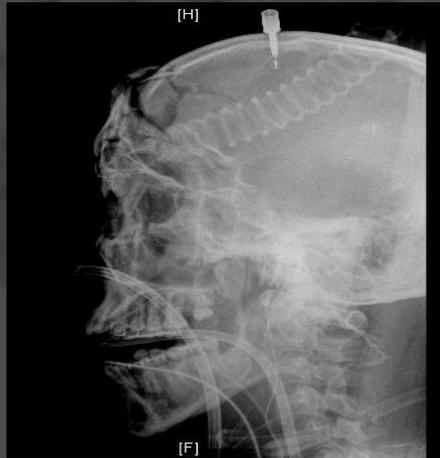


Traumatisme crânien sévère

RENAU RESUV al RESQ Le REULIAN CRAU

Printemps 2012 de la médecine d'urgence

Lyon, mardi 15 mai 2012



Dr Laurent Gergelé
Réanimation Polyvalente B
Pr AUBOYER
CHU St Etienne



Il n'y a pas de TC peu sévère

→ Ne jamais sous estimer la gravité d'un TC avec PCI ...



J Neurosurg 75:256-261, 1991

Head-injured patients who talk and deteriorate into coma

Analysis of 211 cases studied with computerized tomography

RAMIRO D. LOBATO, M.D., JUAN J. RIVAS, M.D., PEDRO A. GOMEZ, M.D.,
MARIO CASTAÑEDA, M.D., JOSÉ M. CAÑIZAL, M.D., ROSARIO SARABIA, M.D.,
ANTONIO CABRERA, M.D., AND MARIA J. MUÑOZ, M.D.

Neurosurgery Service, Hospital "12 Octubre" and Faculty of Medicine, Complutense University,
Madrid, Spain

Patients with a Head Injury Who "Talk and Die" in the 1990s

Laurence T. Dunn, PhD, FRCS(NS), Michael O. Fitzpatrick, MD, FRCS(NS), Diana Beard, RGN, MBA, and
Jennifer M. Henry, BSc

J Trauma. 2003;54:497-502.

Journal
© 2003
doi: 10.1054/jtra.2001.0815, available online at <http://www.idealibrary.com> on IDEAL[®]

Review

Brain injury: the pathophysiology of the first hours. 'Talk and Die revisited'

Peter L. Reilly MD FRCS

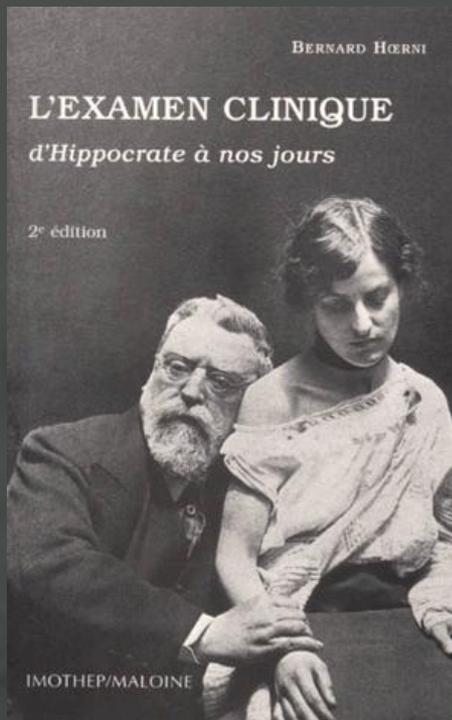
Legal Medicine (Legal Med) 2000 ; 2 : 175-80

Discrepancy of clinical symptoms and prognosis of a patient — forensic
significance of "talk and die" head injury

Kazuhiko KIBAYASHI¹, Paul M. NG'WALALI², Kisei HAMADA², Kohji HONJO², Shigeyuki TSUNENARI²

¹Department of Forensic Medicine, Saga Medical School, Saga 849-8501, Japan

²Department of Forensic Medicine, Kumamoto University School of Medicine, Kumamoto 860-0811, Japan



L'examen clinique

→ Le meilleur stéthoscope du cerveau

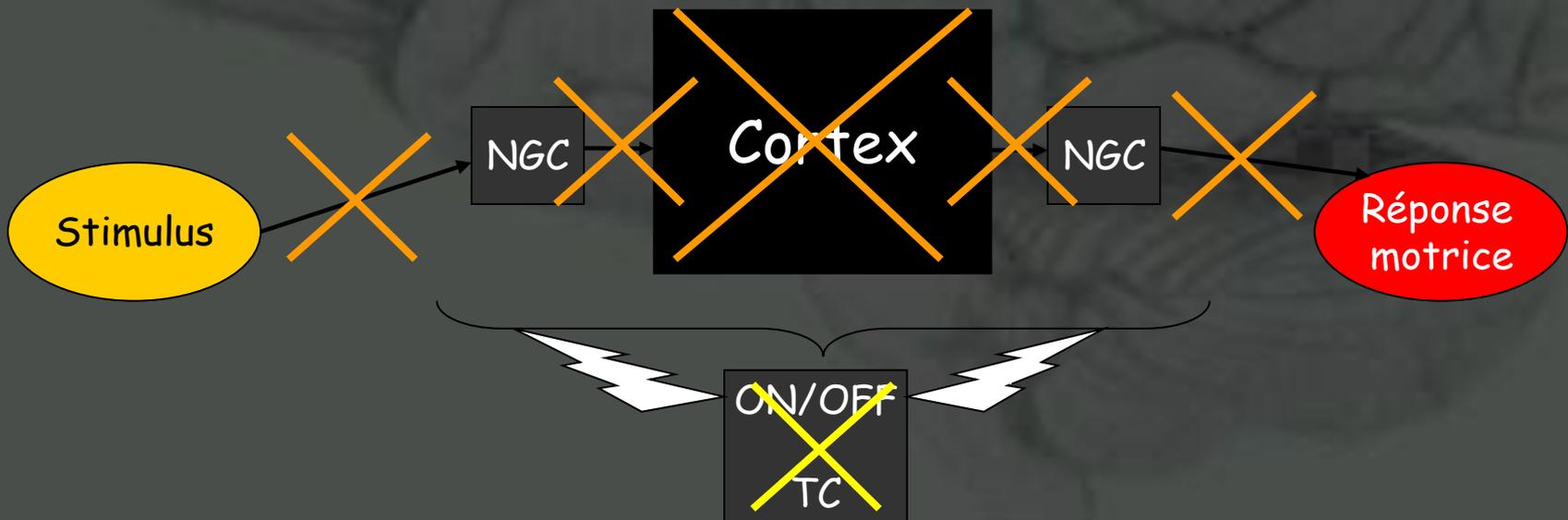
Une fois sous AG ...



Evaluation de la sévérité Glasgow

Conscience et vigilance = fonctions cérébrales supérieures permettant un éveil et une réactivité comportementale

Stimulus → intégration cortico-souscorticale → Réponse M



Glasgow

Le Glasgow → Rigueur +++ dans le recueil

- Nociception (Stylo sur ongle)
- Après normalisation Hd et O₂
- Détailler le Score (M) +++++
- Attention aux toxiques (OH)



$$Y_{n_1} V_{n_2} M_{n_3} \rightarrow G = n_1 + n_2 + n_3$$

Glasgow

Score de Glasgow:

- Classification du TC

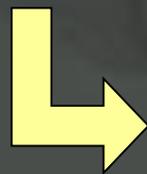
 - $G=14-15$ → TC mineur

 - $G > \text{ou} = \text{à } 8$ et < 14 → TC intermédiaire

 - $G < 8$ = TC grave

- Conditionne la **PEC initiale et l'orientation du patient**

→ Ex: $G < 8$ → IOT (Score moteur +++)



$G=8$ → Y4/M2/V2

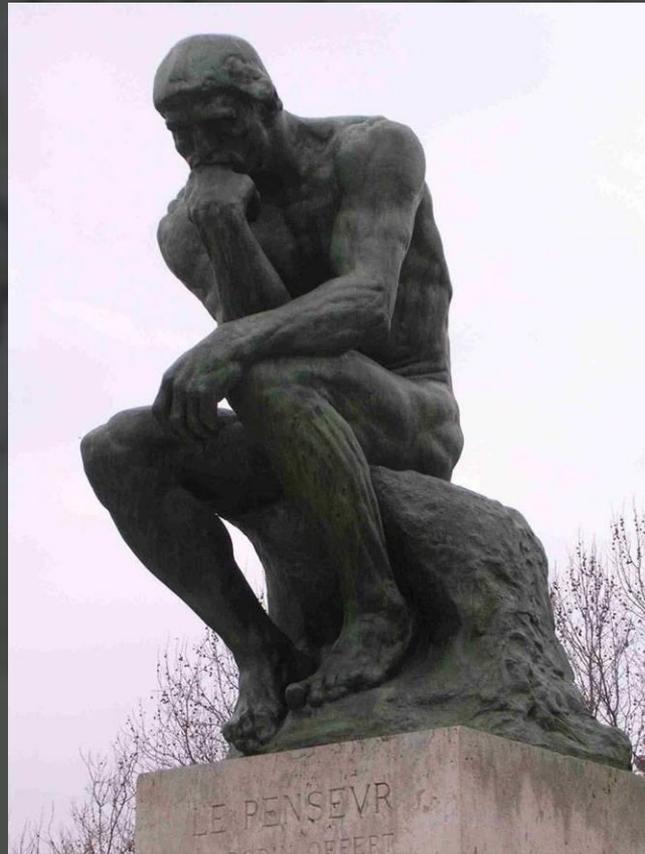
≠

$G=8$ → Y2/M4/V2



Bonne orientation du malade améliore la survie *Dubose et al Ann Surg 2008*

Philosophie de la neuroréanimation



ACSOS / A« N »SOS

Lésions initiales

Lésions directes



Lésions définitives

Séquelles

Lésions secondaires

ACSOS



Dès la PEC initiale sur le terrain ...

- Evaluation clinique initiale
- Lésions associées
- Le bilan lésionnel



→ Conditionne les moyens à mettre en place

Hôpital de proximité ou déchocage de référence ?

• Et **Combattre les ACSOS**

→ Lutte contre Hypoxie, hypo/hypercapnie *Keir et al Trauma 2008*

→ IOT rapide

et Hypotension *Jones PA, J Neurosurg Anesth 1994*

OSMOTHERAPIE FORTES DOSES SI SIGNES D'ENGAGEMENT



La sédation



- Objectifs initiaux:

Adapter le patient à la VM **et** annuler les stimuli nociceptifs

- Quelle sédation ?

- Sédation initiale: Rapidement réversible (Propofol et Remifentanyl)
- Sédation prolongée: BZD (Midazolam) + Morphinique (Sufentanil)

Le nesdonal ...

BARBITURIQUES... Journal Neurotraumatology 2007

The Cochrane group thus concluded: “There is no evidence that barbiturate therapy in patients with acute severe head injury improves outcome. Barbiturate therapy

Utilization of barbiturates for the prophylactic treatment of ICP is not indicated

Brain Trauma Foundation
Improving the Outcome of Brain Trauma Patients Worldwide



La vasoconstriction métabolique → **Traitement de l'HTIC**

(Nesdonal et autres vasoconstricteurs métaboliques) sous couvert monitoring DSC

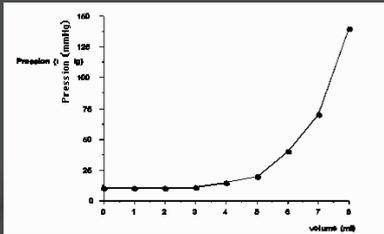
Quel objectif à l'hôpital ?

