CytoSorbents™

Working to save lives together.

Turning the Tide of Sepsis and Septic Shock:

Real World Insights with CytoSorb

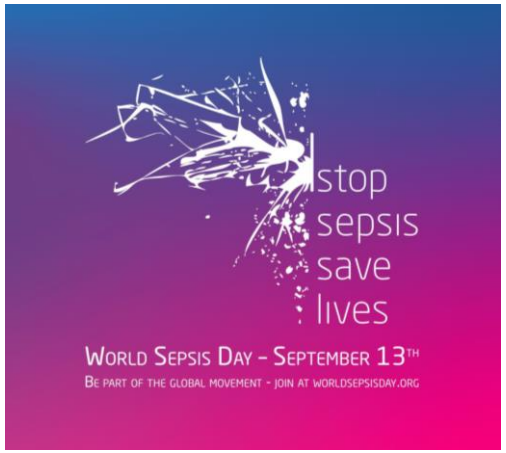
September 10, 2025

Link to webinar replay: https://cyto.news/webinar-sepsis/sep10

Purpose and Agenda

In recognition of World Sepsis Day (September 13th) and Sepsis Awareness Month (September) and our commitment to help to solve the sepsis crisis:

- Summarize the current understanding of how CytoSorb® broadly treats sepsis and septic shock
- Help to optimize CytoSorb treatment with the "Right Patient at the Right Time with the Right Dosage"
- Emphasize the therapeutic goals of CytoSorb to prevent or treat organ failure
  - Break the cycle of deadly, uncontrolled inflammation
  - Stabilize the patient (reverse shock, improve oxygenation, support organ function, etc)
  - Promote reversal of capillary leak
  - Get the excessive fluid off!
- Highlight recent studies that support this new simplified approach
- Learn from expert users of the therapy on how they are driving successful treatment



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Participants

Dr. Phillip Chan, MD, PhD - Moderator
Chief Executive Officer – CytoSorbents

Dr. med. Tobias Hübner, MD DESA EDIC MHBA
Chief Physician of the Intensive Care Unit and Vice Head of the Department of Anesthesia and Intensive Care Hospital of Münsterlingen – Münsterlingen, Switzerland

Prof. Dr. Zsolt Molnár, MD, PhD
Head of Department of Anesthesiology and Intensive Therapy
Semmelweis University – Budapest, Hungary

Associate Prof. Dr. med. Kevin Pilarczyk, MD MHBA
Chief Physician of Intensive Care and Emergency Medicine and Associate Professor
Hochsauerland Hospital – Arnsberg, Germany

Prof. Dr. med. Daniel Wendt, MD, PhD, MHBA, FETCS
Vice President Medical Affairs – CytoSorbents
University of Essen - Essen, Germany

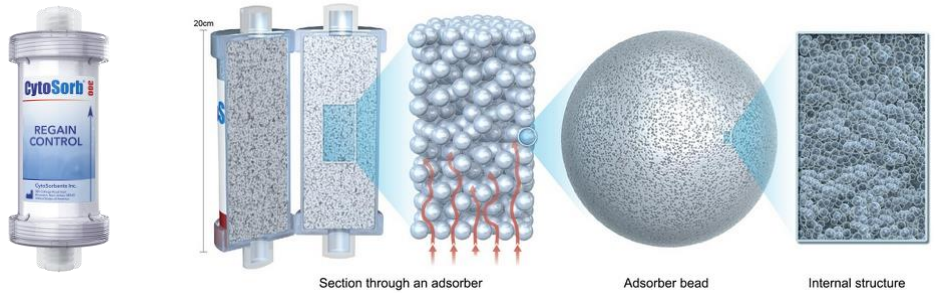
CytoSorbents™

The CytoSorb Adsorber

CytoSorb is a broad-spectrum blood purification device approved as the first specifically approved extracorporeal cytokine adsorber in the European Union in 2011 and used in more than 70 countries and nearly 300,000 human treatments worldwide. It is also approved to remove bilirubin (e.g. liver failure), myoglobin (e.g. rhabdomyolysis), and the antithrombotic agents ticagrelor and rivaroxaban

- Uses hemocompatible, highly porous polymer beads that act like tiny sponges to remove harmful substances up to ~60 kDa from blood by pore capture, adsorption, and concentration. Solid-state porous polymer technology – no antibodies, ligands, cells, or biologics.

- CytoSorb is plug-and-play compatible with the existing installed base of blood pumps found in the hospital

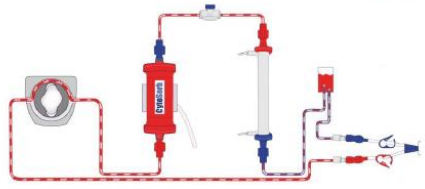


Disclaimer:

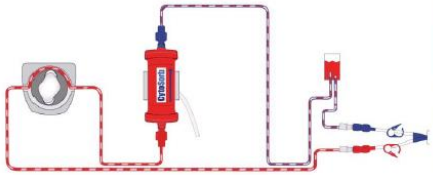
- CytoSorb has also received U.S FDA Emergency Use Authorization to treat patients 18 years of age or older, with confirmed COVID-19 admitted to the ICU with confirmed or imminent respiratory failure to reduce pro-inflammatory cytokine levels. The device is authorized only for the duration of the declaration that circumstances exist justifying the authorization of the emergency use of the CytoSorb device under EUA, unless the authorization is terminated or revoked earlier.

- The CytoSorb device has neither been cleared or approved in the U.S.

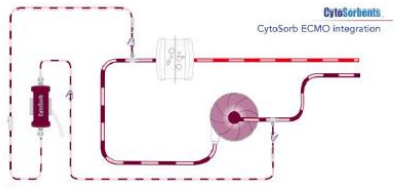
Dialysis or CRRT
(Continuous Renal Replacement Therapy)



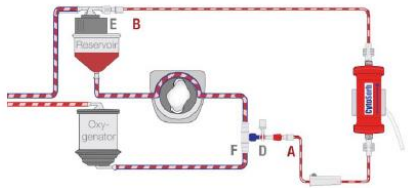
Hemoperfusion
(Standalone Treatment)



ECMO
(Extracorporeal Membrane Oxygenation)

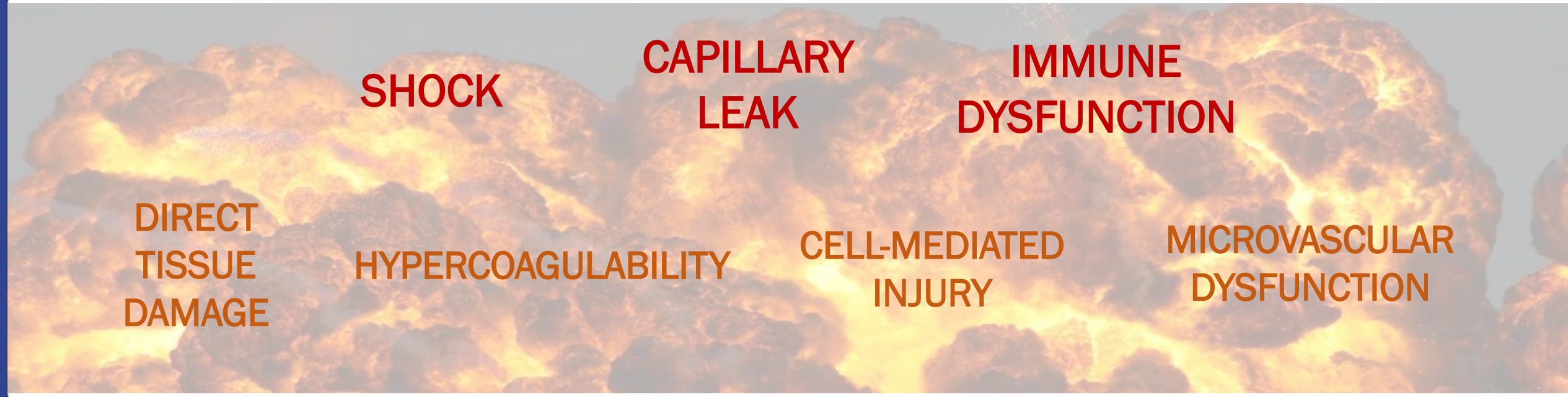


CPB
(Cardiopulmonary Bypass)



CytoSorb Controls Massive Inflammation - the Heart of Critical Illness

- Acute inflammation is the body's mechanism to fight injury and infection
- However, severe inflammation, driven by cytokine storm, can cause a chain reaction of problems that can end in organ failure and death



- Severe inflammation is the common thread amongst most critical illnesses and impacts up to 60% of patients in the ICU. Is directly correlated to increased severity of illness, organ failure, and mortality

CytoSorb controls deadly inflammation and has demonstrated the reversal or prevention of many of these complications

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Sepsis and Septic Shock are Deadly

- Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection.
- This “dysregulated host response” often manifests as a worsening inflammatory response that can spiral out of control, fueled by the excessive production of cytokines (cytokine storm), bacterial toxins, activated complement, and many other inflammatory mediators
- Unchecked, massive inflammation can lead to Septic Shock – an often fatal complication marked by circulatory collapse and a lethal drop in blood pressure and a host of other problems like capillary leak and fluid overload that can lead to multiple organ failure and death
- Sepsis and septic shock afflict an estimated 49 million people worldwide each year, killing 11 million, and accounts for 1 in 5 deaths globally
- Supportive care treatment has improved, but mortality is still unacceptably high, despite antibiotics, fluids, vasopressors, and mechanical “life support”

CytoSorbents™

The Duality of Sepsis

For more than a decade, CytoSorbents has collaborated with clinicians and scientists around the world to advance the treatment of sepsis and septic shock by complementing traditional antibiotics with the broad-spectrum capability of CytoSorb

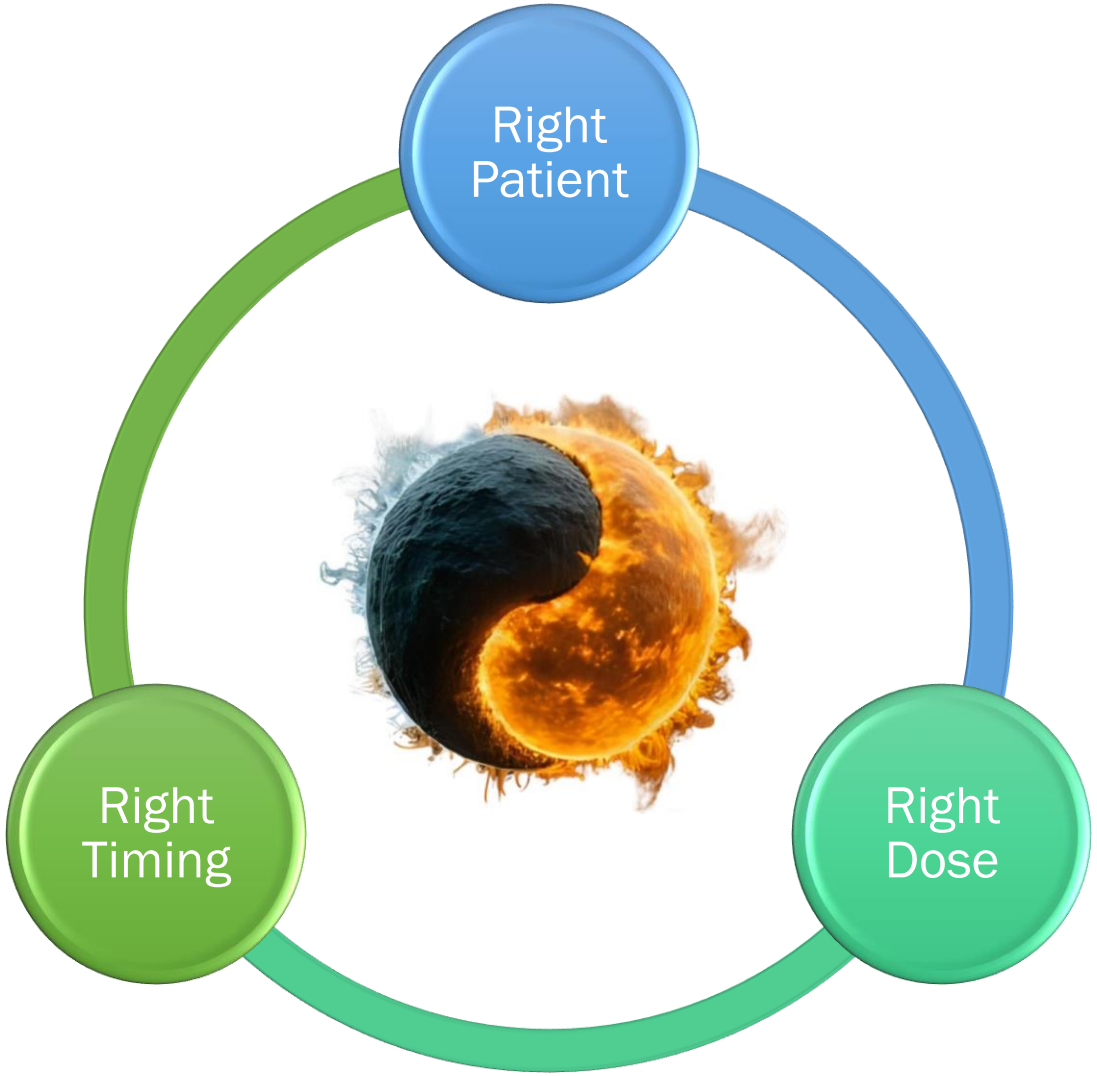
Antibiotics treat the infection

CytoSorb treats the deadly inflammatory response by removing the “fuel to the fire” that causes a system crash

Our understanding about CytoSorb in the treatment of septic shock has continued to evolve. We view CytoSorb as more than an “adjunctive” therapy to buy time, but rather as a fundamental part, just like antibiotics, of an end-to-end strategy to manage the septic patient

CytoSorbents™

The Key to Success: Right Patient, Right Timing, Right Dosing



Just like antibiotics, CytoSorb works most effectively when:

- Treat Early
- Treat Intensively
- Compete the Full Course of Treatment

CytoSorbents™

CytoSorb Attacks Sepsis in a Comprehensive & Multi-faceted Way

- Reduce inflammation by reducing a broad range of cytokines (cytokine storm) & other inflammatory mediators
- Bacterial toxins (hemolysins, super antigens, etc)
- Reversal of shock (macro) & improvement in microcirculation and lactate clearance 
- Improve respiratory function and gas exchange
- Protect the endothelium, promote repair of capillary leak, and **enable excess fluid removal** 
- Improve or protect organ function (e.g. shock, ARDS, acute kidney injury, liver dysfunction)
- Re-establish proper leukocyte trafficking to prevent cell-mediated organ injury

CytoSorb is very unique in its broad range of activity

CytoSorbents™

CytoSorb Core Treatment Goals

A wealth of published, peer-reviewed studies support the broad mechanisms of action of CytoSorb that enable a comprehensive and multi-faceted attack on septic shock – from beginning to end

CytoSorb helps to achieve the
Core Treatment Goals in Septic Shock to
Prevent or Treat Organ Failure and
Help Patients Recover

- Break the vicious cycle of massive, uncontrolled inflammation
- Stabilize the patient (reverse shock, improve oxygenation, restore oxygenated blood flow, etc)
- Promote the repair of capillary leak
- Actively remove excessive fluid and reduce fluid overload in organs

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CytoSorb Treatment Goal #1:

Break the Vicious Cycle of Massive Uncontrolled Inflammation

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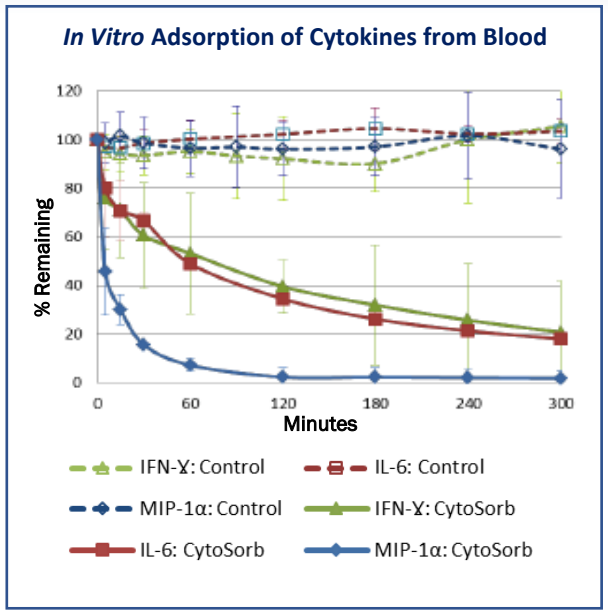
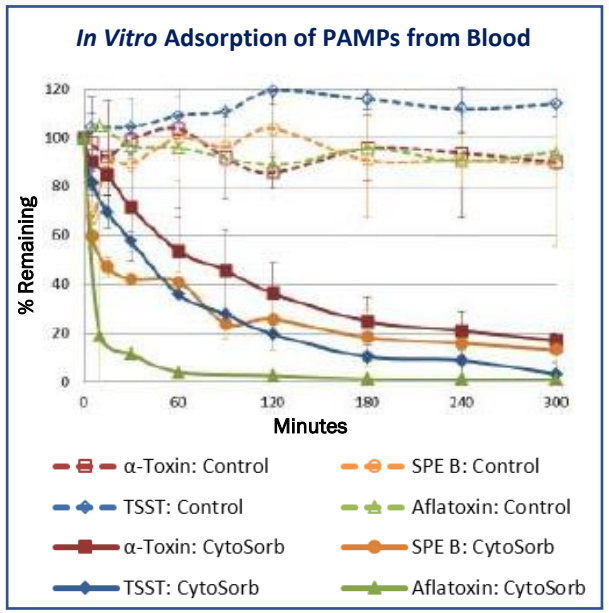
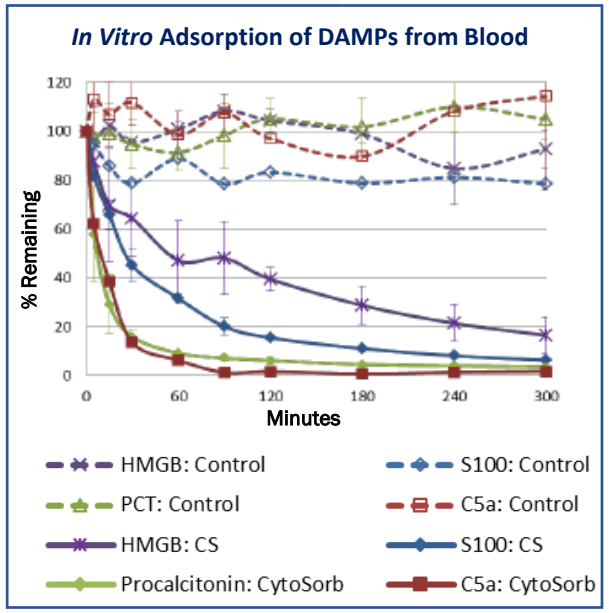
CytoSorb Broadly Reduces Inflammatory Mediators



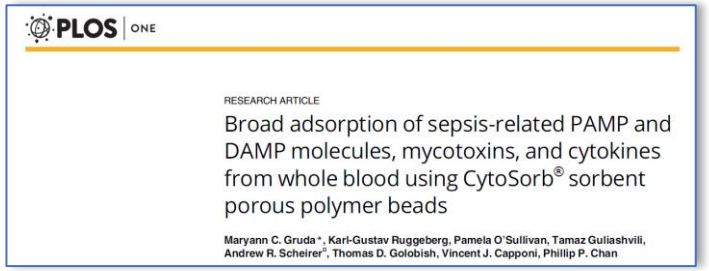
Cytokines



Pathogen associated molecular patterns (PAMPs)



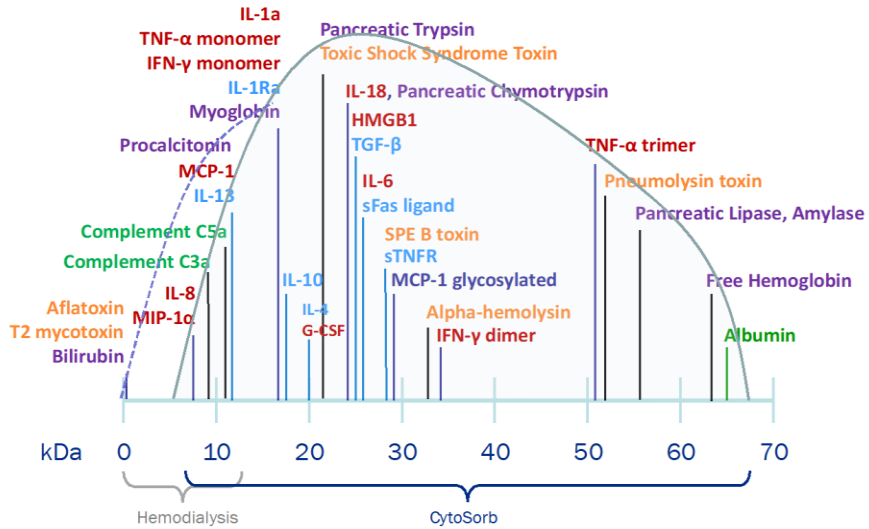
Damage associated molecular patterns (DAMPs)


PLOS ONE

RESEARCH ARTICLE

Broad adsorption of sepsis-related PAMP and DAMP molecules, mycotoxins, and cytokines from whole blood using CytoSorb® sorbent porous polymer beads

Maryann C. Gruda*, Karl-Gustav Ruggeberg, Pamela O'Sullivan, Tamaz Guliashvili, Andrew R. Scheirer, Thomas D. Golobish, Vincent J. Capponi, Phillip P. Chan



Gruda, M.C., et al. PLOS ONE 13.1 (2018): e0191676.

CytoSorbents™

CytoSorb Blunts the Inflammatory Response

Jansen et al. Critical Care (2023) 27:117
https://doi.org/10.1186/s13054-023-04391-z

Critical Care

RESEARCH Open Access

CytoSorb hemoperfusion markedly attenuates circulating cytokine concentrations during systemic inflammation in humans in vivo

Aron Jansen[1,2*], Nicole J. B. Waalders[1,2†], Dirk P. T. van Lier[1,2†], Matthijs Kox[1,2] and Peter Pickkers[1,2*]

In a well-controlled endotoxin-challenge model in 24 healthy human volunteers, CytoSorb treatment demonstrated a marked attenuation of cytokines compared to controls. Importantly, following repeat endotoxin challenge 1 week later (no blood purification), there was no significant difference in cytokine response, showing no immune suppression

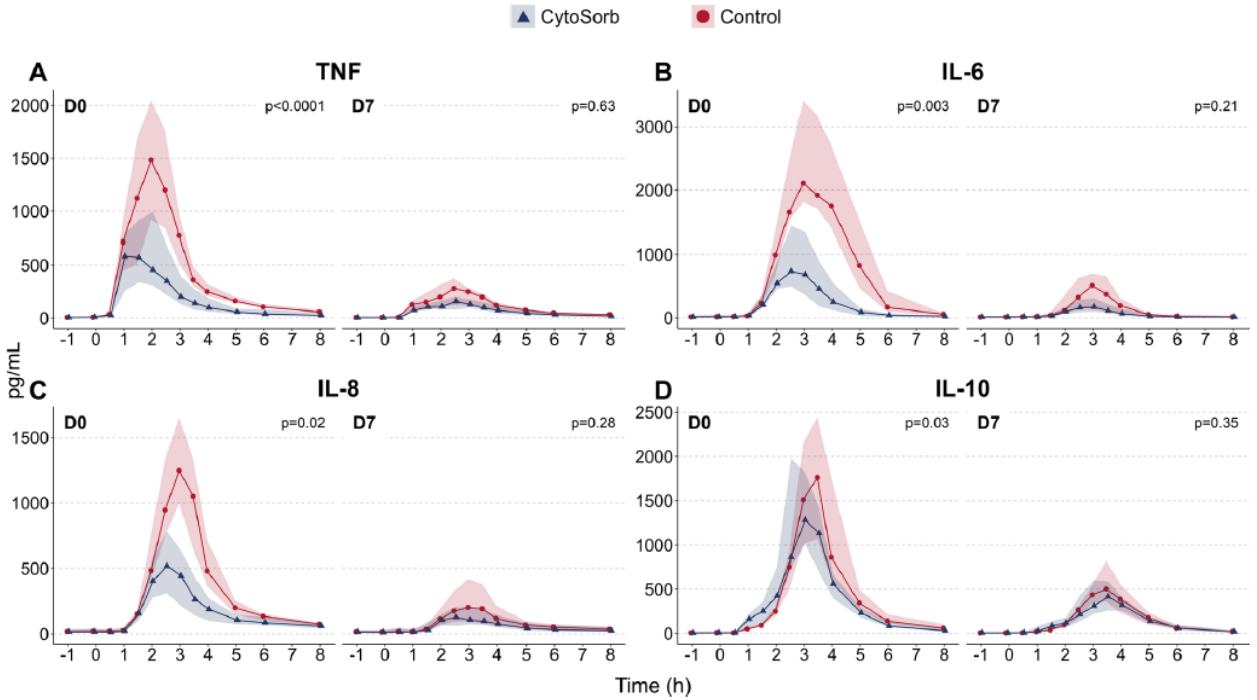


Fig. 2 Plasma concentrations of **A** TNF, **B** IL-6, **C** IL-8 and **D** IL-10 during the first (D0) and second (D7) LPS challenge day. Data are displayed as median (line) and interquartile range (shaded area). *P* values were computed using two-way repeated measures analysis of variance (time × group interaction term). D0 = day 0, D7 = day 7, TNF = tumor necrosis factor, IL = interleukin

CytoSorb Treatment Goal #2:

Stabilize the Patient

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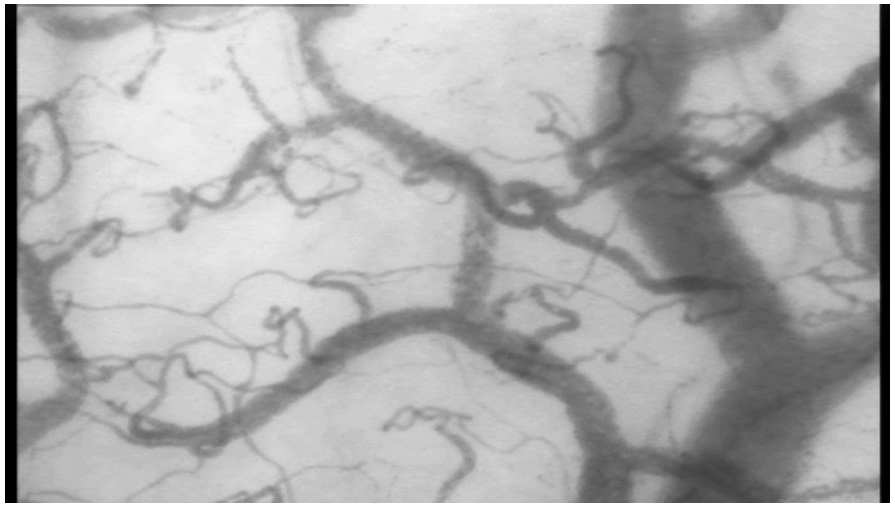
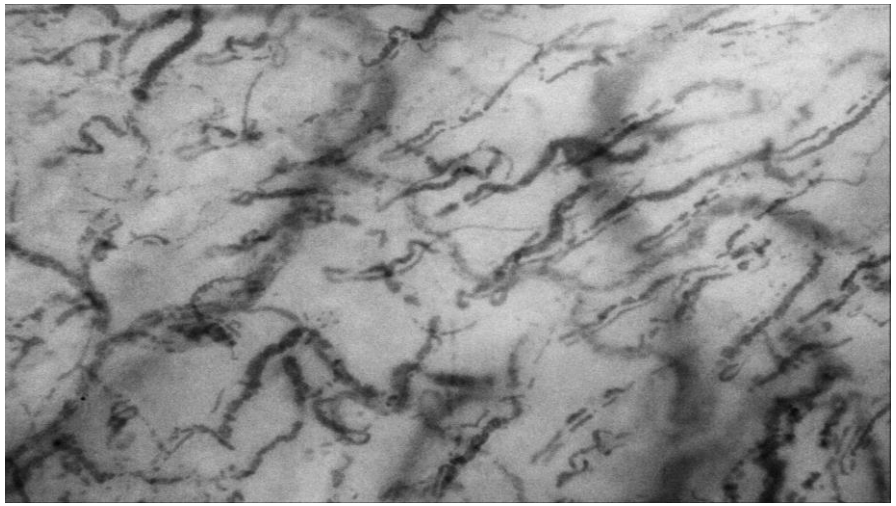
CytoSorb Improves Microcirculation, Promotes Lactate Clearance

Normal



Sepsis



www.nature.com/scientificreports

scientific reports

OPEN

Hemoadsorption improves kidney microcirculatory oxygenation and oxygen consumption, ameliorates tubular injury, and improves kidney function in a rat model of sepsis-induced AKI

Bülent Ergin[1], Deniz Erol Kutucu[2], Aysegul Kapucu[2], Wijnie van Dam[1], Lorenza Moretto[1,3], Paul Heyman[4] & Can Ince[1]

Blood Purification

Sublingual Microcirculatory Evaluation of Extracorporeal Hemoadsorption with CytoSorb® in Abdominal Sepsis: A Case Report

Servet Duran[a] Dietske Miedema[b] Bülent Ergin[b] Can Ince[b]

[a]Intensive Care Units, Wilhelmina Gasthuis Assen, Groningen, The Netherlands; [b]Department of Intensive Care Medicine, Erasmus MC, University Medical Center Rotterdam, Rotterdam, The Netherlands

Table 1. Patient monitoring values

Parameters	T_0 baseline	T_1 (24 h) 12 h on CySo	T_2 (48 h) 36 h on CySo	T_3 (72 h) 12 h after CySo	T_4 (144 h) 84 h after CySo
Days in ICU	1	2	3	4	7
Hb, mmol/L	5.6	5.4	5.1	4.8	6.6
Lactate	9.2	8.6	2.2	1.6	1.0
pH	7.19	7.28	7.38	7.39	7.37

biomedicines

MDPI

Article

The Sublingual Microcirculation in Critically Ill Children with Septic Shock Undergoing Hemoadsorption: A Pilot Study

Gabriella Bottari [1,*], Valerio Confalone [1], Jacques Creteur [2], Corrado Cecchetti [1] and Fabio Silvio Taccone [2]

Volume 49, Issue 1-2
March 2020
Blood Purification

RESEARCH ARTICLES | AUGUST 21 2019

Changes in Cytokines, Haemodynamics and Microcirculation in Patients with Sepsis/Septic Shock Undergoing Continuous Renal Replacement Therapy and Blood Purification with CytoSorb

Subject Area: Nephrology

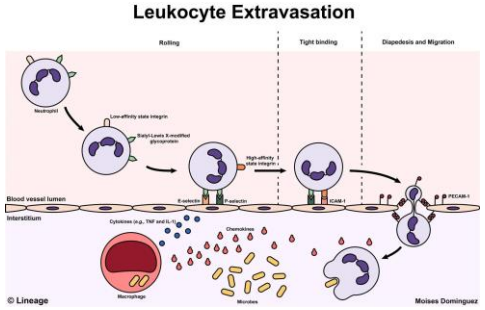
Samuele Zuccari; Elisa Damiani; Roberta Domizi; Claudia Scorcella; Mario D'Arezzo; Andrea Carsetti; Simona Pantanetti; Sara Vannicola; Erika Casarotta; Andrea Ranghino; Abele Donati; Erica Adrario

Blood Purif (2020) 49 (1-2): 107–113.
https://doi.org/10.1159/000502540 Article history

 Courtesy of Prof. Can Ince, Department of Translational Intensive Care Medicine, Erasmus Medical Center, Rotterdam, Netherlands

CytoSorb Can Help Prevent Immune Cell Mediated Organ Injury

- Local chemokine expression and endothelial cell adhesion molecules (ECAMs) are critical to activated leukocyte recruitment to an area of infection



- Cytokine storm creates “immune confusion” by masking this mechanism, resulting in widespread ECAM expression in blood vessels throughout the body resulting in abnormal leukocyte margination

- These activated leukocytes often extravasate into healthy organs, releasing myeloperoxidase (MPO), enzymes, and reactive oxygen species that cause cell-mediated damage to organs

- By treating cytokine storm, CytoSorb can prevent cell-mediated organ injury by redirecting activated WBCs to the infection and away from healthy vital organs

Peng et al. Critical Care 2014, 18:R141
http://ccforum.com/content/18/4/R141

CRITICAL CARE

RESEARCH Open Access

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

Zhi-Yong Peng[1,2], Jeffery V Bishop[2], Xiao-Yan Wen[1,2], Michele M Elder[1,2], Feihu Zhou[1,2], Anan Chuasuwan[1,2], Melinda J Carter[2], Jason E Devlin[3], A Murat Kaynar[1,2], Kai Singbartl[1,2], Francis Pike[1,2], Robert S Parker[1,2,5,6], Gilles Clermont[1,2,5,6], William J Federspiel[1,2,4,6] and John A Kellum[1,2,4,6,7*]

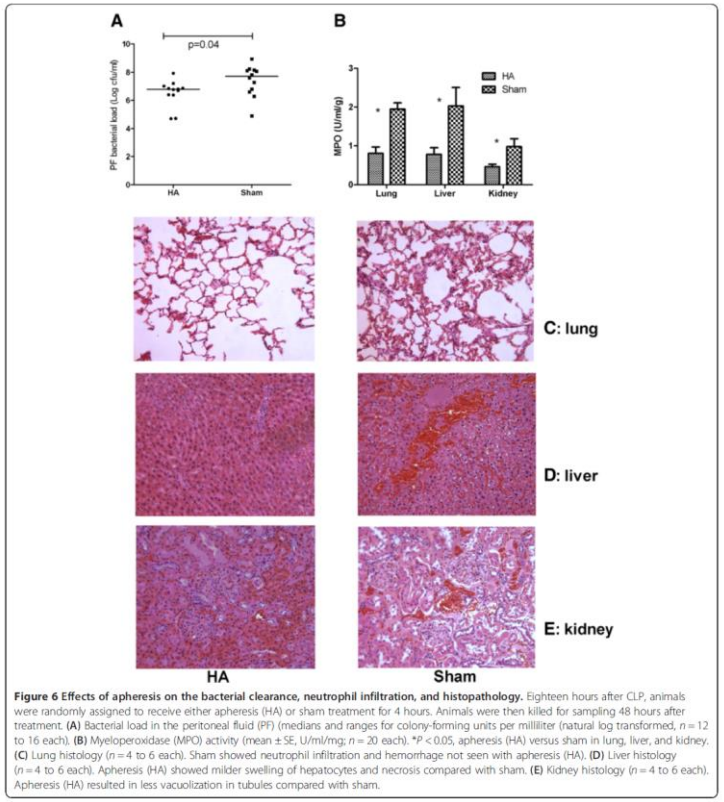


Figure 6 Effects of apheresis on the bacterial clearance, neutrophil infiltration, and histopathology. Eighteen hours after CLP, animals were randomly assigned to receive either apheresis (HA) or sham treatment for 4 hours. Animals were then killed for sampling 48 hours after treatment. **(A)** Bacterial load in the peritoneal fluid (PF) (medians and ranges for colony-forming units per milliliter (natural log transformed, $n = 12$ to 16 each). **(B)** Myeloperoxidase (MPO) activity (mean ± SE, U/ml/mg; $n = 20$ each). *$P < 0.05$, apheresis (HA) versus sham in lung, liver, and kidney. **(C)** Lung histology ($n = 4$ to 6 each). Sham showed neutrophil infiltration and hemorrhage not seen with apheresis (HA). **(D)** Liver histology ($n = 4$ to 6 each). Apheresis (HA) showed milder swelling of hepatocytes and necrosis compared with sham. **(E)** Kidney histology ($n = 4$ to 6 each). Apheresis (HA) resulted in less vacuolization in tubules compared with sham.

