



Physiologie appliquée à la défaillance circulatoire

**29^e Congrès francophone
ACTUALITÉS EN RÉANIMATION**
Médecine Intensive, Surveillance Continue
et Urgences Graves

Alexandra Beurton

Service de Médecine Intensive – Réanimation
Hôpital Tenon, AP-HP, Paris

Je déclare les liens d'intérêt suivants :

Aucun

Physiologie du système circulatoire

Physiopathologie de la défaillance circulatoire

Mécanismes compensateurs

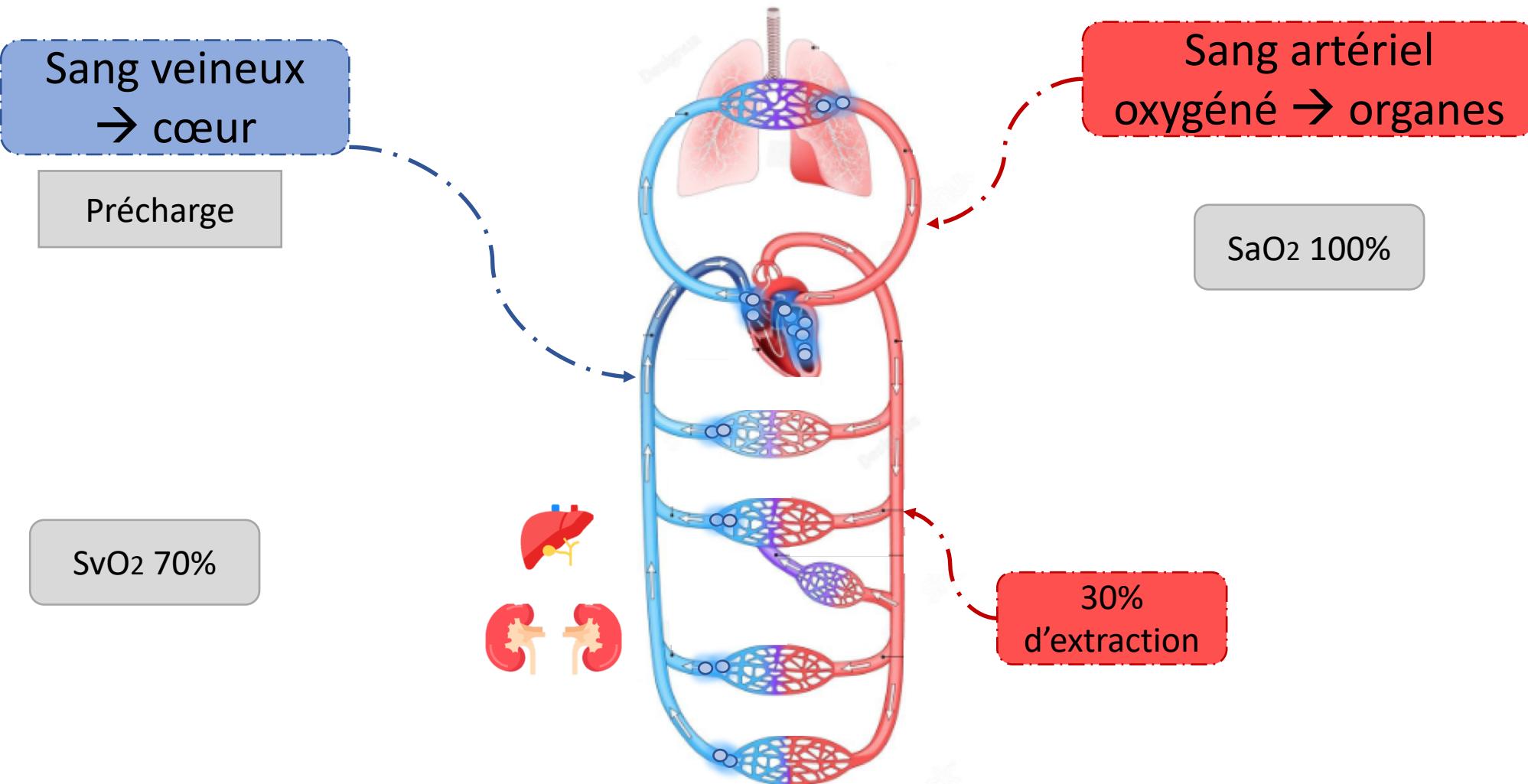
Le cas du choc septique

Impact des états de choc

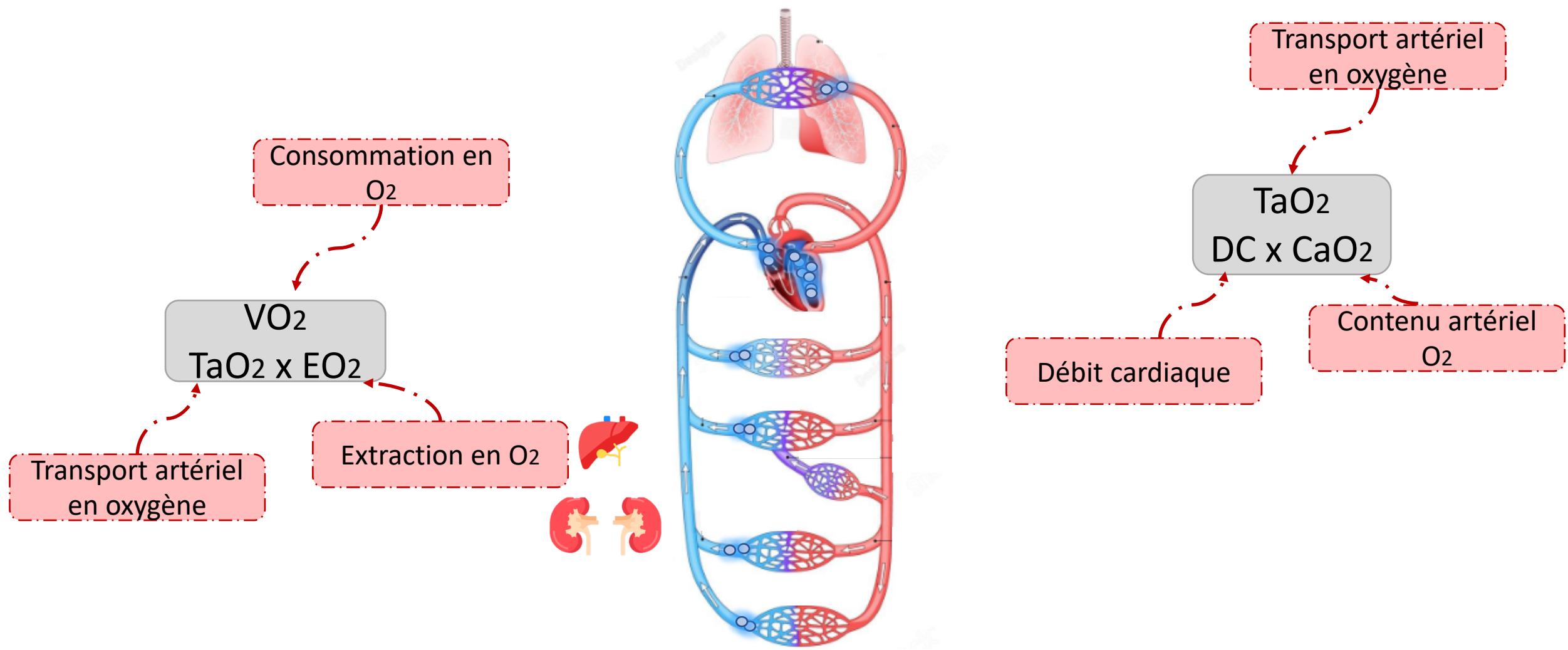


Physiologie système circulatoire

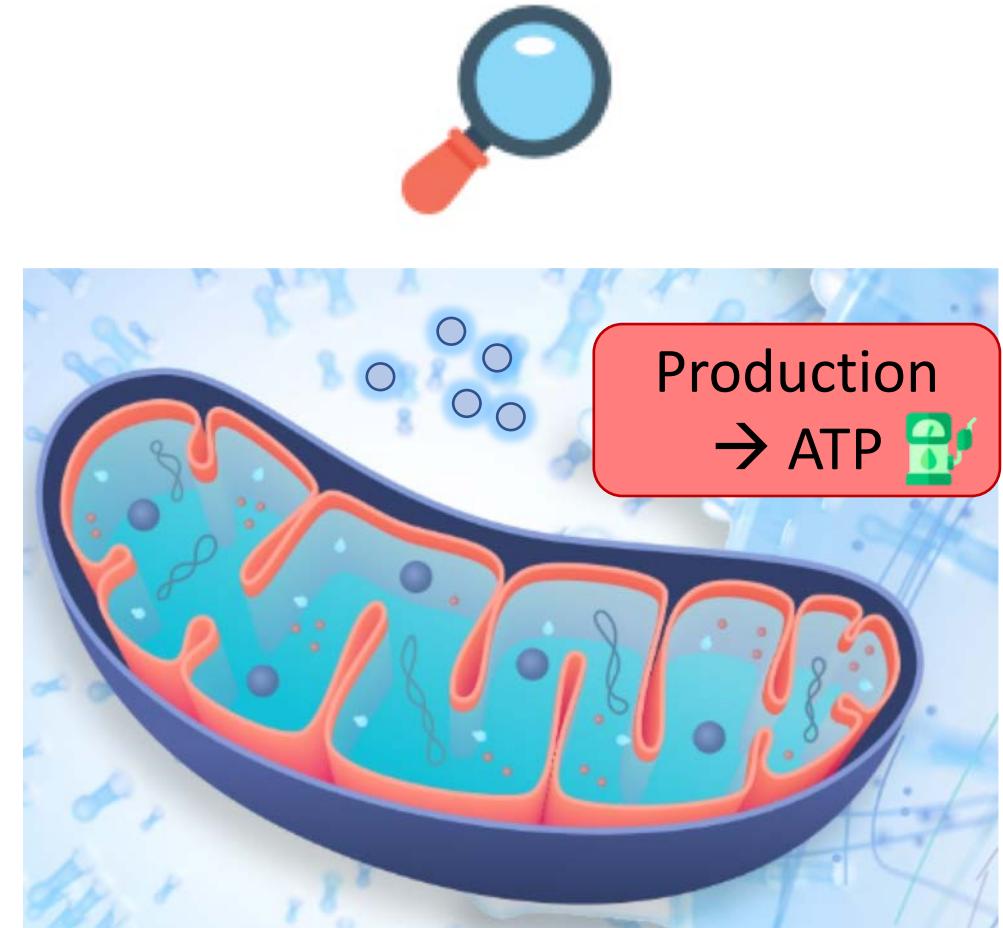
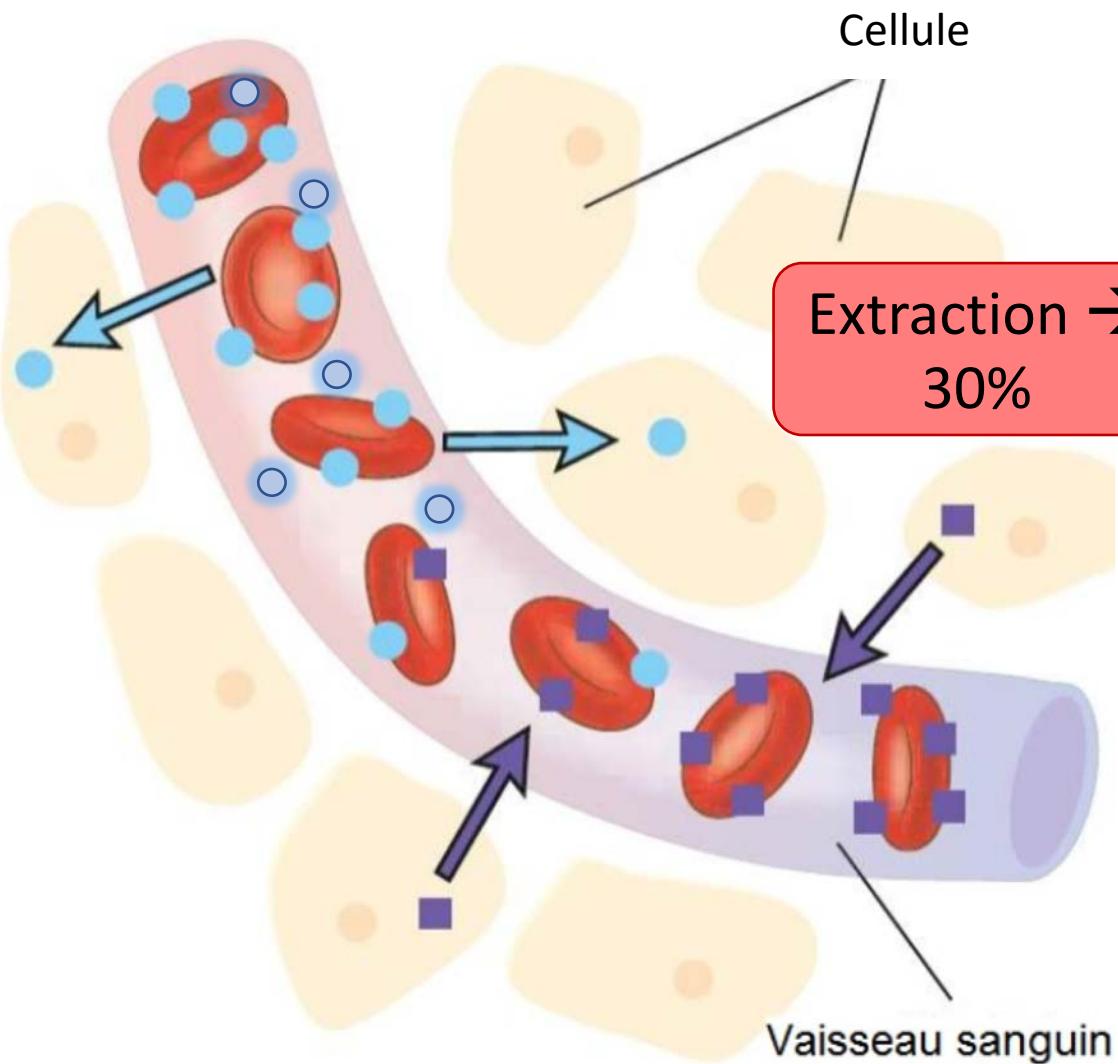
Physiologie système circulatoire



Physiologie système circulatoire



Physiologie système circulatoire

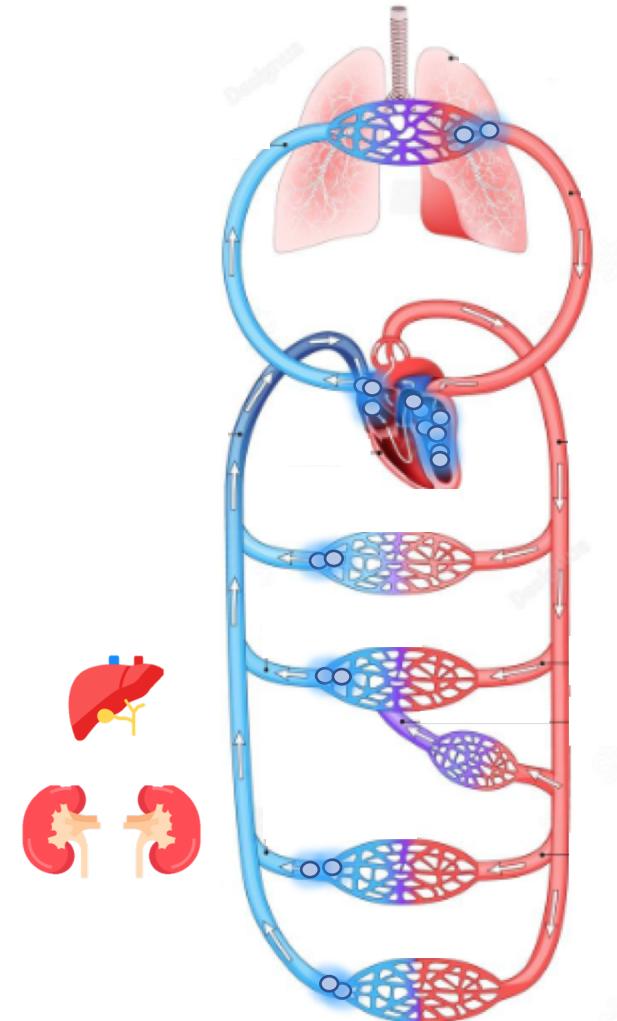


Physiopathologie de la défaillance circulatoire

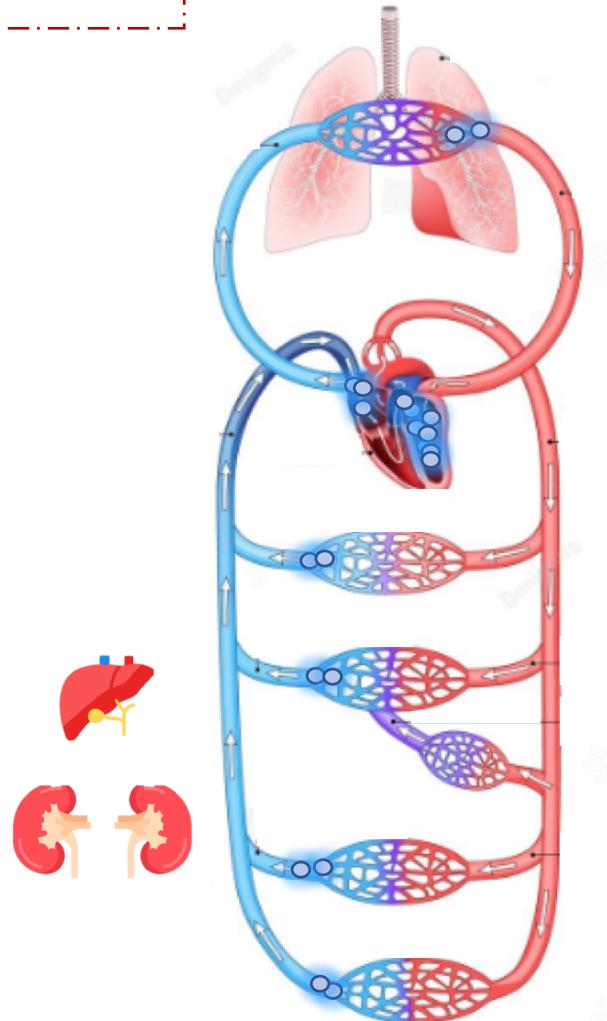
Quels sont les caractéristiques de la défaillance circulatoire ?

