Provider Training Manual

EPIC-TBI
Excellence in Prehospital Injury Care
Traumatic Brain Injury Project

The EPIC Blue Book

ARIZONA FIRE CHIEFS ASSOCIATION 1966

Bureau of EMS & Trauma System
Arizona Department of Health Services

AZAA

THE UNIVERSITY OF ARIZONA

“And whoever saves a life, it is considered as if he saved an entire world.”

The Talmud

The purpose of this manual is to disseminate the background, rationale and protocols, related to the EPIC Project

Our vision is that all victims of acute traumatic brain injury receive the best prehospital assessment and treatment to maximize their chance of survival with good neurologic outcome, in Arizona and around the world.

It is because of the remarkable Arizona fire departments, EMS organizations, and their leaders that this novel project to save lives from traumatic brain injury is feasible. We recognize and appreciate the tremendous, lifesaving work that EMS providers do every day for our citizens.

Dan Spaite, MD
Co-Director, EPIC Project

Ben Bobrow, MD
Co-Director, EPIC Project

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Excellence in Prehospital Injury Care
Traumatic Brain Injury Project

Thank you for your commitment to prehospital care and improving the outcome of TBI patients.
For questions or comments please visit www.epic.arizona.edu

EPIC Project Team

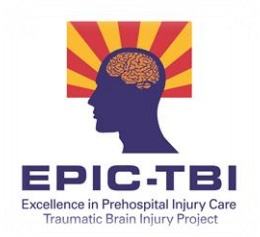
Directors
Dan Spaite, MD, FACEP
Ben Bobrow, MD, FACEP

Faculty
Chad Viscusi, MD
David Adelson, MD
Duane Sherrill, PhD
Jason Roosa, MD
Josh Gaither, MD
Kurt Denninghoff, MD, FACEP
Terry Mullins, MBA

Data Management
Mike Sotelo
Karen Rogge-Miller
Uwe Stolz, PhD, MPH
Vatsal Chikani, MPH
Cerét Clark
Julian Jiminez
Shelley Bissell
Janine Jaber

Training, Education & Implementation
Bruce Barnhart, RN, CEP
Amy Boise, NREMTP, FP-C
John Tobin, CEP
Willie Haro, CEP
Crystal Silva

References that support the recommendations are included for each section.
Further EPIC Project details can be found by visiting www.epic.arizona.edu



Traumatic Brain Injury (TBI)

Significance: Every year, over 1.4 million patients are evaluated in U.S. Emergency Departments (EDs) after Traumatic Brain Injury (TBI). Of these patients, 235,000 require hospitalization and 50,000 die.[1] The total cost for the care of this patient population in 2000 was estimated to be 60 billion dollars, with more than 2% of the US population requiring long-term assistance with activities of daily living secondary to TBI.[2] Major trauma is a leading cause of death in children and 80% of these injuries include TBI. It is difficult to overstate the massive impact of this major public health problem on our society.

What is EPIC? The Excellence in Prehospital Injury Care (EPIC) Project is a unique, statewide effort to improve survival and neurologic outcome for victims of major TBI who are cared for by the EMS agencies in Arizona. Over 5 years, the EPIC Project will work with EMS agencies to implement and evaluate TBI care and outcomes. This will happen through the linkage of prehospital data and the Arizona State Trauma Registry (ASTR), to fully document the impact of implementing the nationally-vetted TBI Guidelines in moderate and severe TBI patients throughout the state. EPIC will implement the TBI Guidelines in EMS systems that respond to 911 calls across the urban, suburban, rural, and wilderness areas of Arizona. Interventions in the "EPIC Protocol" include: optimizing the management of hemodynamics, oxygenation, and ventilation in the field in major TBI victims, with special emphasis on patients who are intubated. Our primary goal is to help EMS systems save as many lives as possible from TBI, and improve the quality of those lives saved.

Importance of EMS in TBI Care: As with other intensely time-sensitive medical emergencies, survival after TBI is profoundly impacted by *early* care of patients immediately after the event. The time-sensitive nature of these injuries is shown by the fact that half of the patients who ultimately die from TBI, do so within the first 2 hours after injury. One of the reasons outcome is so dramatically impacted by the early care is because survival is not determined solely by the severity of the *initial* insult, termed "primary brain injury." Secondary, potentially-preventable damage to the central

nervous system (CNS) often occurs after the primary injury. If the consequences of the injury are not properly identified and rapidly treated, this additional insult can quickly become irreversible. Thus, even if the patient receives *optimal* management later in the hospital, the outcome will be much worse due to the permanent damage that occurred in the *prehospital* environment. There is growing evidence that the care provided in the ***first few minutes*** after major TBI may be *more* important than what happens later. In fact, the prehospital and in-hospital care are probably powerfully synergistic. The success of the subsequent critical care and surgical interventions is probably dramatically enhanced by optimal prehospital care, which gives the patients a chance to benefit from "definitive care" at the trauma center. This means that the EMS care of TBI victims (like other time-sensitive illnesses such as cardiac arrest, STEMI, and acute stroke) ***hinges*** on the care provided by the prehospital providers. In other words, your care is what makes the difference in the outcomes for TBI victims…likely even more than the neurosurgeon. This has created both an enormous responsibility ***and*** an incredible opportunity for EMS systems to impact care and save lives.

Historical Understanding, Pathophysiology and Prehospital Management of Severe TBI: Initial observations in the early 1970s revealed that patients with an intracranial pressure (ICP) of <20 mmHg had a neurologically intact survival rate of 56% compared to only 8% for those with an ICP of >40 mmHg.[3] At that time, the treatment of severe TBI focused on the treatment/manipulation of the blood pressure and/or ICP. Given the well-known relationship between cerebral perfusion pressure (CPP), mean arterial pressure (MAP) and ICP in the equation CPP = MAP – ICP, it was believed that doing whatever was necessary to *decrease ICP* was the best way to treat TBI. Initial attempts to increase MAP were found to be ineffective in maintaining CPP in the setting of increased ICP. However, several methods were known to decrease ICP by reducing cerebrospinal fluid (CSF) volume. These included infusions of hypertonic solutions such as Mannitol and the use of "therapeutic hyperventilation." Hyperventilation became the preferred non-surgical method to reduce ICP and, for years, it was commonly used in both the prehospital and in-hospital settings to treat, and sometimes even to prevent, increased ICP.

