Percorso shock cardiogeno
4 ottobre 2019

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SC Cardiologia Ospedale Santa Maria Annunziata
Azienda USL Toscana Centro

Shock cardiogeno

Ipoperfusione sistemica tessutale secondaria a una gettata cardiaca ridotta nonostante un volume circolatorio e pressioni di riempimento del ventricolo sinistro
normali o aumentati.

SHOCK Trial[9]*

Clinical criteria:
SBP <90 mm Hg for ≥30 min OR
Support to maintain SB
AND
End-organ hypoperfusi
<30 mL/h or cool extre

Hemodynamic criteria:
CI of ≤2.2 L·min^{-1}·m^{-2} A
PCWP ≥15 mm Hg

Hochma

IABP-SHOCK II[1]†

Clinical criteria:
SBP <90 mm Hg for ≥30 min OR
intain SBP >90 mm Hg

ngestion

erfusion (altered
ammy skin and
put <30 mL/h, or

Thiele H et al; NEJM 2012

ESC HF Guidelines[15]

SBP <90 mm Hg with adequate volume and clinical or laboratory signs of hypoperfusion

Clinical hypoperfusion:
Cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure

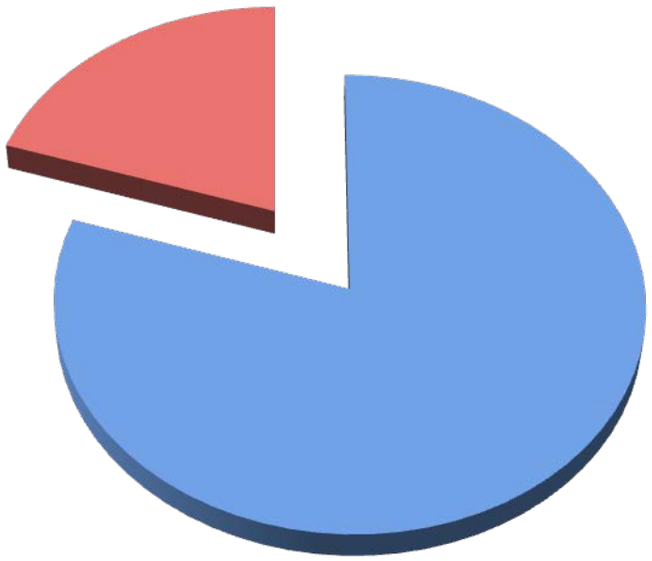
Laboratory hypoperfusion:
Metabolic acidosis, elevated serum lactate, elevated serum creatinine

Ponikowski et al; EHJ 2016

Shock cardiogeno

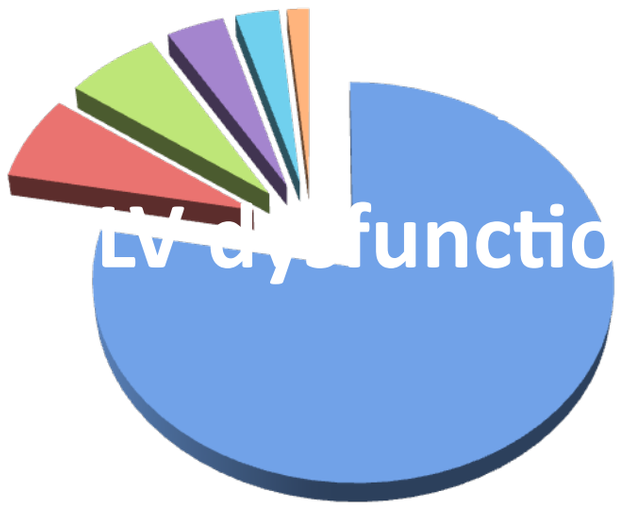
Non-Ischaemic 20%

- Valvulopathy
- Cardiomyopathies
- Myocarditis
- Pulmonary Embolysm



Ischaemic 80%

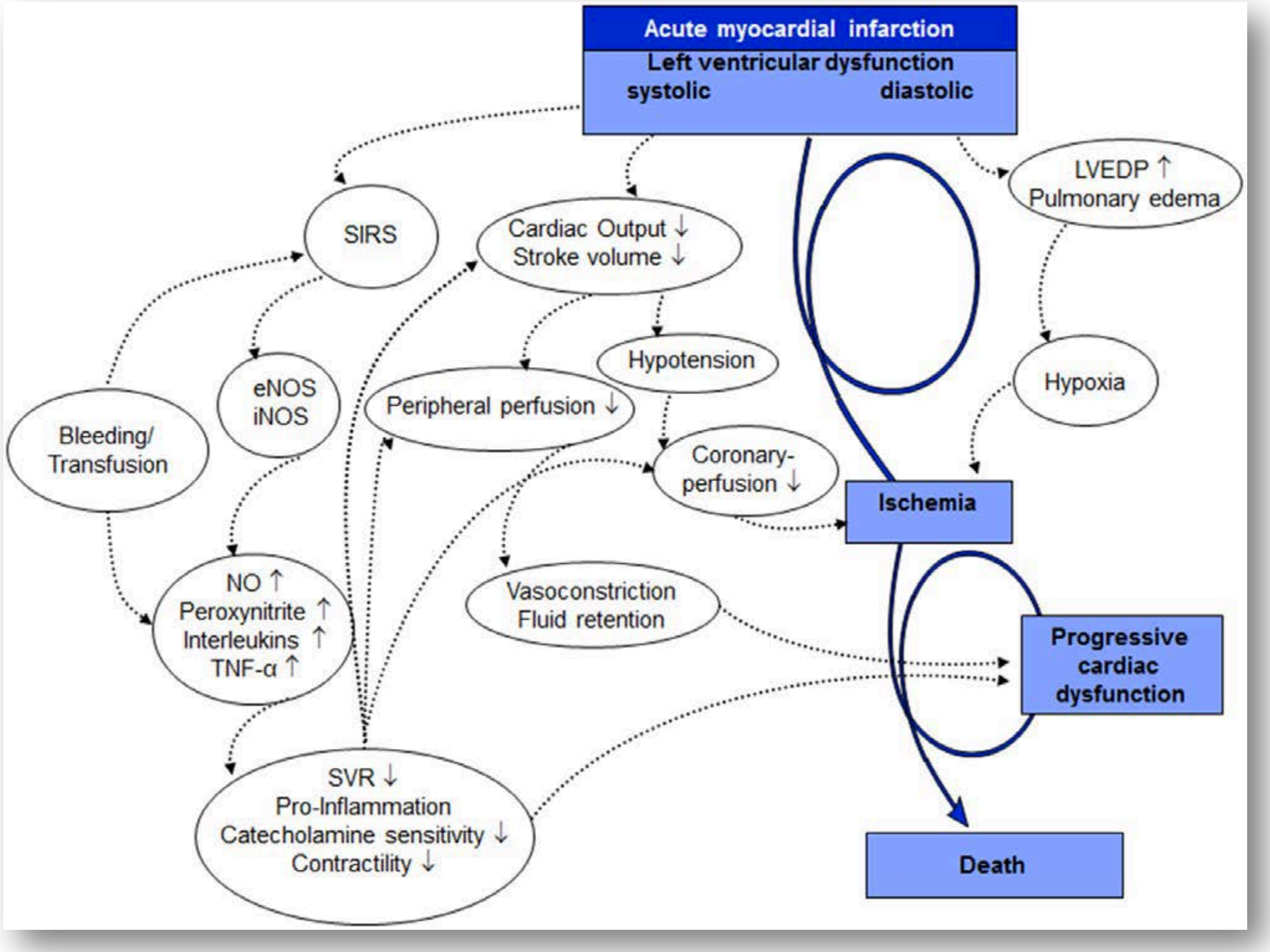
- Acute Mitral Regurgitation 7%
- IVS Rupture 4%
- RV dysfunction 3%
- Cardiac Tamponade 1.5%
- Other 6.5%



Harjola VP et al. Eur J Heart Fail. 2015

Cardiogenic Shock Spiral

Pathophysiological Concept



van Diepen et al, AHA Statement. Circulation 2017

Evidenze su inotropi-vasopressori

Inotropic agents – dobutamine, dopamine, levosimendan, phosphodiesterase III (PDE III) inhibitors			
Short-term, i.v. infusion of inotropic agents may be considered in patients with hypotension (SBP <90 mmHg) and/or signs/ symptoms of hypoperfusion despite adequate filling status, to increase cardiac output, increase blood pressure, improve peripheral perfusion and maintain end-organ function.	IIb	C	
An intravenous infusion of levosimendan or a PDE III inhibitor may be considered to reverse the effect of beta-blockade if beta-blockade is thought to be contributing to hypotension with subsequent hypoperfusion.	IIb	C	
Inotropic agents are not recommended unless the patient is symptomatically hypotensive or hypoperfused because of safety concern.	III	A	556, 557
Vasopressors			
A vasopressor (norepinephrine preferably) may be considered in patients who have cardiogenic shock, despite treatment with another inotrope, to increase blood pressure and vital organ perfusion.	IIb	B	558
It is recommended to monitor ECG and blood pressure when using inotropic agents and vasopressors, as they can cause arrhythmia, myocardial ischaemia, and in the case of levosimendan and PDE III inhibitors also hypotension.	I	C	540, 559–563
In such cases intra-arterial blood pressure measurement may be considered.	IIb	C	

2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

Inotropi e Vasopressori

- Migliorano velocemente i parametri emodinamici
- Beneficio immediato (aumento PA)migliorando la perfusione miocardica
- Sono raccomandati dalle linee guida in corso di shock

- Aumentano il consumo miocardico di O2
- Possono causare ischemia miocardica
- Possono causare aritmie
- Possono aumentare l'area di necrosi
- Danno diretto da overload Ca intracellulare

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*

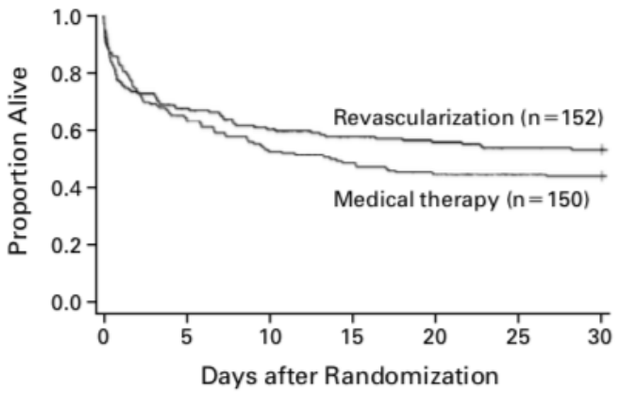


Figure 1. Overall 30-Day Survival in the Study.

The 30-day survival rate was 53.3 percent for patients assigned to revascularization and 44.0 percent for those assigned to medical therapy.

This study was a randomized trial evaluating early revascularization therapy to reduce the high mortality rate associated with cardiogenic shock complicating acute myocardial infarction. The primary end point, overall mortality at 30 days, was not significantly reduced by early revascularization. However, a benefit in terms of mortality was apparent six months after infarction.

Thirty Year Trends (1975-2005) in the Magnitude, Management, and Hospital Death Rates Associated With Cardiogenic Shock in Patients with Acute Myocardial Infarction: A Population-Based Perspective

Robert J. Goldberg, Ph.D.[1], **Frederick A. Spencer, M.D.**[2], **Joel M. Gore, M.D.**[1], **Darleen Lessard, M.S.**[1], and **Jorge Yarzebski, M.D., M.P.H.**[1]

[1]Department of Medicine, Division of Cardiovascular Medicine, University of Massachusetts Medical School, Worcester, MA 01655

[2]Department of Medicine, McMaster University, Hamilton, Ontario Canada

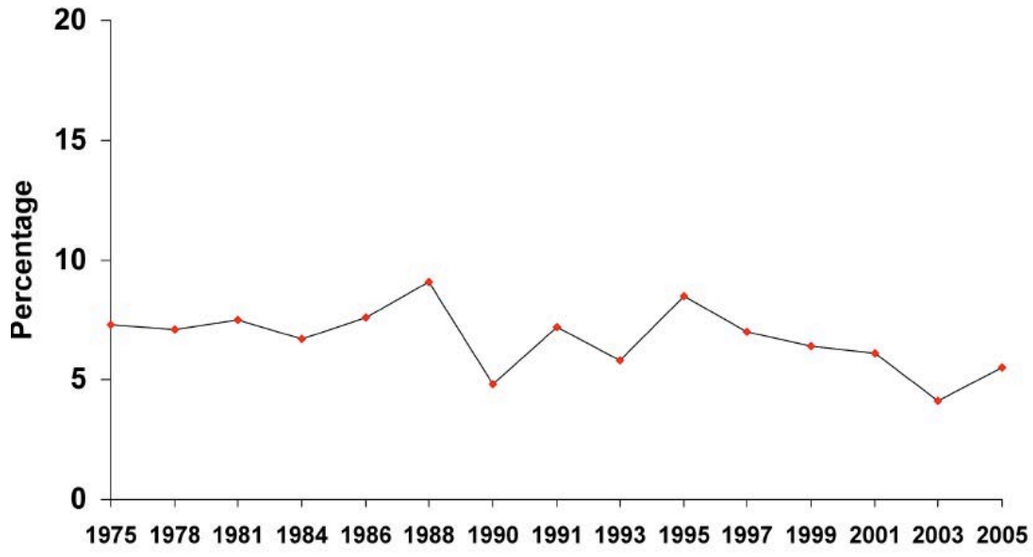


Figure 1.
Trends in the Incidence Rates of Cardiogenic Shock in Patients With Acute Myocardial Infarction

