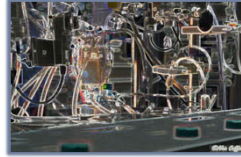
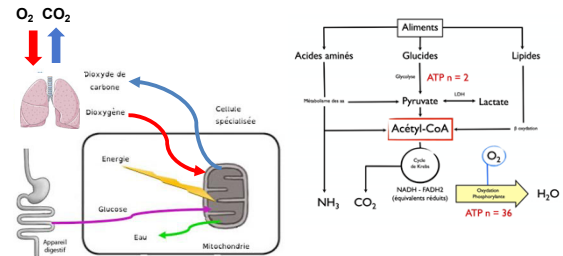


Echanges gazeux appliqués à la CEC

Prof. Alexandre OUARTARA
Service d'Anesthésie-Réanimation cardiovasculaire
Hôpital Haut-Lévêque hospital
33600 Pessac, France
E-mail: alexandre.ouattara@chu-bordeaux.fr



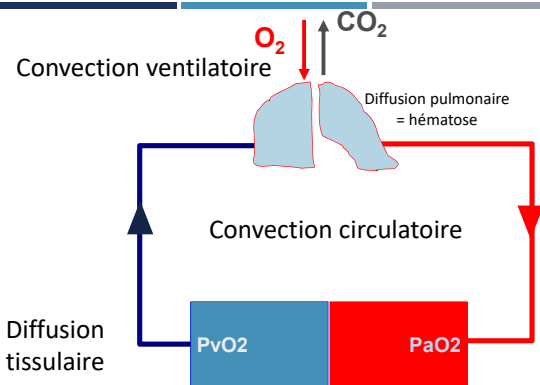
Respiration cellulaire



PRIMUM MOVENS CELLULAIRE

Les échanges gazeux entre l'air ambiant et la cellule sont assurés par...

LA POMPE CARDIO-THORACIQUE



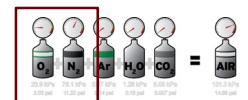
PHASE GAZEUSE => LOI DE DALTON (LOI DES PRESSIONS PARTIELLES)

- Tout gaz d'un mélange exerce une pression partielle proportionnelle à sa fraction (assimilée à son pourcentage)
- Pression partielle P d'un gaz donné « x » est égale au produit de la pression totale, P_T , par la fraction F_x de ce gaz dans le mélange
- Somme des pressions partielles est égale à la pression totale du mélange

$$P_x = P_T \times F_x \leftrightarrow P_x = P_B \times F_x$$

Composition de l'air ambiant

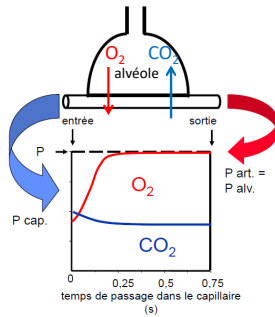
Gaz	Fraction (%)	Pression partielle (mmHg)
N ₂	78.6	597
O ₂	20.9	159
CO ₂	0.04	0.3
H ₂ O	0.46	3.7
Total	100%	760



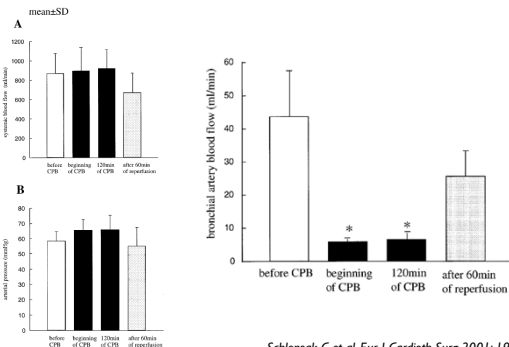
$$P_{Gaz} = \text{Fraction} \times P_{atmosphérique}$$

$$P_{ATM} = 760 \text{ mmHg au niveau de la mer}$$

Temps de transit dans la barrière alvéolo-capillaire (effet PvO₂)

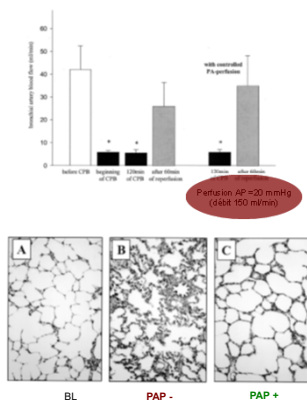


Bronchial artery perfusion during cardiopulmonary bypass does not prevent ischemia of the lung in piglets: assessment of bronchial blood flow with fluorescent microspheres[®]

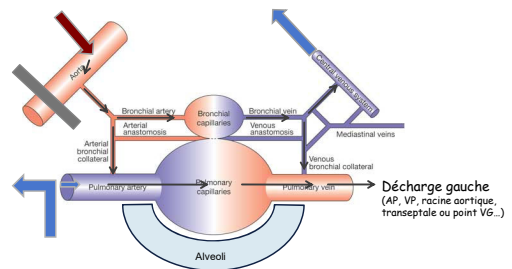


Schlensak C et al. Eur J Cardioth Surg 2001; 19:329-32

Schlensak C et al. J Thorac Vasc Surg 2002;123:1199-205



Rappels anatomiques « Shunts physiologiques de la circulation du poumon »



de Leval MR. The circulation of the lung
Nature Clinical Practice Cardiovascular Medicine (2005) 2, 202-208

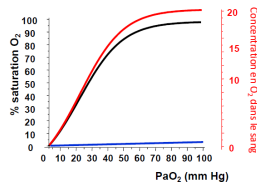
Contenu de l'oxygène dans le sang

$CaO_2 = O_2$ transporté par Hb + O_2 dissous

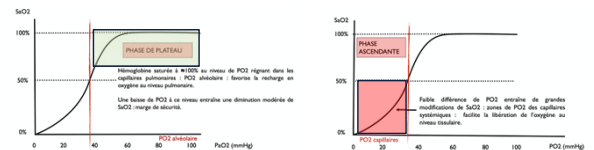
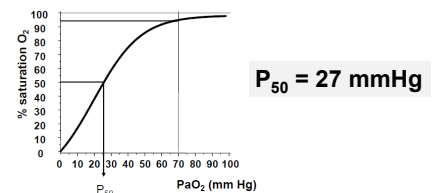
$$CaO_2 = (SaO_2 \times Hb \times 1,34) + PaO_2 \times 0,031$$

