Particularités du circuit CEC en pédiatrie (miniaturisation)

Hematocrit idéal et transfusion

Température et débit de CEC

Pression de perfusion

Hypothermie et protection cérébrale

Monitoring

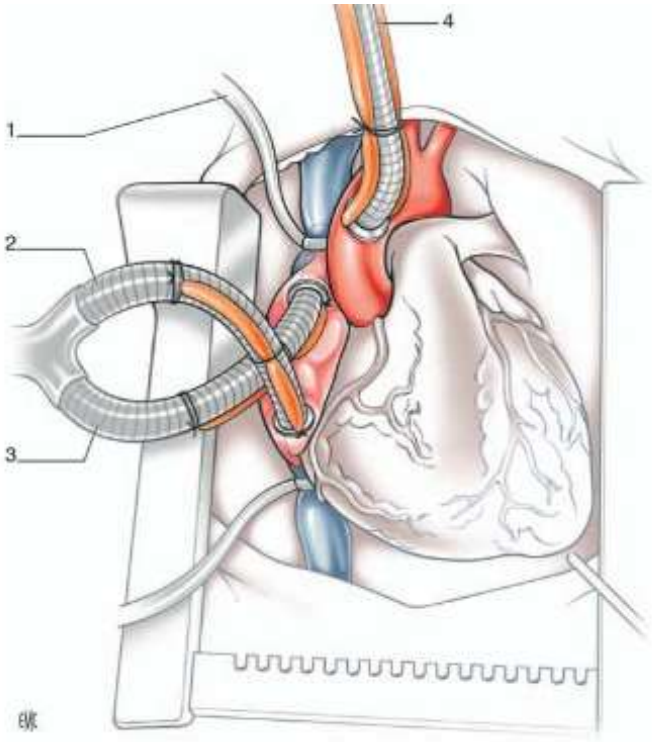
Inflammation

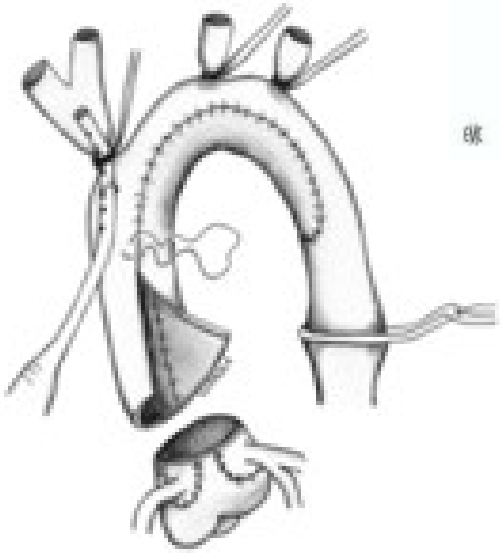
Cardioplégie

Sevrage de la CEC en pédiatrie

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



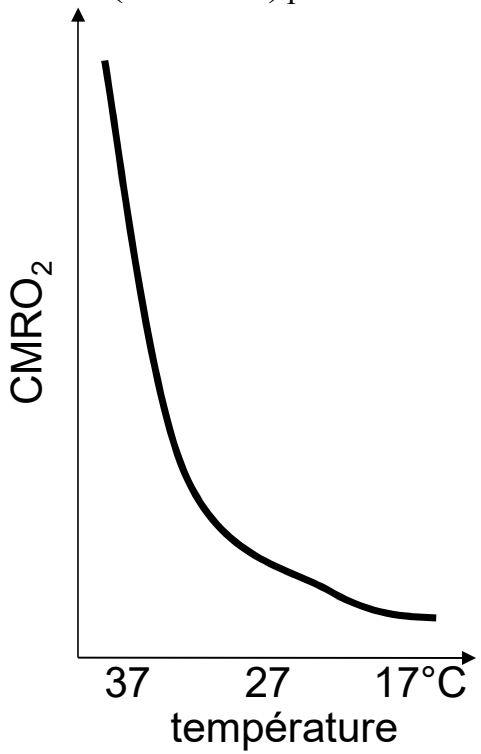


Température de la CEC

Normothermie > 36°C
Hypothermie légère 30-36°C
Hypothermie modérée 22-30°C
Hypothermie profonde 17-22°C