- In a cecal ligation puncture peritonitis sepsis rat model (n=76) without antibiotics, CytoSorb treatment (compared to sham control) significantly led to:
  - Markedly higher chemokine concentrations in infected vs healthy tissues
  - Re-direction and greater recruitment of activated neutrophils to the peritoneum
  - Better source control with a decrease in bacterial counts
  - Decreased cell-mediated injury (hemorrhage, tissue damage) and decreased MPO release in the lung, liver, and kidney
  - Decreased infiltration of tagged neutrophils in the lung

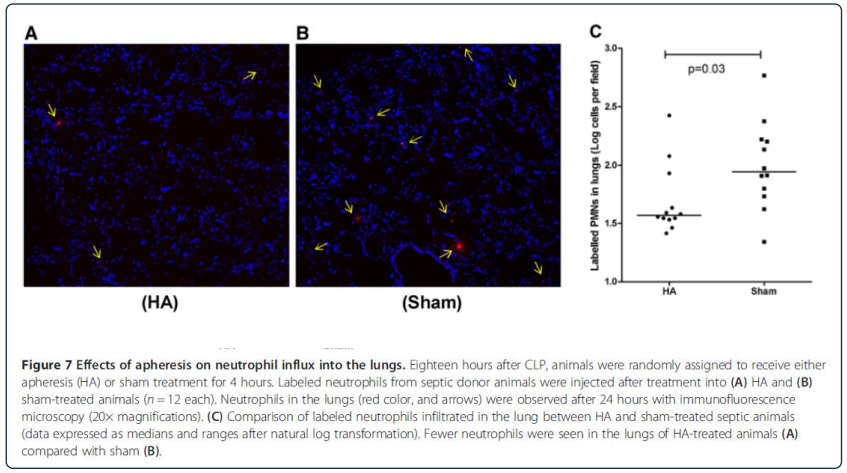


Figure 7 Effects of apheresis on neutrophil influx into the lungs. Eighteen hours after CLP, animals were randomly assigned to receive either apheresis (HA) or sham treatment for 4 hours. Labeled neutrophils from septic donor animals were injected after treatment into **(A)** HA and **(B)** sham-treated animals ($n = 12$ each). Neutrophils in the lungs (red color, and arrows) were observed after 24 hours with immunofluorescence microscopy (20× magnifications). **(C)** Comparison of labeled neutrophils infiltrated in the lung between HA and sham-treated septic animals (data expressed as medians and ranges after natural log transformation). Fewer neutrophils were seen in the lungs of HA-treated animals **(A)** compared with sham **(B)**.

 Peng, ZY, et al. Critical Care (2014); 18:R1141

CytoSorb Enables Shock Reversal and Improved Fluid Balance

Reversal of shock is a key feature of CytoSorb usage, resulting in the ability to wean vasopressors, restore both macro and microcirculation, reduce lactate, promote capillary leak reversal, and enable fluid removal

Shock Reversal

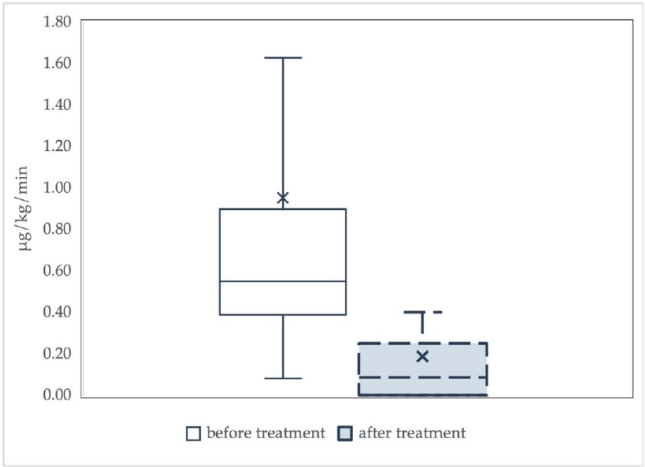


Figure 2. Norepinephrine requirements before and after treatment with Cytosorb. Data are summarized as boxplots. The "x" in the box represents the mean value. There is a significant decline in median norepinephrine requirements before and after hemoadsorption with Cytosorb (from 0.55 (0.39–0.9) µg/kg/min to 0.09 (0.0–0.25) µg/kg/min, $p < 0.001$).

Meta-analysis from 33 studies and 353 patients with shock requiring vasopressors demonstrating a significant reduction in vasopressor need with CytoSorb usage

Improved Fluid Balance

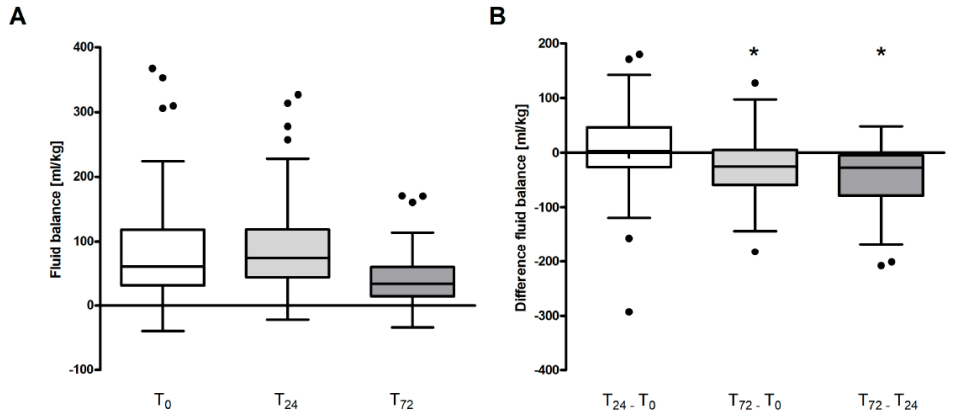


Figure 1. Absolute fluid balance (**A**) and calculated differences in fluid balance (**B**) for the entire study population. Depicted are Tukey boxplots with equal whisker lengths of 1.5 IQR for both whiskers. Dots represent outliers. A p-value of 0.05, as represented by an asterisk (*), was considered significant.

Retrospective study in 124 septic shock patients evaluating fluid balance after 24 and 72 hours of CytoSorb treatment

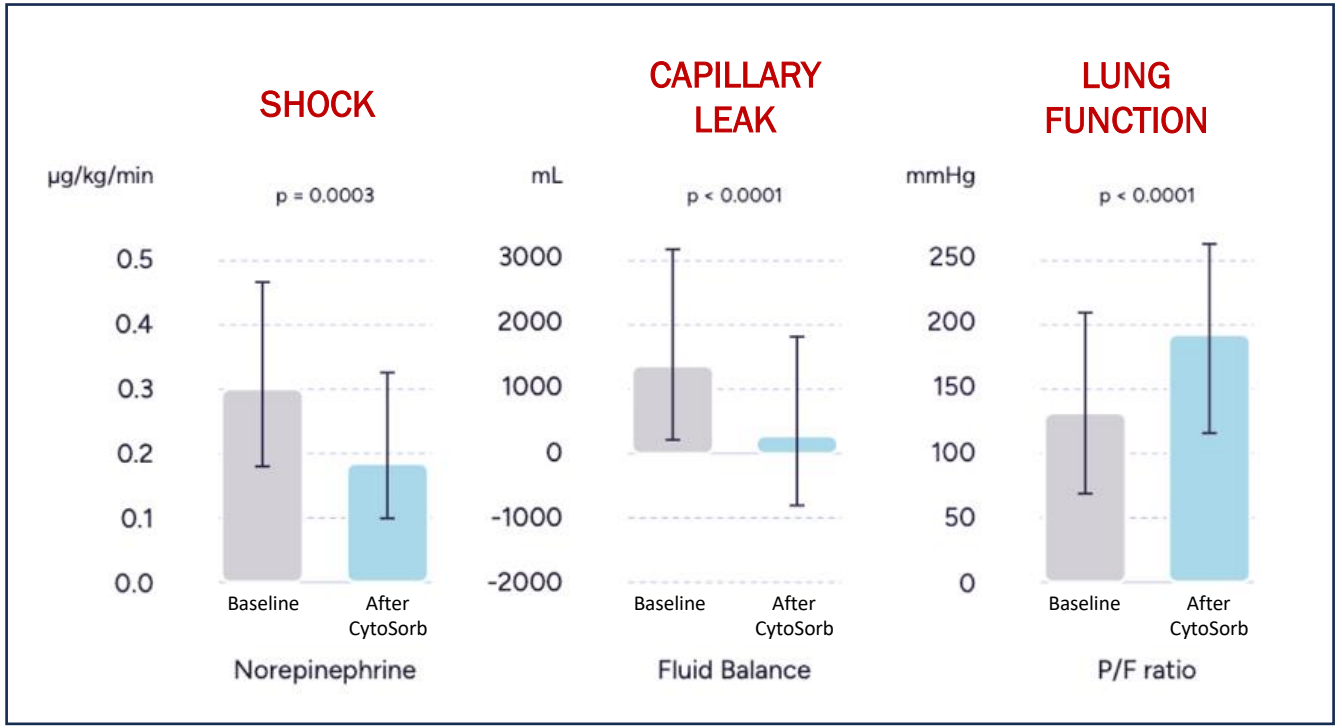
Hawchar, F., et al. Biomedicines 2021. 9(7):768
Kogelmann, K,, et al. J Clin Med 2024. 13-294

CytoSorbents™

2025: COSMOS Registry Reaffirms Results in Real-World Setting

COSMOS registry, patient characteristics

- Data from 150 patients analyzed
- Critical care indications:
  - Septic shock (57.6%)
  - Cardiogenic shock (12.9%)
  - Rhabdomyolysis (10.6%)
  - Acute/acute-on-chronic liver failure (10.6%)
  - Acute respiratory distress syndrome (6.8%)
  - Others (9.1%)



When added to standard therapy, CytoSorb treatment:

- Led to significant improvements in fundamental problems in critical illness: Shock, capillary leak, and lung function
- Observed mortality rates in Registry participants were lower compared with the predicted mortality rates according to standardized and established critical care risk scores

Ferrer, R, et al., J. Intensive Med. 2025 In press;

CytoSorbents™

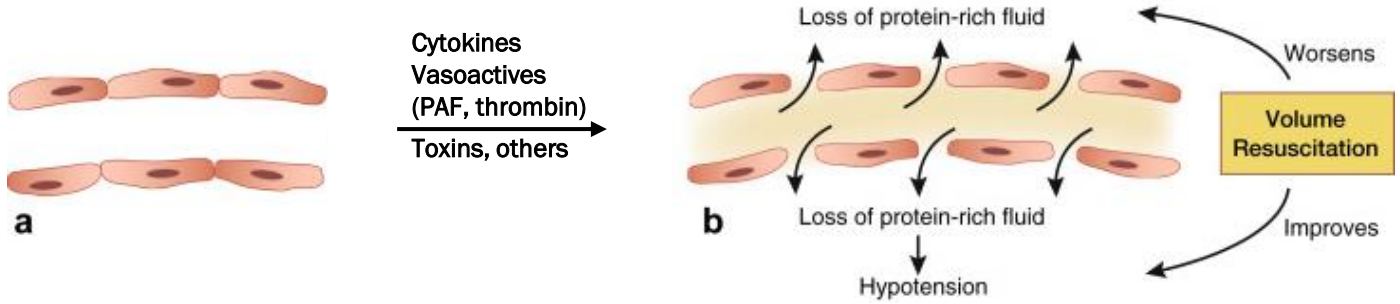
CytoSorb Treatment Goal #3:

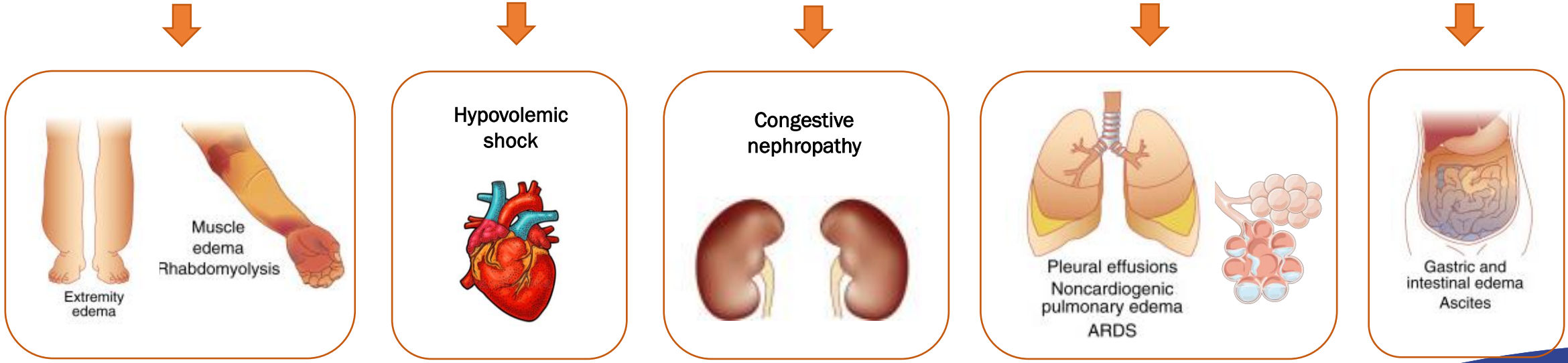
Promote Repair of Capillary Leak

CytoSorbents™

Capillary Leak Causes Organ Failure & Prevents Fluid Removal

Cytokine storm and other toxins cause damage to endothelial tight junctions and capillary leak syndrome. A reduction in cytokine storm through CytoSorb treatment helps to protect against this ongoing injury



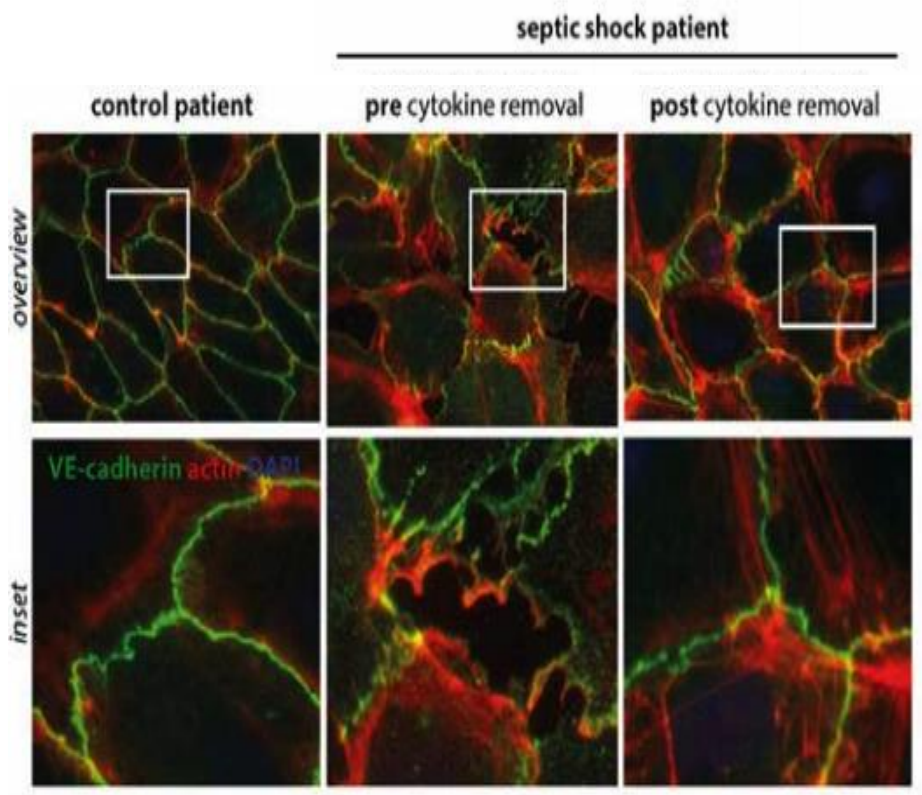


Siddall, E, et al. "Capillary leak syndrome: etiologies, pathophysiology, and management." Kidney Intl 2017. 92(1):37-46

CytoSorbents™

CytoSorb Protects Against Capillary Leak Syndrome

- A 32 yo woman was found unconscious and severely hypoxic from influenza pneumonia, and had ARDS on ECMO, septic shock, and acute kidney injury with a SOFA score = 18
- In spite of a broad anti-infective regimen, adequate fluid resuscitation, and high doses of vasopressors, she remained in refractory shock
- Her serum was obtained before and after 24-hour CytoSorb treatment and added to human umbilical endothelial cells (HUVEC) cultured *in-vitro* and analyzed via transendothelial electrical resistance and immunocytochemistry



Results showed that serum from the patient prior to treatment caused wide disruption of endothelial tight junctions, whereas serum from the same patient collected after CytoSorb treatment no longer disrupted tight junctions. It suggests that CytoSorb removes toxic agents to the endothelium.

