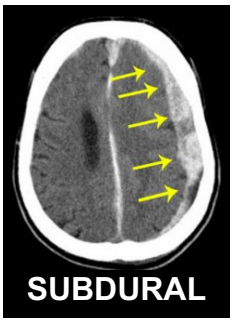
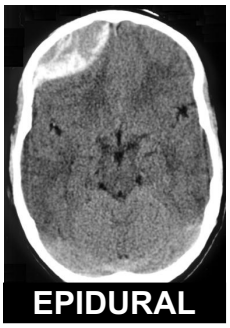
PEM GUIDE: HEAD TRAUMA

INTRODUCTION (RACHEL KOWALSKY, M.D. MPH, 2/2015)
The term “head trauma” is used to describe a spectrum of injuries. The clinician must determine which patients are at risk of intracranial injury (i.e. hemorrhage, diffuse axonal injury, cerebral edema, and increased ICP) and therefore require imaging. While there are guidelines to assist in clinical decision-making, no guideline is perfect, and the topic remains controversial. (See PEM Guide: Child Protection: Child Abuse and Neglect for abusive head trauma, See PEM Guide: Trauma: Concussion)

Subdural Hematomas are 5-10 times more common than epidural hematomas. They represent underlying cortical injury and are associated with worse outcomes. Consider shaken-impact syndrome. The extracranial blood follows the brain contours on the head CT.



Epidural Hematomas present typically with a loss of consciousness and rarely with a lucent interval. They represent arterial bleeding (the middle meningeal artery runs below the pterion or point of junction of frontal, parietal, temporal, and sphenoid bones). The extra cranial blood forms a lenticular shape on head CT.



Diffuse Axonal Injury results from axonal shearing with edema and punctate hemorrhage. Presentation may be delayed for a few hours. Coma without an extracranial hematoma or brain parenchymal injury is more likely to represent diffuse axonal injury than brainstem injury. Diffuse swelling is seen on head CT.

DIAGNOSIS
The evaluation of a patient with head trauma includes: the mechanism of injury, patient symptoms and physical exam findings. In the patient at high risk for intracranial injury, the use of neuroimaging is warranted. However, neuroimaging places the patient at risk from radiation exposure and sedation adverse events. A multi-center study of pediatric head trauma by the Pediatric Emergency Care Applied Research Network (PECARN) has been published. (See Appendix A)

SIGNS OF INCREASED INTRACRANIAL PRESSURE (ICP)
Headache
Vomiting
Papilledema
Decorticate posturing
Transtentorial herniation - brain transverses tentorium at level of incisura
Ipsilateral fixed, dilated pupil - 3rd cranial nerve compression
Contralateral hemiparesis
Foramen magnum herniation – cerebellar tonsils, brainstem
Depressed consciousness
Cushing triad - bradycardia, hypertension, irregular respirations

SIGNS OF BASILAR SKULL FRACTURE
Battle sign (bruising over the mastoid)
Raccoon eyes
Hemotympanum, blood in ear canal
Hearing loss
Facial nerve paralysis
CSF otorrhea or rhinorrhea

INITIAL MANAGEMENT OF HEAD TRAUMA

AIRWAY & BREATHING	If C-spine injury is suspected, use the jaw-thrust technique to position the airway. Apply a semi rigid cervical collar or use manual inline immobilization. Use a log-roll maneuver when turning the patient
CIRCULATION	Goal is to maintain cerebral perfusion pressure. If hypotension is present, treat with fluids. Vasocontrictors may be indicated in neurogenic shock
DISABILITY	Assess mental status using AVPU or age appropriate Glasgow Coma Scale. (Appendix B) Assess for signs of herniation (e.g. dilated fixed pupil)
EXPOSURE	Examine for signs of penetrating head trauma Facial/back trauma that may be associated with intracranial injury Assess for signs of basilar skull fracture

MANAGEMENT OF ELEVATED INTRACRANIAL PRESSURE

The Monroe-Kellie Doctrine states that intracranial volume is a constant made up of brain (70%), blood (10%), CSF (10%) and interstitial fluid (10%). With the addition of volume (hemorrhage, edema) compensatory mechanism will maintain ICP by decreasing the volume of one of the components. When the limits of compensatory mechanisms are met intracranial pressure (ICP) will increase.

