31 GIORNATE CARDIOLOGICHE TORINESI

TURIN
October
24th-26th
2019

MANAGEMENT FOR CARDIOGENIC SHOCK: CLINICAL SESSION

Management of cardiogenic shock: watchful escalating approach or early support?

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Conflict of interest

nessuno

AZIENDA OSPEDALIERO - UNIVERSITARIA
Città della Salute e della Scienza di Torino

1. diagnosi

↕

2. caratterizzazione
(eziologia, gravità, ecografica, emodinamica,
prospettive di trattamento a medio-lungo termine)

↕

3. trattamento

1. diagnosi

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

The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)

Table 1. Pragmatic and Clinical Trial Definitions of CS

Clinical Definition	SHOCK Trial[9]*	IABP-SHOCK II[11]†	ESC HF Guidelines[15]
Cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Support to maintain SBP ≥90 mm Hg AND End-organ hypoperfusion (urine output <30 mL/h or cool extremities) Hemodynamic criteria: CI of ≤2.2 L·min^{-1}·m^{-2} AND PCWP ≥15 mm Hg	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Catecholamines to maintain SBP >90 mm Hg AND Clinical pulmonary congestion AND Impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output <30 mL/h, or lactate >2.0 mmol/L)	SBP <90 mm Hg with adequate volume and clinical or laboratory signs of hypoperfusion Clinical hypoperfusion: Cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure Laboratory hypoperfusion: Metabolic acidosis, elevated serum lactate, elevated serum creatinine

CI indicates cardiac index; CS, cardiogenic shock; ESC, European Society of Cardiology; HF, heart failure; IABP-SHOCK II, Intraaortic Balloon Pump in Cardiogenic Shock II; LV, left ventricular; MI, myocardial infarction; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure; and SHOCK, Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock.

*In setting of MI complicated by predominantly LV dysfunction.

†In setting of acute MI.

Letter to the Editor

Cardiogenic shock: How to overcome a clinical dilemma. Unmet needs in Emergency medicine

Nuccia Morici [a,*], Alice Sacco [a], Roberto Paino [a], Jacopo Andrea Oreglia [a], Maurizio Bottiroli [a], Michele Senni [b], Michele Nichelatti [c], Paolo Canova [b], Claudio Russo [a], Andrea Garascia [a], Silvio Kulgmann [a], Maria Frigerio [a], Fabrizio Oliva [a]

Inclusion criteria	• Age ≥ 18, males and females Eligible patients have to fit at least TWO of the following criteria/items: • SBP < 100 mm Hg or MAP < 60 mm Hg, after fluid challenge (at least 1000 ml of a crystalloid solution or 500 ml of a colloidal solution with right atrial pressure >4 mm Hg) or with a CVP > 12 mm Hg or WCP > 14 mm Hg. • Mixed venous oxygen saturation <60% • Arterial lactates > 2 • Oliguria <0.5 ml/kg/h

of their chronic disease. Treatment of patients with chronic HF presenting in CS can differ substantially from the treatment of other types of CS because the hemodynamic condition and neurohormonal milieu are often strikingly different. Patients with HF often have profound upregulation of vasoconstrictor substances such as angiotensin II, endothelin-1, and norepinephrine.[61,62] Among patients who had cardiac sur-

2. caratterizzazione

- eziologia

Chronic HF can present in an acute decompensated state and may account for up to 30% of CS cases.[60]

The natural history of HF is a progressive decline in ventricular function as compensatory remodeling ultimately fails and patients present with recurrent episodes of acutely decompensated HF and ultimately CS owing to advanced HF (CS-HF). A recent analysis of the Interagency for Mechanical Circulatory Support (INTERMACS) registry identified that 52.5% of patients with advanced HF referred for surgical left ventricular (LV) assist device (LVAD) placement present with CS-HF defined as INTERMACS levels 1 or 2 HF[6]. By 2030, 8 million people in the United States alone will be diagnosed with HF[7]. Collectively, these data identify CS as a persistent clinical problem and further suggest that the distribution of CS patients may be shifting from CS-AMI to CS-HF over the next decade.

