Kardiogenní šok
2026

Ivo Varvařovský

Kardiologické centrum Agel, Pardubice

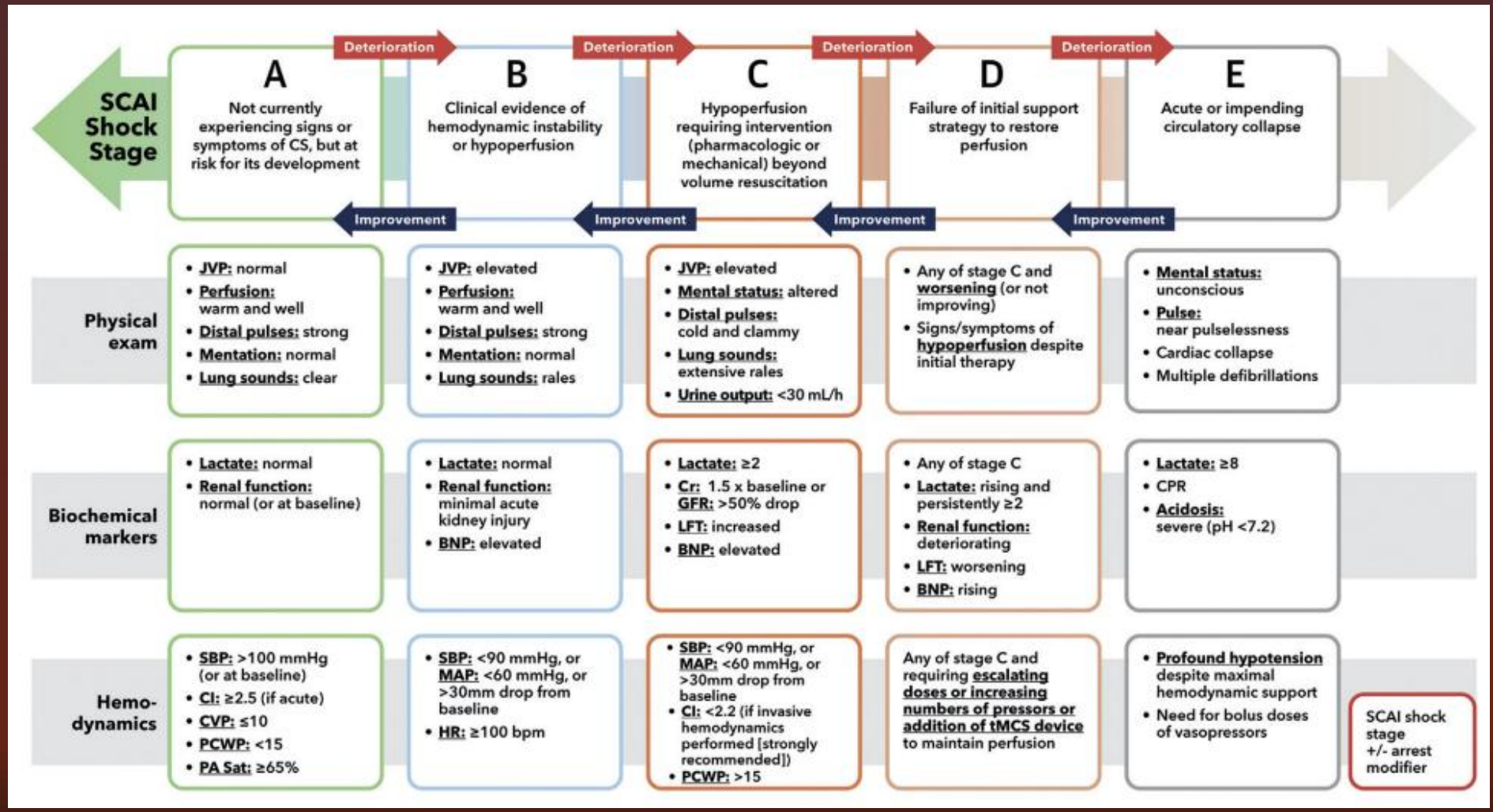
Workshop ČAIK, Třinec, 16.4.2026

Kardiogenní šok : definice

Table 1. Pragmatic and Clinical Trial Definitions of CS (Table view)

Clinical Definition	SHOCK Trial[9]*	IABP-SHOCK II[1]†	ESC HF Guidelines[15]
Cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Support to maintain SBP ≥90 mm Hg AND End-organ hypoperfusion (urine output <30 mL/h or cool extremities)Hemodynamic criteria: CI of ≤2.2 $L \cdot min^{-1} \cdot m^{-2}$ AND PCWP ≥15 mm Hg	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Catecholamines to maintain SBP >90 mm Hg AND Clinical pulmonary congestion AND Impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output <30 mL/h, or lactate >2.0 mmol/L)	SBP <90 mm Hg with adequate volume and clinical or laboratory signs of hypoperfusionClinical hypoperfusion: Cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressureLaboratory hypoperfusion: Metabolic acidosis, elevated serum lactate, elevated serum creatinine

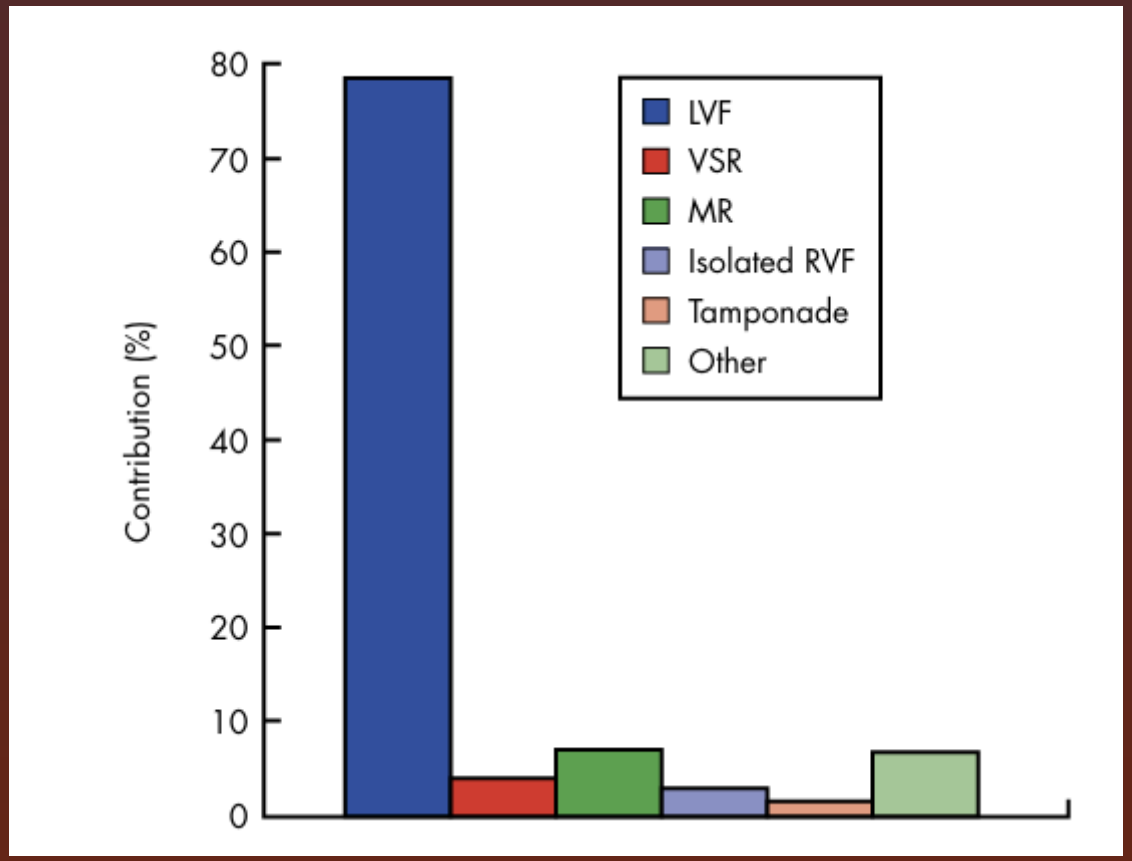
Kardiogenní šok : klasifikace klinická (SCAI)



Kardiogenní šok : klasifikace hemodynamická

		Volume Status	
		Wet	**Dry**
Peripheral Circulation	**Cold**	Classic Cardiogenic Shock (↓CI; ↑SVRI; ↑PCWP)	Euvolemic Cardiogenic Shock (↓CI; ↑SVRI; ↔PCWP)
	Warm	Vasodilatory Cardiogenic Shock or Mixed Shock (↓CI; ↓/↔SVRI; ↑PCWP)	Vasodilatory Shock (Not Cardiogenic Shock) (↑CI; ↓SVRI; ↓PCWP)

Kardiogenní šok : epidemiologie AMI-CS



Heart 2002;**88**:531–537

Kardiogenní šok : prognóza

- SCAI – 3 axis score
- CardShock score, Cardiogenic Shock Score
- SHOCK trial and registry score, IABP-SHOCK II score
- 4 biomarkers score (cystatin C, laktát, NT-pro BNP, IL-6)
- Breton DPP 3 score
- ENCOURAGE (VA-ECMO survival)
- Skorovací systémy založené na AI

