Prise en charge du TCC 2026

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Plan

1. Introduction
2. Révision ATLS
3. Prise en charge anesthésique
4. Lignes directrices de la Brain Trauma Foundation
5. Physiologie cérébrale et TCC
6. Prise en charge avancée
7. Conclusion

Kevin, Bobby et Jessica reviennent d’un party

Accident auto-décor haute vélocité

- coussins gonflables déployés
- deux passagers avant inconscients à l’arrivée des secours
- incarcérés environs 30 minutes
- Kevin 23 ans PA 165/100, FC 60, sat 89%
- Jessica 21 ans PA 85/60, FC 125, sat 94%
- Bobby 24 ans PA 110/75, FC 100, sat 98%

Introduction

- **Cause majeure d’invalidité, surtout chez une population jeune**
- **75% TCC léger 15% TCC modéré et 10% TCC sévère**
- **Souvent en association avec d’autres traumatismes**
- **20-25% de mortalité pour les TCC sévères**
- **60% de déficits sévères**

TCC

- Insulte primaire

- Insultes secondaires

TCC

L’objectif numéro 1 lors de la prise en charge initiale d’un TCC => limiter les insultes secondaires

L’objectif numéro 2 si on a une suspicion de traumatisme crânien => procéder à un scan cérébral pour identifier et potentiellement évacuer une lésion qui a un effet de masse

TCC

2 principes de base pour limiter l'insulte secondaire

=> maintenir l'oxygénation

=> maintenir une pression de perfusion cérébrale

TCC

Multiples études

=> une saturation < 80% triple la mortalité

=> une pression systolique < 90 mmHg double la mortalité

Révision ATLS

A

B

C

D si possible avant sédation/paralysie et en absence d’hypotension

Révision ATLS

Priorité => régler la cause de l’hypotension

Ne pas retarder une laparotomie pour faire un scan cérébral si instabilité importante

Aller au scan dès que possible

TABLE 6-2 GLASGOW COMA SCALE (GCS)

ORIGINAL SCALE	REVISED SCALE	SCORE
Eye Opening (E)	Eye Opening (E)	
Spontaneous	Spontaneous	4
To speech	To sound	3
To pain	To pressure	2
None	None	1
	Non-testable	NT
Verbal Response (V)	Verbal Response (V)	
Oriented	Oriented	5
Confused conversation	Confused	4
Inappropriate words	Words	3
Incomprehensible sounds	Sounds	2
None	None	1
	Non-testable	NT
Best Motor Response (M)	Best Motor Response (M)	
Obeys commands	Obeys commands	6
Localizes pain	Localizing	5
Flexion withdrawal to pain	Normal flexion	4
Abnormal flexion (decorticate)	Abnormal flexion	3
Extension (decerebrate)	Extension	2
None (flaccid)	None	1
	Non-testable	NT

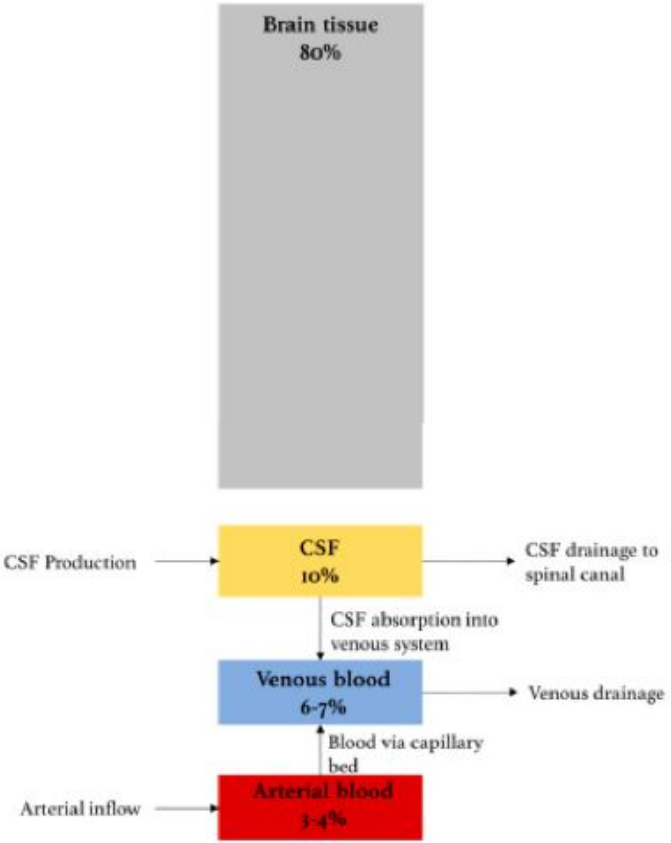


Figure 1. The Monro–Kellie model for the contents of the intracranial compartment. 'Brain tissue' includes neurons, glia, extracellular fluid and cerebral microvasculature. 'Venous' and 'Arterial blood' represents the intracranial blood volume in macro-vasculature and cerebral venous sinuses. 'CSF' includes ventricular and cisternal CSF.

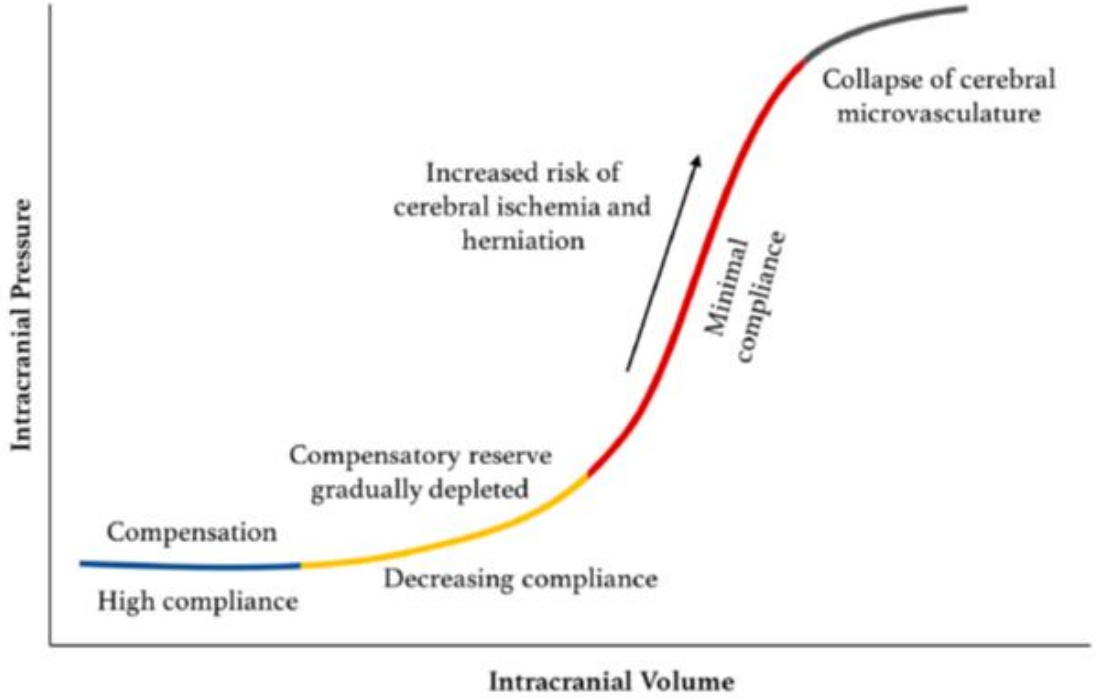
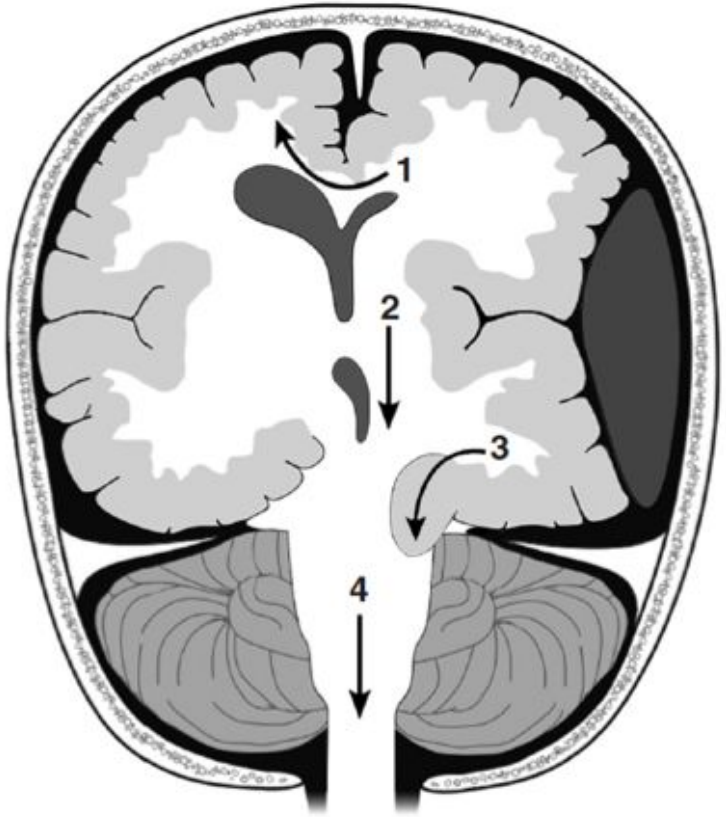


Figure 2. Pressure–volume curve for ICP. The pressure–volume curve has four 'zones': (1) baseline intracranial volume with good compensatory reserve and high compliance (blue); (2) gradual depletion of compensatory reserve as intracranial volume increases (yellow); (3) poor compensatory reserve and increased risk of cerebral ischemia and herniation (red); and (4) critically high ICP causing collapse of cerebral microvasculature and disturbed cerebrovascular reactivity (grey).



1: Subfalcine herniation
2: Central herniation
3: Transtentorial (uncal) herniation
4: Tonsillar herniation

Herniation Subtype	Physical Findings
Subfalcine	• Early unilateral motor deficits of lower extremities • Bladder incontinence • Late same-sided motor and sensory deficit • Late speech difficulty
Central	• Forced downward gaze • Dilated, unreactive pupils
Transtentorial (uncal herniation)	• Dilation of pupil ipsilateral to injury • Eye deviation downward and peripherally • Contralateral or ipsilateral (Kernohan notch phenomenon) hemiparesis
Tonsillar	• Early gradual decrease in level of consciousness • Late respiratory failure • Late flaccid paralysis

Kevin

Glasgow 4/15

Pupille D dilatée

Décérèbre à G

Jessica

Glasgow 7/15

Pupilles dilatées réactives

Retire

Liquide libre abdominal

Bobby

Glasgow 14/15

Ecchymoses au visage

Fractures ouvertes femur G

Comment on intube un TCC?

Quels sont les objectifs?

1. **éviter la désaturation**
2. **éviter l'hypercapnie**
3. **éviter l'hypotension**

Donc, prendre le contrôle rapidement des voies aériennes et de la ventilation

Quels agents d’induction?

1. Un agent d’induction qui agit rapidement
2. Un agent d’induction qui amène moins d’hypotension
3. Des curares à haute dose
4. Ce qu’il faut pour prévenir l’hypotension une fois l’induction faite

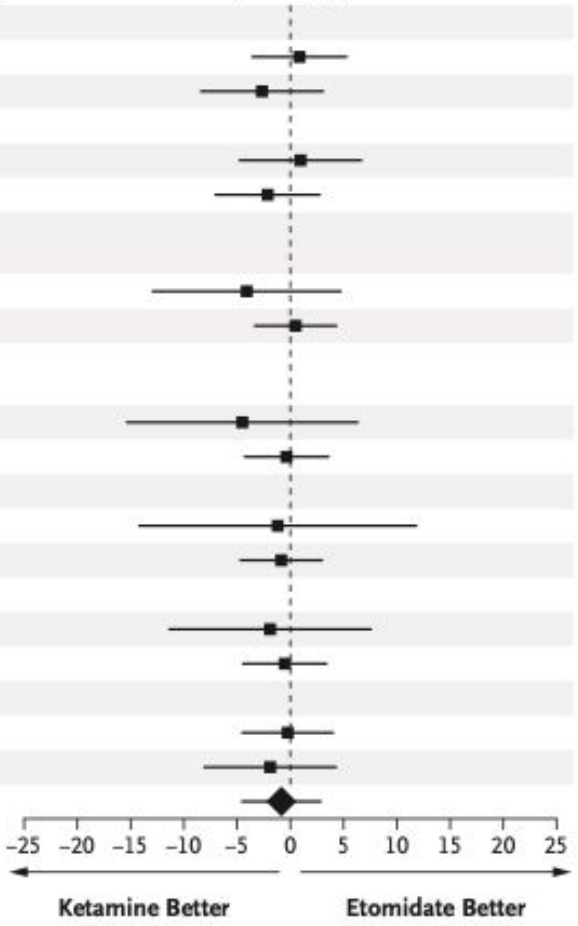
Ketamine or Etomidate for Tracheal Intubation of Critically Ill Adults

J.D. Casey,[1,2] K.P. Seitz,[2] B.E. Driver,[3] K.W. Gibbs,[4] A.A. Ginde,[5] S.A. Trent,[5,6] D.W. Russell,[7,8] A.L. Muhs,[2] M.E. Prekker,[3,9] J.P. Gaillard,[10,11] D. Resnick-Ault,[5] L.J. Stewart,[12,13] M.R. Whitson,[7,14] S.C. DeMasi,[15] A.E. Robinson,[3] J.A. Palakshappa,[4] N.R. Aggarwal,[16] J.C. Brainard,[12] D.J. Douin,[12] T.K. Marvi,[16] B.K. Scott,[12] S.M. Alber,[12] C. Lyle,[6] S. Gandotra,[7] G.W. Van Schaik,[15] A.J. Lacy,[15] K.C. Sherlin,[17] H.L. Erickson,[9] J.M. Cain,[4] B. Redman,[11] L.L. Beach,[14] B. Gould,[18] J. McIntosh,[19] A.A. Lewis,[1] B.D. Lloyd,[1] T.L. Israel,[15] B. Imhoff,[20] L. Wang,[20] A.B. Spicer,[21] M.M. Churpek,[21] T.W. Rice,[1,2] W.H. Self,[1,15] J.H. Han,[15,22] and M.W. Semler,[1,2] for the RSI Investigators and the Pragmatic Critical Care Research Group*

Table 2. Characteristics of the Intubation Procedure.

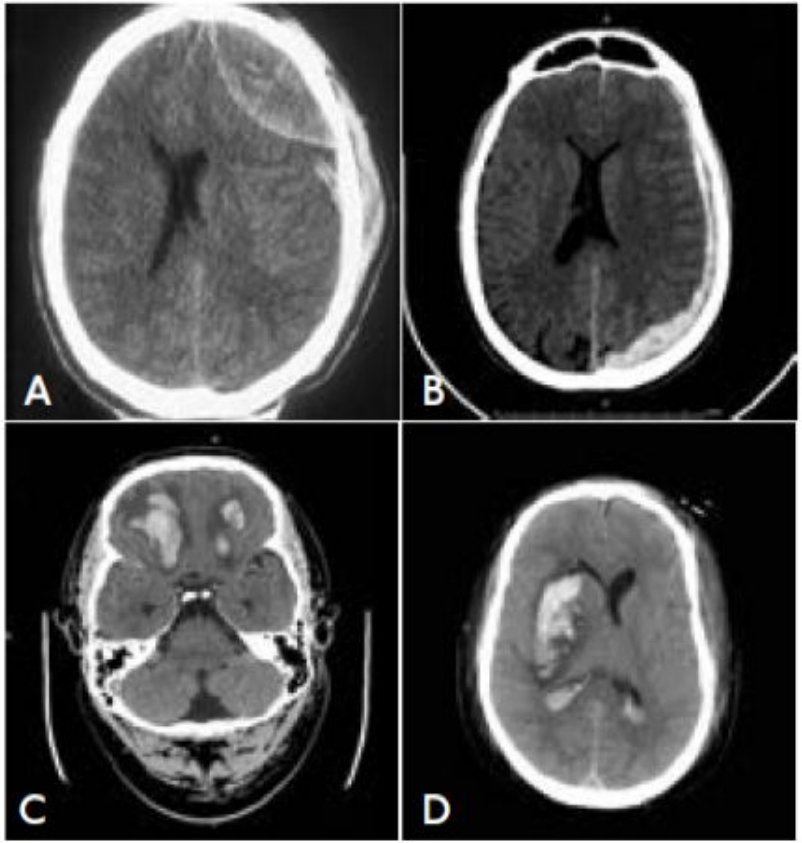
Characteristic	Ketamine (N=1176)	Etomidate (N=1189)	Difference (95% CI)*
Primary medication for induction of anesthesia — no. (%)†			
Ketamine	1167 (99.2)	3 (0.3)	99.0 (98.4 to 99.6)
Etomidate	6 (0.5)	1184 (99.6)	−99.1 (−99.6 to −98.5)
None	3 (0.3)	2 (0.2)	0.1 (−0.3 to 0.5)
Neuromuscular blocking agent — no. (%)‡			
Rocuronium	810 (69.0)	819 (69.0)	0.0 (−3.7 to 3.7)
Succinylcholine	362 (30.8)	365 (30.7)	0.1 (−3.6 to 3.8)
None	3 (0.3)	3 (0.3)	0.0 (−0.4 to 0.4)
Measurements or treatments at induction of anesthesia			
Median oxygen saturation (IQR) — %§	99 (97–100)	99 (97–100)	0 (−1 to 1)
Preoxygenation — no. (%)	1172 (99.7)	1186 (99.7)	−0.1 (−0.5 to 0.4)
Median systolic blood pressure (IQR) — mm Hg¶	127 (110–147)	127 (110–148)	0 (−3 to 3)
Vasopressor bolus or increased infusion rate — no. (%)	207 (17.6)	234 (19.7)	−2.1 (−5.2 to 1.1)
Laryngoscope used on the first attempt — no. (%)			
Video	1124 (95.6)	1127 (94.8)	0.8 (−0.9 to 2.5)
Direct	49 (4.2)	60 (5.0)	−0.9 (−2.6 to 0.8)
Other‖	3 (0.3)	2 (0.2)	0.1 (−0.3 to 0.5)
Instrument used on the first intubation attempt — no. (%)**			
Endotracheal tube with stylet	672 (57.3)	689 (58.1)	−0.8 (−4.7 to 3.2)
Bougie	455 (38.8)	446 (37.6)	1.2 (−2.7 to 5.1)
Neither endotrachal tube with stylet nor bougie	45 (3.8)	51 (4.3)	−0.5 (−2.1 to 1.1)

Subgroup	Ketamine	Etomidate
	no. of patients with event/total no. of patients (%)	
Location of intubation		
Emergency department	128/661 (19.4)	121/653 (18.5)
Intensive care unit	202/512 (39.5)	224/533 (42.0)
Sepsis or septic shock		
Yes	209/538 (38.8)	215/563 (38.2)
No	121/635 (19.1)	130/623 (20.9)
Vasopressor use <1 hr before enrollment		
Yes	115/246 (46.7)	136/273 (49.8)
No	215/927 (23.2)	209/913 (22.9)
Adrenal insufficiency or long-term glucocorticoid use		
Yes	45/136 (33.1)	48/129 (37.2)
No	285/1037 (27.5)	297/1057 (28.1)
Acute neurologic condition		
Yes	44/125 (35.2)	45/120 (37.5)
No	286/1048 (27.3)	300/1066 (28.1)
Acute cardiac condition		
Yes	87/216 (40.3)	95/222 (42.8)
No	243/957 (25.4)	250/964 (25.9)
APACHE II score		
<20	118/649 (18.2)	123/658 (18.7)
≥20	212/524 (40.5)	222/528 (42.0)
Overall	**330/1173 (28.1)**	**345/1186 (29.1)**



Dans les résultats

Plus de collapsus cardiovasculaires chez les patients avec un score Appache II plus élevé dans le groupe kétamine



■ **FIGURE 6-7 CT Scans of Intracranial Hematomas. A.** Epidural hematoma. **B.** Subdural hematoma. **C.** Bilateral contusions with hemorrhage. **D.** Right intraparenchymal hemorrhage with right to left midline shift and associated biventricular hemorrhages.

