



GIOVEDÌ 29

La golden hour nella gestione dello shock: fare tanto, fare in fretta

Marco Marini, *Ancona*



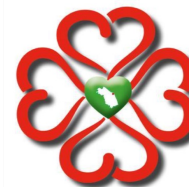


Conoscere e Curare il Cuore 2024



**“There is a golden hour between life and death.
If you are critically injured you have less than 60
minutes to survive. You might not die right then;
it may 3 days or 2 weeks later but something has
happened to your body that is irreparable”**

Dr. Adams Cowley pioneer of management of shock



OSPEDALI
RIUNITI

UNIVERSITÀ
POLITECNICA
DEL MARCHE

LANCISI

Centro Cardiovascolare
di Alta Specializzazione
del Medio Adriatico



Conoscere e Curare il Cuore 2024

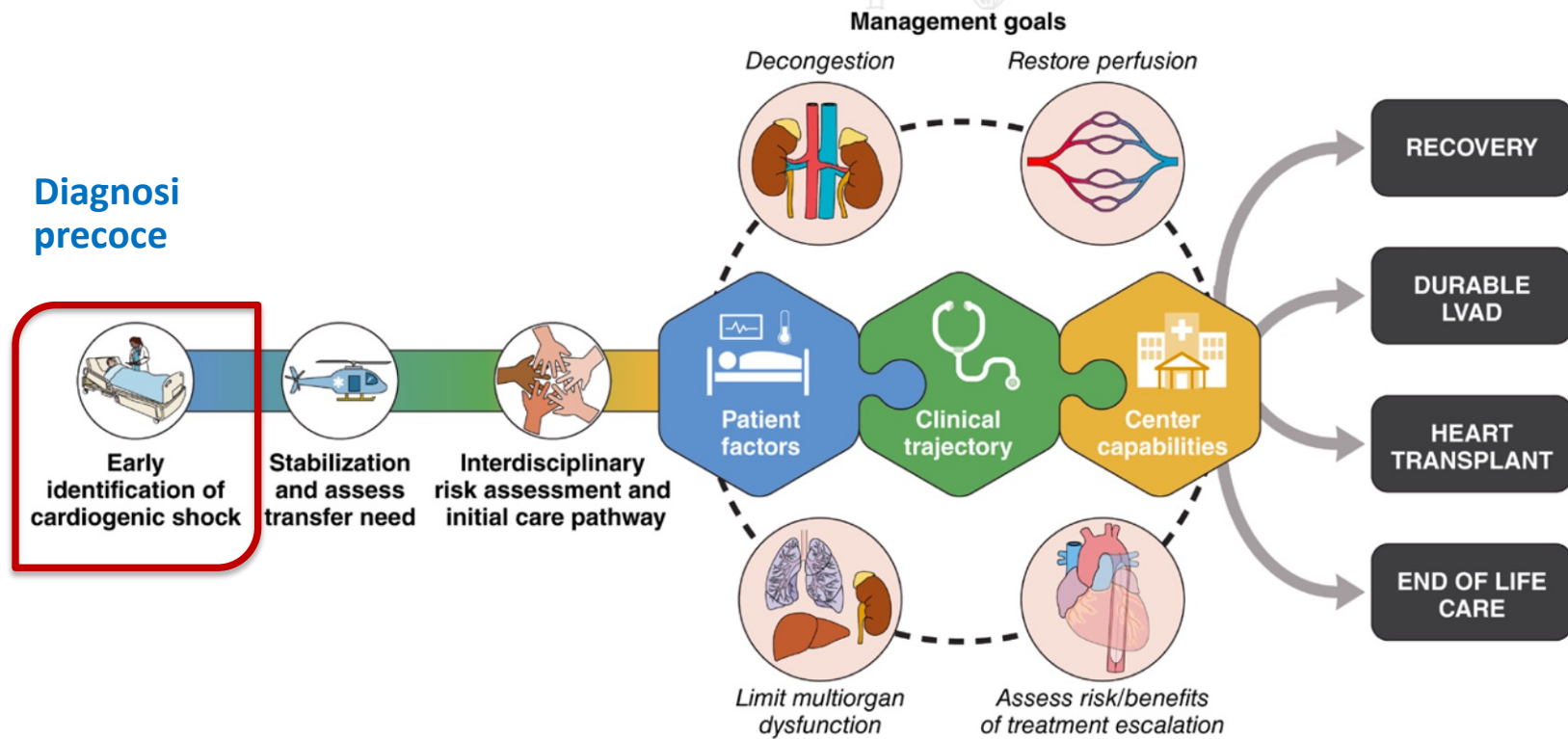
Disclousure

Member of Advisory Board for Abiomed, Astra Zeneca, Boehringer Ingelheim
Consultant for GADA

La golden hour nella gestione dello shock: fare tanto, fare in fretta

- Diagnosi precoce
- Stabilire “il grado”
- Etiologia
- Iniziare un trattamento
- Ipotizzare una traiettoria







La definizione di shock cardiogeno

SHOCK (1999) [13]	CARDSHOCK (2015) [8]	SCAI (2019) [22]					ESC (2021) [4]
		A—At Risk	B—Beginning	C—Classic	D—Deteriorating	E—Extremis	
		Clinical Criteria					
ACS cause Clinical criteria: a. SBP < 90 mmHg for at least 30 min to maintain an S/need for supportive measures, BP ≥ 90 mm Hg and b. end-organ hypoperfusion (cool extremities or UO < 30 mL per hour, and HR ≥ 60 bpm)	Cardiac cause and a. SBP < 90 mmHg (after adequate fluid challenge) for 30 min/need for vasopressor therapy to maintain SBP > 90 mmHg and b. signs of hypoperfusion (altered mental status/confusion, cold periphery, oliguria < 0.5 mL/kg/h for the previous 6 h, or lactate > 2)	SBP ≥ 100 mm Hg	a. SBP < 90 or MAP < 60 or >30 mmHg drop from baseline b. HR ≥ 100	SBP < 90 or MAP < 60 or >30 mmHg drop from baseline and drugs/MCS to maintain SBP > 90 mmHg	any of stage C and requiring multiple pressors OR addition of MCS to maintain perfusion	- No SBP - PEA or refractory VT/VF - BP < 90 mmHg despite maximal support	Cardiac cause and a. clinical signs of hypoperfusion (cold sweated extremities, oliguria, mental confusion, dizziness, narrow PP) b. biochemical manifestations of hypoperfusion (creatinine ↑, metabolic acidosis, lactate ↑)
		Biochemical markers					
		Normal creatinine, lactate	Lactate < 2, creatinine ↑/-, BNP ↑	-lactate > 2 -creatinine × 2 -LFT, BNP ↑	Any of Stage C and deteriorating	- CPR (A-modifier) - pH ≤ 7.2 - lactate ≥ 5	
Hemodynamic criteria: a. CI ≤ 2.2 L/min/m ² and b. PCWP ≥ 15 mm Hg		Physical exam					
		Normal JVP, clear lung sounds, strong distal pulses, normal mentation	↑ JVP, rales, strong distal pulses, normal mentation	↑ JVP, rales, unwell/agitated, cold, clammy extremities, UO < 30 mL/h	Any of stage C	Cardiac collapse	



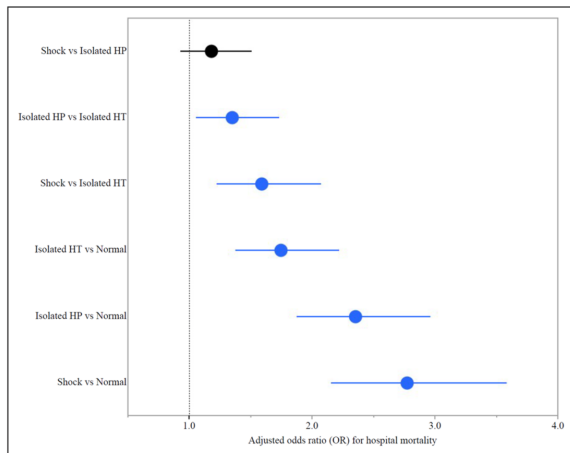
La golden hour nella gestione dello shock: fare tanto, fare in fretta

Shock: international Consensus Conference 2007

- Maldistribuzione del flusso ematico:
 - ✓ Insufficiente delivery o utilizzazione di O₂
 - ✓ Ipossia da ipoperfusione

- Ipotensione: PAS < 90 mmHg, Δ ↓ 40 mmHg PAS, PAM < 65 mmHg
- Spesso presente ma non necessaria
- L'inadeguata perfusione tissutale è l'elemento centrale nello SHOCK:
"NEI CASI AMBIGUI E' MANDATORIO GUARDARE I MARKER DI IPOPERFUSIONE".





