Kraniotrauma – pohled intenzivisty

Eva Wittenbergová Kongres ČSARIM, Brno, 20.9.2024



KLINIKA ANESTEZIOLOGIE, PERIOPERAČNÍ A INTENZIVNÍ MEDICÍNY FAKULTY ZDRAVOTNICKÝCH STUDIÍ UNIVERZITY J. E. PURKYNĚ V ÚSTÍ NAD LABEM A KRAJSKÉ ZDRAVOTNÍ, a. s. – MASARYKOVY NEMOCNICE V ÚSTÍ NAD LABEM, o. z. Platón - Obrana Sokratova: "Zdá se tedy, že alespoň v této maličkosti jsem moudřejší, protože nejsem toho názoru, že něco vím o věcech, o kterých nic nevím."

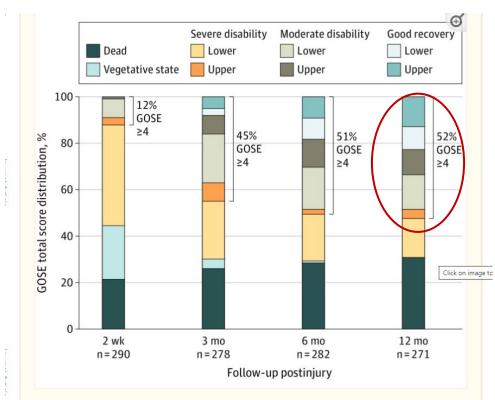
Co řeším(e) v současné době

- Smysluplnost
- Multimodálnost monitorace
- Optimální hodnoty nitrolebního tlaku (ICP), mozkového perfuzního tlaku (CPP), autoregulaci mozku

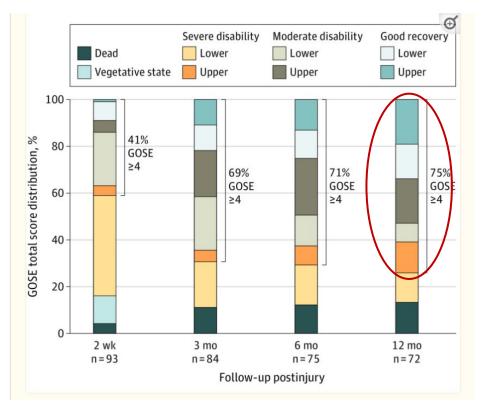


Functional outcomes over the first year after moderate to severe traumatic brain injury in the prospective, longitudinal TRACK-TBI Study

Severe



Moderate

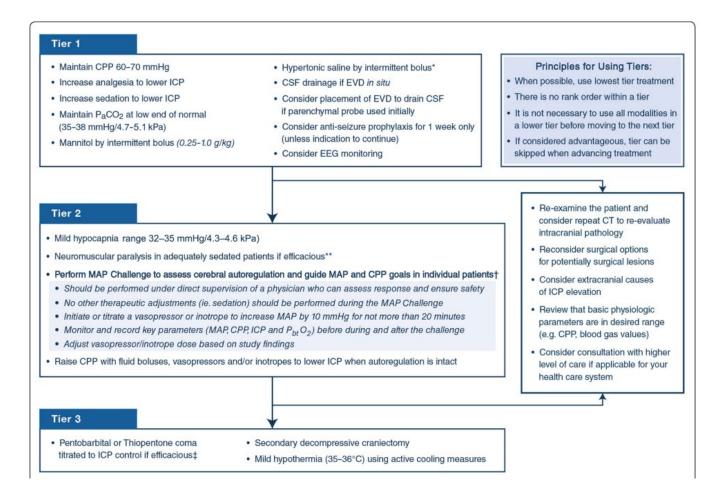


The Seattle Internatiol Severe Traumatic Brain Injury Consensus Conference (SIBICC)