Table 1 CT grading system for diffuse brain injury after Marshall and colleagues.[52] The cisterns referred to are the ones surrounding the midbrain as assessed on CT head scan, that is, the interpeduncular, ambient, and quadrigeminal plate cisterns

Category of diffuse injury	Definition	Mortality (%)
I	No visible intracranial injury	10
II	Cisterns present 0–5 mm midline shift and small, high, or mixed density lesions <25 cc	14
III	Cisterns compressed or absent+I or II	34
IV	Midline shift >5 mm +I, II, or III	56

TABLE 6-1 CLASSIFICATIONS OF TRAUMATIC BRAIN INJURY

Severity	• Mild • Moderate • Severe		• GCS Score 13–15 • GCS Score 9–12 • GCS Score 3–8
Morphology	• Skull fractures	• Vault	• Linear vs. stellate • Depressed/nondepressed
		• Basilar	• With/without CSF leak • With/without seventh nerve palsy
	• Intracranial lesions	• Focal	• Epidural • Subdural • Intracerebral
		• Diffuse	• Concussion • Multiple contusions • Hypoxic/ischemic injury • Axonal injury

Source: Adapted with permission from Valadka AB, Narayan RK. Emergency room management of the head-injured patient. In: Narayan RK, Wilberger JE, Povlishock JT, eds. *Neurotrauma*. New York, NY: McGraw-Hill, 1996:120.

Kevin

Hématome épidural D

Jessica

?

Comment les aider avant leur prise en charge chirurgicale?

1. Hyperventilation/pCO2 visée?
2. Osmothérapie?
3. Pression artérielle visée?
4. Sédation?
5. Transfusions?
6. Acide tranexamique?
7. Température?

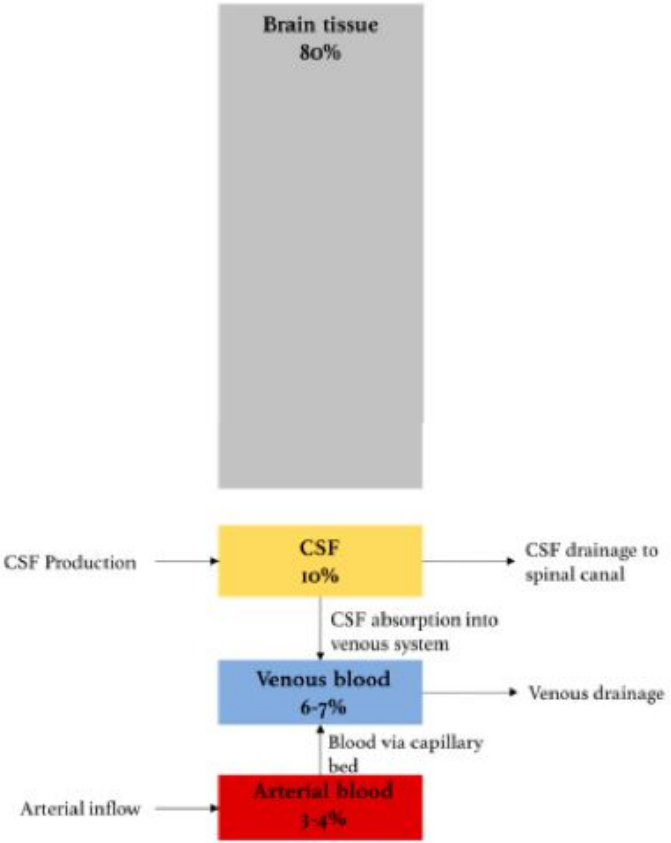
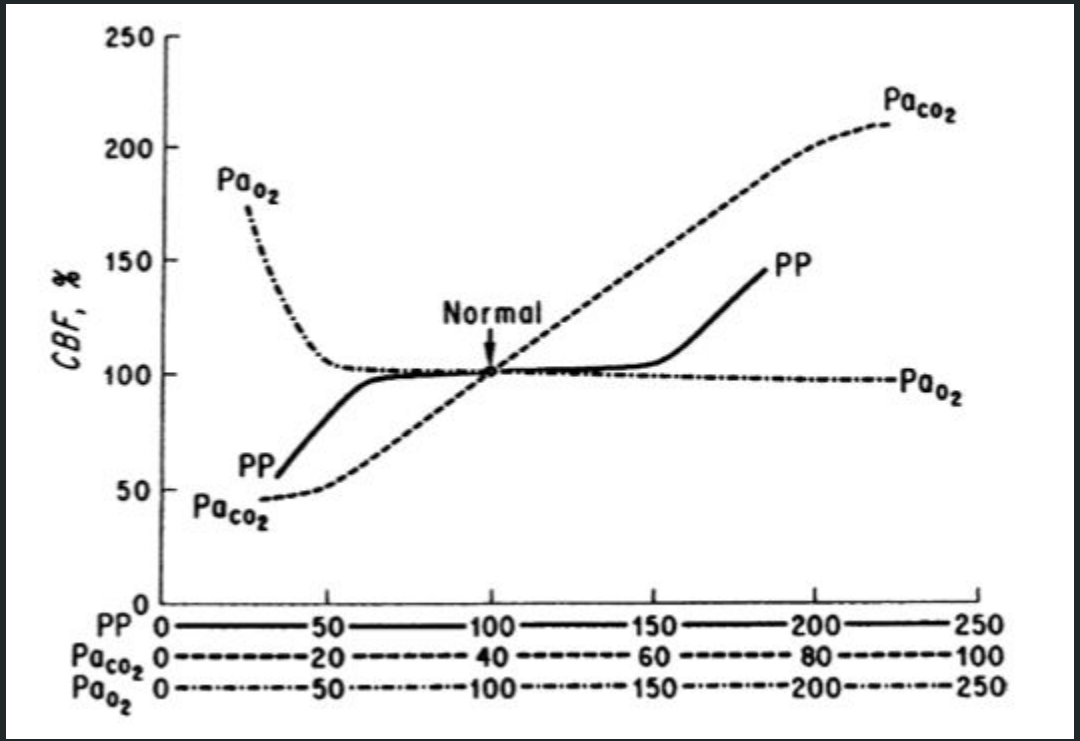


Figure 1. The Monro–Kellie model for the contents of the intracranial compartment. 'Brain tissue' includes neurons, glia, extracellular fluid and cerebral microvasculature. 'Venous' and 'Arterial blood' represents the intracranial blood volume in macro-vasculature and cerebral venous sinuses. 'CSF' includes ventricular and cisternal CSF.

pCO2 est LE facteur le plus important déterminant du débit sanguin cérébral

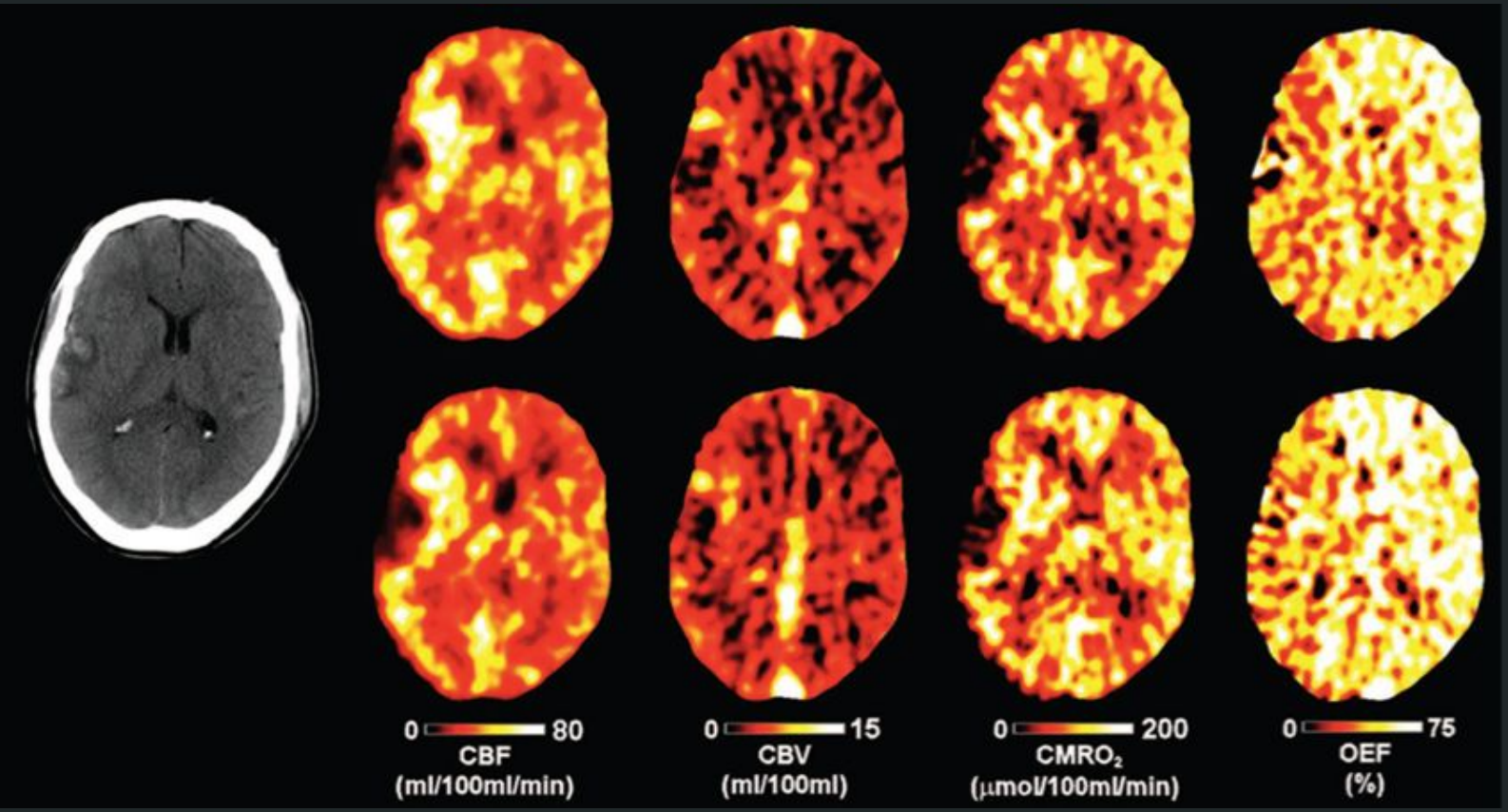
=> relation linéaire



Hyperventilation peut être une mesure temporaire chez un patient qui s’engage

On ne devrait pas aller sous une paCO2 de 25 mmHg

Si elle est utilisée, des mesures d’oxygénation cérébrale sont indiquées pour suivre son effet (plus en phase chronique)



pCO2 35
PIC 22
PPC 73

pCO2 29
PIC 17
PPC 80

Thérapies hyperosmolaires

- Tout le monde s’entend sur leur utilité dans le traitement des TCC pour diminuer la PIC
- Effet aussi sur la rhéologie
- Pas d’études convaincantes/bien construites de leur effet sur le devenir des patients
- Beaucoup d’études qui comparent le mannitol et le salin hypertonique
    - => HTS plus efficace que le mannitol pour diminuer la PIC, mais pas d’influence sur l’évolution

Thérapies hyperosmolaires

- **Pierre angulaire dans le traitement initial et la temporisation avant une intervention neurochirurgicale**

Quel agent utiliser chez

Kevin

Jessica

Pression artérielle visée?

Pression systolique

- **> 110 mmHg 15-49 ans et > 70 ans**
- **> 100 mmHg 50-69 ans**

Sédation

- **Barbituriques et propofol diminuent la PIC**
- **Barbituriques et propofol diminuent le métabolisme cérébral et sa consommation en O2**
- **Par contre => diminution du débit cardiaque et de la pression artérielle**

Sédation

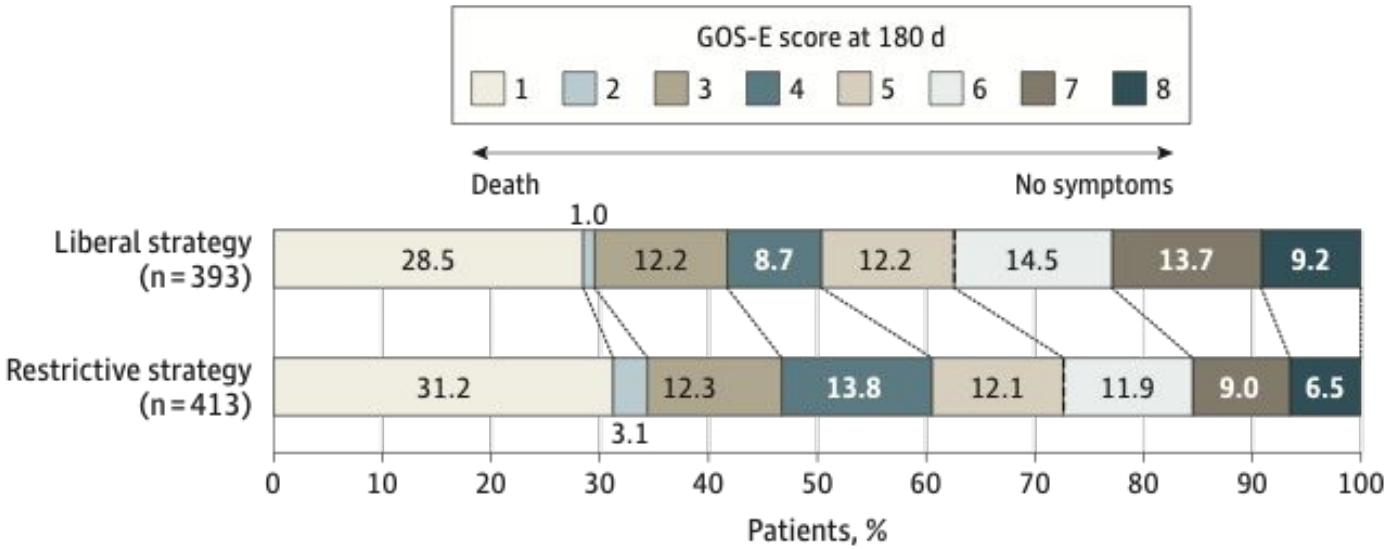
- Une utilisation « libérale » des barbituriques n’est pas recommandée
- Ils peuvent être utilisés dans les cas réfractaires au traitement médical et chirurgical
- Attention au propofol infusion syndrome
- Pas de différence sur le devenir dans une étude qui comparait le midazolam et le propofol

Transfusions

2 grosses études dans les dernières années avec un groupe libéral et un groupe restrictif

Hb > 70 VS Hb > 90 - étude positive -

Figure 4. Distribution of Glasgow Outcome Scale Extended (GOS-E) Scores 180 Days After Randomization (Secondary Outcome)



Hb > 70 VS Hb > 100 - étude négative -

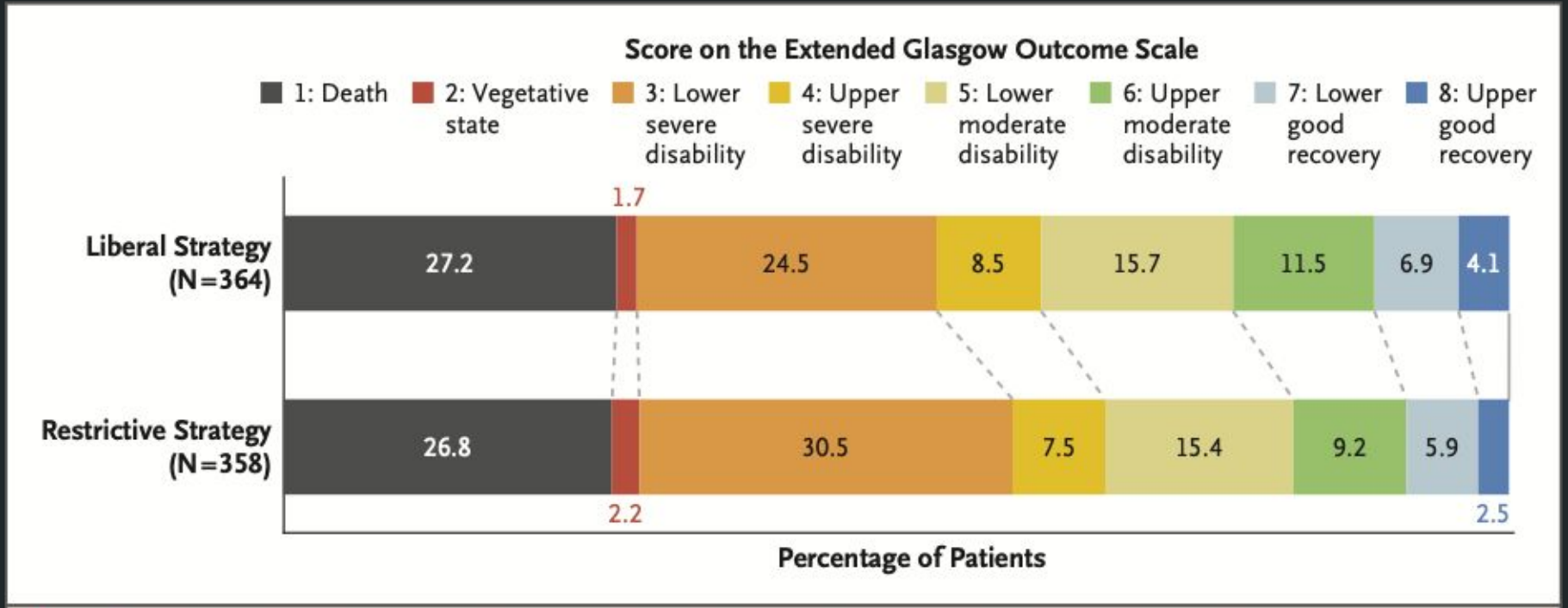


Figure 3. Scores on the Glasgow Outcome Scale–Extended at 6 Months According to Trial Group.

Température

Very early hypothermia induction in patients with severe brain injury (the National Acute Brain Injury Study: Hypothermia II): a randomised trial

Guy L Clifton, Alex Valadka, David Zygun, Christopher S Coffey, Pamala Drever, Sierra Fourwinds, L Scott Janis, Elizabeth Wilde, Pauline Taylor, Kathy Harshman, Adam Conley, Ava Puccio, Harvey S Levin, Stephen R McCauley, Richard D Bucholz, Kenneth R Smith, John H Schmidt, James N Scott, Howard Yonas, David O Okonkwo

	Poor outcome			Died		
	n (%)	RR (95% CI)	p value	n (%)	RR (95% CI)	p value
Primary analysis						
All patients (n=97)	56 (58%)	..	..	20 (21%)	..	..
Hypothermia (n=52)	31 (60%)	1·08 (0·76–1·53)	0·67	12 (23%)	1·30 (0·58–2·89)	0·52
Normothermia (n=45)	25 (56%)	..	..	8 (18%)	..	..
Subgroup analysis						
Diffuse brain injury (n=69)	42 (61%)	..	..	13 (19%)	..	..
Hypothermia (n=37)	26 (70%)	1·44 (0·95–2·17)	0·09	10 (27%)	2·88 (0·87–9·57)	0·08
Normothermia (n=32)	16 (50%)	..	..	3 (9%)	..	..
Surgically removed haematomas (n=28)	14 (50%)	..	..	7 (25%)	..	..
Hypothermia (n=15)	5 (33%)	0·44 (0·22–0·88)	0·02	2 (13%)	0·35 (0·08–1·50)	0·16
Normothermia (n=13)	9 (69%)	..	..	5 (39%)	..	..

Data are number (%). RR=relative risk.