AMI	miocardite	ADHF
↓	↓	↓
noradrenalina Impella	no amine ECMO-Vent	inodilatazione IAoBP

Contemporary Management of Cardiogenic Shock
A Scientific Statement From the American Heart Association
van Diepen et al *Circulation.* 2017;136:00–00.

2. caratterizzazione

- gravità

SCAI clinical expert consensus statement on the classification of cardiogenic shock

This document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019

Cardiogenic Shock Classification to Predict Mortality in the Cardiac Intensive Care Unit

Jacob C. Jentzer, MD,[a,b] Sean van Diepen, MD, MSc,[c] Gregory W. Barsness, MD,[a] Timothy D. Henry, MD,[d] Venu Menon, MD,[e] Charanjit S. Rihal, MD, MBA,[a] Srihari S. Naidu, MD,[f] David A. Baran, MD[g]

E Extremis

Hypoperfusion WITH deterioration AND refractory shock

mortalità intraospedaliera 70%

Stage E "Extremis". A patient with circulatory collapse, frequently (but not always) in refractory cardiac arrest with ongoing cardiopulmonary resuscitation (CPR) or are being supported by multiple simultaneous acute interventions including ECMO-facilitated CPR. These are patients with multiple clinicians at bedside laboring to address multiple simultaneous issues related to the lack of clinical stability of the patient.

D Deteriorating

Hypoperfusion WITH deterioration NOT refractory shock

mortalità intraospedaliera 35%

Stage D "Deteriorating or Doom". A patient that is similar to category C but is getting worse. They have failure to respond to initial interventions.

C Classic

Hypoperfusion WITHOUT deterioration

mortalità intraospedaliera 15%

Stage C "Classic" Cardiogenic Shock. A patient that manifests with hypoperfusion that requires intervention (inotrope, pressor or mechanical support, ECMO) beyond volume resuscitation to restore perfusion. These patients typically present with relative hypotension.

lactate > 2

B Beginning

Stage B "Beginning" Cardiogenic Shock. A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.

