The Neurological and Medical Complications Of TBI

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Traumatic head injury is an insult to the brain, not of a degenerative or congenital nature but caused by an external physical force, that may produce a diminished or altered state of consciousness, which results in impairments of cognitive abilities or physical functioning. It can also result in the disturbance of behavioral or emotional functioning. These impairments may be either temporary or permanent and cause partial or total functional disability or psychosocial maladjustment.

National Head Injury Foundation

EPIDEMIOLOGY TBIMS:(1989-2006)

M/F: 3:1
Bimodal peaks at ages 16-25 (33%) and over 66 (10%)
White(66%) > African American (22%) > Hispanic (8%) > Asian (3%)
Vehicular > Falls > violence (44% alcohol related)
Cost: $9,347 acute rehab; $2,119 (daily)
EPIDEMIOLOGY (CDC)

- Falls (28%): greatest among the elderly (>65) and the young (0-4)
- MVA (20%): most hospitalizations; greatest between 15-19
- Stuck by/Against Events: This includes colliding with a moving or stationary object. Approximately 1.3-3.8 million sports related injuries, most of which are not seen in hospitals or ER's
- Interpersonal Violence (11%): firearm use is the leading case of TBI related deaths. Nearly 2/3 of firearm-related TBIs are suicidal in intent

Epidemiology (CDC)

- 5.3 million Americans with TBI related disabilities...about Denmark's population
- Yearly incidence: 1.4 M
  - 50,000 deaths with decreasing mortality (still a 30% 30 day mortality esp. in the elderly)
  - 235,000 hospitalizations; steady over the last 30 years
  - 1.1 Million ER visits with 1:4 admission vs. discharge ratio

COST

- $60 billion
  - $9.222 B (medical cost)
  - $51.212 B (productivity loses)

*the 10 year survival after 6 months parallels age matched normals
PATHOPHYSIOLOGY

BIOMECHANICS: TBI

• LINEAR FORCES
  – direct (coup and Contre Coup)
  – or indirect (acceleration/deceleration)
  *inferior frontal and superior temporal lobes almost always affected

CEREBRAL CONTUSIONS

BIOMECHANICS

• ROTATIONAL FORCES
  – shearing of neurons
  – Diffuse Axonal Injury affecting:
    • Midbrain
    • Pons
    • Corpus Callosum
    • white matter of the cerebral hemispheres.
PATHOPHYSIOLOGY

• DAI (Diffuse Axonal Injury)
  – “retraction bulbs” or swollen axonal endings that appear severed from their distal segments
  – consistent with wallerian degeneration
  – (+) LOC

CLASSIFICATION: PRIMARY VS SECONDARY INJURY

• Primary Injury refers to the direct disruption of the brain parenchyma from the shear forces of the impact. It is immediate (minutes to hours after the impact) and is not amenable to resuscitation.
  - Direct cortical disruption
  - Diffuse axonal injury
  - Impact depolarization
  * Clinically, the coupling of the injury to these structures lead to the picture of white matter petechial hemorrhages characteristic of DAI.

CLASSIFICATION: SECONDARY INJURY

Secondary injury refers to the cascade of biochemical, cellular, and molecular events which includes both indigenous damage in the brain as well as extra-cerebral damage that comes with trauma.
  - ischemia, excitotoxicity, and energy failure
  - Secondary Cerebral swelling
  - Diffuse Axonal Injury
  - inflammation and Regeneration
DIFFUSE AXONAL INJURY

- The classical definition of Diffuse Axonal Injury implies the immediate disruption of the axons due to rotational forces which cause shearing upon impact. However, there is also evidence of a secondary axotomy due to increased axolemal permeability and calcium influx and cytoskeletal abnormalities that propagate after the injury.

