

Azərbaycan
Kardiologiya
Cəmiyyəti

İleri Evre Kalp Yetersizliđi: inotrop bađımlı organ disfonksiyonu olan hastaya yaklaşım

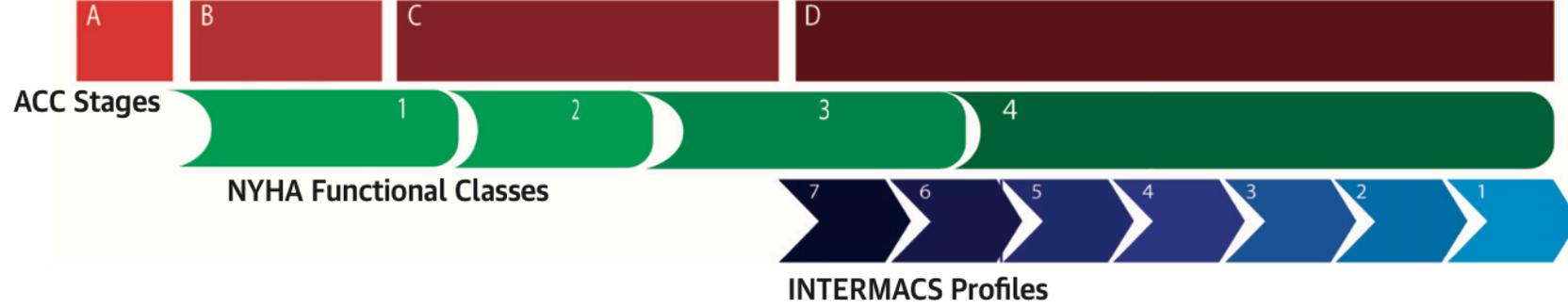
Prof Dr Sanem Nalbantgil, FESC, FHFA

Ege Üniversitesi Kardiyoloji AD

İzmir

2023 Haziran / Bakü

INTERMACS SINIFLAMASI



INTERMACS	1	2	3	4	5	6	7
	Kardiyojenik şok	Giderek kötüleşme	Stabil ama inotrop bağımlı	İstirahatte yakınmalar	Egzersiz intoleransı	Egzersiz kısıtlaması	İleri evre KY
	"Umutsuzlar"	"İnotropa rağmen kötüye gidenler"	"İnotrop bağımlı stabiller"	"Bir ayağı hastanede olanlar"	"Eve bağımlılar"	"Zor yürüyenler"	"Belirsizler"
<u>Hastanın tanımı</u>	İnotrop tedavi ve mekanik destek cihazlarına rağmen hayatı tehdit edici organ perfüzyon bozukluğu mevcuttur	İnotrop tedavi ile KB korunsa da, böbrek işlevleri, beslenme ve konjesyon bulgularında ilerleyici kötüleşme mevcuttur	Düşük-orta doz inotrop tedavi ile stabil olmakla birlikte, tedavi kesilmesiyle böbrek işlevleri, beslenme ve konjesyon bulgularında kötüleşme olur	İnotrop tedaviye ara verilebilse de, tekrarlayan belirti ve bulgularla sık hastaya başvurur	İstirahatte yakınmasız olmakla birlikte, efor yapamaz ve KY belirti ve bulguları kısmen devam etmektedir	Çabuk yorulmakla birlikte, hafif eforu yapabilir ve istirahatte konjesyon bulguları yoktur	Fonksiyonel kapasitesi NYHA III'dür ve yakın zamanda KY bulgu ve belirtileri tekrarlamamıştır
ACC/AHA sınıflaması	Evre D			Evre C			
NYHA sınıflaması ¹	Kardiyojenik Şok			Ambulatuvar IV	Ambulatuvar IV	IIIB	IIIA

Kardiyojenik Őok

- Kardiyojenik Őok, yeterli doluŐ basıncına karŐın hipotansiyon (sKB < 90 mmHg) ve hipoperfuzyon bulguları (oliguri, soĐuk ekstremiteler, deĐiŐen mental durum, laktat > 2 mmol/L, metabolic asidoz, SVo2 < %65) ile karakterize klinik tablodur
- Kardiyojenik Őok hemodinamik olarak kompleks bir sendrom olup çoĐu zaman çoklu organ yetmezliĐi ile sonuçlanan düşük kardiyak debi ile karakterizedir
- Klinik sonlanım kötü olup mortalite %40'ı aŐmaktadır

Kardiyojenik Şok: patofizyoloji

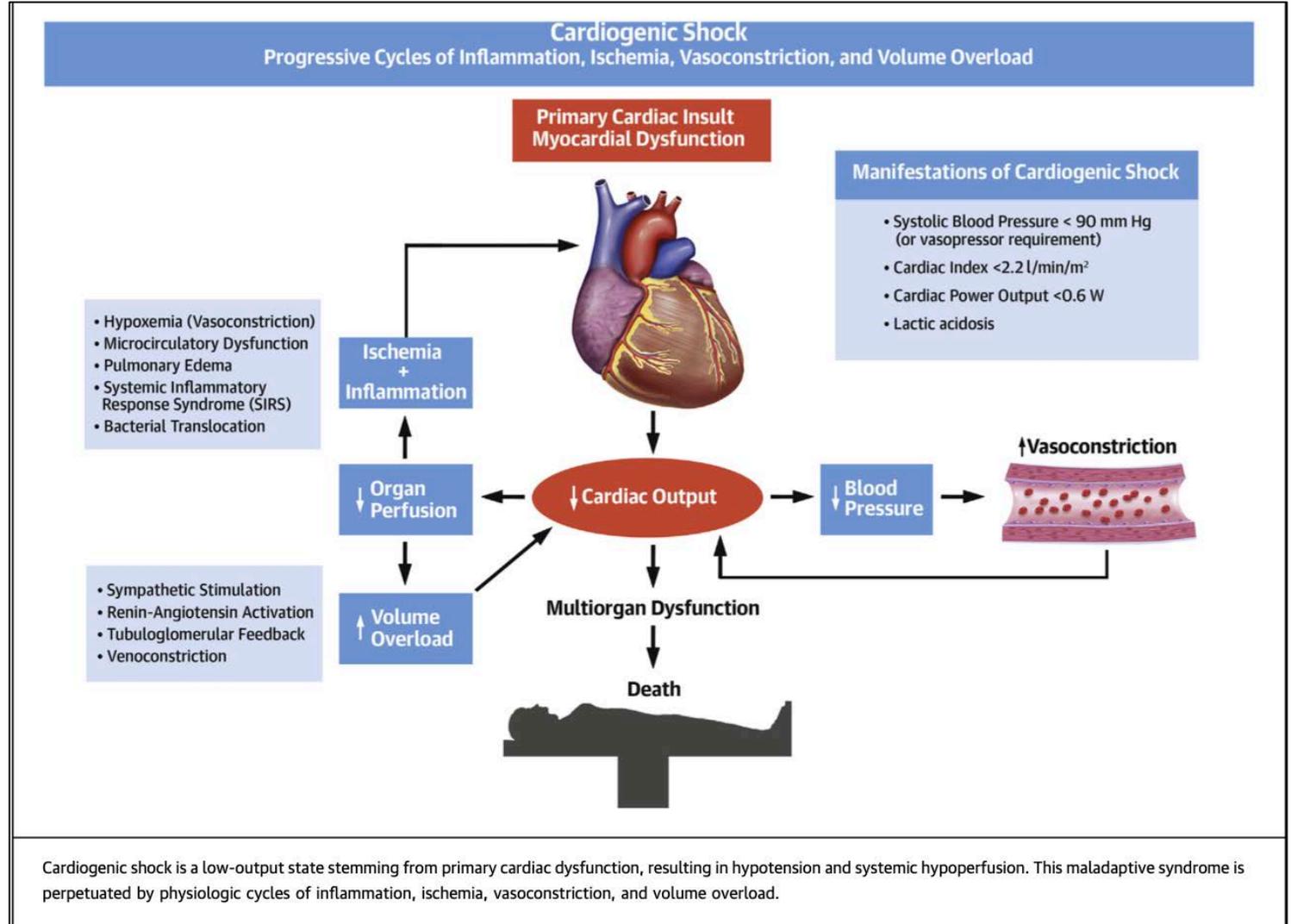
JACC: HEART FAILURE
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VOL. 8, NO. 11, 2020

MINI-FOCUS: HEART FAILURE AND CARADIOGENIC SHOCK

STATE-OF-THE-ART REVIEW

A Standardized and Comprehensive Approach to the Management of Cardiogenic Shock



Cardiogenic shock is a low-output state stemming from primary cardiac dysfunction, resulting in hypotension and systemic hypoperfusion. This maladaptive syndrome is perpetuated by physiologic cycles of inflammation, ischemia, vasoconstriction, and volume overload.

Kardiyojenik Őok: tedaviye yaklařım

- Etiyoloji
- Fenotipleme
- Evreleme
- Medikal Tedavi
- Mekanik Destek Cihazları



Tedaviye yaklařım için önemli



Etiyoloji:

Left ventricular failure

- Acute myocardial infarction
- Hypertrophic obstructive cardiomyopathy
- Myocarditis
- Myocardial contusion
- Peripartum cardiomyopathy
- Post-cardiotomy
- Progressive cardiomyopathy
- Septic cardiomyopathy
- Stress cardiomyopathy (takotsubo)
- Ventricular outflow obstruction

Right ventricular failure

- Acute myocardial infarction
- Myocarditis
- Post-cardiotomy
- Progressive cardiomyopathy
- Pulmonary embolism
- Septic cardiomyopathy
- Worsening pulmonary hypertension

Arrhythmia

- Atrial fibrillation or flutter
- Ventricular tachycardia or fibrillation
- Bradycardia or heart block

Pericardial disease

- Tamponade
- Progressive pericardial constriction

Chemotherapeutic, toxic, metabolic

- Calcium-channel antagonists
- Adrenergic receptor antagonists
- Thyroid disorders

Valvular or mechanical dysfunction

- Aortic regurgitation—acute bacterial endocarditis
- Mechanical valve dysfunction or thrombosis
- Mitral regurgitation—myocardial ischemia or infarction
- Progressive mitral stenosis
- Progressive aortic stenosis
- Ventricular septal defect or free wall rupture

Kardiyojenik Şok Profilleri...

		Volume Status	
		Dry	Wet
Peripheral Perfusion	Warm	Vasodilatory shock (not CS) Increased cardiac index, low SVRI, low/ normal PCWP	Mixed CS Low cardiac index, low / normal SVRI, Elevated PCWP
	Cold	Euvolemic CS Low Cardiac index, high SVRI, low / normal PCWP	Classic CS Low cardiac index, High SVRI, Elevated PCWP

Kardiyojenik Şok Profilleri...

ORIGINAL RESEARCH

Phenotyping Cardiogenic Shock

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1959 pts

-CSWG registry (MI and acute on chronic HF)

-Danish Retroshock MI registry

Table 2. Selection of Outstanding Characteristics of the Phenotypes

Characteristics	Cluster/Phenotype I <u>"Noncongested" CS</u>	Cluster/Phenotype II <u>"Cardiorenal" CS</u>	Cluster/Phenotype III <u>"Cardiometabolic" CS</u>
Mean age, y	≈60	≈70	≈65
Comorbidities	Few	DM2, CKD, hypertension...	Few
Blood pressure	↓	↓	↓↓
Congestion	None	Left ventricular	Right ventricular
Heart rate	↔	↔	↑↑
Hemoglobin	↔	↓	↔
Transaminases	↔	↔	↑↑
Lactate	↔ or ↑	↓	↑↑
Kidney function	↔	↓↓	↓

CKD indicates chronic kidney disease; CS, cardiogenic shock; and DM2, type 2 diabetes mellitus.

Kardiyojenik Şok Profilleri...

What Is New?

- Using an unbiased machine learning approach, we were able to identify 3 distinct cardiogenic shock (CS) clinical phenotypes ("noncongested," "cardiorenal," and "cardiometabolic" shock) with specific characteristics and associations with outcomes.
- These phenotypes were identified and validated in CS attributable to myocardial infarction as well as acute-on-chronic heart failure in 2 different data sets.
- Our data validate the clinical assumption that hemometabolic shock is associated with a higher mortality and stress the importance of renal function, systemic congestion, and metabolic failure for CS outcomes.

