ICP Management

Lap-For Eddie Chan MD, MSc

Neurosurgical section
Kaohsiung Armed Forces General Hospital

ICP Physiology

- Four components of ICP related to :
  - Arterial blood flow
  - Venous blood flow
  - CSF circulation
  - Brain volumetric changes (including contusions)

Pathophysiology of Acquired Brain Injury

Traumatic causes:-

- Motor vehicles
- Falls
- Struck by/against
- Assaults

Acquired causes:-

- Ischemic
- Spontaneous ICH
- Aneurysm
- Hypoxia
- Infection
- Tumors

Cerebral Physiology

- Brain/Cranium: Average 1400g/1700ml
- Intracranial fluid content:-
  - CSF 150ml (25ml ventrices & 125ml in subarachnoid spaces)
- CBF: 45-60ml/100g/min
- CPP: 50-150mmHg (CPP=MAP-ICP)
- Cerebral metabolism:-
  - 25% body glucose, 20% body O2 consumption, 15% cardiac output
- Intracranial temperature: 37.39-39.5°C
- ICP: 5-15mmHg in supine

Monro-kellie Doctrine(1824)

- Cerebral blood flow is passively control by the intracranial volume change.
- In an intact skull, the volume of the CSF, blood and brain is constant. An increase in one must result in a decrease in one or both of the other substances. (Harvey Cushing 1926)

Cerebral Hemodynamics

- Cerebral autoregulation:-
  - ensures stable perfusion of the brain, independent of the systemic blood pressure.
- Vasomotor reactivity:-
  - O2 demand -> adapts hemodynamics to arterial CO2/pH of the tissue.
- Neurovascular coupling:-
  - Increase brain activity -> adapts the perfusion to increase metabolic demand.

(Woff M. Front Neurosci. 2015; 36:40-56)

Why Brain Temperature Higher than our Core Temperature?

- Human brain comprises about 20-25% of whole body energy budget. Possibly related to the brain's need in complex thinking and learning. Primates (8-10%), other mammals(3-5%)
- All the neuron activity affected by minute temperature variations:-
  - Ionic currents, membrane potential, input resistance, action potential, nerve conduction velocity, and synaptic transmission.
- CBF: Principal mechanism to maintain brain and body temperature coupling.
- Whole body cooling -> brain hypothermia -> decrease CBF

(Wang H et al. Front Neurosci. 2014; Oct (8); 307)

Pathophysiology of TBI
Primary and Secondary Brain Injury

- Primary brain injury (unavoidable injury)
- Secondary brain injury (Preventable damages)
  - Edema, Impaired metabolism, Altered CBF, Free radical formation, Excitotoxicity.
  - Systemic factors: Hypertension, Hypoxia, Anemia, Hyperthermia, Hypercapnia, Hyperglyemia, Acid-base imbalance, Metabolic disorders, Systemic inflammation and infection.
  - Intracranial factors: IICP, Space-occupying lesions, Brain edema, Cerabral vasospasm, Hydrocephalus, Intracranial infections, Seizures, acute attacks and NCSE(non-convulsive status epilepticus), decrease CBF, Cerebral metabolic dysfunction, Electrolytes imbalance, Free radical production.

Intensive Care Med (2019) 45:1783–1794
https://doi.org/10.1007/s00134-019-05805-9

CONFERENCE REPORTS AND EXPERT PANEL

A management algorithm for patients with intracranial pressure monitoring: the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC)

Gregory W. J. Hawryluk[1], Sergio Aguilera[2,3], Andras Buki[4,5], Eileen Bulger[6], Giuseppe Citerio[7,8], D. Jamie Cooper[9,10], Ramon Diaz Arrastia[11], Michael Diringer[12,13], Anthony Figaji[14], Guoyi Gao[15], Romergryko Geocadin[16], Jamshid Ghajar[17], Odette Harris[18], Alan Hoffer[19], Peter Hutchinson[20], Mathew Joseph[21], Ryan Kitagawa[22], Geoffrey Manley[23], Stephan Mayer[24], David K. Menon[25], Geert Meyfroidt[26], Daniel B. Michael[27], Mauro Oddo[28], David Okonkwo[29], Mayur Patel[30], Claudia Robertson[31], Jeffrey V. Rosenfeld[32,33], Andres M. Rubiano[34,35], Juan Sahuquillo[36], Franco Servadei[37], Lori Shutter[38], Deborah Stein[39], Nino Stocchetti[40,41], Fabio Silvio Taccone[42], Shelly Timmons[43], Eve Tsai[44], Jamie S. Ullman[45], Paul Vespa[46,47,48,49], Walter Videtta[50], David W. Wright[51], Christopher Zammit[52] and Randall M. Chesnut[53,54,55,56*]

Tier Zero (Basic Severe TBI Care - Not ICP Dependent)

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 1w only (in the absence of an indication to continue)
- Maintain CPP initially ≥ 60 mmHg
- Maintain Hb > 7g/dL
- Avoid hyponatremia
- Optimize venous return from head (eg. keeping head midline, ensure cervical collars are no too tight)
- Arterial line continuous blood pressure monitoring
- Maintain SpO2 ≥ 94%

Recommended Interventions:

- Insertion of a central line
- End-tidal CO_2 monitoring

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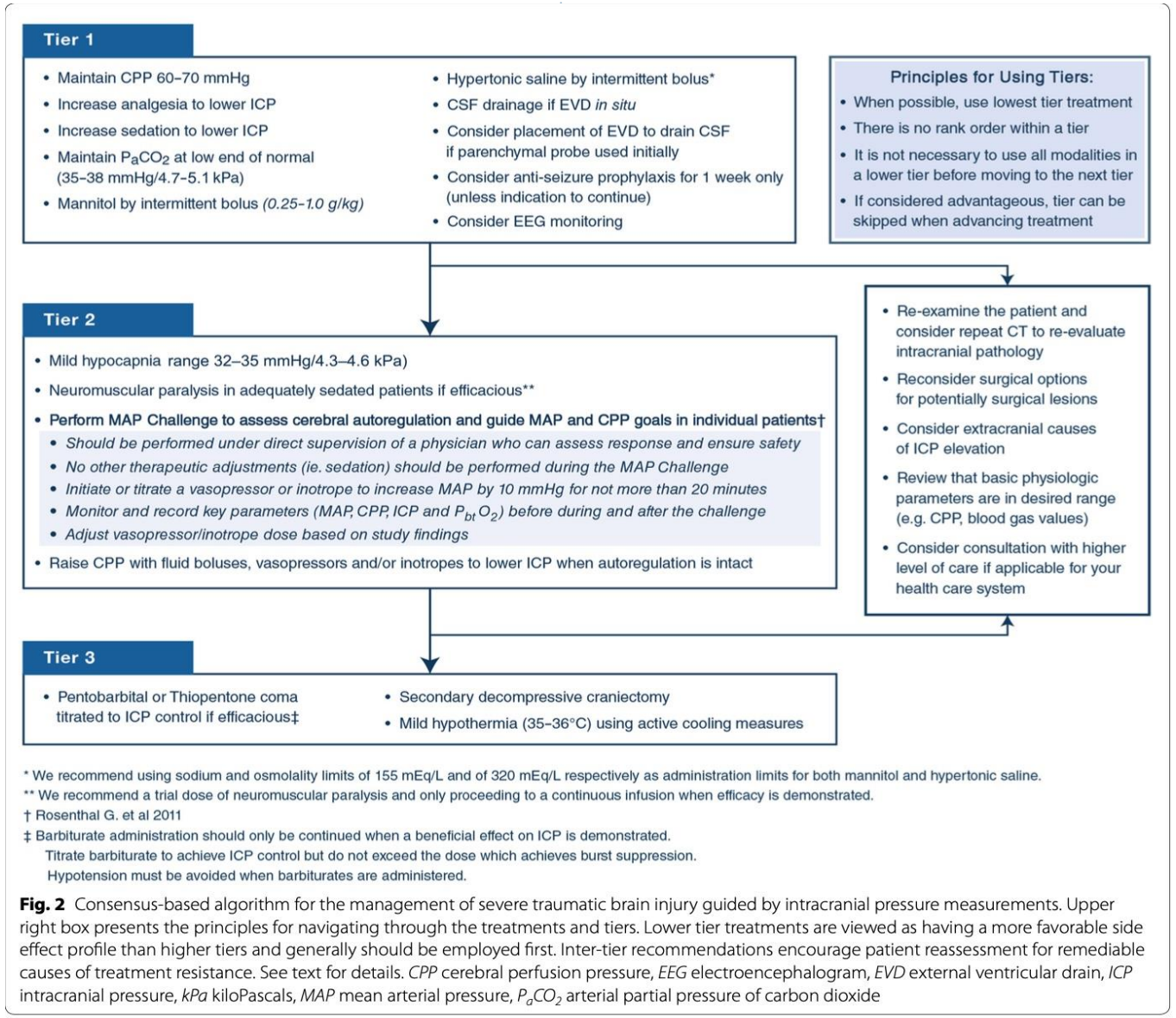

