PARACELSUS MEDIZINISCHE PRIVATUNIVERSITÄT

Klinikum Nürnberg
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University of Ljubljana
Faculty of Medicine

OVERVIEW ABOUT INTRAOPERATIVE USE OF HEMOADSORPTION IN ENDOCARDITIS PATIENTS

Prof. Jurij M. Kališnik, MD, Ph.D

70th ESCVS
International congress of the European Society for Cardiovascular and Endovascular Surgery

7th IMAD meeting

INTRODUCTION – Clinical question

70-yrs male, 185 cm, 90 kg,
combined aortic valve vitium with leading insufficiency
Paroxysmal AF
Left Ventricular EF 50%
COPD
Arterial Hypertension
Diabetes Mellitus II (oral medication)
former Nicotin Abuse

Scheduled surgery:
Aortic Valve Replacement (AVR)

70-yrs male, 185 cm, 90 kg
Mitral valve insufficiency
Paroxysmal AF
Left Ventricular EF 50%
COPD
Arterial Hypertension
Diabetes Mellitus II (oral medication)
former Nicotin Abuse

Scheduled surgery:
Mitral Valve Replacement (MVR)

Increase of Mortality Risk

STS Adult Cardiac Surgery Database Version 4.20
RISK SCORES

	Elective	Complicated urgent	Emergent due to Endocarditis
AVR	1.0%	4.2%	30.1%
MVR	1.6%	6.6%	33.2%

Sepsis!

Klinikum Nürnberg

BACKGROUND

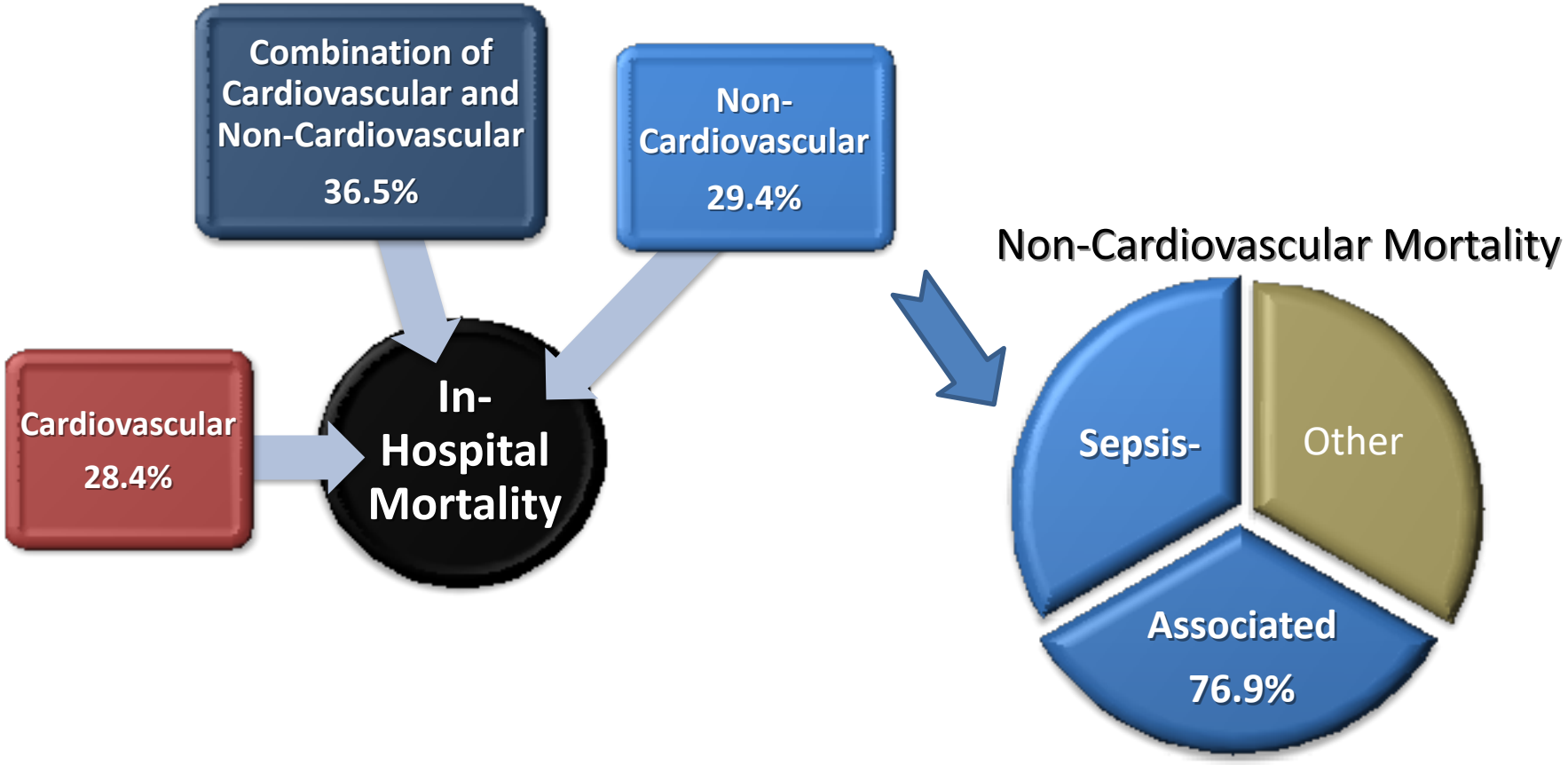
Cardiac surgery in patients with infective endocarditis (IE) is associated with high mortality even up to 60%.

Boss K et al. Extracorporeal cytokine adsorption: Significant reduction of catecholamine requirement in patients with AKI and septic shock after cardiac surgery. Plos One 2021

Postoperative septic multi-organ failure remains crucial factor of mortality, even after curative successful surgery.

Habib G et al. Clinical presentation, aetiology and outcome of infective endocarditis. Results of the ESC-EORP EURO-ENDO (European infective endocarditis) registry: a prospective cohort study. Eur Heart J 2019

Causes of In-Hospital Mortality



Habib G et al. Clinical presentation, aetiology and outcome of infective endocarditis. Results of the ESC-EORP EURO-ENDO (European infective endocarditis) registry: a prospective cohort study. Eur Heart J 2019

Infective Endocarditis and Mortality

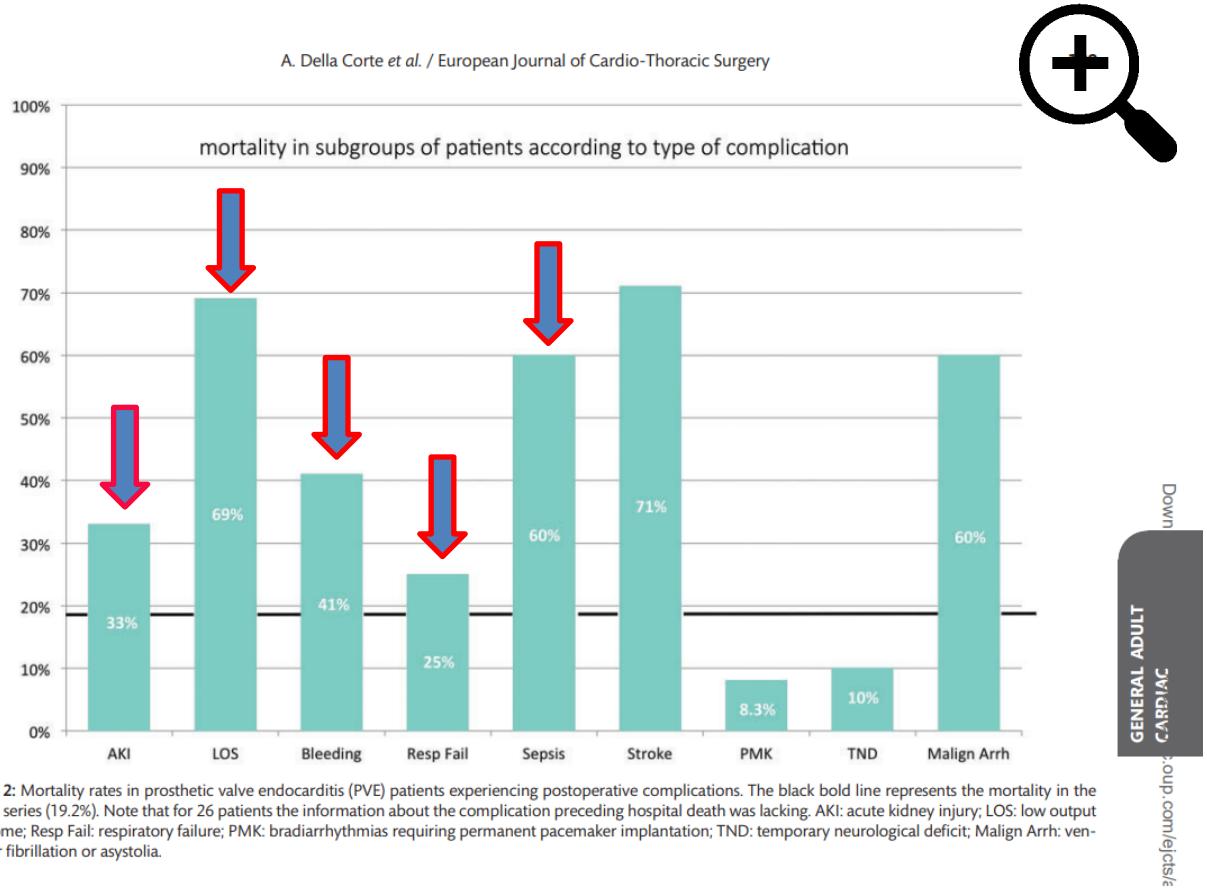
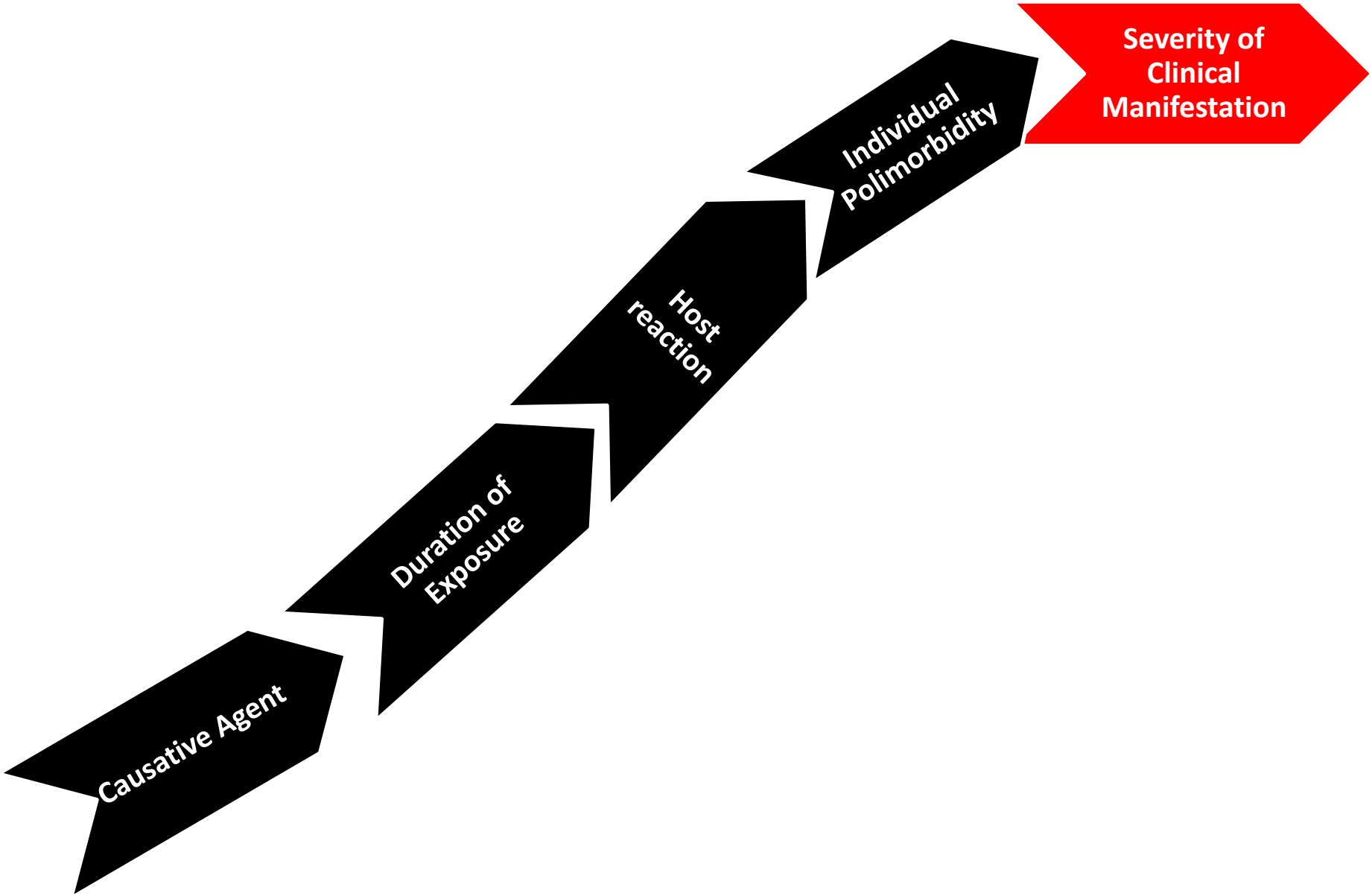


