Complications neurologiques de la chirurgie des cardiopathies congénitales

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NEM

- Epidémiologie
- Rappels physiopathologiques concernant la perfusion cérébrale
- Techniques de perfusion
- Neuromonitoring peropératoire
- Particularités du cerveau néonatal
- Surveillance postopératoire
- Prévention / traitement …

Epidémiologie des principales complications neurologiques après chirurgie cardiaque

Atteintes sévères (2-25%)

- AVC
- Convulsions
- Choréoathetose (survient à J2-J6 → <1% depuis la perfusion cérébrale sélective)
- altération de l'état de vigilance / coma

Majnemer, J Pediatrics 2006

Autres atteintes

- 41% ont anomalie à l'ex neuro < 2 ans
- 26% ont un retard global du développement

Limeropoulos, J Pediatrics 2002

- 7 ans après cure de TGV: une altération de la reconnaissance des expressions faciales *Calderon, Dev Med Child Neurol. 2014*

Table 1. Factors Influencing Neurologic Injury

Fixed factors
Genetic syndromes
Structural CNS malformations
Socioeconomic status
Genetic predispositions
Modifiable factors
Preoperative
Hypotension
(Glycemic control)
(Hyperthermia)
Operative
Emboli—gas, particulate
CPB circuit/inflammation
Modified ultrafiltration
pH-stat blood gas management
Cerebral ischemia
Steal
Hemodilution
Free radicals/reoxygenation

Hsia, ATS 2006

Facteurs de risque de l'atteinte neurologique postopératoire

- Le génotype ϵ 2 de l'apolipoprotéine E (APO E2) (rôle dans le remodelage de la membrane neuronale et des synapses)

  Gaynor, SeminThorac Cardiovasc Surg 2004

- chirurgie palliative

  Majnemer, J Pediatrics 2006

- ↓ débit cérébral néonatal préop à 20ml/100g/min (vn : 50) et faible réactivité au CO_2 chez les NN porteurs de cardiopathie → 28% LPV, 24% microcéphalie

  Licht, JTCVS 2004

- HLHS : étude postmortem (n = 41): 10% agénésie du corps calleux et holoprosencephalie, 27% microcéphalie, 21% anomalies giration corticale

  Glauser, Pediatrics 1990

Facteurs de risque de l'atteinte neurologique postopératoire
La réponse inflammatoire

chez l'adulte : 10% encéphalopathie, 70% altérations tests psychométriques

Puskas, ATS 2000

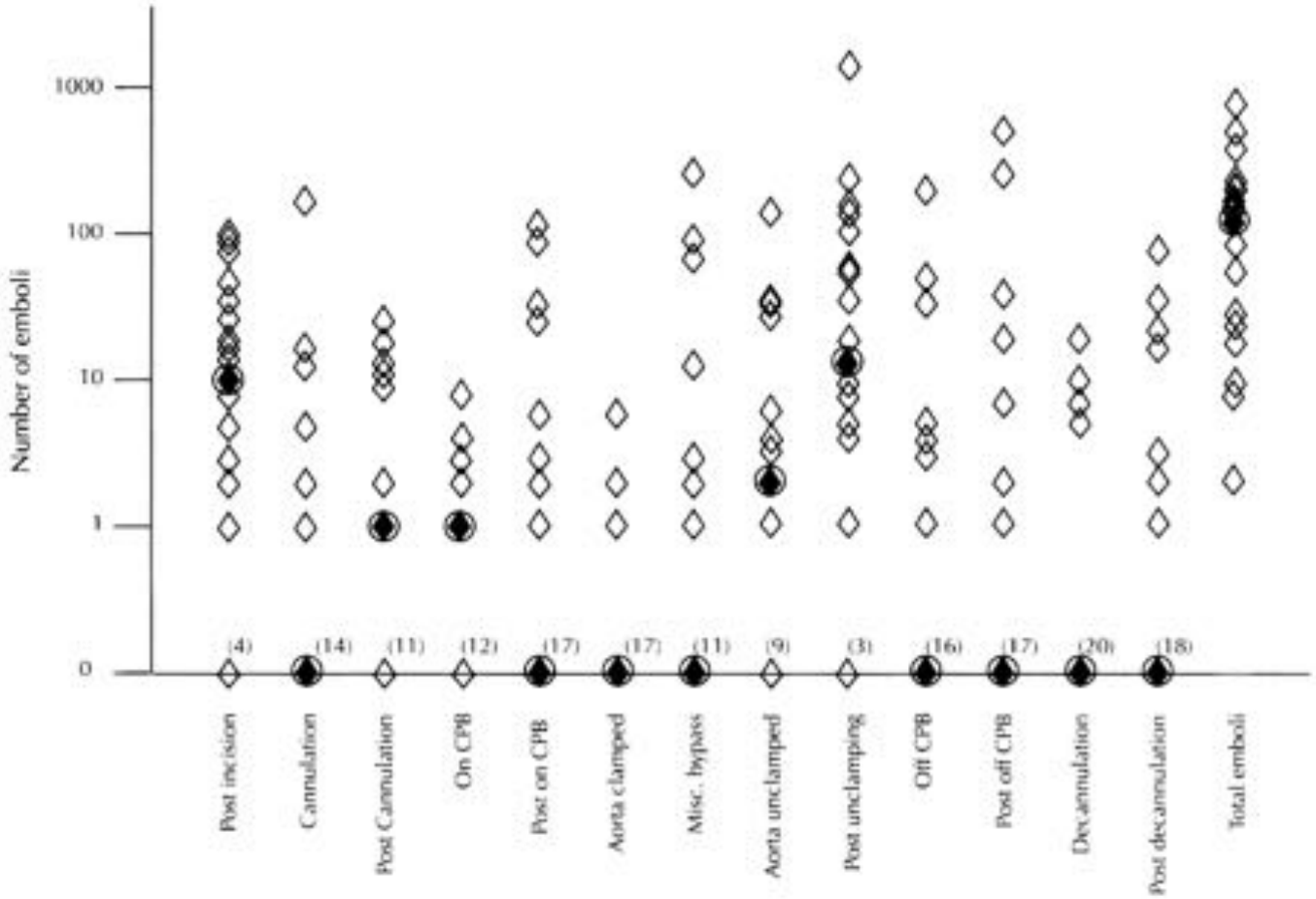
chez le NN et N risque accru de LPV, corrélé aux niveaux TNFα, IL-6, IL1-β

Gaynor, SeminThorac Cardiovasc Surg 2004

Facteurs de risque de l'atteinte neurologique postopératoire
L'embolie cérébrale gazeuse, particulaire

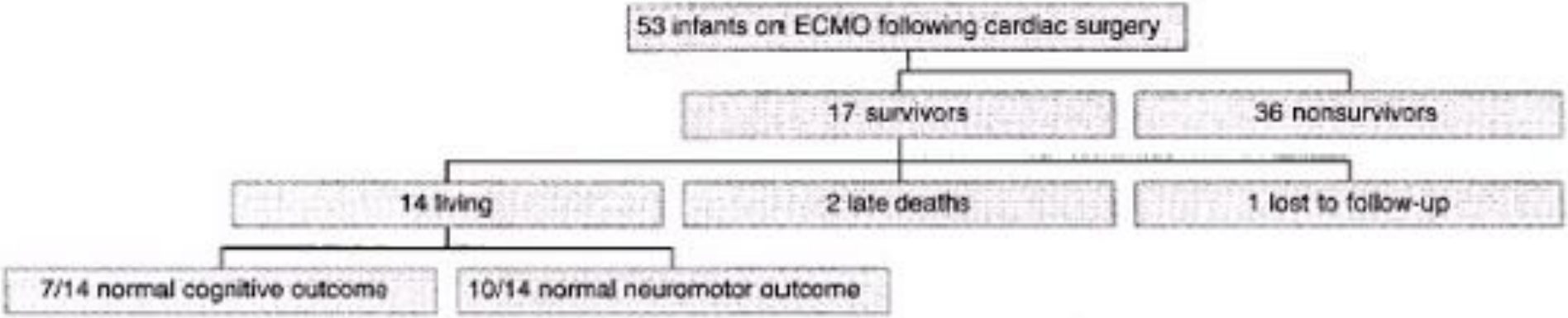
2-2664 embols / patient détectés par Doppler carotidien, risque élevé (shunt D-G, TGV) :152/patient