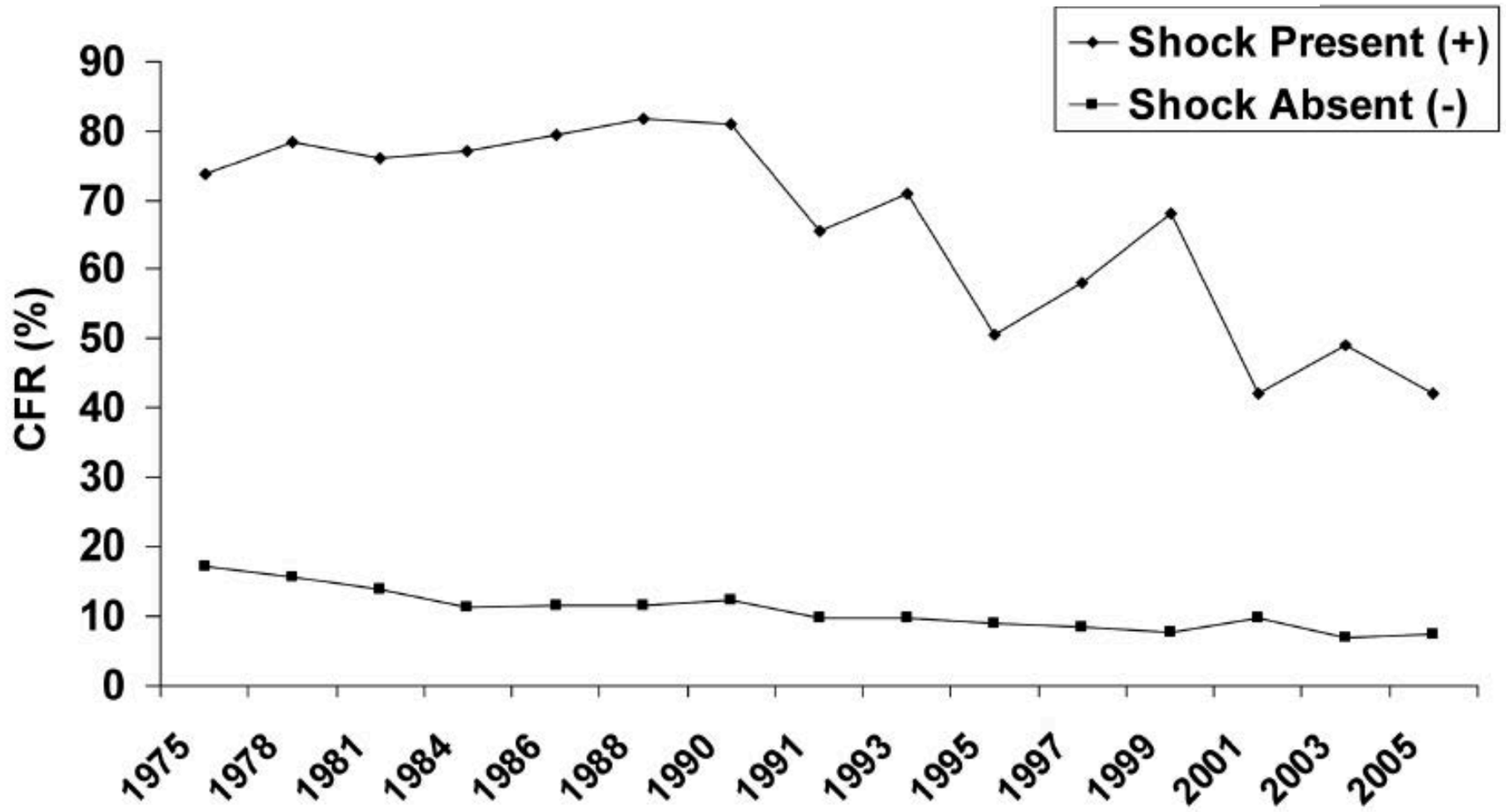
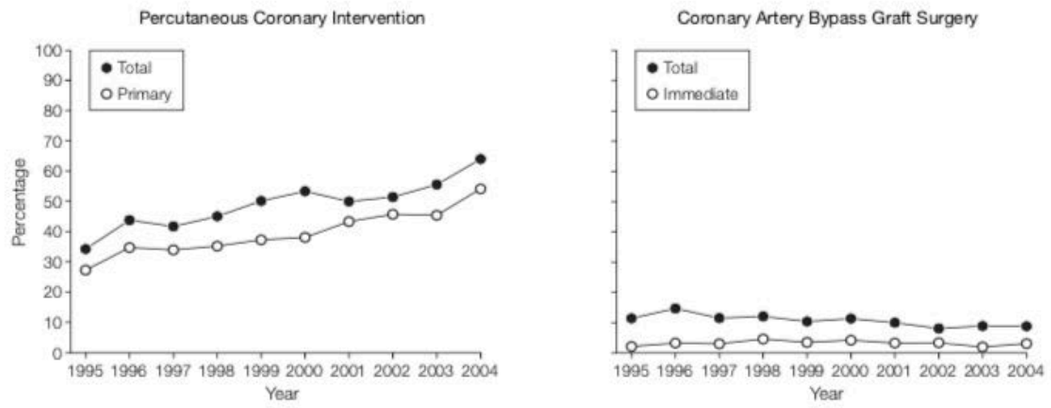


Figure 2.
Trends in Hospital Case-Fatality Rates (CFR's) in Patients With Acute Myocardial Infarction According to the Presence of Cardiogenic Shock

Circulation. 2009 March 10; 119(9): 1211–1219. doi:10.1161/CIRCULATIONAHA.108.814947.

Trends in Management and Outcomes of Patients With Acute Myocardial Infarction Complicated by Cardiogenic Shock

Figure 2. Revascularization Rates in Patients With Cardiogenic Shock at Presentation (n = 7356)



Data are through May 2004. *P* values indicate trends over time: total percutaneous coronary intervention (PCI), $P<.001$; primary PCI, $P<.001$; total coronary artery bypass graft (CABG) surgery, $P<.001$; and immediate CABG surgery, $P = .88$.

Table 2. In-Hospital Mortality Rates*

	No. (%) of Patients in NRMI Registry Presenting With Cardiogenic Shock Who Died		
	Total	<75 y	≥75 y
1995	434 (60.3)	274 (55.8)	160 (69.9)
1996	510 (59.8)	290 (51.4)	220 (76.1)
1997	530 (60.7)	313 (53.3)	217 (75.9)
1998	413 (58.0)	225 (49.2)	188 (73.7)
1999	554 (55.9)	324 (50.3)	230 (66.3)
2000	475 (56.6)	258 (47.9)	217 (72.1)
2001	416 (52.1)	222 (43.9)	194 (66.4)
2002	339 (49.8)	187 (40.8)	152 (68.5)
2003	282 (51.3)	162 (44.7)	120 (63.8)
2004†	163 (47.9)	88 (39.5)	75 (64.1)
P value	<.001	<.001	<.001

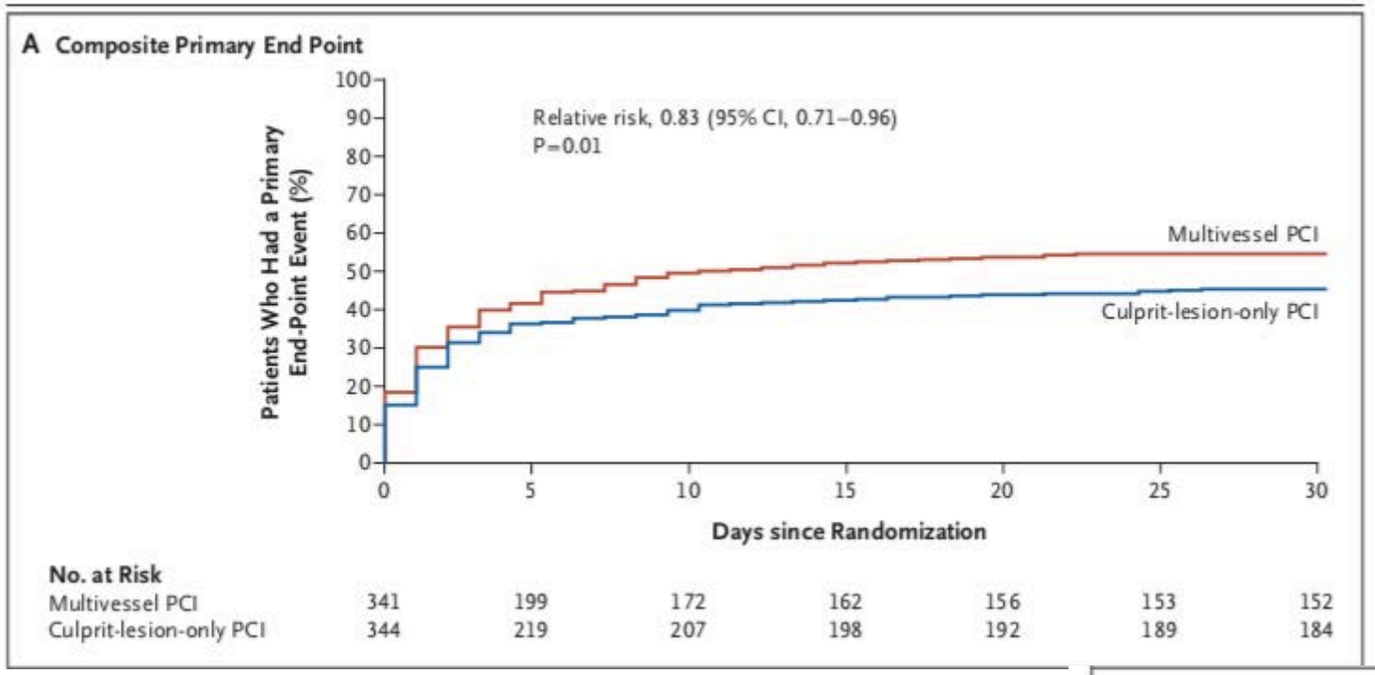
Abbreviation: NRMI, National Registry of Myocardial Infarction.
*The Mantel-Haenszel χ^2 probability for the 2-sided alternative hypothesis that a linear association exists is presented.
†Through May.

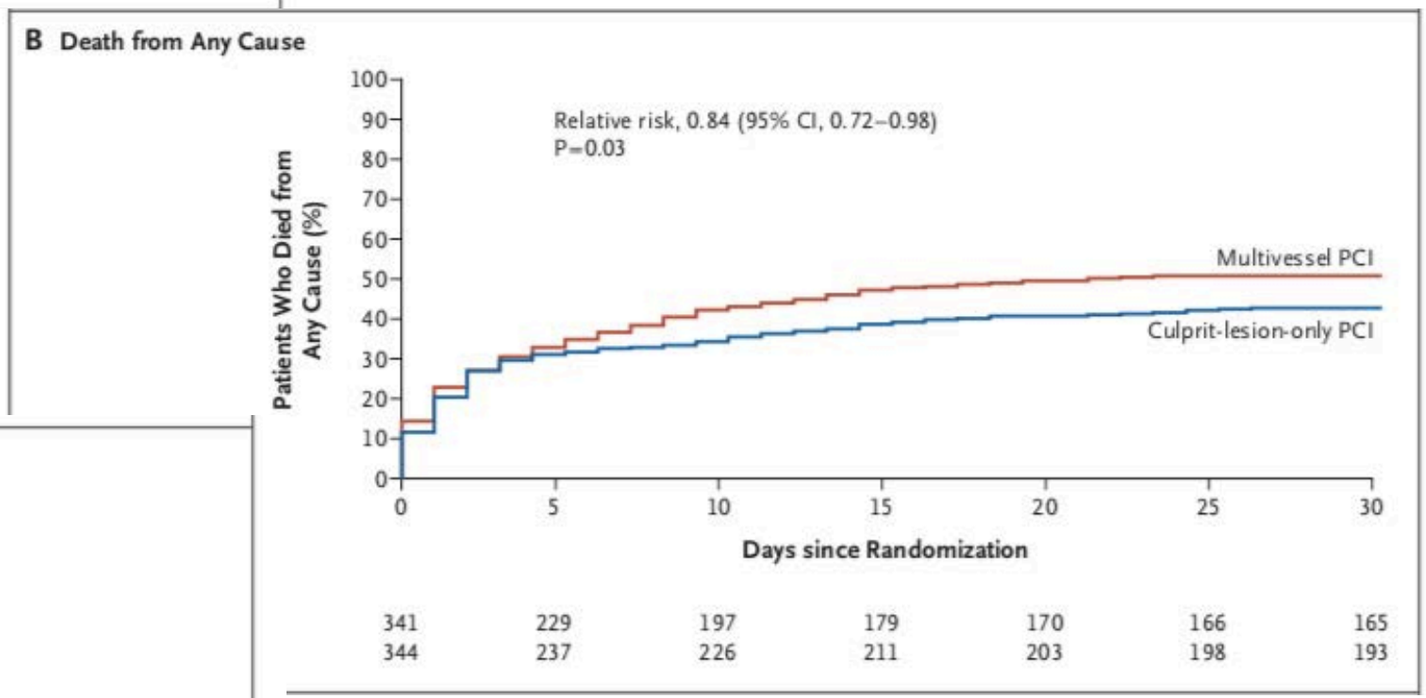
The NEW ENGLAND JOURNAL of MEDICINE

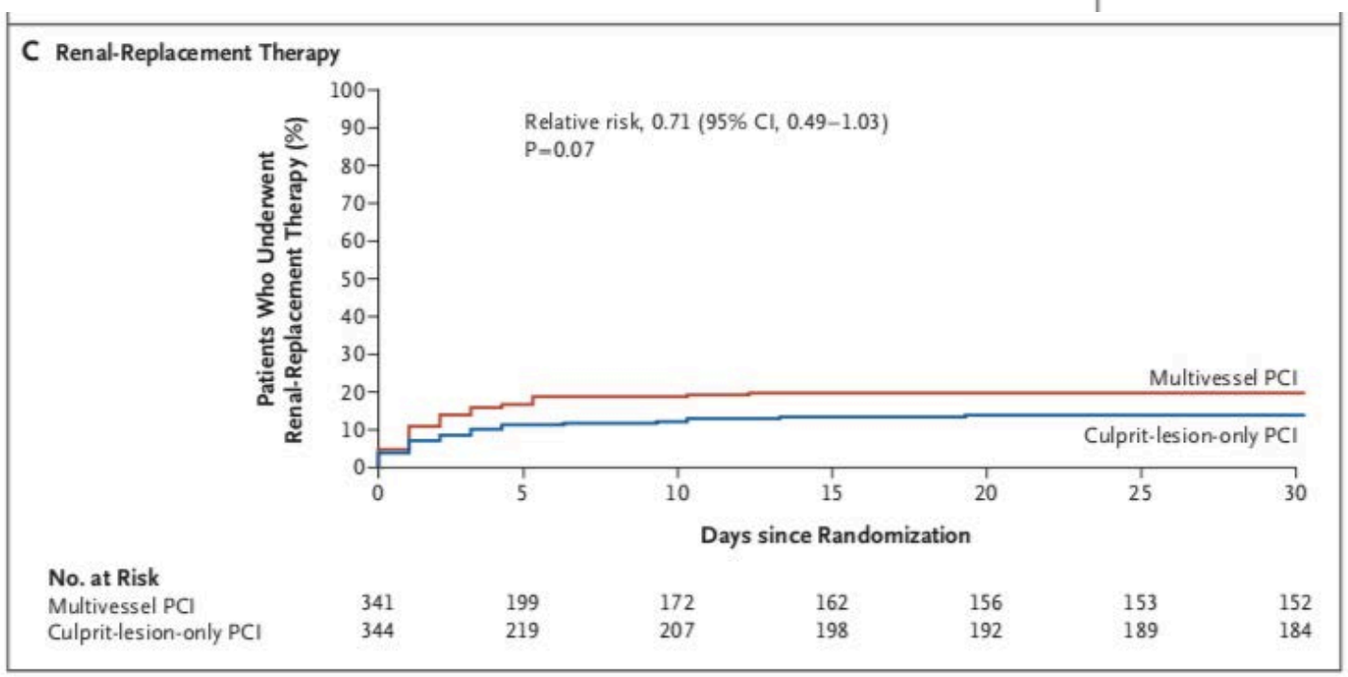
ESTABLISHED IN 1812 DECEMBER 21, 2017 VOL. 377 NO. 25

PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock

H. Thiele, I. Akin, M. Sandri, G. Fuernau, S. de Waha, R. Meyer-Saraei, P. Nordbeck, T. Geisler, U. Landmesser, C. Skurk, A. Fach, H. Lapp, J.J. Piek, M. Noc, T. Goslar, S.B. Felix, L.S. Maier, J. Stepinska, K. Oldroyd, P. Serpytis, G. Montalescot, O. Barthelemy, K. Huber, S. Windecker, S. Savonitto, P. Torremante, C. Vrints, S. Schneider, S. Desch, and U. Zeymer, for the CULPRIT-SHOCK Investigators*







	IABP	Impella 2.5	Impella CP	Impella 5.0	TandemHeart	ECMO
Meccanismo	Pompa pneumatica	Pompa assiale	Pompa assiale	Pompa assiale	Pompa centrifuga	Pompa centrifuga
Dimensioni della cannula	7-9 Fr	12 Fr	14 Fr	21 Fr	21 Fr afflusso 15-17 Fr efflusso	18-21 Fr afflusso 15-22 Fr efflusso
Tecnica di inserimento	Percutanea a. femorale fino all'aorta discendente	Percutanea a. femorale, posizionato a cavallo della valvola aorta	Percutanea a. femorale, posizionato a cavallo della valvola aorta	Isolamento chirurgico dell'a. femorale, posizionato a cavallo della valvola aortica	Percutanea: cannula di afflusso da vena femorale fino ad atrio sinistro (puntura transettale); cannula di efflusso dall'a. femorale	Percutanea: cannula di afflusso da vena femorale fino ad atrio destro; cannula di efflusso dall'a. femorale fino all'aorta discendente
Portata cardiaca	0.3-0.5 l/min	2.5 l/min	3.7 l/min	5 l/min	4-5 l/min	>4.5 l/min
N. max di giorni di impianto	Settimane	5-7	5-7	5-7	14	Settimane
Complessità di inserimento	Bassa	Media	Media	Alta	Alta	Media
Rischio di emolisi	Basso	Medio	Medio	Medio	Medio	Medio
Rischio di complicanze						
Ischemiche	Basso	Medio	Medio	Alto	Alto	Alto
Emorragiche	Basso	Medio	Medio	Medio	Alto	Alto
Infettive	Basso	Basso	Basso	Medio	Medio	Medio

Battistoni I. et al G Ital Cardiol 2017;18(10):708-718

The NEW ENGLAND JOURNAL of MEDICINE

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., Hans-Georg Olbrich, M.D., Jörg Hausleiter, M.D., Gert Richardt, M.D., Marcus Hennersdorf, M.D., Klaus Empen, M.D., Georg Fuernau, M.D., Steffen Desch, M.D., Ingo Eitel, M.D., Rainer Hambrecht, M.D., Jörg Fuhrmann, M.D., Michael Böhm, M.D., Henning Ebelt, M.D., Steffen Schneider, Ph.D., Gerhard Schuler, M.D., and Karl Werdan, M.D., for the IABP-SHOCK II Trial Investigators*

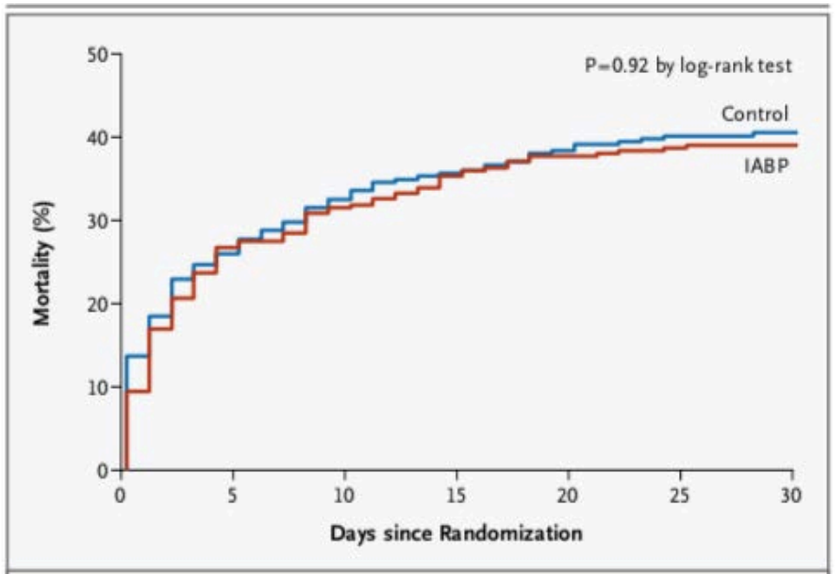


Figure 1. Time-to-Event Curves for the Primary End Point.
Time-to-event curves are shown through 30 days after randomization for the primary end point of all-cause mortality. Event rates represent Kaplan–Meier estimates.

Inotropic/vasopressor agents may be considered for haemodynamic stabilization.	IIb	C
Short-term mechanical support[c] may be considered in patients in refractory shock.	IIb	C
Routine intra-aortic balloon pumping is not indicated.[177,437]	III	B

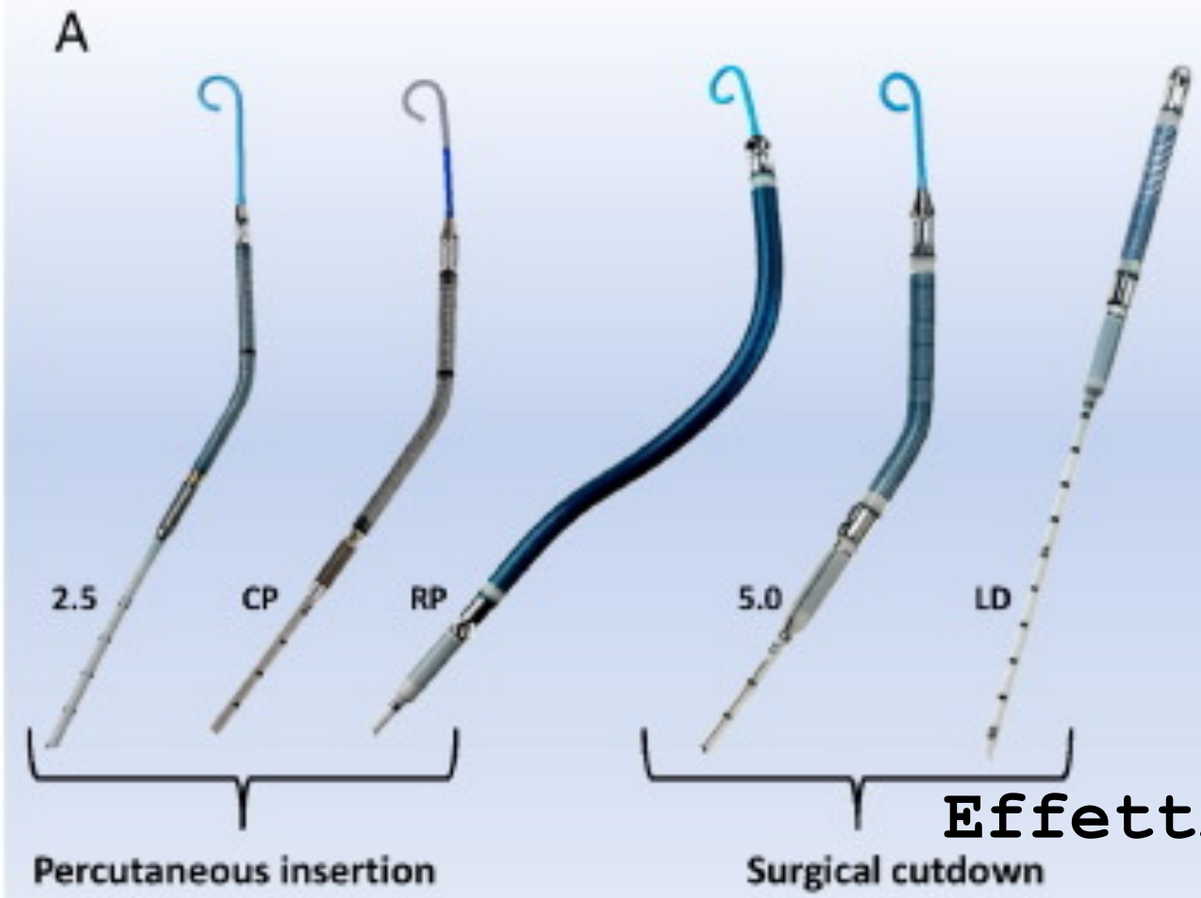
CABG = coronary artery bypass graft surgery; ECLS = extracorporeal life sup-

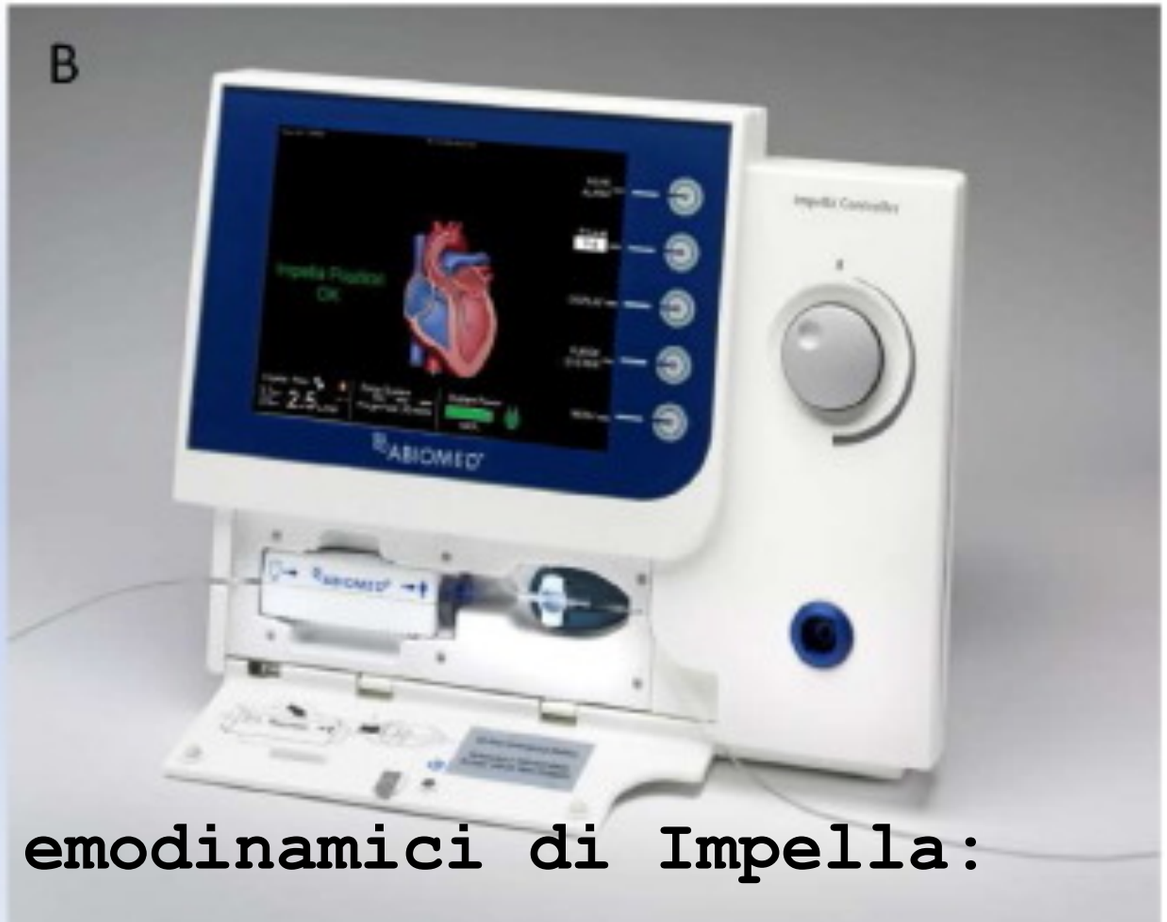
2014 ESC/EACTS Guidelines on myocardial revascularization

haemodynamic instability.			
IABP insertion should be considered in patients with haemodynamic instability/cardiogenic shock due to mechanical complications.	IIa	C	
Patients with mechanical complication after acute myocardial infarction require immediate discussion by the Heart Team.	I	C	

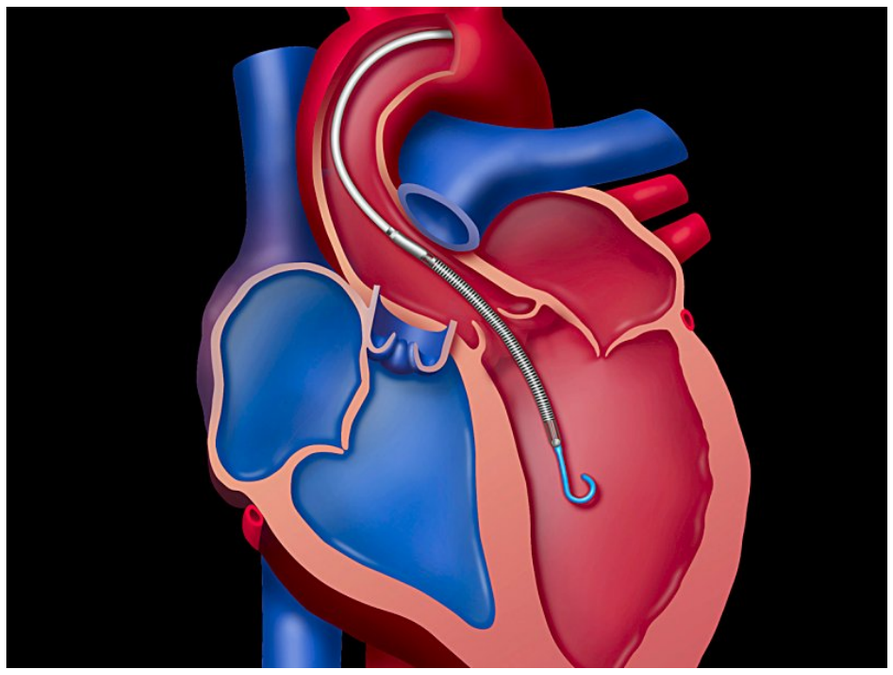
2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