Figure 2: Mortality rates in prosthetic valve endocarditis (PVE) patients experiencing postoperative complications. The black bold line represents the mortality in the overall series (19.2%). Note that for 26 patients the information about the complication preceding hospital death was lacking. AKI: acute kidney injury; LOS: low output syndrome; Resp Fail: respiratory failure; PMK: bradiarrhythmias requiring permanent pacemaker implantation; TND: temporary neurological deficit; Malign Arrh: ventricular fibrillation or asystolia.

Della Corte A, Di Mauro M, Actis Dato G, Barili F, Cugola D, Gelsomino S *et al.* Surgery for prosthetic valve endocarditis: a retrospective study of a national registry. Eur J Cardiothorac Surg 2017;52:105–11.

Sepsis → Septic Schock Progression



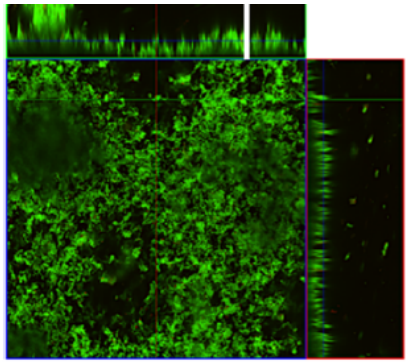
Di Domenico et al. BMC Microbiology (2019) 19:228
https://doi.org/10.1186/s12866-019-1596-2

BMC Microbiology

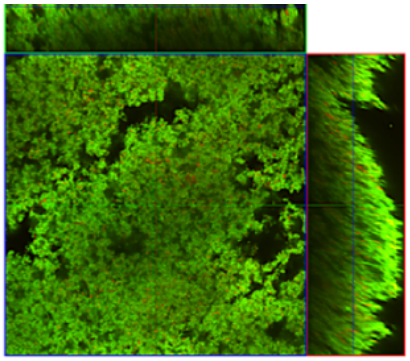
RESEARCH ARTICLE Open Access

Microbial biofilm correlates with an increased antibiotic tolerance and poor therapeutic outcome in infective endocarditis

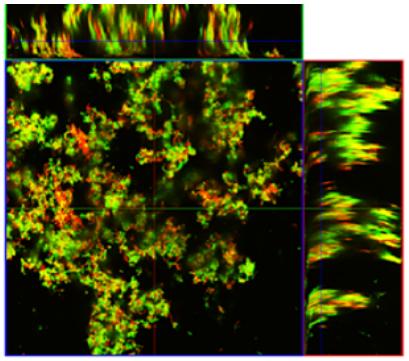
Enea Gino Di Domenico[1*], Sara Giordana Rimoldi[2], Ilaria Cavallo[1], Giovanna D'Agosto[1], Elisabetta Trento[1], Giovanni Cagnoni[3], Alessandro Palazzin[2], Cristina Pagani[2], Francesca Romeri[2], Elena De Vecchi[4], Monica Schiavini[5], Daniela Secchi[2], Carlo Antona[3], Giuliano Rizzardini[5], Rita Barbara Dichirico[2], Luigi Toma[6], Daniela Kovacs[7], Giorgia Cardinali[7], Maria Teresa Gallo[1], Maria Rita Gismondo[2] and Fabrizio Ensoli[1]



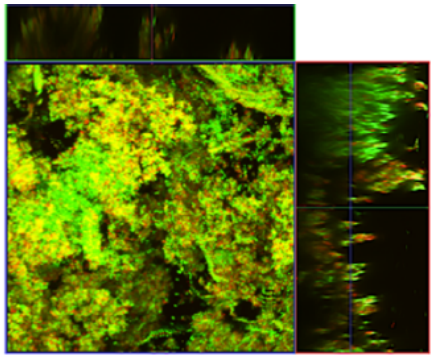
N1 - *S. aureus* (H)



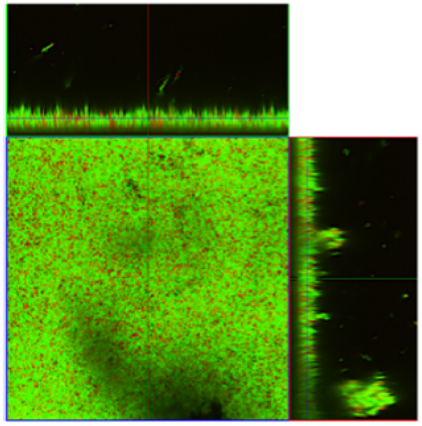
N2 - MRSA (M)

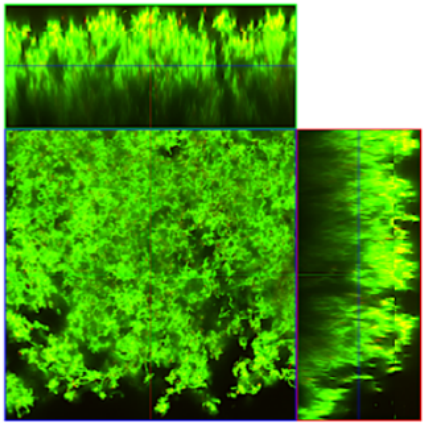


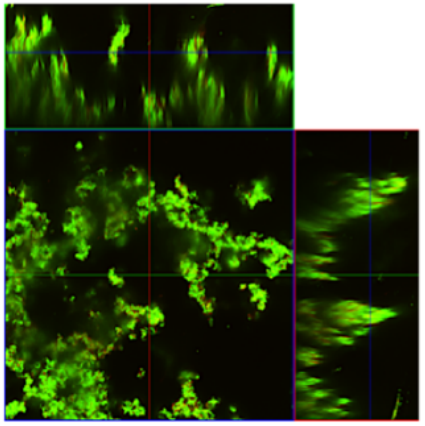
N3 - *S. gallolyticus* (M)

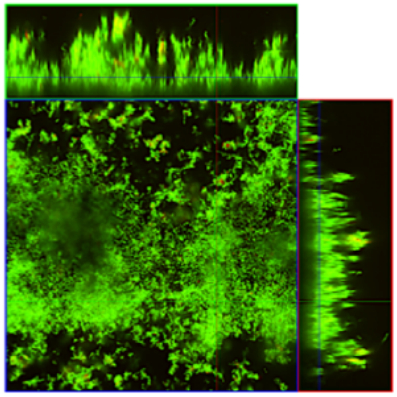


N4 - *S. aureus* (H)

