Traumatic Brain Injury: ICU and Beyond

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Outline

• Management of Acute with Focus on Rehabilitation Concerns
  – BTF Guidelines for Acute Management
  – Consequences of Immobilization
  – TBI Specific Consequences

• Determining Next Steps: After the ICU
Learning Objectives

• Relate body system responses to immobility to nursing and rehabilitation management solutions

• Recognize the unique contribution of TBI to worsening the consequences of immobility

• Review prognostic factors that determine outcomes after TBI
TBI in the United States

At least 1.7 million TBIs occur in the United States each year.*

- 50,000 Deaths
- 235,000 Hospitalizations
- 1,111,000 Emergency Department Visits
- 57 million living with TBI worldwide
- ??? Receiving Other Medical Care or No Care

Faul, CDC, 2010  *  Average annual numbers, 1995-2007
• **Comparison of Annual Incidence**
  
  – Traumatic Brain Injuries >1,000,000
    • 80% mild = 200,000 moderate/severe
  – Breast Cancer 176,300
  – HIV/AIDS 43,681
  – Spinal Cord Injuries 11,000
  – Multiple Sclerosis 10,400
Pre-Hospital Assessment of TBI

• Oxygenation and Blood Pressure
  – Hypoxemia (<90% arterial hemoglobin oxygen saturation)
  – Hypotension (<90 millimeters mm Hg systolic blood pressure)

• Glasgow Coma Score
  – Measured after the initial assessment, after a clear airway is established, and after resuscitation and prior to the administration of sedatives or paralytics

• Pupillary assessment - >1mm asymmetry
Pre-hospital Treatment

• Airway, ventilation, oxygenation (SaO2 >90)
  – Supplemental O2, intubation, avoid hyperventilation (assoc with worse outcome)

• Fluid resuscitation (SBP >90mmHg)
  – Isotonic crystalloids, hypertonic saline for GCS < 8

• Brain-targeted therapy
  – Options: hyperventilation (briefly for herniation)
TBI in the ICU
Guidelines for Acute Management of TBI

• Brain Trauma Foundation


• Brain Trauma Foundation, 523 East 72nd Street, New York, NY 10021, USA
Standards of Care in ICU

- Monitoring of intracranial pressure (2)
  - Appropriate for patients with abnormal CT scans and GCS 3-8 after resuscitation or those with normal CT scans who are >40 years age, posturing, or have SBP <90

- When to treat elevated ICP?
  - ICP thresholds above 20 mm Hg (2)
  - Combination of ICP, CT scan, clinical evaluation

- CPP Target – 50-70

Critical Care Nurses, 2011
• Brain O2 monitoring – still evolving
• Avoidance of hypotension (2) (systolic < 90) /hypoxemia (PaO2<60, O2 sat < 90)
• Use of hyperosmolar agents (2/3)
  – Mannitol for control of ICP (doses 0.25 gm/kg to 1 gm/kg body weight) as a single dose for temporary control
  – (Hypertonic saline)
Standards of Care in ICU

• Use of barbiturates (2)
  – Refractory elevated ICP
    • Propofol (no improvement mortality, outcome)

• Nutrition (2)
  – Start feeding within 72 hours (no good data on formulation, jejunal vs. gastric)
  – Feed to full caloric replacement by Day 7 after injury
  – Mean of 140% expected metabolitic expenditure in rested comatose patients
• Seizure prophylaxis (1)
  – may be used to prevent *early* post-traumatic seizures in patients at high risk for seizures following head injury
  • Control ICP, BP changes, excitatory neurotransmitter release
• Early PTS not associated with worse outcomes
Standards of Care in ICU

• DVT Prophylaxis (3)
  – Compression stockings or intermittent pneumatic compression until ambulatory (?), unfractionated heparin or LMWH (increased risk for expansion of intracranial hemorrhage)
    • Insufficient evidence for preferred agent, dose, or timing

• Hypothermia (3)
  – Better outcome scores, no difference in mortality
Acute Rehabilitation/Medical Concerns: Mobilization and Positioning are Key

• Avoiding consequences of immobility
• Control of muscle tone and preservation of limb mobility
• Evaluation of cognition
• Management of behavioral disorders
• Prognosis for outcome
Avoid unwarranted pessimism!

See later slides on prognosis.
Effect of Immobility on Body Systems

• Musculoskeletal
• Respiratory
• Cardiovascular
• Hematological
• Neurological
• Psychiatric

• Metabolic
• Neuroendocrine
• Gastrointestinal
• Urological
• Dermatological
Musculoskeletal

• Bone density loss
  – moderate ↓ in bone resorption, even larger ↓ in mineralization of bone matrix, poor absorption of Ca, GI Ca losses
  – Additional factors in TBI: paralysis
Muscle weakness and atrophy

- Protein breakdown and decreased synthesis
- Antioxidant disruption
- Additional factors in TBI: hyper-catabolic state
Joint Contractures

- Reduction in length and flexibility, increased connective tissue, disorganized collagen fibers
  - Knee/hip flexion (walking), knee extension (sitting)
Additional Issues in TBI

1. Posturing due to spasticity and dystonia
2. Heterotopic ossification

Prevention of Contractures

1. Appropriate bed position
2. Splinting or casting
3. ROM
Respiratory System

• Diminished functional residual lung capacity, diffusion capacity, pulmonary blood flow
  – Additional risks in TBI: tracheostomy, impaired alertness and respiratory drive, dysphagia

• Management: Good bed positioning, early mobilization, aggressive pulmonary toilet with mobilization of secretion (in-exsufflation)
Cardiovascular System