IABP is not routinely recommended in cardiogenic shock.	III	B
Short-term mechanical circulatory support may be considered in refractory cardiogenic shock depending on patient age, comorbidities and neurological function.	IIb	C





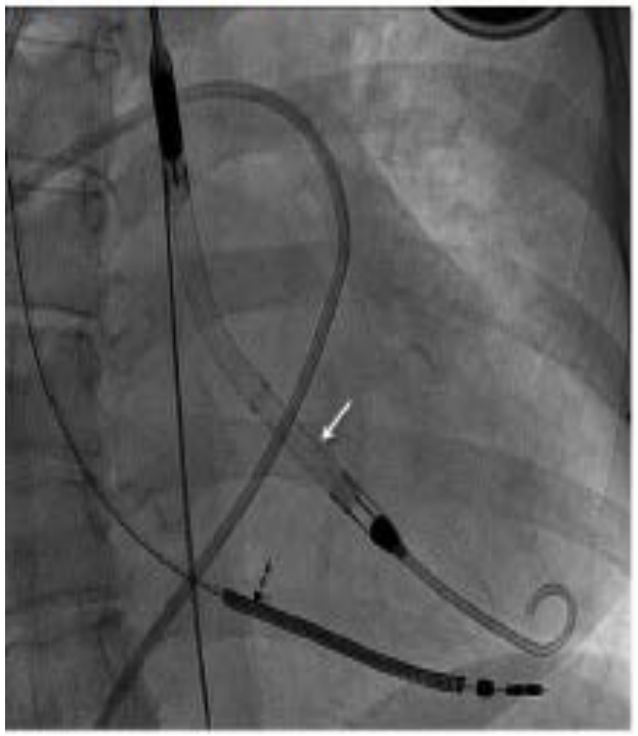
Effetti emodinamici di Impella:



- riduce il consumo miocardico di O2
- Aumenta il flusso coronarico
- Migliora la pressione arteriosa media
- Riduce la pressione telediastolica e quindi la congestione polmonare
- Riduce il postcarico del ventricolo dx

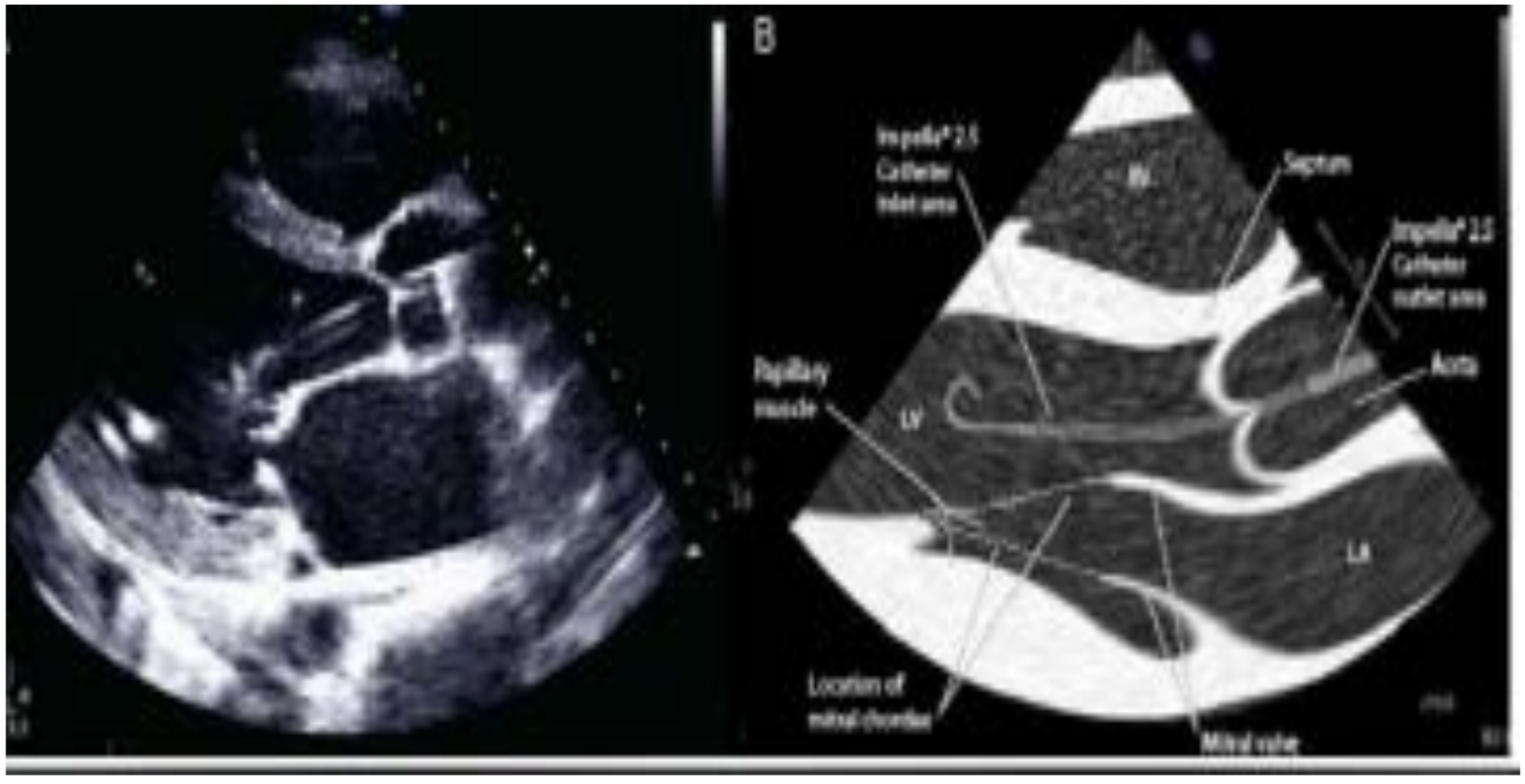
Attenzione alla disfunzione

Fluoroscopia

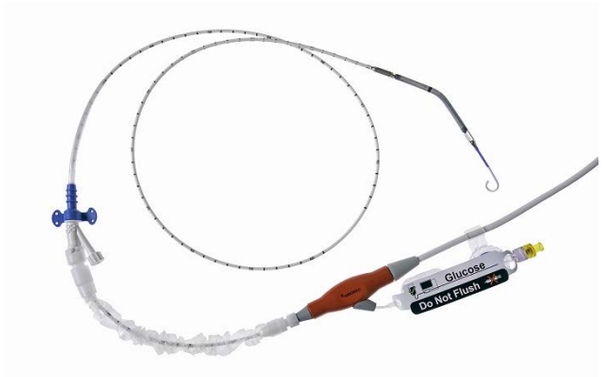


Valutazione ecocardiografica

TTE e TEE: la proiezione parasternale asse lungo è la consigliata



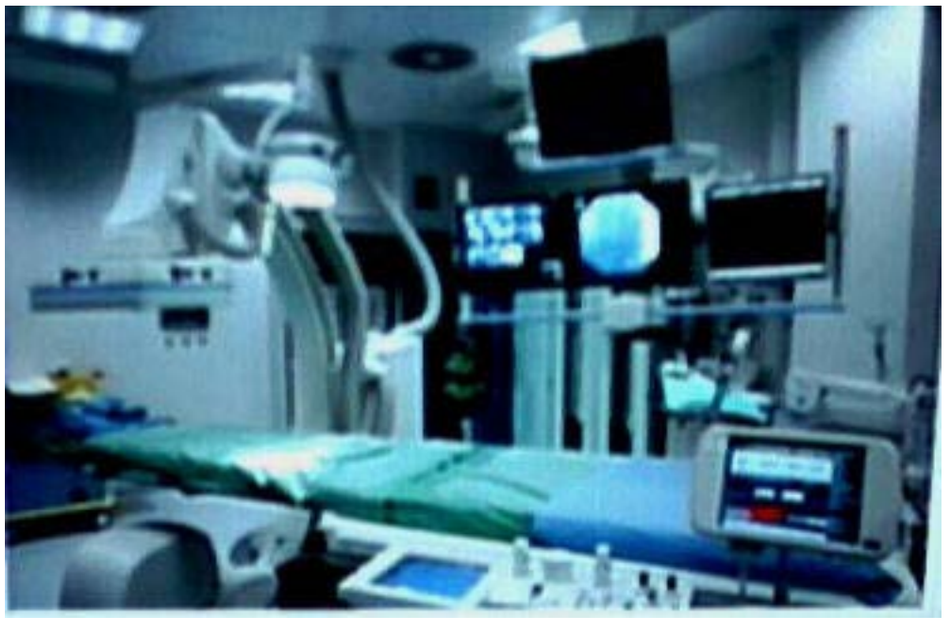
L'area di INFLOW deve trovarsi circa 3.5 cm sotto la valvola aortica



Impella: indicazioni

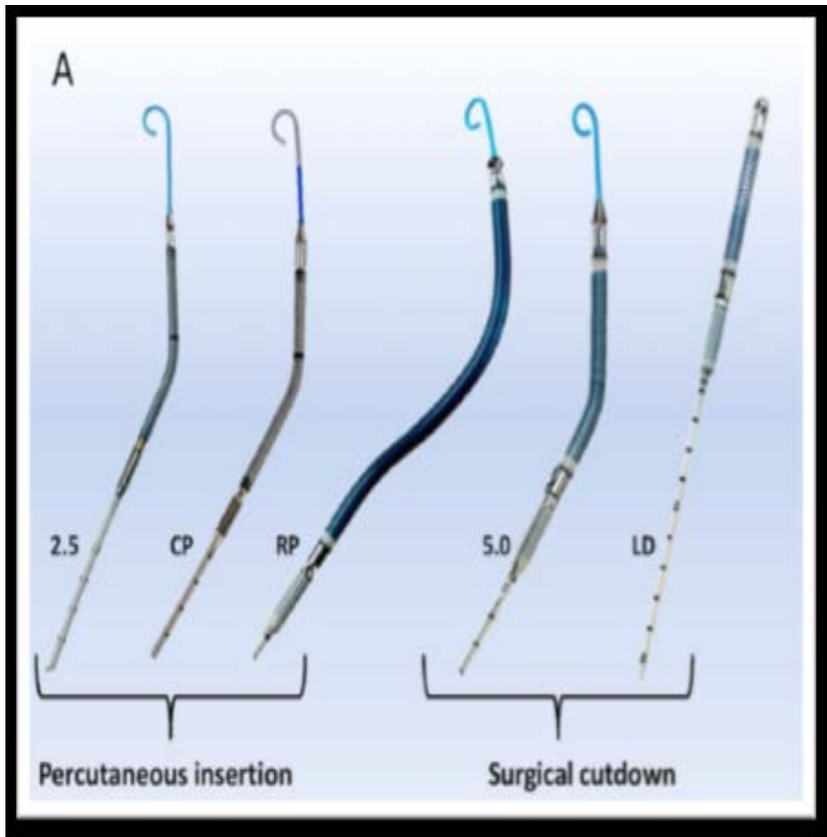
Shock cardiogeno

PTCA ad alto rischio



Impella- Specifiche Tecniche

	Impella 2.5	Impella CP	Impella 5.0/LD	Impella RP
Flusso (L/min)	**2.5**	**3.7 – 4.0**	**5.0**	**4.4**
VAD	Sinistro			Destro
Supporto circolatorio (EU Approval CE Mark)	Parziale (5gg)	Parziale/Alto (5gg)	Alto (10gg)	Parziale/Alto (14gg)
Dimensione catetere	9 F	9 F	9 F	11 F
Dimensione pompa	12 F	14 F	21 F	22 F
Dimensione introduttore	13 F	14 F	--	23 F
Metodo di impianto	Percutaneo	Percutaneo	Chirurgico	Percutaneo (venoso)

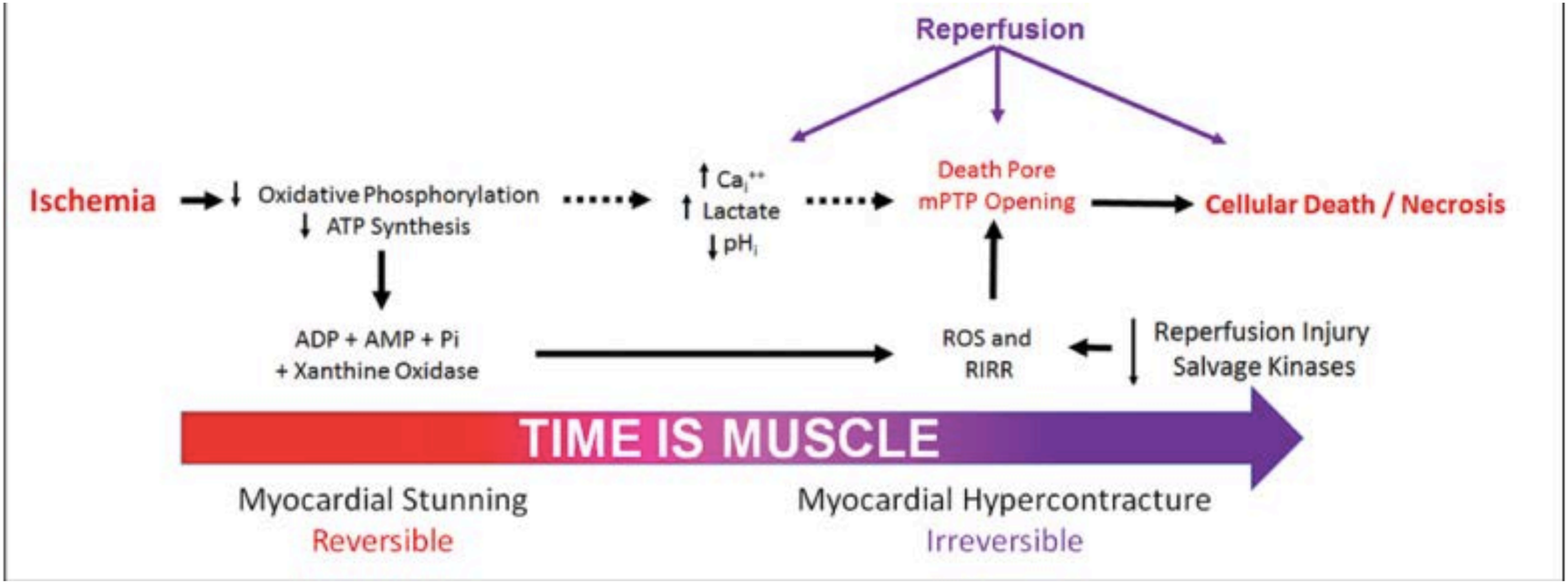


considerare le controindicazioni…

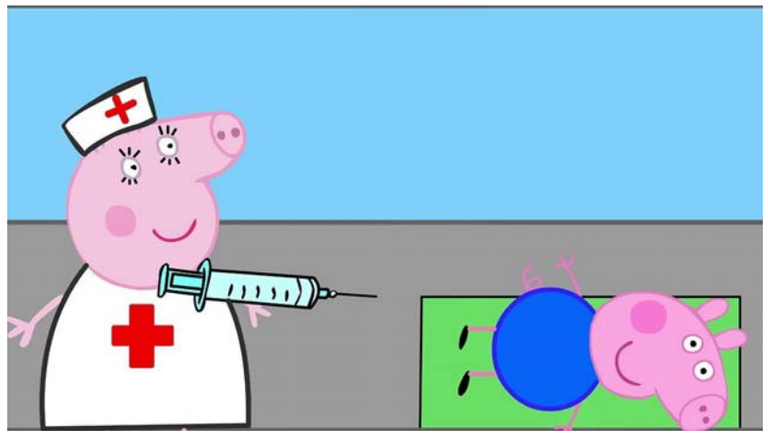
- **Insufficienza aortica severa (rischio di peggioramento)**
- **Stenosi valvolare aortica severa (relativa) rischio di embolizzazione sistemica e perforazione della valvola**
- **Protesi valvolare aortica meccanica**
- **Patologie dell'aorta**
- **Trombosi ventricolare sinistra (rischio di aspirazione)**
- **Vasculopatia periferica grave (nb: angiografia pre-impianto, considerare approccio ascellare)**

