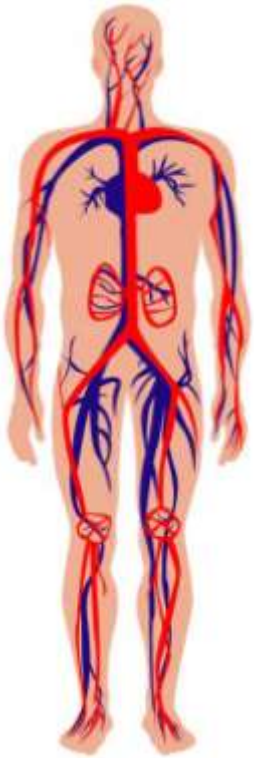
Xoc

Sandra Beltrán de Heredia Marrodán
Enric Samsó Sabé
Anestesiologia i Reanimació
Unitat de Critics Quirúrgics
Hospital del Mar. Parc de Salut Mar

Shock



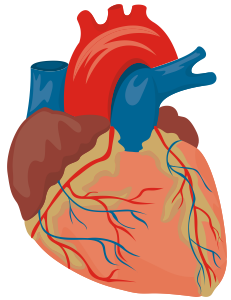
Insuficiència circulatòria
(hipotensió arterial) ?????

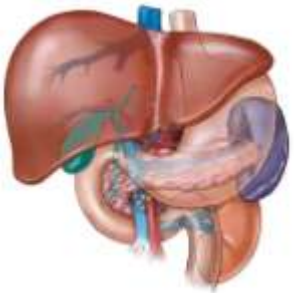
↓

Hipoperfusió tissular
(oliguria, obnubilació, ictericia...)

↓

Hipoxia cel·lular
(acidosi metabòlica)

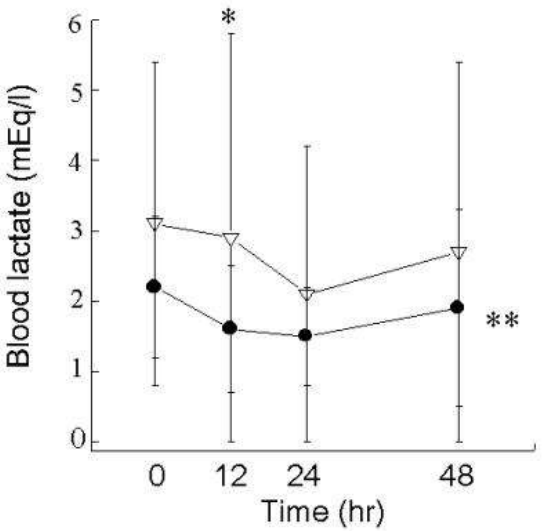


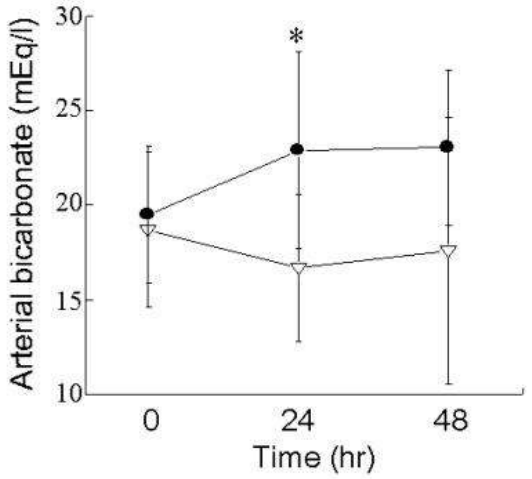


Occult hypoperfusion is associated with increased mortality in hemodynamically stable, high-risk, surgical patients

Critical Care April 2004 Vol 8 No 2 Meregalli et al

Elevated blood lactate levels are associated with a higher mortality rate and postoperativecomplications in hemodynamically stable surgical patients.

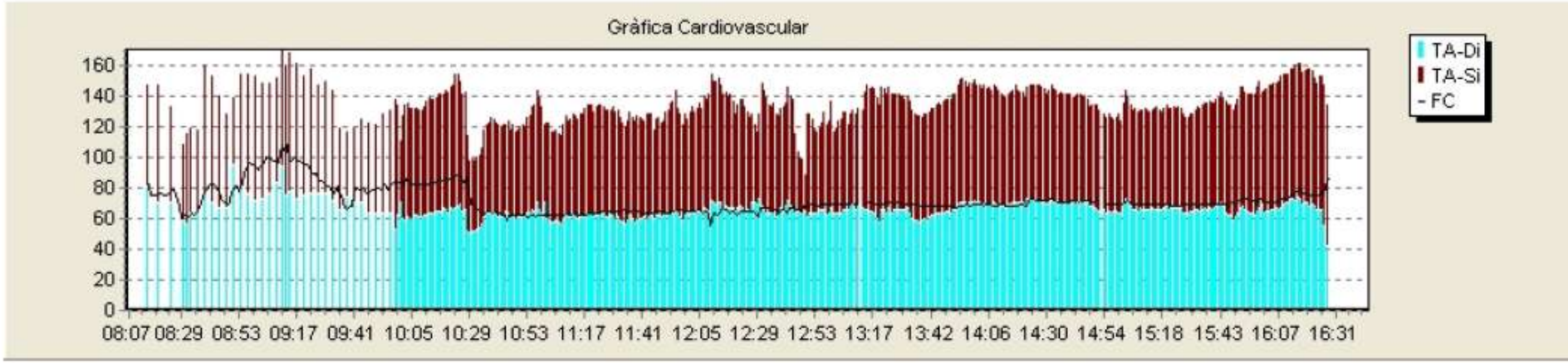




Intraoperative options for flow measurement

It is well known that none of the traditional parameters (HR, systemic arterial pressure, central venous pressure [CVP] and diuresis) are useful to accurately detect the volemic status of patients and that these indexes fail to detect persistent global tissue hypoxia or imbalance in whole-body oxygen supply and demand.[35,

NO CAL HIPOTENSIO



64 años

Laparotomía para ampuloma

Cirugía de Whipple

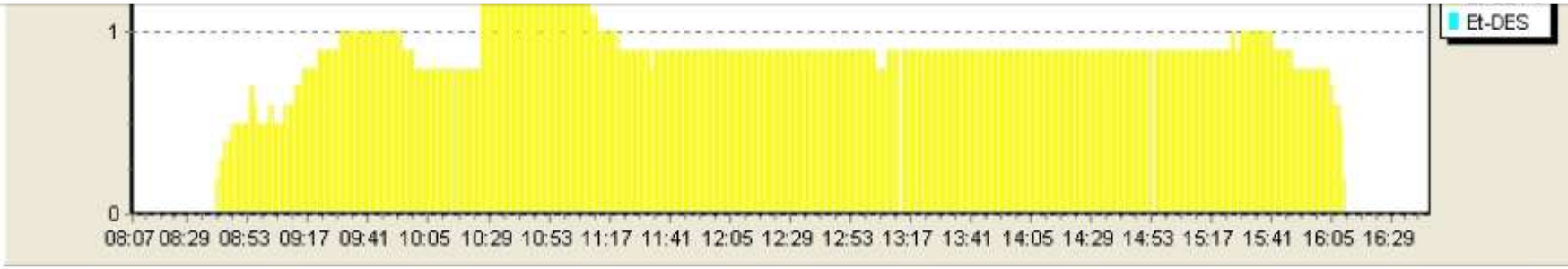
Linea arterial y cvc

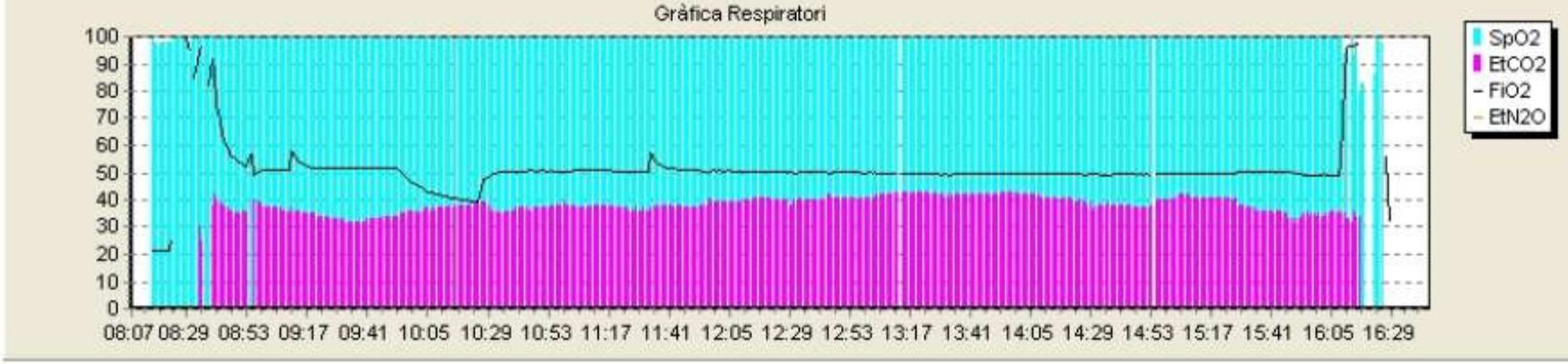
Sin incidencias en el intraoperatorio

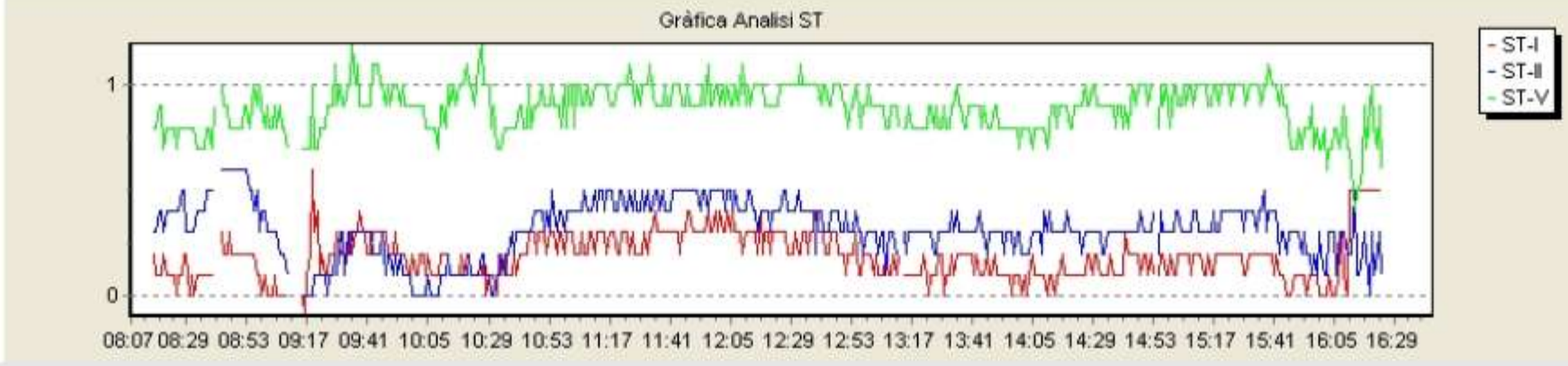
PVC?

Sangrado aproximado de 500 ml

Liquidos administrados: cristaloides (2000 ml)


1
0
Et-DES
08:07 08:29 08:53 09:17 09:41 10:05 10:29 10:53 11:17 11:41 12:05 12:29 12:53 13:17 13:41 14:05 14:29 14:53 15:17 15:41 16:05 16:29


Gràfica Respiratori
100
90
80
70
60
50
40
30
20
10
0
SpO2
EtCO2
FiO2
EtN2O
08:07 08:29 08:53 09:17 09:41 10:05 10:29 10:53 11:17 11:41 12:05 12:29 12:53 13:17 13:41 14:05 14:29 14:53 15:17 15:41 16:05 16:29


Gràfica Analisi ST
1
0
ST-I
ST-II
ST-V
08:07 08:29 08:53 09:17 09:41 10:05 10:29 10:53 11:17 11:41 12:05 12:29 12:53 13:17 13:41 14:05 14:29 14:53 15:17 15:41 16:05 16:29

	04-06-13 18:11	04-06-13 06:46	03-06-13 21:40	03-06-13 17:34
POTASSI Serum		4.18		3.88
CLOR Serum		102.6		104.6
pH/arterial		7.40	7.36	7.32
pCO2/arterial		43	41	44
HCO3/arterial		26.6	23.2	22.7
pO2/arterial		108	104	101
CO2 TOTAL/arterial		27.9	24.5	24.1
EXC BASE BE/arterial		1.8	-2.2	-3.4
BIC STD SBC/arterial		26.1	23.2	22.3
SATUR. O2/arterial		98	98	97
pH/venosa		PACLI		7.28
pCO2/venosa		X		49
pO2/venosa		X		
HCO3/venosa		X		
TCO2/venosa		X		24.5
EXC BASE BE/venosa		X		-3.7
SBE/s. venosa		X		-4.0
BIC STD SBC/venosa		X		21.1
SAT O2/venosa		X		69
OSMOLALITAT Serum		281	285	
Ac. LACTIC		1.7	4.6	5.0
CREATINA CINASA (CK) Seru				

Valors
Mínim
Màxi

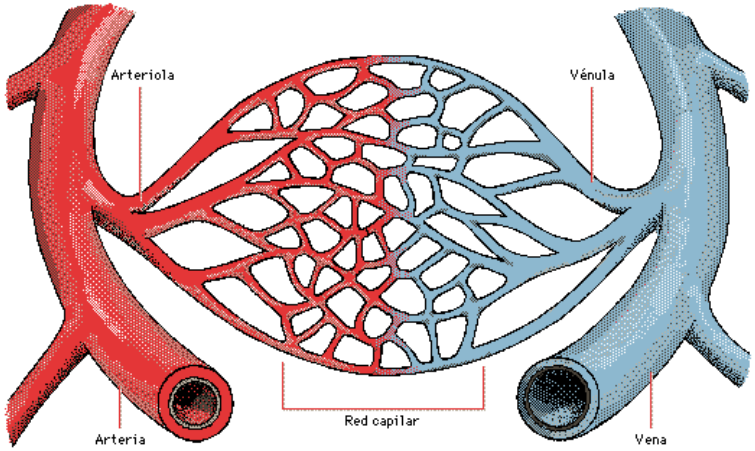
Líquidos
Concentrado de hematíes
Calentar