1 gramme d'Hb fixe 1,34 mL d'O₂

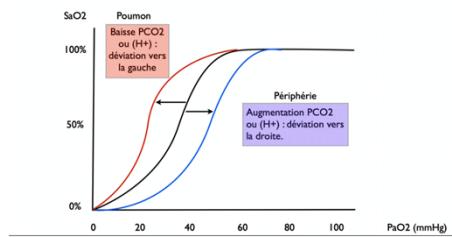
Coefficient de solubilité de l'O₂ dans le sang (en mL d'O₂/L sang/mmHg)



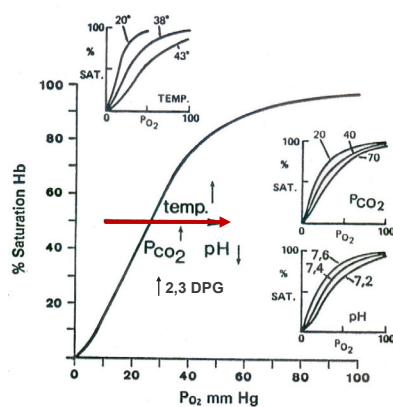
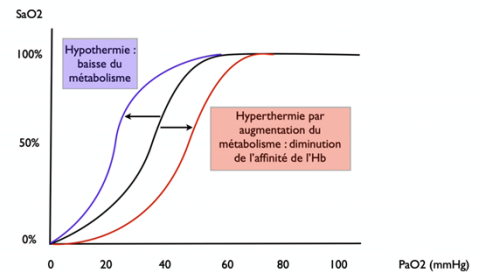
Courbe de BARCROFT



EFFET BOHR*



Physiologiste Danois (1904)



TRANSPORT EN OXYGÈNE

$$DO_2 (TaO_2) = DC \times CaO_2$$

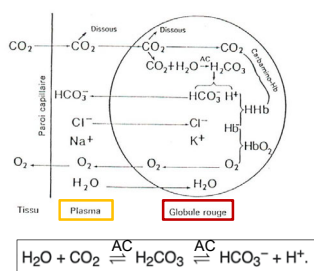
$$= VES \times FC \times (SaO_2 \times Hb \times 1,34 + 0,03 \times PaO_2)$$

$$= \mathbf{VES \times FC \times SaO_2 \times Hb \times 1,34}$$

$$= \mathbf{Débit\ pompe \times SaO_2 \times Hb \times 1,34}$$

(Valeur normale 10 ml d'O₂/kg/min ou 500 ml/min/m²
et VO₂ 3,5 ml/kg/min)

TRANSPORT EN CO₂

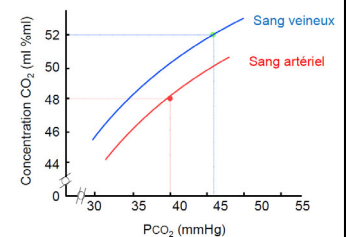


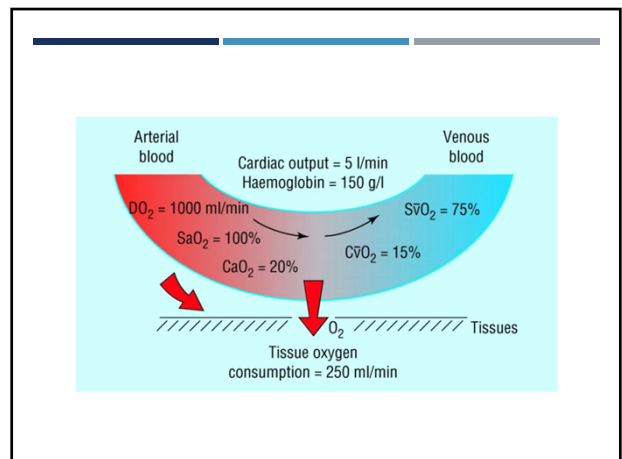
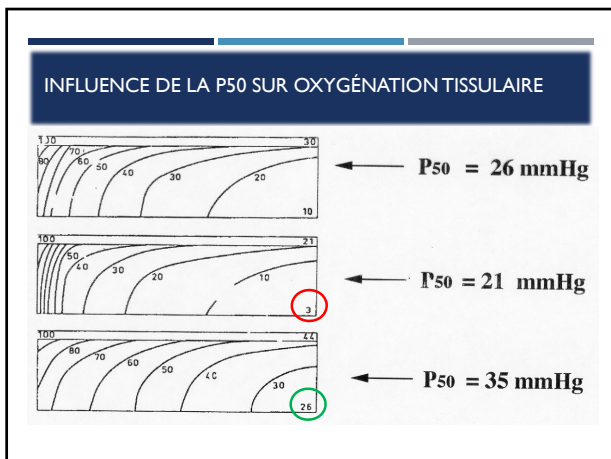
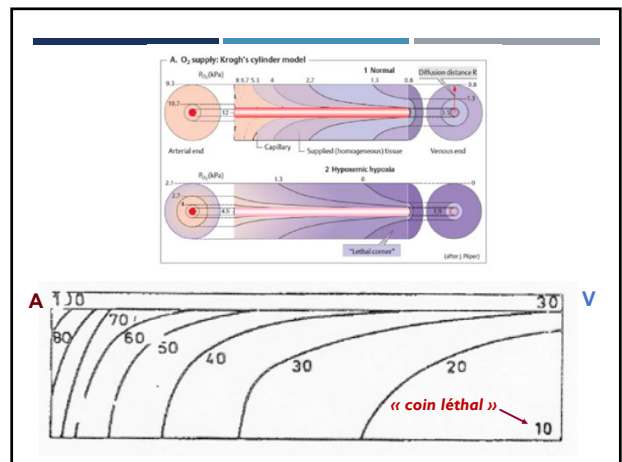
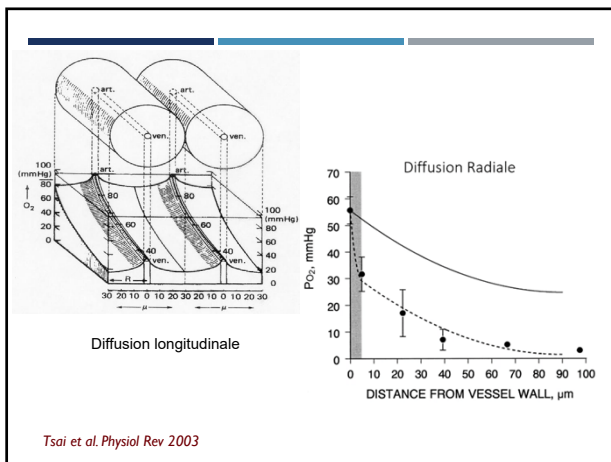
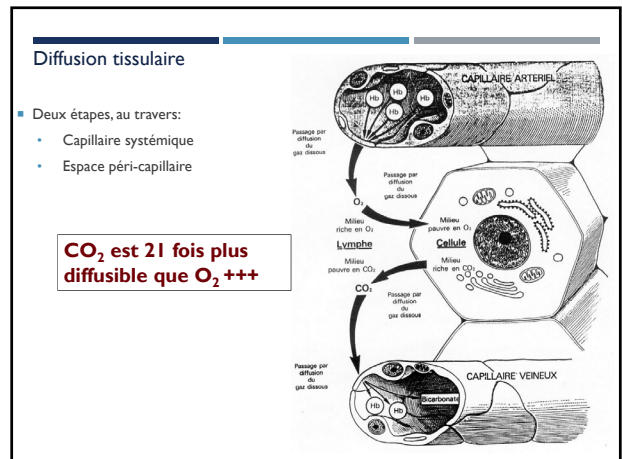
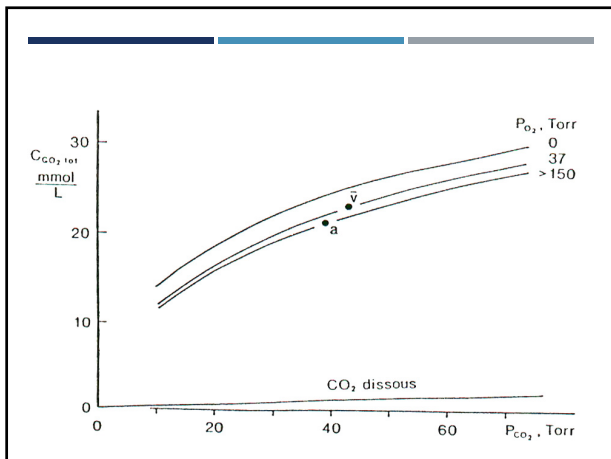
EFFET HALDANE