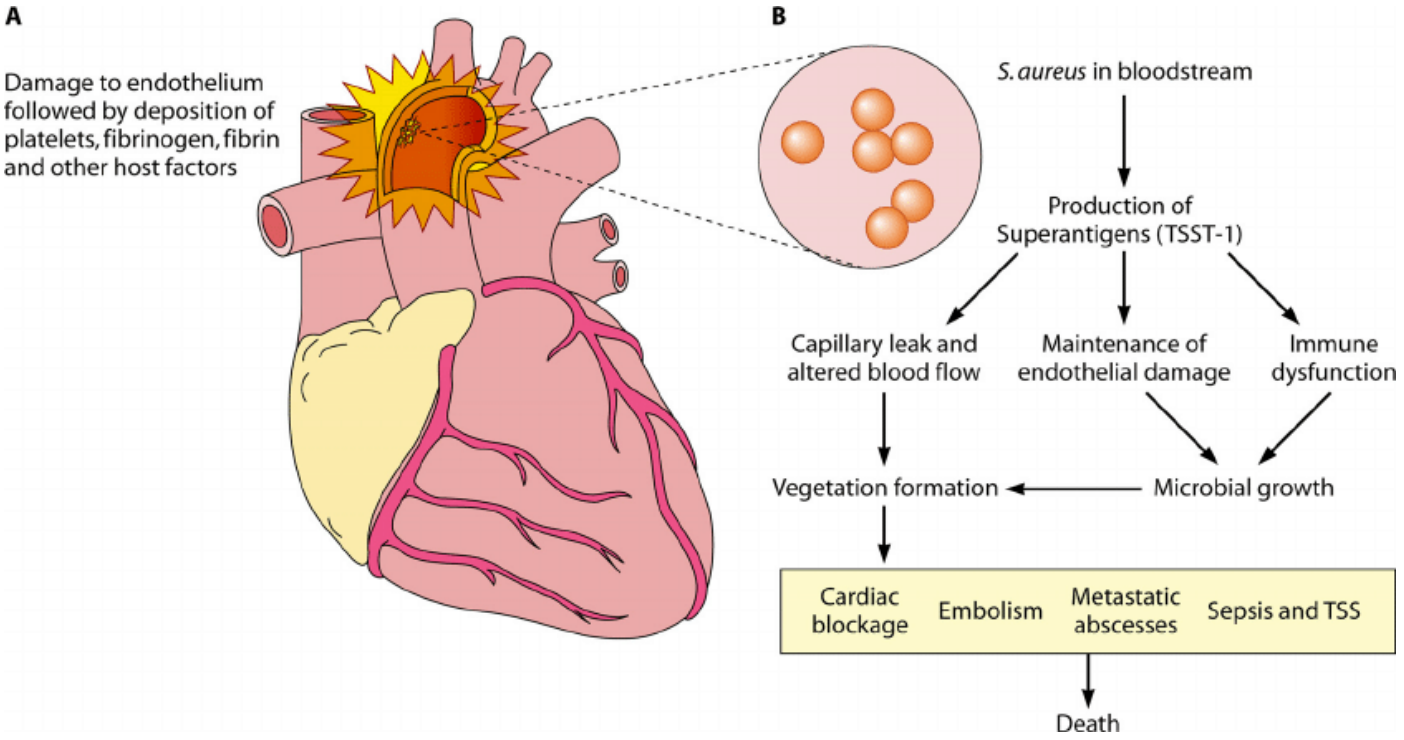








Staphylococcus aureus in bloodstream & endocarditis



S. aureus is the leading cause of IE and its mortality has remained high despite better diagnostic and therapeutic procedures over time.

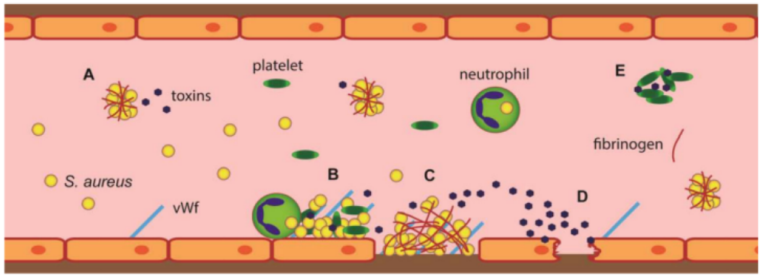


Figure 2. Virulence mechanisms of *S. aureus* inside the bloodstream.

Immune Reaction determines the mode of response in staphylococcus aureus infection

Kwiecinski and Horswill

Curr Opin Microbiol. 2020 February ; 53: 51–60. doi:10.1016/j.mib.2020.02.005.

***Staphylococcus aureus* bloodstream infections: pathogenesis and regulatory mechanisms**

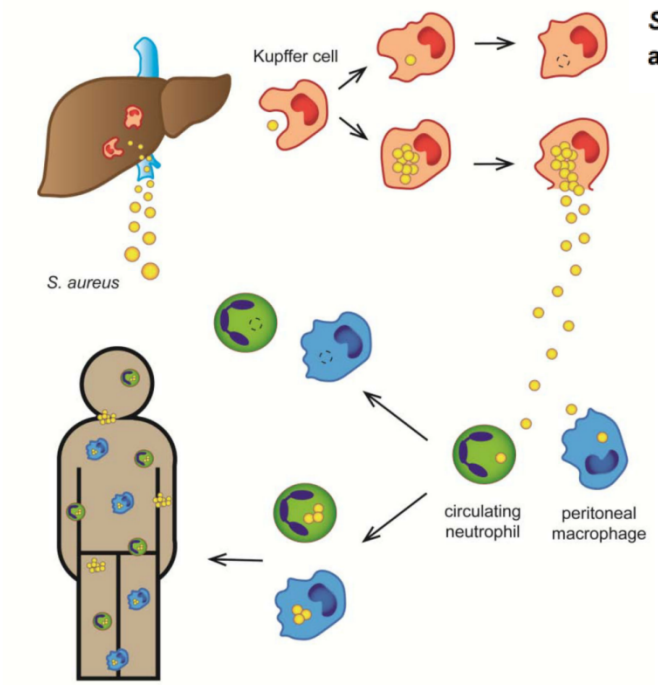


Figure 1. The role of the immune system in clearance and systemic spread of *S. aureus* in the bloodstream.

S. aureus is initially cleared from the bloodstream by Kupffer cells (liver macrophages). While Kupffer cells can kill the majority of phagocytosed bacteria, a small fraction of *S. aureus* survives and proliferates intracellularly, eventually killing the cells and being released back into the bloodstream and peritoneum. Subsequently, *S. aureus* is phagocytosed by neutrophils in liver circulation and by peritoneal macrophages. If these host cells fail to kill bacteria, they turn into “Trojan Horses”, carrying intracellular *S. aureus* throughout the body and causing a disseminated infection.

Infective Endocarditis, Sepsis and Dialysis

Kidney Int. 2019 November ; 96(5): 1083–1099. doi:10.1016/j.kint.2019.05.026.

Acute kidney injury from sepsis: current concepts, epidemiology, pathophysiology, prevention and treatment

Sadudee Peerapornratana[1,2,3,4], **Carlos L. Manrique-Caballero**[1,2], **Hernando Gómez**[1,2], **John A. Kellum**[1,2]

Dialysis is independent predictor of mortality also after surgery for IE

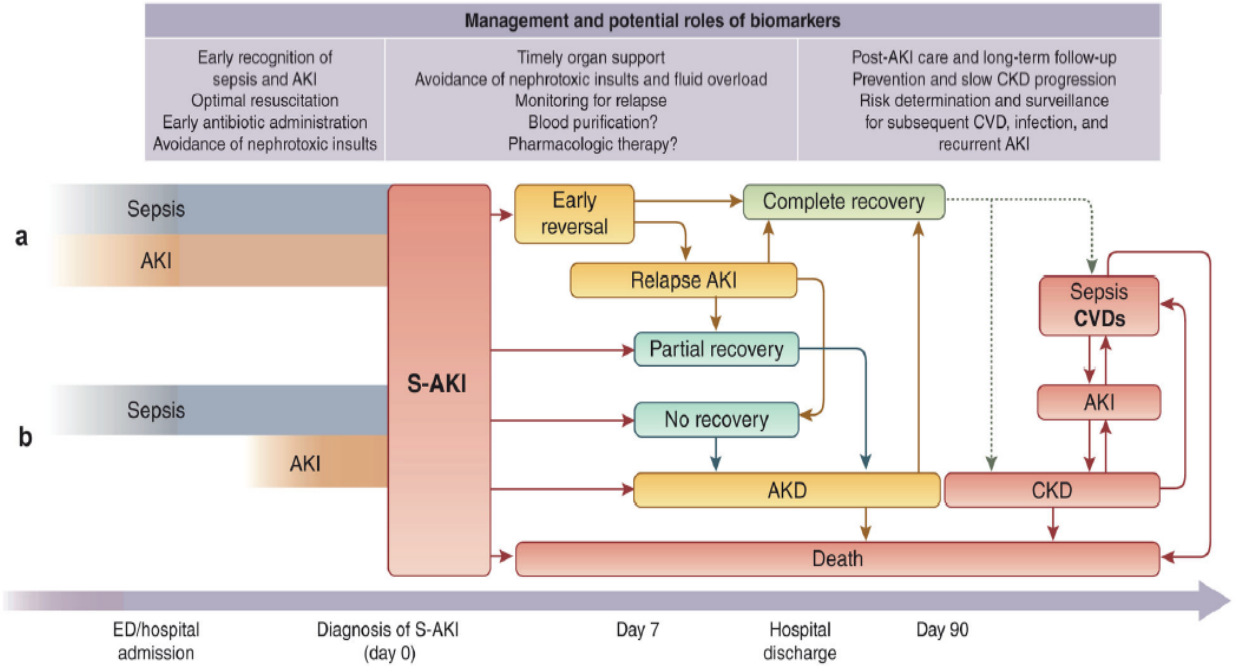
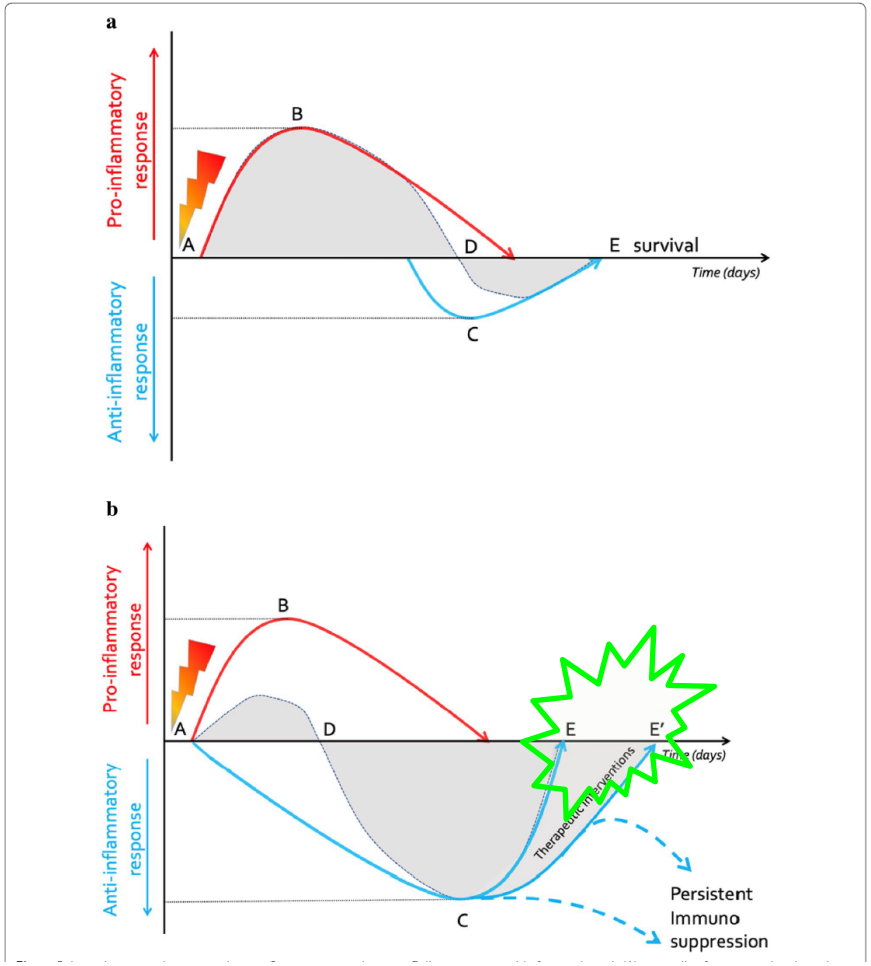


Figure 1 |. Clinical course and outcomes of sepsis-associated acute kidney injury (S-AKI).

