UNIVERSITÄTS
KLINIKUM
HEIDELBERG

Von der akuten Herzinsuffizienz und der Umsetzung der neuen Leitlinien

Mechanical Circulatory Support und ECPR im Kardiogenen Schock und unter Reanimation

Matthias Eden 2023

UK HD

Interessenskonflikte

Vortrags- und Beratertätigkeit: Astra Zeneca, Abiomed, Novartis

Guidelines und Pharmakotherapie in der chronischen Herzinsuffizienz (HEF-REF)

2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

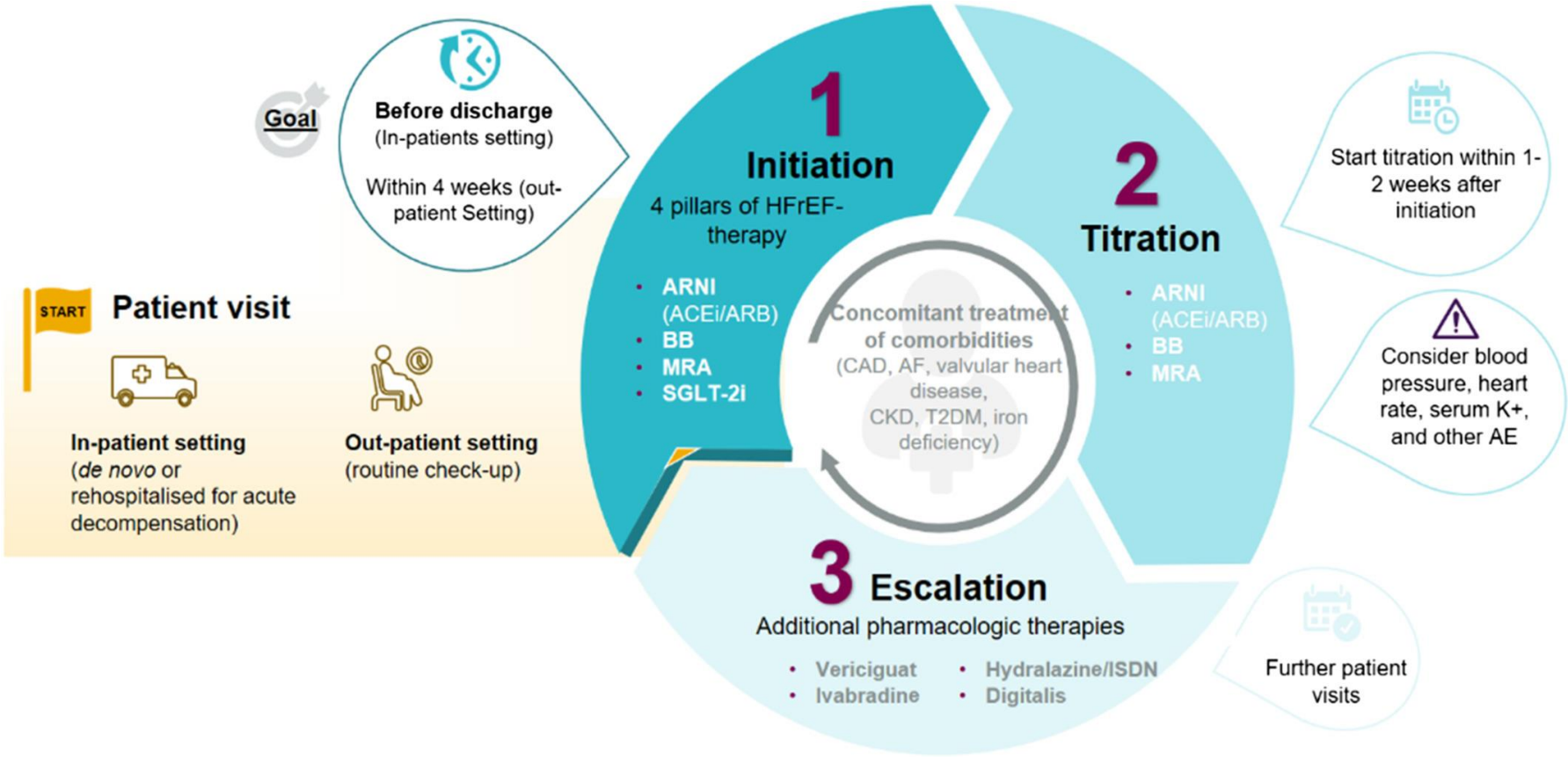
Developed by the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)

A practical approach to the guideline-directed pharmacological treatment of heart failure with reduced ejection fraction

Amr Abdin[1*], Johann Bauersachs[2], Samira Soltani[2], Matthias Eden[3], Norbert Frey[3] and Michael Böhm[1]

[1]*Department of Internal Medicine III—Cardiology, Angiology and Intensive Care Medicine, Saarland University Medical Center, Saarland University, Saarbrücken, Germany;* [2]*Department of Cardiology and Angiology, Hannover Medical School, Hannover, Germany; and* [3]*Department of Internal Medicine III, University of Heidelberg, Heidelberg, Germany*

Guidelines und Pharmakotherapie in der chronischen Herzinsuffizienz (HEF-REF)



Guidelines und Therapie in der akuten Herzinsuffizienz

2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

Developed by the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)

Akut dekompensierte Herzinsuffizienz

Akutes Lungenödem

Isoliertes Rechtsventrikuläres Versagen

Kardiogener Schock

ESC European Society of Cardiology
European Heart Journal (2021) 42, 3599 – 3726
doi:10.1093/eurheartj/ehab368

Akut dekompensierte Herzinsuffizienz

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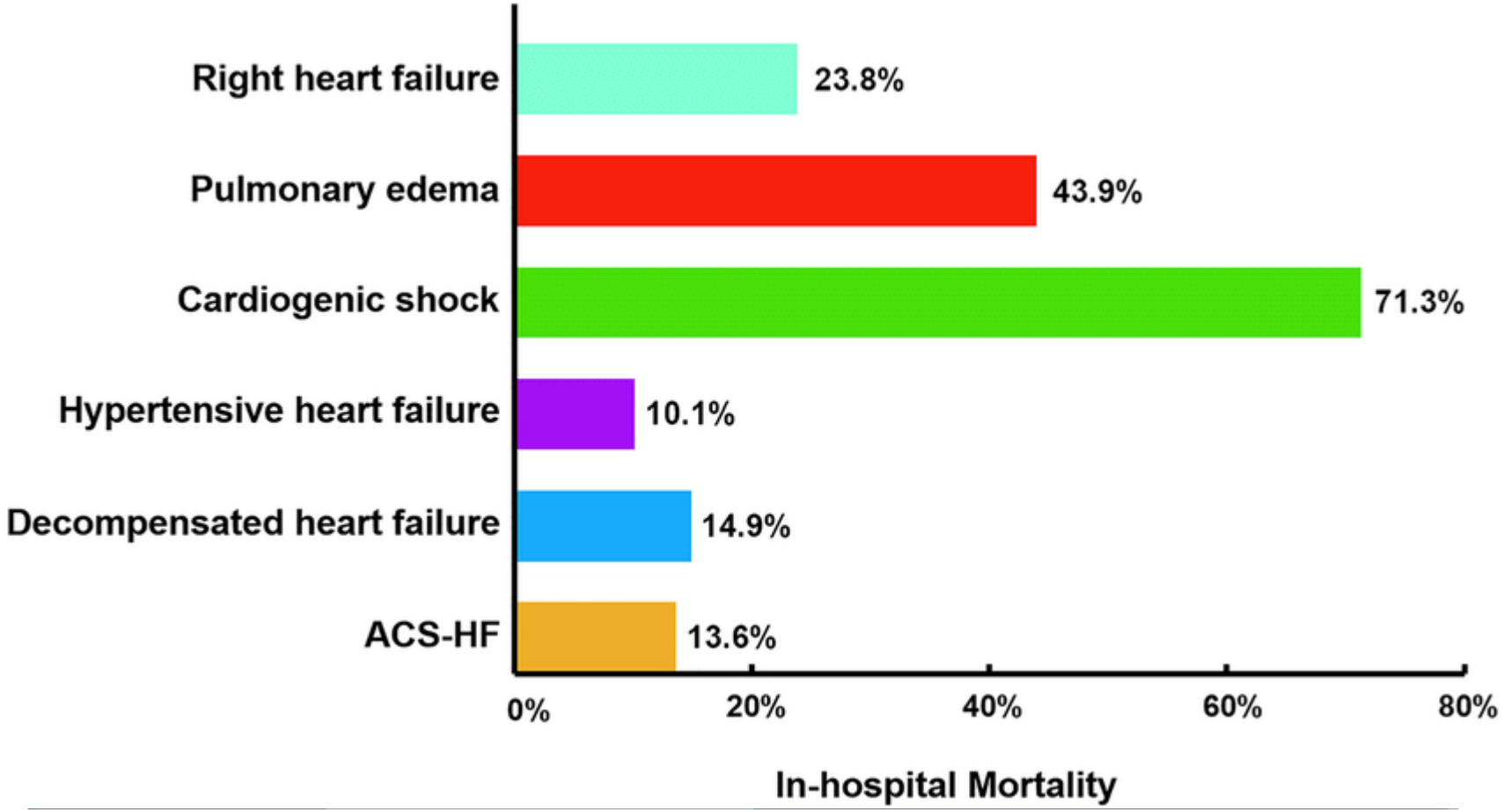


ESC Heart Failure2021;8:423–437

Ätiologie der akuten Herzinsuffizienz

Acute Heart Failure

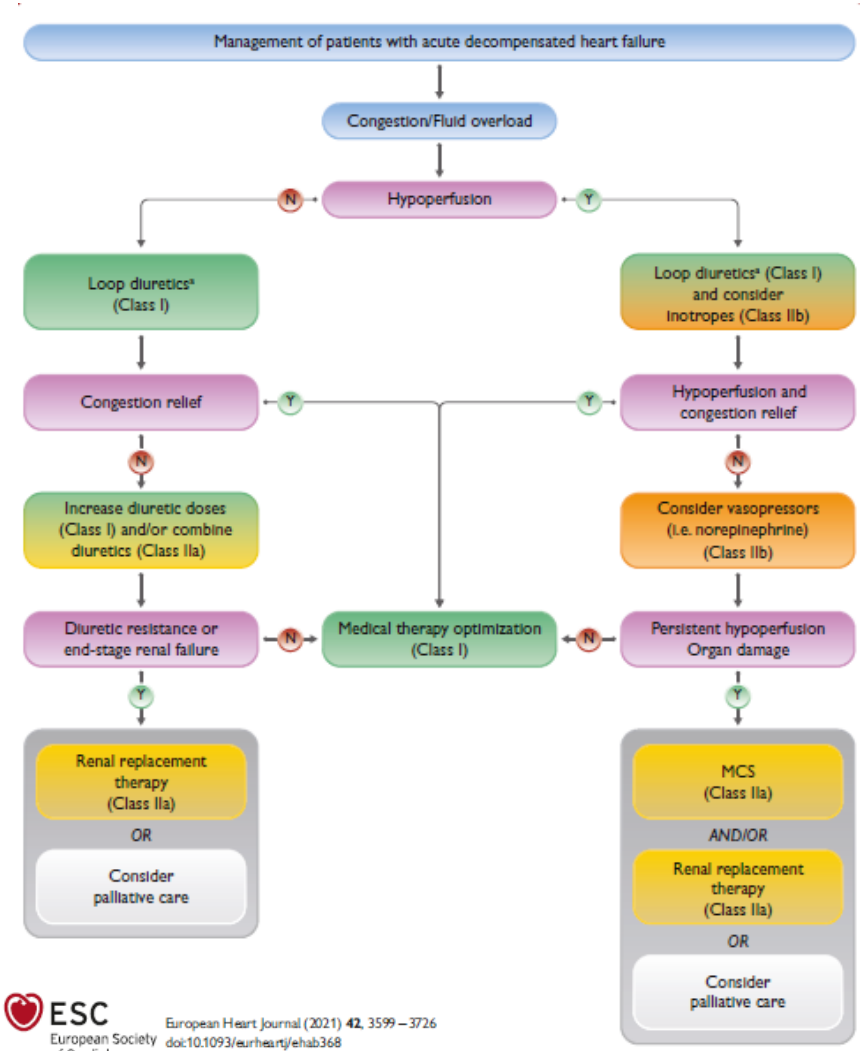
- ACS, myocardial infarction
- Takotsubo-Cardiomyopathy
- Severe myocarditis
- Peripartum Cardiomyopathy
- Endocarditis

Acute decompensated Chronic Heart Failure

- Arrhythmias
- ACS, myocardial infarction
- Aggravated comorbidities (DM, AHT)
- Physical exhaustion
- Fluid overload
- Withdrawal/changes/intolerance in medication

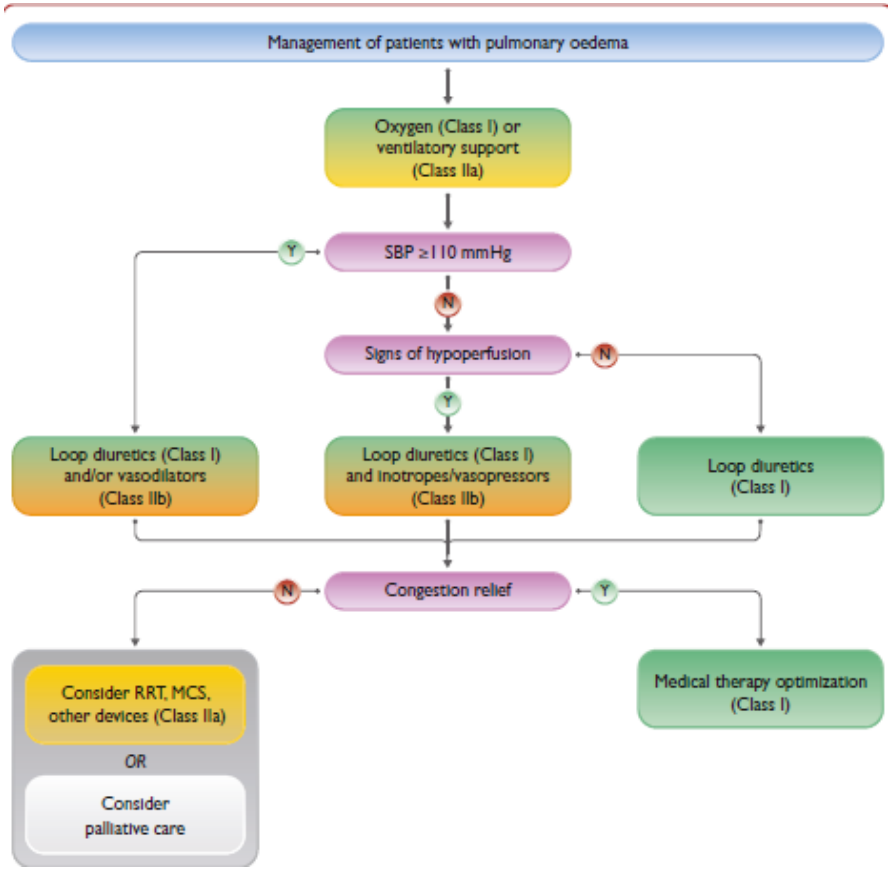
ESC HEART FAILURE
ESC Heart Failure 2020; **7**: 1118–1124

Akut dekompensierte Herzinsuffizienz

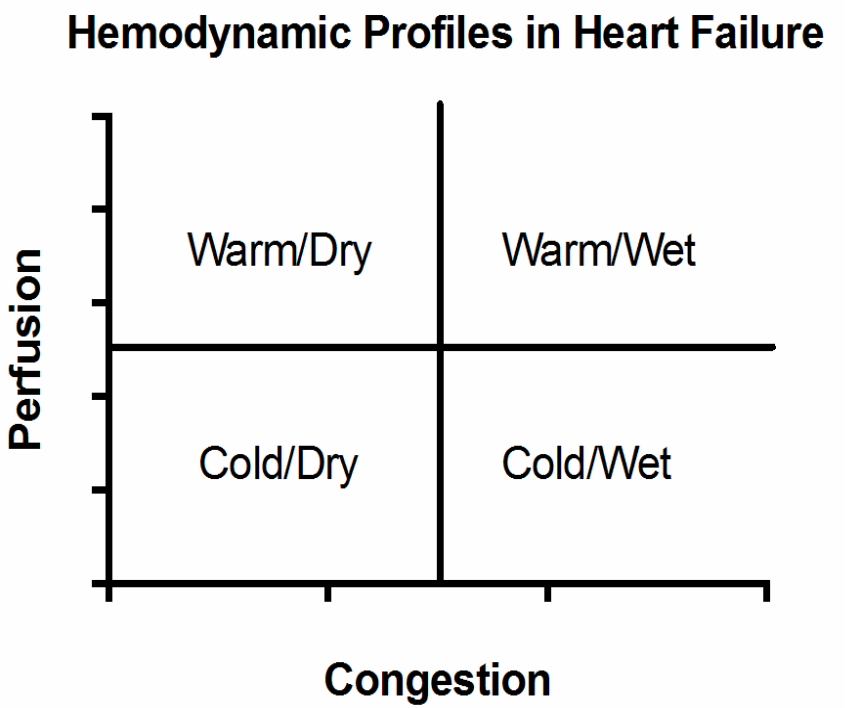


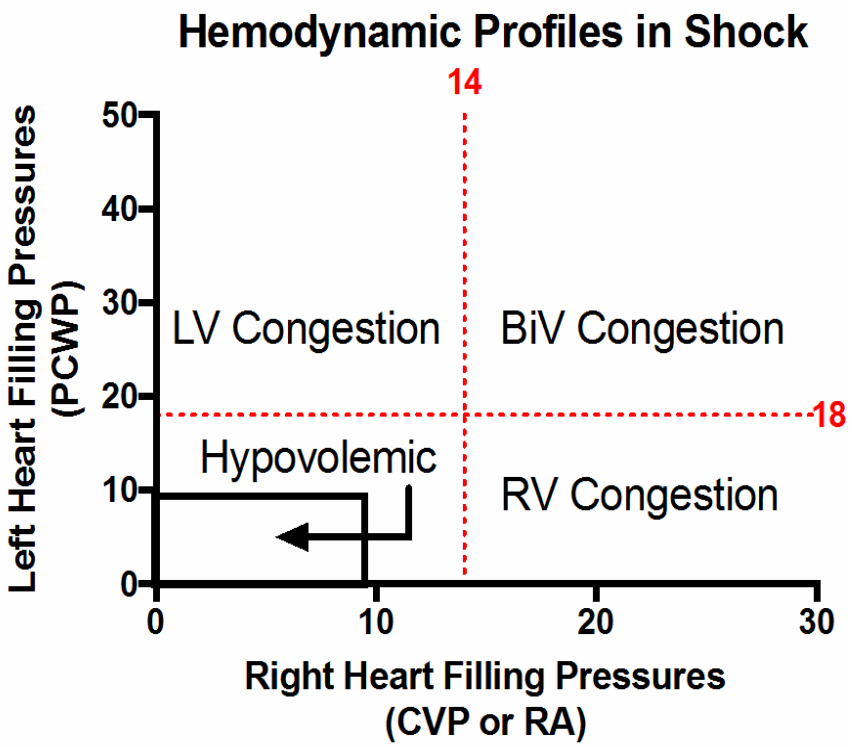
ESC European Society of Cardiology
European Heart Journal (2021) 42, 3599–3726
doi:10.1093/eurheartj/ehab368