Žádný skórovací systém nerozhoduje o iniciaci/ukončení terapie

Léčba kardiogeního šoku : základní strategie

Kardiogenní šok : farmakoterapie

TABLE 2 Vasoactive Agents Used in CS

Category	Agent(s)	Mechanism of Action/Receptor Binding	Dosing	Hemodynamic Effects			
				SVR	BP	CO	HR
Inopressor	Norepinephrine	α1 (+++), β1 (++), β2 (+)	0.05-1 μg/kg/min	↑↑	↑↑	↑	↑
	Epinephrine	β1 (+++), α1 (++), β2 (++)	0.01-0.5 μg/kg/min	↑↑	↑↑	↑↑	↑↑
	Dopamine	D1 (+++), β1 (++), α1 (+)	Low: 2-5 μg/kg/min Intermediate: 5-10 μg/kg/min High: 10-20 μg/kg/min	↑↑	↑↑	↑	↑↑
Inodilator	Dobutamine	β1 (+++), β2 (++)	2-10 μg/kg/min	↓↔	↓↔	↑↑	↑
	Milrinone	PDE-3 inhibitor	0.125-0.5 μg/kg/min	↓↓	↓↓	↑↑	↔↑
Vasopressor	Phenylephrine	α1 (+++)	0.1-10 μg/kg/min	↑	↑↑	↔↓	↔↓
	Vasopressin	Vasopressin receptor	0.01-0.04 U/min	↑↑	↑↑	↔↓	↔↓
Vasodilator	Nitroprusside	NO production	0.3-10 μg/kg/min	↓	↓	↑↔	↑↔
	Nitroglycerin	Converts to NO	25-200 μg/min	↓	↓	↑↔	↑↔
Chronotrope	Isoproterenol	β1 (+++), β2 (+++)	2-20 μg/min	↓	↔	↑	↑↑
	Dopamine	See above					
Inotrope	Levosimendan*	Binds to troponin C, making it more sensitive to calcium thereby improving interaction between troponin C and I	0.05-0.2 μg/kg/min	↓	↓	↑	↔

Kardiogenní šok : farmakoterapie

Table 3. Randomized Controlled Trials of Vasoactive Medications in Patients With Cardiogenic Shock

Study	Setting	Population	Sample size	Agents assessed	Mortality	Adverse events
Milrinone as Compared With Dobutamine in the Treatment of Cardiogenic Shock (CAPITAL-DOREMI), 2021[56]	ICU, single center	Cardiogenic shock	192	Dobutamine vs milrinone	No difference	Nil
Epinephrine Versus Norepinephrine for Cardiogenic Shock After Acute Myocardial Infarction (Optima CC), 2018[31]	ICU, multicenter	Post PCI AMI-CS	57	Epinephrine vs norepinephrine	No difference	Increased refractory shock in epinephrine Increased cardiac double product with epinephrine Increased serum lactate
Sepsis Occurrence in Acutely Ill Patients-II (SOAP-II), 2010[41]	ICU, multicenter	Undifferentiated shock	1679	Dopamine vs norepinephrine	No difference	Increased arrhythmic events with dopamine Increased mortality in cardiogenic shock treated with dopamine.
A Comparison of Epinephrine and Norepinephrine in Critically Ill Patients (CAT), 2008[42]	ICU, multicenter	Undifferentiated shock	280	Epinephrine vs norepinephrine	No difference	Epinephrine associated with metabolic acidosis requiring agent cessation No difference in mortality for cardiogenic shock subgroup
Effect of Tilarginine Acetate in Patients With Acute Myocardial Infarction and Cardiogenic Shock (TRIUMPH), 2007[53]	ICU, multicenter	Post PCI AMI-CS	398	Tilarginine acetate vs placebo	No difference	Nil
Levosimendan vs Dobutamine for Patients With Acute Decompensated Heart Failure (SURVIVE), 2007[54]	In-hospital, multicenter	Decompensated heart failure +/– low cardiac output	1327	Levosimendan vs dobutamine	No difference	Increased atrial fibrillation, hypokalemia, and headache with levosimendan
Efficacy and Safety of Intravenous Levosimendan Compared With Dobutamine in Severe Low-Output Heart Failure (LIDO), 2002[55]	In-hospital, multicenter	Low output heart failure (including decompensated heart failure and post cardiotomy)	203	Levosimendan vs dobutamine	No difference	Increased arrhythmia events and angina with dobutamine treatment

Kardiogenní šok : farmakoterapie

Vasopressors					
SOAP II (CS subgroup)	28 days	50/145	64/135		0.73 (0.54;0.97)
Levy et al.	28 days	4/15	5/15		0.80 (0.27;2.30)
OptimaCC	28 days	8/30	13/27		0.55 (0.27;1.10)
Total		**62/190**	**82/177**	Noreprinephrine better / Dopamine or epinephrine better	**0.70 (0.54;0.91)**

EuroIntervention 2021;17:451-465

Kardiogenní šok : časná revaskularizace

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.,

Median time from MI to randomization (hr)	11.0 (5.9–19.4)	12.0 (6.3–21.8)
<6 hr from MI to randomization (%)	25.0	23.7
Lowest systolic blood pressure (mm Hg)‡	66.4±14.3	69.8±11.3
Systolic blood pressure (mm Hg)§	89.0±22.8	86.5±17.4
Diastolic blood pressure (mm Hg)§	53.9±16.8	55.1±13.6
Heart rate (beats/min)§	103.3±22.0	100.1±22.7
Pulmonary-capillary wedge pressure (mm Hg)§¶	24.2±7.1	24.3±7.7
Cardiac index (liters/min/m²)§‖	1.8±0.7	1.7±0.5
Left ventricular ejection fraction (%)**	29.1±10.6	32.5±13.9
Number of diseased vessels (%)††		
≤1	14.0	11.5
2	21.7	24.0
3	64.3	64.6
Left main coronary artery disease (%)‡‡	23.4	17.5

TREATMENT	REVASCULARIZATION (N=152)	MEDICAL THERAPY (N=150)
CPR, VT, or VF before randomization (%)*	32.7	23.9
Thrombolytic therapy (%)	49.3	63.3
Inotropes or vasopressors (%)	99.3	98.6
Intraaortic balloon counterpulsation (%)	86.2	86.0
Pulmonary-artery catheterization (%)	93.4	96.0
Left ventricular assist device (%)†	3.6	0.9
Heart transplantation (%)	2.0	0.7
Coronary angiography (%)	96.7	66.7
Angioplasty (%)	54.6	14.0
Stent placed‡	35.7	52.3
Platelet glycoprotein IIb/IIIa receptor antagonist§	41.7	25.0
Coronary-artery bypass grafting (%)	37.5	11.3
Angioplasty or coronary-artery bypass grafting (%)	86.8	25.3
Median time from randomization to revascularization (hr)¶	1.4 (0.6–2.8)	102.8 (79.0–162.0)

Kardiogenní šok : časná revaskularizace

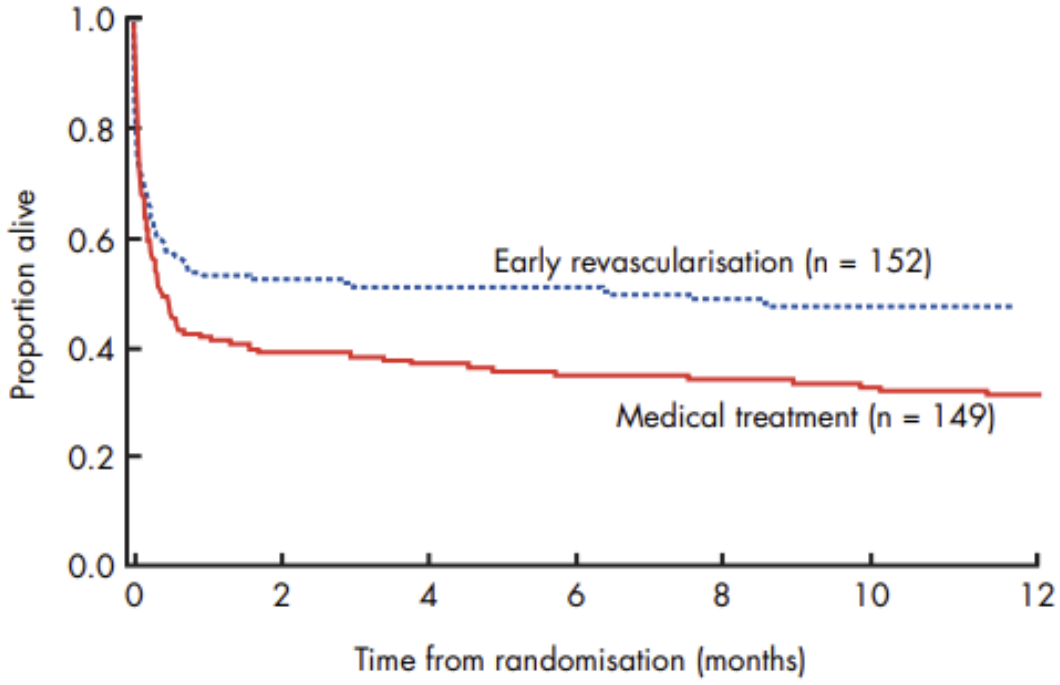
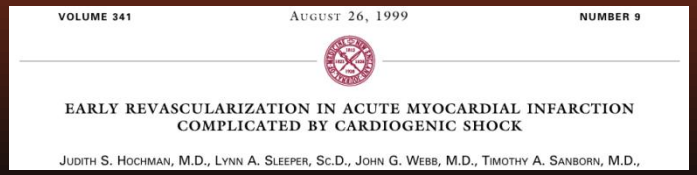


Figure 2 Kaplan-Meier curve showing 12 month survival in the early revascularisation and initial medical stabilisation arms of the SHOCK trial. Reproduced from Hochman *et al*,[2] with permission of the American Medical Association.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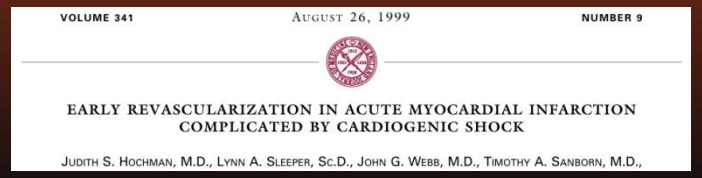
Kardiogenní šok : časná revaskularizace – KOMU?

