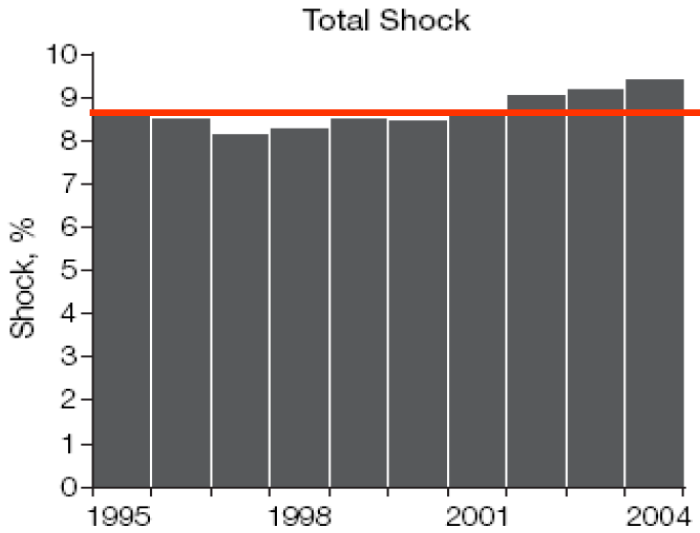
# QUELLO A CUI NON PUO' PIU' RINUNCIARE IL CARDIOLOGO INTENSIVISTA DI UNA MODERNA UTIC



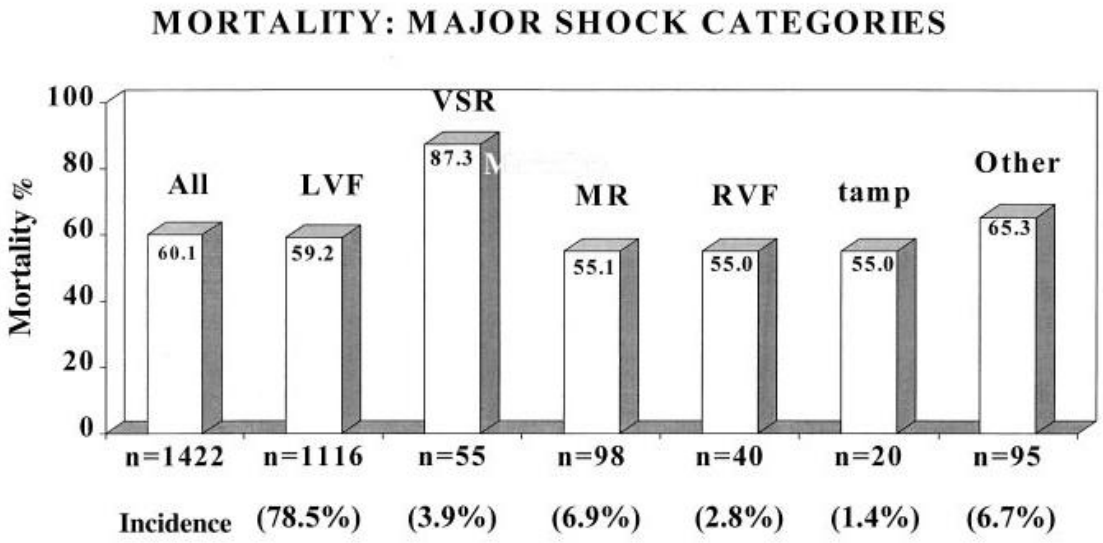
Cardiologia 2 Insufficienza Cardiaca e Trapianto Cardiaco
Dipartimento Cardiologico "A. De Gasperis"
Azienda Ospedaliera Niguarda Cà Granda Milano

# Shock Cardiogeno….i numeri



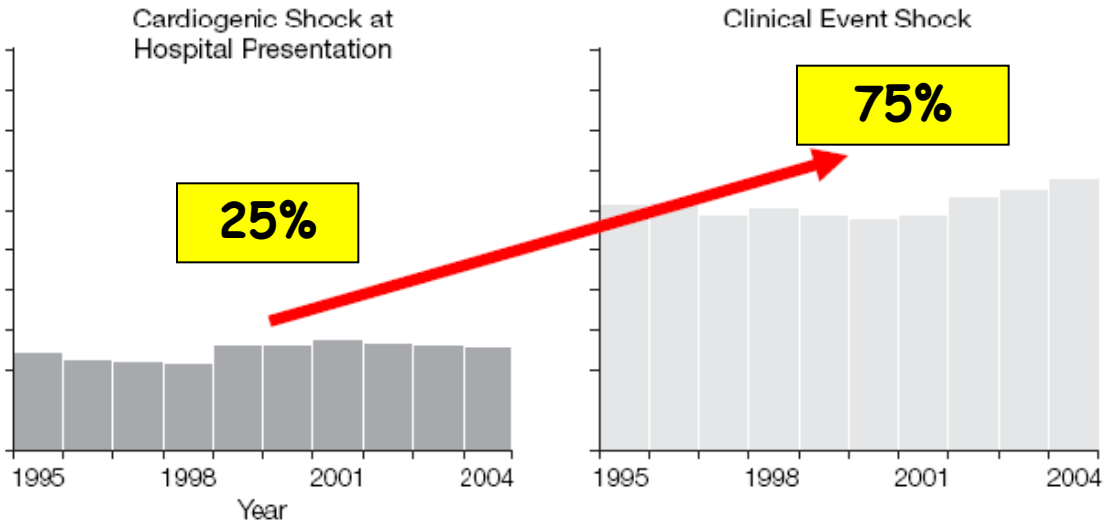

8,6%

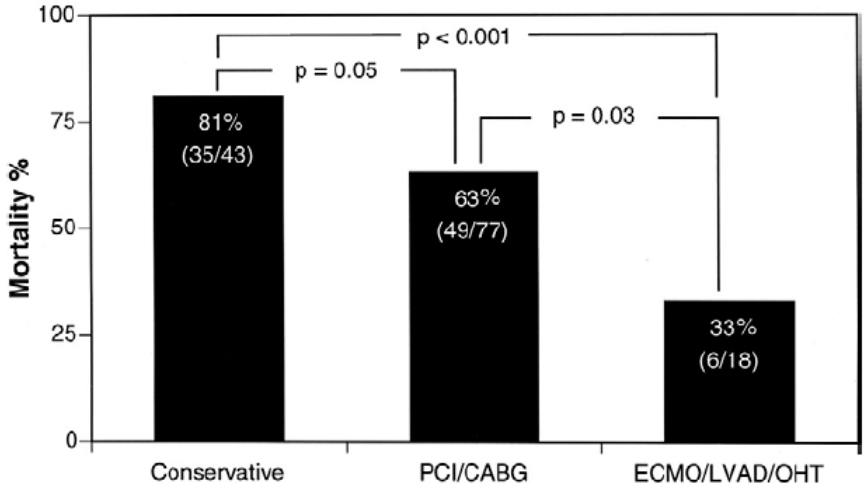
*Babaev et al. JAMA 2005*

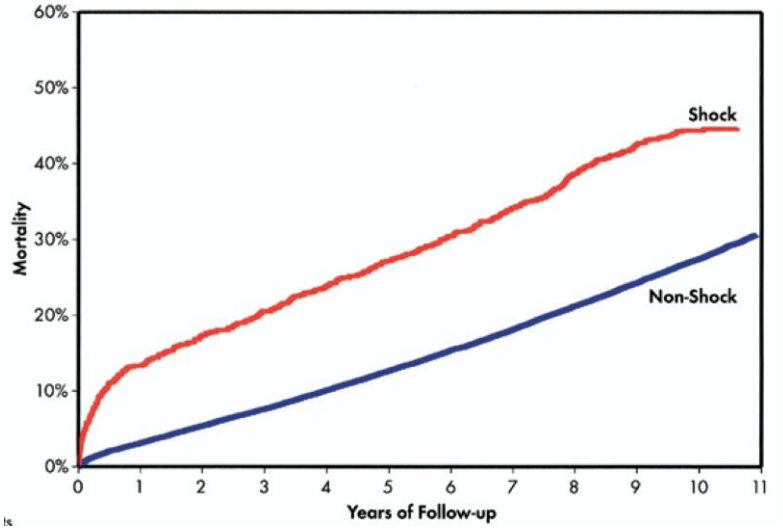

*Shock trial Registry 2000*

# Shock Cardiogeno….i numeri



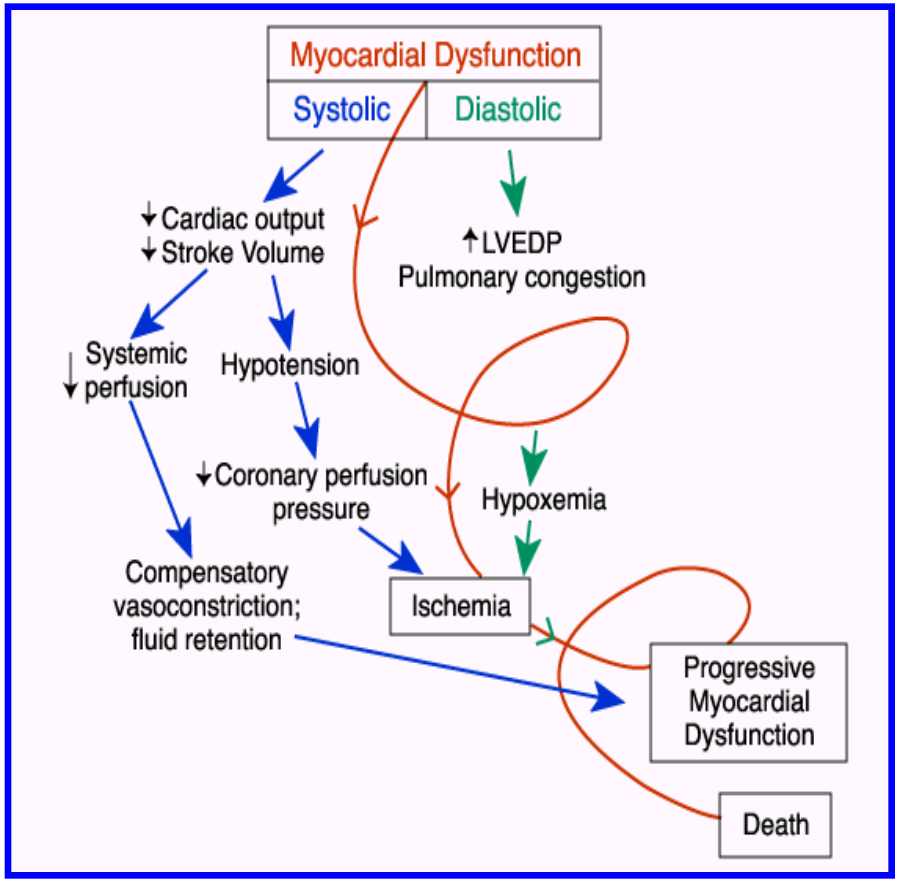

Tayara et al. 2006




GUSTO I. 2007

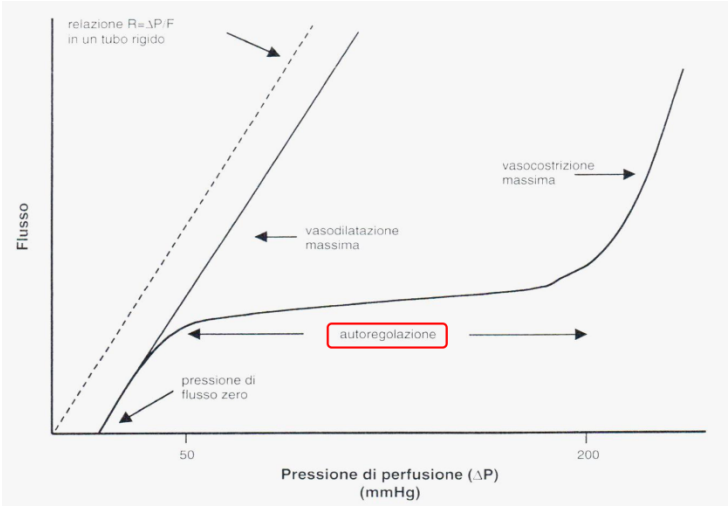
# Shock Cardiogeno
## *definizione*

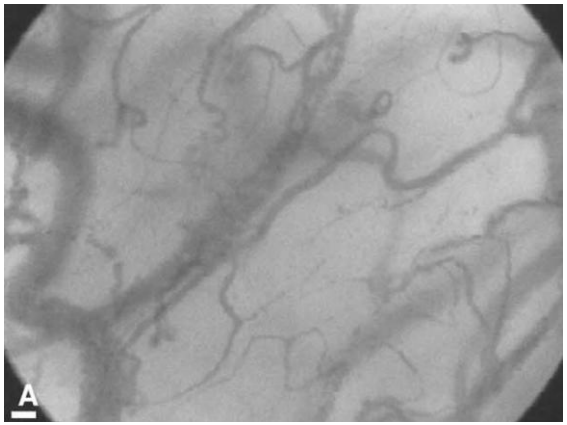
*Lo Shock Cardiogeno è una condizione in cui il cuore, in presenza di un corretto volume circolante, è incapace di mantenere una adeguata portata e quindi un apporto di sangue ai tessuti sufficiente per soddisfare il loro fabbisogno metabolico e per mantenere una normale perfusione coronarica*

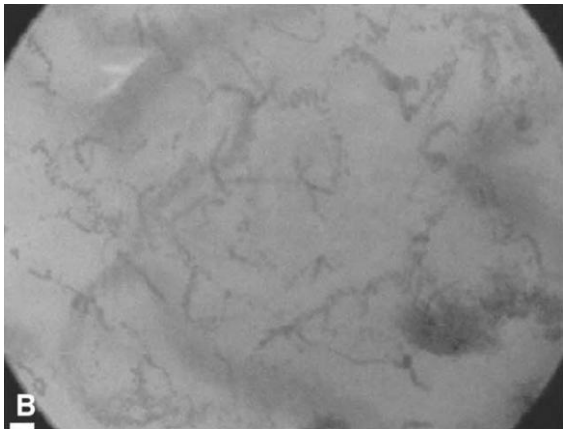


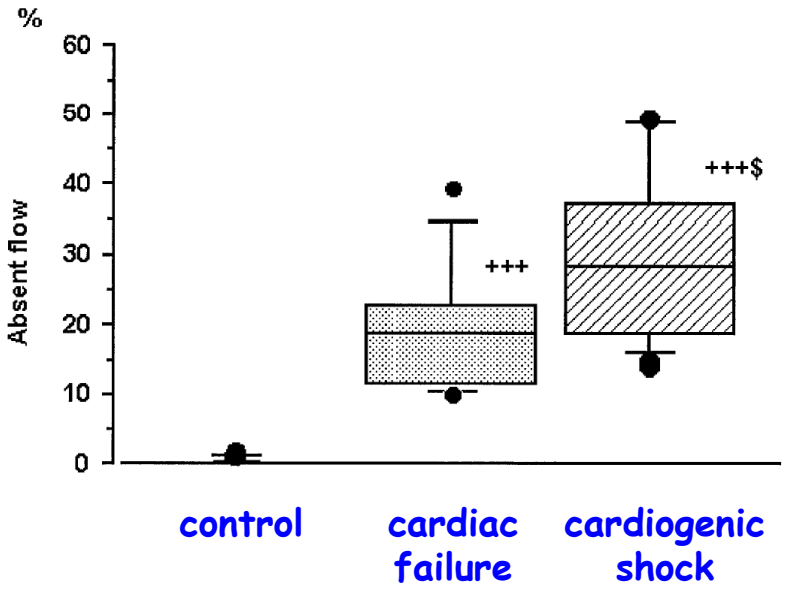

*Hollemberg, Ann. Intern. Med. 1999*

# Shock Cardiogeno
## *patologia del sistema circolatorio*



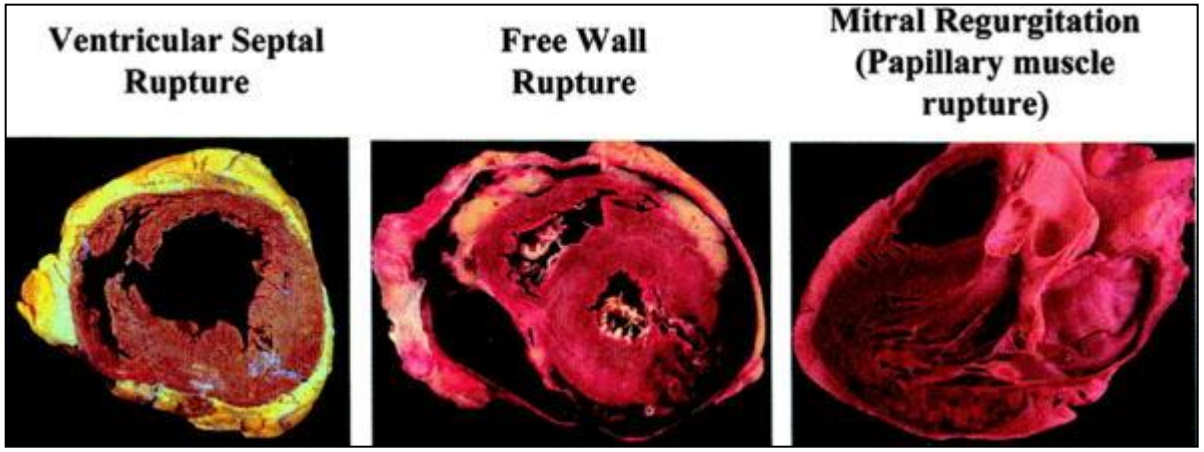

*De Backer et al. Am Heart J 2004*

# Eziologia – Cause cardiache

- INFARTO MIOCARDICO ACUTO

  - Grande estensione area ischemica
  - Sfavorevole evoluzione post rivascolarizzazione
  - Infarto del Ventricolo Destro
  - Complicanze meccaniche IMA

90%



# Eziologia – Cause cardiache

- ✓ Cardiomiopatia Avanzata
- ✓ Miocardite
- ✓ Contusione miocardica
- ✓ Post Cardiotomy
- ✓ Ostruzione tratto efflusso VS
  - Stenosi Aortica
  - CM Ipetrofica Ostruttiva
- ✓ Ostruzione al riempimento VS
  - Stenosi Mitralica
  - Mixoma Atriale sinistra
- ✓ Tamponamento Cardiaco
- ✓ TEP massiva
- ✓ TakoTsubo
- ✓ Feocromocitoma