In the 1990s, major questions began to emerge related to this ventilatory intervention. It was found that prolonged periods of hyperventilation decreased the rate of favorable outcome in severe TBI.[4-45] It was also found that even ***short periods*** of hyperventilation, causing hypocarbia [reduced

carbon dioxide (CO_2) in the blood], decreased cerebral perfusion and cerebral blood flow and increased morbidity and mortality.[12, 33, 35, 39, 46-53]

In 1995, armed with this knowledge, the first evidence-based guidelines for the management of severe TBI were established, which recommended ***against*** prophylactic hyperventilation.[54] The TBI Guidelines emphasized that there are areas of the brain that, after acute injury, are susceptible to secondary injury. Furthermore, these areas are at risk of conversion from "borderline cerebral ischemia into frank ischemia with ensuing neuronal death."[55] In other words, brain cells that are injured can die if *even moderate* hyperventilation occurs. Because of this new evidence, the therapeutic goals of management shifted from focusing on the classic CPP/MAP/ICP relationship to maintaining tissue oxygenation at the cellular level in the portions of the brain that were damaged or susceptible to damage.

There is now powerful evidence that the optimal field treatment of TBI is to focus on maintaining blood flow to the brain.

These findings have been supported by numerous studies demonstrating that even brief episodes of hypotension and hypoxia are very harmful to victims of TBI.[4-11, 46, 47, 56-71] For example, a single episode of hypotension (SBP<90 mmHg) has been shown to be associated with a ***doubling*** of mortality in TBI and a single non-spurious reading of 0_2 saturation <90% is independently associated with a ***doubling*** of mortality.[4]

We know that hyperventilation, hypotension, and hypoxia are well-established causes of secondary brain injury and each of these occurs commonly in the prehospital management of TBI.[4] Aggressive measures to prevent and treat these complications have been widely accepted and practiced in the ICU setting with improved patient outcomes.[72, 73] Although a full discussion of the CNS physiology and changes associated with hyperventilation is outside the scope of this training guide, a brief description of the most important and well-accepted physiologic changes that occur with hyperventilation can be found below and is summarized in Table 1 and Figure 1.

Table 1. Pathophysiology of Secondary CNS Injury during Hyperventilation

Parameter/ Treatment	Physiologic Change	Secondary Injury	Reference
↓ $PaCO_2$	Global CNS vasoconstriction	↓ CBF	1, 2, 37, 50, 74-77
	↑pH → left shift of oxygen-hemoglobin association curve	↓ O2 delivery to tissue	50, 75
	Cell Membrane Permeability alteration of membrane permeability → Apoptosis (programmed cell death)	Neuronal cell death	50, 75, 80, 81
↑Intrathoracic Pressure	↓ Cardiac Output (effects increase with hemorrhage)	↓MAP	50, 66, 78, 79
	↑ JVP (if JVP > ICP); ↓CPP according to CPP = MAP – JVP	↓ CBF	50, 75
Alterations in MAP and ICP	MAP response is variable during hyperventilation and may decrease significantly	Thus, CPP still *decreases* despite ↓ ICP	50, 66, 78, 79
Variations in ventilatory rate, depth, mechanics	Hyperventilation → global ↓CBF → periods of normal ventilation → ↑ blood flow to healthy brain "steals" blood from injured brain	↓ blood flow to area of injury ("Post-hyperventilatory Steal")	82-86

Abbreviations:

CBF – Cerebral Blood Flow

CNS – Central Nervous, System

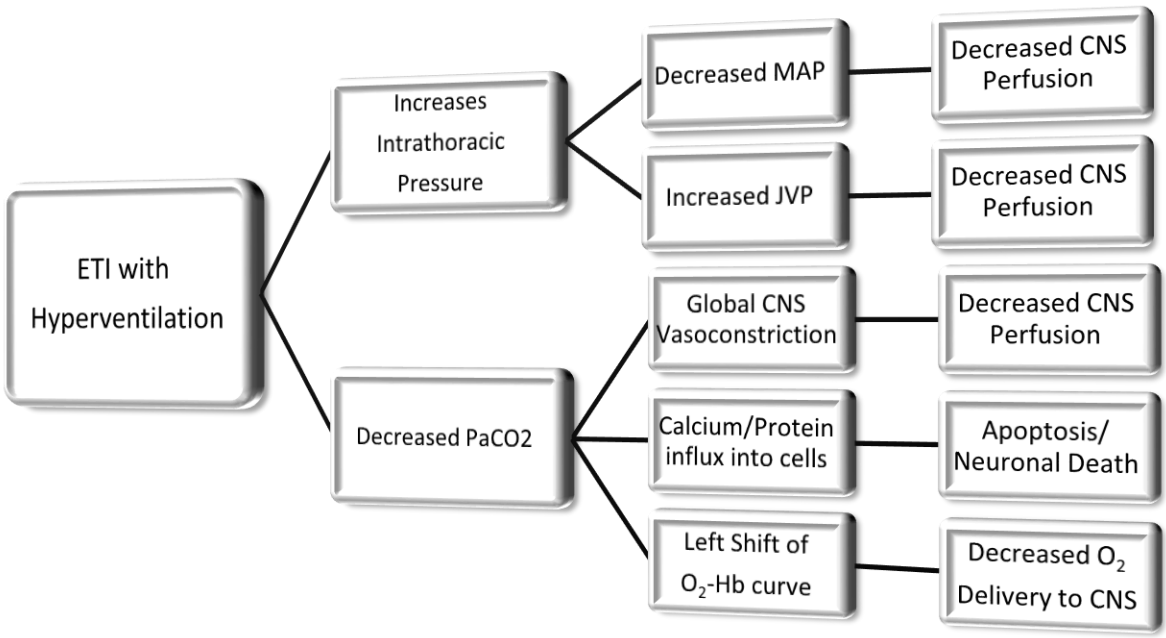
CPP—Cerebral Perfusion Pressure

ICP – Intracranial Pressure

JVP – Jugular Venous Pressure

MAP – Mean Arterial Pressure

$PaCO_2$ – Arterial Partial Pressure of Carbon Dioxide**Figure 1. Mechanisms of Secondary Injury Induced During Hyperventilation**



