

Putting Clinical Policies into Practice: Management of Neurological Emergencies. Traumatic Brain Injuries

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Disclosures

- Executive Committee, Foundation for Education and Research in Neurologic Emergencies
- Speakers board: GSK, BMS, Sanofi-Aventis
- Advisory Board: HeartScape Technologies, Brainscope Incorporation

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Overview

Global Objectives

- Understand disease state (TBI)
- Review the Brain Trauma Foundation 2007 guidelines for severe TBI
- Review the ACEP / CDC minor TBI practice parameters
- Detail trephination and antibiotic use

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Epidemiology

TBI Incidence

- 1.6 million head injuries per year
- 800,000 receive ED, outpatient care
- 270,000 hospital admissions
- 52,000 deaths
- 90,000 permanent neuro disabilities

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Health Care Costs

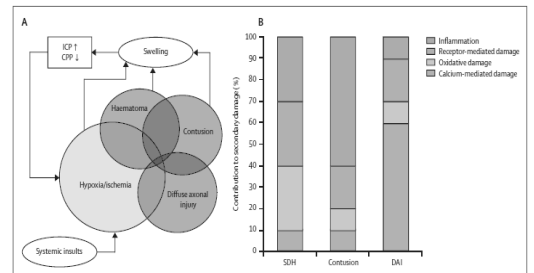
TBI Effects

- Leading cause of death & disability
- Loss of life
- Loss of productivity
- Significant health care costs
- Annual cost: \$40 billion
- 52% of all trauma deaths due to TBI

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Pathophysiology



Maas AI, et al. Lancet Neurol 2008; 7: 728-41 Brian O'Neil, MD, FACEP



Pathophysiology

Brain Edema and ICP

- Brain edema:
 - Vasogenic, hydrostatic, osmotic effects
 - Cytotoxic effects
 - Interstitial edema
- Normal intracranial pressure
 - $CPP = MAP - ICP$
 - $80 = 90 - 10$ (mm Hg)

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Pathophysiology

Elevated ICP

- ICP < 15 mm Hg is normal
- Altered mental status patients:
 - 40% will have increased ICP
- CBF is disturbed above 40 mm Hg
- ICP > 60 mm Hg is lethal

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Severe TBI Case

History

- 58 year old male
- Struck by auto crossing street
- Coming to your trauma center ED
- Prehospital care: IV, O2, monitor
- Pt is immobilized
- Pt responds only to painful stimuli

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Severe TBI Case

Physical Exam

- 98.8 100/60 110 12 approx 70 kg
- Gen: ? Non-purposeful mvmt on cart
- Head: Large laceration, contusion over R temporal-parietal region
- Face: Several abrasions, contusions
- Eyes: 4 mm, equal, reactive, EOM OK

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Severe TBI Case

Physical Exam

- Chest: BSBE, no crep pox 95%
- Cor: Tachycardia without murmur
- Abd: Soft, ? non-tender, no peritonitis
- Pelvis: Stable to compression
- Ext: No fracture evident, abrasions

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Severe TBI Case

Neurologic Exam

- Motor: Withdraws to painful stimuli
- Sensory: No apparent anesthesia level
- Eyes: Open to painful stimuli
- Verbal: Moans to painful stimuli
- Reflex: No posturing, pathological reflex

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Severe TBI Case Provisional Diagnosis

- Severe TBI (GCS Score approx 8-9)
- R/O skull fracture
- R/O cerebral contusion
- R/O epidural hematoma

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Description of the Process

Strength of evidence (Class of evidence)

- I: Randomized, double blind interventional studies for therapeutic effectiveness; prospective cohort for diagnostic testing or prognosis
- II: Retrospective cohorts, case control studies, cross-sectional studies
- III: Observational reports; consensus reports