TABLE 4. MORTALITY AMONG STUDY PATIENTS.*

OUTCOME AND SUBGROUP	REVASCULARIZATION	MEDICAL THERAPY	DIFFERENCE BETWEEN GROUPS (95% CI)	RELATIVE RISK (95% CI)	P VALUE
	percent (number in subgroup)		percent		
30-day mortality					
Total	46.7 (152)	56.0 (150)	−9.3 (−20.5 to 1.9)	0.83 (0.67 to 1.04)	0.11
Age <75 yr	41.4 (128)	56.8 (118)	−15.4 (−27.8 to −3.0)	0.73 (0.56 to 0.95)	0.01†
Age ≥75 yr	75.0 (24)	53.1 (32)	+21.9 (−2.6 to 46.4)	1.41 (0.95 to 2.11)	
6-mo mortality‡					
Total	50.3 (151)	63.1 (149)	−12.8 (−23.2 to −0.9)	0.80 (0.65 to 0.98)	0.027
Age <75 yr	44.9 (127)	65.0 (117)	−20.1 (−31.6 to −7.1)	0.70 (0.56 to 0.89)	0.003†
Age ≥75 yr	79.2 (24)	56.3 (32)	+22.9 (0.7 to 46.6)	1.41 (0.97 to 2.03)	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Kardiogenní šok : časná revaskularizace – JAK?

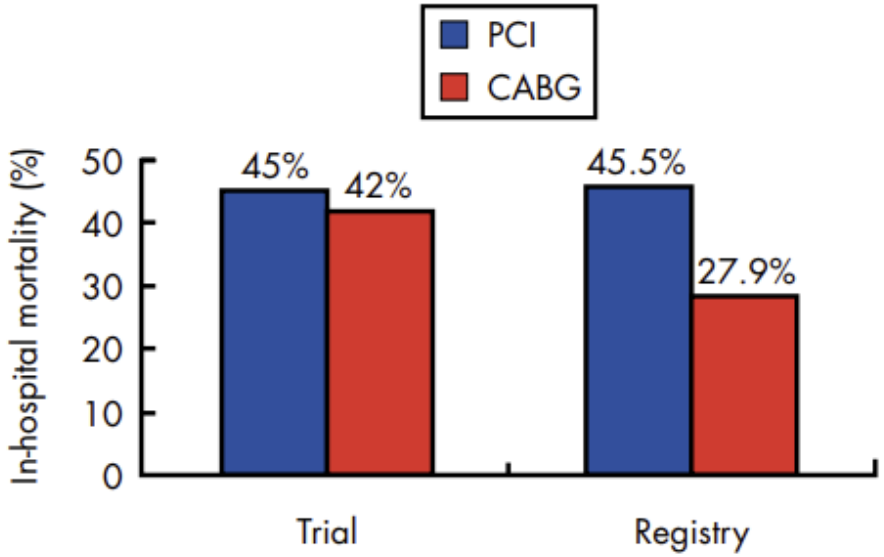

Figure 4 In-hospital mortality with percutaneous coronary intervention (PCI) and coronary artery bypass graft surgery (CABG) in the early revascularisation arm of the randomised SHOCK trial compared to the non-randomised larger SHOCK registry.

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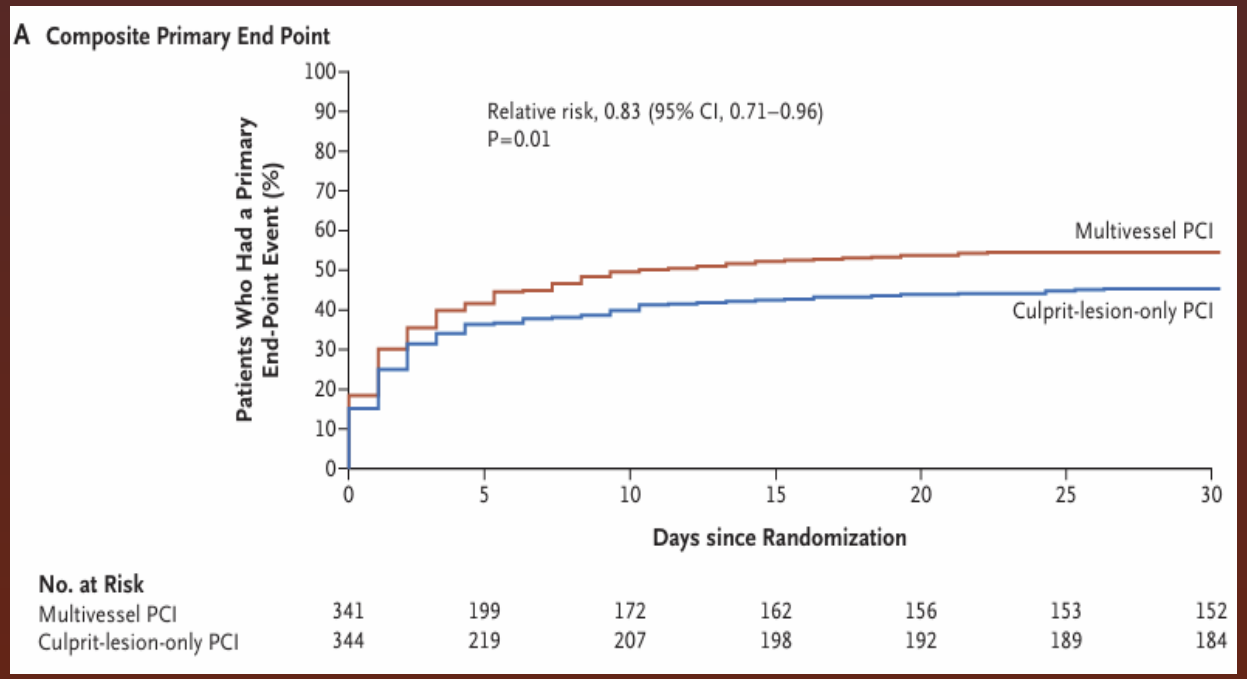
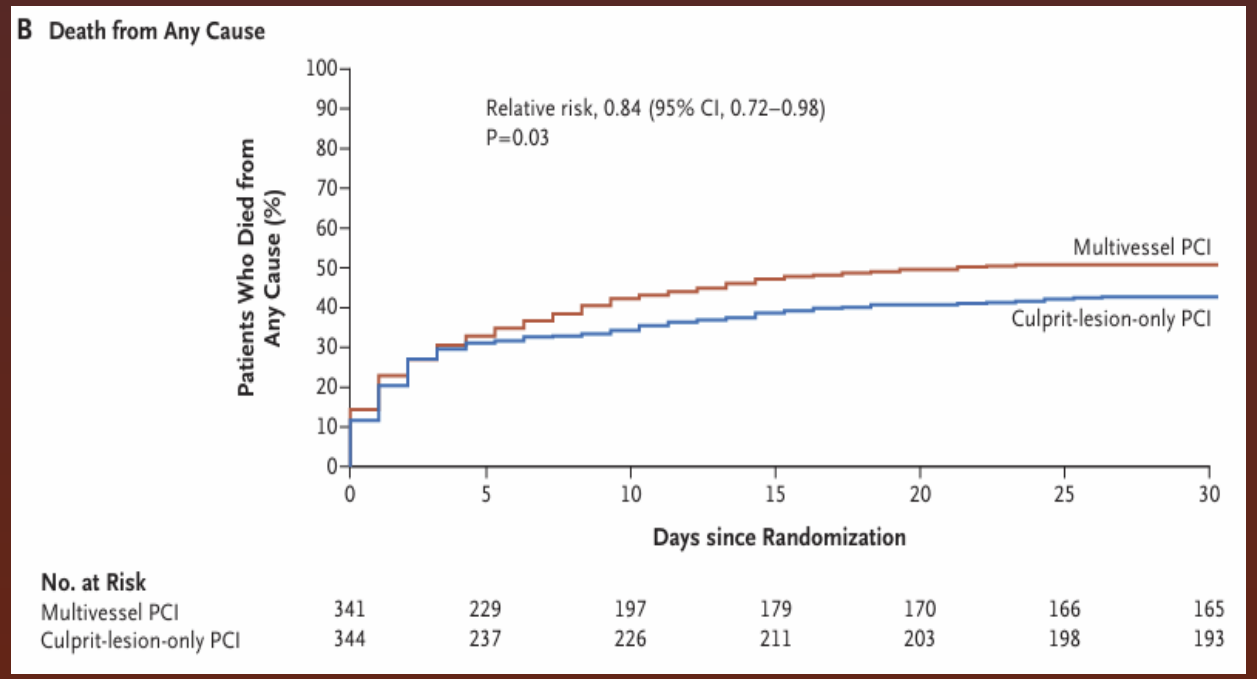
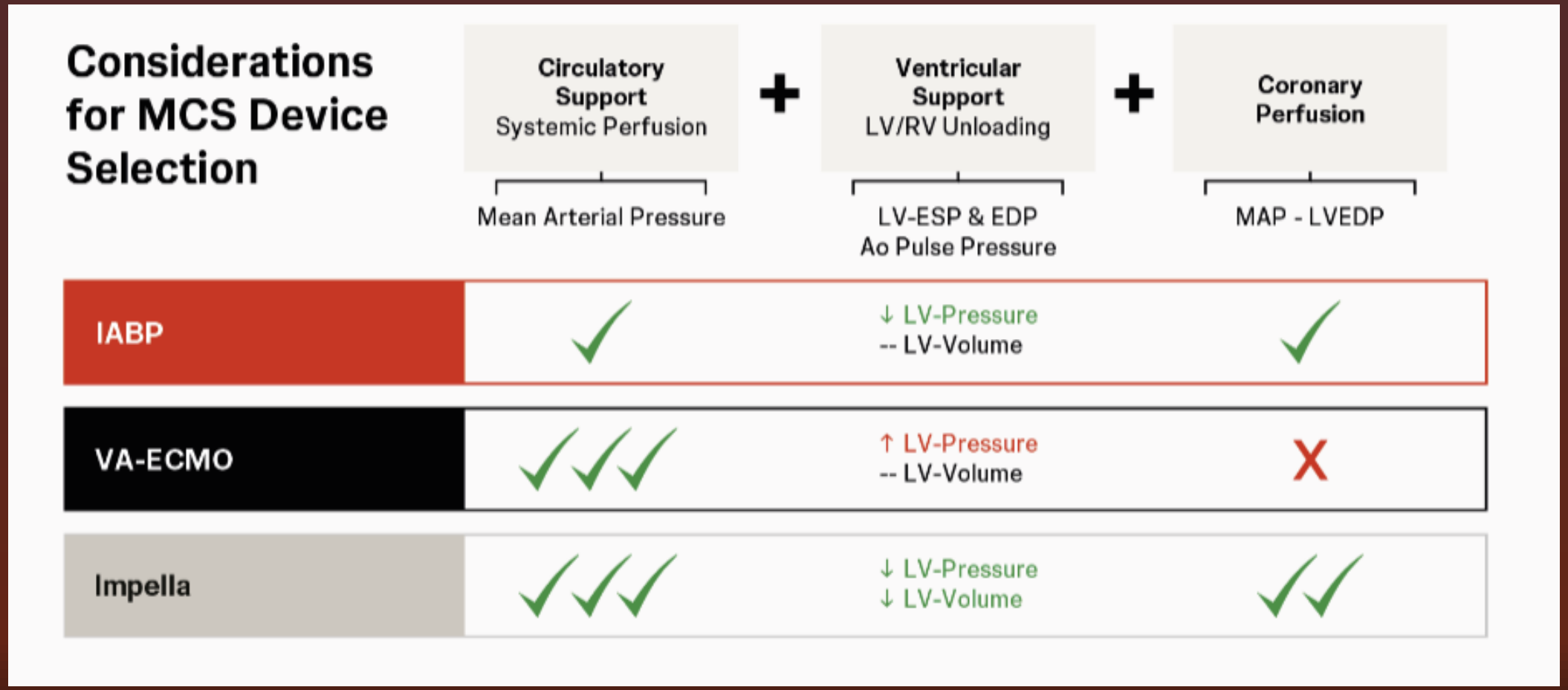
Kardiogenní šok : časná revaskularizace (CULPRIT-SHOCK)





