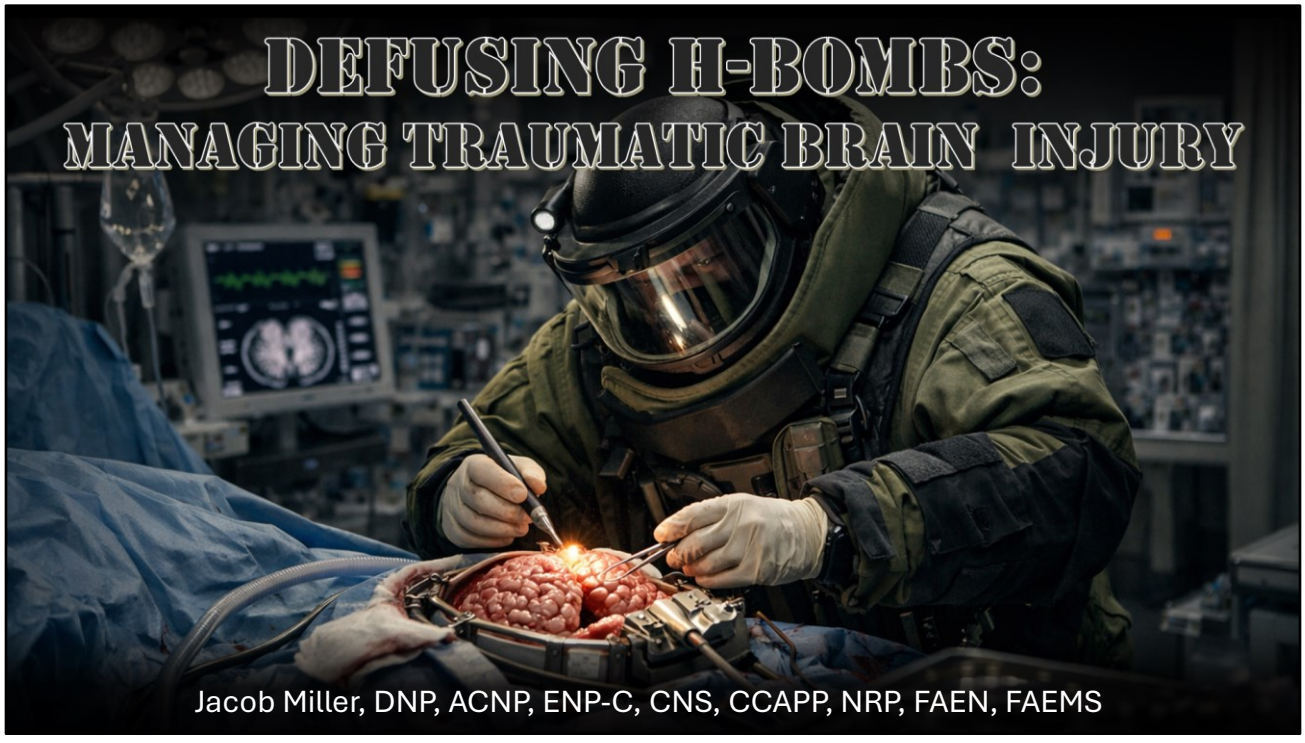


Standard disclaimers:

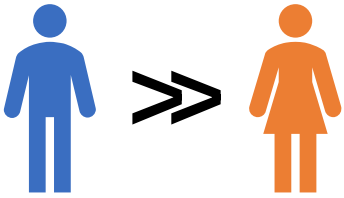
- Views/opinions are entirely my own and may not be reflective of my employers or others.
- Any specific brands or products mentioned are illustrative and/or for clarity and ***not*** intended as product endorsement.
- This presentation is NOT a substitute for medical judgment or common sense. Seek expert consultation as indicated.
- **I am neither a neurosurgeon nor bomb technician*

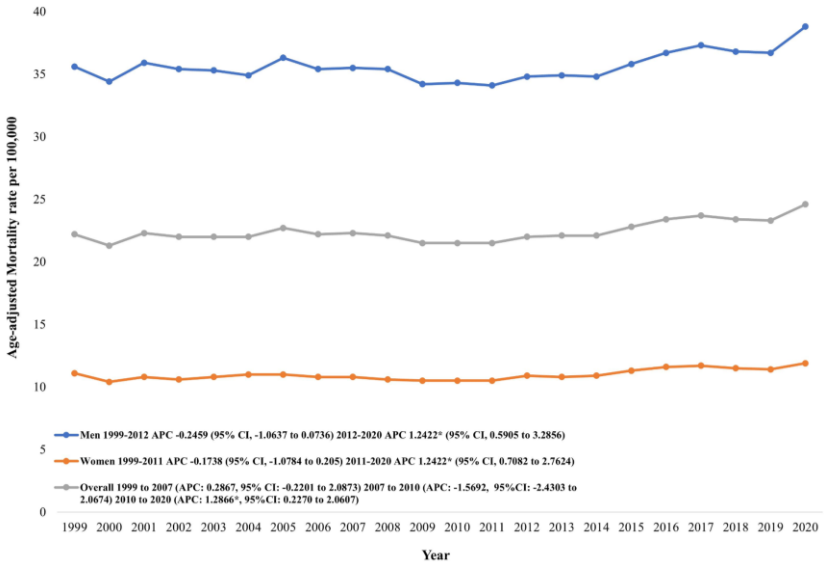
Epidemiology

Overall incidence of TBI is estimated at 64-74 million cases per year globally, with approximately 1.7 million annual cases of TBI in the United States.
The overall prevalence of TBI is estimated at 18.2% - meaning nearly 1 in 5 people in the United States has had a TBI at some point in their life.

Epidemiology

Mortality
by gender

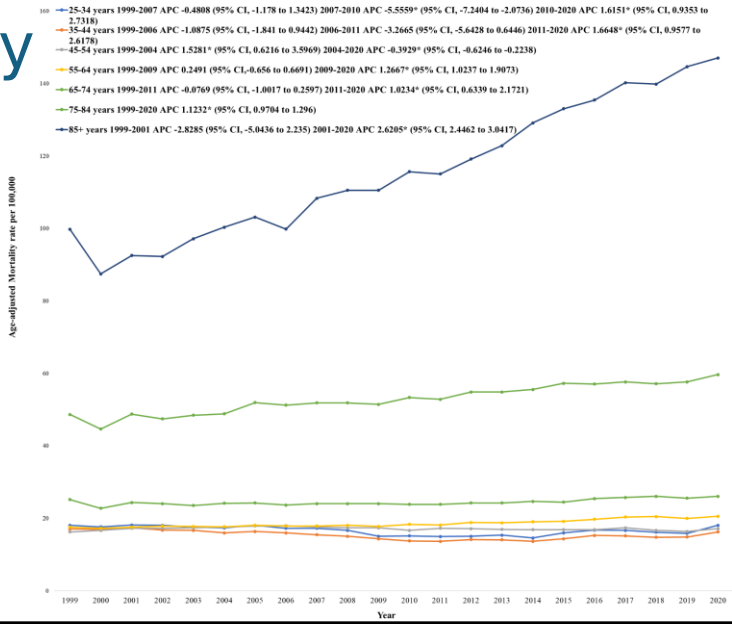




BMC Public Health 2025;25:482

The age-adjusted mortality in the United States from TBI had been relatively flat since the turn of the century, but in the last few years has had a sudden and statistically significant increase; this has caused the overall trend line to show a slight increase in mortality over time. Males are consistently and considerably more likely to suffer a fatal TBI than females.