Secondary Injury after TBI

Glutamate release
Glutamate receptors
(NMDA, AMPA, KA)

\[ \text{Mg}^{2+}, \text{Zn}^{2+}, \text{glycine, PCP (receptor antagonists)} \]

\[ \text{[Ca}^{2+} \text{]} \]

\[ \text{[Na}^{+} | \text{K}^{+} \text{]} \]

Proteases
Lipases
Free radicals

\[ \text{Na}^{+}/\text{K}^{+} \text{ pumps} \]

\[ \text{ATP} \]

Neuronal death

FOCAL INJURY: Involves a localized injury in the brain occurring immediately after the injury and could be easily visualized by CT or MRI

- Cerebral contusions
- Focal Ischemia
- Focal hemorrhages

Focal Vs. Diffuse Injury
DIFFUSE INJURY: Widespread cerebral injury

- Diffuse Axonal Injury
- Excitotoxicity
- Hypoxia
- Apoptosis: Programmed cell death defined by cell shrinkage, nuclear condensation, intranucleosomal DNA fragmentation with dissolution of the cell membrane. It has both intracellular (Cytochrome C, AIF) and extracellular (TNF) triggers

NEUROCOGNITIVE/ BEHAVIORAL ISSUES

- AGITATION
  - State of aggression during PTA in the absence of any physical, medical, or psychiatric causes
  - Physical aggression, explosive anger, increased psychomotor activity, impulsivity, verbal aggression, disorganized thinking, and perceptual disturbances
  - Different from akathisia or the state of motor restlessness which is a component of agitation

- AGITATION
  - INCIDENCE: 35-96% in TBI Patients
    - Occurs more acutely up to 10 days post injury
    - Associated with LOC, subsequent injuries, major depression, frontal lobe lesions, poor premorbid social functioning, alcohol and substance abuse
NEUROCOGNITIVE/ BEHAVIORAL ISSUES

• AGITATION
  – PATHOPHYSIOLOGY: A combination of structural lesions, biochemical deficits, and external factors
  – STRUCTURAL LESIONS
    • Hypothalamus
    • Limbic system (amygdala)
    • Frontal Neocortex
  – NEUROTRANSMITTERS
    • Increased Norepinephrine
    • Decreased Serotonin
    • Increased dopamine

Management Paradigm

• 1. Identify the undesirable behavior: akathisia, impulsivity, confusion, verbal/physical aggressiveness
• 2. Consider the pathology: CNS Infection/sepsis, Acute Neurological event, pain, metabolic encephalopathy, medications.
• 3. Restore Sleep-wake Cycles: sleep hygiene/judicious use of medications

Management Paradigm

• 4. Environmental Modifications: Low noise and light levels; decrease restraints, decrease monitoring/blood draws
• 5. Pharmacologic Agents
  – Atypical antipsychotics
  – Antidepressants
  – Mood stabilizers/ Neuroleptics
  – Stimulants
  – GABAergics/ benzodiazepines
Medical Complications Following Stroke and Brain Injury

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Treatment Paradigm

Identification of undesirable behaviors

Identification of possible differential diagnosis:
- Drug withdrawal
- Infection
- Pain
- Hypoxia
- Seizure disorder

Consideration of environmental issues

Medication Management
- Minimizing benzodiazepines and typical antipsychotic agents as possible
- Anticonvulsants
  - Valproic acid (monitor liver function)
  - Tegretol (monitor sodium levels)
- Neurostimulants
  - Beta blocker
- And/or

Consideration of environmental issues

- Ensure good sleep cycle regulation
- Medication Management
- Minimizing benzodiazepines and typical antipsychotic agents as possible

ACUTE MEDICAL ISSUES:
Traumatic Brain Injury

• ICP
  - dependent on brain parenchymal tissue and CSF
  - increased with brain tissue edema or the presence of space occupying lesions
  - increased with CSF Flow obstruction

ACUTE MEDICAL ISSUES:
Traumatic Brain Injury

• BRAIN PRESSURE DYNAMICS
  - cranial vault
    - closed space
    - can only take a limited amount of volume
  - PVI (pressure volume index)
    - volume needed to raise the ICP 10x
      - PVI = 26 cc
      - 26 cc = 10x
      - 52 cc = 100x
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PHYSICAL EXAMINATION

- Serial neurological examinations (GCS scores)/ eye exams
  - Anisocoria: impending transtentorial herniation to the ipsilateral side
  - Bilateral fixed dilated pupils indicate a poor outcome
  - Gaze deviations point toward the site of the lesion
  - Rhythmic eye movements might indicate a seizure
  - Loss of corneal and oculocephalic reflexes are indicia of a severe TBI
- Abnormal respiratory patterns may indicate increasing ICP and herniation. Patients initially present with tachynea and hyperventilation followed by irregular breathing patterns (apnea) bradycardia, and the Cushingoid pattern of breathing

