The EPIC Blue Book

Provider Training Manual

EPIC-MT

An EPIC-TBI Partner

This program and manual was adopted from EPIC TBI Program.

Emergency Medical Services & Trauma Systems Section
Department of Public Health & Human Services

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Excellence in Prehospital Injury Care in Montana (EPIC-MT)

MISSION

"To reduce death and disability of Montanans who experience traumatic brain injury (TBI) through evidence-based best practices, education, and continuous quality improvement."

BACKGROUND

Each year, over 300 Montanans die from TBI and another 800 are hospitalized. Nationally, TBI leads to 2.2 million emergency department visits, 280,000 hospitalizations and 52,000 deaths. Montana has an elevated injury and death rate from TBI compared to the nation. The Emergency Medical Services and Trauma Systems Section (EMSTS), in partnership with Montana's EMS providers and trauma receiving centers is moving forward with participating in the EPIC protocol in attempt to improve the quality of care, and outcomes, that people suffering from TBI receive.

Excellence in Prehospital Injury Care (EPIC) for TBI is a treatment protocol and quality improvement initiative. In Arizona, where the program began, the protocol demonstrated a doubling of survival to discharge for patients with severe TBI and a tripling of survival for intubated patients with severe TBI. In children with severe TBI, the adjusted odds of survival are seven-fold. Similar results have been achieved by partner agencies across the nation.

STRATEGIES

- Educate a cadre of EPIC-MT Trainers who will provide training to EMS agencies and providers within their region/county.
- Recognize and incentivize agencies that participate in the EPIC-MT program.
- Support participating agencies by providing initial equipment inventory and ongoing training opportunities.
- Train Emergency Department staff in the EPIC-TBI Protocols.
- Encourage joint training/education with EMS agencies and receiving hospitals to ensure continuous adherence to the EPIC-TBI Protocols.

Student Pre-Test

1. The main goal of prehospital management of a TBI is:
   - o Keep blood sugar ≥ to 90
   - o Maintain SBP > 150
   - o Prevent secondary brain injury
   - o Provide positive pressure ventilation

2. At what rate should you ventilate an intubated adult with TBI?
   - o 8 BPM
   - o 10 BPM
   - o 12 BPM
   - o 14 BPM

3. In treating an adult TBI patient, the SaO2 should be maintained at or above:
   - o 80%
   - o 85%
   - o 90%
   - o 95%

4. What is the target EtCO2 for an intubated TBI patient?
   - o 20-30 mmHg
   - o 25-35 mmHg
   - o 30-40 mmHg
   - o 35-45 mmHg

5. When managing an adult TBI patient, the SBP should be maintained at or above:
   - o 60 mmHg
   - o 70 mmHg
   - o 80 mmHg
   - o 90 mmHg

6. When managing a 5 year-old TBI patient, the SBP should be maintained at or above:
   - o 60 mmHg
   - o 70 mmHg
   - o 80 mmHg
   - o 90 mmHg

7. Decreasing CO2 will cause which of the following (check all that apply):
   - o Cerebral artery vasoconstriction
   - o Increased secondary injury
   - o Decreased cerebral perfusion
   - o Increased cerebral perfusion

8. The effect of a SINGLE episode of hypoxia results in:
   - o Increased morbidity and mortality
   - o Bradycardia
   - o Hypotension
   - o No impact on neurological outcome

9. At what rate should an intubated 5 year-old with a TBI be ventilated? (check all that apply)
   - o 10 BPM
   - o 15 BPM
   - o 20 BPM
   - o To keep EtCO2 40 mmHg

10. If the EtCO2 in an intubated patient falls below 35, what is the most likely cause? required
    - o Hypoventilation
    - o Hypoxia
    - o Acidosis
    - o Hyperventilation

TBI Background & History

SIGNIFICANCE

Every year, over 1.4 million patients are evaluated in U.S. Emergency Departments (EDs) after 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 started as 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, which has since been adopted by EMS agencies across the nation. Over 5 years, the EPIC Project Team worked with EMS agencies to implement and evaluate TBI care and outcomes. This happened through the linkage of prehospital and trauma registry data, to fully document the impact of implementing the nationally vetted TBI Guidelines in moderate and severe TBI patients. EPIC implemented the TBI Guidelines in EMS systems that respond to 911 calls across the urban, suburban, rural, and wilderness areas. Interventions in the “EPIC Protocol” include optimizing the management of hemodynamics, oxygenation, and ventilation in the field for major TBI victims, with special emphasis on patients who received positive pressure ventilation. The primary goal was to help EMS systems save as many lives as possible from TBI and improve the quality of those lives saved.