Les mêmes qu'en pré-hospitalier

Eviter l'ischémie cérébrale secondaire

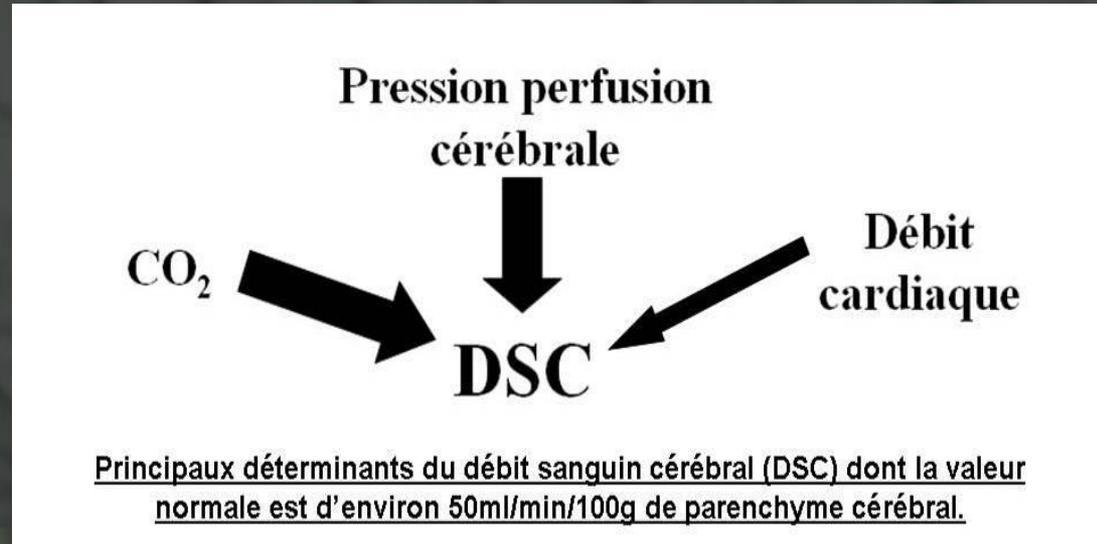
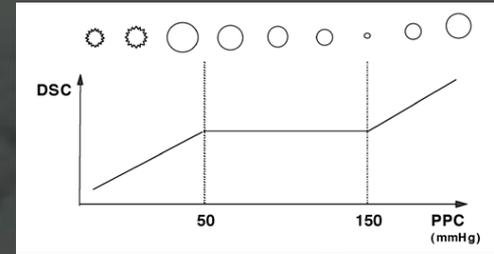
→ Maintenir le DSC

Ventilation



$$PPC = PAM - PIC$$

→ PIC



Hémodynamique et transport en oxygène



Quel monitoring en réanimation spécialisée ?

Contrôle des déterminants du DSC = Monitoring basique

Pression artérielle invasive

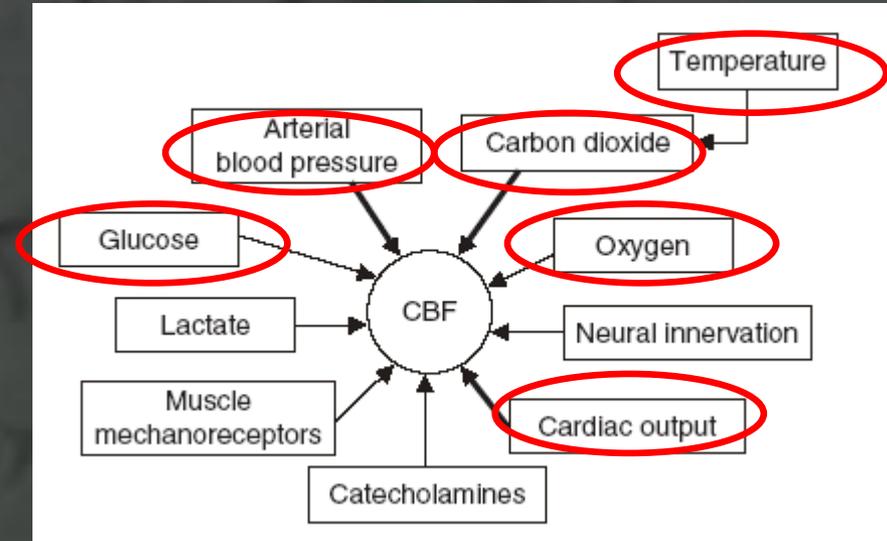
Pression intracrânienne (PIC)

→ PPC

Capnographie (EtCO₂)

Saturation en O₂, Dextro, Temp.

Monitoring du débit cardiaque



Evaluation du DSC:

Doppler Trans-crânien +/- en Continu (DTC)

Saturation veineuse jugulaire en Oxygène (SVjO₂)

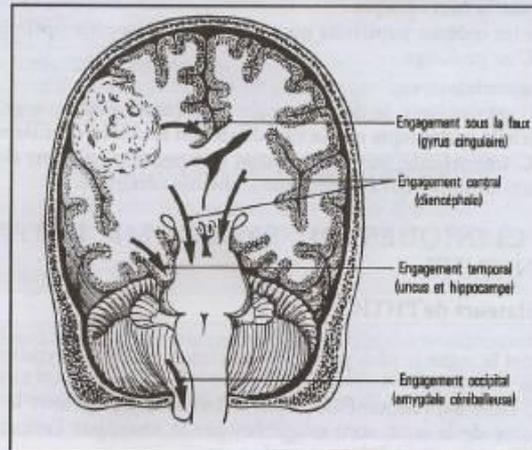
Pression tissulaire en Oxygène (PtiO₂)

Microdialyse



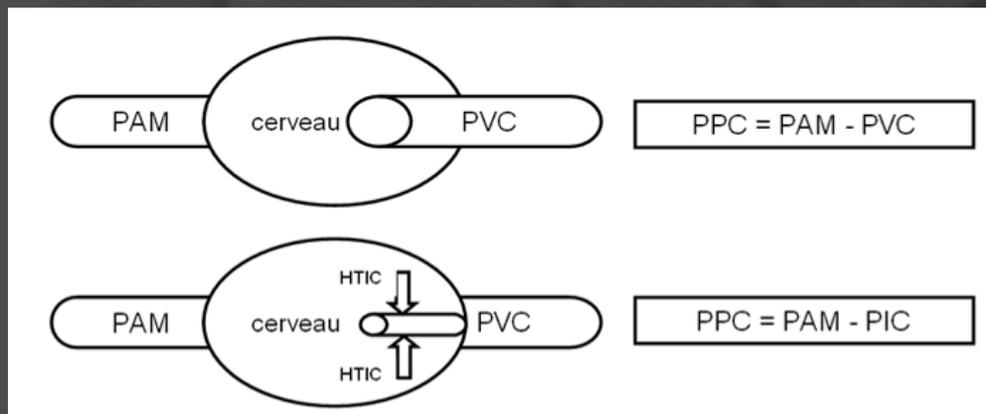
Pourquoi monitorer la PIC ...

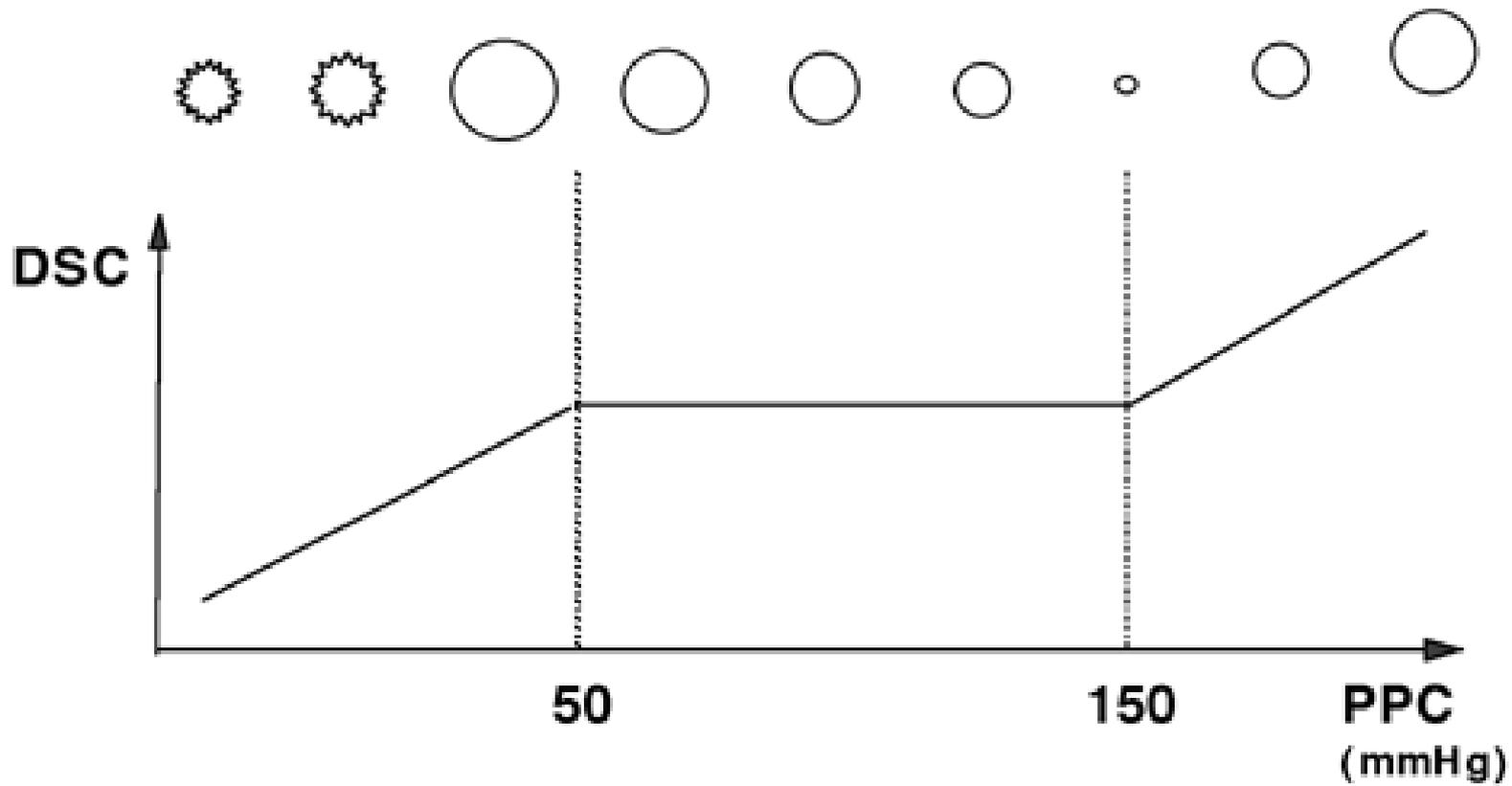
Figure 1 : Les différents types d'engagement cérébral
(Tiré de Cambier J., Masson M., Dehen H., « Neurologie », Éd. Masson, 1994)

