TRAITEMENT NON MÉDICAMENTEUSE DU CHOC CARDIOGÉNIQUE POST - IDM

PR LEVY BRUNO
RÉANIMATION MÉDICALE NANCY
INSTITUT DU CŒUR ET DES VAISSEAUX
GROUPE CHOC, INSERM
FRANCE

DÉFINITION

- Pression artérielle systolique < 90 mmHg pendant plus de 30 min
- Vasopresseur pour pour obtenir une pression artérielle systolique ≥ 90 mmHg
- Index cardiaque <1,8 l/min/m^2 ou l'utilisation de vasopresseur/inotropes
- Une augmentation des pressions de remplissage ventriculaire gauche
- Altération de la perfusion tissulaire:
  - Trouble de conscience
  - Oligurie
  - Extrémités froides
  - Augmentation du lactate

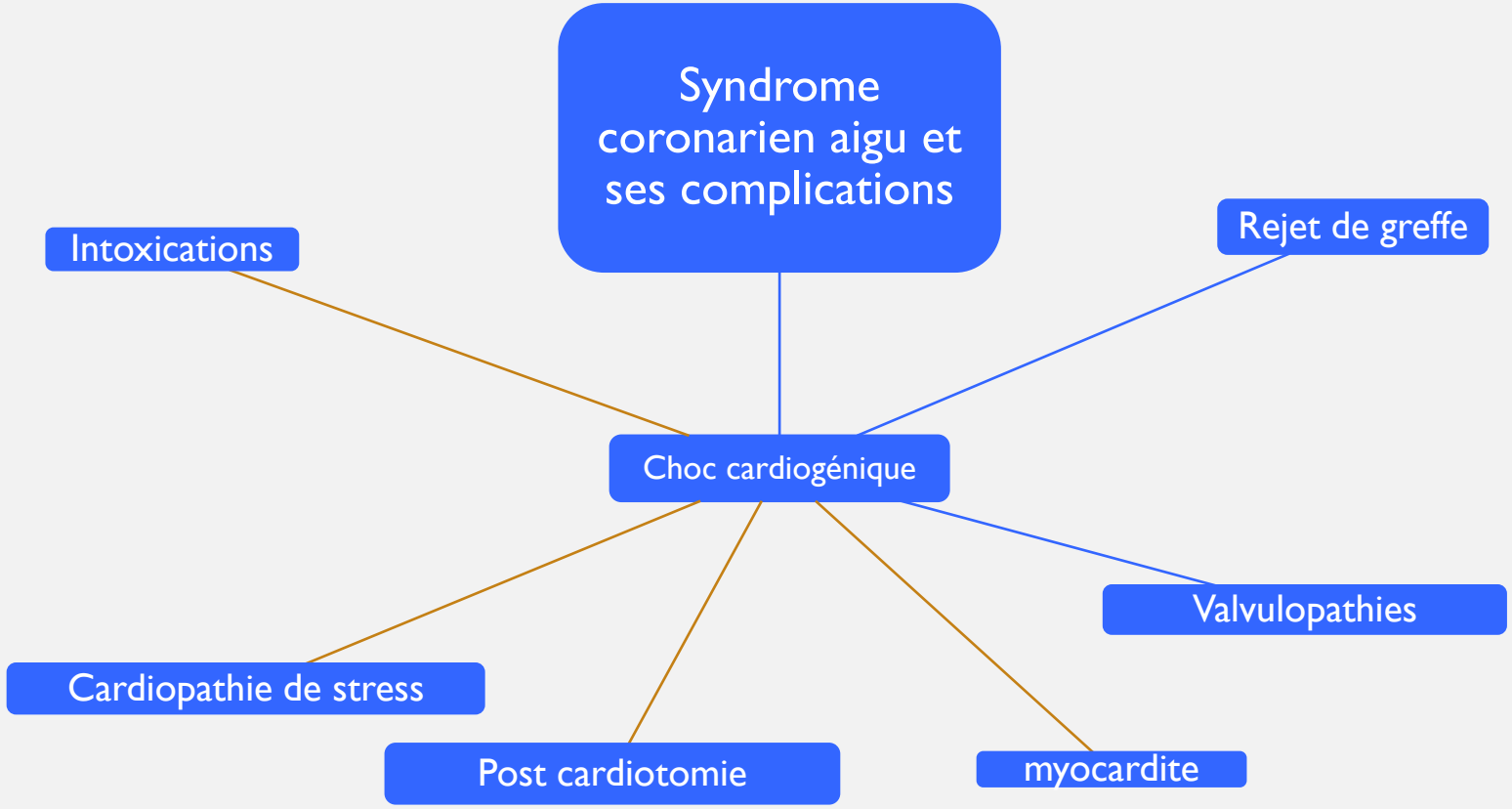
Thiele H et al. **European Heart Journal** 2015

CONFIRMATION PARACLINIQUE

- Par cathérisme de Swan-Ganz historiquement
  - Index cardiaque
  - Pression artérielle pulmonaire d'occlusion
  - Résistances vasculaires systémiques et pulmonaires
- Par thermodilution transpulmonaire
- Par échocardiographie

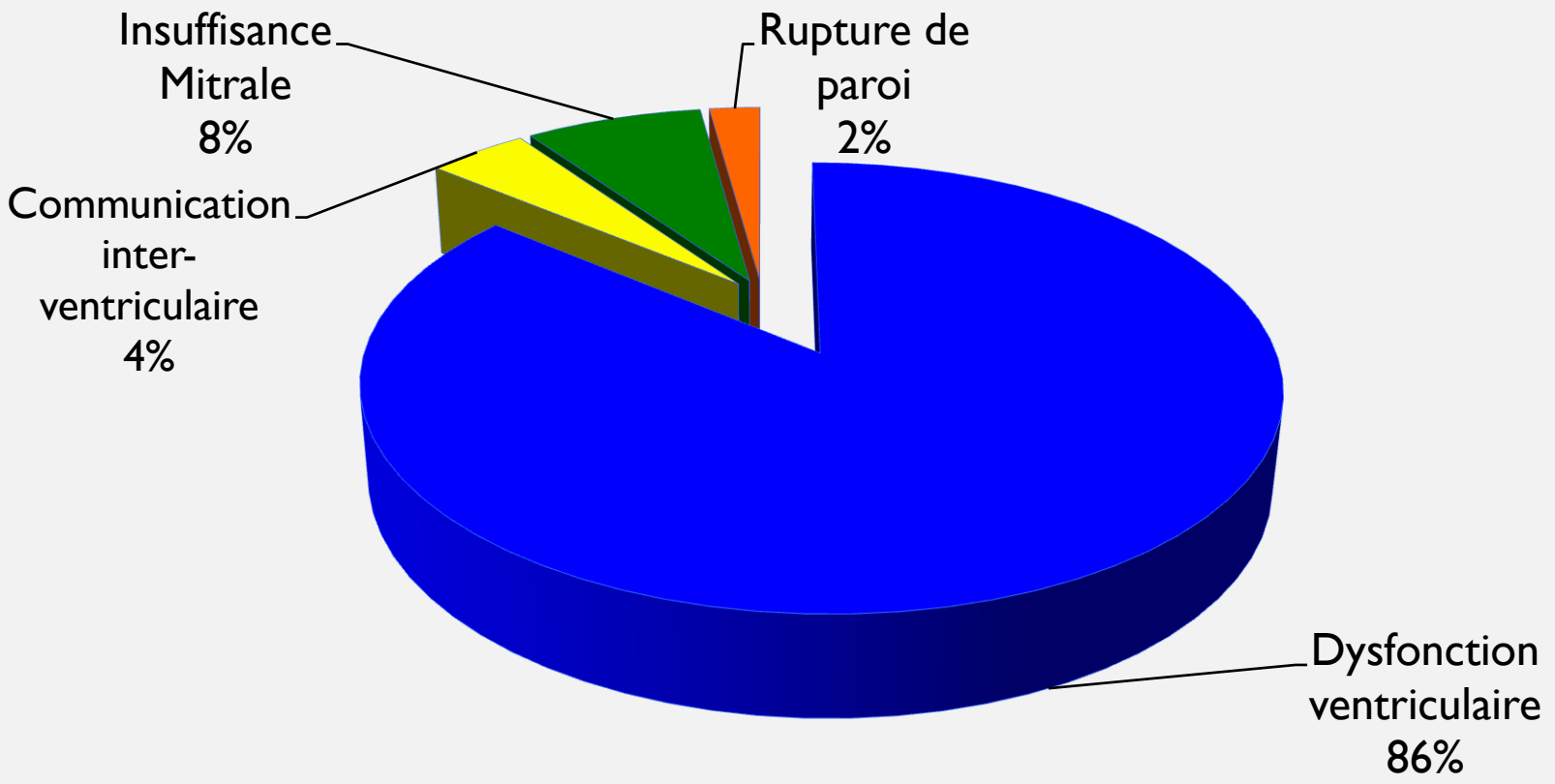
Gabriele V et al. **J Am Soc Echocardiogr** 2014;27:683.e1-683.e33.

CONSTELLATION D'ÉTIOLOGIES



Aucune donnée épidémiologique de qualité…

ETIOLOGIES AU SEIN DU SCA

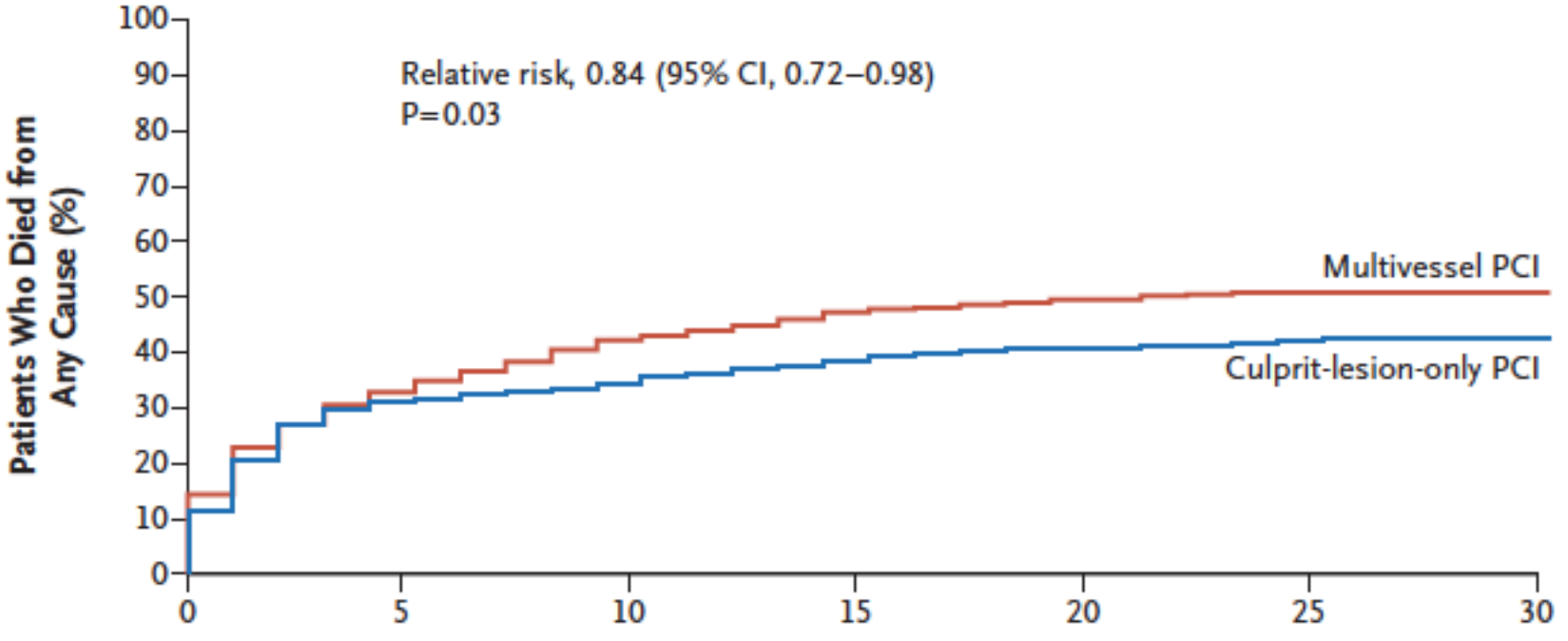


Hochman JS et al. **J Am Coll Cardiol** 2000;36:1063–70

ORIGINAL ARTICLE

PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock

B **Death from Any Cause**



No. at Risk							
Multivessel PCI	341	229	197	179	170	166	165
Culprit-lesion-only PCI	344	237	226	211	203	198	193

REVASCULARISATION CORONAROGRAPHIQUE

TABLE 4. MORTALITY AMONG STUDY PATIENTS.*

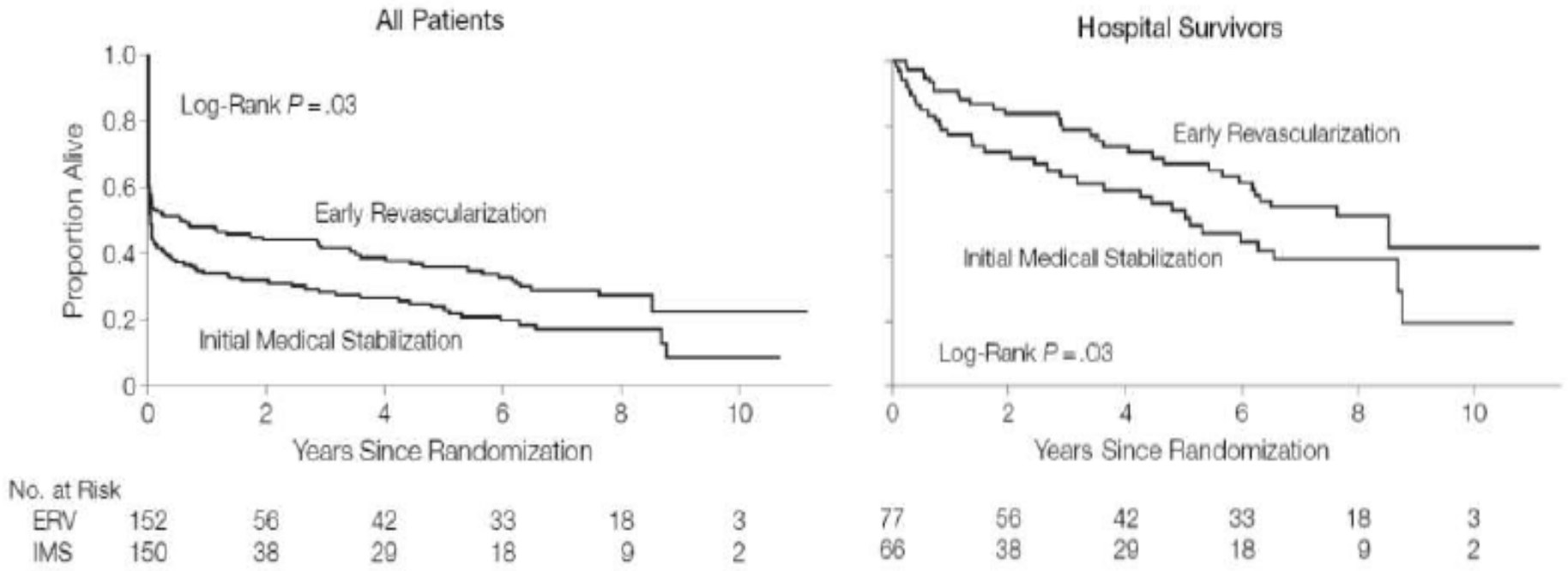
OUTCOME AND SUBGROUP	REVASCULARIZATION	MEDICAL THERAPY	DIFFERENCE BETWEEN GROUPS (95% CI)	RELATIVE RISK (95% CI)	P VALUE
	percent (number in subgroup)		percent		
30-day mortality					
Total	46.7 (152)	56.0 (150)	−9.3 (−20.5 to 1.9)	0.83 (0.67 to 1.04)	0.11
Age <75 yr	41.4 (128)	56.8 (118)	−15.4 (−27.8 to −3.0)	0.73 (0.56 to 0.95)	0.01†
Age ≥75 yr	75.0 (24)	53.1 (32)	+21.9 (−2.6 to 46.4)	1.41 (0.95 to 2.11)	
6-mo mortality‡					
Total	50.3 (151)	63.1 (149)	−12.8 (−23.2 to −0.9)	0.80 (0.65 to 0.98)	0.027
Age <75 yr	44.9 (127)	65.0 (117)	−20.1 (−31.6 to −7.1)	0.70 (0.56 to 0.89)	0.003†
Age ≥75 yr	79.2 (24)	56.3 (32)	+22.9 (0.7 to 46.6)	1.41 (0.97 to 2.03)	

Hochman JS et al. **N Engl J Med** 1999;341:625-34

Early Revascularization and Long-term Survival in Cardiogenic Shock Complicating Acute Myocardial Infarction

Judith S. Hochman, MD
Lynn A. Sleeper, ScD
John G. Webb, MD
Vladimir Dzavik, MD
Christopher E. Buller, MD
Philip Aylward, MD
Jacques Col, MD
Harvey D. White, DSc
for the SHOCK Investigators

JAMA, June 7, 2006—Vol 295, No. 21



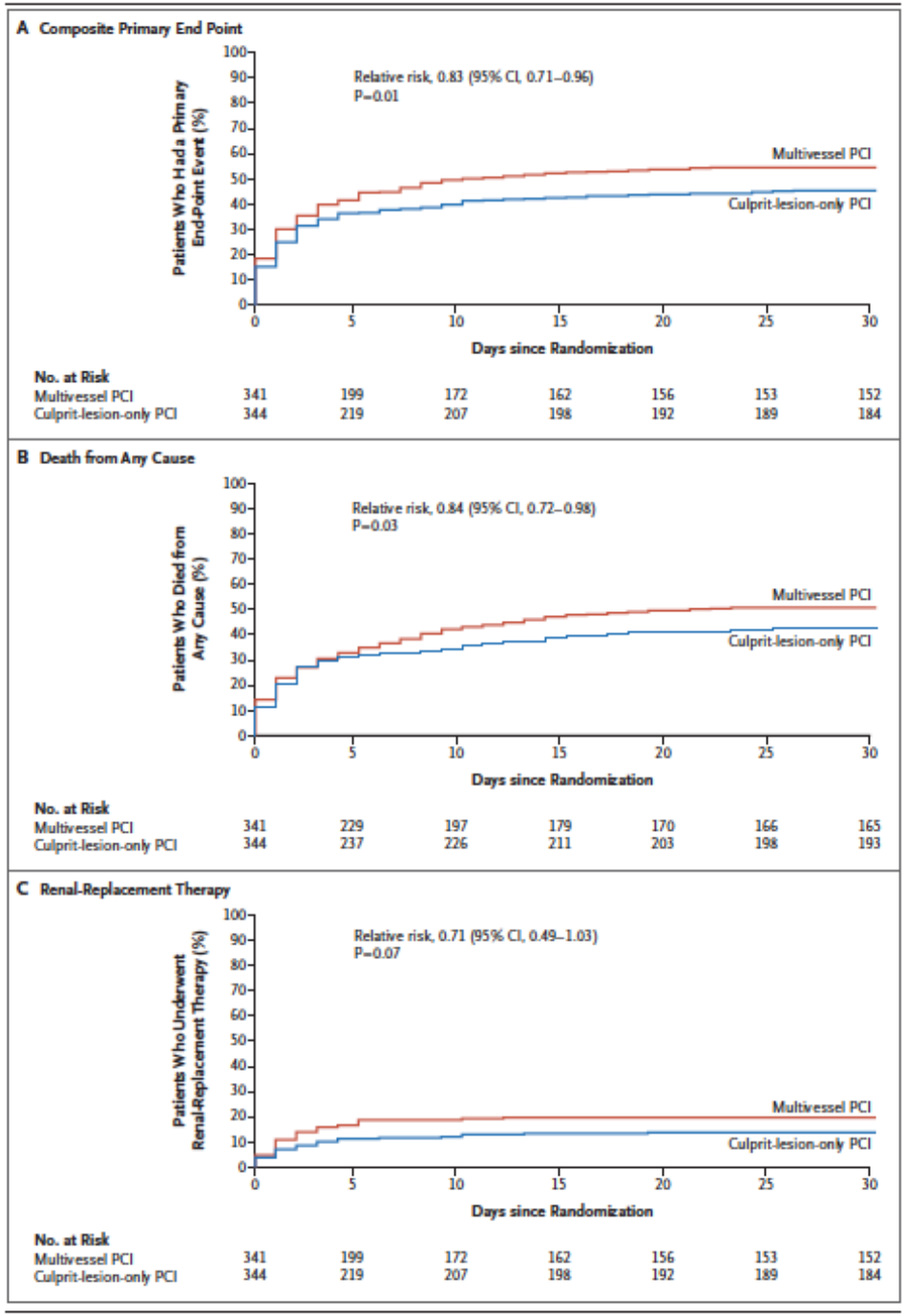
No. at Risk						
ERV	152	56	42	33	18	3
IMS	150	38	29	18	9	2

