Diagnostic & épidémiologie du choc cardiogénique

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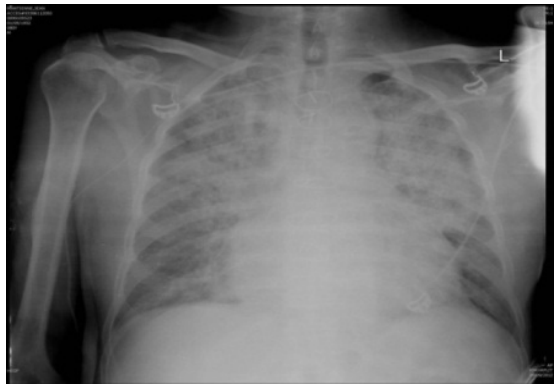
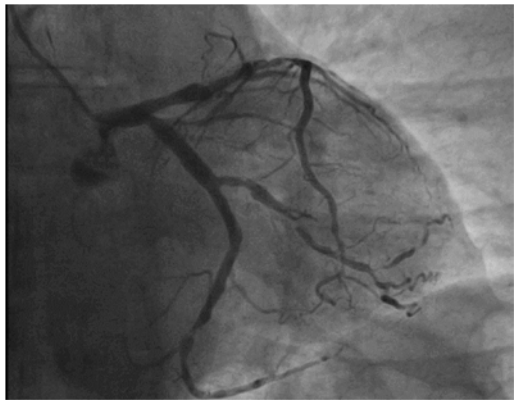
Université Paris Cité

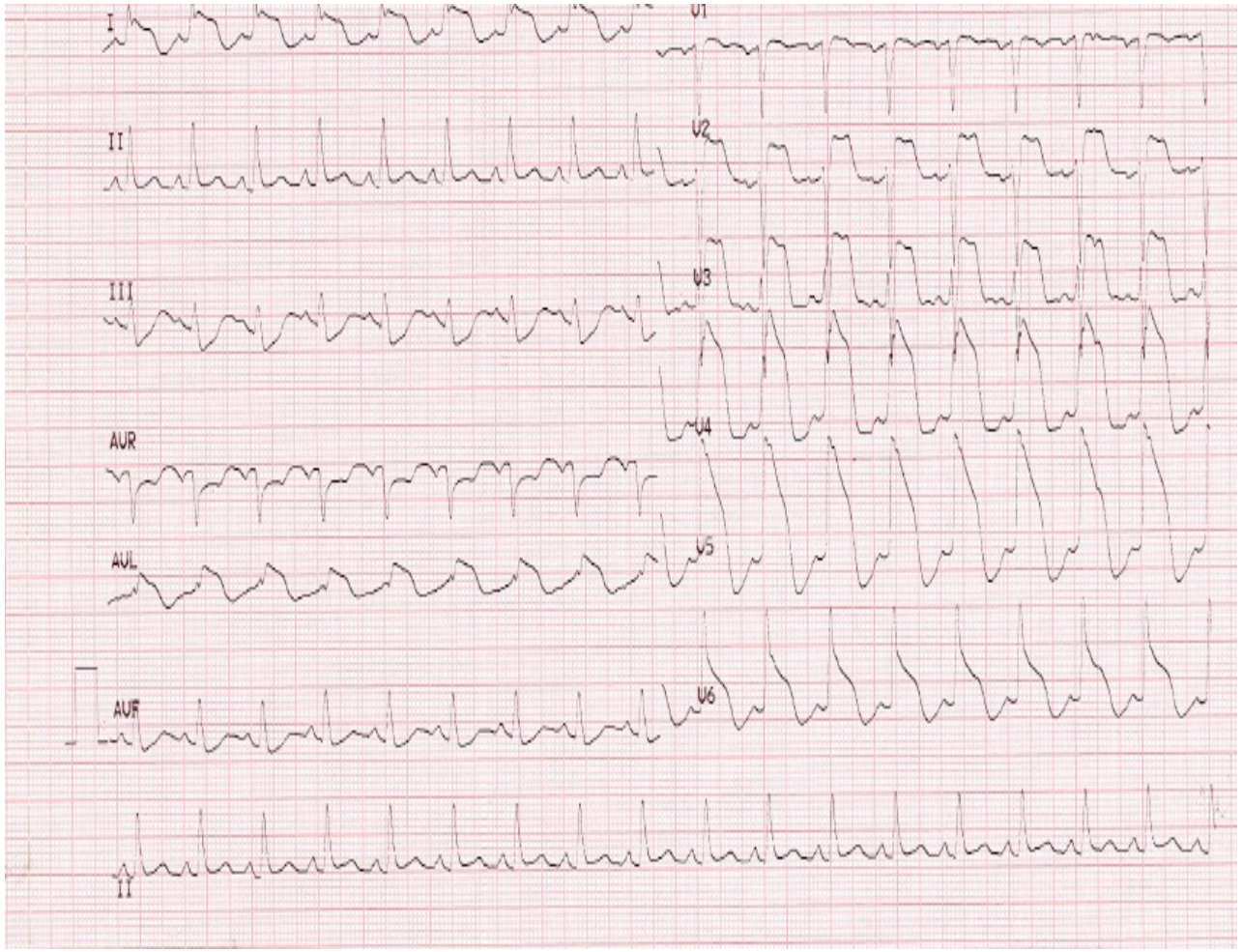
Conflits d’intérêt : aucun

Le choc cardiogénique il y a 20 ans….



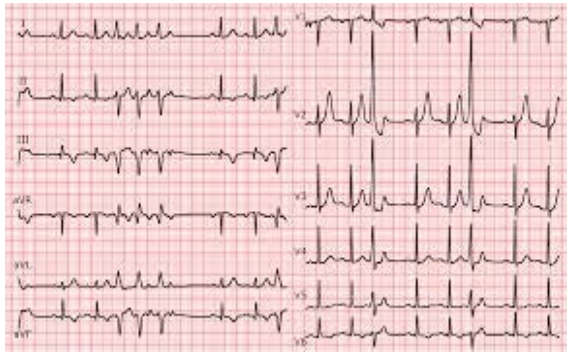


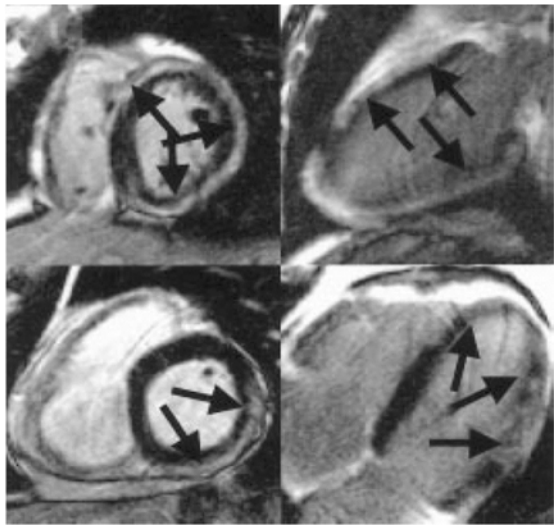
- TA 80/60 mmHg, FC 100/min
- Dyspnée dans un contexte de douleurs thoraciques
- Crépitants des bases
- Genoux froids
- Bilan biologique : pH 7,36, lactate à 3 mmol/L, créatinine à 120 mmol/L, urée 10 mmol/L, facteur V normal, bilirubine 12



USIC, HEGP le 21 novembre 2024

Chambre 8, Iliyan , 17 ans





USIC, HEGP le 21 novembre 2024

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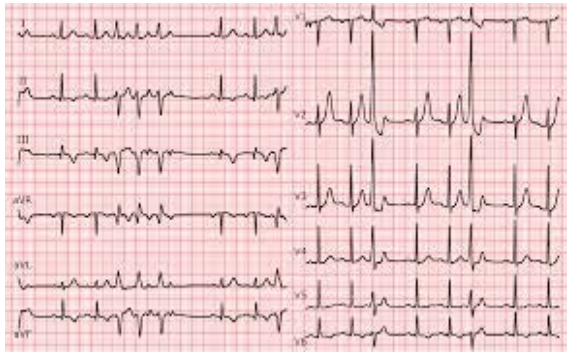
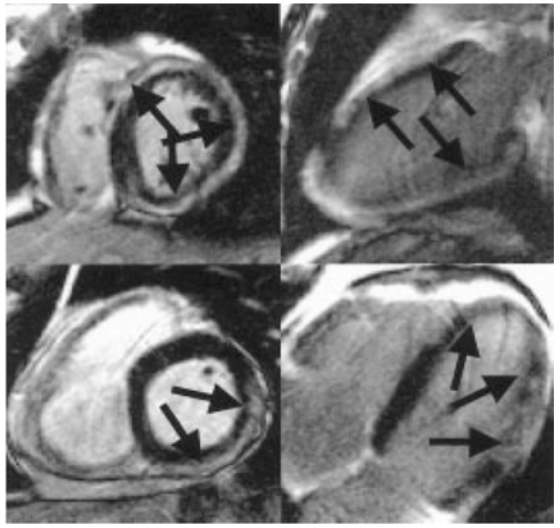
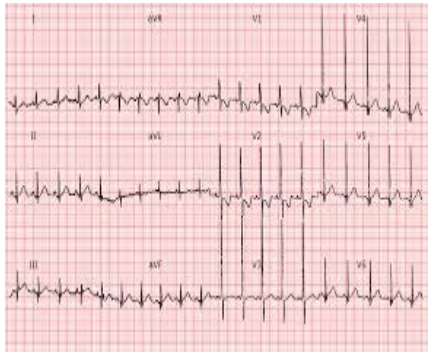
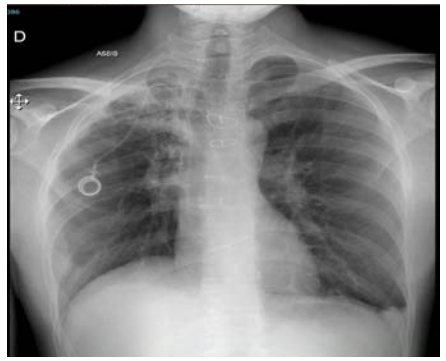
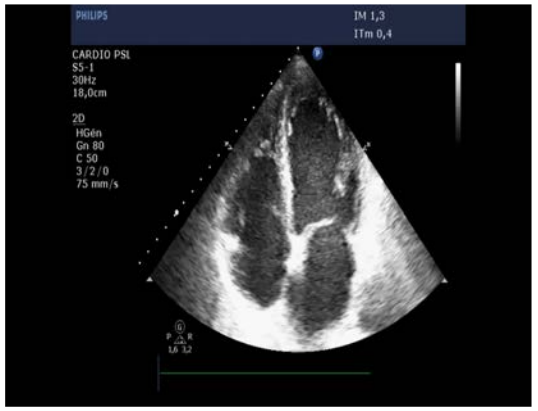




Chambre 11, Mohammed 23 ans

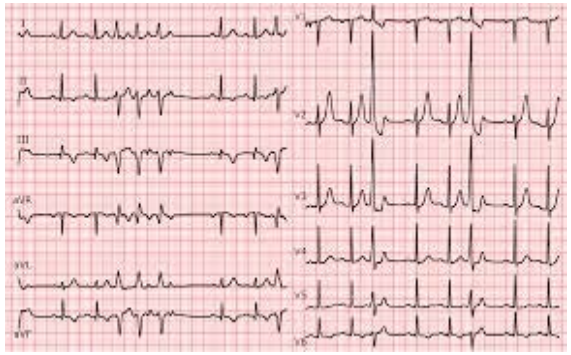


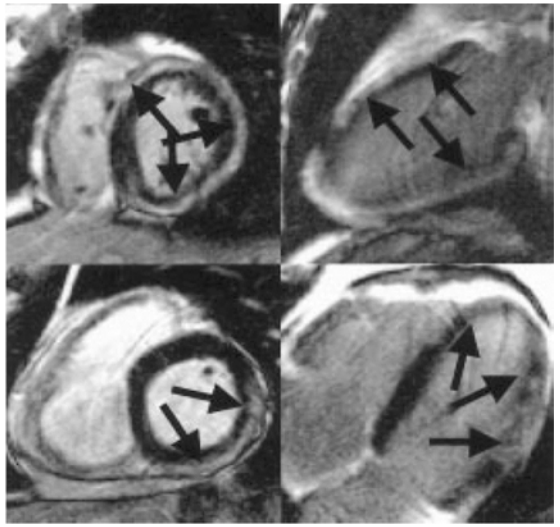




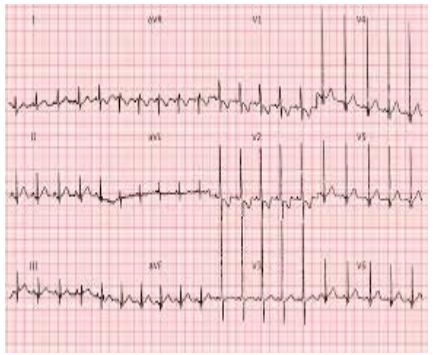
USIC, HEGP le 21 novembre 2024

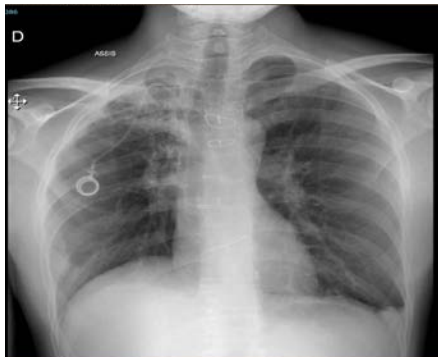
Chambre 8, Iliyan , 17 ans

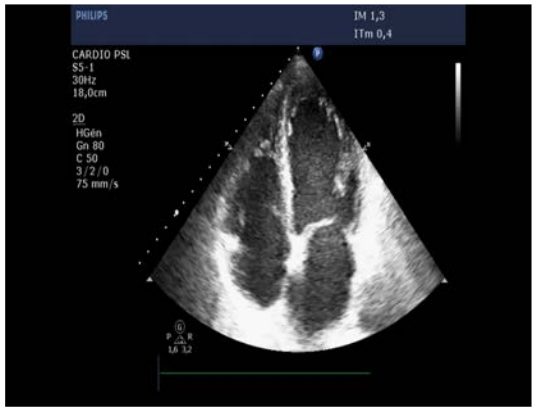




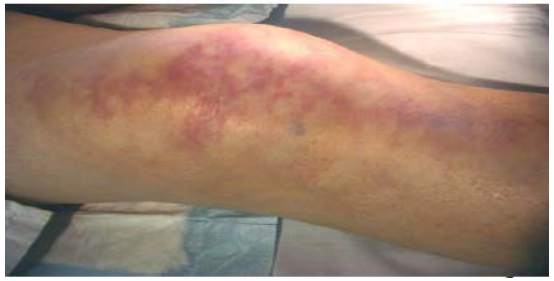
Chambre 11, Mohammed 23 ans

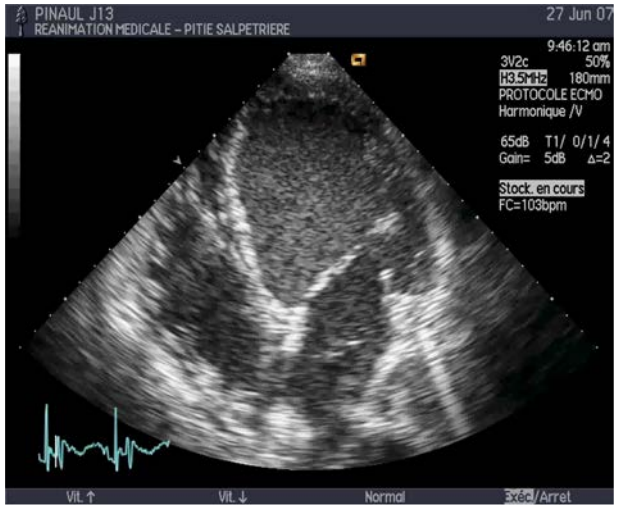






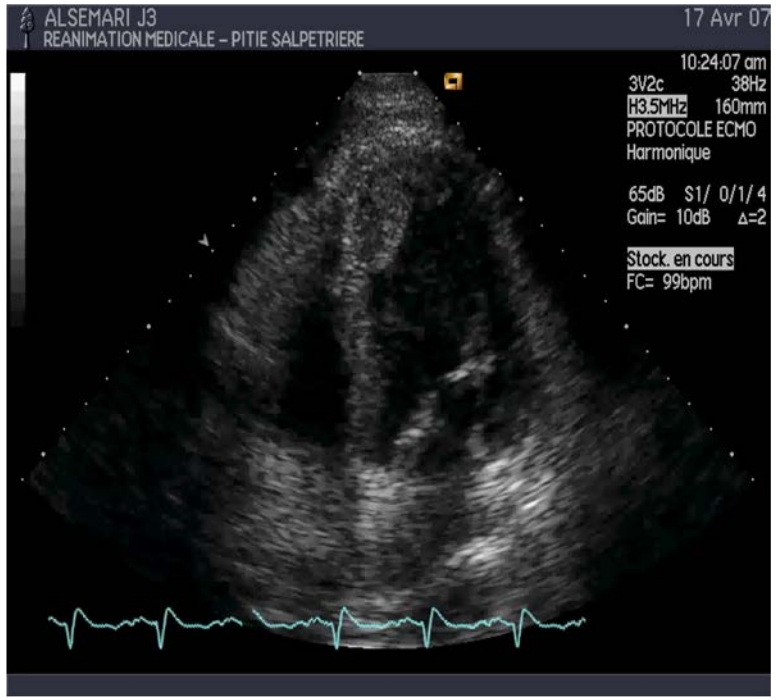
Chambre 16, Pierre 58 ans

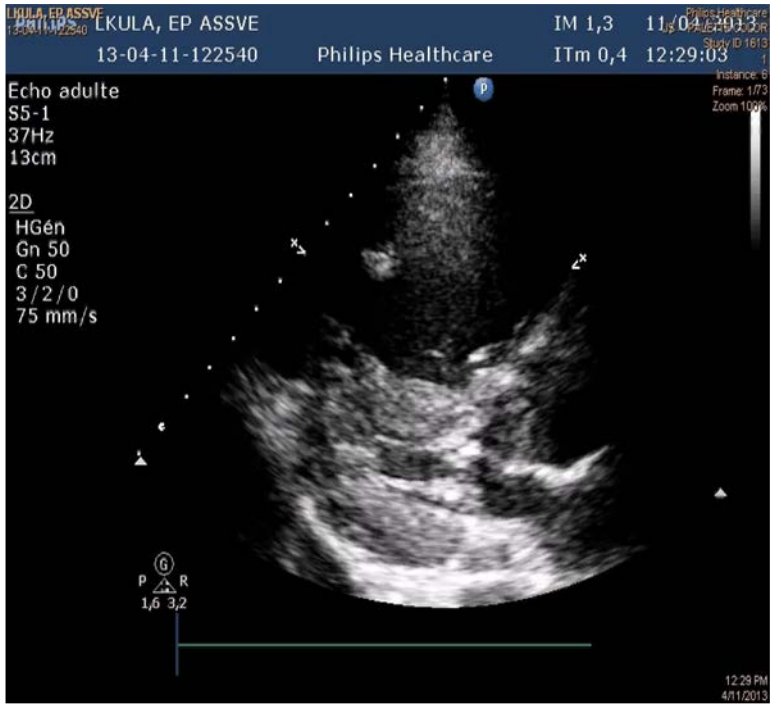


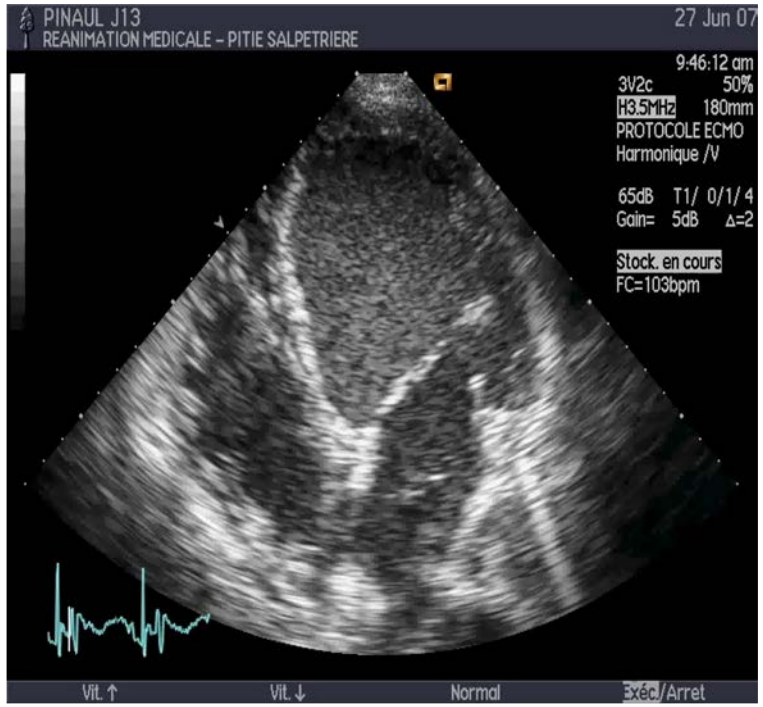


Un seul diagnostic : le choc cardiogénique

Le choc cardiogénique







Insuffisance circulatoire provoquant un déséquilibre entre transport en oxygène et besoins métaboliques, lié à une diminution du débit cardiaque, en l'absence d'hypovolémie

Circulation. 2017;136:e232–e268.