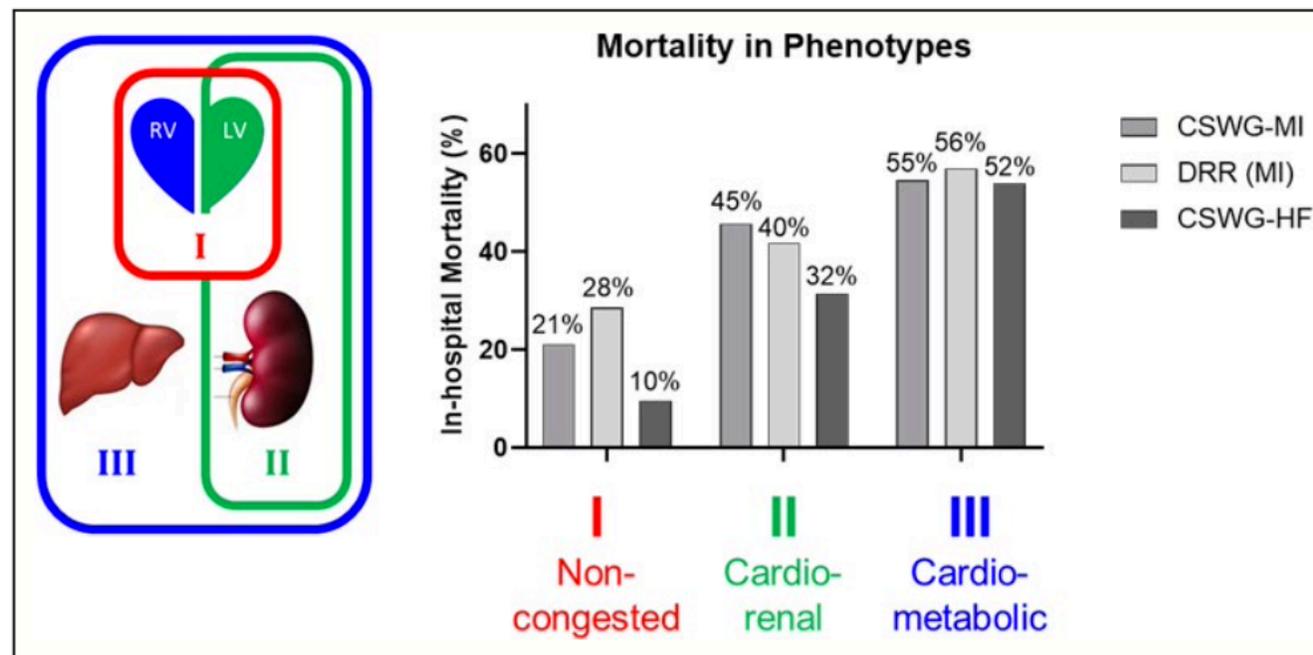


Figure 3. In-hospital mortality in the 3 distinct phenotypes of cardiogenic shock (CS).

Phenotype I (noncongested), phenotype II (cardiorenal), and phenotype III (cardiometabolic) are associated with in-hospital mortality across 2 international multicenter registries of CS attributable to acute myocardial infarction (MI) and a multicenter registry of CS attributable to acute-on-chronic heart failure. CSWG indicates Cardiogenic Shock Working Group Registry; and DRR, Danish Retrospective MI Registry.

Kardiyojenik Şok Evreleri...

Received: 23 April 2019 | Accepted: 24 April 2019
DOI: 10.1002/ccd.28329

CLINICAL DECISION MAKING

WILEY

SCAI clinical expert consensus statement on the classification of cardiogenic shock

This document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019

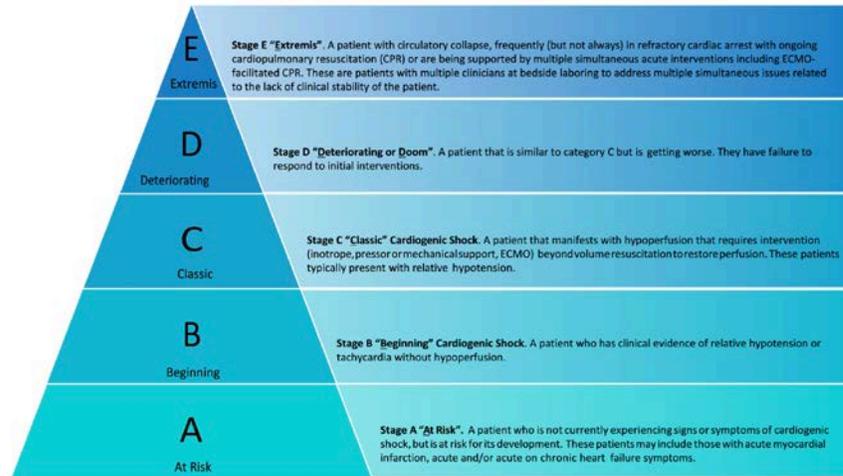


FIGURE 1 The pyramid of CS classification

SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies



This statement was endorsed by the American College of Cardiology (ACC), American College of Emergency Physicians (ACEP), American Heart Association (AHA), European Society of Cardiology (ESC) Association for Acute Cardiovascular Care (ACVC), International Society for Heart and Lung Transplantation (ISHLT), Society of Critical Care Medicine (SCCM), and Society of Thoracic Surgeons (STS) in December 2021.

TABLE 1 Characteristics of Studies Validating the Association Between the SCAI SHOCK Stage and Mortality

Study	Years Included	Population	Design	Patients, n	Primary Outcome
Schrage et al 2020 ^a	2009-2017	CS or large MI	Retrospective single-center	1007	30-day survival
Baran et al 2020	2019-2020	CS	Prospective single-center	166	30-day survival
Thayer et al 2020	2016-2019	CS	Prospective multicenter ^b	1414	In-hospital mortality
Hanson et al 2020	2016-2019	AMICS	Prospective multicenter ^b	300	Survival to discharge
Jentzer et al 2021 ^a	2007-2015	CS	Retrospective single-center	934	30-day survival
Jentzer et al 2019	2007-2015	CICU	Retrospective single-center	10,004	In-hospital mortality
Lawler et al 2021	2017-2019	CICU or CS	Retrospective multicenter	1991	In-hospital mortality
Jentzer et al 2020	2007-2015	CICU survivors	Retrospective single-center	9096	Postdischarge survival
Pareek et al 2020	2012-2017	OHCA	Retrospective single-center	393	30-day mortality

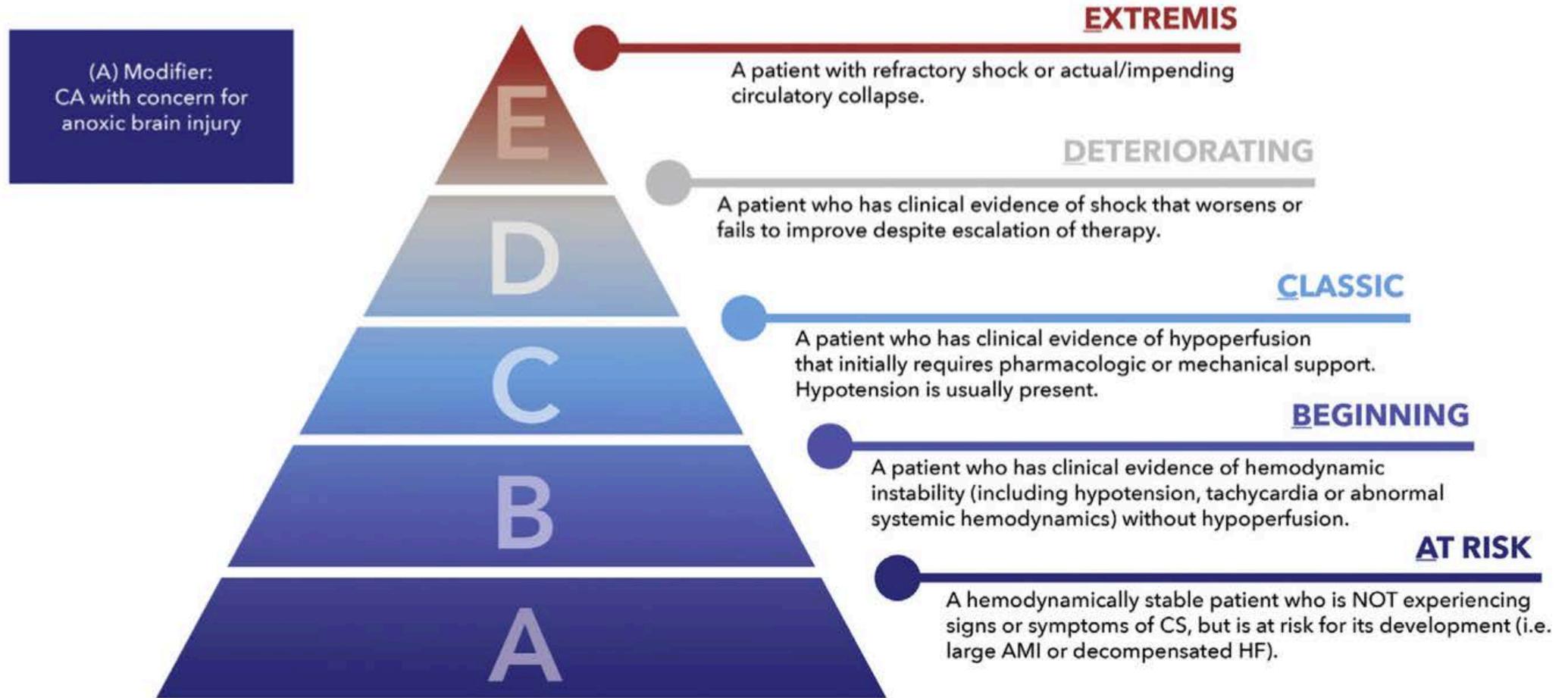
Duplicate data from the same cohort are not shown. AMICS = CS from acute myocardial infarction; CICU = cardiac intensive care unit; CS = cardiogenic shock; MI = myocardial infarction; OHCA = out-of-hospital cardiac arrest; SCAI = Society for Cardiovascular Angiography and Interventions.

^aPatients with CS from the Schrage 2020 study were included in the Jentzer 2021 study, so only the nonduplicated patients are reported for the Jentzer 2021 study.

^bPatient enrollment in these studies was prospective, but the SCAI SHOCK stage was assigned retrospectively.

JACC 2022:933-946

Kardiyojenik Şok Evreleri...



Stage	Description	Hemodynamics	Biochemical Markers
A At risk	No signs or symptoms of CS but at risk for CS development. May include patients with large acute myocardial infarction.	Normotensive (SBP \geq 100 or normal for patient) If hemodynamics done: - Cardiac index \geq 2.5 - CVP < 10 - PA sat \geq 65%	Normal labs - Normal renal function - Normal lactic acid
B Beginning CS	A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.	SBP <90 or MAP <60 or >30 mmHg drop from baseline. - Pulse \geq 100 - If hemodynamics done - Cardiac index \geq 2.2 - PA sat \geq 65%	- Normal lactate - Minimal renal function impairment - Elevated BNP
C Classic CS	A patient that manifests with hypoperfusion that requires intervention (inotrope, pressor, or mechanical support, including ECMO) beyond volume resuscitation to restore perfusion. These patients typically present with relative hypotension.	May include any of: SBP <90 or MAP <60 or >30 mmHg drop from baseline and drugs/device used to maintain BP above these targets Hemodynamics: - Cardiac index < 2.2 - PCWP >15 - RAP/PCWP \geq 0.8 - PAPI < 1.85 - Cardiac power output \leq 0.6	May include any of the following: - Lactate \geq 2 - Creatinine doubling OR >50% drop in GFR - Increased LFTs - Elevated BNP
D Deteriorating	A patient that is similar to category C but is getting worse. They have failure to respond to initial interventions.	Any of Stage C and : Requiring multiple pressors OR addition of mechanical circulatory support devices to maintain perfusion	Any of Stage C and : Deteriorating
E Extrimis	A patient that is experiencing cardiac arrest with ongoing CPR and/or ECMO being supported by multiple interventions.	No SBP without resuscitation PEA or refractory VT/VF hypotension despite maximal support	"Trying to die" - CPR (A-modifier) - pH \leq 7.2 - Lactate \geq 5

Epidemiology, pathophysiology and contemporary management of cardiogenic shock – a position statement from the Heart Failure Association of the European Society of Cardiology

