SIBICC SEVERE TBI ALGORITHM

FOR PATIENTS WITH ICP AND BRAIN TISSUE OXYGEN MONITORING

A comprehensive protocol designed to assist clinicians managing sTBI patients undergoing ICP and $P_{bt}O_2$ monitoring.
These recommendations are based on combined expert opinion and reflect neither a standard-of-care nor a substitute for thoughtful individualized management.

BASIC CARE Applies to all Severe TBI Patients

TIER 0

Expected Interventions:
- Admission to ICU
- Endotracheal intubation and mechanical ventilation
- Serial evaluations of neurological status and pupillary reactivity
- Elevate HOB 30–45°
- Analgesia to manage signs of pain (not ICP directed)
- Sedation to prevent agitation, ventilator asynchrony, etc. (not ICP directed)
- Temperature management to prevent fever
  - Measure core temperature
  - Treat core temperature above 38°C
- Consider anti-seizure medications for 1 week only (in the absence of an indication to continue)
- Maintain CPP initially ≥ 60mmHg
- Maintain Hb > 7g/dL
- Avoid hyponatremia
- Optimize venous return from head (e.g. head midline, ensure cervical collars are not too tight)
- Arterial line for continuous blood pressure monitoring
- Maintain SpO_2 ≥ 94%

Recommended Interventions:
- Insertion of a central line
- End-tidal CO_2 monitoring

	ICP < 22 mmHg	ICP > 22 mmHg
$P_{bt}O_2$ > 20 mmHg	BASIC CARE Applies to all sTBI Patients Type A	Type B
$P_{bt}O_2$ < 20 mmHg	Type C	Type D

TYPE B ICP Elevated – Brain Oxygenation Normal

TIER 1
- Maintain CPP 60–70 mmHg
- Increase analgesia to lower ICP
- Increase sedation to lower ICP
- Maximum P_aCO_2 at low end of normal (35–38 mmHg/4.7–5.1 kPa)
- CSF drainage if EVD *in situ*
- Mannitol by intermittent bolus (0.25–1.0 g/kg)
- Hypertonic saline by intermittent bolus[1]
- Consider anti-seizure prophylaxis for one week only (unless indication to continue)
- Consider EEG monitoring

TIER 2
- Mild hypocapnia (range 32–35mmHg/ 4.3–4.6 kPa)
- Neuromuscular paralysis in adequately sedated patients if efficacious in lowering ICP[2]
- Perform MAP challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients[3].
  - Should be performed under direct supervision of a physician who can assess response and ensure safety
  - No other therapeutic adjustments (i.e. sedation) should be performed during the MAP Challenge
  - Initiate or titrate a vasopressor or inotrope to increase MAP by 10mmHg for not more than 20 minutes
  - Monitor and record key parameters (MAP, CPP, ICP and $P_{bt}O_2$) before during and after the challenge
  - Adjust vasopressor/inotrope dose based on study findings
- Raise CPP with fluid boluses, vasopressors and/or inotropes to lower ICP when autoregulation is intact

TIER 3
- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious[4]
- Secondary decompressive craniectomy
- Mild hypothermia (35–36°C) using active cooling measures
- Hyperventilation to P_aCO_2 of 30–32 mmHg/ 4.0–4.3 kPa

- Reexamine the patient and consider repeat CT to reevaluate intracranial pathology
- Reconsider surgical options for potentially surgical lesions
- Consider extracranial causes of ICP elevation
- Review that basic physiologic parameters are in desired range (e.g. CPP, blood gas values)
- Consider consultation with higher level of care if applicable for your health care system

[1] We recommend using sodium and osmolality limits of 155 mEq/L and of 320mEq/L respectively as administration limits for both mannitol and hypertonic saline.
[2] We recommend a trial dose of neuromuscular paralysis and only proceeding to a continuous infusion when efficacy is demonstrated.
[3] Rosenthal G, Sanchez-Mejia RO, Phan N, Hemphill JC 3rd, Martin C, Manley GT. Incorporating a parenchymal thermal diffusion cerebral blood flow probe in bedside assessment of cerebral autoregulation and vasoreactivity in patients with severe traumatic brain injury. J Neurosurg. 2011;114(1):62–70. doi:10.3171/2010.6. JNS091360
[4] Barbiturate administration should only be continued when a beneficial effect on ICP is demonstrated.
–Titrate barbiturate to achieve ICP control but do not exceed the dose which achieves burst suppression.
–Hypotension must be avoided when barbiturates are administered.

