I presupposti teorici

Come inquadro clinicamente la bassa portata e lo shock e la strategia terapeutica volta alla stabilizzazione : inotropi, vasodilatatori.

Erminio Sisillo
U.O. di Anestesia e Rianimazione
IRCCS Centro Cardiologico Monzino

PATIENT-CENTRED ACUTE CARE TRAINING

PACT

ESICM
EUROPEAN SOCIETY OF INTENSIVE CARE MEDICINE

The Intensive Connection

AN ESICM MULTIDISCIPLINARY DISTANCE LEARNING PROGRAMME FOR INTENSIVE CARE TRAINING

INTRODUCTION

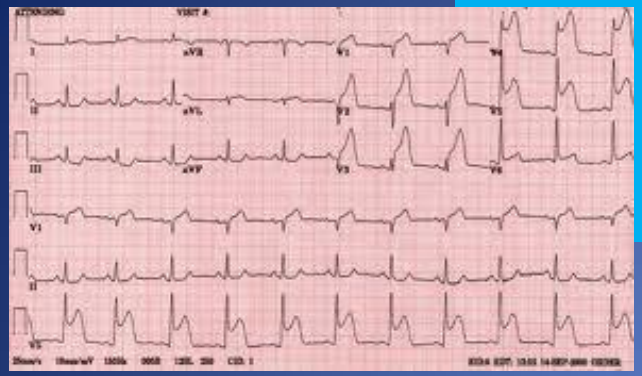
Haemodynamic instability is common in critically ill patients. When associated with signs of inadequate organ or tissue perfusion, whatever the cause, it may present as shock; a constellation of symptoms, signs and laboratory abnormalities that are a manifestation of tissue hypoperfusion.

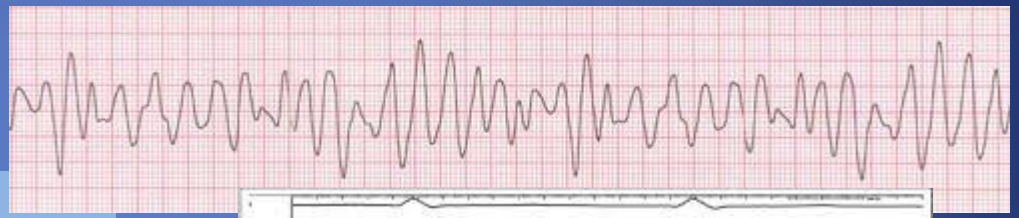
Haemodynamic instability, whatever the cause, is called circulatory shock.

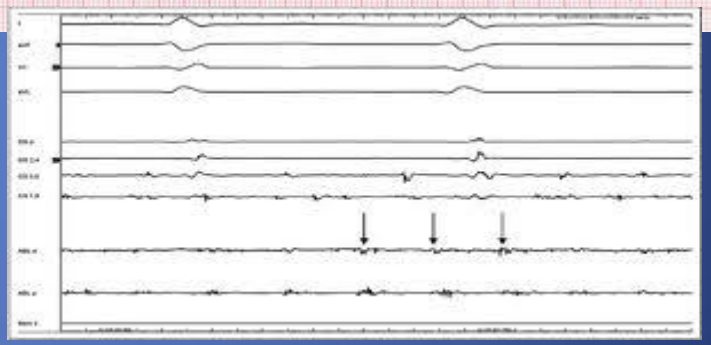
Patients who survive the initial phase of shock may then develop the multiple organ dysfunction syndrome (MODS), which is a major cause of late death in the intensive care unit (ICU). Although the pathophysiology of MODS is multifactorial and not always precisely defined, haemodynamic instability, reduced organ perfusion and alterations in tissue microcirculation resulting in tissue hypoxia play key roles in the onset and maintenance of the syndrome.

Sindrome coronarica acuta:

a) infarto miocardico acuto
b) angina instabile con ampia area ischemica
c) complicanze meccaniche dell'IMA
d) infarto del ventricolo destro



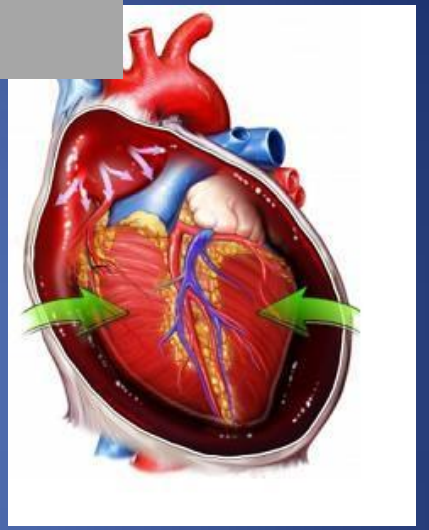


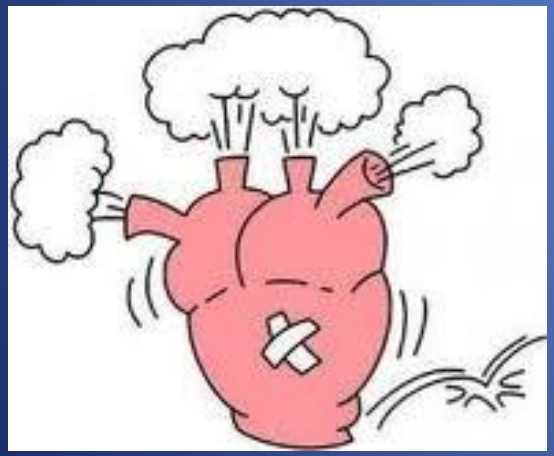


Aritmie acute : TV, FV,
FA, FLA, TPSV;
bradiaritmie critiche

Scompenso cardiaco cronico acutizzato
Miocardite acuta
Tamponamento cardiaco
Crisi ipertensiva
Stenosi aortica severa
Scompenso destro (*ipertensione polmonare, embolia polmonare, BPCO riacutizzata, polmonite massiva)*

Tumori cardiaci
Cardiomiopatia post-partum
Fattori extracardiaci (*farmaci, abuso di alcool o di droghe, insufficienza renale, feocromocitoma, etc*.)





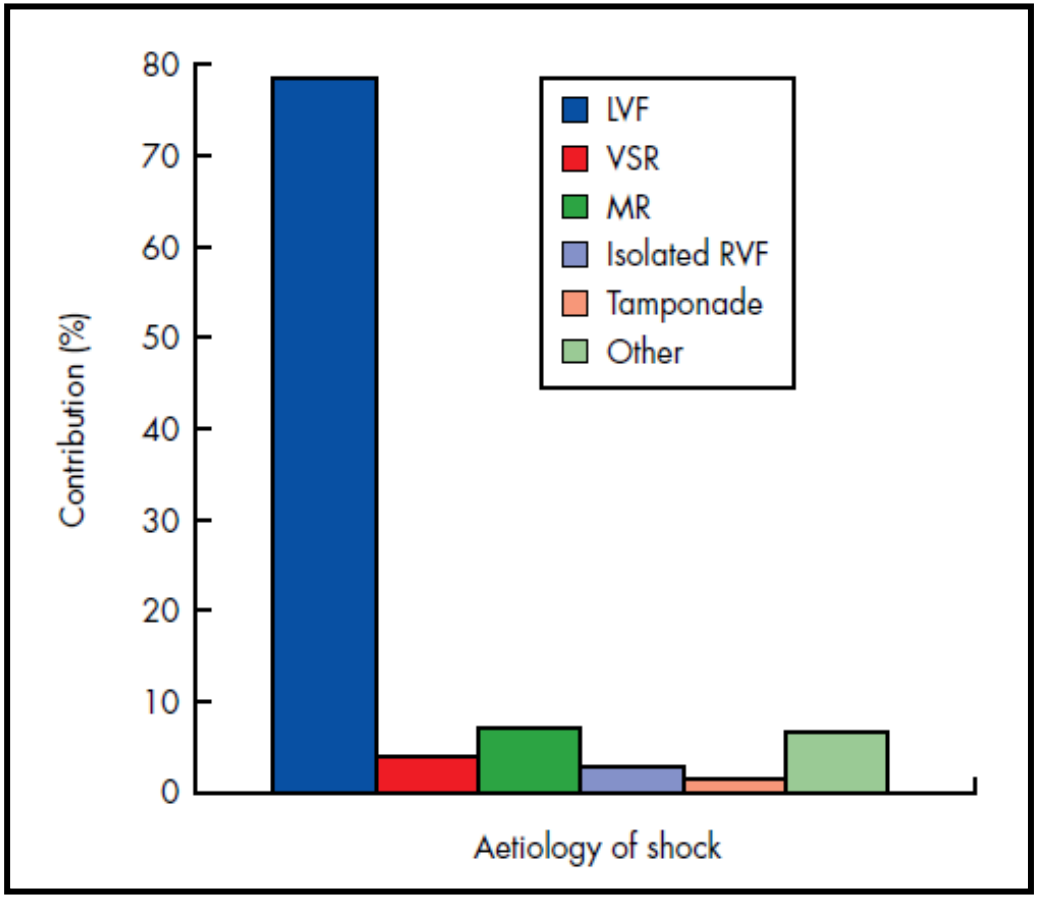
Sindromi da alta gettata

- setticemia
- anafilassi
- anemia
- crisi tireotossica
- sindromi da shunt

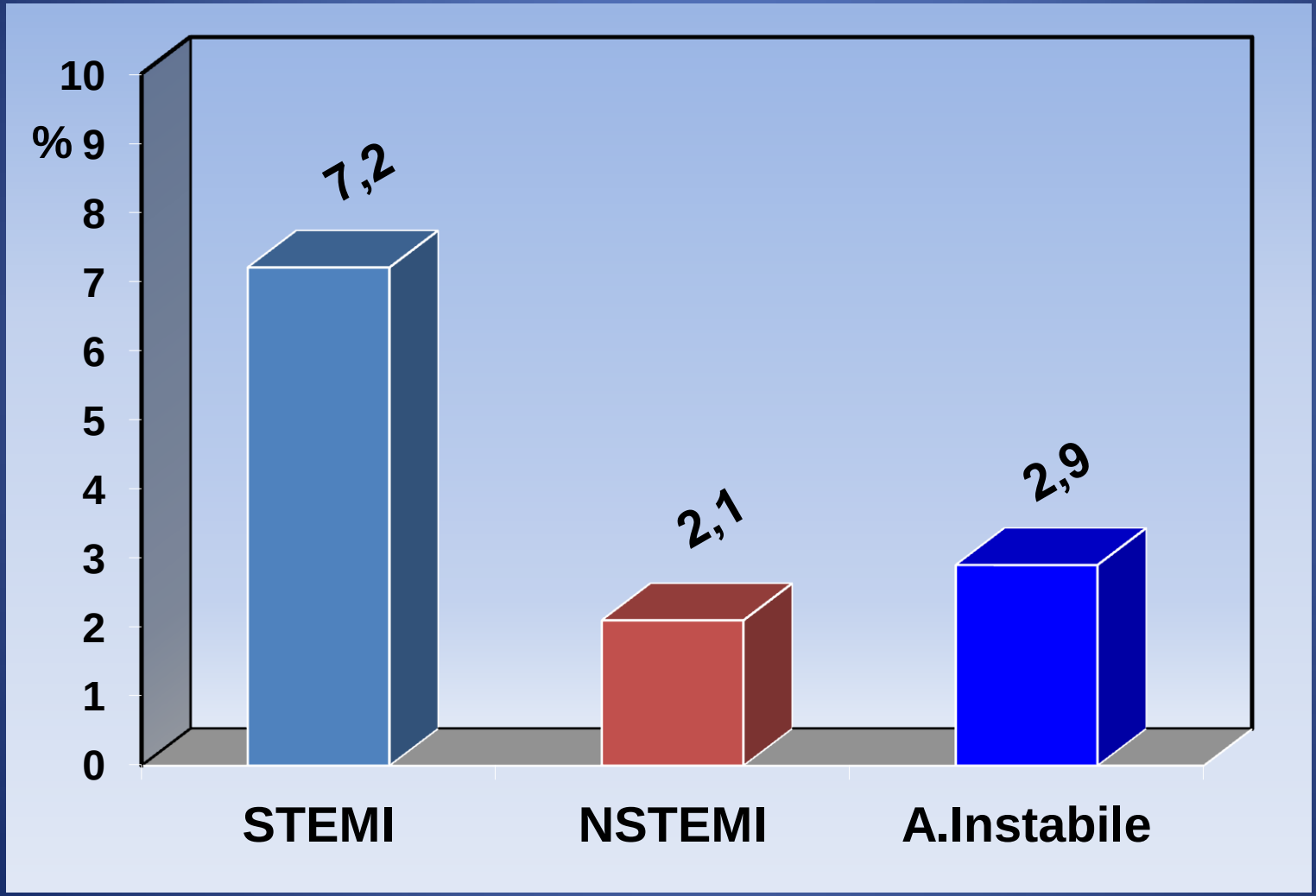
MANAGEMENT OF CARDIOGENIC SHOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION