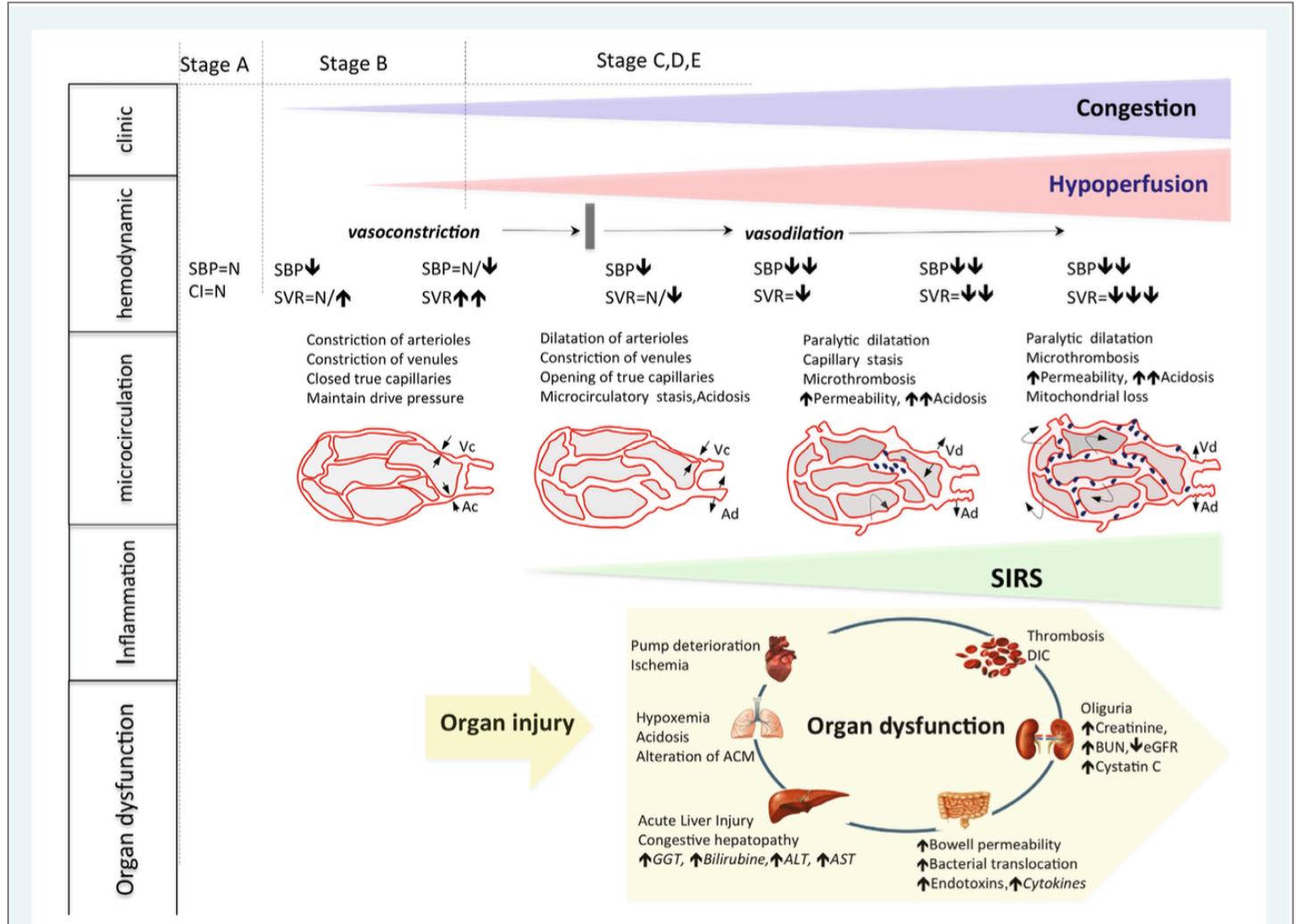
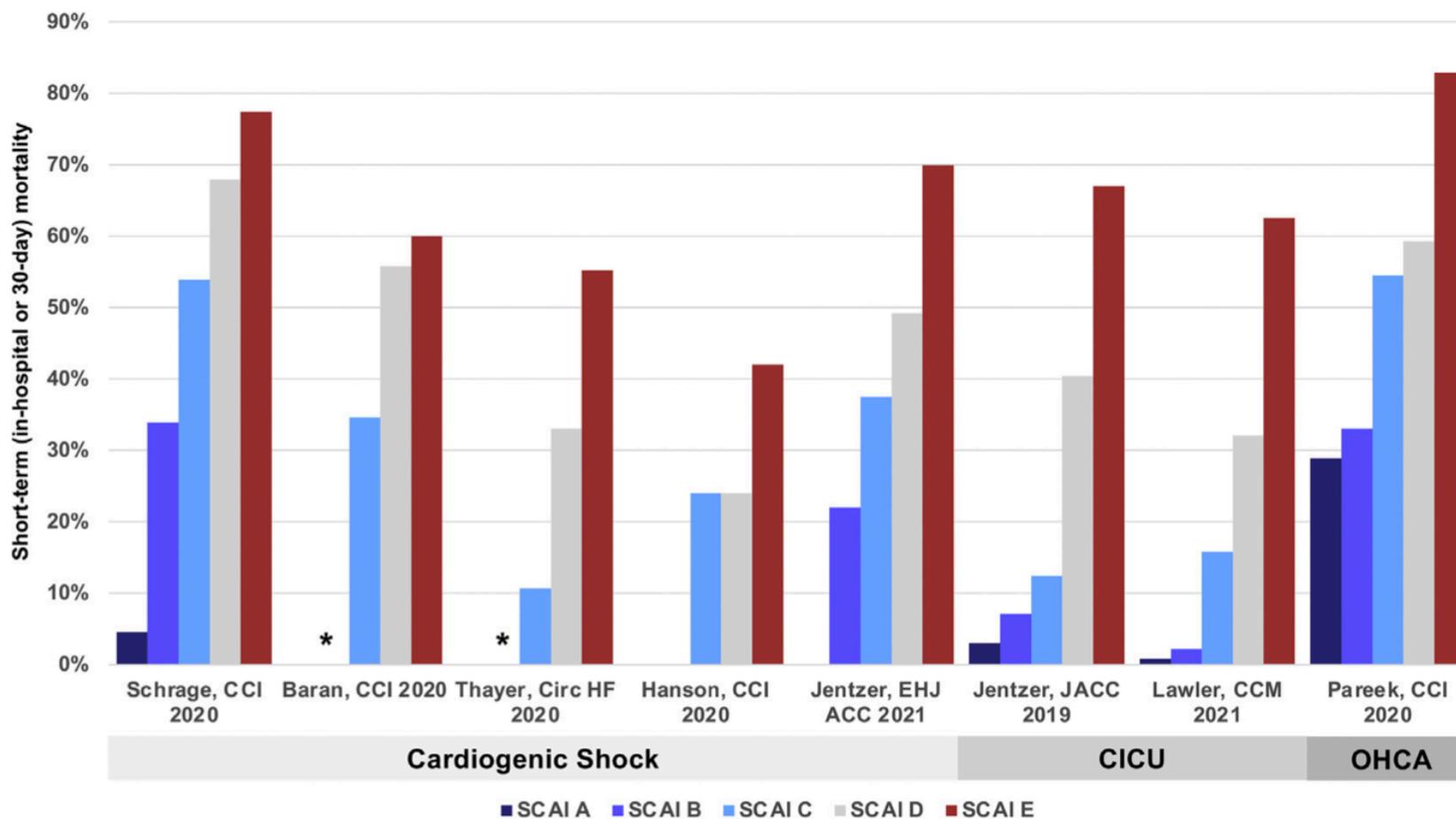


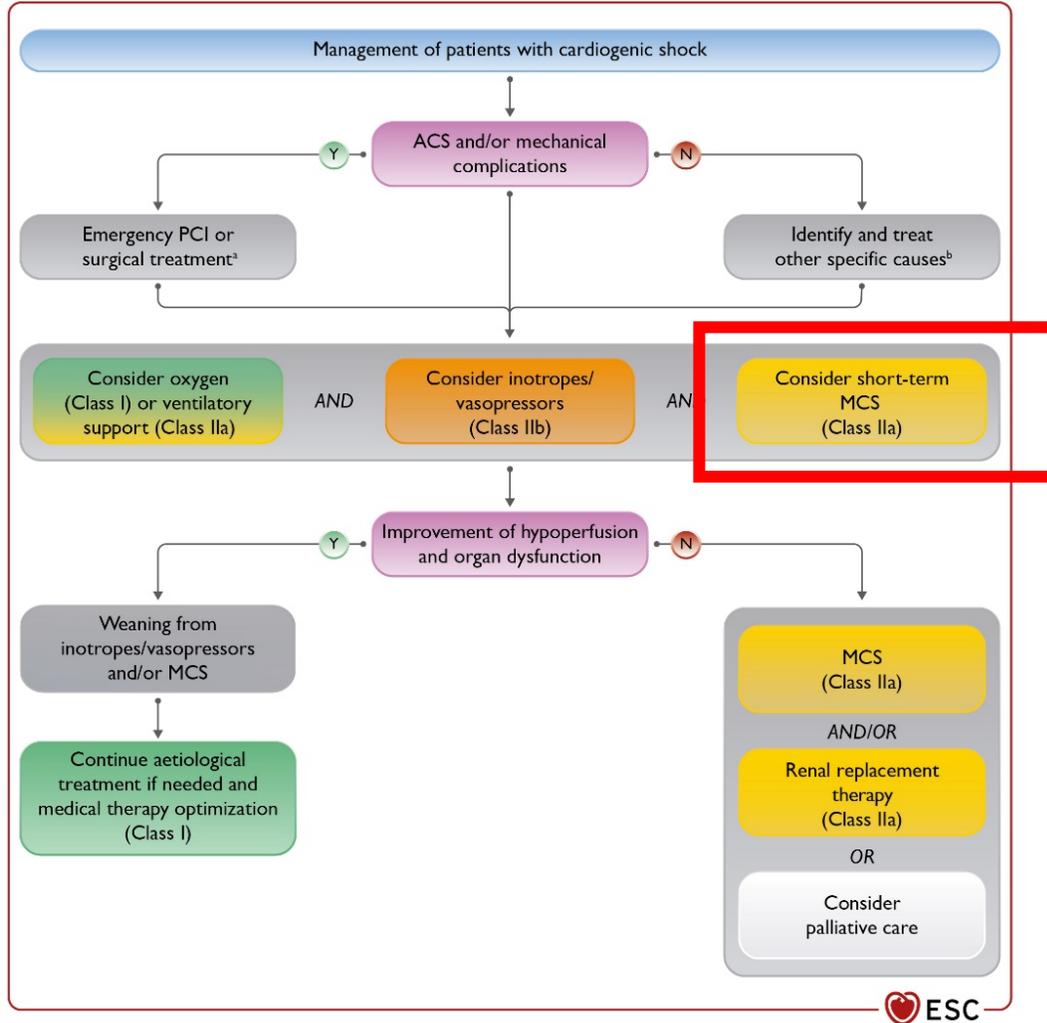
Figure 2 Pathophysiology of cardiogenic shock with staged abnormalities of clinic examination, haemodynamics, microcirculatory dysfunction and organ failure. On the upper row, the SCAI classification is presented. Ac, arteriolar constriction; Ad, arteriolar dilatation; ACM, alveolar-capillary membrane; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CI, cardiac index; DIC, disseminated intravascular coagulation; eGFR, estimated glomerular filtration rate; GGT, gamma glutamyltransferase; SBP, systolic blood pressure; SIRS, systemic inflammatory response syndrome; SVR, systemic vascular resistance; TMAO, trimethylamine N-oxide; Vc, venous constriction; Vd, venous dilatation.

FIGURE 2 Short-Term Mortality as a Function of SCAI SHOCK Stages in Each Study



*denotes that no deaths were observed in patients with SCAI stage B in these studies. CICU = cardiac intensive care unit; OHCA = out-of-hospital cardiac arrest; SCAI = Society for Cardiovascular Angiography and Interventions.

Kardiyojenik Şok Tedavi...



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Kardiyojenik Şok: medikal tedavi

Table 4. Mechanism of Action and Hemodynamic Effects of Common Vasoactive Medications in CS

Medication	Usual Infusion Dose	Receptor Binding				Hemodynamic Effects
		α_1	β_1	β_2	Dopamine	
Vasopressor/inotropes						
Dopamine	0.5–2 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	–	+	–	+++	↑CO
	5–10 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	+	+++	+	++	↑↑CO, ↑SVR
	10–20 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	+++	++	–	++	↑↑SVR, ↑CO
Norepinephrine	0.05–0.4 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	++++	++	+	–	↑↑SVR, ↑CO
Epinephrine	0.01–0.5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	++++	++++	+++	–	↑↑CO, ↑↑SVR
Phenylephrine	0.1–10 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	+++	–	–	–	↑↑SVR
Vasopressin	0.02–0.04 U/min	Stimulates V_1 receptors in vascular smooth muscle				↑↑SVR, ↔PVR
Inodilators						
Dobutamine	2.5–20 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	+	++++	++	–	↑↑CO, ↓SVR, ↓PVR
Isoproterenol	2.0–20 $\mu\text{g}/\text{min}$	–	++++	+++	–	↑↑CO, ↓SVR, ↓PVR
Milrinone	0.125–0.75 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	PD-3 inhibitor				↑CO, ↓SVR, ↓PVR
Enoximone	2–10 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	PD-3 inhibitor				↑CO, ↓SVR, ↓PVR
Levosimendan	0.05–0.2 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$	Myofilament Ca^{2+} sensitizer, PD-3 inhibitor				↑CO, ↓SVR, ↓PVR

CO indicates cardiac output; CS, cardiogenic shock; PD-3, phosphodiesterase-3; PVR, pulmonary vascular resistance; and SVR, systemic vascular resistance.