O'Brien, Anesthesiology 1997



Facteurs de risque de l'atteinte neurologique postopératoire
L'ECMO

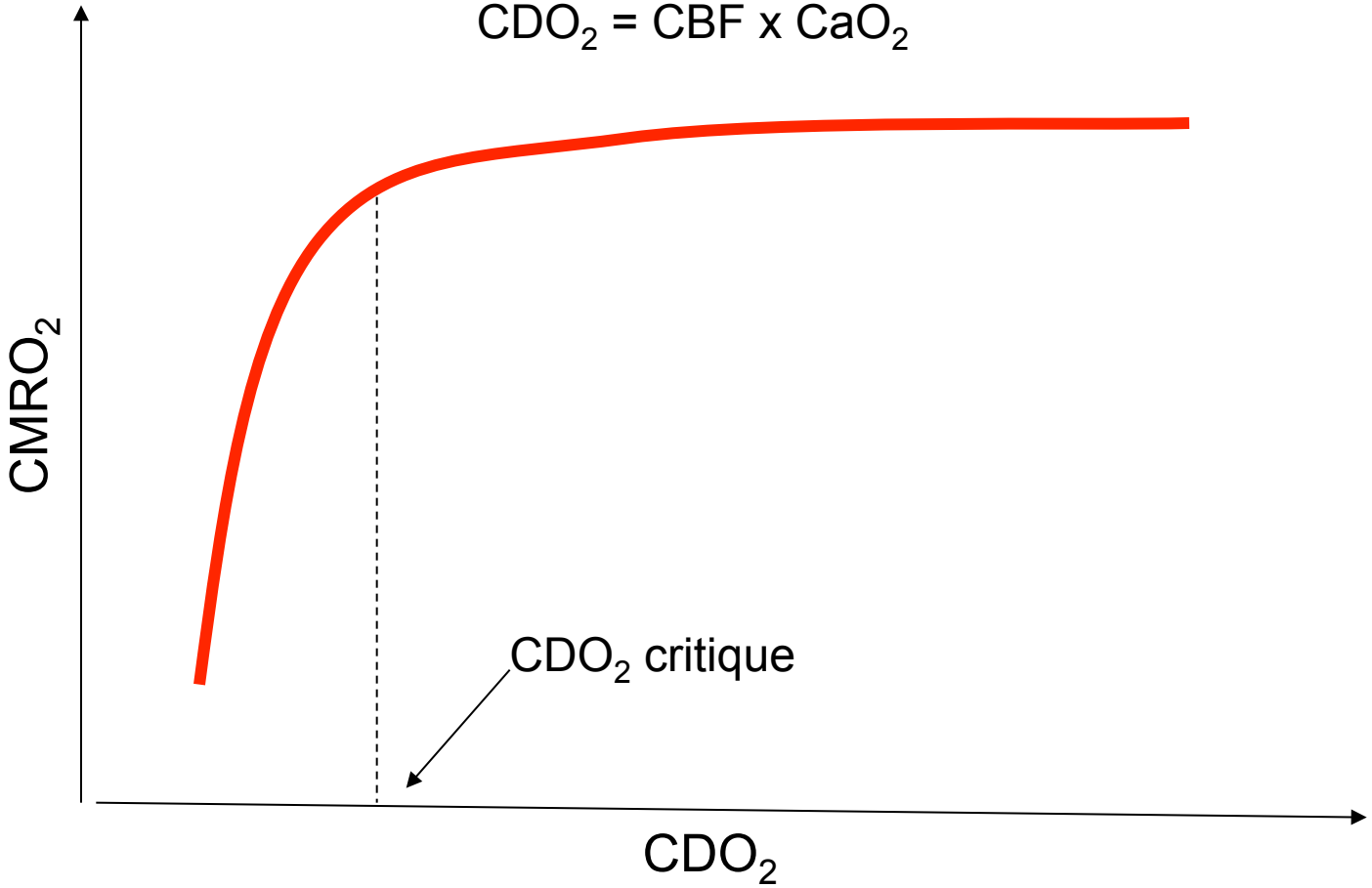
ECLS Registry Report 2003; survie NN 37%, enfants 42%



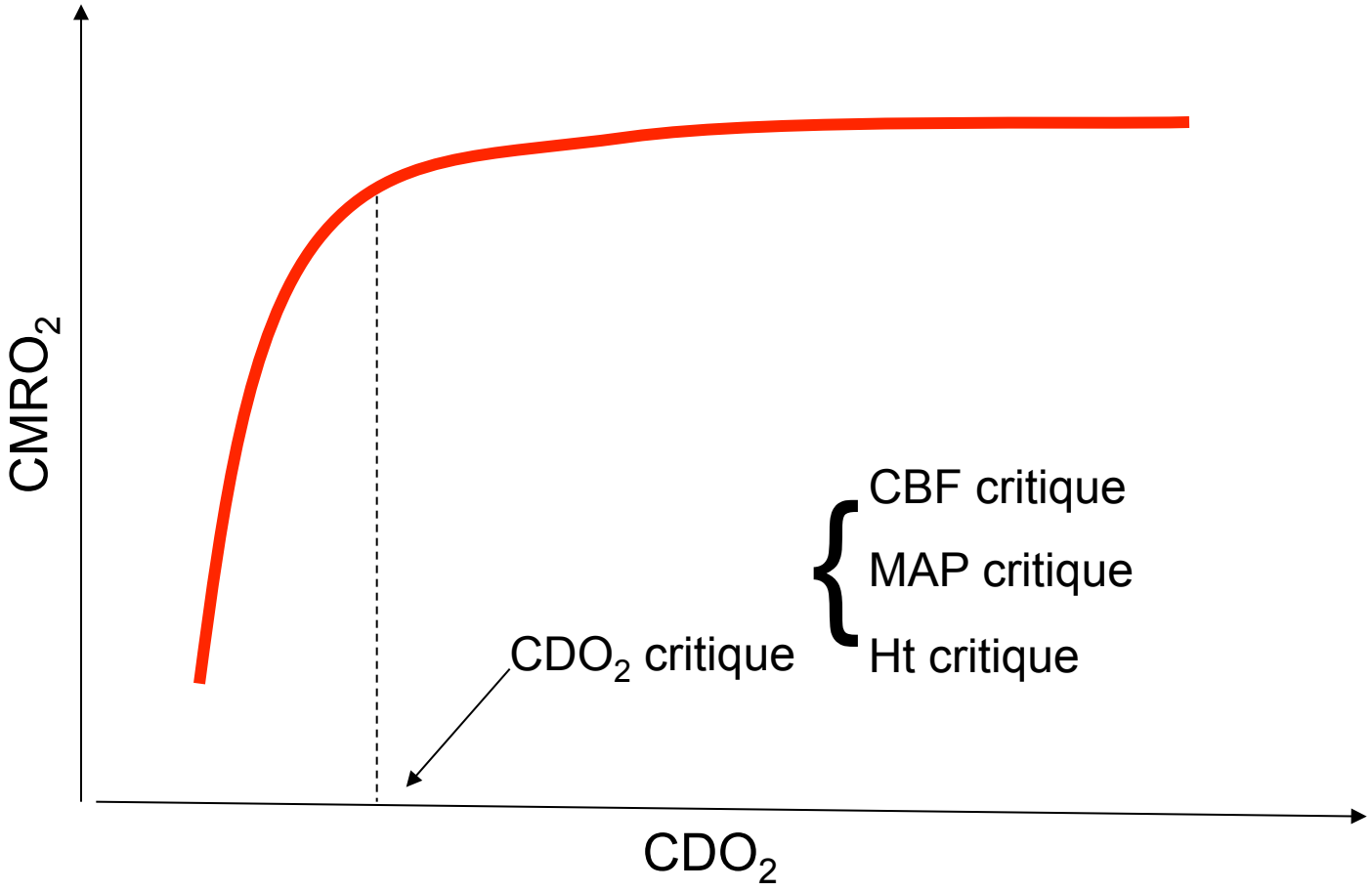
Le rapport $CMRO_2/CD0_2$

$CMRO_2 = CBF\ (CaO_2\text{-}CvjO_2)$

$CDO_2 = CBF \times CaO_2$

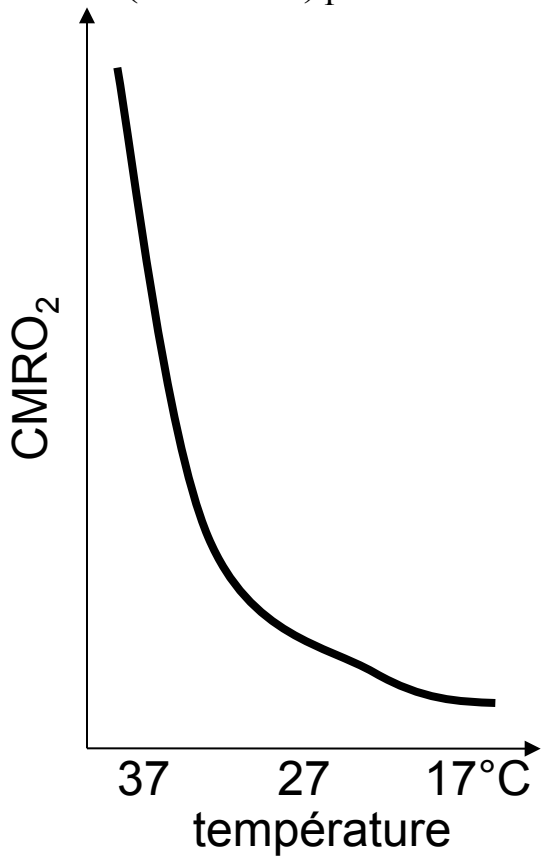


Le rapport CMRO2/CD02



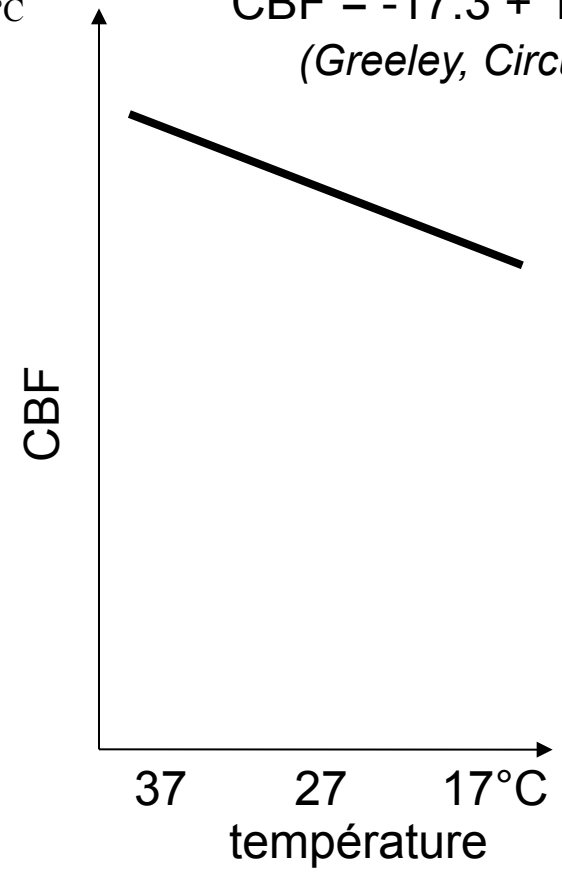
Effet de l'hypothermie sur la $CMRO_2$ et CDO_2

