



Physiologie appliquée à la défaillance circulatoire

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ACTUALITÉS EN RÉANIMATION
Médecine Intensive, Surveillance Continue
et Urgences Graves

☒ Je déclare les liens d'intérêt suivants :

Aucun

Physiologie du système circulatoire

Physiopathologie de la défaillance circulatoire

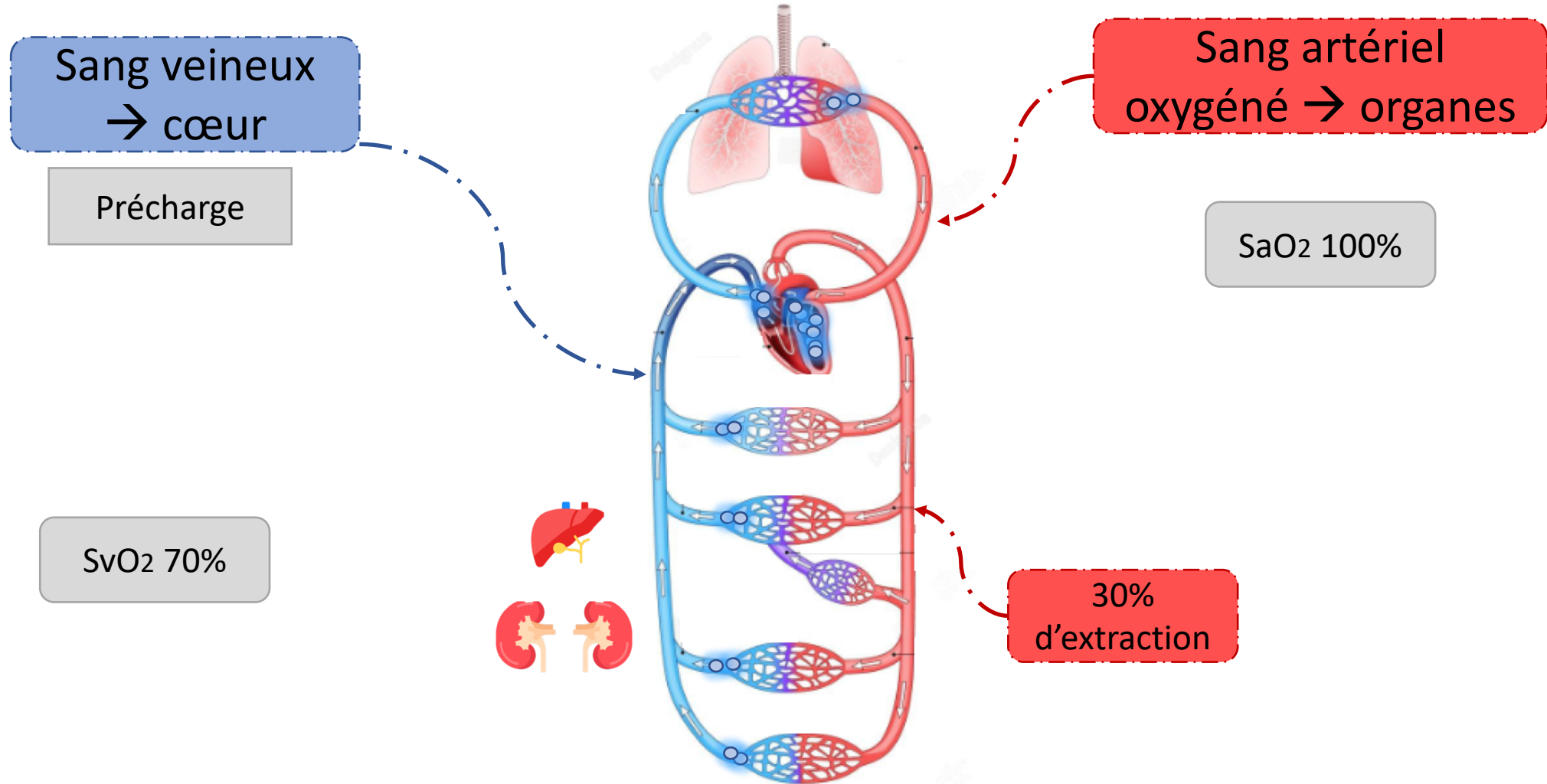
Mécanismes compensateurs

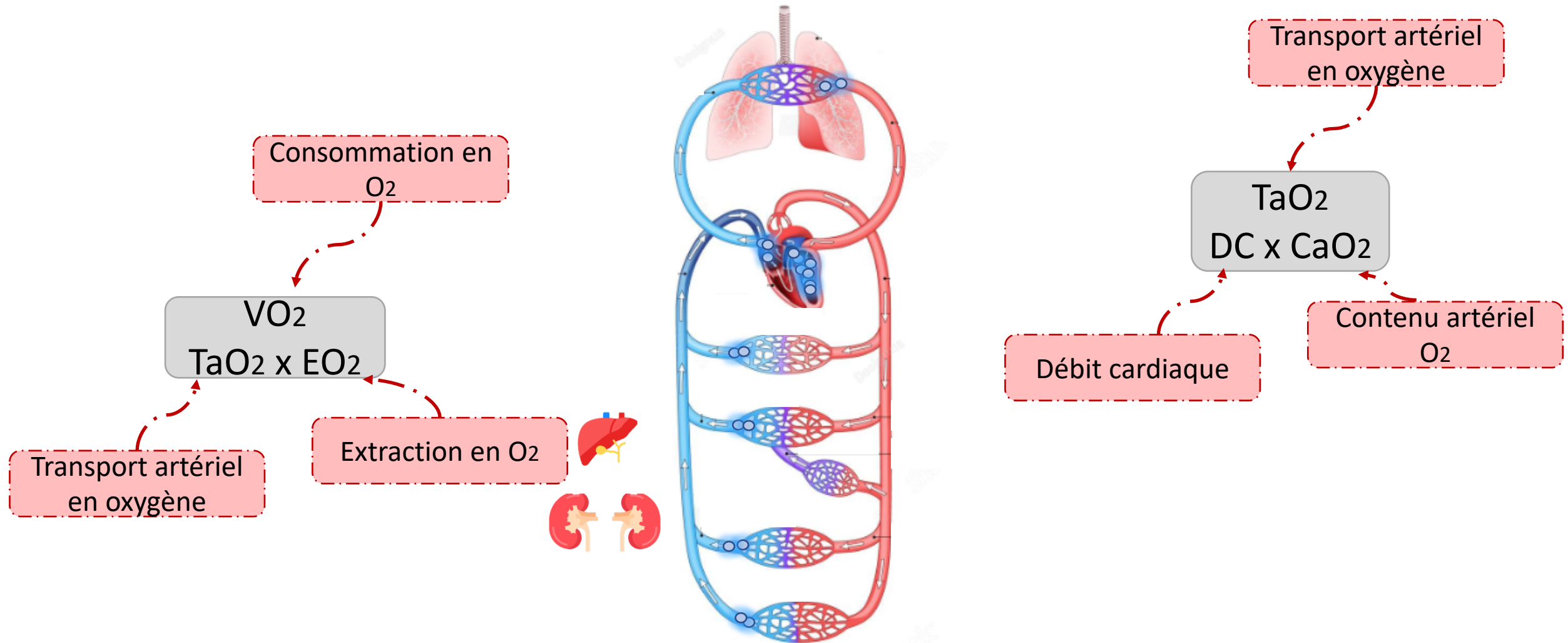
Le cas du choc septique

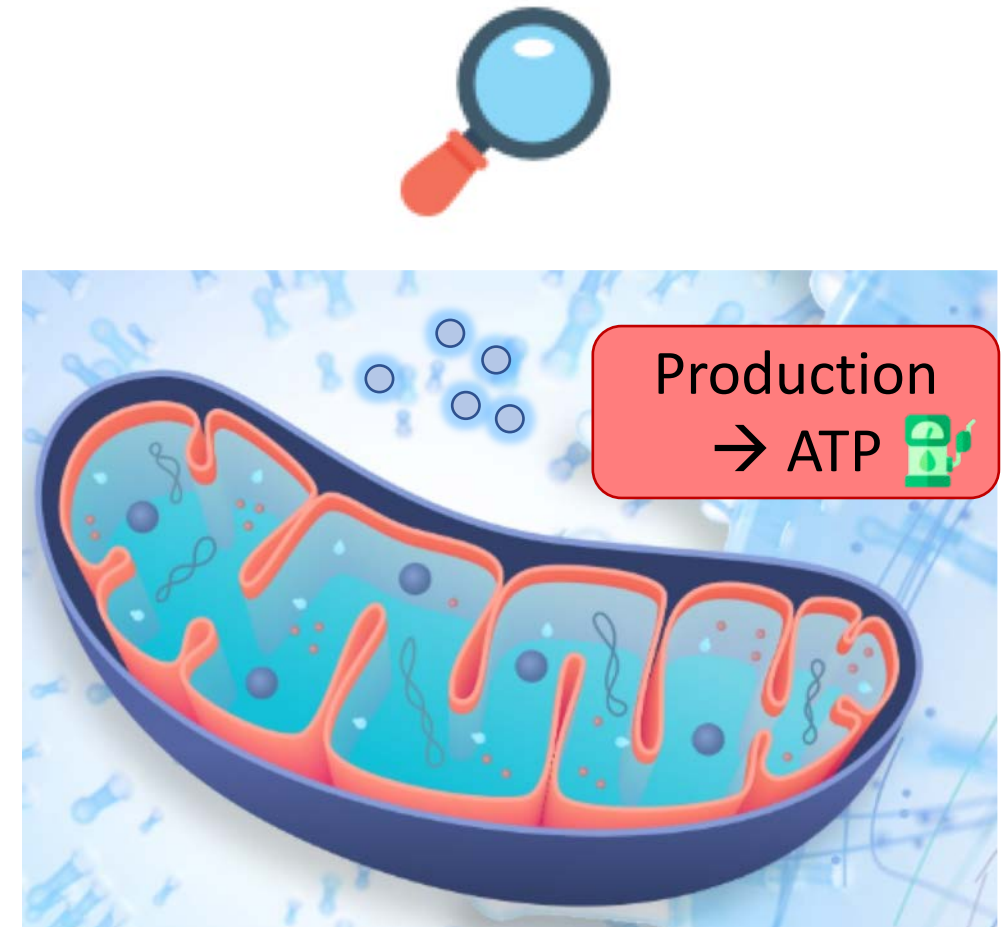
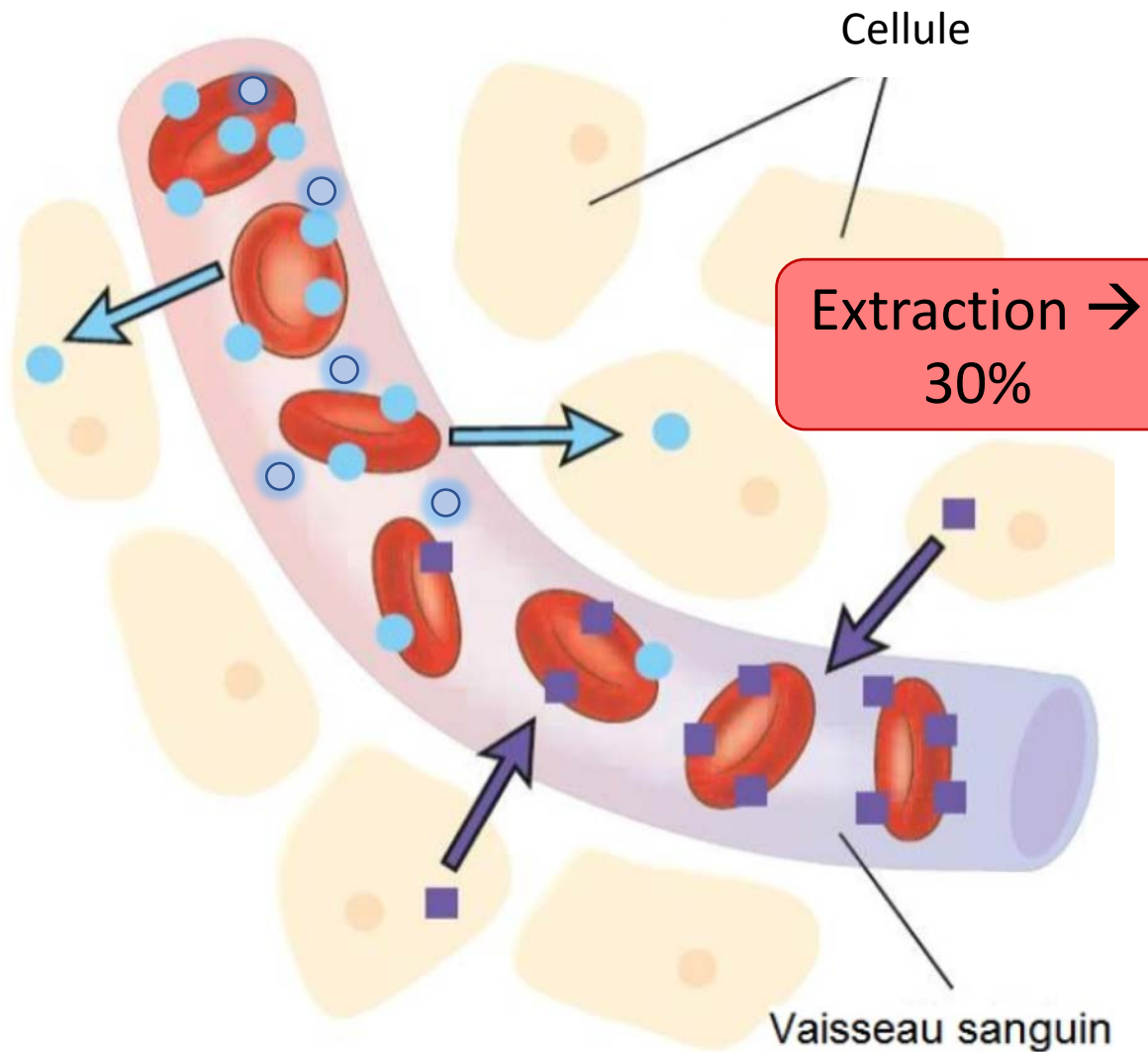
Impact des états de choc









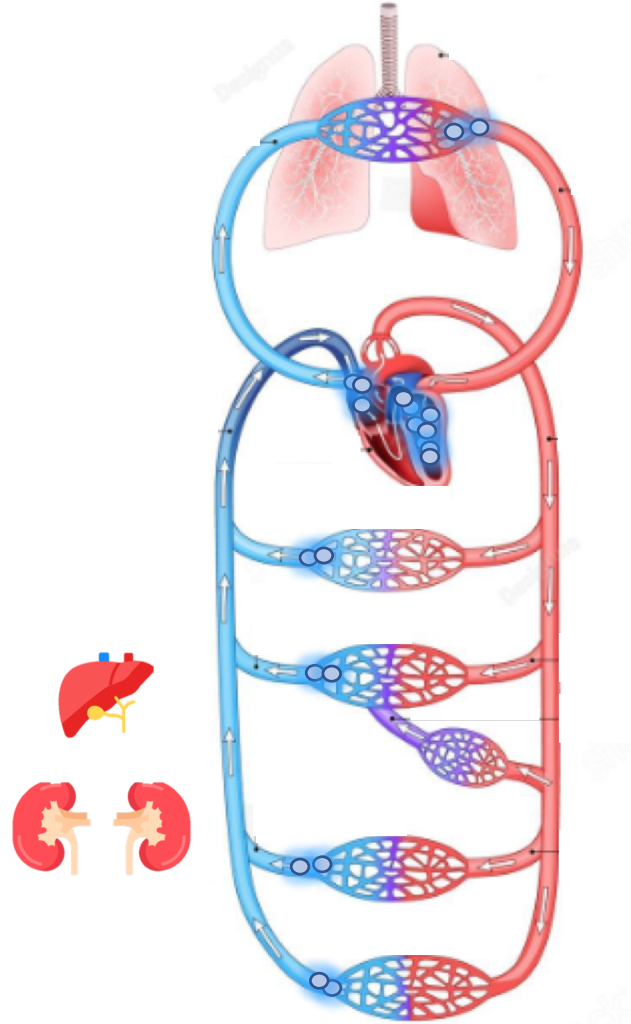




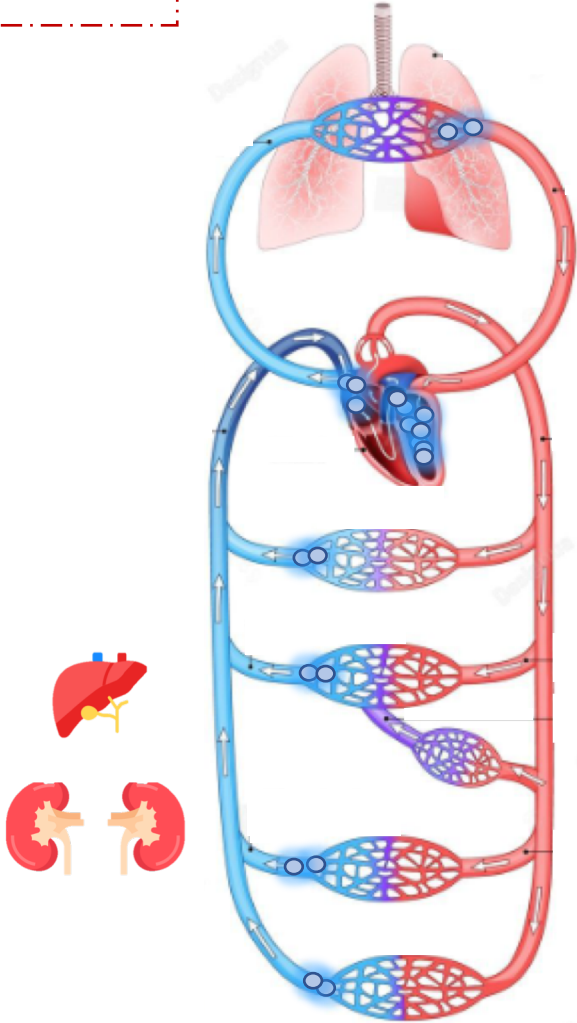
Quels sont les caractéristiques de la défaillance circulatoire ?