Cardiogenic Shock: Short-term MCS

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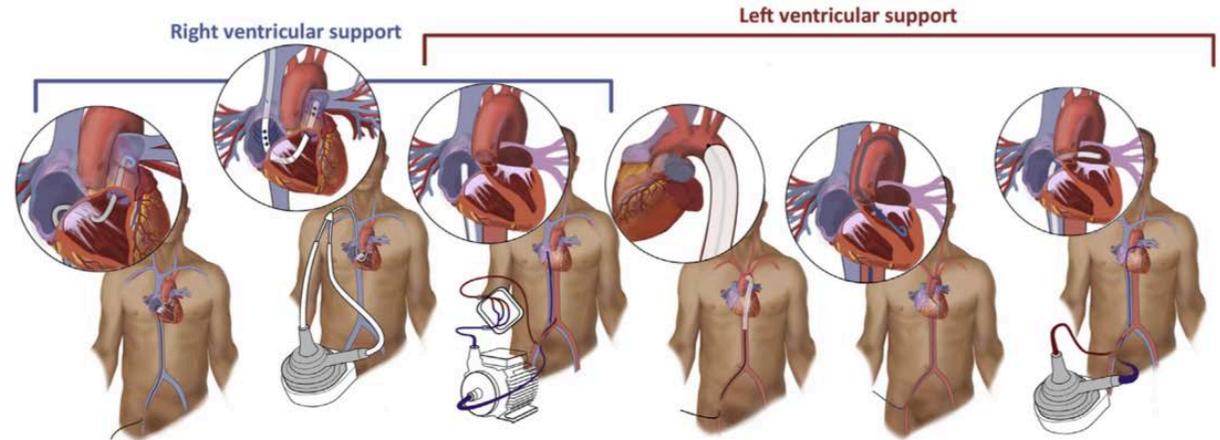
MINI-FOCUS: HEART FAILURE AND CARDIOGENIC SHOCK

STATE-OF-THE-ART REVIEW

A Standardized and Comprehensive
Approach to the Management of
Cardiogenic Shock



FIGURE 2 Current Mechanical Circulatory Support Devices Used for the Treatment of Cardiogenic Shock



	Impella RP	TandemHeart RA-PA	VA-ECMO	IABP	Impella (2.5, CP, 5.0, 5.5)	TandemHeart LA-FA
Flow	max 4.0 l/min	max 4.0 l/min	max 7.0 l/min	0.5 l/min	2.5 - 5.5 l/min	max 4.0 l/min
Pump Speed	33000 rpm	max 7500 rpm	max 5000 rpm	NA	max 51,000 rpm	max 7500 rpm
Mechanism	Axial flow continuous pump (RA-to-PA)	Centrifugal flow continuous pump (RA-to-PA)	Centrifugal flow continuous pump (RA-to-AO)	Balloon inflation-deflation (AO)	Axial flow continuous pump (LV-to-AO)	Centrifugal flow continuous pump (LA-to-AO)
Cannula Size	22 F venous	29 F venous	14-19 F arterial 17-21 F venous	7-8 F arterial	13-21 F arterial	12-19 F arterial 21 F venous
Insertion/Placement	Femoral vein	Internal jugular vein	Femoral vein Femoral artery	Femoral artery Axillary artery	Femoral artery Axillary artery	Femoral artery Femoral vein
LV Unloading	-	-	-	+	++ to +++	++
RV Unloading	+	+	++	-	-	-
Cardiac Power	-	-	↑↑	↑	↑↑	↑↑
Afterload	-	-	↑↑	↓	↓↓	↑
Coronary Perfusion	-	-	-	↑	↑	-
Considerations	<ul style="list-style-type: none"> RECOVER RIGHT: 73% survival-to-30 days in RVF post LVAD, AMI or cardiomy May 2019 - FDA post-approval study: 33% survival-to-30 days 	<ul style="list-style-type: none"> IJ access may facilitate early ambulation 	<ul style="list-style-type: none"> Bi-V + oxygenation support for CS following: <ul style="list-style-type: none"> AMI, ADHF or cardiac arrest Cardiotomy Myocarditis Allograft rejection 	<ul style="list-style-type: none"> Requires stable cardiac rhythm and native heart function May consider in select cases of post-AMI mechanical complications 	<ul style="list-style-type: none"> June 2008 – FDA 510(k) approval for HR-PCI April 2016: Expanded Indication for CS Contraindicated with mechanical aortic valve, LV thrombus 	<ul style="list-style-type: none"> Requires transeptal access Oxygenator may be added to the circuit

Intraaortic Balloon Pump in Cardiogenic Shock Complicating Acute Myocardial Infarction

Long-Term 6-Year Outcome of the Randomized IABP-SHOCK II Trial

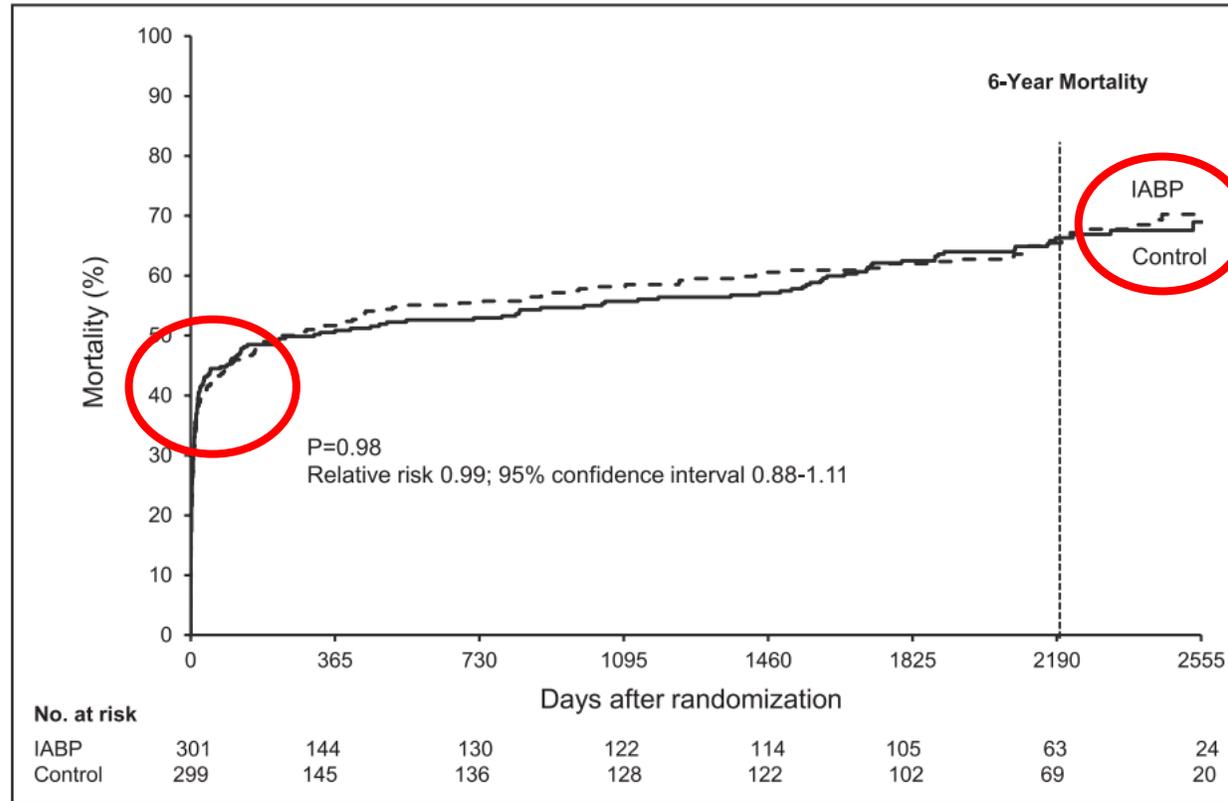
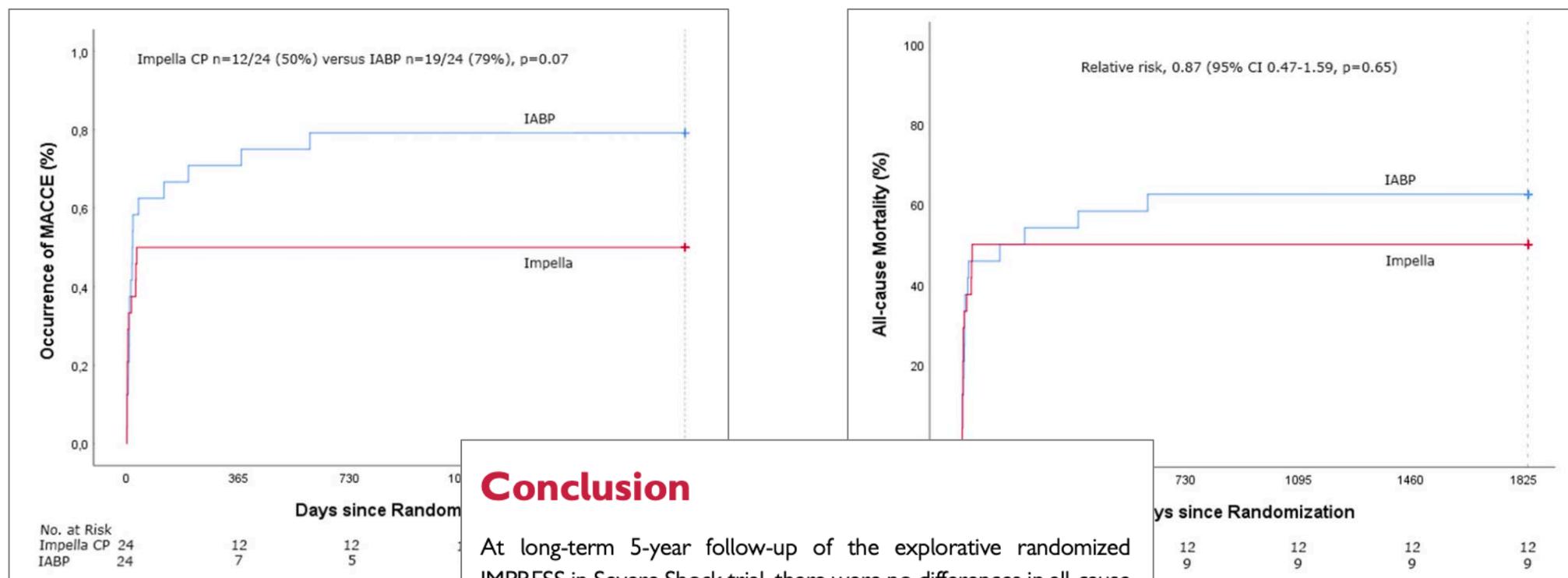


Figure 2. Time-to-event curves through 6 years.

Time-to-event curves through 6 years for all-cause mortality. *P* value is based on the log-rank test. Event rates represent Kaplan–Meier estimates. IABP indicates intraaortic balloon pump.

Long-term 5-year outcome of the randomized IMPRESS in severe shock trial: percutaneous mechanical circulatory support vs. intra-aortic balloon pump in cardiogenic shock after acute myocardial infarction



Conclusion

At long-term 5-year follow-up of the explorative randomized IMPRESS in Severe Shock trial, there were no differences in all-cause mortality and functional status between pMCS and IABP treated patients, supporting previously published short-term data and in accordance with other long-term CS trials.