SVcO < 65%

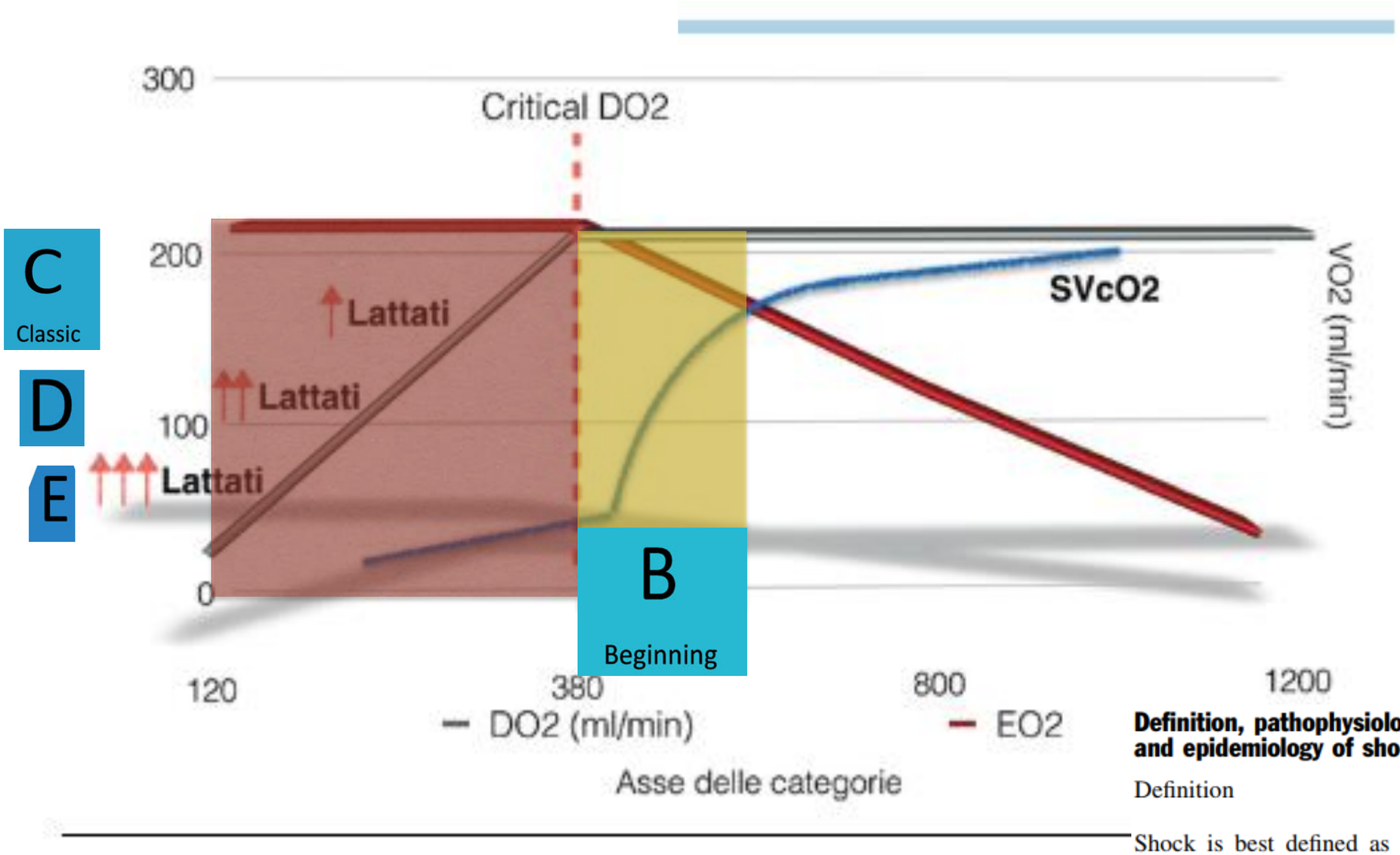
A At Risk

Stage A "At Risk". A patient who is not currently experiencing signs or symptoms of cardiogenic shock, but is at risk for its development. These patients may include those with acute myocardial infarction, acute and/or acute on chronic heart failure symptoms.

Intensive Care Med (2014) 40:1795–1815
DOI 10.1007/s00134-014-3525-z

Maurizio Cecconi
Daniel De Backer
Massimo Antonelli
Richard Beale
Jan Bakker
Christoph Hofer
Roman Jaeschke
Alexandre Mebazaa
Michael R. Pinsky
Jean Louis Teboul
Jean Louis Vincent
Andrew Rhodes

Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine



Definition, pathophysiology, features and epidemiology of shock

Definition

Shock is best defined as a life-threatening, generalized form of acute circulatory failure associated with inadequate oxygen utilization by the cells. It is a state in which the circulation is unable to deliver sufficient oxygen to meet the demands of the tissues, resulting in cellular dysfunction. The result is cellular dysoxia, i.e. the loss of the physiological independence between oxygen delivery and oxygen consumption, associated with increased lactate levels. Some clinical symptoms suggest an impaired microcirculation, including mottled skin, acrocyanosis, slow capillary refill time and an increased central-to-toe temperature gradient.

2. caratterizzazione

- ecografica: funzione VD

European Journal of Heart Failure (2016)
doi:10.1002/ejhf.504

Prognostic incremental role of right ventricular function in acute decompensation of advanced chronic heart failure

