Syndrome de défaillance multivicérale (SDM): physiologie et diagnostic

Dr Ines FATHALLAH
Service de réanimation médicale
Hôpital régional de Ben Arous

INTRODUCTION

Concept apparu dans les années 70

ANNALS OF SURGERY
Vol. 178 August 1973 No. 2

Sequential System Failure after Rupture of Abdominal Aortic Aneurysms:

An Unsolved Problem in Postoperative Care

NICHOLAS L. TILNEY, M.D.,* GEORGE L. BAILEY, M.D.,** ALFRED P. MORGAN, M.D.***

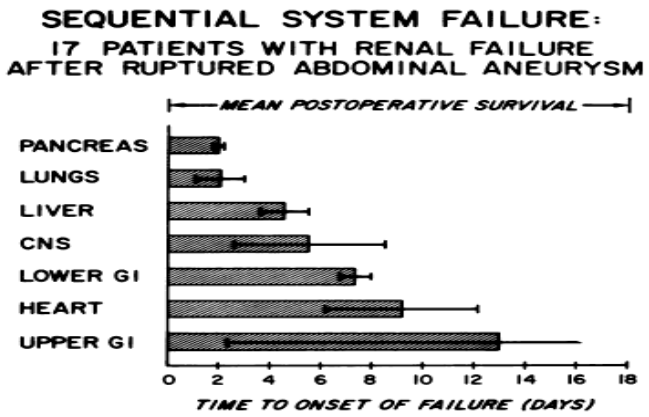

FIG. 1. The order of appearance of organ system failure is shown by normalizing survival time for each patient to mean duration of

Results

Although 17 of the 18 patients died, there was a wide distribution of survival times. One died on the 5th postoperative day, one on the 48th; the remaining 15 deaths occurred between these extremes. Despite these differ-

INTRODUCTION

Plusieurs synonymes utilisés

- ✓ Sequential systems failure
- ✓ Multiple organ failure
- ✓ Multiple system organ failure
- ✓ Multiple organ system failure

INTRODUCTION

- Incidence 11–40%

CHEST

Volume 127, Issue 3, March 2005, Pages 942-951

Clinical Investigations in Critical Care

Incidence and Impact of Organ Dysfunctions Associated With Sepsis

Bertrand Guidet MD [a], Philippe Aegerter MD, PhD [b], Remy Gauzit MD [c], Patrick Meshaka MD [d], Didier Dreyfuss MD [e], † On behalf of the CUB-Rea Study Group

Measurements and main results

We collected information on demographic characteristics, type of admission, underlying disease, organ dysfunction, organ support, McCabe and Charlson-Deyo scores, simplified acute physiology score II, length of stay, and outcome. The incidence of SS was 27.7%

INTRODUCTION

- Mortalité 27-100%

International Journal of Clinical Medicine, 2012, 3, 722-730
http://dx.doi.org/10.4236/ijcm.2012.37A127 Published Online December 2012 (http://www.SciRP.org/journal/ijcm)

Multiple Organ Dysfunction Syndrome (MODS): Is It Preventable or Inevitable?*

Ayman El-Menyar[1,2#], Hassan Al Thani[3], El Rasheid Zakaria[4], Ahmad Zarour[3], Mazin Tuma[3], Husham AbdulRahman[3], Ashok Parchani[3], Ruben Peralta[3], Rifat Latifi[2,3,5]

[1]The Clinical Research, Trauma Surgery, Hamad Medical Corporation (HMC), Doha, Qatar; [2]Clinical Medicine, Weill Cornell Medical School, Doha, Qatar; [3]Trauma Surgery, Hamad Medical Corporation (HMC), Doha, Qatar; [4]Medical Research Center, Hamad Medical Corporation (HMC), Doha, Qatar; [5]Department of Surgery, University of Arizona, Tucson, Arizona, USA.
Email: #aymanco65@yahoo.com

Received September 26th, 2012; revised November 11th, 2012; accepted November 24th, 2012

Multiple organ dysfunction syndrome (MODS) is a systemic, dysfunctional inflammatory response that requires long intensive care unit (ICU) stay and has high mortality rate of 27% - 100% depending on the number of organs involved [1-3]. However, there is no gold standard scor-

INTRODUCTION

Analytic Review

Journal of Intensive Care Medicine
1-12
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DOI: 10.1177/0885066619871452
journals.sagepub.com/home/jic
SAGE

Multiple Organ Dysfunction Syndrome

Nicholas M. Gourd, BSc[1,2], and Nikitas Nikitas, MD, PhD[1]

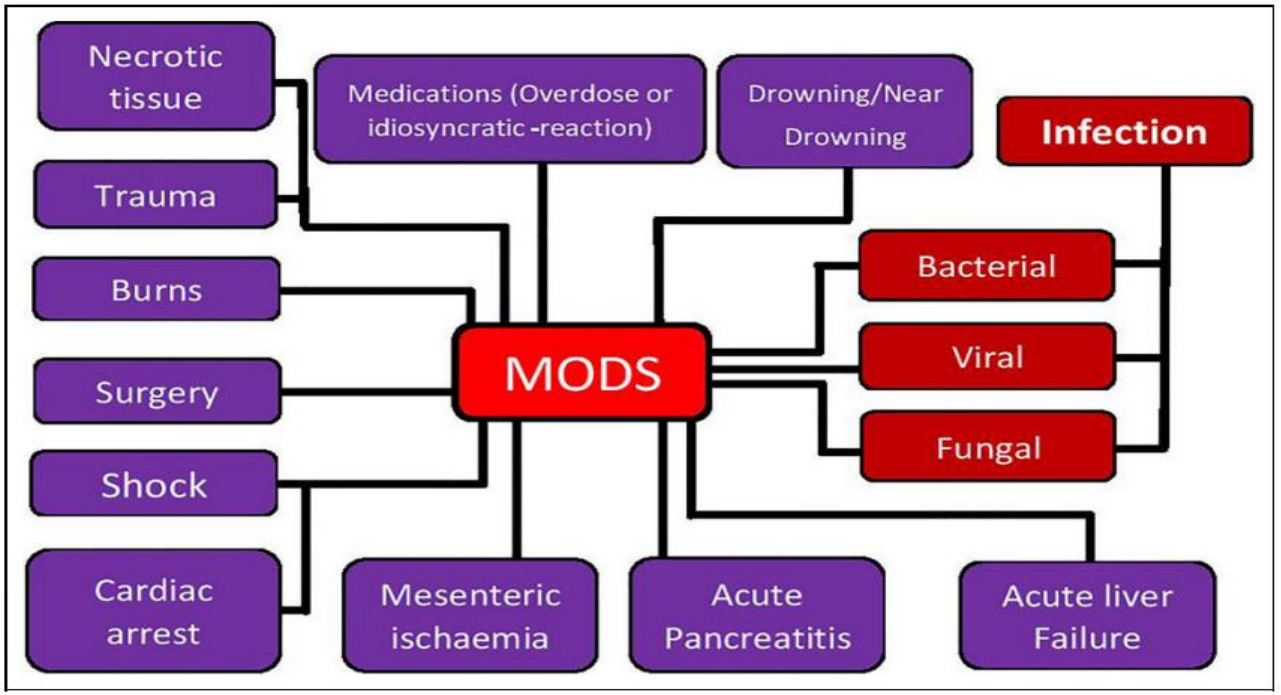
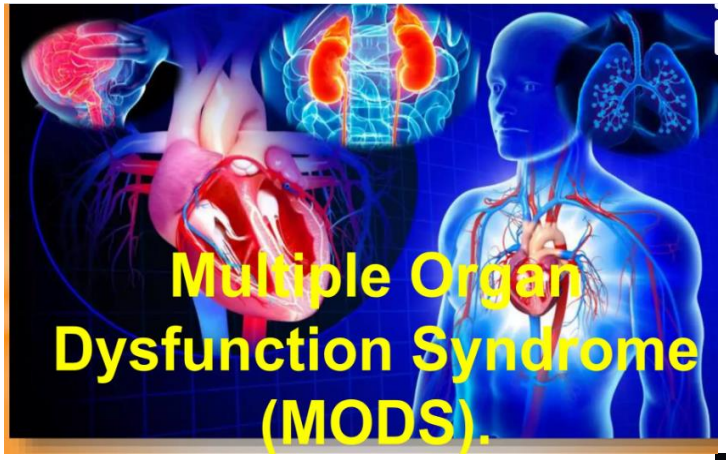
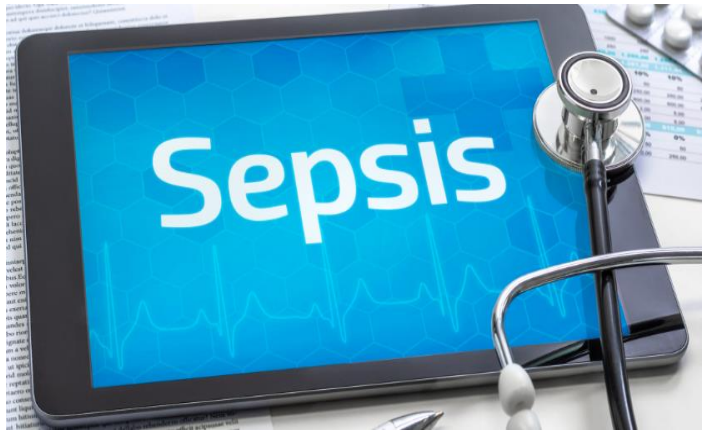


Figure 1. The broad diversity of triggers for multiple organ dysfunction syndrome (MODS) development with infectious shown in red (sepsis) and noninfectious in purple.