- ↓ du débit cardiaque
- ↓ de la précharge cardiaque
- ↓ de la contractilité
- ↓ de la pression artérielle
- Vasodilatation artérielle et veineuse

Etat de choc



Etat de choc



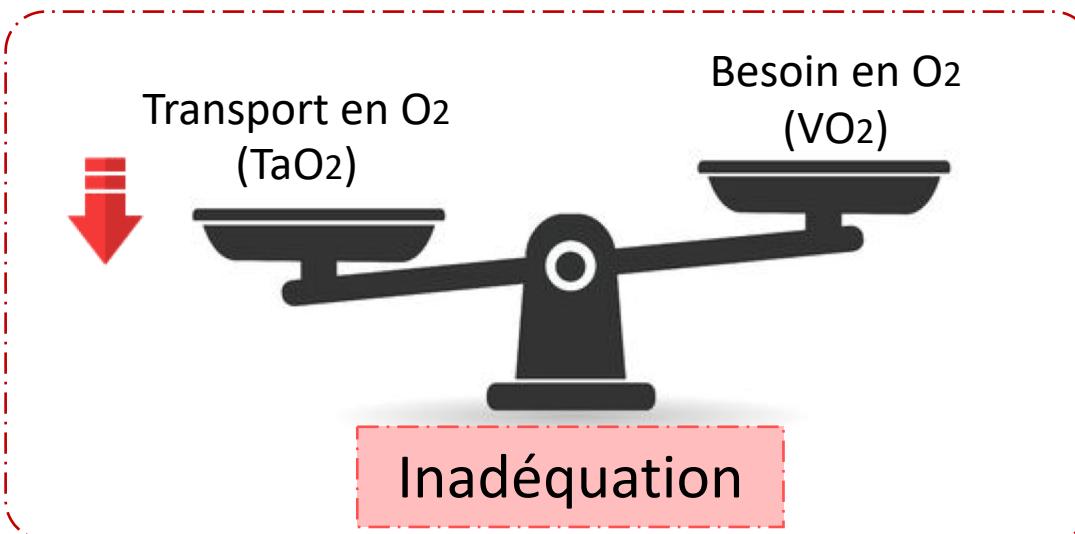
Phase ultime de défaillance circulatoire



↓ de la perfusion des organes

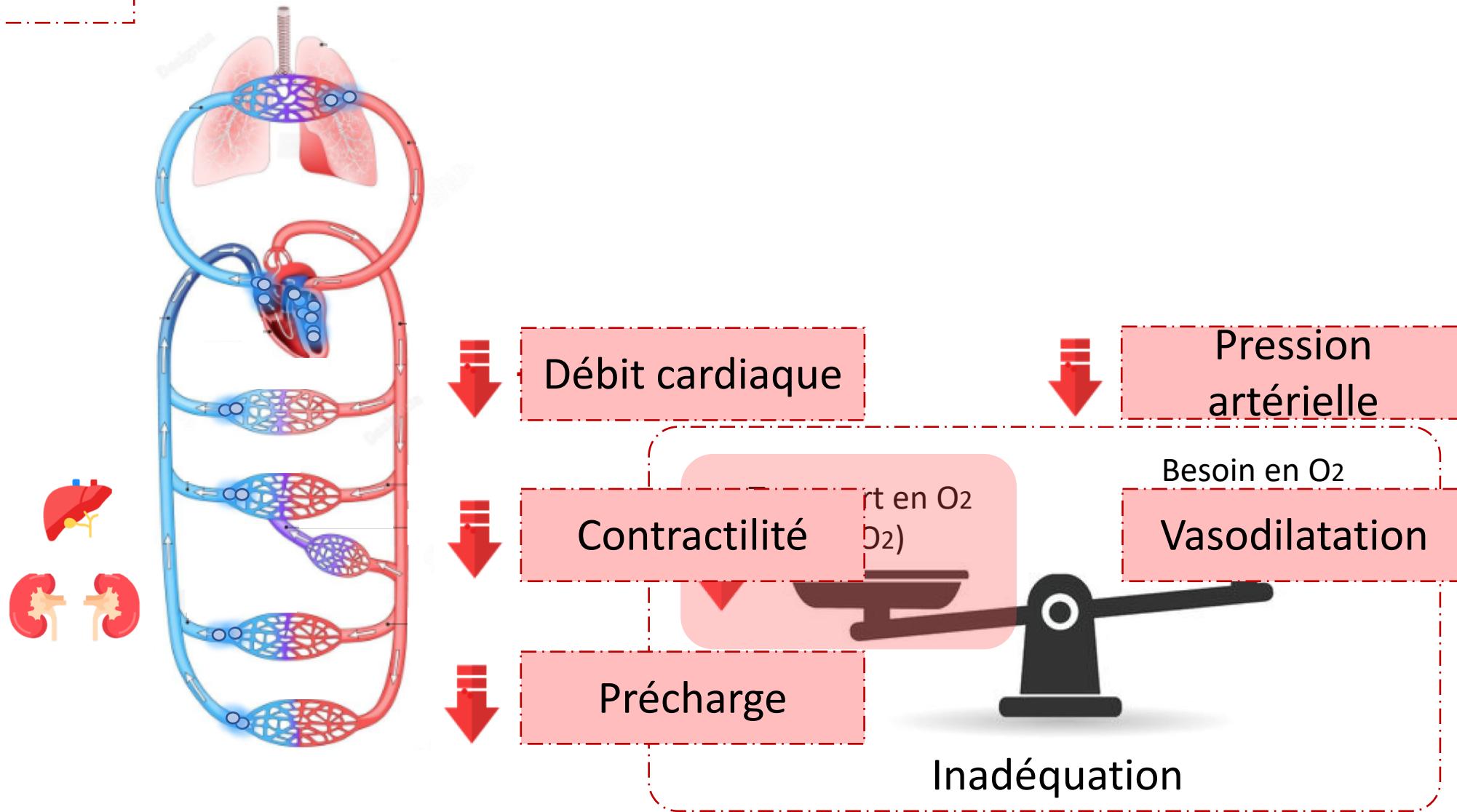


Inadéquation entre apports et besoins en O₂



Physiopathologie de la défaillance circulatoire

Etat de choc



Physiopathologie de la défaillance circulatoire

ESICM guidelines on circulatory shock and hemodynamic monitoring 2025.

Monnet X, et al. Intensive Care Med. 2025. PMID: 41236566



Insuffisance circulatoire

HOW SHOULD ONE DEFINE SHOCK?

→ Shock is defined as a life-threatening acute circulatory failure characterized by decreased tissue perfusion, leading to inadequate oxygen delivery and/or oxygen utilization to meet cellular metabolic demands.

2014 We define circulatory as a life-threatening, generalized form of acute circulatory failure associated with inadequate oxygen utilization by the cells. As a result, there is tissue hypoxia, associated with increased blood lactate levels. Shock can be associated with four underlying patterns: three associated with a low flow state (obstructive, hypovolemic, and septic) and one associated with a hyperkinetic state (distributive). Shock can be due to a combination of processes. [UNGRADED, STATEMENT OF FACT]

→ The typical features are hypotension, tachycardia, and signs of hypoperfusion, such as abnormal skin perfusion, decreased urine output, and altered mental status. Although hypotension is commonly present, it is not required to define shock.

2014 Shock is typically associated with evidence of inadequate tissue perfusion on physical examination. The three organs most commonly affected are the: skin (degree of cutaneous perfusion); kidneys (urine output); and brain (mental status). [UNGRADED, STATEMENT OF FACT]

→ Lactate levels are typically increased (>2 mmol/L) in shock states.

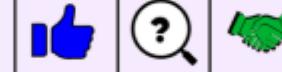
2014 Lactate levels are typically >2 mEq/L (or mmol/L) in shock states. [UNGRADED, STATEMENT OF FACT]

↗ lactate

→ Do not use a single variable (for the diagnosis and/or management of shock). [UNGRADED, BEST PRACTICE STATEMENT]



Inadéquation entre besoin et apport en O₂



↓PA, ↑FC, signes hypoperfusion



Etat de choc



Hypovolémique

Cardiogénique

Distributif

Obstructif

Etat de choc



Hypovolémique

Cardiogénique

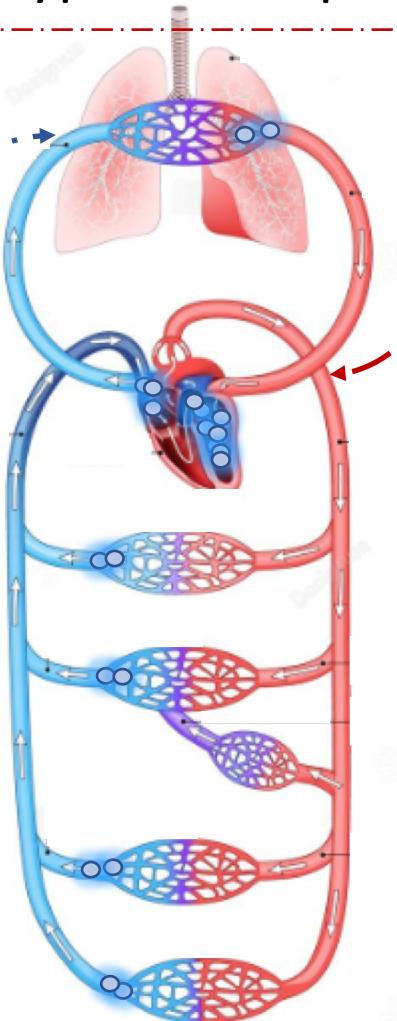
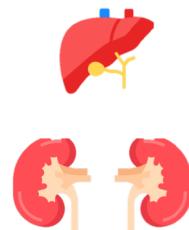
Distributif

Obstructif

Physiopathologie de la défaillance circulatoire

Etat de choc hypovolémique

↓ volume sanguin



↓ débit cardiaque

↓ TaO_2
 $\text{DC} \times \text{CaO}_2$



Précharge



Perte liquide



Perte sang

Pertes digestives, cutanées...

Polytraumatisé, hémorragie dig

Etat de choc



Hypovolémique

Cardiogénique

Distributif

Obstructif

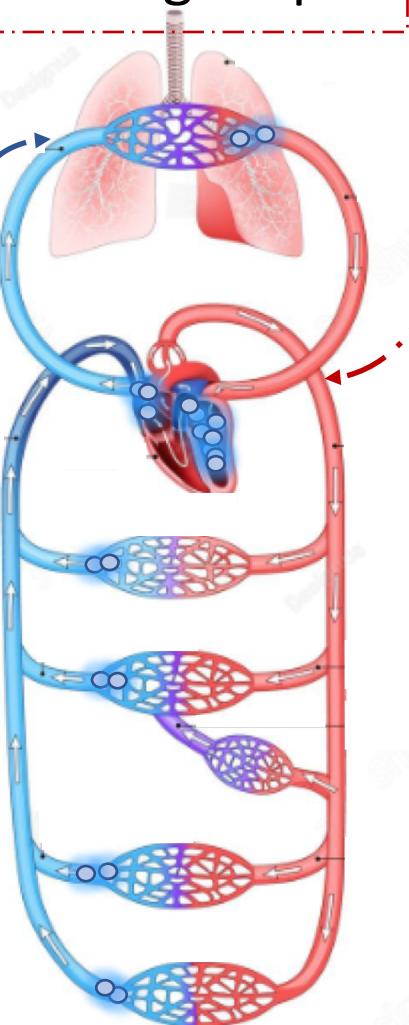
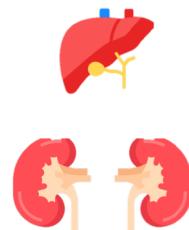
Physiopathologie de la défaillance circulatoire

Etat de choc cardiogénique

↓
contractilité

↓ débit
cardiaque

↓ TaO_2
 $DC \times CaO_2$



Contractilité



Infarctus

Lésions
ischémiques
coronariennes

Myocardite

Inflammation
du tissu
myocardique

Etat de choc



Hypovolémique

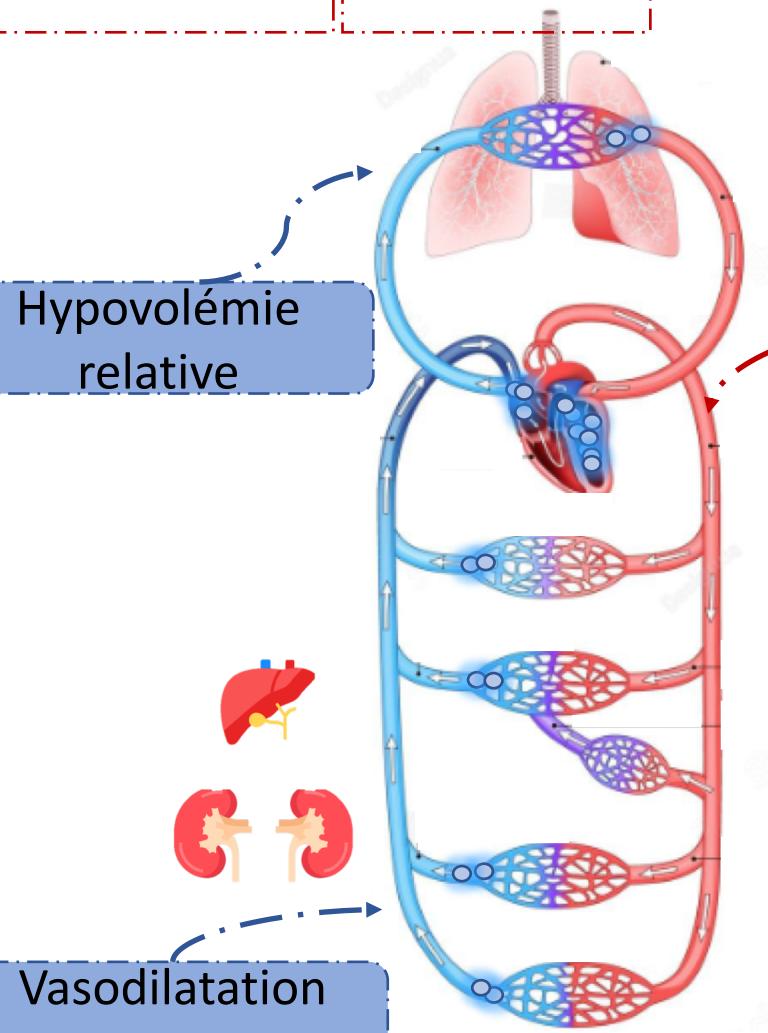
Cardiogénique

Distributif

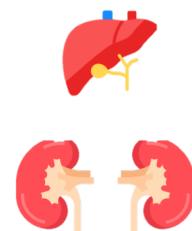
Obstructif

Physiopathologie de la défaillance circulatoire

Etat de choc distributif



Hypovolémie relative



Vasodilatation veineuse

Vasodilatation artérielle

↓ Contractilité

↓ Extraction O₂



Pression artérielle



Choc septique

Choc anaphylactique

Pneumonie, pyélonéphrite

Allergie médicaments

Etat de choc



Hypovolémique

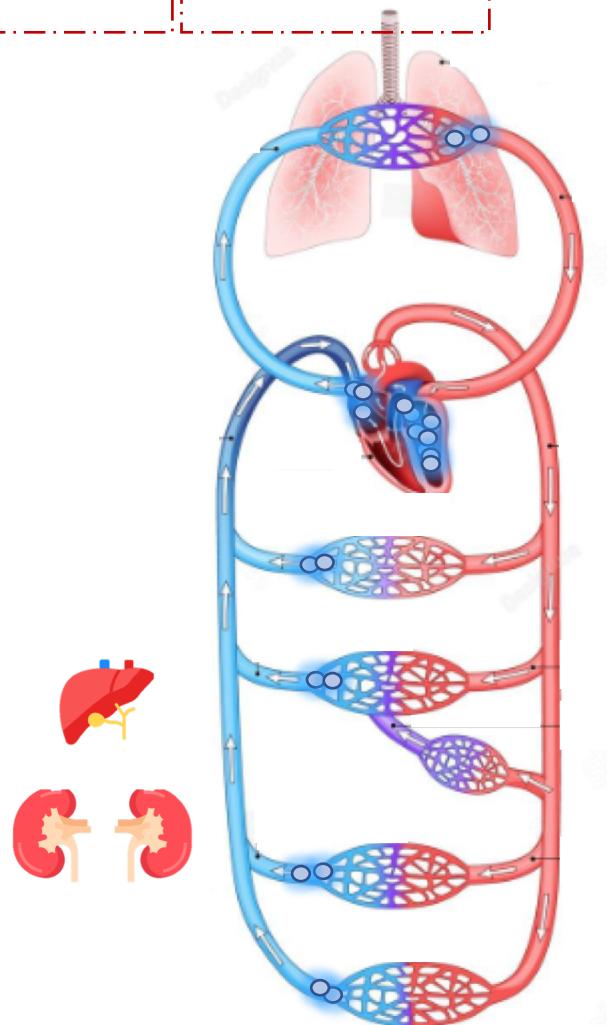
Cardiogénique

Distributif

Obstructif

Physiopathologie de la défaillance circulatoire

Etat de choc obstructif



Obstacle
éjection VD



Choc obstructif

EP

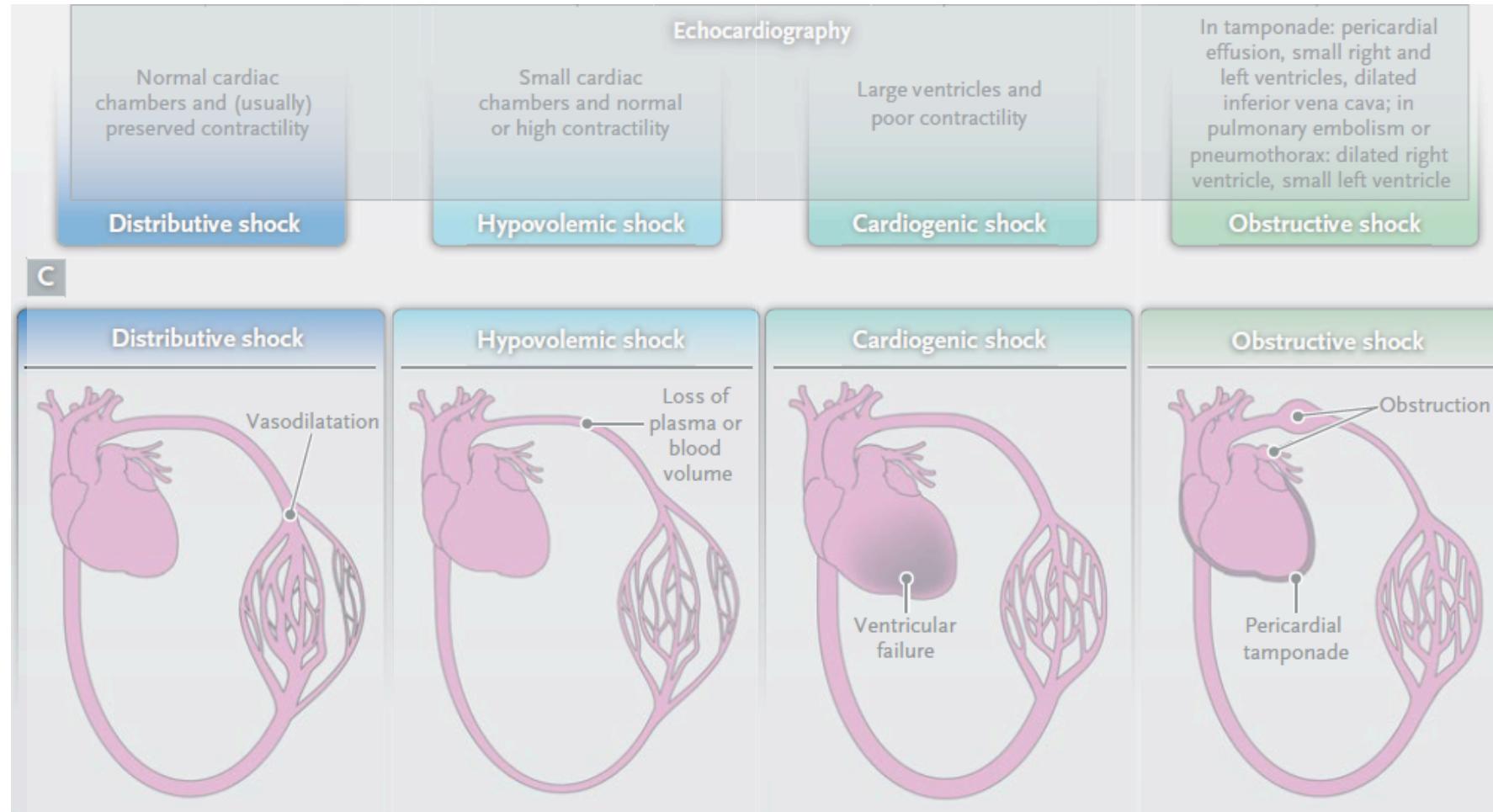
Tamponnade

Circulatory shock.