Venu Menon, Judith S Hochman

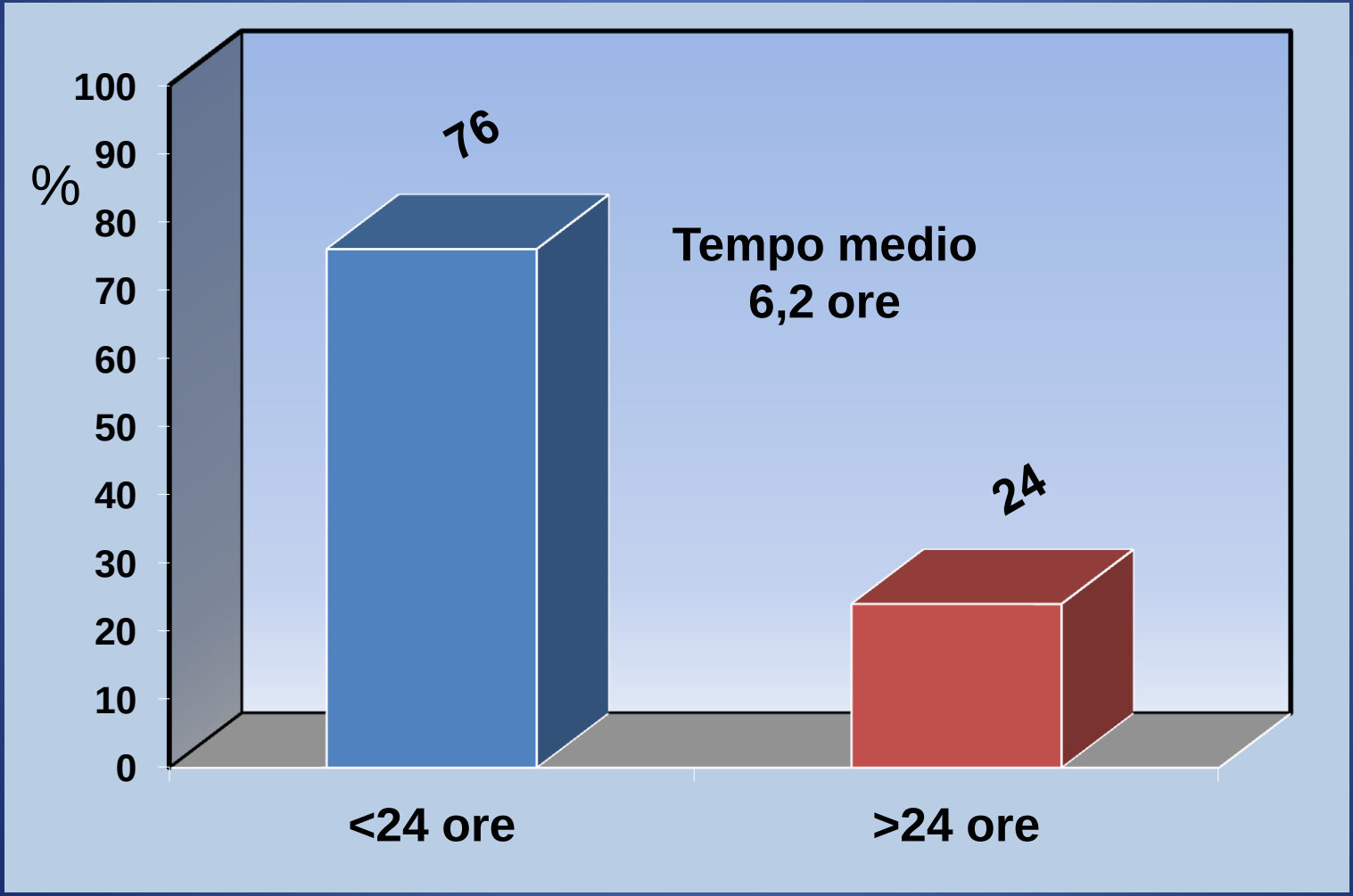
Heart 2002;**88**:531–537



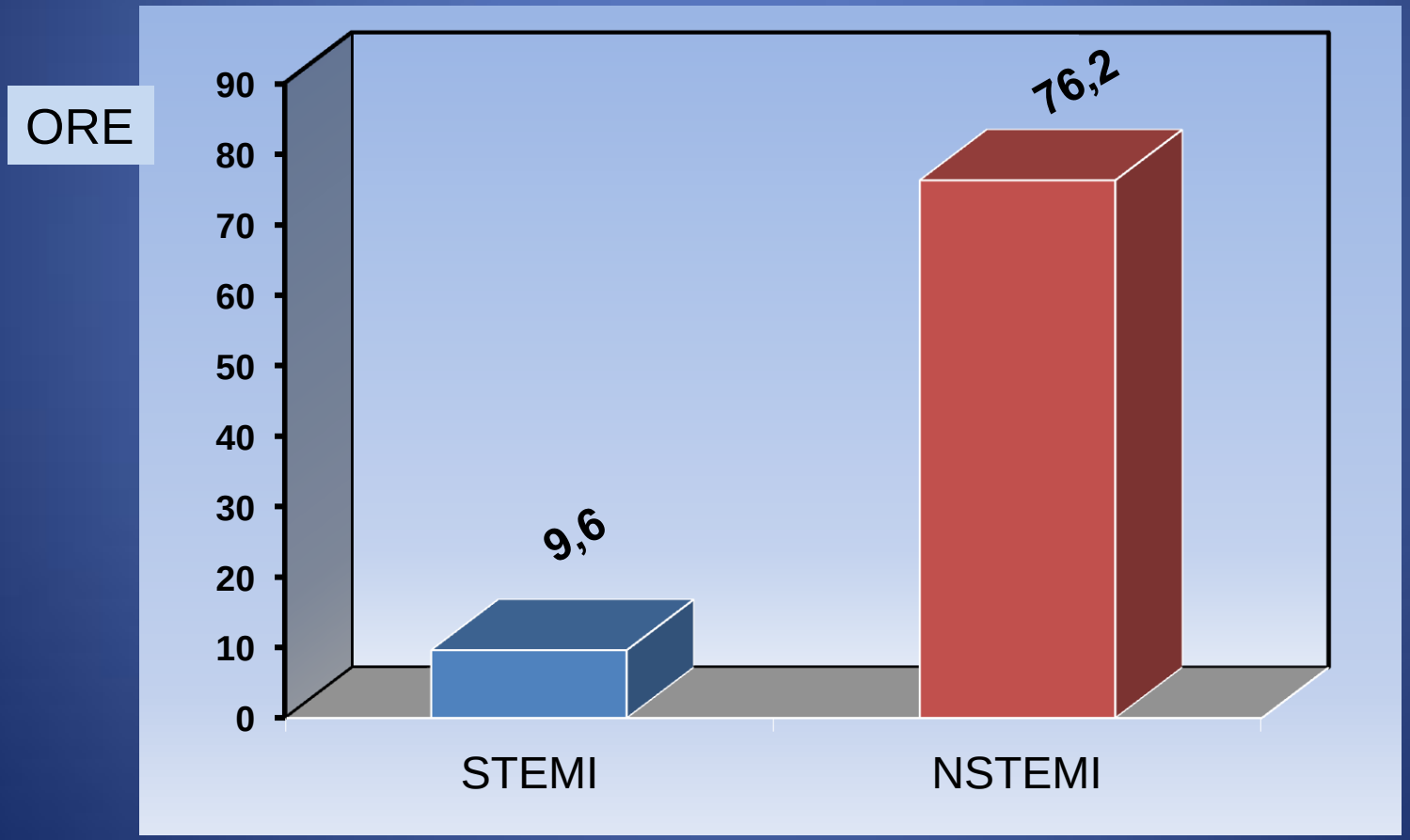
Shock e tipo di Sindrome Coronarica



Tempi di insorgenza dello shock cardiogeno



Tempi di insorgenza dello SHOCK e tipo di IMA



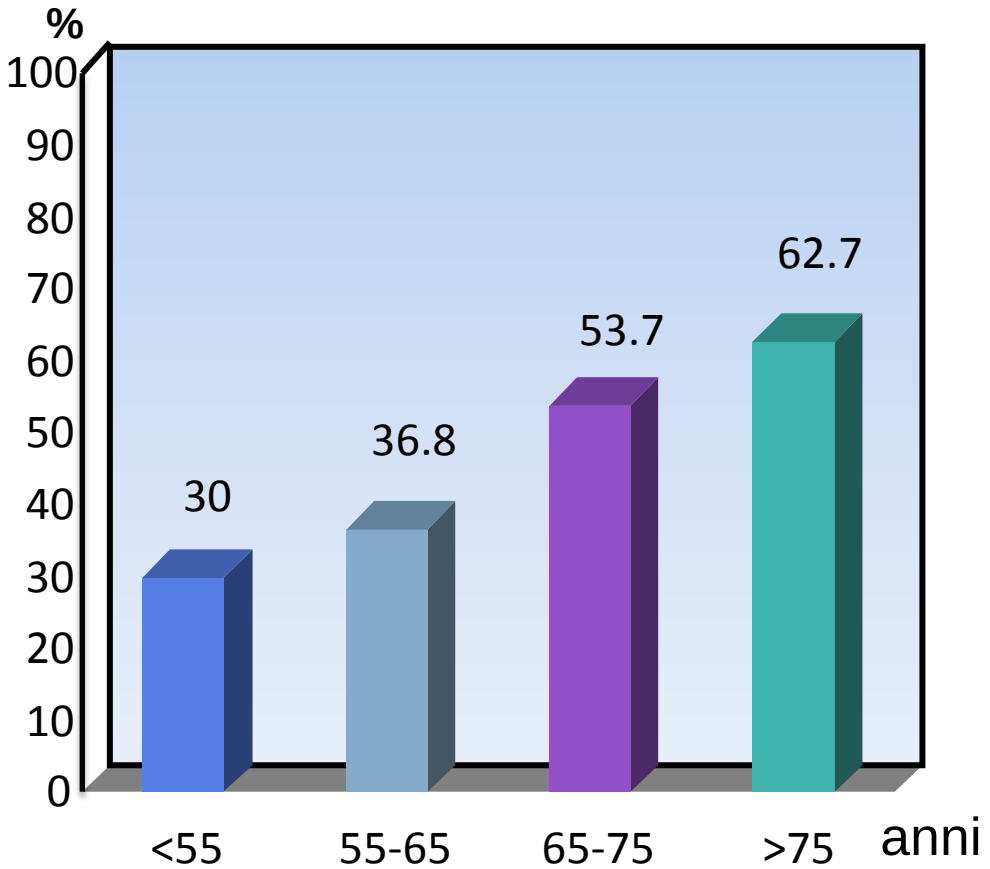
JACC Vol. 35, No. 1, 2000
January 2000:136–43

Predictors of Cardiogenic Shock After Thrombolytic Therapy for Acute Myocardial Infarction

David Hasdai, MD,* Robert M. Califf, MD, FACC,† Trevor D. Thompson, BS,†
Judith S. Hochman, MD, FACC,‡ E. Magnus Ohman, MD, FACC,† Matthias Pfisterer, MD, FACC,§
Eric R. Bates, MD, FACC,‖ Alec Vahanian, MD,¶ Paul W. Armstrong, MD, FACC,#
Douglas A. Criger, MPH,† Eric J. Topol, MD, FACC,** David R. Holmes, Jr., MD, FACC††
Petah Tikva, Israel; Durham, North Carolina; New York, New York; Basel, Switzerland; Ann Arbor, Michigan; Paris, France; Edmonton, Alberta, Canada; Cleveland, Ohio; and Rochester, Minnesota.

Table 2. Baseline Independent Predictors of Developing Cardiogenic Shock

Characteristic	Wald χ^2	*df*	p-Value	Hazard Ratio	95% CI
Age	285.14	1	< 0.001	1.47*	(1.40, 1.53)
Systolic BP	279.55	2	< 0.001		
Heart rate	225.28	3	< 0.001		
Killip class	161.35	2	< 0.001		
II vs. I				1.70	(1.52, 1.90)
III vs. I				2.95	(2.39, 3.63)
MI location	77.05	2	< 0.001		
Anterior vs. other				1.62	(1.21, 2.15)
Inferior vs. other				1.07	(0.80, 1.43)
U.S.	43.92	1	< 0.001	1.39	(1.26, 1.53)
Treatment	36.87	3	< 0.001		
SK-IV vs. tPA				1.39	(1.22, 1.59)
Combo vs. tPA				1.22	(1.07, 1.40)
SK-SQ vs. tPA				1.46	(1.28, 1.66)
Previous MI	25.61	1	< 0.001	1.34	(1.20, 1.50)
Previous CABG	15.38	1	< 0.001	1.46	(1.21, 1.76)
Weight	13.65	1	< 0.001	0.94*	(0.91, 0.97)
Female	12.69	1	< 0.001	1.22	(1.09, 1.35)
Hypertension	8.42	1	0.004	1.15	(1.05, 1.26)
Previous PTCA	7.31	1	0.007	0.70	(0.54, 0.91)
Diastolic BP	5.73	2	0.017	1.06*	(1.01, 1.11)



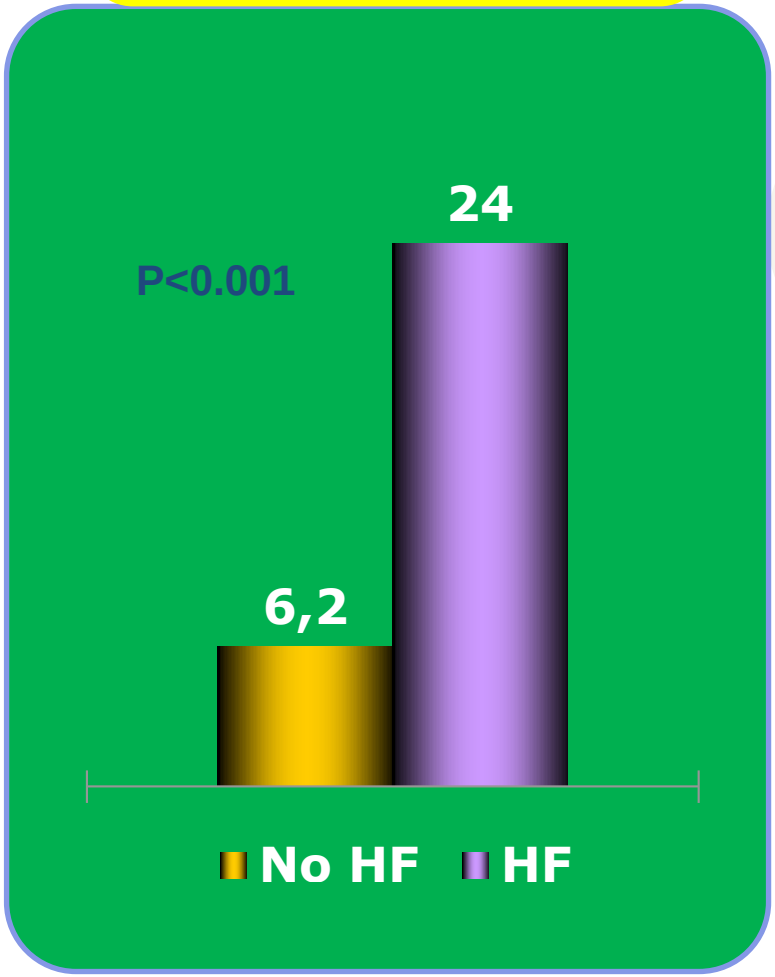
Heterogeneity in the Management and Outcomes of Patients With Acute Myocardial Infarction Complicated by Heart Failure

The National Registry of Myocardial Infarction

Frederick A. Spencer, MD; Theo E. Meyer, MD, PhD; Joel M. Gore, MD; Robert J. Goldberg, PhD

(*Circulation*. 2002;105:2605-2610.)