INTRODUCTION





DEFINITON

SEPSIS sévère

Dysfonction d'organe secondaire à une **réponse inappropriée** de l'hôte envers **une infection**

Journal of *Clinical Medicine*

MDPI

Review

2023 Update on Sepsis and Septic Shock in Adult Patients: Management in the Emergency Department

Matteo Guarino [1], Benedetta Perna [1], Alice Eleonora Cesaro [1], Martina Maritati [2], Michele Domenico Spampinato [1], Carlo Contini [2,†] and Roberto De Giorgio [1,*,†]

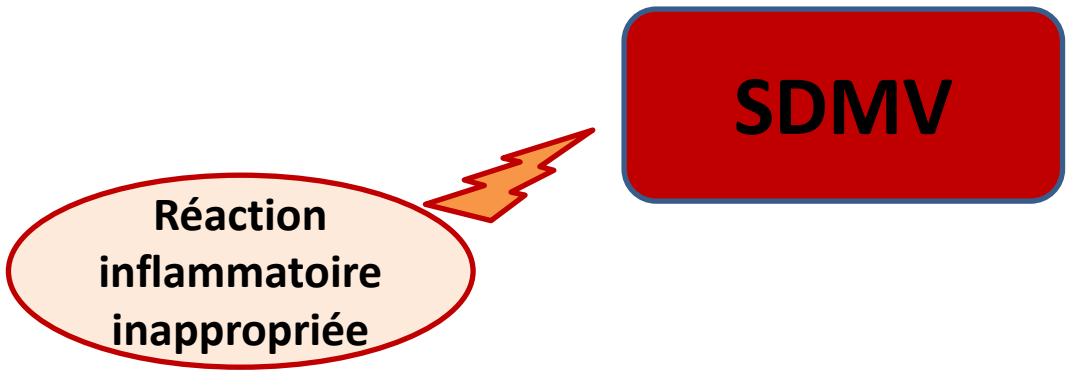
DEFINITION

SDMV

- Des altérations physiologiques
- Potentiellement **réversibles**
- Atteignant au moins **deux** organes
- Non impliqués dans l'**affection initiale**

PHYSIOPATHOLOGIE

HYPOTHESES PHYSIOLOGIQUES



PHYSIOPATHOLOGIE

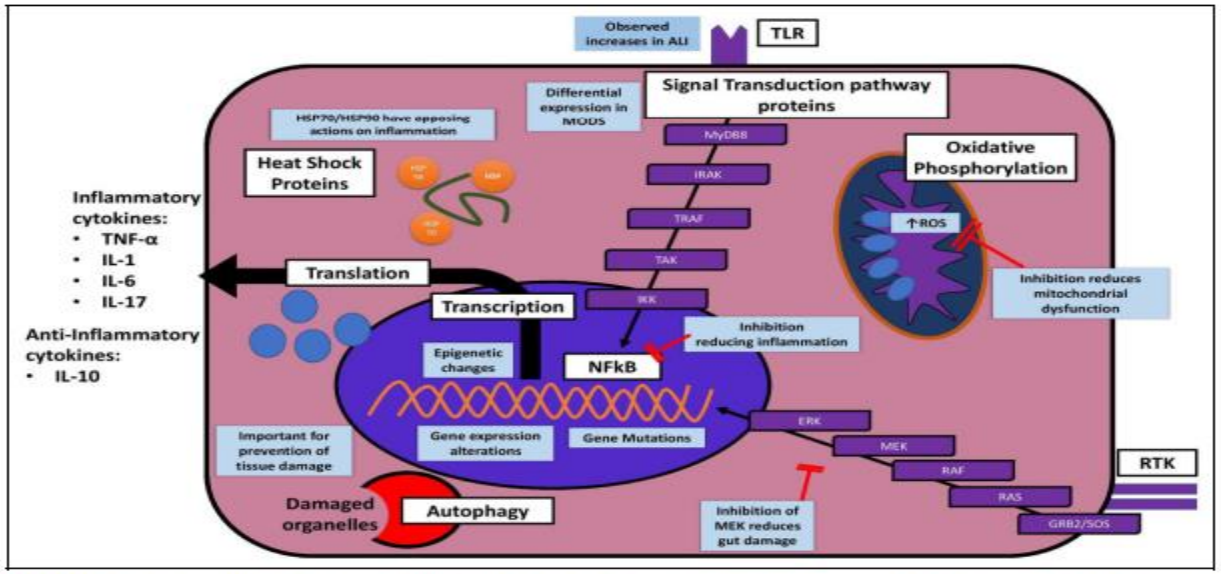


Figure 4. Multiple organ dysfunction syndrome (MODS) has been shown to alter in some way numerous cellular processes including genetic, epigenetic, signal transduction pathways, cell surface receptor expression/function, and mediator release/response. ERK indicates extracellular signal-related kinase; GRB2, growth factor receptor-bound protein 2; IKK, IkB kinase; IRAK, interleukin-1 receptor-associated kinase; MEK, mitogen-activated protein kinase/ERK kinase; MYD88, myeloid differentiation primary response 88; NF-kB, nuclear factor kappa B; RAF, rapidly activating fibrosarcoma; ROS, reactive oxygen species; RTK, receptor tyrosine kinases; SOS, son of sevenless; TAK, Tat-associated kinase; TLR, Toll-like receptor; TRAF, TNF receptor-associated factor.

Analytic Review

Multiple Organ Dysfunction Syndrome

Nicholas M. Gourd, BSc[1,2], and Nikitas Nikitas, MD, PhD[1]

Journal of Intensive Care Medicine
1-12

DOI: 10.1177/0885066619871452
journals.sagepub.com/home/jic
SAGE

PHYSIOPATHOLOGIE

Réaction inflammatoire inappropriée

libérations massives de cytokines pro-inflammatoires (IL-1), IL-6 et (TNF)

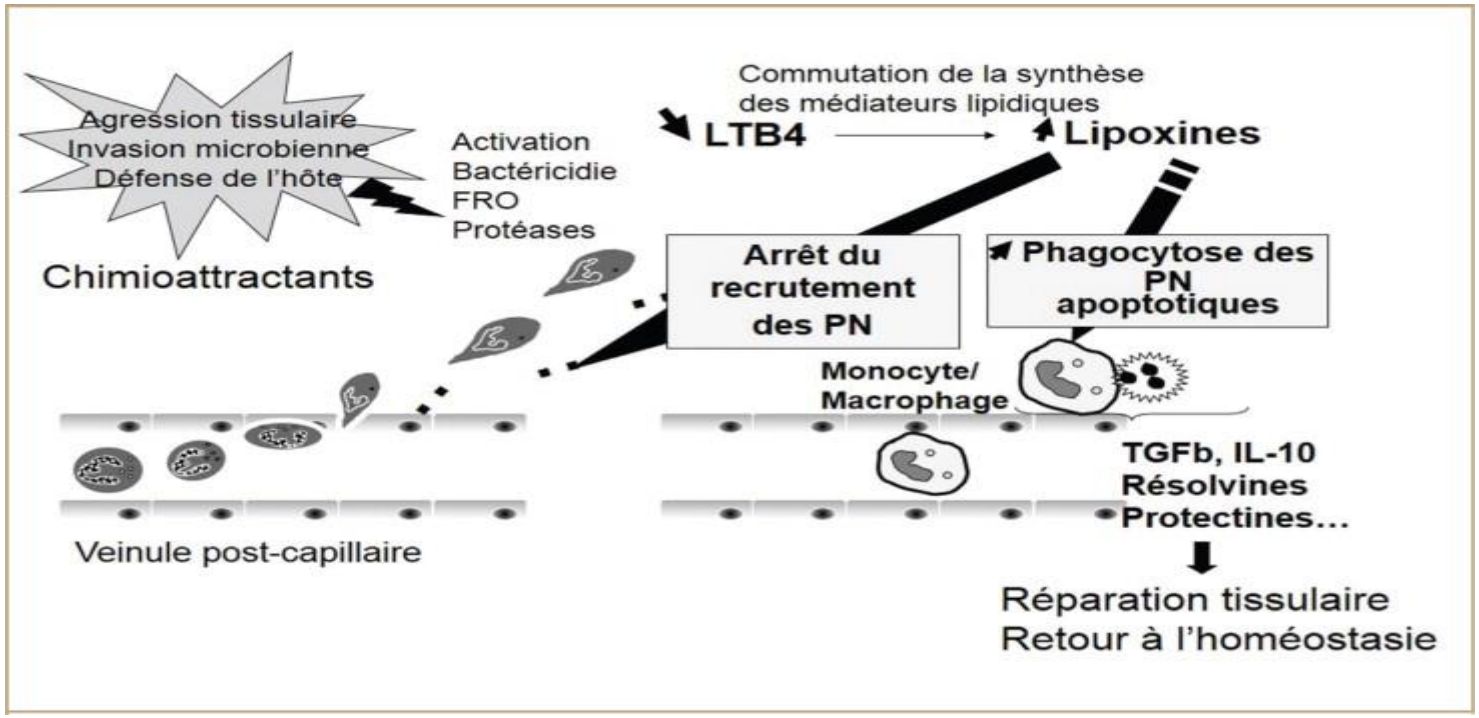
Médiateurs anti-inflammatoires IL-10 et le récepteur soluble au TNFα