Fig. 2 Consensus-based algorithm for the management of severe traumatic brain injury guided by intracranial pressure measurements. Upper right box presents the principles for navigating through the treatments and tiers. Lower tier treatments are viewed as having a more favorable side effect profile than higher tiers and generally should be employed first. Inter-tier recommendations encourage patient reassessment for remediable causes of treatment resistance. See text for details. *CPP* cerebral perfusion pressure, *EEG* electroencephalogram, *EVD* external ventricular drain, *ICP* intracranial pressure, *kPa* kiloPascals, *MAP* mean arterial pressure, P_aCO_2 arterial partial pressure of carbon dioxide

Tier Zero (Basic Severe TBI Care - Not ICP Dependent)

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- Arterial line continuous blood pressure monitoring
- Maintain SpO2 ≥ 94%

Recommended Interventions:

- Insertion of a central line
- End-tidal CO_2 monitoring

Expected Interventions: Tier Zero (SIBICC)

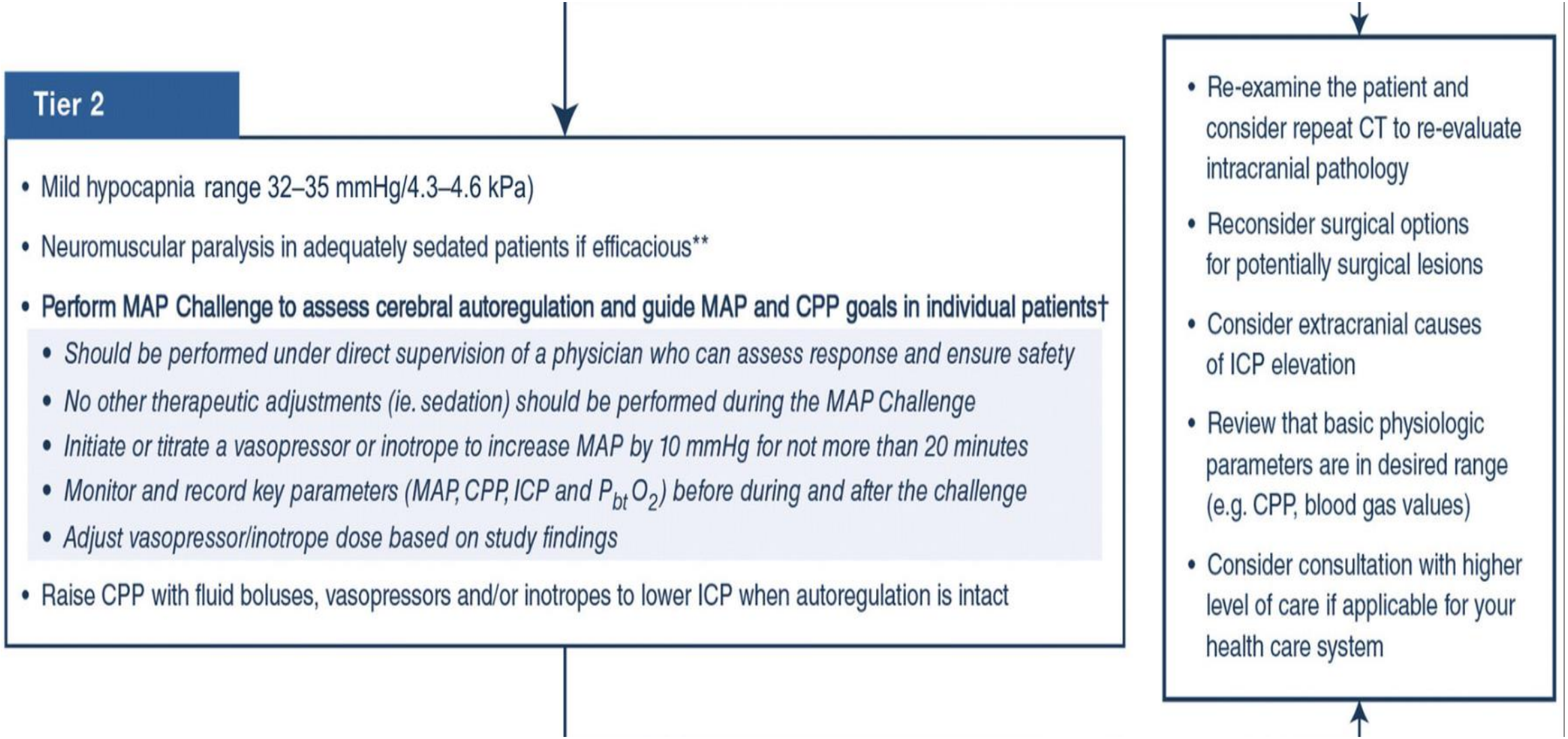
- ICU admission
- Endotracheal intubation & mechanical ventilation
- Serial neurological evaluations
- HOB elevation (30-45°)
- Pain control (not ICP-directed)
- Sedation to prevent agitation (not ICP-directed)
- Treat fever > 38°C
- Consider short-term anti-seizure medication (1 week)
- Maintain CPP ≥ 60mmHg, Hb > 7g/dl, & SpO2 ≥ 94%
- Avoid hyponatremia
- Optimize venous return from the head (keep head midline, not over-tight neck collar)
- Continuous arterial blood pressure monitoring
- Recommended Interventions: Central line insertion, End-tidal CO2 monitoring

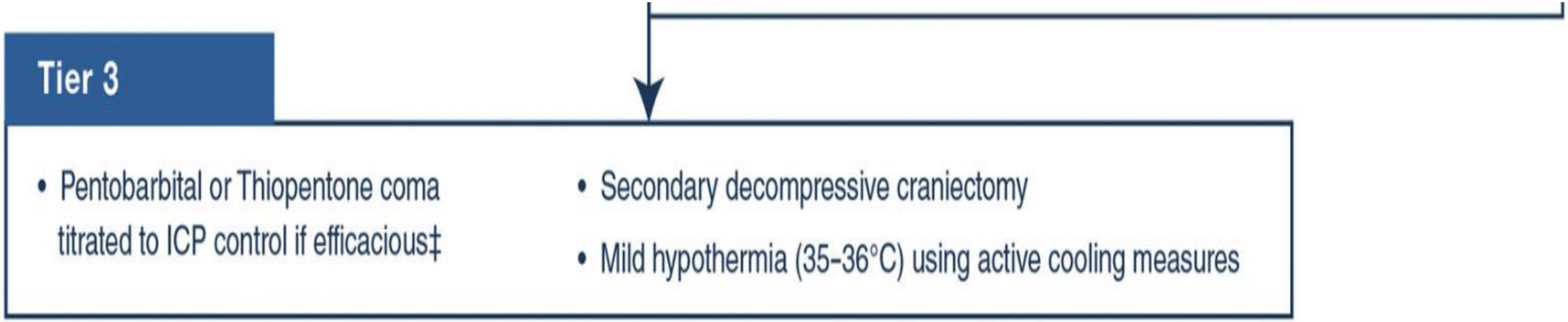
Tier 1

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

Principles for Using Tiers:

- When possible, use lowest tier treatment
- There is no rank order within a tier
- It is not necessary to use all modalities in a lower tier before moving to the next tier
- If considered advantageous, tier can be skipped when advancing treatment


Tier 2
• Mild hypocapnia range 32–35 mmHg/4.3–4.6 kPa)
• Neuromuscular paralysis in adequately sedated patients if efficacious**
• Perform MAP Challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients†
• Should be performed under direct supervision of a physician who can assess response and ensure safety
• No other therapeutic adjustments (ie. 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 with fluid boluses, vasopressors and/or inotropes to lower ICP when autoregulation is intact
• Re-examine the patient and consider repeat CT to re-evaluate 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



* 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.