TRANSTENTORIAL HERNIATION

- Herniation of the cerebral peduncle through the transverse foramina
- Ipsilateral dilated pupil due to unopposed sympathetic action
- Contralateral hemiparesis
UNCAL HERNIATION

- Innermost temporal lobe (uncus) pressed into the brainstem
- Can result in brainstem infarcts/ ipsilateral weakness

INITIAL RESUSCITATION

- Airway Management: Endotracheal intubation is indicated in patients whose GCS < 9. Rapid Sequence Intubation (RSI) with sedating/ paralytic agents (Succinylcholine, thiopental, fentanyl) could be used in emergent situations. Careful attention should be given to the cervical spine (Avoiding neck manipulation/ hard cervical collars) in patients with severe TBI or those with a high index of suspicion for spine trauma.
- Oxygenation: PO2 should be kept between 100 and 120 mmHg while PCO2 should be kept between 28-32 mmHg.
- Circulation: Keep CPP > 60 mmHg; use of hypertonic solutions, osmotic diuretics and crystalloids to decrease intracranial pressure.

ICP MANAGEMENT

- 1. Ventricular Drainage
- 2. Hyperosmolar Agents
- 3. Sedation and Paralysis
- 4. Hyperventilation
- 5. Evacuation
Post Traumatic Hydrocephalus

- Affects 5% of severe TBI (GCS<8)
- 2 weeks - 2 years
- Non-communicating vs communicating
- Normal Pressure Hydrocephalus
  - Most common
  - In DI, wacky only
- Hydrocephalus ex-vacuo
  - 27-42%

ACUTE MANAGEMENT: Prevention of Secondary Injury

- Hypothermia* – Decreases ICP and brain metabolism
- Systemic Steroids - CRASH study aborted due to increased 2 wk. mortality
- Neuroprotective agents: NMDA antagonists, Ca channel blockers, etc.

CENTRAL DYSAUTONOMIA

- Paroxysmal Autonomic Instability and Dystonia (PAID)
- Hypertension, tachycardia, hyperthermia, spasticity and perspiration due to a surge of circulating catecholamines released from direct trauma to the autoregulatory centers.
- 2 weeks post-injury
- MANAGEMENT:
  - Lipophilic Beta Blockers for hypertension/tachycardia
  - Dantrolene Sodium for malignant hyperthermia
  - NSAIDS (Indomethacin), Acetaminophen
  - Cooling Blankets, NG tube lavage
  - Dopamine Agonists:
    - Anterior hypothalamus-temperature sensitive
    - Posterior hypothalamus-heat dissipation center
TEMPERATURE REGULATION

- Anterior preoptic hypothalamus has heat sensitive neurons that promotes sweating and ADH secretion
- Posterior hypothalamus has cold receptors that trigger shivering, vasoconstriction, and increase tone

PAID: THEORIES

- Direct injury to the rostral hypothalamic region causing PG release to anterior hypothalamus resetting thermostats
- Direct frontal damage interrupt inhibitory pathways to the hypothalamus
- Axonal stretch causing inhibition of release of neurotransmitters (dopamine)
- Dopamine causing hypothermia in animals and its abundant receptors in the hypothalamus

MEDS: MODE OF ACTION

<table>
<thead>
<tr>
<th>MEDICATION</th>
<th>ACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bromocriptine/Amantidine</td>
<td>Dopaminergic D2 agonist</td>
</tr>
<tr>
<td>Clonidine</td>
<td>β-adrenergic agonist/ decreased sympathetic outflow from the CNS/ decreased BP</td>
</tr>
<tr>
<td>Ativan</td>
<td>Interacts with GABA receptor site modulating its activity</td>
</tr>
<tr>
<td>Propanolol</td>
<td>β-adrenergic blockade/ decreased BP/ crosses BB barrier</td>
</tr>
<tr>
<td>Dantrolene Sodiuim</td>
<td>Dissociates excitation contraction by interfering with Ca release from the sarcoplasmic reticulum</td>
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Medical Complications Following Stroke and Brain Injury

**Figure 1. Diagnostic fever protocol in TBI patients**

- Temperature > 38.2°C
  - Clinical exam for source of infection
    - CBC with differential
    - Blood cultures
    - Chest x-ray
    - Abdominal films
  - CT scan of head (Positive) or infectious disease consultation (Negative)
    - Culture invasion lines, sputum, throat, urine
    - Duplex scan, venogram of plethysmography for DVT
    - Consider drug fever
    - Extended chemistry profile and sedimentation rate
    - Lumbar puncture (if indicated by clinical exam)
    - Arterial blood gas and V/Q scan