Dépression immune

Médiateurs humoraux

PHYSIOPATHOLOGIE

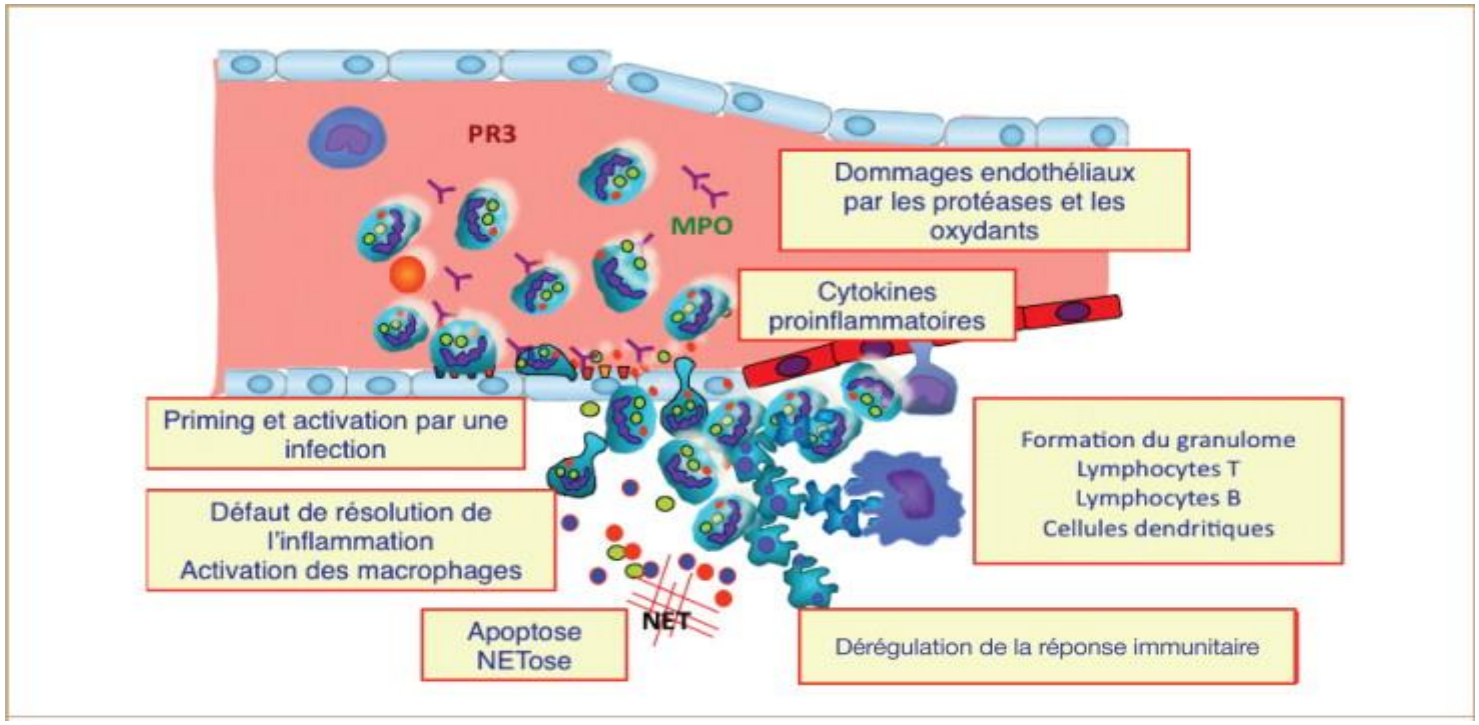
Réaction inflammatoire inappropriée



Médiateurs cellulaire

PHYSIOPATHOLOGIE

Réaction inflammatoire inappropriée



Médiateurs cellulaire

PHYSIOPATHOLOGIE

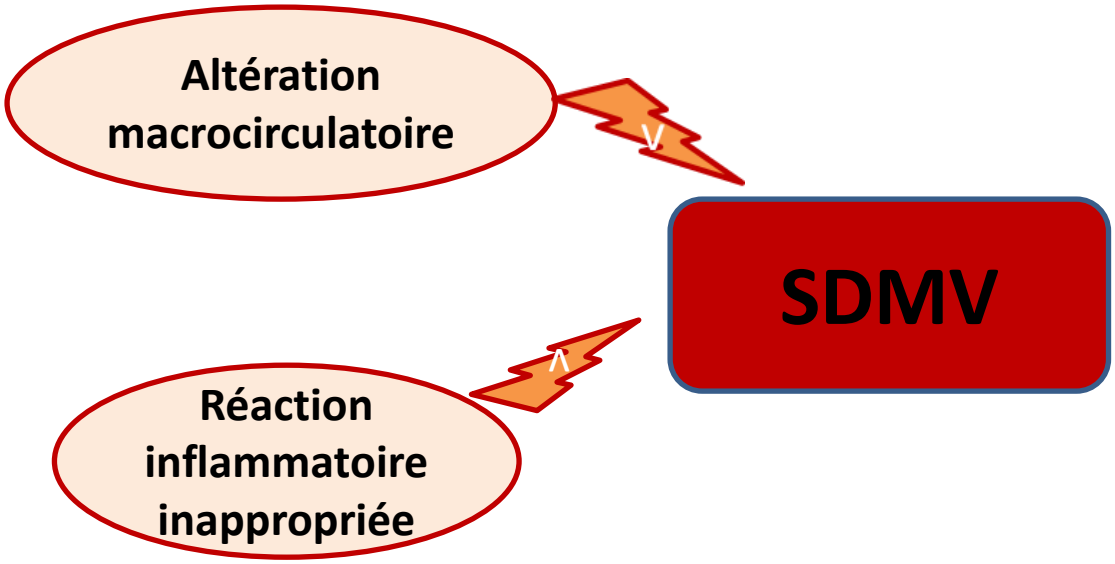
Réaction inflammatoire inappropriée

Activation dérégulée et disproportionnée des PNN et des macrophages

Apoptose retardée des PNN
Lésion endothéliale et vasculaire

PHYSIOPATHOLOGIE

HYPOTHESES PHYSIOLOGIQUES



PHYSIOPATHOLOGIE

Etat de choc

Défaut de perfusion cellulaire

Manque d'apport en O2

Hypoxie cellulaire

Souffrance cellulaire

PHYSIOPATHOLOGIE

Etat de choc

Redistribution de la perfusion

Rein/Peau/ Muscle/Intestin

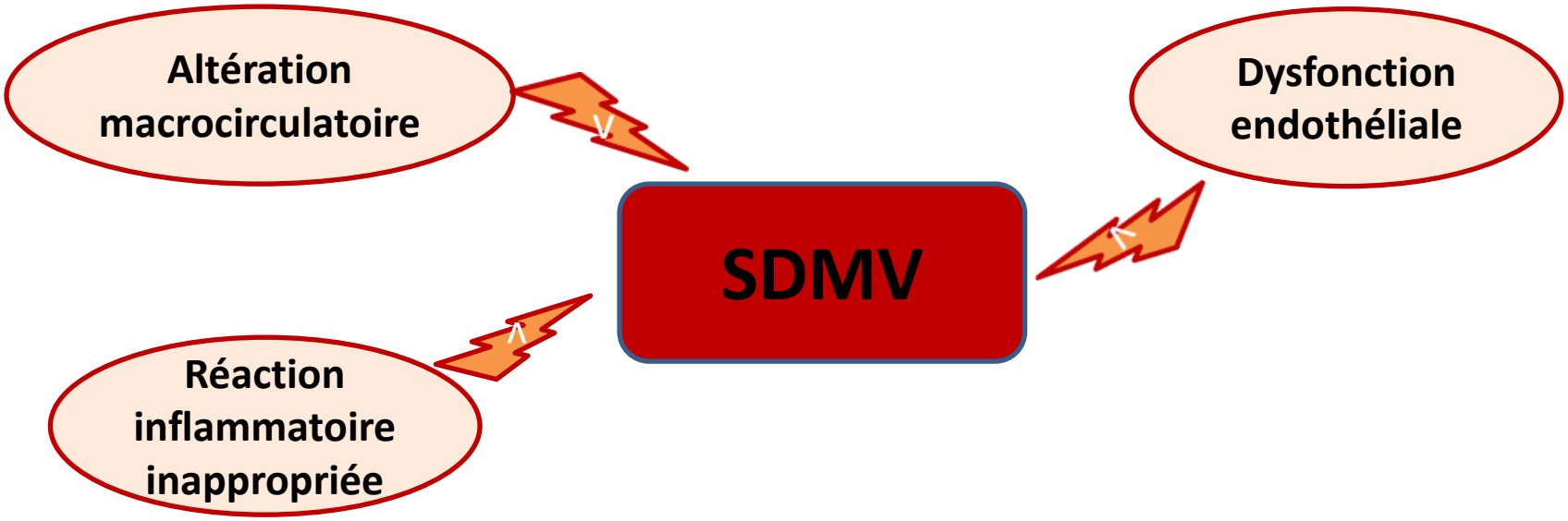
Cœur/cerveau

Souffrance tissulaire

Défaillance multivicérale

PHYSIOPATHOLOGIE

HYPOTHESES PHYSIOLOGIQUES



PHYSIOPATHOLOGIE

Intensive Care Med (2010) 36:1286–1298
DOI 10.1007/s00134-010-1893-6

REVIEW

H. Ait-Oufella
E. Maury
S. Lehoux
B. Guidet
G. Offenstadt

The endothelium: physiological functions and role in microcirculatory failure during severe sepsis

