Echanges gazeux appliqués à la CEC

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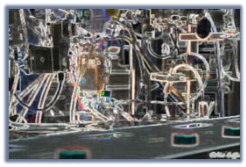

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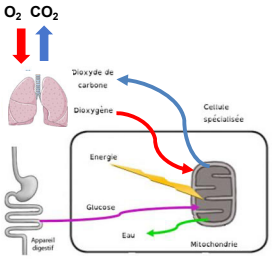
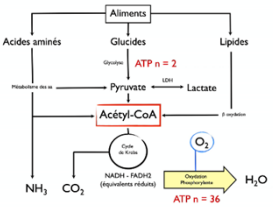
Respiration cellulaire



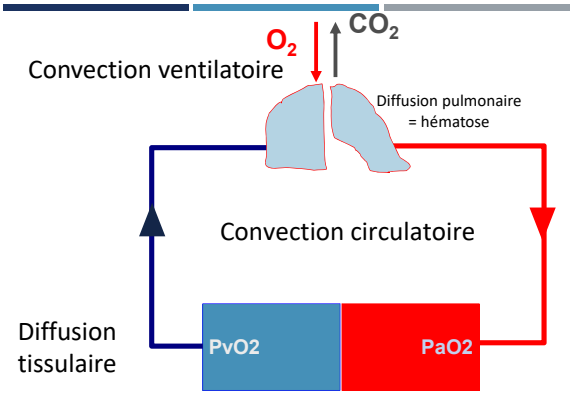


Quotient respiratoire = $\frac{VCO_2}{VO_2}$ = 0,8

PRIMUM MOVENS CELLULAIRE

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

LA POMPE CARDIO-THORACIQUE



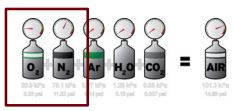
PHASE GAZEUSE => LOI DE DALTON (LOI DES PRESSIONS PARTIELLES)

- 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, de ce gaz dans le mélange
- Somme des pressions partielles est égale à la pression totale du mélange

$Px = P_T \times Fx \leftrightarrow \mathbf{Px = P_B \times Fx}$

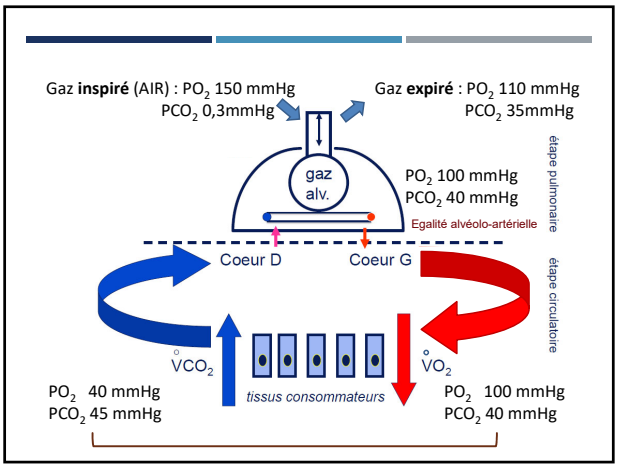
Composition de l'air ambiant

Gaz	Fraction (%)	Pression partielle (mmHg)
N_2	78.6	597
O_2	20.9	159
CO_2	0.04	0.3
H_2O	0.46	3.7
Total	**100%**	**760**

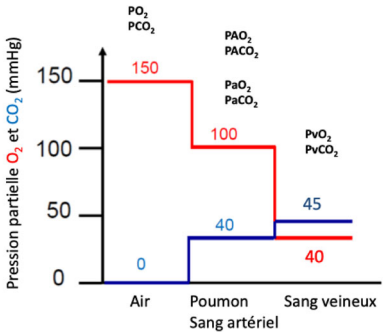


P_{GAZ} = Fraction X $P_{atmosphérique}$

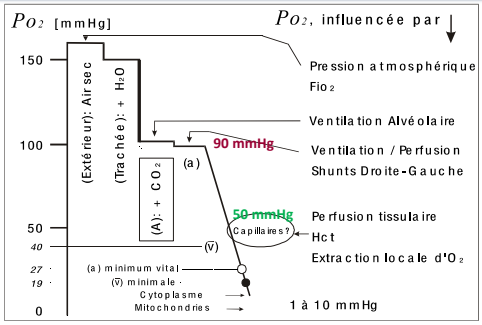
P_{ATM} = 760 mmHg au niveau de la mer



Cascade des PO_2 et PCO_2



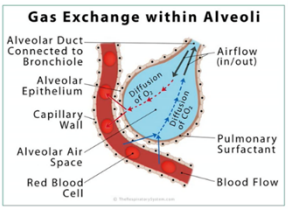
CASCADE DES PO_2



ETAPE PULMONAIRE: TRANSFERT DES GAZ

- **IMPLIQUE**
  - **phase gazeuse et liquidienne (sang)**
  - **séparées par la membrane alvéolo-capillaire**
- **DEPEND**
  - **de la composition de l'air alvéolaire**
  - **de la diffusion**
  - **rapports ventilation-perfusion**