77	56	42	33	18	3
66	38	29	18	9	2

ORIGINAL ARTICLE

PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock

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



< 2 hours

STEMI or high-risk ECG signs or symptoms suggesting AMI, < 3 hours & hemodynamic instability or resuscitated cardiac arrest

↓

PCI should ideally be performed < 120 min:
alert & transfer to cardiac shock center for primary PCI

↓

- *During the transport*, start:
  - aspirin orally or iv
  - consider potent $P2Y_{12}$ inhibitor (prasugrel or ticagrelor), or clopidogrel if contraindicated, orally
  - Anticoagulation iv
- *If hemodynamic instability*: volume loading < 250 ml & norepinephrine on peripheral line
- *If SpO_2 < 90%*: O_2 therapy

↓

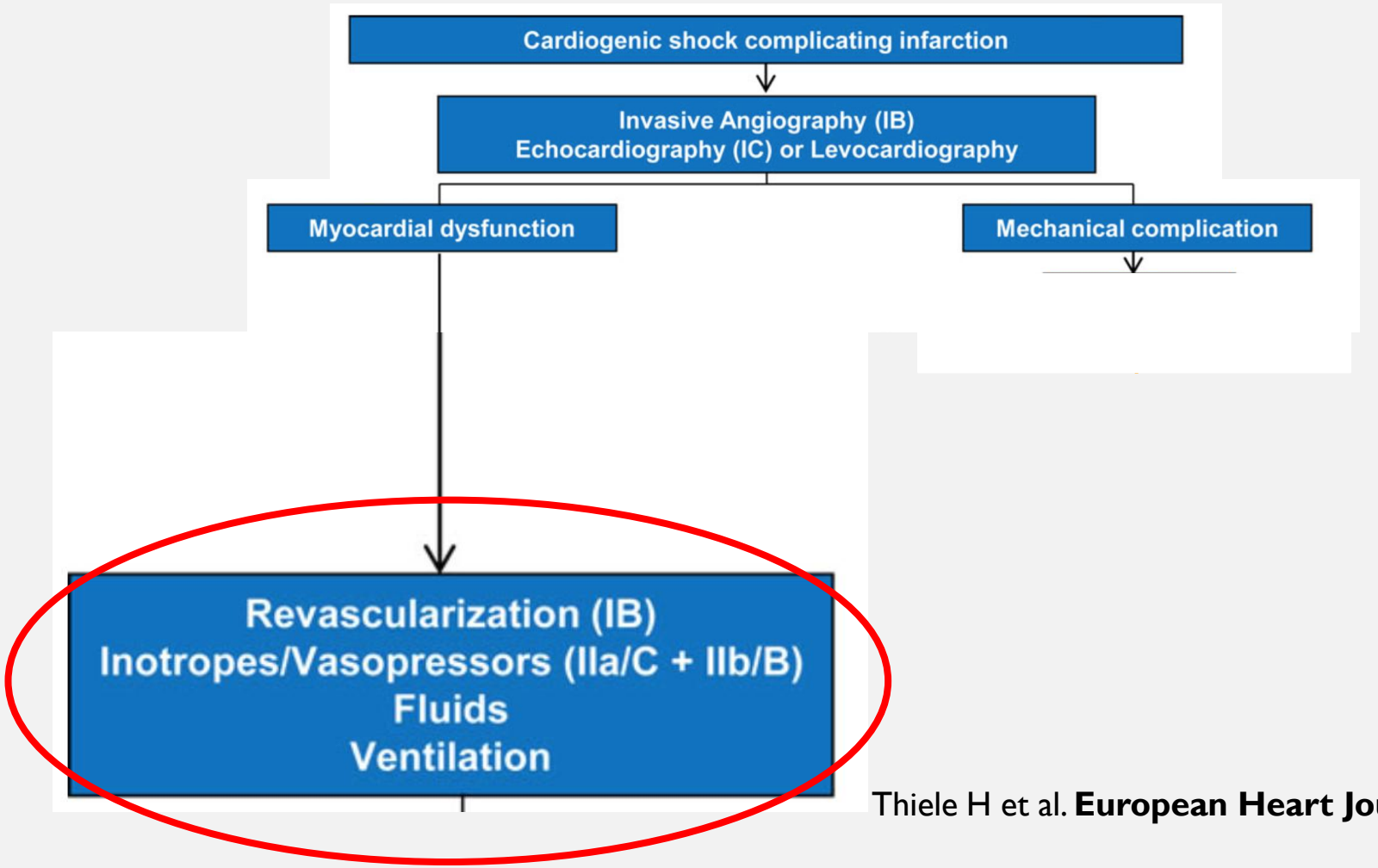
cardiac shock center

Figure 1: pre-hospital management

BACK TO THE ICU

- Arterial line
- Central venous line with SVO_2 measurements
- Echocardiography
- PAC : especially in case of high dose vasopressor and right ventricular dysfunction
- Lactate kinetic
- Impact of shock on organ function : liver, kidney
- Troponin, BNP

DIAGRAMME DE PRISE EN CHARGE: SOINS DE SUPPORT

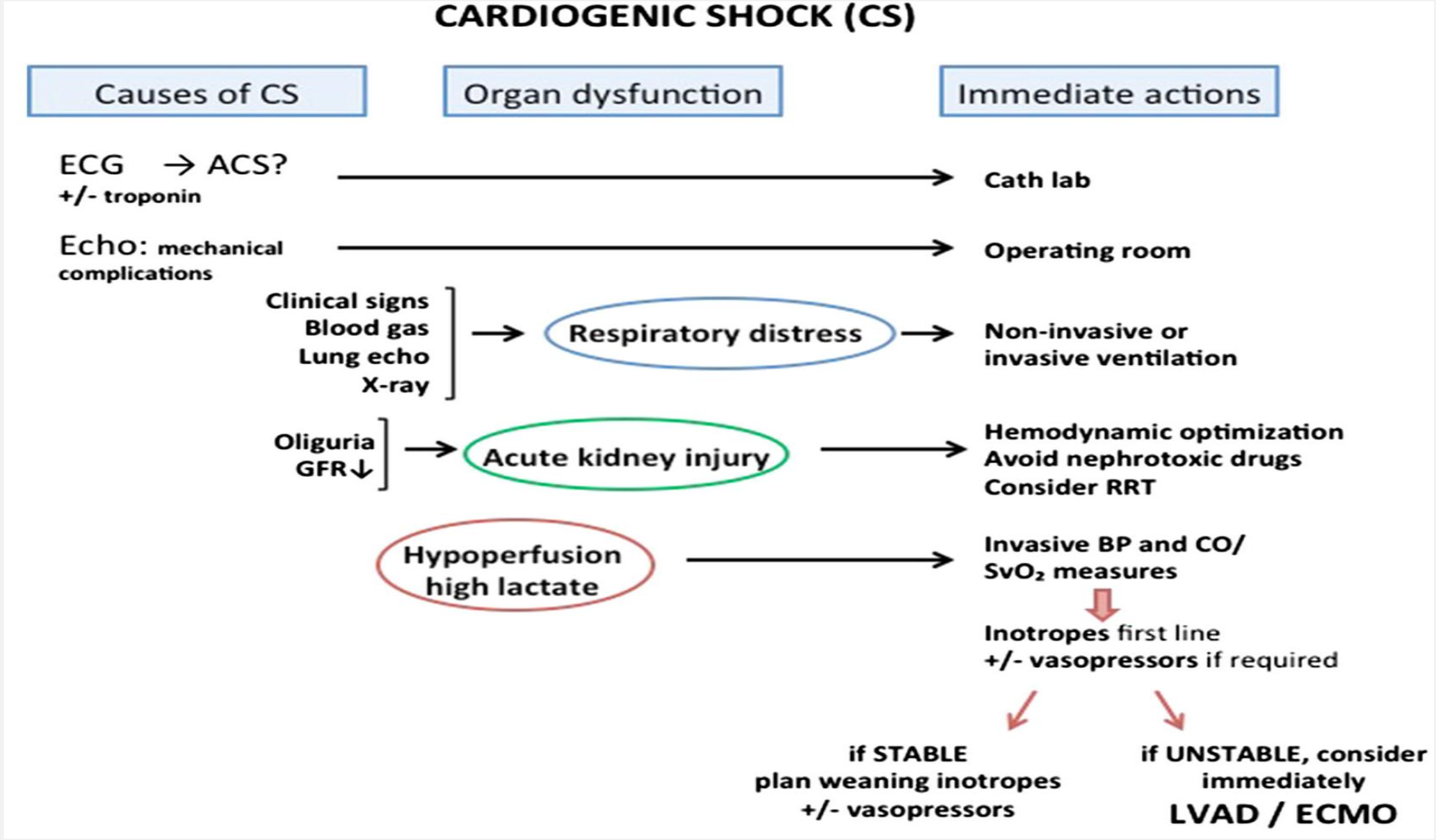


Thiele H et al. **European Heart Journal** 2015

VENTILATION MÉCANIQUE

Study	n	Description of study	Patient groupings	Definition of LV dysfunction	Amount of PEEP (cm H_2O)	Cardiopulmonary changes with PEEP
Grace and Greenbaum[38]	21	Medical ICU; titration of PEEP to maximal CO	Divided by PCWP (≤12, 14–18, ≥19)	AMI, CS or CHF requiring MV	0–8	CO increase in 12/13 of patients with PCWP ≥19
Mathru *et al*,[54]	290	Surgical ICU, post CABG; MV by CMV, IMV or IMV +PEEP	Divided by EF and LVEDP	EF <60% (mean 34%) and LVEDP >16 Torr (mean 19)	5	Improvement in RAP, PCWP, CI, SI
Dongelmans, 1986[10]	121	Surgical ICU, post CABG; MV with high versus low PEEP	High (10) versus Low (5) PEEP	None provided	5–10	Improved lung compliance, PaO_2 and decreased need for supplemental O_2 on discharge, but longer duration of MV
Malbouisson *et al*,[55]	10	Surgical ICU, post CABG; recruitment manoeuvres with high PEEP	–	Requirement of inotropic support for CS (CI <2.5)	Up to 40	Improved PaO_2/FIO_2, reduced intrapulmonary shunting, no decrease in MAP or CI
Kontoyannis *et al*,[39]	28	Medical ICU; patients with myocardial infarction complicated by CS requiring IABP	IABP alone versus IABP plus elective MV+PEEP	Systolic blood pressure <80 mm Hg with end organ damage	10	Improved ability to wean mechanical support (90% vs 56%), PCWP, CI, UO and discharge survival (80% vs 28%)

Wiesen J et al. **Heart**. 2013; 99:1812–1817



Mebazaa et al. Intensive Care Medicine 2016

Contemporary Management of Cardiogenic Shock

A Scientific Statement From the American Heart Association

		Volume Status	
		Wet	**Dry**
Peripheral Circulation	**Cold**	Classic Cardiogenic Shock (↓CI; ↑SVRI; ↑PCWP)	Euvolemic Cardiogenic Shock (↓CI; ↑SVRI; ↔PCWP)
	Warm	Vasodilatory Cardiogenic Shock or Mixed Shock (↓CI; ↓/↔SVRI; ↑PCWP)	Vasodilatory Shock (Not Cardiogenic Shock) (↑CI; ↓SVRI; ↓PCWP)

Circulation. 2017;136:00–00. DOI: 10.1161/CIR.0000000000000525

Contemporary Management of Cardiogenic Shock

A Scientific Statement From the American Heart Association

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

Circulation. 2017;136:00–00. DOI: 10.1161/CIR.0000000000000525

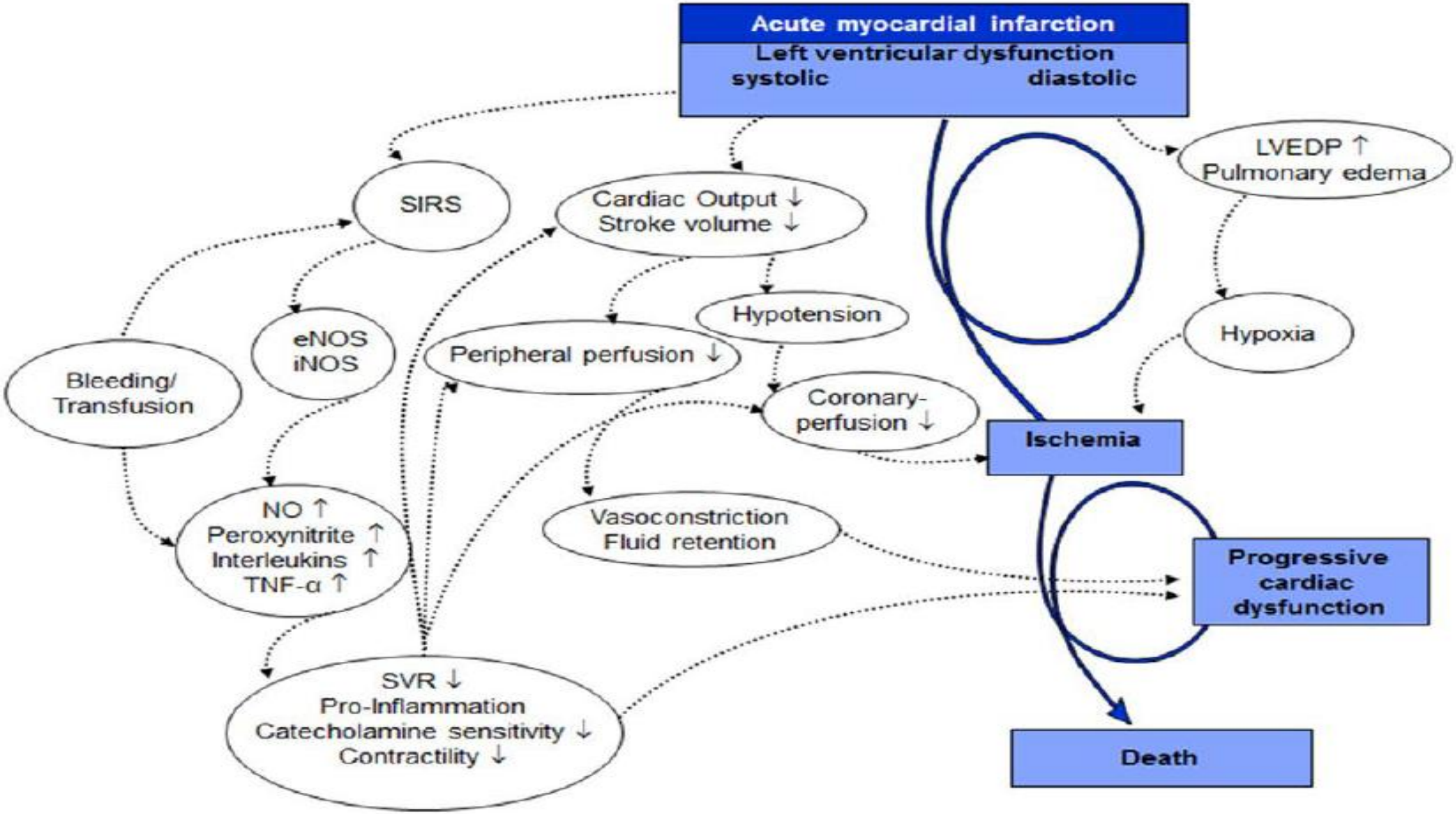
Current ESC STEMI-guideline (2017)

Recommendations for the management of cardiogenic shock in ST-elevation myocardial infarction

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

Class IIb	***Usefulness/efficacy is less well established by evidence/opinion.***	**May be considered**
Class III	**Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.**	**Is not recommended**

Level of evidence B	**Data derived from a single randomized clinical trial or large non-randomized studies.**
Level of evidence C	**Consensus of opinion of the experts and/or small studies, retrospective studies, registries.**


Acute myocardial infarction
Left ventricular dysfunction
systolic
diastolic
SIRS
Cardiac Output ↓
Stroke volume ↓
LVEDP ↑
Pulmonary edema
Hypotension
Hypoxia
eNOS
iNOS
Peripheral perfusion ↓
Bleeding/
Transfusion
Coronary-
perfusion ↓
Ischemia
NO ↑
Peroxynitrite ↑
Interleukins ↑
TNF-α ↑
Vasoconstriction
Fluid retention
Progressive
cardiac
dysfunction
SVR ↓
Pro-Inflammation
Catecholamine sensitivity ↓
Contractility ↓
Death

VASOPRESSOR USE

POTENTIAL PROBLEMS WITH VASOPRESSOR

- Excessive increase in afterload
  - Further decrease in flow
  - Excessive increase in peripheral resistances
  - Further decrease in perfusion pressure at the organ level
- Risk of ischemia exacerbation
- Excessive tachycardia
  - Increase in MVO_2
  - Decrease in diastolic time
- Cellular increase in calcium
- Arrhythmias

THE QUESTIONS?

- Which vasopressor?
- Alone or in combination with inotropes?
- Mean arterial pressure level?
- Cardiac index/SVO_2 level?
- Monitoring?
- Timing and indication : vasopressor versus ECMO?

ESTABLISHED IN 1812 MARCH 4, 2010 VOL. 362 NO. 9

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*

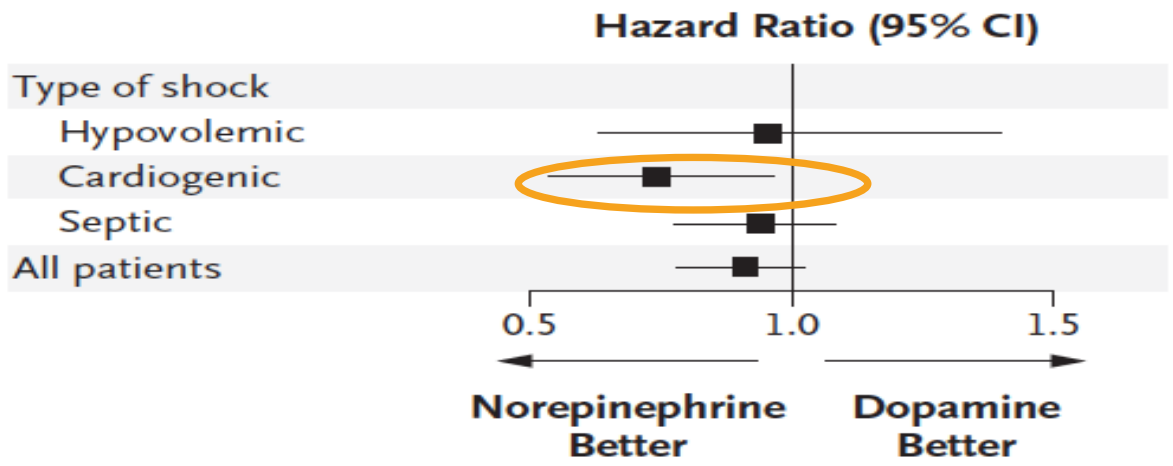


Figure 3. Forest Plot for Predefined Subgroup Analysis According to Type of Shock.

