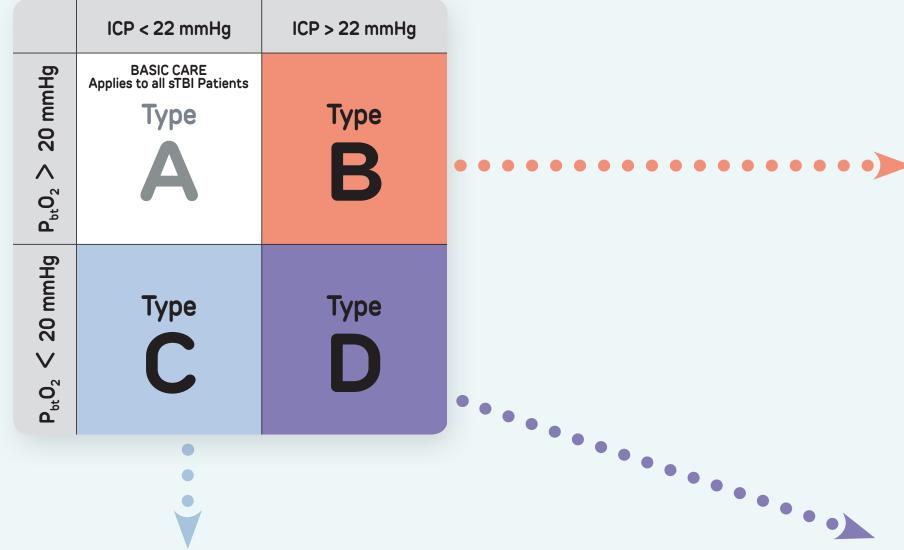
# SIBICC SEVERE TBI ALGORITHM

# FOR PATIENTS WITH ICP AND BRAIN TISSUE OXYGEN MONITORING

A comprehensive protocol designed to assist clinicians managing sTBI patients undergoing ICP and PbtO2 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 **Expected Interventions** Optimize venous return from head Temperature management to prevent (e.g. head midline, ensure cervical collars Admission to ICU are not too tight) Measure core temperature · Endotracheal intubation and mechanical · Arterial line for continuous blood - Treat core temperature above 38°C pressure monitoring · Consider anti-seizure medications Serial evaluations of neurological status Maintain SpO<sub>2</sub> ≥ 94% for 1 week only (in the absence of an and pupillary reactivity indication to continue) Recommended Interventions: • Elevate HOB 30-45° Maintain CPP initially ≥ 60mmHg · Insertion of a central line Analgesia to manage signs of pain (not ICP directed) Maintain Hb > 7g/dL • End-tidal CO<sub>2</sub> monitoring · Sedation to prevent agitation, ventilator Avoid hyponatremia asynchrony, etc. (not ICP directed) ICP > 22 mmHg ICP < 22 mmHg



# TYPE C ICP Normal – Brain Hypoxic

· Maximum CPP 60 - 70 mmHg · Increase CPP to a maximum of 70 mmHg with fluid, vasopressors and/or inotropes • Maintain  $P_aCO_2 > (35 \text{ mmHg/4.7 kPa})$ 

 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<sub>bt</sub>O<sub>2</sub>

 Neuromuscular paralysis in adequately sedated patients if efficacious in increasing P<sub>bt</sub>O<sub>2</sub><sup>1</sup> Perform MAP challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients<sup>2</sup>

- Should be performed under direct supervision of a physician who can assess response and ensure safety

• Increase  $P_aCO_2$  to 45-50 mmHg/6.0-6.7 kPa

(but avoid intracranial hypertension)

above 150 mmHg/20 kPa

· Consider normobaric hyperoxia to a P<sub>a</sub>O<sub>2</sub>

• If P<sub>bt</sub>O<sub>2</sub> remains < 20 mmHg despite P<sub>a</sub>O<sub>2</sub>

transfusing 1 unit of PRBCs if Hbg <9g/L

and CPP/MAP optimization, consider

- No other therapeutic adjustment (i.e. sedation) should be performed during the MAP challenge

• If  $P_aO_2$  is already in desired range,

Consider EEG monitoring

further increase PaO2 by increasing

- 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  $\cdot$  Raise CPP to increase  $P_{bt}O_2$  when supported by MAP Challenge Increase CPP above 70mmHg with fluid boluses, vasopressors and/or inotropes<sup>3</sup>

 Consider extracranial causes of ICP elevation Review that basic physiologic parameters are in desired range (e.g. CPP,

 Consider consultation with higher level of care if applicable for your health care system