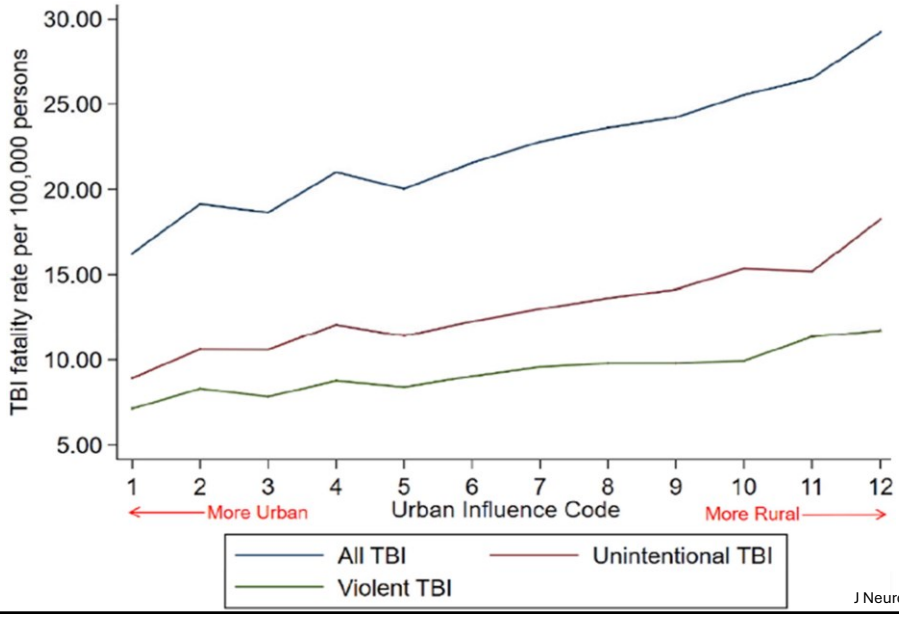
Mortality
by age



BMC Public Health 2025;25:482

The bulk of the rising mortality tends to be in patients aged 75 or greater. While the mortality rate for those 65-75 is slightly higher than younger counterparts, the mortality rates for those 75-84 and those 85 and over are markedly higher and consistently increasing over time.

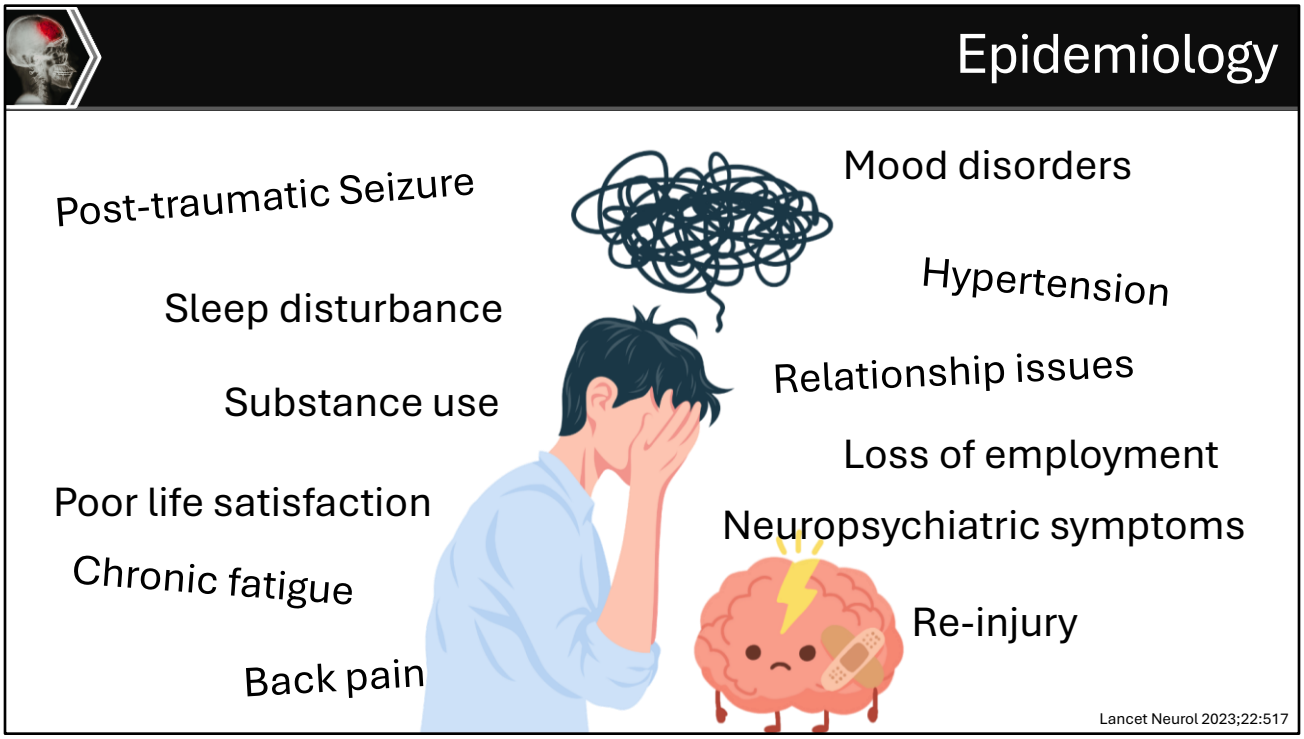
Epidemiology



J Neurosci Rural Pract 2019;10:10

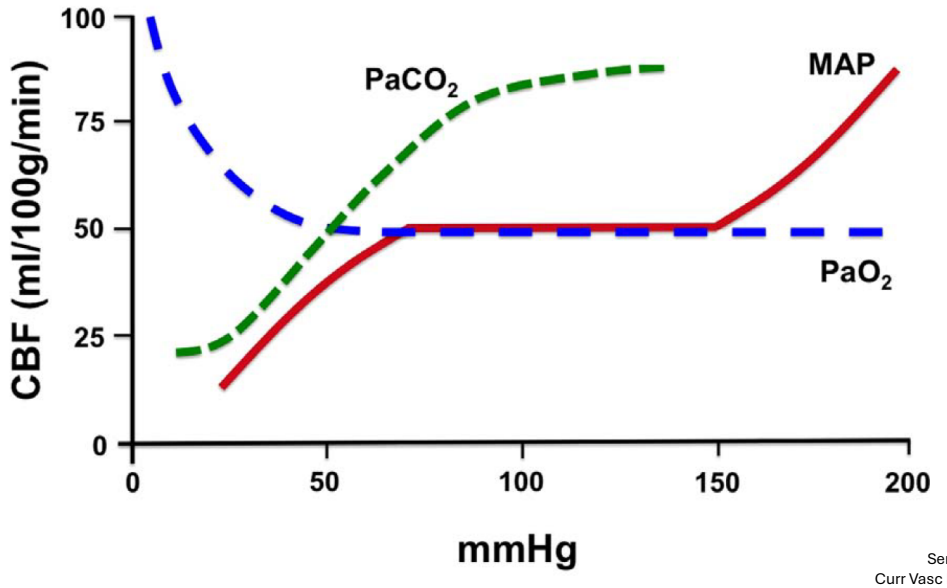
In addition to older Americans, geographical location affects mortality rates, with death becoming increasingly likely with more rural location of injury.

Epidemiology



And those who survive their TBI can be met with a high degree of morbidity across physical, psychological, and social domains.

Physiology & Pathophysiology



Semin Neurol 2016;36:560
Curr Vasc Pharmacol 2013;11:170

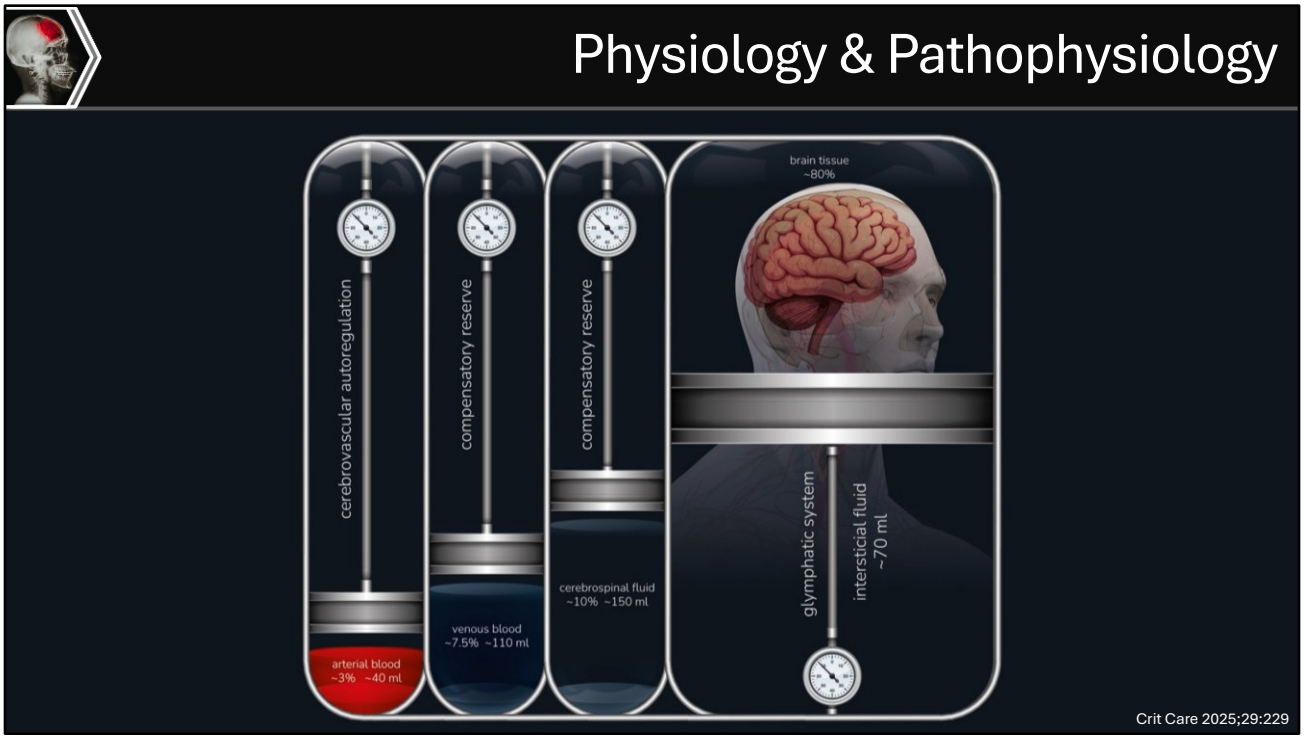
It's also important to understand cerebral autoregulation, especially in the context of Monroe-Kellie's hypothesis and the possible effects of a traumatic brain injury.

