

EDITORIAL

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# How I manage intracranial hypertension



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## Why and when to manage intracranial hypertension

The detrimental effects of intracranial hypertension (HICP, high intracranial pressure) are well documented [1, 2]. HICP can cause secondary brain injury and death, and therefore, intracranial pressure (ICP) elevations should be aggressively treated.

HICP has been classically defined as an ICP > 20 mmHg, and this threshold has been considered the trigger for treatment [3]. Recent BTF guidelines have moved this threshold to 22 mmHg [4], grounded on a single-centre, retrospective study. This modification is trivial [5]. As for many other treatment options in intensive care, a single threshold is debatable. In fact, recent evidence suggests that not a single value but the time spent over the threshold and its intensity, the so-called ICP dose, is more important [6]. Moreover, Guiza demonstrated that not only higher values but also prolonged exposure to values below the classical threshold are associated with negative outcomes [7]. In addition, if cerebral perfusion pressure (CPP, i.e. MAP-ICP) is critically low (< 50 mmHg), ICP is no longer a predictor for poor outcome and lower ICP values might be barely tolerated. On the contrary, ICP insults in the range 18–23 mmHg can be tolerated for a longer duration at higher CPPs. In my practice, the ICP alarm is set at 20 mmHg and low CPP alarm at 55 mmHg. This is a warning signal for nurses at the bedside. Before starting any treatments for high ICP, I consider both the intensity and duration of HICP. I am flexible with thresholds putting them in the clinical contest, considering also CPP. Short-lasting, low-intensity episodes (low ICP dose with normal CPP) are observed and not treated. On the contrary, higher ICP doses, progressively rising trends, or/and HICP impacting CPP require prompt treatment.

## How I manage intracranial hypertension

Figure 1 summarises the algorithm that I use in clinical practice. Before starting any ICP-directed therapies, I try to correct any reversible cause and systemic abnormality affecting intracranial volumes and causing raised ICP

(see Additional file 1). I always consider the surgical option with a neurosurgeon; mass-occupying space should be promptly evacuated when indications are met, and hydrocephalus should be drained.

When I decide to administer ICP-lowering therapies, I use a “staircase” approach [1] with escalating treatment intensity (starting with low risk-benefit profiles) [8]. The first-line ICP-lowering strategies that I consider (without a priority between them) include:

- Head-up positioning (15–30°),
- Hemodynamic stability aimed to maintain an appropriate cerebral perfusion pressure (CPP 50–70 mmHg according to autoregulatory status. Increasing mean arterial pressure + 10% might be considered as a test for exploring pressure autoregulation),
- Sedation and analgesia (propofol, 4–6 mg/kg/h and opioids, fentanyl 1–4 µg/kg/h used at the lowest dose producing ICP control. Maintain CPP with vasopressors, if needed) [9],
- Mechanical ventilation to prevent hypercapnia and hypoxia (target PaCO<sub>2</sub> at 35 mmHg, and oxygen saturation ≥ 94%),
- Normothermia; if the temperature is > 37.5 °C (internal), I start Diclofenac infusion [10].
- Crystalloids as preferred maintenance fluids [11] to maintain euvolemia and to prevent drops in plasma osmolarity. I do not use colloids or hypotonic solutions w/o glucose as maintenance fluids.

If HICP persists, I subsequently escalate to osmotic agents, mannitol (up to 0.5–1 g/kg every 4–6 h) or hypertonic saline (7.5% solution, 100 ml every 4–6 h). They have several transient mechanisms (lasting 4–6 h) mainly due to osmotic effects but also hemodilution, increased cardiac output and increased blood pressure. I prefer testing both of them (using an equimolar bolus) for evaluating their efficacy in the individual patient. Their efficacy is higher if started at an ICP > 25 mmHg [11].