Mortalità ospedaliera



- 606.500 pazienti con STEMI (1994-2000)
- 20.4% HF alla presentazione
- 8.6% HF dopo la presentazione

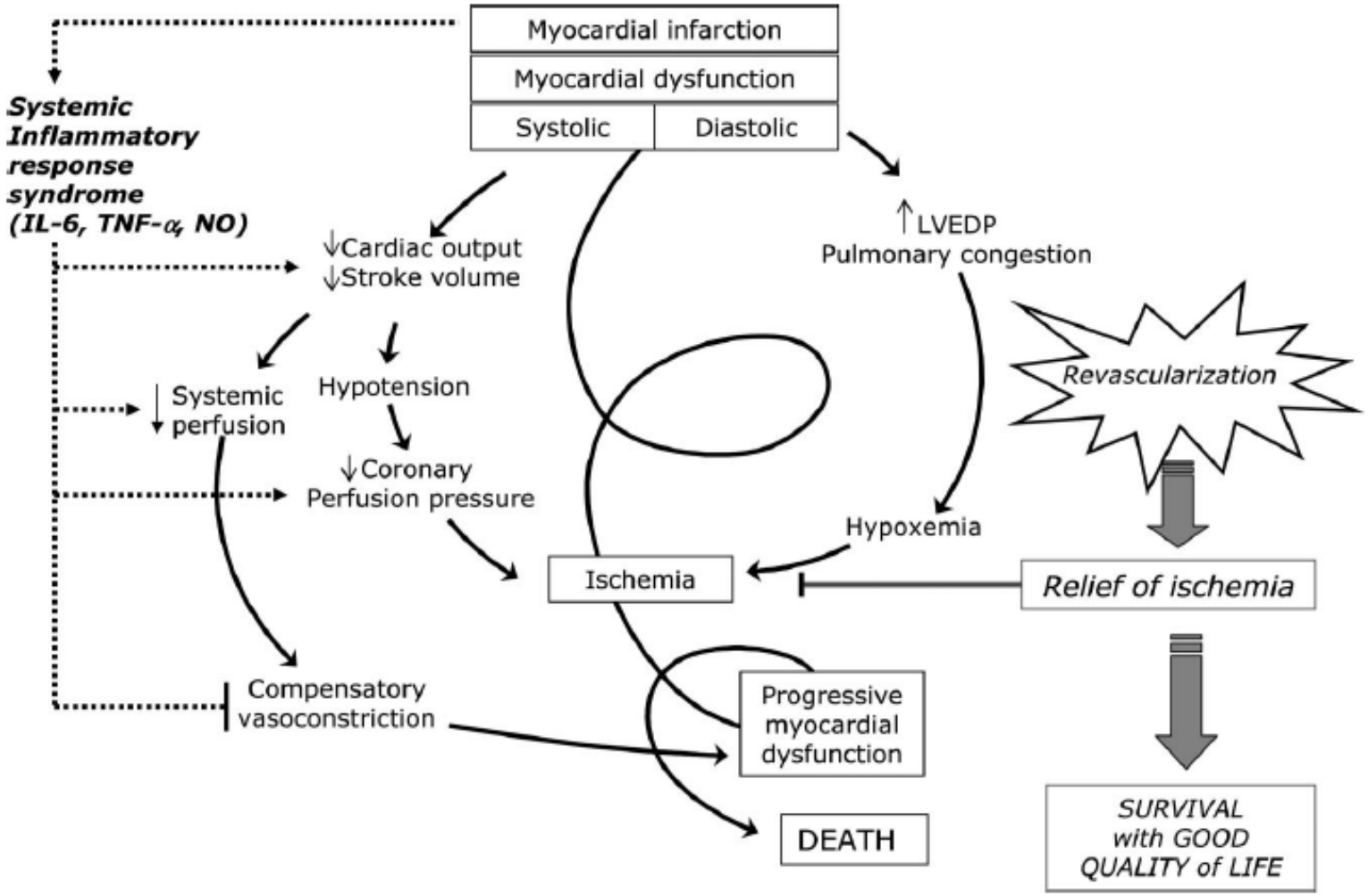
Contemporary Reviews in Cardiovascular Medicine

Cardiogenic Shock

Current Concepts and Improving Outcomes

Harmony R. Reynolds, MD; Judith S. Hochman, MD

(*Circulation*. 2008;117:686-697.)



DIAGNOSI

CONFERMARE

Disfunzione Miocardica

ESCLUDERE

- *Ipovolemia Acuta*
- *Rottura aneurisma aortico*
- *Dissezione aortica*
- *Stato settico*
- *Embolia polmonare*
- *Tamponamento cardiaco*

Executive summary of the guidelines on the diagnosis and treatment of acute heart failure

The Task Force on Acute Heart Failure of the European Society of Cardiology

Endorsed by the European Society of Intensive Care Medicine (ESICM)

Table 2 Terminology and common clinical and haemodynamic characteristics

Clinical status	Heart rate	SBP mmHg	CI L/min/m^2	PCWP mmHg	Congestion Killip/ Forrester	Diuresis	Hypoperfusion	End organ hypoperfusion
I Acute decompensated congestive heart failure	+/ −	Low normal/ High	Low normal/ High	Mild elevation	K II/F II	+	+/ −	−
II Acute heart failure with hypertension/hypertensive crisis	Usually increased	High	+/ −	>18	K II-IV/FII-III	+/ −	+/ −	+, with CNS symptoms
III Acute heart failure with pulmonary oedema	+	Low normal	Low	Elevated	KIII/FII	+	+/ −	−
IVa Cardiogenic shock*/ low output syndrome	+	Low normal	Low, <2.2	>16	K III-IV/F I-III	low	+	+
IVb Severe cardiogenic shock	>90	<90	<1.8	>18	K IV/F IV	Very low	++	+
V High output failure	+	+/ −	+	+/ −	KII/FI-II	+	−	−
VI Right sided acute heart failure	Usually low	Low	Low	Low	F I	+/ −	+/−, acute onset	+/ −

There are exceptions; the above values in table II are general rules.
*The differentation from low cardiac output syndrome is subjective and the clinical presentation may overlap these classifications.
SBP = systolic blood pressure; CI = cardiac index; PCWP = pulmonary capillary wedge pressure; CNS = central nervous system.

European Heart Journal (2005) 26, 384–416

Practical recommendations for prehospital and early in-hospital management of patients presenting with acute heart failure syndromes

Alexandre Mebazaa, MD, PhD; Mihai Gheorghiade, MD, FACC; Ileana L. Piña, MD, FACC; Veli-Pekka Harjola, MD; Steven M. Hollenberg, MD; Ferenc Follath, MD; Andrew Rhodes, MD; Patrick Plaisance, MD; Edmond Roland, MD; Markku Nieminen, MD; Michel Komajda, MD; Alexander Parkhomenko, MD; Josep Masip, MD; Faiez Zannad, MD, PhD; Gerasimos Filippatos, MD

Table 1. Clinical scenarios in acute heart failure syndrome

Clinical Scenario	Characteristics
CS1	SBP >140 mm Hg Symptoms develop abruptly Predominantly diffuse pulmonary edema Minimal systemic edema (patient may be euvolemic or hypovolemic) Acute elevation of filling pressure often with preserved LVEF Vascular pathophysiology
CS2	SBP 100–140 mm Hg Symptoms develop gradually, together with a gradual increase in body weight Predominantly systemic edema Minimal pulmonary edema Chronic elevation of filling pressure, including increased venous pressure and elevated pulmonary arterial pressure Manifestations of organ dysfunction (renal impairment, liver dysfunction, anemia, hypoalbuminemia)
CS3	SBP <100 mm Hg Rapid or gradual onset of symptoms Predominantly signs of hypoperfusion Minimal systemic and pulmonary edema Elevation of filling pressure Two subsets: Clear hypoperfusion or cardiogenic shock No hypoperfusion/cardiogenic shock
CS4	Symptoms and signs of acute heart failure Evidence of ACS Isolated elevation of cardiac troponin is inadequate for CS4 classification
CS5	Rapid or gradual onset No pulmonary edema Right ventricular dysfunction Signs of systemic venous congestion

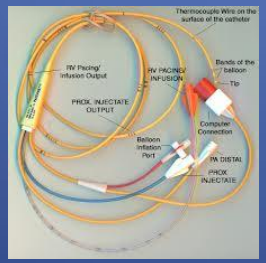
Crit Care Med 2008 Vol. 36, No. 1 (Suppl.)

CLINICA

- *Polsi periferici piccoli, iposfigmici*
- *Sensorio obnubilato*
- *Oliguria (diuresi<30 ml/h)*
- *Sudorazione fredda, pallore, cianosi delle estremità*

DATI STRUMENTALI

- *Ipotensione arteriosa (< 90 mmHg) persistente*
- *Tendenza all'acidosi metabolica*
- *Incremento dei lattati*
- *C.I. < 2,2 L/min/m2*
- *PCWP < 15 mmHg*



CCM

REVIEW ARTICLE

Martin J. London, MD
Section Editor

Journal of Cardiothoracic and Vascular Anesthesia, Vol 19, No 1 (February), 2005: pp 97-108

New Pharmacologic Approaches for the Perioperative Treatment of Ischemic Cardiogenic Shock

Andreas Lehmann, MD, and Joachim Boldt, MD

Hochman et al[2]	Goldberg et al[3]	Killip and Kimball[13]	Ducas et al[14]	Adams et al[15]
AP_{sys} <90 mmHg or support to >90 mmHg	AP_{sys} <80 mmHg absence of hypovolemia	AP_{sys} <90 mmHg	AP_{sys} <90 mmHg or support to >90 mmHg	AP_{sys} <90 mmHg
HR ≥60/min				
Oliguria <30 mL/h	Oliguria	Oliguria	Oliguria <30 mL/h	Oliguria
Cold extremities	Cold extremities	Cold extremities	Cold extremities	Cold extremities
	Cyanosis	Cyanosis	Cyanosis	Cyanosis
	Change in mental status		Change in mental status	Change in mental status
	Congestive heart failure			
CI <2.2 L/min/m²				CI <2.2 L/min/m²
PCWP >15 mmHg				PCWP >18 mmHg

NOTE. Diagnosis of cardiogenic shock according to different authors. Cardiogenic shock is primarily diagnosed by clinical signs. The leading clinical symptoms are hypotension and hypoperfusion. Only 2 authors refer to hemodynamic data as additional information for the diagnosis of cardiogenic shock.

ECOCARDIOGRAFIA

Table 1 Usefulness of echocardiography in cardiogenic shock

- Evaluate left ventricular function and myocardium at risk
- Evaluate remote myocardial segments
- Screen for ventricular septal rupture
- Screen for severe mitral regurgitation and proceed to transoesophageal echocardiography as needed
- Look for tamponade/rupture
- Assess right ventricular function
- Look for aortic dissection

PATIENT-CENTRED ACUTE CARE TRAINING PACT

ESICM EUROPEAN SOCIETY OF INTENSIVE CARE MEDICINE

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INTRODUCTION

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Haemodynamic instability, whatever the cause, is called circulatory shock.

Patients who survive the initial phase of shock may then develop the multiple organ dysfunction syndrome (MODS), which is a major cause of late death in the intensive care unit (ICU). Although the pathophysiology of MODS is multifactorial and not always precisely defined, haemodynamic instability, reduced organ perfusion and alterations in tissue microcirculation resulting in tissue hypoxia play key roles in the onset and maintenance of the syndrome.

Trasporto d'ossigeno

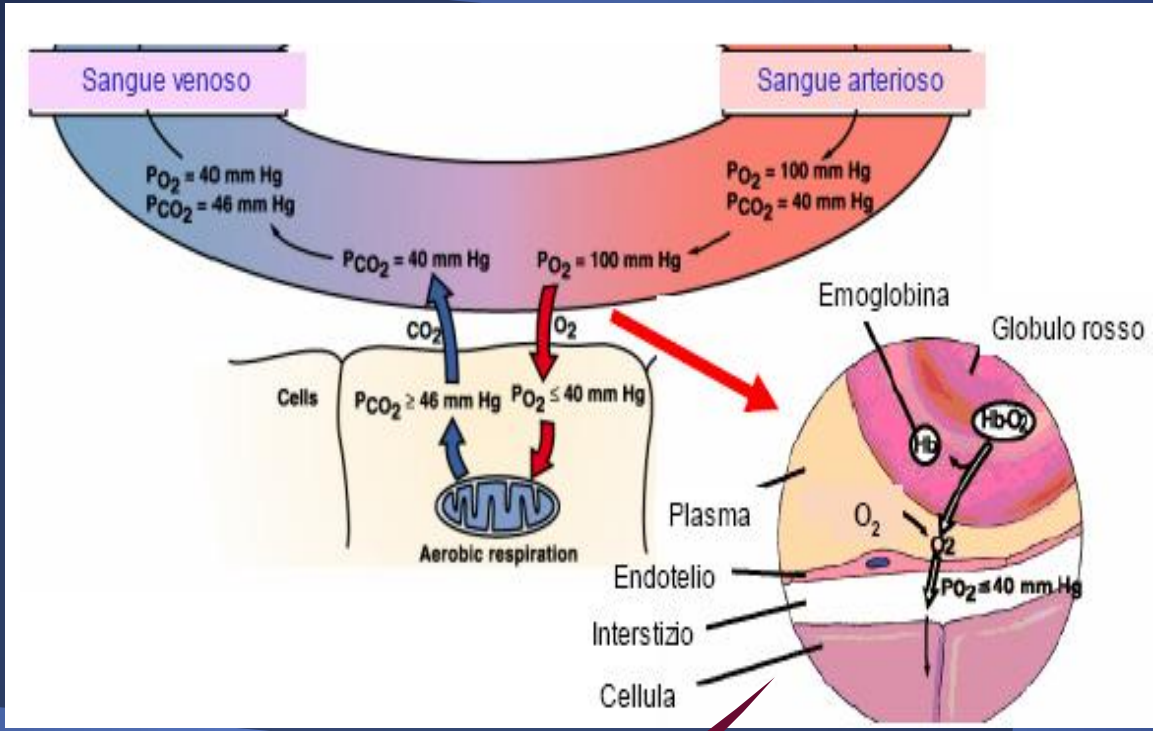
Scambi gassosi polmonari
Trasporto O2 alveolo-capillare
Distribuzione V/Q (Shunt)

