

LE CHOC CARDIOGENIQUE DANS L'URGENCE

Nicolas DEYE

MD, PhD

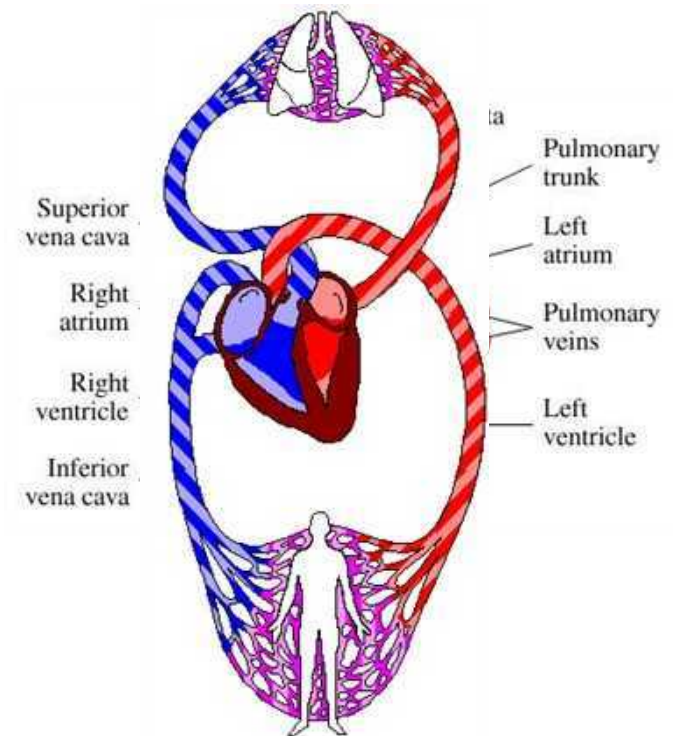
Réanimation Médicale Toxicologique

Inserm UMR-S 942

Hôpital Universitaire Lariboisière

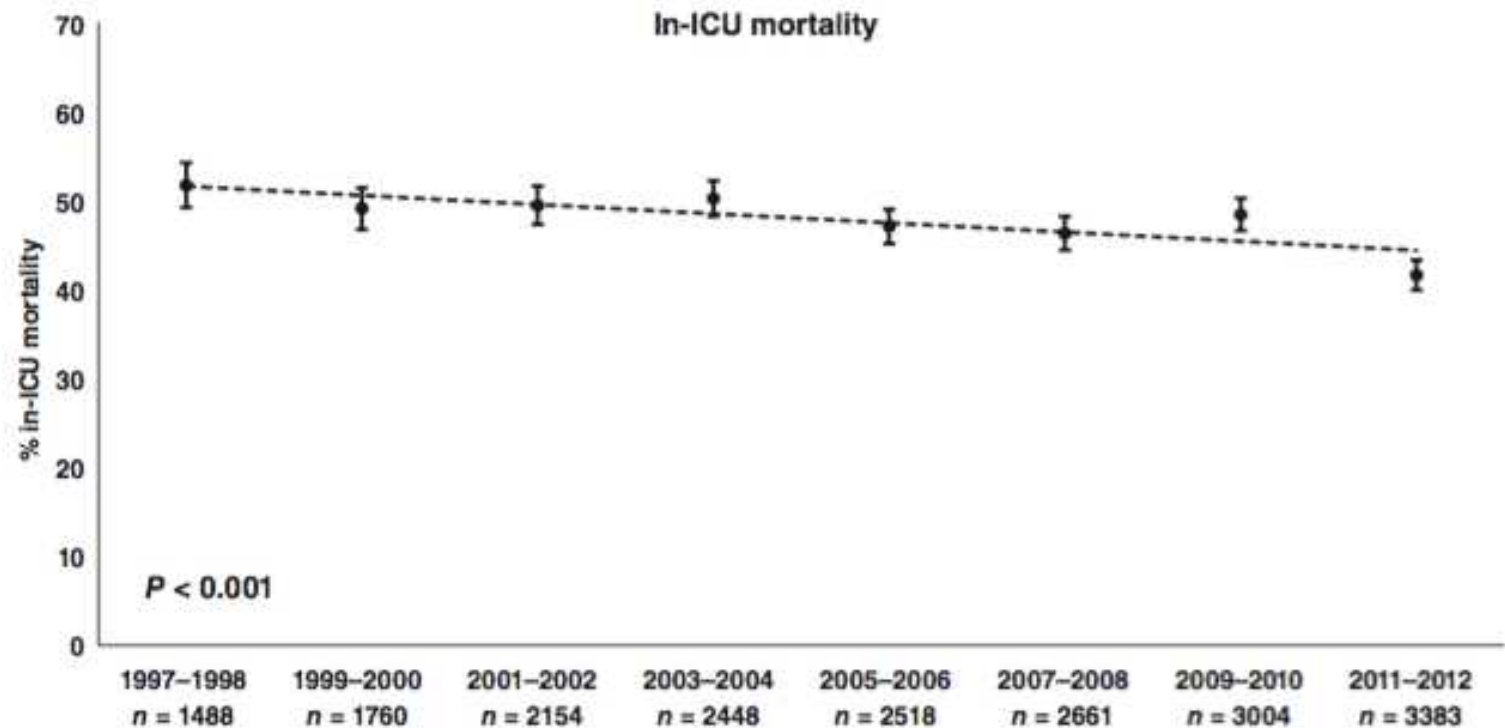
Paris 10^{ème} arrondissement

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Pas de conflit d'intérêt pour cette présentation
(past congress and travel fees for Bard and Zoll: cardiac arrest)

Épidémiologie du choc cardiogénique



- ✓ 1997 – 2012 : base CUB-Réa (316.905 admissions)
- ✓ Choc cardiogénique : 6,1% des admissions en réanimation
- ✓ **Mortalité en baisse ~ 40-50%**

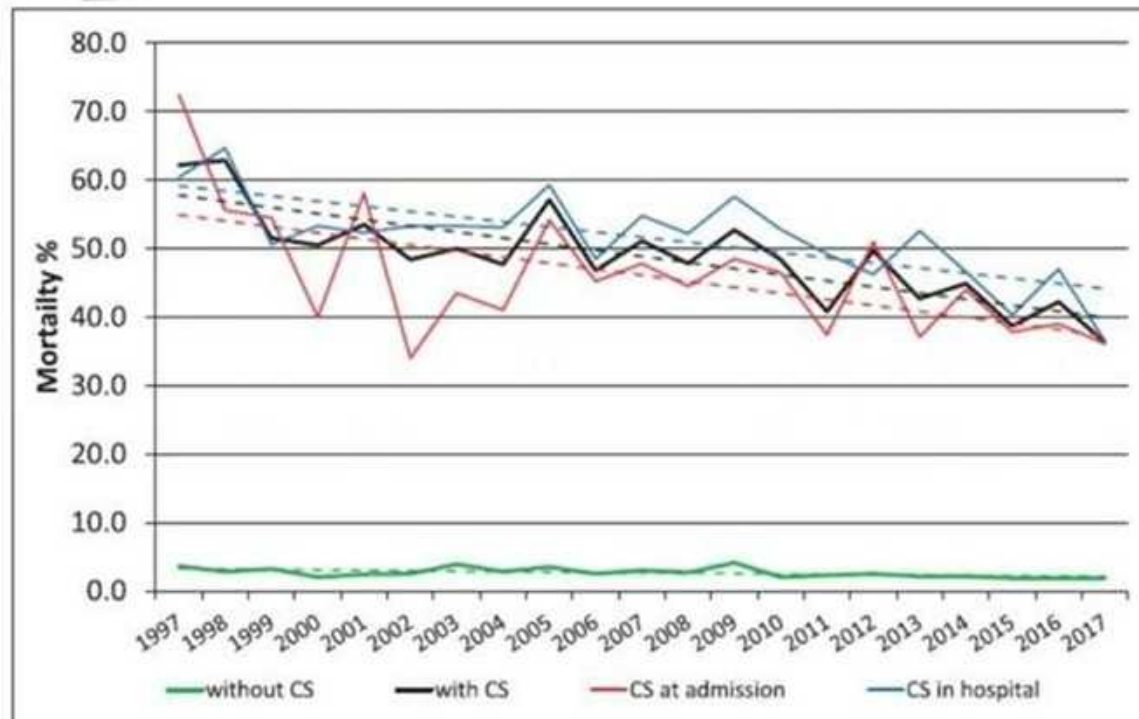
Épidémiologie du choc cardiogénique

Circulation: Cardiovascular Interventions

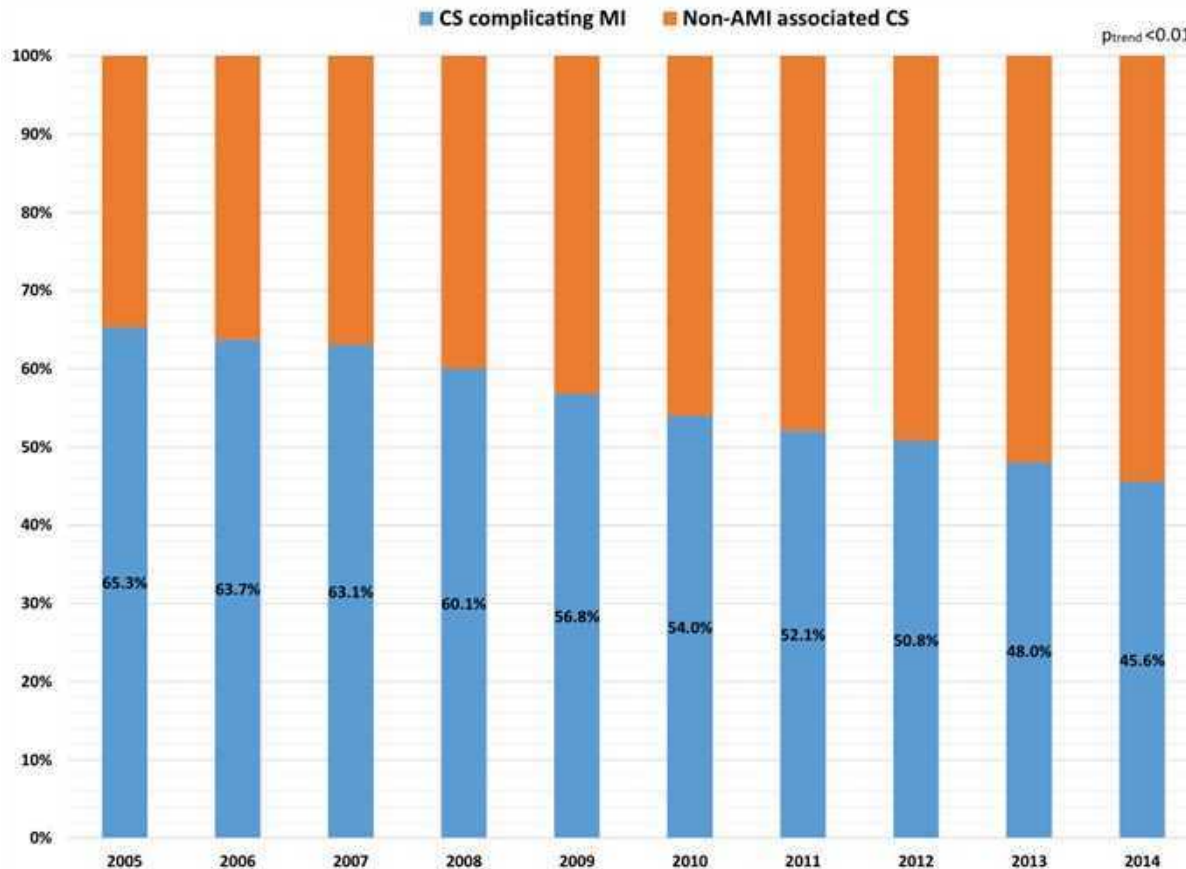
ORIGINAL ARTICLE

Twenty-Year Trends in the Incidence and Outcome of Cardiogenic Shock in AMIS Plus Registry

Figure 4. Trends in in-hospital mortality of patients with acute myocardial infarction according to cardiogenic shock (CS). Percentage in the table indicates percentage of overall CS. Dotted lines indicate trendlines.



Épidémiologie du choc cardiogénique



- Ischemic cardiogenic shock:
 - Main etiology
 - Net decrease
- CS from others etiologies:
 - Net increase
 - Miscellaneous

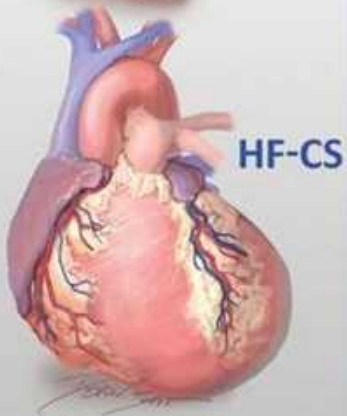
Épidémiologie du choc cardiogénique

Cardiogenic Shock From Heart Failure
Versus Acute Myocardial Infarction: Clinical
Characteristics, Hospital Course, and 1-Year
Outcomes

Sinha. *Circulation Heart Failure* 2022

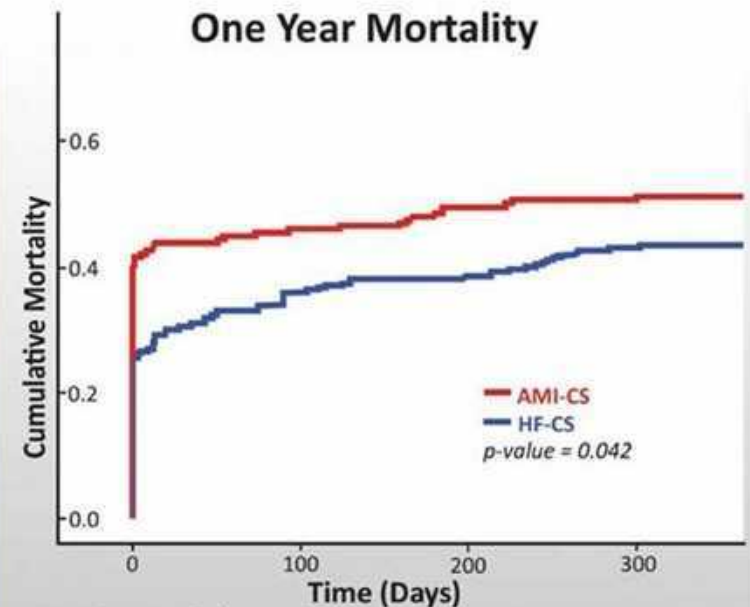


AMI-CS



HF-CS

Baseline Characteristics	Hospital Course
↑ Age ↑ Diabetes ↑ Vasopressors ↑ Cardiac Arrest	↑ Temporary MCS ↑ Major Bleeding ↑ Vascular Access Complications
↓ LV Ejection Fraction ↓ Cardiac Power Output ↑ Pulmonary Capillary Wedge Pressure ↑ Pulmonary Artery Pulsatility Index	↑ Durable MCS ↑ Heart Transplant ↑ Length of Stay



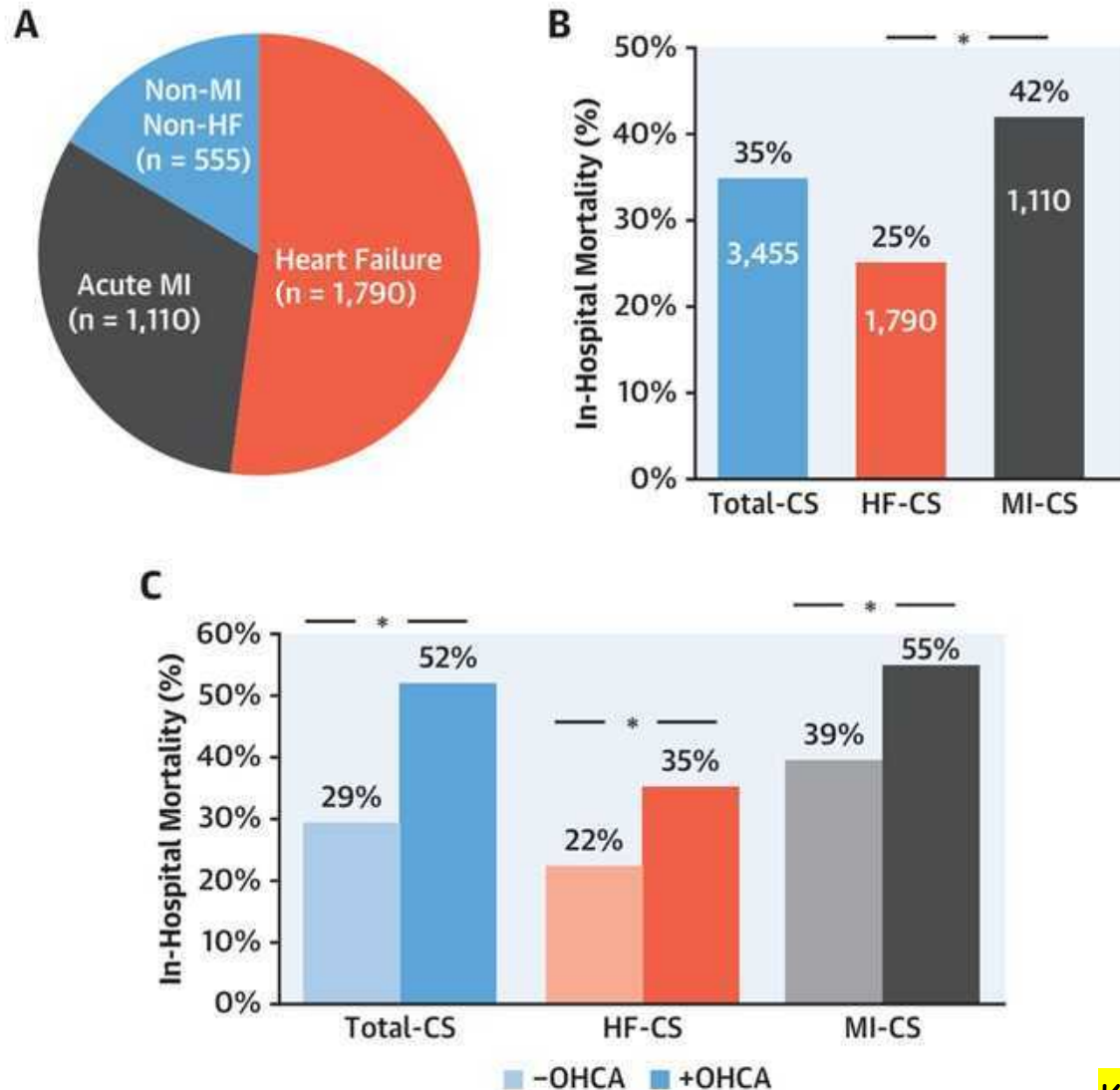
Number at risk

AMI-CS	219	109	100	90
HF-CS	301	183	170	155
	0	100	200	300

Time (Days)

Épidémiologie du choc cardiogénique

FIGURE 1 Etiology and Clinical Outcomes of Cardiogenic Shock Using the CSWG Registry

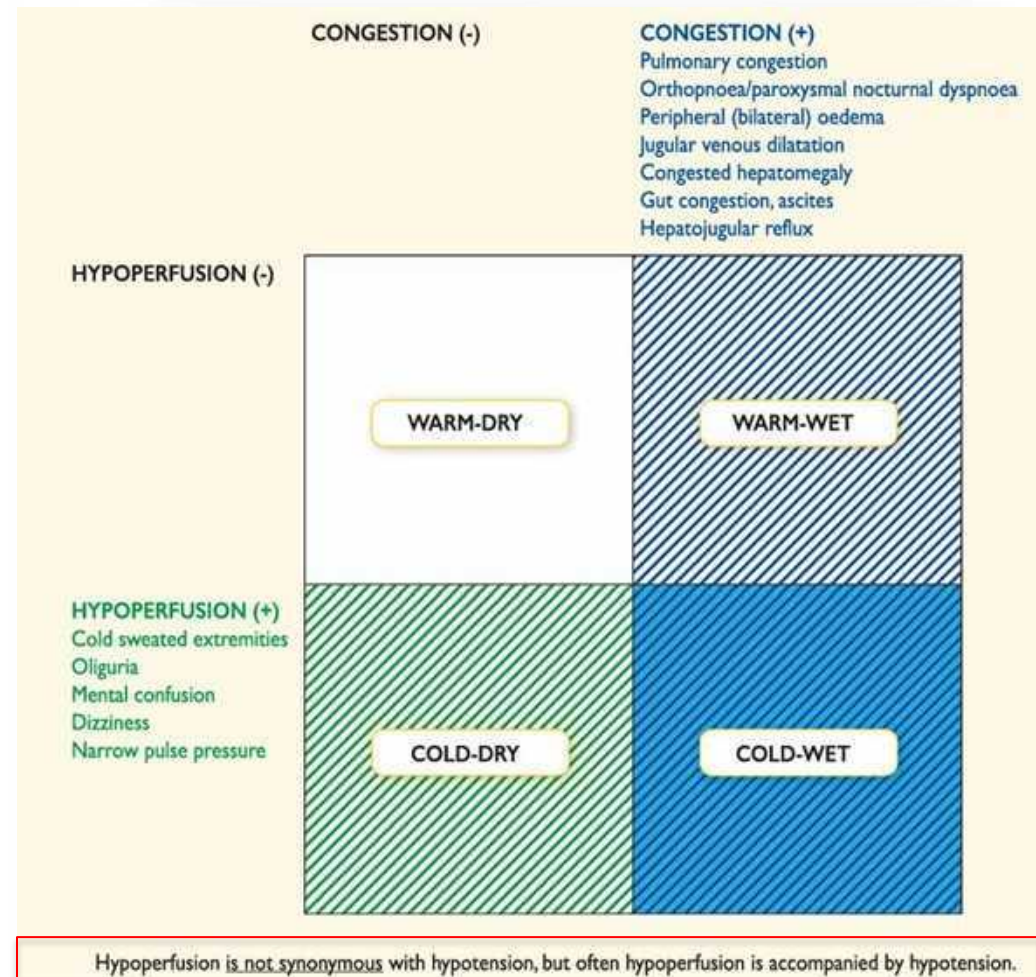
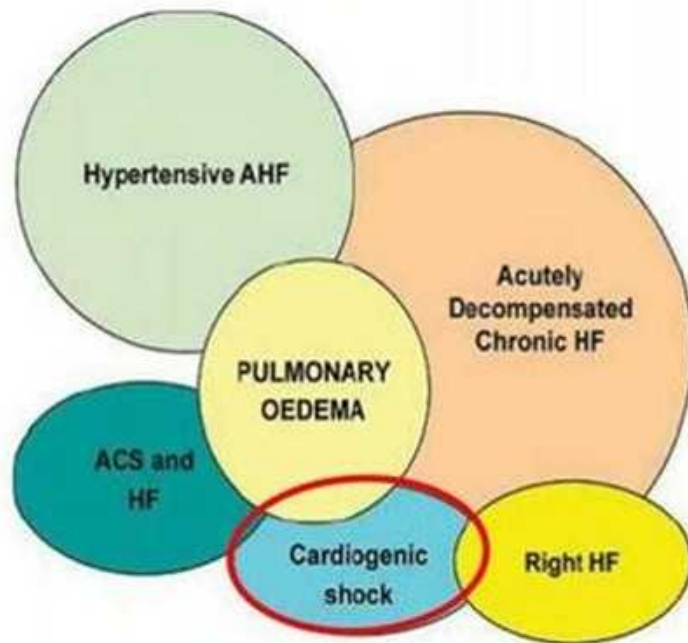


Définition « originelle » du choc cardiogénique

Baisse du débit cardiaque avec hypoxie tissulaire en présence d'une volémie normale (... ou normalisée après optimisation)

- ✓ Pression artérielle systolique < 90 mm Hg (ou nécessité de catécholamines)
- ✓ Index cardiaque $< 2,0$ L/min/m² (ou ≤ 2.2)
- ✓ Pression artérielle pulmonaire d'occlusion > 18 mmHg (15 si non ventilé) : **congestion pulmonaire**
- ✓ **SvO₂ $< 60\%$** (Sv_mO₂ ... Sv_cO₂ après optimisation volémie)
- ✓ DAVO₂ $> 5,5$ mL O₂ /dL
- ✓ **Dysfonction contraction myocardique** : FE_{VG} $< 40\%$ / **Augmentation pressions** de remplissage (PTD_{VG})
- ✓ **Hypoperfusion tissulaire** (altération conscience, extrémités $\pm$ froides, marbrures, oligurie, lactate > 2)

Acute heart failure & cardiogenic shock



Ponikowski. *European Heart J.* 2016

Définition(s) du choc cardiogénique variable(s)

Clinical Definition	SHOCK Trial ^{9*}	IABP-SHOCK II ^{††}	ESC HF Guidelines ¹⁵
Cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion	Clinical criteria: SBP <90 mmHg for ≥30 min OR Support to maintain SBP ≥90 mmHg 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 mmHg	Clinical criteria: SBP <90 mmHg for ≥30 min OR Catecholamines to maintain SBP >90 mmHg 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 mmHg 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

		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)

Définition(s) du choc cardiogénique variable(s)

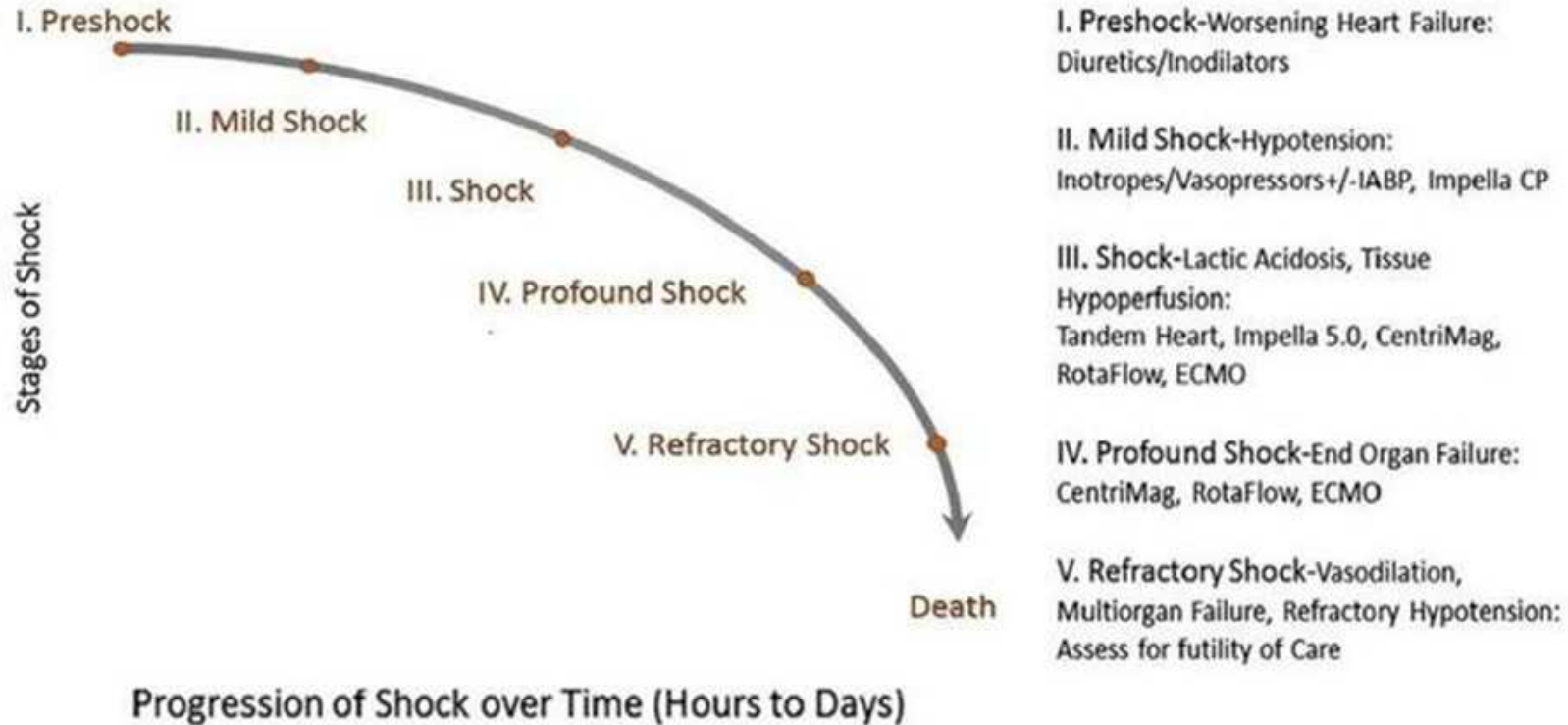
Design and preliminary results of FRENSHOCK 2016: A prospective nationwide multicentre registry on cardiogenic shock

Delmas. Archives Cardiovasc Dis 2019

Design et résultats préliminaires de l'étude FRENSHOCK 2016 : une étude nationale prospective multicentrique de cohorte sur le choc cardiogénique toute cause

Table 1 Inclusion criteria (FRENSHOCK definition of cardiogenic shock).	
Component	Criteria
Low cardiac output	SBP < 90 mmHg or need for vasopressors/inotropes to maintain SBP > 90 mmHg CI < 2.2 L/min/m ² (by echocardiography and/or invasive haemodynamic evaluation with right heart catheterization)
Right and/or left overload	Clinical assessments (dyspnoea, rales and crepitations, jugular venous distension and/or abdominojugular test, oedema) Biological tests (NT-proBNP > 900 pg/mL and/or BNP > 400 pg/mL) Radiology (overload signs on chest X-ray and/or chest tomodensitometry) Echocardiography (E/A > 2 if LVEF < 45% or E/Ea > 13 if LVEF normal; or sPAP > 35 mmHg and/or E deceleration time < 150 ms and/or Ap-Am > 30 ms and/or E/Vp ≥ 2.5) Invasive haemodynamic evaluation with right heart catheterization (PCWP > 15 mmHg and/or mPAP > 25 mmHg)
Organ malperfusion	Clinical (oliguria < 0.5 mL/kg/h, confusion, cold/clammy skin and extremities and/or marbling) Biology (lactate > 2 mmol/L, metabolic acidosis, liver insufficiency and/or renal failure)
To be considered to have cardiogenic shock, patients had to fulfil at least one criterion from each of the three components: low cardiac output; left and/or right overload; and organ malperfusion. BNP: B-type natriuretic peptide; CI: cardiac index; LVEF: left ventricular ejection fraction; mPAP: mean pulmonary arterial pressure; NT-proBNP: N-terminal prohormone of B-type natriuretic peptide; PCWP: pulmonary capillary wedge pressure; SBP: systolic blood pressure.	

Définition du choc cardiogénique = évolutive !

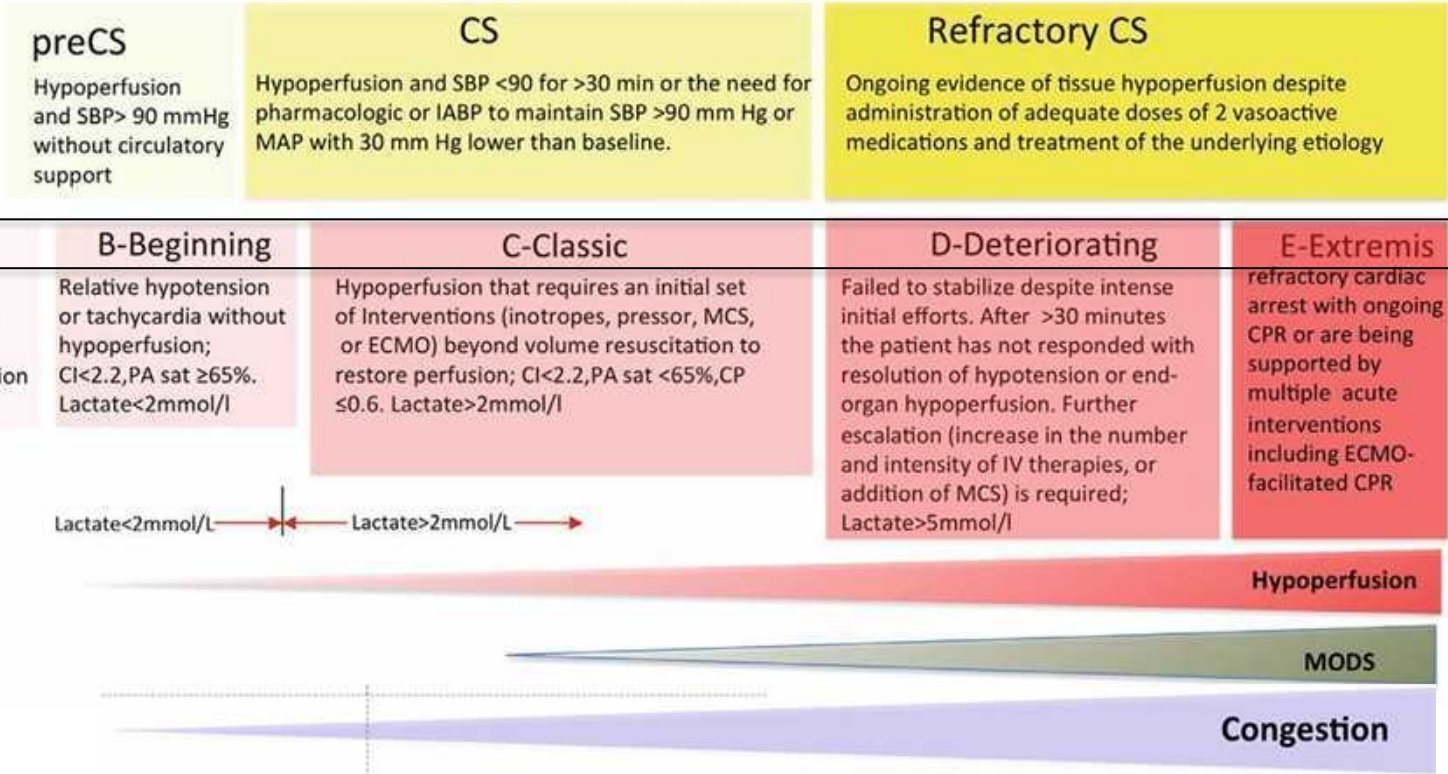


Classification du choc cardiogénique



Classification du choc cardiogénique

A Clinical classifications of CS



B Hemodynamic classification of CS

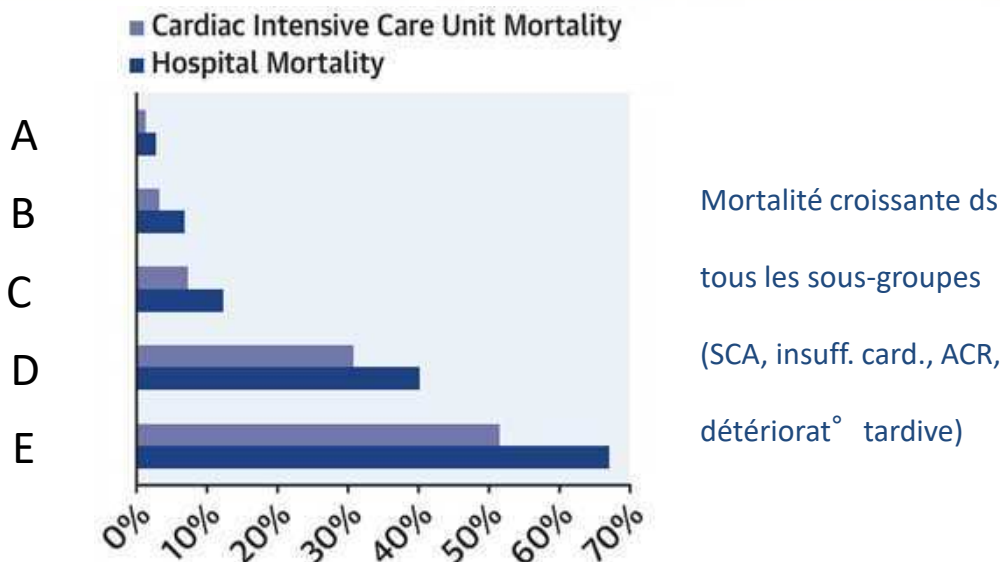
SVR ↓;PCWP ↓;CVP N ↓ "warm-dry"	SVR ↓;PCWP ↑;CVP ↑ "warm-wet"
SVR ↑;PCWP N ↓;CVP N ↓ "cold-dry"	SVR ↑;PCWP ↑;CVP ↑ "cold-wet"