$$(CMRO2)_T = e^{0.1171(T-37)} * (CMRO2)_{37°C}$$

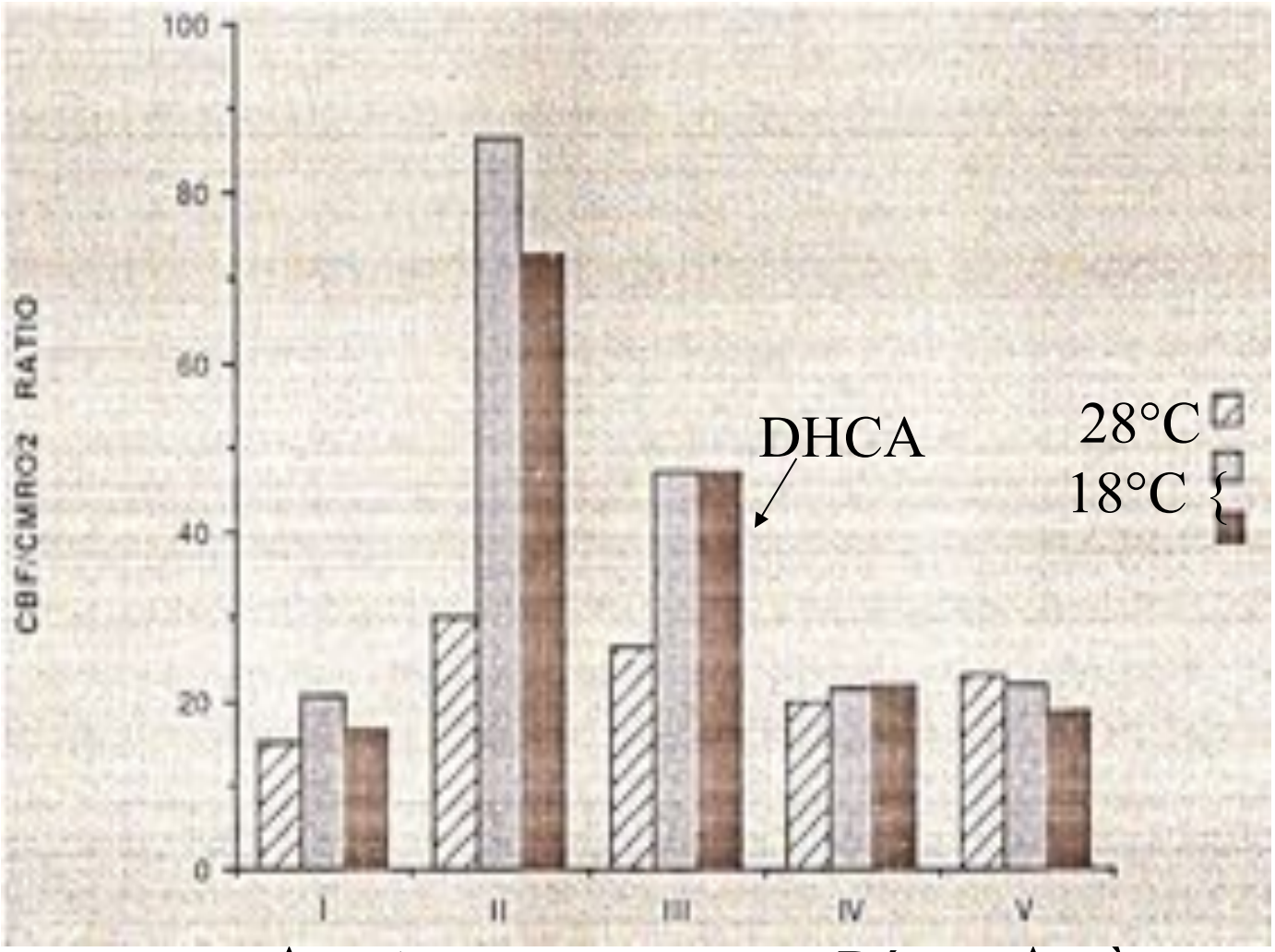


CBF = -17.3 + 1.47 x temp

(Greeley, Circulation 1989)



Effet de l'hypothermie sur la CMRO2 et CDO2



Avant CEC | CEC | Ré-chauffement | Après CEC

J Thorac Cardiovasc Surg. 1991 May;101(5):783-94.

The effect of hypothermic cardiopulmonary bypass and total circulatory arrest on cerebral metabolism in neonates, infants, and children.

Greeley WJ[1], Kern FH, Ungerleider RM, Boyd JL 3rd, Quill T, Smith LR, Baldwin B, Reves JG.

Q_{10} (index thermique) = % de variation du paramètre lorsque la température varie de 10°C

$Q_{10} = 3.65$

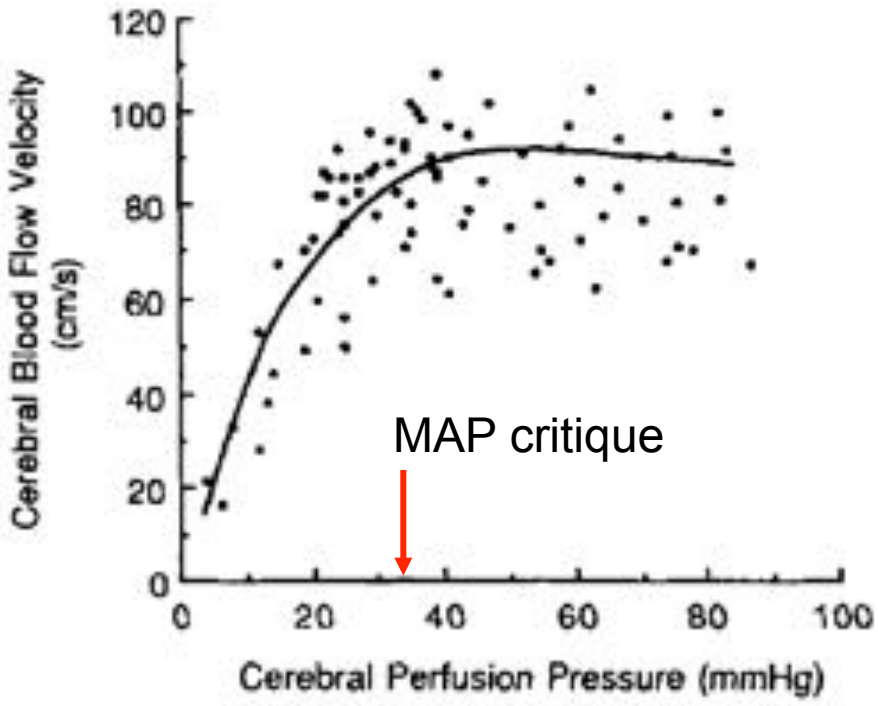
exponential relationship between temperature and cerebral metabolism and an average temperature coefficient of 3.65. There was no significant difference in the rate of metabolism reduction (temperature coefficient) in patients cooled to 28 degrees and 18 degrees C. From these data we were able to derive an equation that numerically expresses a hypothermic metabolic index, which quantitates duration of brain protection provided by reduction of cerebral metabolism owing to hypothermic bypass over any temperature range. Based on this index, patients cooled to 28 degrees C have a predicted ischemic tolerance of 11 to 19 minutes. The predicted duration that the brain can tolerate ischemia ("safe" period of deep hypothermic circulatory arrest) in patients cooled to 18 degrees C, based on our metabolic index, is 39 to 65 minutes, similar to the safe period of deep hypothermic circulatory arrest known to be tolerated

Effet de l'hypothermie sur la perfusion cérébrale

- Consommation en O2 diminue jusqu'à 27°C au dépend de la diminution du métabolisme cellulaire, jusqu'à 18°C au dépend de la diminution de l'activité électrique (silence à 20°C).
- perte de l'autorégulation, DSC ~ PAM à 18-22°C
- ↑ viscosité et résistance vasculaire
- courbe de dissociation de l'Hb à gauche
- la perte de l'autorégulation persiste en postop, (H6-H20)

