVCs killed >2.8 million people since 1900!
TRAUMATIC HEAD INJURY - CLASSIFICATION

- Minor head injury – GCS 13-15
- Moderate Head Injury – GCS 9-12
- Severe Head Injury – GCS 8
- Head Injuries divided into focal and diffuse
  - **Focal Injuries** - Fractures (linear, depressed, basilar)
    - Hematomas (SAH, epidural, subdural, intracerebral)
    - Brain contusions, lacerations
  - **Diffuse Injuries** – Concussion
    - Diffuse Axonal Injuries (mild, moderate, severe)
GLASGOW COMA SCORE

- Not statistically affected by alcohol until BAC >200mg/dL is reached

- **Severe head injury or coma = GCS <8**
  - overall mortality 40%

- **Moderate head injury = GCS 9-12**
  - Mortality 20% but morbidity is substantially higher

- **Minor head injury = GCS 13-15**
  - up to 10% patients with GCS of 15 have an abnormal CT
  - 1-3% of those get a craniotomy
PRIMARY INJURY

• Occurs at impact
• Continues at the scene
• Direct mechanical damage to neurons, dendrites, axons, blood vessels, myelin sheaths
• Focal impact vs. deceleration
PRIMARY INJURY AT COLLISION

• **3 separate collisions**
  1. Initial collision of vehicle
  2. Impact of the occupant vs. vehicle interior
  3. Impact of occupant’s organ vs body
A. Hyperflexion

B. Axial Loading

C. Hyperextension
SECONDARY INJURY

- Complications intracranial/ extracranial injury
- Delayed in onset
- Hypoxemia/ischemia
- Global vs. Focal
- Systemic physiologic responses to initial injury
- Number of biochemical substances released play a role in propagation of neuronal injury following TBI
- Substances include excitatory amino acids glutamate and aspartate, cytokines and free radicals
From the time primary injury to onset secondary injury, a window of time exists for susceptible tissue to die or survive!

Some cells that are only partially injured and have potential for recovery
MECHANISMS SECONDARY INJURY

- Hypoxia
- Hypotension
- Apnea
- Intracranial Hypertension (ICP)
- Seizures
- Cerebral Edema
- Hyperglycemia
BRAIN’S RESPONSE TO TRAUMA

- Edema
- Hyperemia
- Ischemia
- Loss of Cerebral Autoregulation
- Release of neurotransmitters
Secondary Injury — Miller et. al, JAMA 1978

1. 35% Hypoxic (PaO2 < 65mm Hg)
2. 15% Hypotensive (Systolic BP < 95mm HG)
3. 12% Anemic (Hct <30%)

ATLS
Secondary Brain Injury – Jones et.al., J. Trauma ‘95

1. ICU admission does not eliminate risk secondary injury
2. Jones demonstrated in 124 patients, while in ICU
   - 40% Hypoxic (PaO2 < 60 torr)
   - 73% Hypotensive (SBP < 90mm Hg)

While in ICU!
MILD HEAD INJURY

- GCS 13-15
- **Concussion Definition**: temporary neurologic and/or cognitive impairment
  - can be due to direct or indirect force
  - usually associated with the acute onset of symptoms, but symptoms may evolve over minutes to hours
- **Symptoms of acute concussion**: any neurologic and/or cognitive impairment of any kind including headache, sleep disturbance, phonophobia, photophobia, dizziness, weakness, fogginess, difficulty concentrating, memory loss, more irritable or emotional than baseline, unsteadiness or ataxia
- Concussion are cumulative
- 35% of college football players with concussions had no impact to the head
- Players sustaining a concussion have 4-6X rate of sustaining another concussion
SPORTS HEAD INJURIES

Concussions giving NHL big headache

RICH STROM
Chicago Tribune

Wednesday, March 7, 2001

Cowboys waive Aikman after 12 seasons, 10 concussions, 3 Super Bowls
By JAIME ARON
AP Sports Writer

Steelers/NFL
Update on Concussions & the NFL: Medicine fast framing theories with hard data
Sunday, June 17, 2007
FOOTBALL HEAD INJURIES

• Concussions evenly divided between practices & games
• Only 30% players do not return to play same day
• 35% concussions in college football had no impact to head
• Player receiving concussion 4-6X more likely to sustain another
• High school players sustaining concussions, 20% have multiple
NFL Concussion Case

I have concerns about the fairness, reasonableness, and adequacy of

Judge Anita Brody

NFL Pay $765 Million To Settle Concussion Case, Still Wins

The NFL has settled the lawsuit that could have destroyed it.

And the price? A pittance: less than 0.5% of the NFL’s annual revenue, to be paid out over
Judge Anita Brody denies preliminary approval for NFL Concussion Settlement

On Aug. 29, 2013, the NFL and over 4,000 former players who had sued the league agreed to a settlement totaling $760 million. The lawsuits stemmed from the contention that the NFL knew about the dangers of on-field head injuries long before it did enough about them, and that those players affected have not been helped enough in their post-football lives. The settlement came after more than two months of intense negotiations, and was given to Judge Anita Brody for preliminary approval.