Reexamine the patient

pathology

lesions

and consider repeat CT

to reevaluate intracranial

Reconsider surgical options

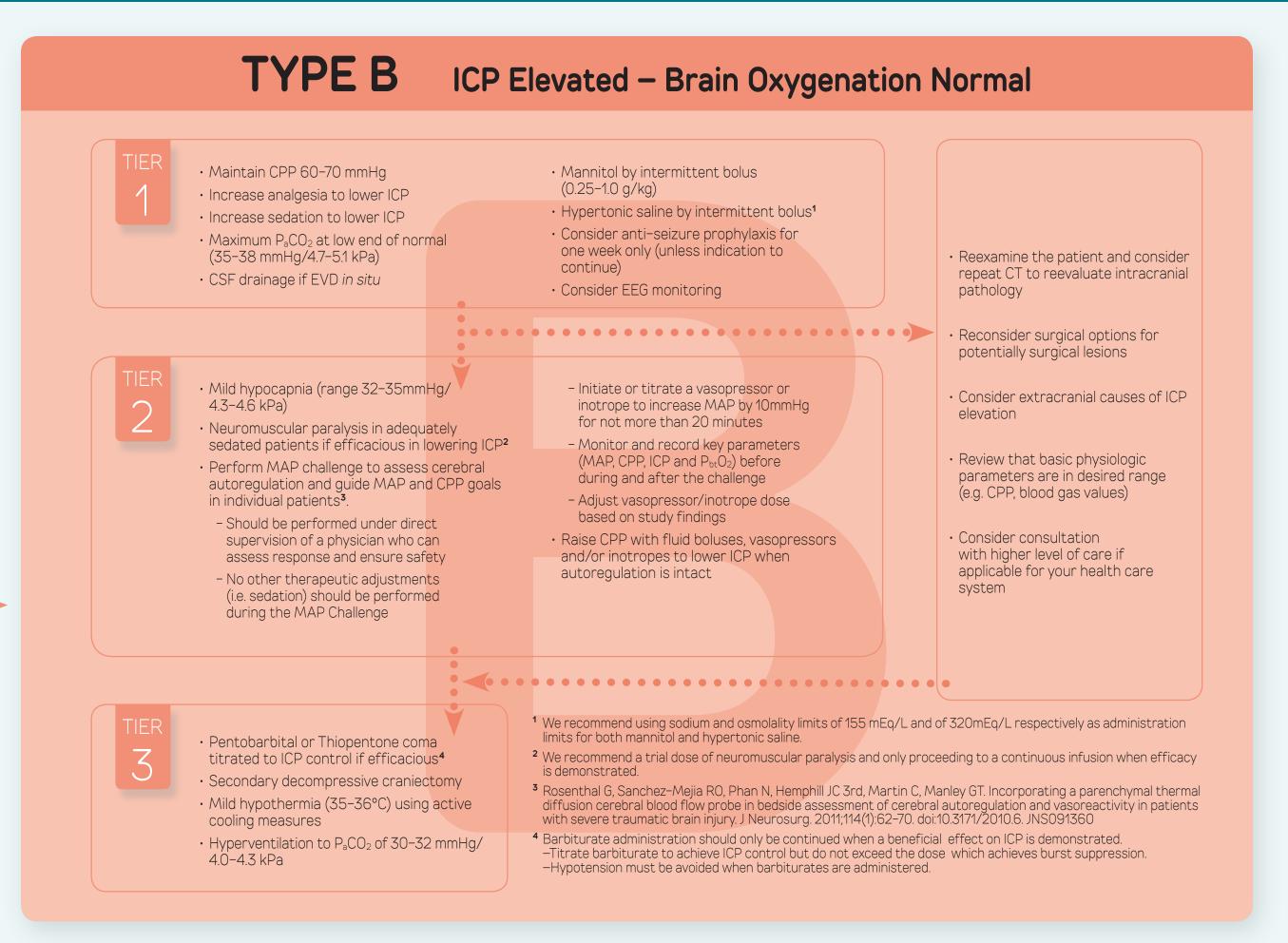
for potentially surgical

blood gas values)

<sup>1</sup> We recommend a trial dose of neuromuscular paralysis and only proceeding to a continuous infusion when efficacy is demonstrated.

<sup>2</sup> 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.