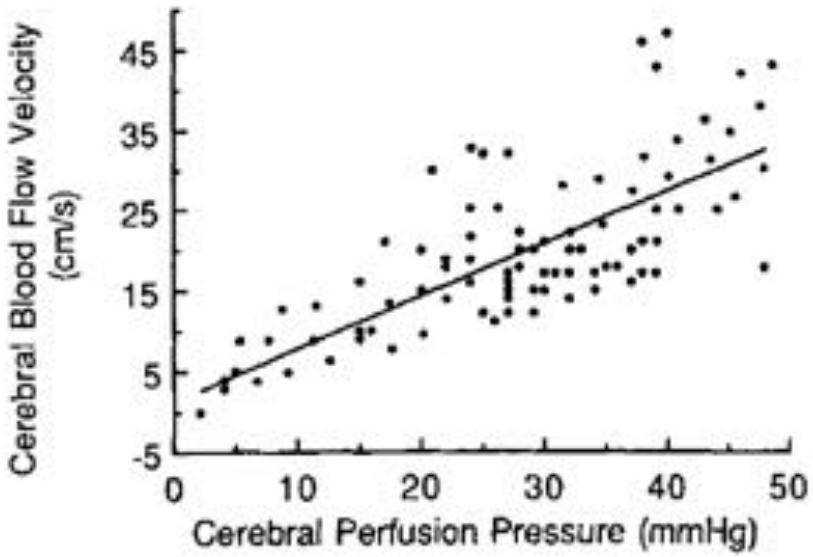
Bassan, Pediatr Res 2005

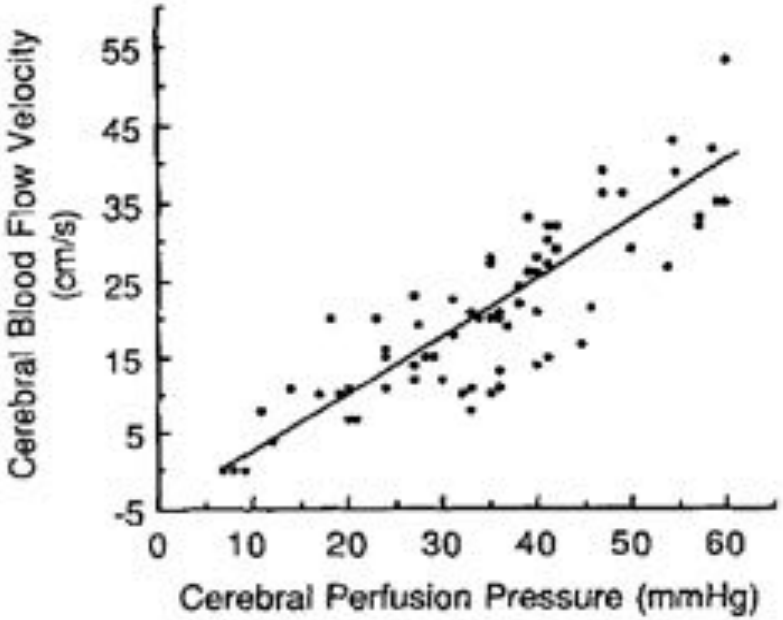
L'autorégulation du débit sanguin cérébral



nouveau-né et nourrisson
36-37°C
(Taylor, Anesth Analg 1992)

Perte de l'autorégulation en hypothermie





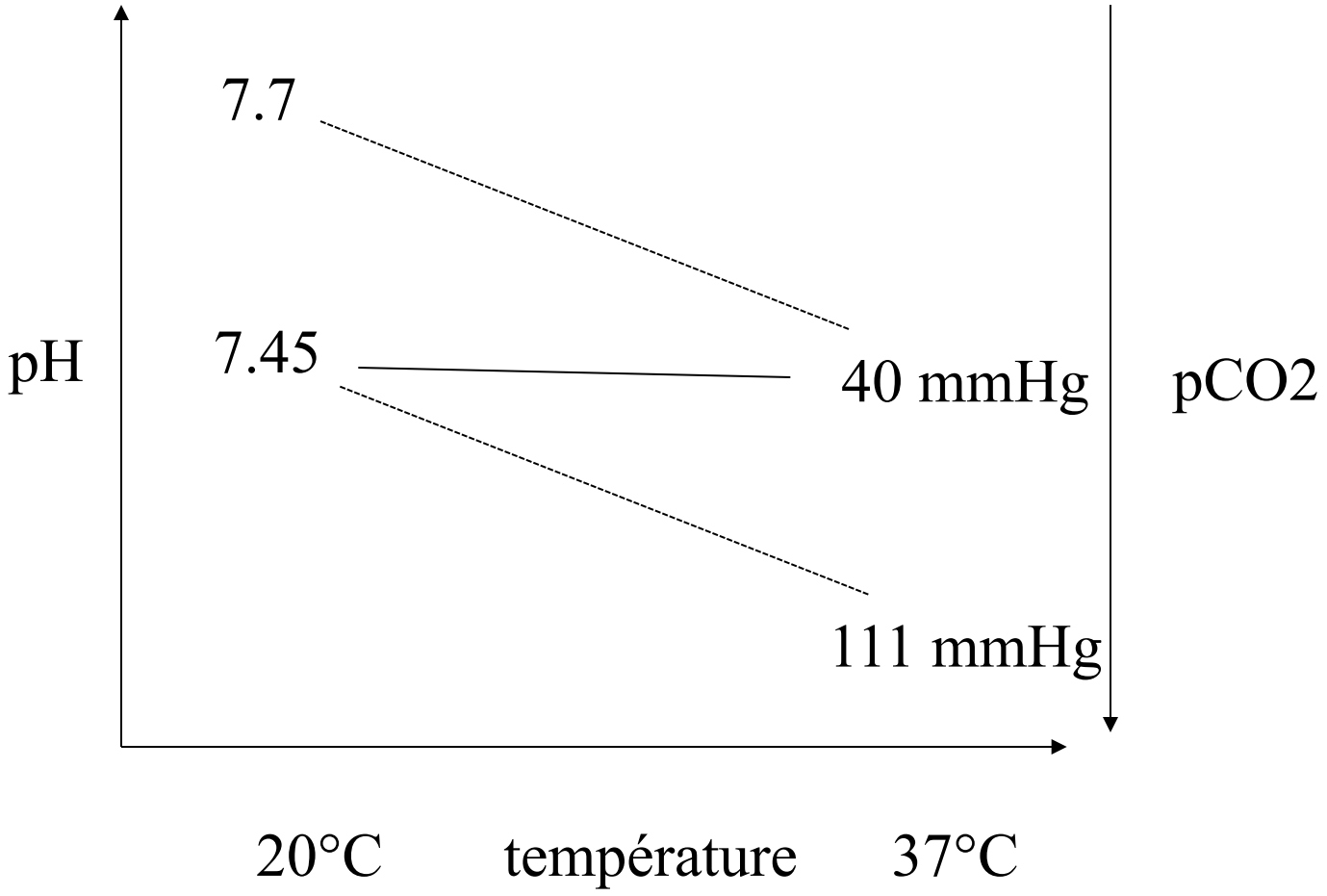
nouveau-né et nourrisson
23-25°C
(Taylor, Anesth Analg 1992)

nouveau-né et nourrisson
14-20°C
(Taylor, Anesth Analg 1992)

Solubilité des gaz et constante de dissociation diminuent en hypothermie

- L'eau devient alcaline en hypothermie
- La chaîne respiratoire fonctionne de manière optimale en hypothermie lorsque le pH intracellulaire suit la variation du pH de l'eau

α–stat ou pH–stat ?

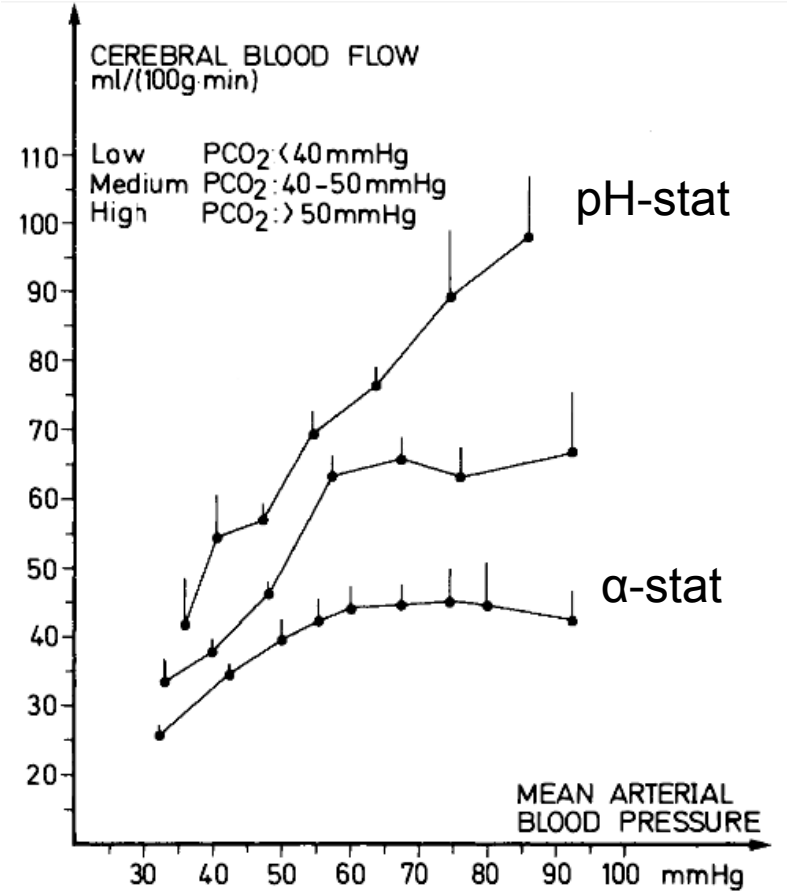


Solubilité des gaz et constante de dissociation diminuent en hypothermie

α-stat:
pCO2 = const
pH = var

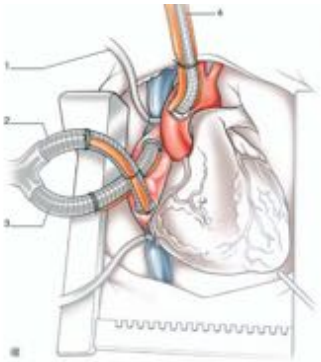
pH-stat:
pCO2 = var
pH = const

1. -> vasodilatation cérébrale
2. -> perte de l'autorégulation CBF



adultes, 22-28°C
(Henriksen, J Cereb Blood Flow Metab,1986)