Adapted from: Chioncel.
EHJ Heart Failure 2020

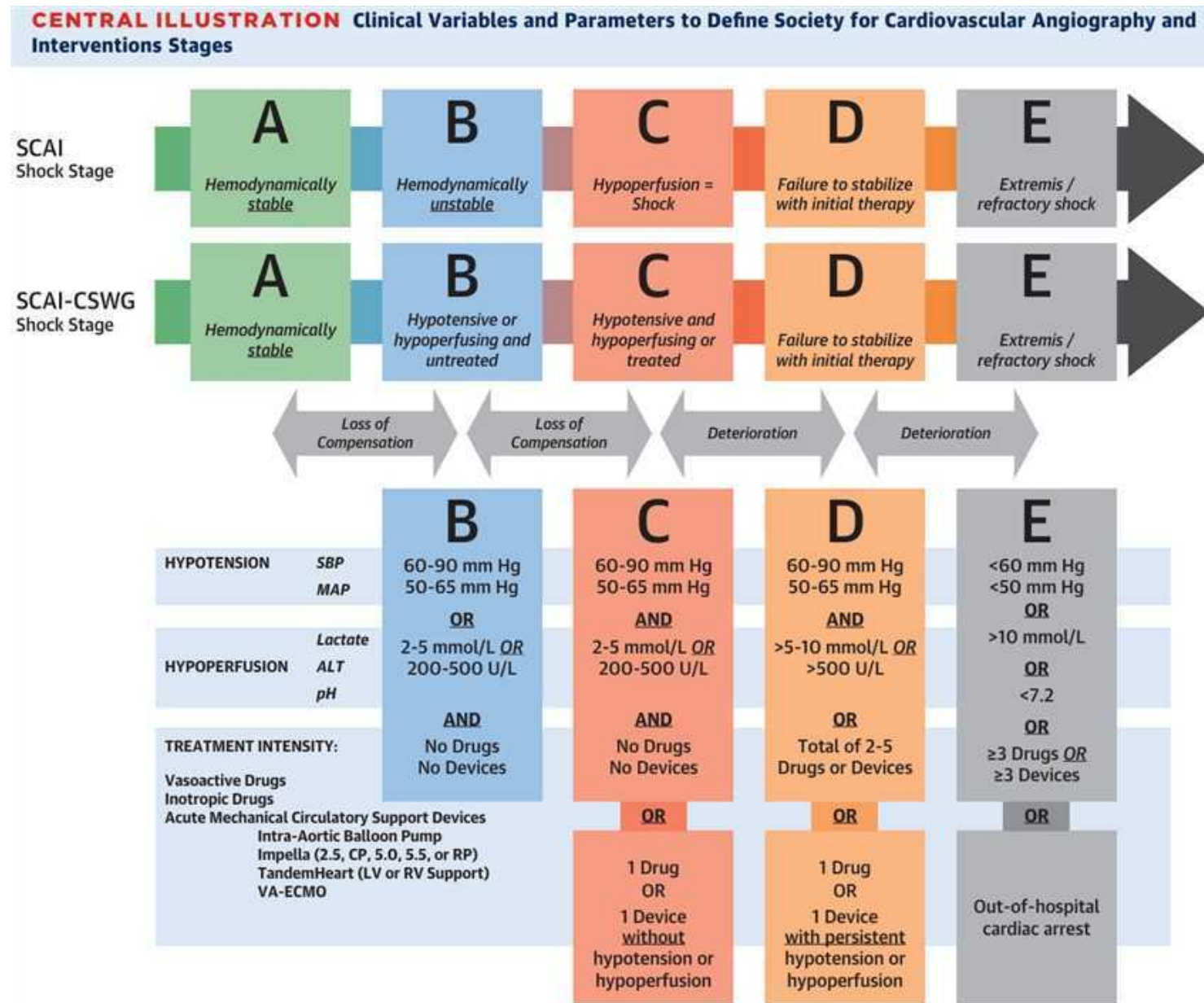
Classification du choc cardiogénique

Term	Definition
Hypotension/ tachycardia	Presence of any of the following criteria: - Admission systolic BP <90 mm Hg - Minimum systolic BP <90 mm Hg during first 1 h - Admission MAP <60 mm Hg - Minimum MAP <60 mm Hg during first 1 h - Admission HR >100 beats/min - Maximum HR >100 beats/min during first 1 h - Admission HR > admission systolic BP - Mean HR > mean systolic BP during first 1 h
Hypoperfusion	Presence of any of the following criteria: - Admission lactate >2 mmol/l - Urine output <720 ml during first 24 h - Creatinine increased by ≥0.3 mg/dl during first 24 h
Deterioration	Presence of any of the following criteria: - Maximum lactate > admission lactate - Number of vasoactives during first 24 h > number of vasoactives during first 1 h - Maximum VIS during first 24 h > VIS during first 1 h - Maximum NEE during first 24 h > NEE during first 1 h
Refractory shock	Presence of any of the following criteria: - Mean systolic BP during first 1 h <80 and on vasoactives - Mean systolic MAP during first 1 h <50 and on vasoactives - Number of vasoactives during first 1 h >2 - Number of vasoactives during first 1 h >1 and IABP during first 24 h - Admission lactate ≥10 mmol/l

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



Classification du choc cardiogénique



Tous les patients qui ont
présenté un ACR ont été
inclus dans le groupe E

Physiopathologie : il y a longtemps... le SIRS

Table 4. Logistic Regression Model for Predictors of Culture-Positive SIRS for 210 Study Patients

Predictor	OR for Culture-Positive SIRS (95% CI)	P Value
Age*	0.67 (0.45-1.00)	.047
Coronary artery bypass grafting (yes vs no)	3.01 (1.32-6.86)	.009
SVR†	1.21 (1.04-1.40)	.01

Table 5. Time-Dependent Cox Regression for 30-Day Survival by SIRS Grouping After Age Adjustment in the 297 Study Patients

Covariate*	Hazard Ratio for Mortality (95% CI)	P Value†
Overall association between survival and SIRS grouping		.008
Culture-positive vs non-SIRS control	2.22 (1.32-3.76)	.008
Culture-negative vs non-SIRS control	1.36 (0.55-3.37)	>.99
Culture-positive vs SIRS culture-negative	1.64 (0.61-4.42)	.99
Age†	1.38 (1.17-1.61)†	<.001

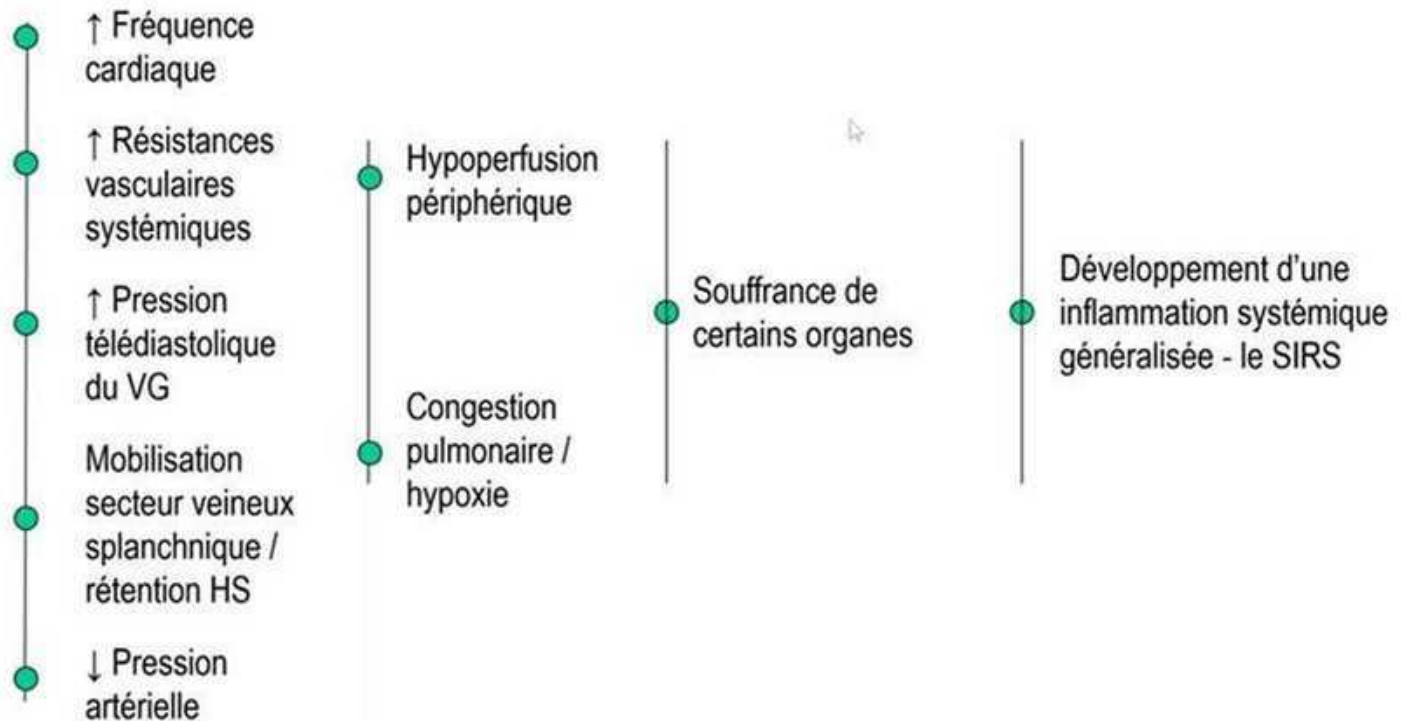
- ✓ Analyse *a posteriori* de l'étude princeps SHOCK
- ✓ Près de 20 % des patients en choc présentent un SIRS
- ✓ 75% de ces patients ont des hémocultures positives
- ✓ Association entre SIRS et mortalité à 30 jours

Physiopathologie : paradigme classique

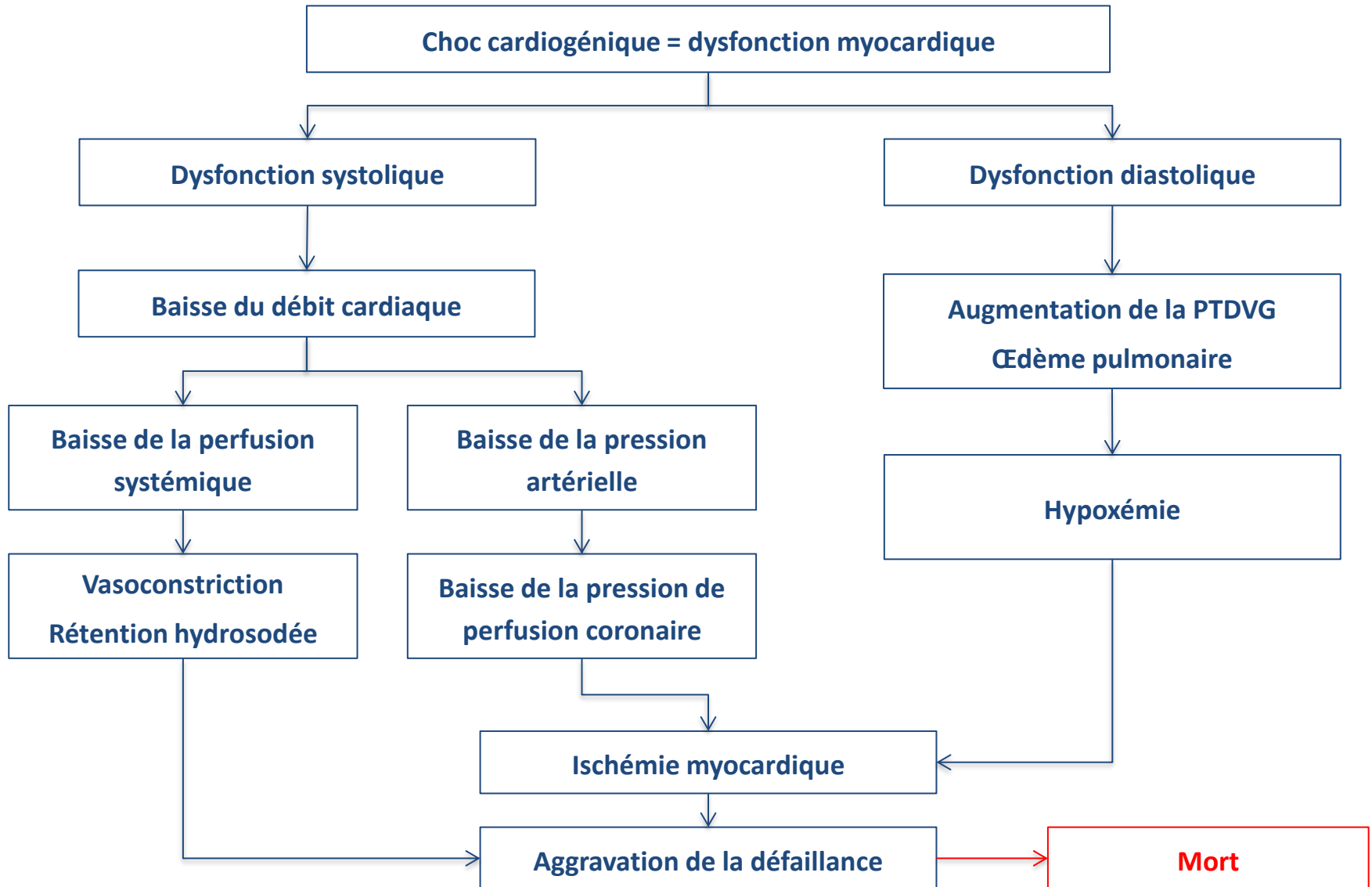
“État d'hypoperfusion critique des organes et des tissus due à un débit cardiaque réduit / inadéquat”



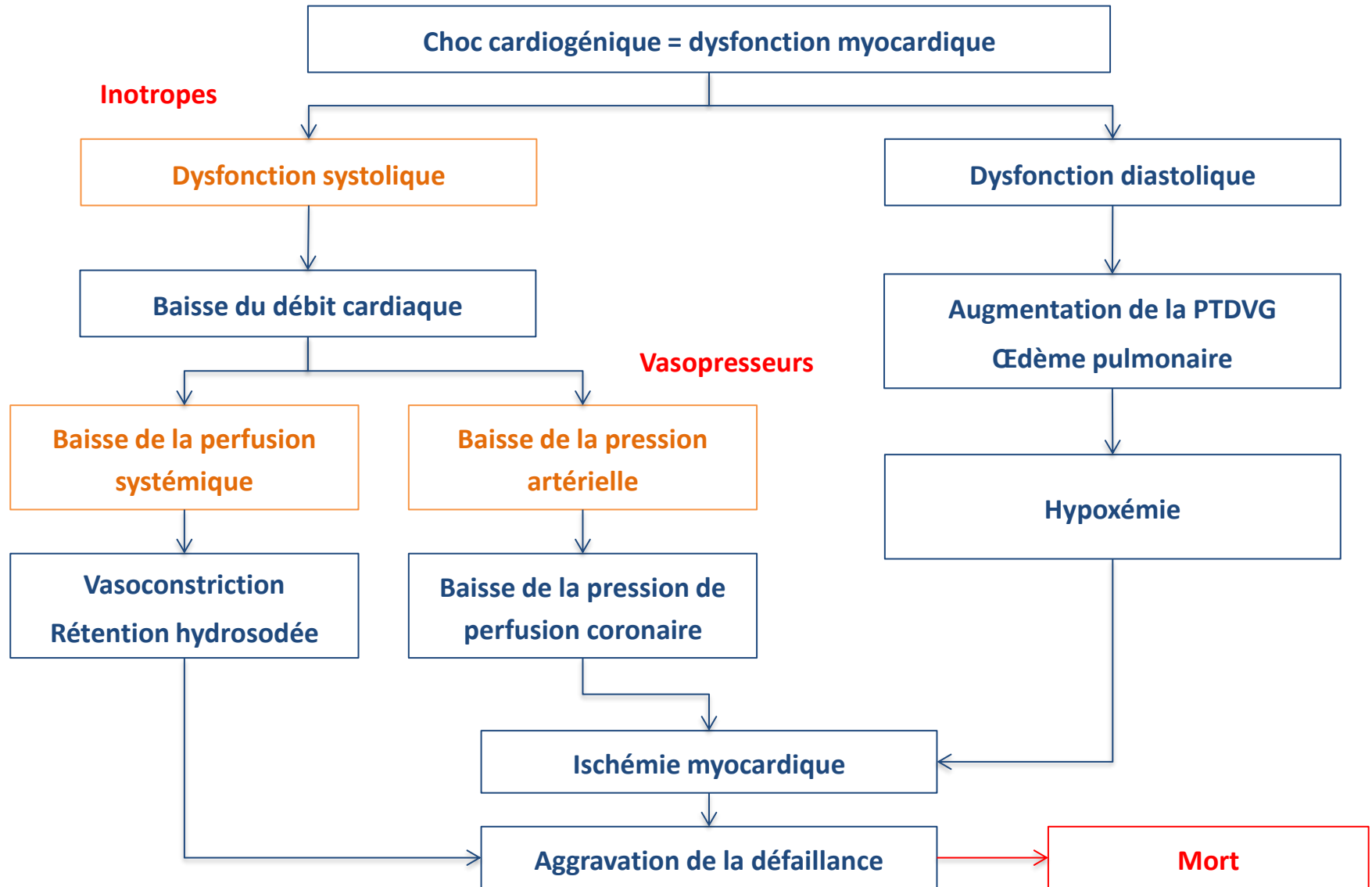
REDUCTION
CRITIQUE DU
VES et du DEBIT
CARDIAQUE



Physiopathologie : paradigme classique

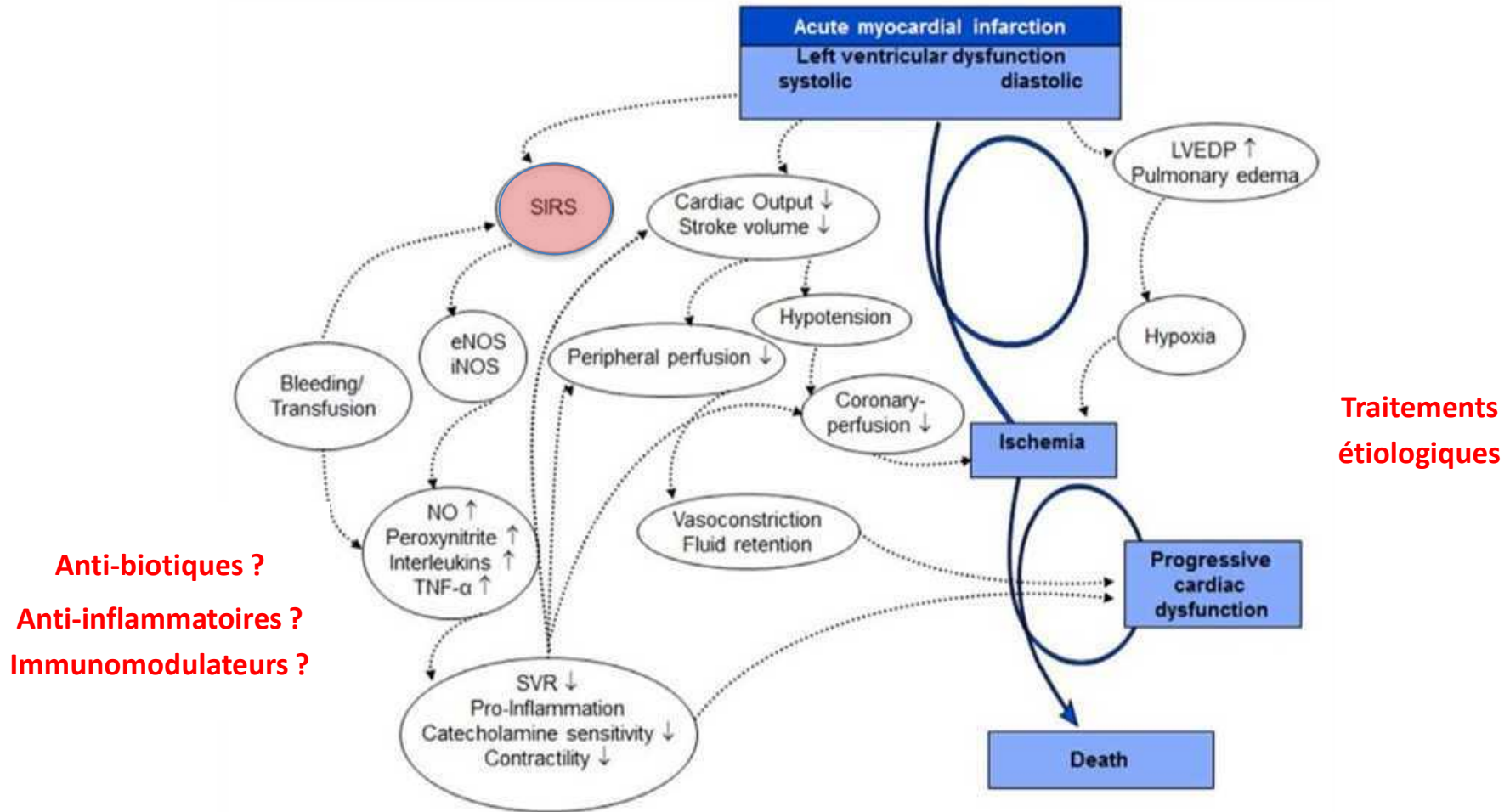


Physiopathologie : paradigme classique

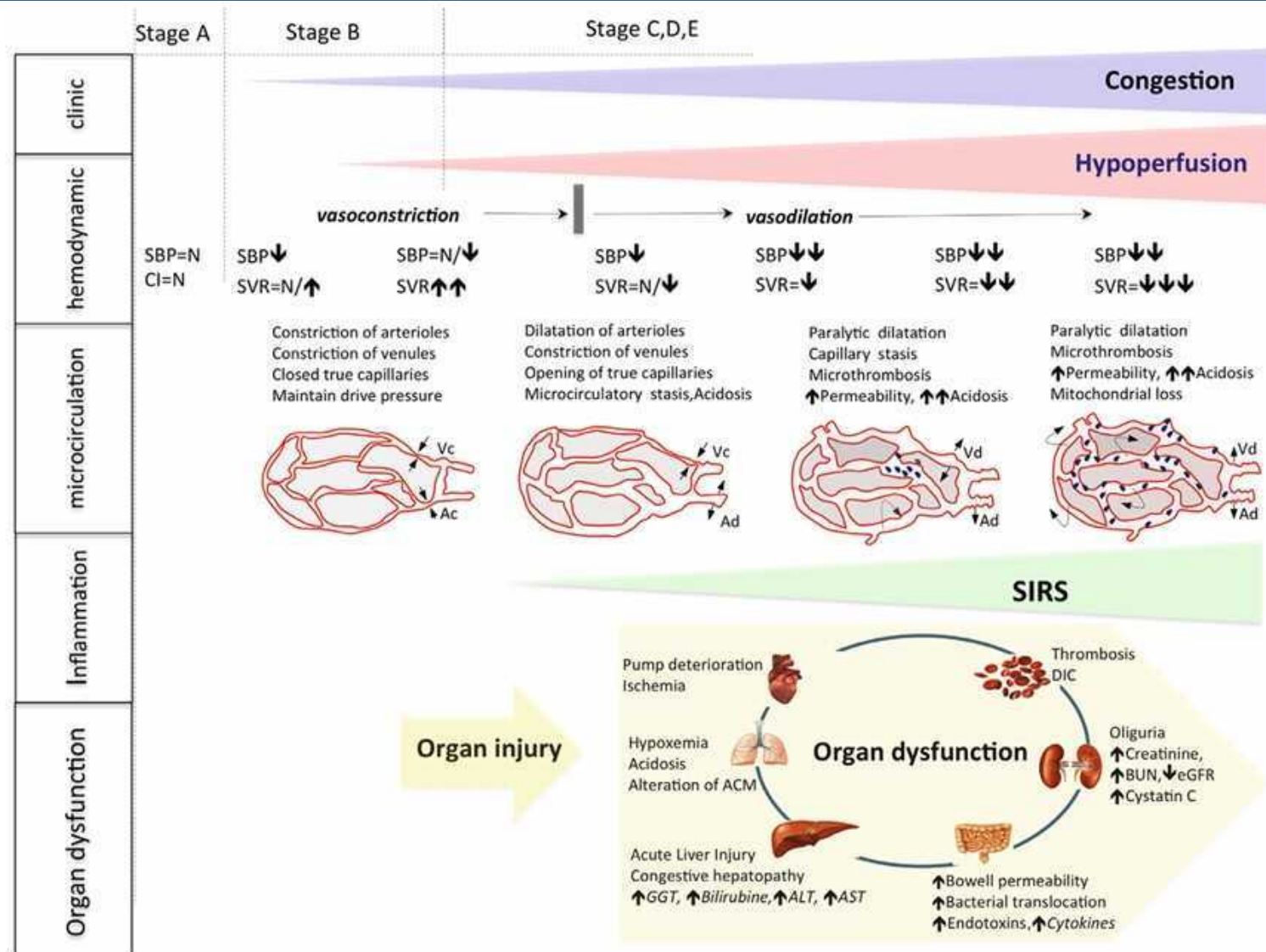


Physiopathologie : « nouveau » paradigme

Le choc cardiogénique (grave) = un choc (+) vasoplégique



Physiopathologie : « nouveau » paradigme



Les messages – choc cardiogénique



- Choc cardiogénique = défaillance hémodynamique initiale puis circulatoire globale avec une souffrance tissulaire diffuse
- Enorme réserve physiologique et activation de mécanismes d'adaptation en support et « achète » du temps pour sortir du choc
- Analyse clinique et hémodynamique situe le choc
- La réponse initiale au traitement doit être interprétée très vite
- Le passage au premier plan des aspects inflammatoires signent la perte de contrôle



Cardiogenic-Vasodilatory Shock

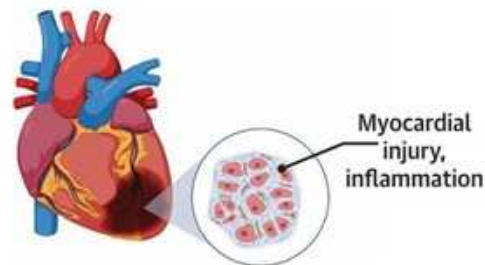
'Two-Hit'
Pathogenesis
Model

Vasodilatory-Cardiogenic Shock

'Single-Hit'
Model

Primary Mixed Shock

Etiology



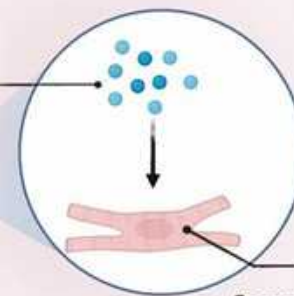
Primary Cardiac Insult

Hemodynamic Features

Prototypical Cardiogenic Shock:
Low Cardiac Index

Maladaptive Reduction in SVR

Trigger of proinflammatory cascade with microvascular dysfunction, oxidative stress, and abnormal mitochondrial oxygen utilization



Primary Vasodilatory Insult

Primary Microvascular and Systemic Vasodilation

Myocardial Depression:
Low Cardiac Index

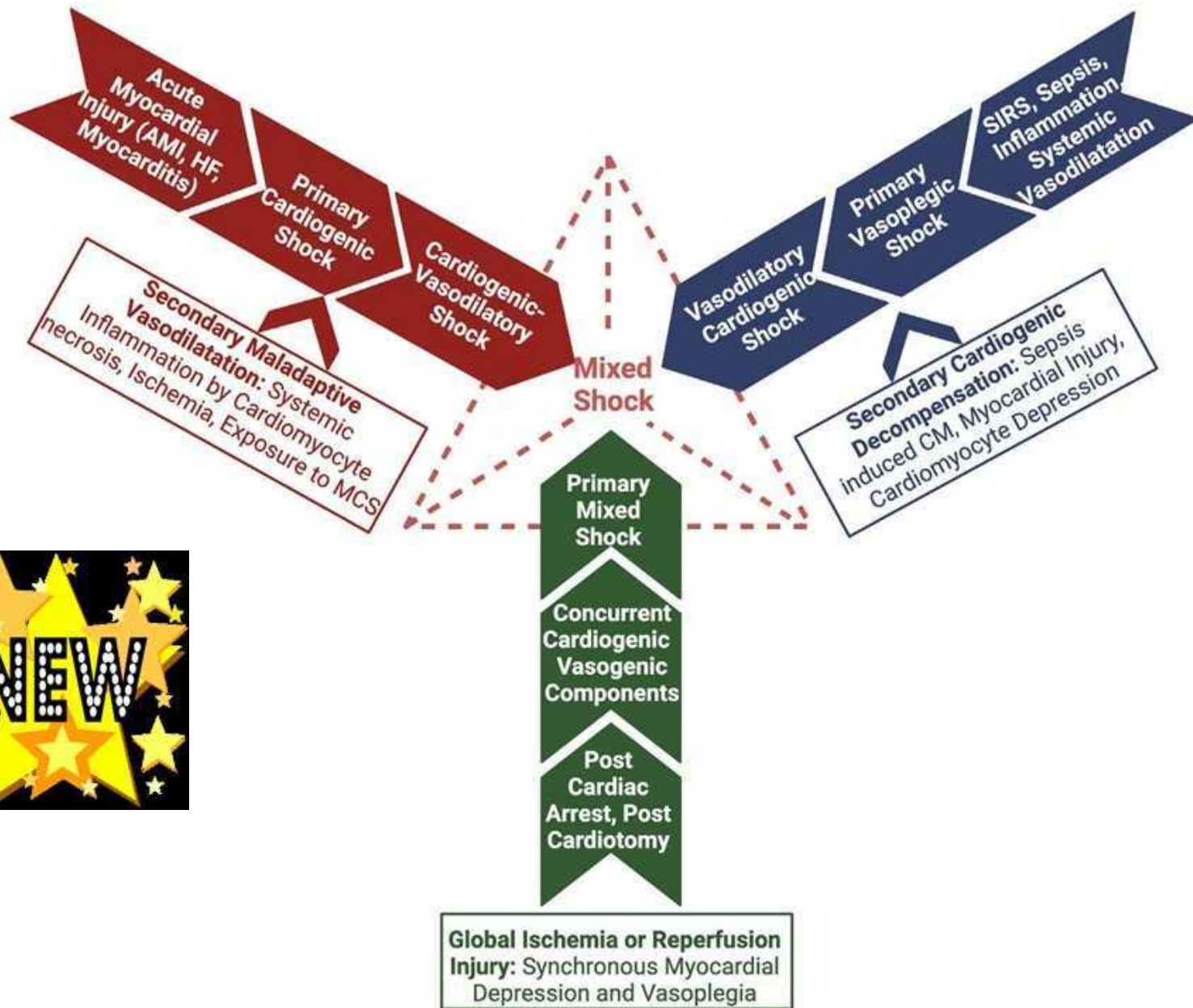
Post-cardiotomy;
Post-cardiac arrest syndrome

Ischemia reperfusion injury

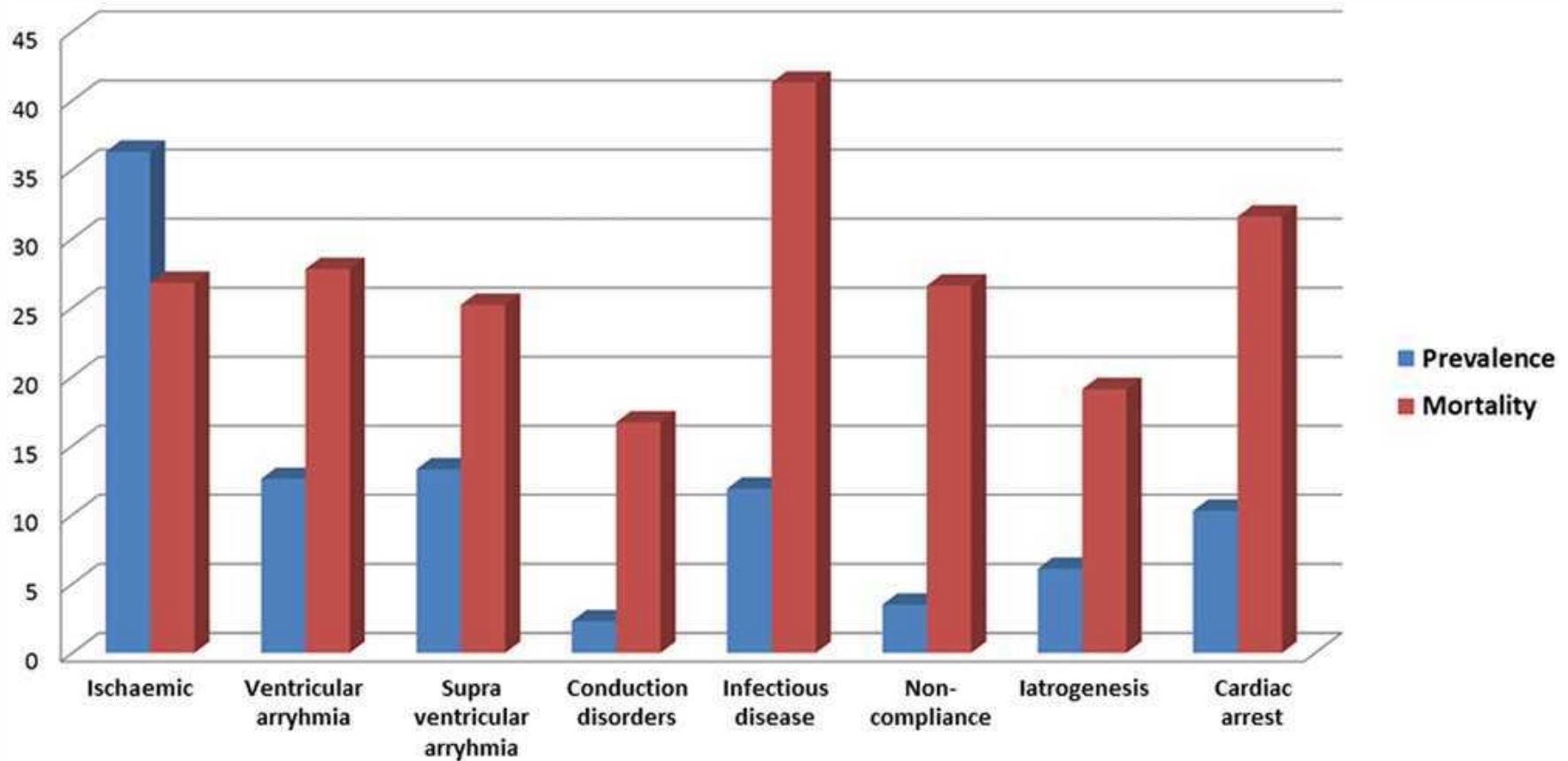
Simultaneous or Rapid Sequential Cardiac Dysfunction and Vasoplegia

Primary Insult Triggering Low Cardiac Output and Vasoplegia

FIGURE 1 Proposed Classification of Mixed Shock



Facteurs de gravité = grand danger (FRENSHOCK)



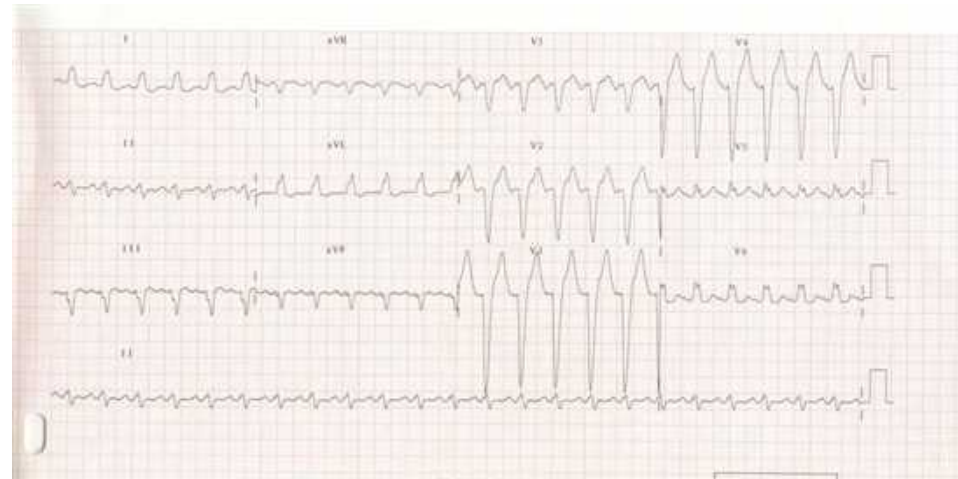
- ✓ 48 centres ; 772 patients
- ✓ Critères hémodynamiques + surcharge droite et/ou gauche + « malperfusion »
- ✓ Choc cardiogénique d'origine ischémique : 36,3% ; non-ischémique 63,7% (jusqu'à 3 « triggers » / patient)
- ✓ Nécessité d'un support d'organe chez 58,3% des patients

Facteurs de gravité = grand danger (SCA)

Dans 2/3 des cas, le choc n'est pas présent à l'admission et survient dans les 48 premières heures après admission pour IDM.

