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

ADULTS

--Definitions:
--Adults: 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:
--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

--Overall approach to monitoring and continuous evaluation:
Continuous O\textsubscript{2} saturation (sat) via pulse oximetry, continuous quantitative end-tidal CO\textsubscript{2} (ETCO\textsubscript{2}) monitoring in intubated patients, and systolic blood pressure (SBP) every 3-5 minutes.

--Specific, guideline-based therapy:
I. Management of airway/oxygenation:
--CLINICAL AXIOM: A single non-spurious O\textsubscript{2} sat of <90% is independently associated with a doubling of mortality. Hypoxia kills neurons!
A. Management is initiated by continuous high-flow O\textsubscript{2} for all potential TBI cases. Emphasis is placed on prevention, identification, and treatment of hypoxia (O\textsubscript{2} sat <90% and/or cyanosis).\textsuperscript{1-6} If high-flow O\textsubscript{2} fails to correct hypoxia, basic maneuvers for airway repositioning will be attempted, followed by reevaluation. If this does not restore O\textsubscript{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.\textsuperscript{1,2,5,7-10} Following ETI, tube placement will be confirmed via multiple means including ETCO\textsubscript{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.
--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 dying 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 (Pressure-Controlled Bags-PCBs, Ventilation Rate Timers (VRTs), ETCO\textsubscript{2} monitoring, ventilators). PCBs/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, shallow or irregular respirations, or periods of apnea): If there is evidence of hypoventilation despite high-flow O\textsubscript{2} therapy, assisted ventilation will be performed via BVM (PCB/VRT) and, if ineffective, ETI will be performed if an experienced ALS provider is present.\textsuperscript{1,2,11,12}

B. Intubated patients: After ETI, PCB/VRT is used immediately for ventilation and ETCO\textsubscript{2} levels will be strictly maintained between 35 and 45 mmHg when monitoring is available (target = 40).\textsuperscript{1,2,12-15}
1. All agencies are strongly encouraged to use PCBs/VRTs. In agencies without ETCO₂ monitors, maintain a respiratory rate of 10 breaths per minute to prevent inadvertent hyperventilation. Agencies with ETCO₂ monitors should use PCBs/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.

2. Ventilators will be used post-intubation whenever available to optimize ventilatory mechanics and O₂ therapy. This is the best way to care for an intubated TBI patient. PCBs/VRTs should be used immediately after intubation and until the patient is placed on the ventilator even if this will only take several minutes. Target tidal volume (TV) will be 7cc/kg with vent rates adjusted to keep the ETCO₂ 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.

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 many end up with the misapplication of this “treatment.”

D. Non-intubated patients: All relevant monitoring/treatment will be applied except ETCO₂ 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.

--CLINICAL AXIOM: 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 dying 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. 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.
--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. However, IV fluids should be restricted to a minimal “keep open” rate in patients with SBP ≥140 mmHg.
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 → 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 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/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.
References

44. Seelig JM, Klauber MR, Toole BM, Marshall LF, Bowers SA. Increased ICP and systemic hypotension during the first 72 hours following severe head injury. In: Miller JD, Teasdale GM, Rowan JO, eds. Intracranial Pressure VI. Berlin: Springer-Verlag; 1986:675-9