REVIEW Open Access

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

- Une pression artérielle systolique inférieure à 90 mmHg ou une pression artérielle moyenne inférieure à 65 mmHg pendant 30 minutes ;
- Une congestion pulmonaire ou une élévation des pressions de remplissage ;
- Des signes d'hypoperfusion périphérique : (a) confusion; (b) genoux froids et/ou marbrés; (c) oligurie; (d) lactate augmenté

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

De nombreuses définitons

Clinical Definition	SHOCK Trial[9]*	IABP-SHOCK II[1]†	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 $L \cdot min^{-1} \cdot m^{-2}$ 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

Van Diepen.S, Circulation Sept 2017

- Basées sur le choc ischémique
- Patient trop évolué (pré-choc…)

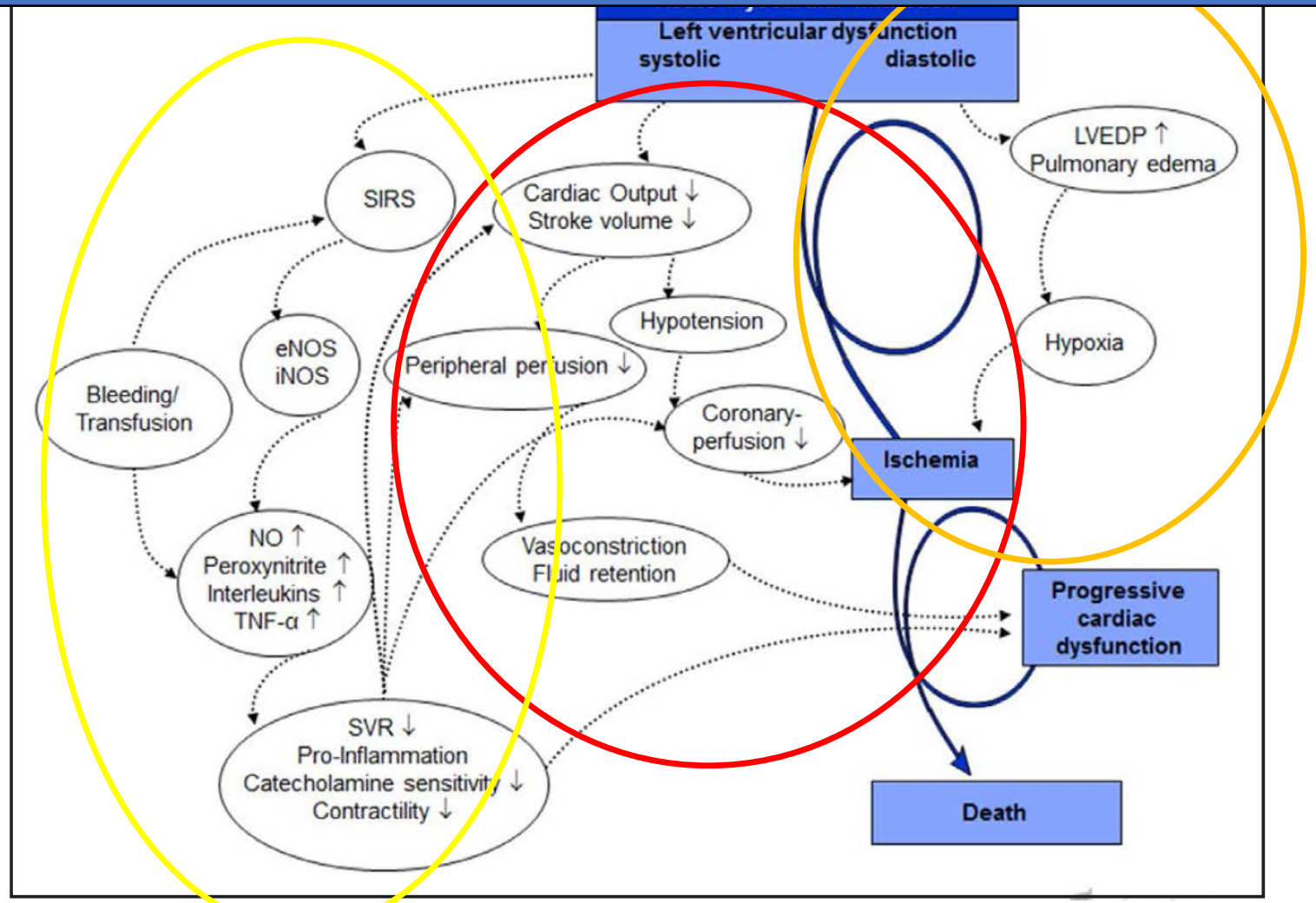
ESC European Society of Cardiology
European Heart Journal (2021) 42, 3599–3726
doi:10.1093/eurheartj/ehab368

2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

	Acute decompensated heart failure	Acute pulmonary oedema	Isolated right ventricular failure	Cardiogenic shock
Main mechanisms	LV dysfunction Sodium and water renal retention	Increased afterload and/or predominant LV diastolic dysfunction Valvular heart disease	RV dysfunction and/or pre-capillary pulmonary hypertension	Severe cardiac dysfunction
Main cause of symptoms	Fluid accumulation, increased intraventricular pressure	Fluid redistribution to the lungs and acute respiratory failure	Increased central venous pressure and often systemic hypoperfusion	Systemic hypoperfusion
Onset	Gradual (days)	Rapid (hours)	Gradual or rapid	Gradual or rapid
Main haemodynamic abnormalities	Increased LVEDP and PCWP[a] Low or normal cardiac output Normal to low SBP	Increased LVEDP and PCWP[a] Normal cardiac output Normal to high SBP	Increased RVEDP Low cardiac output Low SBP	Increased LVEDP and PCWP[a] Low cardiac output Low SBP
Main clinical presentations[1,446]	Wet and warm OR Wet and cold	Wet and warm[b]	Wet and cold	Wet and cold
Main treatment	Diuretics Inotropic agents/vasopressors (if peripheral hypoperfusion/hypotension) Short-term MCS or RRT if needed	Diuretics Vasodilators[b]	Diuretics for peripheral congestion Inotropic agents/vasopressors (if peripheral hypoperfusion/hypotension) Short-term MCS or RRT if needed	Inotropic agents/vasopressors Short-term MCS RRT

Cardiogenic shock is a syndrome due to primary cardiac dysfunction resulting in an inadequate cardiac output, comprising a life-threatening state of tissue hypoperfusion, which can result in multi-organ failure and death.[450–452] Cardiac insult causing severe impairment of cardiac performance may be acute, as a result of the acute loss of myocardial tissue (acute MI, myocarditis) or may be progressive as seen in patients with chronic decompensated HF who may experience a decline in disease stability as a result of the natural progression of advanced HF and/or specific precipitants.[426]

Physiopathologie usuelle



Van Diepen.S et al, Circulation 2017

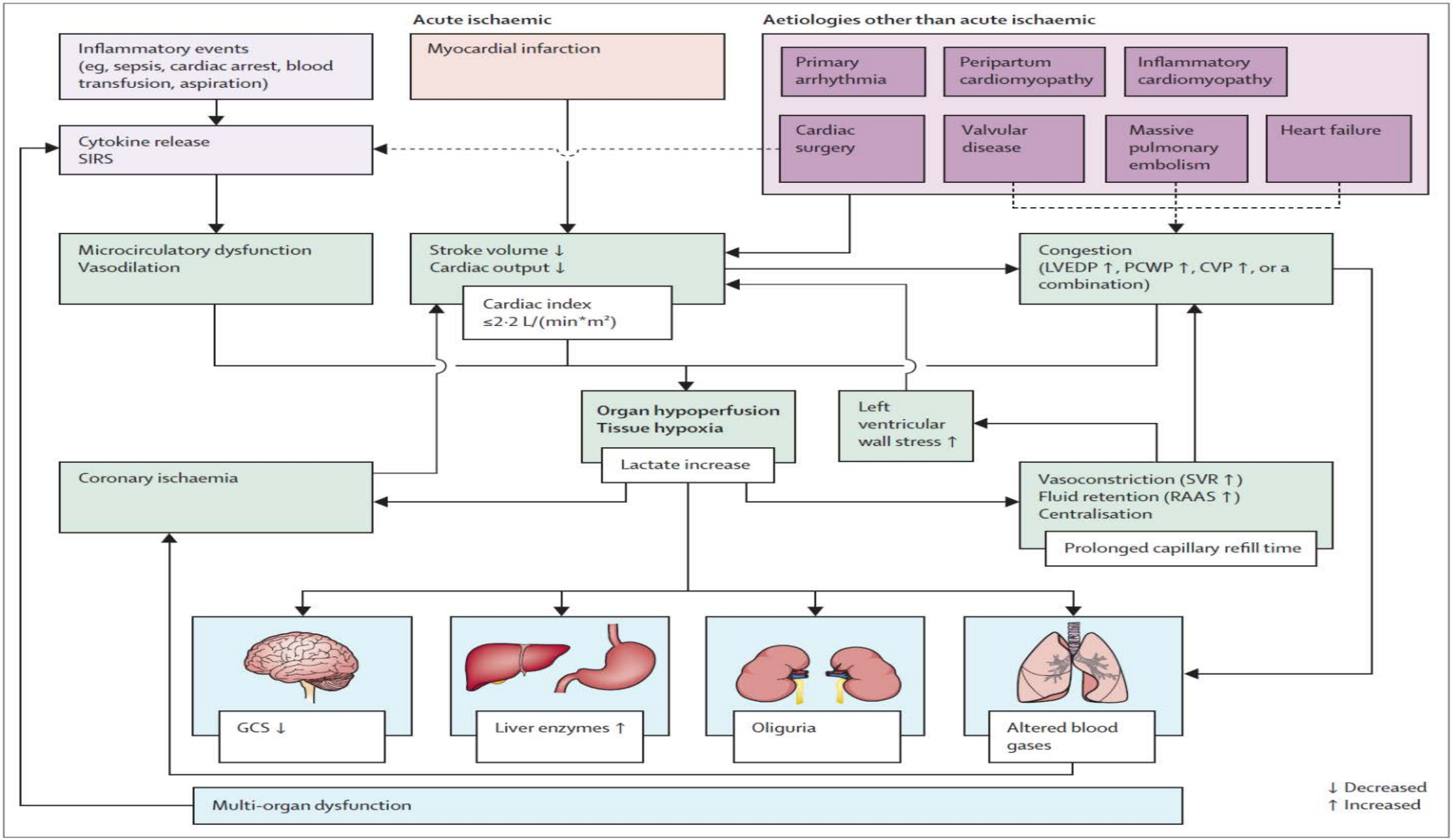
Dysfonction systolique

Dysfonction diastolique

SIRS

+ Congestion

Arjola VP. Eur J Heart Failure 2018



Lüsebrink, Enzo et al. The Lancet, 2024, 404, 10466, 2006 - 2020

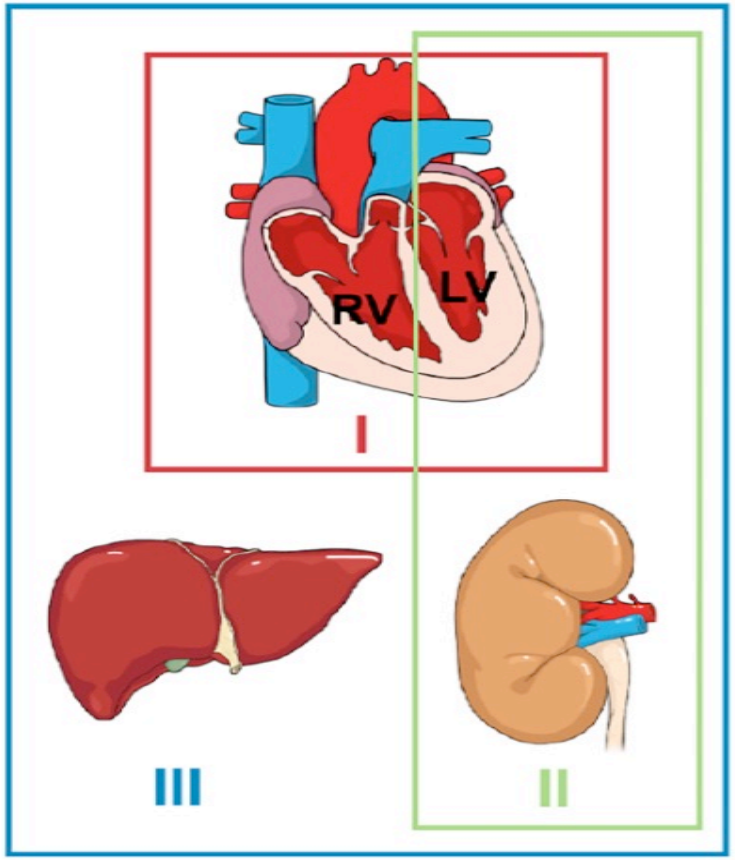
Différentes présentations cliniques ; impact thérapeutique et pronostique

REVIEW Open Access

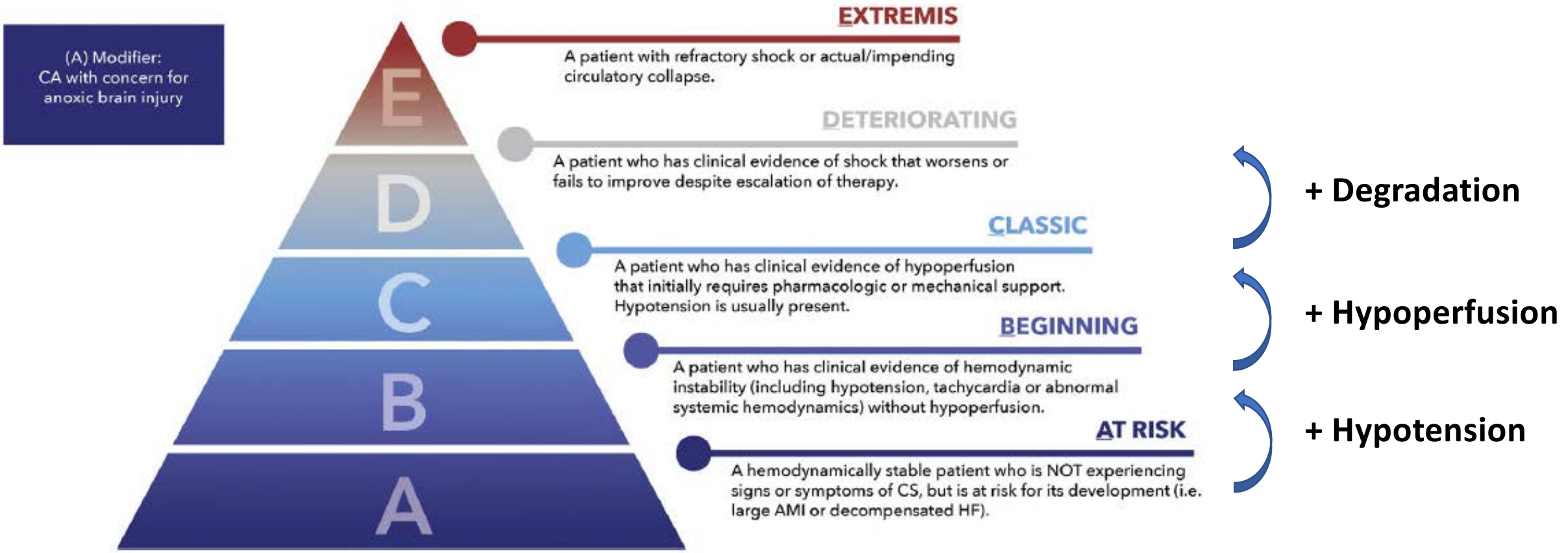
Management of cardiogenic shock: a narrative review

Driss Laghlam[1*], Sarah Benghanem[2,3,10], Sofia Ortuno[4,5], Nadia Bouabdallaoui[6], Stephane Manzo-Silberman[5,7], Olfa Hamzaoui[8,9] and Nadia Aissaoui[2,3,10]

Peripheral Circulation	Volume Status: Wet	Volume Status: Dry
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)



International experts consensus definition/classification of CS



Moosamy.P et al, J Am Col Cardiol 2022

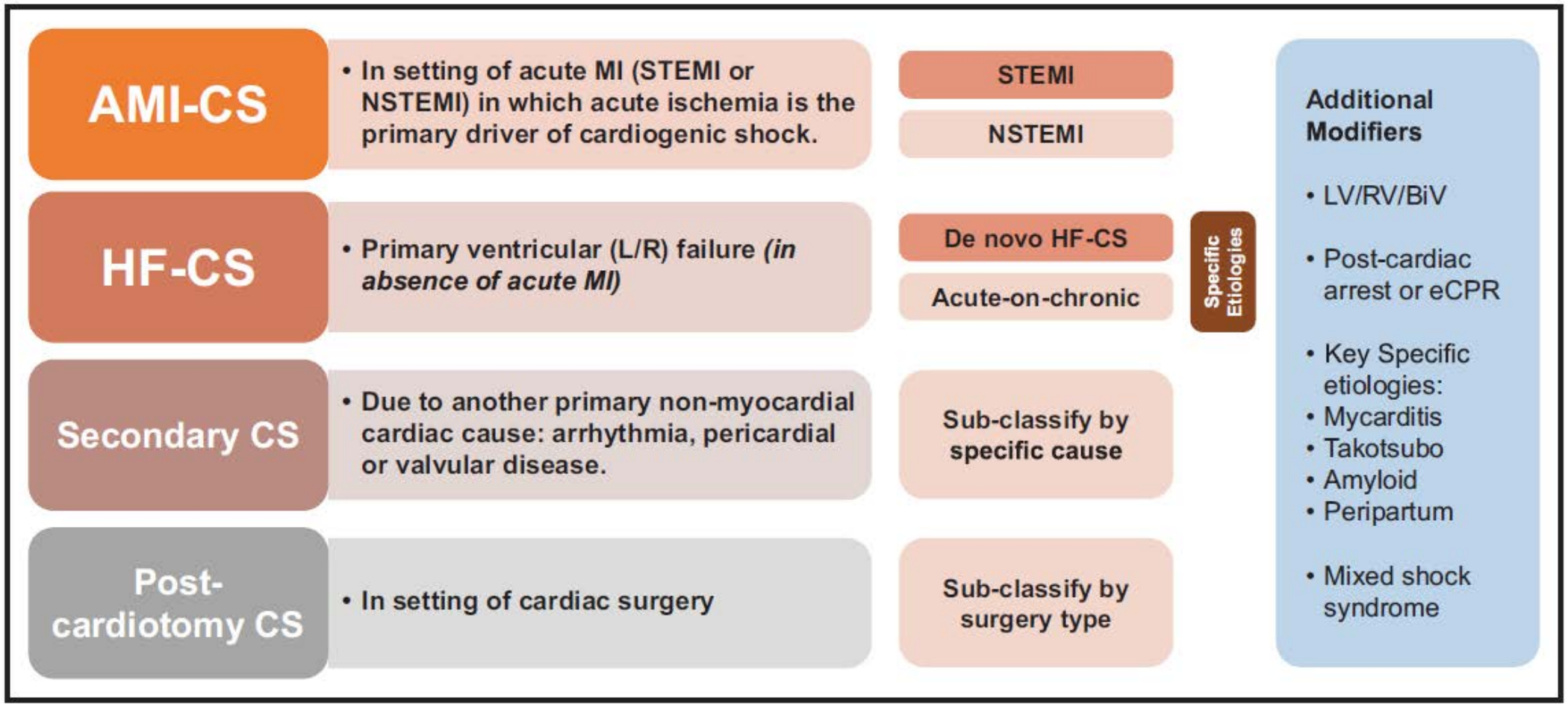
Classification récente:
the SHARC classification (ShockAcademic Research Consortium)

Proposed definition	Cardiac disorder
Definition for clinical practice	Cardiac disorder that results in both clinical and biochemical evidence of sustained tissue hypoperfusion
Definition for clinical trials	Cardiac disorder that results in a systolic blood pressure <90 mm Hg for ≥30 min (or the need for vasopressors, inotropes or mechanical circulatory support to maintain systolic blood pressure ≥90 mm Hg) with evidence of hypoperfusion

Hemodynamic criteria (optional)	
Cardiac index	≤2.2 L/(min·m²)*
Hypoperfusion criteria (≥1)	Elevated arterial lactate (>2 mmol/L)
	Acute kidney injury (creatinine ≥2× upper limit of normal) or oliguria (eg, urine output <0.5 mL/(kg·h)
	Acute hepatic injury (eg, ALT >3× upper limit of normal)
	Cool or mottled extremities
	Altered mental status not explained by an alternative cause
Hemodynamic criteria (optional)	
Cardiac index	≤2.2 L/(min·m²)
Systemic vascular resistance index	>2200 dynes/(cm·sec⁻⁵)*
Normotensive cardiogenic shock subtype cardiac disorder	Systolic blood pressure ≥90 mm Hg without the need for vasopressors, inotropes, or mechanical circulatory support with evidence of hypoperfusion and other potential causes of markers of hypoperfusion have been excluded
Hemodynamic criteria (optional)	
Cardiac index	≤2.2 L/(min·m²)
Systemic vascular resistance index	>2200 dynes/(cm·sec⁻⁵)*

Waksman.R et al, Circulation 2023

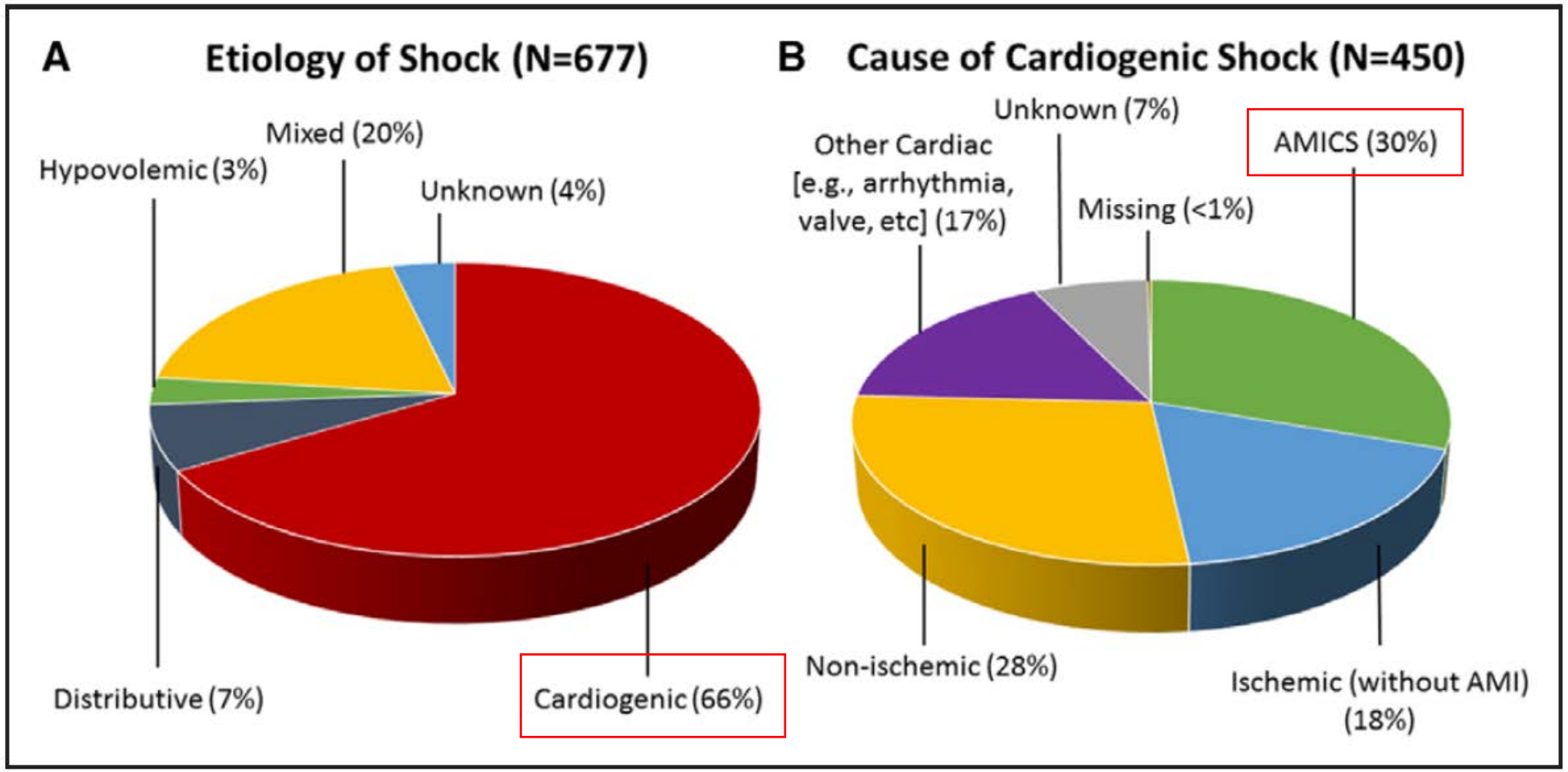
Classification récente:
the SHARC classification (ShockAcademic Research Consortium)