** We recommend a trial dose of neuromuscular paralysis and only proceeding to a continuous infusion when efficacy is demonstrated.

† Rosenthal G. et al 2011

‡ 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.

Critical Neuroworsening

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

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 include:

- 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

** the hyperventilation $PaCO_2$ limit of 30 mmHg/4.0 kPa does not apply here

Treatment Not Recommended for use in the Management of Severe TBI (when only ICP is 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
- High-dose propofol to attempt burst suppression
- Routinely decreasing $PaCO_2$ below 30 mmHg/4.0 kPa
- Routinely raising CPP above 90 mmHg

Effect of Sedation Therapy

Adequate sedation and pain management can help lower metabolic demand, maintain stable blood pressure, and ultimately aid in stabilizing intracranial pressure (ICP).

Main effect of sedation:

1. Reduction of ICP through effective sedation and pain management
   - Reduces sympathetic activation caused by pain and agitation, helping to avoid elevated blood pressure and increased cerebral blood flow (CBF).
   - Enhances patient-ventilator synchrony, which helps prevent ICP elevation due to hypoxia and hypercapnia.
2. Neuroprotection through decreased metabolism
   - Sedatives lower the cerebral metabolic rate of oxygen ($CMRO_2$), resulting in stabilized CBF.
   - This action helps to prevent brain edema and secondary brain injury.
3. Prevention of sudden ICP spikes
   - Reduces coughing reflexes and body movements, which helps prevent the Valsalva reflex (increased intrathoracic pressure leading to decreased venous return and elevated ICP).

Effect of Sedation

Sedatives	Main Effect	Impact on ICP	Considerations
Propofol	Metabolic suppression Anticonvulsant	Lower ICP (fast-acting)	Risk of hypotension Propofol infusion syndrome (PRIS)
Midazolam (BZDs)	Sedation Anxiolysis	Moderate ICP reduction	Accumulates easily Delayed awakening
Dexmedetomidine	Sedation Analgesia Sympatholytic effect	Mild ICP reduction	Risk of bradycardia Hypotension
Fentanyl (Opioid)	Strong analgesia	ICP reduction (initial transient increase)	Risk of hypotension Respiratory depression

Propofol in the first line choice due to its strong ICP-lowering effect, rapid onset & manageable awakening. Midazolam may be used in combination for seizure prevention or post-surgical management.

Head Elevation and Neutral Position

- Advantages of head elevation
  - ICP lowering effect
    - Enhances venous return
    - Faciliates the flow of CSF into the spinal canal

- Disadvantages of head elevation
  - Decreases CPP due to reduced cerebral circulation

Osmotic Therapy: Mannitol vs Hypertonic Saline

Comparison	Mannitol	Hypertonic Saline
Mechanism	Osmotic effect Cerebral vasoconstriction	Strong osmotic effect Uneasy to cross BBB
ICP-lowering effect	Rapid onset	Longer duration
Effect on CPP	May transiently decrease	Tends to increase
Renal impact	Renal clearance-dependent Risk of renal failure	Not dependent on renal clearance
Side effects	Hypotension Renal dysfunction Rebound edema	Hypernatremia Metabolic acidosis
Clinical preference	Effective if BP is stable	Preferred in hypotensive or renal-impaired patients

Agent selection should be tailored to individual patient conditions.
Close monitoring of serum sodium and osmolarity is essential.

Brain Trauma FOUNDATION

Guidelines for the Management of Severe Traumatic Brain Injury

4th Edition

Nancy Carney, PhD	Oregon Health & Science University, Portland, OR
Annette M. Totten, PhD	Oregon Health & Science University, Portland, OR
Cindy O'Reilly, BS	Oregon Health & Science University, Portland, OR
Jamie S. Ullman, MD	Hofstra North Shore-LIJ School of Medicine, Hempstead, NY
Gregory W. J. Hawryluk, MD, PhD	University of Utah, Salt Lake City, UT
Michael J. Bell, MD	University of Pittsburgh, Pittsburgh, PA
Susan L. Bratton, MD	University of Utah, Salt Lake City, UT
Randall Chesnut, MD	University of Washington, Seattle, WA
Odette A. Harris, MD, MPH	Stanford University, Stanford, CA
Niranjan Kissoon, MD	University of British Columbia, Vancouver, BC
Andres M. Rubiano, MD	El Bosque University, Bogota, Colombia; MEDITECH Foundation, Neiva, Colombia
Lori Shutter, MD	University of Pittsburgh, Pittsburgh, PA
Robert C. Tasker, MBBS, MD	Harvard Medical School & Boston Children's Hospital, Boston, MA
Monica S. Vavilala, MD	University of Washington, Seattle, WA
Jack Wilberger, MD	Drexel University, Pittsburgh, PA
David W. Wright, MD	Emory University, Atlanta, GA
Jamshid Ghajar, MD, PhD	Stanford University, Stanford, CA

Reviewed for evidence-based integrity and endorsed by the American Association of Neurological Surgeons and the Congress of Neurological Surgeons.

September 2016

Level of Recommendation

Recommendations in this edition are designated as Level I, Level II A, Level II B, or Level III. The Level of Recommendation is determined by the assessment of the quality of the body of evidence, rather than the class of the included studies.

- The levels were *primarily* based on the quality of the body of evidence as follows:
  - Level I recommendations were based on a high-quality body of evidence.
  - Level II A recommendations were based on a moderate-quality body of evidence.
  - Level II B and III recommendations were based on a low-quality body of evidence.

1. Decompressive Craniectomy

RECOMMENDATIONS*

Level I

- There was insufficient evidence to support a Level I recommendation for this topic.

Level II A

- Bifrontal DC is not recommended to improve outcomes as measured by the Glasgow Outcome Scale–Extended (GOS-E) score at 6 months post-injury in severe TBI patients with diffuse injury (without mass lesions), and with ICP elevation to values >20 mm Hg for more than 15 minutes within a 1-hour period that are refractory to first-tier therapies. However, this procedure has been demonstrated to reduce ICP and to minimize days in the intensive care unit (ICU).
- A large frontotemporoparietal DC (not less than 12 x 15 cm or 15 cm diameter) is recommended over a small frontotemporoparietal DC for reduced mortality and improved neurologic outcomes in patients with severe TBI.

2. Prophylactic Hypothermia

RECOMMENDATIONS

Level I and II A

- There was insufficient evidence to support a Level I or II A recommendation for this topic.

Level II B

- Early (within 2.5 hours), short-term (48 hours post-injury) prophylactic hypothermia is not recommended to improve outcomes in patients with diffuse injury.

3. Hyperosmolar Therapy

RECOMMENDATIONS

Level I, II, and III

- Although hyperosmolar therapy may lower intracranial pressure, there was insufficient evidence about effects on clinical outcomes to support a specific recommendation, or to support use of any specific hyperosmolar agent, for patients with severe traumatic brain injury.

4. Cerebrospinal Fluid Drainage

RECOMMENDATIONS

Level I and II

- There was insufficient evidence to support a Level I or II recommendation for this topic.

Level III

- An EVD system zeroed at the midbrain with continuous drainage of CSF may be considered to lower ICP burden more effectively than intermittent use.
- Use of CSF drainage to lower ICP in patients with an initial Glasgow Coma Scale (GCS) <6 during the first 12 hours after injury may be considered.

5. Ventilation Therapies

RECOMMENDATIONS

Level I and II A

- There was insufficient evidence to support a Level I or II A recommendation for this topic.

Level II B

- Prolonged prophylactic hyperventilation with partial pressure of carbon dioxide in arterial blood (PaCO2) of 25 mm Hg or less is not recommended.

6. Anesthetics, Analgesics, and Sedatives

RECOMMENDATIONS

Level I and II A

- There was insufficient evidence to support a Level I or Level IIA recommendation for this topic.

Level II B

- Administration of barbiturates to induce burst suppression measured by EEG as prophylaxis against the development of intracranial hypertension is not recommended.
- High-dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment. Hemodynamic stability is essential before and during barbiturate therapy.
- Although propofol is recommended for the control of ICP, it is not recommended for improvement in mortality or 6-month outcomes. Caution is required as high-dose propofol can produce significant morbidity.

7. Steroids

RECOMMENDATIONS

Level I

- The use of steroids is not recommended for improving outcome or reducing ICP. In patients with severe TBI, high-dose methylprednisolone was associated with increased mortality and is contraindicated.