shock

OFERTA O_2

DO2: GC X CaO2

TA



CONSUMO O_2

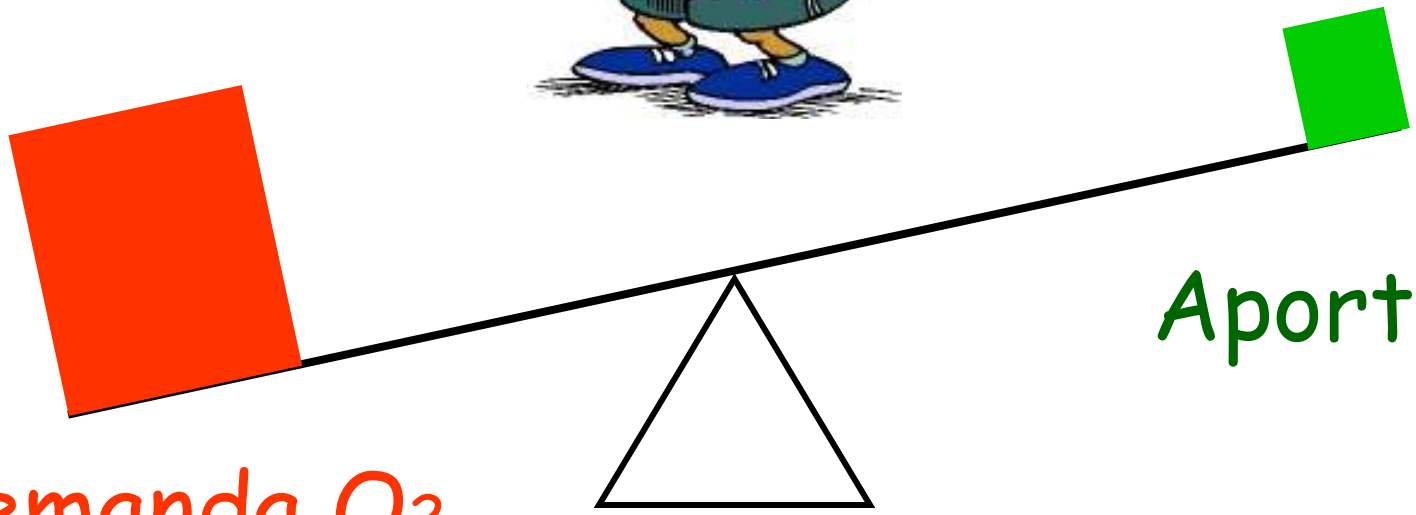
Láctico

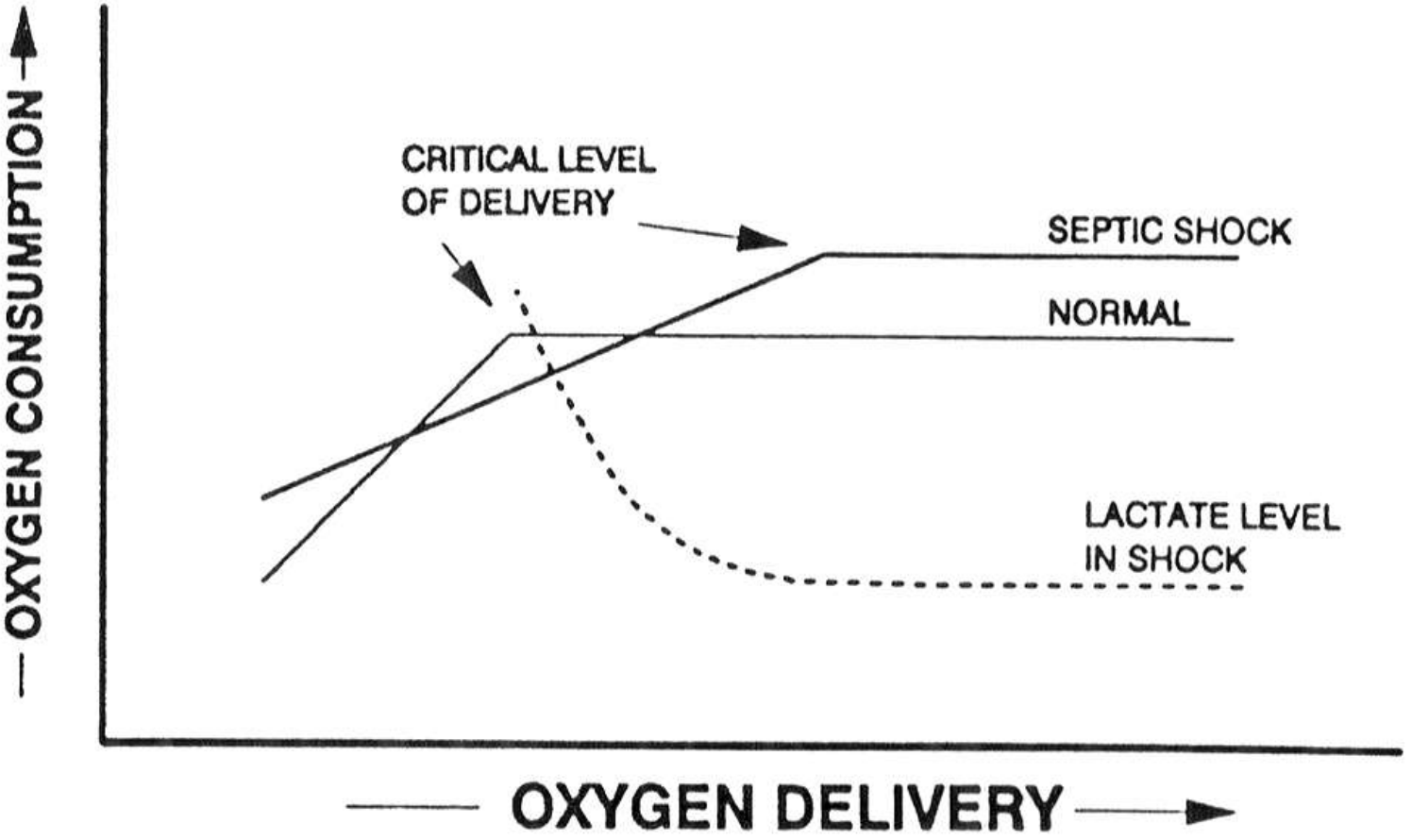
ΔCO_2

$ScvO_2$

XOC

HIPÒXIA Tissular

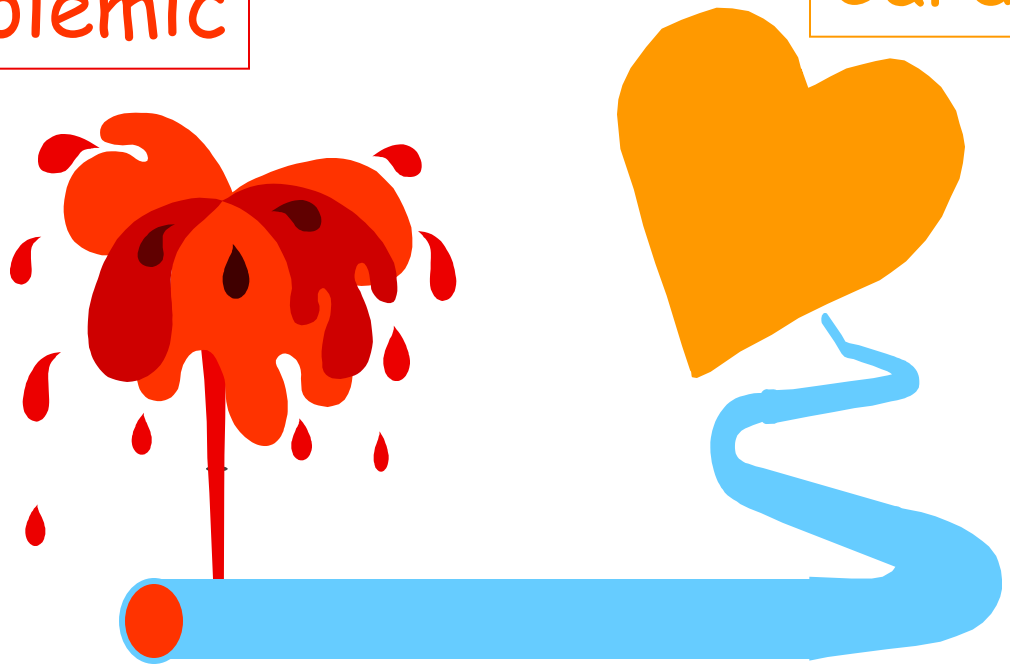



OXYGEN CONSUMPTION
CRITICAL LEVEL
OF DELIVERY
SEPTIC SHOCK
NORMAL
LACTATE LEVEL
IN SHOCK
OXYGEN DELIVERY

Xoc

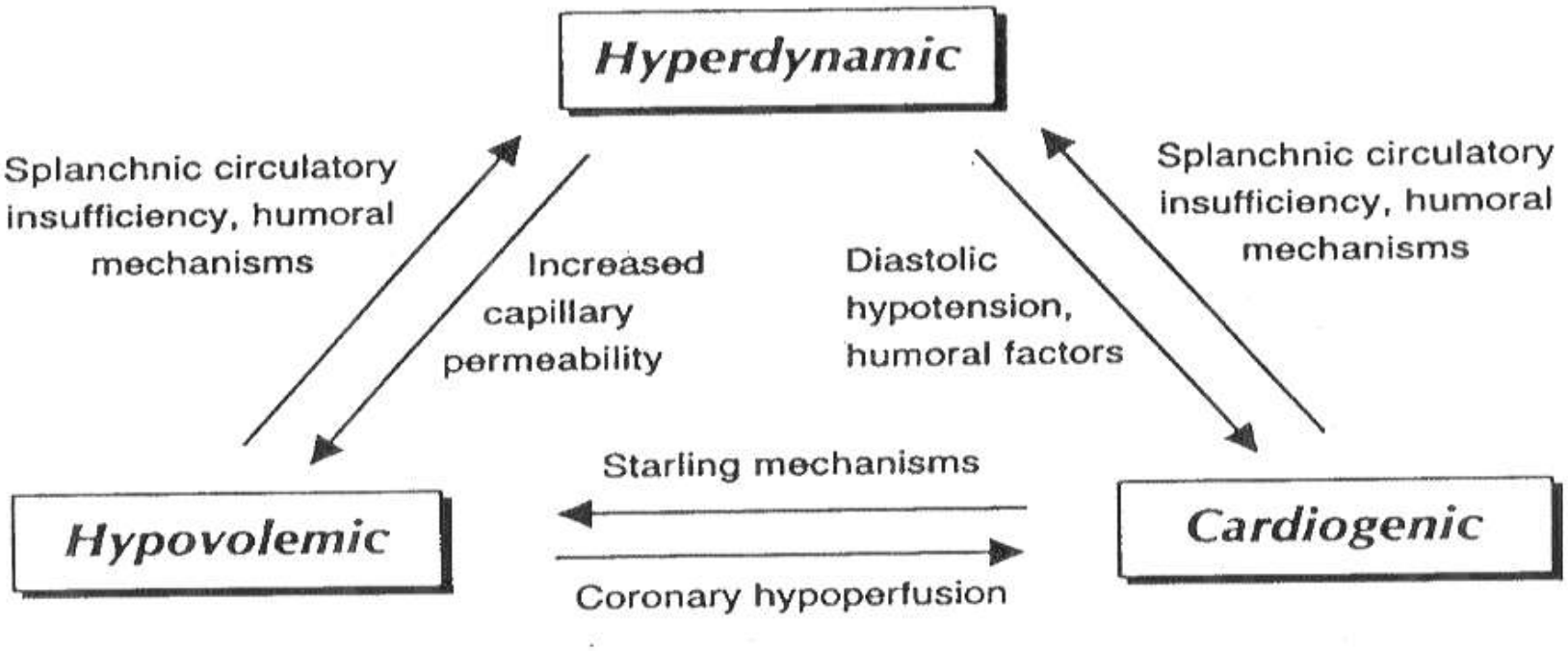
Hipovolèmic

Cardiogènic

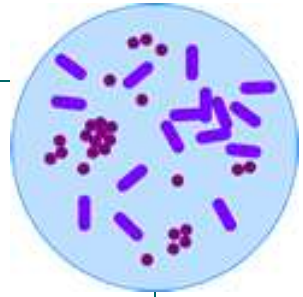


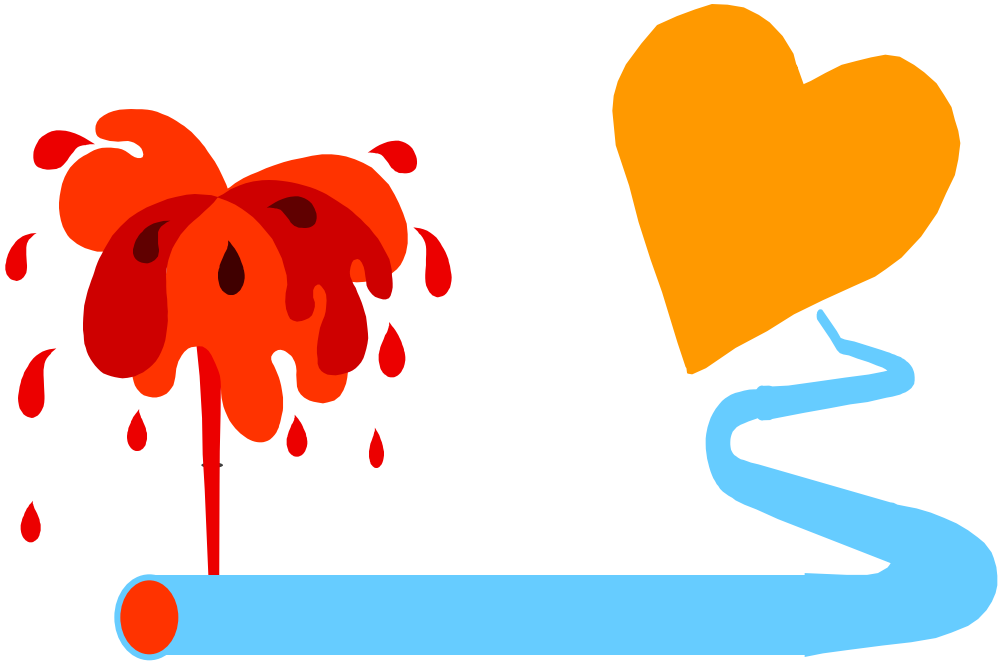
Distributiu

Хос


Hyperdynamic
Splanchnic circulatory insufficiency, humoral mechanisms
Increased capillary permeability
Diastolic hypotension, humoral factors
Splanchnic circulatory insufficiency, humoral mechanisms
Starling mechanisms
Hypovolemic
Cardiogenic
Coronary hypoperfusion

Xoc Sèptic



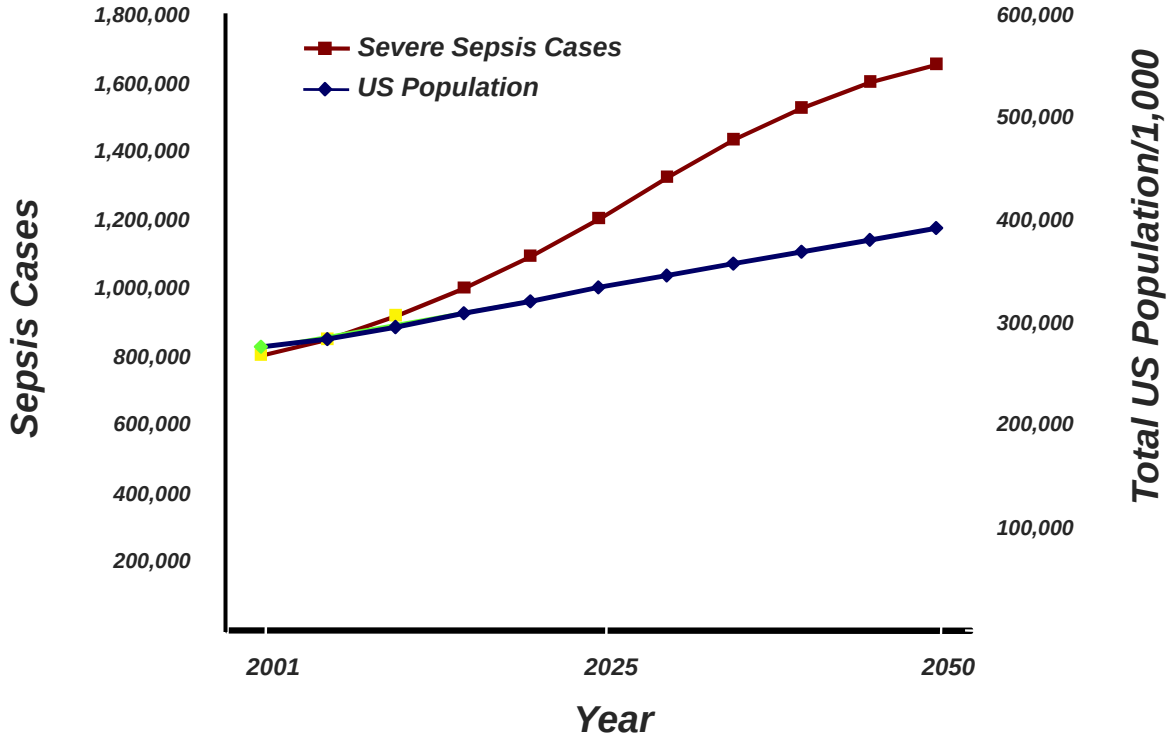


Sepsis, shock séptico

Presente

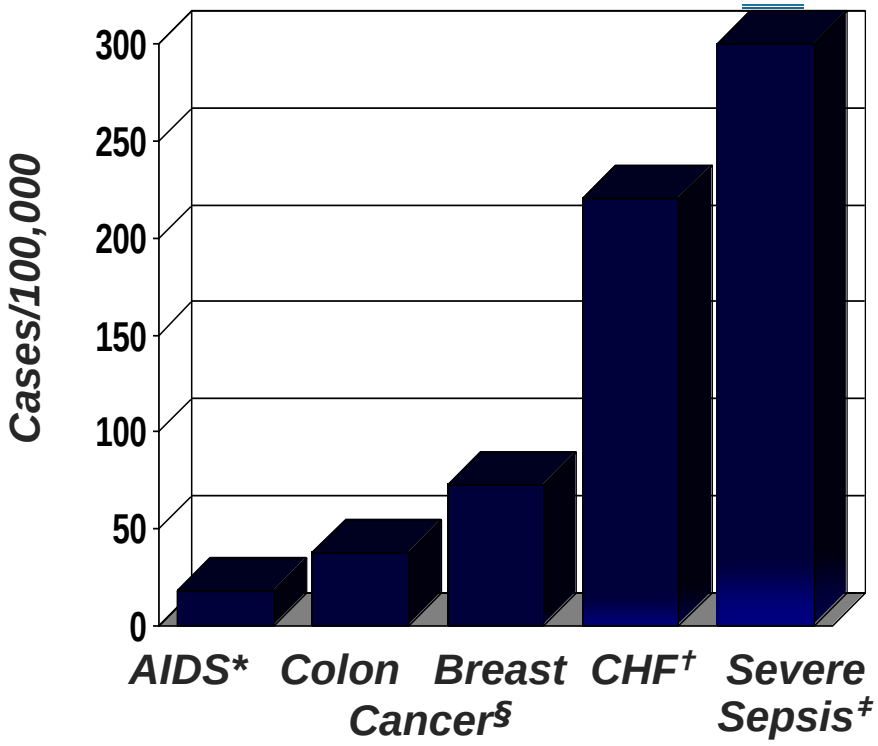
>750,000 sépsis severa / año en los EE.UU

Futuro

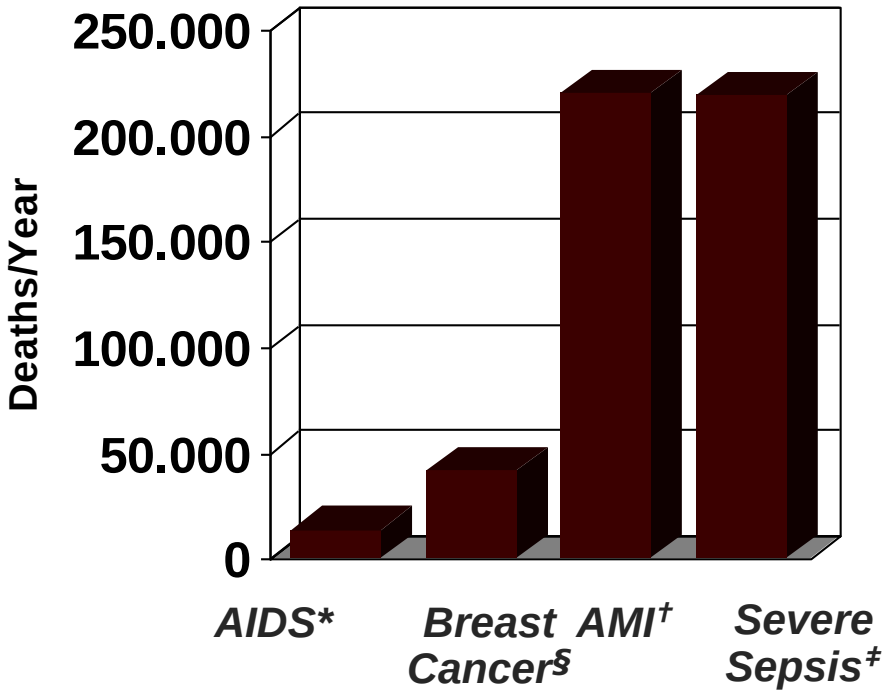


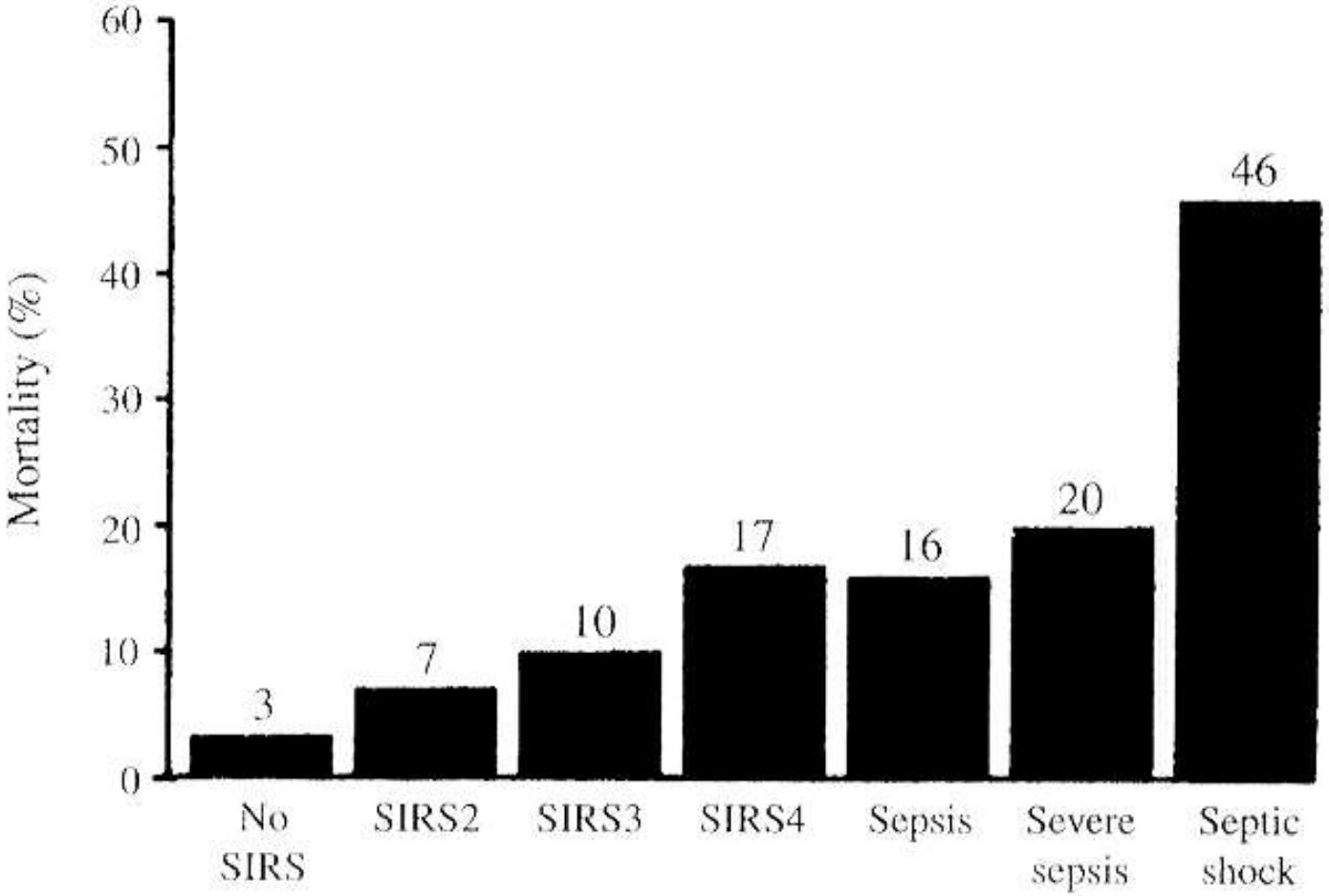
Angus DC. Crit Care Med. 2001;29(7):1303-1310.

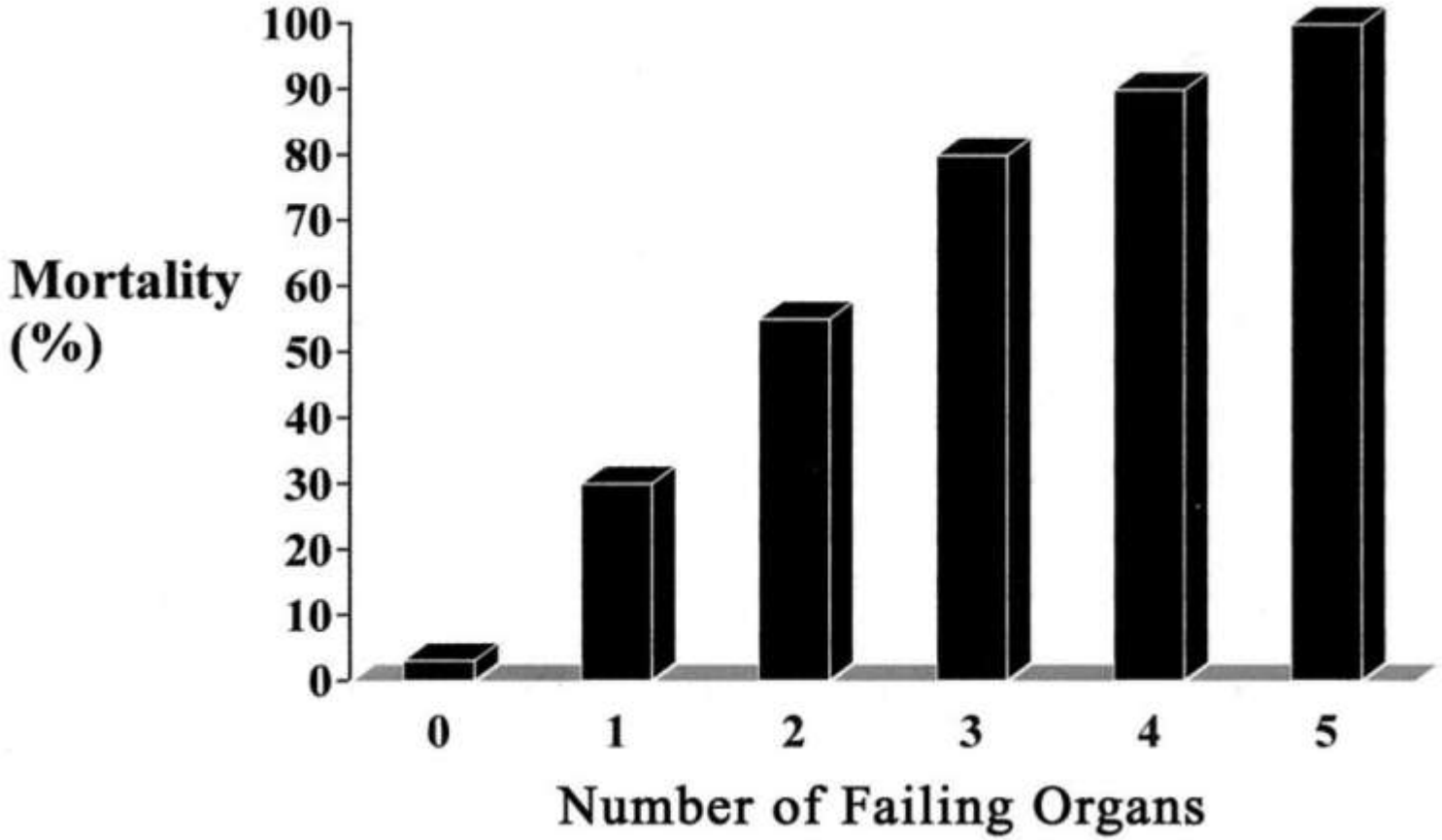
Incidencia de la Sépsis Severa



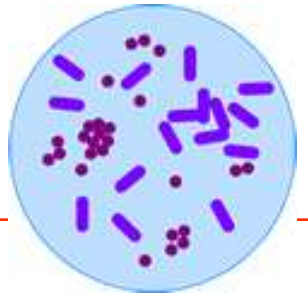
Mortalidad de la Sépsis Severa




Mortality (%)
60
50
40
30
20
10
0
3
7
10
17
16
20
46
No SIRS
SIRS2
SIRS3
SIRS4
Sepsis
Severe sepsis
Septic shock


Mortality (%)
100
90
80
70
60
50
40
30
20
10
0
0
1
2
3
4
5
Number of Failing Organs

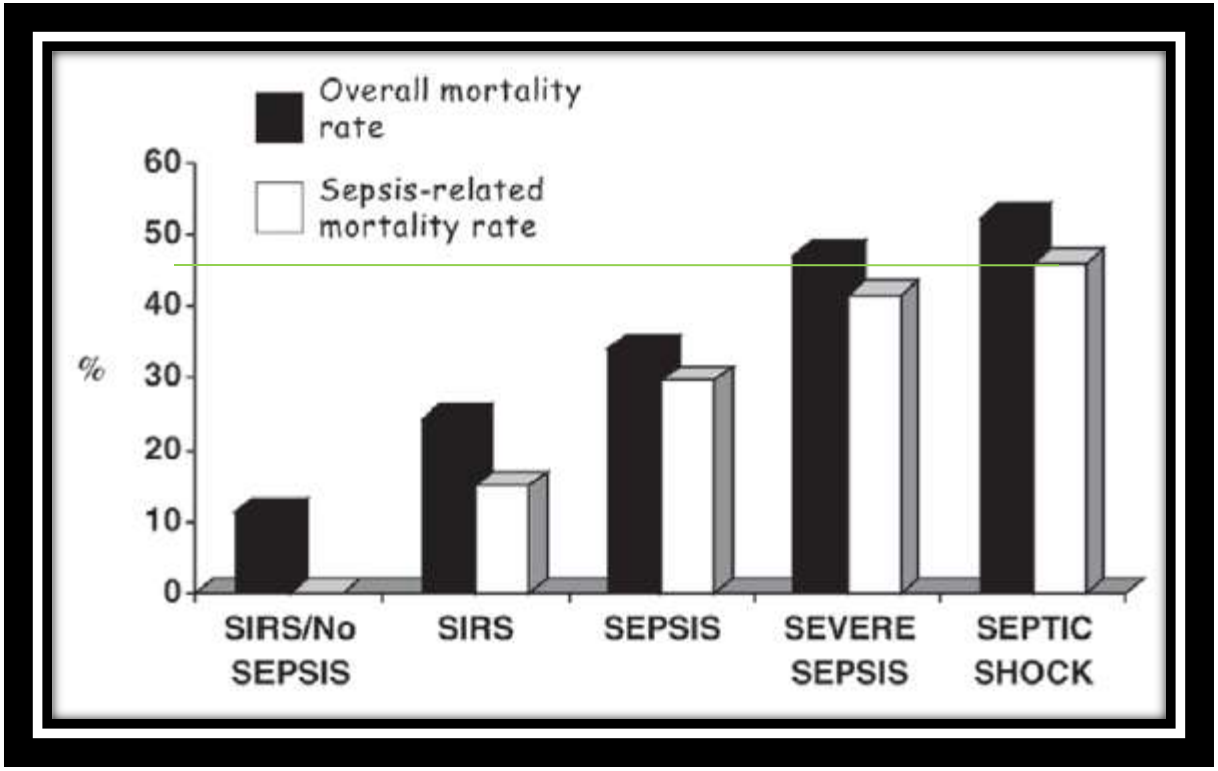
Factors de risc de mortalitat



- Edat > 70a.
- Traumatismes
- Diabetes
- Cirrosi Hepàtica
- Cremats
- Neoplasies
- Immunosupressors
- Resistencia a Antibiòtics
- Insuficiència Renal
- Insuficiència Respiratòria