Waksman.R et al, Circulation 2023

Epidemiology of shock in modern ICCU

3049 ICCU admission on 16 centers : 677 shock (22%)



Berg.DD et al, Circ Cardiovasc Qual Outcomes 2019

Evolution des étiologies

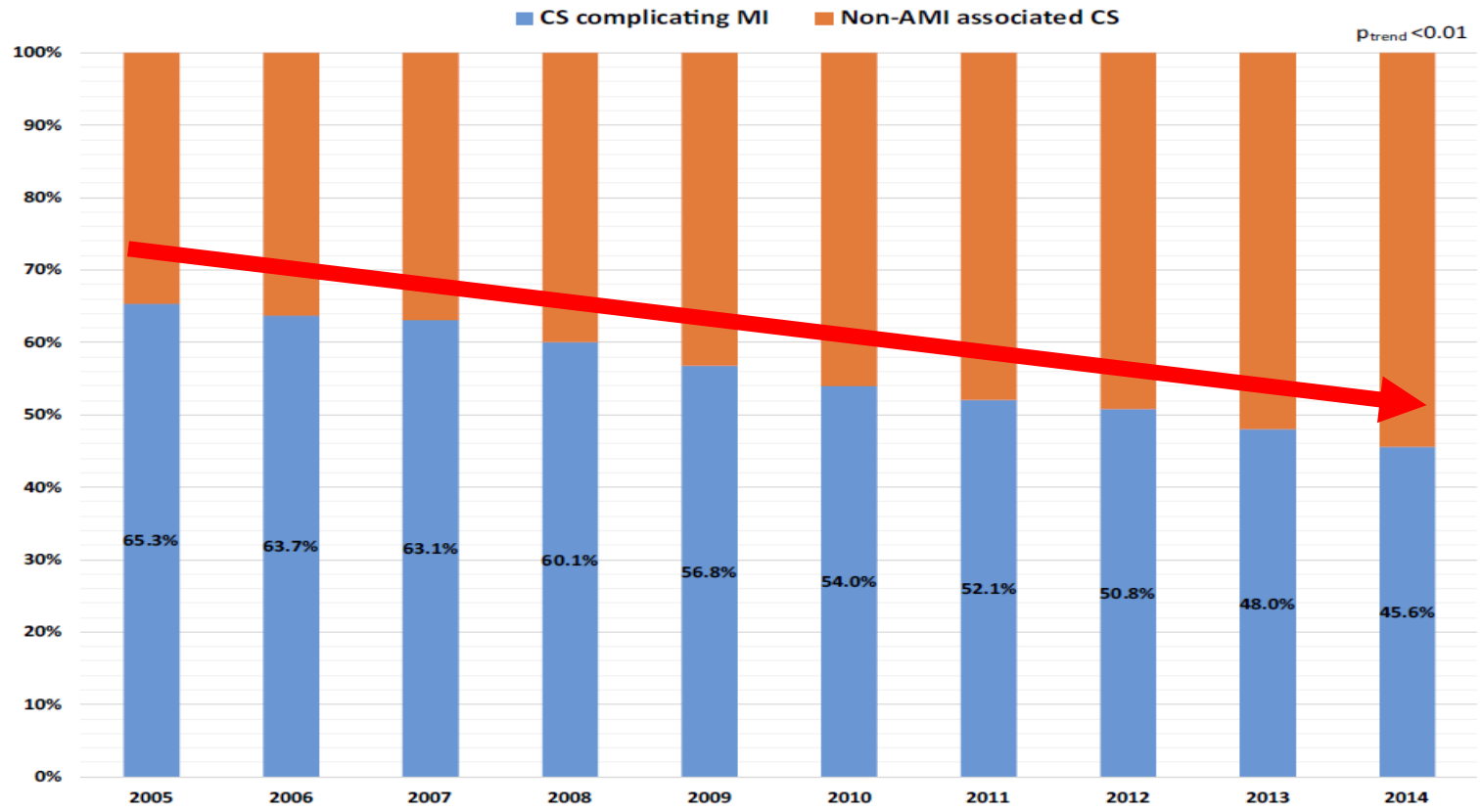


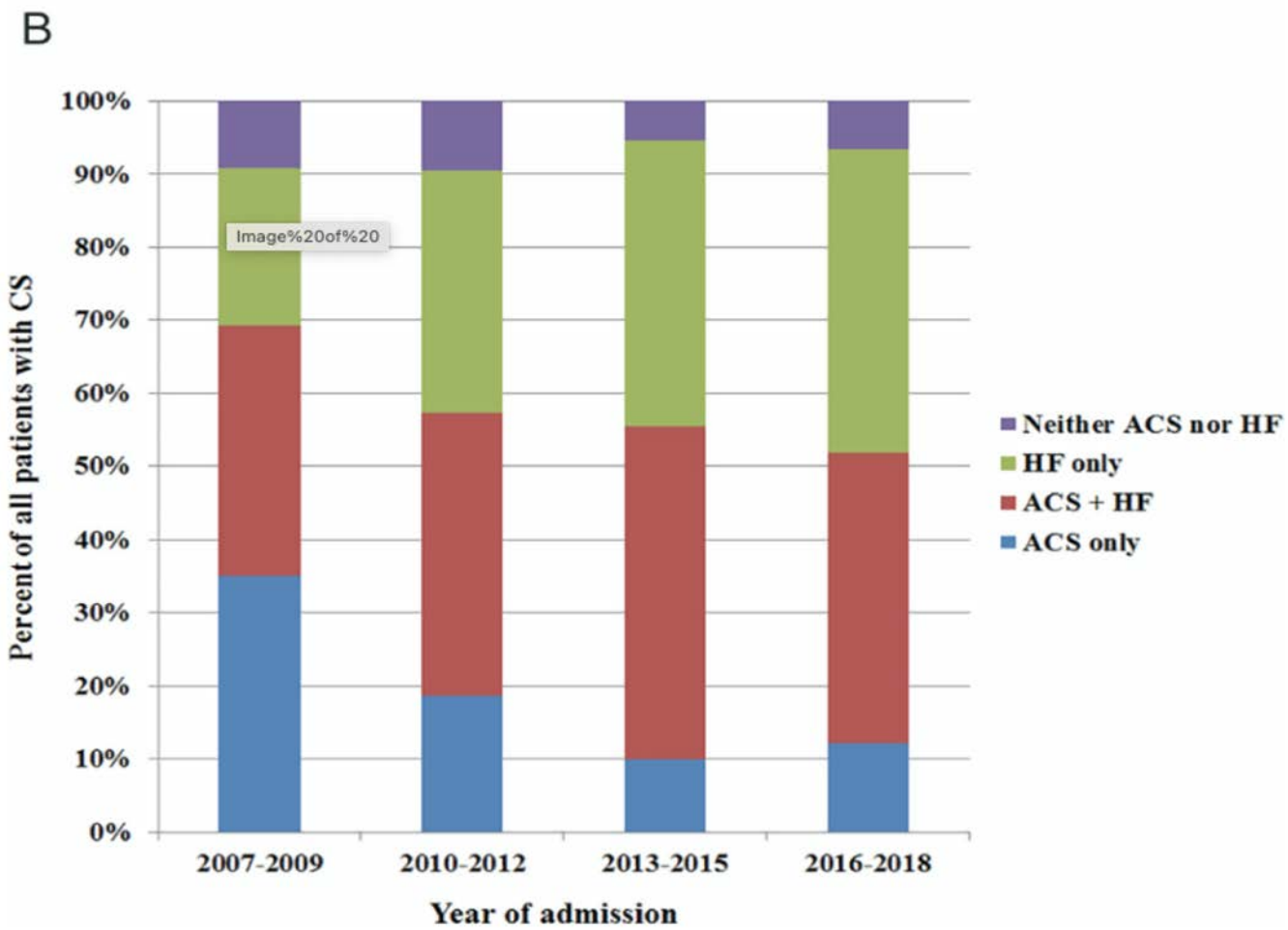
Fig. 4 Proportion of patients admitted with cardiogenic shock depending on status of associated acute myocardial infarction

- **Ischemic CS**:
  - Main etiology +++

- **Others CS**:
  More frequent
  - Dilated cardiopathy. terminal HF. obstructive cardiopathy. myocarditis. intoxication. valvulopathy. PE. takotsubo. sepsis. …

Shah M et al. Clin Res Cardiol 2017

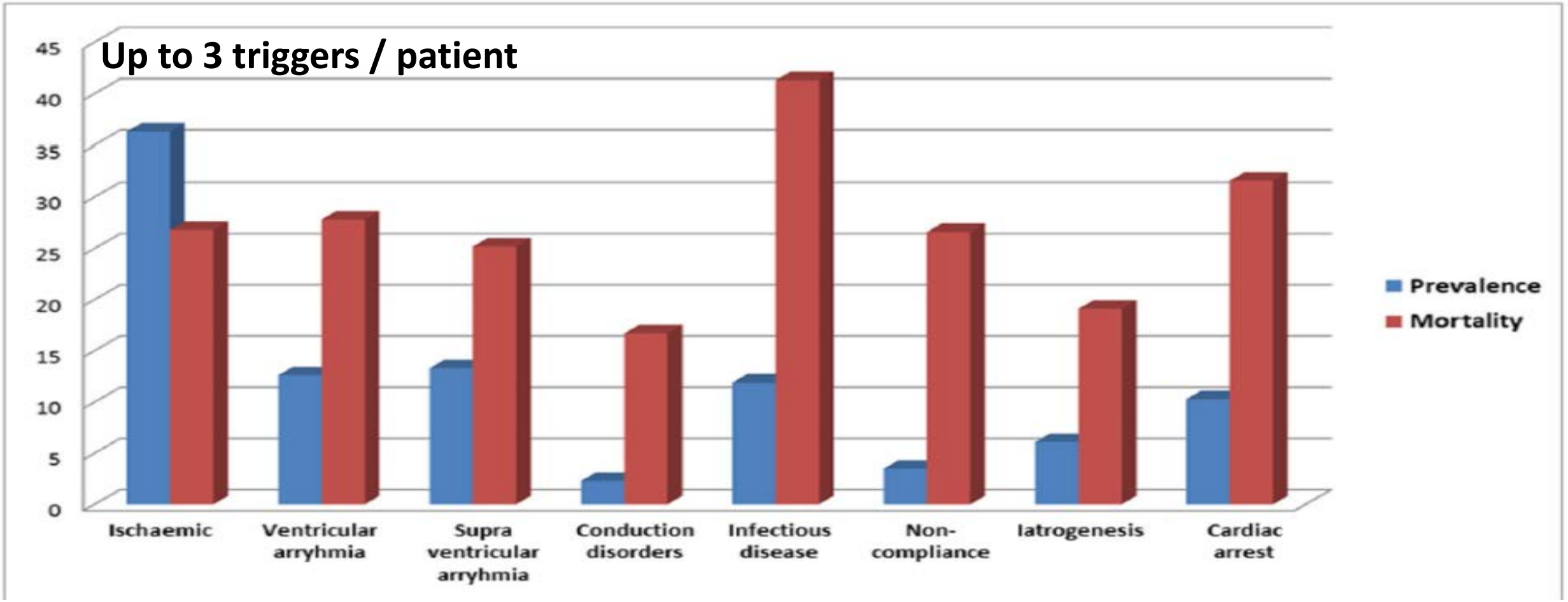
HF-CS has become as prevalent as AMI-CS



HF-CS is overtaking AMI-CS

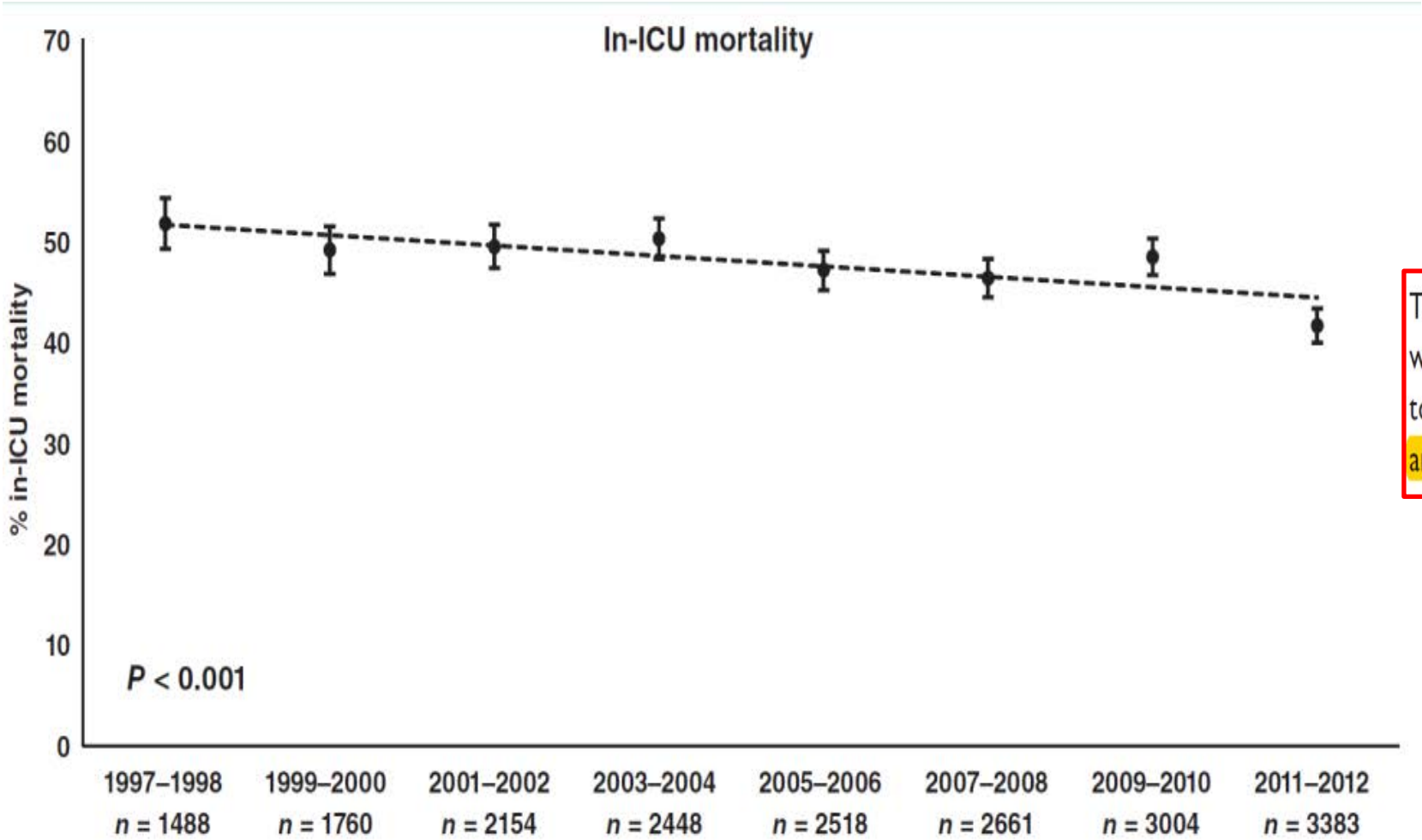
Jentzer, JC. *et al*. *Am Heart J* 2020

Ne pas oublier les facteurs déclenchants



Delmas.C et al, ESC Heart Fail 2022

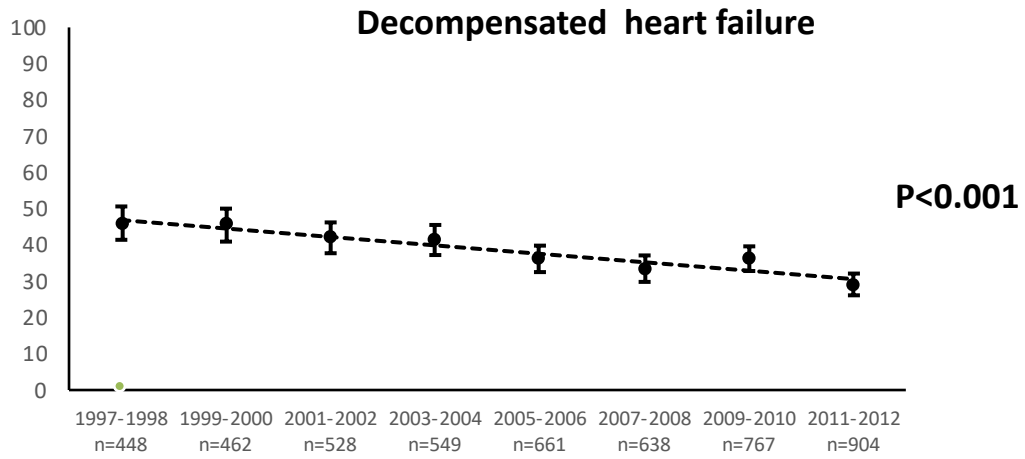
Cardiogenic shock in intensive care units: evolution of prevalence, patient profile, management and outcomes, 1997–2012

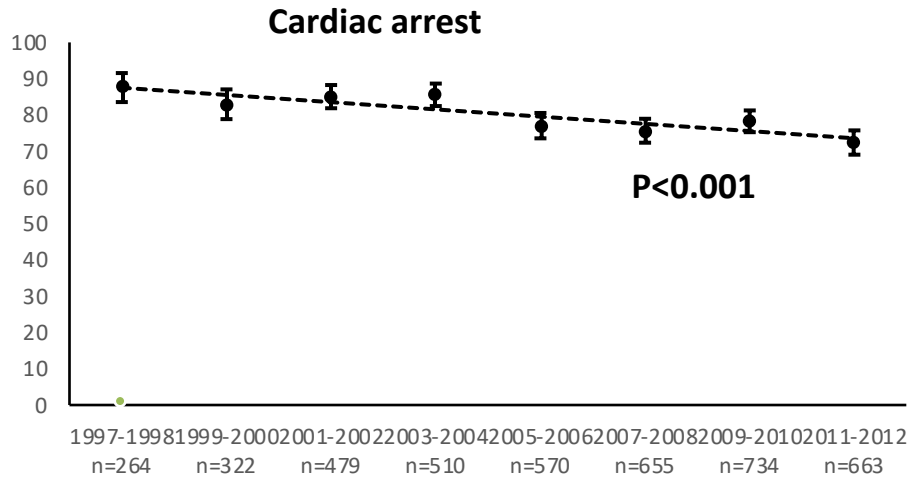


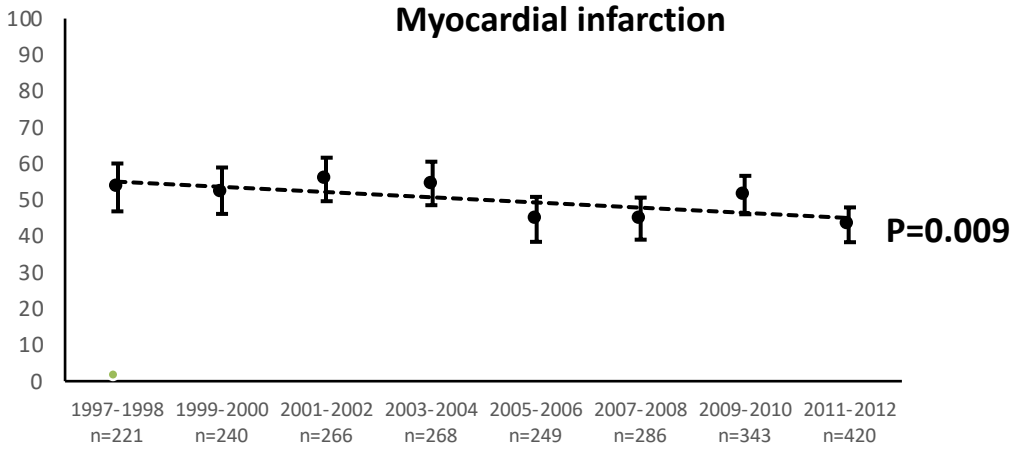
The average crude in-ICU mortality over the entire study period was 47.4% (9205/19 416) and decreased by −5.6% (95% CI −7.7 to −3.5) from 50.3% (period 1) to 44.8% (period 4), representing an 11% relative decrease in mortality (*Table 1* and *Figure 2*).

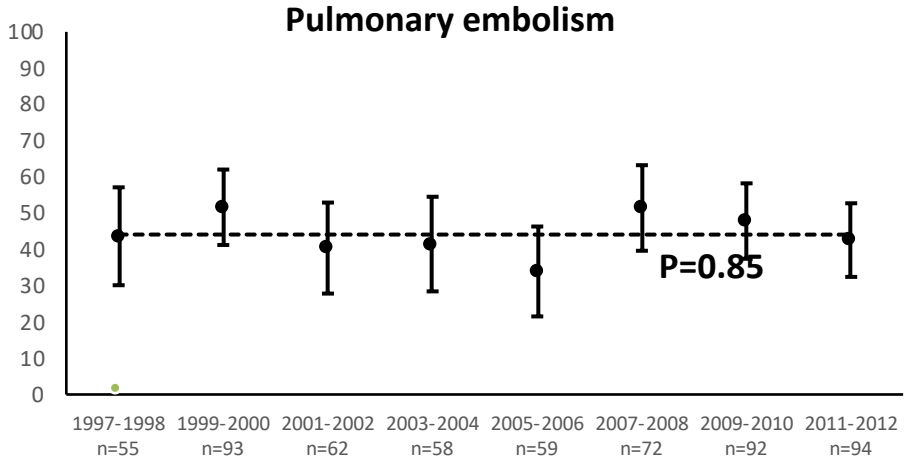
European Journal of Heart Failure (2017) **19**, 192–200

Difference in CS mortality according to its etiology









European Journal of Heart Failure (2017) 19, 192–200

Difference AMICS and ADCHF

Acute = AMICS

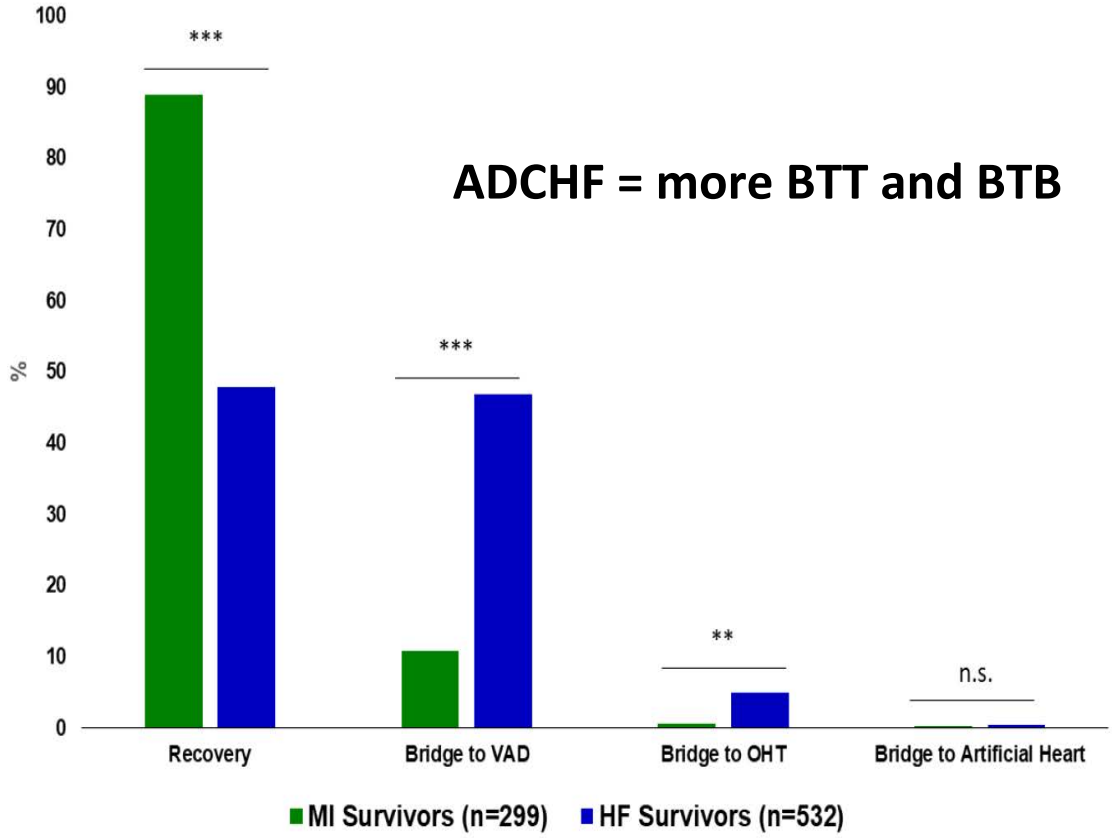
- Older
- **Lower SVr**
- **More severe state: stade D and E**
- **Higher number/doses of inotropes, vasopressors and aMCS (ECMO or Impella)**

Chronic = ADCHF

- Younger
- **Lower LVEF**
- **More severe renal and hepatic impairment**
- **Lower CI and higher mPAP**

In-hospital mortality

AMICS: 39.5% **HF: 25.3%** **P < 0.0001**

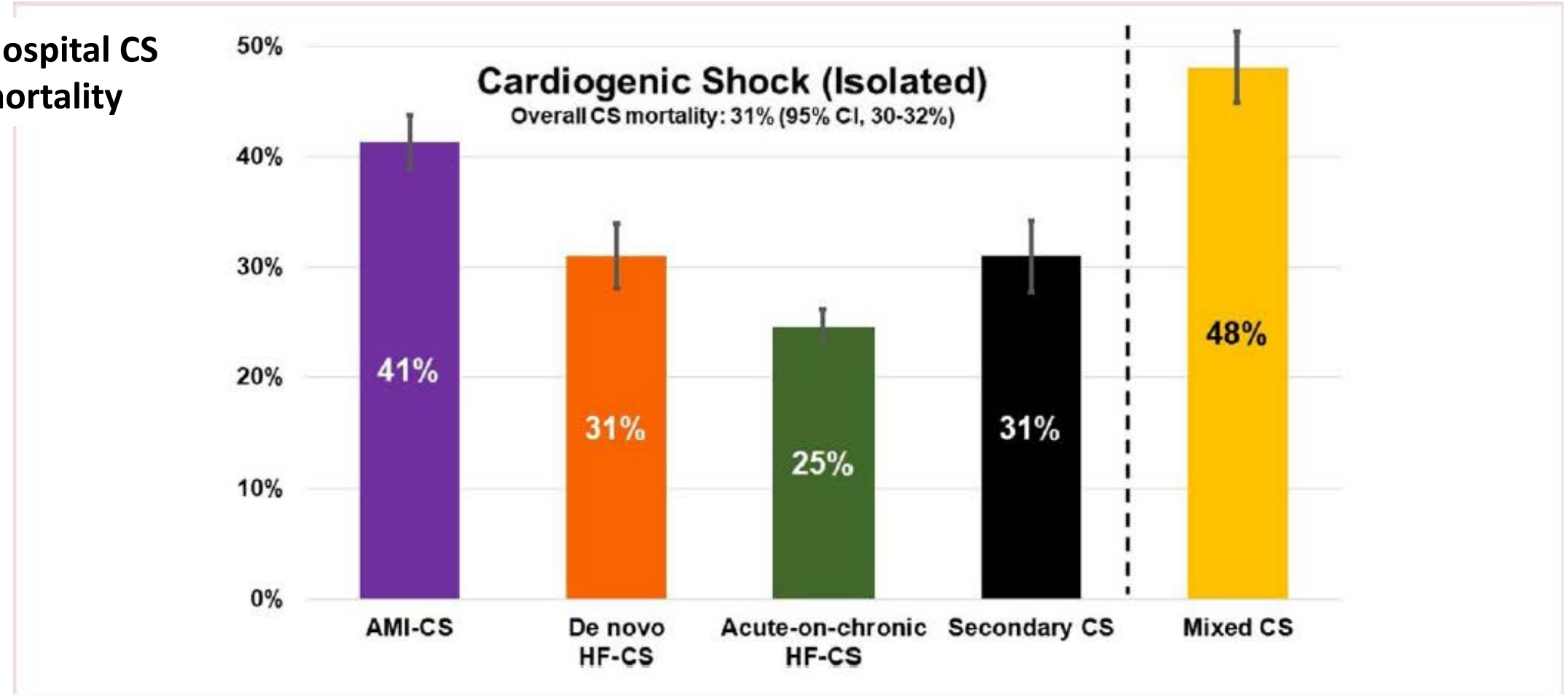


ADCHF = more BTT and BTB

Thayer.KL et al, Circ Heart Fail 2020 / Gaubert.M et al, J Clin Med 2020

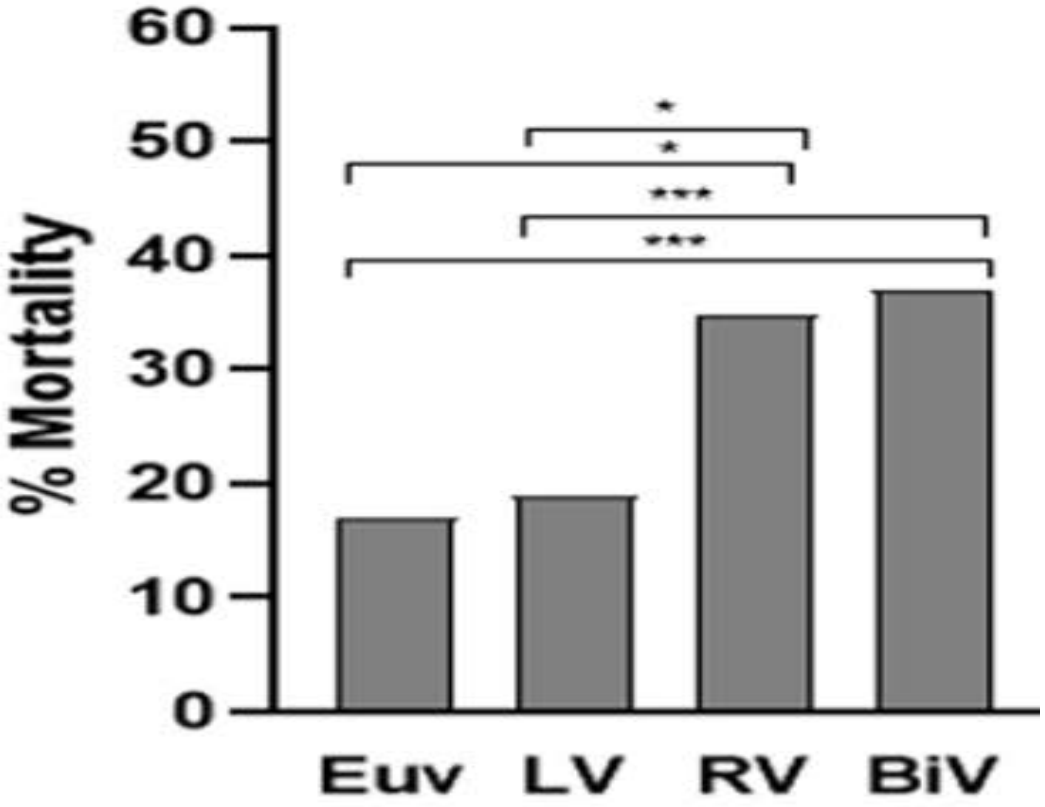
Prognosis according to the SHARC CS classification