Vincent JL, De Backer D.

N Engl J Med. 2013 Oct 31;369(18):1726-34.

Etat de choc



1

2

3

4

1

Etat de choc → inadéquation entre apports et besoins en O₂

2

3

4

1

Etat de choc → inadéquation entre apports et besoins en O₂

2

4 grands types de choc caractérisés par une ↘ du TaO₂ (DC) et/ou ↘ de PA

3

4

Mécanismes adaptatifs

Quels sont les mécanismes adaptatifs ?

- ↑ l'extraction en oxygène
- ↓ l'inotropisme
- ↑ l'inflammation
- Vasodilatation la microcirculation
- Vasoconstriction artérielle et veineuse

1 Système Σ

1 Système Σ



Barorécepteurs

Sinus carotidien
Crosse aortique

↗ fréquence cardiaque

↗ débit cardiaque



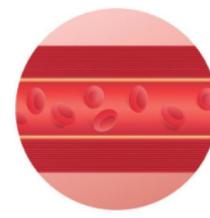
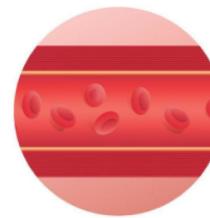
Stimulation adrénaline physiologique

Ionotropisme ↑ Vasoréactivité ↓ Inflammation

Activation adrénaline persistante

Ionotropisme ↓ Vasoréactivité Immunosuppression

Dysautonomie



Vasoconstriction

↗ pression artérielle

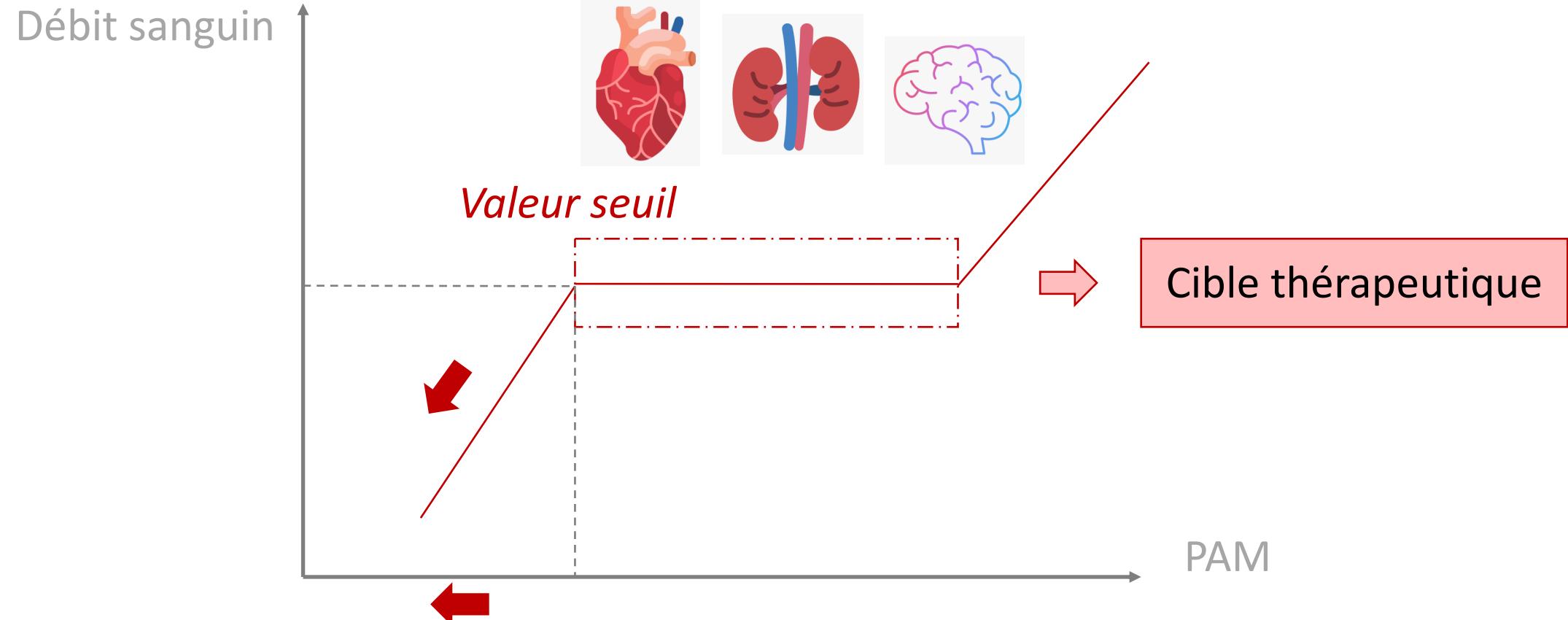


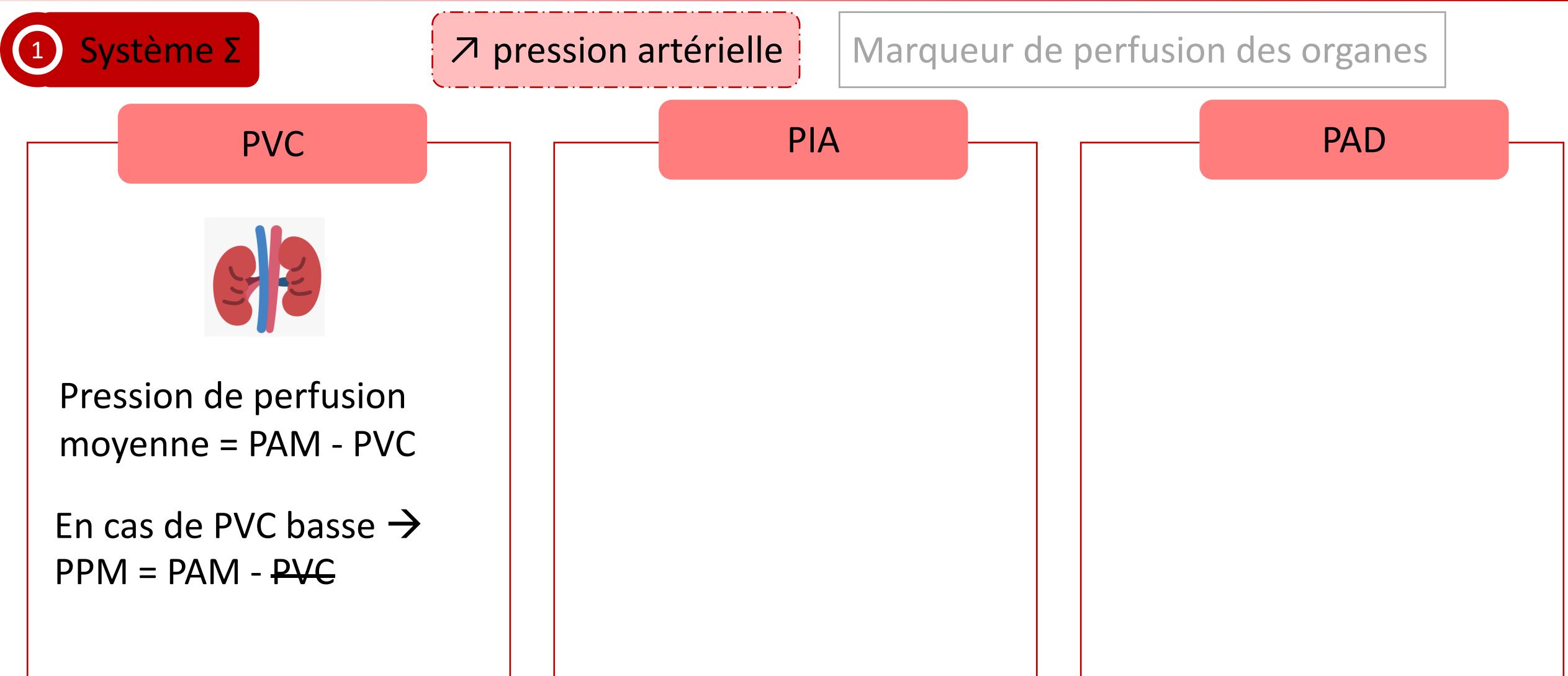
PA Moyenne

1 Système Σ

↗ pression artérielle

Marqueur de perfusion des organes





1 Système Σ

↗ pression artérielle

Marqueur de perfusion des organes

Low **mean perfusion pressure** is a risk factor for progression of **acute kidney injury** in critically ill patients - A retrospective analysis.

Ostermann M, Hall A, Crichton S.

BMC Nephrol. 2017 May 3;18(1):151.

Rétrospectif
n=2,118

Parameter	OR (95% CI) ^a	p-value
First arterial lactate following diagnosis of AKI I [mmol/L]	1.45 (1.12–1.89)	0.005
SOFA score on day of AKI I	1.20 (1.05–1.37)	0.01
First DO ₂ I in 12 h period after diagnosis of AKI I [ml/min/m ²]	0.997 (0.994–0.99)	0.01
First calculated MPP	0.995 (0.92–0.99)	0.03
Age [years]	1.02 (0.997–1.05)	0.09
Cumulative fluid balance on day of AKI I [ml]	1.00 (0.99–1.00)	0.98
MAP <65 mmHg for >1 h in first 12 h after diagnosis of AKI I	0.97 (0.48–1.96)	0.93

Pression de perfusion moyenne
est associée à l'IRA

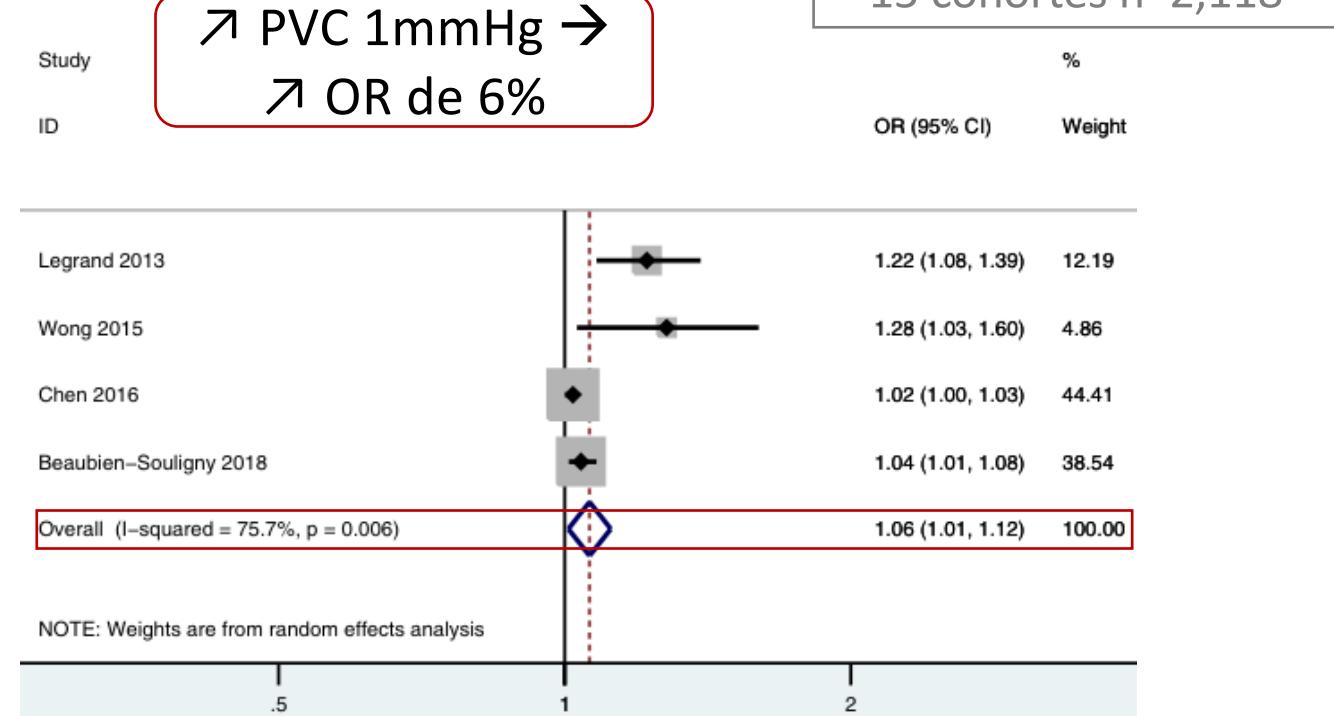
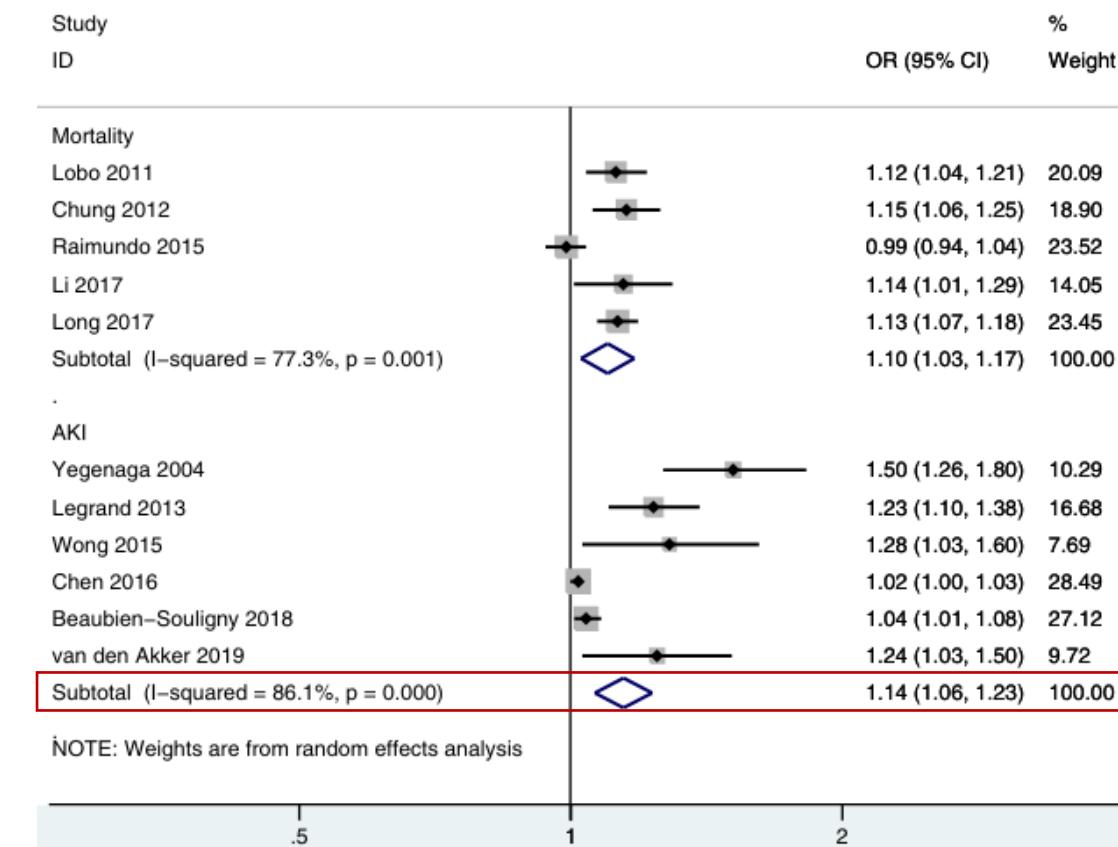
1 Système Σ

↗ pression artérielle

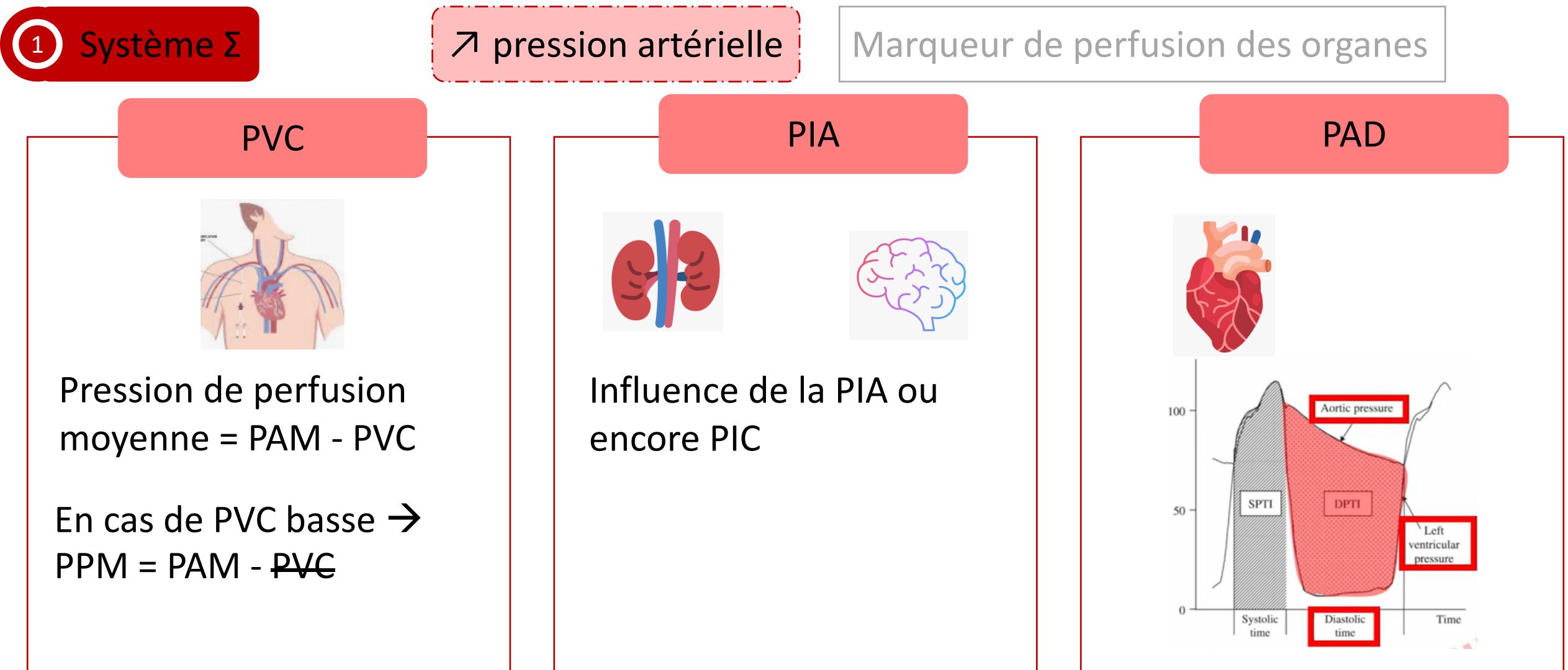
Marqueur de perfusion des organes

Elevated central venous pressure is associated with increased mortality and acute kidney injury in critically ill patients: a meta-analysis.