Diffusion des gaz



Loi de Fick de Diffusion

$$Rate\ of\ gas\ diffusion = k_{gas}\ x\ A\ x\ \frac{\Delta P_{gas}}{D}$$

k_{gas} : constante de diffusion (gas dépendante)

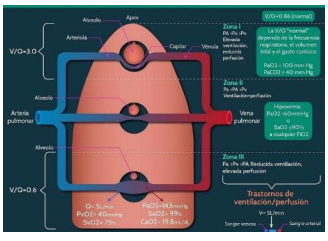
A: surface d'échange

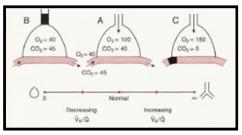
D: épaisseur de la membrane

Delta P_{gas}: gradient de pression partielle entre l'alvéole et le sang capillaire pulmonaire (gas dépendant)

Rapport ventilation-perfusion

Zone de West I-III



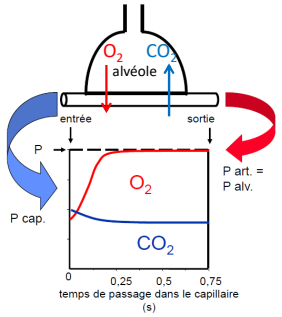


Zone I = PA > Pa > Pv

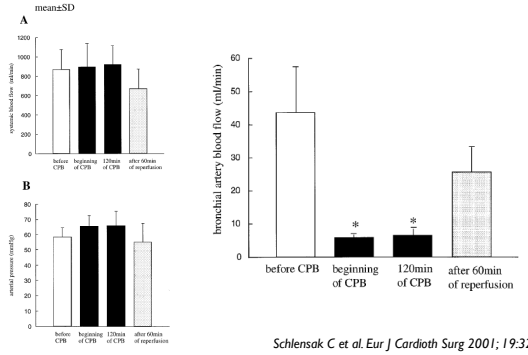
Zone II = Pa > PA > Pv

Zone III = Pa > Pv > PA

Temps de transit dans la barrière alvéolo-capillaire (effet $Pv0_2$)

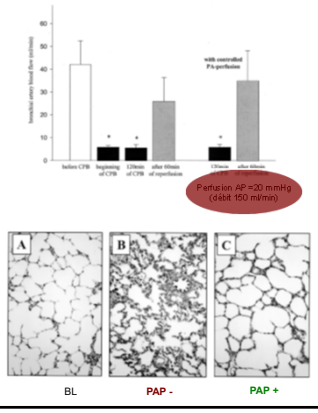


Bronchial artery perfusion during cardiopulmonary bypass does not prevent ischemia of the lung in piglets: assessment of bronchial artery blood flow with fluorescent microspheres☆



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

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



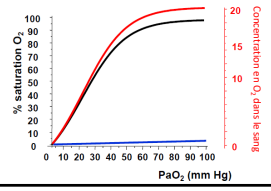
Contenu de l'oxygène dans le sang

CaO_2 = O_2 transporté par Hb + O_2 dissous

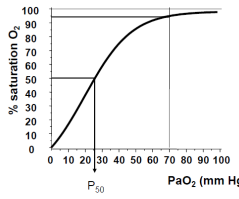
$$CaO_2 = (SaO_2 \times Hb \times 1{,}34) + PaO_2 \times 0{,}003$$

1 gramme d'Hb fixe 1,34 mL d'O_2

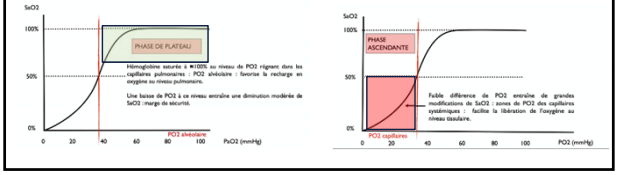
Coefficient de solubilité de l'O_2 dans le sang (en mL d'O2/L sang/mmHg)