Akutes Lungenödem



Klinische Variabilität der akuten Herzinsuffizienz und des kardiogenen Schock





Kapur & Esposito. F1000 Reviews 2017

Vasopressoren und Inotropika

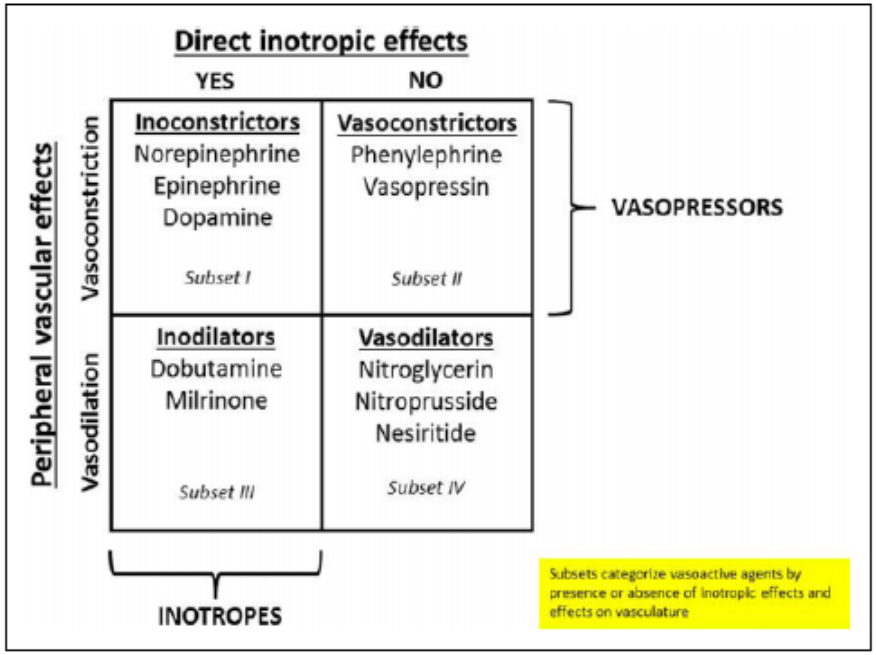


Figure 1. Proposed classification of vasoactive agents.

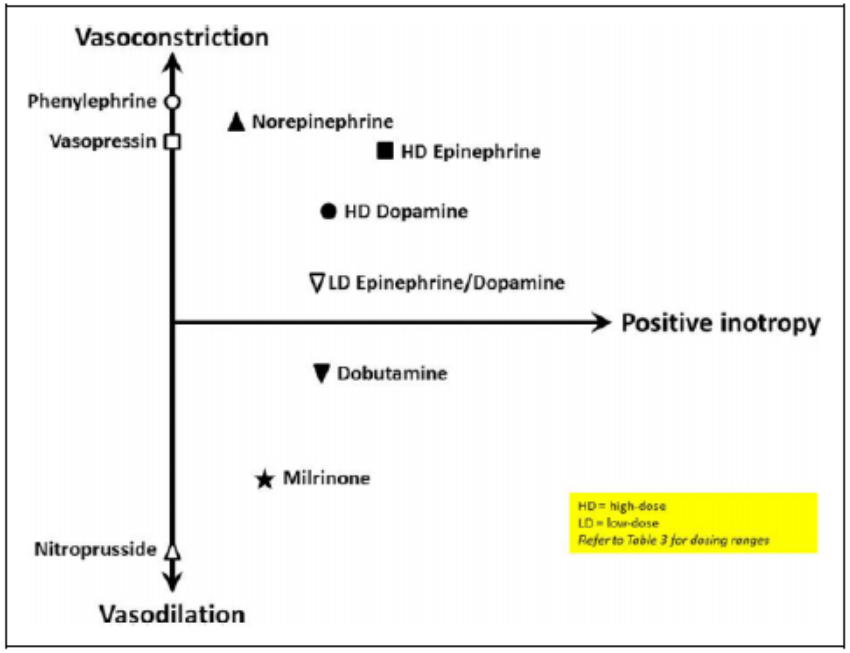


Figure 2. Vascular response to vasoactive medications.

Journal of Cardiovascular Pharmacology and Therapeutics 2015, Vol. 20(3) 249-260

Vasopressoren und Inotropika

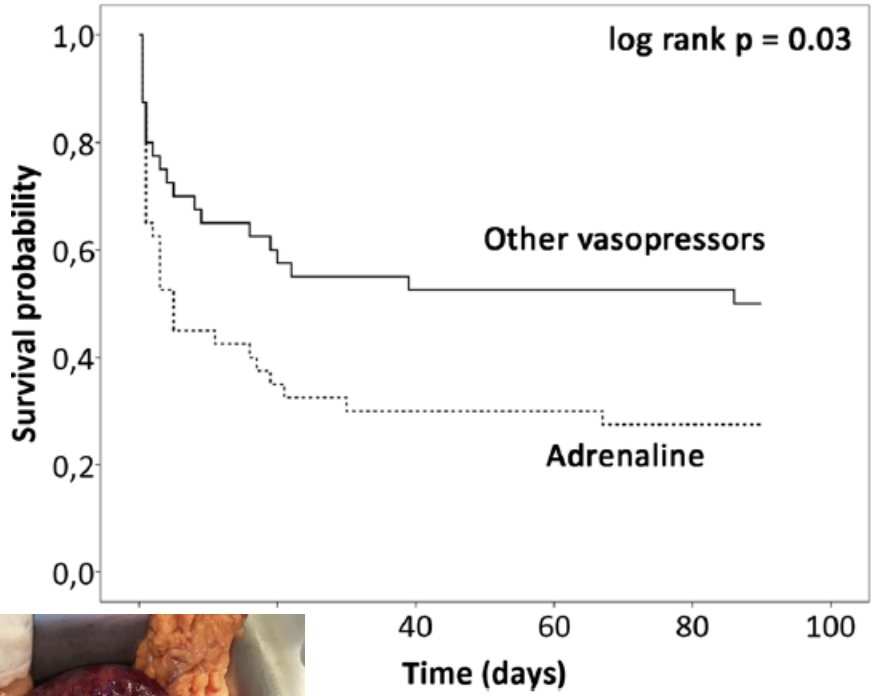
Tarvasmäki *et al. Critical Care* (2016) 20:208
DOI 10.1186/s13054-016-1387-1

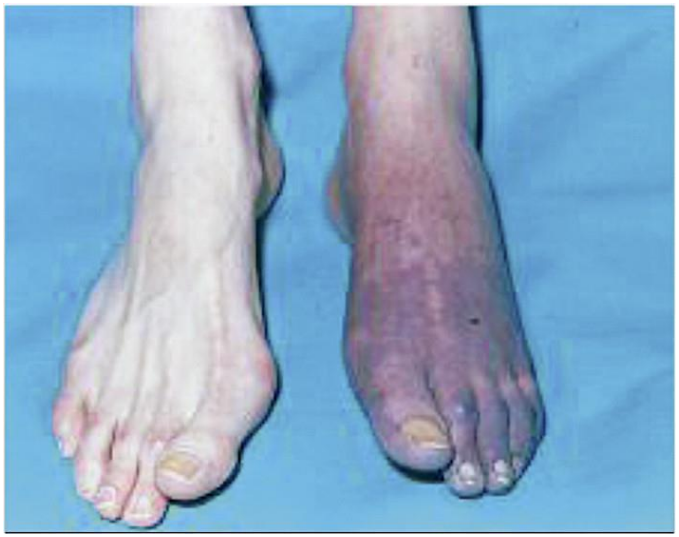
Critical Care

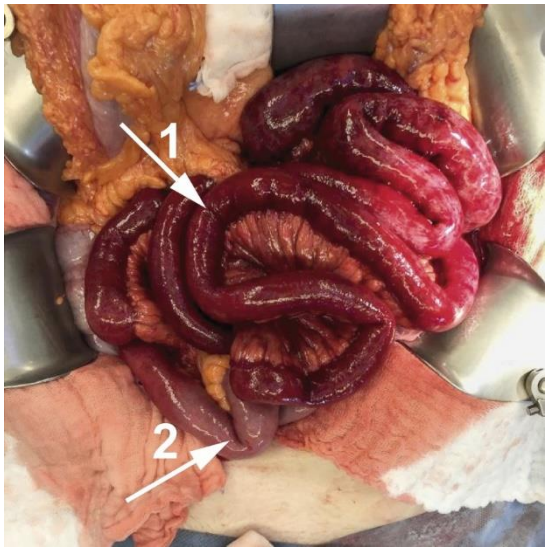
RESEARCH Open Access

Current real-life use of vasopressors and inotropes in cardiogenic shock - adrenaline use is associated with excess organ injury and mortality

Tuukka Tarvasmäki[1*], Johan Lassus[2], Marjut Varpula[2], Alessandro Sionis[3], Reijo Sund[4], Lars Køber[5], Jindrich Spinar[6], John Parissis[7], Marek Banaszewski[8], Jose Silva Cardoso[9], Valentina Carubelli[10], Salvatore Di Somma[11], Alexandre Mebazaa[12], Veli-Pekka Harjola[1] and for the CardShock study investigators







The bumpy road to drug development for acute heart failure

Carine E. Hamo[1], Javed Butler[1], Mihai Gheorghiade[2], and Ovidiu Chioncel[3]*

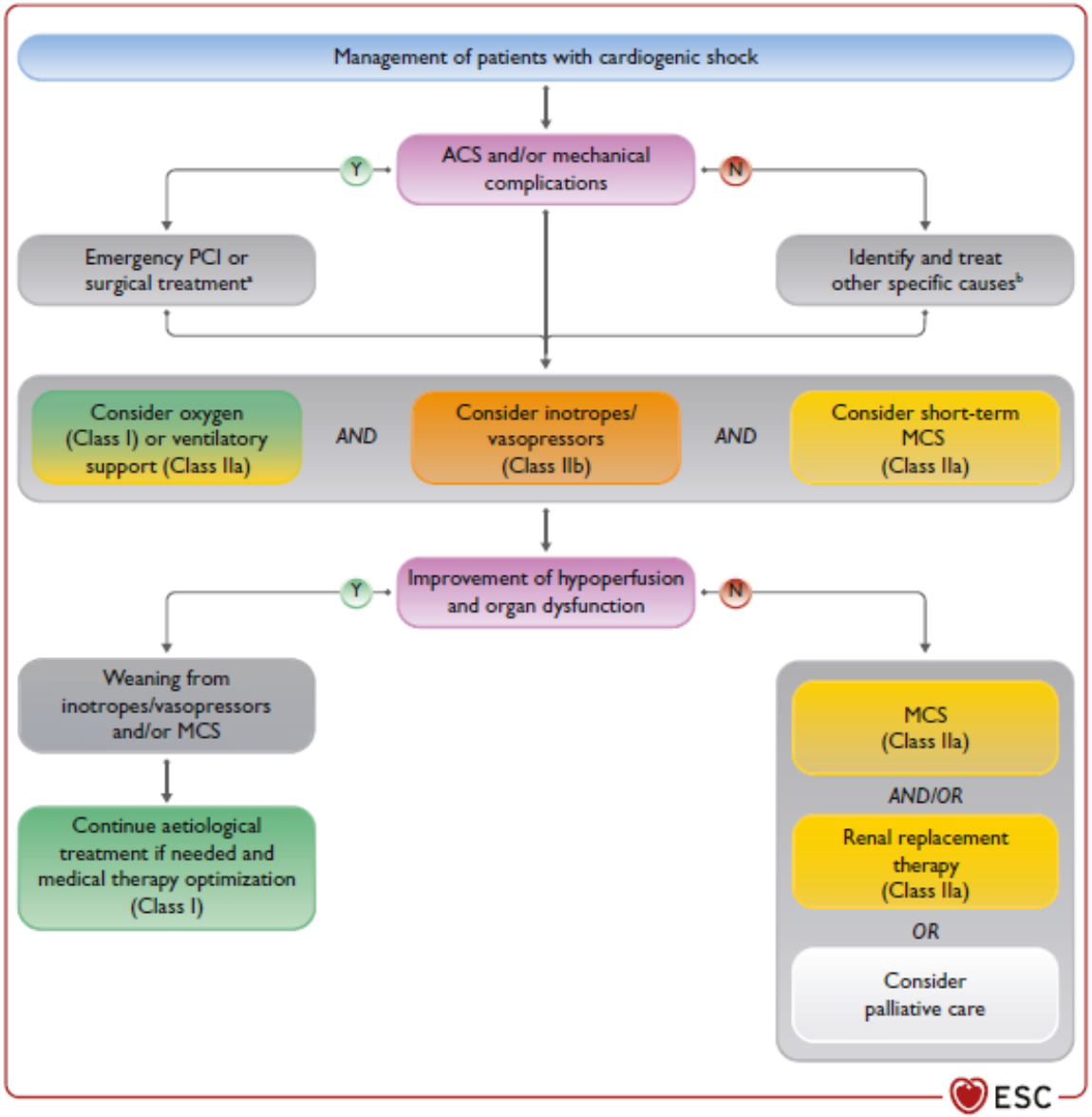
SIRIUS II 2006[22]	Intravenous ularitide (at 7.5, 15, and 30 ng/kg/min) vs. placebo as 24 h continuous infusion in ADHF	• Change in PCWP at 6 h compared with placebo • Change in patient's self-assessed dyspnoea score at 6 h compared with placebo	• Ularitide 7.5 ng/kg/min ($P = 0.014$), 15 ng/kg/min ($P = 0.031$), and 30 ng/kg/min ($P = 0.015$) • Ularitide 7.5 ng/kg/min ($P = 0.0026$), 15 ng/kg/min ($P = 0.0026$), 30 ng/kg/min ($P = 0.0026$)
SURVIVE 2007[29]	Intravenous levosimendan vs. i.v. dobutamine in patients with ADHF requiring inotropic support	All-cause mortality at 180 days	HR 0.91 (CI 0.74-1.13); $P = 0.4$
EVEREST 2007[33]	*Per os* tolvaptan vs. placebo within 48 h of admission for WCHF	• All cause mortality • CV death or hospitalization for HF	• HR 0.98 (CI 0.87-1.11); $P = 0.68$ • HR 1.04 (CI 0.95-1.14); $P = 0.55$
VERITAS 2007[16]	Intravenous tezosentan vs. placebo for 24-72 h within 24 h of WCHF	• Change in dyspnoea over 24 h • Incidence of death or WHF at 7 days	– VERITAS 1: −12(CI − 105 to 81) mm·h; $P = 0.8$; VERITAS 2: −25 (CI − 119 to 69) mm·h; $P = 0.6$-OR 0.99 (CI 0.82-1.21); $P = 0.95$
ECLIPSE 2008[32]	*Per os* tolvaptan (15, 30, 60 mg) vs. placebo in advanced HF	PCWP peak change from baseline within 3-8 h of administration	$P < 0.05$ for all tolvaptan doses vs. placebo
HORIZON-HF 2009[30]	Intravenous istaroxime vs. placebo as 6 h infusion in patients with ADHF	−Change in PCWP compared with placebo after 6 h continuous infusion	Istaroxime PCWP change from baseline −3.7 ± 6 ($P < 0.0001$)
REACH UP 2010[87]	Intravenous rolofylline vs. placebo in patients with ADHF as a 4 h infusion for three consecutive days	Proportion of treatme within 30 days (dea mission for HF with worsening sign/sym >24 h after drug to charge, WRF)	
PROTECT 2010[25]	Intravenous rolofylline vs. placebo within 24 h of WCHF	Proportion of patient ment success, treat ure, or no change i clinical condition	
ASCEND-HF 2011[18]	Intravenous nesiritide vs. placebo	• Change in dyspnoea at 6 and 24 h • Rehospitalization for HF or death within 30 days	• 6 h (44.5% vs. 42.1%); $P = 0.03$ • 24 h (68.2% vs. 66.1%); $P = 0.007$ • OR − 0.7 (CI − 2.1 to 0.7); $P = 0.31$
DOSE 2011[13]	Intravenous furosemide administered as bolus or continuous infusion (administered at low dose or high dose)	• Patient's global assessment of symptoms • Change in serum creatinine from baseline to 72 h	• Mean AUC, 4326 ± 1440 and 4372 ± 1404; $P = 0.47$ • 0.05 ± 0.3 mg/dL and 0.07 ± 0.3 mg/dL; $P = 0.45$
ASTRONAUT 2013[45]	*Per os* aliskiren vs. placebo initiated at median 5 days of WCHF	CV death or HF rehospitalization at 6 months	HR 0.92 (CI 0.76-1.12); $P = 0.41$
REVIVE-II 2013[27]	Intravenous levosimendan vs. placebo for 24 h	Clinical course during first 5 days (improved, unchanged, worse)	58 vs. 44 patients were improved at 6 h, 24 h, and 5 days; 58 vs. 82 patients were worse ($P = 0.015$)
ROSE-AHF 2013[26]	Intravenous dopamine or i.v. nesiritide vs. placebo in ADHF and renal dysfunction	• 72 h cumulative urine volume • Change in serum cystatin C from enrolment to 72 h	• Dopamine vs. placebo: 229 mL (CI − 714 to 1171); $P = 0.59$ • Nesiritide vs. placebo: 279 mL (CI − 618 to 1176); $P = 0.49$ • Dopamine vs. placebo: 0.01 (−0.08 to 0.10); $P = 0.72$ • Nesiritide vs. placebo: −0.04 (CI − 0.13 to 0.05); $P = 0.36$
RELAX-AHF 2013[21]	Intravenous seralexin vs. placebo for 48 h	• Improvement in dyspnoea from baseline to Day 5 • Patient reported relative to baseline	• VAS AUC 448 mm·h (CI 120-775); $P = 0.007$ • Likert scale 26% vs. 27%; $P = 0.7$
ARTS-HF 2015[88]	*Per os* finerenone (at one of five doses) vs. eplerenone for 90 days in patients with DM and/or CKD	Proportion of patients with a relative decrease in NT-proBNP of > 30% from baseline to 90 days	The proportion of patients who had NT-proBNP decrease of > 30% at Day 90 compared with baseline was similar in the finerenone groups and eplerenone group
SOCRATES 2015[37]	*Per os* sGC stimulator vericiguat vs. placebo for 12 weeks	SOCRATES-REDUCED: change in NT-proBNP from baseline to 12 weeks SOCRATES-PRESERVED: change in NT-proBNP from baseline to 12 weeks and change in LAV from baseline to 12 weeks	No change in the pooled vericiguat arms vs. placebo in NT-proBNP levels but dose-dependent reductions were seen with higher doses in SOCRATES REDUCED
FIGHT 2015[43]	SC GLP-1 agonist liraglutide vs. placebo for 180 days	Time to death, time to HF hospitalization, time-averaged proportional change in NT-proBNP (from baseline to 180 days)	No change in mean global rank score between placebo (155) vs. liraglutide (146); $P = 0.309$
BLAST-AHF 2016[89]	Intravenous TRV027 at three doses vs. placebo in ADHF	Death, rehospitalization for HF, WCHF, change in dyspnoea VAS score, length of hospital stay	No potentially efficacious dose of TRV027 was identified in the targeted AHF population