ORIGINAL ARTICLE

Clinical Outcomes Associated With Acute Mechanical Circulatory Support Utilization in Heart Failure Related Cardiogenic Shock (acute HF on chronic HF)

CSWG registry:

3 outcome categories

- I) mortality
- II) heart replacement therapy
- III) native heart survival

712 pts:

Mortality: 25.3%

HRT: 38.9%

NHS: 35.8%

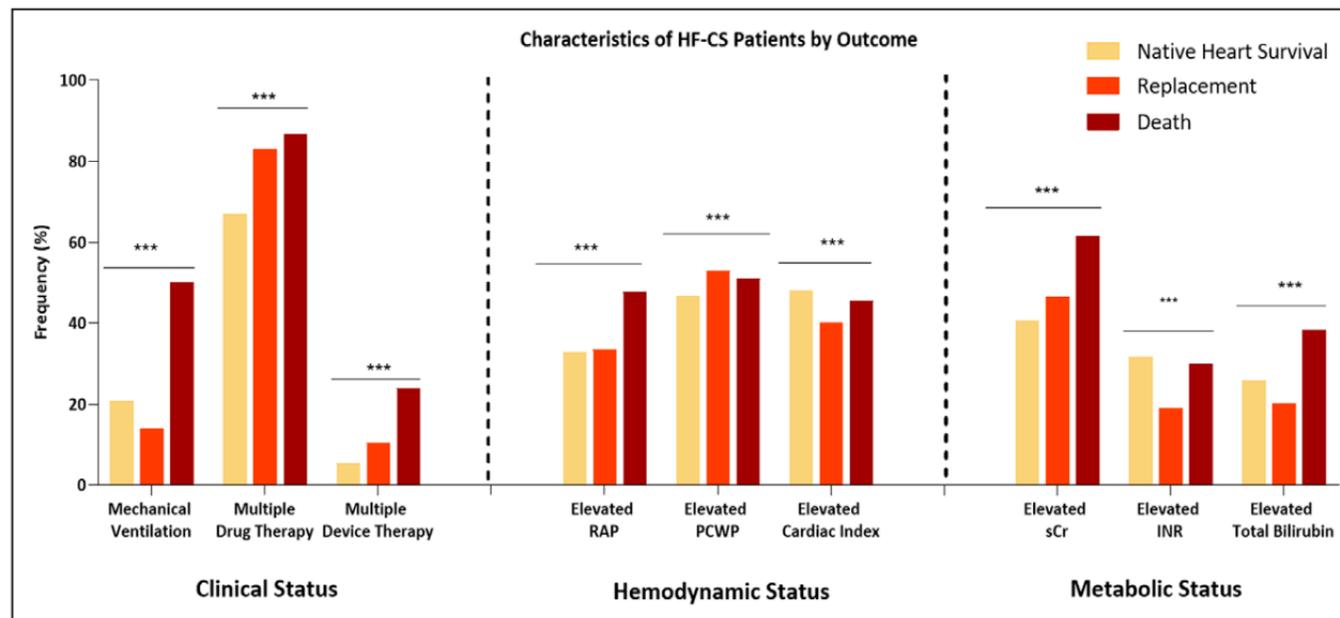


Figure 1. Clinical, hemodynamic, and metabolic characteristics of patients with cardiogenic shock resulting from decompensated heart failure (HF-CS) by outcome.

Patients experiencing in-hospital mortality were more frequently mechanically ventilated and treated with multiple vasoactive drugs and mechanical support devices than those who survived or went on to replacement therapies (***) $P < 0.001$. INR indicates international normalized ratio; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; and sCr, serum creatinine.

****IABP was most commonly used**

****IABP was most commonly used in pts who underwent HRT**

****Pts receiving > 1 MCS had highest in hospital mortality (irrespective of drug therapy)**

****ECMO was used in more severe cases of shock (D and E)**

****Mortality was highest in ECMO:**

ECMO: 54.7%

Impella: 45.3%

IABP: 23%

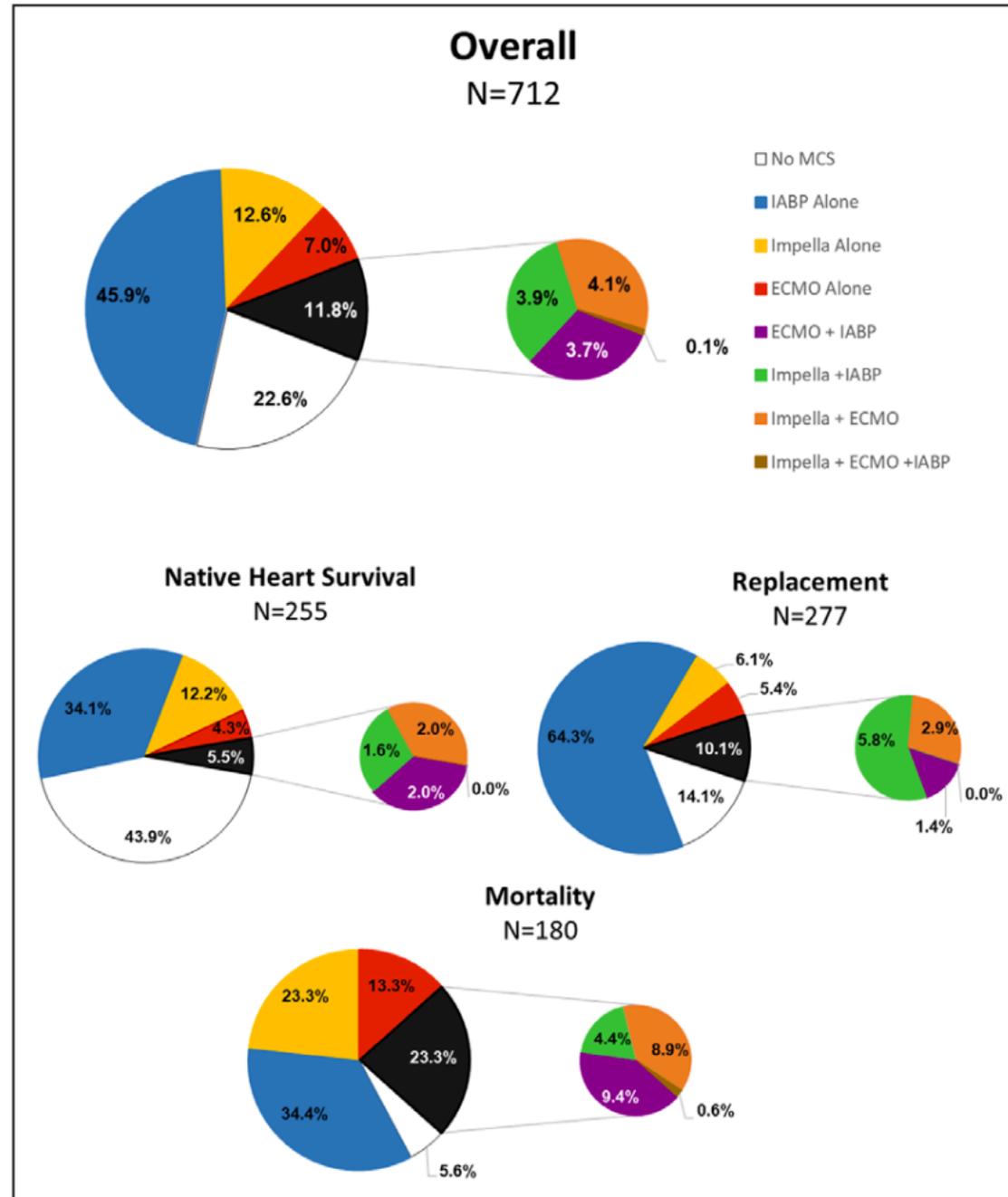


Figure 2. Distribution of acute mechanical circulatory support (AMCS) devices used alone and in combination in the overall study cohort and among patients in each outcome group.

ECMO indicates extracorporeal membrane oxygenation; and IABP, intraaortic balloon pump.

Mortality increased with deteriorating stage

Most common stage was
D (63%) > C (22%) > E (8%) > B (6%)

B: 82.5% survived without HRT
17.5% had HRT
no mortality

C: 53.5% survived without HRT
35.6% had HRT
10.8% died

D: 26.6% survived without HRT
44% had HRT
29.4% died

E: 14.5% survived without HRT
30.9% had HRT
54.6% died

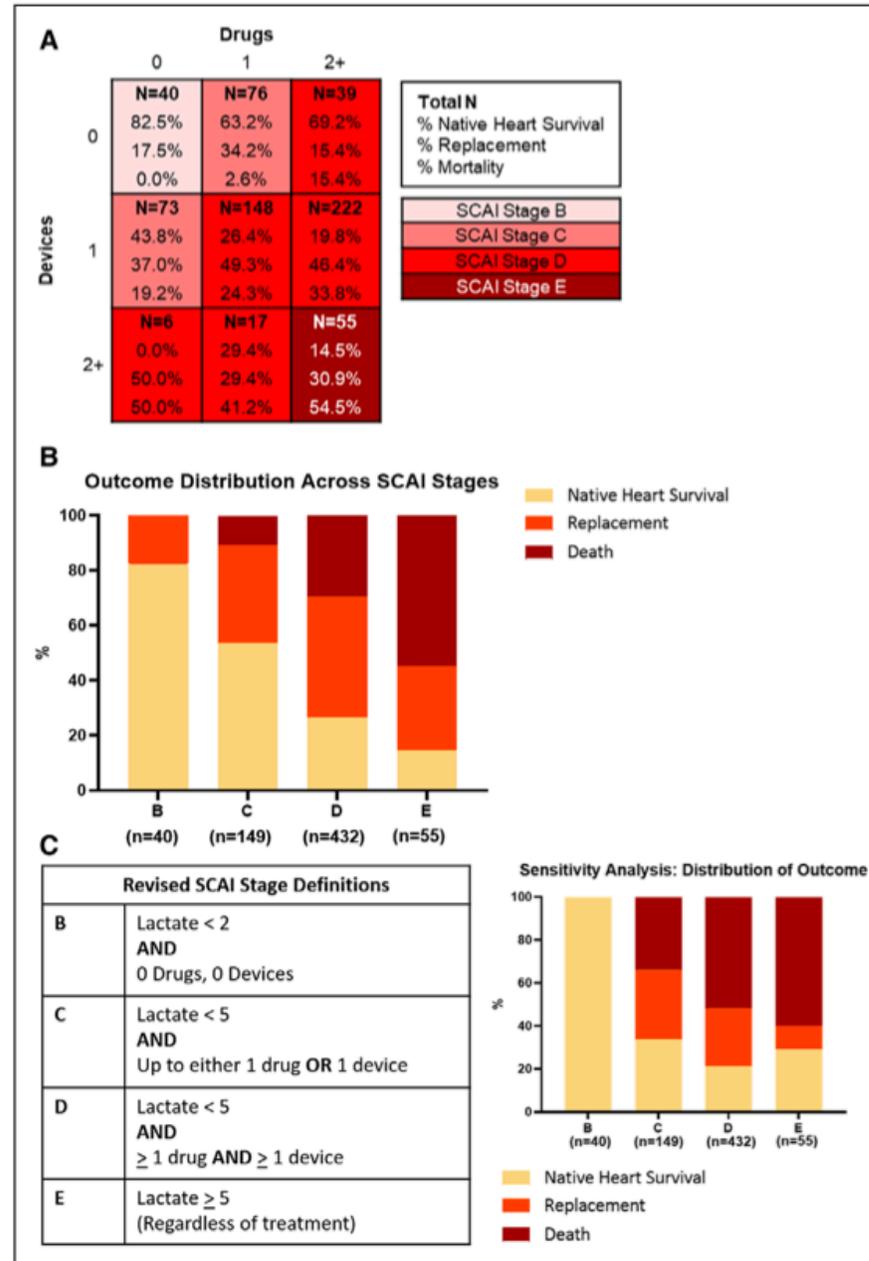


Figure 3. Clinical outcomes according to Society for Cardiovascular Angiography and Intervention (SCAI) stages as defined by the Cardiogenic Shock Working Group (CSWG) according to treatment intensity. A, Grid analysis of heart failure outcomes by drug and device utilization and CSWG definitions of SCAI stages. B, Increasing SCAI stage is associated with increased in-hospital mortality and decreased native heart survival (CSWG definitions of SCAI stages: B: no drugs or acute mechanical circulatory support [AMCS], C: up to 1 drug or 1 device, D: >1 drug OR >1 device, E: >1 drug AND >1 device). C, Sensitivity analysis of CSWG definitions of SCAI stages including lactate cutoffs.