Fixation du CO₂ à l'hémoglobine (extrémités NH₂)

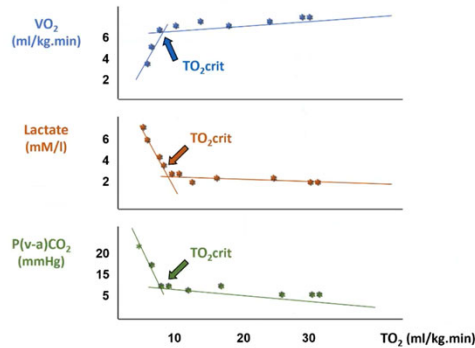
Pour une valeur de PCO₂ donnée, le sang contient d'autant plus de CO₂ lié à l'hémoglobine qu'il est riche en hémoglobine réduite (non liée à l'oxygène = desoxyhémoglobine) et donc du sang veineux.

C'est l'effet HALDANE





Adaptation TaO_2/VO_2



Déterminants SvO_2

$$VO_2 = (CaO_2 - CvO_2) \cdot DC$$

Or :

$$CaO_2 = (1,39 \cdot Hb \cdot SaO_2) + (0,0031 \cdot PaO_2)$$

$$CvO_2 = (1,39 \cdot Hb \cdot SvO_2) + (0,0031 \cdot PvO_2)$$

négligeable

D'où :

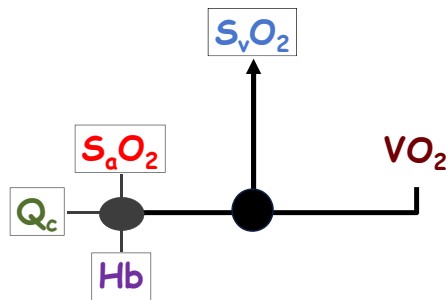
$$VO_2 \approx (SaO_2 - SvO_2) \cdot (Hb \cdot 1,39 \cdot DC)$$

$$SaO_2 - SvO_2 \approx \frac{VO_2}{Hb \cdot 1,39 \cdot DC}$$

$$SvO_2 \approx SaO_2 - \frac{VO_2}{Hb \cdot 1,39 \cdot DC}$$

$$ERO_2 = I - SvO_2$$

SvO_2 : FACTEURS DÉTERMINANTS



Adult: Perioperative Management

Ranucci et al

Goal-directed perfusion to reduce acute kidney injury: A randomized trial

Check for updates

2018

Marco Ranucci, MD, FESC,¹ Ian Johnson, CCP,^{2,3} Timothy Willcox, CCP,^{2,3} Robert A. Baker, PhD, CCP,¹ Christa Boer, MD, PhD,^{4,5} Andreas Baumann, MD,⁶ George A. Jostson, CCP,¹ Filip de Somer, CCP,¹ Paul Exton, BSc (Hon) ACP,¹ Seema Agarwal, FRCA,⁷ Rachael Parke, PhD,^{8,9} Richard F. Newland, CCP,¹ Renard G. Haumann, CCP,¹ Dirk Buchwald, PhD, CCP,¹ Nathan Weitzel, MD,¹ Rajamanyer Venkateswarani, MD, FRCS(Cth),¹ Federico Ambroggi, PhD,¹ and Valeria Pinardi¹

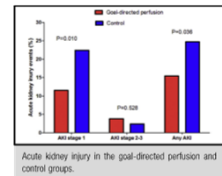
ABSTRACT

Objective: To determine whether a goal-directed perfusion (GDP) strategy aimed at maintaining oxygen delivery (DO_2) at ≥ 250 mL·min⁻¹·m⁻² reduces the incidence of acute kidney injury (AKI).