and

−No change in dyspnoea response on Likert Scale

European Heart Journal Supplements (2016) **18**(Supplement G), G19-G32
The Heart of the Matter
doi:10.1093/eurheartj/suw045

Der kardiogenen Schock

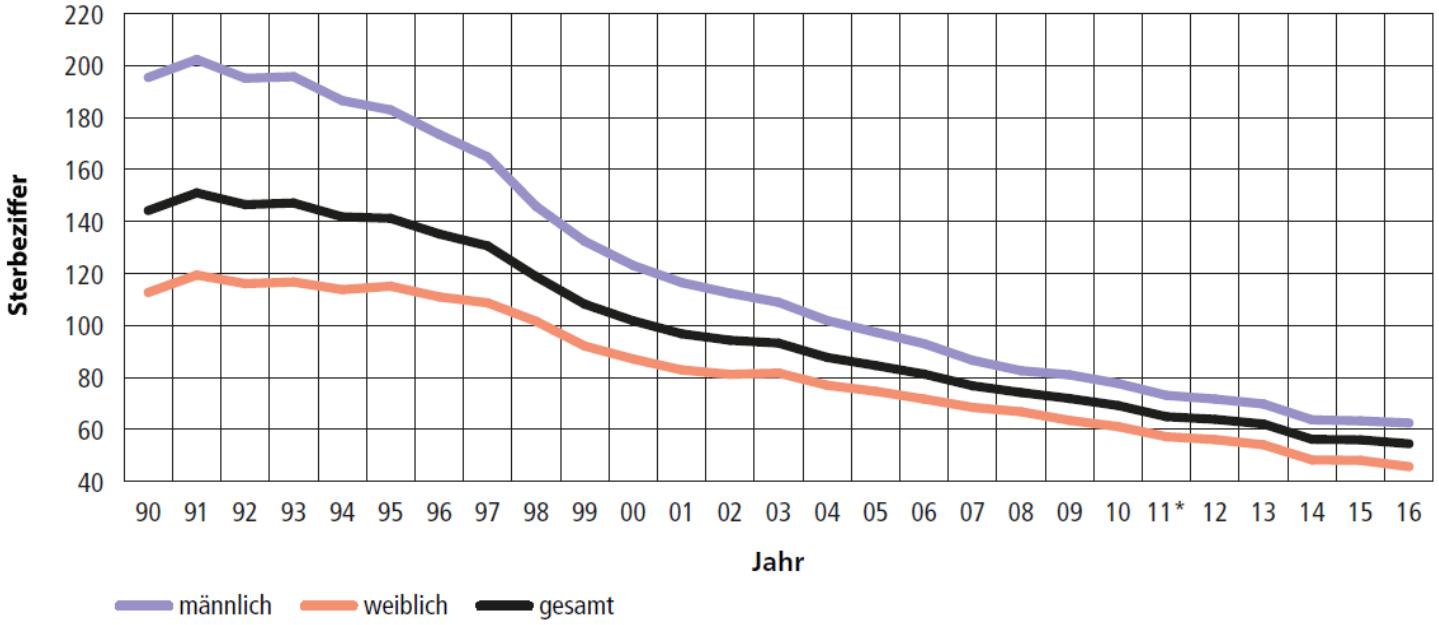


ESC European Society of Cardiology
European Heart Journal (2021) 42, 3599 – 3726
doi:10.1093/eurheartj/ehab368

Der kardiogene Schock: Ein lebensbedrohlicher Notfall

Der kardiogene Schock

Der akute Myokardinfarkt ist das mit ca. 75-80% häufigste auslösende Ereignis für einen kardiogenen Schock.

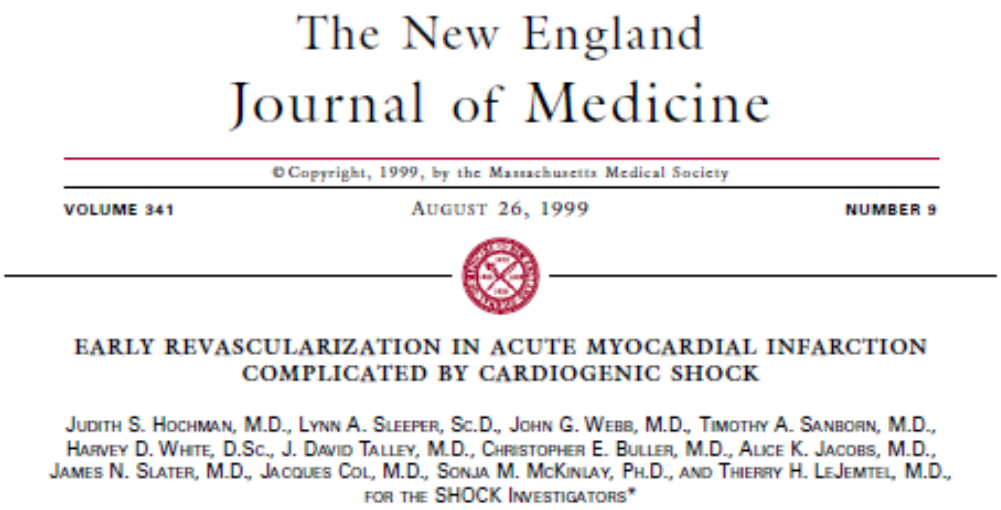


Berechnung auf Grundlage von Daten des Statistischen Bundesamtes
* ab 2011 Bevölkerung auf Grundlage des Zensus 2011

Herzinfarktsterblichkeit in Deutschland (1990-2016)

Kardiogener Schock ist definiert als eine Endorganhypoperfusion (kalte Extremitäten, Oligurie, Bewusstseinsstörung, Laktaterhöhung) bei persistierender Hypotension (RR < 80 mmHg für ≥ 30 min) infolge eines Herzversagens (CI < 1,8 l/min/m²) - trotz ausgeglichenem Volumenstatus.
Erhöhte kardiale Füllungsdrücke (PCWP>18mmHg)

Kardiogener Schock – Synonym: **Kardiales Pumpversagen**



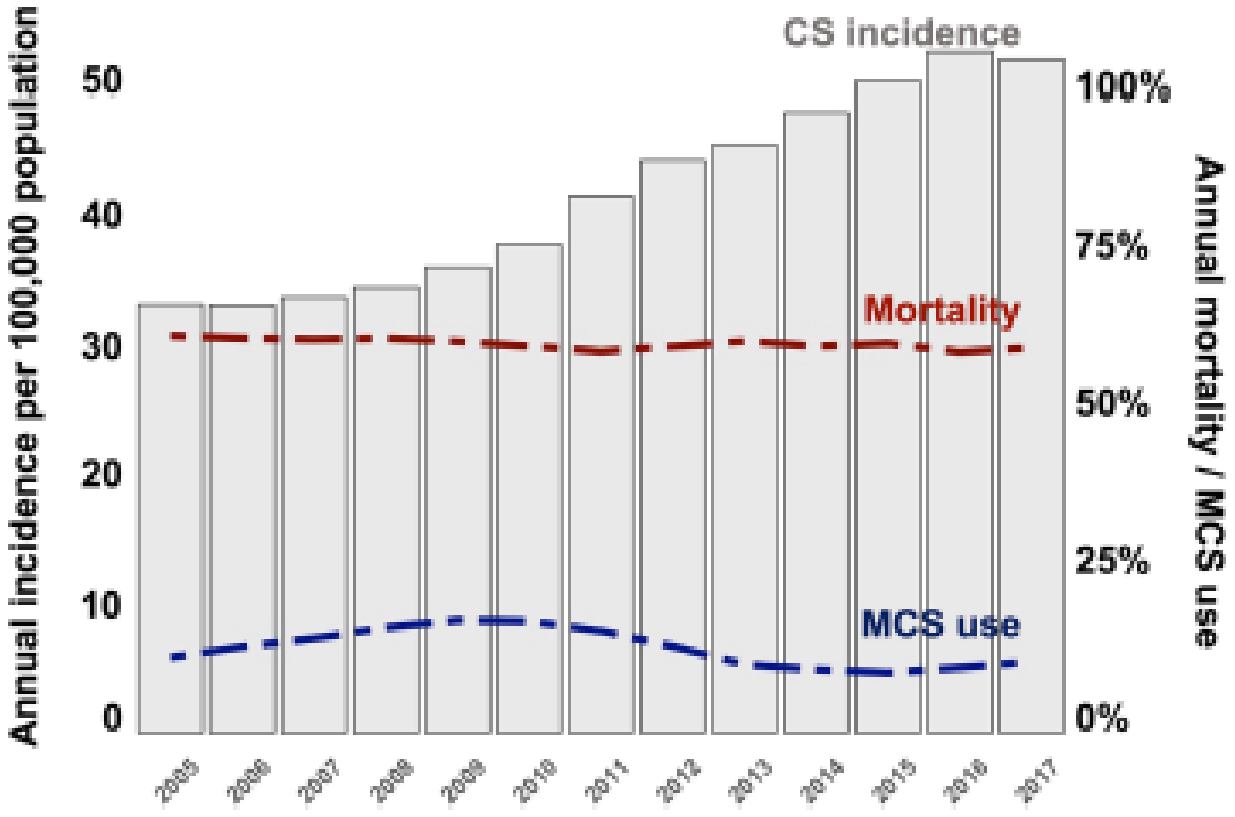
The New England
Journal of Medicine

VOLUME 341 AUGUST 26, 1999 NUMBER 9

EARLY REVASCULARIZATION IN ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK

JUDITH S. HOCHMAN, M.D., LYNN A. SLEEPER, SC.D., JOHN G. WEBB, M.D., TIMOTHY A. SANBORN, M.D., HARVEY D. WHITE, D.SC., J. DAVID TALLEY, M.D., CHRISTOPHER E. BULLER, M.D., ALICE K. JACOBS, M.D., JAMES N. SLATER, M.D., JACQUES COL, M.D., SONJA M. MCKINLAY, PH.D., AND THIERRY H. LEJEMTEL, M.D., FOR THE SHOCK INVESTIGATORS*

Epidemiologie des kardiogenen Schock



ESC Heart Failure 2021; 8: 1295–1303
DOI: 10.1002/ehf2.13202

Verwendung von Protokollen im kardiogenen Schock

„Fast Entry“

ECPR

ECLS (VA ECMO)
Geprimetes Gerät im BZ
Extrahospitale Kanülierung (MIC)
Prüfe Checkliste ECPR

HKU/PCI
+/-Impella CP
Venting
(LV Distension, LVEDP >30-35mmHg, HF >100bpm (SR))
Vor PCI

Alternativ „*Neurescue*“ Studien-Einschluss prüfen

„Slow Entry“

Kardiogener Schock

Refraktär (>30min) auf Volumentherapie, Inotropika und Vasokonstriktoren (SBP <90mmHg, ZvSO2 <60% und Laktatakkumulation, LVEDP >30-35mmHg, CI<1,8l/min/m2, Klinik

Prüfe Checkliste Pro und Contra
Htx Kandidat? LVAD/BiVAD Kandidat?

Schwere Hypoxie (HI <150)	**BiV Versagen**	**RV Versagen**	**LV Versagen**
VA ECMO Ggf. VA-V ECMO	VA ECMO +/- Impella CP	VA ECMO *Ggf. Tandem Heart*	Impella CP oder 5.5

-Idealerweise Hämodynamisches Monitoring
(PAK: >CI 2,2l/Min x m2, SVR 800-1000 dyn/cm x s-5, PCWP 12-18mmHg)
(MAP>65mmHg, kein oder regredientes Laktat, ZvSO2 >70%, pulsatiler Auswurf, Minimum Katecholamin
-Monitoring der Zugangswege
-Monitoring von BB, Elyte, Krea/Hst, Billirubin, Transaminasen, Haptoglobin, ACT, AT III, D-Dimere, Fibrinogen, CK, LDH, Hs-TnT, NT-pro BNP, CRP, PCT
- Monitoring der Urinausscheidung (20ml/h)
-Lungenprotektive Beatmung (ggf. ultraprotektiv)
-Narkose und Neuromonitoring (*Narcotrend*)
-Täglich Echokardiographie (Eskalation/Deeskalation)
-Regelmäßig Evaluation Therapieziel/Erfolg/RS HCH

REVERSIBLE URSACHEN?