Strength of evidence downgraded based on methodologic flaws

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Recommendations

Year of publication	Description	Type	Topics (n)	Recommendations (n)		
				Class I	Class II	Class III
Maas and co-workers*	1997	European Brain Injury Consortium guidelines on management of severe head injury in adults	Consensus/report opinion	-	-	-
Bullock and co-workers**	1996	Management of severe TBI (best edition)	Evidence based	13	1	10
Bullock and co-workers**	2000	Management and prognosis of severe TBI	Evidence based	13	3	10
Brain Trauma Foundation*	2000	Prophylactic management	Evidence based	7	0	5
Asadi and co-workers**	2001	Penetrating brain injury	Evidence based	7	0	0
Va and co-workers†	2002	European Federation of Neurological Societies guidelines on mild traumatic brain injury	Evidence based/consensus	-	-	-
Adelson and co-workers**	2002	Pediatric guidelines	Evidence based	17	0	6
Brain Trauma Foundation*	2002	Field management of combat related head trauma	Evidence based	5	0	3
Bullock and co-workers**	2006	Surgical management of TBI	Evidence based	5	0	0
Brain Trauma Foundation**	2007	Revised guidelines for management of severe TBI	Evidence based	15	1	14
Italian Society of Neurology**	1996	Italian guidelines for management of patients with minor head injuries	Consensus/report opinion	-	-	-
Bartlett and co-workers**	1999	UK guidelines for the initial management of head injuries	Expert opinion	-	-	-
Newcombe and Murray**	1999	Management of acute neurotrauma in rural and remote locations of Australia	-	-	-	-
UK National Institute for Health and Clinical Excellence††	2003	UK guidelines for triage, assessment, investigation, and management of TBI	Evidence based	27	3	16

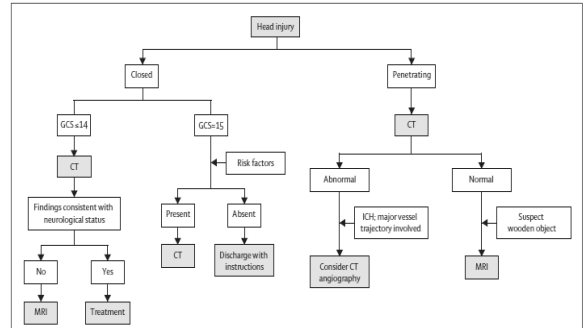
*<http://www.braintrauma.org/>; **<http://www.who.int/>; †to the NICE guidelines (<http://www.nice.org/>), the grading scheme for level of recommendations was adapted from the Oxford Centre for Evidence Based Medicine levels of evidence as level A-D, for consistency, we considered grade A as class I, grade B as class II, and grade C and D as class III.
 ††<http://www.nice.org.uk/>

Maas AI, et al. *Lancet Neurol* 2008; 7: 728-41

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Use of CT



Maas AI, et al. *Lancet Neurol* 2008; 7: 728-41

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Risk factors with GCS of 15

- Vomiting
- Age
- Duration of Amnesia
- MOI
- Neuro deficits
- Anticoagulation

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Severe TBI Case Therapy Questions

- What are the indications for
 - Fluids, hypertonic saline, blood?
 - Hyperventilation?
 - Mannitol?
 - Barbiturates?
 - Hypothermia?
 - Steroids?
 - Seizure prophylaxis?

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Treatment

Blood Pressure / Oxygenation

- **Level II**
- BP monitored and hypotension (SBP <90 mm Hg) avoided
 - **No target BP**
- **Level III**
- Oxygenation monitored and hypoxia (PaO₂ <60 mm Hg or O₂ saturation <90%) avoided
- **Guidelines** Customary to treat hypotension with fluids. Hypertonic saline in the pre-hospital phase is safe in doses <500 mL and can be used for hypovolemia.

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Treatment

Hyperventilation

Level II

- Prophylactic hyperventilation (PaCO₂ of 25 mm Hg or less) is not recommended

Level III

- Hyperventilation is recommended as a temporizing measure for the reduction of elevated intracranial pressure (ICP)
- Hyperventilation should be avoided during the first 24 hrs when CBF is often critically reduced.
- If hyperventilation is used, jugular venous oxygen saturation or brain tissue oxygen tension measurements are recommended

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Treatment

Hyperventilation Options

- Option: Hyperventilation useful briefly
 - Acute neurologic deterioration
 - Longer use if intracranial HTN persists despite other medical therapies (sedation, paralysis, mannitol, CSF drainage)
- Rapidly lowers ICP via vasoconstriction, which reduces cerebral blood flow
- One RCT

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Hypothermia for TBI

Level III*

- Pooled data indicate that prophylactic hypothermia is not significantly associated with decreased mortality
- Preliminary findings suggest that a greater decrease in mortality risk when target temperatures are maintained for more than 48 hours.
- Prophylactic hypothermia is associated with significantly higher Glasgow Outcome Scale (GOS) scores when compared to scores for normothermic controls.

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Treatment

Mannitol

Level II

- Mannitol is effective for control of raised intracranial pressure (ICP) at doses of 0.25 g/kg to 1 g/kg body weight. SBP <90 mm Hg should be avoided.