Tier Zero (Basic Severe TBI Care - Not ICP Dependent) **Expected Interventions:** Admission to ICU · Consider anti-seizure medications for 1w only (in the absence of an indication to continue) · Endotracheal intubation and mechanical ventilation Maintain CPP initially ≥ 60 mmHg · Serial evaluations of neurological status and pupillary reactivity Maintain Hb > 7g/dL Elevate HOB 30-45° Avoid hyponatremia · Analgesia to manage signs of pain (not ICP directed) · Optimize venous return from head · Sedation to prevent agitation, ventilator asynchrony, etc. (eg. keeping head midline, ensure cervical collars are no too tight) (not ICP directed) · Arterial line continuous blood pressure monitoring · Temperature management to prevent fever Maintain SpO2 ≥ 94% Measure core temperature Treat core temperature above 38°C **Recommended Interventions:** · Insertion of a central line . End-tidal CO2 monitoring

Fig. 1 Consensus-based basic severe traumatic brain injury care for patients with an ICP monitor in situ. These are basic treatments recommended as fundamental to the care of patients with sTBI, to be initiated ("Expected interventions") or considered ("Recommended interventions") upon ICU admission of a patient with an ICP monitor, regardless of the monitored pressure. CO₂ carbon dioxide, CPP cerebral perfusion pressure, Hg hemoglobin, HOB head of bed, ICP intracranial pressure, ICU intensive care unit, spO₂ arterial oxygen saturation

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



Váha doporučení

We present a series of consensus-based algorithms for adult sTBI management. Our consensus working group (CWG) established 18 interventions as fundamental to sTBI care and ten treatments not to be used. It also established a three-tier algorithm focused on treating elevated ICP wherein higher tiers involve therapies with higher risk. We suggest considerations to address when advancing from lower to higher tiers and recommendations for critical neuroworsening aimed at assisting the recognition, workup, and treatment of declining patients. Novel elements include guidance for autoregulation-based ICP treatment and the performance of MAP Challenges, as well as two sets of heatmaps to guide (1) consideration of sedation holidays to facilitate neurological examination and (2) ICP-monitor removal.

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.

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.

Level Land II

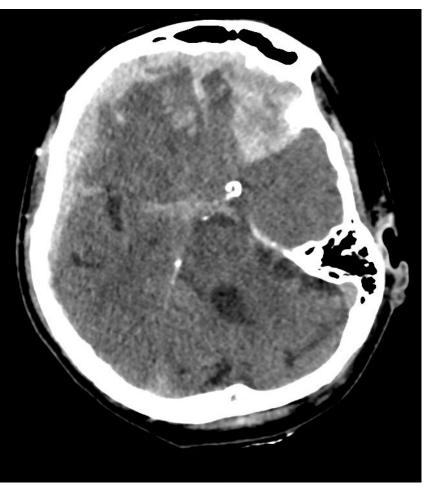
There was insufficient evidence to support a Level I or II recommendation for this topic.

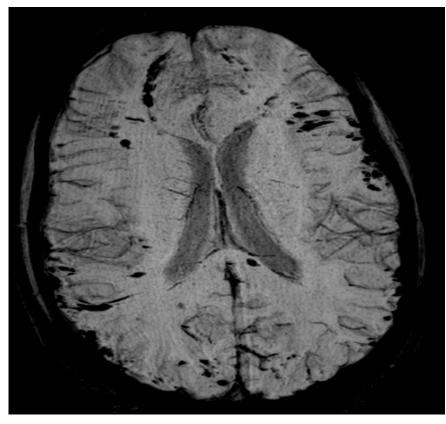
Level III

- 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)
 during the first 12 hours after injury may be considered.

Nejsou všichni stejní





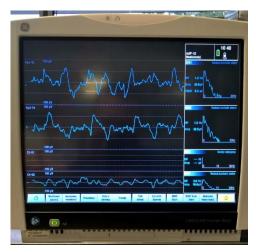


From: Use a "GHOST-CAP" in acute brain injury



Multimodální monitorace- Jen ICP nestačí









Brain Tissue Oxygen Monitoring and Management in Severe Traumatic Brain Injury (BOOST-II): a Phase II Randomized Trial. <u>David O. Okonkwo</u>, et al. Crit Care Med. 2017. Nov 45

ſ	Values in mmHg	ICP ≤ 22	ICP > 22
	PbtO ₂ ≥ 20	Type A No interventions needed	Type B Interventions to lower ICP
	PbtO ₂ < 20	Type C Interventions to increase PbtO ₂	Type D Interventions to lower ICP and increase PbtO ₂

