

La CEC pédiatrique

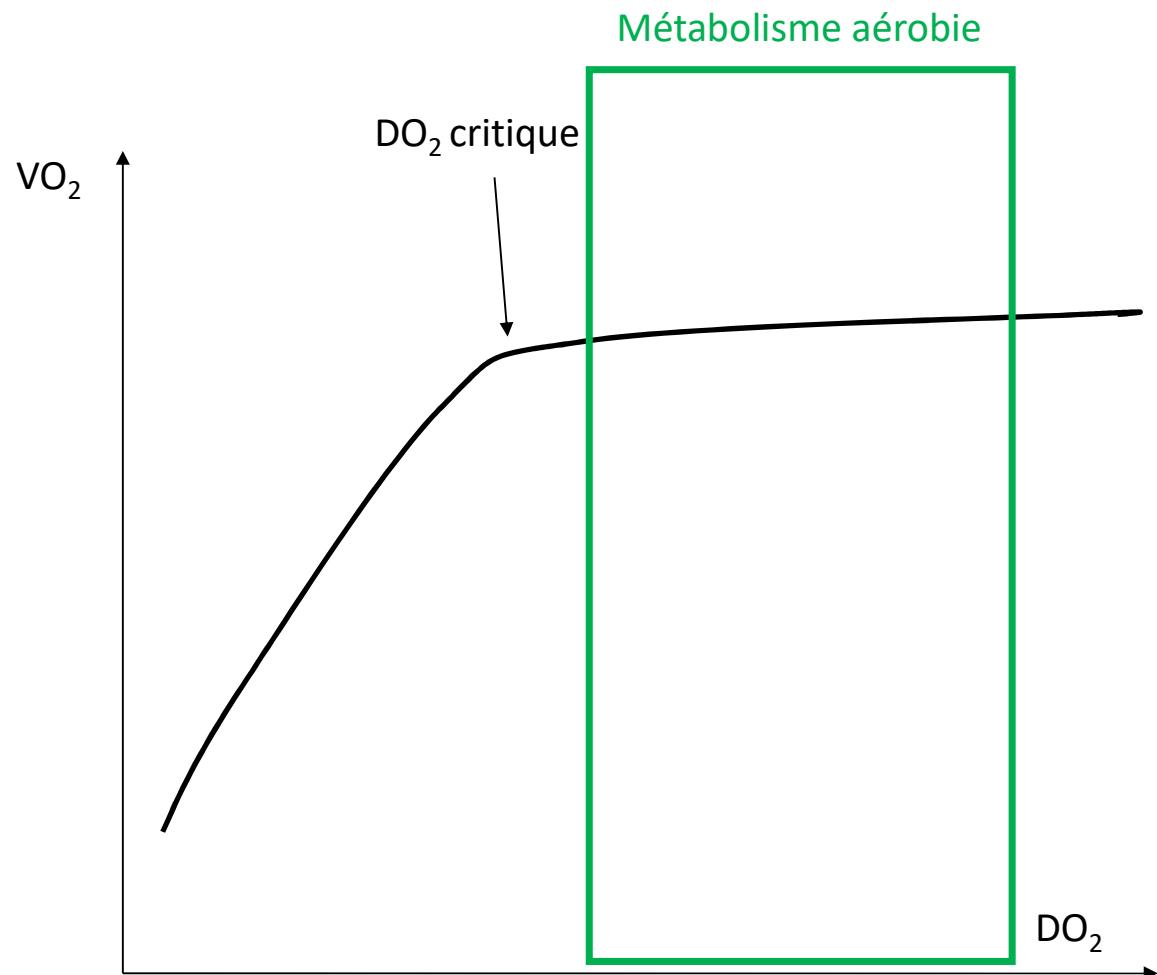
Mirela Bojan, MD. PhD.
Hôpital Marie Lannelongue

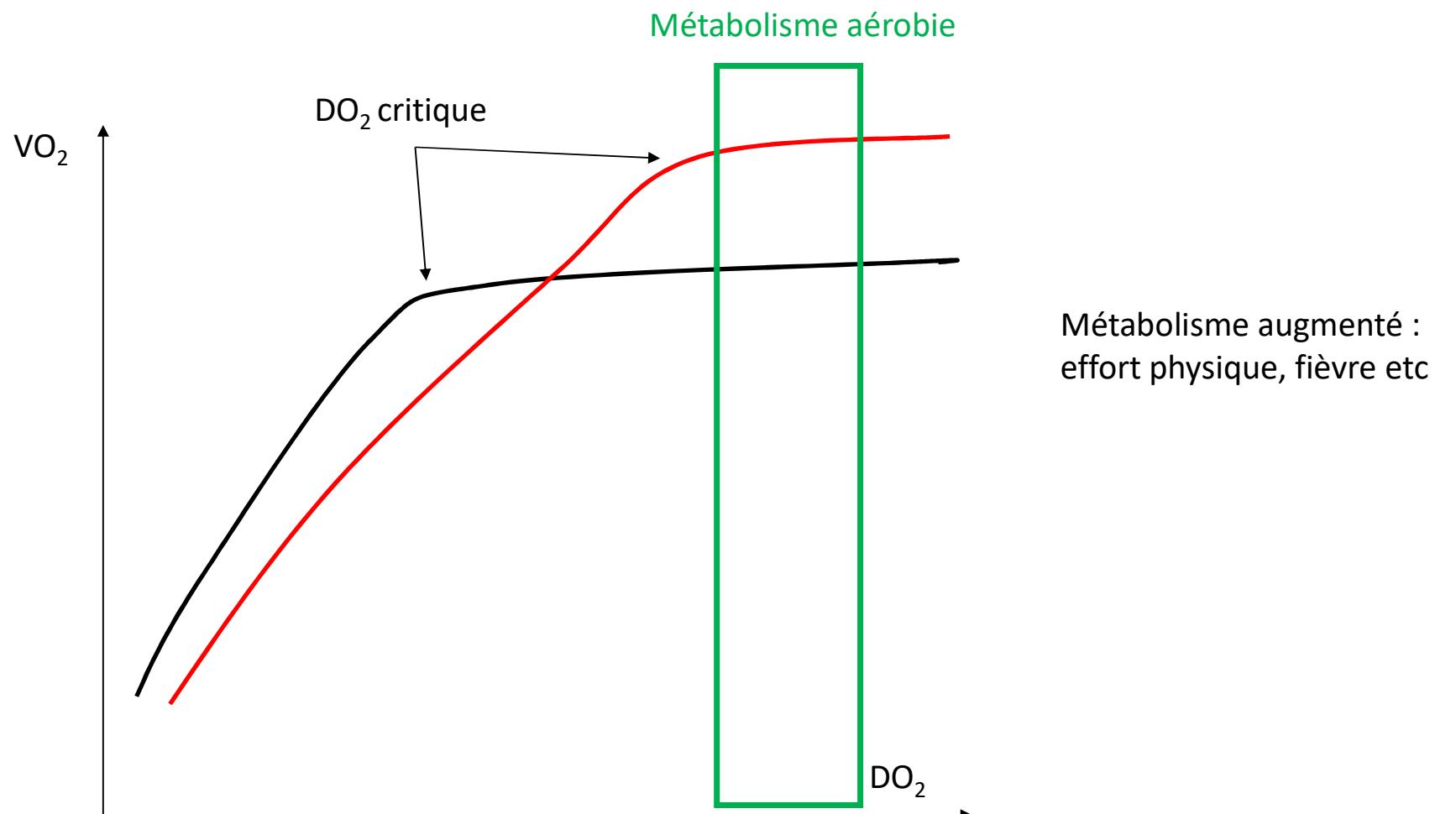
m.bojan@ghpsj.fr

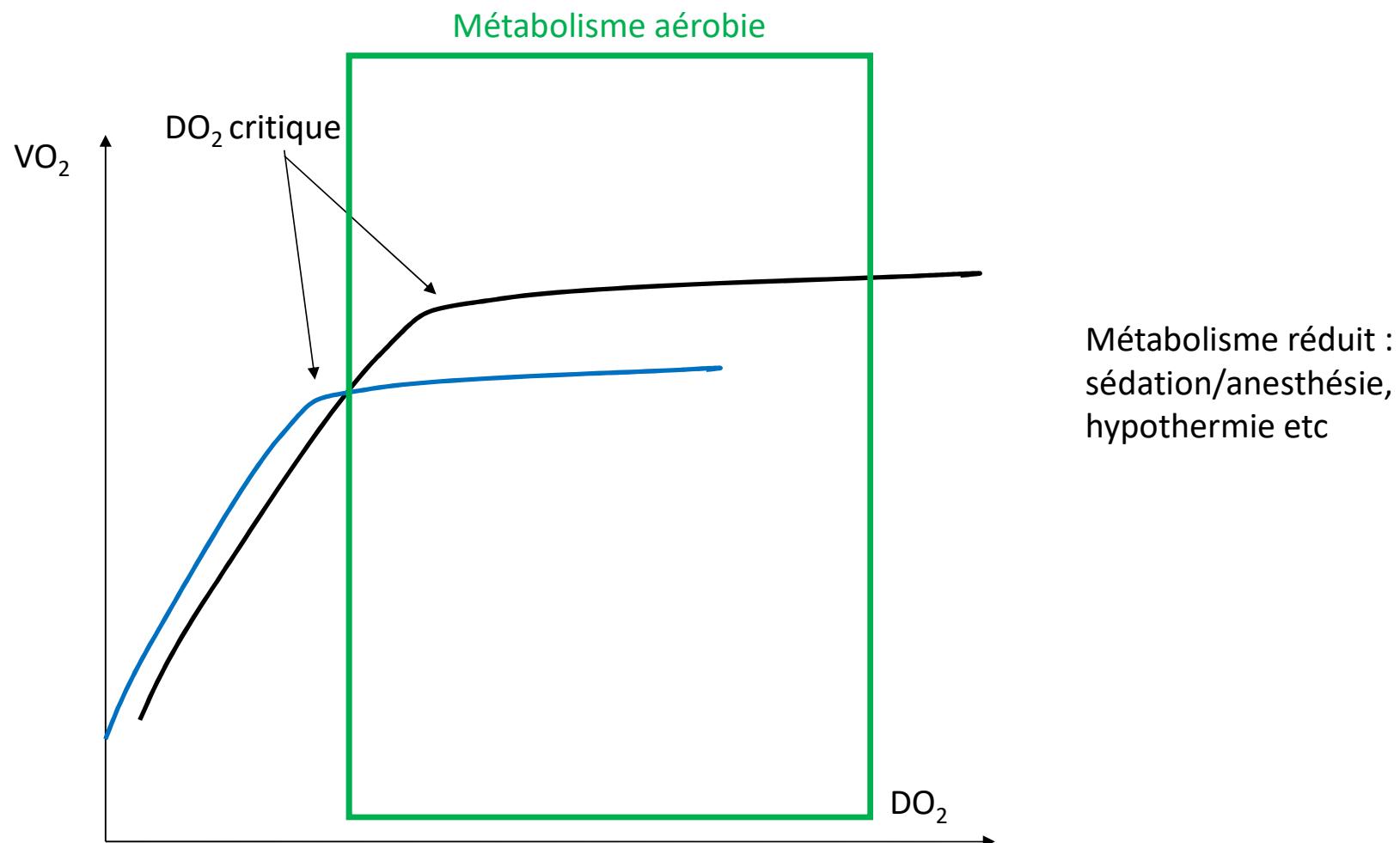


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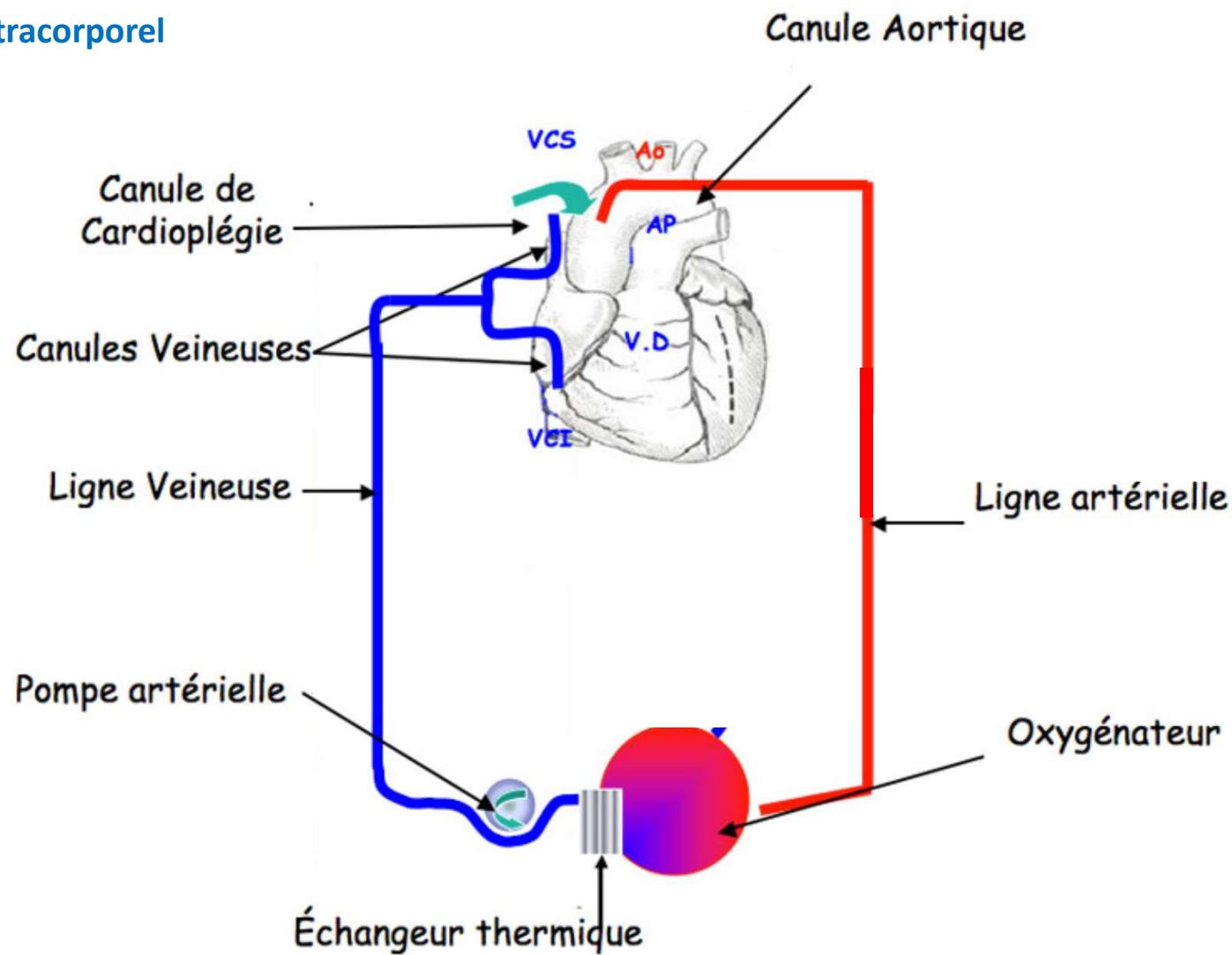




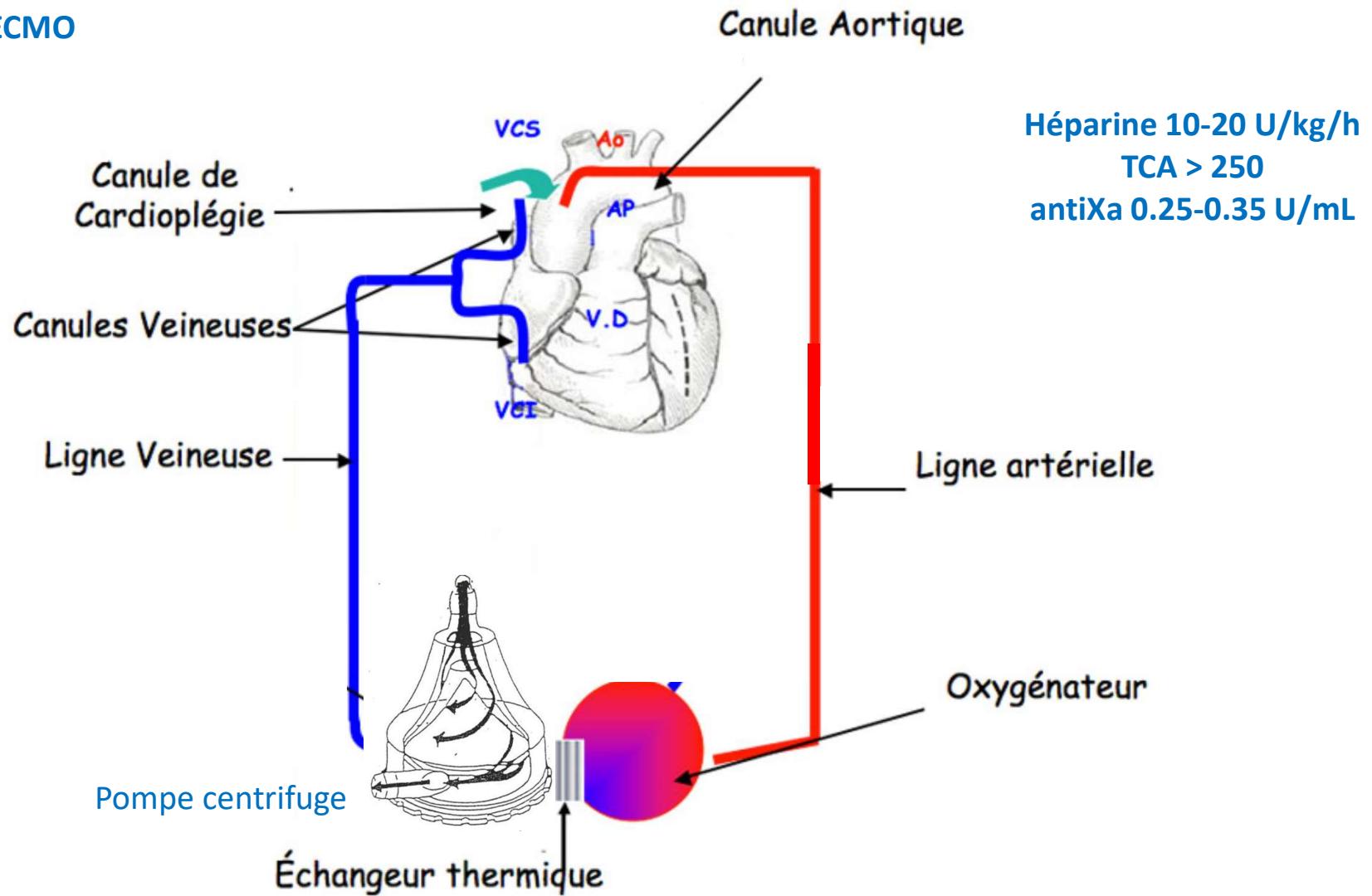
$\text{DO}_2 \text{ (mL min}^{-1} \text{ m}^{-2}\text{)} = 10 * \text{pump flow indexed (L min}^{-1} \text{ m}^{-2}\text{)} \times \text{arterial O}_2 \text{ content (mL 100 mL}^{-1}\text{)}$

$\text{Arterial O}_2 \text{ content (mL 100 mL}^{-1}\text{)} = \text{Haemoglobin (g dL}^{-1}\text{)} * 1.34 \text{ (mL O}_2 \text{ g}^{-1} \text{ of Haemoglobin)} \times \text{SaO}_2 (\%)$
+ 0.003 * $\text{PO}_2 \text{ (mmHg)}$

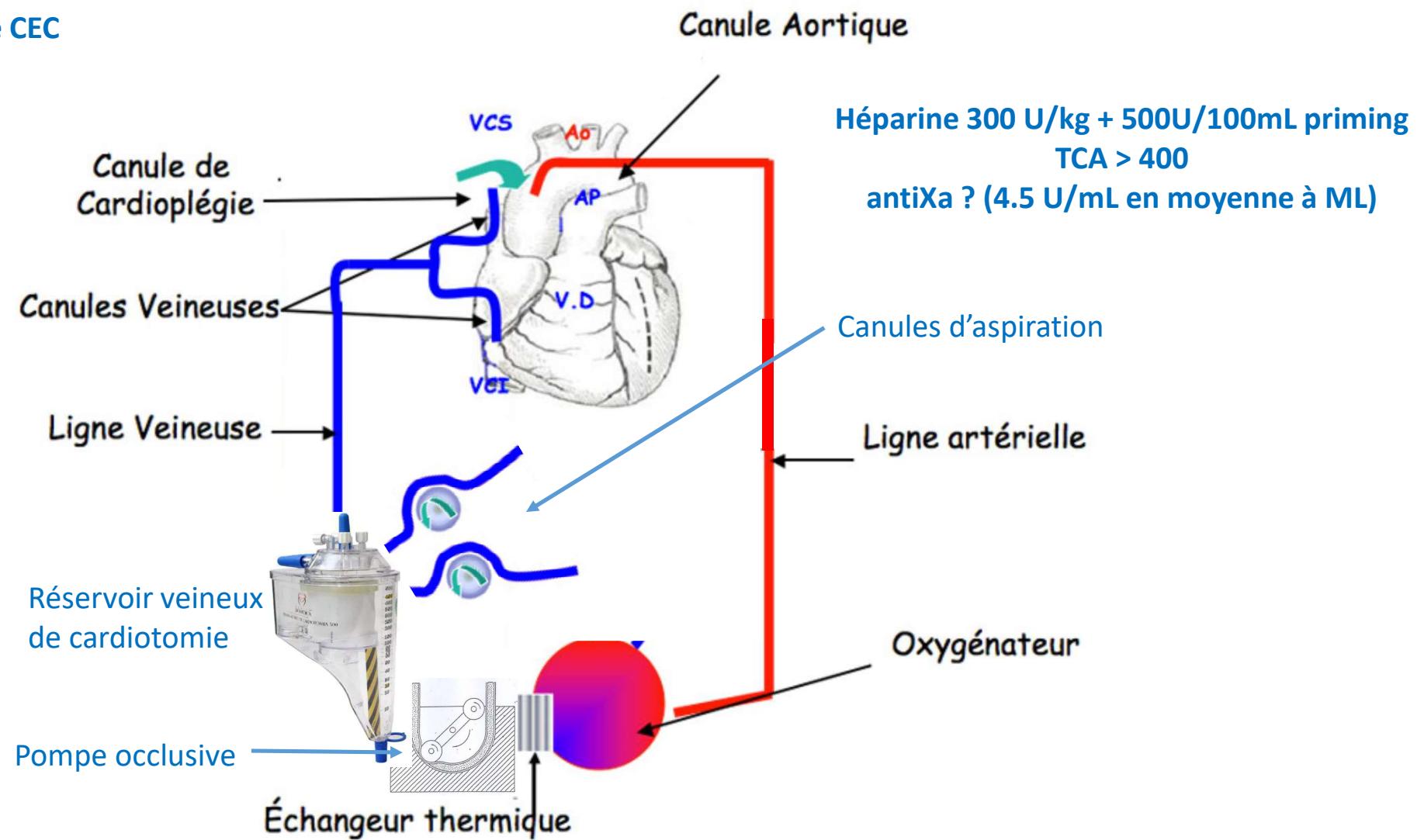
Le circuit extracorporel



Le circuit d'ECMO



Le circuit de CEC



Pompes de CEC

Occlusive (à galets)



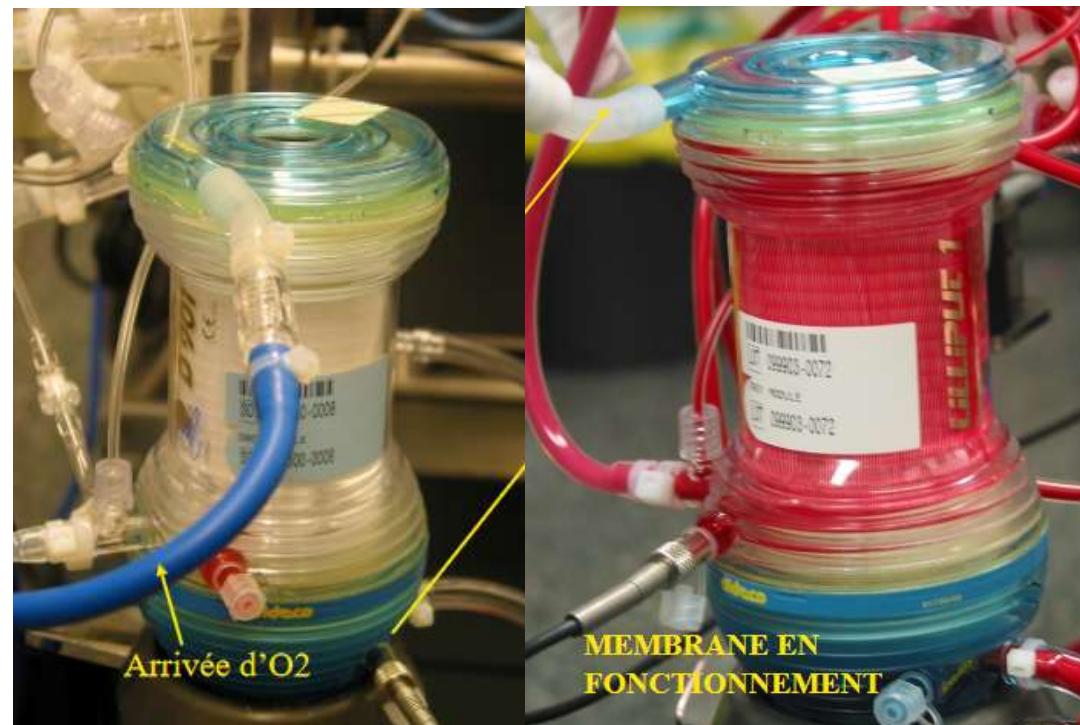
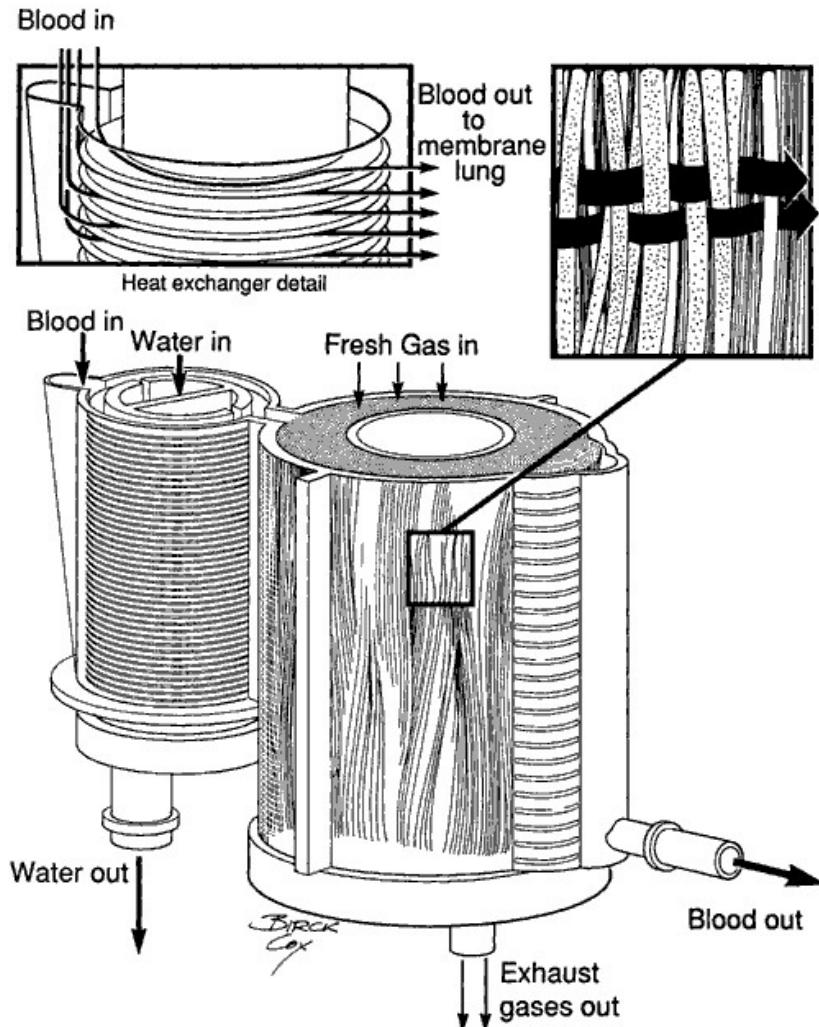
Centrifuge
Effet vortex



Occlusive	Centrifuge
Précision du débit (délivré même en présence d'une résistance au flux)	Le débit dépend de la postcharge -> nécessite un système de monitorage du débit
Risque de générer des fortes pressions (positives ou négatives) - risque accru d'hémolyse	Risque de retro-flow
Risque plus élevé d'embolie gazeuse	Moindre risque d'embolie gazeuse
Circuit (réservoir) ouvert – contact sang/air	Circuit fermé (réservoir souple)
Faible coût	Coût élevé

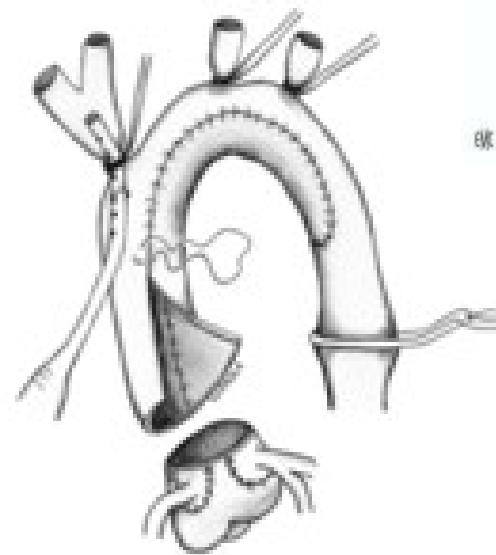
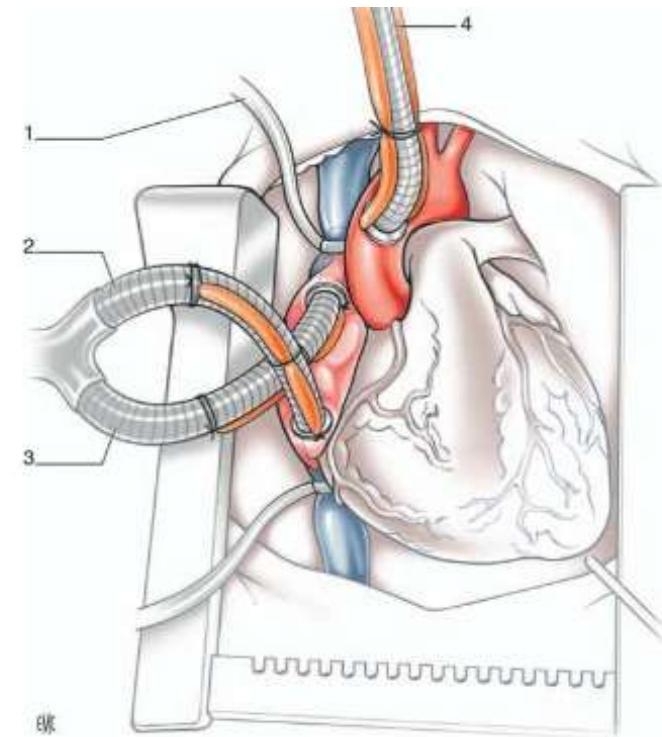
le réservoir :





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



Particularités du circuit en CEC pédiatrique (néonatale)



Hematocrite idéal et transfusion

Température et débit de CEC

Pression de perfusion

Hypothermie et protection cérébrale

Monitoring

Inflammation

Cardioplégie

Sevrage de la CEC en pédiatrie

La principale limitation dans l'effort de miniaturisation du circuit néonatal est la taille de l'oxygénateur



Kids pédiatrique
87 ml prime, 0.61 m²
Q max = 2.5 L/min

Ajout filtre artériel = 28 ml

Kids néonatal
31 ml prime, 0.22 m²
Qmax = 0.7 L/min

Ajout filtre artériel = 16 ml



Quadrox neonatal
Filtre intégré
38 ml prime, 0.38 m²
Qmax = 1.5 L/min

Affinity Pixie
48 ml prime, 0.48 m²
Q max = 2 L/min

CAPIOX® RX
Family of Hollow Fiber Oxygenators

Perfectly balanced performance for every patient

A collection of various oxygenator models from different manufacturers, including CAPIOX, MAQUET, and TERUMO, are displayed against a light background. The models vary in size and design, showing different configurations of filters and connectors.