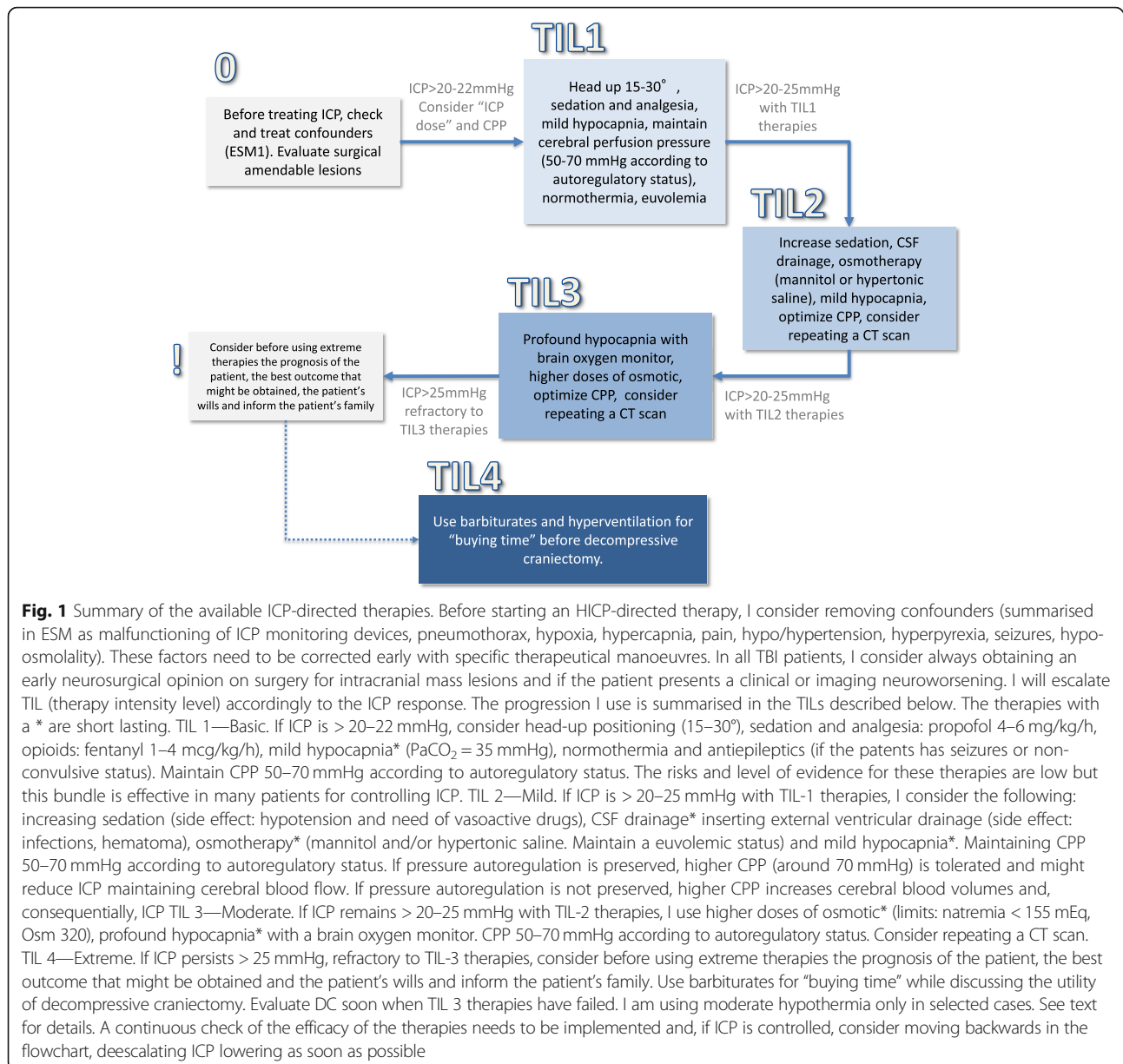
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### When and how to escalate to upper tier therapies

I generally reserve to patients with refractory intracranial hypertension ICP-lowering strategies associated with significant side effects and potential complications as hyperventilation, metabolic suppression and decompressive craniectomy [8, 12].

Hyperventilation produces a reduction of HICP by inducing cerebral vasoconstriction and reducing cerebral blood volume [13]. The effect is short lasting and cease when the interstitial pH, alkalotic during the immediate hyperventilation phase, returns to normality. However, because of the theoretical risk of hypoperfusion, I aim to achieve mild hyperventilation, i.e. a PaCO<sub>2</sub> ~ 30–32 mmHg, only in patients in whom ICP remains abnormally elevated

despite first- and second-line treatments, considering adding for safety a brain oxygenation monitor. I use more aggressive hyperventilation only in life-threatening cases with the risk of cerebral herniation and death.