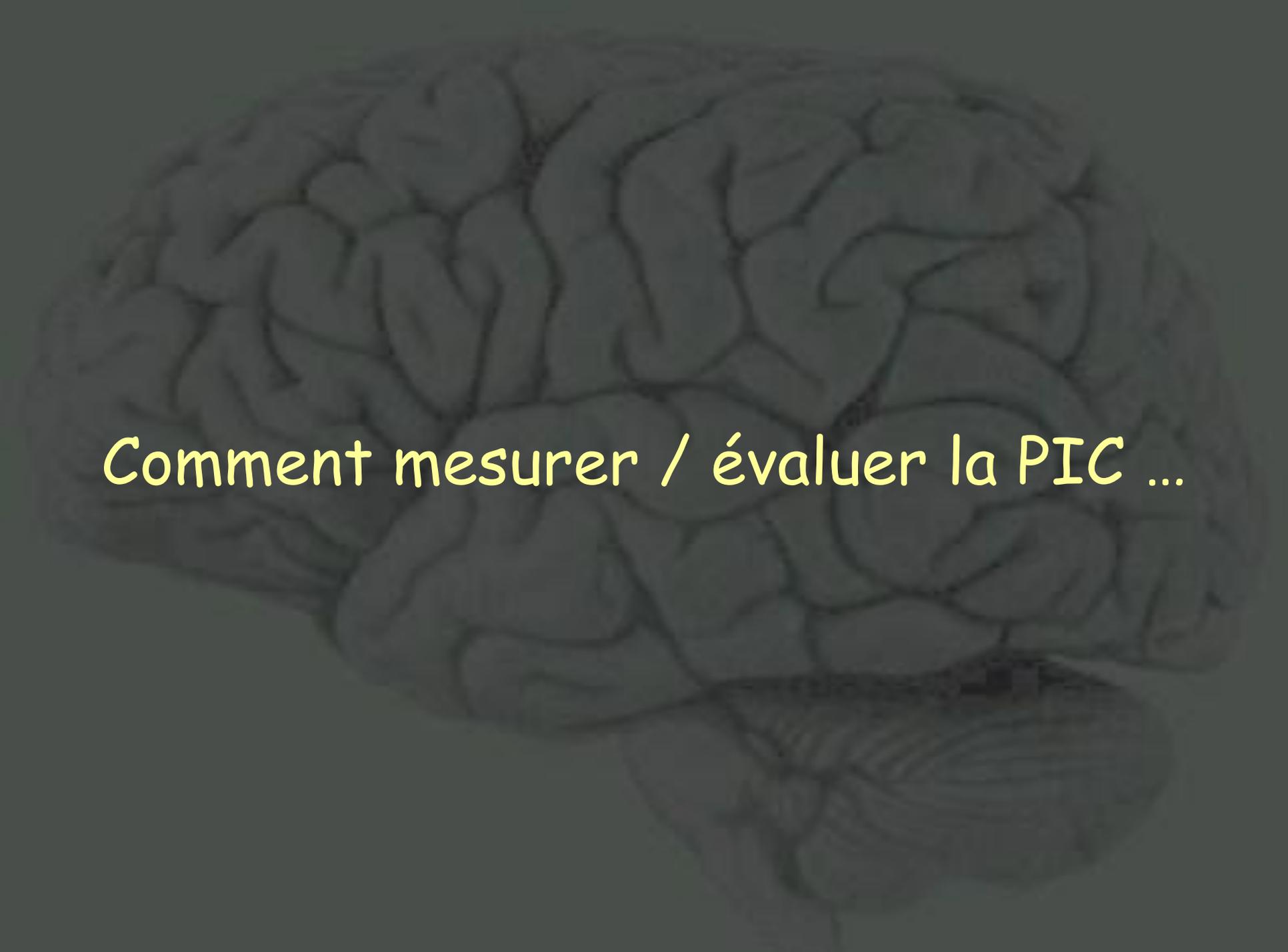


Pour connaître la pression de perfusion cérébrale en cas d'HTIC

- Nécessaire pour maintenir un débit tissulaire
- Particularité du cerveau → Boîte crânienne = inextensible
- Physiologiquement → $PPC = PAM - PVC$
- En cas d'HTIC → $PPC = PAM - PIC$
- $PPC =$ Principal déterminant du DSC global.

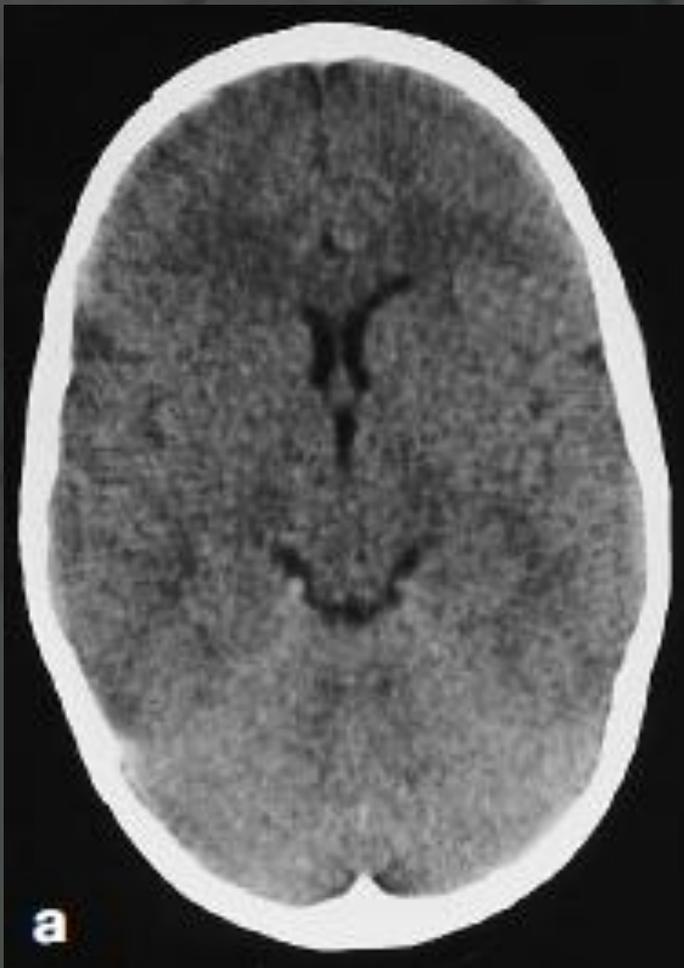






Comment mesurer / évaluer la PIC ...

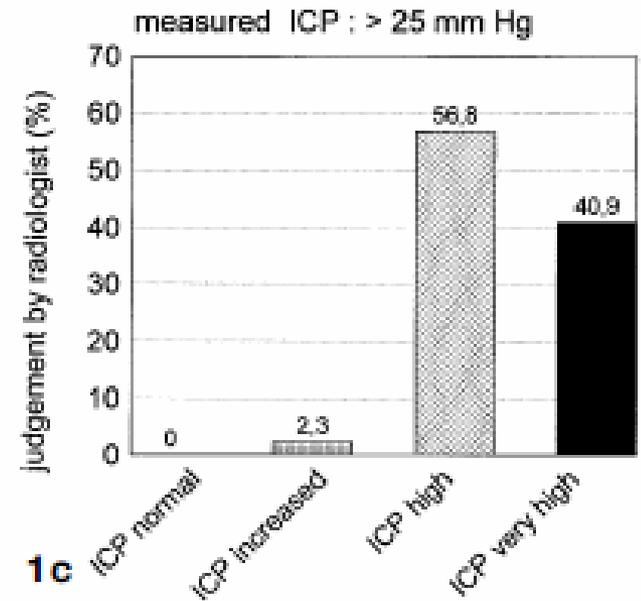
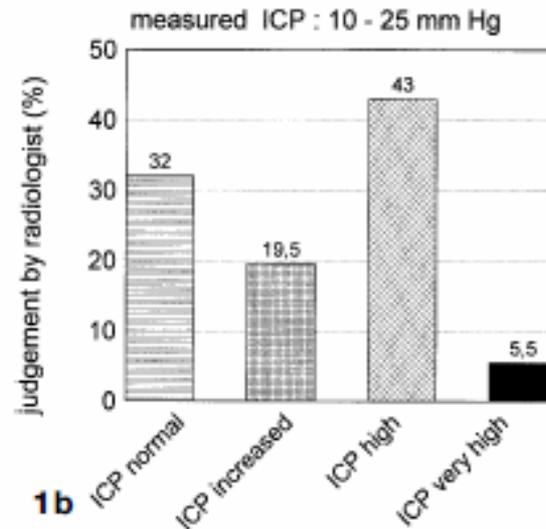
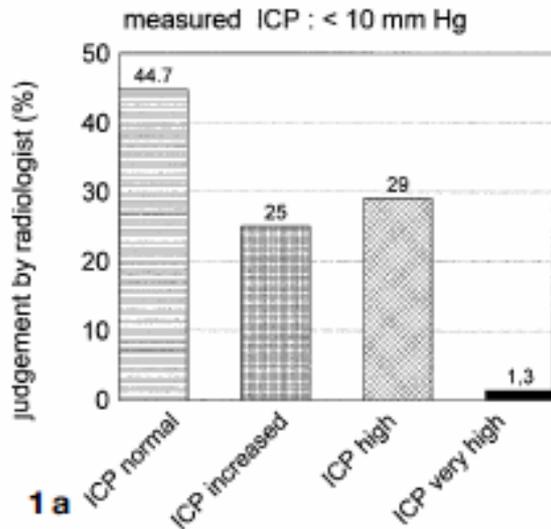
Le scanner ...



PIC = ?

Wolfgang Hirsch
Rainer Beck
Curt Behrmann
Axel Schobess
Rolf-P. Spielmann

Reliability of cranial CT versus intracerebral pressure measurement for the evaluation of generalised cerebral oedema in children



In conclusion, we have found that in children we cannot use CT to differentiate between reduction of the fluid reserve space without any rise of ICP and when there is a critical rise of ICP. This would explain why both of

Clinical Article

The relationship between intracranial pressure and size of cerebral ventricles assessed by computed tomography

P. K. Eide

Department of Neurosurgery, The National Hospital, University of Oslo, Oslo, Norway