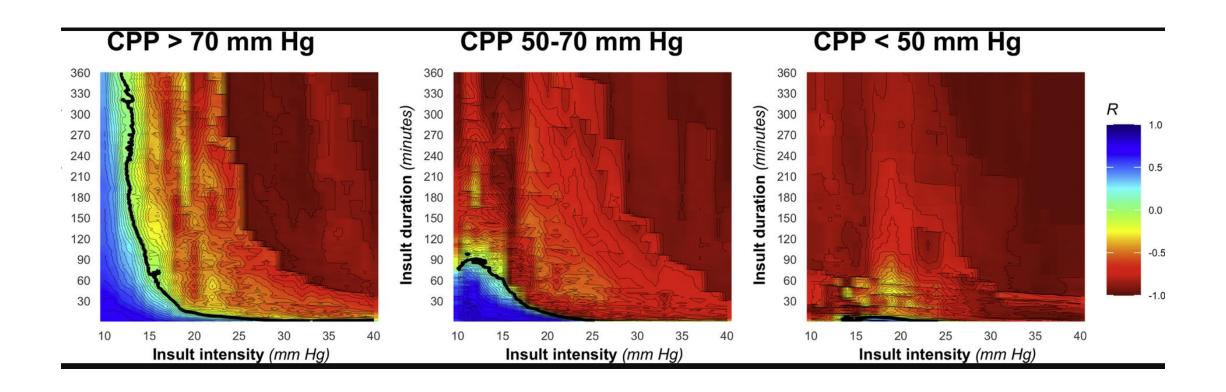
Scenario	Tier	Task#	Task	ICP Only	ICP PbtO
		1	Adjust head of bed to lower ICP	61	44
		2	Ensure temperature < 38	72	33
В		3	Adjust sedation and analgesia	324	181
	1	4	Adjust ventilation to obtain PaCO2 between 35-40 mmHg	38	10
		5	CSF drainage	165	59
		6	Standard dose mannitol (0.25 – 1.0 g/kg)	63	23
		7	Hypertonic saline	70	26
		1	Adjust ventilation to lower PaCO2 to 32-35 mmHg.	0	
		2	High dose mannitol (>1g/kg), or higher frequency of standard dose	6	6
	2	3	Repeat CT – look for increased size of intracranial mass lesions	14	7
		4	Treat surgically remediable lesions with craniotomy	0	1
		5	Adjust temperature to 35 – 37 °C	4	4
		1	Pentobarbital coma	1	0
	,	2	Decompressive craniectomy	3	0
	3	3	Adjust Temperature to 32 – 34.5 °C	1	0
		4	Neuromuscular paralysis	8	2

Effect of Haemoglobin Transfusion Treshold on Cerebral Haemodynamics and Oxygention. Jamal JM. Neurotrauma 2015. Aug 15

	7 g/dL group	10 g/dL group	p
	n=92	n=95	value
Time PbtO ₂ was monitored (h), median (IQR)	76.5 (78.5)	103 (95)	0.04
Developed brain tissue hypoxia, n (%) ^a	31/92 (33.7)	26/95 (27.4)	0.43
When probe was in normal tissue, n (%)	16/64 (25)	6/59 (10.2)	0.04
When probe was in abnormal tissue, n (%)	15/28 (53.6)	20/36 (55.6)	>0.99
Time PbtO ₂ was <10 mm Hg (h), median (IQR) ^b	0 (9.5)	0 (7)	0.59
When probe was in normal tissue (h, n=123), median (IQR)	0 (5)	0 (0)	0.06
When probe was in abnormal tissue (h, n=64), median (IQR) $$	9 (28.5)	9 (20.5)	0.86
		Open in a separat	e window

- Skupina s prahem 10g/dl měla menší riziko úmrtí do 72h, pak se rizika srovnala
- 28denní mortalita stejná
- Nebyla hodnocena rizika transfuze

Visualizing the Pressure and time burden of intracranial hypertension in adult and pediatric brain injury. Guiza F et al. Intensive Care Med 2015 Jun; 41(6)



Mozková autoregulace

= schopnost udržet konstantní cerebral blood flow (CBF) za měnícího se CPP díky změnám cerebrovaskulární rezistence, tj. vasodilataci a vasokonstrikci Published and copyright by

THE AMERICAN PHYSIOLOGICAL SOCIETY, INC.