TOM BRIDGE photo, Independent Record


MICHAEL GALLACHER photo, Missoulian

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 patient outcomes are 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 (CO2) 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.


photo, MSU Billings

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 02 saturation <90% is

independently associated with a doubling of mortality.[4]

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

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.

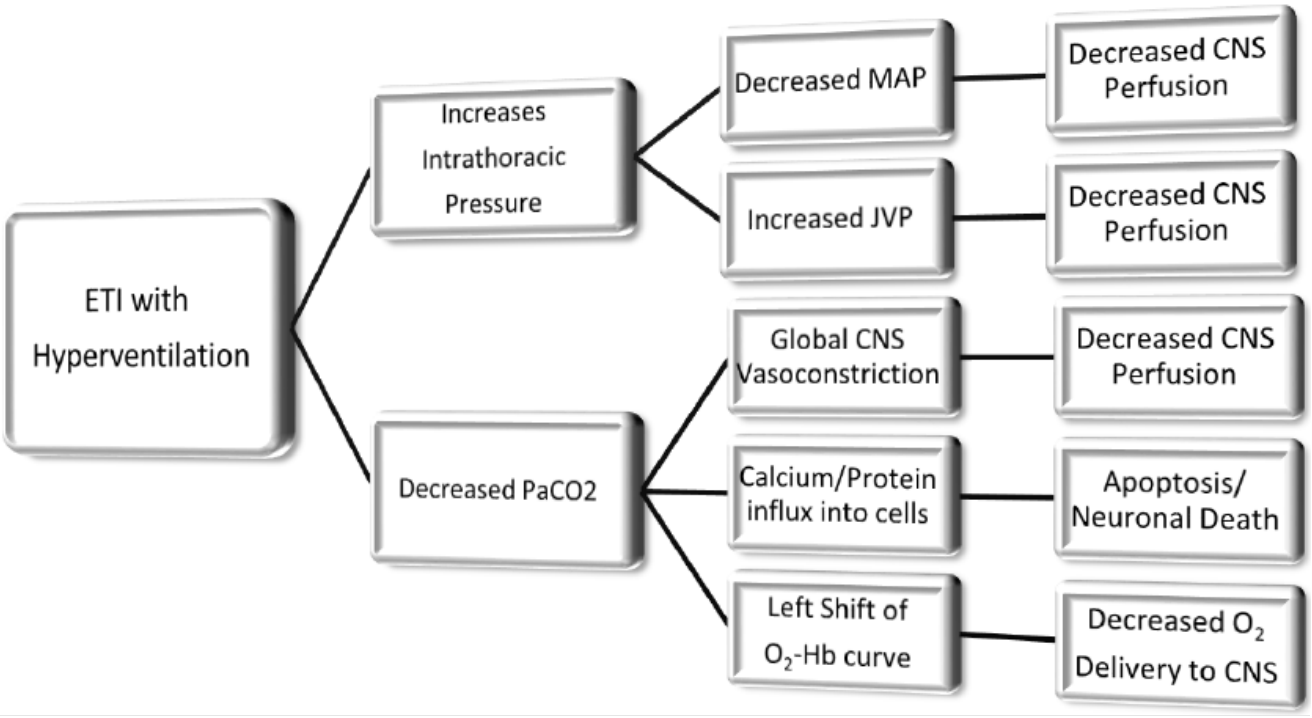
Table 1. Pathophysiology of Secondary CNS Injury during Hyperventilation

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
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
JVP - Jugular Venous Pressure
MAP - Mean Arterial Pressure
O2 - Oxygen
PaCO2 - Arterial Partial Pressure of Carbon Dioxide