Emoglobina
Concentrazione
Affinità per l'O2

Flusso ematico
Portata cardiaca e distribuzione distrettuale
Estrazione tissutale O2

Ossigenazione tissutale

Ossigenazione dei tessuti



Svo2

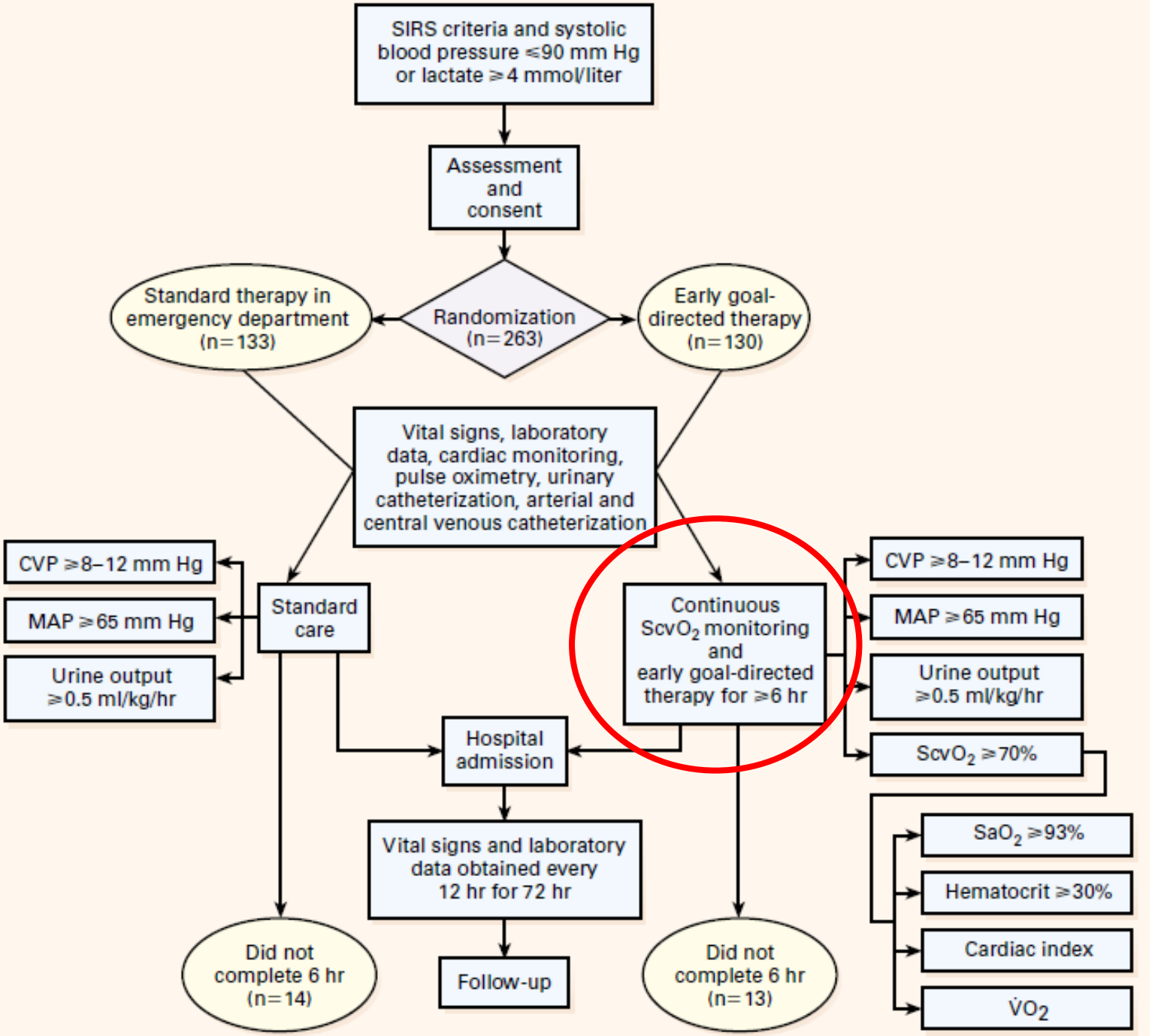
An SvO2 of less than 65% indicates an increased oxygen extraction by the cells wich is suggestive of impaired tissue perfusion or increased metabolic rate

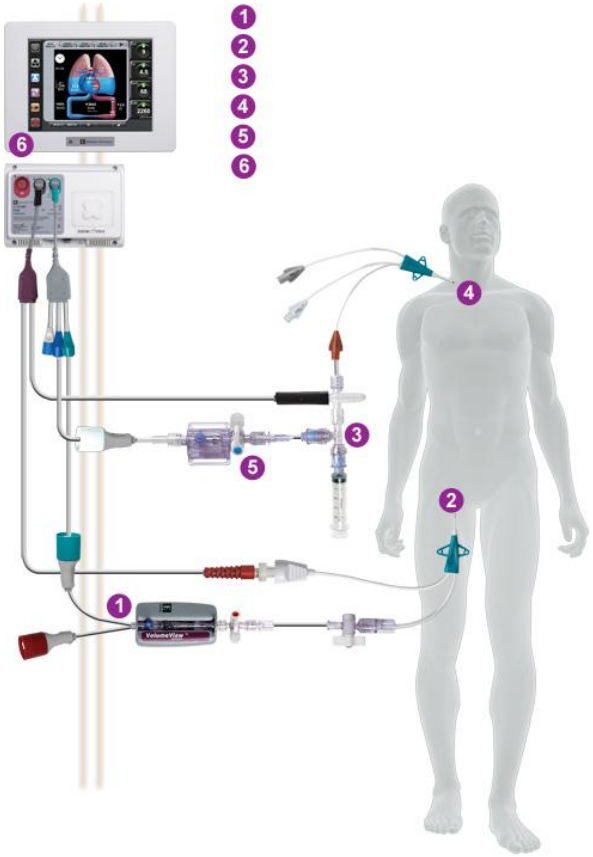
Anesthesiol Clin North America. 2001;19(4):769