Methods: This multicenter randomized trial enrolled a total of 350 patients undergoing cardiac surgery in 9 institutions. Patients were randomized to receive either GDP or conventional perfusion. A total of 326 patients completed the study and were analyzed. Patients in the treatment arm were treated with a GDP strategy during cardiopulmonary bypass (CPB) aimed to maintain DO_2 at ≥ 250 mL·min⁻¹·m⁻². The perfusion strategy for patients in the control arm was based on body surface area and temperature. The primary endpoint was the rate of AKI. Secondary endpoints were intensive care unit length of stay, major morbidity, and blood cell transfusions, and operative mortality.

Results: Acute Kidney Injury Network (AKIN) stage 1 was reduced in patients treated with GDP (relative risk [RR], 0.45; 95% confidence interval [CI], 0.25-0.83; $P = .01$). AKIN stage ≥ 2 did not differ between the 2 study arms (RR, 1.66; 95% CI, 0.46-6.0; $P = .328$). There were no significant differences in secondary outcomes. In a prespecified analysis of patients with a CPB time between 1 and 3 hours, the difference in favor of the treatment arm were more pronounced, with an RR for AKI of 0.49 (95% CI, 0.27-0.89; $P = .017$).

Conclusions: A GDP strategy is effective in reducing AKIN stage 1 AKI. Further studies are needed to define perfusion interventions that may reduce more severe levels of renal injury (AKIN stage 2 or 3). J Thorac Cardiovasc Surg 2018;156:1916-27.



ECHANGE GAZEUX EXTRA-CORPORELS

- "Un poumon simplifié" = oxygénateur.
- Principe de la membrane: interface entre sang et gas.
- Large surface de diffusion: supérieure aux besoins standards en CEC
- Permet une relative tolérance de l'hémodilution.

	Poumon	Oxygénateur
Surface d'échange	100-150 m ²	1-4 m ²
Surface/volume	300 cm ²	28 cm ²
Distance de diffusion	1-2 µm	10-30 µm
Échange gazeux pour O ₂	210-3200 ml/min	200-400 ml/min

OXYGÉNATEUR

- Permet l'**oxygénation** et la **décarboxylation** du sang veineux.
- +/- administration agents anesthésiques inhalés.
- En aval de la pompe artérielle car résistance à l'écoulement.
- Technique moderne : oxygénateur à **MEMBRANES** :
 - évite contact air / sang.
 - moindre traumatisme des éléments sanguins.
 - risque d'embolie gazeuse quasi nul.
- Echanges gazeux par **DIFFUSION**: basés sur la différence en pression partielle du gaz entre la phase sanguine et la phase gazeuse.
- Echanges thermiques par **CONDUCTION**: entre 2 fluides (sang et eau).

MEMBRANES

POLYPROPYLENE

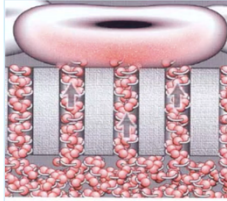


Figure 1: Typical hollow fiber design with a direct gas to blood interface through an open pore.

CEC
Fuite plasmatique et dégradation des échanges si > 6 heures

POLYMETHYLPENTHENE

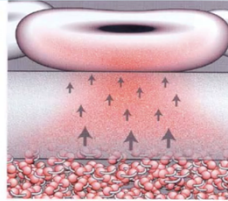
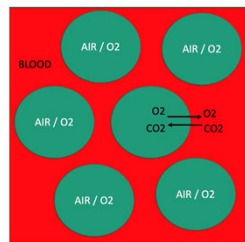
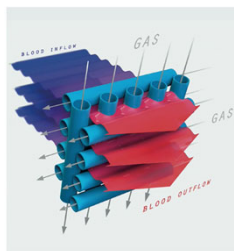
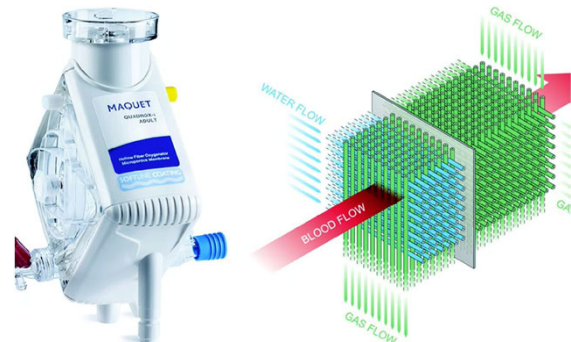


Figure 2: Gas exchange by diffusion through a semi-permeable membrane.

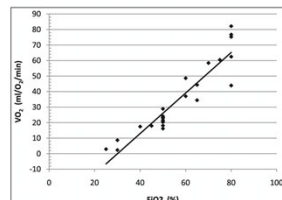
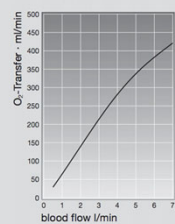
ECMO+++
Hémocompatible, durée de vie supérieure
Imperméable aux halogénés



Oxygénation

- Peu dépendante du débit de gaz.
- Dépendante :
 - de la **FdO₂** du mélange gazeux.
 - du **DEBIT DE SANG** (notion de débit sanguin corrigé ou rated flow : débit max de sang 75% - 95%).
 - des caractéristiques de la membrane : coefficient de diffusion et surface de contact.
 - de l'état de la membrane (thrombose).
 - de la [Hb] et de la SaO₂ de l'entrée (gradient).