… *e le possibili complicanze*

- **Ischemia arto inferiore**
- **Emolisi (riducibile con corretta posizione del device)**
- **Allarme di suzione (valutare volemia e funzione ventricolare destra)**
- **Sanguinamento (nb: posizionamento di dispositivo di chiusura intravascolare)**


Reperfusion
Ischemia
Oxidative Phosphorylation
ATP Synthesis
↑ Ca$_i^{++}$
↑ Lactate
↓ pH$_i$
Death Pore
mPTP Opening
Cellular Death / Necrosis
ADP + AMP + Pi
+ Xanthine Oxidase
ROS and
RIRR
Reperfusion Injury
Salvage Kinases
TIME IS MUSCLE
Myocardial Stunning
Reversible
Myocardial Hypercontracture
Irreversible

Left Ventricular Unloading Before Reperfusion Promotes Functional Recovery After Acute Myocardial Infarction



Michele L. Esposito, MD,* Yali Zhang, MD, PhD,* Xiaoying Qiao, PhD,* Lara Reyelt, BS, Vikram Paruchuri, MD, Gavin R. Schnitzler, PhD, Kevin J. Morine, MD, Shiva K. Annamalai, MD, Courtney Bogins, BS, Peter S. Natov, BS, Robert Pedicini, BS, Catalina Breton, BS, Andrew Mullin, BS, Emily E. Mackey, MD, Ayan Patel, MD, Ethan Rowin, MD, Iris Z. Jaffe, MD, PhD, Richard H. Karas, MD, PhD, Navin K. Kapur, MD

ABSTRACT

BACKGROUND Heart failure after an acute myocardial infarction (AMI) is a major cause of morbidity and mortality worldwide. We recently reported that activation of a transvalvular axial-flow pump in the left ventricle and delaying myocardial reperfusion, known as primary unloading, limits infarct size after AMI. The mechanisms underlying the cardioprotective benefit of primary unloading and whether the acute decrease in infarct size results in a durable reduction in LV scar and improves cardiac function remain unknown.

OBJECTIVES This study tested the importance of LV unloading before reperfusion, explored cardioprotective mechanisms, and determined the late-term impact of primary unloading on myocardial function.

METHODS Adult male swine were subjected to primary reperfusion or primary unloading after 90 min of percutaneous left anterior descending artery occlusion.

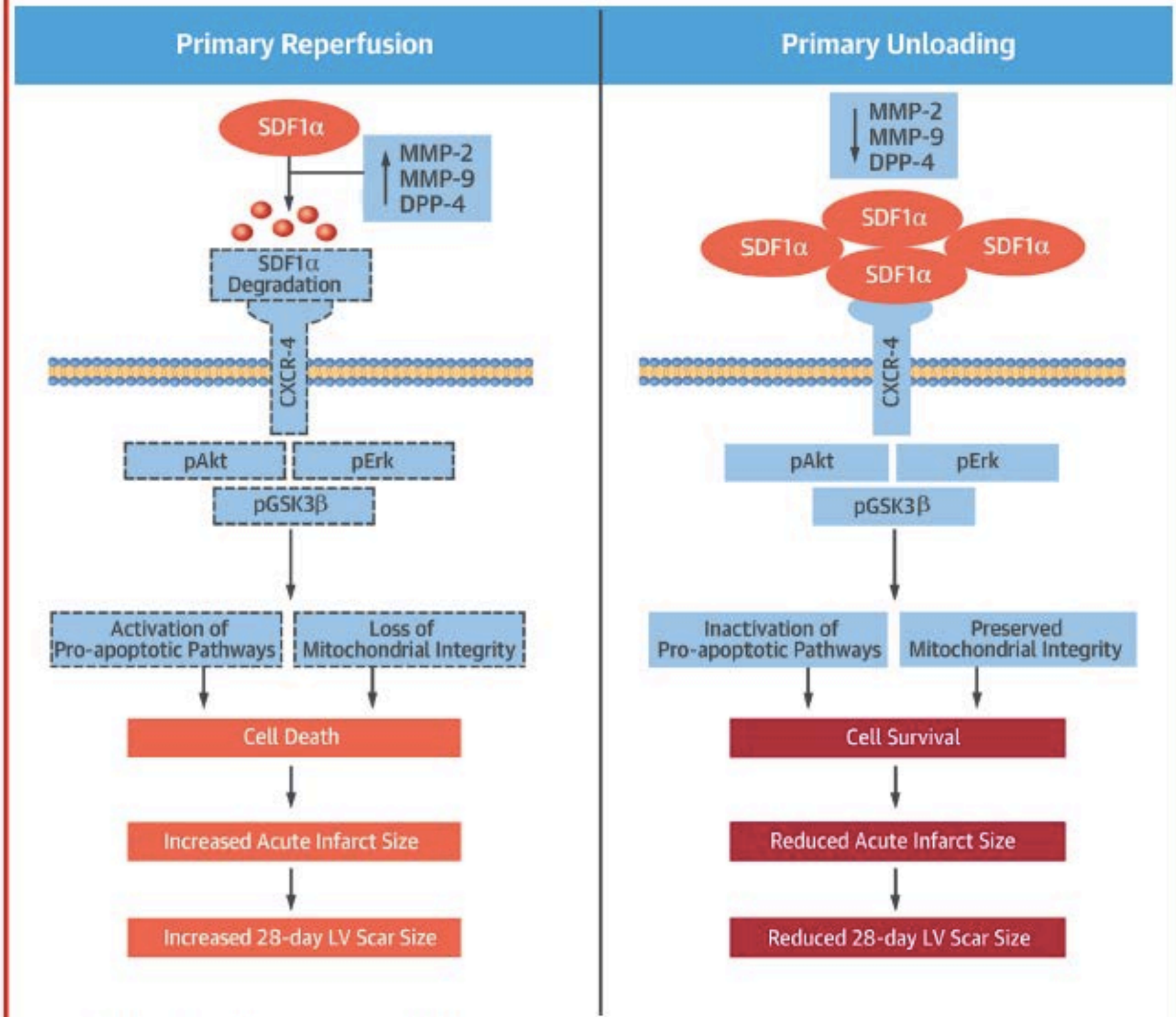
RESULTS Compared with primary reperfusion, 30 min of LV unloading was necessary and sufficient before reperfusion to limit infarct size 28 days after AMI. Compared with primary reperfusion, primary unloading increased expression of genes associated with cellular respiration and mitochondrial integrity within the infarct zone. Primary unloading for 30 min further reduced activity levels of proteases known to degrade the cardioprotective cytokine, stromal-derived factor (SDF)-1α, thereby increasing SDF-1α signaling via reperfusion injury salvage kinases, which limits apoptosis within the infarct zone. Inhibiting SDF-1α activity attenuated the cardioprotective effect of primary unloading. Twenty-eight days after AMI, primary unloading reduced LV scar size, improved cardiac function, and limited expression of biomarkers associated with heart failure and maladaptive remodeling.

CONCLUSIONS The authors report for the first time that first mechanically reducing LV work before coronary reperfusion with a transvalvular pump is necessary and sufficient to reduce infarct size and to activate a cardioprotective program that includes enhanced SDF-1α activity. Primary unloading further improved LV scar size and cardiac function 28 days after AMI. (J Am Coll Cardiol 2018;72:501–14) © 2018 Published by Elsevier on behalf of the American College of Cardiology Foundation.

Left Ventricular Unloading Before Reperfusion Promotes Functional Recovery After Acute Myocardial Infarction

Michele L. Esposito, MD,* Yali Zhang, MD, PhD,* Xiaoying Qiao, PhD,* Lara Reyelt, BS, Vikram Paruchuri, MD, Gavin R. Schnitzler, PhD, Kevin J. Morine, MD, Shiva K. Annamalai, MD, Courtney Bogins, BS, Peter S. Natov, BS, Robert Pedicini, BS, Catalina Breton, BS, Andrew Mullin, BS, Emily E. Mackey, MD, Ayan Patel, MD, Ethan Rowin, MD, Iris Z. Jaffe, MD, PhD, Richard H. Karas, MD, PhD, Navin K. Kapur, MD

CENTRAL ILLUSTRATION Mechanistic Insight Into the Biological Impact of Primary Unloading



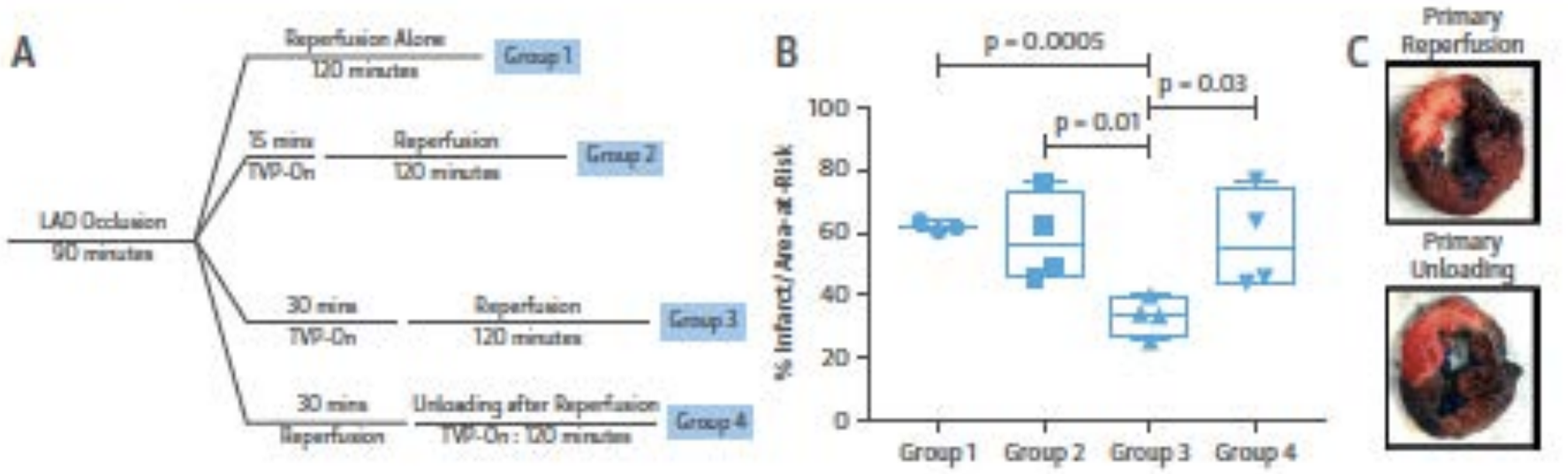
Esposito, M.L. et al. J Am Coll Cardiol. 2018;72(5):501-14.

Mechanically unloading the left ventricle for a minimum of 30 min before reperfusion limits expression of proteolytic enzymes that degrade stromal-derived factor-1α (SDF1α), thereby increasing cardioprotective signaling improving cell survival, and reducing both acute infarct size and subsequent myocardial scar size 28 days after acute myocardial infarction. DPP-4 = dipeptidyl peptidase-4; LV = left ventricular; MMP = matrix metalloproteinase.

Left Ventricular Unloading Before Reperfusion Promotes Functional Recovery After Acute Myocardial Infarction

Michele L. Esposito, MD,* Yali Zhang, MD, PhD,* Xiaoying Qiao, PhD,* Lara Reyelt, BS, Vikram Paruchuri, MD, Gavin R. Schnitzler, PhD, Kevin J. Morine, MD, Shiva K. Annamalai, MD, Courtney Bogins, BS, Peter S. Natov, BS, Robert Pedicini, BS, Catalina Breton, BS, Andrew Mullin, BS, Emily E. Mackey, MD, Ayan Patel, MD, Ethan Rowin, MD, Iris Z. Jaffe, MD, PhD, Richard H. Karas, MD, PhD, Navin K. Kapur, MD

FIGURE 1 Timing of Unloading Between Groups and Infarct Quantification



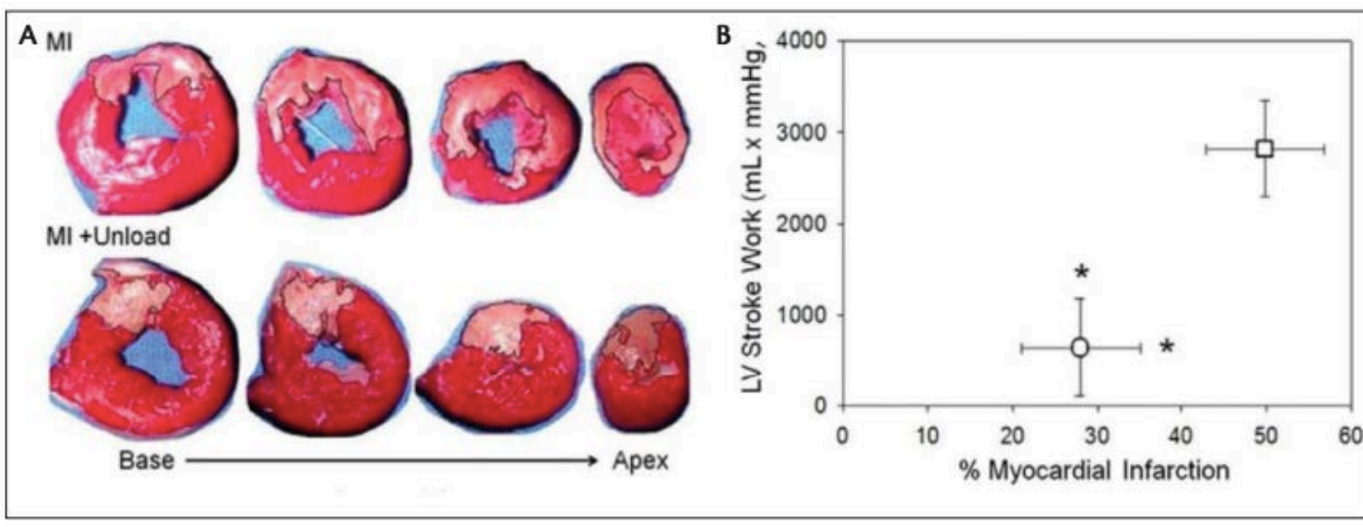
(A) Flowchart illustrating the effect of reperfusion alone (group 1), left ventricular unloading for 15 min (group 2) or 30 min (group 3) before reperfusion, or left ventricular unloading after reperfusion (group 4). (B) Infarct area as a percentage of the area at risk for each group (1-way analysis of variance = 0.017 across all 4 groups). (C) Representative images of infarct quantification using triphenyltetrazolium chloride and Evan's blue counterstaining from group 1 and group 3 (n = 4 per group). **Red** staining indicates the area at risk, **white** staining indicates infarcted myocardium, and **blue** staining indicates myocardial tissue outside the area at risk. LAD = left anterior descending; TVP = transvalvular pump.

