

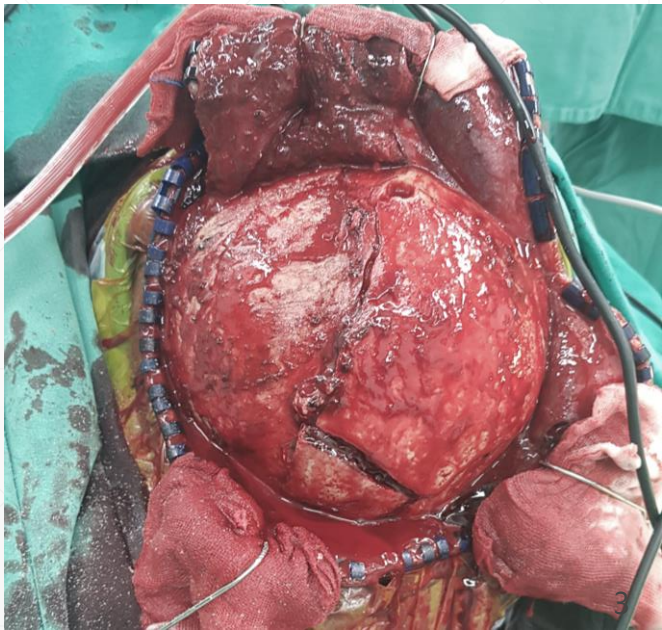
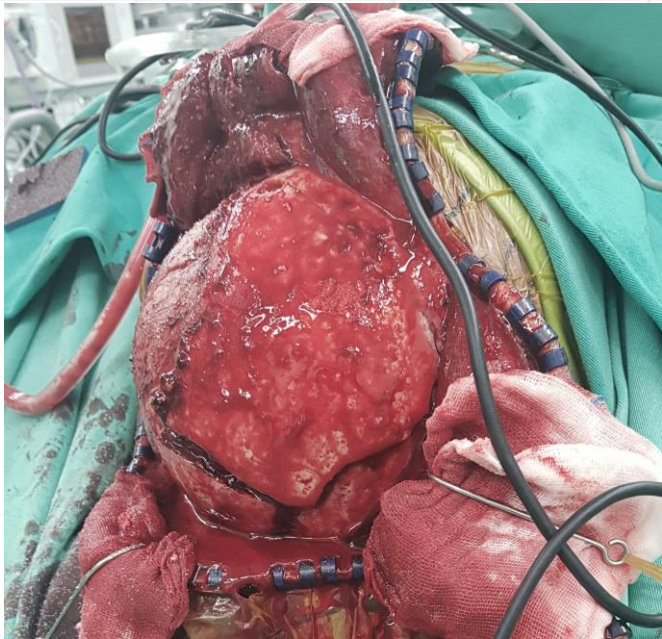
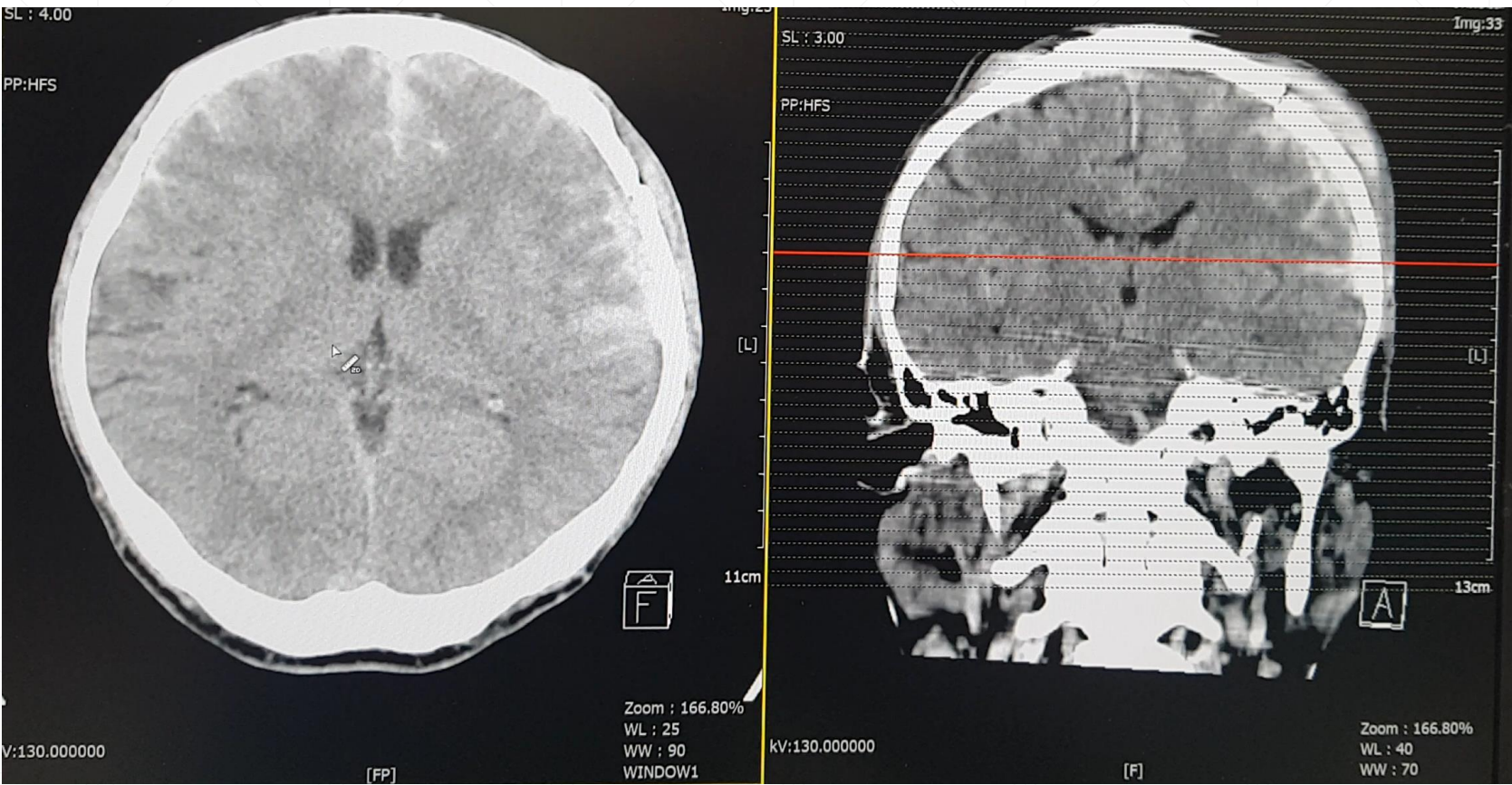
An Update in Traumatic Brain Injury