Oxygen Transfer



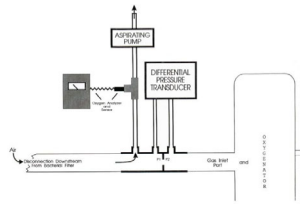
Ginther et al, Perfusion, 2013

Décarboxylation

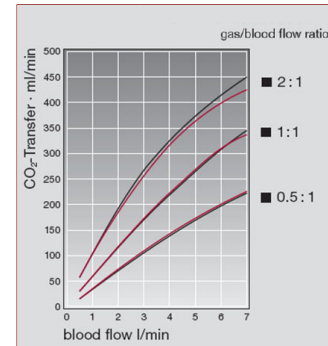
Débit de sang (non limitant, 25% du débit physiologique suffit).

- Dépendante :
 - du DEBIT DE GAZ (**BALAYAGE** ou sweep gas).
 - du gradient de diffusion (PaCO₂ d'entrée).
 - des caractéristiques de la membrane (surface...).

GAZ FRAIS



Kirson et al. J Cardiothorac Vasc Anesth, 1994

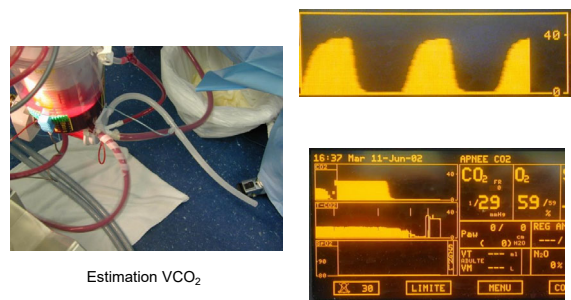


MONITORAGE GAZ DU SANG

- 1) Spectrophotométrie par réflexion
→ saturation sanguine en O_2
- 2) Capteurs électrochimiques
→ pressions partielles O_2 & CO_2
→ pH
→ autres paramètres : K^+
- 3) Mesure répétées des GDS

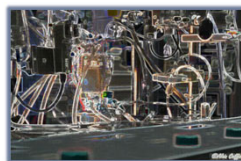


MONITORAGE GAZ (ÉVENT MEMBRANE)



Quel niveau d'oxygénation en CEC ?

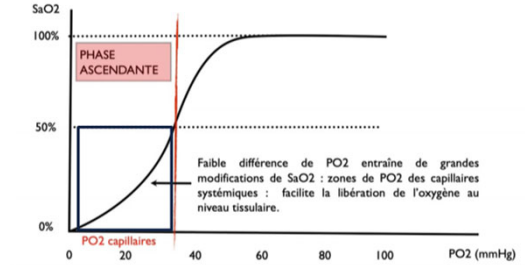
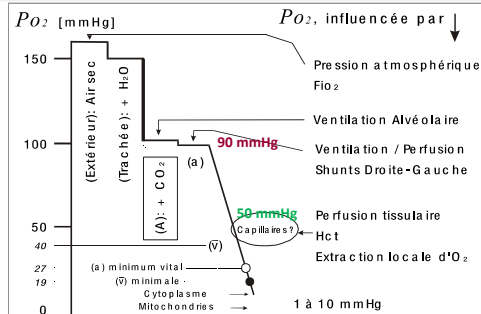
Prof. Alexandre OUARTARA
Département d'Anesthésie-Réanimation
Centre Médico-chirurgical Magellan
Hôpital Haut-Lévêque hospital
33600 Pessac, France
E-mail: alexandre.ouattara@chu-bordeaux.fr



TERMINOLOGIE

- **Hyperoxémie ou hypoxémie** = teneur en oxygène dans sang
- **Hyperoxie ou hypoxie** = teneur en oxygène dans tissu
- Hyperoxémie ne se traduit pas obligatoirement une hyperoxie
- Hypoxie tissulaire peut se voir sans hypoxémie

CASCADE DES PO₂



CONTENU ARTÉRIEL EN OXYGÈNE (CONCENTRATION EN OXYGÈNE)

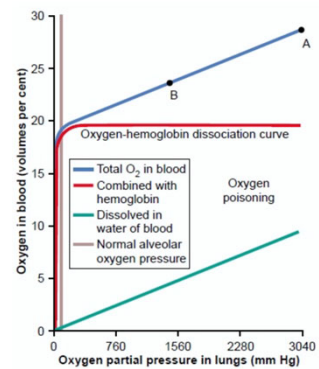
$$CaO_2 = O_2 \text{ transporté par Hb} + O_2 \text{ dissous}$$

$$CaO_2 = (SaO_2 \times Hb \times 1,34) + PaO_2 \times 0,003$$

1 gramme d'Hb fixe 1,34 mL d'O₂

Coefficient de solubilité de l'O₂ dans le sang (en mL d'O₂/L sang/mmHg)

$$CaO_2 = 18-20 \text{ ml d'O}_2/100 \text{ ml de sang}$$



TRANSPORT EN OXYGÈNE (OXYGEN DELIVERY)

$$TaO_2 (DO_2) = CaO_2 \times \text{Débit cardiaque}$$

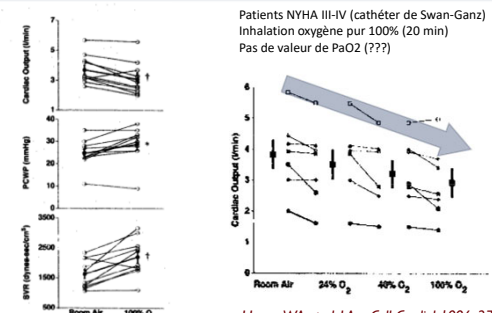
ml d'O₂/100 ml de sang

L.min⁻¹

Valeur normale 600-800 ml d'O₂. min⁻¹

EFFETS HEMODYNAMIQUES DE L'HYPEROXIE

Patients NYHA III-IV (cathéter de Swan-Ganz)
Inhalation oxygène pur 100% (20 min)
Pas de valeur de PaO₂ (???)