A total of 1044 patients were in septic shock (542 in the dopamine group and 502 in the norepinephrine group), 280 were in cardiogenic shock (135 in the dopamine group and 145 in the norepinephrine group), and 263 were in hypovolemic shock (138 in the dopamine group and 125 in the norepinephrine group). The P value for interaction was 0.87.

- Eventuelle utilisation chez les patients bradycardes

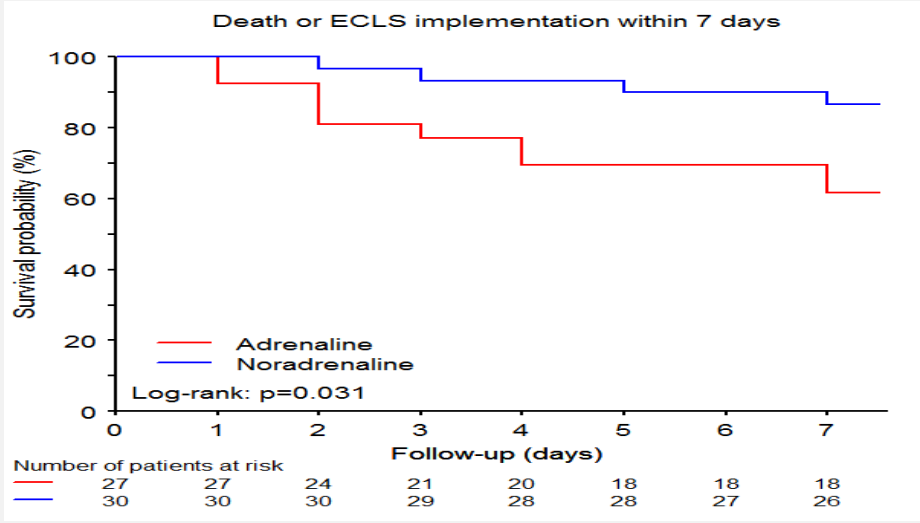
STUDY COMPARING THE EFFICACY AND TOLERABILITY OF EPINEPHRINE AND NOREPINEPHRINE IN CARDIOGENIC SHOCK (OPTIMACC) NCT01367743

- Prospective, double blind, randomized, multicenter study
- Cardiogenic shock due to myocardial infarction treated by angioplasty and needing a vasopressor support
- Epinephrine or norepinephrine titrated to MAP : 70 mmHg
- Monitoring with a Swan-Ganz catheter.
- 57 patients

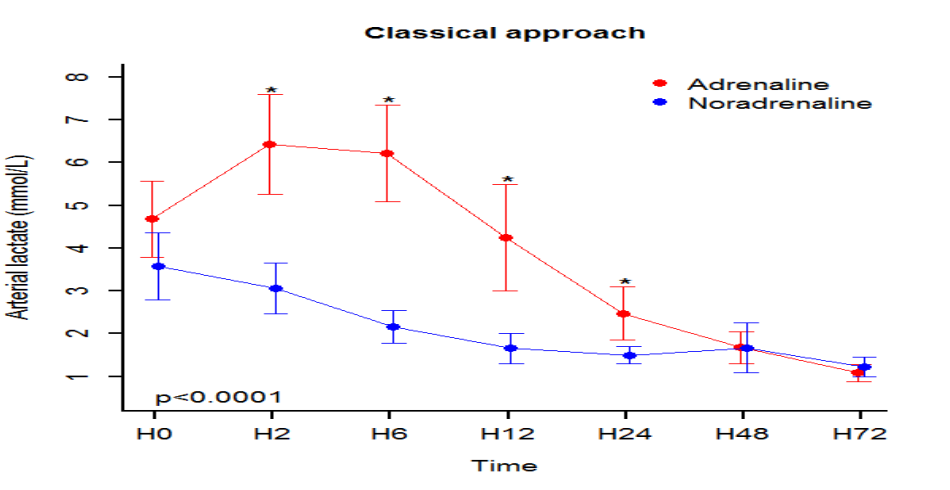
STUDY COMPARING THE EFFICACY AND TOLERABILITY OF EPINEPHRINE AND NOREPINEPHRINE IN CARDIOGENIC SHOCK (OPTIMACC) NCT01367743

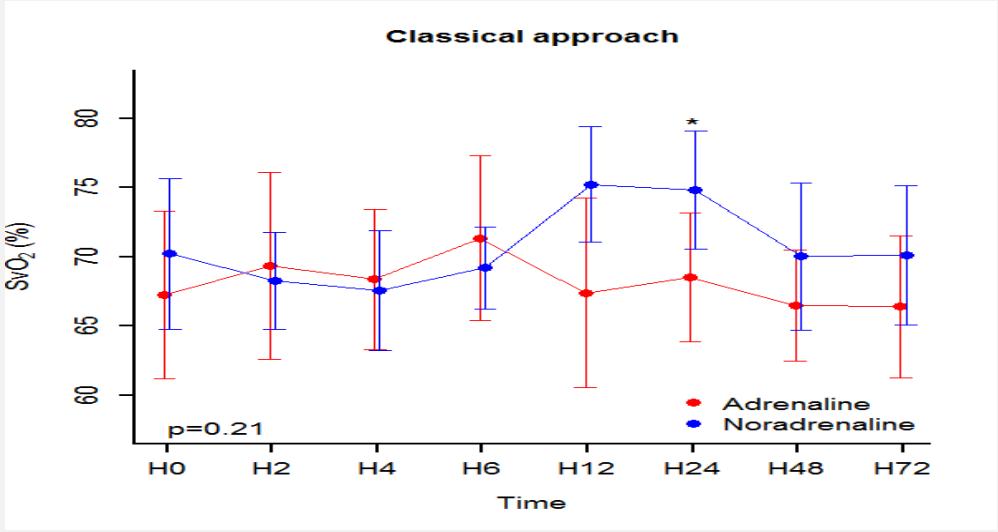
B LEVY ET AL, JACC IN PRESS

- **More refractory cardiogenic shock in the epinephrine group**.
  - 39% in the epinephrine group versus 7 % in the Nor group (p= 0.008)
- **Epinephrine use was associated with a trend of an increased risk of death** (p=0.08) and an increased risk of death plus ECMO (p=0.031) at 7 days. There was a trend of an increased risk of death plus ECMO at J28 (p= 0.064)



- No differences in vasopressor and dobutamine use
- **Higher heart rate in the epinephrine group with a similar cardiac index and similar stroke volume.**
- **Epinephrine induced lactic acidosis**



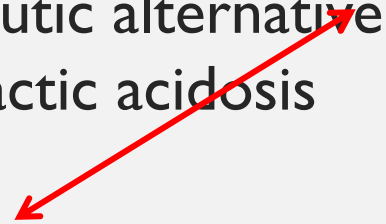


Refractory shock was associated with a higher cardiac index in the epinephrine group likely due to beta-2 adrenergic receptors stimulation evoking a cardiac over-stimulation

Experts' recommendations for the management of adult patients with cardiogenic shock

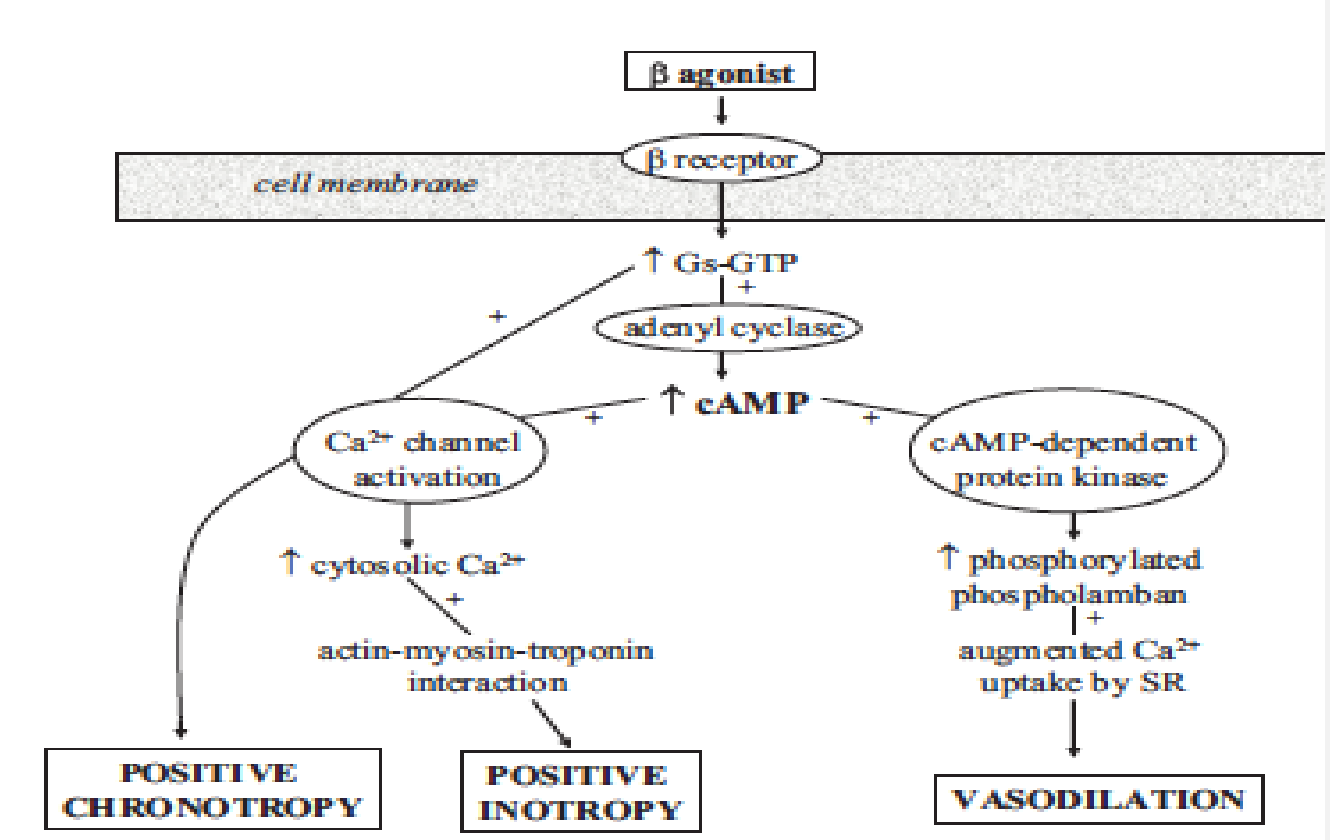
Bruno Levy[1*], Olivier Bastien[2], Karim Bendjelid[3], Alain Cariou[4], Tahar Chouihed[5], Alain Combes[6], Alexandre Mebazaa[7], Bruno Megarbane[8], Patrick Plaisance[9], Alexandre Ouattara[10], Christian Spaulding[11], Jean-Louis Teboul[12], Fabrice Vanhuyse[13], Thierry Boulain[14] and Kaldoun Kuteifan[15]

- A MAP of at least 65 mmHg is recommended
- Norepinephrine should be used to restore perfusion pressure (strong agreement).
- Epinephrine can be a therapeutic alternative but is associated with a greater risk of arrhythmia, tachycardia and lactic acidosis



INOTROPE USE

INTRACELLULAR ACTIONS OF BETA1-AGONIST



- Dobutamine
  - Short-half life
  - Beta-1 and beta-2 (3/1)
  - Mild vasodilation
  - Increase MVO_2
  - Tolerance (receptor internalization)

SHOCK, Vol. 41, No. 4, pp. 269–274, 2014

INCREASING MEAN ARTERIAL PRESSURE IN CARDIOGENIC SHOCK SECONDARY TO MYOCARDIAL INFARCTION: EFFECTS ON HEMODYNAMICS AND TISSUE OXYGENATION

Pierre Perez,* Antoine Kimmoun,*†‡ Vincent Blime,* and Bruno Levy*†‡

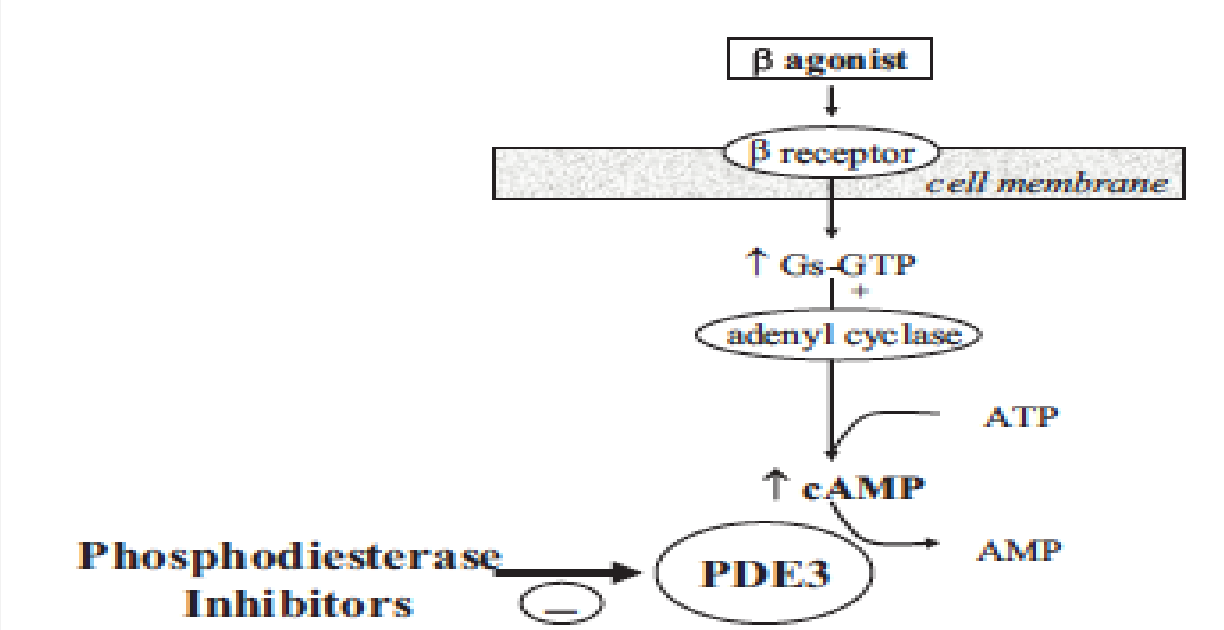
**CHU Nancy, Service de Réanimation Médicale Brabois, Pole Cardiovasculaire et Réanimation Médicale, Hôpital Brabois; and †INSERM, Groupe Choc, U1116, Faculté de Médecine, Vandoeuvre-les-Nancy; and ‡Université de Lorraine, Nancy, France*

INCREASING MEAN ARTERIAL PRESSURE IN CARDIOGENIC SHOCK SECONDARY TO MYOCARDIAL INFARCTION: EFFECTS ON HEMODYNAMICS AND TISSUE OXYGENATION

Pierre Perez,* Antoine Kimmoun,*†‡ Vincent Blime,* and Bruno Levy*†‡

	MAP 65 mmHg	MAP 85 mmHg	p=
Heart rate (bpm)	102 +/- 8	105 +/- 7	p > 0,05
CI (l/min/m²)	2,3 +/- 0,4	2,8 +/- 0,3	p < 0,05
CPI (watt/m²)	0,38 +/- 0,03	0,58 +/- 0,04	p < 0,01
SVO_2 (%)	73 +/- 2	79 +/- 2	p < 0,05

INTRACELLULAR ACTIONS OF PHOSPHODIESTERASE INHIBITOR



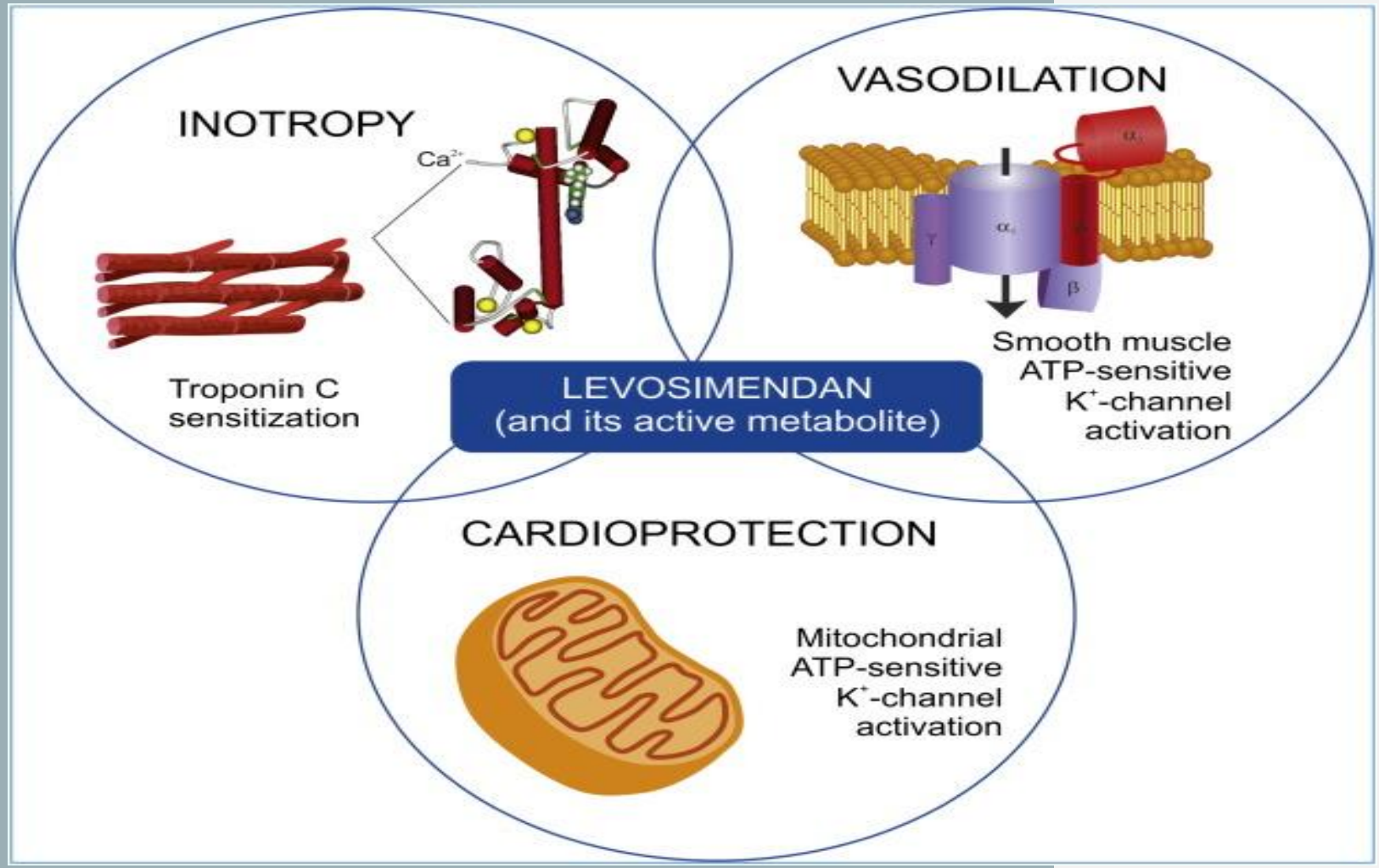
- Milrinone
  - Long-half life
  - Inodilatator
- // Dobutamine :
  - Less increases in heart rate
  - More vasodilation
  - Less increase in MVO_2
  - No tolerance
  - Efficient in beta-blocking patients
  - Thrombocytopenia