Level III

- Restrict mannitol use prior to ICP monitoring to patients with signs of transtentorial herniation or progressive neurological deterioration not attributable to extracranial causes.

Summary

- Mannitol is effective in reducing ICP in the management of traumatic intracranial hypertension. Current evidence is not strong enough to make recommendations on the use of hypertonic saline

Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons.
Guidelines for the management of severe traumatic brain injury. Hypertonic therapy. 1
Neurosurgery 2007;24(Suppl):S14-S20.

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Treatment

Mannitol - CR

- Initially osmotic load that increase BP
 - Lowers ICP
- May reverse brain swelling
 - Osmotic agent
- Few eligible RCTs
- Considerable uncertainty
- May be superior:
 - to pentobarbital for increased ICP
 - in setting of measured increased ICP

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Treatment

High Dose Barbiturates

- Standards: None
- Guides: Controls increased ICP
 - May be useful when maximal therapies fail
- Lower ICP via lower cerebral metabolism
- Few eligible RCTs
- No evidence of improved outcome
- Noted hypotension in 1 of 4 patients

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Treatment

Cerebral Perfusion Pressure

Level II

- Aggressive attempts to maintain CPP above 70 mm Hg should be avoided because of ARDS

Level III

- CPP of <50 mm Hg should be avoided.
- CPP value to target lies within the range of 50–70 mm Hg
- Ancillary monitoring of cerebral parameters that include blood flow, oxygenation, or metabolism facilitates CPP management.

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Treatment

ICP Rx Algorithm

- Insert ICP monitor, maintain CPP 50- 70 mmHg
- Ventricular drainage
- Repeat CT
- Hyperventilate to pCO₂ 30-35 mm hg
- Mannitol 0.25 to 1.0 gr/kg
- Second tier Rx: barbiturates, pCO₂ < 30

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Treatment

ICP Monitoring

Level II

- ICP should be monitored in all salvageable patients with a severe traumatic brain injury (GCS score of 3 to 8 after resuscitation) and an abnormal CT scan

Level III

- ICP monitoring is indicated in patients with severe TBI with a normal CT scan if: two or more of
 - age over 40 years
 - unilateral or bilateral motor posturing
 - SBP <90 mm Hg

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ICP Treatment

- Level II
- Treatment should be initiated with intracranial pressure (ICP) thresholds above 20 mm Hg.
- Level III
- A combination of ICP values and clinical and brain CT findings should be used to determine the need for treatment.

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Treatment

Seizure Prophylaxis

- Adult patients with severe TBI (typically with prolonged LOC or amnesia, ICH on CT scan, and/or depressed skull fracture)
- Prophylactic treatment with phenytoin, beginning with IV loading, should be initiated ASAP after injury to decrease the risk of post-traumatic seizures within the first 7 days (Level A).
- Prophylactic treatment with phenytoin, carbamazepine, or valproate should not routinely be used beyond the first 7 days after injury to decrease the risk of post-traumatic seizures occurring beyond that time (Level B).

Chang BS, Lewentz DH. Practice parameter: antiepileptic drug prophylaxis in severe traumatic brain injury: report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2003 Jan 14;60(1):10-6.

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Treatment

Seizure Prophylaxis, Rx -CR

- Reduced secondary damage due to increased metabolism, ICP, glutamate
- Six RCTs
- RR for early sz prophylaxis: 0.34
(95% CI:21-0.54)
- For every 100 patients treated, 10 would remain seizure-free for the first week
- No reduction in late seizures or outcome

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Treatment

Steroids

Level I

- The use of steroids is not recommended for improving outcome or reducing ICP in patients with moderate or severe traumatic brain injury (TBI)
- high-dose methylprednisolone is associated with increased mortality and is contraindicated

Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons. Guidelines for the management of severe traumatic brain injury. Steroids. J Neurotrauma 2007;24(Suppl 1):S91-S95. |

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Treatment

Calcium Channel Blockers-CR

- Prevent vasospasm, keep blood flow
- Four RCTs
- Two RCTs, traumatic SAH, nimodipine
 - Pooled OR 0.59 for death (95% CI .37-.94)
 - Pooled OR 0.67 for death, disability

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Antibiotics Prophylaxis

Level II

- **Periprocedural antibiotics for intubation should be administered to reduce the incidence of pneumonia**
 - it does not change length of stay or mortality.