„Fast Entry“ Heidelberger ECPR Checkliste

Universitätsklinikum Heidelberg
Klinik für Kardiologie, Angiologie und Pneumologie
Cardiac Arrest Center
Im Neuenheimer Feld 410
69120 Heidelberg

ECPR/ECLS Checkliste

Patientenaufkleber

Datum:

Uhrzeit Beginn CPR:

Primäre ECLS Kriterien (alle sollten zutreffen):

- ☐ Beobachteter Herzstillstand
- ☐ Suffiziente Laienreanimation
- ☐ Initial schockbarer Rhythmus (gilt nicht für LAE)
- ☐ Keine limitierende Grunderkrankung (Sepsis, MODS, Aktives Malignom) [1]
- ☐ Keine vorliegende ausschließende Patientenverfügung
- ☐ Keine Lysetherapie erhalten

Sekundäre ECLS Kriterien (sollte maximal 2 „Nein“- Antworten enthalten):

Nein ☐	JA ☐	CPR („low flow“-Zeit) unter 60 Min.
Nein ☐	JA ☐	Laktat <20mmol/L und/oder pH >6,8
Nein ☐	JA ☐	Alter <75 Jahre
Nein ☐	JA ☐	Hb >7mg/dl oder keine Kontraindikation gegen Vollantikoagulation [2]
Nein ☐	JA ☐	SaO2 in erster BGA >85%
Nein ☐	JA ☐	Latenz („no flow“- Zeit) maximal 10 Min
Nein ☐	JA ☐	Keine klinische Zeichen der schweren Hirnschädigung
Nein ☐	JA ☐	keine prolongierte Reanimation >20 Min bei Asystolie / pers. VF/VT >120 Min [3]

1. Terminale Herzinsuffizienz, -limitierende COPD, fortgeschrittene Demenz; 2. schwere Blutung, Trauma, Hämatothorax; 3. Ausnahme: akzidentelle Hypothermie, Intoxikation, Beinahe-Ertrinken, V.a. LAE

Wenn Entscheidung nicht kongruent zu o.g. Kriterien, Begründung:

Universitätsklinikum Heidelberg
Klinik für Kardiologie, Angiologie und Pneumologie
Cardiac Arrest Center
Im Neuenheimer Feld 410
69120 Heidelberg

Einstimmige ECLS Entscheidung

Nein ☐ ☐ JA

Oberarzt/-ärztin Kardiologie | Stationsarzt/-ärztin Kardiologie | Pflegekraft HKL/Intensivstation

Dieses Formular verbleibt in der Patientenakte

- Entscheidung Beginn ECPR:
- Implantation ECLS:
- Ende Therapie ECLS:

☐ Einschluss ECLS-Register erfolgt

Quellen:

1. ELSO ECPR Supplement to the ELSO General Guidelines 12/2015
2. Empfehlungen zur extrakorporalen kardiopulmonalen Reanimation (eCPR) Konsensuspapier der DGIIN, DGK, DGTHG, DGfK, DGNI, DGAI, DIVI und GRC. Kardiologe https://doi.org/10.1007/s12181-018-0268-z

Medical Intervention Car (MIC) HD – präklinische ECPR bei OHCA

Einsatz der extrakorporalen Zirkulation (ECLS/ECMO) bei Herz- und Kreislaufversagen (AWMF-S3-Leitlinie): Bedeutung für die präklinische und klinische Notfallmedizin

Guido Michels[1] · Stephan Ensminger[2] · Alexander Assmann[3] · Christof Schmid[4] · Karl Werdan[5] · Malte Kelm[6] · Andreas Beckmann[7] · Udo Boeken[3] · S3 Guideline Group

Michels et al. Notfall Rettungsmed 2021

Medical Intervention Car (MIC) HD – präklinische ECPR bei OHCA

Tab. 1 Indikationen und Kontraindikationen [5, 6]

Empfehlung	COR	LOE
Die Entscheidung bezüglich einer ECLS sollte nach Abwägung von Pro- und Kontrakriterien individuell, im klinischen Kontext und im ECLS-Team erfolgen (adaptiert nach eCPR-Konsensuspapier 2018)	B	EK
Im kardiogenen Schock kann eine ECLS in Erwägung gezogen werden	0	+/++
Bei „in-hospital cardiac arrest" (IHCA) kann eine ECLS-Therapie (eCPR) in ausgewählten Fällen erwogen werden. Diese Entscheidung sollte frühzeitig getroffen werden	0 B	+++ +++
Bei „out-of-hospital cardiac arrest" (OHCA) kann eine ECLS-Therapie (eCPR) in ausgewählten Fällen erwogen werden. Diese Entscheidung sollte frühzeitig getroffen werden	0 B	++/+++ ++/+++

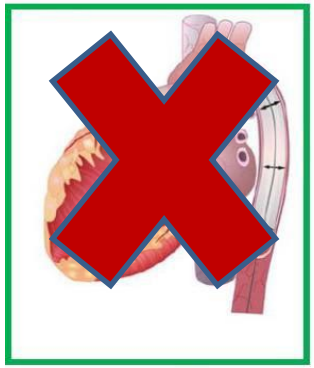
Michels et al. Notfall Rettungsmed 2021

Darstellung verschiedener temporärer MCS

Left Ventricle

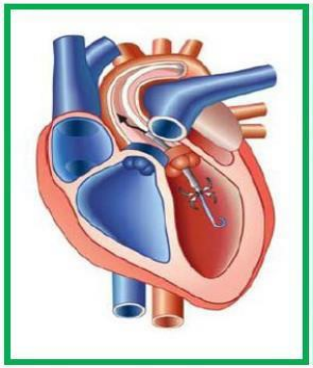
Continuous Flow Pumps

Pulsatile

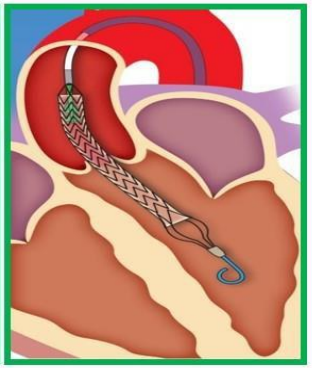


IABP

Axial-Flow



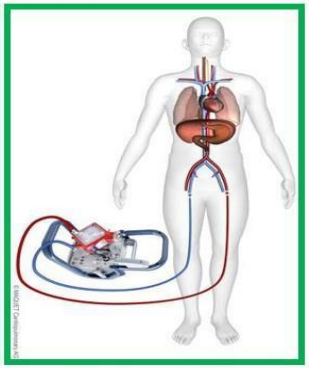
Impella CP



PHP *

Centrifugal Flow

TandemHeart

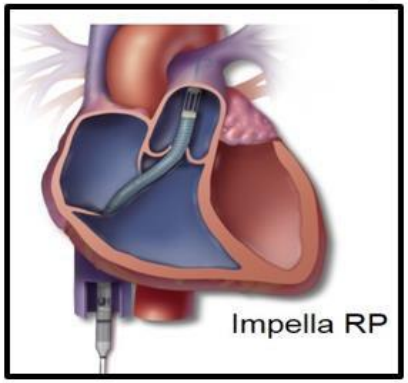


VA-ECMO

Intracorporeal | **Extracorporeal**

Right Ventricle

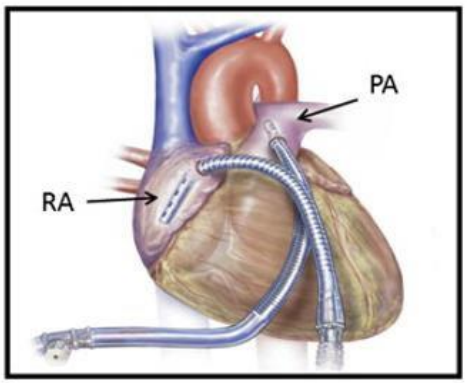
Axial Flow



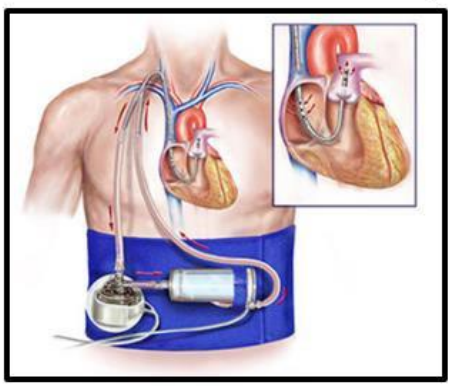
Impella RP

Centrifugal Flow

VA-ECMO



Tandem pRVAD

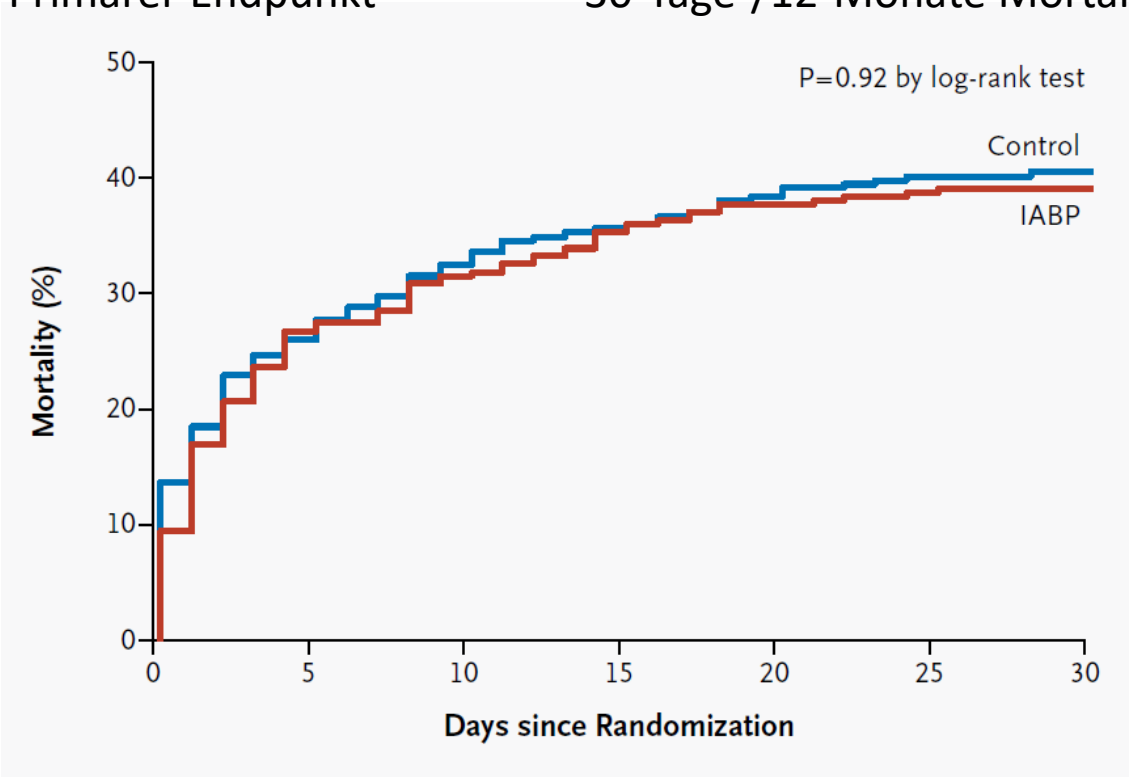


Protek Oxy-RVAD

Darstellung verschiedener temporärer MCS

IABP-SHOCK II Trial

Primärer Endpunkt 30-Tage-/12-Monate Mortalität



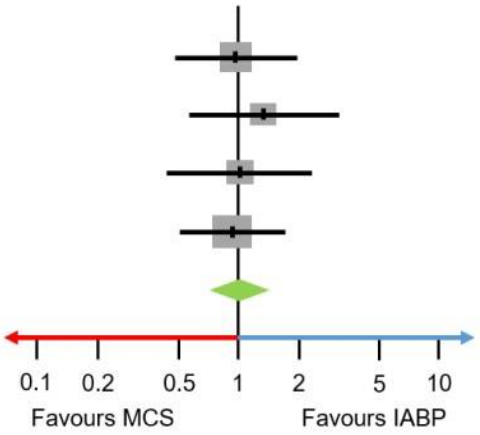
ABER: Kein Überlebensvorteil – Keine Standardtherapie mehr!

Thiele, H, NEJM 2013

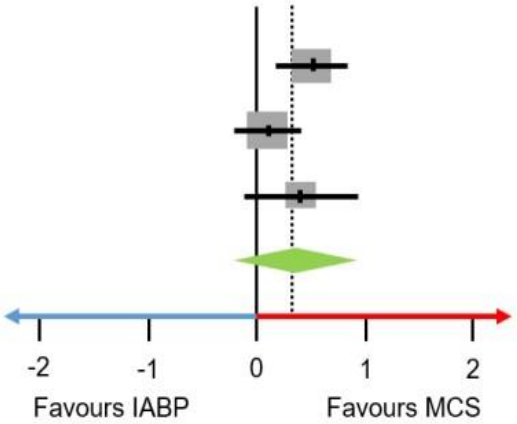
Hemodynamic Efficacy Without Clear Clinical Benefit in Small RCTs Comparing Acute MCS Options

	MCS		IABP		30-day mortality	RR	95%CI	Weight
	Events	Total	Events	Total				
Thiele et al.	9	21	9	20		0.95	[0.48;1.90]	26.8%
Burkhoff et al.	9	19	5	14		1.33	[0.57;3.10]	17.9%
ISAR-SHOCK	6	13	6	13		1.00	[0.44;2.29]	18.6%
IMPRESS-IN-SEVERE-SHOCK	11	24	12	24		0.92	[0.51;1.66]	36.7%
Overall	**35**	**77**	**32**	**71**		**1.01**	**[0.70;1.44]**	**100%**

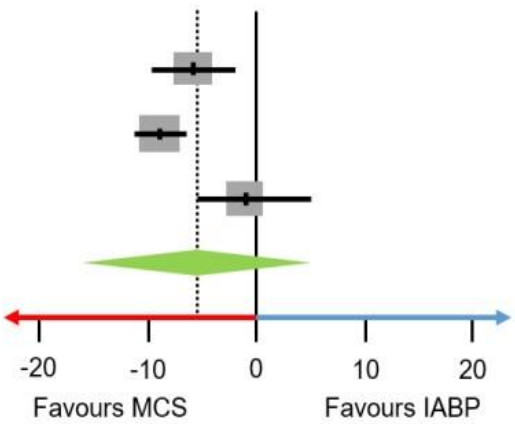
Heterogeneity: τ^2=0, I^2=0%, p=0.91
Test for overall effect: p=0.98



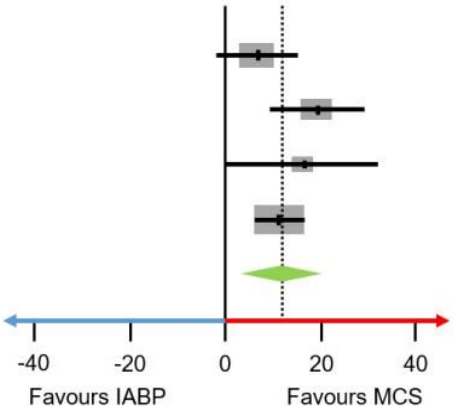
Cardiac index



PCWP

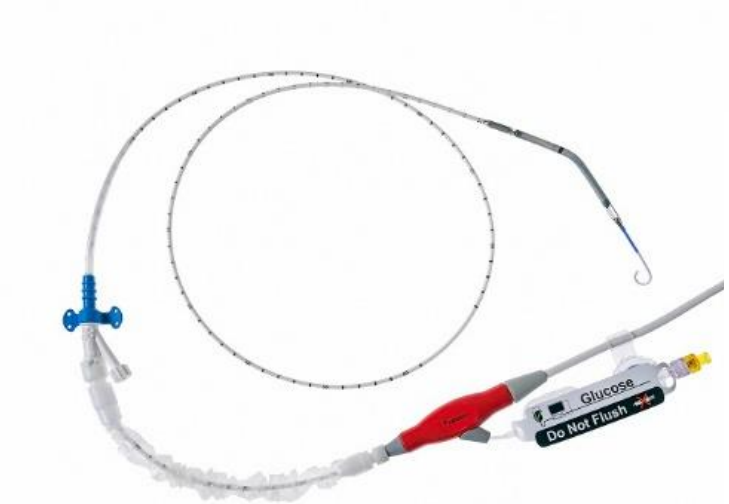


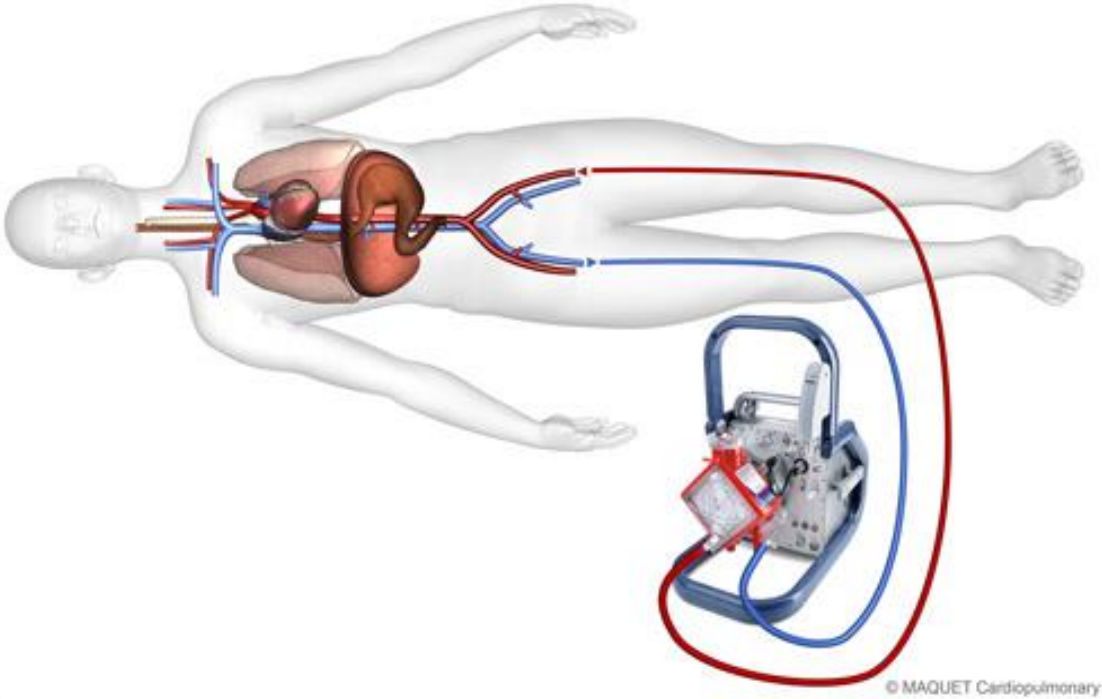
Mean arterial pressure

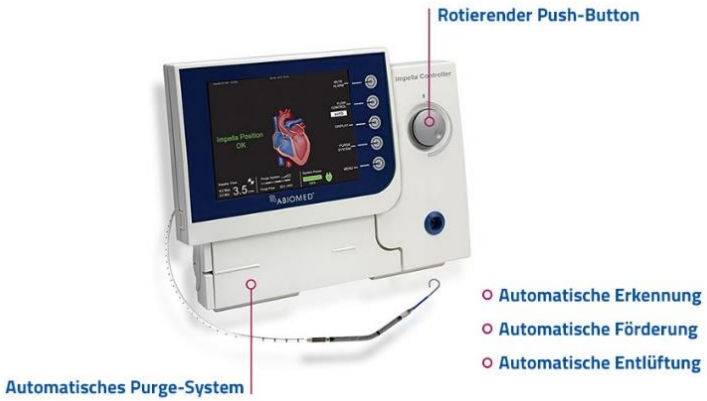


Thiele and de Waha et al EHJ 2017

Am häufigsten verwendete Mechanical Circulatory Support Devices (MCS)