Pro- and Anti-inflammatory Balance Resuscitation



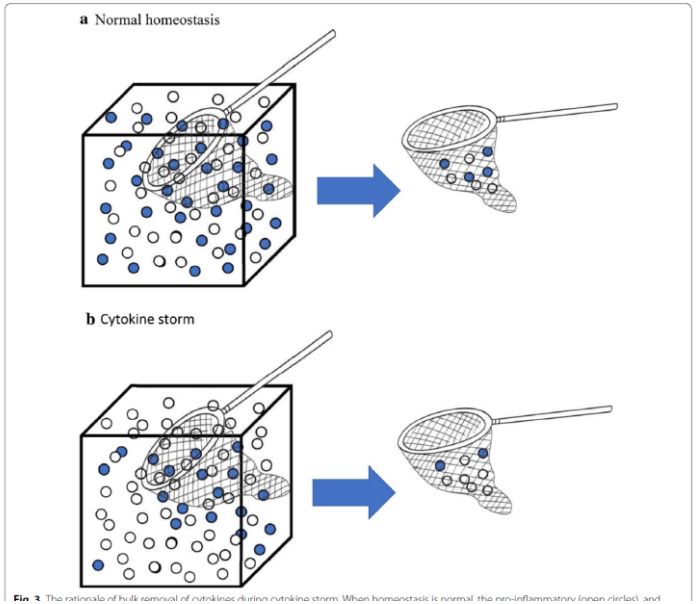
Honore et al. Ann. Intensive Care (2019) 9:56
https://doi.org/10.1186/s13613-019-0530-y

Annals of Intensive Care

REVIEW Open Access

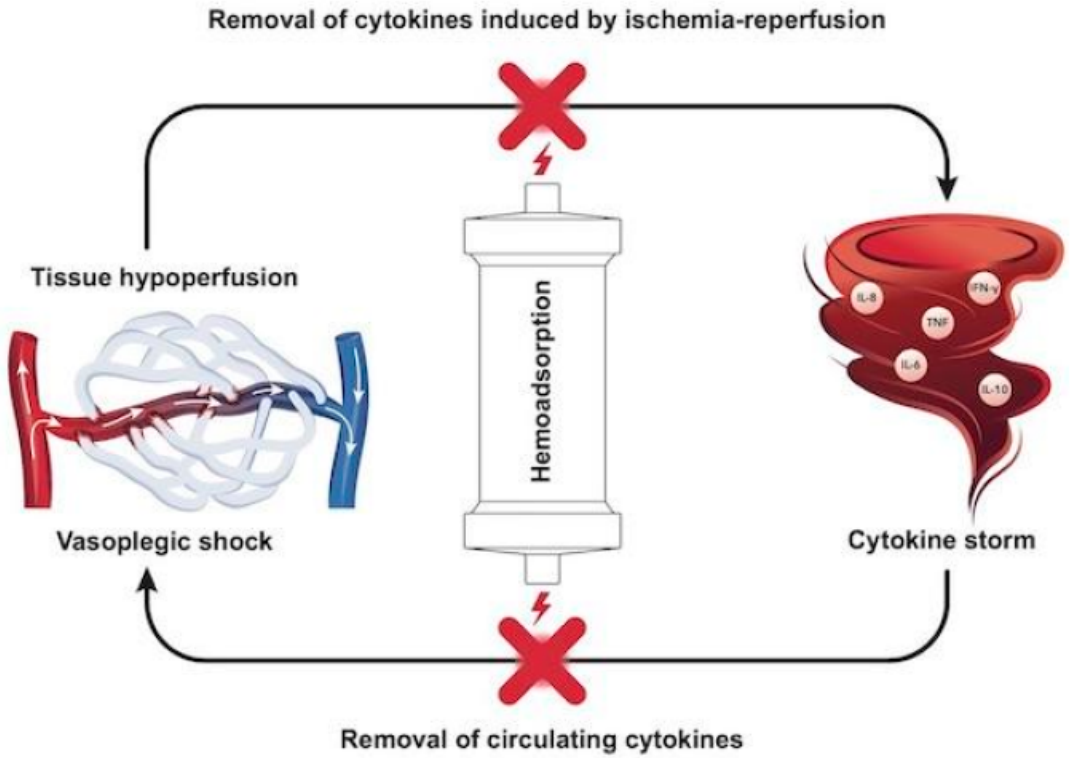
Cytokine removal in human septic shock: Where are we and where are we going?

Patrick M. Honore[1*], Eric Hoste[2], Zsolt Molnár[3], Rita Jacobs[4], Olivier Joannes-Boyau[5], Manu L. N. G. Malbrain[4,6] and Lui G. Forni[7,8]



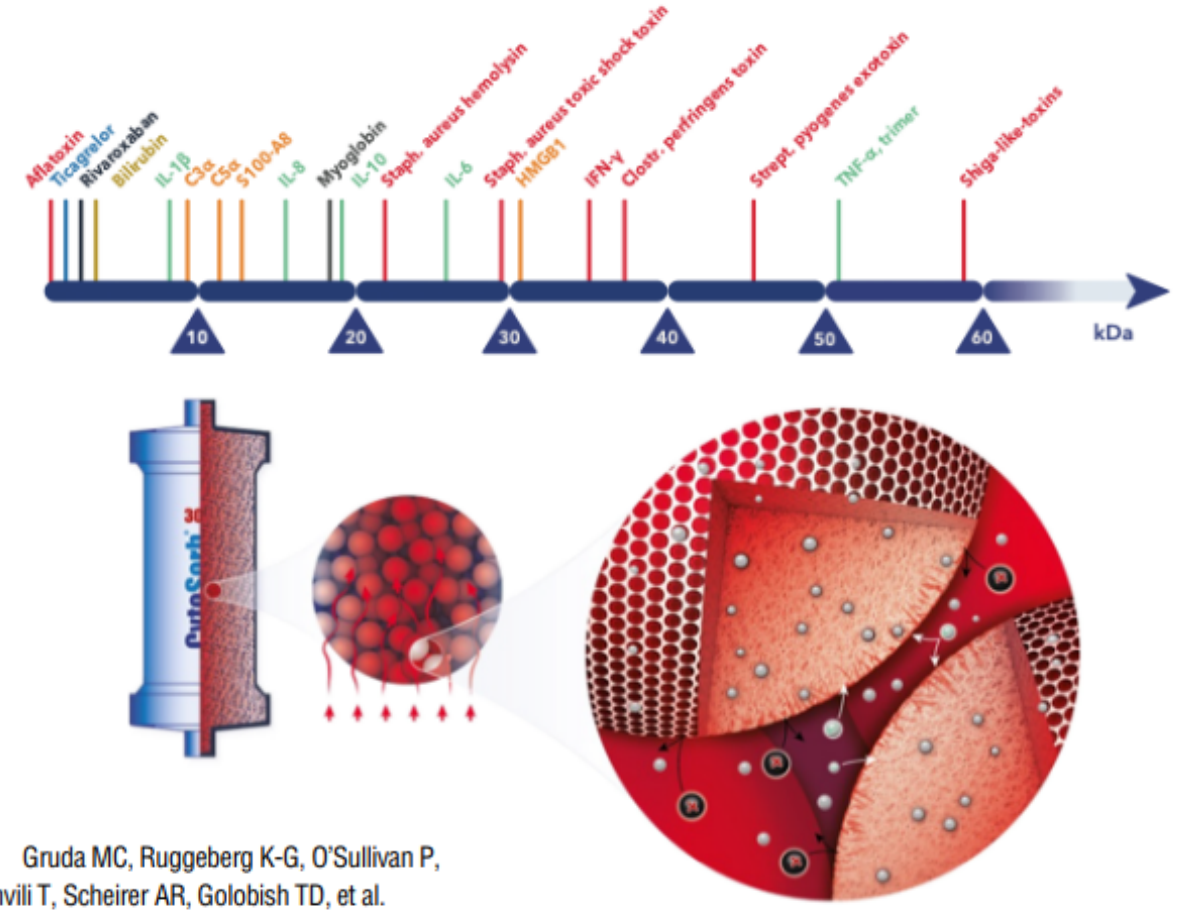
Possible Mechanism of Septic Response Alleviation

Hemoadsorption therapy interrupts vicious circle of septic shock.



Hawchar F et al. The Potential Role of Extracorporeal Cytokine Removal in Hemodynamic Stabilization in Hyperinflammatory Shock. Biomedicines 2021;9(7):768.

