

D.U. THERAPEUTIQUE ANTI-INFECTIEUSE 2026

CHU BDX CENTRE HOSPITALIER UNIVERSITAIRE BORDEAUX

ImmunoConcEPT Inserm

université de BORDEAUX

Sepsis

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ImmunoConcEPT, Université Bordeaux

2017

47 - 50 millions des cas par an¹

Au moins **11 millions** de morts²

1 décès sur 5 dans le monde est lié au sepsis³

Le sepsis est la première cause de décès à l'hôpital⁴
cause de ré-hospitalisation⁵
cause des coûts de soins de santé⁶

Plus de **50%** des survivants du sepsis souffrent sur le long terme de problèmes physiques et/ou de problèmes psychologiques⁷

40% des cas sont des enfants de moins de cinq ans⁸

80% des cas de sepsis interviennent en dehors de l'hôpital⁹

LE SEPSIS est souvent causé par une infection telle que la pneumonie ou maladies diarrhéiques¹⁰

Le **SEPSIS** est une urgence médicale : si vous ou quelqu'un présente les signes de sepsis, consultez immédiatement un médecin en urgence ou appelez le numéro d'urgence ! Les heures sont comptées !¹¹

N Engl J Med. 2017 Aug 3;377(5):414-417
www.worldsepsisday.org

Définition

Si William Osler on sepsis: "Except on few occasions, the patient appears to die from the body's response to infection rather than from it."

Syndrome of infection complicated by acute organ dysfunction

400 B.C. (Sepsis) → Blood poisoning (1870-1904) → Sepsis-1 (1992) → Sepsis-2 (2003) → Sepsis-3 (2016)

Hippocrates uses the term sepsis meaning the process of decay or decomposition of organic matter during, when continuing fever is present, it is dangerous if the outer parts are cold, but the inner parts are burning hot.¹

Sepsis defined as a systemic inflammatory response syndrome (SIRS) to infection.²

Sepsis defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Septic shock is a subset of sepsis in which underlying respiratory and circulatory/metabolic abnormalities are profound enough to substantially increase mortality. Specific clinical criteria used to identify sepsis include a change in Sepsis-related Organ Failure Assessment Score (SOFA) ≥2 above baseline values, and for septic shock supressor requirements to maintain a mean arterial pressure ≥65 mmHg and a serum lactate >2 mmol/l in the absence of hypovolemia.³

SIRS (Systemic Inflammatory Response Syndrome) criteria

Temperature	> 38 or < 36°C
White blood cell count	> 12000 or < 4000/ml ³ or < 20% bands
Heart rate	> 90 beats/min
Respiratory rate	> 20 breaths/min or PaCO ₂ < 32 mm Hg

Immunity. 2021 Nov 9;54(11):2450-2464. doi: 10.1016/j.immuni.2021.10.012.

Définition

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)

Mervyn Singer, MD, FRCP; Clifford S. Deutschman, MD, MSc; Christopher Warren, Seymour, MD, MSc; Marcio Shankar-Hari, MSc, MD, FFICM; Djillali Annane, MD, PhD; Michael Bauer, MD; Ronald Bellomo, MD; Gordon B. Bernard, MD; Jean-Daniel Chiche, MD, PhD; Cheng M. Cooperwelle, MD; Richard C. Cook, MD; Mitchell M. Levy, MD, MSc; John C. Marshall, MD, MSc; Marzio Moles, MD, MSc; Steven M. Opal, MD; Gordon D. Rubenfeld, MD, MSc; Tom van der Poll, MD, PhD; Jean-Louis Vincent, MD, PhD; Derrick C. Angus, MD, MPH

- Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection.
- Organ dysfunction can be identified as an acute change in total SOFA score ≥2 points consequent to the infection.

	3	4			
PaO ₂ /F _i O ₂ , mm Hg (S _i DF)	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support
Coagulation Platelets, ×10 ³ /μL	≥150	<150	<100	<50	<20
Liver Bilirubin, mg/dL (μmol/L)	<1.2 (20)	1.2-1.9 (20-32)	2.0-5.9 (33-101)	6.0-11.9 (102-204)	>12.0 (204)
Cardiovascular MAP ≥70 mm Hg	MAP ≥70 mm Hg	MAP <70 mm Hg	Dopamine <5 or dobutamine (any dose) ^a	Dopamine 5.1-15 or epinephrine <0.1 or norepinephrine <0.1 ^b	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1 ^b
Central nervous system Glasgow Coma Scale score ^c	15	13-14	10-12	6-9	<6
Renal Creatinine, mg/dL (μmol/L)	<1.2 (110)	1.2-1.9 (110-170)	2.0-3.4 (171-299)	3.5-4.9 (300-440)	>5.0 (440)
Urine output, mL/d			<500	<200	

JAMA. 2016;315(8):801-810. doi:10.1001/jama.2016.0287

Définition

System	Score	0	1	2	3	4
Respiration PaO ₂ /F _i O ₂ , mm Hg (kPa)	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support	
Coagulation Platelets, ×10 ³ /μL	≥150	<150	<100	<50	<20	
Liver Bilirubin, mg/dL (μmol/L)	<1.2 (20)	1.2-1.9 (20-32)	2.0-5.9 (33-101)	6.0-11.9 (102-204)	>12.0 (204)	
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Central nervous system Glasgow Coma Scale score ^c	15	13-14	10-12	6-9	<6	
Renal Creatinine, mg/dL (μmol/L)	<1.2 (110)	1.2-1.9 (110-170)	2.0-3.4 (171-299)	3.5-4.9 (300-440)	>5.0 (440)	
Urine output, mL/d				<500	<200	

JAMA. 2016;315(8):801-810. doi:10.1001/jama.2016.0287

Définition

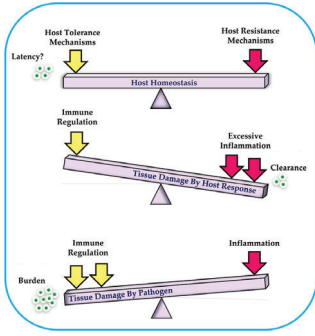
Quick SOFA score Criteria

- Respiratory rate ≥22/min
- Altered mentation
- Systolic blood pressure ≤100 mm Hg

JAMA. 2016;315(8):801-810

Définition → Physiopathologie

Tolérance



Résistance

Front Immunol. 2019 Jan 29;10:66. doi: 10.3389/fimmu.2019.00066

Définition → Physiopathologie

	Pro-inflammatory immune response	Counter-regulatory immune response
Beneficial effects	<ul style="list-style-type: none"> Local activation of defensins and cytokines Activation of phagocytosis and killing of cells Local endothelial activation for increased cell recruitment 	<ul style="list-style-type: none"> Regulation and control of inflammation Increase in tissue repair
Deleterious effects	<ul style="list-style-type: none"> Systemic cytokine release with endothelial activation and hypotension Systemic activation of complement Disseminated intravascular coagulation Organ dysfunction 	<ul style="list-style-type: none"> Too strong inhibition of antimicrobial mechanisms Immunosuppression Opportunistic infections