Conventional Hemodynamic Resuscitation May Fail to Optimize Tissue Perfusion: An Observational Study on the Effects of Dobutamine, Enoximone, and Norepinephrine in Patients with Acute Myocardial Infarction Complicated by Cardiogenic Shock

Corstiaan A. den Uil[1]*, Wim K. Lagrand[2], Martin van der Ent[3], Koen Nieman[1], Ard Struijs[1], Lucia S. D. Jewbali[1], Alina A. Constantinescu[1], Peter E. Spronk[4], Maarten L. Simoons[1]

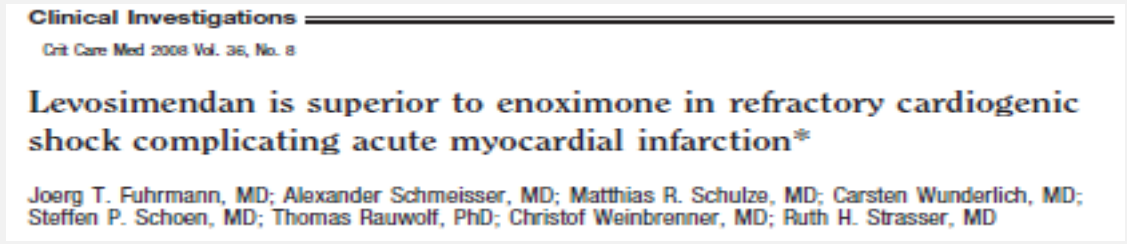
	Dobutamine (n = 14)	Enoximone (n = 10)	Norepinephrine (n = 9)	P-value
ΔHR, bpm	+9 [0; +16]**	+4 [−11; +9]	+1 [−15; +4]	NS
ΔMAP, mmHg	+6 [−5; +21]	+8 [+1; +14]	+17 [+13; +32]**	NS
ΔCVP, mmHg	−1 [−3; +1]	−2 [−3; −1]*	+2 [−4; +4]	NS
ΔPCWP, mmHg	−2 [−4; −1]**	−2 [−3; −1]**	+5 [−1; +7]	NS
ΔMPAP, mmHg[a]	0 [−3; +3]	−1 [−9; 0]	+4 [−1; +7]	NS
ΔCI, $L.min^{-1}.m^{-2}$	+0.8 [+0.3; +1.4]**	+0.6 [−0.1; +1.5]	0.0 [−0.5; +0.1]	0.006
ΔSVR, $dynes.sec.cm^{-5}$	−201 [−623; +220]	−119 [−491; +175]	+390 [+237; +505]*	0.03
ΔSvO2, %	+6 [+2; +12]**	0 [−3; +4]	0 [−3; +6]	0.04
ΔLactate, $mmol.L^{-1}$	−0.4 [−2.5; −0.1]**	0.0 [−0.6; +0.2]	0.0 [−0.2; +0.5]	NS
ΔDelta-T, °C	−0.4 [−0.8; 0]	−1.1 [−1.9; +0.6]	0.0 [−2.2; +0.6]	NS
ΔPCD, $mm.mm^{-2}$	+0.6 [−0.9; +2.3]	+2.0 [+0.5; +3.4]*	−0.4 [−3.3; 0.0]	0.01

LEVOSIMENDAN



- Effective very long half-life
- No tolerance
- Specific action on right ventricule
- Inodilator
- Neutral impact on left ventricular efficiency and MVO_2
- Improved myocardial efficiency.

Levosimendan in cardiogenic shock: Overview of main publications

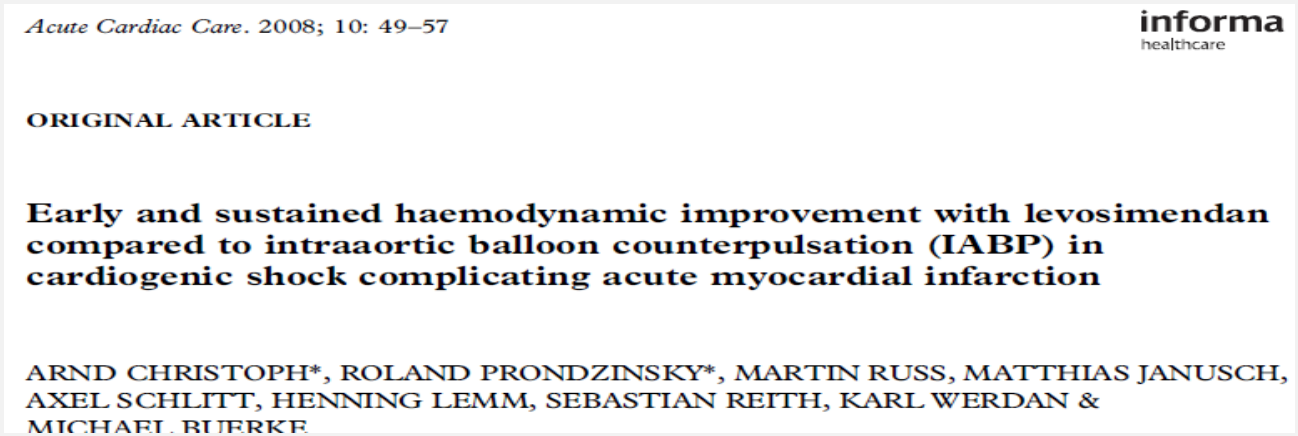


Clinical Investigations

Crit Care Med 2008 Vol. 36, No. 8

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



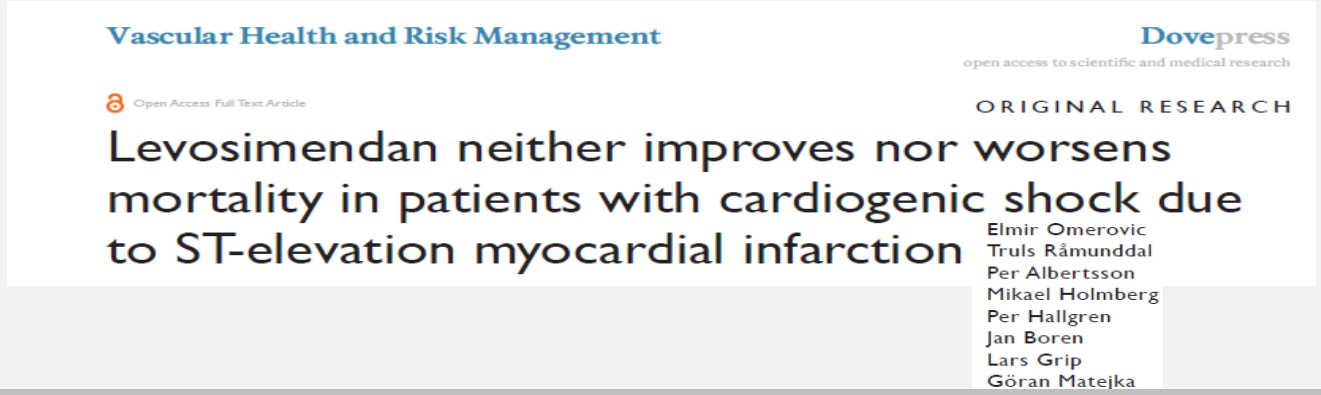
Acute Cardiac Care. 2008; 10: 49–57

informa healthcare

ORIGINAL ARTICLE

Early and sustained haemodynamic improvement with levosimendan compared to intraaortic balloon counterpulsation (IABP) in cardiogenic shock complicating acute myocardial infarction

ARND CHRISTOPH*, ROLAND PRONDZINSKY*, MARTIN RUSS, MATTHIAS JANUSCH, AXEL SCHLITT, HENNING LEMM, SEBASTIAN REITH, KARL WERDAN & MICHAEL BUERKE



Vascular Health and Risk Management

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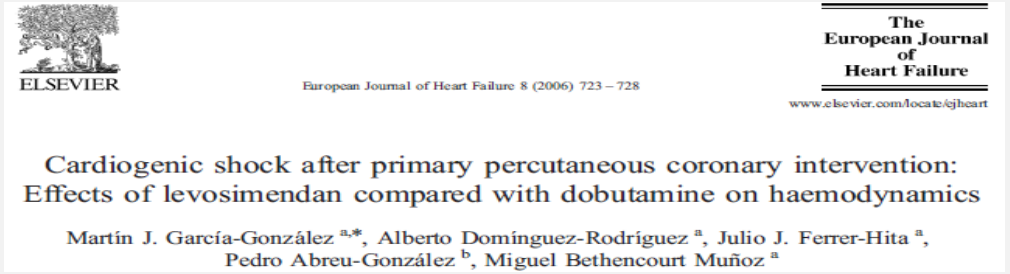
ORIGINAL RESEARCH

Levosimendan neither improves nor worsens mortality in patients with cardiogenic shock due to ST-elevation myocardial infarction

Elmir Omerovic
Truls Råmunddal
Per Albertsson
Mikael Holmberg
Per Hallgren
Jan Boren
Lars Grip
Göran Matejka

Same patients in the 3 publications below



ELSEVIER

European Journal of Heart Failure 8 (2006) 723–728

The European Journal of Heart Failure

www.elsevier.com/locate/ejheart

Cardiogenic shock after primary percutaneous coronary intervention: Effects of levosimendan compared with dobutamine on haemodynamics

Martín J. García-González [a,*], Alberto Domínguez-Rodríguez [a], Julio J. Ferrer-Hita [a], Pedro Abreu-González [b], Miguel Bethencourt Muñoz [a]

ELSEVIER

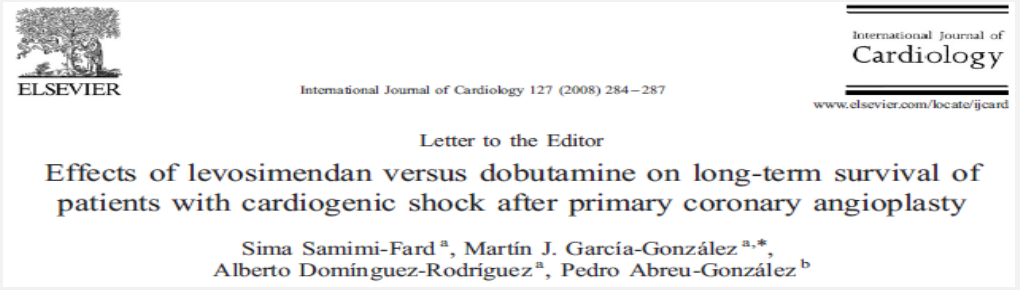
International Journal of Cardiology 128 (2008) 214–217

International Journal of Cardiology

www.elsevier.com/locate/ijcard

Effects of levosimendan versus dobutamine on left ventricular diastolic function in patients with cardiogenic shock after primary angioplasty

Alberto Dominguez-Rodriguez [a,*], Sima Samimi-Fard [a], Martin J. Garcia-Gonzalez [a], Pedro Abreu-Gonzalez [b]



ELSEVIER

International Journal of Cardiology 127 (2008) 284–287

International Journal of Cardiology

www.elsevier.com/locate/ijcard

Letter to the Editor

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]

Conclusions First Part

- In cardiogenic shock complicating AMI, adding levosimendan to standard therapy
  - Improves haemodynamics
  - Seems to be safe
- The current studies are too small to draw conclusions about effects on the incidence of refractory shock, ECMO implantation and survival

WHEN AND HOW TO USE LEVOSIMENDAN IN CARDIOGENIC SHOCK

- Dobutamine remains the first choice
  - Levo as a first choice in patients previously treated with beta-blockers ?
- In cases of failure/insufficient efficacy of dobutamine
  - Stable arterial pressure or low doses of norepinephrine
  - No hypovolemia, no sepsis
  - No loading doses

WHEN AND HOW TO USE LEVOSIMENDAN IN CARDIOGENIC SHOCK

- During ECMO therapy
  - To increase ECMO withdrawal
  - To decrease time on ECMO
- When/how :
  - As soon as possible
  - Stop dobutamine

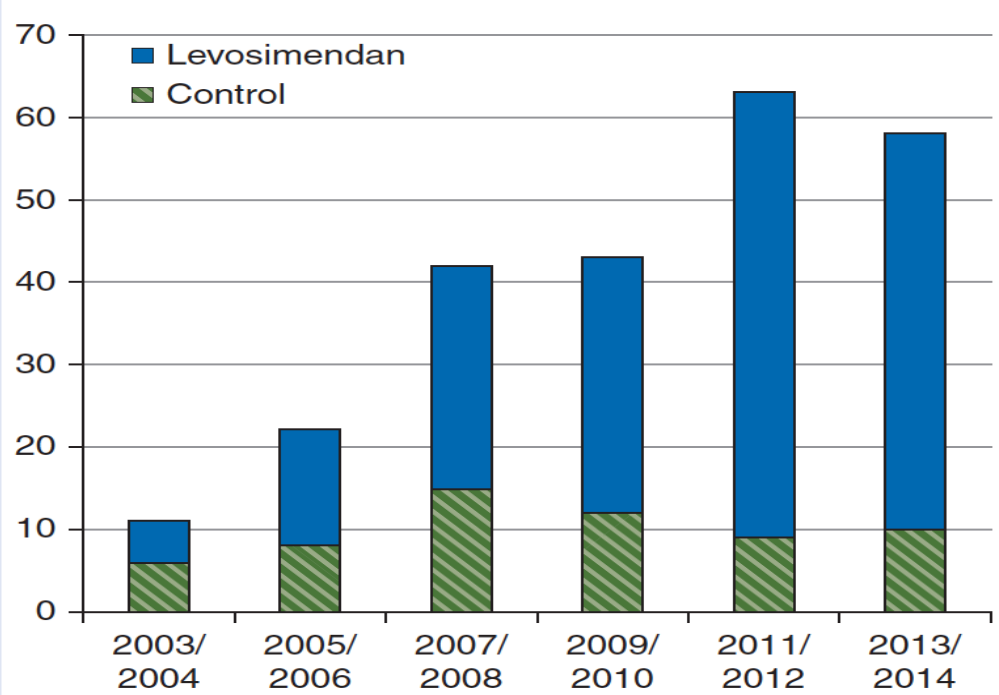
BJA

British Journal of Anaesthesia, 117 (1): 52–8 (2016)

doi: 10.1093/bja/aew151
Cardiovascular

Beneficial effects of levosimendan on survival in patients undergoing extracorporeal membrane oxygenation after cardiovascular surgery

K. Distelmaier[1], C. Roth[1], L. Schrutka[1], C. Binder[1], B. Steinlechner[2], G. Heinz[1], I. M. Lang[1], G. Maurer[1], H. Koinig[3], A. Niessner[1], M. Hülsmann[1], W. Speidl[1] and G. Goliasch[1,*]



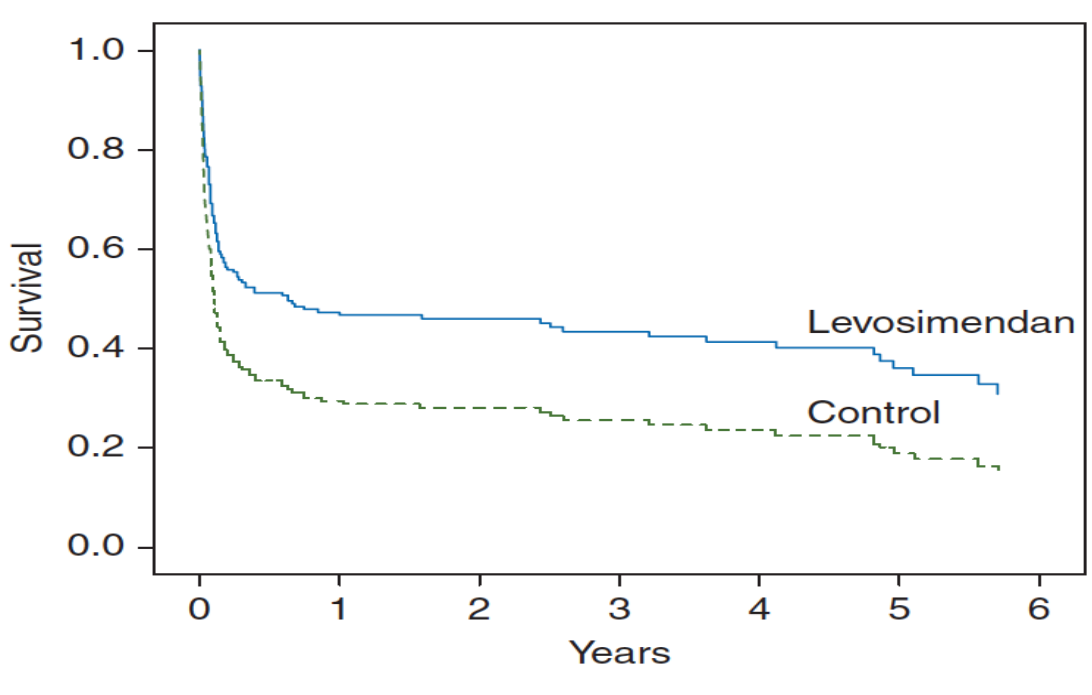


Table 2 Unadjusted and adjusted Cox proportional hazard model analysing the effect of levosimendan treatment on ECMO weaning failure, 30 day mortality, and long-term mortality. Hazard ratios are adjusted for all variables in the clinical confounder model (i.e. for age, sex, SAPS-3, SOFA score, hypertension, diabetes, maximal norepinephrine dose within first 24 h, left ventricular function, duration of ECMO support, and type of cardiovascular surgery). CI, confidence interval; ECMO, extracorporeal membrane oxygenation; HR, hazard ratio; SAPS-3, simplified acute physiology score 3; SOFA, sequential organ failure assessment

	Crude HR (95% CI)	P-value	Adjusted HR (95% CI)	P-value
ECMO weaning failure	0.54 (0.31–0.93)	**0.03**	0.41 (0.22–0.80)	**0.008**
30 day mortality	0.61 (0.39–0.96)	**0.03**	0.52 (0.30–0.89)	**0.016**
Long-term mortality	0.77 (0.54–1.09)	0.14	0.64 (0.42–0.98)	**0.04**

Experts' recommendations for the management of adult patients with cardiogenic shock

Bruno Levy[1*], Olivier Bastien[2], Karim Bendjelid[3], Alain Cariou[4], Tahar Chouihed[5], Alain Combes[6], Alexandre Mebazaa[7], Bruno Megarbane[8], Patrick Plaisance[9], Alexandre Ouattara[10], Christian Spaulding[11], Jean-Louis Teboul[12], Fabrice Vanhuyse[13], Thierry Boulain[14] and Kaldoun Kuteifan[15]

- Dobutamine should be used to treat low cardiac output in cardiogenic shock. (strong agreement)
- Phosphodiesterase inhibitors or levosimendan should not be used first-line
- CS refractory to catecholamines can be treated by perfusion of phosphodiesterase inhibitors or levosimendan
- There is a pharmacological rationale for the use of this strategy in patients on chronic beta-blocker treatment. (weak agreement)
- In CS refractory to catecholamines it seems logical to consider the use of circulatory support rather than increased pharmacological support

Levy *et al. Annals of Intensive Care* (2015) 5:17

TAKE HOME MESSAGE (1)

- Inotropes : Indication and titration
  - Symptomatic low cardiac output : heart rate, CI/SVO_2, lactate, echo
  - No hypovolemia
- Dobutamine : start at 2 microgramme/kg/min
- Levosimendan : no bolus, start at 0.1 microgramme/kg/min
- Enoximone no bolus, start at 1 vial//24h

  (2 to 10 microgramme/kg/min)

TAKE HOME MESSAGE (2)

Norepinephrine plus dobutamine

Medical treatment

- Contra-indication to ECLS
- Improvement on medical treatment

ECLS

- Crash and burn
  - Lactic acidosis, increase in NE, organ failure
- Refractory cardiogenic shock

LEVOSIMENDAN?