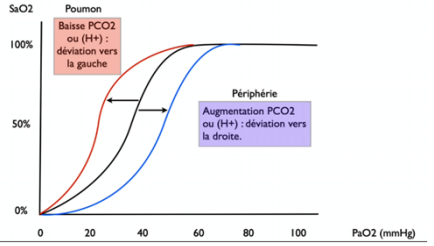
Courbe de BARCROFT



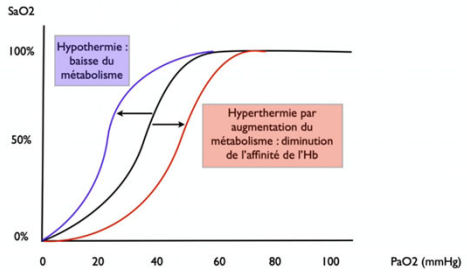
P_{50} = 27 mmHg

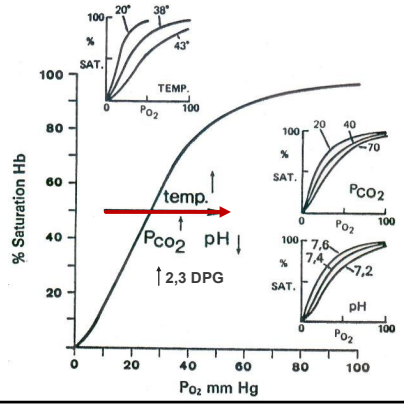


EFFET BOHR*

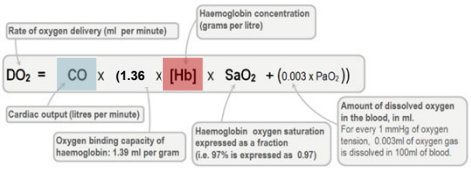


Physiologiste Danois (1904)





OXYGEN DELIVERY (DO_{2i})



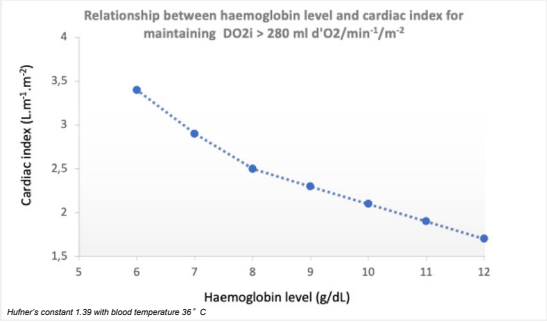
TRANSPORT EN OXYGÈNE

DO_2 (TaO_2) = DC X CaO_2

= VES X FC X (SaO2X Hb X 1,34 + 0,03 X PaO2)

= Débit pompe X SaO_2 X Hb X 1,34

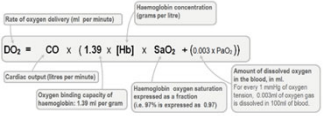
= Débit pompe X 1 X **Hb** X 1,34



THEORETICAL VALUE OF HÜFNER'S CONSTANT

1.39

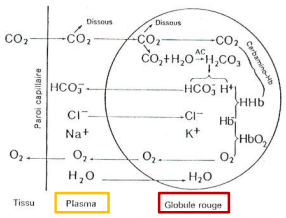
Comroe JH. Physiology of Respiration, 2nd edn. Chicago: Year Book. Medical Publishers 1974



1.31

Lumb AB et al. Nunn's Applied Respiratory Physiology, 5th edition Oxford: Butterworth-Heinemann 2000

TRANSPORT EN CO_2



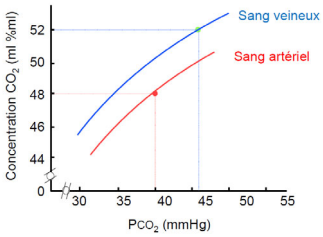
$$H_2O + CO_2 \overset{AC}{\rightleftharpoons} H_2CO_3 \overset{AC}{\rightleftharpoons} HCO_3^- + H^+.$$

EFFET HALDANE

Fixation du CO_2 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.

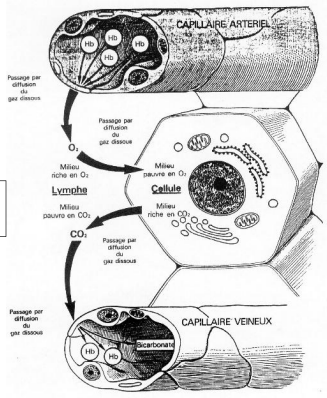
C'est l'effet HALDANE

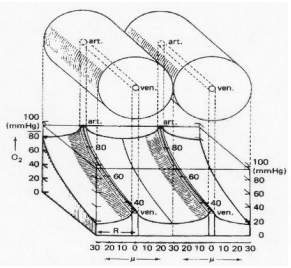


Diffusion tissulaire

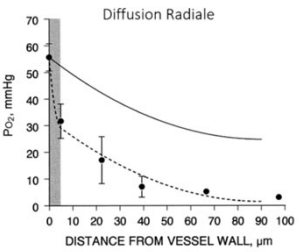
- Deux étapes, au travers:
  - Capillaire systémique
  - Espace péri-capillaire