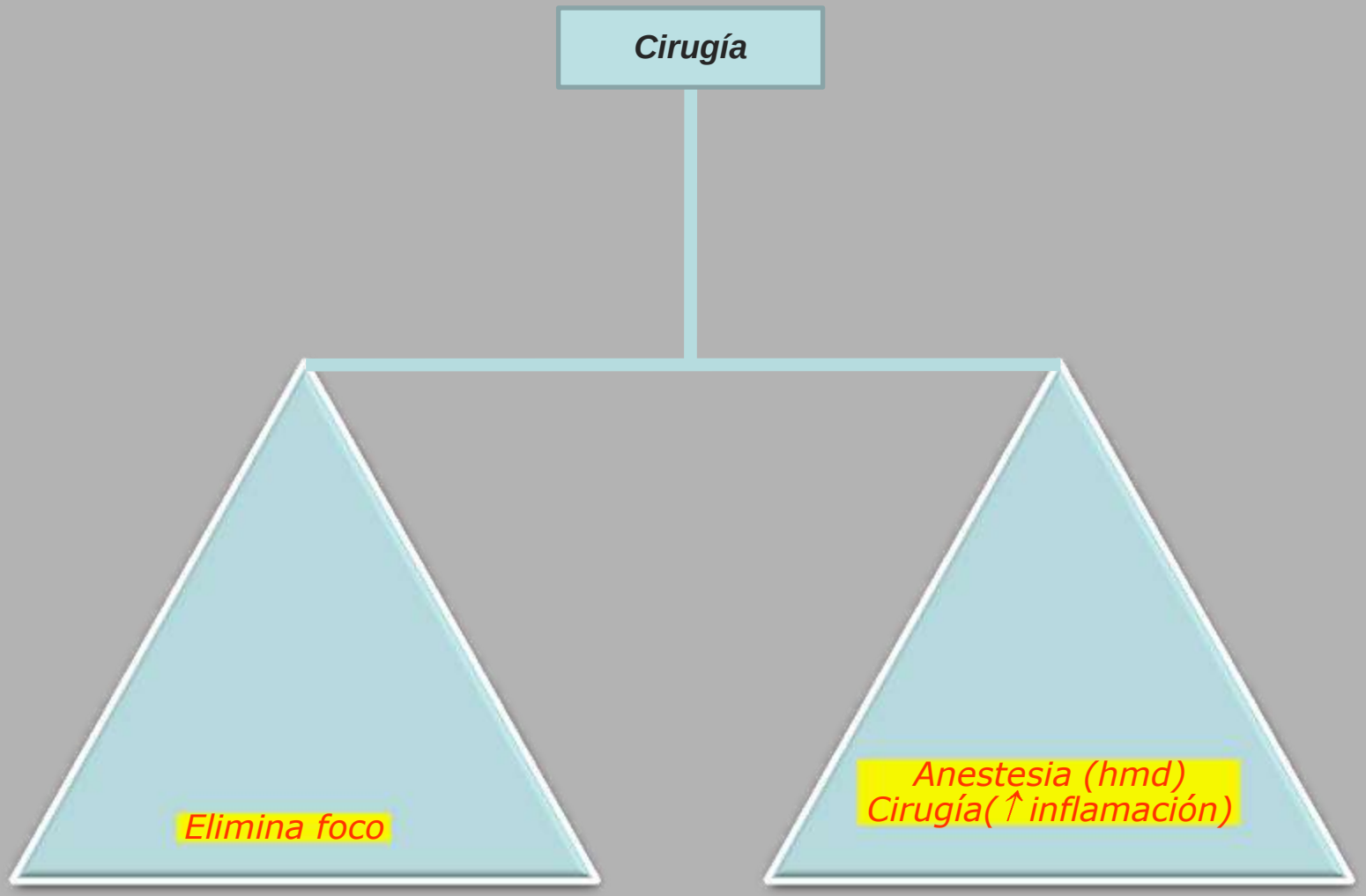
Brazilian Sepsis Epidemiological Study (BASES study)

Critical Care 2004, **8**:R251-R260



45%

Malalt médic ≠ Quirúrgic



… no hay antibiótico tan rápido…..

… aumentamos morbilidad..

Millors resultats amb cirurgía

CRITICAL CARE MEDICINE

Simon R. Finfer, M.D., and Jean-Louis Vincent, M.D., Ph.D., *Editors*

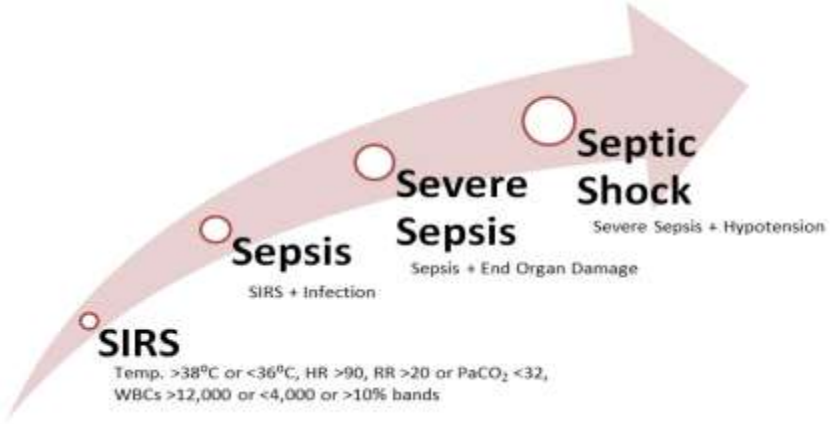
Severe Sepsis and Septic Shock

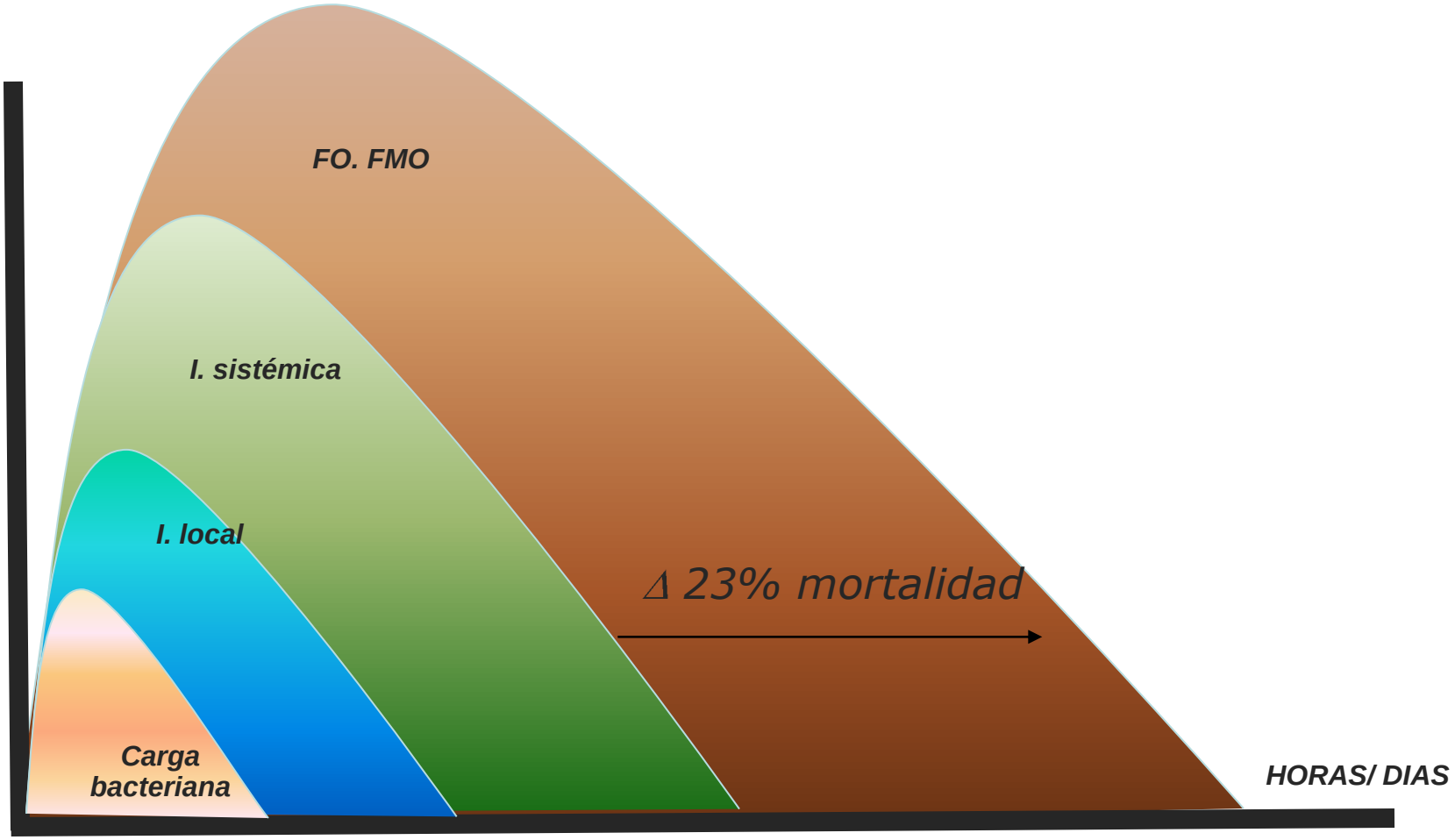
Derek C. Angus, M.D., M.P.H., and Tom van der Poll, M.D., Ph.D.

However, with the advent of modern antibiotics, germ theory did not fully explain the pathogenesis of sepsis: many patients with sepsis died despite successful eradication of the inciting pathogen. Thus, researchers suggested that it was the host, not the germ, that drove the pathogenesis of sepsis.[3]

N Engl J Med 2013;369:840-51.

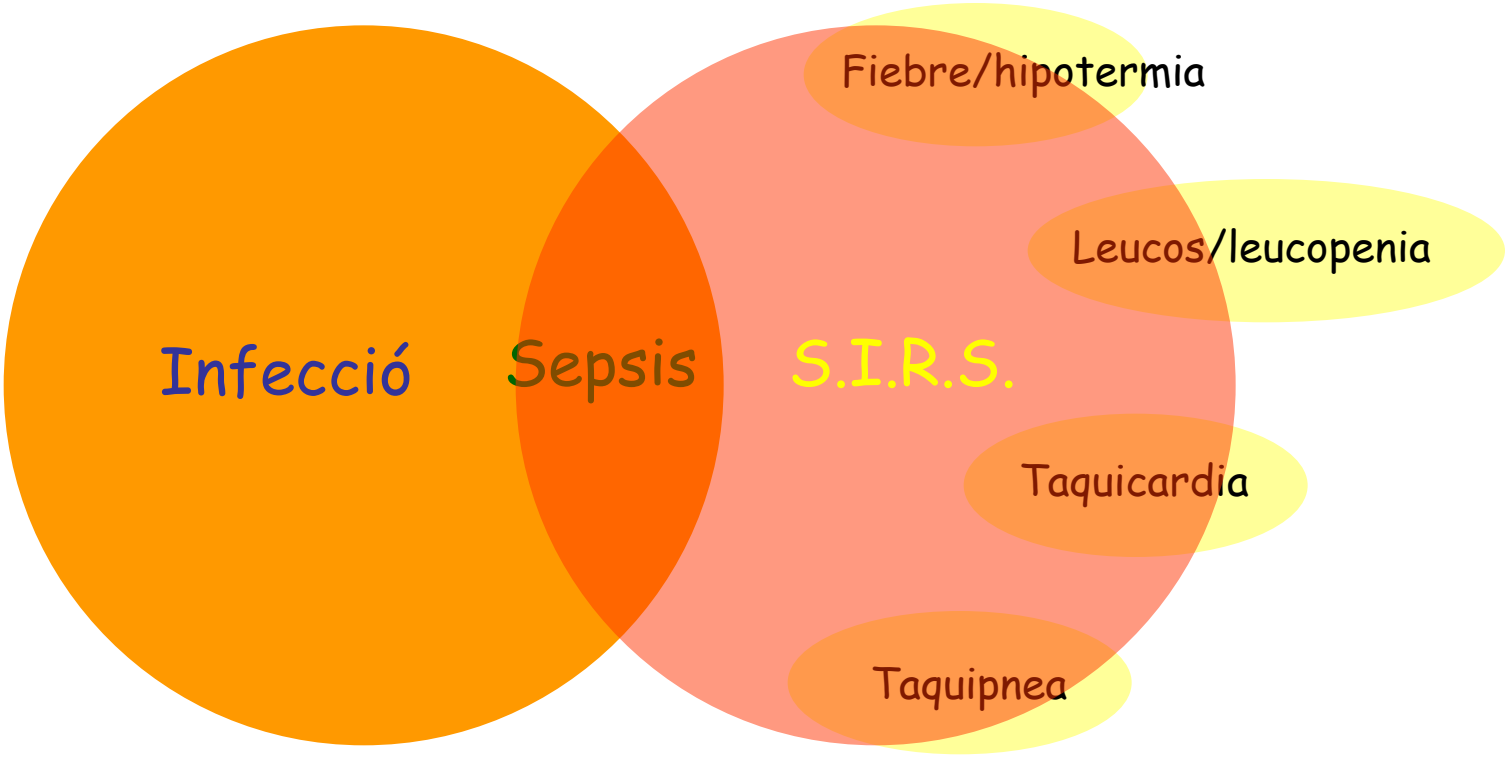
No todo son los antibióticos y la cirugía


Septic Shock
Severe Sepsis + Hypotension
Severe Sepsis
Sepsis + End Organ Damage
Sepsis
SIRS + Infection
SIRS
Temp. >38°C or <36°C, HR >90, RR >20 or PaCO2 <32,
WBCs >12,000 or <4,000 or >10% bands


FO. FMO
I. sistémica
I. local
Carga bacteriana
Δ 23% mortalidad
HORAS/ DIAS

Definicions:

SIRS	Resposta inflamatòria a una agressió externa
Sepsis	SIRS + infecció
Sepsis greu	Sepsis amb disfunció orgànica x hipoperfusió
Xoc sèptic	Sepsis + hipotensió malgrat reposició líquids



Definicions:

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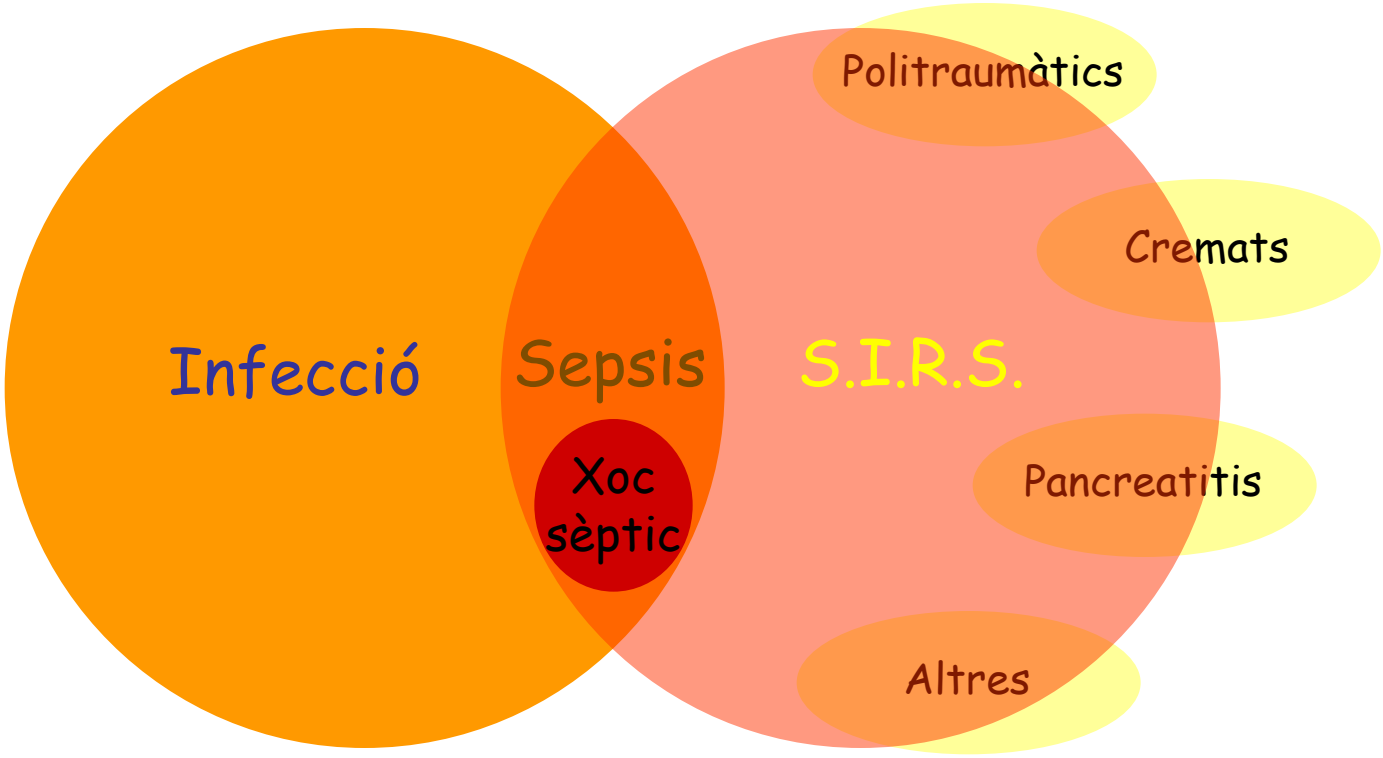
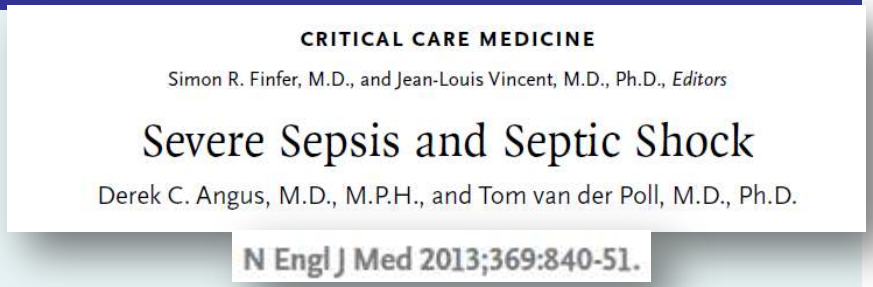


Sepsis: infección documentada y 1 o + de los siguientes

Variables generales
- Fiebre (>38,3ºC) ó hipotermia (<36ºC)
- Taquicardia (> 90 lpm)
- Taquipnea
- Alt. estado mental
- Edema notable ó balance + (>20 ml/kg en 24h)
- Hiperglucemia (>120 mgr/dl)



Variables inflamatorias
- Leucocitosis (>12000) o leucopenia (< 4000) ó > 10% de bandas
- PRC o PCR elevada (> 2 ds normal)

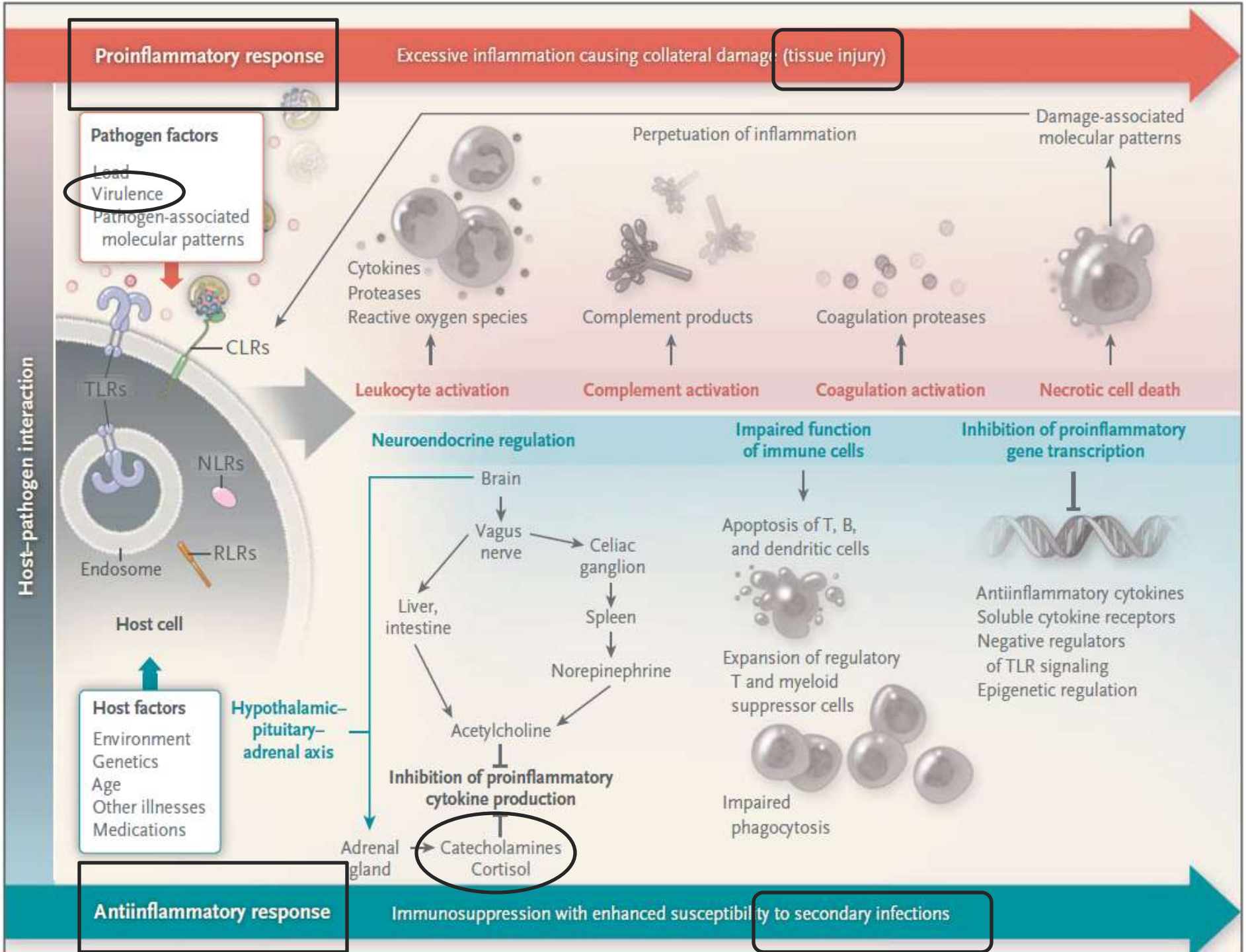
Varibles hemodinámicas
- Hipotensión (TAS<90 mmHg, TAM < 70 mmHg, ↓ TAS> 40 mmHg ó > 2 DS)
- SvO_2 > 70%
- IC > 3,5