APRIL 1959 VOLUME 39 NUMBER 2.

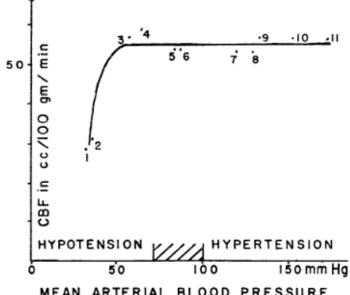
Cerebral Blood Flow and Oxygen Consumption in Man

NIELS A. LASSENI

April 1959

CEREBRAL BLOOD FLOW AND OXYGEN UPTAKE





MEAN ARTERIAL BLOOD PRESSURE

Fig. 1. Cerebral blood flow and blood pressure. Mean values of 11 groups of subjects reported in 7 studies have been plotted. Various and chronic conditions have been selected, characterized in a change in blood pressure. In all, this figure is based on a change in blood pressure.nations.

1 and 2, Drug-induced severe hypotension (81). 3 and 4, Drug-induced moderate hypotension (206). 5 and 6, Normal pregnant women and normal young men (206, 173). 7, Drug-induced hypertension (230). 8, Hypertensive toxemic pregnancy (206). 11, Essential hypertension (229, 131, 228).

Differential Hemodynamic Response of Pial Arterioles Contributes to a Quadriphasic Cerebral Autoregulation Physiology

Samuel P. Klein D, MD, PhD; Veerle De Sloovere, MD; Geert Meyfroidt, MD, PhD; Bart Depreitere D, MD, PhD

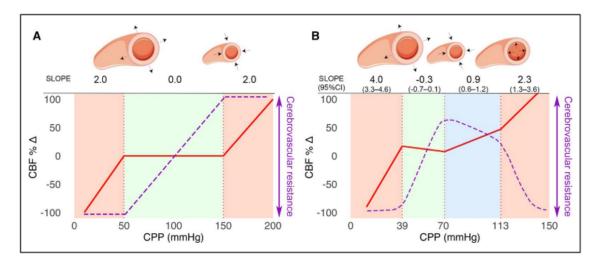


Figure 5. Quadriphasic curve compared with the classic triphasic cerebral perfusion pressure (CPP)–cerebral blood flow (CBF) curve.

A, Cerebrovascular autoregulation (CA) is classically visualized as a triphasic curve consisting of a wide plateau of steady CBF (red curve) between a CPP of 50 and 150 mm Hg (green area). Red areas indicate absent CA with a passive CPP-CBF relationship. Cerebrovascular resistance is represented by the purple dashed line. The underlying physiology is explained by maximal vasodilation at the lower limit of autoregulation (LLA) and maximal vasoconstriction at the upper limit of autoregulation (ULA). **B**, We have demonstrated a quadriphasic curve with a relatively narrow plateau between the LLA and ULA1 (green area). Between ULA1 and ULA2, there is progressive failure of CA starting at the level of the smallest arterioles and progressing toward larger arterioles (blue area) until all arterioles fail to resist increasing CPP and flow becomes completely pressure passive (right-sided red area). During maximally maintained vasoconstriction (blue area), the CPP-CBF relationship is sloped but attenuated.

Cerebrovascular Autoregulation Monitoring in the Management of Adult Severe Traumatic Brain Injury: A Delphi Consensus of Clinicians

B. Depreitere^{1*}, G. Citerio², M. Smith³, P. David Adelson^{4,5}, M. J. Aries⁶, T. P. Bleck⁷, P. Bouzat⁸, R. Chesnut⁹, V. De Sloovere¹⁰, M. Diringer¹¹, J. Dureanteau¹², A. Ercole¹³, G. Hawryluk¹⁴, C. Hawthorne¹⁵, R. Helbok¹⁶, S. P. Klein¹⁷, J. O. Neumann¹⁸, C. Robba¹⁹, L. Steiner^{20,21}, N. Stocchetti²², F. S. Taccone²³, A. Valadka²⁴, S. Wolf²⁵, F. A. Zeiler^{26,27,28,29,13} and G. Meyfroidt³⁰