神經醫學中心 神經重症科 葉美吟

Traumatic Brain Injury

創傷性腦損傷







台灣版 - 輕度及嚴重頭部外傷治療準則



台灣版 -- 輕度頭部外傷治療準則

輕度頭部外傷之定義與分類

輕度頭部外傷病人接受電腦斷層檢查準則

輕度頭部外傷病患住院照護

腦震盪症候群

台灣版 -- 嚴重頭部外傷治療準則

急診處置

腦灌流壓之原則

輸液之原則

顱內壓監測

鎮定劑之使用

營養

顱內壓上升之處置

預防性抗癲癇藥物之投予

二線療法

頭部外傷 (Traumatic Brain Injury, TBI) 之定義

本研究所使用之頭部外傷定義為「頭部受到外力的傷害，係長期沿用英文Head Injury 之翻譯名詞，可使用腦傷害、腦受傷、腦傷害...等」。

前言

意外事故在台灣仍然是很重要的死因，頭部外傷雖然在安全帽法實施後大量減少，然而嚴重頭部外傷發生後之死亡率仍然高達35%，如何使嚴重頭部外傷發生後得到最好的照顧實為當務之急。嚴重頭部外傷治療之進步，實有賴於國內神經外科專家學者共同努力建立台灣本土嚴重頭部外傷的重症治療共識(指引)。有關頭部外傷的治療理論最近二、三十年來有相當大的改變，由於對顱內壓升高的病理生理機制及腦部循環動力學的了解，相關的治療已有重大的突破。以往將治療的目標專注在處理顱內壓升高，例如：限水(fluid restriction)、高張利尿劑(mannitol、glycerol)、過度通氣(hyperventilation)及類固醇等使用均被重新再評估。主要的改變是因腦部監測(cerebral monitoring)技術的進步以及預防腦部缺血(ischemia)為概念的發展。目前腦部重症加護照顧的中心目標已經由顱內壓的控制轉為缺血的預防，因此，除了降低顱內壓之外，如何提升腦部的灌流(cerebral perfusion)及降低腦部代謝以避免缺血，已成為另一注意的焦點。

美國神經外科醫學會(American Association of Neurological surgeons)與腦外傷基金，於1995及2000年發表了嚴重頭部外傷處理之指引(Guidelines for the management of severe head injury)，我們將參考國外學者及台灣專家學者之意見，特別針對有關嚴重頭部外傷神經加護照顧當中重要的觀念，包括急診之處理原則、顱內壓監測、顱內壓升高的處理原則、腦灌流、過度通氣、癲癇大發作之預防、營養及二線療法等加以討論說明。

The Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC)



西雅圖國際嚴重創傷性腦損傷共識會議

- Delphi-method based
- 42 international, multidisciplinary neurotrauma experts
- Class III evidence
- Brain-specific parameter
 - Intracranial pressure
 - Brain tissue oxygen ($P_{bt}O_2$) monitoring
- BOOST-3 trial protocol
 - Brain Oxygen Optimization in Severe Traumatic Brain Injury



BMJ Open Brain Oxygen Optimization in Severe Traumatic Brain Injury (BOOST-3): a multicentre, randomised, blinded-endpoint, comparative effectiveness study of brain tissue oxygen and intracranial pressure monitoring versus intracranial pressure alone

Accepted 02 February 2022

Francis Bernard ^{1,2} William Barsan,³ Ramon Diaz-Arrastia,⁴ Lisa H Merck,⁵ Sharon Yeatts,⁶ Lori A Shutter ⁷

- Trial registration number: ClinicalTrials.gov Registry (NCT03754114)



BASIC CARE Applies to all Severe TBI Patients

TIER

0

Expected Interventions:

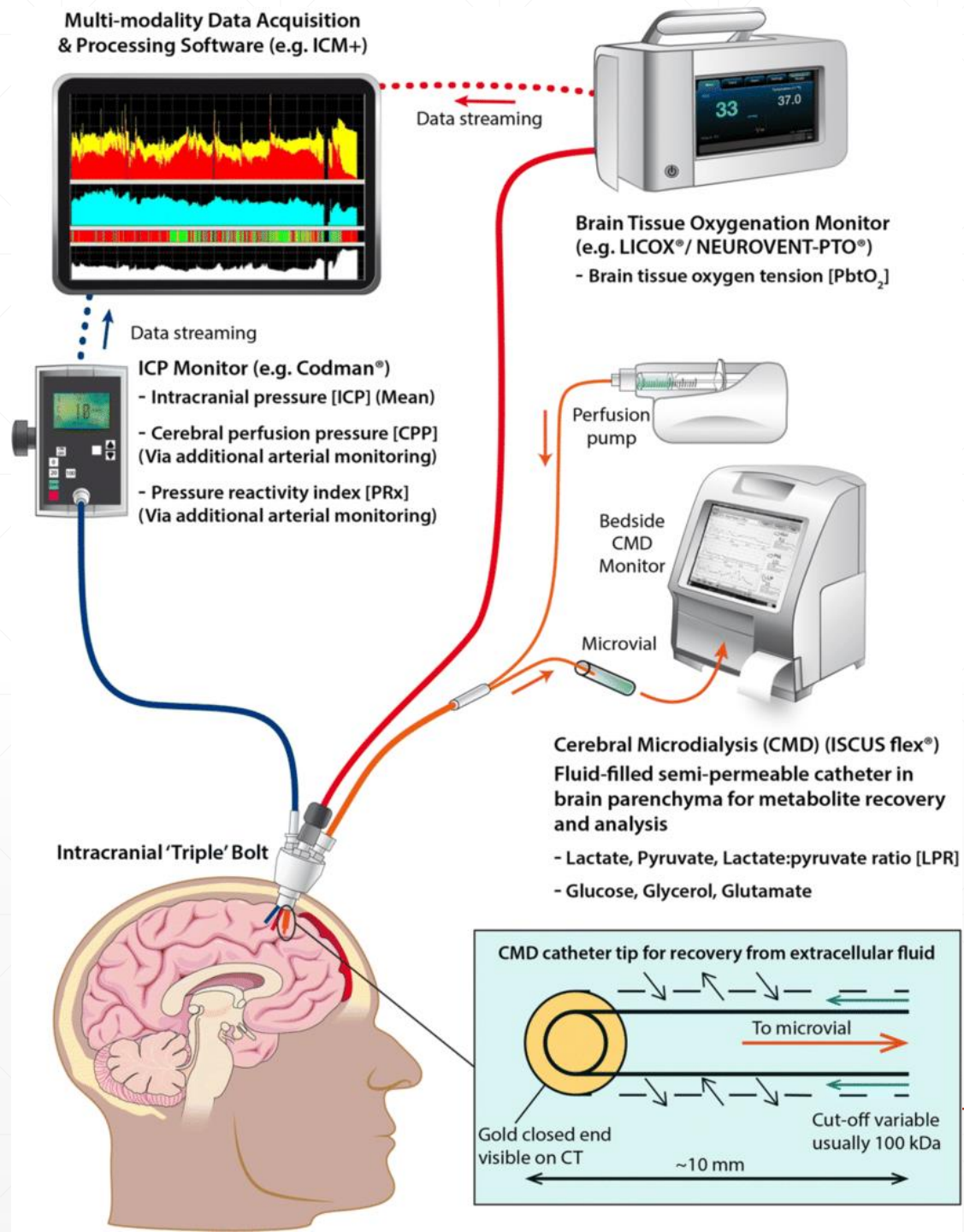
- Admission to ICU
- Endotracheal intubation and mechanical ventilation
- Serial evaluations of neurological status and pupillary reactivity
- Elevate HOB 30–45°
- Analgesia to manage signs of pain (not ICP directed)
- Sedation to prevent agitation, ventilator asynchrony, etc. (not ICP directed)

- 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 $\geq 60\text{mmHg}$
- Maintain Hb > 7g/dL
- Avoid hyponatremia

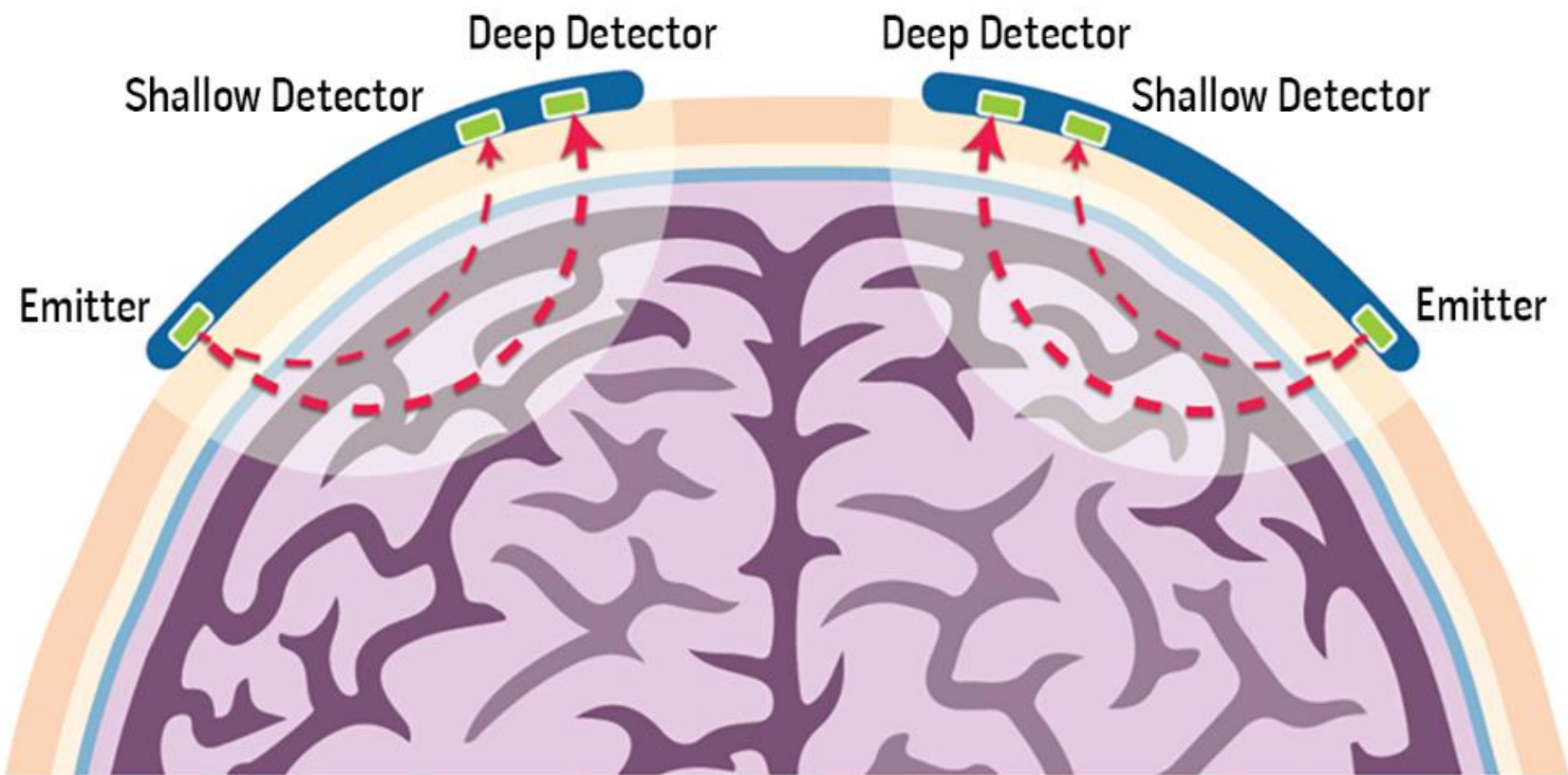
- Optimize venous return from head (e.g. head midline, ensure cervical collars are not too tight)
- Arterial line for continuous blood pressure monitoring
- Maintain $\text{SpO}_2 \geq 94\%$