Defining Shock and Preshock for Mortality Risk Stratification in Cardiac Intensive Care Unit Patients

Jacob C. Jentzer, MD*; Barry Burstein¹, MD*; Sean Van Diepen², MD, MSc; Joseph Murphy, MD; David R. Holmes Jr., MD; Malcolm R. Bell, MD; Gregory W. Barsness, MD; Timothy D. Henry³, MD; Venu Menon, MD; Charanjit S. Rihal, MD, MBA; Srihari S. Naidu, MD; David A. Baran, MD

Term	Definition
Hypotension	Presence of any of the following criteria
	Admission systolic BP <90 mm Hg
	Minimum systolic BP <90 mm Hg during first 1 h
	Admission MAP <60 mm Hg
Hypoperfusion	Minimum MAP <60 mm Hg during first 1 h
	Presence of any of the following criteria
	Admission lactate >2 mmol/L
	Urine output <720 mL during first 24 h
Tachycardia	Creatinine increased by ≥0.3 mg/dl during first 24 h
	Presence of any of the following criteria
	Admission HR >100 BPM
	Maximum HR >100 BPM during first 1 h
Group	Admission HR > admission systolic BP
	Mean HR > mean systolic BP during first 1 h
Normal	Study definition
Isolated hypotension	Neither hypotension nor hypoperfusion
Isolated hypoperfusion	Hypotension without hypoperfusion
Shock	Hypoperfusion without hypotension
	Both hypotension and hypoperfusion



Home/Satellite Centre



Ambulance

Oxygen administration
Peripheral vein catheter
Fluid infusion
ECG suggesting MI-Aspirin

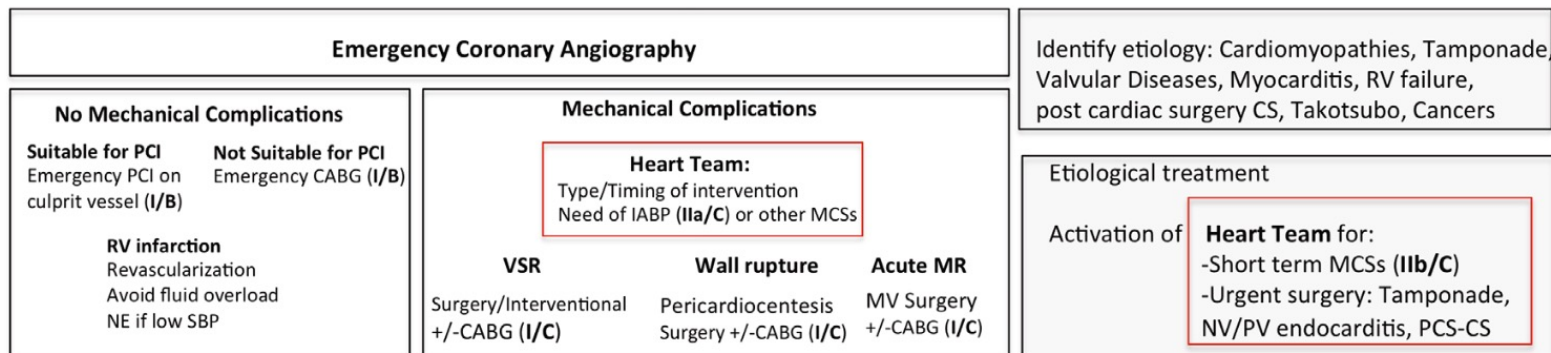


Emergency Department

O² or Ventilatory support
Hemodynamic instability: NE or/and Dob; if CA, continues IMV and ECMO
12 leads ECG and Echocardiography (I/C)

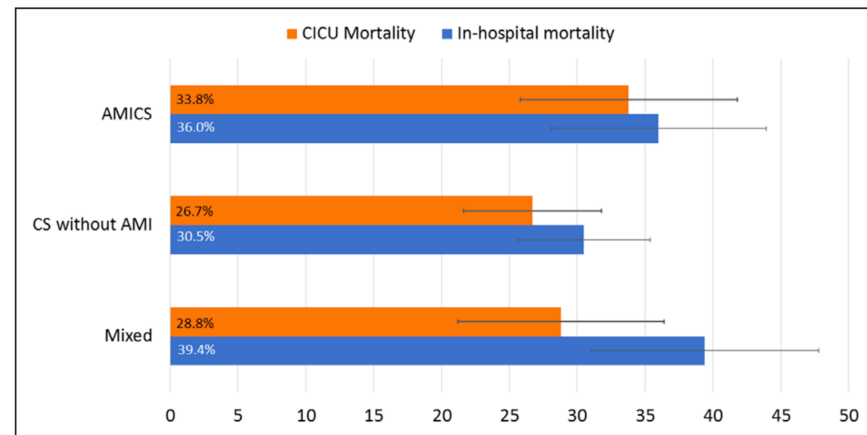
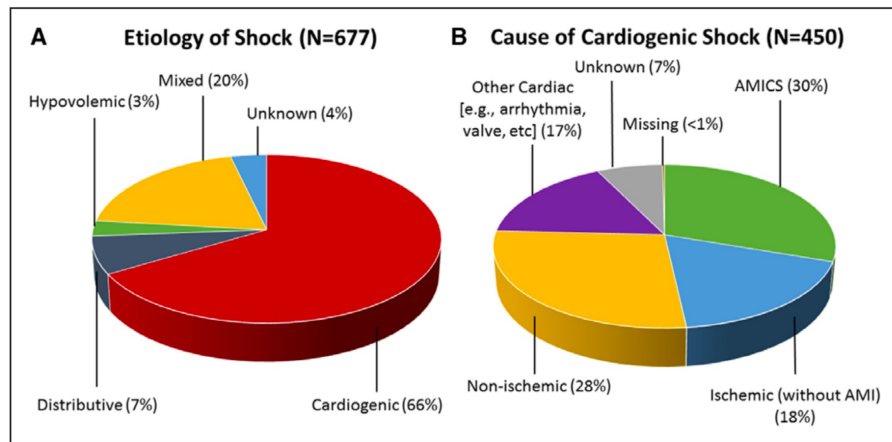
AMI related CS