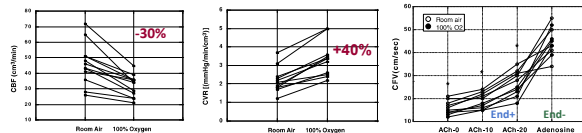


Haque WA et al. J Am Coll Cardiol 1996; 27:353-7

HYPEROXIE ET CIRCULATION CORONAIRE

Patients (n=27) bénéficiant d'un cathétérisme cardiaque
O₂ pur durant 15 min (**273 ± 43 mmHg**)

	Blood O ₂ Content, ml/l		Art-CS O ₂ Content Difference, ml/l	Cardiac Vo ₂ , ml/min
	Art	CS		
Room air	181 ± 24	62 ± 20	119 ± 26	5.2 ± 0.6
100% Oxygen	194 ± 22*	73 ± 21*	120 ± 23	3.8 ± 0.4*



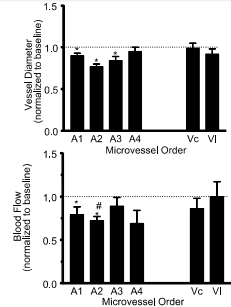
HYPEROXEMIE ET MICROCIRCULATION

Etude animale (hamster)
O₂ pur pendant 30 min
Etude microvasculaire

Table 1. Systemic parameters and blood gas analysis during baseline and 100% O₂ inspiration

	Baseline	100% O ₂
Blood pressure, mmHg	88.3 ± 6.4	87.2 ± 7.1
Heart rate, beats/min	420.0 ± 28.3	389.0 ± 28.8
Arterial P _{O₂} , mmHg	60.0 ± 1.2	477.9 ± 19.9*
Arterial P _{O₂} , mmHg	59.8 ± 4.1	44.4 ± 2.8*
Arterial p _H	7.38 ± 0.02	7.33 ± 0.01*
Venous P _{O₂} , mmHg	21.7 ± 7.1	38.4 ± 3.3*
Venous P _{O₂} , mmHg	79.4 ± 2.8	82.9 ± 8.2*
Venous p _H	7.35 ± 0.01	7.27 ± 0.00*
Cardiac index, ml·min ⁻¹ ·kg ⁻¹	196 ± 13	144 ± 31*
(normalized to baseline)	(1.00 ± 0.00)	(0.75 ± 0.07)
Vascular resistance, mmHg·min·kg ⁻¹ ·ml ⁻¹	0.46 (1.00)	0.61 (1.33)
(normalized to baseline control)		

Values are means ± SE. *P < 0.05 (statistically significantly different from baseline). Numbers in parentheses refer to results normalized to control.



Tsai AG et al. Am J Physiol Heart Circ Physiol 2003

EFFETS DE L'HYPEROXIE SUR OXYGENATION MUSCULAIRE

Patients de chirurgie cardiaque (n=10)
Mesure de la pression partielle musculaire en O₂ au cours de l'hyperoxémie (83-108 mmHg versus 281-356 mmHg)

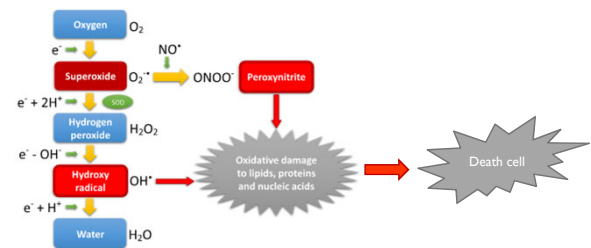
Clinical course. No positive bacterial cultures from the electrode application sites or clinical complications from that site were seen. Similarly no other clinical complications were noted.

Discussion

In this study hyperoxemia increased global oxygen delivery and oxygen saturation in mixed venous blood during CPB. This effect might be assumed to provide improved tissue oxygenation and safety. However, muscle oxygenation deteriorated during hyperoxemia. This paradoxical finding and other results suggest that hyperoxemia may be harmful to tissue oxygenation.

Joachimsson PO et al. J Thorac Cardiovasc Surg 1996;112:812-9

PRODUCTION ESPÈCES RÉACTIVES DE L'OXYGÈNE (STRESS OXYDATIF)



Nakane M J Intensive Care 2020; 8:95

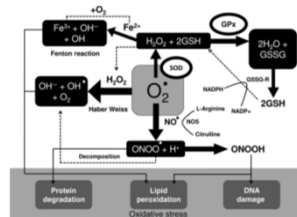
STRESS OXYDATIF ET CEC



Table 1: Potential triggers for increased ROS generation during ECMO

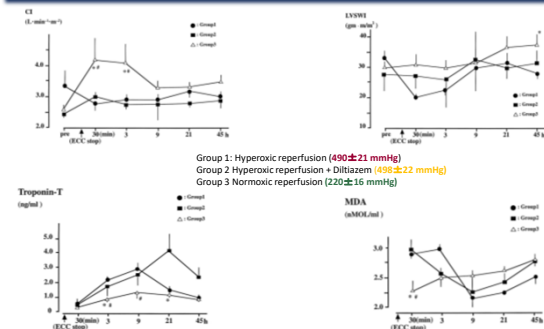
Potential triggers for increased ROS generation during ECMO

- Exposure to ECC (ECMO circuits) [43] dialysis [13]
- SIRS syndrome [43]
- Acute respiratory distress syndrome [16]
- Hyperoxia [26]
- Mechanical ventilation [44]
- Sepsis [43]
- Ischemia-reperfusion [46]
- Transfusion [27, 28]
- Sequestration of antioxidant trace elements by the ECMO circuit [19]



McDonald CI et al. Eur J Cardiothorac Surg 2014;46:937-43

HYPEROXEMIE ET REPERFUSION MYOCARDIQUE



Group 1: Hyperoxic reperfusion (**490 ± 21 mmHg**)
Group 2: Hyperoxic reperfusion + Diltiazem (**498 ± 22 mmHg**)
Group 3: Normoxic reperfusion (**220 ± 16 mmHg**)

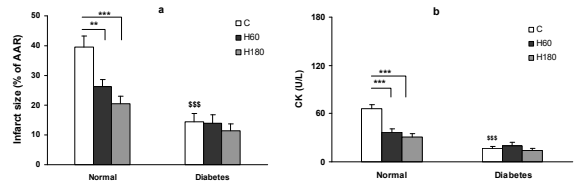
HYPEROXEMIE ET MICRO-BULLES

- Accélération dissolution microbulles
- Effet dénitrogénéation (azote moins soluble que l'oxygène)

Nollert G et al. J Thorac Cardiovasc Surg 1999;117:1166-71

HYPEROXIE ET PRECONDITIONNEMENT MYOCARDIQUE

Etude expérimentale chez rat soumis à O₂ pur (60 ou 180 min, effet dose?)
I/R 30/120 min sur préparation de Langendorff (cœur isolé)
Evaluation taille IDM/AR



Doit-on appliquer une hyperoxie préCEC (pré-oxygénation)?