Published online March 3, 2003

© Springer-Verlag 2003

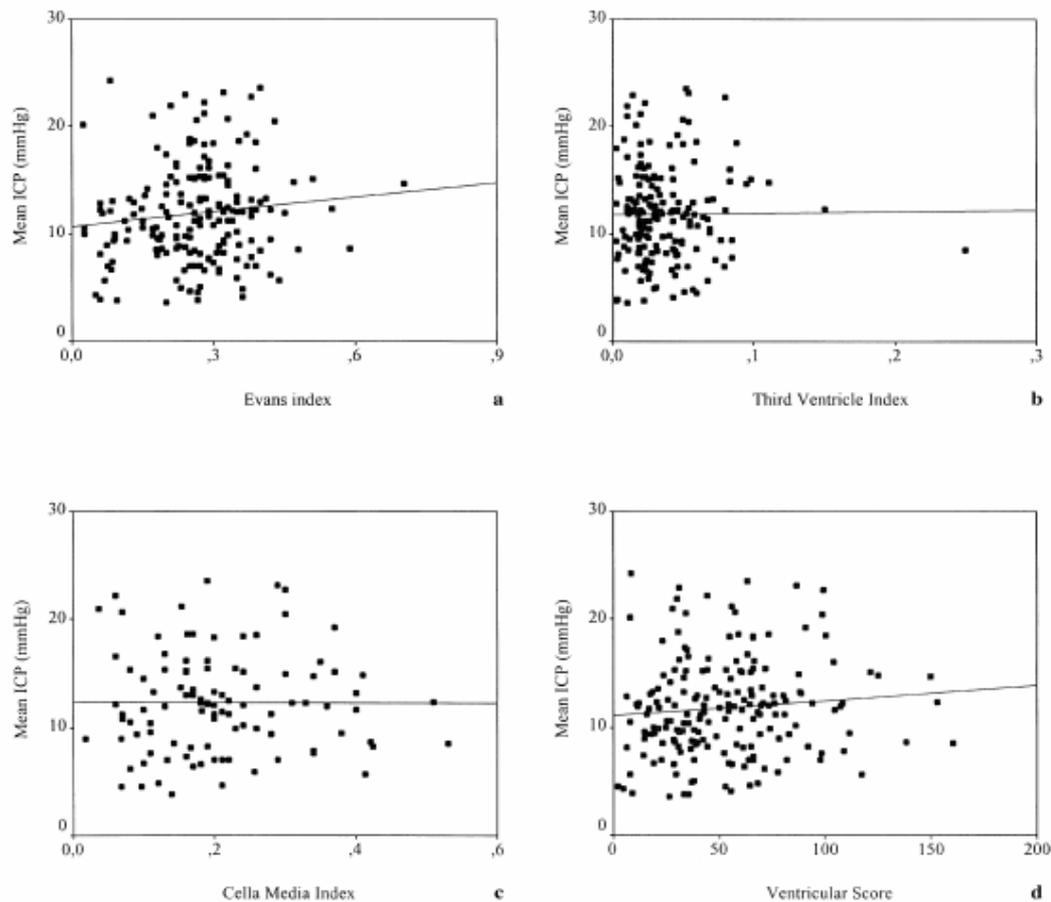
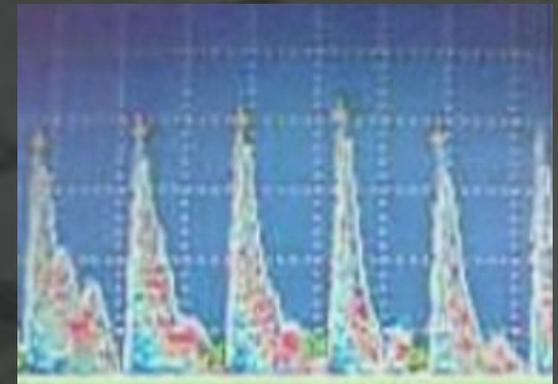
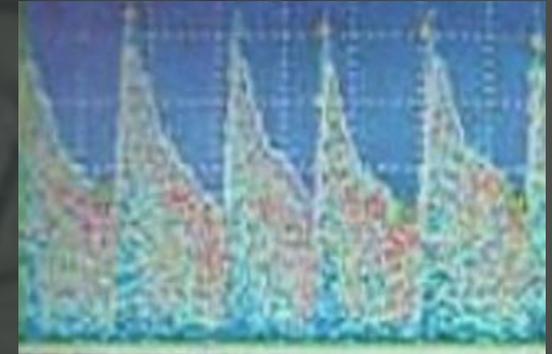


Fig. 1. The relationship between mean ICP (mmHg) and either (a) Evans index, (b) third ventricle index, (c) cella media index, or (d) ventricular score in the whole group of 184 cases. Data presented as individual recordings and lines of regression

According to these results, great care should be taken when predicting ICP on the basis of cranial CT scans with the estimation of cerebral ventricular size.

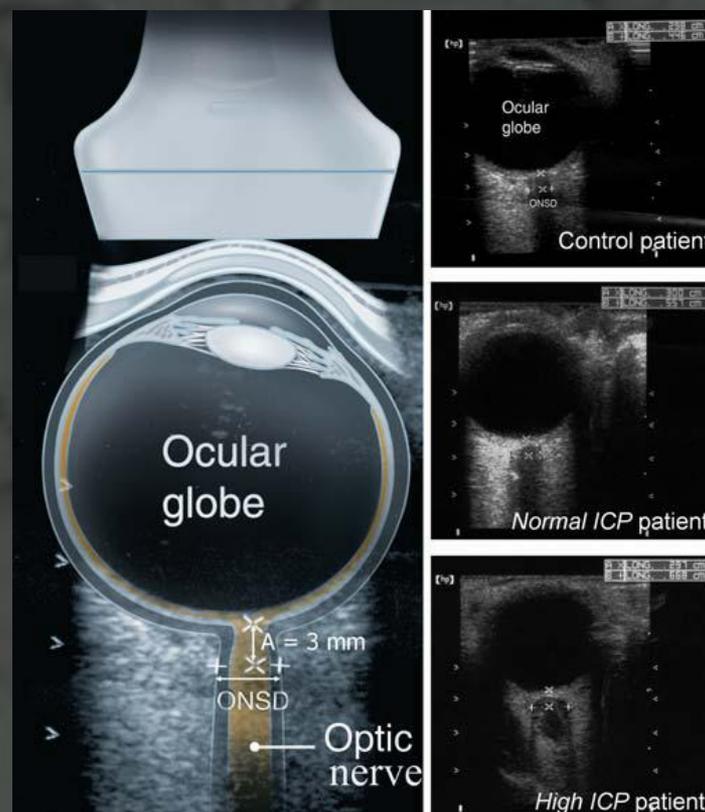
Le Doppler transcrânien



+++ à la phase aigue
Index de pulsatilité

Julie Dubourg
Etienne Javouhey
Thomas Geeraerts
Mahmoud Messerer
Behrouz Kassai

Ultrasonography of optic nerve sheath diameter for detection of raised intracranial pressure: a systematic review and meta-analysis



Le capteur de PIC +++

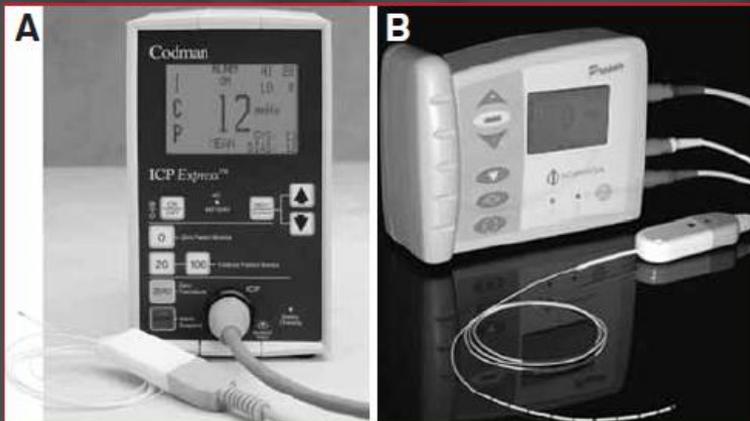
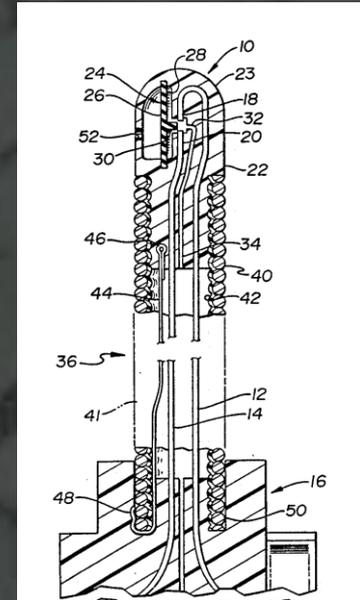
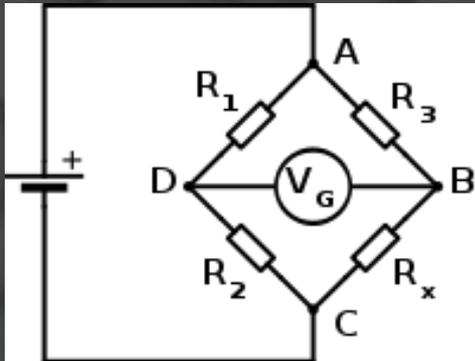


FIGURE 1. General views of the Codman Express intracranial pressure (ICP) monitor (A) and the Sophysa Pressio ICP monitor (B).



La PIC

Plusieurs techniques →

Intraparenchymateux +++

Indications bien codifiées:

→ TCPC grave $GSC \leq 8$ **et** TDM anormal / Moteur +++
(hématomes, contusions, œdèmes, effacement citernes de la base)

Journal of Neurotrauma 2007

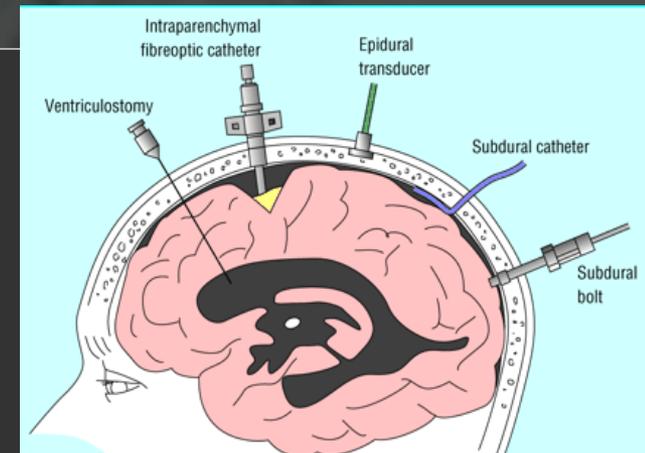
Intérêt:

Contrôler la PIC et éviter les épisodes de PIC > 20-25
améliore le pronostic des patients.

Saul et al, J Neurosurg 1982

Patel et al, ICM 2002

Lane et al Can J Surg 2000

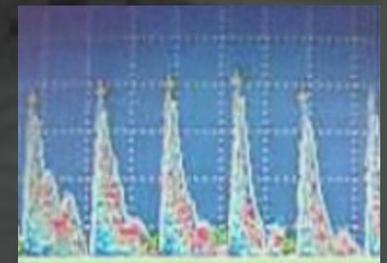
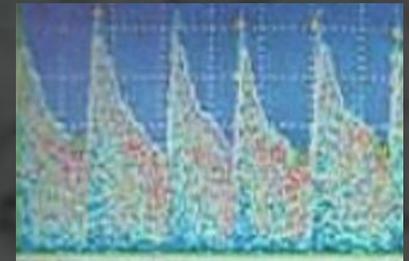
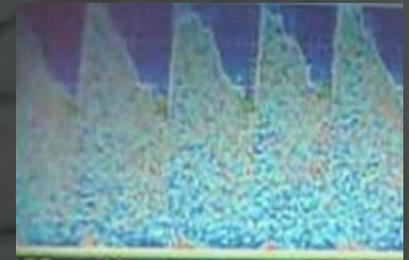
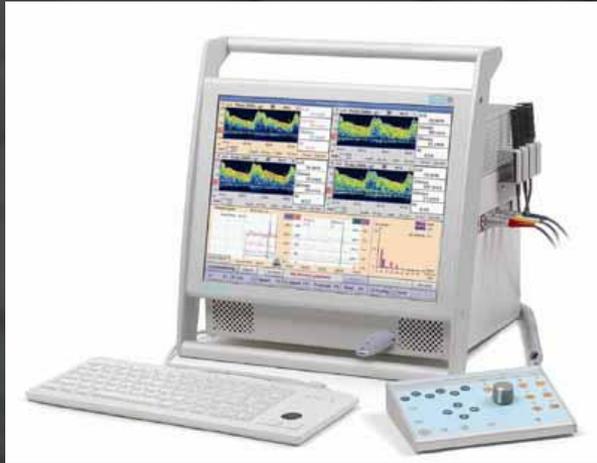




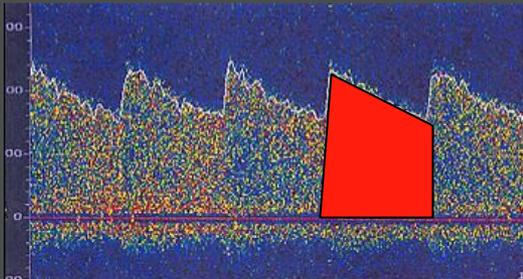
Le Débit Sanguin Cérébral

Monitoring multimodale

Le Doppler transcrânien



*En réanimation la VM = ITV du cerveau
Normale = 60 +/- 10 cm/s*



La Pression Tissulaire en Oxygène (PtiO₂)

Positionnement

→ *En zone saine pour le TC*

Reflet du DSC local (PaO₂, Hb, PaCO₂)

Nortje et al BJA 2006

Attention PaO₂ → Utilisation rapport PtiO₂/PaO₂ ?