Not AMI related CS





Etiologia



Circulation: Cardiovascular Quality and Outcomes

ORIGINAL ARTICLE

Epidemiology of Shock in Contemporary Cardiac Intensive Care Units

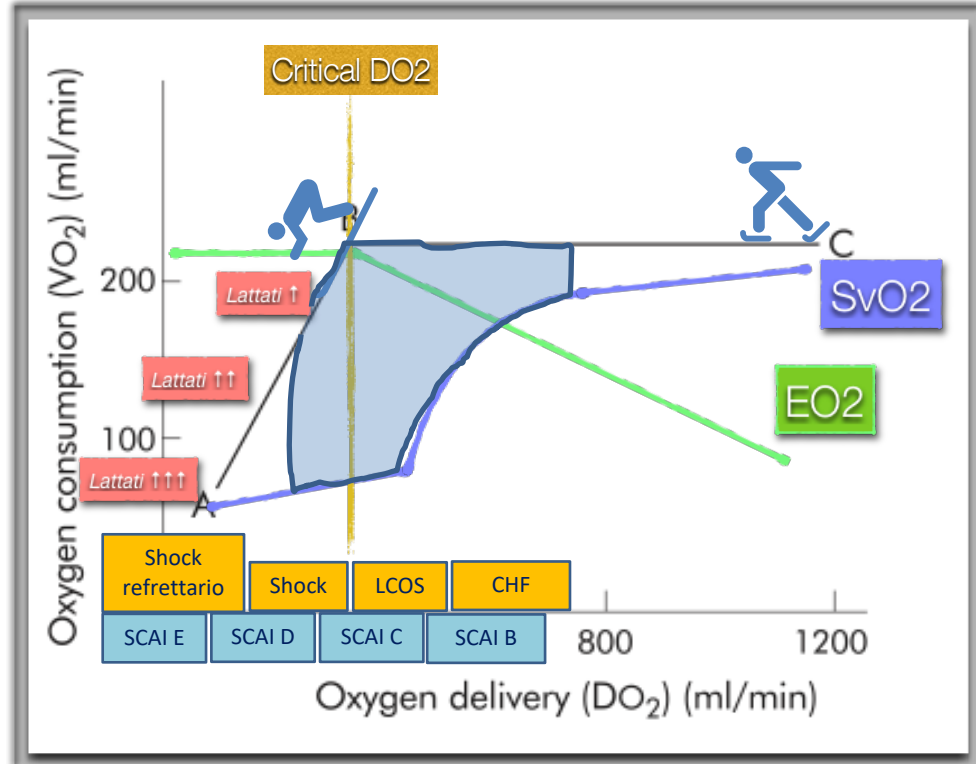
Data From the Critical Care Cardiology Trials Network Registry

Circulation: Cardiovascular Quality and Outcomes 2019



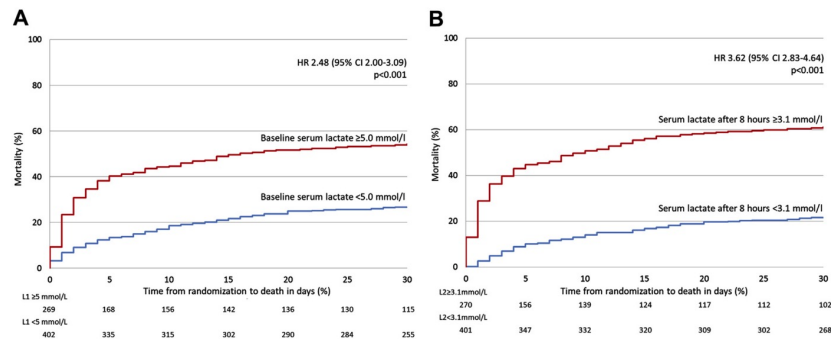
Stabilire il “grado”

SCAI SHOCK STAGE	A	B	C	D	E	BIOMARKERS
Perfusion						
Microvasculature			Endothelial activation swelling and vasoconstriction	Capillary rarefaction, stasis and leakage, microthrombosis, leukocyte adhesion		
Kidney			Oliguria (<0.5ml/h/kg) Hypoperfusion	Anuria (<100ml/24h) Tubulus necrosis		Creatinine
Liver			Hypoxic hepatitis Hepatocyte damage GOT/GPT ↑	Acute liver failure Liver cell necrosis Ischemic cholangiopathy → SSC-CIP; Bilirubin ↑		GOT/GPT Bilirubin L-FABP?
Gut			Loss of Integrity Bacterial translocation	NOMI Intestinal necrosis		Lactate LDH? IFABP? L-FABP?
Immune System			HMGB1, S100, HSPs, mtDNA IL-6, IFN-γ, TNF-α, MCP-1, IL-1, IL-8	HMGB1, S100, HSPs, mtDNA INOS activation, DPP-3 IL-6, IFN-γ, TNF-α, MCP-1, IL-1, IL-8		mtDNA?
Inflammatory activation						CRP IL-6 DPP3?
Systemic Vascular Resistance						
Lactate						Lactate
Systemic consequences			Multi organ dysfunction syndrome (MODS)	Multi organ failure Death		
Therapeutic options	Vasodilator		Dobutamine, Levosimendan Norepinephrine		Mechanical circulatory support	

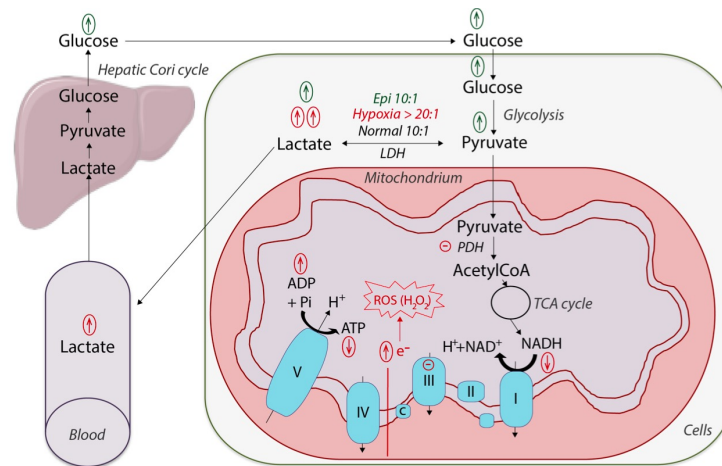


Arterial Lactate in Cardiogenic Shock

Prognostic Value of Clearance Versus Single Values



Stabilire il “grado”



HYPOXIC STATE

Glucose production continues
Pyruvate production continues
Mitochondrial dysfunction
Lower ATP:ADP and NADH:NAD ratios
Pyruvate dehydrogenase (PDH) inhibition
Increased lactate production
Higher Lactate:Pyruvate ratio ($> 20:1$)

HIGH DEMAND STATE (e.g. Epinephrine)

Increased glucose production as stress response
Increased pyruvate production
Increased lactate production
Lactate:Pyruvate ratio maintained at 10:1