Name	Brand	Integrated ALF	Maximum Blood Flow	Priming Volume	Surface area	Minimum-level in the reservoir	Biocoating
Quadrox-i	Maquet	yes	1.5 L min ⁻¹	40 mL	0.38 m ²	15 mL	SOFTLINE (polymer coating)
Neonatal	Getinge Group						BIOLINE (heparin coating)
Capiox FX 0.5	Terumo	yes	1.5 L min ⁻¹	43 mL	0.50 m ²	15 mL	Xcoating™ (amphiphilic polymer)
KIDS D100	LivaNova	no	0.7 L min ⁻¹	31 mL	0.22 m ²	10 mL	Phosphorylcholine coating
Affinity Pixie	Medtronic	no	2 L min ⁻¹	48 mL	0.48 m ²	20 mL	CORTIVA™ (heparin coat) BALANCE® (hydrophilic)
HILITE 1000	Medos	no	1 L min ⁻¹	57 mL	0.39 m ²	50 mL	Rheoparin® (heparin coating)

Le tubing

Diamètre tubulure (in.)	Diamètre tubulure (mm)	Volume par mètre de longueur
1/2	12.7	126
3/8	9.53	71
1/4	6.35	33
3/16	4.76	18

Poids patient	Canulation artérielle	Canulation veineuse
< 10 kg	3/16	1/4
10-20 kg	1/4	3/8
20-30 kg	3/8	3/8

Le drainage veineux est passif (déclive 30 cm) ou assisté (max -30 cmH20)

Volume du priming néonatal

Oxygénateur	31
Filtre artériel	16
Réservoir	≥ 30
Ligne artérielle 3/16	27 - 36
Ligne veineuse 1/4	50 - 66
Filtre à UF	20
Circuit de cardioplégie	10
Total	184 - 210 ml

Volémie d'un nouveau-né = $0.85 * \text{poids}$ ($0.9 * \text{poids si prématuré}$)

Particularités du circuit CEC en pédiatrie (miniaturisation)

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Composition du priming néonatal

Variation Ht si priming cristalloïde:

$$\Delta \text{Ht} = (\text{Ht patient} * \text{volémie}) / (\text{volémie} + \text{volume priming})$$

Volume CGR a inclure dans le priming :

$$\text{CGR} = \text{Ht désiré} * (\text{volémie} + \text{volume priming}) - (\text{volémie} * \text{Ht patient}) / 0.6$$

Volémie d'un nouveau-né = 0.85 * poids (0.9 * poids si prématuré)

Volémie estimée = 275 ml

Composition priming :

Albumine 5%

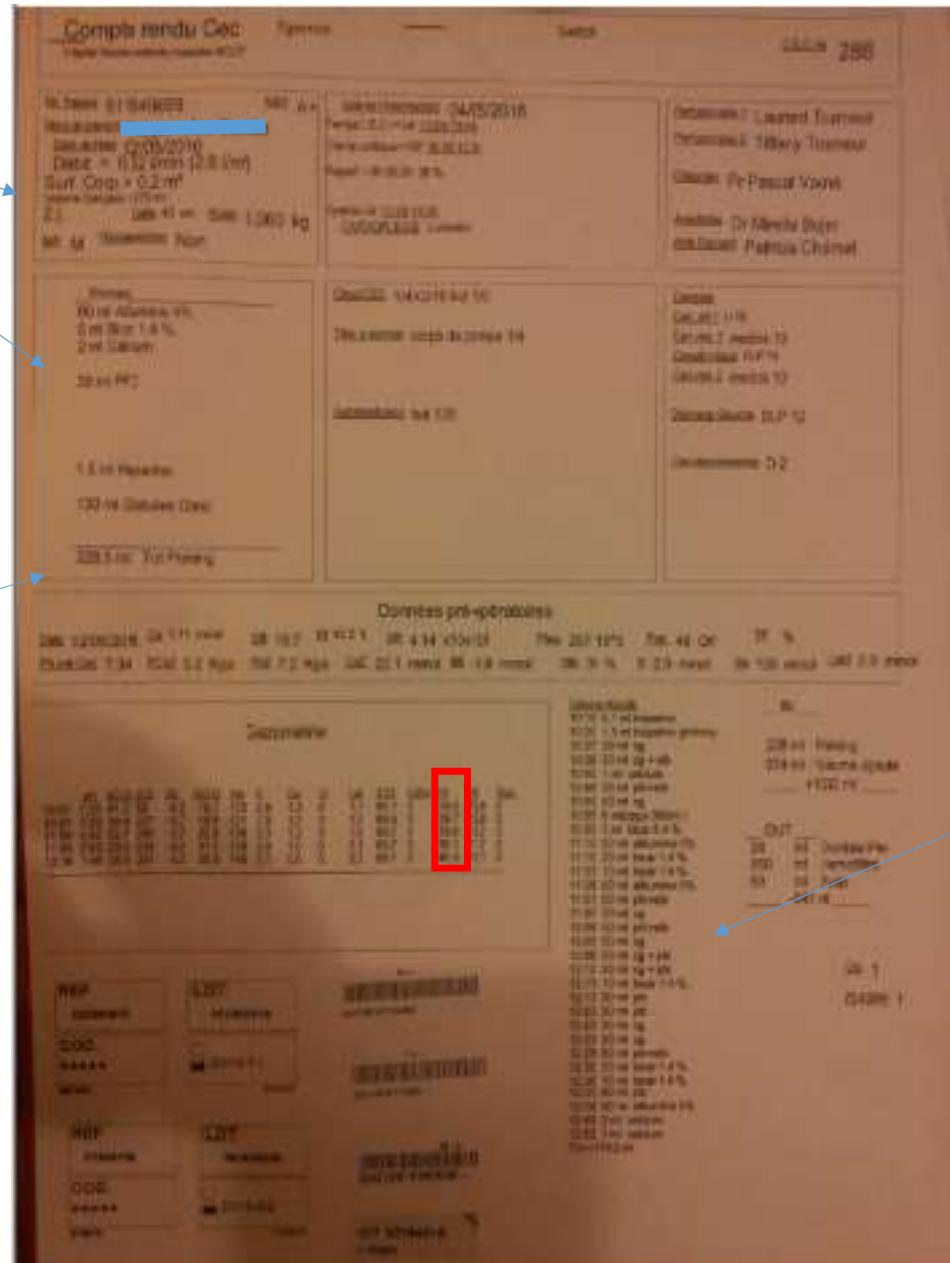
Bicarbonate

Ca

PFC

CGR = 130 ml

Volume priming = 228 ml



Volume ajouté = 974 ml
CGR, PFC, bicarbonate,
albumine

Hématocrite idéal en CEC ?

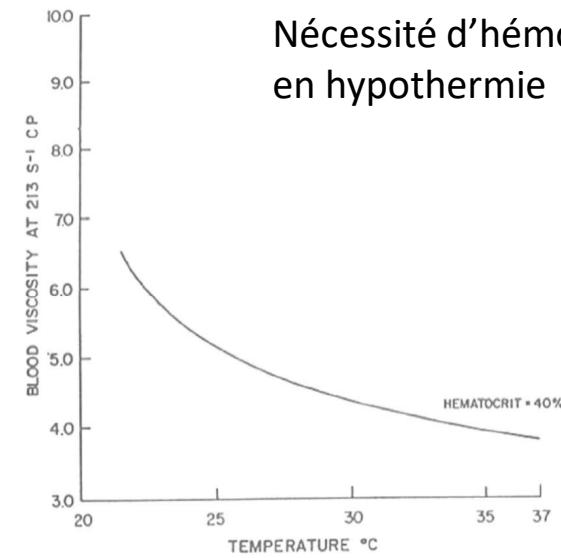
Loi d'Ohm : $Q = (\text{Part} - \text{Pvein}) / \text{Résistance}$

Loi de Poiseuille : Résistance = $8 * \text{viscosité} * \text{Long} / \pi * \text{Rayon}^4$

Viscosité plasma = $\exp [-5.64 + 1800/(T^\circ\text{C} + 273)]/100$

Viscosité sang = Viscosité plasma * $\exp (2.31 * \text{Ht})$

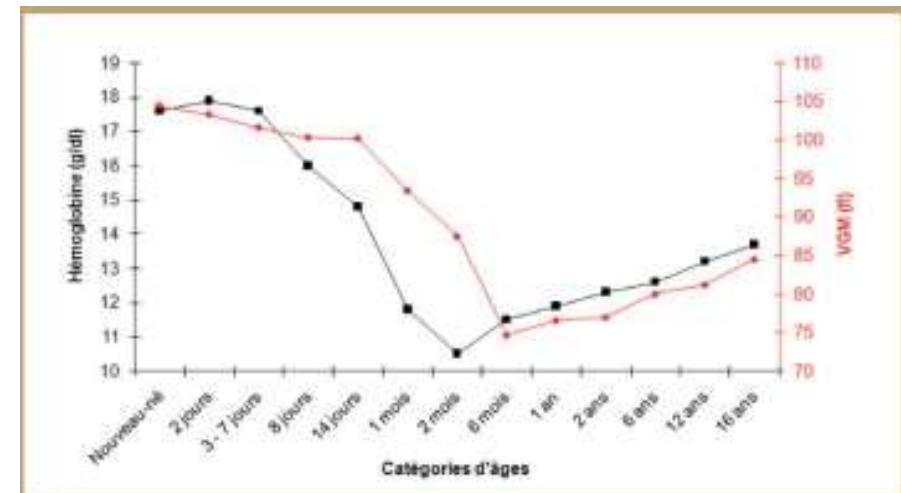
Lainey, 2009



Nécessité d'hémodiluer
en hypothermie

MB1

From Rand PW, Lacombe E, Hunt HE, Austin WH. Viscosity of normal human blood under normothermic and hypothermic conditions. J Appl Physiol 1964;19:117-122, as modified by Gordon RJ, Ravin MB, Daintoff GR. Blood rheology. In Cardiovascular physiology for anaesthesiologists. Springfield, Illinois: Charles C Thomas, 1979:27-71



Diapositive 21

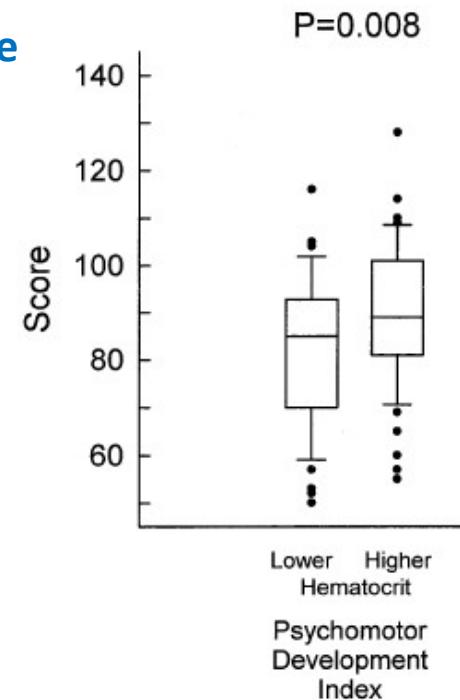
MB1

Mirela Bojan; 07/11/2025

Hématocrite idéal en CEC néonatale

147 nourrissons, 74 Ht=20%, 73 Ht=30%
Low-flow, 18°C, pH-stat
Tests psychométriques à 1 an

(Jonas, JTCVS 2003)



124 nourrissons, 56 Ht=25%, 68 Ht=35%
Low-flow, pH-stat
Tests psychométriques et IRM à 1 an
-> aucune différence

(Newburger, JTCVS 2008)

Ne pas descendre en-dessous de 30%, même en hypothermie

[Interact Cardiovasc Thorac Surg](#). 2013 Jun;16(6):890-1. doi: 10.1093/icvts/ivt010. Epub 2013 Mar 4.

Strategic and operational aspects of a transfusion-free neonatal arterial switch operation.

Schweiger M¹, Dave H, Kelly J, Hübler M.

[Tex Heart Inst J](#). 2011; 38(5): 562–564.

PMCID: PMC3231527

Transfusion-Free Complex Cardiac Surgery with Use of Deep Hypothermic Circulatory Arrest in a Preterm 2.96-kg Jehovah's Witness Neonate

Michael Huebler, MD, Helmut Habazettl, MD, Wolfgang Boettcher, ECCP, Hermann Kuppe, MD, Roland Hetzer, MD, and Matthias Redlin, MD

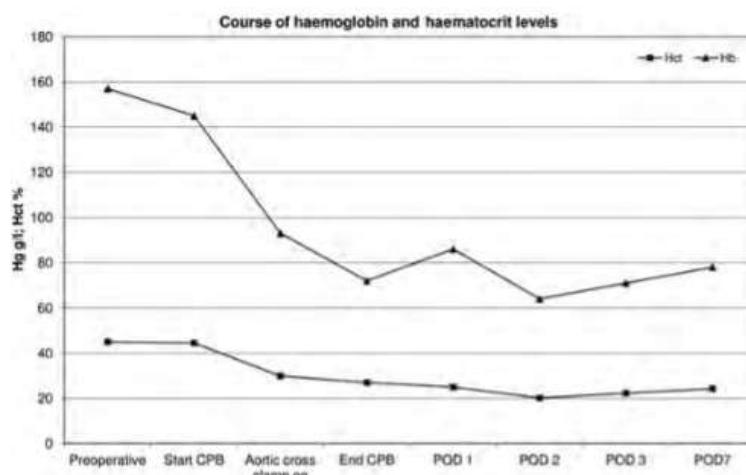


Figure 1: Course of haemoglobin (Hb) and haematocrit (Hct) levels; CPB: cardiopulmonary bypass; POD: postoperative day.

D130, Sorin Group) were used. The total priming volume of the circuit consisted of 95 mL of Deltajonin

lines and 1/8" for the arterial lines. Vacuum-assisted drainage was implemented with a peak negative pressure of 50 mmHg. A hollow-fiber membrane oxygen-



November 15, 2016

Clinical Practice Guidelines From the AABB : Red Blood Cell Transfusion Thresholds and Storage

Jeffrey L. Carson, MD¹; Gordon Guyatt, MD²; Nancy M. Heddle, MSc³; et al

» Author Affiliations

JAMA. 2016;316(19):2025-2035. doi:10.1001/jama.2016.9185



Findings It is good practice to consider the hemoglobin level, the overall clinical context, patient preferences, and alternative therapies when making transfusion decisions regarding an individual patient. Recommendation 1: a restrictive RBC transfusion threshold in which the transfusion is not indicated until the hemoglobin level is 7 g/dL is recommended for hospitalized adult patients who are hemodynamically stable, including critically ill patients, rather than when the hemoglobin level is 10 g/dL (strong recommendation, moderate quality evidence). A restrictive RBC transfusion threshold of 8 g/dL is recommended for patients undergoing orthopedic surgery, cardiac surgery, and those with preexisting cardiovascular disease (strong recommendation, moderate quality evidence). The restrictive transfusion threshold of 7 g/dL is likely comparable with 8 g/dL, but RCT evidence is not available for all patient categories. These recommendations do not apply to patients with acute coronary syndrome, severe thrombocytopenia (patients treated for hematological or oncological reasons who are at risk of bleeding), and chronic transfusion-dependent anemia (not recommended due to insufficient evidence). Recommendation 2: patients, including neonates, should receive RBC units selected at any point within their licensed dating period (standard issue) rather than limiting patients to transfusion of only fresh (storage length: <10 days) RBC units (strong recommendation, moderate quality evidence).

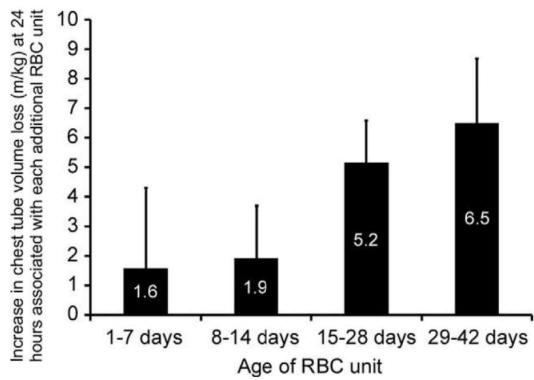
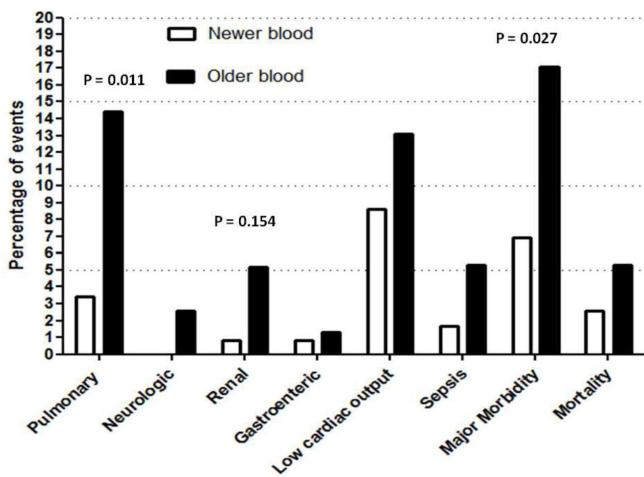
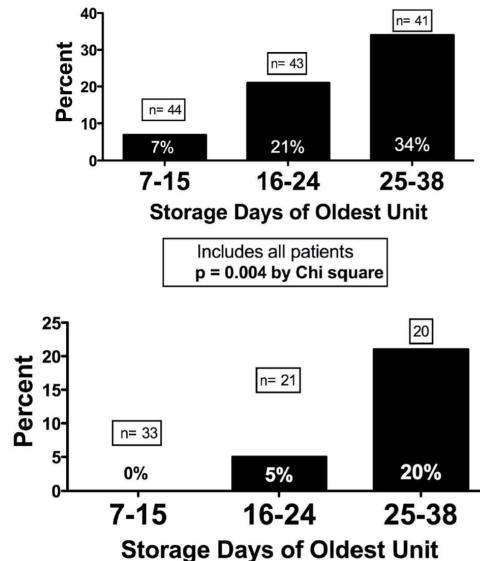


Fig 2. Increase in chest tube drainage over the first 24 postoperative hours for each additional allogeneic red blood cell (RBC) unit by duration of storage.

Saignement postop correlé avec âge CGR (Manhiet ATS 2013)



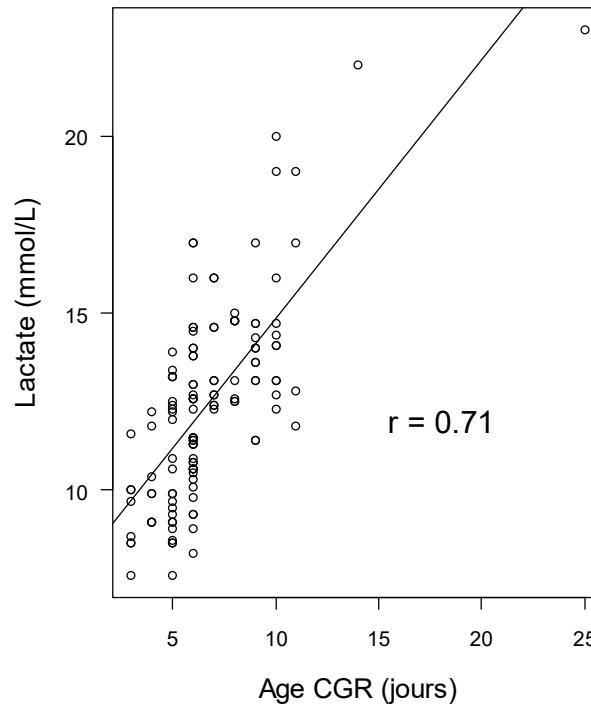
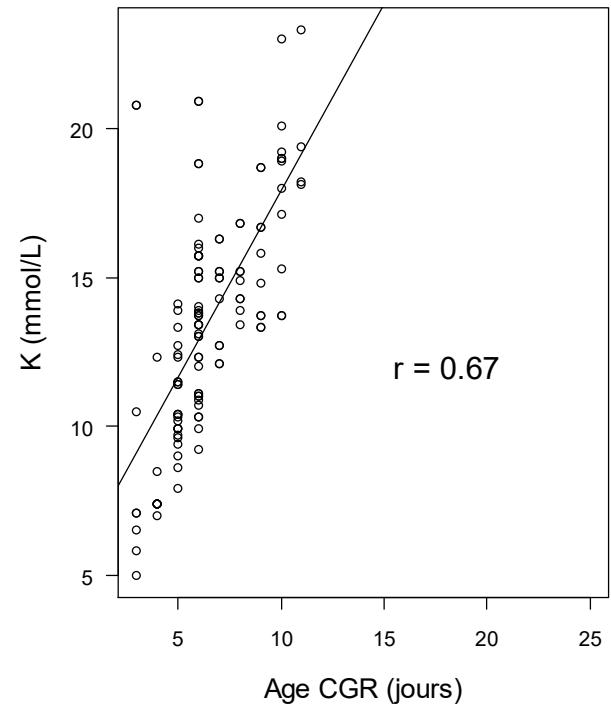
Morbidity and mortality in patients receiving newer vs. older blood in the cardiopulmonary bypass circuit.