TABLE 1 Hemodynamic Variables 28 Days After Acute Myocardial Infarction

	Primary Reperfusion	Primary Unloading	p Value
Heart rate, beats/min	63 ± 9	73 ± 12	NS
LV EDV, ml	190 ± 13	248 ± 54	NS
LV ESV, ml	150 ± 15	195 ± 47	NS
LV stroke volume, ml	40 ± 6	54 ± 7	0.02
LV cardiac output, l/min	2.5 ± 0.2	3.9 ± 0.6	0.006
LV stroke work, ml × mm Hg	2,195 ± 307	3,075 ± 339	0.008
LV systolic pressure, mm Hg	79 ± 3	78 ± 10	NS
LV end-diastolic pressure, mm Hg	11.3 ± 2.5	7.4 ± 1.5	0.02

Values are mean ± SD.

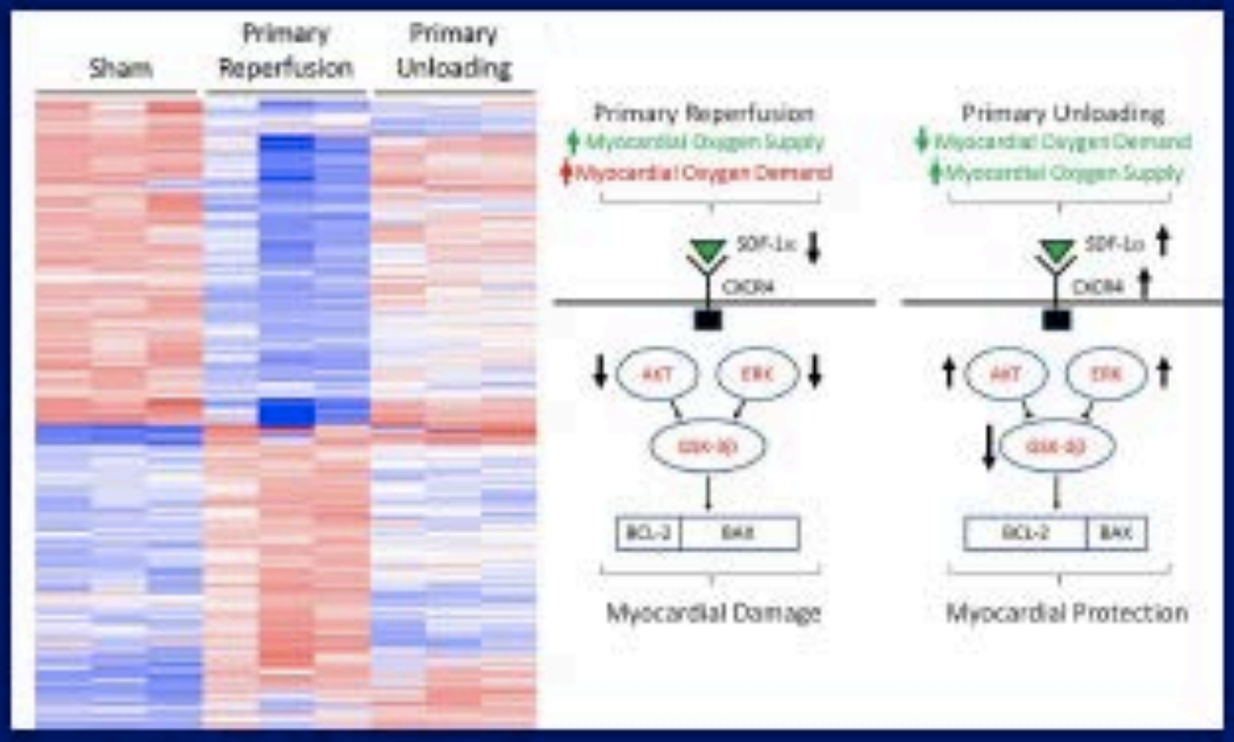
EDV = end-diastolic volume; ESV = end-systolic volume; LV = left ventricular; NS = not significant.

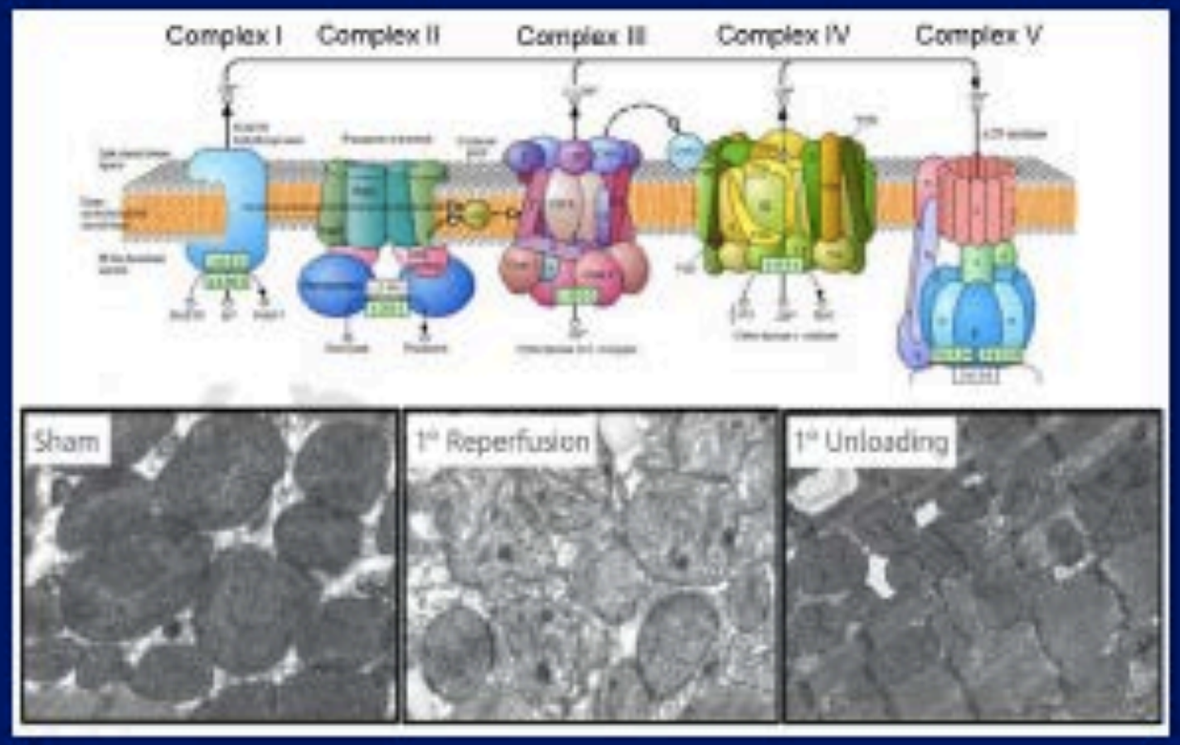


Preclinical Development of the DTU Concept

2012 — 2018

- **TandemHeart** — <u>Delayed</u> Reperfusion (Circulation 2013)
- **Impella CP** — <u>Delayed</u> Reperfusion (JACC HF 2015)
- **Impella CP** — Cardioprotective Biology (JACC 2018)
- **Impella CP** — Preclinical Pilot (JACC 2018)
- **Impella CP** — DTU-STEMI Pilot Trial (Circulation 2018)





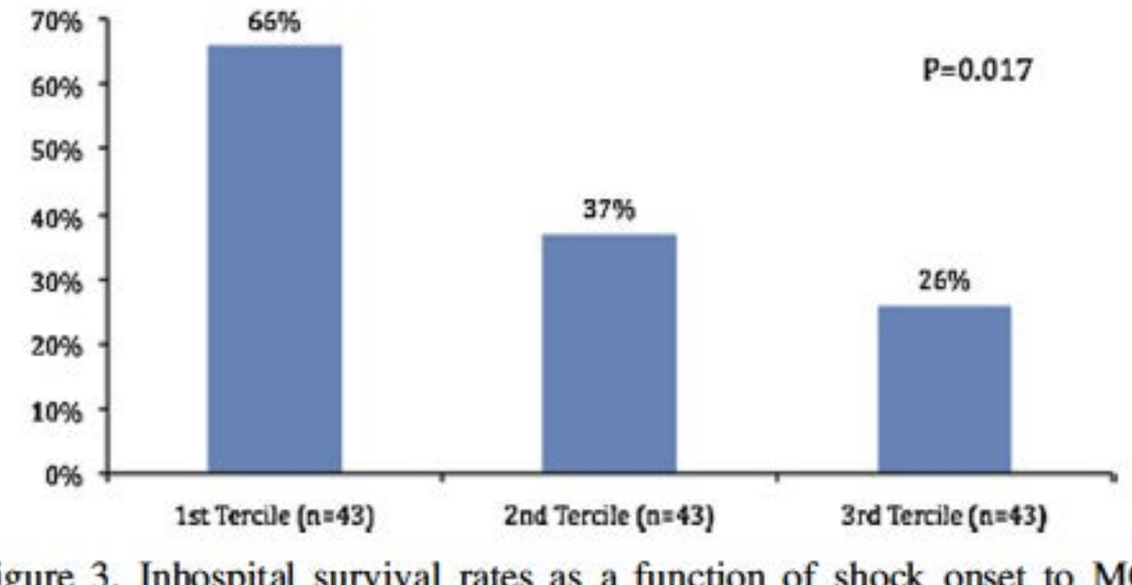
Trans-valvular LV Unloading Limits Myocardial Ischemia and Promotes a Cardioprotective Shift in Myocardial Biology

Esposito, Zhang, Qiao and Kapur et al JACC 2018

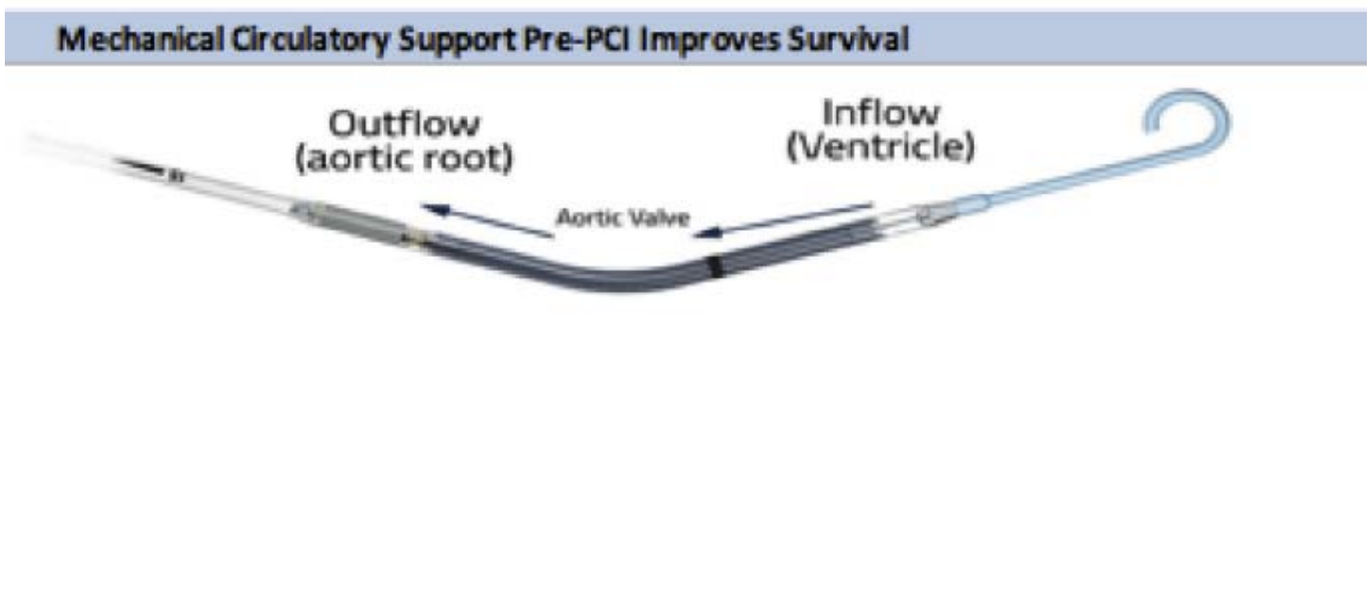
Effect of Early Initiation of Mechanical Circulatory Support on Survival in Cardiogenic Shock