CO_2 est 21 fois plus diffusible que O_2 +++

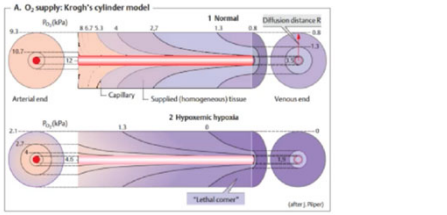


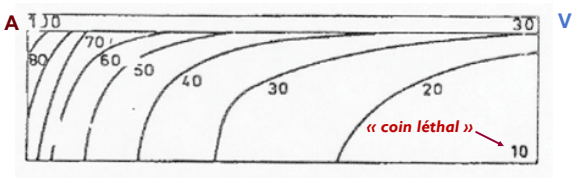


Diffusion longitudinale

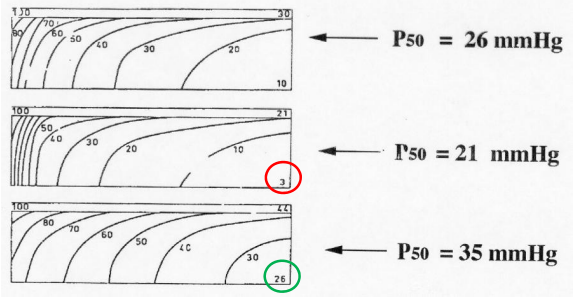


Tsai et al. Physiol Rev 2003





INFLUENCE DE LA P50 SUR OXYGÉNATION TISSULAIRE

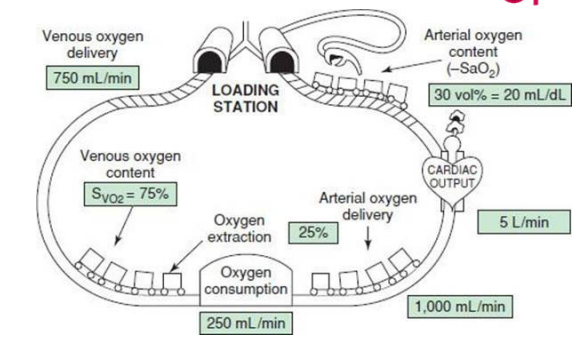


$DO_2 = DC \times [(Hb \times SaO_2 \times 1.36) + PaO2 \times 0.003]$

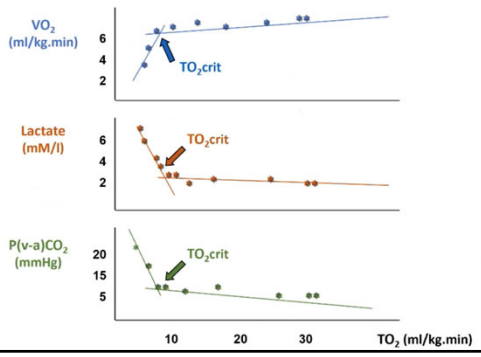
$VO_2 = DC \times (CaO_2\text{-}CvO_2)$

$ERO_2 = VO_2 / DO_2$

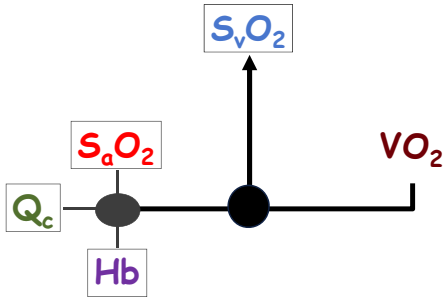
$EO_2 = 1 - SvO_2$



Adaptation TaO_2/VO_2

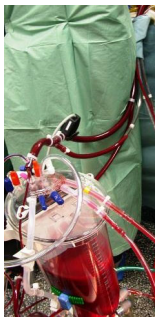


S_vO_2 : FACTEURS DÉTERMINANTS



HO km et al. Shock 2008;29:3-6

VENOUS OXYGEN SATURATION



After ventilation with 100% inspired oxygen for 5 min

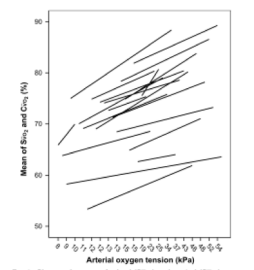


Fig. 1. Changes in mean of mixed (SvO_2) and central (CvO_2) venous oxygen saturation with changes in arterial oxygen tension in 20 critically ill patients with circulatory failure.

