Echanges gazeux appliqués à la CEC

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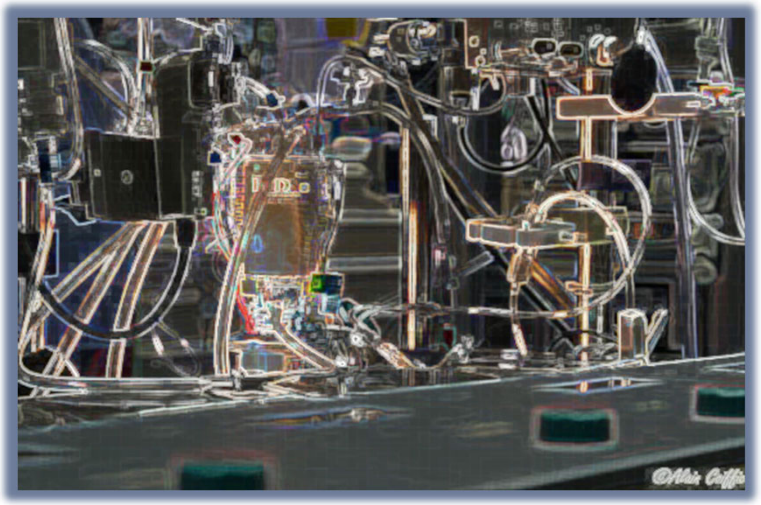

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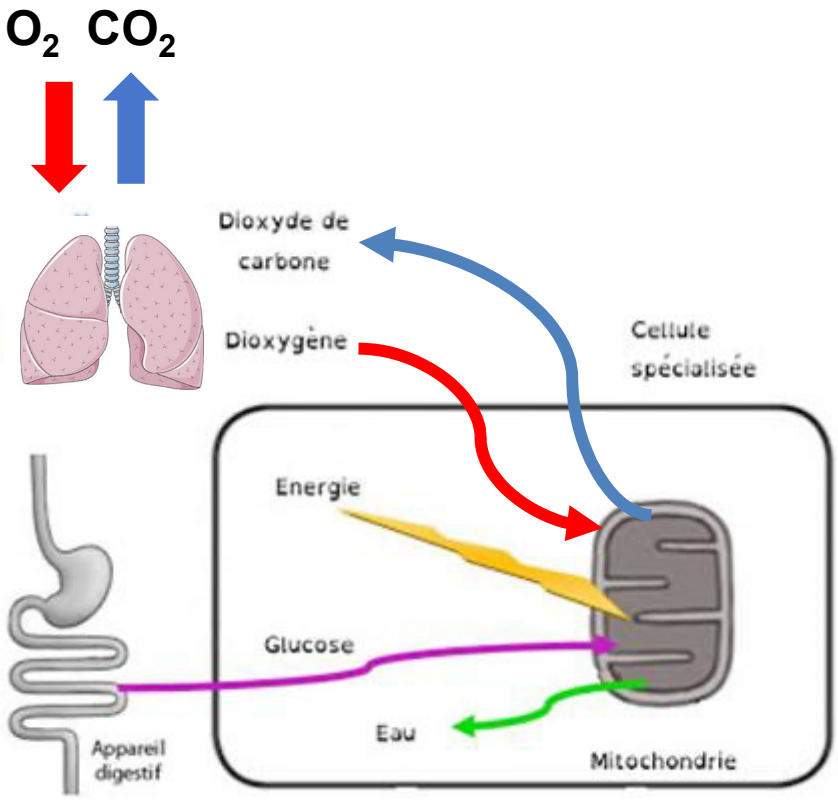
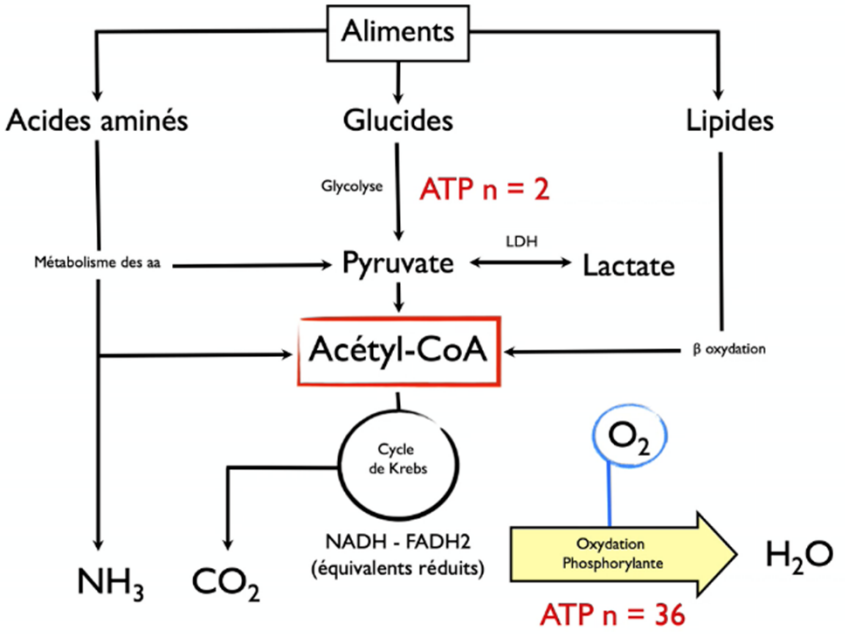
Inserm U1034

Respiration cellulaire

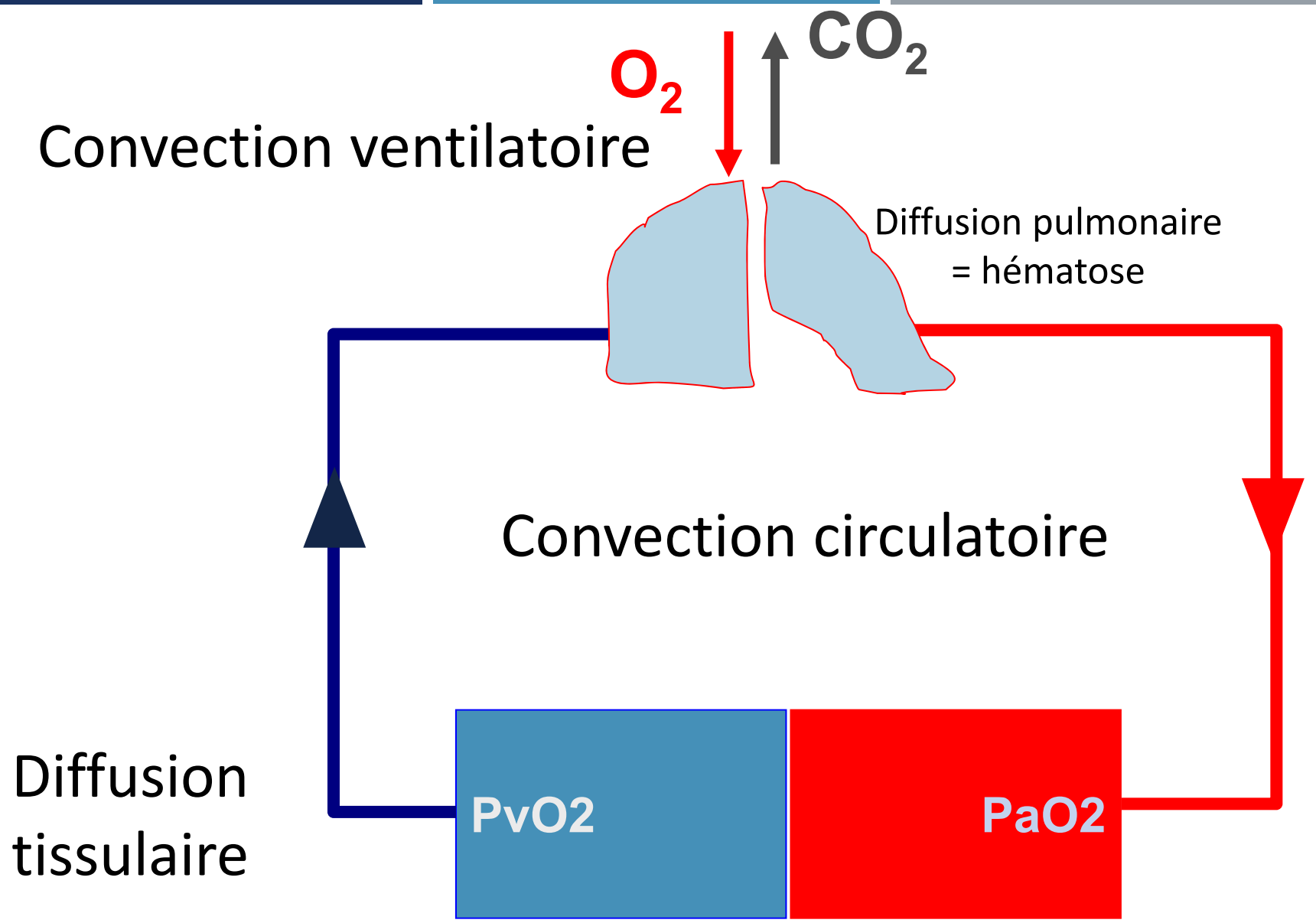




PRIMUM MOVENS CELLULAIRE

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

LA POMPE CARDIO-THORACIQUE


O_2
CO_2
Convection ventilatoire
Diffusion pulmonaire
= hématose
Convection circulatoire
Diffusion
tissulaire
PvO2
PaO2

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)

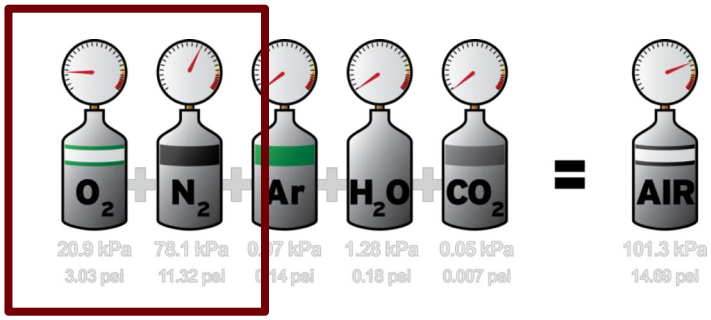
- Pression partielle P d'un gaz donné « x » est égale au produit de la pression totale, P_T, par la fraction F, de ce gaz dans le mélange

- Somme des pressions partielles est égale à la pression totale du mélange

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

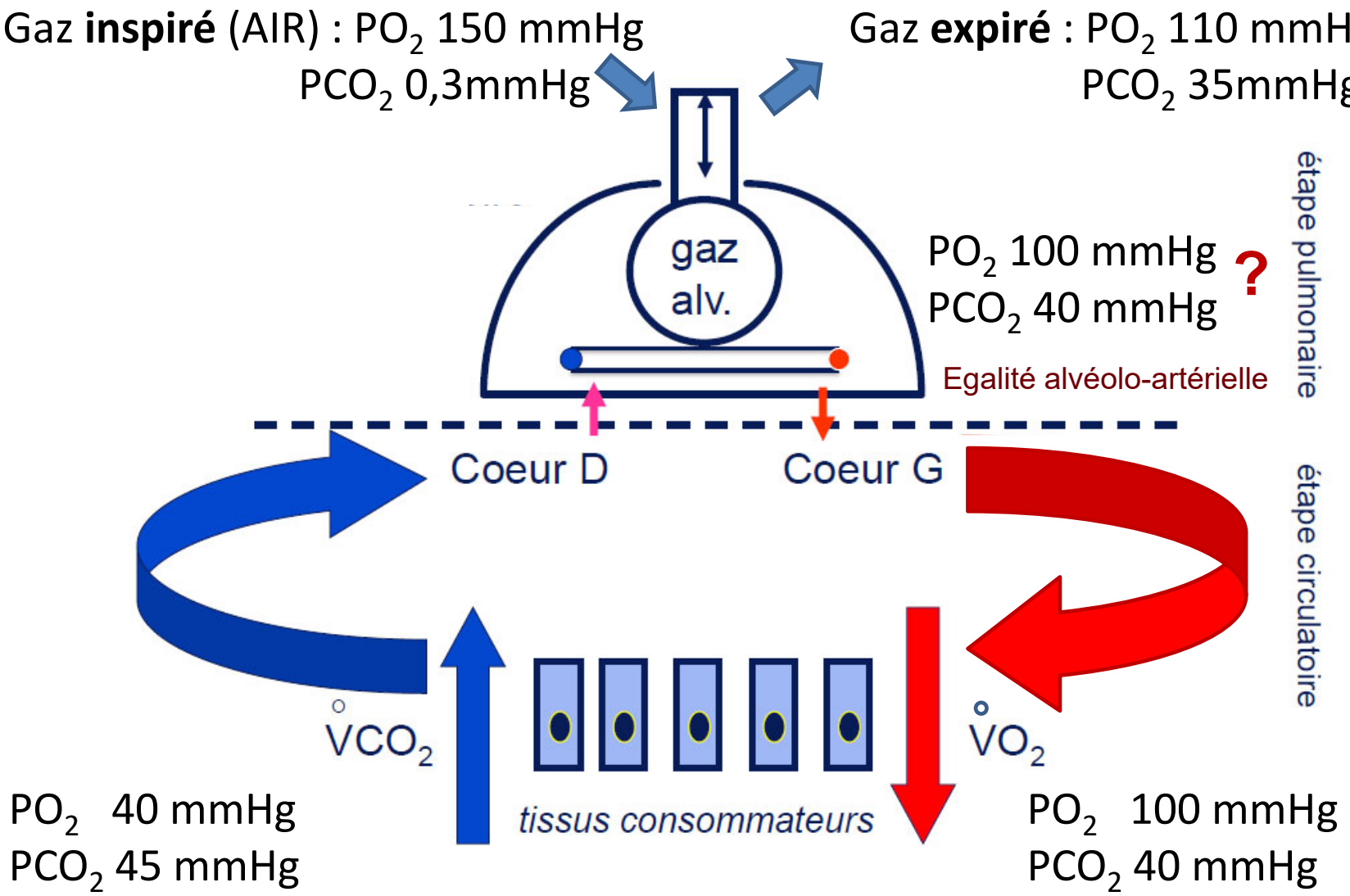
Composition de l'air ambiant

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**

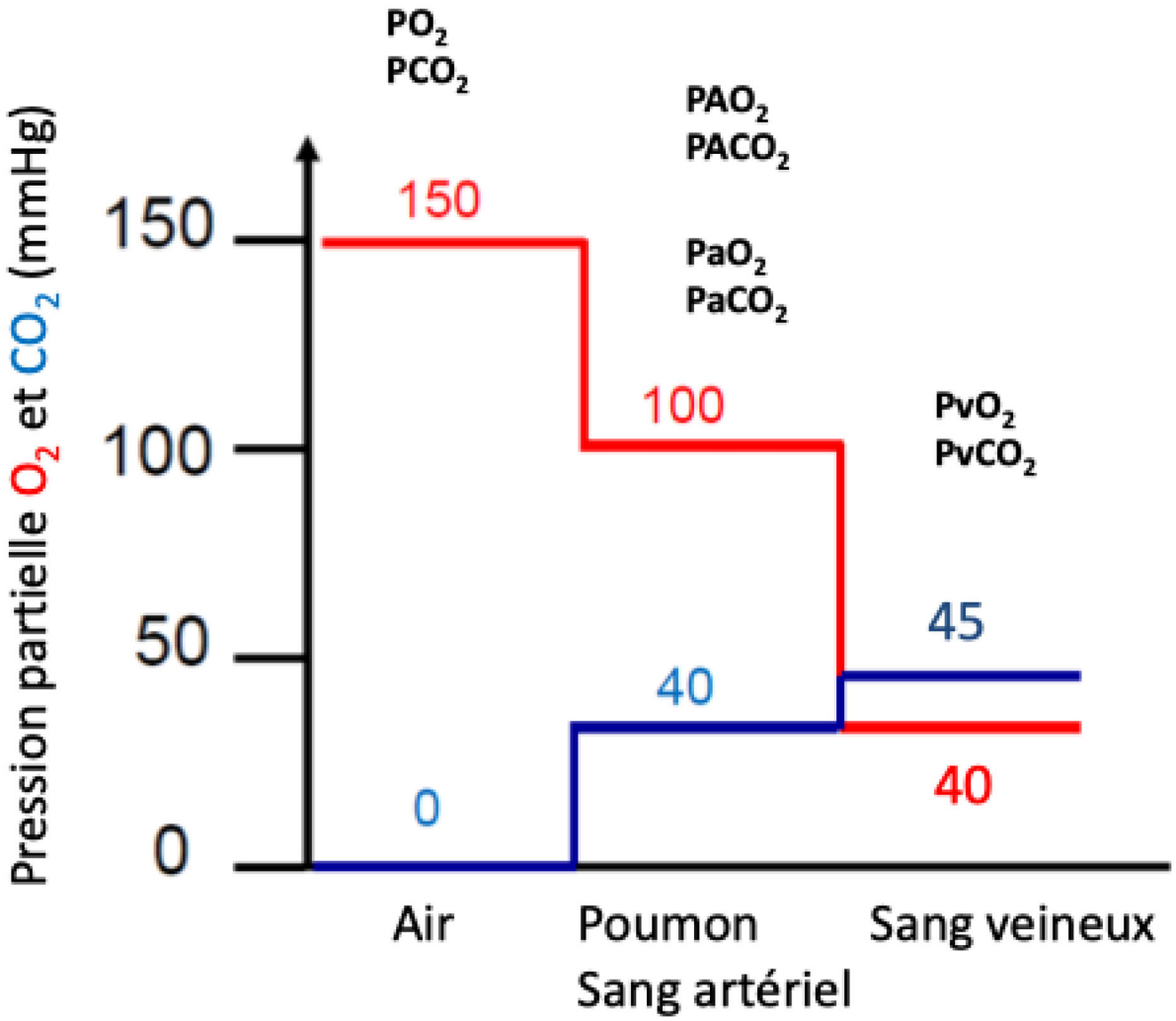


P_{GAZ} = Fraction X $P_{atmosphérique}$

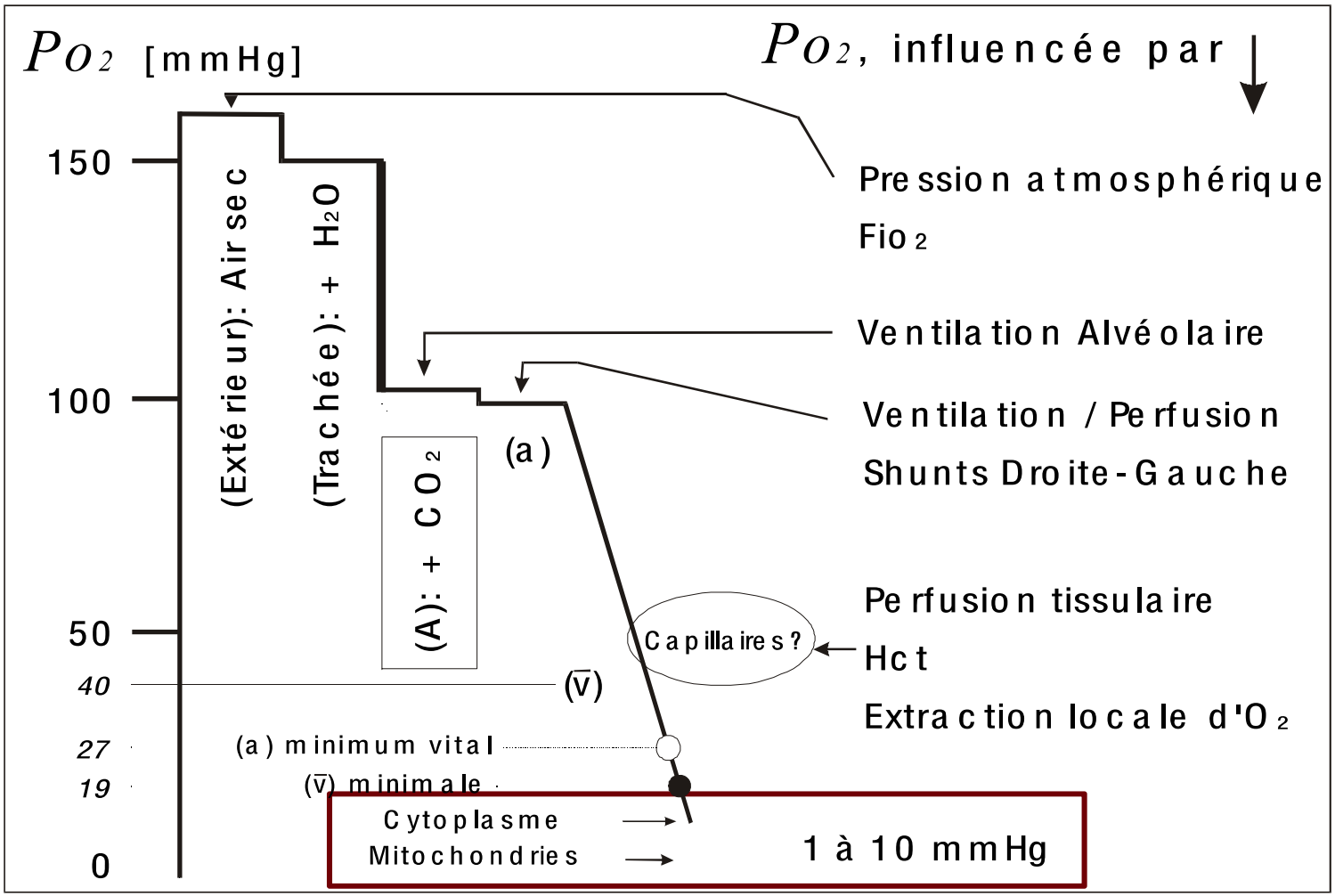
P_{ATM} = 760 mmHg au niveau de la mer


Gaz **inspiré** (AIR) : PO_2 150 mmHg
PCO_2 0,3mmHg
Gaz **expiré** : PO_2 110 mmHg
PCO_2 35mmHg
gaz alv.
PO_2 100 mmHg ?
PCO_2 40 mmHg
Egalité alvéolo-artérielle
étape pulmonaire
Coeur D
Coeur G
étape circulatoire
$\dot{V}CO_2$
$\dot{V}O_2$
tissus consommateurs
PO_2 40 mmHg
PCO_2 45 mmHg
PO_2 100 mmHg
PCO_2 40 mmHg