L’utilisation de l’hypothermie en CEC pédiatrique

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



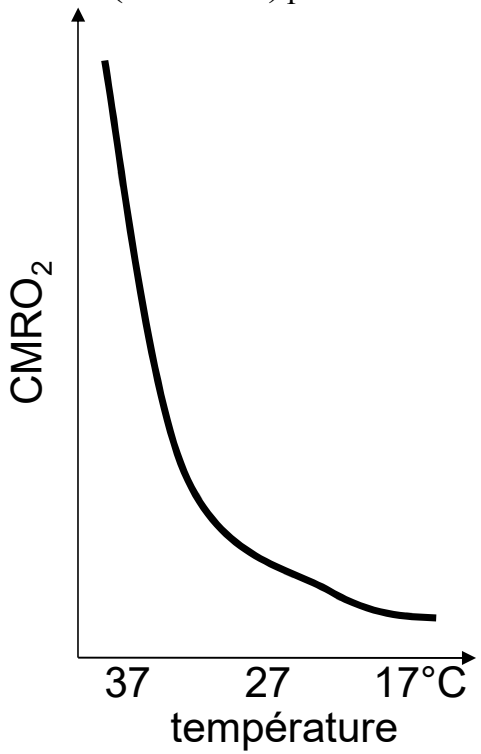
Q_{10} (index thermique) = % de variation du paramètre lorsque la température varie de 10°C

$Q_{10} = 3.65$

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).

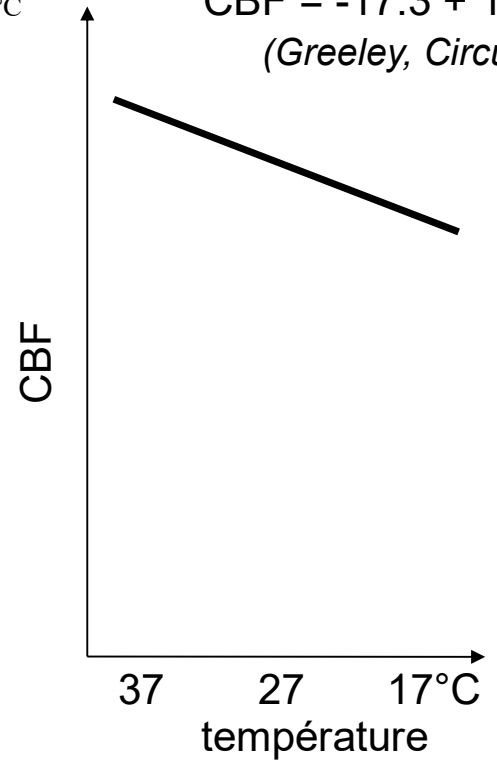
L’utilisation de l’hypothermie en CEC pédiatrique

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



CBF = -17.3 + 1.47 x temp

(Greeley, Circulation 1989)



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.

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

Hypothermie en CEC pédiatrique: inconvénients

- perte de l'autorégulation, DSC ~ PAM < 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