Pourkhalili K et al. 2012

ETUDE CLINIQUE I: DOMMAGE MYOCARDIQUE ET DYSFONCTION ORGANES

Patients opérés de PAC isolé (>18 ans)
CEC non pulsée hypothermie modérée (34-36° C)

Groupe libéral PaO₂ 200-220 mmHg en CEC (CDI 500)
et 130-150 mmHg en réanimation

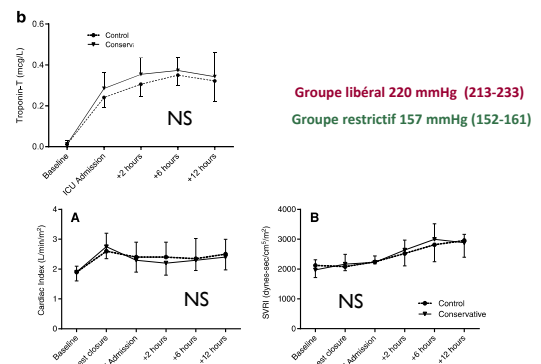
versus

Groupe restrictif PaO₂ 130-150 mmHg en CEC (CDI 500)
et 80-100 mmHg en réanimation

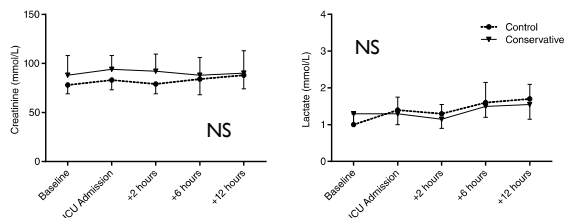
C_{JP}= Dommages myocardiques (AUC Troponine T)

CJS= morbi-mortalité postopératoire

Smit B et al. Crit Care 2016;20:55



Groupe libéral 220 mmHg (213-233)
Groupe restrictif 157 mmHg (152-161)



ETUDE CLINIQUE II: FONCTION COGNITIVE

Patients opérés de PAC isolé (>65 ans)

FiO₂ 35% (PaO₂ >70 mmHg et SpO₂>92%) avant CEC
et PaO₂ entre 100 et 150 mmHg durant CEC (n=51)

versus

FiO₂= 100% durant toute la chirurgie (n=49)

C_{JP}= Montreal Cognitive Assessment Score (J2 postopératoire)

CJS= MCAS à M1, M3, M6 et morbi-mortalité postopératoire

Shaeff A et al. Anesthesiology 2021;134:189-201

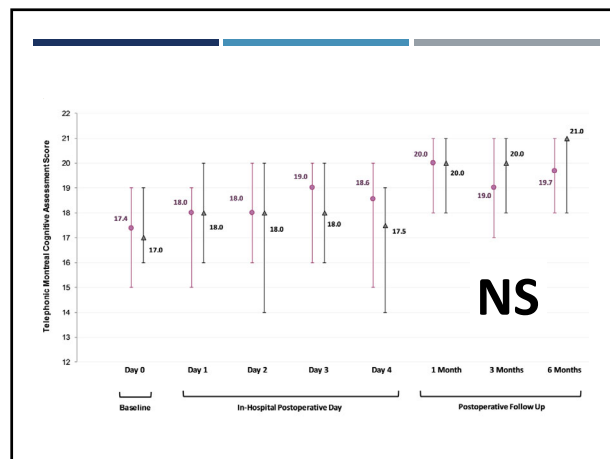
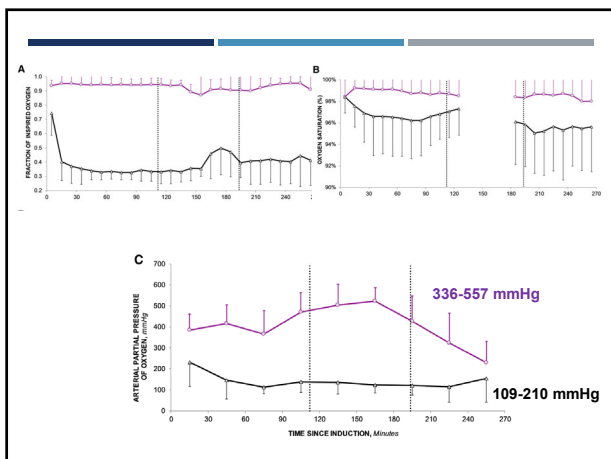


Table 3. Study Outcomes

	Hyperoxia (n = 49)	Normoxia (n = 51)	P Value
Delirium	15 (30.61)	16 (31.37)	0.93
Delirium severity (worst)	11 (8-13)	8 (7-11)	0.23
Time to delirium	1 (1-2)	2 (1-3)	0.17
Time characteristics			
Hospital length of stay, days	8 (5-11)	7 (5-10)	0.70
Intensive care unit length of stay, days	2 (1-3)	1 (1-3)	0.34
Hours of initial intubation	4.8 (3.7-6.7)	5.5 (3.7-6.7)	0.76
Adverse clinical outcomes			
Mortality			
In-hospital	0 (0)	0 (0)	0.32
After 30 days	0 (0)	1 (1.96)	0.37
After 6 months†	0 (0)	1 (2.56)	0.37
Stroke	0 (0)	0 (0)	0.36
Pneumonia	3 (6.12)	1 (1.96)	0.32
Renal failure	0 (0)	1 (1.96)	0.32
Reoperation (bleeding)	0 (0)	1 (1.96)	0.32
Atrial fibrillation	14 (28.57)	16 (31.37)	0.83

NS

Shaeefi A et al. *J Card Surg* 2022 Jun 9. doi: 10.1111/jocs.16661. Online ahead of print.