Cascade des PO_2 et PCO_2



Cascade des PO_2

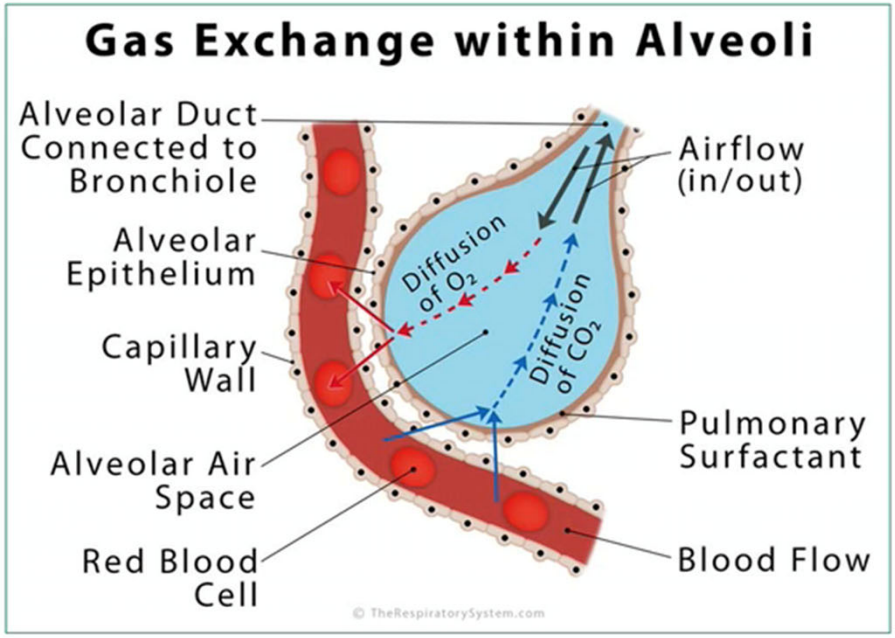


ETAPE PULMONAIRE:TRANSFERT DES GAZ

- **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)

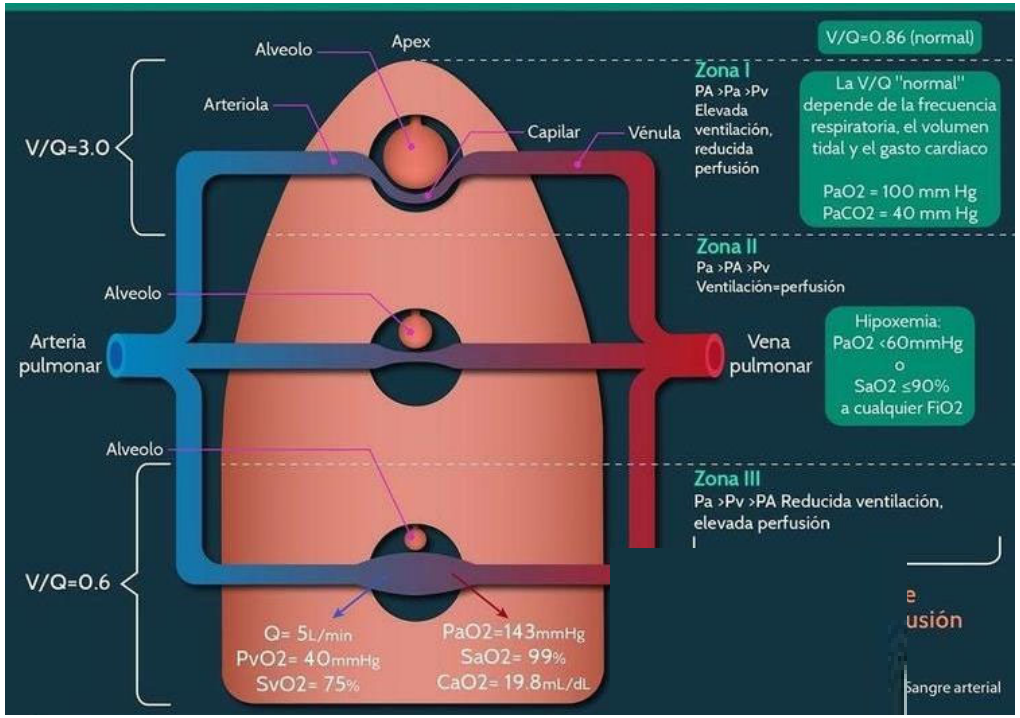
A: surface d'échange

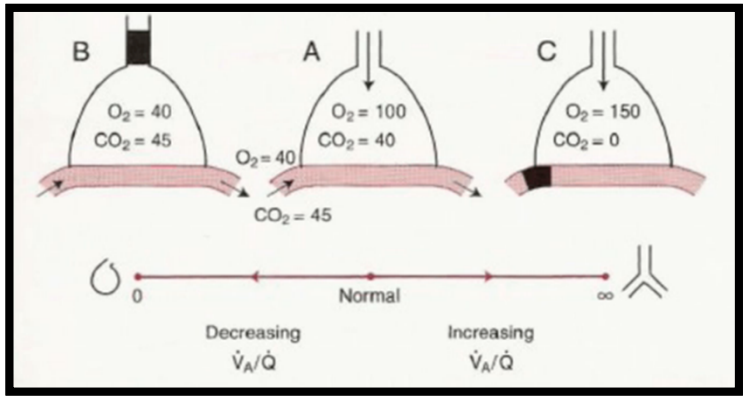
D: épaisseur de la membrane

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

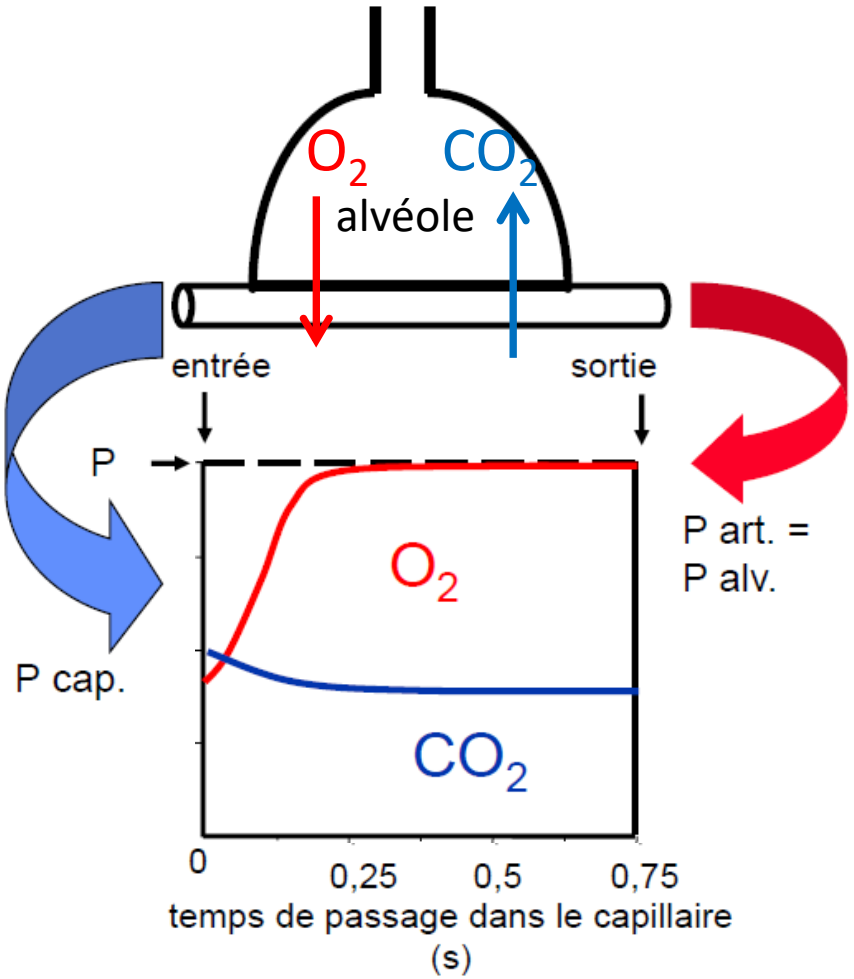
Rapport ventilation-perfusion

Zone de West I-III

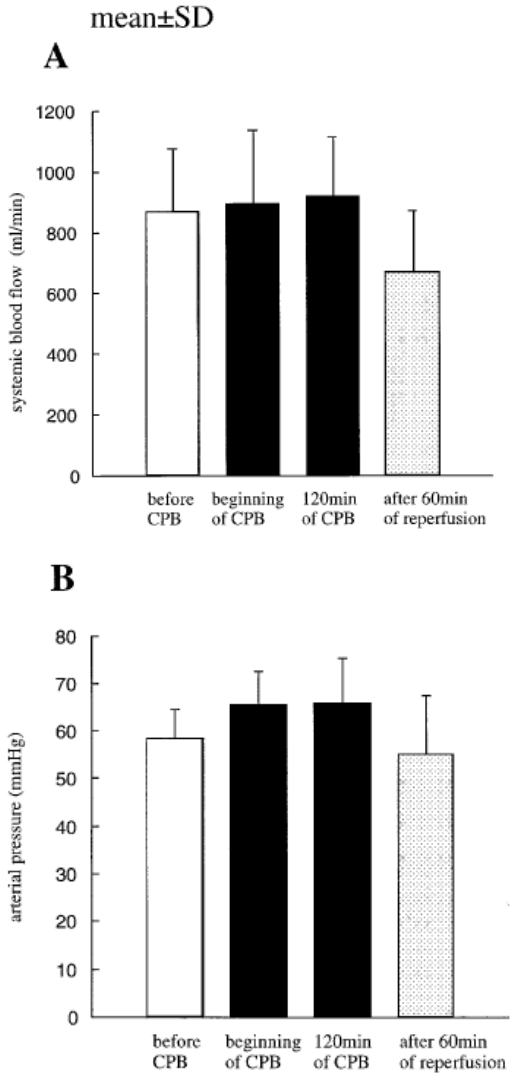


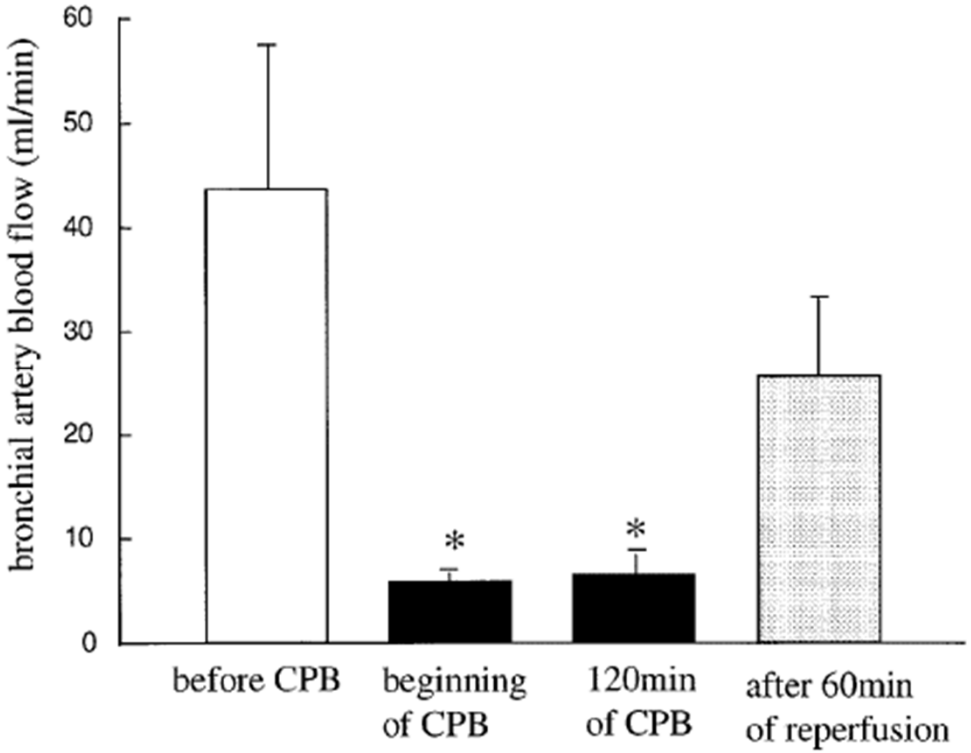


Temps de transit dans la barrière alvéolo-capillaire (effet $Pv0_2$)



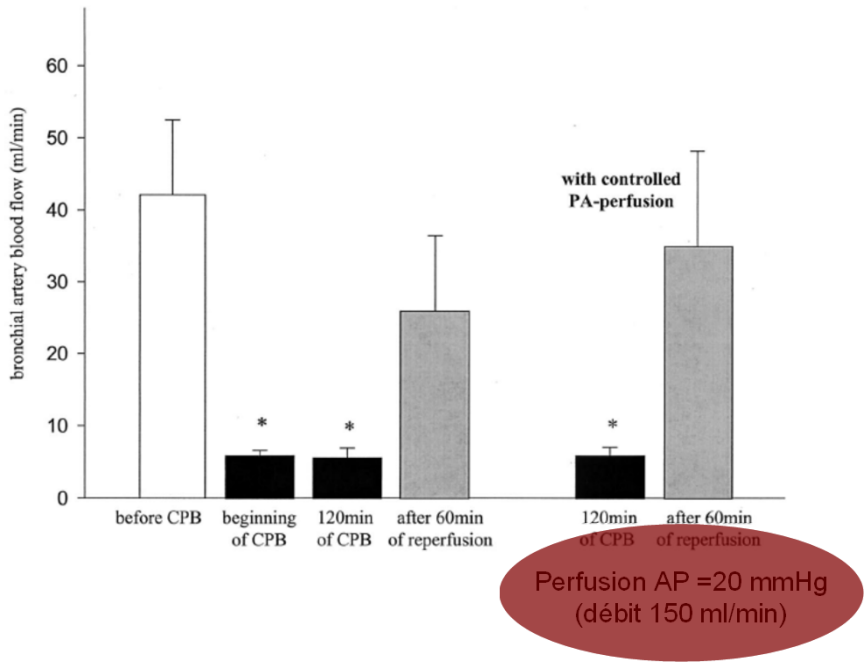
Bronchial artery perfusion during cardiopulmonary bypass does not prevent ischemia of the lung in piglets: assessment of bronchial artery blood flow with fluorescent microspheres☆

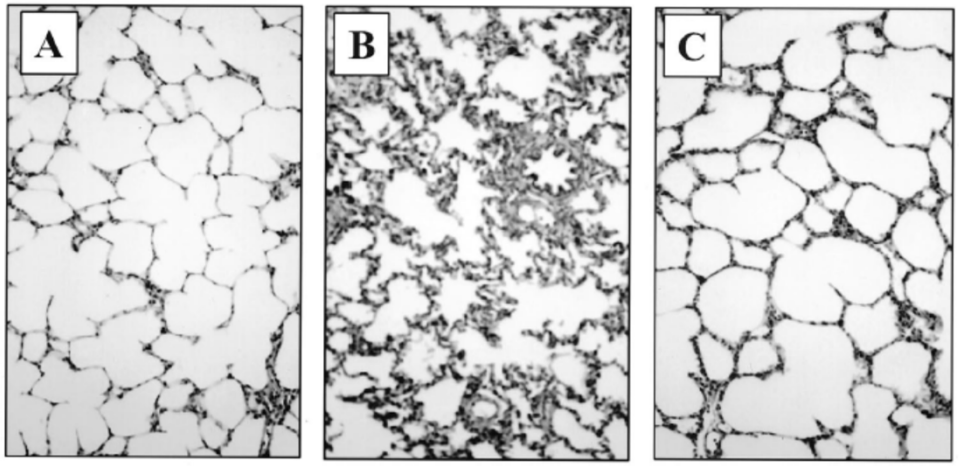




Schlensak C et al. Eur J Cardioth Surg 2001; 19:329-32

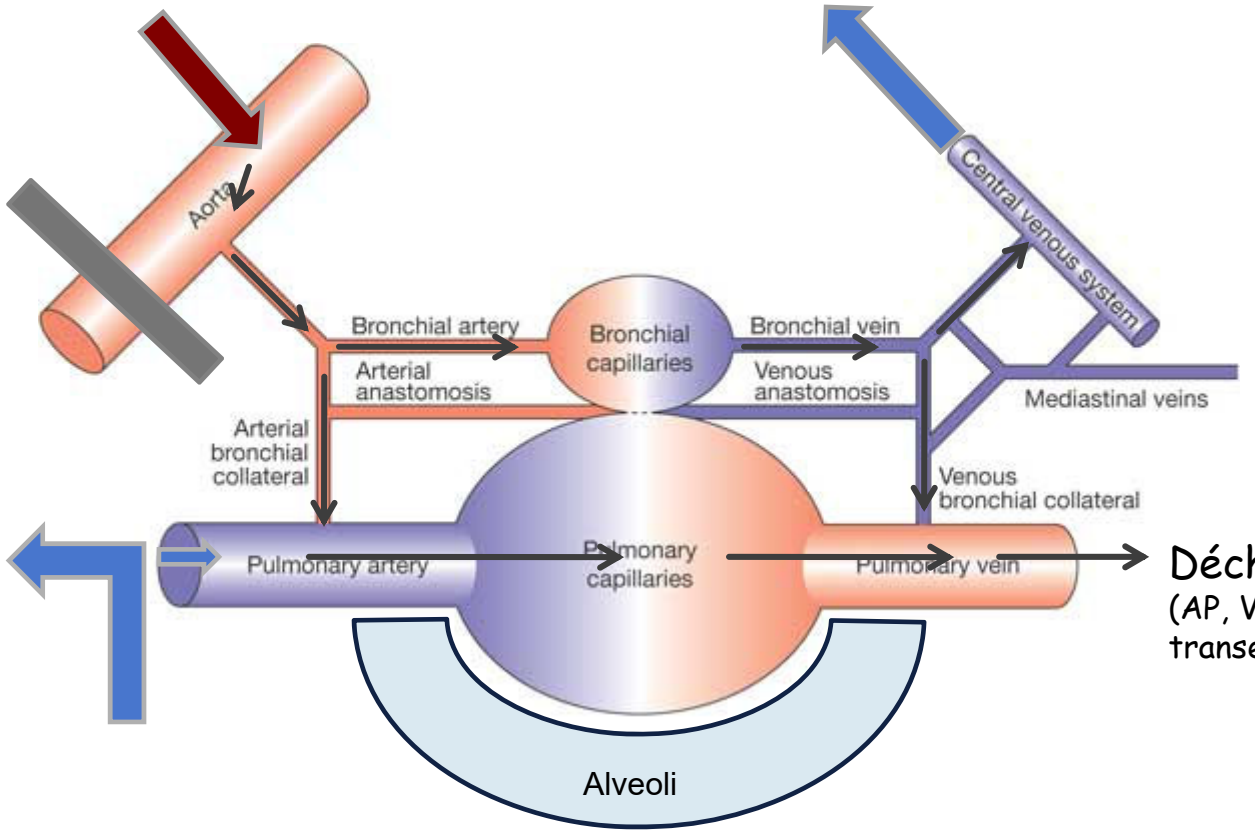
Schlensak C et al. J Thorac Vasc Surg 2002;123:1199-205





BL **PAP -** **PAP +**

Rappels anatomiques
« Shunts physiologiques de la circulation du poumon »



Décharge gauche
(AP, VP, racine aortique, transeptale ou point VG…)

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

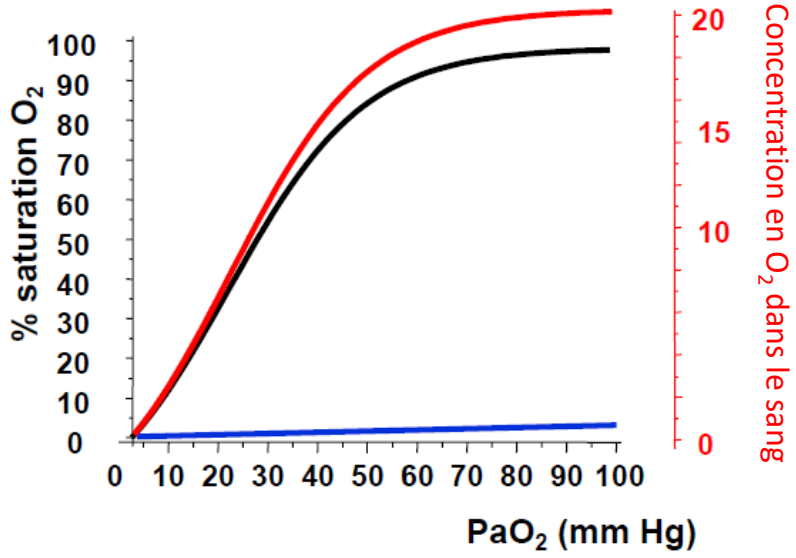
Contenu de l'oxygène dans le sang

$CaO_2 = O_2$ transporté par Hb + O_2 dissous

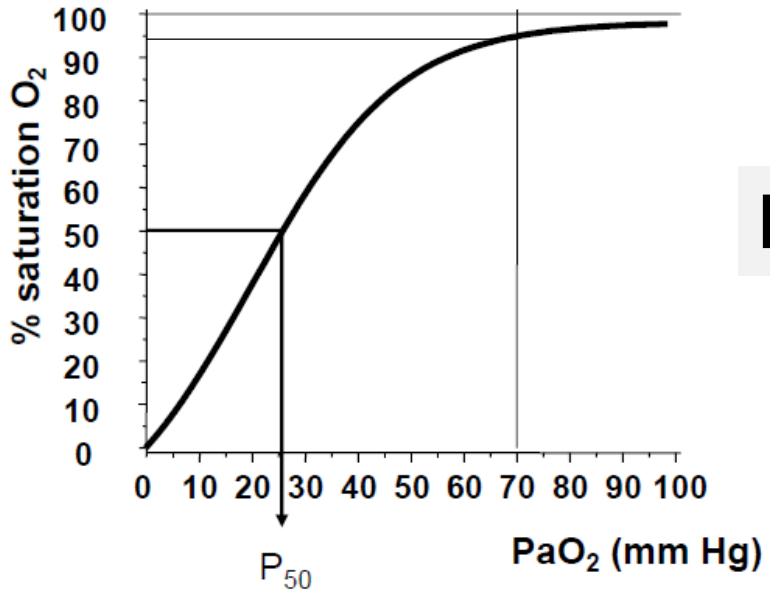
$$CaO_2 = (SaO_2 \times Hb \times 1{,}34) + PaO_2 \times 0{,}031$$

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

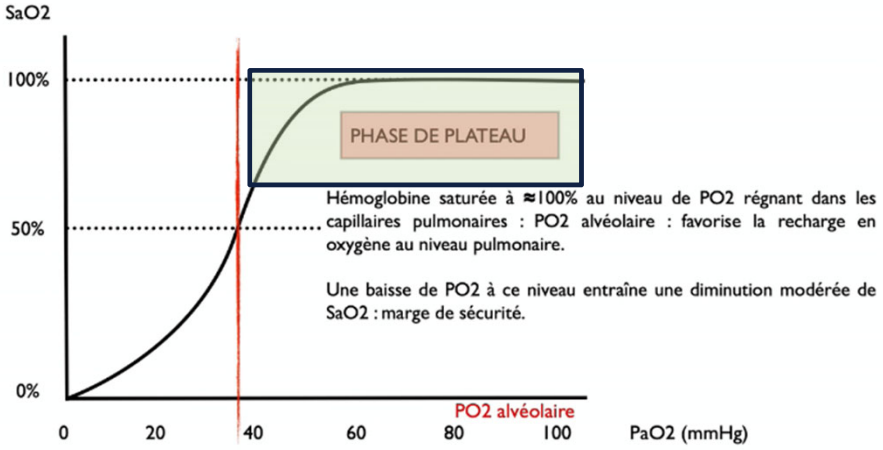
Coefficient de solubilité de l'O_2 dans le sang (en mL d'O2/L sang/mmHg)