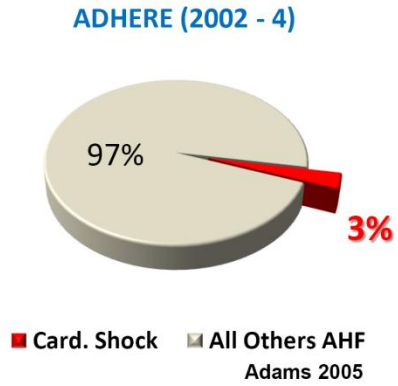
**10%**

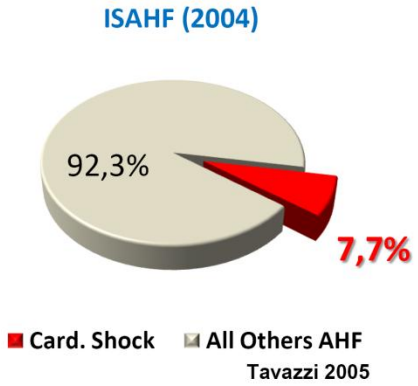
# Eziologia – Cause cardiache

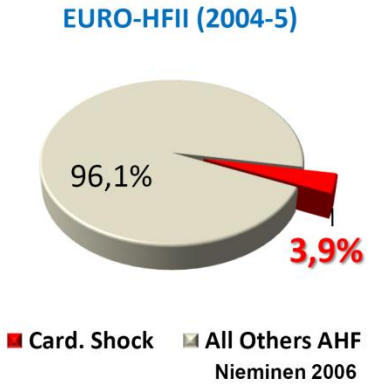
- CARDIOMIOPATIA AVANZATA

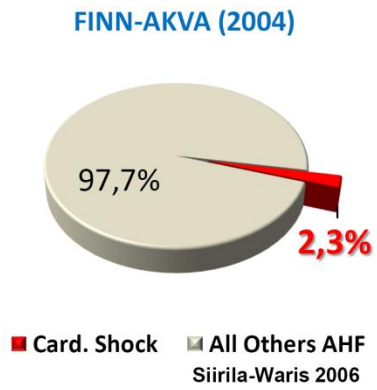
# Eziologia – Cause cardiache

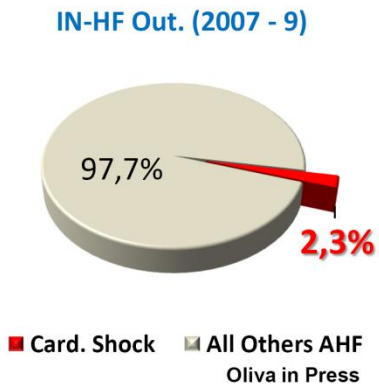
- CARDIOMIOPATIA AVANZATA

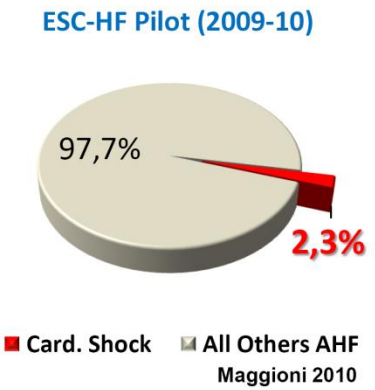


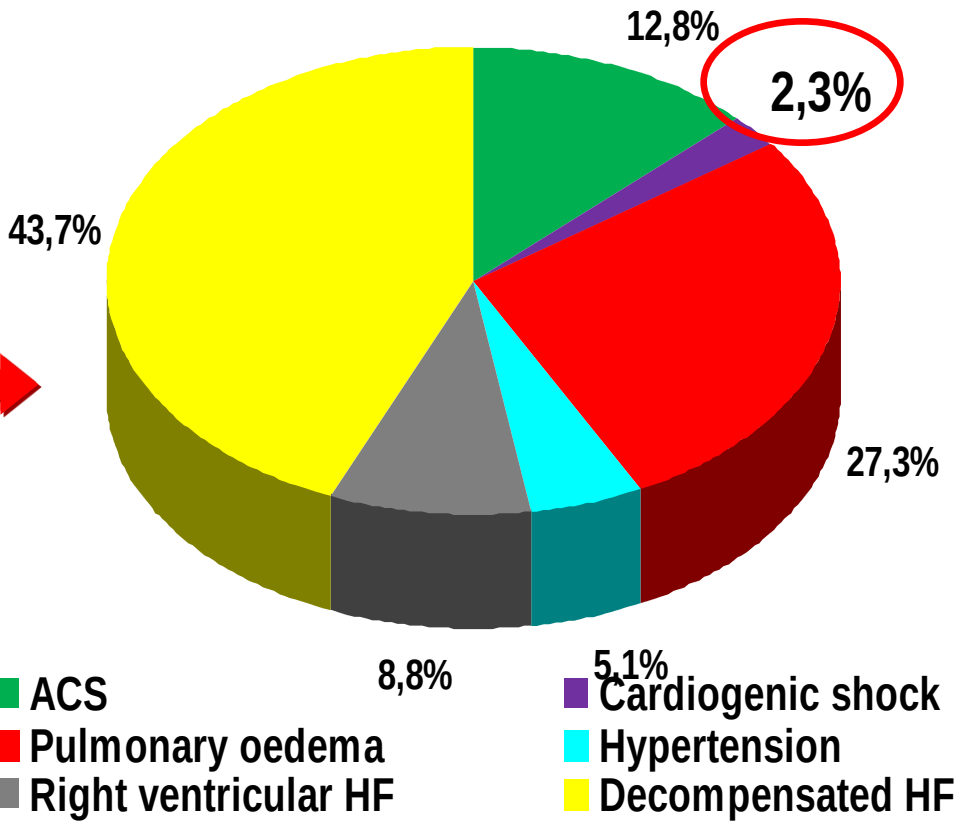



**5610 patients enrolled**

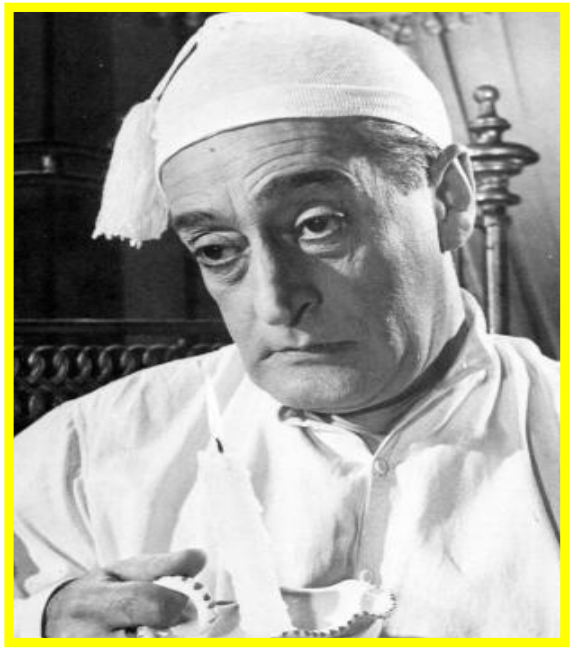

*Enrollment period from*
November 23, 2007
December 31, 2009

# Shock Cardiogeno:
## *Quale monitoraggio?*

- Inquadramento clinico anamnestico
- Monitoraggio organi bersaglio
- Monitoraggio perfusione tissutale

## Diagnosi clinica

- Cianosi/pallore
- Diaforesi
- Estremità ipotermiche
- Polsi periferici iposfigmici
- Sensorio alterato
- Oliguria (QU< 0,5 ml/kg/h)



## Diagnosi strumentale

- PAS< 90mmHG per più di 30'
- SVO2< 60% (prelievo da CVC)
- Lattato≥ 2 mMol/lt (prelievo da puntura arteriosa per EGA)
- Indice Cardiaco < 2,2 l/min/$m^2$ e P Capillare > 18 mmHg

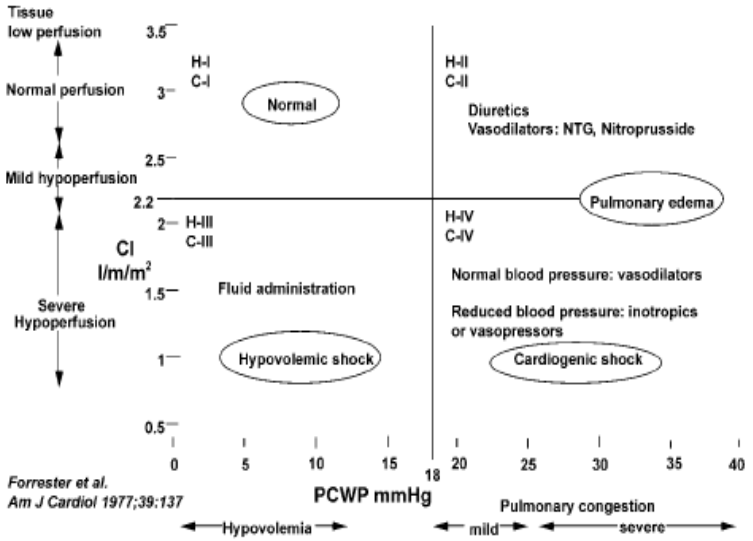
**Stadio I**: No heart failure.

**Stadio II**: Heart failure.

**Stadio III**:Severe HF.

**Stadio IV**: Cardiogenic shock.

*Killip, T. Am J Cardiol 1967*




*Forrester. Am J Cardiol 1977*

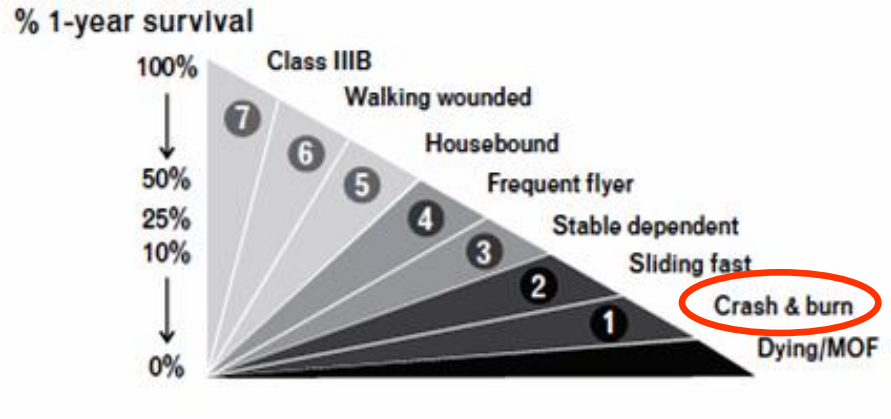
CONGESTION

LOW PERFUSION

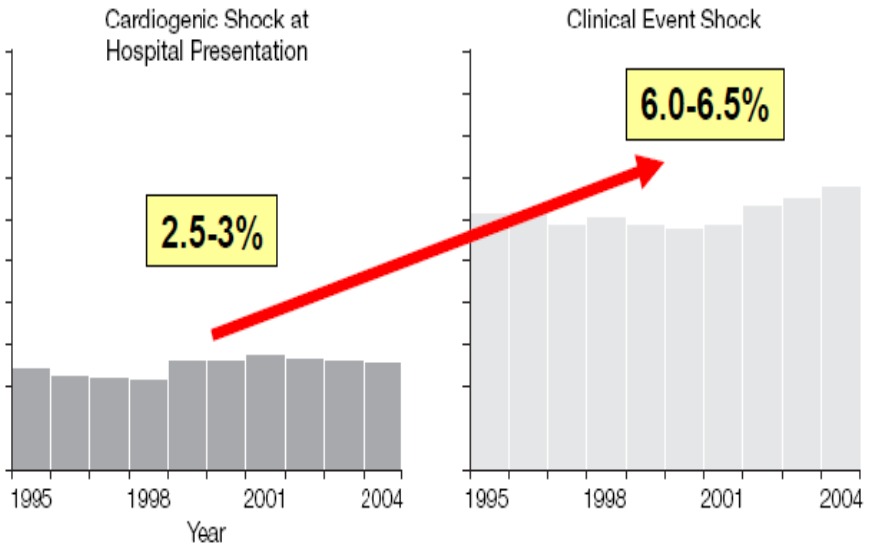
| | |
|---|---|
| **Warm and dry** A | **Warm and wet** B |
| **Cold and dry** L | **Cold and wet** C |

*Nohria et al, JAMA 2002; 287: 628-640*

INTERMACS

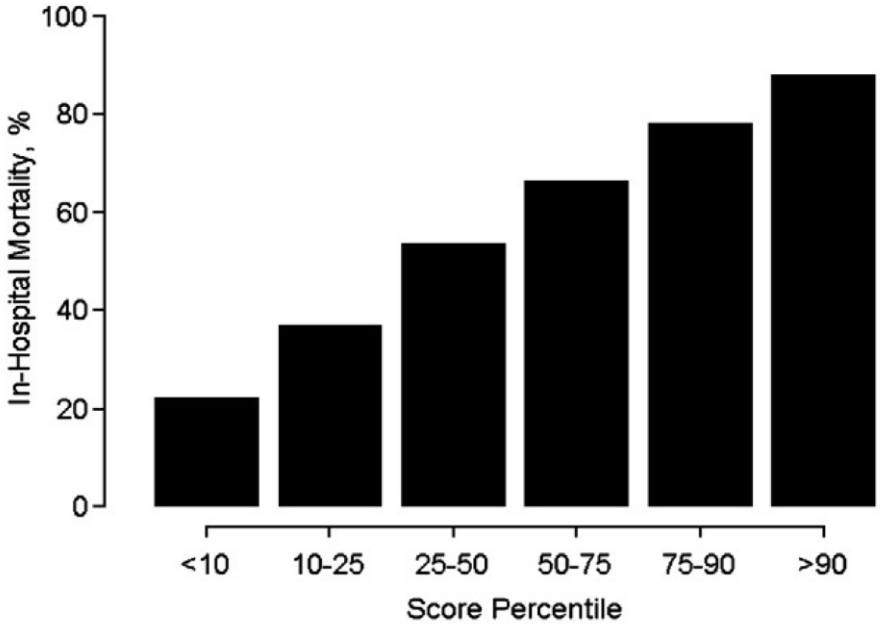



*J Heart Lung Transplant 2009*


Cardiogenic Shock at
Hospital Presentation
Clinical Event Shock
6.0-6.5%
2.5-3%
1995
1998
2001
2004
Year
1995
1998
2001
2004

# Predittori di insorgenza di Shock Cardiogeno

| | Variable | Estimate | SE | Odds ratio | *P* value |
|---|---|---|---|---|---|
| ✗ | Age | 0.047 | .006 | 1.27 per 5-y increase | <.001 |
| | Anoxic brain damage | 3.069 | .799 | 21.52 | .0001 |
| ✗ | End-organ hypoperfusion | 1.425 | .333 | 4.16 | <.001 |
| | Shock on admission | 0.654 | .179 | 1.92 | .0003 |
| | Prior CABG | 0.694 | .235 | 2.00 | .0032 |
| ✗ | Noninferior MI* | 0.327 | .137 | 1.39 | .0172 |
| | Creatinine ≥1.9 mg/dL | 0.516 | .162 | 1.68 | .0016 |
| ✗ | Systolic BP, mmHg† | –0.018 | .003 | 1.09 per 5–mm Hg decrease | <.001 |




| | Variable | Estimate | SE | Odds ratio | *P* value |
|---|---|---|---|---|---|
| ✗ | Stroke work, g/m†,‡ | –.0358 | .0069 | 0.84 per 5 units | <.001 |
| ✗ | LVEF <28%† | 0.7880 | .1924 | 2.20 | <.001 |
| | Age, y | .0413 | .0072 | 1.23 per 5 yr | <.001 |
| | Anoxic brain damage | 2.4902 | .8168 | 12.1 | .002 |
| | End-organ hypoperfusion | 1.3667 | .3889 | 3.92 | <.001 |

*Shock trial Registry 2010*

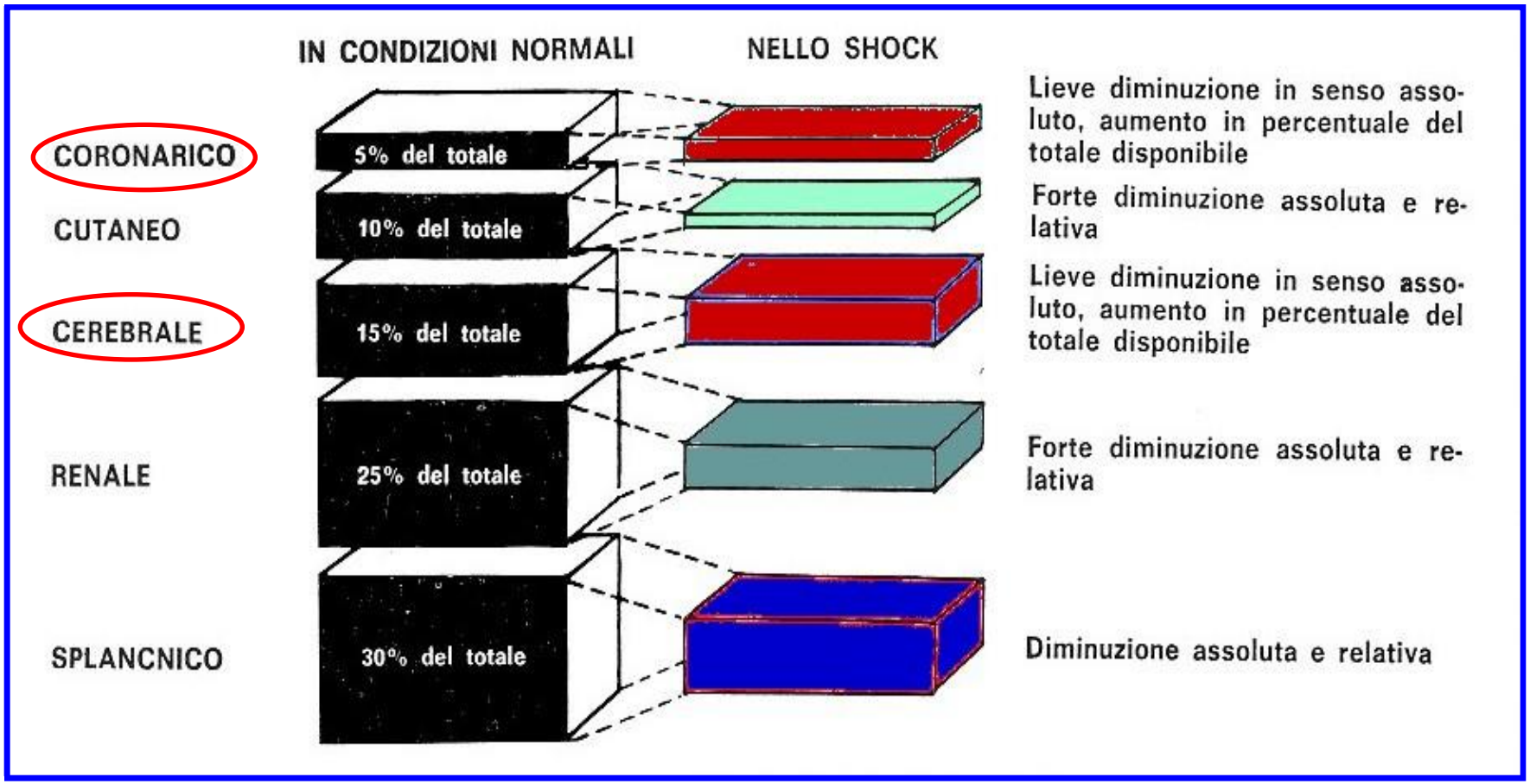
# Shock Cardiogeno:
# *Quale monitoraggio?*

- Inquadramento clinico anamnestico

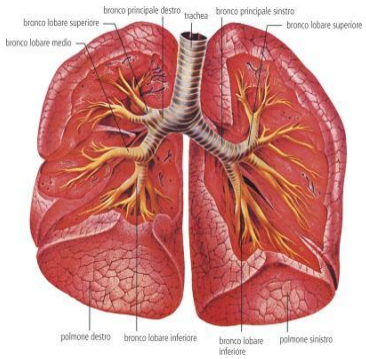
- **Monitoraggio organi bersaglio**
  - Controllo quotidiano esami ematochimici comprendenti funzione renale (urea e creatinina) epatica (AST, ALT gamma-GT, bilirubina), elettroliti, parametri coagulativi

- Monitoraggio perfusione tissutale

# Shock Cardiogeno
## *distribuzione della Gittata Cardiaca*



## POLMONE

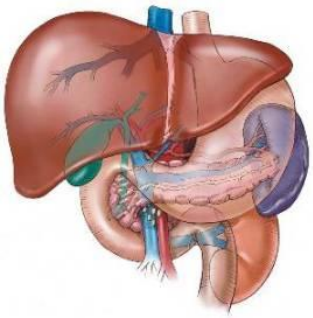



Compaiono precocemente

Riduzione della compliance polmonare e grave alterazione degli scambi gassosi

Insorgenza della Sindrome da Distress respiratorio dell'Adulto (ARDS)

## FEGATO E APP G.I.



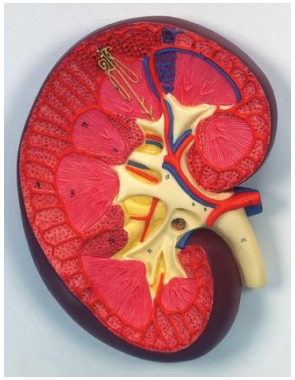
Rialzo transaminasi e indici di colestasi. Scoagulazione spontanea. Difetto di sintesi con riduzione colesterolo e colinesterasi

Ileo paralitico

Colecistite alitiasica

Pancreatite

## RENE



Riduzione diuresi come marker di ipoperfusione

Compenso iniziale con aumento tono arteriole afferenti

Necrosi tubulare acuta e IRA

## SNC

Ipoperfusione

Ipossiemia

Alterazioni acido-basiche

Alterazioni elettrolitiche

Obnubilamento del sensorio

# Shock Cardiogeno: Quale monitoraggio?

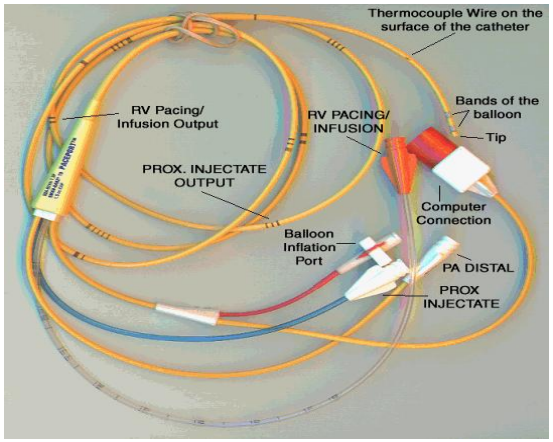
- Inquadramento clinico anamnestico
- Monitoraggio organi bersaglio

- Monitoraggio perfusione tissutale
  - Catetere di Swan Ganz
  - EGA artero-venosa
  - Lattato ematico
  - Biomarkers (IL-1ra, IL-8. TNF-a)

# Shock Cardiogeno: Quale monitoraggio?
## *Catetere di Swan Ganz*

Guida per interventi correttivi:

- espansione volemica
- supporto inotropo
- modulazione dei vasodilatatori




Indice Cardiaco > 2,0

WP < 18mmHg

AD < 10 mmHg

45% dei pz deceduti per Shock Cardiogeno avevano un CI> 2,2 l/min/m2.