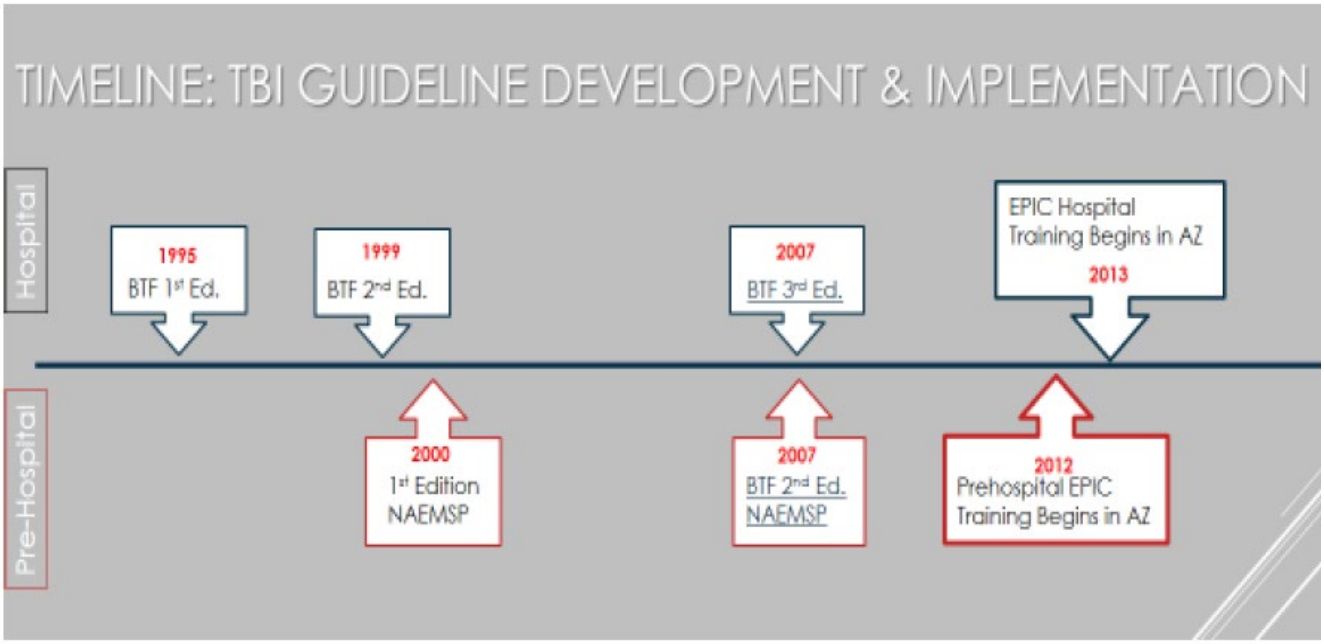
Hyperventilation (and the resulting decrease in the $PaC0_2$) causes the blood vessels supplying the brain to constrict. This results in a decrease in cerebral blood flow and is a powerful factor leading to secondary brain injury. At the cellular/neuronal level, low $C0_2$ can initiate a chain of reactions 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 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]

Figure 2. Timeline for TBI Guideline Development & Implementation



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, hypotension, 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:
1. 2. Hypoxia: Prevention, immediate recognition, and urgent treatment of Hypoxia
3. Hypotension: Prevention, rapid identification, and aggressive treatment of Hypotension
4. Hyperventilation: Strict avoidance and immediate correction of Hyperventilation/low ETC02
5. Hypoglycemia: Assess early and correct

EPIC Guidelines & Algorithm for Adults

DEFINITIONS

- Adults: Age ≥ 18 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:
    - Any injured patient with loss of consciousness, especially those with GCS < 15 or 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**

OVERALL APPROACH TO MONITORING AND CONTINUOUS EVALUATION

1. Continuous O_2 saturation (sat) via pulse oximetry,
2. Continuous quantitative end-tidal CO_2 (ETCO2) monitoring in intubated patients, and
3. Systolic blood pressure (SBP) every 3-5 minutes.

SPECIFIC, GUIDELINE-BASED THERAPY

I. Management of airway/oxygenation:

CLINICAL AXIOM: A single non-spurious O2 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 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).

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 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.

CLINICAL AXIOM: 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!

COROLLARY: 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 (Flow-Controlled Bags (FCB), Ventilation Rate Timers (VRT), ETCO2, ventilators). FCBs/VRTs should be used immediately after intubation and until the patient can be placed on a mechanical ventilator even if this will only take 3-5 minutes (note: that's all the hyperventilation it takes to begin killing neurons).

A. 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 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, use FCB/VRT immediately for ventilation and ETCO2 levels will be strictly maintained between 35 and 45 mmHg when monitoring is available (target = 40).[1, 2,12-15]

- All agencies are strongly encouraged to use FCBs/VRTs. Agencies without ETCO2 monitors, maintain age-appropriate ventilatory rates and decrease the risk of inadvertent hyperventilation.[1, 2, 10-12, 16-24] Agencies with ETCO2 monitors should use FCBs/VRTs for the initial rate of manual ventilation and then gently modify the ventilation to obtain the target ETCO2 of 40 mmHg. Beware of the tendency to only use the ETCO2 monitor to verify tube placement and then to fail to carefully maintain ETCO2 in target range.