8. Nutrition

RECOMMENDATIONS

Level I

- There was insufficient evidence to support a Level I recommendation for this topic.85

Level II A

- Feeding patients to attain basal caloric replacement at least by the fifth day and, at most, by the seventh day post-injury is recommended to decrease mortality.

Level II B

- Transgastric jejunal feeding is recommended to reduce the incidence of ventilator-associated pneumonia.

9. Infection Prophylaxis

RECOMMENDATIONS

Level I

- There was insufficient evidence to support a Level I recommendation for this topic.

Level II A

- Early tracheostomy is recommended to reduce mechanical ventilation days when the overall benefit is felt to outweigh the complications associated with such a procedure. However, there is no evidence that early tracheostomy reduces mortality or the rate of nosocomial pneumonia.
- The use of povidone-iodine (PI) oral care is not recommended to reduce ventilator-associated pneumonia and may cause an increased risk of acute respiratory distress syndrome.

Level III

- Antimicrobial-impregnated catheters may be considered to prevent catheter-related infections during EVD.

10. Deep Vein Thrombosis Prophylaxis

RECOMMENDATIONS

Level I and II

- There was insufficient evidence to support a Level I or II recommendation for treatment of deep vein thrombosis (DVT) in severe TBI patients.

Level III

- Low molecular weight heparin (LMWH) or low-dose unfractioned heparin may be used in combination with mechanical prophylaxis. However, there is an increased risk for expansion of intracranial hemorrhage.

11. Seizure Prophylaxis

RECOMMENDATIONS

Level I

- There was insufficient evidence to support a Level I recommendation for this topic.

Level II A

- Prophylactic use of phenytoin or valproate is not recommended for preventing late PTS.
- Phenytoin is recommended to decrease the incidence of early PTS (within 7 days of injury), when the overall benefit is felt to outweigh the complications associated with such treatment. However, early PTS have not been associated with worse outcomes.

At the present time there is insufficient evidence to recommend levetiracetam over phenytoin regarding efficacy in preventing early post-traumatic seizures and toxicity.

12. Intracranial Pressure Monitoring

RECOMMENDATIONS

Level I and II A

- There was insufficient evidence to support a Level I or II A recommendation for this topic.

Level II B

- Management of severe TBI patients using information from ICP monitoring is recommended to reduce in-hospital and 2-week post-injury mortality.

13. Cerebral Perfusion Pressure Monitoring

RECOMMENDATIONS

Level I

- There was insufficient evidence to support a Level I recommendation for this topic.

Level II B

- Management of severe TBI patients using guidelines-based recommendations for CPP monitoring is recommended to decrease 2-week mortality.

14. Advanced Cerebral Monitoring

RECOMMENDATIONS

Level I and II

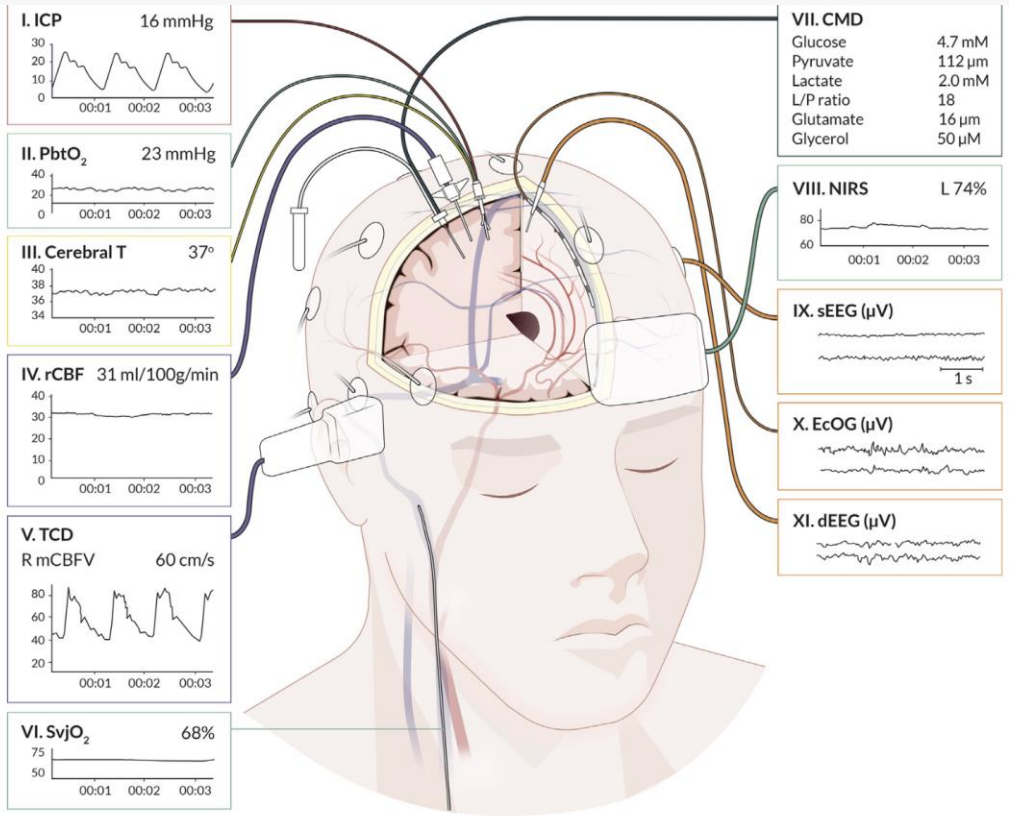
- There was insufficient evidence to support a Level I or II recommendation for this topic. (Although patients with desaturations identified with advanced cerebral monitoring have poorer outcomes, Level II evidence showed no improvement in outcomes for monitored patients.)

Level III

- Jugular bulb monitoring of arteriovenous oxygen content difference (AVDO2), as a source of information for management decisions, may be considered to reduce mortality and improve outcomes at 3 and 6 months post-injury.

Cerebral Multimodality Monitoring

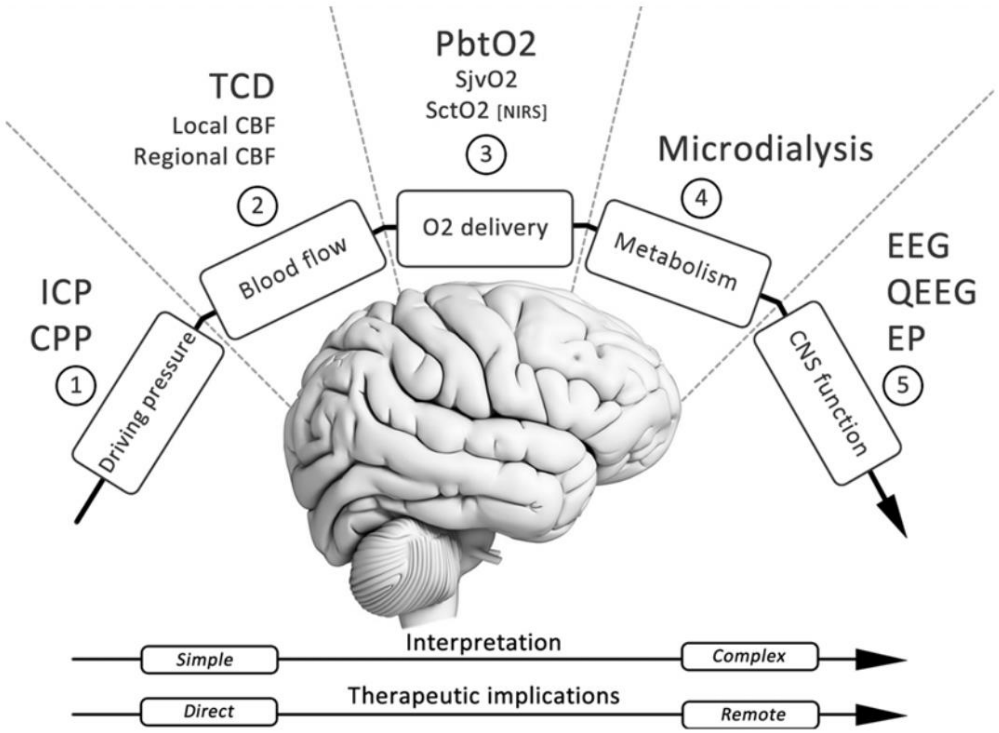
Cerebral multimodality monitoring in adult neurocritical care patients with acute brain injury: A narrative review



Front. Physiol., 01 December 2022
Sec. Computational Physiology and Medicine
Volume 13 - 2022 |

Cerebral T, cerebral temperature; CMD, cerebral microdialysis; dEEG, depth electroencephalography; ECoG, electrocorticography; ICP, intracranial pressure; NIRS, near-infrared spectroscopy; $PbtO_2$, partial pressure of brain tissue oxygenation; rCBF, regional cerebral blood flow; sEEG, surface electroencephalography; $SvjO_2$, jugular bulb venous oximetry; TCD, transcranial Doppler. Professional illustration by Anna Sieben (Sieben Medical Art).