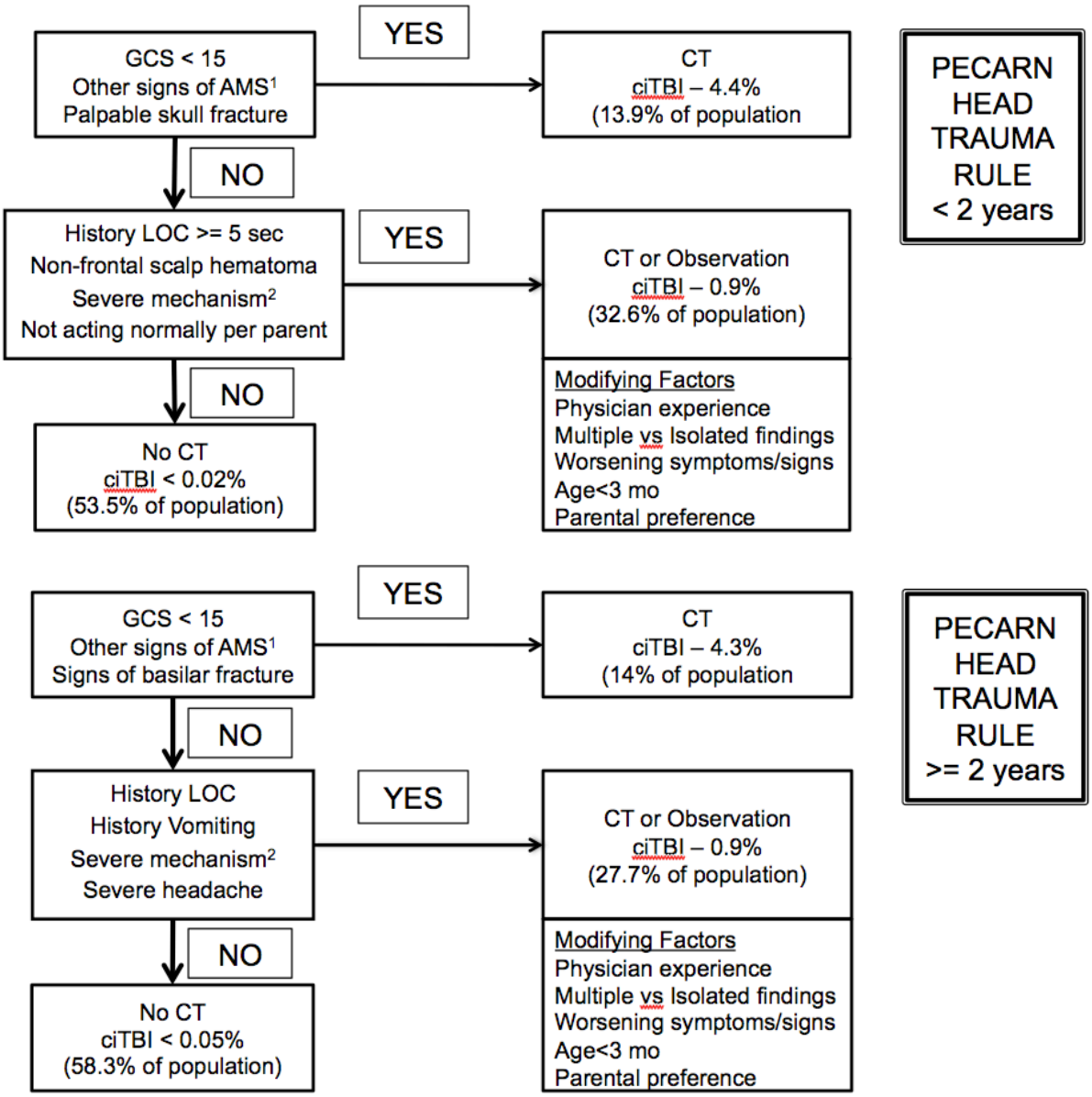
Conceptually, the management of increased ICP has been replaced by management of cerebral perfusion pressure (CPP). (CPP = mean arterial pressure (MAP) – ICP)). An increase in ICP will result in a decrease in CPP unless MAP increases. A decrease in CPP will result in cerebral ischemia and edema (secondary brain injury) further increasing ICP/