Barbiturates have been historically used for decreasing brain metabolism and consequently cerebral blood flow/volume and therefore HICP at the cost of serious side effects including hypotension and infections. I avoid long-term administration, and I generally administer thiopentone (10 mg/kg bolus, checking its efficacy, followed by 3–8 mg/kg/h infusion) as temporary “bridge” to decompressive craniectomy (DC) in refractory cases. I prefer, as third tier therapy, DC that has a long-lasting effect on the control of refractory HICP. DC performed

without severe refractory HICP increases the rate of unfavourable neurologic outcome and should be avoided [14]. On the other hand, DC in patients with severe refractory HICP reduces mortality (22 more survivors for every 100 patients treated) [15]. At 12 months, 13/22 survivors (59%) had favourable outcomes while 9/22 (41%) were in a vegetative state or in lower severe disability. For these reasons, DC needs to be wisely pondered in the context of refractory HICP and it should be undertaken timely in subjects with a potentially acceptable prognosis (i.e. before irreversible damages occurred), considering individual patient's preferences and family's quality of life expectations.

In conclusion, my approach to ICP-lowering strategies has a stepwise fashion associated with a continuous check of the efficacy of the therapies. This will allow me to deescalate ICP-lowering strategies as soon as possible (ICP control > 24 h). Tapering therapies (as hyperventilation and osmotic) might produce a rebound effect, and it needs to be done slowly and under ICP monitoring.

Alternatively, if the therapies are ineffective, I intensify treatments until the patients are judged salvable. When, in more severe unsalvageable cases, everything is ineffective and DC is not an option, a wise limitation of the therapies has to be evaluated.

## Additional file

**Additional file 1:** Summary of the remediable causes of intracranial hypertension. (DOCX 15 kb)

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## References

1. Stocchetti N, Maas AIR. Traumatic intracranial hypertension. *N Engl J Med*. 2014;370:2121–30.
2. Maas AIR, Menon DK, Adelson PD, Andelic N, Bell MJ, Belli A, et al. Traumatic brain injury: integrated approaches to improve prevention, clinical care, and research. *Lancet Neurol*. 2017;16:987–1048.
3. Marmarou A, Anderson RL, Ward JD, Choi SC, Young HF, Eisenberg HM, et al. Impact of ICP instability and hypotension on outcome in patients with severe head trauma. *J Neurosurg*. 1991;75:S59–66.
4. Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GWJ, Bell MJ, et al. Guidelines for the management of severe traumatic brain injury, fourth edition. *Neurosurgery*. 2017;80:6–15.
5. Helbok R, Meyfroidt G, Beer R. Intracranial pressure thresholds in severe traumatic brain injury: con : The injured brain is not aware of ICP thresholds! *Intensive Care Med*. 2018;44:1318–20.
6. Vik A, Nag T, Fredriksli OA, Skandsen T, Moen KG, Schirmer-Mikalsen K, et al. Relationship of "dose" of intracranial hypertension to outcome in severe traumatic brain injury. *J Neurosurg*. 2008;109:678–84.
7. Güiza F, Depreitere B, Piper I, Citerio G, Chambers I, Jones PA, et al. Visualizing the pressure and time burden of intracranial hypertension in adult and paediatric traumatic brain injury. *Intensive Care Med*. 2015;41:1067–76.
8. Stocchetti N, Carbonara M, Citerio G, Ercole A, Skrifvars MB, Smielewski P, et al. Severe traumatic brain injury: targeted management in the intensive care unit. *Lancet Neurol*. 2017;16:452–64.
9. Opendakker O, Vanstraelen A, De Sloovere V, Meyfroidt G. Sedatives in neurocritical care: an update on pharmacological agents and modes of sedation. *Curr Opin Crit Care*. 2019;25:97–104.
10. Cormio M, Citerio G. Continuous low dose diclofenac sodium infusion to control fever in neurosurgical critical care. *Neurocrit Care*. 2007;6:82–9.
11. Oddo M, Poole D, Helbok R, Meyfroidt G, Stocchetti N, Bouzat P, et al. Fluid therapy in neurointensive care patients: ESICM consensus and clinical practice recommendations. *Intensive Care Med*. 2018;44:449–63.
12. Stocchetti N, Zanaboni C, Colombo A, Citerio G, Beretta L, Ghisoni L, et al. Refractory intracranial hypertension and "second-tier" therapies in traumatic brain injury. *Intensive Care Med*. 2008;34:461–7.
13. Stocchetti N. Hyperventilation in head injury: a review. *Chest*. 2005; 127:1812–27.
14. Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, et al. Decompressive craniectomy in diffuse traumatic brain injury. *N Engl J Med*. 2011;364:1493–502.
15. Hutchinson PJ, Kollias AG, Timofeev IS, Corteen EA, Czosnyka M, Timothy J, et al. Trial of decompressive craniectomy for traumatic intracranial hypertension. *N Engl J Med*. 2016;375:1119–30.

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