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

Etat de choc



Etat de choc



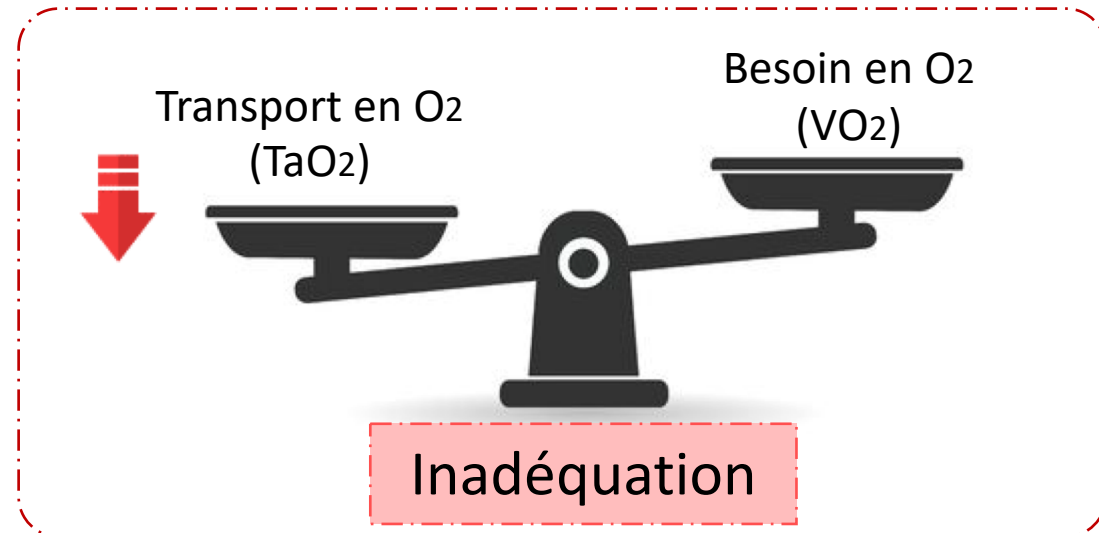
Phase ultime de défaillance circulatoire



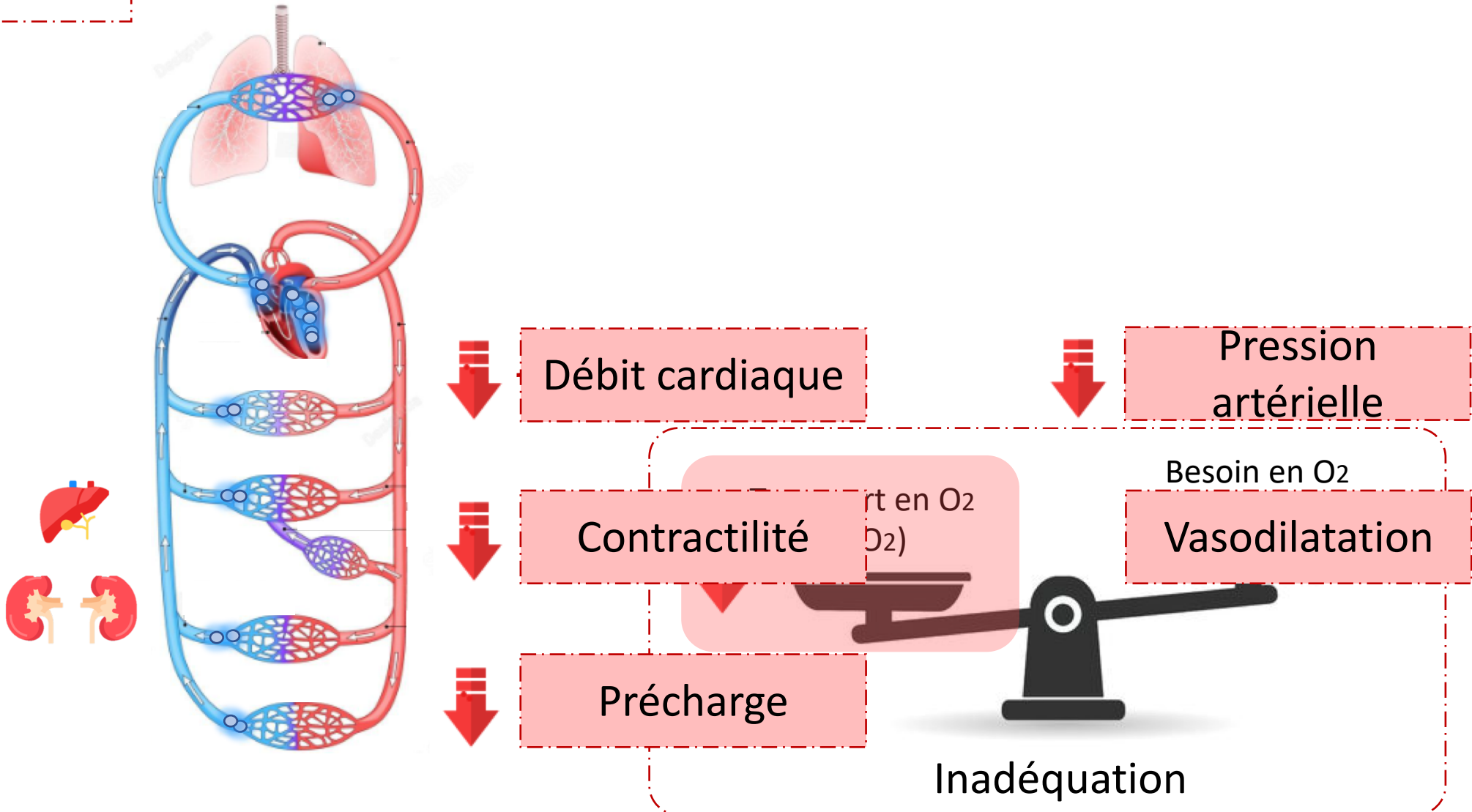
↘ de la perfusion des organes



Inadéquation entre apports et besoins en O₂

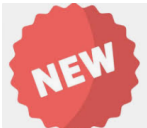


Etat de choc












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?				
	<p>→ 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.</p>			
2014	<p>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 (cardiogenic, obstructive) and one associated with a hyperkinetic state (distributive). Shock can be due to a combination of processes. [UNGRADED, STATEMENT OF FACT]</p>			
	<p>→ 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.</p>			
2014	<p>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]</p>			
	<p>→ Lactate levels are typically increased (>2 mmol/L) in shock states.</p>			
	<p>Lactate levels are typically >2 mmol/L (>2 mg/dL) in shock states. [UNGRADED, STATEMENT OF FACT]</p>			

Inadéquation entre besoin et apport en O₂

⋮ PA, ⋈ FC, signes hypoperfusion

⋈ lactate

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

Etat de choc



Hypovolémique

Cardiogénique

Distributif

Obstructif

Etat de choc



Hypovolémique

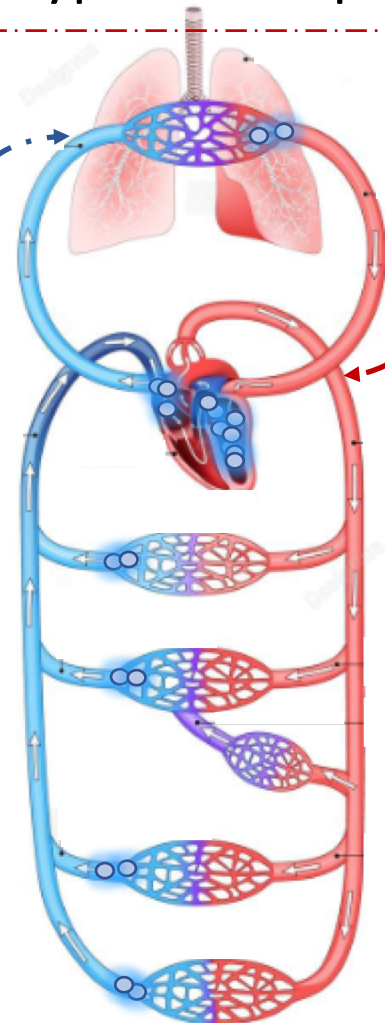
Cardiogénique

Distributif

Obstructif

Etat de choc hypovolémique

↘ volume sanguin



↘ débit cardiaque

↘ TaO_2
 $DC \times CaO_2$



Précharge



Perte liquide

Perte sang

Pertes digestives, cutanées...