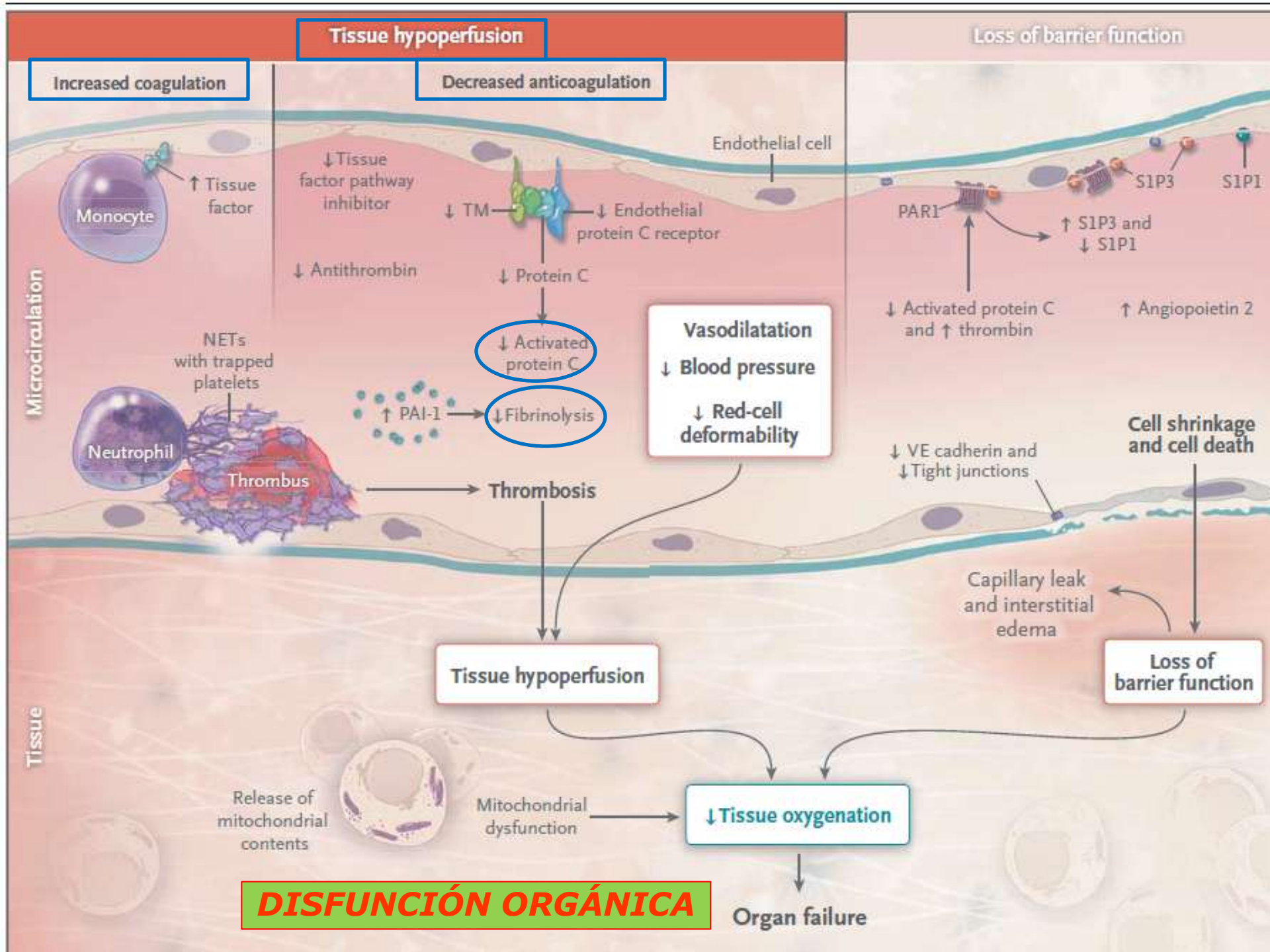
Disfunción orgánica
- Hipoxemia (Pa/FiO2<300)
- Oliguria (<0,5 ml/kg/h al meno en 2 horas
- Creatinina (> 0,5 nivel basal)
- INR > 1,5 ó TTPa > 60 sec
- Íleo paralitico
- Plaquetas < 100.000
- BiT > 4 mg/dl

Perfusión tisular
- Láctico elevado (> 1 mmol/L) (?)
- Relleno capilar > 3 seg o piel moteada

Sepsis severa (sepsis + disfunción orgánica)

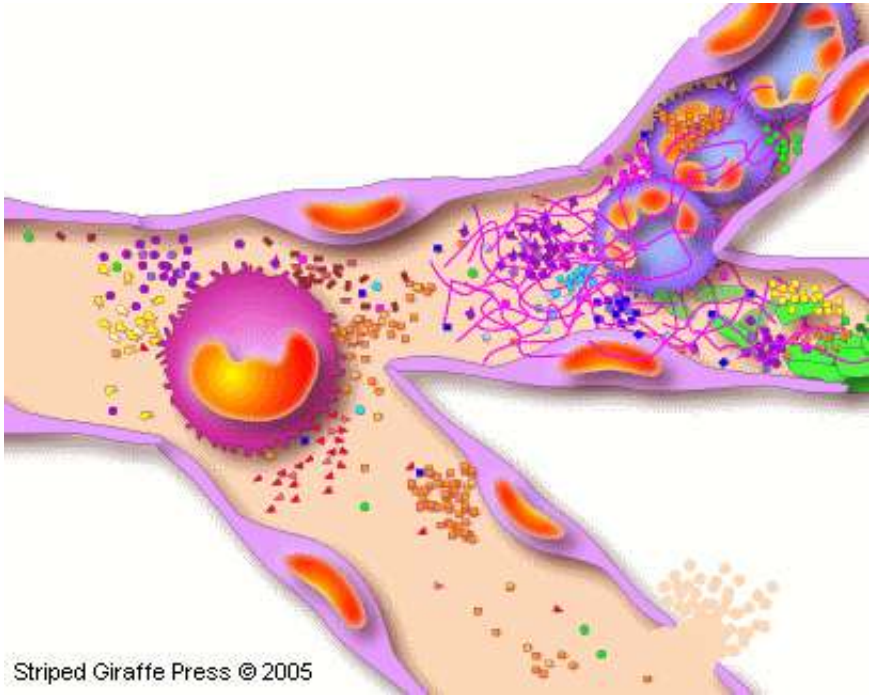
Shock séptico (sepsis e hipotensión (refractaria líquidos) o láctico elevado


Proinflammatory response
Excessive inflammation causing collateral damage (tissue injury)
Host–pathogen interaction
Pathogen factors
Load
Virulence
Pathogen-associated molecular patterns
CLRs
TLRs
NLRs
RLRs
Endosome
Host cell
Perpetuation of inflammation
Damage-associated molecular patterns
Cytokines
Proteases
Reactive oxygen species
Complement products
Coagulation proteases
Leukocyte activation
Complement activation
Coagulation activation
Necrotic cell death
Neuroendocrine regulation
Brain
Vagus nerve
Celiac ganglion
Liver, intestine
Spleen
Norepinephrine
Acetylcholine
Inhibition of proinflammatory cytokine production
Adrenal gland
Catecholamines
Cortisol
Hypothalamic–pituitary–adrenal axis
Impaired function of immune cells
Apoptosis of T, B, and dendritic cells
Expansion of regulatory T and myeloid suppressor cells
Impaired phagocytosis
Inhibition of proinflammatory gene transcription
Antiinflammatory cytokines
Soluble cytokine receptors
Negative regulators of TLR signaling
Epigenetic regulation
Host factors
Environment
Genetics
Age
Other illnesses
Medications
Antiinflammatory response
Immunosuppression with enhanced susceptibility to secondary infections


Tissue hypoperfusion
Loss of barrier function
Increased coagulation
Decreased anticoagulation
Endothelial cell
Monocyte
↑ Tissue factor
↓ Tissue factor pathway inhibitor
↓ TM
↓ Endothelial protein C receptor
↓ Antithrombin
↓ Protein C
PAR1
S1P3
S1P1
↑ S1P3 and ↓ S1P1
↓ Activated protein C and ↑ thrombin
↑ Angiopoietin 2
Microcirculation
NETs with trapped platelets
↓ Activated protein C
↑ PAI-1
↓ Fibrinolysis
Vasodilatation
↓ Blood pressure
↓ Red-cell deformability
Neutrophil
Thrombus
Thrombosis
↓ VE cadherin and ↓ Tight junctions
Cell shrinkage and cell death
Capillary leak and interstitial edema
Tissue hypoperfusion
Loss of barrier function
Tissue
Release of mitochondrial contents
Mitochondrial dysfunction
↓ Tissue oxygenation
DISFUNCIÓN ORGÁNICA
Organ failure

XOC SÈPTIC

Causes HIPÒXIA Tissular



- Microembolismes
- Lesió endotelial : EDEMA
  - ↑ distancia capilar - cèl·lula
  - compressió capilar
- Bloqueig de la captació mitocondrial d'O_2

XOC SÈPTIC

Causes HIPÒXIA Tissular

- Apertura de shunts A-V

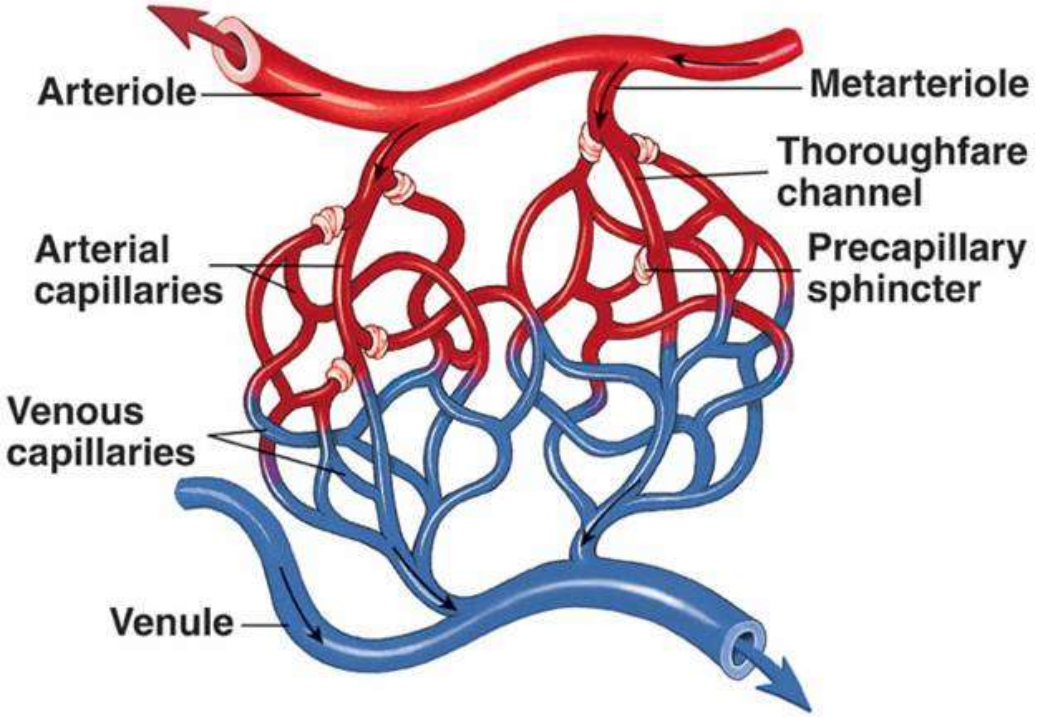


Tabla 1 Disfunciones orgánicas secundarias a la sepsis

- Respiratoria: infiltrados pulmonares e hipoxemia con Pa02/Fi02 <300 mmHg
- Oliguria (diur <0,5 mL/Kg/h durante al menos 2 h)
- Creatinina >2 mg/dl o incremento >0,5 mg/dl
- Coagulopatía (INR >1,5 o aPTT > 60 s)
- Trombocitopenia < 100.000/mm^3
- Hiperbilirrubemia (bilirrubina >2 mg/dl)
- Hiperlactacidemia (>3 mmol/L o 24 mg/dl)
- Hipotensión (presión arterial sistólica < 90 mm Hg, presión arterial media < 70 o descenso de presión arterial sistólica > 40 mmHg)

NIDUS OF INFECTION

- Pneumonitis
- Peritonitis
- Cellulitis
- Abscess
- Other infection sites

ORGANISMS

EXOGENOUS TOXINS

- Organism
- Structural Component
- Exotoxin (TSST-1, Toxin A)
- Endotoxin

ENDOGENOUS MEDIATORS

CYTOKINES
- Interleukin 1,2...6
- Tumor Necrosis Factor (TNF)

PLATELET ACTIVATING FACTOR (PAF)

ARACHIDONIC ACID METABOLITES

HUMORAL DEFENSE SYSTEMS
- Complement
- Kinins
- Coagulation

OTHERS
- Myocardial Depressant Substance (MDS)
- Endorphins
- Histamine

SEVERE DECREASE SVR

HYPOTENSION

DEPRESSED CO

DEATH

CARDIOVASCULAR INSUFFICIENCY

MOSF

MULTIPLE ORGAN SYSTEM FAILURE

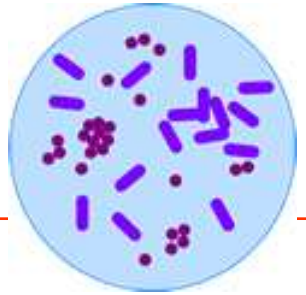
RECOVERY

MYOCARDIUM
- Depression
- Dilatation

VASCULATURE
- Vasodilatation
- Vasoconstriction
- Maldistribution of Blood Flow
- Endothelial Destruction

XOC SÈPTIC

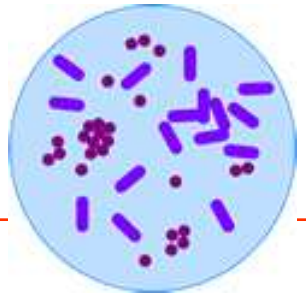
ETIOLOGIA



- FOCUS:
  - Genitourinari
  - Respiratori
  - Gastrointestinal
  - Pell
  - Ferides
  - Catéters

XOC SÈPTIC

ETIOLOGIA



- FOCUS:
  - Genitourinari
  - Respiratori
  - Gastrointestinal
  - Pell
  - Ferides
  - Catéters

- Organismes:
  - BGN (>80%): *E. Coli, Pseudomona, Klebsiella…*
  - Stafilococ Aureus
  - Streptococ pneumoniae

RESISTENCIA

Virulencia

MARSA (*)
BLEE
PSEUDOMONA MR
ACINETOBACTER
STENOTROPHOMONA
E. FAECIUM….

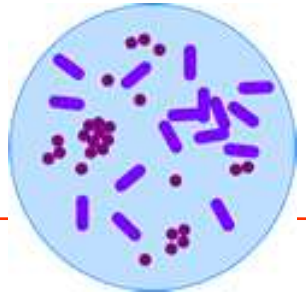
RESISTENCIA

VIRULENCIA

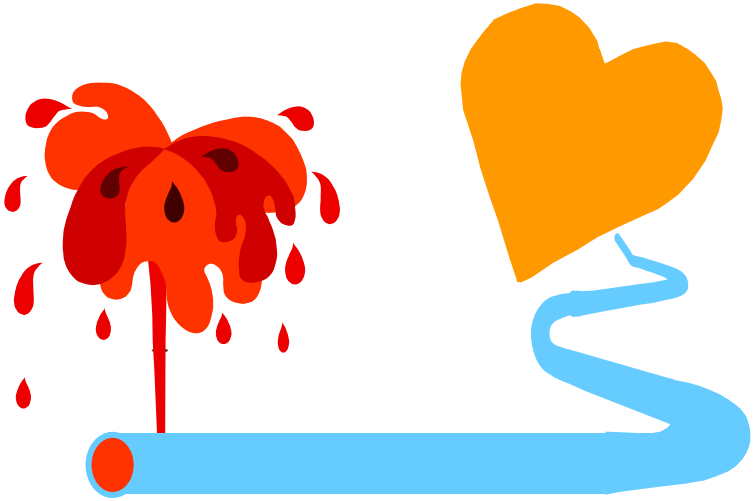
E. COLI

XOC SÈPTIC

FISIOPATOLOGIA



- Hipovolemia
- Disfunció miocàrdica
- Hipoxia tissular



XOC SÈPTIC

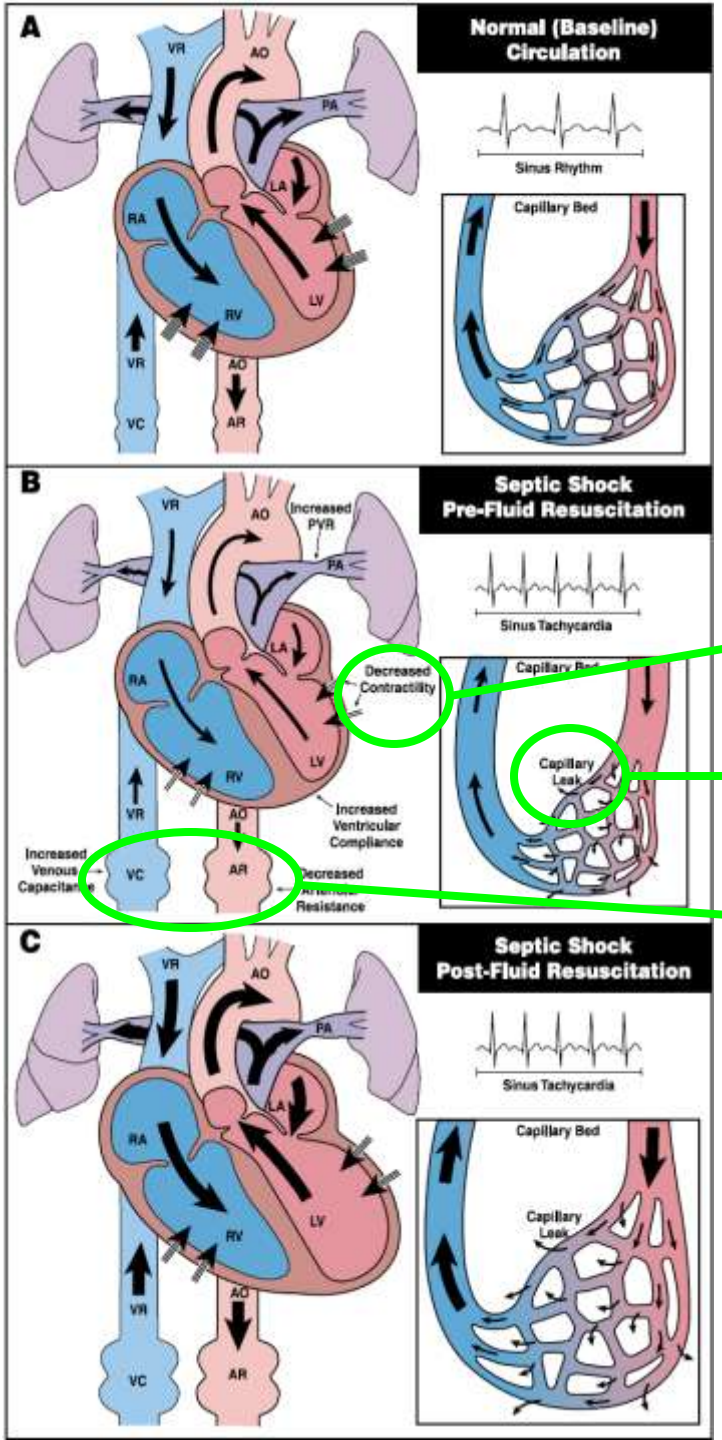
Causes HIPOVOLÈMIA

- Dèficit de Volum Previ
- Exudació Capilar
- Tercer Espai
- Vasodilatació mediada per l'Òxid Nítric

XOC SÈPTIC

Disfunció Miocàrdica

- Biventricular
- Dilatació ventricular i ↓ F.E.
- Reversible
- Indicador de bon pronòstic si dilatació ventricular
- Causes:
  - isquèmia miocàrdica
  - mediadors humorals:
    - Substància depresora del miocardi (MDS)
    - Tumor necrosis factor (TNF)
    - Interleuquines ?
    - Endotoxina ?



Cardiovascular management of septic shock

R. Phillip Dellinger, MD

Crit Care Med 2003 Vol. 31, No. 3

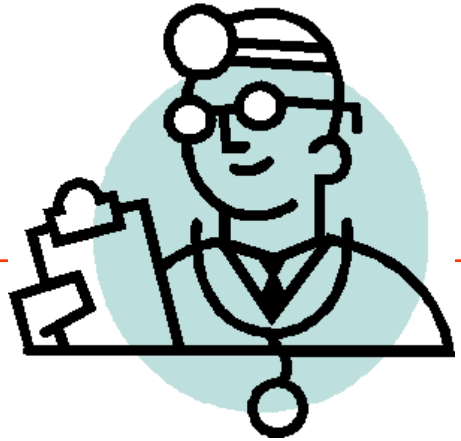
Depresión miocárdica

Hipovolemia

Vasodilatación

XOC SÈPTIC

CLINICA

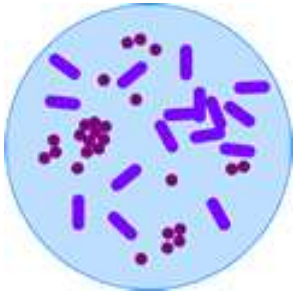


- Febre
- Hiperventilació (per efecte de l'endotoxina sobre el SNC)
- Taquicardia
- Hipotensió
- Hipoperfusió tisular:
  - Ronyo → oliguria
  - SNC → obnubilació
  - Cor → isquèmia coronària
  - Gastrointestinal → isquemia intestinal i hepàtica
  - Pell → fredor cutània

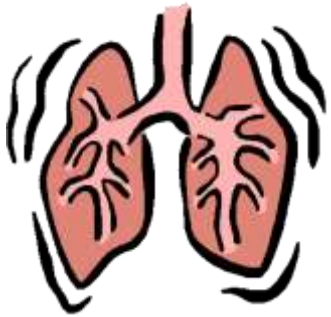
XOC SÈPTIC

COMPLICACIONS

- Respiratòries
  - Alteració V/Q
  - Fatiga Diafragmàtica
  - SDRA
- Insuficiència Renal
- CID
- Fracàs Multiorgànic (M.O.F.)



Sepsis i SDRA



Sepsis

↓

Mediadors imflamatoris: LPS, citoquines, TNF,...

↓

Acumulació neutròfils a l'endoteli i interstici pulmonar

↓

Activació neutròfils

↓

Alliberament metabolits oxidatius i enzims proteolítics

↓

Lesió epitelial i endotelial

↓

SDRA

Tractament xoc sèptic

Intensive Care Med (2013) 39:165–228
DOI 10.1007/s00134-012-2769-8

GUIDELINES

Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock, 2012

R. P. Dellinger
Mitchell M. Levy
Andrew Rhodes
Djillali Annane
Herwig Gerlach
Steven M. Opal
Jonathan E. Sevransky
Charles L. Sprung
Ivor S. Douglas
Roman Jaeschke
Tiffany M. Osborn
Mark E. Nunnally
Sean R. Townsend
Konrad Reinhart
Ruth M. Kleinpell
Derek C. Angus
Clifford S. Deutschman
Flavia R. Machado
Gordon D. Rubenfeld
Steven Webb
Richard J. Beale
Jean-Louis Vincent
Rui Moreno
The Surviving Sepsis Campaign Guidelines Committee
including The Pediatric Subgroup*

Tractament

Table 1 Grading system

Grading recommendations	
A.	Supported by at least 2 level I investigations
B.	Supported by 1 level I investigation
C.	Supported by level II investigations only
D.	Supported by at least 1 level III investigation
E.	Supported by level IV or V evidence
Grading of evidence	
I.	Large, randomized trials with clearcut results; low risk of false-positive (alpha) error or false-negative (beta) error
II.	Small, randomized trials with uncertain results; moderate-to-high risk of false-positive (alpha) and/or false-negative (beta) error
III.	Non-randomized, contemporaneous controls
IV.	Non-randomized, historical controls and expert opinion
V.	Case series, uncontrolled studies, and expert opinion

Antecedents

Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock

R. Phillip Dellinger, MD; Jean M. Carlet, MD; Henry Masur, MD; Herwig Gerlach, MD, PhD; Thierry Calandra, MD; Jonathan Cohen, MD; Juan Gea-Banacloche, MD, PhD; Didier Keh, MD; John C. Marshall, MD; Margaret M. Parker, MD; Graham Ramsay, MD; Janice L. Zimmerman, MD; Jean-Louis Vincent, MD, PhD; Mitchell M. Levy, MD; for the Surviving Sepsis Campaign Management Guidelines Committee

Sponsoring Organizations: American Association of Critical-Care Nurses, American College of Chest Physicians, American College of Emergency Physicians, American Thoracic Society, Australian and New Zealand Intensive Care Society, European Society of Clinical Microbiology and Infectious Diseases, European Society of Intensive Care Medicine, European Respiratory Society, International Sepsis Forum, Society of Critical Care Medicine, Surgical Infection Society.