TYPE C ICP Normal – Brain Hypoxic

TIER 1
- Maximum CPP 60 – 70 mmHg
- Increase CPP to a maximum of 70 mmHg with fluid, vasopressors and/or inotropes
- Maintain P_aCO_2 > (35 mmHg/4.7 kPa)
- If P_aO_2 is already in desired range, further increase P_aO_2 by increasing F_iO_2 to 60%
- Consider EEG monitoring

TIER 2
- Ventilator management to increase P_aO_2 as high as 150 mmHg/20 kPa
- Decrease ICP to a threshold < 22 mmHg
- *Consider CSF drainage*
- Increase sedation to improve mechanical ventilation and $P_{bt}O_2$
- Neuromuscular paralysis in adequately sedated patients if efficacious in increasing $P_{bt}O_2$[1]
- Perform MAP challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients[2]
  - Should be performed under direct supervision of a physician who can assess response and ensure safety
  - No other therapeutic adjustment (i.e. sedation) should be performed during the MAP challenge
  - Initiate or titrate a vasopressor or inotrope to increase MAP by 10 mmHg for not more than 20 minutes
  - Monitor and record key parameters (MAP, CPP, ICP and $P_{bt}O_2$) before during and after the challenge
  - Adjust vasopressor/inotrope dose based on study findings
- Raise CPP to increase $P_{bt}O_2$ when supported by MAP Challenge
- Increase CPP above 70mmHg with fluid boluses, vasopressors and/or inotropes[3]

TIER 3
- Increase P_aCO_2 to 45–50 mmHg/6.0–6.7 kPa (but avoid intracranial hypertension)
- Consider normobaric hyperoxia to a P_aO_2 above 150 mmHg/20 kPa
- If $P_{bt}O_2$ remains < 20 mmHg despite P_aO_2 and CPP/MAP optimization, consider transfusing 1 unit of PRBCs if Hbg <9g/L

- Reexamine the patient and consider repeat CT to reevaluate intracranial pathology
- Reconsider surgical options for potentially surgical lesions
- Consider extracranial causes of ICP elevation
- Review that basic physiologic parameters are in desired range (e.g. CPP, blood gas values)
- Consider consultation with higher level of care if applicable for your health care system

[1] We recommend a trial dose of neuromuscular paralysis and only proceeding to a continuous infusion when efficacy is demonstrated.
[2] Rosenthal G, Sanchez-Mejia RO, Phan N, Hemphill JC 3rd, Martin C, Manley GT. Incorporating a parenchymal thermal diffusion cerebral blood flow probe in bedside assessment of cerebral autoregulation and vasoreactivity in patients with severe traumatic brain injury. J Neurosurg. 2011;114(1):62–70. doi:10.3171/2010.6. JNS091360
[3] Careful monitoring for respiratory complications is required when CPP is raised above 70 mmHg (Robertson C.S. et al. Prevention of secondary ischemic insults after severe head injury. Crit Care Med. 1999;27(10):2086–2095)

TYPE D ICP Elevated – Brain Hypoxic

TIER 1
- Maintain CPP 60 – 70 mmHg
- Increase CPP to a maximum of 70 mmHg with fluid, vasopressors and/or inotropes
- Increase analgesia to lower ICP/improve ventilation and $P_{bt}O_2$
- Increase sedation to lower ICP/improve ventilation and $P_{bt}O_2$
- Maintain P_aCO_2 > (35 mmHg/4.7 kPa)
- Mannitol by intermittent bolus (0.25–1.0 g/kg)
- Hypertonic saline by intermittent bolus[1]
- CSF drainage if EVD *in situ*
- Consider placement of EVD to drain CSF if parenchymal probe used initially
- If P_aO_2 is already in desired ranged, further increase P_aO_2 by increasing F_iO_2 to 60%
- Consider anti-seizure prophylaxis for 1 week only (unless indication to continue)
- Consider EEG monitoring