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

2018

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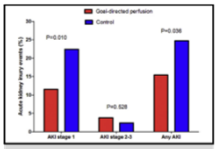
ABSTRACT

Objective: To determine whether a goal-directed perfusion (GDP) strategy aimed at maintaining oxygen delivery (DO_2) at ≥280 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 ≥280 mL·min^{-1}·m^{-2}. The perfusion strategy for patients in the control arm was factored 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, red 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 ≥3 did not differ between the 2 study arms (RR, 1.66; 95% CI, 0.46-6.0; $P = .528$). There were no significant differences in secondary outcomes. In a prespecified analysis of patients with a CPB time between 1 and 3 hours, the differences 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;156:1918-27)



Acute kidney injury in the goal-directed perfusion and control groups.

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 tolerance de l'hémodilution.

	Poumon	Oxygénateur
Surface d'échange	100-150 m²	1-4 m²
Surface/volume	300 cm⁻¹	28 cm⁻¹
Distance de diffusion	1-2 µm	10-30 µm
Échange gazeux pour O_2	210-3200 ml/mn	200-400 ml/mn

OXYGENATEUR

- Permet l'**oxygenation** 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

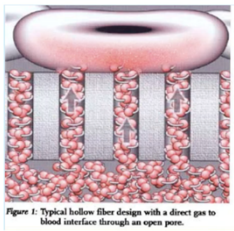


Figure 1: Typical hollow fiber design with a direct gas to blood interface through an open pore.

CEC
Fuite plasmatique et degradation des échanges si > 6 heures

POLYMETHYLPENTHENE

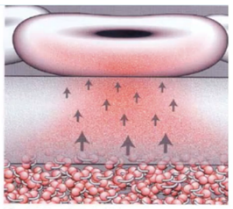


Figure 2: Gas exchange by diffusion through a semi-permeable membrane.

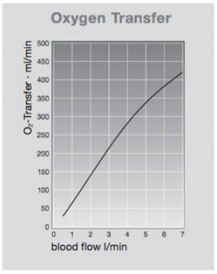
ECMO++++
Hémocompatible, durée de vie supérieure
Imperméable aux halogénés

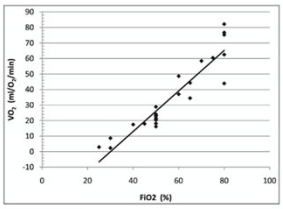
Oxygénation

•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'Hb d'entrée (gradient).





Ginther et al, Perfusion, 2013

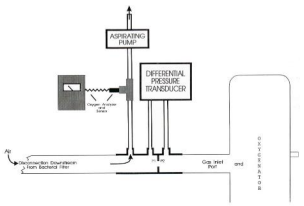
Décarboxylation

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

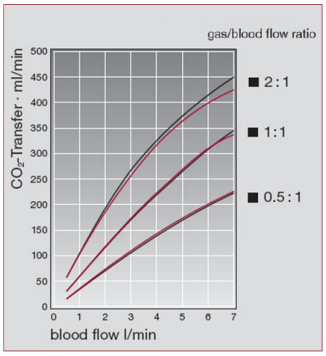
•Dépendante :

- 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

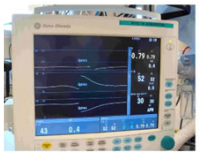


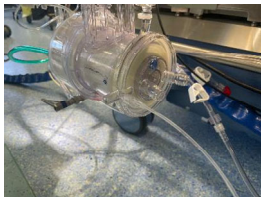
MONITORAGE GAZ DU SANG

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

VCO_2 MEASURED IN REAL TIME ON CPB CIRCUIT





$$V_{CO_2}\ \text{indexed}\ (mL \cdot min^{-1} \cdot m^{-2}) = \frac{eCO_2(mm\ Hg) \cdot Ve(L/min)}{760 \cdot BSA(m^2)}$$

CO_2 DERVIED PARAMETERS?

- CO_2 is the final product of cellular metabolism
- **In aerobic conditions**, CO_2 is produced by the Krebs cycle proportionally to oxygen consumption (respiratory quotient VCO_2/VO_2 =0.8)
- **In anaerobic conditions**, a decrease in VCO_2 is observed and related to a decrease in VO_2. Because CO_2 anaerobic production (residual hydrolysis ATP into ADP and neutralization H+ from lactic acid synthesis by bicarbonate ion), the decrease is proportionally less. Consequently, **an increase in VCO_2/VO_2 ratio is observed.**

BTPS (Body Temperature and Pressure, Saturated) vers STPD (Standard Temperature and Pressure, Dry)