- Ventilators should 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. FCBs/VRTs should be used immediately after intubation and until the patient is placed on the ventilator even if this will only take several minutes.

  i. Target tidal volume (TV) will be 7cc/kg with rates adjusted to keep the ETCO2 within target range (35-45 mmHg).

  ii. Note: This is consistent with the TBI guidelines and 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. Impending cerebral herniation:
- The EPIC guidelines do not encourage even mild hyperventilation for "impending cerebral herniation" for the following reasons:

  i. There is no evidence that it improves outcome in any setting.

  ii. There is much evidence that even mild hyperventilation harms moderate and severe TBI patients.

  iii. The "practical application" of this "treatment" is that many patients who do not have actual impending herniation end up being hyperventilated since the real-world interpretation often ends up thinking… "The worse a TBI is, the faster you should ventilate." Thus, many patients who will be harmed by hyperventilation may end up with the misapplication of this "treatment."

D. Non-intubated patients: All relevant monitoring/treatment will be applied, including ETCO2 monitoring where available.

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 SBP <90mmHg.[1-5, 35-48]

CLINICAL AXIOM: A *single* episode of SBP <90 mmHg 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, do not hesitate to continue aggressive fluid resuscitation.

- Note: If rapid infusion of initial crystalloid bolus does not correct the hypotension, continue aggressive fluid resuscitation.

- Note: Do not wait for the patient to become hypotensive. If the SBP is dropping, or if there are any other signs of compensated shock such as increasing heart rate with decreasing SBP, begin aggressive treatment *before* the patient becomes hypotensive.

- 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, 2, 49] However, IV fluids will be restricted to a minimal "keep open" rate in patients with SBP ≥140 mmHg.

IV. 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., diabetic on insulin who misses a meal → leads to low blood sugar → leads to decreased LOC → leads to motor vehicle crash in a hypoglycemic driver).

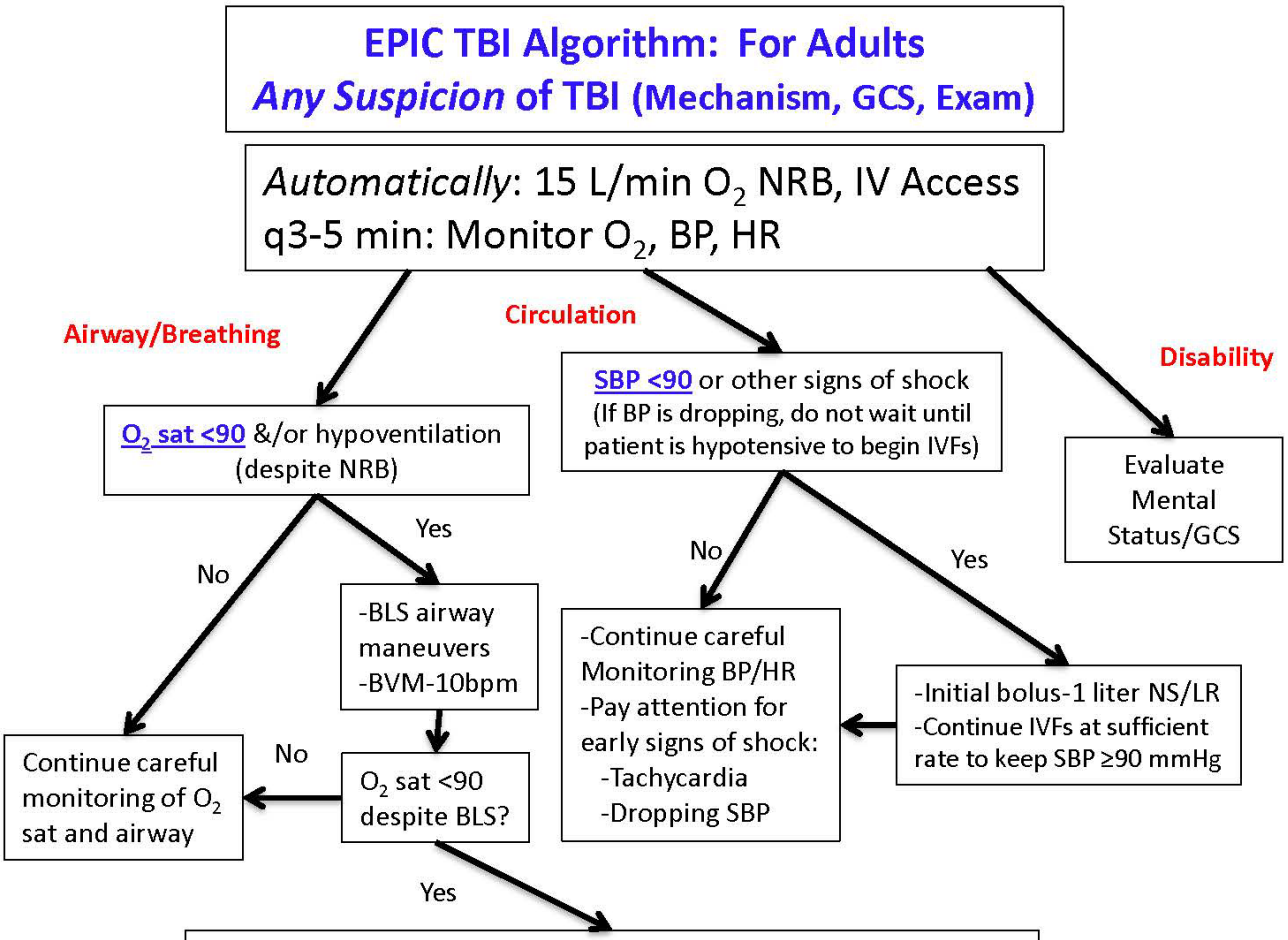
A. Assess blood glucose: Obtain fingerstick or serum glucose level. If glucose level is <70mg/dl, then administer dextrose according to local protocol or medical direction.