Facteurs prédictifs de survenue d'un choc cardiogénique d'origine ischémique :

- ✓ Age
- ✓ Fréquence cardiaque > 75 bpm (admission)
- ✓ Diabète
- ✓ ATCD d'IDM et / ou de pontages coronaires
- ✓ Présence de signes d'insuffisance cardiaque (admission)
- ✓ Nécrose localisée en antérieur



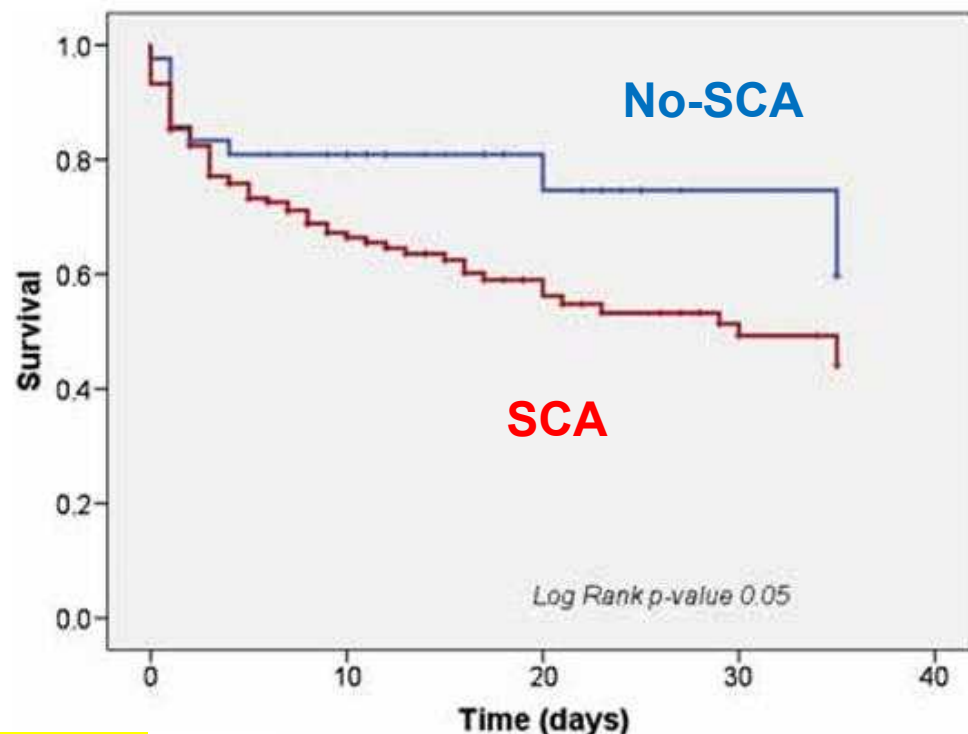
Facteurs de gravité = grand danger (SCA)

Clinical picture and risk prediction of short-term mortality in cardiogenic shock

Veli-Pekka Harjola^{1*,†}, Johan Lassus^{2†}, Alessandro Sionis³, Lars Køber⁴,
Tuukka Tarvasmäki⁵, Jindrich Spinar⁶, John Parissis⁷, Marek Banaszewski⁸,
Jose Silva-Cardoso⁹, Valentina Carubelli¹⁰, Salvatore Di Somma¹¹, Heli Tolppanen²,
Uwe Zeymer¹², Holger Thiele¹³, Markku S Nieminen², and Alexandre Mebazaa¹⁴,
for the CardShock study investigators and the GREAT network

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



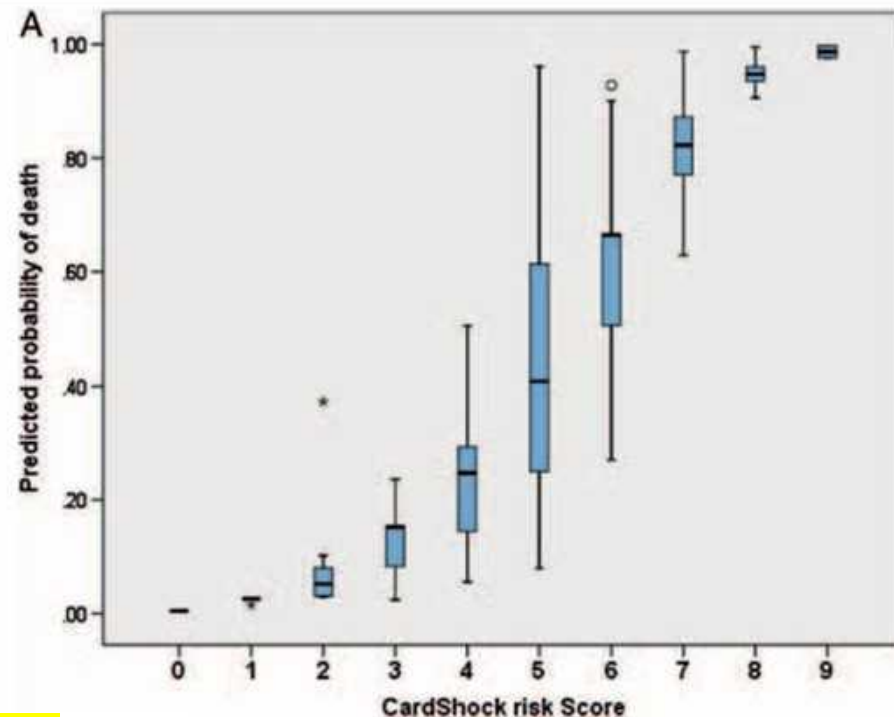
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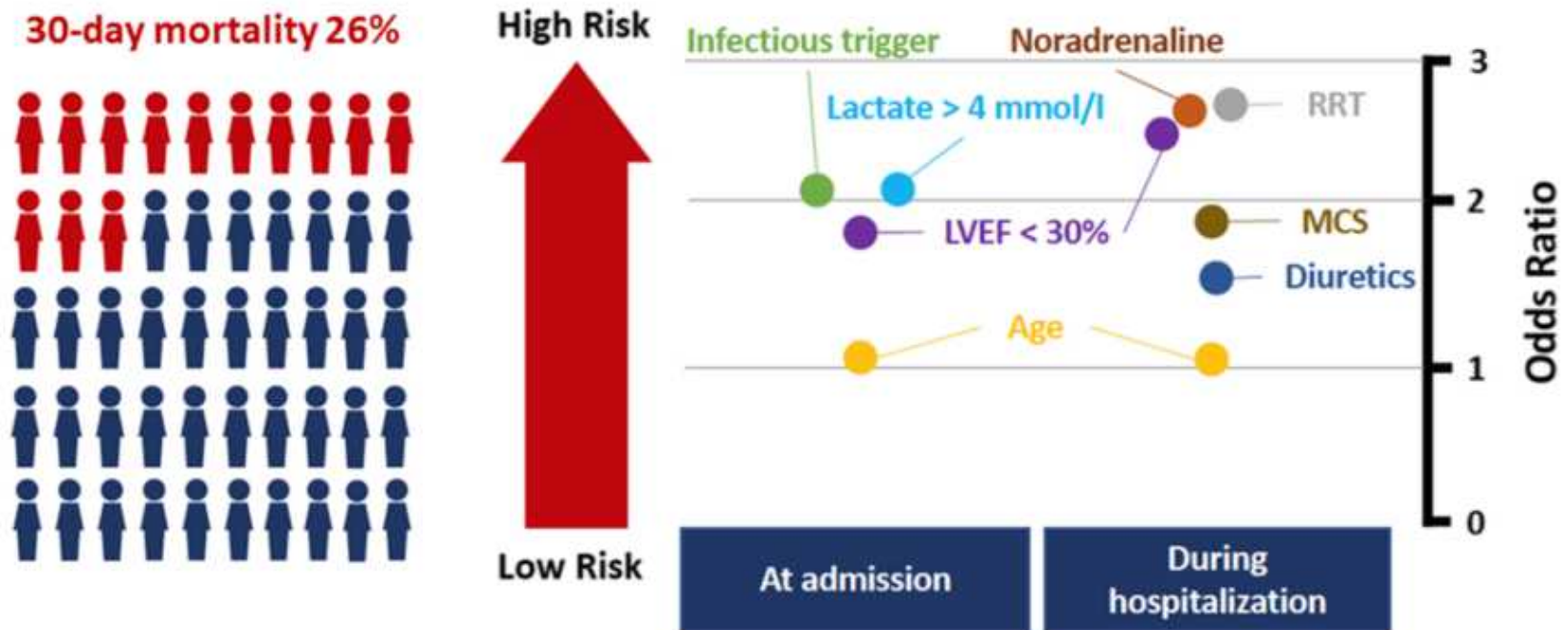
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Table 4 The CardShock risk Score for risk prediction of in-hospital mortality in cardiogenic shock

Variable	CardShock risk Score
Age >75 years	1
Confusion at presentation	1
Previous MI or CABG	1
ACS aetiology	1
LVEF <40%	1
Blood lactate	
<2 mmol/L	0
2–4 mmol/L	1
>4 mmol/L	2
eGFR _{CKD-EPI}	
>60 mL/min/1.73 m ²	0
30–60 mL/min/1.73 m ²	1
<30 mL/min/1.73 m ²	2
Maximum points	9



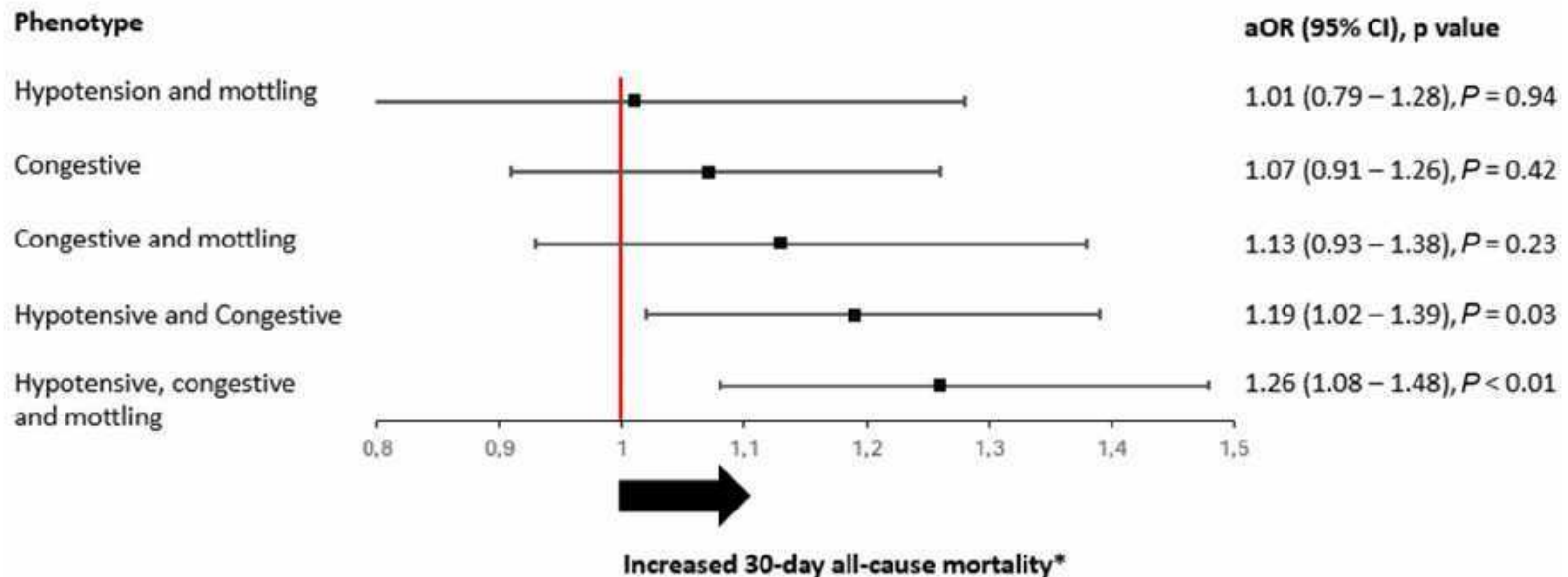
Facteurs de gravité = grand danger (FRENSHOCK)



- ✓ 48 centres ; 772 patients
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Clinical phenotyping of cardiogenic shock at a glance: A rapid, costless, streamlined approach

	Normotensive		Hypotensive	
Non-congestive	NA N = 0	NA N = 0	8.8% N = 3/34	11.8% N = 2/17
Congestive	16.2% N = 16/99	21.2% N = 7/33	26.5% N = 43/162	32.3% N = 42/130
	No mottling	Mottling	No mottling	Mottling



*The reference group was that with hypotension without congestion or mottling. Analysis were adjusted for age, sex, history of cardiomyopathy and ischaemic CS trigger.

Diagnostic du choc cardiogénique

Definition

SBP < 90 mmHg

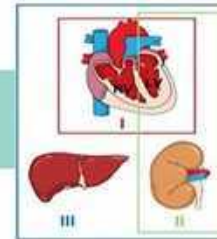
End-organ hypoperfusion: cold extremities, confusion, oliguria < 30 mL/h

Lactate > 2 mmol/L

CI < 2.2 L/min/m²

Phenotyping

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



Identify cause of CS

Acute myocardial infarction

Cardiac arrest

Ventricular / Supraventricular arrhythmia

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

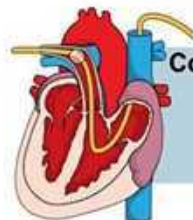


Considering invasive hemodynamic monitoring

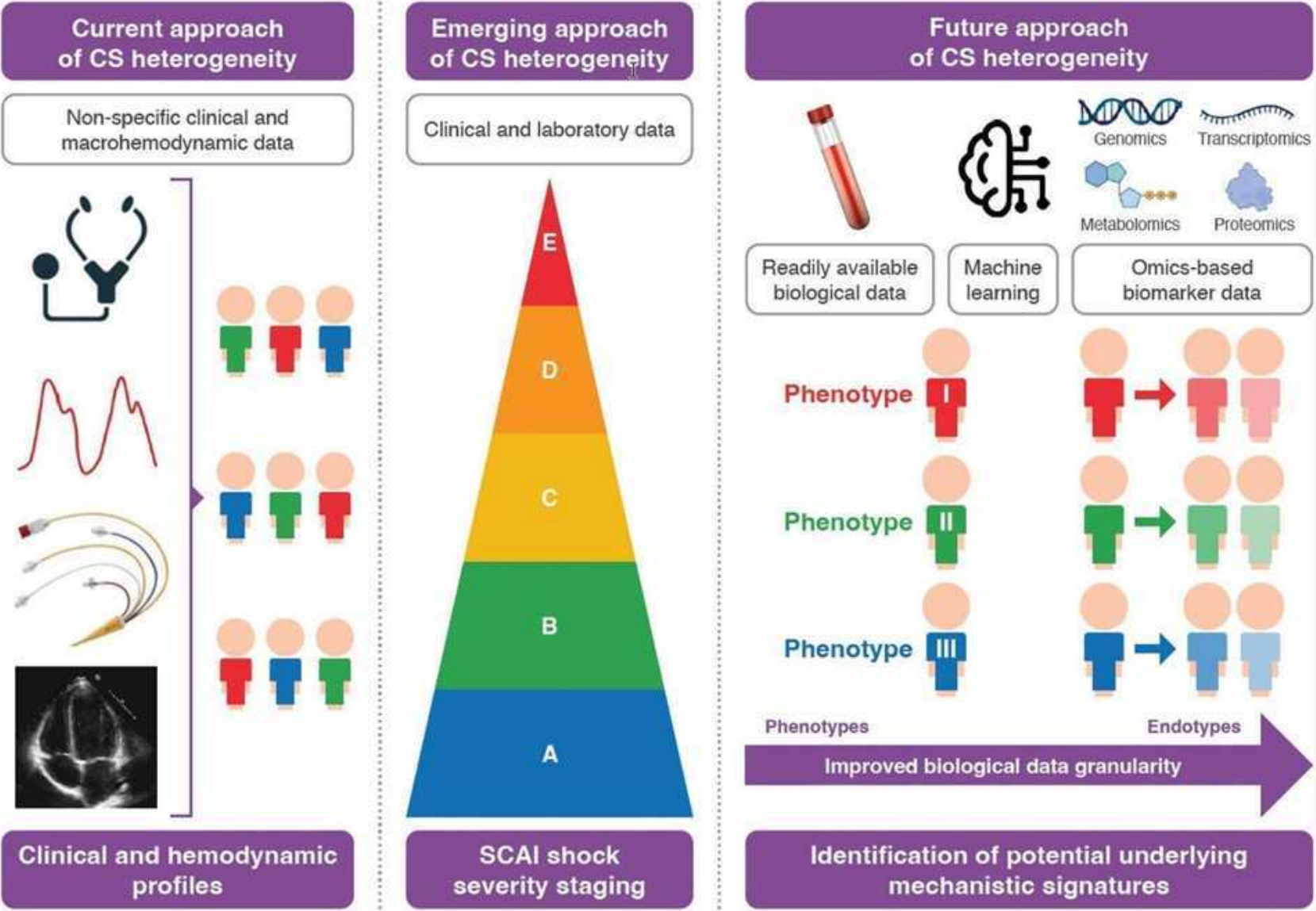
Arterial catheter

Transpulmonary thermodilution

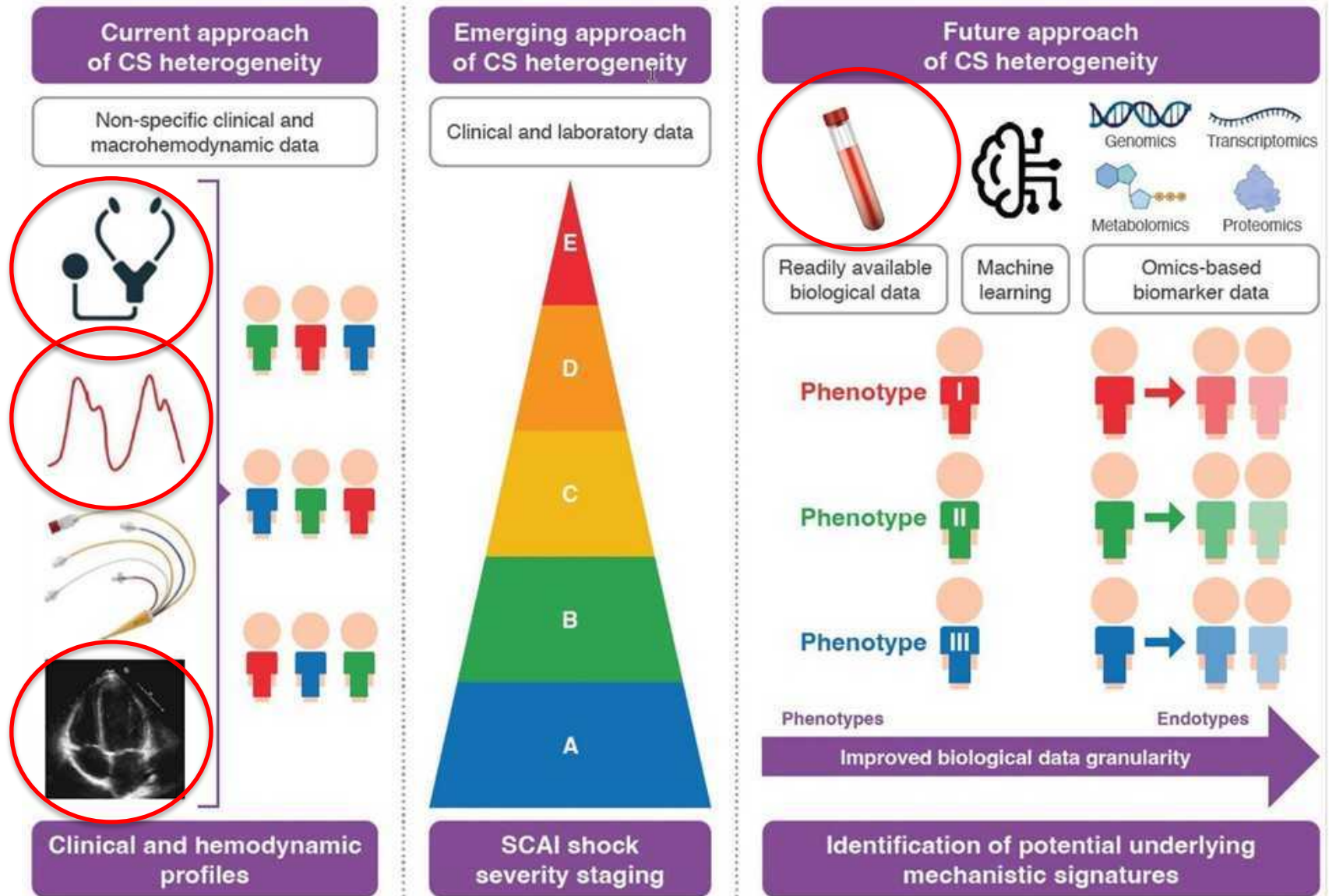
Discussed pulmonary artery catheterization



Diagnostic du choc cardiogénique



Outils du choc cardiogénique (diagnostic/pronostic)



Outils du choc cardiogénique (diagnostic/pronostic)

Identifying biomarker-driven subphenotypes of cardiogenic shock: analysis of prospective cohorts and randomized controlled trials

Sabri. *Lancet* 2025

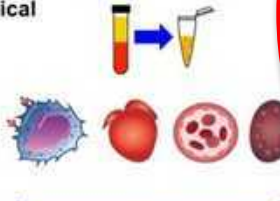
Identification of biomarker-driven subphenotypes of cardiogenic shock

Molecular characterization of the identified subphenotypes

Evaluation of subphenotypes association with outcome

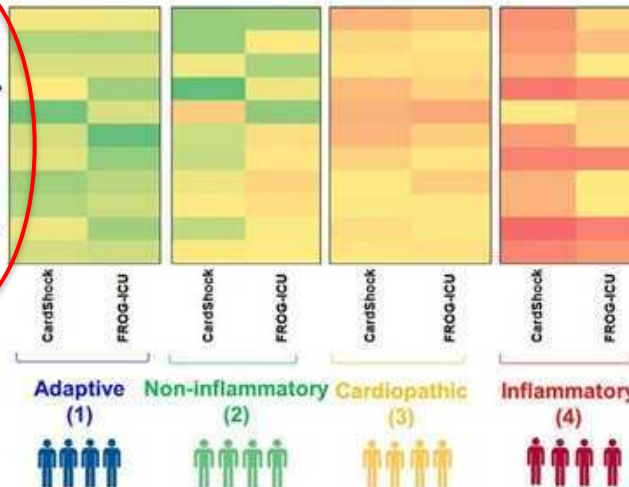
Observational prospective cohorts (CardShock & FROG-ICU; N=433)

Cardiogenic shock as a heterogeneous clinical syndrome

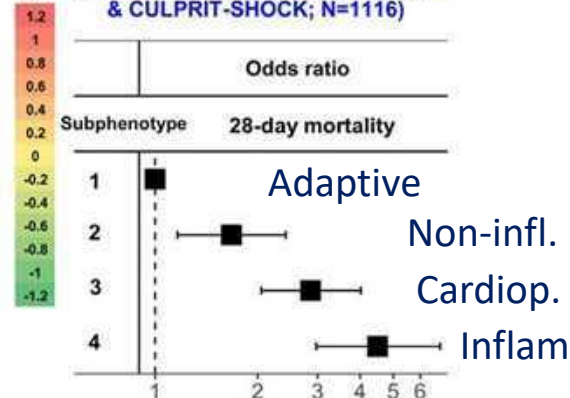


Organ injury and host-response biomarkers

CRP
PCT
IL-6
NT-proBNP
Hs-cTnI
ST2
Bio-ADM
Lactate
DPP3
Cystatin C
NGAL



Observational prospective cohorts & clinical trials (OptimaCC, DOREMI & CULPRIT-SHOCK; N=1116)



Inflammation : CRP, PCT, IL6
Autres : ALAT, plaquettes

Rénal : créatinine, NGAL,
cystatine C

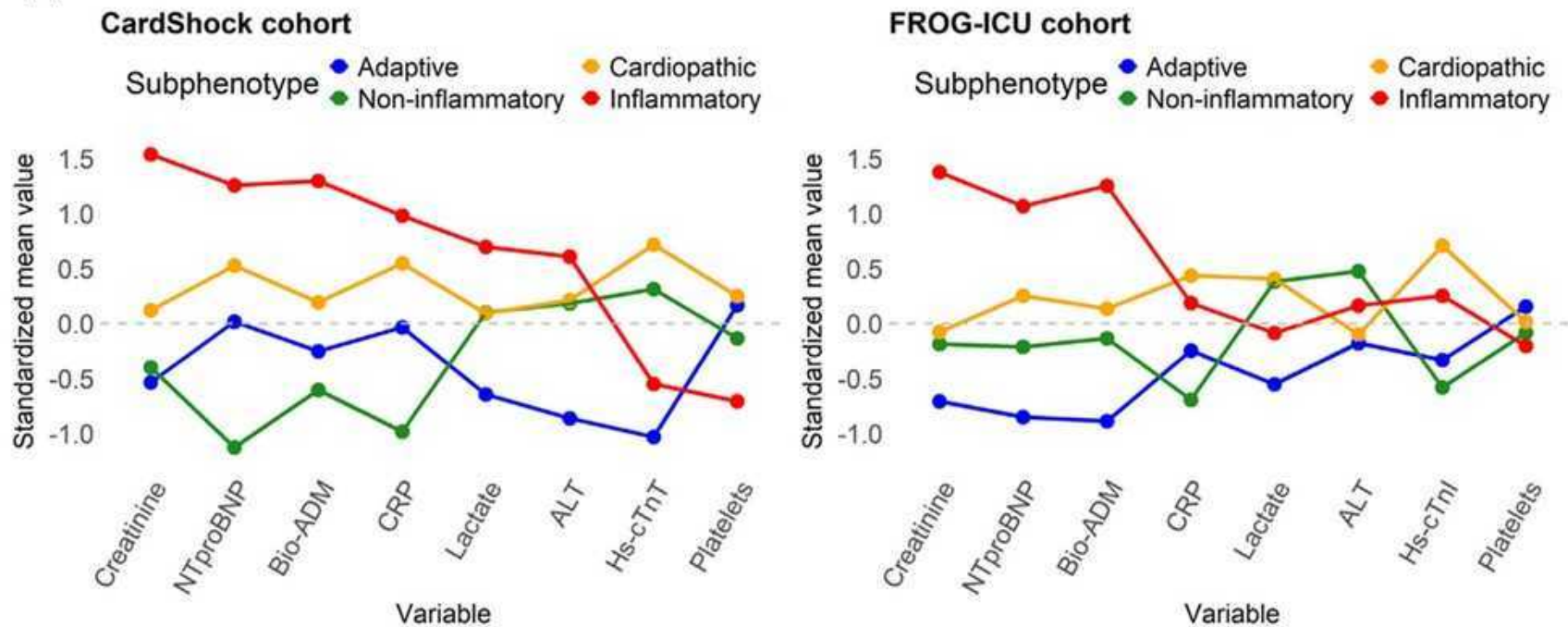
Dysfonction/lésion CV :
NT-proBNP, cTnI HS, ST2,
bio-ADM, Lactate, DPP3

Outils du choc cardiogénique

Identifying biomarker-driven subphenotypes of cardiogenic shock: analysis of prospective cohorts and randomized controlled trials

Sabri. *Lancet* 2025

A



Interpretation Subphenotypes with the highest concentration of biomarkers of endothelial dysfunction and inflammation (inflammatory) or myocardial injury/fibrosis (cardiopathic) were associated with mortality independently from the SCAI shock stages.

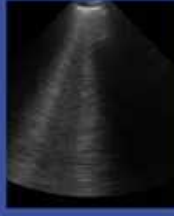
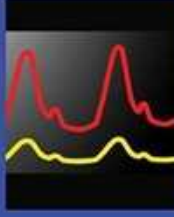
How we use critical care ultrasonography in the management of cardiogenic shock: a strategic game of chess in intensive care

The queen: “critical care echocardiography”



San Filippo et al. ICM 2025

- Grande valeur prédictive pour la contraction myocardique (FE) et le débit cardiaque Qc (= inverse des résistances !).
- Bonne VPN pour la dysfonction diastolique (évaluation des pressions de remplissage)
- Diagnostic étiologique +++

CHES PIECE	MONITORING TOOLS	USEFULNESS IN CARDIOGENIC SHOCK LIMITATIONS IN CARDIOGENIC SHOCK
		CCE is first-line imaging modality for diagnosis of CS; Preferred modality to assess for complications (i.e. valve dysfunction); Unique for monitoring response to therapy; Essential for escalation to MCS, Cath-lab or cardiac surgery; Estimation of LVFP; Assessment of interventricular and heart-lung interaction Monitoring is not continuous; Filling pressures are estimated; Advanced CCE is not always available; Education in CCE is currently a challenge
		LUS permits the rapid identification and quantification of pulmonary edema; Monitoring response to therapy; Complements the CCE in assessing LVFP It is not diagnostic for the etiology of CS; does not address the genesis of pulmonary edema
		Pulse contour analysis: Allows continuous blood pressure monitoring; can track CO changes in response to interventions; may be used to test FR PAC: Gold standard for measuring CO; allows direct measurement of PAP and LVFP; differentiates pre- and post-capillary pulmonary hypertension Both: not diagnostic of CS etiology of shock; Not feasible as first-line approach; Variations in use among centers PAC: requires greater knowledge and practice; proficiency and utilization have decreased over the years.; intermittent CO monitoring (most cases), unable to assess FR
		Venous congestion allows to identify congestive phenotypes; Quantification of maladaptive consequences of congestion; Monitoring of decongestion in case of off-loading strategy Validation studies still lacking; Not established role in general intensive care; Low inter-rater reproducibility; Difficult to master
		Telemedicine and AI might allow provision of distant support in remote centers with image transfer and case discussion; Identification of phenotypes and early prognostication Not yet established role; Sensible data sharing; Need of adequate data quality.

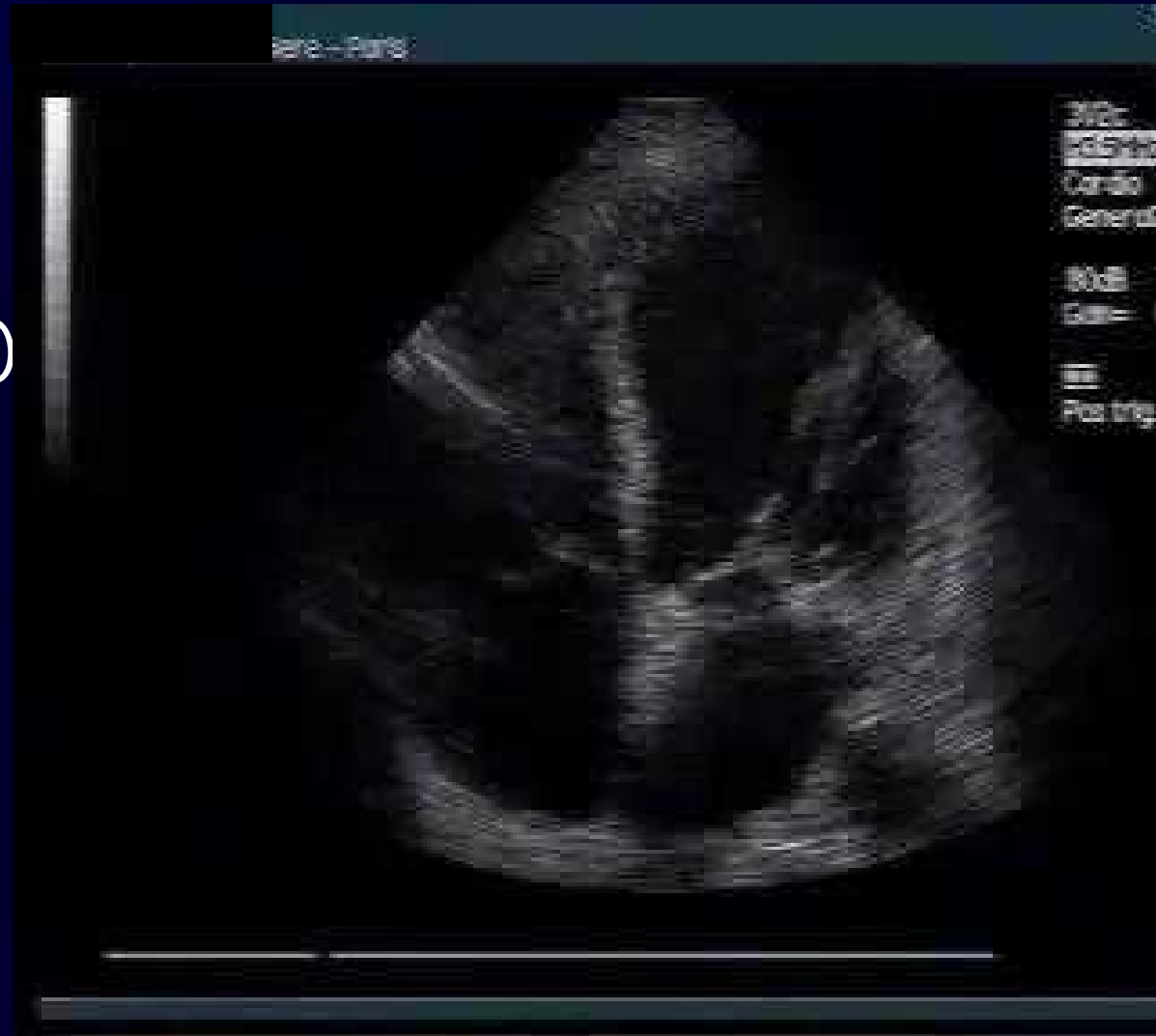
CAS 1 : diagnostic ?

Femme 48 ans

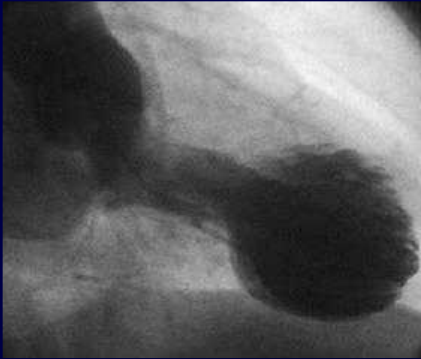
Surdosage aux bêta-bloquants et AA2 +
ulcère duodénal (stress)

Survenue d'un choc
cardiogénique à QRS
fins et QT normal

Qc 2,2 l/min
PTDVG élevée
NA 3, Adré 10 mg/h



ASPECT ÉCHOCARDIOGRAPHIQUE COMPATIBLE AVEC UN TAKO-TSUBO



4 critères de cardiomyopathie de Tako-Tsubo :

- **Akinésie ou dyskésie apicale** $\pm$ moyenne transitoire du VG avec anomalies régionales de mobilité myocardique non systématisés. Normokinésie des autres segments.
- **Absence de coronaropathie** aiguë ou de signe angiographique en faveur d'une rupture aiguë de plaque d'athérome.
- **Anomalies récentes de l'ECG** : élévation du segment ST et/ou inversion des ondes T en précordial
- **Absence de traumatisme crânien grave** récent, hémorragie intracrânienne, phéochromocytome, **coronaropathie** évoluée, **myocardite**, cardiomyopathie hypertrophique

Aspect échocardiographique de Tako-Tsubo

Age ans	IGS II	Etiologie	IS max mg/h	Adr ^e max mg/h	NA max mg/h	Qc l/min	CEC	Tako inversé (vs nl)	FE _{VG} (%)	Décès
37	41	Hémorragie	397	10	5	2	Oui	Oui	20	Non
64	36	I. Rénale A.	0	0	0	2.5	Non	Non	30	Non
75	54	AC	255	3	4	3.1	Non	Non	38	Non
55	48	Intoxicat ^o	572	10	3	1.2	Oui	Non	15	Non
42	60	AC	227	5	7.5	6	Non	Oui	50	Oui

Score inotropique de Chen :

IS ($\mu\text{g/kg/min}$) = dobutamine + dopamine + 100xAdr^e + 100xNoradr^e

ECLS si choc persistant et IS > 60 $\mu\text{g/kg/min}$???

Majoration des catécho (inotropes) !!!