Plus d'infections nosocomiales avec vieux CGR (Cholette, Ped Crit Care 2015)

Plus de morbidité postop avec des vieux CGR : pneumopathie, insuffisance rénale postop, complications neurologiques (Ranucci Crit Care 2009)

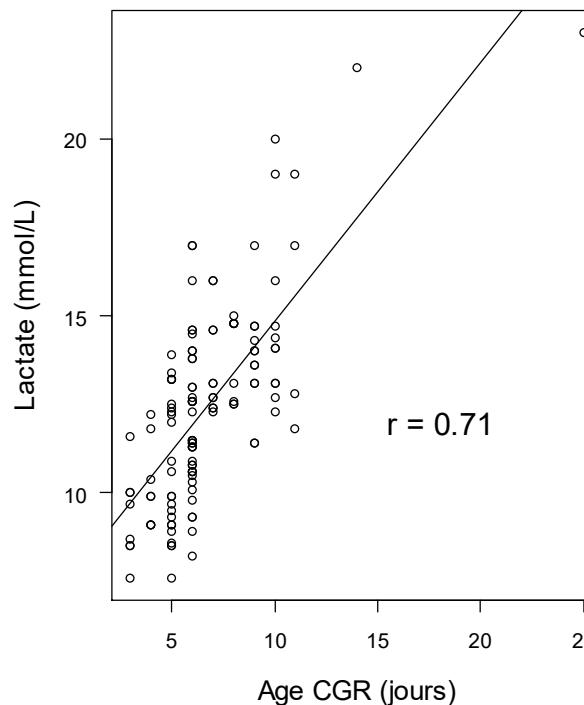
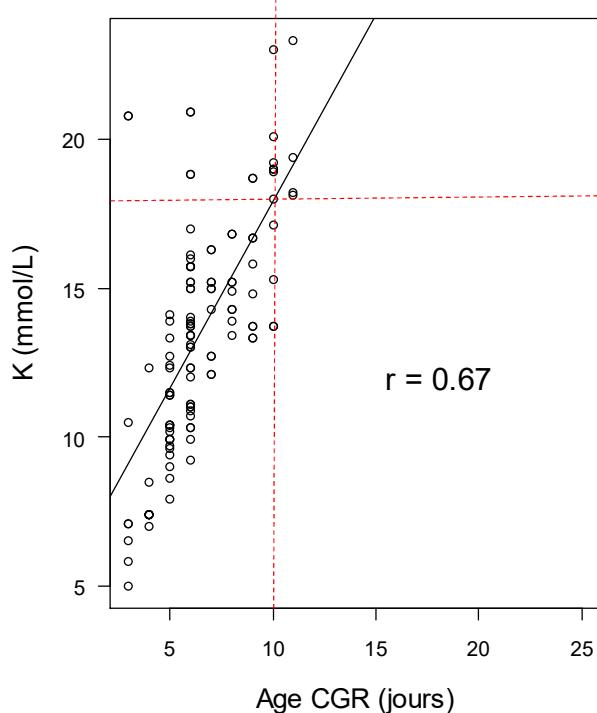
Concentration en potassium et lactate des concentrés globulaires selon leur durée de conservation



données de Necker

! Potassium plus élevé encore si utilisation de culots irradiés (patients atteints du syndrome DiGeorge)

Concentration en potassium et lactate des concentrés globulaires selon leur durée de conservation



données de Necker

$$[\text{K actuel}] = ([\text{K patient}] * \text{volémie} + [\text{K priming}] * \text{volume priming}) / (\text{volémie} + \text{volume priming})$$

Kaliémie dans un priming constitué de $\frac{1}{2}$ CGR de 10 jours et $\frac{1}{2}$ PFC = environ 9 mmol/L

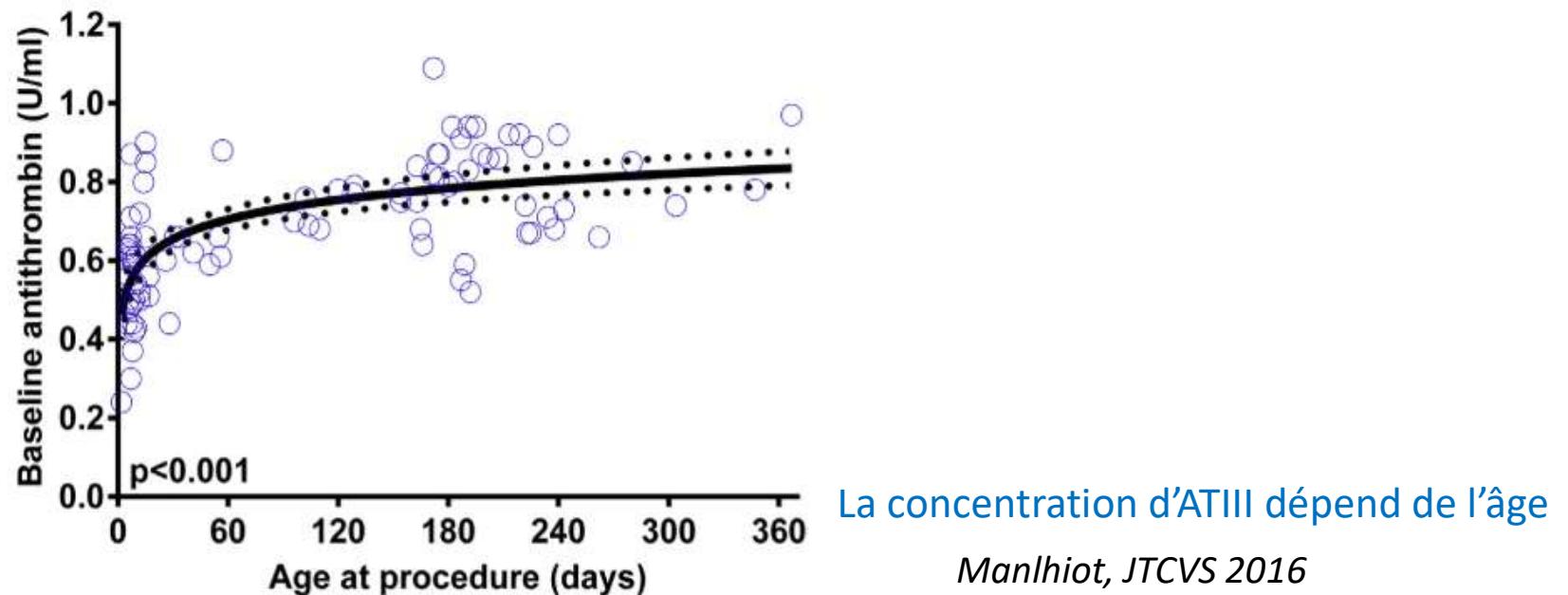
Kaliémie d'un nouveau-né de 3 kg (volémie estimé 240ml) au démarrage de sa CEC (volume du priming 250ml) avec un CGR de 10 jours :

$$[\text{K}] = (4 * 240 + 9 * 250) / (240 + 250) = 6.5 \text{ mmol/L}$$

Pour un culot de 15 jours ça peut approcher 7.6 mmol/L, qui peut causer un arrêt cardiaque au démarrage de la CEC

Priming au CGR + PFC chez les nouveau-nés

Héparine 250-300 U/kg (selon si circuit pré-hépariné ou pas), objectif ACT > 400s



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Température de la CEC

Normothermie > 36°C

Hypothermie légère 30-36°C

Hypothermie modérée 22-30°C

Hypothermie profonde 17-22°C

- Le débit nécessaire en CEC varie avec la température (variation de la VO₂)

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 WJ¹, Kern FH, Ungerleider RM, Boyd JL 3rd, Quill T, Smith LR, Baldwin B, Reves JG.

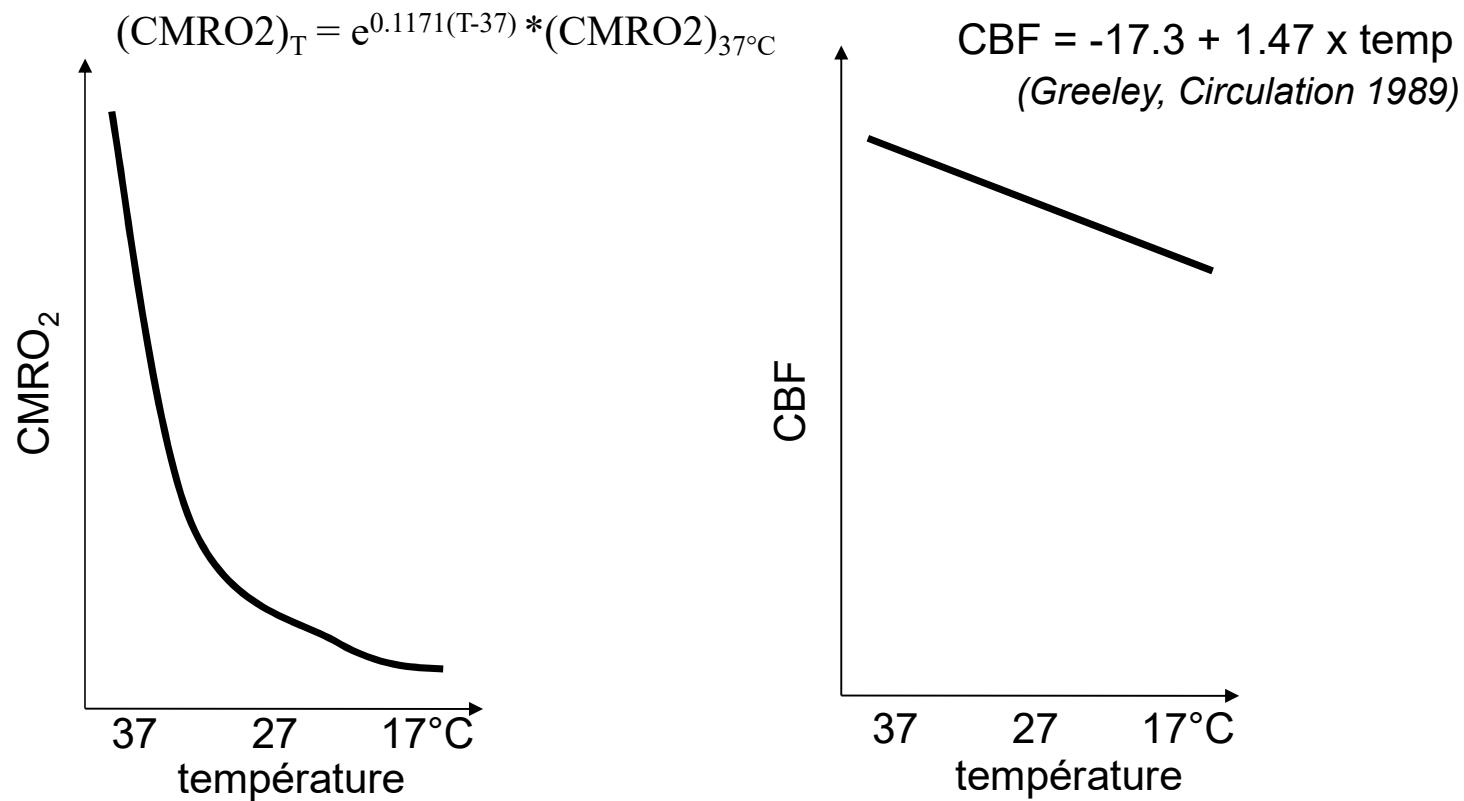
Q_{10} (index thermique) = % de variation de la CMRO₂ lorsque la température varie de 10°C

$$Q_{10} = 3.65$$

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

Protection cérébrale par l'hypothermie

Effet de l'hypothermie sur la CMRO₂ et CDO₂



27 patients âgés 3j-13ans

Température et débit CEC

Table 3. Calculated Cerebral Oxygen Consumption Reduction and Predicted Minimal Flow Rates^a

Temperature (°C)	CMRO ₂ (mL · 100 g ⁻¹ · min ⁻¹)	Predicted MPFR (mL · kg ⁻¹ · min ⁻¹)
37	1.48	100
32	0.823	56
30	0.654	44
28	0.513	34
25	0.362	24
20	0.201	14
18	0.159	11
15	0.112	8

(Kern, ATS 1993)

De manière consensuelle, le débit en CEC néonatale normothermique est 150 ml/kg/min, soit environ 2.7 l/min/m² voire plus si anastomoses aorto-pulmonaires

Effet de l'hypothermie sur la CMRO₂ et 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
- ↑ 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

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 II A 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)

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Sevrage de la CEC en pédiatrie

La pression de perfusion Le seuil d'autorégulation de la circulation cérébrale

Monitoring Cerebral Blood Flow Pressure Autoregulation in Pediatric Patients During Cardiac Surgery

Ken M. Brady, MD; Jennifer O. Mytar, BS; Jennifer K. Lee, MD; Duke E. Cameron, MD;
Luca A. Vricella, MD; W. Reid Thompson, MD; Charles W. Hogue, MD; R. Blaine Easley, MD

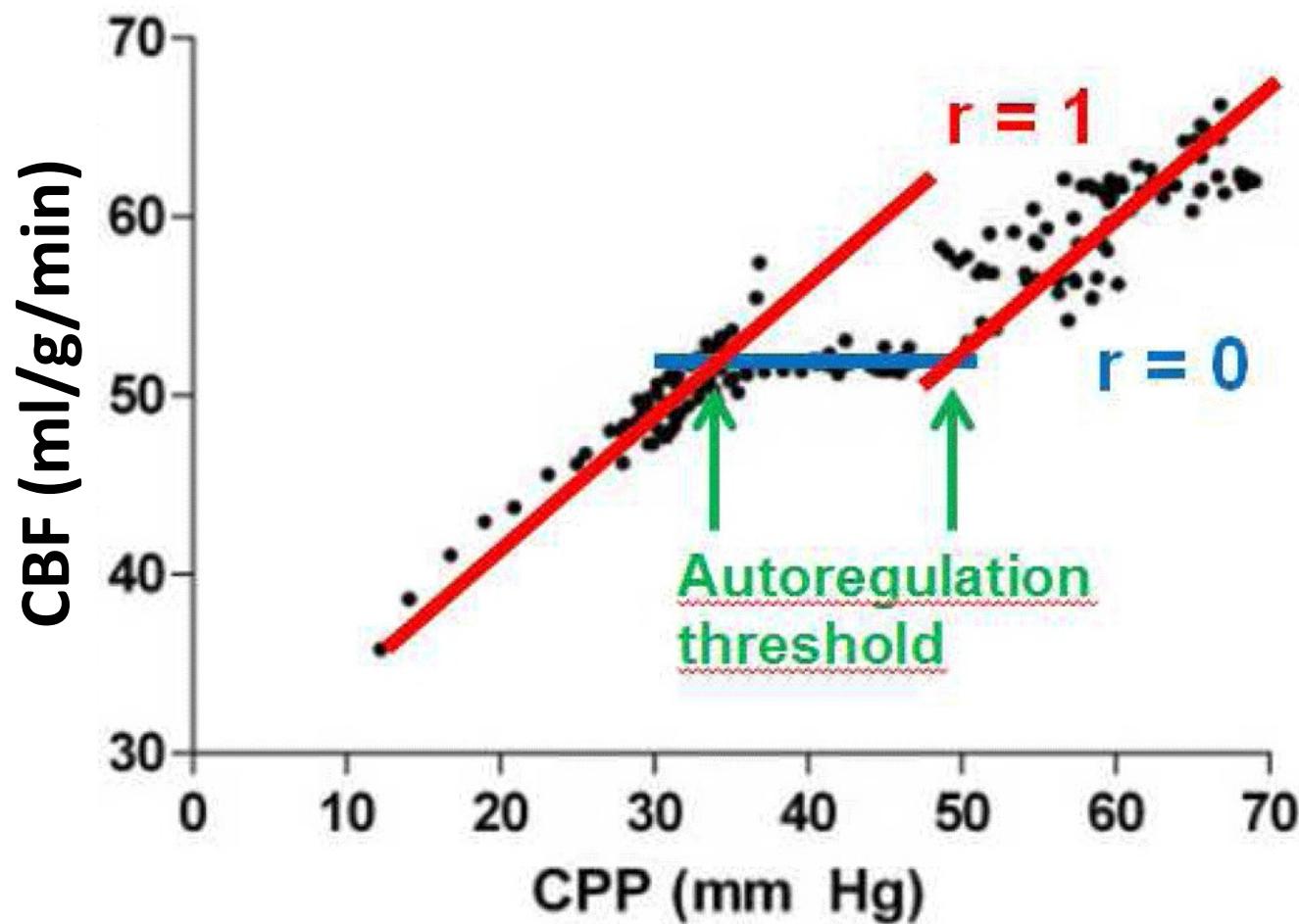
N = 54 (nouveau-nés, nourrissons et enfants)

Results—Hypotension was associated with increased values of COx ($P<0.0001$). For 77% of patients, an individual estimate of lower limits of pressure autoregulation could be determined using a threshold COx value of 0.4. The mean lower limits of pressure autoregulation for the cohort using this method was 42 ± 7 mm Hg.

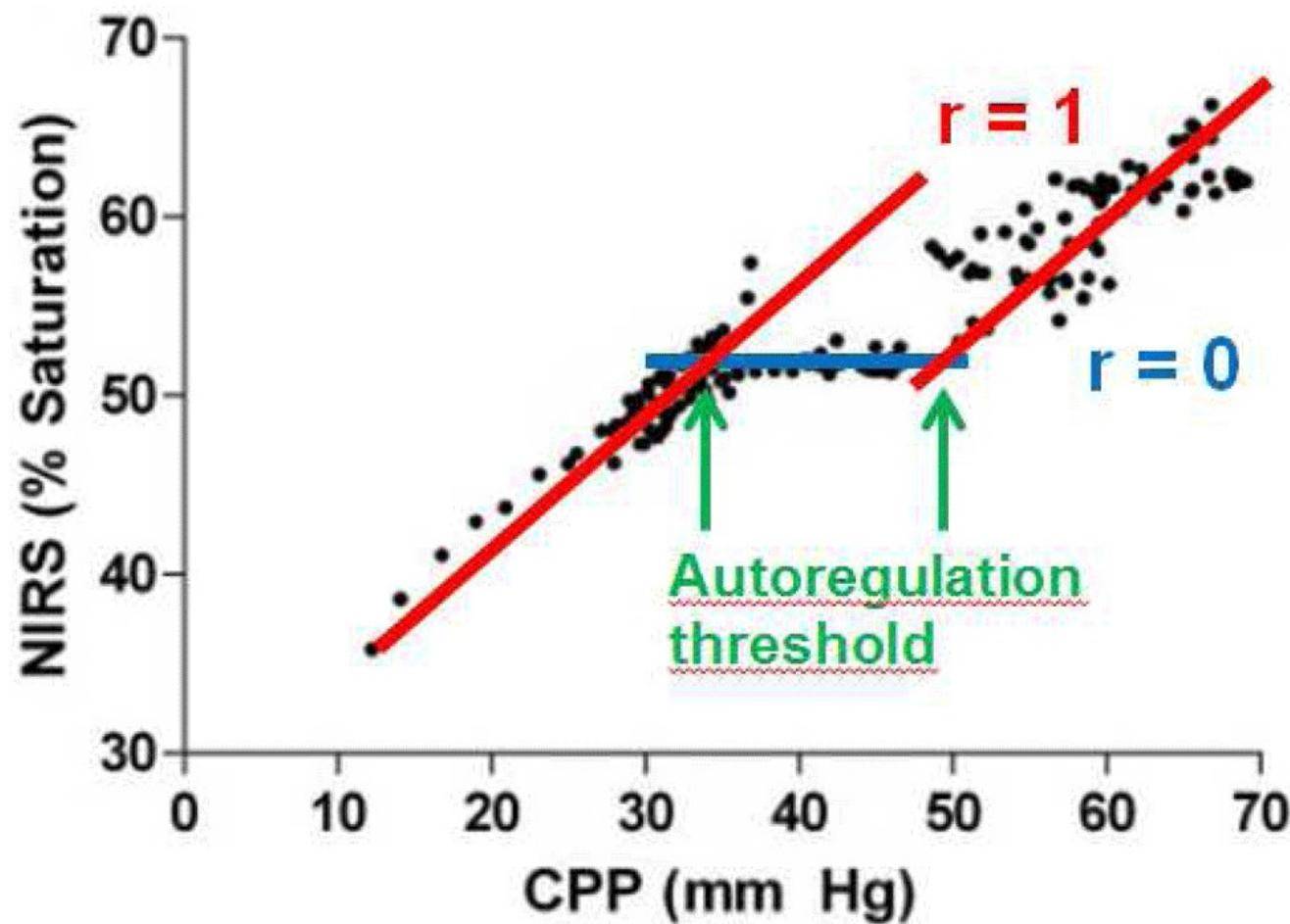
Conclusions—This pilot study of COx monitoring in pediatric patients demonstrates an association between hypotension during cardiopulmonary bypass and impairment of autoregulation. The COx may be useful to identify arterial blood pressure-dependent limits of cerebral autoregulation during cardiopulmonary bypass. Larger trials with neurological outcomes are indicated. (*Stroke*. 2010;41:1957-1962.)

(Cox: « index de corrélation mouvante » entre la NIRS et la PAM, normal < 0.4)

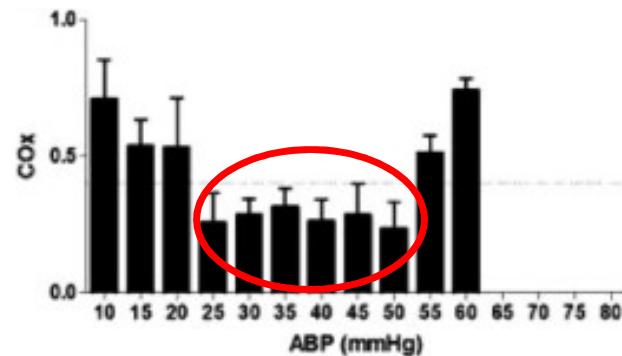
Cerebral blood flow autoregulation



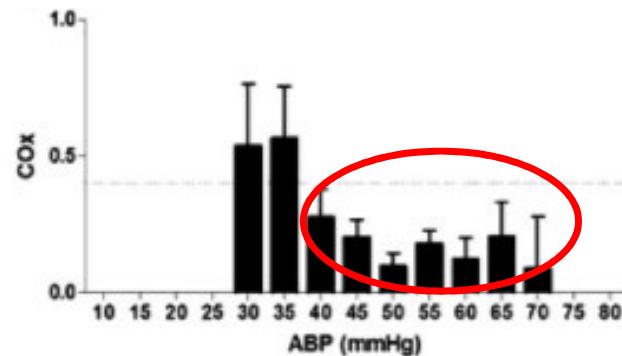
Cerebral blood flow autoregulation



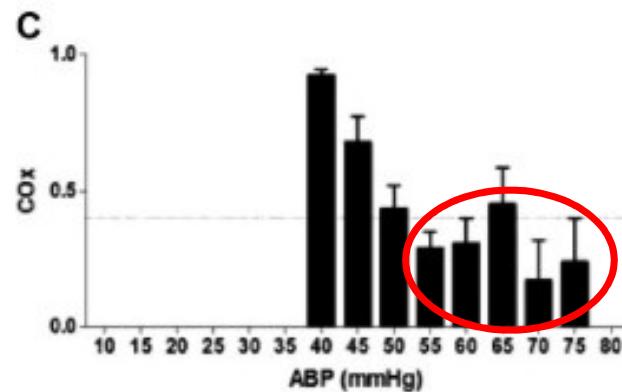
6 jours, interruption de l'arche aortique



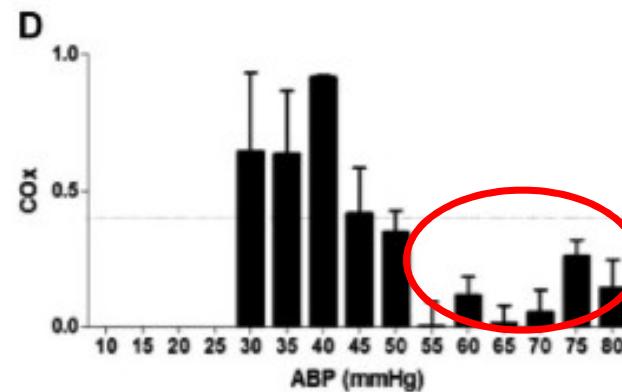
7 mois, CIV



2 ans, CIV



7 ans, ALCAPA

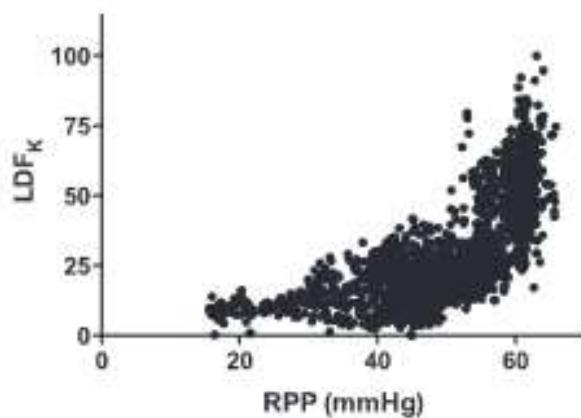
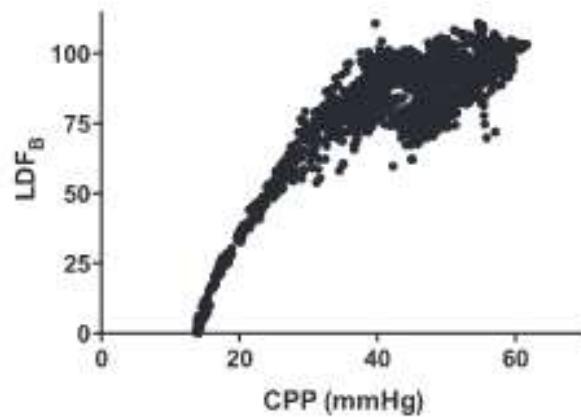


Il existe un seuil de pression de perfusion, en dessous duquel il y a perte de l'autorégulation cérébrale en CEC

Pression de perfusion = MAP – PVC

- 30-40mmHg chez le nouveau-né
- 40-50mmHg < 2 ans
- 50 mmHg > 2 ans

Renovascular reactivity measured by near-infrared spectroscopy



compared with cerebral laser-Doppler measurements. Renal blood flow decreased to 75%, 50%, and 25% of baseline at perfusion pressures of 60, 45, and 40 mmHg, respectively, whereas in the brain these decrements occurred at pressures of 30, 25, and 15 mmHg, respectively. The RVx compared favorably to the renal laser-Doppler

Cerebral autoregulation and anesthesia

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Current Opinion in Anaesthesiology 2009,
22:547–552

Purpose of review

This review will examine the recent literature on anesthesia and monitoring techniques in relation to cerebral autoregulation. We will discuss the effect of physiologic and pharmacological factors on cerebral autoregulation alongside its clinical relevance with the help of new evidence.

Recent findings

Intravenous anesthesia, such as combination of propofol and remifentanil, provides best preservation of autoregulation. Among inhaled agents sevoflurane appears to preserve autoregulation at all doses, whereas with other agents autoregulation is impaired in a dose-related manner.

Summary

Intraoperative cerebral autoregulation monitoring is an important consideration for the patients with neurologic disease. Transcranial Doppler based static autoregulation measurements appears to be the most robust bedside method for this purpose.

Keywords

anesthetics, cerebral autoregulation, cerebral blood flow, cerebral blood flow velocity, cerebral perfusion pressure, transcranial Doppler

Particularités du circuit CEC en pédiatrie (miniaturisation)

Hematocrite idéal et transfusion

Température et débit de CEC

Pression de perfusion

Hypothermie et protection cérébrale

Monitoring

Inflammation

Cardioplégie

Sevrage de la CEC en pédiatrie

Température de la CEC

Normothermie > 36°C

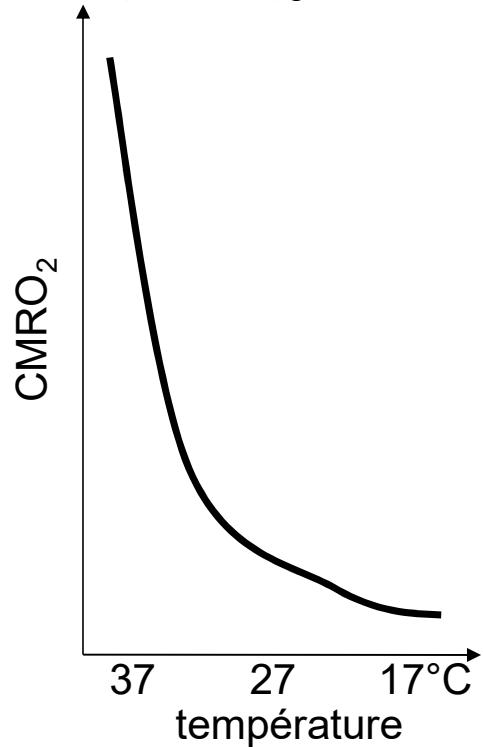
Hypothermie légère 30-36°C

Hypothermie modérée 22-30°C

Hypothermie profonde 17-22°C

L'utilisation de l'hypothermie en CEC pédiatrique

$$(CMRO_2)_T = e^{0.1171(T-37)} * (CMRO_2)_{37^\circ C}$$

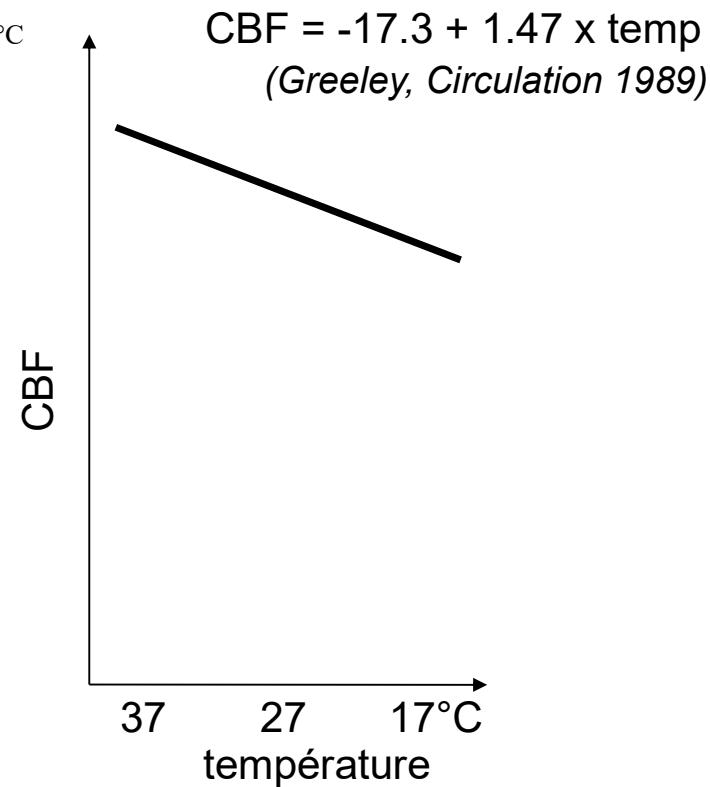
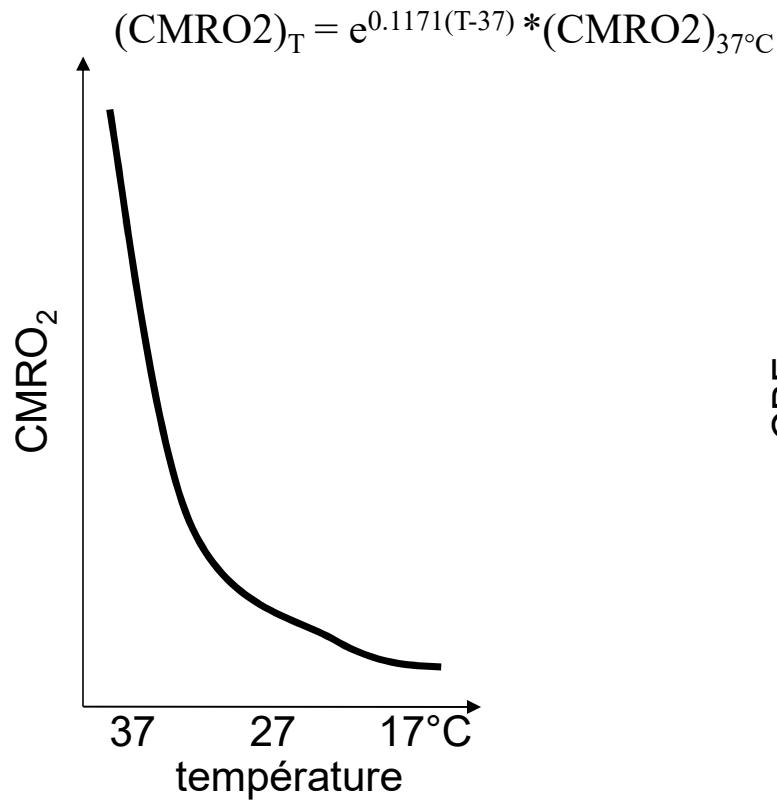