Chen CY, et al. Crit Care. 2020.



PVC élevée → mortalité ↗



Perfusion des organes dépend aussi d'autres déterminants hémodynamiques

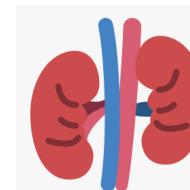
1 Système Σ

↗ pression artérielle

Marqueur de perfusion des organes

Dysfonction d'organes

- ✓ Facteur de risque indépendant
- ✓ Selon la profondeur, la durée de l'hypotension



< 65mmHg < 70mmHg < 65mmHg
↗ troponine ↗ créat ou EER ↗ délirium

Mortalité

- ✓ Facteur de risque indépendant
- ✓ Selon la profondeur, la durée de l'hypotension

Seuil optimal variable

1 Système Σ

↗ pression artérielle

Marqueur de perfusion des organes

ESICM guidelines on circulatory shock and hemodynamic monitoring 2025.

Monnet X, et al. Intensive Care Med. 2025. PMID: 41236566



WHAT IS THE TARGET OF ARTERIAL PRESSURE IN SHOCK?

→ The target blood pressure should be individualized during resuscitation of patients with shock.

Individualisation



2014 We recommend individualizing the target blood pressure during shock resuscitation. [LEVEL 1, QoE B]

→ An initial MAP of 65-70 mmHg should be targeted in patients with septic shock.

PAm

65-70mmHg



2014 We recommend to initially target a MAP of ≥ 65 mmHg. [LEVEL 1, QoE C]

→ A higher MAP target may be considered in patients with septic shock and a history of chronic arterial hypertension who show clinical improvement with higher blood pressure.



→ A higher MAP target may be considered in patients with septic shock with high CVP values who show clinical improvement with higher blood pressure.



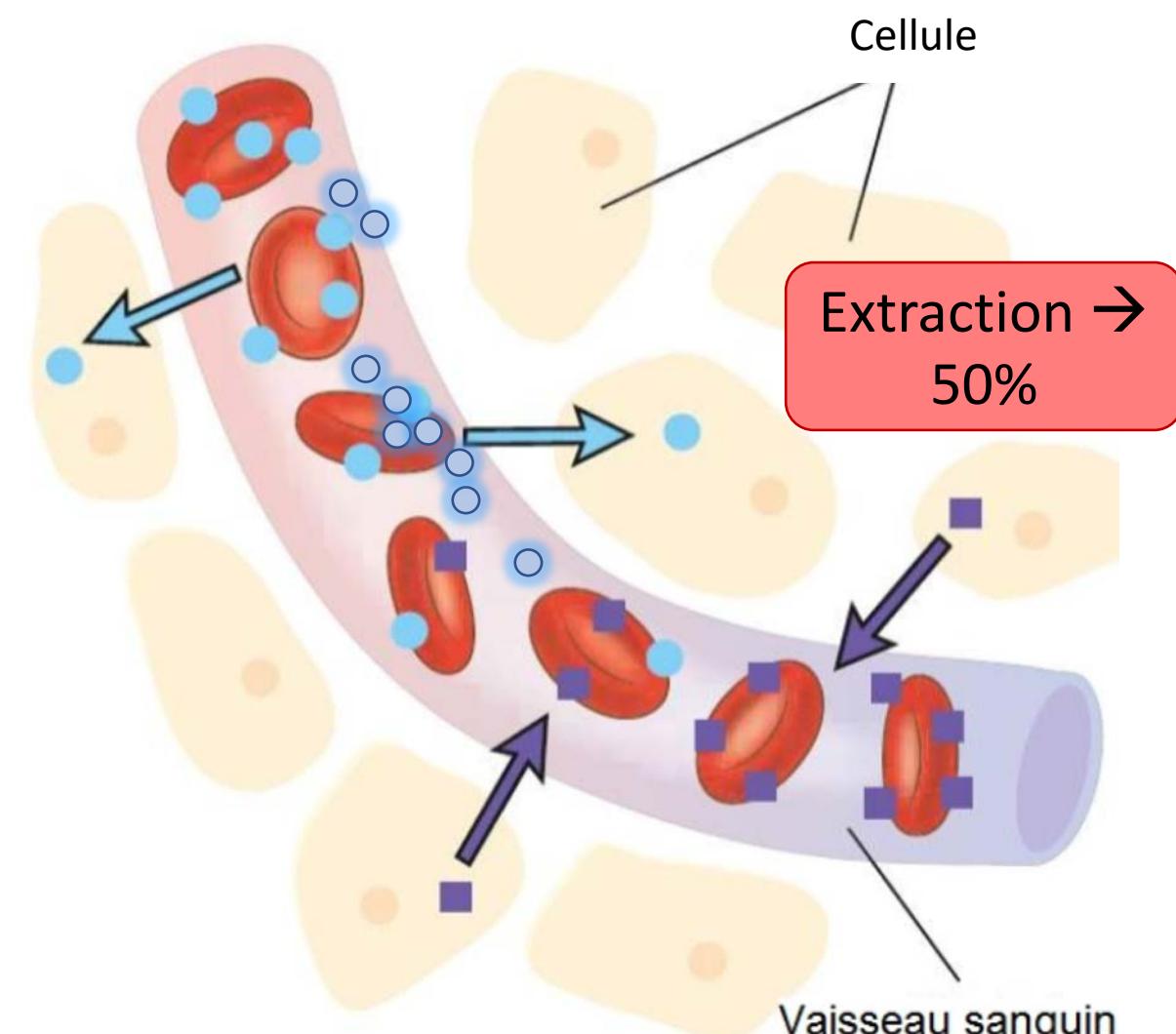
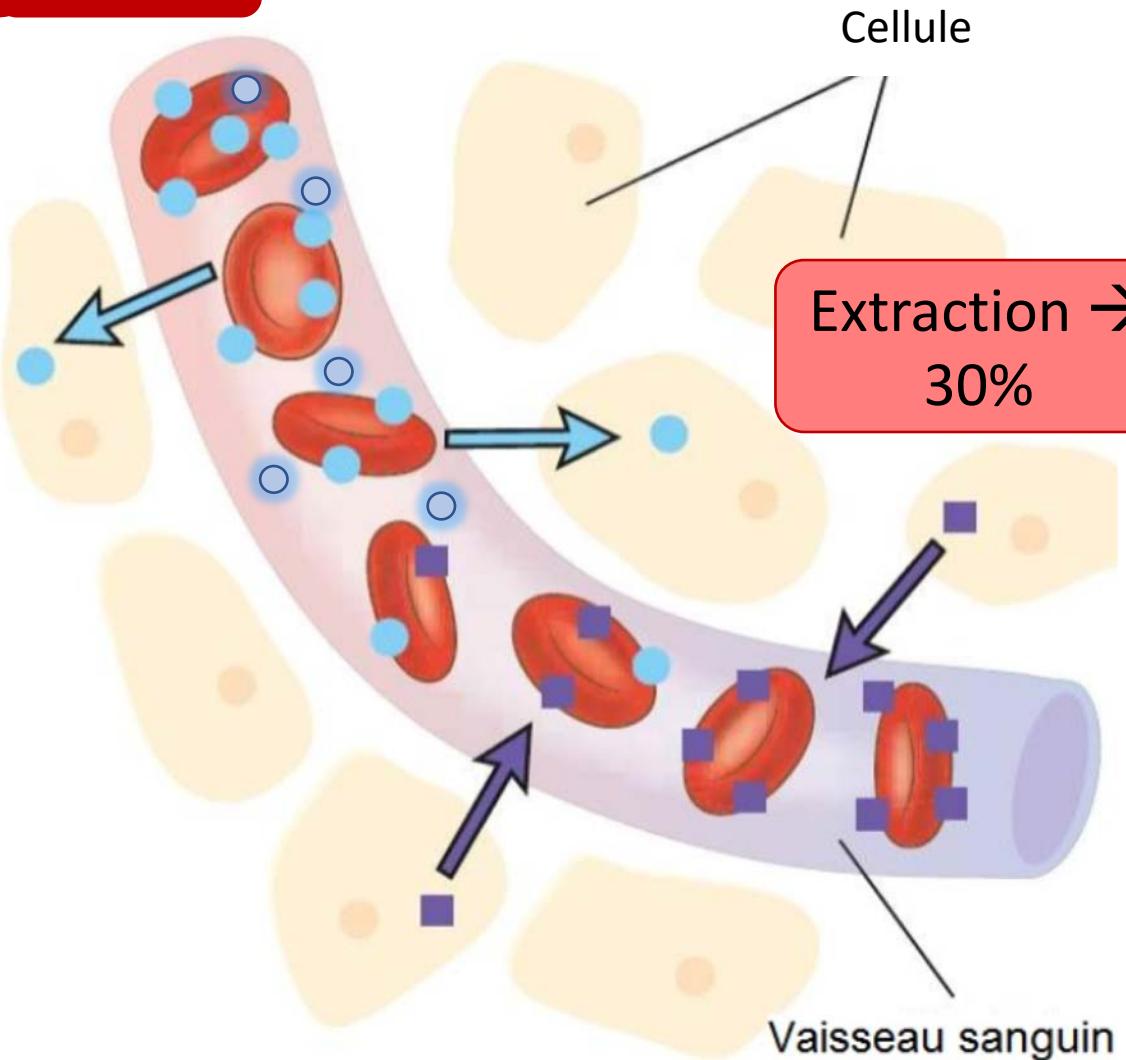
↗ PAm si
PVC élevée