Production de CO_2 (VCO_2) :
VCO_2 en mL/min = VE en L/min x 1000 × (P_ECO_2 en mmHg / (P_B-P_{H2O}) en mmHg)

Conversion du volume de gaz mesurée aux conditions BTPS aux conditions STPD

Application loi des Gaz parfaits $\frac{\text{Pression X Volume}}{\text{T (kelvin)}}$ = Constante

$$\frac{P_{STPD} \times V_{STPD}}{T_{STPD}} = \frac{V_{BTPS} \times P_{BTPS}}{T_{BTPS}} \quad \text{donc } V_{STPD} = \left[\frac{P_{BTPS} \times T_{STPD}}{T_{BTPS} \times P_{STPD}}\right] \times V_{BTPS}$$

Facteur correcteur K de BTPS en STPD (VCO_2 corrigée)

$$V_{STPD} = \left[\frac{P_{BTPS} \times T_{STPD}}{T_{BTPS} \times P_{STPD}}\right] \times V_{BTPS}$$

$$V_{STPD} = \frac{(760-P_{H2O}) \times 273}{(273+T^\circ) \times 760} \times V_{BTPS}$$

Facteur correcteur K1 de BTPS en STPD (VCO_2 corrigée)

T° température du compartiment gazeux (ligne artérielle)

Influence de la température sur le coefficient K de conversion de BTPS en STPD

Temperature (°C)	K_1
32	0.853
34	0.842
35	0.837
37	0.826

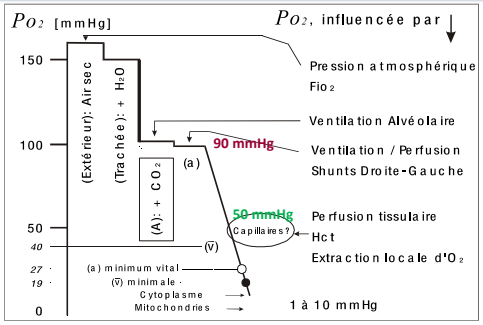
Volume STPD est toujours plus petit que volume BTPS
VCO_2 corrigée toujours plus faible que la VCO_2 mesurée sur évent (K1<1)

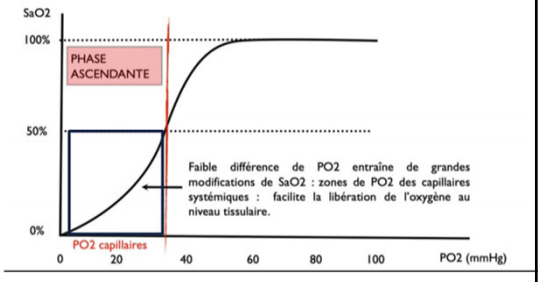
Quel niveau d'oxygénation au cours de la CEC?

TERMINOLOGIE

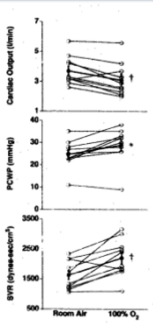
- Hyperoxémie ou hypoxémie = teneur en oxygène dans sang
- Hyperoxie ou hypoxie = teneur en oxygène dans tissu
- Hyperoxémie ne se traduit pas obligatoirement une hyperoxie
- Hypoxie tissulaire peut se voir sans hypoxémie

CASCADE DES PO_2

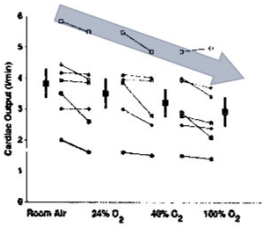




EFFETS HEMODYNAMIQUES DE L'HYPEROXIE



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



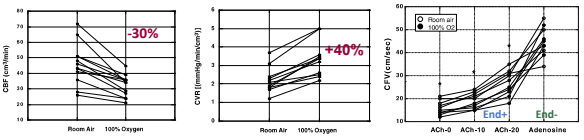
Haque WA et al. J Am Coll Cardiol 1996; 27:353-7

McNulti PH et al. Am J Physiol Heart Circ Physiol 2005

HYPEROXIE ET CIRCULATION CORONAIRE

Patients (n=27) bénéficiant d'un cathétérisme cardiaque
O_2 pur durant 15 min (**273±43 mmHg**)

	Blood O_2 Content, ml/l		Art-CS O_2 Content Difference, ml/l	Cardiac $\dot{V}O_2$, 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*



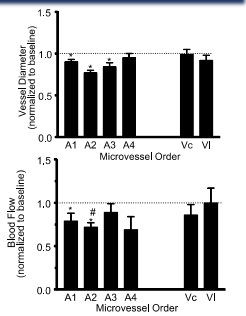
HYPEROXEMIE ET MICROCIRCULATION

Etude animale (hamster)
O_2 pur pendant 30 min
Etude microvasculaire

Table 1. *Systemic parameters and blood gas analysis during baseline and 100% O_2 inspiration*