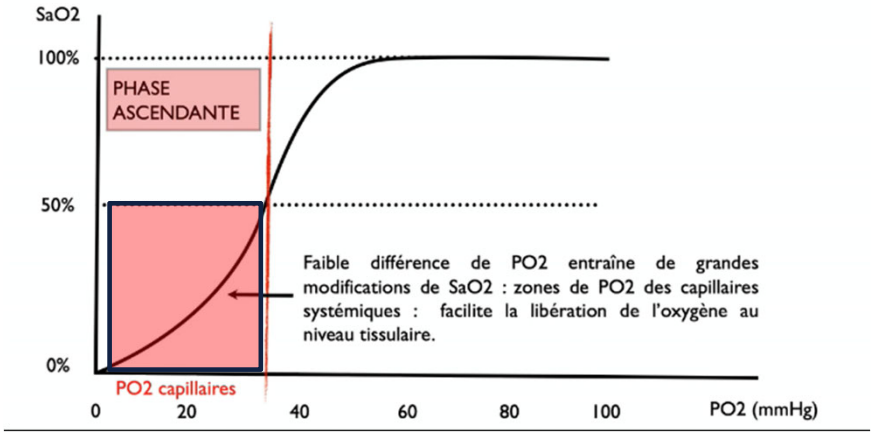


Courbe de BARCROFT

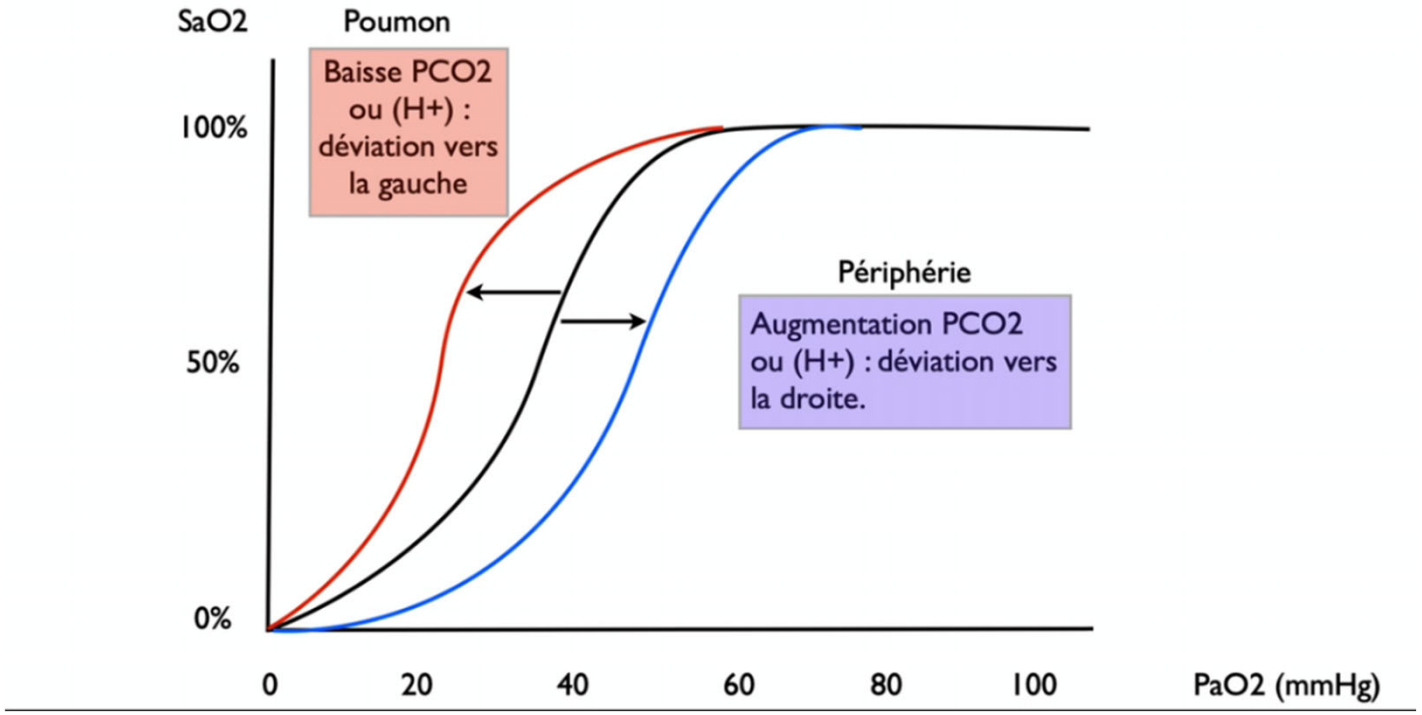


P_{50} = 27 mmHg

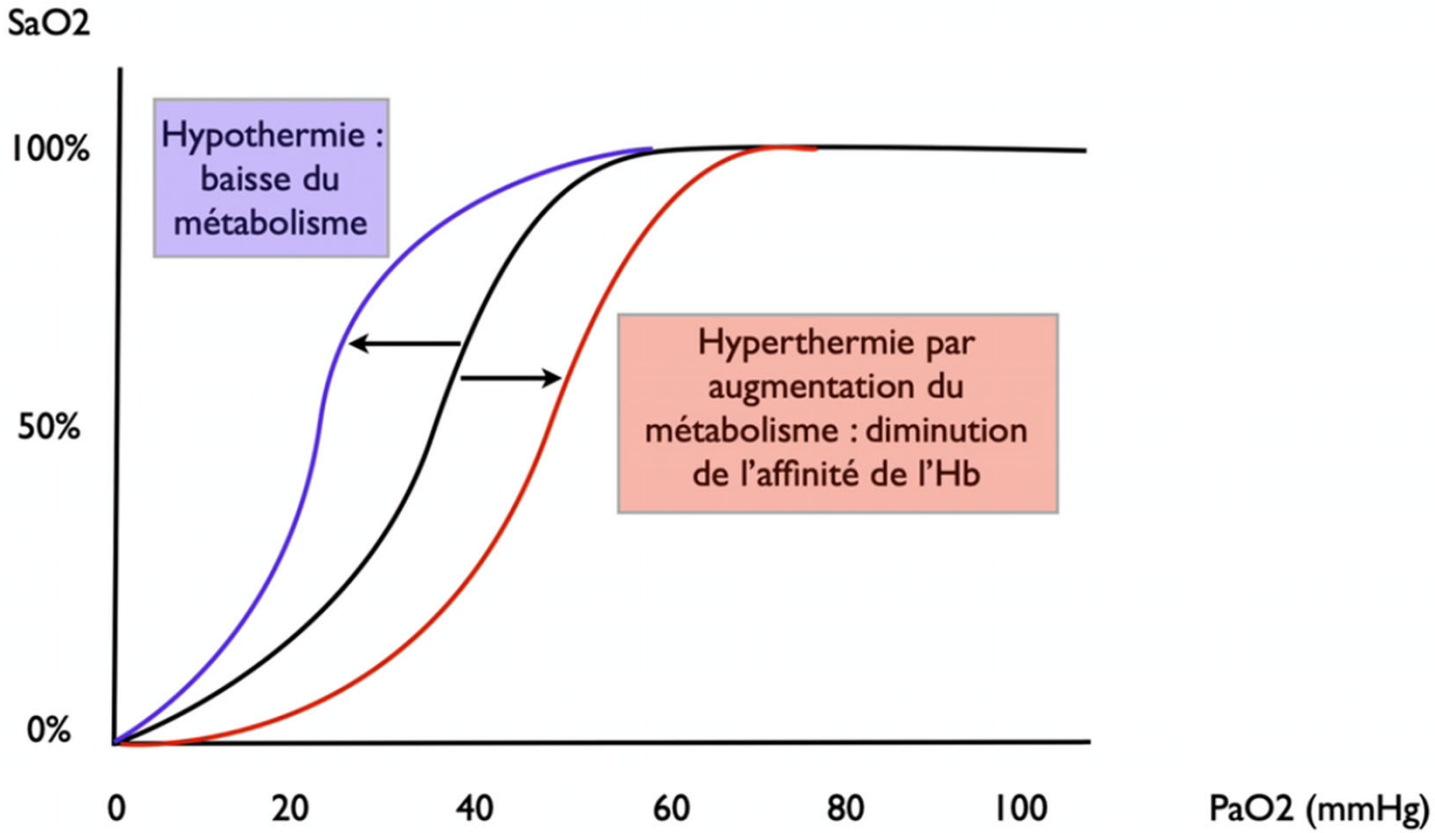


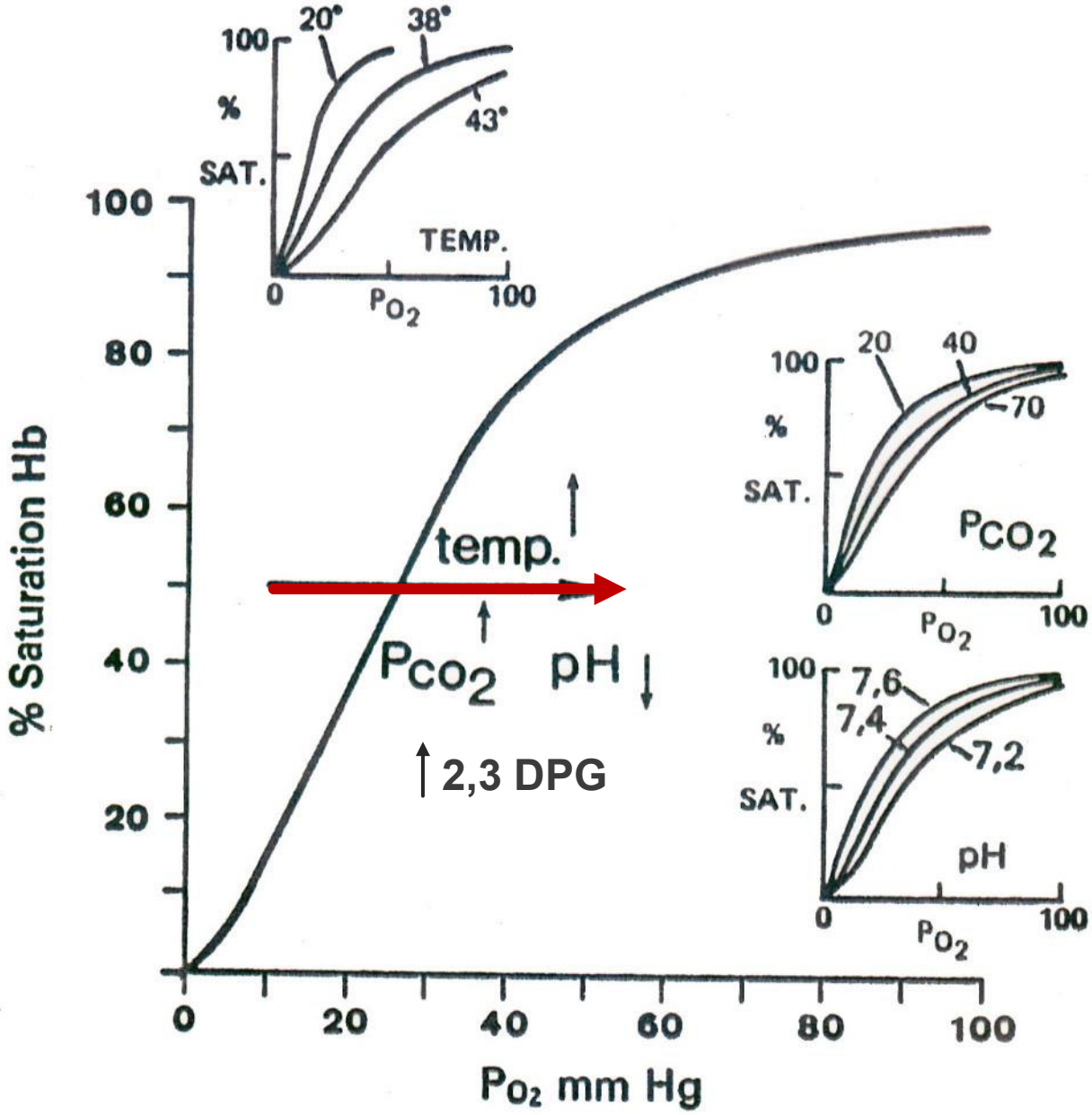


EFFET BOHR*



Physiologiste Danois (1904)


SaO2
100%
50%
0%
Hypothermie : baisse du métabolisme
Hyperthermie par augmentation du métabolisme : diminution de l'affinité de l'Hb
0
20
40
60
80
100
PaO2 (mmHg)


% Saturation Hb
100
80
60
40
20
0
20
40
60
80
100
Po2 mm Hg
temp.
Pco2
pH
2,3 DPG
100
%
SAT.
20°
38°
43°
TEMP.
0
Po2
100
100
%
SAT.
20
40
70
Pco2
0
Po2
100
100
%
SAT.
7,6
7,4
7,2
pH
0
Po2
100

TRANSPORT EN OXYGÈNE

DO_2 (TaO_2) = DC X CaO2

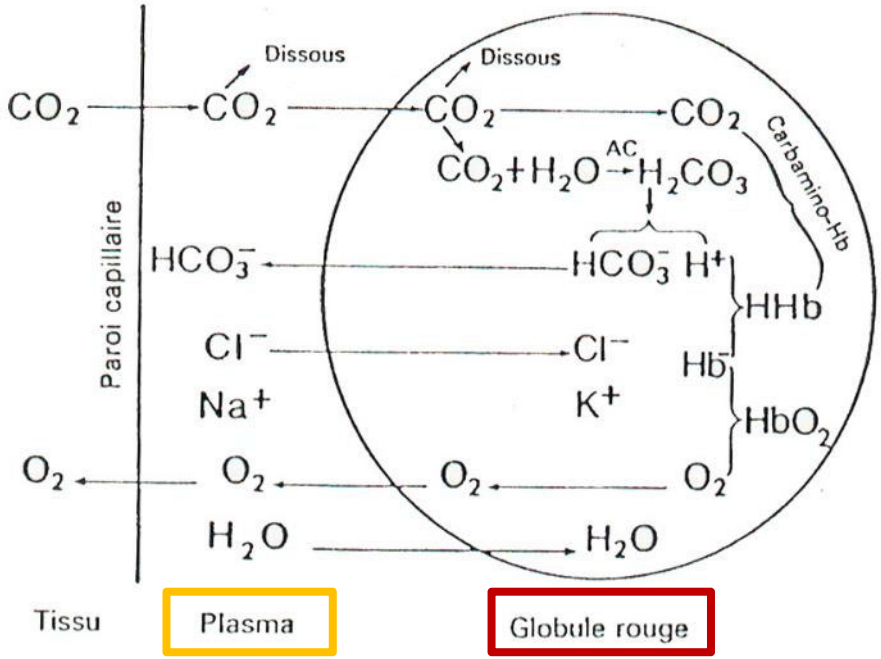
= VES X FC X (SaO2X Hb X 1,34 + 0,03 X PaO2)

= **VES X FC X SaO2 X Hb x 1,34**

= **Débit pompe X SaO2 X Hb X 1, 34**

(Valeur normale 10 ml d'O_2/kg/min ou 500 ml/min/m^2)
et VO_2 3,5 ml/kg/min

TRANSPORT EN CO_2



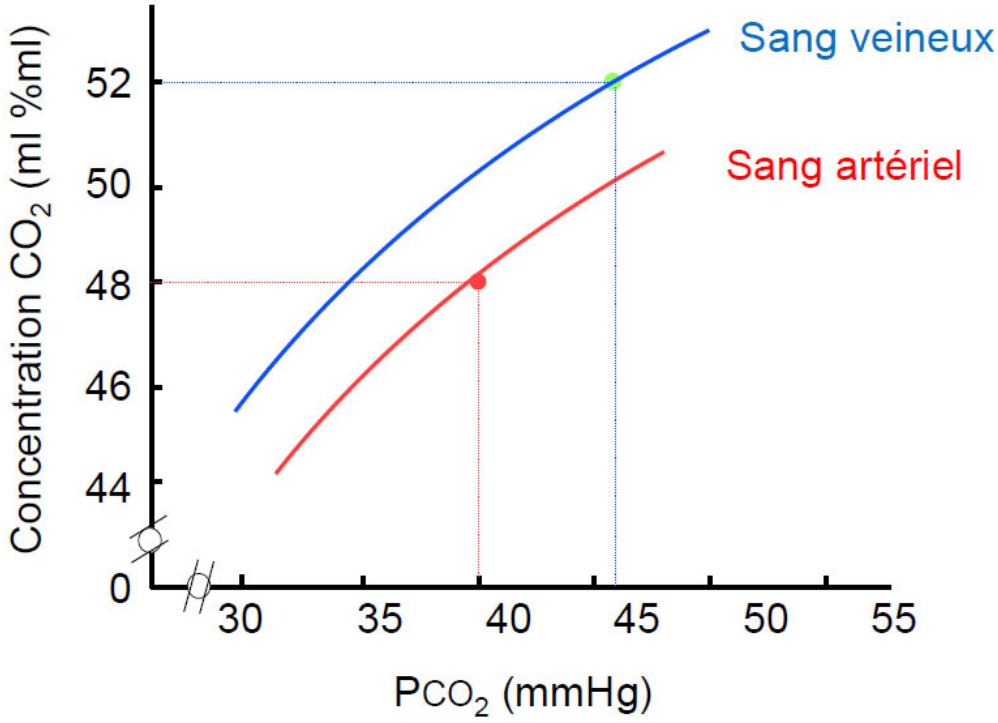
$$H_2O + CO_2 \overset{AC}{\rightleftharpoons} H_2CO_3 \overset{AC}{\rightleftharpoons} HCO_3^- + H^+.$$