B. Reassess blood glucose: Repeat fingerstick in 10 minutes and, if still <70mg/dl, repeat dose x 1.

- If no response, contact medical control for further direction.
- If IV access unsuccessful, dextrose may be given IO.
- If IV and IO unsuccessful, give Glucagon 0.03mg/kg IM, max dose 1mg.

-- EPIC Guidelines & Algorithm for Adults References start on page 32 --

EPIC ALGORITHM FOR ADULTS



EPIC4Kids Guidelines & Algorithm 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/interpret), decreased level of consciousness, decreased responsiveness, or any deterioration of mental status.

OVERALL APPROACH TO MONITORING AND CONTINUOUS EVALUATION

1. Continuous O_2 saturation (sat) via pulse oximetry,
2. Continuous quantitative end-tidal CO_2 (ETCO2) monitoring in intubated patients, and
3. Systolic blood pressure (SBP) every 3-5 minutes.

SPECIFIC, GUIDELINE-BASED THERAPY

I. Management of airway/oxygenation:

CLINICAL AXIOM: A single non-spurious O2 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 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.

CLINICAL AXIOM: 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!

COROLLARY: 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 (Flow-Controlled Bags (FCB), Ventilation Rate Timers (VRT), ETCO2, ventilators). FCBs/VRTs should be used immediately after intubation and until the patient can be placed on a mechanical ventilator even if this will only take 3-5 minutes (note: that's all the hyperventilation it takes to begin killing neurons).

A. 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 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, use FCB/VRT immediately for ventilation and ETCO2 levels will be strictly maintained between 35 and 45 mmHg when monitoring is available (target = 40).[1-3,15-17]

- All agencies are strongly encouraged to use FCBs/VRTs. Agencies without ETCO2 monitors, maintain age-appropriate ventilatory rates and decrease the risk of inadvertent hyperventilation.[1-3,11,14,15,18-26] Agencies with ETCO2 monitors should use FCBs/VRTs for the initial rate of manual ventilation and then gently modify the ventilation to obtain the target ETCO2 of 40 mmHg. Beware of the tendency to only use the ETCO2 monitor to verify tube placement and then to fail to carefully maintain ETCO2 in target range.

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)

- 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. FCBs/VRTs should be used immediately after intubation and until the patient is placed on the ventilator even if this will only take several minutes.

i. Target tidal volume (TV) will be 7cc/kg with rates adjusted to keep the ETCO2 within target range (35-45 mmHg).

ii. Note: This is consistent with the TBI guidelines and 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. Impending cerebral herniation: The EPIC guidelines do not encourage even mild hyperventilation for "impending cerebral herniation" for the following reasons:

- There is no evidence that it improves outcome in any setting.
- There is much evidence that even mild hyperventilation harms moderate and severe TBI patients.
- The "practical application" of this "treatment" is that many patients who do not have actual impending herniation end up being hyperventilated since the real-world interpretation often ends up thinking… "The worse a TBI is, the faster you should ventilate." Thus, many patients who will be harmed by hyperventilation may end up with the misapplication of this "treatment."