Table 2: **Outcome and mortality rates**

Kevin

Hématome épidural D

Craniectomie D, volet non remis en place

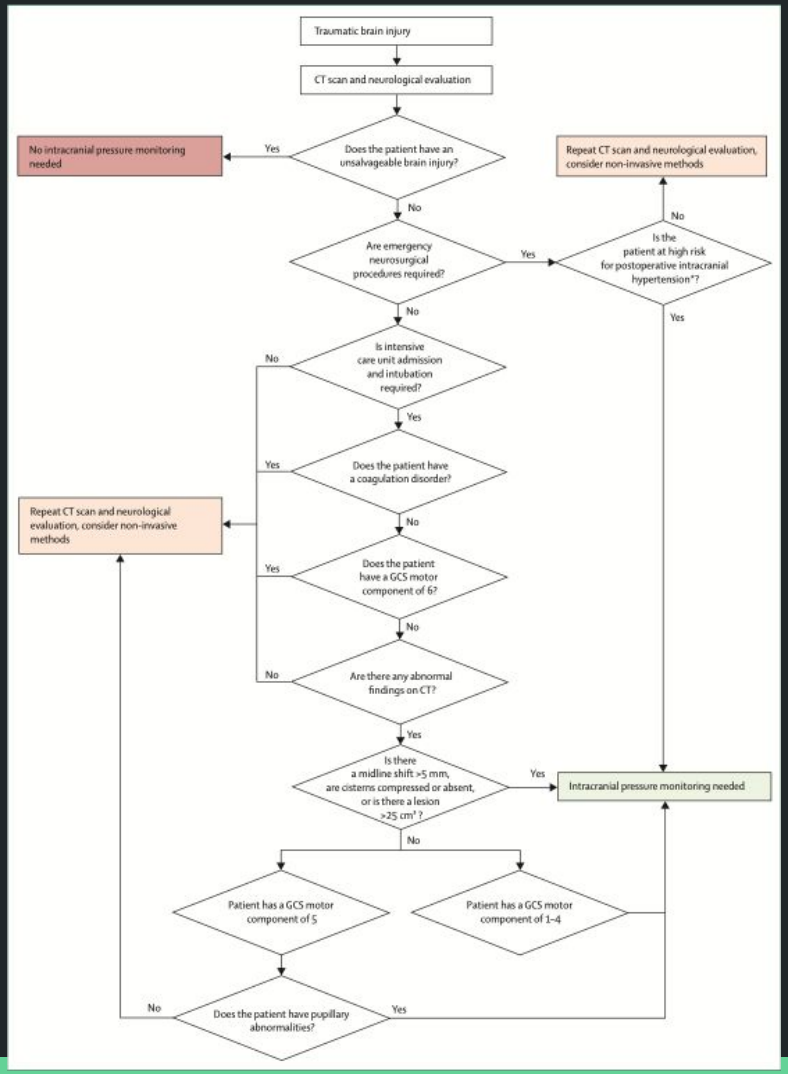
Moniteur de PIC mis en place - 13 mmHg

Jessica

Splénectomie/paquetage hépatique

Scan post op -> contusions bi frontales

Moniteur de PIC?


Traumatic brain injury
CT scan and neurological evaluation
Does the patient have an unsalvageable brain injury?
Yes
No intracranial pressure monitoring needed
No
Are emergency neurosurgical procedures required?
Yes
Is the patient at high risk for postoperative intracranial hypertension*?
No
Repeat CT scan and neurological evaluation, consider non-invasive methods
Yes
No
Is intensive care unit admission and intubation required?
Yes
Yes
Does the patient have a coagulation disorder?
No
Repeat CT scan and neurological evaluation, consider non-invasive methods
Yes
Does the patient have a GCS motor component of 6?
No
No
Are there any abnormal findings on CT?
Yes
Is there a midline shift >5 mm, are cisterns compressed or absent, or is there a lesion >25 cm³ ?
Yes
Intracranial pressure monitoring needed
No
Patient has a GCS motor component of 5
Patient has a GCS motor component of 1–4
No
Does the patient have pupillary abnormalities?
Yes

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*

Table 2. Clinical Outcomes.*

Variable	Pressure-Monitoring Group (N=157)	Imaging–Clinical Examination Group (N=167)	P Value	Proportional Odds Ratio (95% CI)†
Patients assessed at 6 mo — no. (%)	144 (92)	153 (92)		
Primary outcome‡			0.49§	1.09 (0.74–1.58)
Median	56	53		
Interquartile range	22–77	21–76		
Cumulative mortality at 6 mo — %	39	41	0.60¶	1.10 (0.77–1.57)
GOS-E scale at 6 mo — no. (%)‖				
Death	56 (39)	67 (44)**	0.40§	1.23 (0.77–1.96)
Unfavorable outcome	24 (17)	26 (17)		
Favorable outcome	63 (44)	60 (39)		

Ce que disent les lignes directrices

- Indiqué si GCS de 3-8 et scan anormal
- Indiqué si GCS de 3-8 et scan normal si
  - Âge > 40 ans
  - Posture motrice
  - Hypotension - pression artérielle systolique < 90 mmHg
- Une valeur de plus de 22 mmHg devrait être traitée

Jessica

Moniteur de PIC mis en place => 28 mmHg

Craniectomie décompressive?

Trial of Decompressive Craniectomy for Traumatic Intracranial Hypertension

P.J. Hutchinson, A.G. Kolias, I.S. Timofeev, E.A. Corteen, M. Czosnyka, J. Timothy, I. Anderson, D.O. Bulters, A. Belli, C.A. Eynon, J. Wadley, A.D. Mendelow, P.M. Mitchell, M.H. Wilson, G. Critchley, J. Sahuquillo, A. Unterberg, F. Servadei, G.M. Teasdale, J.D. Pickard, D.K. Menon, G.D. Murray, and P.J. Kirkpatrick, for the RESCUEicp Trial Collaborators*

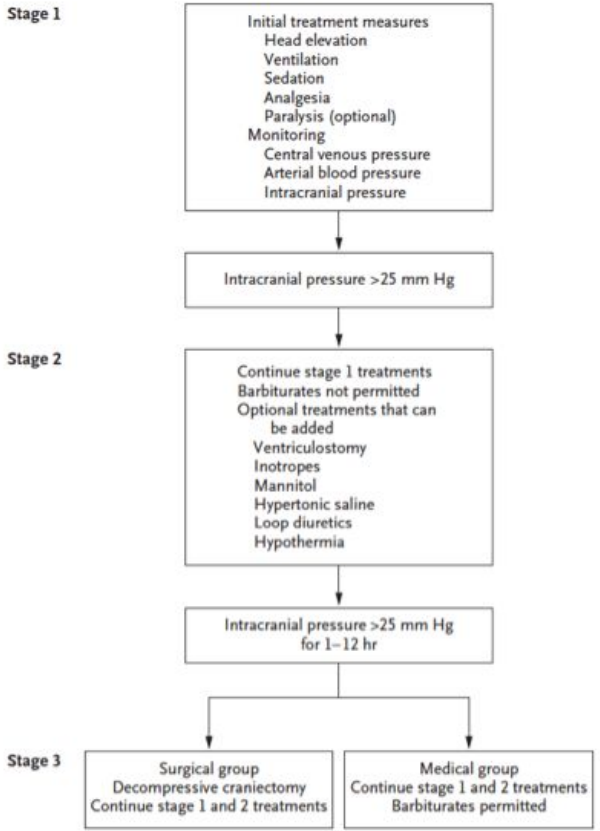
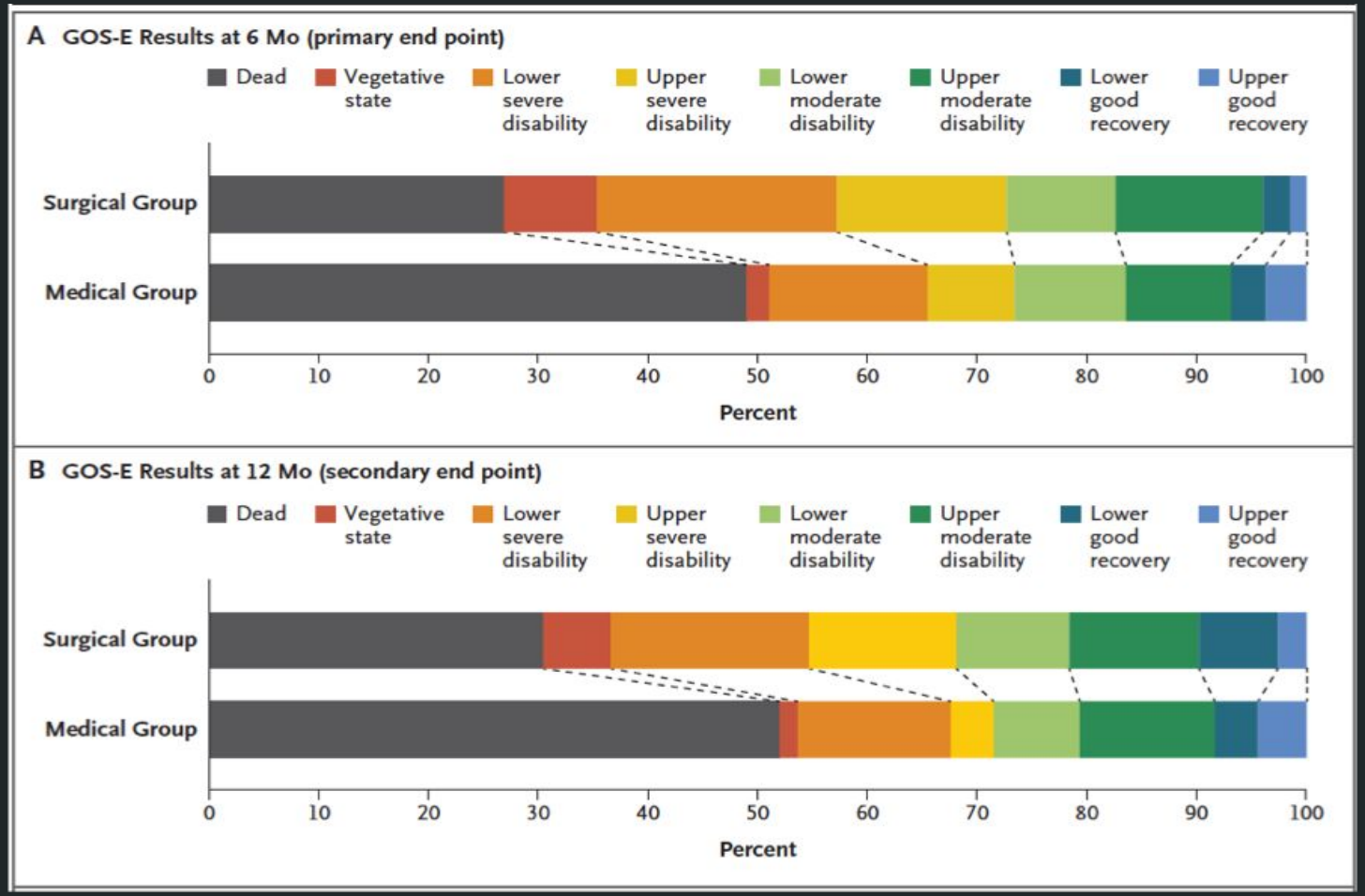


Figure 1. Stages of Therapeutic Management.

Agreement for participation was obtained from the nearest relative or a person who had been designated to give consent preemptively on admission of the patient in order to avoid delays in treatment. Randomization was performed after stage 2 if the intracranial pressure was more than 25 mm Hg for 1 to 12 hours. The protocol stages 1 and 2 reflected the therapeutic protocols that were followed in the participating units.

Table 1. Characteristics of the Patients at Baseline.*

Characteristic	Surgical Group (N = 202)	Medical Group (N = 196)
Age — yr	32.3±13.2	34.8±13.7
Male sex — no./total no. (%)	165/202 (81.7)	156/195 (80.0)
GCS motor score at first hospital — no./total no. (%)†		
1 or 2	96/181 (53.0)	85/170 (50.0)
3–6	85/181 (47.0)	85/170 (50.0)
Pupillary abnormality — no. (%)‡	59 (29.2)	57 (29.1)
Hypotension — no. (%)§	40 (19.8)	42 (21.4)
Hypoxemia — no. (%)¶	49 (24.3)	52 (26.5)
History of drug or alcohol abuse — no. (%)	50 (24.8)	69 (35.2)
Extracranial injury — no. (%)	75 (37.1)	83 (42.3)
Injury classification on basis of CT imaging — no./total no. (%)‖		
Diffuse injury	161/198 (81.3)	141/186 (75.8)
Mass lesion	37/198 (18.7)	45/186 (24.2)


A GOS-E Results at 6 Mo (primary end point)
Dead
Vegetative state
Lower severe disability
Upper severe disability
Lower moderate disability
Upper moderate disability
Lower good recovery
Upper good recovery
Surgical Group
Medical Group
0
10
20
30
40
50
60
70
80
90
100
Percent
B GOS-E Results at 12 Mo (secondary end point)
Dead
Vegetative state
Lower severe disability
Upper severe disability
Lower moderate disability
Upper moderate disability
Lower good recovery
Upper good recovery
Surgical Group
Medical Group
0
10
20
30
40
50
60
70
80
90
100
Percent

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Decompressive Craniectomy in Diffuse Traumatic Brain Injury

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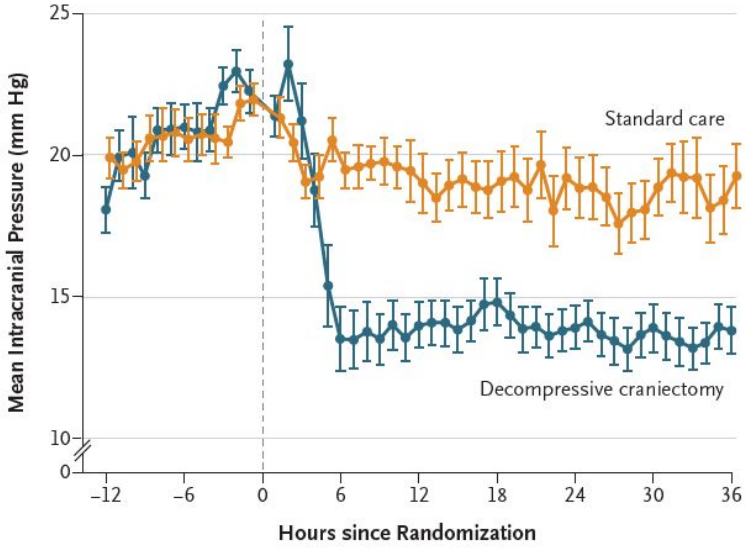


Figure 1. Intracranial Pressure before and after Randomization.

Shown are the mean measurements of intracranial pressure in the two study groups during the 12 hours before and the 36 hours after randomization. The I bars indicate standard errors.

Table 2. Primary and Secondary Outcomes.*

Outcome	Decompressive Craniectomy (N=73)	Standard Care (N=82)	P Value†
Intracranial pressure and cerebral perfusion pressure			
Intracranial pressure after randomization — mm Hg	14.4±6.8	19.1±8.9	<0.001
No. of hr of intracranial pressure >20 mm Hg — median (IQR)	9.2 (4.4–27.0)	30.0 (14.9–60.0)	<0.001
Intracranial hypertension index — median (IQR)‡	11.5 (5.9–20.3)	19.9 (12.5–37.8)	<0.001
Cerebral hypoperfusion index — median (IQR)§	5.7 (2.5–10.2)	8.6 (4.0–13.8)	0.03
Duration of hospital intervention			
Days of mechanical ventilation — median (IQR)	11 (8–15)	15 (12–20)	<0.001
Days of ICU stay — median (IQR)	13 (10–18)	18 (13–24)	<0.001
Days of hospitalization — median (IQR)	28 (21–62)	37 (24–44)	0.82
Extended Glasgow Outcome Scale			
Score — no. (%)			
1 (dead)	14 (19)	15 (18)	
2 (vegetative state)	9 (12)	2 (2)	
3 (lower severe disability)	18 (25)	17 (21)	
4 (upper severe disability)	10 (14)	8 (10)	
5 (lower moderate disability)	13 (18)	20 (24)	
6 (upper moderate disability)	6 (8)	13 (16)	
7 (lower good recovery)	2 (3)	4 (5)	
8 (upper good recovery)	1 (1)	3 (4)	
Median score (IQR)	3 (2–5)	4 (3–5)	0.03
Unfavorable score of 1 to 4 — no. (%)	51 (70)	42 (51)	0.02

Hypotension intracrânienne

Céphalée

Vertiges

NoVo

Symptômes visuels et auditifs

Changements cognitifs

Engagement

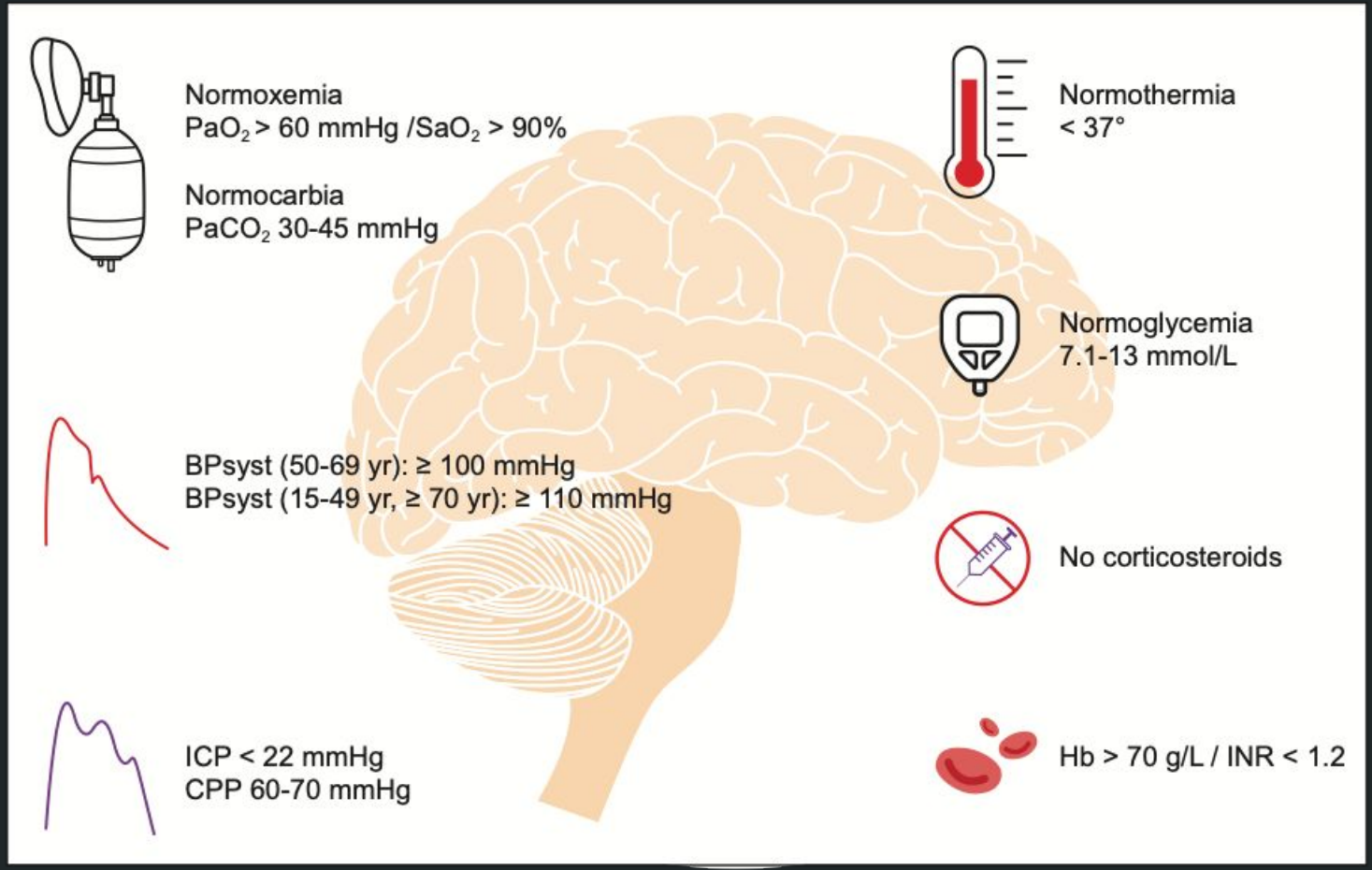


FIGURE 1. Overview of basic management principles for patients with traumatic brain injury.

On oubliait Bobby…

Après la laparotomie et la craniotomie, c’est au tour de l’orthopédiste de se manifester

Scan pré op?

Conduite anesthésique?