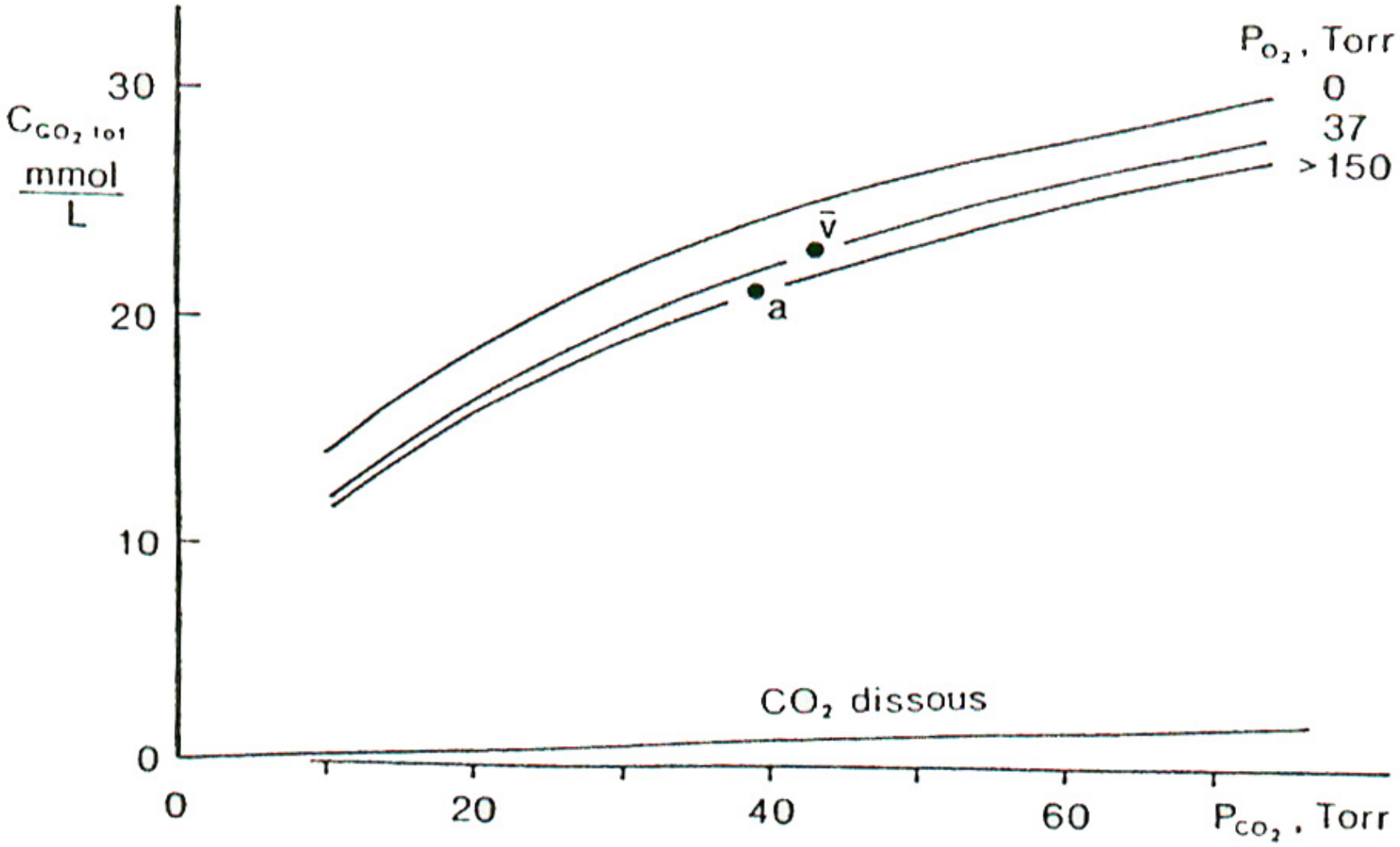
EFFET HALDANE

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

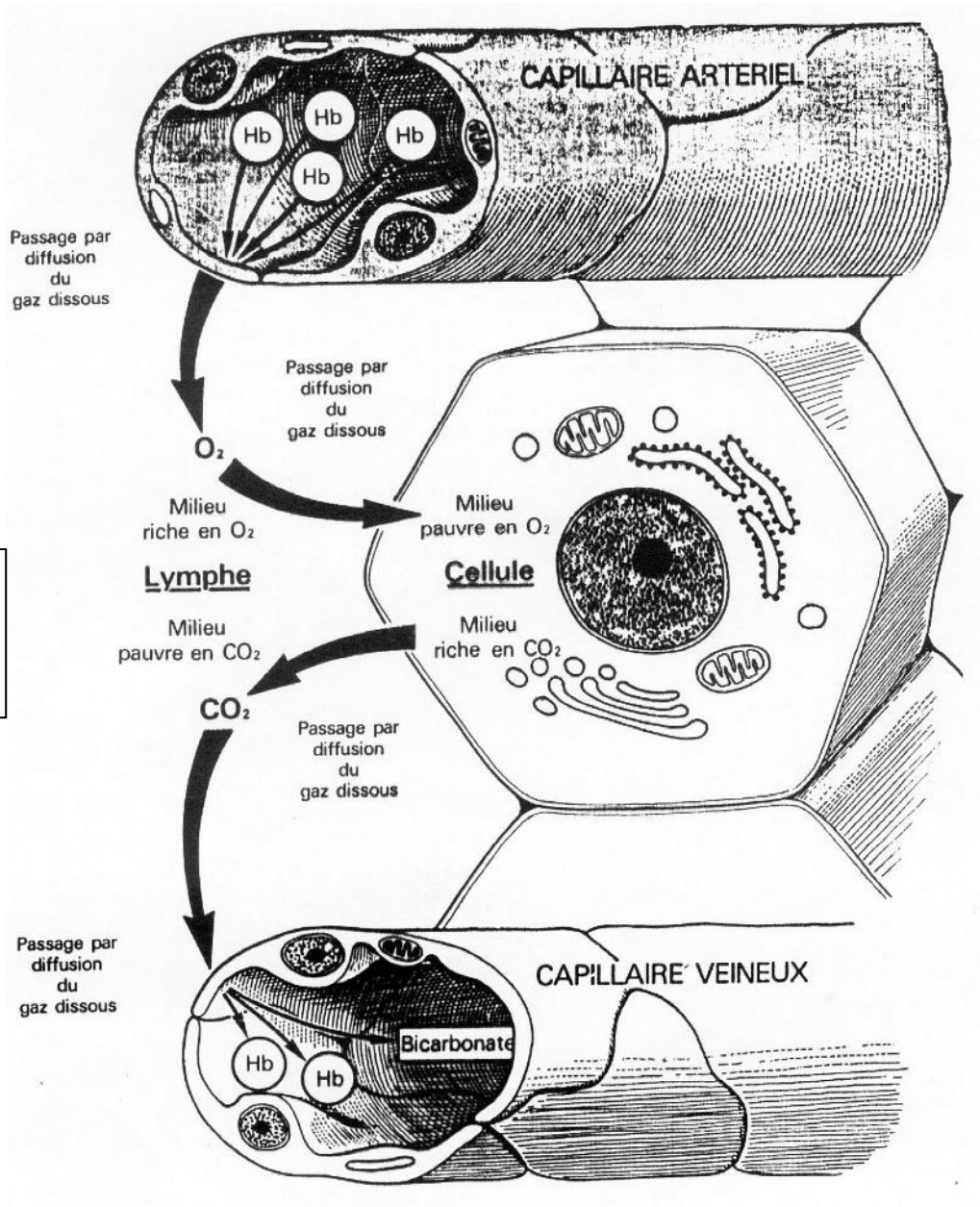


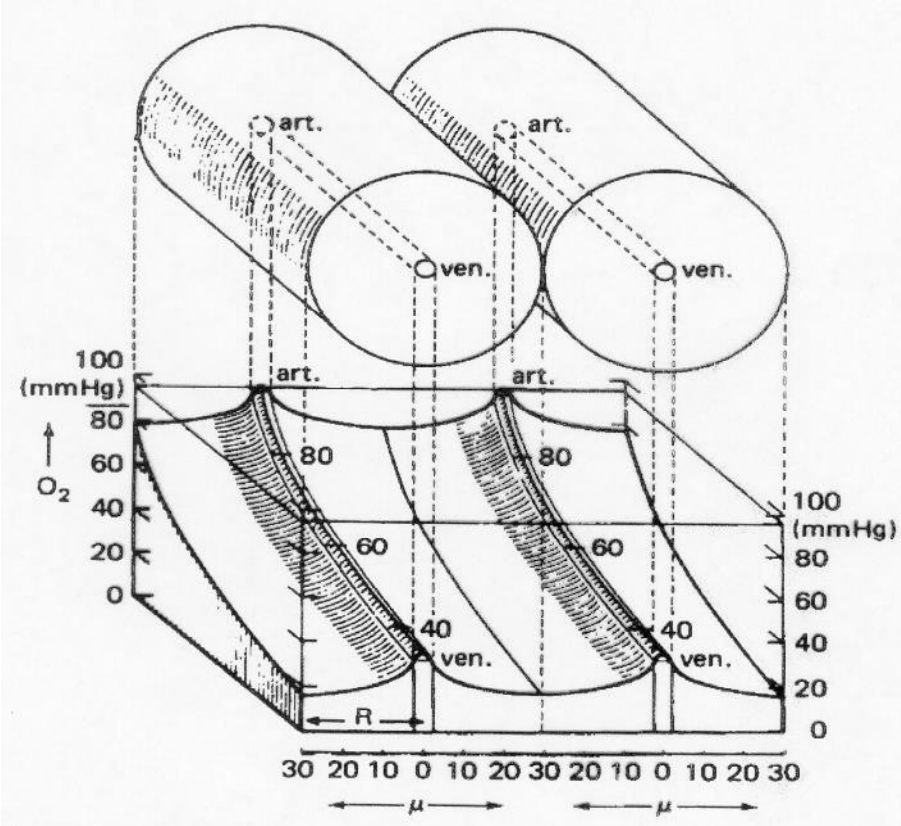

$C_{CO_2\ tot}$
mmol
L
30
20
10
0
0
20
40
60
P_{CO_2}, Torr
P_{O_2}, Torr
0
37
>150
$\bar{v}$
a
CO_2 dissous

Diffusion tissulaire

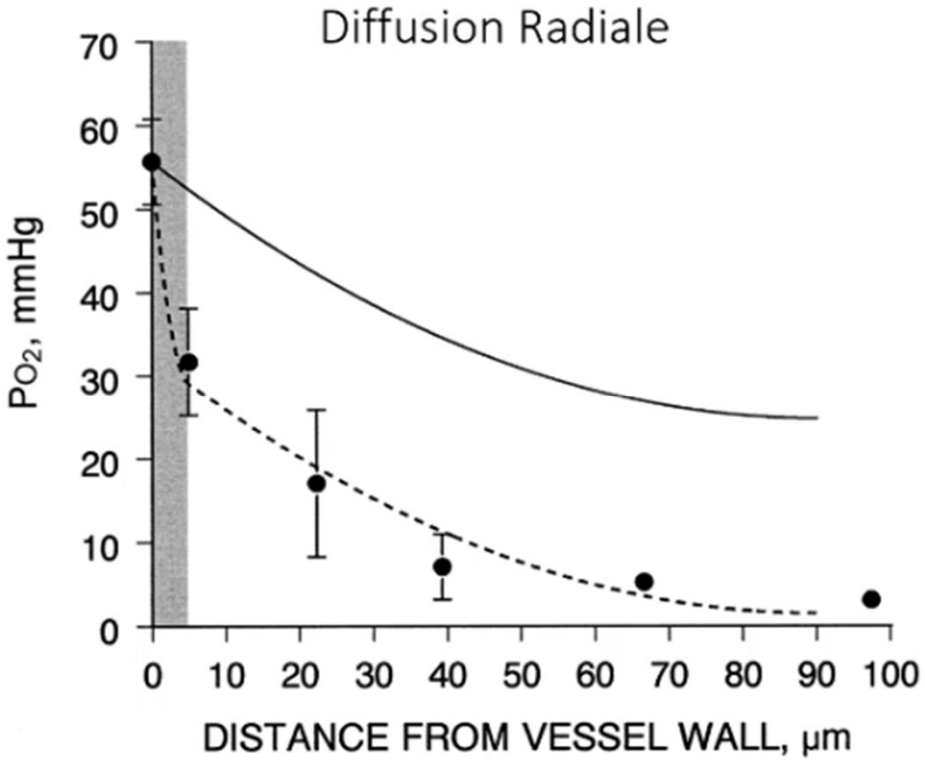
- Deux étapes, au travers:
  - Capillaire systémique
  - Espace péri-capillaire

CO_2 est 21 fois plus diffusible que O_2 +++

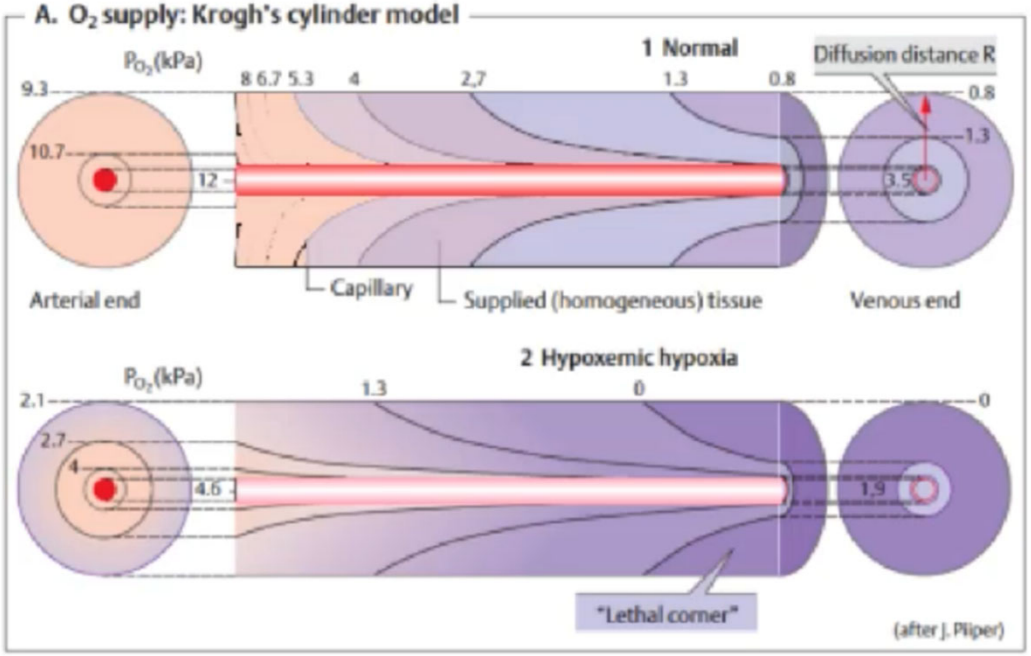




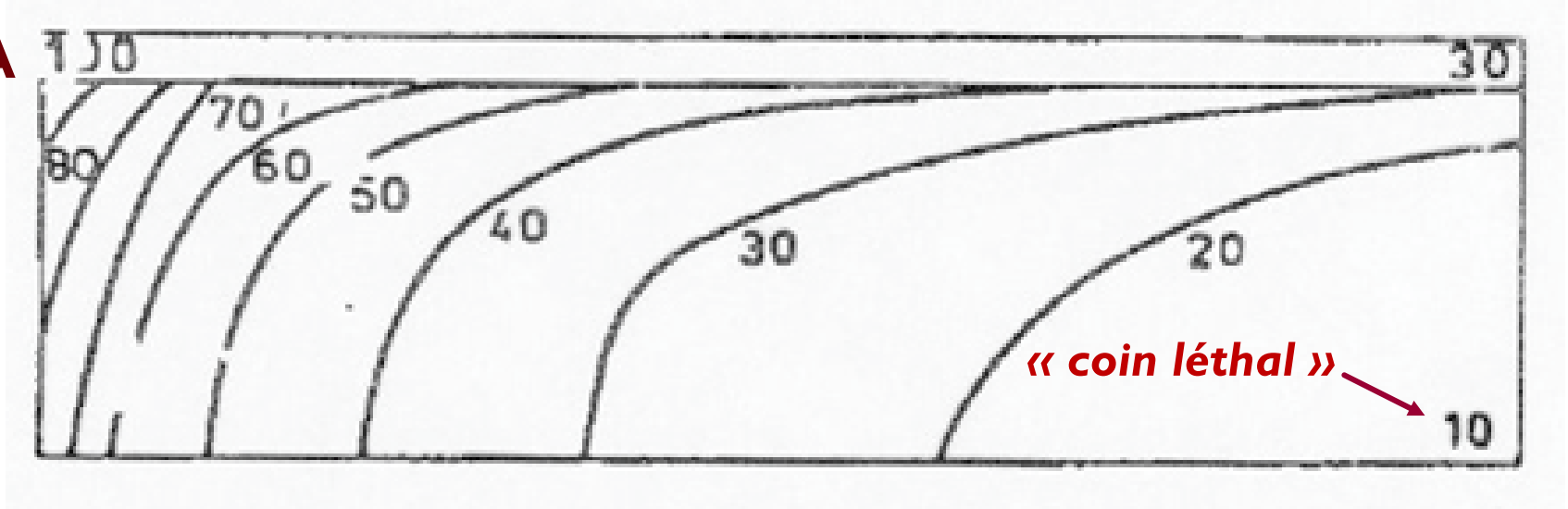
Diffusion longitudinale



Tsai et al. Physiol Rev 2003

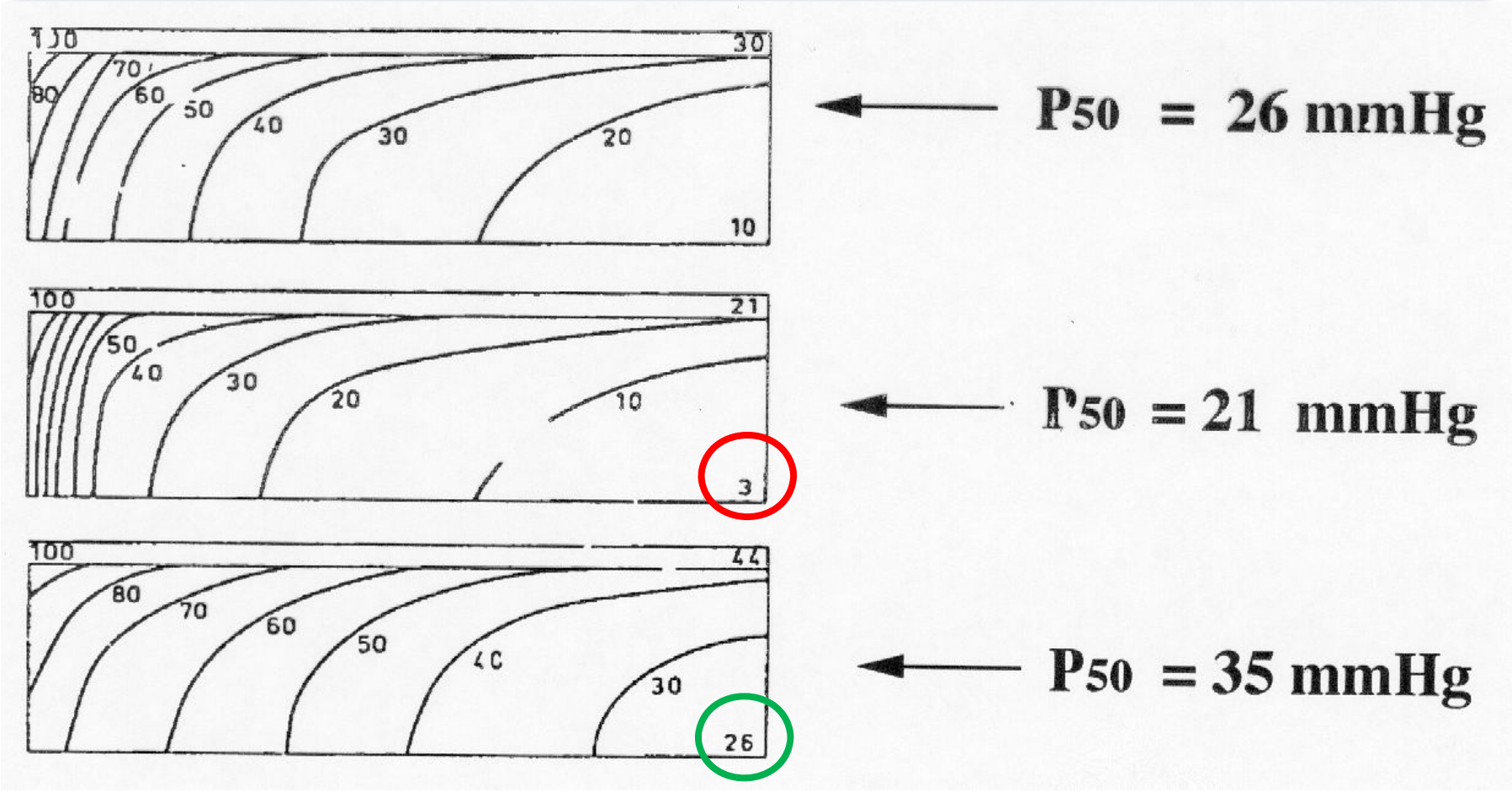

A. O_2 supply: Krogh's cylinder model
1 Normal
P_{O_2}(kPa)
9.3
8 6.7 5.3
4
2,7
1.3
0.8
Diffusion distance R
0.8
1.3
10.7
12
3.5
Arterial end
Capillary
Supplied (homogeneous) tissue
Venous end
2 Hypoxemic hypoxia
P_{O_2}(kPa)
2.1
1.3
0
0
2.7
4
4.6
1.9
"Lethal corner"
(after J. Piiper)

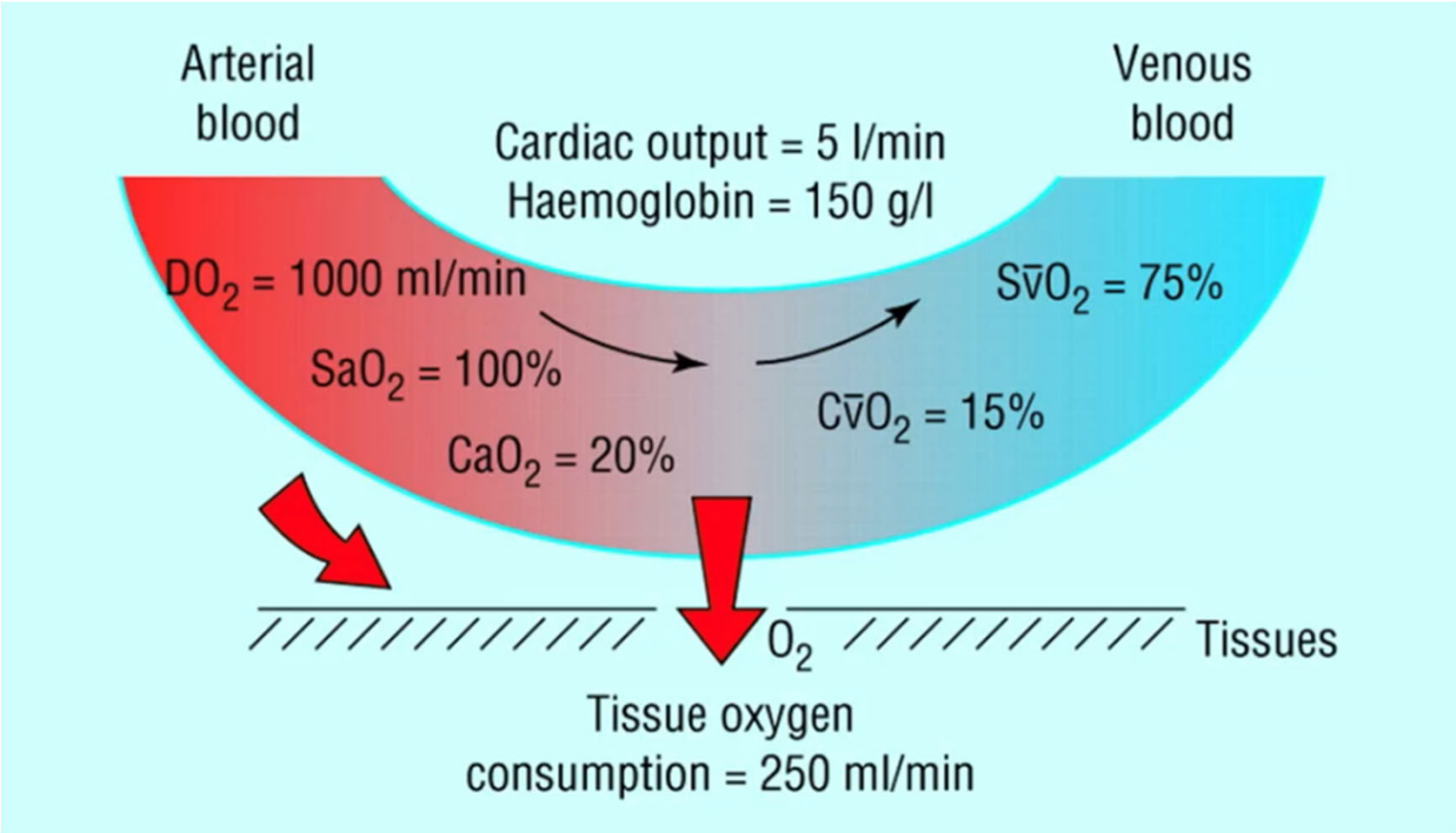
A


100
90
70
60
50
40
30
20
10
« coin léthal »

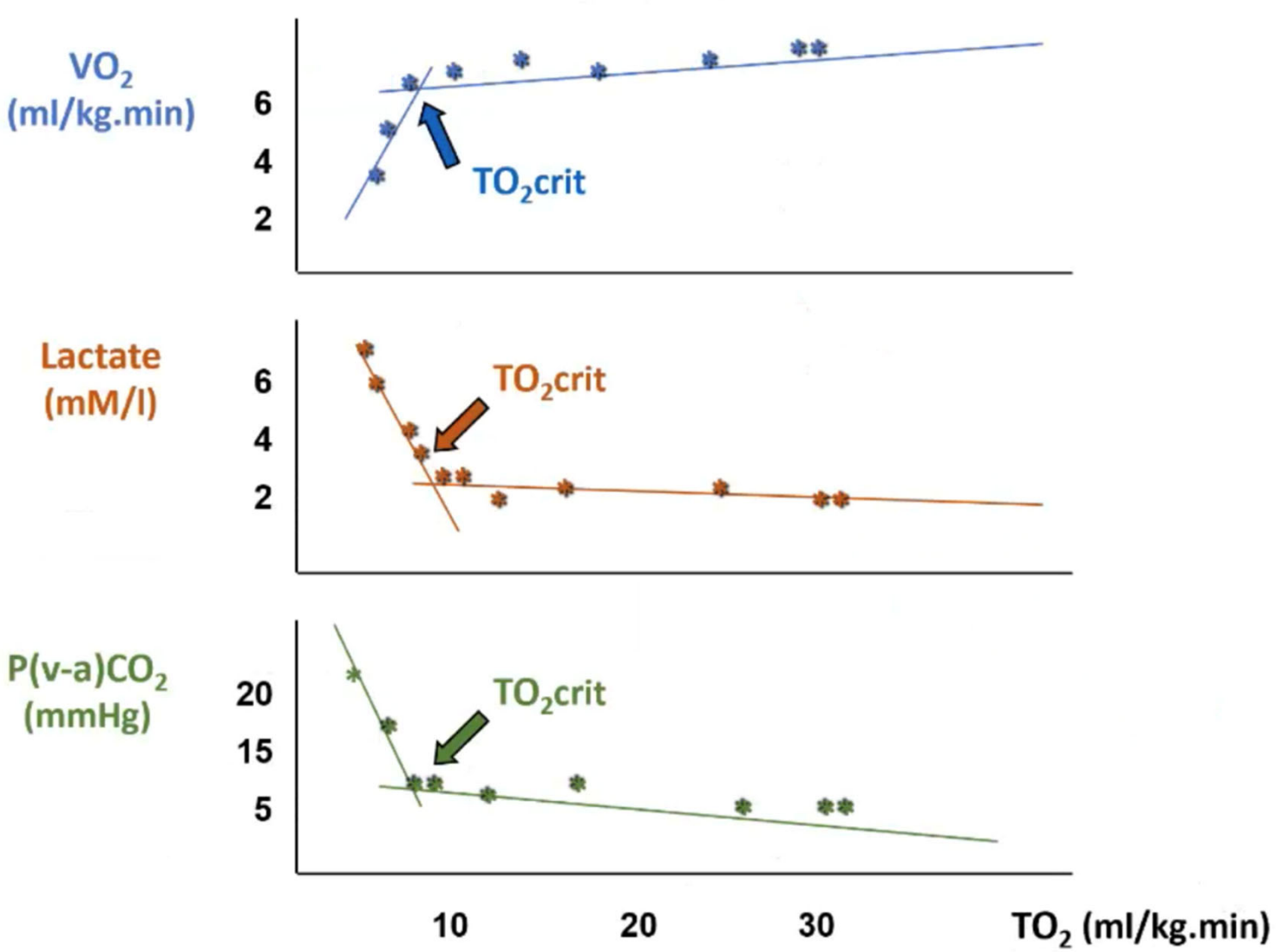
V

INFLUENCE DE LA P50 SUR OXYGÉNATION TISSULAIRE




Arterial blood
Venous blood
Cardiac output = 5 l/min
Haemoglobin = 150 g/l
DO_2 = 1000 ml/min
SaO_2 = 100%
CaO_2 = 20%
$S\bar{v}O_2$ = 75%
$C\bar{v}O_2$ = 15%
O_2
Tissues
Tissue oxygen consumption = 250 ml/min