• Reduced functional capacity and orthostatic hypotension
  – Loss of plasma volume resulting in decreased stroke volume
  – Attenuated baroreflex responses
  – Loss of aerobic capacity and conditioning

• Bed positioning and early mobilization
Hematologic/Thrombogenesis

- Potential effects of decreased red cell mass
- Risk factors: immobility, hemiparesis, hypercoagulability after trauma
- Risks of prophylaxis or DVT treatment: intracranial bleeding, weakness or paralysis combined with impulsivity or neglect and increased risk of falls
Neurological/Psychiatric System

• Depressed scores on cognitive testing, mood disorders and distress, insomnia, balance impairment

• Associated with TBI: Significant sleep disorders, confusion and cognitive impairment, disorientation, balance impairment
Endocrine/Metabolism

• Less known about interaction of immobilization and endocrine issues
  – TBI: Growth hormone, cortisol, thyroid hormones

• Metabolism
  – Hypercalcemia (esp. in young men)
  – Hyperhidrosis
GI/Urologic system

• Constipation/Diminished absorption of nutrients
  – TBI: catabolic state and increased need for nutrition, hyperhidrosis leading to volume loss
  – Altered CNS input into enteric nervous system
  – Medications slowing GI motility common
  – Fiber supplements and rectal suppository motility agents

• Reflux, Stress ulcers
Urinary system

• Urinary incontinence: cognitive impairment, damage to micturition centers, peripheral nerve damage, or even bladder trauma

• Stasis and infection:
  – Indwelling catheters
Dermatological System

• Pressure ulcers
  – Inadequate tissue perfusion
  – Malnutrition
  – Impaired consciousness and paralysis

• Turning and positioning

• Use of specialty mattresses

• Nutrition and hydration
Take Home Messages for TBI and Acute Care

- Consequences of immobility can adversely affect outcome in patients with TBI
- Musculoskeletal changes can be profound with weakness, atrophy, and contracture
- Impaired cough and swallow mechanisms, low lung volumes, and ventilator use predispose these patients to pneumonia and prolonged ventilator use
Take Home Messages for TBI and Acute Care

• DVT prophylaxis and monitoring for effects in intracranial bleeding

• Enhanced potential for pressure ulcers dictates good positioning/turning and nutrition

• Cardiovascular sequelae will lengthen recovery
Planning for the Next Step: After the ICU
Determining potential for recovery

- GCS score – no clear cut-off point
- Length of coma – < 2 weeks suggests “good” recovery; > 3 months poor
- Post-traumatic amnesia - <2 months “good” recovery; > 3 months poor
- Age – 65 and older
- Neuroimaging – bilateral brainstem lesions
Assessment of TBI and severity

• Clinical assessment
  – Glasgow Coma Scale
  – Length of loss of consciousness

• Neuroimaging
  – Computerized tomography
  – Magnetic resonance imaging
  – (Single photon emission computerized tomography)
  – (Positron emission tomography – not used clinically)

• Neuropsychological examination

• Electrophysiologic techniques (not commonly used clinically)
Assessment of Severity of TBI

Glasgow Coma Scale (1974) used to

– assess depth and duration of impaired consciousness and coma

– gauge deterioration or improvement at the emergent and acute stages of brain damage or lesions

– predict the ultimate outcome
Glasgow Coma Scale

- **Best Eye Response. (4)**
  - No eye opening.
  - Eye opening to pain.
  - Eye opening to verbal command.
  - Eyes open spontaneously.

- **Best Verbal Response. (5)**
  - No verbal response
  - Incomprehensible sounds.
  - Inappropriate words.
  - Confused
  - Orientated

- **Best Motor Response. (6)**
  - No motor response.
  - Extension to pain.
  - Flexion to pain.
  - Withdrawal from pain.
  - Localising pain.
  - Obey Commands.

- **E + V + M = Total**
  
  **Severe** 3-8
  **Moderate** 9-12
  **Mild** 13-15
Limitations of the GCS

• Not a predictor of functional outcome in the middle ranges
• Upper ranges not predictive of persistence of symptoms
• May be affected by eye swelling, endotracheal tubes, alcohol, young children, non-English speakers, persons with chronic neurological conditions
Imaging

• CT scan – evaluate for intracranial masses/mass effect, fragments
• MRI – adjunctive role, sequelae
• SPECT – controversial (confounded by medical and neurological disorders, learning disabilities, substance use, emotional disorders) – not routinely clinically indicated
Neuropsychological examination

- Standardized normed collection of measures examining cognitive function
  - Most convincing if baseline data exists
    - Athletics (ImPACT – Immediate Post Concussion Assessment and Cognitive Testing), Military (ANAM – Automated Neuropsychological Assessment Metrics)
  - Need to have skilled neuropsychologist who does not overread or underread NPEs (but still currently best method to document impairment)
Prognosis

• Nonspecific: CT scan, initial GCS
• More specific:
  – Length of unconsciousness/coma:
    • > 4 months – persistent vegetative state*
  – Post traumatic amnesia (PTA) – ability to remember events happening after the injury
  – Age: >65
Recovery after TBI

• Moderate-Severe TBI
  – “Rule of thumb”
    • Rapid recovery over first 12 months
    • Return to community activities over 12-24 months
  – Recovery can continue for extended period of time

• Recovery from coma or minimally responsive state cannot be determined before 4 months!
Critical Care Nurses and TBI

- Neurophysiological Interventions
- Psychosocial Interventions
- Preventative Interventions
- Maintaining the therapeutic milieu

McNett, Gianakis. J Neurosci Nurs, 42 (2), 2010
Thank You!

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