“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.

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Co-Director, EPIC Project

Ben Bobrow, MD
Co-Director, EPIC 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
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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. 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. 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.\textsuperscript{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 \textit{against} prophylactic hyperventilation.\textsuperscript{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.”\textsuperscript{55} In other words, brain cells that are injured can die if \textit{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.

\begin{center}
\textbf{There is now powerful evidence that the optimal field treatment of TBI is to focus on maintaining blood flow to the brain.}
\end{center}

These findings have been supported by numerous studies demonstrating that even brief episodes of hypotension and hypoxia are very harmful to victims of TBI.\textsuperscript{4-11, 46, 47, 56-71} For example, a single episode of hypotension (SBP<90 mmHg) has been shown to be associated with a \textit{doubling} of mortality in TBI and a single non-spurious reading of O$_2$ saturation <90% is independently associated with a \textit{doubling} of mortality.\textsuperscript{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.\textsuperscript{4} Aggressive measures to prevent and treat these complications have been widely accepted and practiced in the ICU setting with improved patient outcomes.\textsuperscript{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

<table>
<thead>
<tr>
<th>Parameter/Treatment</th>
<th>Physiologic Change</th>
<th>Secondary Injury</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ PaCO₂</td>
<td>Global CNS vasoconstriction</td>
<td>▼ CBF</td>
<td>1, 2, 37, 50, 74-77</td>
</tr>
<tr>
<td></td>
<td>↑pH → left shift of oxygen-hemoglobin association curve</td>
<td>▼ O2 delivery to tissue</td>
<td>50, 75</td>
</tr>
<tr>
<td></td>
<td>Cell Membrane Permeability</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>alteration of membrane permeability → Apoptosis (programmed cell death)</td>
<td>Neuronal cell death</td>
<td>50, 75, 80, 81</td>
</tr>
<tr>
<td>↑ Intrathoracic</td>
<td>▼ Cardiac Output (effects increase with hemorrhage)</td>
<td>▼ MAP</td>
<td>50, 66, 78, 79</td>
</tr>
<tr>
<td>Pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ JVP (if JVP &gt; ICP); ▼ CPP according to CPP = MAP – JVP</td>
<td>▼ CBF</td>
<td>50, 75</td>
</tr>
<tr>
<td>Alterations in MAP</td>
<td>MAP response is variable during hyperventilation and may decrease significantly</td>
<td>Thus, CPP still decreases despite</td>
<td>50, 66, 78, 79</td>
</tr>
<tr>
<td>and ICP</td>
<td></td>
<td>▼ ICP</td>
<td></td>
</tr>
<tr>
<td>Variations in</td>
<td>Hyperventilation → global ▼ CBF → periods of normal ventilation → ▲ blood flow</td>
<td>▼ blood flow to area of injury</td>
<td>82-86</td>
</tr>
<tr>
<td>ventilatory rate,</td>
<td>to healthy brain “steals” blood from injured brain</td>
<td>(&quot;Post-hyperventilatory Steal&quot;)</td>
<td></td>
</tr>
<tr>
<td>depth, mechanics</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations:
CBF – Cerebral Blood Flow  
CNS – Central Nervous, System  
CPP—Cerebral Perfusion Pressure  
ICP – Intracranial Pressure  
JVP – Jugular Venous Pressure  
MAP – Mean Arterial Pressure  
PaCO₂ – 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
O₂ – Oxygen
PaCO₂ – Arterial Partial Pressure of Carbon Dioxide
Hyperventilation (and the resulting decrease in the PaCO₂) 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 CO₂ can initiate a chain of reactions leading to cellular death through a mechanism called “apoptosis.” In addition, pH changes associated with low CO₂ 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 O₂ 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 eight times!!!**

1. Hyperventilation: Strict avoidance and immediate correction of Hyperventilation/low ETCO₂
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:*

<table>
<thead>
<tr>
<th>Avoid</th>
<th>Monitor and Document</th>
<th>Maintain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperventilation</td>
<td>ETCO2 readings q 5 min</td>
<td>35 – 45mmHg</td>
</tr>
<tr>
<td>Hypotension</td>
<td>B/P readings q 5 min</td>
<td>Systolic &gt; 90mmHg</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>O2 Sat readings q 5 min</td>
<td>&gt; 90%</td>
</tr>
</tbody>
</table>