Adaptation TaO_2/VO_2



$ERO_2 = 1 - SvO2$

Déterminants SvO_2

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

Or :

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

$$CvO_2 = (1{,}39 \cdot Hb \cdot SvO_2) + \underbrace{(0{,}0031 \cdot PvO_2)}_{\text{négligeable}}$$

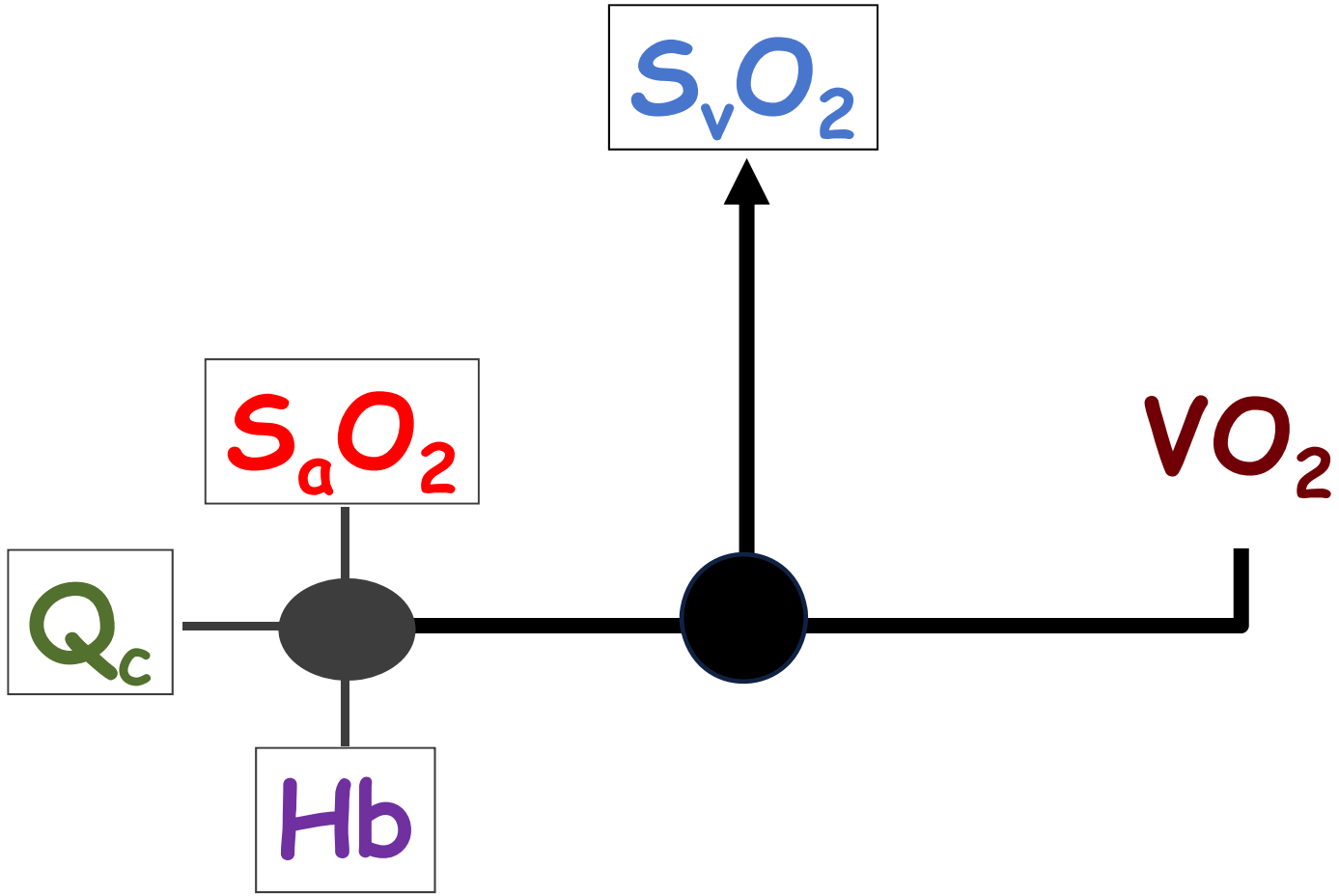
D'où :

$$VO_2 \simeq (SaO_2 - SvO_2) \cdot (Hb \cdot 1{,}39 \cdot DC)$$

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

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

S_vO_2 : FACTEURS DÉTERMINANTS



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

Check for updates

2018

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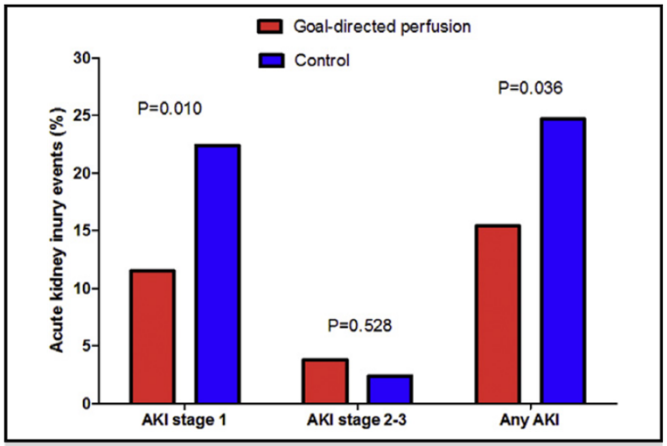
ABSTRACT

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 2-3 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 m^2	1-4 m^2
Surface/volume	300 cm^{-1}	28 cm^{-1}
Distance de diffusion	1-2 µm	10-30 µm
Échange gazeux pour O_2	210-3 200 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

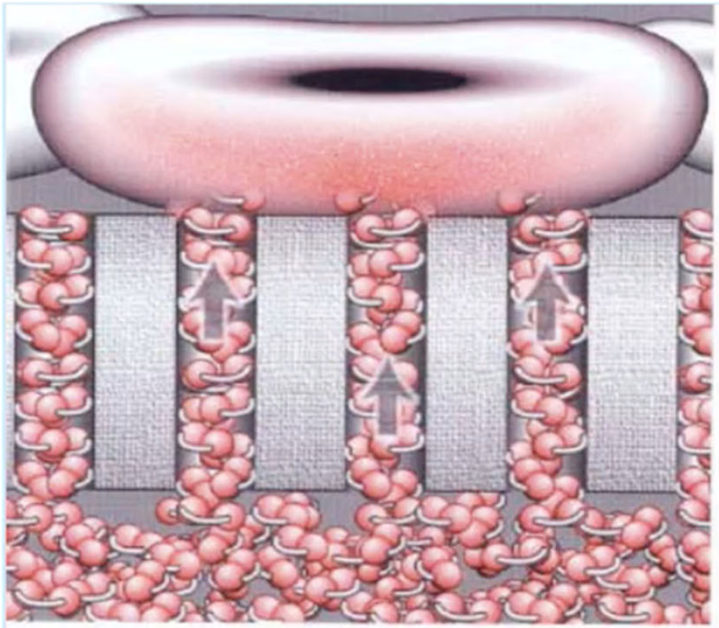


Figure 1: Typical hollow fiber design with a direct gas to blood interface through an open pore.

CEC

Fuite plasmatique et degradation des échanges si > 6 heures

POLYMETHYLPENTHENE

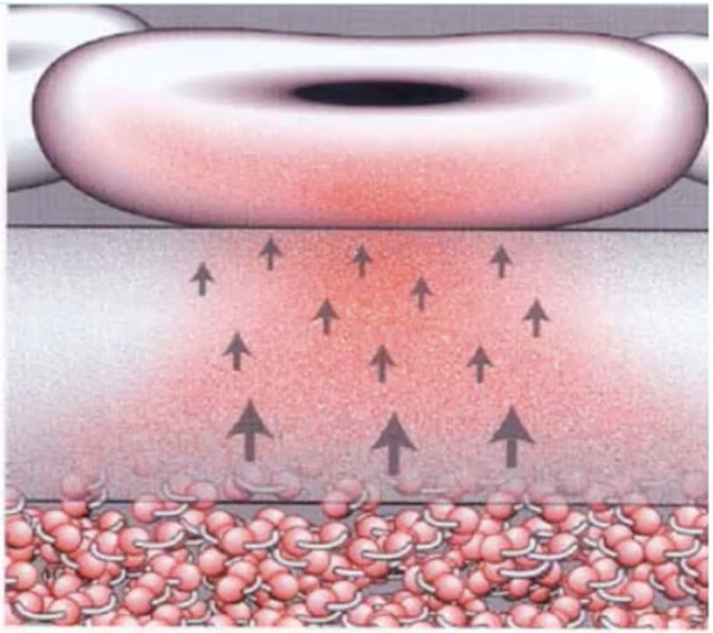


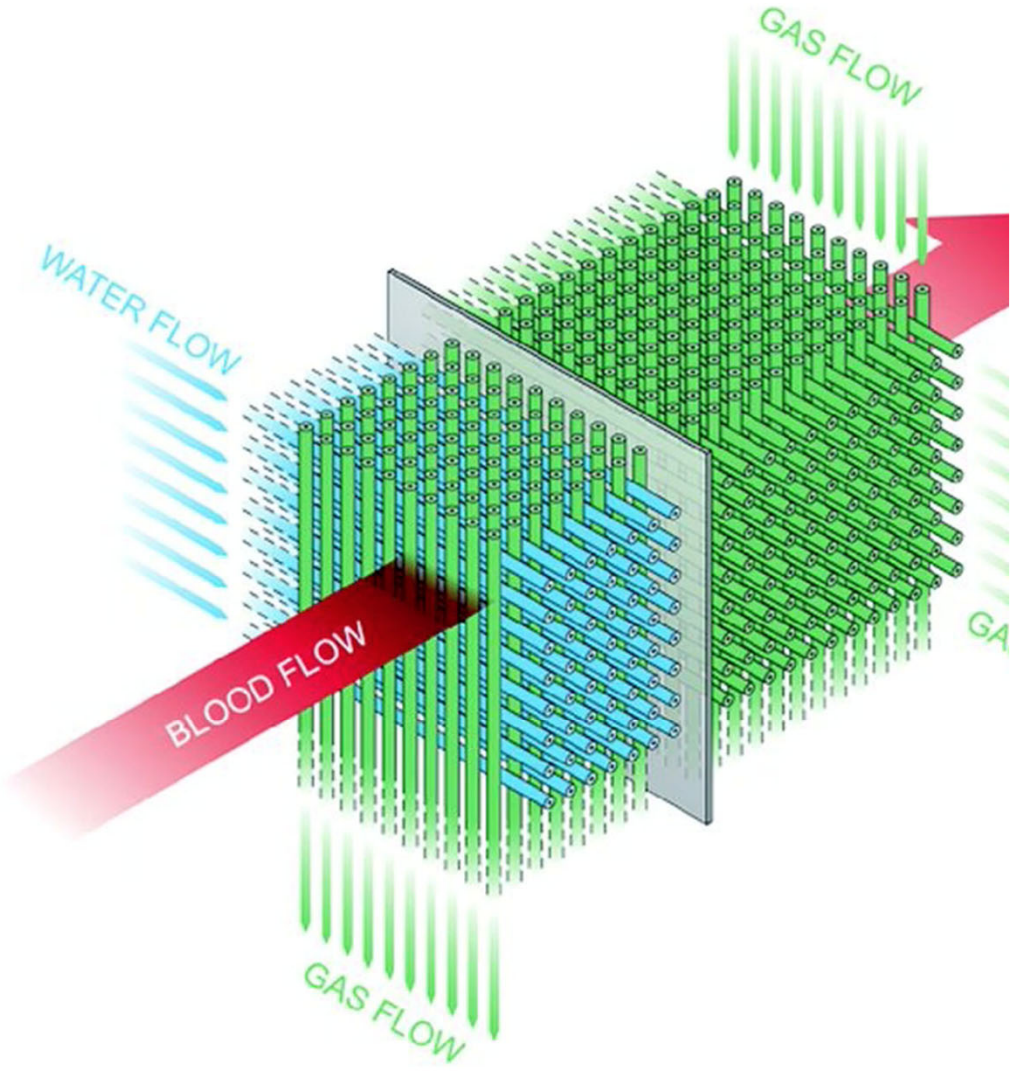
Figure 2: Gas exchange by diffusion through a semi-permeable membrane.

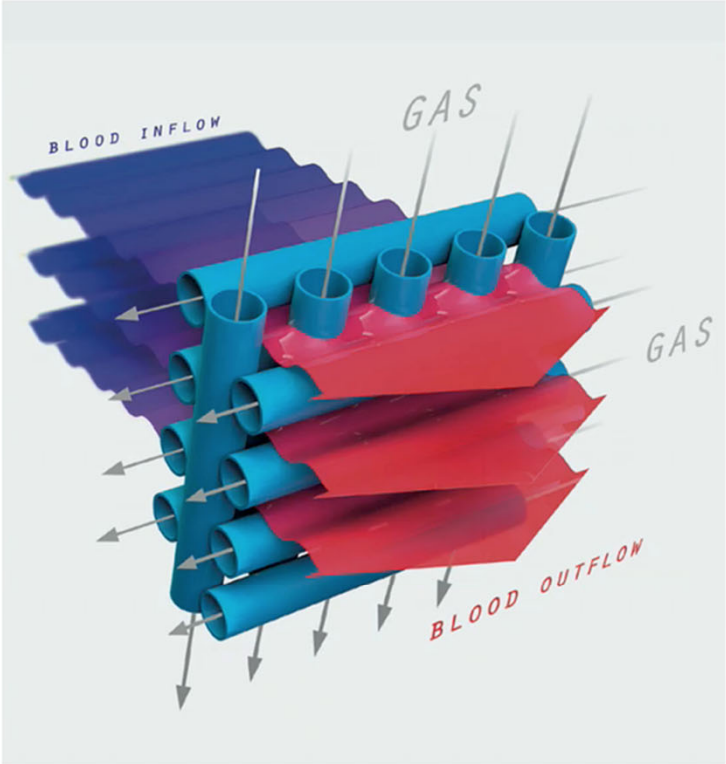
ECMO++++

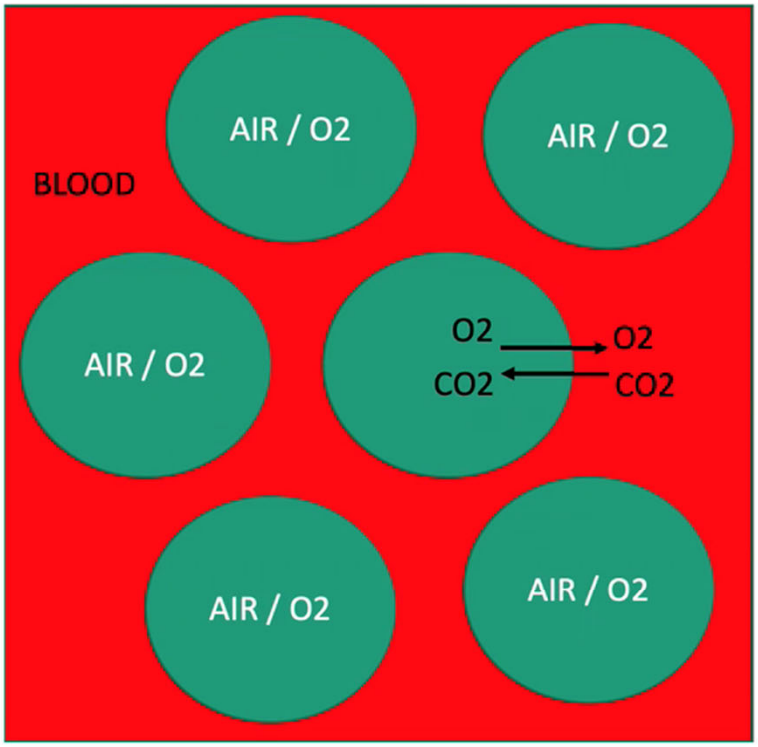
Hémocompatible, durée de vie supérieure

Imperméable aux halogénés


MAQUET
QUADROX-i
ADULT
Hollow Fiber Oxygenator
Microporous Membrane
SOFTLINE COATING


GAS FLOW
WATER FLOW
BLOOD FLOW
GAS FLOW


BLOOD INFLOW
GAS
GAS
BLOOD OUTFLOW


BLOOD
AIR / O2
AIR / O2
AIR / O2
O2
O2
CO2
CO2
AIR / O2
AIR / O2

Oxygénation

•Peu dépendante du débit de gaz.

•Dépendante :

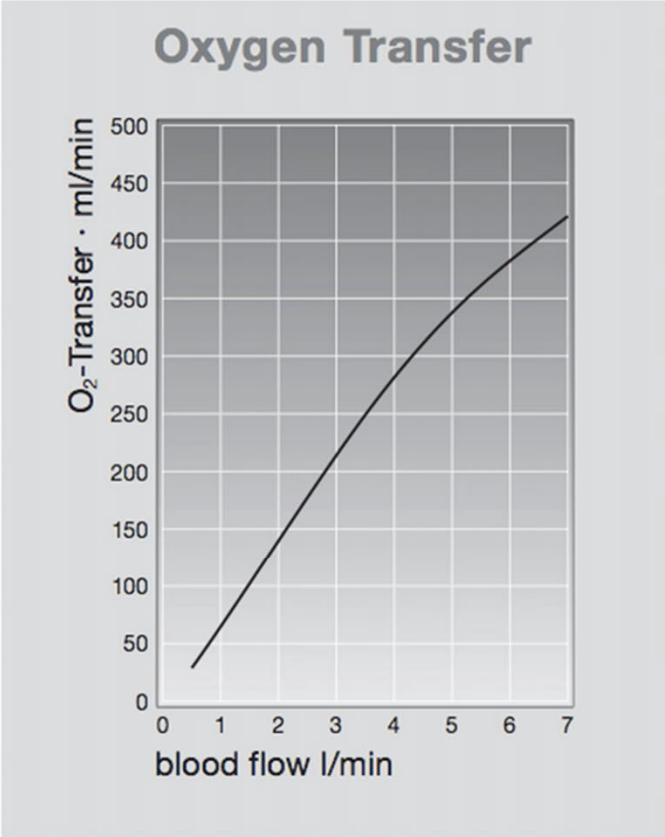
- de la **FdO2** du mélange gazeux.

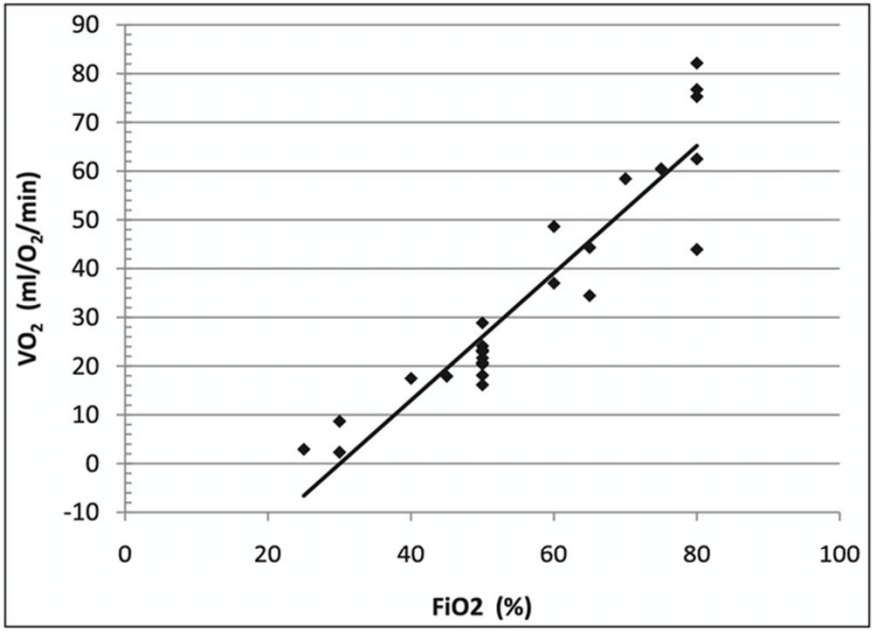
- du DEBIT DE SANG (notion de débit sanguin corrigé ou rated flow : débit max de sang 75% - 95%).

- 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

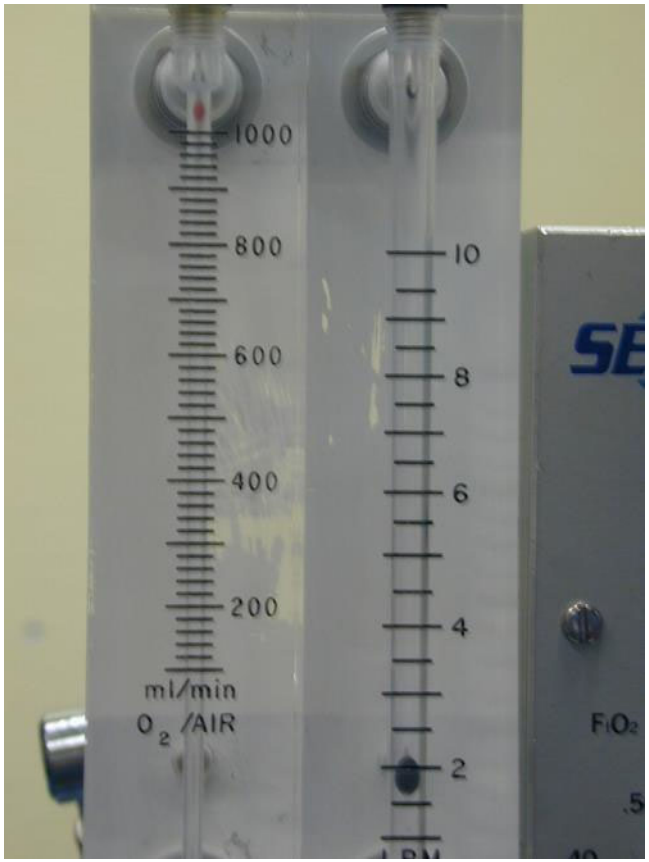
Décarboxylation

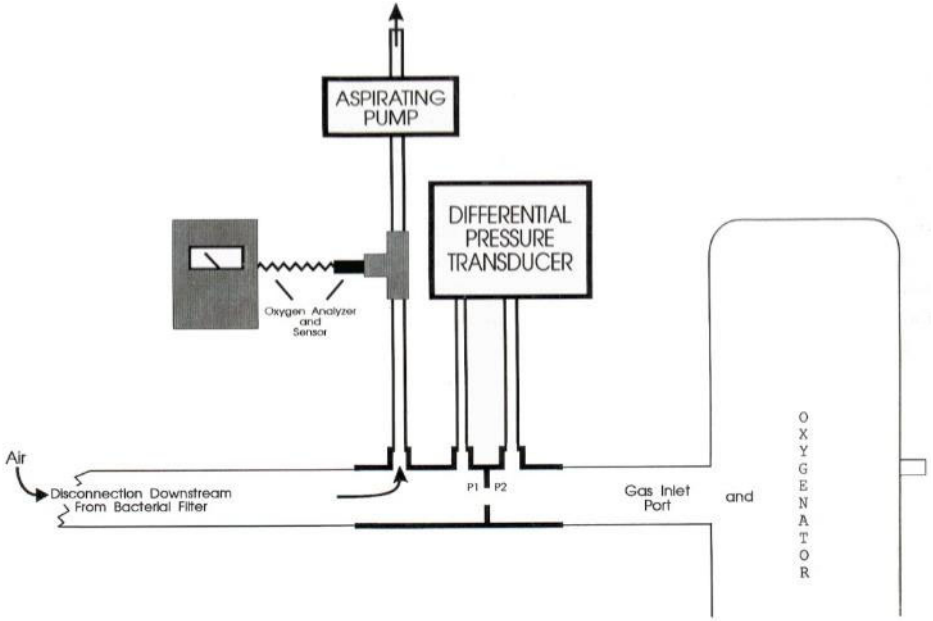
Débit de sang (non limitant, 25% du débit physiologique suffit).