Crit Care Med 2004 Vol. 32, No. 3

Intensive Care Med (2008) 34:17–60
DOI 10.1007/s00134-007-0934-2

SPECIAL ARTICLE

R. Phillip Dellinger
Mitchell M. Levy
Jean M. Carlet
Julian Bion
Margaret M. Parker
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Konrad Reinhart
Derek C. Angus
Christian Brun-Buisson
Richard Beale
Thierry Calandra
Jean-Francois Dhainaut
Herwig Gerlach
Maurene Harvey
John J. Marini
John Marshall
Marco Ranieri
Graham Ramsay
Jonathan Sevransky
B. Taylor Thompson
Sean Townsend
Jeffrey S. Vender
Janice L. Zimmerman
Jean-Louis Vincent

Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008

Antecedents

- Sepsis severa i shock sèptic

- Alta prevalença

- Alta mortalitat

- Incidència en augment

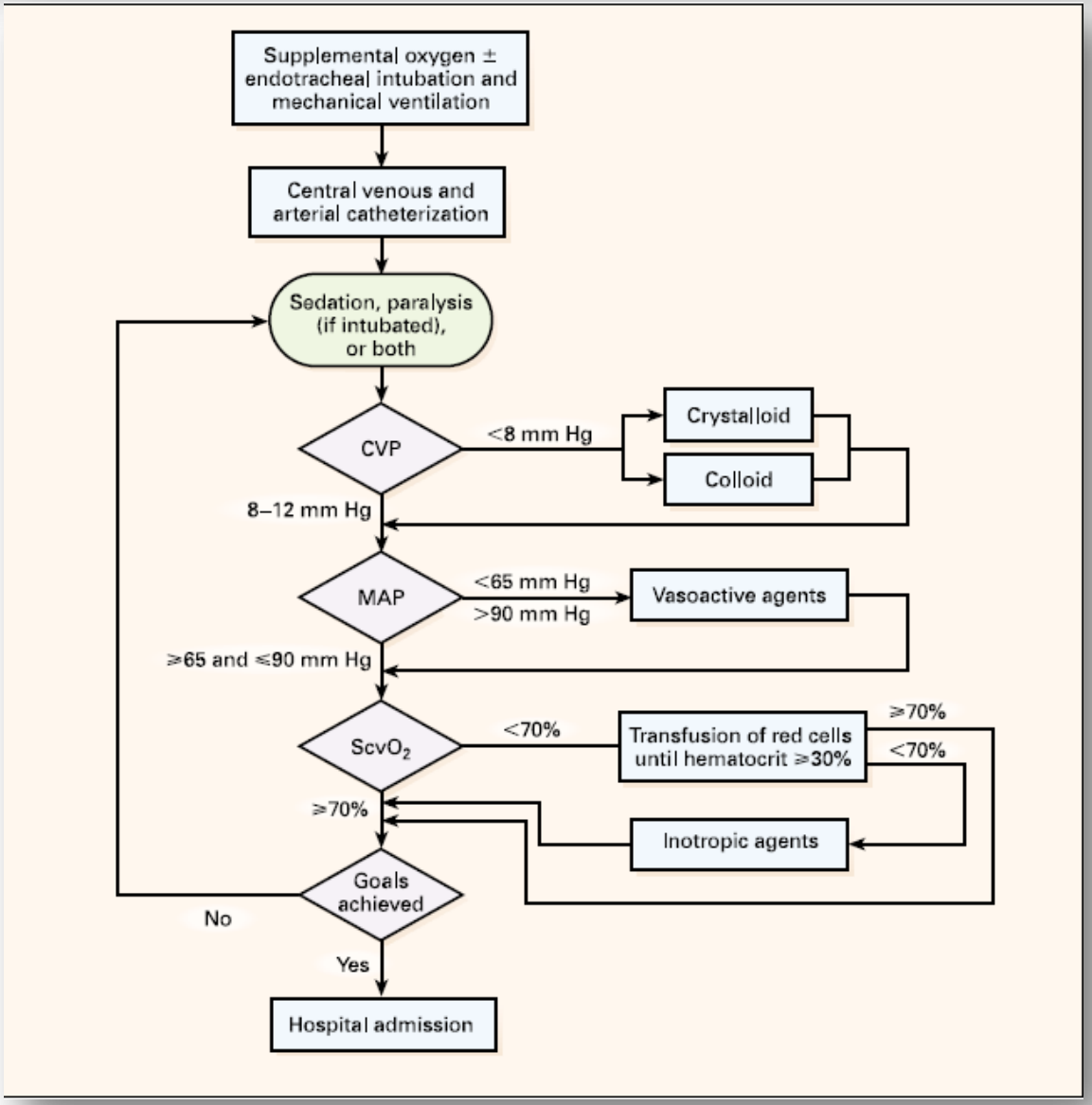
"Golden hours"

A. Ressucitació inicial

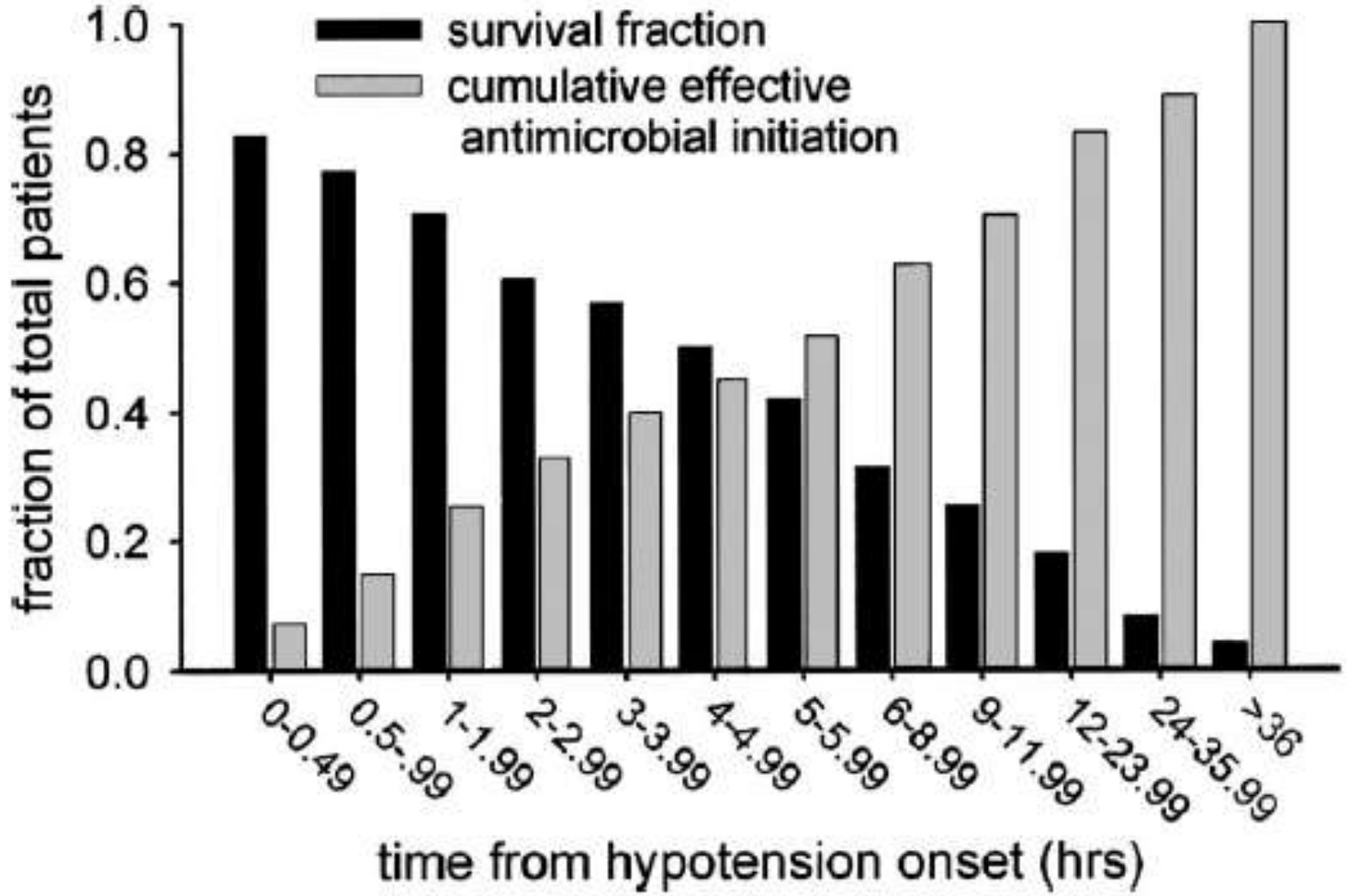
A. Initial resuscitation

1. We recommend the protocolized, quantitative resuscitation of patients with sepsis- induced tissue hypoperfusion (defined in this document as hypotension persisting after initial fluid challenge or blood lactate concentration ≥4 mmol/L). This protocol should be initiated as soon as hypoperfusion is recognized and should not be delayed pending ICU admission. During the first 6 h of resuscitation, the goals of initial resuscitation of sepsis-induced hypoperfusion should include all of the following as a part of a treatment protocol (grade 1C):
   (a) CVP 8–12 mmHg ?
   (b) MAP ≥65 mmHg
   (c) Urine output ≥0.5 mL kg h^{-1}
   (d) Superior vena cava oxygenation saturation ($ScvO_2$) or mixed venous oxygen saturation (Svo_2) 70 or 65 %, respectively.
2. We suggest targeting resuscitation to normalize lactate in patients with elevated lactate levels as a marker of tissue hypoperfusion (grade 2C).

EARLY GOAL-DIRECTED THERAPY IN THE TREATMENT OF SEVERE SEPSIS AND SEPTIC SHOCK



(Rivers E, N Engl J Med, 2001)



(Crit Care Med 2006; 34:1589–1596)

SSC 2012: Hemodinamia. ***Fluidos***

- *De preferencia:* ***Cristaloides*** *hasta un máximo de 30 ml/kg en Sepsis Severa*
  - *Con hipoperfusión órgano +*
  - *Sospecha de hipovolemia*

- ***No recomiendan coloides (HES)***

Intensive Insulin Therapy and Pentastarch Resuscitation in Severe Sepsis	N Engl J Med 2008;358:125-39.
Assessment of hemodynamic efficacy and safety of 6% hydroxyethylstarch 130/0.4 vs. 0.9% NaCl fluid replacement in patients with severe sepsis: The CRYSTMAS study	Guidet *et al. Critical Care* 2012, **16**:R94
Hydroxyethyl Starch 130/0.42 versus Ringer's Acetate in Severe Sepsis	N Engl J Med 2012;367:124-34.
Hydroxyethyl Starch or Saline for Fluid Resuscitation in Intensive Care	N Engl J Med 2012;367:124-34.

- ***Albúmina*** *cuando se requieran de grandes cantidades de cristaloides*

Líquids

G. Fluid therapy of severe sepsis

1. We recommend crystalloids be used as the initial fluid of choice in the resuscitation of severe sepsis and septic shock (grade 1B).
2. We recommend against the use of hydroxyethyl starches (HES) for fluid resuscitation of severe sepsis and septic shock (grade 1B). (This recommendation is based on the results of the VISEP [128], CRYSTMAS [122], 6S [123], and CHEST [124] trials. The results of the recently completed CRYSTAL trial were not considered.)
3. We suggest the use of albumin in the fluid resuscitation of severe sepsis and septic shock when patients require substantial amounts of crystalloids (grade 2C).

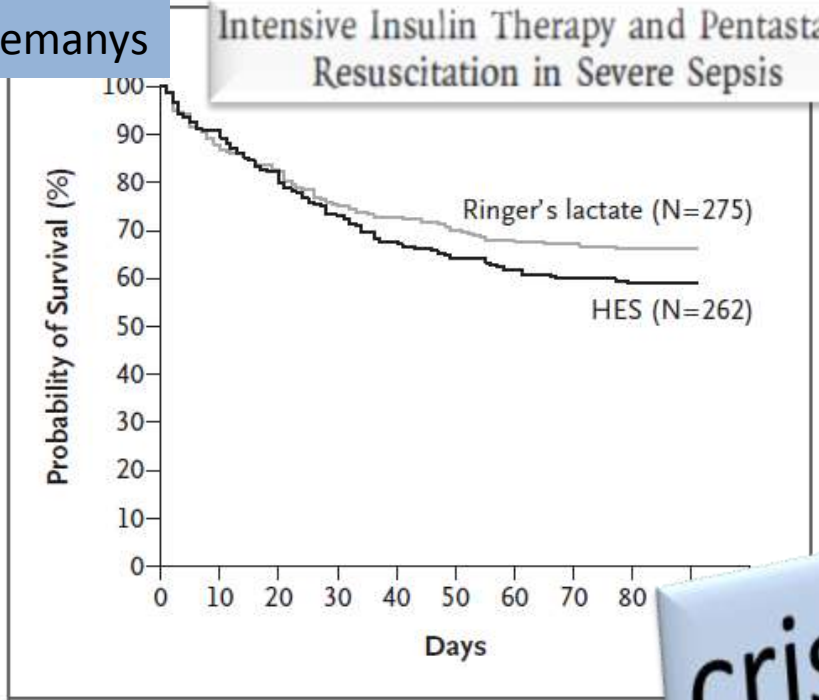
→ *fins 30ml/kg*

RECOMANEN FER FLUIDS CHALLENGE !!

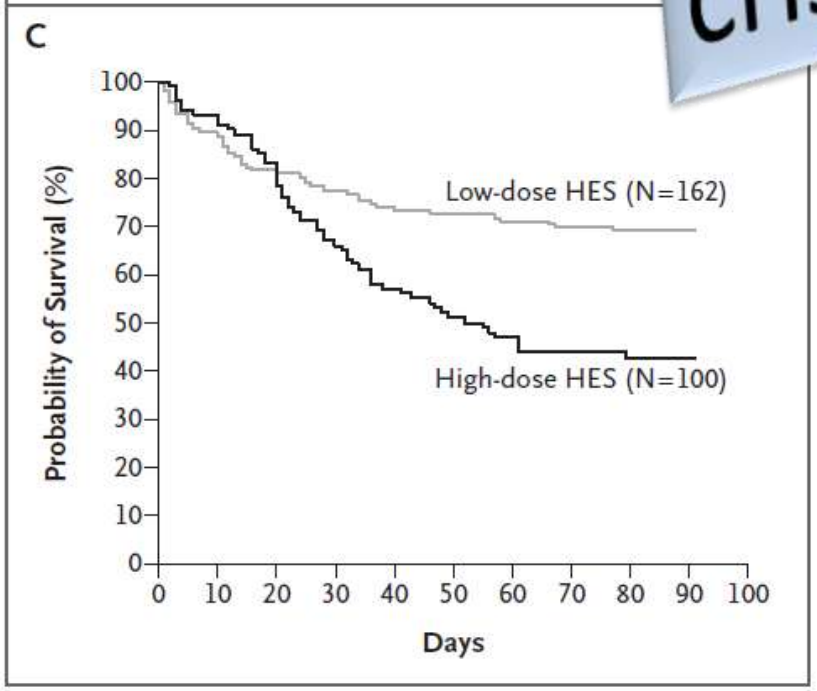
Sépticos

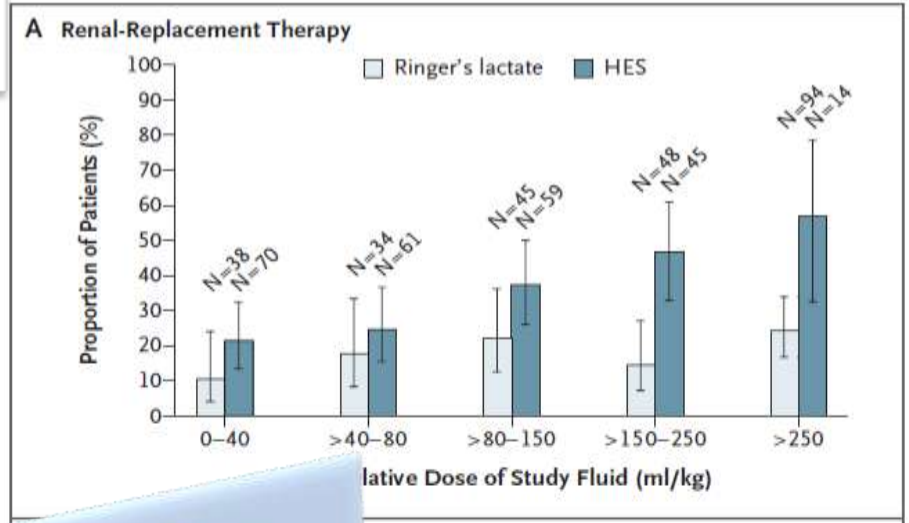
	Alemán-VISEP	Scandinavo-6S	Francés-CRYSTMAS
	NEMJ-2008	**NEMJ-2012**	**Crit Care-2012**
Pacientes	Médico-quirúrgicos	Médico-quirúrgicos	Médico-quirúrgicos
Tipo	**Sépticos**	**Sépticos**	**Sépticos**
Cristaloide	R-Lactato	R-Acetato	SS 0,9%
Coloide	HES (200/0,5, 10%)	HES (130/0,4, 6%)	HES (130/0,4, 6%)
Rip 28 d	=	=	=
Rip 90 d	= (0.09)	↑ **HES**	=
Inf. Renal	↑ HES	= (0.08)	=
T S Renal	↑ HES	↑ HES	=
Coag /sangrado	↑ HES	↑ HES	=
Comentarios	Underpowered Estudio “raro” ***38% HES >20 ml/kg/d***	Ya reanimados Estabilización = Coloids hemodiluyen	Underpowered Tiempo Excluyen IR severa **HMD+rápida (2,5 h)**

Alemanys



cristaloides





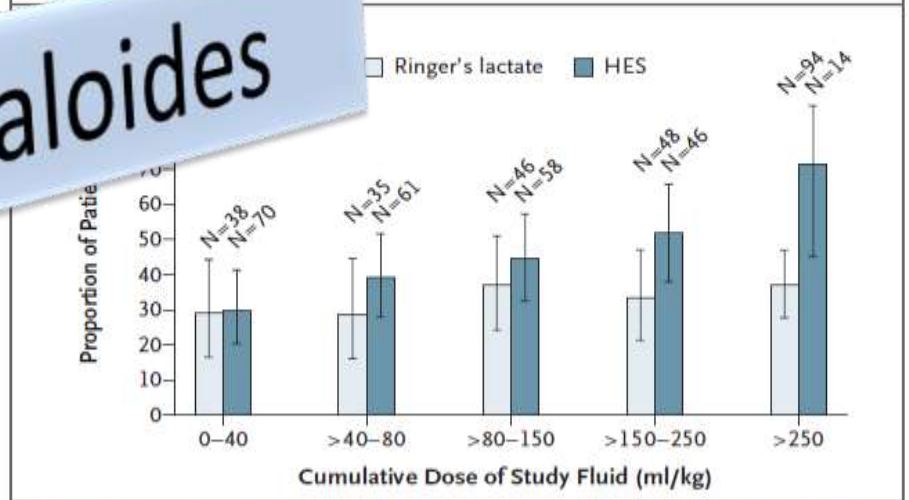


Figure 3. Cumulative Effect of Volume Resuscitation on the Need for Renal-Replacement Therapy and the Rate of Death at 90 Days.

Panel A shows the relationship between the cumulative dose of either pentastarch (HES) or Ringer's lactate and the percentage of patients who needed renal-replacement therapy (Panel A) and the rate of death at 90 days (Panel B). The need for renal-replacement therapy and 90-day mortality were significantly correlated with the cumulative dose of HES ($P<0.001$ and $P=0.001$, respectively) but not with the dose of Ringer's lactate ($P=0.11$ and $P=0.31$, respectively). All P values were calculated with the Cochran–Armitage test for trend. I bars denote 95% confidence intervals.

Francesos

Assessment of hemodynamic efficacy and safety of 6% hydroxyethylstarch 130/0.4 vs. 0.9% NaCl fluid replacement in patients with severe sepsis: The CRYSTMAS study

Guidet et al. Critical Care 2012, 16:R94

És igual

Abstract

Introduction: Inadequate initial treatment (HDS) may be associated with increased risk of death in severe sepsis pat

Methods: In order to compare the hemod and safety of 6% HES 130/0.4 and NaCl 0.9% for HDS in patients with severe sepsis, we designed a pective, multicenter, active-controlled, double-blind, randomized study in intensive care units.

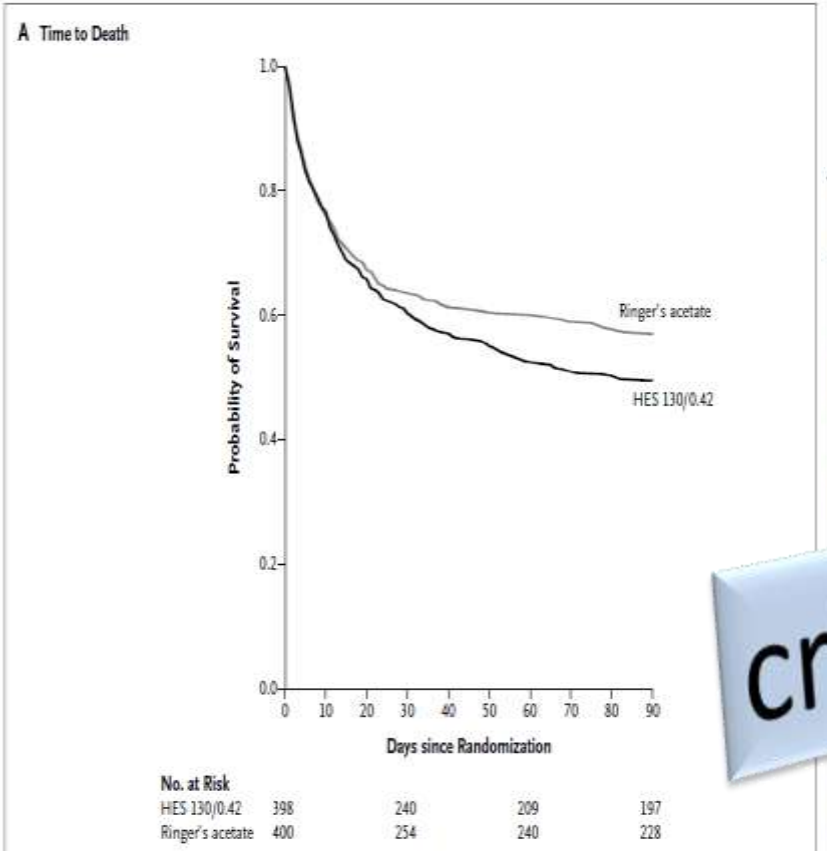
Results: 174 out of 196 patients reached HDS (88 and 86 patients for HES and NaCl, respectively). Significantly less HES was used to reach HDS vs. NaCl (1,379 ±886 ml in the HES group and 1,709 ±1,164 ml in the NaCl group (mean difference = -331± 1,033, 95% CI -640 to -21, $P = 0.0185$). Time to reach HDS was 11.8 10.1 hours vs. 14.3 ±11.1 hours for HES and NaCl, respectively. Total quantity of study drug infused over four consecutive days, ICU and hospital LOS, and area under the curve of SOFA score were comparable. Acute renal failure occurred in 24 (24.5%) and 19 (20%) patients for HES and NaCl, respectively ($P = 0.454$). There was no difference between AKIN and RIFLE criteria among groups and no difference in mortality, coagulation, or pruritus up to 90 days after treatment initiation.