Q_{10} (index thermique) = % de variation du paramètre lorsque la température varie de 10°C

$$Q_{10} = 3.65$$

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



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 WJ¹, 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*)

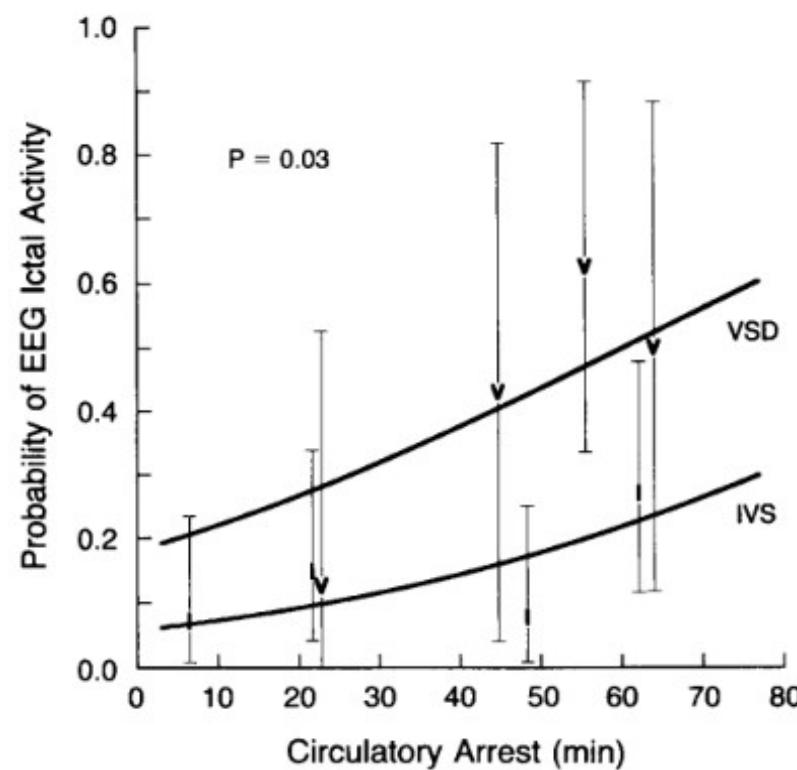
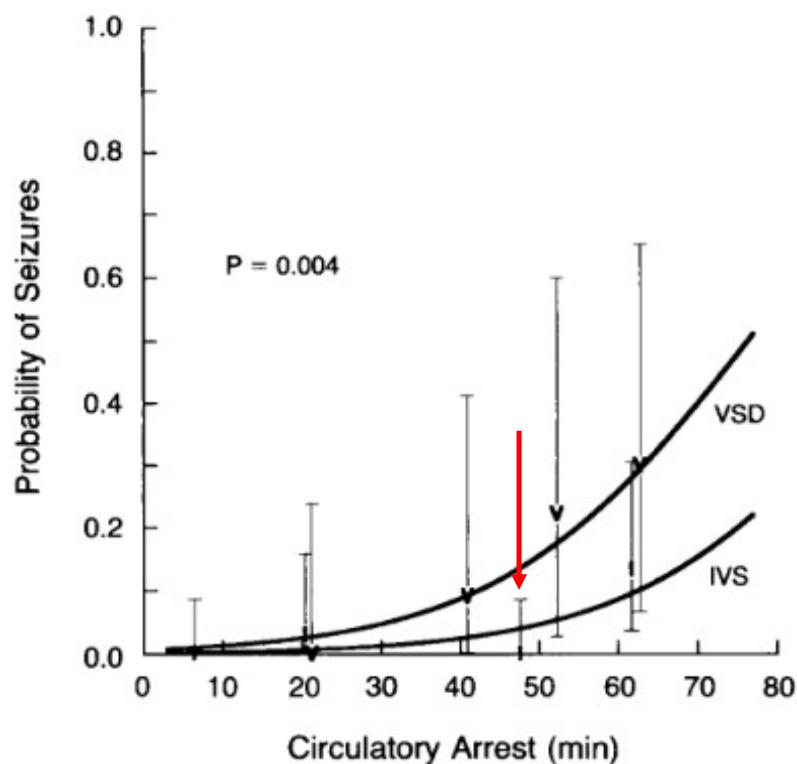
171 nouveau-nés avec TGV, 87 DHCA + 84 low-flow, 18°C, α-stat, Ht>20%

- > 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

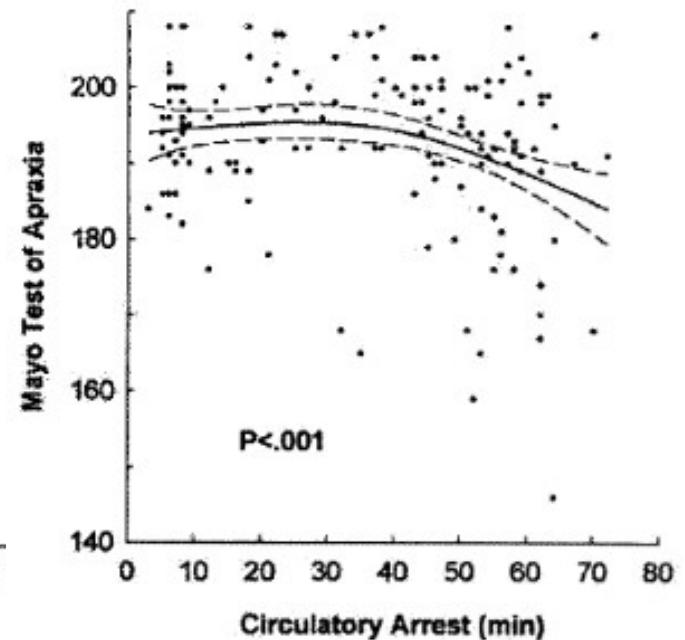
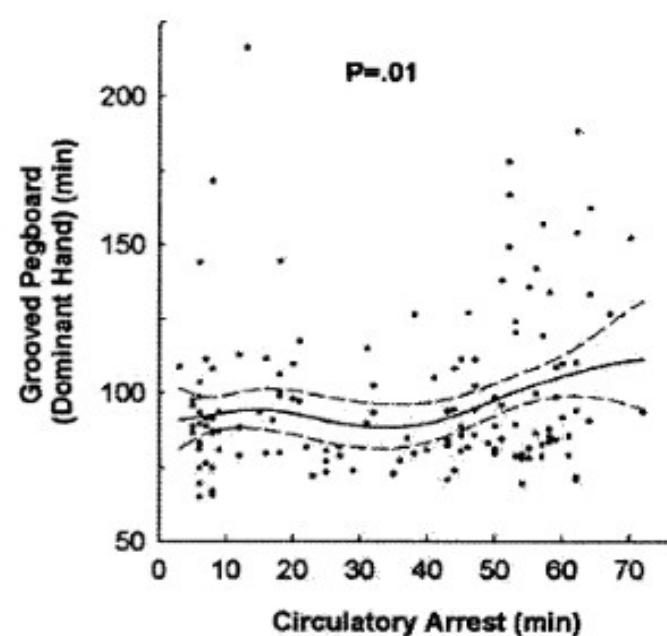
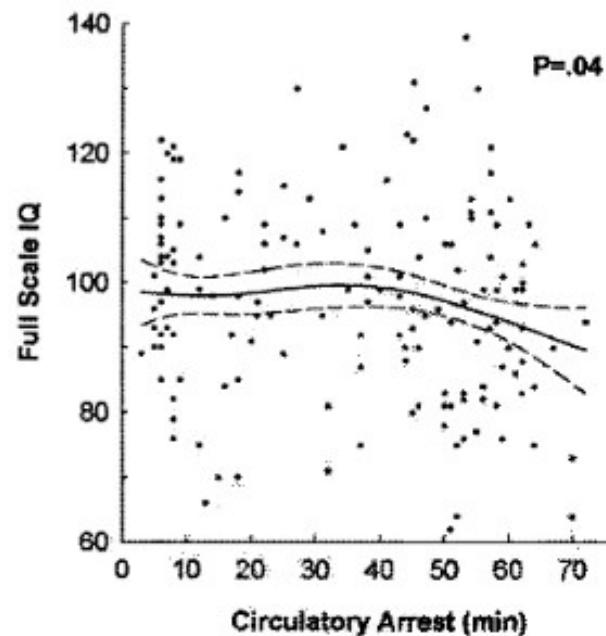


(Newburger, NEJM 1993)

Boston Circulatory Arrest Study

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

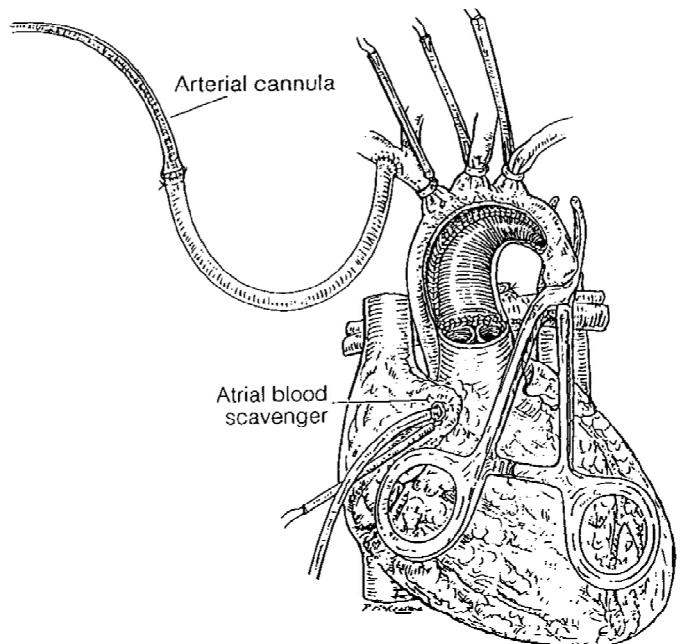
tests psychomoteurs à 8 ans



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

(Wypij, JTCVS 2003)

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

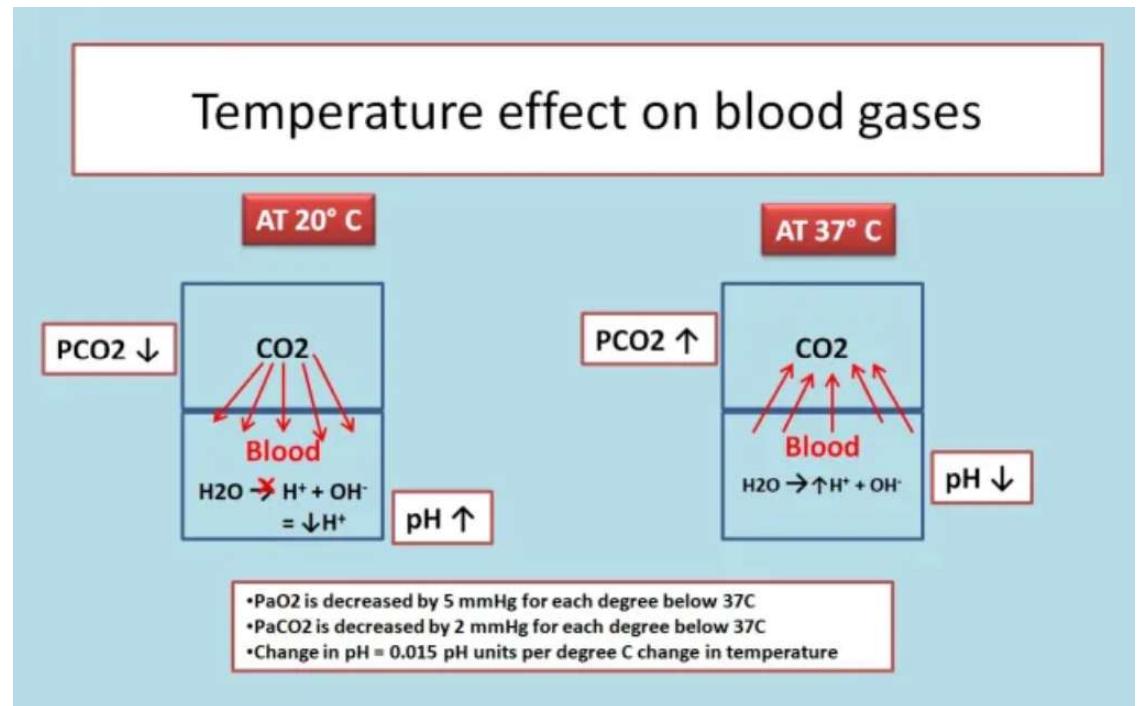
Pigula, JTCVS 2000 : collatéralité responsable d'un reflux dans l'aorte descendante

Andropoulos, JTCVS 2003 : 24 – 94 ml/kg min !!! en utilisant un monitoring multimodal (rSO₂ et DTC)

Quelle pression ?

Tanaka, JTCVS 1998 : pas moins de 40 mmHg

Solubilité CO₂ ~ 1/ température



α -stat:

$pCO_2 = \text{const}$

$pH = \text{var}$

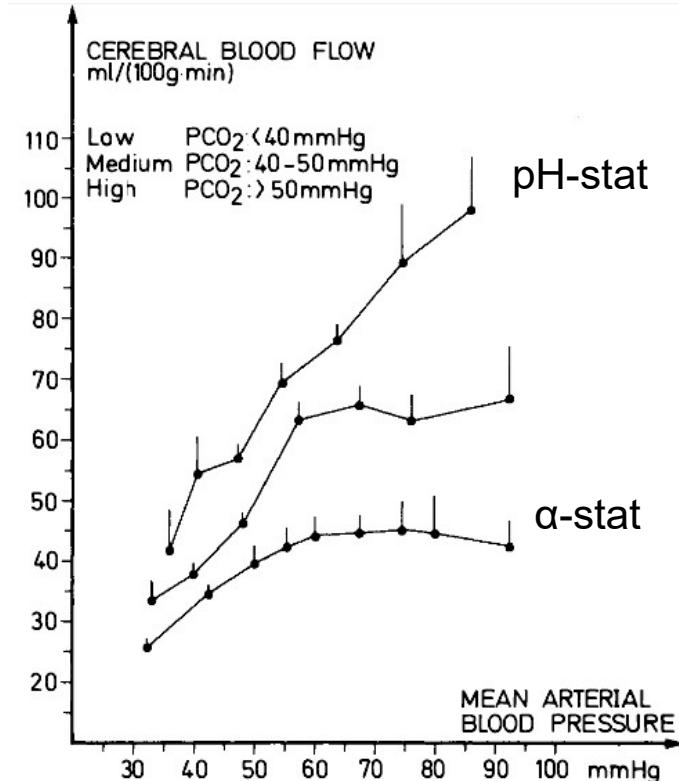
-> vasoconstriction cérébrale, meilleur maintien de l'autorégulation ?

pH-stat:

$pCO_2 = \text{var}$

$pH = \text{const}$

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



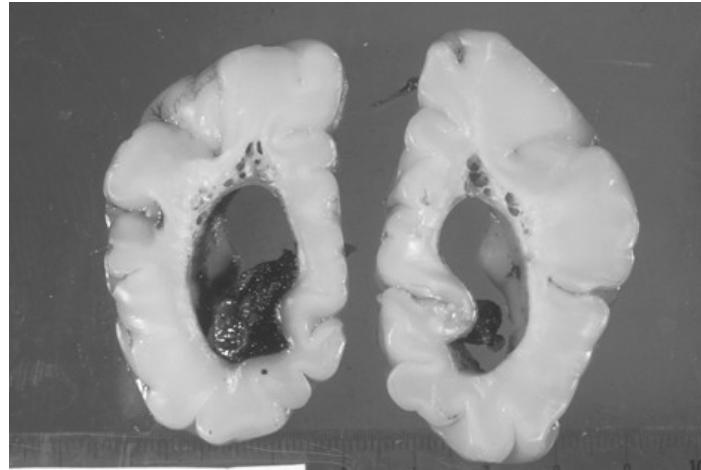
adultes, 22-28°C

(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*

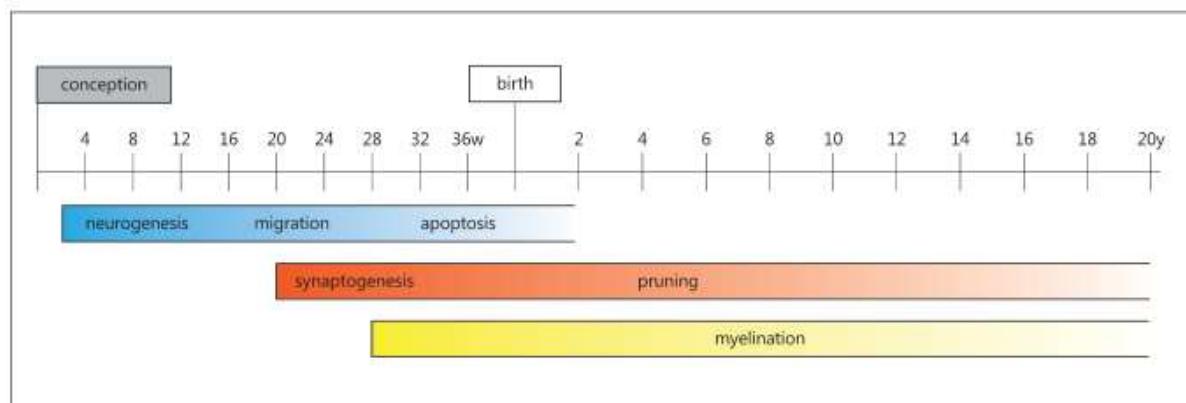
n = 24NN
dont 8 HLHS

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	—

Perspective
MARCH 9, 2017

Anesthesia and Developing Brains — Implications of the FDA Warning

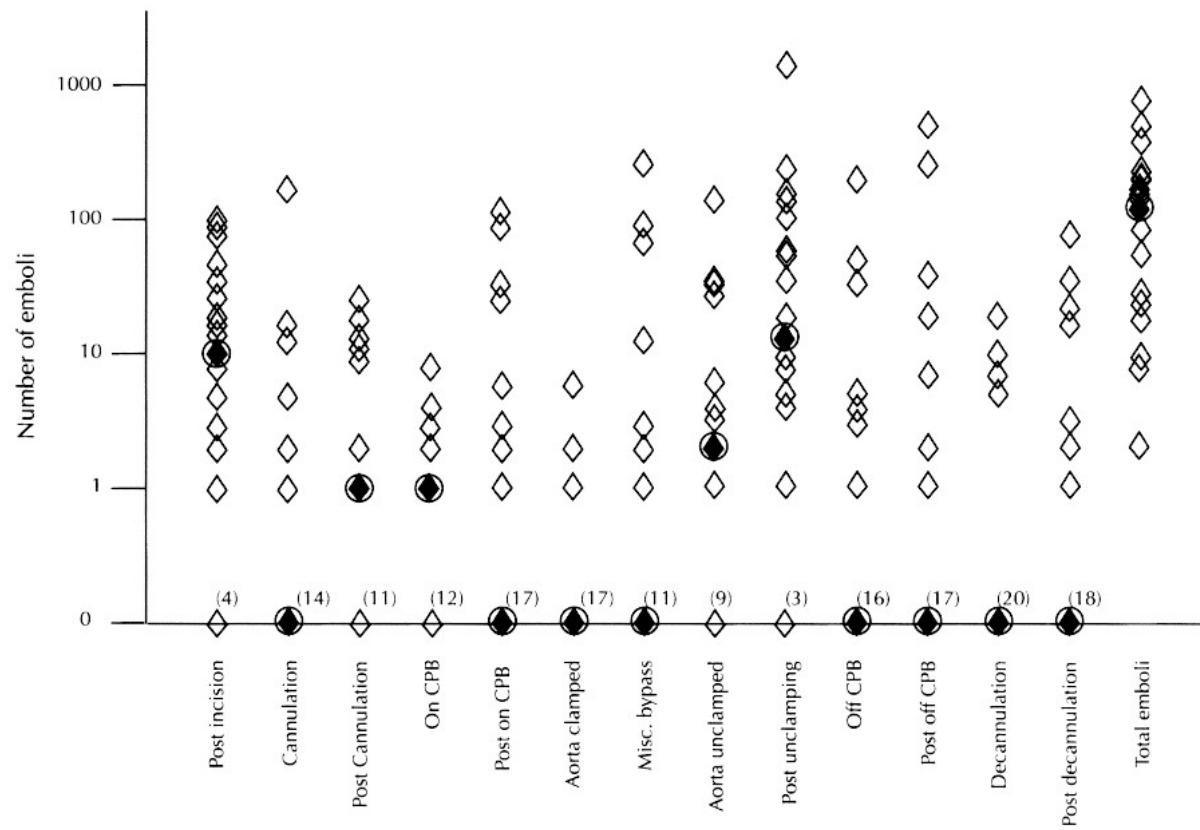
Dean B. Andropoulos, M.D., M.H.C.M., and Michael F. Greene, M.D.



L'embolie cérébrale gazeuse, particulière

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

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



Particularités du circuit CEC en pédiatrie (miniaturisation)

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Sevrage de la CEC en pédiatrie

Monitoring de la perfusion

IC et DO2

De Somer, Crit Care 2011: O₂-derived parameters [...], « time for a goal-directed perfusion management »

Développement du logiciel CONNECT par Livanova pour le monitorage continu de la DO2

DO2 critique:

Adulte:

*Liebermann, Anesthesiology 2000, 8 volontaires conscients : 7.3 mL kg⁻¹ min⁻¹
≈ 300 mL min⁻¹ m⁻²*

Ranucci ATS 2005, sous anesthésie en CEC : 260 - 270 mL min⁻¹ m⁻²

DO₂ critique

Enfant :

Abraham, JTCVS 2010

CIA, âge moyen 4.5 ans

débit CEC « critique » = $100 \text{ ml kg}^{-1} \text{min}^{-1}$

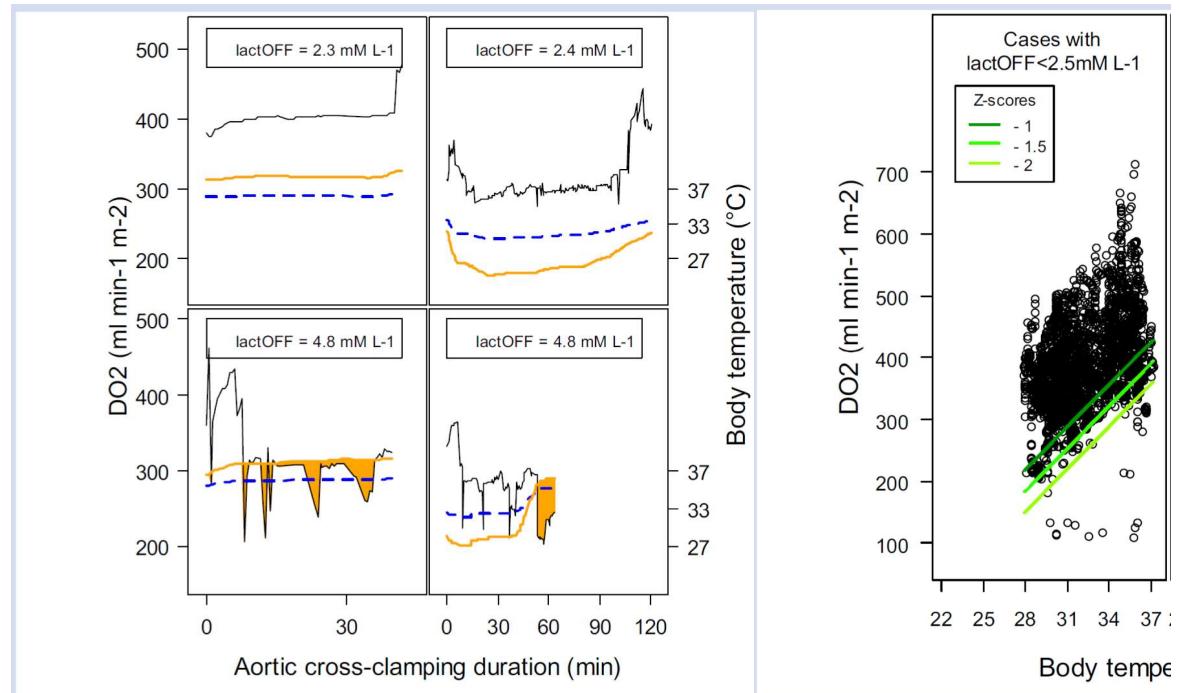
Hb 9g/dL, BSA 0.76 m² \rightarrow DO₂ critique **330 mL min⁻¹ m⁻²**

Nouveau-né

Bojan, BJA 2020, 180 nouveau-nés

22896 mesures per-CEC

DO₂ critique = **340 ml min⁻¹ m⁻²**



Bojan, BJA 2020

Monitoring de la perfusion

Le CO₂ éliminé par l'oxygénateur

Durant la CEC, il y a 2 sources de CO₂:

- Le métabolisme aérobie
- Le tamponnement des protons par le bicarbonate

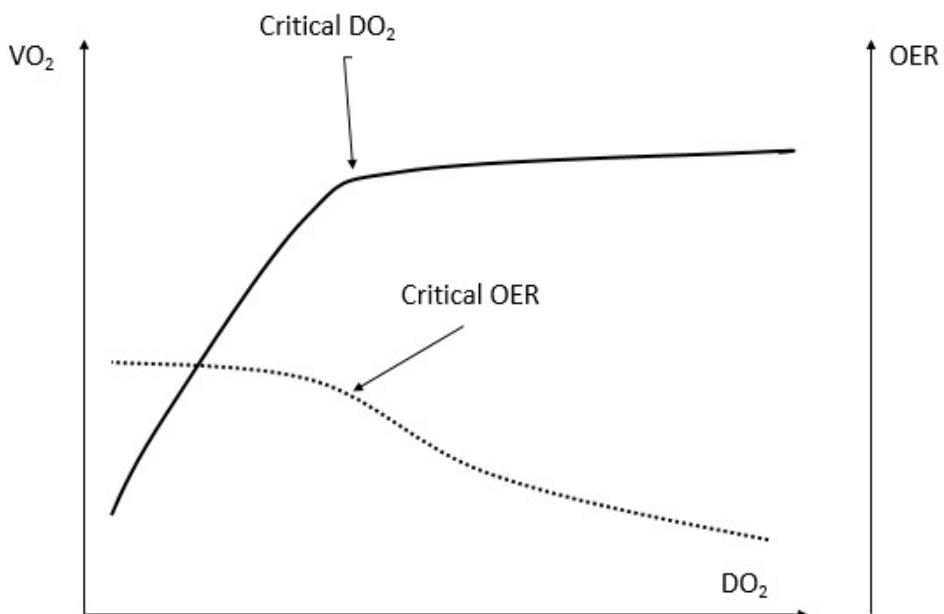
Indicateurs de la présence du métabolisme anaérobie:

- $VCO_2 > 60 \text{ ml/min/m}^2$
- $VCO_2/VO_2 > 0.9$
- $DO_2/VCO_2 < 5$

Ranucci, ATS 2006

Monitoring de la perfusion

La SvO₂



$$\text{OER} = (\text{SaO}_2 - \text{SvO}_2) / \text{SaO}_2$$

$$\text{SvO}_2 = (1 - \text{OER}) \times \text{SaO}_2 \text{ (mais SaO}_2 \text{ souvent 100\%)}$$