•Dépendante :

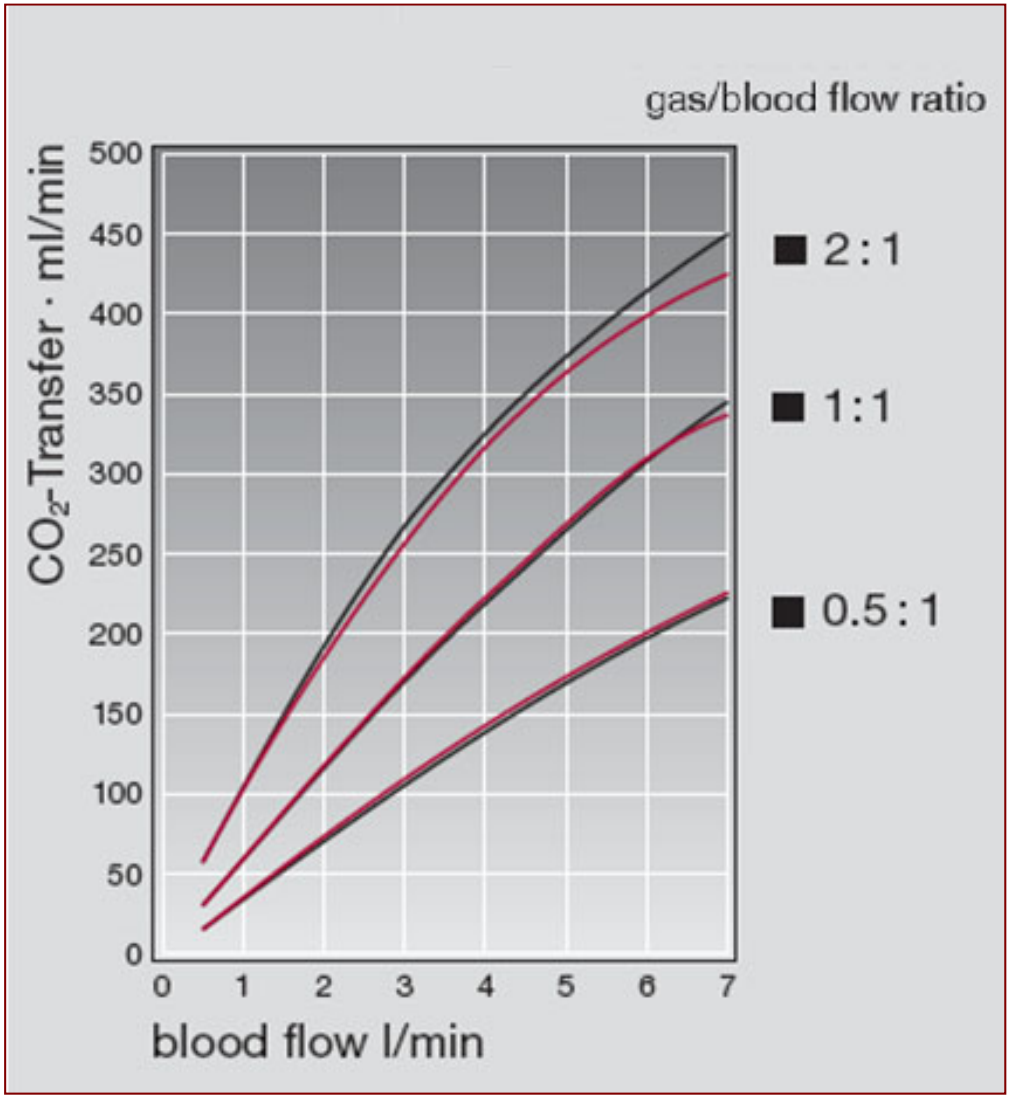
- du DEBIT DE GAZ (BALAYAGE ou sweep gas).
- du gradient de diffusion (PaCO2 d'entrée).
- des caractéristiques de la membrane (surface…).

GAZ FRAIS





Kirson et al. J Cardiothorac Vasc Anesth, 1994

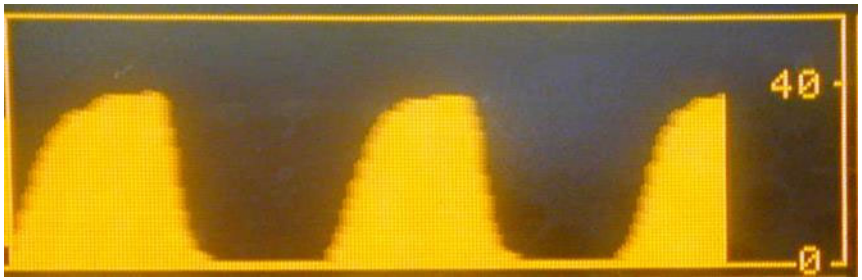

gas/blood flow ratio
CO_2-Transfer · ml/min
500
450
400
350
300
250
200
150
100
50
0
0
1
2
3
4
5
6
7
blood flow l/min
2 : 1
1 : 1
0.5 : 1

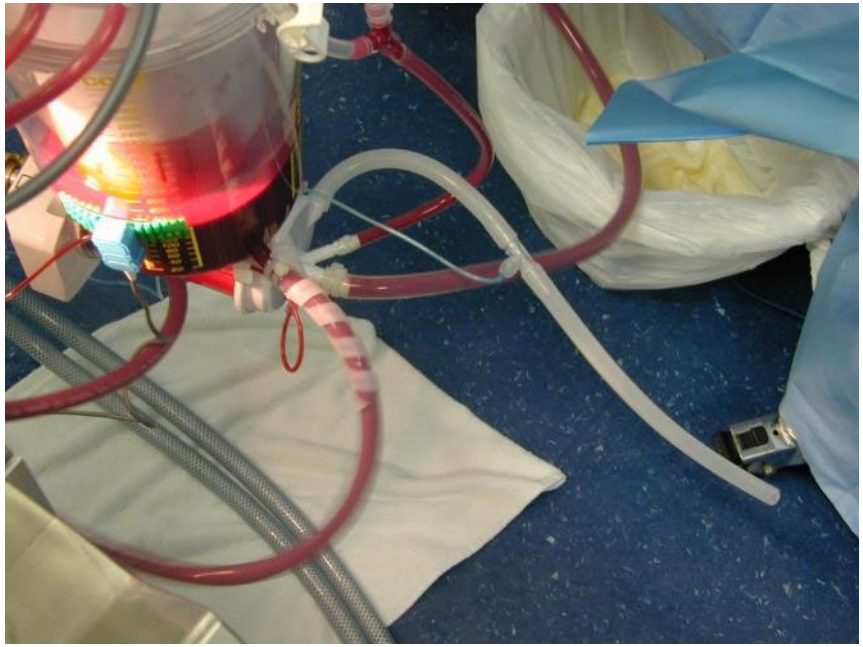
MONITORAGE GAZ DU SANG

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

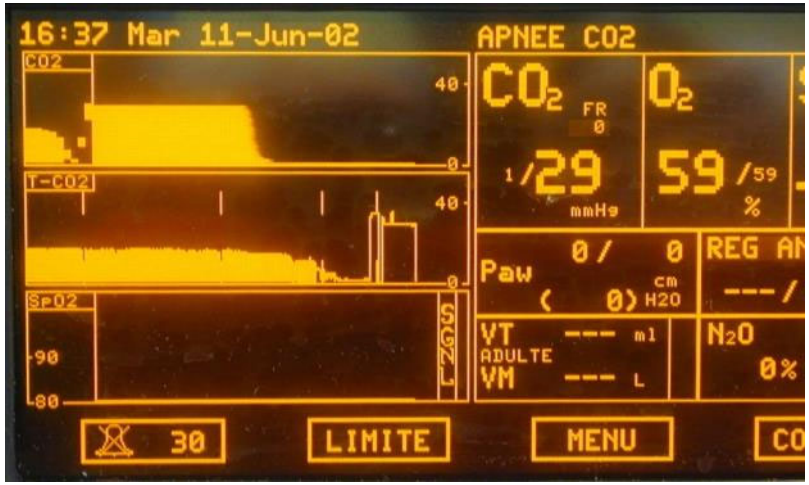
3) Mesure répétées des GDS

MONITORAGE GAZ (ÉVENT MEMBRANE)





Estimation VCO_2



Quel niveau d'oxygénation en CEC ?

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Hôpital Haut-Lévêque hospital
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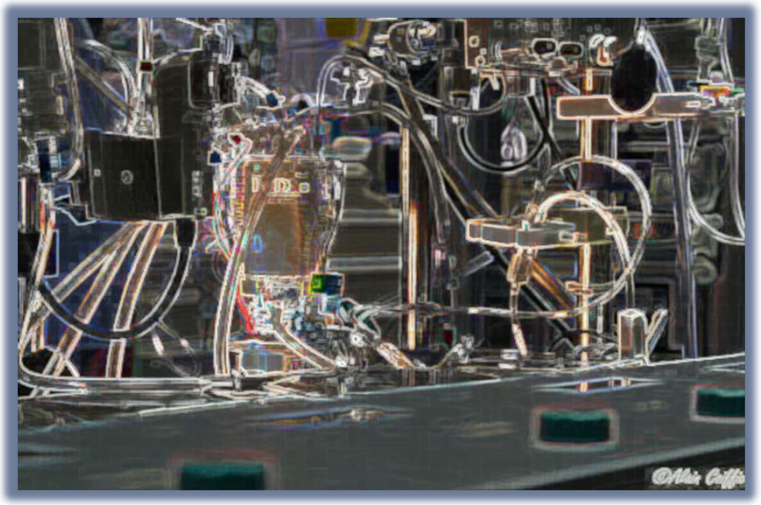


CHU Hôpitaux de Bordeaux

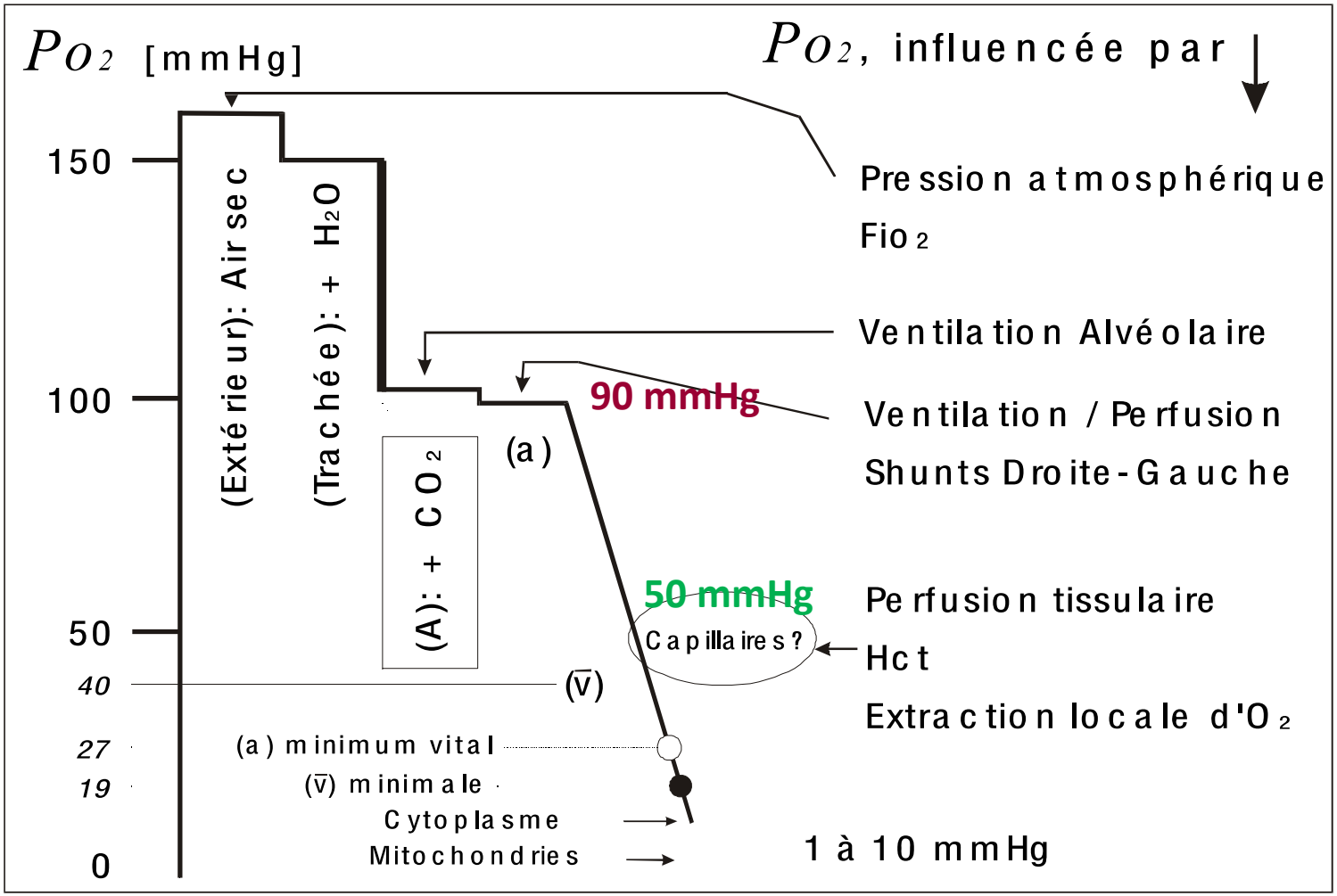
université de BORDEAUX

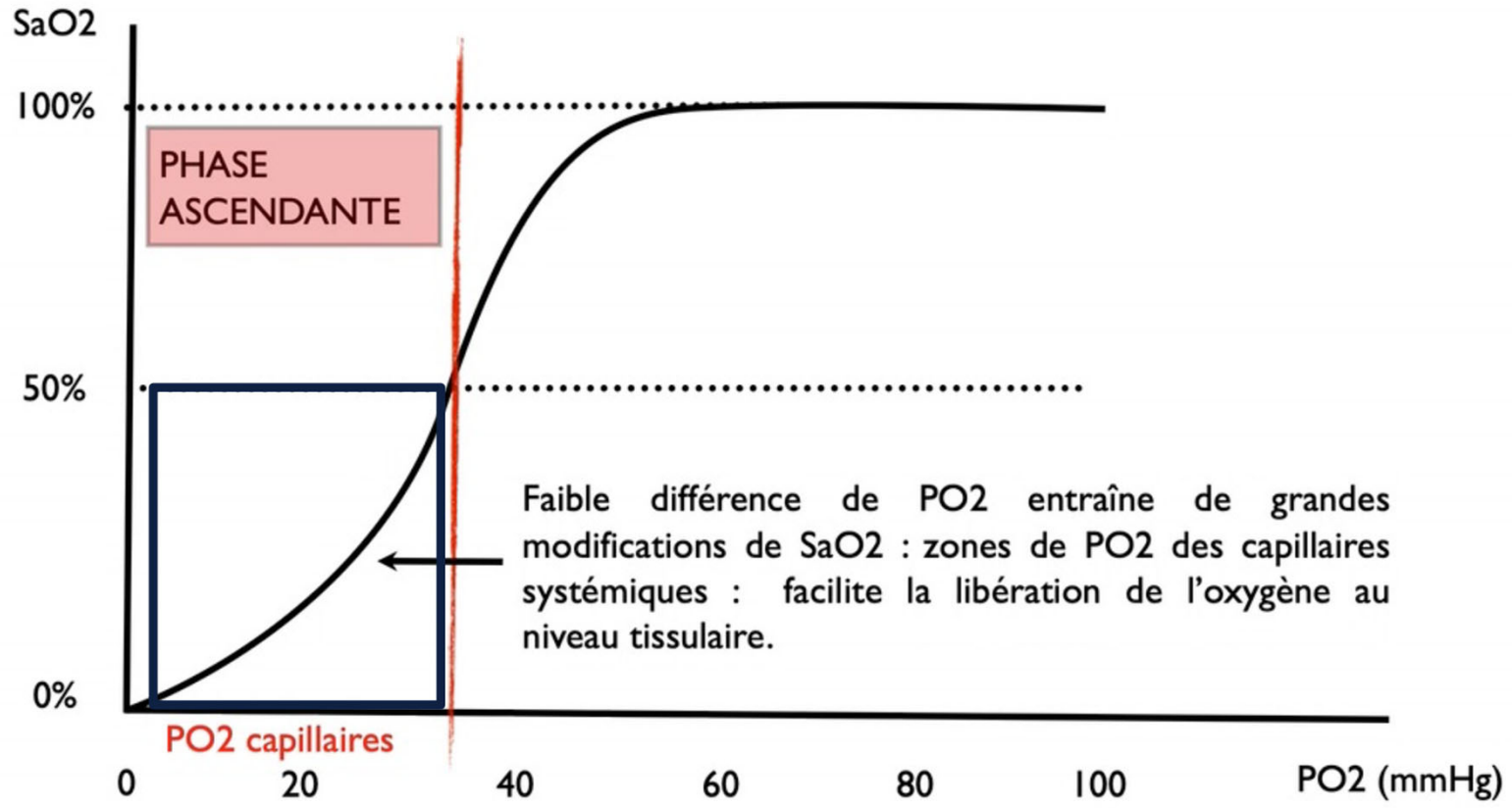
Inserm U1034

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




SaO2
100%
50%
0%
PHASE ASCENDANTE
Faible différence de PO2 entraîne de grandes modifications de SaO2 : zones de PO2 des capillaires systémiques : facilite la libération de l'oxygène au niveau tissulaire.
PO2 capillaires
0
20
40
60
80
100
PO2 (mmHg)

CONTENU ARTÉRIEL EN OXYGÈNE (CONCENTRATION EN OXYGÈNE)

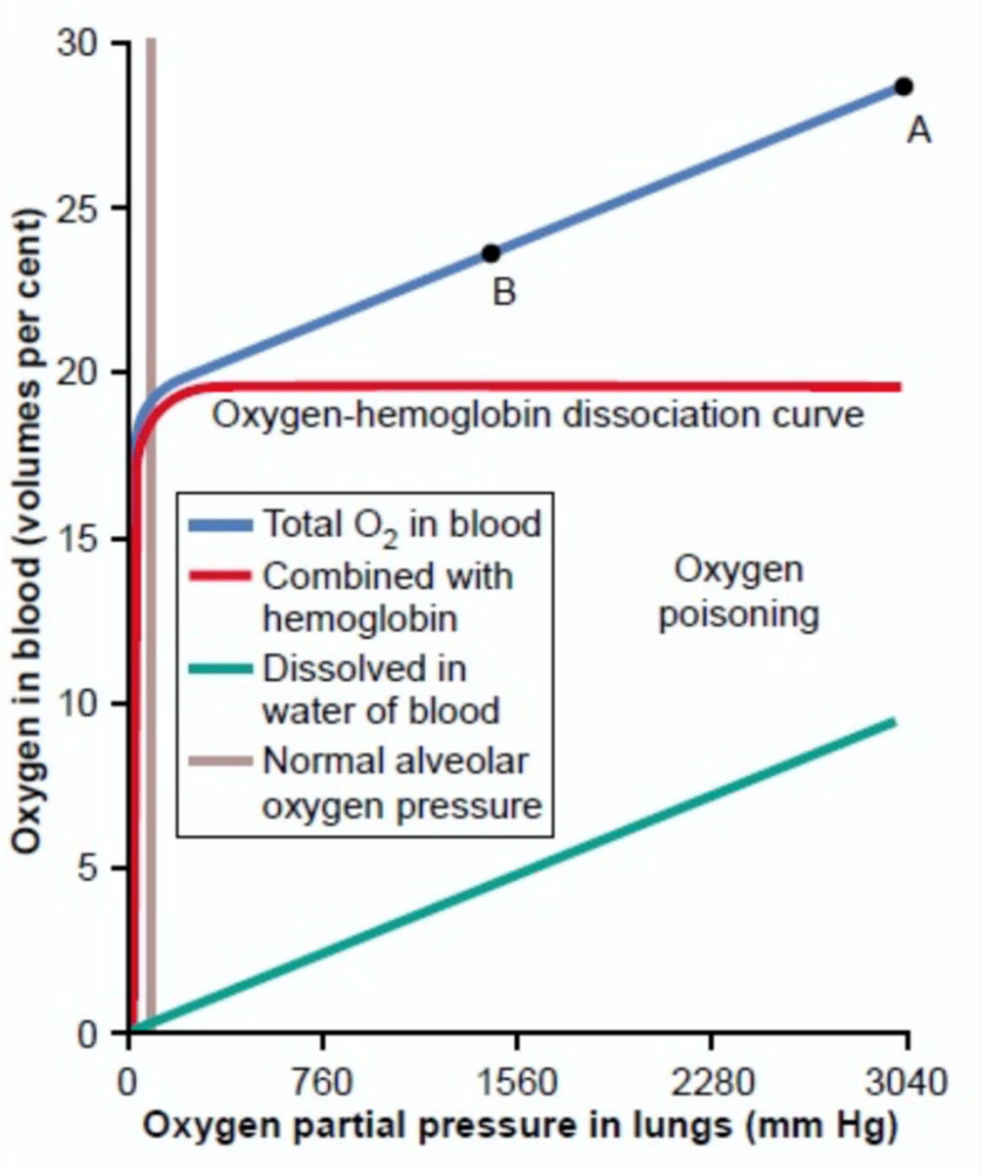
CaO_2 = O_2 transporté par Hb + O_2 dissous

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

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)