Techniques de perfusion : DHCA vs low-flow

Boston Circulatory Arrest Study 1988-1992

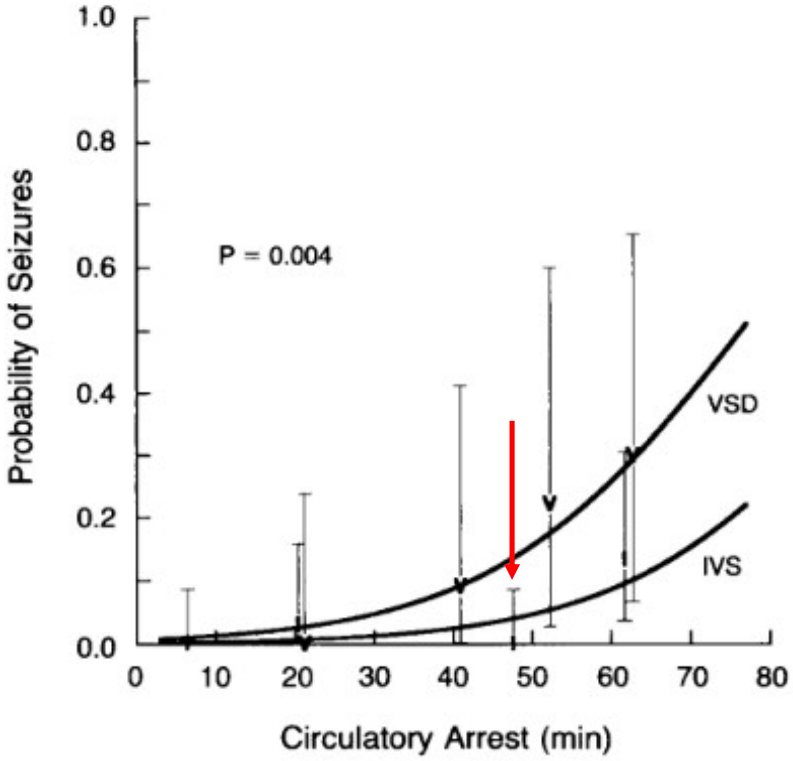
(Newburger NEJM 1993, Bellinger, NEJM 1995, Bellinger, Circulation 1999, Bellinger, JTCVS 2003)

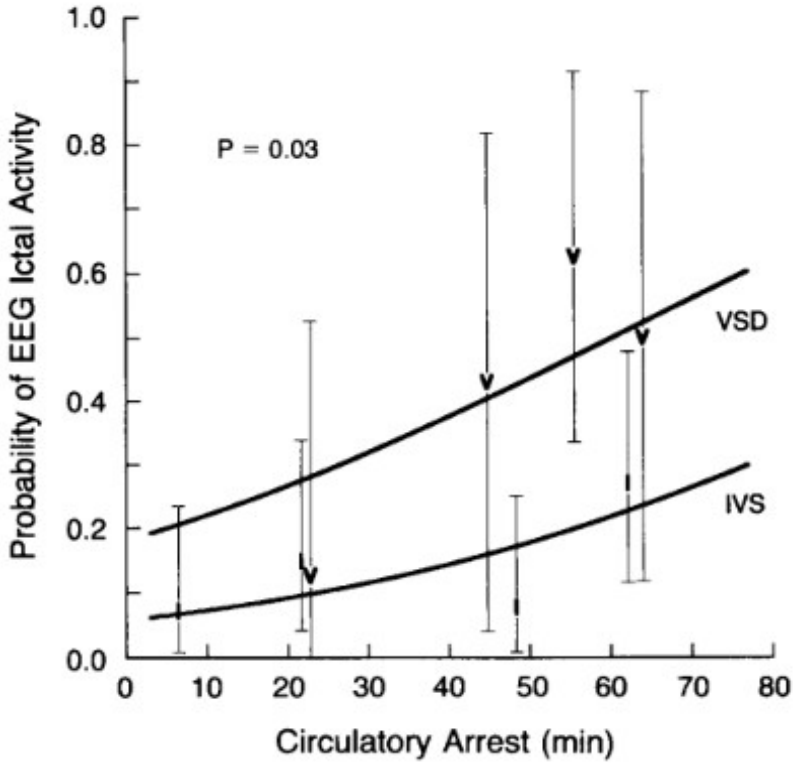
171 nouveau-nés avec TGV, 87 DHCA + 84 low-flow, 18°C, α-stat, Ht>20%

-> 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 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.