TABLE 6-4 INDICATIONS FOR CT SCANNING IN PATIENTS WITH MILD TBI

Head CT is required for patients with suspected mild brain trauma (i.e., witnessed loss of consciousness, definite amnesia, or witnessed disorientation in a patient with a GCS score of 13–15) *and* any one of the following factors:

High risk for neurosurgical intervention:	Moderate risk for brain injury on CT:
• GCS score less than 15 at 2 hours after injury • Suspected open or depressed skull fracture • Any sign of basilar skull fracture (e.g., hemotympanum, raccoon eyes, CSF otorrhea or rhinorrhea, Battle's sign) • Vomiting (more than two episodes) • Age more than 65 years • Anticoagulant use*	• Loss of consciousness (more than 5 minutes) • Amnesia before impact (more than 30 minutes) • Dangerous mechanism (e.g., pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from height more than 3 feet or five stairs)

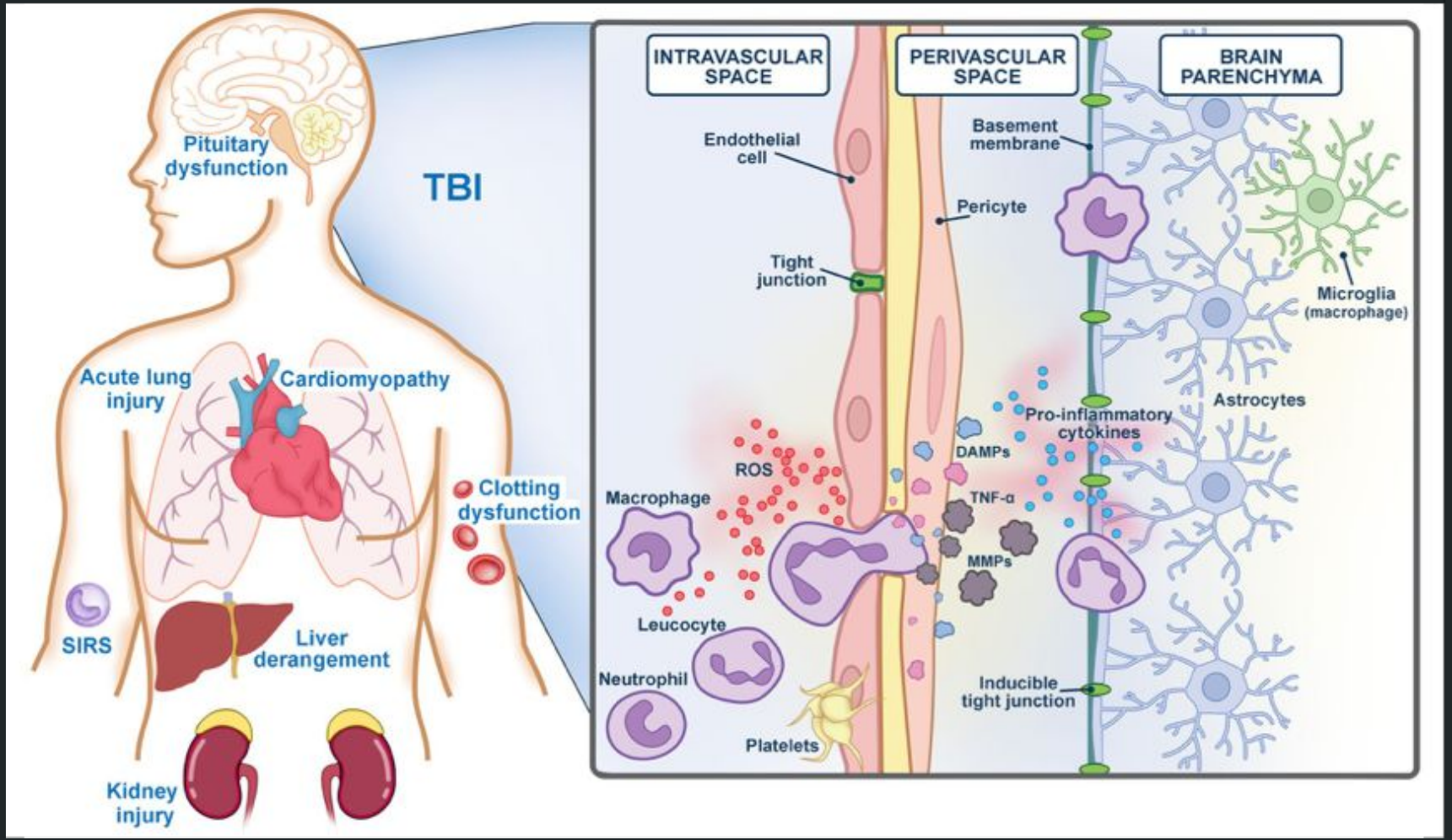
Source: Adapted from Stiell IG, Wells GA, Vandemheen K, et al. The Canadian CT Head Rule for patients with minor head injury. *Lancet* 2001; 357:1294.

Ok, on est rendu aux soins intensifs

Physiologie cérébrale post TCC

Monitoring

Buts


Pituitary dysfunction
TBI
Acute lung injury
Cardiomyopathy
Clotting dysfunction
SIRS
Liver derangement
Kidney injury
INTRAVASCULAR SPACE
PERIVASCULAR SPACE
BRAIN PARENCHYMA
Endothelial cell
Basement membrane
Pericyte
Tight junction
Microglia (macrophage)
Astrocytes
Pro-inflammatory cytokines
DAMPs
ROS
Macrophage
TNF-α
MMPs
Leucocyte
Neutrophil
Inducible tight junction
Platelets

TEMPERATURE
36°C - < 37.5°C (Core)

HEMOGLOBIN
8 – 12 gr/dL

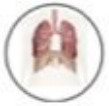
ELECTROLYTES AND ACID-BASIC STATUS
Na+ 135-145 mEq/L
pH 7.35 – 7.45
P50 26-28 mmHg

METABOLISM
$SvjO_2$ > 55%
$PtiO_2$ > 18 mmHg
CPP 60-70 mmHg

ARTERIAL BLOOD PRESSURE
SABP > 110 mmHg

NUTRITION AND GLUCOSE
Glycemia 110-180 mg/dL

TARGET OF OXYGEN
PaO_2 80 – 110 mmHg



LUNG PROTECTIVE VENTILATION
Vt 6-10 ml/kg PBW
RR to achieve $PaCO_2$ 35-45 mmHg
PP < 24
DP < 13 cm H_2O
MP < 17 J/min

EDEMA AND ICP CONTROL
ICP < 22 mmHg
ONSD < 5.5 mm; PI < 1.2
Serial CT scan

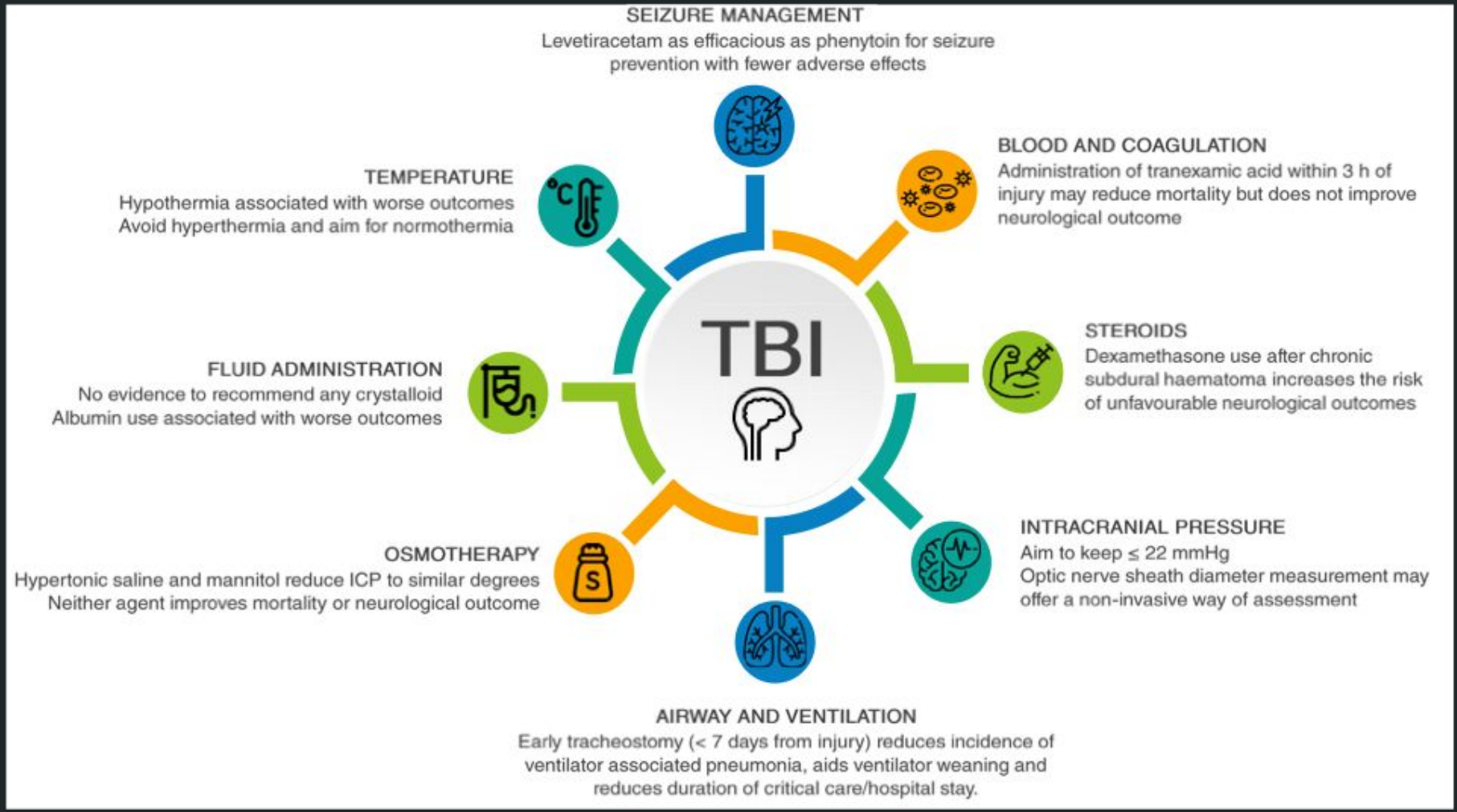

SEIZURE MANAGEMENT
Levetiracetam as efficacious as phenytoin for seizure prevention with fewer adverse effects
BLOOD AND COAGULATION
Administration of tranexamic acid within 3 h of injury may reduce mortality but does not improve neurological outcome
TEMPERATURE
Hypothermia associated with worse outcomes
Avoid hyperthermia and aim for normothermia
TBI
STEROIDS
Dexamethasone use after chronic subdural haematoma increases the risk of unfavourable neurological outcomes
FLUID ADMINISTRATION
No evidence to recommend any crystalloid
Albumin use associated with worse outcomes
INTRACRANIAL PRESSURE
Aim to keep ≤ 22 mmHg
Optic nerve sheath diameter measurement may offer a non-invasive way of assessment
OSMOTHERAPY
Hypertonic saline and mannitol reduce ICP to similar degrees
Neither agent improves mortality or neurological outcome
AIRWAY AND VENTILATION
Early tracheostomy (< 7 days from injury) reduces incidence of ventilator associated pneumonia, aids ventilator weaning and reduces duration of critical care/hospital stay.

Table 1 Summary of key recommendations from guidelines on the management of traumatic brain injury.

	National Institute for Health and Care Excellence: head injury: assessment and early management [7]	Brain Trauma Foundation: guidelines for the management of severe traumatic brain injury, 4th edn [8]	Association of Anaesthetists: guidelines for safe transfer of the brain-injured patient: trauma and stroke, 2019 [9]
Blood pressure	MAP ≥ 80 mmHg	SBP > 100 mmHg (age 50–69 y) SBP ≥ 110 mmHg (ages 15–49 and >70 y)	SBP 110–150 mmHg MAP > 90 mmHg
Ventilation	$PaCO_2$ 4.5–5.0 kPa PaO_2 > 13 kPa	Avoid $PaCO_2$ < 3.33 kPa	$PaCO_2$ 4.5–5.0 kPa PaO_2 ≥ 13 kPa
Intracranial pressure	No recommendation made	≤ 22 mmHg	No recommendation made
Cerebral perfusion pressure	No recommendation made	60–70 mmHg	No recommendation made
Steroids	No recommendation made	Not recommended	No recommendation made
Osmotherapy	No recommendation made	No recommendation made	Mannitol or hypertonic saline if impending uncal herniation
Temperature	No recommendation made	Prophylactic hypothermia not recommended	Maintain normothermia (36–37°C)

SBP, systolic blood pressure; MAP, mean arterial pressure.

Table 1. Unchallenged management principles and areas of debate in perioperative management of traumatic brain injury patients

Parameter	Unchallenged therapeutic goals	Areas of debate
SBP	>100 mmHg for patients ≥50–69 years old and >110 mmHg for patients 15–49 or over 70 years old	
CPP	60–70 mmHg	Autoregulation-oriented CPP management
ICP	Threshold 22 mmHg	Individual ICP thresholds
Airway management and respiratory management	P_aCO_2 in the low normal range	PEEP in TBI RSI or DSI
Temperature	Normothermia	
Glycemia	Normoglycemia	Slightly elevated values
Drugs	No steroids	Tranexamic acid Albumin

CPP, cerebral perfusion pressure; DSI, delayed sequence induction; ICP, intracranial pressure; MAP, mean arterial pressure; PEEP, positive end-expiratory pressure; RSI, rapid sequence induction; SBP, systolic blood pressure; TBI, traumatic brain injury.

Physiologie cérébrale lors de TCC

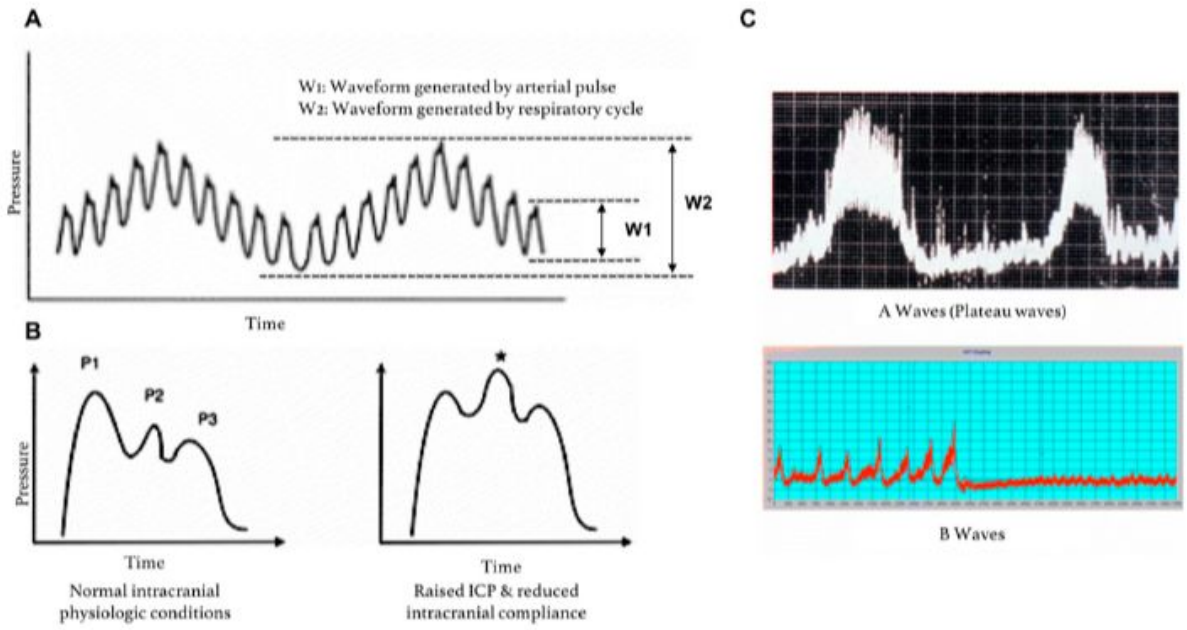
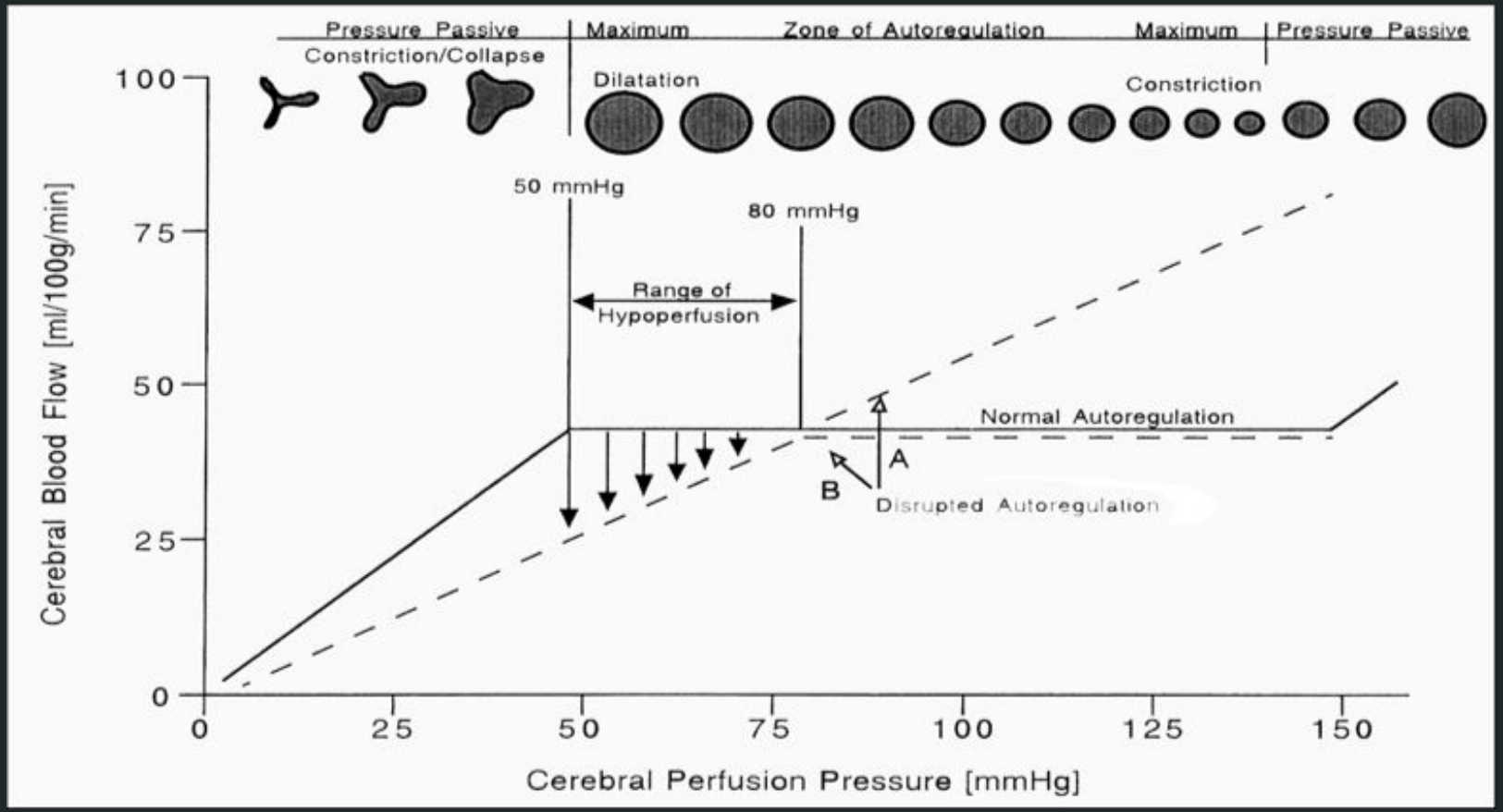
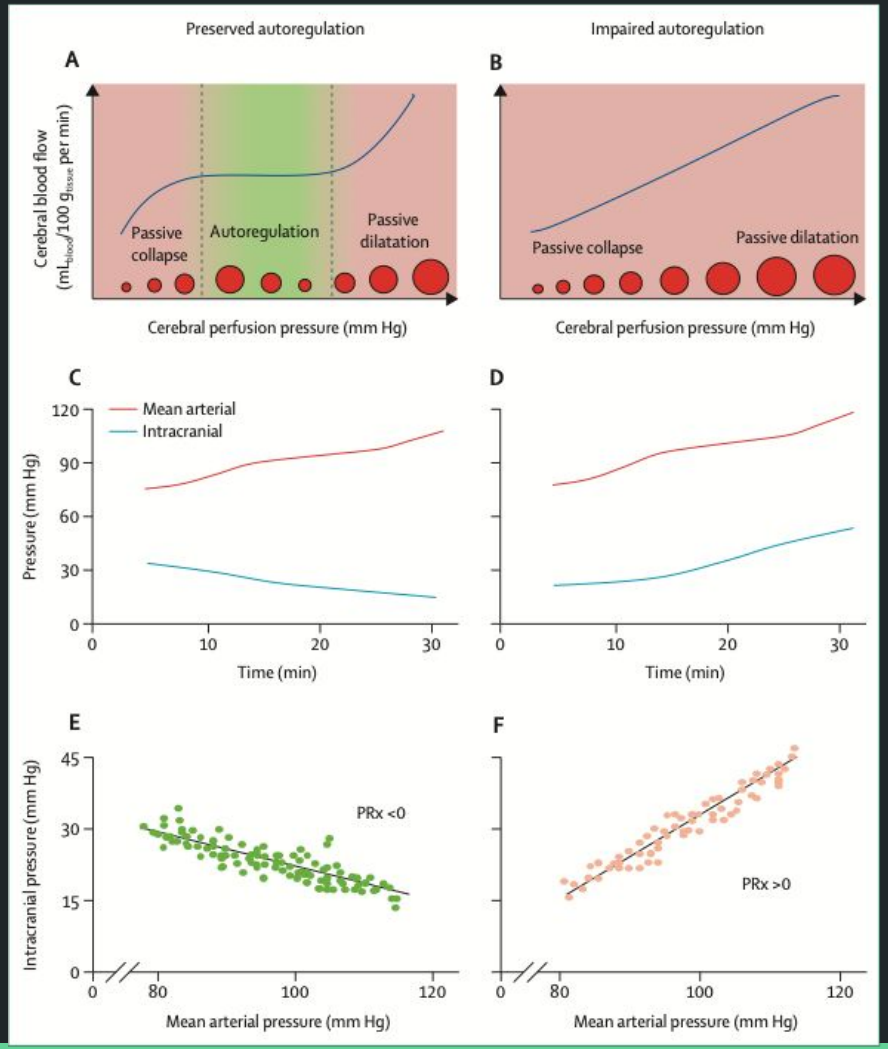
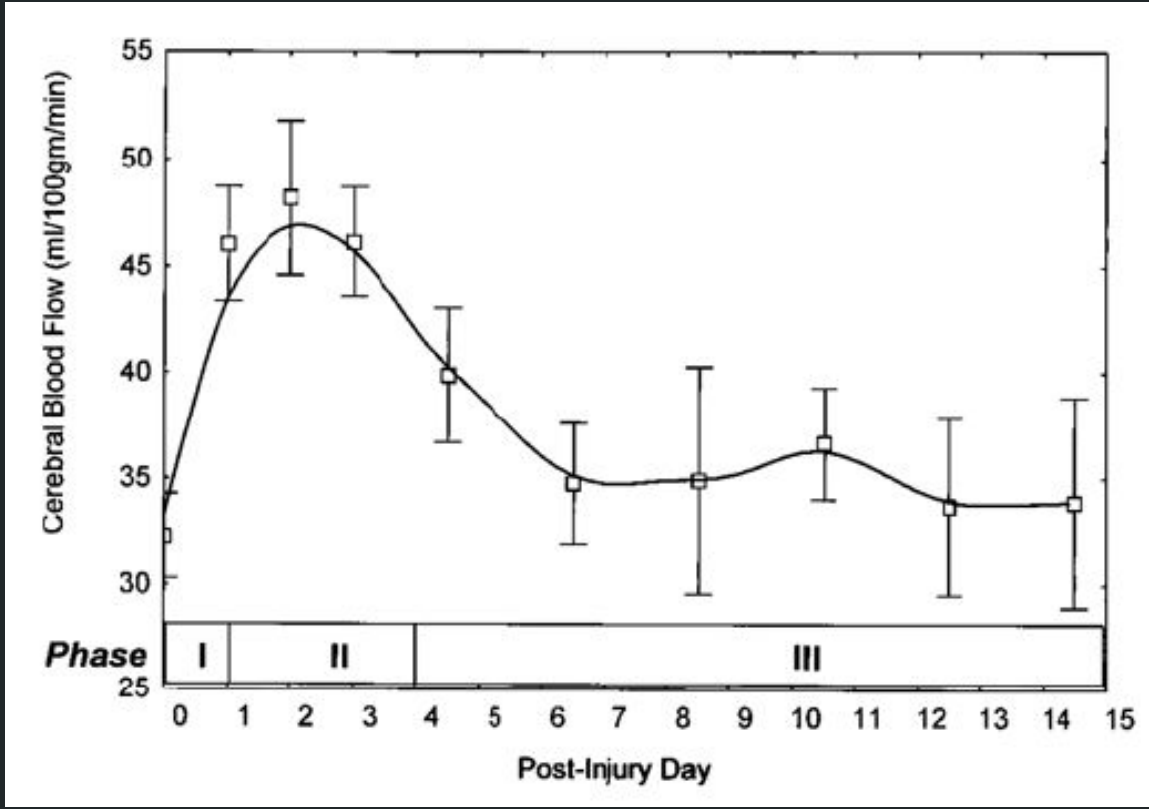