D. Non-intubated patients: All relevant monitoring/treatment will be applied, including ETCO2 monitoring where available.

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.

CLINICAL AXIOM: 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:

SBP Target Thresholds:[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 (by age) will initiate intravenous (IV) fluid resuscitation. For hypotension or other signs of shock, give IV normal saline. 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 a sufficient rate to keep the patient non-hypotensive.

i. Note: If rapid infusion of initial crystalloid bolus does not correct the hypotension, continue aggressive fluid resuscitation.

ii. Note: Do not wait for the patient to become hypotensive. If the SBP is dropping, or if there are any other signs of compensated shock such as increasing heart rate with decreasing SBP, begin aggressive treatment *before* the patient becomes hypotensive.

iii. 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. 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.

A. Assess blood glucose: Obtain fingerstick or serum glucose level. If glucose level is <70mg/dl, then provide dextrose according to local protocol or medical direction.

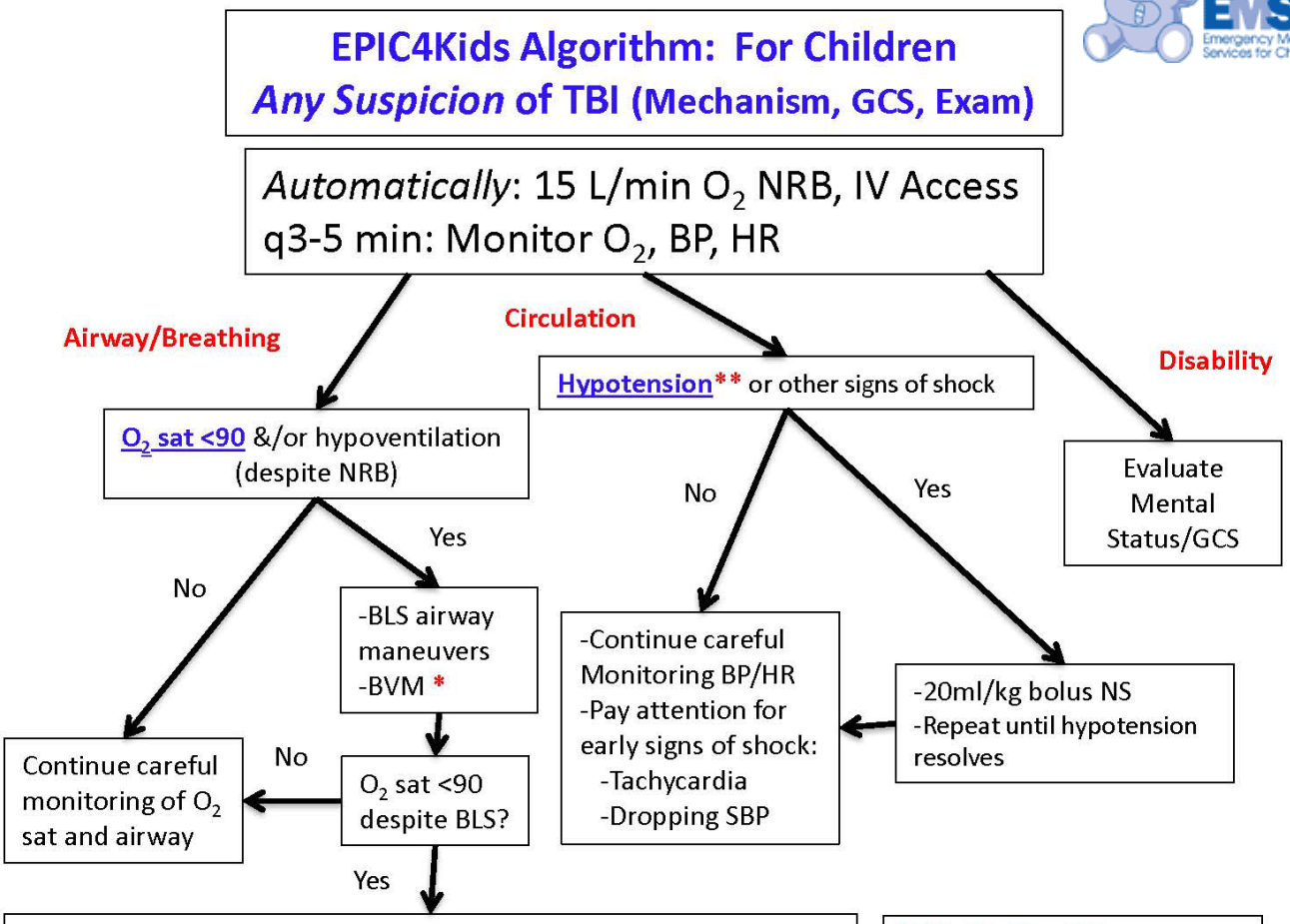
B. Reassess blood glucose: Repeat fingerstick in 10 minutes and, if still <70mg/dl, repeat dose x 1.