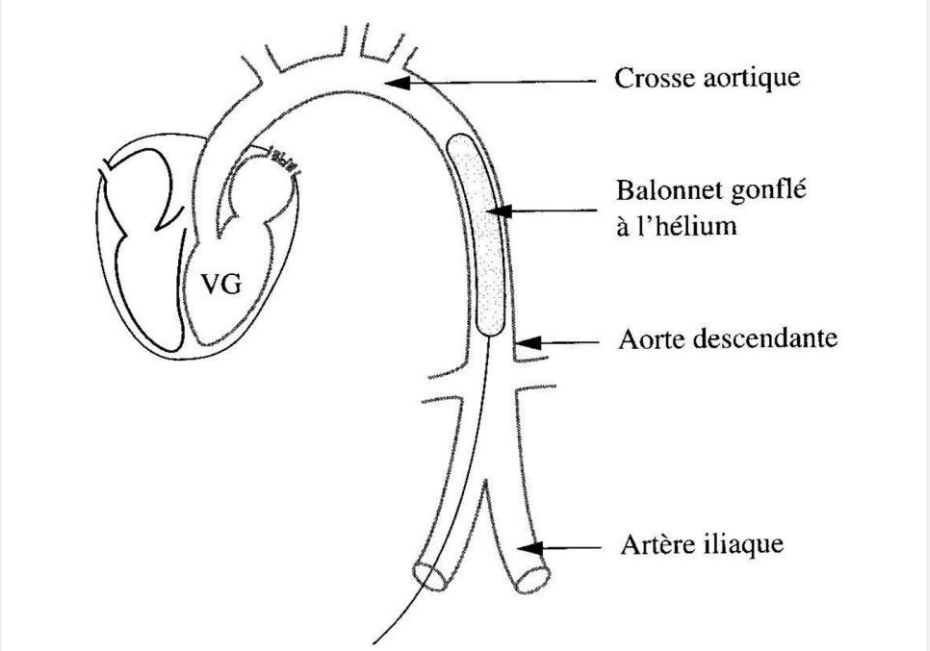
WHEN CARDIOGENIC SHOCK IS BECOMING REFRACTORY
MECHANICAL ASSISTANCES DEVICES

Un objectif prioritaire:
Rétablir un débit circulatoire

BALLON DE CONTRE PULSION INTRA-AORTIQUE: DISPOSITIFS

Ballon :

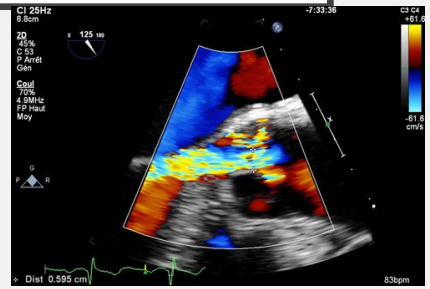
- 30 à 40 cm de long
- Entre la sous-clavière et les artères rénales
- Synchronisé sur l'ECG
- Qui se gonfle à l'hélium en diastole (à la dicrote)
- Qui se dégonfle activement



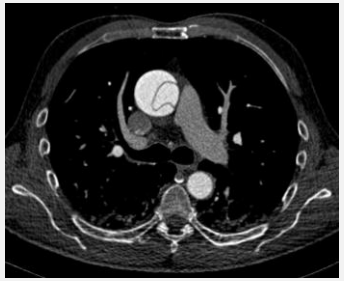
Ballon de contre pulsion intra-aortique: Indications initiales

- Infarctus antérieur non revascularisé
- Infarctus antérieur revascularisé
- Choc cardiogénique
- Angioplastie coronarienne à risque
- CIV post infarctus
- Infarctus VD
- Décharge VG sous ECMO

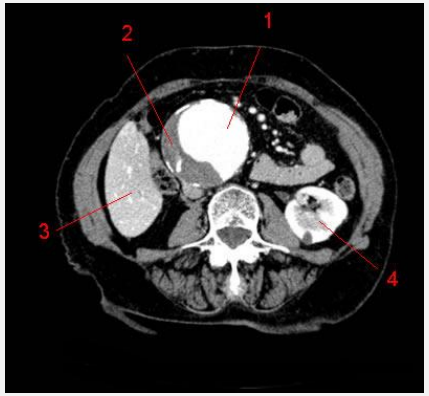
BALLON DE CONTRE PULSION INTRA-AORTIQUE: CONTRE-INDICATIONS



- Insuffisance aortique préexistante



- Dissection aortique



- Dilatations anévrismales de l'aorte

- Les patients « trop » tachycardes

BALLON DE CONTRE PULSION INTRA-AORTIQUE: COMPLICATIONS

- Ischémiques du membre inférieur
- Hémorragique
- Thrombopénie
- Hypoperfusion rénale
- Hypoperfusion mésentérique

Si malposition

BALLON DE CONTRE PULSION INTRA-AORTIQUE: AVANTAGES ET INCONVÉNIENTS

AVANTAGES

- Facilité d'implantation
- Physiologique

INCONVÉNIENTS

- Ne restaure pas de débit
- Temporaire
- Complications:
  - Hémodynamiques
  - Infectieuses

LA CONTRE PULSION INTRA AORTIQUE

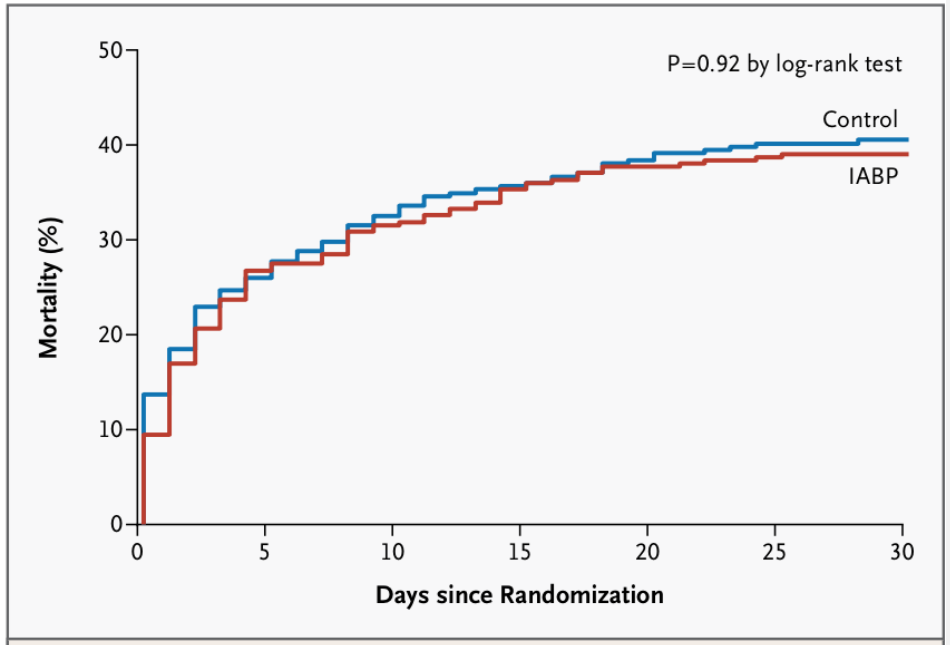


Figure 1. Time-to-Event Curves for the Primary End Point.

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

Thiele H et al. **N Engl J Med** 2012;367:1287-96

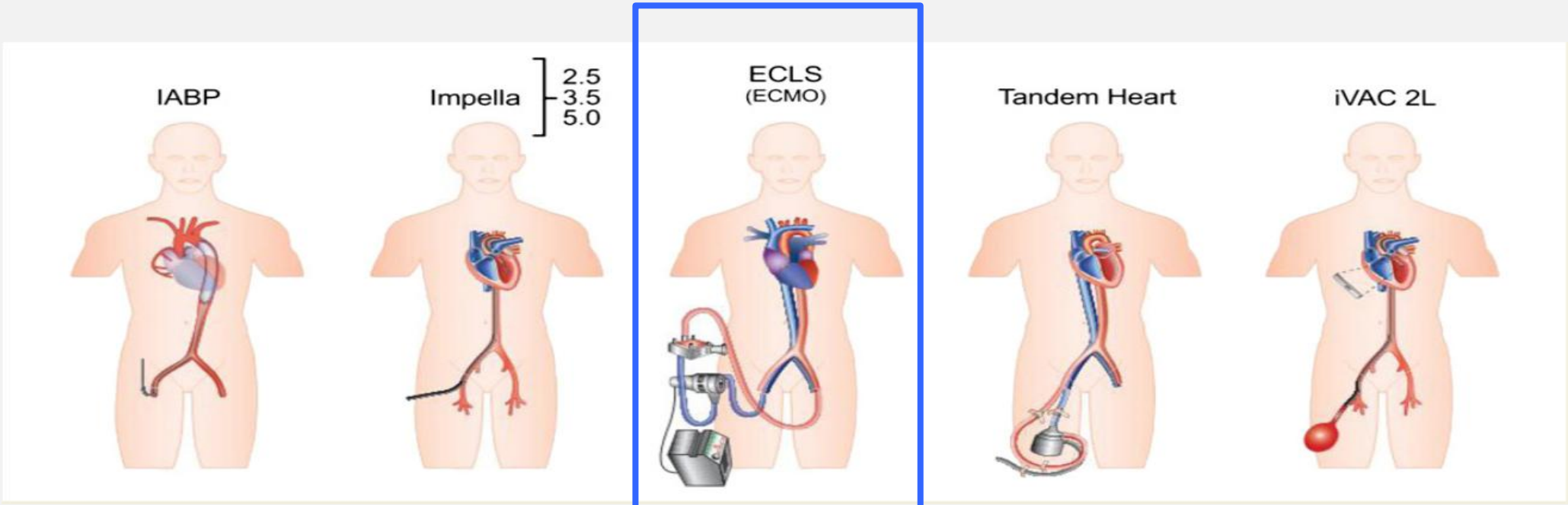
2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC)

Recommendations for the management of cardiogenic shock in ST-elevation myocardial infarction

Intra-aortic balloon pumping should be considered in patients with haemodynamic instability/cardiogenic shock due to mechanical complications.	IIa	C
Routine intra-aortic balloon pumping is not indicated.[177,437]	III	B

QUELLE TECHNIQUE?



Thiele H et al. **European Heart Journal** 2015

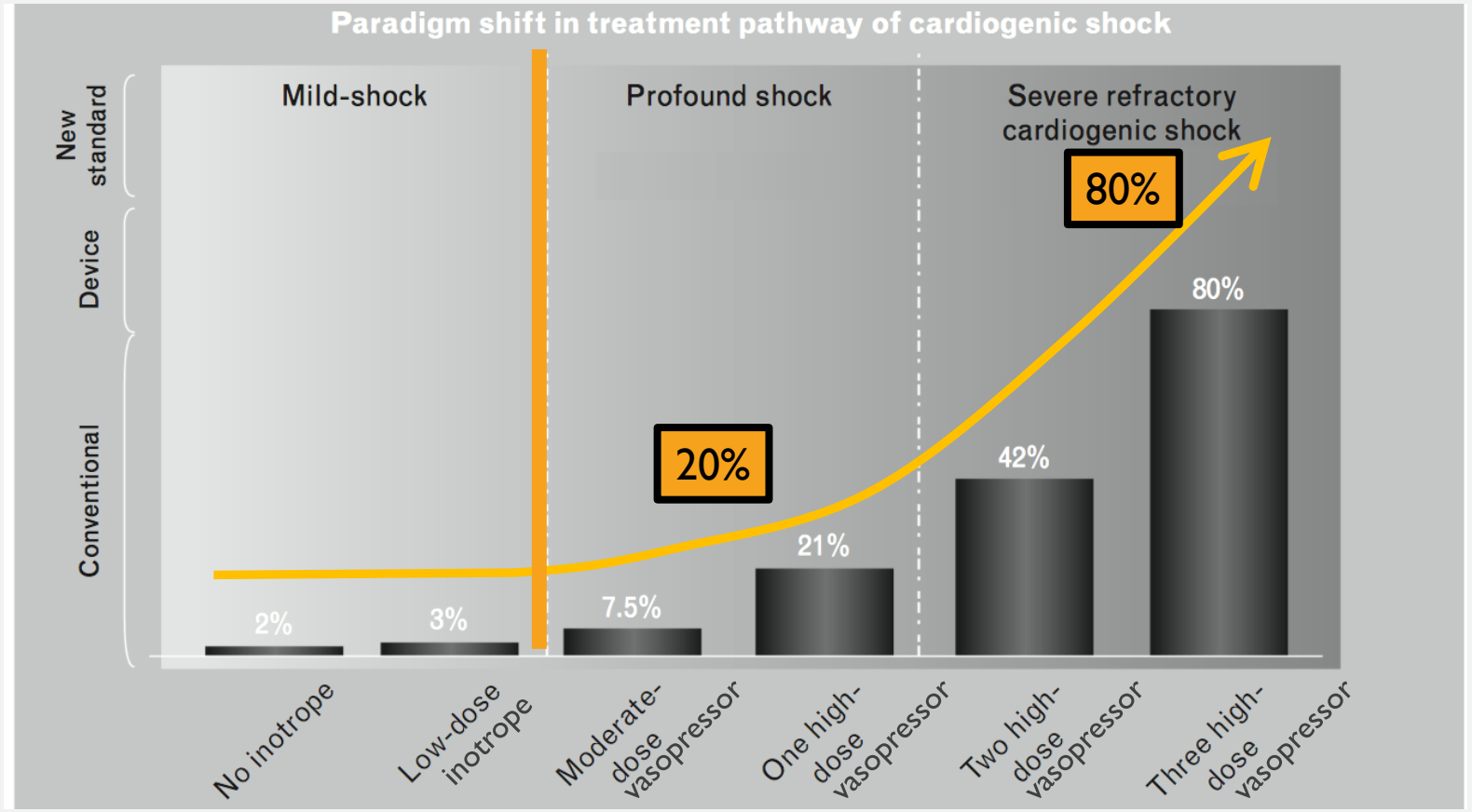
VA-ECMO : INDICATIONS

CHOC CARDIOGÉNIQUE

- **Pression artérielle systolique < 90 mmHg ou pression artérielle moyenne < 65 mmHg pendant plus de 30 min**
- **Index cardiaque <1,8 l/min/m^2**
- Augmentation des pressions de remplissage ventriculaire gauche
- **Inotrope ou Vasopresseur**
- **Altération de la perfusion tissulaire:**
  - Trouble de conscience
  - Oligurie
  - Extrémités froides
  - Augmentation du lactate

Levy B et al. **Ann intensive Care** 2015

GRADATION DE LA GRAVITÉ DANS LE CHOC CARDIOGÉNIQUE



Basra SS et al. **Current Opinion in Cardiology** 2011

DÉFINITION DU CHOC CARDIOGÉNIQUE RÉFRACTAIRE

Choc cardiogénique **ne répondant pas au traitement médical (inotrope/vasopresseur/étiologique) bien conduit** :

Augmentation non contrôlée des posologies de vasopresseurs

Signes cliniques de profond bas débit
Atteinte hépatique et/ou rénale évolutive

Confirmé par l'échocardiographie ou par le monitorage invasif

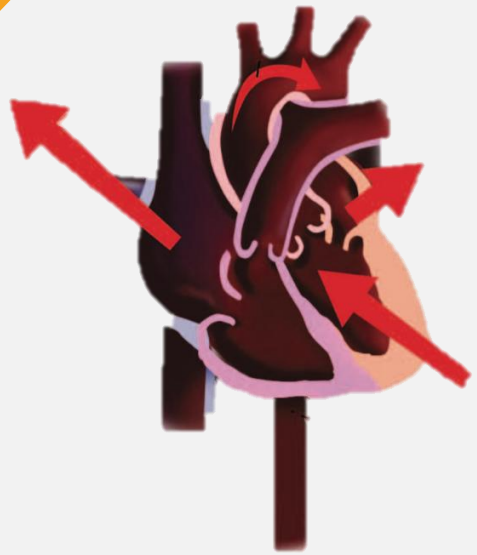
Pas de consensus

CONSÉQUENCES PHYSIOPATHOLOGIQUE DU CHOC CARDIOGÉNIQUE RÉFRACTAIRE

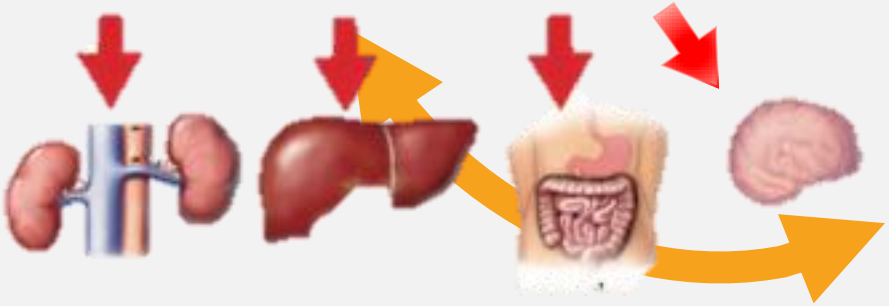
Harjola et al. **Eur J Heart Fail** 2017

Bas débit

Congestion

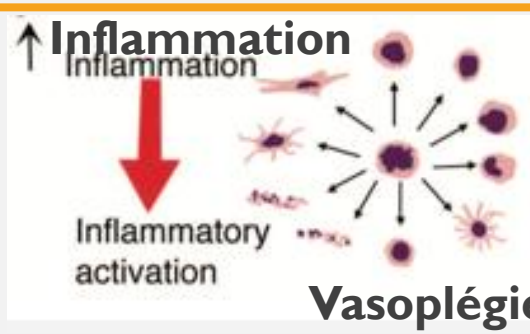


Ischémie, défaillance



DECES

↑Inflammation

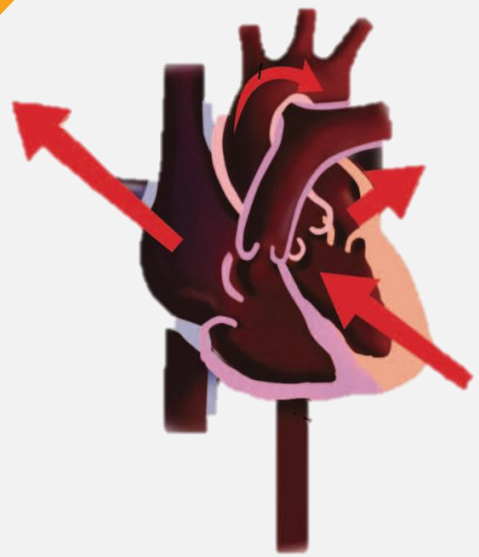


Vasoplégie

Noradrénaline

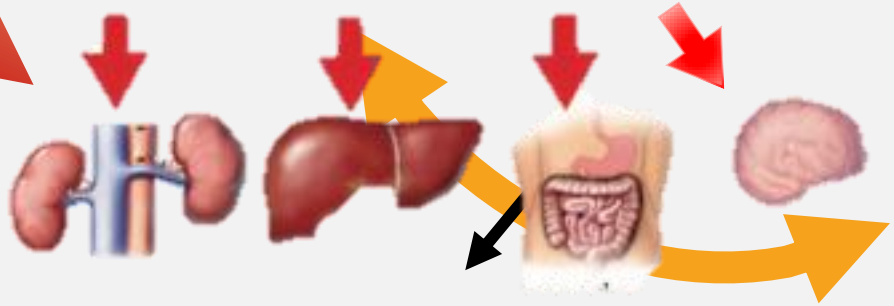
L'ASSISTANCE CIRCULATOIRE ECMO VA : EVITER LE CERCLE VICIEUX