Seuil Ischémique = 15 mmHg ...

Son utilisation semble améliorer le pronostic chez le TC

→ 25% avec PtiO₂ vs 44% DCD

Stiefel et al, J Neurosurg 2005

Leroux et al, CCM 2009

Pradeep et al, JNS 2009



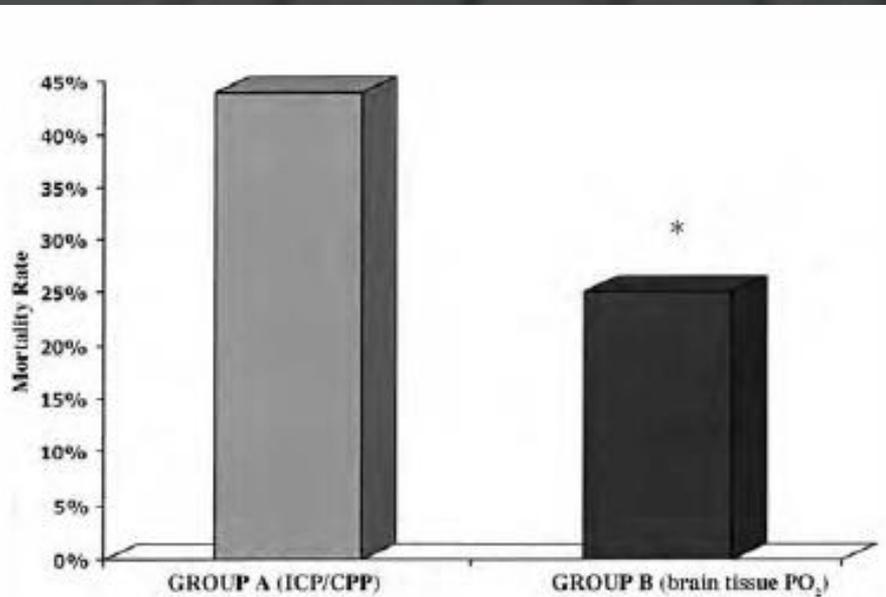


FIG. 1. Bar graph illustrating the mortality rates in patients who received traditional ICP/ CPP therapy (Group A, 25 patients) or combined ICP/ CPP and brain tissue PO₂ treatment (Group B, 28 patients). *p < 0.05.

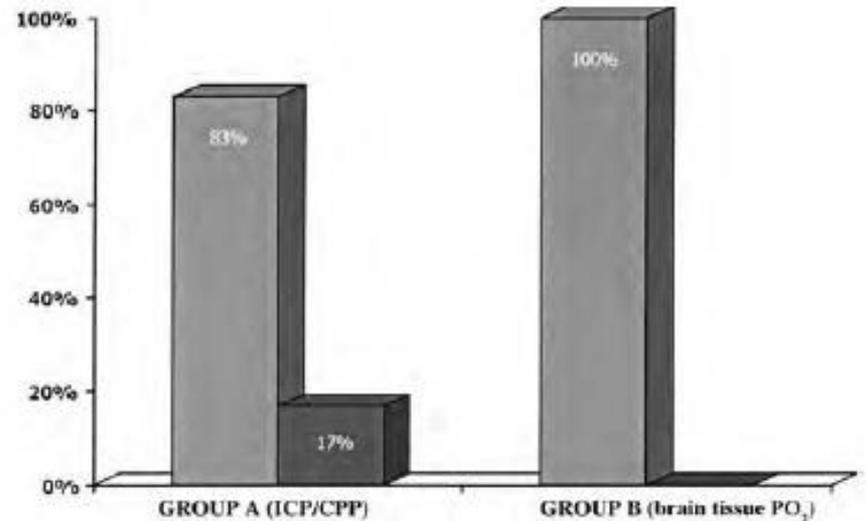
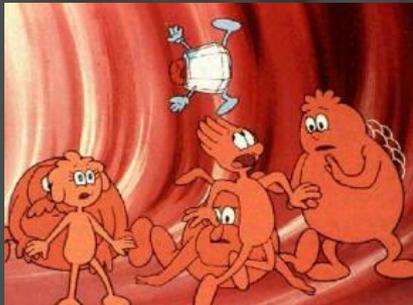


FIG. 2. Bar graph illustrating the discharge disposition among surviving patients who received traditional ICP/ CPP treatment (Group A, 14 patients) or combined ICP/ CPP and brain tissue PO₂ treatment (Group B, 21 patients). Discharge outcome was considered favorable if the patient was discharged to home or a rehabilitation center and unfavorable if the patient required additional hospitalization or was admitted to a nursing home. *Light gray bar* indicates favorable outcome; *dark gray bar* indicates an unfavorable outcome.

Prise en charge thérapeutique



Traitement médical

Objectif N°1 → Maintenir le DSC

adapté à la demande: Prévenir les lésions ischémiques secondaires

Objectif N°2 → Lutter contre l'HTIC

DIFFERENTS MOYENS THERAPEUTIQUES:

Lutte contre les ACSOS et optimiser le DSC

- La PPC
- Le CO₂

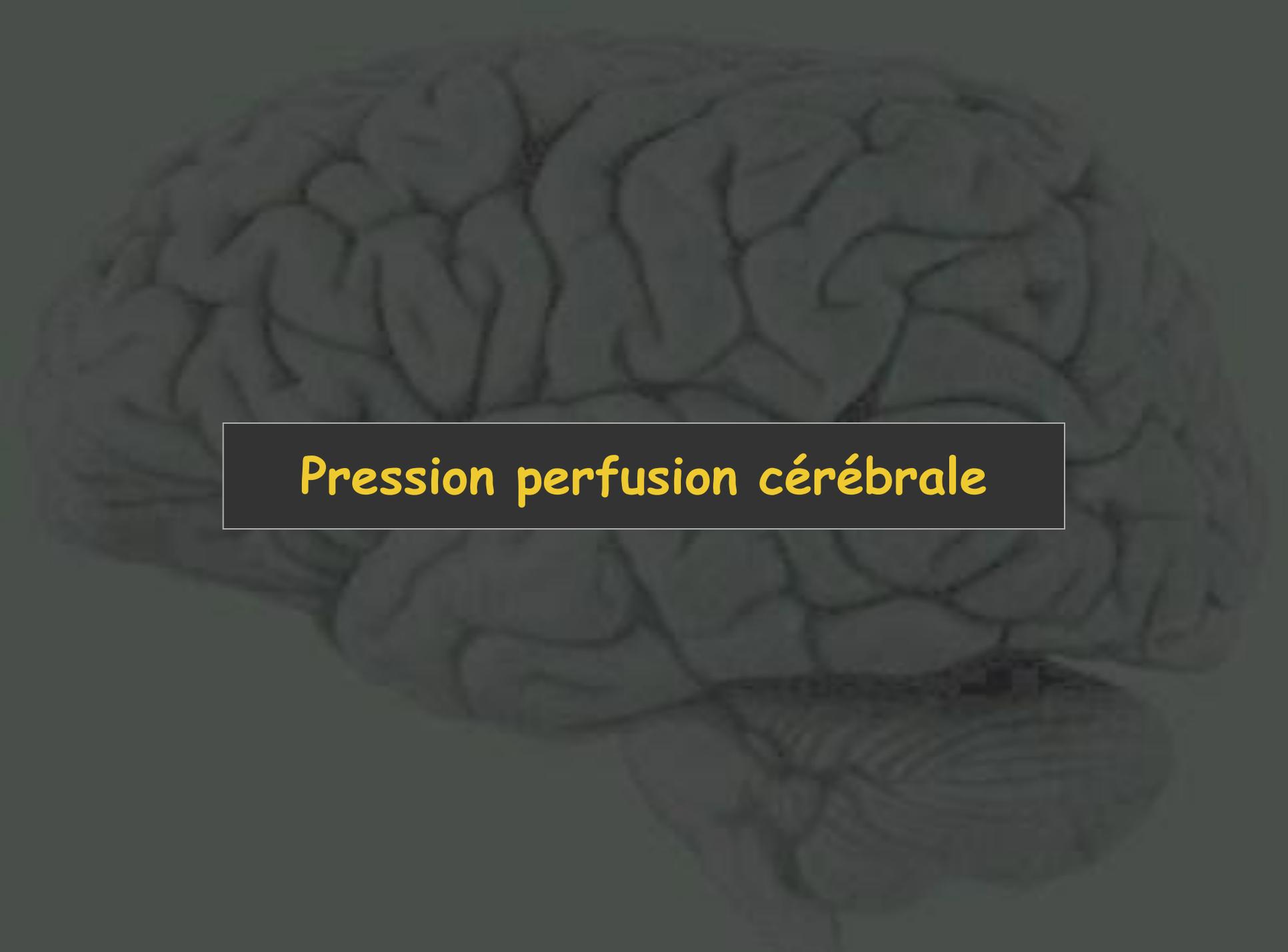
Vasoconstriction métabolique

Osmothérapie

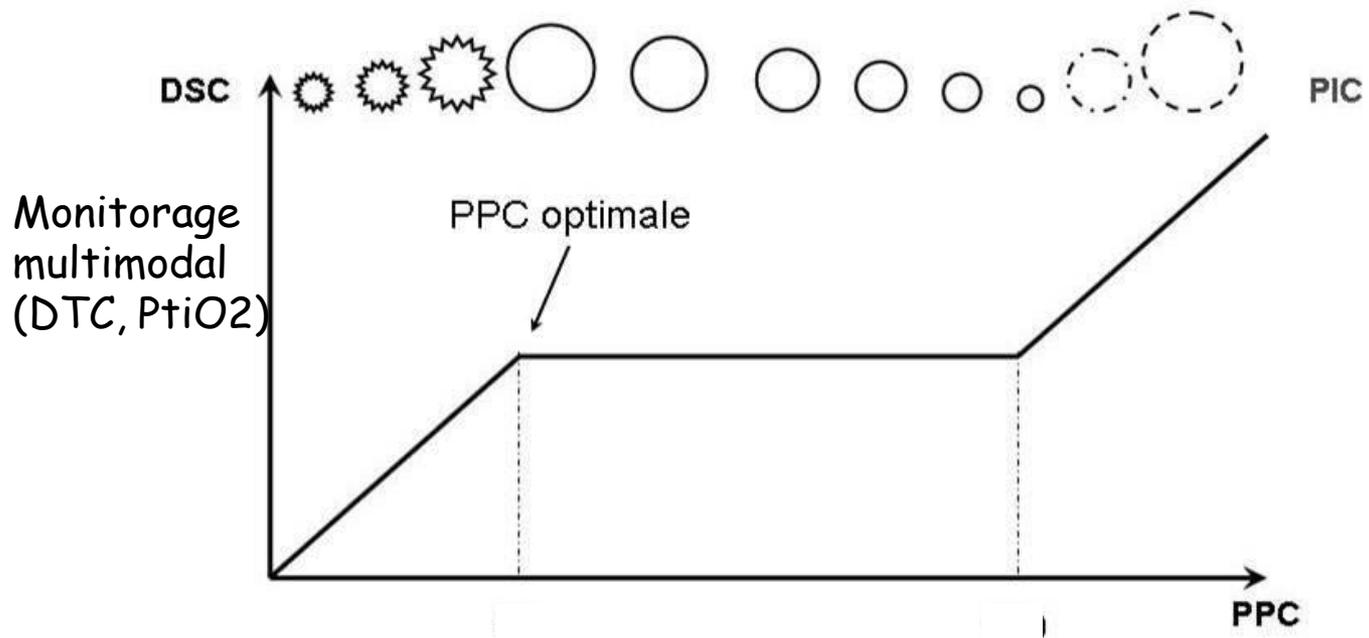
Lutte contre les A«N»SOS

« « Le tout normal » »

- PPC et CO2
- L'oxygénation: IOT précoce si hématoxémie anormale
- Régulation de la glycémie: Eviter l'hyperglycémie et surtout l'hypoglycémie
- Contrôle de la température: Eviter hyperthermie
- Equilibre électrolytique
- Traitement anticomitial
- Anémie et coagulopathie ...