David, S., et al. "Effect of extracorporeal cytokine removal on vascular barrier function in a septic shock patient." *Journal of intensive care* 5.1 (2017): 12.

CytoSorb Removes Endothelial Toxic Substances

frontiers | Frontiers in Immunology

Major endothelial damage markers identified from hemadsorption filters derived from treated patients with septic shock – endoplasmic reticulum stress and bikunin may play a role

Robin Kasper[1], Armando Rodriguez-Alfonso[2,3], Ludger Ständker[2], Sebastian Wiese[3] and E. Marion Schneider[1*]

[1]Clinic of Anesthesiology and Intensive Care Medicine, University Hospital Ulm, Ulm, Germany, [2]Core Facility Functional Peptidomics, Ulm University Medical Center, Ulm, Germany, [3]Core Unit Mass Spectrometry and Proteomics (CUMP), Ulm University, Ulm, Germany

Conclusion: Our results indicate that hemadsorption is efficient to transiently remove strong endothelial damage mediators from the blood of patients with septic shock, which explains a rapid clinical improvement of inflammation and endothelial function. The current work indicates that a combination of stressors leads to the most detrimental effects. Oxidized ssDNA, likely derived from mitochondria, SAA1, the chemokine CXCL7 and the human neutrophil peptide alpha-defensin 1 (HNP-1) were unique for their significant negative effect on endothelial cell viability. However, the strongest damage effect occurred, when, bikunin – cleaved off from alpha-1-microglobulin was present in high relative amounts (>65%) of protein contents in the most active fraction. Thus, a relevant combination of stressors appears to be removed by hemadsorption therapy which results in fulminant and rapid, though only transient, clinical restitution.

Fractions that inhibit endothelial cell growth

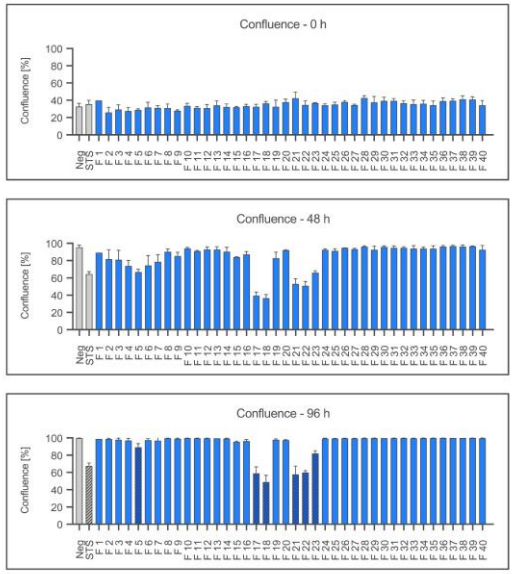


FIGURE 1
Proliferation of Ea.hy926 cells under the influence of CytoSorb fractions. Shown is the mean confluence and standard deviation of the endothelial cells measured using IncuCyte® ZOOM after 0, 48, and 96 hours in percent. Fractions (F) 1-40, the negative control with IMDM (Negative) and the positive control staurosponine (STS) 100 nM are displayed. Statistical analysis was performed after 96 hours. Significant differences to the negative control are marked as hatched columns.

Fractions that kill endothelial cells

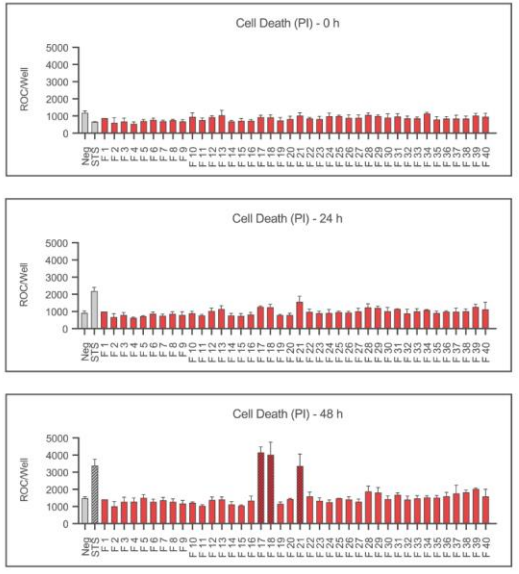


FIGURE 2
Cell death of Ea.hy926 cells under the influence of CytoSorb fractions. Displayed is the cell death of endothelial cells measured using IncuCyte® ZOOM after 0, 24 and 48 hours as mean red object count per well along with the standard deviation. Fractions (F) 1-40, the negative control with IMDM (Negative) and the positive control staurosporine (STS) 100 nM are shown. Statistical analysis was performed after 48 hours. Significant differences to the negative control are marked as hatched columns.

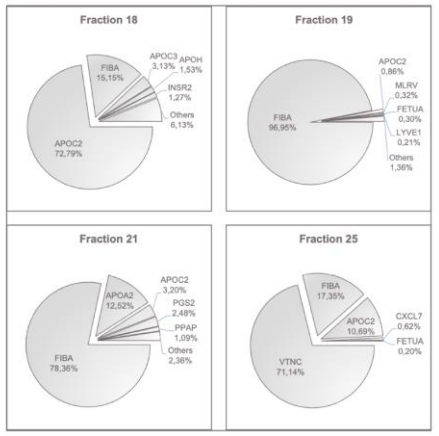


FIGURE 6
Peptides with the highest contents in fractions 18, 19, 21 and 25. The most prevalent peptides in each fraction are shown in percent of the determined intensities. APOC2, Apolipoprotein C-II; FIBA, Fibrinogen Alpha Chain; APOC3, Apolipoprotein C-III; APOH, Beta-2-Glycoprotein 1; INSR2, Insulin Isoform 2; MLRV, Myosin Regulatory Light Chain 2; FETUA, Fetuin A, Alpha-2-HS-Glycoprotein; LYVE1, Lymphatic Vessel Endothelial Hyaluronic Acid Receptor 1; APOA2, Apolipoprotein A-II; PGS2, Decorin; PPAP, Prostatic Acid Phosphatase; VTNC, Vitronectin; CXCL7, Platelet Basic Protein.

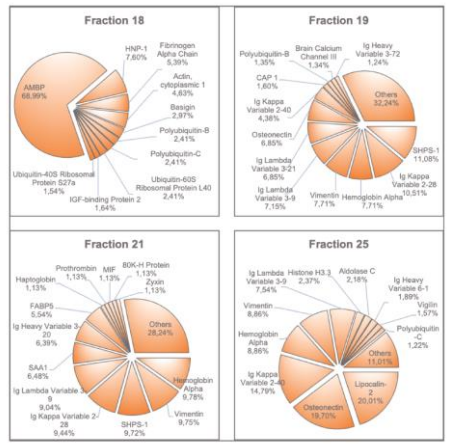


FIGURE 7
Proteins with the highest amounts found in fractions 18, 19, 21 and 25. The most prevalent proteins in each fraction are shown in percent of intensities. AMBP, Alpha-1-Microglobulin/Bikunin Precursor; HNP-1, Neutrophil Defensin 1; SHPS1, Tyrosine-Protein Phosphatase; CAP1, Adenylyl Cyclase-Associated Protein 1; SAA1, Serum Amyloid A-1 Protein; FABP5, Fatty Acid-Binding Protein 5; MIF, Macrophage Migration Inhibitory Factor; 80K-H Protein, Glucosidase 2 Subunit Beta.

By Removing Toxic Insults, the Endothelium Can Now Heal

- Healing of the endothelium, regeneration of the glycocalyx, and the reversal of capillary leak requires time on the order of days

- Some healing will take place while on CytoSorb therapy. However, this healing is expected to continue AFTER CytoSorb treatment is discontinued. The weaning of vasopressors is often used as an endpoint for CytoSorb therapy.

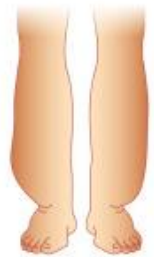
- From a fluid management standpoint, while on CytoSorb therapy, we recommend:
  - Liberal fluid resuscitation as needed in the midst of severe refractory shock
  - The use of packed red blood cells as a good intravascular volume expander and an important carrier of oxygen and carbon dioxide
  - As shock begins to resolve, consolidate IV fluids to limit fluid intake rather than trying to remove fluid via CRRT which directly removes volume from the intravascular space and can exacerbate shock
  - With each device change, it is essential that the blood in the device is flushed back into the patient (as long as the blood has not clotted). There is about 150 mL of blood, or a 1/3 of a pRBC unit, in each device, which if discarded, could also exacerbate anemia, hypovolemia, and shock and make it difficult to wean off vasopressors

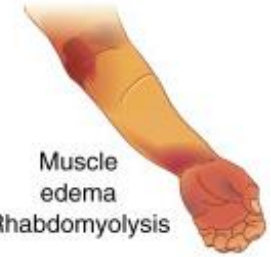
Treatment Goal #4:

Actively Remove Excessive Fluid

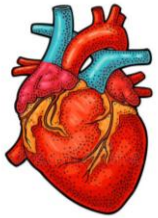
CytoSorbents™

Removal of Excessive Fluid Can Help Organ Function

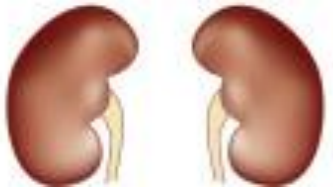


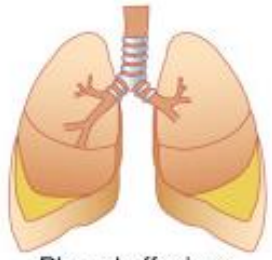


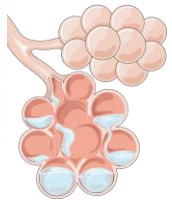
Hypovolemic shock



Congestive nephropathy







Pleural effusions
Noncardiogenic pulmonary edema
ARDS

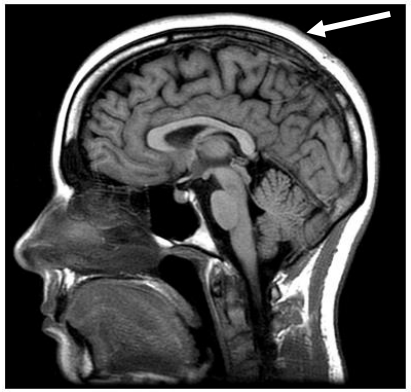
Gastric and intestinal edema
Ascites

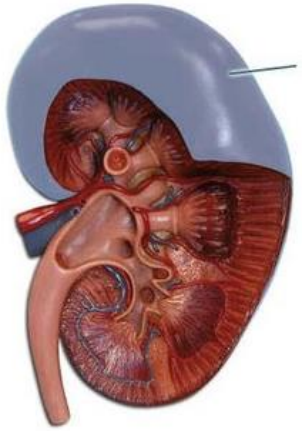
- Weaning of vasopressors and a resolution of shock, and an improvement in capillary leak offers an ideal starting point for active fluid removal via CRRT or diuresis
- Gradual fluid removal is recommended initially to avoid hypoperfusion and hypotension and then as tolerated
- Target is Dry Weight ± 5% (as measured by a bed scale) over time

CytoSorbents™

The Kidney is Like the Brain – Trapped in an Unexpandable Case

- Sepsis-associated Acute Kidney Injury (S-AKI) occurs in an estimated 1 in 6 ICU patients with sepsis
- Severe S-AKI significantly increases the risk of mortality and poses major challenges in fluid balance, acid-base status, and toxin removal
- Interestingly, in histopathologic studies of cadaver kidneys or renal biopsies, only ~30-40% of cases have identifiable damage (e.g. ATN, glomerular disease, etc) at a cellular level. The majority of cases have no significant pathology, just interstitial edema and some cellular infiltration
- Capillary leak causes renal edema, but just like the brain, because the kidneys are in a non-expandable fibrous renal capsule, at some point anuric AKI can develop as:
  - Increased swelling causes increased intra-renal pressures and decreased renal blood flow – particularly microcirculation – and regional ischemia
  - Importantly, nephrons collapse, choking off urine output





By helping to promote capillary leak reversal, CytoSorb enables the active removal of fluid via CRRT or diuresis, with the goal of decompressing the kidneys. Users have often observed a rapid return of urine output and improvement in renal function, consistent with renal edema, rather than structural damage to the kidney

White, KC, et al., Intensive Care Med (2023); 49(9):1079-1089.
Langenberg, C, et al., Critical Care (2008); 12:R38

CytoSorbents™

Simple Way to Think about CytoSorb Therapy

Right Patient		Hyperinflamed with organ dysfunction/failure
Right Timing		Early intervention following standard of care
Right Dosing	Frequency	Change device q8-q12h initially. As severity decreases, switch to q24h
	Flow	Higher flow rate = higher blood volumes treated (200-250 mL/min)
	Finish	Treat to clinical effect (particularly shock reversal), NOT just 2-3 devices

Treatment Goals:

1) Break the Vicious Cycle of Massive Uncontrolled Inflammation

2) Stabilize the patient (shock reversal, other organ dysfunction)

3) Promote Capillary Leak Reversal

4) Now Actively Remove Excessive Fluid from the Patient

Need to address #1, 2, and 3 to effectively achieve active fluid removal via CRRT/dialysis or diuresis

CytoSorbents™

Common Findings with CytoSorb Treatment

- Inflammatory marker reductions
  - IL-6 and other cytokines
  - PCT, CRP
  - Ferritin
- Improved Hemodynamic stabilization
  - Decreased need for vasopressors
  - Stabilization of fluid balance
  - Decrease in lactate levels
- Objective improvement in lung function (may be delayed)
  - On mechanical ventilation:
    - Improved P/F ratio, reduced FIO_2, ease of mechanical ventilation (decreased peak or plateau pressures, PEEP, improved lung compliance), improved ABG
  - On ECMO:
    - Reduced O_2, reduced hypercarbia, reduced sweep gas flow, improved ABG

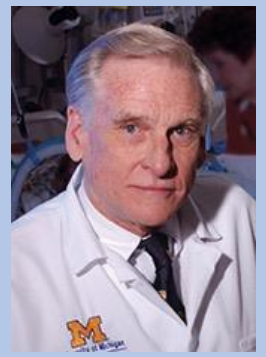
Expect improvement in lung function but do not expect to wean patients off of mechanical ventilation or decannulate from ECMO while on CytoSorb. CytoSorb removes the inflammatory toxins causing ongoing lung injury. Then it takes time for capillary leak to reverse and for the lungs to heal.

Also, in very significant inflammation, it may take more than one device change to begin to see improvements in hemodynamics.