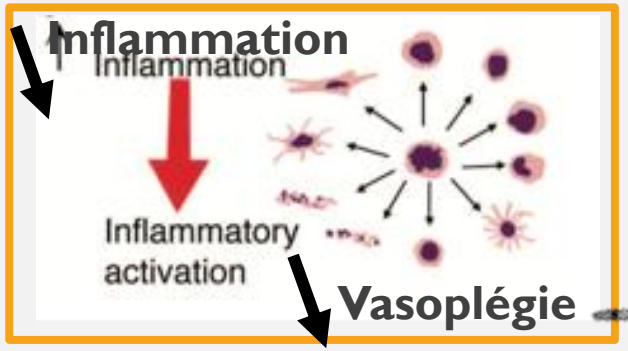
Bas débit



DECES

Ischémie, défaillance





Précoce avant l'installation des défaillances rénales et hépatiques

Noradrénaline

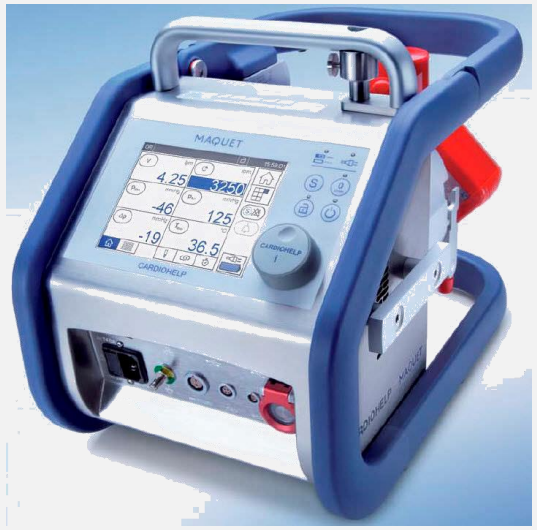
Hanjola et al. **Eur J Heart Fail** 2017

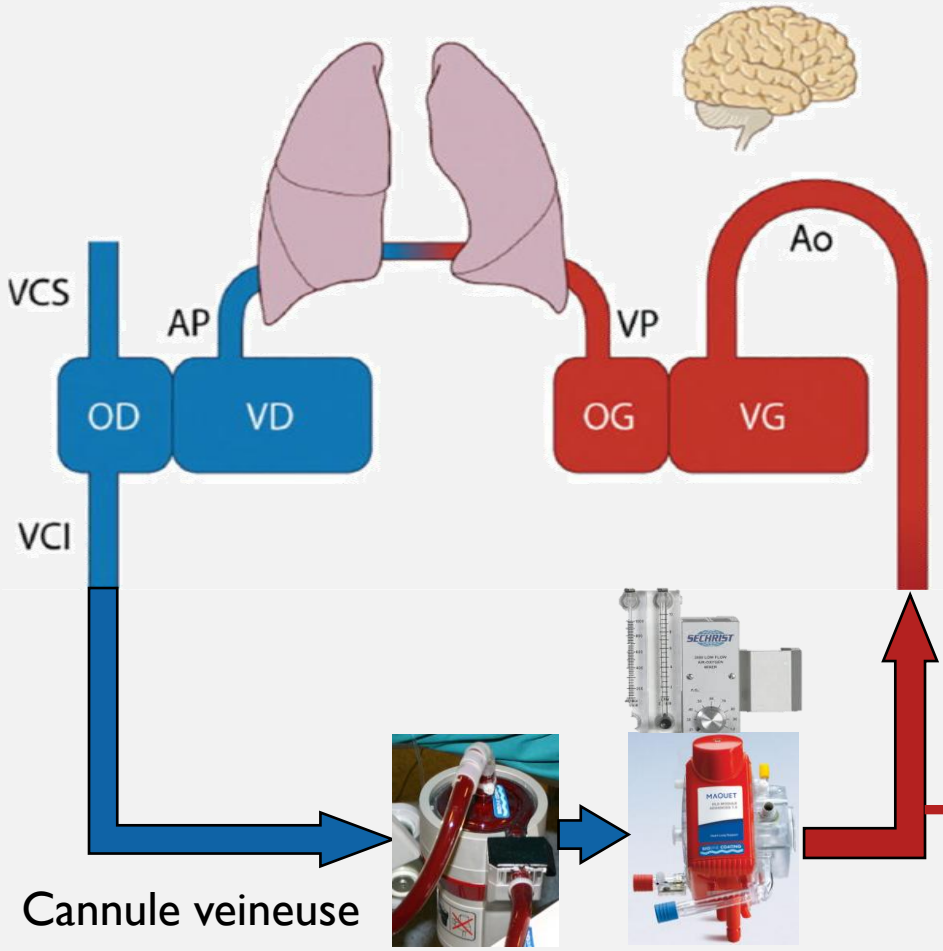
TECHNIQUES

L'ASSISTANCE CIRCULATOIRE: CAHIER DES CHARGES

Vrai débit circulatoire : PRIMORDIAL
Facilité d'implantation <20 min
Décharge ventriculaire gauche et droite
Faible taux de complications

ECMO VA : PRINCIPE TECHNIQUE





Décharge ventricule droit

Débit circulatoire de 1 à 7 l/min

Sang oxygéné et épuré en CO_2

Réinjecte à "contre-courant"

Adapté de Calderaon J et al. **Traité d'Anesthésie Réanimation** 2014

DESCRIPTION DU DISPOSITIF ET IMPACTS

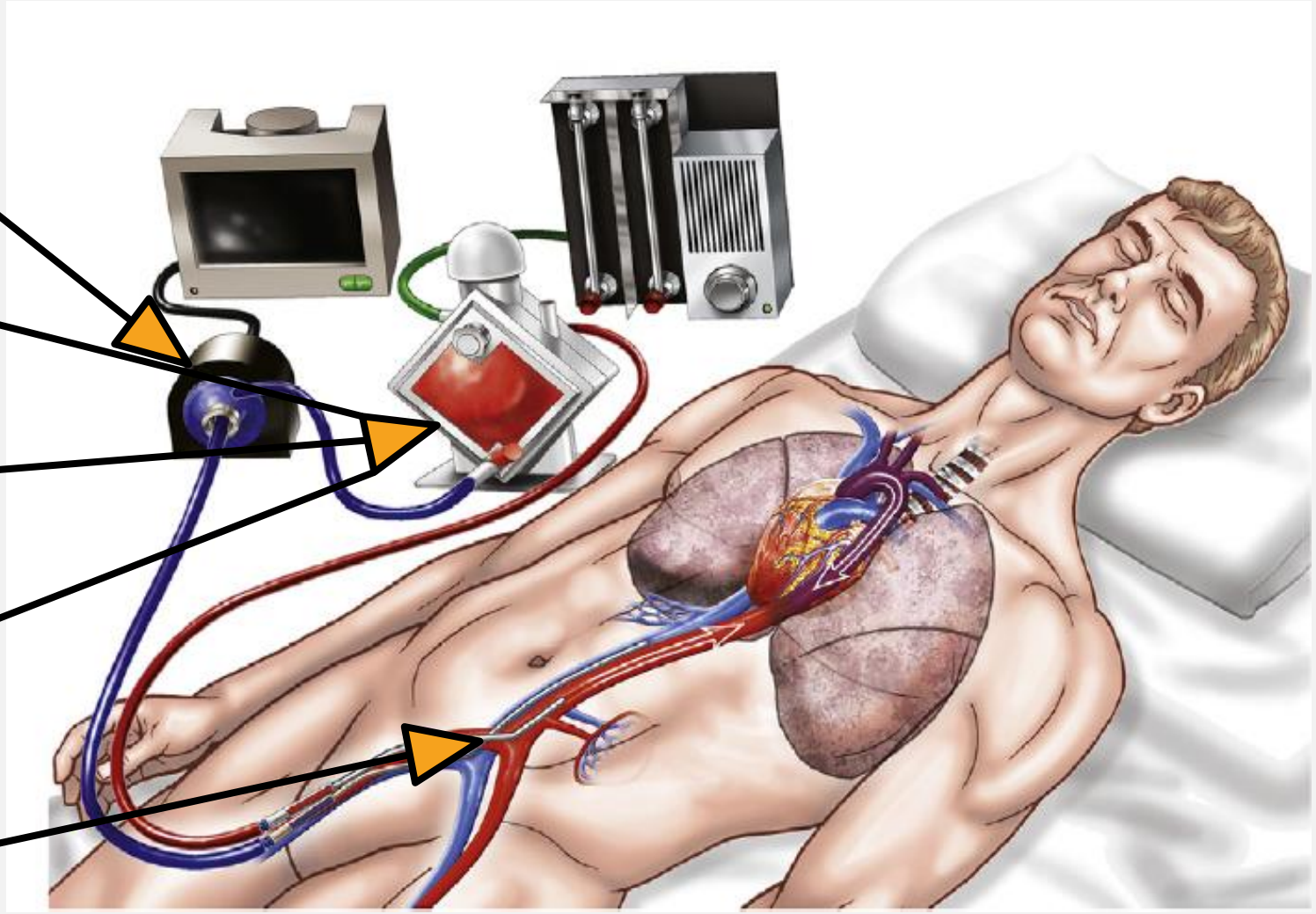
Débit non pulsatile

PaO_2 de 100 à 400 mmHg

Activation de l'inflammation

Activation de la coagulation

Circulation rétrograde



IMPACT D'UNE CIRCULATION RÉTROGRADE SUR LA FONCTION CARDIAQUE

Normal

Choc cardiogénique

↓Emax et ↓Ea

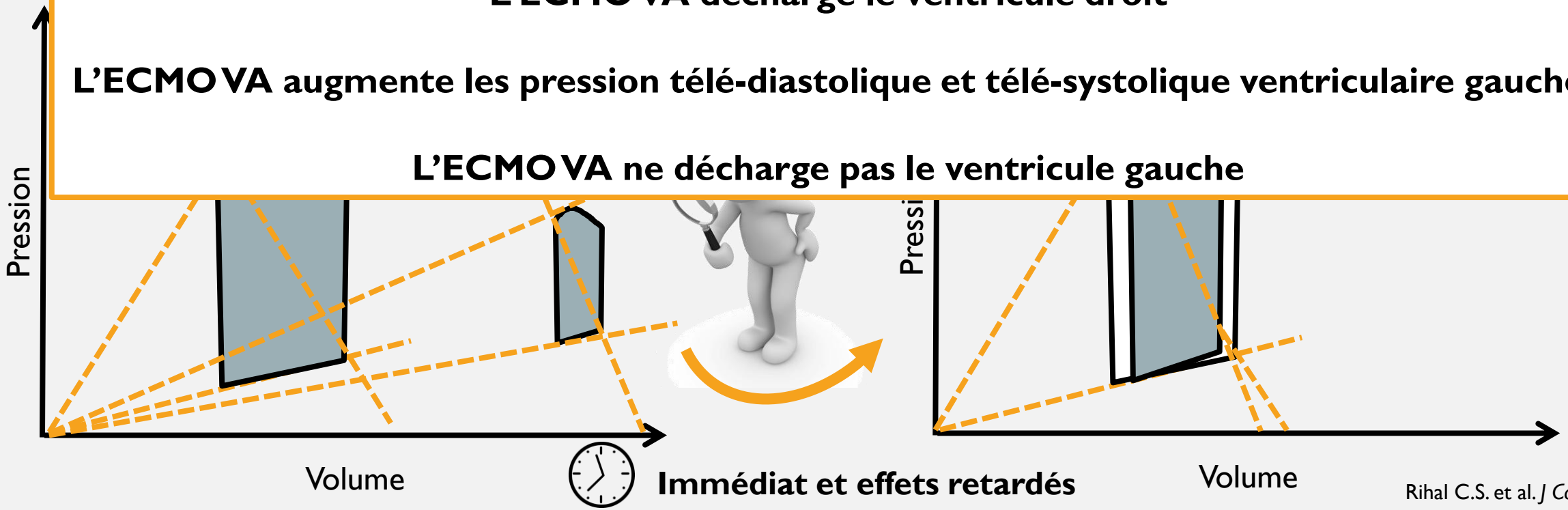
ECMO

= Emax ↓Ea ↓SV

L'ECMO VA décharge le ventricule droit

L'ECMO VA augmente les pression télé-diastolique et télé-systolique ventriculaire gauche