# Shock Cardiogeno: Quale monitoraggio?

## *Catetere di Swan Ganz*

Guida per interventi correttivi:

- espansione
- supporto i
- modulazio


L'ottimizzazione dei parametri emodinamici da solo non basta……..CI scarso indicatore della perfusione distrettuale

Indice Cardiac

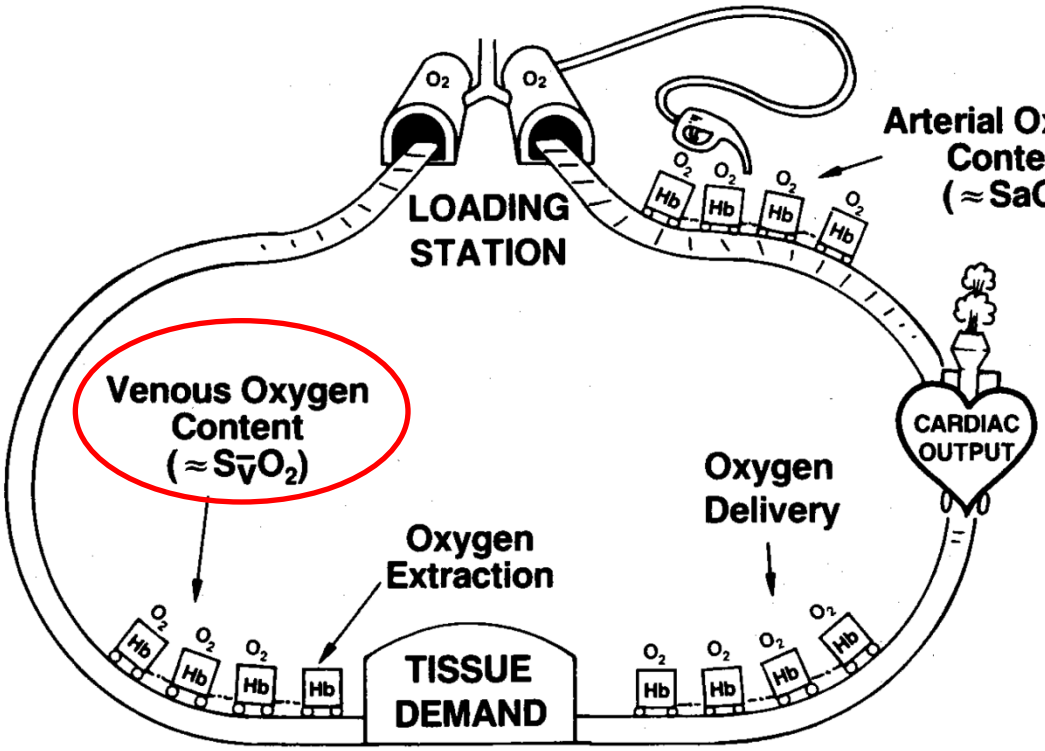
WP < 18mmHg

AD < 10 mmHg

uti per Shock Cardiogeno avevano un CI> 2,2 l/min/m2.

# Catetere di Swan Ganz: SvO2

ESPRIME IL BILANCIO TRA TRASPORTO (DaO2) E CONSUMO DI OSSIGENO (VO2). SvO2= DaO2/VO2




- Hb
- SaO2
- PaO2

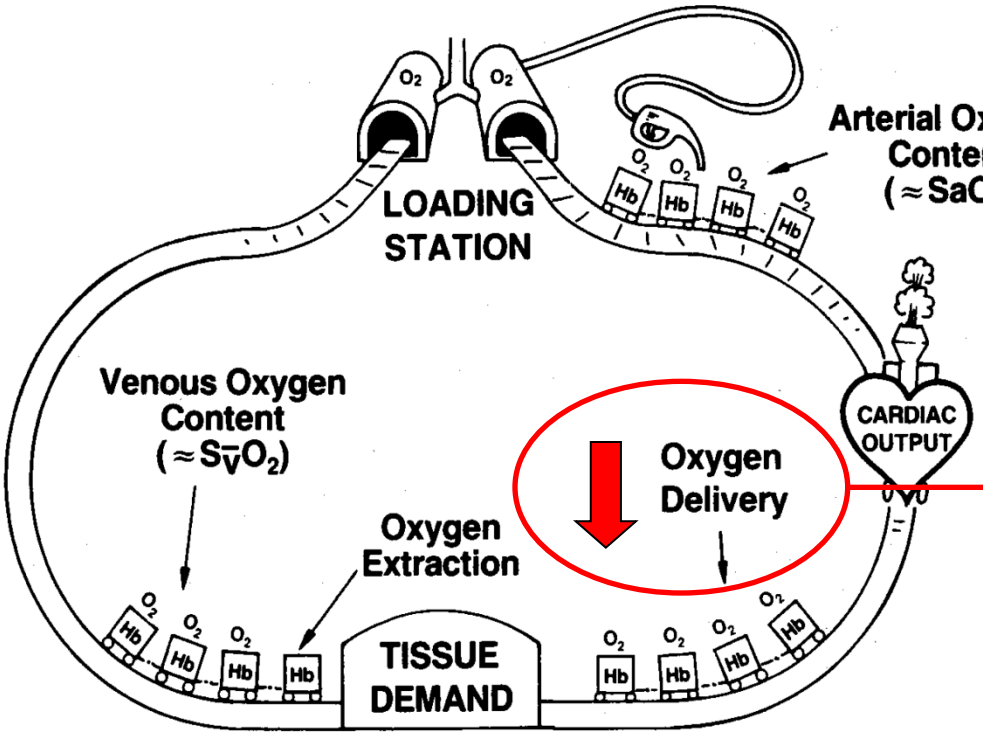
- Stroke Volume
- Heart Rate

Consumo di O2 (VO2)

# Catetere di Swan Ganz: SvO2

Valori normali: 70 ± 5%

Patologici quando < 60%




| | |
|---|---|
| < Hb | •Anemia<br>•Emorragia |
| < SaO2 | •Ipossiemia<br>•Patologie Polmonari |
| < CO | •Ipovolemia<br>•Disfunzione VS<br>•Shock |

↑ VO2

- Febbre-Sepsi
- Brivido
- Convulsioni
- Lavoro respiratorio

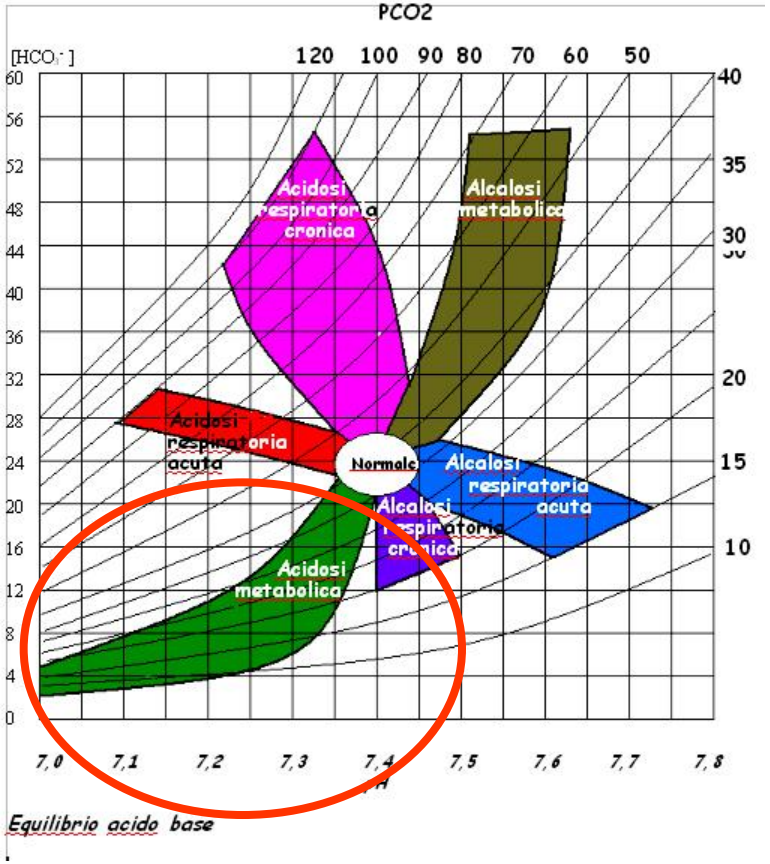
# Shock Cardiogeno:Quale monitoraggio?
## *EGA*

- Valutazione equilibrio acido-base
- Ossigenazione del sangue
- Valutazione andamento CO2
- SvO2
- Valutazione lattati

| | |
|---|---|
| pH | 7,38-7,42 |
| PaCO2 | 38-42 mmHg |
| PaO2 | 90-100 mmHg |
| SaO2 | 95-98% |
| Ht | 37-46% |
| Hb | 12-6 g/dl |
| HCO3 | 21-27 mmol/l |
| Lattato | < 1 mmol/l |
| SVO2 | 70 ± 5% |

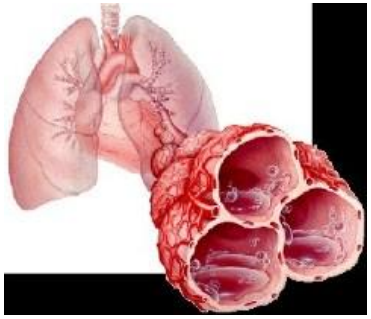
# EGA: equilibrio acido-basico

| | |
|---|---|
| pH | 7,38-7,42 |
| PaCO2 | 38-42 mmHg |
| PaO2 | 90-100 mmHg |
| SaO2 | 95-98% |
| Ht | 37-46% |
| Hb | 12-16 g/dl |
| HCO3 | 21-27 mmol/l |
| Lattato | < 1 mmol/l |
| SVO2 | 70 ± 5% |



# EGA: ossigenazione

| | |
|---|---|
| pH | 7,38-7,42 |
| PaCO2 | 38-42 mmHg |
| PaO2 | 90-100 mmHg |
| SaO2 | 95-98% |
| Ht | 37-46% |
| Hb | 12-16 g/dl |
| HCO3 | 21-27 mmol/l |
| Lattato | < 1 mmol/l |
| SVO2 | 70 ± 5% |



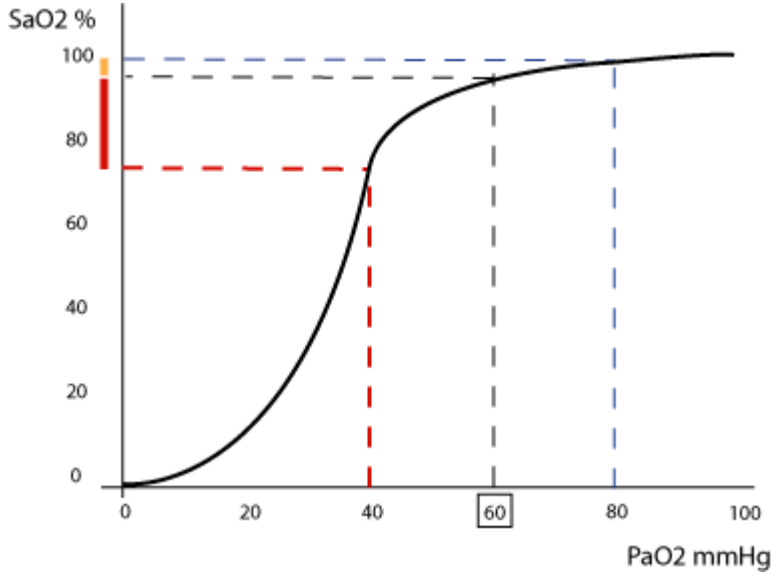
Edema alveolare

⇓

Ipossiemia

⇓

PaO2 < 60 mmHg



# EGA: valutazione CO2

| | |
|---|---|
| pH | 7,38-7,42 |
| PaCO2 | 38-42 mmHg |
| PaO2 | 90-100 mmHg |
| SaO2 | 95-98% |
| Ht | 37-46% |
| Hb | 12-16 g/dl |
| HCO3 | 21-27 mmol/l |
| Lattato | < 1 mmol/l |
| SVO2 | 70 ± 5% |

Iperventilazione compensatoria

↓

PaCO2 < 30mmHg FR > 35 atti/min

↓

Esaurimento muscolare


VAM

# EGA: lattati

| | |
|---|---|
| pH | 7,38-7,42 |
| PaCO2 | 38-42 mmHg |
| PaO2 | 90-100 mmHg |
| SaO2 | 95-98% |
| Ht | 37-46% |
| Hb | 12-16 g/dl |
| HCO3 | 21-27 mmol/l |
| Lattato | < 1 mmol/l |
| SVO2 | 70 ± 5% |