Figure 5. ICP pressure waves. (**A**) ICP fluctuations in response to the respiratory cycle (W2) and the arterial cycle (W1); (**B**) close-up of ICP waveform due to the systemic arterial cycle. Components are P1 (Percussion wave = representative of arterial pulsation), P2 (Tidal wave = a proxy for intracranial compliance) and P3 (Dicrotic wave = pressure transmission of aortic valve closure). A raised P2 wave is an indicator of raised ICP and reduced intracranial compliance (*); (**C**) Lundberg A (plateau) and B waves; adapted from Hall et al. [55].

Physiologie cérébrale lors de TCC




Preserved autoregulation
Impaired autoregulation
A
B
Cerebral blood flow (ml$_{blood}$/100 g$_{tissue}$ per min)
Passive collapse
Autoregulation
Passive dilatation
Passive collapse
Passive dilatation
Cerebral perfusion pressure (mm Hg)
Cerebral perfusion pressure (mm Hg)
C
D
Mean arterial
Intracranial
Pressure (mm Hg)
120
90
60
30
0
0
10
20
30
Time (min)
Time (min)
E
F
Intracranial pressure (mm Hg)
45
30
15
0
PRx <0
PRx >0
0
80
100
120
Mean arterial pressure (mm Hg)
Mean arterial pressure (mm Hg)


Cerebral Blood Flow (ml/100gm/min)
55
50
45
40
35
30
25
Phase
I
II
III
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
Post-Injury Day

Auparavant

2 grands types de thérapie

Rosner =>

cristalloïdes + vasopresseurs pour PPC > 70 mmHg

Lund =>

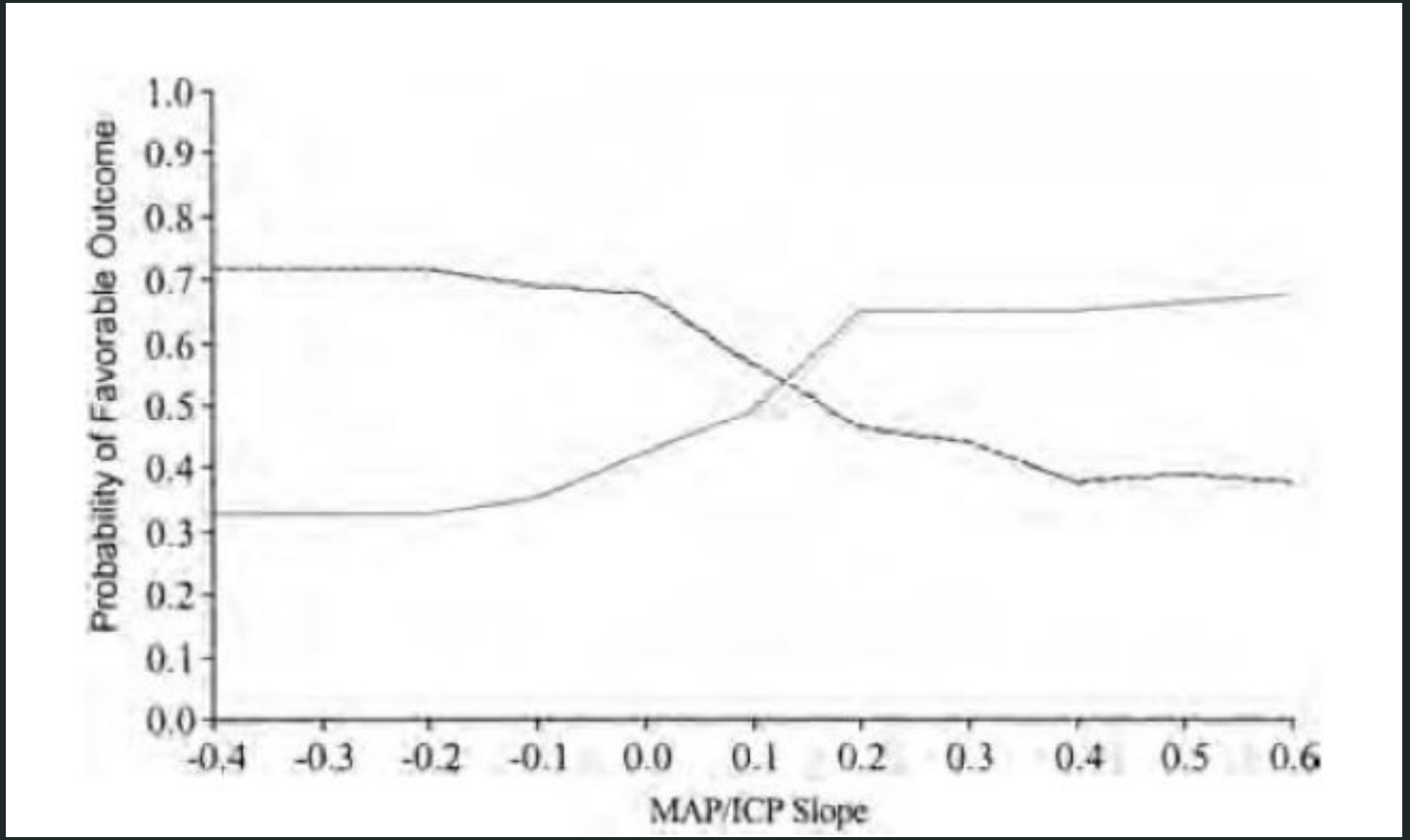
basée sur une diminution de la pression hydrostatique et un maintient de la pression oncotique

PPC 60-70, mais qui peut aller ad 50 mmHg

Metoprolol + clonidine

Albumine + Hb >120

Traitement à moduler en fonction de la physiologie du patient



Monitoring multimodal

Le manque de corrélation entre la PIC et la PPC et le devenir des patients a amené la recherche d’autres moyens de quantifier le flot sanguin cérébral et la livraison d’O2

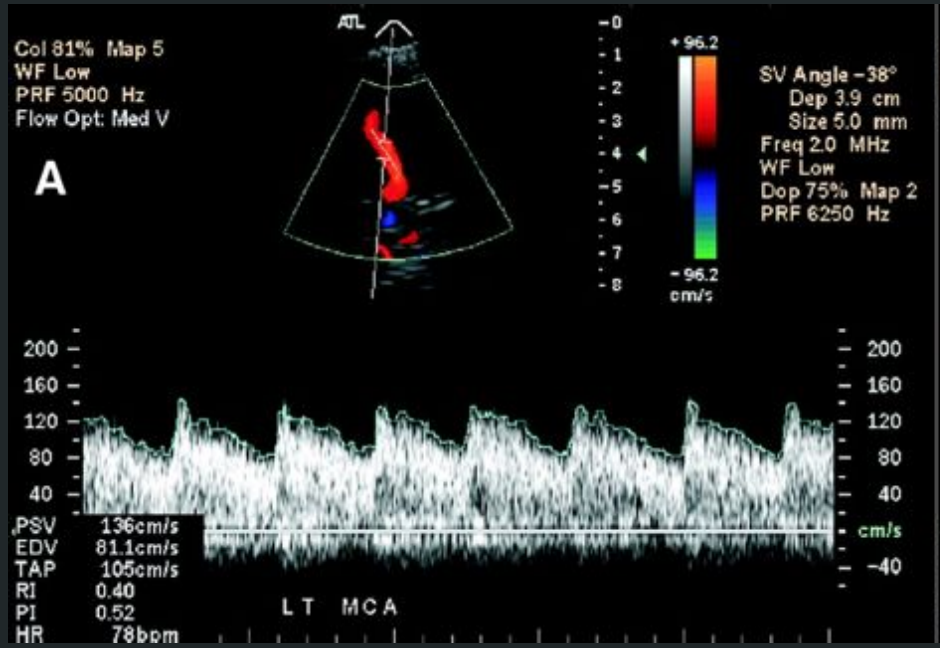
Mesure de la perfusion cérébrale

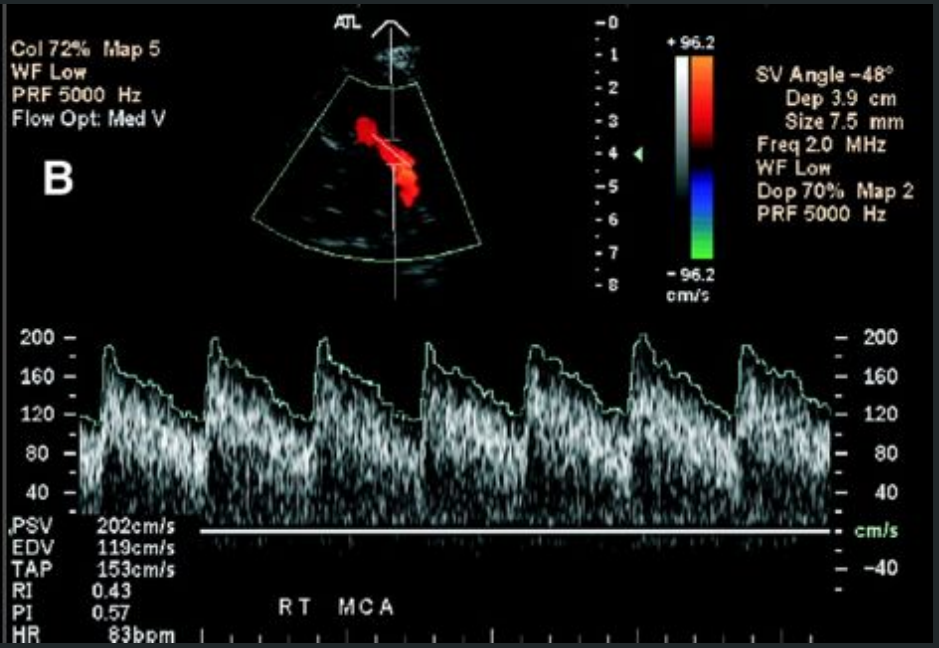
Méthodes variées pour le faire

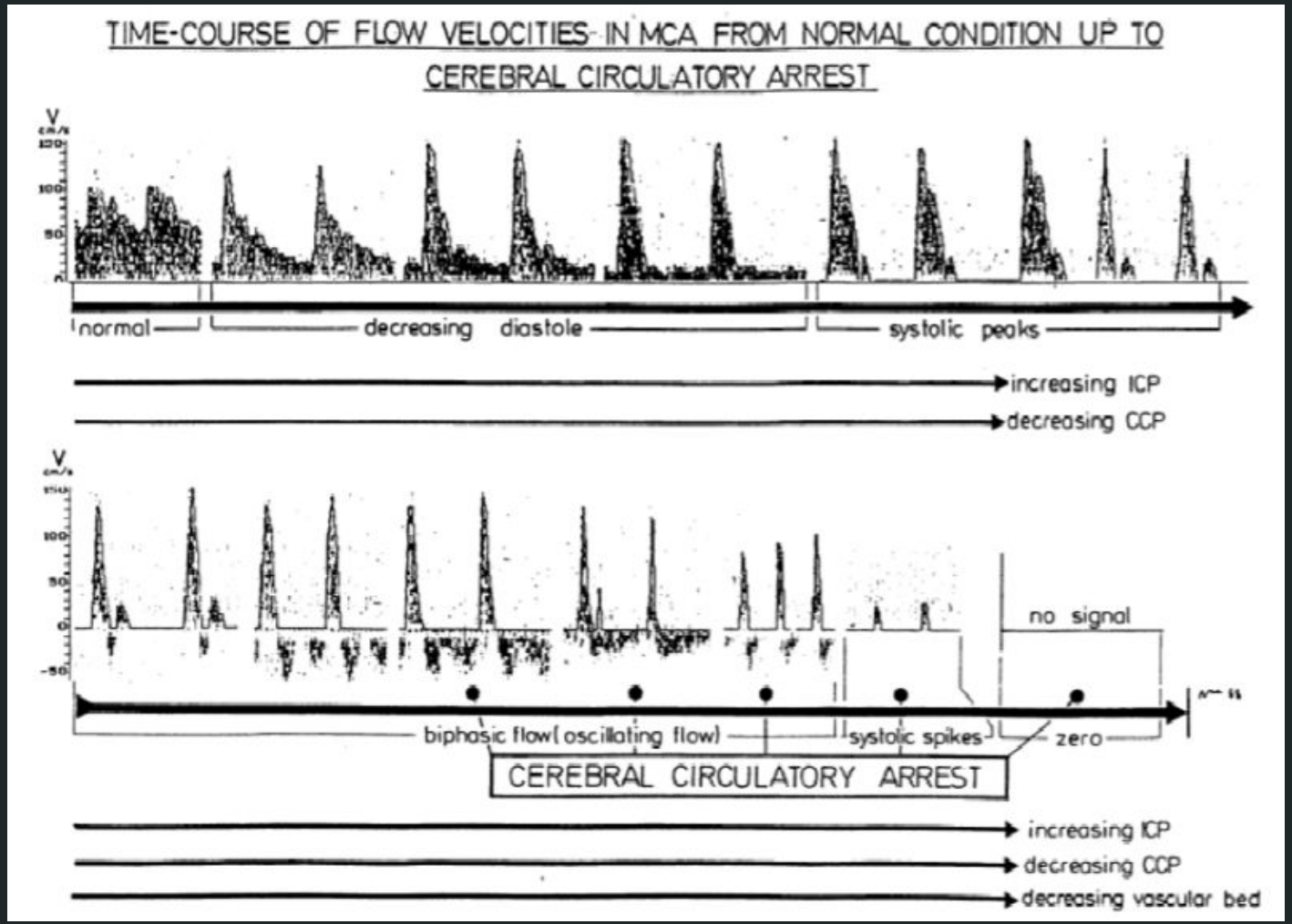
- **scan - RMN - PET : donnent beaucoup d’informations, mais constituent un «snapshot»**
- **doppler transcrânien**
- **cathéter intracrânien : *licox***

Doppler transcrânien

- **Utilise effet doppler pour mesurer la vélocité du flot sanguin cérébral (CBF)**
- **Doppler pulsé**
- **Courbe vélocité - temps**
- **Mesure directe de la vélocité systolique maximale et la vélocité en fin de diastole**


Col 81% Map 5
WF Low
PRF 5000 Hz
Flow Opt: Med V
A
ATL
+ 96.2
- 96.2
cm/s
SV Angle -38°
Dep 3.9 cm
Size 5.0 mm
Freq 2.0 MHz
WF Low
Dop 75% Map 2
PRF 6250 Hz
200
160
120
80
40
cm/s
-40
PSV 136cm/s
EDV 81.1cm/s
TAP 105cm/s
RI 0.40
PI 0.52
HR 78bpm
LT MCA


Col 72% Map 5
WF Low
PRF 5000 Hz
Flow Opt: Med V
B
ATL
+ 96.2
- 96.2
cm/s
SV Angle -48°
Dep 3.9 cm
Size 7.5 mm
Freq 2.0 MHz
WF Low
Dop 70% Map 2
PRF 5000 Hz
200
160
120
80
40
cm/s
-40
PSV 202cm/s
EDV 119cm/s
TAP 153cm/s
RI 0.43
PI 0.57
HR 83bpm
RT MCA


TIME-COURSE OF FLOW VELOCITIES IN MCA FROM NORMAL CONDITION UP TO CEREBRAL CIRCULATORY ARREST
V
normal
decreasing diastole
systolic peaks
increasing ICP
decreasing CCP
V
no signal
biphasic flow (oscillating flow)
systolic spikes
zero
CEREBRAL CIRCULATORY ARREST
increasing ICP
decreasing CCP
decreasing vascular bed

Doppler transcrânien

- Vélocité vs Débit sanguin cérébral
- Débit sanguin cérébral (cm^3 x s) = Vélocité (cm/s) x Aire du vaisseau (cm^2)
- Donc données fiables à calibre d'artère constant
- Études sur le calibre de l'artère cérébrale moyenne
- Quasi absence de variation du diamètre en réponse aux changements physiologiques
- Donc proportionnalité existe entre la vélocité et le débit sanguin
- Attention à l'hémorragie sous arachnoïdienne et au vasospasme!