The flat part of the **RED** curve in the center indicates consistent "autoregulated" blood flow (blood flow is shown on the y-axis), where normally the intrinsic autoregulation mechanisms can maintain a consistent level of blood flow and perfusion across a wide variety of blood pressures (the x-axis of the red curve)

However, changes in oxygen and carbon dioxide tension can ALSO affect cerebral blood flow:

- The **BLUE** curve shows that cerebral blood flow increases as oxygen tension falls (below, in this graph, a PaO_2 of around 50 mmHg)
- Conversely, the **GREEN** curve shows the effect of carbon dioxide changes on cerebral blood flow: LOW $PaCO_2$ levels cause vasoconstriction and reduce blood flow, while HIGH $PaCO_2$ levels result in vasodilation and increased cerebral blood flow.

Physiology & Pathophysiology



The well-known Monro Kellie doctrine (and yes, I'm well aware that this is an imperfect model) holds that the intracranial pressure is a function of the three *primary* components within the calvarium:

- Brain tissue
- Blood (both arterial and venous)
- CSF

Because the cranium is essentially a fixed container, the total volume of these components has a direct effect on the overall pressure – raise the total intracranial volume (by any one or more components), and the total intracranial pressure (ICP) will also rise, unless there is a balancing measure (e.g., a decrease in a different component)

While not *typically* in the cranium, any other mass effect (whether tumor, hemorrhage, or otherwise) will also contribute to the total intracranial volume and, thus, ICP.

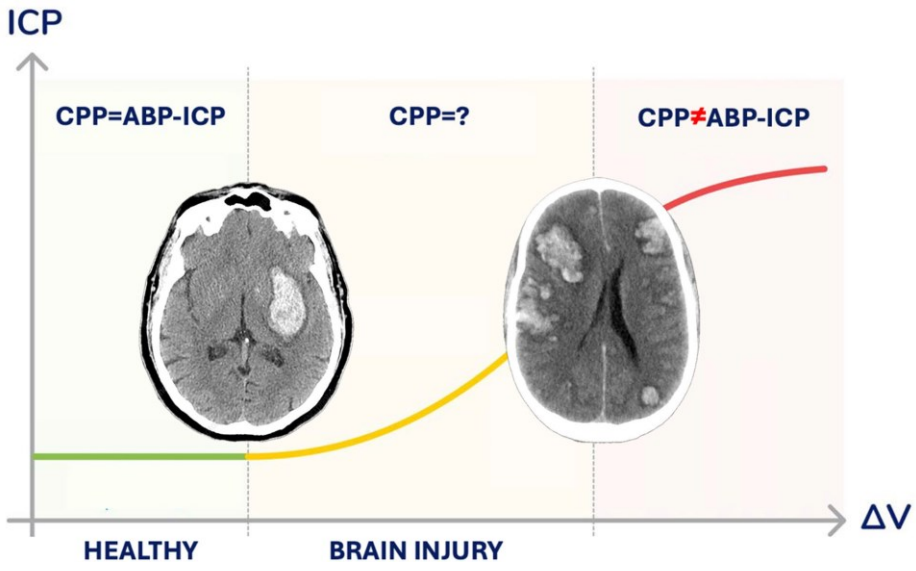
CPP ≟ MAP - ICP

Semin Neurol 2016;36:560
Curr Vasc Pharmacol 2013;11:170

We've all been taught that cerebral perfusion pressure (CPP) is the difference between the mean arterial pressure (MAP) and the intracranial pressure (ICP). For this reason, the brain autoregulates to achieve a relatively constant MAP when given a relatively constant ICP, and to a certain extent can "autoregulate" itself if ICP rises or falls slightly.

But this may be a gross oversimplification of a more complex process.

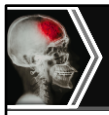
Physiology & Pathophysiology



Crit Care 2025;29:229

Newer evidence is suggesting that CPP is not as simple as “MAP minus ICP” – and that even when we know MAP and ICP, actual brain tissue perfusion might not be occurring as well as we predict it to from a calculated CPP. This seems to be especially true at extremely high ICP values.

Patient Assessment



Primary Trauma Survey

M ASSIVE HEMORRHAGE
A IRWAY
R ESPIRATORY
C IRCULATION
H EAD INJURY
E NVIRONMENT
D EXTROSE / DIFFERENTIALS

J Spec Oper Med 2022;21(1):11
National Model EMS Clinical Guidelines, v 3.0; 2022

The primary survey has classically been taught in an "ABC" approach, but this misses some key elements that have been shown to have a significant impact on morbidity and mortality.

For that reason, the military's Tactical Combat Casualty Care (TCCC), and the more recent National Model EMS Guidelines, teach the MARCH mnemonic for primary survey. I personally have expanded this to "MARCHED" focusing on the following:

- **M:** Identify and control any major/massive external hemorrhage. This may include use of tourniquet, wound packing, direct pressure, or other mechanisms available
- **A:** Maintain a patent airway. **RARELY** does this mean intubate; during the primary survey, the focus should be on clearing secretions, positioning, and placing simple adjuncts (OPA/NPA) to maintain airway patency. If RSI/intubation is required, that is best done AFTER completing the primary survey
- **R:** Ensure adequate respirations and respiratory support. This may include provision of oxygen, BVM ventilations, or decompression of actual/suspected tension pneumothorax. It's important to recognize that, in a spontaneously breathing patient, hypotension is a VERY LATE sign of tension pneumothorax (and may not occur until peri-arrest or imminent arrest) – the most common sign of TPTx in a spontaneously breathing patient is severe, progressive dyspnea!
- **C:** We'll get into hemodynamic goals later, but here, circulation should include ensuring adequate perfusion and early acquisition of baseline vital signs (this can be delegated during the primary survey, or obtained after the primary survey is completed)
- **H:** More than just "disability" assessment, you should be identifying patients with potential TBI in this survey, because that will likely change some management considerations. If head injury is suspected or confirmed, ensure steps are taken to appropriately manage the patient and prevent secondary brain injury (literally, the entire rest of this presentation)
- **E:** Aggressively protect the patient from hypothermia. Remove wet clothing, keep the patient covered, place a barrier between the patient and the ground (tons of heat is lost through conduction this way!), and crank the heat in the ambulance, aircraft, or trauma bay.

Hypothermia can independently more than double a trauma patient's risk of death. In excessively HOT environments, heat loss is still a concern, but also be mindful to avoid hyp**er**thermia.

- **D:** I put this in here because it's important to pause and (1) for ANY altered mental status, obtain a blood glucose to exclude an easily correctable cause of their AMS – especially before invasive procedures like intubation, and (2) consider that not all trauma is "trauma" – some patients may have a preceding medical even that leads to trauma, or may have had a purely medical event that is masked as trauma.