Multimodality Monitoring in Neurocritical Care



	Goal-directed variable target
ICP	Less than 20 mmHg
CPP	Greater than 50–60 mmHg
$PbtO_2$	Greater than 10–15 mmHg
JvO_2	Greater than 50%
EEG	Avoid electrographic, non-convulsive seizures
BrGluc	Greater than 0.7 mol/L
BrLac	Less than 2.9 mmol/L
BrPyr	Greater than 166 μmol/L
LPR	Less than 40

(heustein Peripheral Brain pbrainmd.wordpress.com. December 13, 2015)

Multimodality Monitoring in Neurocritical Care

1. The driving pressure method relies on continuous monitoring of ICP and CPP.
2. For CBF assessment, TCD is the standard technique, although local and regional CBF should also be considered.
3. PbtO2 has gradually replaced the traditional SjvO2 for monitoring O2 delivery, while cerebral oximetry remains an indicator of O2 diffusion rather than a direct metabolic metric.
4. Metabolism is primarily assessed through microdialysis, where increased glutamate, a high lactate/pyruvate ratio, and low glucose levels indicate CNS cellular hypoxia.
5. Electrophysiology is uniquely capable of providing insights into CNS function and neuronal activity.

(heustein Peripheral Brain pbrainmd.wordpress.com. December 13, 2015)

15. Blood Pressure Thresholds

RECOMMENDATIONS

Level I and II

- There was insufficient evidence to support a Level I or II recommendation for this topic.

Level III

- Maintaining SBP at ≥100 mm Hg for patients 50 to 69 years old or at ≥110 mm Hg or above for patients 15 to 49 or over 70 years old may be considered to decrease mortality and improve outcomes.

16. Intracranial Pressure Thresholds

RECOMMENDATIONS*

Level I and II A

- There was insufficient evidence to support a Level I or II A recommendation for this topic.

Level II B

- Treating ICP above 22 mm Hg is recommended because values above this level are associated with increased mortality.

Level III

- A combination of ICP values and clinical and brain CT findings may be used to make management decisions.

17. Cerebral Perfusion Pressure Thresholds

RECOMMENDATIONS

Level I and II A

- There was insufficient evidence to support a Level I or II A recommendation for this topic.
- ***Level II B***
- The recommended target cerebral perfusion pressure (CPP) value for survival and favorable outcomes is between 60 and 70 mm Hg. Whether 60 or 70 mm Hg is the minimum optimal CPP threshold is unclear and may depend upon the patient’ s autoregulatory status.
- ***Level III***
- Avoiding aggressive attempts to maintain CPP above 70 mm Hg with fluids and pressors may be considered because of the risk of adult respiratory failure.

18. Advanced Cerebral Monitoring Thresholds

RECOMMENDATIONS

Level I and II

- There was insufficient evidence to support Level I or II recommendation for this topic.

Level III

- Jugular venous saturation of <50% may be a threshold to avoid in order to reduce mortality and improve outcomes.

Targeted Temperature Mangement for TBI

Lavinio *et al. Critical Care* (2024) 28:170
https://doi.org/10.1186/s13054-024-04951-x

Critical Care

GUIDELINE Open Access

Targeted temperature control following traumatic brain injury: ESICM/NACCS best practice consensus recommendations

Andrea Lavinio[1,2*], Jonathan P. Coles[1,2], Chiara Robba[3], Marcel Aries[4,5], Pierre Bouzat[6], Dara Chean[7], Shirin Frisvold[8,9], Laura Galarza[10], Raimund Helbok[11,12], Jeroen Hermanides[13], Mathieu van der Jagt[14], David K. Menon[1,2], Geert Meyfroidt[15], Jean-Francois Payen[6], Daniele Poole[16], Frank Rasulo[17], Jonathan Rhodes[18], Emily Sidlow[19], Luzius A. Steiner[20], Fabio Silvio Taccone[21,22] and Riikka Takala[23,24]

Tier Zero (not ICP dependent): Treat core temperature >38.0°C

- Sedation, endotracheal intubation and mechanical ventilation
- CPP >60 mmHg
- SpO_2 >94% and Hb >7g/dL
- Consider EEG monitoring and seizure prophylaxis, avoid hyponatraemia

Tier 1: Controlled normothermia (target core temperature 36.0–37.5°C)

- Titrate sedation and analgesia to control ICP
- CPP 60–70 mmHg
- $PaCO_2$ 35–38 mmHg / 4.7–5.1 kPa
- Consider osmotherapy and external ventricular drainage

Tier 2: Controlled normothermia (target core temperature 36.0–37.5°C)

- CPP individualised goals
- $PaCO_2$ 32–35 mmHg / 4.3–4.6 kPa
- Consider neuromuscular blocker

Tier 3: Mild hypothermia (target core temperature 35.0–36.0°C)

- Consider decompressive craniectomy
- Consider barbiturate coma

Fig. 2 Intracranial pressure management algorithm for severe TBI edited from SIBICC 2019 [28]. * Including TTC in tiers 1 and 2 is the suggested addition from the TTC-TBI group to the original SIBICC tiers (green bars). *When possible, the lowest tier should be used. It is not necessary to use all modalities in a previous tier before moving to the next tier. Consider repeat CT and surgical options for space occupying lesions. *CPP* cerebral perfusion pressure, *CT* computed tomography, *EEG* electroencephalography, *Hb* haemoglobin, *kPa* kilopascal, *mmHg* milimetre of mercury, $PaCO_2$ arterial partial pressure of carbon dioxide, SpO_2 arterial oxygen saturation

Targeted Temperature Mangement for TBI

Clinical term	Definition
Mild hypothermia	Core temperature 34.0–36.0 °C
Therapeutic hypothermia	Core temperature < 36.0 °C
Controlled normothermia	Core temperature 36.0–37.5 °C
Fever	Core temperature > 37.5 °C

Lavinio *et al. Critical Care* *(2024) 28:170*

Targeted Temperature Mangement for TBI

Recommendation	Level of consensus	Stage reached
Pathophysiology		
Temperature measurement and control is an essential aspect of high-quality care in patients with severe traumatic brain injury (TBI)	Strong consensus (100%)	Round 3
In patients with impending cerebral herniation, temperature control is essential	Strong consensus (89%)	Round 3
Monitoring		
Continuous temperature monitoring is preferable over intermittent temperature measurements in patients with severe TBI	Strong consensus (100%)	Round 1
Monitoring core temperature (e.g., bladder, oesophageal, brain) is strongly recommended over measuring or monitoring superficial temperature (e.g., skin, tympanic) in severe TBI	Strong consensus (94%)	Round 3
Monitoring brain temperature is recommended in addition to monitoring core systemic temperature as a therapeutic target	*No consensus (61%)*	
When brain temperature monitoring is not immediately available, alternative sources of core temperature (oesophageal, bladder, intravascular) are acceptable	Strong consensus (89%)	Round 3
When brain temperature monitoring is in place, it is advisable to also assess core temperature	Strong consensus (100%)	Round 3

Targeted Temperature Mangement for TBI

Pathophysiology

1. Temperature measurement and control is an essential aspect of high-quality care in patients with severe TBI.
2. In patients with impending cerebral herniation, temperature control is essential.

Targeted Temperature Mangement for TBI

Monitoring

1. Continuous temperature monitoring is preferable over intermittent temperature measurements in patients with severe TBI.
2. Monitoring core temperature (e.g., bladder, oesophageal, brain) is strongly recommended over measuring or monitoring superficial temperature (e.g., skin, tympanic) in severe TBI.
3. When brain temperature monitoring is in place, it is advisable to assess an additional source of core temperature monitoring (i.e. oesophageal, bladder).