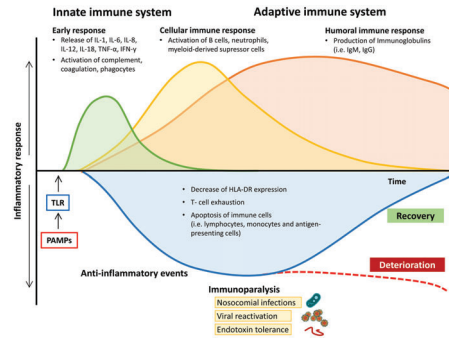
Nature Immunology, Giamarellos-Bourboulis et al, 2024

Définition → Physiopathologie

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Nature Immunology, Giamarellos-Bourboulis et al, 2024

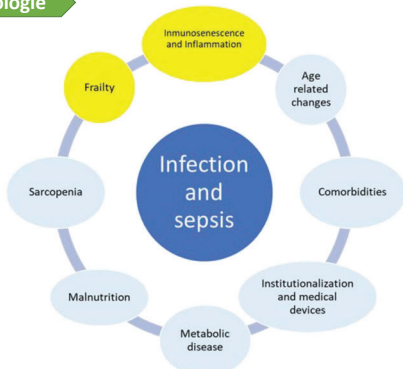
Définition → Physiopathologie



Front. Med. 8:628302. doi: 10.3389/fmed.2021.628302

Définition → Physiopathologie

- Score de fragilité (index de Charlson)
- « Inflammaging »
- Réduction des réserves physiologiques et métaboliques associée au vieillissement



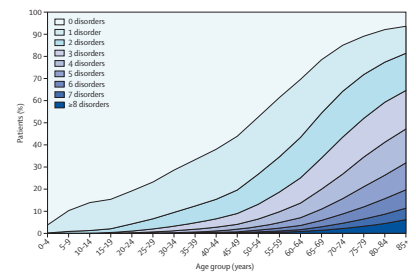
Ann Intensive Care. 2024 Jan 10;14(1):6

Définition → Physiopathologie

Epidemiology of multimorbidity and implications for health care, research, and medical education: a cross-sectional study

Karen Barnett, Stewart W Meace, Michael Nashroy, Graham Witt, Sally Wyke, Bruce Guthrie

- 78 % des patients en sepsis présentent au moins une comorbidité
- 60 % en présentent 3 ou +
- Les patients de 65 à 84 ans présentent 2,6 ± 2,2 comorbidités
- Ceux âgés de >85 ans en présentent 3,6 ± 2,3

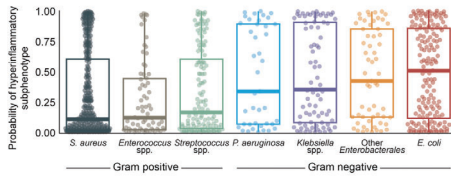


Lancet. 2012 Jul 7;380(9836):37-43

Définition Physiopathologie

Pathogen characteristics are key determinants of distinct host response phenotypes of sepsis

Rohit Choudhry, Bharat Bhatia, Kathleen A. Singer, Mohamad H. Taha, Michael W. Spolnik, Ying He, Mark Nagappa, Kirk S. Braggs, Mark D. Adams, Sunny S. Liu, Y. Eric Kenchappa, Matthew W. Churpek, Carole S. Calfee, Sandhya Toppo, Debra M. Foster, John A. Kalam, Robert P. D'Antonio, Frank Sista



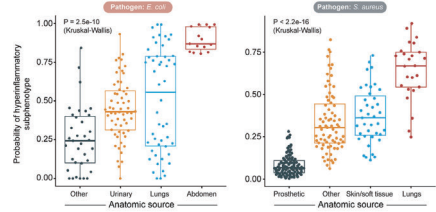
Among 2,108 bacteremic patients with sepsis, the hyperinflammatory subphenotype was strongly predicted by the **identity of the pathogen**, specifically gram-negative members of the Enterobacteriales order (*E. coli*, *Klebsiella* spp.).

J Clin Invest. 2026;136(6):e197346

Définition Physiopathologie

Pathogen characteristics are key determinants of distinct host response phenotypes of sepsis

Rohit Choudhry, Bharat Bhatia, Kathleen A. Singer, Mohamad H. Taha, Michael W. Spolnik, Ying He, Mark Nagappa, Kirk S. Braggs, Mark D. Adams, Sunny S. Liu, Y. Eric Kenchappa, Matthew W. Churpek, Carole S. Calfee, Sandhya Toppo, Debra M. Foster, John A. Kalam, Robert P. D'Antonio, Frank Sista



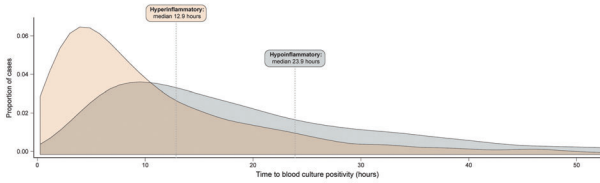
Among patients with the same species of pathogen (*E. coli* [n = 161], *S. aureus* [n = 240]), the hyperinflammatory subphenotype was strongly predicted by **anatomic site of initial infection**

J Clin Invest. 2026;136(6):e197346

Définition Physiopathologie

Pathogen characteristics are key determinants of distinct host response phenotypes of sepsis

Rohit Choudhry, Bharat Bhatia, Kathleen A. Singer, Mohamad H. Taha, Michael W. Spolnik, Ying He, Mark Nagappa, Kirk S. Braggs, Mark D. Adams, Sunny S. Liu, Y. Eric Kenchappa, Matthew W. Churpek, Carole S. Calfee, Sandhya Toppo, Debra M. Foster, John A. Kalam, Robert P. D'Antonio, Frank Sista



Time to culture positivity, which is inversely correlated with blood bacterial burden, was shorter among patients in the hyperinflammatory subphenotype (n = 2,108).

J Clin Invest. 2026;136(6):e197346

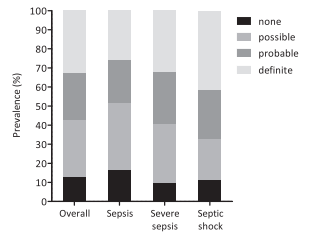
Définition Physiopathologie

Likelihood of infection in patients with presumed sepsis at the time of intensive care unit admission: a cohort study

Peter M. C. Klein Klouwenberg^{1,2,3}, Olaf L. C. Cremer¹, Laurette A. van Vught¹, David S. Y. Ong^{1,2,3}, Jos F. Frencken^{1,3}, Marcus J. Schultz², Marc J. Bonten^{1,2} and Tom van der Poll¹

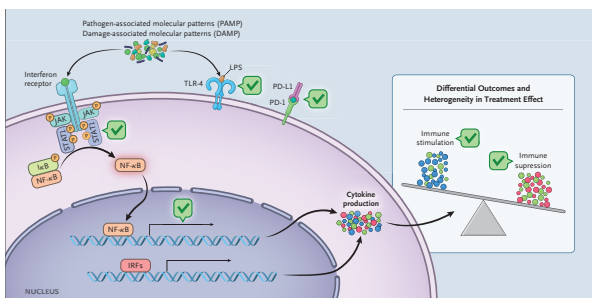


Un tiers ou plus des patients diagnostiqués comme sepsis ont en réalité une ou des défaillances d'organe d'origine non infectieuse



Klein Klouwenberg et al. Critical Care (2015) 19:319

Définition Physiopathologie



October 25, 2022 NEJM Evid 2022; 1 (11) DOI: <https://doi.org/10.1056/EVID2020118>