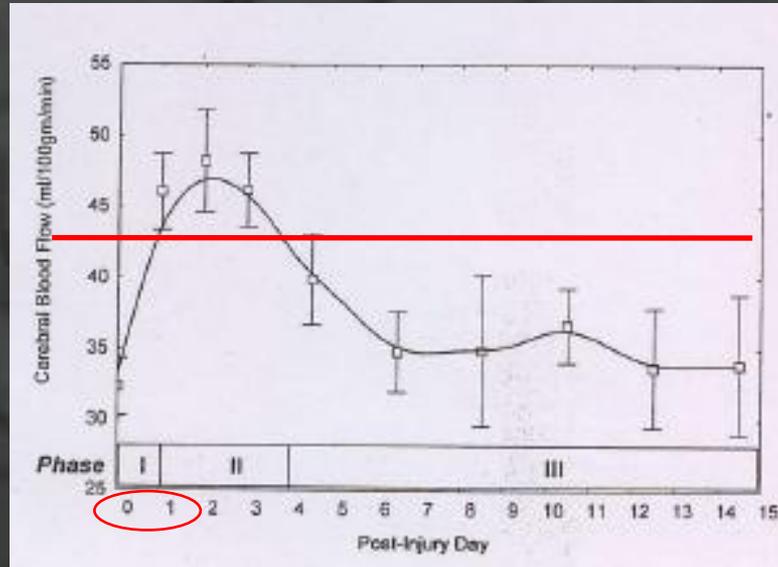


Pression perfusion cérébrale

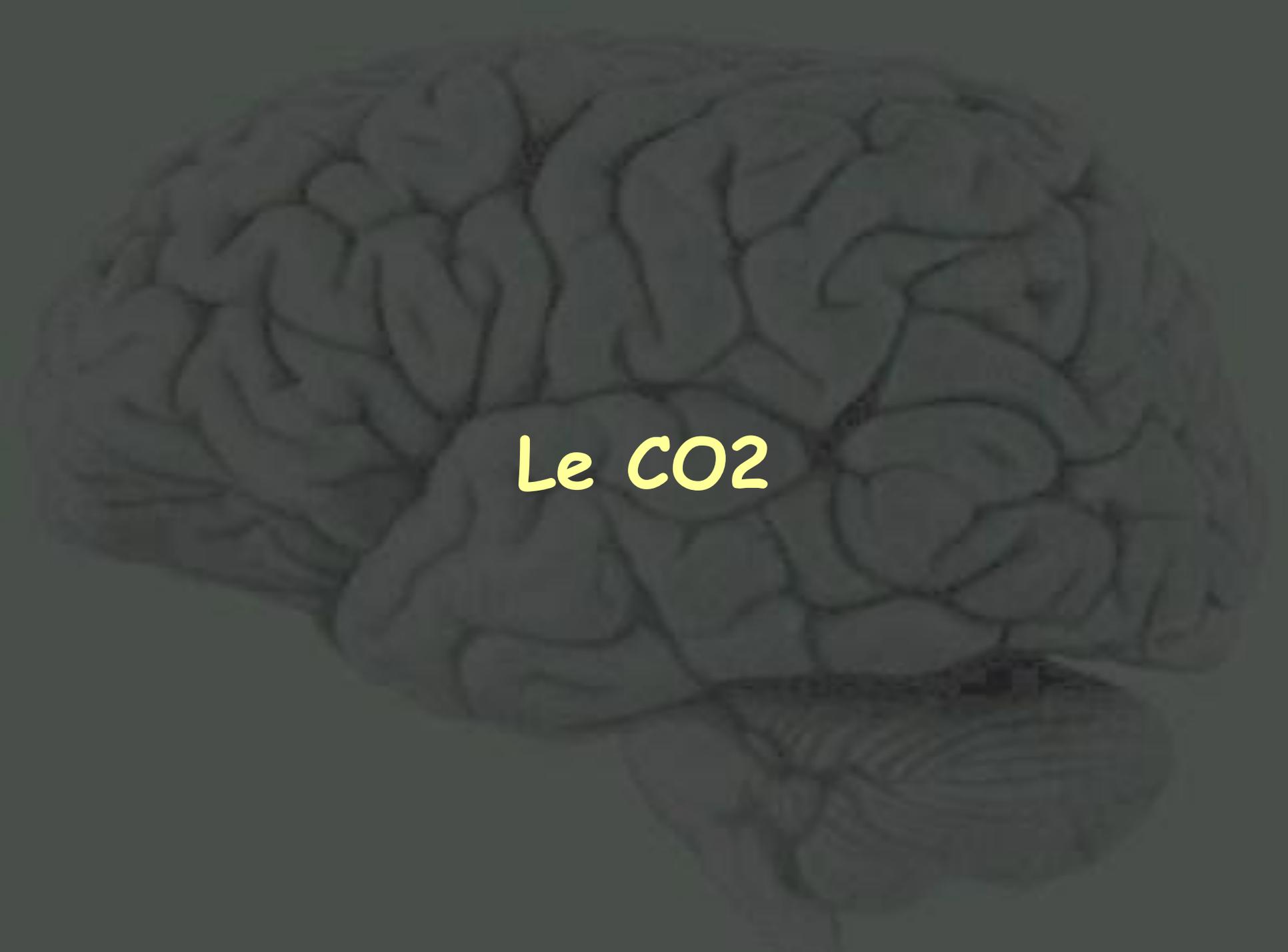


Courbe d'autorégulation cérébrale.

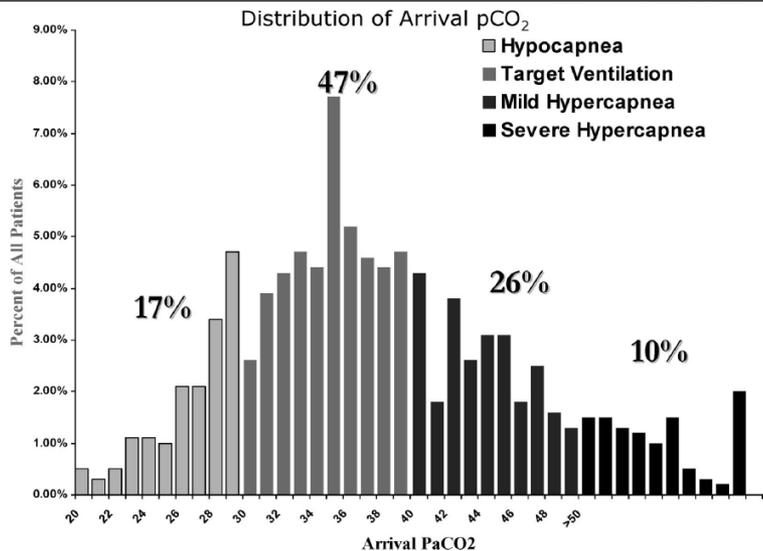
Quelle PPC en l'absence du monitoring multimodal ?



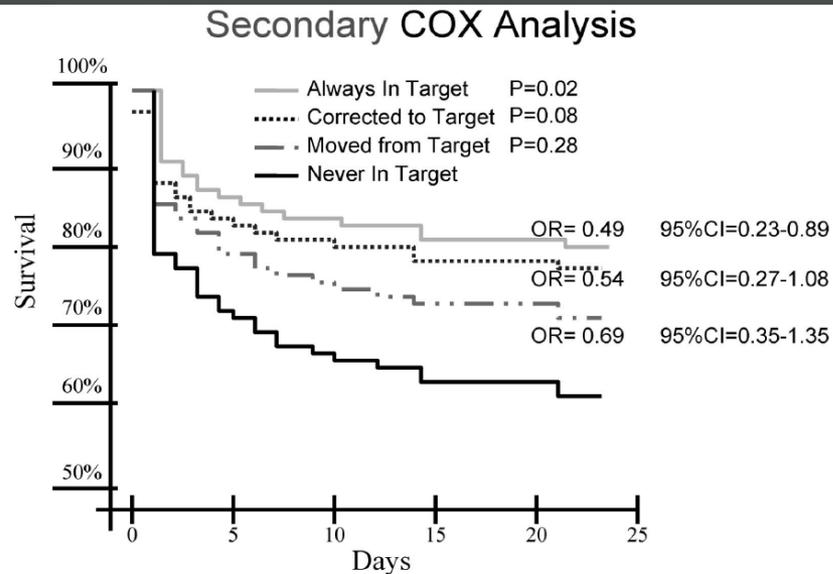
- PPC = 70-80 mmHg dans les premières 24h → PAM autour de 90mmHg à la phase initiale si suspicion HTIC
- Savoir rapidement diminuer la PPC sur les données du monitoring multimodal dans les 12 à 24h



Le CO₂



Shows the distribution of arrival arterial PCO₂ for patients intubated in the field and transported to our trauma center.



The Journal of TRAUMA®

February 2008

Emergency Ventilation in Brain Injury

Table 1 Demographic Data (N = 422)

Demographics	Always Out of Target N = 138 (32.7%)	Achieved Target N = 284 (67.3%)	p
Mean age	36.7 (18)	35.7 (18.7)	0.624
Age > 55	21 (15.2%)	48 (16.9%)	0.661
% Male	97 (70.3%)	208 (73.2%)	0.531
Prehospital hypotension (SBP < 90)	18 (16.5%)	40 (14.0%)	0.550
Mechanism			
% blunt trauma	117 (84.8%)	234 (82.4%)	0.673
Motor vehicle crash	47.1%	37.7%	
Fall	10.9%	14.4%	
Pedestrian vs. vehicle	9.4%	8.2%	
Motorcycle crash	8.0%	8.2%	
Gunshot wound	10.1%	9.2%	
Prehospital GCS			
Mild (14–15)	18.3%	16.5%	0.051*
Moderate (9–13)	6.1%	15.1%	
Severe (≤8)	75.7%	68.3%	
ED hypotension	32 (23.2%)	60 (21.1%)	0.010*
ED hypoxic (Po ₂ < 100)	15 (10.9%)	28 (9.9%)	0.731
ED acidosis (ABD >6)	45 (32.6%)	70 (24.6%)	0.043*
Mean ISS	28.0 (13)	26.4 (13.1)	0.253
ISS > 25	75 (54.3%)	162 (57%)	0.665
AIS chest	2.0	1.8	0.201
Head injury severity			
AIS head 4–5	60 (43.5%)	141 (49.6%)	0.233
AIS head 3–5	83 (60.1%)	179 (63.0%)	0.574

Student's *t* test for continuous variables χ^2 for categorical variables.

* Variable included in multivariate logistic regression model.

Prehospital Hypocapnia and Poor Outcome After Severe Traumatic Brain Injury

Eileen V. Caulfield, PhD, RN, Richard P. Dutton, MD, MBA, Douglas J. Floccare, MD, MPH, Lynn G. Stansbury, MD, MPH, and Thomas M. Scalea, MD

J Trauma. 2009;66:1577–1583.