Level III

- **Routine ventricular catheter exchange or prophylactic antibiotic use for ventricular catheter placement is not recommended**

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Prognosis

CT classification	n	OR (95% CI)	OR (95% CI)
Class I	5209	0.45 (0.31-0.67)	0.47 (0.32-0.70)
Class III		2.50 (2.09-3.00)	2.20 (1.54-2.63)
Class IV		3.03 (2.12-4.35)	2.22 (1.44-3.42)
Class V/VI		2.18 (1.83-2.61)	1.48 (1.27-1.71)
Traumatic subarachnoid haemorrhage	7407	2.64 (2.42-2.89)	2.01 (1.83-2.21)
Laboratory testing			
Glucose	4831	1.68 (1.54-1.83)	1.45 (1.36-1.55)
Haemoglobin	3872	0.69 (0.60-0.78)	0.76 (0.66-0.88)

Data from Murray et al.¹⁸

Table 3- Predictors of outcome in TBI

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Prognosis

	n	Odds ratio (95% CI)	
		Unadjusted	Adjusted
Age	8719	2.14 (2.00-2.28)	-
Motor score	8199		
None		5.30 (3.49-8.04)	-
Extensor		7.48 (3.60-9.95)	-
Abnormal flexion		3.58 (2.71-4.73)	-
Flexion		1.74 (1.44-2.11)	-
Pupils	7310		
Non-reactive		7.31 (5.35-9.99)	-
One reactive		2.71 (2.36-3.12)	-
Hypoxia	5661	2.08 (1.69-2.56)	1.65 (1.37-2.00)
Hypotension	6629	2.67 (2.09-3.41)	2.06 (1.64-2.69)

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Head CT Prognosis Subarachnoid Hemorrhage

- Blood in basal cisterns, 70% PPV bad
- Extent of SAH is related to outcome
 - I: Age > 45 & > 5 mm shift, 78% PPV bad
 - II: Shift > 15 mm, 70% unfavorable outcome

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Emergent Cranial Decompression Indications

- Hippocrates utilized trephination
- To evacuate extradural hematomas
- To reverse signs of tentorial herniation
- Rapid, progressive neurologic deterioration
- Coma, fixed, dilated pupil, hemiplegia and presumed skull fx on side of pupil
- Likely intracranial hematoma on same side

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Emergent Cranial Decompression Procedure

- 4 cm vertical incision
- External auditory canal is key landmark
 - > Three cm superior to zygoma
 - > Two cm anterior to ear



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Emergent Cranial Decompression Procedure

- Drill a hole, enlarge with a Burr
 - > Speed is your friend, pressure your enemy
- Careful as the inner table is perforated
 - > Resistance- give-resistance
- Epidural: clotted, unless bleeding persists
- Be prepared to replace blood loss
- Bilateral fixed pupils, or no clot, repeat on contra-lateral side

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Severe TBI Case Acute Management

- IV NS 500 cc bolus, BVM O2 100%
- Rapid sequence induction
 - > Lidocaine 100 mg IVP
 - > Midazolam 4 mg IVP
 - > Succinylcholine 100 mg IVP
- Endotracheal intubation
- Ventilator: 100%, TV 600, IMV 14, PEEP 5

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Severe TBI Case Test Results

- No fractures on x-ray
- CT head: skull fracture, epidural
- ABG: 7.30 35 280 100% BD -3
- Hb 11.4, other labs OK
- Fast exam is negative

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Severe TBI Case

ED Diagnoses

- Linear skull fracture, non-depressed
- Epidural hematoma
- Severe TBI, GCS 8-9
- Scalp laceration
- Multiple abrasions and contusions

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Severe TBI Case

ED, Hospital Disposition

- Trauma service consultation
- Neurosurgery consultation
- To OR: epidural hematoma evacuation
- Admitted to ICU, intubated 8 days
- Discharged to rehab facility: day 20

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Severe TBI Case

Patient Outcome

- Six month assessment
- Glasgow Outcome Scale Score
- Functions at home OK
- Just now beginning to drive
- Short work days
- Persistent headaches, amnesia

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Conclusions

E.D. TBI Therapy

- Despite few standards, an algorithm exists
- Treat hypotension, hypoxia, elevated ICP
- ICP monitor and ventricular drainage
- Mild hyperventilation, bolus mannitol
- Barbiturates, other ICU interventions
- Use all aggressively with decompensation

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Clinical Policy: Neuroimaging and Decision Making in Adult Mild Traumatic Brain Injury in the Acute Setting