QUANDO LA SVCO₂ NON È VALUTABILE O ATTENDIBILE?... Δ PCO₂ A-V

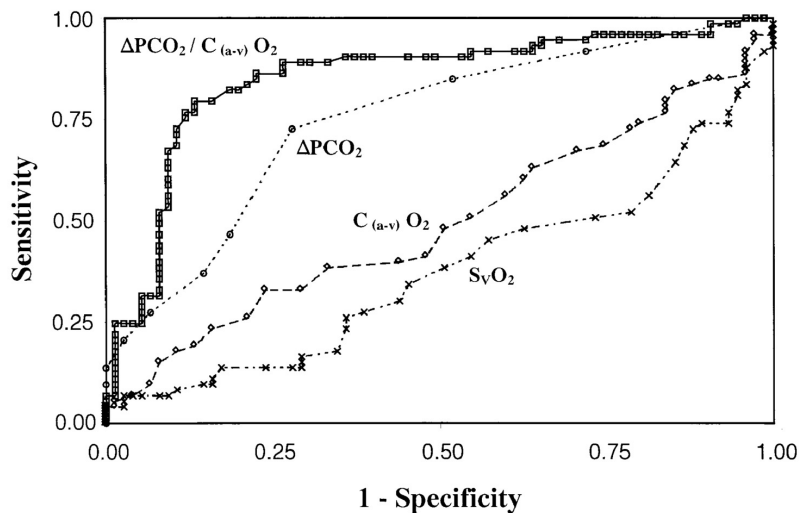
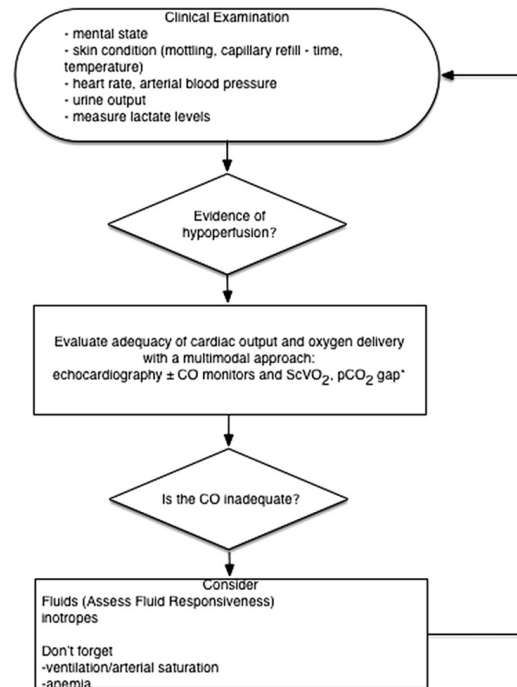


Fig. 1 Receiver operating characteristic curves of the studied O₂- and CO₂-derived parameters for the prediction of hyperlactatemia (arterial lactate level ≥ 2 mmol/l)

VALORI PATOLOGICI > 6 MMHG



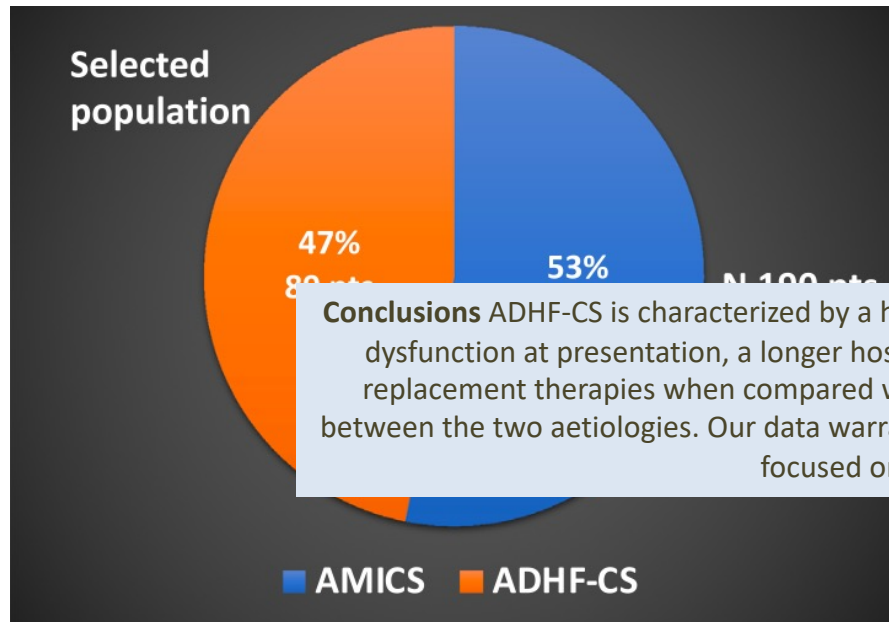
*
if ScVO₂ > 70% assess pCO₂ gap. A pCO₂ gap > 0.8 kpa or 6 mmHg suggests that inadequacy of cardiac output is still present



Etiologia

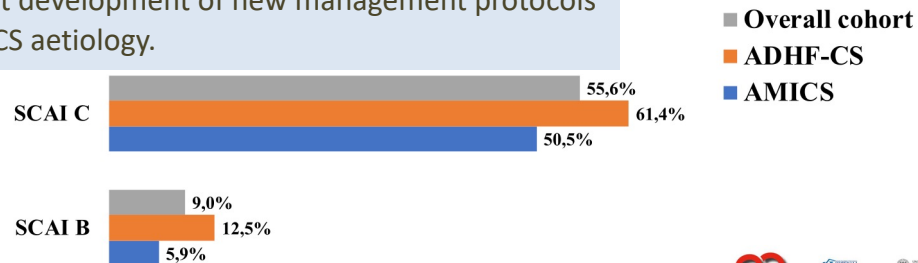
ESC Heart Failure (2023)

Published online in Wiley Online Library (wileyonlinelibrary.com) DOI: 10.1002/ehf2.14510



Conclusions ADHF-CS is characterized by a higher prevalence of end-organ and biventricular dysfunction at presentation, a longer hospital length of stay, and higher need of heart replacement therapies when compared with AMI-CS. In-hospital mortality was similar between the two aetiologies. Our data warrant development of new management protocols focused on CS aetiology.

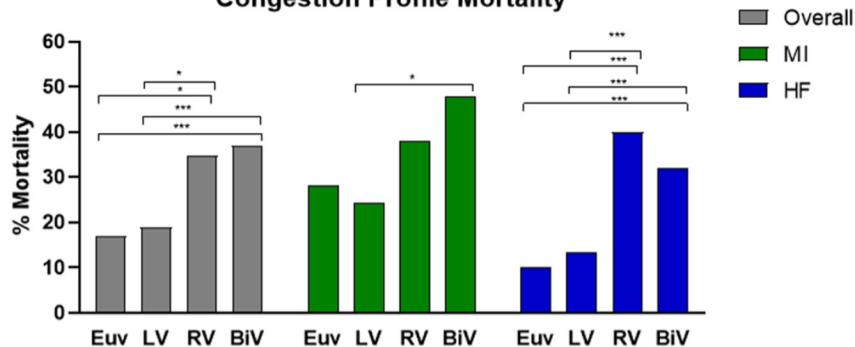
Differences between cardiogenic shock related to acute decompensated heart failure and acute myocardial infarction





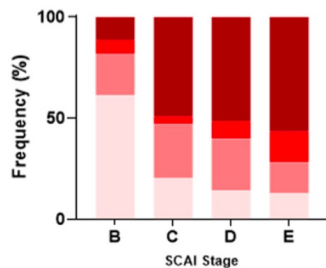
Etiologia

Congestion Profile Mortality

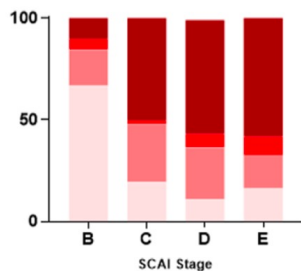


Circ Heart Fail. 2020;13:e007099.