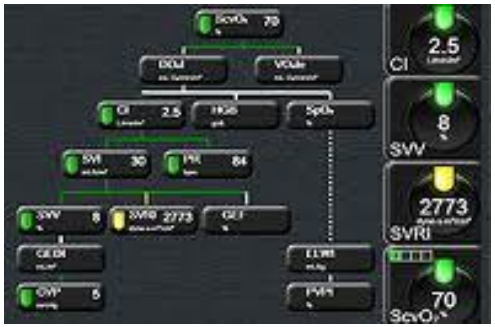
CCM

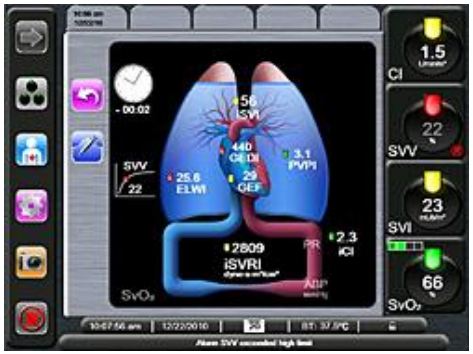
EARL

EM
ALEXANDI

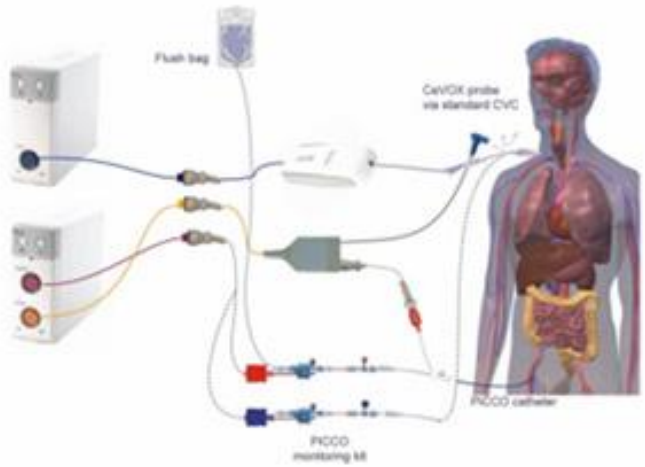


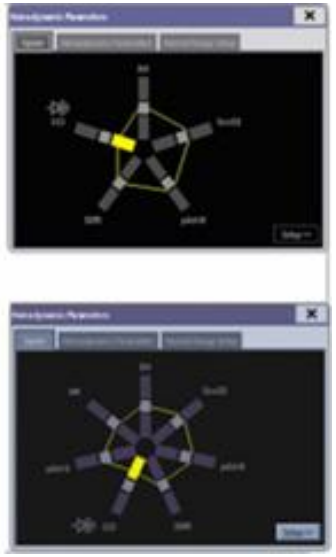

1
2
3
4
5
6


2.5
CI
8
SVV
2773
SVRI
70
ScvO2

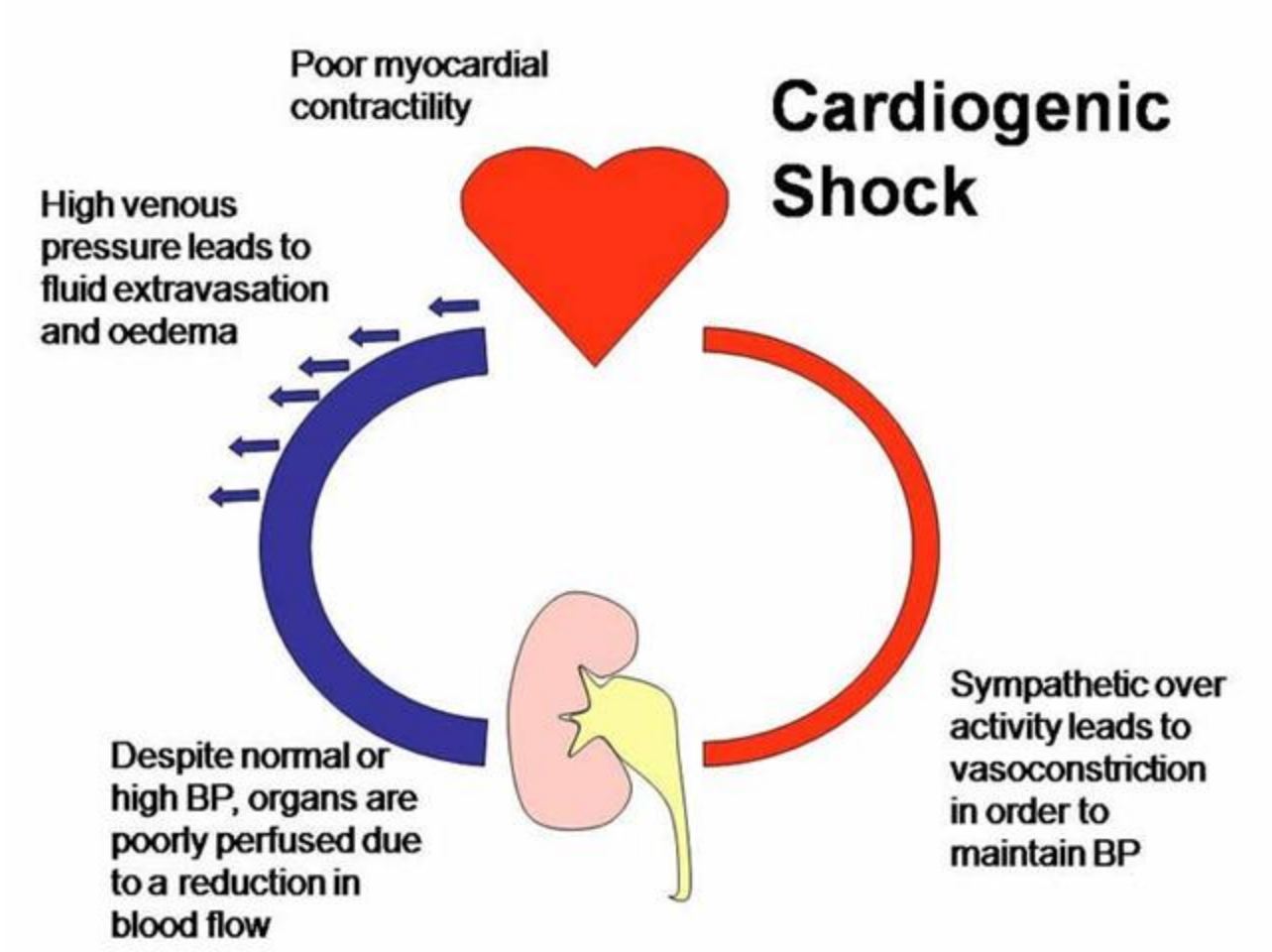

1.5
CI
22
SVV
23
SVI
66
SvO2
SVV
22
25.6
ELWI
2809
iSVRI
2.3
ICI
SvO2

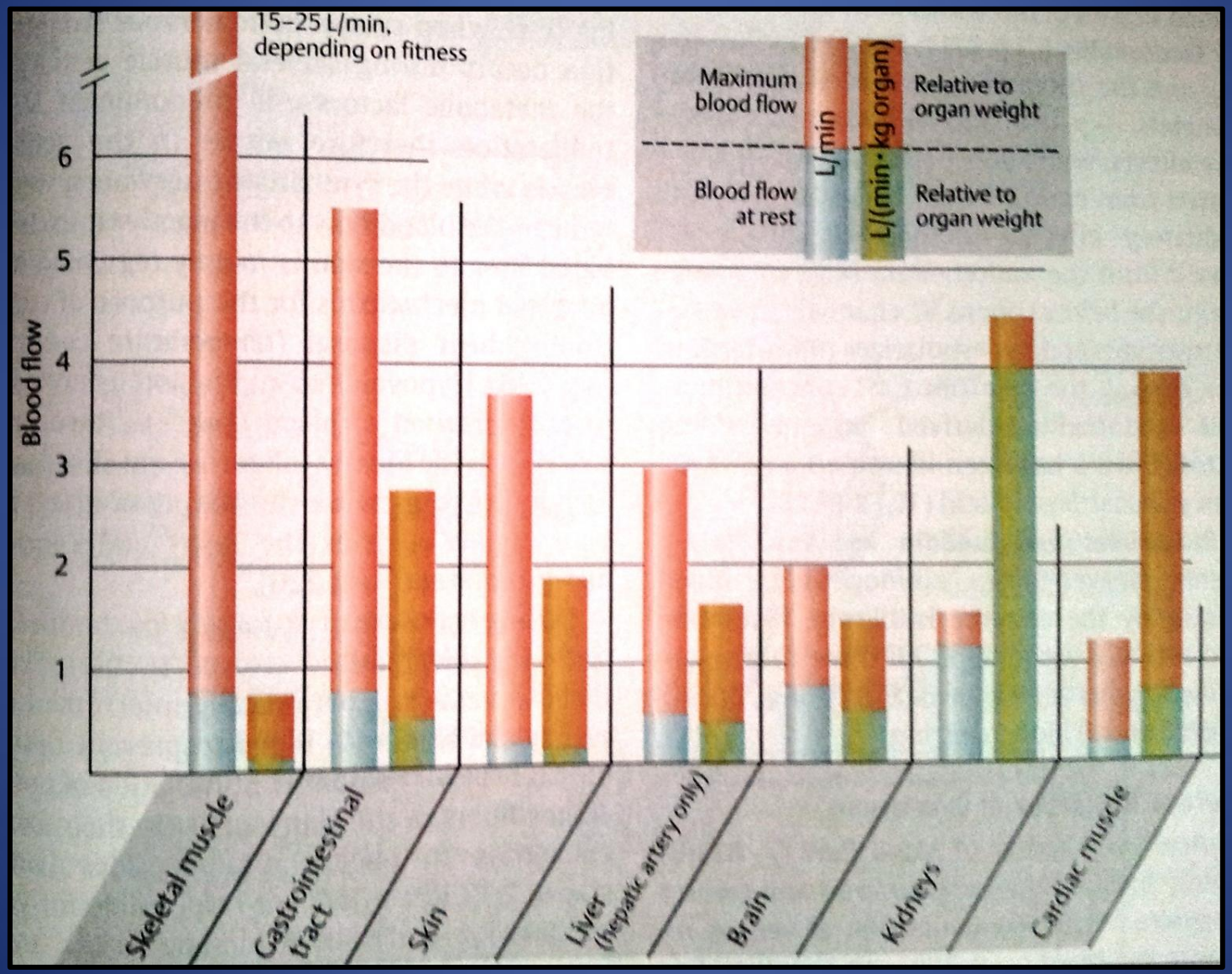

3.6
CI
11
SVV
1668
SVRI
76
ScvO2

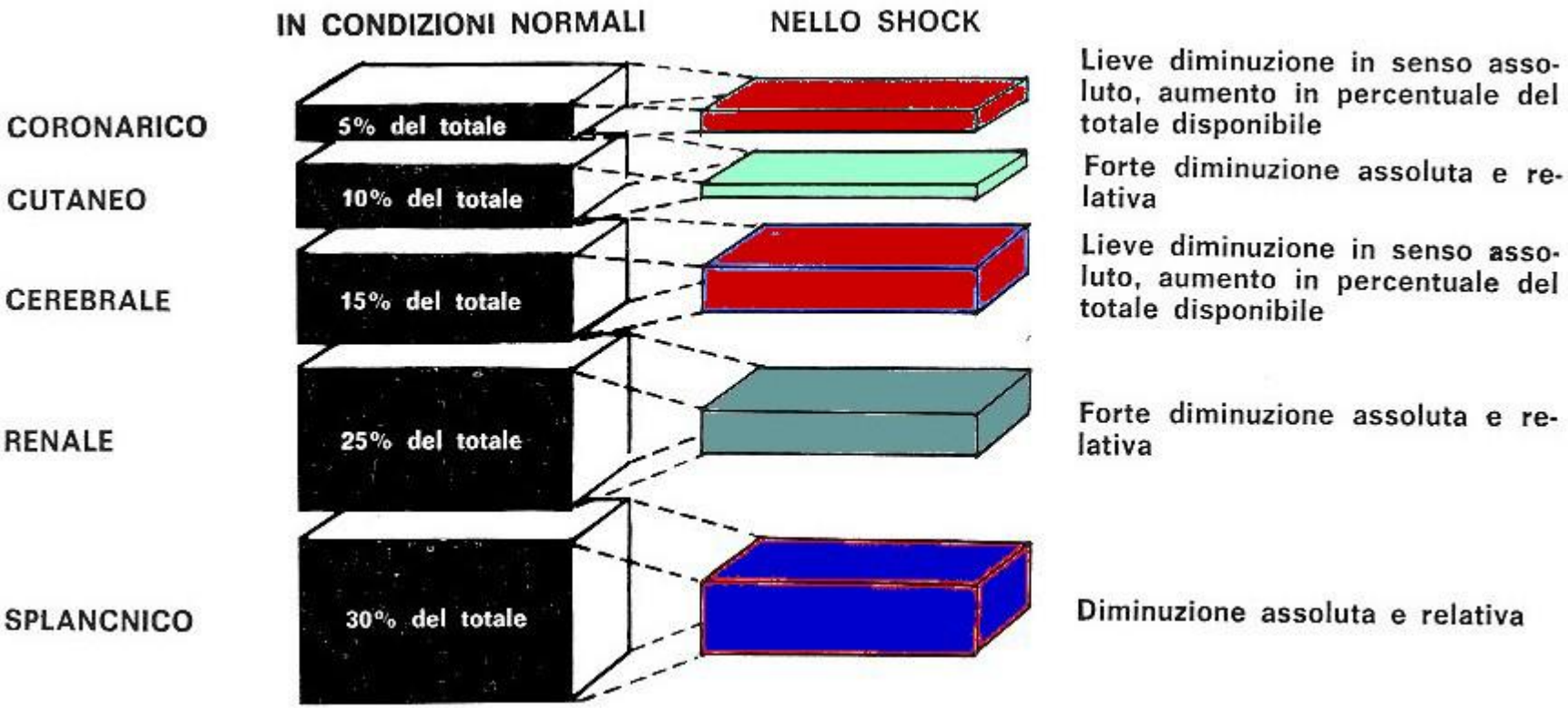

Flush bag
CalVOX probe
via standard CVC
PiCCO catheter
PiCCO
monitoring kit



Cardiogenic Shock




15–25 L/min,
depending on fitness
Maximum blood flow
Blood flow at rest
L/min
L/(min · kg organ)
Relative to organ weight
Relative to organ weight
Blood flow
6
5
4
3
2
1
Skeletal muscle
Gastrointestinal tract
Skin
Liver (hepatic artery only)
Brain
Kidneys
Cardiac muscle


IN CONDIZIONI NORMALI
NELLO SHOCK
CORONARICO
5% del totale
Lieve diminuzione in senso assoluto, aumento in percentuale del totale disponibile
CUTANEO
10% del totale
Forte diminuzione assoluta e relativa
CEREBRALE
15% del totale
Lieve diminuzione in senso assoluto, aumento in percentuale del totale disponibile
RENALE
25% del totale
Forte diminuzione assoluta e relativa
SPLANCNICO
30% del totale
Diminuzione assoluta e relativa

Mantenimento iniziale del GFR

↓

Progressiva riduzione della diuresi

↓

AKI -> Necrosi Tubulare Acuta

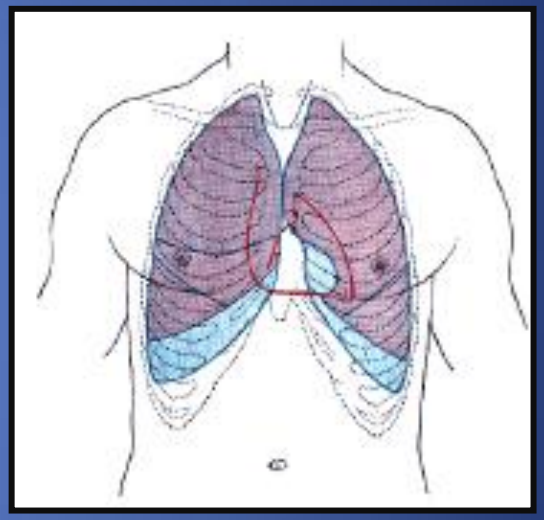
Alterazione della compliance polmonare

↓

Progressiva alterazione degli scambi gassosi

↓

ARDS



Alterazione della mucosa e passaggio in circolo di tossine

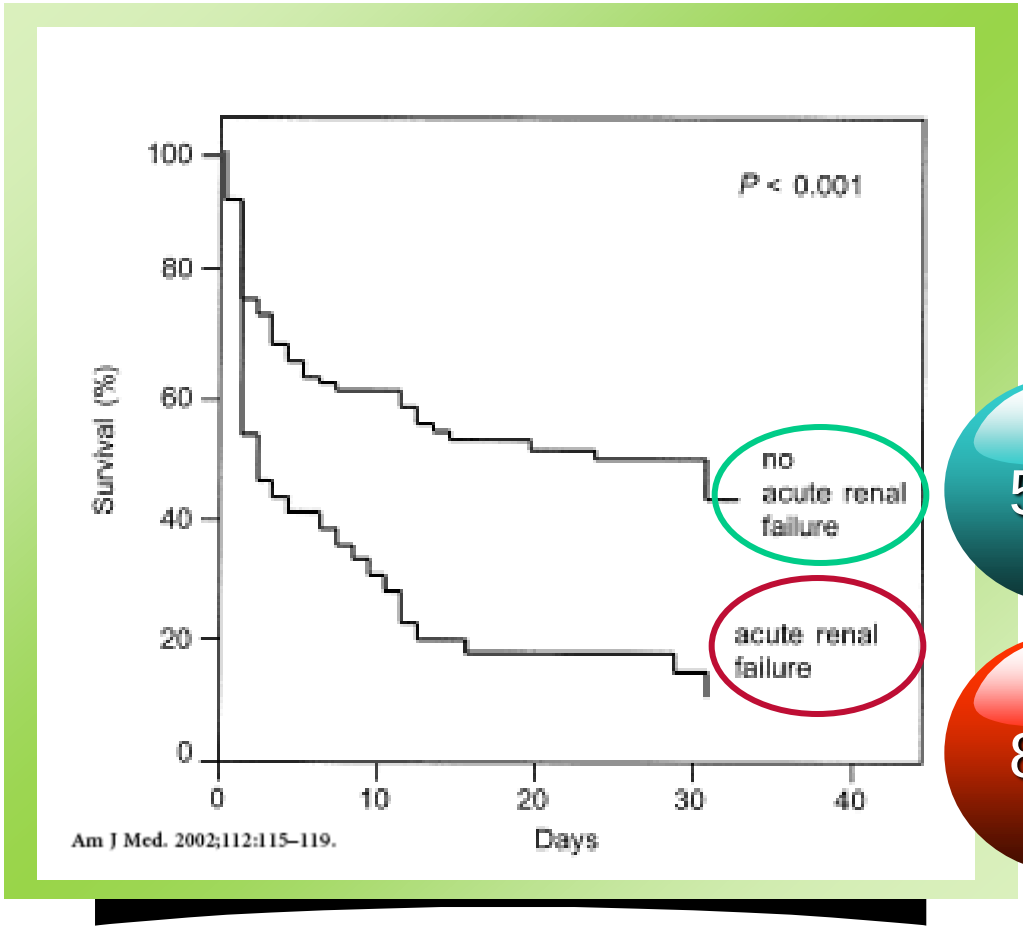
Colecistite, Pancreatite,

Ileo paralitico

Sofferenza epatica e disturbi della coagulazione

Prognosis of Patients Who Develop Acute Renal Failure during the First 24 Hours of Cardiogenic Shock after Myocardial Infarction

Maria Koreny, MD, Georg Delle Karth, MD, Alexander Geppert, MD, Thomas Neunteufl, MD, Ute Priglinger, MD, Gottfried Heinz, MD, Peter Siostrzonek, MD



Practical recommendations for prehospital and early in-hospital management of patients presenting with acute heart failure syndromes

Alexandre Mebazaa, MD, PhD; Mihai Gheorghiade, MD, FACC; Ileana L. Piña, MD, FACC; Veli-Pekka Harjola, MD; Steven M. Hollenberg, MD; Ferenc Follath, MD; Andrew Rhodes, MD; Patrick Plaisance, MD; Edmond Roland, MD; Markku Nieminen, MD; Michel Komajda, MD; Alexander Parkhomenko, MD; Josep Masip, MD; Faiez Zannad, MD, PhD; Gerasimos Filippatos, MD