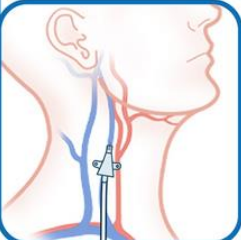
Recommended Interventions:


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



	ICP < 22 mmHg	ICP > 22 mmHg
$P_{btO_2} > 20$ mmHg	Type A	Type B
$P_{btO_2} < 20$ mmHg	Type C	Type D

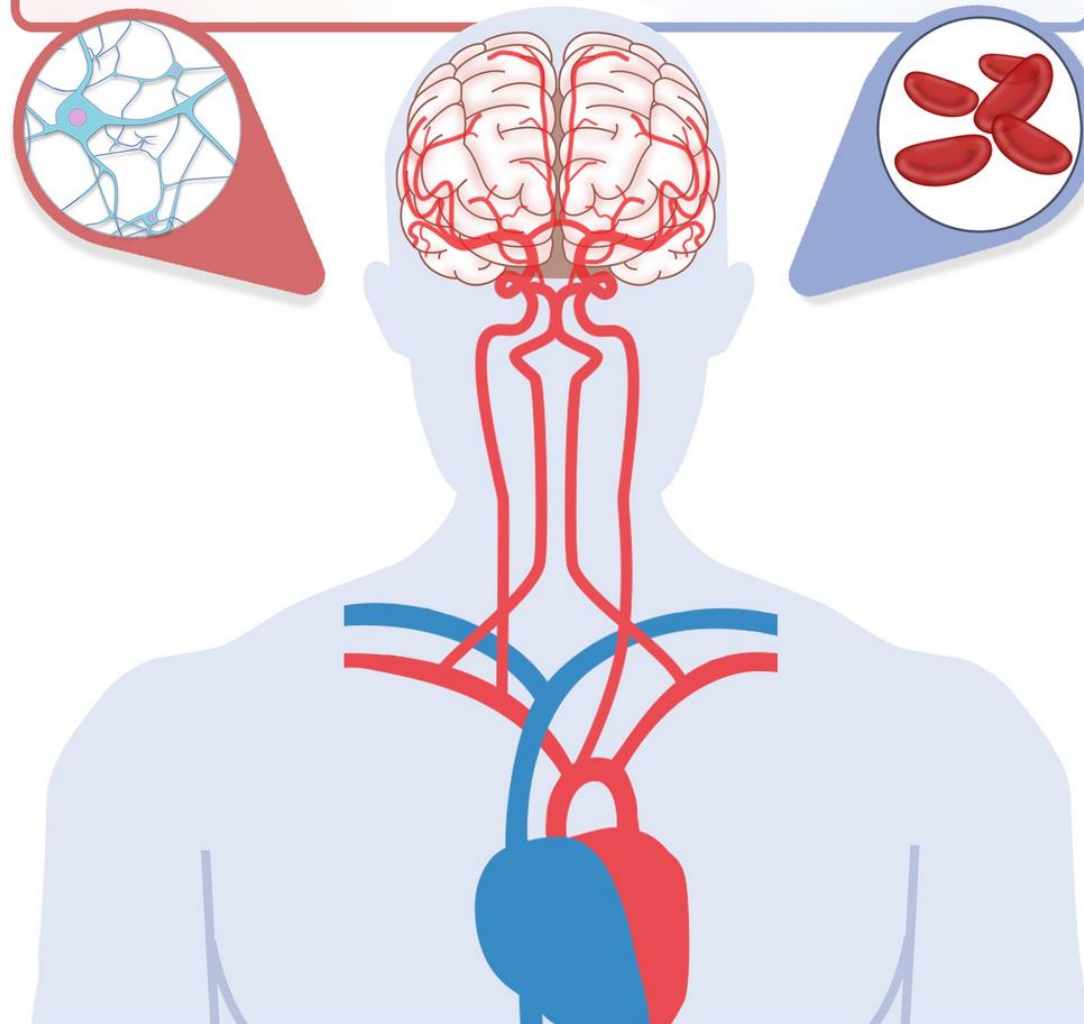


	Pros	Pitfalls
Jugular Bulb Oximetry 	<ul style="list-style-type: none"> • Global oxygen assessment • Reflects dynamic balance of brain oxygen supply and consumption 	<ul style="list-style-type: none"> • Invasive • Not focal • Risk of vein thrombosis • Possible extracranial contamination • Intermittent (if no fiber optic probes)