Abbreviations:

CNS – Central Nervous, System

ETI – Endotracheal Intubation

Hb – Hemoglobin

ICP – Intracranial Pressure (ICP)

JVP – Jugular Venous Pressure (JVP)

MAP – Mean Arterial Pressure

O_2 – Oxygen

$PaCO_2$ – Arterial Partial Pressure of Carbon Dioxide

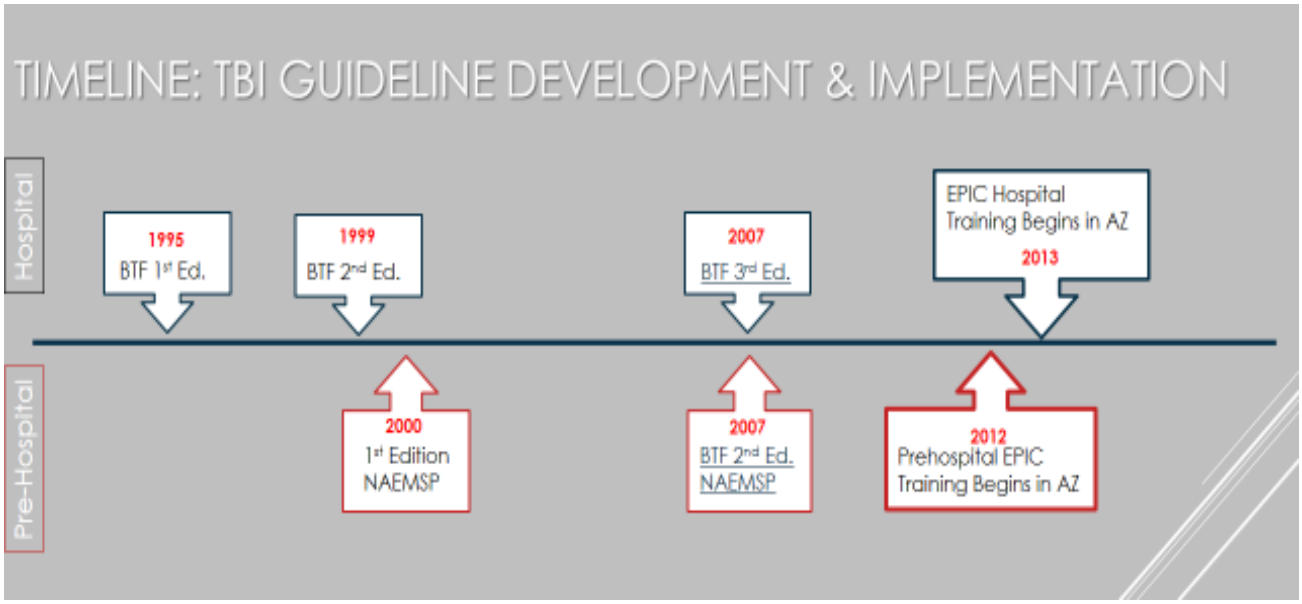
Hyperventilation (and the resulting decrease in the $PaC0_2$) causes the blood vessels supplying the brain to constrict. This results in a decrease in cerebral blood flow and is a powerful factor leading to secondary brain injury. At the cellular/neuronal level, low $C0_2$ can initiate a chain of reactions leading to cellular death through a mechanism called "apoptosis." In addition, pH changes associated with low $C0_2$ result in increased cell membrane permeability and protein shifts. This cascade of events creates free radicals and irreversible cellular damage. All of these effects from hyperventilation ultimately result in failure to supply adequate oxygen to the neurons and the greatest compromise of 0_2 delivery is in the injured regions of the CNS. The cumulative effect of hyperventilation results in significant secondary brain injury and dramatically increases morbidity and mortality from TBI.

******The optimal treatment of TBI patients in the prehospital setting centers on strictly preventing and aggressively treating alterations in cerebral blood flow and oxygenation. These changes include hypotension, hyperventilation (both intentional and inadvertent) and hypoxemia.******

What has Happened in the Past: Historically, the prehospital management of patients with severe TBI focused on reducing ICP by performing endotracheal intubation (ETI) and then intentionally hyperventilating patients. However, because of the overwhelming recent evidence revealing very detrimental effects, TBI management guidelines vetted by authoritative national organizations have radically reversed the previous approach. The guidelines have changed the focus of early management to emphasize *strict avoidance* of hyperventilation (see timeline below). This shift began in the 1990s when data indicated that prophylactic hyperventilation was associated with worse outcomes. Over the last ten years, there have been many animal and human studies demonstrating that *even short periods* of *moderate* hyperventilation during the early treatment of TBI result in increased morbidity and mortality. In fact, some studies have shown as much as a six fold increase in mortality from hyperventilating patients with severe TBI.[67]

The timeline demonstrates the shift in TBI Guideline care to encourage strict avoidance of hyperventilation as well as recognition and management of the other 3 H-Bombs: hypoxia, hypertension, and hypoglycemia. Note that the guidelines are 18 years old!**Prehospital Providers must avoid the four "H-Bombs" in caring for TBI patients.**
The "H-Bombs" of the EPIC Protocol are:

NOTE: Repeated episodes of hypotension have been associated with an increase in the likelihood of death by as much as <u>eight</u> times!!!



1. **Hyperventilation: Strict avoidance and immediate correction of Hyperventilation/low ETC0$_2$**
2. **Hypoxia: Prevention, immediate recognition, and urgent treatment of Hypoxia**
3. **Hypotension: Prevention, rapid identification, and aggressive treatment of Hypotension**
4. **Hypoglycemia: [add verbiage here]**

Why EPIC in Arizona? Arizona was selected for the EPIC Project by the National Institutes of Health (NIH) in an extremely competitive process. This is largely based upon the fact that Arizona EMS has clearly proven it is unique among EMS systems around the country. Arizona EMS has consistently been able to work collectively and cooperatively to implement, measure, and improve care for victims of many medical emergencies. The EMS systems have *tripled* neurologically intact survival from cardiac arrest, resulting in over 700 cardiac arrest survivors since 2004. In this collaborative program, through the rapid statewide adoption and implementation of the latest cardiac resuscitation protocols, Arizona EMS has accomplished what no other EMS system in the country has.