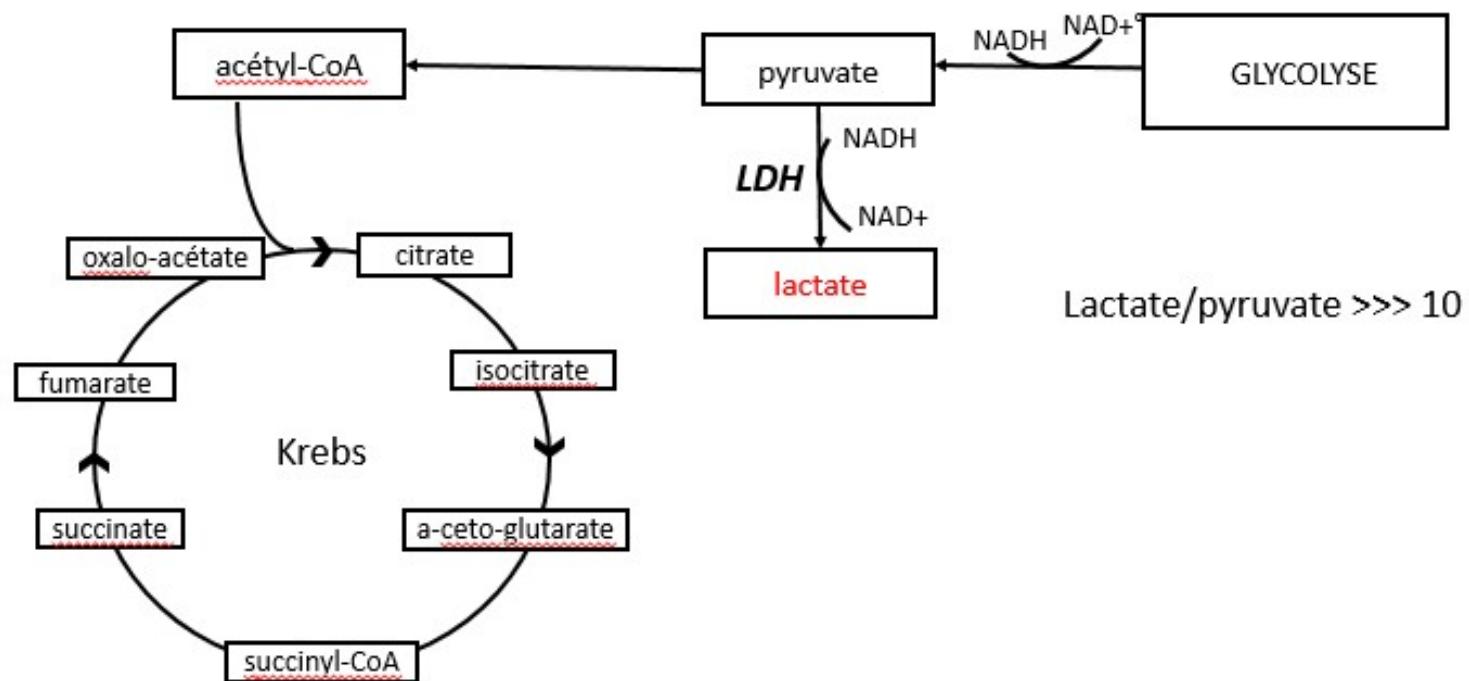
$$\text{SvO}_2 = 1 - \text{OER}$$

Maintien **OER < 30%** -> nécessité de maintenir **SvO₂ > 70%**

Monitoring de la perfusion

Le lactate

- Est un reflet de l'adéquation DO₂/VO₂ en CEC
- Est une excellent indicateur du risque de complications postop (>3.5 mmol/L)



Monitoring de la perfusion

La NIRS

- Positionnement des canules
- Monitoring des circulations régionales

OER cérébral = 35%, OER somatique (rénal) = 10-15%

→ *le gradient rSO₂ somatique-cérébral est normalement 10-15%*

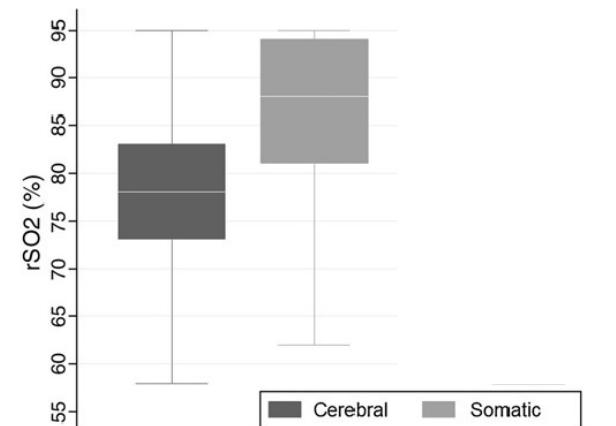
La circulation cérébrale est sous le contrôle de l'autorégulation du DSC

La circulation somatique (rénale) est sous contrôle adrénnergique

→ *le gradient rSO₂ somatique-cérébral se réduit lors de la stimulation sympathique (état de choc)*

Bernal, J Pediatr Surg 2010

Cerebral and somatic near-infrared spectroscopy in normal newborns



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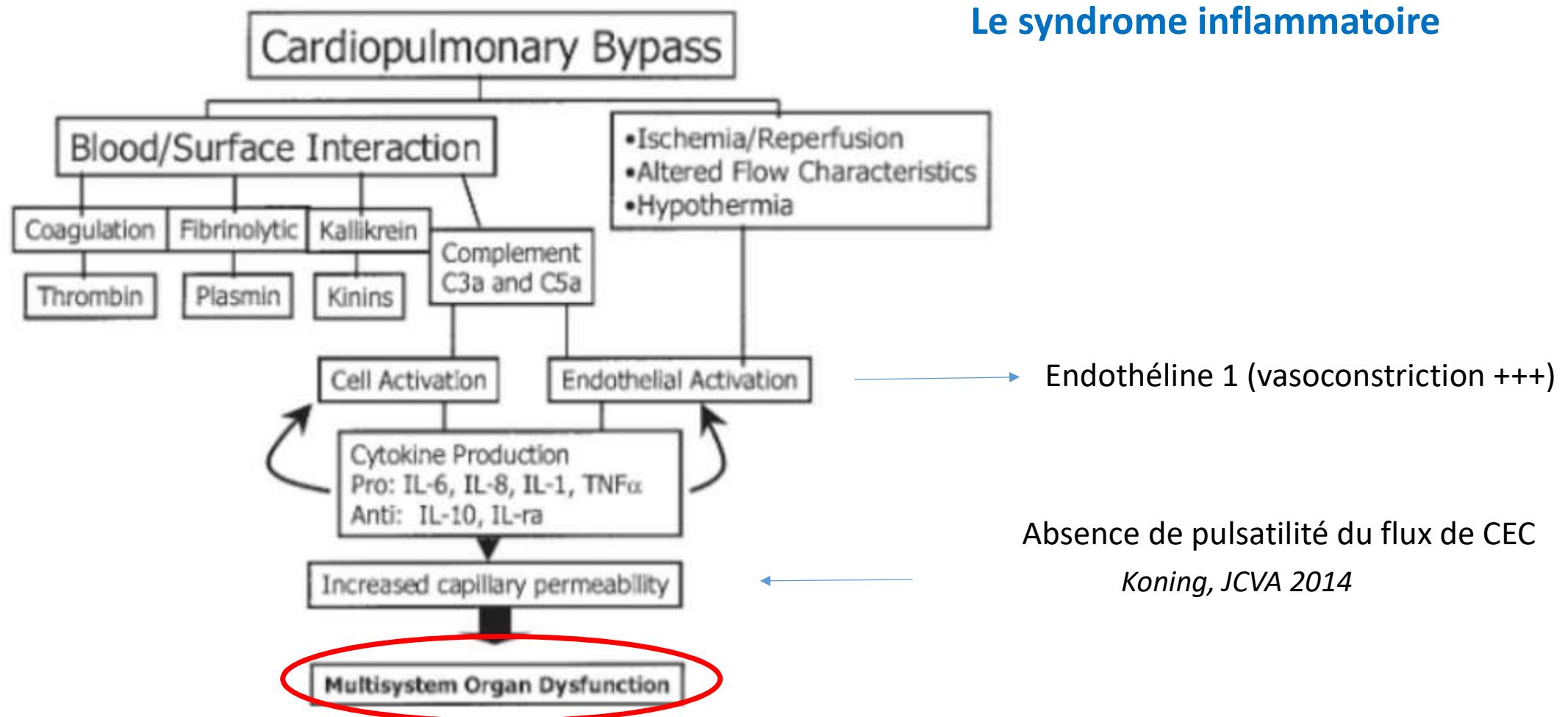
Monitoring

Inflammation

Cardioplégie

Sevrage de la CEC en pédiatrie

Le syndrome inflammatoire



Kozik, ATS 2006

Traitements anti-inflammatoires : les corticoïdes

Méthylprédnisolone 30 mg/kg avant CEC et 30 mg/kg durant CEC

Effets bénéfiques : diminuent le ratio IL6 / IL 10
augmentent la concentration plasmatique de noradrénaline (inhibition de la COMT)

Effets indésirables : hyperglycémie, nécessité du recours à l'insuline (risque hypoGly chez le nouveau-né ++)
risque d'insuffisance surrénalienne postop (*Crawford, Pediatric Anesth 2016*)
durée de séjour plus longue, risque infectieux accru (*Pasquali, Circulation 2010*)

aucun bénéfice postop !

Traitements anti-inflammatoires : l'hypothermie = NON

The influence of bypass temperature on the systemic inflammatory response and organ injury after pediatric open surgery: A randomized trial

Christian F. Stocker, MD,^{a,b} Lara S. Shekerdemian, MD,^{b,c,f} Stephen B. Horton, PhD,^{d,f}
Katherine J. Lee, PhD,^g Rob Eyes, MD,^e Yves D'Udekem, MD, PhD,^{d,f} and Christian P. Brizard, MD^{b,d,f}

Objective: Systemic cooling for cardiopulmonary bypass is widely used to attenuate the systemic inflammatory response syndrome and organ injury in children after open surgery. We compared the effects of moderate (24°C) and mild (34°C) hypothermia during bypass on markers of the systemic inflammatory response syndrome and organ injury, and on clinical outcome after corrective surgery for congenital heart disease.

Methods: Sixty-six children (mean age, 6.8 ± 5.7 months; mean weight, 6.2 ± 2.3 kg) were randomized to 24°C or 34°C bypass temperature during cardiac surgery. Perfusion strategies were otherwise strictly identical. Clinical data and blood samples were collected before bypass, 5 minutes after aortic crossclamp release, and 4, 24, and 48 hours after bypass. Patients were followed up until discharge from the hospital.

Results: In the 54 children with outcome data, bypass temperature did not influence the duration of mechanical ventilation between the 24°C group and the 34°C group (median [interquartile range] 22 [13–40] hours vs 14 [8–40] hours, $P = .14$), intensive care unit stay (43 [24–49] hours vs 29 [23–47] hours, $P = .79$), blood loss (29 [20–38] mL/kg vs 23 [13–38] mL/kg, $P = .36$), or incidence of postoperative infection (9% vs 11%, $P = 1.0$). There was no evidence of an influence of bypass temperature on the markers of acute inflammation, innate immune response, organ injury, coagulation, or hemodynamics.

Conclusions: There is no evidence that the systemic inflammatory response syndrome and organ injury after pediatric open surgery are influenced by bypass temperature. The routine use of hypothermic bypass may not be warranted in the pediatric population. (J Thorac Cardiovasc Surg 2011;142:174–80)

Traitements anti-inflammatoires : UF, MUF

Ultrafiltration conventionnelle	Ultrafiltration modifiée
Pendant la CEC, à tout moment ?, surtout après déclampage Oxygénateur -> hémofiltre -> réservoir Volémie constante, compensation du volume filtré par transfusion/remplissage cristalloïde	A la fin de la CEC, avant l'ablation des canules, pendant 15-20 min Canule aortique -> hémofiltre -> oxygénateur -> canule veineuse Volémie constante, compensation par le sang du réservoir

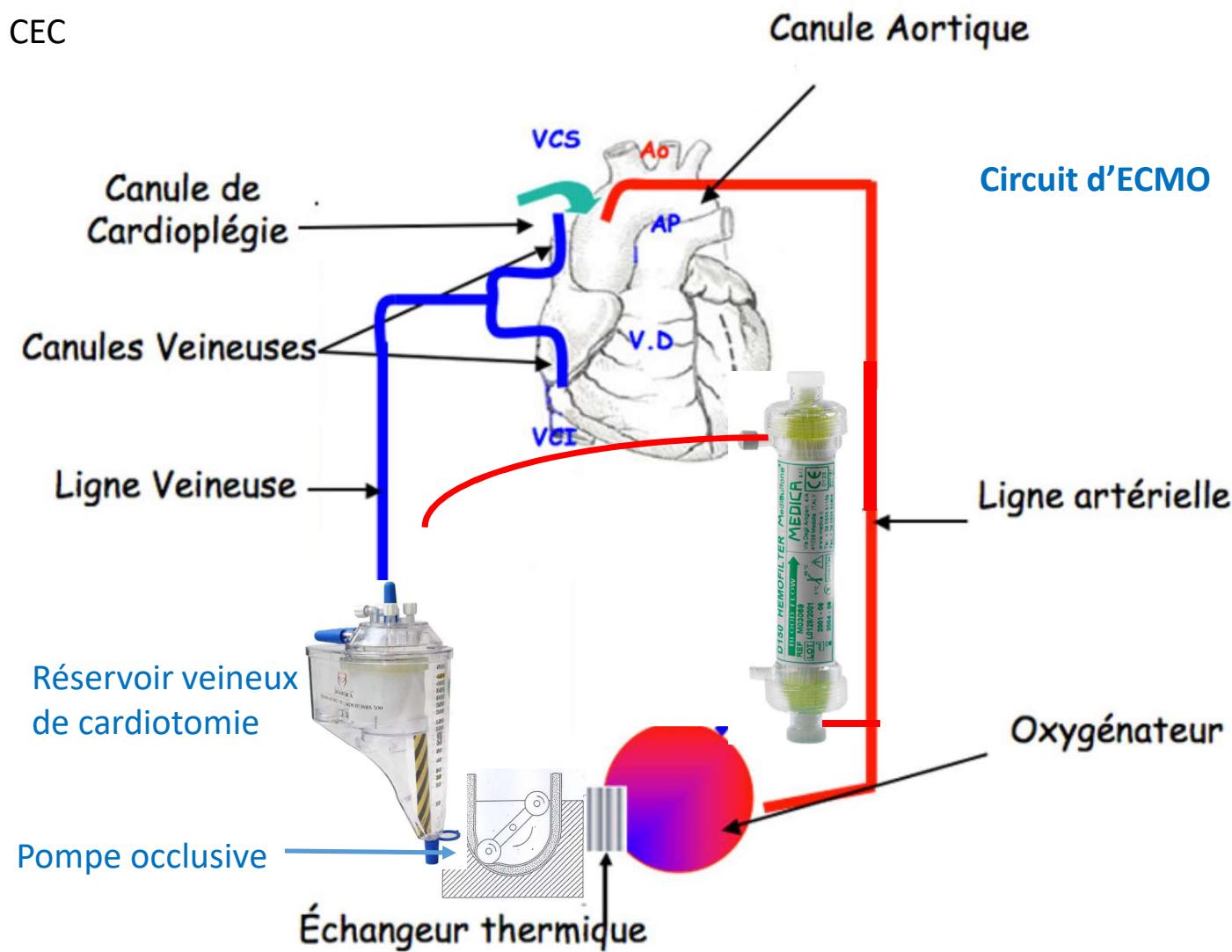
Effets bénéfiques :

- Hémoconcentration isovolumique
- Diminution des médiateurs inflammatoires (IL-8, complément)
- Diminution de l'eau extravasculaire pulmonaire et de la RVP

Désavantages:

- MUF: délai pour l'administration de la protamine , impatience du chirurgien

Le circuit de CEC



Controlled reoxygenation during cardiopulmonary bypass decreases markers of organ damage, inflammation, and oxidative stress in single-ventricle patients undergoing pediatric heart surgery

Traitements anti-inflammatoires

Massimo Caputo, MD,^{a,b} Amir Mokhtari, FRCS,^c Antonio Miceli, MD,^c Mohamed T. Ghorbel, PhD,^c Gianni D. Angelini, FRCS,^c Andrew J. Parry, FRCS,^b and Saadeh M. Suleiman, DSc^c

Objective: Single-ventricle patients undergoing pediatric heart surgery are a high-risk group owing to reoxygenation injury during cardiopulmonary bypass (CPB). The present study investigated the effects of controlled reoxygenation CPB on biomarkers of organ damage, inflammation, stress, and long-term functional outcomes in cyanotic patients with either a single or double ventricle during open heart surgery.

Methods: Cyanotic patients with either a single ($n = 32$) or double ($n = 47$) ventricle undergoing surgical correction were randomized to receive CPB using either standard oxygen levels or controlled reoxygenation. The markers of cardiac injury, inflammation, stress, and cerebral and hepatic injury were measured preoperatively, at 10 and 30 minutes after starting CPB, and at 10 minutes and 4 and 24 hours after CPB. The data were analyzed using a mixed regression model.

Results: No difference was found in the pre- or intraoperative characteristics between the standard and controlled reoxygenation CPB groups for single- or double-ventricle patients. In the single-ventricle patients, controlled reoxygenation CPB significantly ($P < .05$) decreased the markers of organ damage, inflammation, stress, and oxidative stress. In contrast, the markers of inflammation and cardiac injury were not altered by controlled reoxygenation CPB in the double-ventricle patients.

Conclusions: Controlled reoxygenation CPB decreased the markers of organ damage, stress, inflammation, and oxidative stress in single-ventricle patients undergoing cardiac surgery. (J Thorac Cardiovasc Surg 2014;148:792-801)

PaO ₂	Groupe « standard »	Groupe « Réoxygénéation contrôlée »
At start of CPB	167.8 ± 70.2	55.4 ± 13.7
At 5 min of CPB	169.6 ± 61.5	63.7 ± 20.5
At 10 min of CPB	171.8 ± 55.9	65.8 ± 18.2
At 30 min of CPB	155.6 ± 39.0	114.4 ± 29.7
Immediately after CPB	173.2 ± 56.2	159.4 ± 41.1

Particularités du circuit CEC en pédiatrie (miniaturisation)

Hematocrite idéal et transfusion

Température et débit de CEC

Pression de perfusion

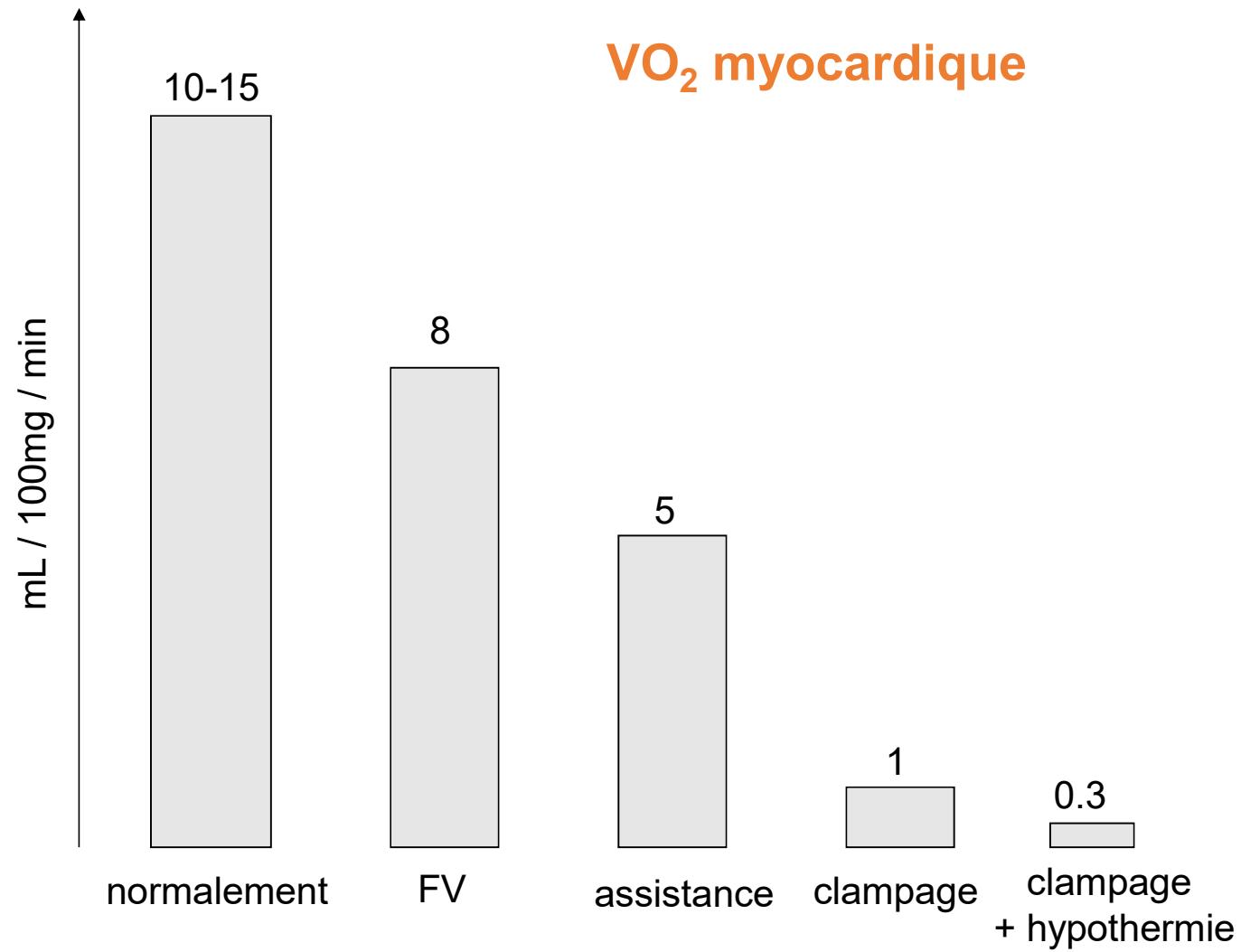
Hypothermie et protection cérébrale

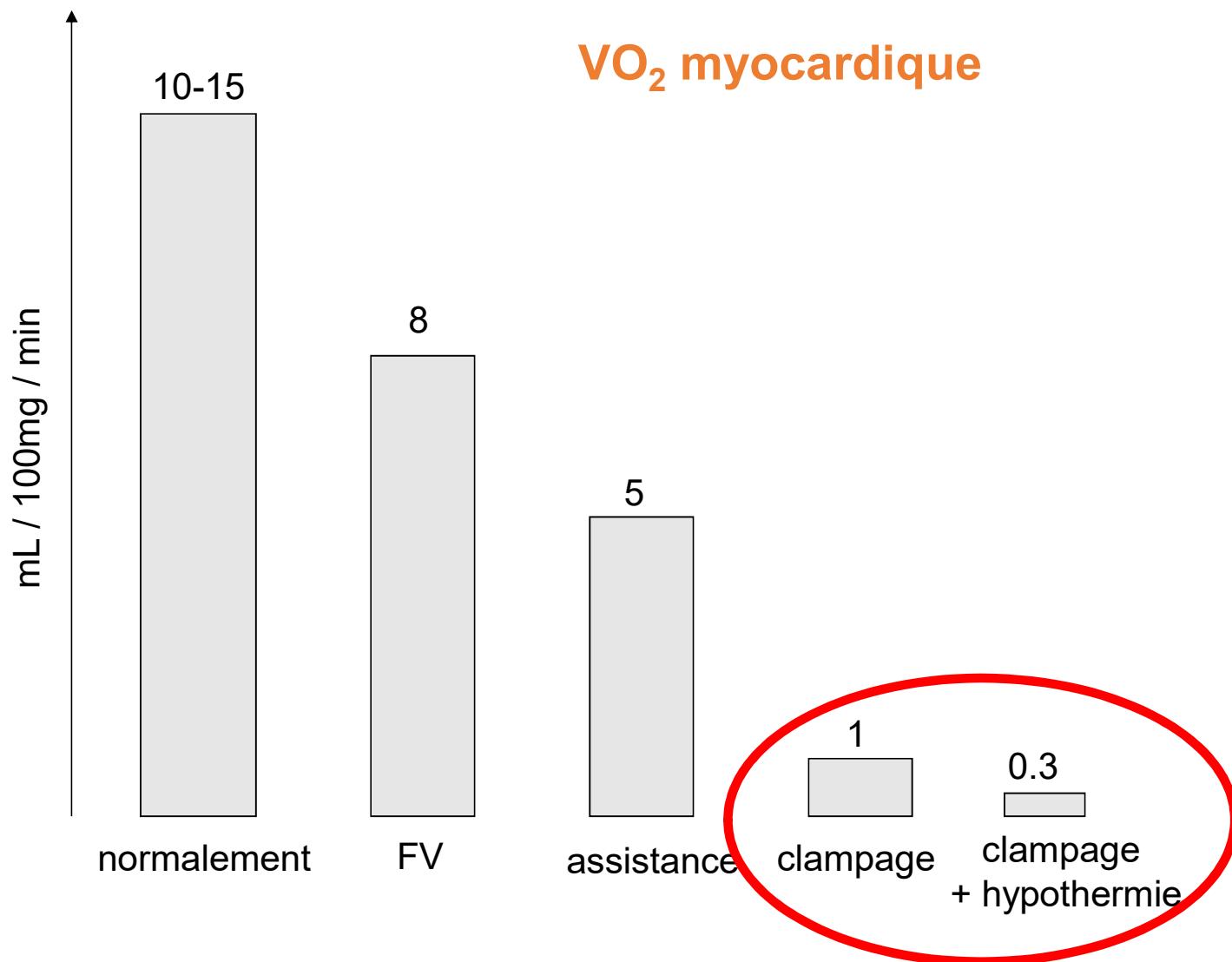
Monitoring

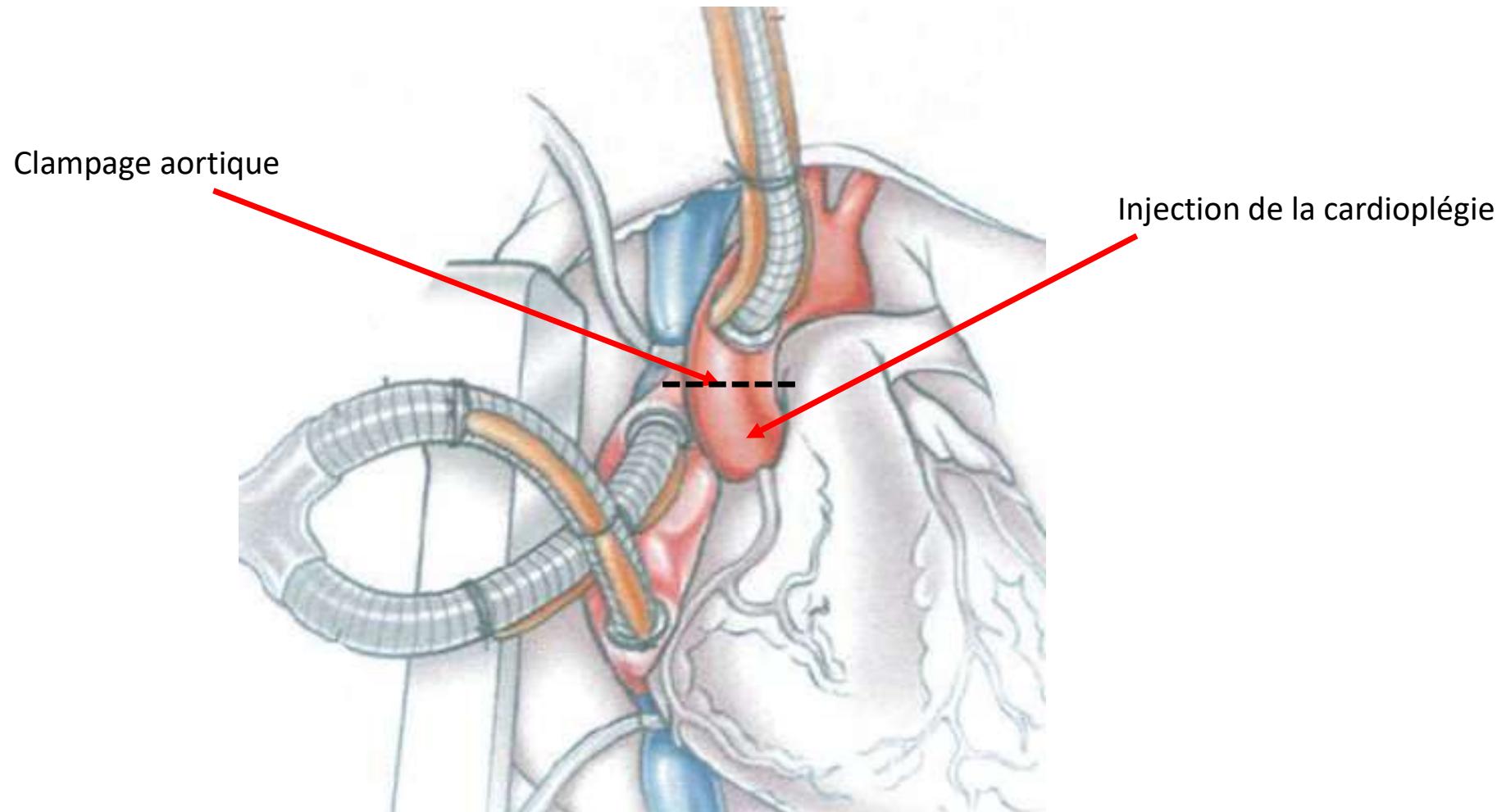
Inflammation

Protection myocardique

Sevrage de la CEC en pédiatrie







Particularités du myocarde néonatal

Fonctionnelles

1. Aucune réserve contractile, augmentation de l'IC au dépend de la fréquence
2. Mauvaise compliance
3. Sensibilité réduite aux catécholamines (nombre réduit de récepteurs à l'adenyl-cyclase)

Métaboliques

1. Utilise quasi-exclusivement du glucose (réserves glycogène)
2. Réticulum sarcoplasmique sous-développé
 - > la capacité de réserve du Ca est réduite, et la contraction dépendante du Ca extracellulaire,
 - > déficit Ca-ATP-ase sarcoplasmique (redirige le Ca vers le réticulum sarcoplasmique) -> susceptibilité à la surcharge en Ca après la période d'ischémie
3. Système anti-radicalaire (supéroxyde dysmutase, catalase, glutathion-reductase) déficitaire
- 4 . *déficit en 5' nucléotidase (conversion AMP en adénoside, qui peut quitter la cellule)*
 - > résistance à l'ischémie
 - ... pas de déficit chez le patient cyanosé ...