Définition Physiopathologie

Cytokine Storm

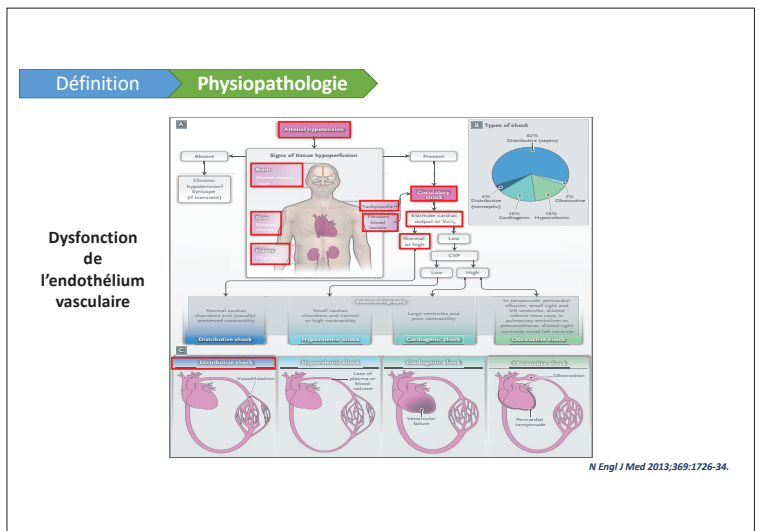
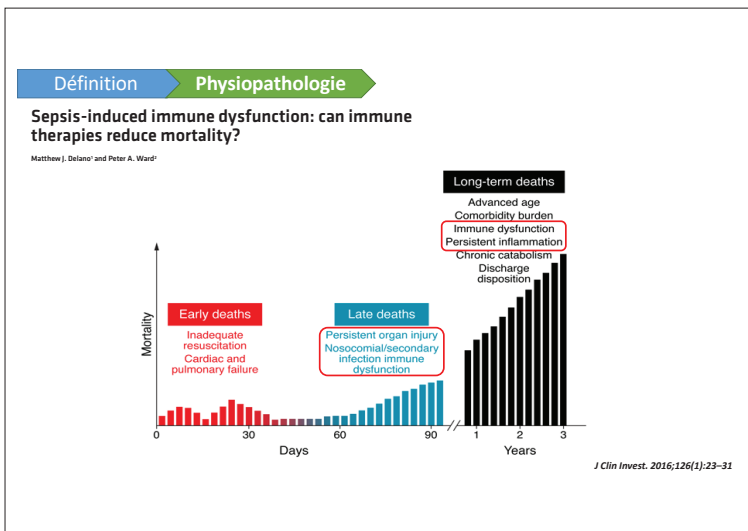
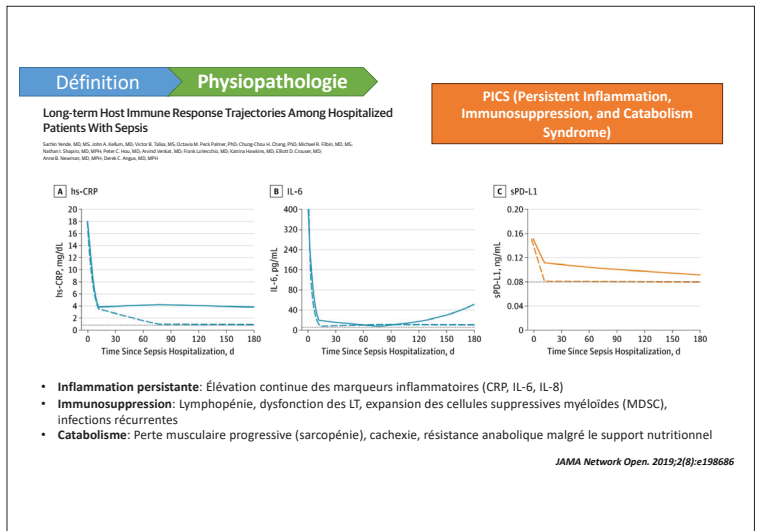
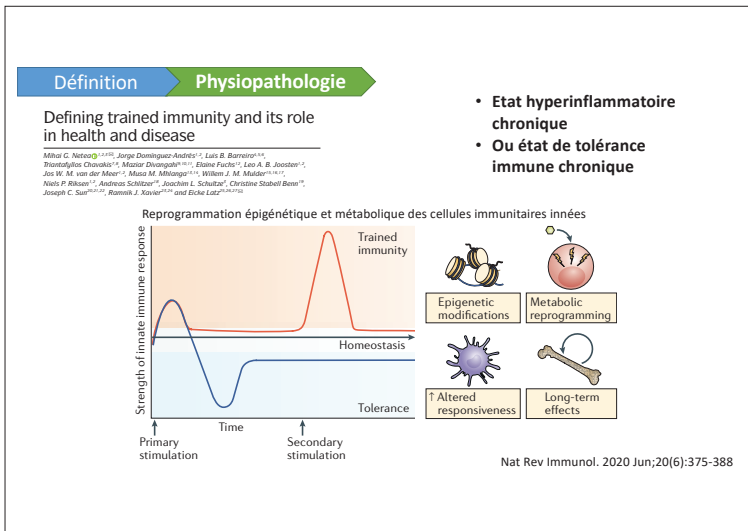
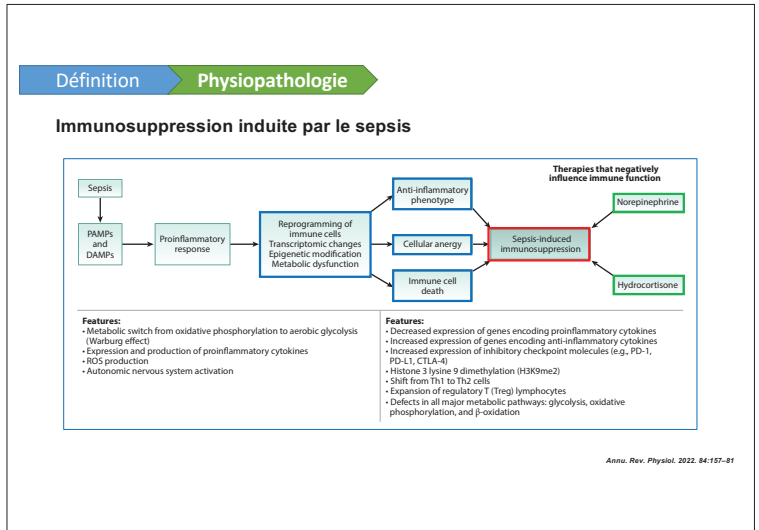
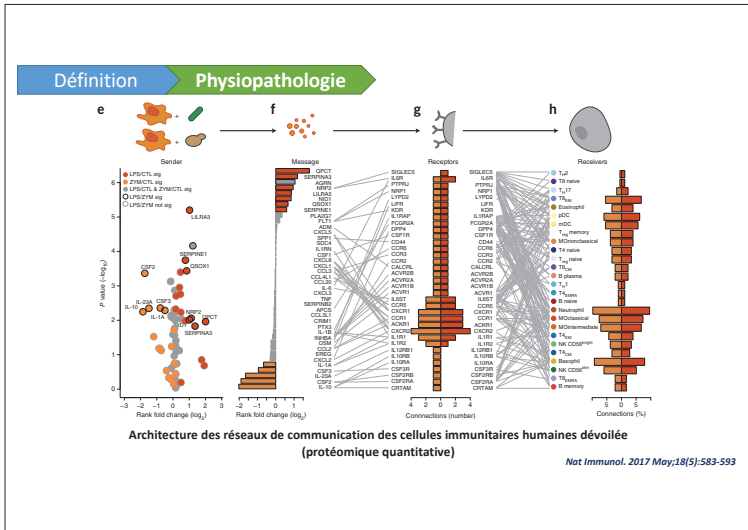
David C. Fajgenbaum, M.D., and Carl H. June, M.D.