In-hospital CS mortality



Berg.DD Eur Heart J ACVC 2024

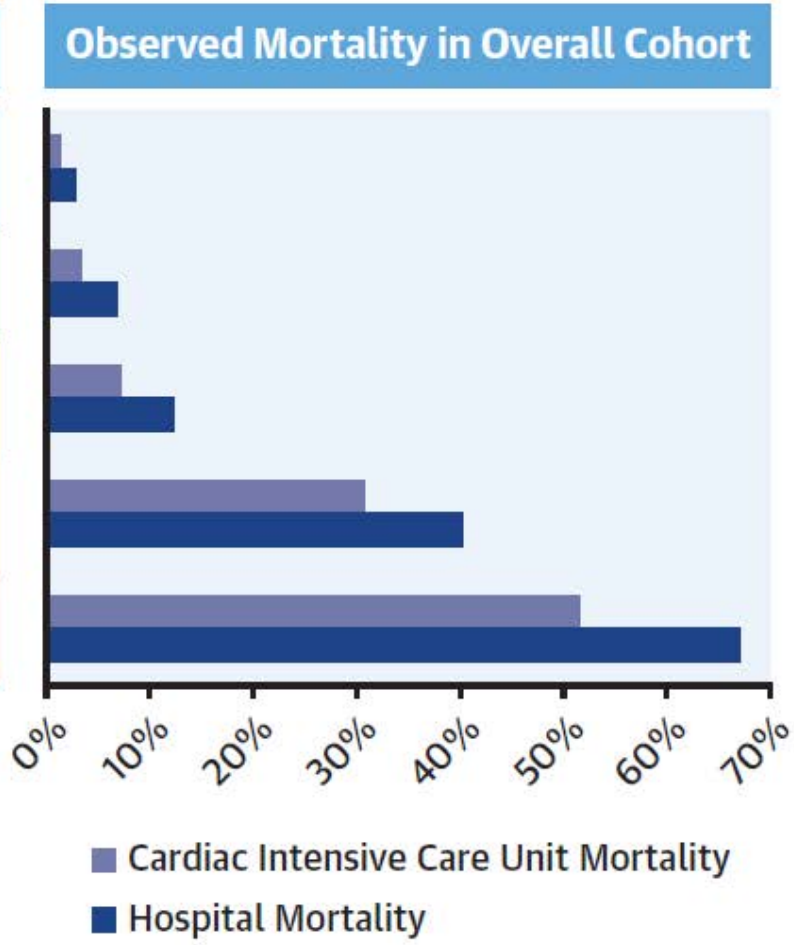
Difference in Left / Right ventricular function and congestion



Thayer.KL *et al, Circ Heart Fail 2020*

Prognostic role in unselected CS at admission

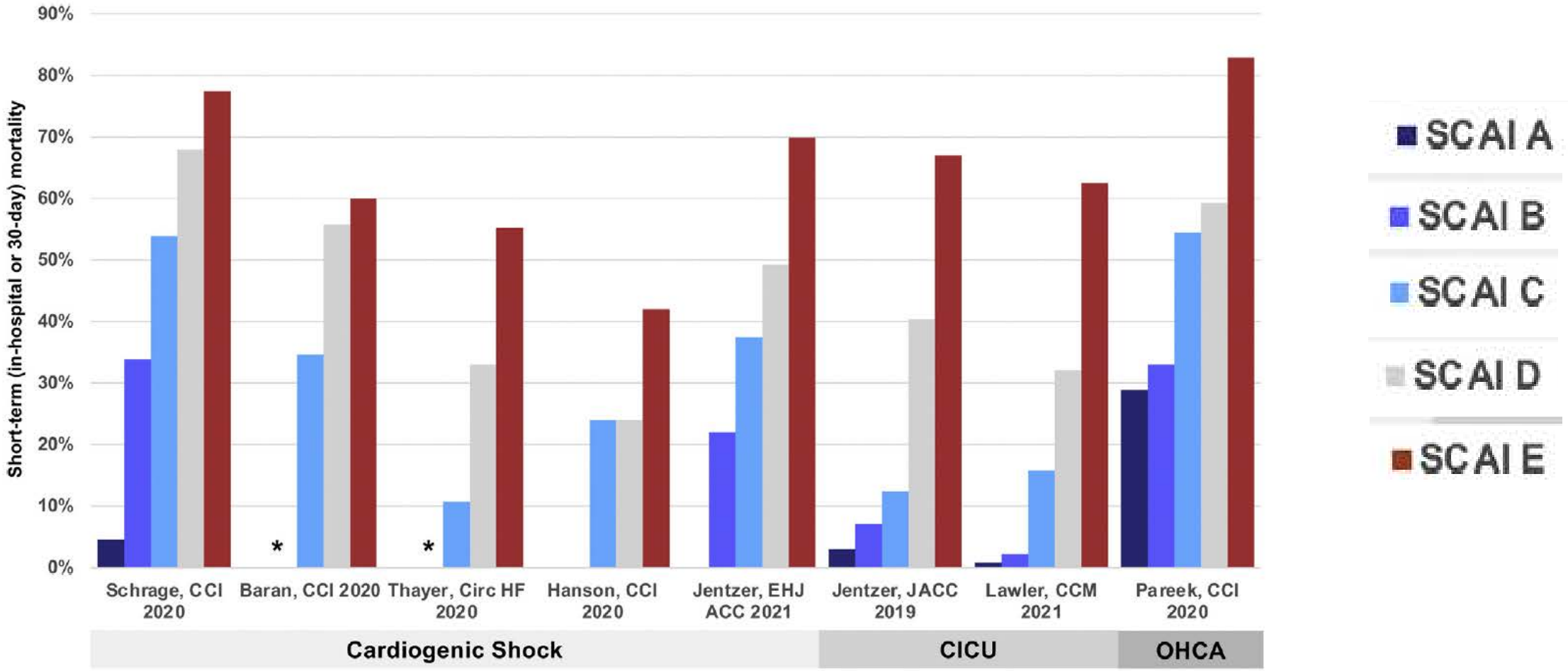
Cardiogenic Shock Stage	Study Definition
Stage A ("At risk")	Neither hypotension/tachycardia nor hypoperfusion
Stage B ("Beginning")	Hypotension/tachycardia WITHOUT hypoperfusion
Stage C ("Classic")	Hypoperfusion WITHOUT deterioration
Stage D ("Deteriorating)"	Hypoperfusion WITH deterioration NOT refractory shock
Stage E ("Extremis")	Hypoperfusion WITH deterioration AND refractory shock



Jentzer, J.C. et al. J Am Coll Cardiol. 2019;■(■):■-■.

10,004 pts, Mayo Clinic, Retrospective, 43,1% ACS 46.1% HF 12.1% CA

Association with Short-term mortality



- **Ischemic and non-ischemic CS**
- **Stronger effects for the most severe CS patients (D/E)**

Naidu.SS et al, J Am Col Cardiol 2022

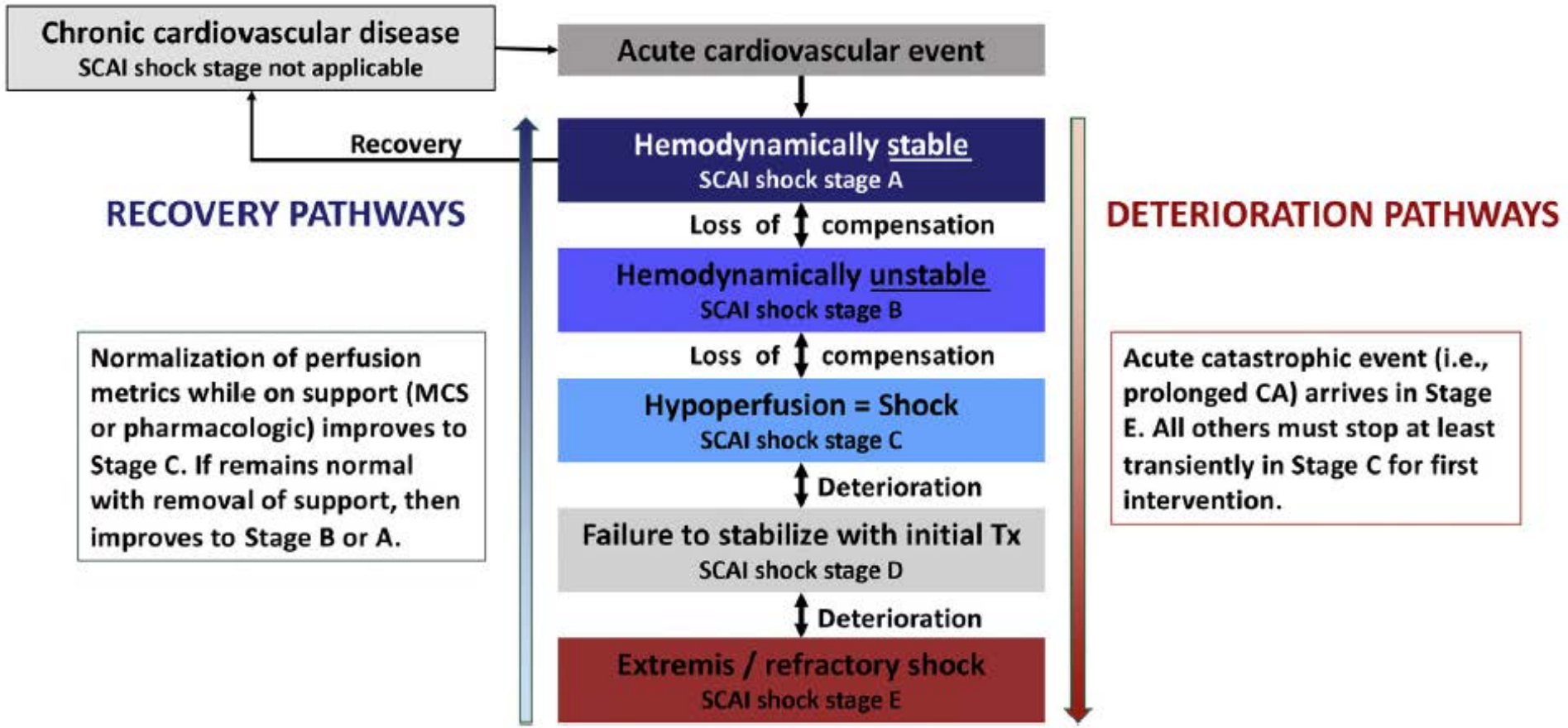
Potential risk modifiers on top of the SCAI shock stages

Study	Population	Design	Patients, n	Variable of Interest	Conclusions
Jentzer et al 2019	CICU				ciated with
Baran et al 2020	CS				ed with higher
Garan et al 2020	CS				nd HR or lower
Hanson et al 2020	AMICS				ed with higher
Jentzer et al 2020	CICU				r mortality
Jentzer et al 2020	CICU				ality
Padkins et al 2020	CICU				r mortality
Thayer et al 2020	CS				r mortality
Jentzer et al 2021	CS				survival
Jentzer et al 2021	CICU				d with higher
Jentzer et al 2021	CICU				mortality across
Jentzer et al 2021	CICU				ner mortality
Zweck et al 2021	CS	Prospective multicenter	1959	Biochemical phenotype	"Cardiometabolic" phenotype associated with higher mortality

- **Prior cardiac arrest**
- **Change in SCAI stage**
- **Invasive hemodynamics: higher RAP and HR, lower MAP**
- **Associated SIRS**
- **Age**
- **Non invasive hemodynamics: low SVI and high E/e'**
- **AKI**
- **Acidosis**
- **Cardiometabolic phenotype**

Moosamy.P et al, J Am Coll Cardiol 2022

Integration of spontaneous or under treatment evolution of patients with CS



But:

- Same stage for patients stabilized by dobutamine 2.5g/kg/min or ECPELLA…..
- Same stage in case of previous CA, mixed shock,…

Need to integrate « modifiers » and pronostic evaluation available at patient's bedside as a « point of care » !

Moosamy.P et al, J Am Col Cardiol 2022

SCAI evolution and associated prognosis in CS

CSWG registry 3268pts (57% HF-CS, 27% AMICS)

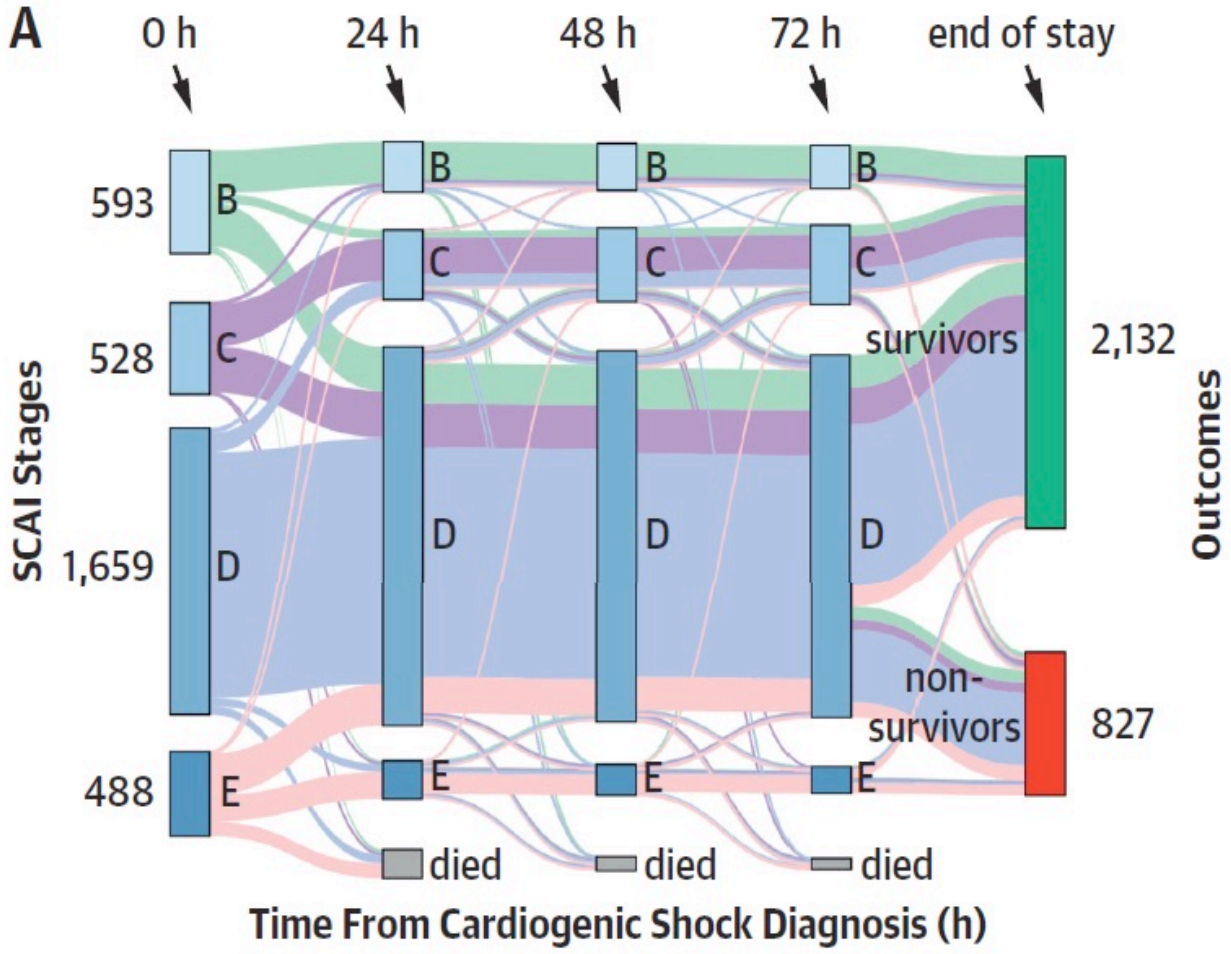


TABLE 2 Unadjusted Mortality for Patients in Each Baseline SCAI Stage Who Were Reclassified at 24 Hours

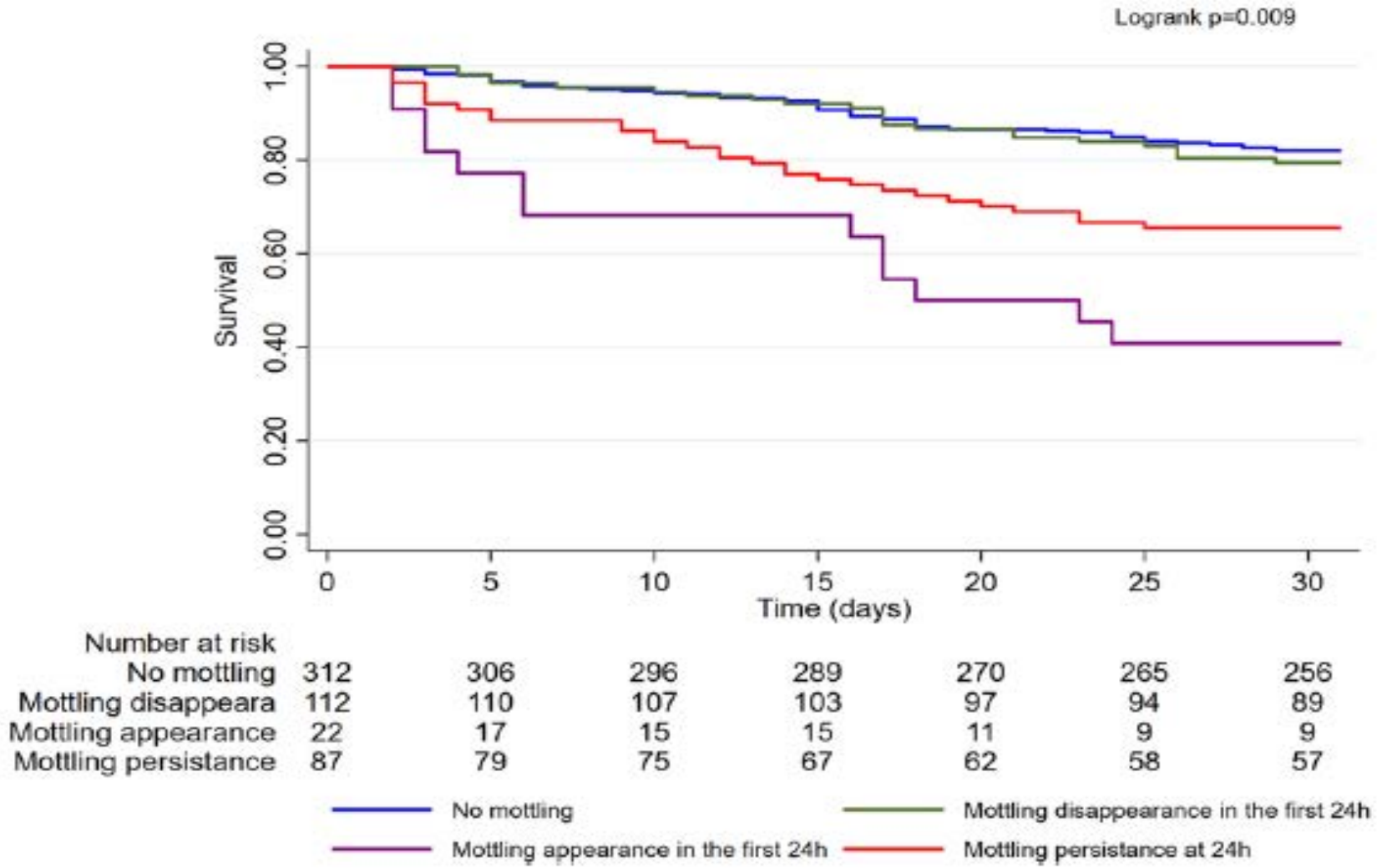
	Mortality				
Baseline	B (24 h)	C (24 h)	D (24 h)	E (24 h)	*P* Value
B	20.3 (246)	25.9[a] (54)	27.4[b] (255)	71.4 (14)	<0.001
C	16.6 (12)	15.8 (241)	25 (264)	80 (5)	0.001
D	16.6 (30)	12.9 (101)	32.4 (1,432)	62.2 (45)	<0.001
E	33.3 (3)	12.5 (8)	47.1 (238)	59.7 (154)	0.005

Values are % (n). [a]*P* = 0.1 comparing stage B worsening to stage C vs stage C remaining at stage C. [b]*P* = 0.6 comparing stage B worsening to stage D vs stage C worsening to stage D.

SCAI = Society for Cardiovascular Angiography and Interventions.