Substances removed during Hemoadsorption



Gruda MC, Ruggeberg K-G, O'Sullivan P, Guliashvili T, Scheirer AR, Golobish TD, et al. (2018) Broad adsorption of sepsis-related PAMP and DAMP molecules, mycotoxins, and cytokines from whole blood using CytoSorb® sorbent porous polymer beads. PLoS ONE 13(1): e0191676. https://doi.org/10.1371/journal.pone.0191676

Modes of Hyperinflammatory Host Response and Hemoadsorption

Honore *et al. Ann. Intensive Care* (2019) 9:56

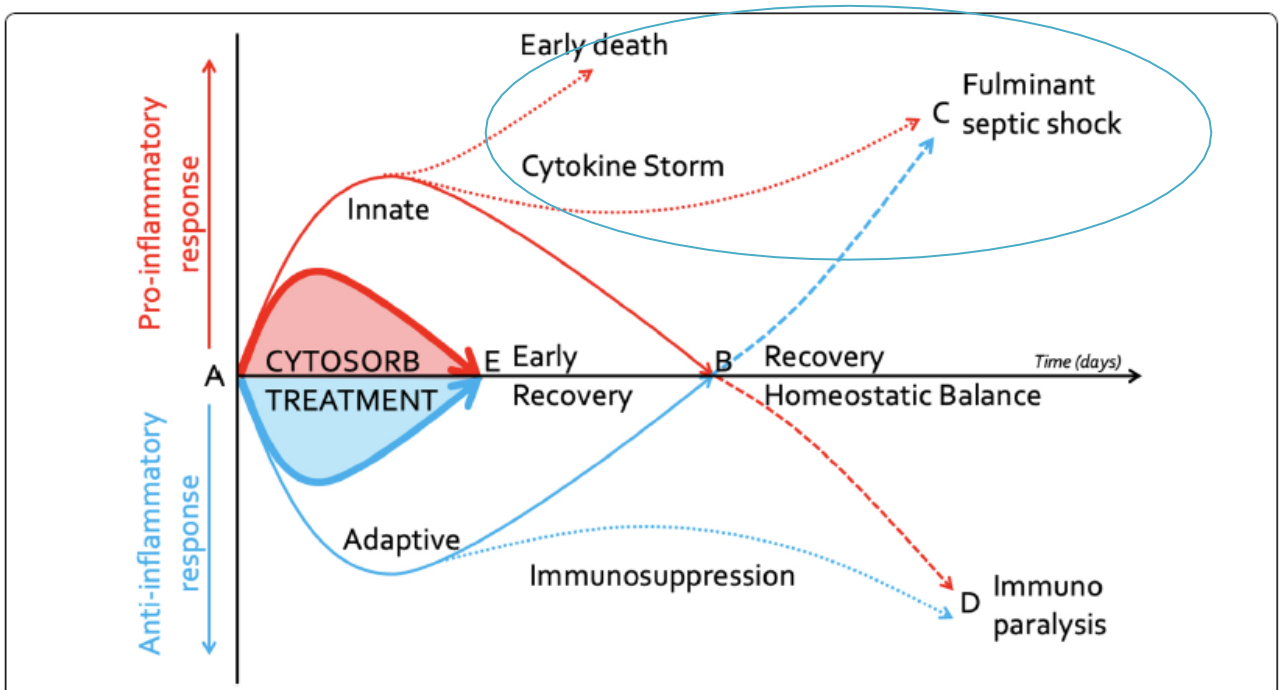
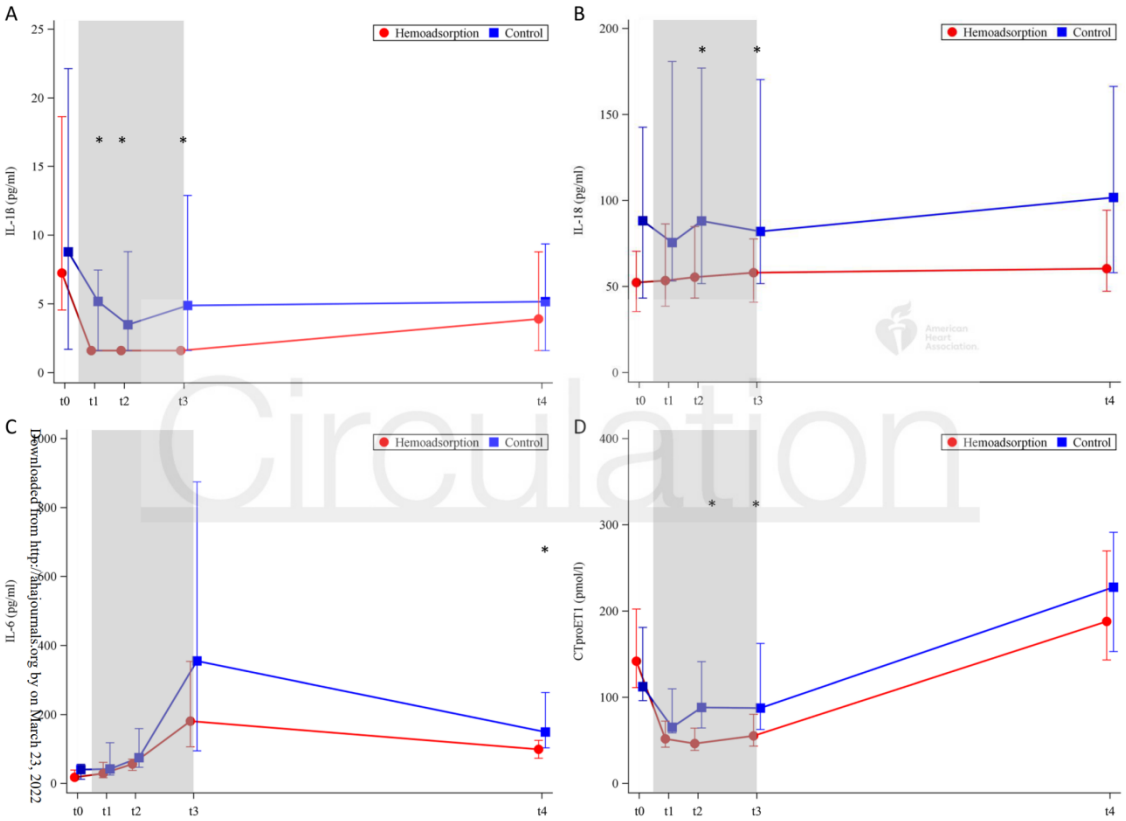


Fig. 1 Cytokine response after sepsis. Normal and abnormal immune response after an (infectious) insult (A). Recovery with regaining of the homeostatic balance occurs when pro-inflammatory (solid red line) and anti-inflammatory (solid blue line) mediators (B) return back to baseline levels. Early death or fulminant septic shock (C) can occur following early increased innate pro-inflammatory response (cytokine storm, dotted red line) or after initial adaptive immunosuppression (dashed blue line). Immunoparalysis (D) can occur following early increased adaptive anti-inflammatory response (immunosuppression, dotted blue line) or after initial pro-inflammatory response (dashed red line). Haemoadsorption with Cytosorb® may attenuate the initial pro- (bold red line) and anti-inflammatory (bold blue line) response resulting in early recovery (E)

Intraoperative Hemoadsorption reduced Hyperinflammatory Response



Cytokine Hemoadsorption During Cardiac Surgery Versus Standard Surgical Care for Infective Endocarditis (REMOVE): Results From a Multicenter Randomized Controlled Trial

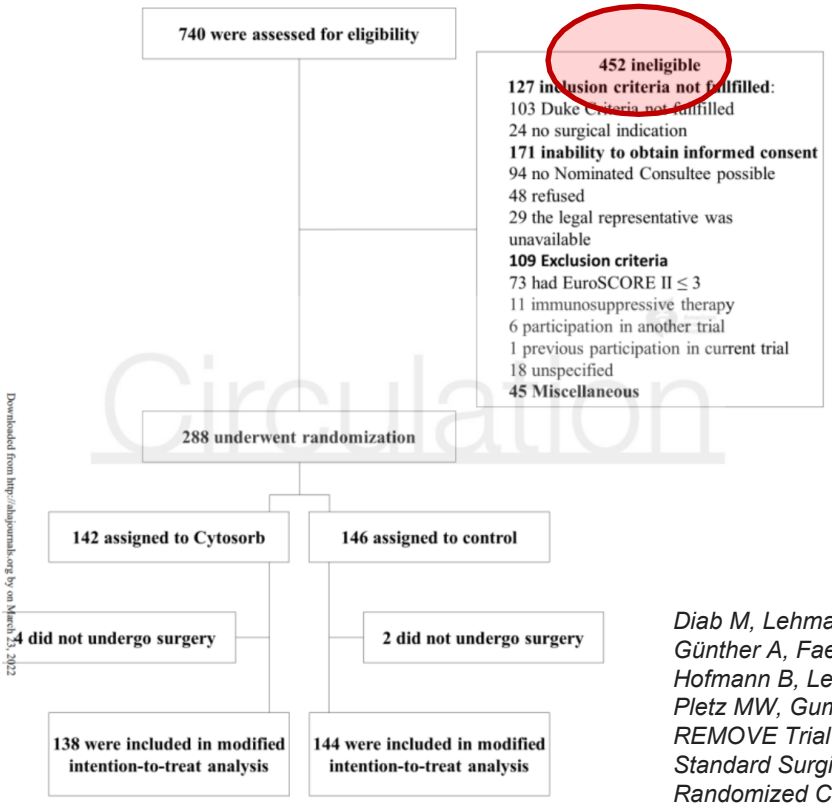
Mahmoud Diab, Thomas Lehmann, Wolfgang Bothe, Payam Akhyari, Stephanie Platzer, Daniel Wendt, Antje-Christin Deppe, Justus Strauch, Stefan Hagel, Albrecht Günther, Gloria Faerber, Christoph Sponholz, Marcus Franz, André Scherag, Ilia Velichkov, Miriam Silaschi, Jens Fassl, Britt Hofmann, Sven Lehmann, Rene Schramm, Georg Fritz, Gabor Szabo, Thorsten Wahlers, Klaus Matschke, Artur Lichtenberg, Mathias W. Pletz, Jan F. Gummert, Friedhelm Beyersdorf, Christian Hagl, Michael A. Borger, Michael Bauer, Frank M. Brunkhorst and Torsten Doenst See fewer authors
and on behalf of the REMOVE Trial Investigators*

Originally published 25 Feb 2022 | https://doi.org/10.1161/CIRCULATIONAHA.121.056940 | Circulation. 2022;145:959–968

No detectable clinical benefit after Hemoadsorption

Conclusions:

This randomized trial failed to demonstrate a reduction in postoperative organ dysfunction through intraoperative hemoadsorption in patients undergoing cardiac surgery for IE. Although hemoadsorption reduced plasma cytokines at the end of cardiopulmonary bypass, there was no difference in any of the clinically relevant outcome measures.