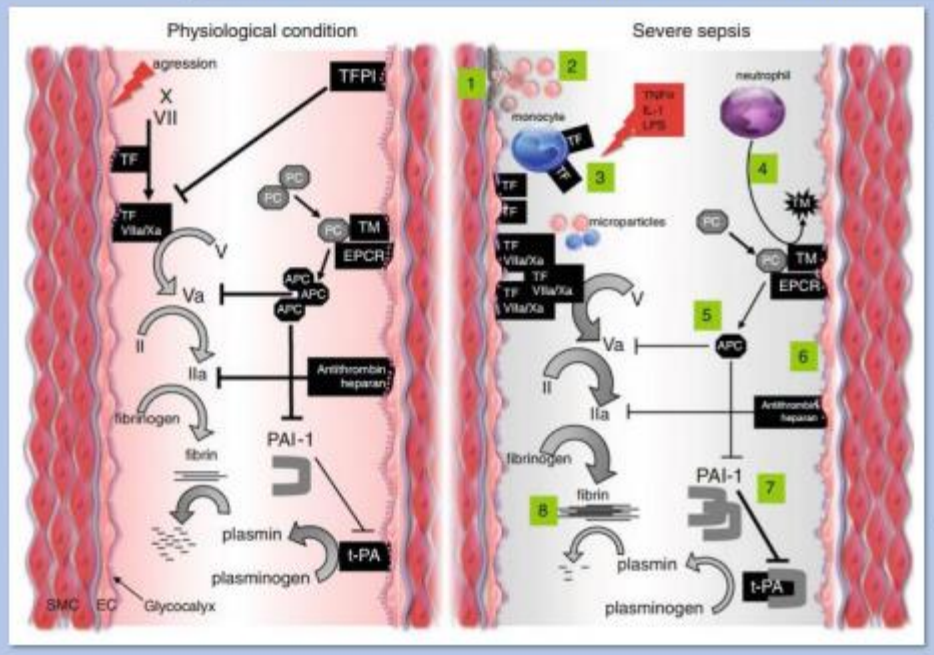
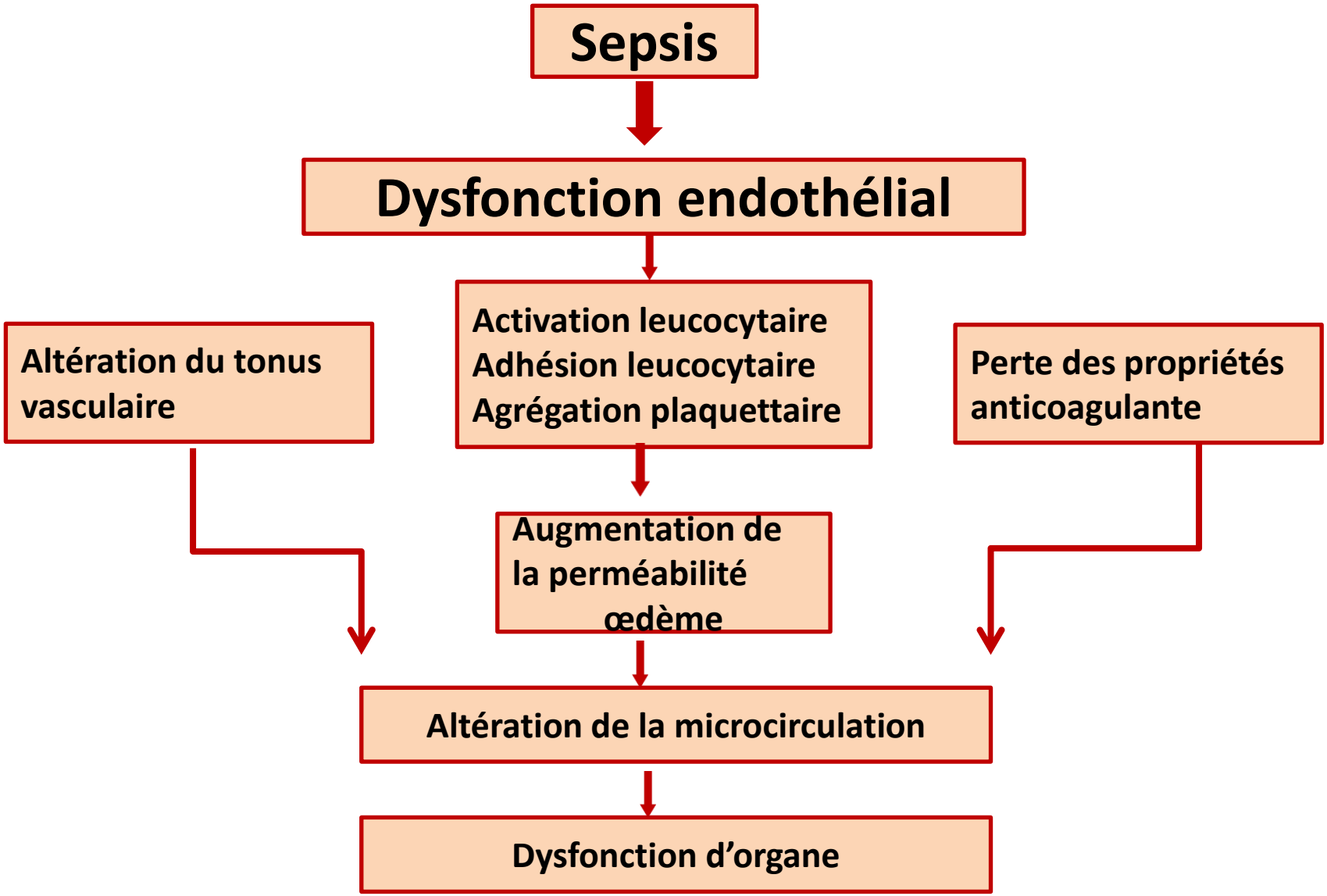


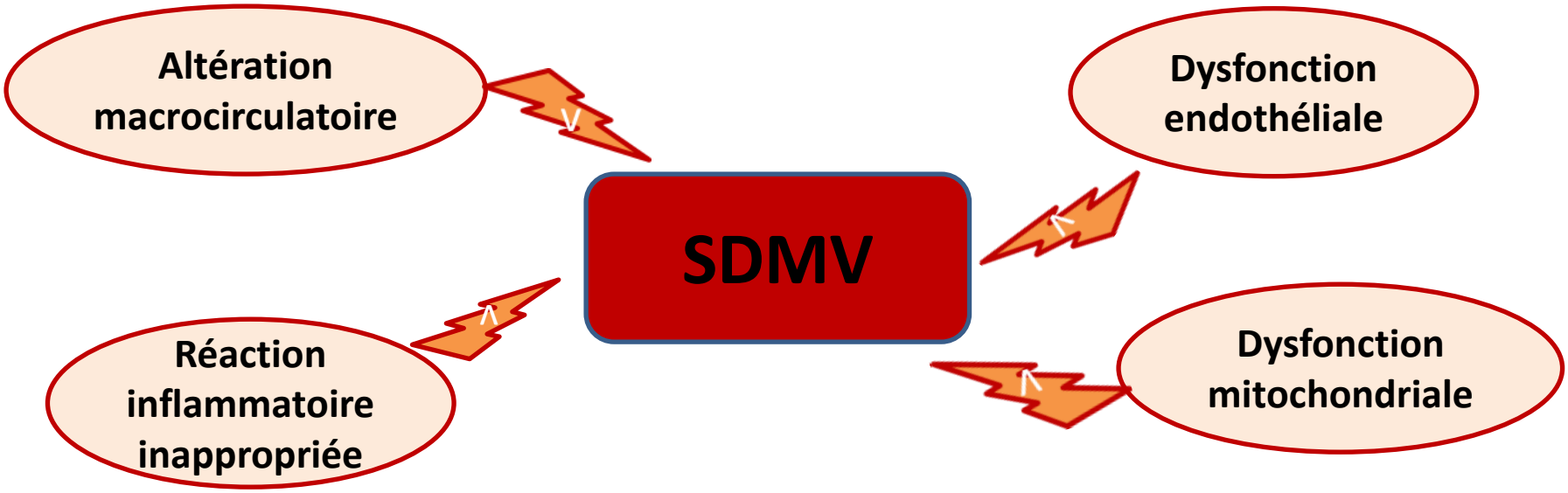
Fig. 1 Physiologically, after vascular aggression, endothelial cells express TF and initiate the coagulation cascade that leads to thrombin activation and fibrin deposition. At the same time, anticoagulant pathways and fibrinolysis are activated to avoid disseminated coagulation and limit fibrin accumulation. In severe sepsis, several disorders occur: endothelial cell apoptosis (*1*), microparticle shedding (*2*), procoagulant phenotype switch with TF expression (ECs, monocytes and microparticles) (*3*) and activation of the coagulation cascade. Alteration of anticoagulant pathways involve: TM degradation by neutrophil elastase (*4*), decreased APC levels (*5*), glycocalyx degradation (*6*), alteration of fibrinolytic pathways with increased PAI-1 activity (*7*) and consecutive fibrin accumulation (*8*)