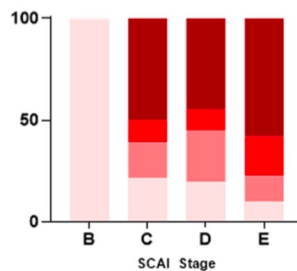
Overall



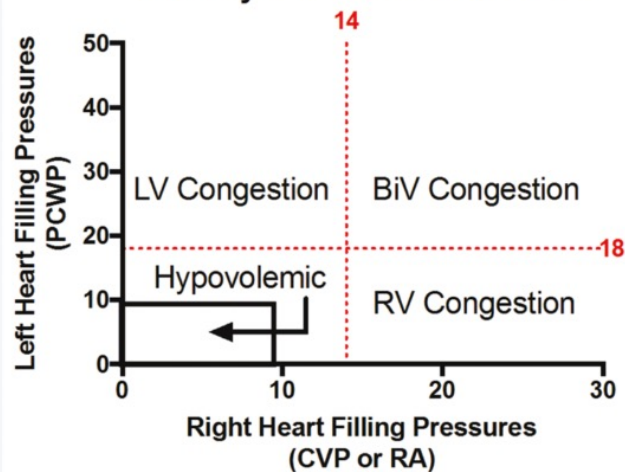
HF



MI

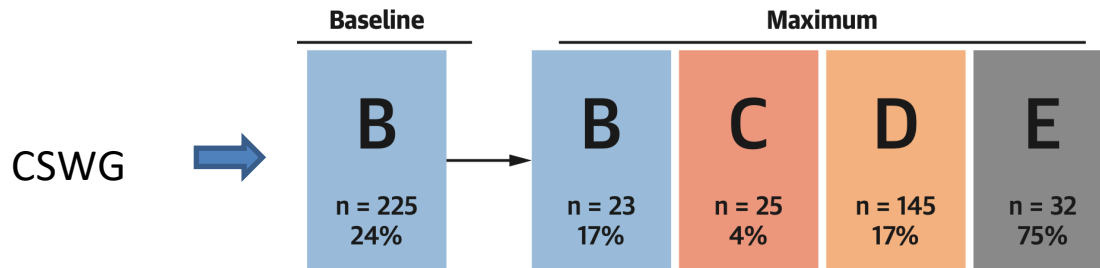


Hemodynamic Profiles in Shock

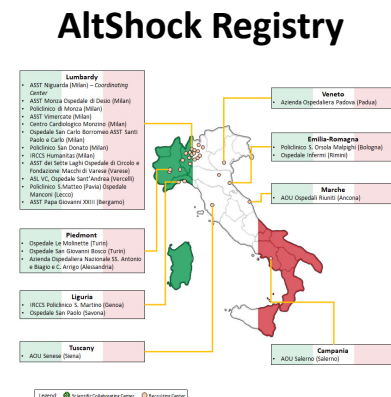


Background: SCAI B patients deteriorate in higher stages with contemporary therapeutic approaches

Altshock-2 Registry Population (237 patients)												
Basal Evaluation	SCAI B			SCAI C			SCAI D			SCAI E		
Oh	20 pts			131 pts			61 pts			25 pts		
(Available for 237 patients)	(8%)			(55%)			(26%)			(11%)		
Re-Evaluation												
SCAI Class Variations	41.7%	50%	8.3%	6.7%	61.2%	32.2%	11.3%	43.4%	45.3%	0%	37.5%	62.5%
	Worsened	Unchanged	Improved	Worsened	Unchanged	Improved	Worsened	Unchanged	Improved	Worsened	Unchanged	Improved



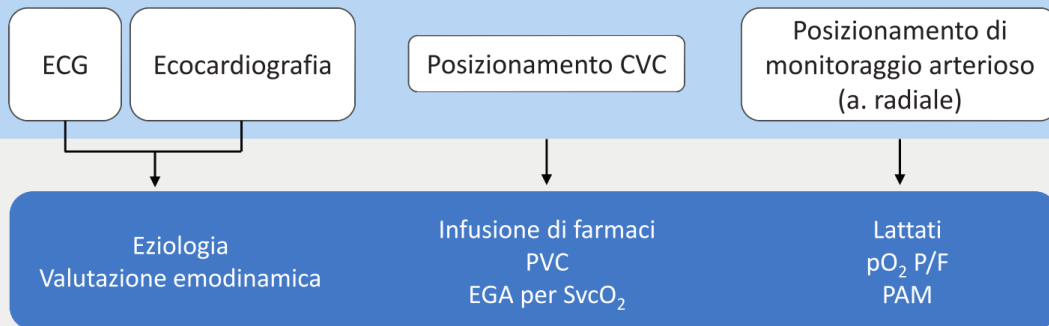
JACC 2022; 80: 185-198



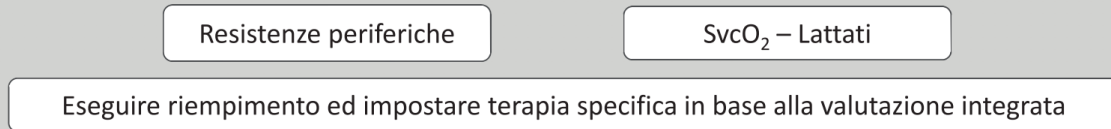


PAZIENTE CON SOSPETTO SHOCK CARDIOGENO

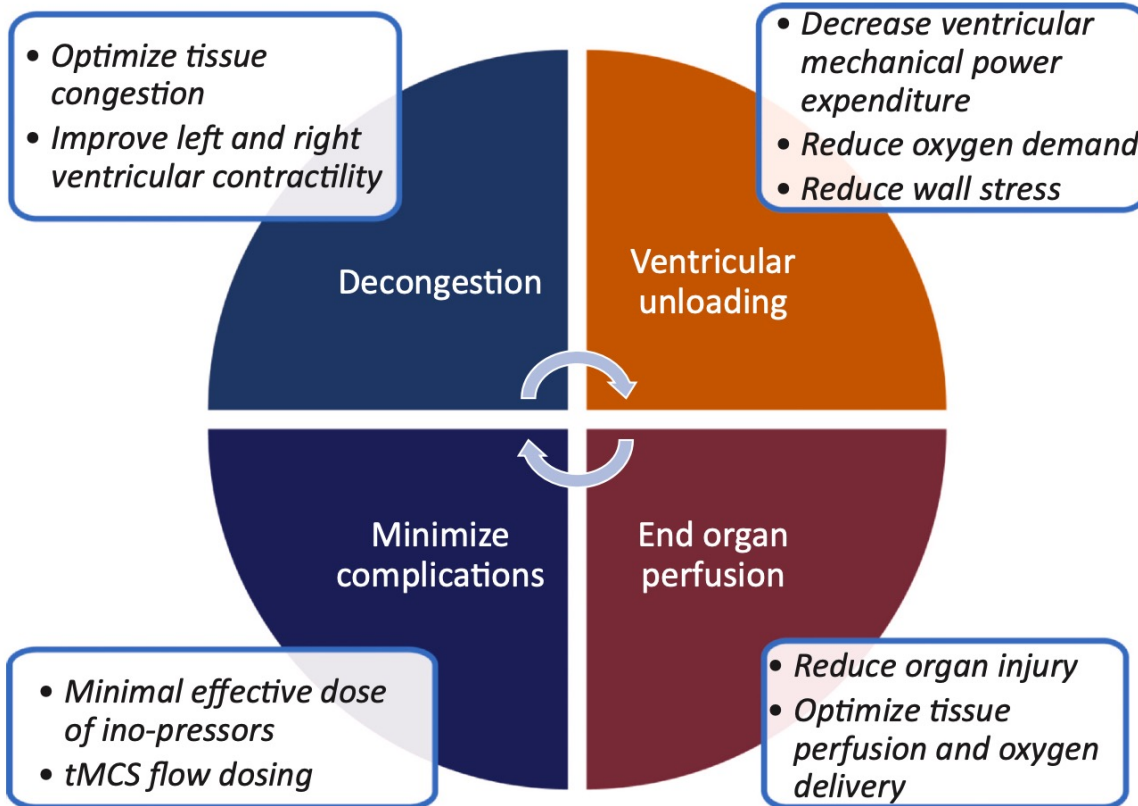
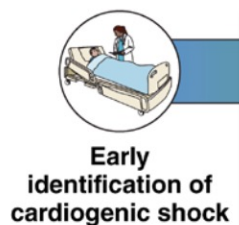
Primo step da effettuare subito