Diab M, Lehmann T, Bothe W, Akhyari P, Platzer S, Wendt D, Deppe AC, Strauch J, Hagel S, Günther A, Faerber G, Sponholz C, Franz M, Scherag A, Velichkov I, Silaschi M, Fassl J, Hofmann B, Lehmann S, Schramm R, Fritz G, Szabo G, Wahlers T, Matschke K, Lichtenberg A, Pletz MW, Gummert JF, Beyersdorf F, Hagl C, Borger MA, Bauer M, Brunkhorst FM, Doenst T; REMOVE Trial Investigators. Cytokine Hemoadsorption During Cardiac Surgery Versus Standard Surgical Care for Infective Endocarditis (REMOVE): Results From a Multicenter Randomized Controlled Trial. Circulation. 2022 Mar 29;145(13):959-968.*

Comparable rates of postoperative complications in REMOVE trial

10.1161/CIRCULATIONAHA.121.056940

Table 3: Adverse events

	Hemoadsorption group (n=138)	Control group (n=144)	P
Pericardial tamponade	6 (4.3)	6 (4.2)	1.00
Bleeding anemia	23 (16.7)	26 (18.1)	0.88
Coagulopathy	15 (10.9)	15 (10.4)	1.00
Thrombocytopenia	22 (15.9)	20 (14.1)	0.74
Re-exploration	23 (17.0)	18 (12.5)	0.40
Low cardiac output syndrome	10 (7.2)	11 (7.6)	1.00
Left ventricular failure	7 (5.1)	9 (6.3)	0.80
Cardiac arrest	1 (0.7)	2 (1.4)	1.00
Distributive shock	94 (68.1)	102 (70.8)	0.70
Atrioventricular block	13 (9.4)	18 (12.5)	0.45
Pneumonia	8 (5.8)	7 (4.9)	0.80
Delirium	10 (7.2)	18 (12.5)	0.17
Small cerebral infarctions	4 (3)	0	0.06
Acute kidney injury	78 (57)	80 (55.6)	0.91
Respiratory insufficiency	47 (34.1)	58 (40.8)	0.27

Data are presented as n (%)

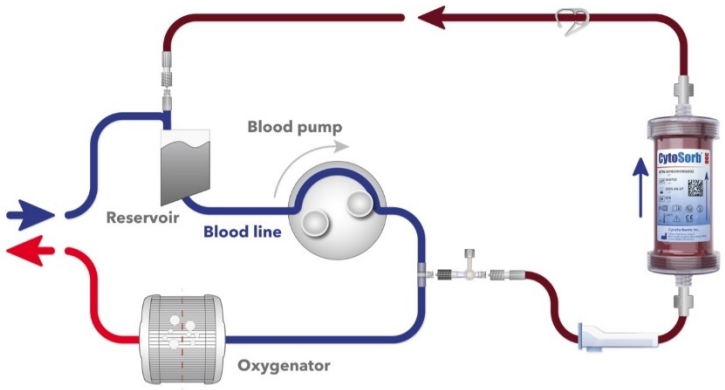
Diab M, Lehmann T, Bothe W, Akhyari P, Platzer S, Wendt D, Deppe AC, Strauch J, Hagel S, Günther A, Faerber G, Sponholz C, Franz M, Scherag A, Velichkov I, Silaschi M, Fassl J, Hofmann B, Lehmann S, Schramm R, Fritz G, Szabo G, Wahlers T, Matschke K, Lichtenberg A, Pletz MW, Gummert JF, Beyersdorf F, Hagl C, Borger MA, Bauer M, Brunkhorst FM, Doenst T; REMOVE Trial Investigators. Cytokine Hemoadsorption During Cardiac Surgery Versus Standard Surgical Care for Infective Endocarditis (REMOVE): Results From a Multicenter Randomized Controlled Trial. Circulation. 2022 Mar 29;145(13):959-968.*

Nuremberg Infective Endocarditis Cohort

Retrospective analysis of 202 patients with left-sided infective endocarditis (IE) and urgent indication for surgery was performed:

135 patients with native valve IE

67 patients with prosthetic valve IE



103 patients without intraoperative hemoadsorption
operated between January 2015 and December 2018

99 patients with intraoperative hemoadsorption during cardiopulmonary bypass
operated between January 2018 and May 2021

Preoperative Patient Demographics

Preoperative characteristics of Patients				
		Control (n=103)	Hemoadsorption (n=99)	*P*-value
Demographics				
Age (years)		69 [58;77]	67 [58;75]	0.612
BMI (kg/m²)		26.4 [23.8;30.8]	26.8 [24.0;30.6]	0.891
Gender (% male)		83 (80.6%)	81 (81.8%)	0.964
EuroScore II (%)		9 [3.6;22.2]	9.9 [5.5;21.8]	0.805
Native valve endocarditis		76 (37.6%)	49 (24.1%)	0.106
Prosthetic valve endocarditis		27 (13.3%)	40 (19.9%)	0.169
Coronary artery disease		28 (27.5%)	28 (28.3%)	1.000
Dilated ascending aorta		5 (4.9%)	9 (9.1%)	0.407
Previous surgery of one valve		34 (33%)	40 (40.4%)	0.345
Previous multiple valve surgery		2 (1.9%)	5 (5.1%)	0.272
Previous aortic root surgery		3 (2.9%)	5 (5.1%)	0.492
Previous bypass surgery		8 (7.8%)	7 (7.1%)	1.000
Arterial hypertension		75 (72.8%)	76 (76.8%)	0.628
Diabetes Mellitus Type II		25 (24.3%)	23 (23.2%)	0.993
Peripheral Arterial Occlusive Disease		6 (5.8%)	3 (3%)	0.499
Hypercholesterinemia		56 (54.9%)	56 (56.6%)	0.924

		Control (n=103)	Hemoadsorption (n=99)	*P*-value
History of Cerebrovascular Insult		19 (18.4%)	32 (32.3%)	**0.035**
Septic embolisms (last 3 weeks)		19 (18.4%)	21 (21.2%)	0.752
Chronic Obstructive Lung Disease		20 (19.4%)	14 (14.1%)	0.416
Liver cirrhosis		4 (3.9%)	6 (6.1%)	0.533
Preoperative Atrial Fibrillation		20 (19.4%)	27 (27.3%)	0.248
$eGFR_{CKD\text{-}EPI}$ (mL/min)		62 [38;79]	52 [40;74]	0.380
Preoperative Dialysis		10 (9.7%)	8 (8.1%)	0.874
Antiplatelet therapy		32 (31.1%)	22 (23.2%)	0.276
Oral anticoagulant therapy		13 (12.7%)	10 (10.4%)	0.772

Preoperative Patient Characteristics

Clinical status	Control (n=103)	Hemoadsorption (n=99)	*P*-value
NYHA (III-IV)	73 (70.9%)	78 (78.8%)	0.257
Preoperative inotropes	36 (35.0%)	40 (40.8%)	0.477
Septic shock (within 48h)	17 (16.5%)	19 (19.2%)	0.753
Preoperatively intubated	12 (11.7%)	9 (9.2%)	0.733
Mean arterial pressure (mmHg)	86 [77;100]	87 [80;98.5]	0.760
Laboratory Parameters			
CRP preop (mg/dL)	6 [3;10.9]	5 [2;10.3]	0.166
Preoperative Platelets (x 10^3/µL)	236 [160;307]	238 [171;300]	0.920
Preoperative Leucocytes (x 10^3/µL)	9.7 [7.3;14.1]	8.5 [6.2;12.5]	*0.077*
Preoperative Hemoglobin (ng/mL)	10.2 [9.1;11.4]	10.1 [9.1;11.5]	0.836
c-TnT (ng/mL)	0.06 [0.02;0.17]	0.04 [0.02;0.1]	0.159
CK-MB (ng/mL)	1.36 [1;1.83]	1.44 [1;2.12]	0.673
Lactate (start of surgery; mmol/l)	0.8 [0.6;1.1]	0.7 [0.5;1]	0.183

Echocardiographic/Radiologic Characteristics	Control (n=103)	Hemoadsorption (n=99)	*P*-value
LVEF lower than 50%	29 (28.2%)	23 (23.2%)	0.523
Vegetations	98 (95.1%)	94 (94.9%)	1.000
Paravalvular extension or Abscess	37 (35.9%)	44 (44.4%)	0.275
Concomitant right-sided endocarditis	5 (4.9%)	2 (2.1%)	0.446
Causative infective agent			
Staphylococcus species	40 (38.8%)	31 (32%)	0.385
Staphylococcus aureus	26(26%)	19 (21.3%)	0.563
Streptococcus species	29 (29%)	18 (20.2%)	0.221
Enterococcus faecalis	13 (13%)	15 (16.9%)	0.590
Gram- bacteria	5 (4.9%)	5 (5.1%)	1.000
Preoperative Targeted Antibiotic (d)	6 [2;11]	5 [3;10.8]	0.702