ORIGINAL ARTICLE

Intraoperative hyperoxygenation may negatively affect postoperative cognitive functions in coronary artery bypass graft operations: A randomized controlled study

Tugba Onur MD, Ümran Karaca MD, Filiz Ata MD, Halil E. Sayan MD, Anil Onur MD, Canan Yilmaz MD, Ayşe N. Balkaya MD, Cüneyt Eriş MD

PaO₂ entre 100 et 180 mmHg versus PaO₂>180 mmHg

ETUDE CLINIQUE III: TROUBLES DU RYTHME POST-OPERATOIRES

Patients opérés d'une chirurgie cardiaque sous CEC (>18 ans)
CEC normothermique (>36° C)

PaO₂ < 150 mmHg avec possibilité d'augmenter la FiO₂ si SvO₂ < 60% (n=167)

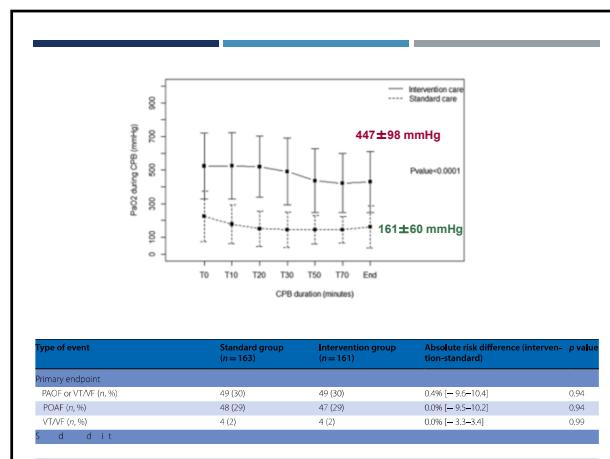
versus

FiO₂= 100% durant toute la CEC (n=163)

CJP= Troubles du rythme dans les 15 jours postopératoires (ACFA/FV/TV)

CJS= Morbi-mortalité postopératoire

Abou-Arab O et al. *Intensive Care Med* 2019; 45:1413-21

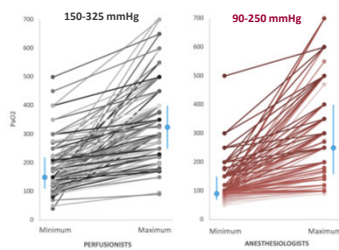


Type of event	Standard group (n = 163)	Intervention group (n = 161)	Absolute risk difference (intervention-standard)	p value
Secondary endpoints				
MACCE (n, %)	34 (21)	39 (24)	3.4% [-5.7-12.5]	0.47
Outcomes (n, %)				
Cardiac arrest	2 (1)	3 (2)	0.6% [-2.0-3.3]	0.64
In-hospital mortality	0 (0)	4 (2)	2.5% [-0.06-5.3]	0.06
Acute kidney injury	30 (18)	35 (22)	3.3% [-5.4-12.0]	0.45
Stroke	2 (1)	1 (1)	-0.6% [-2.7-1.5]	0.57
Mesenteric ischemia	0 (0)	2 (1)	1.2% [-0.5-2.9]	0.25
Cardiac troponin (ng/L)				
6 h post CPB	7.4 [4.3-14.4]	8.0 [4.3-15.4]	-	0.79
Day 1 post CPB	4.3 [2.5-8.0]	4.6 [2.8-8.4]	-	0.53
Day 2 post CPB	1.9 [1.0-3.9]	2.0 [1.0-3.7]	-	0.80
Norepinephrine (n, %)	60 of 153 (39)	47 of 156 (30)	-9.1% [-19.7-1.5]	0.10
Dobutamine (n, %)	11 of 153 (7)	10 of 156 (6)	-0.7% [-6.3-4.9]	0.81
ICU discharge (days)	2 [2-3]	2 [2-3]	-	0.94
Hospital discharge (days)	11 [9-14]	10 [9-12]	-	0.09
Out-of-hospital mortality at month 6	3 (2)	0 (0)	-	0.08

NS

NS

QUELLE CIBLE? LES AVIS DIVERGENT...



« Too low » value?

Perfu = 100 mmHg

MAR = 60 mmHg

Calhoun A et al. J Cardiothorac Vasc Anesth 2022



European Journal of Cardio-Thoracic Surgery 68 (2019) 1-42
doi:10.1093/ejcts/ezy287



2019 EACTS/EACTA/EBCP guidelines on cardiopulmonary bypass in adult cardiac surgery

Authors/Task Force Members: Alexander Wahba ^{48,77} (Chairperson) (Norway), Milan Milojevic ^{44,77} (Serbia, Netherlands), Christa Boer ⁶ (Netherlands), Filip M.J. De Somer ¹ (Belgium), Tomas Gudbjartsson ² (Iceland), Jenny van den Groot ⁸ (Netherlands), Timothy J. Jones ⁹ (UK), Vladimir Lomivorotov ¹ (Russia), Frank Merkle ⁸ (Germany), Marco Ranucci ¹ (Italy), Gudrun Kunst ^{10,77} (Chairperson) (UK) and Luc Puis ^{6,77} (Chairperson) (Belgium)

7.14.2 Pharmacological interventions.

7.14.2.1 Hyperoxia. Exposure of the alveolus to 100% oxygen leads to alveolar collapse and the generation of oxygen radicals, which might exacerbate ischaemia-reperfusion injury after CPB. Small RCTs assessed the effect of hyperoxia on postoperative ventilation times, with conflicting results, demonstrating prolonged ventilation times with intraoperative hyperoxia in 1 RCT but not in others [304]. The lengths of the stays in the critical care unit and in the hospital were not affected [304].

CONCLUSION

- Hyperoxémie est souvent argumentée dans un souci sécuritaire en CEC
- Elle ne s'accompagne pas obligatoirement d'une hyperoxie tissulaire (impact micro-circulatoire)
- Effets potentiels délétères via le stress oxydatif induit (dommage cellulaire)
- Impact en pratique clinique?
- Cible **100-120 mmHg QSP > 96%**
- Meilleure interprétation SvO₂
- Hyperoxémie discutable en pré-CEC (induction anesthésique)