Cerebral Oximetry (NIRS) 	<ul style="list-style-type: none"> • Noninvasive • Safe • Minimal expertise needed • Low cost • Global tissue oxygen assessment 	<ul style="list-style-type: none"> • Extra-cranial contamination • Lack of precision as absolute number and changes over time • Contusions or blood in the explored area interfere with the signal
---	--	---

Brain Tissue Oxygen Probe 	<ul style="list-style-type: none"> • No extra-cranial contamination • Good temporal resolution 	<ul style="list-style-type: none"> • Invasive • Low spatial resolution • Higher cost
--	--	---

Cause of cerebral hypoxia	Treatment
↓ CBF, ↓ CPP	↑ ABP, ↑ PaCO ₂
↓ PaO ₂	↑ FiO ₂
↓ Hb	Consider transfusion
↑ Metabolism	↑ Sedation, ↓ Brain temperature



Type B

ICP Elevated - Brain Oxygenation Normal

Tier 1

- Maintain CPP 60-70 mm Hg
- Increase analgesia to lower ICP
- Increase sedation to lower ICP
- Maintain P_aCO_2 at low end of normal (35-38 mm Hg/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

Tier 2

- Mild hypocapnia (range 32-35 mmHg/4.3-4.7 kPa)
- Neuromuscular paralysis in adequately sedated patients if efficacious in lowering ICP**
- **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

Tier 3

- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious‡
- 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

Type B

ICP Elevated - Brain Oxygenation Normal

Tier 1

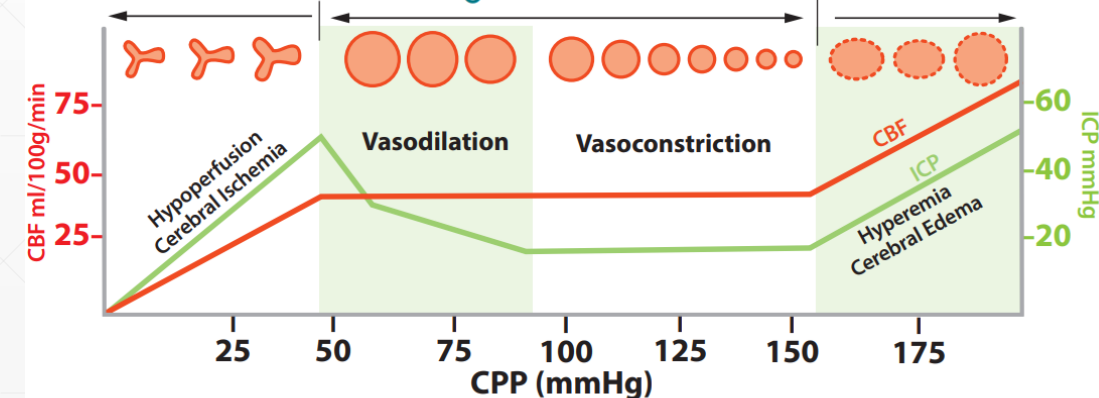
- Maintain CPP 60-70 mm Hg
- Increase analgesia to lower ICP
- Increase sedation to lower ICP
- Maintain P_aCO_2 at low end of normal (35-38 mm Hg/4.7-5.1 kPa)
- Mannitol by intermittent bolus (0.25-1.0 g/kg)
- Hypertonic saline by intermittent bolus*
- CSF drainage if EVD *in situ*
- Consider placement 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

Tier 2

- Mild hypocapnia (range 32-35 mmHg/4.3-4.7 kPa)
- Neuromuscular paralysis in adequately sedated patients if efficacious in lowering ICP**
- **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

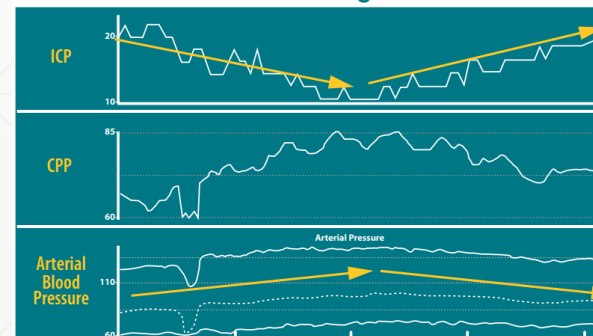
- Re-examine the patient and consider repeat CT to re-evaluate intracranial pathology
- Reconsider surgical options for potentially surgical lesions
- Consider extracranial causes of ICP elevation
- Review that basic physiologic parameters are in desired range (e.g. CPP, blood gas values)
- Consider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

Autoregulation Curve

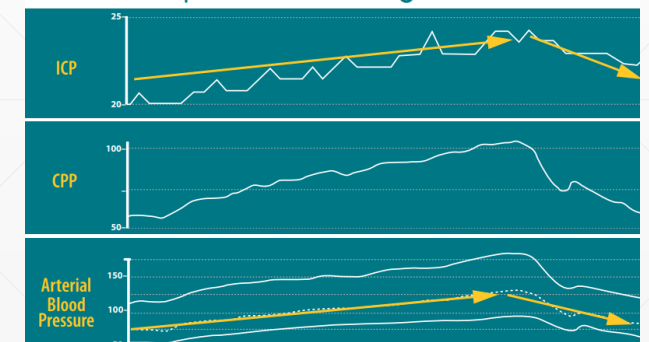


Lassen NA (1959). Cerebral blood flow and oxygen consumption in man. *Physiol Rev.* 39, 183-238.