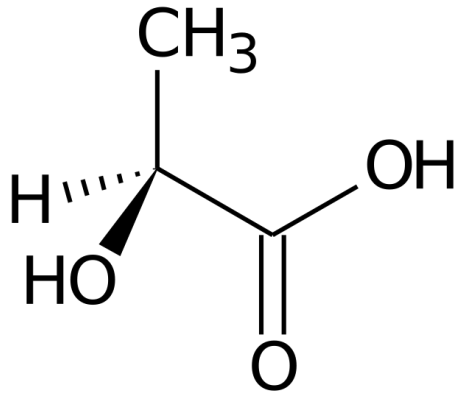
Shock Cardiogeno

↓

Ridotto trasporto di Ossigeno

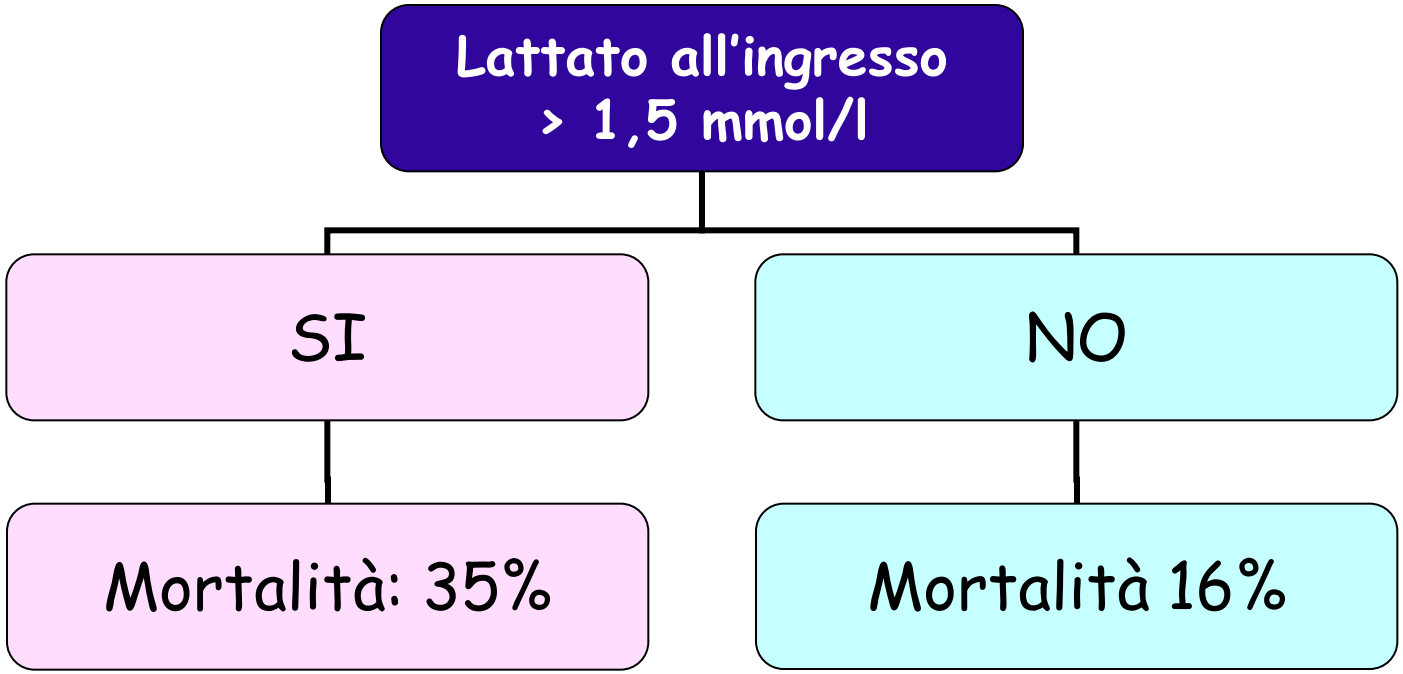
↓

Anaerobiosi tissutale



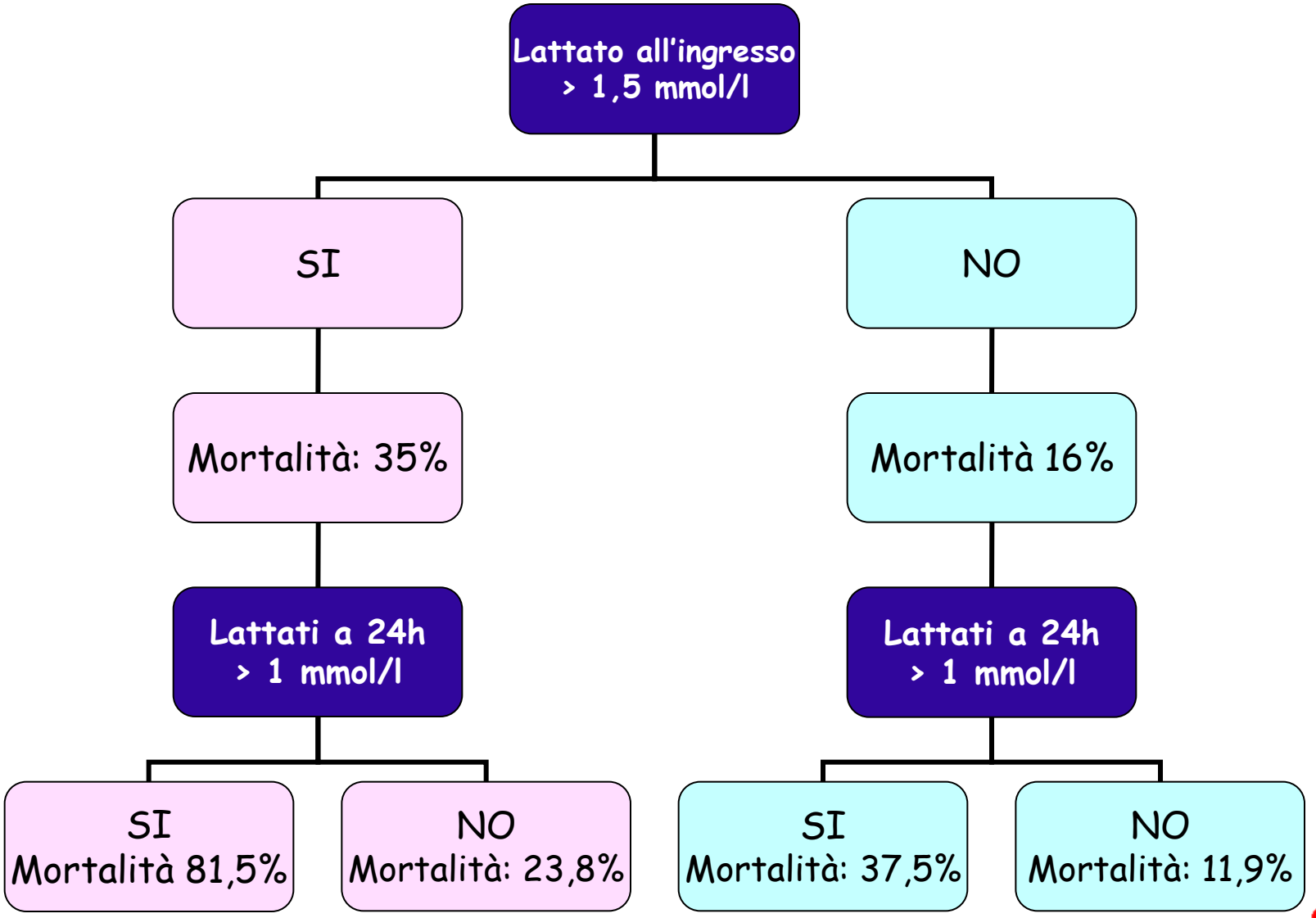

↑ ACIDO LATTICO

# EGA: lattati



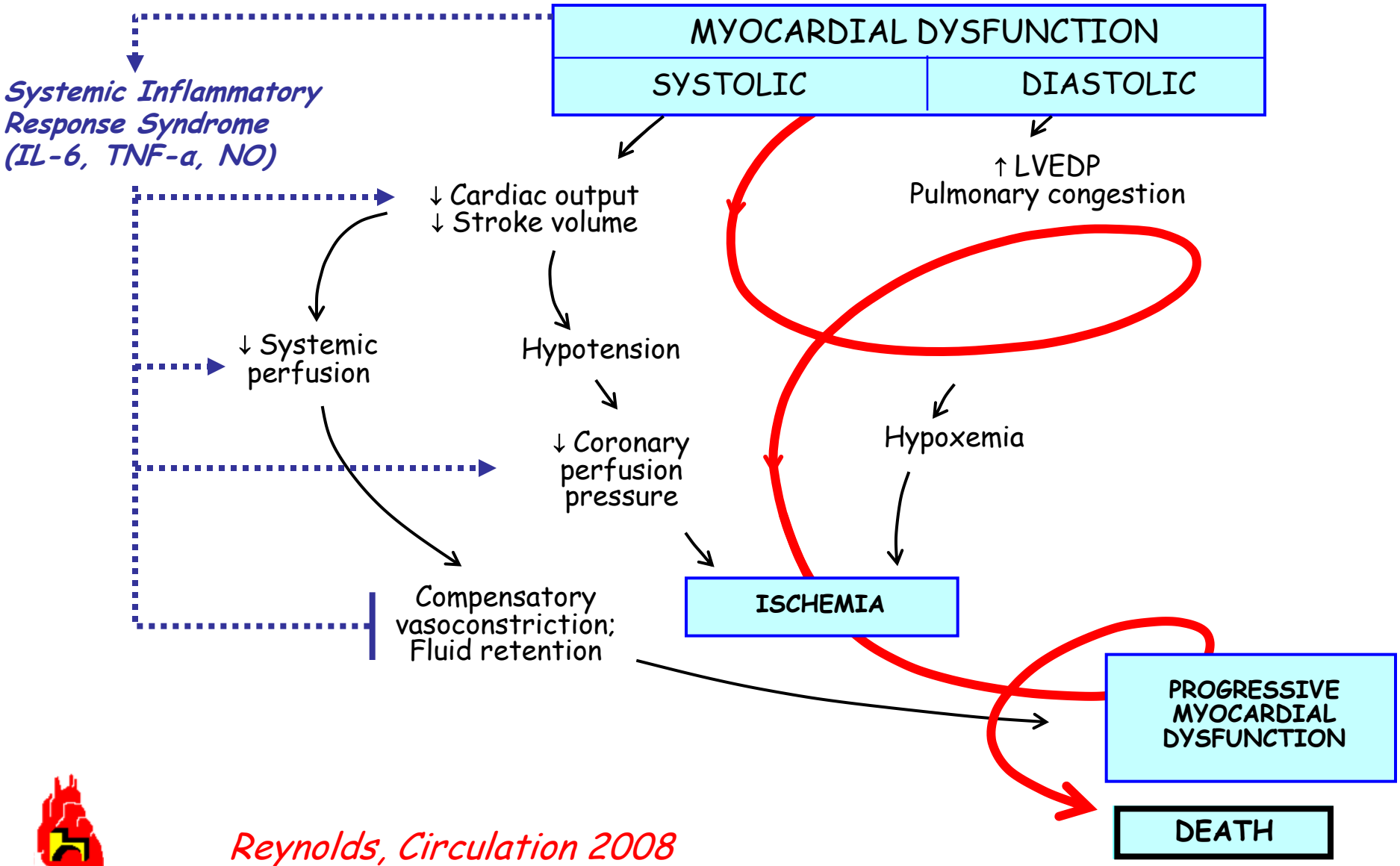

*Smith et al. Intensive Care Med 2001*

# EGA: lattati




*Smith et al. Intensive Care Med 2001*

# Shock Cardiogeno:Quale monitoraggio?
## *Biomarkers umorali*




*Reynolds, Circulation 2008*

# Shock Cardiogeno:Quale monitoraggio?

## *Biomarkers umorali*

Ipoperfusione

↓

risposta infiammatoria sistemica SIRS

↓

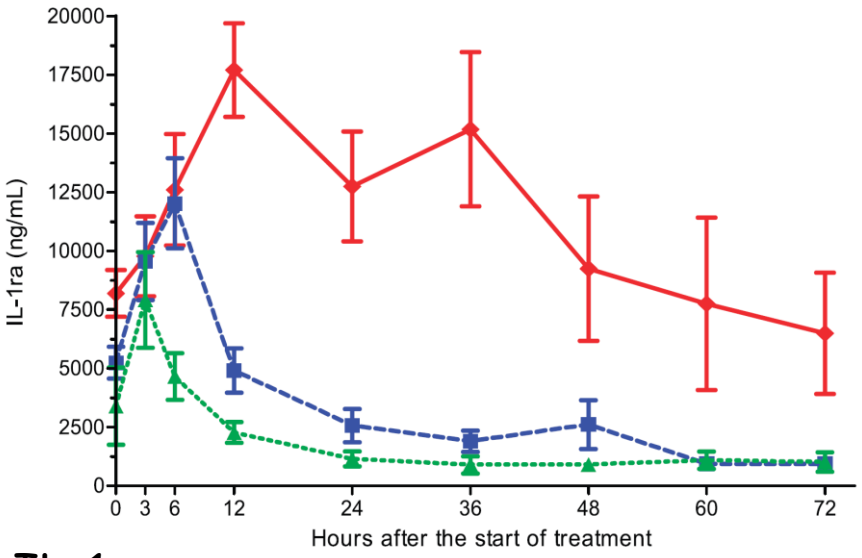
rilascio di mediatori dell'infiammazione tissutale e di neuroormoni

↓

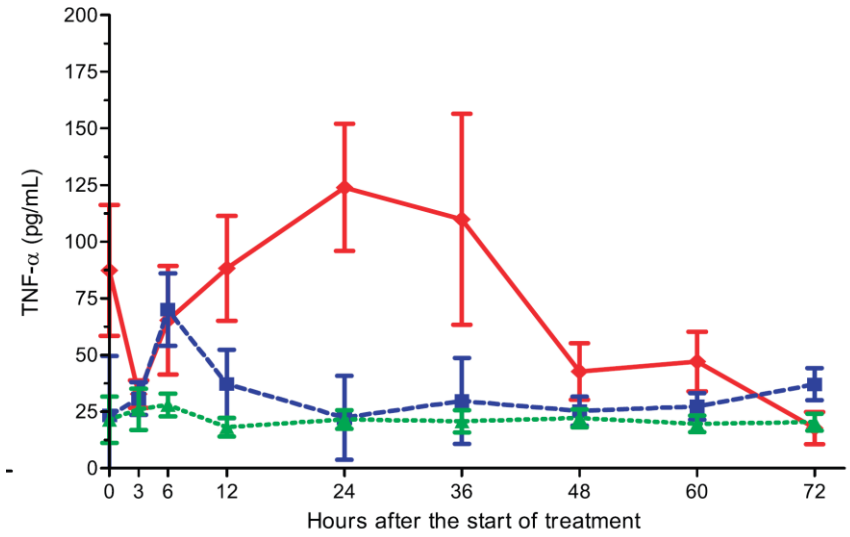
alterazione del microcircolo tissutale MOF.

- Interleukin-1 receptor antagonist (IL-1ra)
- Intercellular adhesion molecule-1 (ICAM-1)
- Tumor necrosis factor a (TNF-a)
- Caspase-3
- Interleukin-8 (IL-8)

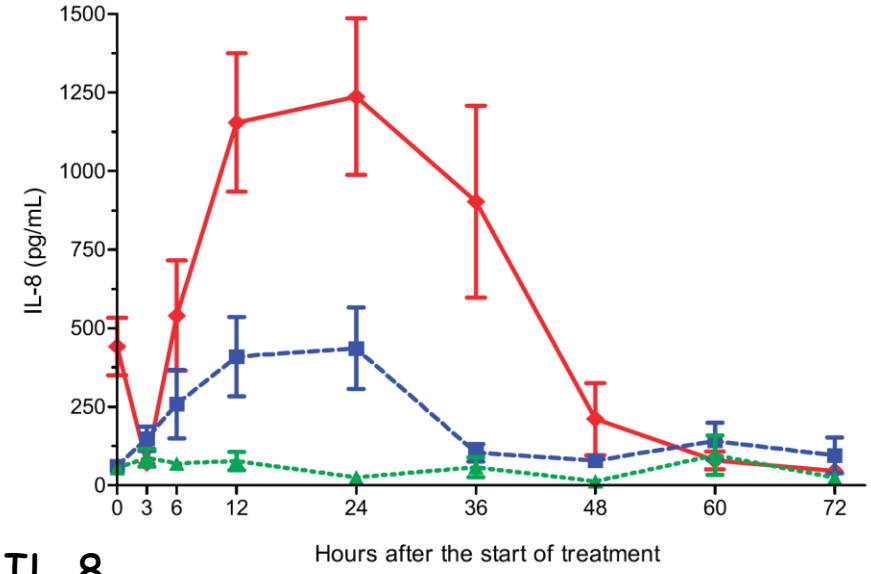
*Andamento dei markers della SIRS in corso di shock sono predditivi di mortalità a breve termine*




IL-1ra



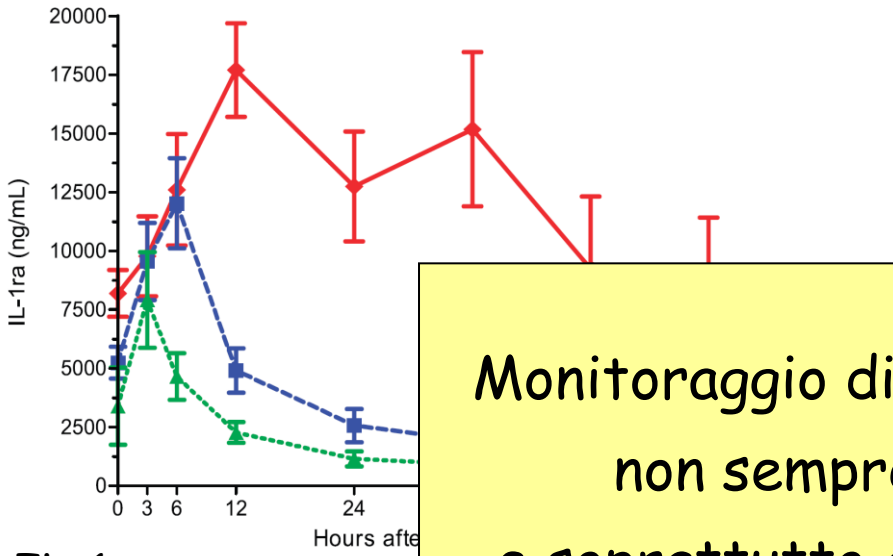

TNF-α



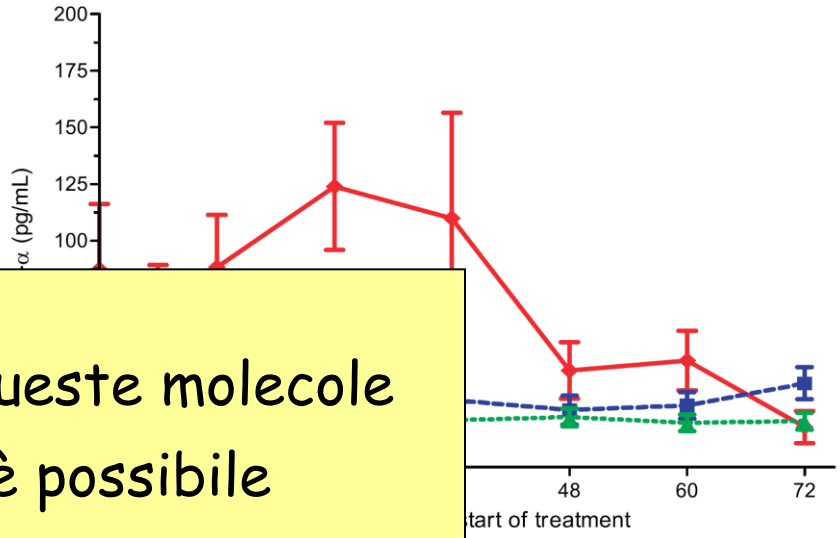

IL-8

- Lactate ≥ 4 and $ScvO_2$ < 70%
- Lactate ≥ 2 and $ScvO_2$ < 70%
- Lactate < 2 and $ScvO_2$ ≥ 70%

*Rivers et al. Crit Care Med 2007*

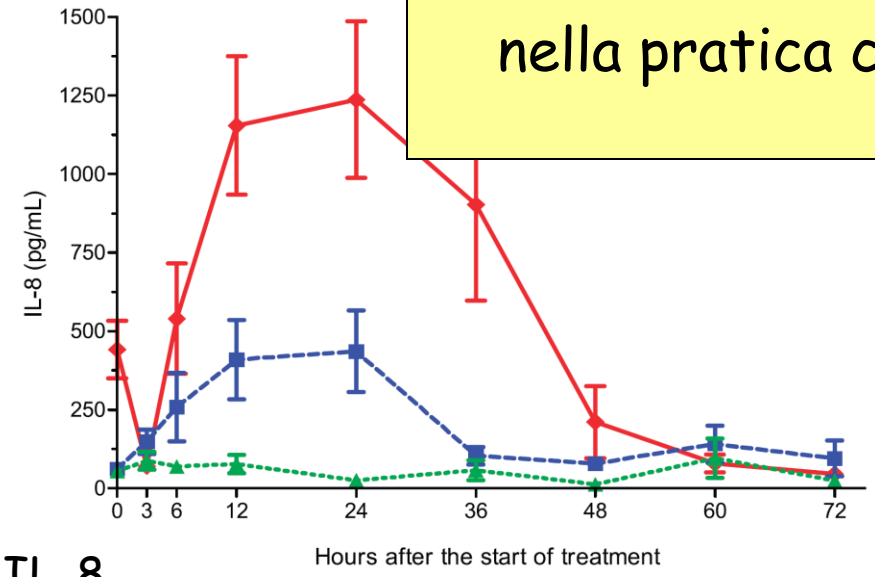



IL-1ra




Monitoraggio di queste molecole
non sempre è possibile
e soprattutto diventa difficile
da utilizzare di routine
nella pratica clinica quotidiana

d $ScvO_2$ < 70%
d $ScvO_2$ < 70%
d $ScvO_2 \geq 70\%$




IL-8

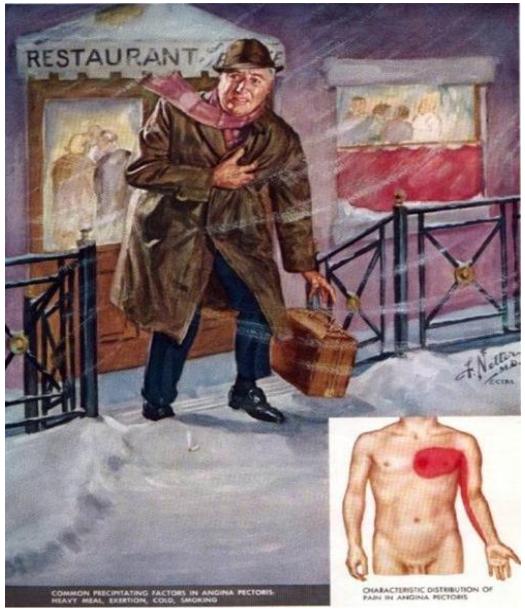
*Rivers et al. Crit Care Med 2007*

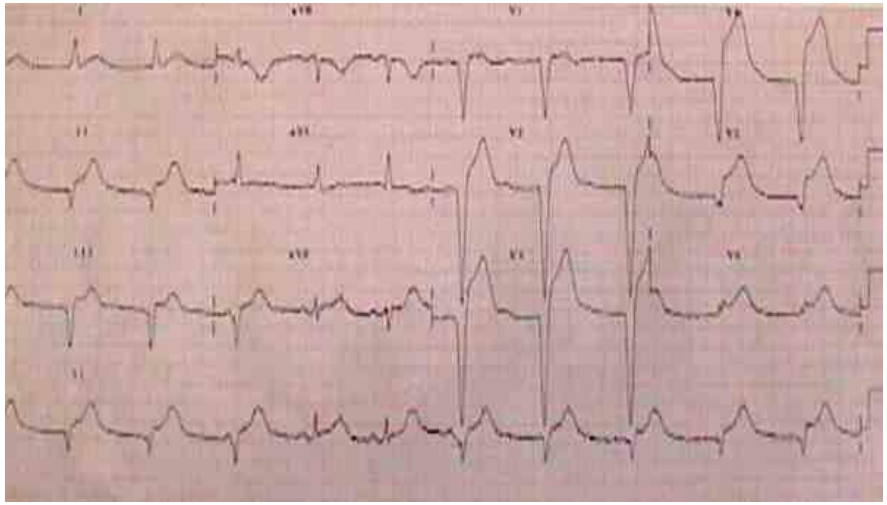
# Shock Cardiogeno: Terapia

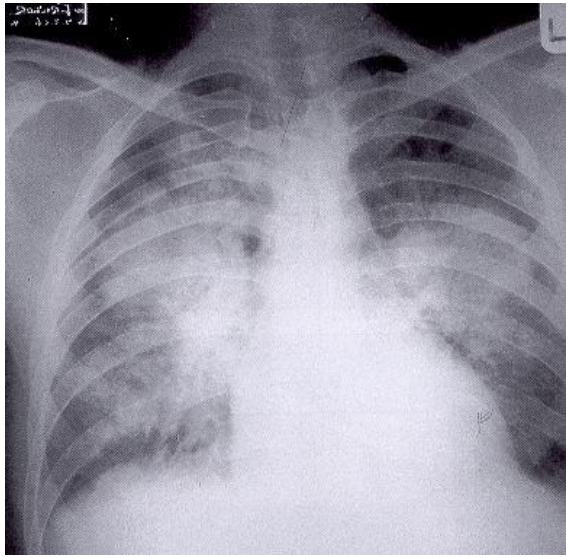
Inquadramento clinico anamnestico:

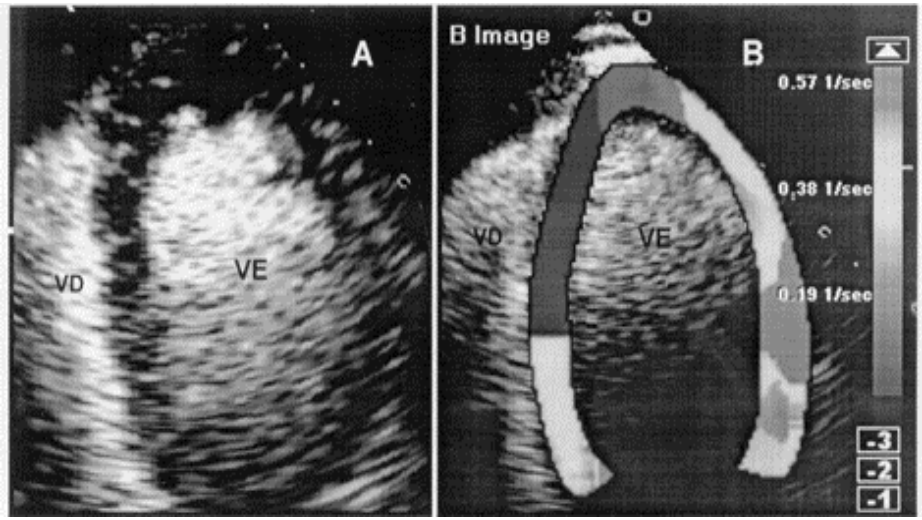
Essenziale per l'immediata impostazione terapeutica:

# Shock Cardiogeno: Caso 1

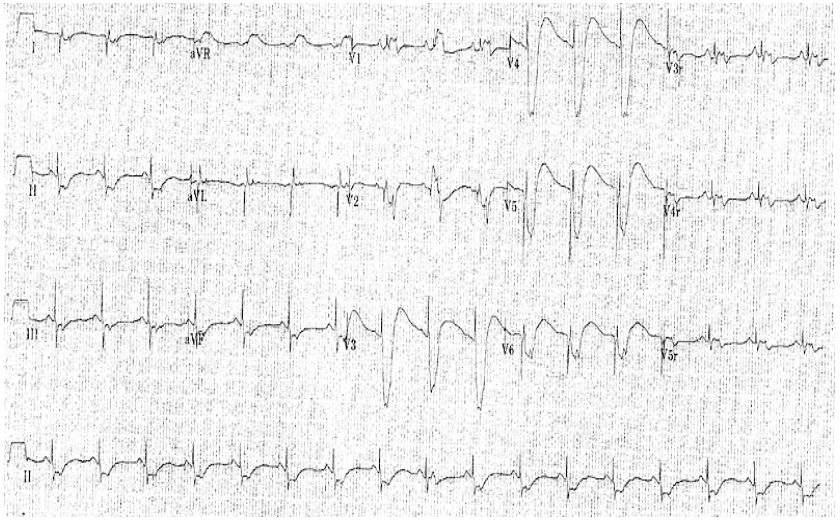


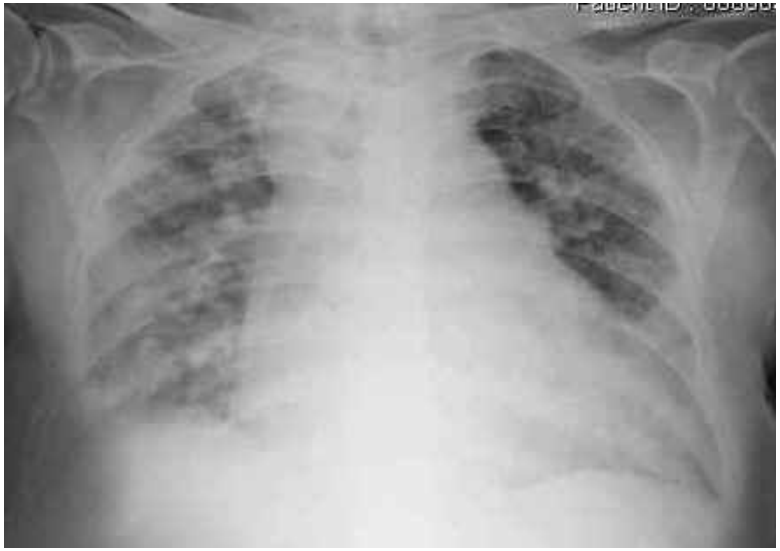



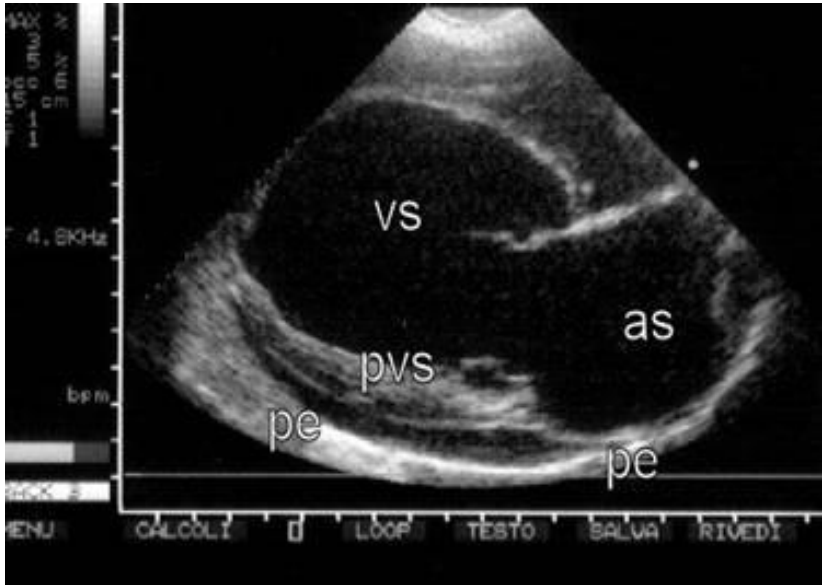




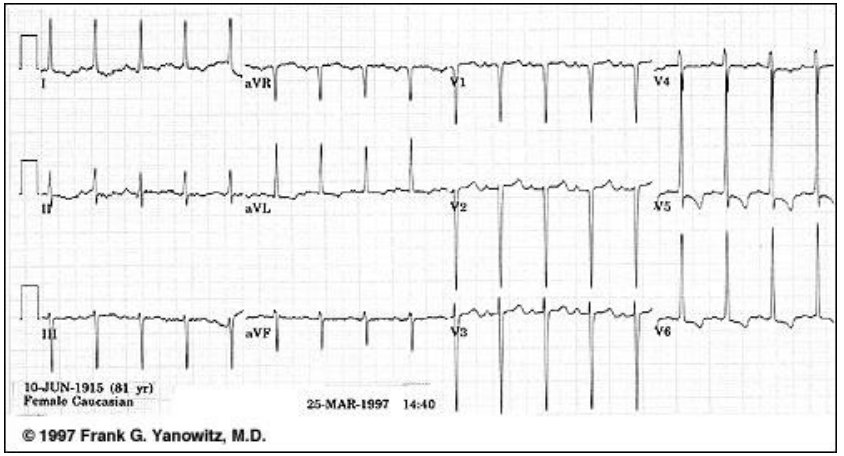
# Shock Cardiogeno: Caso 2

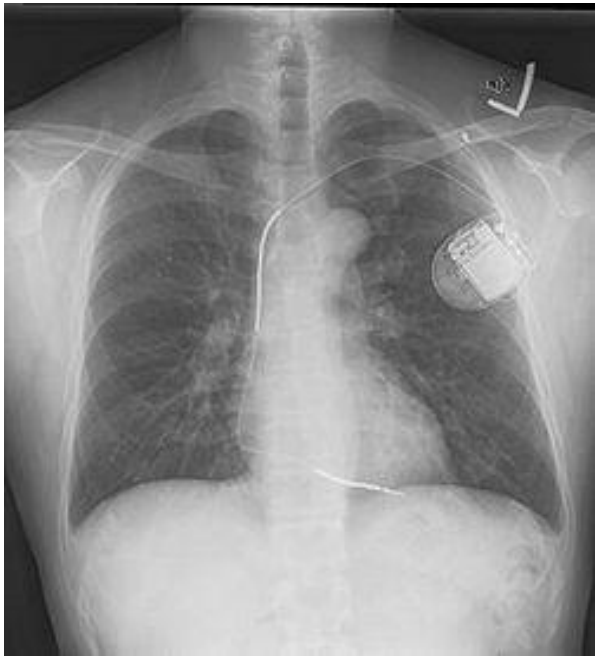


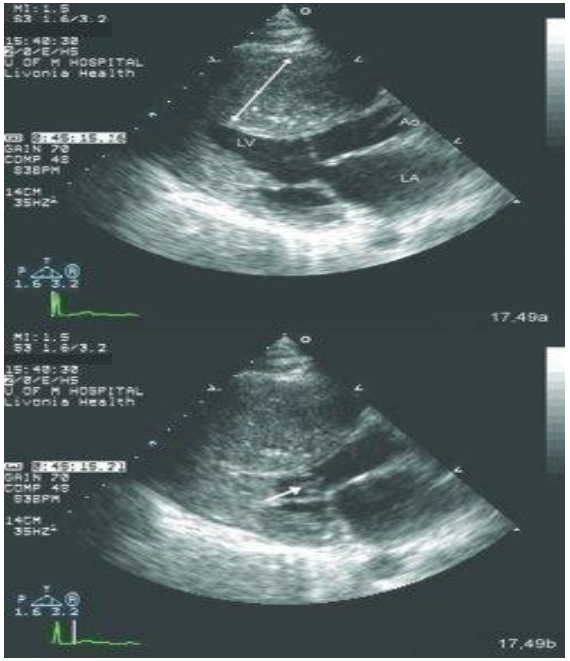




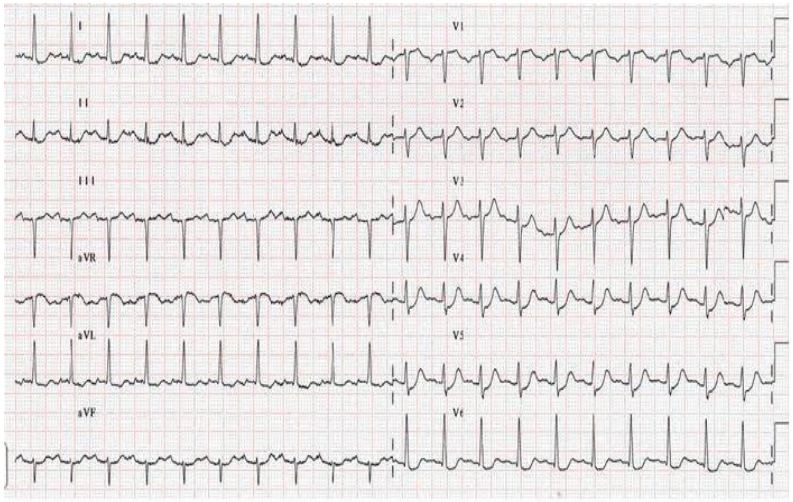
# Shock Cardiogeno: Caso 3

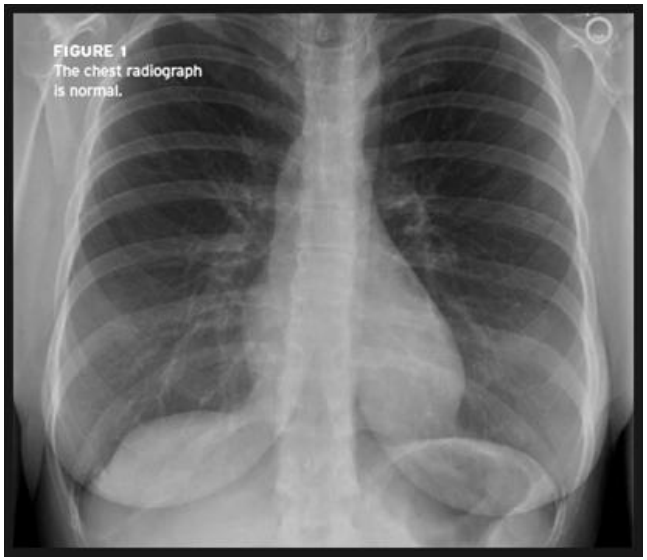


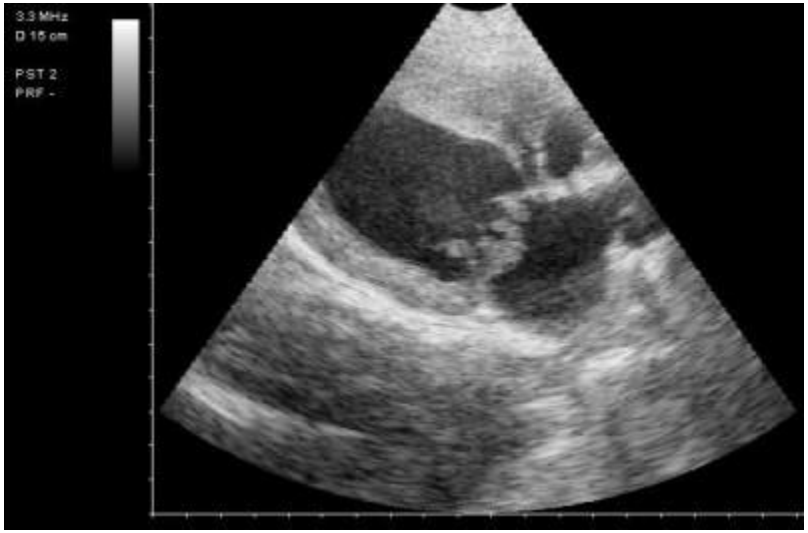





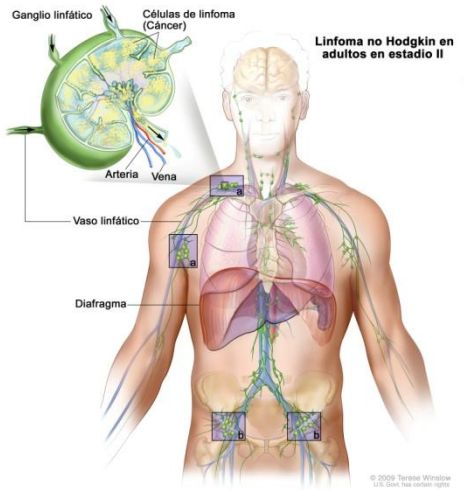
# Shock Cardiogeno: Caso 4

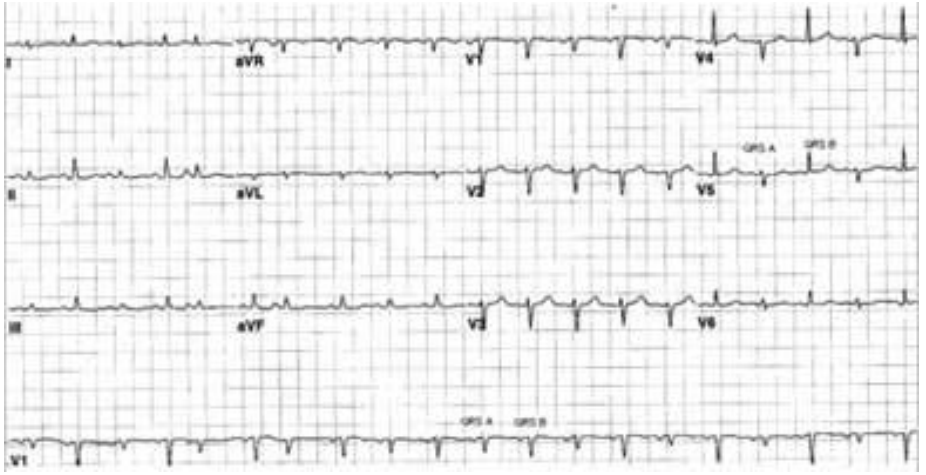


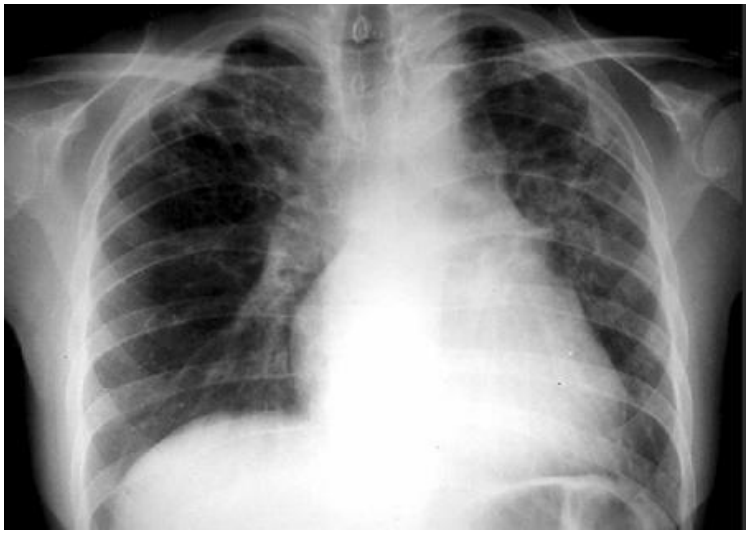



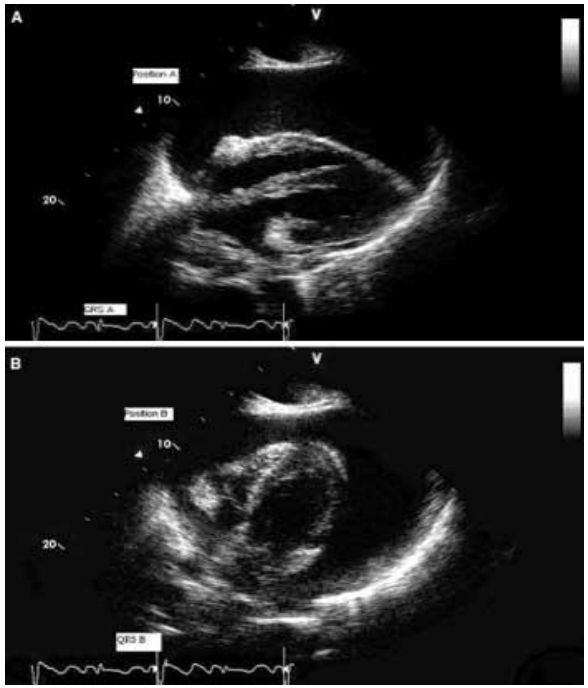



# Shock Cardiogeno: Caso 5

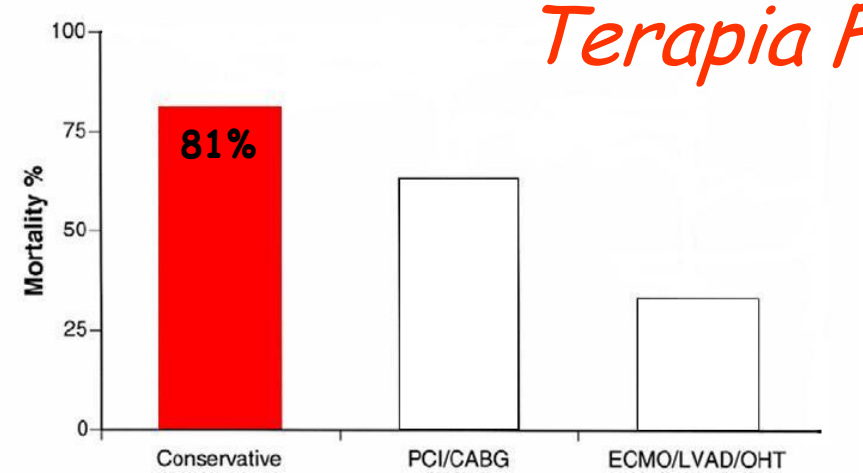


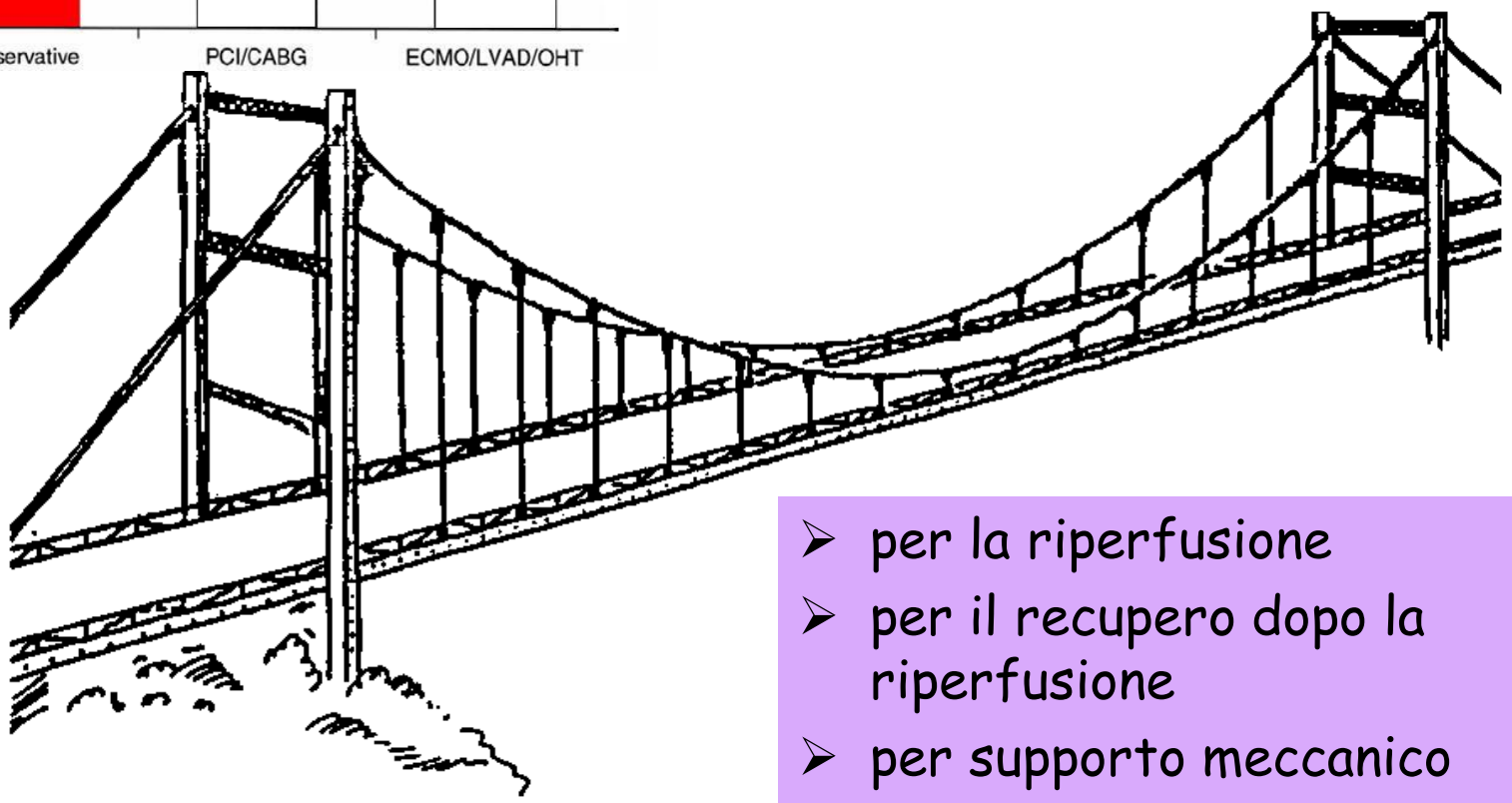





# Shock Cardiogeno:
## *Terapia Farmacologica*



- per la riperfusione
- per il recupero dopo la riperfusione
- per supporto meccanico

# IN-HF *Outcome*


***Enrollment period from***
**November 23, 2007**
**December 31, 2009**

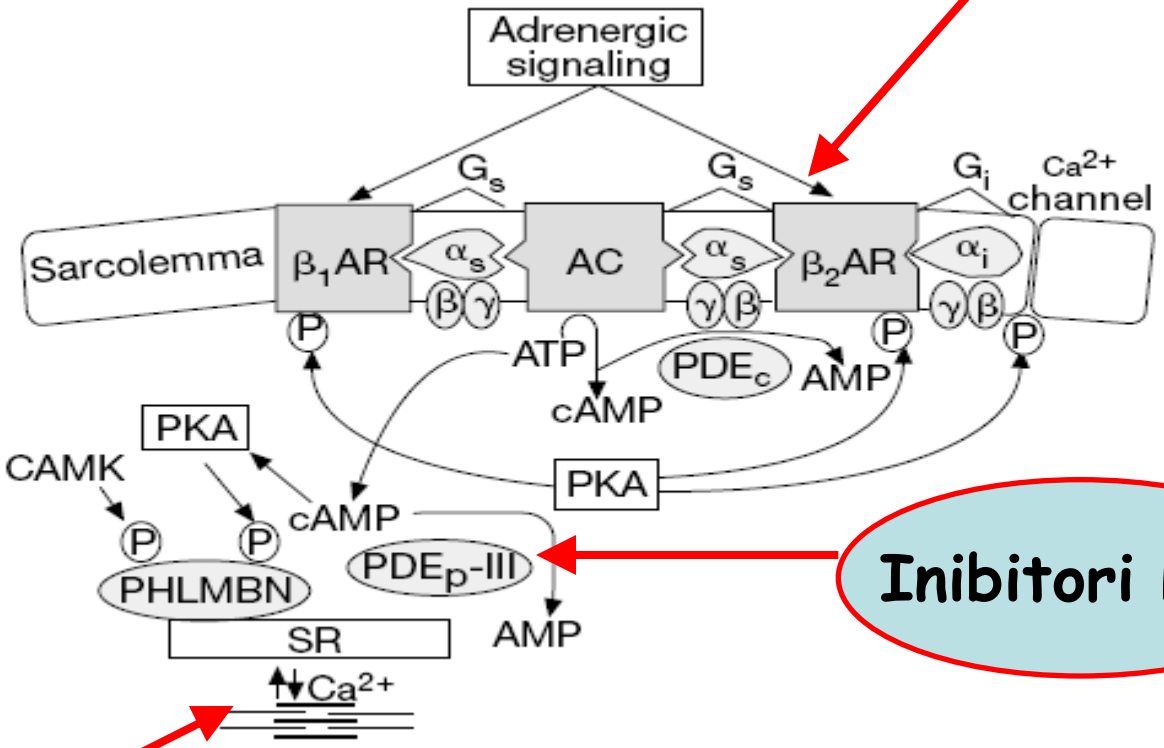
**5610** patients enrolled

| | |
|---|---|
| IV diuretics | |
| IV nitrates | |
| Inotropes | 21% |
| - Dopamine | 14% |
| - Dobutamine | 9% |
| - Levosimendan | 3% |

**Nei pz con Shock 75%**

# Shock Cardiogeno: Terapia Farmacologica

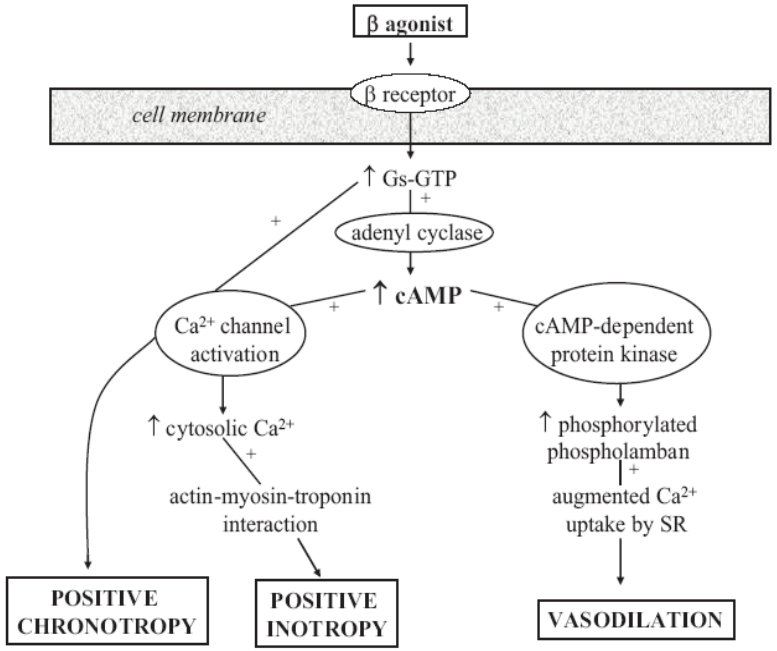
Simpaticomimetici

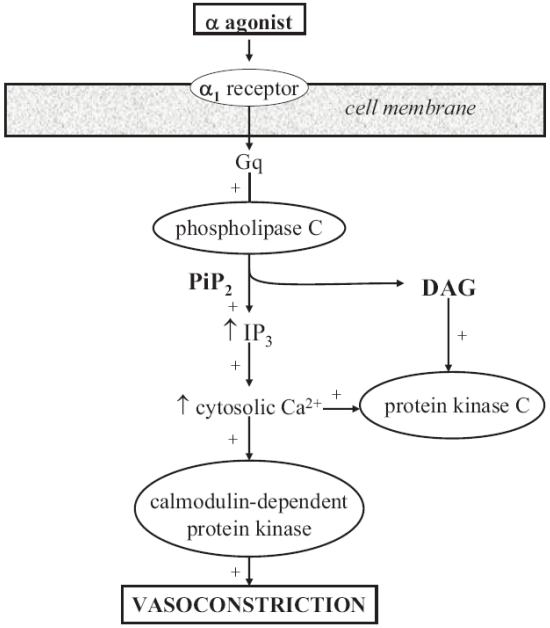



Inibitori PDE

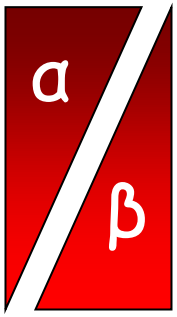
Ca sensibilizz

# Shock Cardiogeno:terapia farmacologica
# Amine Simpaticomimetiche




- ✓ Norepinephrine
- ✓ Epinephrine
- ✓ Dopamine
- ✓ Dobutamine
- ✓ Isoproterenol



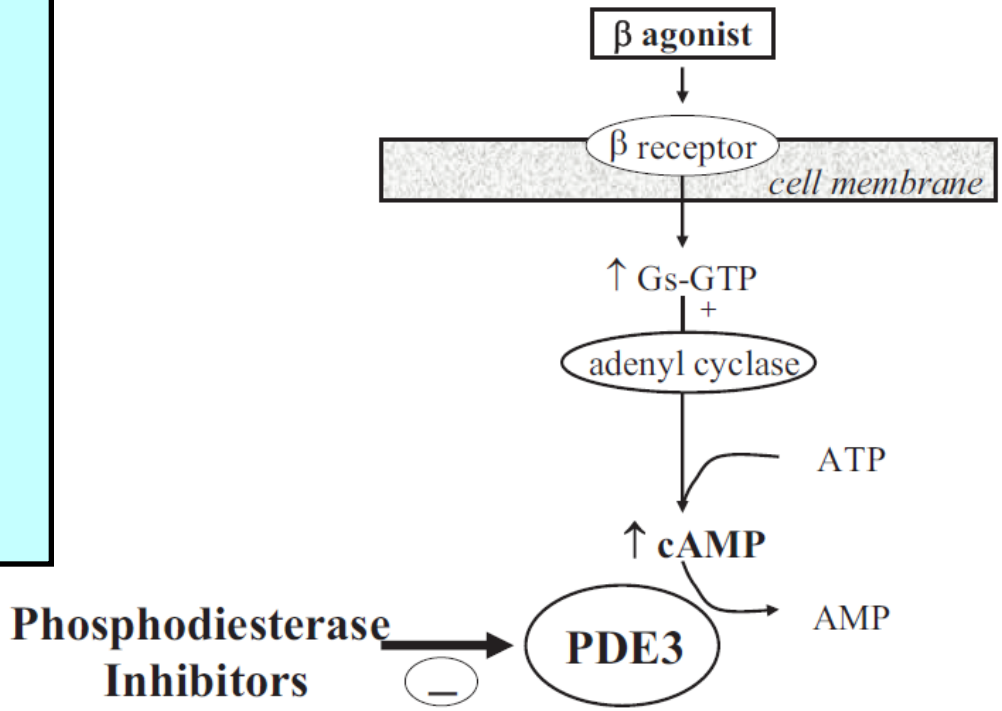

*Christpher et al. Circulation 2008*

# Shock Cardiogeno:terapia farmacologica
# Inibitori Fosfodiesterasi III

- Inodilatatori
- Attivi indipendentemente dallo stato dei recettori adrenergici, in particolare beta
  - Downregulation
  - Tolerance
  - Inattivati da bb
- Incremento mortalità nei pz ischemici
- Ipotensione

Enoximone

Milrinone



# Shock Cardiogeno:terapia farmacologica
# Sensibilizzatore al calcio

**Calcio-sensibilizzatore puro** (alle dosi terapeutiche):

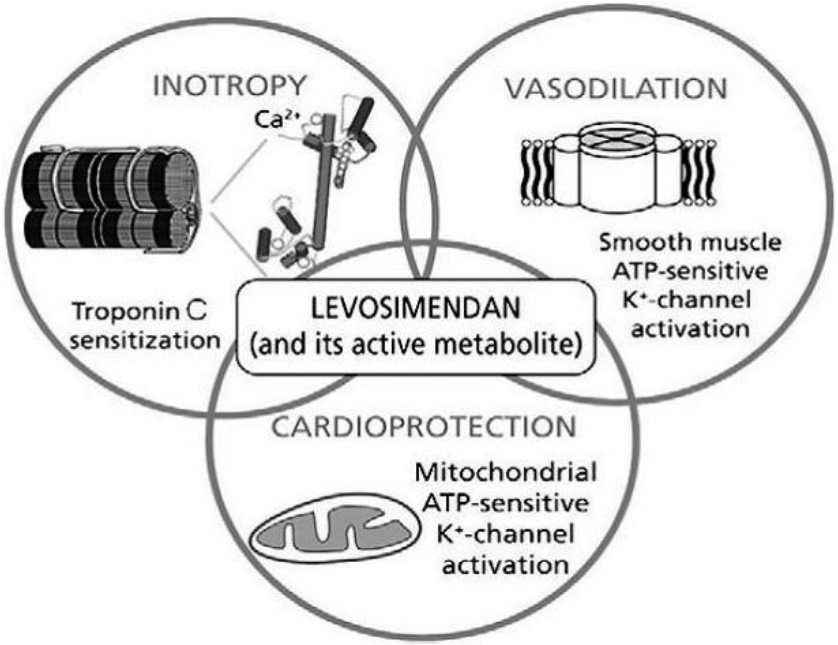
si lega alla TnC **quando ad essa è già legato il $Ca^{++}$** → aumento della responsività dei miofilamenti

↓

Aumento della contrattilità senza incremento del $Ca^{++}$ intracellulare

- senza incrementare il consumo di $O_2$
- senza alterare la funzione diastolica
- senza aritmogenicità

## LEVOSIMENDAN




**Apertura dei canali $K_{ATP}$**

| Nelle fibre muscolari lisce vascolari | Nella membrana interna dei mitocondri |
|---|---|
| ↓ | ↓ |
| Vasodilatazione coronarica e sistemica | Effetto antischemico, anti-apoptotico anti-infiammatorio |
| ↓ | ↓ |
| **CARDIOPROTEZIONE** | |

# Management of acute myocardial infarction in patients presenting with persistent ST-segment elevation

**The Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology:**

| **Treatment of shock (Killip class IV)** | | |
|---|---|---|
| $O_2$ | I | C |
| Mechanical ventilatory support according to blood gasses | I | C |
| Haemodynamic assessment with balloon floating catheter | IIb | C |
| Inotropic agents: dopamine | IIb | B |
| and dobutamine | IIa | C |
| Intra-aortic balloon pump | I | C |
| LV assist devices | IIa | C |
| Early revascularization | I | B |

European Heart Journal (2008) **29**, 2909–2945
doi:10.1093/eurheartj/ehn416

**Dobutamine**

Dobutamine, a positive inotropic agent acting through stimulation of $\beta_1$-receptors to produce dose-dependent positive inotropic and chronotropic effects, is usually initiated with a 2–3 μg/kg/min infusion rate without a loading dose. The infusion rate may then be progressively modified according to symptoms, diuretic response, or clinical status. Its haemodynamic actions are dose-related, which can be increased to 15 μg/kg/min. BP should be monitored, invasively or non-invasively. In patients receiving β-blocker therapy, dobutamine doses may have to be increased to as high as 20 μg/kg/min to restore its inotropic effect.[234] The elimination of the drug is rapid after cessation of infusion. Care should be exercised in weaning patients from dobutamine infusion. Gradual tapering (i.e. decrease in dosage by steps of 2 μg/kg/min) and simultaneous optimization of oral therapy are essential.

**Class of recommendation IIa, level of evidence B**

**Dopamine**

Dopamine, which also stimulates β-adrenergic receptors both directly and indirectly with a consequent increase in myocardial contractility and cardiac output, is an additional inotropic agent. Infusion of low doses of dopamine (≤2–3 mg/kg/min) stimulates dopaminergic receptors but has been shown to have limited effects on diuresis. Higher doses of dopamine may be used to maintain SBP, but with an increasing risk of tachycardia, arrhythmia, and α-adrenergic stimulation with vasoconstriction. Dopamine and dobutamine should be used with caution in patients with a heart rate >100 b.p.m.[232] The alpha stimulation at higher doses may lead to vasoconstriction and elevated systemic vascular resistance. Low-dose dopamine is frequently combined with higher doses of dobutamine.