PHYSIOPATHOLOGIE

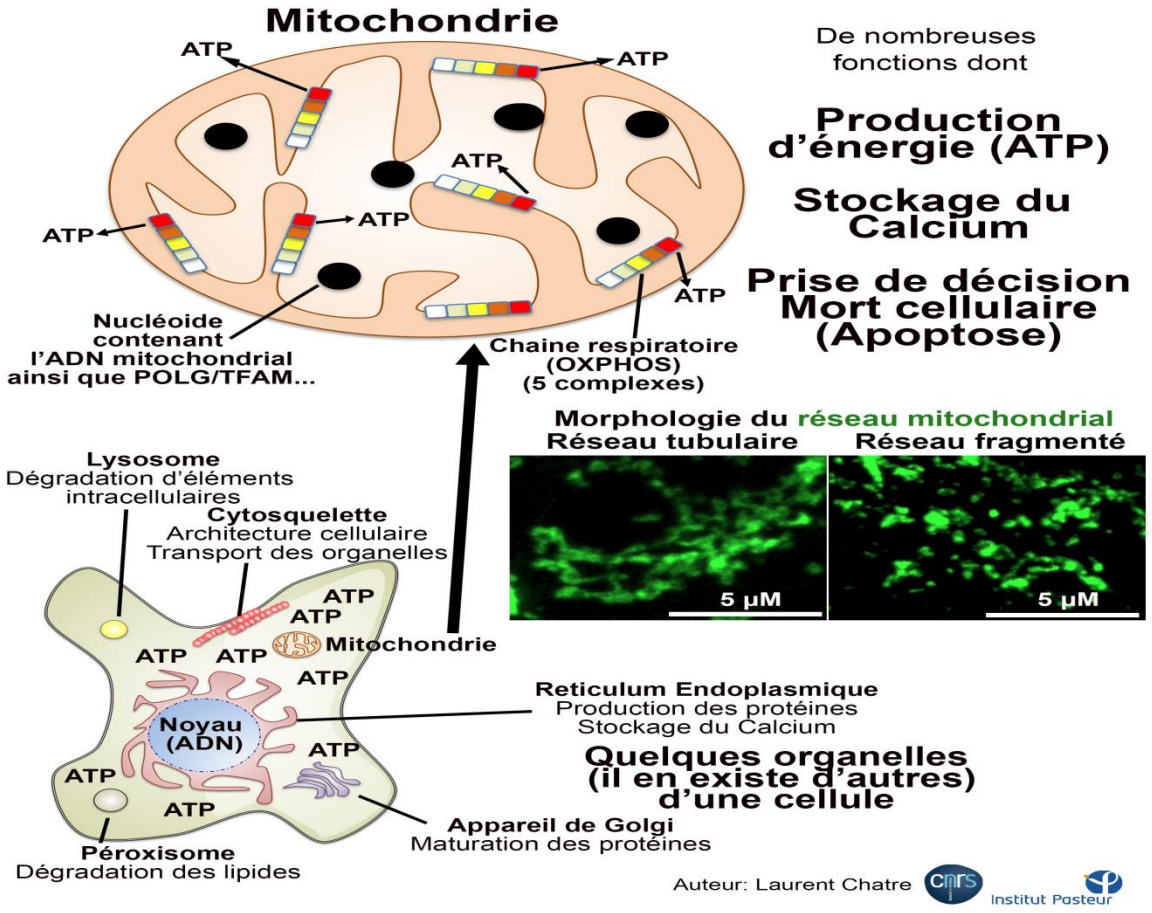


PHYSIOPATHOLOGIE

HYPOTHESES PHYSIOLOGIQUES



PHYSIOPATHOLOGIE



PHYSIOPATHOLOGIE

Dysfonction mitochondriale

WJCCM World Journal of Critical Care Medicine

Submit a Manuscript: https://www.f6publishing.com

World J Crit Care Med 2023 June 9; 12(3): 139-152

DOI: 10.5492/wjccm.v12.i3.139

ISSN 2220-3141 (online)

MINIREVIEWS

Sepsis-induced mitochondrial dysfunction: A narrative review

Wagner Nedel, Caroline Deutschendorf, Luis Valmor Cruz Portela

PHYSIOPATHOLOGIE

Sepsis

Altérations du métabolisme cellulaire

Difficulté intrinsèque de la cellule à utiliser l'O2

LA THEORIE DE LA CYTOPATHIE HYPOXIQUE

Bench-to-bedside review: Cytopathic hypoxia

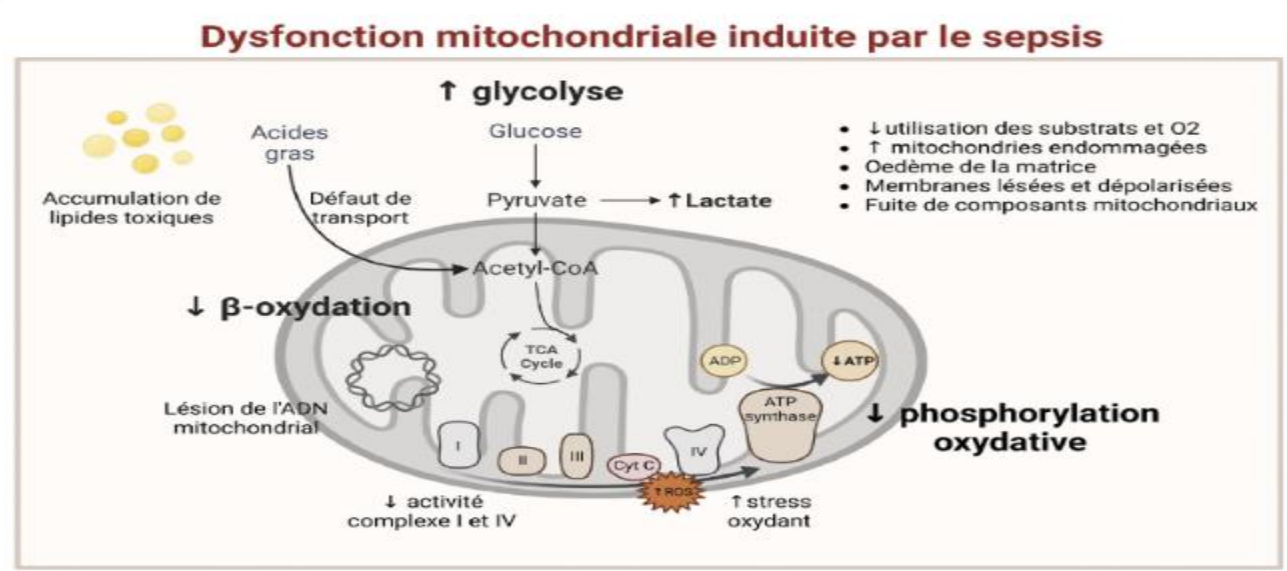
Mitchell P Fink

Professor and Chairman, Department of Critical Care Medicine, Watson Chair in Surgery, University of Pittsburgh Medical School, Pittsburgh, Pennsylvania, USA

Critical Care December 2002 Vol 6 No 6 Fink

PHYSIOPATHOLOGIE

Méd. Intensive Réa. 33(1):29-46



MISE AU POINT / *UPDATE*

RÉANIMATEUR-RICE ADULTE / *INTENSIVIST*

Dysfonction énergétique au cours du sepsis

Energetic dysfunction in sepsis

Alexandre Pierre[1,2*] • Raphael Favory[1,2] • Benoit Brassart[2] • Arthur Durand[2] • Claire Bourel[2] • Steve Lancel[1] Sébastien Préau[1,2]

Reçu le 2 février 2023 ; accepté le 11 septembre 2023.