Targeted Temperature Mangement for TBI

ICP		
Temperature control is a key component of intracranial pressure (ICP) management in severe TBI cases	Strong consensus (100%)	Round 1
Controlled normothermia (i.e., target core temperature 36.0–37.5 °C) should be included as an addition to the Tier 1 and Tier 2 treatments defined within the SIBICC 2019 guidelines	Moderate consensus (83%)	Round 3
Therapeutic hypothermia (i.e., target core temperature ≤ 36.0 °C) should be considered in cases where tier 1 and 2 treatments (as per SIBICC guidance) have failed to control ICP	Moderate consensus (83%)	Round 3
If hypothermia is considered to control ICP, target temperature should be managed as close to physiological temperature as possible	Strong consensus (94%)	Round 3
In patients with impending brain herniation, therapeutic hypothermia should be considered as a temporising strategy, and should be induced rapidly	*No consensus (61%)*	
In patients with impending herniation awaiting surgical evacuation or decompression, the lowest target core temperature at which hypothermia should be initiated as a short-term temporising strategy is	*No consensus*[a] (61%—35.0 °C; 17%—34.0 °C; 6%—33.0 °C; 17%—N/A)	Round 3
In patients with exhausted intracranial volume buffering reserve and labile ICP with occasional spikes > 25 mmHg, the lowest target core temperature that a medium term ICP-control strategy should be implemented at is	*No consensus*[a] (56%—35.0 °C; 33%—34.0 °C; 6%—33.0 °C; 6%—N/A)	Round 3
In tier 3 treatment in SIBICC guidelines, before considering decompressive craniectomy, hypothermia (< 36.0 °C) should be attempted	*No consensus (44%)*	
Before considering barbiturate burst suppression, hypothermia (< 36.0 °C) should be attempted	*No consensus (61%)*	

Targeted Temperature Mangement for TBI
ICP Mangement

1. Temperature control is a key component of ICP management in severe TBI.
2. Controlled normothermia (i.e., target core temperature 36.0–37.5 °C) should be included as an addition to the Tier 1 and Tier 2 treatments defined within the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC) 2019 guidelines.
3. Therapeutic hypothermia (i.e., target core temperature ≤ 36.0 °C) should be considered in cases where tier 1 and 2 treatments (as per SIBICC guidance) have failed to control ICP.
4. If hypothermia is considered to control ICP, target temperature should be managed as close to physiological temperature (normothermia) as possible.

Targeted Temperature Mangement for TBI

Fever		
Uncontrolled fever (neurogenic or secondary to inflammation or infection) can precipitate secondary brain injury in patients with severe TBI	Strong consensus (100%)	Round 3
Fever control is recommended in patients with severe TBI who have seizures or are perceived to be at high risk of seizures	Strong consensus (94%)	Round 3
Fever increases the risk of intracranial hypertension in patients with severe TBI	Strong consensus (94%)	Round 3
Neurogenic fever (core temperature > 37.5 °C driven by neurological dysregulation in the absence of sepsis or clinically significant inflammatory process) is relatively common in traumatic brain injury cases, and it should be promptly detected and treated (i.e., with controlled normothermia targeting 36.0–37.5 °C), irrespective of ICP	Moderate consensus (83%)	Round 3
Controlled normothermia should be considered when pyrexia is secondary to sepsis or inflammatory processes, and when the patient is perceived to be at risk of secondary brain injury, especially in the acute phase of TBI	Strong consensus (94%)	Round 3
In patients with severe TBI who are sedated and ventilated, controlled normothermia, irrespective of ICP, should be initiated reactively when fever is detected	Strong consensus (94%)	Round 3
When neurogenic fever is detected in TBI cases, controlled normothermia should be continued for as long as the brain remains at risk of secondary brain damage	Strong consensus (89%)	Round 3

Targeted Temperature Mangement for TBI
Fever Control

1. Uncontrolled fever (neurogenic or secondary to inflammation or infection) can precipitate secondary brain injury in patients with severe TBI.
2. Fever control is recommended in patients with severe TBI who have seizures or are perceived to be at high risk of seizures.
3. Fever increases the risk of intracranial hypertension in patients with severe TBI.
4. Neurogenic fever (core temperature>37.5 °C) driven by neurological dysregulation in the absence of sepsis or a clinically significant systemic inflammatory process is relatively common in TBI, and it should be promptly detected and treated (i.e., with controlled normothermia targeting 36.0 °C to 37.5 °C), irrespective of ICP level.
5. Controlled normothermia should be considered when pyrexia is secondary to sepsis or inflammatory processes, and when the patient is perceived to be at risk of secondary brain injury, especially in the acute phase of TBI.

Targeted Temperature Mangement for TBI

Hypothermic TTC induction		
It is recommended that the rapid induction of hypothermia in TBI cases should be achieved with automated feedback-controlled temperature management devices	Strong consensus (89%)	Round 3
It is advisable that neurotrauma ICUs should stock readily available NaCl solutions of different concentrations stored at ice-cold temperature for the management of intracranial hypertension crises	No consensus *(50%)*	
TTC maintenance		
An automated feedback-controlled TTC device that enables precise temperature control is desirable for the initiation of TTC and maintenance at target temperature in patients with severe TBI	Strong consensus (100%)	Round 1
The maximum temperature variation that a patient should experience during normothermia is less than or equal to ± 0.5 °C per hour and ≤ 1 °C per 24-h period	Moderate consensus (78%)	Round 3
When hypothermia is indicated, treatment should be continued for as long as the brain is considered to be at risk of secondary brain injury	Strong consensus (89%)	Round 3

Targeted Temperature Mangement for TBI
Hypothermic TTC induction

- It is recommended that the rapid induction of hypothermia in TBI should be achieved with automated feedback-controlled temperature management devices.

Targeted Temperature Mangement for TBI
TTC maintenance

1. An automated feedback-controlled TTC device that enables precise temperature control is desirable for the initiation of TTC and maintenance at target temperature in patients with severe TBI.
2. The maximum temperature variation that a patient should experience during normothermia is less than or equal to +/− 0.5 °C per hour and ≤1 °C per 24-h period
3. When hypothermia is indicated, treatment should be continued for as long as the brain is considered to be at risk of secondary brain injury.

Targeted Temperature Mangement for TBI

Rewarming following hypothermic TTC

Obtaining an interval scan and/or an alternative assessment of intracranial compliance, in addition to the absolute number of ICP, is recommended before rewarming	Strong consensus (89%)	Round 3
When rewarming a patient from therapeutic hypothermia, rewarming should be controlled by an automated feedback-controlled TTC device and should not exceed 1.0 °C per 24-h period	*No consensus (44%)*	
Rebound hyperthermia should be prevented whenever possible or promptly treated in cases when the brain is perceived to be at risk of secondary brain injury	Strong consensus (100%)	Round 3

Targeted Temperature Mangement for TBI
Rewarming

1. Obtaining an interval scan and/or an alternative assessment of intracranial compliance, in addition to the absolute number of ICP, is recommended before rewarming.
2. Rebound hyperthermia should be prevented whenever possible or promptly treated in cases when the brain is perceived to be at risk of secondary brain injury.

Targeted Temperature Mangement for TBI

Shivering		
It is important to assess, document and manage shivering in severe TBI patients	Strong consensus (100%)	Round 3
Whenever ICP is labile and shivering is detected, neuromuscular blockers should be considered after ensuring appropriate depth of sedation	Strong consensus (94%)	Round 3
In self-ventilating patients in the subacute phase of severe TBI, an individualised risk–benefit assessment should be undertaken regarding the indications of controlled normothermia	Strong consensus (100%)	Round 3
Permissive hyperthermia should be considered in cases where risk of secondary brain injury resulting from pyrexia is thought to be low, and when shivering cannot be controlled with first line treatments such as NSAIDs, opiates, magnesium or counter warming	Moderate consensus (83%)	Round 3
Auditing		
Time within target range, burden of fever and similar metrics can be considered as indicators of quality of temperature management	Strong consensus (94%)	Round 3

Targeted Temperature Mangement for TBI
Shivering

1. It is important to assess, document and manage shivering in severe TBI patients.
2. Whenever ICP is labile and shivering is detected, neuromuscular blockers should be considered after ensuring appropriate depth of sedation.
3. In self-ventilating patients in the subacute phase of severe TBI, an individualised risk–benefit assessment should be undertaken regarding the strict indications of controlled normothermia.
4. Permissive hyperthermia should be considered in cases where risk of secondary brain injury resulting from pyrexia is thought to be low, and when shivering cannot be controlled with first line treatments such as NSAIDs, opiates, magnesium or counter warming.