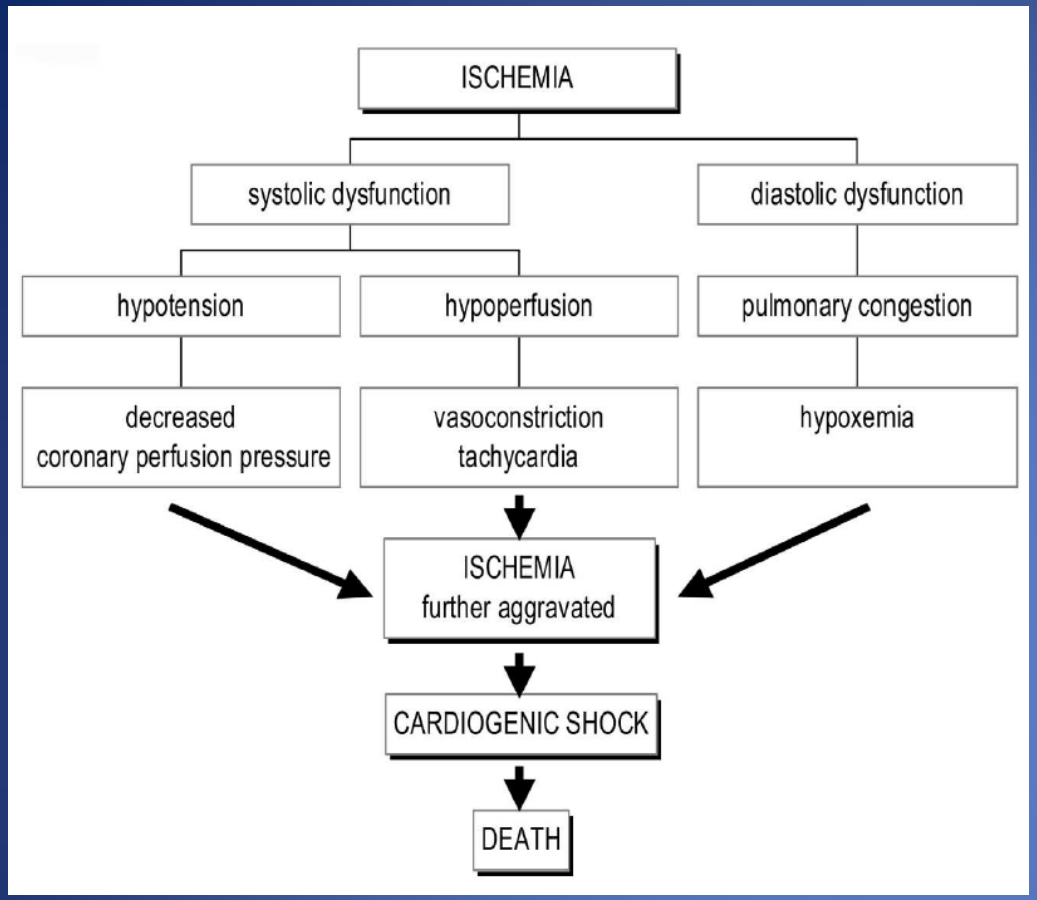
Management at admission

- Non-invasive monitoring (SaO_2, BP, temperature)
- O_2
- Non-invasive ventilation (NIV) as indicated
- Physical exam
- Lab tests
- BNP or NT-pro BNP when diagnosis is uncertain
- ECG
- Chest X-Ray

TERAPIA

Treatment Objectives

- Decrease dyspnea
- Improve well being
- Decrease heart rate
- Urine output >0.5 ml/kg/min
- Maintain/improve SBP
- Restore adequate perfusion



FARMACI

CARDIOATTIVI

MANTENIMENTO DI UN'ADEGUATA PERFUSIONE TESSUTALE

Variabilità della patologia di base

↓

Quadri clinici molto diversi

Diagnosi

- Identificare meccanismo
- Guidare la terapia

EMODINAMICA → PAC

IMAGING → TEE

IPOSISTOLIA
(deficit contrattilità)

↓

β-stimolanti

IPODIASTOLIA
(deficit rilasciamento)

↓

PDE III inibitori

CCM

QUALI FARMACI

CATECOLAMINE
- adrenalina
- dobutamina
- dopamina
- Nor-adrenalina
- Isoproterenolo
- dopexamina

INODILATATORI
- PDE-III inibitori
  - Milrinone
  - Enoximone
- Ca++-sensitizer
  - Levosimendan

QUALI FARMACI

Drug	CO	dp/dt	HR	SVR	PVR	PCWP	MVO_2
Dobutamine 2-12 μg/kg/min	⇑⇑⇑	⇑	⇑⇑	⇓	⇓	⇓ or ⇔	⇑
Dopamine							
0-3 μg/kg/min	⇑	⇑	⇑	⇓	⇓	⇑	⇑
3-8 μg/kg/min	⇑⇑	⇑	⇑	⇓	⇓	⇑	⇑
>8 μg/kg/min	⇑⇑	⇑	⇑ (⇓)	⇑	⇔ (⇑)	⇑ or ⇔	⇑⇑
Dopexamine 0.5-6 μg/kg/min	⇑⇑	⇑	⇑	⇓⇓	⇓	⇓	(⇑)
Epinephrine 0.01-0.4 μg/kg/min	⇑⇑	⇑	⇑⇑	⇑ (⇓)	(⇑)	⇑ or ⇔	⇑⇑
Norepinephrine 0.01-0.3 μg/kg/min	⇑	⇑	⇔ (⇑⇓)	⇑⇑	⇔	⇔	⇑
PDE inhibitors	⇑⇑	⇑	⇑	⇓⇓	⇓	⇓⇓	⇓

NOTE. PDE inhibitors are usually given as a loading dose followed by a continuous infusion: Amrinone: 0.5-1.5 mg/kg loading dose, 10-30 μg/kg/min continuous infusion; Milrinone: 50 μg/kg loading dose, 0.375-0.75 μg/kg/min continuous infusion; Enoximone: 0.5-1.0 mg/kg loading dose, 2.5-20 μg/kg/min continuous infusion.

The indicated doses represent the most common dose ranges. For the individual patient, a deviation from these recommended dose might be indicated.

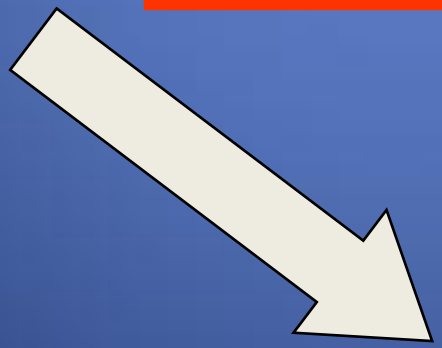
Abbreviations: CO, cardiac output; dp/dt, myocardial contractility; HR, heart rate; SVR, systemic vascular resistance; PVR, pulmonary vascular resistance; PCWP, pulmonary capillary wedge pressure; MVO_2, myocardial oxygen consumption; PDE inhibitors, phosphodiesterase inhibitors.

STRATEGIE TERAPEUTICHE

FARMACI VASODILATATORI

FARMACI INOTROPI

IMPIEGO DI AMINE AD ALTO DOSAGGIO:
- aumento della necrosi perioperatoria
- esaurimento energetico miocardico
- aritmie ventricolari e sopraventricolari
- riduzione funzione renale
- prolungata ventilazione artificiale
- maggiore permanenza in TI



SUPPORTO MECCANICO
IABP → VAD → TAH

Journal of Cardiothoracic and Vascular Anesthesia, Vol 18, No 4 (August), 2004: pp 401-403

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GOLD STANDARD

ADRENALINA

0.01- 0.05 γ/Kg/min

PDE-III i

Enoximone 3 – 5 γ/Kg/min

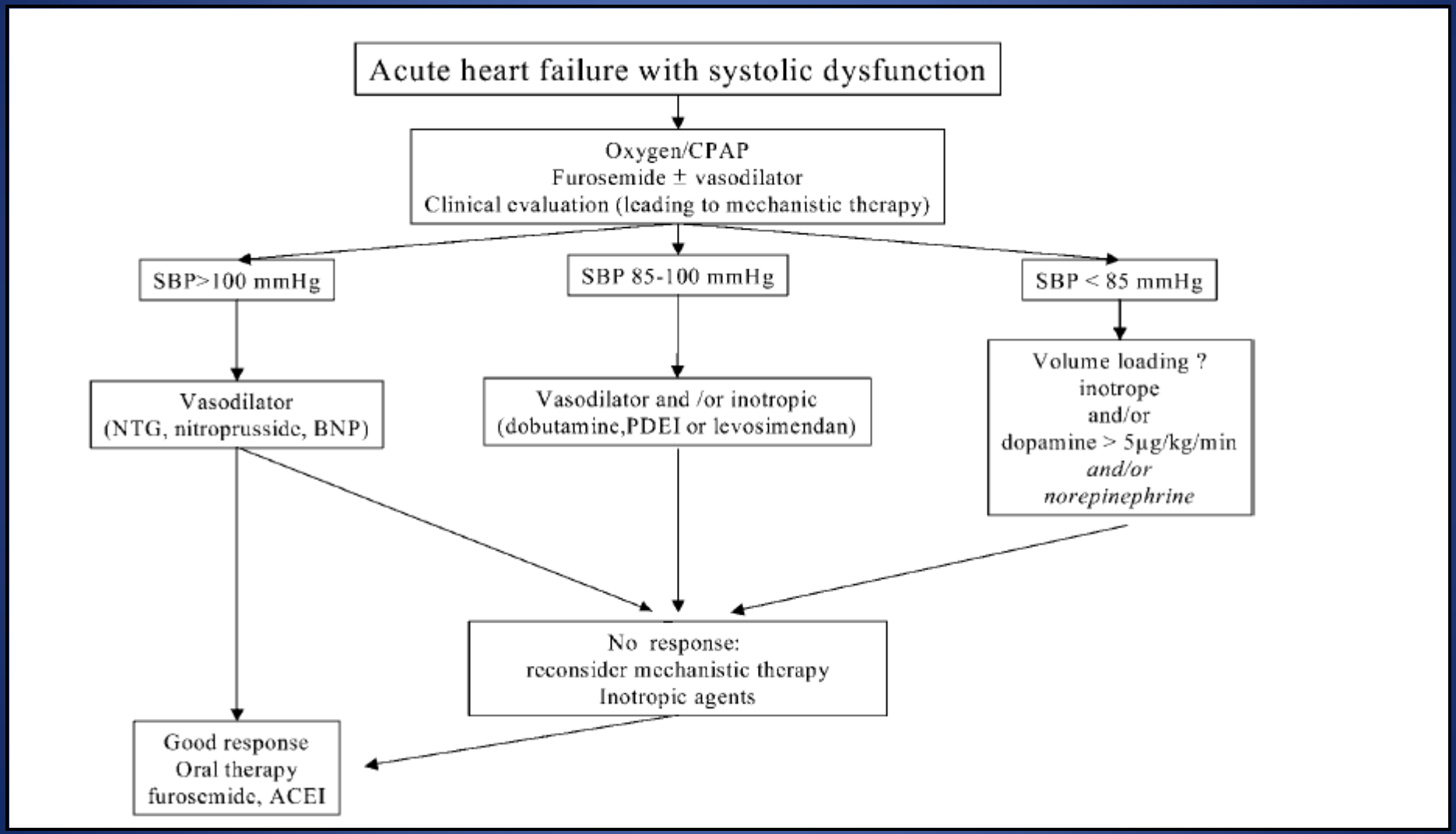
↑↑ cAMP

- **effetto inotropo**
- **vasodilazione**
- **rilasciamento diastolico**

EFFETTO SINERGICO

- **FE < 35%**
- **IC < 2.0 l/min/m2**
- **PCWP > 15 mmHg**
- **scompenso Vdx**
- **ipertensione polm.**

Algoritmo terapeutico per l'impiego degli inotropi nello scompenso cardiaco acuto



European Heart Journal (2005) **26**, 384–416

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Executive summary of the guidelines on the diagnosis and treatment of acute heart failure

The Task Force on Acute Heart Failure of the European Society of Cardiology

Endorsed by the European Society of Intensive Care Medicine (ESICM)

European Heart Journal (2005) **26**, 384–416

Inotropic agents are indicated in the *presence of peripheral hypoperfusion* (hypotension, decreased renal function) with or without congestion or *pulmonary oedema* refractory to diuretics and vasodilators at optimal doses. **(Class IIa, level of evidence C)**

Their use *is potentially harmful* as they increase oxygen demand and calcium loading and they should be used with caution.

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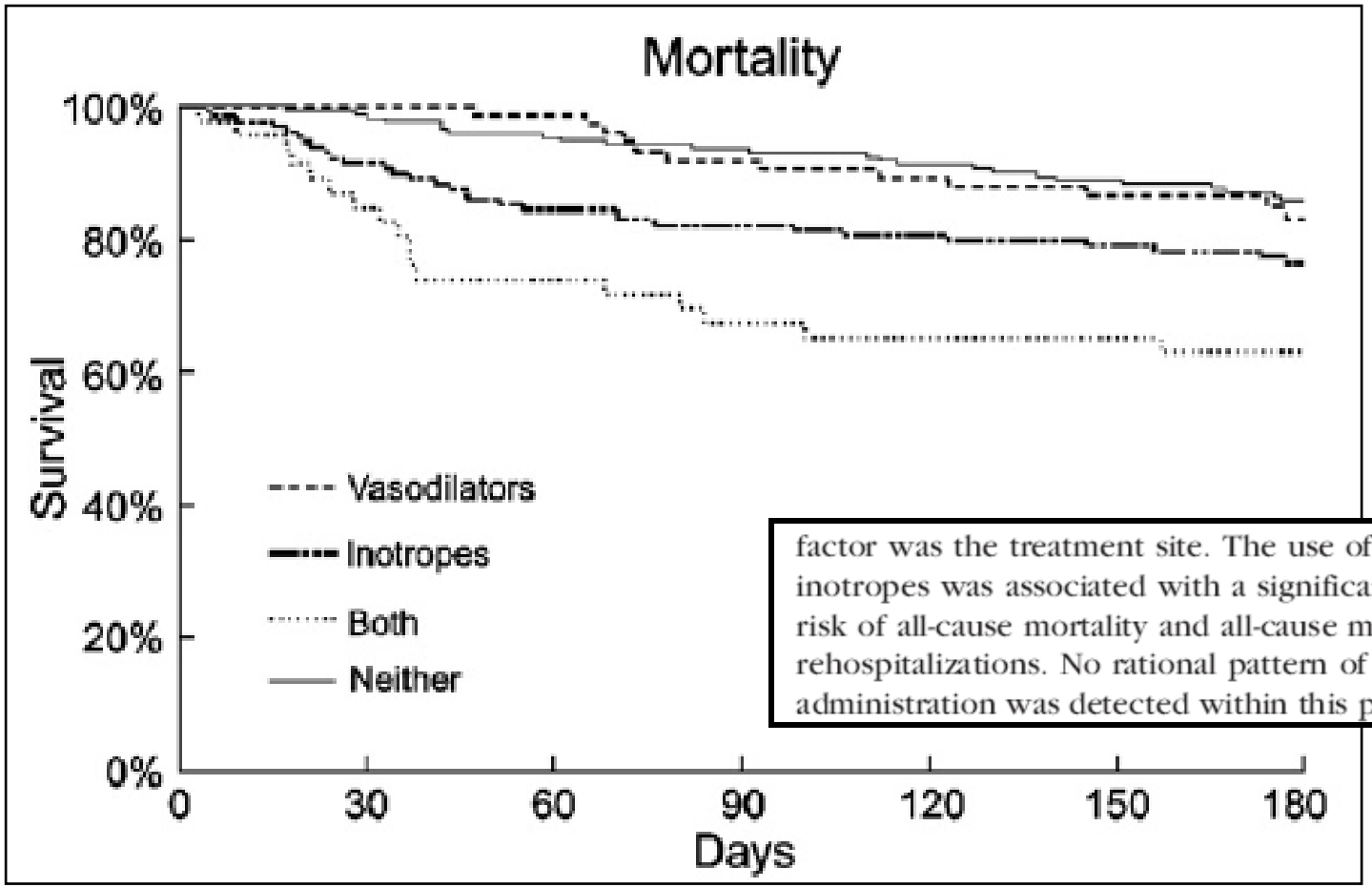
Use and impact of inotropes and vasodilator therapy in hospitalized patients with severe heart failure