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



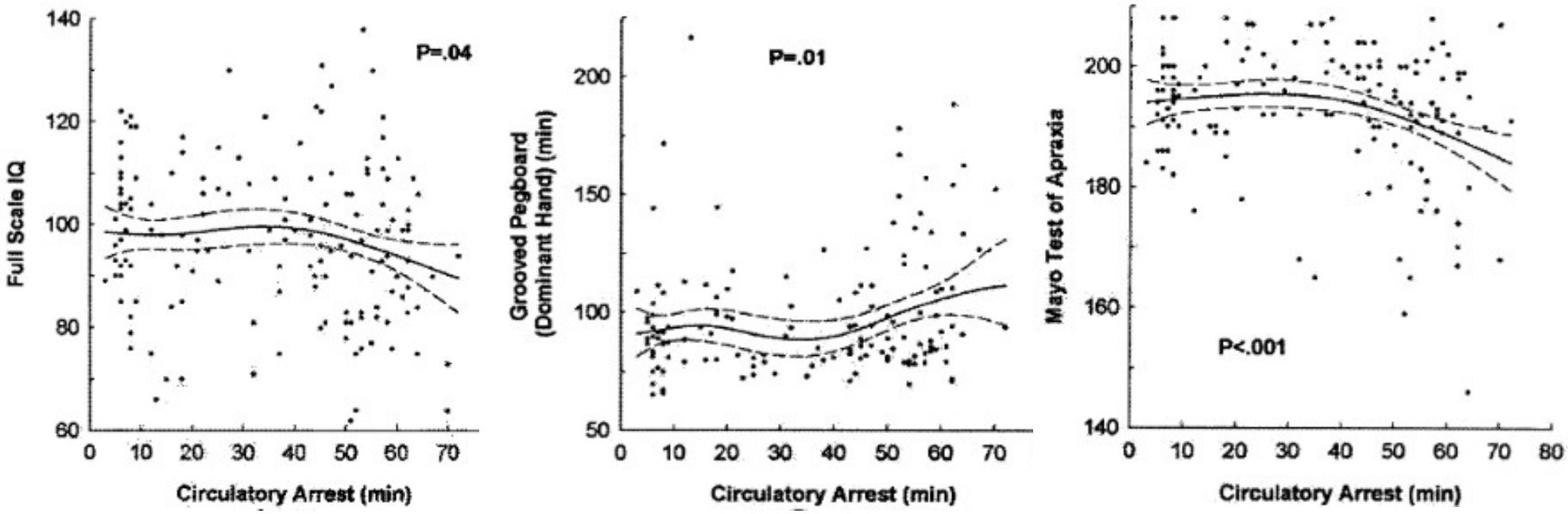


(Newburger, NEJM 1993)

Boston Circulatory Arrest Study

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

tests psychomoteurs à 8 ans



La limite de l'IC 95% pour le DHCA était à 32min

(Wypij, JTCVS 2003)

The Society of Thoracic Surgeons, The Society of Cardiovascular Anesthesiologists, and The American Society of ExtraCorporeal Technology: Clinical Practice Guidelines for Cardiopulmonary Bypass—Temperature Management during Cardiopulmonary Bypass