N Engl J Med 2017;377:2419-32

Kardiogenní šok : časné použití MCS



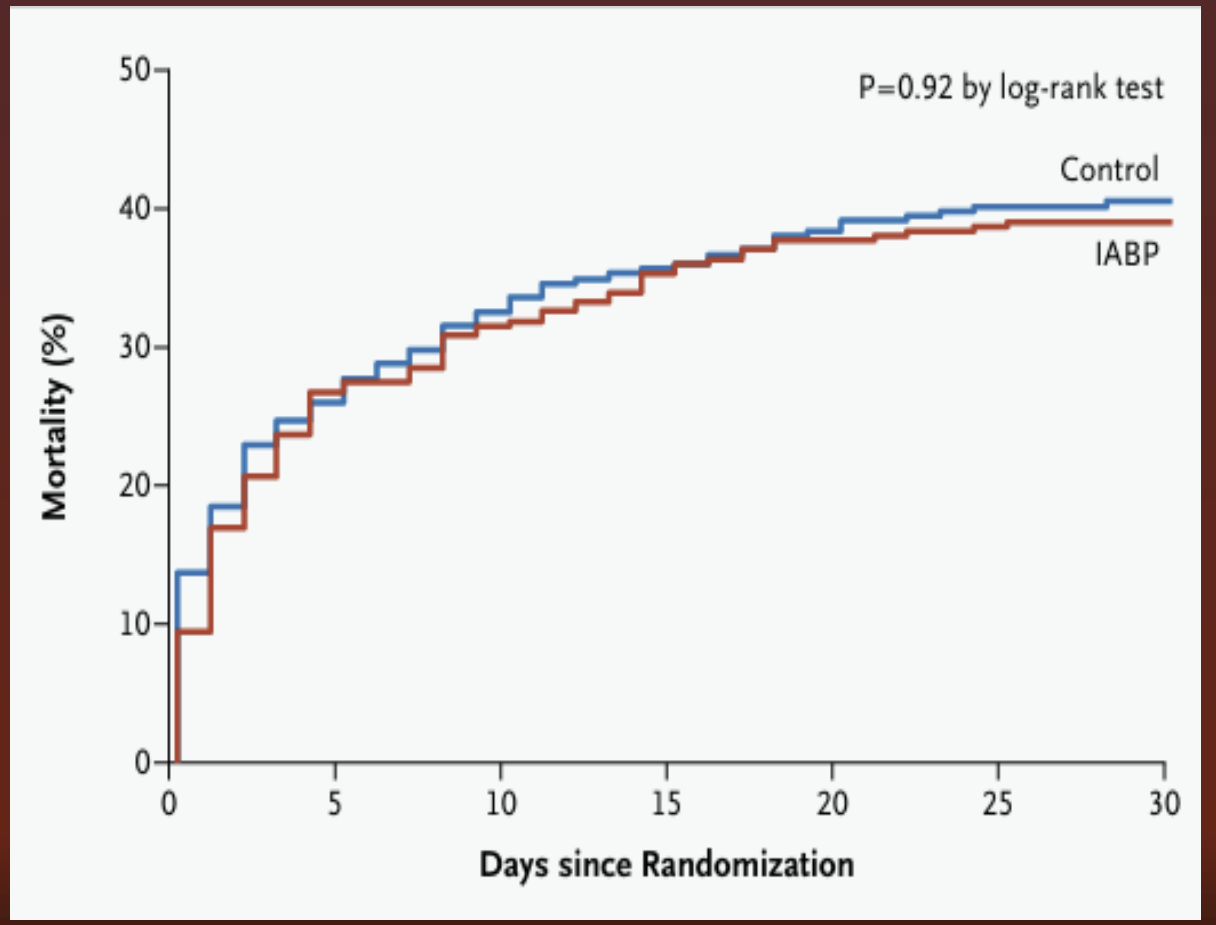
Kardiogenní šok : časné použití MCS (IABP)

ESTABLISHED IN 1812 OCTOBER 4, 2012 VOL. 367 NO. 14

Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

Holger Thiele, M.D., Uwe Zeymer, M.D., Franz-Josef Neumann, M.D., Miroslaw Ferenc, M.D.,

Outcome	IABP (N=300)	Control (N=298)	P Value	Relative Risk with IABP (95% CI)
	number (percent)			
Primary end point: all-cause mortality at 30 days	119 (39.7)	123 (41.3)	0.69	0.96 (0.79–1.17)
Reinfarction in hospital	9 (3.0)	4 (1.3)	0.16	2.24 (0.70–7.18)
Stent thrombosis in hospital	4 (1.3)	3 (1.0)	0.71	1.32 (0.30–5.87)
Stroke in hospital	2 (0.7)	5 (1.7)	0.28	0.40 (0.08–2.03)
Ischemic	2 (0.7)	4 (1.3)	0.45	0.49 (0.09–2.71)
Hemorrhagic	0	1 (0.3)	0.50	—
Peripheral ischemic complications requiring intervention in hospital	13 (4.3)	10 (3.4)	0.53	1.29 (0.58–2.90)
Bleeding in hospital*				
Life-threatening or severe	10 (3.3)	13 (4.4)	0.51	0.76 (0.34–1.72)
Moderate	52 (17.3)	49 (16.4)	0.77	1.05 (0.74–1.50)
Sepsis in hospital	47 (15.7)	61 (20.5)	0.15	0.77 (0.54–1.08)