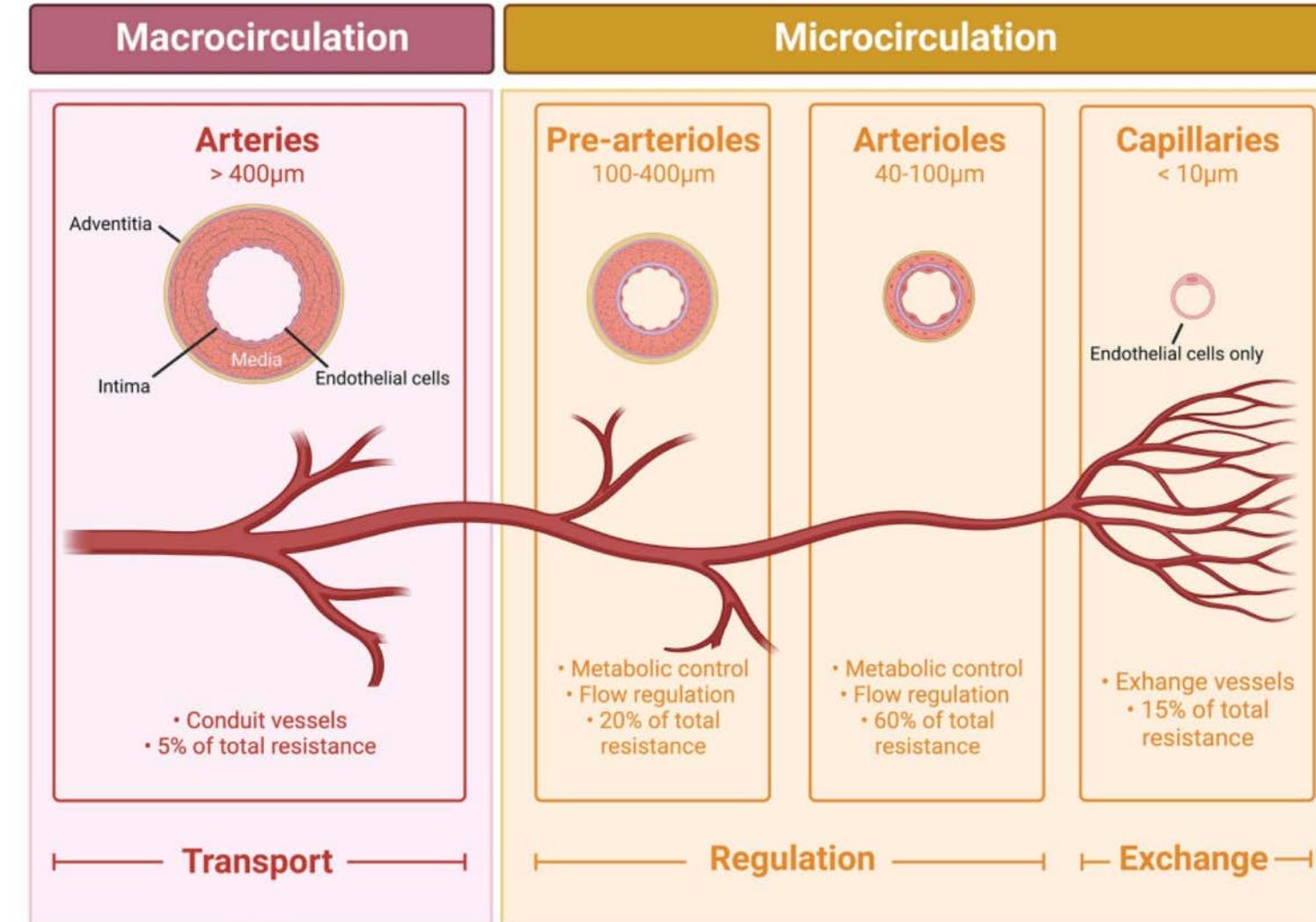
② $\uparrow \text{EO}_2$

Mécanismes adaptatifs

② $\uparrow \text{EO}_2$



② \rightarrow EO₂

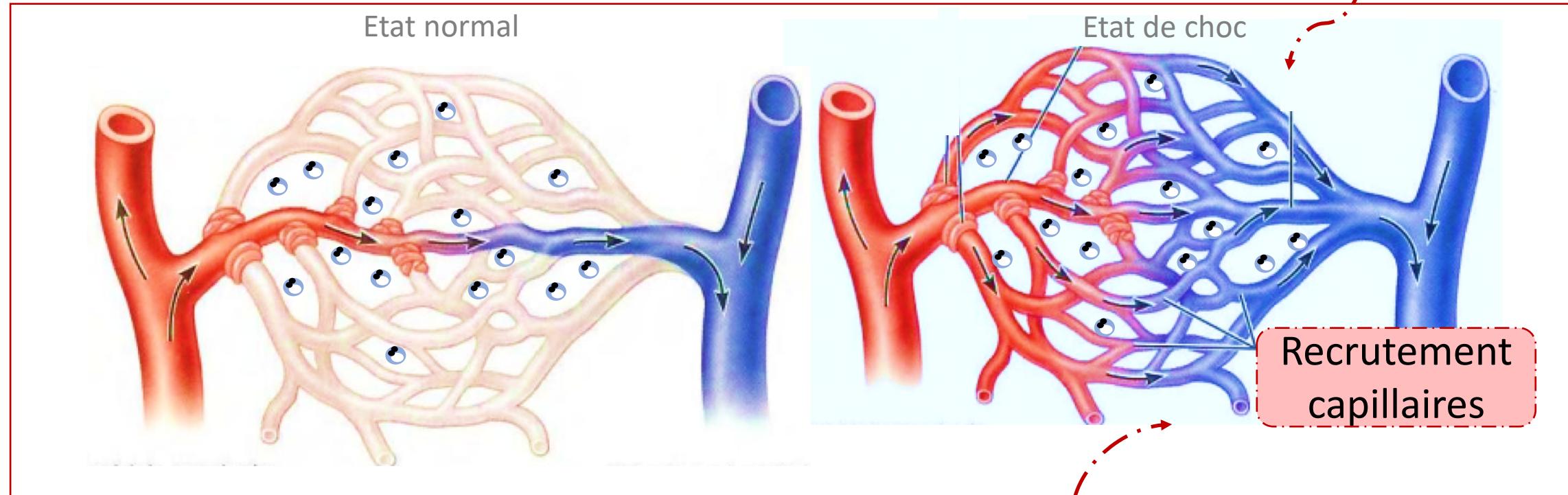


Mécanismes adaptatifs

② $\uparrow EO_2$

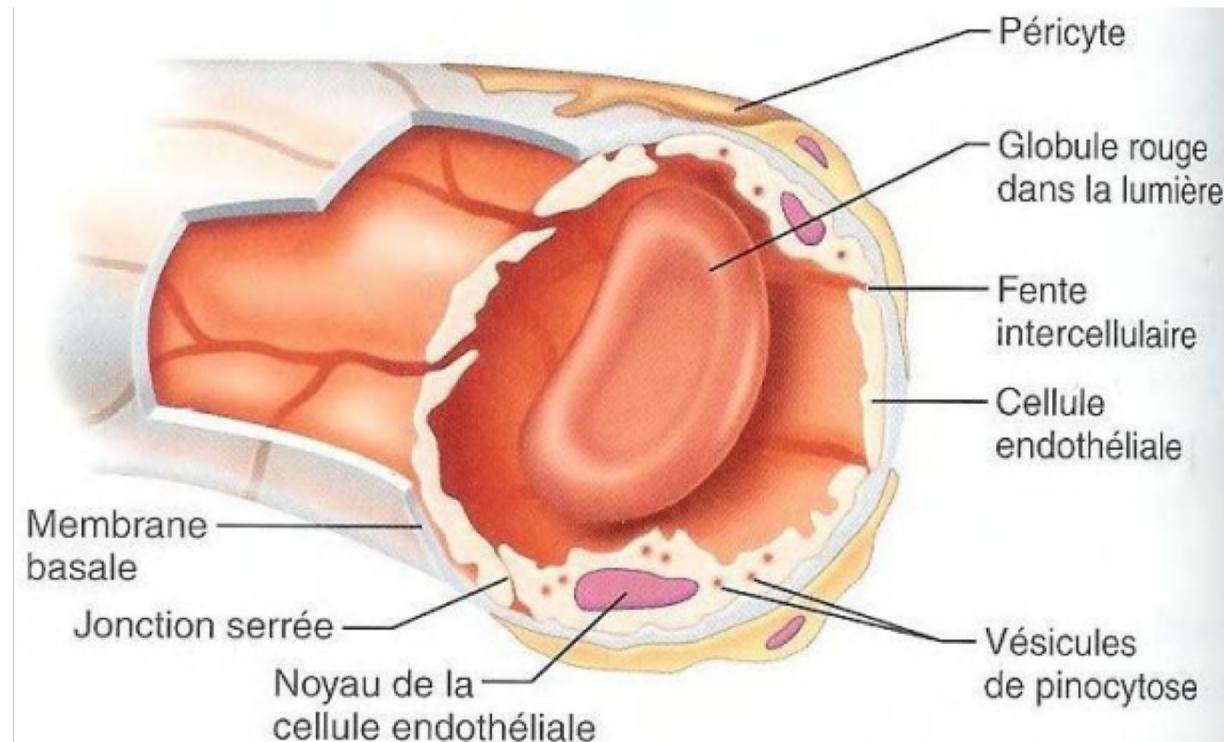


\uparrow surface d'échanges

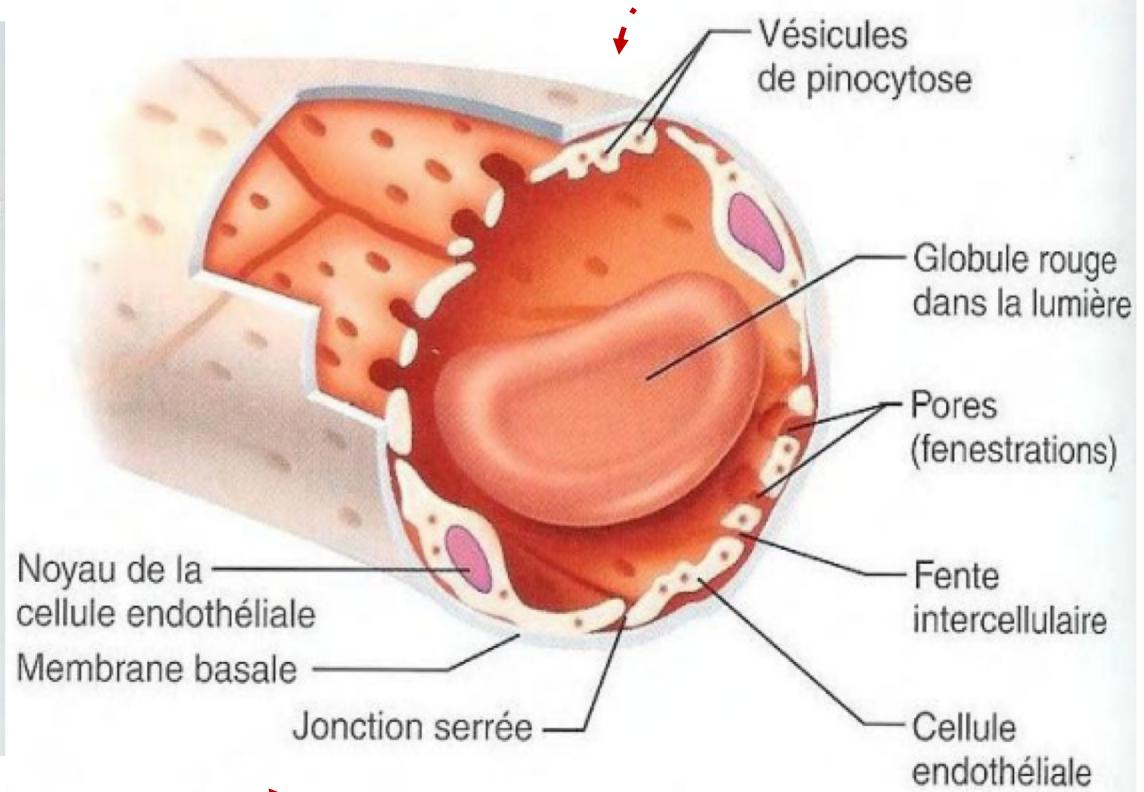


Vasodilatation μcirculation

② \uparrow EO₂

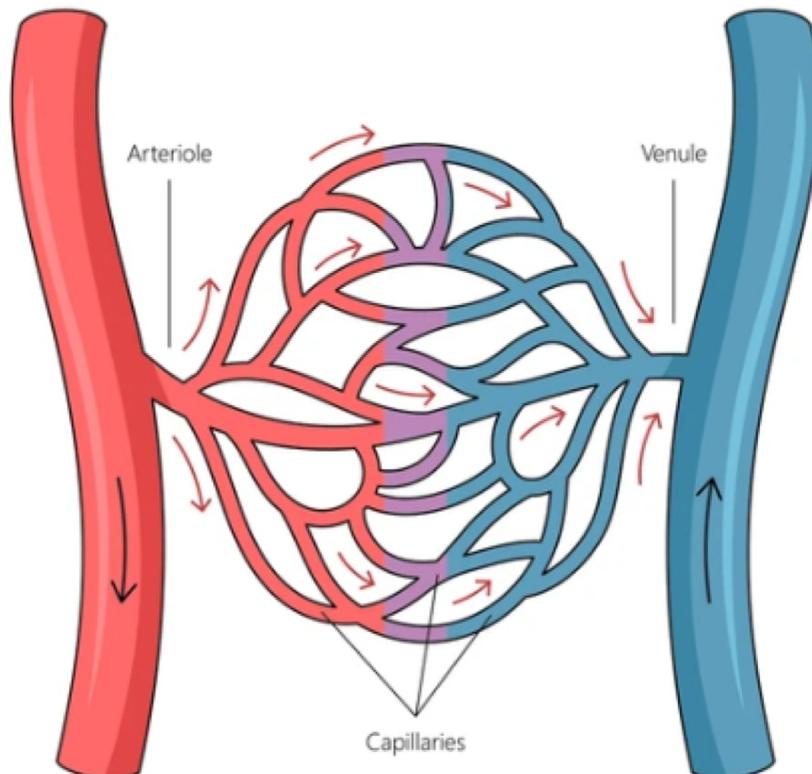


Ouverture de pores



\uparrow perméabilité

② $\uparrow EO_2$

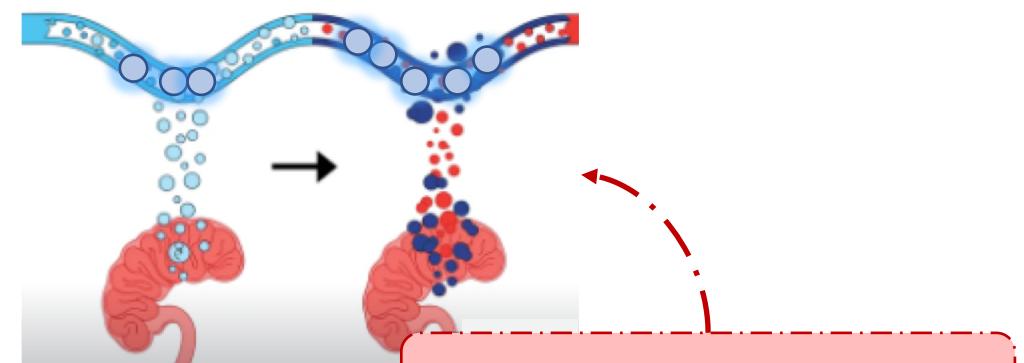


Vasodilatation μ circulation



NO

Prostaglandines

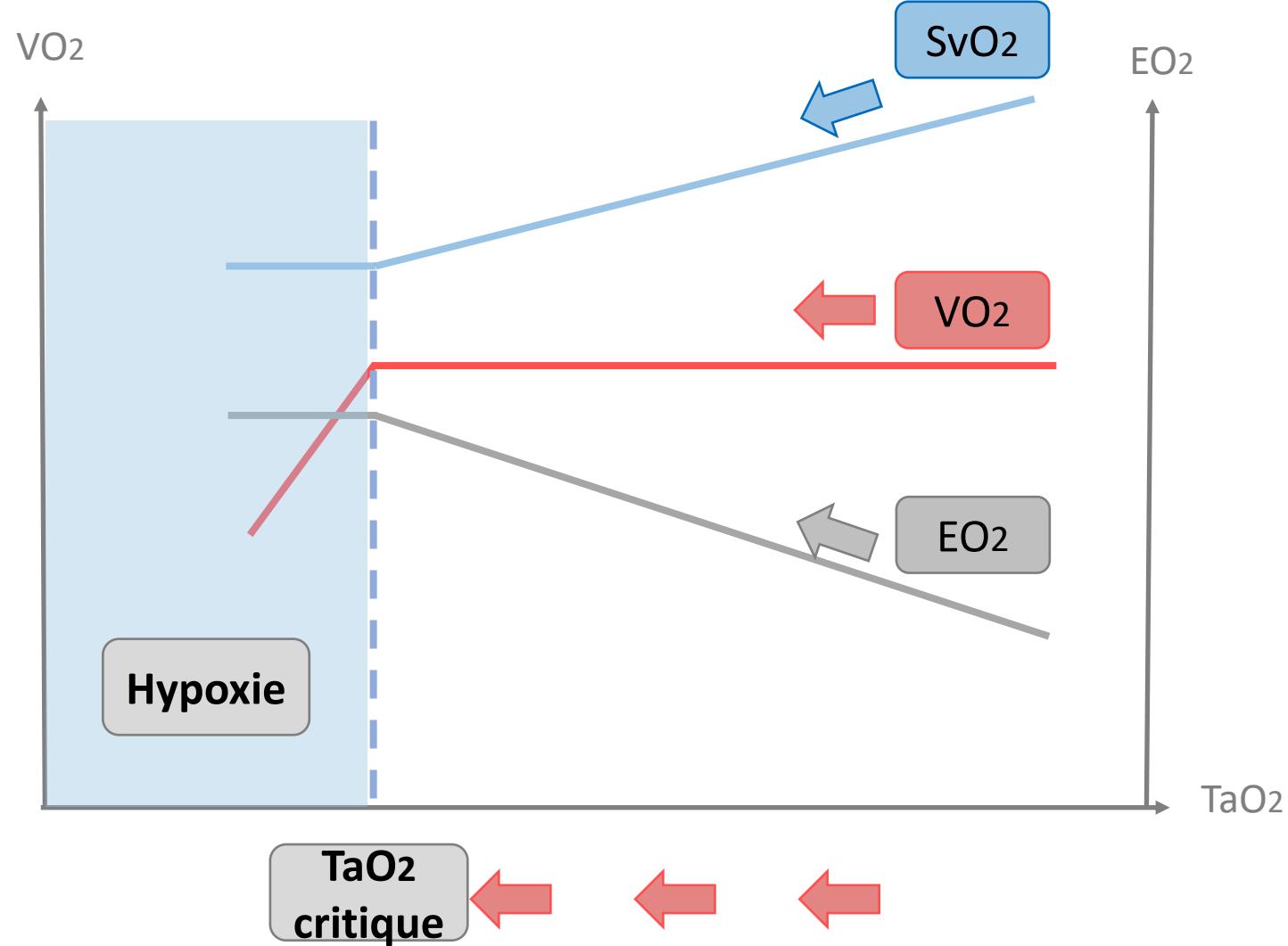


\uparrow Extraction

\uparrow surface d'échanges

Mécanismes adaptatifs

② $\uparrow EO_2$



Mécanismes adaptatifs

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Monnet X, et al. Intensive Care Med. 2025. PMID: 41236566



WHAT MARKERS OF THE REGIONAL CIRCULATION AND TISSUE OXYGENATION IN SHOCK SHOULD BE USED?

→ Serial assessment of tissue perfusion should be performed to follow shock evolution and to help assess the underlying pattern and the adequacy of CO and vascular function.



2014 We recommend serial measurements of blood lactate to guide, monitor, and assess. [LEVEL 1, QoE C]

→ Monitoring skin perfusion should be performed using the assessment of CRT and this could be complemented with the assessment of skin temperature and mottling.



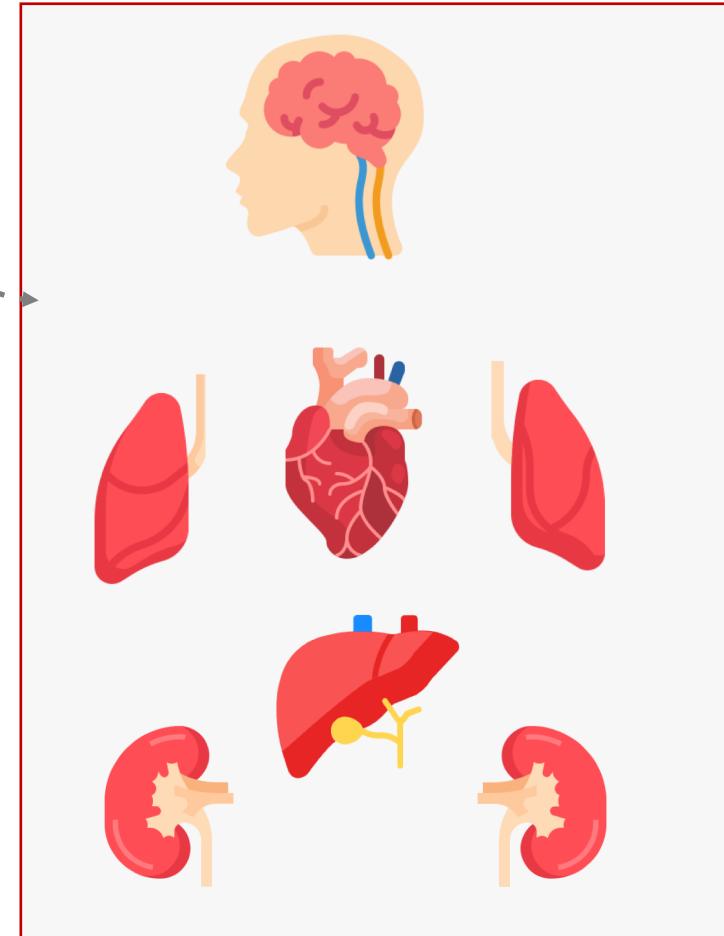
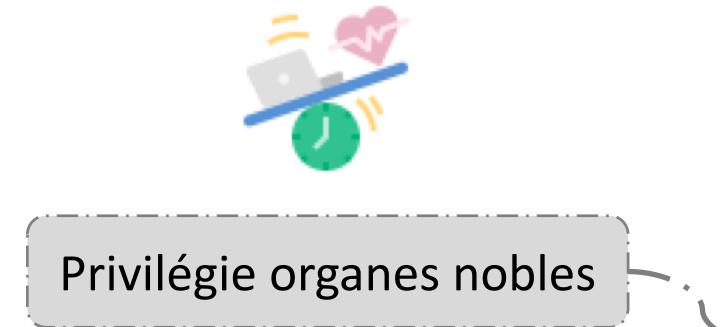
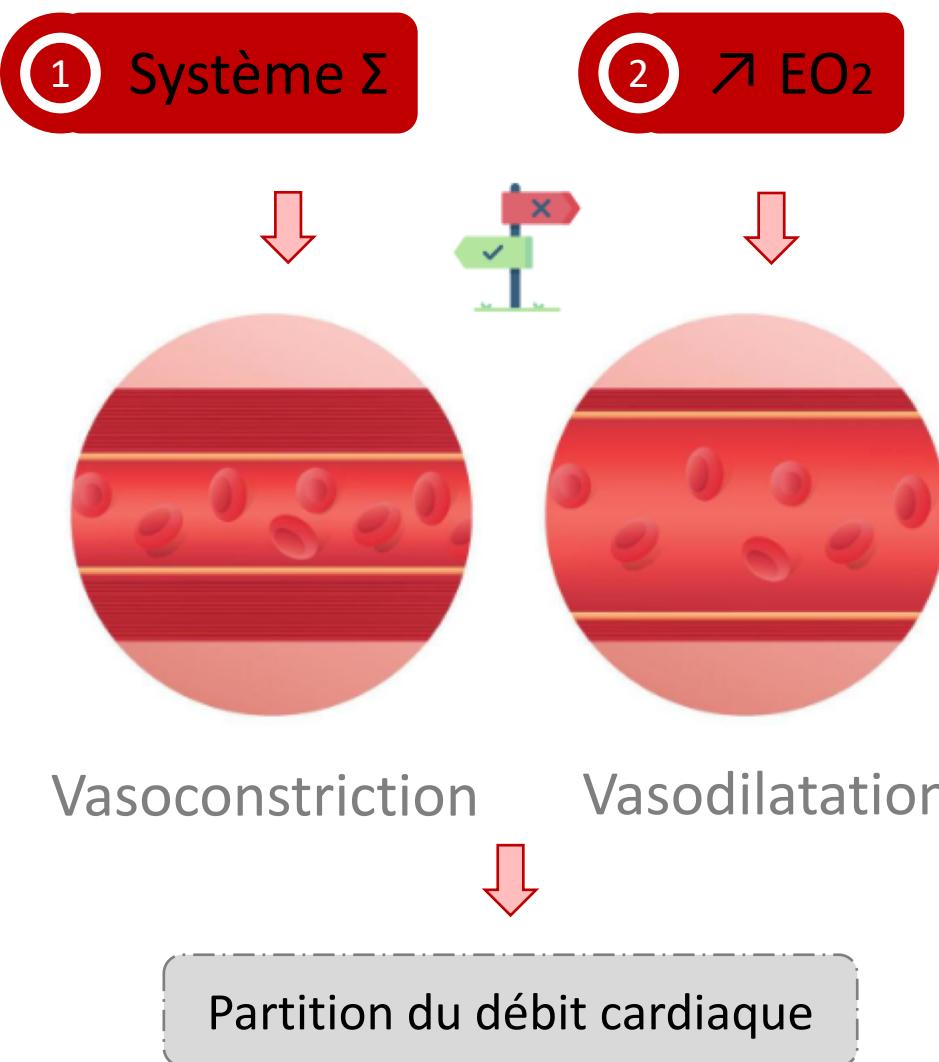
→ In patients with a central venous catheter, serial measurements of (central) venous oxygen saturation ($S_{(c)v}O_2$) should be performed.