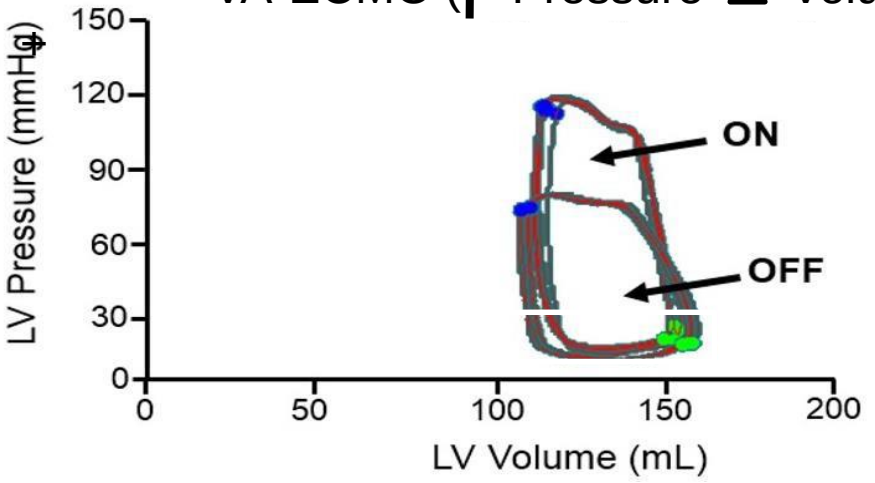




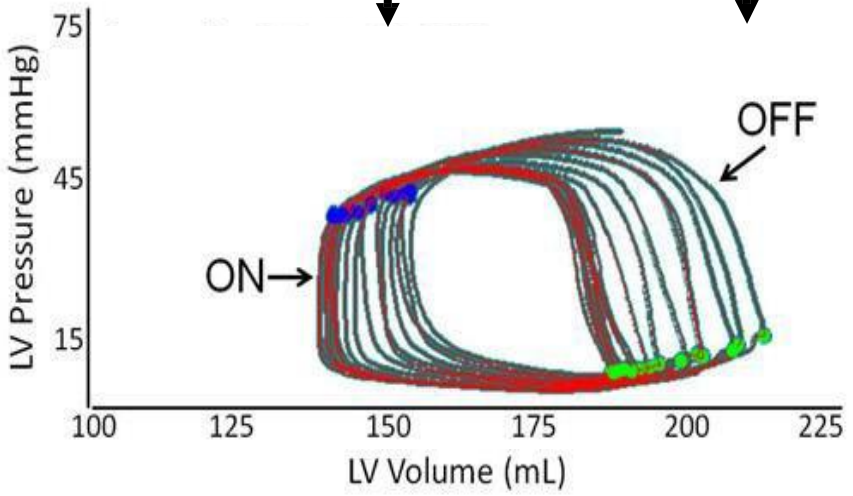


Vorteile/Unterschiede ECLS/IMPELLA

VA-ECMO (↑ Pressure Δ Volume)



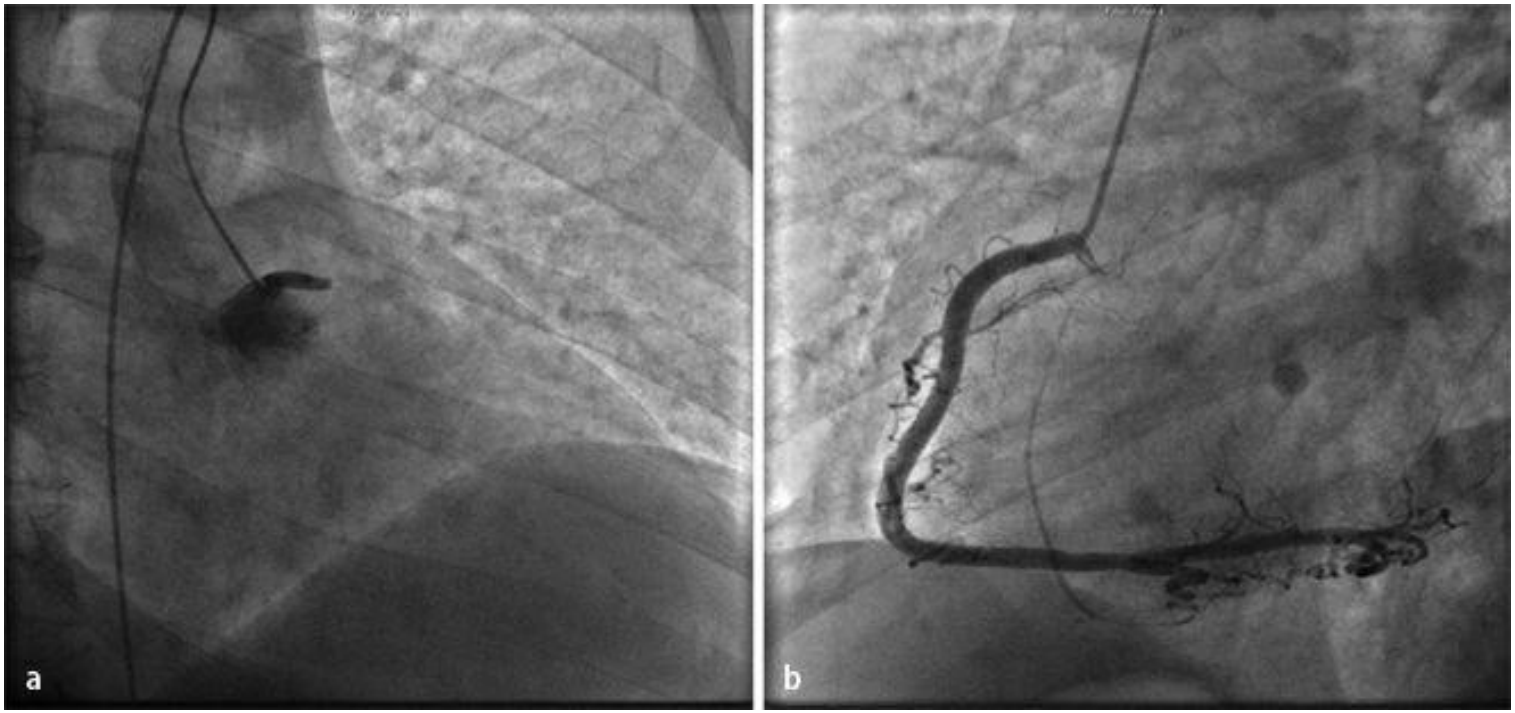
IMPELLA (Pressure ↓ Δ Volume ↓)



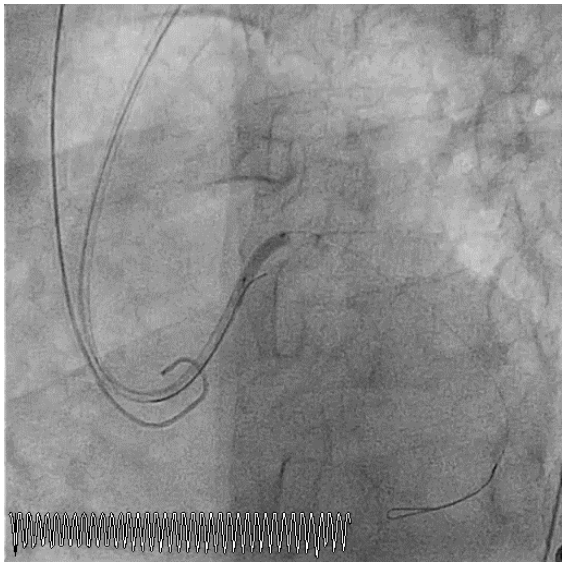
Therapie des Kardiogenen Schocks
Schnellstmögliche Behebung reversibler Ursachen

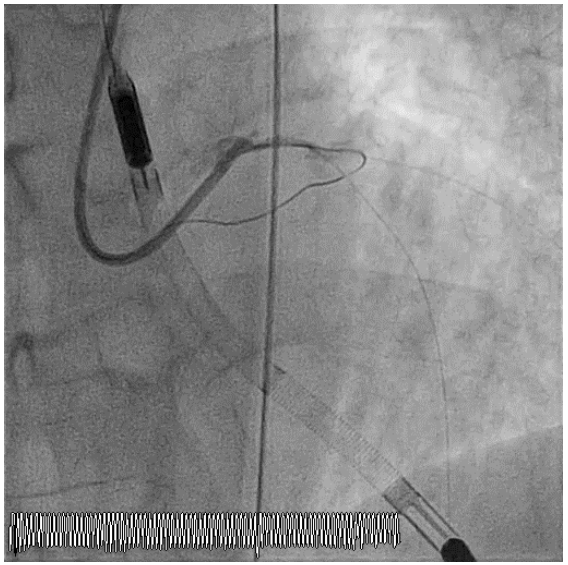
- In 70-80 % aller Fälle wird der kardiogene Schock durch eine akute myokardiale Ischämie verursacht!
- 5-10% der akuten Myokardinfarkte führen zu einem kardiogenen Schock!

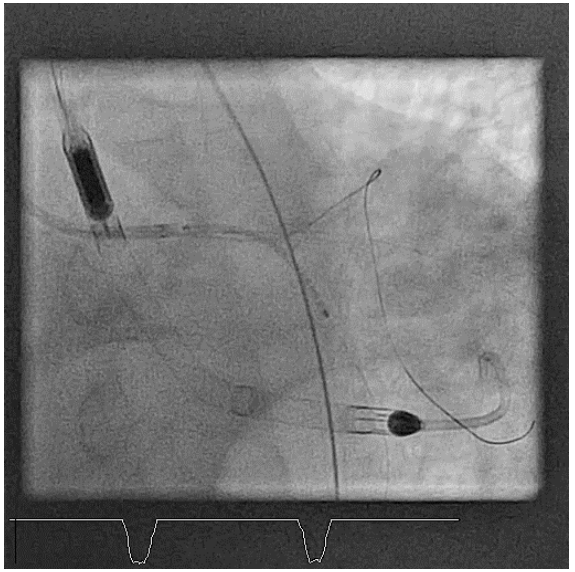
Fallbeispiel

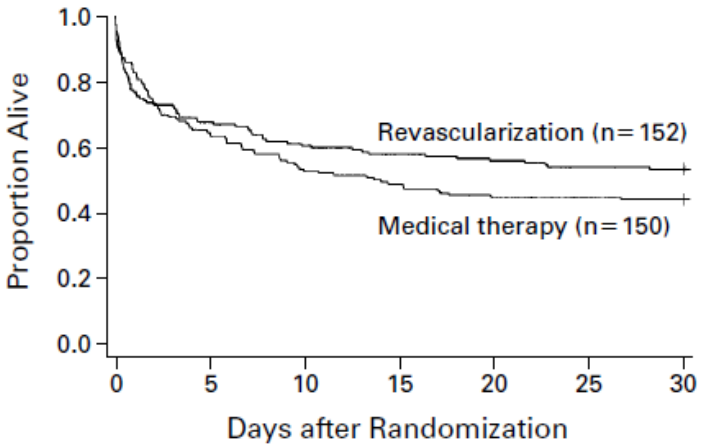


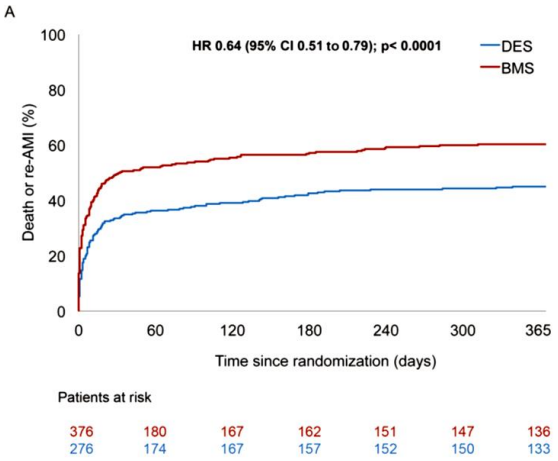
Schnellstmögliche Behebung reversibler Ursachen