Techniques de perfusion



1. Au débit théorique en normothermie
2. Low flow en hypothermie
3. Perfusion cérébrale sélective (regional low-flow) en hypothermie
4. Arrêt circulation en hypothermie profonde

Techniques de perfusion : Low-flow

Boston Circulatory Arrest Study 1988-1992

(Newburger NEJM 1993, Bellinger, NEJM 1995, Bellinger, Circulation 1999, Bellinger, JTCVS 2003)

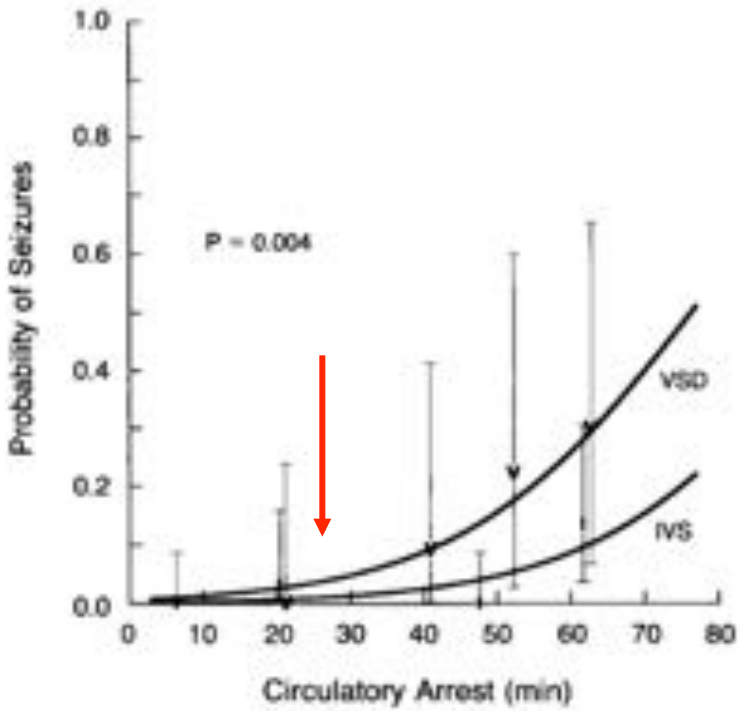
171 nouveau-nés avec TGV, 87 DHCA + 84 low-flow, 18°C, α-stat, Ht>20%

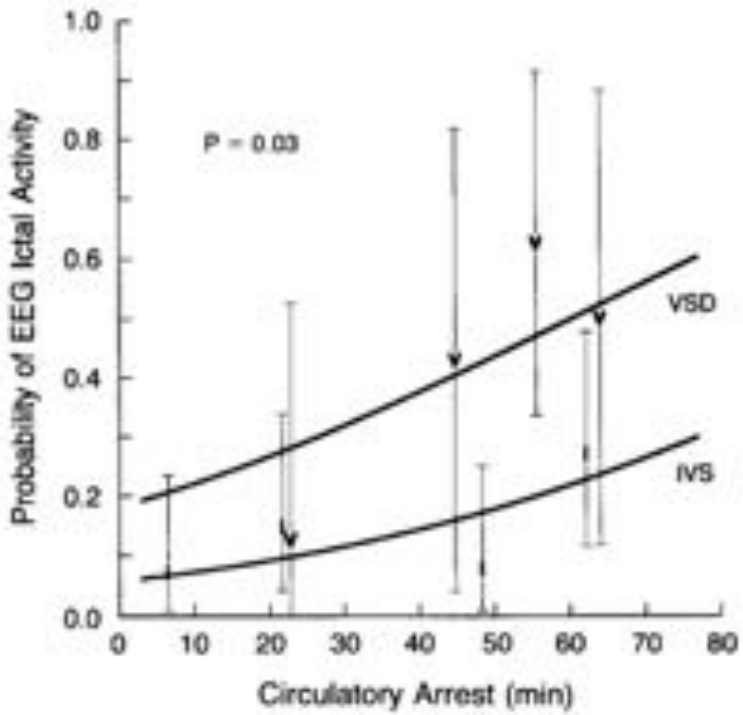
-> convulsions < 48h: OR=11.4 pour DHCA
-> activité électrique critique < 48h: OR=2.5 pour DHCA
-> index de développement psychomoteur à 1 an plus bas dans le groupe DHCA (différence 6.5 points)
-> plus d'apraxie motrice à 4 ans dans le groupe DHCA
-> plus d'apraxie motrice et idéatoire à 8 ans dans le groupe DHCA

CONCLUSIONS: Use of total circulatory arrest to support vital organs during heart surgery in infancy is generally associated with greater functional deficits than is use of low-flow cardiopulmonary bypass, although both strategies are associated with increased risk of neurodevelopmental vulnerabilities.

Techniques de perfusion (3)
Arrêt circulatoire en hypothermie profonde

Boston Circulatory Arrest Study
87 nouveau-nés avec TGV, DHCA, 18°C, α-stat, Ht>20%
survenue de convulsions < 48h





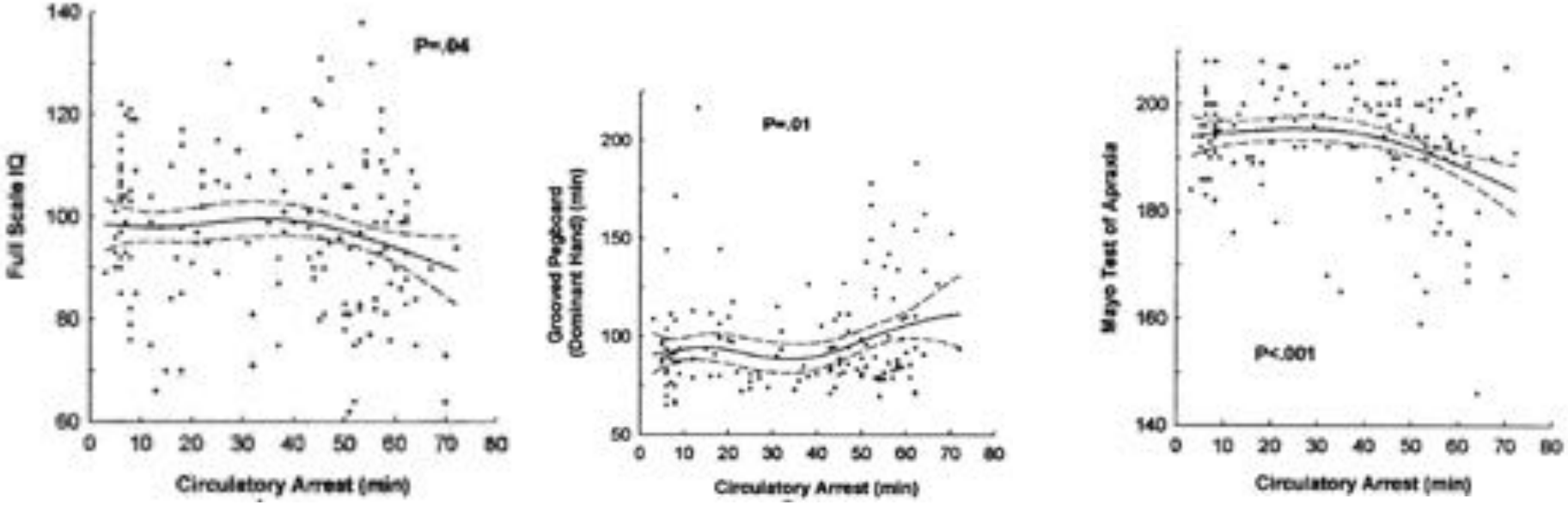
(Newburger, NEJM 1993)

Techniques de perfusion (3)
Arrêt circulatoire en hypothermie profonde

Boston Circulatory Arrest Study

155 nouveau-nés avec TGV, 18°C, α-stat, Ht > 20%

tests psychomoteurs à 8 ans



(Wypij, JTCVS 2003)