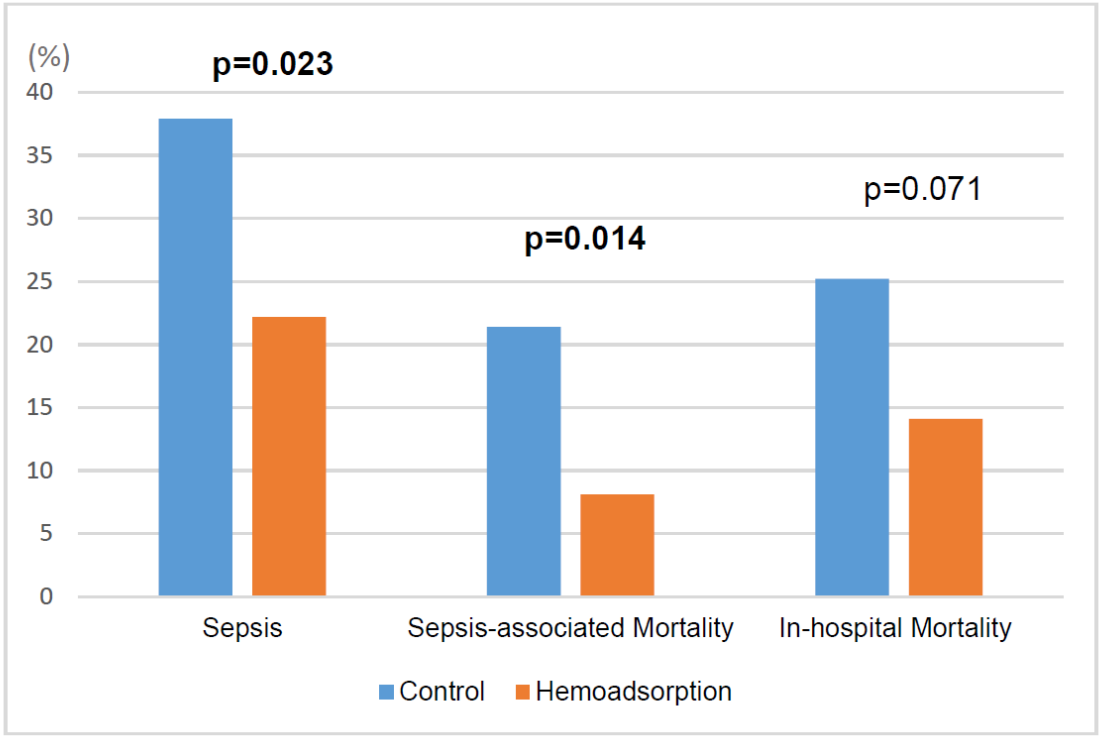
Journal of Clinical Medicine

MDPI

Article

Intraoperative hemoadsorption in left-sided infective endocarditis

Jurij Matija Kalisnik[1,2,*], Spela Leiler[1], Hazem Mamdooh[1], Janez Zibert[3], Thomas Bertsch[4], Ferdinand Aurel Vogt[5,6], Erik Bagaev[1], Matthias Fittkau[1], Theodor Fischlein,[1,6]



Complications

	Control (n=103)	Hemoadsorption (n=99)	*P*-value
Re-thoracotomy for bleeding	17 (16.7%)	10 (10.2%)	0.258
Intra-Aortic Balloon Pump	4 (3.9%)	4 (4.0%)	*1.000*
Extracorporeal Life Support	1 (1.0%)	4 (4.0%)	*0.205*
Mechanical ventilation (h)	22 [8;68.2]	19.0 [10;65.5]	0.856
Chest tube output (24h; ml)	500 [300;700]	500 [250;950]	0.761
Postoperative Atrial Fibrillation	17 (16.5%)	13 (13.1%)	0.634
Postoperative Acute Kidney Injury	54 (52.4%)	59 (59.6%)	0.377
New Dialysis	25 (24.3%)	19 (19.2%)	0.481
Central Neurological Complications	2 (1.9%)	8 (8.1%)	*0.055*
Pneumonia	10 (9.7%)	6 (6.1%)	0.484
Deep Sternal Wound Infection	1 (1.0%)	0	1.000
Urinary Tract infection	2 (1.9%)	1 (1.0%)	1.000
New Pacemaker	12 (11.7%)	13 (13.1%)	0.916

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Laboratory-associated Parameters after Surgery

	Control (n=103)	Hemoadsorption (n=99)	*P*-value
Lactate End Surgery (mmol/L)	1.3 [1;1.7]	1.2 [0.9;1.8]	0.656
Peak c-TnT (ng/mL)	0.56 [0.28;1.03]	0.65 [0.4;1.07]	0.178
Peak CK-MB (ng/mL)	19.7 [12.4;32.2]	23 [13.6;36.2]	0.214
Cumulative Inotropes POD1 (mg)	12.6 [4.9;40.1]	17.8 [7.8;37.2]	0.204
Lactate POD1 (mmol/L)	1.2 [0.9;1.6]	1.3 [0.9;1.8]	0.191
Hemoglobin POD 1 (g/dL)	9.2 [8.4;9.9]	9.7 [8.7;10.3]	**0.011**
Platelets POD1 (x 10^3/µL)	172 [127;235]	151 [114;204]	**0.032**
Leucocytes POD1 (10^3/µL)	11.4 [9.1;17.1]	10.4 [8.1;15.6]	*0.065*
CRP POD 1 (mg/dL)	10 [7.2;15.4]	8.8 [5;12.9]	**0.026**

	Control (n=103)	Hemoadsorption (n=99)	*P*-value
Cumulative Inotropes POD2 (mg)	5.8 [0.9;15.2]	4.9 [1.1;16.9]	0.927
Lactate POD2 (mmol/L)	1 [0.7;1.2]	1 [0.8;1.3]	0.142
Hemoglobin POD2 (g/dL)	8.8 [8.1;9.3]	8.9 [8.4;9.6]	0.166
Platelets POD2 (x10^3/µL)	168 [123;228]	136 [106;201]	*0.069*
Leucocytes POD2 (x10^3/µL)	12.1 [8.8;15.1]	9.9 [7.40;14.2]	**0.025**
CRP POD2 (mg/L)	15.4 [11.5;21.8]	16.2 [11;20.6]	0.824
RBC transfused (Units)	3 [1;6]	1 [0;4]	**0.016**
RBC transfused (frequency)	79 (76.7%)	59 (59.6%)	**0.014**
FFP transfused (Units)	0 [0;4]	1 [0;6]	**0.014**
FFP transfused (frequency)	30 (29.1%)	45 (45.5%)	**0.024**
Platelets transfused (Units)	0 [0;2]	0 [0;1]	0.719
Platelets transfused (frequency)	38 (36.9%)	35 (35.4%)	0.935

Parameters of Sepsis-associated and In-hospital Mortality

Variables related to sepsis-associated mortality* and in-hospital mortality**

	Univariate Analysis		*Multivariate analysis*	
Variable	OR [CI_L, CL_U]	P-value	OR [CI_L, CL_U]	P-value
CRP Day 1 postoperatively	1.09 [1.03,1.16] *	0.005	1.15 [1.02,1.299]	**0.023**
Leucocytes Day 2 postoperatively	1.26 [1.16,1.37] *	< 0.001	1.23 [1.08,1.4]	**0.002**
Abscess	2.21 [1.11,4.84] *	0.048	4.65 [0.81,26.91]	*0.086*
Chest tube output	1.001 [1,1.001] **	0.003	1.01 [1,1.01]	**0.04**
Lactate End of surgery	2.66 [1.78,3.99] **	< 0.001	1.96 [0.914,4.21]	**0.049**
Preoperative inotropes	6.4 [2.95,13.85] **	< 0.001	5.57 [1.3,23.83]	**0.010**
Inotropes Day 1 Postoperatively	1.051 [1.034,1.07] **	< 0.001	1.023 [1, 1.047]	*0.055*
Leucocytes Day 1 Postop	1.18 [1.1,1.25] **	< 0.001	1.18 [1.13,1.42]	*0.078*

	Univariate Analysis		*Multivariate Analysis*	
Variable	OR [CI_L, CL_U]	P-value	OR [CI_L, CL_U]	P-value
Hemoadsorption therapy	0.32 [0.14,0.77] *	0.01	0.11 [0.02,0.68]	**0.018**
	0.48 [0.238,1.001] **	0.049	0.07 [0.01,0.76]	*0.064*
Inotropes Day 2 postoperatively	1.01 [1,1.01] *	< 0.001	1.01 [1,1.01]	**0.015**
	1.049 [1.03,1.7] **	< 0.001	1.03 [1,1.05]	**0.020**
Need for postoperative Dialysis	7.02 [3.06,16.12] *	< 0.001	7.43 [1.4,39.44]	**0.019**
	6.68 [3.12,14.3] **	< 0.001	10.46 [1.87,58.37]	**0.022**

Multivariate analysis was performed for $p < 0.05$

* for Sepsis-associated mortality

** for In-hospital mortality

Relevant decrease of SOFA Score after intraoperative Hemoadsorption

Intraoperative hemoadsorption in high-risk patients with infective endocarditis

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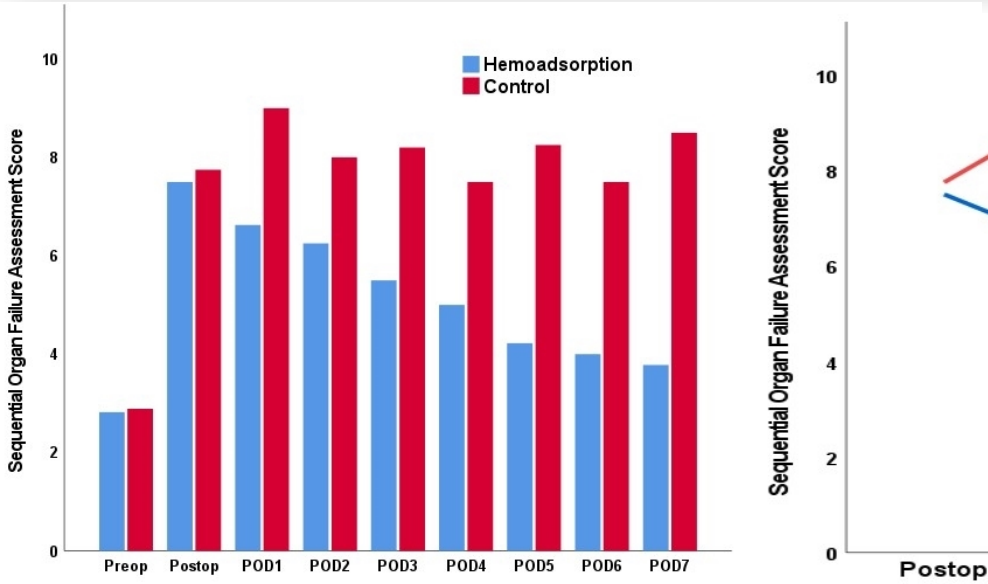
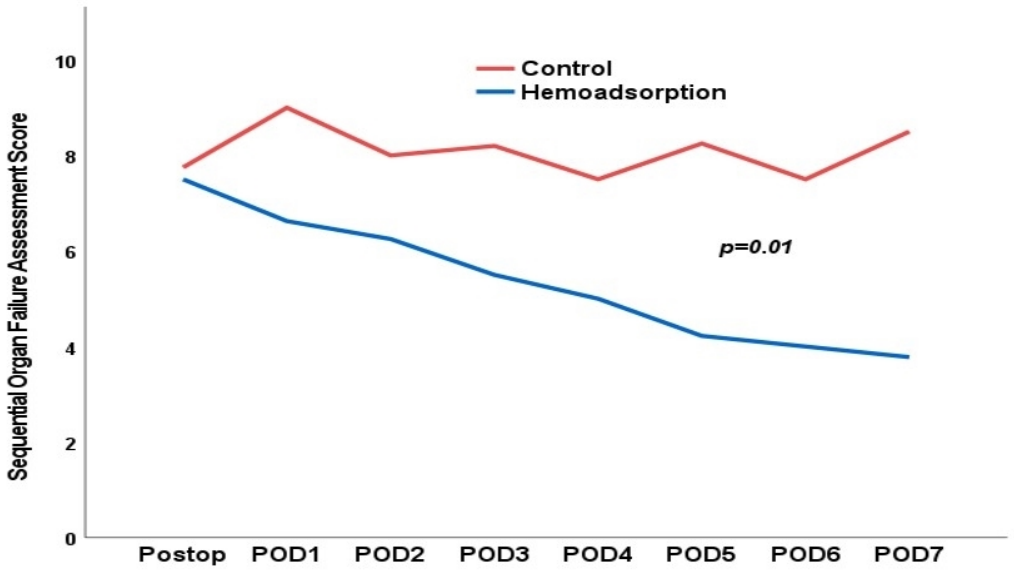