EPIC is a logical extension of this statewide network of prehospital providers and agencies. You have proven your commitment to saving lives from medical emergencies and collecting the accurate data necessary to help demonstrate to other communities how to do the same. The EPIC Team is truly honored to work on this statewide effort alongside your EMS agency.

Our collective goal is for all victims of acute traumatic brain injury in Arizona and around the world to receive the best prehospital assessment and treatment to maximize their chance of survival and good neurologic outcome.

Excellence in Prehospital Injury Care (EPIC)
Documentation Guide

A Public Health initiative to improve the quality of care for traumatic brain injury patients

Documentation necessary for optimal recognition of your care of traumatic brain injuries includes:*

Avoid	Monitor and Document	Maintain
Hyperventilation	ETCO2 readings q 5 min	35 – 45mmHg
Hypotension	B/P readings q 5 min	Systolic > 90mmHg
Hypoxia	O2 Sat readings q 5 min	> 90%
Assess	**Monitor and Document**	**Maintain**
Level of Consciousness	At least 2 accurate GCS (E/V/M components- 3/4/5 rather than just 12)	N/A
Blood Glucose	Capillary Blood Glucose Check	Glucose>70 mg/dL
Hypotension	Total amount of fluids given	Systolic>90mmHg

*Please keep in mind that all standard requirements should also be documented.

The four "H-Bombs" components of the EPIC Protocol need special monitoring and documentation:

- Hyperventilation STRICT AVOIDANCE
- Hypotension EARLY DETECTION AND RAPID CORRECTION
- Hypoxia EARLY DETECTION AND RAPID CORRECTION
- Hypoglycemia EARLY DETECTION AND RAPID CORRECTION

Why: Reduce mortality and improve outcomes of victims of moderate and severe brain injury.
How: Implementation and evaluation of current national TBI guidelines.
What: Accurate documentation of prehospital trauma care.

IMPORTANT: No individual prehospital personnel information is maintained in EPIC. The data collected is used specifically to measure the impact on patient outcome of implementing these guidelines.

EPIC4KIDS Guidelines and Algorithm*

The ABCDs in Caring for Moderate and Severe TBI in Infants and Children

*References starting on Page 28

EMS Care of moderate and severe TBI
Treatment and Monitoring Guidelines/Protocols

Infants and Children

- **Definitions:**

  - Age Definitions for Monitoring and Management:[1]
    - "Infant": Age 0-24 months
    - "Child": Age 2-14 years
    - "Late adolescence": 15-17 years

  - The prehospital identification of moderate or severe TBI: Anyone with physical trauma and a mechanism consistent with the *potential* to induce a brain injury and:
    - GCS of 12 or less
      OR
    - GCS <15 with decreasing GCS or increasing confusion
      OR
    - Multisystem trauma requiring intubation whether the primary need for intubation was from TBI or from other potential injuries
      OR
    - Post-traumatic seizures, whether they are continuing or not
      OR
    - In infants (where GCS may be difficult to obtain or interpret), decreased level of consciousness, decreased responsiveness, or any deterioration of mental status.

Overall approach to monitoring and continuous evaluation:

- Continuous O_2 saturation (sat) via pulse oximetry, continuous quantitative end-tidal CO_2 ($ETCO_2$) monitoring in intubated patients and systolic blood pressure (SBP) every 3-5 minutes.

Specific, guideline-based therapy

I. Management of airway/oxygenation:

AVOID THE H-BOMB!!!: A *single* accurate O_2 sat of <90% is *independently* associated with a *doubling* of mortality. Hypoxia kills neurons!

A. Management is initiated by continuous high-flow O_2 for all *potential* TBI cases. Emphasis is placed on prevention, identification, and treatment of hypoxia (O_2 sat <90% and/or cyanosis).[1-7] If high-flow O_2 fails to correct hypoxia, basic maneuvers for airway repositioning will be attempted, followed by reevaluation. If this does not restore O_2 saturation to 90% or greater, or if there is inadequate ventilatory effort, bag-valve-mask (BVM) ventilation will be performed using appropriate airway

adjuncts (e.g., oropharyngeal airway). It should be noted that most infants and children can have their airway managed well using basic maneuvers and BVM.

B. If airway compromise or hypoxia persists after these interventions, ETI will be performed when an experienced ALS provider is available.[1-3,6,8-13] Following ETI, tube placement will be confirmed via multiple means including $ETCO_2$ detection and/or capnography.

II. Management of ventilation: Special emphasis is placed on identifying and treating hypoventilation as well as preventing hyperventilation when assisting ventilation.

AVOID THE H-BOMB!!!: In intubated patients, hyperventilation is *independently* associated with *at least* a doubling of mortality and some studies have shown that *even moderate* hyperventilation can increase the risk of death by *six* times. Hyperventilation kills neurons!

IMPORTANT!!!: It has been shown repeatedly that inadvertent hyperventilation happens *reliably* if not meticulously prevented by proper external means. No one, no matter how experienced, can properly ventilate without ventilatory adjuncts (ventilators, ventilation rate timers (VRTs), $ETCO_2$ monitoring, cadence devices, resistance-controlled BVMs).