Chen CCM 2006

CMP de stress dans les suites d'une forte imprégnation en catécholamines pour choc hémorragique en péri-partum

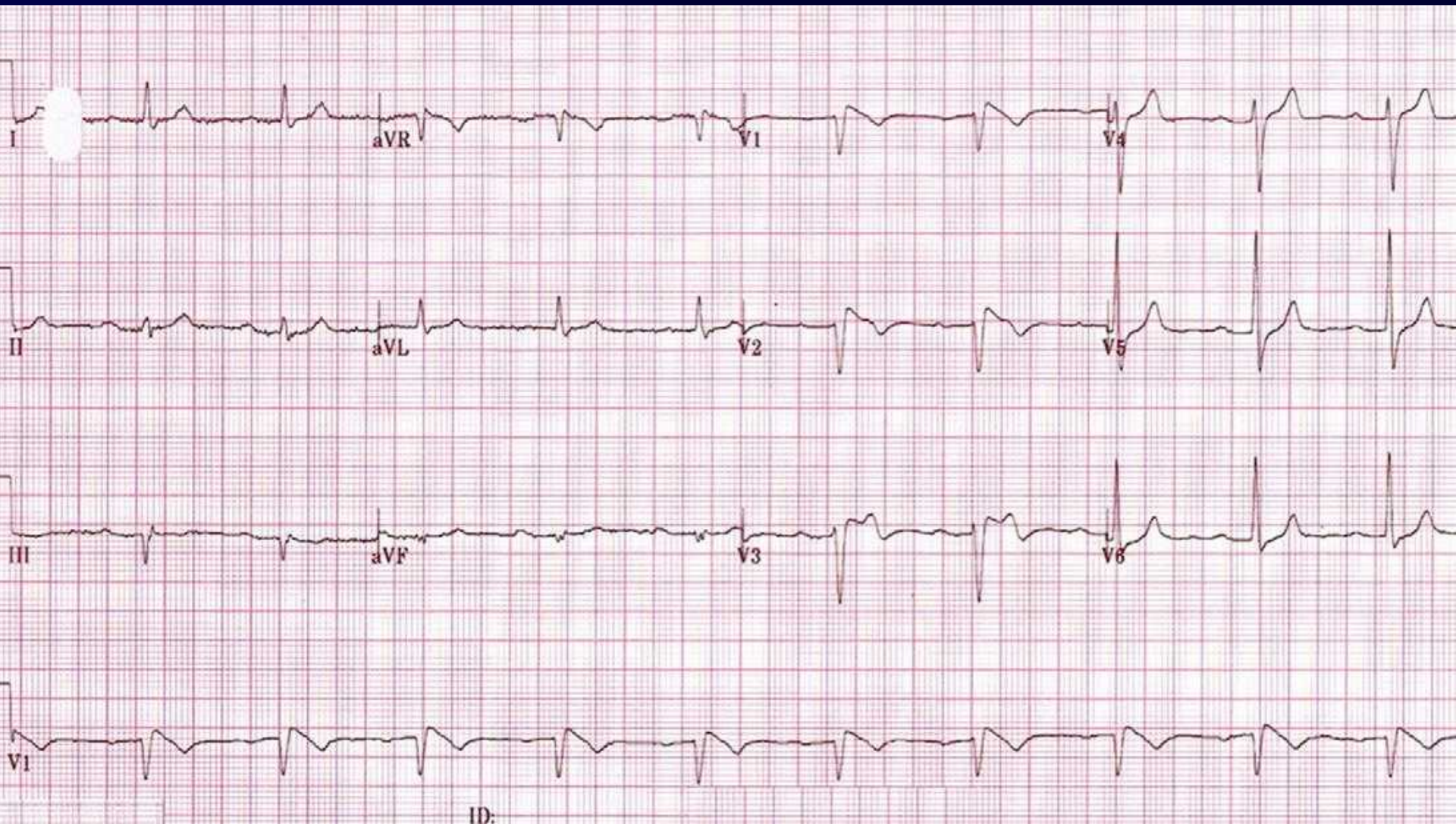


Takotsubo inversé ou reverse
(attention à Takotsubo médian vs myocardite segmentaire)

FE_{VG} 20 %, $Q_c \approx 2.2$ L/min, AK globale mais hyperkinésie apicale, épanchement pleuro-péricardique non compressif, VD non dilaté.

CAS 2 : diagnostic ECG ?

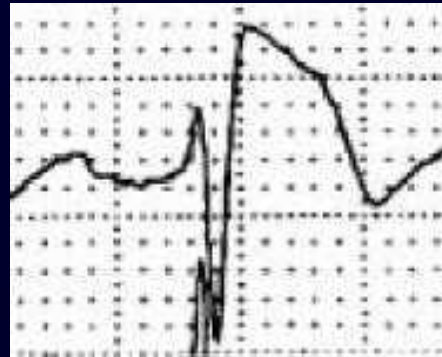
Hospitalisation pour coma convulsif (traitement au long cours par lithium). Survenue secondaire d'un choc cardiogénique.



ASPECT ÉLECTROCARDIOGRAPHIQUE COMPATIBLE AVEC UN SYNDROME DE BRUGADA DE TYPE 1 (par blocage des canaux sodiques : effet stabilisant de membrane)

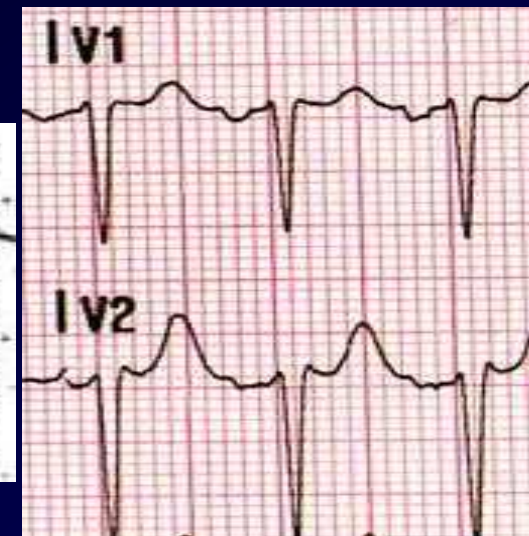
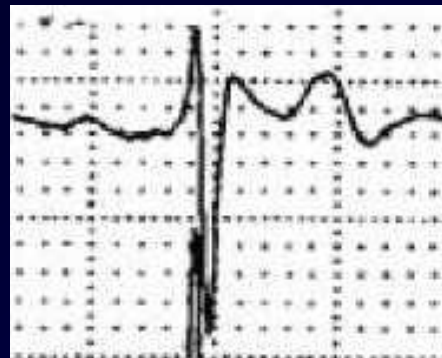
SDB type 1

élévation du point J en V1 $\geq 2\text{mm}$
onde T-
ST en dôme (« coved »)
(BBID)



SDB type 2 ou 3

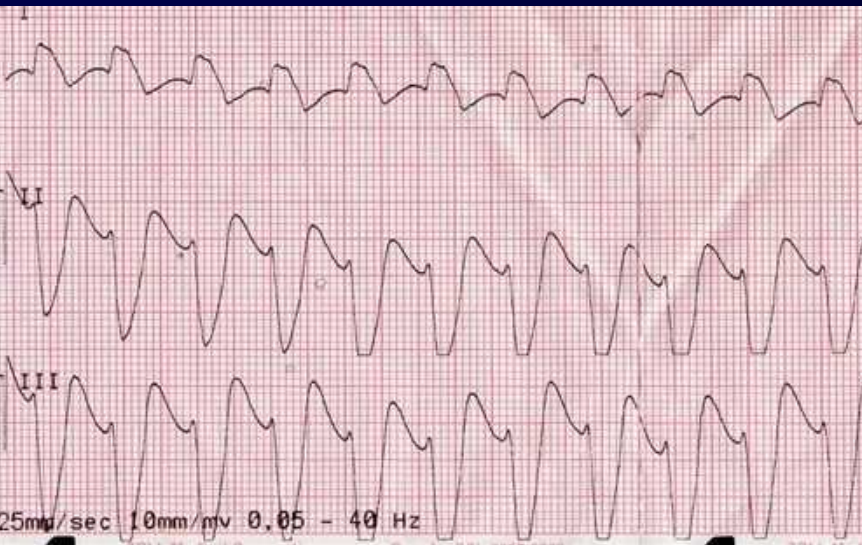
élévation du point J en V1 $\geq 2\text{mm}$
onde T+
ST en selle (« saddleback »)
($\pm$ sus-ST)



CHOCs CARDIOGÉNIQUES RÉFRACTAIRES TOXIQUES

EN BAS DÉBIT OU EN AC

Intoxications aiguës (cardiotropes : ESM)



- ANTI-ARYTHMIQUES (classe 1)
- Certains BÉTA-BLOQUANTS
- Certains ANTI-DÉPRESSEURS (polycycliques)
- Certains PSYCHOTROPES (phénothiazines, IRS, carbamazépine)
- ANTI-PALUDÉENS (chloroquine)
- STUPÉFIANTS (cocaïne, dextropropoxyphène)

AC & CHOC REFRACTAIRES TOXIQUES

- La RCP doit être prolongée en cas d'AC d'origine toxique survenue devant témoin.
- L'ECLS doit être envisagée en cas d'AC ou de choc toxique réfractaire au traitement médical conventionnel optimal (incluant les fortes doses de catécholamines...).

Conférence d'Experts SRLF/SFAR 2006-2020



Quels patients doit-on assister ?



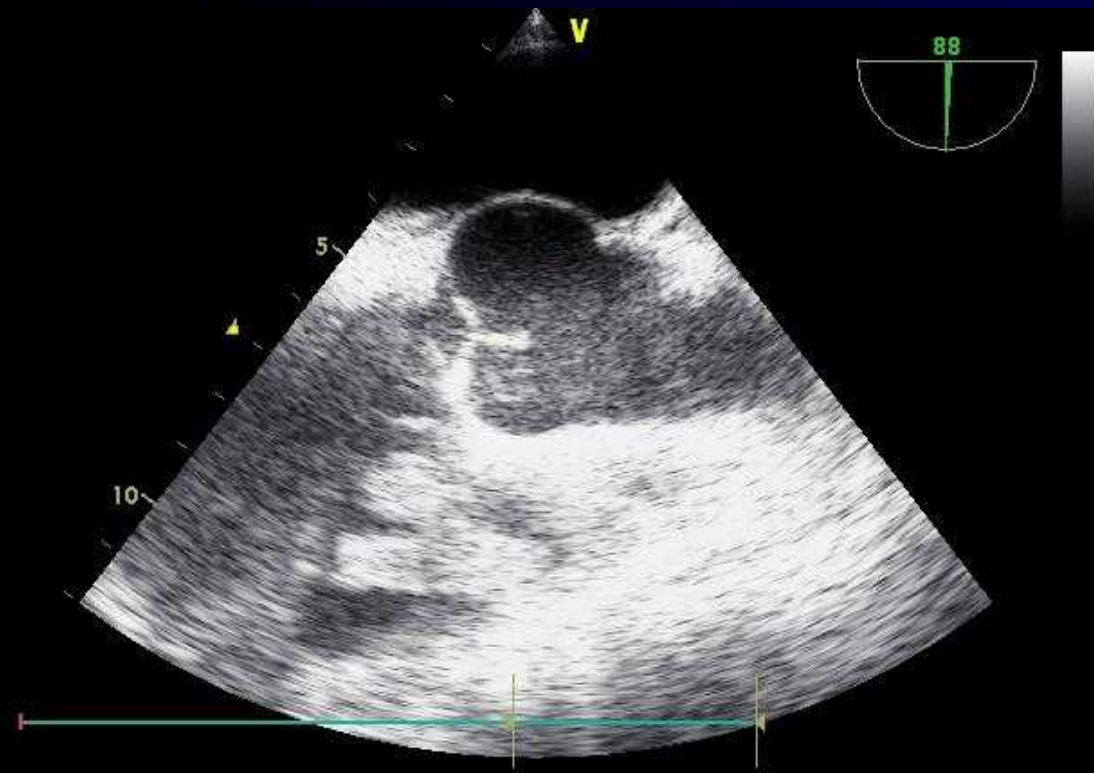
Ni trop tardif (AC) : risque d'encéphalopathie anoxique ou de défaillance multiviscérale

Ni trop facile (choc non réfractaire) : patients qui auraient guéri avec un traitement médical bien conduit = **choc cardiogénique prouvé** ($IC \leq 2,5$ l/min/m²) malgré antidotes/catécho fortes doses + défaillance d'organe persistante (pulmonaire, rénale)

Donc, CC: échec des ttt usuels + catécholamines croissantes



CAS 3 :
diagnostic ?



Ventricular fibrillation during termination of pregnancy

Nicolas Verroust, Rachid Zegdi, Vlad Ciobotaru, Vassilis Tsatsaris, François Goffinet, Jean-Noel Fabiani, Alexandre Mignon

EA après ITG à 35 SA mise sous ECLS

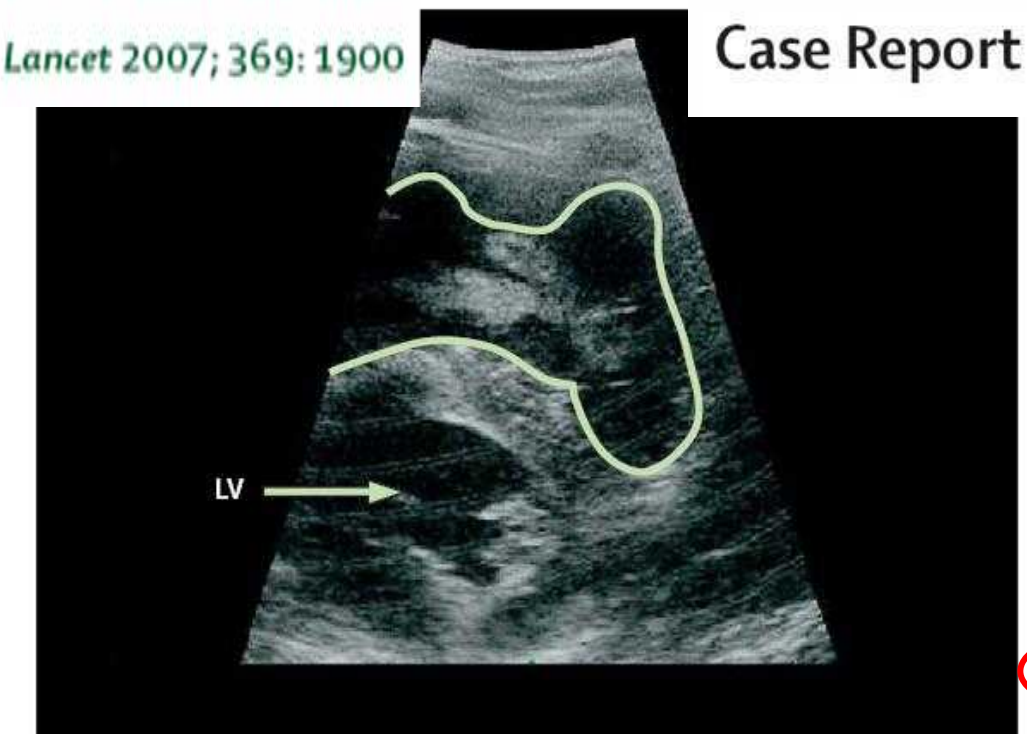


Figure: Echocardiography, showing AFE
The grossly enlarged right ventricle is outlined. At least three intracardiac masses are visible. The left ventricle (LV) is also visible.

3 précédentes grossesses : RAS

Déclenchement W (Misoprostol)

Rupture des membranes artificielle

Min 15 : coma, arrêt respiratoire

FV : CEE, MCE

CIVD $\Rightarrow$ Hémorragie de la délivrance

Massage utérin, ocytocine, packing utérin, sulprostone, CGR

Prélèvement sanguin (anapath) : cellules amniotiques et fœtales (coloration Bleu de Nile et Wright)

Embolisation utérine : stabilisation

Choc réfractaire ($FE_{VG} < 15\%$) :

ECLS à H8

SIRS : corticoïdes (J12)

Décanulation J20, extubation J26

Sortie J40 (pas de séquelle)



IM : 8.8
 2.8/2.8-M/C
 28 MARS 99
 12:10:53
 TRAIT 8/E
 CHU GRENOBLE
 CARDIO PR DENIS
 CARDIO
 PERICARDE

HBOUVAIST

046829.19
 GAIN 80
 COMP 70

18cm
 29Hz



Tamponnade



IM : 8.8
 2.8/2.8-M/C
 28 MARS 99
 12:11:36
 TRAIT 8/E
 CHU GRENOBLE
 CARDIO PR DENIS
 CARDIO
 PERICARDE

HBOUVAIST

046848.21
 GAIN 80
 COMP 70

18cm
 29Hz



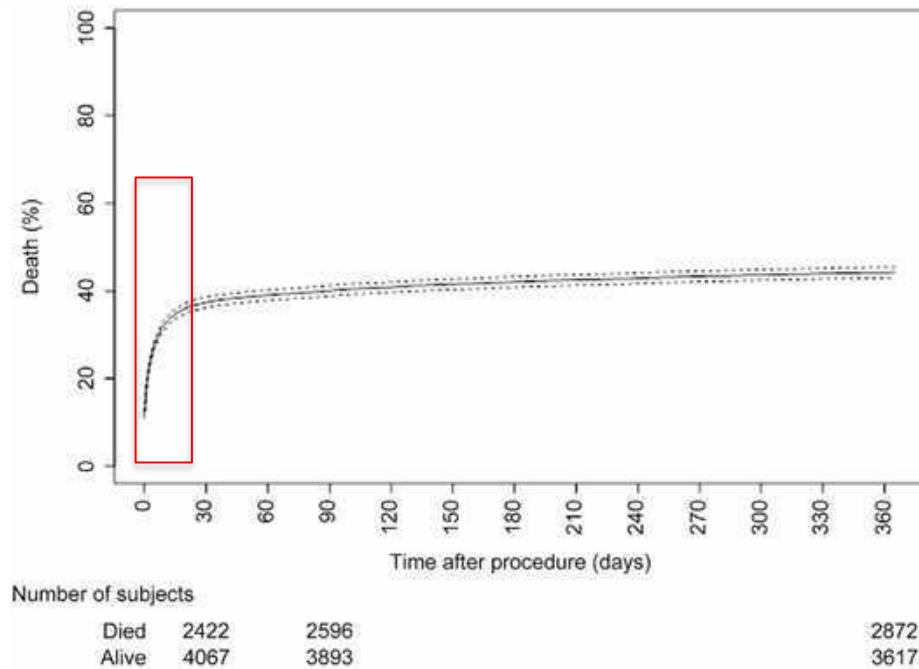
131.04
 30% 27%
 240mm
 Cardio
 General

83.8 51/43/1/
 Date: 005

FC 75bpm

Prise en charge **immédiate**

**FORTE MORTALITE PRECOCE (>48H) :
DONC PRISE EN CHARGE ACTIVE ET URGENTE**



Kunadian. *JACC Cardiovasc Interv.* 2014

Plus de 40% de la mortalité hospitalière dans les 24 premières heures !
Le pronostic ensuite semble meilleur après (CS vs. No-CS)

Assaoui. *CCM.* 2012

DIAGNOSTIC PRECOCE + STRATIFICATION + TRAITEMENT

Prise en charge **immédiate**

CS: Physiopathology and therapeutics approaches

1. Reperfusion

ESC 2014 = Classe IB
ESC 2016 = Classe IB
ESC 2017 = Classe IB

2. Inotropes

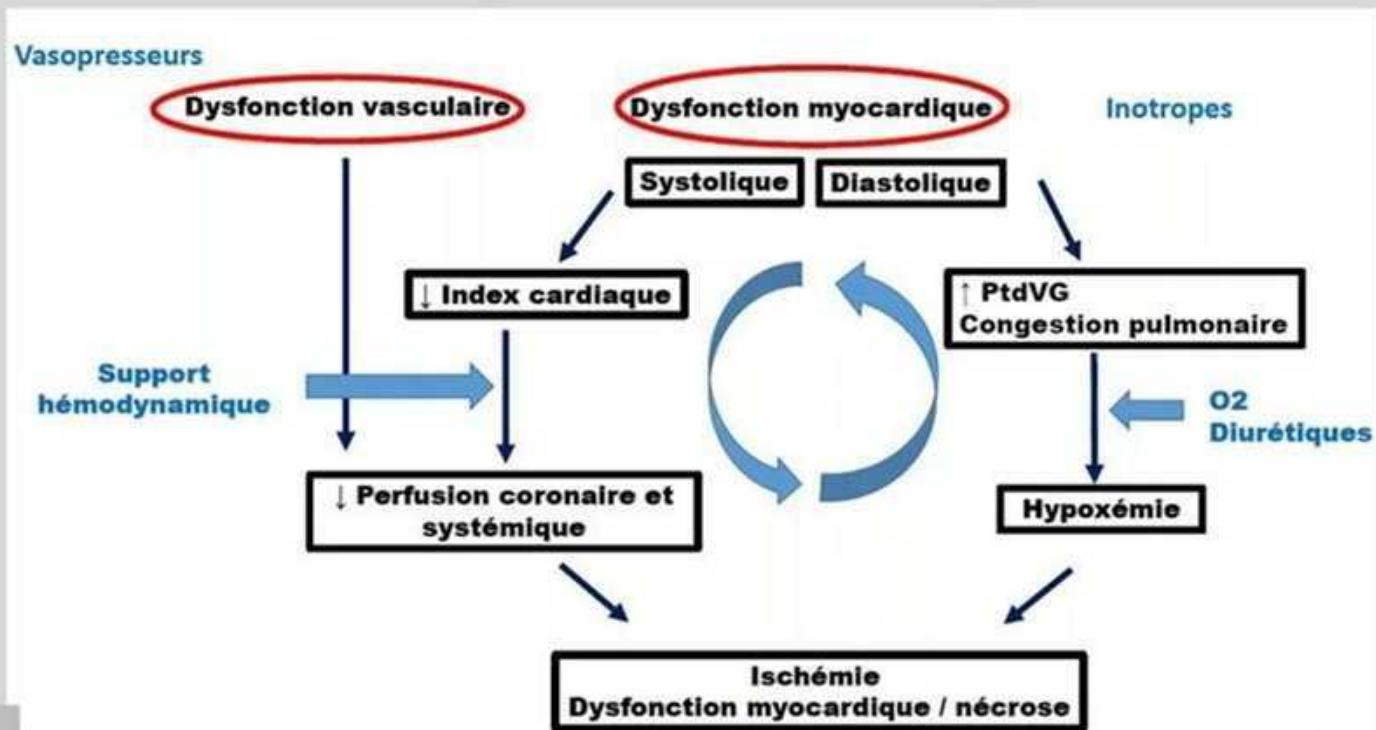
Dobutamine

ESC 2017 = Classe IIbC
ESC 2016 = Classe IIaC
SRLF = strong agreement

3. Vasopressors

Noradrénaline

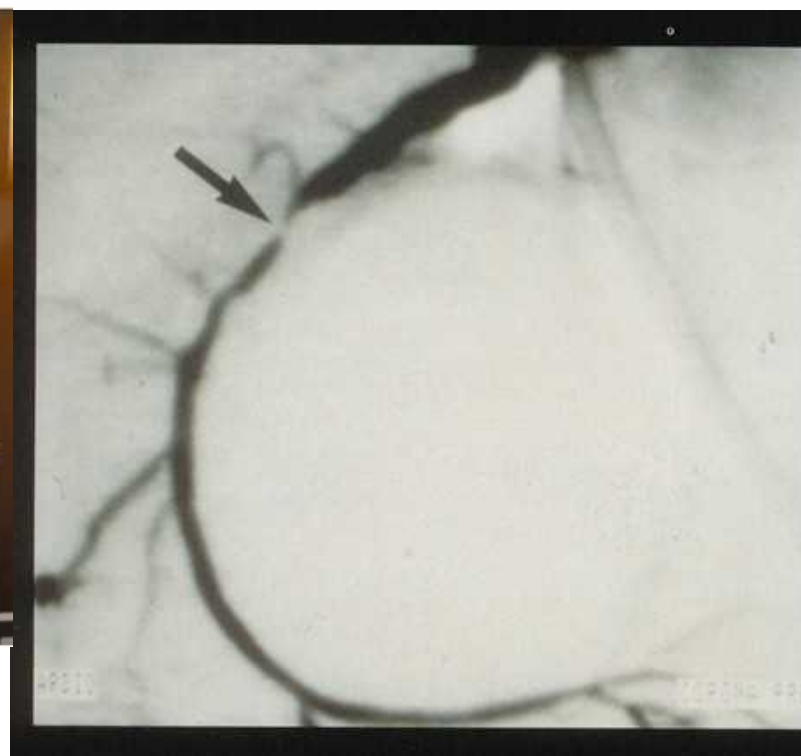
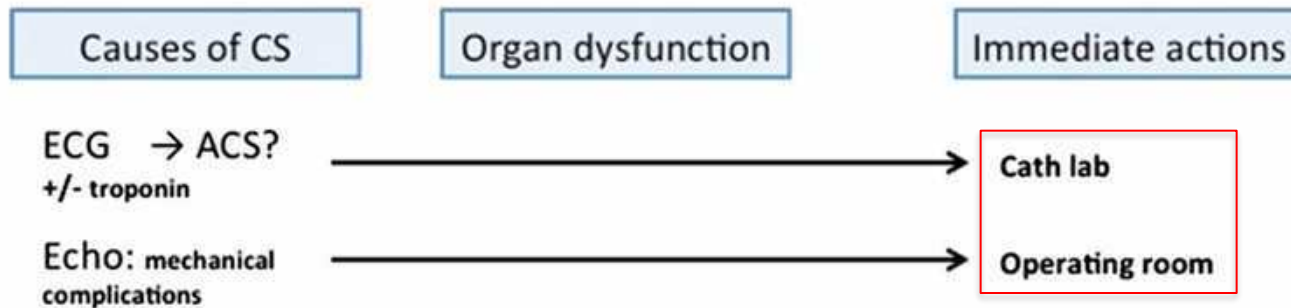
ESC 2017 = Classe IIbC
ESC 2016 = Classe IIaB
SRLF 2015 = strong agreement



Delmas C et al, MIR 2017

Prise en charge **immédiate**

TRAITEMENT ETIOLOGIQUE +++



DESOBSTRUCTION PULMONAIRE : EP
DRAINAGE PERICARDIQUE : TAMPONNADE
CHIRURGIE CARDIAQUE : DISSECTION...
TRAITER LE FACTEUR DECLENCHANT :
CEE si TDR, ATB si infection...

REVASCULARISATION CORON

Prise en charge : revascularisation

Étude SHOCK (N=302) = shock +STEMI

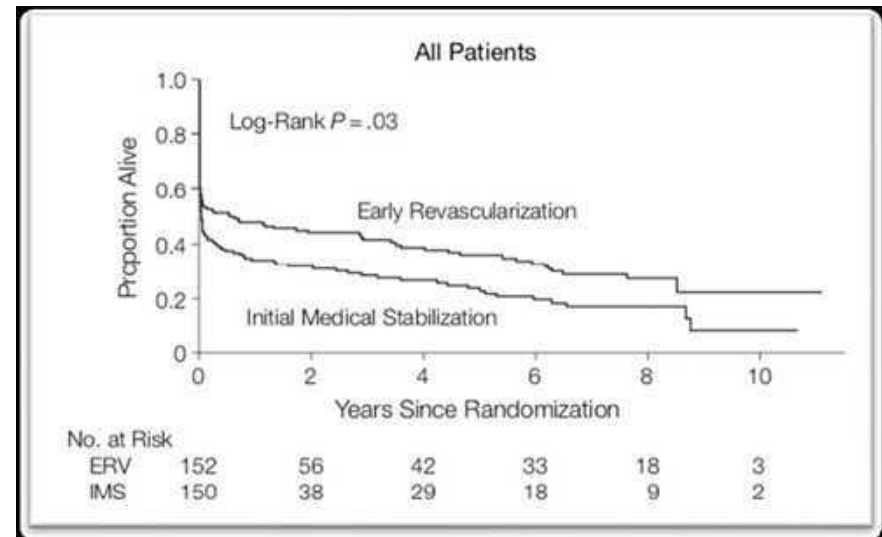
Groupe revascularisation

- ✓ Revascularisation < 6h
- ✓ Pontages ou angioplastie
- ✓ Ballon de contre-pulsion recommandé

Traitement médical

- ✓ Thrombolyse recommandée
- ✓ Ballon de contre-pulsion recommandé

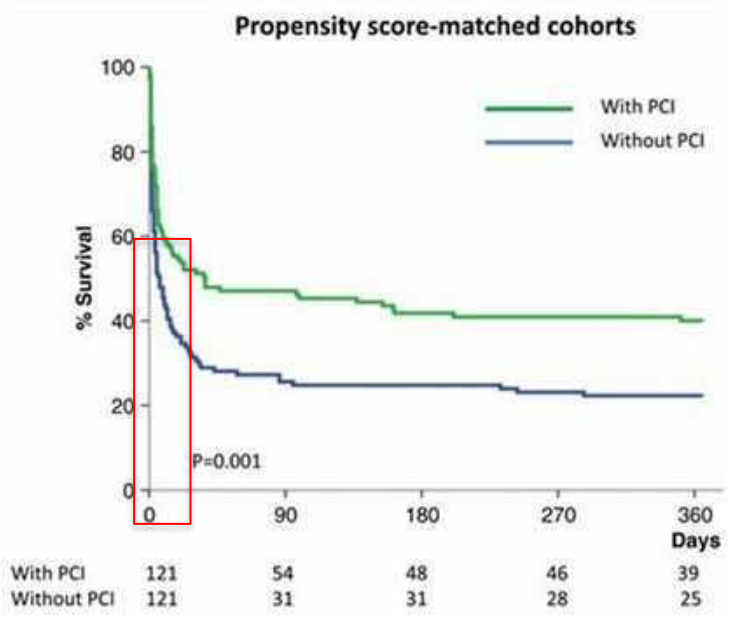
- ✓ Revascularisation :
angioplastie 54,6%, PAC 37,5%
- ✓ Traitement médical : thrombolyse 66%,
revascularisation 25,3%
- ✓ IABP 86% des patients



Prise en charge : revascularisation (STEMI / Non-STEMI)

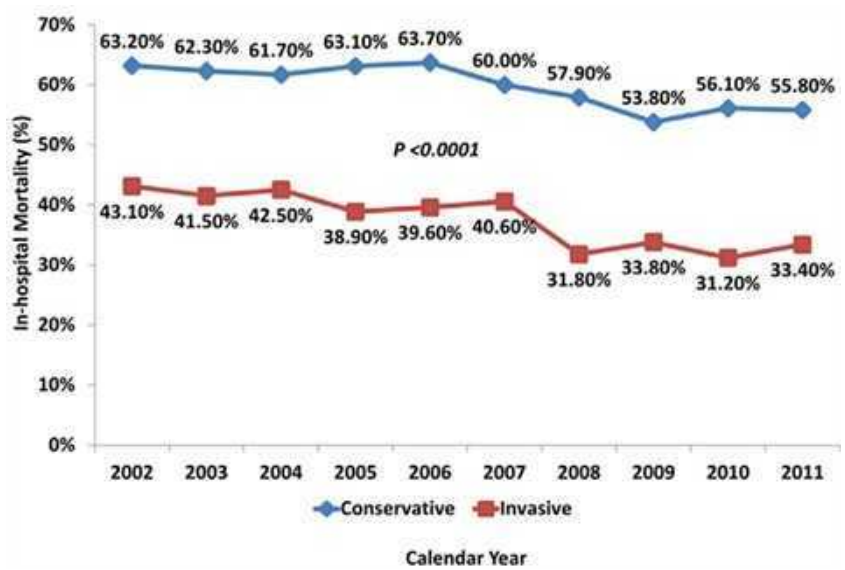
Improved outcome of cardiogenic shock at the acute stage of myocardial infarction: a report from the USIK 1995, USIC 2000, and FAST-MI French Nationwide Registrie

Assaoui. EHJ. 2012



Outcomes with Invasive vs Conservative Management of Cardiogenic Shock Complicating Acute Myocardial Infarction

Bangalore. Am J Med. 2015

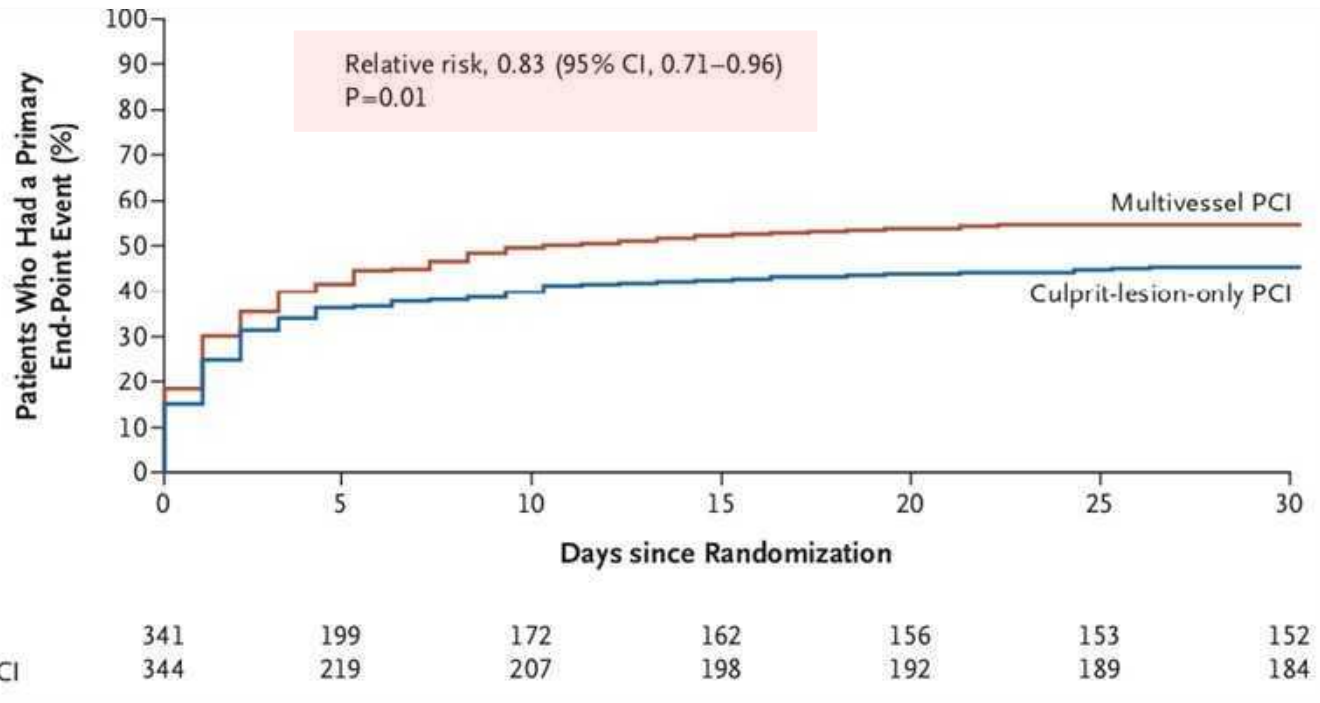


Recommendations	Class ^a	Level ^b
Immediate PCI is indicated for patients with cardiogenic shock if coronary anatomy is suitable. If coronary anatomy is not suitable for PCI, or PCI has failed, emergency CABG is recommended. ^{2,48}	I	B
Fibrinolysis should be considered in patients presenting with cardiogenic shock if a primary PCI strategy is not available within 120 min from STEMI diagnosis and mechanical complications have been ruled out.	IIa	C

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

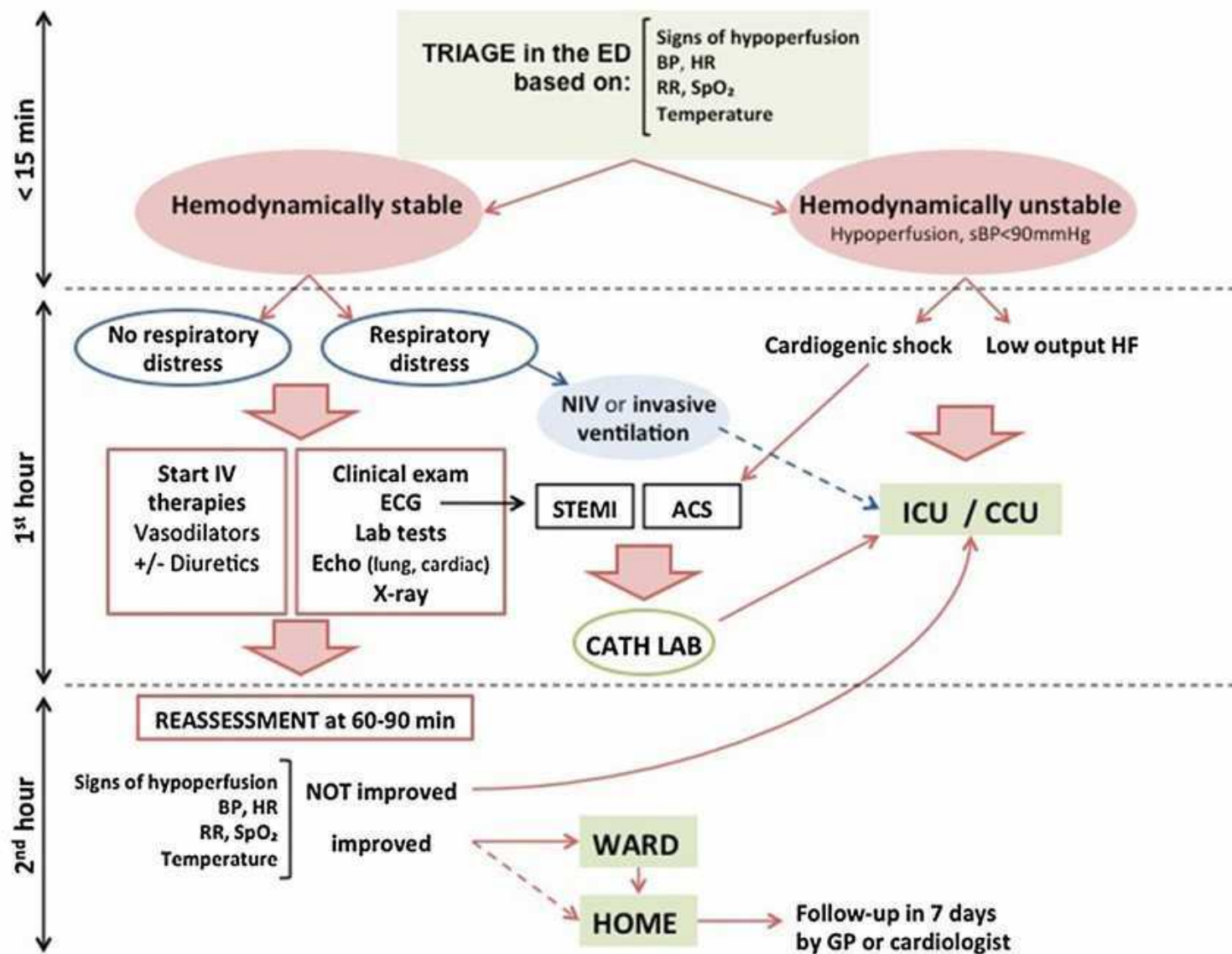
Ibanez. EHJ. 2018

En cas de lésions multiples : traiter l'artère coupable



- ✓ Choc cardiogénique sur SCA : revascularisation complète versus lésion coupable
- ✓ A 30 jours, critère composite (décès et EER) significativement plus bas dans le groupe *Culprit vessel* que dans le groupe *Multivessel* : 45.9% vs. 55.4%; relative risk, 0.83; 95% [CI], 0.71 to 0.96; P = 0.01
- ✓ Support mécanique 28,8% vs. 27,8%