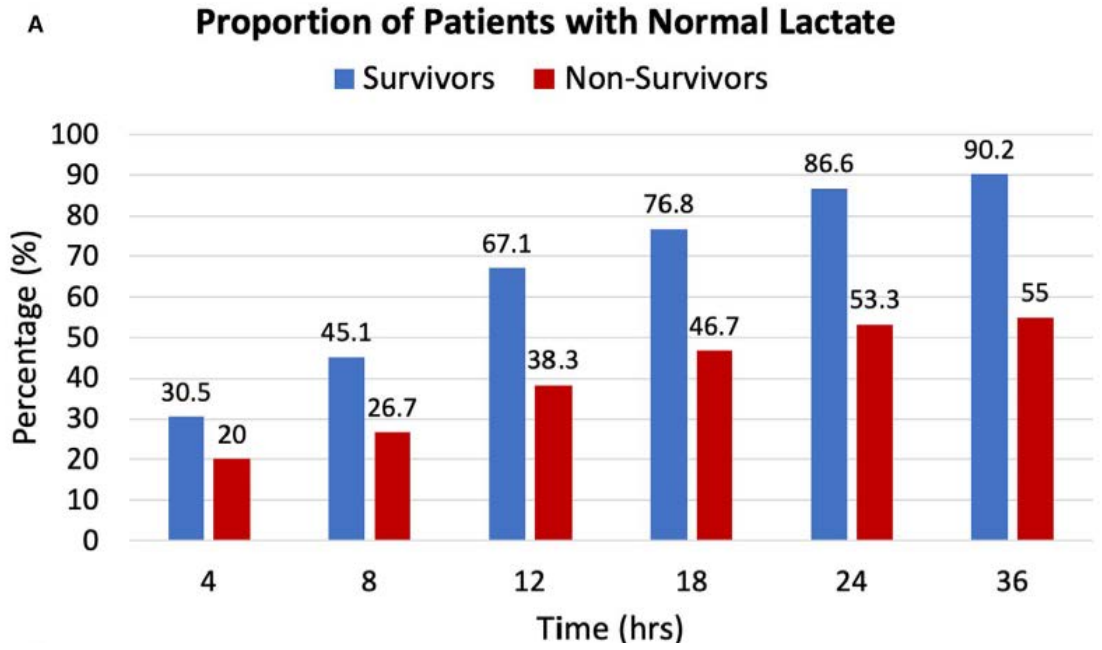
Ton VH et al, JACC 2024

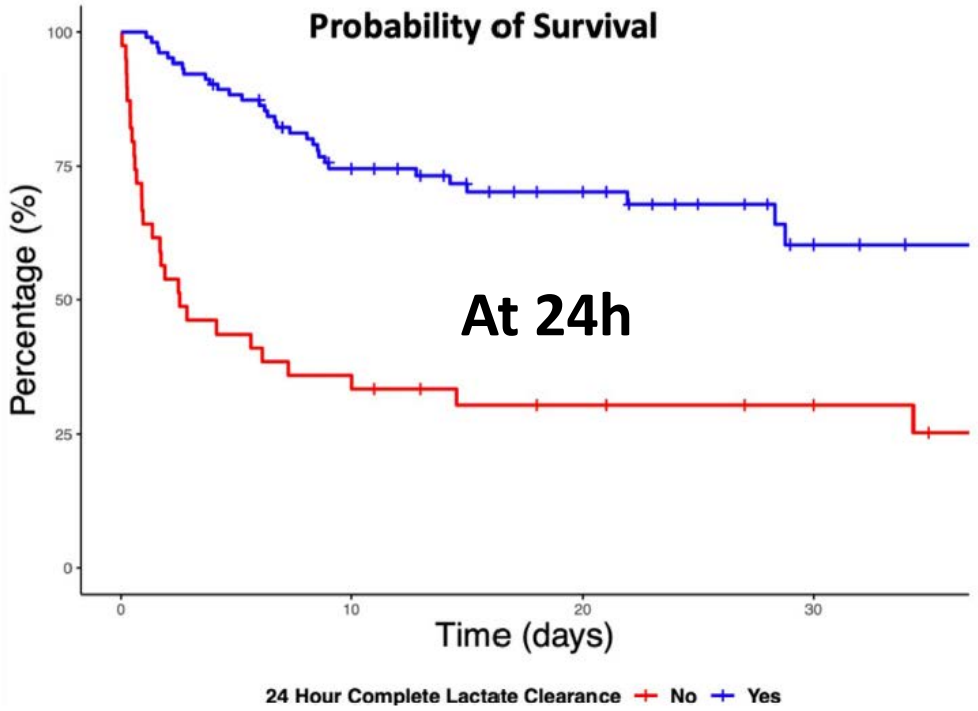
Clinical monitoring +++ = Mottling !

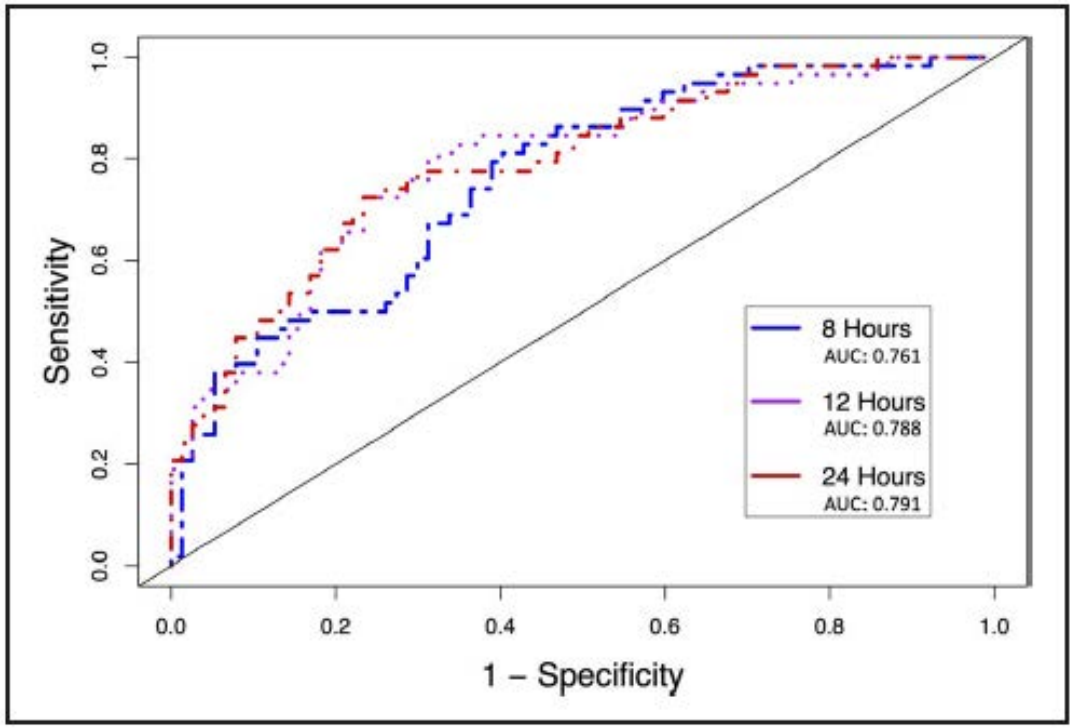


Merdji.H et al, Ann Int Care 2023

Prognostic role of lactate clearance in CS



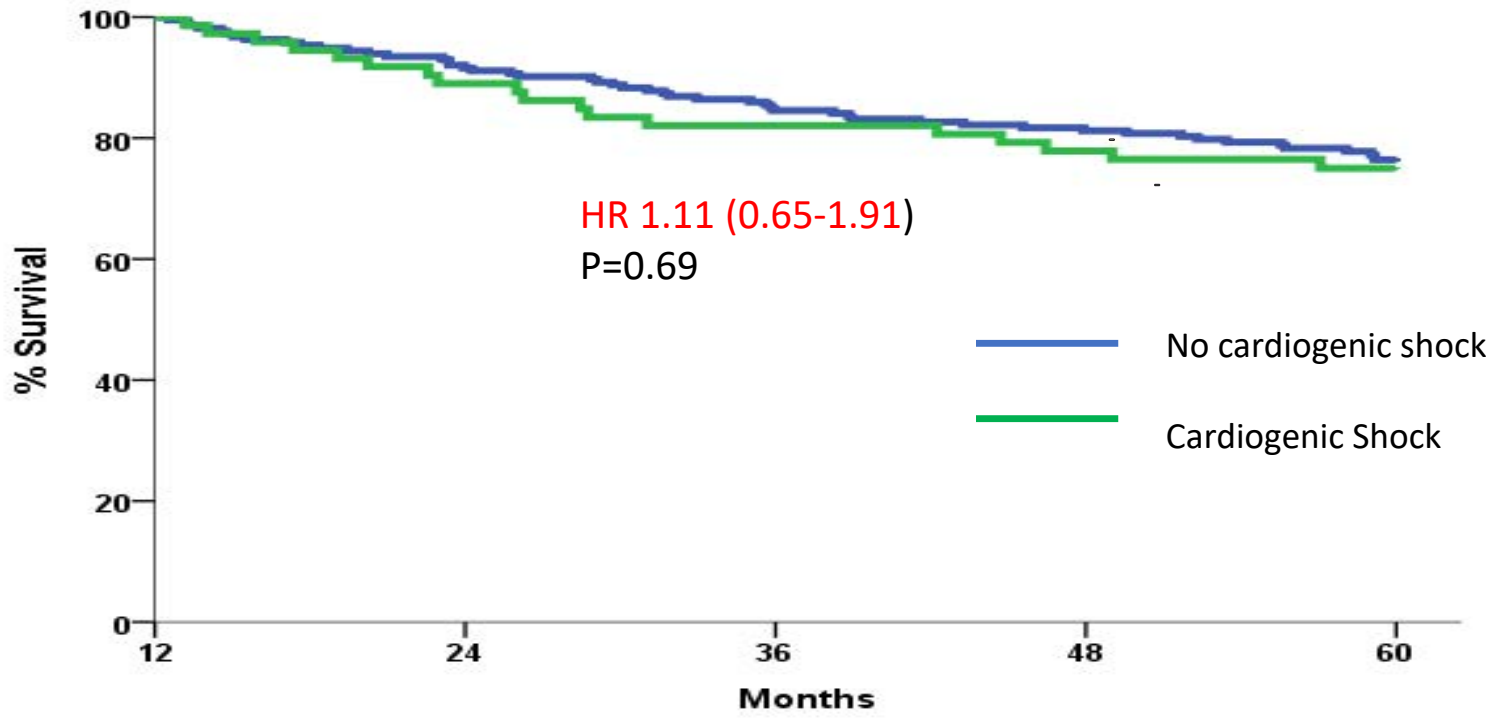




Marbach.JA et al, J Am Heart Assoc 2022

Long-term outcome in early survivors of cardiogenic shock at the acute stage of myocardial infarction: a landmark analysis from the French registry of Acute ST-elevation and non-ST-elevation Myocardial Infarction (FAST-MI) Registry

Nadia Aissaoui[1,2], Etienne Puymirat[1,2,3], Tabassome Simon[4,5,6], Eric Bonnefoy-Cudraz[7], Denis Angoulvant[8], Francois Schiele[9], Hakim Benamer[10], Philippe Quandalle[11], Fabrice Prunier[12], Eric Durand[13], Laurence Berard[4], Didier Blanchard[14] and Nicolas Danchin[1,2*]

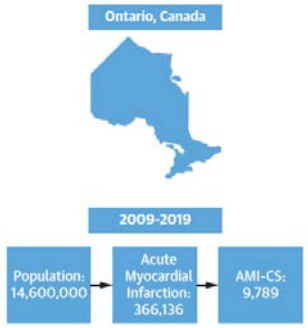


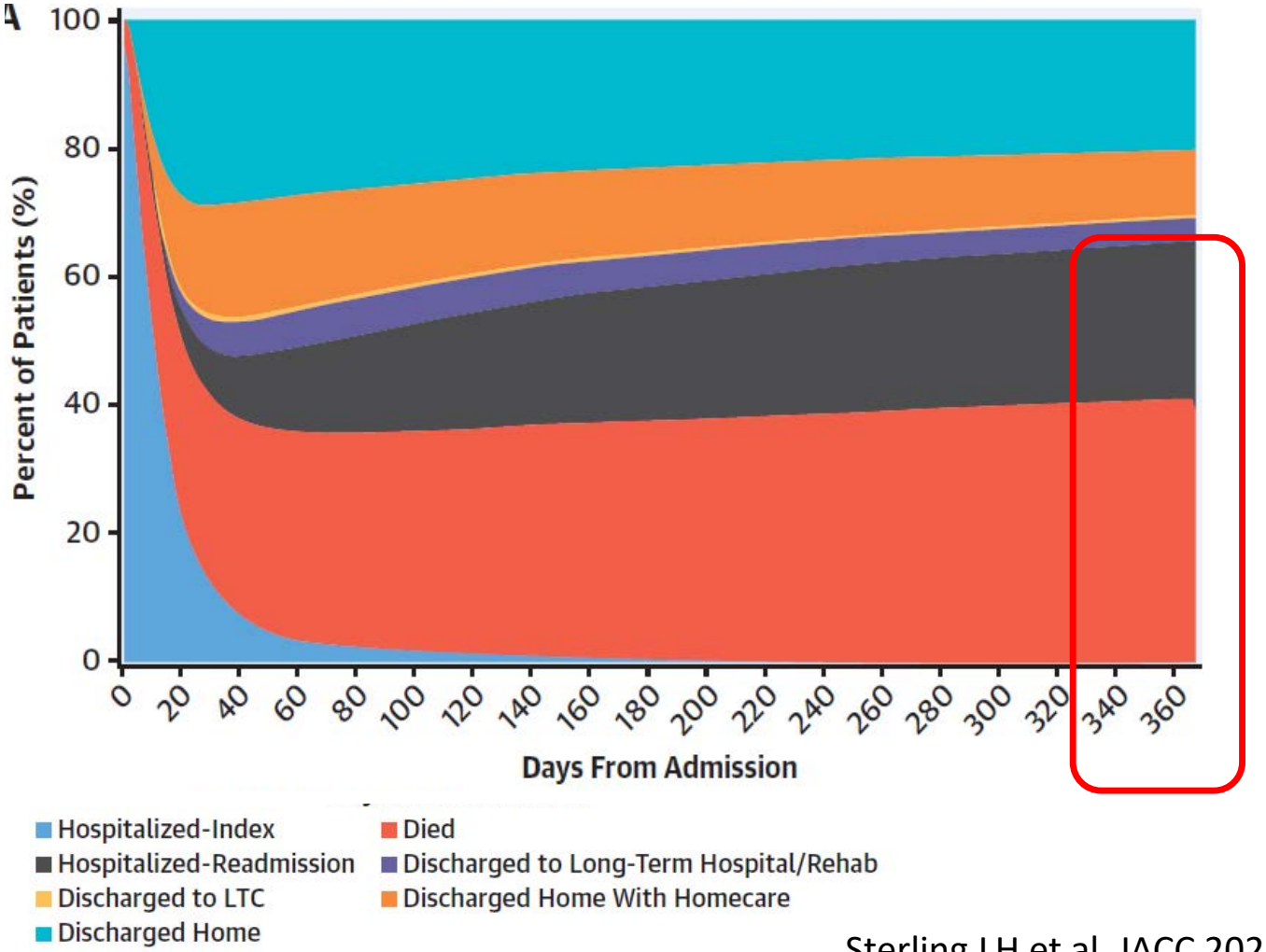
FAST MI 2005 : 3670 pts
3411 survivants à J30
99 pts en choc

Chez les patients en choc cardiogénique qui survivent à la phase aigue, la mortalité après un an est la meme quelque soit le tableau à la phase initiale

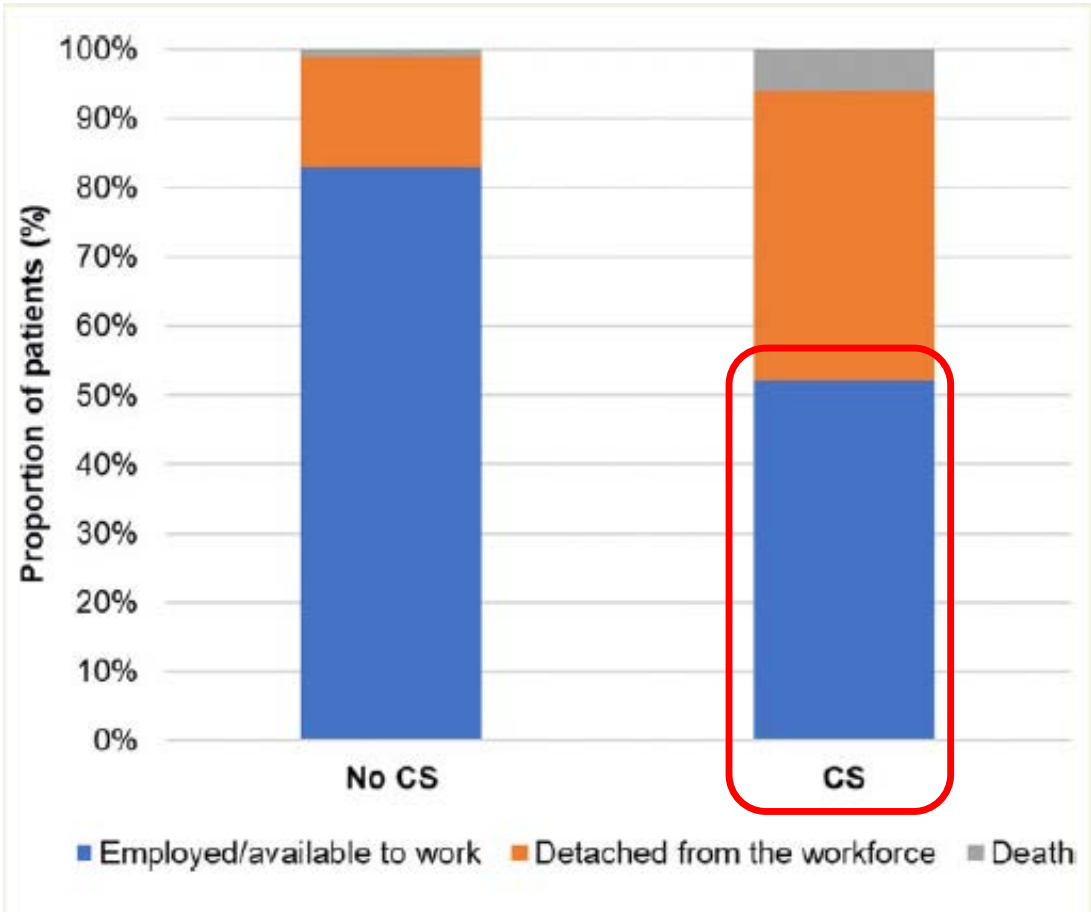
Critical Care 2014, **18**:516

Mild-term prognosis in AMICS





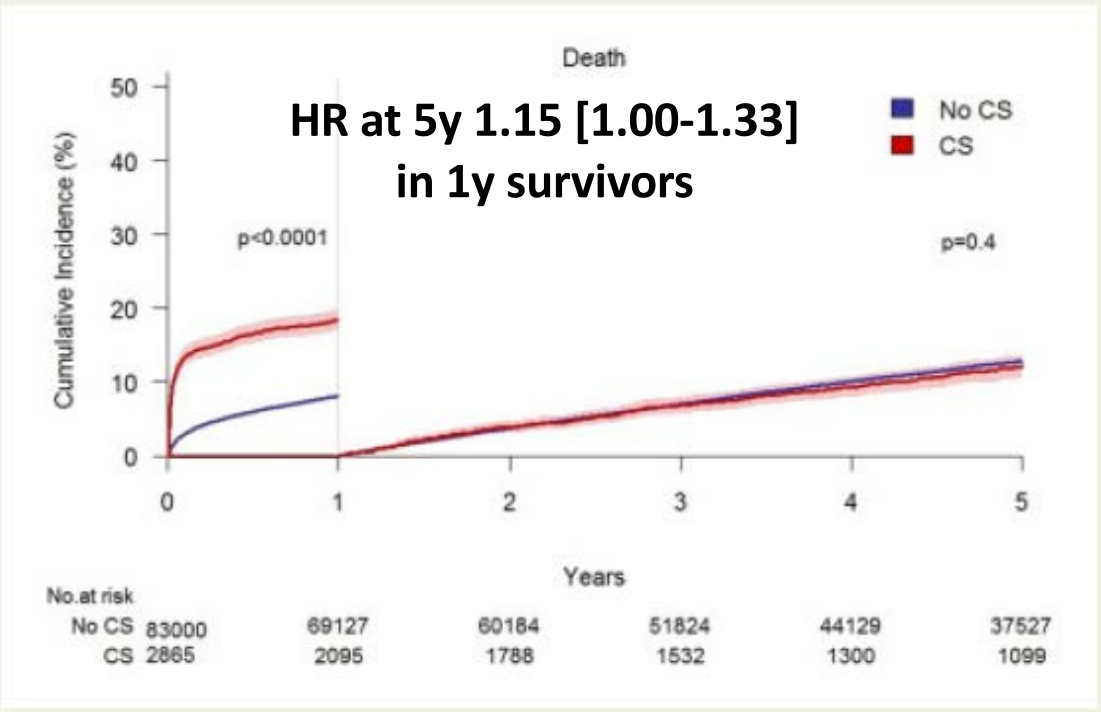
Sterling.LH et al, JACC 2023

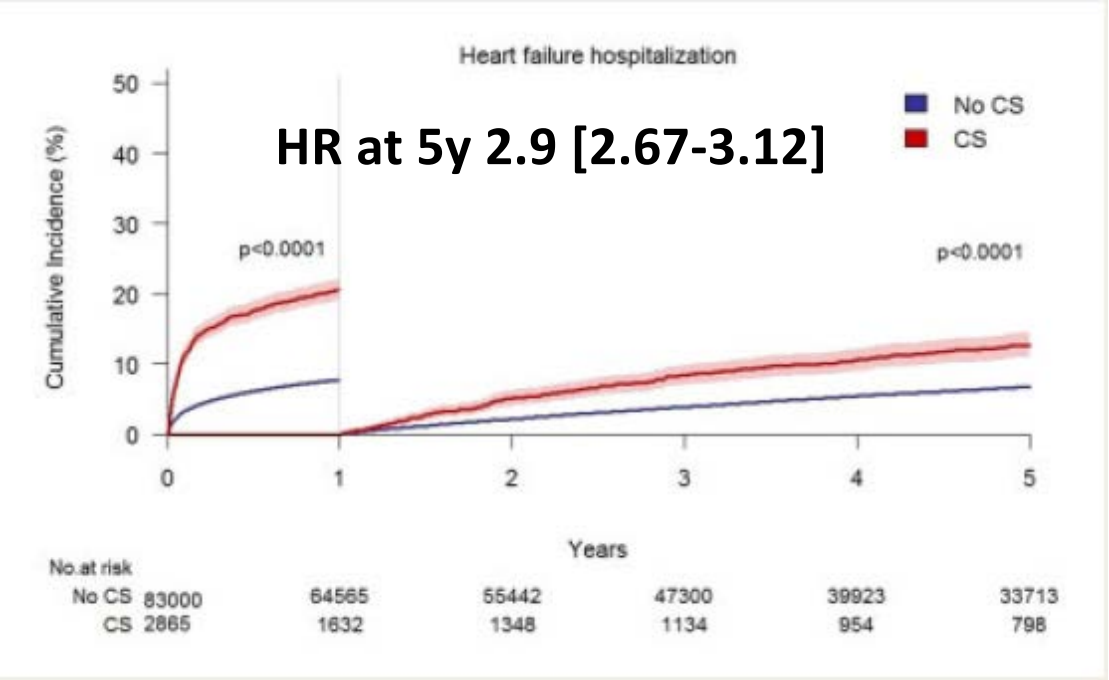


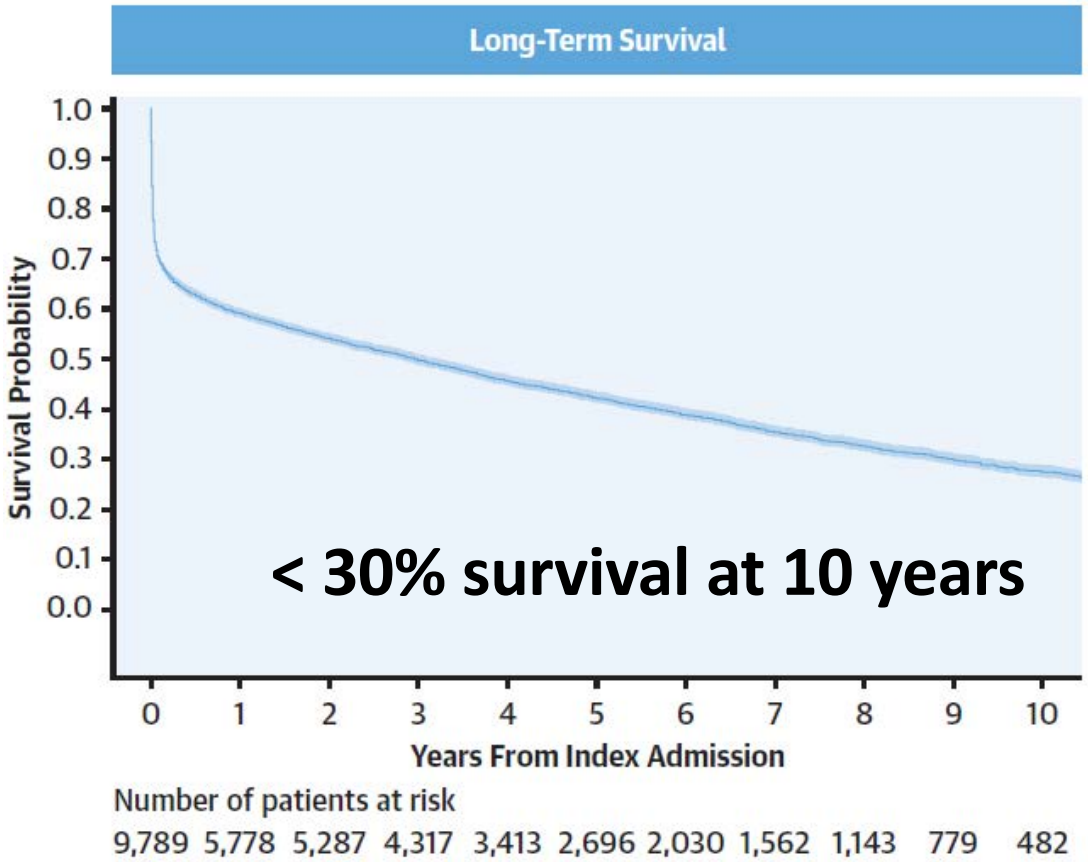
19799 Danish AMI pts (2005-2015) = 653 AMICS (3%)