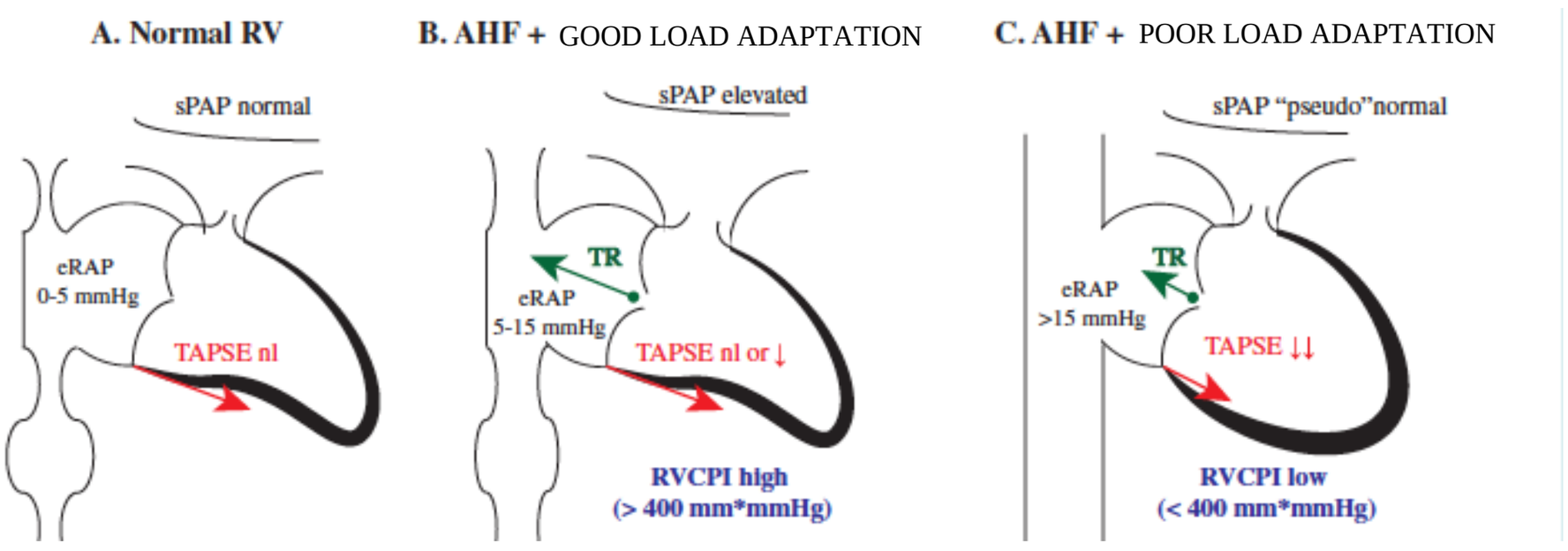
Simone Frea[1]*, Stefano Pidello[1], Virginia Bovolo[1], Cristina Iacovino[1], Erica Franco[2], Francesco Pinneri[2], Alessandro Galluzzo[1], Alessandra Volpe[1], Massimiliano Visconti[1], Andrea Peirone[1], Mara Morello[1], Serena Bergerone[1], and Fiorenzo Gaita[1]

European Journal of Heart Failure (2016) 18, 573–575
doi:10.1002/ejhf.533

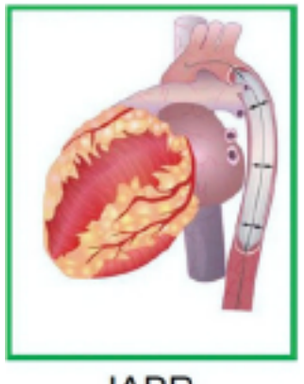
EDITORIAL COMMENT

Advanced heart failure: look right to prognosticate right!

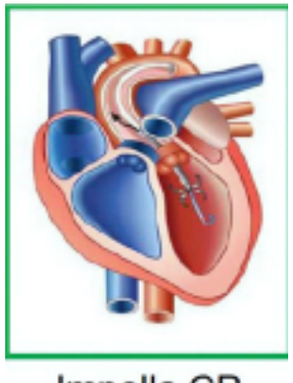
Lucas N.L. Van Aelst[1,2,3], Jelena Čelutkienė[4], and Alexandre Mebazaa[3,5,6]*



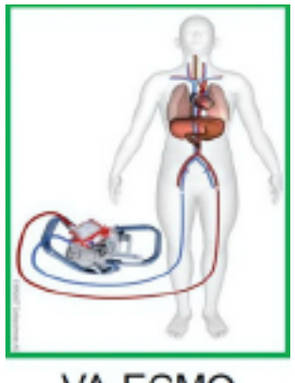
INTERMACS profile 1 or 2 at 48 h	11 (6.0%)	16 (19.5%)	0.002