	Baseline	100% O_2
Blood pressure, mmHg	88.3 ± 6.4	87.7 ± 7.1
Heart rate, beats/min	420.0 ± 28.3	392.0 ± 26.8
Arterial Po_2, mmHg	60.0 ± 1.2	477.9 ± 19.9*
Arterial Pco_2, mmHg	59.8 ± 4.1	64.3 ± 2.1*
Arterial pH	7.36 ± 0.02	7.33 ± 0.01*
Venous Po_2, mmHg	21.7 ± 7.1	26.4 ± 3.3*
Venous Pco_2, mmHg	70.4 ± 2.8	82.9 ± 8.2*
Venous pH	7.33 ± 0.01	7.27 ± 0.03*
Cardiac index, ml·min^{-1}·kg^{-1} (normalized to baseline)	196 ± 13 (1.00 ± 0.00)	144 ± 31* (0.75 ± 0.07)
Vascular resistance, mmHg·min·kg·ml^{-1} (normalized to baseline control)	0.46 (1.00)	0.61 (1.33)

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

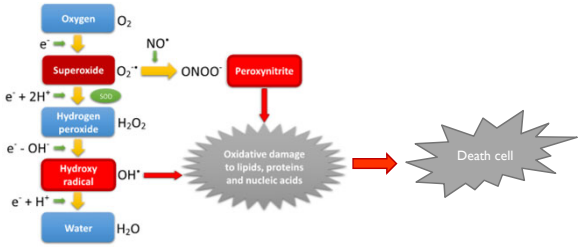
In this study hyperoxemia increased global oxygen delivery and oxygen saturation in mixed venous blood during CPB. This effect might be assumed to provide improved tissue oxygenation and safety. However, muscle oxygenation deteriorated during hyperoxemia. This paradoxic finding and other re-

between normoxemic and hyp
at any of the four studied stag
Cardiac index was increased during rewarmin
was otherwise similar at all stages. Pao_2 values
be held within the intended ranges during mea
ments, were significantly different during nor
emic and hyperoxemic conditions ($p < 0.001$)
did not overlap. Venous oxygen saturations
higher during hyperoxemia, reaching statistica
nificance during hypothermic CPB and after C

Tissue oxygenation. A common pattern d
change from normoxemia to hyperoxemia was

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

HYPEROXEMIE ET MICRO-BULLES

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

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

ETUDE CLINIQUE III:TROUBLES DU RYTHME POST-OPERATOIRES

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

PaO_2 < 150 mmHg avec possibilité d'augmenter la FiO_2 si SvO_2< 60% (n=167)

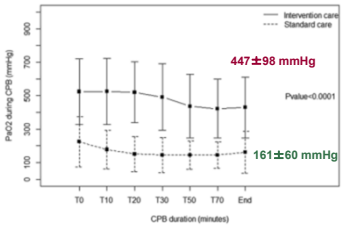
versus

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
Primary endpoint				
PAOF or VT/VF (n, %)	49 (30)	49 (30)	0.4% [− 9.6–10.4]	0.94
POAF (n, %)	48 (29)	47 (29)	0.0% [− 9.5–10.2]	0.94
VT/VF (n, %)	4 (2)	4 (2)	0.0% [− 3.3–3.4]	0.99
S d d i t				

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% [0.08–6.3]	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

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 (3.7–8.7)	5.5 (3.7–8.7)	0.76
Adverse clinical outcomes			
Mortality			
In-hospital	0 (0)	0 (0)	
After 30 days	0 (0)	1 (1.96)	0.32
After 6 months†	0 (0)	1 (2.56)	0.37
Stroke	0 (0)	0 (0)	
Pneumonia	3 (6.12)	1 (1.96)	0.36
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

ETUDE CLINIQUE 1I: 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)