THE NEW ENGLAND JOURNAL OF MEDICINE

Mediator	Main Cell Source	Type and Function	Chemokine	Function	
Interleukin-1	Macrophages, epithelial cells, pyroptotic cells	Proinflammatory alarm cytokine; pyrogenic function, macrophage and Th1 cell activation	Interleukin-4 (CXCL12)	Recruitment of neutrophils	
Interleukin-2	T cells	Effector T cell and regulatory T cell growth factor	MIP-1α (CXCL3)	Interleukin-4-dependent recruitment of Th1 cells, NK cells, plasmacytoid dendritic cells, NK cells	
Interleukin-6	Macrophages, T cells, endothelial cells	Proinflammatory cytokine; pyrogenic function, increased antibody production, induction of acute-phase reactants	MIP-1β (CXCL4)	Interleukin-4-dependent recruitment of macrophages, Th1 cells, NK cells	
Interleukin-9	Th9 cells	Protection from helminth infections, activation of mast cells, association with type 1 (inflammation in Covid-19)	MIP-1β (CXCL4)	Recruitment of Th1 cells, monocytes, dendritic cells, basophils, eosinophils	
Interleukin-10	Regulatory T cells, Th1 cells	Anti-inflammatory cytokine; inhibition of Th1 cells and cytokine release	MIP-1β (CXCL4)	Recruitment of macrophages, Th1 cells, NK cells, eosinophils, dendritic cells, progenitor function	
Interleukin-12	Dendritic cells, macrophages	Activation of the Th1 pathway; induction of interferon-γ from Th1 cells, CTLs, and NK cells; synergism with interleukin-18	IL-8 (CXCL8)	Recruitment of neutrophils, endothelium	
Interleukin-17	Th17 cells, NK cells, group 3 innate lymphoid cells	Protecting neutrophilic inflammation, protection from bacterial and fungal infections	IL-8 (CXCL8)	Recruitment of Th1 cells, NK cells, dendritic cells	
Interleukin-18	Macrophages, macrophages, dendritic cells	Proinflammatory alarm cytokine; activation of Th1 pathway, acting in synergy with interleukin-12		Recruitment of Th1 cells, NK cells, dendritic cells	
Interleukin-21	Macrophages, dendritic cells, mast cells, epithelial cells	Proinflammatory alarm cytokine; amplification of Th1 and Th2 cells, activation of NK cells, CTLs, and mast cells		Recruitment of Th1 cells, NK cells, dendritic cells	
Interleukin-23	Th17 cells, CTLs, group 3 innate lymphoid cells, and NK cells	Proinflammatory alarm cytokine; activation of macrophages		Recruitment of Th1 cells, NK cells, dendritic cells	
Tumor necrosis factor-α	Macrophages, T cells, NK cells, mast cells	Increasing vascular permeability; pyrogenic function		Recruitment of Th1 cells, NK cells, dendritic cells	
GM-CSF	Th17 cells	Proinflammatory cytokine		Recruitment of Th1 cells, NK cells, dendritic cells	
VEGF	Macrophages	Angiogenesis		Recruitment of Th1 cells, NK cells, dendritic cells	
			Plasma proteins		
			CSP	Hepatocytes	Microscopic CSP increases interleukin-8 and MCP-1 secretion, interleukin-8 increases CSP expression
			Complement	Hepatocytes, other cells	Complement activation contributes to tissue damage in cytokine storm; complement inhibitors can reduce immunopathologic effects of cytokine storm
			Fibrin	Fibrinolytic	Primary site of iron storage in cells

Médiateurs solubles dans l'orage cytokinique

N Engl J Med 2020;383:2255-73



Définition **Physiopathologie**

Personalized Hemodynamic Resuscitation Targeting Capillary Refill Time in Early Septic Shock
The ANDROMEDA-SHOCK-2 Randomized Clinical Trial

JAMA

QUESTION Does a personalized hemodynamic resuscitation strategy targeting capillary refill time improve outcomes in patients with early septic shock vs usual care?

CONCLUSION In patients with early septic shock, a personalized hemodynamic resuscitation protocol targeting capillary refill time (CRT-PHR) was superior to usual care.

POPULATION	INTERVENTION	FINDINGS
831 Men 636 Women Adults 18 years or older with septic shock Mean age: 66 years	1501 Patients randomized 1467 Patients analyzed 720 CRT-PHR Usual care PHR targeted at normalizing CRT over a 6-hour period	Total No. of wins CRT-PHR: 131 131 (48.9%) Usual care: 112 787 (42.1%) CRT-PHR was superior to usual care: Win ratio, 1.16 (95% CI, 1.02 to 1.33, P = .04)

LOCATIONS 86 Sites in 19 countries

PRIMARY OUTCOME Hierarchical composite outcome: all-cause mortality, duration of vital support, and length of hospital stay at 28 days as an overall win ratio

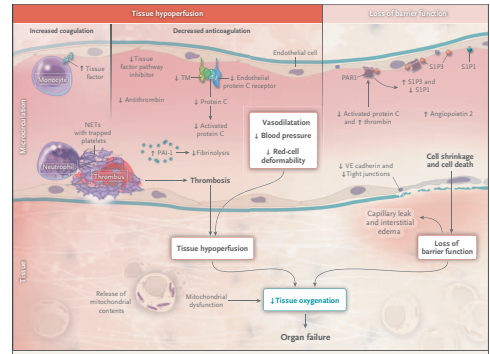
The ANDROMEDA-SHOCK-2 Investigators. Personalized hemodynamic resuscitation targeting capillary refill time in early septic shock. JAMA. Published online October 29, 2025. doi:10.1001/jama.2025.20402



JAMA. 2025;334(22):1988-1999.