PHYSIOPATHOLOGIE

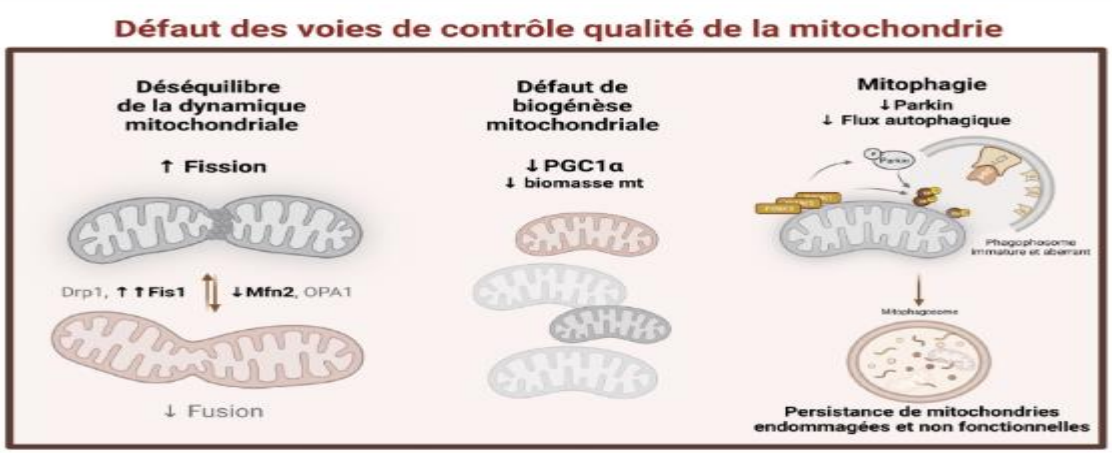


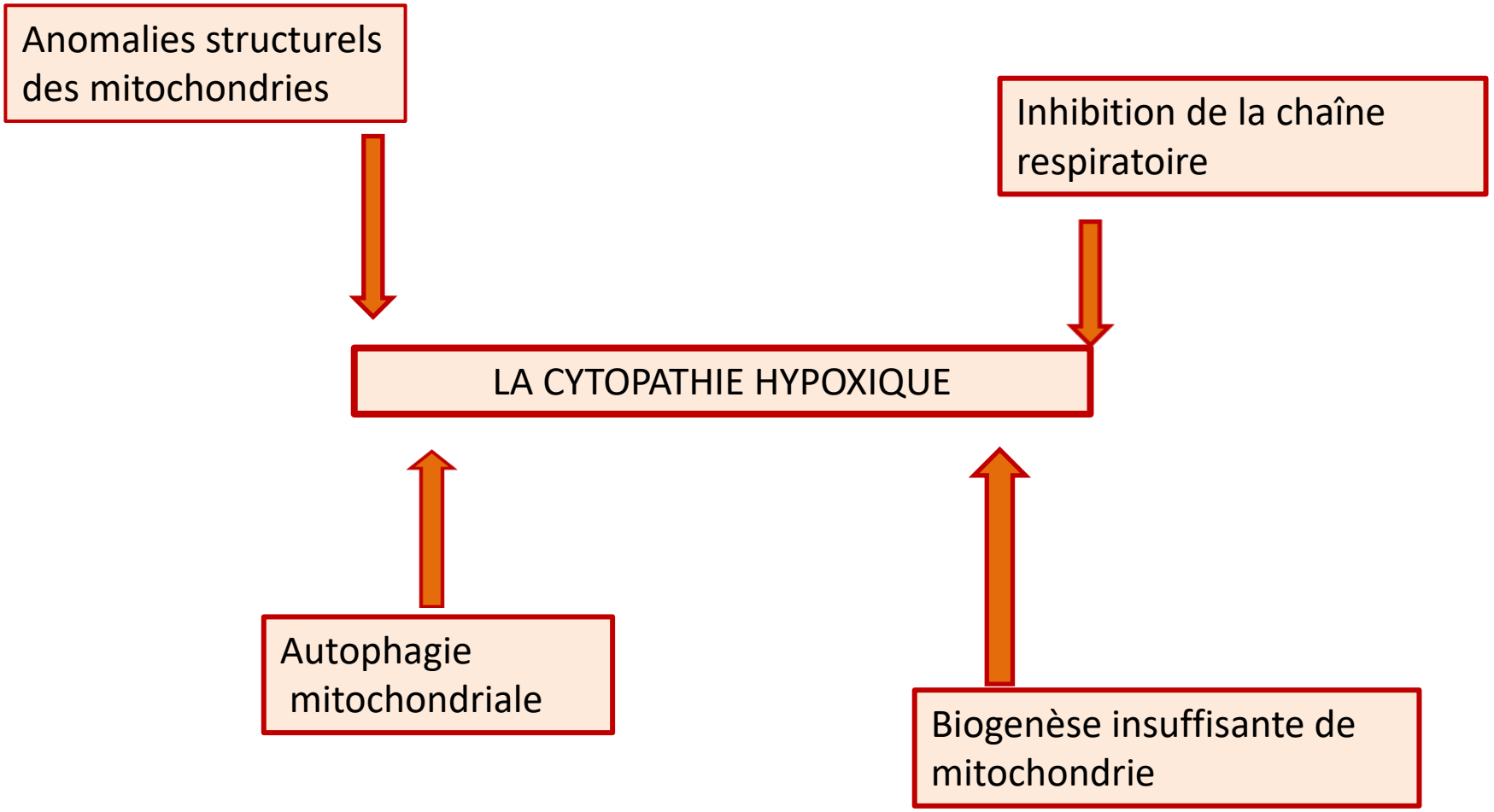
FIGURE 6 – Altérations mitochondriales au cours du sepsis.

Le panel du haut résume les caractéristiques fonctionnelles et morphologiques de la population mitochondriale.

Le panel du bas résume les anomalies des voies de contrôle qualité de la mitochondrie.

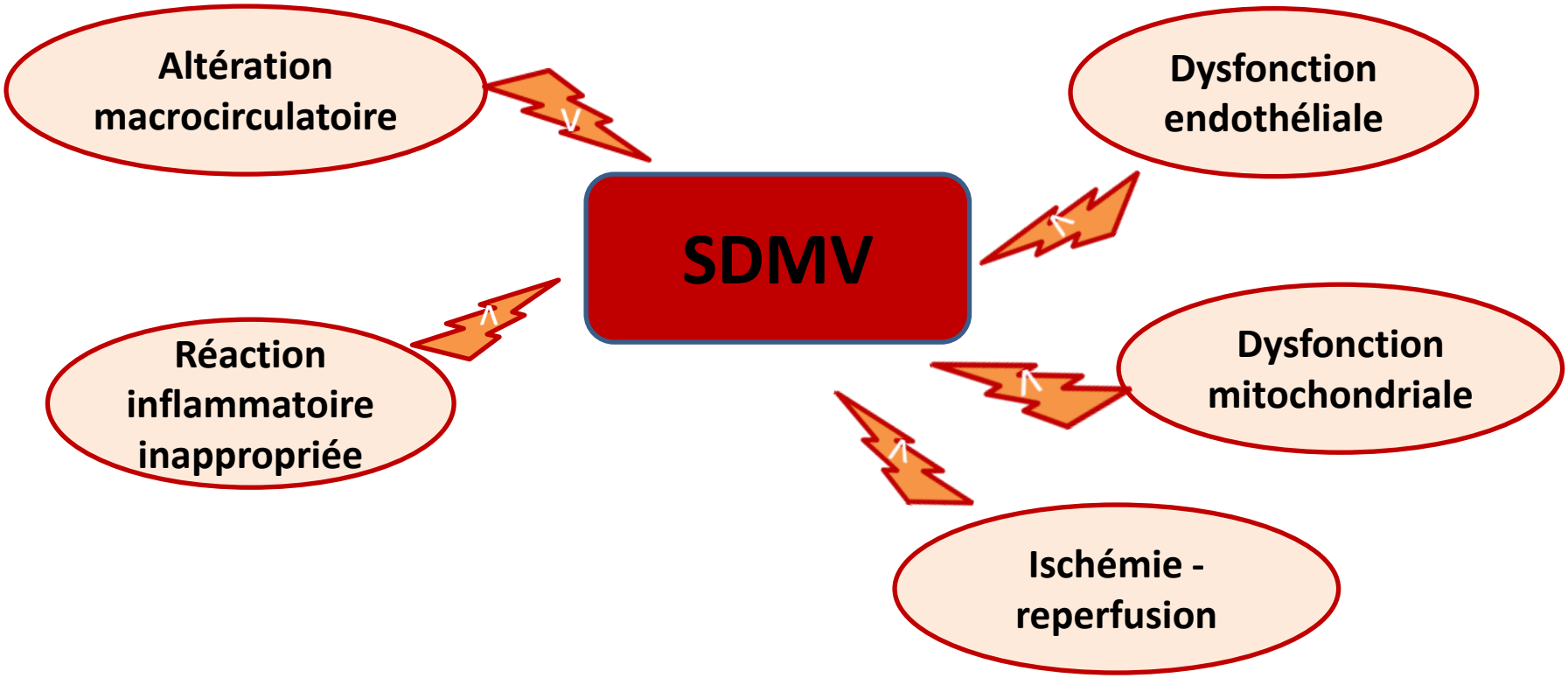
Cyt c : cytochrome c ; ROS : reactive oxygen species pour espèces réactives de l'oxygèn ; Drp1 : Dynamin-related protein 1 ; Fis1 : mitochondrial Fission 1 protein ; Mfn2 : Mitofusin 2, OPA1 : optic atrophy 1 ; PGC1α : Peroxisome proliferator-activated receptor gamma coactivator 1-alpha ; PINK : PTEN-induced kinase 1 ; LC3 : microtubule associated protein 1 Light Chain 3.