IABP



Impella CP



VA-ECMO

2. caratterizzazione

- emodinamica

SVR ⇢ noradrenalina vs. inotropo vs. inodilatazione

CPO ⇢ prognosi

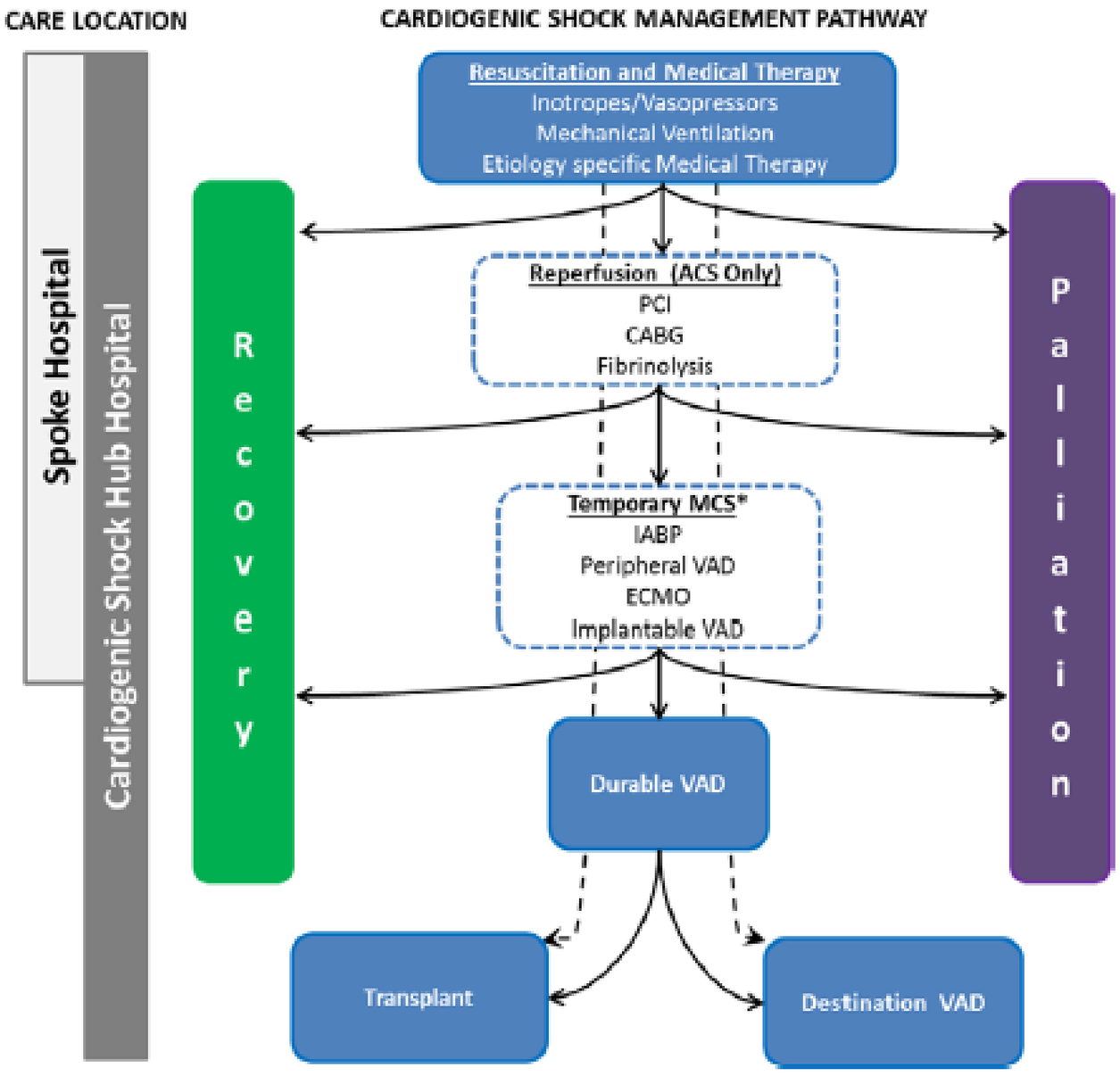
PAPi ⇢ risposta a IAoBP

Standardized Team-Based Care for Cardiogenic Shock

Behnam N. Tehrani, MD,[a] Alexander G. Truesdell, MD,[a,b] Matthew W. Sherwood, MD,[a] Shashank Desai, MD,[a] Henry A. Tran, MD,[a] Kelly C. Epps, MD,[a] Ramesh Singh, MD,[a] Mitchell Psotka, MD, PhD,[a] Palak Shah, MD,[a] Lauren B. Cooper, MD,[a] Carolyn Rosner, NP,[a] Anika Raja, BS,[a] Scott D. Barnett, PhD,[a] Patricia Saulino, RN, MPA,[a] Christopher R. deFilippi, MD,[a] Paul A. Gurbel, MD,[a] Charles E. Murphy, MD,[a] Christopher M. O'Connor, MD[a]