Définition **Physiopathologie**

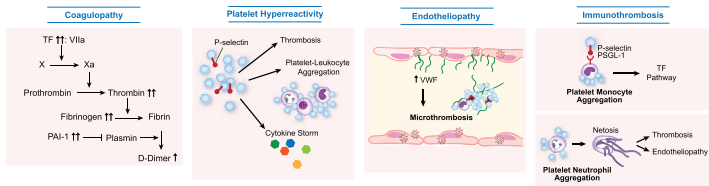
Dysfonction de l'endothélium vasculaire



N Engl J Med 2013; 369:840-851

Définition **Physiopathologie**

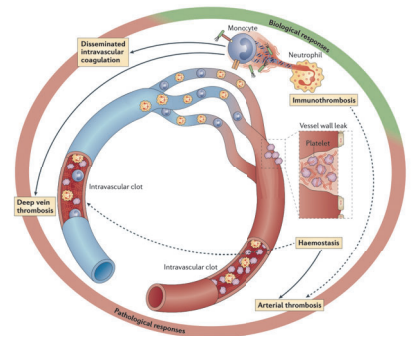
Atteinte microthrombotique



Curr Opin Hematol 2021; 28:445 - 453

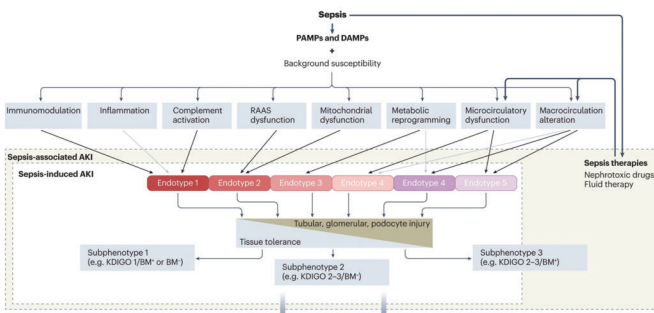
Définition **Physiopathologie**

Immunothrombose et coagulation induite par le sepsis



Nat Rev Immunol. 2013 Jan;13(1):34-45

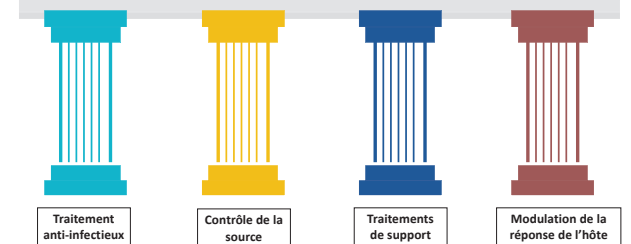
Définition **Physiopathologie**



Nat Rev Nephrol. 2023 Feb 23

Définition **Physiopathologie** **Traitement**

Traitement du sepsis



Définition **Physiopathologie** **Traitement**

Critical Care Medicine
 April 2026 • Volume 54 • Number 4 • Pages 725-812

Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2026

2021: THE OLD GUIDELINES

- Physical exam bundles
- Standardized care pathways
- qSOFA (added) and LEMS emphasis on practice
- Early broad-spectrum antibiotics
- Fluid 30 ml/kg fluids
- Fluid dynamic monitoring
- Standard organ ventilation
- Meets consensus recommendations
- Focus on acute phase

2026: THE NEW GUIDELINES

- System-wide QI programs
- Adaptive, patient-centered care
- NICE (NG102) / AHA/ACC guideline
- Prognostic screening
- Individualized organ support
- Early RRT more targeted strategy
- Strong focus on de-escalation & stewardship
- Dynamic assessment
- Early responses
- HFOV preferred early
- Lung protective ventilation
- Customized organ targets
- 2026 (Clear Shift)
- Standardized
- Immunoglobulins
- Blood purification
- Early detection
- ICU Care
- Post-Intensive Care

Crit Care Med. 2026 Apr 1;54(4):725-812

Définition **Physiopathologie** **Traitement**

Traitements de support

- ✓ Vasopressors 1-6 hours after onset
 - Norepinephrine
 - Dopamine
 - Phenylephrine
- ✓ Enteral feeding
- ✓ Insulin therapy
- ✗ Deep sedation
- ✓ Fluids Several liters initially
 - Colloids
 - Crystalloids
 - Starches
 - High chloride
- ✗ Goal oriented therapy
- ✗ EGDT Early goal directed therapy
- ✓ Lung protective ventilation
- ✓ Urinary catheter

BMJ 2016; 353 doi: <https://doi.org/10.1136/bmj.f1585>

ORIGINAL ARTICLE

Initiation Strategies for Renal-Replacement Therapy in the Intensive Care Unit

Critères d'inclusion:

- AKI compatible with a diagnosis of acute tubular necrosis in the context of ischemic or toxic injury
- KDIGO stage 3 acute kidney injury
- Invasive mechanical ventilation or catecholamine infusion

Critères d'exclusion:

- Urée > 40 mmol/L
- Potassium > 6 mmol/L
- pH < 7,15 (acidose métabolique pure ou mixte avec Paco2 > 50 mmHg)
- Acute pulmonary edema due to fluid overload responsible for severe hypoxemia requiring an oxygen flow rate > 5 L/mn or FiO2>50%

N Engl J Med. 2016 Jul 14;375(2):122-33

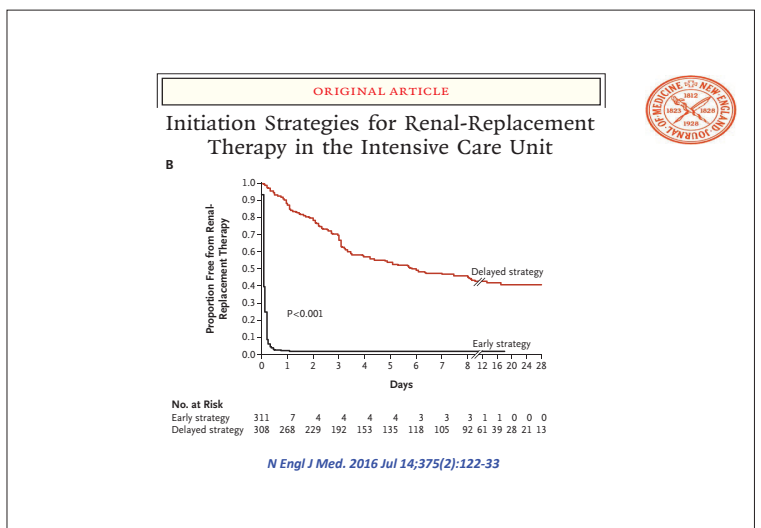
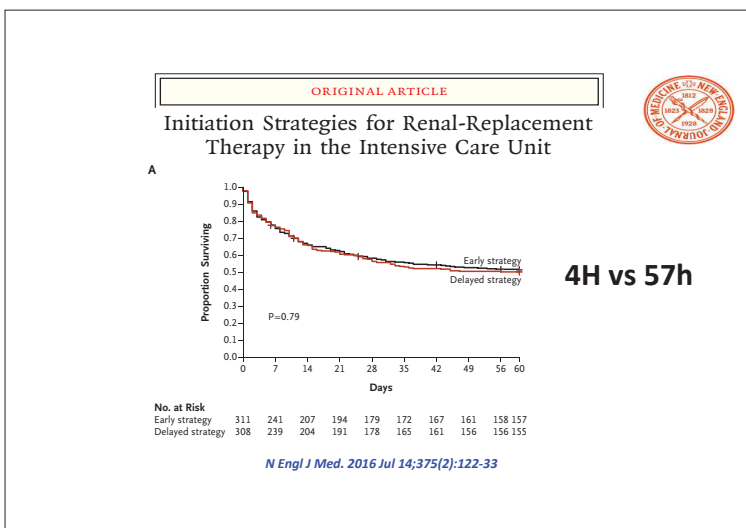
ORIGINAL ARTICLE