Nursing Practice
Review
Neurology

Keywords: Consciousness/Glasgow Coma Scale/Clinical assessment/ Standardisation
● **This article has been double-blind peer reviewed**

Forty years after its initial implementation, the Glasgow Coma Scale has been updated to address variations in technique that have developed over time

Forty years on: updating the Glasgow Coma Scale

Author Sir Graham Teasdale is emeritus professor of neurosurgery, Institute of

Nurs Times 2014;110:12

Because, historically, the GCS alone determined TBI severity, it's worth taking a few slides to review the CORRECT assessment of GCS.

This review article was authored by Prof. Teasdale in 2014 – one of ***the*** original co-authors of the original 1974 paper first describing GCS. I encourage you to look up this paper (it's only a few pages long), but I've summarized some of the key components that I think we should be aware of.

Forty years on: updating the Glasgow Coma Scale

Self-explanatory

5: Person, place, and time

4: ANY disorientation

3: "Lacks structured phrases"
Intelligible single words

2: "Sounds"

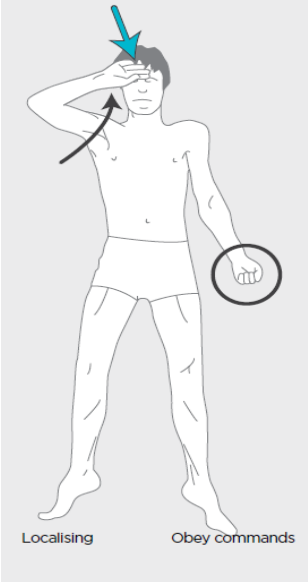
Nurs Times 2014;110:12

Eye response is fairly self explanatory, although Dr. Teasdale does note that eyes "to voice" (3) does not have to be a command to open eyes – this counts with any verbal stimulus.

For VERBAL response, Dr. Teasdale makes the following notes:

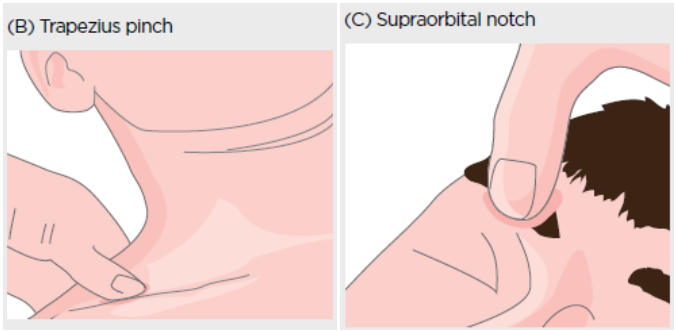
- A verbal score of "5" may only be applied if the patient is oriented to self, place, and time. Place does not have to be incredibly specific, but at least a correct general location should be provided (e.g., "hospital" is acceptable, does not have to be "ABC Medical Center" specifically). Orientation to event/circumstance is not mentioned as a requirement.
- A verbal score of "4" is applied if there is any disorientation to any of the above 3 features (person, place, time), or any other obvious incorrect orientation or confusion, even if the patient otherwise speaks with full and coherent sentences.
- A verbal score of "3" is applied when the patient speaks in either single words or a "lack of structure" in forming complete thoughts. The term "inappropriate words" is no longer considered adequate, since "inappropriate" can be fairly subjective. Thus, a patient who can respond to questions in full sentences with formed thought is likely going to score at least a "4"
- Lastly, a verbal score of "2" is provided when the patient makes only sounds that are not words. Similar to the previous score, the term "incomprehensible speech" is no longer used as some found the term "speech" to imply the patient must be attempting to speak or form words, but this is clarified to apply to patients making any "sounds" – e.g., grunting, moaning, *or* incomprehensible attempts at speech.

Forty years on: updating the Glasgow Coma Scale



6: Follows TWO-STEP command

5: Localize ABOVE clavicle

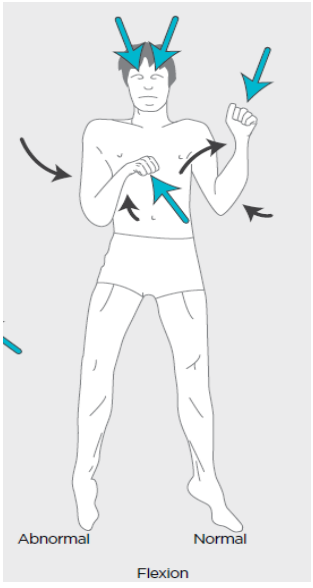


J Crit Care 2014;29:627
Nurs Times 2014;110:12

For the MOTOR component of the GCS, the following distinctions are noted:

- A motor score of “6” should only be applied when the patient clearly follows commands. If it’s even remotely unclear if the patient is following versus performing reflexive actions, a two-step command should be given, e.g., “make a fist and then open it”
- A motor score of “5” applies when the patient LOCALIZES a stimulus. Likewise, if this is unclear, the noxious stimulus should be applied above the level of the neck (i.e., either trapezius squeeze or supraorbital pressure). Here, Teasdale notes that localizing should involve bringing the hand “above the clavicle towards a stimulus on the head or neck”, further noting that “bringing a hand to the opposite side of the body is not sufficient”

Forty years on: updating the Glasgow Coma Scale



6: Follows TWO-STEP command

5: Localize ABOVE clavicle

4: Normal flexion

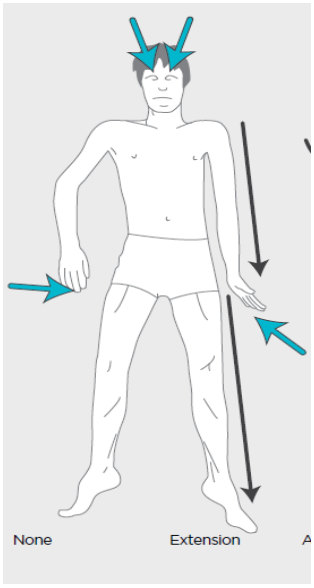
3: Abnormal flexion

Abnormal Flexion	Normal Flexion
● Slow	● Rapid
● Stereotyped (the same response each time)	● Variable (or varying)
● Arm moves across chest	● Arm moves away from body
● Forearm rotates, thumb clenched ● Leg extends	

J Crit Care 2014;29:627
Nurs Times 2014;110:12

- *Normal flexion* (no longer called "withdrawal") scores "4" – this is defined as a more rapid flexion that can be variable or varying (i.e., might be slightly different each time a noxious stimulus is applied), often with the arm moving away from the body. This is best tested by application of fingernail pressure, which should cause the arm to move away from the body and the stimulus.
- By contrast, *abnormal flexion* – a score of "3" – is a more slow and stereotyped (i.e., the same response is seen every time) flexion. The arm moves across the chest, the forearm rotates, and thumb is clenched. This is also generally associated with extension of the leg.