CytoSorb + ECMO for Severe ARDS May Enable Enhanced Lung Rest

CytoSorb, in combination with ECMO, represents a novel and potentially effective Enhanced Lung Rest strategy to treat severe ARDS

Dr. Robert Bartlett - Pioneer of ECMO, and former CytoSorbents Chief Medical Officer for 10 years



- ECMO provides gas exchange and rests the lungs to prevent ventilator-induced lung injury (VILI)
- CytoSorb reduces circulating inflammatory cytokines and other mediators to prevent ongoing endothelial injury, allowing a reversal of capillary leak and the lungs to heal

The goal of cytokine adsorption with CytoSorb + ECMO in severe ARDS patients is to promote lung healing – a requisite to potentially faster weaning from mechanical ventilation and decannulation from ECMO

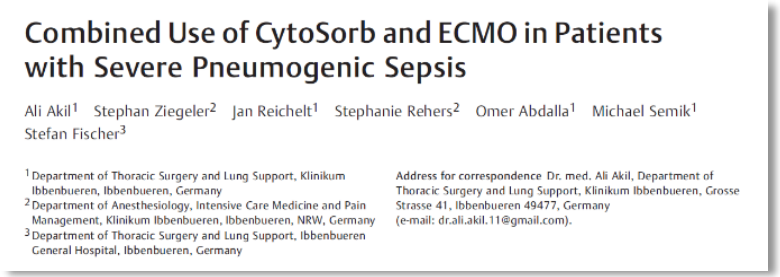
CytoSorbents™

Early Use of CytoSorb + ECMO to Treat Severe ARDS

- 13 patients prospectively enrolled with severe ARDS due to pneumonia and severe shock treated early with CytoSorb + ECMO (compared to ECMO only historical control; n=7)
  - Cause of pneumonia: 3 Influenza, 9 Bacterial, 1 Fungal vs 2 flu, 2 bacterial, 1 fungal, 2 unknown
  - Required: norepinephrine > 0.3 µg/kg/min, lactate > 2.0 mmol/L; PCT > 1 ng/ml
- CytoSorb + ECMO started ≤ 6 hrs from ICU admission and 12 hrs from sepsis diagnosis
  - All patients received at least 2 CytoSorb (max 3) changed every 24 hours
  - Blood flow rates 200-400 mL/min
- Baseline SAPS II score:
  - CytoSorb: 58±2 (range 49-66; predicted mortality 60%+)
  - Control: 50±2 (range 42-55; predicted mortality 50%+)

Results:

- CytoSorb use resulted in rapid hemodynamic stabilization
- Shorter mean duration on ECMO: CytoSorb: 8±2 days (range 2-23 days) versus Control: 19±3 days (range 13-30 days). Note: 8 days is 5 days after end of CytoSorb
- Mean ICU stay: CytoSorb: 26±6 days (range 7-63 days) versus Control: 26±5 days (range 8-43 days) impacted by survival bias
- Lower 30-day mortality: 0% treatment vs 57% control. All CytoSorb treated patients were alive at 60 days. Cause for death in control patients was sepsis with multi-organ failure



Combined Use of CytoSorb and ECMO in Patients with Severe Pneumogenic Sepsis

Ali Akil[1] Stephan Ziegeler[2] Jan Reichelt[1] Stephanie Rehers[2] Omer Abdalla[1] Michael Semik[1] Stefan Fischer[3]

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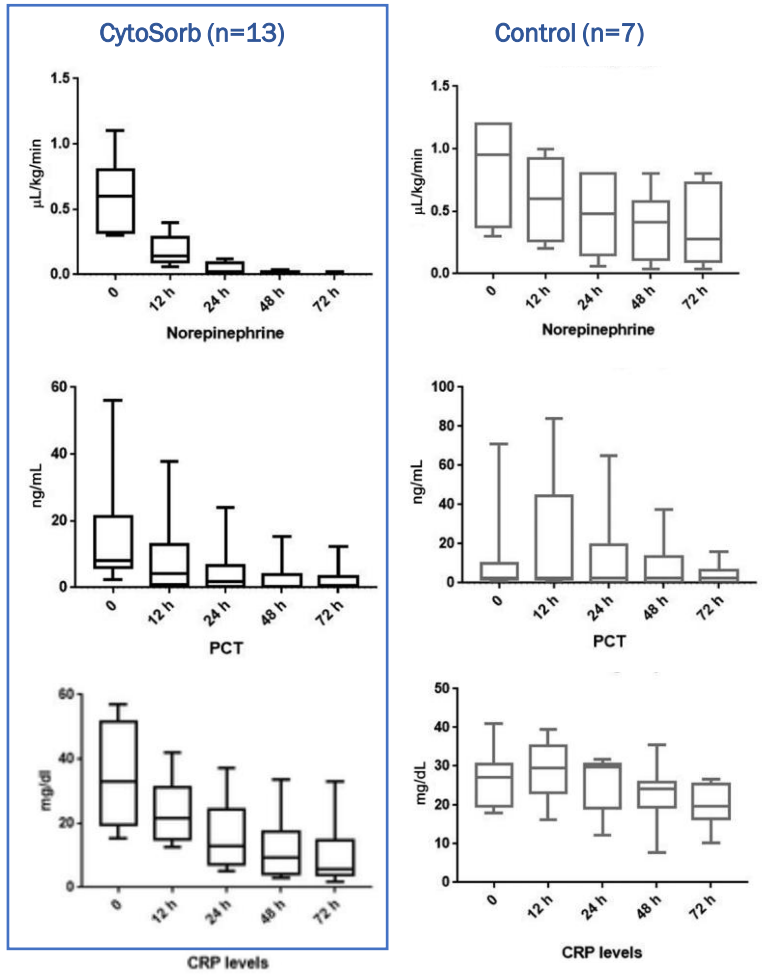
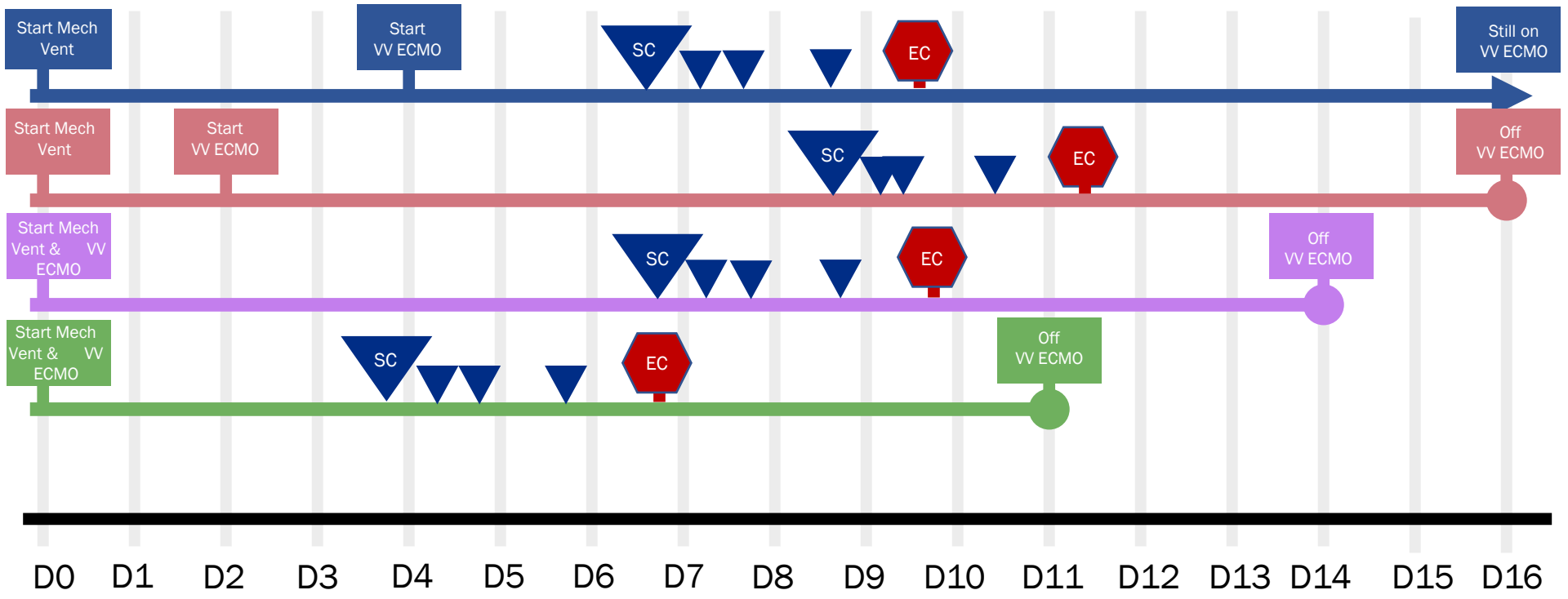
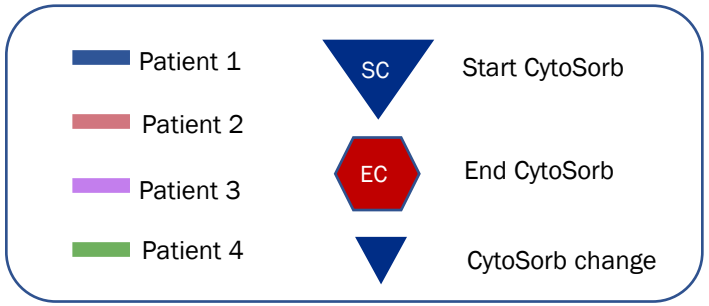


Akil, A, Fischer, S et al., Combined use of CytoSorb and ECMO in Patients with Severe Pneumogenic Sepsis. Thorac Cardiovasc Surg. 2021. 69(3):246-251.

NYU Patient Intervention Timeline with CytoSorb + ECMO on First 4 COVID-19 Patients Pre-EUA





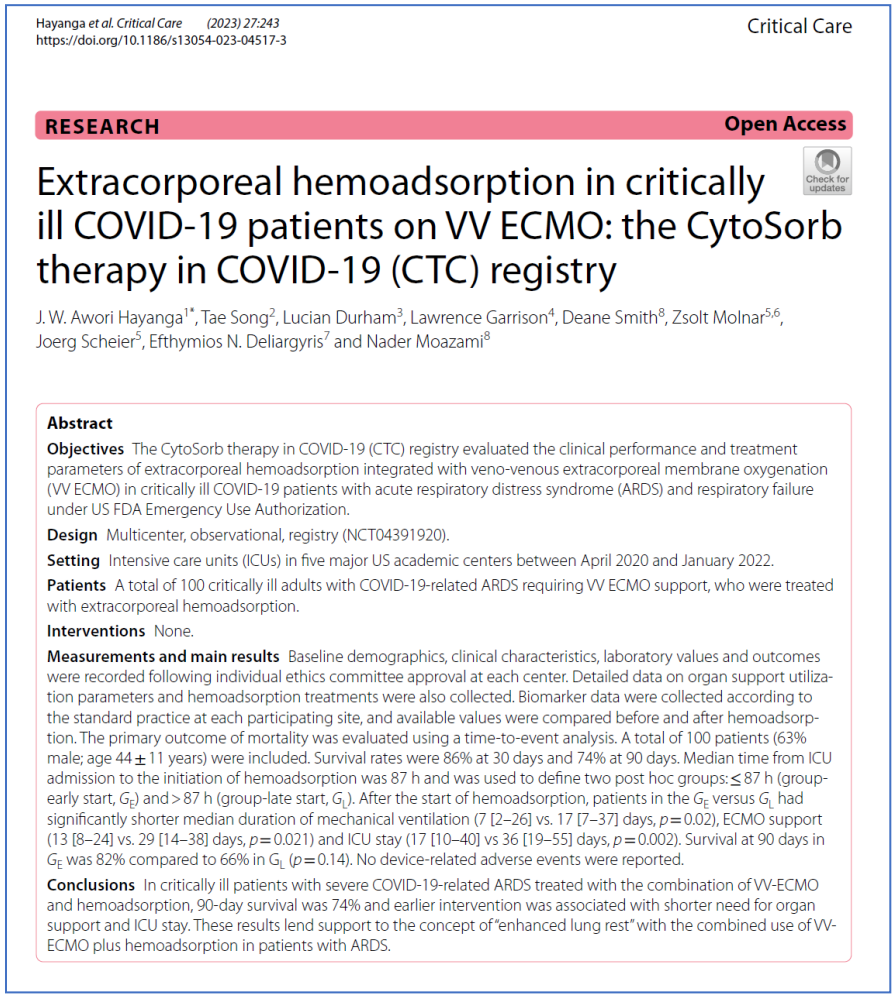
Small patient cohort but trend is similar to previous Akil paper and raises interesting question of whether earlier intervention with ECMO AND CytoSorb may help patients get off ECMO faster or more reproducibly (within 5 days of last CytoSorb treatment)

 * Data courtesy of Dr. Nader Moazami

NYU Langone MEDICAL CENTER

CytoSorbents™

CytoSorb Therapy in COVID-19 (CTC) Registry under FDA EUA



Hayanga *et al. Critical Care* (2023) 27:243
https://doi.org/10.1186/s13054-023-04517-3

Critical Care

RESEARCH Open Access

Extracorporeal hemoadsorption in critically ill COVID-19 patients on VV ECMO: the CytoSorb therapy in COVID-19 (CTC) registry

J. W. Awori Hayanga[1*], Tae Song[2], Lucian Durham[3], Lawrence Garrison[4], Deane Smith[8], Zsolt Molnar[5,6], Joerg Scheier[5], Efthymios N. Deliargyris[7] and Nader Moazami[8]

Abstract

Objectives The CytoSorb therapy in COVID-19 (CTC) registry evaluated the clinical performance and treatment parameters of extracorporeal hemoadsorption integrated with veno-venous extracorporeal membrane oxygenation (VV ECMO) in critically ill COVID-19 patients with acute respiratory distress syndrome (ARDS) and respiratory failure under US FDA Emergency Use Authorization.

Design Multicenter, observational, registry (NCT04391920).

Setting Intensive care units (ICUs) in five major US academic centers between April 2020 and January 2022.

Patients A total of 100 critically ill adults with COVID-19-related ARDS requiring VV ECMO support, who were treated with extracorporeal hemoadsorption.

Interventions None.

Measurements and main results Baseline demographics, clinical characteristics, laboratory values and outcomes were recorded following individual ethics committee approval at each center. Detailed data on organ support utilization parameters and hemoadsorption treatments were also collected. Biomarker data were collected according to the standard practice at each participating site, and available values were compared before and after hemoadsorption. The primary outcome of mortality was evaluated using a time-to-event analysis. A total of 100 patients (63% male; age 44 ± 11 years) were included. Survival rates were 86% at 30 days and 74% at 90 days. Median time from ICU admission to the initiation of hemoadsorption was 87 h and was used to define two post hoc groups: ≤ 87 h (group-early start, G_E) and > 87 h (group-late start, G_L). After the start of hemoadsorption, patients in the G_E versus G_L had significantly shorter median duration of mechanical ventilation (7 [2–26] vs. 17 [7–37] days, $p = 0.02$), ECMO support (13 [8–24] vs. 29 [14–38] days, $p = 0.021$) and ICU stay (17 [10–40] vs 36 [19–55] days, $p = 0.002$). Survival at 90 days in G_E was 82% compared to 66% in G_L ($p = 0.14$). No device-related adverse events were reported.

Conclusions In critically ill patients with severe COVID-19-related ARDS treated with the combination of VV-ECMO and hemoadsorption, 90-day survival was 74% and earlier intervention was associated with shorter need for organ support and ICU stay. These results lend support to the concept of "enhanced lung rest" with the combined use of VV-ECMO plus hemoadsorption in patients with ARDS.