LICOX®
Brain Tissue Oxygen Monitoring System


Camino
ICP
51
22.1
INTEGRA
Licox
341
21.4
INTEGRA

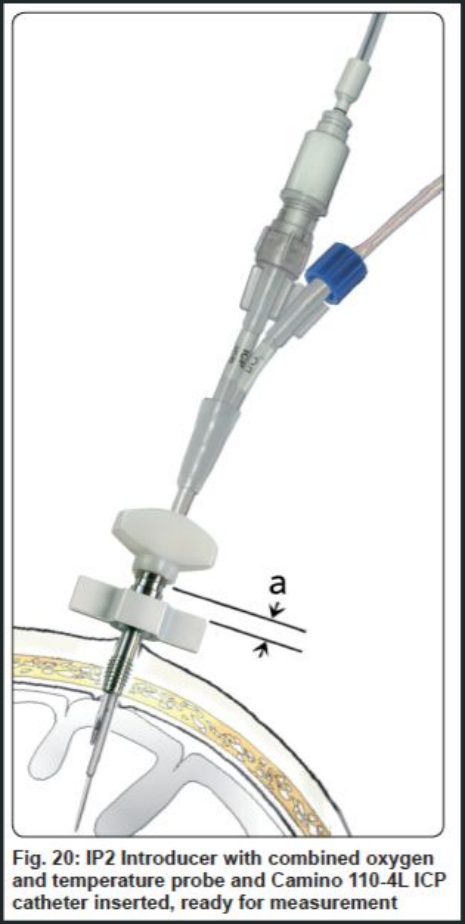
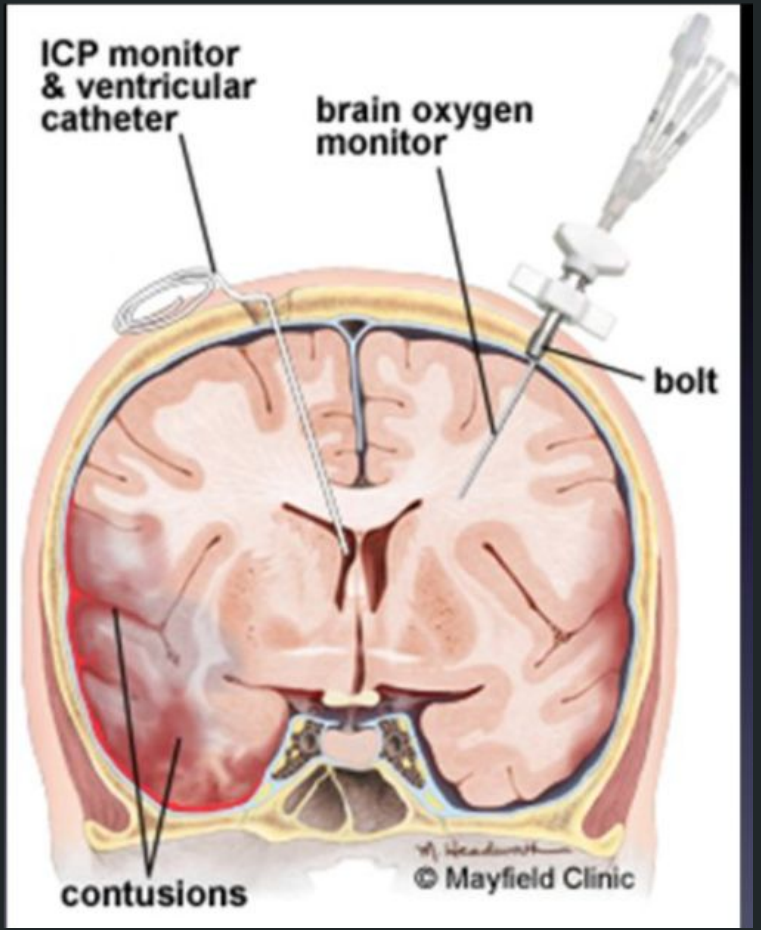
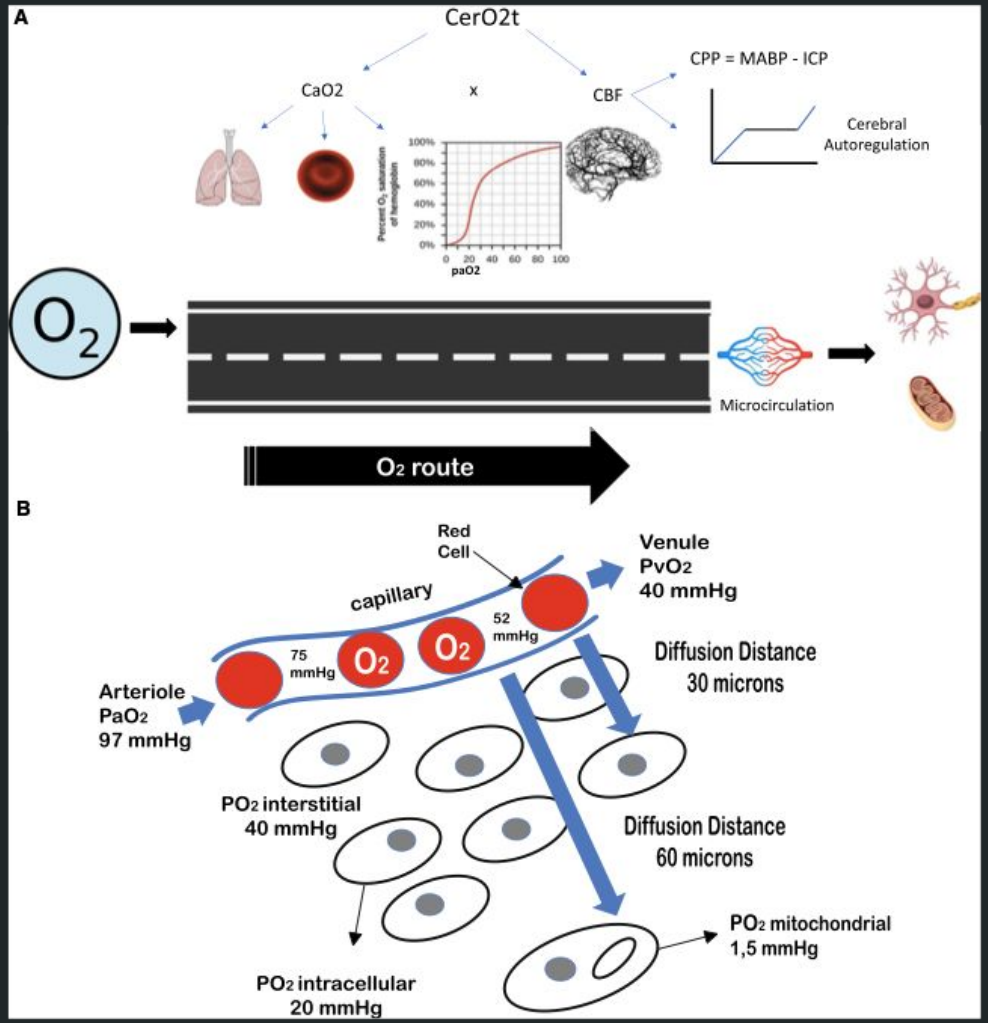


Fig. 20: IP2 Introducer with combined oxygen and temperature probe and Camino 110-4L ICP catheter inserted, ready for measurement




A
CerO2t
CaO2
x
CBF
CPP = MABP - ICP
Cerebral Autoregulation
100%
80%
60%
40%
20%
0%
0 20 40 60 80 100
paO2
O2
Microcirculation
O2 route
B
Red Cell
capillary
Venule
PvO2
40 mmHg
52 mmHg
75 mmHg
O2
O2
Arteriole
PaO2
97 mmHg
Diffusion Distance
30 microns
PO2 interstitial
40 mmHg
Diffusion Distance
60 microns
PO2 mitochondrial
1,5 mmHg
PO2 intracellular
20 mmHg

Oxygénation tissulaire cérébrale comme moniteur de flot sanguin

Livraison en O2 = débit sanguin cérébral x Hb x saturation

À consommation cérébrale en O2 constante, la saturation cérébrale en oxygène sera proportionnelle au débit sanguin cérébral

Vrai dans des conditions stables

- Hb
- Sat
- Température

Il y a une influence de la paO2 aussi sur la PbrO2

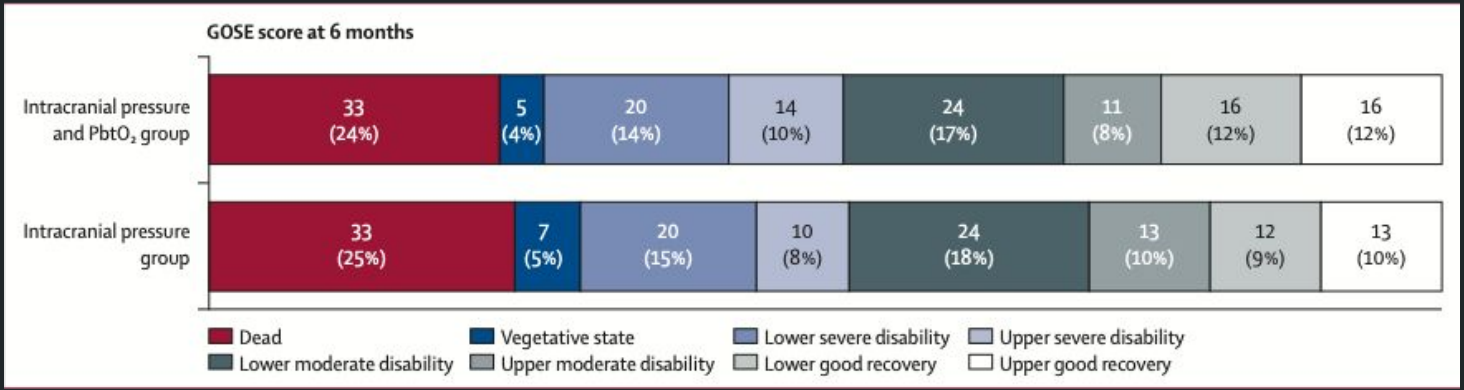
Brain Hypoxia Is Associated With Short-term Outcome After Severe Traumatic Brain Injury Independently of Intracranial Hypertension and Low Cerebral Perfusion Pressure

TABLE 5. Outcome in Patients With Intracranial Hypertension (Intracranial Pressure > 20 mm Hg) and Low Cerebral Perfusion Pressure (< 60 mm Hg) According to the Presence or Absence of Brain Hypoxia ($PbtO_2$ < 15 mm Hg)[a]

	Patients With Favorable Outcome, n (%)	
	Intracranial Hypertension (n = 74)	Low CPP (n = 75)
Brain hypoxia	20/43 (46)	18/46 (39)
No brain hypoxia	25/31 (81)	24/29 (83)
P	< .01	< .01

Intracranial pressure monitoring with and without brain tissue oxygen pressure monitoring for severe traumatic brain injury in France (OXY-TC): an open-label, randomised controlled superiority trial

*Jean-François Payen, Yoann Launey, Russell Chabanne, Samuel Gay, Gilles Francony, Laurent Gergele, Emmanuel Vega, Ambroise Montcriol, David Couret, Vincent Cottenceau, Sebastien Pili-Floury, Clement Gakuba, Emmanuelle Hammad, Gerard Audibert, Julien Pottecher, Claire Dahyot-Fizelier, Lamine Abdennour, Tobias Gauss, Marion Richard, Antoine Vilotitch, Jean-Luc Bosson, Pierre Bouzat for the OXY-TC trial collaborators**



ALGORITHME SIBICC DU TC GRAVE
POUR LES PATIENTS AVEC MONITORAGE DE PIC

Protocole détaillé conçu pour aider les cliniciens à prendre en charge les patients TC graves nécessitant un monitorage de PIC.
Ces recommandations sont basées sur une combinaison d'avis d'experts et ne reflètent ni une norme de soins ni un substitut à une prise en charge individualisée et réfléchie.

PRINCIPES D'UTILISATION DES PALIERS

- Si possible, utiliser le palier de traitement le plus bas.
- Il n'y a pas d'ordre d'application au sein d'un palier.
- Il n'est pas nécessaire d'utiliser toutes les modalités d'un palier avant de passer au suivant.
- Si c'est jugé bénéfique, il est possible de passer à un palier supérieur directement.

PALIER 0

Soins de base du TC grave – non-dépendants de la PIC :

Actions attendues:
- admission en réanimation
- Intubation endotrachéale et ventilation mécanique
- Evaluations répétées de l'état neurologique et de la réactivité pupillaire
- Surélévation de la tête de lit 30–45°
- Analgésie pour contrôler les signes de douleur (non guidée par la PIC)
- Sédation pour prévenir l'agitation, les asynchronies ventilatoires, etc… (non guidée par la PIC)
- Contrôle de la température pour prévenir la fièvre
  - Mesure de la température centrale
  - Traiter la température centrale au-dessus de 38°C
- Evaluer l'intérêt d'un traitement anti-épileptique pour 1 semaine seulement (sauf indication à poursuivre)
- Maintenir une PPC initiale ≥ 60mmHg
- Maintenir une Hb > 7g/dL
- Eviter l'hyponatrémie
- Optimiser le retour veineux cérébral (tête centrée dans l'axe, s'assurer que la minerve cervicale ne soit pas trop serrée)
- Cathéter artériel pour monitorage continu de la pression artérielle
- Maintenir la SpO_2 ≥ 94%

Actions recommandées:
- Mise en place d'une voie veineuse centrale
- Monitorage du CO_2 télé-expiratoire

PALIER 1

- Maintenir la PPC à 60–70 mmHg
- Augmenter l'analgésie pour diminuer la PIC
- Augmenter la sédation pour diminuer la PIC
- Maintenir la P_aCO_2 à la limite inférieure de la normale (35–38 mmHg/4.7–5.1 kP_a)
- Mannitol par bolus intermittent (0.25–1.0 g/kg)
- Sérum salé hypertonique par bolus intermittent[1]
- Drainage du LCS si DVE en place
- Envisager la pose de DVE pour drainer le LCS si utilisation initialement d'une fibre intraparenchymateuse
- Evaluer l'intérêt d'une prophylaxie anti-comitiale pour une semaine seulement (sauf indication à poursuivre)
- Envisager un monitorage EEG

PALIER 2

- Hypocapnie légère 32–35 mmHg/4.3–4.6 kP_a
- Curarisation chez les patients correctement sedatés si efficace[2]
- Effectuer une épreuve de variation de la PAM pour tester l'autorégulation cérébrale et guider les objectifs individuels de PAM et PPC des patients[3]
  - Devrait être réalisée sous la supervision directe d'un praticien qui peut évaluer la réponse et s'assurer de la sécurité de cette épreuve
  - Aucun autre ajustement thérapeutique (ex. sédation) ne devrait être réalisé pendant l'épreuve de PAM
  - Initier ou titrer un vasopresseur ou un inotrope pour augmenter la PAM de 10mmHg sur 20 minutes au plus.
  - Monitorer et enregistrer les paramètres clés (PAM, PPC, PIC et PtiO2) avant, pendant et après l'épreuve
  - Ajuster la dose de vasopresseur/inotrope basé sur les résultats de l'épreuve
- Monter la PPC avec des boli de fluides, des vasopresseurs et/ou inotropes pour baisser la PIC lorsque l'autorégulation est intacte

PALIER 3

- Coma induit par Pentothal ou Thiopental, titré pour contrôle de PIC si cette option est efficace[4]
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- Hypothermie légère (35–36°C) en utilisant des mesures actives de refroidissement.

- Ré-examiner cliniquement le patient avec répétition du scanner pour réévaluer les lésions intracrâniennes
- Discuter les options chirurgicales pour des lésions potentiellement chirurgicales
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- L'hypotension doit être évitée lorsque les barbituriques sont administrés

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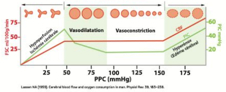
Noter les paramètres du moniteur à la fin de l'épreuve.

Evaluer les réponses observées et les valeurs enregistrées de l'état d'autorégulation cérébrale statique de la pression artérielle (sARC).

Une sARC altérée apparaitra comme une élévation soutenue de la PIC à mesure que la PAM augmente.

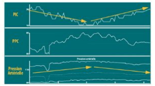
Ajuster la cible: retour à la PAM initiale (sARC altérée) ou choix de la nouvelle PAM (sARC intacte)

Courbe d'autorégulation

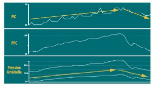


Lassen NA (1959). Cerebral blood flow and oxygen consumption in man. Physiol Rev 39, 183–238.

Autorégulation intacte



Autorégulation altérée



GCS SCORING

Indicateur du niveau de conscience	Terme utilisé: 1974	Terme utilisé: 2014
Ouverture des yeux	Spontanée	Spontanée
	A la voix	Au bruit
	A la douleur	A la pression
	Aucune	Aucune
Réponse verbale	Orientée	Orientée
	Discours confus	Confuse
	Propos Inappropriés	Mots
	Propos incompréhensibles	Sons
	Aucune	Aucune
Réponse motrice	A la demande	A la demande
	Localisatrise	Orientée
	Flexion normale	Evitement
	Flexion anormale	Flexion stéréotypée
	Extension	Extension stéréotypée
	Aucune	Aucune

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DETERIORATION NEUROLOGIQUE SEVERE

Une détérioration sévère de l'état clinique neurologique qui requiert une réponse immédiate du médecin telle que:
- Une diminution spontanée du GCS moteur ≥ 1 point (comparé à l'examen précédent)
- Une nouvelle diminution de la réactivité pupillaire
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ACTION FACE A UNE DETERIORATION NEUROLOGIQUE SEVERE

Evaluation urgente pour identifier une cause possible de détérioration. Si suspicion d'engagement:
- Traitement empirique
  - Hyperventilation[5]
  - Bolus de soluté hypertonique
- Envisager une imagerie urgente ou un autre examen
- Intensification rapide du traitement

[5] La limite de P_aCO_2 d'hyperventilation de 30 mmHg/ 4.0 kP_a ne s'applique pas ici

CAUSES POSSIBLES DE DETERIORATION NEUROLOGIQUE

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AP: Anomalie pupillaire; TDM=Tomodensitométrie; LD: Lésions diffuses définies selon le score TDM cérébrale de Marshall;
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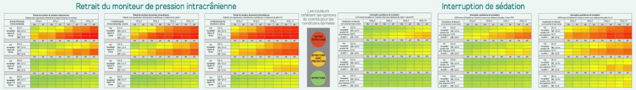
Retrait du moniteur de pression intracrânienne — Interruption de sédation



TRAITEMENTS NON RECOMMANDES

Mannitol par perfusion intraveineuse continue
Perfusion systématique d'osmothérapie (i.e. toutes les 4-6h)
Drainage lombaire du LCS
Furosemide
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Utilisation en routine de l'hypothermie thérapeutique au-dessous de 35°C car risque de complications systémiques
Doses élevées de propofol pour atteindre la burst-suppression
Maintien prolongé de la P_aCO_2 au-dessous de 30 mmHg/4.0 kP_a
Maintien prolongé de la PPC au-delà de 90 mmHg

Soutenu par Global Neuro, Brain Trauma Foundation

Pour des informations complémentaires, se reporter à:
1. A management algorithm for patients with intracranial pressure monitoring: The Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC)
2. Echelle de Glasgow
3. Guidelines for the management of severe TBI 4th edition
4. Article du Score TDM de Marschall
5. Présentation de Randall Chesnut à CNS 2019

Auteurs et participants de la réunion: Gregory W. J. Hawryluk, Sergio Aguilera, Andras Buki, Eileen Bulger, Giuseppe Citerio, D. Jamie Cooper, Ramon Diaz Arrastia, Michael Diringer, Anthony Figaji, Guoyi Gao, Romergryko Geocadin, Jamshid Ghajar, Odette Harris, Alan Hoffer, Peter Hutchinson, Mathew Joseph, Ryan Kitagawa, Geoffrey Manley, Stephan Mayer, David K. Menon, Geert Meyfroidt, Daniel B. Michael, Mauro Oddo, David Okonkwo, Mayur Patel, Claudia Robertson, Jeffrey V. Rosenfeld, Andres M. Rubiano, Juan Sahuquillo, Franco Servadei, Lori Shutter, Deborah Stein, Nino Stocchetti, Fabio Silvio Taccone, Shelly Timmons, Eve Tsai, Jamie S. Ullman, Paul Vespa, Walter Videtta, David W. Wright, Christopher Zammit and Randall Chesnut; Creation du poster: Randall Chesnut, Gregory Hawryluk, Karen March, Linda Littlejohns, Tyler Harmon, Global Neuro; Traduction française: Yoann Launey, Fabio Taccone, EURONEURO

ALGORITHME SIBICC DU TC GRAVE

POUR LES PATIENTS AVEC MONITORAGE DE PIC

Protocole détaillé conçu pour aider les cliniciens à prendre en charge les patients TC graves nécessitant un monitorage de PIC.
Ces recommandations sont basées sur une combinaison d'avis d'experts et ne reflètent ni une norme de soins ni un substitut à une prise en charge individualisée et réfléchie.