Engelman JECT 2015

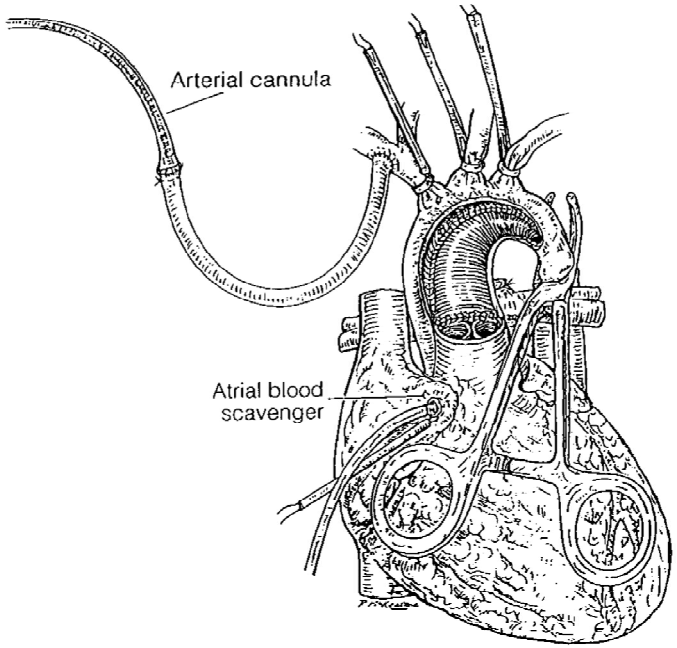
CLASS I RECOMMENDATIONS

1. **The oxygenator arterial outlet blood temperature is recommended to be used as a surrogate for cerebral temperature measurement during cardiopulmonary bypass (CPB). (Class I, Level C)**
2. **To monitor cerebral perfusate temperature during warming, it should be assumed that the oxygenator arterial outlet blood temperature underestimates cerebral perfusate temperature. (Class I, Level C)**
3. **Surgical teams should limit arterial outlet blood temperature to <37°C to avoid cerebral hyperthermia. (Class I, Level C)**
4. **Temperature gradients between the arterial outlet and venous inflow on the oxygenator during CPB cooling should not exceed 10°C to avoid generation of gaseous emboli. (Class I, Level C)**
5. **Temperature gradients between the arterial outlet and venous inflow on the oxygenator during CPB rewarming should not exceed 10°C to avoid outgassing when blood is returned to the patient. (Class I, Level C)**

CLASS IIA RECOMMENDATIONS

1. **Pulmonary artery (PA) or nasopharyngeal temperature recording is reasonable for weaning and immediate post-bypass temperature measurement. (Class IIa, Level C)**
2. **Rewarming when arterial blood outlet temperature ≥30°C:**
   i. **To achieve the desired temperature for separation from bypass, it is reasonable to maintain a temperature gradient between arterial outlet temperature and the venous inflow of ≤4°C. (Class IIa, Level B)**
   ii. **To achieve the desired temperature for separation from bypass, it is reasonable to maintain a rewarming rate ≤ .5°C/min. (Class IIa, Level B)**
3. **Rewarming when arterial blood outlet temperature <30°C: to achieve the desired temperature for separation from bypass, it is reasonable to maintain a maximal gradient of 10°C between arterial outlet temperature and venous inflow. (Class IIa, Level C)**

Qu'en est-il de la perfusion cérébrale sélective ?



Asou, ATS 1996

- Avec / sans tube en Goretex suturé au TABC
- Monitoring de la pression radiale/brachiale droite
- Avec / sans perfusion Ao descendante à travers le CA (motivation pour choisir une température entre 18 – 32°C)
- En sachant que tout le débit n'est pas délivré au cerveau ….

Quel débit ?