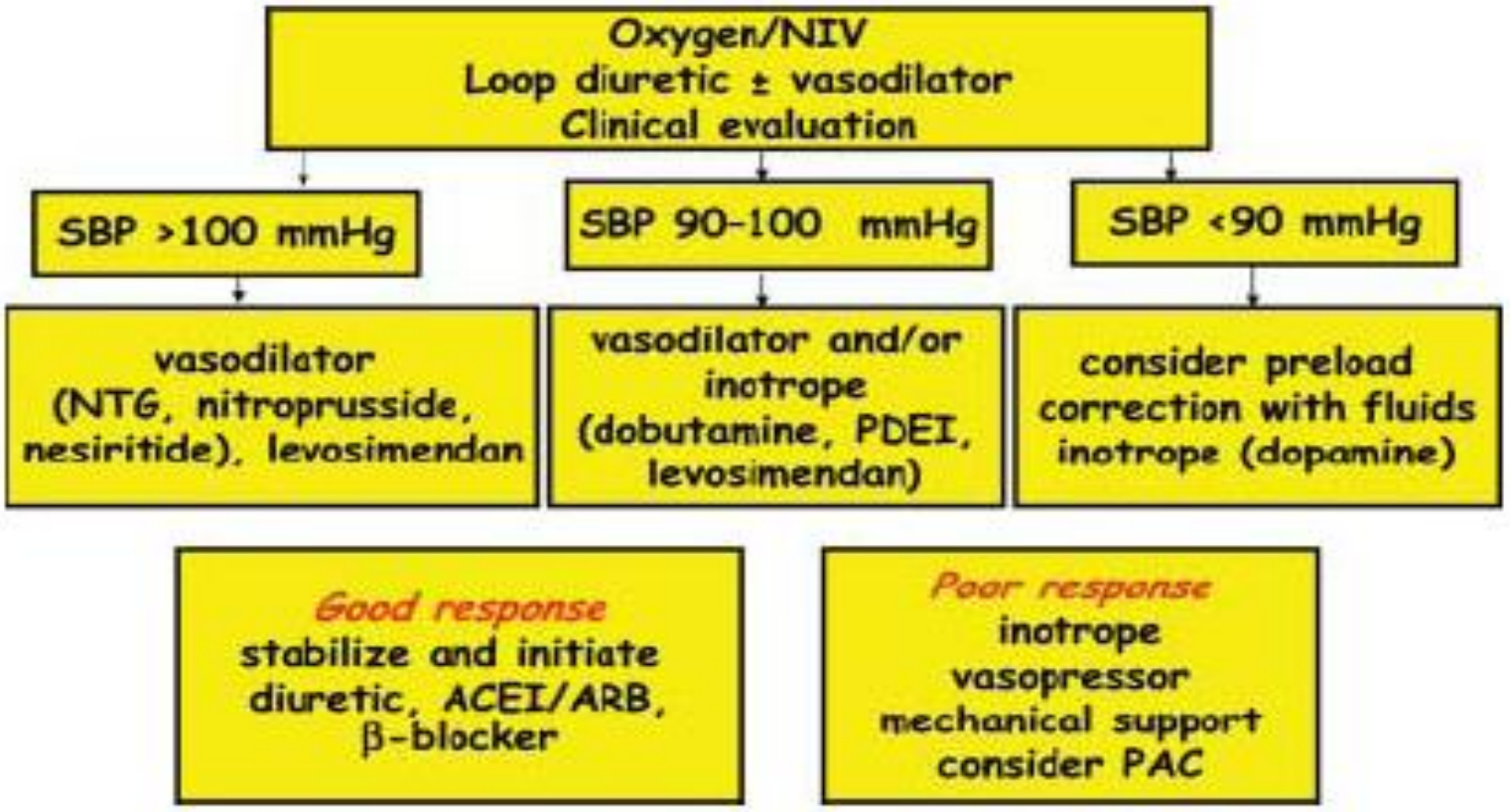
Uri Elkayam, MD,[a] Gudaye Tasissa, PhD,[b] Cynthia Binanay, RN, BSN,[b] Lynne W. Stevenson, MD,[c] Mihai Gheorghiade, MD,[d] J. Wayne Warnica, MD,[e] James B. Young, MD,[f] Barry K. Rayburn, MD,[g] Joseph G. Rogers, MD,[b] Teresa DeMarco, MD,[h] and Carl V. Leier, MD[i] *Los Angeles and San Francisco, CA; Durham, NC; Boston, MA; Chicago, IL; Calgary, Alberta, Canada; Cleveland and Columbus, OH; and Birmingham, AL*

(Am Heart J 2007;153:98-104.)



factor was the treatment site. The use of intravenous inotropes was associated with a significantly increased risk of all-cause mortality and all-cause mortality plus rehospitalizations. No rational pattern of inotrope administration was detected within this population. The

European Journal of Heart Failure (2008), 933–989
doi:10.1016/j.ejheart.2008.08.005



ACC/AHA Practice Guidelines

ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction—Executive Summary

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction)

2. Hypotension

Class I

1. Rapid volume loading with an IV infusion should be administered to patients without clinical evidence for volume overload. (*Level of Evidence: C*)
2. Rhythm disturbances or conduction abnormalities causing hypotension should be corrected. (*Level of Evidence: C*)
3. Intra-aortic balloon counterpulsation should be performed in patients who do not respond to other interventions, unless further support is futile because of the patient's wishes or contraindications/unsuitability for further invasive care. (*Level of Evidence: B*)
4. Vasopressor support should be given for hypotension that does not resolve after volume loading. (*Level of Evidence: C*)
5. Echocardiography should be used to evaluate mechanical complications unless these are assessed by invasive measures. (*Level of Evidence: C*)

3. Low-Output State

Class I

1. LV function and potential presence of a mechanical complication should be assessed by echocardiography if these have not been evaluated by invasive measures. (*Level of Evidence: C*)
2. Recommended treatments for low-output states include:
   a. Inotropic support. (*Level of Evidence: B*)
   b. Intra-aortic counterpulsation. (*Level of Evidence: B*)
   c. Mechanical reperfusion with PCI or CABG. (*Level of Evidence: B*)
   d. Surgical correction of mechanical complications. (*Level of Evidence: B*)

Limit the use of catecholamines to:

LOS, caused by systolic disfunction, volume refractory, documented by TEE or CI < 2.0 L min^{-1} m^2 and with organ hypoperfusion (oliguria and hyperlactacidemia).

Vasodilatatory shock, volume refractory, documented by low SVR values and with organ hypoperfusion

Short duration and replaced on time by IABP/VAD if severe systolic disfunction

Intensive Care Med (2011) 37:290–301
DOI 10.1007/s00134-010-2073-4

ORIGINAL

Alexandre Mebazaa
John Parissis
Raphael Porcher
Etienne Gayat
Maria Nikolaou
Fabio Vilas Boas
J. F. Delgado
Ferenc Follath

Short-term survival by treatment among patients hospitalized with acute heart failure: the global ALARM-HF registry using propensity scoring methods

RETROSPETTIVO - MULTICENTRICO 4953 paz. in 9 paesi

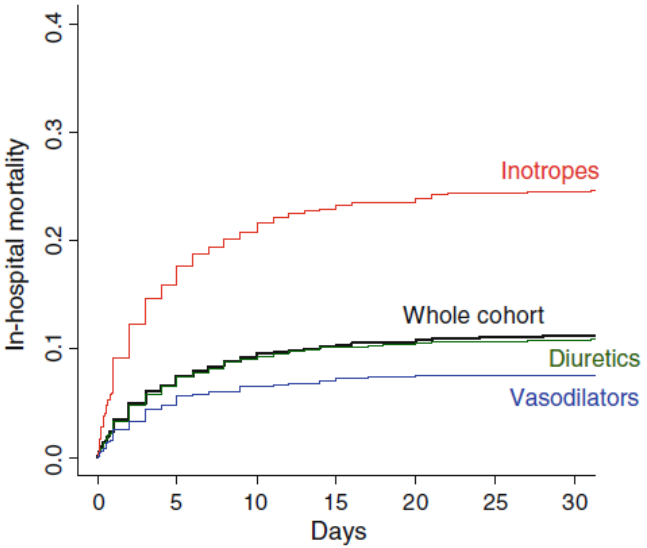


Fig. 1 Effect of the main intravenous (IV) drugs administered during first 48 h in acute heart failure (AHF) patients on in-hospital mortality. Whole cohort ($n = 4{,}953$), IV diuretics ($n = 4{,}167$), IV vasodilators (mostly nitrates, $n = 1{,}930$), IV inotropes and/or IV vasopressors ($n = 1{,}617$)

domized clinical trial. We also observed associations between four IV catecholamines, namely, epinephrine, norepinephrine, dopamine, and dobutamine, and poor in-hospital outcome in this large multinational AHF cohort. The use of levosimendan did not incur a higher risk of in-hospital mortality. This study therefore brings to light the fact that existing guidelines need to be revisited in order to bring safety to the treatment of AH patients. Based on our results, we advocate for a wider use of agents with vasodilator properties and a very limited use of catecholamines in AHF patients.

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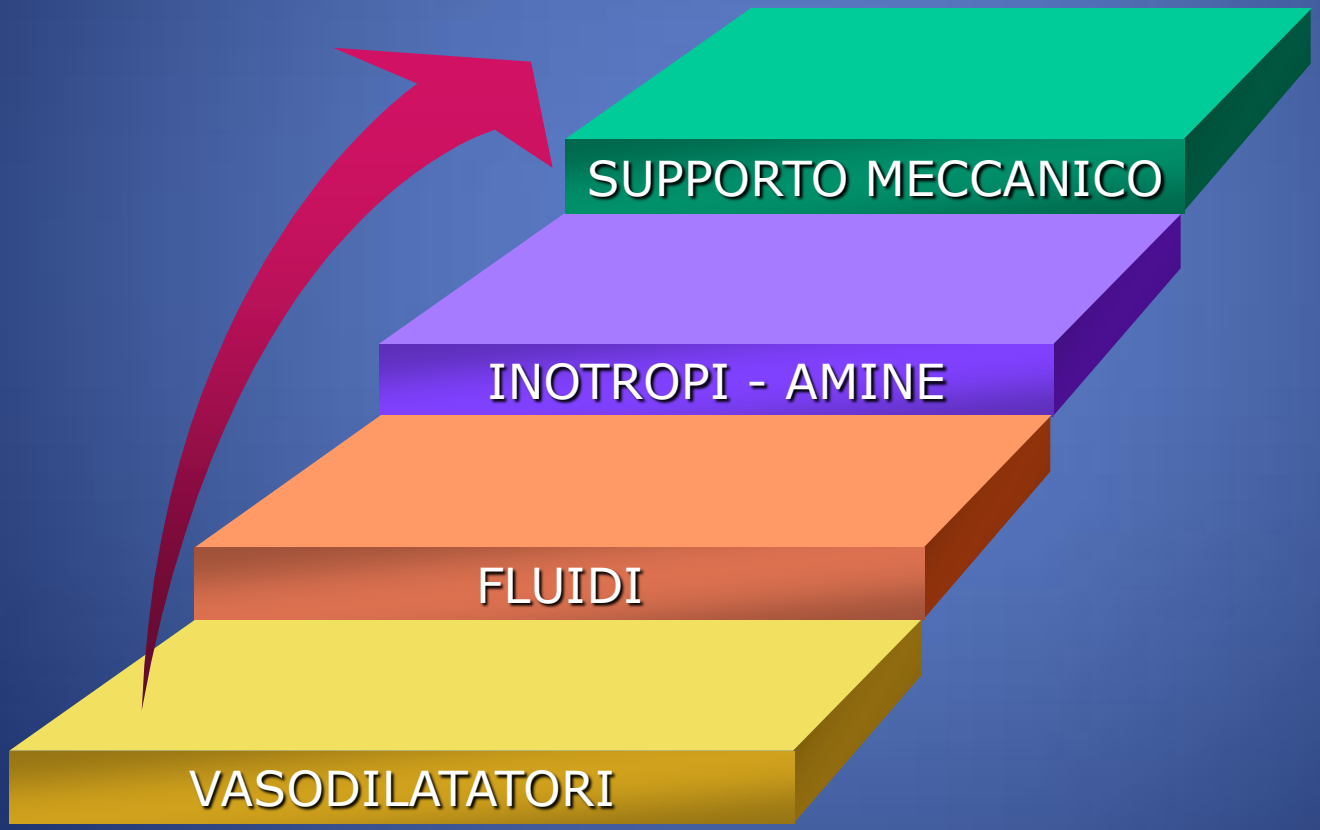
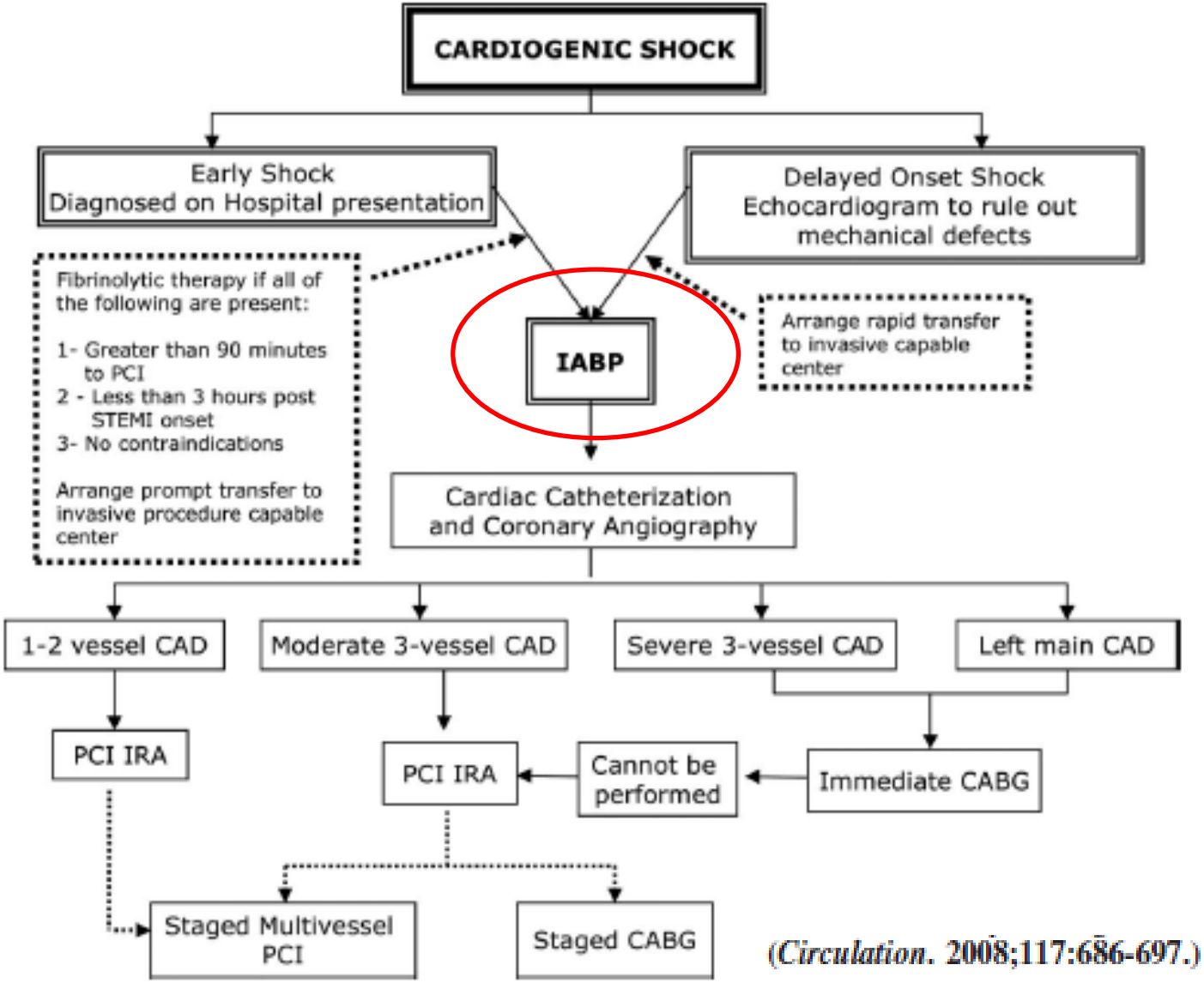
Terapia dello shock cardiogeno