Protection myocardique durant la chirurgie cardiaque

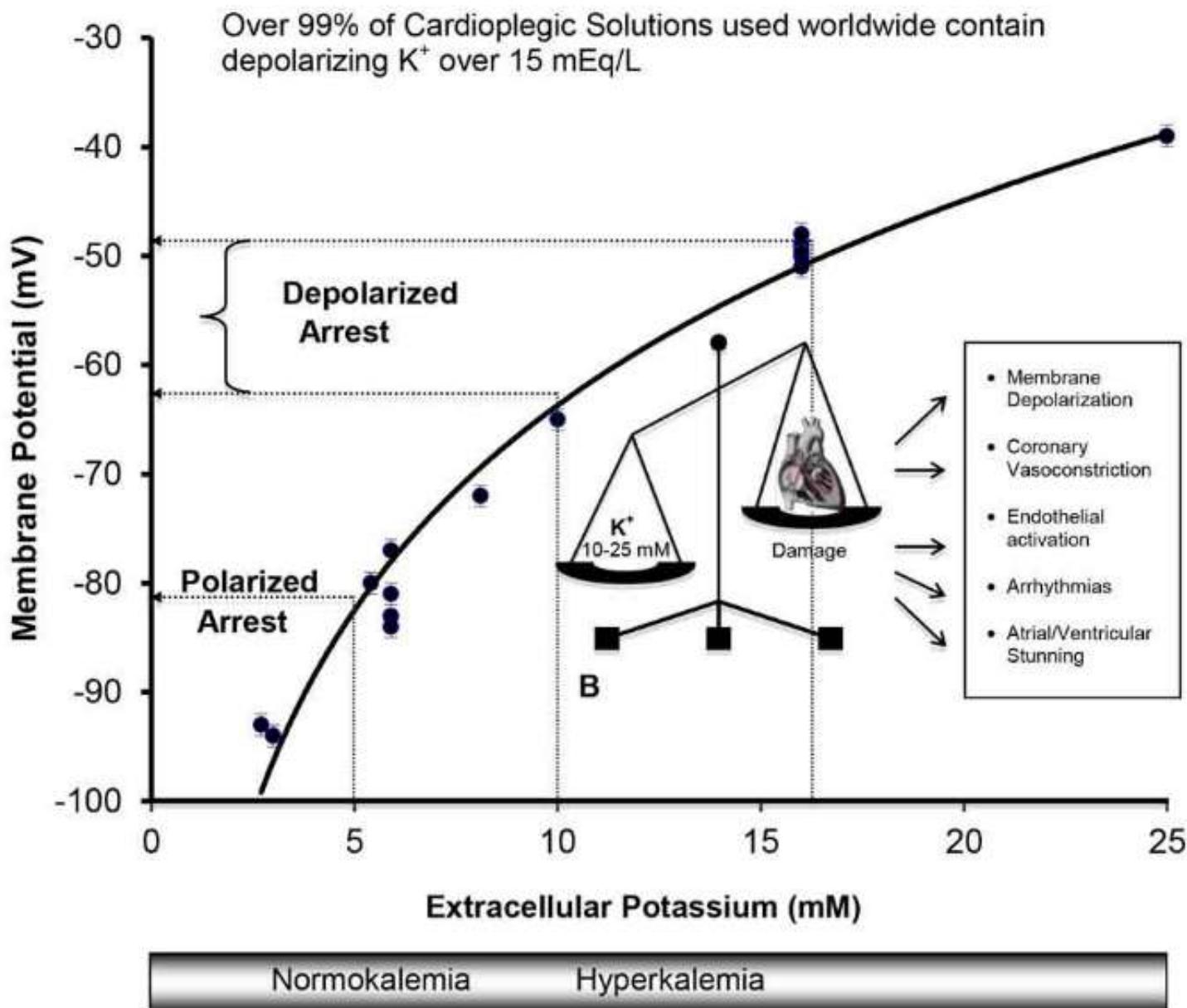
- 1. réduction de la VO₂ myocardique
 - 2. apporter le substrat énergétique nécessaire pour le maintien de l'intégrité cellulaire
 - 3. maintenir le pH intracellulaire
 - 4. prévenir l'œdème cellulaire
 - 5. prévenir les lésions de reperfusion
 - 6. éviction de l'hyperoxygénéation du myocarde cyanosé
 - 7. contrôle de la pression d'injection de la cardioplégie (*Kronon, ATS 1998*)
 - 8. éviction de la distension des cavités lors du rétablissement des retours veineux en fin de CEC
 - 9. homéostasie énergétique, thermique, hydro-éléctrolytique et acido-basique
- }
- choix de la solution de cardioplégie**

167 solutions de cardioplégie (*Demmy, Transplantation 1997*)

224 protocoles de cardioplégie en pédiatrie, dont 86% au sang et 93% froide
(*Kotani, ATS 2013*)

au sang / cristalloïde
chaude / froide
chaude puis froide / froide puis *hot shot*
continue / intermittente
antérograde / rétrograde
a composition intracellulaire / extracellulaire

?



Dobson, *Frontiers Physiol* 2013

Table 10. Cardioplegia solutions used in 2010 by region.

	NA (n = 89) (%)	CSA (n = 14) (%)	Asia (n = 10) (%)	OA (n = 5) (%)	EU (n = 28) (%)	Total (n = 146) (%)
Hyperpolarizing (Custodiol® HTK)*	4	23	0	0	31	10
Depolarizing (high potassium)	64	77	89	100	62	68
Modified depolarizing (del Nido)†	32	0	11	0	8	22

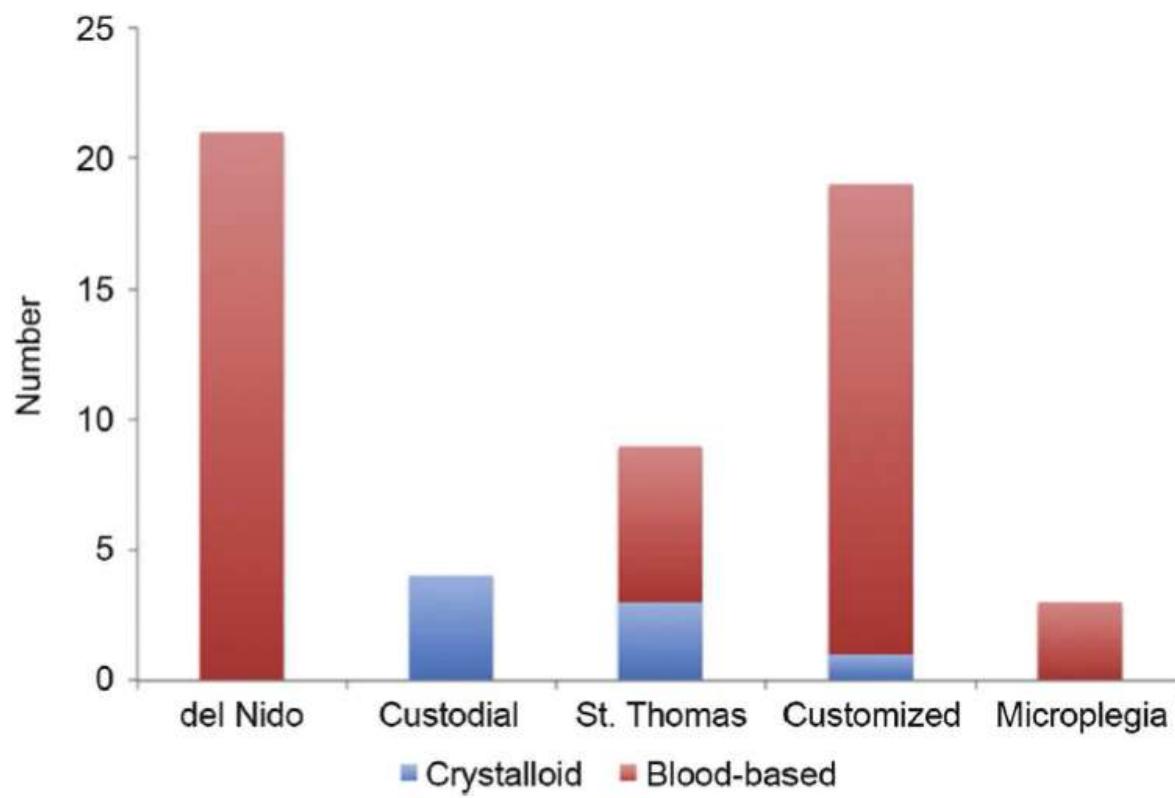
*Custodiol® HTK Solution (Dr. Franz Köhler Chemie GmbH, Alsbach-Hähnlein, Germany).

†Original patent University of Pittsburgh now expired. Composition (8).

NA, North America; CSA, Central and South America; OA, Oceania; EU, European Union.

Harvey, JECT 2012

*Fig 1. Type of cardioplegia and solutions.
The red bar represents blood-based
cardioplegia and the blue bar represents
crystalloid cardioplegia.*

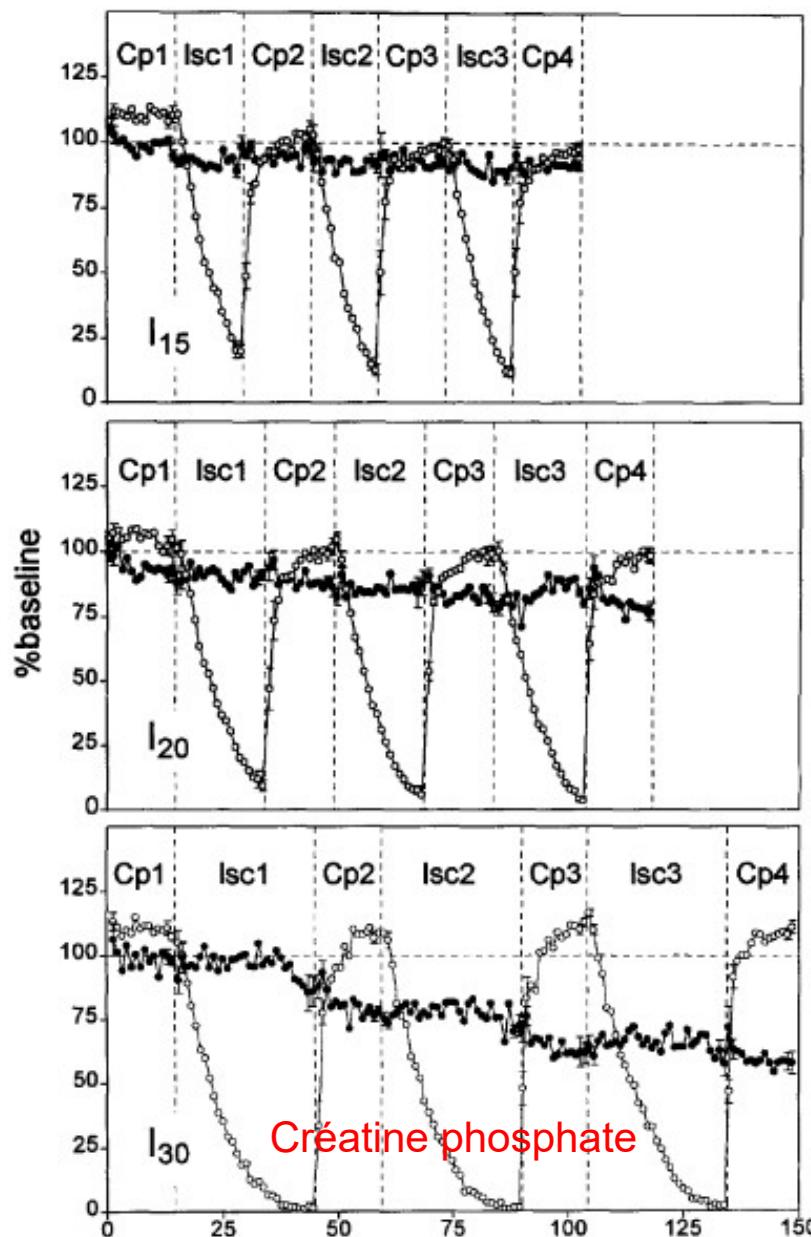


Kotani, ATS 2013

Cardioplégie chaude / froide au sang / cristalloïde

	Chaud au sang	Froide au sang	Froide cristalloïde
Hémodilution	-	++	
Apport O ₂	++	±	-
Inhibiteurs ROS	+	+	±
Pouvoir tampon	++	++	±
Contrôle t°C myocarde	+	- surtout si collatérales	
Substrat nutritif	+	+	±
Réinjections	++	±	

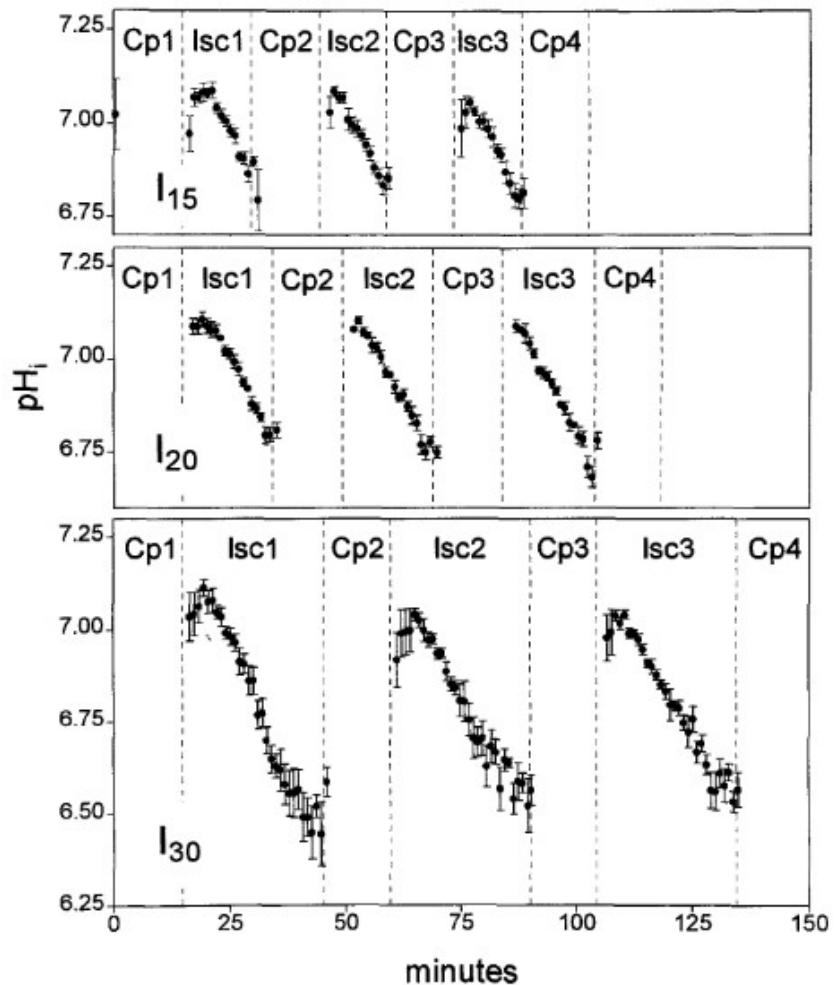
Cardioplégie chaude: intervalle des réinjections



ATP

De Oliveira, JTCVS 1997

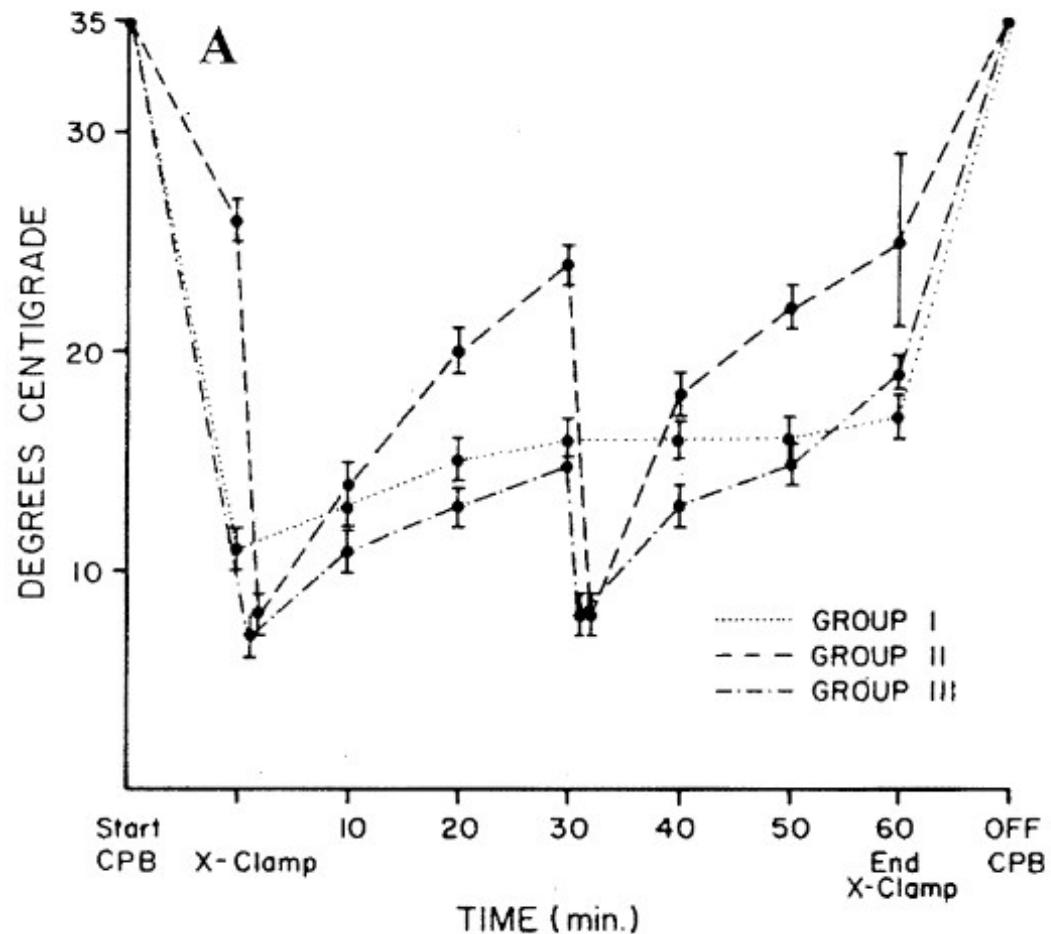
Cardioplégie chaude: intervalle des réinjections



De Oliveira, JTCVS 1997

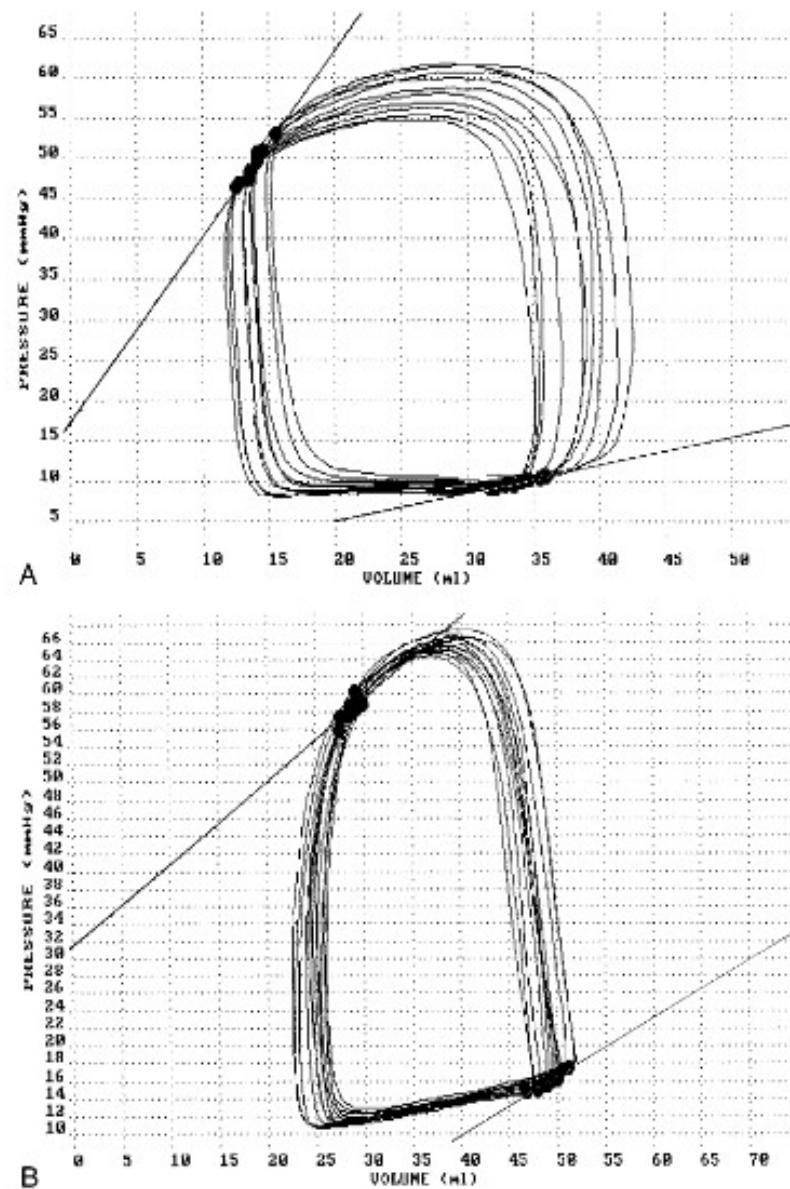
Chez l'adulte: temps d'ischémie chaude <13min (Lichtenstein, Circulation 1995)

Cardioplégie froide: réchauffement du myocarde



Groupe I : CEC 15°C
Groupe II : CEC 28°C, cardioplégie 4°C
Groupe III: CEC 15°C, cardioplégie 4°C

Ganzel, JTCVS 1988
(10 porcelets)



Stunning = phénomène obligatoire après ischémie-reperfusion ?

fermeture CIA !

Chaturvedi, JTCVS 1998

Choix du critère d'évaluation de la qualité de la cardioplégie:

DC ? Infarctus postop ? Durée de séjour ?

Bas débit postop ?

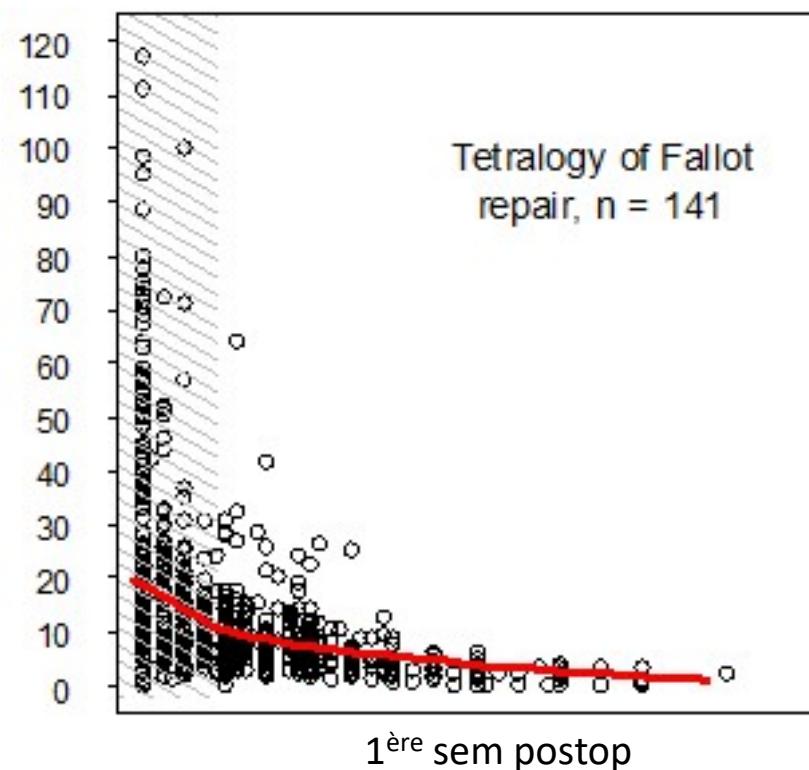
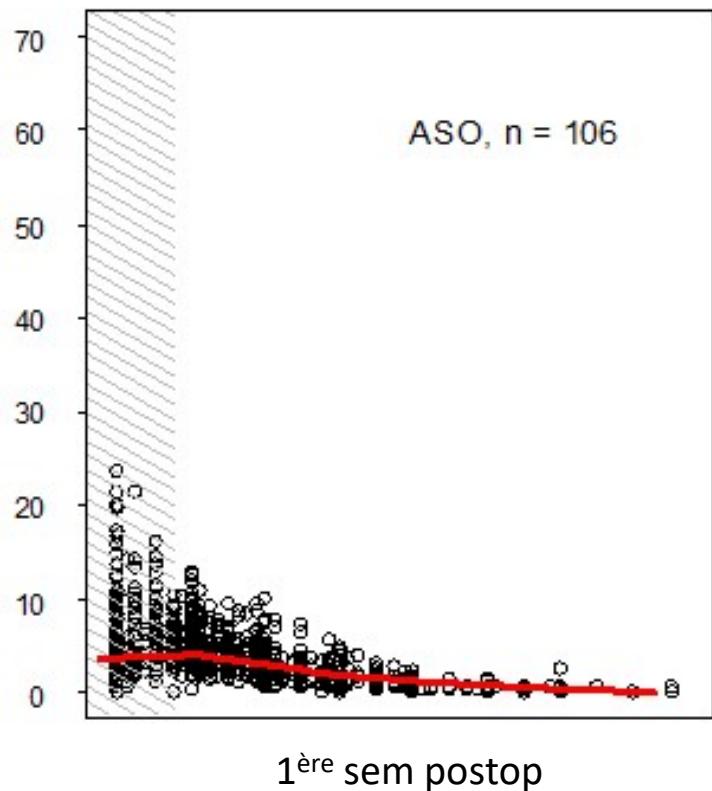
Dysfonction myocardique postop?