Assess

<table>
<thead>
<tr>
<th>Maintain</th>
<th>Monitor and Document</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of Consciousness</td>
<td>At least 2 accurate GCS (E/V/M components- 3/4/5 rather than just 12)</td>
</tr>
<tr>
<td>Blood Glucose</td>
<td>Capillary Blood Glucose Check</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Total amount of fluids given</td>
</tr>
</tbody>
</table>

*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
- GCS <15 with decreasing GCS or increasing confusion
- Multisystem trauma requiring intubation whether the primary need for intubation was from TBI or from other potential injuries
- Post-traumatic seizures, whether they are continuing or not
- 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. Following ETI, tube placement will be confirmed via multiple means including ETCO₂ 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), ETCO₂ 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 ETCO₂)]: If there is evidence of hypoventilation despite high-flow O₂ therapy, assisted ventilation will be performed via BVM and, if ineffective, ETI will be performed if an experienced ALS provider is present.

B. **Intubated patients**: After ETI, ETCO₂ will be strictly maintained between 35 and 45 mmHg when monitoring is available.

1. Agencies without ETCO₂ 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.

   - Target ventilatory rates from the National TBI Guidelines:
     - **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 O₂ therapy. 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 ETCO₂ 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.\textsuperscript{14,18,30-37}

C. \textbf{Non-intubated patients}: All relevant monitoring/treatment will be applied except ETCO\textsubscript{2} monitoring.

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

- AVOID THE H-BOMB!!!: A \textit{single} episode of hypotension is \textit{independently} associated with at least a doubling of mortality. Amazingly, repeated episodes of hypotension can increase the risk of death by as much as \textit{eight} times. Hypotension kills neurons!
- Hypotension will be defined as systolic blood pressure (SBP) below the 5\textsuperscript{th} percentile for age. This will be estimated using the following formula:\textsuperscript{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. \textbf{Treatment of hypotension}: Even a \textit{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 5\textsuperscript{th} 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. \textbf{Treatment of hypertension}: In TBI, treatment of acute hypertension is not recommended.\textsuperscript{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:
   a. Newborn (birth to 2 months):
      o Administer 5ml/kg of D10 solution IV
   b. Infants and toddlers (3 months to 3 years):
      o Administer 2ml/kg of D25 solution, max dose = 100ml (25 g).
   c. Children age 4 and older:
      o 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

**Suspicion of TBI**
- Mechanism
- GCS
- Exam

**Automatically:** 15 L/min O₂ NRB, IV Access
q3-5 min: Monitor O₂, BP, HR

Airway/Breathing

- **O₂ sat <90** &/or hypoventilation (despite NRB)
  - No
  - Yes

  - **BLS** airway maneuvers
  - **BVM**

  **No**

  **Yes**

  - Continue careful monitoring of O₂ sat and airway
  - **O₂ sat <90** despite BLS?

- **Hypotension** or other signs of shock

  **No**

  - Continue careful Monitoring BP/HR
  - Pay attention for early signs of shock:
    - Tachycardia
    - Dropping SBP

  **Yes**

  - -20ml/kg bolus NS
  - Repeat until hypotension resolves

- **Evaluate Mental Status/GCS**

Consider ALS airway if experienced provider available:
- Place advanced airway:
  - Pre-oxygenate: BVM with 100% O₂ at age-appropriate rate
  - Check placement using ETCO₂ detector
  - AVOID even moderate hyperventilation
    - Control Ventilatory Rate *:
      - ETCO₂ available: maintain ETCO₂ between 35-45 mmHg
      - ETCO₂ not available: Utilize cadence device for rate *
    - Control Ventilatory volume:
      - Ventilator available: utilize as soon as possible
      - Tidal volume = 7 cc/kg
      - Ventilator not available: utilize pressure-controlled BVM
  - Monitor: O₂ sat and airway every 3-5 minutes:
    - If O₂ sat <90 mmHg despite above interventions, consider:
      - Tension pneumothorax & needle thoracostomy