Polytraumatisé, hémorragie dig



Etat de choc



Hypovolémique

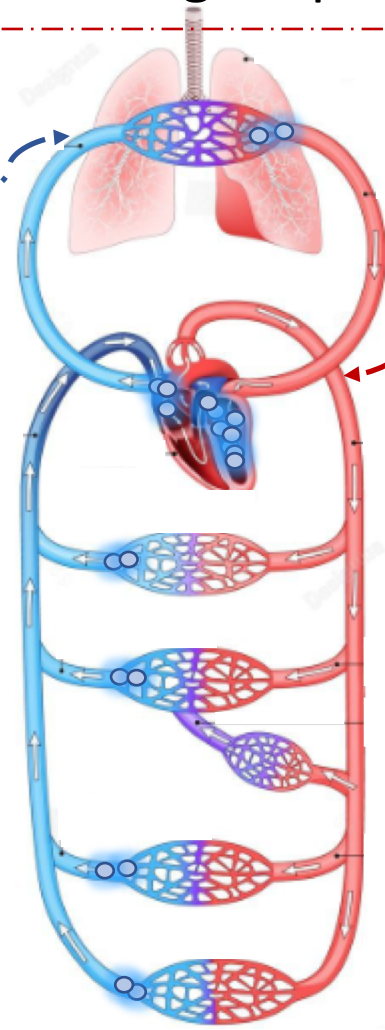
Cardiogénique

Distributif

Obstructif

Etat de choc cardiogénique

↘
contractilité



↘ débit
cardiaque

↘ TaO_2
 $DC \times CaO_2$



Contractilité



Infarctus

Myocardite

Lésions
ischémiques
coronariennes

Inflammation
du tissu
myocardique

Etat de choc



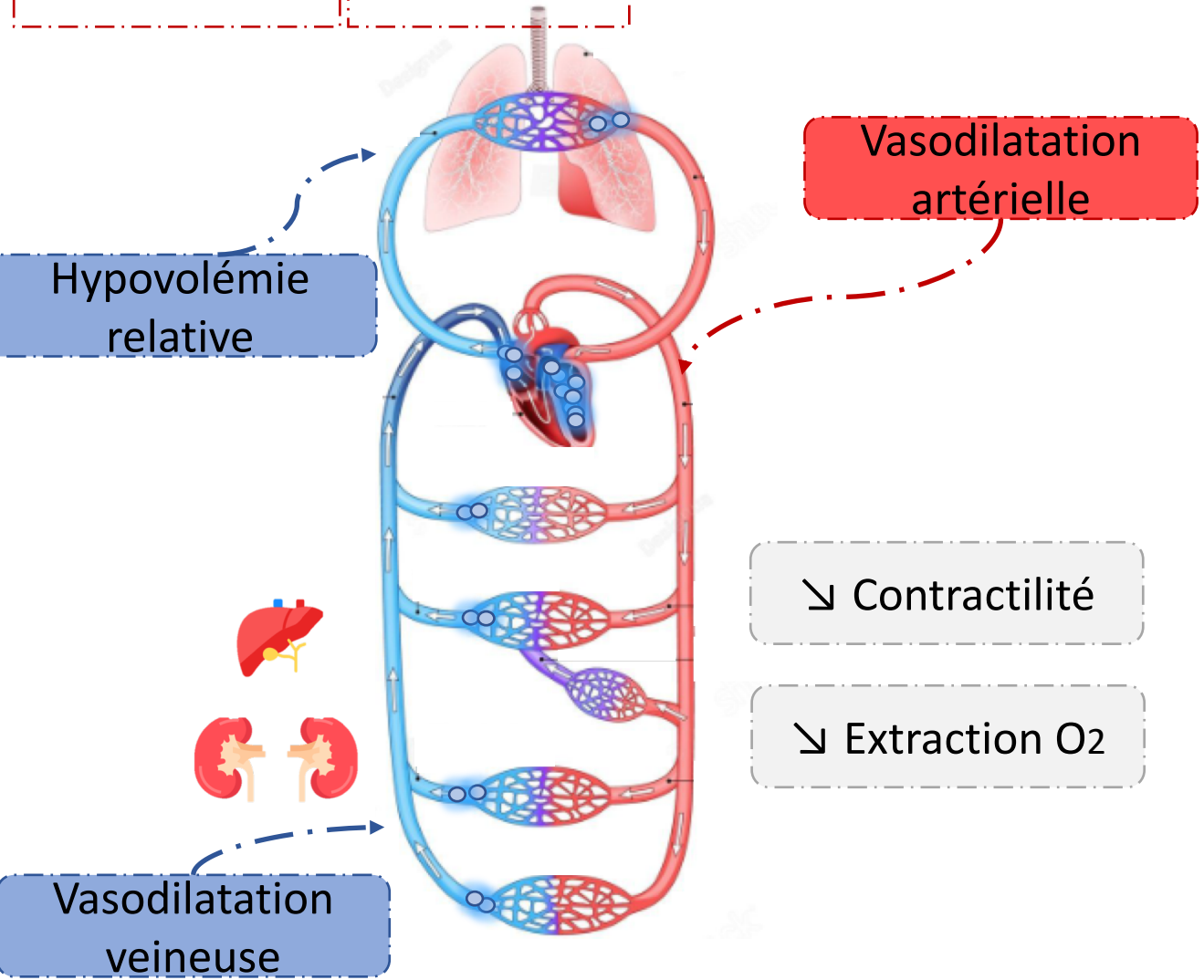
Hypovolémique

Cardiogénique

Distributif

Obstructif

Etat de choc distributif



Pression artérielle



Choc septique

Choc anaphylactique

Pneumonie,
pyélonéphrite

Allergie
médicaments

Etat de choc



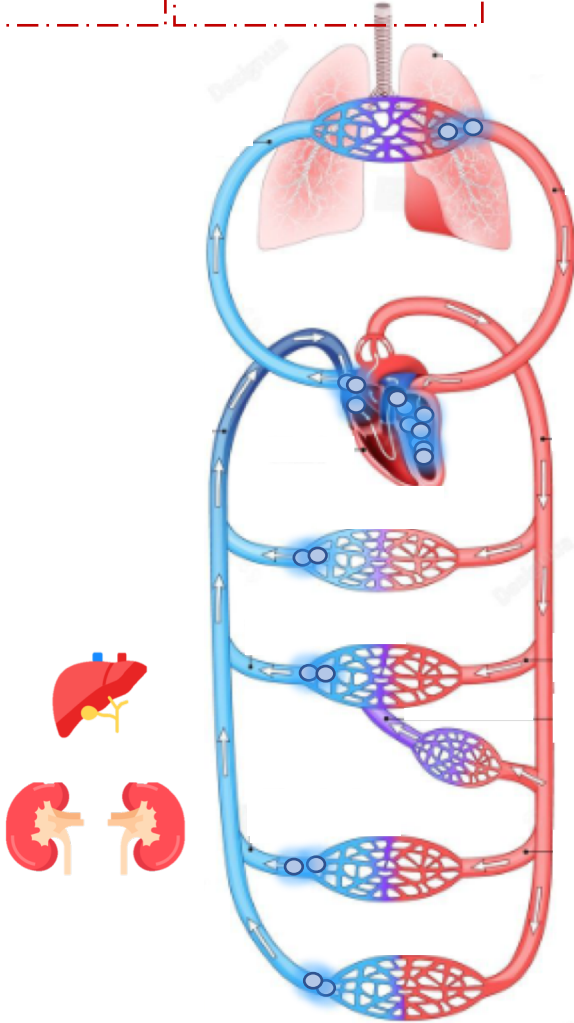
Hypovolémique

Cardiogénique

Distributif

Obstructif

Etat de choc obstructif



Obstacle
éjection VD



Choc obstructif

EP

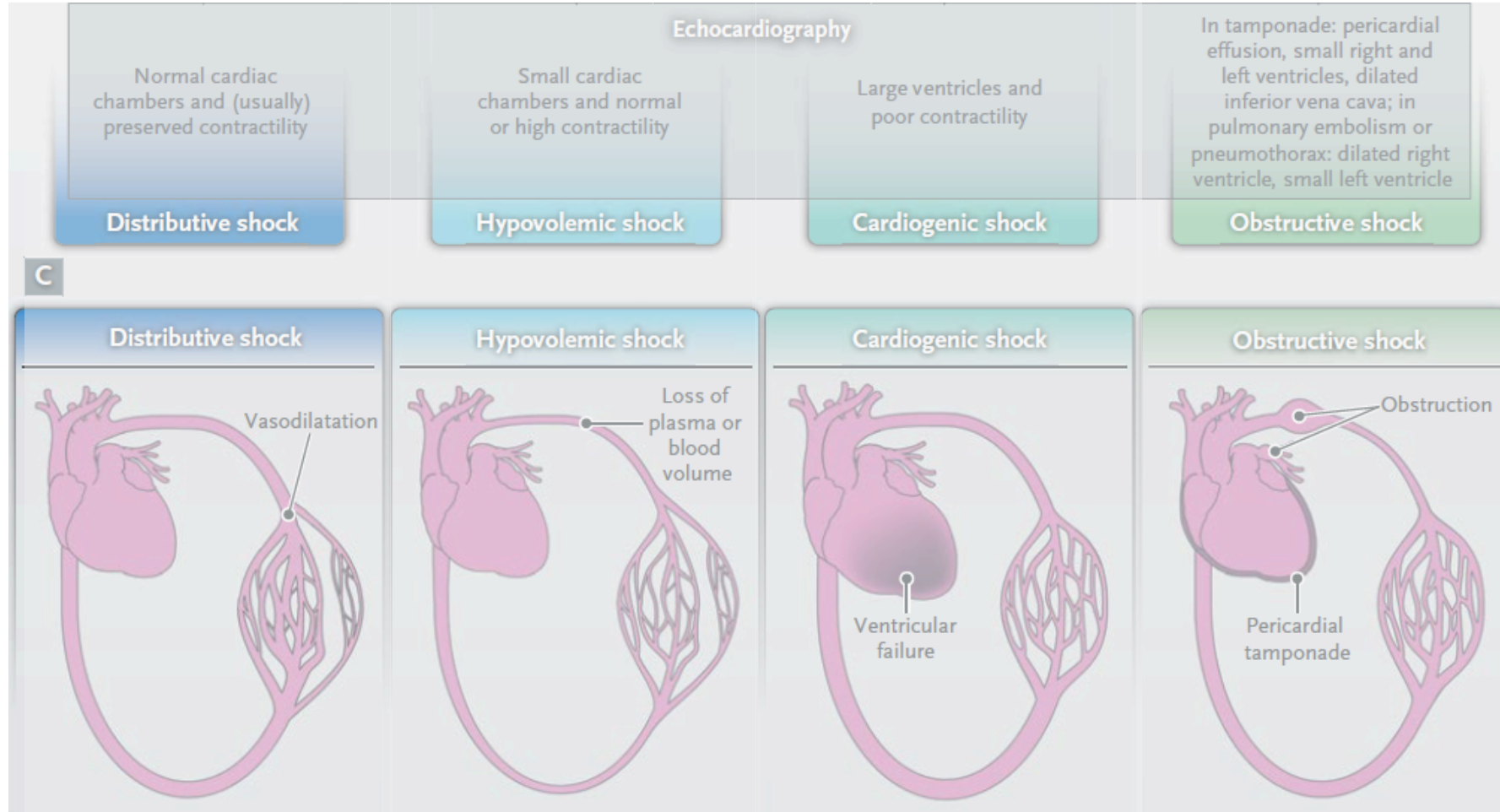
Tamponnade

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 \searrow du TaO₂ (DC) et/ou \searrow de PA

3

4



Quels sont les mécanismes adaptatifs ?

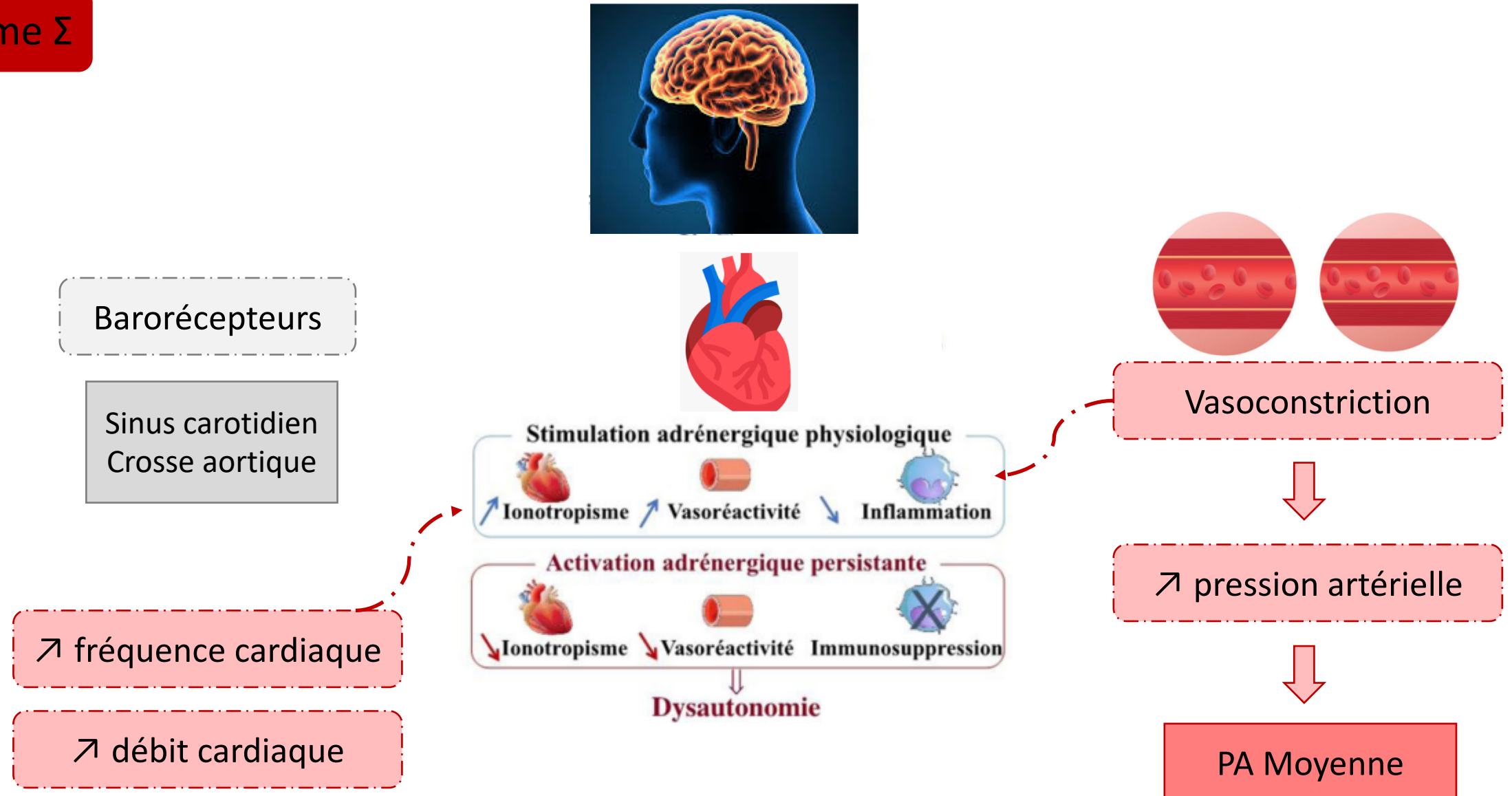
- ☐ ↑ l'extraction en oxygène
- ☐ ↓ l'inotropisme
- ☐ ↑ l'inflammation
- ☐ Vasodilatation la microcirculation
- ☐ Vasoconstriction artérielle et veineuse



1

Système Σ

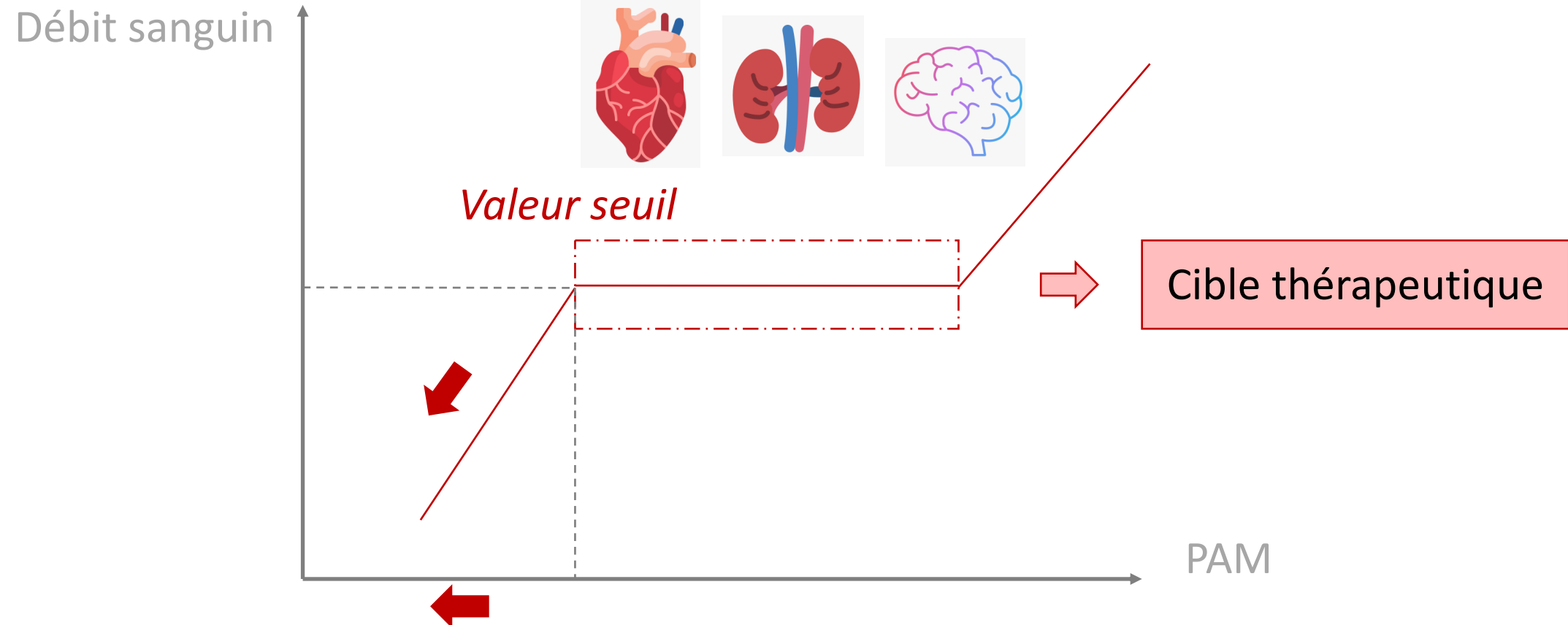
1 Système Σ



① Système Σ

↗ pression artérielle

Marqueur de perfusion des organes

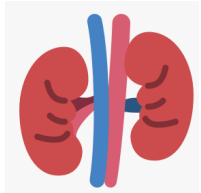


① Système Σ

↗ pression artérielle

Marqueur de perfusion des organes

PVC



Pression de perfusion
moyenne = PAM - PVC

En cas de PVC basse →
 $PPM = PAM - \cancel{PVC}$

PIA

PAD

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

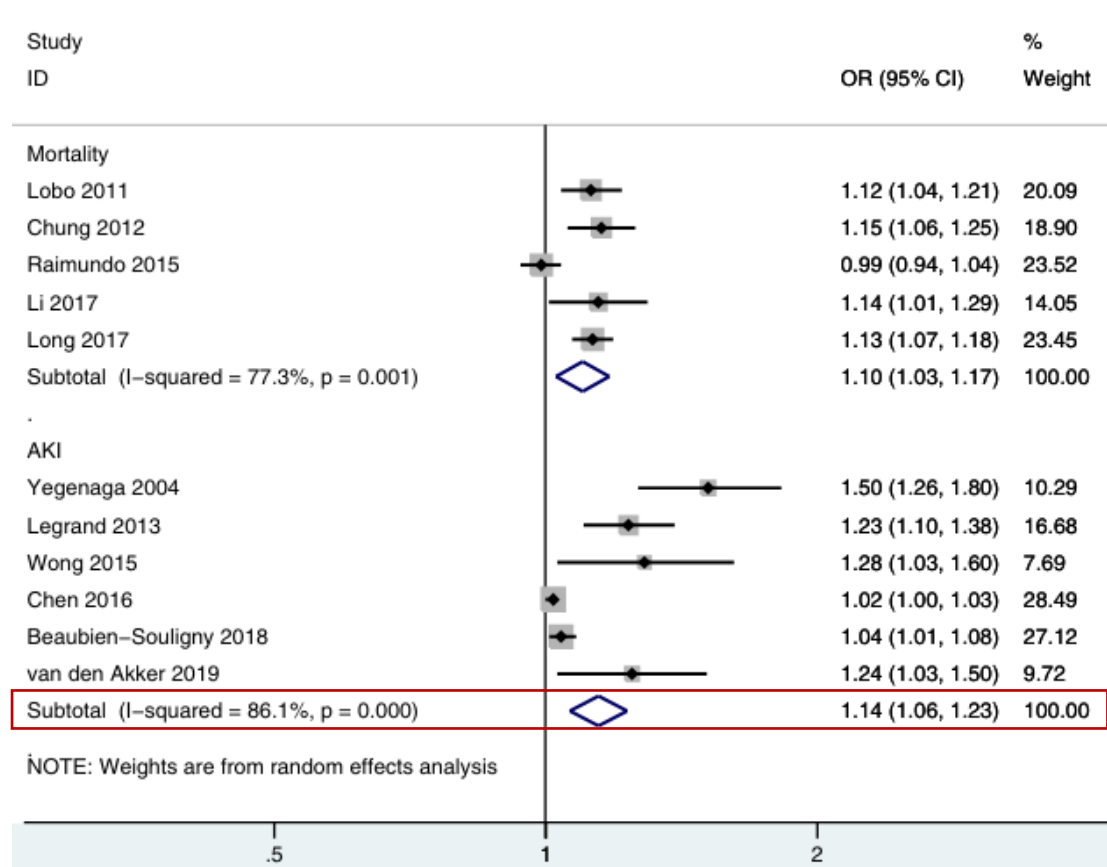
↗ pression artérielle

Marqueur de perfusion des organes

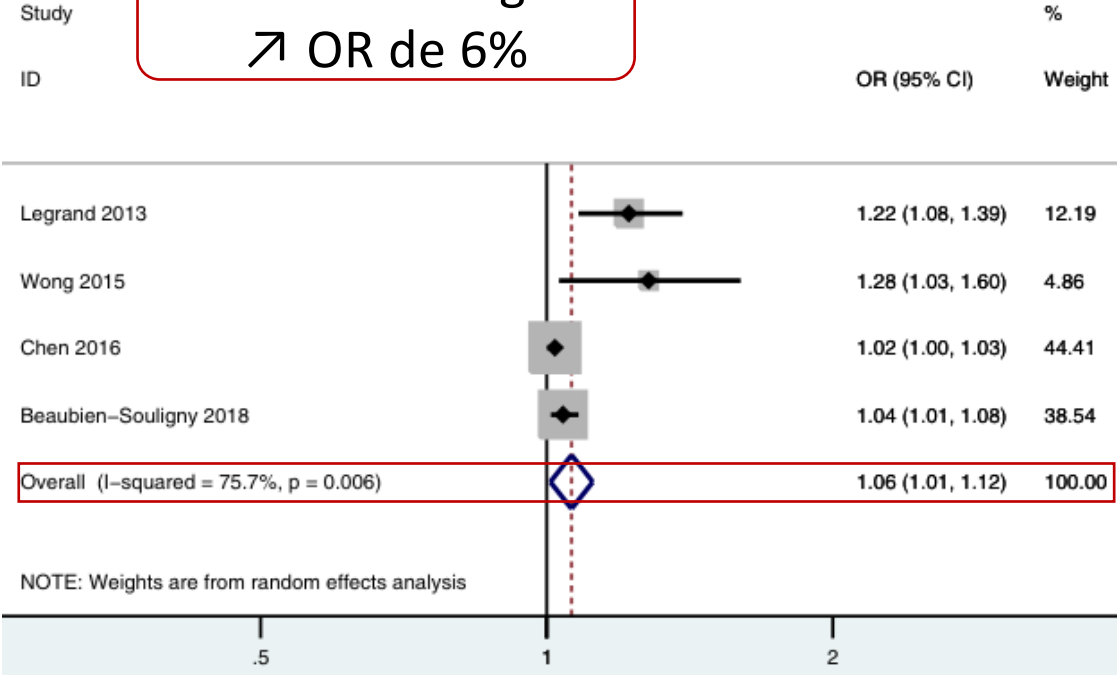
Méta-analyses
15 cohortes n=2,118

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 1mmHg →
↗ OR de 6%



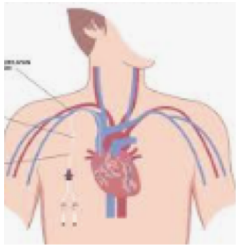
PVC élevée → mortalité ↗

① Système Σ

↗ pression artérielle

Marqueur de perfusion des organes

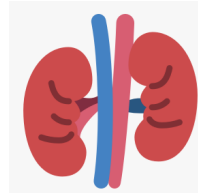
PVC



Pression de perfusion
moyenne = PAM - PVC

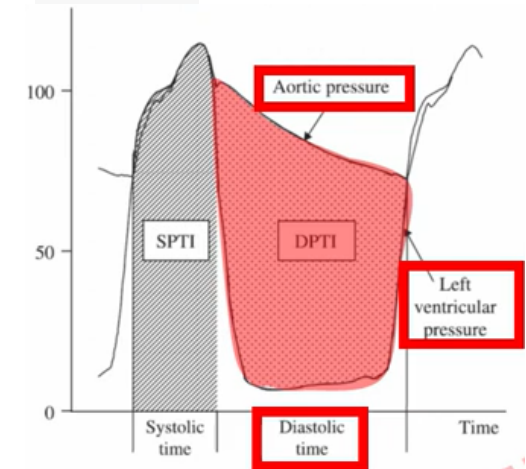
En cas de PVC basse →
 $PPM = PAM - \cancel{PVC}$