Targeted Temperature Mangement for TBI
Auditing

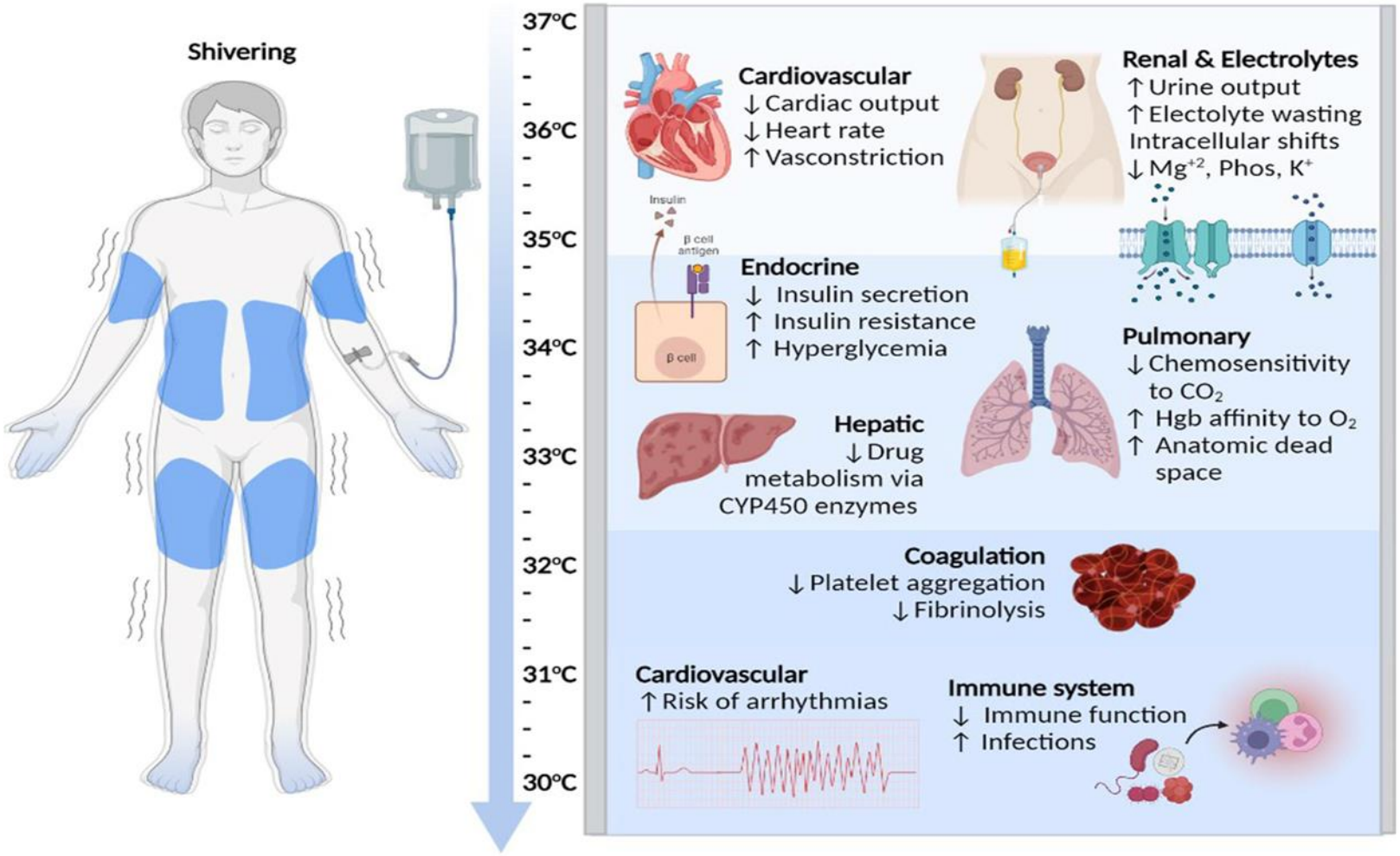
- ‘Time within target range’, ‘burden of fever’ and similar metrics can be considered as indicators of quality of temperature management.

Targeted Temperature Mangement for TBI
Overview of Recommendations

- Managing temperature is a vital aspect of care for TBI .
- It is crucial to control fever to prevent secondary brain injury and should be actively managed.
- The recommended target for controlled normothermia is 36.0 - 37.5 ˚C (Tier 1 & 2 treatment).
- Therapeutic hypothermia should only be considered with caution and as a last option.

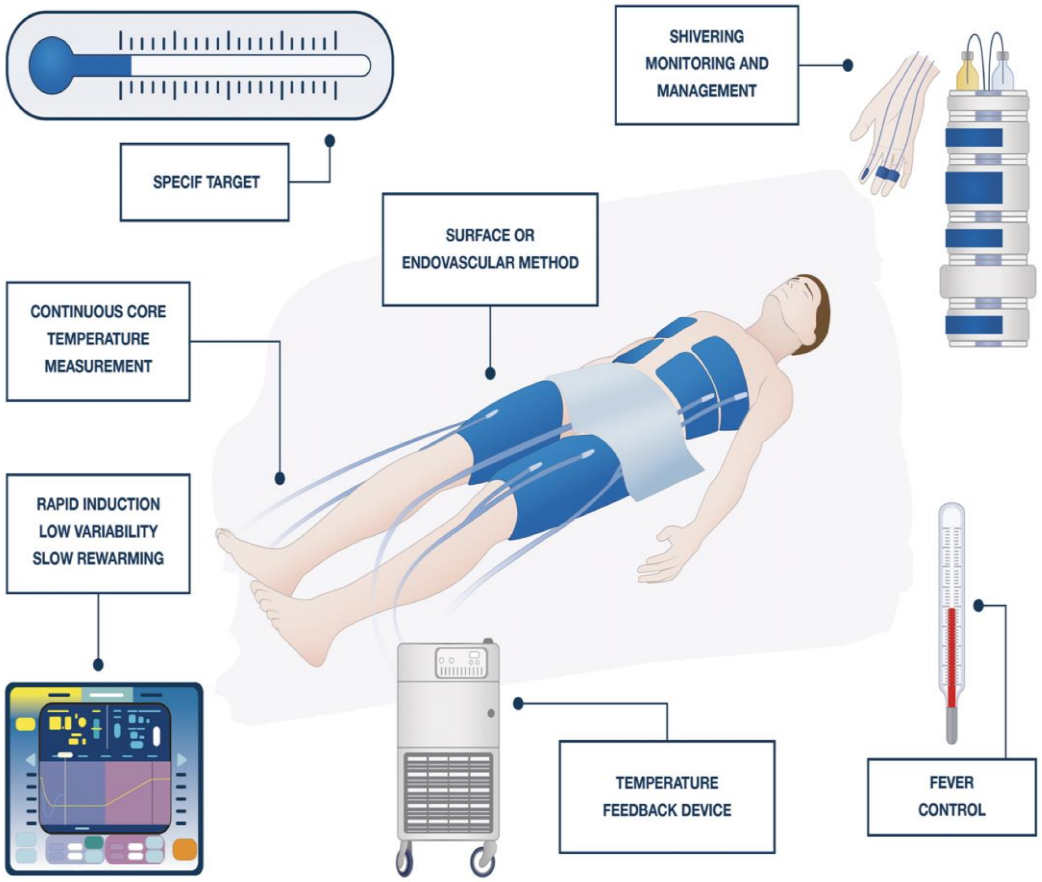
Complications of temperature control

Hernandez et al. Adv Ther (2023) 40:2097–2115



High Quality TTM

Taccone et al. Critical Care 2020 Jan 6;24:6.