The mainstay of therapy is to maintain optimal hemodynamics (MAP) and respiratory support to ensure adequate oxygenation and perfusion. Therapeutic options are listed in the table below. Further management requires consultation with an intensivist and/or neurosurgeon. (The society for pediatric critical care medicines guidelines for the management of severe traumatic brain injury is included in the resource folder)

EMERGENCY MANAGEMENT OF CEREBRAL PERFUSION PRESSURE	
Positioning	Head of bed up to 30 degrees. Neutral head/neck position. (maximizes cerebral venous return)
Temperature	Antipyretics - avoid hyperthermia – Cerebral metabolic demand ↑ 5% for each ↑ 1°C Recent evidence suggests that cooling is ineffective
Sedation and Analgesia	Balanced to maintain blood pressure and ability to assess neurologic status
Systolic blood pressure	Maintain mean arterial pressure > 5% for age Do not restrict fluids if poor perfusion or hypotension Crystalloid bolus – 20 cc/kg for poor perfusion Consider vasoconstrictor for neurogenic shock
Controlled hyperventilation	Consider brief periods hyperventilation (PCO_2 30-35) for signs of herniation only. Otherwise normal PCO_2
Mannitol (20%)*	Dose - 0.25 - 1.0 grams/kg over 10-20 min Diuretic effect may decrease intravascular volume
Hypertonic Saline (3%)*	Dose – 0.1-1.0 ml/kg/hr (dosed to maintain ICP < 20 mm/hg and Serum osmolality < 360 mOsm/L) Benefit of increasing intravascular volume
Anticonvulsants	Keppra (Levetiracetam) – 20 mg/kg IV Indications: > 1 seizure, Seizure > 1 hour post injury Severe intraparenchymal brain injury
Corticosteroids	No proven efficacy
Barbiturates Coma	No proven efficacy, Consider if refractory
* Hyperosmolar therapy: Pediatric guidelines recommend the use of hypertonic saline and state there is insufficient evidence to recommend Mannitol. Adult guidelines recommend the use of Mannitol and state there is insufficient evidence to recommend hypertonic saline. Mannitol may decrease intravascular volume but is readily available.	

APPENDIX A: PECARN HEAD TRAUMA RULE

(Kupperman et al, Lancet 2009, PubMed ID: 19758692)



1. Other signs of AMS
   - Agitation
   - Somnolence
   - Repetitive questioning
   - Slow response to verbal communications

GCS – Glasgow Come Scale
LOC – Loss of Consciousness
AMS – Altered Mental Status

2. Severe Mechanism
   - MVC with patient ejected
   - MVC with other passenger death
   - MVC with rollover
   - Pedestrian/Bicyclist without helmet struck by motorized vehicle
   - Fall > 3 feet (< 2 years), > 5 feet (>=2 years)
   - Head struck by high impact object

APPENDIX B: NEUROLOGIC ASSESSMENT

GLASGOW COMA SCALE				
	< 1 YEAR	>1 YEAR		
Eye Opening	Spontaneous	Spontaneous		4
	To Verbal Command	To shout		3
	To Painful	To Painful		2
	No response	No response		1
Motor Response	Spontaneous	Obeys Commands		6
	Localizes Pain	Localizes Pain		5
	Withdraws to Pain	Withdraws to Pain		4
	Flexion-Decorticate	Flexion-Decorticate		3
	Extension-Decerebrate	Extension-Decerebrate		2
	No Response	No Response		1
	< 2 YEARS	2-5 YEARS	> 5 YEARS	
Verbal	Smile/Coos Appropriately	Appropriate Words/Phrases	Oriented	5
	Cries and is Consolable	Inappropriate Words	Confused / Disoriented	4
	Persistent Inappropriate crying and/or screaming	Persistent Cries Screams	Inappropriate Words	3
	Grunts, Agitated or Restless	Grunts	Incomprehensible Sounds	2
	No Response	No Response	No Response	1

AVPU CLASSIFICATION	
A	Alert
V	Responds to Voice Stimuli
P	Responds to Painful Stimuli
U	Unresponsive to all Stimuli