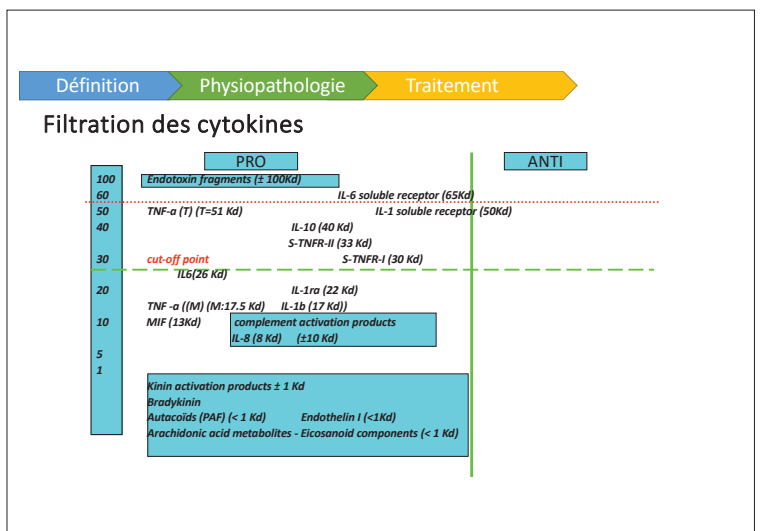
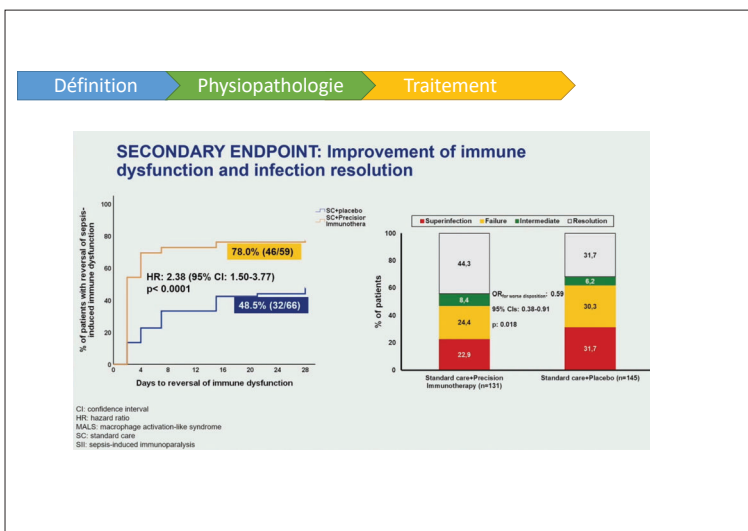
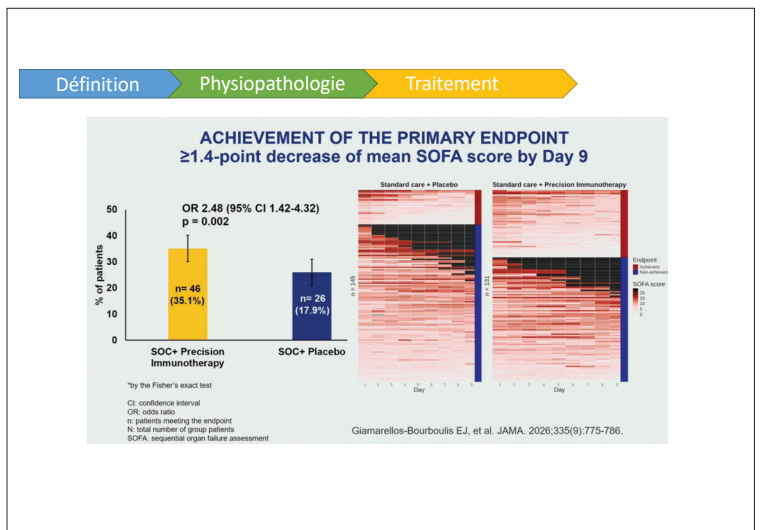
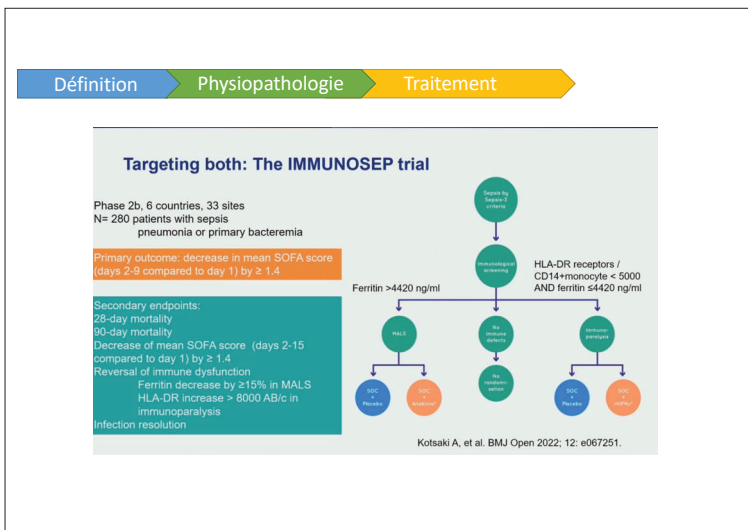
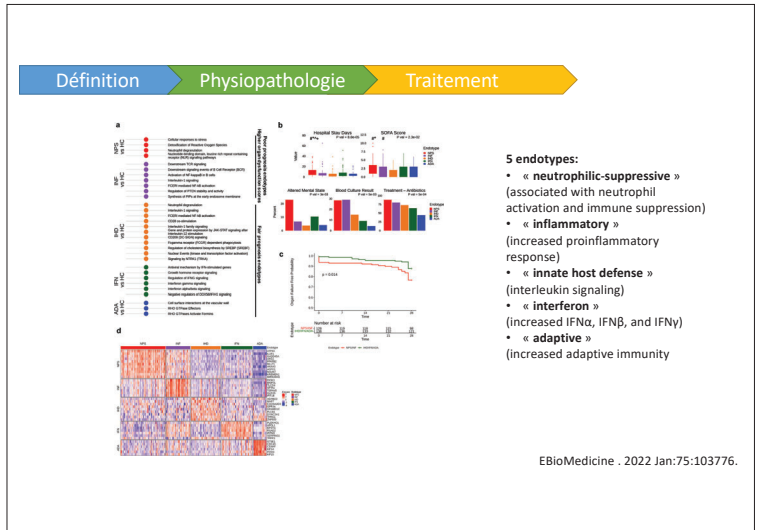
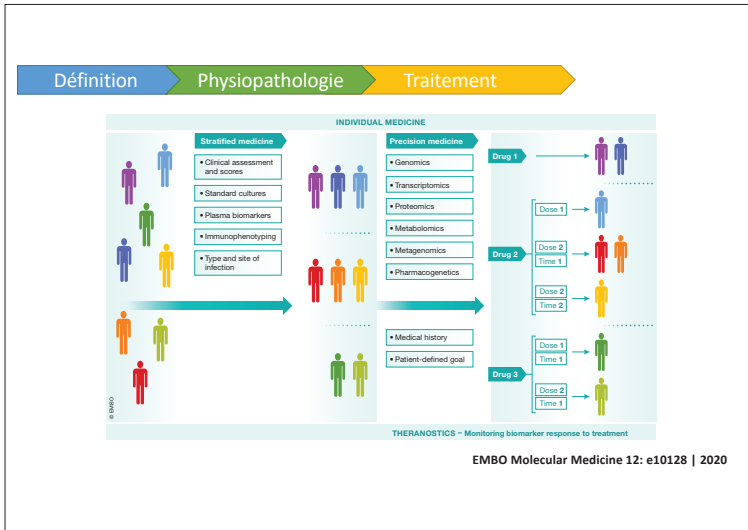
Initiation Strategies for Renal-Replacement Therapy in the Intensive Care Unit

Critères d'initiation de l'EER:

- Une insuffisance rénale KDIGO 3 avec oligurie ou anurie persistante pendant plus de 72 heures ;
- Urée > 40 mmol/L ;
- Potassium > 5,5 mmol/L malgré un traitement médical ;
- pH < 7,15 (acidose métabolique pure (PaCO2<30mmHg) ou acidose mixte (Paco2 > 50 mmHg sans possibilité d'améliorer la ventilation alvéolaire) ;
- Œdème pulmonaire aigu secondaire à une surcharge hydrosodée responsable d'une hypoxémie sévère (débit d'oxygène > 5l/min ou FiO2>50% en ventilation mécanique pour maintenir une SaO2>95%) malgré un traitement diurétique.