Asou, ATS 1996 : 50ml/kg min

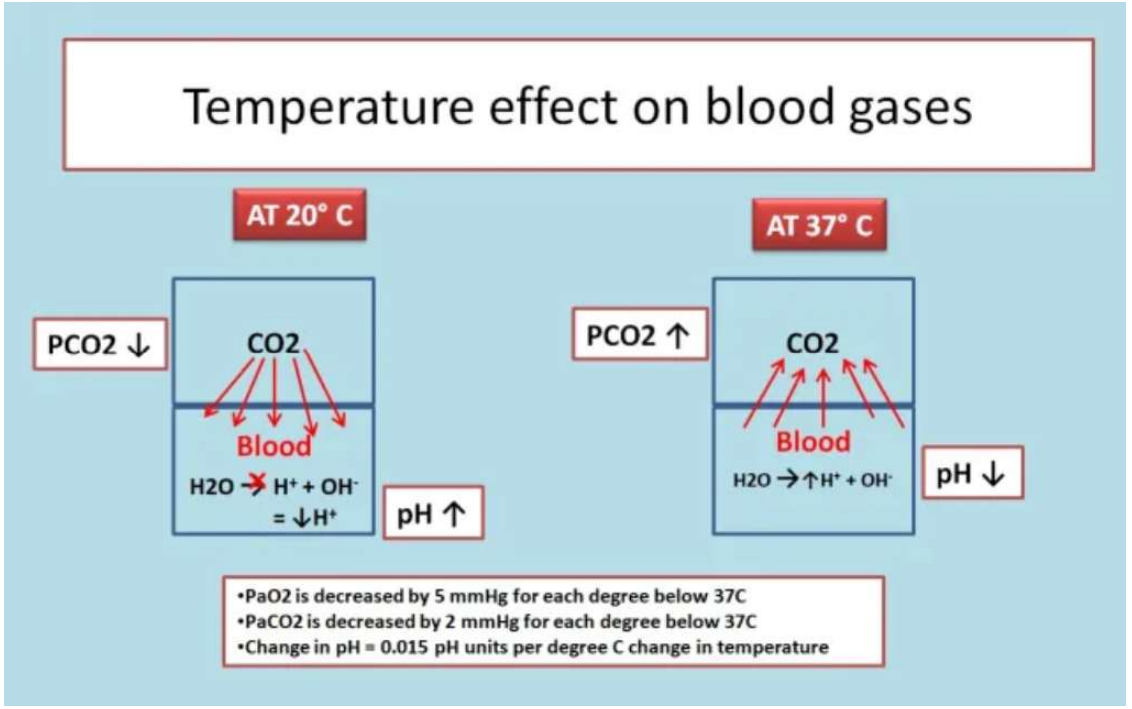
Pigula, JTCVS 2000 : collatéralité responsable d'un reflux dans l'aorte descendante ….

Andropoulos, JTCVS 2003 : 24 – 94 ml/kg min !!! en utilisant un monitoring multimodal (rSO_2 et DTC)

Quelle pression ?

Tanaka, JTCVS 1998 : pas moins de 40 mmHg

Solubilité CO_2 ~ 1/ température

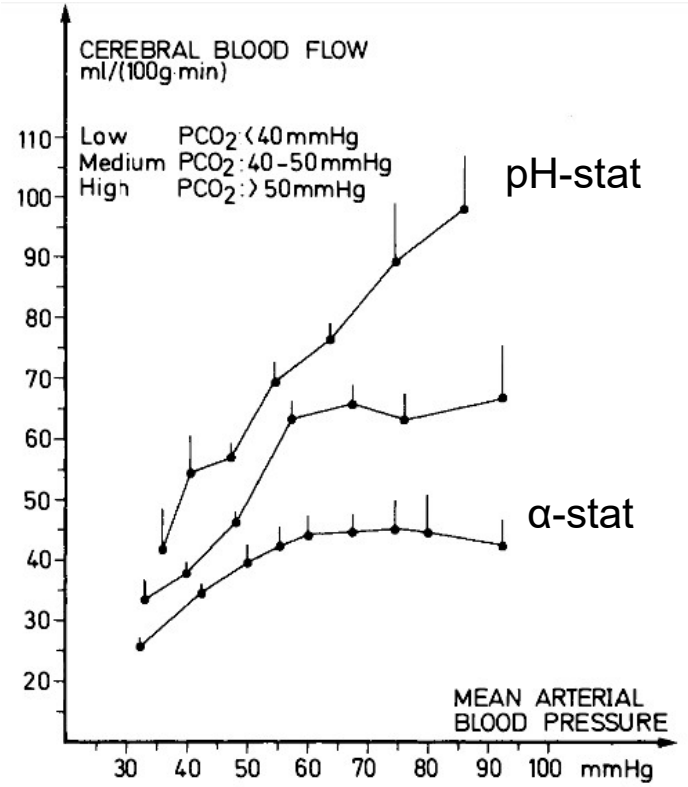


α-stat:
pCO2 = const
pH = var

-> vasoconstriction cérébrale, meilleur maintien de l'autorégulation ?