Conclusion: Significantly less volume was required to achieve HDS for HES vs. NaCl in the initial phase of fluid resuscitation in severe sepsis patients without any difference for adverse events in both groups.

ClinicalTrials.gov: NCT00464204

Hydroxyethyl Starch 130/0.42 versus Ringer's Acetate in Severe Sepsis

Escandinaus



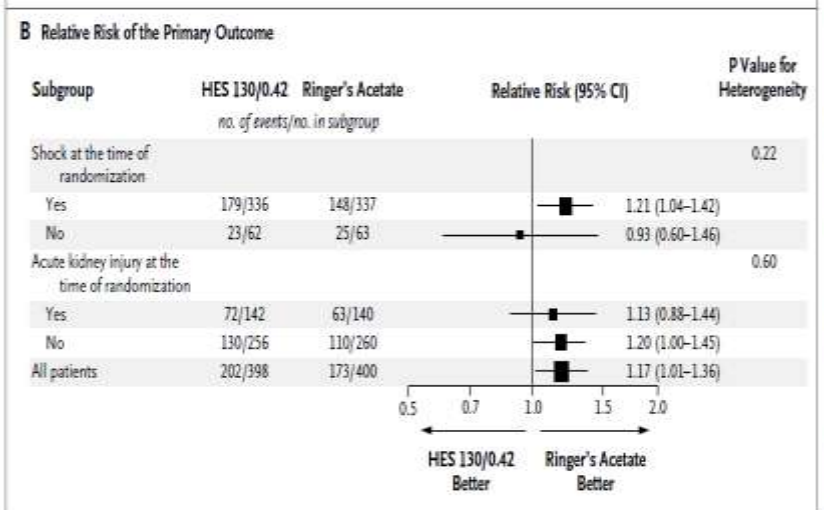


Figure 2. Time to Death and Relative Risk of the Primary Outcome.

Table 3. Primary and Secondary Outcomes.*

Outcome	HES 130/0.42 (N=398)	Ringer's Acetate (N=400)	Relative Risk (95% CI)	P Value
Primary outcome				
Dead or dependent on dialysis at day 90 — no. (%)	202 (51)	173 (43)	1.17 (1.01–1.36)	0.03
Dead at day 90 — no. (%)	201 (51)	172 (43)	1.17 (1.01–1.36)	0.03
D…	1 (0.25)	1 (0.25)	—	1.00
	154 (39)	144 (36)	1.08 (0.90–1.28)	0.43
…— no. (%)†	38 (10)	25 (6)	1.52 (0.94–2.48)	0.09
Severe allergic reaction — no. (%)†	1 (0.25)	0	—	0.32
SOFA score at day 5 — median (interquartile range)	6 (2–11)	6 (0–10)	—	0.64
Use of renal-replacement therapy — no. (%)‡	87 (22)	65 (16)	1.35 (1.01–1.80)	0.04
Use of renal-replacement therapy or renal SOFA score ≥3 — no. (%)§	129 (32)	108 (27)	1.20 (0.97–1.48)	0.10
Doubling of plasma creatinine level — no. (%)†	148 (41)	127 (35)	1.18 (0.98–1.43)	0.08
Acidosis — no. (%)†¶	307 (77)	312 (78)	0.99 (0.92–1.06)	0.72
Alive without renal-replacement therapy — mean % of days‖	91	93	—	0.048
Use of mechanical ventilation — no. (%)†	325 (82)	321 (80)	1.02 (0.95–1.09)	0.61
Alive without mechanical ventilation — mean % of days‖	62	65	—	0.28
Alive and out of hospital — mean % of days‖	29	34	—	0.048

cristaloides

Hydroxyethyl Starch or Saline for Fluid Resuscitation in Intensive Care

Australians

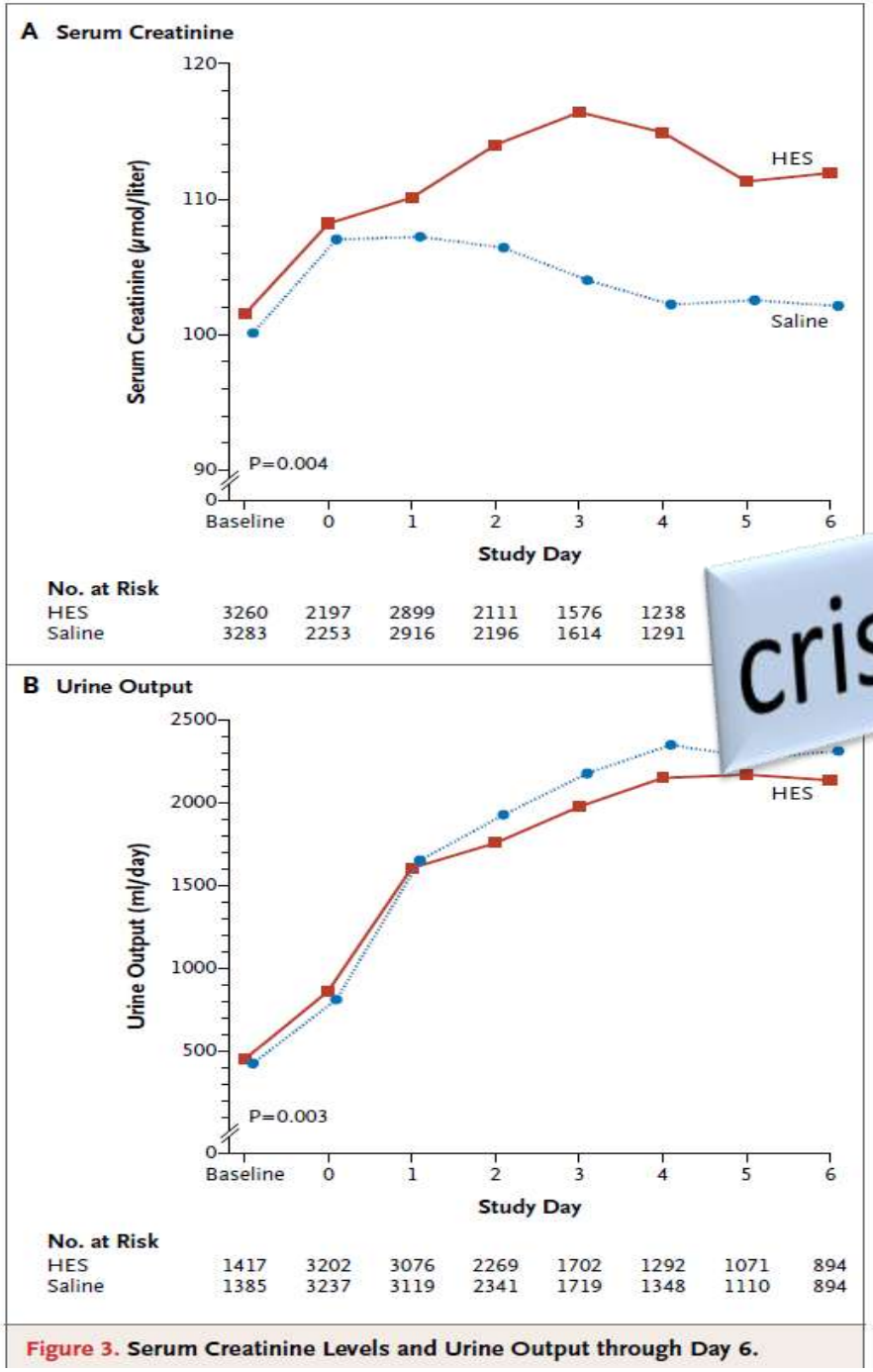
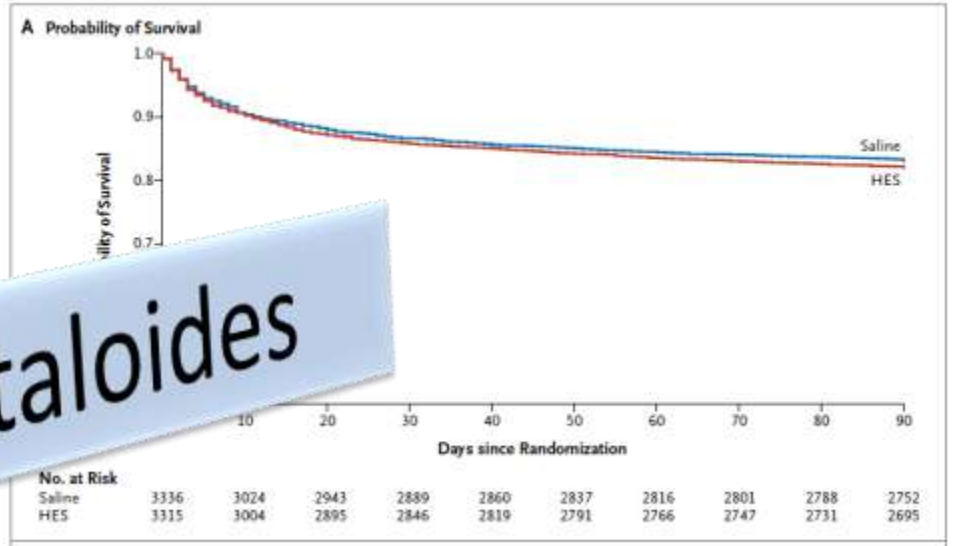


Figure 3. Serum Creatinine Levels and Urine Output through Day 6.

cristaloides



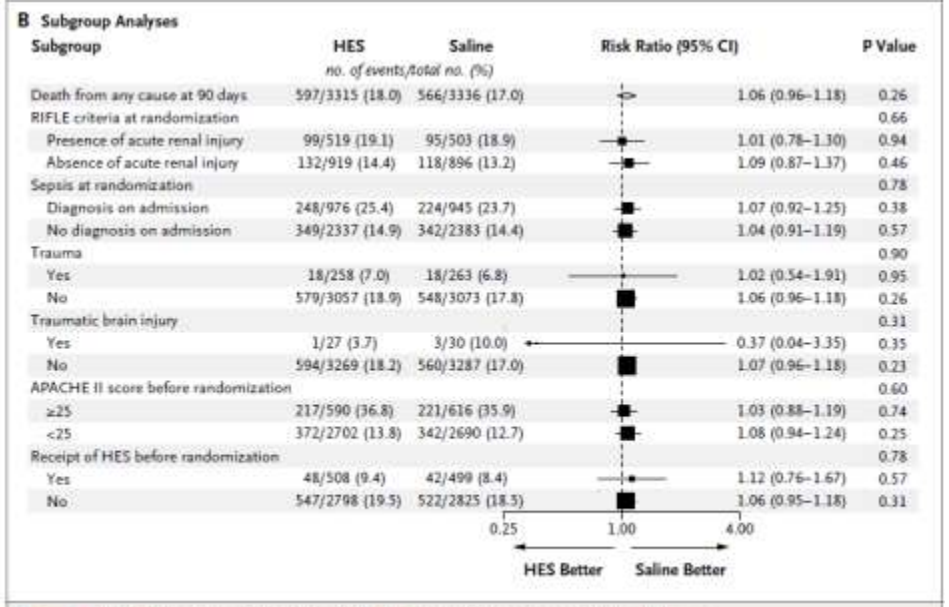


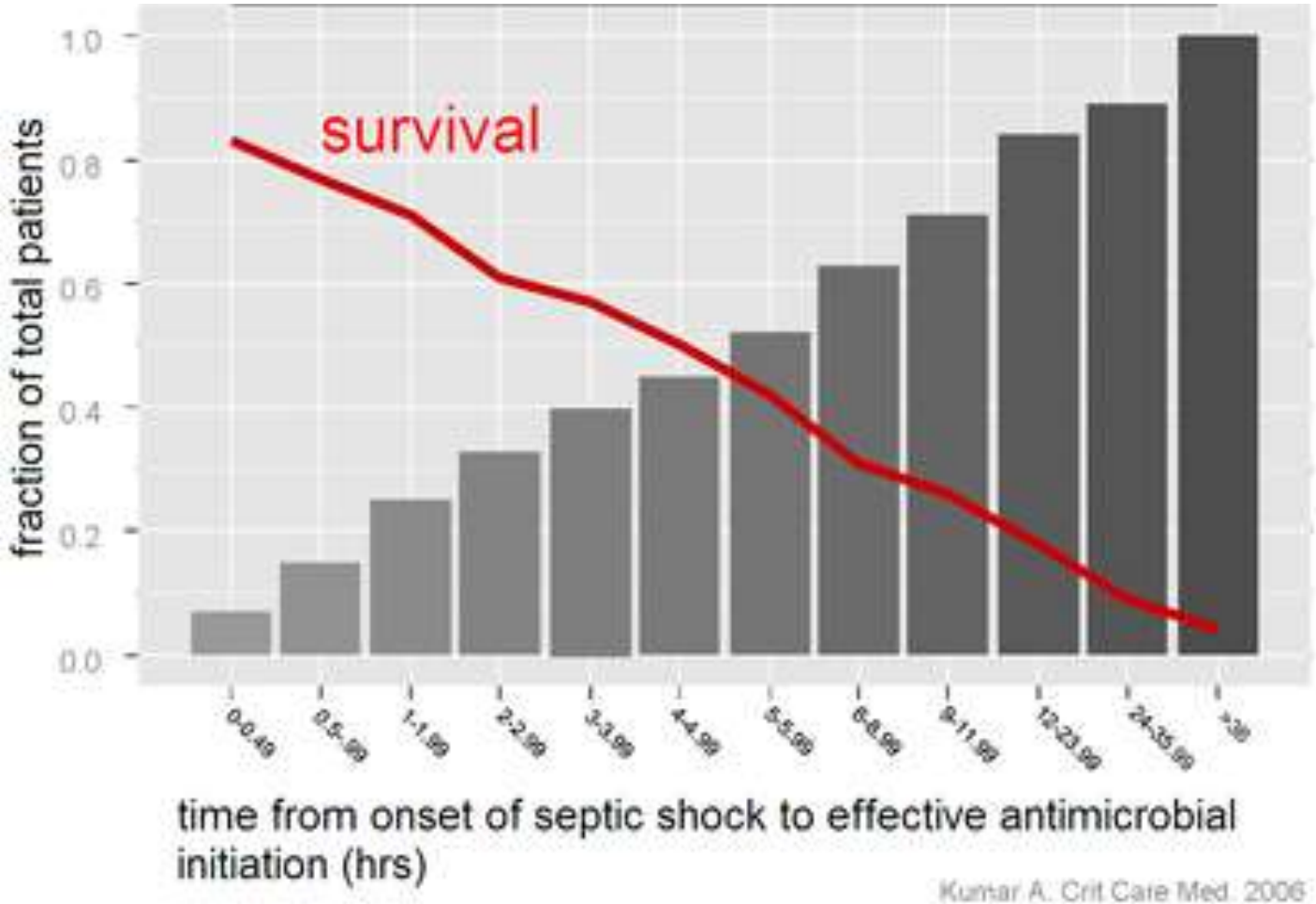
Figure 2. Probability of Survival and the Risk of Death at 90 Days, According to Subgroup.

Diagnòstic. Cultius

- *Hemocultius: abans de començar tractament antibiòtic com a mínim 1 percutani i un altre de cada accés vascular col.locat fa > 48 h*
- *Altres cultius*
- *Sempre que no demori l'inici del tractament*
- *No recomanacions de la utilitat de la procalcitonina o la PCR per diferenciar infecció d'inflamació.*

Tractament antimicrobià

- *Tractament antibiòtic ev efectiu durant la primera hora*
  (cada hora de retràs s'associa a un augment de mortalitat)
- *Antibiòtics d'ampli espectre i adequats per la localització de la infecció.*
- *Desescalar quan sigui possible: evaluar diariament*
- *Si posteriorment no hi ha evidència d'infecció, guiar-se a través de nivells baixos de procalcitonina per suspendre AB empírics*
- *Teràpia combinada empírica no més de 3-5 dies*
- *Durada tractament: 7-10 dies*



Kumar A. Crit Care Med. 2006

D. Tractament antimicrobià

Doble cobertura
- ***Ps. Aeuroginosa***
- ***Acineto***
- ***Sepsis grave***

4a. Empiric therapy should attempt to provide antimicrobial activity against the most likely pathogens based upon each patient's presenting illness and local patterns of infection. We suggest combination empiric therapy for neutropenic patients with severe sepsis (grade 2B) and for patients with difficult-to- treat, multidrug-resistant bacterial pathogens such as *Acinetobacter* and *Pseudomonas spp.* (grade 2B). For selected patients with severe infections associated with respiratory failure and septic shock, combination therapy with an extended spectrum beta-lactam and either an aminoglycoside or a fluoroquinolone is suggested for *P. aeruginosa* bacteremia (grade 2B). Similarly, a more complex combination of beta-lactam and a macrolide is suggested for patients with septic shock from bacteremic *Streptococcus pneumoniae* infections (grade 2B).

Control de focus

E. Source control

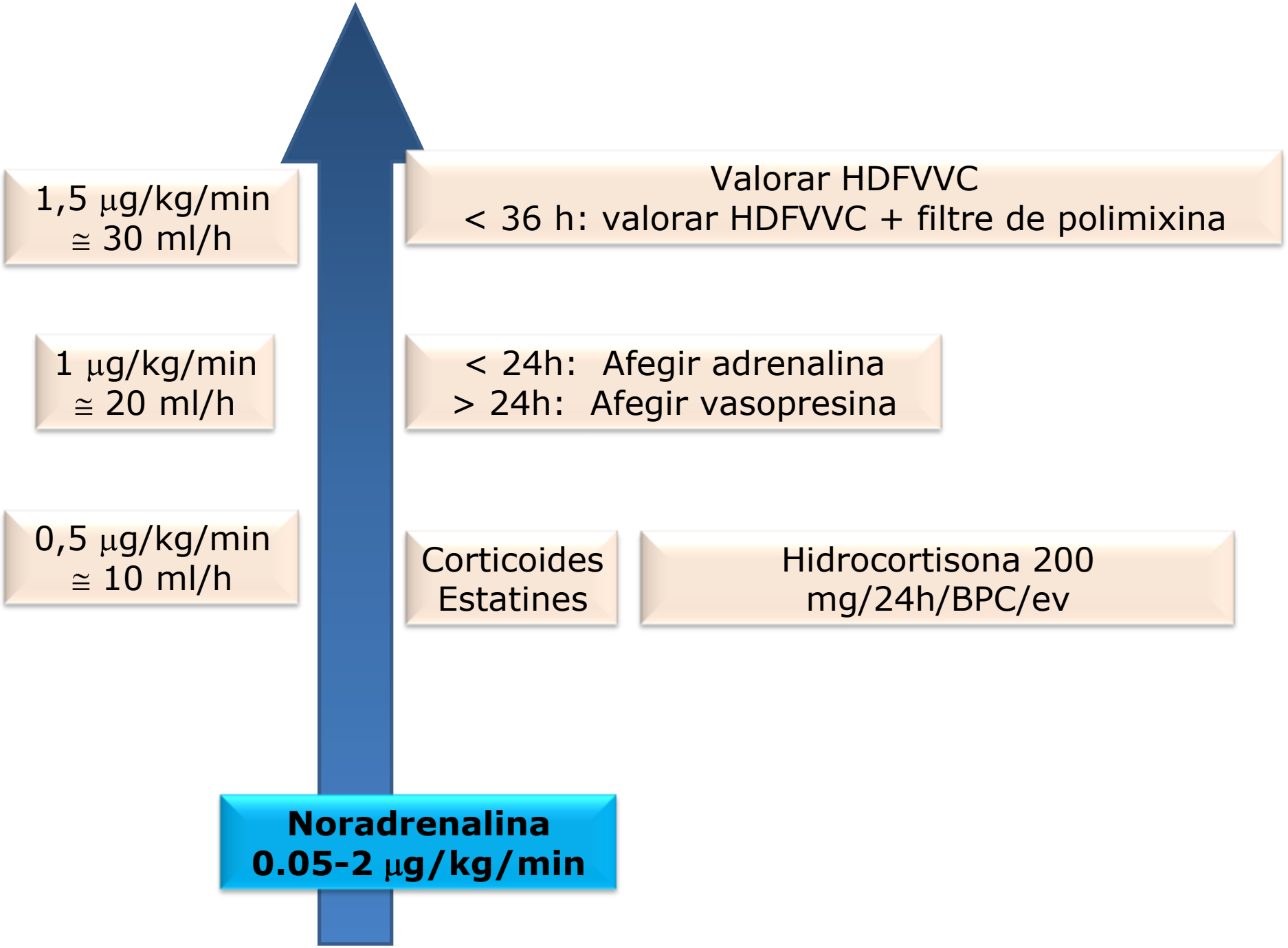
1. We recommend that a specific anatomical diagnosis of infection requiring consideration for emergent source control (e.g., necrotizing soft tissue infection, peritonitis, cholangitis, intestinal infarction) be sought and diagnosed or excluded as rapidly as possible, and intervention be undertaken for source control within the first 12 h after the diagnosis is made, if feasible (grade 1C).
2. We suggest that when infected peripancreatic necrosis is identified as a potential source of infection, definitive intervention is best delayed until adequate demarcation of viable and nonviable tissues has occurred (grade 2B).
3. When source control in a severely septic patient is required, the effective intervention associated with the least physiologic insult should be used (e.g., percutaneous rather than surgical drainage of an abscess) (UG).
4. If intravascular access devices are a possible source of severe sepsis or septic shock, they should be removed promptly after other vascular access has been established (UG).

H. Vasopressors

H. Vasopressors

1. We recommend that vasopressor therapy initially target a MAP of 65 mmHg (grade 1C).
2. We recommend norepinephrine as the first-choice vasopressor (grade 1B).
3. We suggest epinephrine (added to and potentially substituted for norepinephrine) when an additional agent is needed to maintain adequate blood pressure (grade 2B).
4. Vasopressin (up to 0.03 U/min) can be added to norepinephrine with the intent of raising MAP to target or decreasing norepinephrine dosage (UG).
5. Low-dose vasopressin is not recommended as the single initial vasopressor for treatment of sepsis-induced hypotension, and vasopressin doses higher than 0.03–0.04 U/min should be reserved for salvage therapy (failure to achieve an adequate MAP with other vasopressor agents) (UG).
6. We suggest dopamine as an alternative vasopressor agent to norepinephrine only in highly selected patients (e.g., patients with low risk of tachyarrhythmias and absolute or relative bradycardia) (grade 2C).
7. Phenylephrine is not recommended in the treatment of septic shock except in the following circumstances: (a) norepinephrine is associated with serious arrhythmias, (b) cardiac output is known to be high and blood pressure persistently low, or (c) as salvage therapy when combined inotrope/vasopressor drugs and low-dose vasopressin have failed to achieve the MAP target (grade 1C).
8. We recommend that low-dose dopamine not be used for renal protection (grade 1A).