Forty years on: updating the Glasgow Coma Scale



6: Follows TWO-STEP command

5: Localize ABOVE clavicle

4: Normal flexion

3: Abnormal flexion

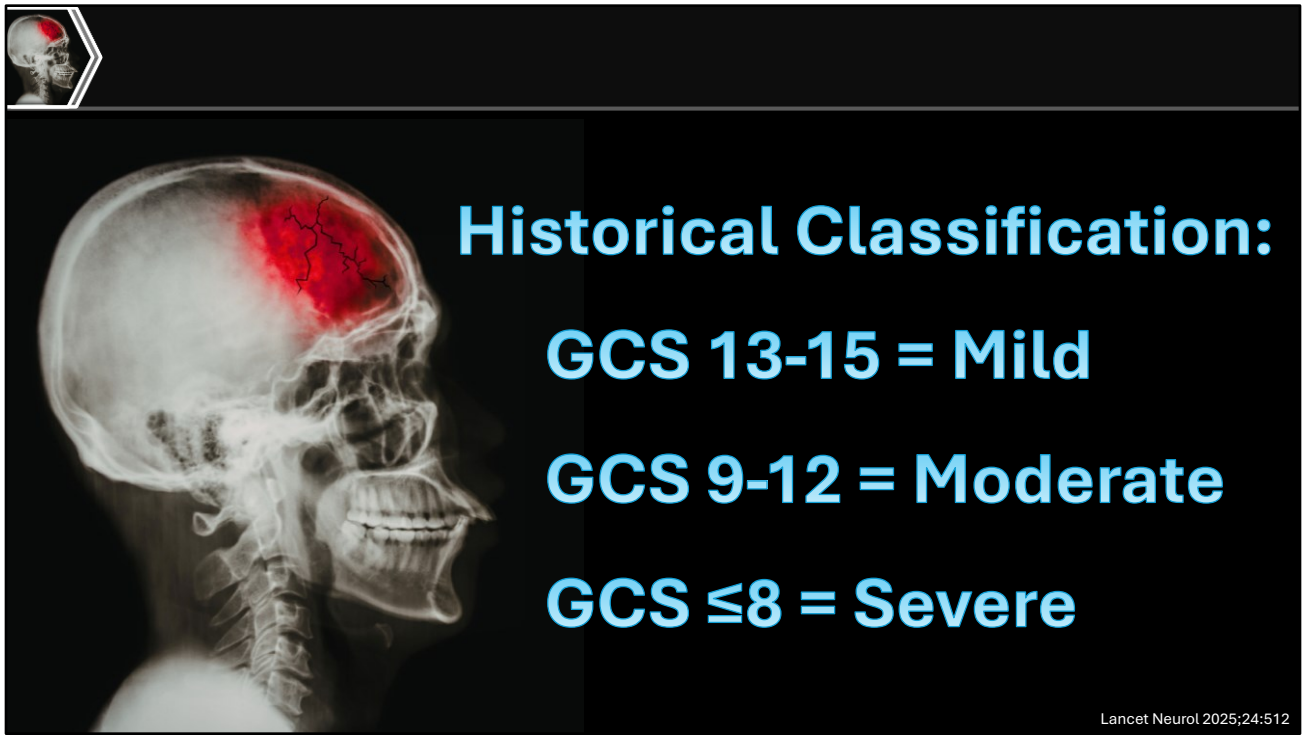
2: Extension

1: No response*

Spinal reflex ≠ "response"

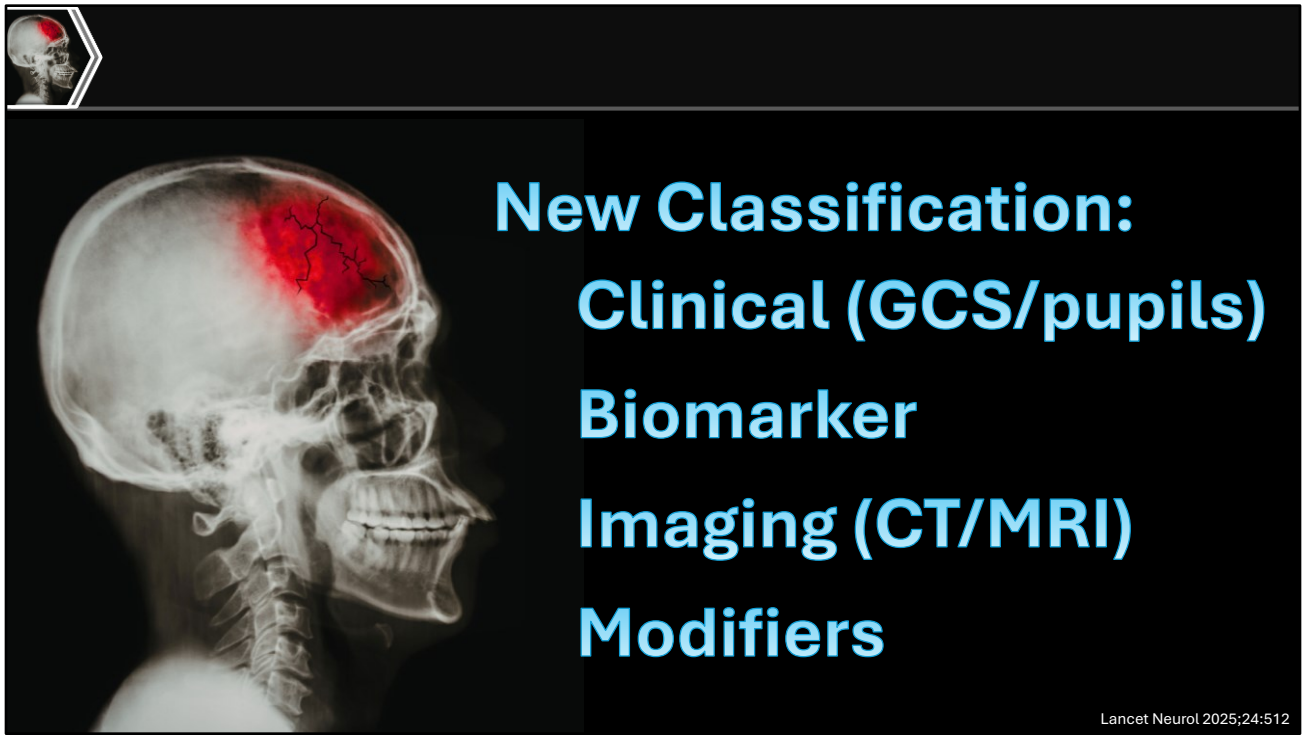
J Crit Care 2014;29:627
Nurs Times 2014;110:12

- Extension response is scored "2" and is characterized by a straightening (extension) of the elbow in addition to the lower extremities.
- Absence of **cerebral** motor activity is scored a "1" – thus, if a patient responds ONLY with spinal reflexes, this is still scored a "1" for no motor response. The most commonly seen spinal reflex may be "triple flexion" of the lower legs.
    - Triple flexion involves a **stereotyped** flexion of the hip, knee, and ankle (dorsiflexion, or upward) that subsides after a few (~6) seconds despite constant stimulus. This is a stereotyped spinal reflex, so appears the same regardless of where stimulus is applied (plantar aspect vs toes, etc).



As noted, the historical (and, likely, still widely-used) classification of TBI was divided into mild, moderate, or severe based on GCS score alone. While this helped classify patients into well-delineated "buckets", there was considerable heterogeneity in type and extent of injury, morbidity, and outcome data even within individual groupings.

In reality, some patients with "mild" TBI have considerable morbidity, and some patients with "severe" TBI are viewed as having a poor prognosis and can be met with nihilism and early withdrawal of treatment or rehabilitative efforts.



The new classification scheme for TBI, called the "CBI-M" classification, includes clinical, biomarker, and imaging results as well as various modifiers.

- Clinical exam findings include total GCS score (so, yes, this is still important!) and pupil reactivity, as well as expanded clinical markers like injury signs, cognitive function, and other assessment parameters.
- The biomarker pillar refers to various lab-based biomarkers that have been shown to correlate with degrees of tissue damage (similar to how we now regularly use troponin in evaluating a cardiac patient). These TBI biomarkers include glial fibrillary acidic protein (GFAP), ubiquitin C-terminal hydrolase L1 (UCH-L1), and S100 calcium-binding protein B (S100B), and research continues to evaluate new biomarkers for triage, treatment, and prognostic purposes.
- Imaging includes various aspects of neuroimaging results, including lesion size and location, presence of mass effect, presence of fracture, and so on.
- Lastly, modifiers include external factors that may affect the treatment or prognosis; these include injury-specific factors (e.g., mechanism of injury, presence of post-traumatic seizure, secondary or comorbid non-CNS injuries, etc.), patient-specific factors (e.g., age, sex, medical history, etc.), and other societal and community factors (e.g., access to healthcare, social support structure, etc.).

While this will help better classify and characterize specific details of TBI patients and inform future research, this removes the blunt (and easy) classification of mild/moderate/severe.