A. AVOID THE H-BOMB!!! Hypoventilation [ineffective respiratory rate for age, shallow or irregular respirations, periods of apnea, or measured hypercarbia (elevated $ETCO_2$)]: If there is evidence of hypoventilation despite high-flow O_2 therapy, assisted ventilation will be performed via BVM and, if ineffective, ETI will be performed if an experienced ALS provider is present.[1-3,12-15]

B. Intubated patients: After ETI, $ETCO_2$ will be strictly maintained between 35 and 45 mmHg when monitoring is available.[1-3,15-17]

1. Agencies without $ETCO_2$ monitors are strongly encouraged to use ventilation cadence devices and to assist in maintaining age-appropriate ventilation rates and decreasing the risk of inadvertent hyperventilation.[1-3,11,14,15,18-26]

- Target ventilatory rates from the National TBI Guidelines:[1,27]
  - Infants: (age 0-24 months): 25 breaths per minute (bpm);
  - Children: (age 2-14): 20 bpm;
  - Older adolescents: (age 15-17): 10 bpm (same as adults)

2. Whenever possible, ventilators should be used post-intubation to optimize ventilatory parameters and O_2 therapy.[1,14,15,28-30] This is the *best* way to care for an intubated TBI patient.

- Target tidal volume (TV) will be 7cc/kg with rates adjusted to keep the $ETCO_2$ within target range (35-45 mmHg).

Note: This is consistent with the TBI guidelines and the recent literature showing that intrathoracic pressure, lung mechanics, hemodynamics, and ICP are optimized by this TV compared to the "classic" 10-12 cc/kg that remains common in many settings.[14,18,30-37]

C. Non-intubated patients: All relevant monitoring/treatment will be applied except $ETCO_2$ monitoring.

III. Management of blood pressure: In patients with a *potential* for TBI, strong emphasis is placed on preventing and *aggressively* treating even a *single* episode of hypotension.

- AVOID THE H-BOMB!!!: A *single* episode of hypotension is *independently* associated with *at least* a doubling of mortality. Amazingly, repeated episodes of hypotension can increase the risk of death by as much as *eight* times. Hypotension kills neurons!
- Hypotension will be defined as systolic blood pressure (SBP) below the 5th percentile for age. This will be estimated using the following formula:[1,38]
  - Infants/children age <10: 70 mmHg + (age X 2)
  - Children age ≥10: 90 mmHg (same as adults)
    - Good "rules of thumb" to remember:
      - ✓ Infant = 70 mmHg
      - ✓ 5 year old = 80 mmHg
      - ✓ 10 and older = 90 mmHg

A. Treatment of hypotension: Even a *single* hypotensive measurement (for age) will initiate intravenous (IV) fluid resuscitation. For hypotension or other signs of shock, IV normal saline will be given. Sufficient volume (via 20cc/kg boluses every 5 minutes) will be given to return SBP to at least the 5th percentile estimate.

- Once hypotension has been corrected, IV administration of NS should occur at sufficient rate to keep the patient non-hypotensive.

  Note: If the rapid infusion of the initial bolus of crystalloid does not correct the hypotension, there should be no hesitation to continue aggressive fluid resuscitation.

- Intraosseous access should be attempted if all three of the following criteria are met: 1) there is hypotension or other signs of shock, 2) peripheral venous access cannot be quickly established, and 3) the patient's mental status is such that they can tolerate the procedure without undue pain.

B. Treatment of hypertension: In TBI, treatment of acute hypertension is not recommended.[1-3,39] However, IV fluids will be restricted to a minimal "keep open"

rate in infants/young children with SBP ≥100 mmHg and in older children/adolescents with SBP ≥130 mmHg.

IV. AVOID THE H-BOMB!!! Assessment and management of hypoglycemia: In patients with any alteration in mental status, *always* check for hypoglycemia early in the clinical course. Hypoglycemia can *mimic* TBI as a cause of altered mental status. Hypoglycemia can also *cause* TBI.

- Obtain fingerstick or serum glucose level. If glucose level is <*70mg/dl* then:

1. Administer dextrose IV:
   - Newborn (birth to 2 months):
     - Administer 5ml/kg of D10 solution IV
   - Infants and toddlers (3 months to 3 years):
     - Administer 2ml/kg of D25 solution, max dose = 100ml (25 g).
   - Children age 4 and older:
     - Administer 1ml/kg of D50 solution, max dose = 50ml
2. Repeat blood sugar in 10 minutes and, if still <70mg/dl, repeat dose x 1.
   - If no response then contact medical personnel for further direction
3. If IV access unsuccessful, dextrose may be given IO.
4. If IV and IO unsuccessful, administer Glucagon 0.03mg/kg IM, max dose 1mg

NOTES:

A. If there are differences between your regional/agency protocols or standing orders for treating hypoglycemia in the setting of TBI, you may use either the EPIC protocol above or your regional/local protocol. If in doubt, check with your medical director.