PIA



Influence de la PIA ou
encore PIC

PAD



Perfusion des organes dépend aussi d'autres déterminants hémodynamiques

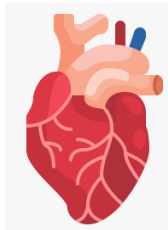
① Système Σ

↗ pression artérielle

Marqueur de perfusion des organes

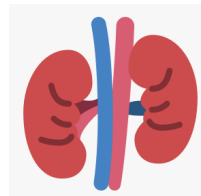
Dysfonction d'organes

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



< 65mmHg

↗ troponine



< 70mmHg

↗ créat ou EER



< 65mmHg

↗ délirium

Seuil optimal variable

Mortalité

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

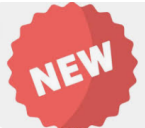
1

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



Individualisation

WHAT IS THE TARGET OF ARTERIAL PRESSURE IN SHOCK?			
→ The target blood pressure should be individualized during resuscitation of patients with shock.		👍	🔍
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.		👍	
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
65-70mmHg

↗ PAm si
PVC élevée

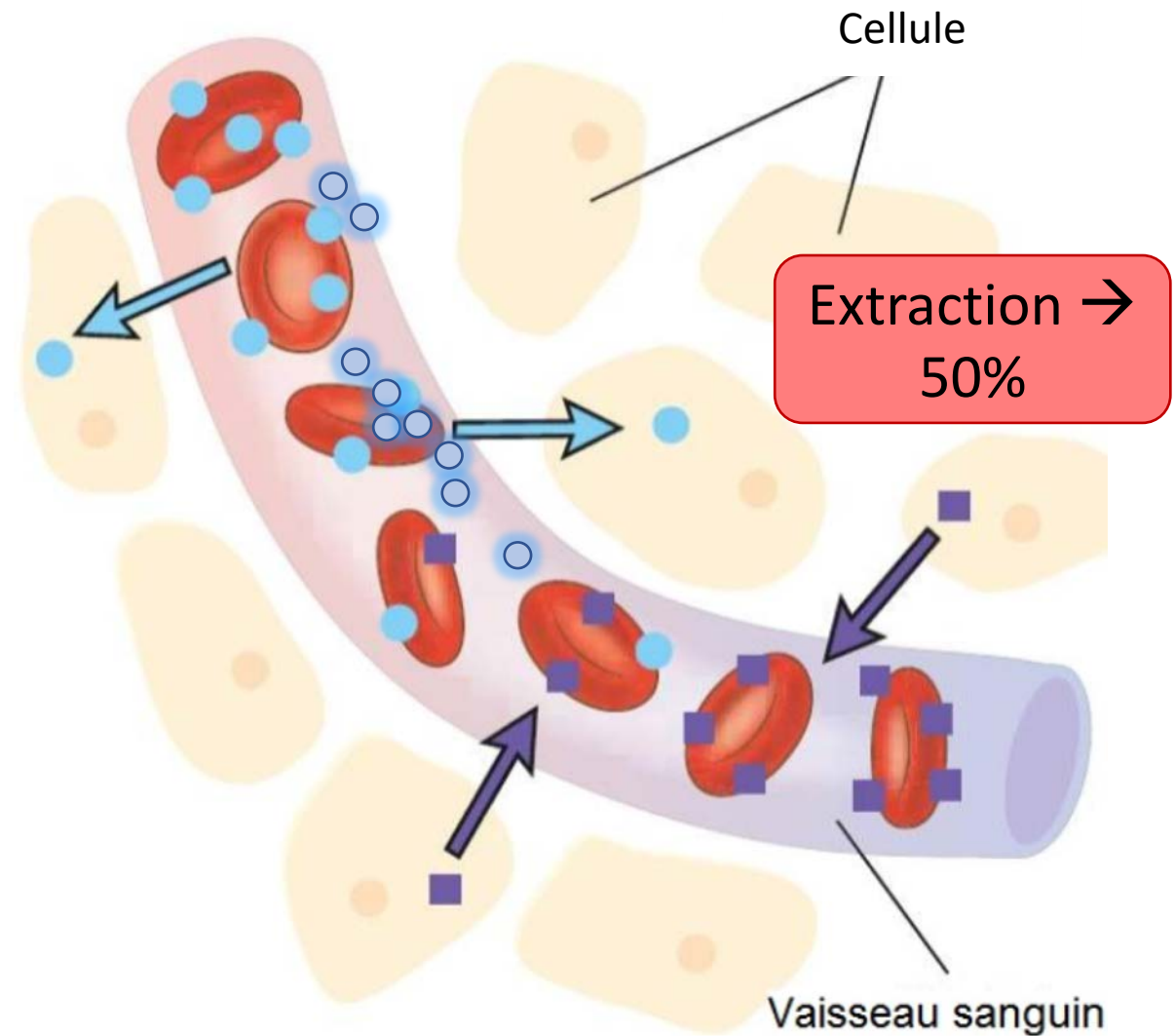
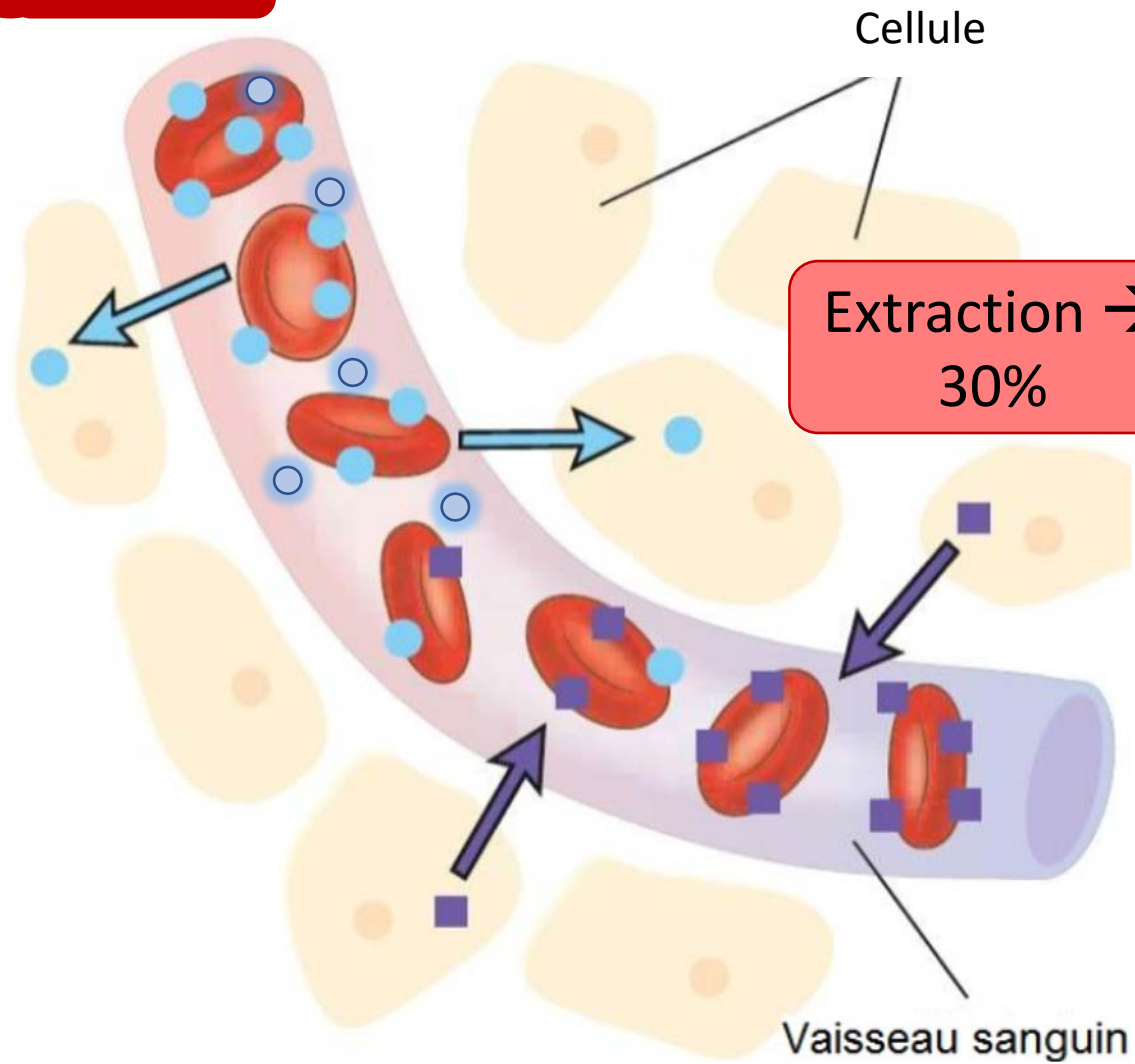