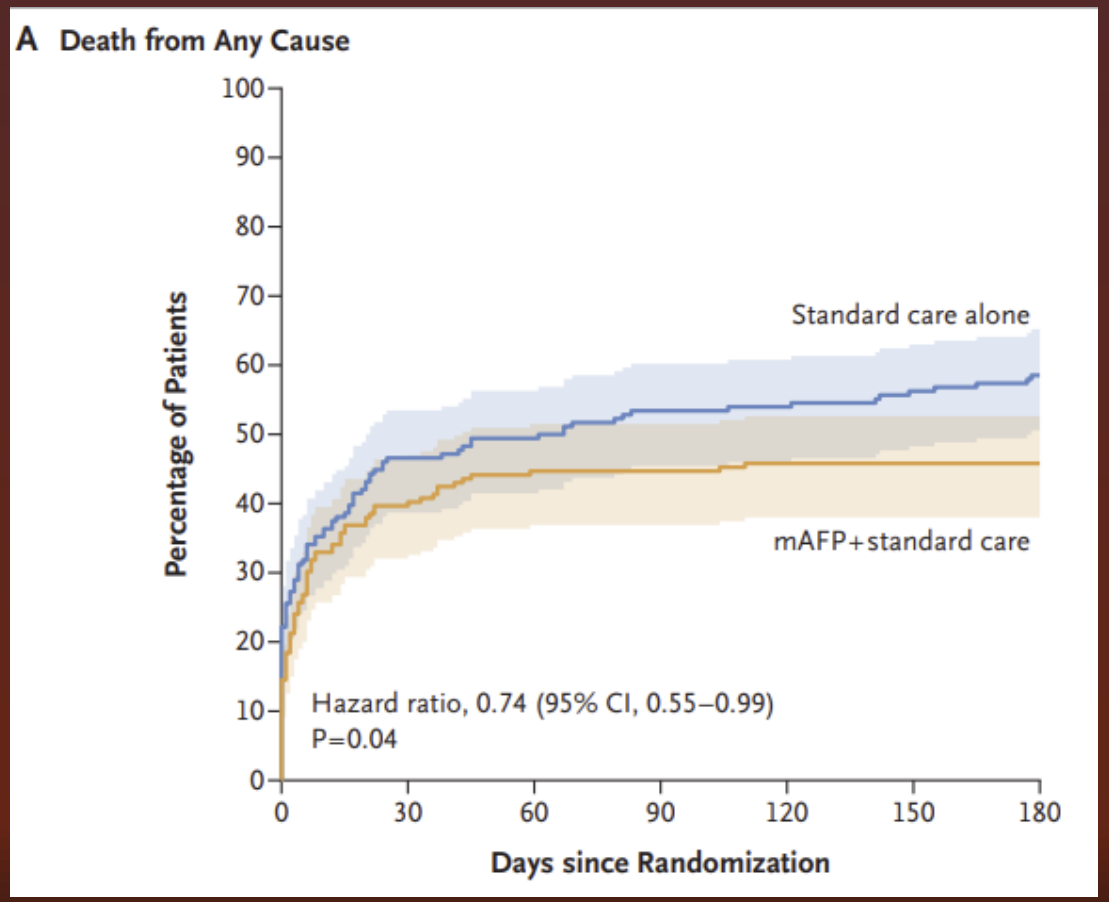


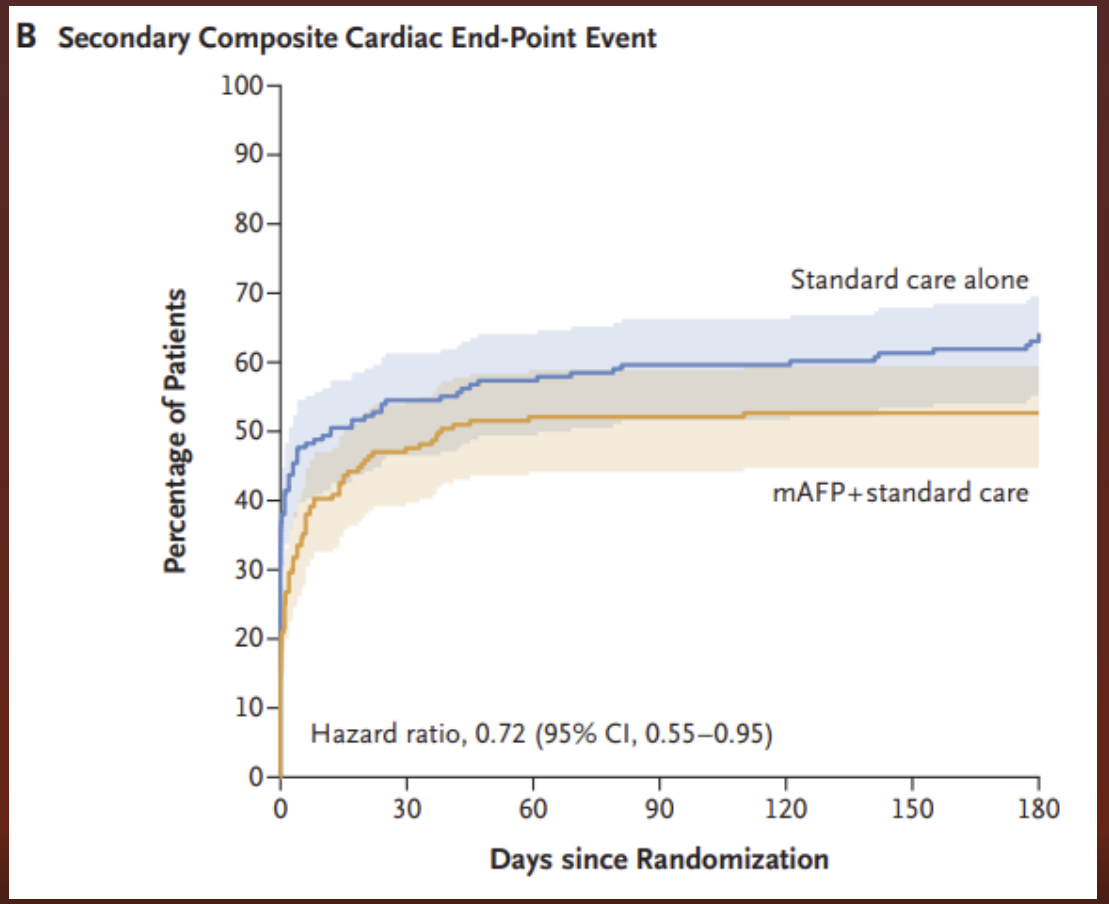
Kardiogenní šok : časné použití MCS (ECMO)

Table 2. Comparative table of studies on the impact of VA-ECMO on survival.

Authors	Year of Randomization	Type of Study	Patient Population	Primary Endpoint	Group	Size	LV unloading Management	30 Day—Mortality
Brunner S. et al. [20]	2019	monocentric, open-label, randomized controlled	CS complicating AMI *	To assess the effect of VA-ECMO on 30-day mortality	ECLS group	21	not reported	19% in the ECLS group and 33% in the control group. ($p = 0.37$)
					no-ECLS group	21		
Banning A.S. et al. (EUROSHOCK trial) [22]	January 2020—January 2022	multicentric, open-label, randomized controlled	CS complicating AMI **	To assess the effect of VA-ECMO on 30-day mortality	ECLS group	17	IABP in all VA-ECMO patients	43.8% in the ECLS group and 61.1% in the control group. ($p = 0.22$)
					no-ECLS group	18		
Ostadal P. et al. (ECMO-CS trial) [23]	September 2014—January 2022	multicentric, open-label, randomized controlled	all non-surgical causes of CS ***	To compare the immediate implementation of VA-ECMO with early conservative therapy	immediate ECMO	58	LV unloading at the discretion of the physician	50% in the immediate ECMO and 47.5% in the early conservative group.
					early conservative therapy	59		
Thiele H. et al. (ECLS-SHOCK trial) [24]	June 219—November 2022	multicentric, open-label, randomized controlled	CS complicating AMI ****	To assess the early addition of ECLS to early revascularization	ECLS group	211	Unloading rate in ECLS group 5.8%	47.8% in the ECLS group and 49.0% in the control group ($p = 0.81$).
					no-ECLS group	209		

Kardiogenní šok : časné použití MCS (Impella)





N Engl J Med 2024;390:1382-93.

Kardiogenní šok : časné použití MCS (Impella)