Prise en charge **immédiate**



Prise en charge **immédiate**

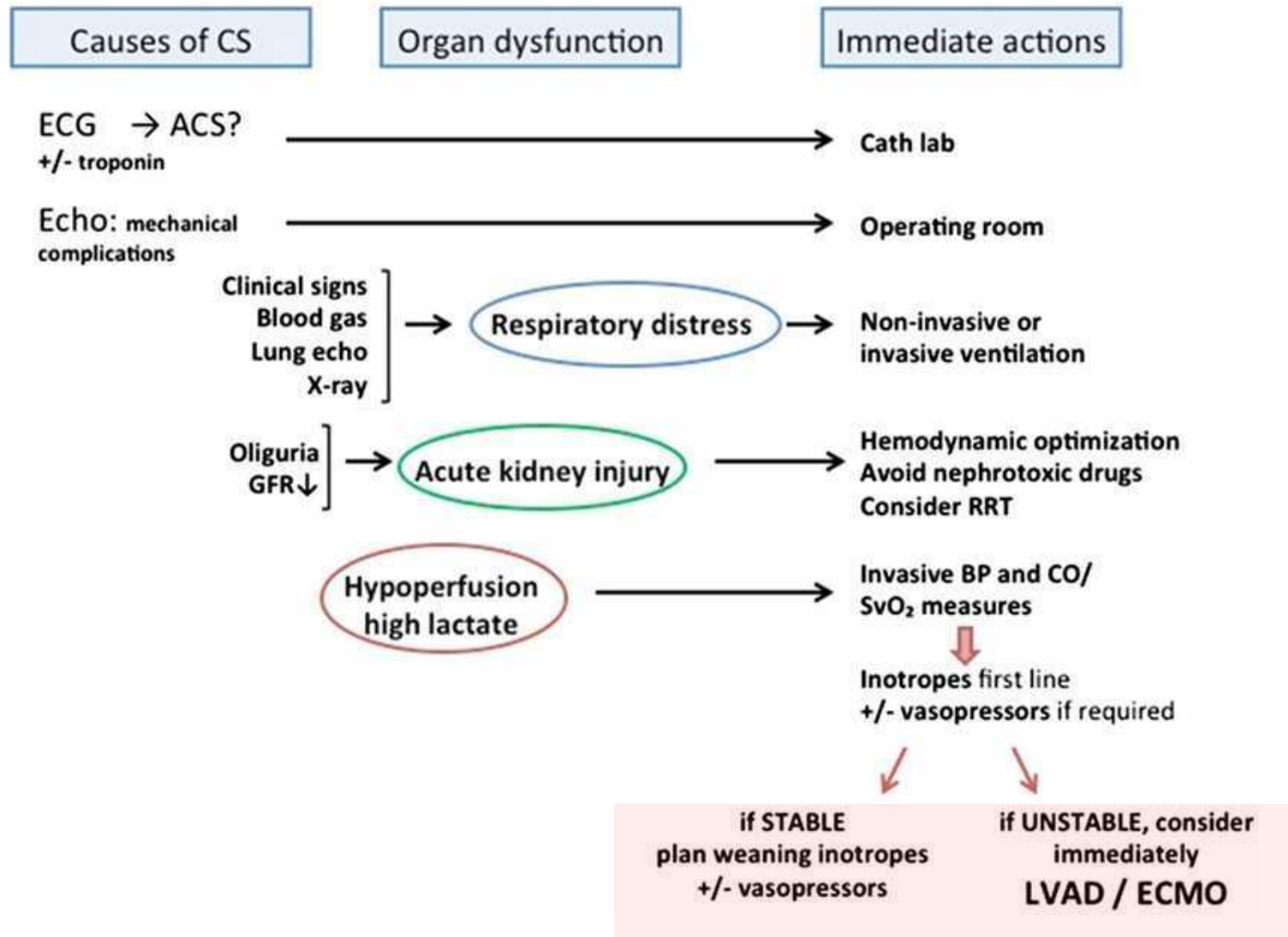
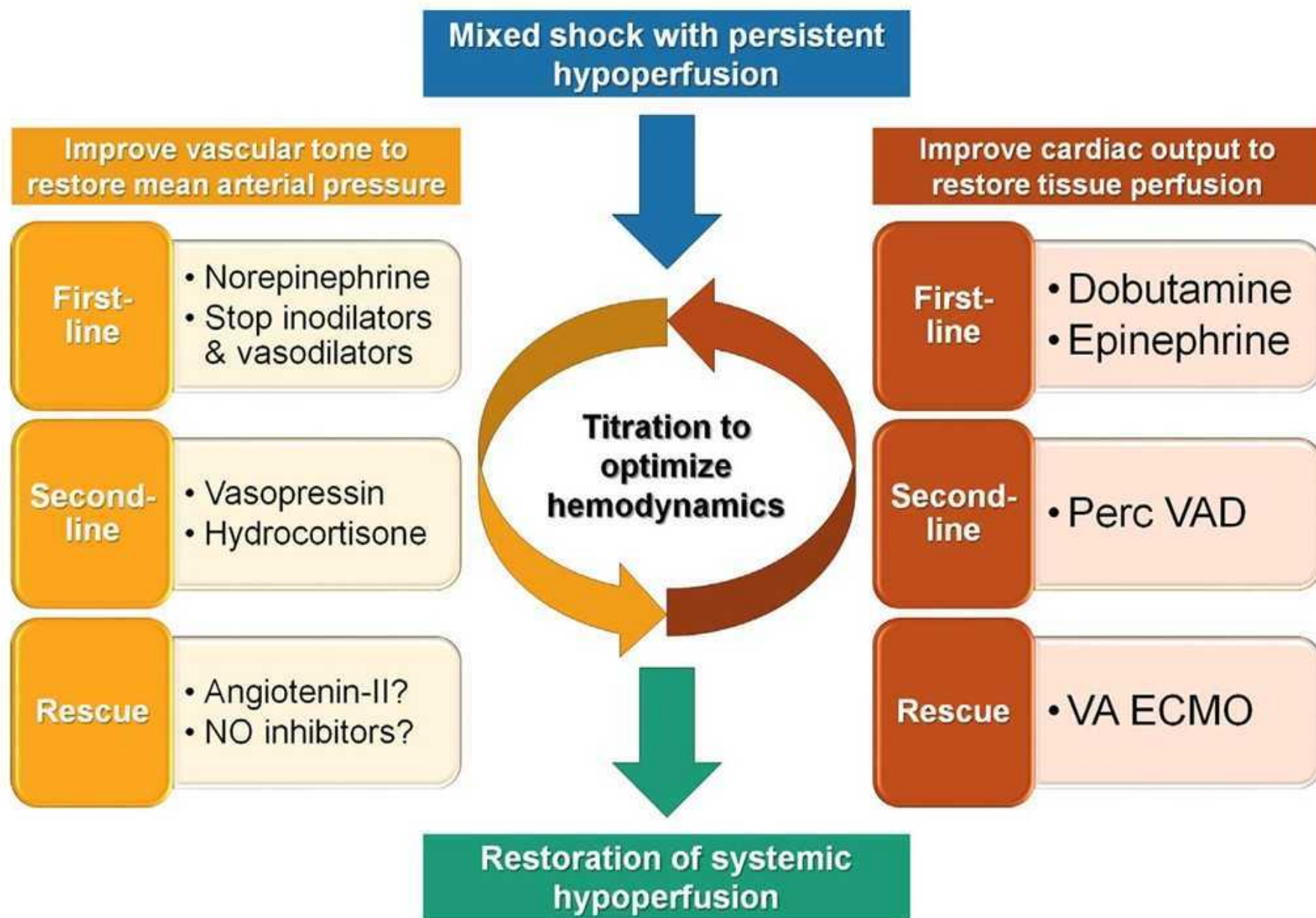


FIGURE 3 Overview of Hemodynamic Support in Mixed Shock



Prise en charge **immédiate** : arrêt du ttt chronique

Table 3 Management of oral therapy in AHF in the first 48 hours

		Hypotension		Low heart rate		Potassium		Renal impairment		
		Normotension/ Hypertension	85–100 mmHg	<85 mmHg	<60 ≥ 50 bpm	<50 bpm	≤3.5 mg/dl	>5.5 mg/dl	Cr<2.5, eGFR>30	Cr > 2.5, eGFR<30
ACE-I /ARB	review/ increase	reduce / stop	stop	No change	No change	review/ increase	stop	review	stop	
Beta-blocker	No change	reduce / stop	stop	reduce	stop	No change	No change	No change	review	
MRA	No change	No change	stop	No change	No change	review/ increase	stop	reduce	stop	
Diuretics	increase	reduce	stop	No change	No change	review/ No change	review/ increase	No change	review	
Other vasodilators (Nitrates)	increase	reduce / stop	stop	No change	No change	No change	No change	No change	No change	
Other heart rate slowing drugs (amiodarone, CCB, Ivabradine)	review	reduce / stop	stop	reduce / stop	stop	review/stop (*)	No change	No change	No change	

Farmakis D et al. Int J Card 2015 *Mebazaa et al. EHJHF 2*

Farmakis.D et al, Int J Card 2015 Mebazaa et al, EHJHF 2015

Recommendations	Class ^a	Level ^b
In case of worsening of chronic HFrEF, every attempt should be made to continue evidence-based, disease-modifying therapies, in the absence of haemodynamic instability or contra-indications.	I	C

Ponikowski P et al, ESC HF guidelines, EHJ 2016

Cardiogenic shock = stop usual chronic HF treatment

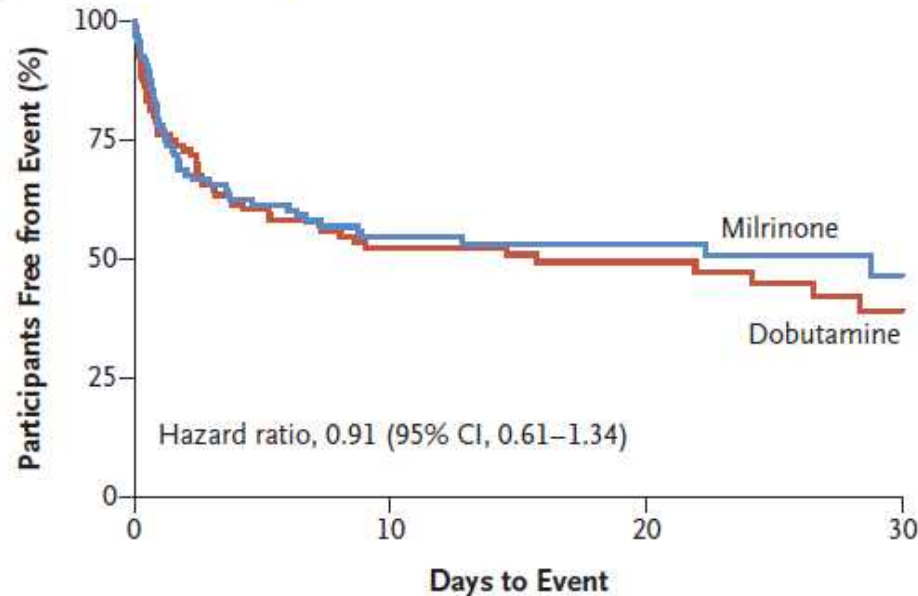
Inotrope idéal ?

- ✓ Titration facile pour un effet on/off
- ✓ Équilibre entre l'apport et la demande en oxygène du myocarde
- ✓ Effet régulier dans le temps (pas de tachyphylaxie)
- ✓ Effet inotrope positif direct
- ✓ Stimulation inotrope positive β -indépendante
- ✓ Peu ou pas d'effets arythmogènes
- ✓ Pas d'augmentation de la surcharge calcique intracellulaire
- ✓ Maintien de la pression de perfusion coronaire
- ✓ Effets bénéfiques sur les lits vasculaires régionaux
- ✓ Rapport bénéfice/risque raisonnable

Aucune étude prospective randomisée n'a prouvé la supériorité d'un inotrope

Dobutamine versus milrinone

A Primary Composite Outcome



Pas de bénéfice de la milrinone sur critère composite, sur mortalité et sur critères secondaires.

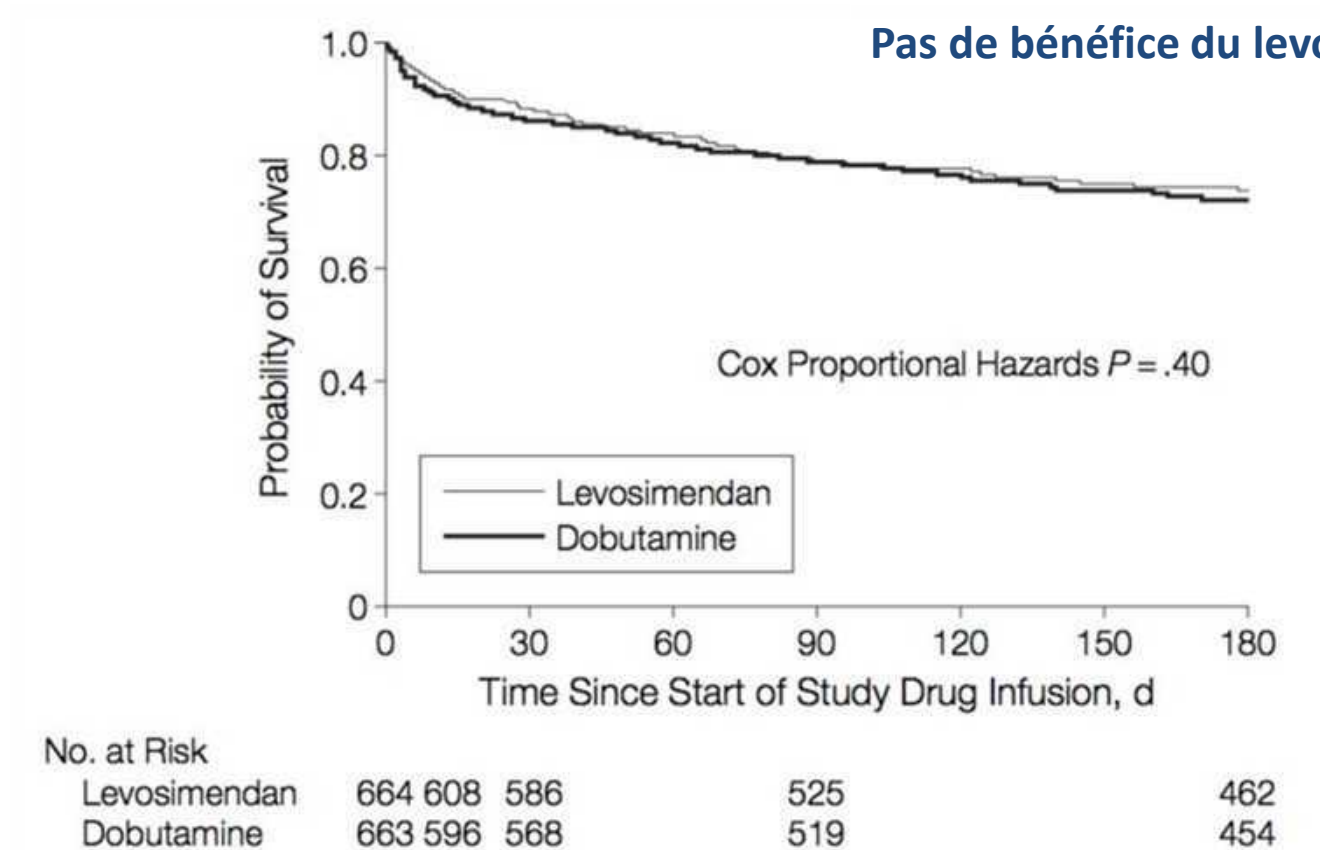
Pas de différence sur diurèse, lactate...

No. at Risk

Milrinone	96	42	26	7
Dobutamine	96	43	25	13

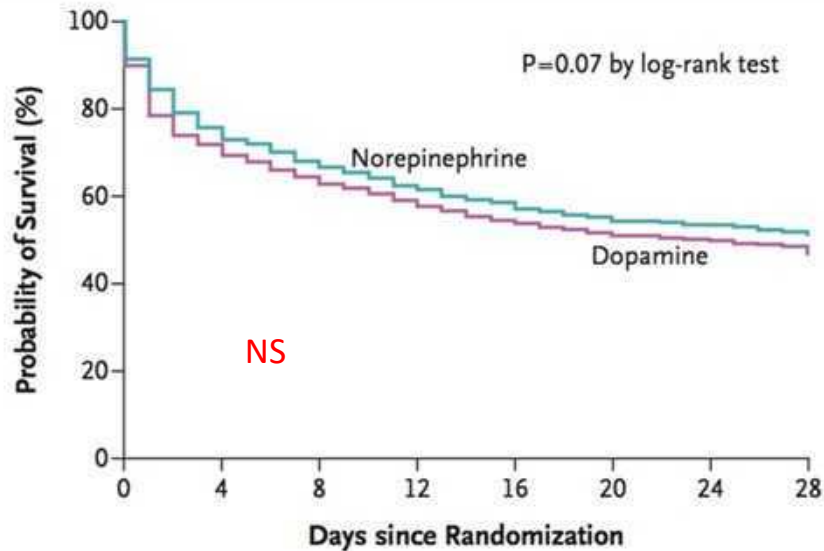
- ✓ DOREMI : étude prospective multicentrique randomisée en double aveugle (SCAI $\geq$ B)
- ✓ Critère de jugement composite : mortalité hospitalière, ACR, transplantation / assistance, SCA, AVC, EER

Levosimendan versus Dobutamine



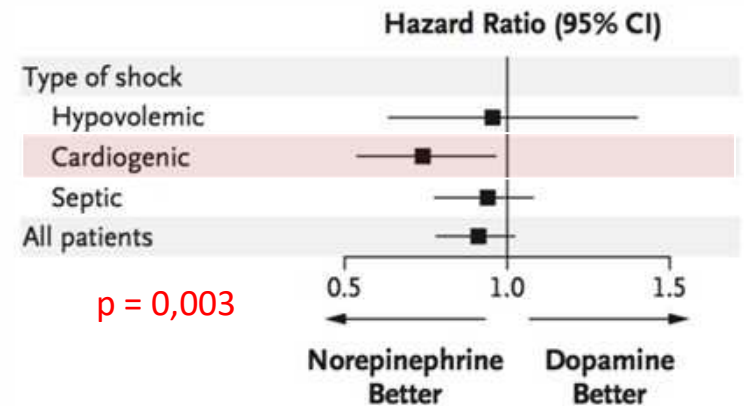
- ✓ Étude prospective multicentrique randomisée
- ✓ Dobutamine (N=663) versus levosimendan (N=664)

Quel vasopresseur ?



No. at Risk									
		0	4	8	12	16	20	24	28
Norepinephrine		821	617	553	504	467	432	412	394
Dopamine		858	611	546	494	452	426	407	386

Vasopresseur = noradrénaline



- ✓ Étude prospective multicentrique randomisée (N=1679 patients)
- ✓ Dopamine versus noradrénaline
- ✓ Choc septique (n=1044), cardiogénique (N=280), hypovolémique (N=263)

Noradrénaline versus Adrénaline

Étude OptimaCC

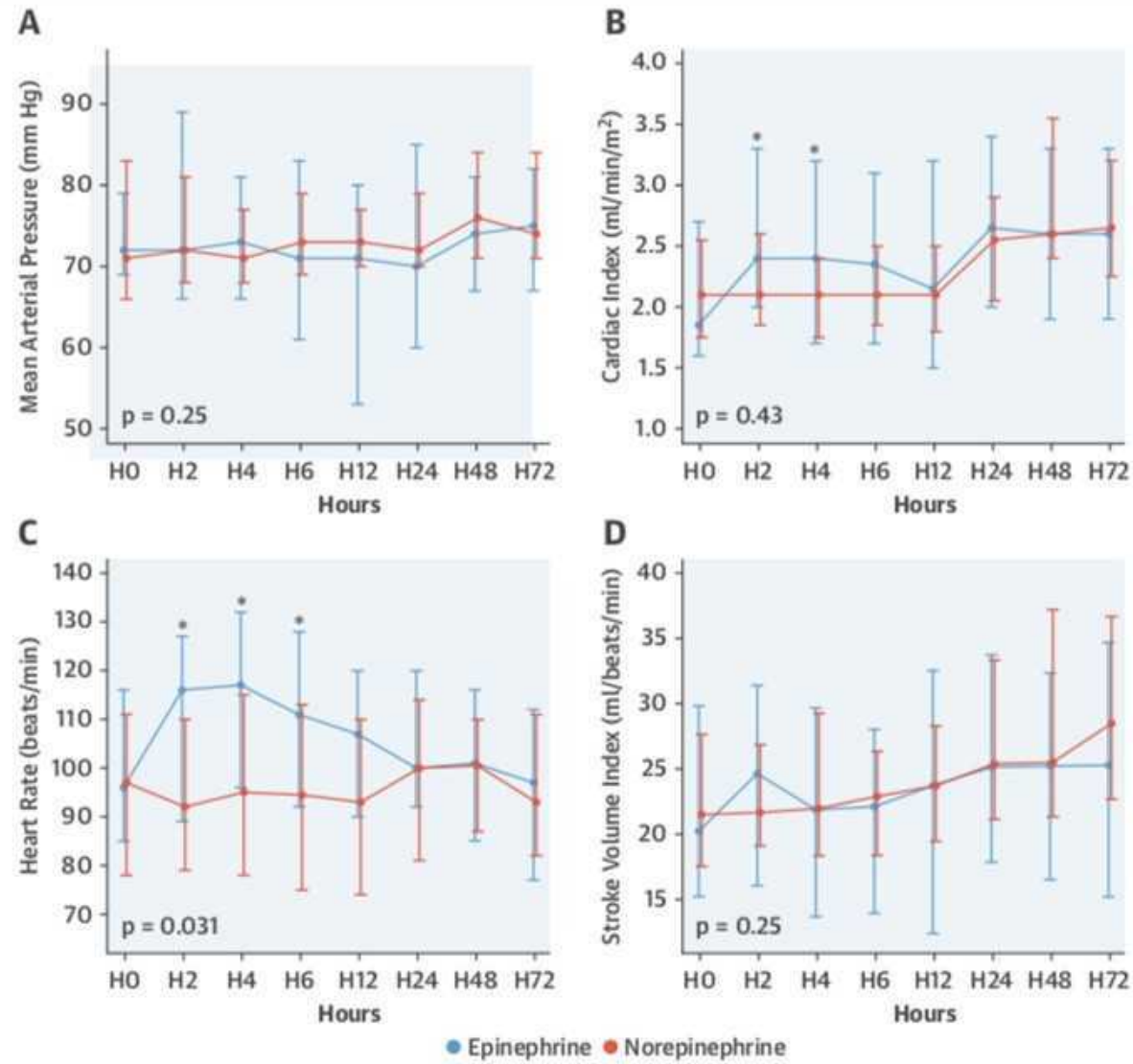
(N=57 CS + STEMI)



Adrénaline (N=27)

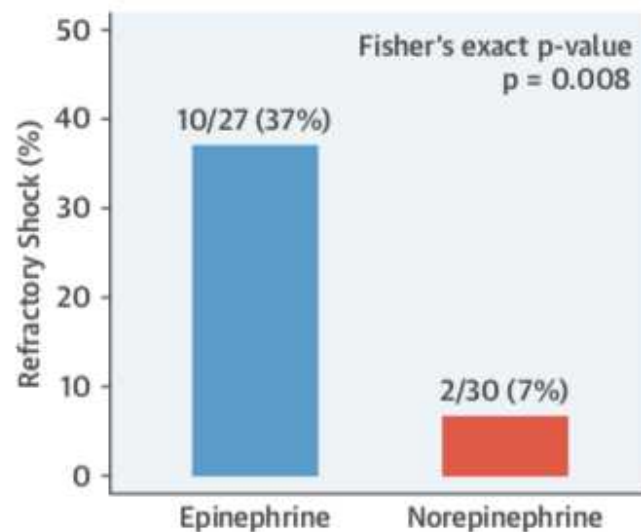
Noradrénaline (N=20)

Critère de jugement principal : index cardiaque



Noradrénaline versus Adrénaline

	Epinephrine (n = 27)	Norepinephrine (n = 30)	p Value*	Odds Ratio (95% Confidence Interval)	p Value†
Refractory shock	10 (37)	2 (7)	0.008	8.24 (1.61-42.18)	0.011
Arrhythmia	11 (41)	10 (33)	0.59	1.37 (0.47-4.05)	0.56
ECLS	3 (11)	1 (3)	0.34	3.62 (0.35-37.14)	0.28
Death	14 (52)	11 (37)	0.29	1.86 (0.65-5.36)	0.25
Death within 7 days	8 (30)	3 (10)	0.093	3.79 (0.89-16.17)	0.072
Death within 28 days	13 (48)	8 (27)	0.11	2.55 (0.84-7.72)	0.097



- ✓ Augmentation des effets secondaires dans le groupe adrénaline (lactate)
- ✓ Augmentation de l'incidence de choc réfractaire dans le groupe adrénaline (10 of 27 [37%] vs. le groupe noradrénaline (2 of 30 [7%]) ; p = 0.008
- ✓ Augmentation de la mortalité

Quelles catécholamines ?

REVIEW

Open Access

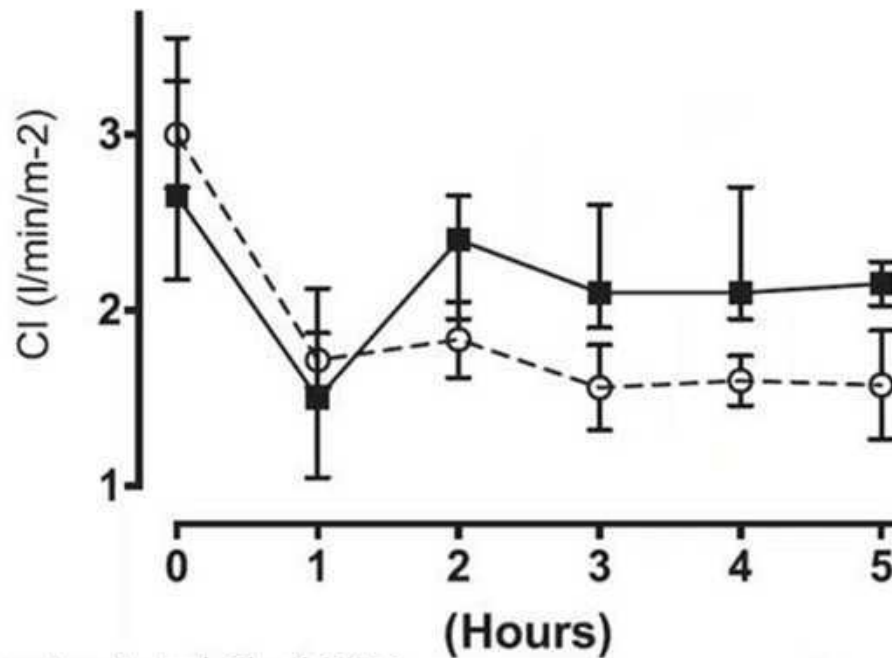


Experts' recommendations for the management of adult patients with cardiogenic shock

1. **Norepinephrine** should be used to restore perfusion pressure during cardiogenic shock (strong agreement)
2. **Dobutamine** should be used to treat low cardiac output in cardiogenic shock (strong agreement)
3. Phosphodiesterase inhibitors or levosimendan should not be used firstline (strong agreement)
4. CS refractory to catecholamines can be treated by perfusion of **phosphodiesterase inhibitors or levosimendan**
5. There is a pharmacodynamic rationale for the use of **levosimendan in patients on chronic beta-blocker treatment**

Quelles catécholamines ?

In case of hypotension: Norepinephrine first !



Effets vasculaires α > action β_1

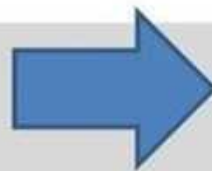
Nad = également un effet inotrope +

Vasopressors

A vasopressor, preferably norepinephrine, may be considered in patients with cardiogenic shock to increase blood pressure and vital organ perfusion.^{485–487}

IIb

B



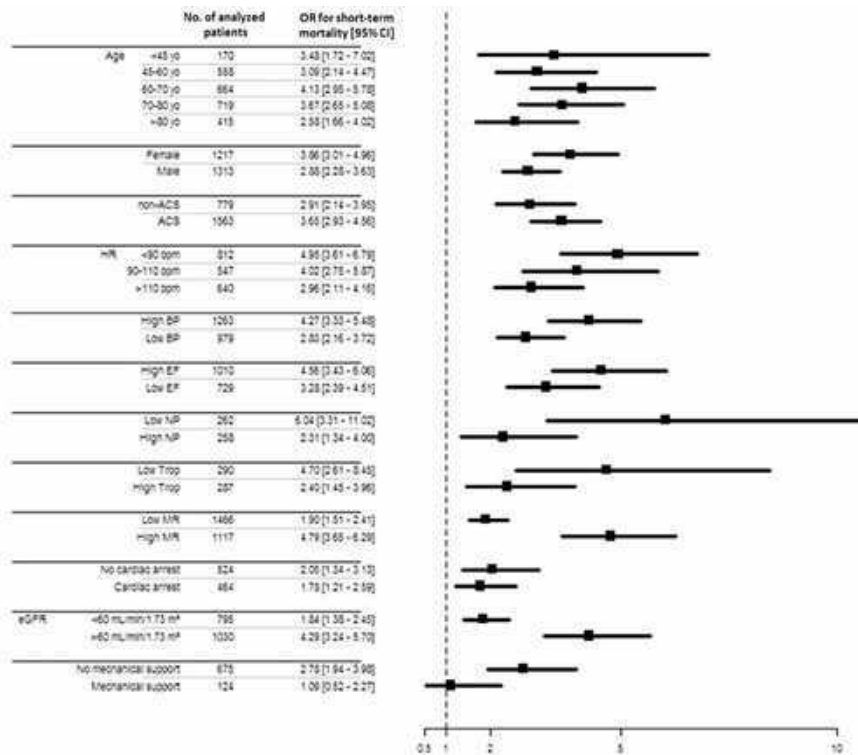
Quelles catécholamines ? **PAS D'ADRENALINE !**

Epinephrine and short-term survival in cardiogenic shock: an individual data meta-analysis of 2583 patients

Leopold V. *ICM. 2018*

12 +2 cohortes / 3 RCT

ADRENALINE = 3 FOIS PLUS DE MORTALITE



	No. of patients	No. of patients receiving epinephrine	OR for short-term mortality [95% CI]
Adler, 2012	40	10	4.00 [0.87 - 18.45]
Adler, unpublished	47	9	4.27 [0.88 - 20.67]
AHEAD, 2011	674	304	15.08 [9.08 - 25.05]
ALARM, 2011	520	86	2.14 [1.34 - 3.42]
Chua, 2011	105	80	0.99 [0.40 - 2.45]
CARDSHOCK, 2016	219	46	6.64 [3.22 - 13.71]
Champion, 2014	192	130	7.27 [2.85 - 18.54]
EFICA, 2006	158	75	3.10 [1.61 - 5.98]
Gaudard, 2015	40	11	3.15 [0.75 - 13.26]
IMPRESS in Severe Shock, 2017	48	14	12.55 [2.38 - 66.01]
OPTIMA CC, 2018	57	27	2.55 [0.84 - 7.72]
Basir, unpublished	45	8	0.96 [0.16 - 5.73]
Popovic, 2011	86	47	1.11 [0.47 - 2.63]
Simonik, 2012	89	25	1.37 [0.53 - 3.55]
SMASH, 1998	111	41	0.62 [0.20 - 1.47]
Valente, 2011	152	34	2.40 [0.38 - 14.96]
All studies	2583	947	3.33 [2.81 - 3.94]

Fig. 3 Forest plot of the meta-analysis of short-term mortality

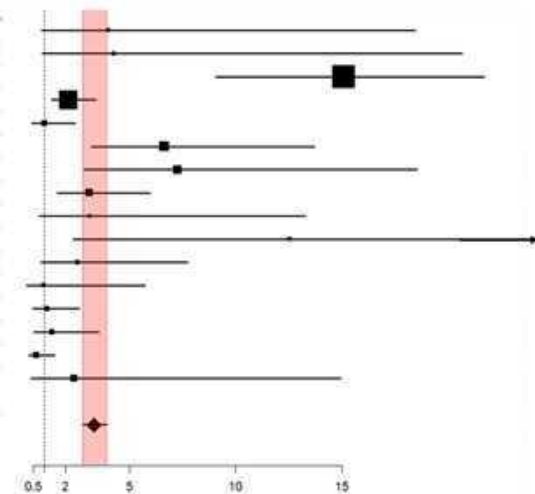
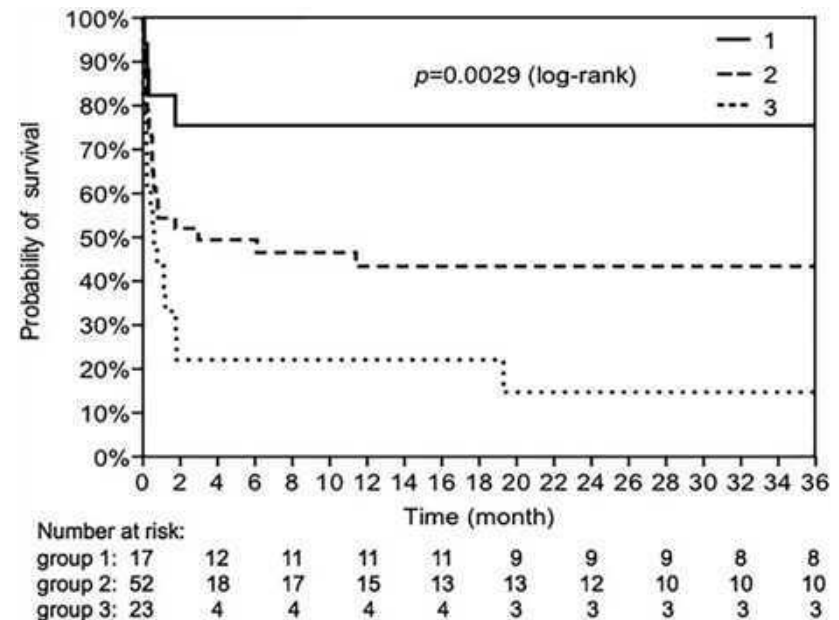
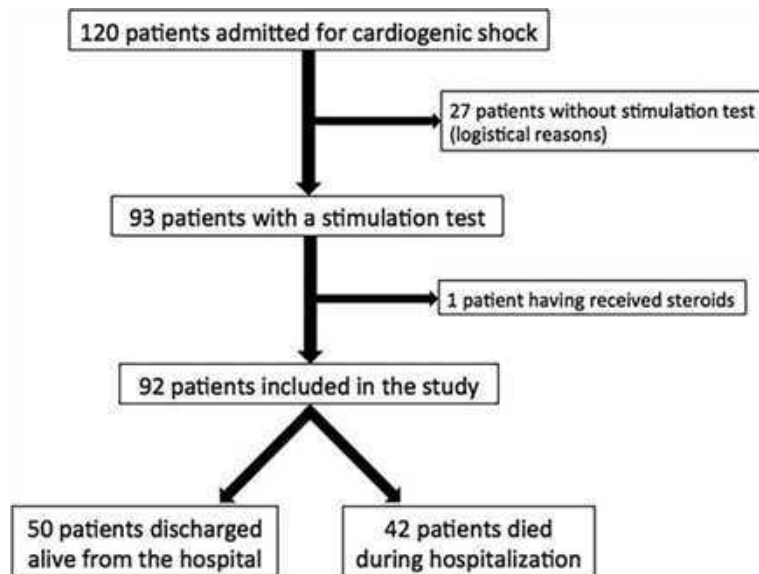


Fig. 5 Forest plot of the meta-analysis of short-term mortality, with subgroup analysis. ACS acute coronary syndrome, HR heart rate, BP blood pressure (low BP, MAP < 65 mmHg or SBP < 90 mmHg), EF ejection fraction, NP natriuretic peptide, Trop troponin, MR mortality rate. Low/high cutoff was defined as the median value within each cohort

Insuffisance surrénale : supplémentation ?