- Rapid induction: initiate as soon as possible
- Specific target temperature: 33 °C or 36 °C
- Continuous core temperature measurement: bladder, esophagus, or a vessel
- Duration of the cooling phase: last at least 24 h.
- Slow rewarming: (0.15–0.25 °C/h)
- Low variability
- Temperature feedback system
- Fever control
- Shivering management

難治型顱內高壓之低溫療法: 健保支付標準

	難治型顱內高壓之低溫療法					
47081B	一首日		v	v	v	6057
47082B	一第二至七日(每日)		v	v	v	2505
	註：					
	1.適應症：因下列原因造成之顱內高壓，以傳統治療方式如：頭部抬高 30 度、降腦壓藥物、開顱手術、深度鎮靜且 ICP 仍處於 20mmHg>10 min 者，皆無法使顱內壓降低：					
	(1)出血性中風。					
	(2)次重度或重度腦創傷 GCS 小於等於 12。					

難治型顱內高壓之低溫療法: 健保支付標準

2. 禁忌症:

(1)任意原因引起之休克。

(2)在意外前即有失智或長期意識障礙。

(3)腦死。

(4)自發性低體溫者<32°C。

(5)顱內有占據顱內空間病灶如血塊、 腦水腫、水腦症等，符合手術適應症，但未進行手術者。

(6)嚴重感染者。

(7)呼吸窘迫症候群。

難治型顱內高壓之低溫療法: 健保支付標準

3. 支付規範:

(1)限 ICU 執行。

(2)同次住院，限申報「首日」一次，「第二至七日(每日)」至多六次。

(3)申報時需檢附生命徵象及藥物使用紀錄。

(4)不得同時申報:47037B、**47038B**、**47039B**、**47094B~**47096B 及 47097B**~**47100B。

4. 限神經科、神經外科、兒科專科醫師執行。

Shivering

Stroke
Volume 39, Issue 12, 1 December 2008; Pages 3242-3247
https://doi.org/10.1161/STROKEAHA.108.523654

ORIGINAL CONTRIBUTIONS

Metabolic Impact of Shivering During Therapeutic Temperature Modulation

The Bedside Shivering Assessment Scale

Neeraj Badjatia, MD, MSc, Evangelia Strongilis, RD, Errol Gordon, MD, Mary Prescutti, RN, Luis Fernandez, MD, Andres Fernandez, MD, Manuel Buitrago, MD, PhD, J. Michael Schmidt, PhD, Noeleen D. Ostapkovich, MSc, and Stephan A. Mayer, MD, FCCM

Shivering
Bedside Shivering Assessment Scale (BSAS)

Score	Definition
0	None: no shivering noted on palpation of the masseter, neck, or chest wall
1	**Mild:** shivering localized to the neck and/or thorax only
2	**Moderate:** shivering involves gross movement of the upper extremities (in addition to neck and thorax)
3	**Severe:** shivering involves gross movements of the trunk and upper and lower extremities

Shivering

Review Article

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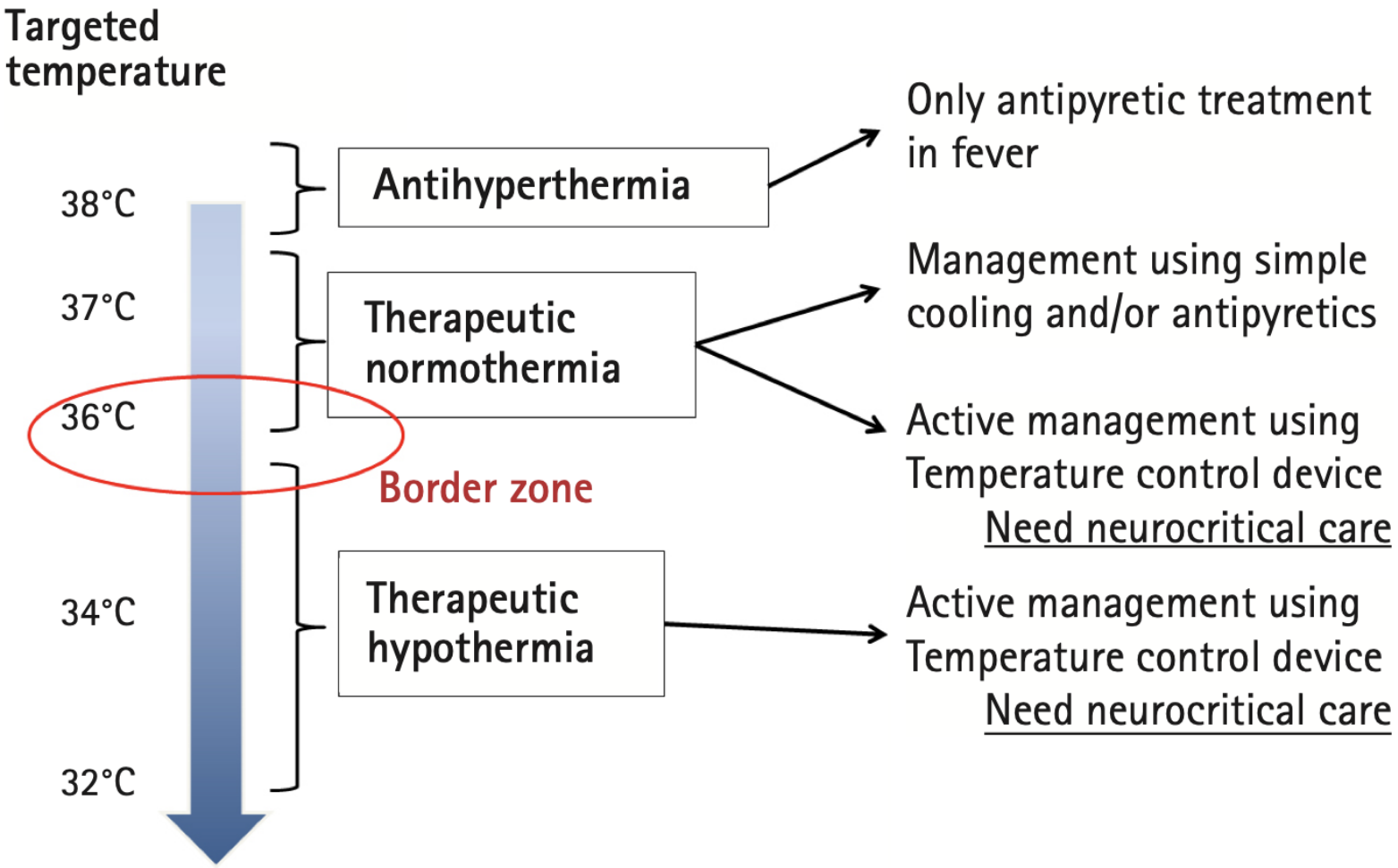
Targeted temperature management for postcardiac arrest syndrome

Yasuhiro Kuroda, MD[1], Kenya Kawakita, MD[2]

[1]Department of Emergency, Disaster, and Critical Care Medicine, Faculty of Medicine, Kagawa University, Kita, Japan

[2]Emergency Medical Center, Kagawa University Hospital, Faculty of Medicine, Kagawa University, Kita, Japan

Shivering
Definition of TTM



Shivering

TEMPERATURE SELECTION: CONSIDERING 36°C

- Therapeutic hypothermia (TTM 32°C to 34°C) is more effective in preventing secondary brain injury than TTM 36°C, but it may lead to coagulopathy and increased bleeding.
- In cases of surgical bleeding, intracranial hemorrhage, hemorrhagic diathesis, or trauma, TTM 36°C is advisable, as it typically does not induce coagulopathy.
- Regarding circulatory management, TTM 32°C to 34°C can cause greater hemodynamic instability compared to TTM 36°C, potentially necessitating vasopressor support.

Shivering
Complications and Warning Signs

- Shivering acts as a physiological homeostatic response to help regulate body temperature, typically beginning at around 36°C.
- At TTM of 36°C, shivering is severe due to the patient's thermoregulatory mechanisms being somewhat diminished at temperatures between 32°C and 33°C.
- Prolonged shivering leads to an elevated metabolic rate and cardiac output, resulting in tachycardia, increased blood pressure, increased lethal cardiac complications, greater CO2 production, wound pain, elevated CMRo2 and ICP, and increased stress response.
- Warning signs of shivering include goosebumps, palpation of the masseter muscle, artifacts in electrocardiograms, difficulty in cooling, and a rise in temperature despite TTM .

Shivering Control

To suppress shivering effectively, a combination of techniques should be employed, and it is important to control shivering with determination.

Remifentanil 6-60 μg/kg/hr
or fentanyl 0.7-1.0 μg/kg/hr
and if necessary add
dexmedetomidine 0.2-0.7 μg/kg/hr
meperidine 0.5 mg/kg every 6 hr

Propofol 0.5-3.0 mg/kg/hr
or midazolam 0.03-0.2 mg/kg/hr