Élévation postop des enzymes cardiaques ?

chez l'enfant: > 5.9ng/mL associé au DC à 30 jours (*Mildh, ATS 2006*)

> 100ng/mL = forte prédiction du DC postop (*Immer, JACC 1999*)

L'élévation postop des enzymes cardiaques chez l'enfant dépend du type de chirurgie



Bojan, JTCVS 2012

Il n'y a pas de démonstration formelle de la supériorité d'un cardioplégie par rapport à une autre en pédiatrie

Comparaison chaude au sang / cristalloïde froide:

- *Rasmussen JCVA 2001*, n = 20 nourrissons 6.5 mois, randomisée, équivalence
- *Caputo, ATS 2005*, n = 59 enfants 6.5 ans, randomisée, troponine ↑ avec cristalloïde
- *Pouard, EJTCS 2006*, n = 40 ASO, rétrospective, équivalence
- *Poncelet EJCTS 2011*, n = 47 nourrissons 19 mois, randomisée, ATP ↑ avec chaude
- *Bojan, ATS 2013*, n = 218 ASO, rétrospective, troponine ↑ avec cristalloïde

Remote Ischemic Preconditioning in Children Undergoing Cardiac Surgery With Cardiopulmonary Bypass: A Single-Center Double-Blinded Randomized Trial

Brian W. McCrindle, MD, MPH; Nadia A. Clarizia, MSc; Svetlana Khaikin, RN, MPH; Helen M. Holtby, MD; Cedric Manlihot, BSc; Steven M. Schwartz, MD; Christopher A. Caldarone, MD; John G. Coles, MD; Glen S. Van Arsdell, MD; Stephen W. Scherer, PhD; Andrew N. Redington, MD

Background—Remote ischemic preconditioning (RIPC) harnesses an innate defensive mechanism that protects against inflammatory activation and ischemia-reperfusion injury, known sequelae of cardiac surgery with cardiopulmonary bypass. We sought to determine the impact of RIPC on clinical outcomes and physiological markers related to ischemia-reperfusion injury and inflammatory activation after cardiac surgery in children.

Methods and Results—Overall, 299 children (aged neonate to 17 years) were randomized to receive an RIPC stimulus (inflation of a blood pressure cuff on the left thigh to 15 mm Hg above systolic for four 5-minute intervals) versus a blinded sham stimulus during induction with a standardized anesthesia protocol. Primary outcome was duration of postoperative hospital stay, with serial clinical and laboratory measurements for the first 48 postoperative hours and clinical follow-up to discharge. There were no significant baseline differences between RIPC ($n=148$) and sham ($n=151$). There were no in-hospital deaths. No significant difference in length of postoperative hospital stay was noted (sham 5.4 versus RIPC 5.6 days; difference +0.2; adjusted $P=0.91$), with the 95% confidence interval (−0.7 to +0.9) excluding a prespecified minimal clinically significant differences of 1 or 1.5 days. There were few significant differences in other clinical outcomes or values at time points or trends in physiological markers. Benefit was not observed in specific subgroups when explored through interactions with categories of age, sex, surgery type, Aristotle score, or first versus second half of recruitment. Adverse events were similar (sham 5%, RIPC 6%; $P=0.68$).

Conclusions—RIPC is not associated with important improvements in clinical outcomes and physiological markers after cardiac surgery in children.

Clinical Trial Registration—URL: clinicaltrials.gov. Unique identifier: NCT00650507. (*J Am Heart Assoc.* 2014;3:e000964 doi: 10.1161/JAHA.114.000964)

Particularités du circuit CEC en pédiatrie (miniaturisation)

Hematocrite idéal et transfusion

Température et débit de CEC

Pression de perfusion

Hypothermie et protection cérébrale

Monitoring

Inflammation

Cardioplégie

Sevrage de la CEC en pédiatrie

Sevrage de la CEC

= début de la réanimation postopératoire

Décision collégiale entre le chirurgien, l'anesthésiste et le perfusioniste

Après retour à l'homéostasie thermique ($> 36^{\circ}\text{C}$), ionique, acido-basique

Après reprise d'une ventilation efficace

Hématocrite adapté à l'âge et à la pathologie (nouveau-né 42-45%)

Inotropes en route

Produits de remplissage disponibles

Les 3 principales raisons du sevrage difficile

- 1. Lésion résiduelle (ETO, saturations étagées si suspicion de shunt résiduel)**
- 2. HTAP**
- 3. Défaillance myocardique**

Les 3 principales raisons du sevrage difficile

- 1. Lésion résiduelle**
- 2. HTAP**
- 3. Défaillance myocardique**

L'immaturité pulmonaire du nouveau-né

Résistances vasculaires pulmonaires élevées à la naissance, diminuent rapidement pour arriver au niveau des RVP de l'adulte à l'âge de 2 sem

Heyman, Prog Cardiovasc Dis 1972

Le nombre d'alvéoles pulmonaires augmente x 10 jusqu'à l'âge de 8 ans

Vidal Melo, Int Anesth Clin 2004

Altérations pulmonaires « obligatoires » à la fin de la CEC

Lésions d'ischémie-reperfusion

Transfusion massive

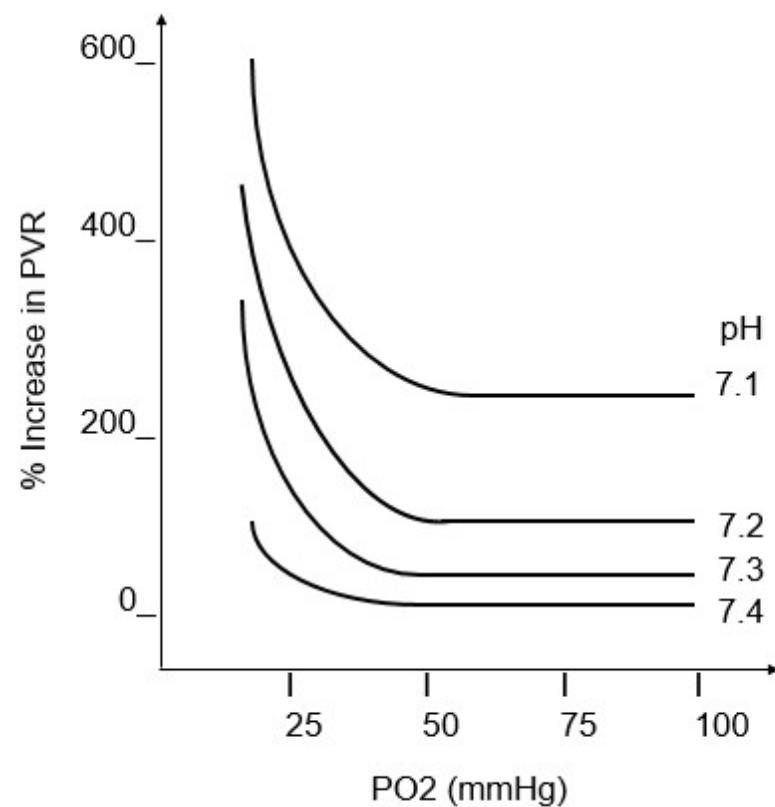
Augmentation de l'eau extra-vasculaire pulmonaire (syndrome inflammatoire, fuite capillaire)

- diminution de la compliance pulmonaire
- augmentation des résistances vasculaires pulmonaires
- risque accru d'interactions cardio-pulmonaires à la fermeture sternale

Prévenir / traiter une HTAP post-CEC

1. Maintenir le volume pulmonaire au niveau de la CRF
2. Maintenir le pH > 7.4
3. Maintenir la paO₂ à environ 120 mmHg
4. Normocapnie paCO₂ 35-45 mmHg
5. Vasodilatateurs pulmonaires inhalés ou IV

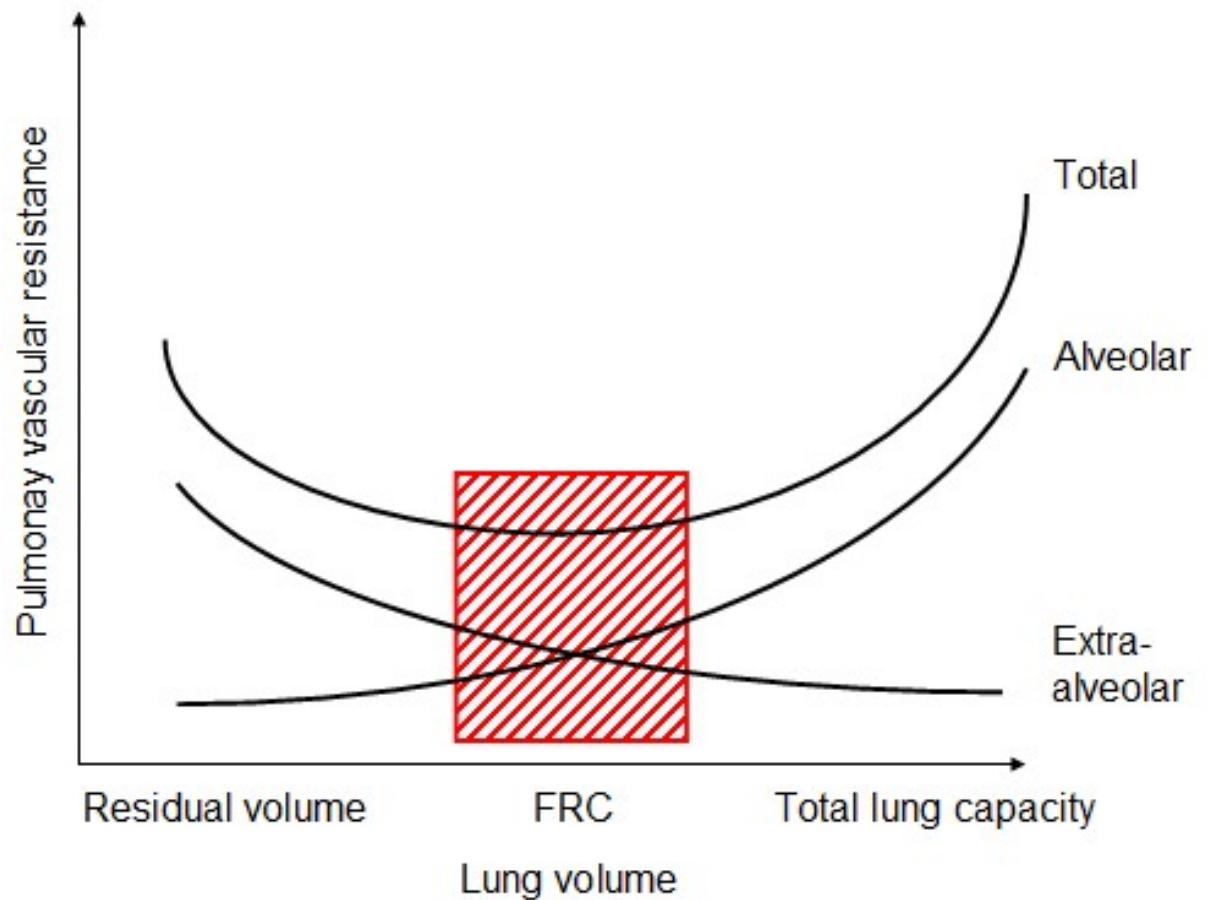
Homéostasie acido-basique et résistances vasculaires pulmonaires



Rudolph, J Clin Invest 1966

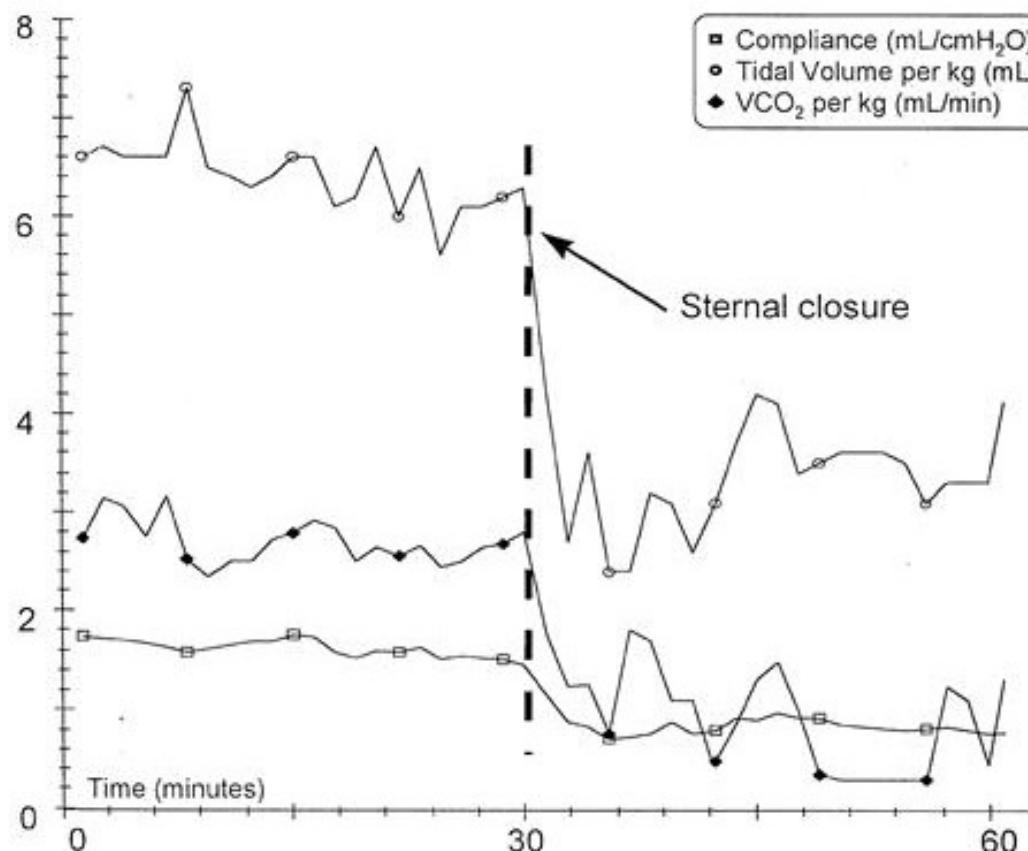
maintien du volume pulmonaire au niveau de la CRF

As lung volume increases from residual volume to total lung capacity, the alveolar vessels become increasingly compressed by the distending alveoli, and their resistance increases, whereas the resistance of the extra-alveolar vessels (which become less tortuous as lung volume increases) falls. The combined effect of increasing lung volume on the pulmonary vasculature produces the typical “U shaped” curve, with its nadir at around normal functional residual capacity (FRC).



Shekerdemian, Arch Dis Child 1999

La compliance thoracique diminue lors de la fermeture sternale

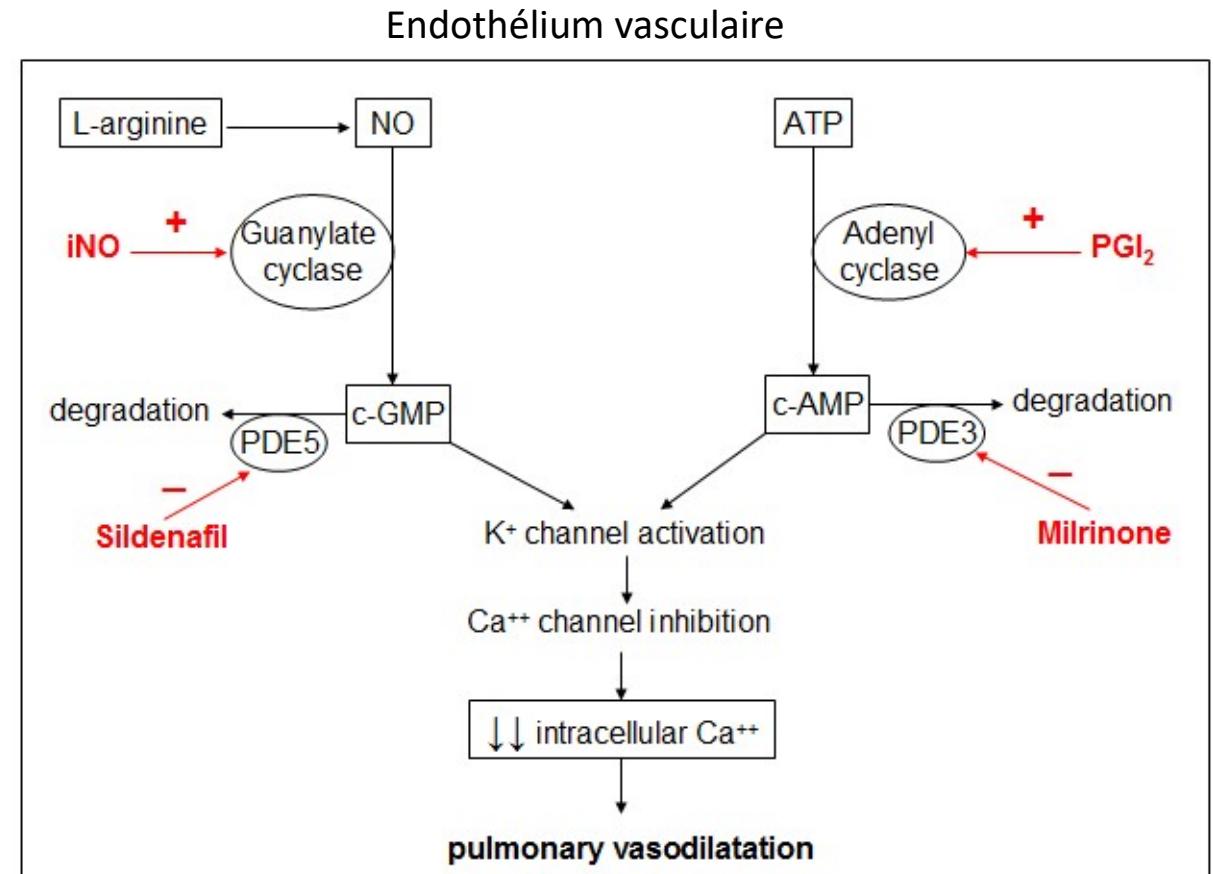


Main, CCM 2001



Les vasodilatateurs pulmonaires

- Dans l'endothélium pulmonaire PDE5 > PDE3
- NOi est un vasodilatateur pulmonaire sélectif ++
- Sildenafil : risque d'effet shunt en cas d'atélectasie
- Epoprostenol IV : effet vasodilatateur pulmonaire et systémique
- Iloprost inhalé est un vasodilatateur pulmonaire sélectif ++, bonne alternative pour le NOi



Ghofrani, JACC 2004

Les 3 principales raisons du sevrage difficile

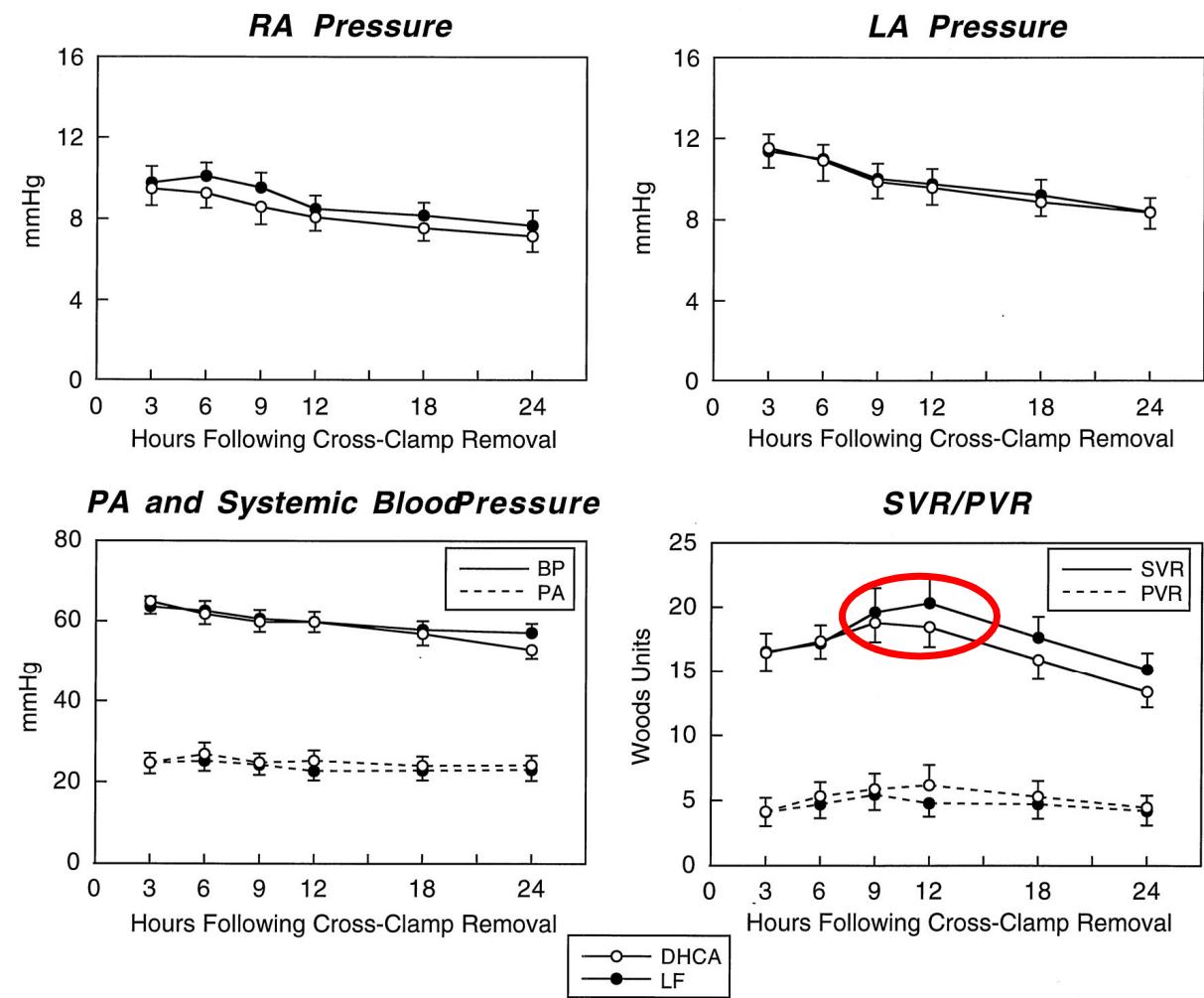
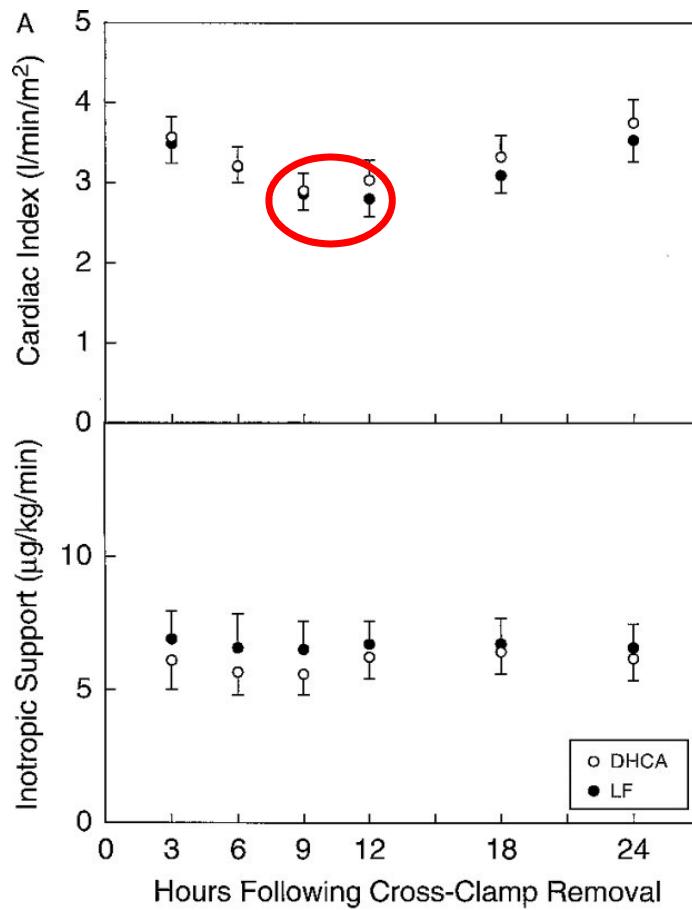
1. Lésion résiduelle
2. HTAP
3. Défaillance myocardique

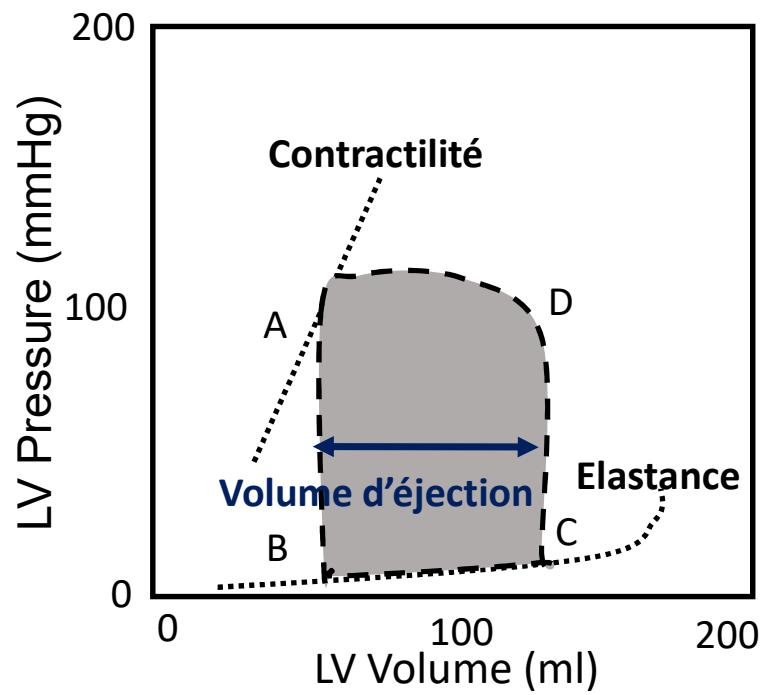
Prévention / traitement d'une défaillance ventriculaire post-CEC

1. Bonne protection myocardique
2. Diminution de la postcharge (vasodilatateurs systémiques, pulmonaires, éviter les interactions cardio-pulmonaires, fermeture sternale retardée)
3. Augmentation de l'inotropisme
4. L'assistance circulatoire