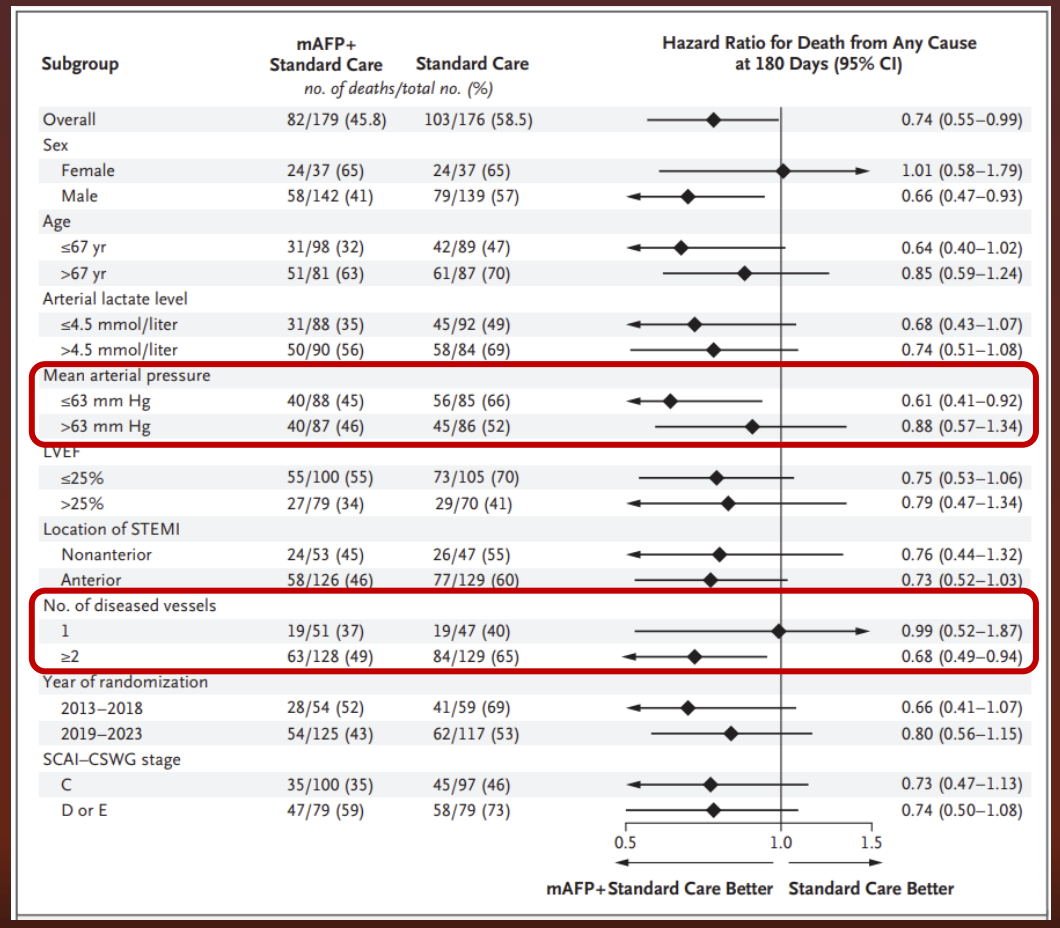


Table 3. End Points and Adverse Events in the Intention-to-Treat Population.*

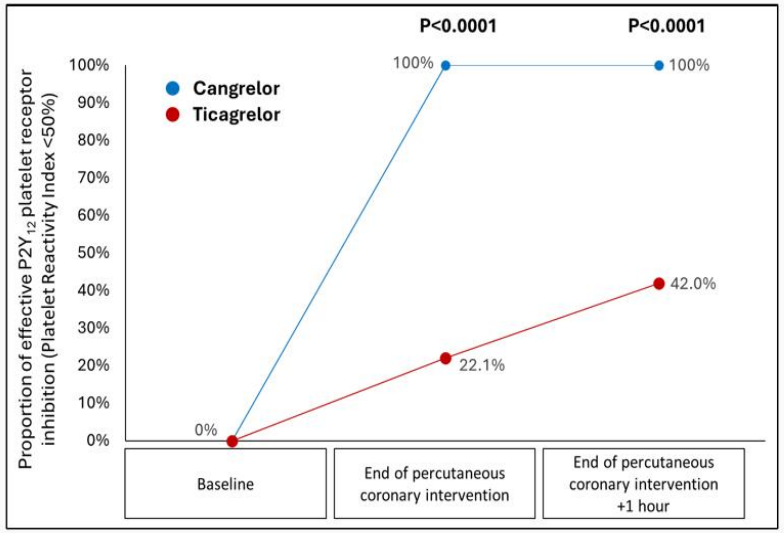
Event	Microaxial Flow Pump plus Standard Care (N=179)	Standard Care Alone (N=176)	Effect Size (95% CI)†
Primary end point: death from any cause at 180 days — no. (%)	82 (45.8)	103 (58.5)	0.74 (0.55 to 0.99)‡
Secondary end point			
Composite cardiac end point — no. (%)§	94 (52.5)	112 (63.6)	0.72 (0.55 to 0.95)
No. of days alive and out of the hospital (range)¶	82 (0 to 177)	73 (0 to 179)	8 (–8 to 25)
Adverse events			
Composite safety end point — no. (%)‖	43 (24.0)	11 (6.2)	4.74 (2.36 to 9.55)
Moderate or severe bleeding — no. (%)**	39 (21.8)	21 (11.9)	2.06 (1.15 to 3.66)
Limb ischemia — no. (%)	10 (5.6)	2 (1.1)	5.15 (1.11 to 23.84)
Renal-replacement therapy — no. (%)	75 (41.9)	47 (26.7)	1.98 (1.27 to 3.09)
Stroke — no. (%)	7 (3.9)	4 (2.3)	1.75 (0.50 to 6.01)
Cardioversion after ventricular tachycardia or fibrillation — no. (%)	59 (33.0)	52 (29.5)	1.17 (0.75 to 1.83)
Sepsis with positive blood culture†† — no. (%)	21 (11.7)	8 (4.5)	2.79 (1.20 to 6.48)

N Engl J Med 2024;390:1382-93.

Kardiogenní šok : antitrombotická léčba

PRIMARY LABORATORY ENDPOINT

VASP-Platelet Reactivity Index < 50% at the end of pPCI



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PRIMARY PCI – PROCEDURAL RESULTS

	Cangrelor (N=298)	Ticagrelor (N=307)	P superiority
TIMI flow after intervention			
0	7 (2.4%)	10 (3.4%)	0.456
1	9 (3.1%)	18 (6.1%)	0.091
2	33 (11.5%)	48 (16.3%)	0.100
3	239 (83.0%)	218 (74.1%)	**0.009**
Complications of primary PCI			
Any complications	22 (7.4%)	43 (14.0%)	**0.009**
Slow flow or no reflow	1 (0.3%)	11 (3.6%)	**0.004**
Distal embolization	4 (1.3%)	7 (2.3%)	0.388
Periprocedural stent thrombosis	1 (0.3%)	7 (2.3%)	**0.037**
Other complications	16 (5.4%)	18 (5.9%)	0.792
Procedural result			
Optimal	239 (83.0%)	220 (75.1%)	**0.014**
Suboptimal	39 (13.5%)	58 (19.8%)	0.052
Failure	10 (3.5%)	15 (5.1%)	0.345

Core Lab (independent experts, blinded to the study arms) evaluated angiograms.

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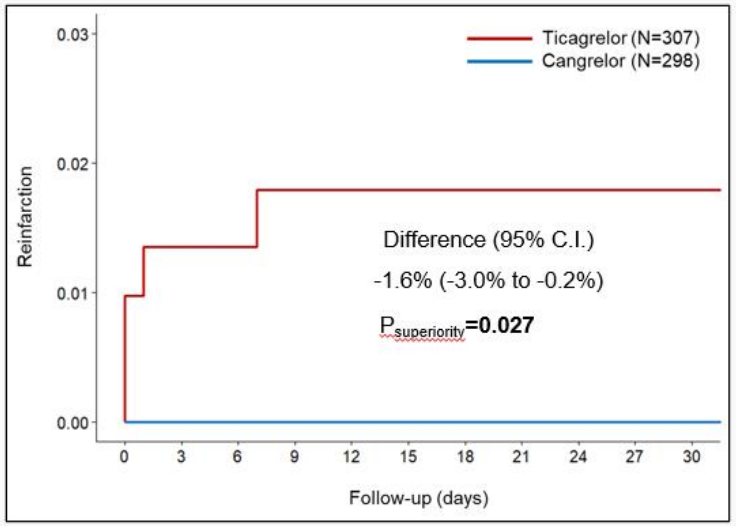
Zuzana MOTOVSKA, August 31st, 2025

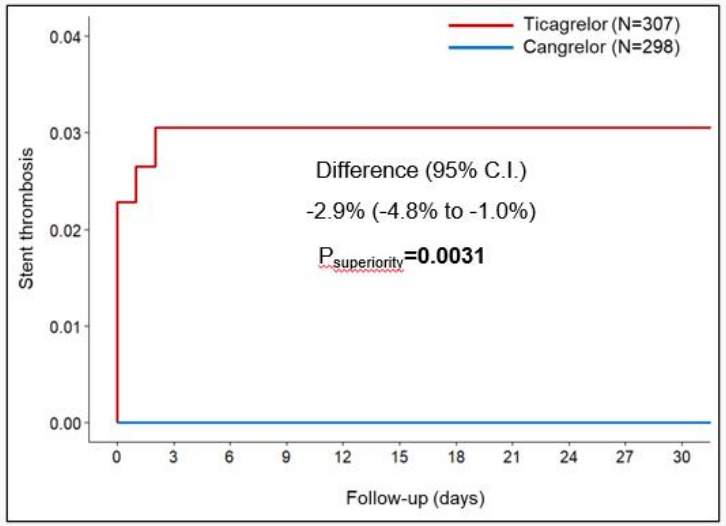
Kardiogenní šok : antitrombotická léčba

DAPT SHOCK AMI

REINFARCTION



STENT THROMBOSIS



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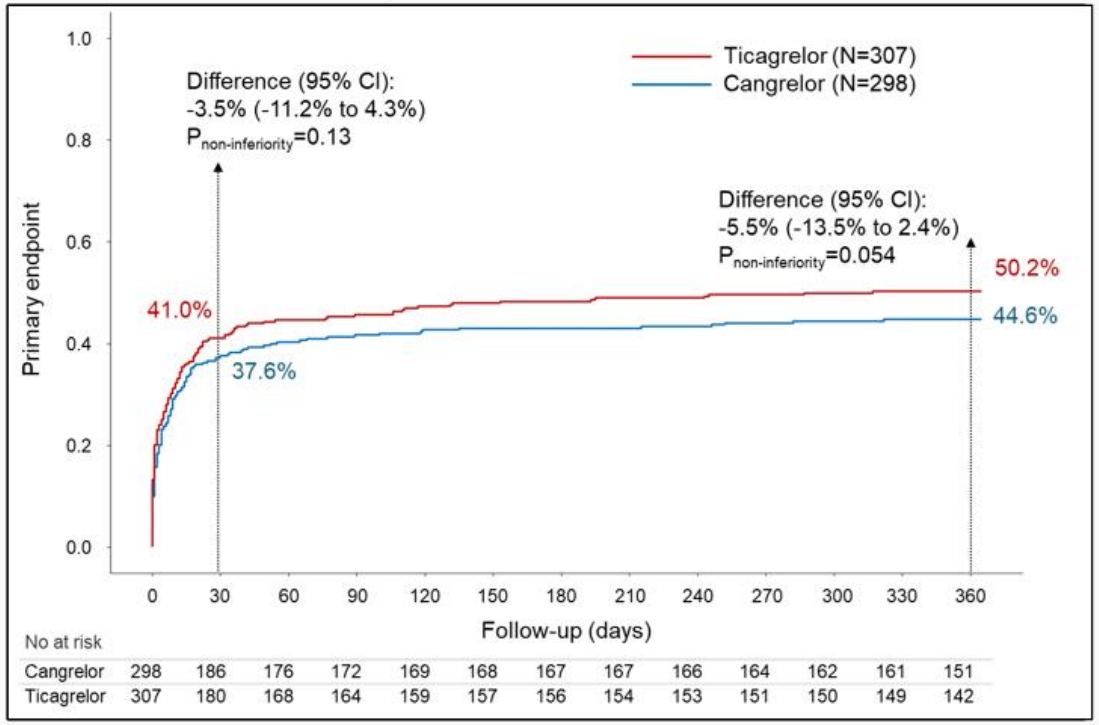
Kardiogenní šok : antitrombotická léčba