Lauridsen.MD et al, EHJACVC2022

Long-term prognosis in AMICS

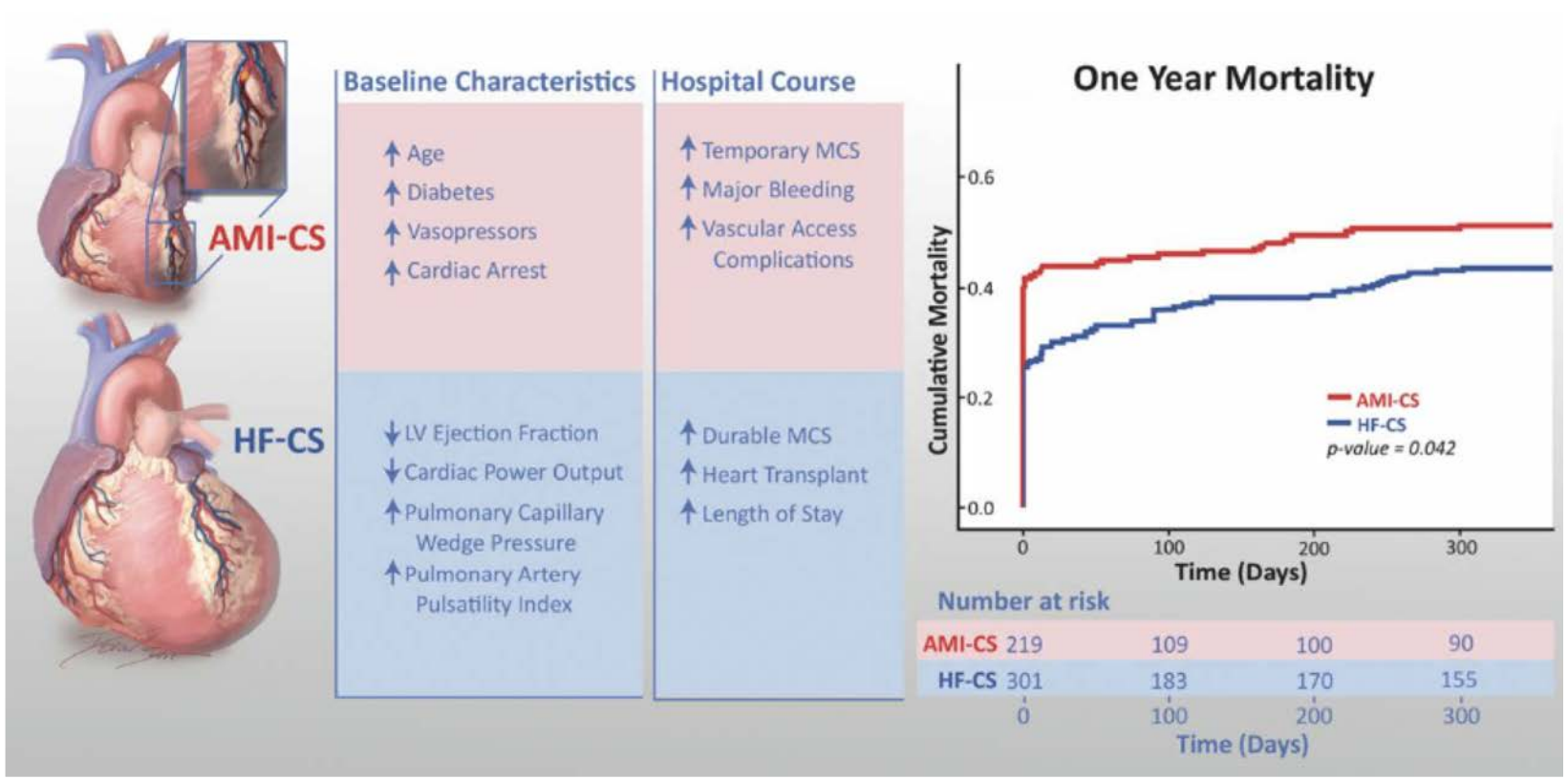




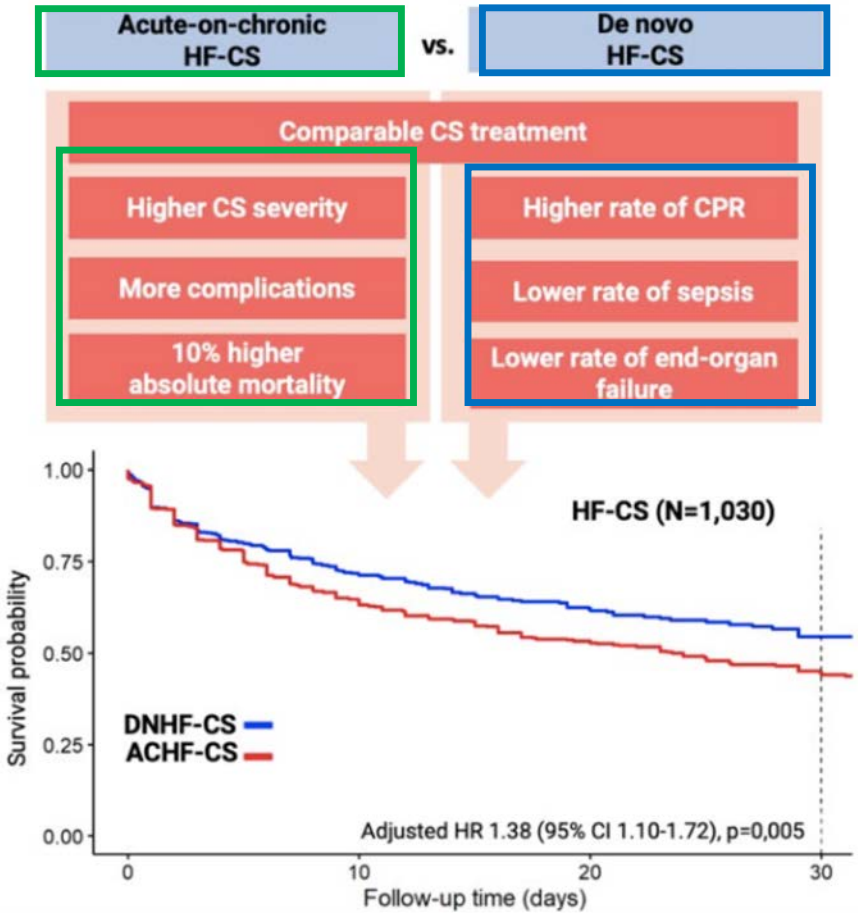


Sterling.LH et al, JACC 2023

AMI-CS and HF-CS have different outcomes



Lower in-hospital and 1-year mortality in HF-CS patients

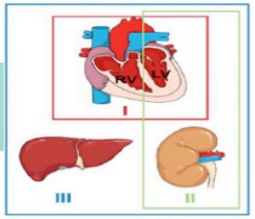


Worse outcomes in acute-on-chronic HF-CS

Sinha, SS. *et al. Circ Heart Fail* 2023; Sundermeyer, J. *et al. Eur J Heart Fail* 2023

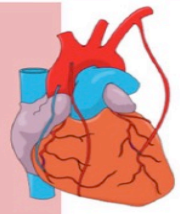
Prise en charge diagnostique ; intégration des nouvelles classifications

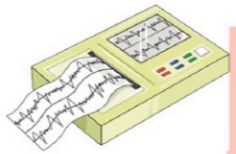
Definition
SBP<90mmHg
End-organ hypoperfusion: cold extremities, confusion, oliguria<30mL/h
Lactate>2mmol/L
CI<2.2L/min/m2



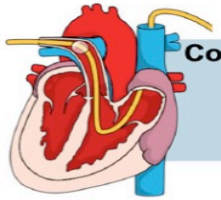
Phenotyping
BiV / LV-predominant / RV-predominant CS
Non congested / CardioRenal /Cardiometabolic

Identify cause of CS
Acute myocardial infarction
Cardiac arrest
Ventricular / Supraventricular arythmia
Conduction disorders
Infectious disease
Iatrogenesis
Non-compliance





Key investigations for etiological assessment
12-lead ECG, X-Ray, Laboratory tests
Transthoracic +/- transoesophageal echocardiography
Lung and pleural ultrasound
Tomography scan for Aortic syndrome or Pulmonary embolism



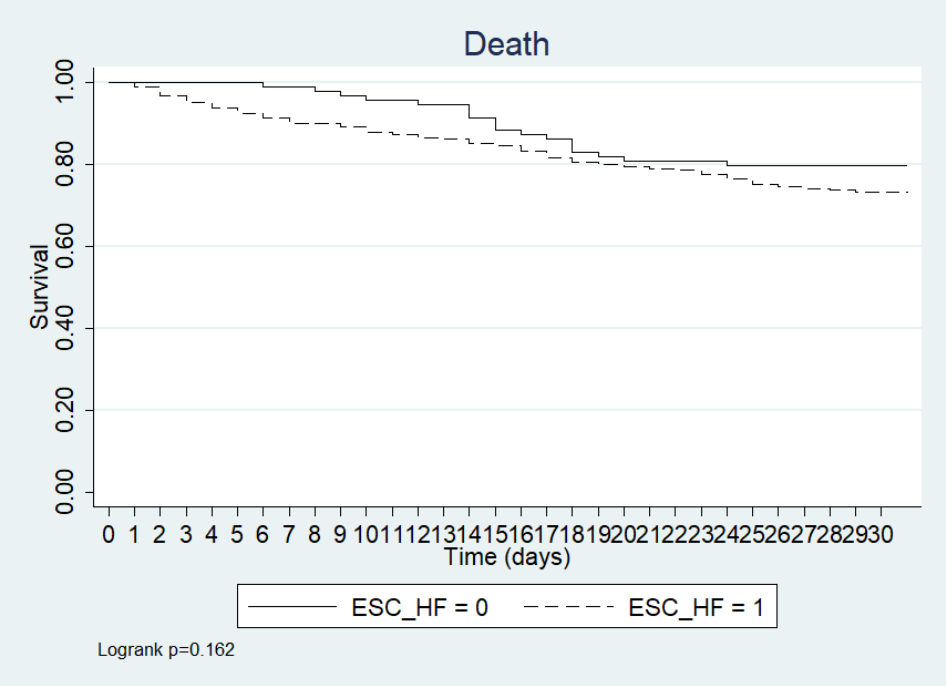
Considering invasive hemodynamic monitoring
Arterial catheter
Transpulmonary thermodilution
Discussed pulmonary artery catheterization

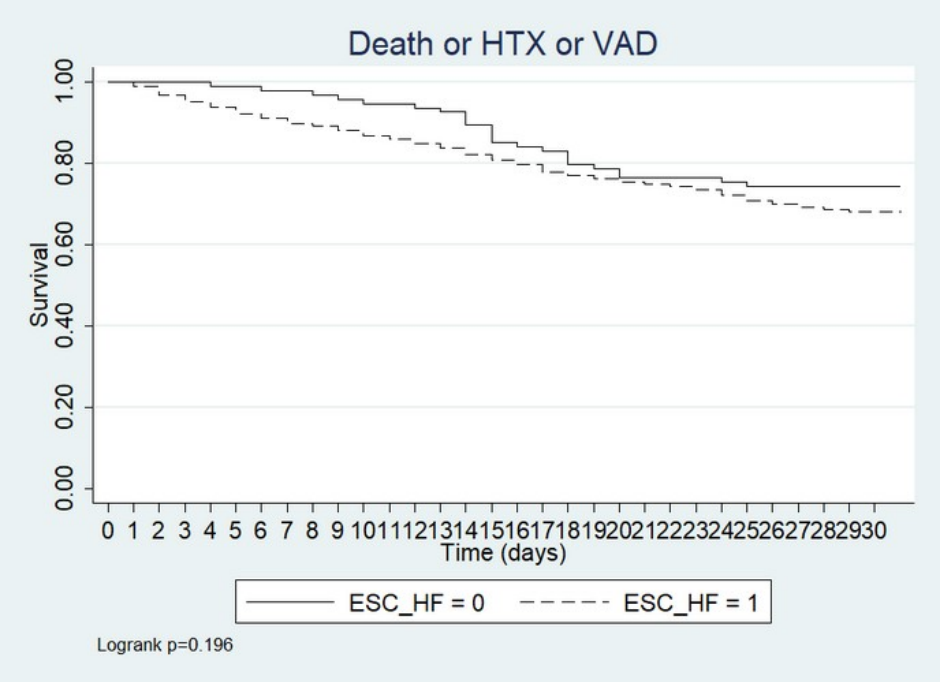
Diagnostic du choc cardiogénique retour à la définiton…

- Une pression artérielle systolique inférieure à 90 mmHg ou une pression artérielle moyenne inférieure à 65 mmHg pendant 30 minutes ;
- Une congestion pulmonaire ou une élévation des pressions de remplissage ;
- Des signes d'hypoperfusion périphérique : (a) confusion; (b) genoux froids et/ou marbrés; (c) oligurie; (d) lactate augmenté

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

Oui mais non : pas de différence de pronostic , pas pas meconnaitre des chiffres de PAS « correctes »



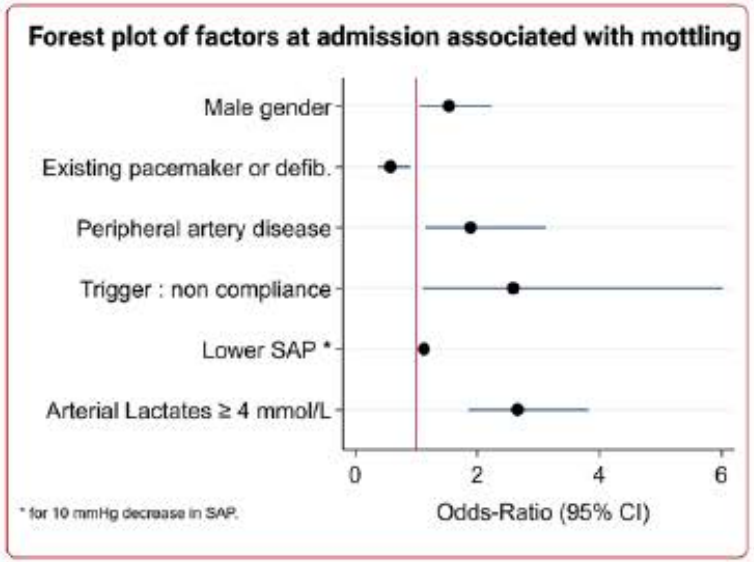


FRENSHOCK
772 patients in 49 centers
France 6 months 2016

Patients with (ESC-HF = 1), or without (ESC-HF=0) hypotension
All with low cardiac output, overload signs and organ malperfusion

Delmas.C et al, JESFC 2019

Tissu malperfusion = Mottling +++



At admission

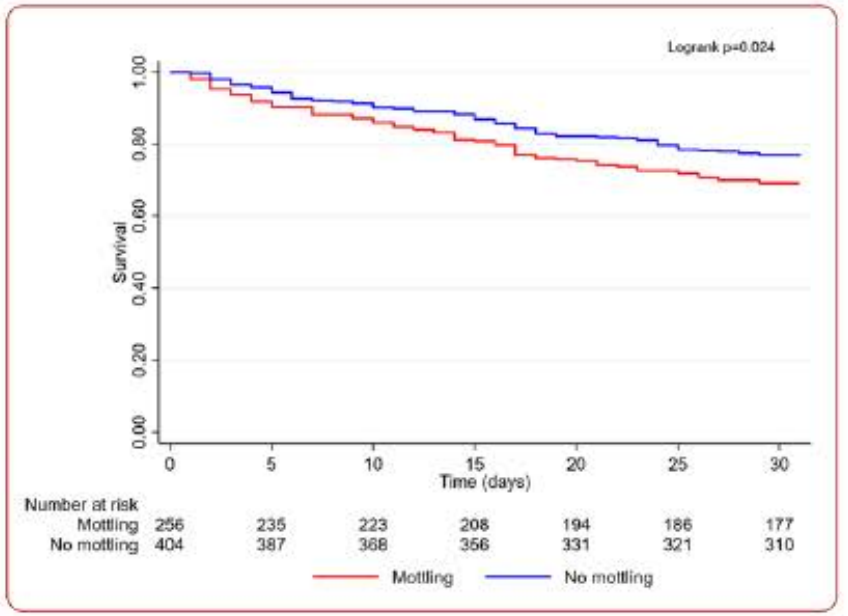
- No major difference regarding past medical history
- Higher HR in patients with mottling (p=0.029)
- Lower SAP & DAP in patients with mottling (p<0.001)
- Higher arterial lactate level (3.8 vs 2.5 mmol/L, p<0.01)

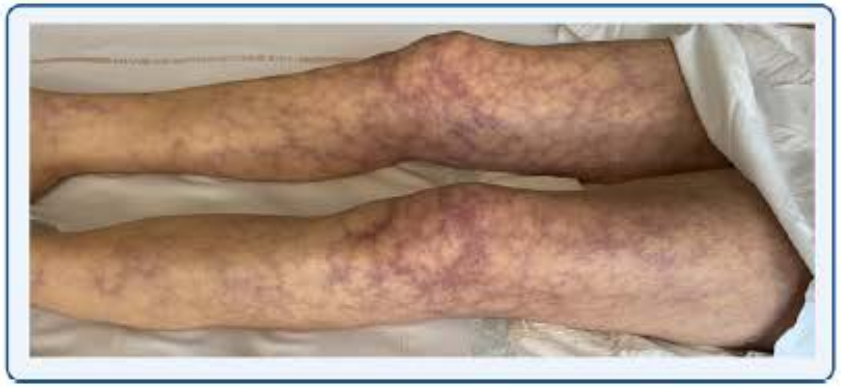
In-hospital management

- Invasive respiratory support significantly higher in patients with mottling (50.2% vs. 30.1%, p<0.001)
- Higher need for renal replacement therapy also (19.9% vs. 12.4%, p=0.09).

Consequences

- Prolonged length of stay (19 vs. 16 days, p=0.033)
- Higher 30-day mortality (31% vs. 23.3%, p=0.031)
- Higher one-year mortality (54% vs. 42%, p=0.003)





Mottling is a simple, easy, costless prognosis marker in cardiogenic shock

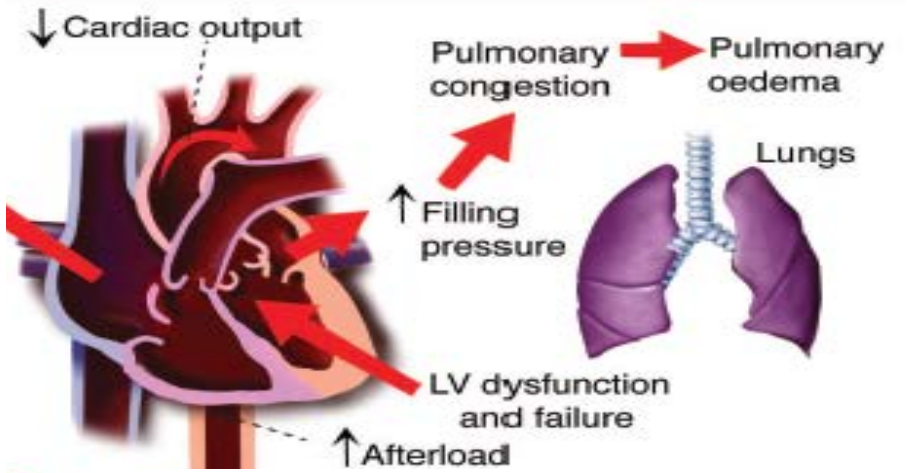
Merdji.H et al, Annales Int Care 2023

Diagnostic du choc cardiogénique retour à la définiton…

- Une pression artérielle systolique inférieure à 90 mmHg ou une pression artérielle moyenne inférieure à 65 mmHg pendant 30 minutes ;
- Une congestion pulmonaire ou une élévation des pressions de remplissage ;
- Des signes d'hypoperfusion périphérique : (a) confusion; (b) genoux froids et/ou marbrés; (c) oligurie; (d) lactate augmenté

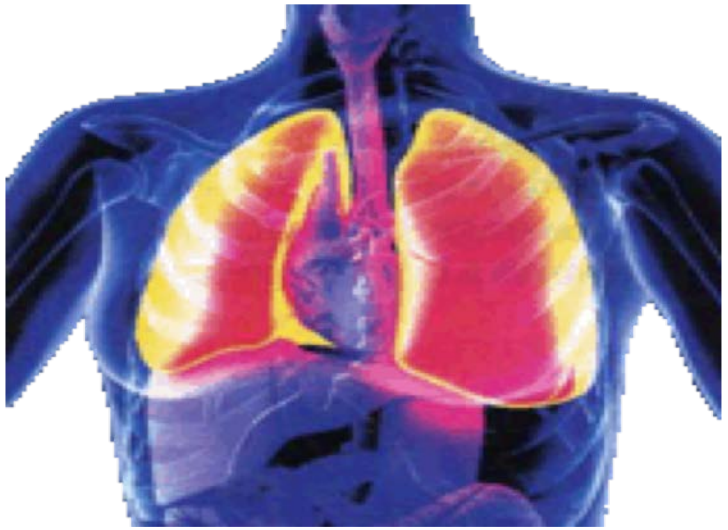
Levy *et al. Annals of Intensive Care* (2015) 5:17

Congestion à gauche



Arjola VP. Eur J Heart Failure 2018

Clinical



Biological

Nt-proBNP/ BNP

Blood gaz

!! Underlying COPD

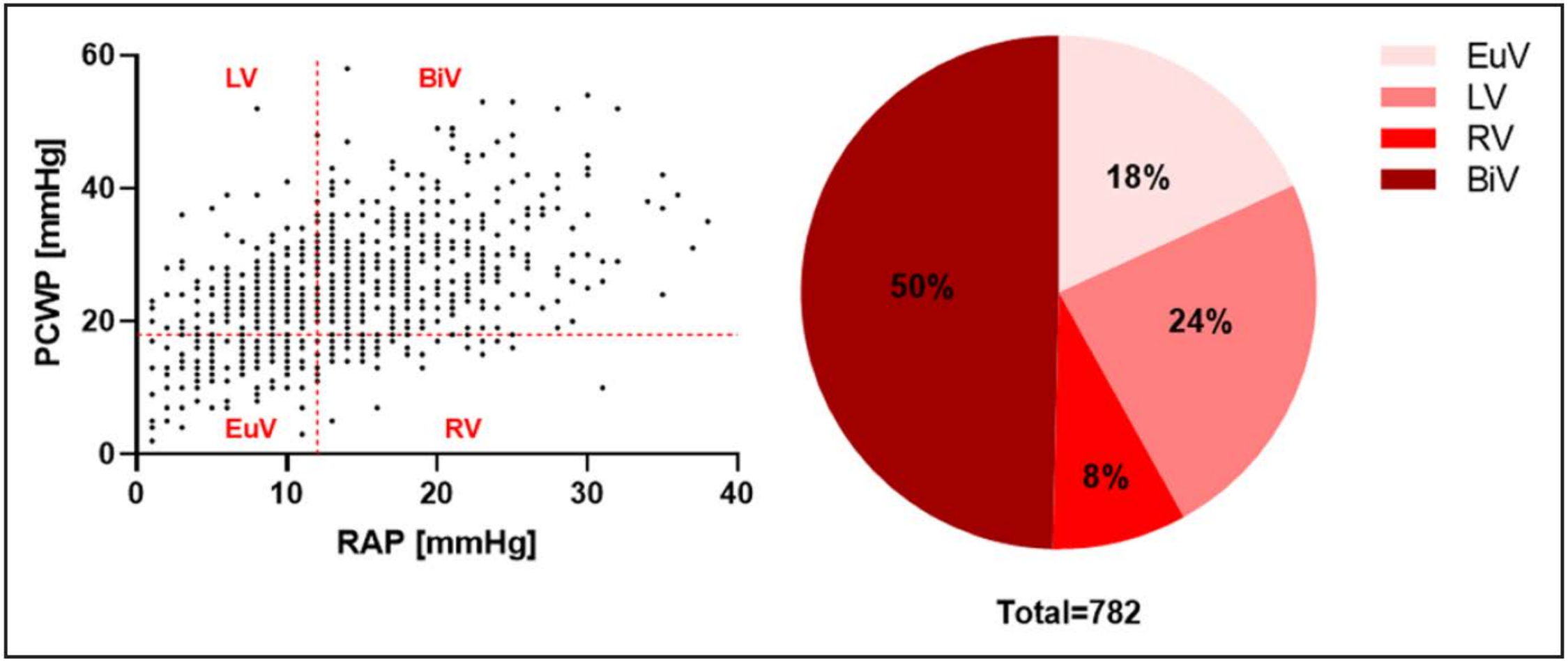
Hemodynamic

- **Swan** (PCWP > 15 mmHg)
- PICCO (EPEI?)