- If no response, contact medical control for further direction.
- If IV access unsuccessful, dextrose may be given IO.
- If IV and IO unsuccessful, give Glucagon 0.03mg/kg IM, max dose 1mg.

Note: Consult with your medical director if there are differences between the guidelines outlined above and your regional/agency protocols/standing orders for treating hypoglycemia in the setting of TBI.

-- EPIC4Kids Guidelines & Algorithm References start on page 34 --

EPIC4KIDS ALGORITHM



Consider ALS airway if experienced provider available:
- -Place advanced airway:
  - • Pre-oxygenate: BVM with 100% O_2 @ age-appropriate rate *
  - • Check placement using $ETCO_2$ monitor/detector
- -*AVOID* even *MILD* hyperventilation with *Ventilation Rate Timer* and *Pressure-controlled Bag*:
  - • Carefully keep rate @ age-appropriate rate *
  - • **ETCO2 available: Target $ETCO_2$ 40 mmHg (range: 35-45)**
  - •Control Ventilatory volume:
    - •Ventilator available: utilize *as soon as possible*
      - •Tidal volume = 7 cc/kg
    - • Ventilator *not* available: Continue Pressure-controlled BVM
- -Monitor: O_2 sat and airway *every 3-5 minutes*:
  - • If O_2 sat <90% despite above interventions, consider:
    - • Tension pneumothorax & needle thoracostomy

NOTE: NO ONE (not even Respiratory Therapists) can manually ventilate at the proper rate without ventilatory adjuncts (EVERYONE inadvertently hyperventilates unless meticulously preventing it):
- • Ventilation Rate timers
- • Pressure-controlled bags
- • $ETCO_2$ monitoring with someone watching the level continuously
- • Mechanical ventilator with careful $ETCO_2$ monitoring

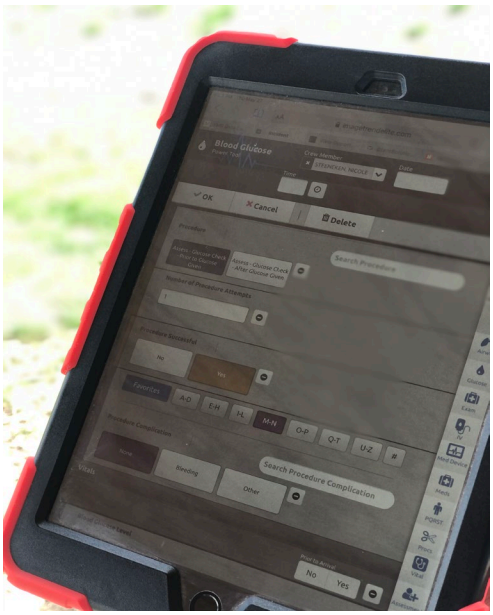
Ventilation Rates:
-Infants: (0-24 mo.):
-25 breaths/min (bpm)
-Children: (2-14 yrs):
-20 bpm
-Adolescents: (15-17 yrs):
-10 bpm (same as adults)

****Identifying Hypotension in children***
0-9 yrs: 70 + (age x 2)
≥10 yrs: <90mmHg
-*Rules of thumb*:
Newborn: <70mmHg
5 year old: <80mmHg
10 and older: <90mmHg →
(Same as adults)

Documentation & Quality Improvement Guide

EMS DOCUMENTATION

In addition to all standard requirements, documentation necessary for optimal recognition of your care in TBI includes the following:



- ✓ **Glasgow Coma Score (GCS)** – at least 2 (initial and during transport)
- ✓ **Blood pressure** - readings every 5 minutes*
- ✓ **SPO2** - readings every 5 minutes (as available)*
- ✓ **Capillary blood glucose** - initial (as available)
- ✓ **ETCO2** - readings every 5 minutes (as available)*

*Whenever possible, providers are encouraged to upload and attach/import their cardiac monitor case file to record ongoing blood pressure, SPO2, and ETCO2 readings.

Note: In order to be EPIC-MT Certified, an EMS agency must submit electronic prehospital care data to EMSTS through utilization of Montana's ImageTrend Elite EMS ePCR or export of ePCR data to the EMSTS data registry.