Patient Management

Photo: J. Miller

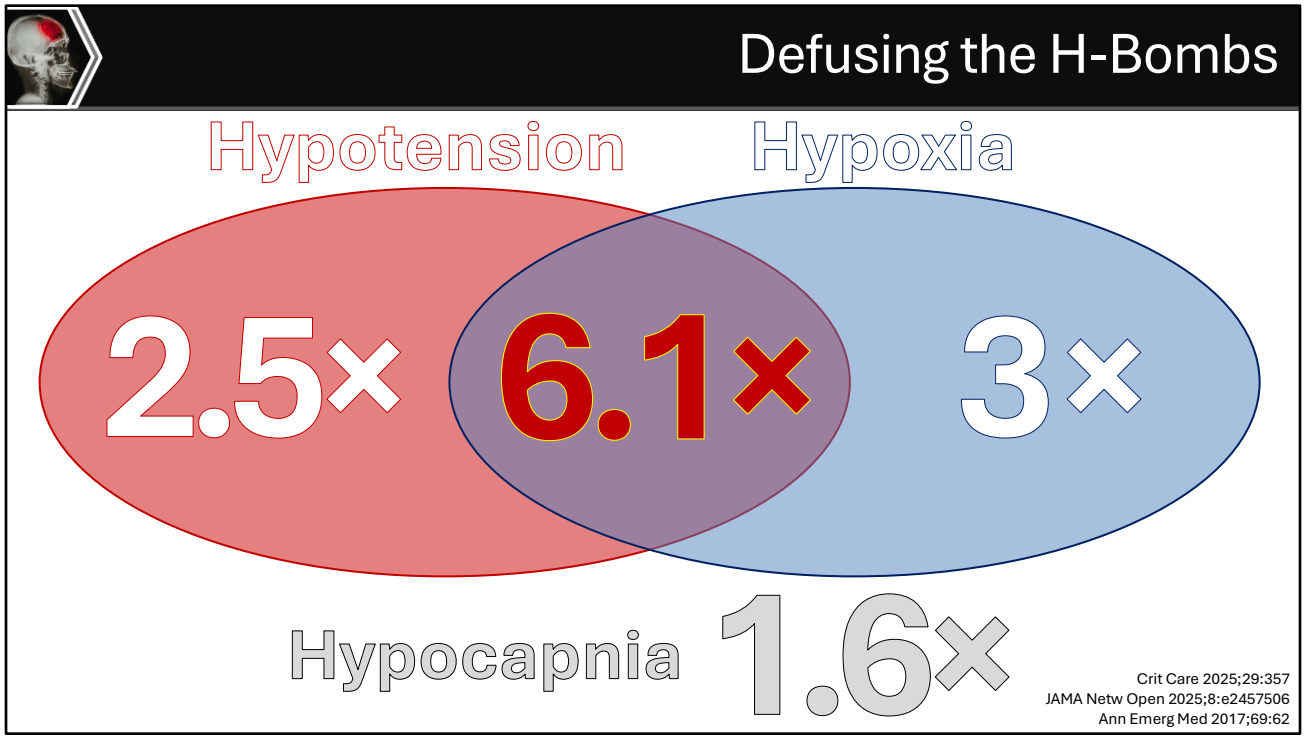
Defusing the H-Bombs

- Hypotension
- Hypoxia
- Hyperventilation

Crit Care 2025;29:357
JAMA Surg 2019;154:e191152
Ann Emerg Med 2017;69:62

The 3 major “H-bombs” of head injury that lead to the highest morbidity and mortality are hypotension, hypoxia, and hyperventilation.

Although not commonly discussed, a 4th “H-bomb”, hypoglycemia, should also be ruled-out for any patient with altered mental status.

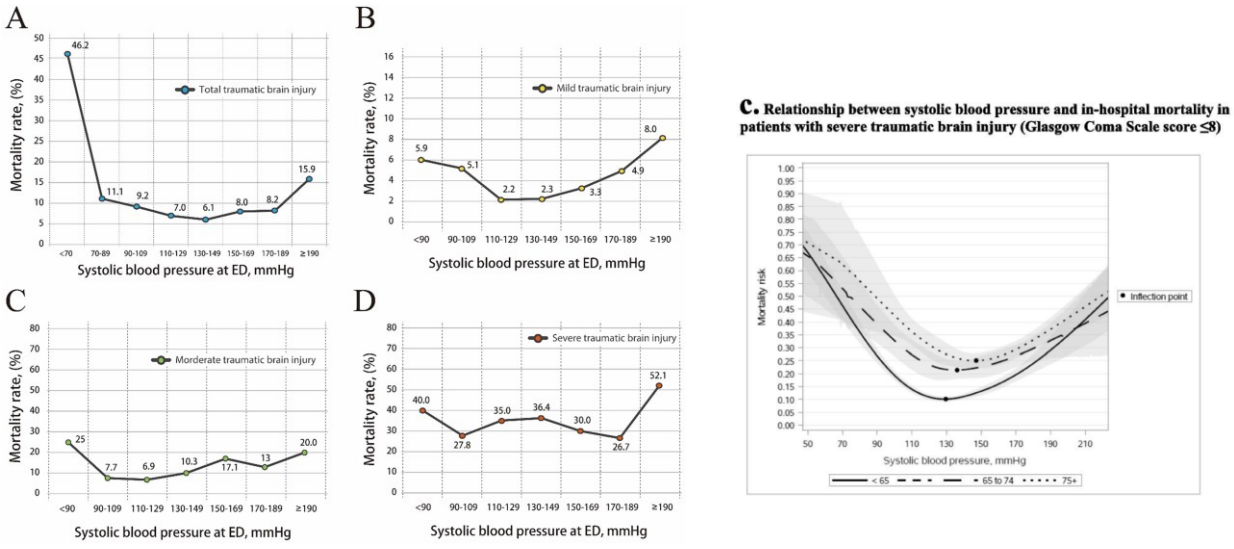


Data from the EPIC-TBI study:
Any single episode of prehospital hypotension (SBP <90) increased odds of death by 2.5
Any single episode of prehospital hypoxia (SpO2 <90) increased odds of death by 3
And any combination of the above (occurring at the same time or separate times) increased odds of death by 6-fold!

A 2025 systematic review added that hypocapnia (most commonly defined as $PaCO_2$ <30 mmHg) was associated with a 1.6-fold increase in odds of death, although one large retrospective study (15,000 patients; over 1,000 intubated) found this to be as high as an **8-fold** increase in risk of death with EtCO2 <35 in those intubated in the prehospital setting.

Note: EPIC-TBI patients were isolated TBI without hemorrhagic shock or polytrauma.

Defusing the H-Bombs



BMC Emerg Med 2023;23:105
Am J Emerg Med 2022;58:265

Across all levels of TBI severity, hypotension is consistently associated with significant increase in odds of death. Interestingly, mortality curves all seem to have a “U”-shaped appearance, with mortality also rising in patients presenting with hypERtension – similar to morbidity and mortality seen in intracranial hemorrhage with elevated blood pressures. This has been replicated in multiple studies across multiple sites and countries, and consistently finds similar patterns.

Importantly, however, while both hypotension and hypertension are *associated* with poor outcomes, it remains to be proven whether they are the *cause* of poor outcomes, or simply a marker of another factor.

Defusing the H-Bombs

Hemodynamic Targets

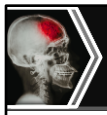
Age	Min SBP
Pediatric	≥ 75th %ile
15-49 yrs	≥ 110
50-69 yrs	≥ 100*
≥70 yrs	≥ 110

Neurosurgery 2017;80:6
Prehosp Emerg Care 2023;27:507
ENLS / Neurocritical Care Society; 2025:333

These are the recommended MINIMUM blood pressure targets for various populations in patients with **isolated** TBI or without multisystem trauma or hemorrhagic shock.

For pediatric population, the general recommendation is to maintain the systolic blood pressure above the 75th percentile for age.

The asterisk is a difference in management suggestions: The prehospital guidelines recommend all adults be maintained >110 mmHg systolic; the ENLS and Brain Trauma Foundation guidelines relax the SBP to >100 for patients aged 50-69 years.



Defusing the H-Bombs

Hemodynamic Targets

SBP ≥ 100

MAP ≥ 80

For <u>Uncontrollable</u> Hemorrhage:

Consider lower target for <u>shortest</u> possible time

World J Emerg Surg 2019;14:53

Hemodynamic targets in the *polytrauma* patient, especially those with hemorrhagic shock become more challenging.

On one hand, we know damage control resuscitation principles for *polytrauma* patients typically allow for *permissive HYPOtension*, and these patients can have worse outcomes if we elevate their blood pressure prior to definitive hemorrhage control.

On the other hand, we *also* know that traumatic brain injured patients do not tolerate hypotension, and that leads to worse morbidity and mortality.

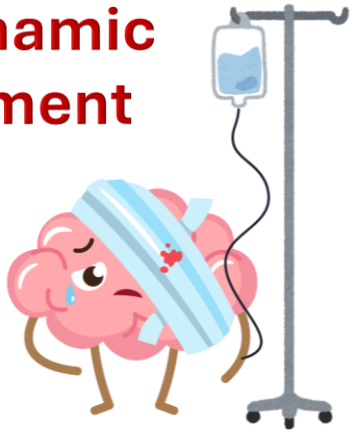
Essentially – the hemodynamic goals for managing these patients are to simultaneously employ two completely divergent therapies. Most current TBI guidelines do not address this patient population in any depth; they focus on the isolated TBI patient. The consensus recommendations from the World Society for Emergency Surgery are to target a systolic BP >100 or a MAP >80 (for the adult patient), but to *<u>consider</u>* a lower blood pressure for the ***<u>shortest possible</u>*** time if the patient has an <u>uncontrollable</u> hemorrhage, recognizing the lower blood pressure may increased the risk of morbidity and TBI-related mortality, but may be necessary to prevent mortality from hemorrhage.