L'ECMO VA ne décharge pas le ventricule gauche

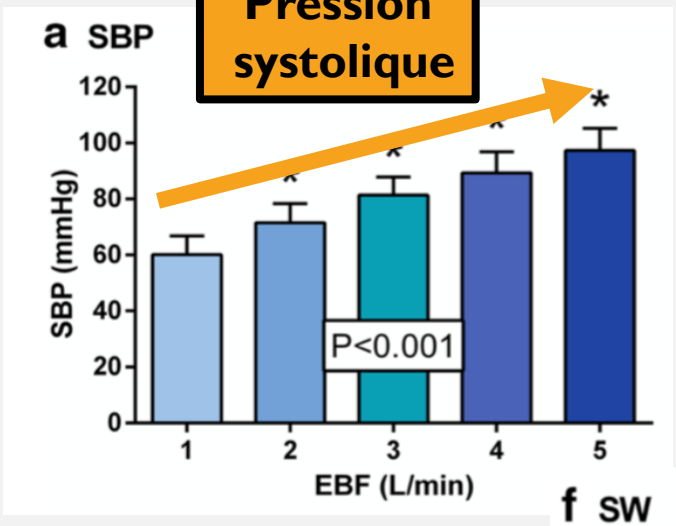


Immédiat et effets retardés

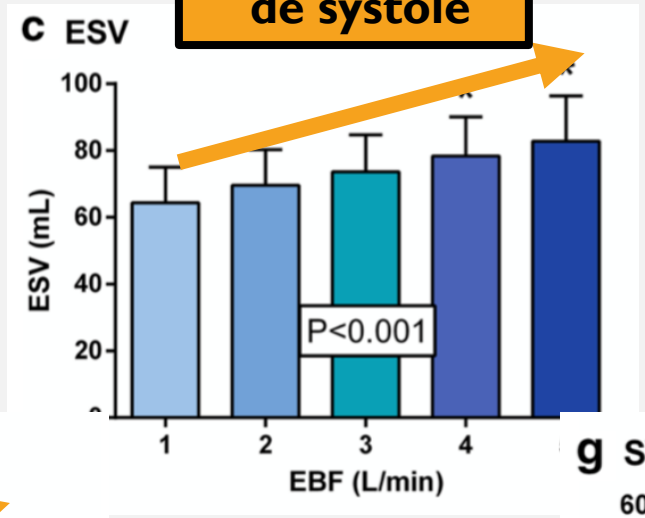
Rihal C.S. et al. *J Card Fail* 2015

PERFORMANCE MYOCARDIQUE ET DÉBIT D'ECMO VA

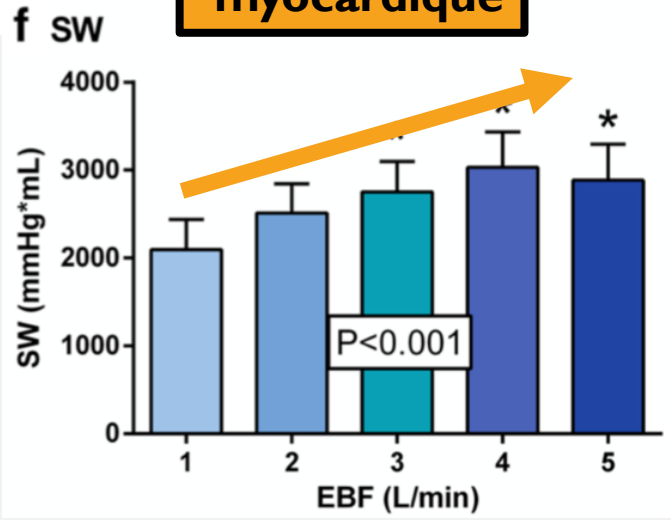
Pression systolique



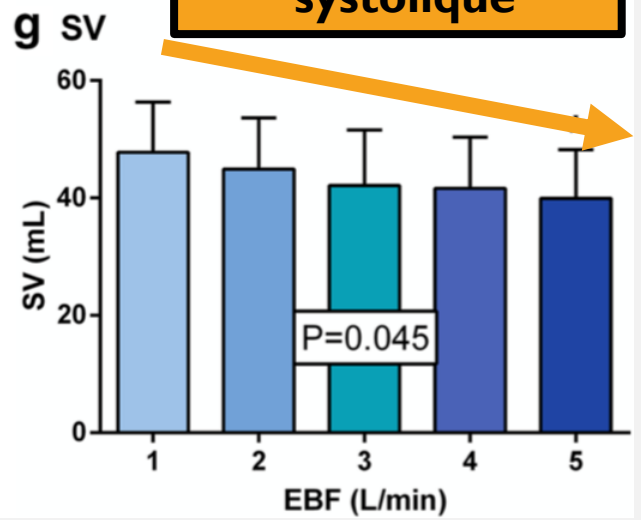
Volume de fin de systole



Travail myocardique



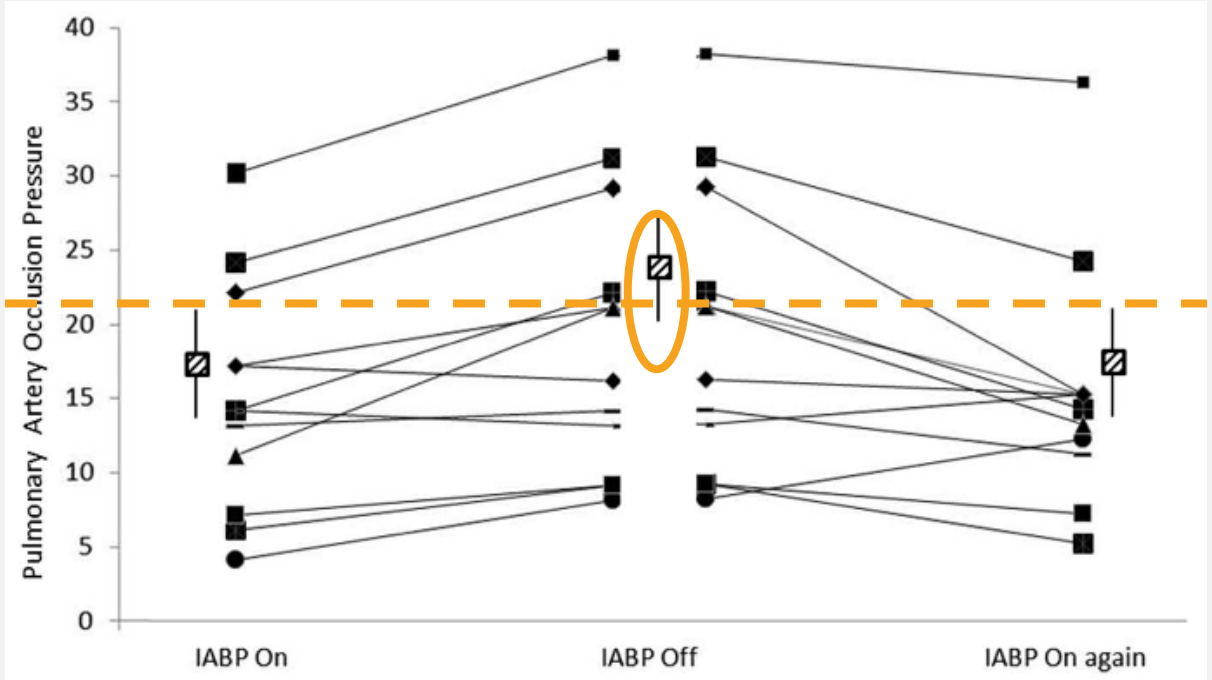
Volume d'éjection systolique



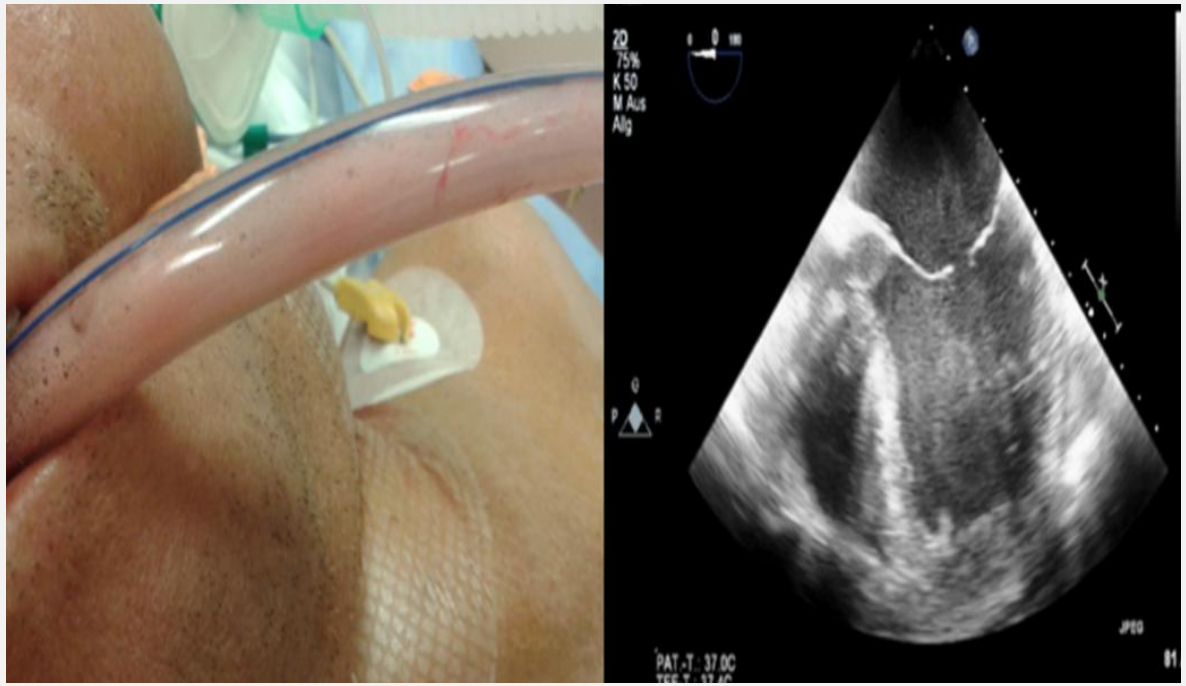
Ostadal et al. *J Transl Med* 2015

CONSÉQUENCES DE LA CIRCULATION RÉTROGRADE SUR LE VENTRICULE GAUCHE

24 mmHg



Œdème aigu pulmonaire

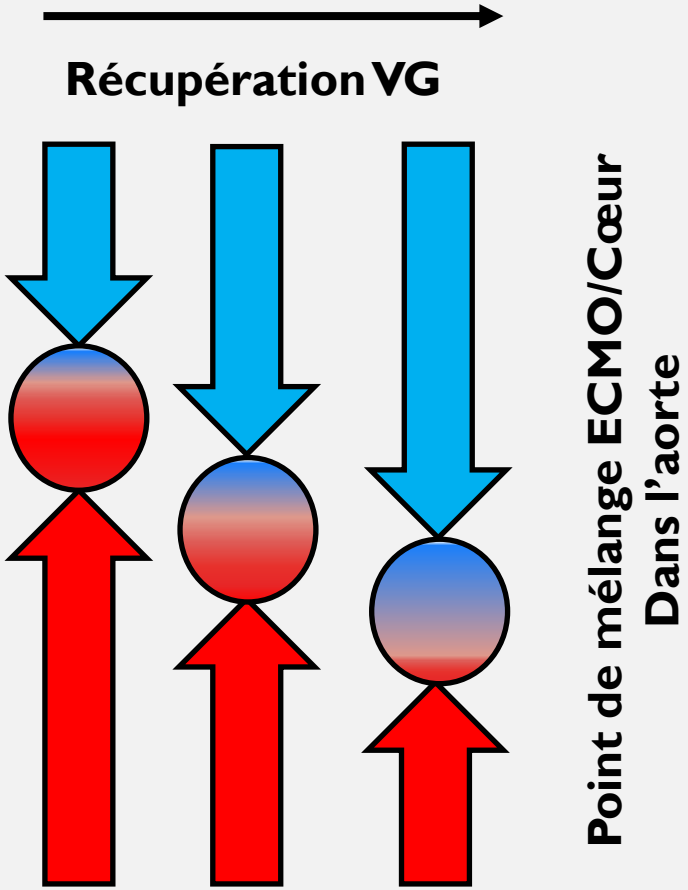


Immédiat et effets retardés

Petroni T et al. *Crit Care Med* 2014

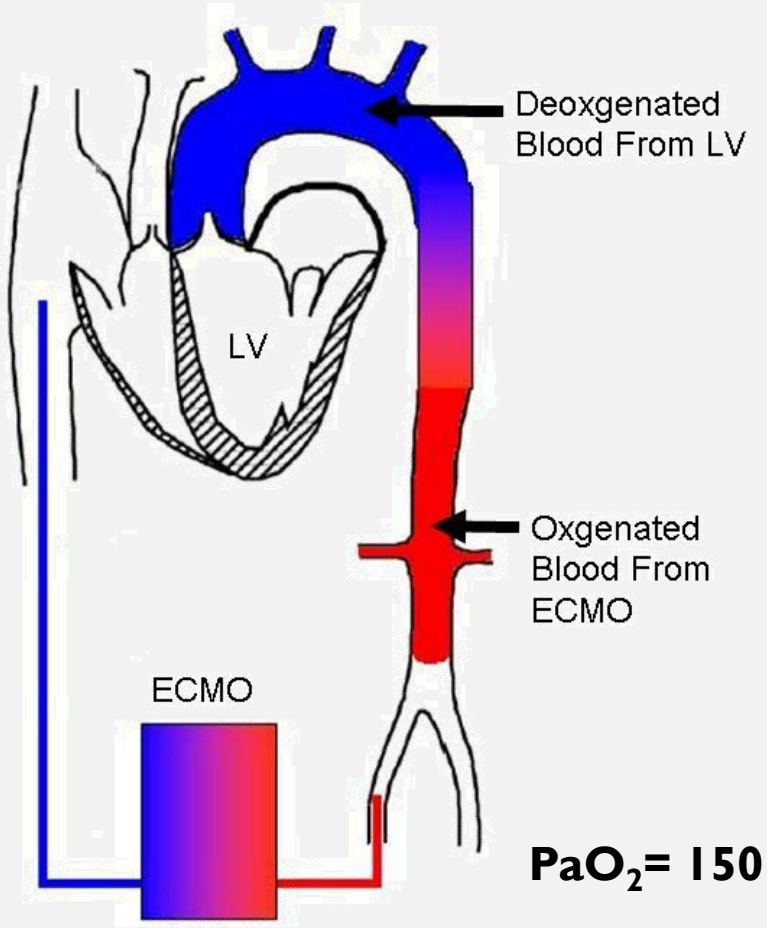
Weymann A et al. *J Cardiothorac Surg* 2014

SYNDROME ARLEQUIN



Point de mélange ECMO/Cœur Dans l'aorte

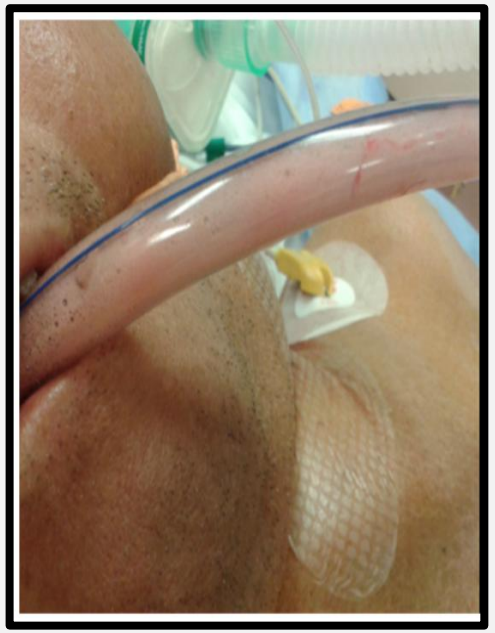
PaO_2= 50 mmHg



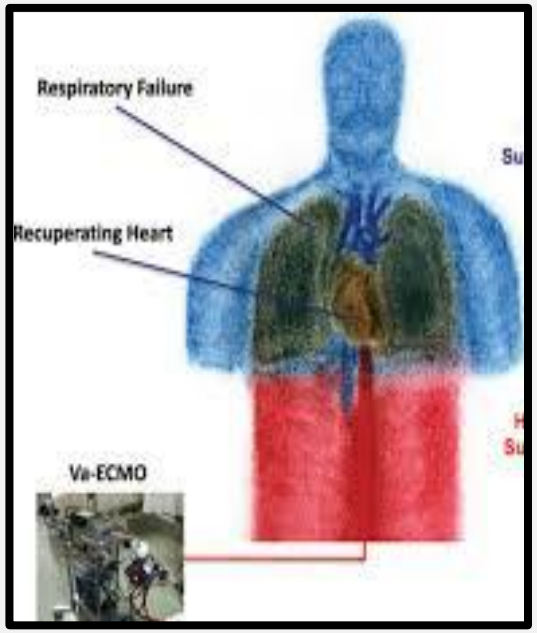
PaO_2= 150 mmHg

COMPLICATIONS

Réinjecte à "contre-courant"

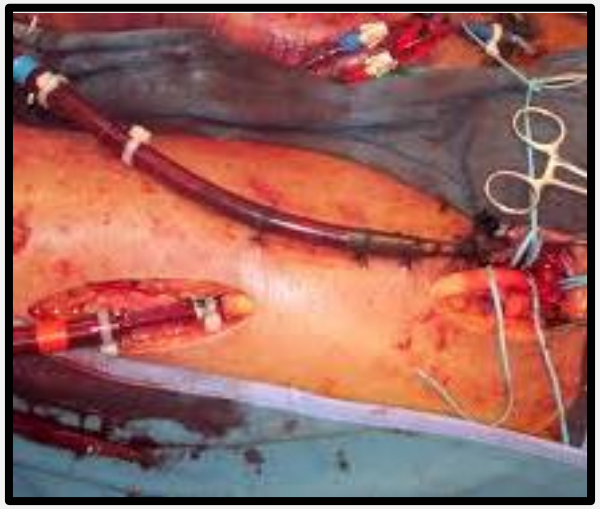


Œdème aigu hydrostatique

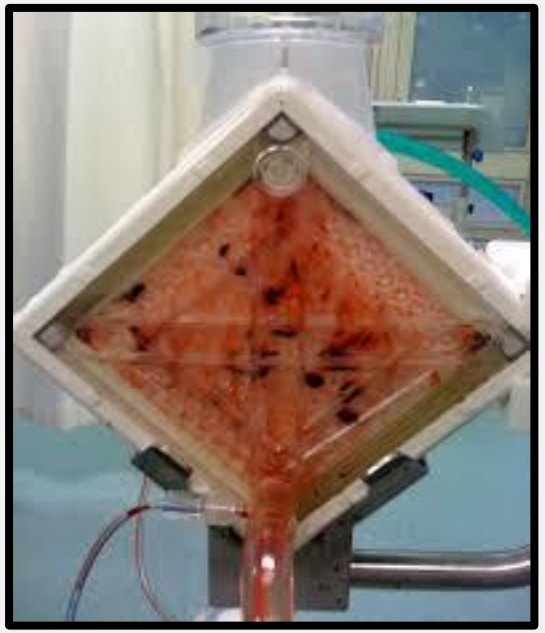


Syndrome Arlequin

Circulation Extra-Corporelle



Infections 90% à J25



Thrombopénie
Thrombose
Hémorragie

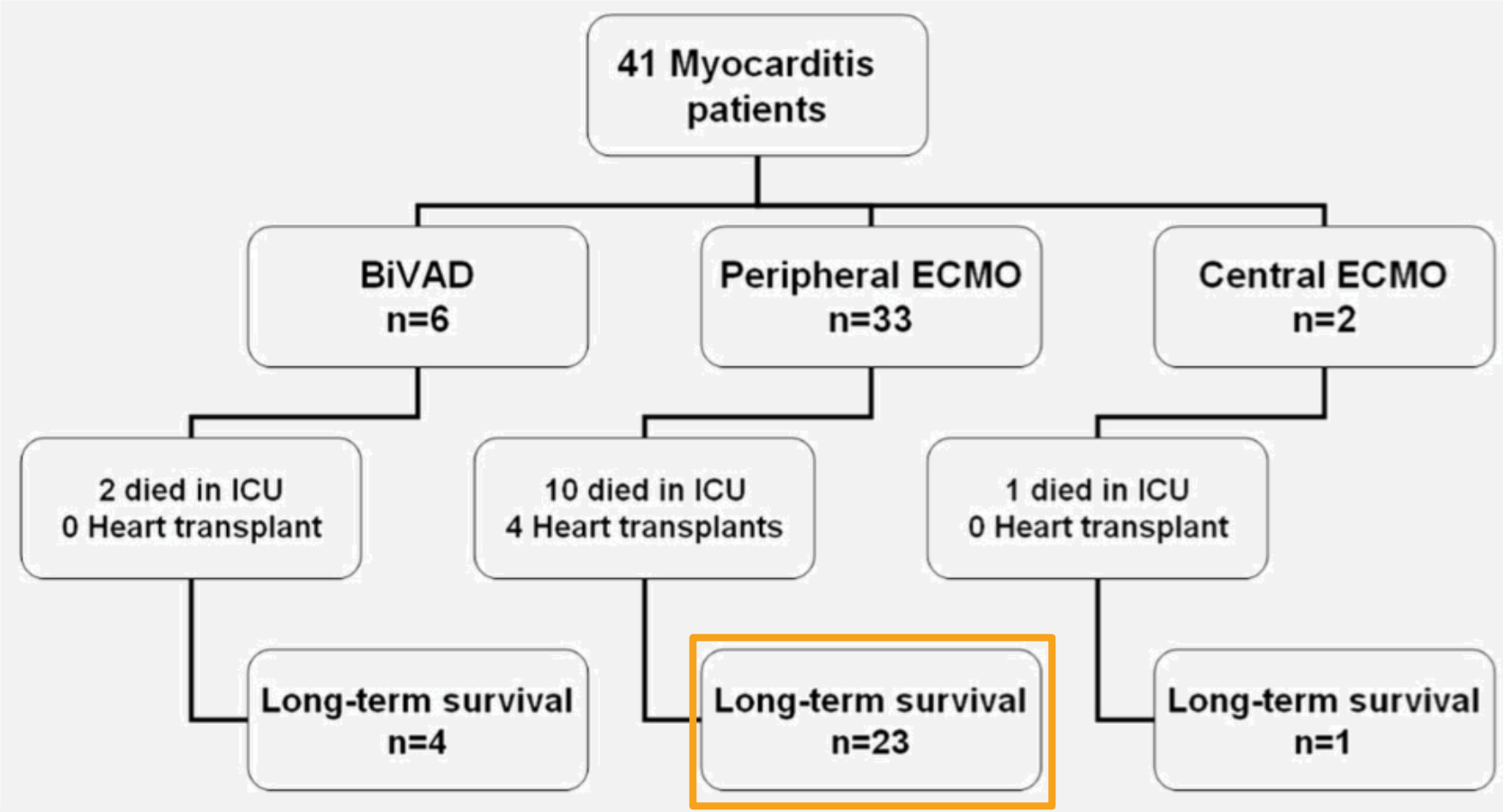
Weymann A et al. **Journal of Cardiothoracic Surgery** 2014 Lotz C et al. **Circulation** 2014 Schmidt M et al. **CID** 2012

ECLS: CAHIER DES CHARGES

Vrai débit circulatoire: PRIMORDIAL	✓
Facilité d'implantation < 20 min	✓
Décharge ventriculaire - droite - gauche	 ✓ ✗
Faible taux de complications	✗

ECMO VA: UNE THÉRAPEUTIQUE EFFICACE

MYOCARDITE



69% de survie

Mirabel M et al. **Crit Care Med** 2011

SYNDROME CORONARIEN AIGU

Table 1 Clinical characteristics of ECMO-treated AMI patients according to ICU survival status

Characteristic	All patients ($n = 138$)	Survivors ($n = 65$)	Non-survivors ($n = 73$)	*P* value
Age, years	55 (46–63)	53 (44–60)	58 (49–65)	0.050
Men	110 (80)	55 (85)	55 (75)	0.170
Body mass index, kg/m^2	26 (24–29)	25 (24–28)	26 (24–30)	0.060
SAPS II	66 (48–82)	60 (40–75)	73 (55–88)	<0.001
SOFA score	11 (8–15)	10 (7–12)	13 (9–16)	0.001
AMI onset to ECMO, days	1 (0–2)	1 (0–4)	1 (0–1)	0.280
Mobile ECMO retrieval	45 (33)	21 (32)	24 (33)	0.940
Pre-ECMO cardiac arrest	79 (57)	37 (57)	42 (58)	0.940
No-flow, min	0 (0–0)	0 (0–0)	0 (0–1)	0.460
Low-flow, min	16 (10–35)	15 (5–30)	29 (10–36)	0.570
Shockable rhythm	42/79 (53)	23/37 (62)	19/42 (45)	0.120
ECMO under CPR	19 (14)	9 (14)	10 (14)	0.980
Thrombolysis for AMI	16 (12)	6 (9)	10 (14)	0.410
AMI location				0.380
Anterior	93 (67)	44 (68)	49 (67)	
Inferior	33 (24)	14 (22)	19 (26)	
Other	12 (9)	7 (11)	5 (7)	
Patients with attempted PCI	112 (81)	54 (83)	58 (80)	0.590
Successful PCI	99/112 (88)	48/54 (89)	51/58 (88)	0.890
ECMO pre-PCI	14 (10)	8 (12)	6 (8)	0.460
ECMO to PCI, h	2 (1–6)	1 (1–5)	4 (1–24)	0.990
Post-PCI ECMO	124 (90)	57 (88)	67 (92)	0.660
PCI to ECMO, h	12 (4–24)	11 (3–24)	12 (5–24)	0.880