→ *TAM > 65mmHg si HTA previa*


1,5 μg/kg/min
≅ 30 ml/h
Valorar HDFVVC
< 36 h: valorar HDFVVC + filtre de polimixina
1 μg/kg/min
≅ 20 ml/h
< 24h: Afegir adrenalina
> 24h: Afegir vasopresina
0,5 μg/kg/min
≅ 10 ml/h
Corticoides
Estatines
Hidrocortisona 200
mg/24h/BPC/ev
Noradrenalina
0.05-2 μg/kg/min

H. Vasopressors

- **Adrenalina**
  - Augmenta nivells de làctic per estimulació d'adrenoreceptor β-2 muscular
  - Invalida làctic per seguir evolució del shock

- **Fenilefrina**
  - Arritmies greus si associat amb noradrenalina
  - GC alt amb TAM persistentement baixa
  - Indicat quan tota la resta fallen

- **Vasopresina**
  - Nivells alts les primeres 24-48 h de shock
  - Nivells baixos en shock persistent *(relative vasopresin deficiency*)
  - Isquèmia cardíaca, digital i esplàcnica

- **Dopamina**
  - Només en bradicardies basals i/o pacients amb baix risc arritmogènic

I. Inotrops

I. Inotropic therapy

1. We recommend that a trial of dobutamine infusion up to 20 μg kg^{-1} min^{-1} be administered or added to vasopressor (if in use) in the presence of: (a) myocardial dysfunction, as suggested by elevated cardiac filling pressures and low cardiac output, or (b) ongoing signs of hypoperfusion, despite achieving adequate intravascular volume and adequate MAP (grade 1C).
2. We recommend against the use of a strategy to increase cardiac index to predetermined supranormal levels (grade 1B).

Ecocardio?

Levosimendan?

Fármac	Dosis/ dilución	Acción	Estimulación Cardíaca	VSC	VSD	CO	Comentarios
Norepinefrina	0.05-2μg/kg/min 50 mgr/250 ml	α-1 β-1 (algo)	++	++++	0	↑/0	↓FC Util en vasodilatacion ↓ Circulación coronaria
Dopamina	0,5-20 μg/kg/min 400 mgr/200 ml	dopa < 5 β: 5-10 α > 10 μg/kg/min	++ a 5-10 μg/kg/min	++ a 7 μg/kg/min	+ a 0.5-5.0 μg/kg/min	↑	Taquicarritmias ↑CMO2 VSD coronaria, renal, cerebral, mesenterio
Fenilefrina	40-200 μg/min 10 mgr/250 ml	α	0	++++	0	↓	↓FC, cefalea, inquietud, ok para taquicardia y/o taquiaritmias supra ventric
Vasopresina	0.01-0,04 U/min 20 U/100 ml	α y V1	0	++++	+	↓	No pasar 0.04 Isquemias variadas
Epinefrina	0.1-0.5 μg/kg/min 1 mgr/250 ml	α, β	++ ++ a 0.03-0,15 μg/kg/min	++ ++ a 0.15-0.30 μg/kg/min	+++	↑	Taquidisrritmias, ↑CMO2, leucocitosis, ↑Láctico
Dobutamina	2,5-20 μg/kg/min 500mgr/250 ml	β-1, algo β-2 y α-1 dosis ↑	++++	+	++	↑	Taquiaritmias, ↑CMO2 Hipotension en hipovolemicos < VSC periferica que dopa
NTG	5-60 μg/min 50 mg/250 ml	Relx musc Coronaria y sistemica	0	0	+++	↓/0	Cefalea, ↑FC, hipersensibilidad

J. Corticoides

Hidrocortisona

- *No, si amb líquids i vasopresors aconseguim estabilitat hemodinàmica*
- *Dosis de 200 mg/día ev en BPC (menys hiperglicemia i hiperNa)*
- *No afegir fludrocortisona*
- *Disminució progressiva quan haguem suspès vasopressors*
- *No realitzar test d'ACTH*

J. Corticoides

Systemic Steroids in Severe Sepsis and Septic Shock

Gourang P. Patel[1] and Robert A. Balk[1]

Am J Respir Crit Care Med Vol 185, Iss. 2, pp 133–139, Jan 15, 2012

Study	Study (*N*)	Study Population	Steroid Treatment	Primary Outcome	28-Day Placebo Mortality (%)	28-Day Treatment Mortality (%)	Conclusion
Bollaert (41)	41	Vasopressor-dependent septic shock on ventilator for >48 h	Hydrocortisone 100 mg q8h for 5 d, then wean over 6 d	Shock reversal	63	32	Hydrocortisone treatment significantly improved hemodynamic abnormalities of septic shock
Briegel (42)	40	Vasopressor-dependent septic shock on ventilator	Hydrocortisone 100 mg load, then 0.18 mg/kg/h continuous infusion until reversal of shock, then wean over 6 d	Shock reversal	30	20	Hydrocortisone treatment significantly decreased time to cessation of vasopressor treatment
Yildiz (43)	40	Patients with sepsis (ACCP-SCCM criteria) (52)	Prednisolone 5 mg at 06:00 and 2.5 mg at 18:00 for 10 d	28-d All-cause mortality	60	40	Trend toward decreased mortality with physiological-dose steroid treatment
Annane (29)	300	Vasopressor-dependent septic shock	Hydrocortisone 50 mg q6h for 7 d and fludrocortisone 50 μg daily for 7 d	28-d Survival distribution from randomization in nonresponders	63	53	Hydrocortisone treatment significantly improved survival and shock reversal in nonresponders to ACTH stimulation test
Oppert (46)	40	Vasopressor-dependent septic shock	Hydrocortisone 50 mg bolus, then 0.18 mg/kg/h until vasopressor discontinued, then wean to 0.06 mg/kg/h for 24 h, then reduced by 0.02 mg/kg/h/d until off	Time to vasopressor discontinuation	48	39	Hydrocortisone treatment significantly improved shock reversal and decreased level of proinflammatory cytokines
Sprung (48)	499	Septic shock	Hydrocortisone 50 mg q6h for 5 d then 50 mg q12h for 3 d, then 50 mg q24h for 3 d	28-d Mortality rate in nonresponders to ACTH stimulation test	36.1 in Nonresponder, 31.5 overall	39.2 in Nonresponder, 34.3 overall	Hydrocortisone treatment did not significantly improve 28-d survival or shock reversal in septic shock nonresponders to ACTH stimulation test

K. Altres teràpies

K. Blood product administration

1. Once tissue hypoperfusion has resolved and in the absence of extenuating circumstances, such as myocardial ischemia, severe hypoxemia, acute hemorrhage, or ischemic coronary artery disease, we recommend that red blood cell transfusion occur when the hemoglobin concentration decreases to <7.0 g/dL to target a hemoglobin concentration of 7.0–9.0 g/dL in adults (grade 1B).

2. We recommend not using erythropoietin as a specific treatment of anemia associated with severe sepsis (grade 1B).

3. We suggest that fresh frozen plasma not be used to correct laboratory clotting abnormalities in the absence of bleeding or planned invasive procedures (grade 2D).

4. We recommend against antithrombin administration for the treatment of severe sepsis and septic shock (grade 1B).

5. In patients with severe sepsis, we suggest that platelets be administered prophylactically when counts are $\leq 10{,}000/mm^3$ ($10 \times 10^9/L$) in the absence of apparent bleeding, as well when counts are $\leq 20{,}000/mm^3$ ($20 \times 10^9/L$) if the patient has a significant risk of bleeding. Higher platelet counts [$\geq 50{,}000/mm^3$ ($50 \times 10^9/L$)] are advised for active bleeding, surgery, or invasive procedures (grade 2D).

Altres teràpies

O. Mechanical ventilation of sepsis-induced acute respiratory distress syndrome (ARDS)

1. Target a tidal volume of 6 mL/kg predicted body weight in patients with sepsis-induced ARDS (grade 1A vs. 12 mL/kg).
2. Plateau pressures be measured in patients with ARDS and initial upper limit goal for plateau pressures in a passively inflated lung be $\leq$30 cm H_2O (grade 1B).
3. Positive end-expiratory pressure (PEEP) be applied to avoid alveolar collapse at end expiration (atelectotrauma) (grade 1B).
4. Strategies based on higher rather than lower levels of PEEP be used for patients with sepsis- induced moderate or severe ARDS (grade 2C).
5. Recruitment maneuvers be used in sepsis patients with severe refractory hypoxemia (grade 2C).
6. Prone positioning be used in sepsis-induced ARDS patients with a PaO_2/FIO_2 ratio $\leq$100 mm Hg in facilities that have experience with such practices (grade 2B).
7. That mechanically ventilated sepsis patients be maintained with the head of the bed elevated to 30–45 degrees to limit aspiration risk and to prevent the development of ventilator-associated pneumonia (grade 1B).
8. That noninvasive mask ventilation (NIV) be used in that minority of sepsis-induced ARDS patients in whom the benefits of NIV have been carefully considered and are thought to outweigh the risks (grade 2B).
9. That a weaning protocol be in place and that mechanically ventilated patients with severe sepsis undergo spontaneous breathing trials regularly to evaluate the ability to discontinue mechanical ventilation when they satisfy the following criteria: a) arousable; b) hemodynamically stable (without vasopressor agents); c) no new potentially serious conditions; d) low ventilatory and end-expiratory pressure requirements; and e) low Fio_2 requirements which can be met safely delivered with a face mask or nasal cannula. If the spontaneous breathing trial is successful, consideration should be given for extubation (grade 1A).
10. Against the routine use of the pulmonary artery catheter for patients with sepsis-induced ARDS (grade 1A).
11. A conservative rather than liberal fluid strategy for patients with established sepsis-induced ARDS who do not have evidence of tissue hypoperfusion (grade 1C).
12. In the absence of specific indications such as bronchospasm, not using beta 2-agonists for treatment of sepsis- induced ARDS. (Grade 1B).

Altres teràpies (maneig SDRA)

- *Ventilar a 6ml/kg*
- *P plateau inicial max 30cmH2O*
- *Aplicar PEEP (si SDRA moderat/greu PEEP elevades)*
- *Maniobres de reclutament en casos d'hipoxèmia refractària*
- *Col.locar en prono si PAFI < 100*
- *Llit a 30-45º per evitar NAVM i disminuir risc BAS*
- *NIV només en casos molt seleccionats*
- *Iniciar weaning quan compleixi criteris (despert, HMD estable, requeriments Pinsp baixes i FiO2 baixes)*
- *Administració restrictiva líquids*
- *No utilitzar CAP rutinàriament*
- *No utilitzar B-adrenèrgics rutinàriament (broncoespasme si)*

Altres teràpies (sedació)

P. Sedation, analgesia, and neuromuscular blockade in sepsis

1. Continuous or intermittent sedation be minimized in mechanically ventilated sepsis patients, targeting specific titration endpoints (grade 1B).
2. Neuromuscular blocking agents (NMBAs) be avoided if possible in the septic patient *without ARDS* due to the risk of prolonged neuromuscular blockade following discontinuation. If NMBAs must be maintained, either intermittent bolus as required or continuous infusion with train-of-four monitoring of the depth of blockade should be used (grade 1C).
3. A short course of NMBA of not greater than 48 hours for patients with *early* sepsis-induced ARDS and a PaO_2/FIO_2 <150 mm Hg (grade 2C).

- *Sedació continua o intermitent mínima si VM (buscar endpoints)*
- *Evitar relaxants musculars si no SDRA (si cal, monitoritzar amb train-of-four)*
- *En SDRA amb PAFI <150mmHg fer tanda curta RM <48h*

Altres terapies

Q. Glucose control

1. We recommend a protocolized approach to blood glucose management in ICU patients with severe sepsis, commencing insulin dosing when two consecutive blood glucose levels are >180 mg/dL. This approach should target an upper blood glucose level ≤180 mg/dL rather than an upper target blood glucose ≤110 mg/dL (grade 1A).
2. We recommend blood glucose values be monitored every 1–2 h until glucose values and insulin infusion rates are stable, then every 4 h thereafter (grade 1C).
3. We recommend that glucose levels obtained with point-of-care testing of capillary blood be interpreted with caution, as such measurements may not accurately estimate arterial blood or plasma glucose values (UG).

Altres teràpies

R. Renal replacement therapy

1. We suggest that continuous renal replacement therapies and intermittent hemodialysis are equivalent in patients with severe sepsis and acute renal failure because they achieve similar short-term survival rates (grade 2B).
2. We suggest the use of continuous therapies to facilitate management of fluid balance in hemodynamically unstable septic patients (grade 2D).

S. Bicarbonate therapy

1. We recommend against the use of sodium bicarbonate therapy for the purpose of improving hemodynamics or reducing vasopressor requirements in patients with hypoperfusion-induced lactic acidemia with pH $\geq$7.15 (grade 2B).

Altres teràpies

T. Deep vein thrombosis prophylaxis

1. We recommend that patients with severe sepsis receive daily pharmacoprophylaxis against venous thromboembolism (VTE) (grade 1B). We recommend that this be accomplished with daily subcutaneous low-molecular weight heparin (LMWH) (grade 1B versus unfractionated heparin [UFH] twice daily and grade 2C versus UFH given thrice daily). If creatinine clearance is <30 mL/min we recommend use of dalteparin (grade 1A) or another form of LMWH that has a low degree of renal metabolism (grade 2C) or UFH (grade 1A).
2. We suggest that patients with severe sepsis be treated with a combination of pharmacologic therapy and intermittent pneumatic compression devices whenever possible (grade 2C).
3. We recommend that septic patients who have a contraindication to heparin use (e.g., thrombocytopenia, severe coagulopathy, active bleeding, recent intracerebral hemorrhage) not receive pharmacoprophylaxis (grade 1B). Rather we suggest they receive mechanical prophylactic treatment, such as graduated compression stockings or intermittent compression devices (grade 2C), unless contraindicated. When the risk decreases, we suggest starting pharmacoprophylaxis (grade 2C).

V. Nutrition

1. We suggest administering oral or enteral (if necessary) feedings, as tolerated, rather than either complete fasting or provision of only intravenous glucose within the first 48 h after a diagnosis of severe sepsis/septic shock (grade 2C).
2. We suggest avoiding mandatory full caloric feeding in the first week, but rather suggest low dose feeding (e.g., up to 500 kcal per day), advancing only as tolerated (grade 2B).
3. We suggest using intravenous glucose and enteral nutrition rather than TPN alone or parenteral nutrition in conjunction with enteral feeding in the first 7 days after a diagnosis of severe sepsis/septic shock (grade 2B).
4. We suggest using nutrition with no specific immunomodulating supplementation in patients with severe sepsis (grade 2C).

Un punt clau: ressucitació inicial

SURVIVING SEPSIS CAMPAIGN CARE BUNDLES

TO BE COMPLETED WITHIN 3 HOURS:
1) Measure lactate level
2) Obtain blood cultures prior to administration of antibiotics
3) Administer broad spectrum antibiotics
4) Administer 30 mL/kg crystalloid for hypotension or lactate ≥ 4 mmol/L

TO BE COMPLETED WITHIN 6 HOURS:
5) Apply vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain a mean arterial pressure (MAP) ≥ 65 mm Hg
6) In the event of persistent arterial hypotension despite volume resuscitation (septic shock) or initial lactate ≥ 4 mmol/L (36 mg/dL):
   - Measure central venous pressure (CVP)*
   - Measure central venous oxygen saturation ($ScvO_2$)*
7) Remeasure lactate if initial lactate was elevated*

*Targets for quantitative resuscitation included in the guidelines are CVP of ≥8 mm Hg, $ScvO_2$ of ≥ 70%, and normalization of lactate.

Recomendaciones en quirófano de manejo de sepsis grave/shock séptico

- ✓ *Hemocultivos*
  - ✓ *Al colocar la vía central*
  - ✓ *Antes de administrar antibióticos*
  - ✓ *Incluso en normotermia*
- ✓ *Al colocar la vía central*
  - ✓ *Muestra la láctico*
  - ✓ *Muestra para ScvO2*
- ✓ *Antibióticos*
  - ✓ ***Lo antes posible***
  - ✓ ***Antes de la cirugía***
  - ✓ *Según protocolo*
- ✓ *Control de foco (cirugía): lo antes posible*
- ✓ *Manejo de hemodinamia*
  - ✓ ***Con GCC***
  - ✓ *Líquidos (cristaloides) según VVS*
  - ✓ *Inotrópicos (dobutamina) según IC*
  - ✓ *Vasopresores (nordrenalina) para TAM > 65 mmHg*
  - ✓ *Hidrocortisona (noradrenalina > 15 ml/h)*
  - ✓ *La TA no es el objetivo, sino un medio para optimizar*

No recomanats

- *Eritropoietina*

- *Inmunoglobulines*

Baixa qualitat de l'evidència científica que existeix

- *Seleni*

Pobre evidència científica

- *Proteína C activada recombinant*

No evidència científica

Retirada del mercat

Per acabar...

Summary and future directions

Although this document is static, the optimum treatment of severe sepsis and septic shock is a dynamic and evolving process. Additional evidence that has appeared since the publication of the 2008 guidelines allows more certainty with which we make severe sepsis recommendations; however, further programmatic clinical research in sepsis is essential to optimize these evidence-based medicine recommendations.

TIPOS DE SHOCK		PVC	GC	RVP	% Sat O_2 venosa
Hipovolémico		↓↓	↓	↑	↓
Cardiogénico		↑	↓↓	↑	↓
Obstructivo		↑↑	↓	↑	↓
Séptico	Hiperdinámico	↓↑	↑	↓	↑
	Hipodinámico o tardío	↓↑	↓		↓↑
Neurogénico		↓	↓	↓	↓
Anafiláctico		↓	↓	↓↓	↓

F. Prevenció infecció

F. Infection prevention

1. We suggest that selective oral decontamination (SOD) and selective digestive decontamination (SDD) should be introduced and investigated as a method to reduce the incidence of VAP; this infection control measure can then be instituted in healthcare settings and regions where this methodology is found to be effective (grade 2B).
2. We suggest oral chlorhexidine gluconate (CHG) be used as a form of oropharyngeal decontamination to reduce the risk of VAP in ICU patients with severe sepsis (grade 2B).

Xoc Hipovolèmic

Xoc Hipovolèmic
ETIOLOGIA

- PÈRDUES EXTERNES:
  - Sagnat
  - Vòmits, diarrees…
  - Drenatges
  - Cremats
- PÈRDUES INTERNES:
  - hemotòrax, hemoperitoni
  - oclusió intestinal, ascitis…
  - fractures, hematomes…
  - cirurgia recent
- RESTRICCIÓ DE LIQUIDS:
  - manca ingesta
  - dejú per exploracions o cirurgia

Xoc Hemorràgic
Respostes Fisiològiques

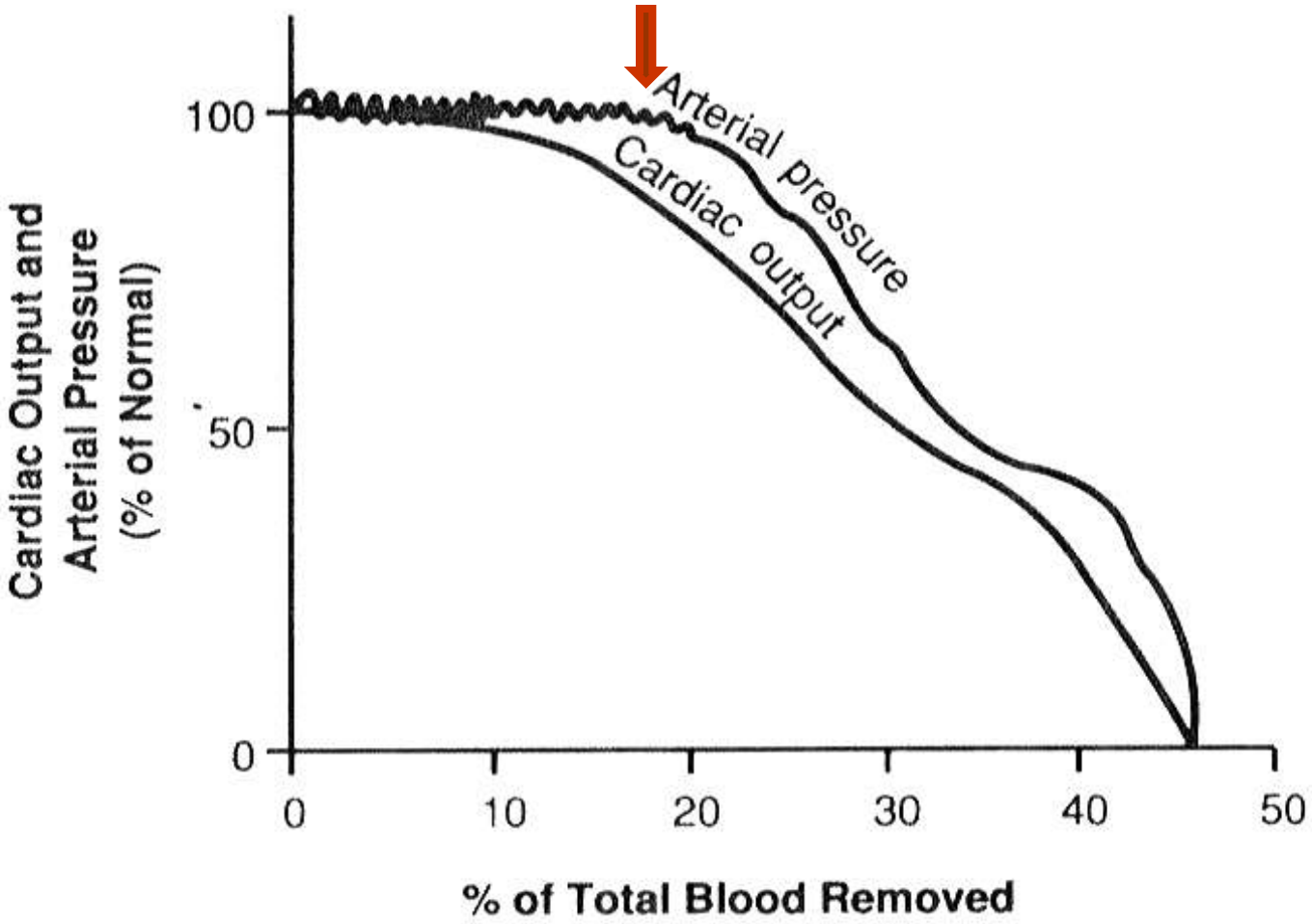
- ACTIVITAT SIMPATICA
  - Taquicardia
  - ↑ Contractilitat
  - ↑ Retorn venòs (modificat per anestèsics)
  - ↑ Resistències vasculars sistèmiques
- RECUPERACIO DE LA VOLEMIA
  - ↑ Osmolaritat plasma: Glucosa, lactat, urea, AA,...
  - ↓ Fluxe transcapilar d'albúmina
  - ↓ P hidrostàtica capilar
- APERTURA DE SHUNTS A-V
- PERFUSIÓ ORGÀNICA SELECTIVA