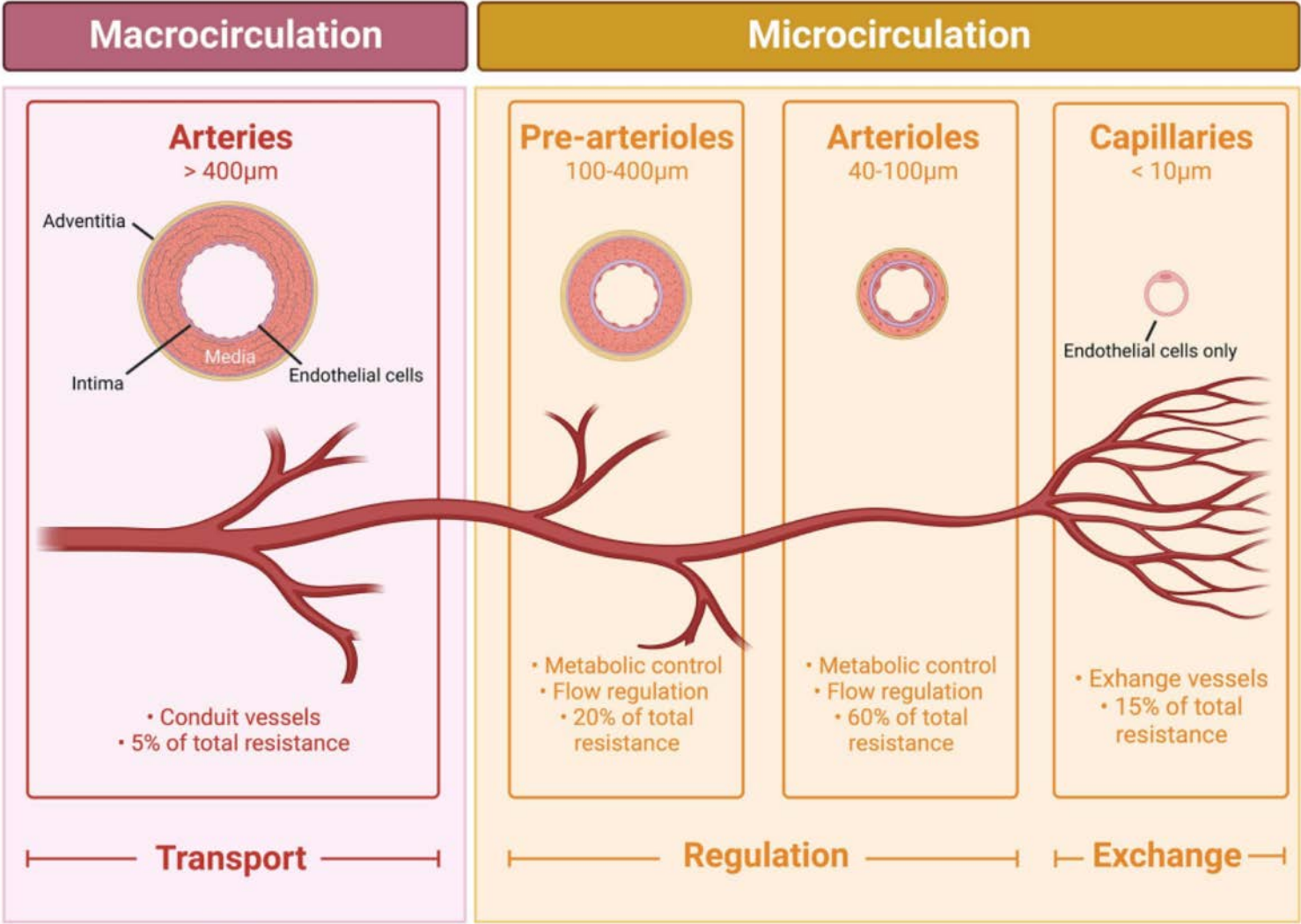
2

↗ EO₂

2 ↗ EO₂



2 ↗ EO₂

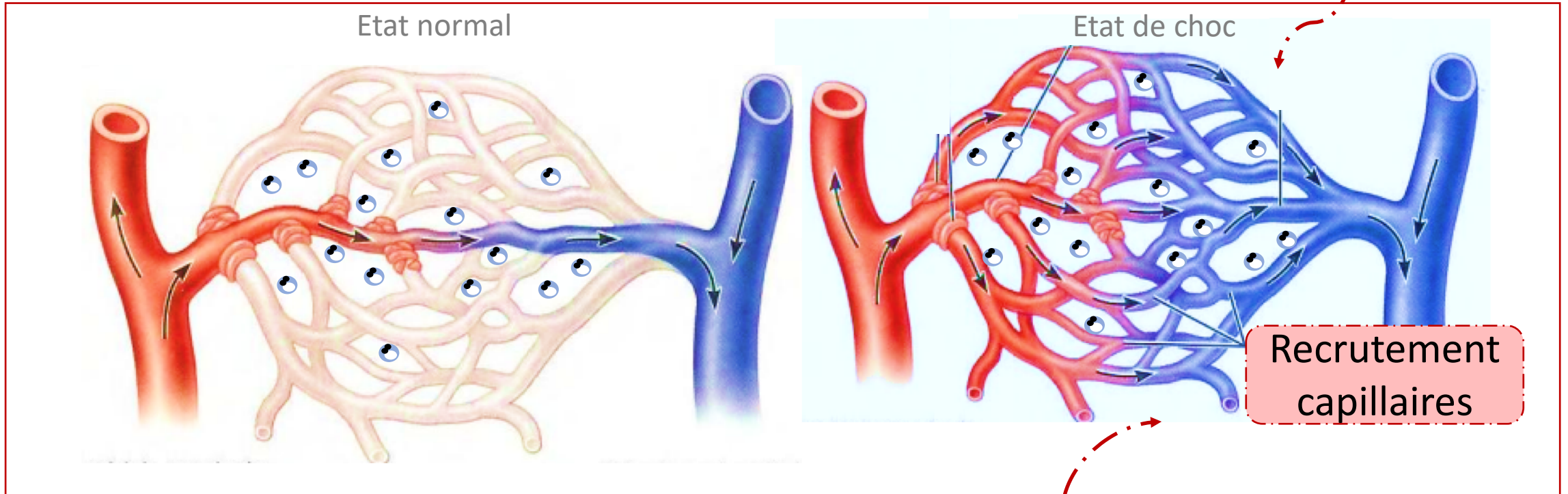


2

↗ EO_2



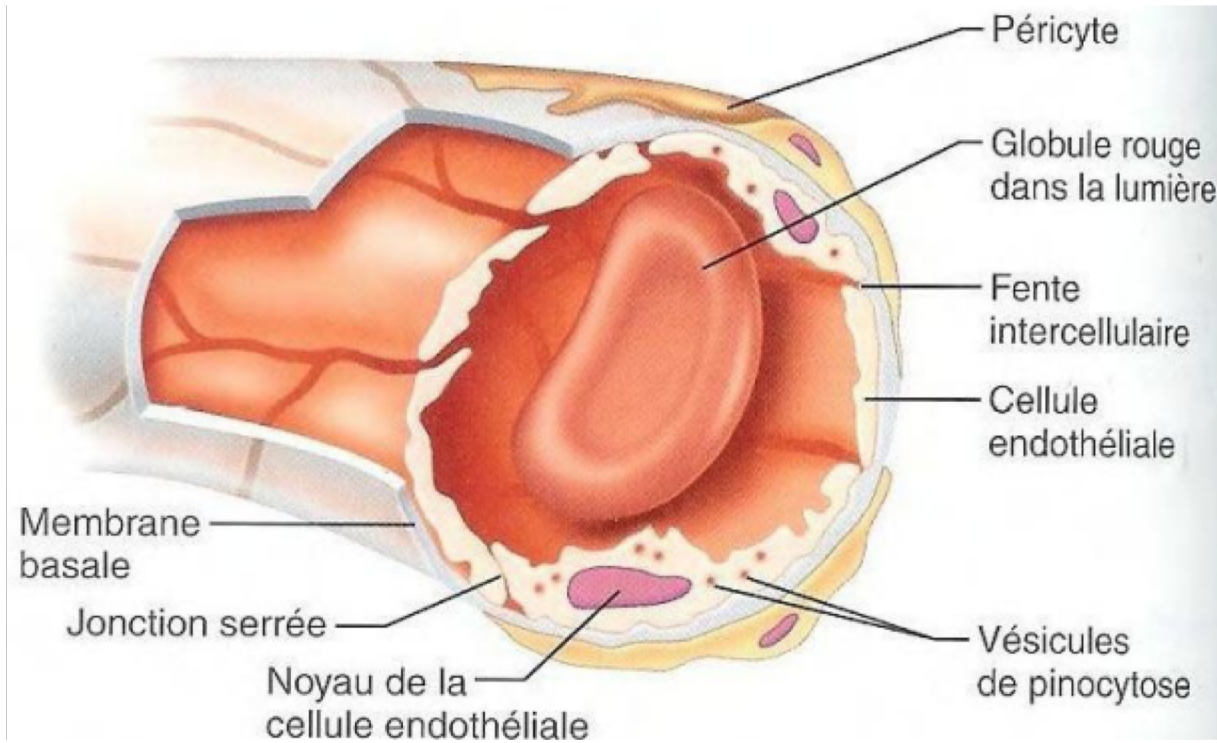
↗ surface d'échanges



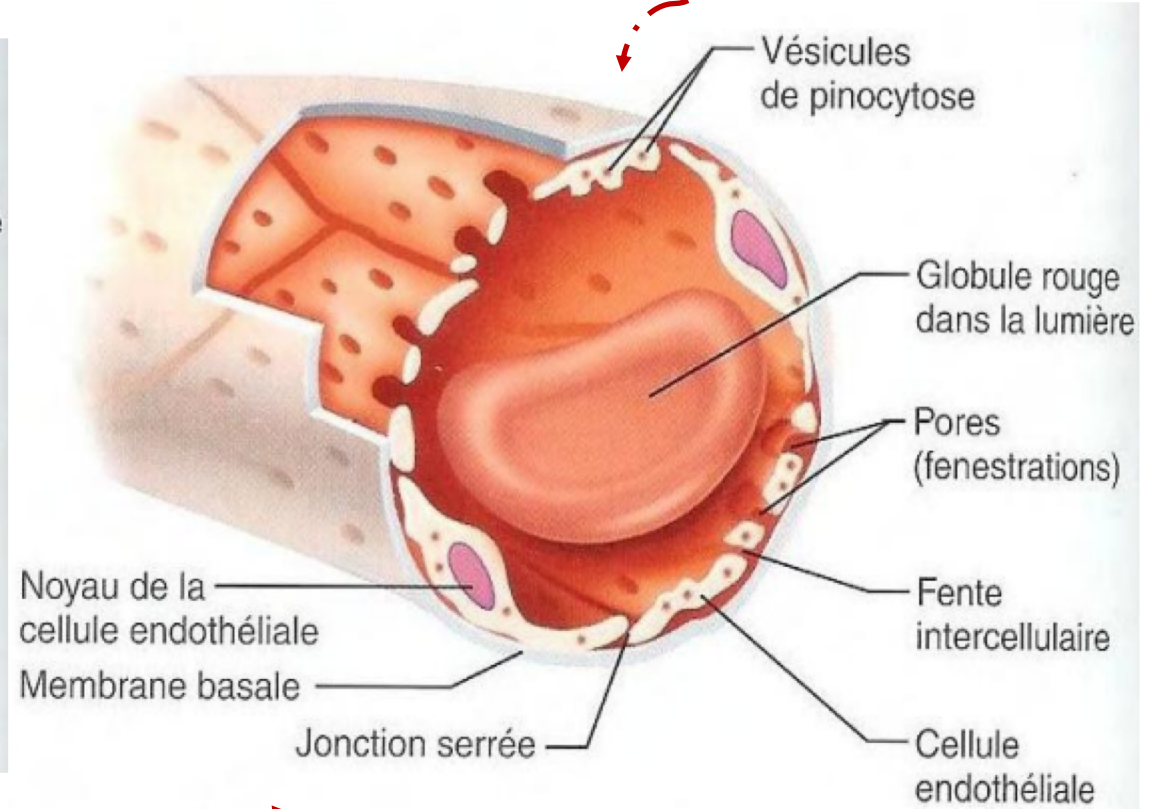
Vasodilatation μ circulation

2

↗ EO₂



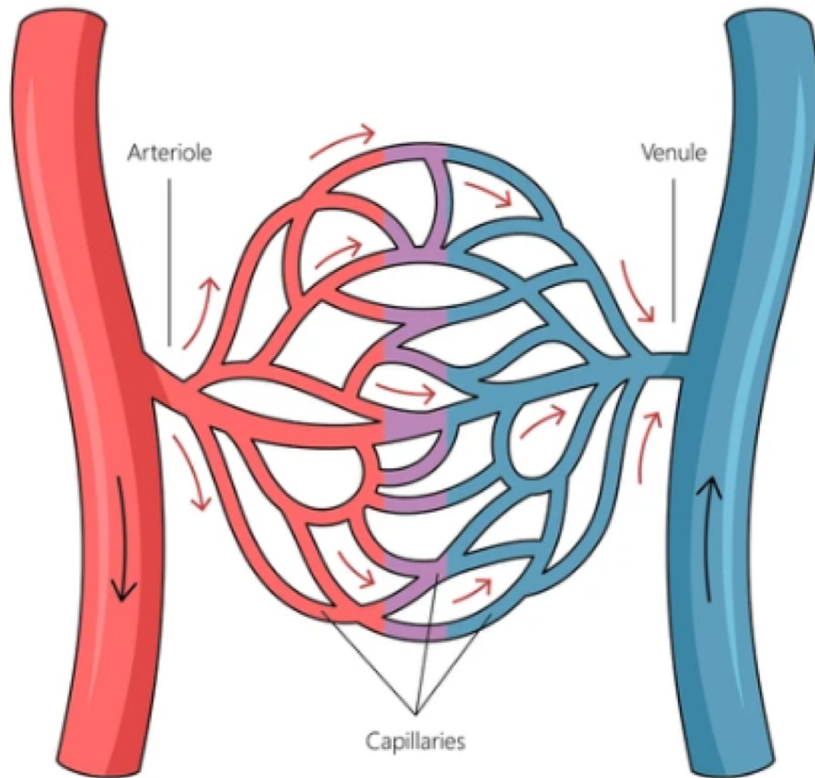
Ouverture de pores



↗ perméabilité

2

↗ EO₂

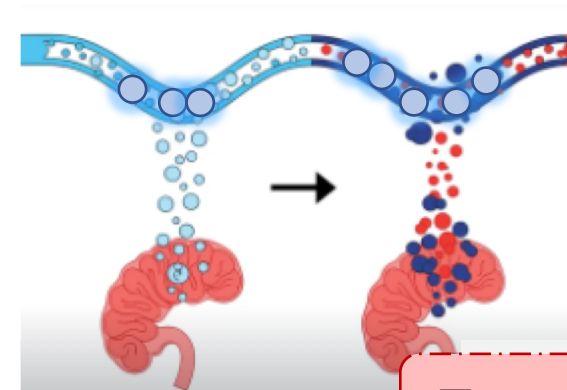


Vasodilatation μ circulation



NO

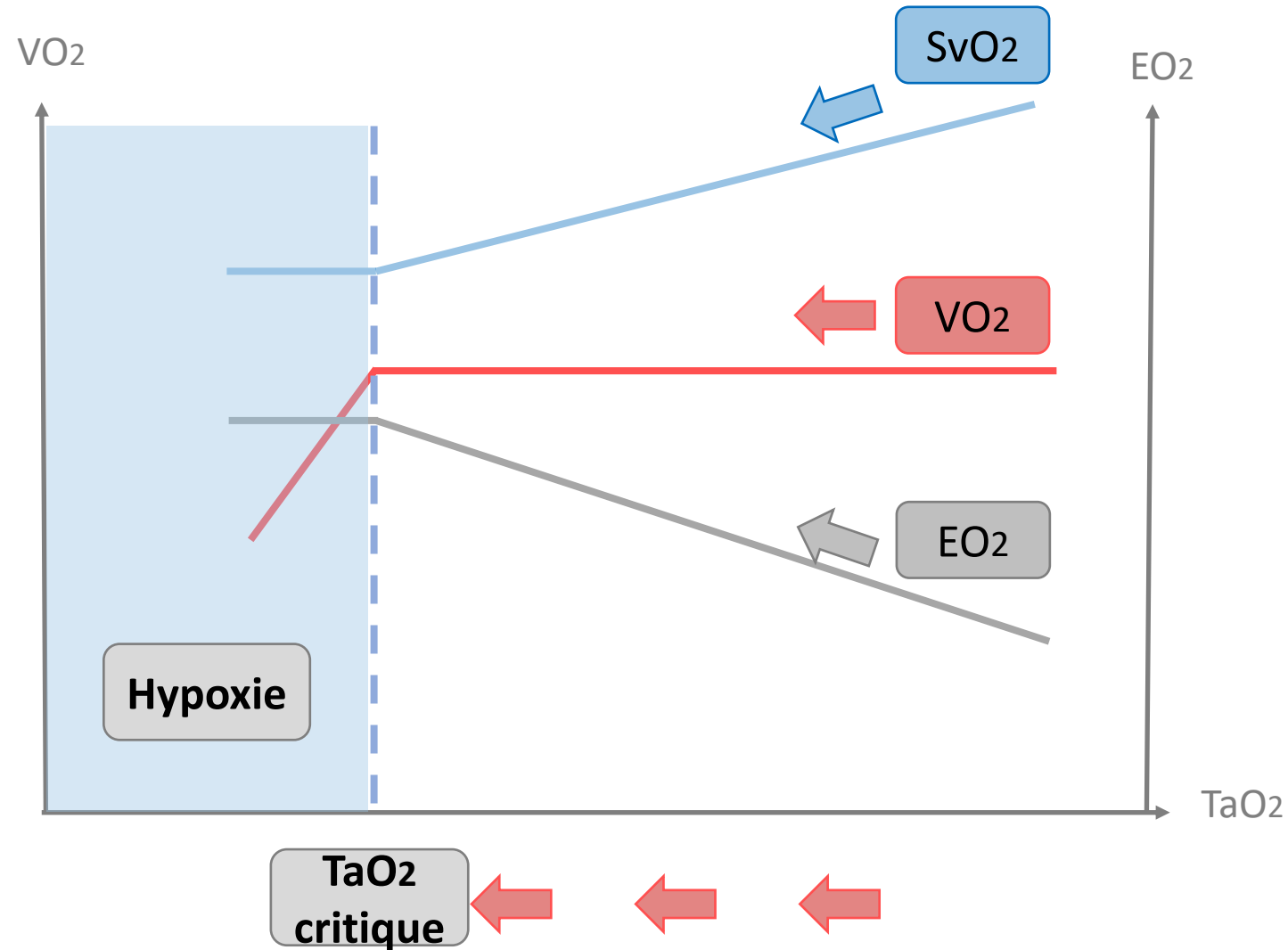
Prostaglandines



↗ surface d'échanges

↗ Extraction










② ↗ EO_2



ESICM guidelines on circulatory shock and hemodynamic monitoring 2025.

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



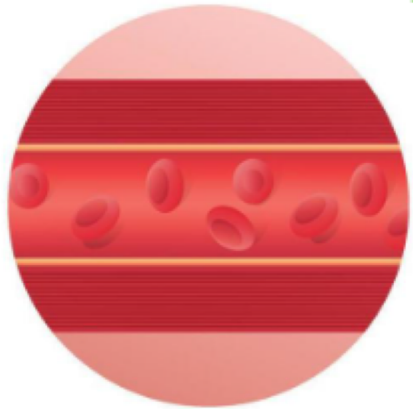
WHAT MARKERS OF THE REGIONAL CIRCULATION AND TISSUE OXYGENATION IN SHOCK SHOULD BE USED?			
→ Serial assessment of tissue perfusion should be performed to follow shock evolution and to help assess the underlying pattern and the adequacy of CO and vascular function.			
2014 We recommend serial measurements 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

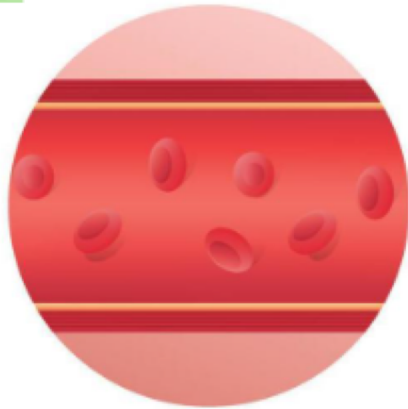
La SvO₂ →apprécier l’adaptation du DC aux besoins tissulaires en O₂