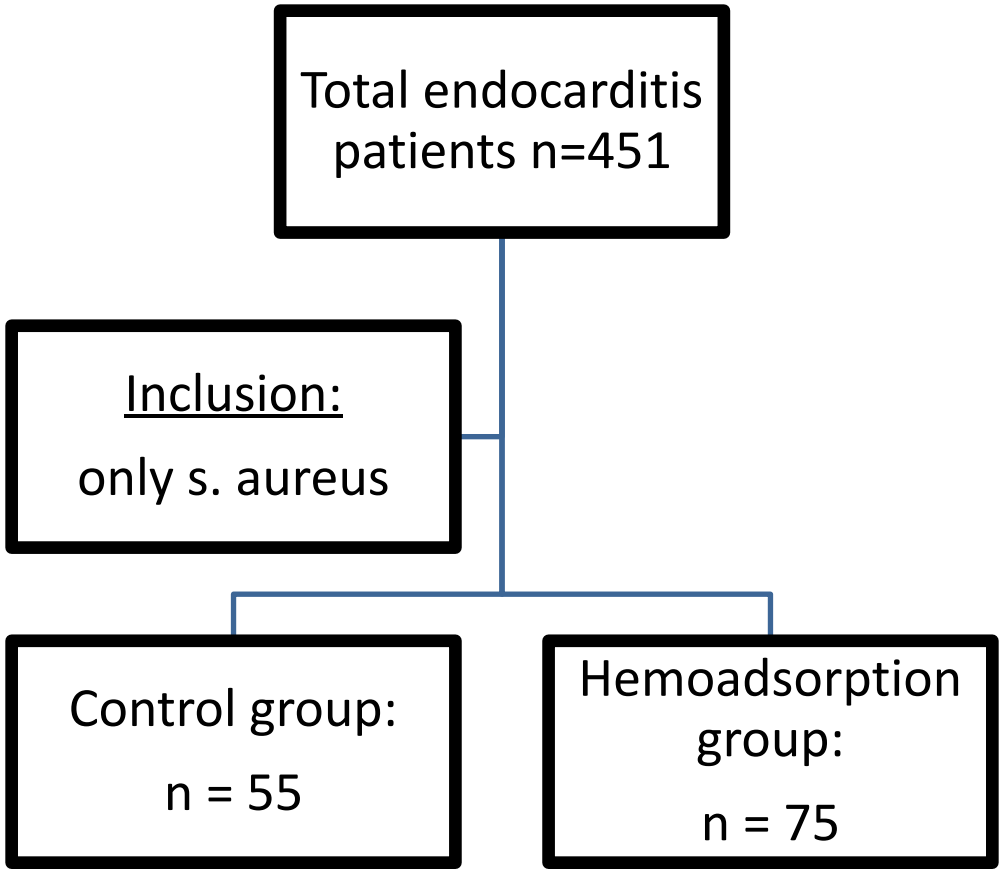


……accepted 03/22

Update staphylococcus aureus endocarditis

Essen & Nuremberg data

- 01/2015 – 03/2022
- consecutive MRSA & MSSA pts.
- only intraoperative CytoSorb use
- all-comers
- local ethics approval
- Retrospective analysis



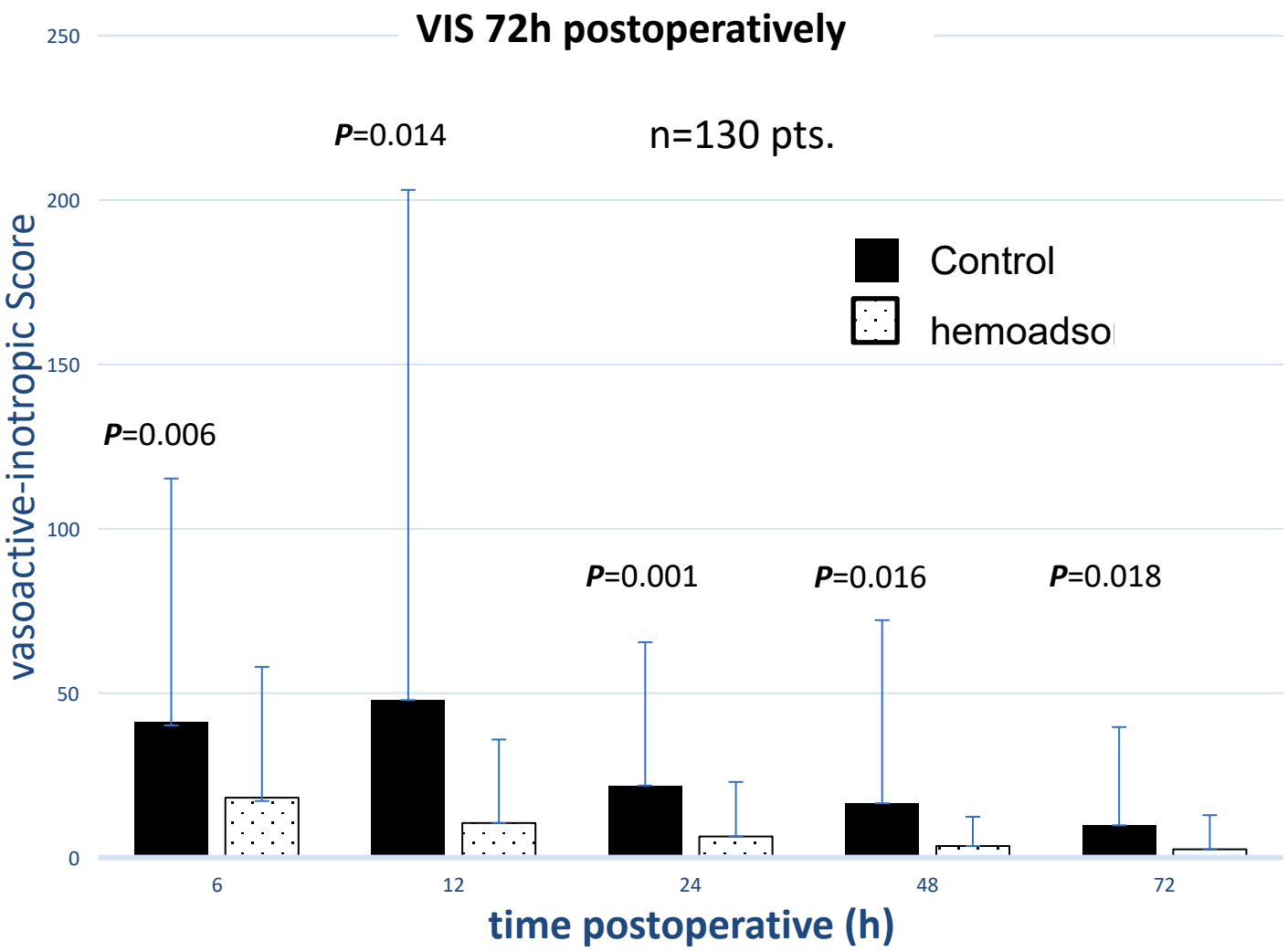
E&N Endocarditis - demographics

Variable, demographics	Control n=55	Hemoadsorption n=75	p
Age, years	60.8±13.5	60.7±16.5	0.99
Gender, male	32 (58.2)	38 (50.1)	0.47
Arterial hypertension	35 (63.6)	47 (62.7)	1.00
IV drug abuse	12 (21.8)	10 (13.3)	0.24
Dialysis dependent	5 (9.1)	6 (8.0)	1.00
COPD	10 (18.2)	12 (16.0)	0.81
Peripheral vascular disease	9 (16.3)	6 (8.0)	0.17
Liver cirrhosis	2 (3.6)	3 (4.0)	1.00
Pulmonary hypertension	3 (5.5)	4 (5.3)	1.000
Coronary artery disease	13 (23.6)	25 (33.3)	0.24

E&N Endocarditis - Comorbidities

EuroSCORE II, %	12.0±11.5	11.9±15.2	0.49
Reoperation	15 (27.2)	19 (25.2)	0.84
Atrial fibrillation	12 (21.8)	26 (34.7)	0.12
History of stroke	18 (32.7)	28 (37.3)	0.71
Fever	45 (81.8)	57 (76.0)	0.51
MSSA	52 (94.5)	69 (92.0)	0.73
MRSA	3 (5.5)	6 (8.0)	0.73

E&N Endocarditis – Inotropic support



Essen&Nuremberg staphylococcus aureus Endocarditis cohort

Mortality

	Control	Hemoadsorption	P
Sepsis-related mortality	12 (21.8)	6 (8.0)	0.02
30-day mortality	18 (32.7)	12 (16.0)	0.03
90-day mortality	22 (40.0)	14 (18.7)	0.009

CONCLUSIONS

The need for inotropes, kidney failure requiring dialysis, excessive bleeding and inflammation were independently associated with sepsis-associated and in-hospital mortality.

Intraoperative hemoadsorption may reduce sepsis, sepsis-associated mortality and inflammatory response after surgery for infective endocarditis in high-risk patients with left-sided native or prosthetic valve endocarditis.

Treatment was safe without CytoSorb® device-related adverse events in all high-risk cohorts presented.

Seemingly, S. Aureus subcohorts benefit more from Hemoadsorption therapy.

Optimal timing, strategy and duration of Hemoadsorption needs to be determined in the future in most responsive subcohorts.