Xoc Hemorràgic
Classificació

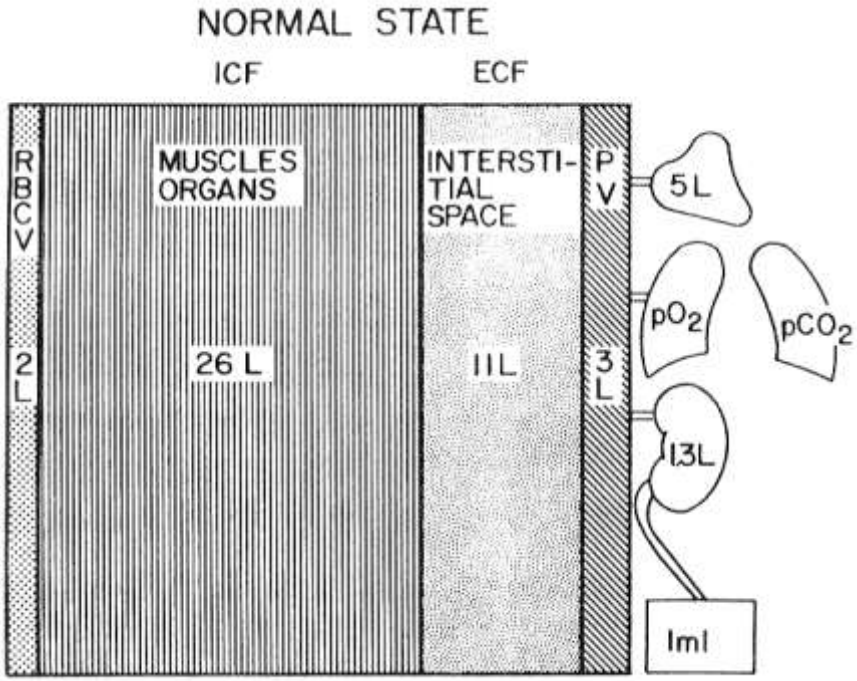
SAGNAT	< 15%	15-30%	30-40%	> 40%
F.C. (bpm)	<100	100-120	120-140	➢140
T.A.	Normal	Normal	↓	↓
P.P.	Normal	↓	↓	↓
F.R. (rpm)	14-20	20-30	30-40	> 35
Diuresis	> 30ml/h	20-30ml/h	5-15ml/h	Anuria
SNC	Normal	Ansietat	Confusió	Letàrgia

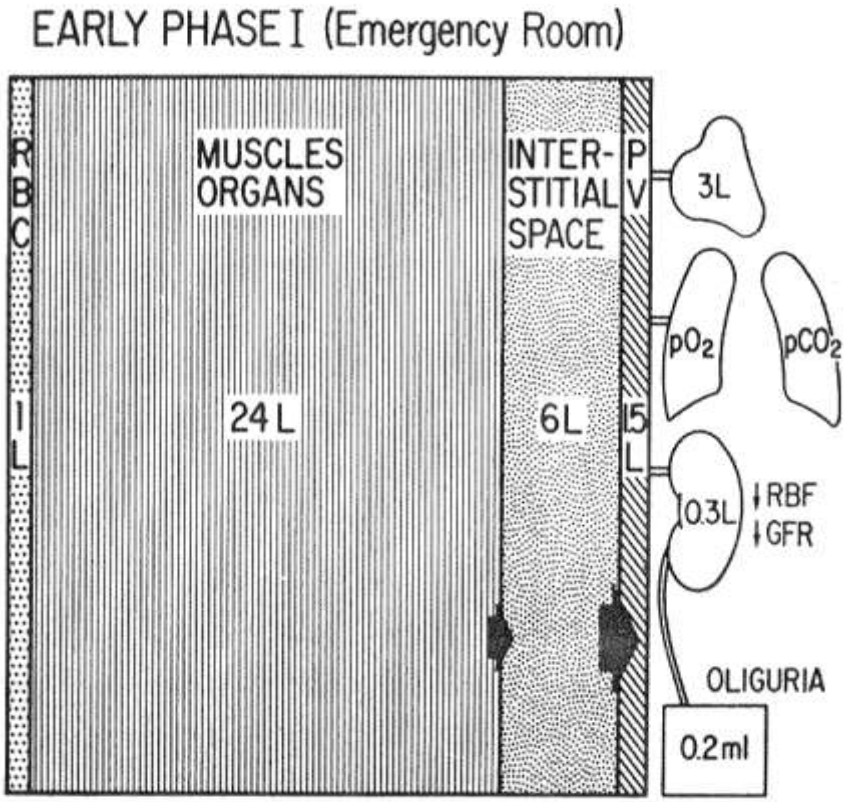
American College of Surgeons of Advanced Trauma Life Support: classificació severitat hemorràgia (ATLS)

Xoc Hemorràgic
Respostes Fisiològiques



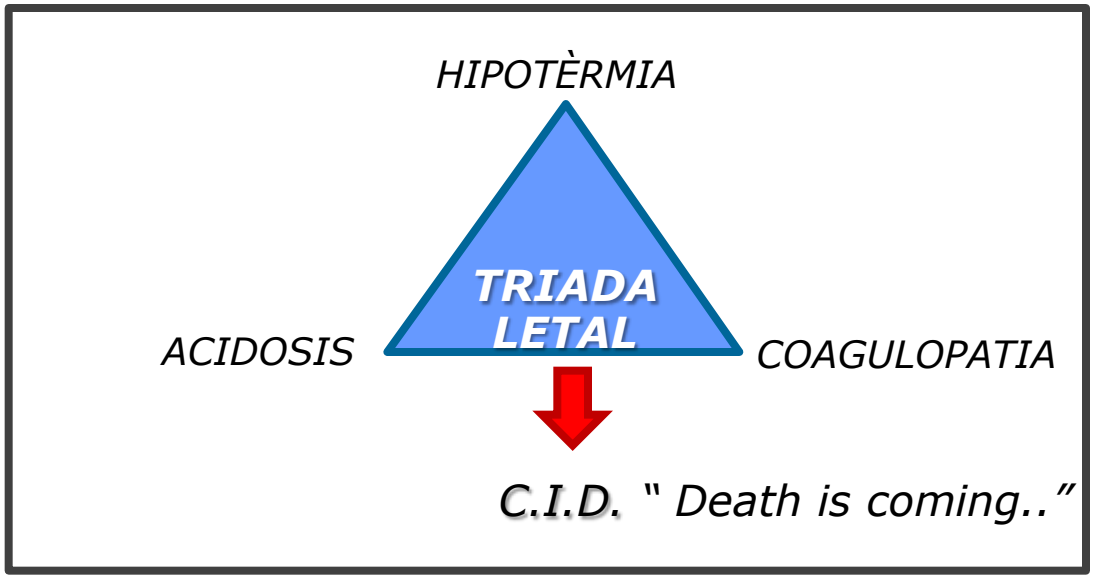
Xoc Hemorràgic
Respostes Fisiològiques





Xoc Hemorràgic
Tractament

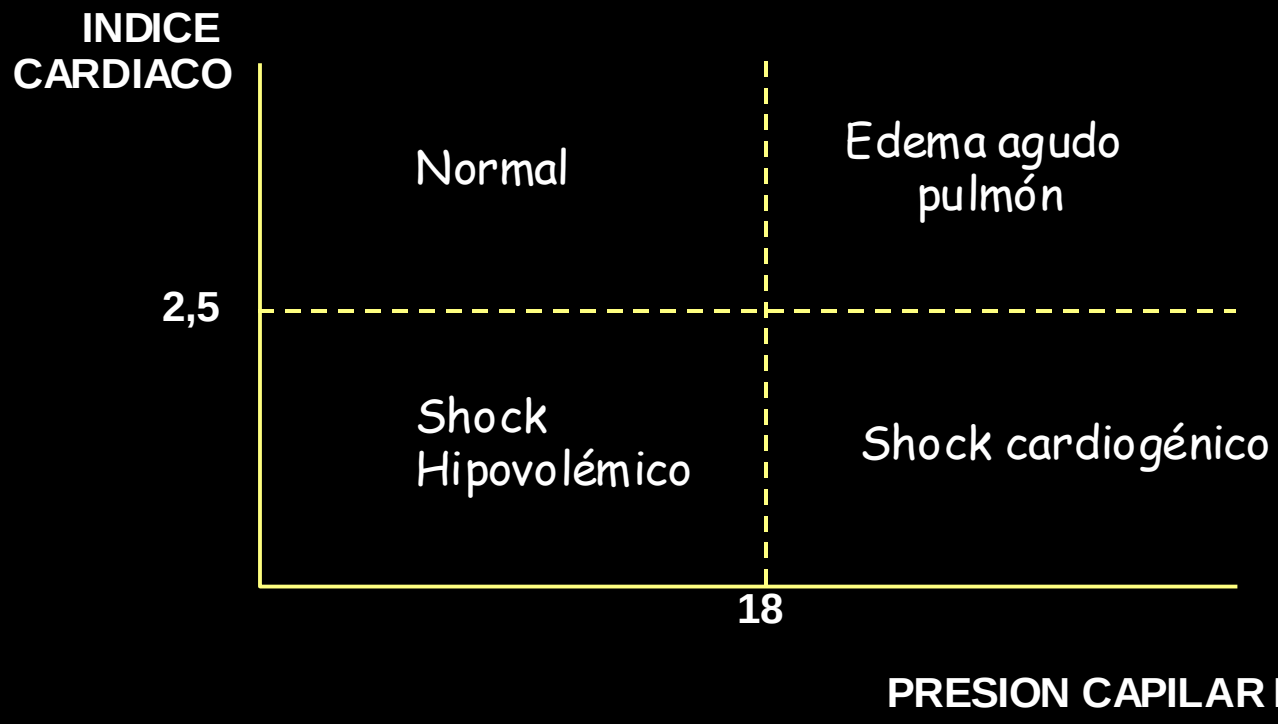
- Control del sagnat
- Si PCR seguir ABC (Airway, Breathing, Circulation)
- Posició correcta del pacient (pla o cames aixecades, no Trendelemburg)
- Hipotensió permisiva
- Reposició volèmia (coloides, cristaloides, sol. balan.)
- Transfusió
- Hiperoxigenació
- Tractar 3H



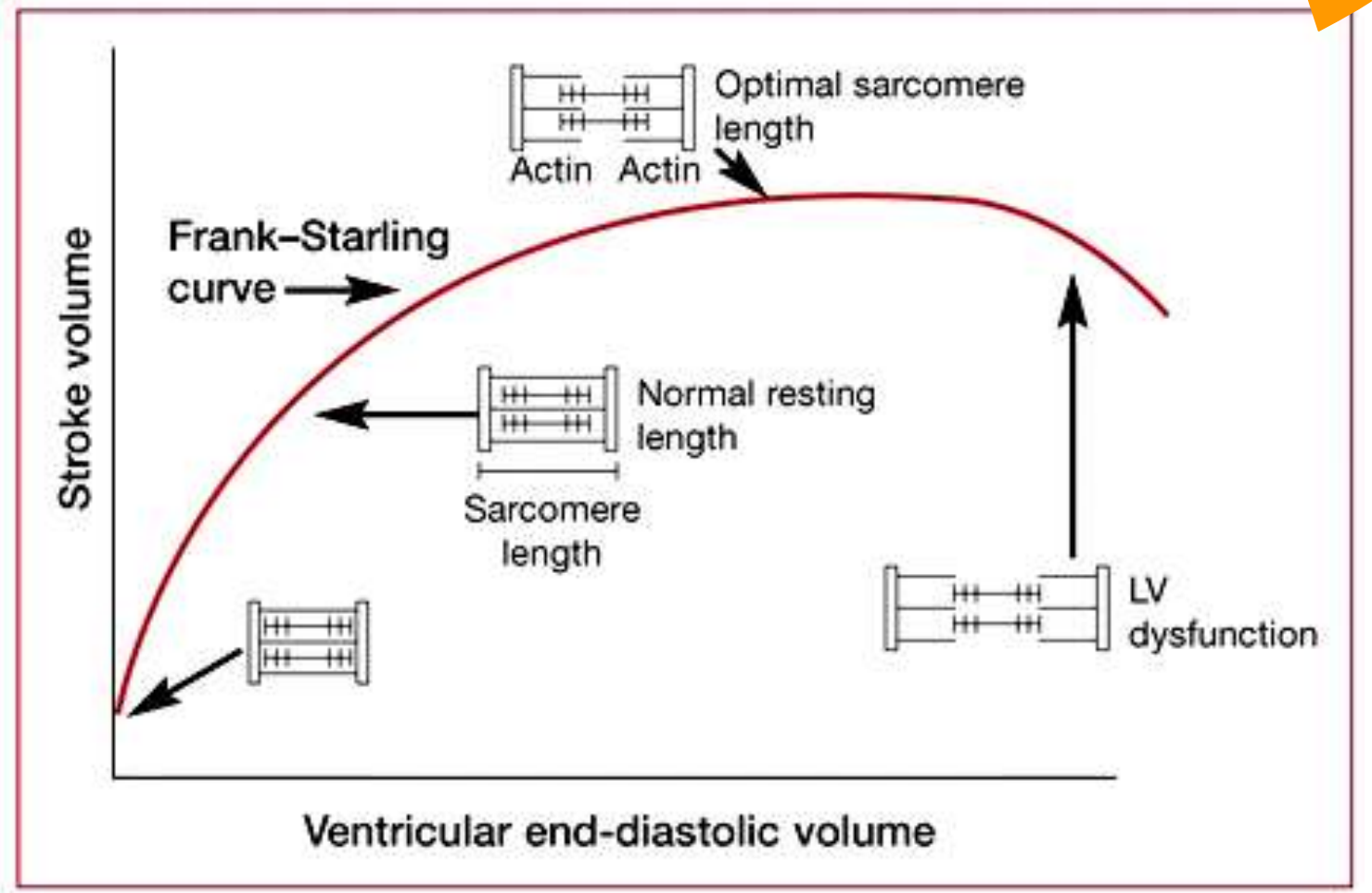
Xoc Cardiogènic

Xoc Cardiogènic
Etiologia

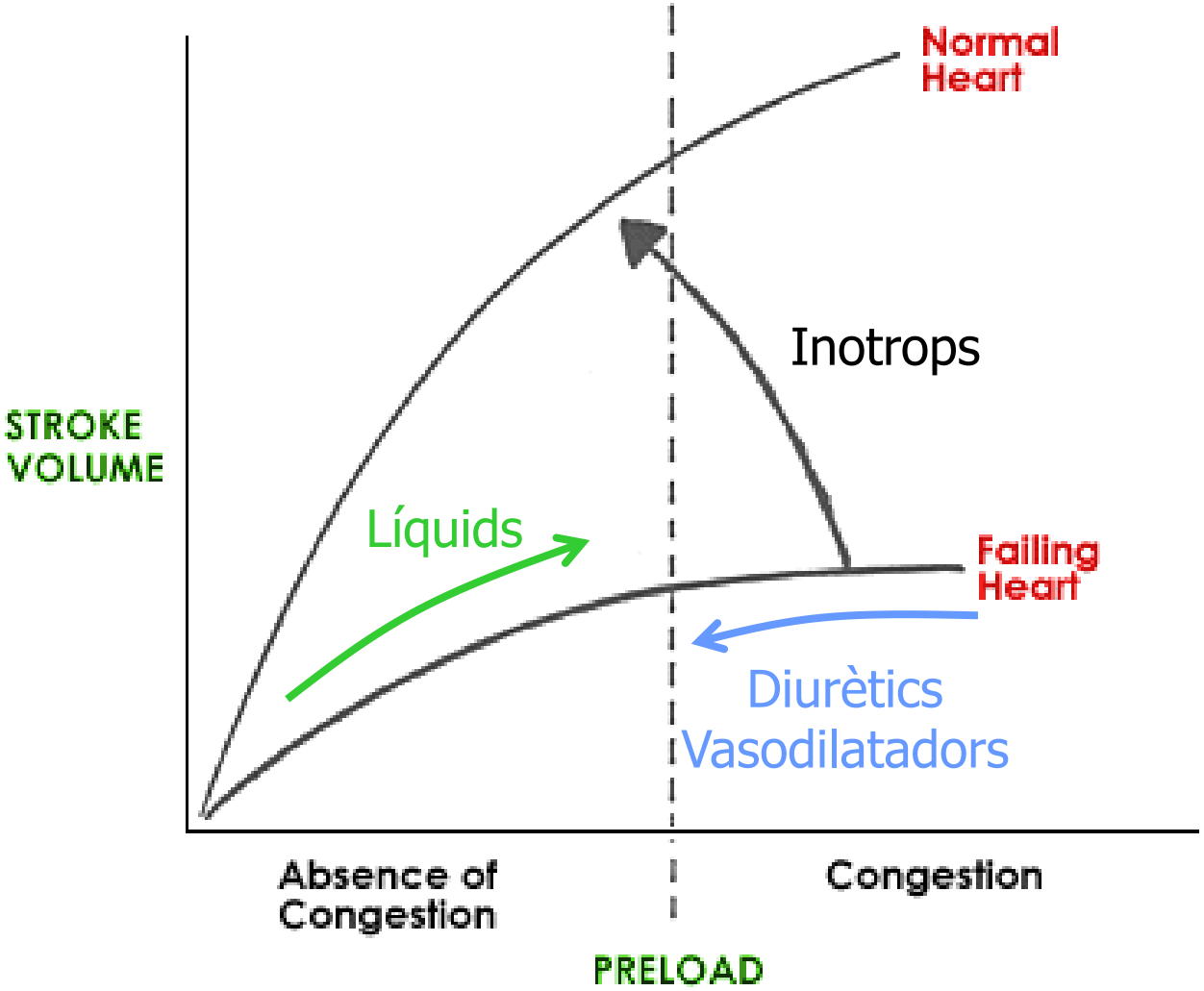
- Miopàtic
  - Infart De Miocardi
  - Miocardiopatia Dilatada
  - Sortida De CEC
- Mecànic
  - Valvulopatia
  - Defecte Pared Ventricular
- Arrítmia
- Obstructiu
  - Taponament Pericàrdic
  - Pericarditis Constrictiva
  - Tromboembolisme Pulmonar
  - Coartació Aòrtica
  - Neumotòrax a Tensió


INDICE CARDIACO
Normal
Edema agudo pulmón
2,5
Shock Hipovolémico
Shock cardiogénico
18
PRESION CAPILAR PULMONAR

Xoc Cardiogènic
Corbes Starling



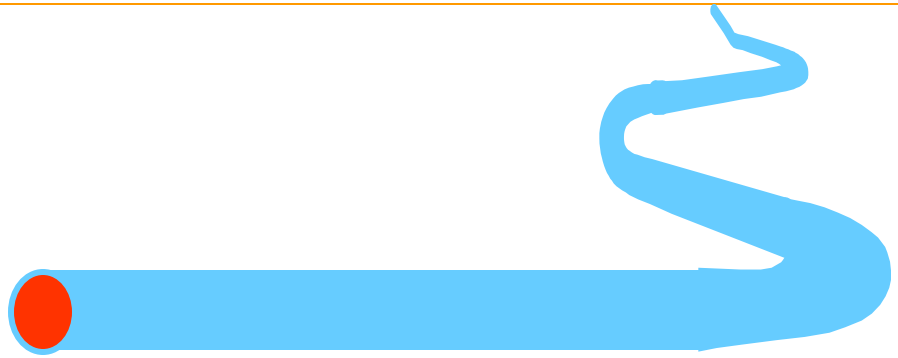
Xoc Cardiogènic
Corbes Starling

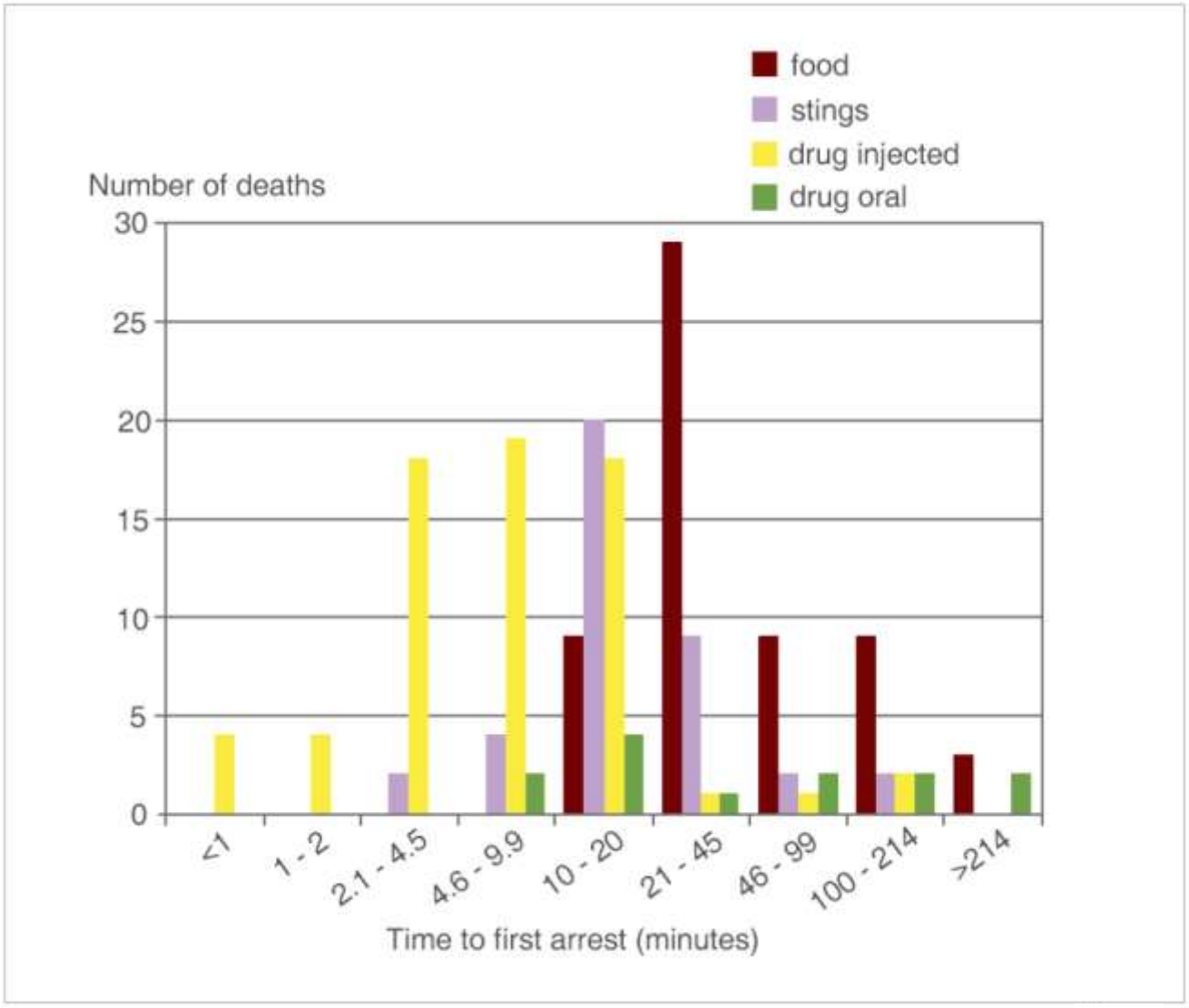


Xoc Distributiu

- Sèptic
- Anafilàctic
- Neurogènic
- Endocrinològic

Xoc Anafilàctic





Time to cardiac arrest following exposure to triggering agent

ETIOLOGIA

- Fàrmacs
  - Antibiòtics
  - Anestèsics:
    - » curares
    - » làtex
    - » agents hipnòtics
    - » substituts del plasma
    - » opioides
    - » benzodiacepines .
  - Contrast radiològic
  - AINES
- Productes naturals
  - Polen, fruits secs, picades insectes...

CLINICA

- Cardiovascular
  - Taquicardia
  - Hipotensió
  - HTA Pulmonar
- Respiratòria
  - Broncoespasme
  - Edema de Glotis
  - Edema Pulmonar no cardiogènic
- Cutània
  - Urticaria
  - Flushing
  - Edema periorbitari

XOC ANAFILÀCTIC

TRACTAMENT

- Retirar al·lèrgen
- Si PCR seguir l'ABC
- Líquids +++
- Adrenalina (100mcg) *efecte $\alpha \rightarrow$ VC*
  efecte $\beta \rightarrow$ BD
- Altres catecolamines: Noradrenalina, fenilefrina
- Dopamina, Efedrina
- Broncodilatadors (...Halogenats)
- Corticoides (efecte a les 4-6h)
- RCP prolongada