Intact Autoregulation



Impaired Autoregulation



Tier 3



- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious†
- Secondary decompressive craniectomy
- Mild hypothermia (35-36°C) using active cooling measures
- Hyperventilation to $P_a\text{CO}_2$ of 30-32 mmHg/4.0-4.3 kPa

Type C

ICP Normal - Brain Hypoxic

Tier 1

- Maintain CPP 60-70 mm Hg
- Increase CPP to a maximum of 70 mmHg with fluid, vasopressors and/or inotropes
- Maintain $P_aCO_2 > (35 \text{ mmHg}/4.7 \text{ kPa})$
- 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/20kPa
- Decrease ICP to a threshold $< 22 \text{ mmHg}$
- Consider CSF drainage
- Increase sedation to improve mechanical ventilation and $P_{bt}O_2$
- Neuromuscular paralysis in adequately sedated patients if efficacious in increasing $P_{bt}O_2$ *
- 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 to increase $P_{bt}O_2$ when supported by MAP Challenge
- Increase CPP above 70 mmHg with fluid boluses, vasopressors and/or inotropes **

- 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

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 $< 20 \text{ mmHg}$ despite P_aO_2 and CPP/MAP optimization, consider transfusing 1 unit of PRBCs if Hgb $< 9 \text{ g/L}$

Type C

ICP Normal - Brain Hypoxic

Tier 1

- Maintain CPP 60-70 mm Hg
- Increase CPP to a maximum of 70 mmHg with fluid, vasopressors and/or inotropes
- Maintain $P_aCO_2 > (35 \text{ mmHg}/4.7 \text{ kPa})$
- If P_aO_2 is already in desired ranged, 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/20kPa
- 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 $P_{bt}O_2$ *
- 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 to increase $P_{bt}O_2$ when supported by MAP Challenge
- Increase CPP above 70 mmHg with fluid boluses, vasopressors and/or inotropes **

- 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



Tier 3

- Increase $P_a\text{CO}_2$ to 45-50 mmHg/6.0-6.7 kPa (but avoid intracranial hypertension)
- Consider normobaric hyperoxia to a $P_a\text{O}_2$ above 150 mmHg/20 kPa
- If $P_{bt}\text{O}_2$ remains < 20 mmHg despite $P_a\text{O}_2$ and CPP/MAP optimization, consider transfusing 1 unit of PRBCs if Hgb < 9g/L

ICP Elevated - Brain Hypoxic

Tier 1

- Maintain CPP 60-70 mm Hg
- Increase CPP to a maximum of 70 mmHg with fluid, vasopressors and/or inotropes
- Increase analgesia to lower ICP/improve ventilation and P_{btO_2}
- Increase sedation to lower ICP/improve ventilation and P_{btO_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*
- 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 range, 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/20kPa
- Increase sedation to improve ICP and P_{btO_2}
- Neuromuscular paralysis in adequately sedated patients if efficacious in decreasing ICP or increasing P_{btO_2} *
- **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_{btO_2}) before during and after the challenge
 - Adjust vasopressor/inotrope dose based on study findings
- Raise CPP to decrease ICP and/or increase P_{btO_2} when supported by MAP Challenge
- Increase CPP above 70 mmHg with fluid boluses, vasopressors and/or inotropes ***

- 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

Tier 3

- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious‡
- Secondary decompressive craniectomy
- Consider normobaric hyperoxia to a P_aO_2 above 150 mmHg/20 kPa
- If P_{btO_2} remains < 20 mmHg despite P_aO_2 and CPP/MAP optimization, consider transfusing 1 unit of PRBC if Hgb < 9 g/L

Type D

ICP Elevated - Brain Hypoxic

Tier 1

- Maintain CPP 60-70 mm Hg
- 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*
- 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 range, 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/20kPa
- Increase sedation to improve ICP and $P_{bt}O_2$
- Neuromuscular paralysis in adequately sedated patients if efficacious in decreasing ICP or increasing $P_{bt}O_2$ *
- **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 to decrease ICP and/or increase $P_{bt}O_2$ when supported by MAP Challenge
- Increase CPP above 70 mmHg with fluid boluses, vasopressors and/or inotropes ***

- 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

Tier 3



- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious†
- 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 PRBC if Hgb < 9g/L

Guidelines for the Management of Severe Traumatic Brain Injury

4th Edition



BRAIN TRAUMA FOUNDATION TBI GUIDELINES

**Guidelines for the Management of Severe Traumatic
Brain Injury: 2020 Update of the Decompressive
Craniectomy Recommendations**



2017

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.

2020

Level IIA—to improve mortality and overall outcomes

1. NEW—Secondary DC performed for *late* refractory ICP elevation is recommended to improve mortality and favorable outcomes.
2. NEW—Secondary DC performed for *early* refractory ICP elevation is not recommended to improve mortality and favorable outcomes†.
3. A large frontotemporoparietal DC (not less than 12 × 15 cm or 15 cm in diameter) is recommended over a small frontotemporoparietal DC for reduced mortality and improved neurological outcomes in patients with severe TBI.
4. NEW—Secondary DC, performed as a treatment for either early or late refractory ICP elevation, is suggested to reduce ICP and duration of intensive care, though the relationship between these effects and favorable outcome is uncertain.

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

Benchmark Evidence from South American Trials: Treatment of Intracranial Pressure (BEST:TRIP) trial

The NEW ENGLAND JOURNAL *of* MEDICINE

ESTABLISHED IN 1812

DECEMBER 27, 2012

VOL. 367 NO. 26

A Trial of Intracranial-Pressure Monitoring in Traumatic Brain Injury

Randall M. Chesnut, M.D., Nancy Temkin, Ph.D., Nancy Carney, Ph.D., Sureyya Dikmen, Ph.D., Carlos Rondina, M.D.,
Walter Videtta, M.D., Gustavo Petroni, M.D., Silvia Lujan, M.D., Jim Pridgeon, M.H.A., Jason Barber, M.S.,
Joan Machamer, M.A., Kelley Chaddock, B.A., Juanita M. Celix, M.D., Marianna Cherner, Ph.D., and Terence Hendrix, B.A.,
for the Global Neurotrauma Research Group*

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
 - Hyperventilation¹
 - Bolus of hypertonic solution
- Consider emergent imaging or other testing
- Rapid escalation of treatment