TIER 2
- Ventilator management to increase P_aO_2 as high as 150 mmHg/20 kPa
- Increase sedation to improve ICP and $P_{bt}O_2$
- Neuromuscular paralysis in adequately sedated patients if efficacious in decreasing ICP or increasing $P_{bt}O_2$[2]
- Perform MAP challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients[3]
  - Should be performed under direct supervision of a physician who can assess response and ensure safety
  - No other therapeutic adjustment (i.e. sedation) should be performed during the MAP challenge
  - Initiate or titrate a vasopressor or inotrope to increase MAP by 10 mmHg for not more than 20 minutes
  - Monitor and record key parameters (MAP, CPP, ICP and $P_{bt}O_2$) before during and after the challenge
  - Adjust vasopressor/inotrope dose based on study findings
- Raise CPP to decrease ICP and/or increase $P_{bt}O_2$ when supported by MAP Challenge
- Increase CPP above 70mmHg with fluid boluses, vasopressors and/or inotropes[4]

TIER 3
- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious[5]
- Secondary decompressive craniectomy
- Consider normobaric hyperoxia to a P_aO_2 above 150 mmHg/20 kPa
- If $P_{bt}O_2$ remains < 20 mmHg despite P_aO_2 and CPP/MAP optimization, consider transfusing 1 unit of PRBCs if Hgb <9g/L

- Reexamine the patient and consider repeat CT to reevaluate intracranial pathology
- Reconsider surgical options for potentially surgical lesions
- Consider extracranial causes of ICP elevation
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[5] Barbiturate administration should only be continued when a beneficial effect on ICP is demonstrated.
–Titrate barbiturate to achieve ICP control but do not exceed the dose which achieves burst suppression.
–Hypotension must be avoided when barbiturates are administered.

TREATMENT **NOT** RECOMMENDED FOR USE IN THE MANAGEMENT OF SEVERE TRAUMATIC BRAIN INJURY (when both ICP and $P_{bt}O_2$ are monitored)

- Mannitol by non-bolus continuous intravenous infusion
- Scheduled infusion of hyperosmolar therapy (e.g., every 4–6 h)
- Lumbar CSF drainage
- Furosemide
- Routine use of steroids
- Routine use of therapeutic hypothermia to temperatures below 35 °C due to systemic complications
- High-dose propofol to attempt burst suppression
- Decreasing P_aCO_2 below 30 mmHg/4.0 kPa
- Routinely raising CPP above 90 mmHg
- Barbiturates as treatment for low $P_{bt}O_2$ unless barbiturates are otherwise indicated
- Hypothermia as treatment for low $P_{bt}O_2$ unless hypothermia is otherwise indicated
- Hypercarbia in "type D" patients

CPP cerebral perfusion pressure, ICP intracranial pressure, kPa kiloPascals, P_aCO_2 arterial partial pressure of carbon dioxide, $P_{bt}O_2$ brain tissue partial pressure of oxygen, MAP Mean arterial pressure

CRITICAL NEUROWORSENING

A serious deterioration in clinical neurologic status which requires an immediate physician response such as:

- Spontaneous decrease in the GCS motor score of ≥ 1 points (compared with the previous examination)
- New decrease in pupillary reactivity
- New pupillary asymmetry or bilateral mydriasis
- New focal motor deficit
- Herniation syndrome or Cushing's Triad

RESPONSE TO CRITICAL NEUROWORSENING

Emergent evaluation to identify possible cause of neuroworsening. If herniation is suspected:

- Empiric treatment
  - –Hyperventilation[1]
  - –Bolus of hypertonic solution
- Consider emergent imaging or other testing
- Rapid escalation of treatment

[1] The hyperventilation P_aCO_2 limit 30 mmHg/4.0 kPa does not apply here

POSSIBLE CAUSES OF NEUROWORSENING

- Expanding intracranial mass lesion
- Cerebral edema
- Elevated ICP
- Stroke
- Electrolyte or other metabolic disturbance
- Medical comorbidity
- Medication effect
- Impaired renal or hepatic function
- Systemic hypotension
- Seizure or post-ictal state
- Hypoxemia/tissue hypoxia
- CNS infection
- Infection or sepsis
- Substance withdrawal
- Dehydration
- Hyper or hypothermia

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