In the largest multi-center study in 100 consecutive critically ill COVID-19 patients with hyperinflammation and refractory respiratory failure treated with CytoSorb and ECMO:

1) Patient were treated with 72 hours of CytoSorb (q12h x 1 day, q24h x 2 days) with CytoSorb integrated into ECMO circuit at 400-600 mL/min

2) Combination of CytoSorb with ECMO resulted in 74% 90-day survival. For context, ELSO Registry reports 53% 90-day survival in North American COVID-19 patients receiving ECMO alone without CytoSorb

3) Early use of CytoSorb (G_E : before the median time from ICU admission to CytoSorb treatment = 87h) with ECMO is associated with significantly shorter times on ECMO and mechanical ventilation, and shorter ICU stays, and a trend to faster reversal of shock and weaning from vasopressors, compared to later use

4) We believe these data support the use of CytoSorb with ECMO in this patient population to achieve **"enhanced lung rest"**, where ECMO rests the lungs and reduces risk of VILI, while CytoSorb removes circulating inflammatory toxins that can otherwise perpetuate or worsen capillary leak and ARDS, giving the lungs a chance to heal and recover

Hayanga, JWA., et al., "Extracorporeal hemoadsorption in critically ill COVID-19 patients on VV-ECMO: the CytoSorb therapy in COVID-19 (CTC) registry," Critical Care 2023; 27:243

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Early Treatment with CytoSorb Improves Outcome

Early treatment with ECMO and CytoSorb led to the best clinical outcomes, including less time on mechanical ventilation, ECMO, and in the ICU, a trend to lower need for vasopressors, and supports the concept of "enhanced lung rest" where ECMO reduces ventilator-induced lung injury while CytoSorb reduces circulating cytokines and other inflammatory toxins to allow the lungs to heal and reduce capillary leak syndrome

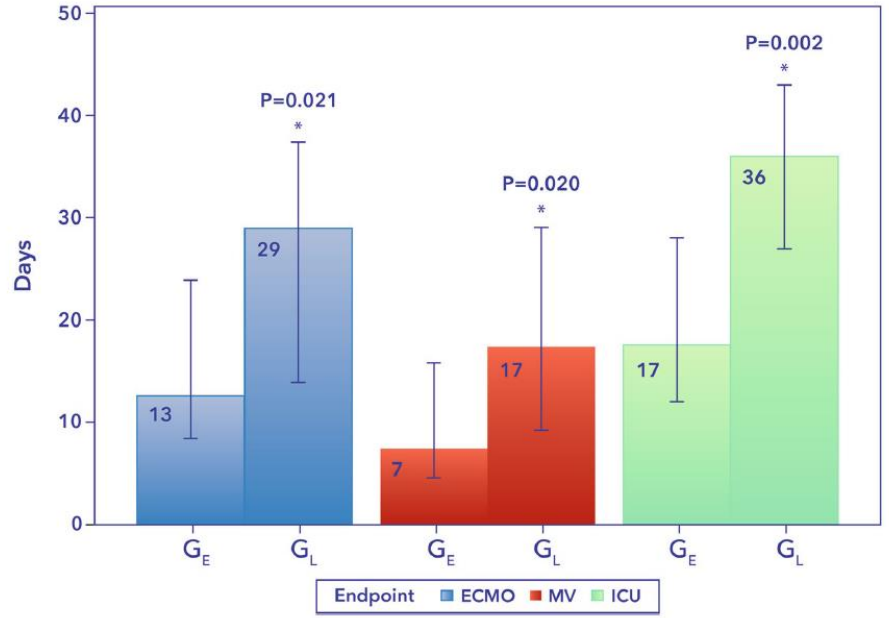


Fig. 1 Days on organ support and ICU stay in G_E and G_L. G_E, early group; G_L, late group; ECMO, extracorporeal membrane oxygenation; MV, mechanical ventilation; ICU, intensive care unit

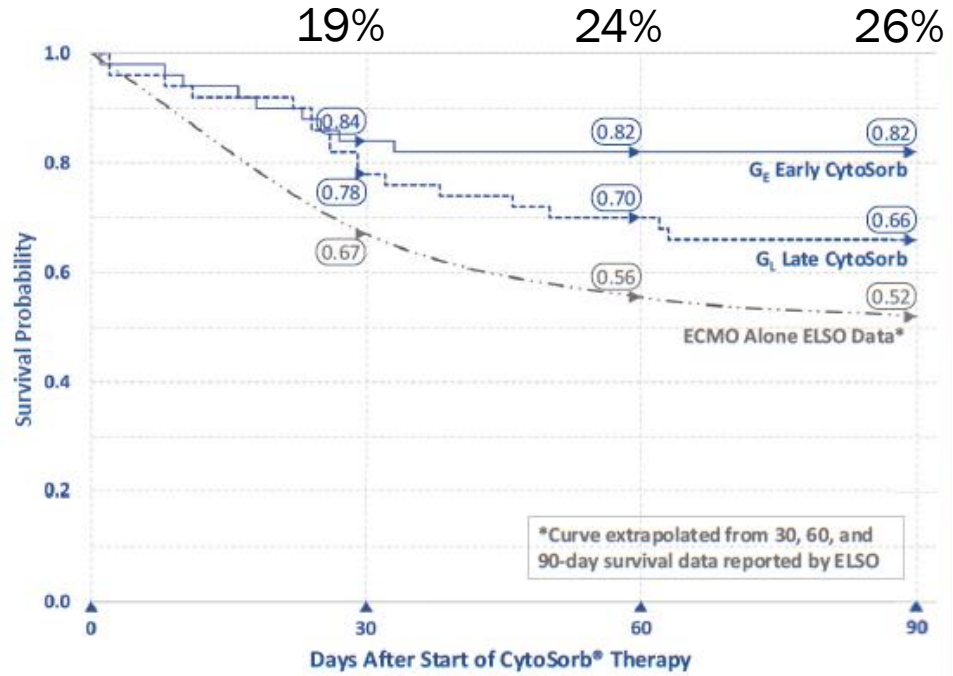


Fig. 2 Kaplan–Meier plot for probability of survival from admission to intensive care unit to 90 days in G_E and G_L. p=0.140 for 90-day survival difference between G_E and G_L. ELSO, Extracorporeal Life Support Organization.

* Modified from the published manuscript

Table 2 Clinical course parameters

	Total $n=100$	G_E $n=50$	G_L $N=50$	p-value
Vasopressor support, n (%)	76 (76)	38 (76)	38 (76)	1.000
Vasopressor support after HA start (days)	5 [3–21]	4 [1–17]	7.5 [4–21]	0.128
Cumulative fluid balance (mL)	1988 [– 933 to 4727]	990 [– 993 to 3894]	2322 [– 969 to 5957]	0.336

Data are presented as n(%) and median[interquartile range]

Hayanga, JWA., et al., "Extracorporeal hemoadsorption in critically ill COVID-19 patients on VV-ECMO: the CytoSorb therapy in COVID-19 (CTC) registry," Critical Care 2023; 27:243

CytoSorbents™

2025: Early & Intensive Treatment with More Blood Treated Works Best

Original Research

Real-World Outcomes of Hemoadsorption with CytoSorb® in Patients with Septic Shock: Insights from a Single-Center Study

Giorgio Berlot, MD[1], Paolo Carocci, MD[1], Valentina Votrico, MD[2], Barbara Iacoviello, MD[1], Nicolò Taverna, MD[1], Ugo Gerini, MD[3], Vittorio di Maso, MD[4], and Ariella Tomasini, MD[1]

- Large, retrospective single center study
- 175 patients with septic shock treated with CytoSorb
- Evaluated the impact of early versus late, and low versus high intensity treatment with CytoSorb on mortality compared to predicted mortality based on illness severity scores

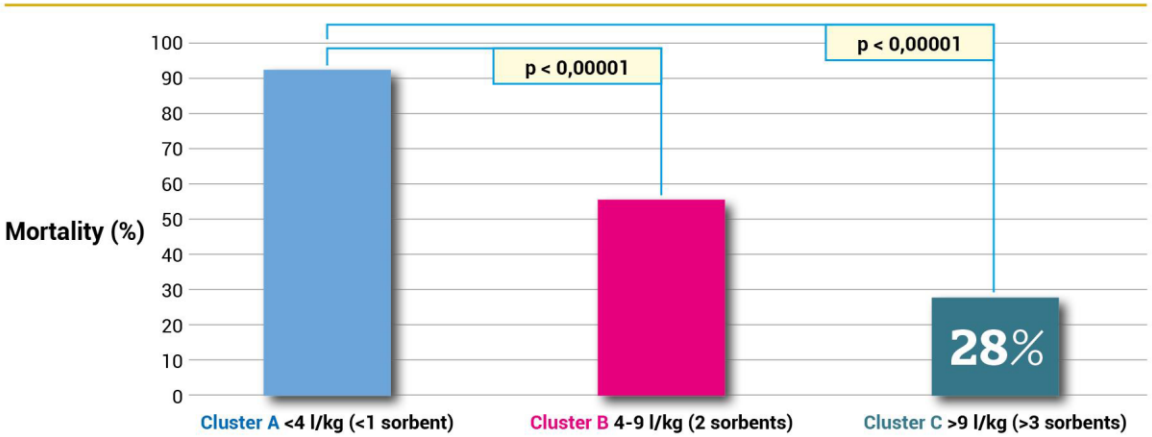
Early and Intensive Treatment with CytoSorb Doubles Survival Expectation

Table 3. Observed Versus Expected Mortality (%).

Categories	Expected mortality (%)	Observed mortality (%)	p-value
All (n = 175)	66	49	0.048
Early starters (n = 102)	66	48	n.s.
Late starters (n = 73)	70	51	n.s.
High intensity (n = 90)	63	30	0.002
Low intensity (n = 85)	71	69	n.s.
Early starters-high intensity (n = 56)	63	30	0.02
Late starters-low intensity (n = 38)	74	68	n.s.

Abbreviations: n.s., nonsignificant.

The More Blood Purified with CytoSorb, the Higher the Survival. Treatment Intensity is Key



*Berlot, G, et al., J. Intensive Care Med. **2025** Sep; 40(9):993-1000; 14(7), 2285; doi: 10.1177/08850666251331905.*

CytoSorb Improves Survival in Septic Shock Meta-Analysis

Journal of Clinical Medicine

MDPI

Systematic Review

Hemoadsorption in the Management of Septic Shock: A Systematic Review and Meta-Analysis

David Steindl [1,†], Tim Schroeder [2,†], Alexander Krannich [3,*] and Jens Nee [2]

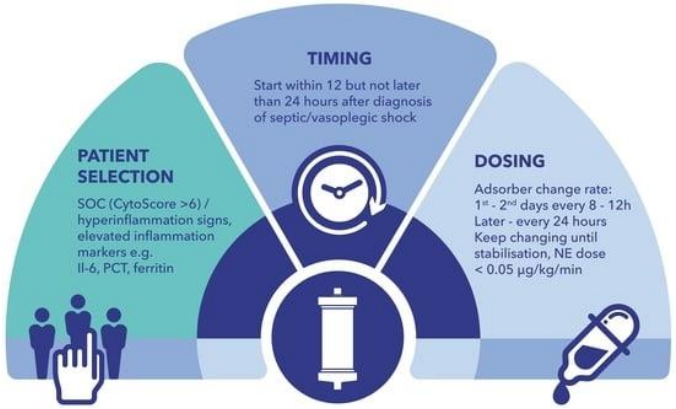
CHARITÉ UNIVERSITÄTSMEDIZIN BERLIN

- Meta-analysis of 744 septic shock patients from 1 RCT and 8 observational studies from 2019-2024, of which 449 patients were treated with CytoSorb
- CytoSorb reduced in-hospital mortality (OR 0.64, p=0.04)
- 28-30-day mortality was also halved with CytoSorb (OR 0.46, p=0.003) than without (p=0.003)
- Significant hemodynamic improvement with reductions in vasopressor need in CytoSorb patients again confirmed

Steindl, D., et a;., J. Clin. Med. 2025, 14(7), 2285; https://doi.org/10.3390/jcm14072285

CytoSorbents™

Summary



- Massive uncontrolled inflammation can rapidly cause organ failure and death

- Treating hyperinflammation requires patience
  - Right Patient — Hyperinflamed with organ dysfunction/failure
  - Right Timing — Early intervention following standard of care
  - Right Frequency — Change device q12h initially. When severity decreases, switch to q24
  - Right Flow — Higher flow rate = higher blood volumes treated (200-250 mL/min)
  - Right Finish — Treat to clinical effect (particularly shock reversal), NOT just 2-3 devices

- Goals of CytoSorb Use:
  - Break the Cycle of Massive Inflammation
  - Reverse Shock and other Instability
  - Help Promote Capillary Leak Reversal
  - **Now Actively Remove Excess Fluid from Patient!**

- Enhanced Lung Rest with CytoSorb + ECMO is an excellent example of what is possible

- Importantly, these same principles can be applied to the treatment of septic shock and a wide range of other critical illnesses

CytoSorbents™

CytoSorbents Corporation

NASDAQ: CTSO

Company Contact:
Dr. Phillip Chan
pchan@cytosorbents.com
908-307-0367
www.cytosorbents.com

Case of acute pancreatitis and septic shock

Prof. Dr. Zsolt Molnár, MD, PhD
Head of Department of Anesthesiology and Intensive Therapy
Semmelweis University – Budapest, Hungary

Initial event: acute pancreatitis

- 36 year old male
  - Acute alcoholic pancreatitis
  - MOF for 2 weeks
  - IAP > 20 cmH_2O
  - Laparotomy – open abdomen with VAC
  - Regular VAC exchanges
  - Perforated colon – Right hemicolectomy

1 day before 3rd VAC exchange

Circulation

Heart rate (min^{-1})	96
MAP (mmHg)	72
$ScvO_2$ (%)	75
Vasopressors	-
Inotropes	-

Respiration

FiO_2	40
PEEP (cmH_2O)	13
PaO_2 (mmHg)	98

Renal function

Creat (umol/L)	73
UO (mL/h)	100-200
pH	7.40
HCO_3 (mmol/L)	21.6
Lactate (mmol/L)	1.8

Inflammation

PCT (nmol/L)	0.61
CRP (mmol/L)	312

During 3rd VAC exchange

- Perforated cecum was found
- Right hemicolectomy
- Intra-operative course:
  - Increasing vasopressor need
  - Increased FiO_2, PEEP

Before-after VAC exchange

Circulation

	Before	After
Heart rate (min^{-1})	96	118
MAP (mmHg)	72	68
$ScvO_2$ (%)	75	78
Norepinephrine (ug/kg/min)	--	0.59
Vasopressin (NE/min)	--	0.02

Renal function

Creat (umol/L)	73	188
UO (mL/h)	100-200	0
pH	7.40	7.31
HCO_3 (mmol/L)	21.6	18.8
Lactate (mmol/L)	1.8	1.6

Respiration

	Before	After
FiO_2	40	80
PEEP (cmH_2O)	13	28/6
PaO_2 (mmHg)	98	91

DAMP, PAMP or both?

Inflammation

PCT (nmol/L)	0.61	63.0
CRP (mmol/L)	312	385

What would you do?

Before-after 12 hrs CytoSorb treatment

Circulation

	Before	After
Heart rate (min^{-1})	118	95
MAP (mmHg)	68	73
$ScvO_2$ (%)	78	72
Norepinephrine (ug/kg/min)	0.59	0.07
Vasopressin (NE/min)	0.02	--

Reduced NE

Respiration

	Before	After
FiO_2	80	40
PEEP (vízcm)	28/6	16
PaO_2 (Hgmm)	91	129

Improved P/F

Renal function

Creat (umol/L)	188	134
UO (mL/h)	0	100
pH	7.31	7.46
HCO_3 (mmol/L)	18.8	23.4
Lactate (mmol/L)	1.6	1.7

Inflammation

PCT (nmol/L)	63.0	20.1
CRP (mmol/L)	385	[illegible]

Reduced inflammation (!)

Take home message

- Surgery can trigger severe hyperinflammatory response
- Early (<12 hrs) treatment seemed extremely effective

Was it due to:

- Source control?
- ABs?
- HA?
- Patient himself?

Case Reports and the Use of the CytoScore

Dr. med. Tobias Hübner, MD DESA EDIC MHBA
Chief Physician of the Intensive Care Unit and Vice Head of the Department of Anesthesia and Intensive Care Hospital of Münsterlingen – Münsterlingen, Switzerland

The CytoScore

Dynamic Scoring System

Journal of Clinical Medicine

MDPI

Article

First Evaluation of a New Dynamic Scoring System Intended to Support Prescription of Adjuvant CytoSorb Hemoadsorption Therapy in Patients with Septic Shock

Klaus Kogelmann [1,*], Tobias Hübner [2], Franz Schwameis [3], Matthias Drüner [1], Morten Scheller [1] and Dominik Jarczak [4]

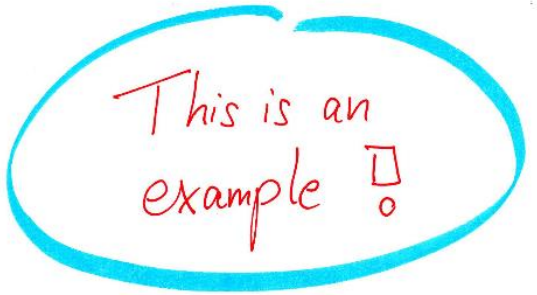
?