**Class of recommendation IIb, level of evidence C**

# ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012

**The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care Medicine (ESICM)**

**Vasopressors**

Vasopressors (norepinephrine) are not recommended as first-line agents and are only indicated in cardiogenic shock when the combination of an inotropic agent and fluid challenge fails to restore SBP >90 mmHg, with inadequate organ perfusion, despite an improvement in cardiac output. Patients with sepsis complicating AHF may require a vasopressor. Since cardiogenic shock is usually associated with a high systemic vascular resistance, all vasopressors should be used with caution and discontinued as soon as possible. Noradrenaline might be used with any of above-mentioned inotropic agents in cardiogenic shock, ideally perfused through a central line. Caution is advised with dopamine that already exerts a vasopressor effect. Epinephrine is not recommended as an inotrope or vasopressor in cardiogenic shock and should be restricted to use as rescue therapy in cardiac arrest.

**Class of recommendation IIb, level of evidence C**

### Milrinone and enoximone

Milrinone and enoximone are the two type III phosphodiesterase inhibitors (PDEIs) used in clinical practice. The agents inhibit the breakdown of cyclic AMP and have inotropic and peripheral vasodilating effects, with an increase in cardiac output and stroke volume, and a concomitant decline in pulmonary artery pressure, pulmonary wedge pressure, and systemic and pulmonary vascular resistance. As their cellular site of action is distal to the β-adrenergic receptors, the effects of PDEIs are maintained during concomitant β-blocker therapy.[236] Milrinone and enoximone are administered by a continuous infusion possibly preceded by a bolus dose in patients with well-preserved BP. Caution should be used with the administration of PDEIs in patients with CAD, as it may increase medium-term mortality.[231]

**Class of recommendation IIb, level of evidence B**

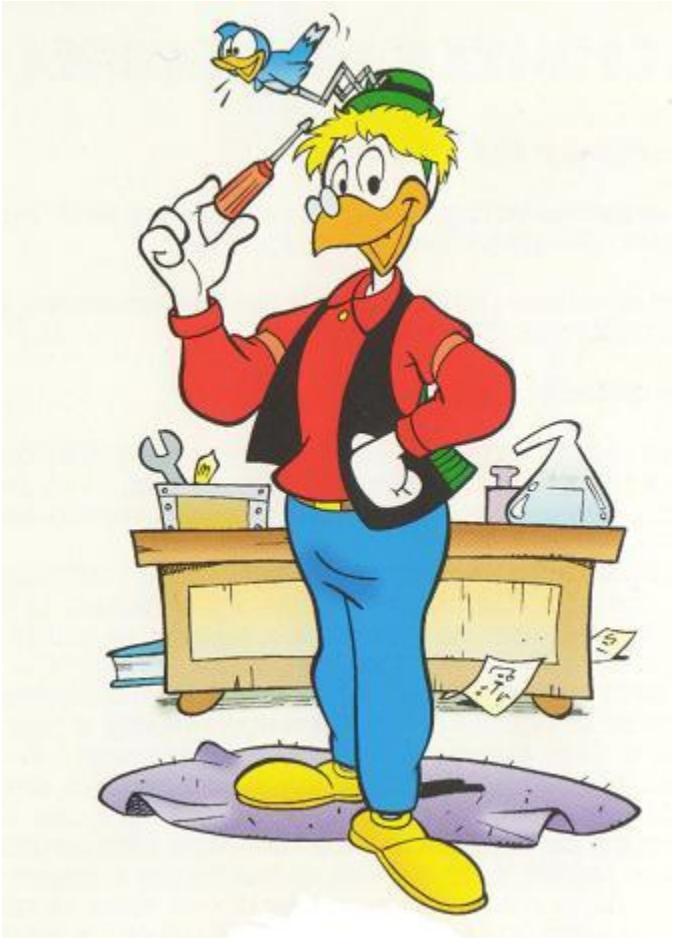
### Levosimendan

Levosimendan is a calcium sensitizer that improves cardiac contractility by binding to troponin-C in cardiomyocytes. It exerts significant vasodilatation mediated through ATP-sensitive potassium channels and has mild PDE inhibitory action. Levosimendan infusion in patients with acutely decompensated HF increases cardiac output and stroke volume and reduces pulmonary wedge pressure, systemic vascular resistance, and pulmonary vascular resistance. The haemodynamic response to levosimendan is maintained over several days. Levosimendan may be effective in patients with decompensated chronic HF. In that the inotropic effect is independent of β-adrenergic stimulation, it represents an alternative for patients on β-blocker therapy. Levosimendan treatment is associated with a slight increase in heart rate and a decrease in the BP, especially if a loading dose is administered.[235,237]

Levosimendan may be administered as a bolus dose (3–12 μg/kg) during 10 min followed by a continuous infusion (0.05–0.2 μg/kg/min for 24 h). The infusion rate may be increased once stability is confirmed. In patients with SBP $<$100 mmHg, the infusion should be started without a bolus dose to avoid hypotension.

**Class of recommendation IIa, level of evidence B**

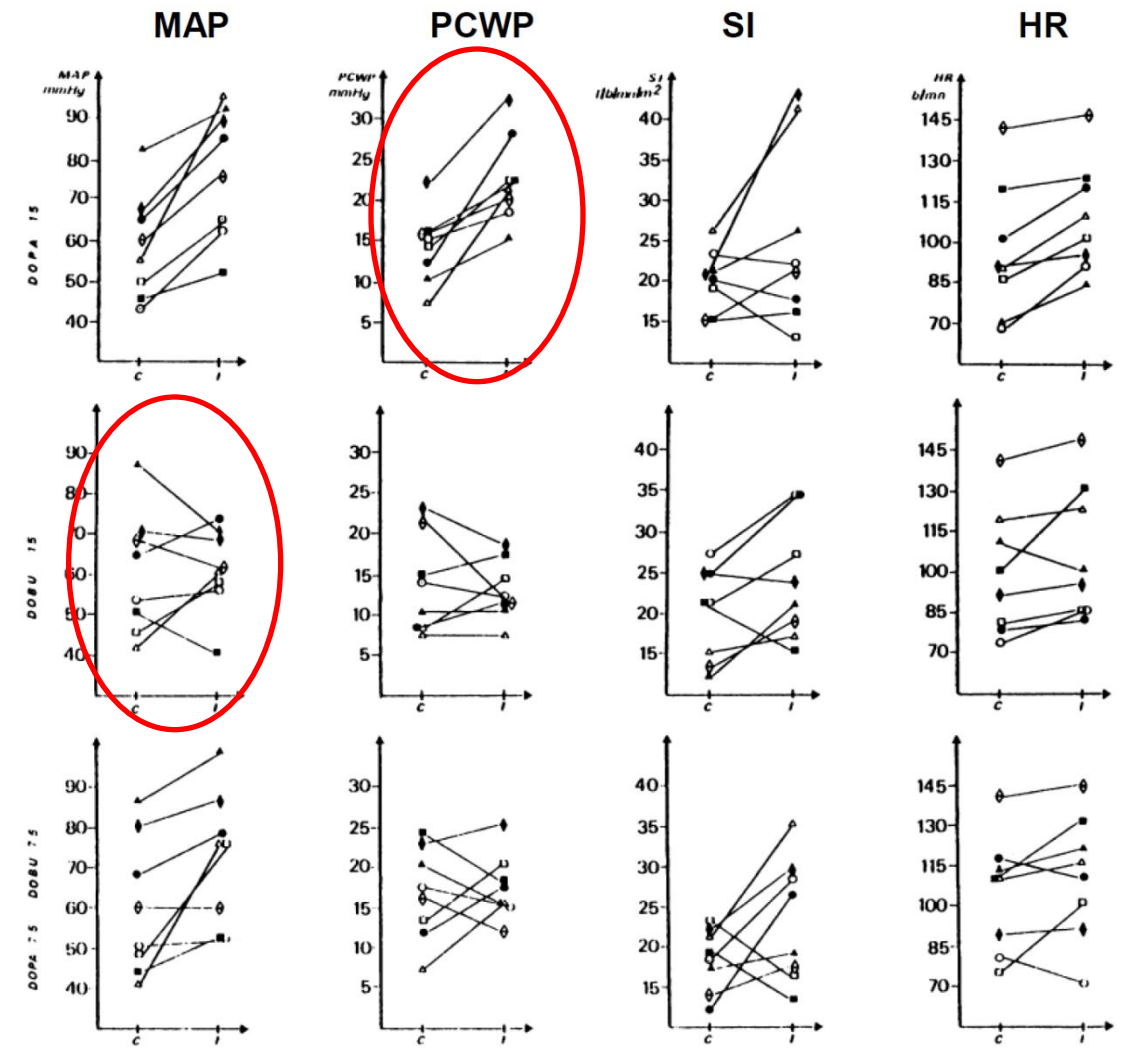
# Quali le evidenze?



# Combined hemodynamic effects of dopamine and dobutamine in cardiogenic shock

C Richard, JL Ricome, A Rimailho, G Bottineau and P Auzepy

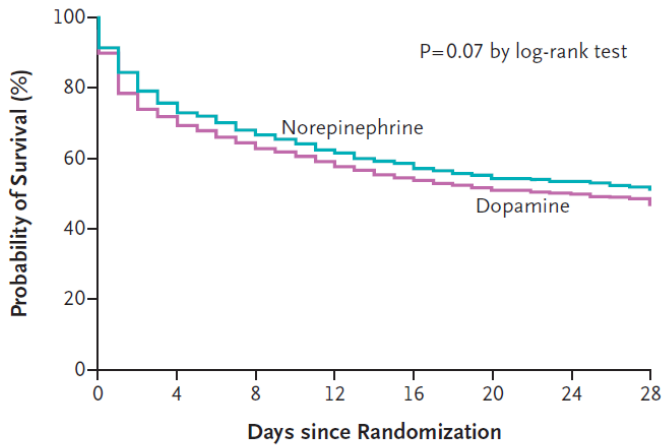
8 PZ




*Circulation 1983*

The NEW ENGLAND JOURNAL of MEDICINE
ESTABLISHED IN 1812 MARCH 4, 2010 VOL. 362 NO. 9

# Comparison of Dopamine and Norepinephrine in the Treatment of Shock




| No. at Risk | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Norepinephrine | 821 | 617 | 553 | 504 | 467 | 432 | 412 | 394 |
| Dopamine | 858 | 611 | 546 | 494 | 452 | 426 | 407 | 386 |

- Studio multicentrico randomizzato
- 1679 pazienti
- Tipi di shock:
  - Settico (62,2%)
  - Cardiogeno (16,7%)
  - Ipovolemico (15,7%)

| | dopamine | norepinephrine | |
|---|---|---|---|
| **Severe arrhythmias** | 52 pts (6.1%) | 13 pts (1.6%) | p<0.001 |

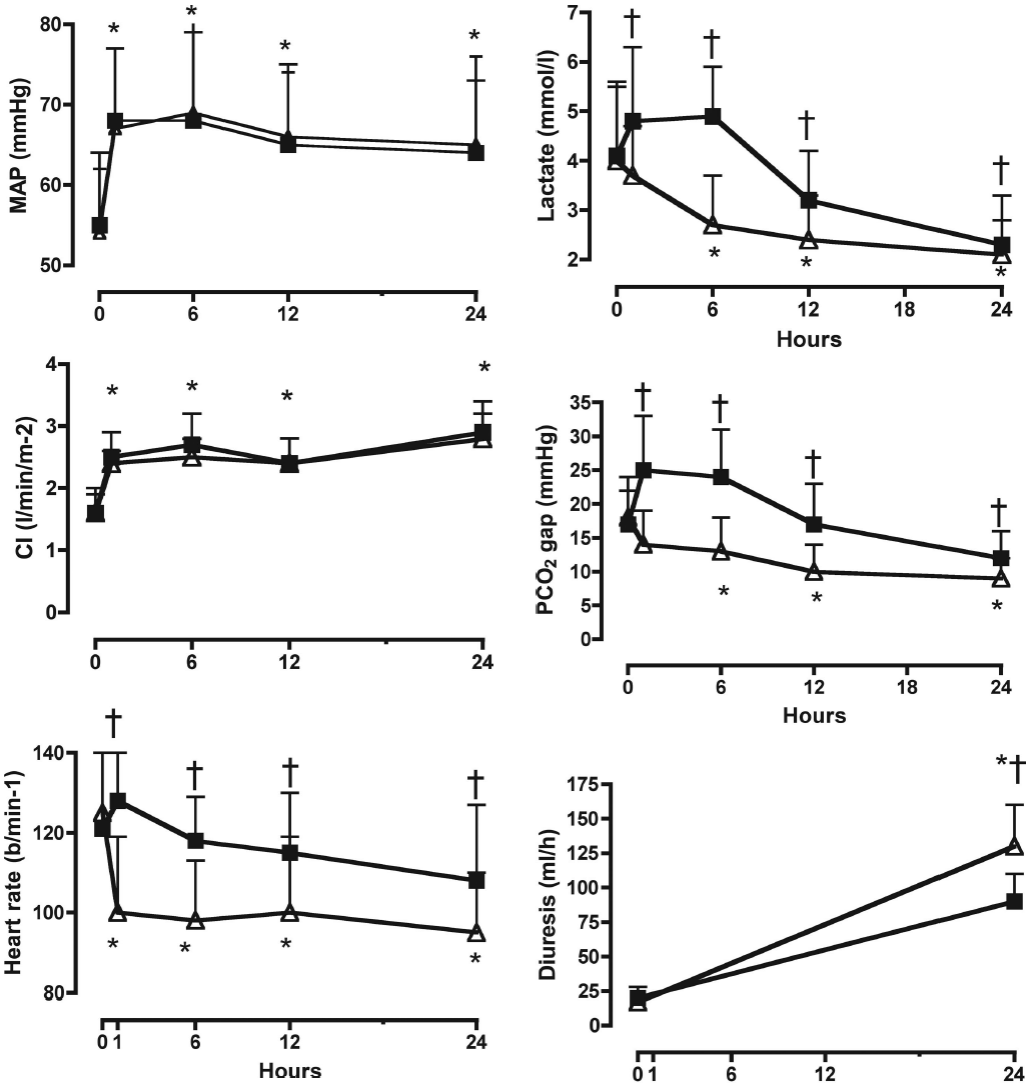
Dose dopamina: 20mcg/Kg/min

# Comparison of norepinephrine-dobutamine to epinephrine for hemodynamics, lactate metabolism, and organ function variables in cardiogenic shock. A prospective, randomized pilot study*

Bruno Levy, MD, PhD; Pierre Perez, MD; Jessica Perny, MD; Carine Thivilier, MD; Alain Gerard, MD

- Studio randomizzato 30 pazienti
- Eziologia non ischemica
- MAP ≤ 60 mmHg
  CI ≤ 2,2 l/min/mq
- Non responders a dobu+dopa

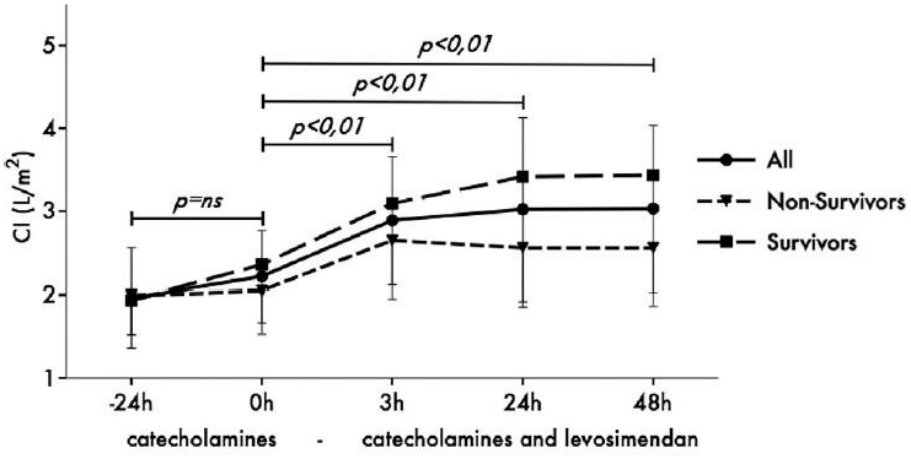
*Crit. Care M. 2011*



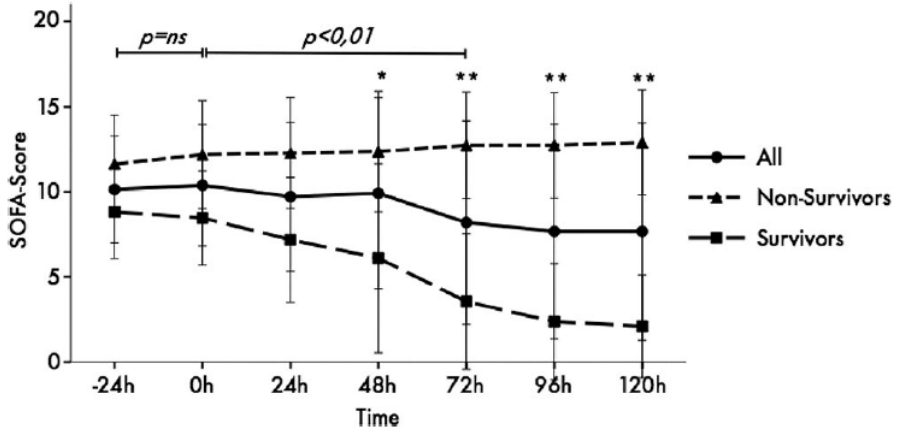
# Hemodynamic improvement following levosimendan treatment in patients with acute myocardial infarction and cardiogenic shock*

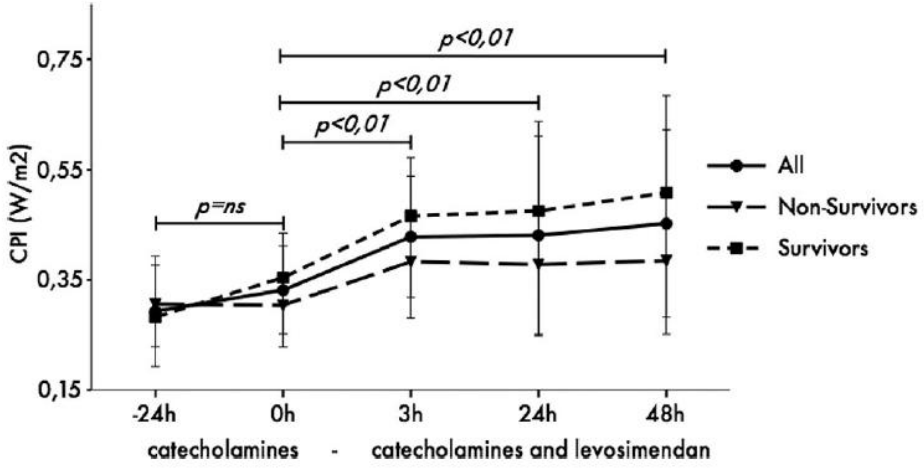
Martin A. Russ, MD; Roland Prondzinsky, MD; Arnd Christoph, MD; Axel Schlitt, MD; Ute Buerke, MD; Gerold Söffker, MD; Henning Lemm; Michael Swyter; Nikolas Wegener; Matthias Winkler, MD; Justin M. Carter, MRCP; Sebastian Reith, MD; Karl Werdan, MD; Michael Buerke, MD