Monitorer la
SvO₂

La SvO₂ → apprécier l'adaptation du DC aux besoins tissulaires en O₂

Mécanismes adaptatifs

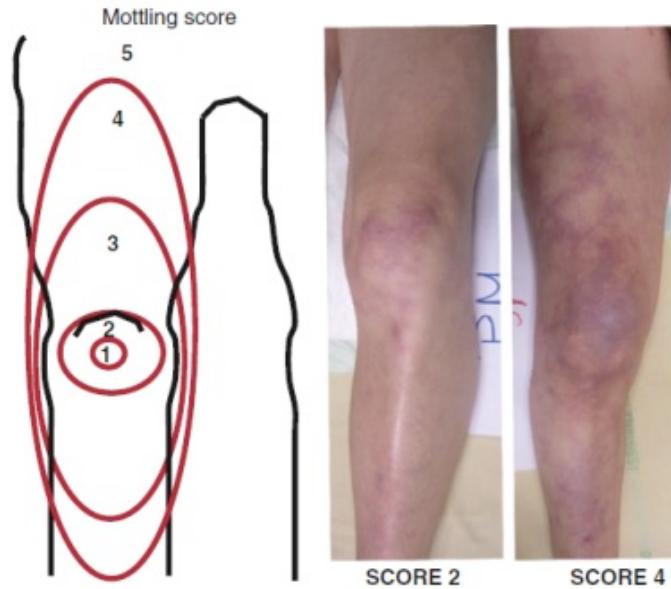


Effets variables de ces mécanismes adaptatifs selon les territoires



Marbrures

Ce que l'on connaît déjà



Signe d'insuffisance circulatoire aiguë

Marbrures

Les nouveautés

NEW

1

Intérêt pronostic

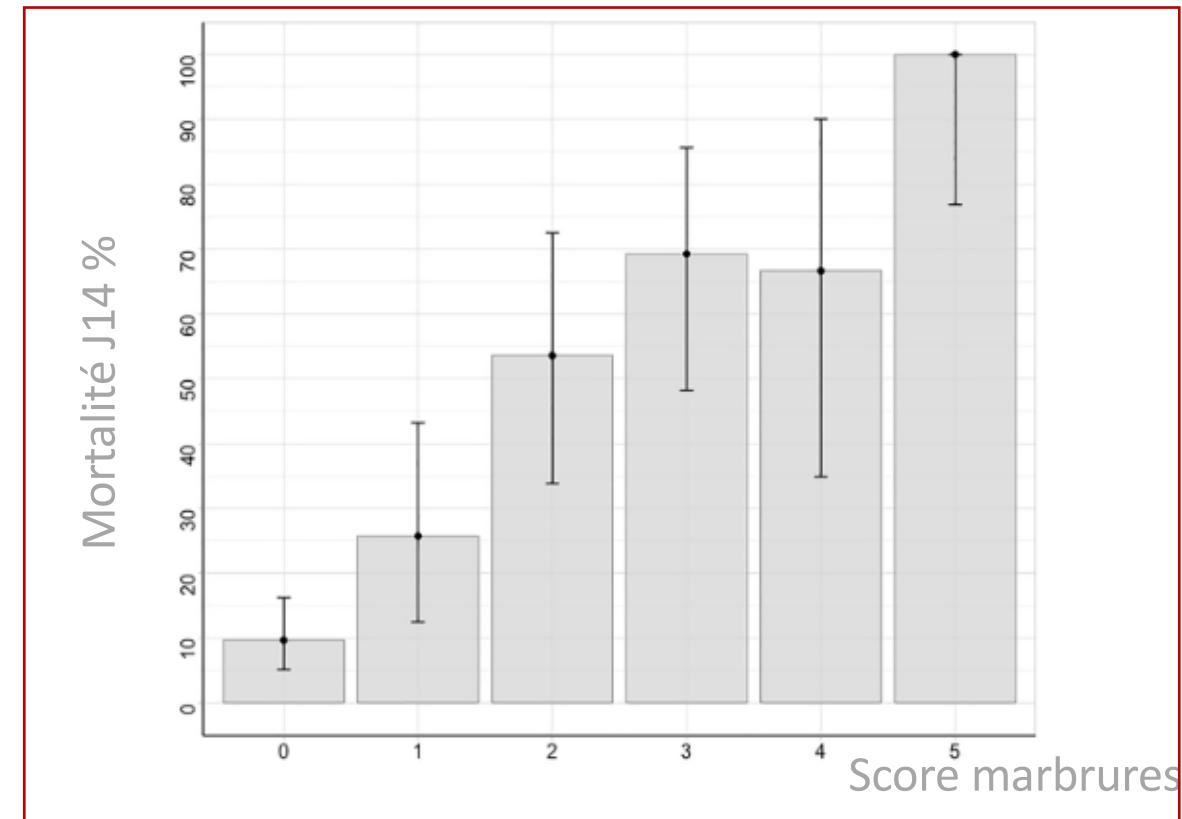
Marbrures → associée à la mortalité dans différents types de choc

Mottling score is a strong predictor of 14-day mortality in septic patients whatever vasopressor doses and other tissue perfusion parameters.

Dumas G, Lavillegrand JR, Joffre J, Bigé N, de-Moura EB, Baudel JL, Chevret S, Guidet B, Maury E, Amorim F, Ait-Oufella H.

Crit Care. 2019 Jun 10;23(1):211.

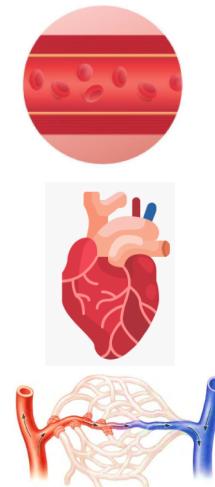
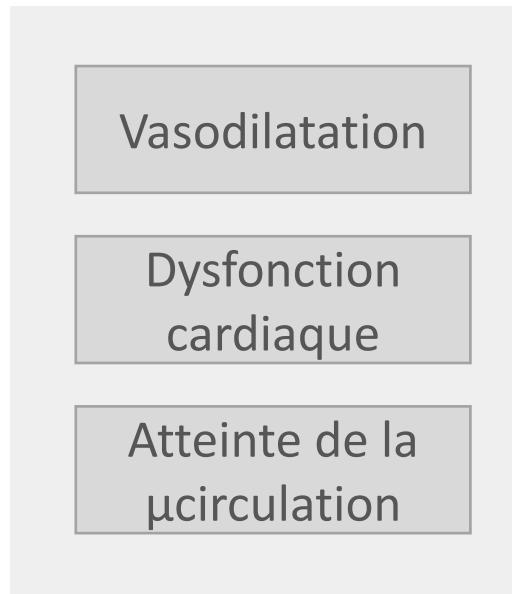
259 chocs septiques



Augmentation mortalité

Mécanismes adaptatifs : le cas du choc septique

Etat de choc



- 
- Hypovolémique
 - Cardiogénique
 - Distributif
 - Obstructif

Mécanismes compensateurs



- 1 Système Σ
- 2 ↑ EO₂

Mécanismes adaptatifs : le cas du choc septique

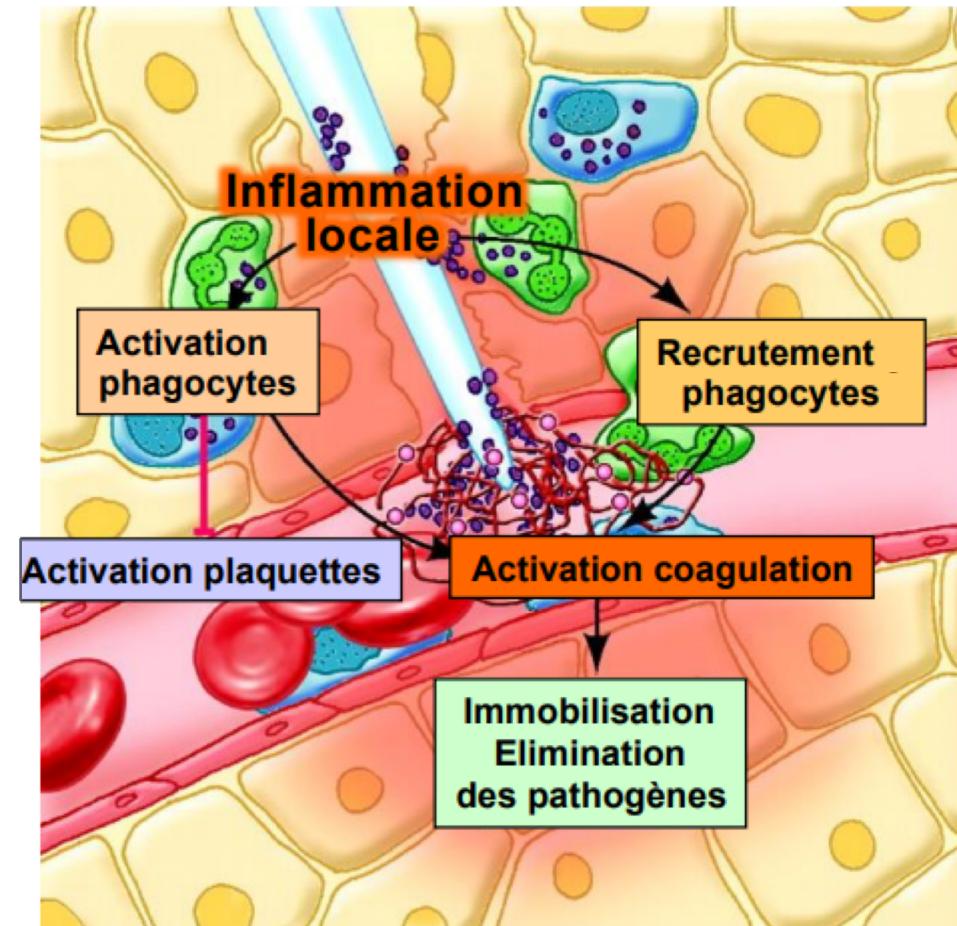
Choc septique

Dysfonction de la microcirculation

Microthrombosis

Shunt AV

Inflammation

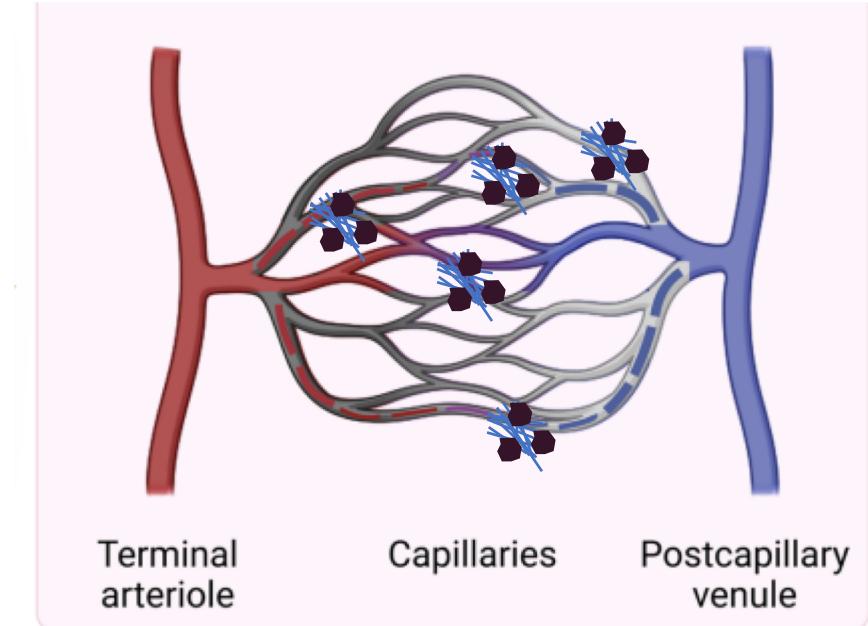




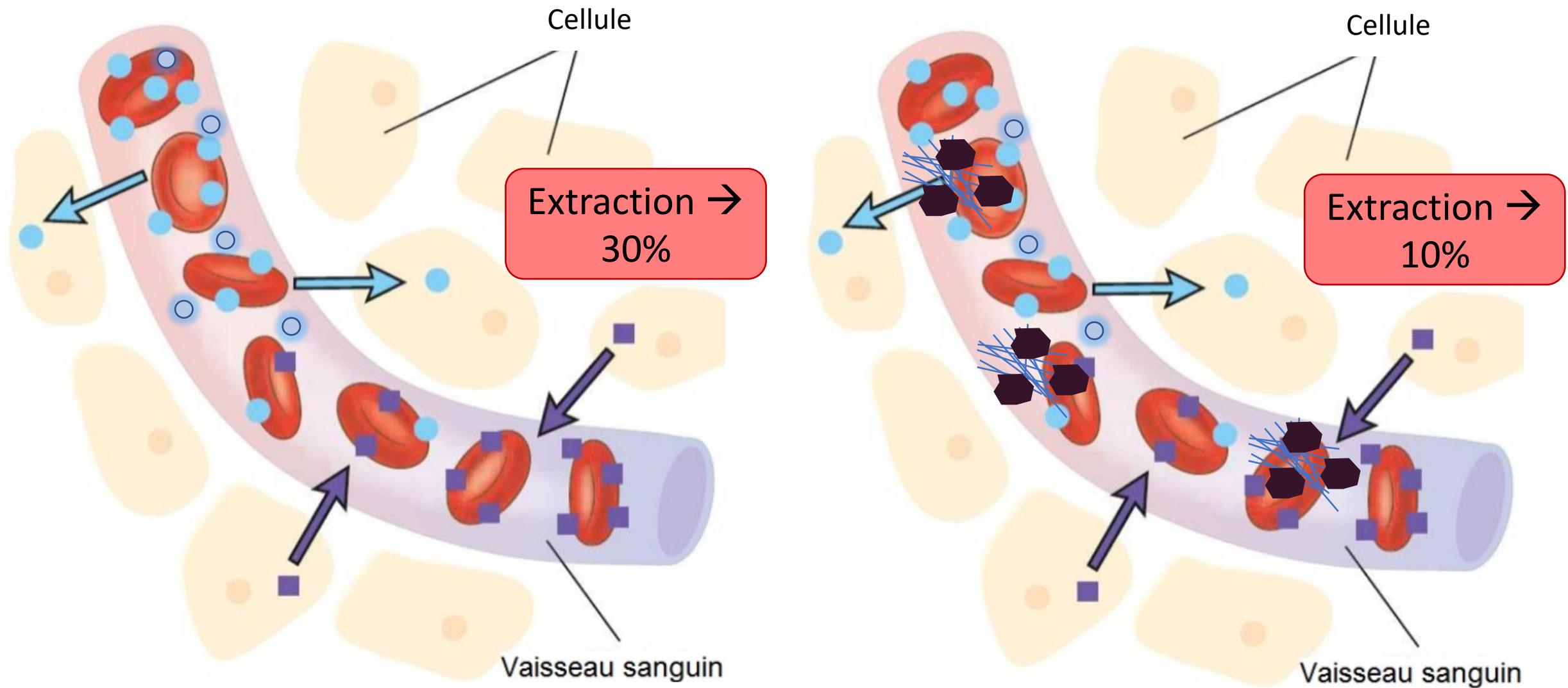
Vasodilatation

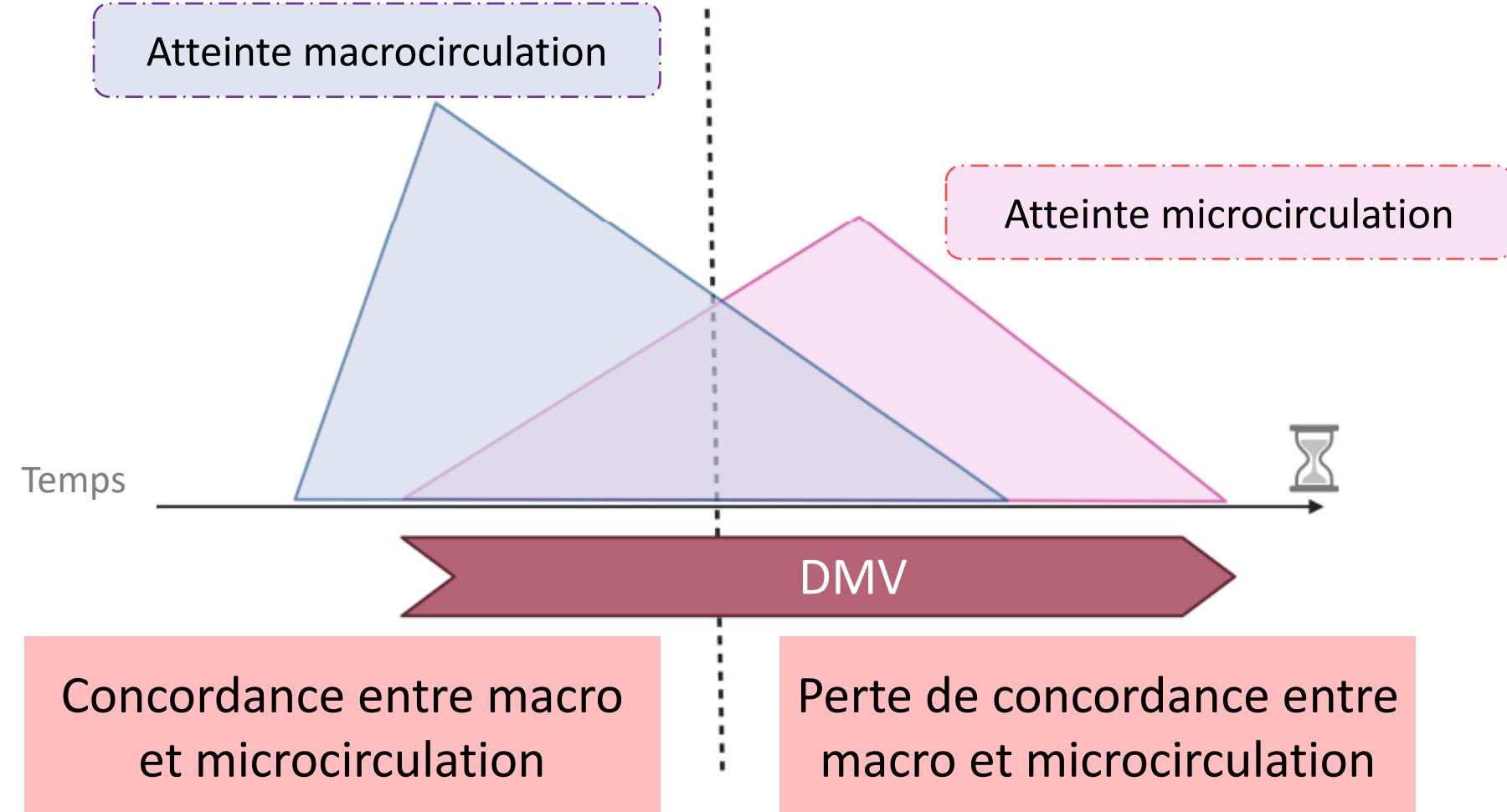
Recrutement capillaires fermés

↗ extraction en O₂



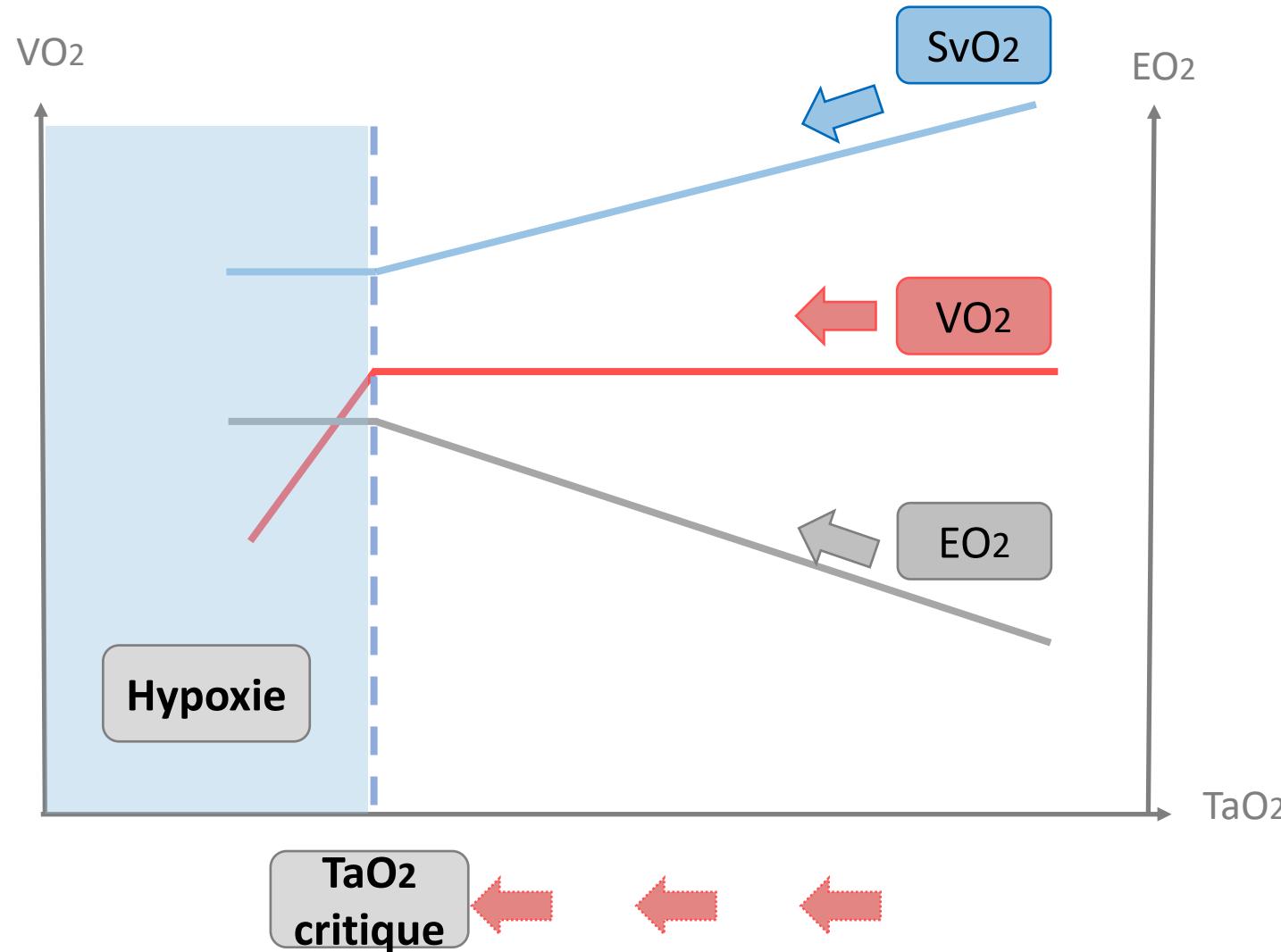
Mécanismes adaptatifs : le cas du choc septique