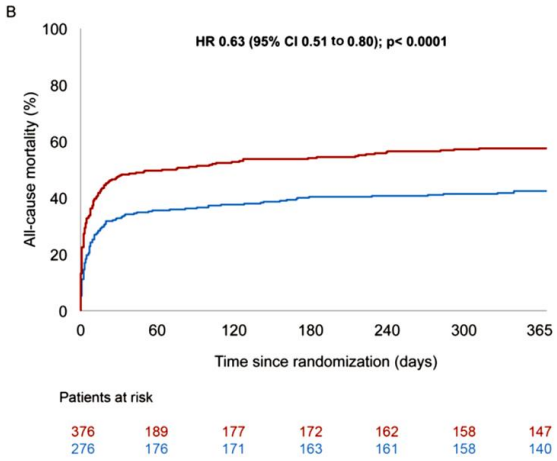






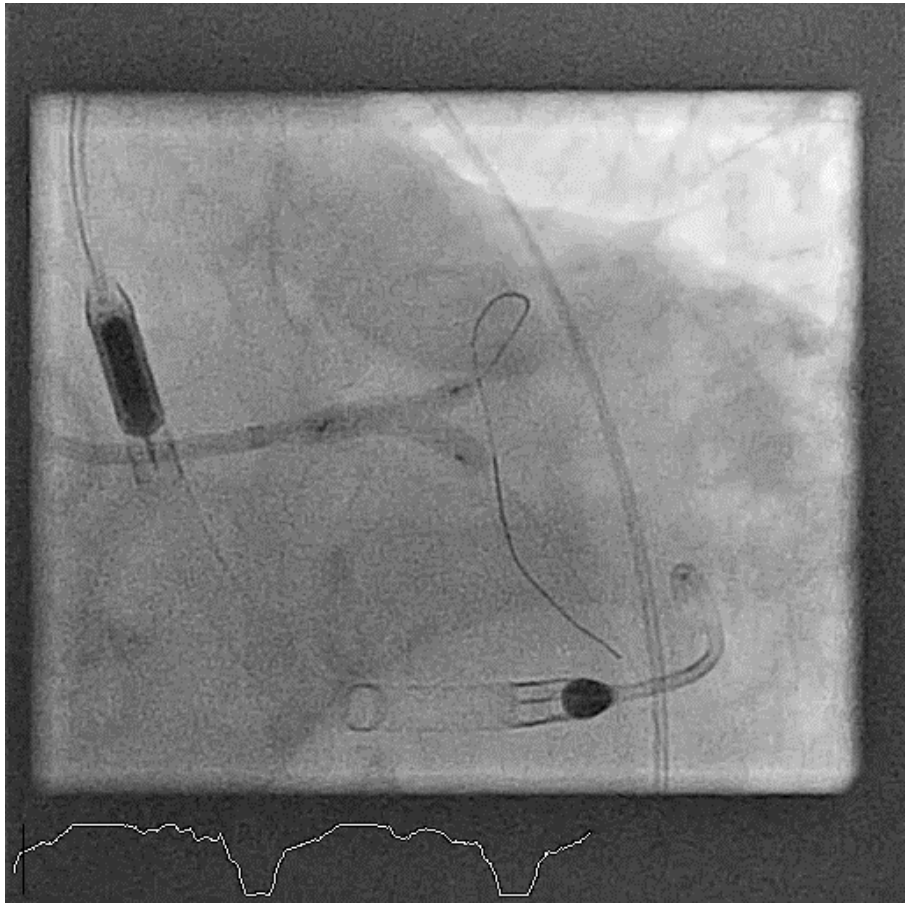


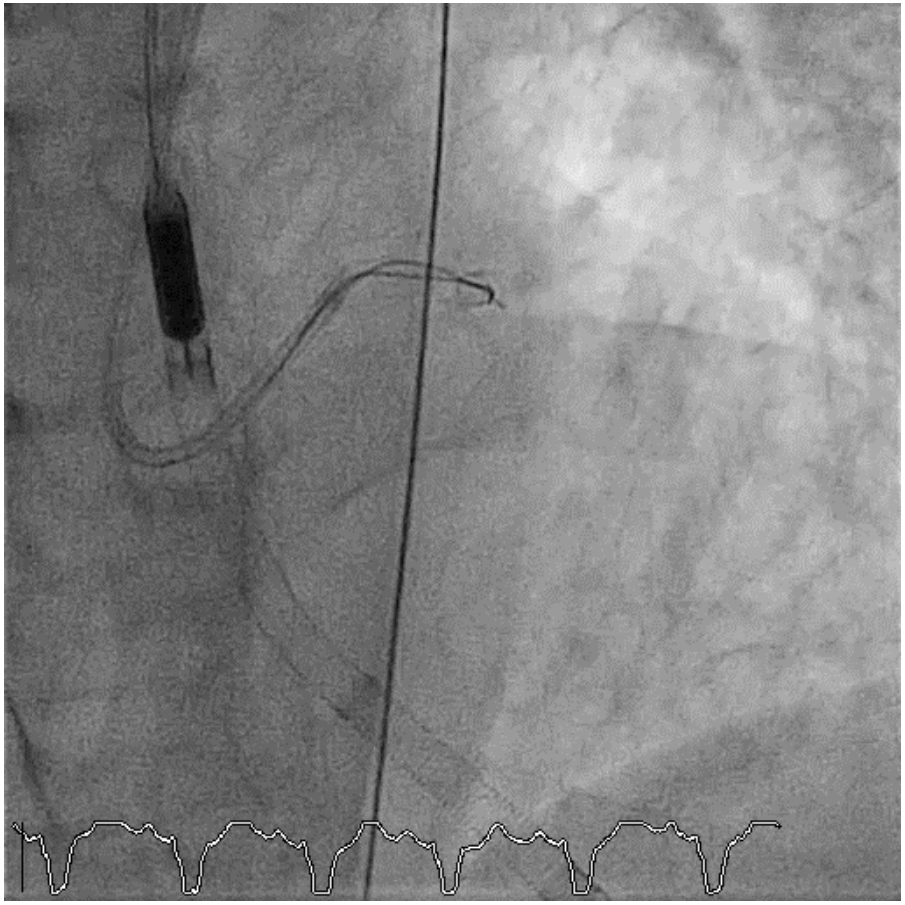




Hochmann JS et al N Engl J Med 1999; 341:625-634

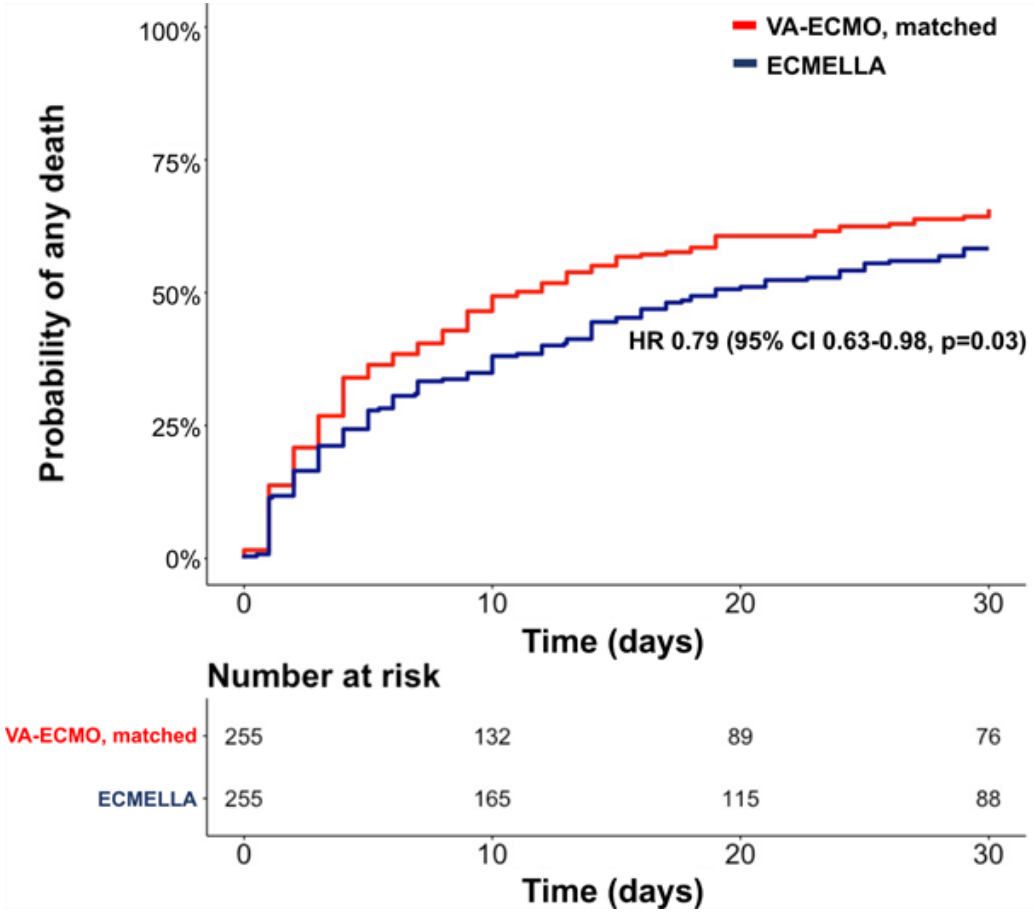
Ledwoch J, Fuernau G, Desch S, et al.Heart 2017;**103**:1177–1184





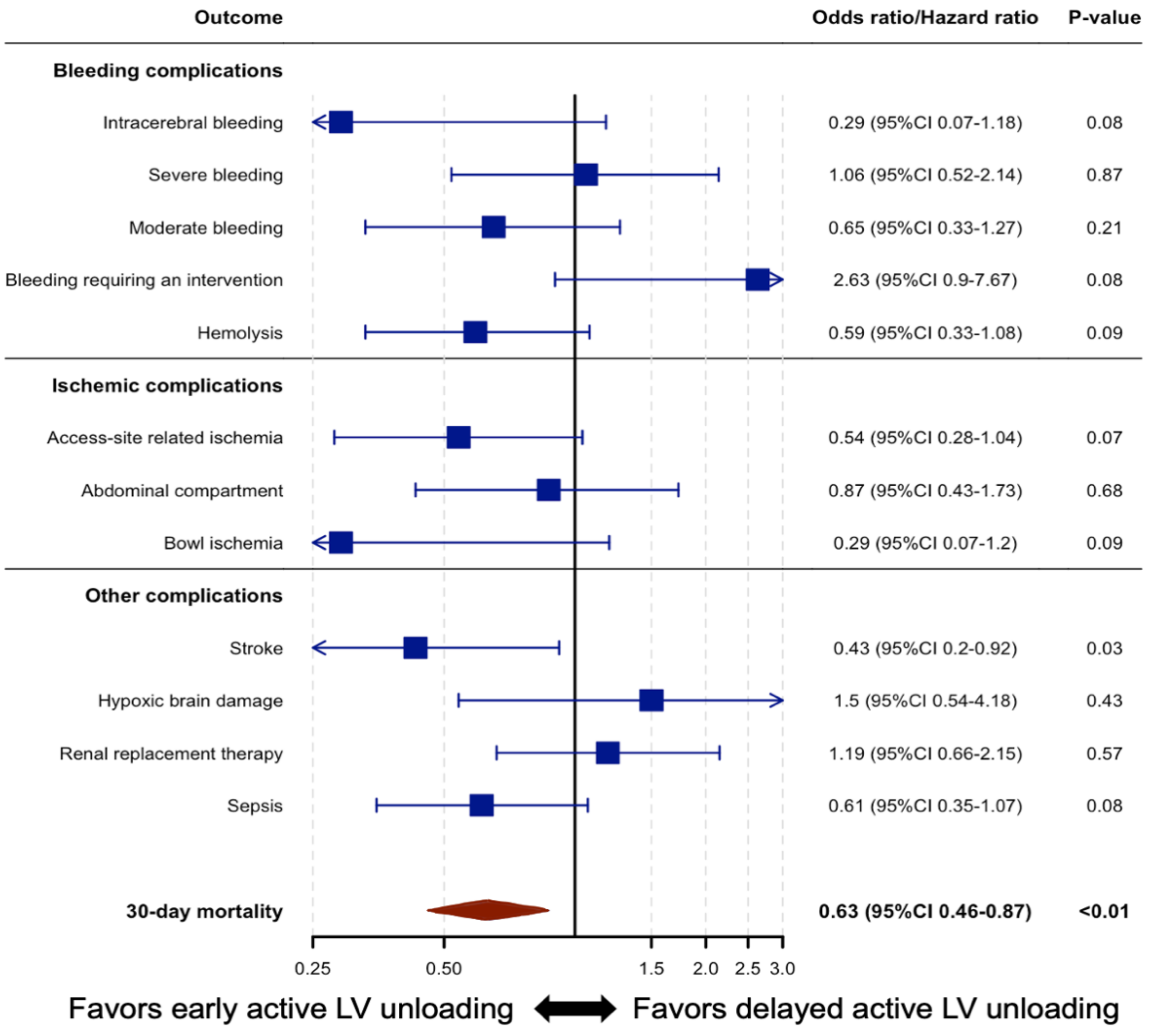
- **Postinterventionell persistierender Schock und Lungenödem, Eskalation um VA ECMO**
- **4 Tage in Folge Hs TnT >100.000pg/ml**
- **Protrahiertes MODS mit Exitus letalis**

ECLS und Impella („ECMELLA“) im Infarktbedingten CS



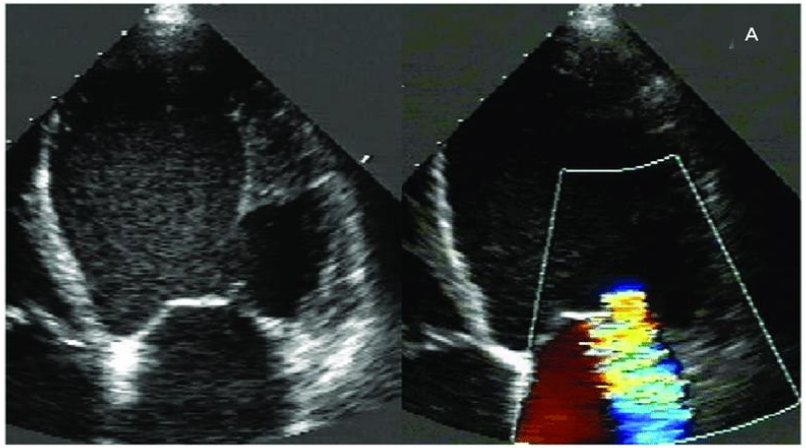
Schrage, Eden, Westermann et al Circulation. 2020;142:2095–2106

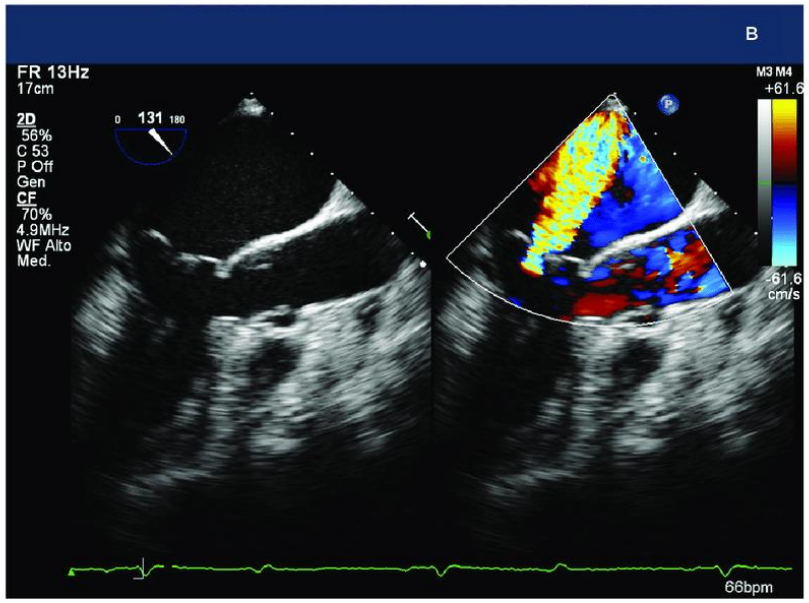
Timing of active left ventricular unloading in patients with cardiogenic shock on veno-arterial extracorporeal membrane oxygenation therapy.



Schrage, …., Eden, Frey, …. Westermann et al JACC 2023

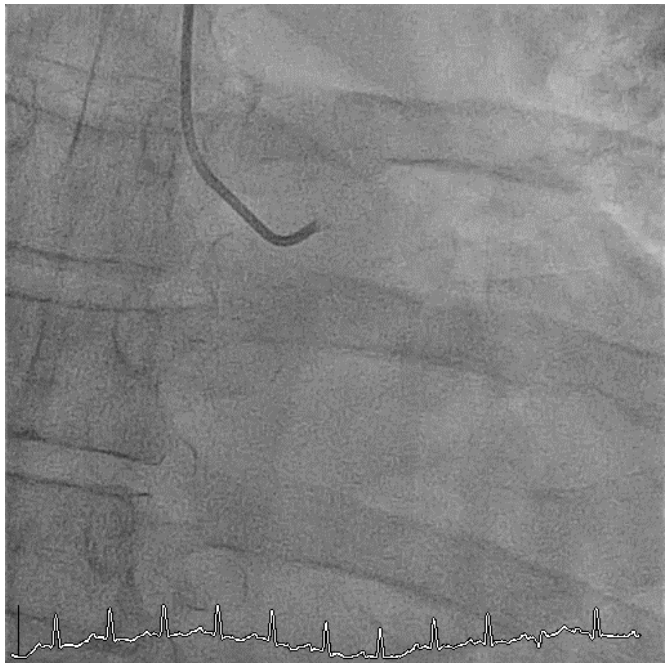
Schnellstmögliche Behebung reversibler Ursachen

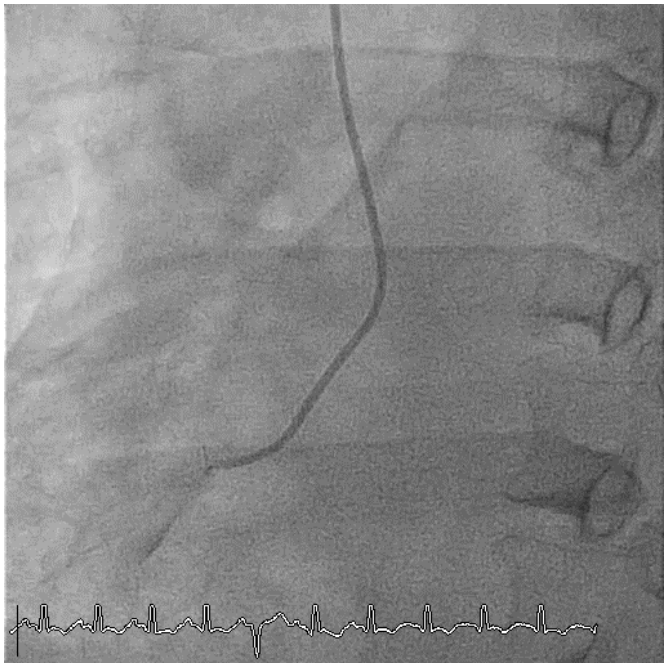




Schnellstmögliche Behebung reversibler Ursachen

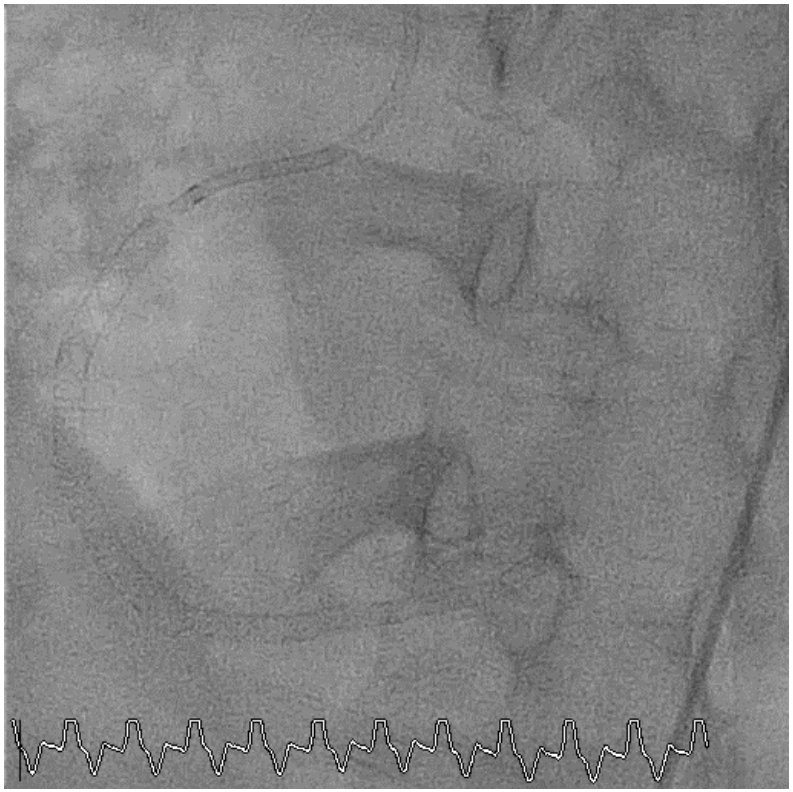
Fallbeispiel 2





Schnellstmögliche Behebung reversibler Ursachen

Fallbeispiel 2



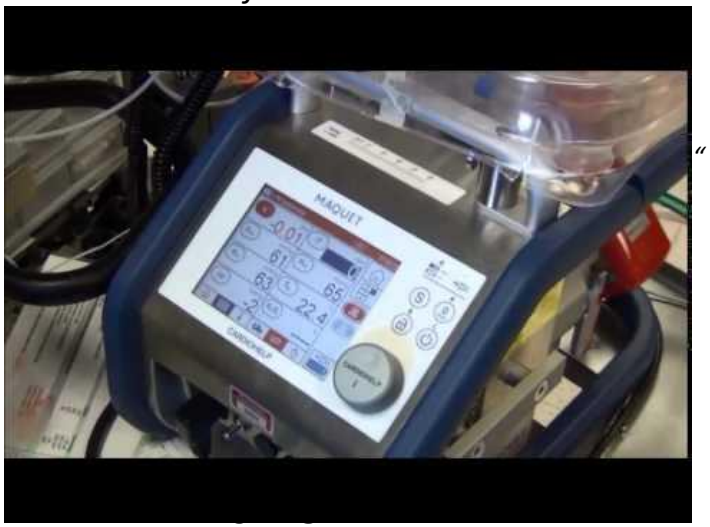
Schnellstmögliche Behebung reversibler Ursachen