PHYSIOPATHOLOGIE



PHYSIOPATHOLOGIE

HYPOTHESES PHYSIOLOGIQUES



PHYSIOPATHOLOGIE

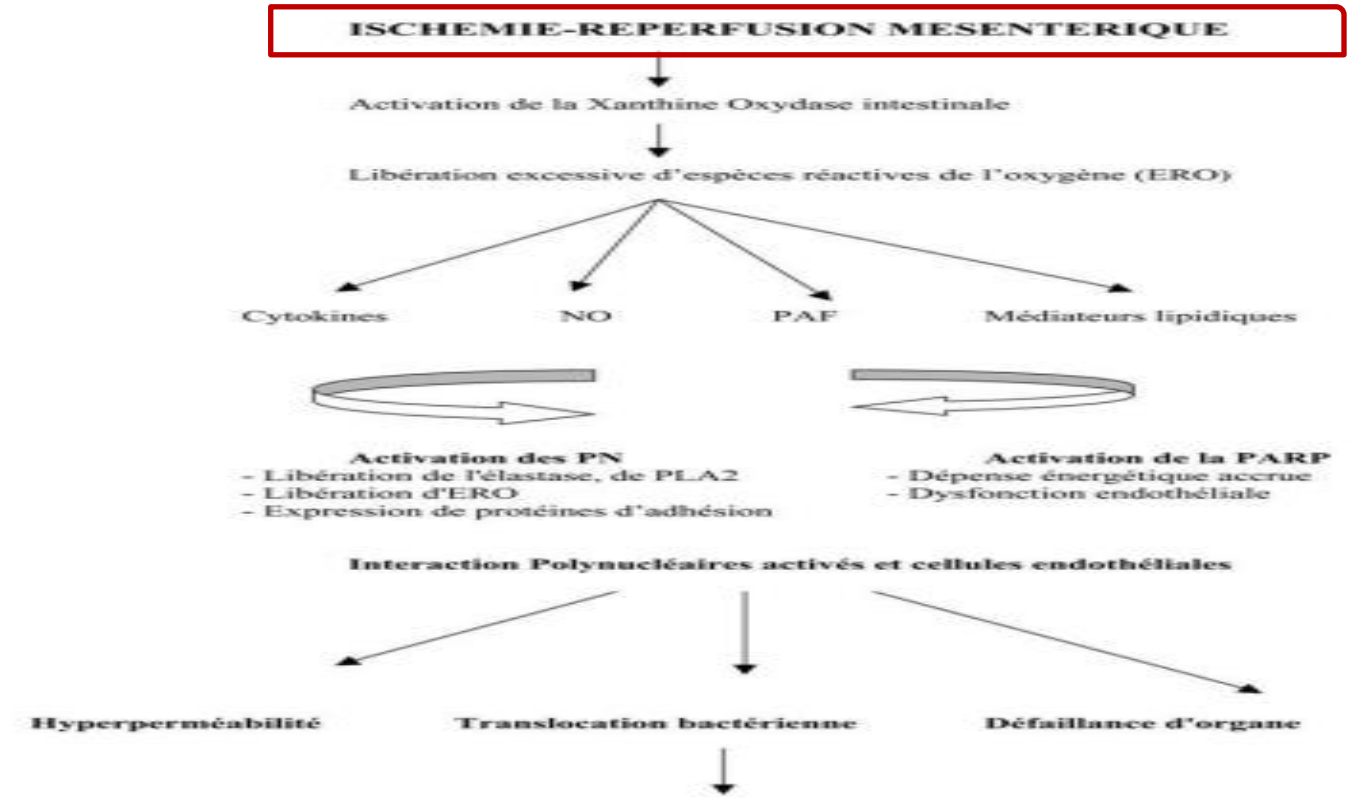


Fig. 1. Mécanismes physiopathologiques de l'ischémie–reperfusion mésentérique.
NO, monoxide d'azote ; PAF, *platelet activator factor* ; PLA2, phospholipase A2 ; PARP, la poly (ADN-ribosome) polymérase ; ERO, espèces réactives de l'oxygène.

ELSEVIER

Available online at www.sciencedirect.com
SCIENCE DIRECT®
Réanimation 12 (2003) 441–448

Réanimation
www.elsevier.com/locate/reaurg

Mise au point

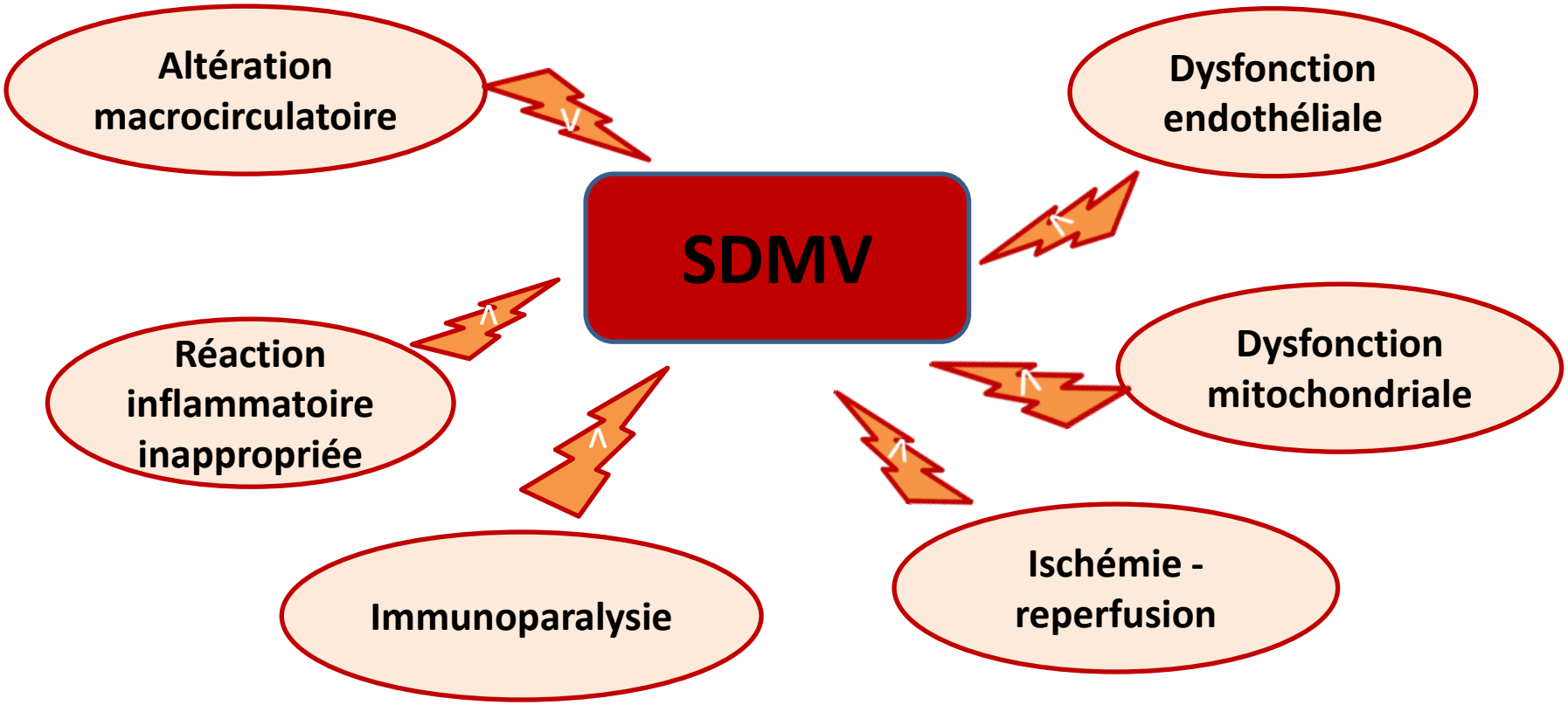
Ischémie–reperfusion mésentérique lors des états de choc : principaux aspects physiopathologiques

Splanchnic ischemia–reperfusion in shock: pathophysiology

F. Tamion *, K. Clabault, G. Bonmarchand
Service de réanimation médicale, hôpital Charles-Nicolle, 1, rue de Germont, 76031 Rouen, France
Reçu le 20 mai 2003 ; accepté le 22 mai 2003

PHYSIOPATHOLOGIE

HYPOTHESES PHYSIOLOGIQUES



PHYSIOPATHOLOGIE

Réponse anti-inflammatoire «compensatrice»

Immunosuppression
(Immunoparalysie)

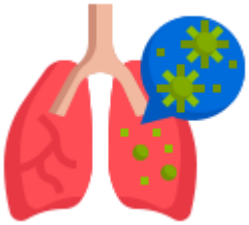
Réduction majeure des fonctions des phagocytes, la désactivation des monocytes

Apoptose massive des lymphocytes et des cellules dendritiques

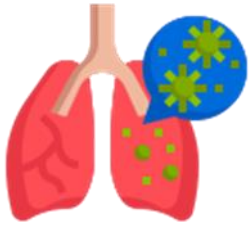
Entravant l'élimination de l'infection primaire

Favorisant des infections nosocomiales secondaires

La réactivation de pathogènes dormants,




Multiple
Organ
Dysfunction
Syndrome



PHYSIOPATHOLOGIE

Médiateurs de l'inflammation

➔ Atteinte vasculaire

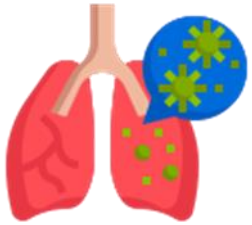
➔ Atteinte endothéliale

⇩

Augmentation de la perméabilité capillaire

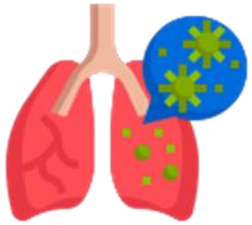
⇩

Œdème interstitiel



PHYSIOPATHOLOGIE

- Collapsus alvéolaire
- Anomalie de ventilation
- Thrombose vasculaire
- Effet shunt



CLINIQUE

- Insuffisance respiratoire
- Anomalie des échange gazeux (hypoxie)
- Atteinte alvéolaire à la radiographie
- SDRA

PHYSIOPATHOLOGIE

Médiateurs de l'inflammation

Atteinte mitochondriale et anomalie énergétique

Augmentation de la demande myocardique en O2

Diminution de la sensibilité adrénergique

↓

Dysfonction myocardique

Curr Heart Fail Rep. Author manuscript; available in PMC 2015 Jun 19.
Published in final edited form as:
Curr Heart Fail Rep. 2015 Apr; 12(2): 130–140.
doi: 10.1007/s11897-014-0247-z

PMCID: PMC447473
NIHMSID: NIHMS66637
PMID: 2547518

Pathophysiology of Sepsis-Related Cardiac Dysfunction: Driven by Inflammation, Energy Mismanagement, or Both?