Hemodynamic Management

Isolated TBI:

Normal Saline

Better outcomes vs balanced crystalloids



JAMA Surg 2019;154:e191152
J Neurotrauma 2022;39:1159
Am J Emerg Med 2026;101;84
Prehosp Emerg Care 2023;27:507
ENLS / Neurocritical Care Society; 2025:333

For isolated TBI patients, guidelines consistently recommend crystalloid resuscitation to prevent or treat hypotension. Several subgroup analyses and systematic reviews have demonstrated a benefit of normal saline over balanced crystalloids (e.g. Lactated Ringers, Normasol, or Plasma-Lyte A) in this specific patient population.

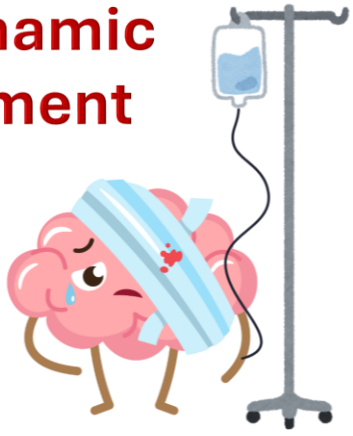
Hypertonic saline should **NOT** be used for routine fluid resuscitation in these patients, but will be discussed later in the context of ICP crises.

Polytrauma/TBI: Hemodynamic Management

Blood Products

Liberal transfusion strategy might(?) be better

Plasma might have benefit?

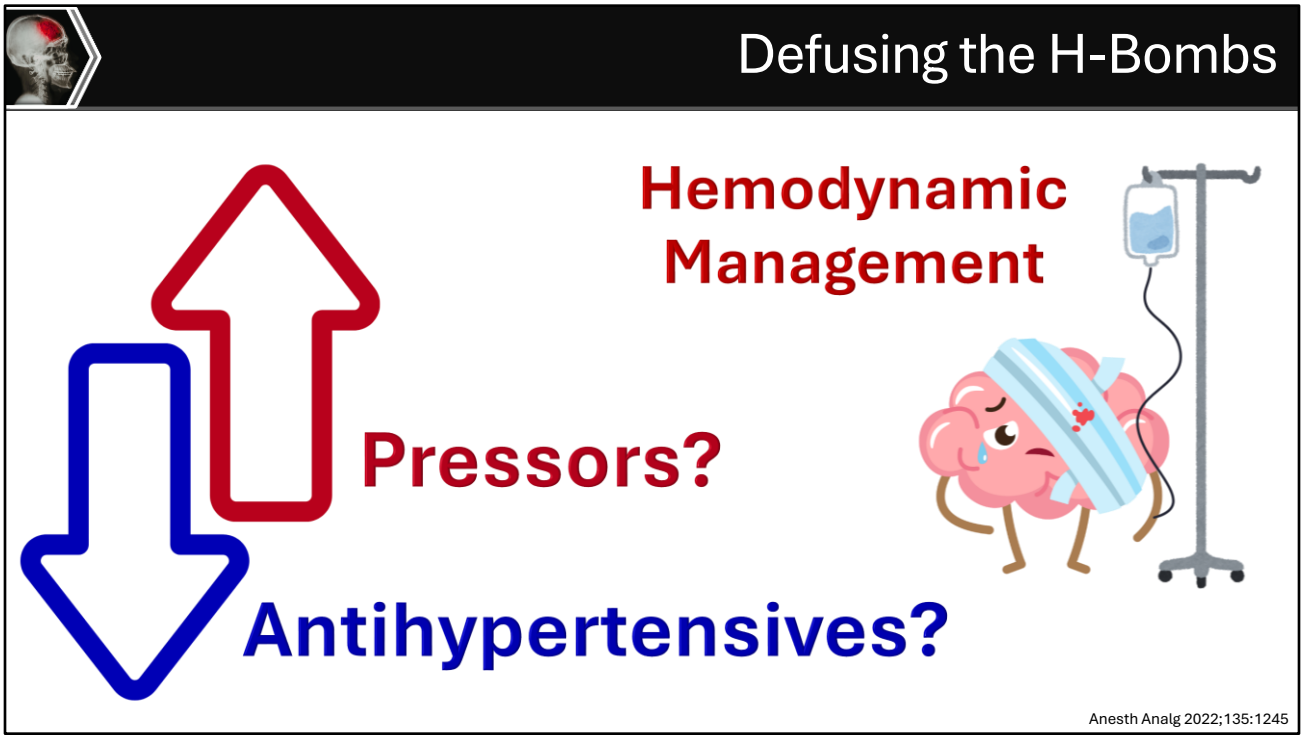


JAMA Netw Open 2020;3:e2016869
Injury 2025;56:112040
BMJ Open 2025;15:e107697
Neurocrit Care 2026;44:322
Prehosp Emerg Care 2023;27:507

Polytrauma patients should be resuscitated with blood products, if available.

When transfusing these patients, it may be reasonable to target a higher hemoglobin concentration; two separate systematic reviews and meta-analyses found that using a more liberal transfusion strategy (targeting a hemoglobin of 9-10 g/dL, rather than the traditional threshold of 7 g/dL) may result in more favorable neurologic outcomes and less morbidity.

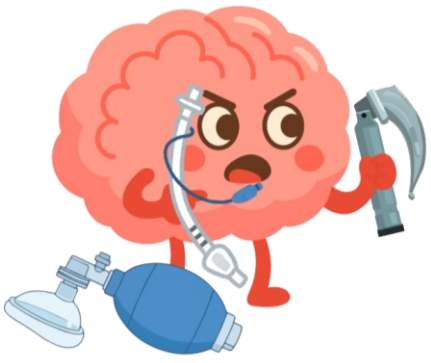
Research is conflicting on the benefit of plasma transfusion in TBI patients. A secondary analysis of PAMPer found better survival in TBI patients receiving plasma versus standard therapy, and another TQIP database study identified that plasma had limited effect on outcomes, but improved mortality in patients with TBI and traumatic hemorrhage.



Interestingly, no current guidelines explicitly mention the use of vasopressors (or avoidance thereof), so this is a bit of a “guidance-free” zone. Still, vasopressor use is common in severe TBI with hypotension, so use may be considered in patients that remain hypotensive (or relatively hypotensive) despite fluid resuscitation.

On the other end, however, it’s generally best to **AVOID** antihypertensives in the acute phase. While profound hypertension is *associated with* poor outcomes, it’s not been proven that the hypertension is the *cause of* this worse morbidity and mortality. It’s also quite possible that the increased blood pressure is a marker of increased ICP (á la Monroe-Kellie) and lowering blood pressure may make things worse.

Defusing the H-Bombs



Shock Index:

$$\frac{HR}{SBP}$$

SI > 0.9 = Bad

Am J Emerg Med 2022;58:229
J Intensive Care Med 2024;39:866

When discussing intubation for definitive airway management, it is important to adequately resuscitate patients BEFORE induction for RSI. While literature specific to the TBI population is limited, an elevated shock index has been shown in multiple settings to predict peri-intubation hypotension, cardiovascular collapse, and even cardiac arrest.

The shock index is easily calculated by dividing the heart rate by the systolic blood pressure, with a value greater that 0.9 predicting higher risk.

Since nobody enjoys performing long division in their head, *the "easy" 3 A.M. math here is that a HR that's higher than (or pretty darn close to) the SBP is a bad thing!*

Defusing the H-Bombs

Peri-RSI Hypotension:

Polytrauma

1.7×

Isolated TBI

13.6×

JAMA Netw Open 2025;8:e2544057

Peri-intubation hypotension is no better than other hypotensive episodes in the TBI patient. In patients with polytrauma and TBI, peri-intubation hypotension increased the odds of mortality by 1.7-fold (so about a 70% increase in odds of death).

For *isolated TBI* patients, however, peri-intubation hypotension increased the odds of death over 13-fold!