PRINCIPES D'UTILISATION DES PALIERS

- Si possible, utiliser le palier de traitement le plus bas.
- Il n'y a pas d'ordre d'application au sein d'un palier.
- Il n'est pas nécessaire d'utiliser toutes les modalités d'un palier avant de passer au suivant.
- Si c'est jugé bénéfique, il est possible de passer à un palier supérieur directement.

PALIER 0

Soins de base du TC grave – non-dépendants de la PIC :

Actions attendues:

- admission en réanimation
- Intubation endotrachéale et ventilation mécanique
- Evaluations répétées de l'état neurologique et de la réactivité pupillaire
- Surélévation de la tête de lit 30-45°
- Analgésie pour contrôler les signes de douleur (non guidée par la PIC)
- Sédation pour prévenir l'agitation, les asynchronies ventilatoires, etc... (non guidée par la PIC)
- Contrôle de la température pour prévenir la fièvre
  - Mesure de la température centrale
  - Traiter la température centrale au-dessus de 38°C
- Evaluer l'intérêt d'un traitement anti-épileptique pour 1 semaine seulement (sauf indication à poursuivre)
- Maintenir une PPC initiale ≥ 60mmHg
- Maintenir une Hb > 7g/dL
- Eviter l'hyponatrémie
- Optimiser le retour veineux cérébral (tête centrée dans l'axe, s'assurer que la minerve cervicale ne soit pas trop serrée)
- Cathéter artériel pour monitorage continu de la pression artérielle
- Maintenir la $SpO_2 \geq 94\%$

Actions recommandées:

- Mise en place d'une voie veineuse centrale
- Monitorage du CO_2 télé-expiratoire

PALIER 1

- Maintenir la PPC à 60–70 mmHg
- Augmenter l'analgésie pour diminuer la PIC
- Augmenter la sédation pour diminuer la PIC
- Maintenir la P_aCO_2 à la limite inférieure de la normale (35–38 mmHg/4.7–5.1 kP_a)
- Mannitol par bolus intermittent (0.25–1.0 g/kg)
- Sérum salé hypertonique par bolus intermittent[1]
- Drainage du LCS si DVE en place
- Envisager la pose de DVE pour drainer le LCS si utilisation initialement d'une fibre intraparenchymateuse
- Evaluer l'intérêt d'une prophylaxie anti-comitiale pour une semaine seulement (sauf indication à poursuivre)
- Envisager un monitorage EEG

PALIER 2

- Hypocapnie légère 32–35 mmHg/4.3–4.6 kP_a
- Curarisation chez les patients correctement sedatés si efficace[2]
- Effectuer une épreuve de variation de la PAM pour tester l'autorégulation cérébrale et guider les objectifs individuels de PAM et PPC des patients[3]
  - Devrait être réalisée sous la supervision directe d'un praticien qui peut évaluer la réponse et s'assurer de la sécurité de cette épreuve
  - Aucun autre ajustement thérapeutique (ex. sédation) ne devrait être réalisé pendant l'épreuve de PAM
  - Initier ou titrer un vasopresseur ou un inotrope pour augmenter la PAM de 10mmHg sur 20 minutes au plus.
  - Monitorer et enregistrer les paramètres clés (PAM, PPC, PIC et PtiO2) avant, pendant et après l'épreuve
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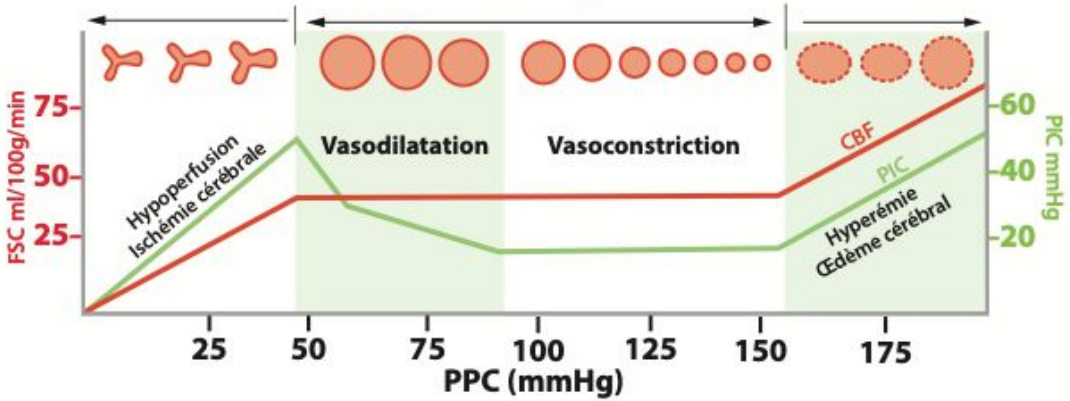
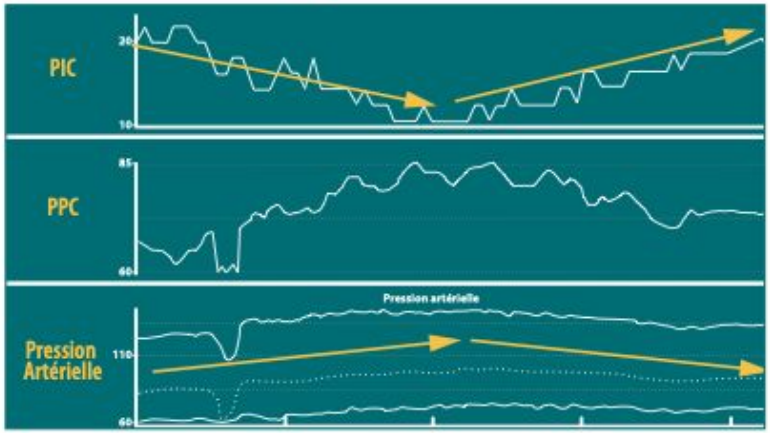
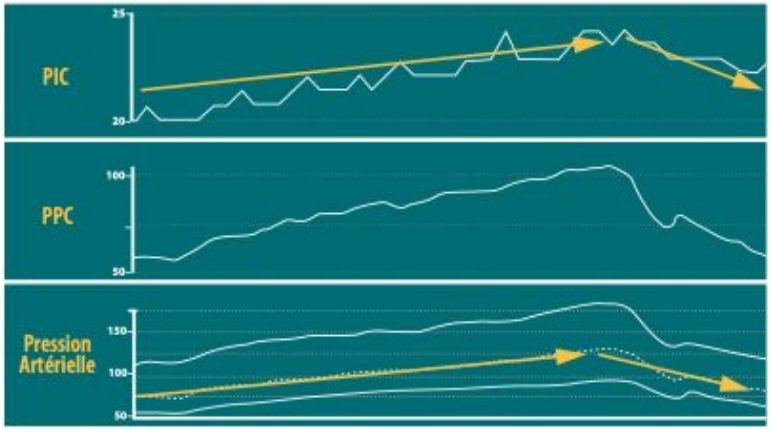


Lassen NA (1959). Cerebral blood flow and oxygen consumption in man. Physiol Rev. 39, 183–238.

Autorégulation intacte



Autorégulation altérée



HEATMAPS INFORMANT SUR LA SECURITE D'UN ARRET DE SEDATION ET DU RETRAIT DU MONITEUR DE PIC

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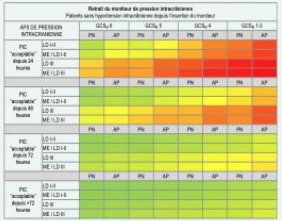
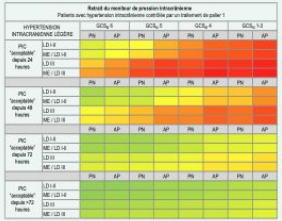
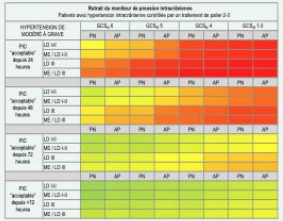
Retrait du moniteur de pression intracrânienne

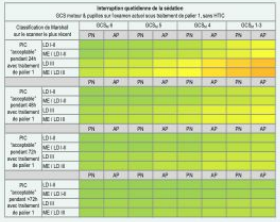


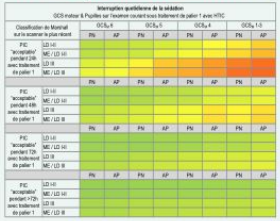


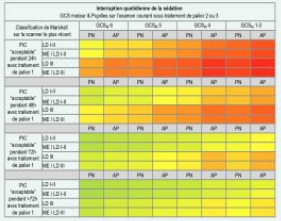


Les couleurs reflètent les opinions du comité pour les conditions données

Interruption de sédation







SIBICC SEVERE TBI ALGORITHM

FOR PATIENTS WITH ICP AND BRAIN TISSUE OXYGEN MONITORING

A comprehensive protocol designed to assist clinicians managing sTBI patients undergoing ICP and $P_{bt}O_2$ monitoring.
These recommendations are based on combined expert opinion and reflect neither a standard-of-care nor a substitute for thoughtful individualized management.

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BASIC CARE Applies to all Severe TBI Patients

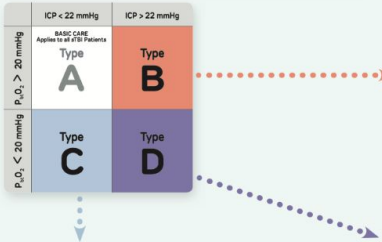
TIER 0

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 ≥ 60mmHg
- 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 $SpO_2 \geq 94\%$

Recommended Interventions:

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



TYPE B ICP Elevated – Brain Oxygenation Normal

TIER 1

- Maintain CPP 60-70 mmHg
- Increase analgesia to lower ICP
- Increase sedation to lower ICP
- Maximum P_aCO_2 at low end of normal (35-38 mmHg/4.7-5.1 kPa)
- CSF drainage if EVD in situ
- Mannitol by intermittent bolus (0.25-1.0 g/kg)
- Hypertonic saline by intermittent bolus*
- Consider anti-seizure prophylaxis for one week only (unless indication to continue)
- Consider EEG monitoring
- Reexamine the patient and consider repeat CT to reevaluate 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 2

- Mild hypocapnia (range 32-35mmHg/ 4.3-4.6 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 10mmHg 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

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

* 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, Sanchez-Mejia RO, Phan N, Hemphill JC 3rd, Martin C, Manley GT. Incorporating a parenchymal thermal diffusion cerebral blood flow probe in bedside assessment of cerebral autoregulation and vasoreactivity in patients with severe traumatic brain injury. J Neurosurg. 2011;114(1):62-70. doi:10.3171/2010.5.JNS09360.

§ Barbiturate administration should only be continued when a beneficial effect on ICP is demonstrated.
- Titrate barbiturates to achieve ICP control but do not exceed the dose which achieves burst suppression.
- Hypotension must be avoided when barbiturates are administered.

TYPE C ICP Normal – Brain Hypoxic

TIER 1

- Maximum CPP 60 – 70 mmHg
- Increase CPP to a maximum of 70 mmHg with fluid, vasopressors and/or inotropes
- Maintain P_aCO_2 > (35 mmHg/4.7 kPa)
- If $P_{bt}O_2$ is already in desired range, further increase $P_{bt}O_2$ by increasing FiO_2 to 60%
- Consider EEG monitoring
- Reexamine the patient, and consider repeat CT to reevaluate 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 2

- Ventilator management to increase $P_{bt}O_2$ as high as 150 mmHg/20 kPa
- Increase CPP 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 adjustment (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 70mmHg with fluid boluses, vasopressors and/or inotropes§

TIER 3

- Increase P_aCO_2 to 45-50 mmHg/6.0-6.7 kPa (but avoid intracranial hypertension)
- Consider normobaric hyperoxia to a $P_{bt}O_2$ above 150 mmHg/20 kPa
- If $P_{bt}O_2$ remains < 20 mmHg despite $P_{bt}O_2$ and CPP/MAP optimization, consider transfusing 1 unit of PRBCs if Hgb <9g/L

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

‡ Rosenthal G, Sanchez-Mejia RO, Phan N, Hemphill JC 3rd, Martin C, Manley GT. Incorporating a parenchymal thermal diffusion cerebral blood flow probe in bedside assessment of cerebral autoregulation and vasoreactivity in patients with severe traumatic brain injury. J Neurosurg. 2011;114(1):62-70. doi:10.3171/2010.5.JNS09360.

§ Careful monitoring for respiratory complications is required when CPP is raised above 70 mmHg (Robertson CS, et al. Prevention of secondary ischemic insults after severe head injury. Crit Care Med. 1999;27(10):2086-2095).

TYPE D ICP Elevated – Brain Hypoxic

TIER 1

- Maintain CPP 60 – 70 mmHg
- 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 $P_{bt}O_2$ is already in desired ranged, further increase $P_{bt}O_2$ by increasing FiO_2 to 60%
- Consider anti-seizure prophylaxis for 1 week only (unless indication to continue)
- Consider EEG monitoring
- Reexamine the patient and consider repeat CT to reevaluate 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 2

- Ventilator management to increase $P_{bt}O_2$ as high as 150 mmHg/20 kPa
- 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 adjustment (i.e. 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 70mmHg with fluid boluses, vasopressors and/or inotropes§

TIER 3

- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious#
- Secondary decompressive craniectomy
- Consider normobaric hyperoxia to a $P_{bt}O_2$ above 150 mmHg/20 kPa
- If $P_{bt}O_2$ remains < 20 mmHg despite $P_{bt}O_2$ and CPP/MAP optimization, consider transfusing 1 unit of PRBCs if Hgb <9g/L

* 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, Sanchez-Mejia RO, Phan N, Hemphill JC 3rd, Martin C, Manley GT. Incorporating a parenchymal thermal diffusion cerebral blood flow probe in bedside assessment of cerebral autoregulation and vasoreactivity in patients with severe traumatic brain injury. J Neurosurg. 2011;114(1):62-70. doi:10.3171/2010.5.JNS09360.

§ Careful monitoring for respiratory complications is required when CPP is raised above 70 mmHg (Robertson CS, et al. Prevention of secondary ischemic insults after severe head injury. Crit Care Med. 1999;27(10):2086-2095).

§ Barbiturate administration should only be continued when a beneficial effect on ICP is demonstrated.
- Titrate barbiturates to achieve ICP control but do not exceed the dose which achieves burst suppression.
- Hypotension must be avoided when barbiturates are administered.

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

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

† The 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

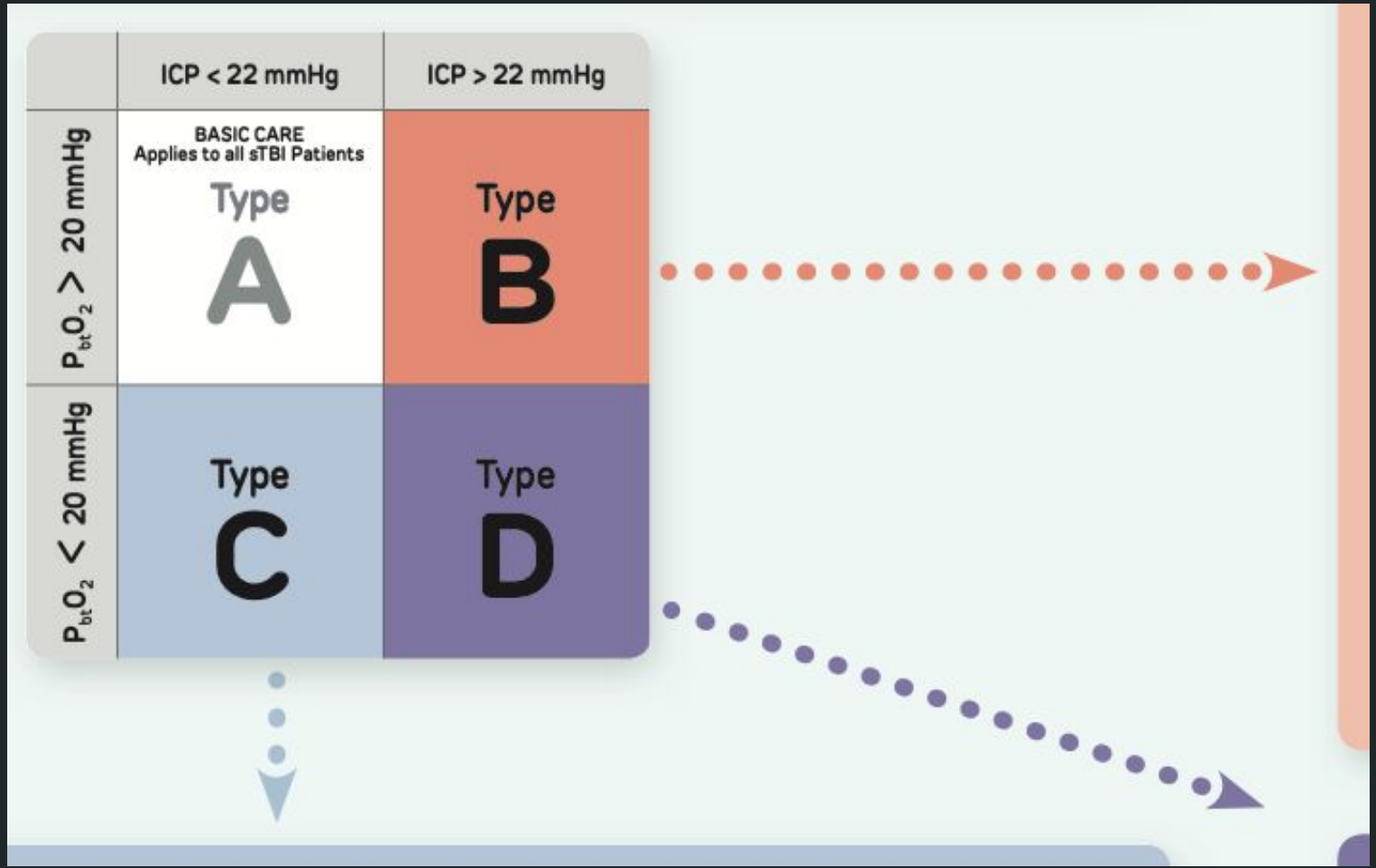
Meeting Participants and Authors: Gregory W. J. Hawryluk, Sergio Aguilera, Andras Buki, Eileen Bulger, Giuseppe Citerio, D. Jamie Cooper, Ramon Diaz Arrastia, Michael Diringer, Anthony Figaji, Guoyi Gao, Romergryko Geocadin, Jamshid Ghajar, Odette Harris, Alan Hoffer, Peter Hutchinson, Mathew Joseph, Ryan Kitagawa, Geoffrey Manley, Stephen Mayer, David X. Menon, Geert Meyfroidt, Daniel B. Michael, Mauro Oddo, David Okonkwo, Mayur Patel, Claudia Robertson, Jeffrey V. Rosenfeld, Andres M. Rubiano, Juan Sahuquillo, Franco Servadei, Lori Shutter, Deborah Stein, Nino Stocchetti, Fabio Silvio Taccone, Shelly Timmons, Eve Tsai, Jamie S. Ullman, Paul Vespa, Walter Videtta, David W. Wright, Christopher Zammit, and Randall Chesnut.