- ✓ 92 patients en choc cardiogénique : réalisation d'un test au synacthène
- ✓ Mortalité hospitalière 46%
- ✓ Insuffisance surrénale chez 15% des patients et associée avec la mortalité hospitalière

Insuffisance surrénale : supplémentation ?

TABLE 3. Variables associated with mortality in patients with cardiogenic shock

Variables	Hazard ratio (95% confidence interval); <i>P</i>	
	Univariable analysis (Cox model)	Multivariable analysis (Cox model)
Smoking	0.65 (0.37–1.15); 0.14	I/NR
Chronic heart failure	1.64 (0.93–2.99); 0.09	I/NR
SAPS II score	1.02 (1.01–1.04); <0.01	1.02 (1.01–1.04); <0.01
LVEF	0.96 (0.92–0.98); <0.01	0.95 (0.92–0.98); <0.01
Cardiac arrest	2.87 (1.62–5.05); <0.01	2.34 (1.31–4.17); <0.01
Adrenal function classification		
Group 1	1	1
Group 2	2.83 (1.10–9.57); 0.03	1.87 (0.63–5.58); 0.26
Group 3	5.27 (1.95–18.32); <0.01	3.33 (1.07–10.36); 0.04

Group 1, lower T0 and higher Δ max; Group 2, lower T0 and lower Δ max or higher T0 and higher Δ max; Group 3, higher T0 and lower Δ max; I/NR, included, but not retained by the final model; LVEF, left ventricular ejection fraction; SAPS II, Simplified Acute Physiology Score II.

- ✓ 92 patients en choc cardiogénique : réalisation d'un test au synacthène
- ✓ Mortalité hospitalière 46%
- ✓ Insuffisance surrénale chez 15% des patients et associée avec la mortalité hospitalière

Place for Oxygen supplementation +++

Oxygen and ventilatory support

Oxygen is recommended in patients with SpO₂ <90% or PaO₂ <60 mmHg to correct hypoxaemia.

I

C

Intubation is recommended for progressive respiratory failure persisting in spite of oxygen administration or non-invasive ventilation.⁴⁴⁸

I

C

Non-invasive positive pressure ventilation should be considered in patients with respiratory distress (respiratory rate >25 breaths/min, SpO₂ <90%) and started as soon as possible in order to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation.⁴⁴⁸

IIa

B

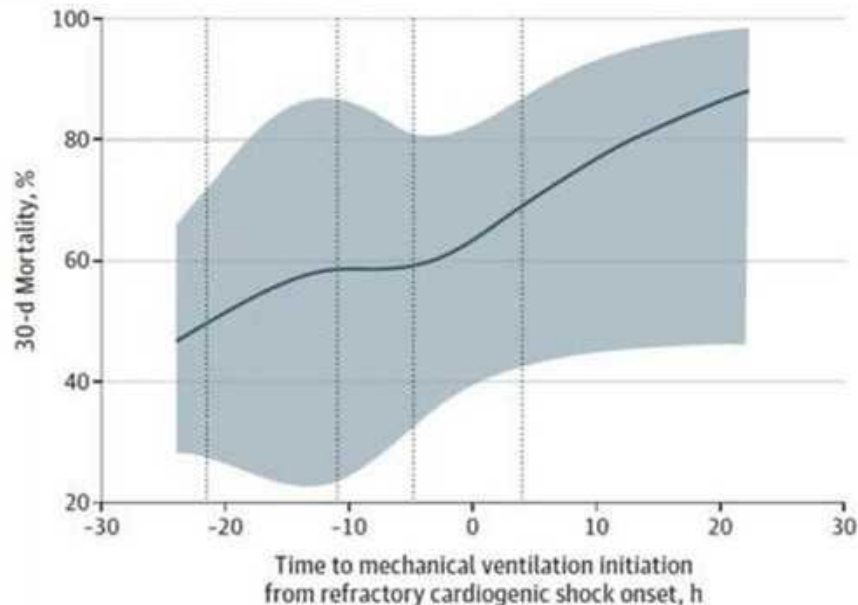
- +/- PEEP
- +/- AI
- +/- VAC
- +/- Sédation
- +/- Curarisation

Mc Donagh. EHJ. 2021

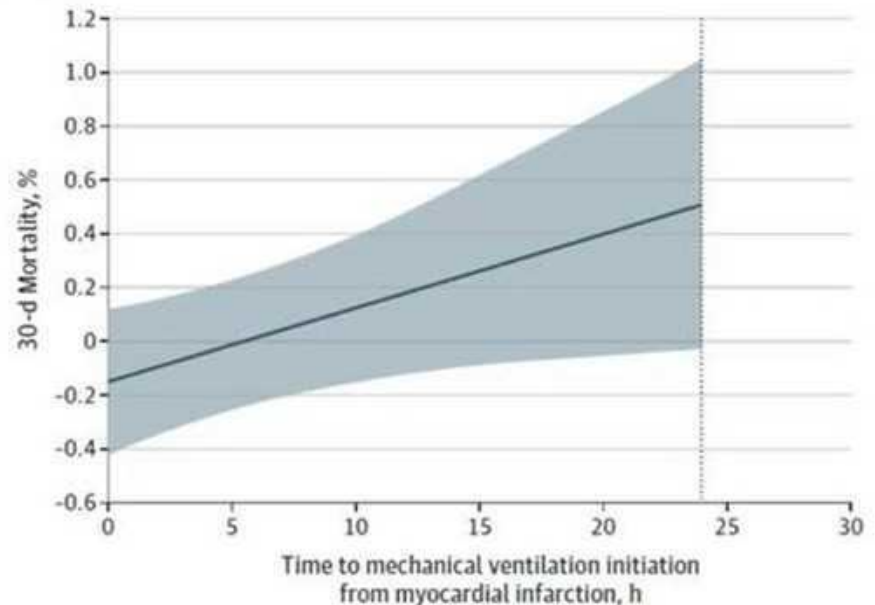
Prise en charge non médicamenteuse : O₂ / VM

What timing to initiate MV in CS?

A Refractory cardiogenic shock



B Myocardial infarction



Each 1-hour delay in mechanical ventilation initiation is associated with an increased risk of 30-day mortality.

OR, 1.03; 95%CI, 1.00-1.06; $p = 0.03$

OR, 1.04; 95%CI, 1.01-1.06; $p > 0.01$

Van Diepen. *JAMA Cardiol.* 2020

ETUDE TRIUMPH : 65% sous VM

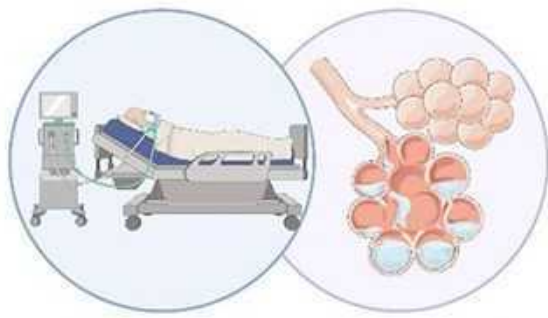
Chaque heure de délai de ventilation mécanique est associée au risque de mortalité à J30

Prise en charge non médicamenteuse : O2 / VM

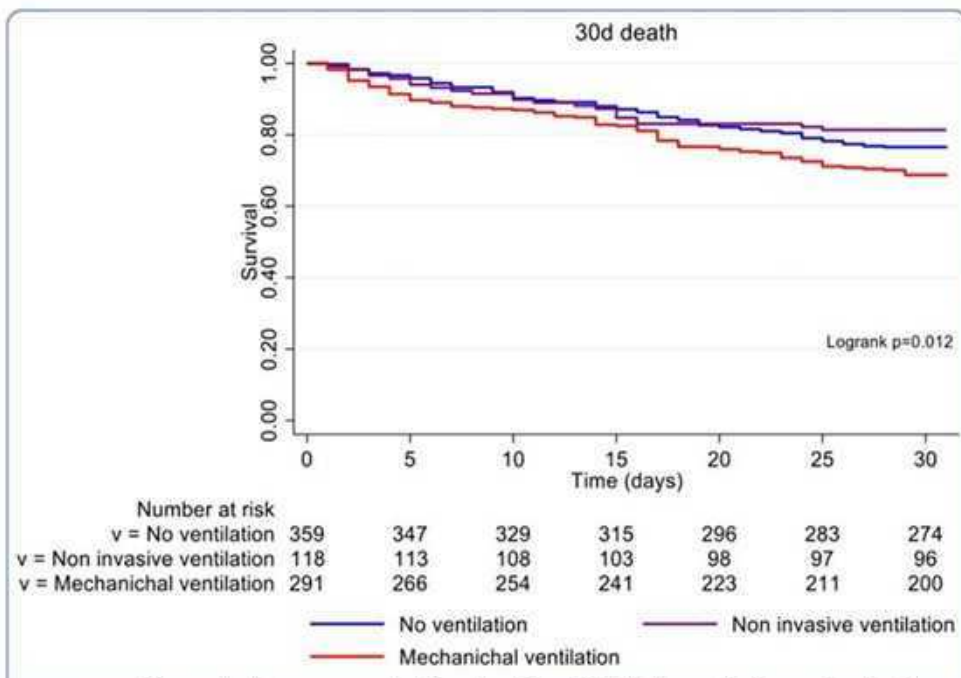
Ventilation strategies in cardiogenic shock: insights from the FRENSHOCK observational registry

Volle. Clin Research Cardiol. 2024

Short and mid-term prognosis according to ventilation strategies in unselected patients with cardiogenic shock



- 49 centers 
- 768 Cardiogenic shock:
 - 359 (46.7%) without ventilation
 - 118 (15.4%) with NIV only
 - 291 (37.9%) with MV
- More severe CS in patient under MV
- No difference in mortality between NIV patients and patients without ventilation



Our study suggests that using NIV is safe in selected patients with less profound shock CS and no other MV indication (mixed shock, post-cardiac arrest management). But special attention should be paid to CS patients under NIV

SURVEILLANCE : A TOUT INSTANT

What perfusion targets ?

- Nad pour PAM > 65 (voir 70 mmHg ?)
- Inotropes pour IC $\geq 2,2 - 2,5\text{L/Min/m}^2$

- Amélioration perfusion d'organes
- Amélioration perfusion myocarde



- Augmentation de la MVO₂ et Ischémie
- Augmentation post charge VG
- Surcharge calcique et Arrhythmies



- Cliniques: marbrure, conscience, diurèse +++
- Lactate, bio hépatique et rénale
- SVO₂ > 65-70%

SURVEILLANCE : A TOUT INSTANT

Overload

Malperfusion

Pain/Anxiety

Monitoring +++

Arterial line, Central venous catheter, urinary tube

+

Telemetry, arterial BP, pulse oxymetry, respiratory rate

+/- Invasive monitoring (CVP, PAC, PICCO,...)



Clinical

- Left and right overload signs
- Diuresis +++
- Marbles
- Confusion,...

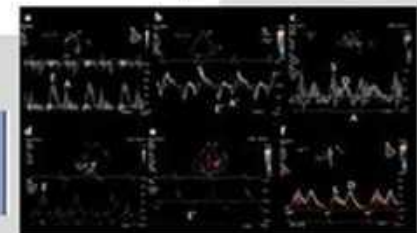
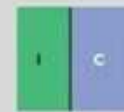
Biological

- SVO2 (4-6h)
- lactate (2-4h)
- Blood gas
- Electrolytes (6-12h)
- Creat-eGFR (12-24h)
- Cytolysis-cholestasis, fact V-TP (24h)
- Troponin, NtproBNP,...



TTE + Lung US (12-24h)

- ITV ssAo -CO/CI
- IVC diam and collapsibility
- SVRi
- ...



Chioncel.O et al, Eur J Heart Fail 2020 / Van Diepen.S et al, Circulation 2017 / Vincent.JL et al, Crit Care 2016 / Price.S et al, Nature Review Cardiol 2017

Prise en charge médicamenteuse = insuffisante

21-01-04-221513

REA CHIR CARDIAQUE

ITm 0,5 22:21:14

Cardiaque

S4-2

27Hz

20,0cm

2D

HGén

Gn 50

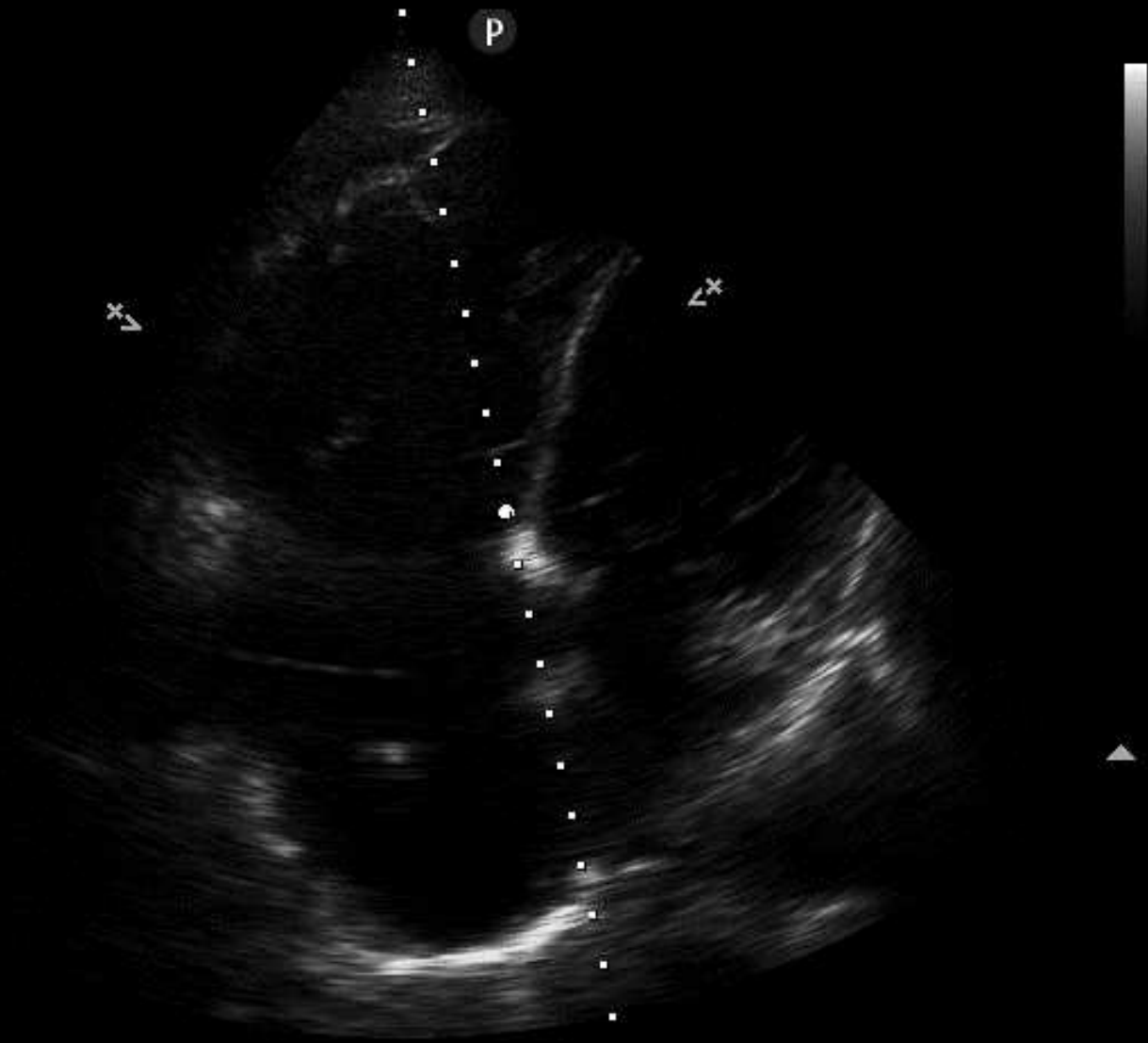
50

4/2/0

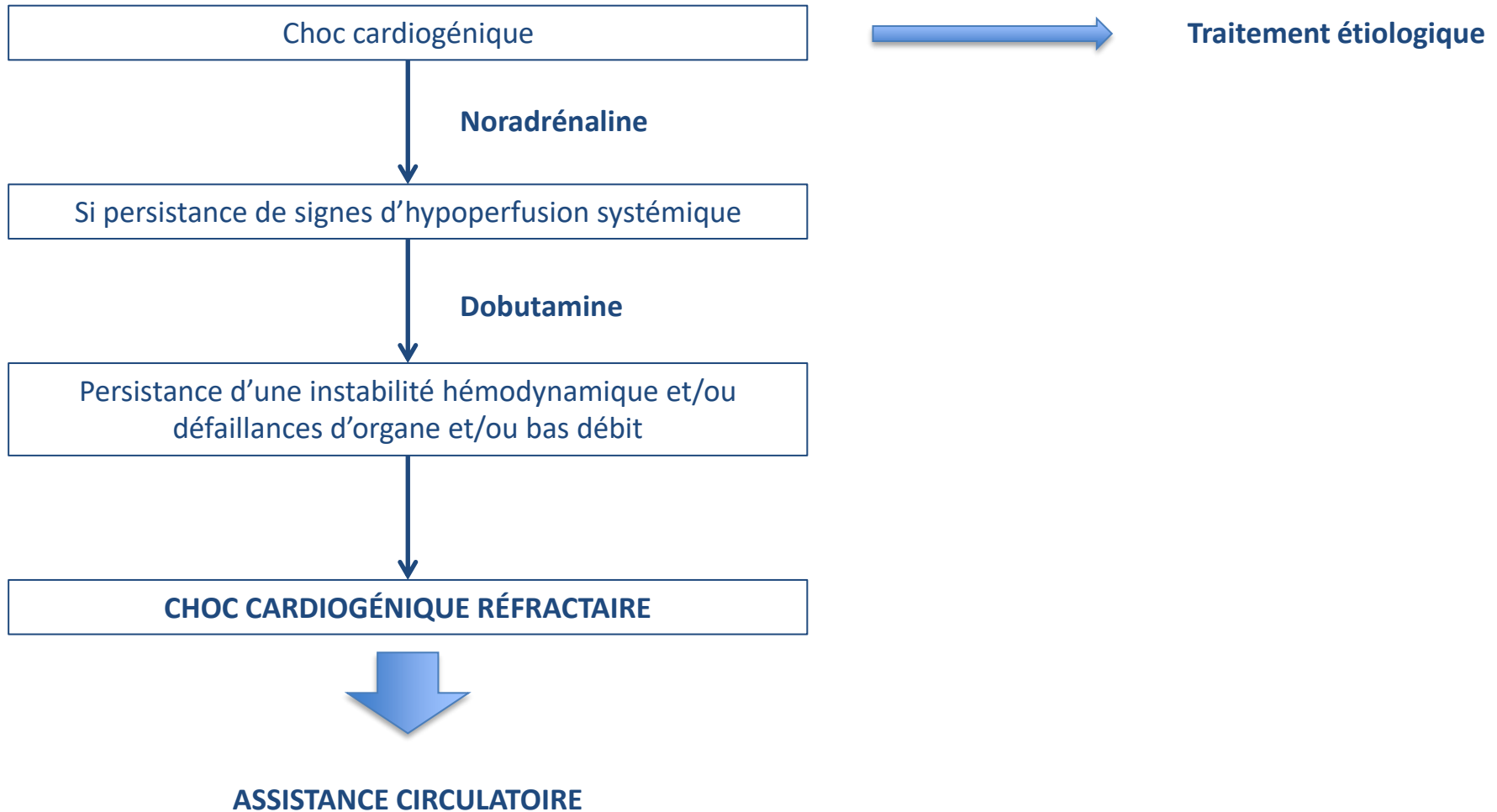
75 mm/s

Dobutamine 10 $\mu\text{g/kg/min}$

G
P R
1,6 3,2



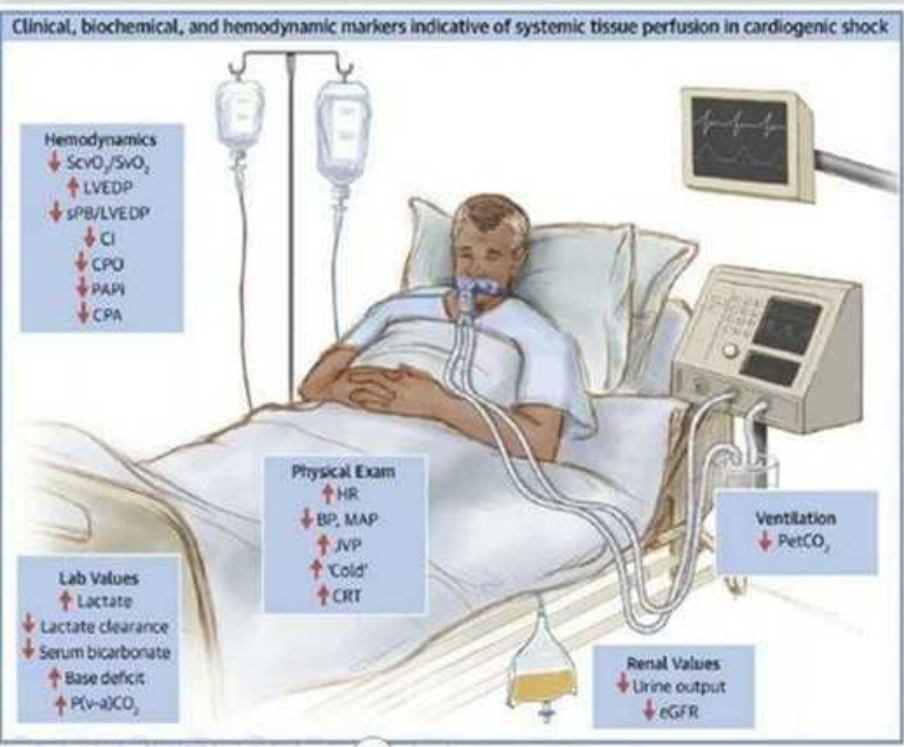
Prise en charge non médicamenteuse



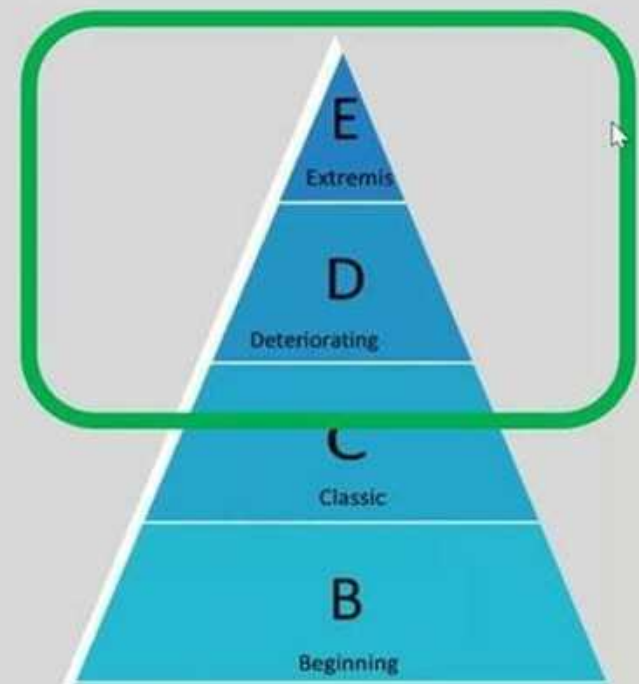
Prise en charge non médicamenteuse

When to think about aMCS ?

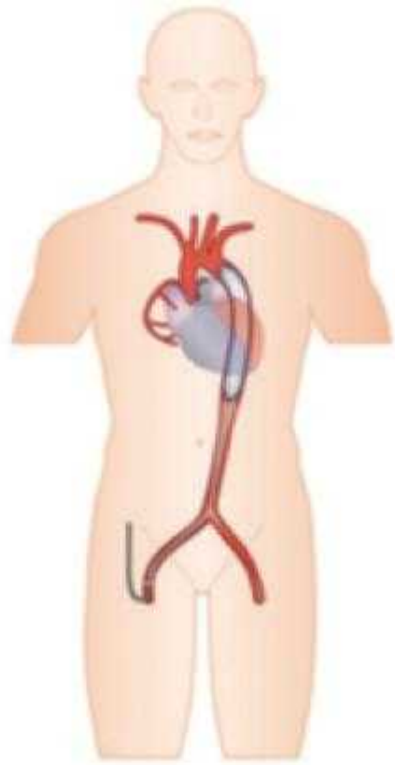
Persistent malperfusion despite initial management



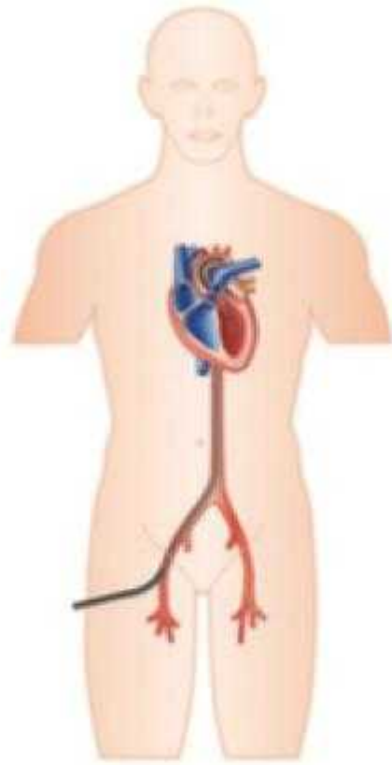
Degradation or Severe/profound initial CS presentation



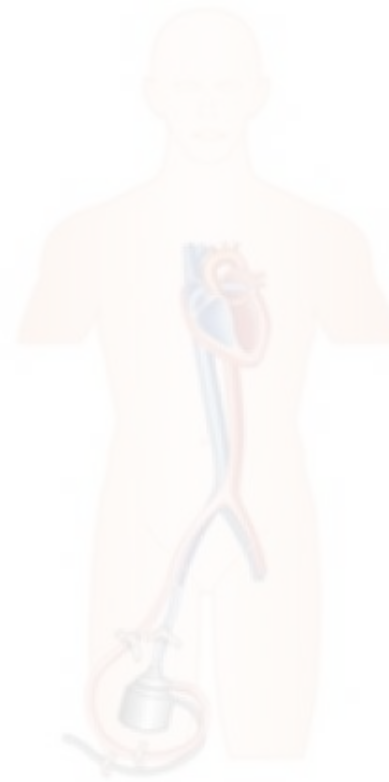
Quand les médicaments ne suffisent plus... LES ASSISTANCES CIRCULATOIRES MECANIQUES



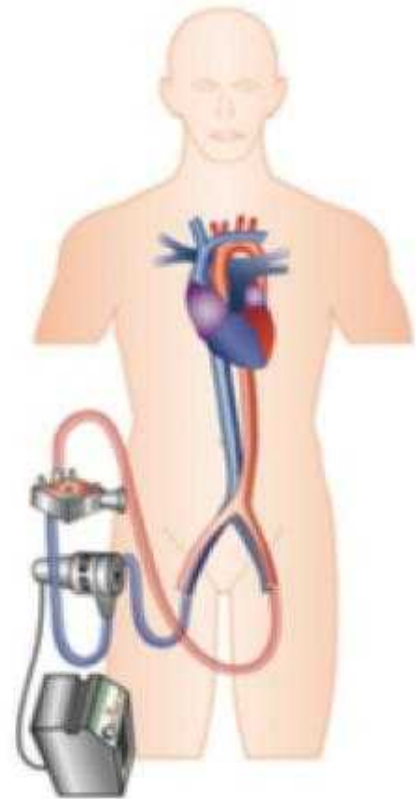
**Ballon de contre-
pulsion intra
aortique**



Impella



TandemHeart



ECMO veino-artérielle

Figure adaptée de Werdan K et al. Eur Heart J (2014)

Orages rythmiques : Sedation, Cordarone, Xylocaïne



Extracorporeal life support to terminate refractory ventricular tachycardia 11 TDR malins sur myocardite, spasme ou SDRA:
82% sevrés de l'ECLS et FEVG normalisée (2 DC)

Feng-Chun Tsai, MD; Yao-Chang Wang, MD; Yao-Kuang Huang, MD; Chi-Nan Tseng, MD;
Meng-Yu Wu, MD; Yu-Sheng Chang, MD; Jaw-Ji Chu, MD; Pyng Jing Lin, MD

CCM 2007



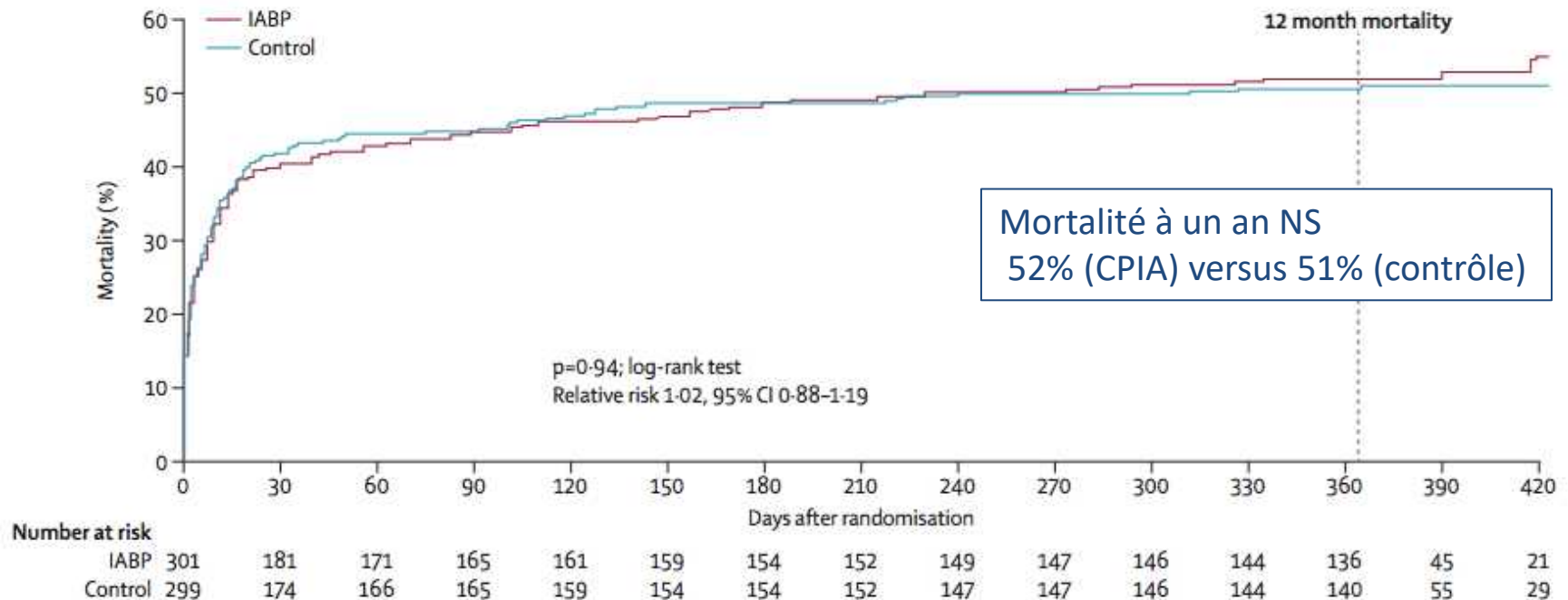
CASE REPORT

Successful extracorporeal life support in cardiac arrest with recurrent ventricular fibrillation unresponsive to standard cardiopulmonary resuscitation☆

Jae-Seung Shin^a, Sung-Woo Lee^{b,*}, Gap-Su Han^b, Won-Min Jo^a,
Sung-Hyuk Choi^b, Yun-Sik Hong^b



Ballon de contre-pulsion intra aortique



- ✓ 600 patients en choc cardiogénique compliquant un infarctus du myocarde (SCA)
- ✓ Randomisation : groupe contrôle vs. CPIA
- ✓ Revascularisation médicale (95,8%) ou chirurgicale (3,5%)

Ballon de contre-pulsion intra aortique

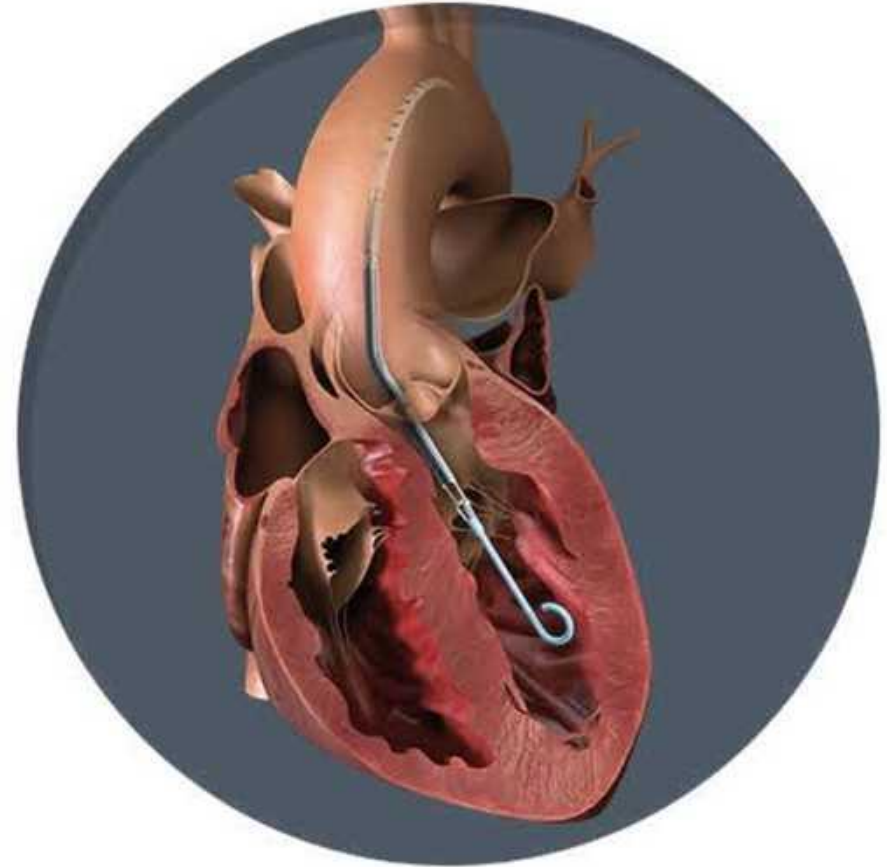
Recommendations	Class ^a	Level ^b	Ref ^c
In all patients with suspected cardiogenic shock, immediate ECG and echocardiography are recommended.	I	C	
All patients with cardiogenic shock should be rapidly transferred to a tertiary care center which has a 24/7 service of cardiac catheterization, and a dedicated ICU/CCU with availability of short-term mechanical circulatory support.	I	C	
In patients with cardiogenic shock complicating ACS an immediate coronary angiography is recommended (within 2 hours from hospital admission) with an intent to perform coronary revascularization.	I	C	
Continuous ECG and blood pressure monitoring are recommended.	I	C	
Invasive monitoring with an arterial line is recommended.	I	C	
Fluid challenge (saline or Ringer's lactate, >200 ml/15–30 min) is recommended as the first-line treatment if there is no sign of overt fluid overload.	I	C	
Intravenous inotropic agents (dobutamine) may be considered to increase cardiac output.	IIb	C	
Vasopressors (norepinephrine preferable over dopamine) may be considered if there is a need to maintain SBP in the presence of persistent hypoperfusion.	IIb	B	558
IABP is not routinely recommended in cardiogenic shock.	III	B	585, 586
Short-term mechanical circulatory support may be considered in refractory cardiogenic shock depending on patient age, comorbidities and neurological function.	IIb	C	

No in AMICS !

Impella (IABP)

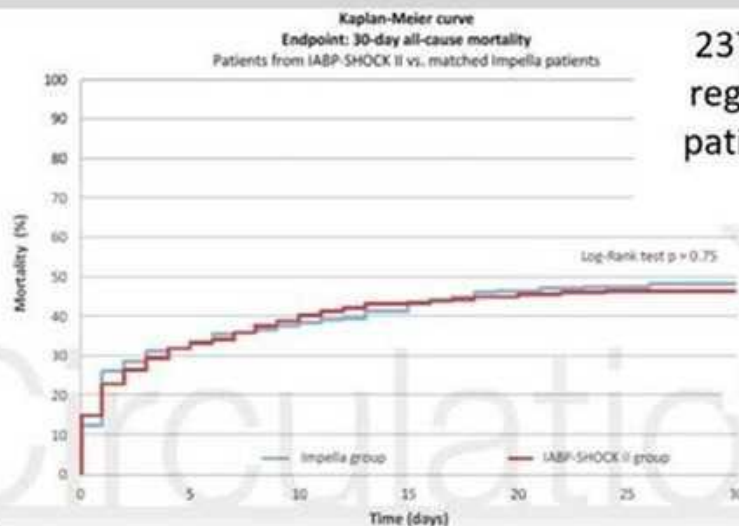
Pompe micro axiale à débit continu

- ✓ Implantation endovasculaire
- ✓ Position transvalvulaire aortique
- ✓ Aspire le sang du VG pour le réinjecter dans l'aorte
- ✓ Assure un débit cardiaque
- ✓ Diminue la pression capillaire (OAP)
- ✓ Diminue la tension pariétale
- ✓ Diminue le travail du VG



Impella (IABP)

Conflicting results in AMICS ...