Secondo step



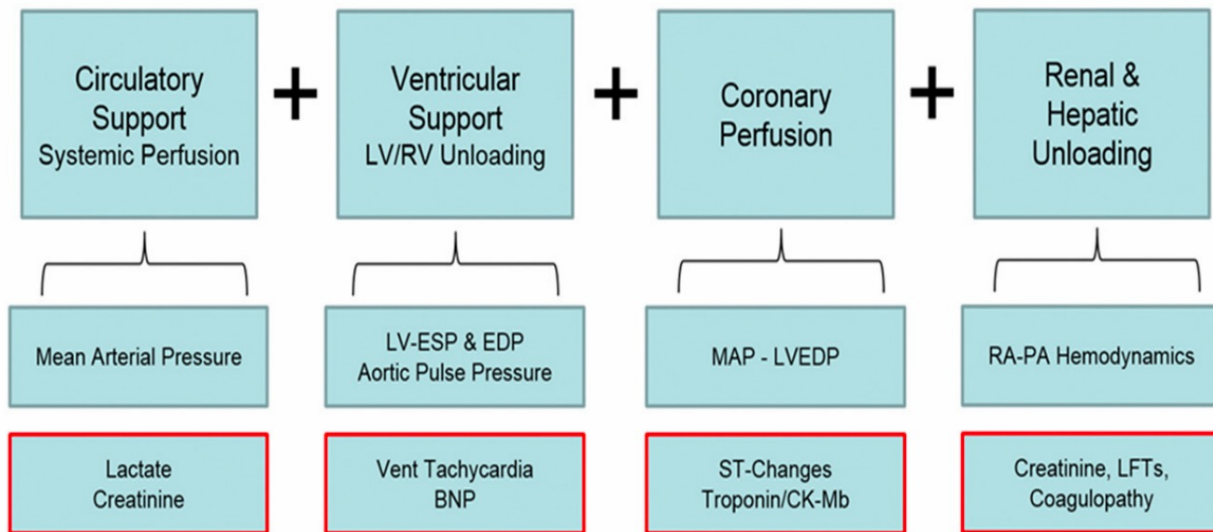
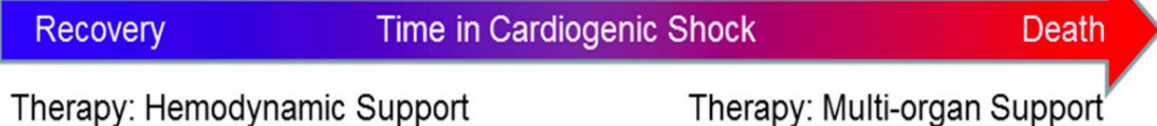
Rivalutazione dopo 30 min ed ogni 30-60 min



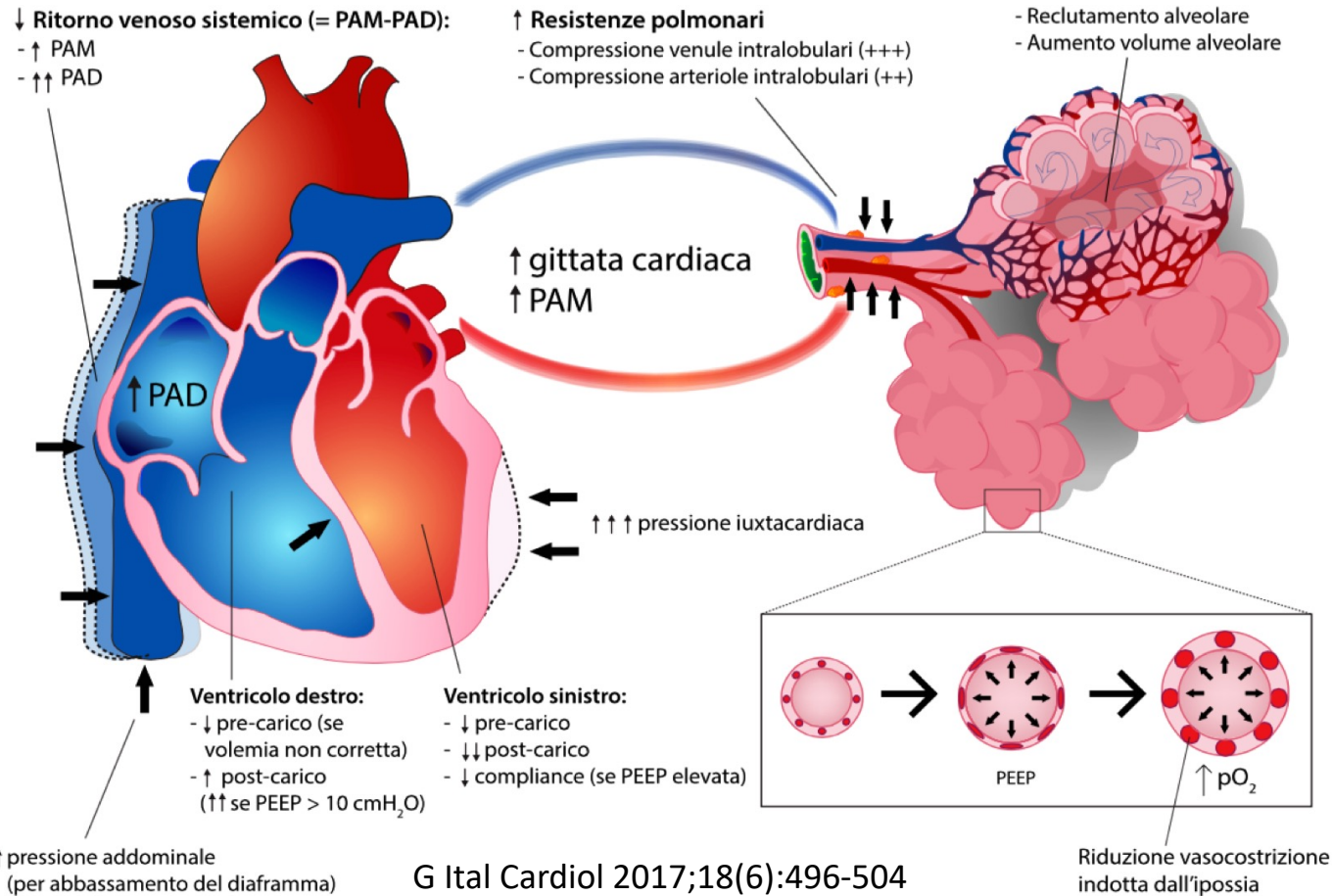


Hemodynamic Problem

Hemo-Metabolic Problem



circulatory support - ventricular unloading - myocardial perfusion - decongestive strategies



J. Clin. Med. 2023, ↑ pressione addominale
(per abbassamento del diaframma)

G Ital Cardiol 2017;18(6):496-504



CARDIOGENIC SHOCK

Inadequate cardiac output

PLUS

Clinical signs of hypoperfusion

AND/OR

Biochemical manifestations of hypoperfusion



Few evidence-based data

AMI-CS

First-line therapy:

- ☞ Norepinephrine (if hypotension prevails)
- ☞ Dobutamine OR Milrinone OR Levosimendan (if CS «normotensive»)

Second-line therapy:

- ☞ Norepinephrine + Dobutamine/Levosimendan

ADHF-CS

First-line therapy:

- ☞ Norepinephrine (if hypotension prevails)
- ☞ Dobutamine OR Milrinone OR Epinephrine + Sodium Nitroprusside OR Levosimendan (if CS «normotensive»)

Second-line therapy:

- ☞ Norepinephrine + Dobutamine/Levosimendan

RV-predominant CS

First-line therapy:

- ☞ Norepinephrine (if hypotension prevails)
- ☞ Milrinone OR Levosimendan (if CS «normotensive»)

Second-line therapy:

- ☞ Norepinephrine + Milrinone/Levosimendan/Dobutamine



Shock cardiogeno: terapia farmacologica e NON... Il ventricolo dx come «cornerstone» nel decision making

Deteriorating shock (SCAI-C and D)—failure to respond to initial therapy

Consider mechanical support

Clinical signs of (relative) hypoperfusion: mottled, cold, clammy, volume overload, extensive rales, (non)-invasive ventilation, alteration in mental status

RV failure

Central venous pressure (CVP) ≥ 15 mmHg

Pulmonary artery pulsatility index (PAPi) ≤ 1.85 ⁷

Right atrial to pulmonary capillary wedge pressure ratio (RA/PCWP) ≥ 0.8 ⁷

LV failure

Systolic blood pressure (SBP) ≤ 90 mmHg or mean arterial pressure (MAP) < 60 or > 30 mmHg drop and inotropes/vasopressors

Cardiac index (CI) < 2.2 L/min/m²

Cardiac power output (CPO) < 0.6 W⁸

Left ventricular end-diastolic pressure (LVEDP) > 15 mmHg

Table 2 Haemodynamic profiles of cardiogenic shock subtypes, from Saxena et al.¹⁴

Haemodynamic variables	Normotensive hypoperfusion	Hypotensive normoperfusion	LV dominant shock	RV dominant shock	BIV shock
SBP, mmHg	>90	<90	<90	<90	<90
CVP, mmHg	Variable	Variable	<14	>14	>14
PCWP, mmHg	Variable	Variable	>18	<18	Variable
CVP/PCWP	Depends on degree of LV and RV involvement	Depends on degree of LV and RV involvement	<0.86	>0.86	>0.86
Pulmonary artery pulsatility index	Depends on degree of RV involvement	Depends on degree of RV involvement	>1.5	<1.5 ^a	<1.5
CI, L/min/m ²	<2.2	≥2.2	<2.2	<2.2	<2.2
SVR, dynes-s//cm ²	>1600	800-1600	800-1600	800-1600	800-1600
CPO, W	Variable	Variable	<0.6	<0.6	<0.6

BIV indicates biventricular; CPO, cardiac power output; CVP, central venous pressure; LV, left ventricular; PAD, pulmonary artery diastolic pressure; PAS, pulmonary artery systolic pressure; PAPl, pulmonary artery pulsatility index PAPl = (PAS - PAD)/CVP; PCWP, pulmonary capillary wedge pressure; RA, right atrial pressure; and SVR, systemic vascular resistance.
^aRight ventricular (RV) dominant shock primarily attributable to RV dysfunction

Table 1 Clinical, echocardiographic, and haemodynamic markers which should aid assessment of congestion and hypoperfusion

	Clinical	Echocardiographic	Haemodynamic
Congestion	Bilateral lung crackles or rales Elevated JVP and/or hepatojugular reflux Peripheral oedema Hepatomegaly Orthopnoea Pleural effusion	Elevated LV or RV filling pressures Dilated and non-collapsing IVC Lung B-lines Altered doppler signal of hepatic, portal, and renal veins (VExUS)	Increased CVP (>12 mmHg) Increased PCWP (>15 mmHg)
Low cardiac output or hypoperfusion	Narrow pulse pressure (pulse pressure proportion < 25%) S ₃ Sensation of impending doom Alteration of mental status Cold and clammy extremities, cyanosis Oligoanuria Delayed capillary refill	Low LVOT VTI	Increased arterial lactate (>2 mmol/L) Reduced cardiac index (<2.2 L/min/m ²) Reduced cardiac power output (<0.6 W) Reduced cardiac power index (<0.4 W/m ²) Occult hypoperfusion Pulmonary artery O ₂ saturation <65% Arteriovenous delta CO ₂ > 6 mmHg

Acute heart failure: differential diagnosis and treatment

Marco Marini^{1*}, Roberto Manfredi^{1,2}, Ilaria Battistoni¹, Matteo Francioni¹,
Maria Vittoria Matassini¹, Giulia Pongetti¹, Luca Angelini¹, Matilda Shkoza¹,
Alessandro Bontempo^{1,2}, Leonardo Belfioretti¹, and Gian Piero Perna¹



Table 1.3 Recommendations for Timing of Acute MCS in CS

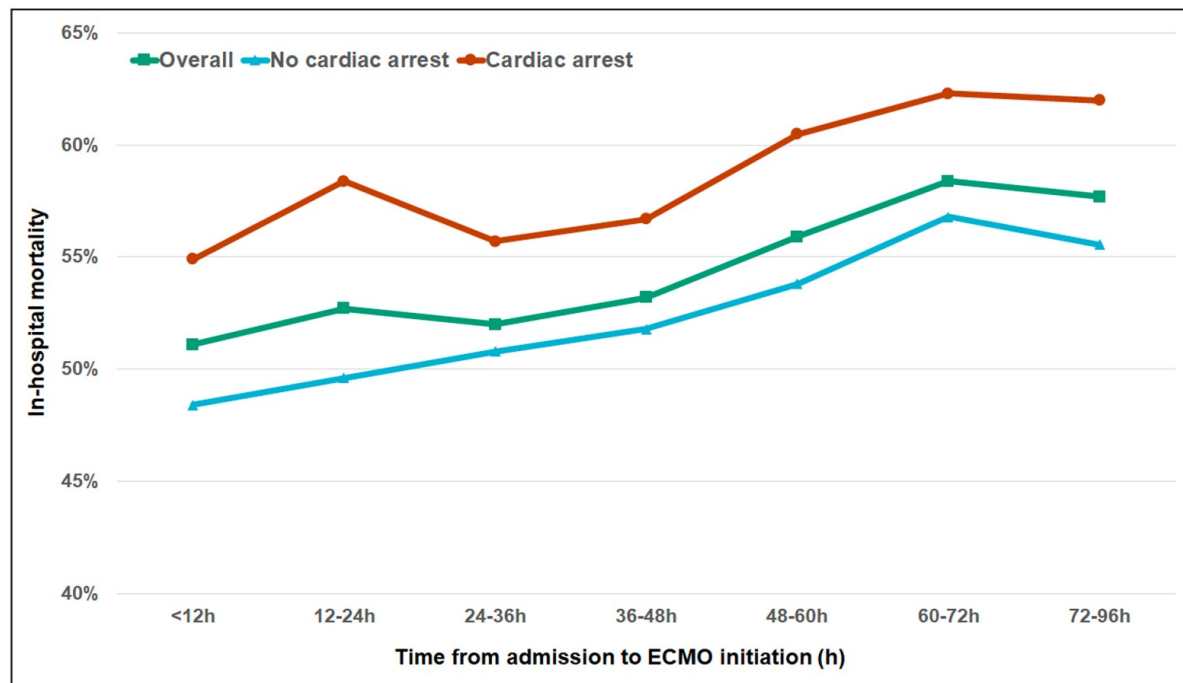
Recommendation	Class of Recommendation	Level of Evidence
Acute MCS should be initiated as soon as possible in patients with CS who fail to stabilize or continue to deteriorate despite initial interventions.	I	B
The use of acute MCS should be considered in patients with multiorgan failure to allow successful optimization of clinical status and neurologic assessment before placement of durable MCS or organ transplantation.	II	C
In patients with cardiac arrest receiving cardiopulmonary resuscitation, VA-ECMO can be considered.	II	C
When considering VA-ECMO, the need for left ventricular venting/unloading (pharmacologic or mechanical) should be considered.	II	B

MCS, mechanical circulatory support; VA ECMO, venoarterial extracorporeal membrane oxygenation.