N Engl J Med. 2016 Jul 14;375(2):122-33

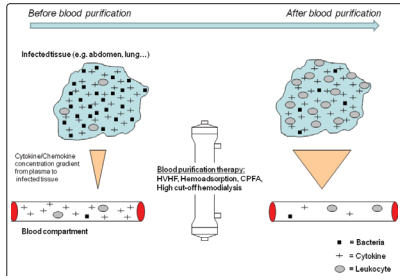




Modulation of chemokine gradients by apheresis redirects leukocyte trafficking to different compartments during sepsis, studies in a rat model

2014

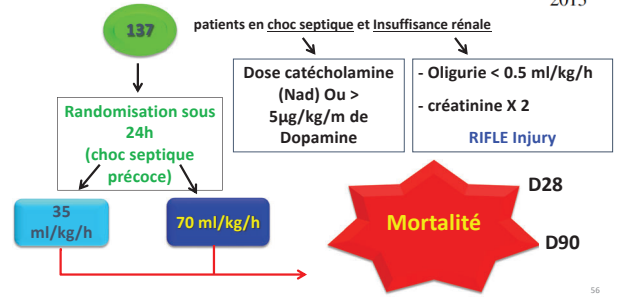
Zhi-Feng Peng^{1,2}, Jeffrey Y. Bishop³, Xiao-Yan Wen^{1,2}, Michele M. Eldor^{1,2}, Feihu Zhou^{1,2}, Anon Chaturvedi^{1,2}, Mehdi K. Carter¹, Jason E. Drenth¹, A. Muneer Ayres^{1,2}, Kai Ding^{1,2}, Francis Phai^{1,2}, Robert S. Fisher^{1,2,4}, Gilles Clermont^{1,2,5,6}, William J. Federspiel^{1,2,6} and John A. Kellum^{1,2,4,6,7}



High-volume versus standard-volume haemofiltration for septic shock patients with acute kidney injury (IVOIRE study): a multicentre randomized controlled trial

Olivier Joannes-Boyau, Patrick M. Honoré, Paul Perez, Sean M. Bagshaw, Hubert Grand, Jean-Luc Canivet, Antoine Dewitte

2013



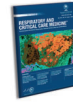
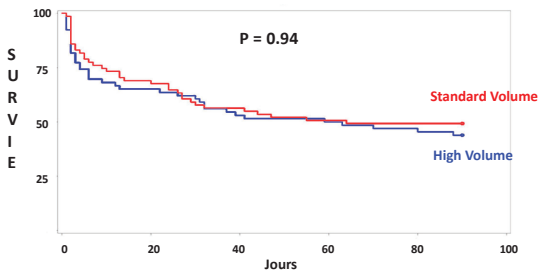
56



High-volume versus standard-volume haemofiltration for septic shock patients with acute kidney injury (IVOIRE study): a multicentre randomized controlled trial

Olivier Joannes-Boyau, Patrick M. Honoré, Paul Perez, Sean M. Bagshaw, Hubert Grand, Jean-Luc Canivet, Antoine Dewitte

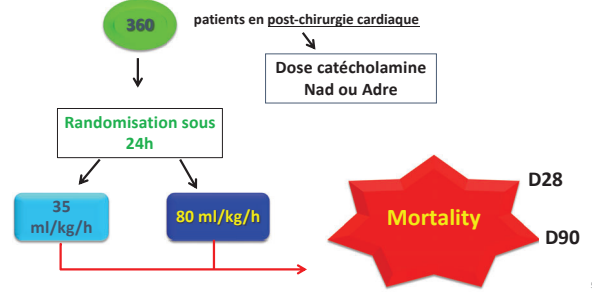
2013



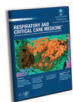
Early High-Volume Hemofiltration versus Standard Care for Post-Cardiac Surgery Shock The HEROICS Study

Alain Combes¹, Nicolas Bréchet¹, Julien Amour², Nathalie Cozic², Guillaume Lebreton⁴, Catherine Guidon⁵, Elie Zoghbi⁶, Jean-Claude Thiranos⁷, Jean-Christophe Rigal⁸, Olivier Bastien⁹, Hamina Benhaoua¹⁰, Bernard Abry¹¹, Alexandre Ouattara¹², Jean-Louis Trouillet¹, Alain Mallet¹, Jean Chastre¹, Pascal Leprince¹, and Charles-Edouard Loyt¹

2015



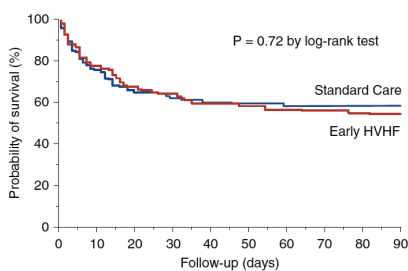
58



Early High-Volume Hemofiltration versus Standard Care for Post-Cardiac Surgery Shock The HEROICS Study

Alain Combes¹, Nicolas Bréchet¹, Julien Amour², Nathalie Cozic², Guillaume Lebreton⁴, Catherine Guidon⁵, Elie Zoghbi⁶, Jean-Claude Thiranos⁷, Jean-Christophe Rigal⁸, Olivier Bastien⁹, Hamina Benhaoua¹⁰, Bernard Abry¹¹, Alexandre Ouattara¹², Jean-Louis Trouillet¹, Alain Mallet¹, Jean Chastre¹, Pascal Leprince¹, and Charles-Edouard Loyt¹

2015



59

High-volume hemofiltration for septic acute kidney injury: a systematic review and meta-analysis

Edward Clark^{1,2}, Amber O. Molnar^{1,2}, Olivier Joannes-Boyau¹, Patrick M. Honoré¹, Lindsey Sikors³ and Sean M. Bagshaw^{1,2}

2014

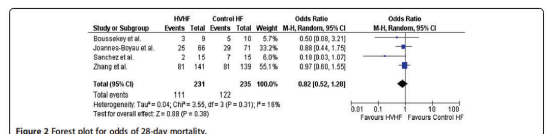


Figure 2 Forest plot for odds of 28-day mortality.