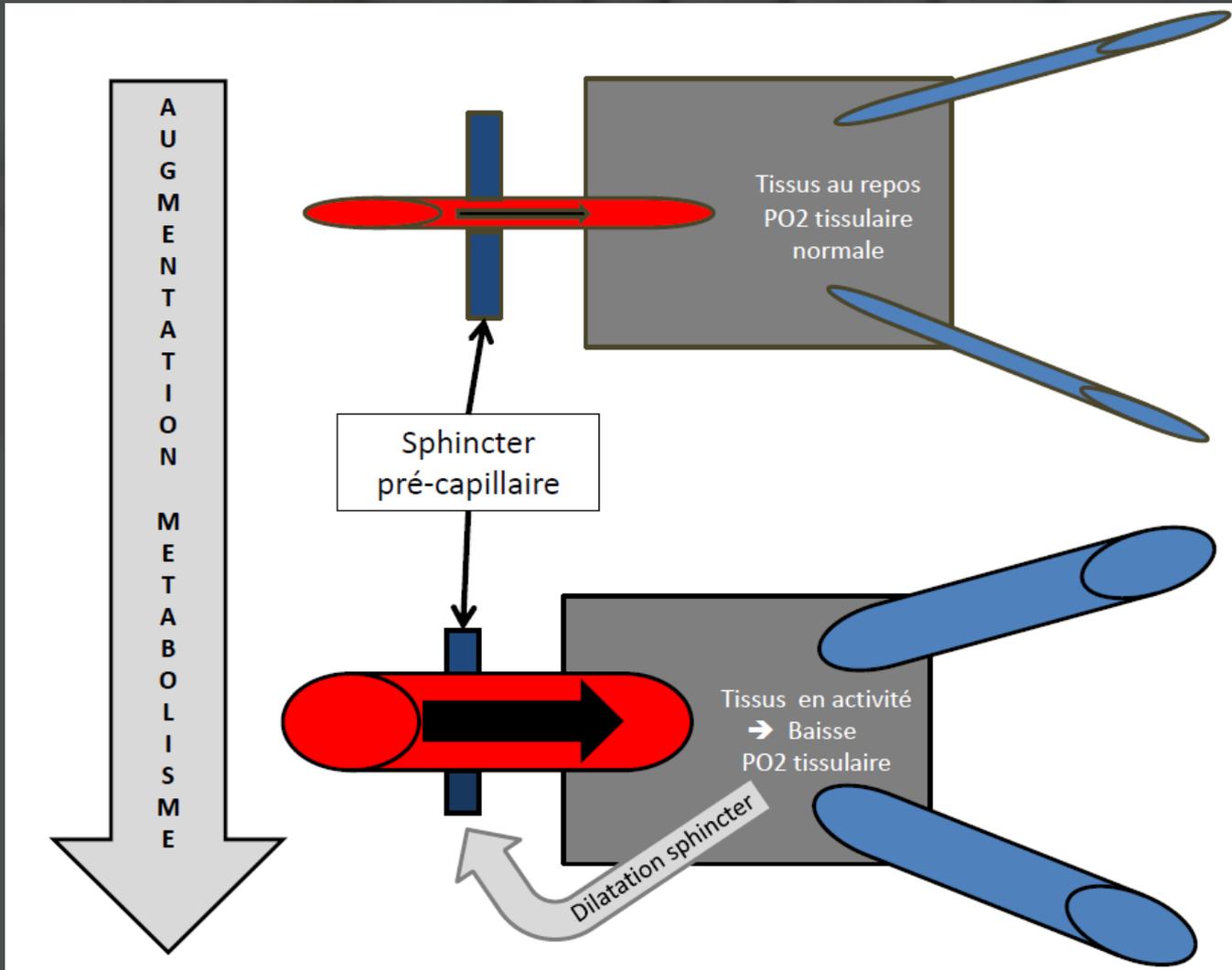
Table 4 Odds Ratio for Mortality by End-Tidal Carbon Dioxide (ETCO₂) Above or Below Levels Recommended by the BTF Guideline on Hyperventilation in Patients With Severe Traumatic Brain Injury Ventilated During Transport

Group	Mortality: n (%)	Odds Ratio (95% CI)	<i>p</i>
"Achieved" (n = 65)	19 (29%)	0.49 (0.21, 1.1)	0.10
"Not achieved" (n = 35)	16 (46%)		

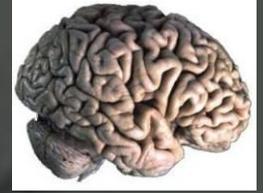
"Achieved," end-tidal carbon dioxide >29 mm Hg; "not achieved," end-tidal carbon dioxide <30 mm Hg.

p, probability associated with no difference between the groups, χ^2 for discrete variables.

Régulation du débit tissulaire (en dehors du cerveau)



Le cerveau



- Le cerveau, un organe à part...
- 2% masse corporelle, 20% de l'ATP produite
- Débit sanguin cérébral (DSC) = 50ml/100g (2/3 SG)
- 40% du DSC → maintien intégrité cellulaire
- DSC = 14% du débit cardiaque
- Réserve en O₂ et glucose
→ 3 minutes
- ➔ O₂ ne peut pas être le médiateur ...

Sinon ...



Couplage métabolique (CM) au niveau cérébral

- Adaptation du DSC au besoin local
- Mécanisme de régulation spécifique au **SNC**
- Adéquation entre les besoins et les apports
- 3 déterminants du DSC
 - Concentration en O_2
 - Concentration en CO_2
 - Concentration en H^+



Doppler study of middle cerebral artery blood flow velocity and cerebral autoregulation during a simulated ascent of Mount Everest

ARAM Ter MINASSIAN, MD; LAURENT BEYDON, MD; MAURO URSINO, PhD; BERNARD GARDETTE, PhD; CLAUDE GORTAN, PhD; JEAN PAUL RICHALET, MD, PhD

Table 1. Physiological parameters at various altitudes

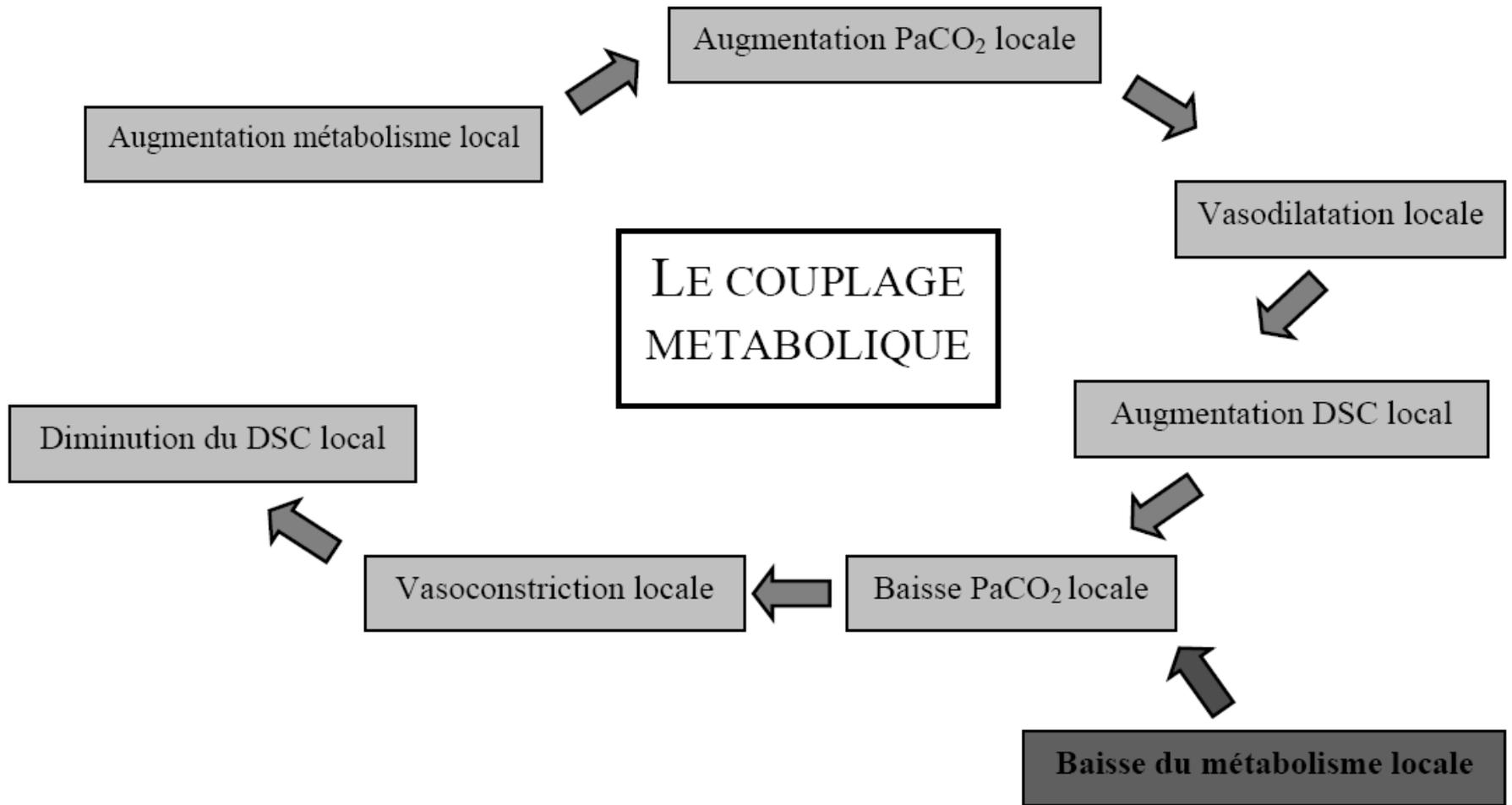
<i>Altitude</i>	<i>Barometric pressure (mm Hg)</i>	<i>MCAv (cm·s⁻¹)</i>	<i>PI</i>	<i>RI</i>	<i>PaO₂ (mm Hg)</i>
SL	760	65	0.83	0.54	101.8
± SD		5	0.12	0.04	3.9
5000 m	422	66	0.82	0.53	51.1*
± SD		7	0.13	0.05	3.4
6000 m	370	67	0.64*	0.44*	49.3*
± SD		8	0.13	0.06	3.3
7000 m	324	74	0.68*	0.46*	40.2*
± SD		15	0.11	0.05	5
8000 m	284	93*	0.59*	0.43*	37*
± SD		28	0.09	0.04	4.6
RSL	760	75	0.79	0.52	102.3
± SD		11	0.07	0.03	6

† Below 8000 m, *n* = 8; at 8000 m, *n* = 7; SL indicates sea level.

* Statistically significant difference between means for each hose significant level.