*Crit Care Med 2007*




- Studio Ossevazionale
56 pazienti
- Shock post IMA
- Levosimendan vs Dobu-Nora

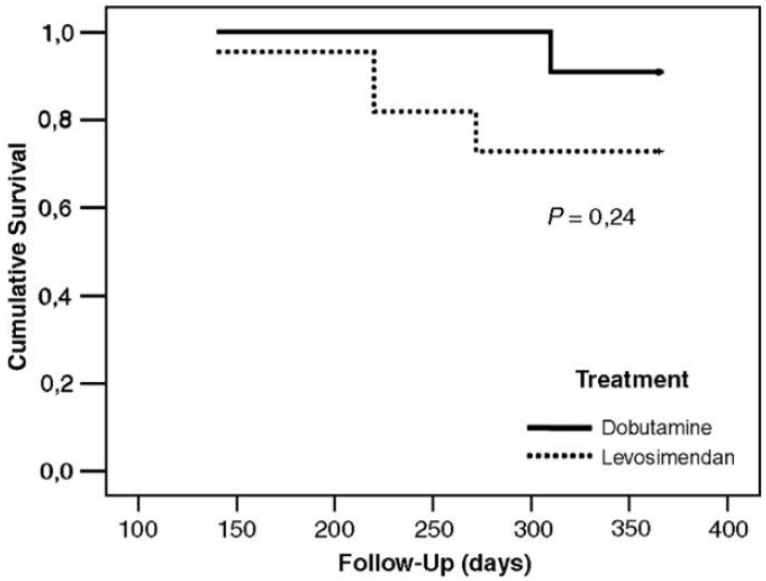


Effects of levosimendan versus dobutamine on long-term survival of patients with cardiogenic shock after primary coronary angioplasty

Sima Samimi-Fard [a], Martín J. García-González [a,*], Alberto Domínguez-Rodríguez [a], Pedro Abreu-González [b]

Studio Randomizzato
22 pazienti

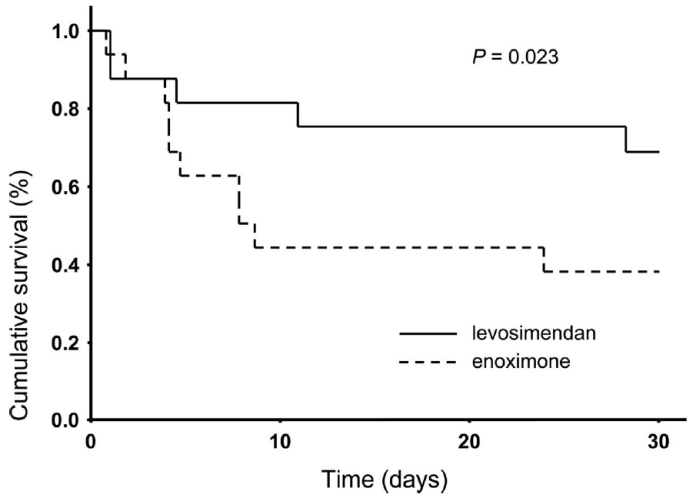



*Int J Cardiol 2008*

Levosimendan is superior to enoximone in refractory cardiogenic shock complicating acute myocardial infarction*

Joerg T. Fuhrmann, MD; Alexander Schmeisser, MD; Matthias R. Schulze, MD; Carsten Wunderlich, MD; Steffen P. Schoen, MD; Thomas Rauwolf, PhD; Christof Weinbrenner, MD; Ruth H. Strasser, MD

Studio Randomizzato
monocentrico:32 pazienti




*Crit Care Med 2008*

# Quali le evidenze?

- Studi “datati”
- Studi monocentrici, osservazionali, non Trials
- Pochi pazienti
- Pazienti eterogenei
- Dosaggi differenti
- Eziologia “mista”
- End Point disomogenei

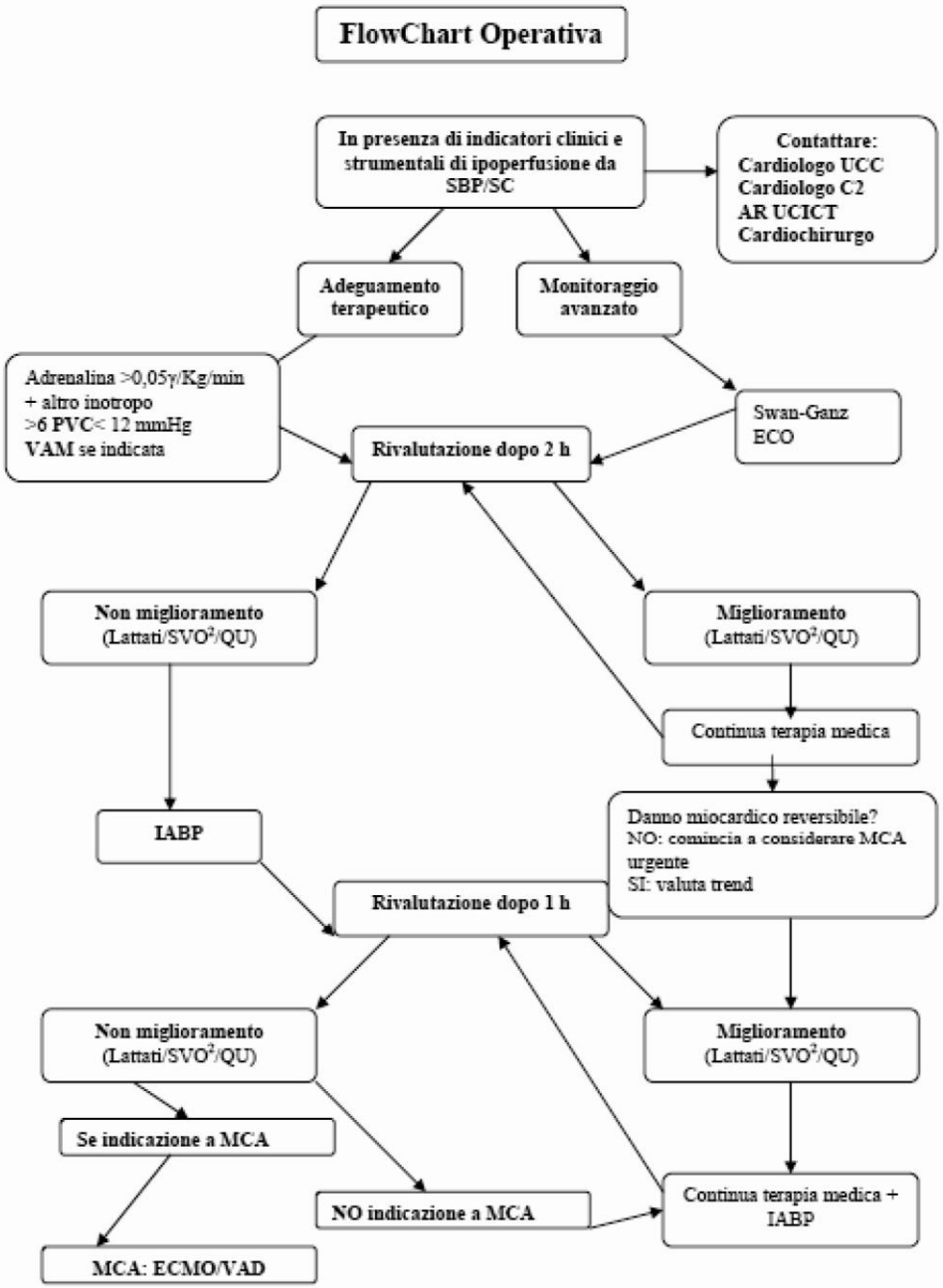
# Shock Cardiogeno:terapia farmacologica

- Dobutamina IIa B
- Dopamina IIb B
- Vasopressori IIb C
- Milrinone IIb B
- Levosimendan IIa B

*"il fine giustifica i mezzi"*

- Aumentare la pressione arteriosa sistemica sistolica e media
- Ridurre la pressione capillare polmonare
- Mantenere una adeguata perfusione degli organi vitali

# PROTOCOLLO OPERATIVO PER LO SHOCK CARDIOGENO

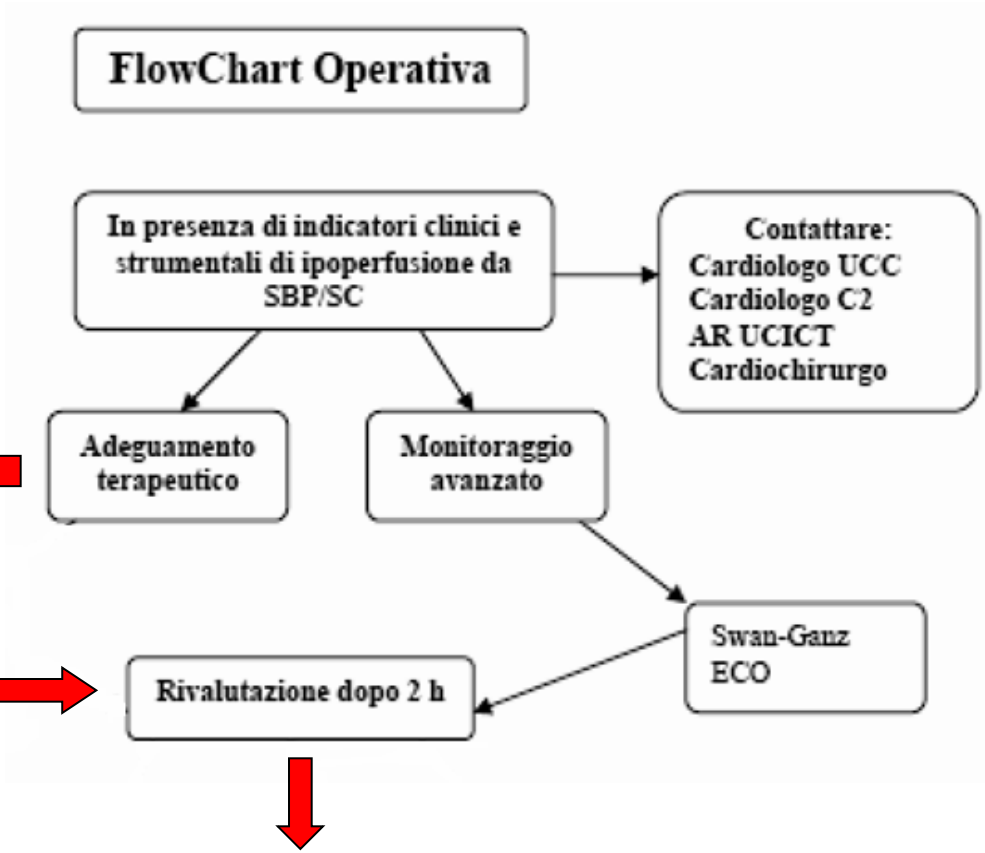



Dipartimento A. De Gasperis
AO Niguarda Cà Granda Milano

- identificare precocemente i pazienti affetti da SBP/SC
- mettere in atto con il timing più corretto tutte le misure di sostegno al circolo
- per impedire l'instaurarsi di danno d'organo o arrestarne la sua progressione

# PROTOCOLLO OPERATIVO PER LO SHOCK CARDIOGENO

Dipartimento A. De Gasperis
AO Niguarda Cà Granda Milano

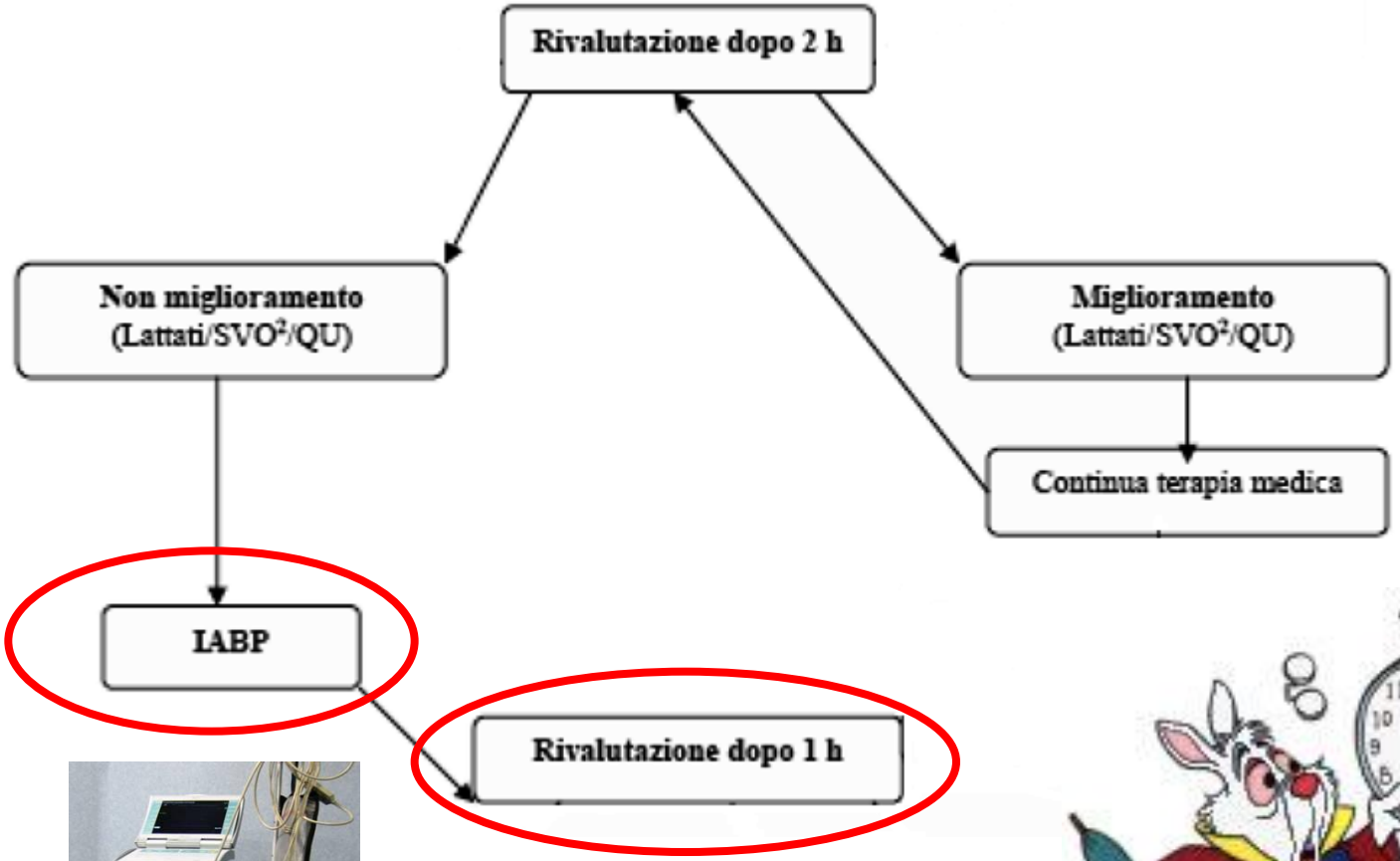


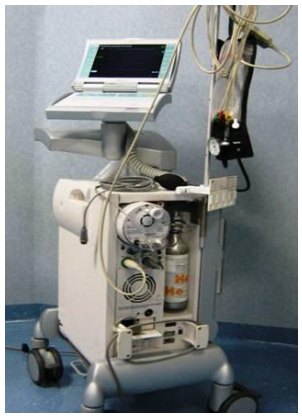

- Adrenalina 0,05mcg/Kg/min da titolare fino a raggiungere MAP> 65mmHg
- Eventualmente associare Dopamina LD (3mcg/Kg/min)
- Se pressione stabile si aggiunge SNP LD
- Diuretico in infusione continua

- Riduzione lattati
- SVO2 > 60%
- MAP > 65 mmHg
- PVC 6-10 mmHg
- Aumento Q.U.

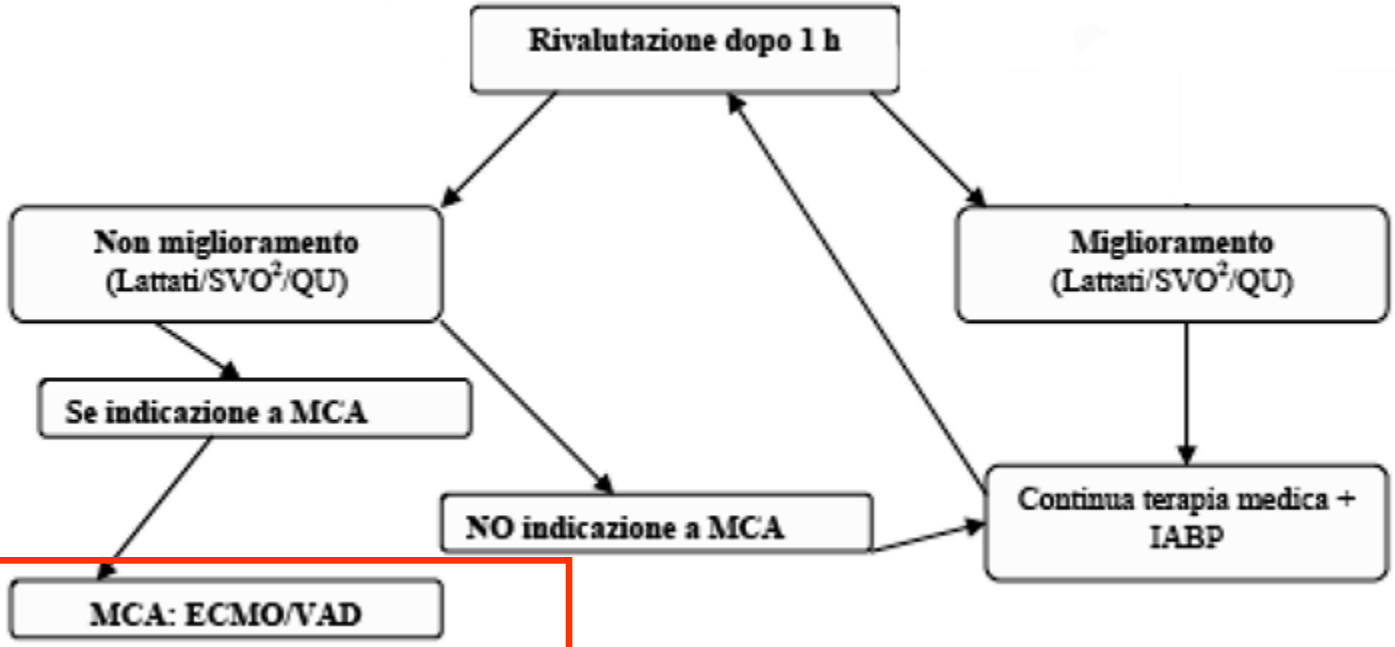
*Morici et al. Int J Cardiol 2011*

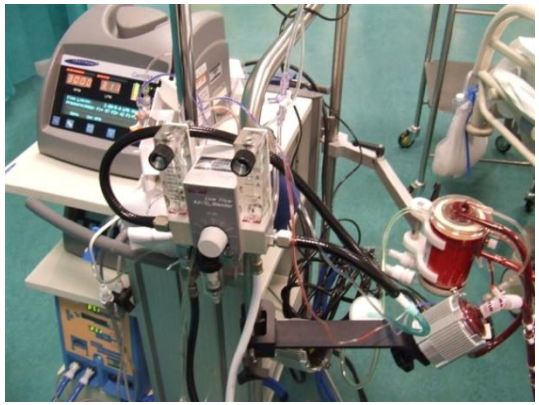
# PROTOCOLLO OPERATIVO PER LO SHOCK CARDIOGENO



# PROTOCOLLO OPERATIVO PER LO SHOCK CARDIOGENO



**DOOR TO VAD:**

**5-6H !!!!**

# Take Home Message


Lo Shock Cardiogeno rappresenta una condizione patologica ad elevata mortalità. La maggior parte dei pazienti sviluppa un quadro di shock cardiogeno durante il ricovero

Il monitoraggio clinico continuo del paziente, degli organi bersaglio e della perfusione tissutale permettono di riconoscere precocemente uno stato di pre-shock cardiogeno e di impostare tempestivamente una corretta strategia terapeutica

# Take Home Message

Gli studi clinici randomizzati sono di scarso aiuto nella scelta dell'inotropo. Di conseguenza il grado di raccomandazione e il livello di evidenza per questi farmaci non sono così "convincenti"

Indipendentemente dal tipo di inotropo, è essenziale iniziare precocemente il trattamento, e verificare il raggiungimento o meno degli obiettivi emodinamici/clinici prefissati


Grazie per l'attenzione