DAPT SHOCK AMI

PRIMARY CLINICAL ENDPOINT (mITT)

Death, MI, or Ischemic Stroke at 30 days



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Zuzana MOTOVSKA, August 31st, 2025

Kardiogenní šok : antitrombotická léčba

SECONDARY EFFICACY ENDPOINTS (Per Protocol)

DAPT SHOCK AMI

*Cangrelor Infusion: 4 µg per kilogram **over at least 2 hours***

	Cangrelor (N=287) - 11	Ticagrelor (N=307)	Difference (95% C.I.)	P non-infer.	P super.
30-day					
KEY SECONDARY EFFICACY ENDPOINT *Death, MI, *UVR of IRA, Stent Thrombosis, or Ischemic Stroke*	102 (35.5%)	128 (41.7%)	-5.4% (-13.2% to 2.4%)	0.055	..
Cardiovascular death	65 (22.6%)	95 (30.9%)	-8.3% (-15.4% to -1.2%)	**0.0052**	**0.023**
All cause death	99 (34.5%)	118 (38.9%)	-4.4% (-12.2% to 3.3%)	0.085	..
Ischemic stroke	4 (1.4%)	1 (0.3%)	1.1% (-0.4% to 2.6%)	0.53	..
CV death, MI, urgent revascularization, and HF	81 (28.2%)	116 (37.8%)	-9.6% (-17.1% to -2.0%)	**0.0036**	**0.013**
365-day					
KEY SECONDARY EFFICACY ENDPOINT	125 (43.6%)	158 (51.5%)	-7.3% (-15.3% to 0.8%)	**0.022**	0.077
Cardiovascular death	69 (24.0%)	102 (33.2%)	-9.8% (-16.4% to -2.0%)	**0.0038**	**0.014**
All cause death	119 (41.5%)	147 (48.5%)	-7.1% (-15.1% to 1.0%)	**0.025**	0.085
Ischemic stroke	6 (2.1%)	1 (0.3%)	1.8% (0.0% to 3.5%)	0.80	..
CV death, MI, urgent revascularization, and HF	88 (30.7%)	128 (41.7%)	-11.0% (-18.7% to -3.4%)	**0.0014**	**0.0056**

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* Urgent Revascularization of Infarct-Related Artery

Non-inferiority margin = 1%

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CONCLUSIONS

DAPT SHOCK AMI

DAPT-SHOCK-AMI, the first-ever randomized trial testing antiplatelet therapy in AMI-CS, showed that compared with ticagrelor:

- Cangrelor provides immediate, effective platelet inhibition, with no high-on-treatment platelet reactivity present.
- While the 1° clinical EP at 30 days was not met, cangrelor improved the final outcome of primary PCI and appears to reduce the risk of early ischemic events without increasing major bleeding.
- Treatment with cangrelor improves LV function, decreases disability, and costs.
- Larger trials are needed to confirm the findings related to numerically lower CV mortality at one year, which if verified, could represent a major advancement in the treatment of cardiogenic shock.

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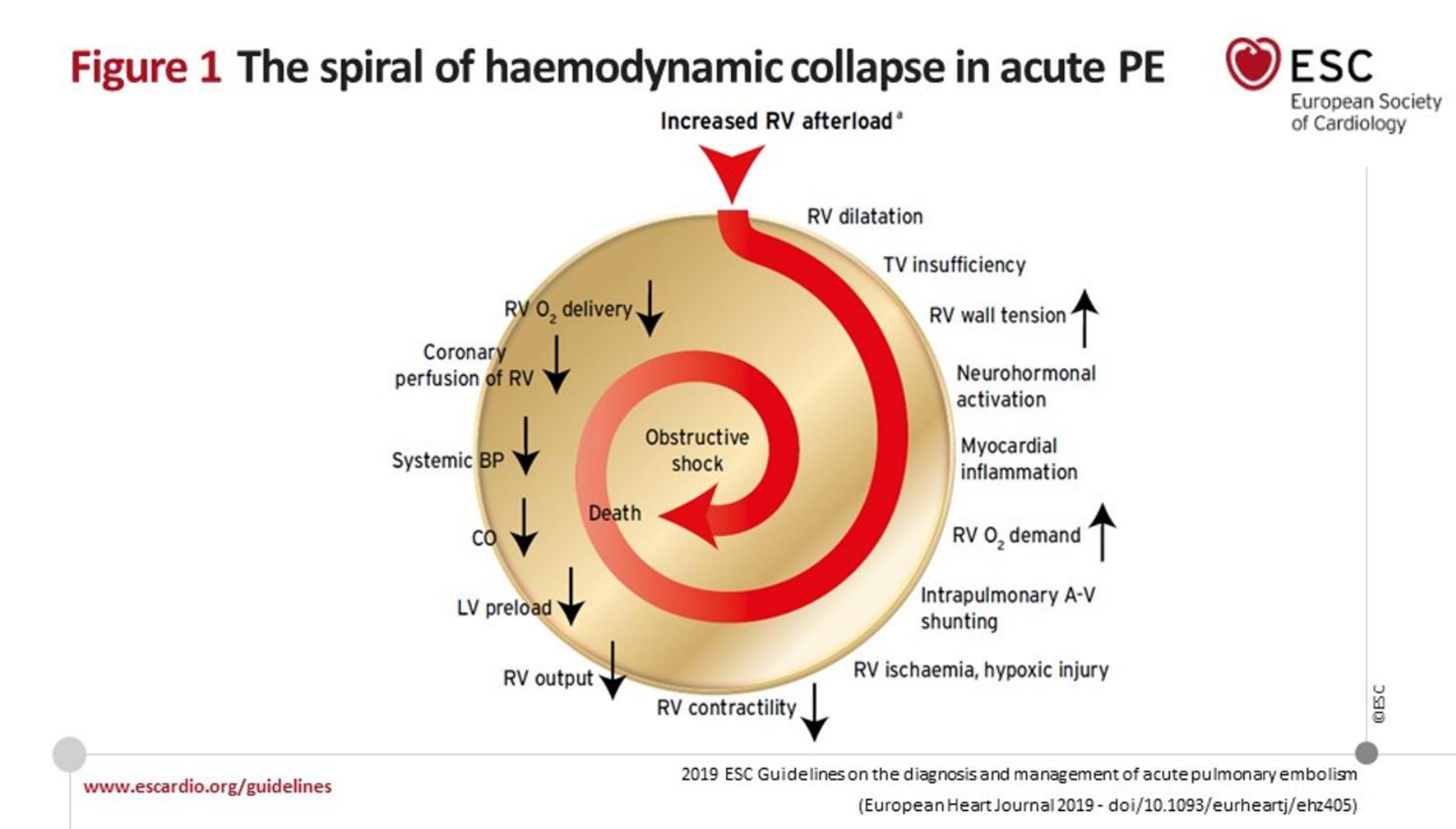
Kardiogenní šok : plicní embolie



Kardiogenní šok : plicní embolie

Table 10 Treatment of RV failure in acute high-risk PE (1)

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Strategy	Properties and use	Caveats
Volume optimization		
Cautious volume loading, saline, or Ringer's lactate, up to 500 mL over 15–30 min	Consider in patients with normal-to-low central venous pressure (due, for example, to concomitant hypovolaemia)	Volume loading can overdistend the RV, worsen ventricular interdependence, and reduce CO
Vasopressors and inotropes		
Norepinephrine, 0.2–1.0 µg/kg/min	Increases RV inotropy, systemic BP; promotes positive ventricular interactions; restores coronary perfusion gradient	Excessive vasoconstriction may worsen tissue perfusion
Dobutamine, 2–20 µg/kg/min	Increases RV inotropy, lowers filling pressures	May aggravate arterial hypotension if used alone, without a vasopressor; may trigger or aggravate arrhythmias

CO = cardiac output; RV = right ventricle/ventricular.