La limite de l'IC 95% était à 32min

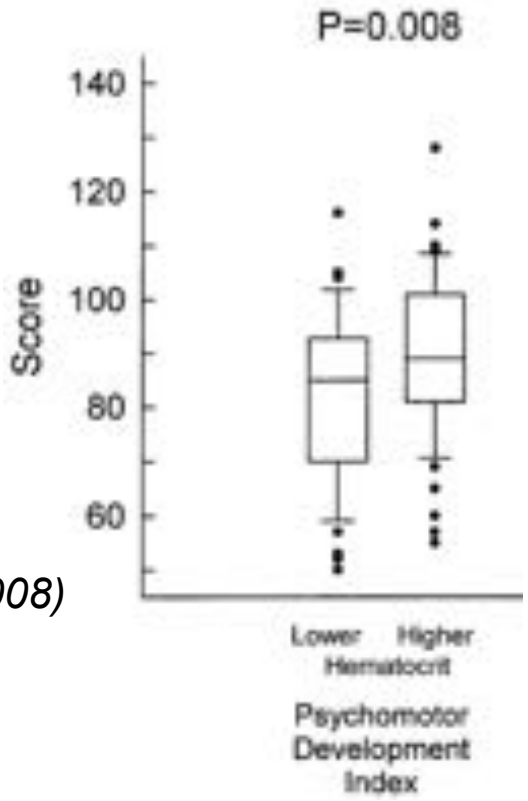
L'hémodilution en hypothermie

147 nouveau-nés, 74 Ht=20%,73 Ht=30%
Low-flow, 18°C, 0.75L/min/m2, pH-stat
Tests psychométriques à 1 an

(*Jonas, JTCVS 2003)*

124 nouveau-nés, 56 Ht=25%, 68 Ht=35%
Tests psychométriques et IRM à 1 an
-> aucune différence

(Newburger,JTCVS 2008)



Autres

- pH-stat en hypothermie *(Hickey, ATS 1998 du Plessis JTCVS 1997)*
- Refroidissement progressif > 20min *(Bellinger, Pediatrics 1991)*
- Reperfusion froide durant 10min après DHCA *(Rodriguez, JTCVS 1995)*
- Eviction hyperthermie post-CEC *(Thong, Anesth Analg 2002)*

Neuromonitoring peropératoire
EEG et index bispectral (BIS)

- obtention du silence électrique en hypothermie avant et durant DHCA

- éviction du réveil lors du réchauffement

- diagnostic précoce de l'embolie

(Andropoulos, Anesth Analg 2004)

Le Doppler transcrânien

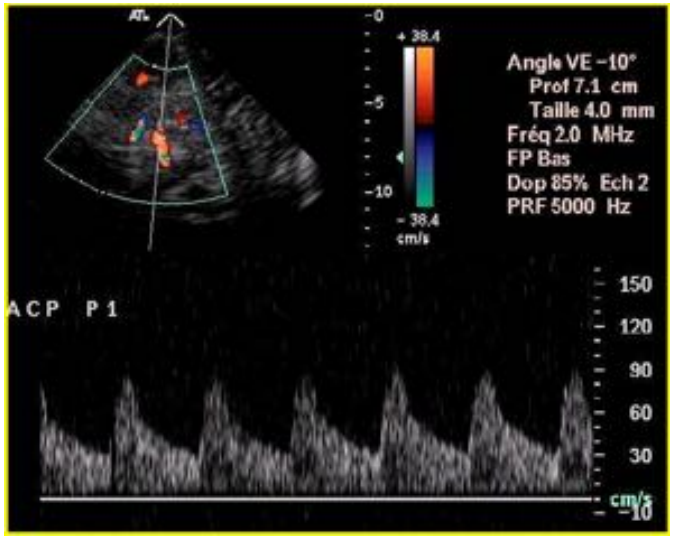


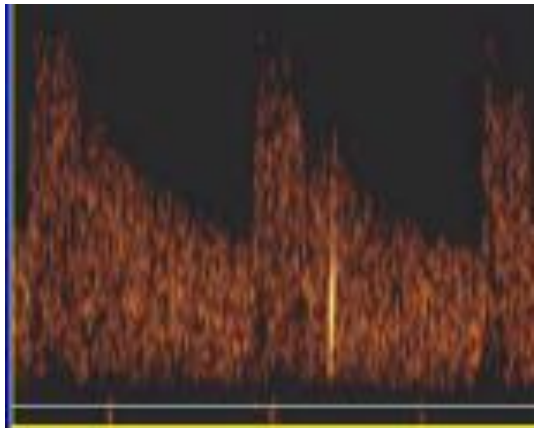
Table 1. Normal Transcranial Doppler Velocities for Infants and Children

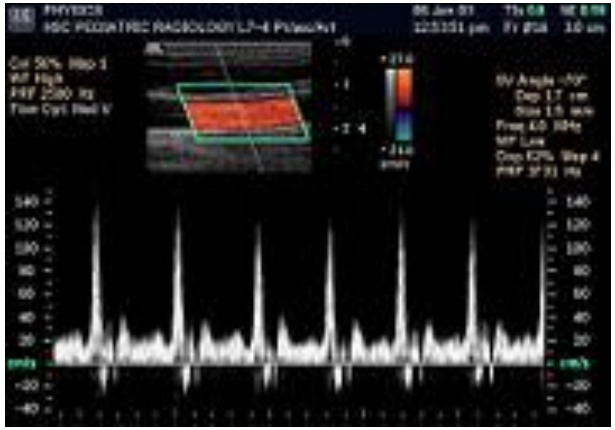
Age	Depth (mm)	Mean velocity (cm/s)	Peak systolic velocity (cm/s)	End-diastolic velocity (cm/s)
0–3 mo	25	24–42 ± 10	46–75 ± 15	12–24 ± 8
3–12 mo	30	74 ± 14	114 ± 20	46 ± 9
1–3 yr	35–45	85 ± 10	124 ± 10	65 ± 11
3–6 yr	40–45	94 ± 10	147 ± 17	65 ± 9
6–10 yr	45–50	97 ± 9	143 ± 13	72 ± 9
10–18 yr	45–50	81 ± 11	129 ± 17	60 ± 8

(Andropoulos, Anesth Analg 2004)

Le Doppler transcrânien

Embolie





Intérêt limité en cas de vol diastolique

Near Infrared Spectroscopy (NIRS):

Neuromonitoring peropératoire multimodal (4)

250 nouveau-nés, nourrissons et enfants, rétrospective
neuromonitoring par EEG, DTC, NIRS
anomalies CDO2/CMRO2 observés dans 176 cas, interventions thérapeutiques dans 130 cas

Problem	*Intervention*	*No intervention*
Perfusion imbalance		
Peak flow velocity high (hyperemia)	3	0
Peak velocity low (aortic obstruction; low pump flow or pressure)	28	6
Doppler signal absent (no cerebral flow)	2	0
Diastolic velocity low (venous obstruction; edema)	4	4
Gas emboli	12	6
Cerebral venous oxygen desaturation		
Airway obstruction	1	0
Hemoglobin low	3	2
Fio_2 low	5	0
Carbon dioxide low	1	0
Arterial pressure or pump flow low	18	41*
Cerebral hypermetabolism with rewarming	10	11†
Cardiac output low	2	3
Insufficient anesthetic depth		
EEG frequency increase	6	2
Insufficient cooling		
EEG activity with deep hypothermia	1	0

Intervention	*No. of cases*
Perfusion imbalance	
Correct mechanical obstruction	
Aortic cannula reposition	22
Venous cannula reposition	3
Vascular clamp reposition	3
Additional deairing; repair air leak	7
Pump flow increase	15
Pump flow decrease	3
Plasma volume expansion	4
Return to cardiopulmonary support	6
Blood pressure increase	29
Carbon dioxide increase	2
Deficient oxygenation	
Increase Fio_2	5
Transfuse whole blood	3
Reintubate	1
Insufficient anesthetic depth	
Increase anesthetic delivery	8
Limited ischemia tolerance	
Neuroprotectant	18
Additional cooling	1

(Austin, JTCVS 1997)

Neuromonitoring multimodal (4)

	No change	*Intervention*	*No intervention*
Neurocomplication	5/74 (7%)	7/130 (6%)	12/46* (26%)
Seizure	3	5	5
Cerebral infarct	1		3
Chorea	1	1	1
Visual neglect		1	
Hypotonia		1	2
Speech disorder			1
Length of stay: median (interquartile range)	6 (4-12)	6 (5-12)	9 (5-13)†
Percent discharged < 7 days	58	51	32‡

(Austin, JTCVS 1997)

Neuromonitoring multimodal (4)

Table 3. Neurological Monitoring Treatment Algorithm for Congenital Heart Surgery