Fallbeispiel 1

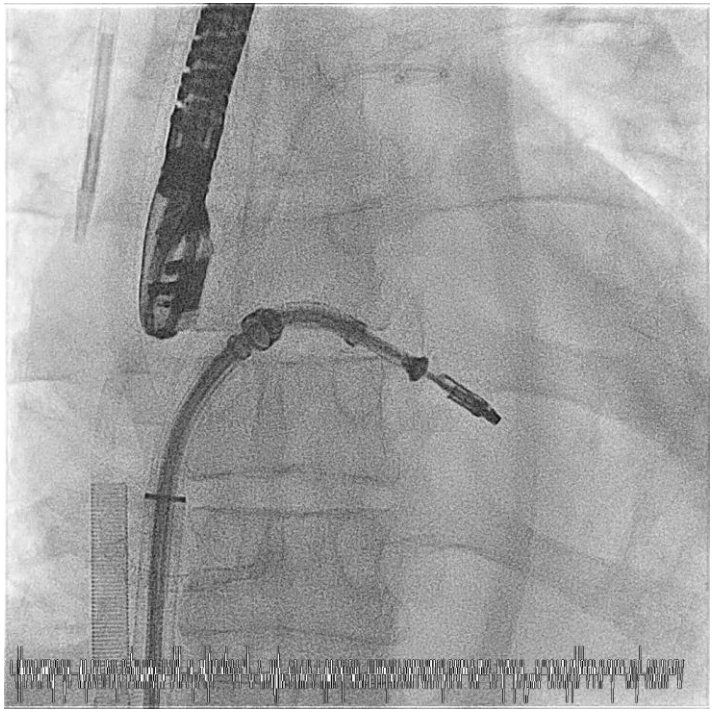
„Fast Entry“

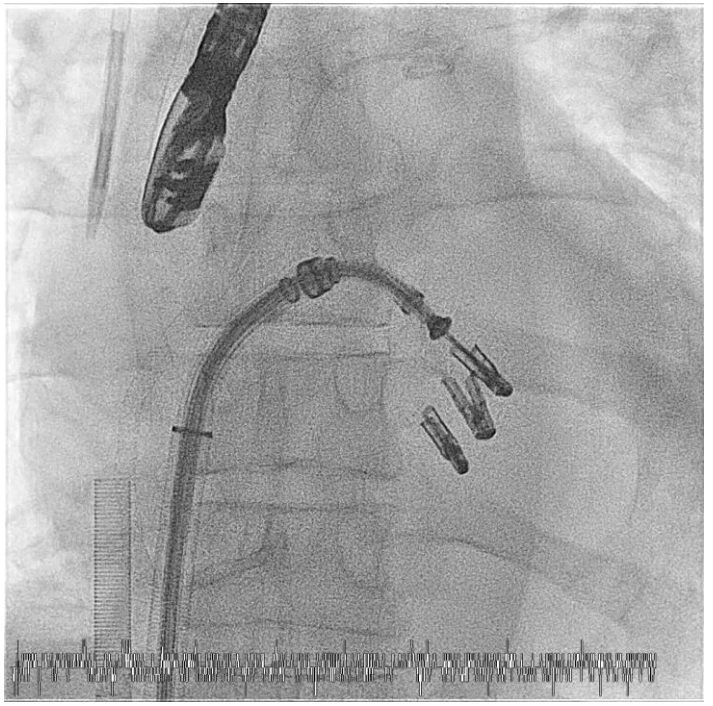
ECPR

ECLS (VA ECMO)
Geprimetes Gerät im BZ
Extrahospitale Kanülierung (MIC)
Prüfe Checkliste ECPR

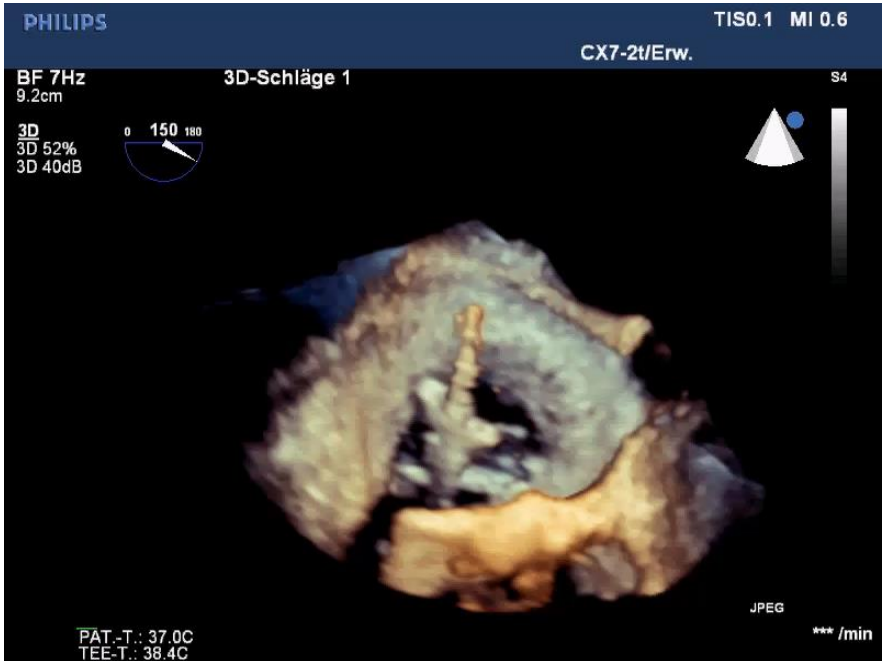


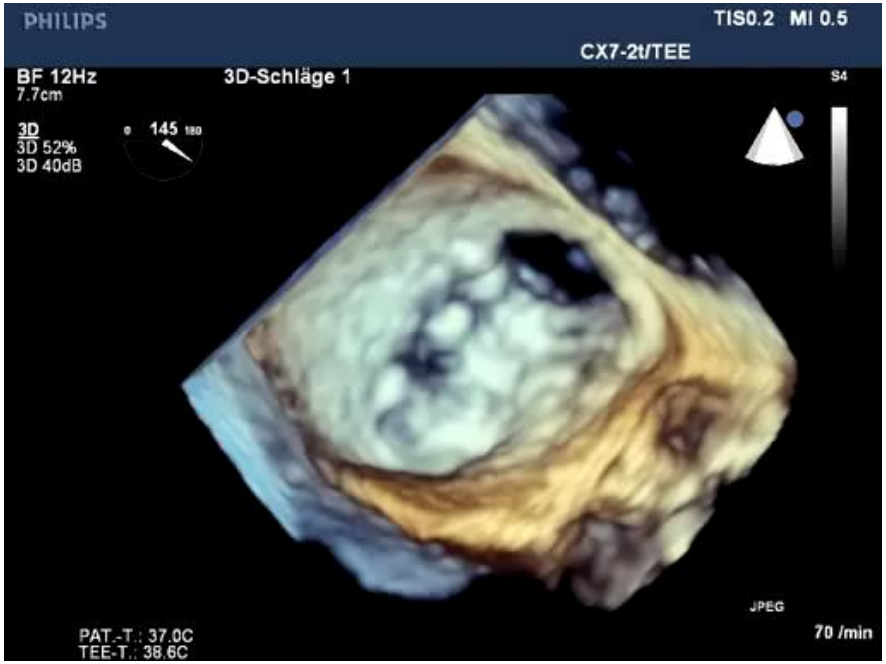
Schnellstmögliche Behebung reversibler Ursachen



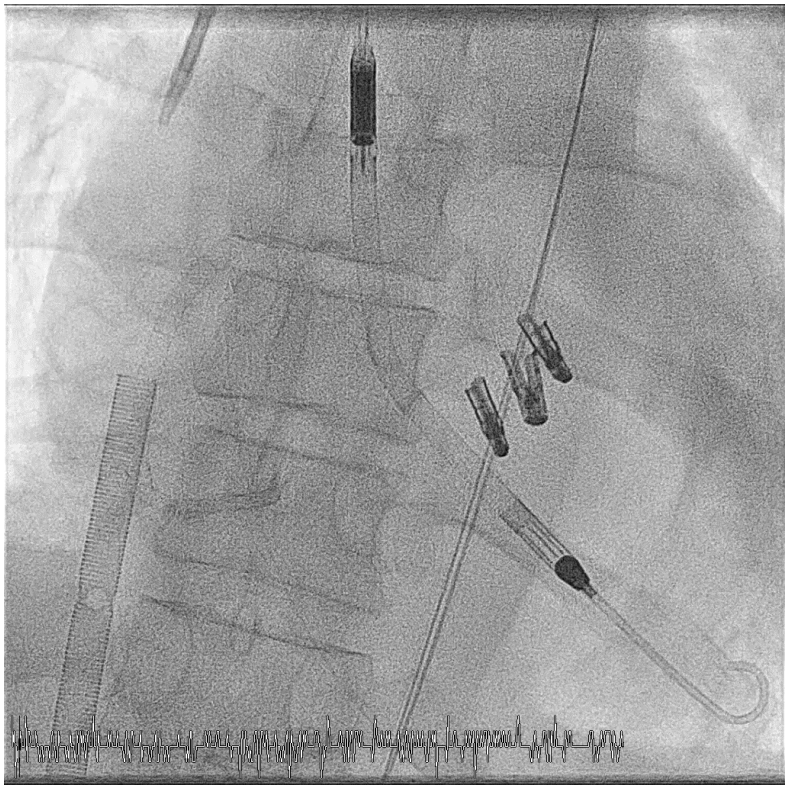


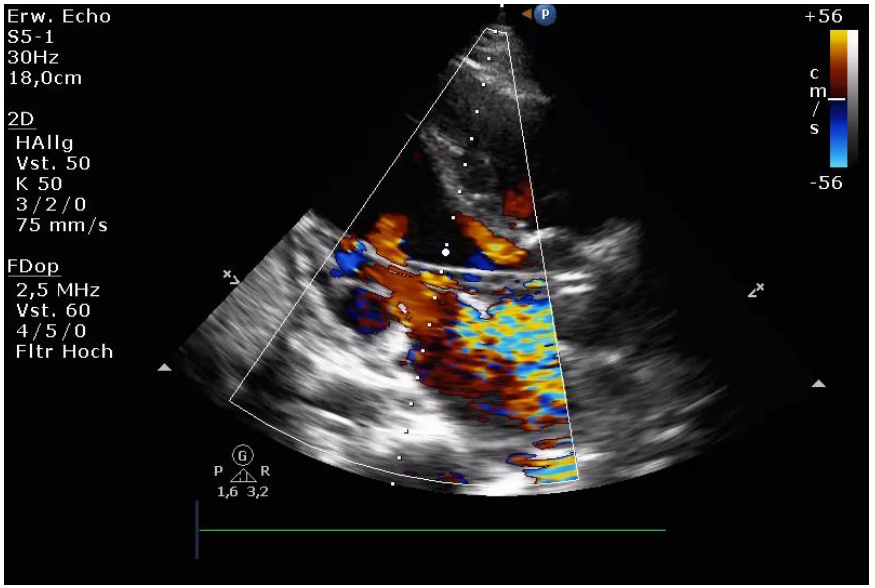
Schnellstmögliche Behebung reversibler Ursachen





Schnellstmögliche Behebung reversibler Ursachen

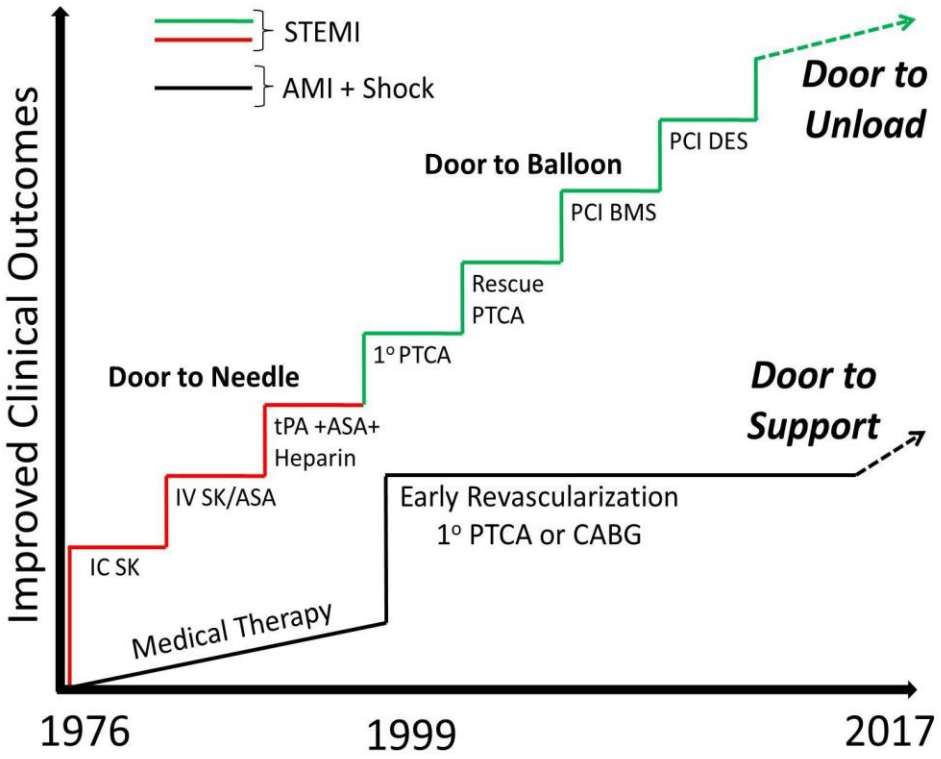




Patient als Fussgänger entlassen

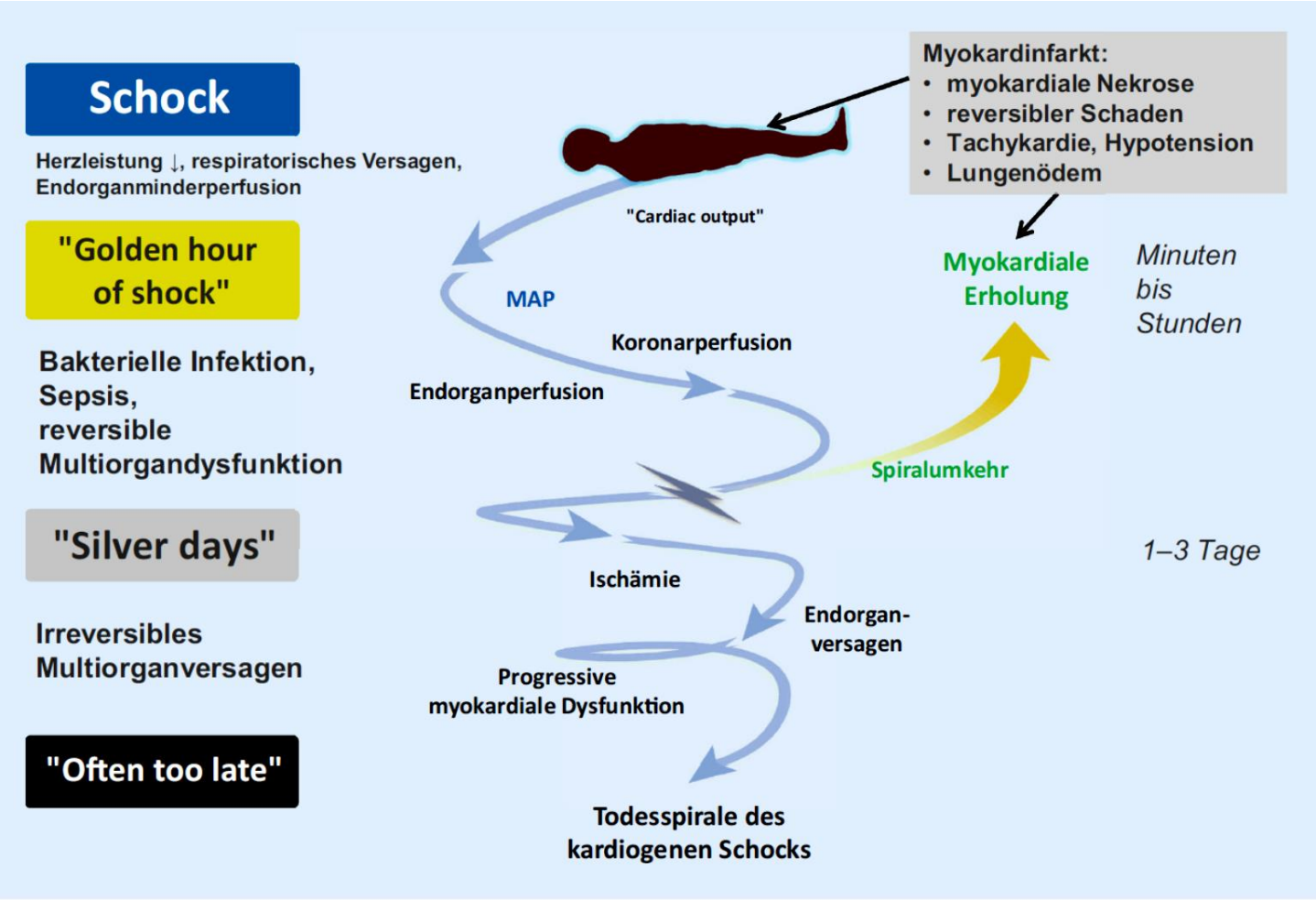
Was ist der ideale Zeitpunkt zur Unterstützung

„Door to support concept“



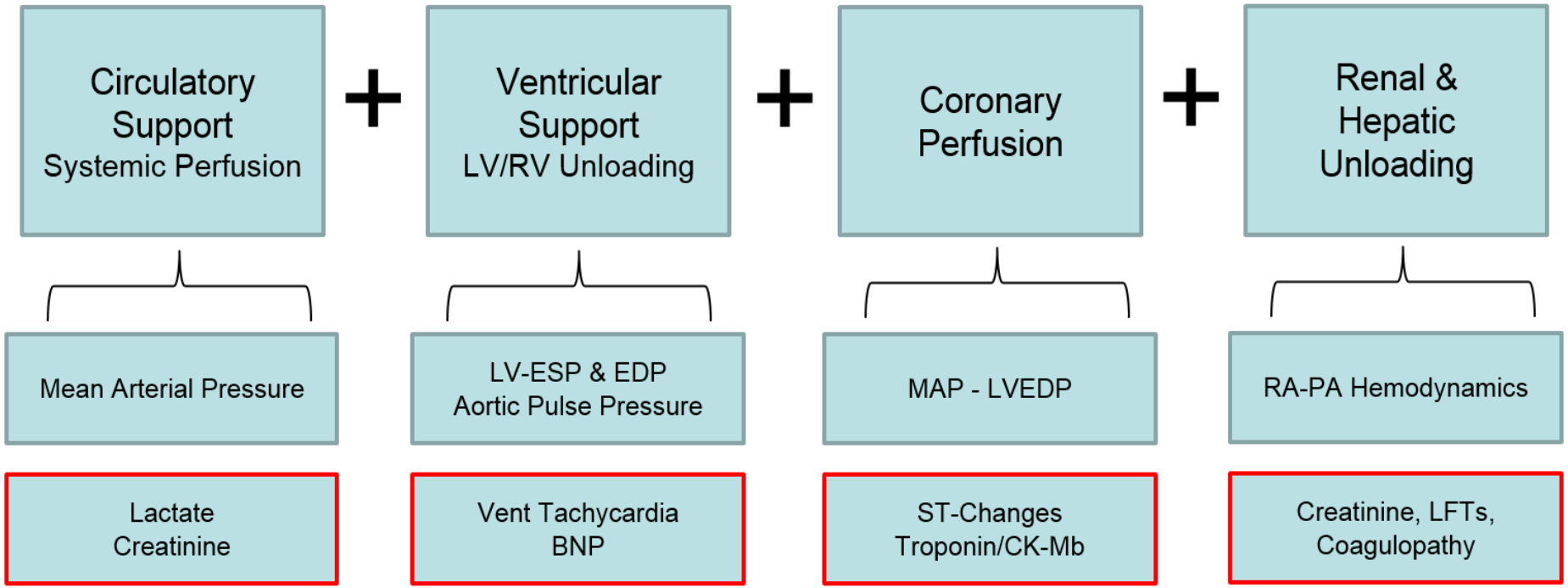
Kapur and Davila Eur H J 2017

Der kardiogenen Schock: Eine zu durchbrechende Abwärtsspirale



Herz 2017 · 42:18–26

The Hemodynamic Support Equation for Acute MCS
From Arithmetic to Calculus



Hemodynamic Problem

Hemo-Metabolic Problem

Recovery — Time in Cardiogenic Shock — Death

Bridge to Recovery
Detroit Cardiogenic Shock Initiative

It's Too Late for AMCS
Impress Trial

LVAD Versorgung im akuten kardiogenen Schock?