Circulation: Heart Failure

ORIGINAL ARTICLE

Clinical Outcomes Associated With Acute Mechanical Circulatory Support Utilization in Heart Failure Related Cardiogenic Shock

- **Highest RAP and heart rate and lowest BP were associated with mortality**
- **Biventricular failure common in pts who died and isolated LV failure common in pts who had HRT**
- **Lactate, BUN, serum creatinine and aspartate aminotransferase were highest in pts who died**
- **In-hospital mortality was associated with biventricular congestion and end-organ hypoperfusion**
- **The study does not clarify whether IABP use was effective or whether one device is more effective**

It is not the device used, but the STAGE & PHENOTYPE of shock that is associated with mortality

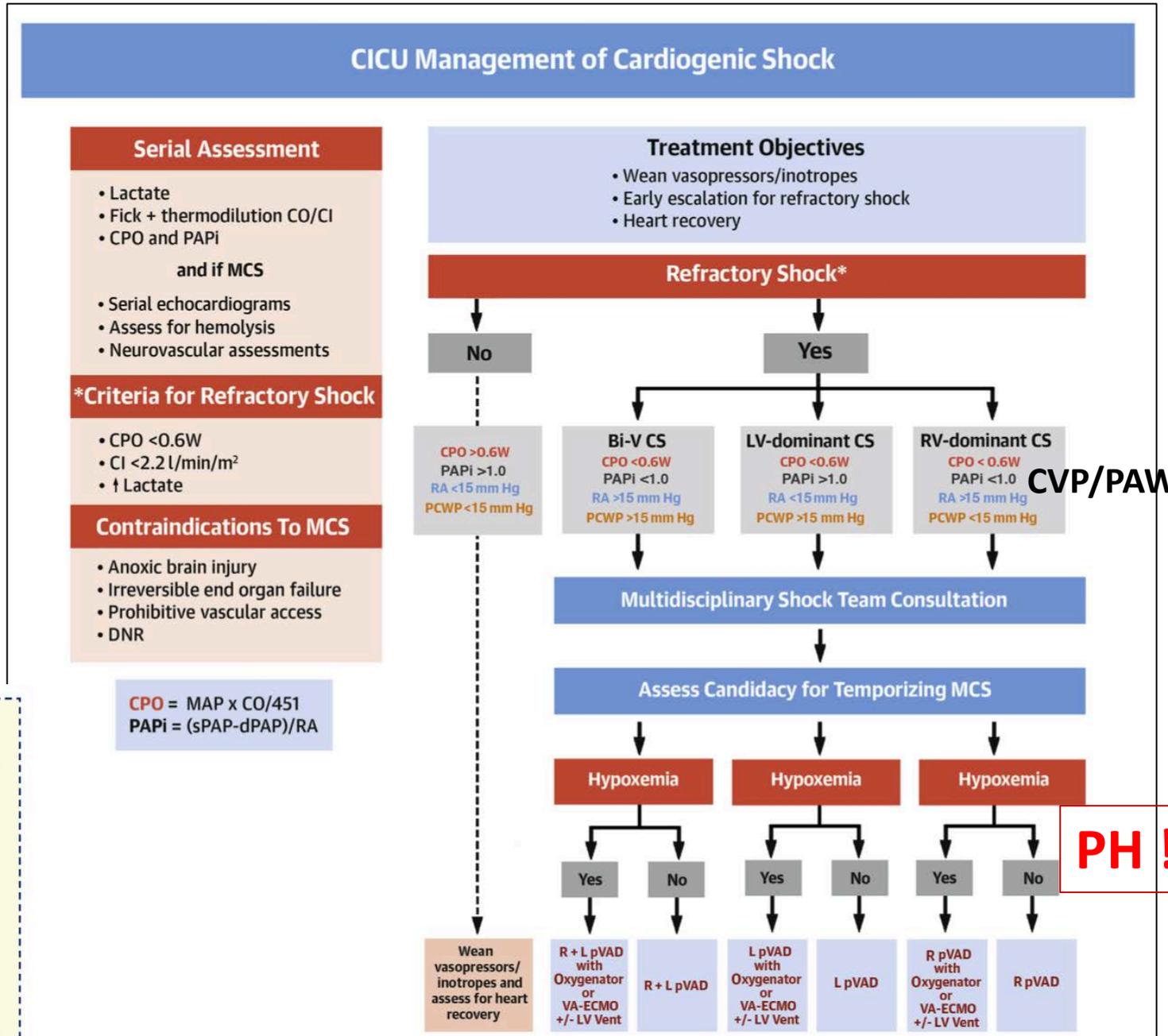
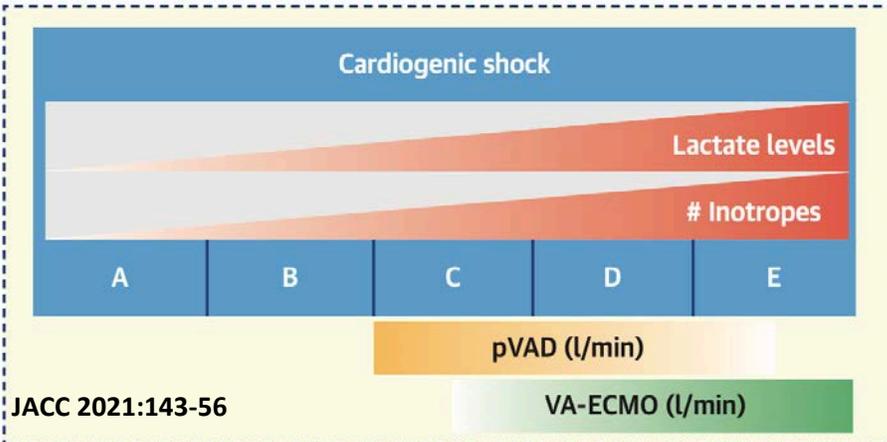
Cardiogenic Shock: Short-term MCS

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STATE-OF-THE-ART REVIEW

A Standardized and Comprehensive Approach to the Management of Cardiogenic Shock

- LV dominant
- RV dominant
- Biventrikular tutulum



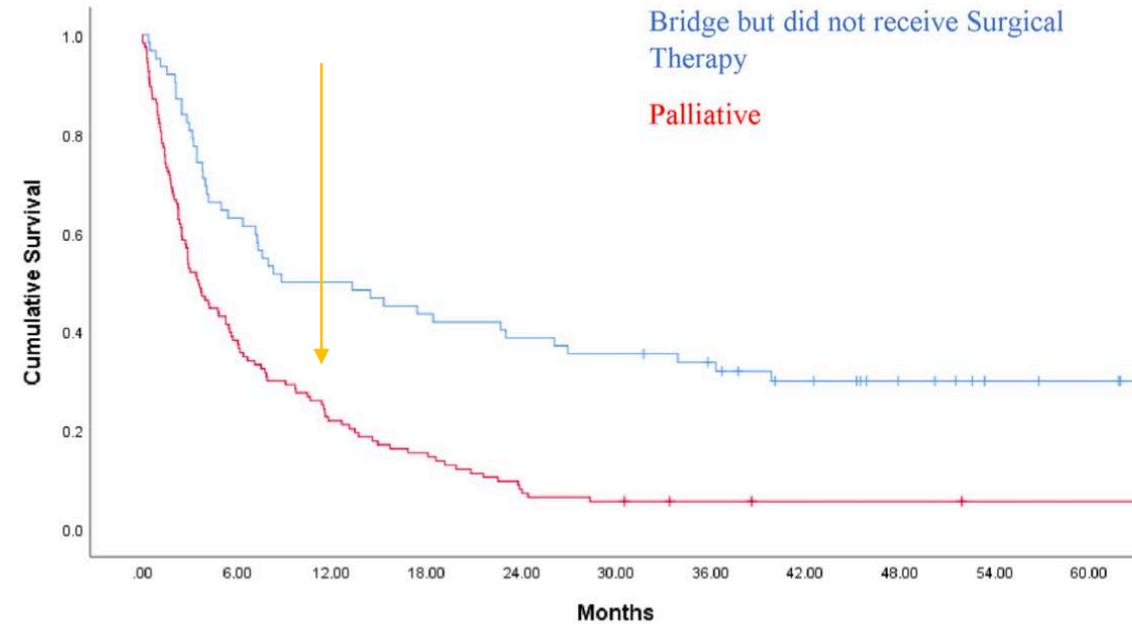
CVP/PAWP >0.63

PH !!!!

Chronic Intravenous Inotropic Support as Palliative Therapy and Bridge Therapy for Patients With Advanced Heart Failure: A Single-Center Experience

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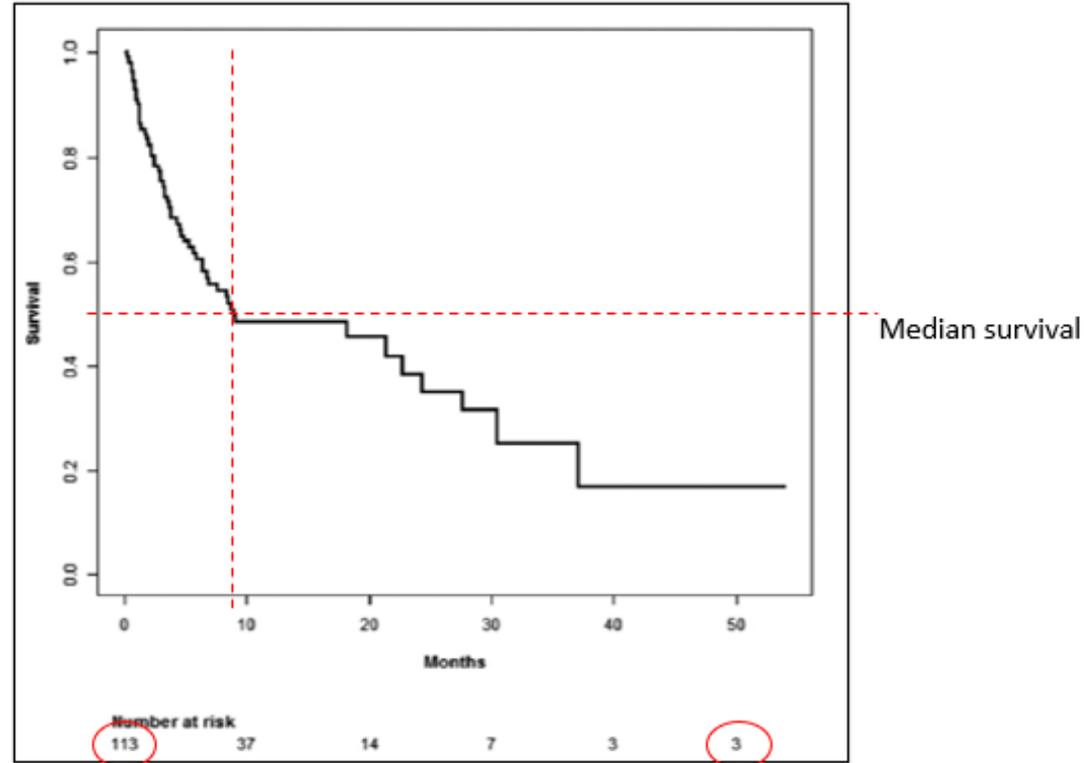


Number at risk

Months	0	6	12	18	24	30	36	42	48	54	60
Bridge	63	39	31	27	24	20	16	9	5	4	2
Palliative	123	47	27	19	10	4	4	4	4	4	3

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9-month survival is in patients on inotropes who did not receive a transplant or left ventricular assist device

ORIGINAL

Causes and predictors of early mortality in patients treated with left ventricular assist device implantation in the European Registry of Mechanical Circulatory Support (EUROMACS)



Sakir Akin^{1,2}, Osama Soliman³, Theo M. M. H. de By⁴, Rahatullah Muslim¹, Jan G. P. Tjissen^{5,6}, Felix Schoenrath⁷, Bart Meyns⁸, Jan F. Gummert⁹, Paul Mohacs¹⁰ and Kadir Caliskan^{1*} on behalf of the EUROMACS investigators

Table 3 Baseline multivariate predictors of early mortality after LVAD implantation using continuous values

Variables	OR	95.0% CI for OR	p value
Age (years)	1.028	1.018–1.038	0.000
Gender (female)	1.339	1.003–1.788	0.048
INTERMACS Class 1–3	1.5	1.121–2.007	0.006
ECMO	1.989	1.431–2.765	0.000
* Creatinine $\mu\text{mol/L}$	1.003	1.002–1.005	0.000
* Total bilirubin g/dL	1.193	1.116–1.275	0.000
Lactate mmol/L	1.011	1.003–1.019	0.008
Hemoglobin g/dL	0.908	0.858–0.961	0.001
* RA/PCWP	1.74	1.292–2.344	0.000
PVR woods unit	1.089	1.044–1.135	0.000
SVR woods unit	0.974	0.957–0.992	0.004
Total implantation time (min)	1.003	1.002–1.004	0.000

For abbreviations, see Table 1

[16]. Furthermore, patients with pre-operative impaired renal and hepatic function, or prolonged peripheral tissue hypoxia (lactate) have increased early mortality following LVAD implantation. We believe that proper timing of LVAD, earlier in the process of end-stage heart failure, before a full-blown cardiogenic shock, is critical in achieving a good survival chance. Furthermore,