On Tuesday, Judge Brody denied that preliminary motion, stating in her ruling that she was concerned with a lack of documentation regarding the fairness of the final monetary figure, and whether the players involved would be diagnosed and paid properly based on their claims.

As Judge Brody also wrote, players diagnosed with head trauma-related illnesses would be eligible for fixed monetary awards — $1.5 million for Level 1 Neurocognitive Impairment; $3 million for Level 2 Neurocognitive Impairment; $3.5 million for Alzheimer’s Disease; $3.5 million for Parkinson’s Disease; $5 million for ALS; and $4 million for Death with CTE. While it may seem cold to attach numbers to such horrible circumstances, class-action cases are often partitioned as such.
LAS VEGAS (AP) -- Veteran boxer Leavander Johnson underwent brain surgery and was in critical condition Saturday night after collapsing in his dressing room following his IBF lightweight title loss against challenger Jesus Chavez.

Johnson's promoter said doctors told him the fighter's brain swelled during surgery and they were inducing a coma to try and control it.

"He's not in good shape," Lou DiBella said.
Fight not stopped until 11th round?

“Basically he was fine? He was alert....I talked to him and there was no change in his neurological state.”
CEREBRAL BLOOD FLOW

- Brain 3% total body wt – 20% C.O.
- N. CBF – 50-65ml/100g/min
- Cerebral autoregulation = constant flow
- BP decreases – vessels dilate
- EEG slowing @ 25ml/100g/min
- Cell Death @ 10-18ml/100g/min
- TBI – autoregulation impaired
  (mechanical, hypoxia, hypercarbia, swelling)
CEREBRAL PERFUSION PRESSURE

- Gradient between Mean Arterial Pressure (MAP) and ICP (resists inflow blood)
- N. ICP is negligible (CPP = MAP)
- Changes CPP (60-150 mm Hg) no effect on CBF
- Increase ICP can lead C. Ischemia when it leads to reduction CPP
- N. CPP ~ 90 mm Hg, need >70 mm Hg to prevent ischemia
Compartment Syndrome

N. Cranial Vault – 500-700mls Neurons
700-900mls Glia
100-150mls CSF
100-150mls Blood

Bone & Fascial Limitation

Falx
HEAD INJURY HEMODYNAMICS

- Autoregulation impaired
- Hypoxia/hypercarbia
- Extravasation Blood/Fluid
- Brain Swelling/Edema
- Vasospasm

RESPONSE

- Increase volume 1 component (edema, clot)
- Compensation occurs:
  1. CSF divert out skull
  2. Ventricles compress
  3. Basal Cisterns absent
ICP

Intracranial Mass Volume

Herniation

Equilibrium

Compensated

Uncompensated
MANAGEMENT RAISED ICP – 4 R’S

• **Reduce** brain size- mannitol, hypertonic saline
• **Reduce** CSF – ventriculostomy
• **Reduce** Blood Volume – hyperventilation? – vasoconstriction
• **Removal** surgically pathologic process
• **Removal**-portion of skull

Alternatively – open skull allow expansion of structures

DECOMPRESSIVE CRANIECTOMY
STEP II

- Mannitol 0.25G – 0.5G/kg or hypertonic saline 2ml/kg of 7.5% saline
- Intermittent boluses, not continuous
- Plasma osmolarity <315mOsm/kg
- Mannitol decreases blood viscosity, expands intravascular volume, increases CBF
- Early high dose Mannitol 1.4/Kg maybe more effective
- Further study – ongoing?
An inert 6 carbon alcohol of the corresponding sugar mannose, causes cellular dehydration by increasing serum osmolarity.

More recent studies have shown that mannitol’s primary mechanism of action is its rheologic effect on cerebral blood flow (CBF) even in cases in which it has only minimal effects on ICP (in other words, it dilutes the blood and increases the deformability of erythrocytes).

Use of mannitol to control elevated ICP in severely head-injured patients is a level II recommendation.

Especially effective when CPP is below 70 mmHg.

Additional level III recommendations include administering bolus doses of mannitol, replacing urinary losses of fluid, and maintaining serum osmolarity below 315-320 mOsm.

To avoid the risk of sudden hypotension from rapid infusion of mannitol, it should be bolused at rates of 1 g/kg or less.
HYPERTONIC SALINE

• Decreases ICP without adversely affecting hemodynamic status
• Possible beneficial effects on excitatory neurotransmitters & immune system
• Recent randomized study comparing mannitol vs. hypertonic saline (7.5%) in 20 pts, HI, ICP, Coma, Saline more effective reducing ICP
• More studies larger numbers needed to confirm or refute?
ICP – 3 STEP TREATMENT

• Step 1 – If ICP > 20 mm Hg slight hyperventilation or least avoidance hypercapnia (PCO2 >40 mmHg)
• Target PCO2 35mm Hg
• Increases pH CSF, produces arterial vasoconstriction, increase CV resistance diminished CBF, Blood Volume
• Drain CSF - ventriculostomy
SCOOP ON HYPERTONIC SALINE