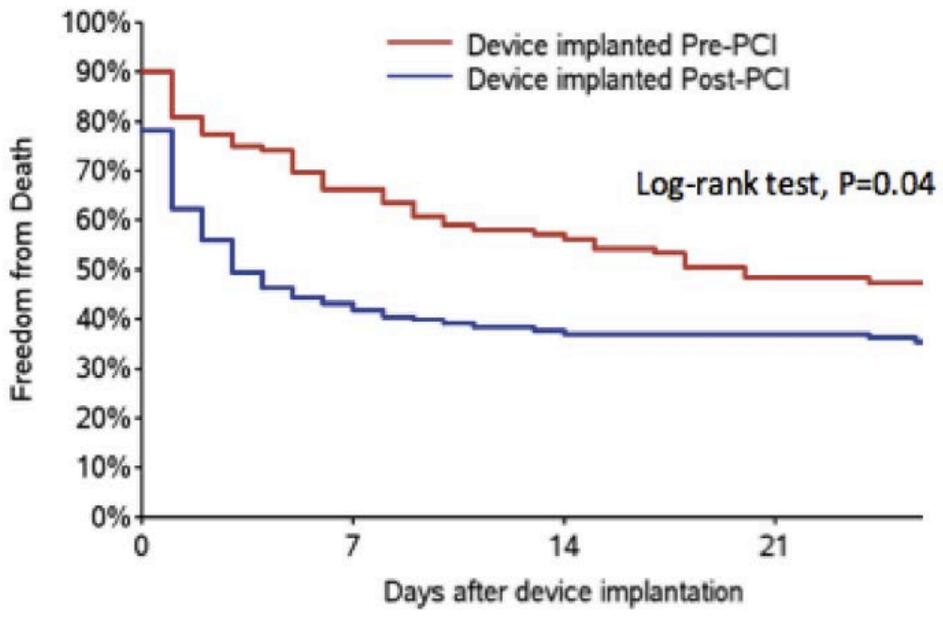
Mir B. Basir, DO[a], Theodore L. Schreiber, MD[b], Cindy L. Grines, MD[b], Simon R. Dixon, MD[c], Jeffrey W. Moses, MD[d], Brijeshwar S. Maini, MD[e], Akshay K. Khandelwal, MD[a], E. Magnus Ohman, MD[f], and William W. O'Neill, MD[a,*]

The role and timing of percutaneous mechanical circulatory support (MCS) devices in the treatment of acute myocardial infarction complicated by cardiogenic shock (AMICS) are not well understood. We sought to evaluate patient characteristics and predictors of outcomes in patients presenting with AMICS supported with an axial flow percutaneous MCS device; 287 consecutive unselected patients enrolled in the catheter-based ventricular assist device registry presenting with AMICS who underwent percutaneous coronary intervention (PCI) were included in this analysis. All patients were supported with either the Impella 2.5 or Impella CP. Mean patient age was 66 ± 12.5 years, 76% were men, and mean left ventricular ejection fraction was 25 ± 12%. Before receiving MCS, 80% of patients required inotropes or vasopressors and 40% were supported with intra-aortic balloon pump; 9% of patients were under active cardiopulmonary resuscitation at the time of MCS implantation. Survival to discharge was 44%. In a multivariate analysis, early implantation of a MCS device before PCI ($p = 0.04$) and before requiring inotropes and vasopressors ($p = 0.05$) was associated with increased survival. Survival was 66% when MCS was initiated <1.25 hours from shock onset, 37% when initiated within 1.25 to 4.25 hours, and 26% when initiated after 4.25 hours ($p = 0.017$). Survival was 68%, 46%, 35%, 35%, and 26% for patients requiring 0, 1, 2, 3, and ≥4 inotropes before MCS support, respectively ($p < 0.001$). In conclusion, MCS implantation early after shock onset, before initiation of inotropes or vasopressors and before PCI, is independently associated with improved survival in patients presenting with AMICS. (Am J Cardiol 2017;119:845–851)



igure 3. Inhospital survival rates as a function of shock onset to MCS nplantation.





Effect of Early Initiation of Mechanical Circulatory Support on Survival in Cardiogenic Shock

Mir B. Basir, DO[a], Theodore L. Schreiber, MD[b], Cindy L. Grines, MD[b], Simon R. Dixon, MD[c], Jeffrey W. Moses, MD[d], Brijeshwar S. Maini, MD[e], Akshay K. Khandelwal, MD[a], E. Magnus Ohman, MD[f], and William W. O'Neill, MD[a,*]

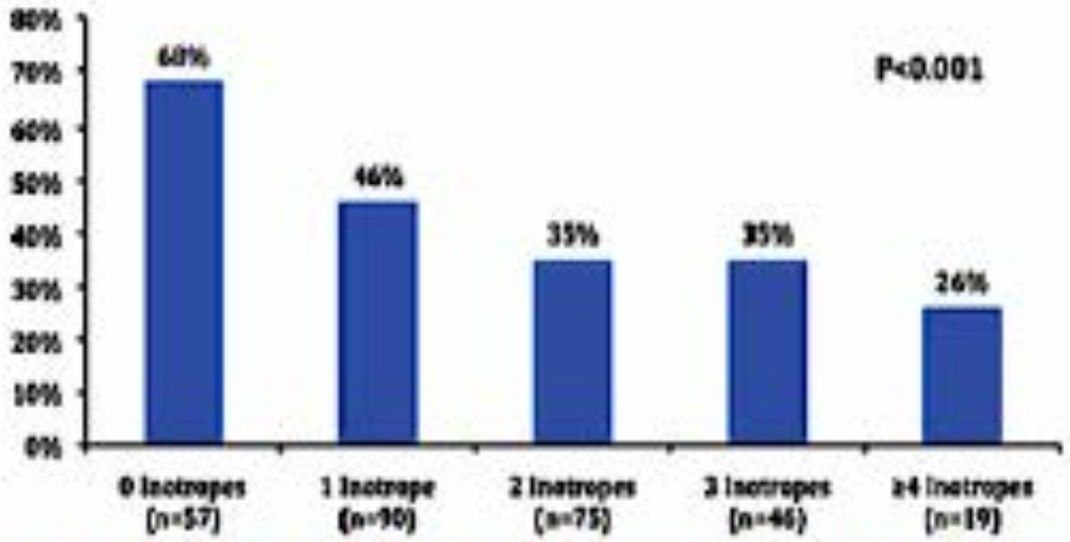


Figure 2. Inhospital survival rates as a function of inotropic support to MCS implantation.

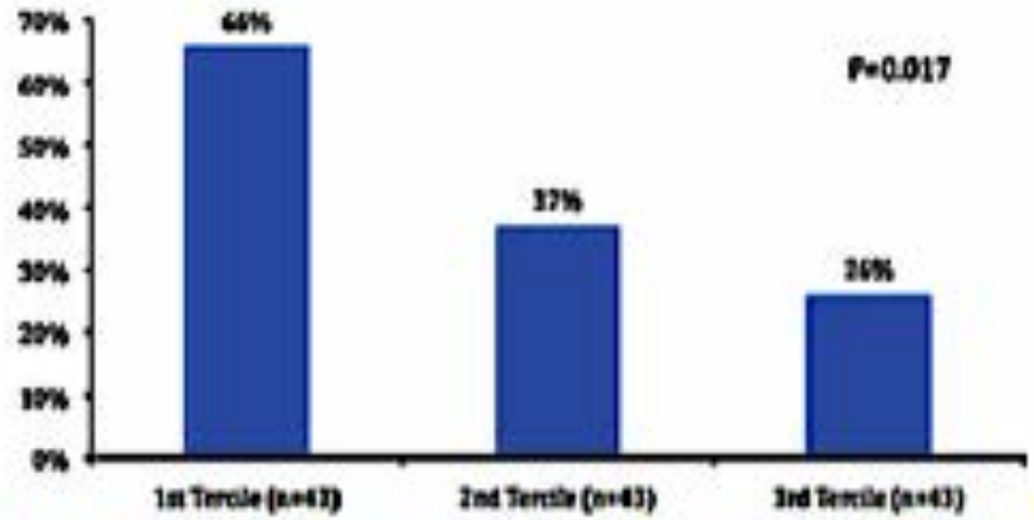
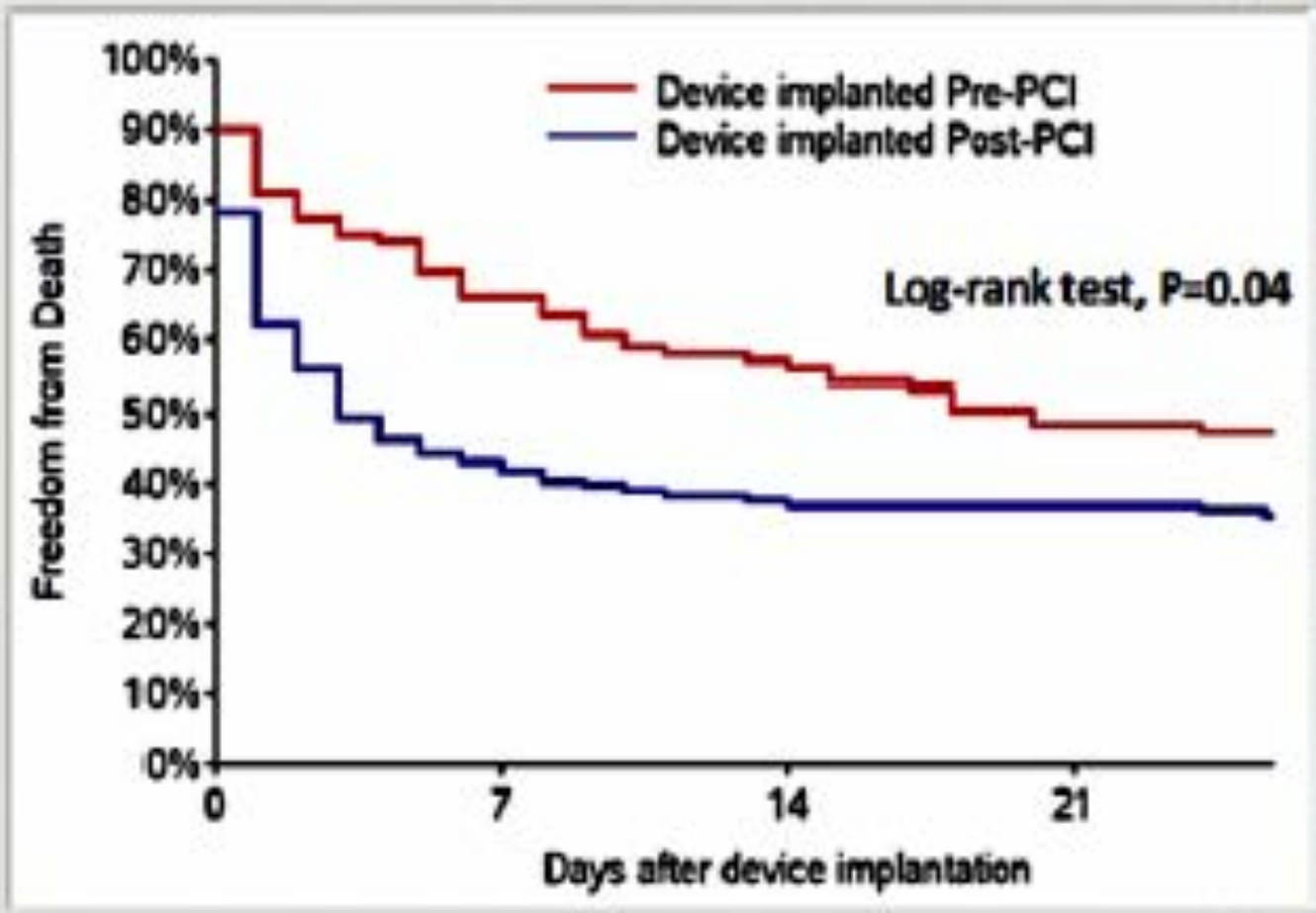


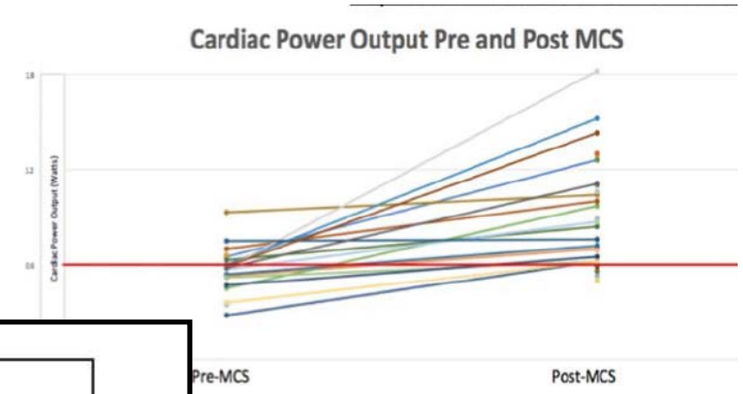
Figure 3. Inhospital survival rates as a function of shock onset to MCS implantation.

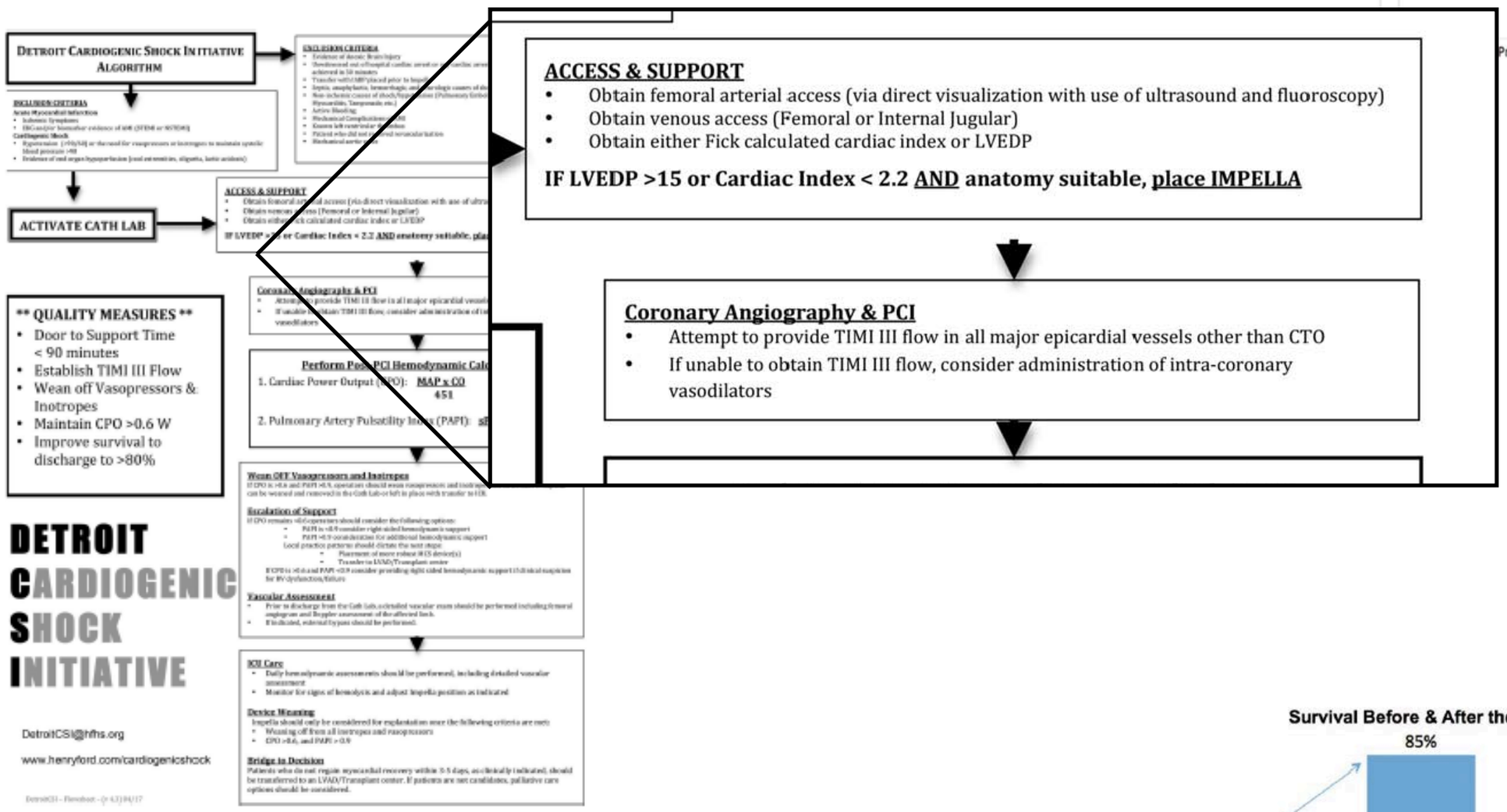


"door to support concept"