**NEUROLOGICAL COMPLICATIONS**

**SEIZURES**

- Post-traumatic Seizure: An initial or recurrent seizure episode not attributable to another obvious cause after penetrating or non-penetrating TBI
  - Immediate/ impact seizure: 1st 24 hrs
  - Early post-traumatic seizure: 1st week
  - Late post-traumatic seizure: after 1st week
- Post-traumatic epilepsy: A disorder characterized by recurrent late seizure episodes not attributable to another obvious cause in patients following TBI.
SEIZURES: PATHOPHYSIOLOGY

- **Ferric Chloride model**: Iron Salts and hemoglobin in neural tissue may contribute to epileptogenesis by initiating lipid peroxidation, damaging cell membranes, and inhibiting NA-K Atpase.
- **Kindling**: repeated electrical stimulation in damaged neurons causes hypersensitivity and decreased seizure threshold.
- **Synaptic Plasticity**: Compensatory collateral axonal sprouting can lead to neural hyperexcitability and the development of new seizure foci.

SEIZURES: EPIDEMIOLOGY

- 5% in closed head injuries
- 30-50% in open head injuries
- 1% of those without early seizures will have late seizures
- 25% of those with early seizures will have late seizures
- Of those with late seizures:
  - 50% will be seizure free within 5-10 years
  - 25% will have good control with AED’s
  - 25% will be uncontrolled

SEIZURES: TREATMENT

**PROPHYLAXIS**: Temkin, AAN

Seizure prophylaxis with AED’s (Dilantin/Valproic Acid) x 7 days can Help prevent early onset posttraumatic seizures. Longer administration does not prevent the onset of late onset seizures….may even increase it
SEIZURE: RISK FACTORS

- Annegers (1980, 1988)
  Age (>65) and Severity of TBI > skull fracture, LOC
- Englander et al (2003) CT findings and seizures
  Biparietal contusions (66%),
  Penetration with bone and metal fragments (62.5%),
  Multiple intracranial procedures (36.5%)
  Multiple subcortical contusions (33.4%)
  Subdural hematoma w/ evacuation (27.8%)
  Midline shift > 5mm (25.8%)
  Multiple or bilateral contusions (25%)

SEIZURES: DIAGNOSIS

- EEG: Utility is in focus localization, determination of seizure persistence, and severity prognostication once documented.
- Prolactin levels: Elevations noted immediately post-ictally
- SPET Studies: 30-40% chance interictally and 70-80% chance ictally

AED’S

- Phenytoin (200-900mg/d): P.O. rapid loading (and newer IV fosphenytoin), ataxia-vestibular, sedation/cognitive/ hirsuitism, gingival hyperplasia, RASH/LFT’s
- Valproate (500-300mg/d): less sedating; mood stabilizer; approved for Migraine, associated with higher mortality in the acute post-traumatic period (Temkin)
AED’S

• Carbamazapine (600-1200mg/d): comes in long-acting bid dose, mood/behavior stabilizer, neuropathic pain; sedation, ataxia, diplopia, aplastic anemia, liver induction, SIADH

• Oxcarbazepine (900-1800mg/d): serum levels not required, not enzyme inducing, mood stabilizing, more expensive, hyponatremia possible

AED’S

• Neurontin (900-5000mg/d): mood stabilizing properties; best choice in liver damage; neuropathic pain; weak anti-spasticity agent; may help tremor; weak seizure control

• Lamotrigine (100-500mg/d): very slow titration: among least sedating and may even be activating; strong mood/behavior stabilizing properties; and neuropathic pain efficacy; rash

Topiramate (Topamax)

■ Mechanism: GABA potentiation, voltage-sensitive Na+ channel and AMPA receptor blockade

■ Dose: 100-200 mg bid

■ Disadvantage: adverse cognitive effects vs. gabapentin and lamotrigine—Martin et al, 1999

■ Advantage: side effect of weight loss usually a plus; approved for Migraine
STATUS EPILEPTICUS

• GCSE (Generalized Convulsive Status Epilepticus): >5 minutes of continuous seizures or recurrent seizures w/o return to baseline consciousness >30 minutes
• Overall mortality is 20% but related more to the underlying pathology