¹The 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

NARRATIVE REVIEW



Management of moderate to severe traumatic brain injury: an update for the intensivist

Intensive Care Med 48, 649–666 (2022)

Geert Meyfroidt^{1*}, Pierre Bouzat², Michael P. Casaer¹, Randall Chesnut³, Sophie Rym Hamada⁴,
Raimund Helbok⁵, Peter Hutchinson⁶, Andrew I. R. Maas⁷, Geoffrey Manley⁸, David K. Menon⁹,
Virginia F. J. Newcombe⁹, Mauro Oddo¹⁰, Chiara Robba¹¹, Lori Shutter¹², Martin Smith¹³,
Ewout W. Steyerberg¹⁴, Nino Stocchetti¹⁵, Fabio Silvio Taccone¹⁶, Lindsay Wilson¹⁷, Elisa R. Zanier¹⁸,
and Giuseppe Citerio^{19,20}

Table 1 Initial resuscitation targets

Parameter	Values/targets	Objectives
Blood pressure	MAP > 80 mmHg SBP > 100 or 110 mmHg	Preserving CBF
SpO ₂	> 90%	Avoiding brain hypoxia
EtCO ₂	30–35 mmHg	Preserving CBF
Hb	> 7 g/dl	Avoiding brain hypoxia
Anticoagulant	Reversal	Limiting blood loss and expansion of hemorrhagic contusions

Table 2 Proposed target values for some neuromonitoring modalities

	Normal	Desirable	Critical
ICP	~ 10 mmHg	< 18–22 mmHg	> 25 mmHg
CPP	50–60 mmHg	60- (80) mmHg	< 50 mmHg
PbtO ₂	~ 30 mmHg	20–25 mmHg	< 15 mmHg
Lactate/Pyruvate Ratio	< 25	< 25	> 40
Brain Glucose	> 1 mmol/l	> 0.8 mmol/l	< 0.5 mmol/l
Brain temperature	~ 36.5 °C	36.5–37 °C	> 37.5 °C

Table 4 Management of severe TBI: conceptual highlights**Initial management**

Initial pre-and in-hospital resuscitation Avoid and treat hypotension, hypoxia, anemia

Secondary injury management

Management of elevated ICP ICP monitoring allows to titrate therapy to severity of intracranial hypertension in severe TBI patients
SIBICC algorithms provide a conceptual framework for a tiered approach
Treating TBI involves more than just treating elevated ICP

Management of CPP Optimizing brain perfusion can be challenging, and ancillary monitoring of brain tissue oxygen or cerebrovascular autoregulation may be helpful

Multimodality monitoring Should be applied to answer a specific pathophysiological question

Extracranial complications

Respiratory management Lung protective ventilation is the preferred strategy
Avoid hypoxia, hyperoxia, hypocapnia, hypercapnia

Fluid management Assessment of volume status like general critically ill patients
Choice of optimal hypertonic solution still uncertain