SYSTEM-WIDE QUALITY IMPROVEMENT

EPIC-MT Certified Agencies are encouraged to participate in system-wide continuous quality improvement (QI). Providers utilizing Montana's ImageTrend Elite ePCR can find a documentation guide to improve data capture (https://dphhs.mt.gov/assets/publichealth/EMSTS/EMS/TSII-TBI.pdf). The guide will also assist agencies not using ImageTrend although the format will be different. Agencies may access their agency's real-time data from Biospatial. Quarterly data is posted to the EMS Data Dashboard (https://dphhs.mt.gov/publichealth/EMSTS/MontanaEMSQIReport).

Note: System-wide quality improvement data is in aggregate form and does not include individual provider identifiers. This data is used to measure state-wide success with implementation and the impact on patient outcomes when using these guidelines.

PERFORMANCE/PROCESS INDICATORS

To assess prehospital utilization of the EPIC guidelines the following performance and process indicators have been selected:

1. Vitals are Documented and Trended
    a. Glasgow Coma Scale
    b. Heart Rate
    c. Respiratory Rate
    d. Systolic Blood Pressure
    e. Pulse Oximetry
    f. Blood Glucose Level

2. Oxygen Administration: High-flow oxygen is administered within 1 minute of arrival at patient.
    a. Oxygen saturation is maintained at or above 90%.
    b. Documented in Medications as well as Procedures

3. Positive Pressure Ventilation: In patients receiving positive pressure ventilation, hyperventilation (ETCO2 <35mmHg) is avoided.
    a. If SGA/ETI airway is placed, hypoxia (SPO2 <90%) does not occur during the intubation period.

4. Fluid Resuscitation: SBP is maintained at or above 90mmHg.
    a. In patients where SBP is below 90mmHg, IV/IO was placed within 3 minutes of arrival at patient and aggressive fluid resuscitation given.

5. Blood Glucose: Blood glucose value is documented.
    a. Dextrose is administered for findings less than 70mg/dl.

For EPIC-MT Certified agencies, the performance/process indicators' performance threshold goal is 80% (minimum).

Student Post-Test & Course Evaluation

POST-TEST LINK

After completing the EPIC-MT training, please visit https://www.cognitoforms.com/DPHHS1/EPICMTPostTest to take the EPIC-MT post-test and complete the course/instructor evaluation. Please complete the post-test prior to leaving class.

A passing score for the post-test is 8/10.
You may retake the post-test as many times as needed.

Upon successful completion of the post-test, you will receive a confirmation email with the following:

- ✓ EPIC-MT course completion and continuing education certificate
- ✓ EPIC-MT Agency Certification Application link

STUDENT LINK

SCAN ME

Note for Train-the-Trainer students: Your confirmation email will also include a link to the instructor registration form. Please complete your instructor registration prior to leaving class.

EPIC-MT Agency Certification

To be an EPIC-MT Certified agency, an EMS agency is making a commitment to meet minimum training standards, maintain necessary equipment, ensure adherence to the treatment protocol, and participate in submission of traumatic brain injury (TBI) patient care and outcome related data to the EMSTS Section. These factors are imperative in the success of the EPIC-MT program and improved outcomes of TBI patients across the state.

REQUIREMENTS

1. Agency agrees to train at least 80% of their EMS/hospital providers to the EPIC-MT Guidelines and provide annual (at minimum) refresher trainings/drills.
2. Front line vehicles will be equipped with at least one adult pressure-controlled bag-valve mask with ventilation rate timing light (i.e., SmartBag).
    a. Ideally, there should also be a pediatric pressure-controlled bag-valve mask and a timer available.
    b. A front-line EMS vehicle is a vehicle that is fully ready to respond to an EMS call without moving equipment from another vehicle. This generally excludes command vehicles, utility trucks, and other support vehicles.
3. Agency actively uses the State ePCR system (ImageTrend) or submits EMS data to EMSTS.

BENEFITS

- ✯ Recognition as an EPIC-MT Certified Agency.
- ✯ Inclusion in EPIC-MT related patient outcome and continuous quality improvement reporting.

EPIC-MT AGENCY CERTIFICATION

To complete the application, please visit
https://www.cognitoforms.com/DPHHS1/EPICMTAgencyCertification

The application includes the following:

- ✓ Chief or EMS Director commitment to meeting the program requirements

With the update in EMS Guidelines (11/1/2024), Medical Director approval is no longer required to implement EPIC-TBI Protocols.

AGENCY LINK

SCAN ME

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