237 patients EUROSHOCK registry matched with 237 patients from IABP Shock 2 study

High rate of complications and associated morbi-mortalities +++

Schrago.B et al, Circulation 2018

We need RCT !

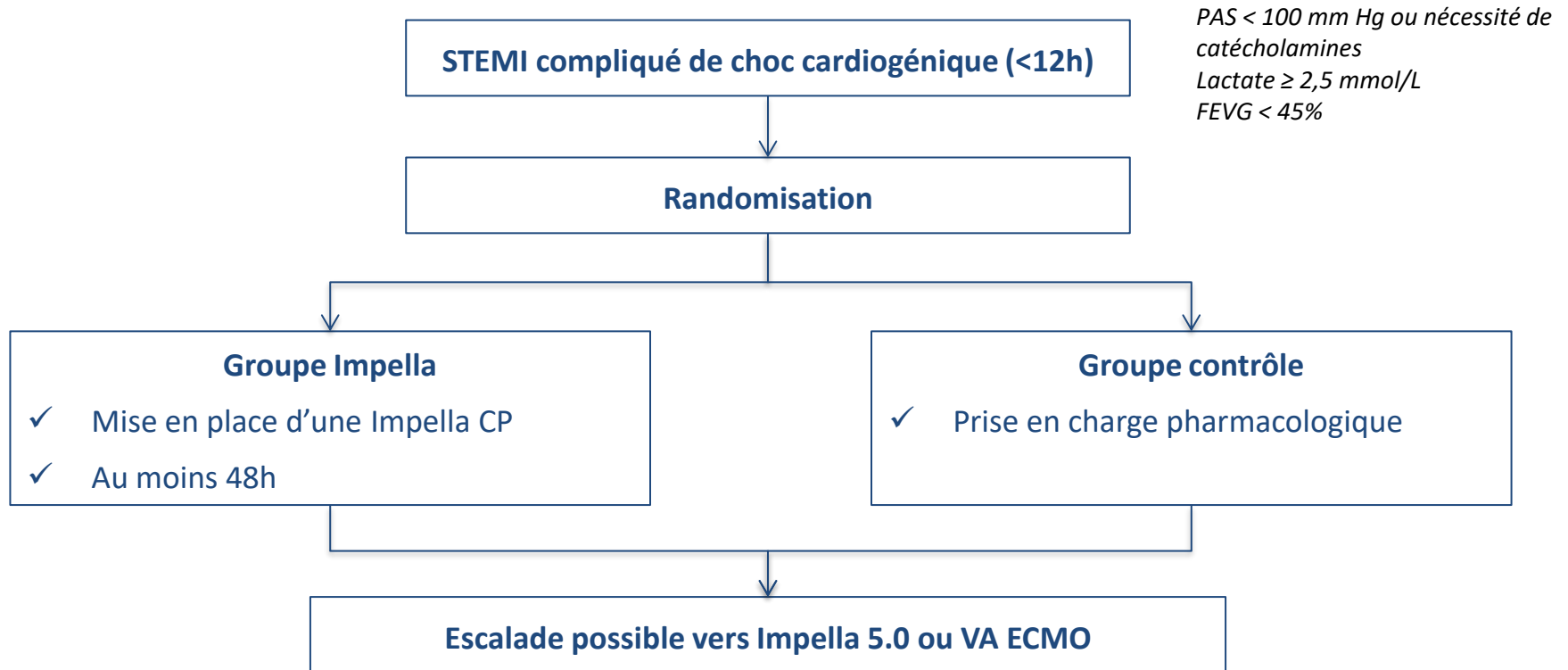
Figure 2. In-Hospital Outcomes Among Propensity-Matched Patients With Acute Myocardial Infarction Complicated by Cardiogenic Shock Undergoing Percutaneous Coronary Intervention With Intravascular Microaxial Left Ventricular Assist Device vs Intra-aortic Balloon Pump

	Intravascular Microaxial Left Ventricular Assist Device		Intra-aortic Balloon Pump		Absolute Risk Difference (95% CI), %	Favors Intravascular Microaxial Left Ventricular Assist Device	Favors Intra-aortic Balloon Pump	P Value
	No. of Patients	Patients, %	No. of Patients	Patients, %				
Overall (n = 1680 matched pairs)								
Mortality	756	45.0	573	34.1	10.9 (7.6-14.2)			<.001
Major bleeding	526	31.3	268	16.0	15.4 (12.5-18.2)			<.001
Device placement before initiation of percutaneous coronary intervention (n = 573 matched pairs)								
Mortality	261	45.5	211	36.8	8.7 (3.1-14.4)			.003
Major bleeding	157	27.4	95	16.6	10.8 (6.1-15.6)			<.001
Device placement after initiation of percutaneous coronary intervention (n = 662 matched pairs)								
Mortality	291	44.0	213	32.2	11.8 (6.6-17.0)			<.001
Major bleeding	228	34.4	104	15.7	18.7 (14.2-23.3)			<.001

Miller.PE JAMA Intern Med

Impella (IABP)

Étude DanGer Shock (N=360)



Critère de jugement principal : mortalité à 180 jours

Critère de jugement secondaire : escalade MCS ; mortalité ; HTx

Impella (IABP)

Étude DanGer Shock (N=360)

Réduction de la mortalité à 180 jours

45,8 versus 58,5% ; HR 0,74 (IC 95% 0,55-0,99) ; P=0,04

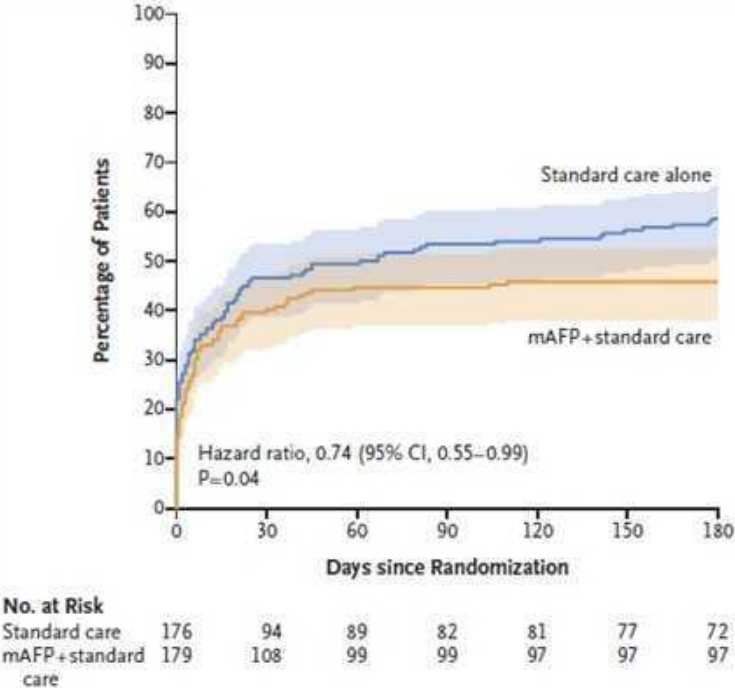
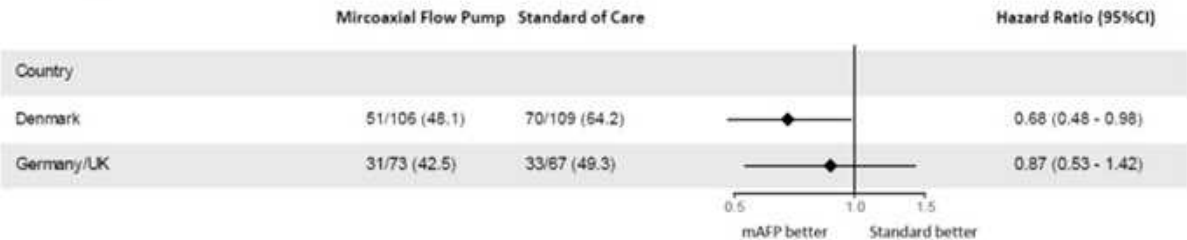
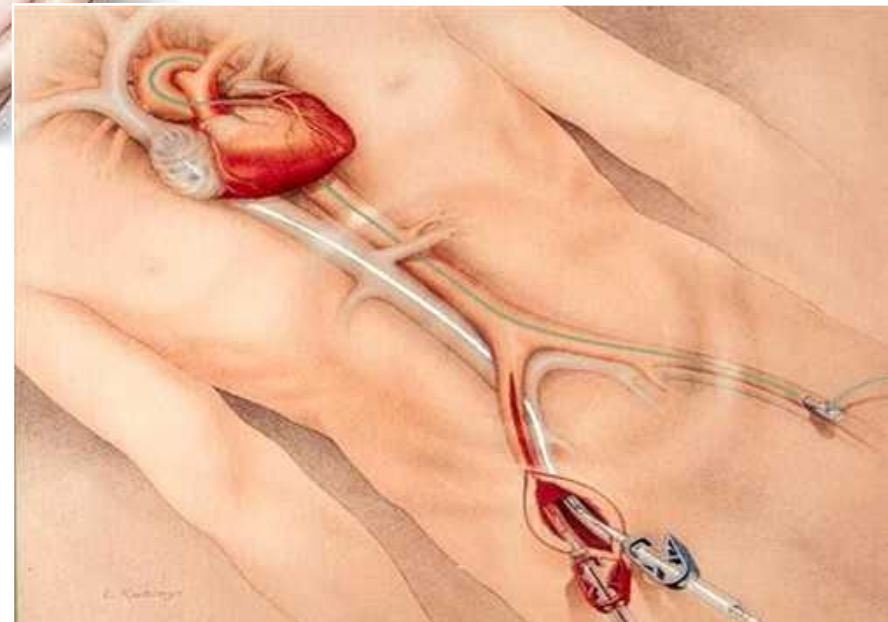
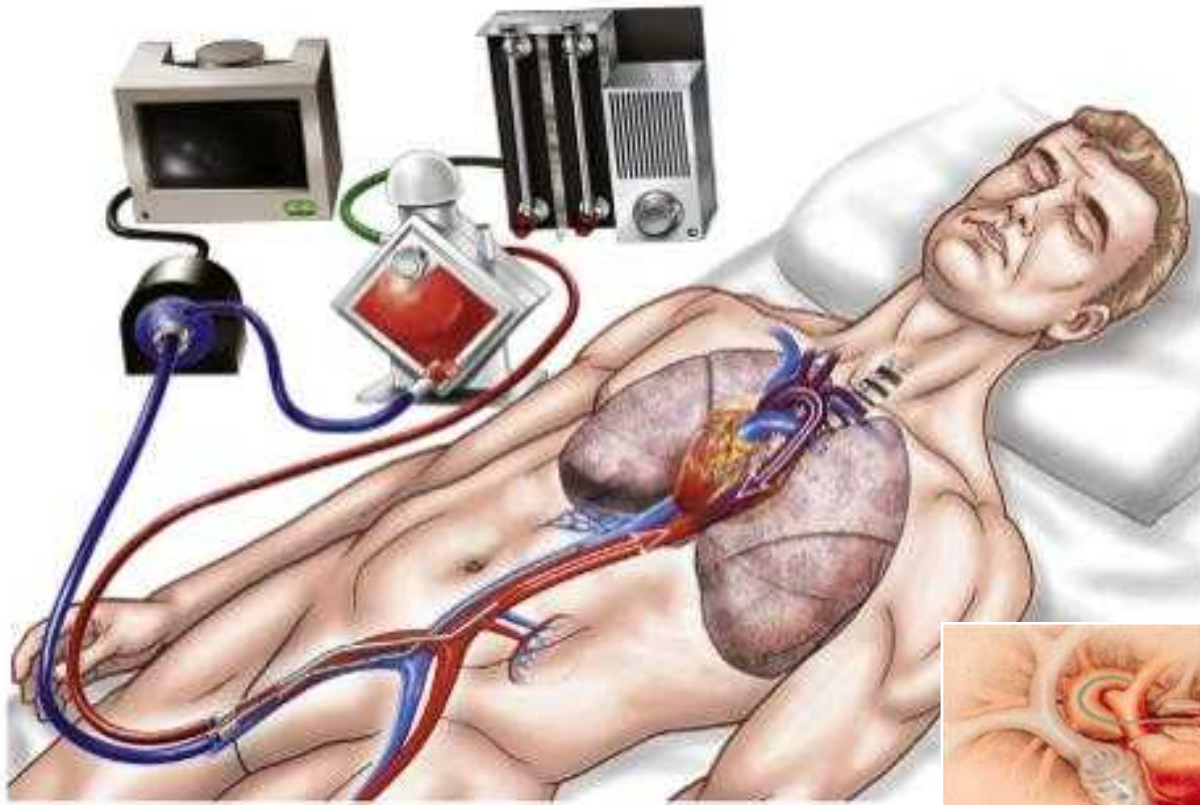


Table 3. End Points and Adverse Events in the Intention-to-Treat Population.*			
Event	Microaxial Flow Pump plus Standard Care (N=179)	Standard Care Alone (N=176)	Effect Size (95% CI)†
Primary end point: death from any cause at 180 days — no. (%)	82 (45.8)	103 (58.5)	0.74 (0.55 to 0.99)‡
Secondary end point			
Composite cardiac end point — no. (%)§	94 (52.5)	112 (63.6)	0.72 (0.55 to 0.95)
No. of days alive and out of the hospital (range)¶	82 (0 to 177)	73 (0 to 179)	8 (–8 to 25)
Adverse events			
Composite safety end point — no. (%)	43 (24.0)	11 (6.2)	4.74 (2.36 to 9.55)
Moderate or severe bleeding — no. (%)**	39 (21.8)	21 (11.9)	2.06 (1.15 to 3.66)
Limb ischemia — no. (%)	10 (5.6)	2 (1.1)	5.15 (1.11 to 23.84)
Renal-replacement therapy — no. (%)	75 (41.9)	47 (26.7)	1.98 (1.27 to 3.09)
Stroke — no. (%)	7 (3.9)	4 (2.3)	1.75 (0.50 to 6.01)
Cardioversion after ventricular tachycardia or fibrillation — no. (%)	59 (33.0)	52 (29.5)	1.17 (0.75 to 1.83)
Sepsis with positive blood culture†† — no. (%)	21 (11.7)	8 (4.5)	2.79 (1.20 to 6.48)

Subgroup Analysis of Death from Any Cause at 180 Days According to Country of Enrollment



Extracorporeal Membrane Oxygenation (ECMO, ECLS)



INDICATIONS DE L'ECLS ?

1. Arrêt cardiaque réfractaire (IH / EH)
2. Intoxication aiguë
3. Hypothermie accidentelle
4. Post cardiectomie (bloc CCV)
5. Myocardites (fulminantes)
6. Infarctus du myocarde
7. Cardiomyopathie dilatée terminale
8. Mycardiopathie aiguë du péri-partum
9. Sidération myocardique
10. Troubles malins du rythme ventriculaire
11. Embolie pulmonaire
12. Hyperkaliémie
13. Noyade
14. ECMO en pédiatrie





ELSEVIER



RESUSCITATION



elsevier.com/locate/resuscitation

Part 4: Advanced life support

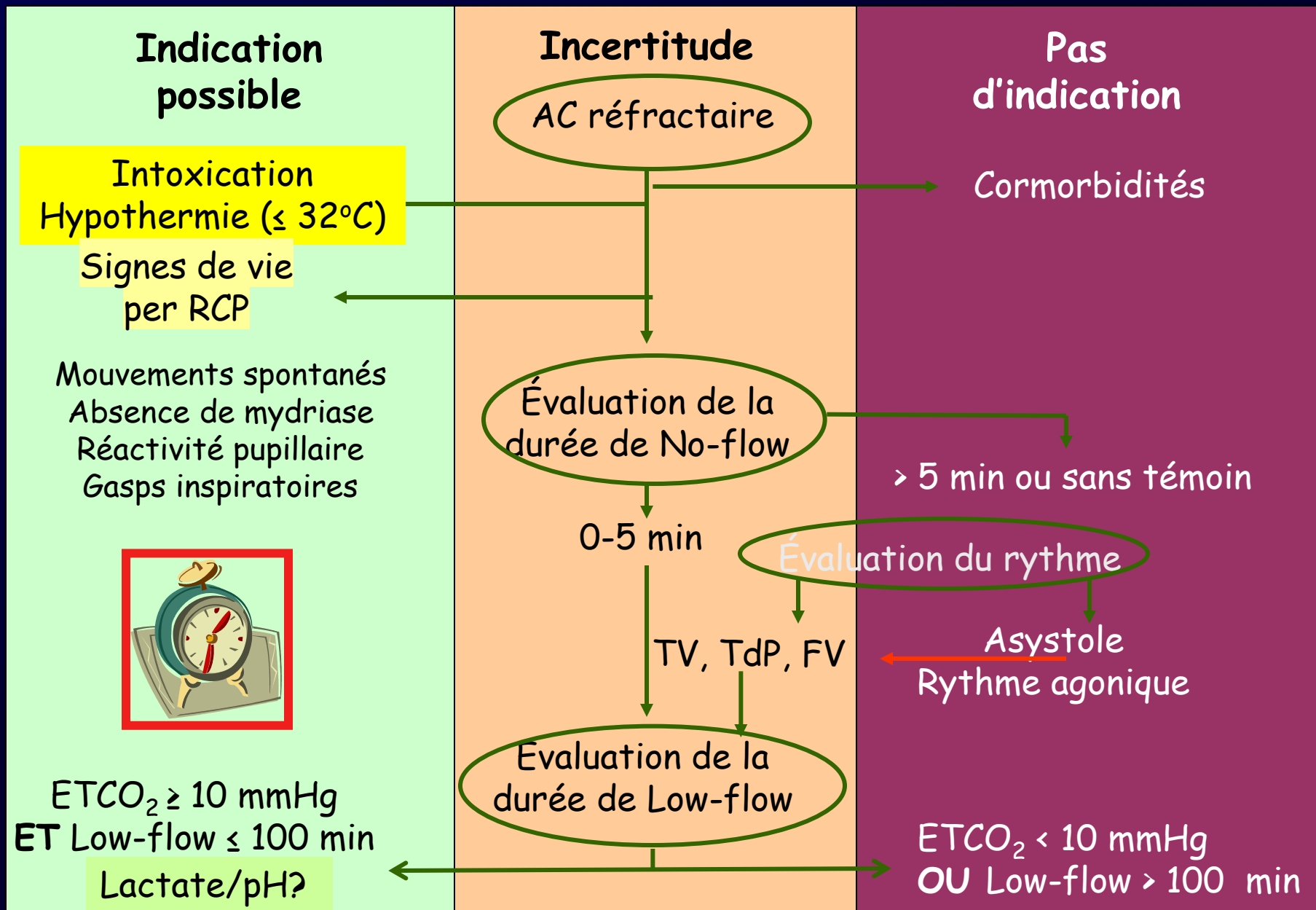
From 2005

International Liaison Committee on Resuscitation

Extracorporeal techniques and invasive
perfusion devices
W28,W82

Consensus on science. The only adult data come from three case series (LOE 5).^{323–325} One of these³²³ indicated that extracorporeal CPR (ECPR) was more successful in postcardiotomy patients than those in cardiac arrest from other causes. The other two studies^{324,325} suggested that ECPR is not beneficial for patients presenting to the emergency department in cardiac arrest with the exception of cardiac arrest associated with hypothermia or drug intoxication.

ECLS si ACEH réfractaire (French 2009 guidelines)



European Resuscitation Council Guidelines 2021: Adult advanced life support

To 2020/21

Consider extracorporeal CPR (eCPR) as a rescue therapy for selected patients with cardiac arrest when conventional ALS measures are failing or to facilitate specific interventions (e.g. coronary angiography and percutaneous coronary intervention (PCI), pulmonary thrombectomy for massive pulmonary embolism, rewarming after hypothermic cardiac arrest) in settings in which it can be implemented. **(weak recommendation, very low LOE)**

There is one recent small randomised controlled trial of eCPR for OHCA refractory VF cardiac arrest, and several others in progress. There is an urgent need for randomised studies of eCPR and large eCPR registries to identify the circumstances in which it works best, establish guidelines for its use and identify the benefits, costs and risks of eCPR

Yannopoulos, the ARREST trial, Lancet 2020

European Resuscitation Council Guidelines 2021: Adult advanced life support

To 2020/21

When to start ECPR?

(no agreed indications for which patients* and optimum time-point during ALS)

Commonly used criteria include:

- Witnessed cardiac arrest with bystander CPR.
- Time to establishing eCPR is less than 60 min from starting CPR.
- Younger patients (e.g. less than 65–70 years) and no major comorbidities precluding a return to independent life.
- Known or suspected treatable underlying cause of cardiac arrest.

*Consider ECPR in CA in special circumstances such as hypothermia, anaphylaxis, hyperkalemia, pulmonary embolism, coronary thrombosis, toxic agents, CA in operating room, cardiac surgery, catheterisation laboratory, drowning, pregnancy).

2023-24: années mitigées pour l'ECMO

THE NEW ENGLAND JOURNAL of MEDICINE

Extracorporeal Life Support in Infarct-Related Cardiogenic Shock

H. Thiele, U. Zeymer, I. Akin, M. Behnes, T. Rassaf, A.A. Mahabadi, R. Lehmann, I. Eitel, T. Graf, T. Seidler, A. Schuster, C. Skurk, D. Duerschmied, P. Clemmensen, M. Hennersdorf, S. Fichtlscherer, I. Voigt, M. Seyfarth, S. John, S. Ewen, A. Linke, E. Tigges, P. Nordbeck, L. Bruch, C. Jung, J. Franz, P. Lauten, T. Goslar, H.-J. Feistritz, J. Pösch, E. Kirchhof, T. Ouarrak, S. Schneider, S. Desch, and A. Freund, for the ECLS-SHOCK Investigators*

n=160

In patients with acute myocardial infarction complicated by cardiogenic shock with planned early revascularization, the risk of death from any cause at the 30-day follow-up was not lower among the patients who received ECLS therapy than among those who received medical therapy alone. (Funded by the Else Kröner Fresenius

THE NEW ENGLAND JOURNAL of MEDICINE

Early Extracorporeal CPR for Refractory Out-of-Hospital Cardiac Arrest

M.M. Suverein, T.S.R. Delnoij, R. Lorusso, G.J. Brandon Bravo Bruinsma, L. Otterspoor, C.V. Elzo Kraemer, A.P.J. Vlaar, J.J. van der Heijden, E. Scholten, C. den Uil, T. Jansen, B. van den Bogaard, M. Kuijpers, K.Y. Lam, J.M. Montero Cabezas, A.H.G. Driessen, S.Z.H. Rittersma, B.G. Heijnen, D. Dos Reis Miranda, G. Bleeker, J. de Metz, R.S. Hermanides, J. Lopez Matta, S. Eberli, D.W. Donker, R.J. van Thiel, S. Akin, O. van Meer, J. Henriques, K.C. Bokhoven, L. Mandigers, J.J.H. Bunge, M.E. Bol, B. Winkens, B. Essers, P.W. Weerwind, J.G. Maessen, and M.C.G. van de Poll

n=420

In patients with refractory out-of-hospital cardiac arrest, extracorporeal CPR and conventional CPR had similar effects on survival with a favorable neurologic outcome. (Funded by the Netherlands Organization for Health Research and Develop-

RESUSCITATION 182 (2023) 109665

Review

Extracorporeal cardiopulmonary resuscitation for cardiac arrest: An updated systematic review

Mathias J. Holmberg^{a,b,*}, Asger Granfeldt^{b,c}, Anne-Marie Guerguerian^d, Claudio Sandroni^e, Cindy H. Hsu^f, Ryan M. Gardner^g, Peter C. Lind^{b,c}, Mark A. Eggertsen^b, Cecilie M. Johannsen^b, Lars W. Andersen^{b,c,h}

Conclusions: Recent randomized trials suggest potential benefit of ECPR, but the certainty of evidence remains low. It is unclear which patients might benefit from ECPR.

Refractory out-of-hospital cardiac arrest and extracorporeal cardiopulmonary resuscitation: A meta-analysis of randomized trials

Artificial
Organs WILEY

Tommaso Scquizzato¹ | Alessandra Bonaccorso¹ | Justyna Swol² |
Lorenzo Gamberini³ | Anna Mara Scandroglio¹ | Giovanni Landoni^{1,4} |
Alberto Zangrillo^{1,4}

Conclusions: Extracorporeal CPR compared with conventional CPR increased survival with favorable neurological outcome in adults with refractory out-of-hospital cardiac arrest, especially when the initial rhythm was shockable.

JAMA Insights

Extracorporeal Cardiopulmonary Resuscitation for Cardiac Arrest

Asger Granfeldt, MD, PhD, DMSc; Mathias J. Holmberg, MD, PhD, MPH; Lars W. Andersen, MD, MPH, PhD, DMSc.

ECPR may increase survival in patients with refractory cardiac arrest, who typically have mortality rates higher than 90%. However, ECPR is resource-intensive and additional research is needed to clarify the optimal timing of ECPR administration and to identify patients for ECPR who are most likely to benefit.

RCT de l'ECMO VA (SCA / CMD)



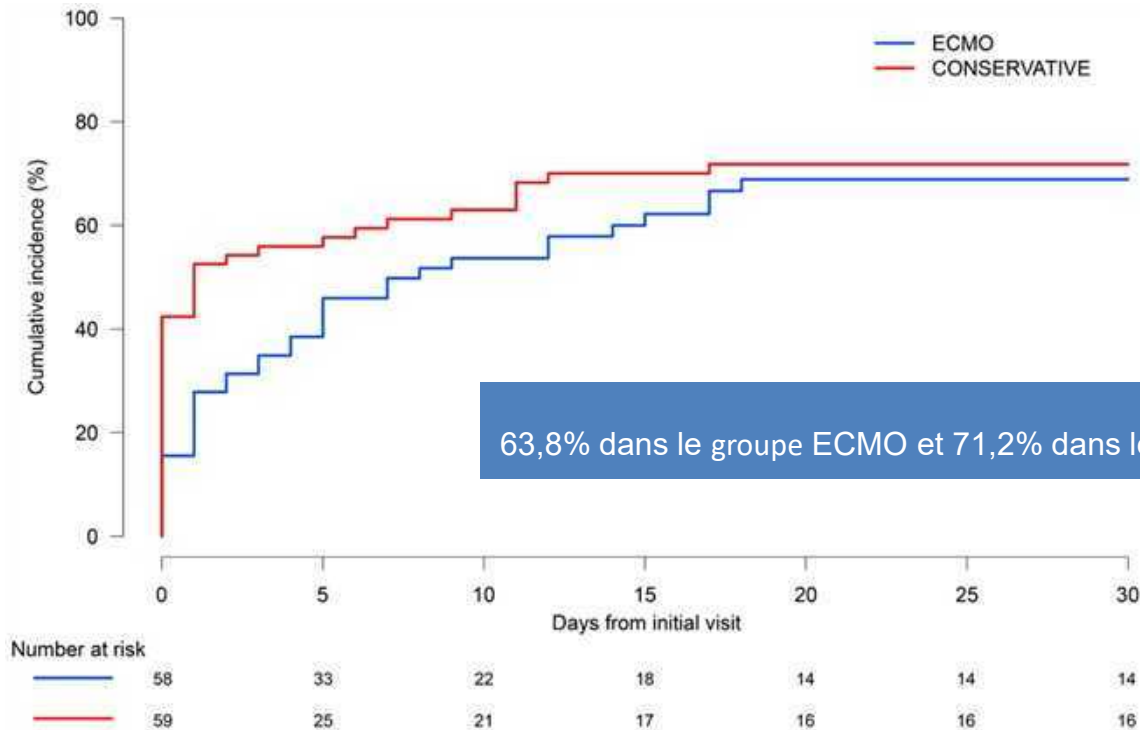
Étude ECMO-CS (N=117)

- ✓ Étude prospective multicentrique randomisée
- ✓ 4 centres en République Tchèque
- ✓ Critères d'inclusion
 - ✓ Choc cardiogénique en aggravation (SCAI D ou E)
 - ✓ Choc cardiogénique grave (SCAI D)
- ✓ Randomisation : ECMO VA immédiate ou traitement conservateur



Critère de jugement composite à 30 jours : décès, arrêt cardiaque récupéré, implantation d'une assistance circulatoire

RCT de l'ECMO VA (SCA / CMD)

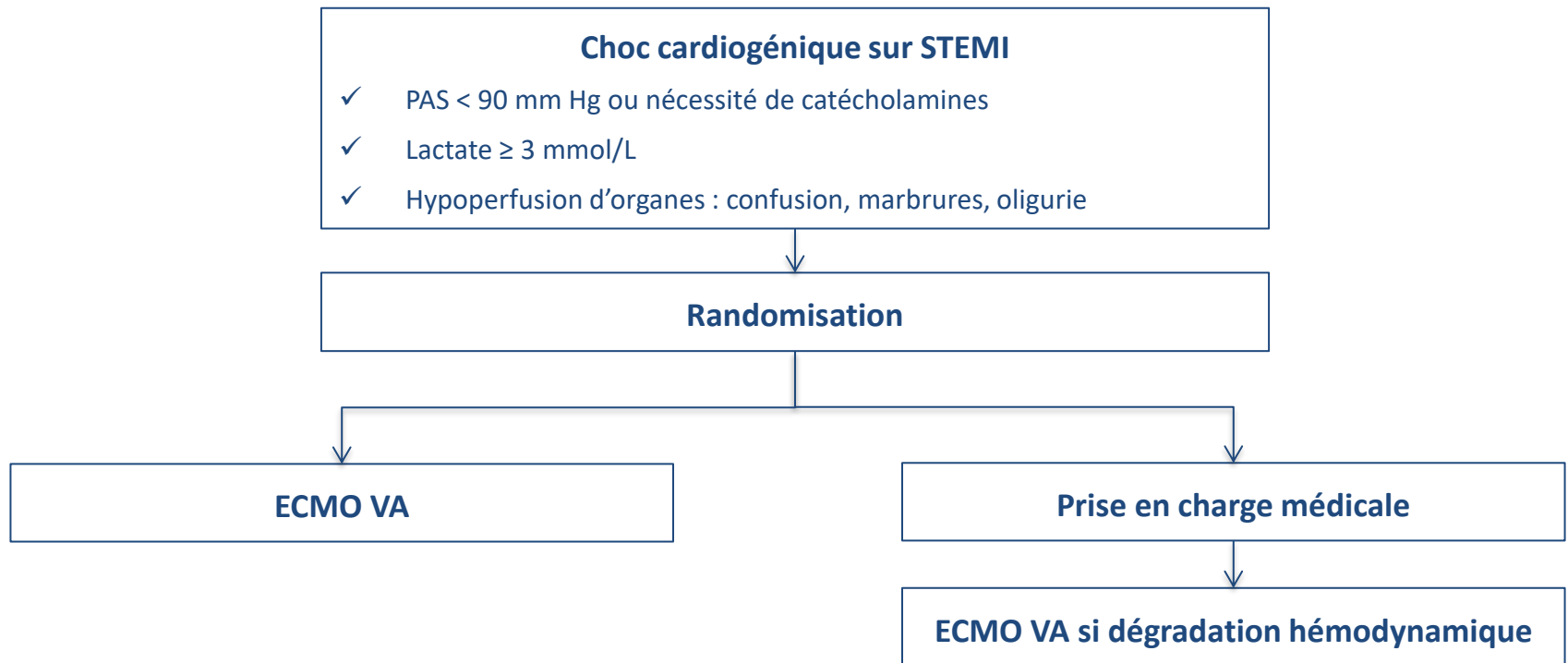


- ✓ 117 patients : 58 VA-ECMO et 59 sans VA-ECMO
- ✓ Sexe masculin 73,5%, âge 66 (59-73) ans ; Vasoactive-inotropic score 61 (30-124) ; lactate 5,0 mmol/L (3,2-8,0)
- ✓ Etiologie : 50,4% SCA ; 23,1% CMD
- ✓ 39% des patients contrôle ont été secondairement assistés

RCT de l'ECMO VA (STEMI + CS)



Étude ECLS-SHOCK (N=420)

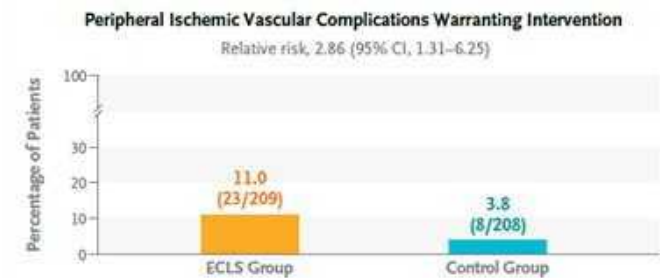
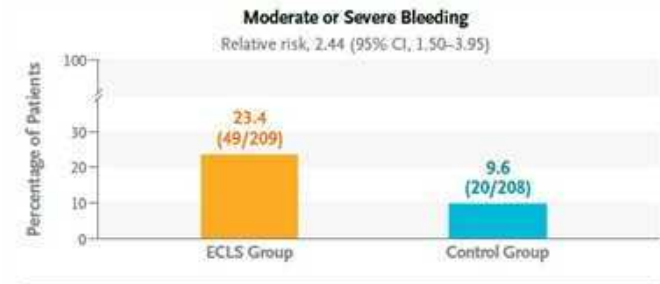
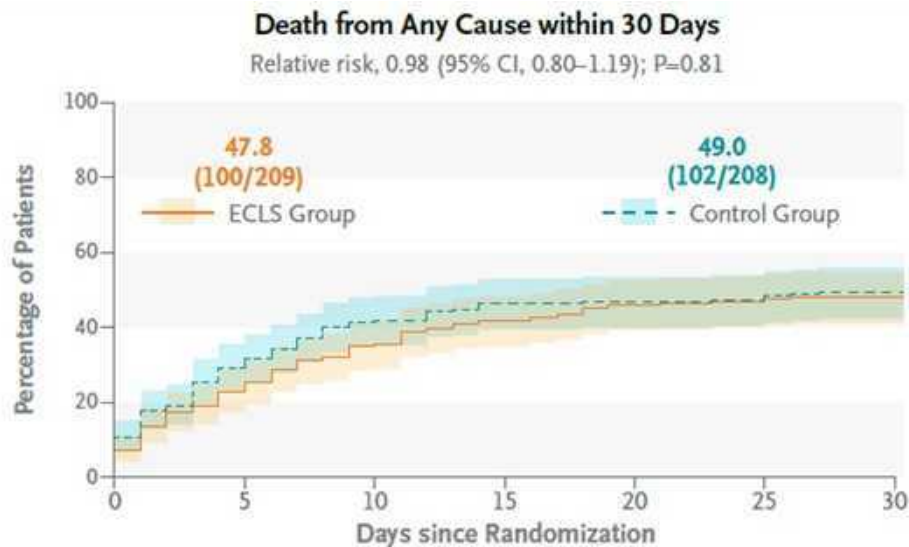


Critère de jugement principal : mortalité à 30 jours

Place de l'ECMO VA (STEMI + CS)



Étude ECLS-SHOCK (N=420)

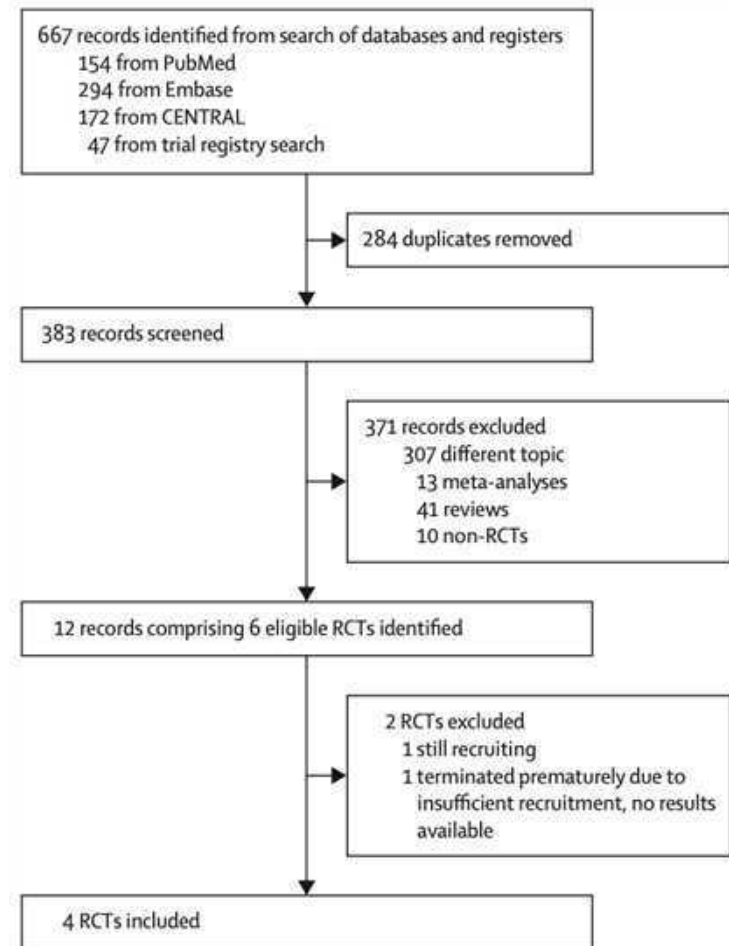


- ✓ N = 417 patients inclus entre 2019 et 2022 (44 centres)
- ✓ 77% des patients ont présenté un arrêt cardiaque récupéré (RCP < 45 minutes)
- ✓ Groupe contrôle : 26 patients implantés d'une ECMO (12,5%) et 28 d'une autre assistance
- ✓ Durée d'ECMO 2,7 jours



ECMO VA & choc cardiogénique ischémique : review

- ✓ Méta analyse basée sur les données patients individualisées
- ✓ **Choc cardiogénique post-infarctus**
- ✓ Critère d'inclusion : **RCTs évaluant l'implantation précoce de l'ECMO VA vs Traitement médical optimal**
- ✓ CJP : **Mortalité à J30 toute causes confondues**
- ✓ CJS : Saftery endpoints pendant 30 jours de suivis (hémorragie, ischémie, AVC)



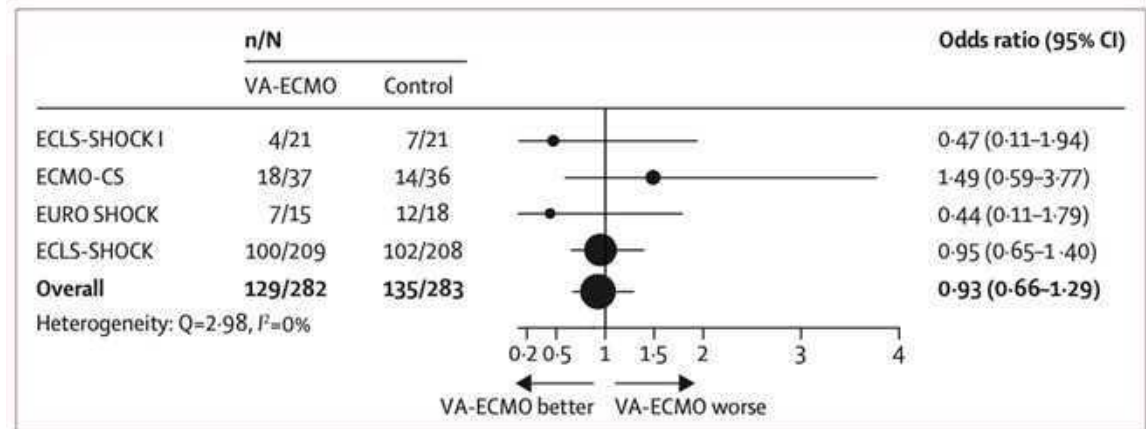
ECMO VA & choc cardiogénique ischémique : review

	Total (n=567)	VA-ECMO (n=284)	Control (n=283)	Effect size (95% CI)
Primary outcome				
All-cause death at day 30	264/565 (47%)	129/282 (46%)	135/283 (48%)	OR 0.93 (0.66-1.29)

Pas d'hétérogénéité entre les articles

Analyse post-hoc après exclusion des cross-over

- Control : **46% (115)**
- ECMO VA : **44% (115)**



ECMO VA & choc cardiogénique ischémique : review

Secondary outcomes				
Moderate or severe bleeding (BARC type 3–5) ¹⁷ within 30 days	104/565 (18%)	70/282 (25%)	34/283 (12%)	OR 2.44 (1.55–3.84)
Stroke within 30 days	19/565 (3%)	11/282 (4%)	8/283 (3%)	OR 1.41 (0.56–3.57)
Peripheral ischaemic vascular complication within 30 days	42/564 (7%)	32/281 (11%)	10/283 (4%)	OR 3.53 (1.70–7.34)
Sepsis within 30 days	87/532 (16%)	45/267 (17%)	42/265 (16%)	OR 1.08 (0.66–1.76)
Additional outcomes				
Poor neurological outcome (CPC 3 or 4) ¹⁸ in survivors	72/267 (27%)	38/134 (28%)	33/133 (25%)	OR 1.20 (0.70–2.07)
Length of intensive care treatment, days	9 (4–15) [n=537]	11 (5–17) [n=268]	8 (4–14) [n=269]	HLE 1.5 (0.0–3.0)
Length of hospital stay, days	12 (5–22) [n=556]	13 (5–22) [n=276]	11 (4–22) [n=280]	HLE 1.5 (0.0–3.0)

Categorical data are shown as n/N (%), where the denominator is the number of patients with valid data. Continuous data are shown as median (IQR) [number of patients with valid data]. Outcome data in the primary studies are shown in the appendix (p 10). The overall OR was calculated in the meta-analytic regression models. ORs were generated by using individual-level data. BARC=Bleeding Academic Research Consortium. CPC=Cerebral Performance Category. HLE=Hodges-Lehmann estimate. OR=odds ratio. VA-ECMO=venoarterial extracorporeal membrane oxygenation.