Annals of Translational Medicine, Vol 8, No 7 April 2020

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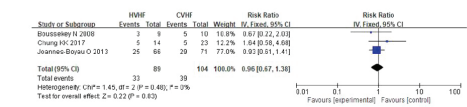
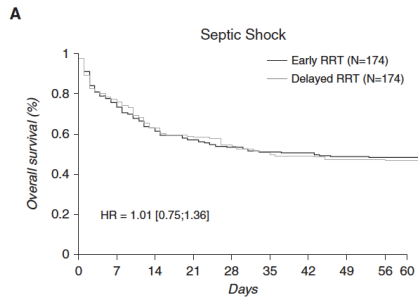


Figure 4 Forest plot of comparison in relative risk of mortality.



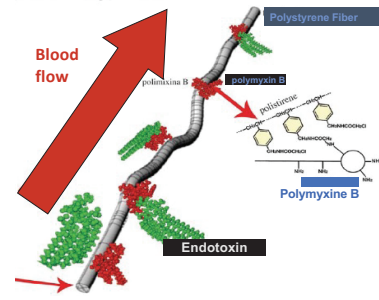
Timing of Renal Support and Outcome of Septic Shock and Acute Respiratory Distress Syndrome
 A Post Hoc Analysis of the AKIKI Randomized Clinical Trial
 Stéphanie Gaudry^{1,2}, David Hajage^{3,4,5}, Frédérique Schortgen⁶, Laurent Martin-Lefevre⁷, Charles Verney¹, Bertrand Pons^{8,9}, Eric Boulet¹⁰, Alexandre Boyer¹¹, Guillaume Chevrel¹², Nicolas Lerolle¹³, Dorothée Carpentier¹⁴

2018



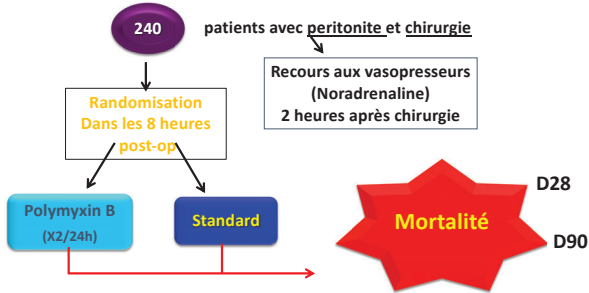
Définition → Physiopathologie → Traitement

Polymyxin B membrane



62

« ABDO-MIX study »



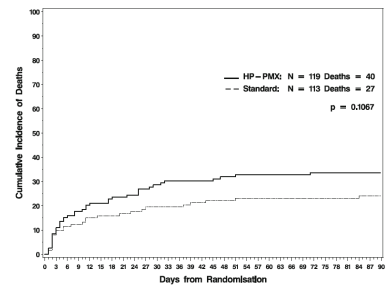
63



Early use of polymyxin B hemoperfusion in patients with septic shock due to peritonitis: a multicenter randomized control trial

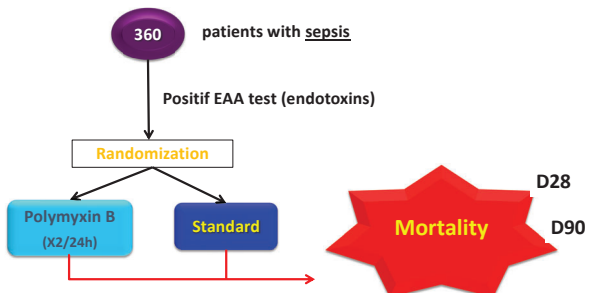
Didier M. Pype
 Justie Guilhot
 Youna Lamey
 Anne Claire Laskowicz
 Mahomed Kasai
 Kamel Yekel
 Julica Pottecher
 Olivier Jusseau-Beyan
 Laurent Martin-Lefevre

2015



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« EUPHRATES study »



65



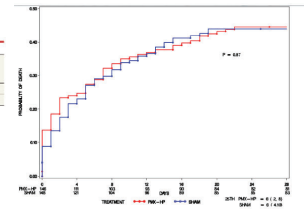
Effect of Targeted Polymyxin B Hemoperfusion on 28-Day Mortality in Patients With Septic Shock and Elevated Endotoxin Level
 The EUPHRATES Randomized Clinical Trial

2018

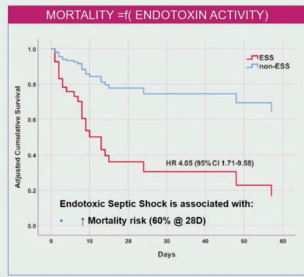
Philip Dellinger, MD, MSc; Sean M. Bagshaw, MD, MSc; Massimo Antonelli, MD; Debra M. Foster, BS; David J. Klein, MD, MBA; John C. Marshall, MD; Paul M. Palevsky, MD; Lawrence S. Weingart, MD; Christa A. Schorr, DNP, MSN, RN; Stephen Trzeciak, MD, MPH; Paul M. Nolan, MD, PhD, for the EUPHRATES Trial Investigators

Table 2. Summary of the Primary End Point of 28-Day Mortality for All Participants and for Patients With MOQS of More Than 9

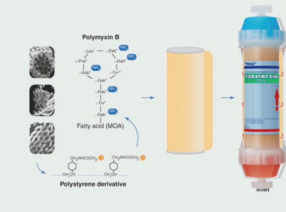
	No./Total (n)	OS (95% CI)	Risk Difference	Risk Ratio	P Value ^a
All Participants	84/223 (37.7)	76/228 (34.5)	3.15 (-5.73 to 12.04)	1.09 (0.85 to 1.39)	.49
>9 MOQS ^b	85/146 (58.2)	82/148 (55.4)	0.80 (-10.75 to 11.97)	1.01 (0.76 to 1.33)	.92



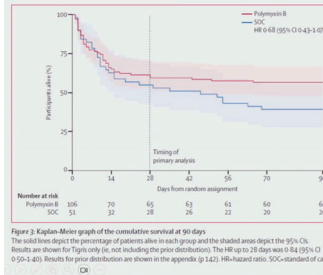
Endotoxic Septic Shock



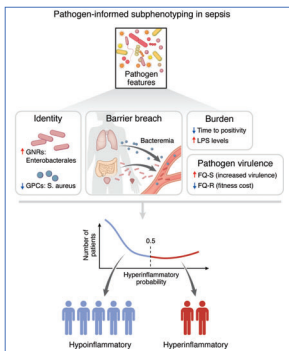
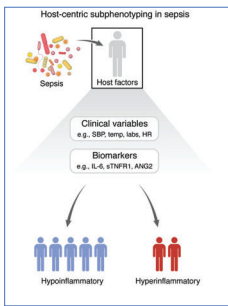
• POLYMYXIN Based Endotoxin Adsorption



Polymyxin B haemoadsorption in endotoxic septic shock (Tigris): a multicentre, open-label, Bayesian, randomised, controlled, phase 3 trial



Endotoxic septic shock defined by high endotoxin activity and multiorgan failure, polymyxin B haemoadsorption was associated with a high probability of lower mortality at 28 days and 90 days



Redefine critical illness based on biological mechanisms, rather than clinical syndromes

J Clin Invest. 2026;136(6):e203658

Remerciements



The ImmunoConcEPT lab focuses on the immune system and its pathologies; it regroups most investigators involved in this field on the Bordeaux campus.

IMMUNOLOGY from CONCEPT and EXPERIMENTS to TRANSLATION



Groupe SEPSIS