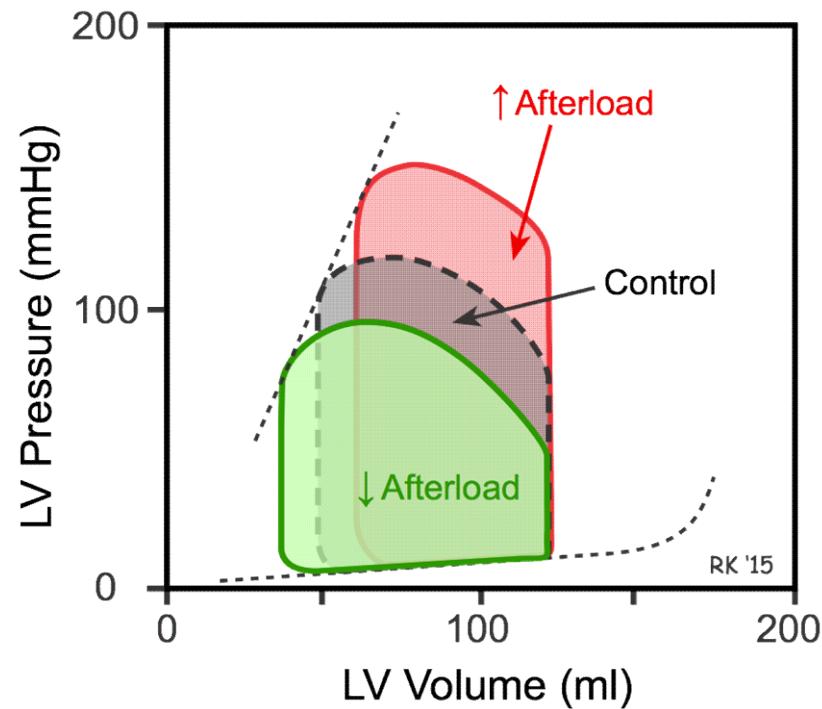
Wernovsky, Circulation 1995

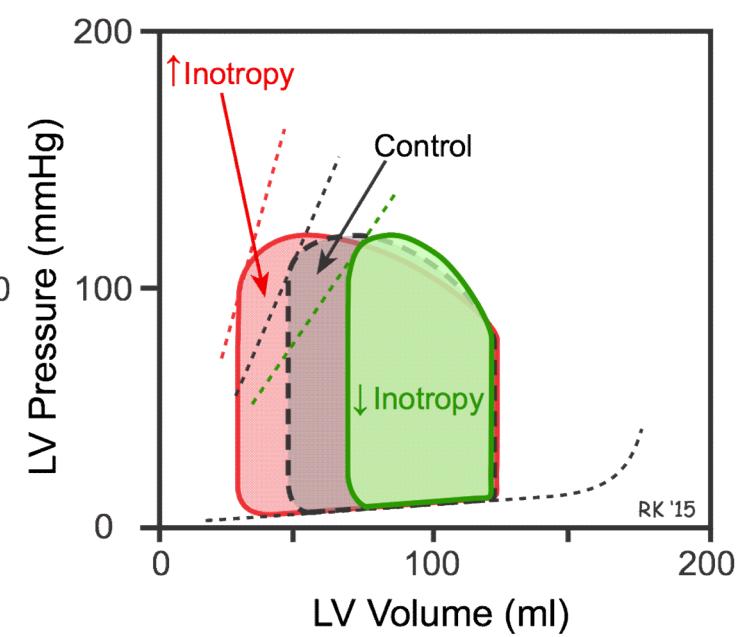
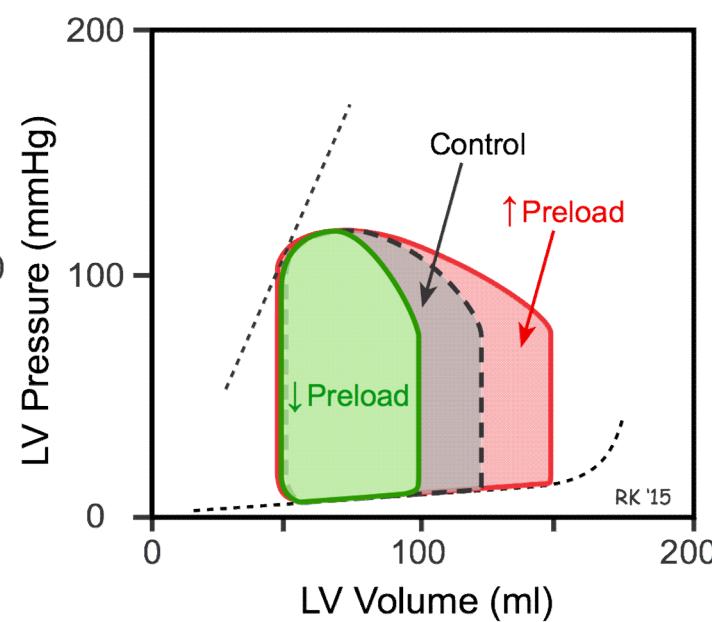
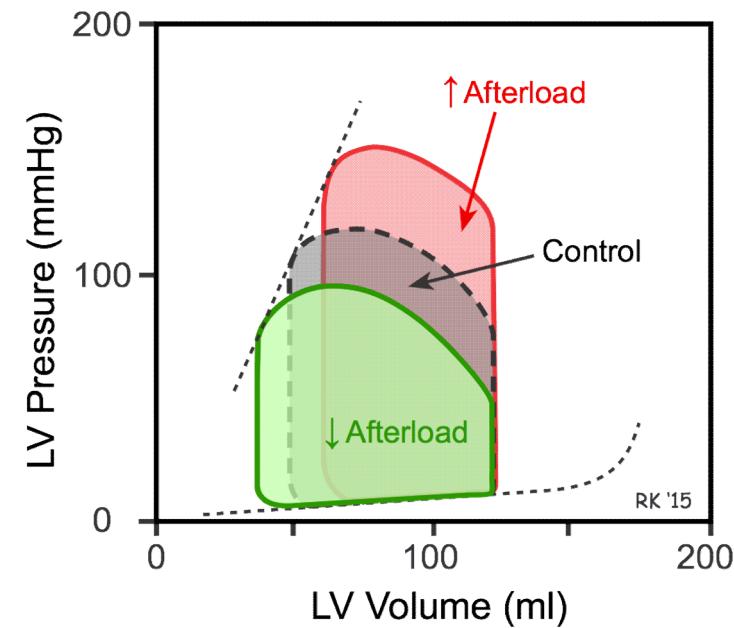
Les résistances vasculaires systémiques augmentent toujours après chirurgie cardiaque pédiatrique



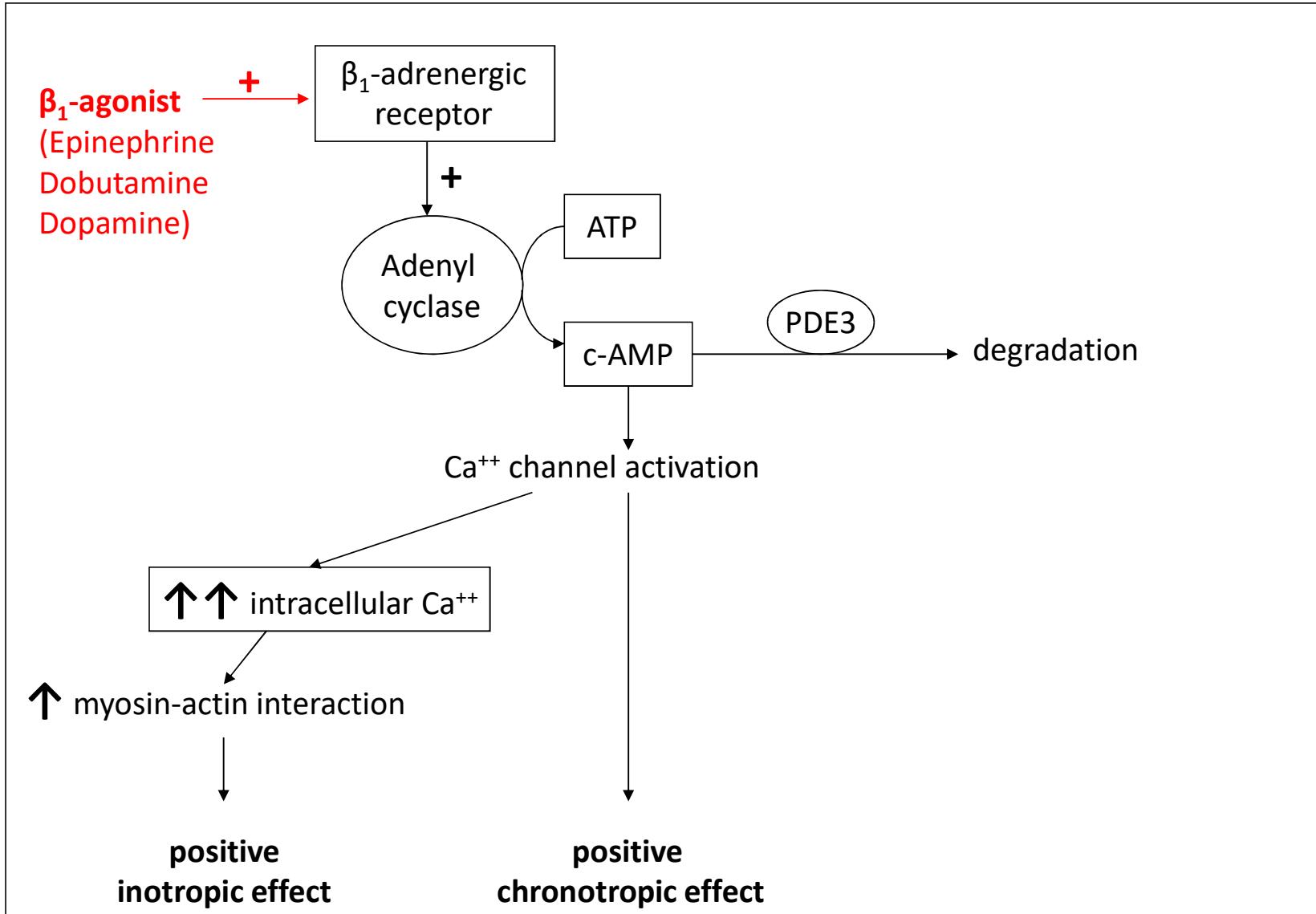


A: fermeture valve Ao, B: ouverture valve mitrale,
C: fermeture valve mitrale, D: ouverture valve Ao

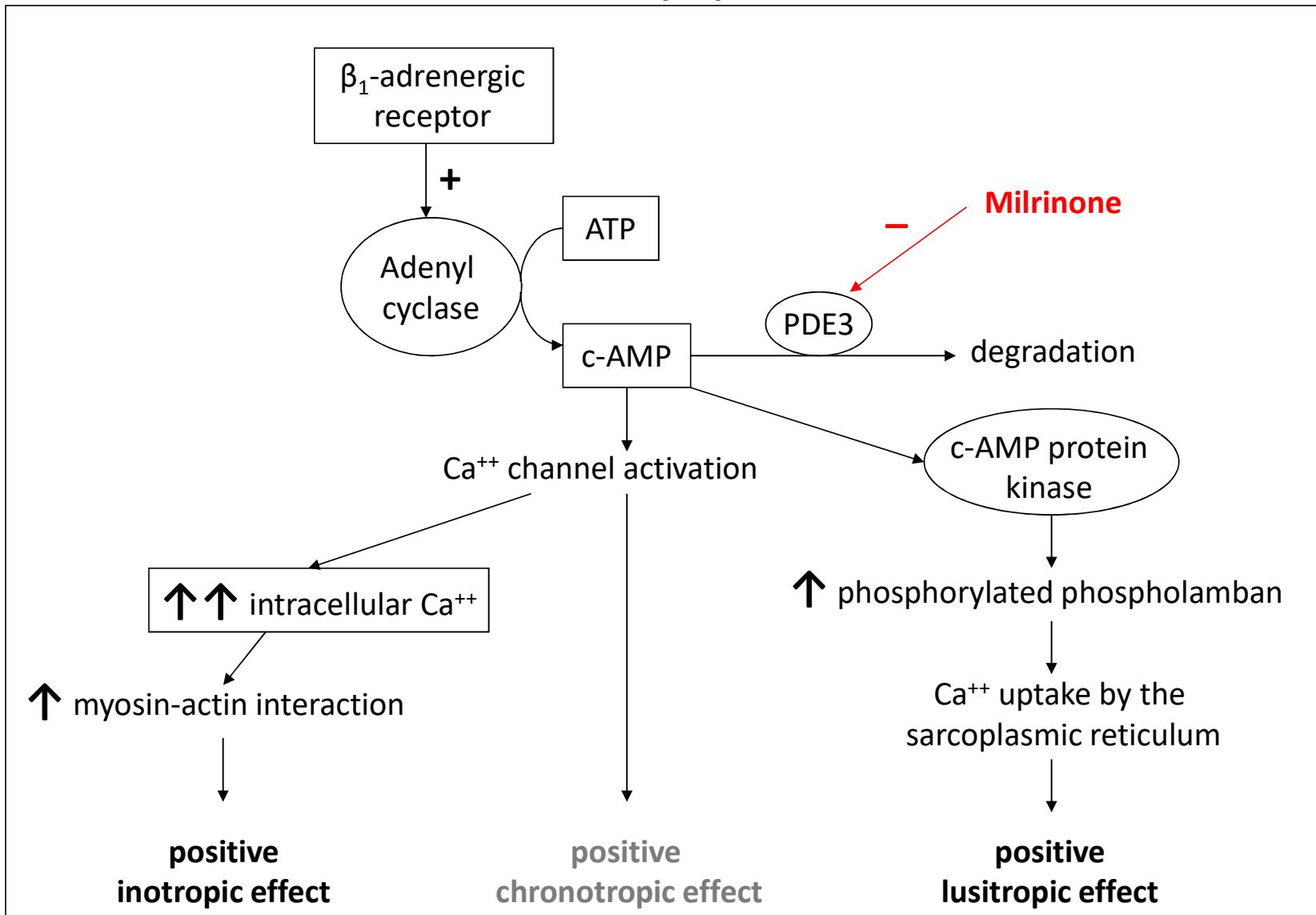




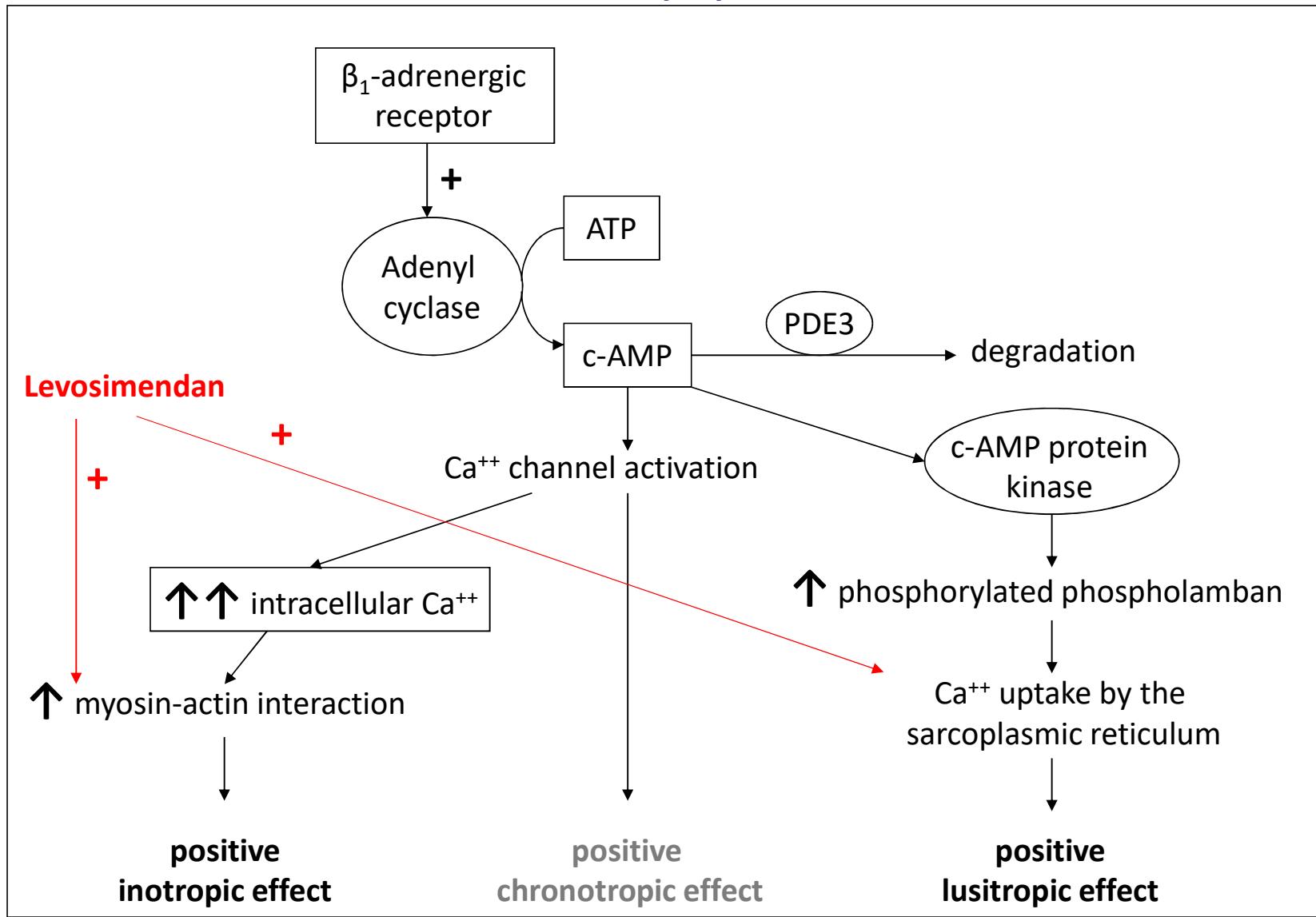
Cardiomyocyte



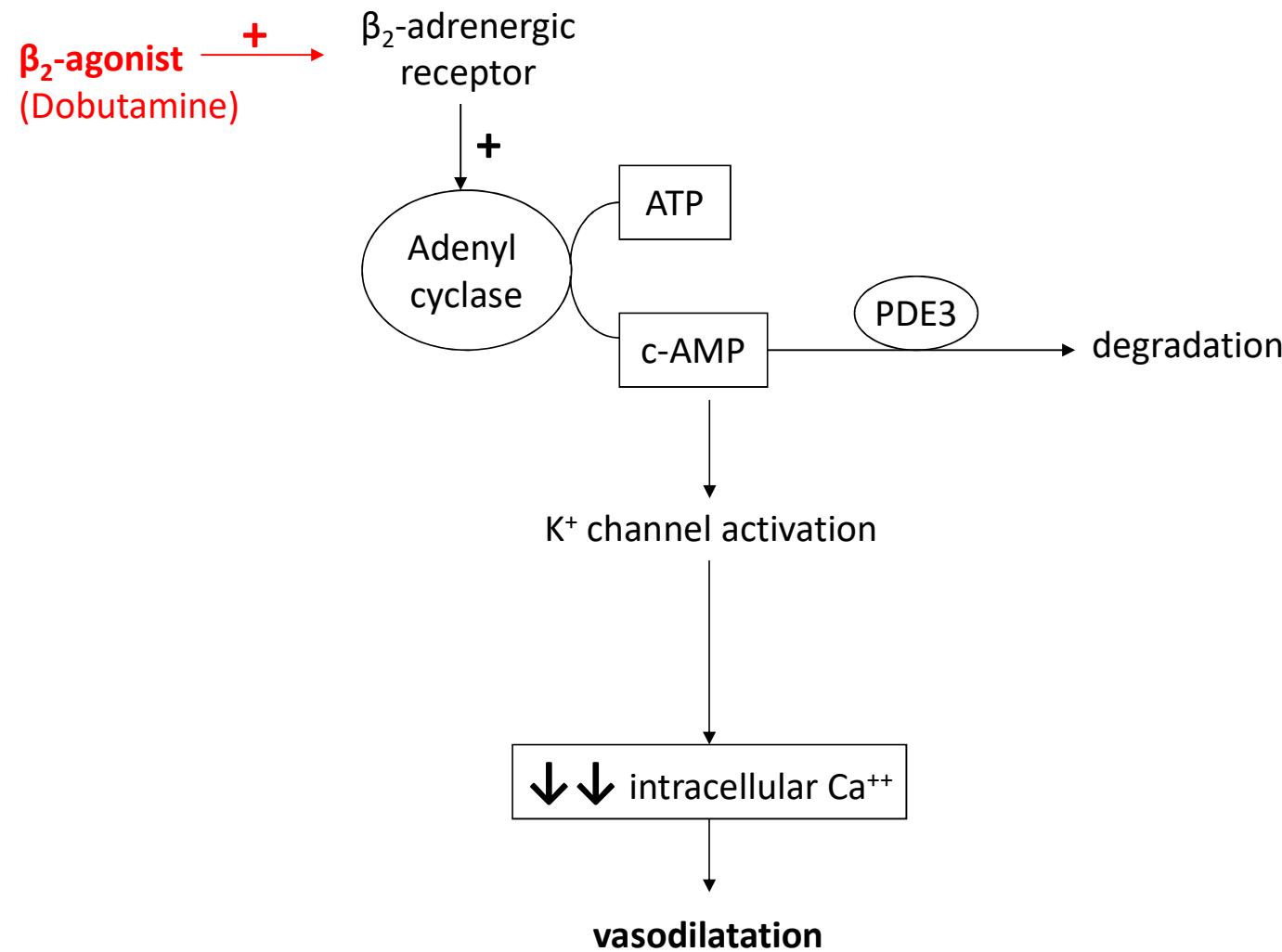
Cardiomyocyte



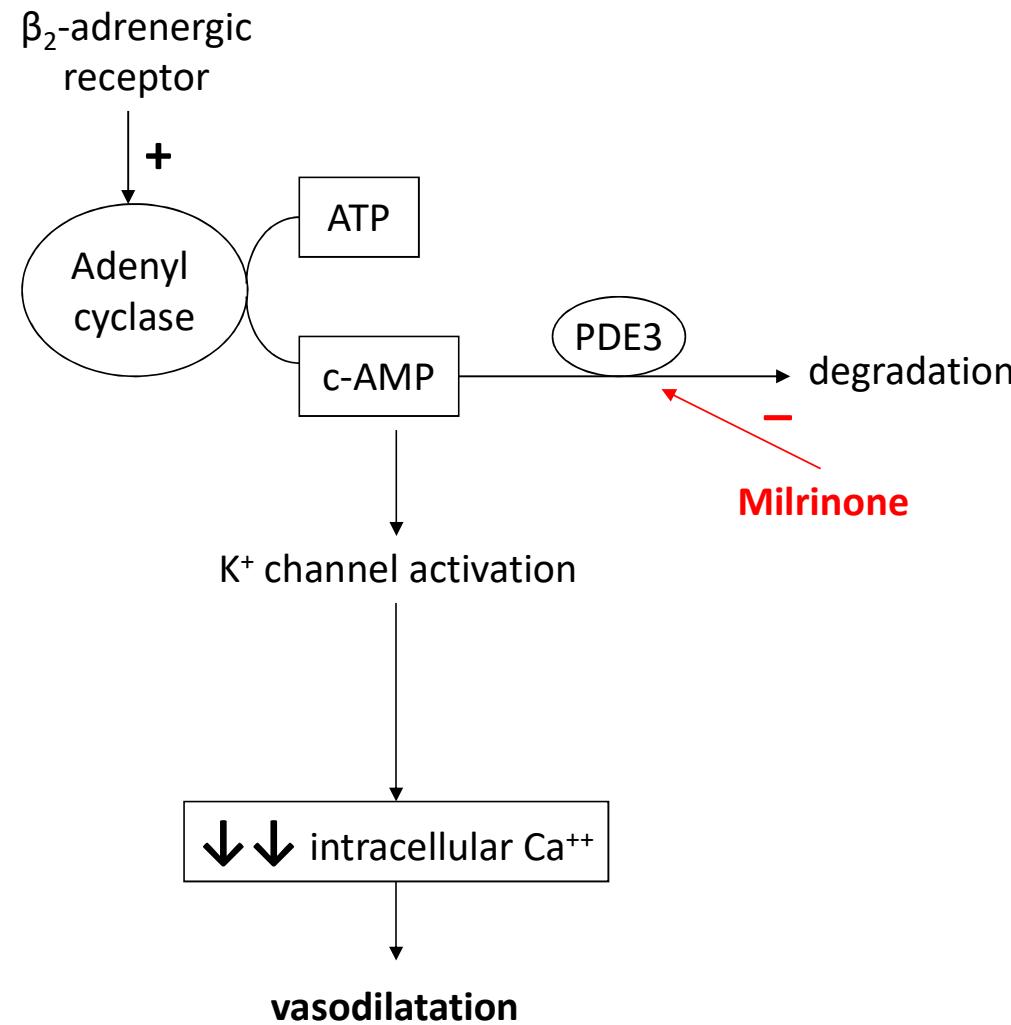
Cardiomyocyte



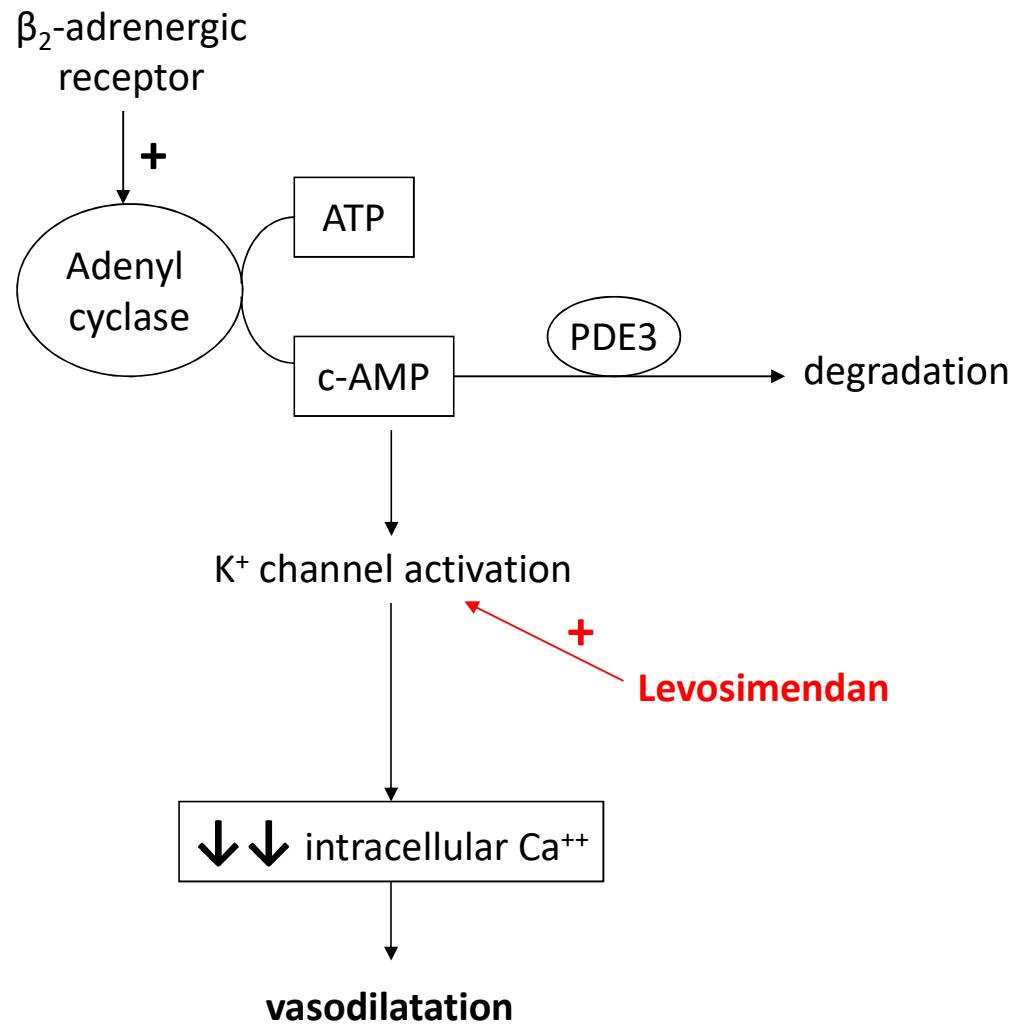
Vascular smooth muscle



Vascular smooth muscle



Vascular smooth muscle



Vasodilatateurs systémiques et inotropes

- Tous les agonistes β_1 augmentent le Ca intracellulaire et la VO₂ myocardique
- Immaturité des récepteurs β_1 chez le nouveau-né
- Tous les agonistes β_1 augmentent la VO₂ globale du fait de leurs effets systémiques (thermogénèse dans la graisse brune, stimulation de la glycolyse)
- Le levosimendan et la milrinone n'augmentent pas la VO₂ myocardique

Prévention du bas débit post-CEC : drogues vaso-actives pour la

Enquête EuLoCOS-Paed
90 centres / 30 pays d'Europe

	<i>n</i> (risk/all) ^a	(%)
Mode of drug use		
Single-drug regimen	31 (25/6)	41.3
Two-drug regimen	43 (36/7)	57.3
Triple-drug regimen	1 (1/0)	1.3
Drug regimens ^b		
Milrinone	19 (15/4)	25.3
Milrinone + dopamine	9 (6/3)	12.0
Milrinone + epinephrine	8 (7/1)	64% 64%
Milrinone + dobutamine	7 (6/1)	9.3
Milrinone + levosimendan	5 (5/0)	6.7
Levosimendan	5 (5/0)	6.7

Enquête PCICS (Pediatric Cardiac Intensive Care Society)
62 centres, dont 70% Nord-Américains

Prophylactic drug regimen	N	%
Milrinone	90/93	97
Adrenaline/epinephrine	42/93	45
Dopamine	35/93	38
Dobutamine	10/93	11
Levosimendan	5/93	5
Other	10/93	11

Vogt, Ped Anesth, 2011

Roeveld, WJ Ped Congenital Heart Surg, 2018

Fermeture sternale retardée



Diminue les interactions cardio-pulmonaires en cas de :

- Défaillance biventriculaire refractaire (adrénaline > 0.2 µg/kg/min)
- Compliance pulmonaire effondrée
- Ischémie myocardique (troponine)
- Faible poids (<3 kg)
- CEC longue (> 2 h)
- HTAP préopératoire

Fermeture en chambre

Antibioprophylaxie par C2G

Pas plus d'infections du site opératoire

Assistance de courte durée

- De type ECMO
- Inotropes maintenues à la dose minimale qui assure une contraction du myocarde et l'ouverture de la valve aortique
- 50 – 80% du débit théorique

En attente de récupération / de ventricule pneumatique / de transplantation

En conclusion

CEC néonatale =

« N » techniques de CEC et de cardioplégie

3 causes principales pour expliquer les difficultés du sevrage

3 inotropes majeurs

« N » raisons pour laisser le thorax ouvert