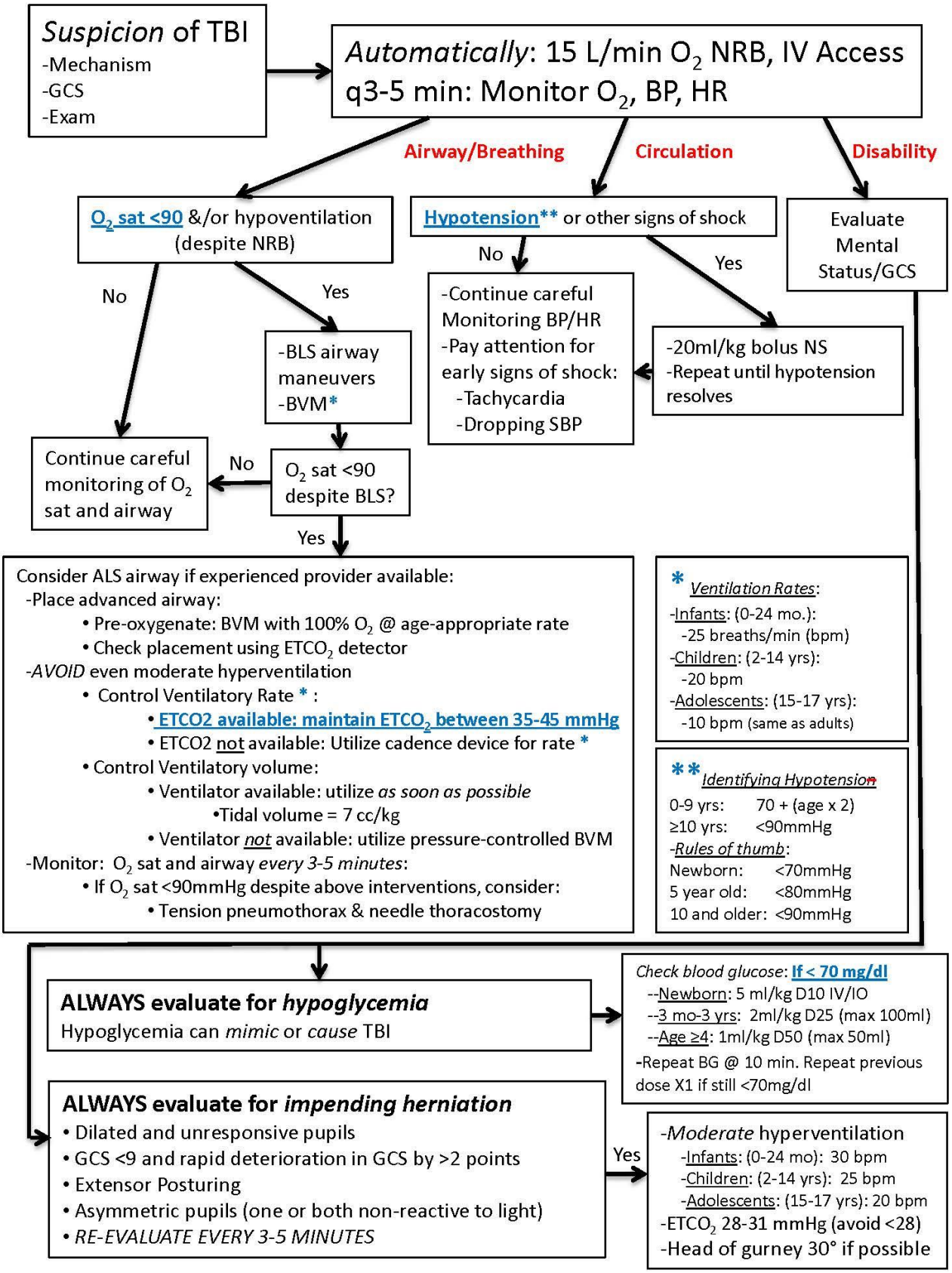
B. All dosing of dextrose and glucagon may be determined by length-based resuscitation tape rather than weight estimations if that is preferred by the agency/medical director

C. Mix D25 or D10 using either your regional/local protocols or the following:

--D25: Make D25 by removing 25CC from a 50CC bag and inject 25 CCs of D50 into the bag. Then remove an appropriate amount of the D25 and administer volume according to weight.

--D10: Make D10NS by removing 50CC from a 250CC bag of NS and then injecting one amp (50CC) of D50 into the bag. Then remove an appropriate amount of the D10NS and administer volume according to weight.

EPIC4KIDS Algorithm



ADULT EPIC Guidelines and Algorithm*

The ABCDs in Caring for

Moderate and Severe TBI

*References starting on Page 29

EMS Care of moderate and severe TBI
Treatment and Monitoring Guidelines/Protocols
ADULTS

Definition: Adult , Age ≥18

The prehospital identification of moderate or severe TBI: Anyone with physical trauma and a mechanism consistent with the *potential* to have induced a brain injury and:

- GCS of 12 or less
  OR
- GCS <15 with decreasing GCS or increasing confusion
  OR
- Multisystem trauma requiring intubation whether the primary need for intubation was from TBI or from other potential injuries
  OR
- Post-traumatic seizures whether they are continuing or not

Overall approach to monitoring and continuous evaluation:

Continuous O_2 saturation (sat) via pulse oximetry, continuous quantitative end-tidal CO_2 ($ETCO_2$) monitoring in intubated patients, and systolic blood pressure (SBP) every 3-5 minutes.

Specific, guideline-based therapy:

I. Management of airway/oxygenation:

AVOID THE H-BOMB!!!: A *single* accurate O_2 sat of <90% is *independently* associated with a *doubling* of mortality. Hypoxia kills neurons!

A. Management is initiated by continuous high-flow O_2 for all potential TBI cases. Emphasis is placed on prevention, identification, and treatment of hypoxia (O_2 sat <90% and/or cyanosis).[1-6] If high-flow O_2 fails to correct hypoxia, basic maneuvers for airway repositioning will be attempted, followed by reevaluation. If this does not restore O_2 sat to 90% or greater, or if there is inadequate ventilatory effort, bag-valve-mask ventilation will be performed using appropriate airway adjuncts (e.g., oropharyngeal airway).

B. If airway compromise or hypoxia persists after these interventions, ETI will be performed when an experienced ALS provider is available.[1,2,5,7-10] Following ETI, tube placement will be confirmed via multiple means including $ETCO_2$ detection and/or capnography.

II. Management of ventilation: Special emphasis is placed on identifying and treating hypoventilation as well as preventing hyperventilation when assisting ventilation.

AVOID THE H-BOMB!!!: In intubated patients, hyperventilation is *independently* associated with *at least* a doubling of mortality and some studies have shown that *even moderate* hyperventilation can increase the risk of death by *six* times. Hyperventilation kills neurons!

IMPORTANT!!!: It has been shown repeatedly that inadvertent hyperventilation happens *reliably* if not meticulously prevented by proper external means. No one, no matter how experienced, can properly ventilate without ventilatory adjuncts (ventilators, $ETCO_2$ monitoring, cadence devices, resistance-controlled ambu bags).

A. AVOID THE H-BOMB!!! Hypoventilation (ineffective respiratory rate, shallow or irregular respirations, or periods of apnea): If there is evidence of hypoventilation despite high-flow O_2 therapy, assisted ventilation will be performed via BVM and, if ineffective, ETI will be performed if an experienced ALS provider is present.[1,2,11,12]

B. Intubated patients: After ETI, $ETCO_2$ levels will be strictly maintained between 35 and 45 mmHg when monitoring is available.[1,2,12-15]

1. Agencies without $ETCO_2$ monitors are strongly encouraged to use ventilation cadence devices and pressure-controlled BVMs to assist in maintaining a respiratory rate of 10 breaths per minute and decrease the risk of inadvertent hyperventilation.[1,2,10-12,16-24]

2. Ventilators will be used post-intubation whenever available to optimize ventilatory mechanics and O_2 therapy.[11,12,25-27] This is the *best* way to care for an intubated TBI patient.