Monitor	Change	Intervention pre/post bypass	Intervention on bypass	Intervention on DHCA
NIRS (rSO_2I%)	≥20% relative to preincision baseline	Cardiac output, hgb, Fio_2, $Paco_2$	Bypass flow, $Paco_2$, MAP, temp, hgb, ↓CBFV adjust cannulae	–
	DHCA: rSO_2I < 30%, or at nadir > 30%	–	–	Reperfuse if possible
	rSO_2I ≥ 95%	–	None, or ↓CBFV – bypass flow or $Paco_2$ if >25% above baseline	–
	rSO_2I <30%	Rapid institution bypass, aggressive measures to O_2 delivery	Aggressive treatment: bypass flow, $Paco_2$, MAP, temp, hgb, ↓CBFV-adjust cannulae	Reperfuse if possible
TCD (mean CBFV, cm/s)	≥25% from preincision baseline	↓ transducer; ↓NIRS—if low, cardiac output, $Paco_2$, MAP	↓ transducer; ↓NIRS—if low: bypass flow, $Paco_2$, MAP, adjust cannulae	–
	≥25% from preincision baseline	$Paco_2$, anesthetic depth MAP	$Paco_2$, anesthetic depth, MAP	–
	Emboli: more than isolated HITS	De-air all infusions, Trendelenberg, stop rapid fluid boluses, search for and treat air entrainment in surgical field, assess TEE for air, return to bypass for de-airing	De-airing maneuvers, Trendelenberg, assess TEE for air, slow wean from bypass	–
BIS	≥80	Anesthetic depth	Anesthetic depth	
	No isoelectric EEG prior to DHCA		Additional cooling time; lower temperature before DHCA	
	<30 during rewarming bypass		Reduce or discontinue volatile agent on bypass pump	

(Andropoulos, Anesth Analg 2004)

Particularités du cerveau néonatal

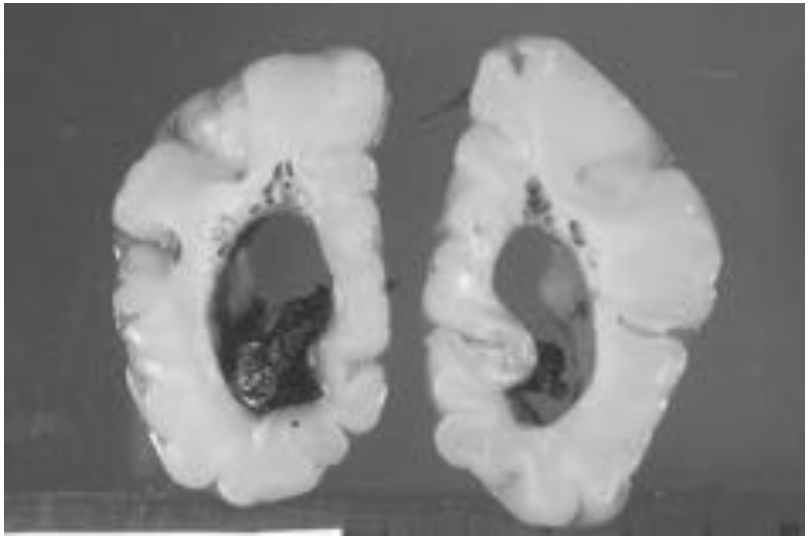
- métabolisme intense, extrait > 35% de l'O2

- débit sanguin = 2-3 x celui de l'adulte

- utilise exclusivement du glucose

- Myélinisation en cours par des olygodendrocytes – matière blanche très susceptible à l'hypoxémie -> LPV (>50% NN et petits N en postop immédiat, *Gaynor, Semin Thorac Cardiovasc Surg 2004*)

La leucomalacie périventriculaire (LPV)



Nécrose cellulaire focale dans la matière blanche entourant la paroi latérale des ventricules latéraux,

- 50% NN à l'IRM à J6-14 postop (*Mahle,2002*)
- ~ Durée CEC, hypoTAD et hypoxémie postop
- infirmité motrice cérébrale + retard mental > difficultés d'apprentissage, déficits visuels, moteurs, de l'attention, hyperactivité
- IRM : nécrose focale et œdème (1-2sem)-> stade kystique (valeur prédictive ?)

-> évolue vers l'atrophie cérébrale, hydrocéphalie, microcéphalie

LPV et cardiopathie congénitale

Mahle,
Circulation 2002

n = 24
dont 8 HLHS

	préop		< 2 sem
Pt	Pre-op MRS	Pre-op MRI	Early Post-op MRI
1	Lactate	Infarct (parietal)	PVL
2	NA	—	PVL
3	—	—	—
4	NA	—	NA
5	Lactate	Infarct (caudate)	Infarct (caudate), hemorrhage (parietal)
6	NA	—	Infarct (parietal-occipital)
7	Lactate	—	NA
8	—	—	—
9	—	—	NA
10	—	—	PVL
11	Lactate	—	PVL, infarct (parietal-occipital)
12	NA	—	PVL
13	Lactate	—	—
14	Lactate	PVL	PVL; infarct (frontal/parietal); hemorrhage (frontal)
15	—	—	Hemorrhage (frontal)
16	Lactate	Hemorrhage (temporal)	Infarct (parietal); hemorrhage (parietal)
17	—	—	PVL
18	Lactate	—	PVL; hemorrhage (frontal)
19	Lactate	PVL	—
20	Lactate	PVL	PVL; hemorrhage (thalamus)
21	—	—	—
22	NA	—	—
23	—	—	PVL, hemorrhage (callosum)
24	—	PVL	PVL

Balloon Atrial Septostomy Is Associated With Preoperative Stroke in Neonates With Transposition of the Great Arteries

Patrick S. McQuillen, MD; Shannon E.G. Hamrick, MD; Marta J. Perez, BA; A. James Barkovich, MD; David V. Glidden, PhD; Tom R. Karl, MD; David Teitel, MD; Steven P. Miller, MD

Background—Preoperative brain injury is common in neonates with transposition of the great arteries (TGA). The objective of this study is to determine risk factors for preoperative brain injury in neonates with TGA.

Methods and Results—Twenty-nine term neonates with TGA were studied with MRI before cardiac surgery in a prospective cohort study. Twelve patients (41%) had brain injury on preoperative MRI, and all injuries were focal or multifocal. None of the patients had birth asphyxia. Nineteen patients (66%) required preoperative balloon atrial septostomy (BAS). All patients with brain injury had BAS (12 of 19; risk difference, 63%; 95% confidence interval, 41 to 85; $P=0.001$). As expected on the basis of the need for BAS, these neonates had lower systemic arterial hemoglobin saturation (Sa_{O_2}) ($P=0.05$). The risk of injury was not modified by the cannulation site for septostomy (umbilical versus femoral, $P=0.8$) or by the presence of a central venous catheter ($P=0.4$).

Conclusions—BAS is a major identifiable risk factor for preoperative focal brain injury in neonates with TGA. Imaging characteristics of identified brain injuries were consistent with embolism; however, the mechanism is more complex than site of vascular access for BAS or exposure to central venous catheters. These findings have implications for the indications for BAS, timing of surgical repair, and use of anticoagulation in TGA. (*Circulation*. **2006;113:280-285.**)

Preoperative Brain Injury in Transposition of the Great Arteries Is Associated With Oxygenation and Time to Surgery, Not Balloon Atrial Septostomy

Christopher J. Petit, MD; Jonathan J. Rome, MD; Gil Wernovsky, MD; Stefanie E. Mason, BS; David M. Shera, ScD; Susan C. Nicolson, MD; Lisa M. Montenegro, MD; Sarah Tabbutt, MD, PhD; Robert A. Zimmerman, MD; Daniel J. Licht, MD

Background—Preoperative brain injury is an increasingly recognized phenomenon in neonates with complex congenital heart disease. Recently, reports have been published that associate preoperative brain injury in neonates with transposition of the great arteries with the performance of balloon atrial septostomy (BAS), a procedure that improves systemic oxygenation preoperatively. It is unclear whether BAS is the cause of brain injury or is a confounder, because neonates who require BAS are typically more hypoxemic. We sought to determine the relationship between preoperative brain injury in neonates with transposition of the great arteries and the performance of BAS. We hypothesized that brain injury results from hypoxic injury, not from the BAS itself.

Methods and Results—Infants with transposition of the great arteries (n=26) were retrospectively included from a larger cohort of infants with congenital heart disease who underwent preoperative brain MRI as part of 2 separate prospective studies. Data collected included all preoperative pulse oximetry recordings, all values from preoperative arterial blood gas measurements, and BAS procedure data. MRI scans were performed on the day of surgery, before the surgical repair. Of the 26 neonates, 14 underwent BAS. No stroke was seen in the entire cohort, whereas 10 (38%) of 26 patients were found to have hypoxic brain injury in the form of periventricular leukomalacia. Periventricular leukomalacia was not associated with BAS; however, neonates with periventricular leukomalacia had lower preoperative oxygenation ($P=0.026$) and a longer time to surgery ($P=0.028$) than those without periventricular leukomalacia.

Conclusions—Preoperative brain injury in neonates with transposition of the great arteries is associated with hypoxemia and longer time to surgery. We found no association between BAS and brain injury. (*Circulation*. 2009;119:709-716.)