Dynamic Scoring System (DSS) to track early evolution of septic shock

	0 Points	1 Points	2 Points	Score
Lactate mmol/l	< 2.0		≥ 2.0	
Lactate change / 6 hours	↓ decreased	↑ ≤ 50 %	↑ > 50 %	
Norepinephrine µg/kg/min (mean arterial pressure = 65 mmHg)	< 0.1		≥ 0.1	
Norepinephrine change / 6 hours	↓ decreased	↑ ≤ 50 %	↑ > 50 %	
2nd catecholamine / vasopressor	No	Yes		
Hydrocortisone use	No	Yes		
Volume bolus 30 ml/kgbw	No	< 2 boli	≥ 2 boli	
			Total	

Case 1: Urosepsis Case Report

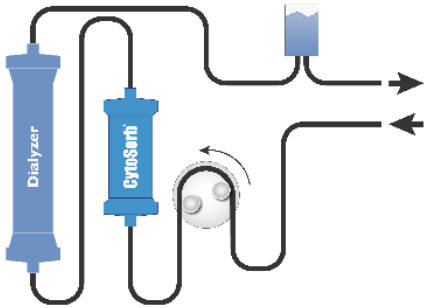
Example from our hospital



Diagnosis Septic shock (Urosepsis) → **Start standard therapy** (Started in ED) → **CytoScore after 6h: 6** → **Start CytoSorb within next 1 hour**

- Lactate 0.8 mmol/l
- NE 0.1 µg/kg/min

- Lactate 3.9 mmol/l
- NE 0.25 µg/kg/min
- Dbt 1.5 µg/kg/min
- Volume therapy ↑



Dynamic Scoring System (DSS) to track early evolution of septic shock

	0 Points	1 Points	2 Points	Score
Lactate mmol/l	< 2.0		≥ 2.0	
Lactate change / 6 hours	↓ decreased	↑ ≤ 50 %	↑ > 50 %	1
Norepinephrine µg/kg/min (mean arterial pressure = 65 mmHg)	< 0.1		≥ 0.1	1
Norepinephrine change / 6 hours	↓ decreased	↑ ≤ 50 %	↑ > 50 %	1
2nd catecholamine / vasopressor	No	Yes		1
Hydrocortisone use	No	Yes		
Volume bolus 30 ml/kgbw	No	< 2 boli	≥ 2 boli	2
			Total	6

Uroseptic Patient:

→ ER to ICU

→ SOFA 8

→ Cytoscore 6

Example from our hospital

This is an example !

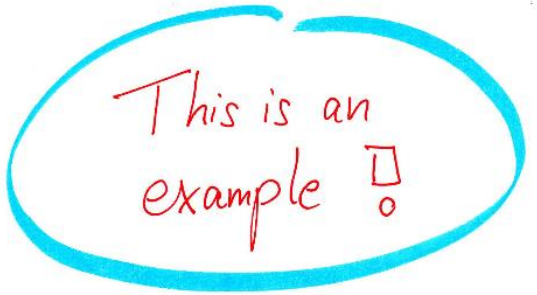
Time

		Ent.Dat. Ein.Dat Befund-Nr. Visum	22.08.2021 So 05:30 22.08.2021 So 06:01 21336592 nicht visiert	21.08.2021 Sa 05:30 21.08.2021 Sa 05:50 21335925 nicht visiert	20.08.2021 Fr 05:30 20.08.2021 Fr 05:34 21334803 nicht visiert
Klinische Chemie					
Calcium	2.1 - 2.6	mmol/l	2.62 H	2.34	
korr. Calcium (Alb.Konz)	2.1 -2.65	mmol/l	2.94 H	2.74 H	
Magnesium	0.7 - 1.10	mmol/l	1.34 H	0.97	1.17 H
Phosphat anorgan.	0.87 - 1.45	mmol/l	0.73 L	1.61 H	1.72 H
Harnstoff	< 11.9	mmol/l	8.7	9.8	18.4 H
Kreatinin	62 - 106	µmol/l	104	129 H	157 H
eGFR CKD-EPI	>= 90	ml/min/1.73m2	64 L (1)	49 L	39 L
C-reaktives Protein	< 5	mg/l	462 H	515 H	372 H
Procalcitonin	< 0.5	ng/ml	59.90 H	85.90 H	9.34 H
Interleukin-6	< 7.0	pg/ml	302.0 H	15525.0 H	129367.0 H

ON TIME

Case 2: Abdominal Septic Shock

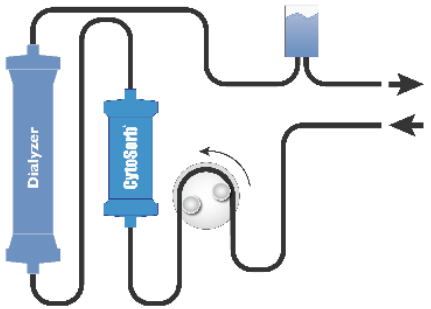
Example from our hospital



Diagnosis Septic shock (Abdominal Sepsis) → **Start standard therapy** (Started in OT) → **CytoScore after 6hrs > 8** → **Start CytoSorb within next 36 hrs**

- Lactate 3 mmol/l
- NE 0.2 µg/kg/min

- Lactate 7.0 mmol/l
- NE 0.55 µg/kg/min
- Dbt 1.5 µg/kg/min
- Hydrocortisone
- Volume therapy ↑



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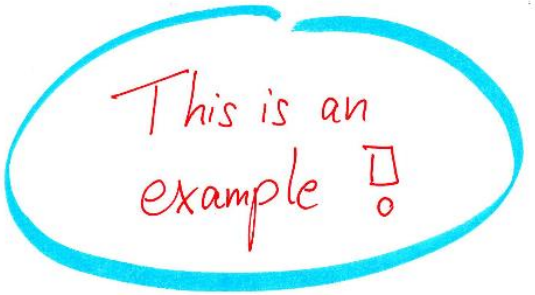
Lactate mmol/l	< 2.0		≥ 2.0	2
Lactate change / 6 hours	↓ decreased	↑ ≤ 50 %	↑ > 50 %	1
Norepinephrine µg/kg/min (mean arterial pressure = 65 mmHg)	< 0.1		≥ 0.1	2
Norepinephrine change / 6 hours	↓ decreased	↑ ≤ 50 %	↑ > 50 %	1
2nd catecholamine / vasopressor	No	Yes		1
Hydrocortisone use	No	Yes		1
Volume bolus 30 ml/kgbw	No	< 2 boli	≥ 2 boli	2
			Total	10

Duodenal Perforation:
→ ER → NW → OT → ICU
→ SOFA 15
→ CytoScore 10

Example from our hospital



TOO LATE

Time

		Befund-Nr. Visum	Do 05:42 18521879 nicht visiert	Mi 23:35 18521802 nicht visiert	Mi 05:21 18521361 nicht visiert	Di 05:18 18520872 nicht visiert	Mo 05:14 18520040 nicht visiert	So 15:38 18515582 nicht visiert
Klinische Chemie								
Natrium	136 - 145	mmol/l						
Kalium	3.4 - 5.0	mmol/l						
Calcium	2.1 - 2.6	mmol/l	2.53	2.44	2.07 L	2.23	2.20	
korr. Calcium (Alb.Konz)	2.1 -2.65	mmol/l	2.81	2.66	2.45	2.57	2.62	
Magnesium	0.7 - 1.10	mmol/l	0.95		1.05	0.85	1.28 H	
Phosphat anorgan.	0.87 - 1.45	mmol/l	1.51 H		0.86 L	1.02	1.10	
Harnstoff	1.7 - 8.3	mmol/l	3.5		3.8	4.0	7.9	
Kreatinin	62 - 106	µmol/l	80		93	105	145 H	
Kreatinin Rapid	62 - 106	µmol/l						
eGFR CKD-EPI	>= 90	ml/min/1.73m2	94		78 (5)	68 (5)	46 (6)	
C-reaktives Protein	< 5	mg/l	161 H		221 H	228 H	183 H	
Procalcitonin	< 0.5	ng/ml				35.82 H	68.41 H	
Interleukin-6	< 3.2	pg/ml				5475.0		>50000.0

Final remarks

Validation

still to be done

Journal of *Clinical Medicine*

MDPI

Article

First Evaluation of a New Dynamic Scoring System Intended to Support Prescription of Adjuvant CytoSorb Hemoadsorption Therapy in Patients with Septic Shock

Klaus Kogelmann [1,*], Tobias Hübner [2], Franz Schwameis [3], Matthias Drüner [1], Morten Scheller [1] and Dominik Jarczak [4]

Past — Present — Future

CytoScore is a decision-making tool to start with CS.

CytoScore is NOT a monitoring tool for effectiveness of CytoSorb therapy!

Final remarks

Journal of Clinical Medicine
MDPI

Article

First Evaluation of a New Dynamic Scoring System Intended to Support Prescription of Adjuvant CytoSorb Hemoadsorption Therapy in Patients with Septic Shock

Klaus Kogelmann [1,*], Tobias Hübner [2], Franz Schwameis [3], Matthias Drüner [1], Morten Scheller [1] and Dominik Jarczak [4]

- **CytoScore can be established at any ICU in the world**
- **It can be easily integrated in daily routine**
- **No need for new devices**

- **CytoScore is helping to do the right thing at the right moment**

Dynamic Scoring System (DSS) to track early evolution of septic shock

	0 Points	1 Points	2 Points	Score
Lactate mmol/l	< 2.0		≥ 2.0	
Lactate change / 6 hours	↓ decreased	↑ ≤ 50 %	↑ > 50 %	
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			Total	

Alexianer
KLINIKUM HOCHSAUERLAND
Ein Unternehmen der Alexianer
und der St. Johannes- und Maria-Stiftung

ecmo
ZENTRUM HOCHSAUERLAND

Case and local approach from Arnsberg

Priv.-Doz. Dr. med. Kevin Pilarczyk, MHBA

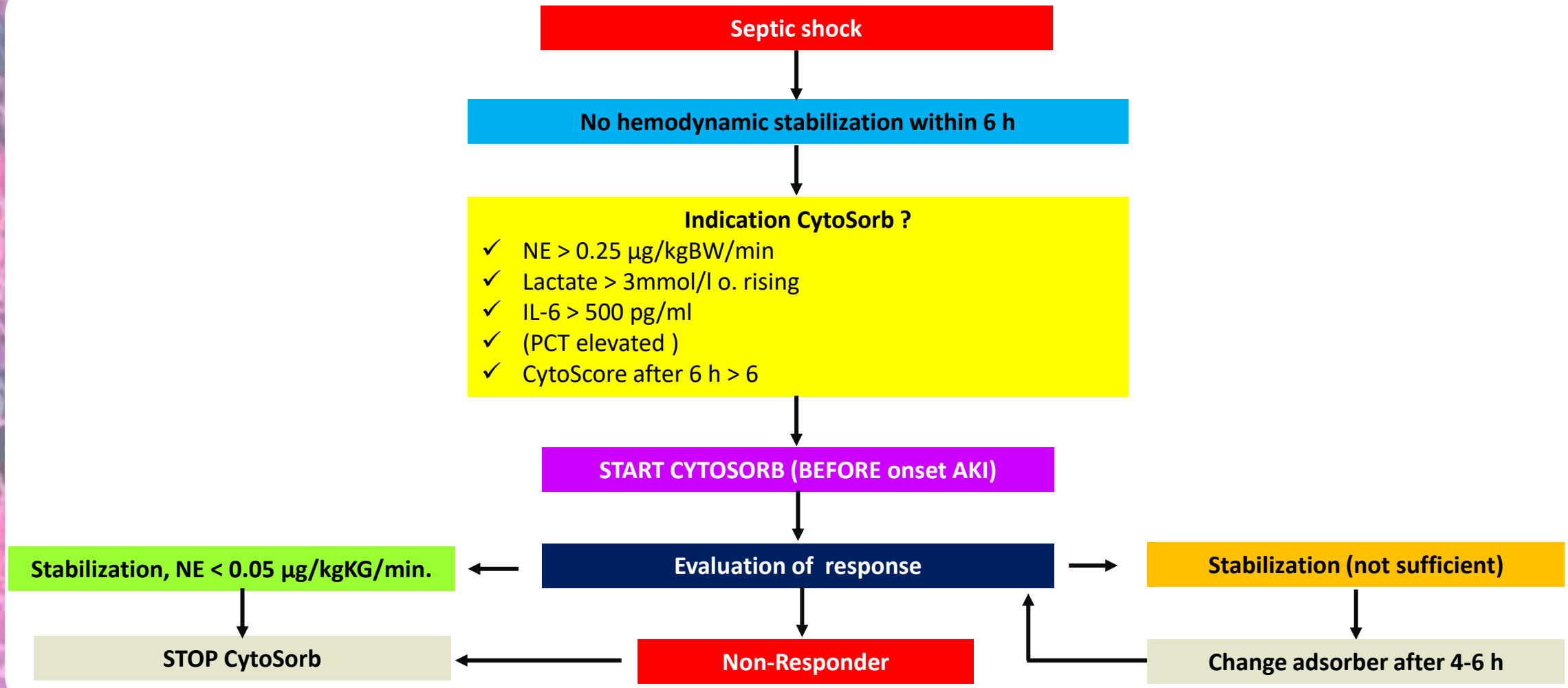
HIT HARD AND EARLY !!!!

When to start ???

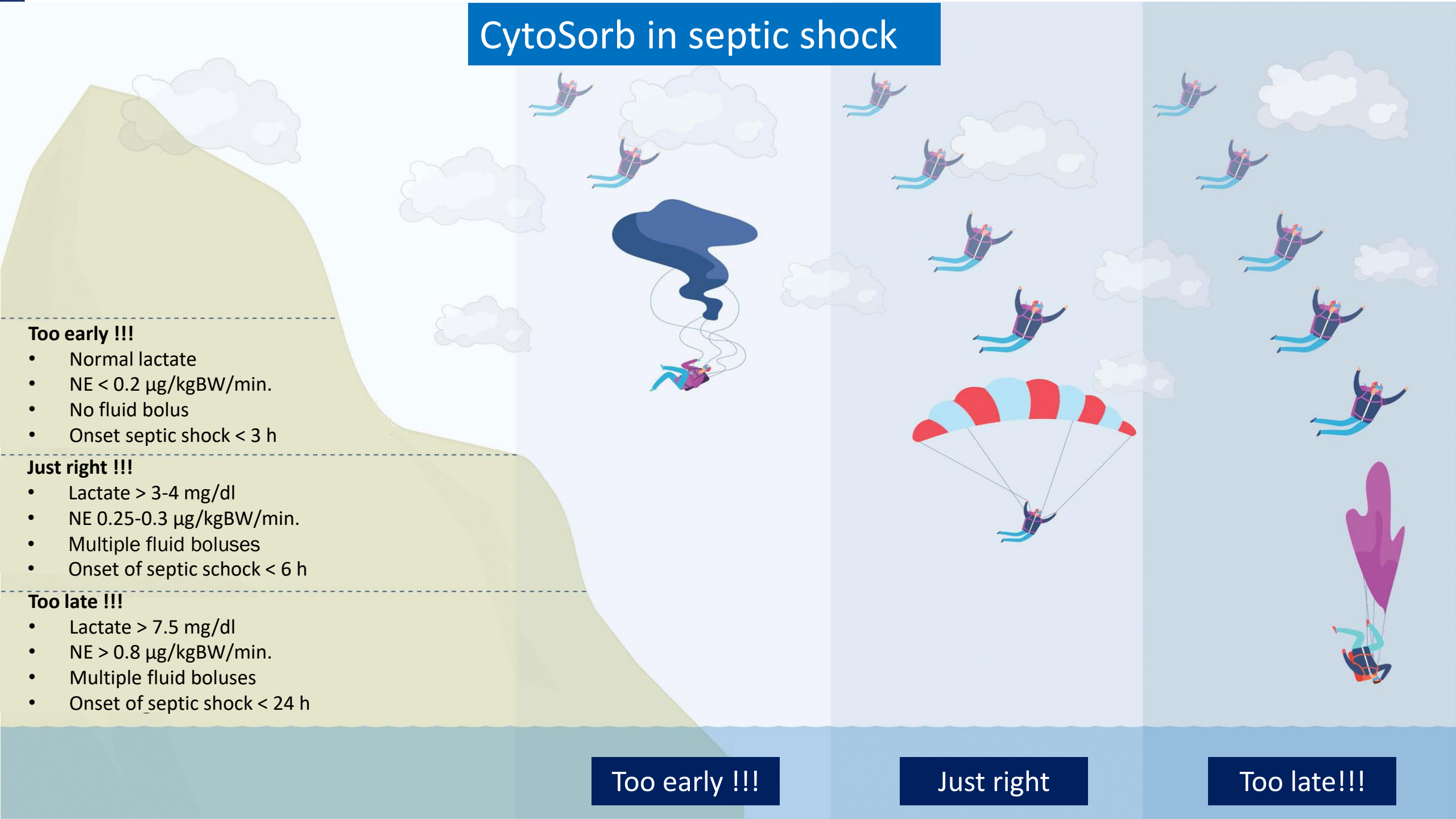
When to change???

How much blood flow ???

CytoSorb in septic shock- Arnsberg approach ?



CytoSorb in septic shock



Too early !!!
- Normal lactate
- NE < 0.2 µg/kgBW/min.
- No fluid bolus
- Onset septic shock < 3 h

Just right !!!
- Lactate > 3-4 mg/dl
- NE 0.25-0.3 µg/kgBW/min.
- Multiple fluid boluses
- Onset of septic schock < 6 h

Too late !!!
- Lactate > 7.5 mg/dl
- NE > 0.8 µg/kgBW/min.
- Multiple fluid boluses
- Onset of septic shock < 24 h

Too early !!!

Just right

Too late!!!