CaO_2= 18-20 ml d'O_2/100 ml de sang


30
25
20
15
10
5
0
Oxygen in blood (volumes per cent)
A
B
Oxygen-hemoglobin dissociation curve
Total O_2 in blood
Combined with hemoglobin
Dissolved in water of blood
Normal alveolar oxygen pressure
Oxygen poisoning
0
760
1560
2280
3040
Oxygen partial pressure in lungs (mm Hg)

TRANSPORT EN OXYGÈNE (OXYGEN DELIVERY)

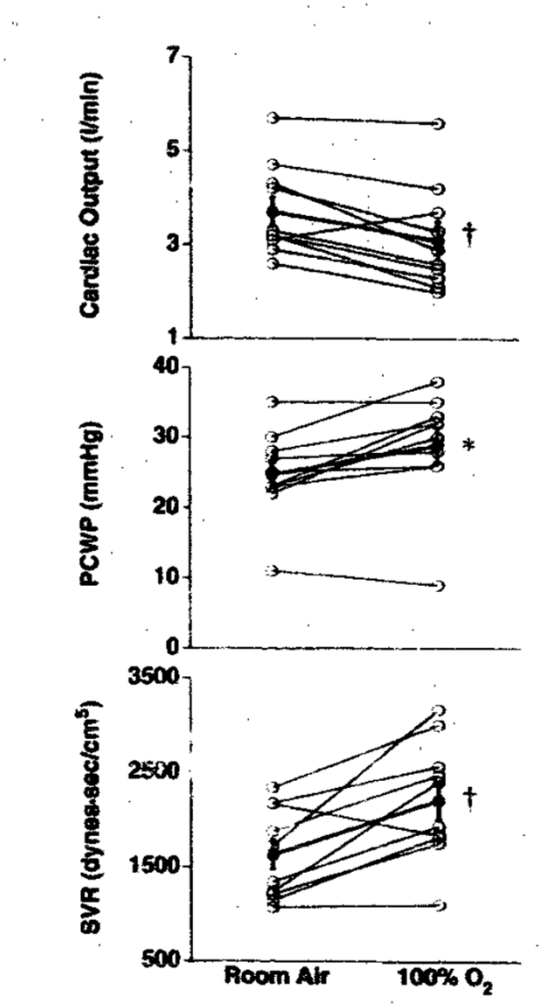
TaO_2 (DO_2)= CaO_2 X Débit cardiaque

↓ ml d'O_2/100 ml de sang

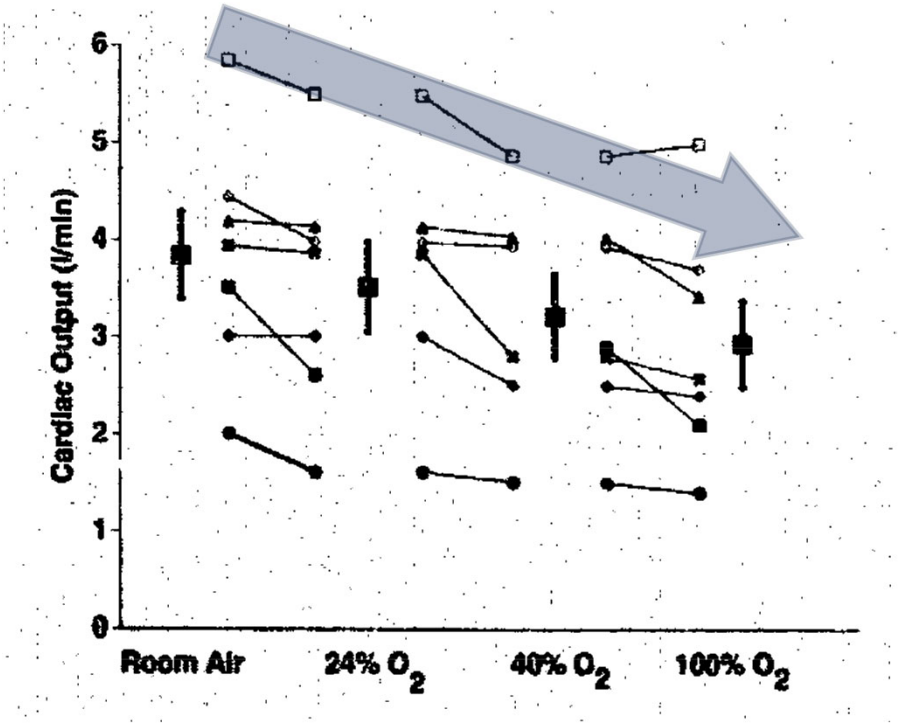
↓ $L.min^{-1}$

Valeur normale 600-800 ml d'O_2. 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

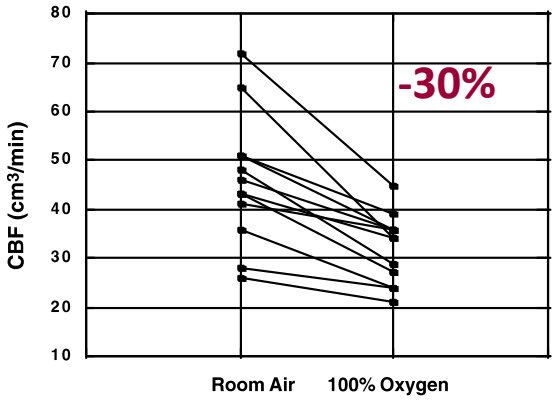
McNulti PH et al. Am J Physiol Heart Circ Physiol 2005

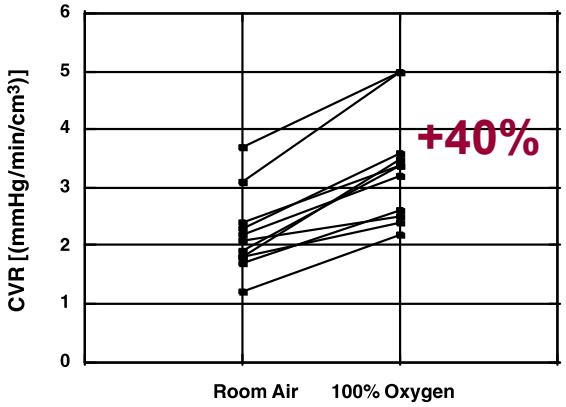
HYPEROXIE ET CIRCULATION CORONAIRE

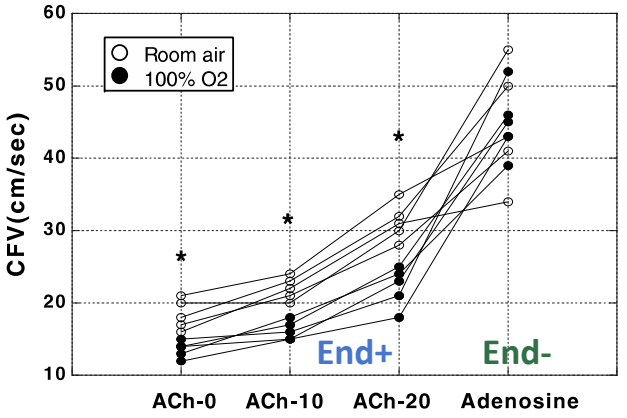
Patients (n=27) bénéficiant d'un cathétérisme cardiaque

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*







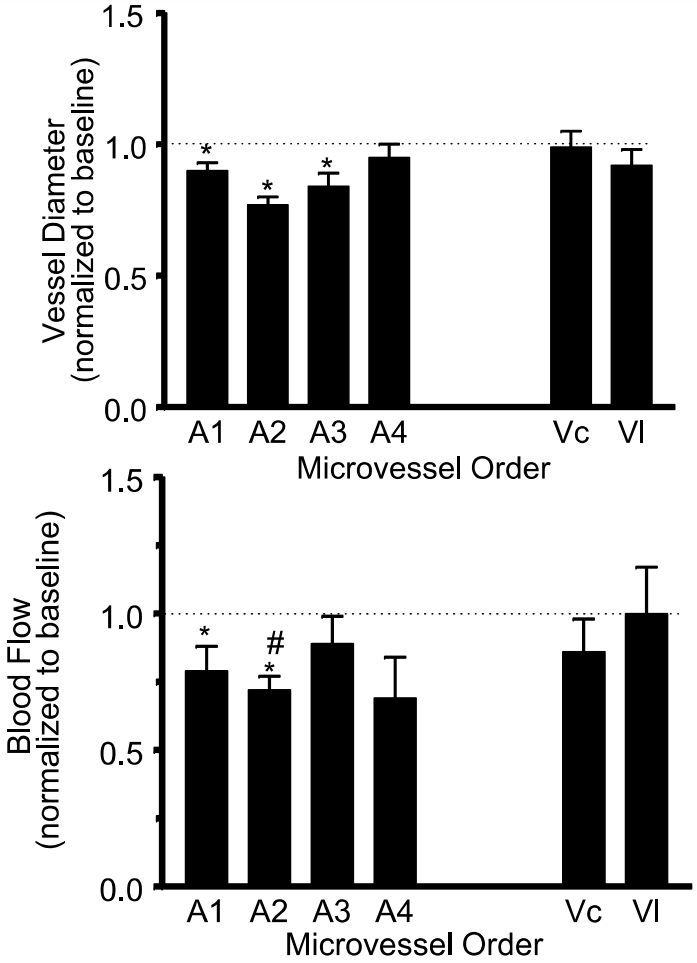
HYPEROXEMIE ET MICROCIRCULATION

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 P_{O_2}, mmHg	60.0 ± 1.2	477.9 ± 19.9*
Arterial P_{CO_2}, mmHg	59.8 ± 4.1	64.3 ± 2.1*
Arterial pH	7.36 ± 0.02	7.33 ± 0.01*
Venous P_{O_2}, mmHg	21.7 ± 7.1	26.4 ± 3.3*
Venous P_{CO_2}, mmHg	70.4 ± 2.8	82.9 ± 8.2*
Venous pH	7.33 ± 0.01	7.27 ± 0.03*
Cardiac index, $ml \cdot min^{-1} \cdot kg^{-1}$	196 ± 13	144 ± 31*
(normalized to baseline)	(1.00 ± 0.00)	(0.75 ± 0.07)
Vascular resistance, $mmHg \cdot min \cdot kg \cdot ml^{-1}$ (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.

TaO2

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-

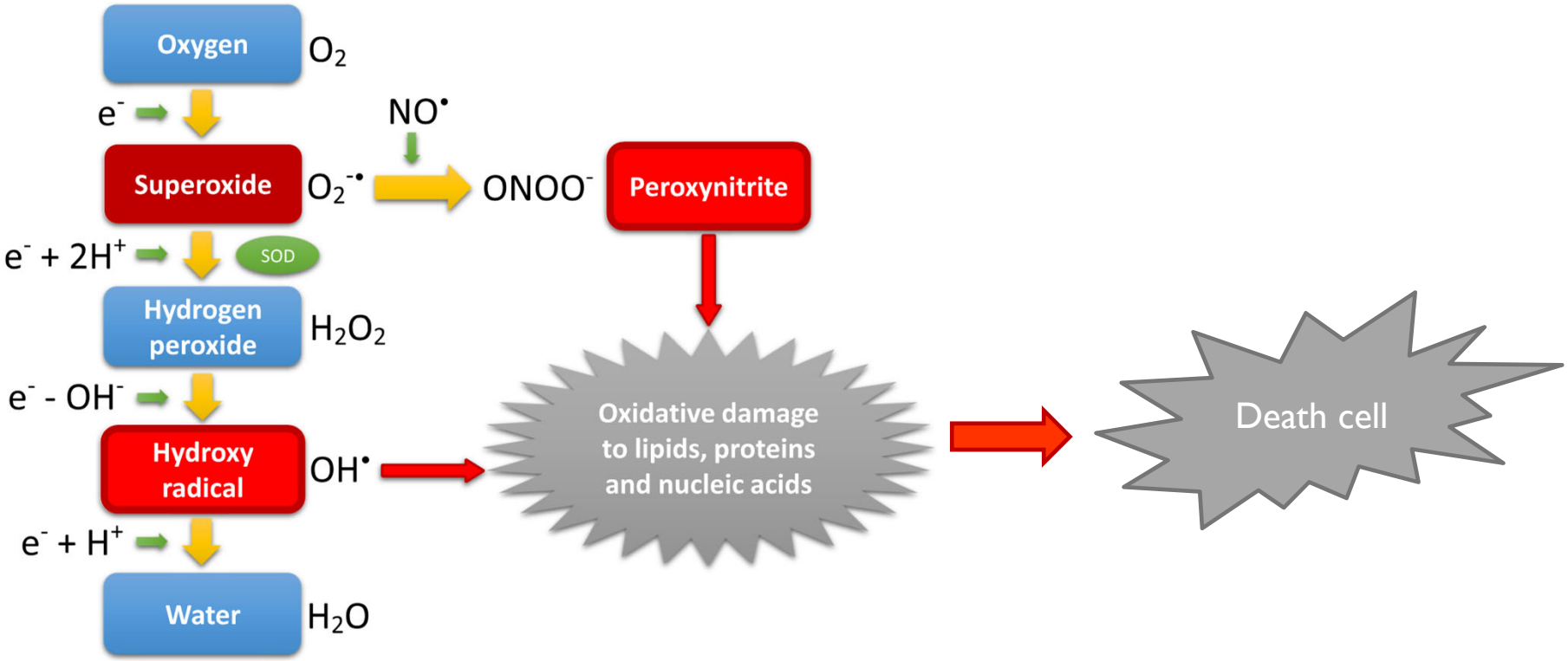
between normoxemic and hyp
at any of the four studied stag
Cardiac index was increased during rewarmin
was otherwise similar at all stages. Pa_{O_2} values
be held within the intended ranges during mea
ments, were significantly different during nor
emic and hyperoxemic conditions ($p < 0.001$)
did not overlap. Venous oxygen saturations
higher during hyperoxemia, reaching statistica
nificance during hypothermic CPB and after C

PO_2 musculaire

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

STRESS OXYDATIF ET CEC

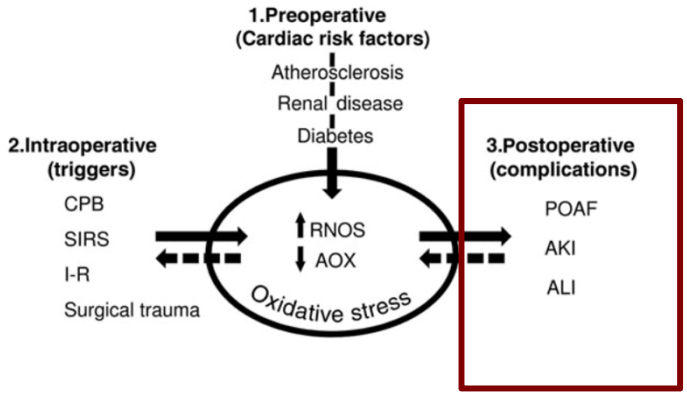
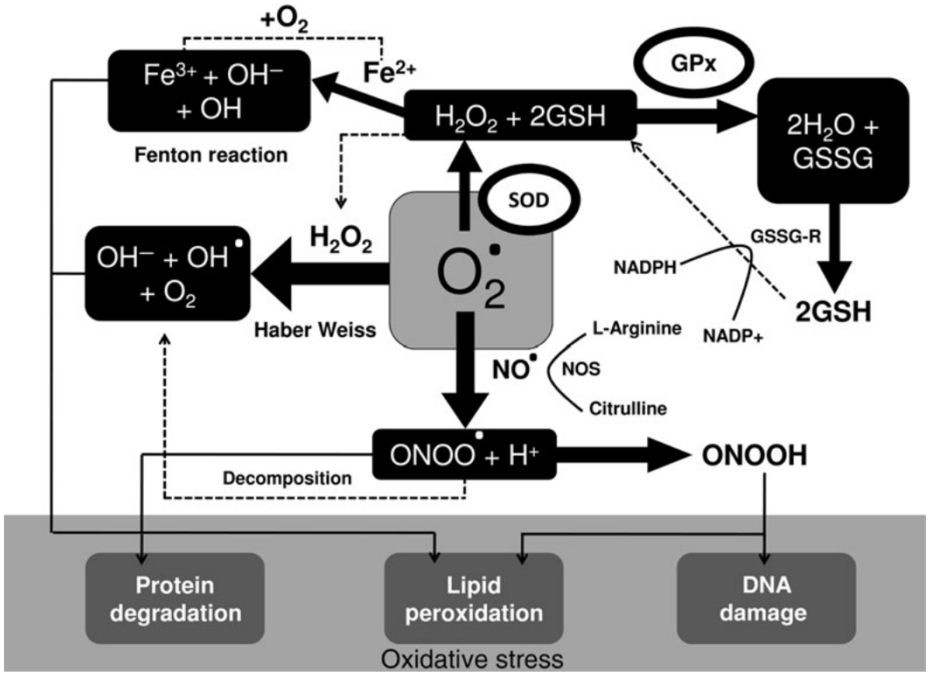


Table 1: Potential triggers for increased RNOS generation during ECMO

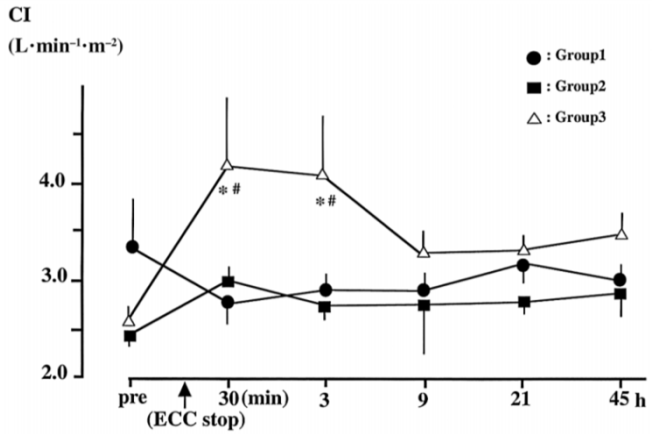
Potential triggers for increased RNOS generation during ECMO
Exposure to ECC (ECMO circuits [42], dialysis [5])
SIRS syndrome [43]
Acute respiratory distress syndrome [16]
Hyperoxia [26]
Mechanical ventilation [44]
Sepsis [45]
Ischaemia-reperfusion [46]
Transfusion [27, 28]
Sequestration of antioxidant trace elements by the ECMO circuit [19]

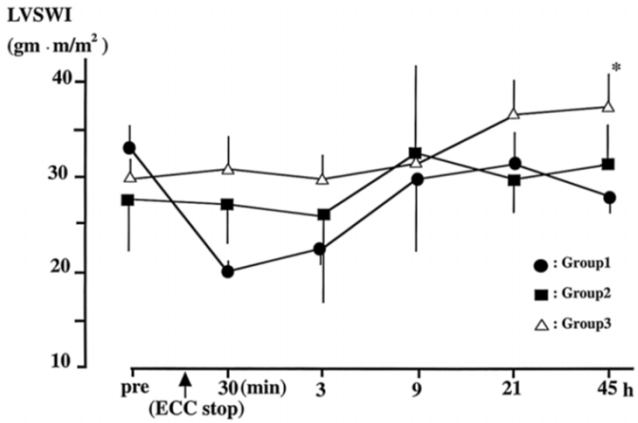


Mc Donald CI et al. Eur J Cardiothorac Surg 2014;46:937-43

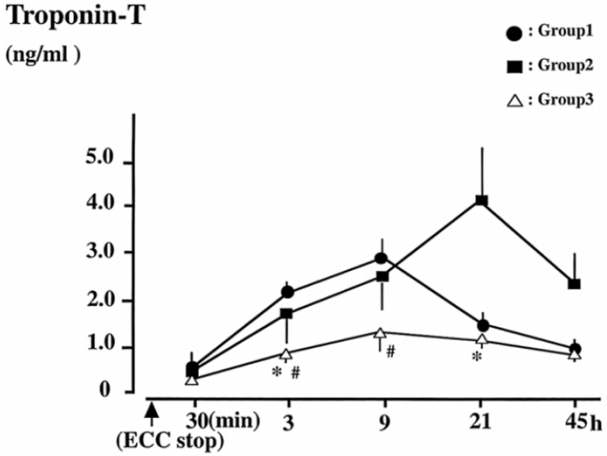
Inoue T et al. Cir J 2022; 66:718-22

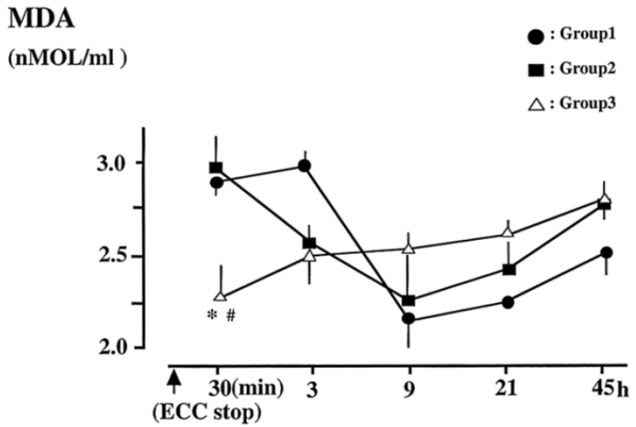
HYPEROXEMIE ET REPERFUSION MYOCARDIQUE





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





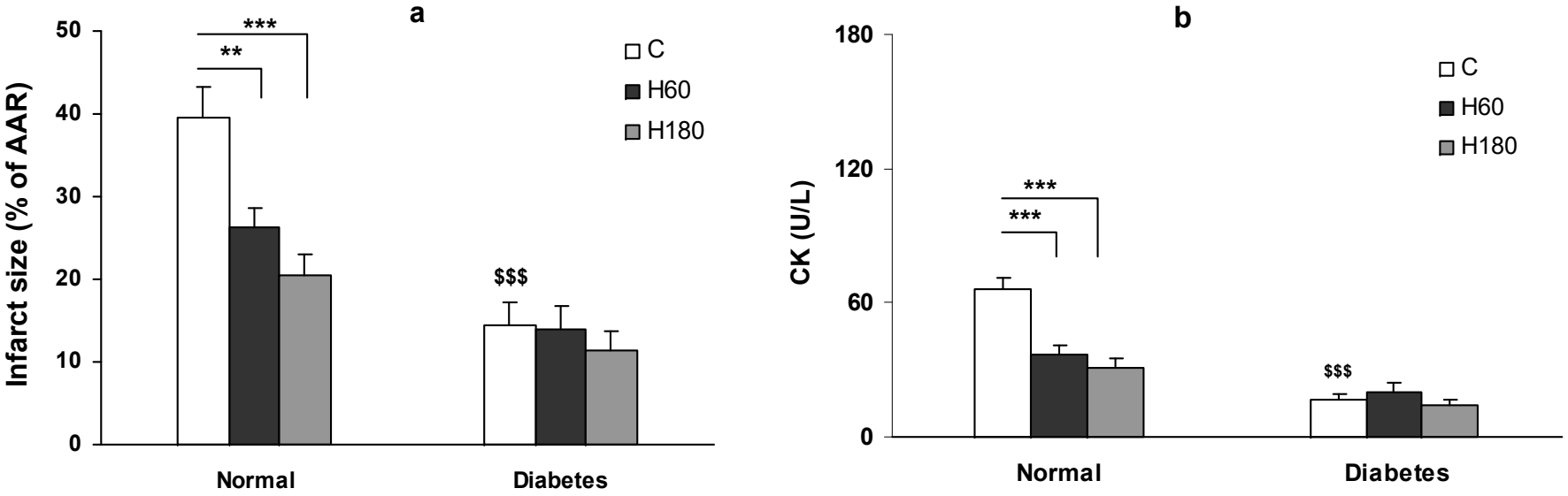
HYPEROXEMIE ET MICRO-BULLES

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

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

HYPEROXIE ET PRECONDITIONEMENT MYOCARDIQUE

Etude expérimentale chez rat soumis à O_2 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 1: 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