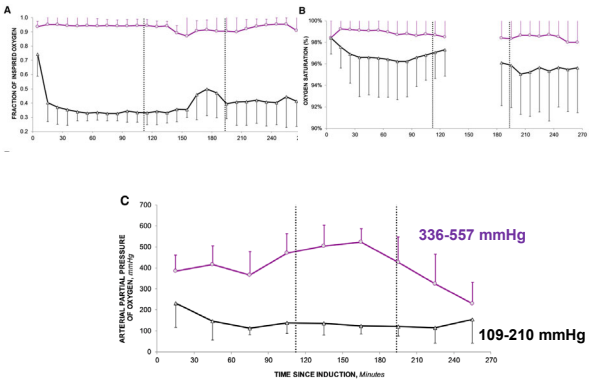
versus

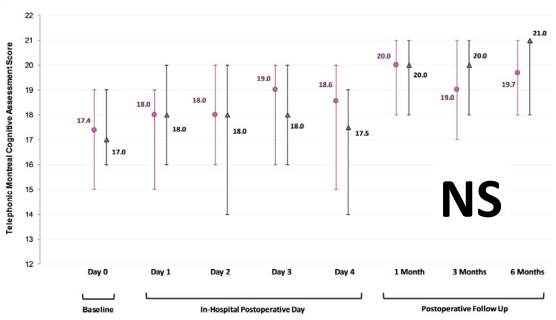
FiO2= 100% durant toute la chirurgie (n=49)

CJP= Montreal Cognitive Assessment Score (J2 postopératoire)

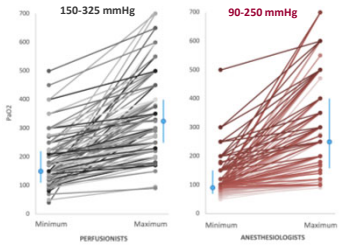
CJS= MCAS à M1, M3, M6 et morbi-mortalité postopératoire

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





QUELLE CIBLE? LES AVIS DIVERGENT…

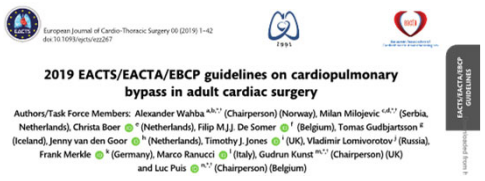


« Too low » value?

Perfu = 100 mmHg

MAR = 60 mmHg

Calhoun A et al. J Cardiothorac Vasc Anesth 2022



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

Authors/Task Force Members: Alexander Wahba [a,b,*,†] (Chairperson) (Norway), Milan Milojevic [c,d,*,†] (Serbia, Netherlands), Christa Boer [e] (Netherlands), Filip M.J.J. De Somer [f] (Belgium), Tomas Gudbjartsson [g] (Iceland), Jenny van den Goor [h] (Netherlands), Timothy J. Jones [i] (UK), Vladimir Lomivorotov [j] (Russia), Frank Merkle [k] (Germany), Marco Ranucci [l] (Italy), Gudrun Kunst [m,†,†] (Chairperson) (UK) and Luc Puis [n,*,†] (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 pro-longed 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 clinque?
- Cible **100-120 mmHg QSP > 96%**
- Meilleure interprétation SvO_2
- Hyperoxémie discutable en pré-CEC (induction anesthésique)

Recommendation Table 41 Recommendations for pump flow management during cardiopulmonary bypass

Recommendations	Class [a]	Level [b]	Ref [c]
It is recommended that the estimated pump flow rate be determined before the initiation of CPB based on the BSA and the planned level of hypothermia.	I	C	-
The adequacy of the pump flow rate during CPB should be considered based on oxygenation and metabolic parameters (SVO_2, O_2ER, rSO_2, VCO_2, VCO_2/VO_2 and arterial blood lactate levels). [d] No validated threshold presently exists.	IIa	B	213,542,543
It is recommended that a minimal value of DO_2 of 280 ml/min/m^2 be used to reduce the risk of AKI stage 1.	I	A	533–536,545,546
Pump flow rates calculated on the basis of lean body mass may be considered as a suggested lower value in patients with obesity.	IIb	B	528

Recommendation Table 5 Recommendations for the use of carbon dioxide flush

Recommendations	Class [a]	Level [b]	Ref [c]
It is recommended that CO_2 flush of the CPB circuit before priming is established as the standard of care to reduce GME.	I	B	67,68
CO_2 insufflation of the operative field may be considered to reduce emboli.	IIb	B	71
When CO_2 insufflation of the surgical field is used, it is recommended that gas flow be adapted to avoid hypercapnic acidosis.	I	C	–

Recommendation Table 43 Recommendations for goal-directed perfusion

Recommendations	Class [a]	Level [b]	Ref [c]
GDP is recommended to reduce the postoperative rate of early stages of acute kidney injury.	I	A	545,546,568
It is recommended that GDP be aimed at limiting the nadir of DO_2 and the length of CPB time with low DO_2 values.	I	B	570–572
It may be considered that individualized DO_2 based on preoperative risk factors, peripheral oxygenation and pulse pressure, be identified preoperatively and maintained during CPB.	IIb	B	570,574
It should be considered to maintain GDP with a lower threshold of DO_2 between 280 and 300 ml/min/m^2 during normothermic CPB in order to improve clinical outcomes.	IIa	B	568–571