(*Circulation*. 2008;117:686-697.)

PRO

- *Relativamenrte semplice, metodica di prima scelta*
- *Bassa incidenza di complicanze*
- *Efficacia per la stabilizzazione emodinamica iniziale*

CONTRO

- *Mancanza di supporto cardiaco attivo*
- *Necessità di un certo livello residuo di funzione del ventricolo sinistro*

European Heart Journal (2012) **33**, 2569–2619
doi:10.1093/eurheartj/ehs215

ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

Treatment of cardiogenic shock (Killip class IV)			
Oxygen/mechanical respiratory support is indicated according to blood gasses.	I	C	-
Urgent echocardiography/Doppler must be performed to detect mechanical complications, assess systolic function and loading conditions.	I	C	-
High-risk patients must be transferred early to tertiary centres.	I	C	-
Emergency revascularization with either PCI or CABG in suitable patients must be considered.	I	B	100
Fibrinolysis should be considered if revascularization is unavailable.	IIa	C	-
Intra-aortic balloon pumping may be considered.	IIb	B	1, 98, 305
LV assist devices may be considered for circulatory support in patients in refractory shock.	IIb	C	-
Haemodynamic assessment with balloon floating catheter may be considered.	IIb	B	316
Inotropic/vasopressor agents should be considered: • Dopamine	IIa	C	-
• Dobutamine	IIa	C	-
• Norepinephrine (preferred over dopamine when blood pressure is low).	IIb	B	300, 317

ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

European Heart Journal (2012) **33**, 2569–2619

2010	IABP insertion is recommended in patients with haemodynamic instability (particularly those in cardiogenic shock and with mechanical complications).	I	C
2012	Intra-aortic balloon pumping may be considered.	IIb	B

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

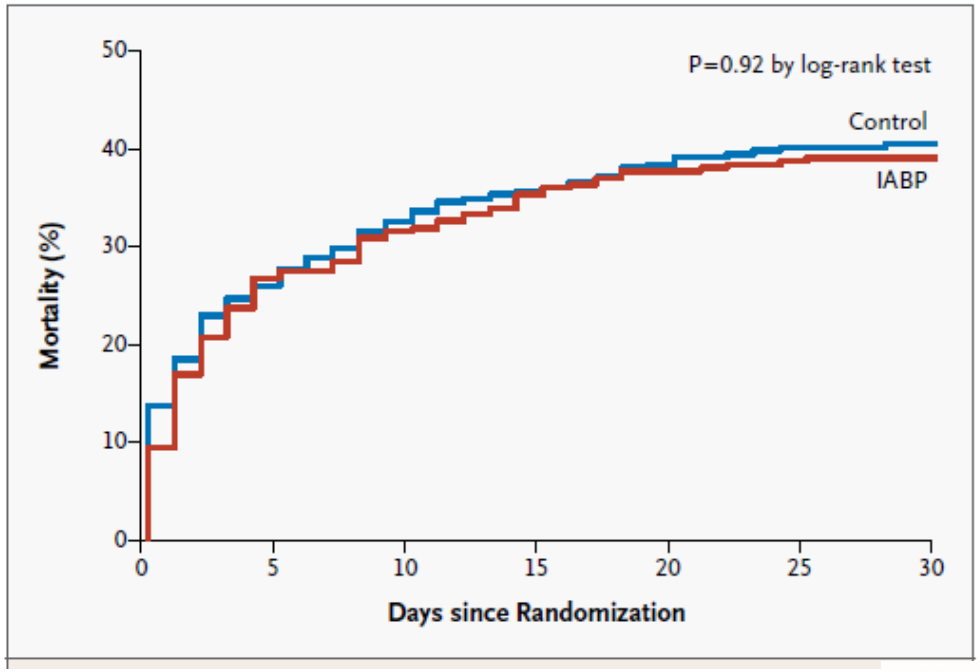
Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

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*

Table 3. Clinical Outcomes.

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)

* Bleeding during the hospital stay was assessed according to the Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO) criteria.



Alain Combes
Service de Réanimation
iCAN, Institute of Cardiometabolism and Nutrition
Hôpital Pitié-Salpêtrière, AP-HP, Paris
Université Pierre et Marie Curie, Paris 6

Therapeutic Strategy: When to initiate mechanical assistance?

- Parameters to evaluate:
  - Etiology/Time course of the disease
  - Treatments administered
  - Clinical status, in particular neurological status:
    - Is it futile to insert a device?
- Other clinical signs associated with rapid deterioration of cardiac function:
  - Nausea, abdominal pain, Alteration of consciousness
  - Tachycardia, rhythm disturbances
  - Ionic disturbances, Acidosis
  - **Hepatic / Renal failure**
- Doppler-Echocardiography +++
  - LVEF <20%
  - Signs of low cardiac output, Ao VTI <8cm

The classical indications of mechanical assistance…

- 4 types of indications:
  - « Bridge to recovery »
  - « Bridge to bridge »
  - « Bridge to transplantation »
  - « Destination therapy »

Effetti delle assistenze circolatorie

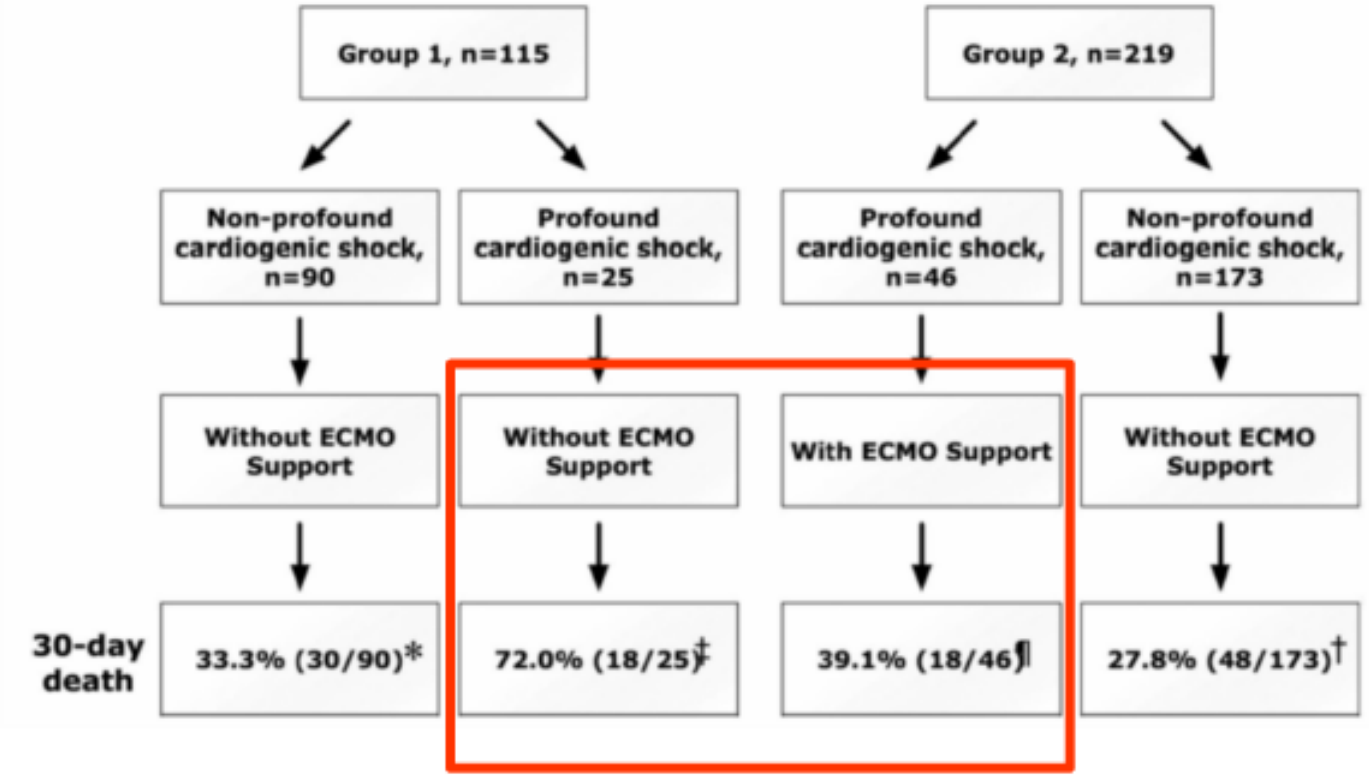
- Riduzione delle pressioni di riempimento del ventricolo sinistro
- Riduzione del lavoro cardiaco (LVWI)
- Riduzione del consumo di ossigeno del miocardio (MVO2)

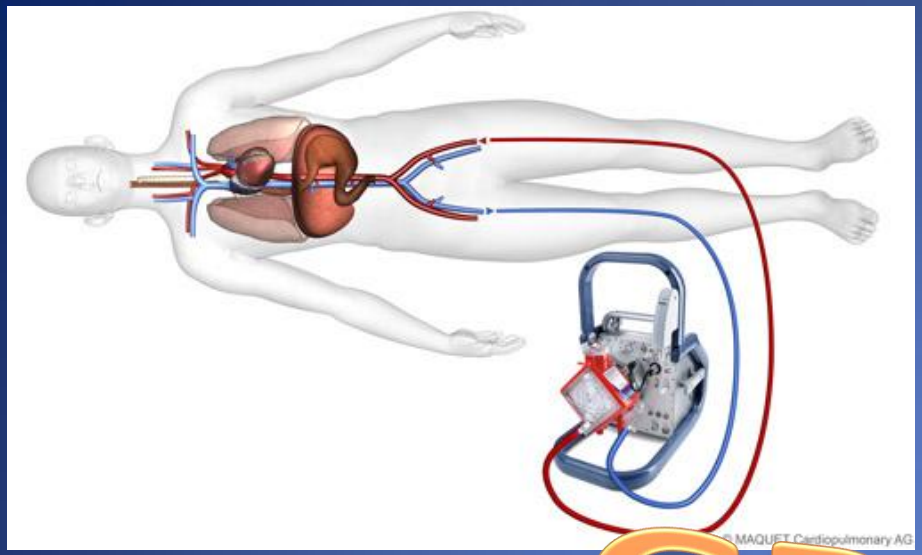
- Recupero del miocardio ischemico dopo rivascolarizzazione
- Riduzione dell'area infartuale

Early extracorporeal membrane oxygenator-assisted primary percutaneous coronary intervention improved 30-day clinical outcomes in patients with ST-segment elevation myocardial infarction complicated with profound cardiogenic shock

Jiunn-Jye Sheu, MD; Tzu-Hsien Tsai, MD; Fan-Yen Lee, MD; Hsiu-Yu Fang, MD; Cheuk-Kwan Sun, MD, PhD; Steve Leu, PhD; Cheng-Hsu Yang, MD; Shyh-Ming Chen, MD; Chi-Ling Hang, MD; Yuan-Kai Hsieh, MD; Chien-Jen Chen, MD; Chiung-Jen Wu, MD; Hon-Kan Yip, MD

Crit Care Med 2010; 38:1810–1817





GRAZIE