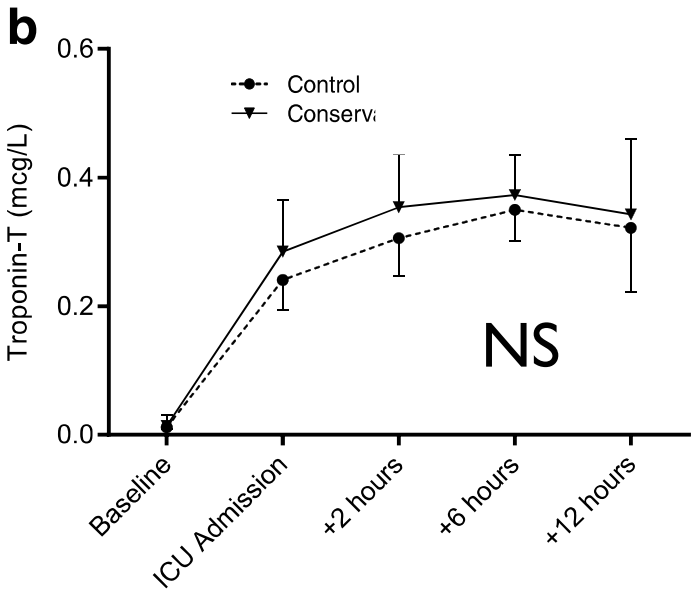
versus

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

CJP= Dommage myocardique (AUC Troponine T)

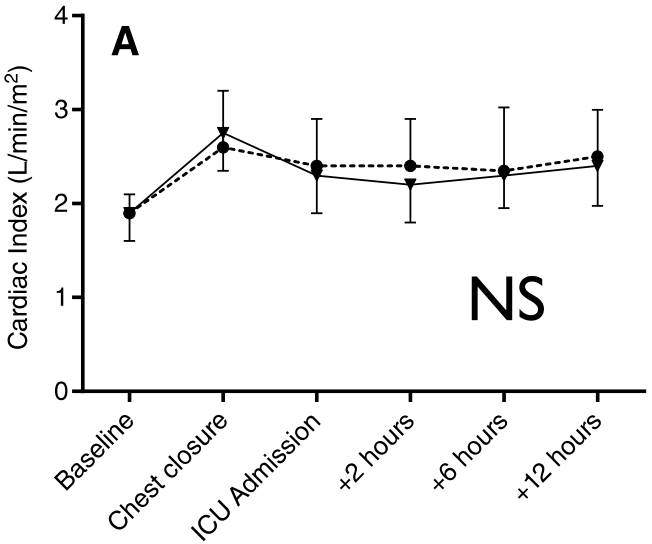
CJS= morbi-mortalité postopératoire

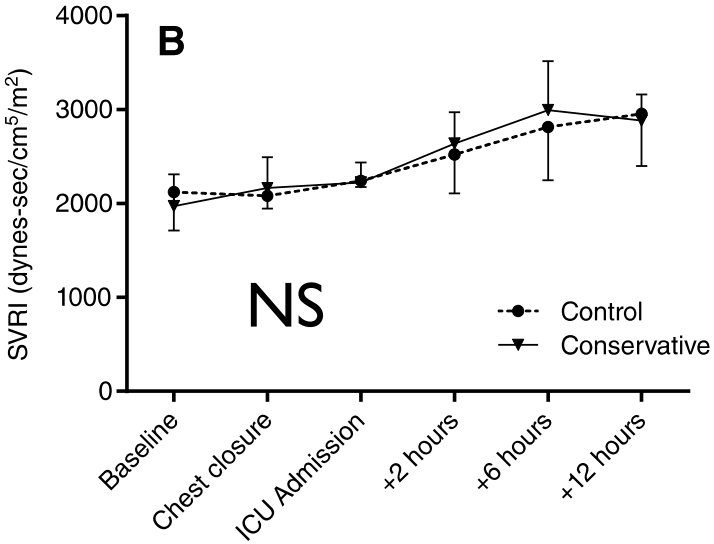
Smit B et al. Crit Care 2016;20:55

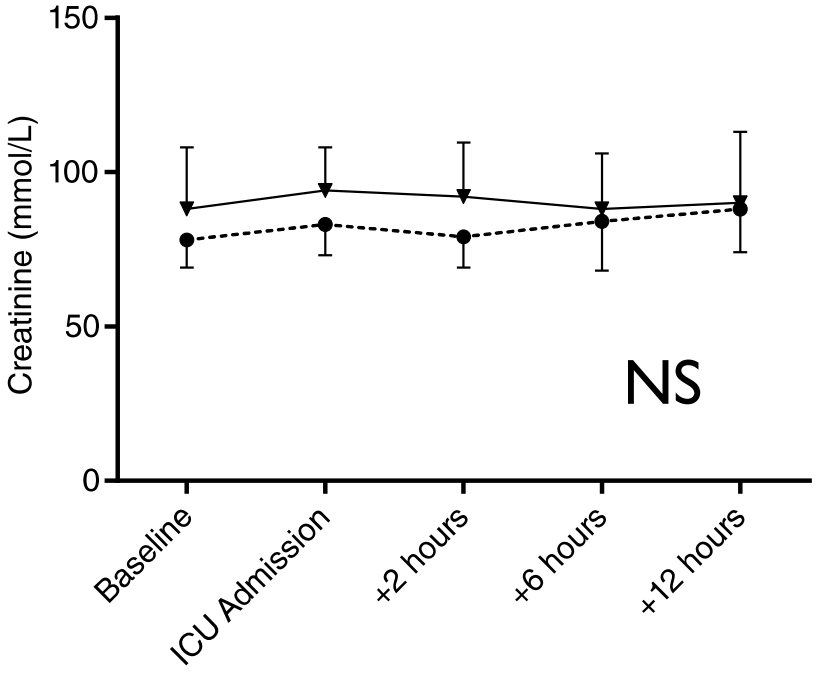


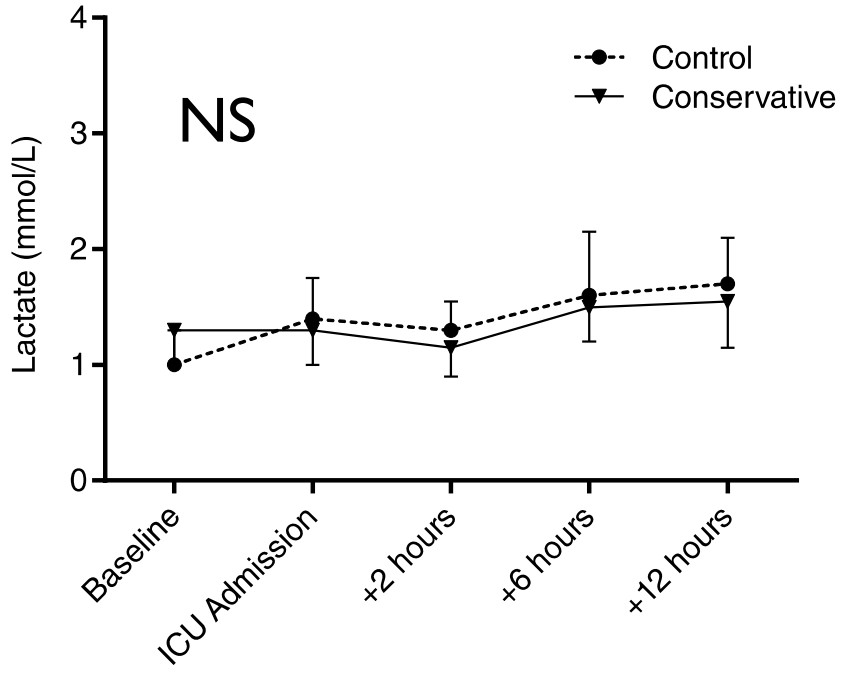
Groupe libéral 220 mmHg (213-233)

Groupe restrictif 157 mmHg (152-161)






150
100
50
0
Creatinine (mmol/L)
NS
Baseline
ICU Admission
+2 hours
+6 hours
+12 hours


4
3
2
1
0
Lactate (mmol/L)
Control
Conservative
NS
Baseline
ICU Admission
+2 hours
+6 hours
+12 hours

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)

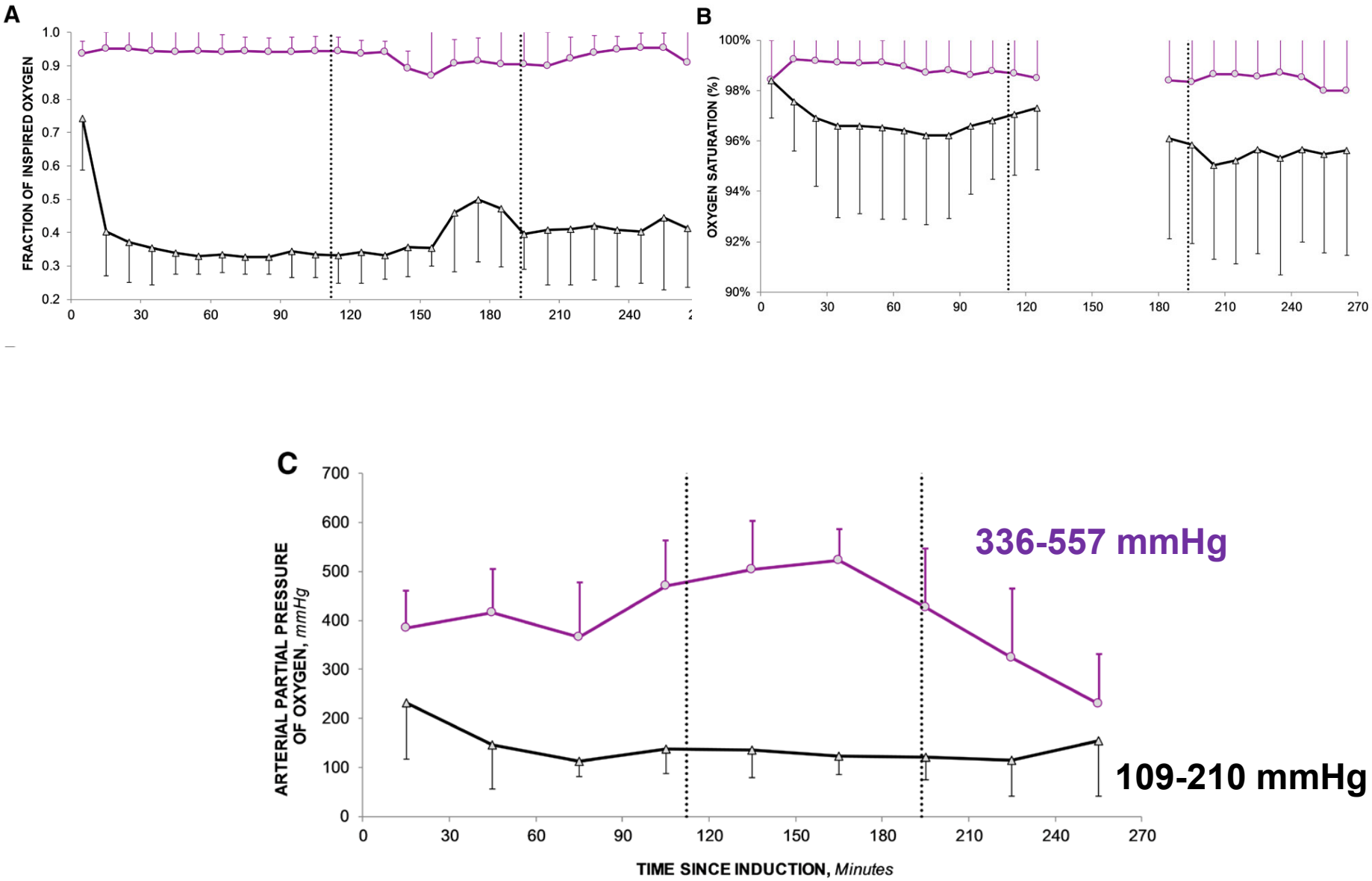
versus

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

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


A
FRACTION OF INSPIRED OXYGEN
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0
30
60
90
120
150
180
210
240
B
OXYGEN SATURATION (%)
100%
98%
96%
94%
92%
90%
0
30
60
90
120
150
180
210
240
270
C
ARTERIAL PARTIAL PRESSURE OF OXYGEN, mmHg
700
600
500
400
300
200
100
0
0
30
60
90
120
150
180
210
240
270
TIME SINCE INDUCTION, Minutes
336-557 mmHg
109-210 mmHg

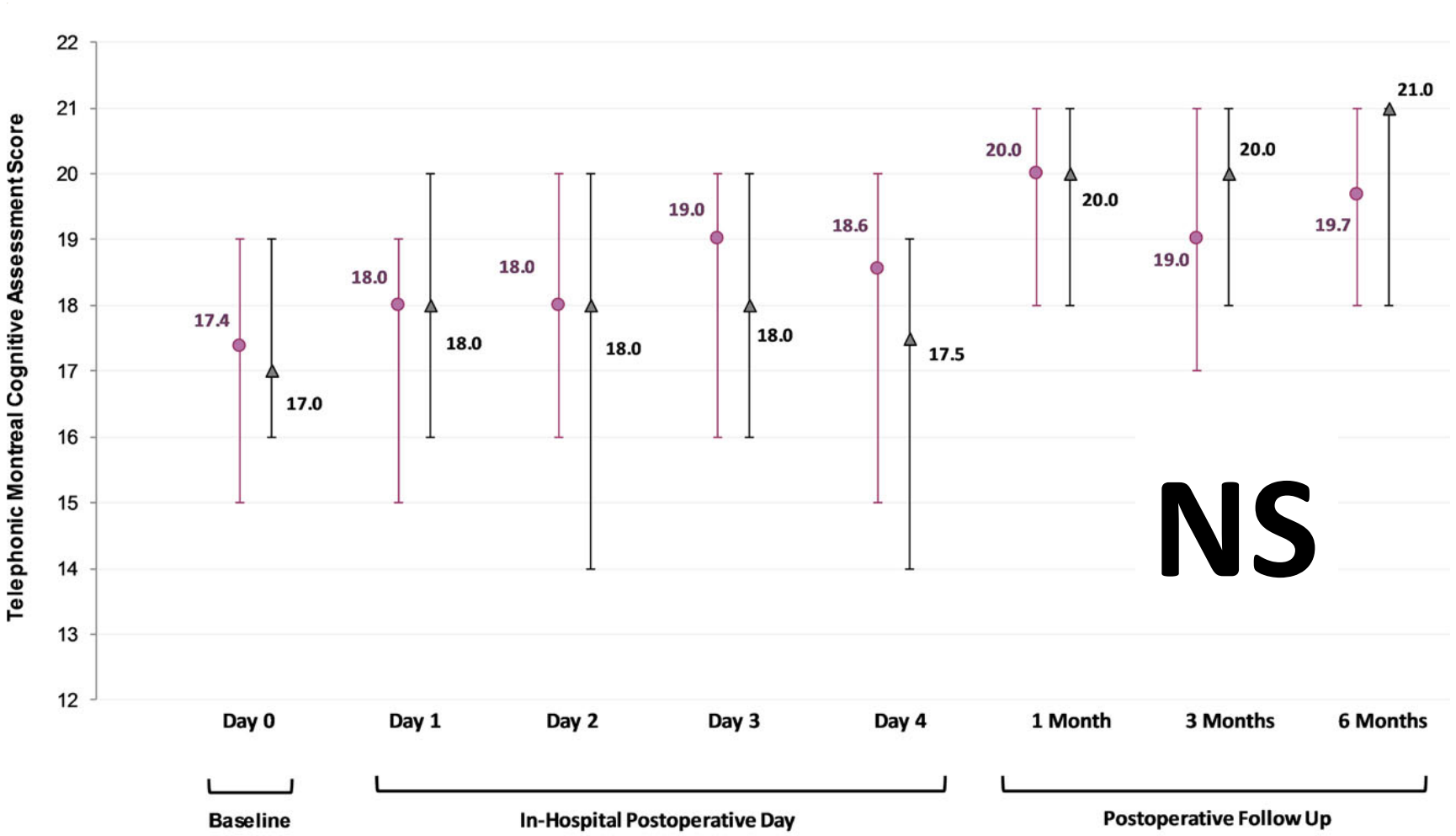

Telephonic Montreal Cognitive Assessment Score
22
21
20
19
18
17
16
15
14
13
12
17.4
17.0
18.0
18.0
18.0
18.0
19.0
18.0
18.6
17.5
20.0
20.0
19.0
20.0
19.7
21.0
NS
Day 0
Day 1
Day 2
Day 3
Day 4
1 Month
3 Months
6 Months
Baseline
In-Hospital Postoperative Day
Postoperative Follow Up

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

Shaefi 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

Tuğba Onur MD ✉, Ümran Karaca MD, Filiz Ata MD, Halil E. Sayan MD, Anıl Onur MD, Canan Yilmaz MD, Ayşe N. Balkaya MD, Cüneyt Eriş MD

PaO_2 entre 100 et 180 mmHg versus PaO_2>180 mmHg

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)

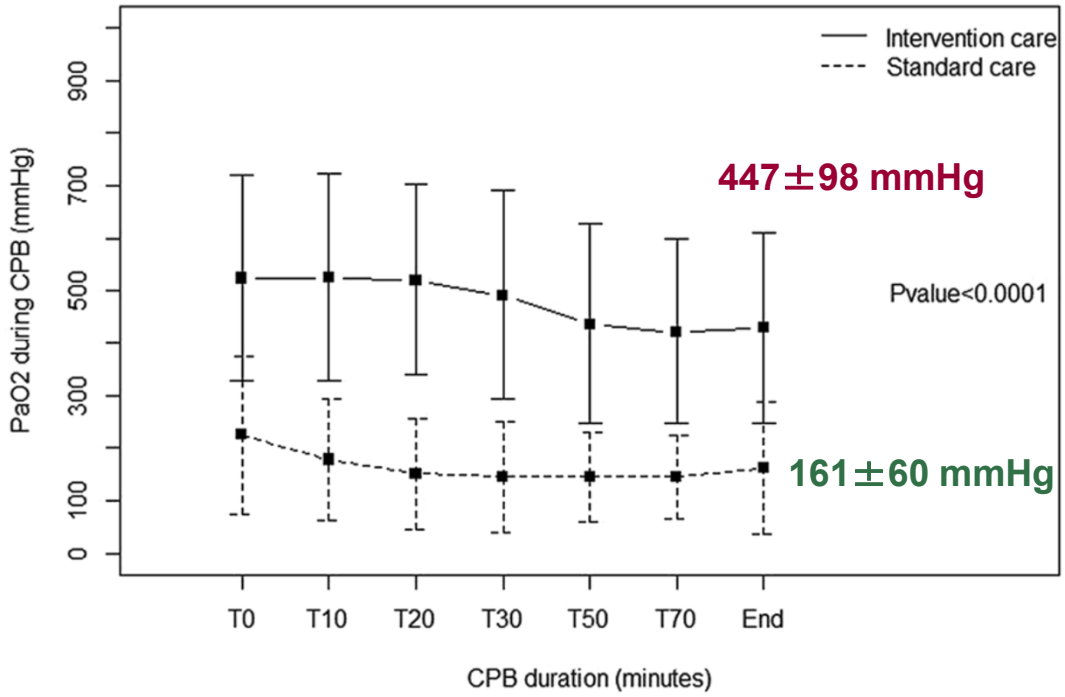
versus

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



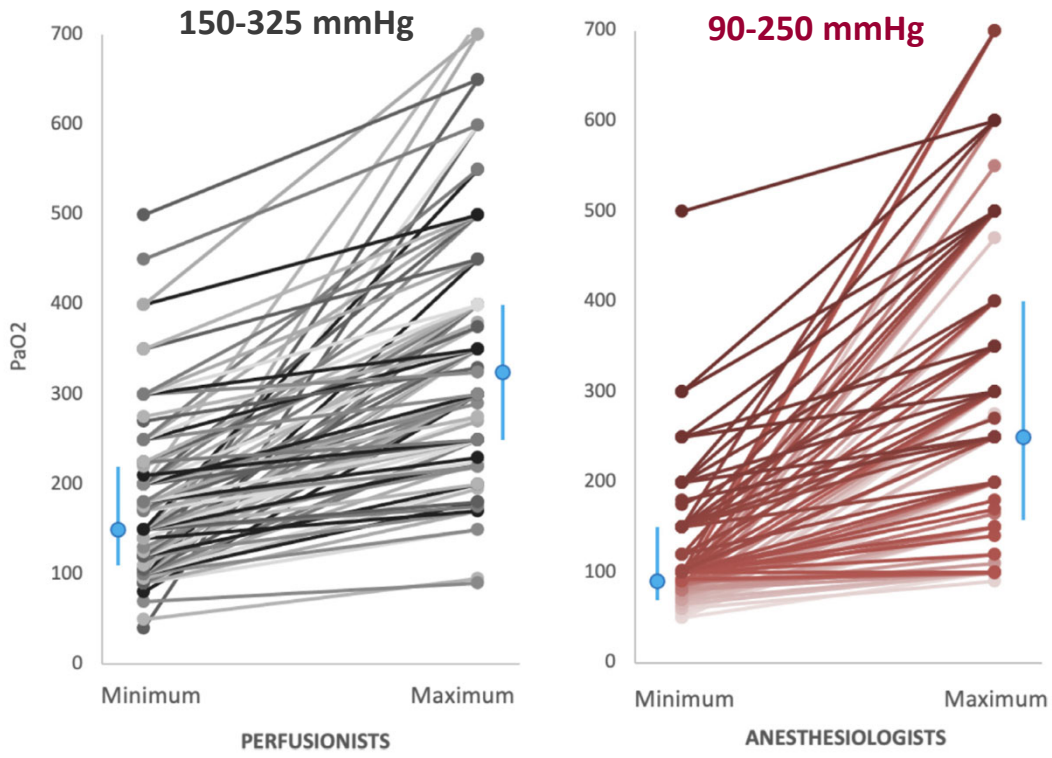
Type of event	Standard group ($n = 163$)	Intervention group ($n = 161$)	Absolute risk difference (intervention-standard)	p value
Primary endpoint				
PAOF or VT/VF (n, %)	49 (30)	49 (30)	0.4% [− 9.6–10.4]	0.94
POAF (n, %)	48 (29)	47 (29)	0.0% [− 9.5–10.2]	0.94
VT/VF (n, %)	4 (2)	4 (2)	0.0% [− 3.3–3.4]	0.99
S d d i				

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

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 00 (2019) 1–42
doi:10.1093/ejcts/ezz267

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

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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 clinque?
- Cible **100-120 mmHg QSP > 96%**
- Meilleure interprétation SvO_2
- Hyperoxémie discutable en pré-CEC (induction anesthésique)