Couplage métabolique (CM) → CO₂

- Spécificité cérébrale → 2 niveaux de régulation
- Permet de maintenir la PtiO₂ au dessus du seuil de régulation de l'oxygène
- « Perfusion de luxe » par rapport aux autres organes



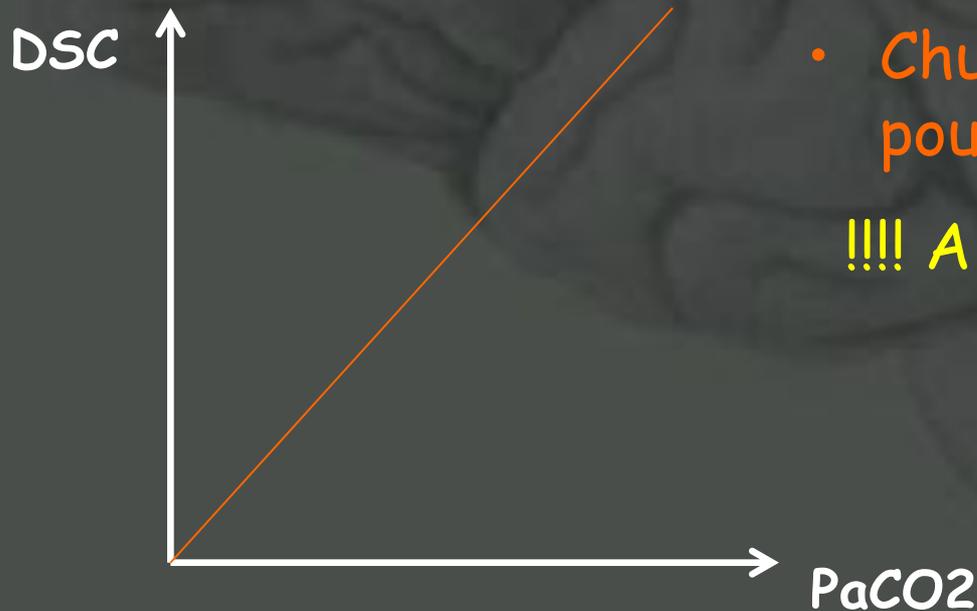
CO₂ = niveau zéro pour le cerveau

CO₂ médiateur utilisé car Tx systémique parfaitement stable

Problème = diffusion du CO₂

PaCO₂ et DSC

➤ Relation linéaire entre la PaCO₂ et le DSC pour des valeurs de PaCO₂ comprises entre 20 et 90mmHg



• Chute de DSC = 1 à 2 ml /100g pour 1 mmHg

!!!! A besoins constants en O₂ !!!!

➔ Ischémie +++

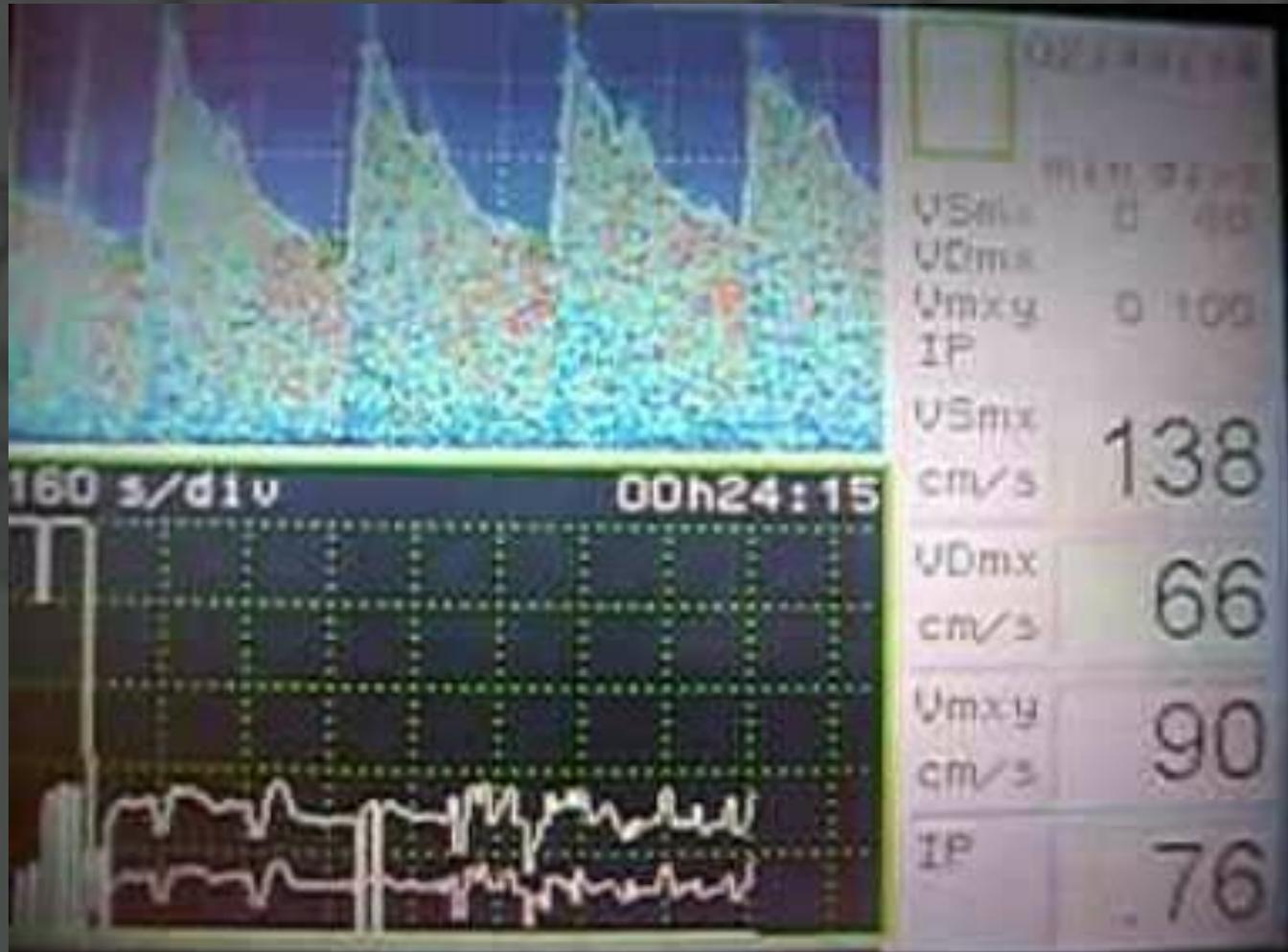
Doppler trans crânién normal



Hypocapnie



Hypercapnie



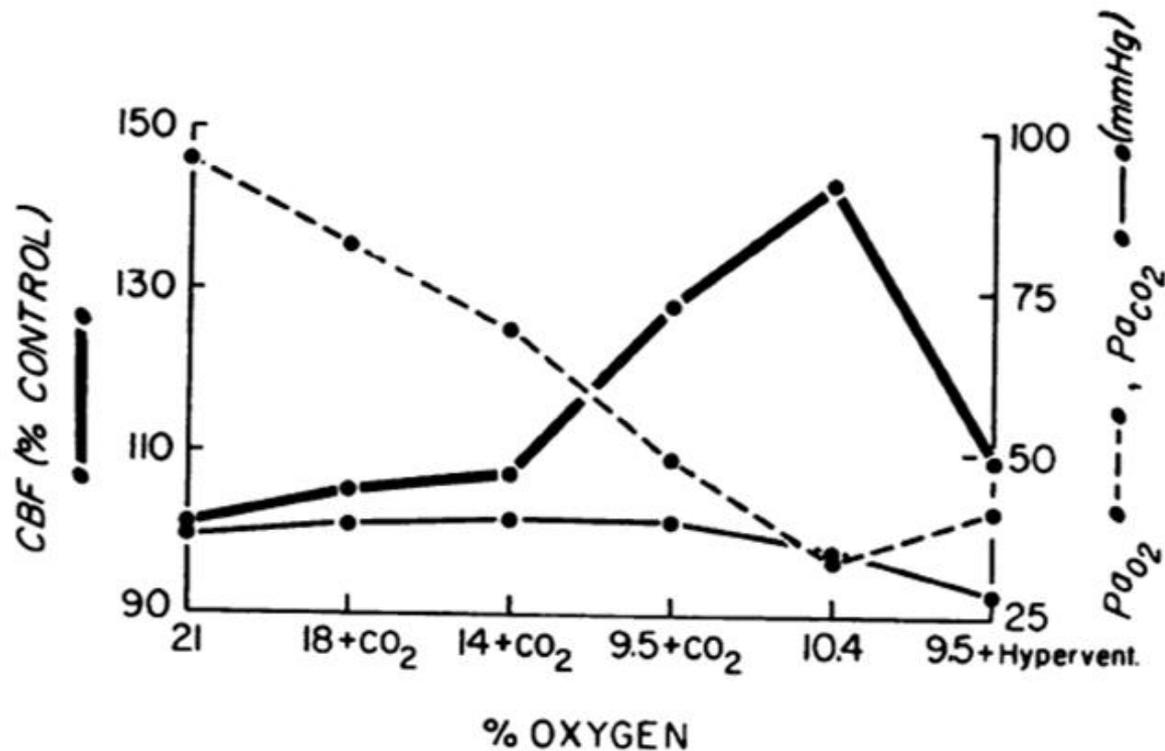


FIGURE 5 The mean effects of eucapnic and hypocapnic hypoxia and of hyperventilation on mean cerebral blood flow and arterial O₂ and CO₂ tensions in 10 subjects. See

Cerebrovascular Response to Acute Hypocapnic and Eucapnic Hypoxia in Normal Man

WILLIAM SHAPIRO, ALBERT J. WASSERMAN, JAMES P. BAKER, and JOHN L. PATTERSON, JR.

The Journal of Clinical Investigation Volume 49 1970



Le CO₂

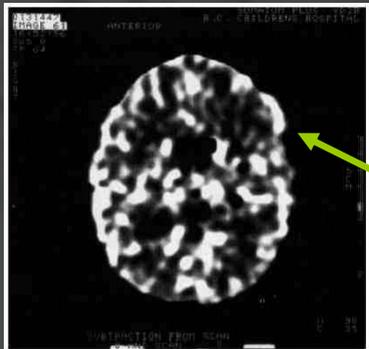


Pas de neuro-réanimation sans monitoring de l' EtCO₂

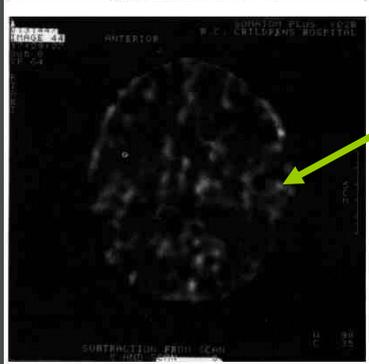
« L'hypocapnie est transitoire, ses effets sont définitifs »
D. Payen JMT SFAR 2006



!!! Vasoconstriction à besoins constants en O₂ !!!



PCO₂=45mmHg, PIC=44mmHg, PPC=54mmHg, DSC global=59ml/min/100g



PCO₂=30mmHg, PIC=15mmHg, PPC=82mmHg, DSC global=14ml/min/100g avec des débits régionaux <10ml/min/100g = seuil d'ischémie irréversible

D'où l'intérêt du monitoring multimodal Skippen et al CCM 1997

NORMOCAPNIE +++ dans la souffrance cérébrale

L'avenir

Perspectives thérapeutiques

- Optimisation hémodynamique générale à la phase aigue
- Les B-Bloquants (RR 0,29) *Bryan et al J of Trauma 2007*
- Anti-apoptose (EPO, CEPO...) ● ● ●
- la Progesterone *Xiao et al, CCM 2008*
- Le lactate hyperosmolaire, Anti-NMDA

Moyens fiables d'évaluer le pronostic (IRM, Electrophysiologie...)

Les réseaux de prise en charge +++

- ➔ Avis précoce neuroréanimateur et neurochirurgien
- ➔ Télétransmission des TDM
- ➔ Pour discuter mutation précoce en neuro-réanimation

Conclusion

- Le TC grave
 - Avis neurochirurgical systématique
 - +/- Avis neuroréanimation
- Monitoring PIC +++ dès que AG + risque HTIC
- Monitoring DTC pour détecter HTIC puis pour optimiser la PPC
- Si HTIC persistante malgré optimisation systémique + sédation standard (Hypnovel + Morphine) → Mutation en centre spécialisé pour optimisation monitoring +/- chirurgie de l'HTIC