- Target tidal volume (TV) will be 7cc/kg with vent rates adjusted to keep the $ETCO_2$ within target range (35-45 mmHg). This is consistent with the National TBI guidelines and with the recent literature showing that intrathoracic pressure, lung mechanics, hemodynamics, and ICP are optimized by this TV compared to the "classical" 10-12 cc/kg that remains common in many settings.[11,16,27-34]

C. Non-intubated patients: All relevant monitoring/treatment will be applied except $ETCO_2$ monitoring.

III. Management of blood pressure: In patients with a *potential* for TBI, SBP ≥ 90 mmHg should be maintained. Strong emphasis is placed on preventing and *aggressively* treating even a *single* episode of SBP <90 mmHg.[1-5,35-48]

AVOID THE H-BOMB!!!: A *single* episode of SBP <90 is *independently* associated with *at least* a doubling of mortality. Amazingly, repeated episodes of hypotension can increase the risk of death by as much as *eight* times. Hypotension kills neurons!

A. Treatment of hypotension: Even a *single* SBP measurement <90 mmHg will initiate intravenous (IV) fluid resuscitation with an initial bolus of 1 liter of normal saline or Ringer's Lactate. This will be followed by IV administration of isotonic fluids at sufficient rate and volume to keep SBP ≥90 mmHg.[1,2] If the rapid infusion of the first liter of crystalloid does not correct the hypotension, there should be no hesitation to continue aggressive fluid resuscitation.

B. Treatment of hypertension: In TBI, treatment of acute hypertension is not recommended.[1,2,49] However, IV fluids should be restricted to a minimal "keep open" rate in patients with SBP ≥140 mmHg.

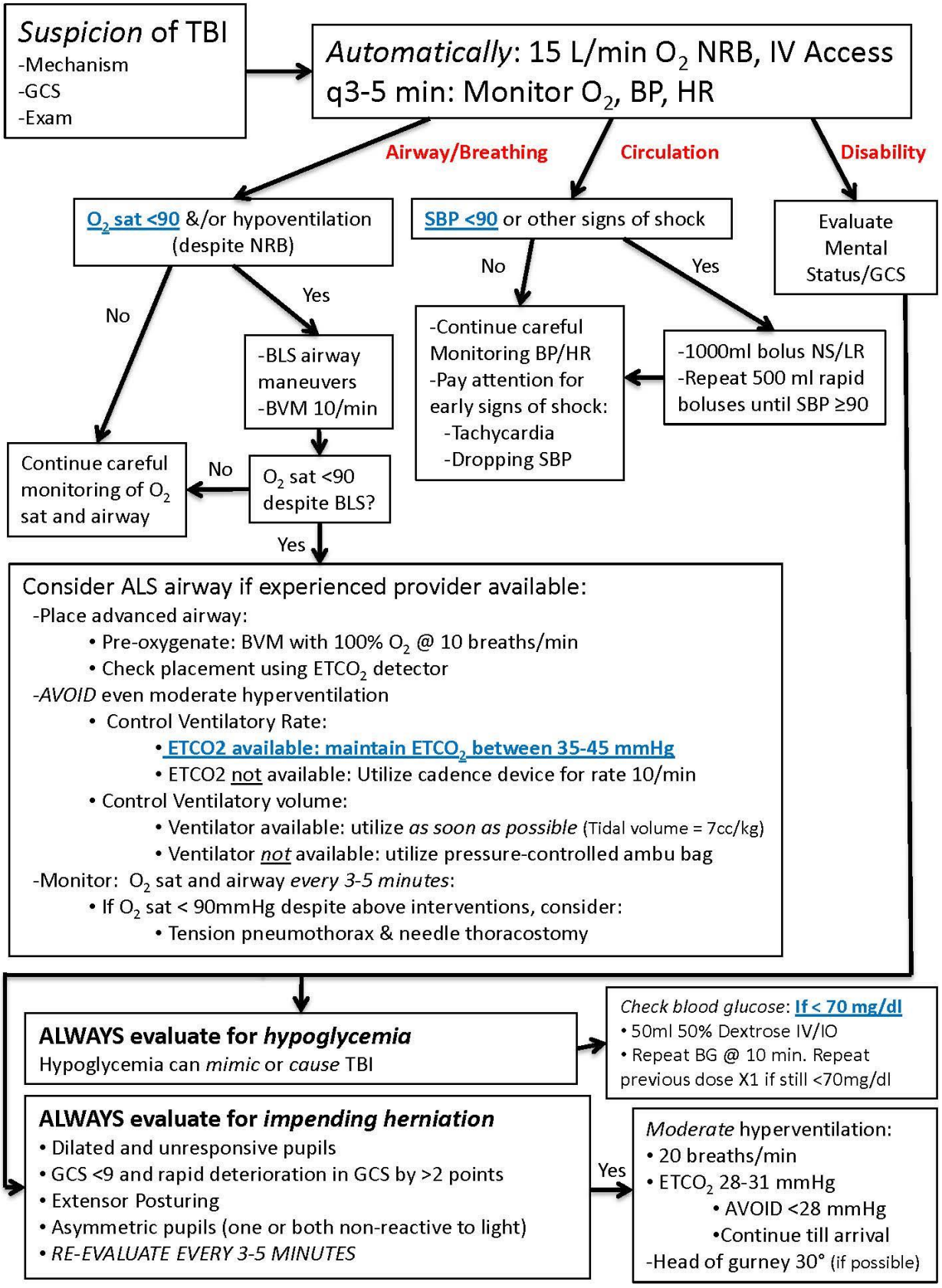
IV. AVOID THE H-BOMB!!! Assessment and management of hypoglycemia: In patients with any alteration in mental status, *always* check for hypoglycemia early in the clinical course. Hypoglycemia can *mimic* TBI as a cause of altered mental status. It can also can *cause* TBI (e.g., low blood sugar → leads to decreased LOC → leads to a motor vehicle crash in a hypoglycemic driver).