2. caratterizzazione

- prospettive di trattamento a medio-lungo termine



dalla teoria alla pratica

P. L.

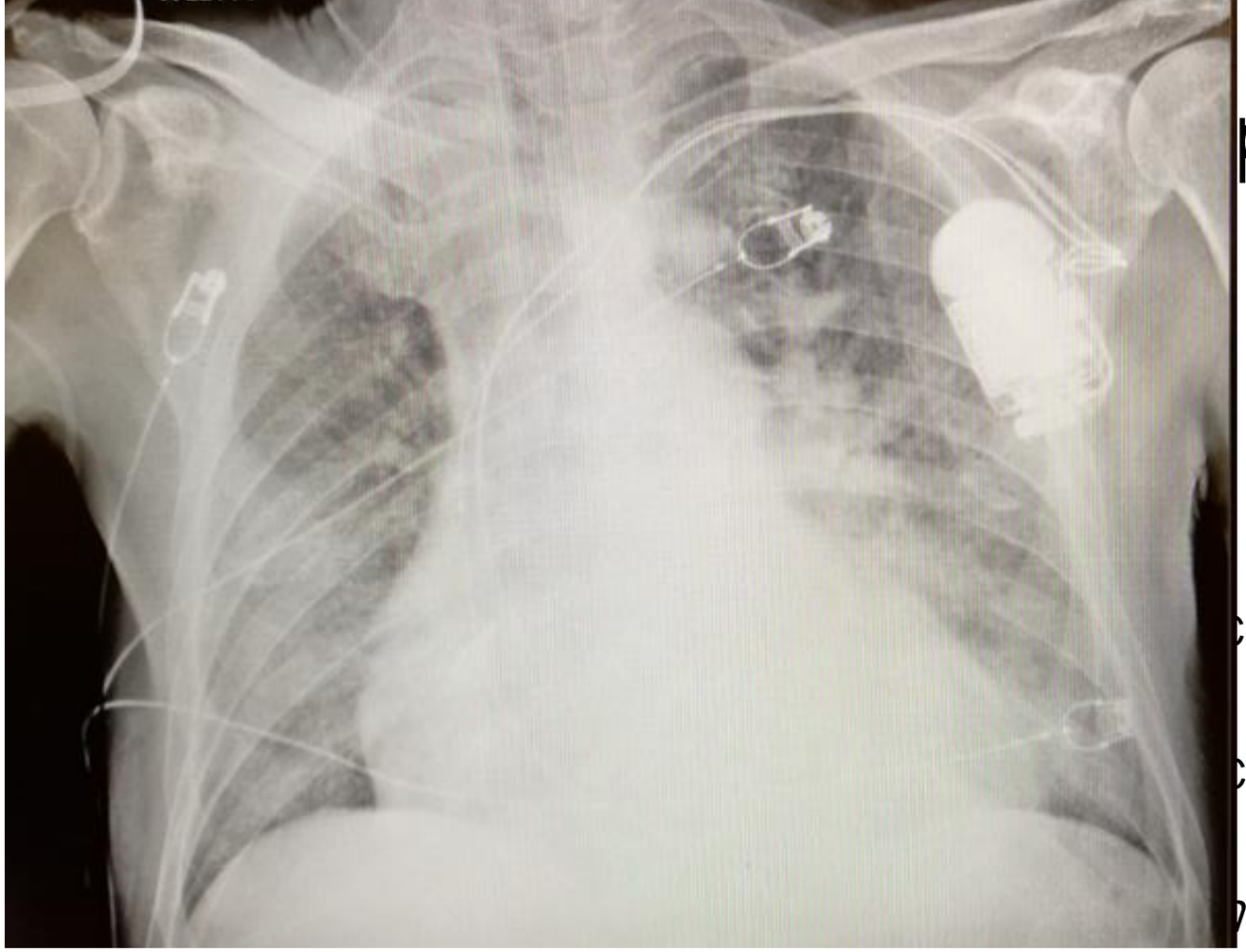
66 anni

2004 STEMI anteriore -> PCI IVA II
2013 STEMI anteriore e inferiore -> PCI IVA II e DX I; FE 33%
2014 ICD prev Iaria

Da 12/2017 NYHA III *(tlv. oppressione toracica)* in TMO

15/5/2018 SC wet and cold (pre-shock), NYHA IV, PAS 100 mmHg, FA de novo, bil tot 1.3, AST 286, ALT 576, crea 1.15 -> shock cardiogeno dopo 24 ore, IOT, dobutamina e levosimendan x 2, TC torace TEP (trombolisi: 33 mm -> 18 mm), sepsi CVC correlata (St, Haemolyticus), TVS x 3

66 anni



pratica

cinetica dilatativa

co

(inotropi da 50 giorni circa)

INTERMACS 2: PA 70/50 mmHg, cachessia, cute fredda e marezzata, sopore, oligoanuria, crepitii medio-basali, giugulari distese a 45°, edemi fino al ginocchio

ECG FA 110/min, Q in inferiore, QS V1-V5, bassi voltaggi su periferiche

Lab: **lattati 4.8**, Hb 10.5 mg/dl, hs-TnT 336 (in aumento), NTproBNP 34.841, **AST/ALT 512/610, INR 2.38 spontaneo, bil tot 6.6**, crea 1.12 (crea 0.45 48h prima)

Eco: VS DTD 67 mm, FE 23%, **IM 4+, IAo 2+**, VD DTD 52 mm, IT 4+, TAPSE 10 mm, grad VA 35 mmHg, VCI 32 mm fissa (eRVSWI 350 mm mmHg, ePAPi 1.5)