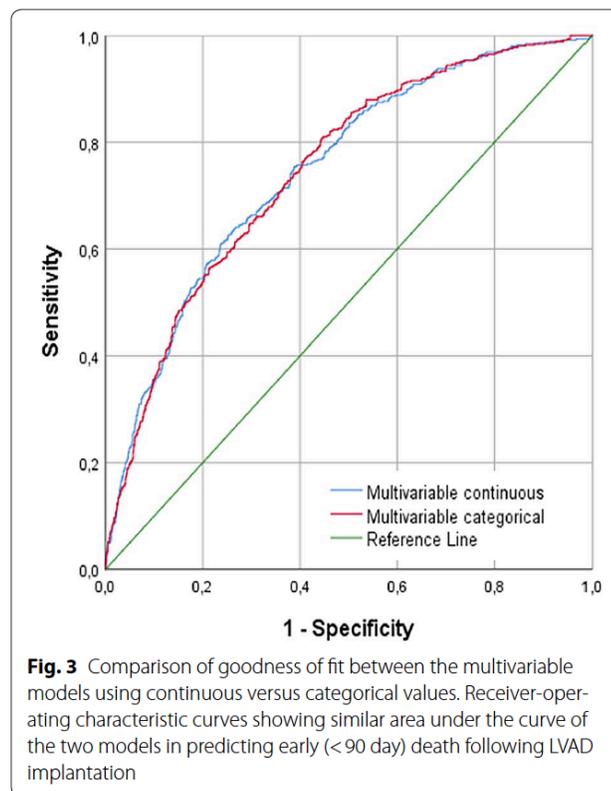


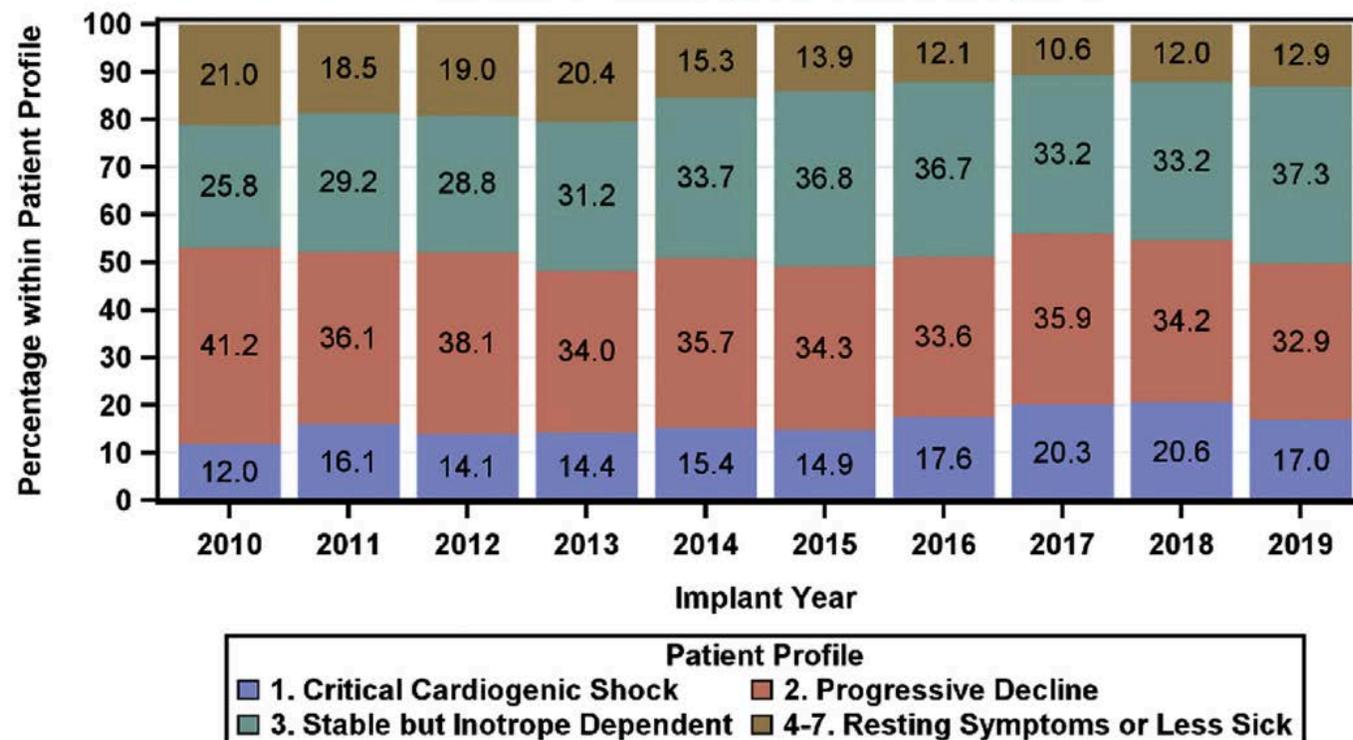
Fig. 3 Comparison of goodness of fit between the multivariable models using continuous versus categorical values. Receiver-operating characteristic curves showing similar area under the curve of the two models in predicting early (< 90 day) death following LVAD implantation

The Society of Thoracic Surgeons InterMACs 2020 Annual Report



Ezequiel J. Molina, MD, Palak Shah, MD, MS, Michael S. Kiernan, MD, MS, William K. Comwell III, MD, MSCS, Hannah Copeland, MD, Koji Takeda, MD, PhD, Felix G. Fernandez, MD, Vinay Badhwar, MD, Robert H. Habib, PhD, Jeffrey P. Jacobs, MD, Devin Koehl, MSDS, James K. Kirklin, MD, Francis D. Pagani, MD, PhD, and Jennifer A. Cowger, MD, MS

**Patient Profile for Primary Continuous Flow LVAD (n=25,472)
InterMACs: January 1, 2010-December 31, 2019**



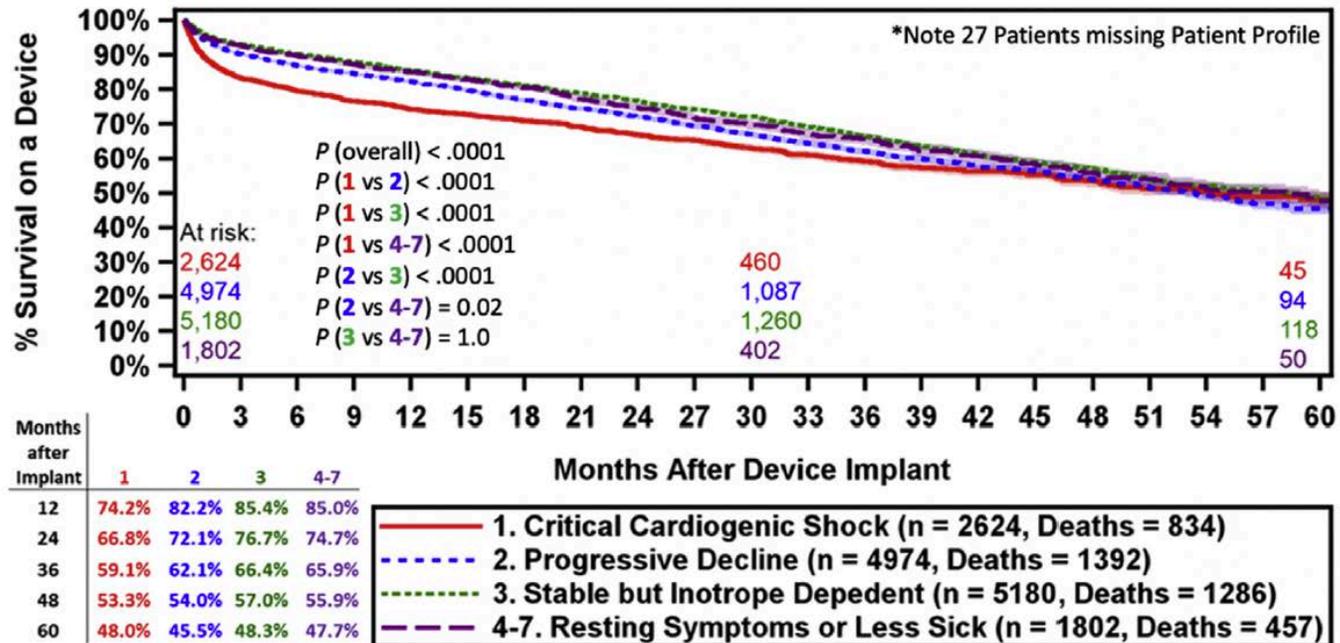
profiles 4-7 were less common in the more recent era

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Kaplan-Meier Survival for Continuous Flow LVAD by Era 2015-2019 (n=14,580)



Shaded areas indicate 70% confidence limits
 p (log-rank) = <.0001
 Event: Death (censored at transplant or cessation of support)

survival is improving over time with all intermacs profiles
survival curves overlap for profile 3 and 4-7

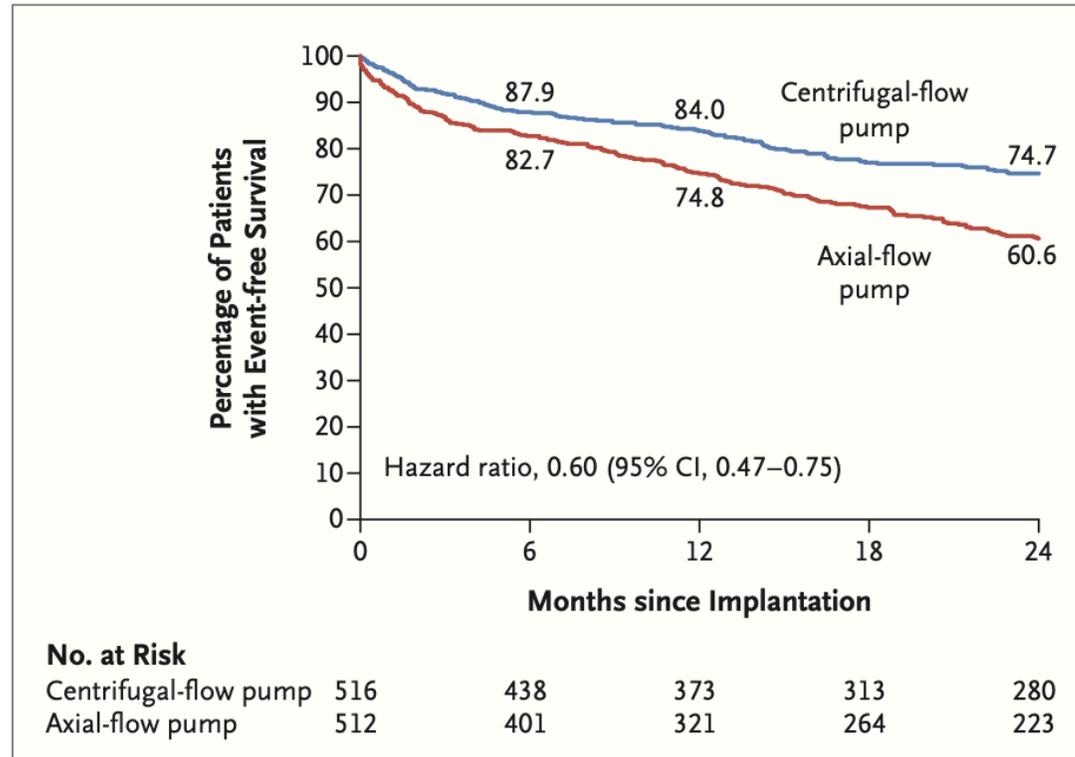
ORIGINAL ARTICLE



A Fully Magnetically Levitated Left Ventricular Assist Device — Final Report

M.R. Mehra, N. Uriel, Y. Naka, J.C. Cleveland, Jr., M. Yuzefpolskaya, C.T. Salerno, M.N. Walsh, C.A. Milano, C.B. Patel, S.W. Hutchins, J. Ransom, G.A. Ewald, A. Itoh, N.Y. Raval, S.C. Silvestry, R. Cogswell, R. John, A. Bhimaraj, B.A. Bruckner, B.D. Lowes, J.Y. Um, V. Jeevanandam, G. Sayer, A.A. Mangi, E.J. Molina, F. Sheikh, K. Aaronson, F.D. Pagani, W.G. Cotts, A.J. Tatoes, A. Babu, D. Chomsky, J.N. Katz, P.B. Tessmann, D. Dean, A. Krishnamoorthy, J. Chuang, I. Topuria, P. Sood, and D.J. Goldstein, for the MOMENTUM 3 Investigators*