- Obtain fingerstick or serum glucose level. If <*70mg/dl* then:

1. Administer 50ml 50% dextrose (D50) IV
2. Repeat blood sugar in 10 minutes and, if still <70mg/dl, repeat dose x 1.
   --If no response then contact medical personnel for further direction
3. If IV access unsuccessful, dextrose may be given IO.
4. If IV and IO unsuccessful, administer glucagon 1.0 mg IM

NOTE: If there are differences between your regional/agency protocols orstanding orders for treating hypoglycemia in the setting of TBI, you may use either the EPIC protocol above or your regional/local protocol. If in doubt, check with your medical director.

ADULT EPIC Algorithm


Suspicion of TBI
-Mechanism
-GCS
-Exam
Automatically: 15 L/min O_2 NRB, IV Access
q3-5 min: Monitor O_2, BP, HR
Airway/Breathing
Circulation
Disability
O_2 sat <90 &/or hypoventilation (despite NRB)
SBP <90 or other signs of shock
Evaluate Mental Status/GCS
No
Yes
No
Yes
-BLS airway maneuvers
-BVM 10/min
-Continue careful Monitoring BP/HR
-Pay attention for early signs of shock:
-Tachycardia
-Dropping SBP
-1000ml bolus NS/LR
-Repeat 500 ml rapid boluses until SBP ≥90
Continue careful monitoring of O_2 sat and airway
No
O_2 sat <90 despite BLS?
Yes
Consider ALS airway if experienced provider available:
-Place advanced airway:
• Pre-oxygenate: BVM with 100% O_2 @ 10 breaths/min
• Check placement using $ETCO_2$ detector
-*AVOID* even moderate hyperventilation
• Control Ventilatory Rate:
• ETCO2 available: maintain $ETCO_2$ between 35-45 mmHg
• ETCO2 not available: Utilize cadence device for rate 10/min
• Control Ventilatory volume:
• Ventilator available: utilize *as soon as possible* (Tidal volume = 7cc/kg)
• Ventilator *not* available: utilize pressure-controlled ambu bag
-Monitor: O_2 sat and airway *every 3-5 minutes*:
• If O_2 sat < 90mmHg despite above interventions, consider:
• Tension pneumothorax & needle thoracostomy
ALWAYS evaluate for *hypoglycemia*
Hypoglycemia can *mimic* or *cause* TBI
Check blood glucose: If < 70 mg/dl
• 50ml 50% Dextrose IV/IO
• Repeat BG @ 10 min. Repeat previous dose X1 if still <70mg/dl
ALWAYS evaluate for *impending herniation*
• Dilated and unresponsive pupils
• GCS <9 and rapid deterioration in GCS by >2 points
• Extensor Posturing
• Asymmetric pupils (one or both non-reactive to light)
• *RE-EVALUATE EVERY 3-5 MINUTES*
Yes
Moderate hyperventilation:
• 20 breaths/min
• $ETCO_2$ 28-31 mmHg
• AVOID <28 mmHg
•Continue till arrival
-Head of gurney 30° (if possible)

The EPIC Project

- Keep SBP > 90mmHg
- Keep O2 Saturation > 90%
- Keep ETCO2 35-45 unless signs of herniation*
- AVOID the H-Bombs:
  - NO Hyperventilation
  - NO Hypoxia
  - NO Hypotension
  - NO Hypoglycemia

GCS

Eye Opening	
Spontaneously	4
To Command	3
To Pain	2
None	1
Best Verbal Response	
Oriented	5
Confused	4
Inappropriate	3
Incoherent	2
None	1
Best Motor Response	
Obeys Commands	6
Localizes Pain	5
Withdraws to Pain	4
Flexes to Pain	3
Extends to Pain	2
None	1

www.epic.arizona.edu

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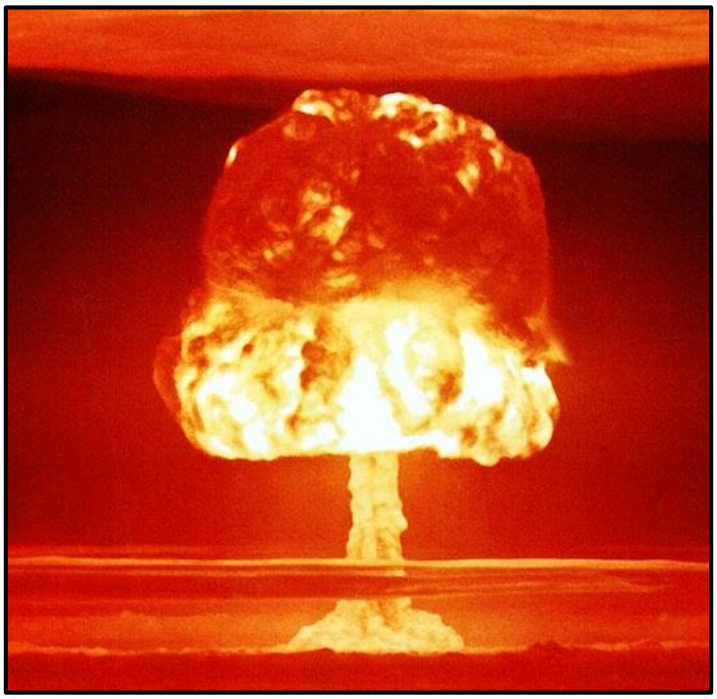

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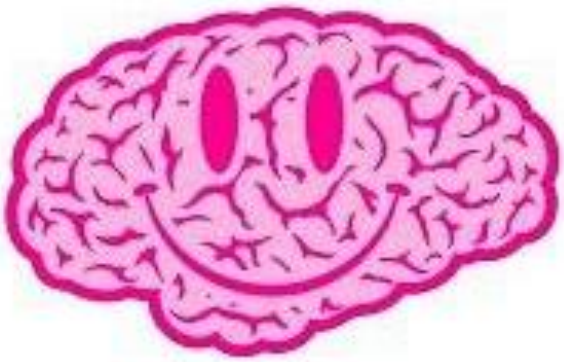

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