Konstantinos Drosatos,✉ Anastasios Lymperopoulos, Peter Johannes Kennel, Nina Pollak, P. Christian Schulze, and Ira J. Goldberg

PHYSIOPATHOLOGIE

Médiateurs de l'inflammation

↓

Vasoplégie

↓

Augmentation de la perméabilité capillaire

↓

Diminution de la précharge

CLINIQUE

- Hypotension
- Signes d’hypoperfusion périphérique
- Tableau d’OAP
- Dosage des lactates
- Echocardiographie (fonction systolique et diastolique VG)

PHYSIOPATHOLOGIE

- Hypoperfusion
- Ischémie
- Translocation microbienne

CLINIQUE

- Ictère cutaméomuqueuse
- Hépatomégalie
- Insuffisance hepatocellulaire
- Biologie: cytolyse

  cholestase

  troubles de coagulation

CLINIQUE

- Cholécystite
- Ischémie mésentérique
- Translocation bactérienne
- Hémorragie digestive

PHYSIOPATHOLOGIE

- Atteinte tubulaire (proximal et distal)

- Multifactorielle :

  Médiateurs de l'inflammation

  Ischémie régionale

  Toxique

PHYSIOPATHOLOGIE

- Atteinte pré rénale et rénale
- Diminution de la perfusion rénale
- Activation du système rénine angiotensine

CLINIQUE

- Oligo-anurie
- Elévation de la créatinine de l’urée
- Ionogramme urinaire
- Trouble hydroélectrolytique
- Acidose métabolique
- Echographie rénale : éliminer une cause obstructive)

PHYSIOPATHOLOGIE

- Hypoperfusion cérébrale (état de choc)
- Encéphalopathie associée au sepsis
- Encéphalopathie hépatique

PHYSIOPATHOLOGIE

- **Encéphalopathie associée au sepsis**

✓ Médiateurs pro-inflammatoire

✓ Activation endothéliale

✓ Altération de la BHE

✓ Passage cérébral de facteurs neurotoxiques, notamment de médiateurs pro-inflammatoires

✓ Alteration de la Microcirculation

PHYSIOPATHOLOGIE

- **Encéphalopathie hépatique**

✓ L'accumulation toxiques (ammoniaque)

✓ Altération du transport de certains acides aminés (AA) à travers la barrière hémato-encéphalique

✓ Production anormale de substances benzodiazepine-like

CLINIQUE

- Alteration de l’état neurologique
- Convulsions
- Confusion
- Tableau d’encéphalopathie hépatique

PHYSIOPATHOLOGIE

Glucocorticoïdes et hormone de stress

Hypermétabolisme

Glycogénolyse

Néo-glycogénolyse

CLINIQUE

- Hyperglycémie
- Resistance à l’insuline
- Hypercatabolisme
- Fente musculaire

PHYSIOPATHOLOGIE

cytokines pro-inflammatoires

Endothélium vasculaire

Cascade de la coagulation

Excès de thrombine

Fibrinolyse retardée

PHYSIOPATHOLOGIE

Diminution des concentrations plasmatiques des anticoagulants naturels (protéine C, protéine S, et antithrombine)

↓

Formation de microthrombi

Immunothrombose

CLINIQUE

- Thrombopénie
- Hyperleucocytose
- Neutropénie
- CIVD

CLINIQUE

Signes cliniques de la CIVD

- Manifestations ischémiques :
  - nécrose des doigts ...
- Manifestations hémorragiques :
  - saignements aux points de ponctions
  - placards echymotiques
  - saignements en nappe

Signes biologiques CIVD :

- ↘ TP
- ↗ TCK
- ↘ facteurs de coag (fact V +++)
- ↗ DDimères et PDF
- ↘ AT III

CLINIQUE

CIVD scores diagnostiques

Paramètres	ISTH	JAAM 2006	JAAM-DIC
	-	**SIRS** [0] < 3 [1] ≥ 3	**ATIII** [0] > 70% [1] < 70%
Plaquettes (G/L)	[0] > 100 [1] 50-99 [2] < 50	[0] > 120 [1] 80-119 ou -30% [3] < 80 ou -50%	[0] > 120 [1] 80-119 ou -30% [3] < 80 ou -50%
Taux de prothrombine (%)	[0] > 64 [1] 35-64 [2] < 35	[0] > 64 [1] < 64	[0] > 64 [1] < 64
Fibrinogène (g/L)	[0] > 1,0 [1] < 1,0	-	-
D-dimères (µg/mL)	[0] < 0,5 [2] 0,5-4,0 [3] > 4,0	[0] < 5,0 [1] 5,0-15,0 [3] > 15,0	[0] < 5,0 [1] 5,0-15,0 [3] > 15,0
Diagnostic de CIVD	**Score ≥ 5/8**	**Score ≥ 4/8**	**Score ≥ 4/8**

Tableau 1 : Scores diagnostiques de la coagulation intravasculaire disséminée [18-20].

Diagnostic

- Scores diagnostiques et pronostiques
- Clinique
- Explorations

Diagnostic

Score SOFA

> Intensive Care Med. 1996 Jul;22(7):707-10. doi: 10.1007/BF01709751.

The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine

J L Vincent [1], R Moreno, J Takala, S Willatts, A De Mendonça, H Bruining, C K Reinhart, P M Suter, L G Thijs

Diagnostic

Scoring system	Description	Parameters	Allotted scores to each parameter
SOFA score (Sequential Organ Failure Assessment) [31]	It is derived from the extent of dysfunction of six different organ systems to predict mortality. Evaluation: On admission & reassessed every 24 h until the patient is discharged utilizing the worst parameters computed during the initial 24 hours. Implications: Development of organ dysfunction can be deduced by the individual scores for each organ. Additionally, as the aggregate of scores on one ICU day and through the whole period of ICU stay, as the aggregate of the worst scores.	• Respiratory (PaO2/FIO2 in mmHg) • Coagulation (platelet count x103/μL) • Hepatic (bilirubin in μmol/L) • Cardiovascular mean arterial pressure or administration of vasoactive agents (dopamine, epinephrine and norepinephrine in μg/kg/min) • Renal (creatinine μmol/L) • Neurological (GCS)	0–4

Diagnostic

QUICK SOFA

qSOFA score [31]	Quick SOFA used in noncritical care setting at the bedside. Patients >18 years of age with suspicion or confirmed infection with increased risk of in-hospital mortality or prolonged ICU stay (>3 days). It is not a diagnostic tool for sepsis. This score is a predictor of mortality (score of 01: low risk of in-hospital mortality; score 23: high risk). Interpretation: a positive score warrants the evaluation of a SOFA score to confirm the presence of sepsis.	• Altered mental status(GCS < 15) • Respiratory rate > or equal to 22/min • Systolic blood pressure < or equal to 100 mmHg	0–1

Diagnostic

Marshall's MOD score

Marshall's MOD score [33]	Simple physiologic measures of dysfunction in six organ systems, which correlated well with the eventual risk of ICU and hospital mortality.	• Respiratory(PaO2/FIO2 in mmHg) • Renal(creatinine in µmol/L) • Hepatic (bilirubin in µmol/L) • Cardiovascular mean arterial pressure or administration of vasoactive agents required (dopamine, epinephrine, and norepinephrine in µg/kg/min) • Coagulation (platelet count x103/µL) • Neurological (GCS)	0–4 OR presence of organ dysfunction/failure

Review > Crit Care Med. 1995 Oct;23(10):1638-52. doi: 10.1097/00003246-199510000-00007.

Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome

J C Marshall [1], D J Cook, N V Christou, G R Bernard, C L Sprung, W J Sibbald
Affiliations + expand
PMID: 7587228 DOI: 10.1097/00003246-199510000-00007

Diagnostic

PIRO score

Predisposition, infection, response, and organ dysfunction (PIRO) score [35]	The PIRO system was developed for ED patients with suspected infection for bedside use at clinical presentation. The four components of this system include various known independent factors that may influence the onset, development, and outcome of sepsis.	• Predisposition (age, COPD, liver disease, nursing home resident, malignancy) • Infection (skin/soft tissue infection, any other infection, pneumonia) • Response [respiratory rate (bpm), bands, heart rate (bpm)] • Organ dysfunction [SBP (mmHg), BUN (mmol/l), respiratory failure/hypoxemia, lactate (mmol/l), platelet count (x109/l)]	0–4

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Risk stratification and prognostic performance of the predisposition, infection, response, and organ dysfunction (PIRO) scoring system in septic patients in the emergency department: a cohort study

Yun-Xia Chen[1] and Chun-Sheng Li[✉1]

Diagnostic

Clinique

- ✓ Insuffisance respiratoire
- ✓ Alteration hémodynamique
- ✓ Alteration neurologique
- ✓ Ictère
- ✓ Hémorragie

Diagnostic

Explorations

- ✓ Evaluation fonction rénale
- ✓ Evaluation du bilan hépatique
- ✓ Numération formule sanguine
- ✓ Bilan d'hémostate
- ✓ GDS
- ✓ Lactaes
- ✓ Echographie rénale et cardiaque


Thank
you