<sup>3</sup> 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)



#### ICP Elevated – Brain Hypoxic Hypertonic saline by intermittent bolus¹ · Maintain CPP 60 - 70 mmHg · CSF drainage if EVD in situ • Increase CPP to a maximum of 70 mmHg with fluid, vasopressors and/or inotropes Consider placement of EVD to drain CSF if parenchymal probe used initially Increase analgesia to lower ICP/improve Reexamine the patient ventilation and P<sub>bt</sub>O<sub>2</sub> • If $P_aO_2$ is already in desired ranged, further and consider repeat CT increase $P_aO_2$ by increasing $F_iO_2$ to 60% Increase sedation to lower ICP/improve to reevaluate intracranial ventilation and P<sub>bt</sub>O<sub>2</sub> · Consider anti-seizure prophylaxis for 1 week only pathology (unless indication to continue) • Maintain $P_aCO_2 > (35 \text{ mmHg}/4.7 \text{ kPa})$ Consider EEG monitoring Mannitol by intermittent bolus (0.25–1.0 g/kg) Reconsider surgical options for potentially surgical lesions Consider extracranial causes of ICP elevation - No other therapeutic adjustment (i.e. sedation) · Ventilator management to increase PaO2 should be performed during the MAP challenge as high as 150 mmHg/20 kPa Review that basic - Initiate or titrate a vasopressor or inotrope to $\cdot$ Increase sedation to improve ICP and $P_{bt}O_2$ physiologic parameters increase MAP by 10 mmHg for not more than are in desired range (e.g. Neuromuscular paralysis in adequately sedated 20 minutes CPP, blood gas values) patients if efficacious in decreasing ICP or - Monitor and record key parameters (MAP, CPP, ICP increasing P<sub>bt</sub>O<sub>2</sub><sup>2</sup> and $P_{bt}O_2$ ) before during and after the challenge · Perform MAP challenge to assess cerebral Consider consultation - Adjust vasopressor/inotrope dose based on study autoregulation and guide MAP and CPP goals in with higher level of care if findings individual patients<sup>3</sup> applicable for your health · Raise CPP to decrease ICP and/or increase PbtO2 care system - Should be performed under direct supervision when supported by MAP Challenge of a physician who can assess response and ensure safety Increase CPP above 70mmHg with fluid boluses, vasopressors and/or inotropes4 <sup>1</sup> We recommend using sodium and osmolality limits of 155 mEq/L and of 320 mEq/L respectively as administration limits for both mannitol and hypertonic saline. Pentobarbital or Thiopentone coma <sup>2</sup> We recommend a trial dose of neuromuscular paralysis and only proceeding to a continuous infusion titrated to ICP control if efficacious<sup>5</sup> when efficacy is demonstrated. Secondary decompressive craniectomy <sup>3</sup> 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 • Consider normobaric hyperoxia to a $P_aO_2$ above 150 mmHg/20 kPa <sup>4</sup> 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) • If $P_{ht}O_2$ remains < 20 mmHg despite

<sup>5</sup> Barbiturate administration should only be continued when a beneficial effect on ICP is demonstrated.

-Hypotension must be avoided when barbiturates are administered.

-Titrate barbiturate to achieve ICP control but do not exceed the dose which achieves burst suppression.

P<sub>a</sub>O<sub>2</sub> and CPP/MAP optimization,

Hgb <9g/L

consider transfusing 1 unit of PRBCs if

## TREATMENT **NOT** RECOMMENDED FOR USE IN THE MANAGEMENT OF SEVERE TRAUMATIC BRAIN INJURY (when both ICP and P<sub>bt</sub>O<sub>2</sub> 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 PaCO2 below 30 mmHg/4.0 kPa
- Routinely raising CPP above 90 mmHg
- · Barbiturates as treatment for low PhtO2 unless barbiturates are otherwise indicated
- Hypothermia as treatment for low P<sub>bt</sub>O<sub>2</sub>
- unless hypothermia is otherwise indicated · Hypercarbia in "type D" patients

CPP cerebral perfusion pressure, ICP intracranial pressure, kPa kiloPascals, PaCO2 arterial partial pressure of carbon dioxide, PbtO2 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

<sup>1</sup>The hyperventilation P<sub>a</sub>CO<sub>2</sub> limit

30 mmHg/4.0 kPa does not apply here

- mvdriasis 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
- -Bolus of hypertonic solution
- Consider emergent imaging or other testing
- · Rapid escalation of treatment

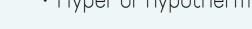
# POSSIBLE CAUSES OF NEUROWORSENING

- Expanding intracranial
- mass lesion
- · Cerebral edema
- Elevated ICP
- Stroke
- Electrolyte or other metabolic disturbance
- Medical comorbidity
- Medication effect
- · CNS infection · Impaired renal or hepatic Infection or sepsis
- function

· Seizure or post-ictal state

- Systemic hypotension
- Substance withdrawal
  - Dehydration
  - Hyper or hypothermia

Hypoxemia/tissue hypoxia





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