CytoSorb

Sepsis and beyond…

Sepsis, vasoplegia

ARDS, CS MCS

Rhabdo-myolysis

Liver failure

Case 1

6 a.m. call from external hospital (distance 140 km):

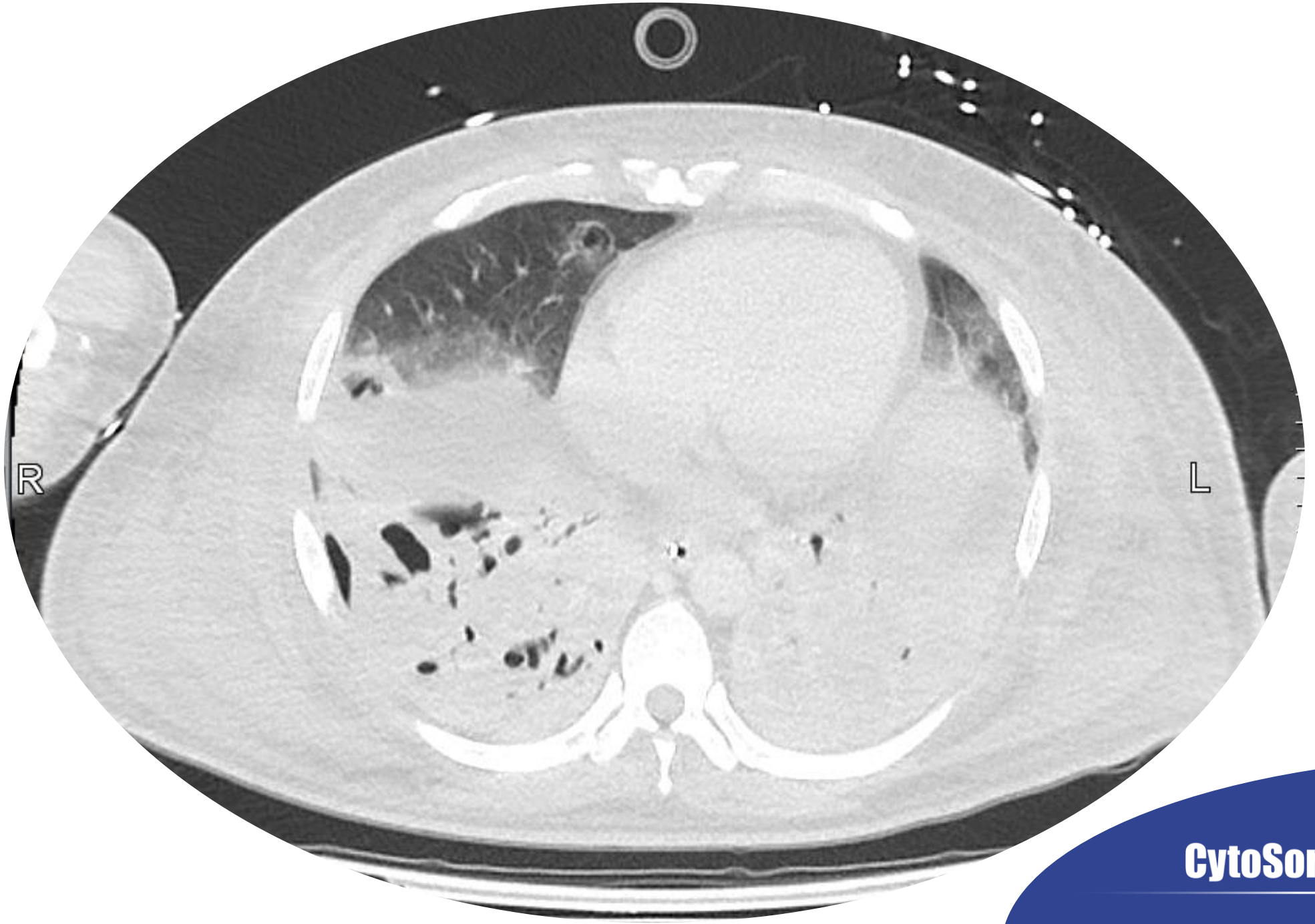
- Male patient, 35 years old, severe ARDS, pneumonia
- No comorbidities
- Current paO_2/FiO_2 45 mmHg.
- Hemodynamically unstable, NE 1.2 µg/kgBW/min, lactate 8 mg/dl.

Start at 6:10 a.m.

Estimated time with car 2h 15min

Switch to helicopter

Arrival at patient at 7:25 a.m.



CytoSorbents™

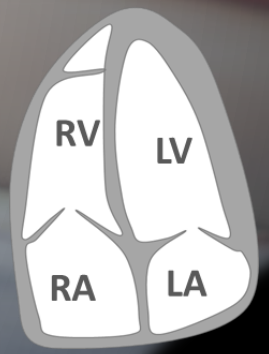
Case 1

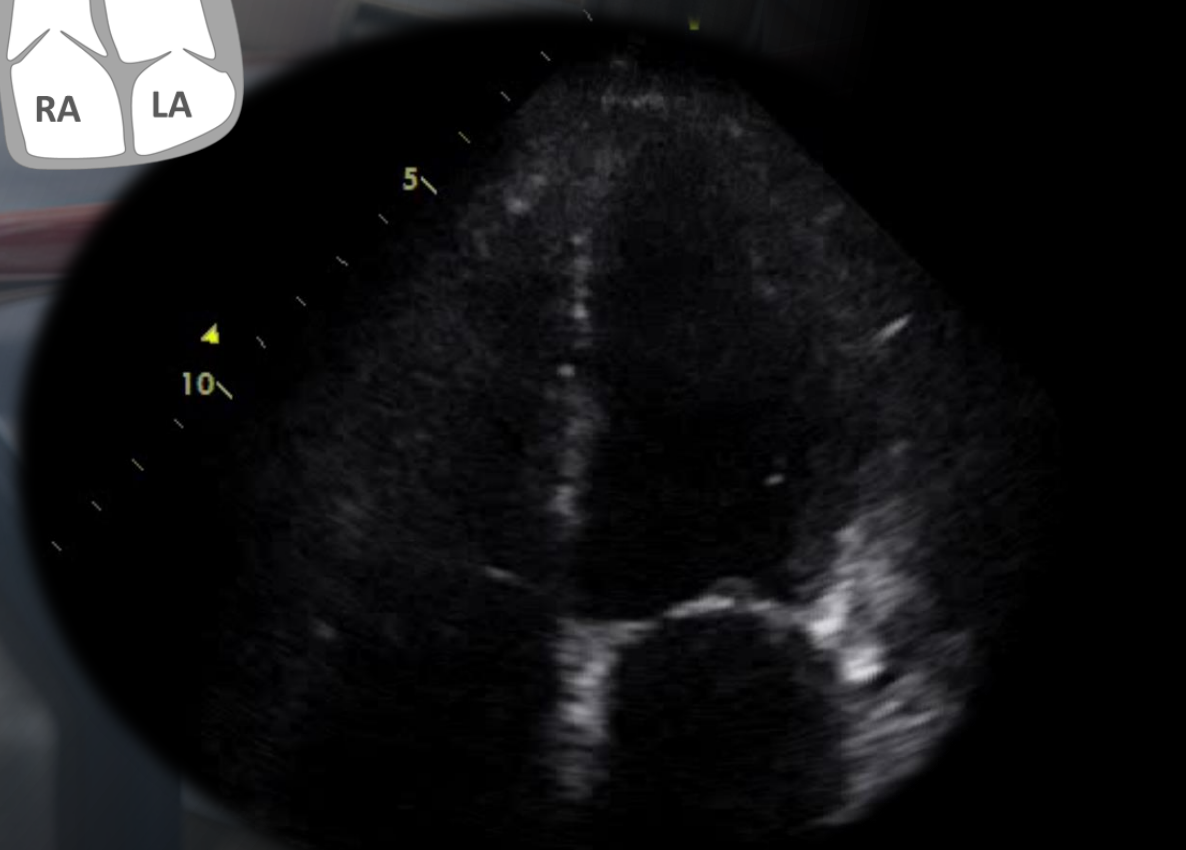
Status on arrival

MAP 55 mmHg, SR 140/min,
Lactate 12 mg/dl, pH 7.0
Mottling 3, NE 1.8 µg/kgBW/min.
Echo: severe septic cardiomyopathy,
SV 18 ml, CPO = 0.32 Watt

Pressure controlled, FiO_2 1.0, PEEP 20 mbar, P_{insp} 50 mbar, RR 24/min, VT = 9 ml/kgBW, SpO_2 33 %, paO_2 36 mmHg, pCO_2 65 mmHg

No diuresis, PCT > 100



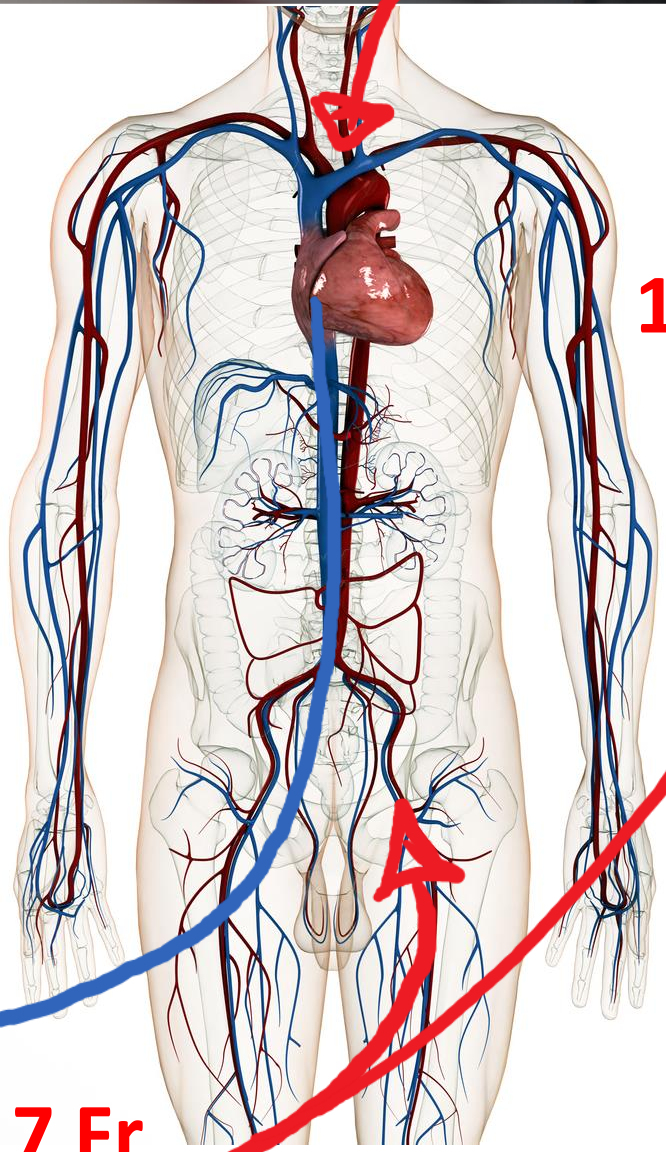


MCS therapy

Veno

Arterio

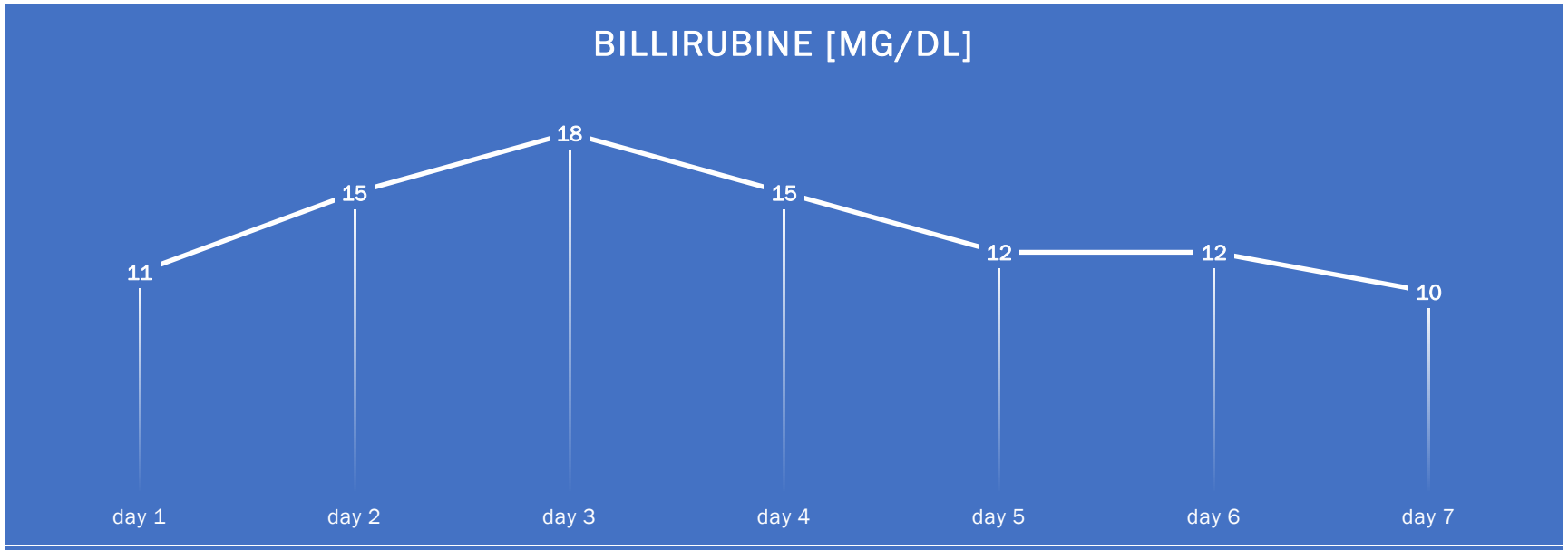
Venous

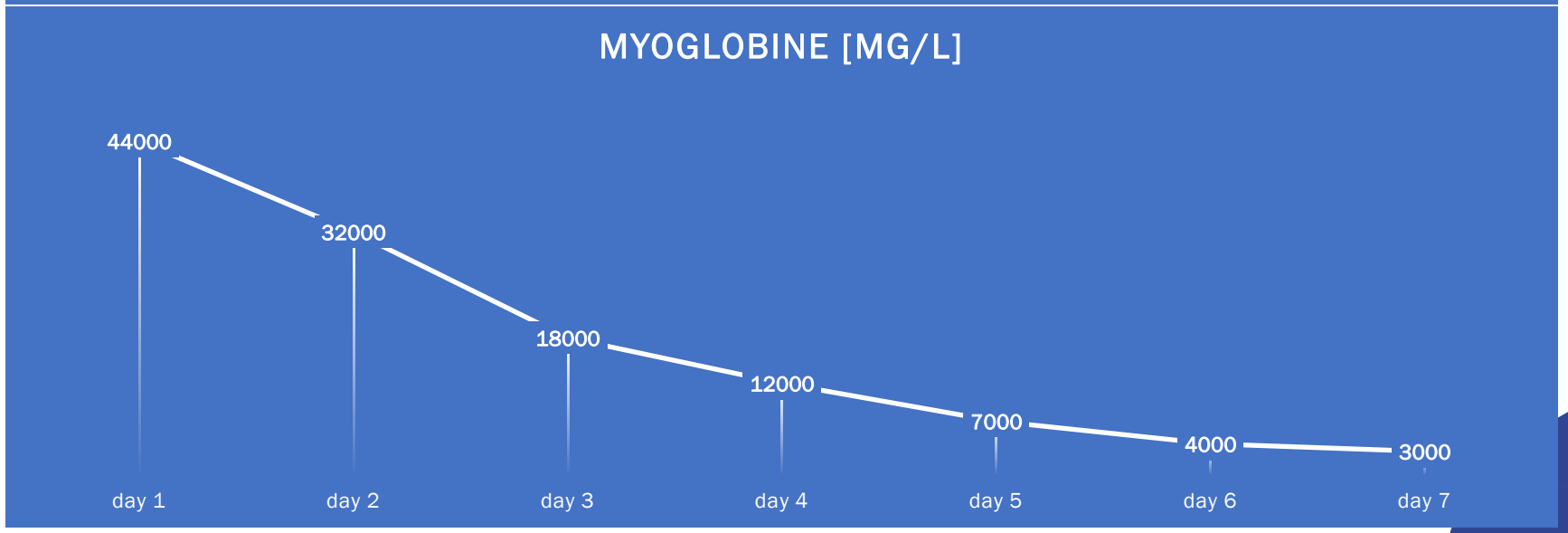


Case 1

Further course

- ✓ AKI with CRRT, liver failure, rhabdomyolsis
- ✓ Hemodynamic improvemevent within 5 days
- ✓ VA-ECMO-weaning with Levosimendan, De-escalation vv-ECMO day 6 + Impella explantation day 8
- ✓ PDT on day 9, vv-ECMO decannulation on day 18


BILLIRUBINE [MG/DL]
11
15
18
15
12
12
10
day 1
day 2
day 3
day 4
day 5
day 6
day 7


MYOGLOBINE [MG/L]
44000
32000
18000
12000
7000
4000
3000
day 1
day 2
day 3
day 4
day 5
day 6
day 7


CytoSorb