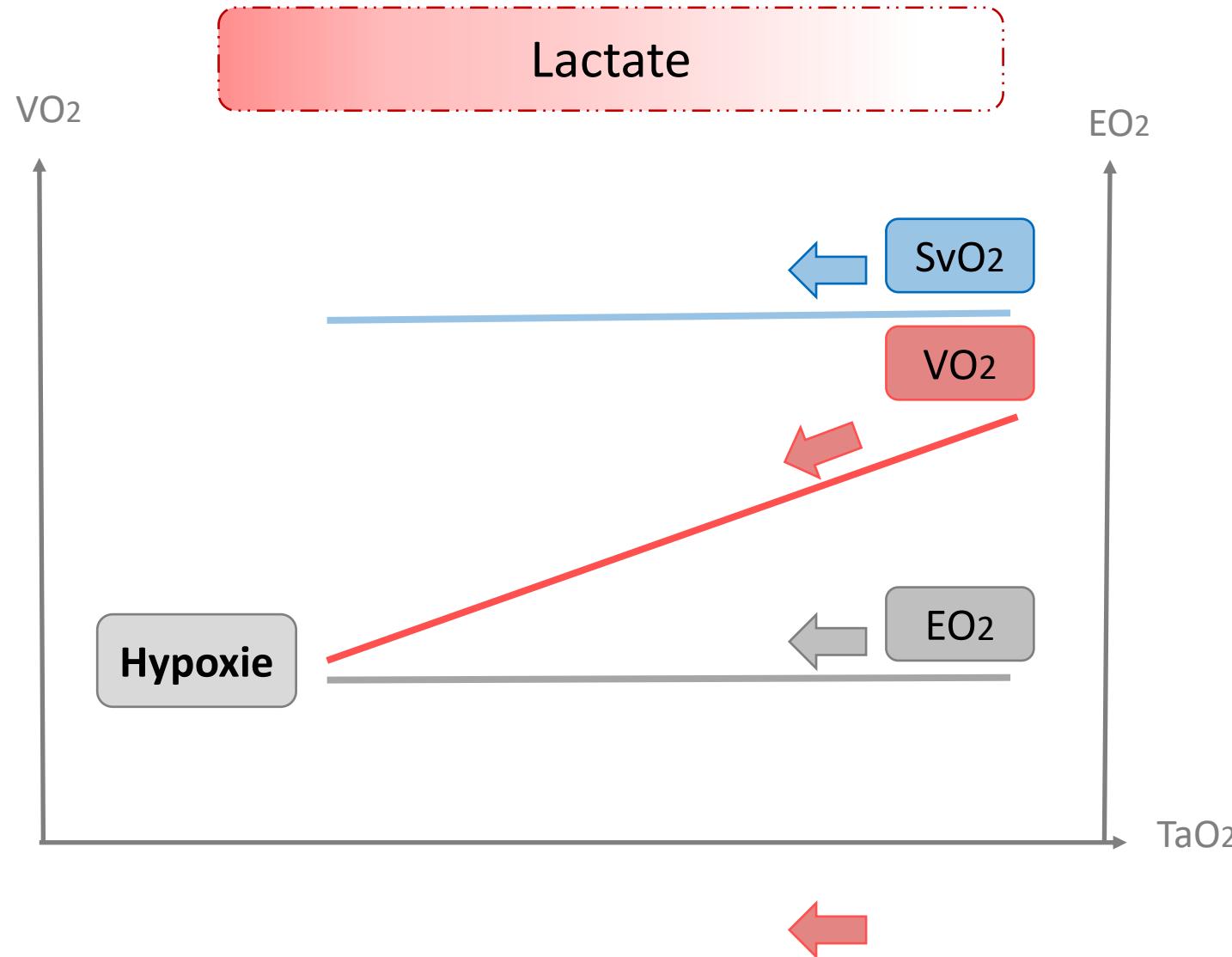
Mécanismes adaptatifs : le cas du choc septique

② $\uparrow EO_2$



Mécanismes adaptatifs : le cas du choc septique

② $\uparrow EO_2$



1

Etat de choc → inadéquation entre apports et besoins en O₂

2

4 grands types de choc caractérisés par une ↘ du TaO₂ (DC) et/ou ↘ de PA

3

4

1

Etat de choc → inadéquation entre apports et besoins en O₂

2

4 grands types de choc caractérisés par une ↘ du TaO₂ (DC) et/ou ↘ de PA

3

↗ EO₂ et l'activation système sympathique → mécanismes compensateurs



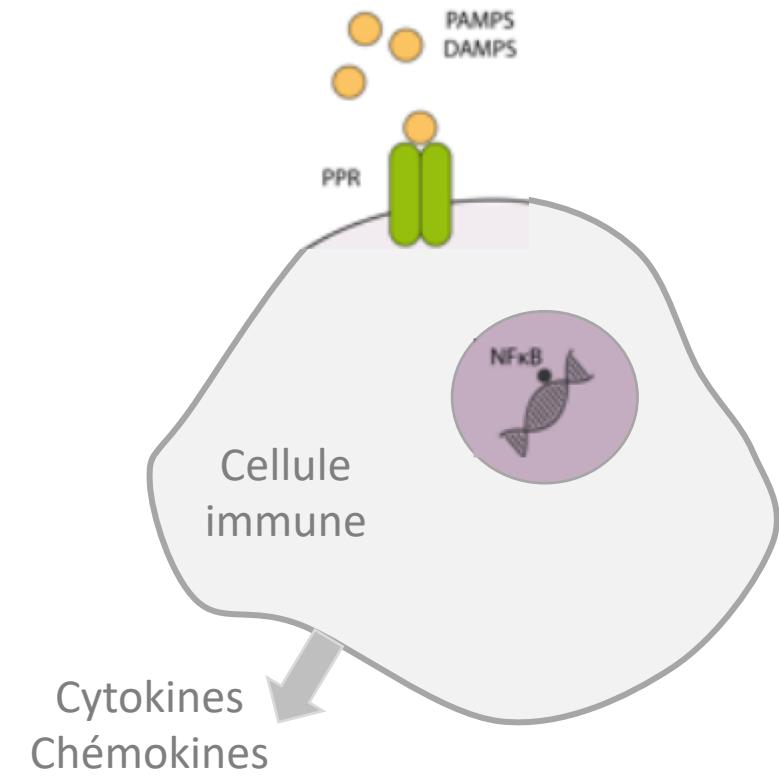
4

Impact des états de choc

Quel est l'impact de l'état de choc ?

- ↑ production cellules pro-inflammatoires
- ↑ production lactate
- Cascade de dysfonction d'organes
- œdème tissulaire