Table 3: Clinical outcomes at 30 days

Significativement plus de saignement et de complications ischémiques

ECMO VA & choc cardiogénique toxique

- La RCP doit être prolongée en cas d'AC d'origine toxique survenue devant témoin.
- L'ECLS doit être envisagée en cas d'AC ou de choc toxique réfractaire au traitement médical conventionnel optimal (incluant les fortes doses de catécholamines...).

Conférence d'Experts SRLF/SFAR 2006-2020



Quels patients doit-on assister ?



Ni trop tardif (AC) : risque d'encéphalopathie anoxique ou de défaillance multiviscérale

Ni trop facile (choc non réfractaire) : patients qui auraient guéri avec un traitement médical bien conduit = **choc cardiogénique prouvé** ($IC \leq 2,5$ l/min/m²) malgré antidotes/catécho fortes doses + défaillance d'organe persistante (pulmonaire, rénale)

Donc, CC: échec des ttt usuels + catécholamines croissantes

ECMO VA & choc cardiogénique toxique

Lavonas et al,
Circulation 2023

2023 American Heart Association Focused Update on the Management of Patients With Cardiac Arrest or Life-Threatening Toxicity Due to Poisoning: An Update to the American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care



Recommendations for the Use of VA-ECMO in Patients With Life-Threatening Poisoning		
COR	LOE	Recommendations
2a	C-LD	1. It is reasonable to use VA-ECMO for persistent cardiogenic shock or cardiac arrest due to poisoning that is not responsive to maximal treatment measures.
2a	C-LD	2. It is reasonable to use VA-ECMO for persistent dysrhythmias due to poisoning when other treatment measures fail.
2b	C-EO	3. The effectiveness of VA-ECMO for poisoned patients with cardiovascular collapse from causes other than cardiogenic shock has not been established.

CLASS 2a (MODERATE) Benefit >> Risk

LEVEL C-LD (Limited Data)

CLASS 2b (WEAK) Benefit ≥ Risk

LEVEL C-EO (Expert Opinion)

ECMO VA & choc cardiogénique

What ECLS results in CS ?

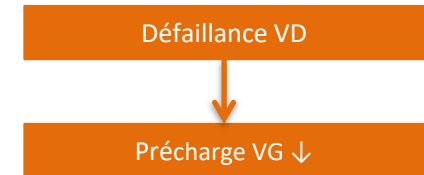
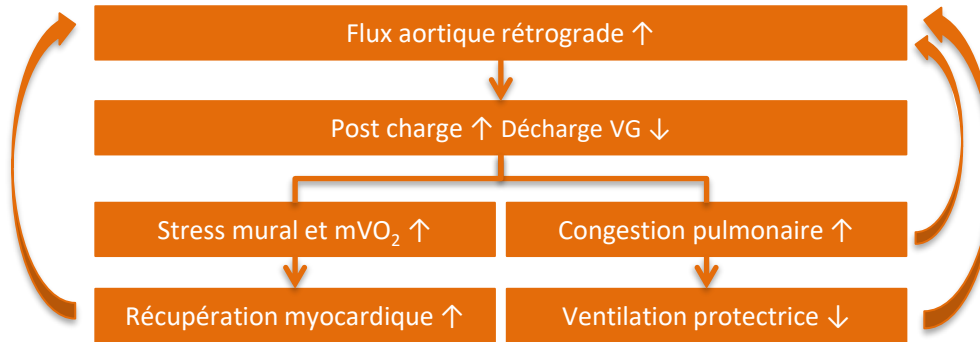
	Survival				
	Hospital (%)		5-Year (%)	HR (95% CI)	5-Year Conditional to Hospital Discharge (%)
PGF	>50%	73.3	57.3	0.30 (0.22-0.42)*	78.2
DCM		53.2	45.3	0.60 (0.45-0.80)*	85.2
Drug overdose		58.6	54.0	0.63 (0.32-1.24)	92.2
Arrhythmic storm		51.6	50.0	0.73 (0.40-1.31)	96.9
Massive PE	30-50%	46.8	38.3	0.93 (0.58-1.50)	81.2
Sepsis-induced CS		44.4	42.4	1.04 (0.61-1.77)	95.5
Fulminant myocarditis		37.9	32.9	0.65 (0.40-1.06)	86.8
AMI		37.3	31.5	1.05 (0.83-1.33)	84.4
Postcardiotomy excluding PGF		34.6	33.3	1.09 (0.89-1.42)	96.2
Refractory vasoplegia shock		11.1	0.0	3.92 (1.92-7.99)*	0.0
Other/unknown etiology	<30%	25.7	22.8	1.64 (1.25-2.14)*	88.7

1253 patients implanted between 2015-2018 at La Pitié University Hospital

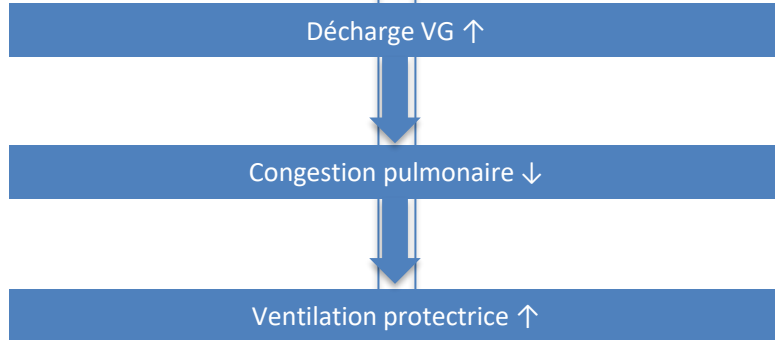
Survival differs between indication +/- previous CA or ongoing CPR

5-year survival is generally stable after hospital survival

ECMO périphérique fémoro-fémorale



ECMELLA (ECMO + Impella)

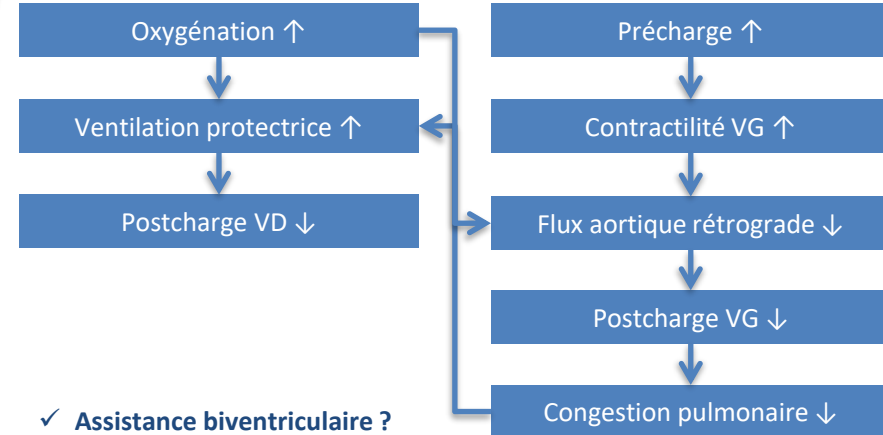


- ✓ Assistance mono gauche
- ✓ Évaluation de la fonction VD
- ✓ Transports difficiles
- ✓ Décubitus ventral ?
- ✓ Coût +++

ECMULSION (ECMO + CPIA)

- ✓ Pas d'assistance VD
- ✓ Transports difficiles
- ✓ Rapide et économique

ECMO VAV



- ✓ Assistance biventriculaire ?
- ✓ Invasif
- ✓ Dispositif unique
- ✓ Rapide et économique

JACC 2025

Mechanical Circulatory Support in Acute Myocardial Infarction-Cardiogenic Shock

2025 Acute Coronary Syndrome Guideline in Context

FIGURE 1 Guideline Changes for Use of MCS in MI With CS

Karthik Murugiah, MBBS, MHS,^{a,b} Theresa A. McDonagh, MD,^c David J. Cohen, MD, MSc,^{d,e} Sanket S. Dhruva, MD, MHS^{f,g,h}

Guideline Changes for use of Mechanical Circulatory Support in Myocardial Infarction With Cardiogenic Shock

	2013 STEMI	2014 NSTEMI	2025 ACS
IABP	2a (Is reasonable)	-	3 (Not recommended)
VA-ECMO			
mAFP			



VA-ECMO



mAFP



ACC/AHA guide

DanGer Shock

Inclusion Criteria



+



+



+



6

VA-ECMO: NO BENEFIT TO EARLY USE

after revascularization) need

Exclusion Criteria

mAFP: UPGRADE IN SELECTED PATIENTS WITH STEMI

GCS <8
after ROSC

precluding
mAFP

expectancy
<1 year

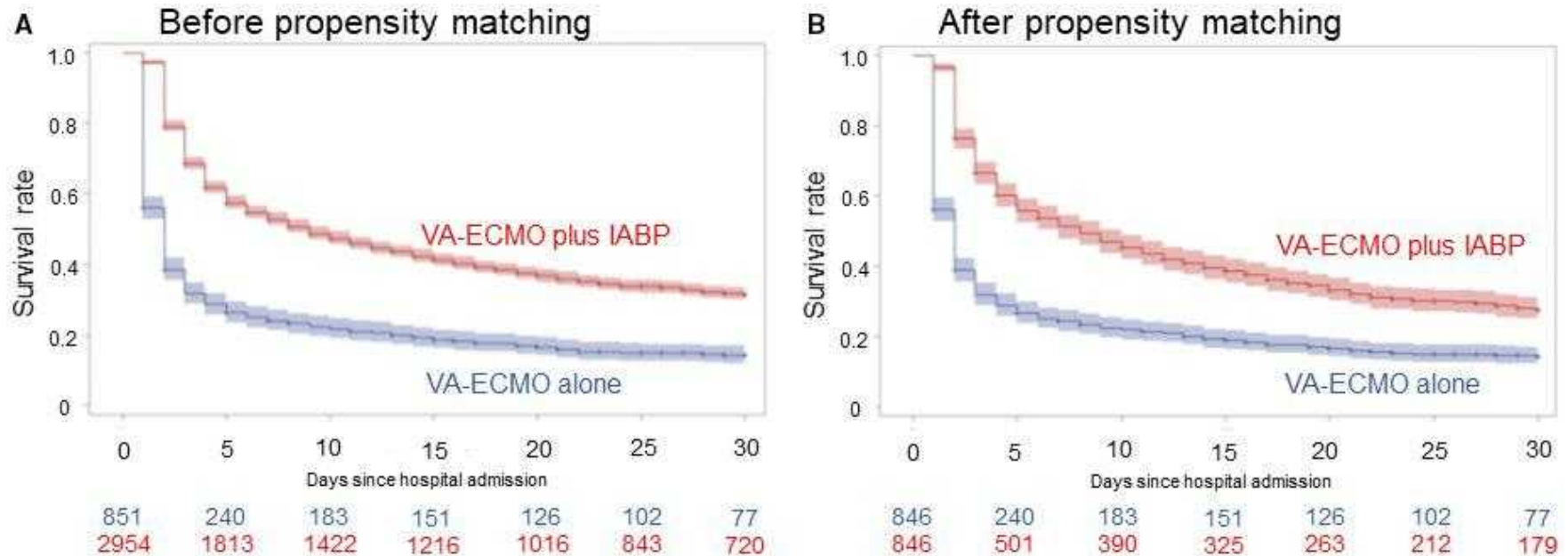
complications
of AMI

Severe aortic
abnormalities
LV thrombus

Unanswered questions and future direction:

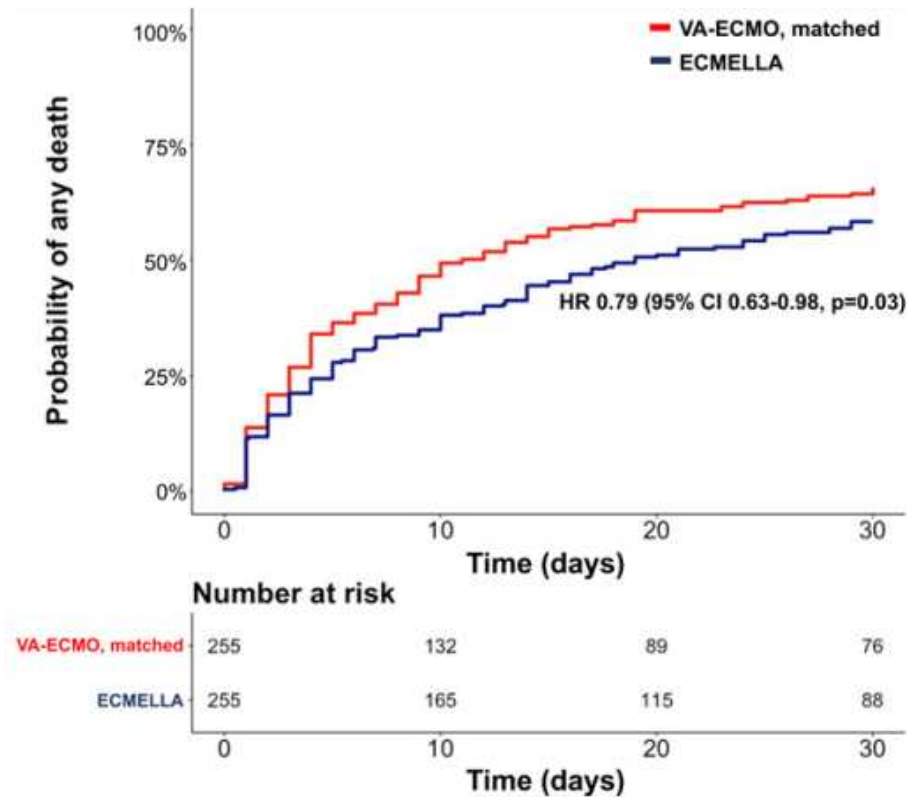
- Would VA-ECMO or IABP have benefit if applied to DanGer Shock-like patients?
- When and how to best use combined MCS modalities in managing CS?
- Physicians and critical care teams need expertise in mAFP management to mitigate risks and optimize benefit.
- Implementation-based research needed to evaluate real-world outcomes and develop best practices.

ECMO + ballon de contre-pulsion intra aortique



- ✓ N = 3815 patients avec choc cardiogénique ischémique assistés par ECMO VA
- ✓ 2964 patients (77.7%) ECMO VA + IABP versus 851 (22.3%) ECMO VA
- ✓ Amélioration de la survie à 7 jours et à 30 jours dans le groupe ECMO VA + IABP

ECMELLA ?



Amélioration de la survie dans le groupe
ECMELLA

Complications plus fréquentes :

- Saignement majeur
- Ischémie de membre
- Syndrome du compartiment abdominal
- AKI

- ✓ 686 patients en choc cardiogénique et assistés par ECMO VA
- ✓ Décharge VG avec IMPELLA (ECMELLA) chez 49% des patients
- ✓ Appariement 255 patients ECMO et 255 patients ECMELLA

Choc cardiogénique et régulation : centres CC

Cardiac Shock Care Centers

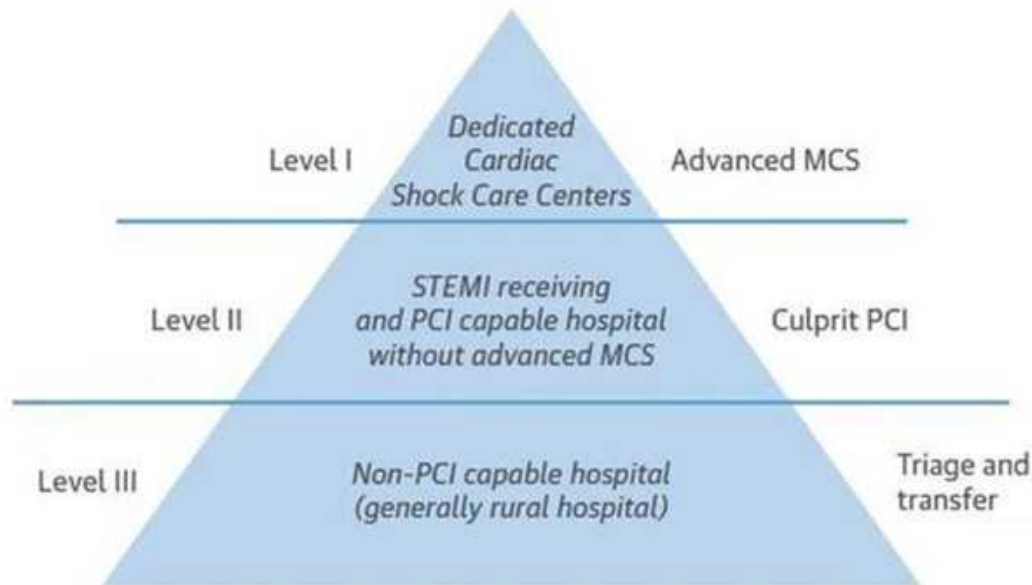


JACC Review Topic of the Week

J Am Coll Cardiol 2018

Tanveer Rab, MD,^a Supawat Ratanapo, MD,^a Karl B. Kern, MD,^b Mir Babar Basir, DO,^c Michael McDaniel, MD,^a Perwaiz Meraj, MD,^d Spencer B. King III, MD,^a William O'Neill, MD^c

FIGURE 2 Levels of Cardiac Shock Care



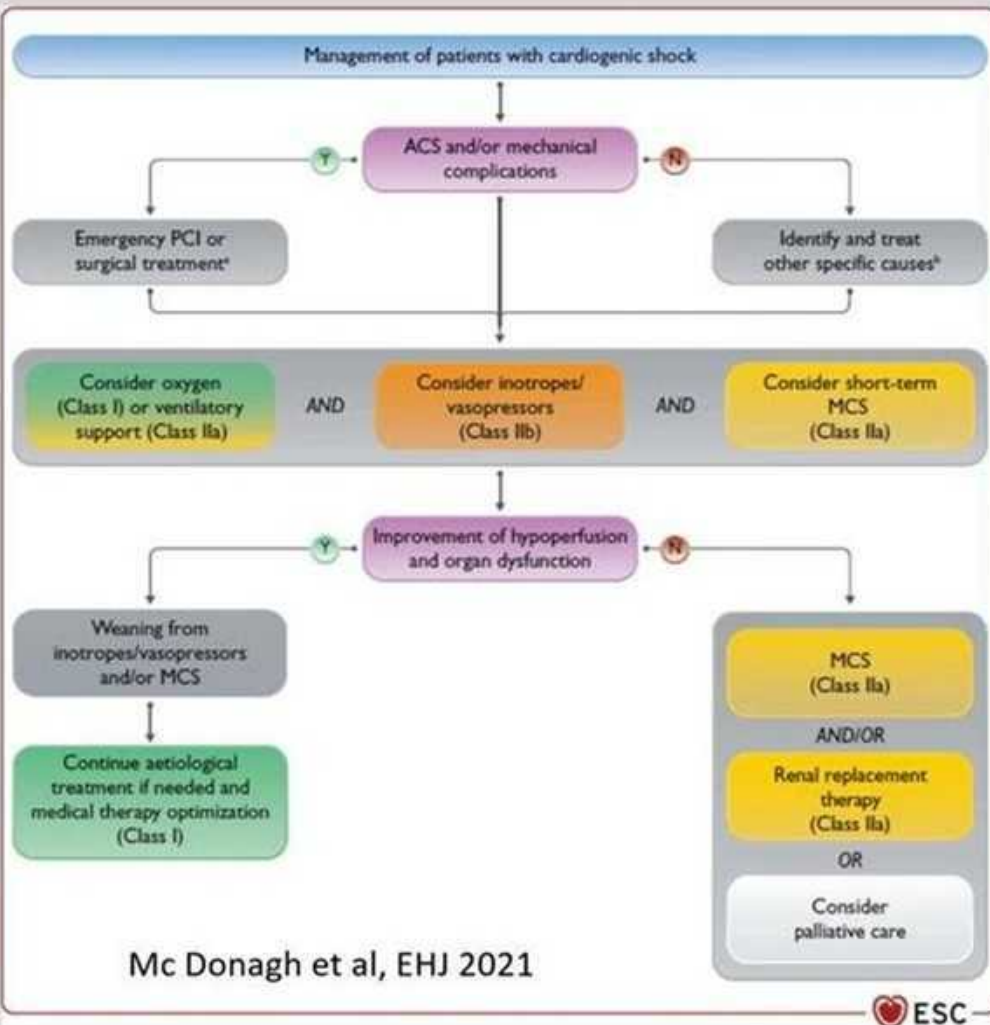
- **Multidisciplinary team:**
 - Intervantional cardiologist
 - Critical care specialist
 - HF specialist
 - Cardiothoracic surgeon
- **PCI on-site**
- **Advanced MCS**
- **Cardiothoracic surgery**

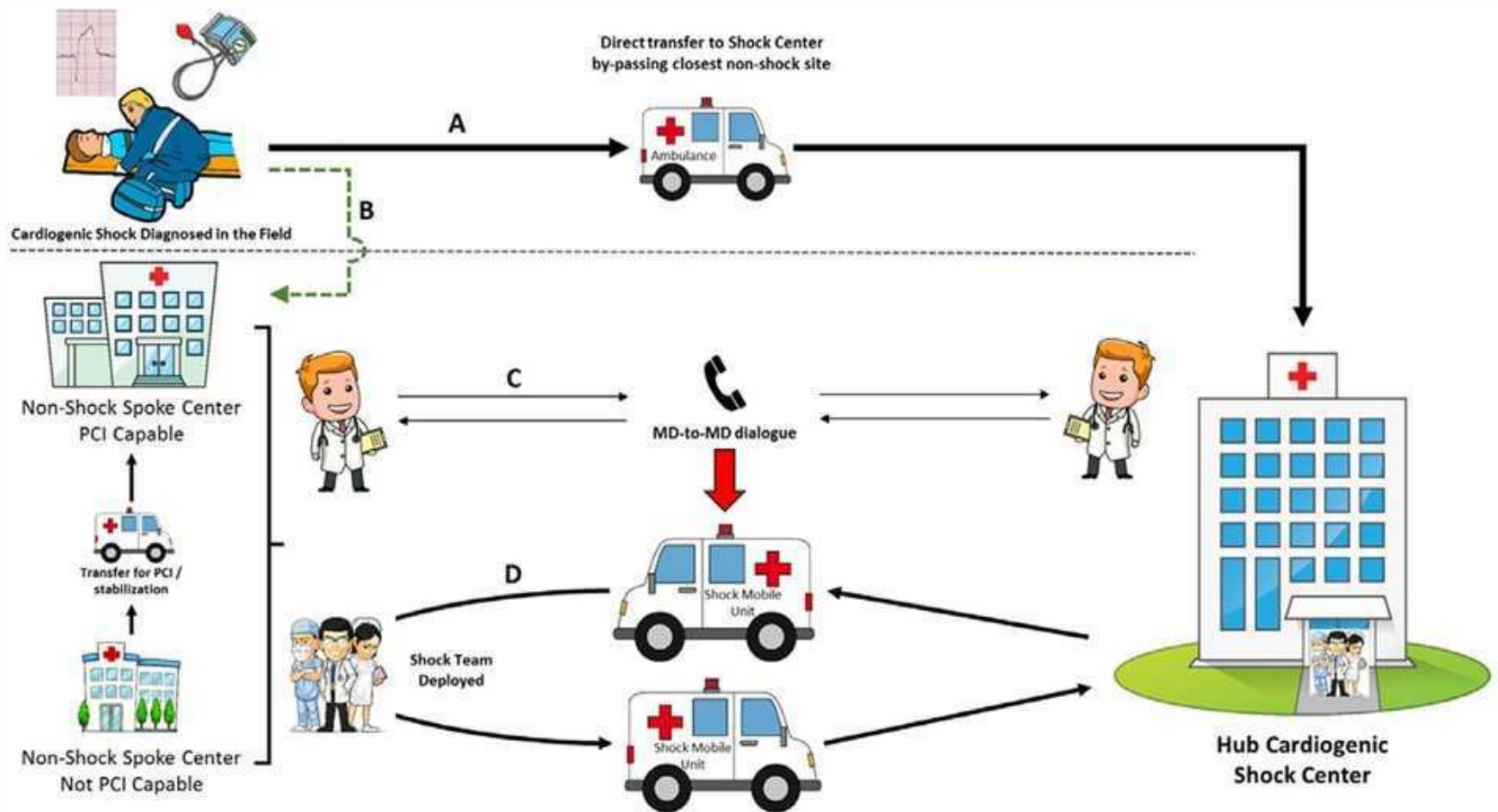
Available 24h/24 and 7days/7

Choc cardiogénique et régulation : protocole

CS management

- **Need a rapid and multiparametric diagnosis (clinic +++)**
- **Management:**
 - Early etiological management
 - Fluid management (diuretics vs fluid challenge)
 - Inotropes (Dobutamin) and Vasopressors (Norepinephrin)
 - aMCS if necessary
- **Local and regional protocols +++**
- **Place of Network and CS Heart team**





(A) A patient with CS diagnosed in the field by EMS can be transported directly to the hub CS center, bypassing the nearest spoke facility. (B) CS pathogenesis, travel time, and spoke center capabilities should factor into the decision to bypass spoke hospitals; STEMI patients can be transferred to a PCI facility for revascularization and stabilization. Patients with unclassified shock should be transferred to the nearest emergency department. (C) For patients presenting to spoke PCI-capable hospitals, revascularization and stabilization can be initiated. Physician-to-physician dialogue with the hub center shock team should occur as soon as possible. (D) A mobile unit from the hub center can be deployed to the spoke hospital to stabilize and initiate transfer to the hub CS center for definitive management. Patients presenting to smaller spoke centers without PCI capabilities should be immediately transferred to the nearest PCI facility, or a shock mobile unit should be requested from the hub CS center, depending on the patient's clinical status and anticipated travel time. CS indicates cardiogenic shock; EMS, emergency medical services; MD, medical doctor; PCI, percutaneous coronary intervention; and STEMI, ST-elevation myocardial infarction.

Figure 4 The systems of care for patients with cardiogenic shock (CS). A model for minimizing time delays and optimizing care has recently been proposed by the American Heart Association, where a network between several satellite centres and a central 'CS centre' exists to facilitate optimal care coordination. The core centre (first level) should be a dedicated CS centre, with expertise in the use of invasive haemodynamics and advanced mechanical circulatory support (MCS), and should be linked with multiple satellite centres [third level triage hospitals or second level percutaneous coronary intervention (PCI) capable centres]. Patients should be transported to the nearest hospital capable of performing 24/7 PCI and intensive care unit/cardiac care unit availability in order to stabilize haemodynamics (type II centre). 'Refractory' CS patients needing MCS will be directed to a higher level of care (type I CS centre). The patient should be hospitalized in an intensive care unit/cardiac care unit depending on hospital availability, and followed by physicians experienced in cardiovascular procedures. CS centres should also be able to provide safe transfer by a mobile extracorporeal membrane oxygenation (ECMO) team (out-of-hospital to hospital or inter-centre transfer), which is a feasible and effective strategy in selected patients. Patients that recover and stabilize should be discharged home or directed to rehabilitation or palliative care centres, depending on the needs. biVAD, biventricular assist device; LVAD, left ventricular assist device.

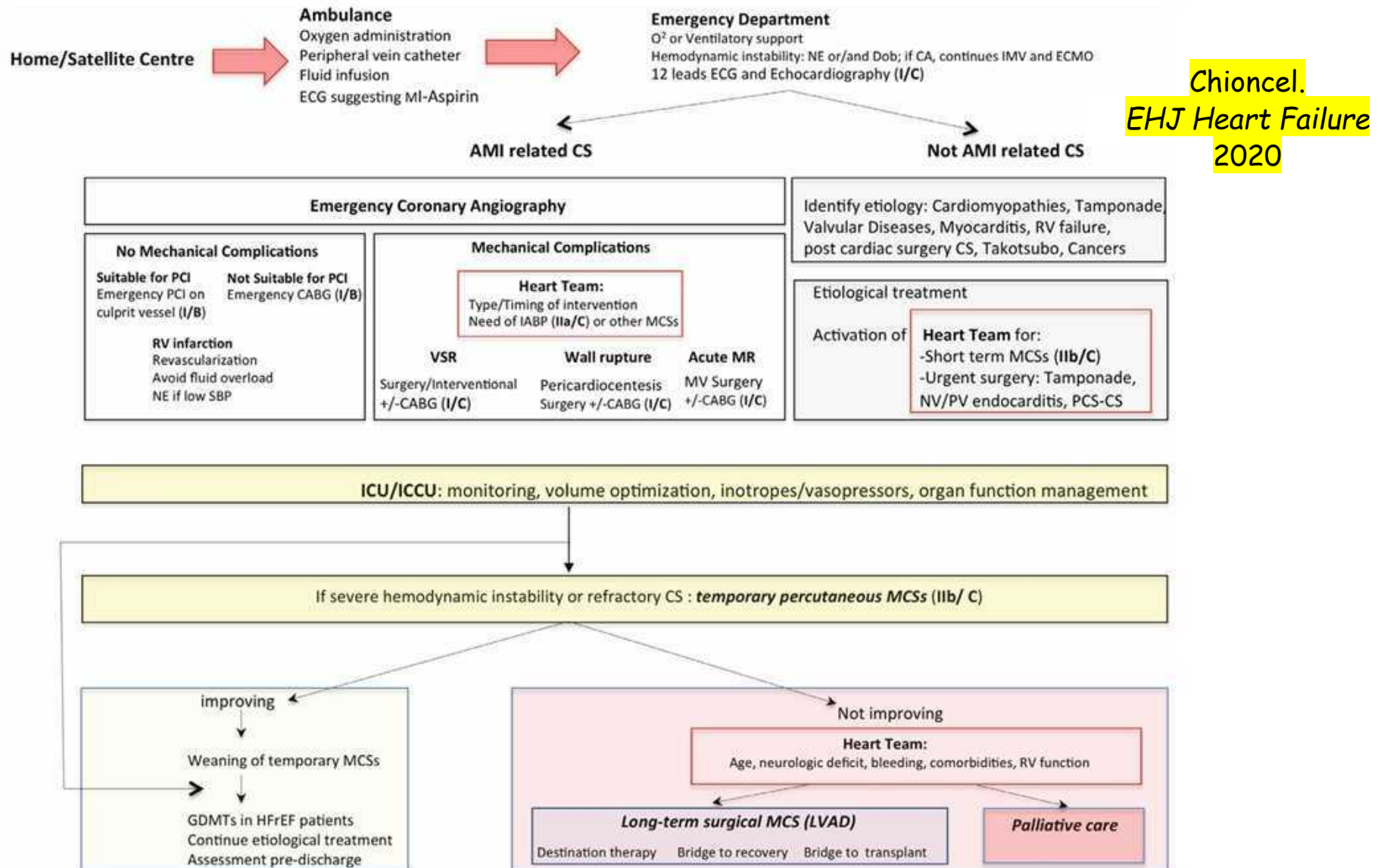


Figure 5 The algorithm for pre- and in-hospital management of patients with cardiogenic shock (CS). The level of decision by multidisciplinary heart team is presented in red rectangles. AMI, acute myocardial infarction; CA, cardiac arrest; CABG, coronary artery bypass graft; Dob, dobutamine; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; GDMT, guideline-directed medical therapy; HFrEF, heart failure with reduced ejection fraction; IABP, intra-aortic balloon pump; ICU, intensive cardiac care unit; ICCU, intensive care unit; IMV, invasive mechanical ventilation; LVAD, left ventricular assist device; MCS, mechanical circulatory support; MI, myocardial infarction; MR, mitral regurgitation; MV, mitral valve; NE, noradrenaline; NV, native valve; PCI, percutaneous coronary intervention; PCS, post-cardiac surgery; PV, prosthetic valve; RV, right ventricle; SBP, systolic blood pressure; VSR, ventricular septal rupture.

Conclusion

- ✓ **Épidémiologie : Incidence en augmentation mais mortalité en baisse**
- ✓ **Prise en charge étiologique et stratification**
- ✓ **Prise en charge pharmacologique : dobutamine et noradrénaline**
- ✓ **Prise en charge non pharmacologique**
 - ✓ Désobstruction coronaire (SCA)
- ✓ **Discussion collégiale d'une assistance mécanique**
 - ✓ En cas de bas débit ou d'instabilité hémodynamique persistant
 - ✓ Intoxication et hypothermie et pathologies réversibles