IC Fick 1.2, SVcO2 15%, PVC 26, SVR 1220, CPO 0.26

CS-ADHF in CAD

Hypoperfusion WITH deterioration
NOT refractory shock

D
Deteriorating

Stage D "Deteriorating or Doom". A patient that is similar to category C but is getting worse. They have failure to respond to initial interventions.

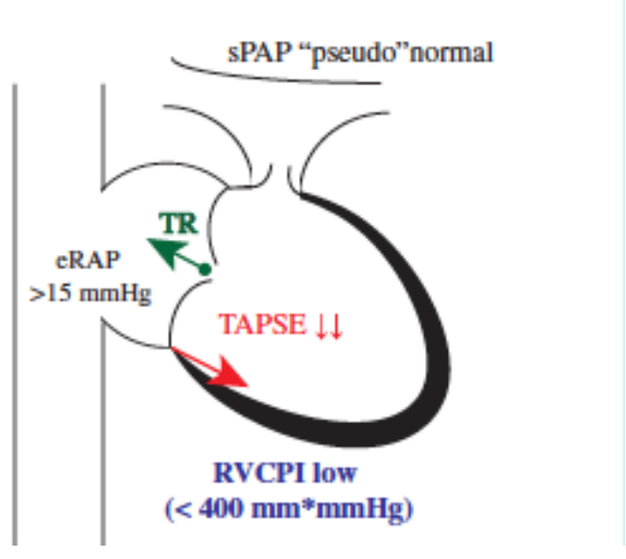
Comparison of Dopamine and Norepinephrine in the Treatment of Shock

Daniel De Backer, M.D., Ph.D., Patrick Biston, M.D., Jacques Devriendt, M.D., Christian Madl, M.D., Didier Chochrad, M.D., Cesar Aldecoa, M.D., Alexandre Brasseur, M.D., Pierre Defrance, M.D., Philippe Gottignies, M.D., and Jean-Louis Vincent, M.D., Ph.D., for the SOAP II Investigators*

Table 2 **INTERMACS Patient Profiles (20)**

Level	Definition	Description
2	Progressive decline	"Sliding fast"

C. AHF + | POOR LOAD ADAPTATION



SVR > 1200 PAPi stimato 1.5

disfunzione VD severa [illegible] sia, necessità di SVAo

Durable VAD

1. diagnosi

↓

2. caratterizzazione

(eziologia, gravità, ecografica, emodinamica,
prospettive di trattamento a medio-lungo termine)

↓

3. trattamento