Radiological

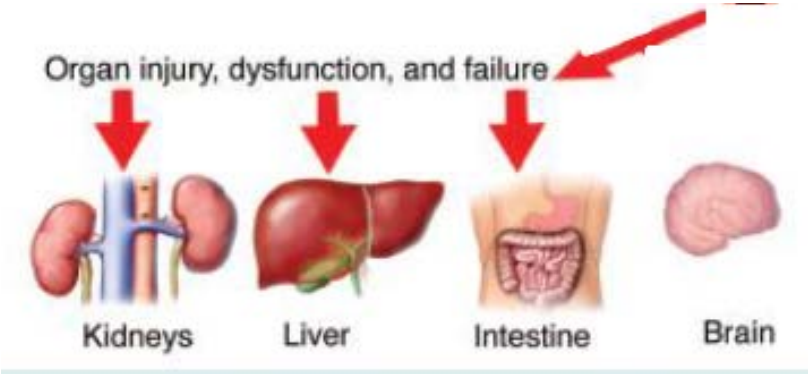
- **Chest X-ray** (Nale > 20% of APE…)
- **TTE** (LV filling pressure)
- **Lung US**. (bilateral B lines)

Congestion gauche et droite+++



Thayer.KL et al, Circ Heart Fail 2020

PCWP ≥ 18 mmHg and/or RAP ≥ 12



Organ malperfusion and congestion = Liver failure

Table 1
Distribution of abnormal liver function test values at different time points

Variable	Number of patients	Survivors median (IQR)	Abnormal	Non-survivors median (IQR)	Abnormal	p value*
Alanine aminotransferase (U/L)						
0 hours	176	38 (18–81)	52 (51%)	56 (25–121)	50 (68%)	0.03
12 hours	152	33 (15–70)	45 (49%)	74 (34–159)	43 (72%)	0.007
24 hours	139	28 (16–63)	39 (43%)	61 (29–130)	34 (71%)	0.002
Alkaline phosphatase (U/L)						
0 hours	176	60 (47–78)	14 (14%)	63 (49–82)	5 (7%)	0.22
12 hours	152	60 (47–72)	9 (10%)	57 (46–70)	4 (7%)	0.57
24 hours	139	57 (43–71)	6 (7%)	54 (42–69)	4 (8%)	0.74
Gamma-glutamyl transferase (U/L)						
0 hours	176	57 (34–106)	47 (46%)	47 (28–79)	31 (42%)	0.35
12 hours	152	55 (31–101)	42 (46%)	46 (32–81)	22 (37%)	0.32
24 hours	139	52 (31–99)	36 (40%)	43 (31–76)	15 (31%)	0.36
Total bilirubin (μmol/L)						
0 hours	176	9.6 (6.0–16.6)	12 (12%)	9.4 (5.6–15.4)	9 (12%)	0.99
12 hours	152	9.9 (6.8–15.7)	10 (11%)	9.2 (5.2–16.8)	3 (5%)	0.25
24 hours	139	10.7 (7.2–16.3)	9 (10%)	8.7 (5.6–18.1)	4 (8%)	0.99

Datas CARDSHOCK

Multivariable Cox regression analysis of factors associated with 90-day mortality

Variable	Hazard ratio (95% CI)	p value*
Altered mental status	1.51 (0.76–3.01)	0.24
Age (year increment)	1.04 (1.01–1.07)	0.01
Prior myocardial infarction	1.64 (0.85–3.17)	0.14
Prior coronary bypass	1.83 (0.73–4.57)	0.20
Acute coronary syndrome etiology	1.11 (0.48–2.60)	0.81
Left ventricular ejection fraction (% increment)	0.98 (0.96–1.00)	0.11
Estimated glomerular filtration rate (mL/min/1.73 m2 increment)	1.00 (0.99–1.02)	0.83
Lactate at baseline (mmol/L increment)	1.11 (1.04–1.19)	0.002
ALT at baseline (U/L increment)	1.00 (1.00–1.00)	0.75
ΔALT>+20%	3.16 (1.72–5.82)	<0.001

Jantti.T et al, Am J Cardiol 2017

Table 3 Predictors of in-hospital mortality in cardiogenic shock

Variable	Adjusted OR (95% CI)	P-value[a]
Prior CABG	10.7 (1.8–64.7)	0.01
ACS aetiology	7.4 (1.9–29.8)	0.005
Confusion	3.0 (1.1–8.1)	0.03
Previous myocardial infarction	3.2 (1.2–8.2)	0.02
Blood lactate (per mmol/L)	1.4 (1.2–1.6)	<0.001
LVEF (per % decrease)	1.06 (1.02–1.09)	0.001
Age (per year)	1.04 (1.00–1.08)	0.08
Systolic blood pressure (per mmHg decrease)	1.03 (0.99–1.06)	0.09

Harjola.VP et al, Eur J Heart Fail 2015

Organ malperfusion and congestion = Cerebral dysfunction

CARDSHOCK: 219 patients

68% with altered mental status

Confusion, drowsiness, coma,…

IABP-SHOCK 2 study

Table 2. Clinical Course before Randomization.*

Variable	IABP (N=301)	Control (N=299)
Sign of impaired organ perfusion — no./total no. (%)		
Altered mental status	215/300 (71.7)	232/299 (77.6)
Cold, clammy skin and extremities	257/300 (85.7)	245/299 (81.9)
Oliguria	90/300 (30.0)	99/299 (33.1)
Serum lactate >2.0 mmol/liter	226/300 (75.3)	218/298 (73.2)
Serum lactate — mmol/liter		
Median	3.6	4.7
Interquartile range	2.1–7.2	2.3–8.2

Thiele.H et al, NEJM 2012

Cerebral dysfunction: pronostic in CS

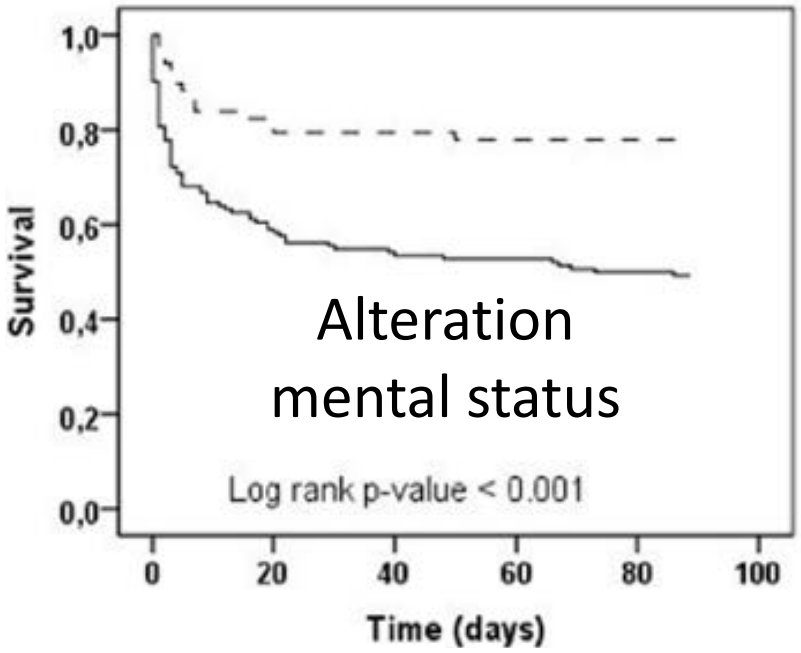


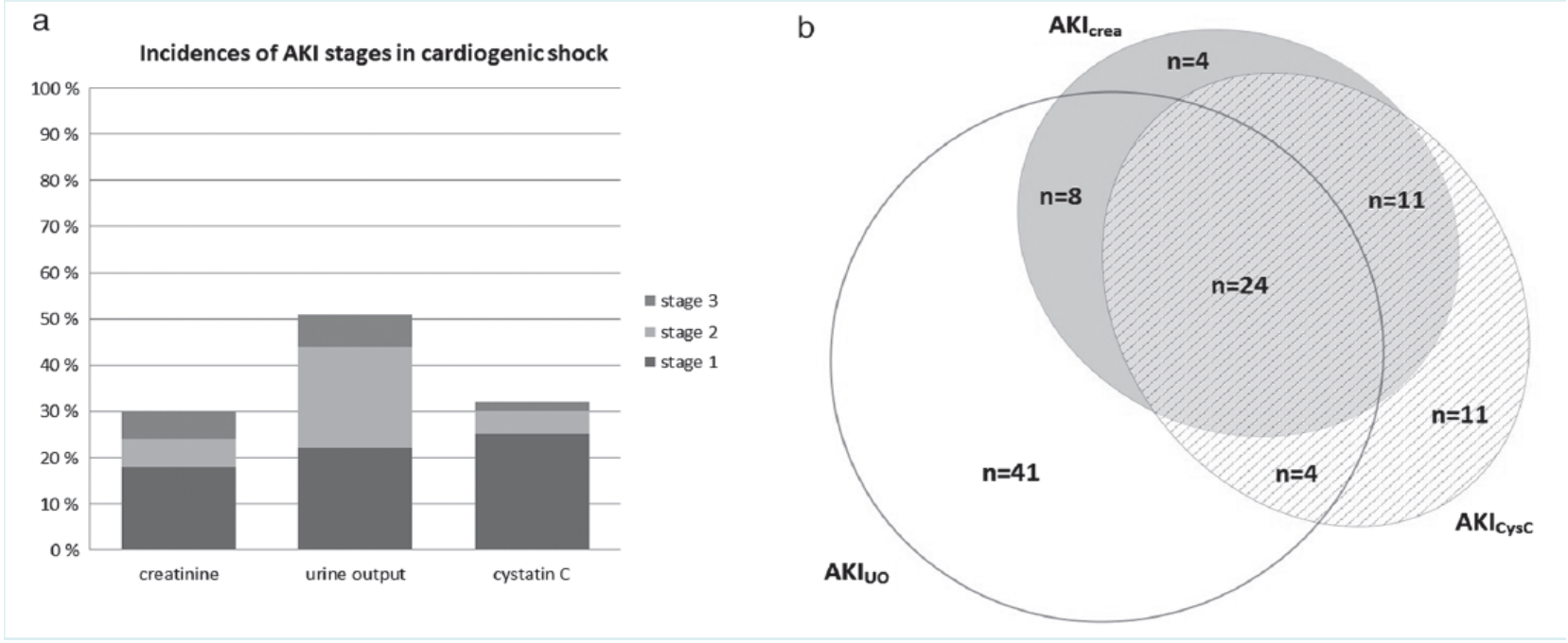
Table 3. Multivariable regression analysis for altered mental status.

	Odds ratio	95% CI	*p*-value
Age, per one year increase	1.03	1.00–1.06	0.09
Male gender	1.2	0.53–2.8	0.6
ACS aetiology	1.6	0.67–4.0	0.3
Systolic blood pressure, per 1 mmHg decrease	1.02	0.99–1.04	0.2
LVEF, per 1% decrease	1.02	1.00–1.05	0.07
Plasma lactate, per 1 mmol/l increase	1.06	0.93–1.2	0.4
Arterial pH, per 0.1 decrease	1.6	1.08–2.2	**0.02**
Plasma glucose, per 1 mmol/l increase	1.01	0.96–1.07	0.7
Resuscitation	1.5	0.66–3.4	0.3

	All	Patients with altered mental status	Patients with normal mental status	*p*-value
Mortality				
In-hospital	79 (37)	67 (46)	12 (18)	< 0.001
90-day	88 (42)	73 (51)	15 (22)	< 0.001

Kataja.A et al, Eur Heart J Acute Cardiovasc Care 2017

Organ malperfusion and congestion = Renal dysfunction



Datas CARDSHOCK

AKI in 30- 50% of CS

Table 2 Unadjusted and adjusted associations of the acute kidney injury definitions with 90-day mortality

Adjustment model	AKI_{crea}		AKI_{UO}		UO < 0.3 mL/kg/h for 6 h	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Unadjusted	7.5 (3.5–12.3)	<0.001	2.1 (1.05–4.0)	0.035	4.7 (2.2–9.8)	0.001
Model 1	7.3 (3.3–16.4)	<0.001	1.7 (0.8–3.4)	0.15	3.7 (1.7–8.0)	0.001
Model 2	7.5 (3.2–17.8)	<0.001	1.6 (0.8–3.5)	0.2	3.9 (1.7–9.0)	0.001
Model 3	12.2 (4.1–36.0)	<0.001	1.5 (0.6–3.5)	0.4	3.6 (1.4–9.3)	0.008

Tarwamaski.T et al, Eur J Heart Fail 2017

Organ malperfusion and congestion = Lactate increase (1)

Characteristic	All ($n = 219$)
Systolic blood pressure, mmHg	78 (14)
Diastolic blood pressure, mmHg	47 (10)
Mean arterial pressure, mmHg	57 (11)
Heart rate, b.p.m.	90 (28)
Sinus rhythm	170 (78)
Clinical findings, *n* (%)	
Cold periphery	207 (95)
Confusion	148 (68)
Oliguria	121 (55)
Lactate >2 mmol/L	155 (71)
Resuscitated from cardiac arrest	62 (28)
Time from detection of shock to study inclusion, min	105 (0–210)
Baseline echocardiography	
LVEDD (mm)	52 (9)
LVEF (%)	33 (14)
LVEF <40%	135 (65)
Mitral regurgitation (moderate or severe), *n* (%)	73 (35)
Biochemistry	
Blood haemoglobin (g/L)	128 (22)
Sodium (mmol/L)	137 (5)
Potassium (mmol/L)	4.2 (0.8)
Arterial blood lactate (mmol/L)	2.8 (1.7–5.8)
Arterial blood pH	7.30 (7.20–7.40)
hsTnT (ng/L)	2190 (388–5418)
NT-proBNP (pg/mL)	2710 (585–9434)
Creatinine (mmol/L)	104 (78–140)
eGFR (mL/min/1.73 m^2)	61 (41–87)
CRP (g/L)	16 (4–54)
In-hospital length of stay, days	12 (7–25)
In-hospital mortality, *n* (%)	80 (37)

Harjola.VP et al, Eur J Heart Fail 2015

CARDSHOCK study

Table 3 Predictors of in-hospital mortality in cardiogenic shock

Variable	Adjusted OR (95% CI)	*P*-value[a]
Prior CABG	10.7 (1.8–64.7)	0.01
ACS aetiology	7.4 (1.9–29.8)	0.005
Confusion	3.0 (1.1–8.1)	0.03
Previous myocardial infarction	3.2 (1.2–8.2)	0.02
Blood lactate (per mmol/L)	1.4 (1.2–1.6)	<0.001
LVEF (per % decrease)	1.06 (1.02–1.09)	0.001
Age (per year)	1.04 (1.00–1.08)	0.08
Systolic blood pressure (per mmHg decrease)	1.03 (0.99–1.06)	0.09

TABLE 2 Results of Multivariable Cox Regression Analysis

	Hazard Ratio (95% CI)	Parameter Estimate	p Value
Age >73 yrs	1.54 (1.16–2.05)	0.43	0.003
History of stroke	2.09 (1.39–3.15)	0.73	0.0004
Glucose >10.6 mmol/l (191 mg/dl)*	1.48 (1.10–2.01)	0.39	0.01
Creatinine >132.6 µmol/l (1.5 mg/dl)*	1.57 (1.17–2.11)	0.44	0.003
Arterial lactate >5 mmol/l*	1.98 (1.47–2.66)	0.68	<0.0001
TIMI flow grade <3 after PCI	2.73 (1.11–6.73)	0.72	0.03

*At admission.

Pöss.J et al, JACC 2017

Organ malperfusion and congestion = Lactate increase (1)

IABP-SHOCK 2 trial

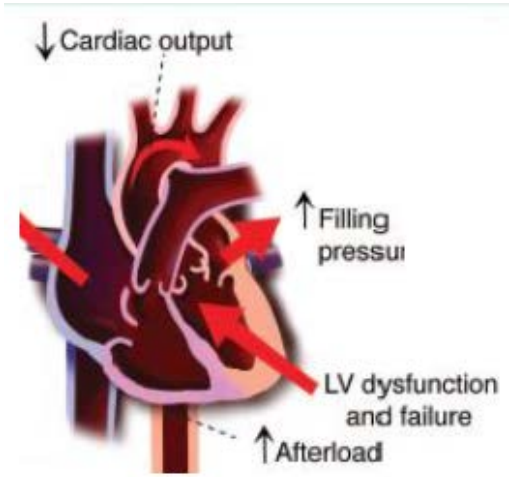
Table 2. Clinical Course before Randomization.*

Variable	IABP (N=301)	Control (N=299)
Sign of impaired organ perfusion — no./total no. (%)		
Altered mental status	215/300 (71.7)	232/299 (77.6)
Cold, clammy skin and extremities	257/300 (85.7)	245/299 (81.9)
Oliguria	90/300 (30.0)	99/299 (33.1)
Serum lactate >2.0 mmol/liter	226/300 (75.3)	218/298 (73.2)
Serum lactate — mmol/liter		
Median	3.6	4.7
Interquartile range	2.1–7.2	2.3–8.2

Thiele.H et al, NEJM 2012

Hyperlactatemia 75% of CS

Intérêt de l'ETT pour évaluer la dysfonction systolique

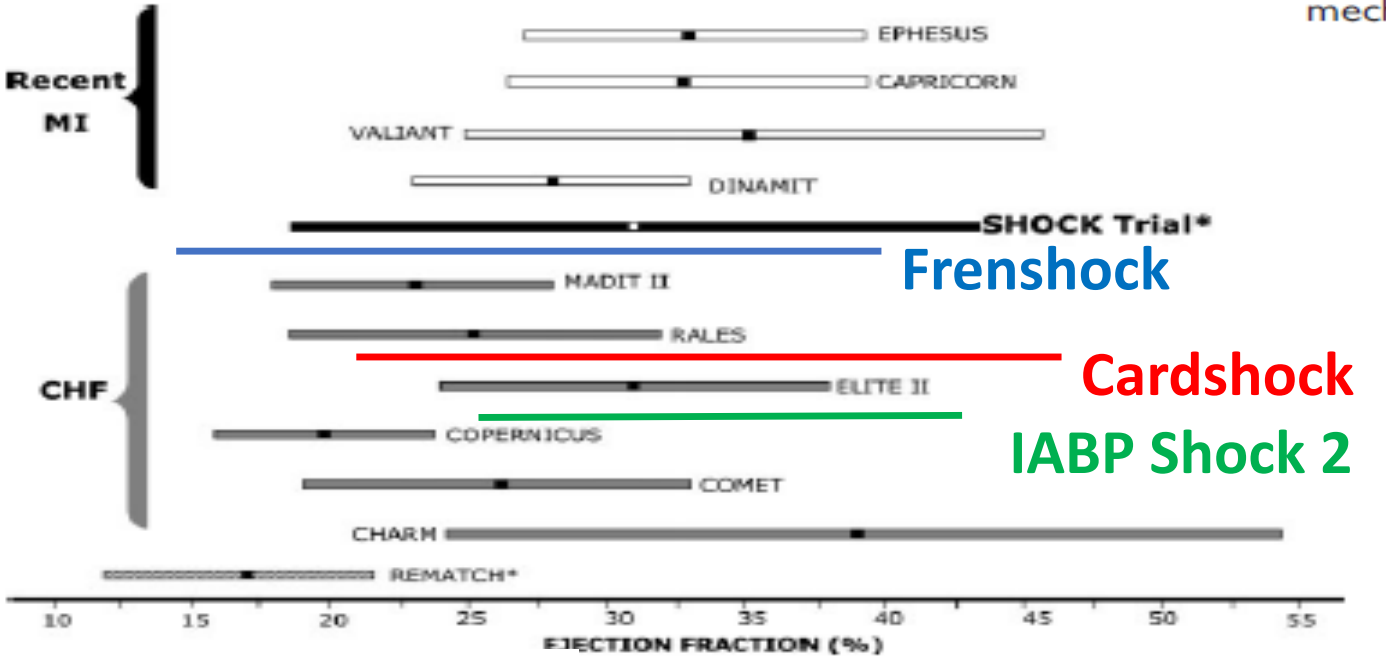


Arjola VP. Eur J Heart Failure 2018

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

Immediate Doppler echocardiography is indicated to assess ventricular and valvular functions, loading conditions, and to detect mechanical complications.	I	C

Ibanez.B et al, Eur Heart J 2017



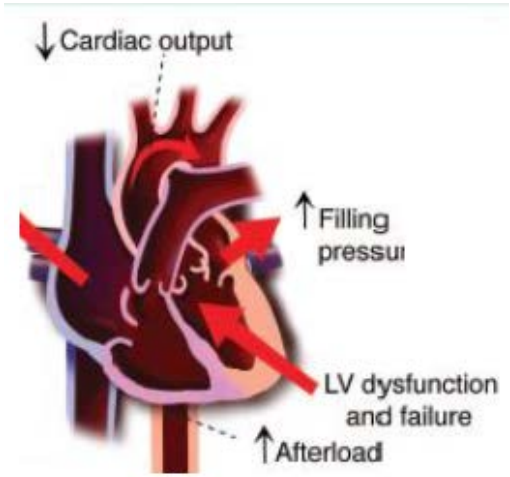
Frenshock

Cardshock

IABP Shock 2

Reynolds HR. Circ 2008

Limits and insufficiency of LVEF evaluation !



Arjola VP. Eur J Heart Failure 2018

Intérêt de l'ETT pour évaluer la dysfonction diastolique

Sensibility 75% / Specificity 74%
PPV 39% / NPV 93%

17%

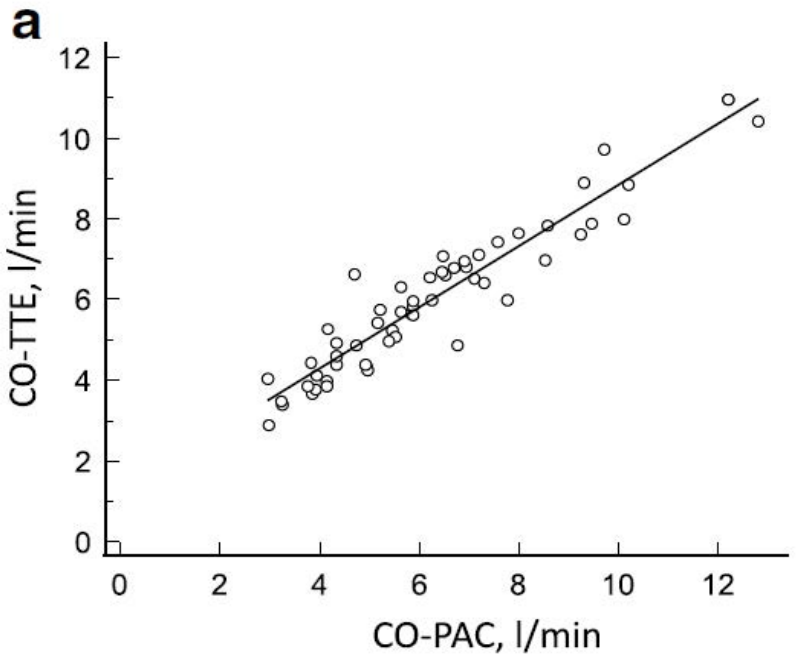
Tables 4 Echo estimates of LVFP and LVEDP according to 2016 ASE/EACVI recommendations

Parameters	Echo Normal LV filling pressure *N* = 108	Echo Elevated LV filling pressure *N* = 24	Echo Indeterminate or Cannot determine *N* = 27
LVEDP < 15 mmHg	**70 (65%)**	**5 (21%)**	**20 (74%)**
LVEF ≥ 50%	62 (57%)	1 (4.2%)	16 (59%)
LVEF < 50%	8 (8%)	4 (16.8%)	4 (15%)
LVEDP ≥ 15 mmHg	**38 (35%)**	**19 (79%)**	**7 (26%)**
LVEF ≥ 50%	32 (30%)	4 (17%)	5 (19%)
LVEF < 50%	6 (5%)	19 (62%)	2 (7%)

ACE, angiotensin converting enzyme; EF, ejection fraction; EDP, end-diastolic pressure; LAV, left atrial volume; LV, left ventricle; TR, tricuspid regurgitation.