Survie ICU 47%

Sévère

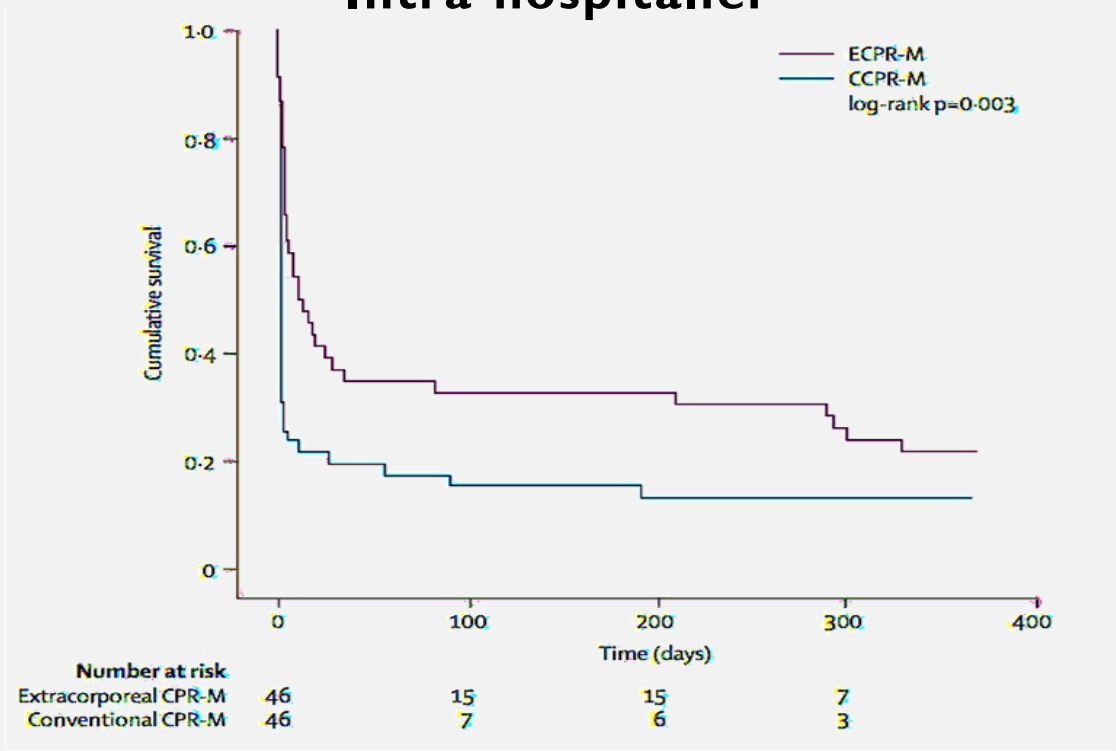
60% ont un ACR pré-canulation

90% canulés après angioplastie dans les 12H

Muller G et al. **Intensive care Med** 2016

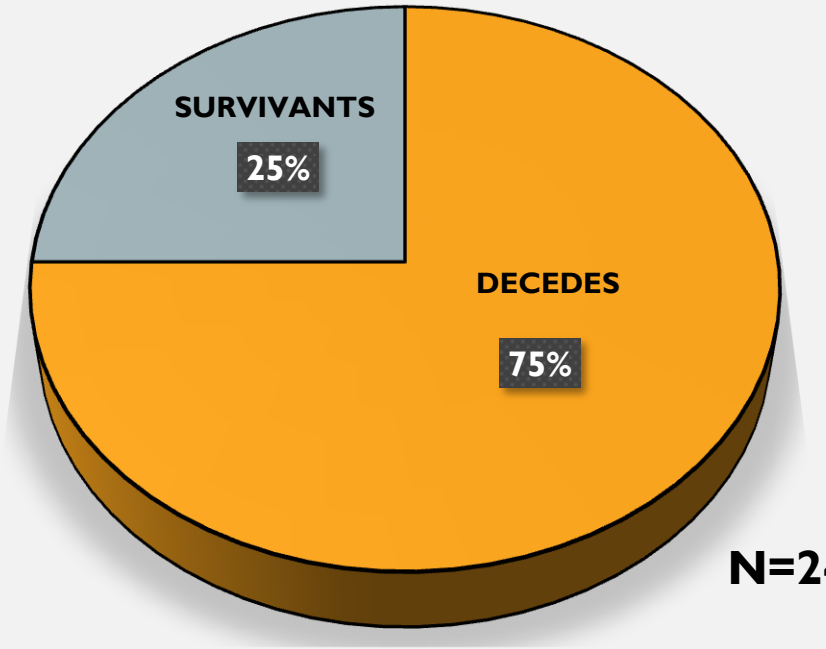
ARRÊT CARDIAQUE RÉFRACTAIRE

Intra-hospitalier



Extra-Hospitalier

N=8



N=24

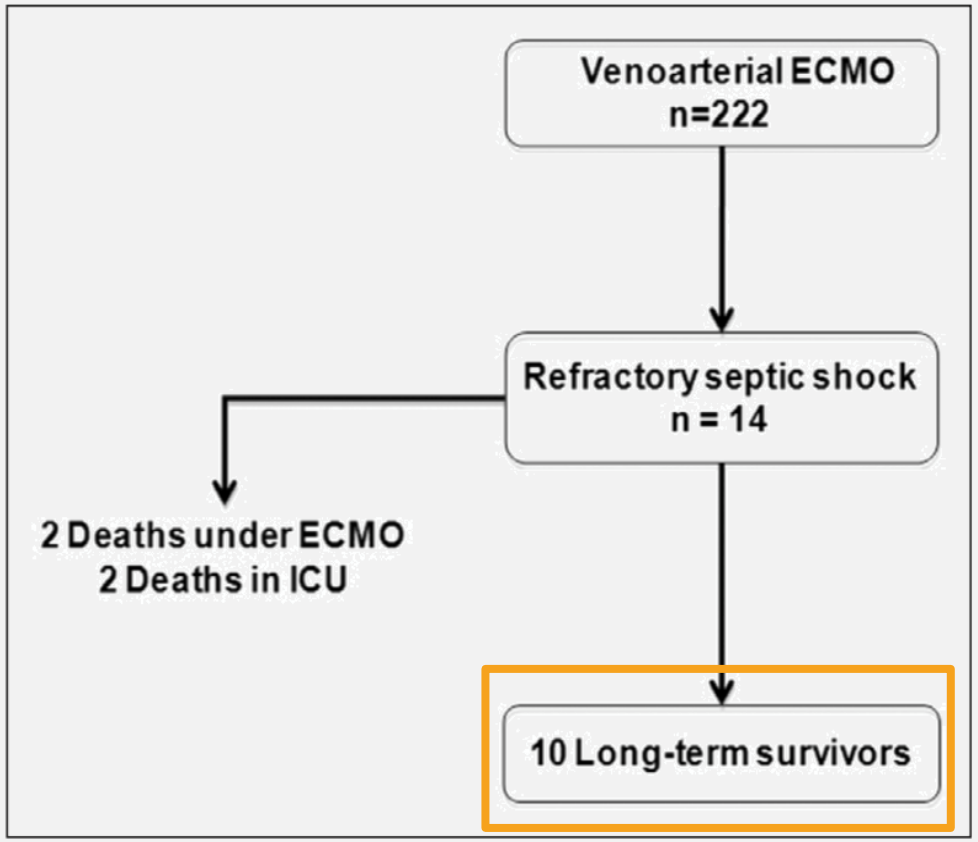
20 études, 833 patients,
22% de survie à la réanimation, 13% CPC 1

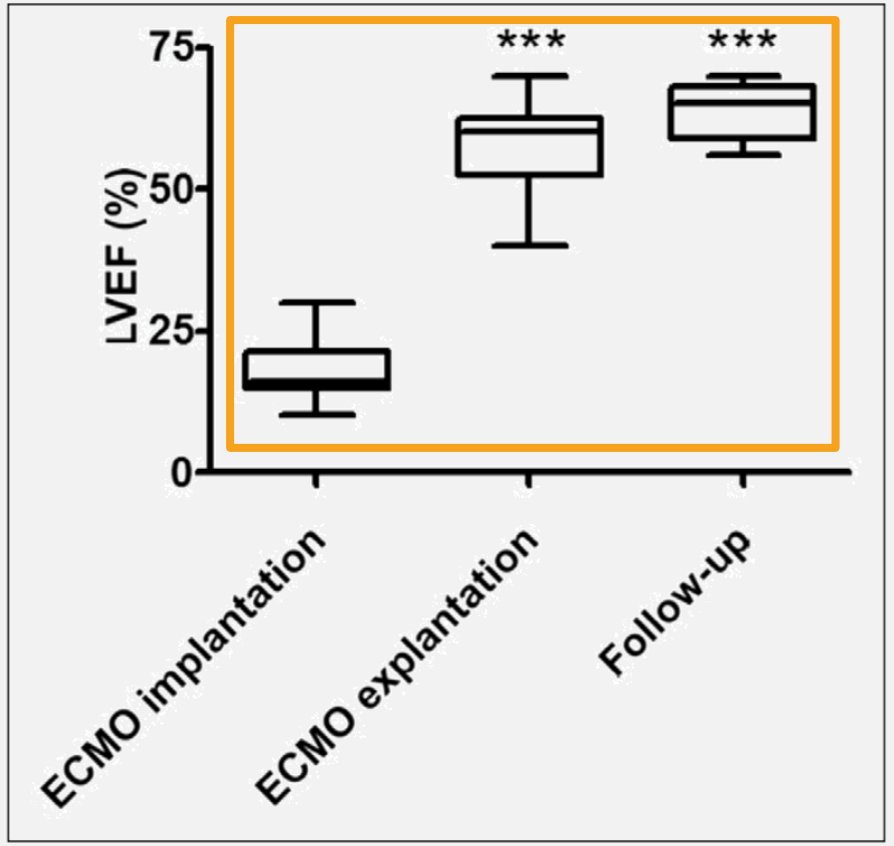
Chen Y-S et al. **Lancet** 2008

Ortega-Deballon I et al. **Resuscitation** 2016

Chouied et al. **Shock** 2018

CARDIOMYOPATHIE SEPTIQUE



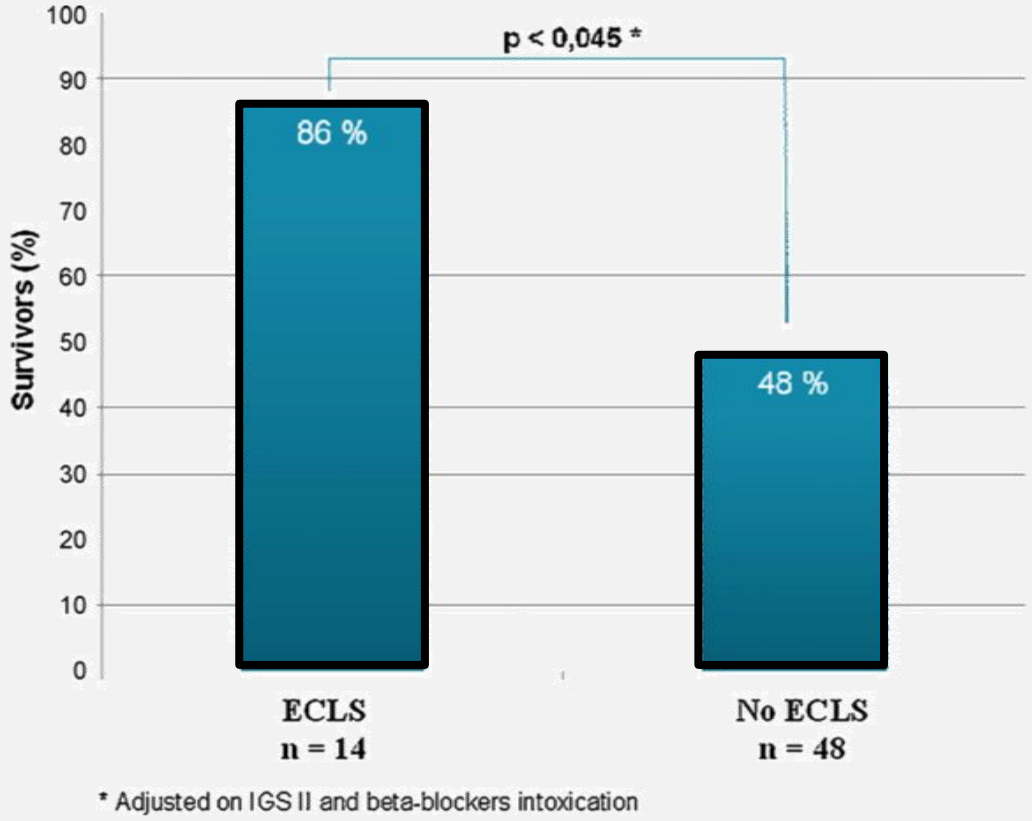


74% de survie

100 % de récupération de la fonction cardiaque

Brechot N et al. **Crit Care Med** 2013

INTOXICATION AUX CARDIOTROPES



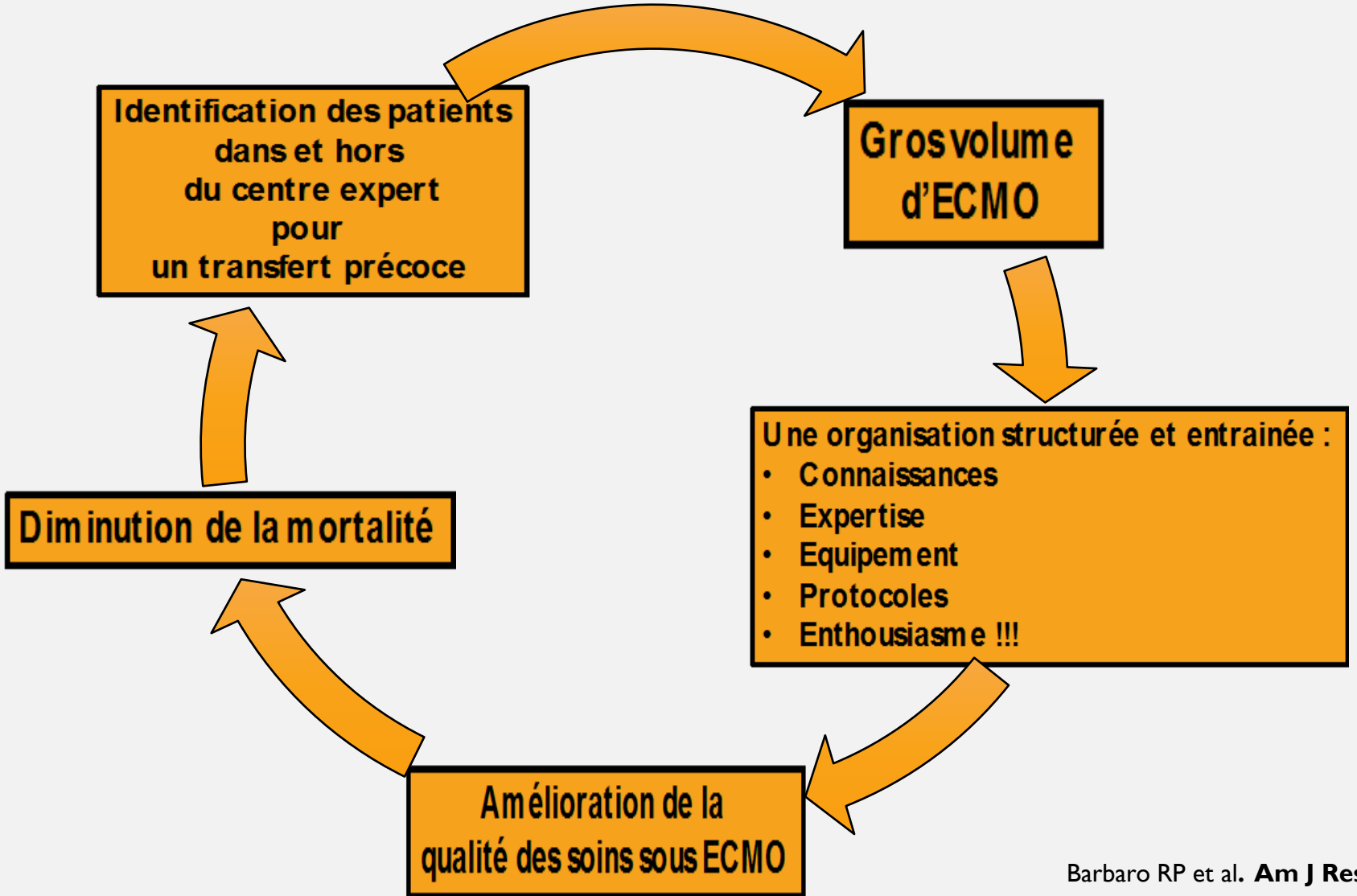
253 chocs sur 2350 intoxications

Pour les Beta bloquants +++

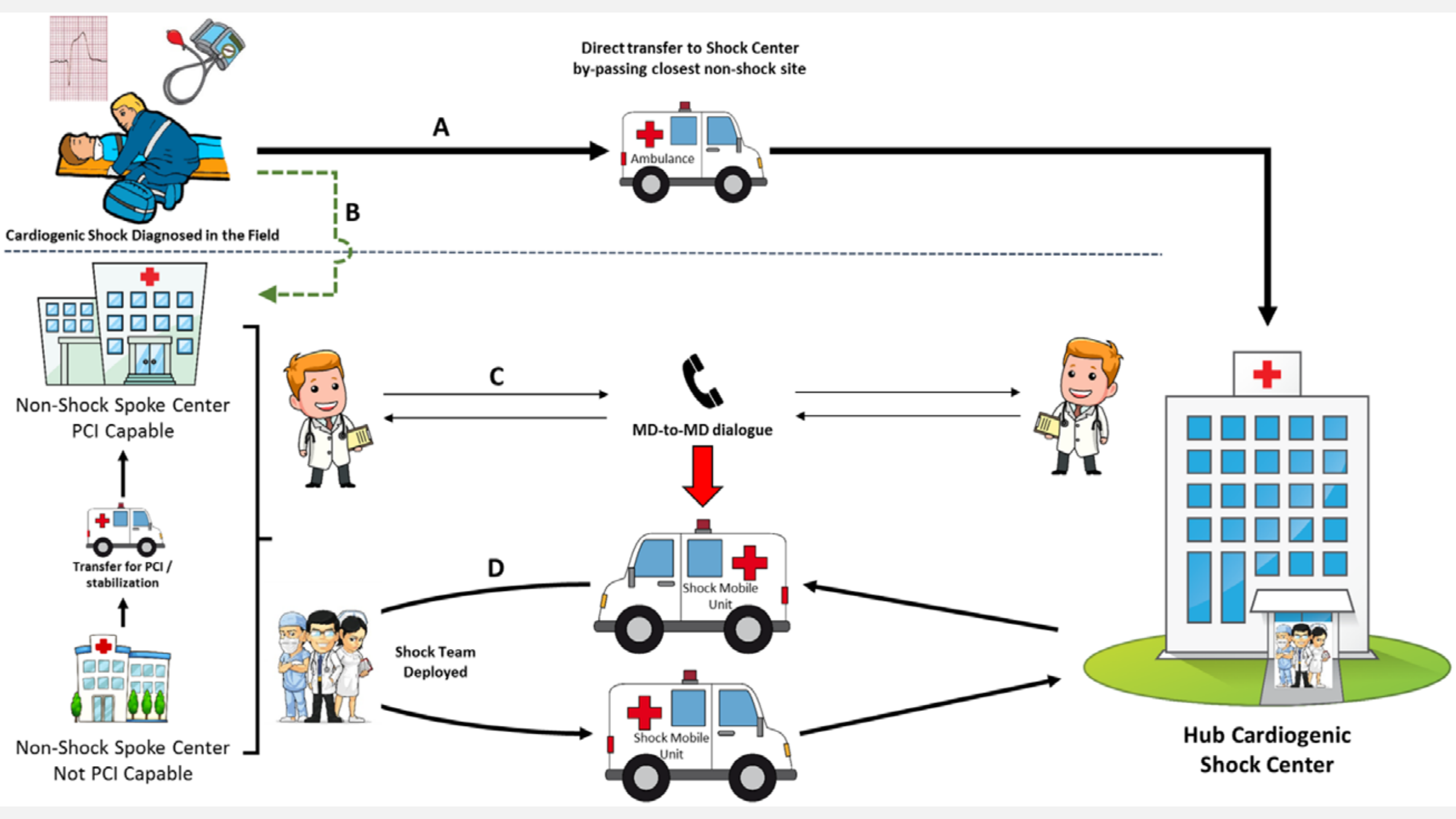
Survie 86% sous ECMO vs 48 %

Masson R et al. **Resuscitation** 2012

UN AVENIR OPTIMISTE ET VERTUEUX SI :



Barbaro RP et al. **Am J Respir Crit Care Med** 2015


Direct transfer to Shock Center
by-passing closest non-shock site
A
Ambulance
B
Cardiogenic Shock Diagnosed in the Field
Non-Shock Spoke Center
PCI Capable
C
MD-to-MD dialogue
Transfer for PCI /
stabilization
D
Shock Mobile
Unit
Shock Team
Deployed
Shock Mobile
Unit
Non-Shock Spoke Center
Not PCI Capable
Hub Cardiogenic
Shock Center