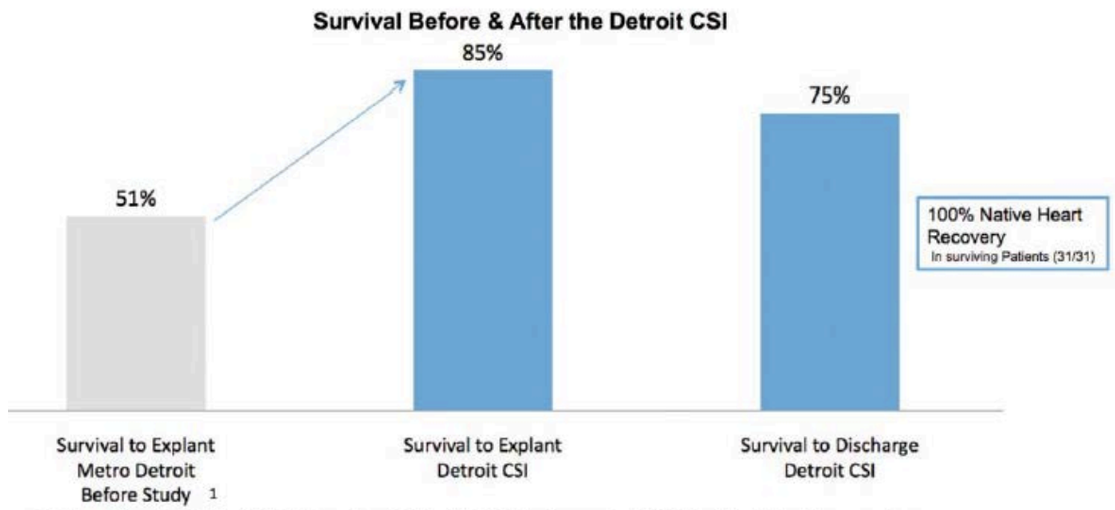
Am J Cardiol 2017;119:845e851)

Feasibility of early mechanical circulatory support in acute myocardial infarction complicated by cardiogenic shock: The Detroit cardiogenic shock initiative





Conclusion: Centers who adopted a regional shock protocol emphasizing the delivery of early MCS with invasive hemodynamic monitoring can achieve rapid door to support times and can improve survival in patients who present with AMICS. Larger national studies will be needed to further validate this pilot feasibility study.



1. *Abiomed Impella Quality (IQ) Database , Jan 2015 to July 2016 for Aggregate DTW Metro Hospitals, all-comers who presented with AMICS, Survival to Explant*

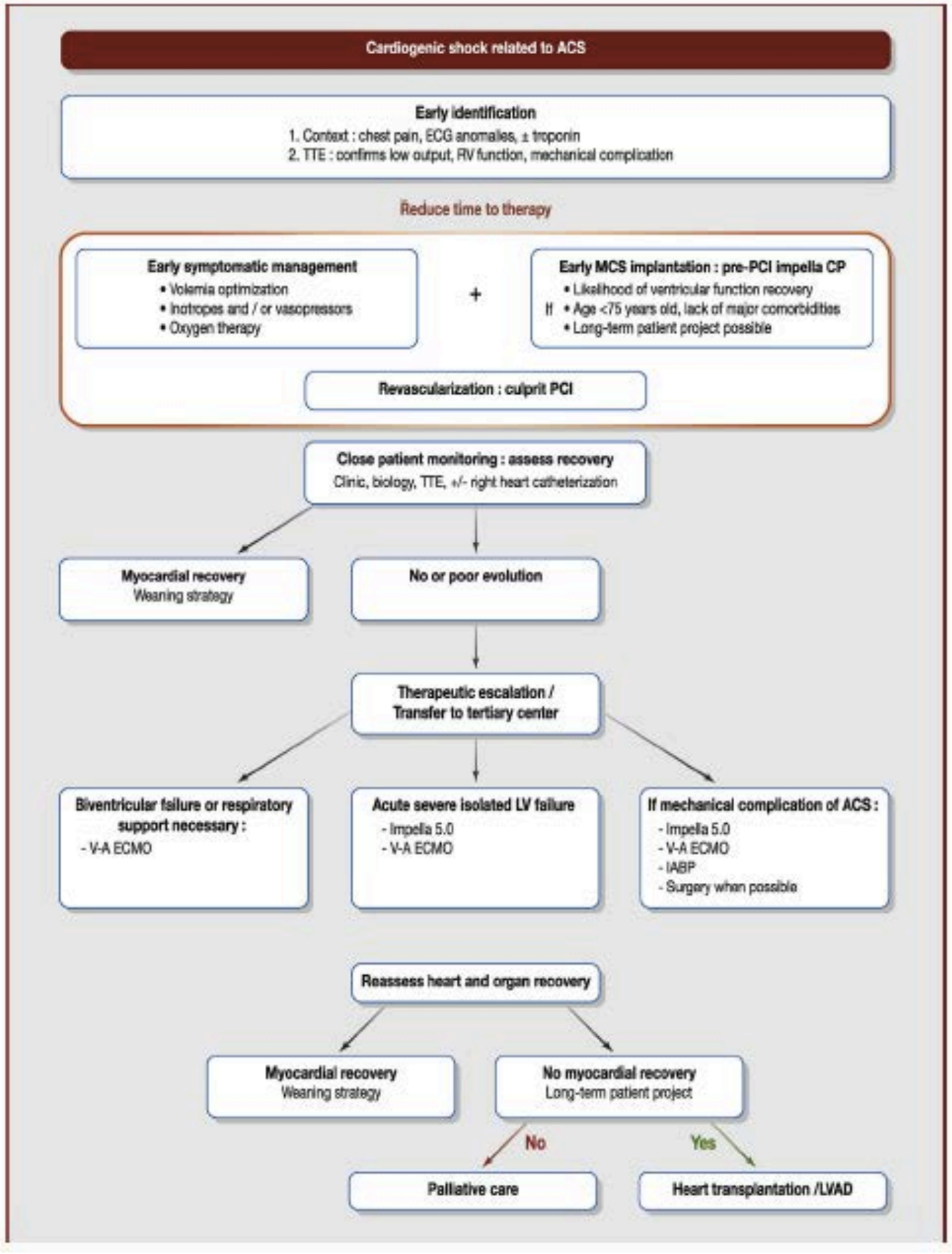
Catheter Cardiovasc Interv. 2017;1–8.

Mechanical circulatory support in patients with cardiogenic shock in intensive care units: A position paper of the ''Unité de Soins Intensifs de Cardiologie'' group of the French Society of Cardiology, endorsed by the ''Groupe Athérome et Cardiologie Interventionnelle'' of the French Society of Cardiology

Laurent Bonello[a,b,*], Clement Delmas[c,d], Guillaume Schurtz[e,f], Guillaume Leurent[g], Eric Bonnefoy[h], Nadia Aissaoui[i], Patrick Henry[j]

CS in the context of ACS

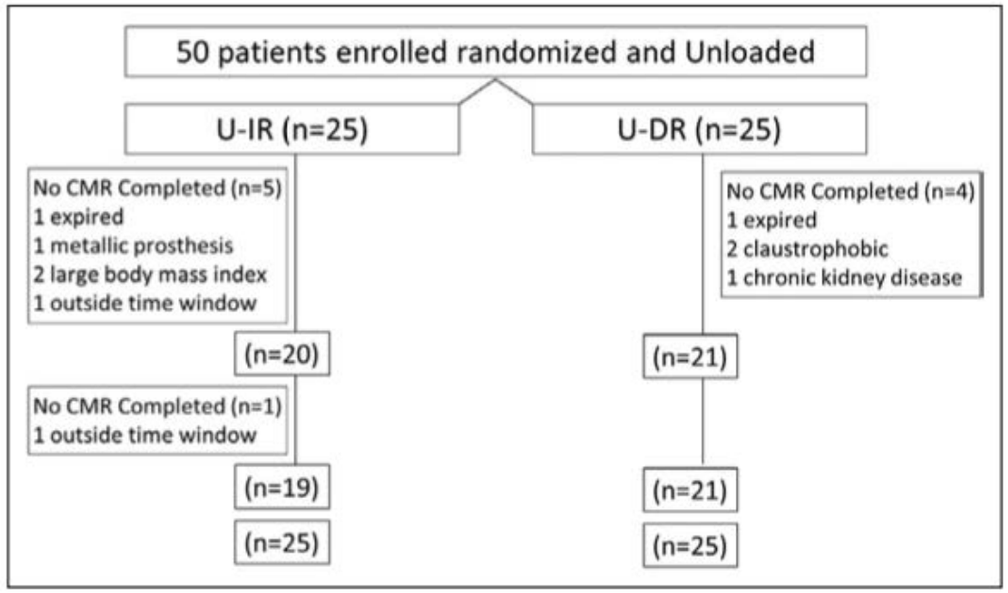
In this situation, MCS should be considered early before PCI in the catheterization laboratory, to provide support and enable a safe revascularization procedure (Fig. 2).



Unloading the Left Ventricle Before Reperfusion in Patients With Anterior ST-Segment–Elevation Myocardial Infarction

A Pilot Study Using the Impella CP

Navin K. Kapur, MD
et al



Events	U-DR n=25	95% CI	U-IR n=25	95% CI	*P* Value
MACCE, n (%)	3 (12)	2.55%–31.22%	2 (8)	0.98%–26.03%	0.99
CV mortality, n (%)	1 (4)	0.10%–20.35%	1 (4)	0.10%–20.35%	0.99
Reinfarction, n (%)	0 (0)	0.00%–13.72%	0 (0)	0.00%–13.72%	—
Stroke/TIA, n (%)	0 (0)	0.00%–13.72%	1 (4)	0.10%–20.35%	0.99
Major vascular events, n (%)	2 (8)	0.98%–26.03%	0 (0)	0.00%–13.72%	0.49

CP indicates cardiac power; CV, cardiovascular; LV, left ventricle; MACCE, major adverse cardiac and cerebrovascular events; TIA, transient ischemic attack; U-DR, delayed reperfusion after 30 minutes of LV unloading by the Impella CP; and U-IR, immediate reperfusion after placement of the Impella CP.

Circulation. 2019;139:337–346. DOI: 10.1161/CIRCULATIONAHA.118.038269

PROTOCOLLO SHOCK CARDIOGENO DA SINDROME CORONARICA ACUTA

CARDIOLOGIE AZIENDALI CON EMODINAMICA H24

Cardiologia dell'ospedale di S. Maria Annunziata
Cardiologia di Prato
Cardiologia di Pistoia
Cardiologia di Empoli.

OSPEDALI INVIANTI ALL'OSPEDALE DI SANTA MARIA ANNUNZIATA

Ospedale Nuovo S. Giovanni di Dio
Ospedale di S. Maria Nuova
Ospedale Serristori (Figline Valdarno)
Nuovo Ospedale del Mugello (Borgo San Lorenzo)

ALGORITMO SHOCK CARDIOGENO DA SINDROME CORONARICA ACUTA

CRITERI DI INCLUSIONE

Sindrome coronarica acuta (sintomi + alterazioni ECG e/o incremento troponina)

+

Shock cardiogeno

- Ipotensione con PAs < 90 mmHg o necessità di vasopressori per mantenere PAs >90 mmHg
- Evidenza di ipoperfusione d'organo:

segni clinici e di lab.(Lattati >3)

EFFETTUARE ECOCARDIOGRAMMA ALL'ARRIVO DEL PAZIENTE
con challenge di fluidi (o sollevamento passivo degli arti)

CRITERI DI ESCLUSIONE

- CAUSE NON ISCHEMICHE DI SHOCK: tamponamento cardiaco, embolia polmonare, pneumotorace, mio
cardiomiopatie, ostruzione dinamica all'efflusso, shock da patologie valvolari)
- Età >75 aa
- ACR con ROSC non ristabilito in 30 minuti
- IR severa in dialisi, BPCO severa, insufficienza epatica moderata-severa, neoplasia con as
 e /o metastasi, demenza, stroke recente e/o con esiti rilevanti
- Quadro di MOF
- Sepsi grave e/o shock settico
- Coma post-anossico o evidenza di lesioni cerebrali post-anossiche
- Sanguinamento attivo
- Recente intervento di chirurgia maggiore
- Complicanze meccaniche di IMA
- Trombo ventricolare sx
- Valvola protesica meccanica aortica e stenosi aortica severa e/o severamente calcifica
- Arteropatia severa arti inferiori

ALGORITMO SHOCK CARDIOGENO DA SINDROME CORONARICA ACUTA

ATTIVAZIONE DELLA SALA DI EMODINAMICA:
incannulamento a. femorale e stima della pressione telediastolica ventricolare sx (LVEDP)

+

ATTIVAZIONE ANESTESISTA:
valutare se necessario IOT, posizion. CVC giugulare

LVEDP> 18 mmhG

IMPELLA E SUCCESSIVA RIVASCOLARIZZAZIONE

Ricovero pz in letti intensi (Margherita)
in cogestione RR-cardiologia
Posizionamento Swan-ganz

CPO: $\frac{MAP \times CO}{451}$

PAPI: $\frac{sPAP - dPAP}{RA}$

CPO>0.6 e PAPI>1:

Scalare inotropi e vasopressori

togliere IMPELLA

se CPO persiste <0.6
Valutare trasferimento per più avanzato supporto emodinamico

CPO>0.6 e PAPI<1:
Valutare trasferimento per diverso supporto emodinamico