pH-stat:
pCO2 = var
pH = const

-> vasodilatation cérébrale, garant d'un refroidissement homogène et rapide du cerveau



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

Particularités du cerveau néonatal

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

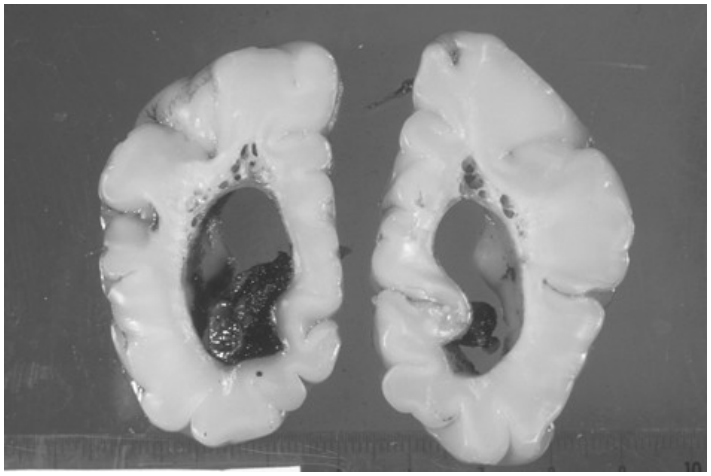
- débit sanguin = 2-3 x celui de l'adulte

- utilise exclusivement du glucose

- Myélinisation en cours par des olygodendrocytes – matière blanche périventriculaire très susceptible à l'hypoxémie -> risque de leucomalacie périventriculaire (LPV)

Gaynor, Semin Thorac Cardiovasc Surg 2004)

La leucomalacie périventriculaire (LPV)



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

- ~ 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

Valeur prédictive de la LPV ?

Mahle, Circulation 2002

n = 24NN
dont 8 HLHS

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	—

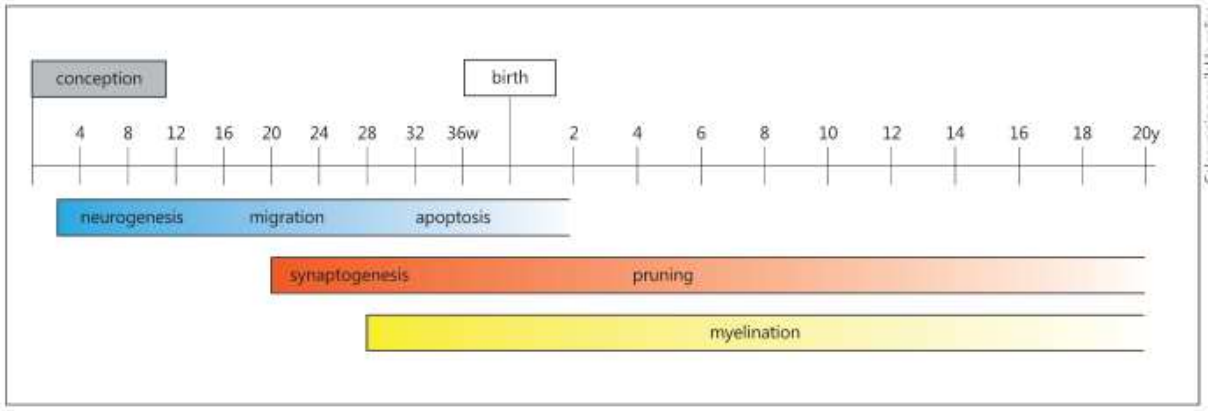
The NEW ENGLAND JOURNAL of MEDICINE

Perspective

MARCH 9, 2017

Anesthesia and Developing Brains — Implications of the FDA Warning

Dean B. Andropoulos, M.D., M.H.C.M., and Michael F. Greene, M.D.



L’embolie cérébrale gazeuse, particulaire

O’Brien, 1997 : 2-2664 embols / patient détectés au Doppler carotidien

risque élevé (shunt D-G, TGV) :152/patient