**ALWAYS evaluate for hypoglycemia**
Hypoglycemia can mimic or cause TBI

**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

**Ventilation Rates:**
- Infants: (0-24 mo.):
  - 25 breaths/min (bpm)
- Children: (2-14 yrs):
  - 20 bpm
- Adolescents: (15-17 yrs):
  - 10 bpm (same as adults)

**Identifying Hypotension**
0-9 yrs: 70 + (age x 2)
10+ yrs: <90mmHg

**Rules of thumb**
- Newborn: <70mmHg
- 5 year old: <80mmHg
- 10 and older: <90mmHg

**Check blood glucose:** If < 70 mg/dl
- Newborn: 5 ml/kg D10 IV/IO
- 3 mo-3 yrs: 2ml/kg D25 (max 100ml)
- >3 yrs: 1ml/kg D50 (max 50ml)
- Repeat BG @ 10 min. Repeat previous dose X1 if still <70mg/dl

**Moderate hyperventilation**
- Infants: (0-24 mo.): 30 bpm
- Children: (2-14 yrs): 25 bpm
- Adolescents: (15-17 yrs): 20 bpm
- ETCO₂ 28-31 mmHg (avoid <28)
- Head of gurney 30° if possible
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
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  OR
- Post-traumatic seizures whether they are continuing or not

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   A. Management is initiated by continuous high-flow O₂ for all potential TBI cases. Emphasis is placed on prevention, identification, and treatment of hypoxia (O₂ sat <90% and/or cyanosis).\textsuperscript{1-6} If high-flow O₂ fails to correct hypoxia, basic maneuvers for airway repositioning will be attempted, followed by reevaluation. If this does not restore O₂ 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.\textsuperscript{1,2,5,7-10} Following ETI, tube placement will be confirmed via multiple means including ETCO₂ 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 $\geq$ 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 *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 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.

**ADULT EPIC Algorithm**
**Suspicion of TBI**
- Mechanism
- GCS
- Exam

**Automatically:** 15 L/min O₂ NRB, IV Access q3-5 min: Monitor O₂, BP, HR

- Airway/Breathing
  - **O₂ sat <90** &/or hypoventilation (despite NRB)
    - No
      - Continue careful monitoring of O₂ sat and airway
    - Yes
      - BLS airway maneuvers
      - BVM 10/min
      - O₂ sat <90 despite BLS?
        - No
          - Continue careful monitoring of O₂ sat and airway
        - Yes
          - SBP <90 or other signs of shock
            - No
              - - Continue careful Monitoring BP/HR
                - Pay attention for early signs of shock:
                  - Tachycardia
                  - Dropping SBP
            - Yes
              - -1000ml bolus NS/LR
                - Repeat 500 ml rapid boluses until SBP ≥90

- Circulation
  - Evaluate Mental Status/GCS

- Disability

**Consider ALS airway if experienced provider available:**
- Place advanced airway:
  - Pre-oxygenate: BVM with 100% O₂ @ 10 breaths/min
  - Check placement using ETCO₂ detector
  - **AVOID** even moderate hyperventilation
    - Control Ventilatory Rate:
      - **ETCO₂ available:** maintain ETCO₂ 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: O₂ sat and airway every 3-5 minutes:
  - If O₂ Sat < 90mmHg despite above interventions, consider:
    - Tension pneumothorax & needle thoracostomy

**ALWAYS evaluate for hypoglycemia**
Hypoglycemia can mimic or cause TBI

**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**

**Check blood glucose:** If < 70 mg/dl
- 50ml 50% Dextrose IV/IO
- Repeat BG @ 10 min. Repeat previous dose X1 if still <70mg/dl

**Moderate hyperventilation:**
- 20 breaths/min
- ETCO₂ 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

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<tr>
<td>To Pain</td>
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**Best Verbal Response**

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**Best Motor Response**

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<td>Extends to Pain</td>
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<tr>
<td>None</td>
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</table>
MAIN REFERENCES:


**PEDIATRIC REFERENCES:**


ADULT REFERENCES:


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

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This is a brain without great EMS Care

Thank you for your commitment to prehospital care and improving the outcome of TBI patients!

EPIC is funded by a grant from the National Institutes of Health. For questions or comments please visit www.epic.arizona.edu