**An ACEP / CDC Collaborative
Project**

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Introduction

- Approximately 1 million ED visits for TBI
 - Majority of TBI are "mild"
 - Challenge is to identify patients with MTBI who have neurosurgical lesions or at risk for post concussive symptoms / syndrome
- "Signature casualty of the war in Iraq"

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Introduction

- Less than 10% of patients with a GCS of 14 / 15 have evidence of an acute intracranial lesion on head CT
- Up to 30% of patients with a GCS of 13 have an acute intracranial lesion on head CT
- Less than 1% of patients with GCS of 15 need intervention

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MTBI: Definition

- Blunt head injury (or blast?)
 - GCS score of 13 – 15
 - Any period of observed or self-reported:
 - ✓ Loss of consciousness
 - ✓ transient confusion, disorientation, or impaired consciousness
 - ✓ dysfunction of memory (amnesia) around the injury
 - ✓ neurological or neuropsychological dysfunction

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MTBI: Definition

- Two large data bases have demonstrated that LOC is not a defining criterion for an intracranial lesion nor neurosurgical lesion in MTBI
- Clinical Policy inclusion criteria
 - Nonpenetrating trauma to the head
 - Presentation to the ED within 24 hours of injury
 - A GCS score of 14 or 15
 - Age 16 years or greater

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2008 Clinical Policy

- Which patients with MTBI should have a non-contrast head CT scan in the ED?
- Is there a role for head magnetic resonance imaging (MRI) over non-contrast CT in the ED evaluation of a patient with acute MTBI?
- In patients with MTBI, are brain specific serum biomarkers predictive of an acute traumatic intracranial injury?
- Can a patient with an isolated MTBI and a normal neurologic evaluation be safely discharged from the ED if a non-contrast head CT scan shows no evidence of intracranial injury?

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Which patients with MTBI should have a noncontrast head CT scan in the ED?

- Level A Recommendation: A non-contrast head Ct is indicated in head trauma patients with LOC or post traumatic amnesia only if one or more of:
 - headache
 - vomiting
 - age greater than 60 years
 - drug or alcohol intoxication
 - deficits in short term memory
 - physical evidence of trauma above the clavicle
 - posttraumatic seizure
 - GCS score less than 15
 - Coagulopathy
 - focal neurologic deficit.

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Which patients with MTBI should have a noncontrast head CT scan in the ED?

- Haydel 2000 NEJM; Class I study; 2 phases
 - Phase I 520 patients to establish predictive criteria
 - Phase II 909 patients to validate criteria
 - 7 predictors identified with 100% sensitivity for predicting intracranial lesion in patients with LOC
 - Use of criteria would decrease head CT by 22%
- High sensitivity (95-100%) but low sensitivity (5-30%) confirmed by large data bases in Italy, Holland, and Spain
- Canadian CT Head Rule has higher specificity (50%) BUT the primary outcome measure is neurosurgical lesion and minor acute intracranial lesions (e.g. small subdurals and SAH) are not considered important

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Which patients with MTBI should have a noncontrast head CT scan in the ED?

- **Level B Recommendation: A non-contrast head CT should be considered in head trauma patients with no LOC or post traumatic amnesia if:**
 - focal neurologic deficit
 - vomiting, severe
 - Headache
 - age 65 years or greater
 - physical signs of a basilar skull fracture
 - GCS score less than 15
 - Coagulopathy
 - dangerous mechanism of injury

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	Smits ¹³ OR (95%CI)	Ibanez ⁹ OR (95%CI)	Faber ⁷ OR (95%CI)
Class of evidence	II	II	III
Variable			
GCS 14	2 (1-3)	7 (4-14)	19 (14 - 26)
Neurologic deficit	2 (1-3)	7 (2-25)	19 (13-28)
Signs of basilar skull fracture	25 (13-47)	11 (6-23)	10 (6-16)
LOC	2 (1-3)	7 (4-11)	2 (2-3)
Post traumatic amnesia	1.5 (1-2)	3 (2-5)	8 (6-12)
Headache mild - moderate	1 (0.8-2)	1 (0.8-2)	-
Headache - severe		3 (2-6)	-
Vomiting	3 (2-4)	4 (2-7)	5 (3-8)
Post traumatic seizure	3 (0.8-10)	2 (0.25-17)	2 (3-5)
Alcohol or drug intoxication	1 (0.6-2)	1 (0.3-3)	-
Anticoagulation	2 (1-5)	4 (3-7)	8 (3-9)
Age ≥65 years	-	2 (1-3)	2 (1-3)
Dangerous mechanism	2 (1-4)	-	3 (2-4)

Is there a role for head MRI over noncontrast CT in the ED evaluation of a patient with acute MTBI

- MRI is up to 30% more sensitive than CT in detecting axonal shear injury
- No studies looked specifically at patients with MTBI within 24 hours of injury
- No studies have shown a clinical correlation with MRI abnormalities with neuropsychologic outcome
- No recommendations

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In patients with MTBI, are brain specific serum biomarkers predictive of an acute traumatic intracranial injury?