Systemic complications

• Hyperthermia
• Acidosis
• Hypotension
• Respiratory failure
• Rhabdomyolysis
• Aspiration

Treatment

• 1. supportive: ABC’s, positioning and environmental modification
• 2. Work up: stat serum glucose, electrolytes (Na and Ca), ABG, Prolactin level, Imaging studies
TREATMENT

• Benzodiazepines (Lorazepam)
  – Sedative hypnotic with short onset of effects and relatively long half-life. By increasing action of GABA, a major inhibitory neurotransmitter in the brain, may depress all levels of CNS, including limbic and reticular formation. Important to monitor patient’s BP after administering dose. Adjust as necessary.
  – 4 mg IV slowly at 2 mg/min; if seizure continues or recurs after 10-15 min, administer an additional 4 mg IV slowly at 2 mg/min

TREATMENT

• AED’S (Dilantin)
  – Loading dose: 18-20 mg/kg IV; hypotension may necessitate slowing administration rate; rate not to exceed 50 mg/min (hypotension and arrhythmias can otherwise occur); if status epilepticus persists, may increase to total of 30 mg/kg

ENDOCRINE: TBI (Hypothalamic Pituitary dysfunction)

- 2/3 of Severe TBI mortalities w/ structural abnormalities in the hypothalamic pituitary region (Crompton, 1971)
- 1/10 TBI’s develop HPA dysfunction (Dimopoulou, 2005)

Pathophysiology:
- Direct or indirect trauma
- Drugs
- Circulating cytokines
- Secondary insults
Normal Anterior Pituitary Hormonal Function

- TSH
- ACTH
- FSH
- LH
- PRL
- GH
- Thyroxine
- Cortisol
- Testosterone
- Estrogen
- IGF-I

Memory
Mood
Metabolism
Energy
Neuromuscular

Memory
Mood
Metabolism
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Post-TBI Neuroendocrine Dysfunction: Results

- ↓ GH
- ↓ IGF-I
- Abnormal Thyroid Function
- ↓ AM Cortisol Levels
- Single Abnormality
- Dual Abnormalities

Prevalence of Hypopituitarism in TBI

- ↓ GH
- ↓ LH and/or FSH
- ↓ ACTH
- ↓ TSH
- ↓ TSH ×1 deficiency


**ENDOCRINE DISORDERS (TBI): HYPONATREMIA**

**SIADH:** Euvolemic hyponatremia
- (0.8-30%) in Neurosurgery and TBI
- Acute Arginine Vasopressin release from the posterior pituitary up to 2 days post injury which persists in the circulation up to 14 days
- Serum Na < 135 Meq/L, Serum osmolarity < 280 mOsM/kg, urine osmolarity > serum osmolarity, no signs of volume depletion, and Normal thyroid, adrenal, and renal function
- Treatment: Fluid Restriction, Demelocycline, Na tablets

**CSW (Cerebral Salt Wasting):** Hypovolemic hyponatremia
- (30% in SAH)/ Vasospasm
- Renal Na loss = hypovolemia and hyponatremia
- Release of natriuretic peptides (ANP, BNP, adrenomedullin)
- TBI can interrupt sympathetic output to the kidney
- Labs: same as SIADH except for hypovolemia (BUN)
- R/o adrenal insufficiency
- Management: Volume replacement, positive Na balance, and mineralocorticoids
**ENDOCRINE DISORDERS (TBI): HYPONATREMIA**

Psychogenic Polydipsia (Polydipsia and Hyponatremia)
- Psychotic disorders and TBI
- Behavioral, dopaminergic and cholinergic systems as well as Hippocampal pathology
- Behavioral modification and fluid restriction
- Clozapine

**Hyponatremia Correction**
- Decreased osmolarity = increased brain edema
- Overcorrection = Central pontine myelinosis

**TBI: HYPERNATREMIA**

Central Diabetes Insipidus: Hypovolemic Hypernatremia
- 1% of TBI (LOC, Skull Fx, and Generalized neurologic findings)
- Lack of ADH secretion due to hypothalamic or pituitary injury
- Acute (60%), Chronic (30-40%), or Triphasic (5-10%)
- Diagnosis: Polydipsia > 3.5L/d, Polyuria 20L/d
- Labs: serum Osm > 310-320 mOsm/kg, Na > 145 meq/L, Plasma ADH < 1.1 ng/L
- Water deprivation test/ Vasopressin challenge test
- Management: Vasopressin, DDAVP, Fluids, Chlorpropamide, HCTZ, Tegretol