① Système Σ

② \nearrow EO₂



Vasoconstriction



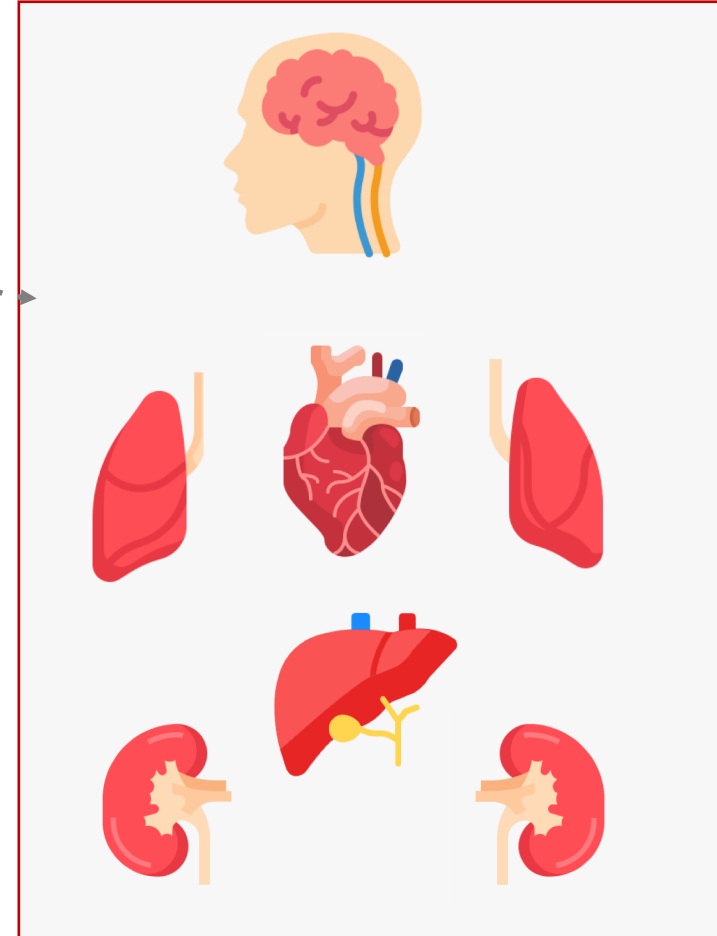
Vasodilatation



Partition du débit cardiaque



Privilège organes nobles

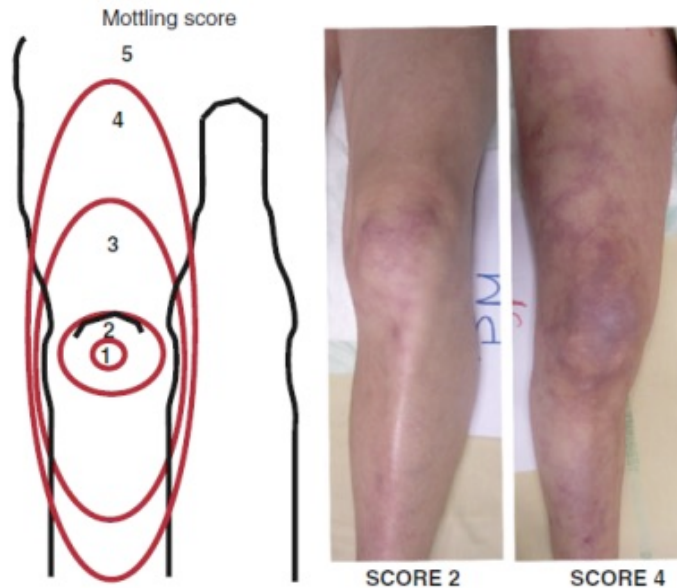


Effets variables de ces mécanismes adaptatifs selon les territoires



Marbrures

Ce que l'on connaît déjà



Signe d'insuffisance circulatoire aiguë

Marbrures

Les nouveautés



1

Intérêt pronostic

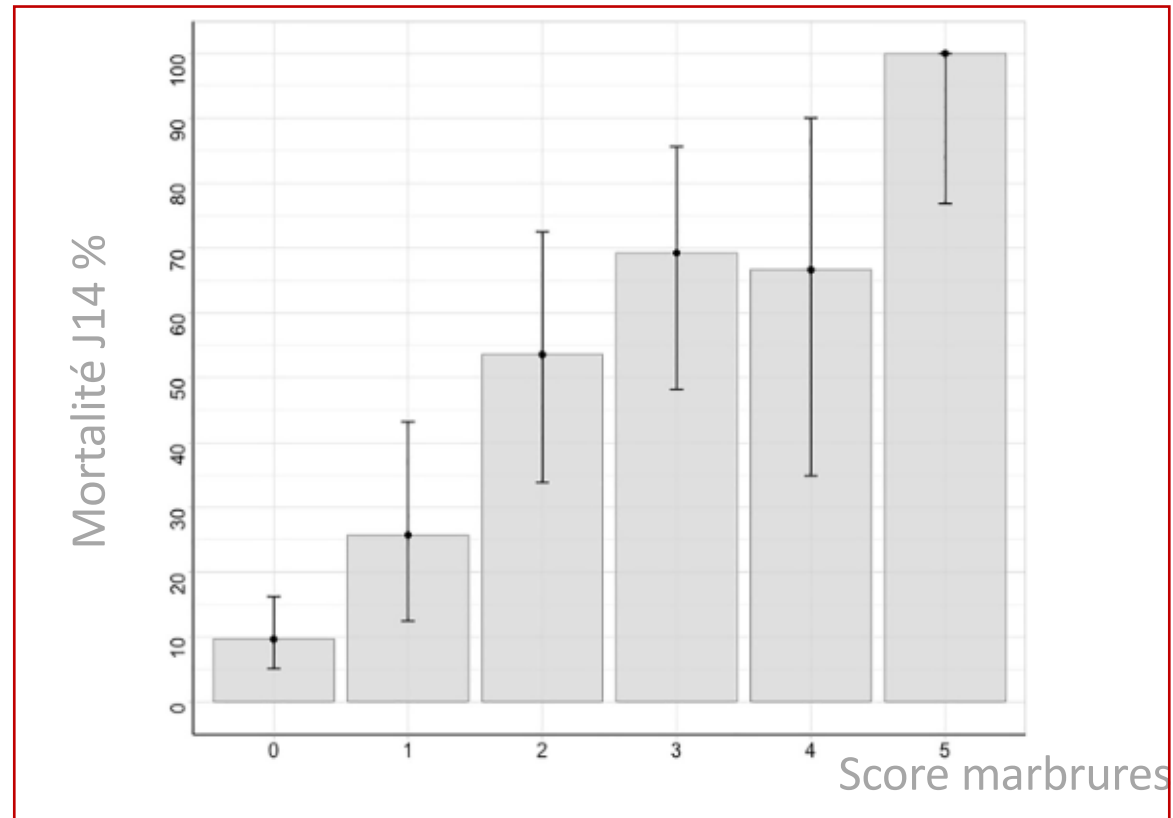
Marbrures → associée à la mortalité dans différents types de choc

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

Etat de choc



Hypovolémique

Cardiogénique

Distributif

Obstructif

Mécanismes compensateurs

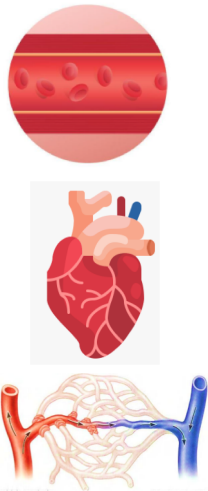


- 1 Système Σ
- 2 \nearrow EO₂

Vasodilatation

Dysfonction cardiaque

Atteinte de la μ circulation



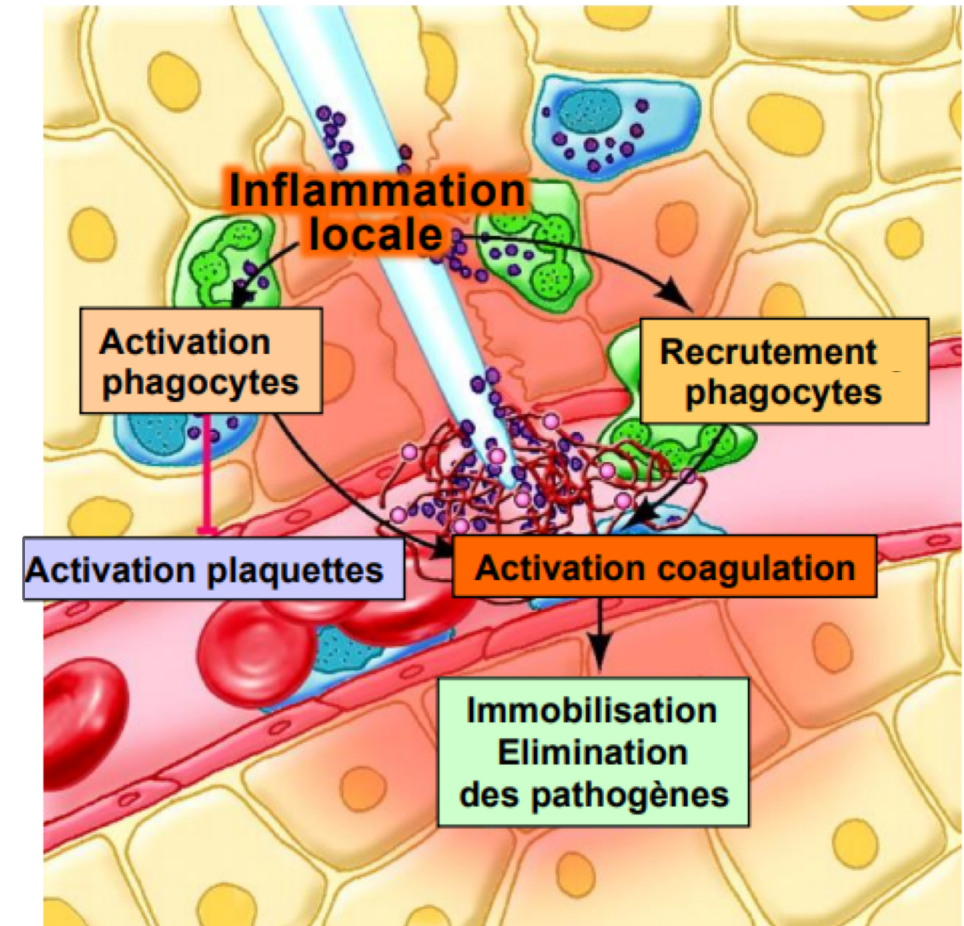
Choc septique

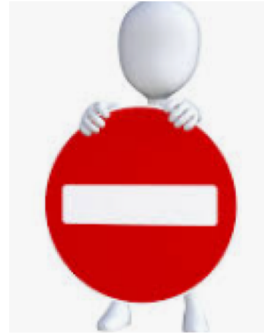
Dysfonction de la
microcirculation

Microthrombosis

Shunt AV

Inflammation

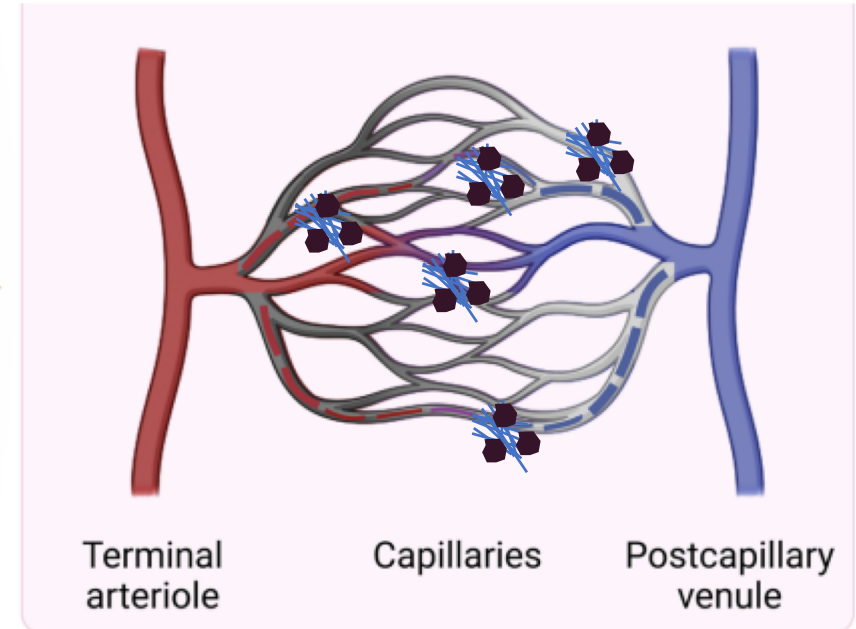




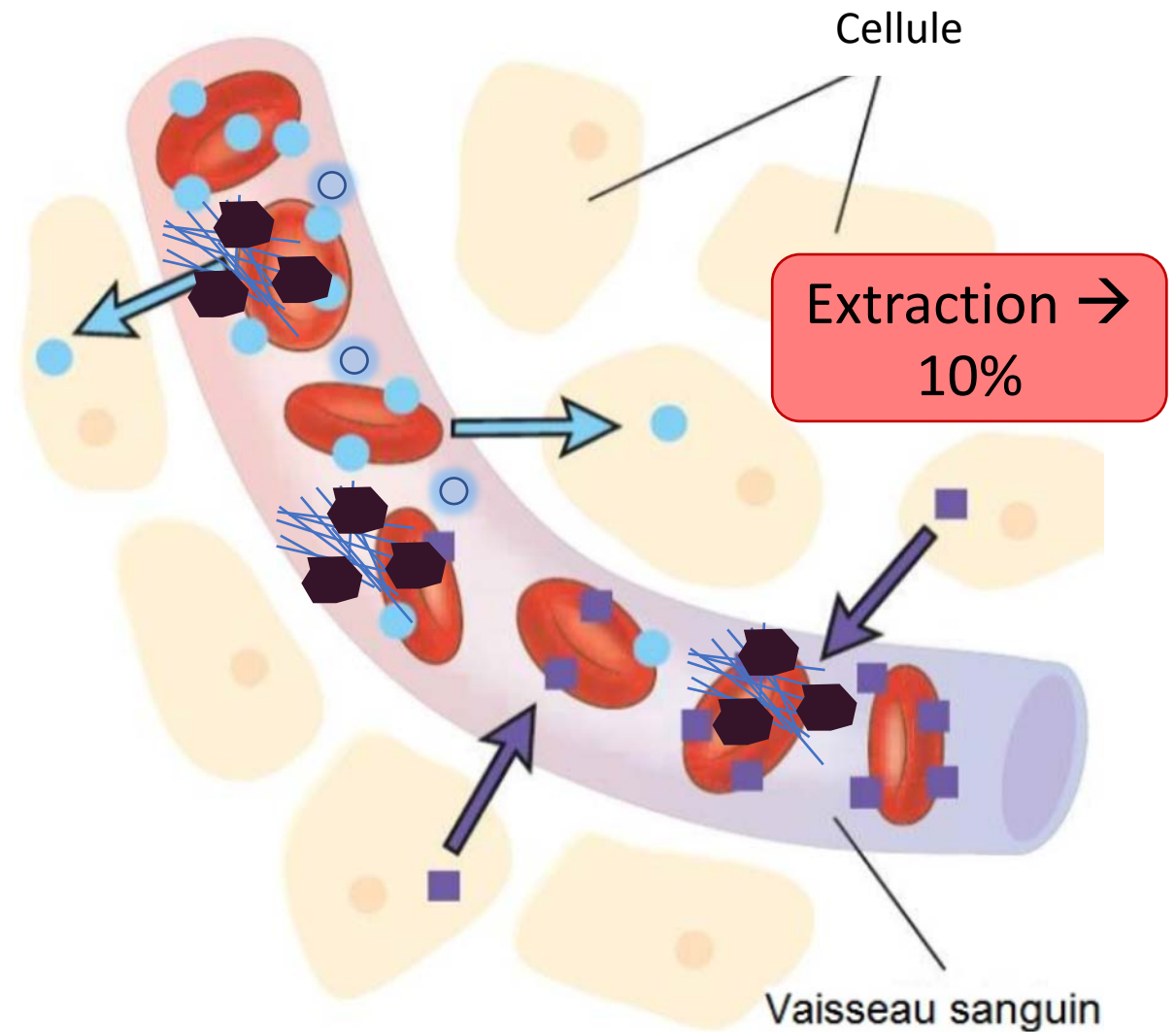
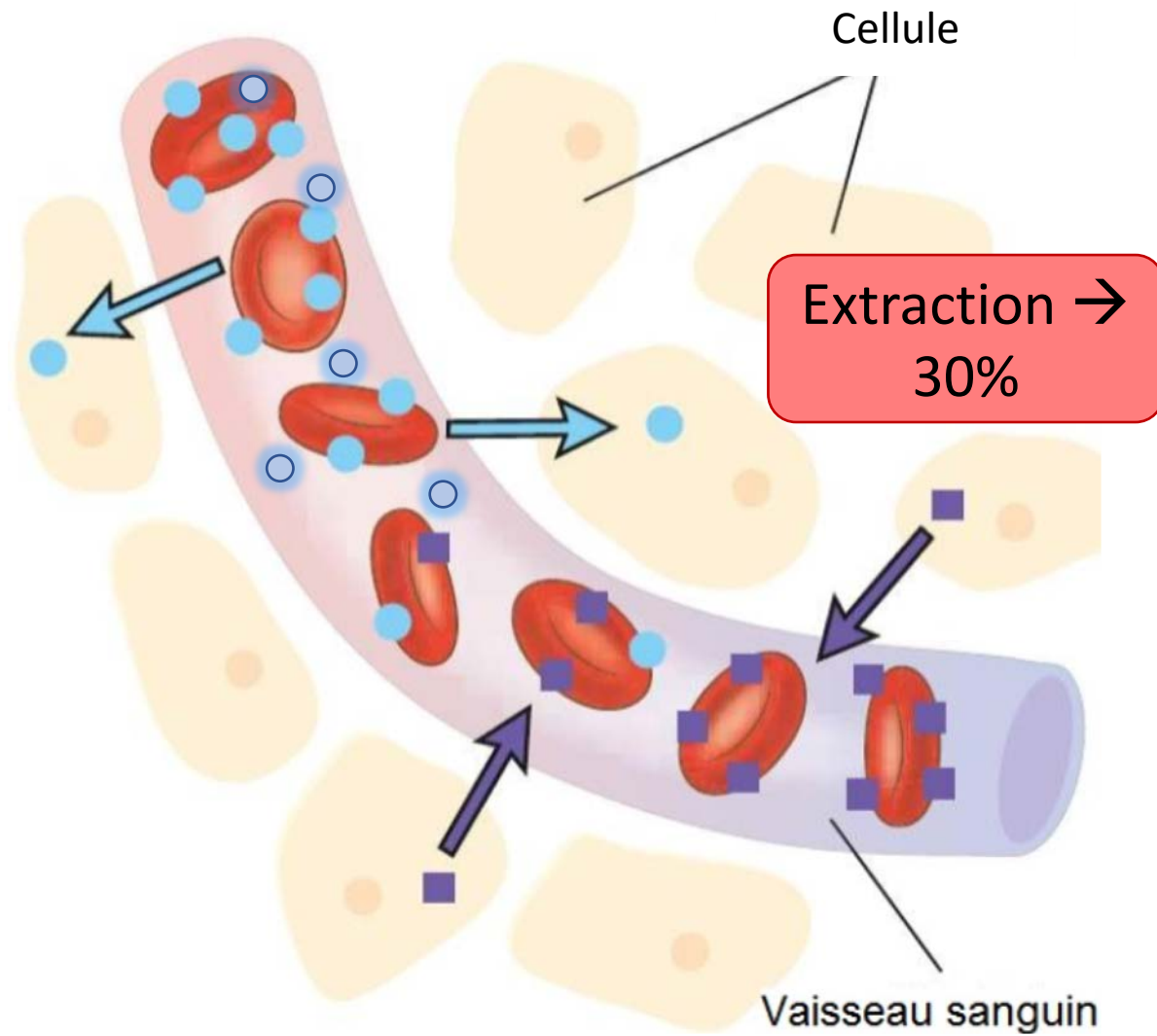
Vasodilatation