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Kardiogenní šok : plicní embolie

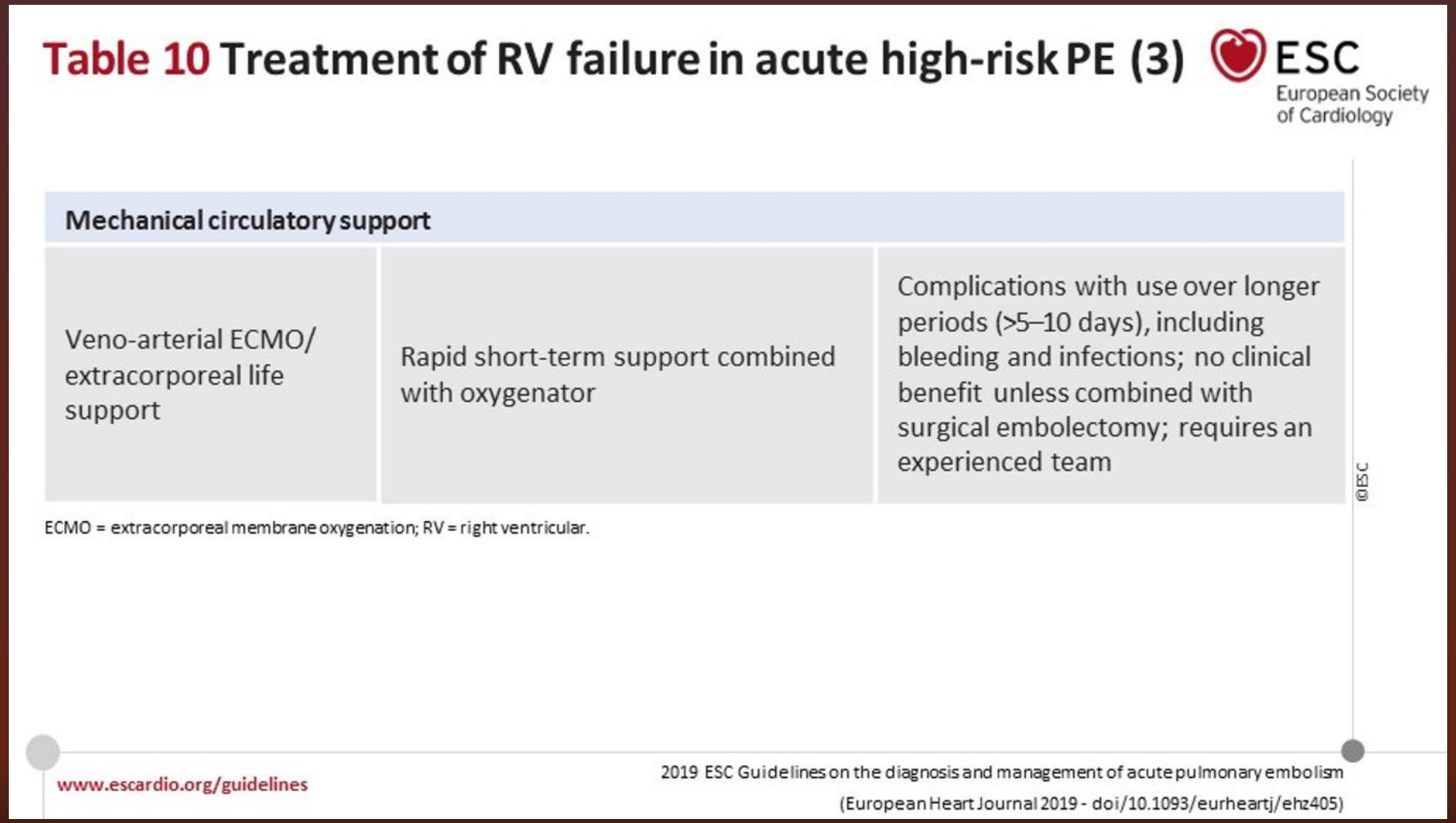

Table 10 Treatment of RV failure in acute high-risk PE (3)

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Mechanical circulatory support		
Veno-arterial ECMO/ extracorporeal life support	Rapid short-term support combined with oxygenator	Complications with use over longer periods (>5–10 days), including bleeding and infections; no clinical benefit unless combined with surgical embolectomy; requires an experienced team

ECMO = extracorporeal membrane oxygenation; RV = right ventricular.

Kardiogenní šok: plicní embolie

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Recommendations for acute-phase treatment of high-risk PE (2)

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Recommendations	Class	Level
Percutaneous catheter-directed treatment should be considered for patients with high-risk PE, in whom thrombolysis is contraindicated or has failed.	IIa	C
Norepinephrine and/or dobutamine should be considered in patients with high-risk PE.	IIa	C
ECMO may be considered, in combination with surgical embolectomy or catheter-directed treatment, in patients with PE and refractory circulatory collapse or cardiac arrest.	IIb	C

ECMO = extracorporeal membrane oxygenation.

2019 ESC Guidelines on the diagnosis and management of acute pulmonary embolism
(European Heart Journal 2019 - doi/10.1093/eurheartj/ehz405)

Kardiogenní šok : plicní embolie

Studie	Typ	Počet pacientů	Status	Populace	Intervence	Komparátor	Primární endpoint	Výsledek (effect)		p-value
FLASH	Registry	~1000	Dokončeno (US)	Intermediate + high-risk	FlowTriever	-	MAE + hemodynamika (48h)	MAE 1.8 %, mortalita 0.8%	MAE 1.8 %, mortalita 0.8 % (30 dní), významné zlepšení RV/LV a mPAP	ns
FLAME	Observační	115	Dokončeno	High-risk	FlowTriever	Standard care	In-hospital kompozit	17% vs 63.9%	17 % vs 63.9 % (výrazně lepší FlowTriever), mortalita 1.9 % vs 29.5 %	<0.01
PEERLESS	RCT	550	Dokončeno	Intermediate-risk	FlowTriever	CDT	Win ratio	WR 5.01	Win ratio 5.01 (p<0.001), méně ICU, bez rozdílu v mortalitě/krvácení	<0.001
PEERLESS II	RCT	1200	Probíhá	Intermediate-risk	FlowTriever	Antikoagulace	Klinický kompozit	-	zatím bez výsledků	-
HI-PEITHO	RCT	544	Dokončeno	Intermediate-high risk	EKOS + AC	AC	Klinický kompozit (7 dní)	4.0% vs 10.3%	4.0 % vs 10.3 % (p=0.005) → ↓ klinické zhoršení (~60 % redukce), bez ↑ ICH	0.005
STRIKE-PE	Registry	>150	Probíhá	Intermediate + high-risk	Indigo	-	RV/LV + MAE	RV/LV ↓	RV/LV výrazně ↓ (p<0.001), MAE ~2.7 %	<0.001
STORM-PE	RCT	100	Dokončeno	Intermediate-high risk	Indigo + AC	AC	RV/LV změna	Δ 0.27	Δ RV/LV 0.27 (p<0.001), MAE bez rozdílu, trend více úmrtí v MT větvi (ns)	<0.001

Kardiogenní šok : plicní embolie

ORIGINAL ARTICLE

Ultrasound-Facilitated, Catheter-Directed Fibrinolysis for Acute Pulmonary Embolism

K. Rosenfield,[1] F.A. Klok,[2,3] G. Piazza,[4,5] A.S.P. Sharp,[6,7] F. Ní Áinle,[6] M.R. Jaff,[8] S. Barco,[3,9] S.Z. Goldhaber,[4,5] N. Kucher,[9] I.M. Lang,[10] I. Schmidtmann,[11] K.M. Sterling,[8] A. Araszkiewicz,[12] V. Arora,[13] R. Cires-Drouet,[14] J. Coghlan,[15] L. Hobohm,[3,16] W.D. Ito,[17] K. Jacobson,[18] C. Kaiser,[19] G. Kopec,[20,21] K. Marx,[22] S. McElwee,[23] N. Meneveau,[24-26] P. Monteleone,[27] J.M. Montero-Cabezas,[28] C.B. Olivier,[29,30] J. Park,[31] M. Roik,[32] R. Sakhuja,[1] A. Tego,[22] M. Theurl,[33] G. Visveswaran,[34] J.A. Vos,[35] M.N. Young,[36,37] F.M. Asch,[38] and S.V. Konstantinides,[3,39] for the HI-PEITHO Investigators*

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Table 2. Clinical Efficacy Outcomes (Intention-to-Treat Population).*

Outcome	Intervention (N=273)		Control (N=271)		Relative Risk (95% CI)†
	no. of patients	*% (95% CI)†*	*no. of patients*	*% (95% CI)†*	
Any primary-outcome event	11	4.0 (2.3–7.1)	28	10.3 (7.2–14.5)	0.39 (0.20–0.77)‡
Components of the primary outcome					
Pulmonary embolism–related death	3	1.1 (0.4–3.2)	1	0.4 (0.1–2.1)	3.0 (0.3–28.5)
Cardiorespiratory decompensation or collapse	10	3.7 (2.0–6.6)	28	10.3 (7.2–14.5)	0.4 (0.2–0.7)
Recurrence of pulmonary embolism	1	0.4 (0.1–2.0)	1	0.4 (0.1–2.1)	1.0 (0.1–15.8)

Table 3. Cumulative Major Bleeding Events through 30 Days (Treated Population).*

Event	Intervention (N=271)	Control (N=271)	Relative Risk (95% CI)†	P Value‡
	no. of patients (%)			
Major bleeding according to ISTH criteria				
Within 72 h	10 (3.7)	4 (1.5)	2.5 (0.8–7.9)	0.17
Within 7 days	11 (4.1)	6 (2.2)	1.8 (0.7–4.9)	0.32
Within 30 days	11 (4.1)	8 (3.0)	1.4 (0.6–3.4)	0.64
Moderate-to-severe bleeding within 7 days according to GUSTO criteria	9 (3.3)	4 (1.5)	2.3 (0.7–7.2)	0.26
Ischemic stroke				
Within 7 days	1 (0.4)	0	NE	1.00
Within 30 days	1 (0.4)	0	NE	1.00
Intracranial hemorrhage				
Within 7 days	0	0	NE	1.00
Within 30 days	0	0	NE	1.00

Krotitelé kardiogenních šoků