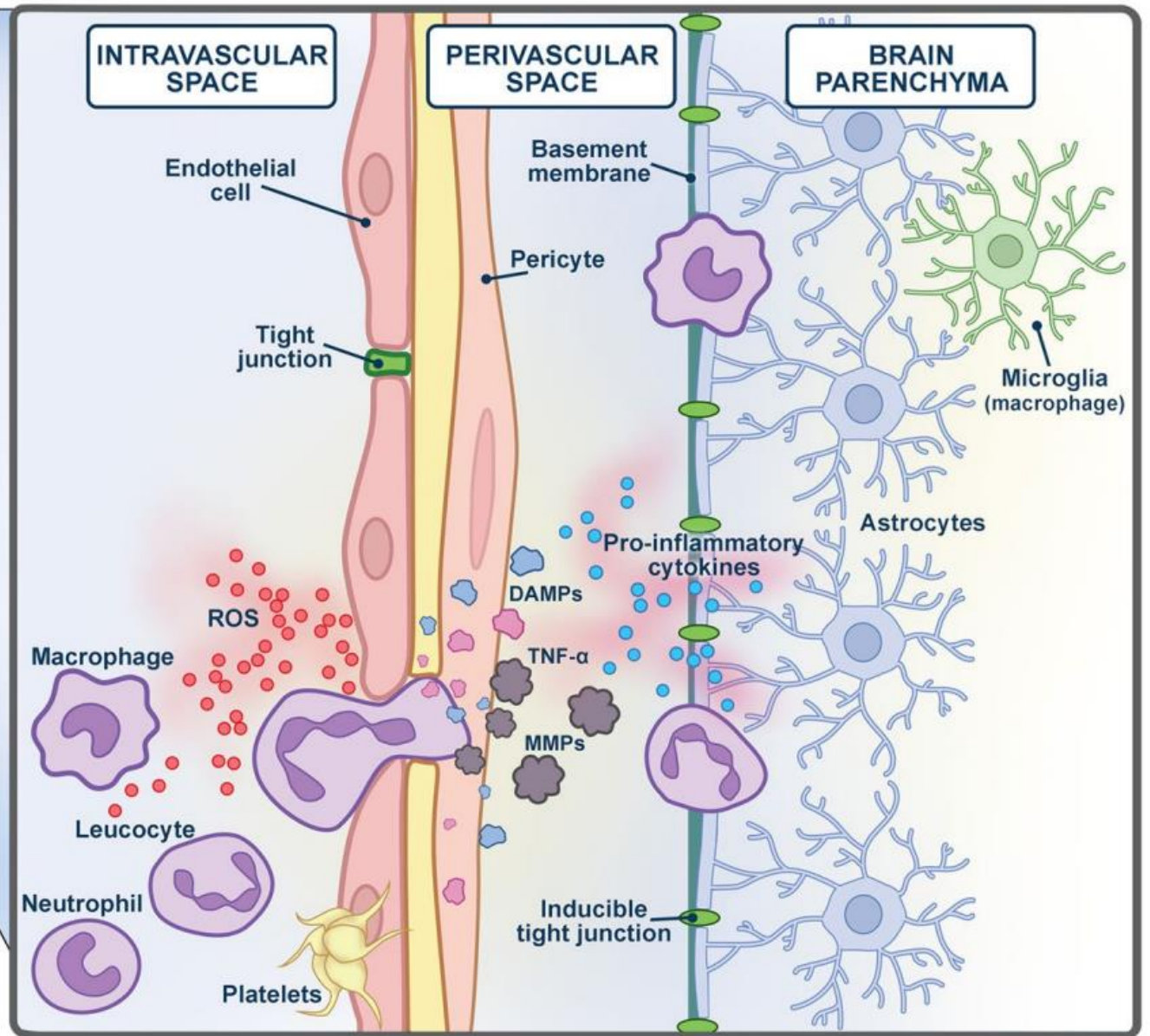
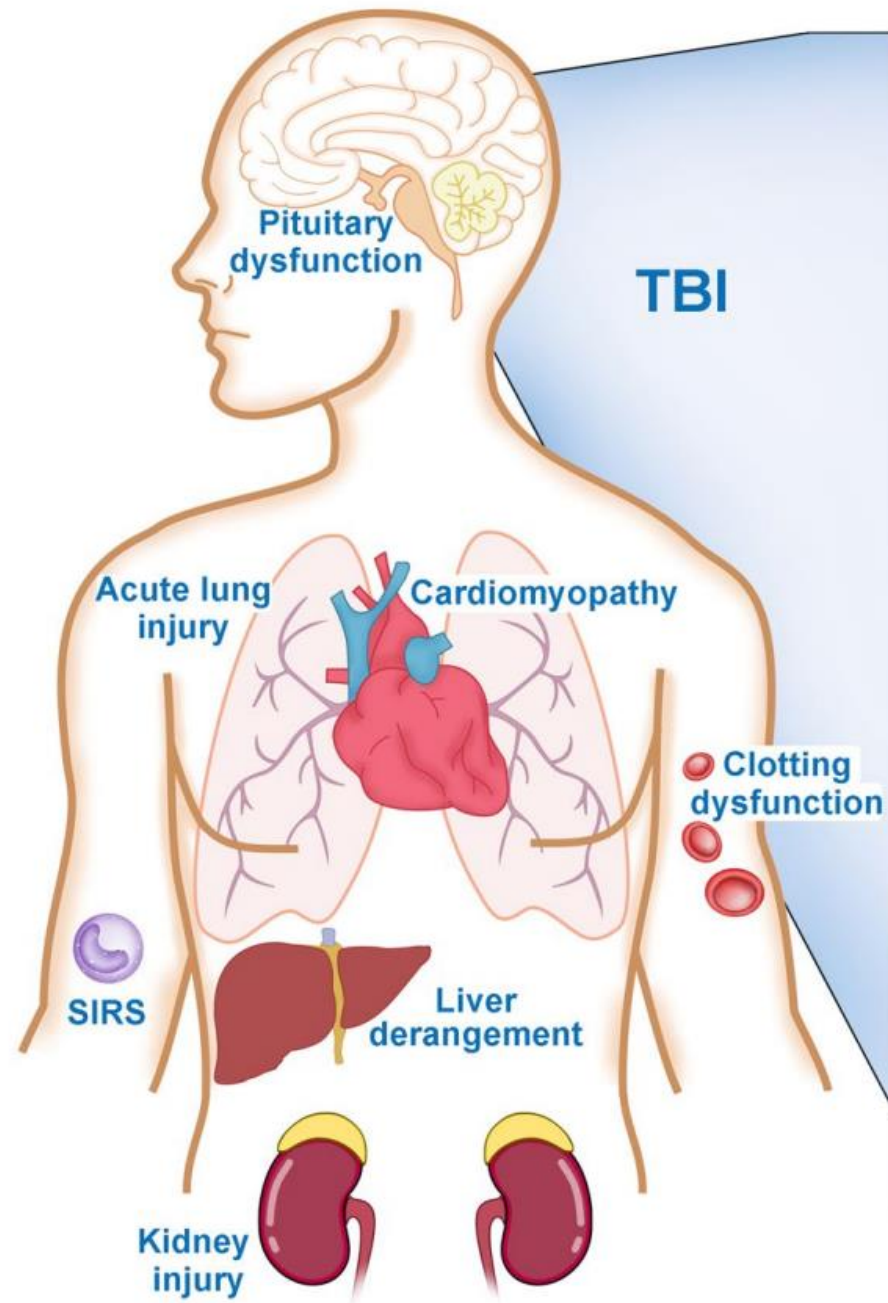
Transfusion Variation in transfusion triggers reflects lack of evidence

Acute kidney injury Occurs in 10% of TBI patients and is associated with poor long-term outcomes

Nutrition management Nutrition management should prioritize the prevention of nutrition induced harm: avoid hyperglycemia, administer micronutrients early on, and delayed enteral nutrition should raise no concern

Mobilization and rehabilitation Early mobilization is feasible, but benefit is unknown
Early rehabilitation referrals might be associated with earlier functional gain

Coagulation management TXA should be administered in all bleeding multiple trauma patients < 3 h. TXA may be considered in isolated mild-to-moderate but not severe TBI
Significant variability in the timing of LMWH initiation exists. Before LMWH can be started, intermittent pneumatic compression should be applied



SIBICC SEVERE TBI ALGORITHM



Only ICP monitoring
(English)



Only ICP monitoring
(Chinese)



ICP and brain tissue
oxygen monitoring



GCS assessment



Guidelines for the Management of
Severe TBI, Fourth Edition



Guidelines for the Management
of Severe TBI: 2020 Update