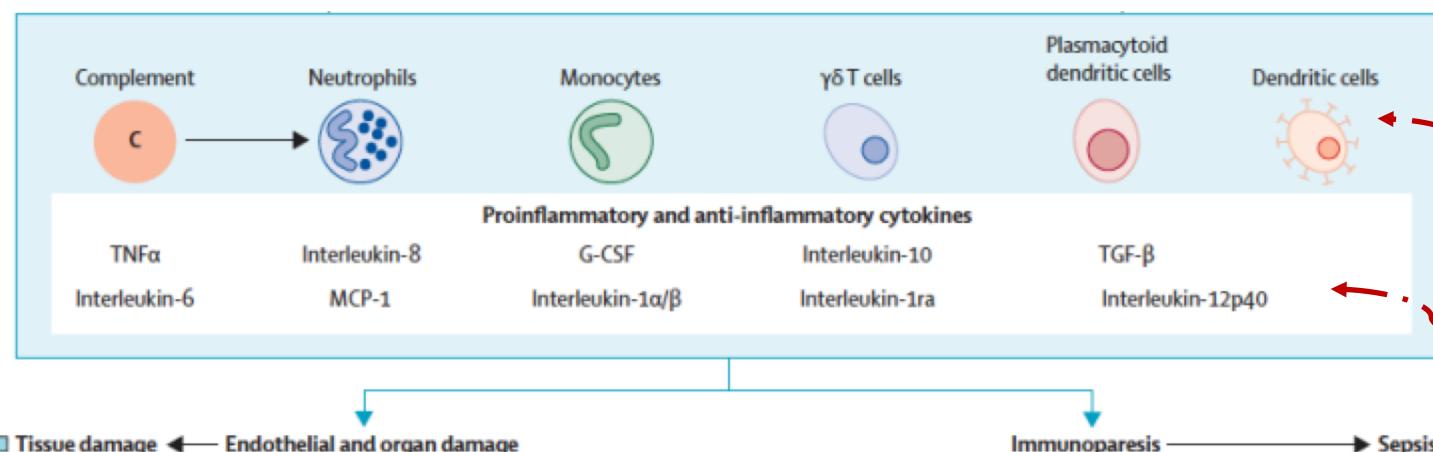
Production cellules



Cytokines
Chémokines

Activation
cytokines pro
inflammatoires

Dommage
tissulaire



↗ Inflammation

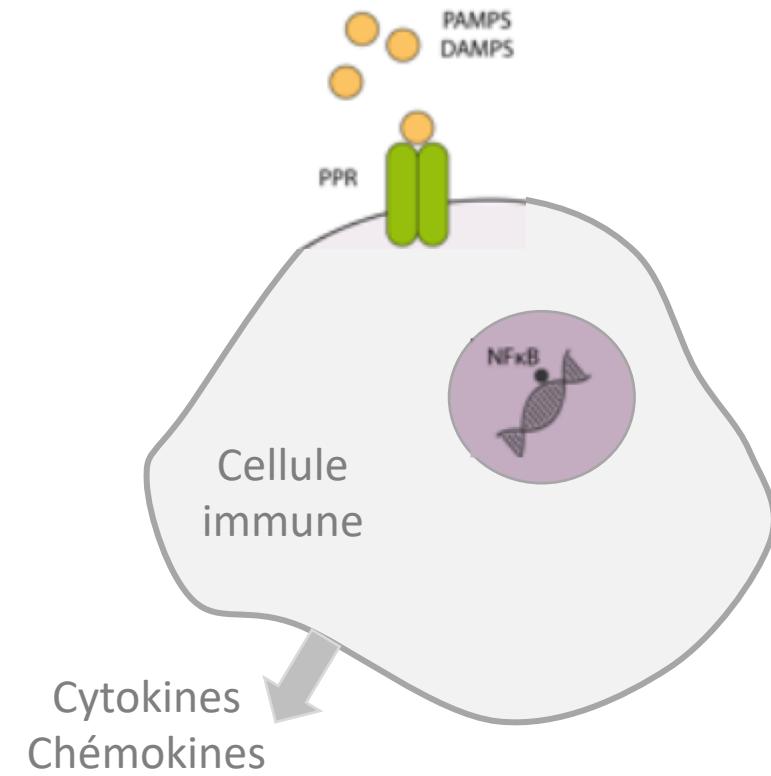
Atteinte
endothéliale

Immunoparesis → Sepsis

Tissue damage ← Endothelial and organ damage

Lord, Lancet 2014

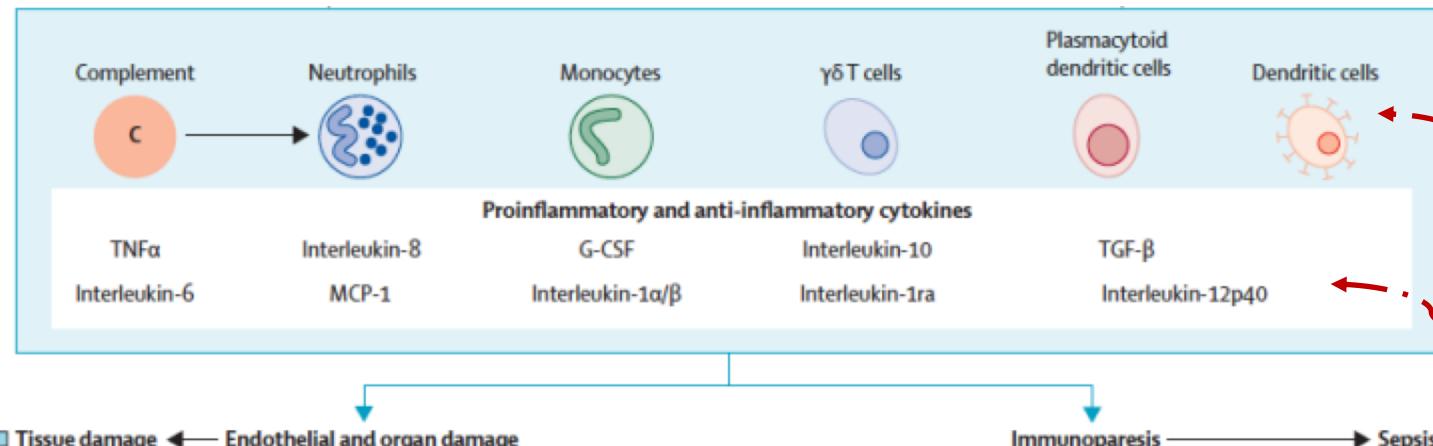
Production cellules



Cytokines
Chémokines

Activation
cytokines pro
inflammatoires

Dommage
tissulaire



↗ Inflammation

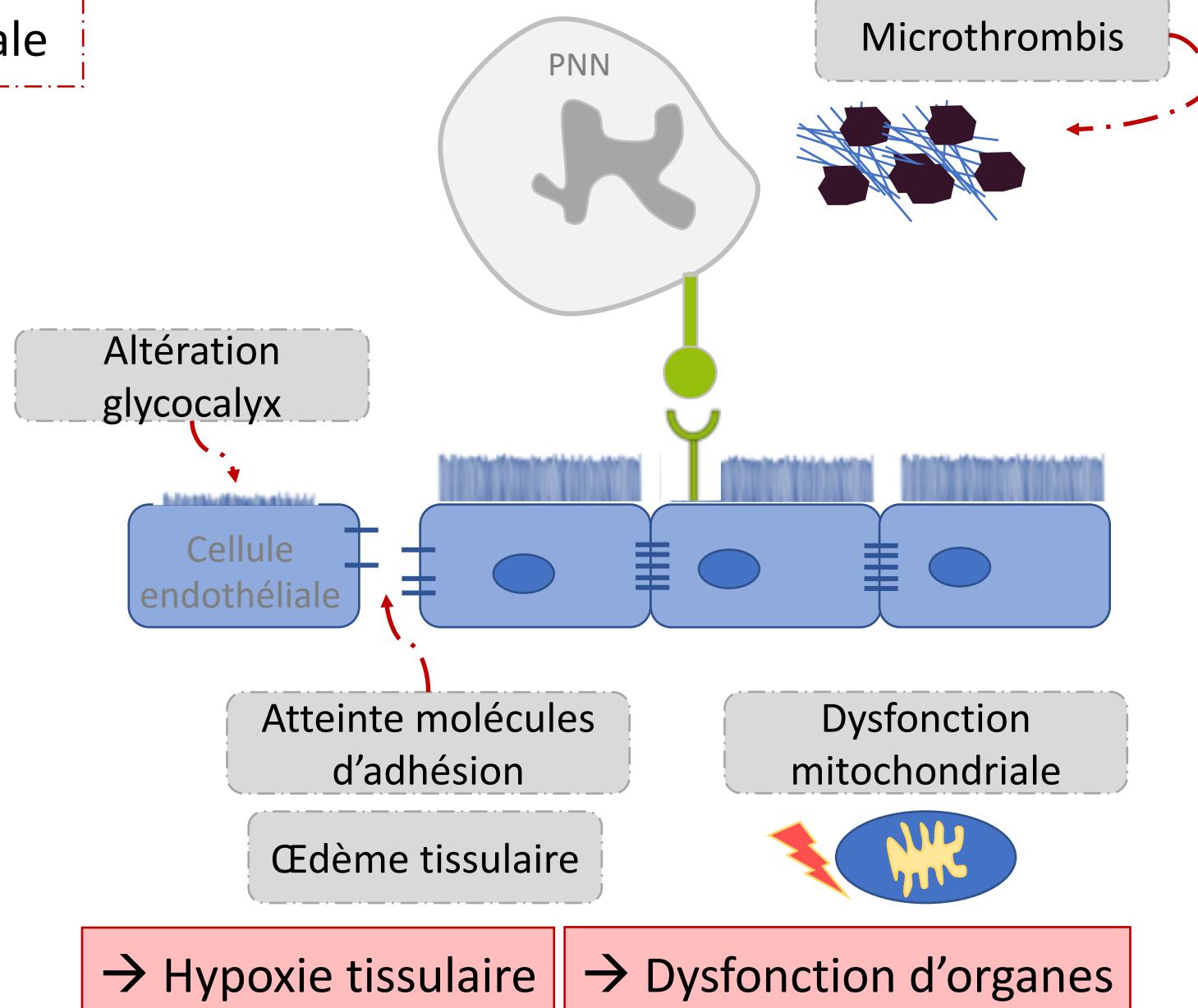
Atteinte
endothéliale

Immunoparesis → Sepsis

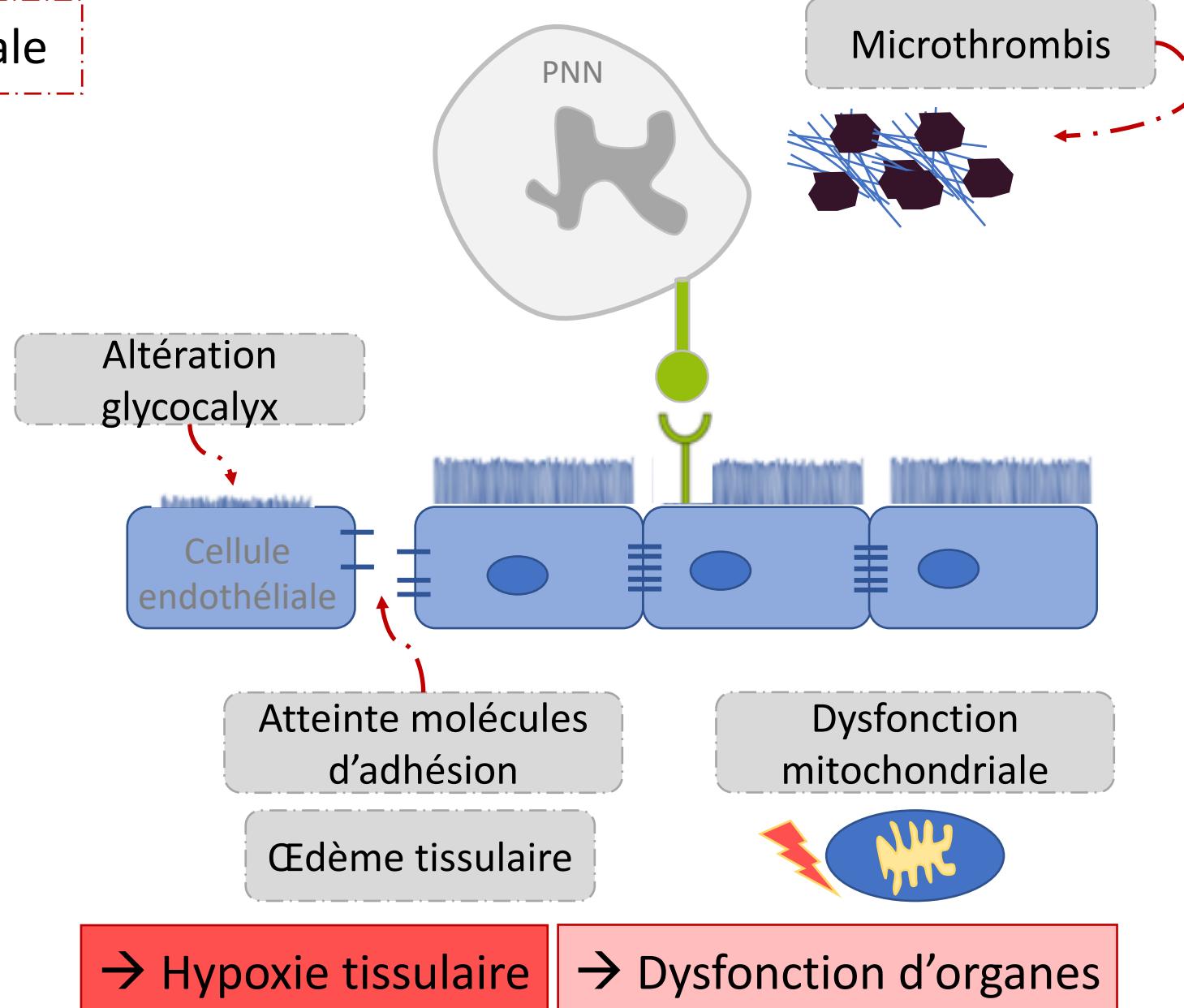
Tissue damage ← Endothelial and organ damage

Lord, Lancet 2014

Atteinte endothéliale



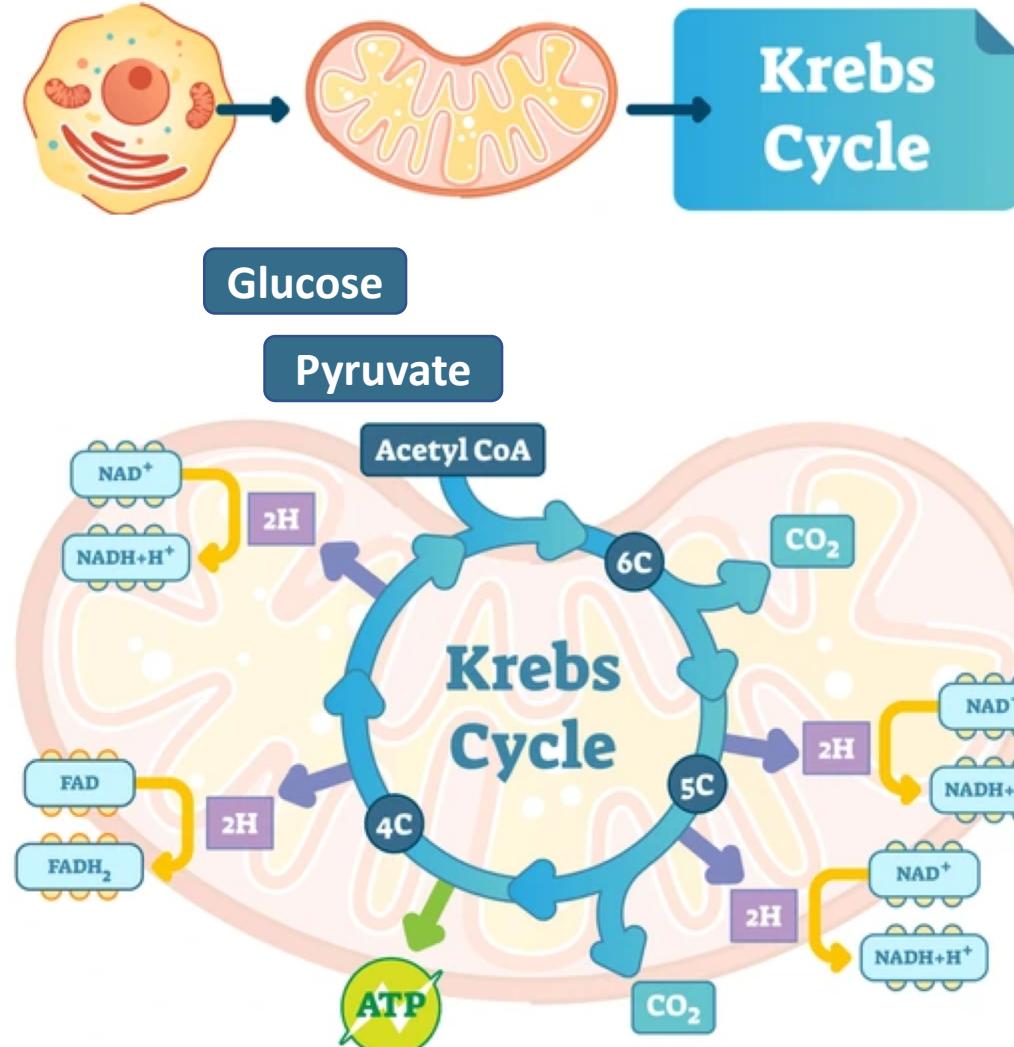
Atteinte endothéliale



Hypoxie tissulaire



Métabolisme
aérobie

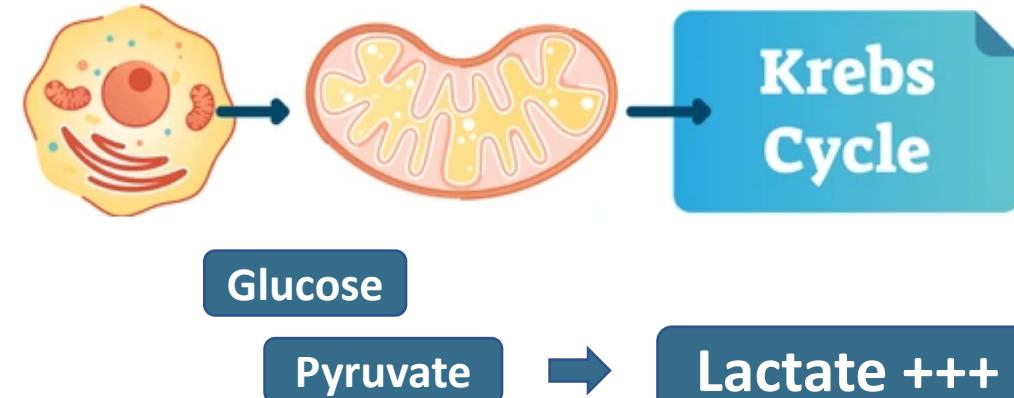


1 molécule de glucose
→ 38 molécules d'ATP

Hypoxie tissulaire



Métabolisme
anaérobie



Indicateur de
l'anaérobiose

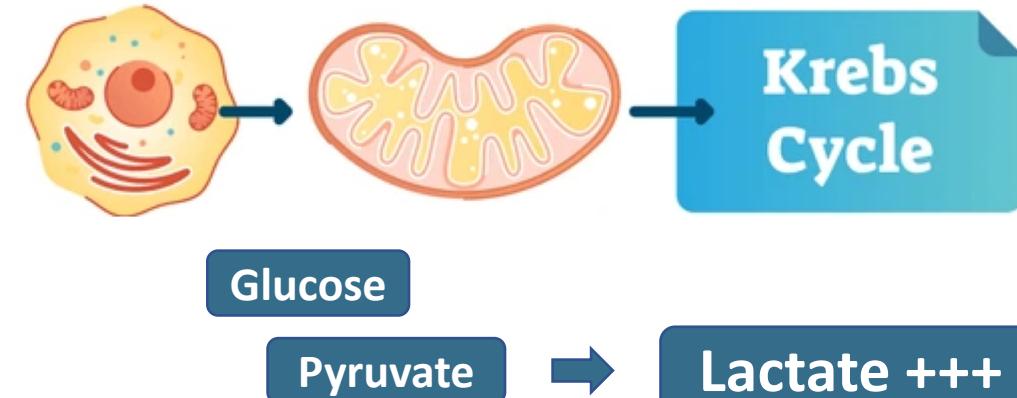
→ Hypoxie tissulaire

→ Dysfonction d'organes

Hypoxie tissulaire



Métabolisme
anaérobie

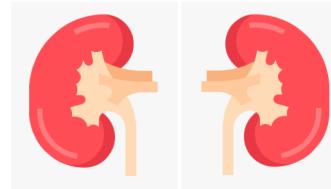
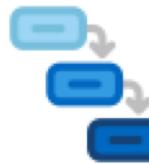


Indicateur de
l'anaérobiose

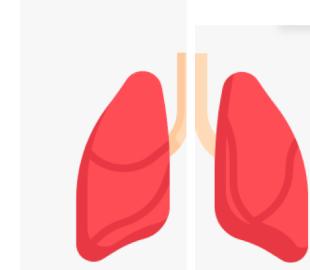
→ Hypoxie tissulaire

→ Dysfonction d'organes

Dysfonction d'organes



Insuffisance
rénale aiguë



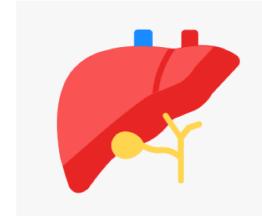
SDRA



Encéphalopathie



CIVD



Foie de choc

→ Défaillance multiviscérale

1

Etat de choc → inadéquation entre apports et besoins en O₂

2

4 grands types de choc caractérisés par une ↘ du TaO₂ (DC) et/ou ↘ de PA

3

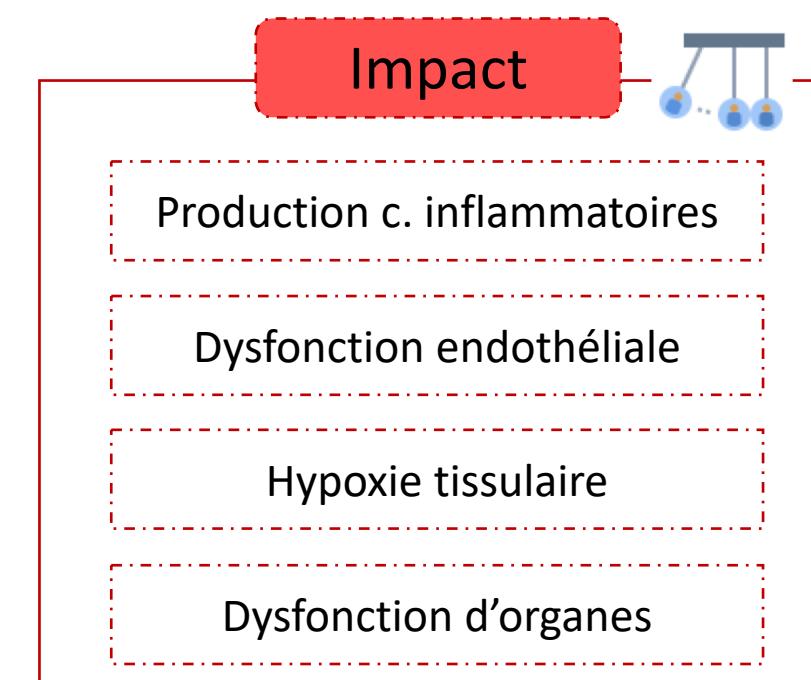
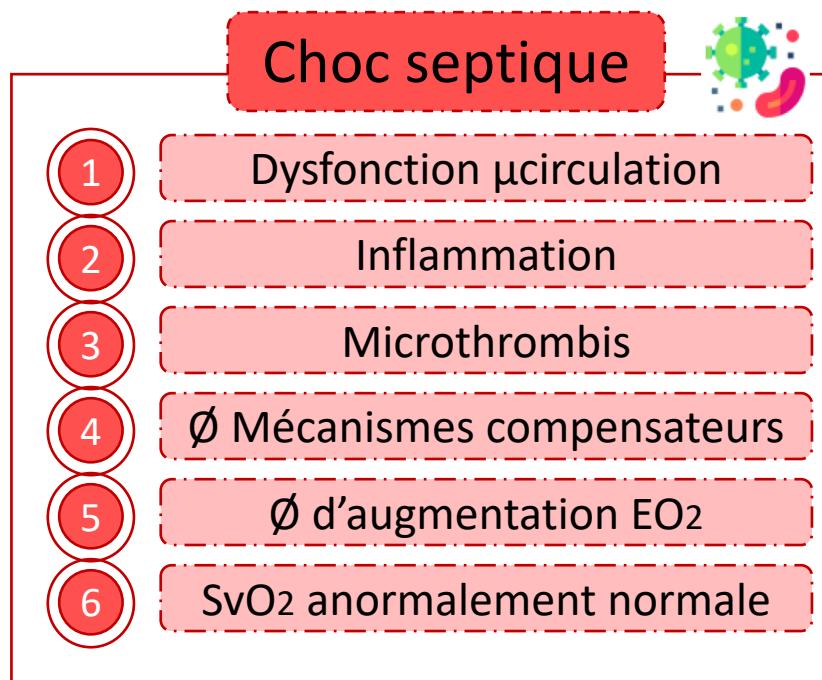
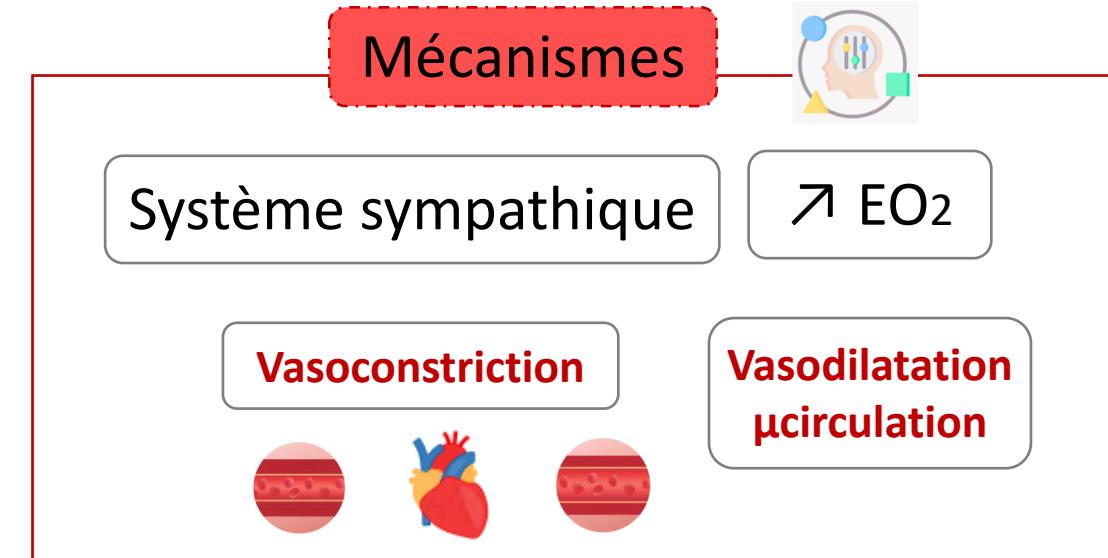
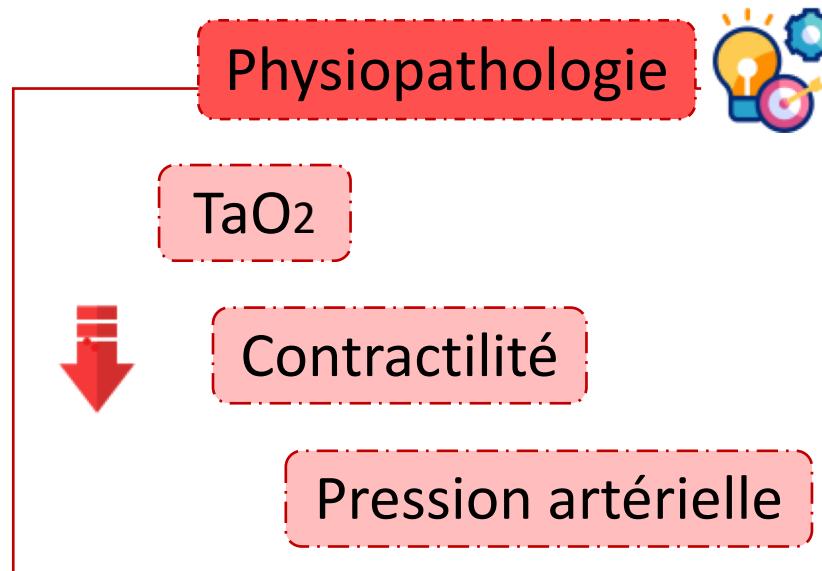
↗ EO₂ et l'activation système sympathique → mécanismes compensateurs



4

- 1 Etat de choc → inadéquation entre apports et besoins en O₂
- 2 4 grands types de choc caractérisés par une ↘ du TaO₂ (DC) et/ou ↘ de PA
- 3 ↗ EO₂ et l'activation système sympathique → mécanismes compensateurs 
- 4 Etat de choc → dysfonction endothéliale, hypoxie tissulaire et DMV

Conclusions



Merci pour votre attention