Timing of Initiation of Extracorporeal Membrane Oxygenation Support and Outcomes Among Patients With Cardiogenic Shock

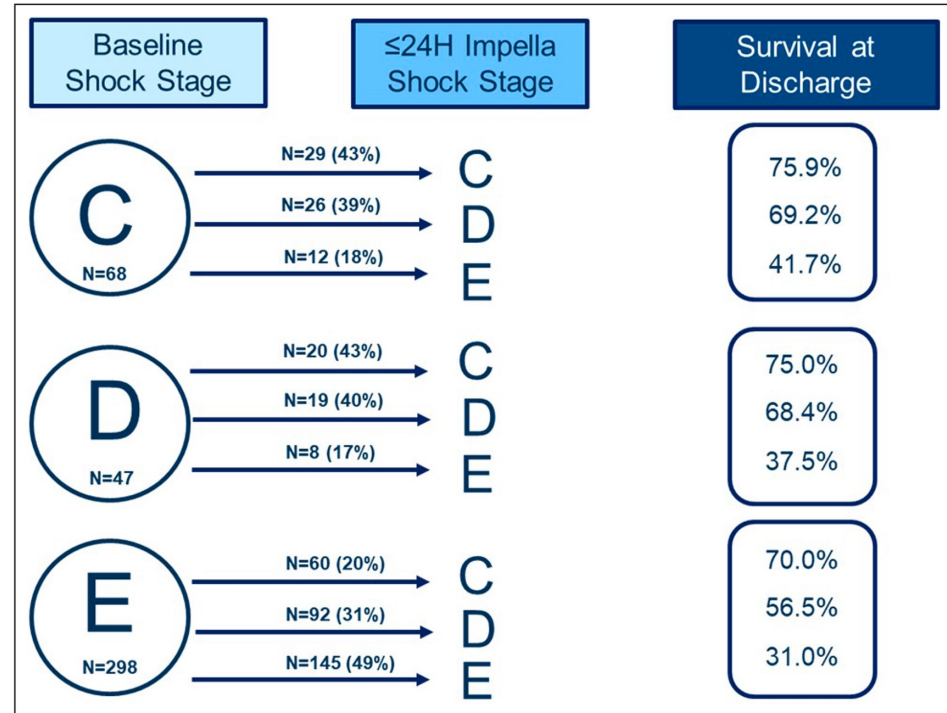


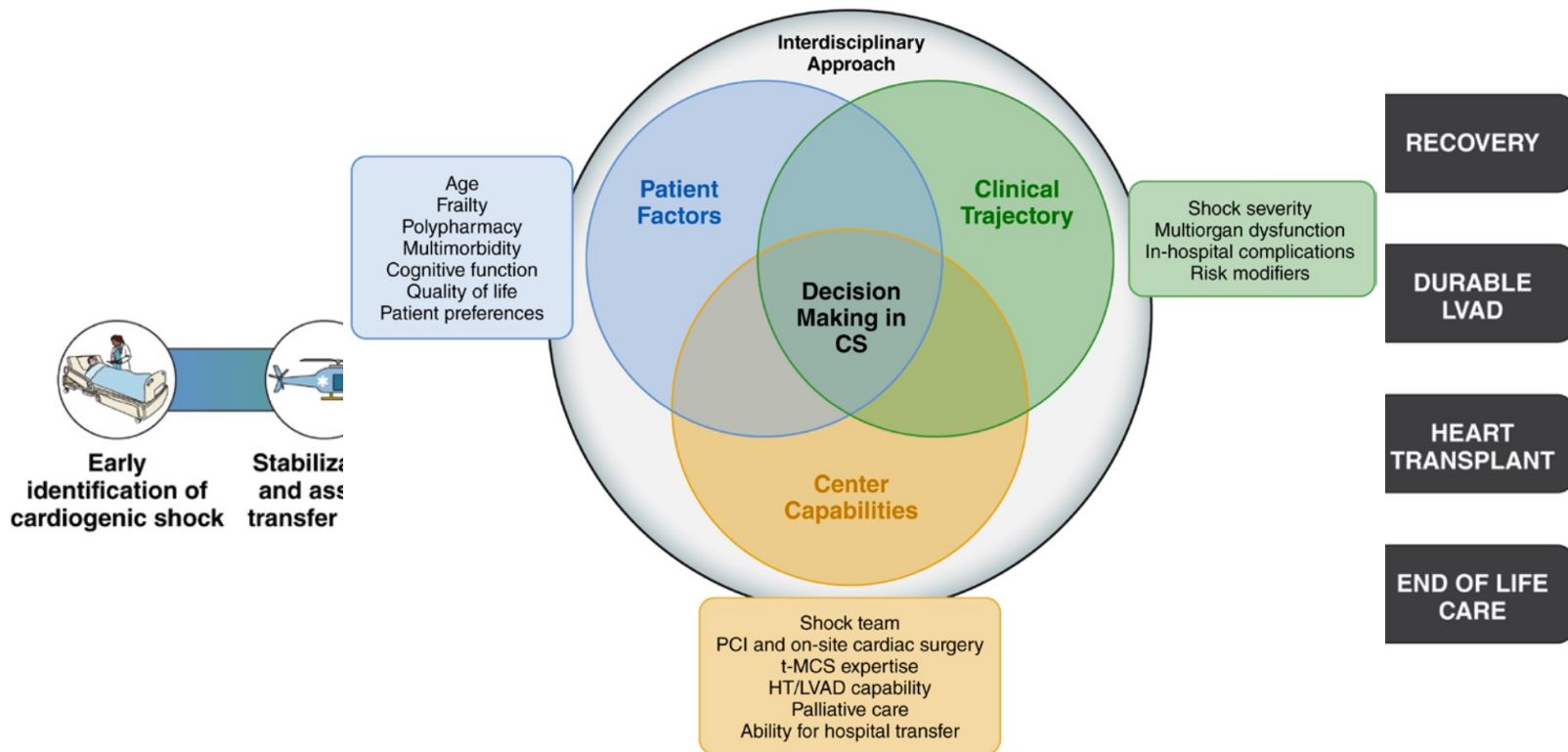


ORIGINAL RESEARCH

Treatment of Acute Myocardial Infarction and Cardiogenic Shock: Outcomes of the RECOVER III Postapproval Study by Society of Cardiovascular Angiography and Interventions Shock Stage

Best Practice	Metric Achieved
Impella Pre PCI	56.0%
Door to support < 90 min	29.9%
Achieve TIMI 3 flow	86.4%
Swan-Ganz/PA Catheter	85.8%
All 4 Best Practices Met?	10.8%





GOLDEN HOUR

nello Shock Cardiogeno

INQUADRAMENTO

Valutazione clinica
Elettrocardiogramma
Ecocardiogramma
Esami ematici ed emogasanalisi

0 min

EZIOLOGIA

Shock correlato a SCA o non correlato a SCA (es. ADHF)

DEFINIZIONE PROGNOSTICA (SCAI)

GESTIONE INIZIALE

Agenti vasoattivi
Supporto ventilatorio
Fluidi e gestione volemica
Studio coronarografico

30 min

SHOCK TEAM

Trasferimento in UTIC
Monitoraggio emodinamico
Supporto meccanico al circolo

60 min

MONITORAGGIO

Eventuale escalation therapy
Rivalutazione emodinamica

90 min

Grazie ai colleghi,
infermieri ed al network

Multidisciplinary
evaluation

Hemodynamics

nal Diagnostics

utic Intervention
Selection

$P = 0.016$

min

23% 29%

CICU Mortality



LANCISI
Centro Cardiovascolare
di Alta Specializzazione
del Medio Adriatico