Poster Creation: Randall Chesnut, Gregory Hawryluk, Karan March, Linda Littlejohns, Global Neuro

Supported by

Brain Trauma Foundation

Global Neuro


ICP < 22 mmHg
ICP > 22 mmHg
$P_{bt}O_2$ > 20 mmHg
$P_{bt}O_2$ < 20 mmHg
BASIC CARE
Applies to all sTBI Patients
Type A
Type B
Type C
Type D

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 ≥ 60mmHg
- 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 SpO_2 ≥ 94%

Recommended Interventions:

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

TYPE B ICP Elevated – Brain Oxygenation Normal

TIER 1

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

TIER 2

- Mild hypocapnia (range 32–35mmHg/ 4.3–4.6 kPa)
- Neuromuscular paralysis in adequately sedated patients if efficacious in lowering ICP[2]
- Perform MAP challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients[3].
  - Should be performed under direct supervision of a physician who can assess response and ensure safety
  - No other therapeutic adjustments (i.e. sedation) should be performed during the MAP Challenge
  - Initiate or titrate a vasopressor or inotrope to increase MAP by 10mmHg 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

TIER 3

- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious[4]
- 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

- Reexamine the patient and consider repeat CT to reevaluate 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

[1] We recommend using sodium and osmolality limits of 155 mEq/L and of 320mEq/L respectively as administration limits for both mannitol and hypertonic saline.

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

[3] Rosenthal G, Sanchez-Mejia RO, Phan N, Hemphill JC 3rd, Martin C, Manley GT. Incorporating a parenchymal thermal diffusion cerebral blood flow probe in bedside assessment of cerebral autoregulation and vasoreactivity in patients with severe traumatic brain injury. J Neurosurg. 2011;114(1):62-70. doi:10.3171/2010.6. JNS091360

[4] 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.

TYPE C ICP Normal – Brain Hypoxic

TIER 1

- Maximum CPP 60 – 70 mmHg
- Increase CPP to a maximum of 70 mmHg with fluid, vasopressors and/or inotropes
- Maintain P_aCO_2 > (35 mmHg/4.7 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/20 kPa
- 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 $P_{bt}O_2$[1]
- Perform MAP challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients[2]
  - Should be performed under direct supervision of a physician who can assess response and ensure safety
  - No other therapeutic adjustment (i.e. 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 70mmHg with fluid boluses, vasopressors and/or inotropes[3]

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 mmHg despite P_aO_2 and CPP/MAP optimization, consider transfusing 1 unit of PRBCs if Hbg <9g/L

- Reexamine the patient and consider repeat CT to reevaluate 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

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

[2] Rosenthal G, Sanchez-Mejia RO, Phan N, Hemphill JC 3rd, Martin C, Manley GT. Incorporating a parenchymal thermal diffusion cerebral blood flow probe in bedside assessment of cerebral autoregulation and vasoreactivity in patients with severe traumatic brain injury. J Neurosurg. 2011;114(1):62-70. doi:10.3171/2010.6.JNS091360

[3] Careful monitoring for respiratory complications is required when CPP is raised above 70 mmHg (Robertson C.S. et al. Prevention of secondary ischemic insults after severe head injury. Crit Care Med. 1999;27(10):2086–2095)

TYPE D ICP Elevated – Brain Hypoxic

TIER 1

- Maintain CPP 60 – 70 mmHg
- 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[1]
- 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 ranged, 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/20 kPa
- Increase sedation to improve ICP and $P_{bt}O_2$
- Neuromuscular paralysis in adequately sedated patients if efficacious in decreasing ICP or increasing $P_{bt}O_2$[2]
- Perform MAP challenge to assess cerebral autoregulation and guide MAP and CPP goals in individual patients[3]
  - Should be performed under direct supervision of a physician who can assess response and ensure safety
  - No other therapeutic adjustment (i.e. 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 70mmHg with fluid boluses, vasopressors and/or inotropes[4]

TIER 3

- Pentobarbital or Thiopentone coma titrated to ICP control if efficacious[5]
- 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 PRBCs if Hgb <9g/L

- Reexamine the patient and consider repeat CT to reevaluate 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

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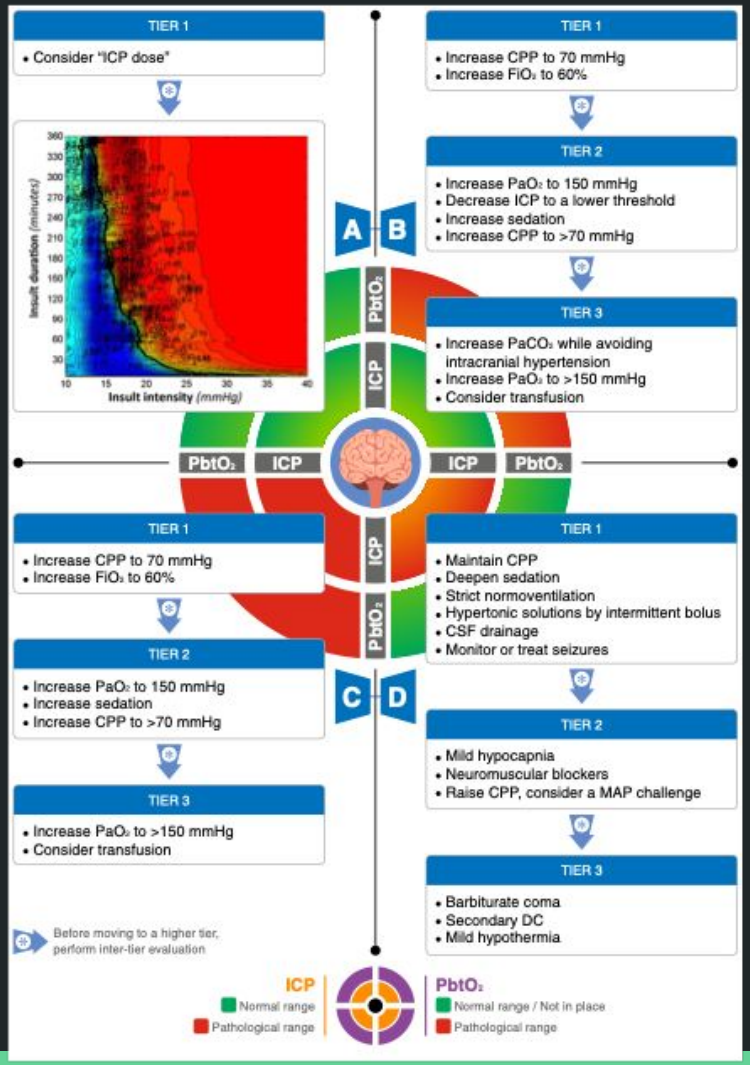
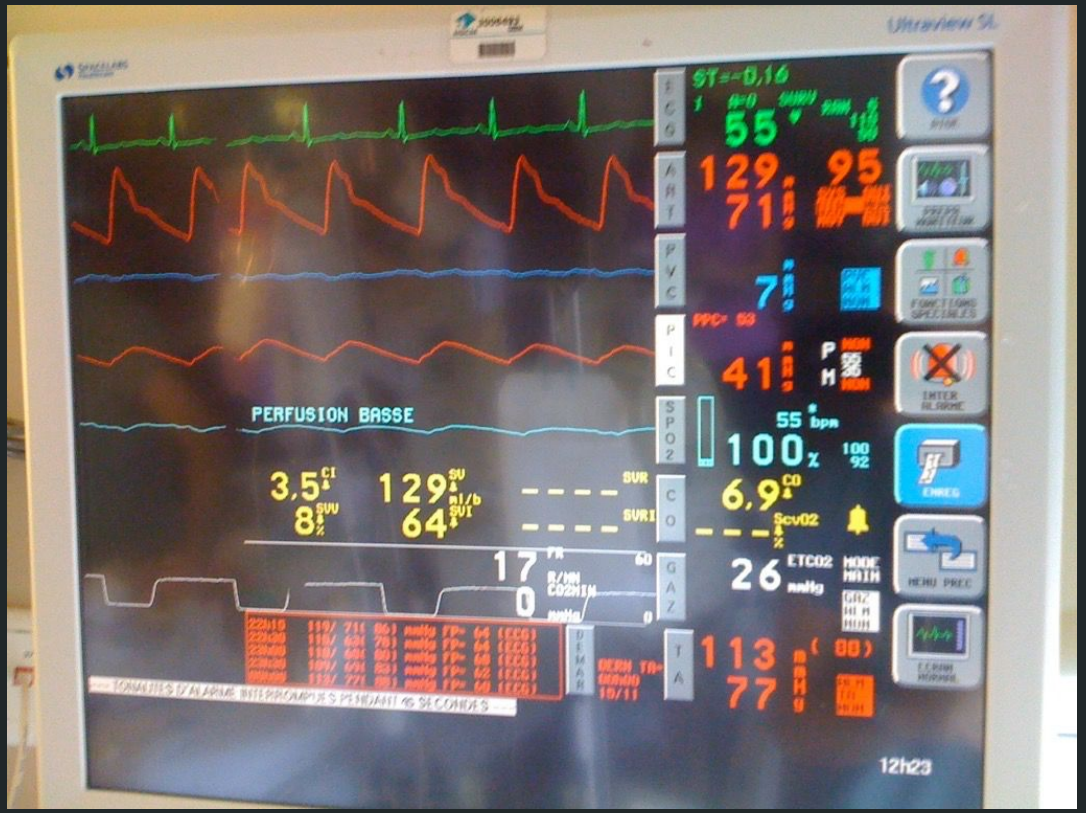
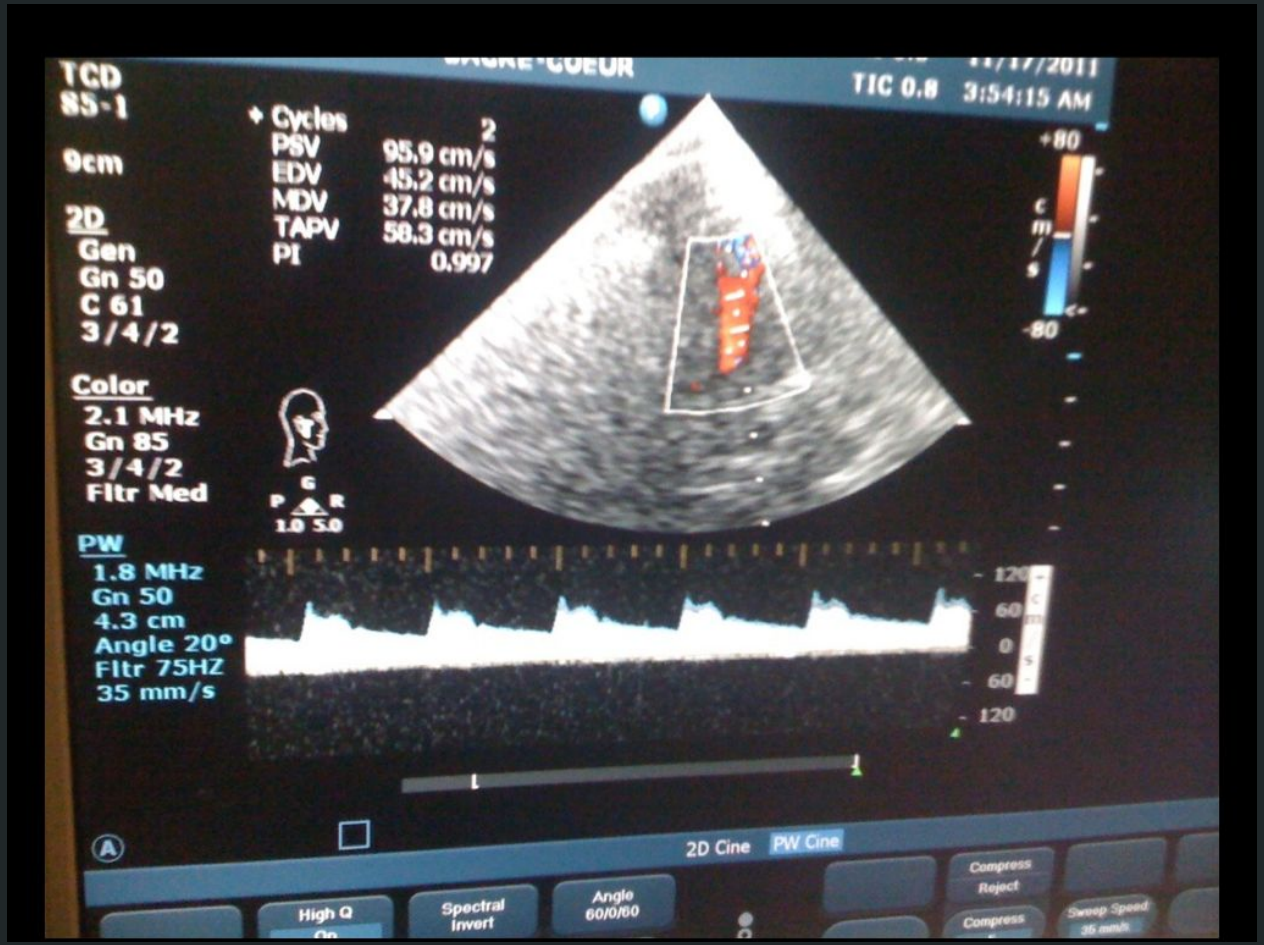
[5] Barbiturate administration should only be continued when a beneficial effect on ICP is demonstrated.
–Titrate barbiturate to achieve ICP control but do not exceed the dose which achieves burst suppression.
–Hypotension must be avoided when barbiturates are administered.


TIER 1
• Consider "ICP dose"
Insult duration (minutes)
Insult intensity (mmHg)
TIER 1
• Increase CPP to 70 mmHg
• Increase FiO₂ to 60%
TIER 2
• Increase PaO₂ to 150 mmHg
• Decrease ICP to a lower threshold
• Increase sedation
• Increase CPP to >70 mmHg
TIER 3
• Increase PaCO₂ while avoiding intracranial hypertension
• Increase PaO₂ to >150 mmHg
• Consider transfusion
A
B
PbtO₂
ICP
ICP
PbtO₂
TIER 1
• Increase CPP to 70 mmHg
• Increase FiO₂ to 60%
TIER 2
• Increase PaO₂ to 150 mmHg
• Increase sedation
• Increase CPP to >70 mmHg
TIER 3
• Increase PaO₂ to >150 mmHg
• Consider transfusion
C
D
TIER 1
• Maintain CPP
• Deepen sedation
• Strict normoventilation
• Hypertonic solutions by intermittent bolus
• CSF drainage
• Monitor or treat seizures
TIER 2
• Mild hypocapnia
• Neuromuscular blockers
• Raise CPP, consider a MAP challenge
TIER 3
• Barbiturate coma
• Secondary DC
• Mild hypothermia
Before moving to a higher tier, perform inter-tier evaluation
ICP
Normal range
Pathological range
PbtO₂
Normal range / Not in place
Pathological range

Jessica

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Gn 50
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+ Cycles 2
PSV 95.9 cm/s
EDV 45.2 cm/s
MDV 37.8 cm/s
TAPV 58.3 cm/s
PI 0.997
TIC 0.8
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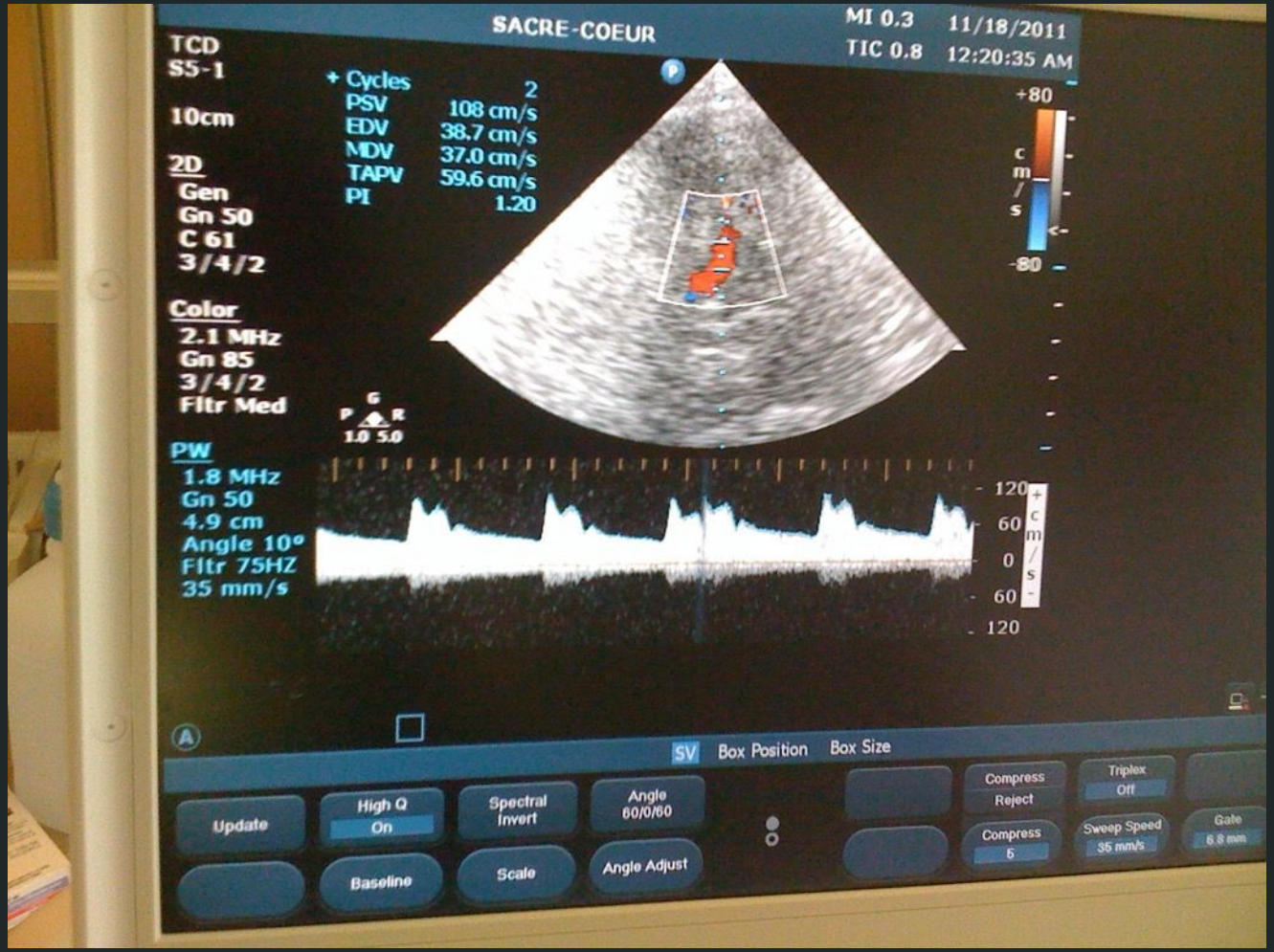
2D Cine
PW Cine
High Q
Spectral Invert
Angle 60/0/60
Compress Reject
Compress
Sweep Speed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35 mm/s
+ Cycles 2
PSV 108 cm/s
EDV 38.7 cm/s
MDV 37.0 cm/s
TAPV 59.6 cm/s
PI 1.20
+80
cm/s
-80
120
60
0
60
120
SV
Box Position
Box Size
Update
High Q On
Spectral Invert
Angle 60/0/60
Baseline
Scale
Angle Adjust
Compress Reject
Compress 5
Triplex Off
Sweep Speed 35 mm/s
Gate 6.8 mm

Jessica

A toujours eu une valeur d’oxygénation cérébrale supérieure à 15 mmHg

N’a jamais eu de craniectomie

Extubation au jour 19 du trauma

À 3 semaines post trauma, exhibait tableau frontal, sans évidence de lésions secondaires

Au suivi en externe - autonome, difficultés rencontrées au retour à l’université

The following passage appeared in the introductory chapter of a text on TBI from 1897: “The manner of treatment is of importance in only a minority of cases, since many subjects of intracranial injury are fated to die whatever measures may be adopted for their relief, and a still greater number are destined to recover though left entirely to the resources of nature. It is probable that in by far the larger proportion of cases in which the issue is determined by treatment it is met in the initial stage, and by insuring restoration from primary shock”