- Traumatic injury results in the release of proteins
 - Neuronal proteins include neuron specific enolase and tau
 - Astrocyte proteins include S-100B, CK-BB
- S-100B is best studied; found within the serum within 30 minutes with a half life of 97 minutes
 - Elevated in multiple trauma
 - Elevated in marathon runners
- Eight studies reviewed; 2 Class II
 - Sensitivities 90 – 100%
 - Specificities 4 – 65%

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In patients with MTBI, are brain specific serum biomarkers predictive of an acute traumatic intracranial injury?

- Biberthaler et al: 1309 patients with isolated MTBI and CT scan
 - Cutoff for serum level 0.1 ug / L
 - 7% had an acute intracranial lesion
 - Sensitivity 0.99 (CI 0.96 – 1.0)
 - Specificity 0.30 (CI 0.29 – 0.31)
- Level B Recommendation: In patients with MTBI without significant extracranial injuries, a CT should be considered if the S-100B serum level is >0.1 ug / L

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Can a patient with an isolated MTBI and a normal neurologic evaluation be safely discharged from the ED if a noncontrast head CT scan shows no evidence of intracranial injury?

- Stein 1992 J Trauma. Retrospective
 - 1339 patients with negative CT, none deteriorated
- Dunham 1996 J Trauma Infect Crit Care. Retrospective review of a prospectively collected data base
 - 2587 patients, no patient with a negative CT deteriorated; those patients who did deteriorate (without initial CT), did so within 4 hours
- Nagy 1999 J Trauma Infect Crit Care. Retrospective
 - 1190 patients with CT and admission
 - No patient with a negative CT deteriorated

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Can a patient with an isolated MTBI and a normal neurologic evaluation be safely discharged from the ED if a noncontrast head CT scan shows no evidence of intracranial injury?

- Geijerstam and Britton reviewed 2187 abstracts and 410 full text papers
 - 62,000 MTBI patients GCS 15
 - 3 cases of delayed adverse outcome identified
- Level B recommendation: Patients with an isolated MTBI who have a negative head CT are at minimal risk for developing an intracranial lesion and therefore may be safely discharged from the ED
- Level C Recommendation: MTBI patients discharged from the ED should be informed about post-concussive symptoms

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Postconcussive symptoms / syndrome

- Symptom complex related to TBI
 - Somatic
 - Headache, sleep disturbance, dizziness, nausea, fatigue, sensitivity to light / sound
 - Cognitive
 - Attention / concentration problems, memory problems
 - Affective
 - Irritability, anxiety, depression, emotional lability
- Incidence in MTBI patients:
 - 80% at 1 month
 - 30% at 3 months
 - 5 - 15% at 12 months



Discharge instructions: design considerations

- Post-concussive symptoms should be identified at the time of assessment. A list of these symptoms should be provided to the patient in written and verbal form and be used as a prompt for the patient to seek referral to a specialist in traumatic brain injury.
- Patients who are experiencing post-concussive symptoms should refrain from strenuous mental or physical activity until they are symptom free. They may require 2 to 3 days off work or school.

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Conclusions

- Predictors of intracranial injury after blunt head trauma exist but no prediction rule has both high sensitivity & high specificity
- There is no evidence to support MRI over CT in the acute management of TBI
- S100-B biomarker has a high sensitivity if assessed within 4 hours of injury: If less than 0.1 ug / l, a head CT can be avoided

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Conclusions

- Patients with MTBI who have a normal exam and a negative head CT can be safely discharged
- Patients with MTBI discharged from the ED should receive both verbal and written information on post-concussive symptoms and provided follow up resources

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Conclusions

Emergency Physicians & TBI

- It is a significant public health problem
- We see it commonly in the EDs
- Mild TBI in all comprehensive EDs
- Severe TBI seen in trauma centers
- EPs manage the airway and early resus
- What happens early can influence outcome

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Questions??

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