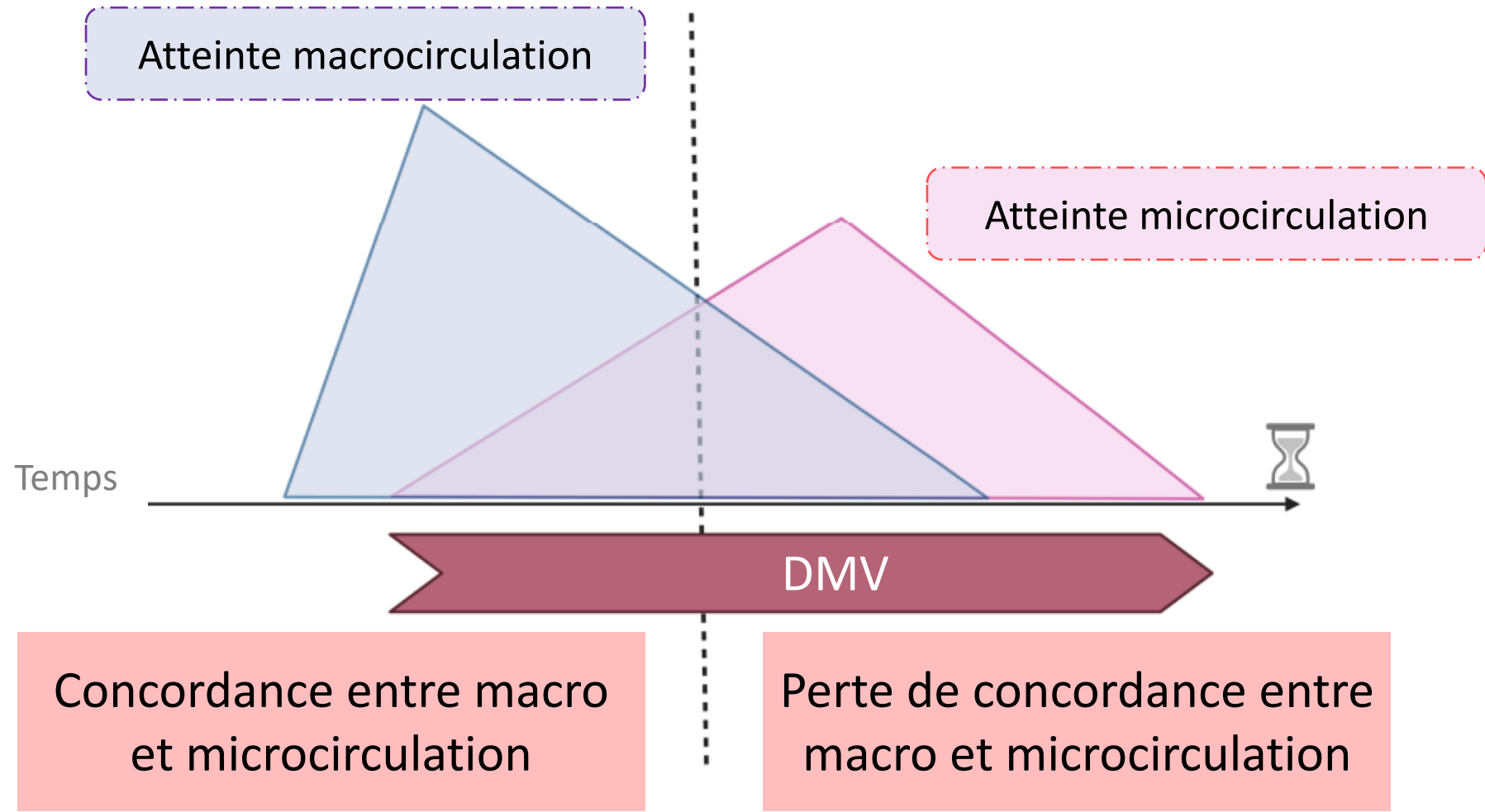
Recrutement capillaires fermés

↗ extraction en O₂

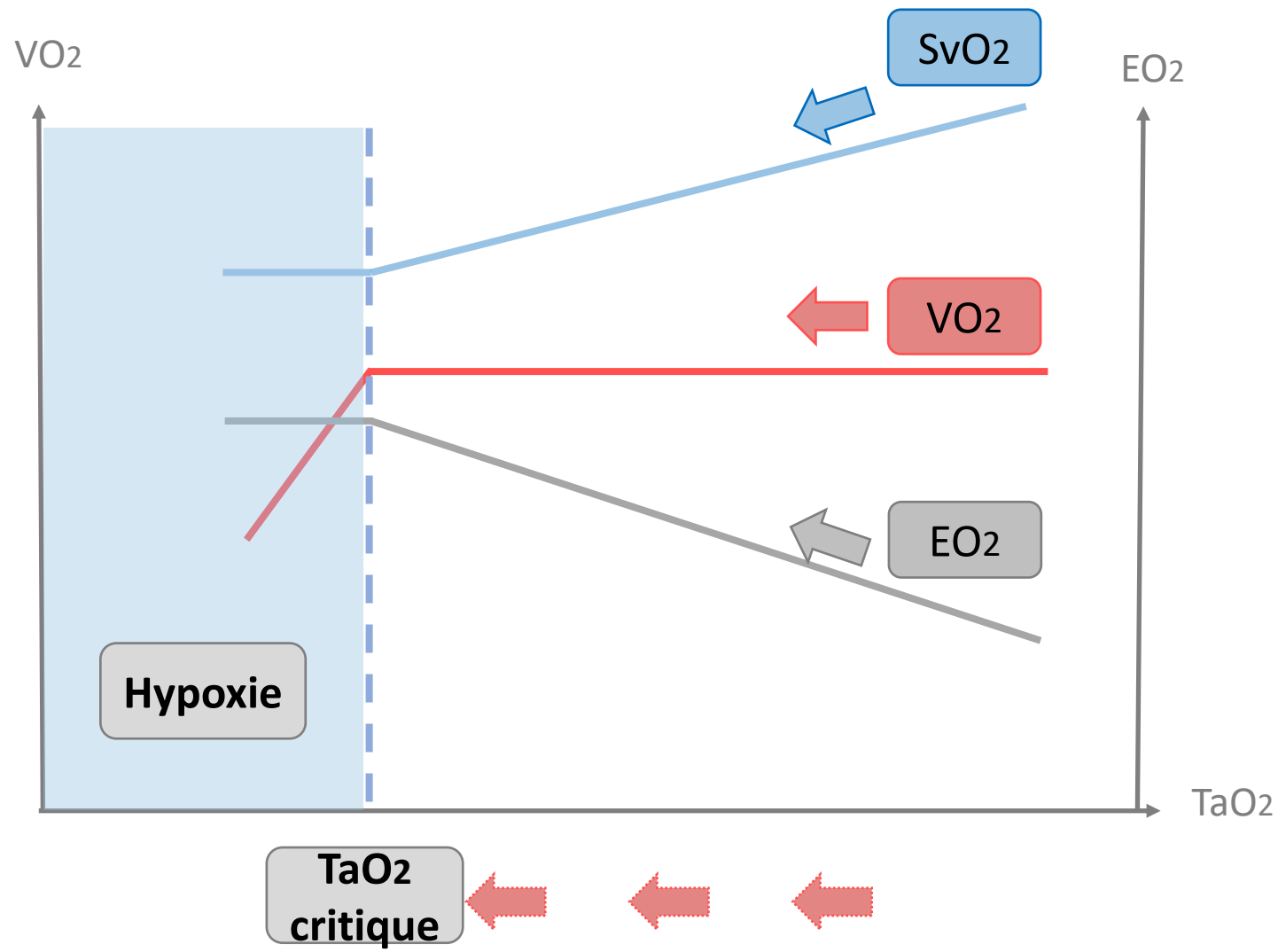


Mécanismes adaptatifs : le cas du choc septique

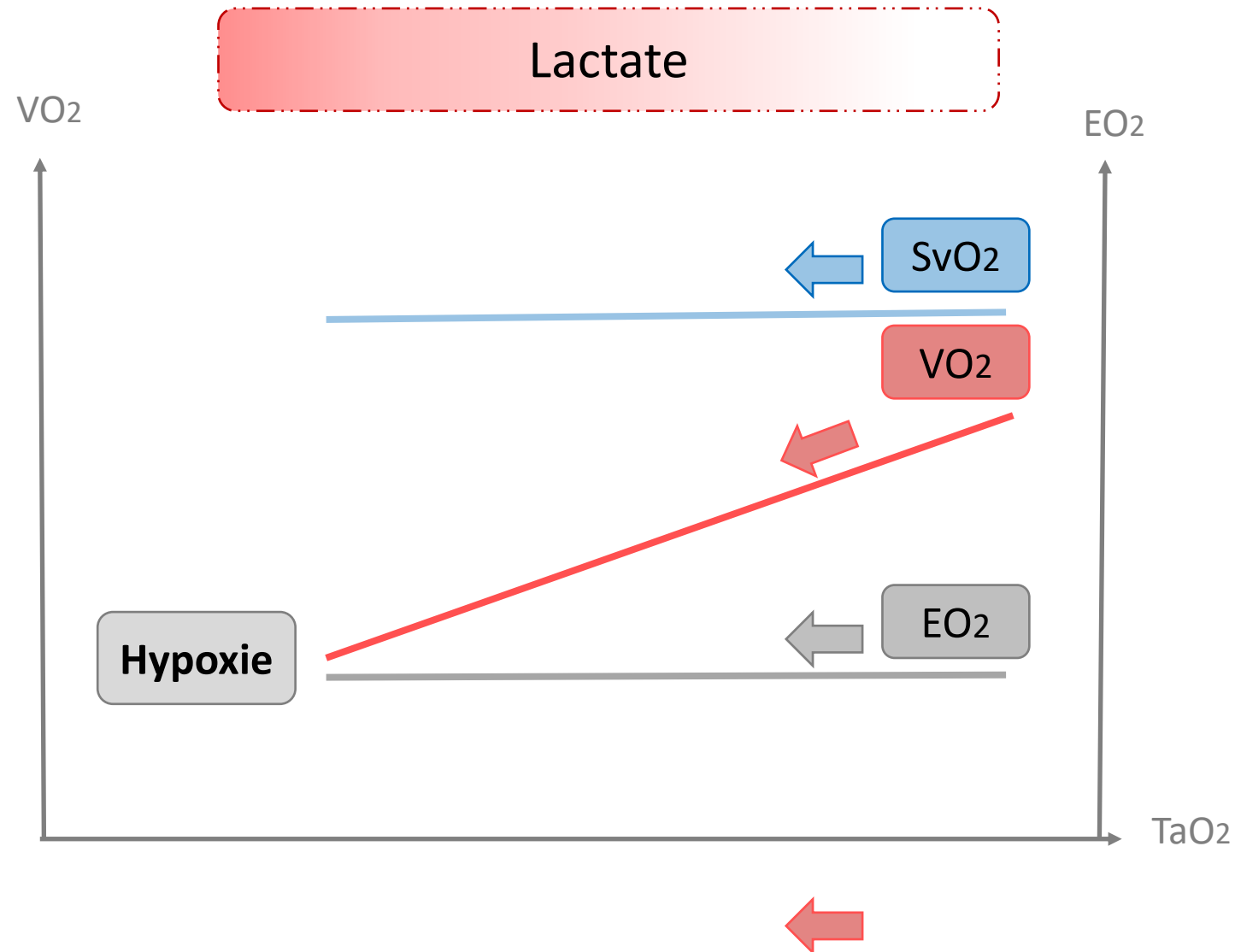




2 ↗ EO_2



2 ↗ EO_2



1

Etat de choc → inadéquation entre apports et besoins en O₂

2

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

3

4

1

Etat de choc → inadéquation entre apports et besoins en O₂

2

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

3

↗ EO₂ et l'activation système sympathique → mécanismes compensateurs



4



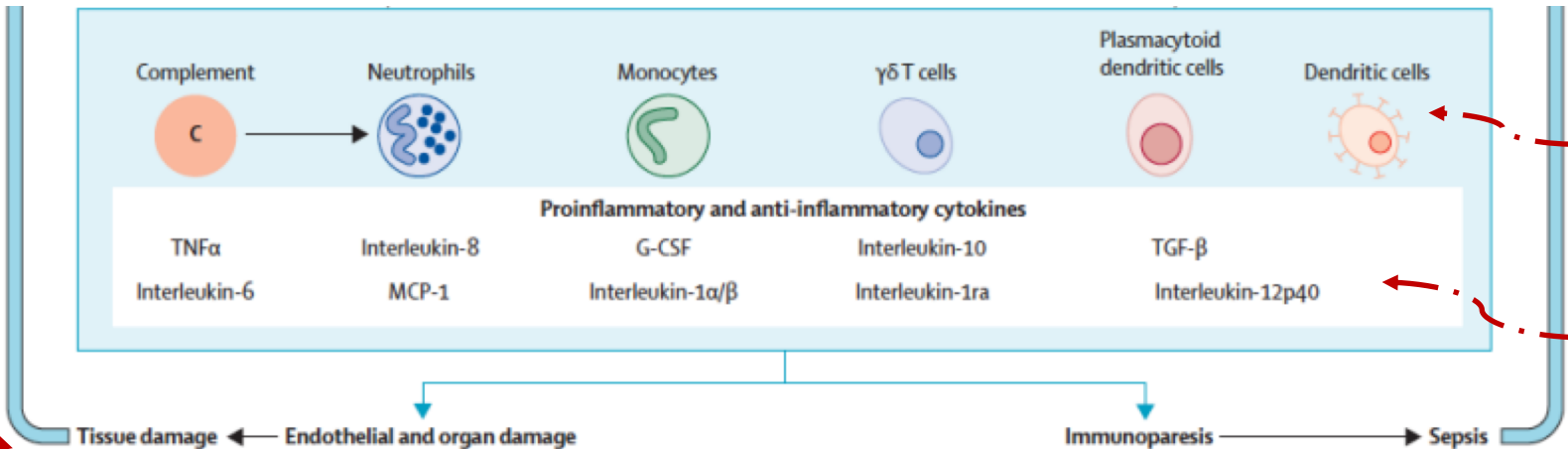
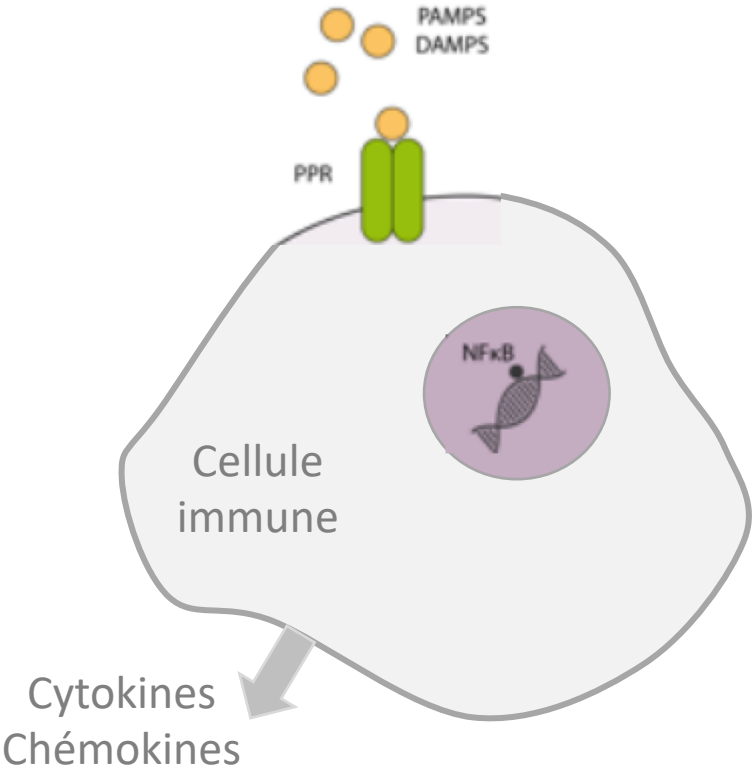
Quel est l'impact de l'état de choc ?