2019



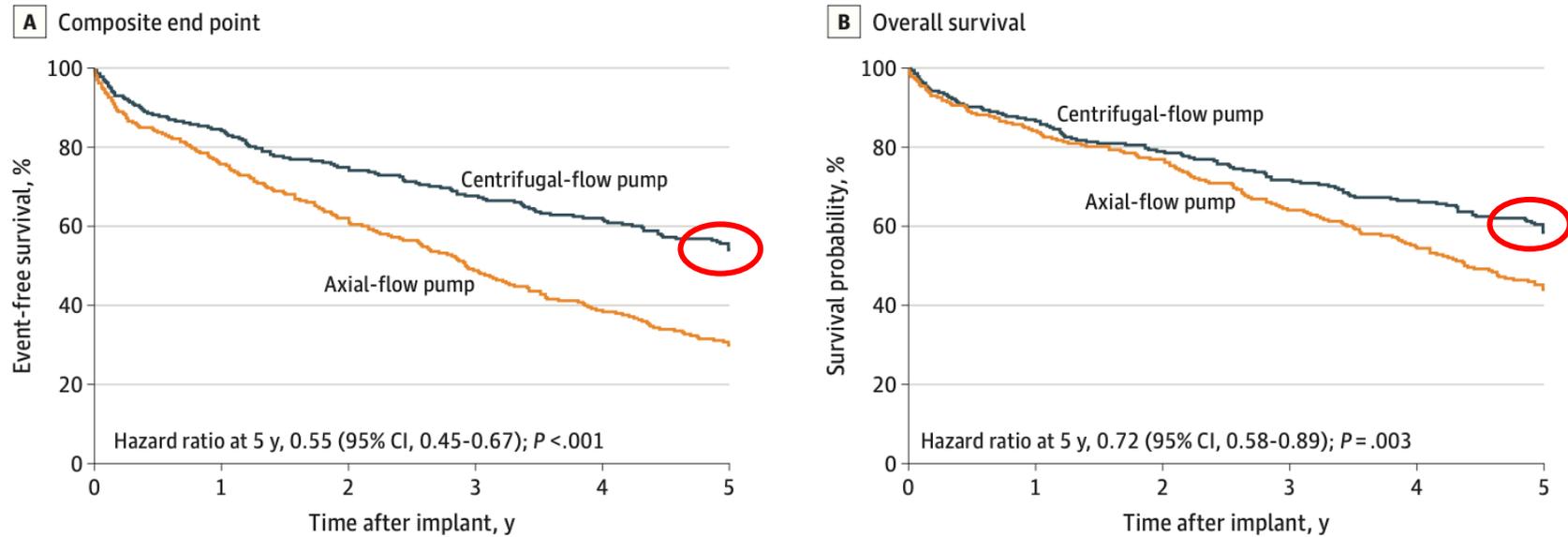
Pr-endpoint: survival free of of disabling stroke or reoperation to replace / remove a malfunctionin device

Five-Year Outcomes in Patients With Fully Magnetically Levitated vs Axial-Flow Left Ventricular Assist Devices in the MOMENTUM 3 Randomized Trial

Mandeep R. Mehra, MD, MSc; Daniel J. Goldstein, MD; Joseph C. Cleveland, MD; Jennifer A. Cowger, MD, MS; Shelley Hall, MD; Christopher T. Salerno, MD; Yoshifumi Naka, MD, PhD; Douglas Horstmanshof, MD; Joyce Chuang, PhD; AiJia Wang, MPH; Nir Uriel, MD, MSc

2022

Figure 2. Composite End Point and Overall Survival in a Study of 5-Year Outcomes in Patients With Fully Magnetically Levitated vs Axial-Flow Left Ventricular Assist Devices (LVADs)

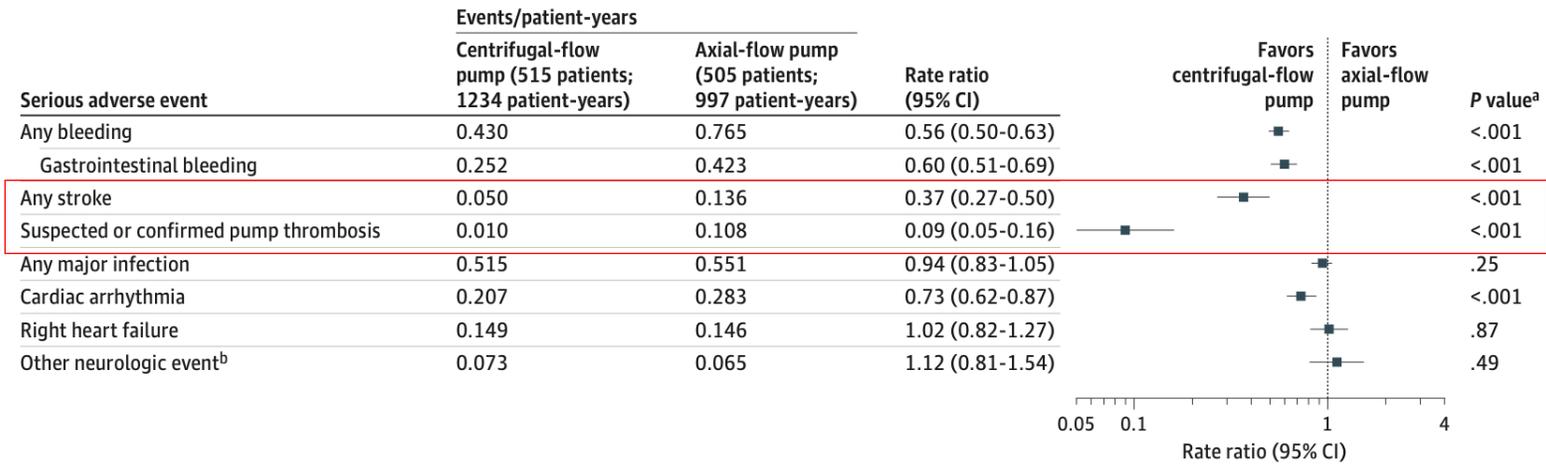


No. of patients	0	1	2	3	4	5
Centrifugal-flow pump	515	373	280	208	177	138
Axial-flow pump	505	321	223	147	106	71

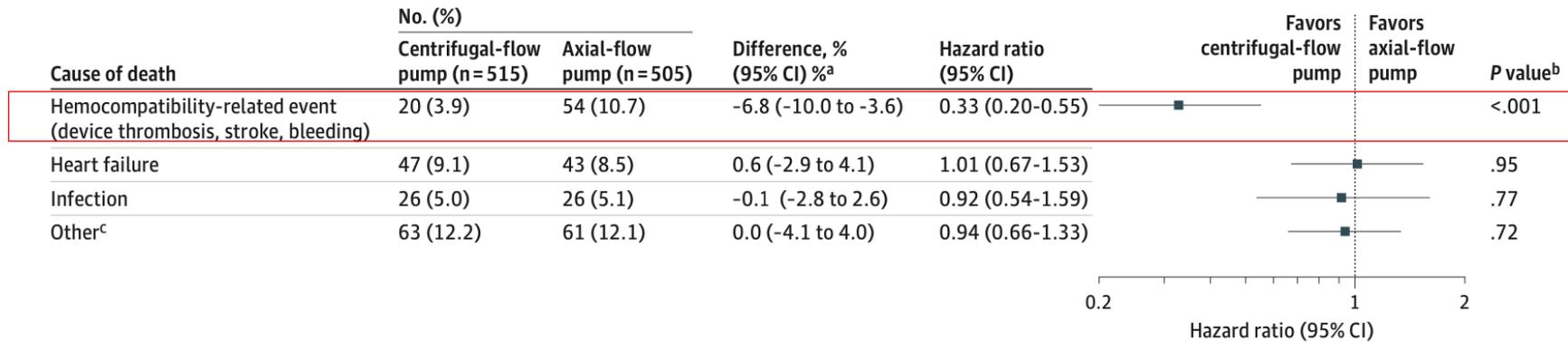
515	383	289	213	184	141
505	339	247	165	124	85

Figure 3. Serious Adverse Events in a Study of 5-Year Outcomes in Patients With Fully Magnetically Levitated vs Axial-Flow Left Ventricular Assist Devices

adverse events

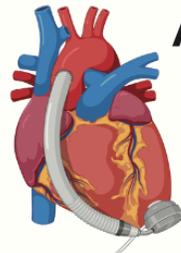


death





Primary results of long-term outcomes in the MOMENTUM 3 pivotal trial and continued access protocol study phase: a study of 2200 HeartMate 3 left ventricular assist device implants



A Study of 2200 HeartMate 3 Left Ventricular Assist Device Implants in the MOMENTUM 3 Trial Portfolio

Are long-term outcomes with the HM3 LVAD in the post-pivotal trial experience different from early pivotal-trial observations?

Two-Year Endpoints

Composite Endpoint

Survival free of disabling stroke or reoperation to replace or remove a malfunctioning device

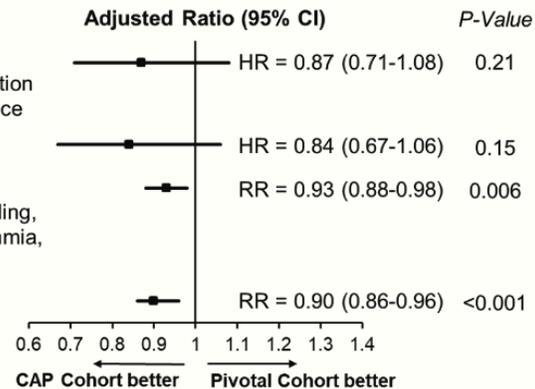
Overall Survival

Overall Adverse Event Burden

Suspected pump thrombosis, stroke, bleeding, infection, right heart failure, cardiac arrhythmia, respiratory failure, and renal dysfunction

All-Cause Readmission Burden

Principal Outcomes



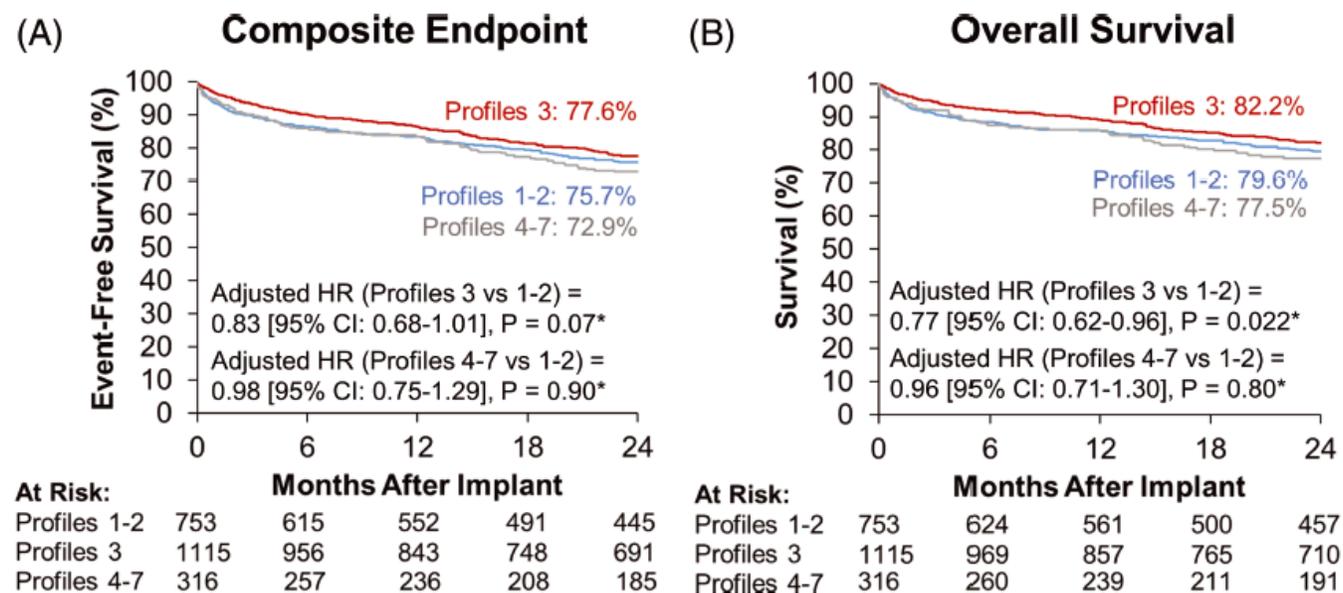
- 2200 HM3 implanted patients - 515 pivotal trial and 1685 continuous access protocol (CAP) patients
- Similar 2-year survival between CAP and pivotal trial cohorts (81.2% vs 79.0%) despite sicker patients (more intra-aortic balloon pump use and INTERMACS 1 profile) and more transplant ineligible patients intended for destination therapy in CAP
- Similar 2-year survival between CAP and pivotal trial cohorts in transplant ineligible patients (79.1% vs 76.7%) even after adjusting for baseline differences (HR=0.89 [95% CI: 0.68-1.16], P=0.38)

Accumulating post-pivotal trial experience with the HM3 LVAD suggests a lower adverse event burden, reduced hospitalizations and similar survival free of disabling stroke or reoperation to replace or remove a malfunctioning pump as compared to the pivotal MOMENTUM 3 trial outcomes at 2 years

These beneficial outcomes were noted across the continuum of clinical severity in advanced heart failure and especially among transplant ineligible patients in whom outcomes may now compare favorably with those in transplant eligible patients at 2 years

Primary results of long-term outcomes in the MOMENTUM 3 pivotal trial and continued access protocol study phase: a study of 2200 HeartMate 3 left ventricular assist device implants

INTERMACS profile			p-value
1	11 (2.1%)	69 (4.1%)	0.036
2	156 (30.4%)	517 (31.0%)	0.79
3	272 (52.9%)	843 (50.5%)	0.33
4–7	75 (14.6%)	241 (14.3%)	0.88



Teşekkür ederim.....😊