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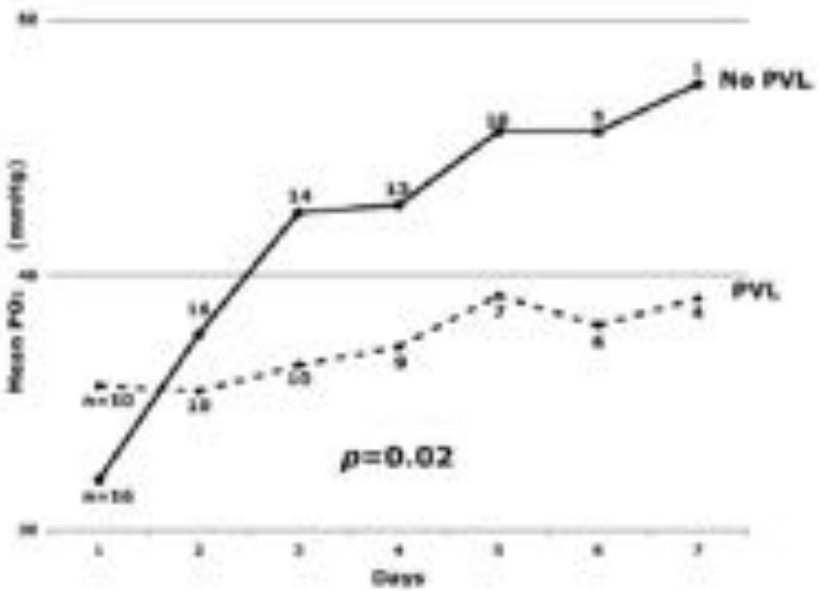

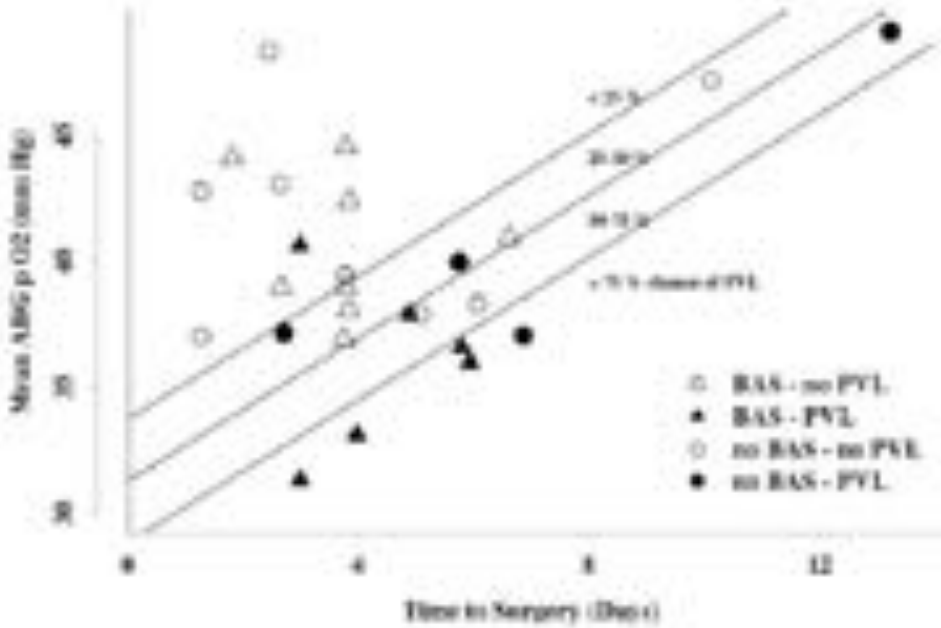

Le vol diastolique

par la collatéralité aorto-pulmonaire, par le CA

-> jusqu'à 30% du Qs

- à partir des vaisseaux des tête et cou-> choréoathétose

Wong, Circulation 1992

- collatérales ralentissent le refroidissement cérébral

Kirshbom, Circulation 1995

L'embolie gazeuse

Johnson, Perfusion 2003

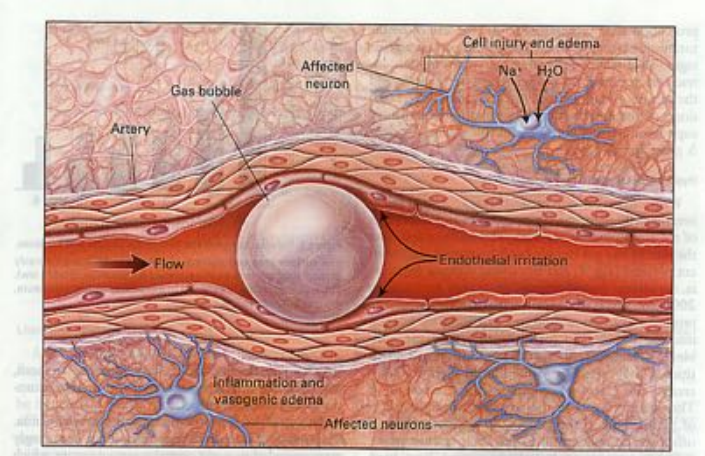

Figure 2. Bubble Obstructing End-Arterial Flow in a Cerebral Vessel with a Diameter of 30 to 60 μm, Causing Distal Ischemia. The obstruction causes the metabolic processes of neurons to fail. Sodium and water enter the vessel, and cytotoxic edema develops. The surface of the bubble generates a foreign-body response through cellular and humoral immune mechanisms. The bubble also mechanically irritates the arterial endothelium. Both processes result in vasogenic edema and greater impairment of perfusion. The neuronal injury extends beyond the area of obstruction.

- veineuse ± paradoxale, artérielle
- gravité : >0,3ml/kg/min
- sources : KTC, ventilation avec fistule pulm
  canulation, coeur battant avec shunt, redux
  désamorçage, cavitation
- ischémie distale et irritation endothéliale ⇒ hypoxie et œdème vasogénique
- Diagnostic peropératoire : variations brutales de l'EEG, bruit caractéristique au Doppler
  ⇒ dénitrogénation, KT aspiratif intraVD
  ⇒ perfusion cérébrale retrograde en séances de 5-15min, 20-25mmHg
  ⇒ oxygénothérapie hyperbare < H6 : ↓volume bulle, ↓œdème
  ⇒ traitement de HTIC
- Diagnostic postopératoire : déficit focal ou arythmie au réveil, au changement de position
  ⇒ oxygénothérapie hyperbare même > 48h, sinon traitement HTIC

Surveillance de l'atteinte neurologique postopératoire
Marqueurs biologiques

EEG

13% convulsions électriques, sans valeur prédictive

Gaynor, JTCVS 2006

- ondes lentes à J2-J5 chez l'enfant, sans valeur prédictive
- silences > 50'' chez le NN prédictif pour séquelles lourdes
- ondes lentes chez le NN si hyponatrémie < 130mmol/L

Schmitt, Pediatr Res 2005

Biologiques: NSE (*Schmitt 1998*), prot S-100 (*Lindberg 1998)*

IRM

Mahle, Circulation 2002

n = 24
dont 8 HLHS

	préop		< 2 sem	4-6 mois
Pt	Pre-op MRS	Pre-op MRI	Early Post-op MRI	Late Post-op MRI
1	Lactate	Infarct (parietal)	PVL	NA
2	NA	—	PVL	—
3	—	—	—	—
4	NA	—	NA	NA
5	Lactate	Infarct (caudate)	Infarct (caudate), hemorrhage (parietal)	—
6	NA	—	Infarct (parietal-occipital)	—
7	Lactate	—	NA	NA
8	—	—	—	—
9	—	—	NA	NA
10	—	—	PVL	—
11	Lactate	—	PVL, infarct (parietal-occipital)	Old infarct, atrophy
12	NA	—	PVL	Acute infarct (parietal)
13	Lactate	—	—	NA
14	Lactate	PVL	PVL; infarct (frontal/parietal); hemorrhage (frontal)	—
15	—	—	Hemorrhage (frontal)	—
16	Lactate	Hemorrhage (temporal)	Infarct (parietal); hemorrhage (parietal)	Atrophy
17	—	—	PVL	—
18	Lactate	—	PVL; hemorrhage (frontal)	—
19	Lactate	PVL	—	—
20	Lactate	PVL	PVL; hemorrhage (thalamus)	NA
21	—	—	—	NA
22	NA	—	—	—
23	—	—	PVL, hemorrhage (callosum)	—
24	—	PVL	PVL	—

Prévention / traitement …

Diagnostic antenatal - prévient les complications neuro périopératoires (OR=4) dans l ’ hypoVG *Mahle, Pediatrics 2001*

Prévention de l’ hypoxémie, acidose, bas débits

Monitoring pré per & post opératoire

Diagnostic rapide des bas débits post CEC, contrôle de la glycémie

Notre attitude

- *Examen clinique préopératoire, imagerie dans le cadre syndromique, des souffrances néonatales*
- *Monitoring peropératoire : NIRS, BIS*
- *Filtre sur la ligne artérielle*
- *ultrafiltration*
- *Refroidissement progressif (20-30') sous contrôle de la température naso-pharyngée et rectale*
- *Hypothermie 20-22°C et perfusion cérébrale séléctive (15-20ml/kg/min à 30-45mmHg), d'une durée < 45min*
- *refroidissement: pHstat -> rechauffement : α stat*
- *recirculation froide 10min puis réchauffement progressif < 0,5-1°/min, Δt°C < 10, surveillance de la Gly*

Notre attitude

- *Purge soigneuse des cavités cardiaques avant déclampage*
- *EEG systématique pour les patients opérés en hypothermie*
- *Doppler transcrânien quotidien des patients sous ECMO*
- *Midazolam pour la sédation*
- *Examen clinique postopératoire quotidien et formation des infirmières à la surveillance neurologique*
- *IRM précoce en cas d'anomalie clinique (dès le retrait des électrodes)*