**THE DILEMA**

- Providing the ICH patient with adequate DVT and PE protection while minimizing the risk of Hemorrhage expansion
WHAT DO WE KNOW: The Numbers

• 50 – 99% of trauma patients will develop DVT’s
• 5% of NSGY patients will develop PE with a mortality between 9%-50%
• TBI seems to be an independent risk factor(4x) for DVT’s(50% in acute, 13-20% in Acute Rehab)
• ICH’s have a 53% risk of DVT and 16% risk of PE’s; hemorrhagic strokes > infarcts
• Elective Craniotomies: 18-50% of DVT, 0-25% of PE’s
• Brain tumors (28-43%), Craniotomies (25%), Head Injury (20%)

TBI

• TBI is an independent risk factor for DVT’s
  – Immobility
  – Increased Clotting Factors (TF, VWF)
• Four fold Increase in DVT Risk between the first 24-72 hours (3.6%-15%), 42% at 2 weeks
• 50% in the Acute phase and 13-20% in Acute Rehab
• Relative safety in terms of systemic and ICH rebleeds in those chemically prophylaxed
• Relative reduction in VTE’s (54-80%) w/ LMWH/UFH

What We Know: Diagnostics

• Physical Examination: calf circumference/ Homan’s sign (poor sensitivity/ specificity)
• D- dimer Assay: 500ug/dl at 2 weeks not useful in TBI pt’s with elevated values up to 8 weeks post
• Real Time Ultrasonography: most specific/sensitive in symptomatic dvt’s; specific but not as sensitive in asymptomatic dvt’s (distal extremities: 59%)
• Venography: Invasive/ potentially nephrotoxic
WHAT WE KNOW: Management

SCD
• Inc. Hemodynamic and Fibrinolytic Activity which ceases immediately when d'ced
• No ICH
• Neuropathies/ Allergies
• Poor Compliance
• 32% DVT formation in NSGY Literature

WHAT WE KNOW: Management

UFH
• Mixture of GAG's w/ MW's between 4-30 kd
• Activates antithrombin III w/ inactivates XA/IIA as well as PLT aggregation
• Short half life: Bid to TID dosing
• Effective and Reversible
• Cheap: 2$/day: prophylaxis, 5$/day: treatment

SCD'S in a Neurorehab Setting: Compliance Difficulties
• SCD's during therapy and mobilization
• The toll on nursing and support staff
• Agitated and demented patients
• Interference with other Rehabilitation equipment: Restraints, orthoses, positioners, External fixators.
WHAT WE KNOW: Management

LMWH
- Fractionation of the Heparin Molecule
- Greater XA/IIA ratio
- Greater bioavailability at lower doses
- More predictable anticoagulant response
- Longer Half life (qd/bid dosing)
- Less HIT
- Not Cheap: 405 for prophylaxis/1095 for treatment
- Cochrane database points to having less complications than UFH

MANAGEMENT

IVC Filters
- Invasive
- Removable
- Does not prevent PE’s from UE DVT’s
- Does not prevent most thrombotic disease
- Can still thrombose around the filter or go through the filter

WHAT’S OUT THERE: Guidelines
- 2001 ACCP (Neurosurgery): Mechanical Methods are Standard of Care, UFH left to the practitioner’s discretion, LMWH: Unsuitable
- 2007 AHA/ASA (Stroke Council, High Blood Pressure Research Council and Quality of Care and Outcomes Research Interdisciplinary Working Group): Mechanical Compression and UFH or LMWH 3-4 days after ICH
- ABPMR/ TBI SAE: TBI: Chemical Prophylaxis 2 weeks post injury
SUMMARY...For now

- All our ICH subgroups are at risk for VTE's
- The Risk starts immediately after the event (24-48 hrs.) hence, a number of them will be coming in with VTE's...or not coming at all if not addressed early
- Dopplers are an acceptable diagnostic tool despite the cost especially in TBI Patients
- SCD’s only work if worn continuously
- Chemical DVT prophylaxis is effective with an acceptable risk
- Timing (24-48hrs-2 weeks) of Chemoprophylaxis is up in the air
- No Conclusive data on UFH vs LMWH in ICH’s other than cost