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

Production cellules

Activation cytokines pro inflammatoires

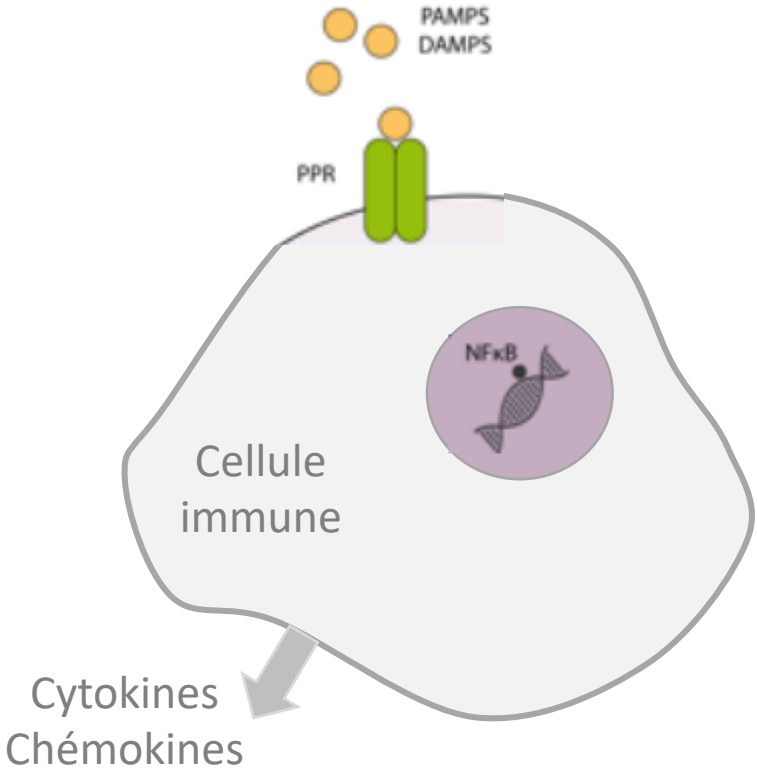
Dommage tissulaire



↗ Inflammation

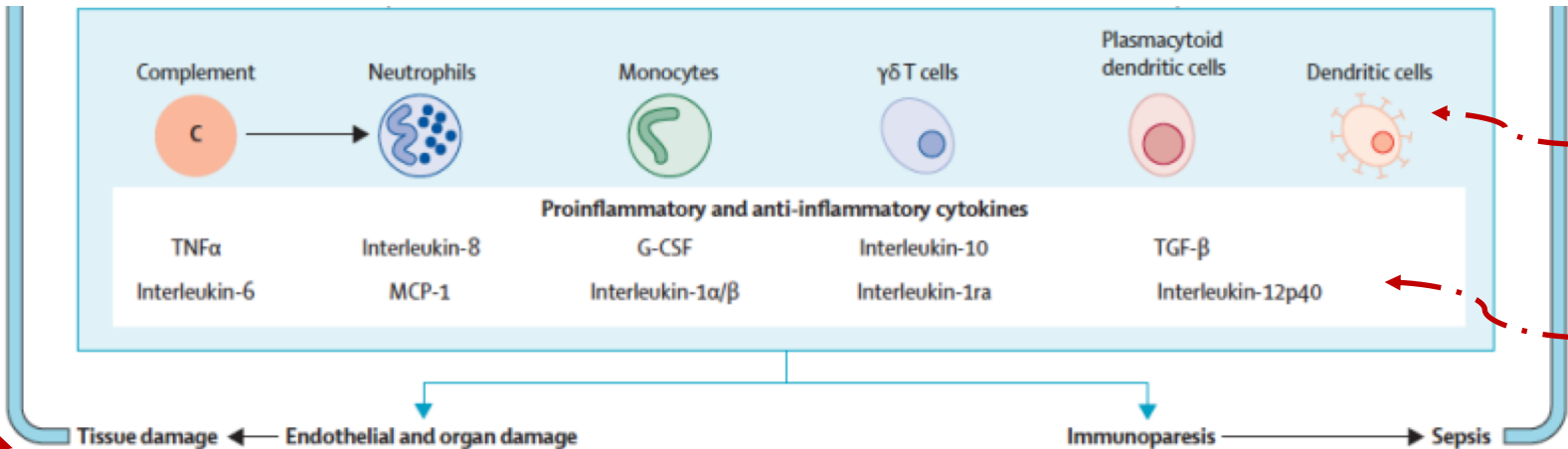
Atteinte endothéliale

Production cellules



Activation
cytokines pro
inflammatoires

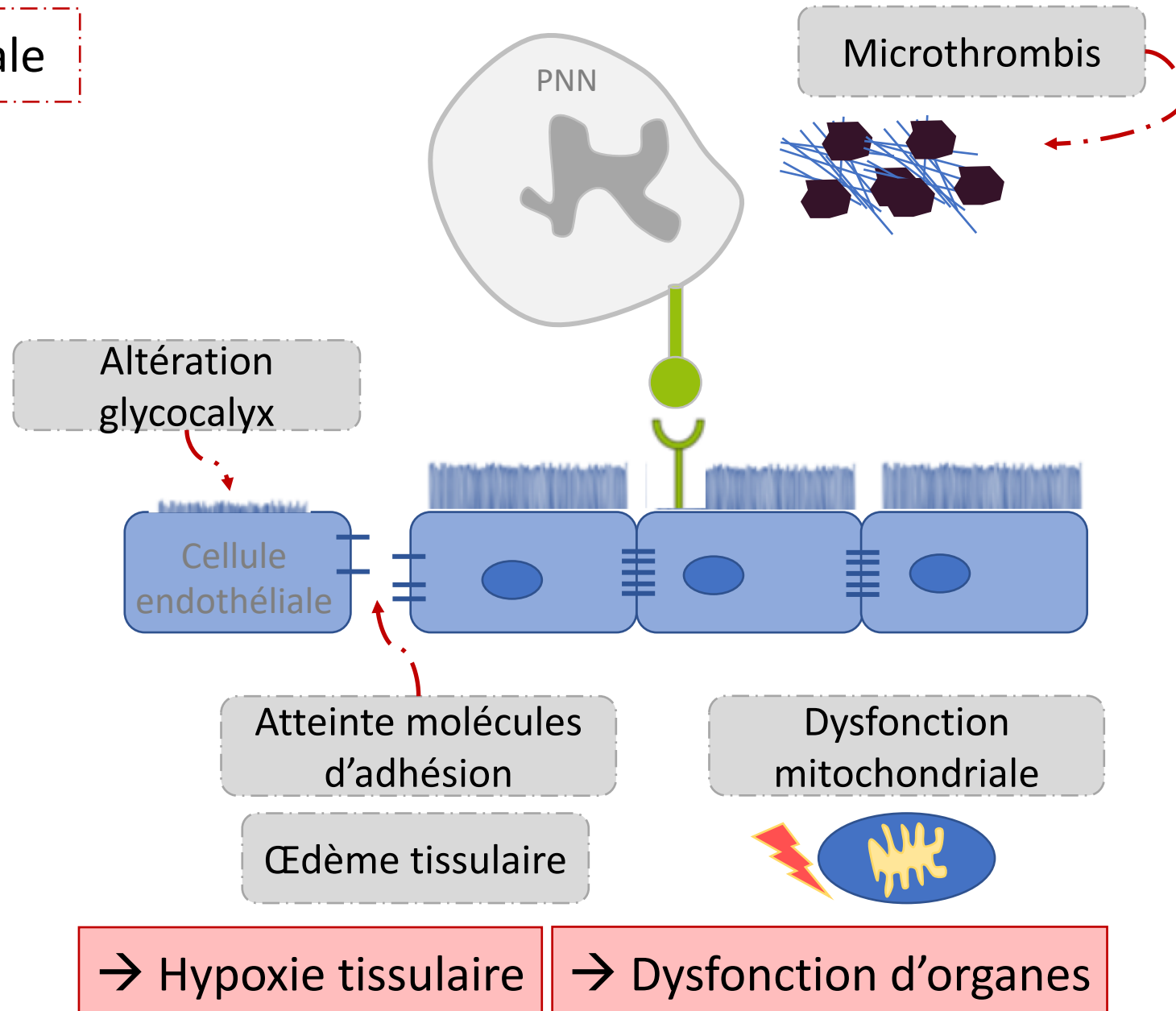
Dommage
tissulaire



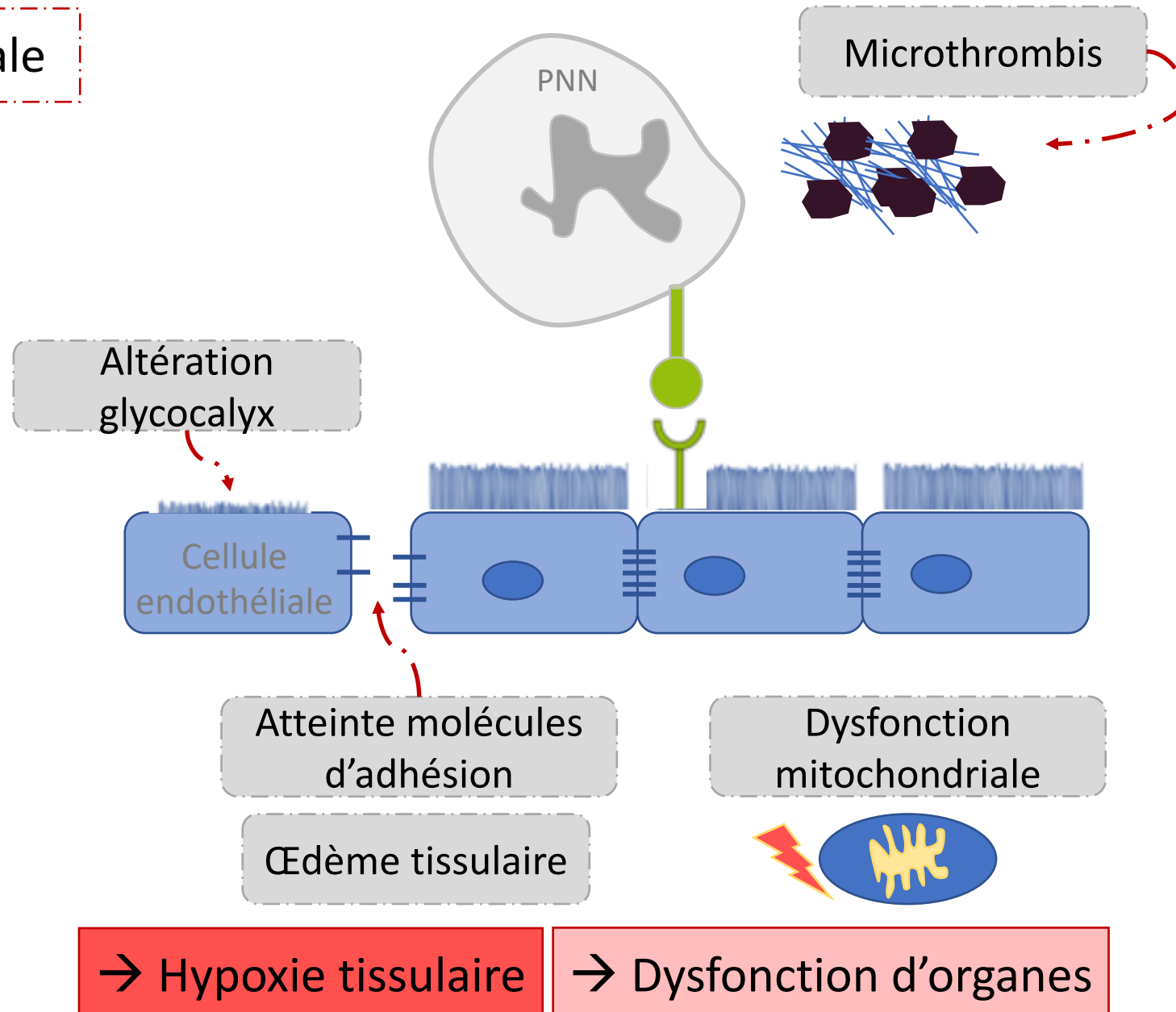
↗ Inflammation

Atteinte
endothéliale

Atteinte endothéliale



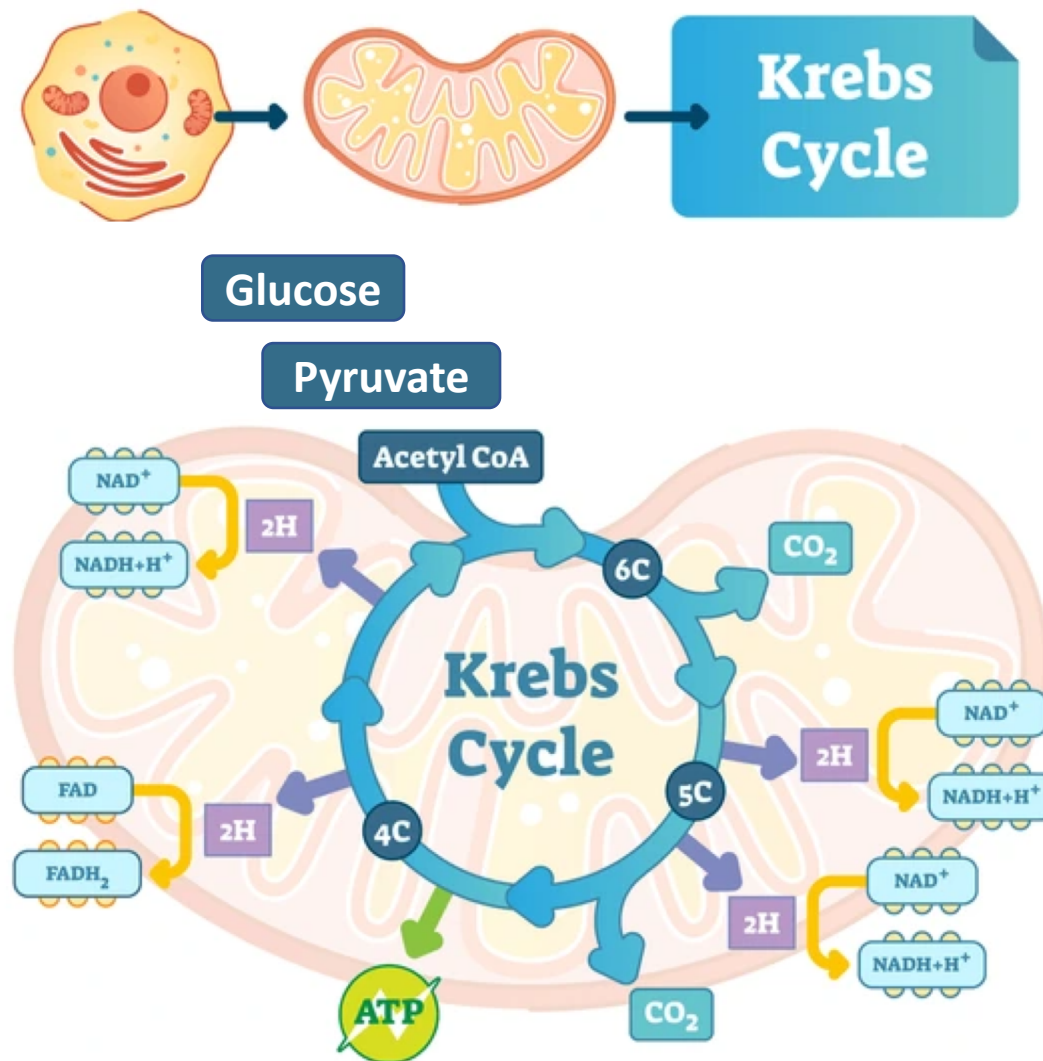
Atteinte endothéliale



Hypoxie tissulaire

O₂

Métabolisme
aérobie

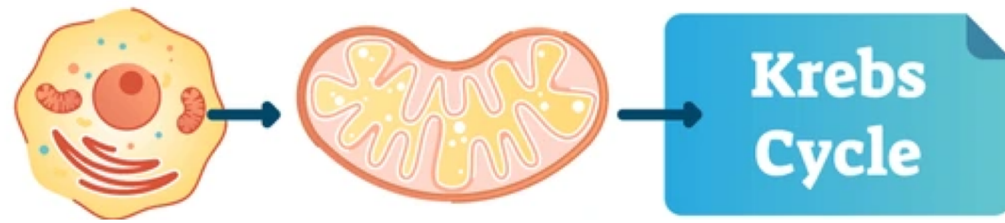


1 molécule de glucose
→ 38 molécules d'ATP

Hypoxie tissulaire



Métabolisme
anaérobie



Glucose

Pyruvate

Lactate +++

Indicateur de
l'anaérobiose

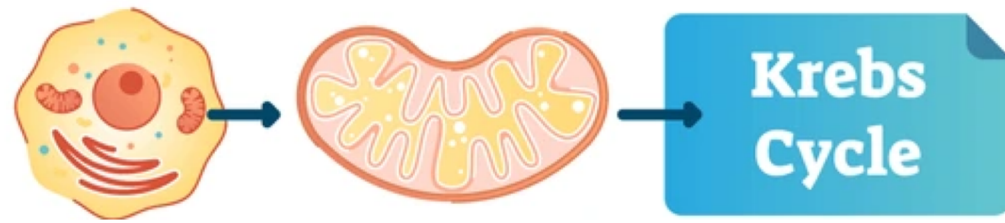
→ Hypoxie tissulaire

→ Dysfonction d'organes

Hypoxie tissulaire



Métabolisme
anaérobie



Glucose

Pyruvate

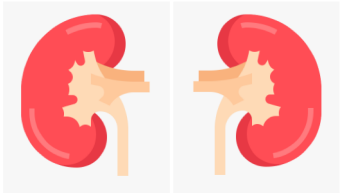
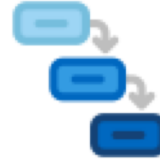
Lactate +++

Indicateur de
l'anaérobiose

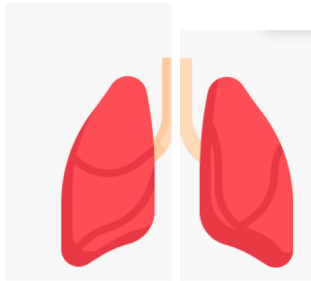
→ Hypoxie tissulaire

→ Dysfonction d'organes

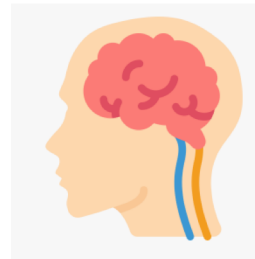
Dysfonction d'organes



Insuffisance
rénale aiguë



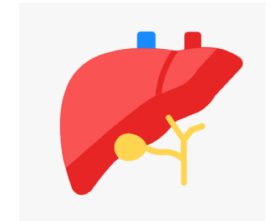
SDRA



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 \searrow du TaO₂ (DC) et/ou \searrow 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 \searrow du TaO₂ (DC) et/ou \searrow de PA

3

↗ EO₂ et l'activation système sympathique → mécanismes compensateurs



4

Etat de choc → dysfonction endothéliale, hypoxie tissulaire et DMV

Conclusions

Physiopathologie



TaO₂



Contractilité

Pression artérielle

Choc septique



1

Dysfonction μ circulation

2

Inflammation

3

Microthrombis

4

Ø Mécanismes compensateurs

5

Ø d'augmentation EO₂

6

SvO₂ anormalement normale

Mécanismes

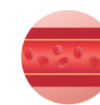
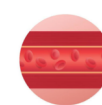


Système sympathique

↗ EO₂

Vasoconstriction

Vasodilatation
 μ circulation



Impact



Production c. inflammatoires

Dysfonction endothéliale

Hypoxie tissulaire

Dysfonction d'organes

Merci pour votre attention