HVAD®, 1

Heart Mate II™, 2

Heart Mate 3™, 2

INTERMACS – Estimated 1-Year Survival

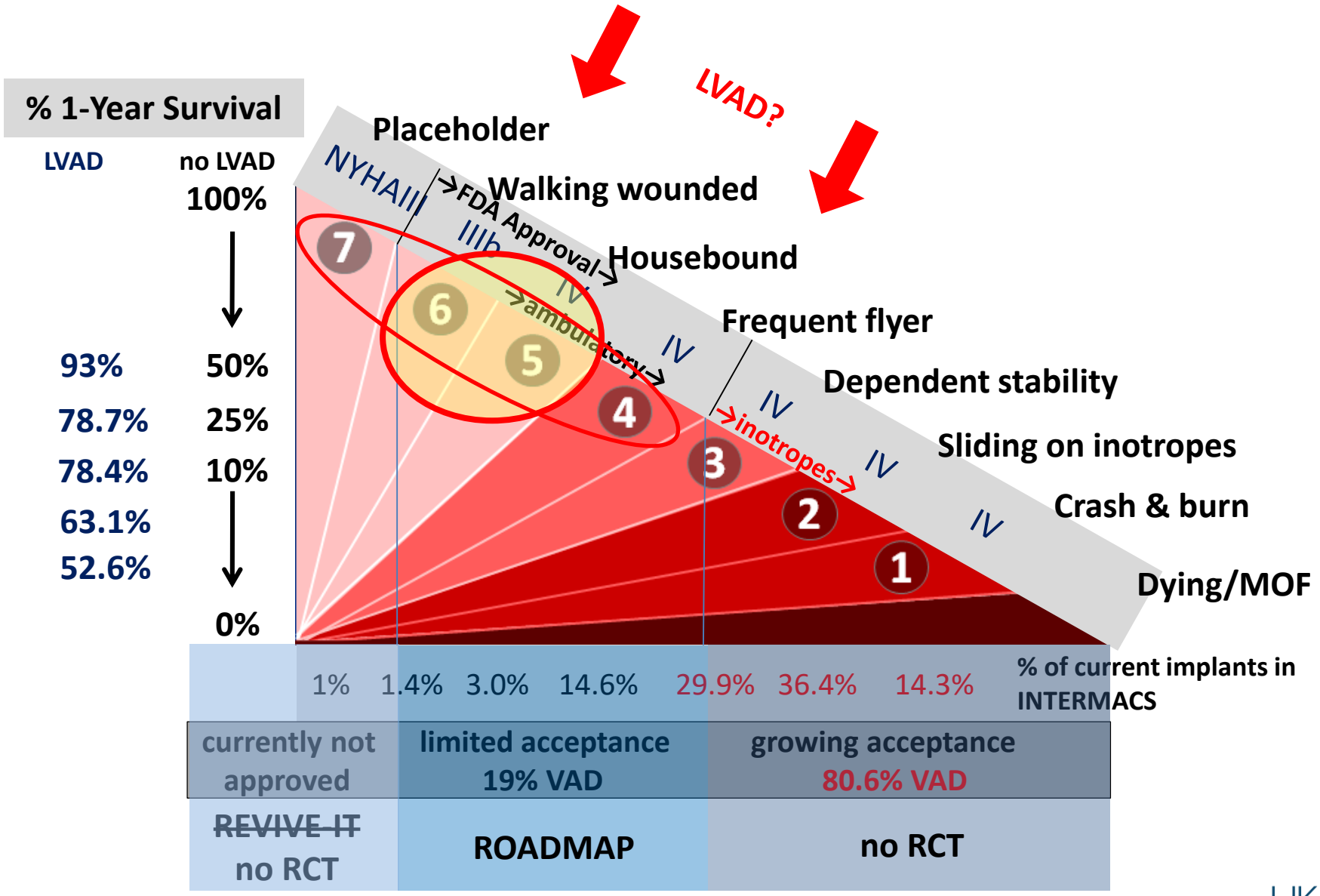
Table 2 INTERMACS levels and outcomes

Level	Description	Number of implants	Number of deaths	Estimated 1-year survival (%)
1	Critical cardiogenic shock	481	121	65
2	Progressive decline	514	102	72
3	Stable but inotrope-dependent	172	20	82
4	Recurrent advanced HF	116	16	75
5	Exertion intolerant	78	16	72
6	Exertion limited	78	16	72
7	Advanced NYHA III	78	16	72
	Overall	1361	275	73

Data includes all types of MCS, including LVAD, RVAD, BIVAD, and TAH.
Implant dates 23 June 2006 to 31 March 2009
Follow-up presumed until 31 March 2009
One-year survival is estimated from Kaplan-Meler curves available in reference.[20]
NYHA, New York Heart Association.

European Journal of Heart Failure (2010) **12**, 434-443 doi: 10.1093/eurjhf/hfq006

VAD Therapy and INTERMACS Levels

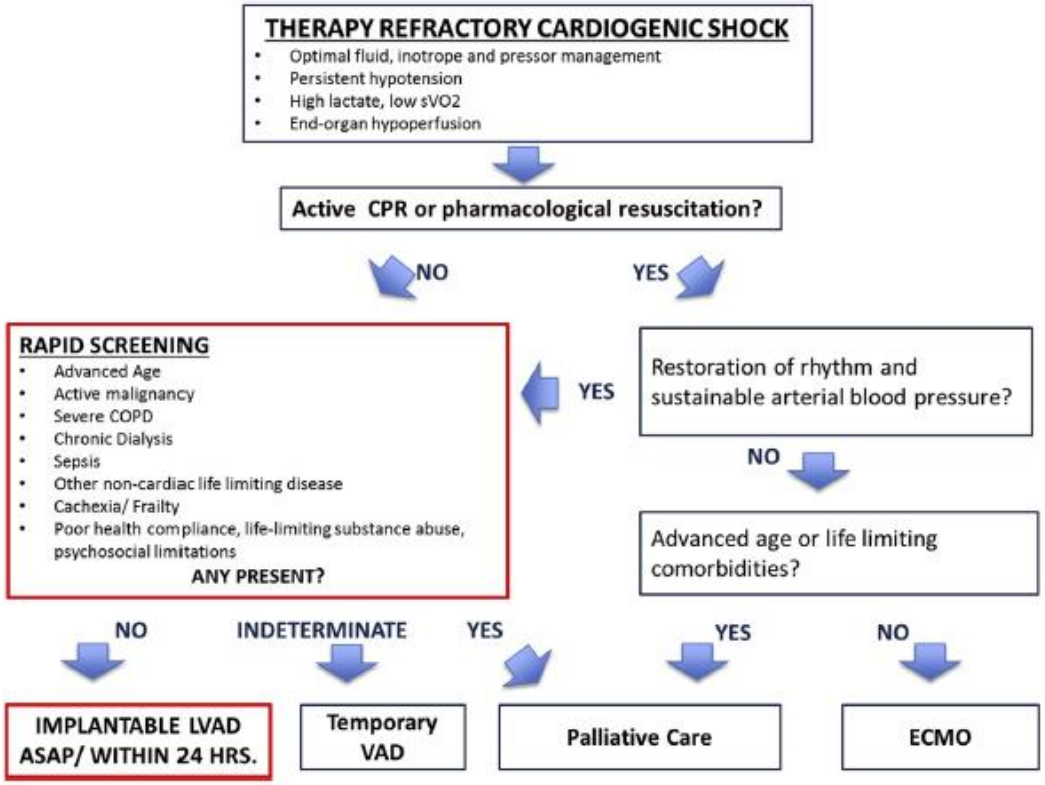


Selective implantation of durable left ventricular assist devices as primary therapy for refractory cardiogenic shock

Amit Pawale, MD,[a] Yosef Schwartz, BS,[a] Shinobu Itagaki, MD,[a] Sean Pinney, MD,[b] David H. Adams, MD,[a] and Anelechi C. Anyanwu, MD[a]

TABLE 1. Patient demographics

Age (y)	54.9 ± 10.8
Males	38 (88)
Acute MI	21 (49)
Decompensated heart failure	22 (51)
CPR within 24 h before operation	8 (18.6)
Unknown neurologic status	5 (11.6)
Preoperative mechanical ventilation	25 (58.1)
IABP	32 (74.4)
Percutaneous LVAD	7 (16.3)
Potent antiplatelet therapy within 48 h	16 (40)
Previous sternotomy	5 (11.6)
Creatinine (mg/dL)	2.0 ± 1.3
Bilirubin (mg/dL)	2.7 ± 3.4
Aspartate aminotransferase (U/L)	934 ± 2896
Lactate (mmol/L)	3.1 ± 3.0
Pulmonary capillary wedge pressure (mm Hg)	28.2 ± 6.8
Right atrial pressure (mm Hg)	19.4 ± 9.9
Pulmonary artery systolic pressure (mm Hg)	49.7 ± 11.2



J Thorac Cardiovasc Surg 2018;155:1059-68

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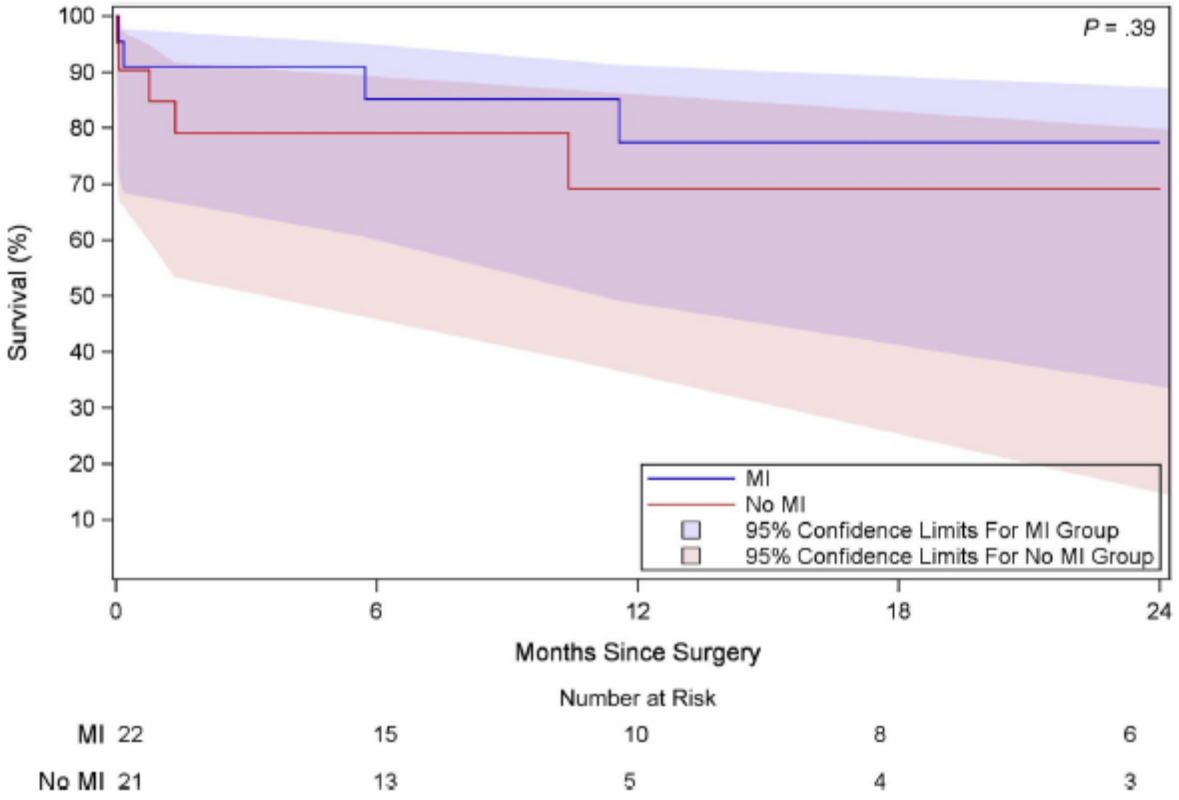

FIGURE 2. One-year survival stratified by myocardial infarction (*MI*) versus non-MI.

J Thorac Cardiovasc Surg 2018;155:1059-68

Outcomes of Hospitalizations for Cardiogenic Shock at Left Ventricular Assist Device Versus Non–Left Ventricular Assist Device Centers

Joseph I. Wang, MD; Daniel Y. Lu, MD; MHS; Dmitriy N. Feldman, MD; Stephen A. McCullough, MD; Parag Goyal, MD; Maria G. Karas, MD; Irina Sobol, MD; Evelyn M. Horn, MD; Luke K. Kim, MD; Udhay Krishnan, MD

Variable	Overall (n=272 075), %	LVAD Hospital (n=70 685), %	Non-LVAD Hospital (n=201 390), %	*P* Value	Standardized Difference, %
Presentation					
Non–AMI-CS	52.5	62.3	49.0	<0.001	27.0
AMI-CS	47.5	37.7	51.0	<0.001	27.0
Procedures					
CPR or intubated <24 h of admission	34.8	31.1	36.1	<0.001	10.4
IABP	18.8	18.7	18.8	0.847	0.3
Percutaneous support (Impella/TandemHeart)	2.1	2.6	1.9	<0.001	5.2
ECMO	1.3	4.3	0.2	<0.001	27.4
PCI or CABG	28.7	25.2	29.9	<0.001	10.3
CABG	8.7	10.7	8.0	<0.001	9.4
PCI	21.2	15.5	23.1	<0.001	19.4
Mechanical ventilation	53.6	51.5	54.3	<0.001	5.6
Pulmonary artery catheter	7.0	14.6	4.3	<0.001	35.4

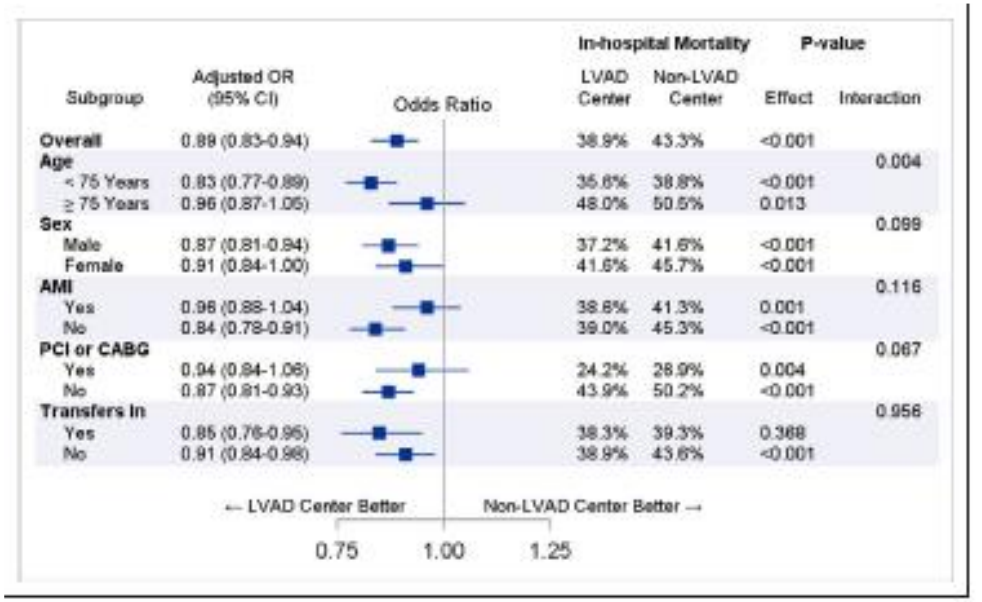

Figure 3. Association between left ventricular assist device (LVAD) centers vs non-LVAD centers and in-hospital mortality in cardiogenic shock.
AMI indicates acute myocardial infarction; CABG, coronary artery bypass grafting; OR, odds ratio; and PCI, percutaneous coronary intervention.

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Zusammenfassung:

- **Im Gegensatz zur chronischen Herzinsuffizienz ist die Therapie der akuten Herzinsuffizienz stagnierend**
- **Der kardiogene Schock (CS) eine immer noch konstant hohe Letalität**
- **Die schnellstmögliche Behandlung reversibler Ursachen ist ein entscheidendes Ziel**
- **Die Verhinderung eines MODS im CS ist das führende Ziel beim Einsatz von mechanischer Unterstützungssystemen**
- **Nur der frühe Einsatz temporärer mechanischer Unterstützungssysteme (MCS) kann ein MODS verhindern**

Danke !