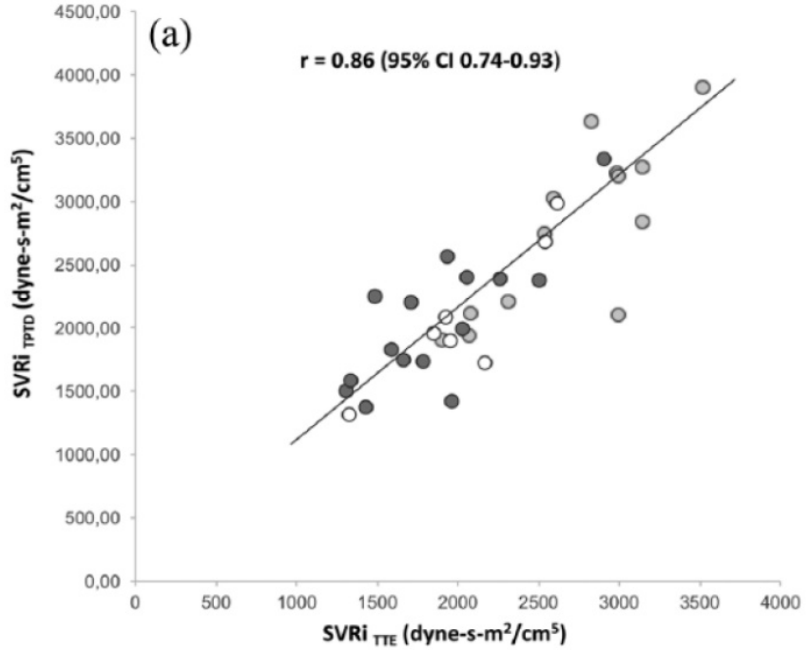
Lancellotti.P et al, Eur J Cardiovasc Imaging 2017

Intérêt de l'ETT pour évaluer le débit cardiaque et les résistance vasculaire systémiques



Mercado.P et al, Crit Care 2017

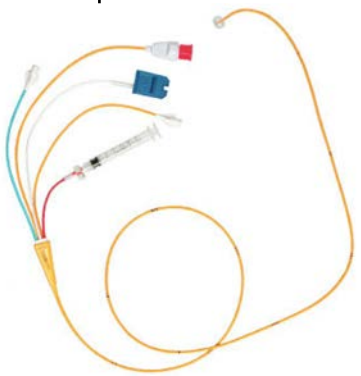
- **CI = (LVOT area x VTI) x HR / BSA**
  - Normal > 2.2l/min/m2
- **SVRi = (MAP-RAP) x 80 / CI**
  - Normal 800-1200

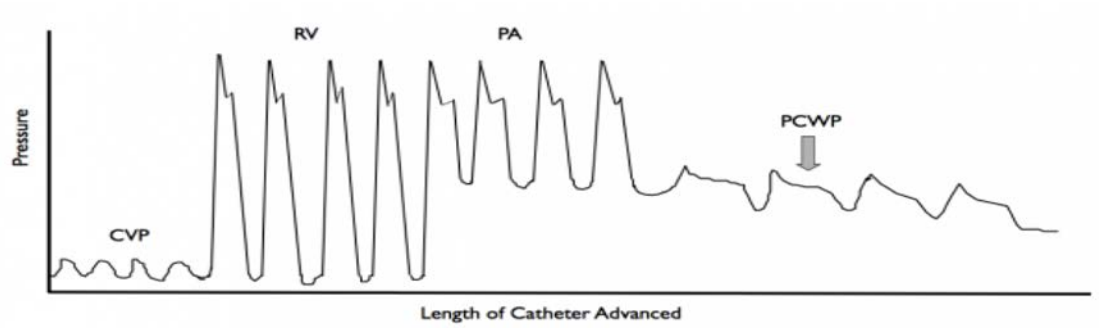


- Approche non invasive au lit du patient
- Discussion Vasopresseurs vs inotropes

Gaubert.M et al, EHJ ACC 2018

Place of invasive hemodynamic evaluation





IIb B

Ibanez.B et al, Eur Heart J 2017

LV function and systemic perfusion

- **CO and CI** (thermodilution)
  - Patho < 1.8 - 2.2L/min/m²
- **PCWP** = LV overload?
  - Patho > 15
- **SVR = (MAP- RAP)/CO**
- **SVO2**

RV and pulmonary function

- **PVR= (mPAP-PCWP) / CO** en UW
  - Normal 0.5-1.1
- **PAPi = (sPAP-dPAP)/RAP**
  - Patho < 1.8 - 2
- **RAP/PCWP**
  - **Patho >** 0.55-0.59

Dynamic evaluation +++: after vascular filling or depletion, inotrops and/or vasopressors

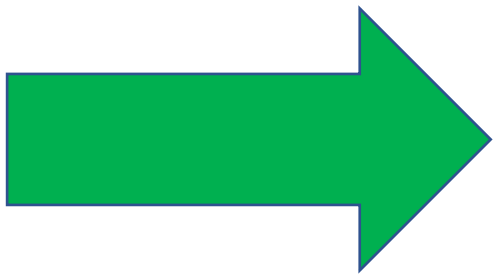
Cathétérisme de l'artère pulmonaire

Avantages

- Close monitoring
- Measure of systemic and pulmonary parameters
- SVO2 monitoring
- Awake or sedated patient

Limites

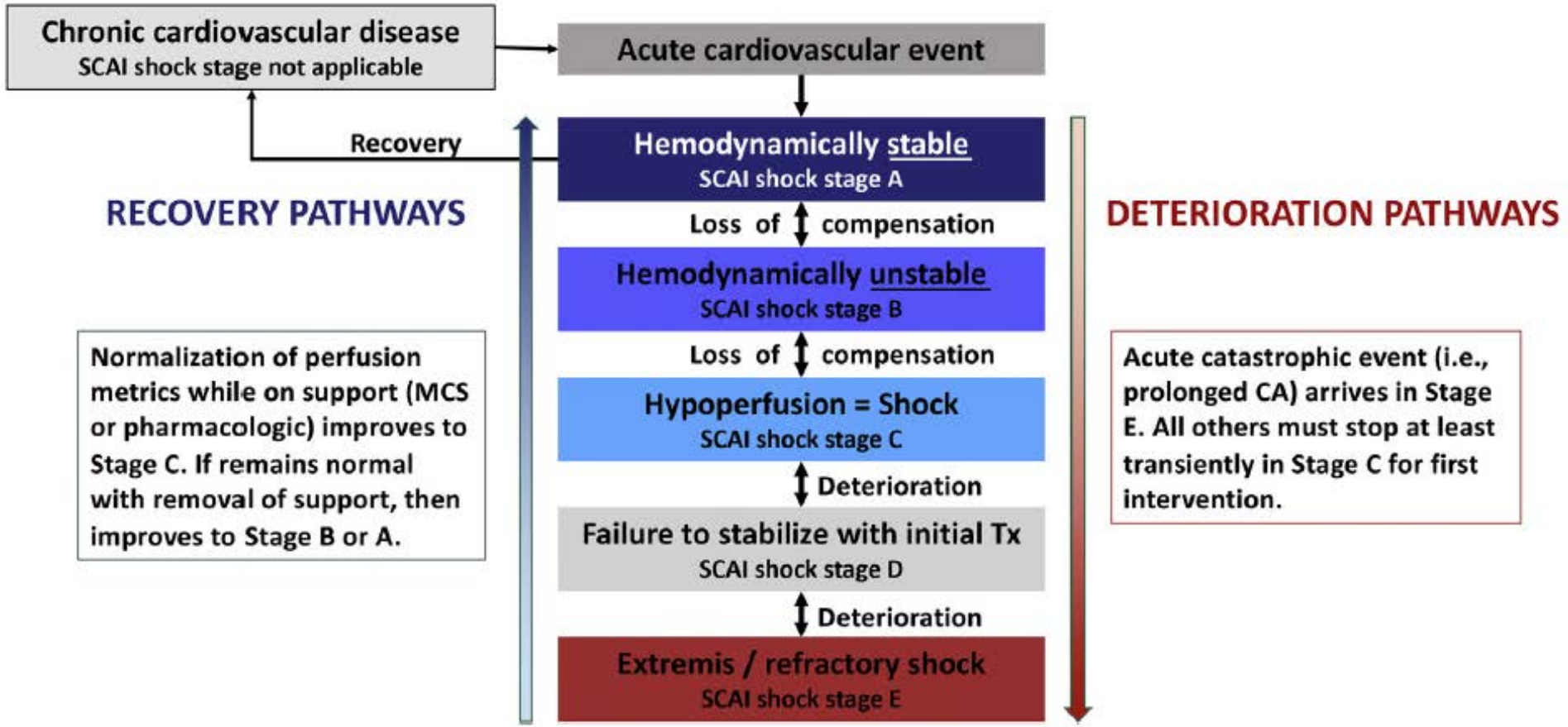
- Invasive
- Need technical skill and training
- Vascular and infectious complications
- No effect found on mortality



- Not systematic ?
- In case of **RV dysfunction**
- In case of **mixed shock**
- In case of **treatment failure**
- Management **monitoring**

Shah.MR et al. JAMA 2005

Peut-on identifier les patients qui vont évoluer vers un choc réfractaire ?



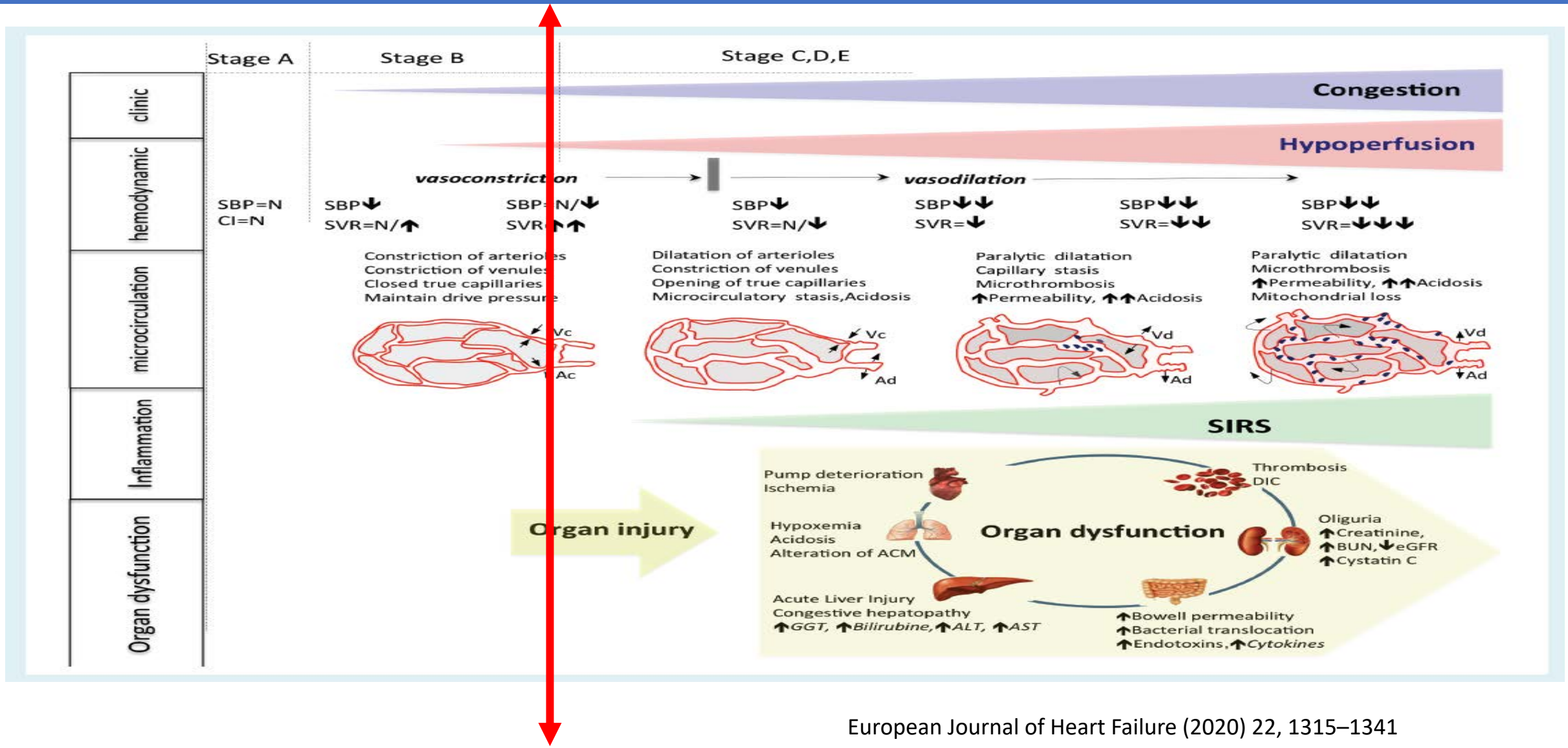
But:

- Same stage for patients stabilized by dobutamine 2.5g/kg/min or ECPELLA…..
- Same stage in case of previous CA, mixed shock,…

Need to integrate « modifiers » and pronostic evaluation available at patient's bedside as a « point of care » !

Moosamy.P et al, J Am Col Cardiol 2022

Identifier précocement /prévenir les dysfonctions d'organe



European Journal of Heart Failure (2020) 22, 1315–1341

Prédicteurs du choc cardiogénique réfractaire

Variables	OR	95% CI	*P*-value
Age >60 years	1.90	1.03–3.49	0.04
Recent history of CPR	2.12	1.27–3.54	0.003
ECMO initiation under CPR	4.12	2.17–7.83	<0.0001
Oligo-anuria	2.61	1.38–4.94	0.002
Inotropic score >20	2.06	1.22–3.46	0.007
pH ≥ 7.30	0.38	0.20–0.73	0.002
Dilated cardiomyopathy vs. non-dilated non-ischaemic acute cardiomyopathy	1.67	0.78–3.57	0.18
AMI vs. non-dilated non-ischaemic acute cardiomyopathy	1.54	0.86–2.78	0.15

2005-2009 75 patients sous ECMo

European Heart Journal (2013) **34**, 112–120

De nombreux scores, avec toutes leurs limites

Management of cardiogenic shock complicating myocardial infarction: an update 2019

Holger Thiele[1,2]*, E. Magnus Ohman[3], Suzanne de Waha-Thiele[4], Uwe Zeymer[5], and Steffen Desch[1,2]

Until recently, a limitation of all published scores in the setting of classical CS was the lack of sufficient validation and also applicability in clinical practice. Currently, there is only one CS score with both internal and external validation derived from the IABP-SHOCK II trial (*Table 2*).[39] Based on six variables—including the biomarkers lactate, creatinine and glucose—with a maximum of nine points this IABP-SHOCK II score divides into three risk categories. Patients in the low (0–2 points), intermediate (3 or 4 points), and high-risk categories (5–9 points) have 30-day mortality risk of 20–30%, 40–60%, and 70–90%, respectively. This score may also be a suitable tool to tailor more aggressive treatment strategies such as MCS. However, this requires further validation in randomized trials. There are also scores for prediction of outcome in patients with MCS mainly ECMO (*Table 2*).

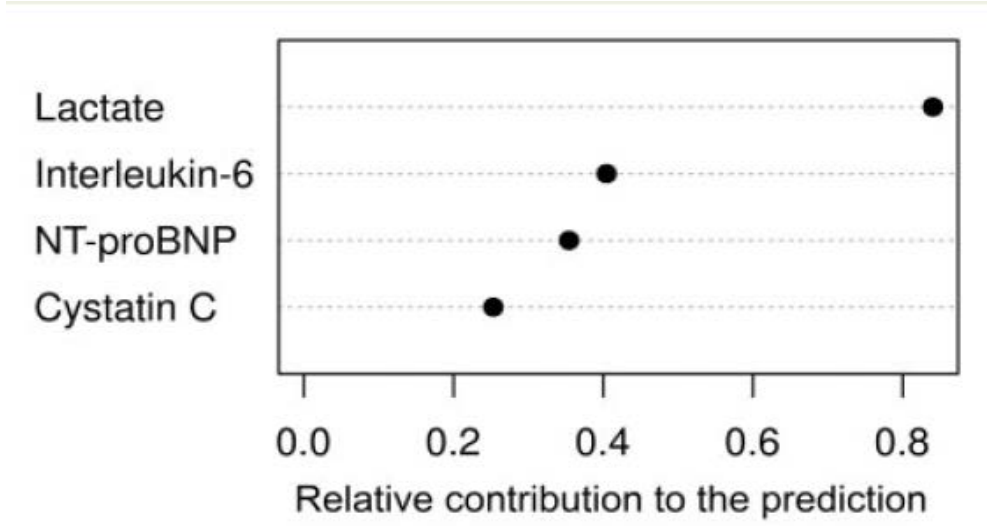
European Heart Journal (2019) **40**, 2671–2683

Intérêts des paramètres biologiques ?

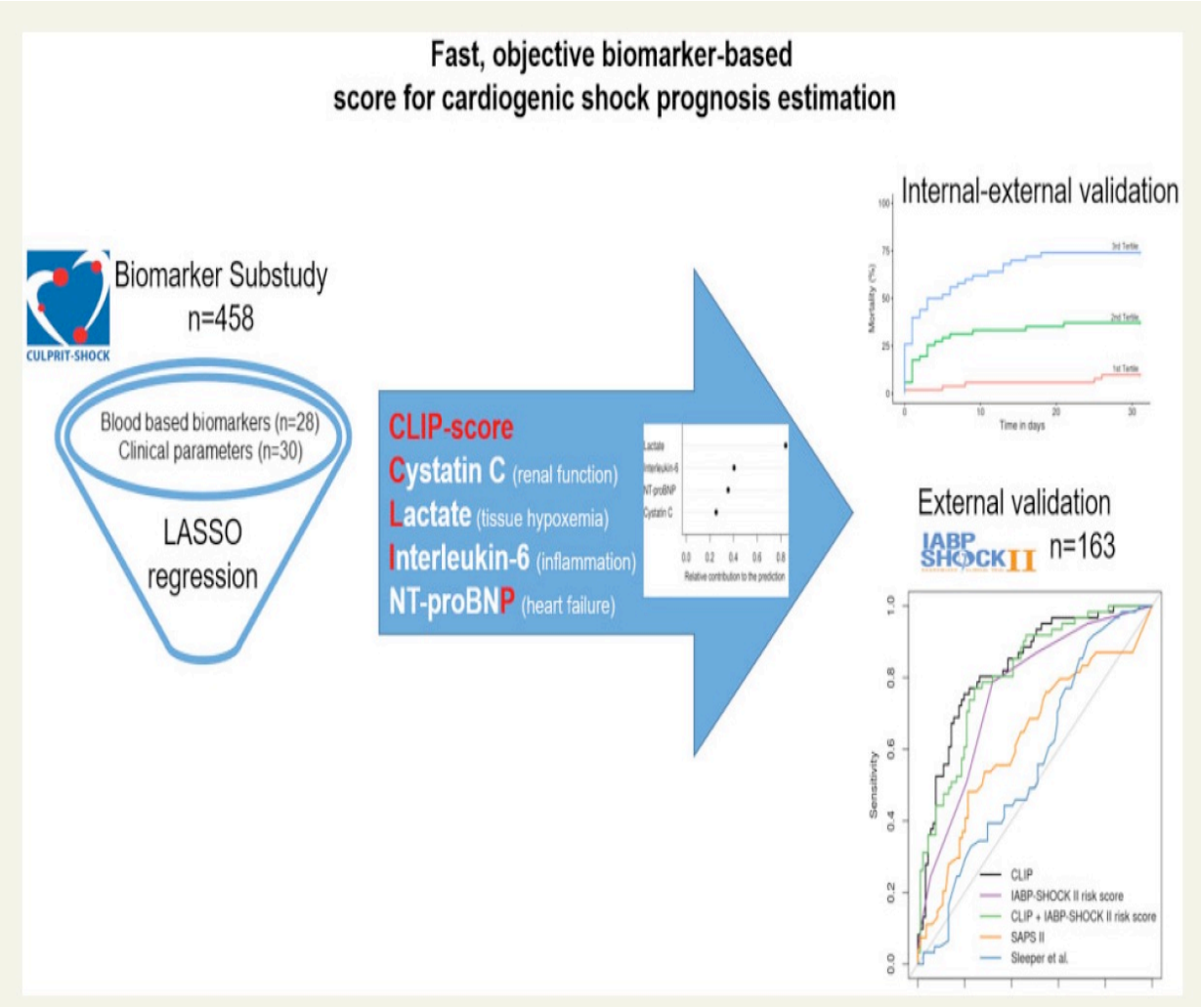
ESC European Society of Cardiology
European Heart Journal (2021) **42**, 2344–2352
doi:10.1093/eurheartj/ehab110

CLINICAL RESEARCH
Heart failure and cardiomyopathies

The novel cystatin C, lactate, interleukin-6, and N-terminal pro-B-type natriuretic peptide (CLIP)-based mortality risk score in cardiogenic shock after acute myocardial infarction



458 patients



Intérêts des paramètres ETT ?

A novel mortality risk score predicting intensive care mortality in cardiogenic shock patients treated with veno-arterial extracorporeal membrane oxygenation

Sakir Akin, MD PhD[a,b,c,*], Kadir Caliskan, MD PhD[a], Osama Soliman, MD PhD[a], Rahatullah Muslem, BSc PhD[d], Goksel Guven, MD[a,b], Robert J. van Thiel, MD[b], Ard Struijs, MD PhD[b], Diederik Gommers, MD PhD[b], Felix Zijlstra, MD PhD[a], Jan Bakker, MD PhD[b,e,f,g], Dinis dos Reis Miranda, MD, PhD[b]

Highlights

- This is the largest echocardiography-based prediction model after VA-ECMO.
- One out of three patients did not survive ICU after VA-ECMO.
- Three out of four non-survivors in the ICU had biventricular failure.
- Adding RV function on echocardiography to the existing SOFA score improves significantly the prediction of ICU mortality.
- Dedicated evaluation of the right ventricular function in patients with VA-ECMO is highly recommended.

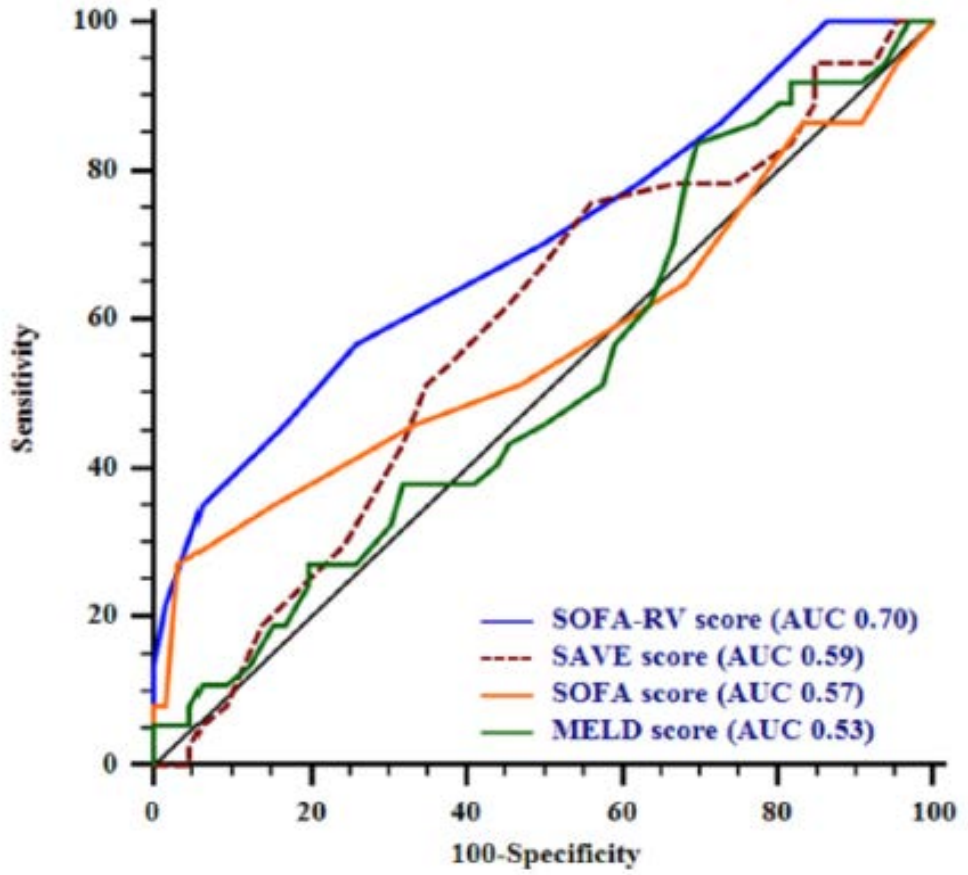


Fig. 4. Receiver Operating Characteristic (ROC) curves of risk scores for predicting ICU mortality.

Conclusions

- Définition clinique et unique
- Plusieurs profils de sévérité indépendamment de l'étiologie
- Mortalité dramatiquement élevée
- Diagnostic rester simple et pragmatique
- Importance d'identifier les patients qui vont développer les formes les plus graves (« phénotypes de patients »)
- Comment ?...