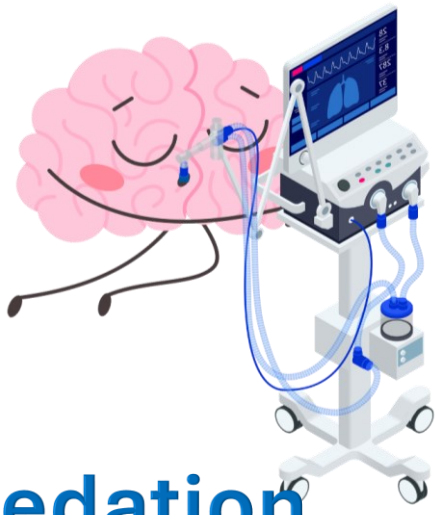
Vent strategies:

Normal PCO_2

V_T < 8 mL/kg IBW

Low PEEP

Adequate analgosedation



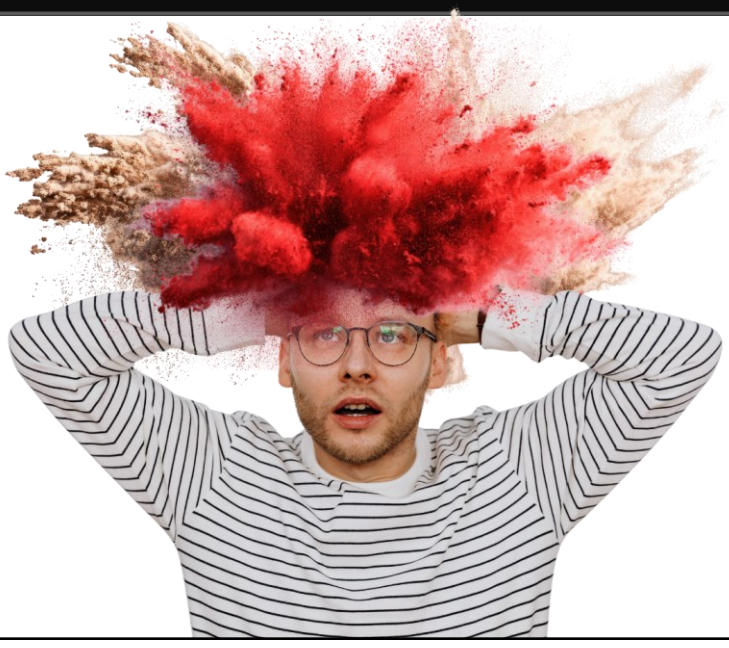
Chest 2025;168:1141
J Clin Med 2025;14:9443

When these patients are placed on the ventilator, in general conventional settings can be used.

Lung-protective tidal volumes, namely <8 mL/kg of ideal body weight, have been shown to improve mortality compared with higher tidal volumes, even in non-ARDS TBI patients.

PEEP (and high intrathoracic pressure, in general) poses a barrier to venous drainage from the cranium and can contribute to increased ICP, as can pain or agitation – so ensure adequate analgosedation in these patients, especially acutely.

Managing ICP Crises



ENLS Tier Zero IICP Management

- **HOB 30-45°**
- **Neck neutral**
- **Neck veins open**
- **Reduce stimuli**
- **Analgosedation**

ENLS / Neurocritical Care Society; 2025:117

These are the **BASIC** strategies that can be taken to prevent and begin to treat increased ICP in **any** patients at risk for IICP:

- Elevate the head of bed to 30-45°; for prehospital teams, even if transporting the patient on a backboard (that's a different topic altogether), the head of the stretcher can still be elevated somewhat with the backboard (I do it frequently in flight) – admittedly, you have to be cautious that the backboard doesn't fall off the bottom end of the stretcher, but achieving some degree of HOB elevation is likely beneficial (even though it will rarely be close to 30°). In the hospital, this can be done with head of bed, or using a reverse-Trendelenburg position.
- Keep the neck in neural alignment, not turned to one side or another. This, and keeping the neck veins open, optimizes the outflow opportunities for blood and CSF in case ICP begins to rise. To that end, try to keep the neck veins free from obstruction; for the EJ (and, to some degree, IJ) veins, this might mean quickly clearing a patient from a cervical collar, or avoiding placement of a cervical collar altogether if possible. Obviously, this is a risk/benefit ratio, and protocol may dictate the need to place a cervical collar, but increasing evidence finds they add little protective value and may actually be harmful in some circumstances (like this!).
- Stimuli should be reduced to the extent possible. Think about a migraine: dark, quiet, cool environment. That's not the reality in EMS, ED, and ICU environments, but we might be able to reduce some unnecessary stimuli (is the 1.5 minutes saved by lights and sirens really going to be worth it, or can you cut them off and travel smoother and with less ambient stimulus?)
- Adequate analgosedation to minimize pain and agitation

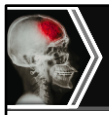
ENLS Tier One IICP Crisis

- **Hyperosmolar therapy (3%HTS)**
- **⇧Analgosedation**
- **$PaCO_2$ 35-38**

ENLS / Neurocritical Care Society; 2025:117

When we believe ICP is elevating, THIS is where we can begin to introduce advanced strategies:

- Hyperosmolar therapy can be used to bring ICP down acutely. There is generally no preference for hypertonic saline over mannitol, but recognize that mannitol is an *osmotic diuretic* and will lead to an eventual loss of volume through the urine. I tend to avoid mannitol in patients with softer blood pressures at baseline, and usually favor HTS as my first-line therapy. Hypertonic saline in typical 3% concentration can be safely administered via peripheral IV line. Symptom-based bolus is better than a slow infusion, since the sudden gradient change is what improves cerebral edema and reduces ICP.
- Increasing analgosedation (even to the point of deep sedation or RASS -5) can significantly reduce the brain's metabolic demands and potentially improve ICP.
- GENTLE hyperventilation to a LOW-NORMAL $PaCO_2$ of around 35-38 can be employed. Excessive hyperventilation and hypocarbia, as was discussed previously, can lead to a relative global hypoperfusion of the brain (from vasoconstriction) with resulting hypoxic insult and actually worsen morbidity and mortality.



The EPIC-TBI Bundle

- q3-5 minute HR/BP/SpO2
- 15 LPM oxygen NRB
- *ANY* SBP <90 mmHg:
  - 1 liter crystalloid bolus, **and**
  - IVF to keep SBP ≥90
- Vent rate timer on BVM; target EtCO2 40
  - NO hyperventilation, even for "herniation"

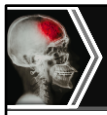
EPIC-TBI
Excellence in Prehospital Injury Care
Traumatic Brain Injury Project

JAMA Surg 2019;154:e191152

This was the EPIC-TBI protocol for included EMS agencies.
Importantly, this was evaluated as an "all-or-nothing" package deal, so it's not really possible to evaluate if one intervention was more impactful than another.

Note: EPIC-TBI patients were isolated TBI without hemorrhagic shock or polytrauma.

Of note – hyperventilation was highly discouraged, and interventions were targeted at maintaining a normal respiratory rate or a normal EtCO2. We know hyperventilation lowers PCO2, which, in turn, causes cerebral vasoconstriction. In theory, in "herniation," that can(?) lower ICP. However, (a) we're not always great at identifying who is truly herniating and who isn't, (b) hyperventilation as a temporizing measure is unlikely to convey a long-term benefit, so unless the patient is being hyperventilated as they're being wheeled into the OR, it's not likely to be helpful, and (c) if we overly hyperventilate patients that don't need it, we may cause more harm from limiting blood flow (i.e., oxygen/glucose delivery) than any purported good.



The EPIC-TBI Bundle

Improved Survival

1.5-2 x
Severe TBI

3x
Severe TBI Intubated

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

JAMA Surg 2019;154:e191152

As a result of the EPIC-TBI bundle, this region saw a 1.5-to-2-fold increase in survival to hospital discharge among patients with severe TBI, and a 3-fold higher survival-to-discharge among those severe TBI patients that required intubation. A limitation of this study is that neurologic outcomes/function wasn't recorded, so it's unclear the extent of deficit that survivors faced or their disposition (e.g., to home, to rehab, or to long-term care).

Mortality was not significantly impacted in those with moderate TBI (i.e., those likely to survive anyhow) or critical TBI (i.e., those likely to die anyhow).

Current Guideline Library

https://braintrauma.org/coma/guidelines-current

The current versions of many TBI management guidelines can be readily accessed online at:
https://braintrauma.org/coma/guidelines-current

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