- Hypertonic saline clearly reduces brain bulk and ICP
- Early studies comparing hypertonic saline to 20% mannitol for reduction of brain bulk and lumbar CSF pressure, two regimens had equal efficacy (Class II data)
- Numerous studies have demonstrated the efficacy of hypertonic saline in reducing ICP, but these studies are not directly comparable because the designs of the studies vary so greatly and because the doses, compositions and infusion regimens of the saline solution differ substantially
- Insufficient evidence to recommend specific protocols for using hypertonic saline
THIRD STEP

• If ICP remains high, Barbiturate therapy?
• Barbs decrease CMR, may limit free radical-mediated cell injury
• Barbs can induce hypotension, decrease myocardial contractility
• Decompressive Craniectomy? Unilateral vs. Bilateral
LIMITATION GCS

- Intubation & orbital swelling
- Sedation & neuromuscular blockade
- Lower end score – motor most NB.
- Upper end score – account for degree stimulation?
- Needs to be repeated and documented
- GCS change is hallmark of deterioration
- Pediatric Score?
INDICATIONS FOR INTUBATION/VENTILATION

- Coma & GCS <8 (ATLS protocol)
- Loss protective laryngeal reflexes
- Ventilatory Insufficiency:
  - PaO2 <60 mm HG
  - PCO2 >45 mm Hg
  - PCO2 <26 mm Hg
- Respiratory arrhythmia
- Loss of airway in transport
INDICATIONS INTUBATION (RELATIVE)

- Before transport within/between hospitals
- Deteriorating LOC
- Copious Bleeding into mouth
- Seizures
- **VENTILATE!!** - PaO2 >100 mm Hg
  - PCO2 >32 mm Hg (1st 24 hrs)
ALTERED MENTAL STATUS

- ABC’s
- Thiamine, Glucose, Naloxone, O2
- A – alcohol
- E – epilepsy
- I – insulin
- O- Opiates
- U- Uremia

- T – trauma, temp
- I – infection
- P – poisoning, psyc
- S – stroke, shock
HYPERVENTILATION

- Avoid prophylactic HV
- CBF < 50% normal in 1st 24 hours (<30cc/100g/min)
- HV – vasoconstricts further reducing CBF
- 1mm Hg - 3%, CBF = 1.1 ml/100g
- 2-4mm Hg PCO2 - ICP by 1mm Hg
- CPP maintained at 70mm Hg – NO HV
The use of prophylactic HV (PaCO2 < 35 mm Hg) during first 24 hrs after severe TBI should be avoided because it can compromise CPP during a time when CBF is reduced.

HV may be necessary brief periods for neuro deterioration.

Longer periods – ICP refractory to sedation, paralysis, osmotics, CSF drain.
ICP MONITORING

- Normal ICP 0-10mm Hg
- 20mm Hg upper limit normal
- Adequate cerebral perfusion pressure (CPP) more NB than ICP
- CPP = MAP – ICP
- Treat hypotension aggressively!
- Aim to keep CPP > 70 mm Hg

HA
AMS
N, V
Papilledema
Visual loss
Cushing Triad
RECOMMENDATIONS ICP MONITORING

- **Severe HI + Normal CT + > 2 criteria**
  1. Age > 40yrs
  2. Posturing Uni/bilateral
  3. SBP <90mm Hg

- **Severe HI + ABN CT with**
  1. Contusion, hematoma, Edema
  2. Compressed Basal Cisterns
ANTICONVULSANTS

- Ineffective preventing late onset SZ
- Reduces incidence early SZ (<7 days)
- Early SZ increase risk late SZ
- Closed HI – 5% early SZ, 5% late
- Temkin et. al. – NEJM ‘91

Long term cognitive impairment
- Hematomas, penetrating,
ANTICONVULSANTS EVIDENCE

- Literature contains reasonable evidence that frequency of early sz after brain injury is reduced by administration of antiepileptic drugs (AEDs).
- Approximately 20-25% patients who sustain serious head injuries can be expected to have at least one PTS.
- Reasonable to treat for 1 week with phenytoin if at high risk for developing early posttraumatic sz (PTS).
- Continuation of such treatment for only 1 week reduces the incidence of early PTS while maintaining an acceptable low risk of adverse effects.
- There is no evidence that prevention of early sz reduces mortality, morbidity or development late posttraumatic epilepsy.
- Thus anticonvulsant prophylaxis is only a level III recommendation in terms of improving outcome.
- Treatment of patients after 1 week for late PTS prophylaxis is not recommended (Level I recommendation.)
NEUROMUSCULAR BLOCKADE

- Use when sedation fails
- Trial sedation (MS, Versed)

**Problems:**
1. Longer ICU stay
2. Increase Pneumonia
3. Increase Sepsis
4. Outcome unchanged

- Refractory ICP, Imaging
“Discovery consists in seeing what everybody else has seen and thinking what nobody has thought.”

S. Gyorgyi
D. PENNEY PEARLS

(20 YEARS TREATING HEAD TRAUMA) IN GEORGIA

6 T’s
THANK YOU