Table 1 Statements on which consensus was reached (R1,2,3 = round 1,2,3)

No (section)	Consensus statement	Score R1	Score R2	Score R3
1 (1)	CA covers several physiological mechanisms alming at adequate nutrient supply to the brain accord- ing to its needs. The current Delphi consensus process focuses on the clinical assessment of the ability to maintain constant global CBF in response to different external stimuli	N/A	N/A	95.8%
2 (1)	CA impairment is not binary, but a process that results in dynamic narrowing of the CBF plateau between the lower and upper limit of CA and probably also in dynamic shifts in the location of the plateau on the CPP axis.	N/A	N/A	91.7%
3 (2)	A CPP below 50 mmHg should never be accepted	N/A	N/A	83.3%
4 (2)	Potential side effects of olevated CPP such as cardiopulmonary complications and brain hyperperfu- sion, may occur in the higher ranges of CPP. In these ranges, additional monitoring for such side effects may be considered	NA	N/A	100%
5 (2)	Both intensity and duration of low CPP insults are determinant in terms of association with poor outcome	83.3%	85.7%	
6 (2)	Both Intensity and duration of high CPP Insults are determinant in terms of association with poor outcome	83.3%	85.7%	
7 (2)	Episodes of low CPP are more detrimental than episodes of high CPP	70.8%	85.7%	
8 (2)	Because of potential dynamic CA Impairment, absolute and universal CPP targets do not exist. The safe	100%	100%	
9 (2)	CPP zone can differ between individuals and can change within individuals The CPP target zone depends on CA status as well as on other variables, and is/can be narrower than	78.3%	100%	
	the area between the lower and upper limit of CA			
10 (4)	The correlation between extracellular glutamate concentration as measured with microdialysis and CPP is inaccurate in reflecting CA	77.8%	85.7%	
11 (4)	The correlation between extracellular glutamate concentration as measured with microdialysis and CPP is not validated as a reflection of CA	76.5%	93.7%	
12 (4)	Current methods to estimate CA status are insufficiently understood. The different indices produce different information	73.9%	95.2%	
13 (4)	Information on CA status may be helpful, but is subordinate to ICP, CPP and PbO2 signals	/8.3%	81.096	
14 (5)	Impaired CA worsens tolerability for high ICP (i.e., association with worse outcome occurs at lower ICP values)	69.6%	90.5%	
15 (5)	Impaired CA worsens tolerability for low PbO2 (i.e., association with worse outcome occurs at higher PbO2 values)	59.1%	85.0%	
16 (5)	Impaired CA worsens overall tolerability for secondary insults (i.e., unfavorably shifts the thresholds associated with worse outcome)	87.0%	95.2%	
17 (5)	Whether overall CA status is intact or deficient, has an independent association with outcome (regardless of actual CPP)	78.3%	100%	
18 (6)	The priority for research on CA is high	69.6%	76.2%	
19 (6)	When a new CA assessment method is developed, it should be validated against a method that includes quantitative CBF analysis in the equation, either in animal research in the lab or in patients	N/A	N/A	91.7%
20 (6)	CA research should move to patient studies, investigating whether CA-based protocols are safe and whether they lead to different treatment strategies and different outcomes	N/A	N/A	100%
21 (6)	Research should focus on prospective patient feasibility studies to test protocols that incorporate CA information	82.6%	85.7%	
22 (6)	Research should focus on prospective patient feasibility studies to test whether dynamic CPP targets from CPPopt algorithms can be achieved/maintained	78.3%	90.5%	
23 (6)	Research should focus on prospective patient safety studies on the implementation of CA information in clinical situations	78.3%	85.7%	
24 (6)	Research should focus on prospective patient safety studies on dynamic CPP targets from CPPopt algorithms	72.7%	85.7%	
25 (6)	Research should focus on randomized controlled trials on dynamic CPP targets from CPPopt algo- rithms versus standard CPP management	69.6%	81.0%	

CA cerebrovascular autoregulation; CBF cerebral blood flow; CPP cerebral perfusion pressure; CPPopt optimal cerebral perfusion pressure; Pb02 partial pressure of oxygen in the brain tissue; R1,2,3 round 1,2,3

Děkuji za pozornost!

One size does not fit all



Má to smysl!

