LE CHOC CARDIOGENIQUE DANS L'URGENCE

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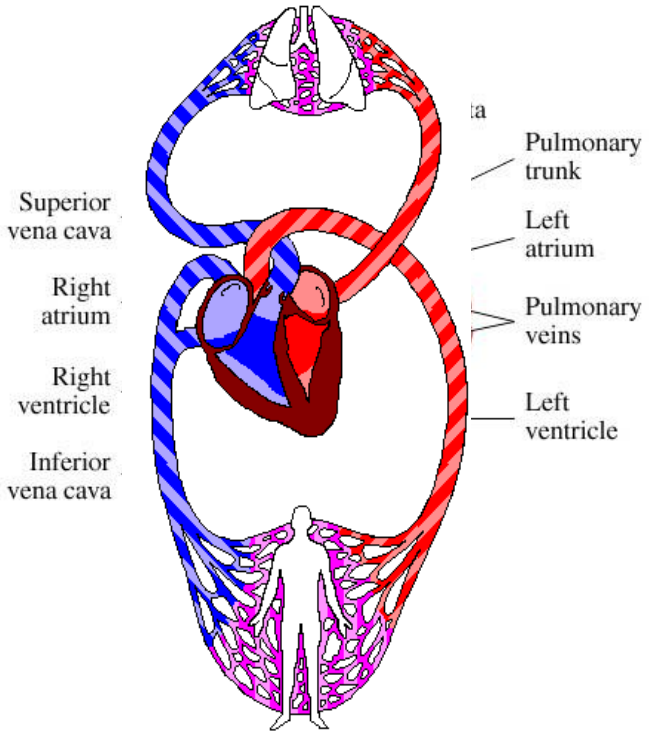
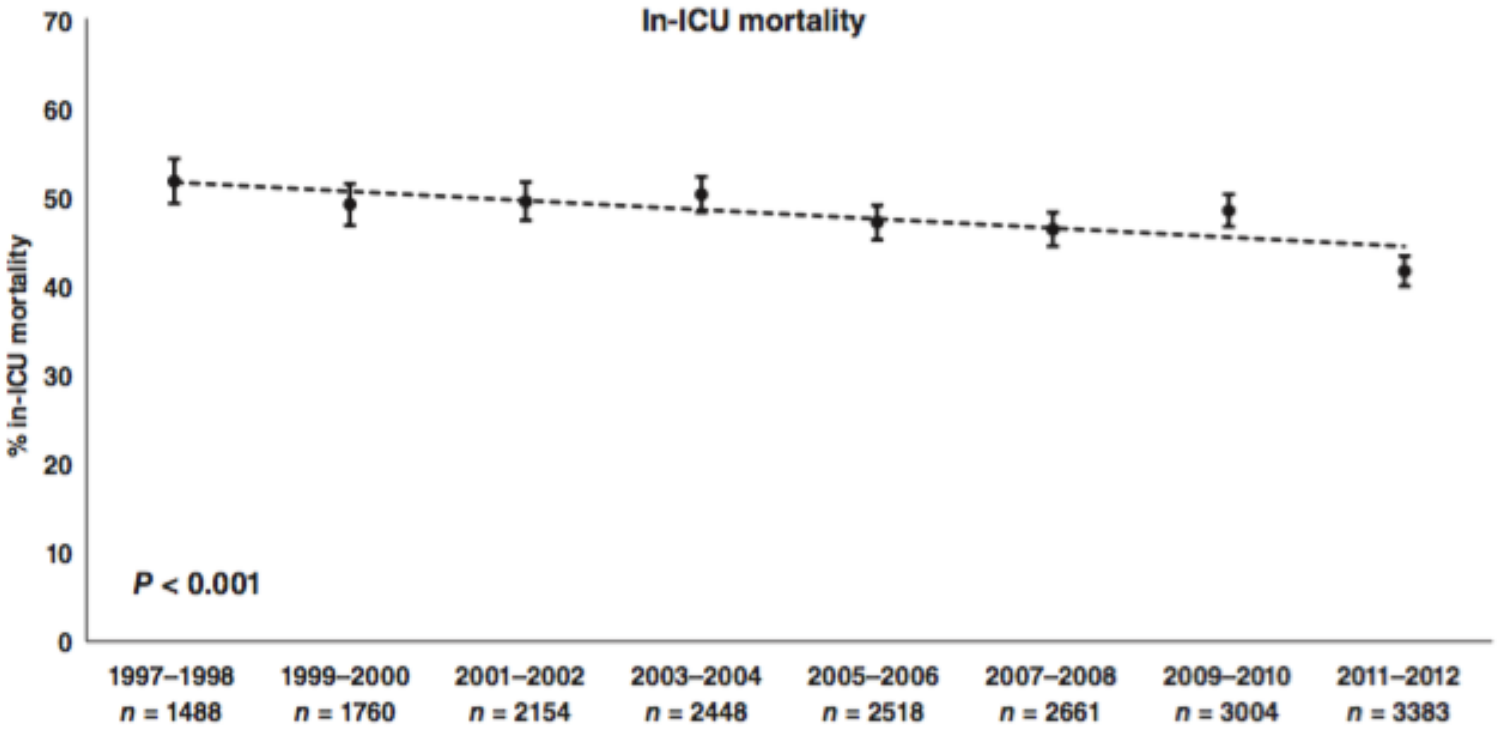


Pas de conflit d'intérêt pour cette présentation
(past congress and travel fees for Bard and Zoll: cardiac arrest)

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%**

Puymirat. *Eur J Heart Failure* 2016

Épidémiologie du choc cardiogénique

Circulation: Cardiovascular Interventions

ORIGINAL ARTICLE

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

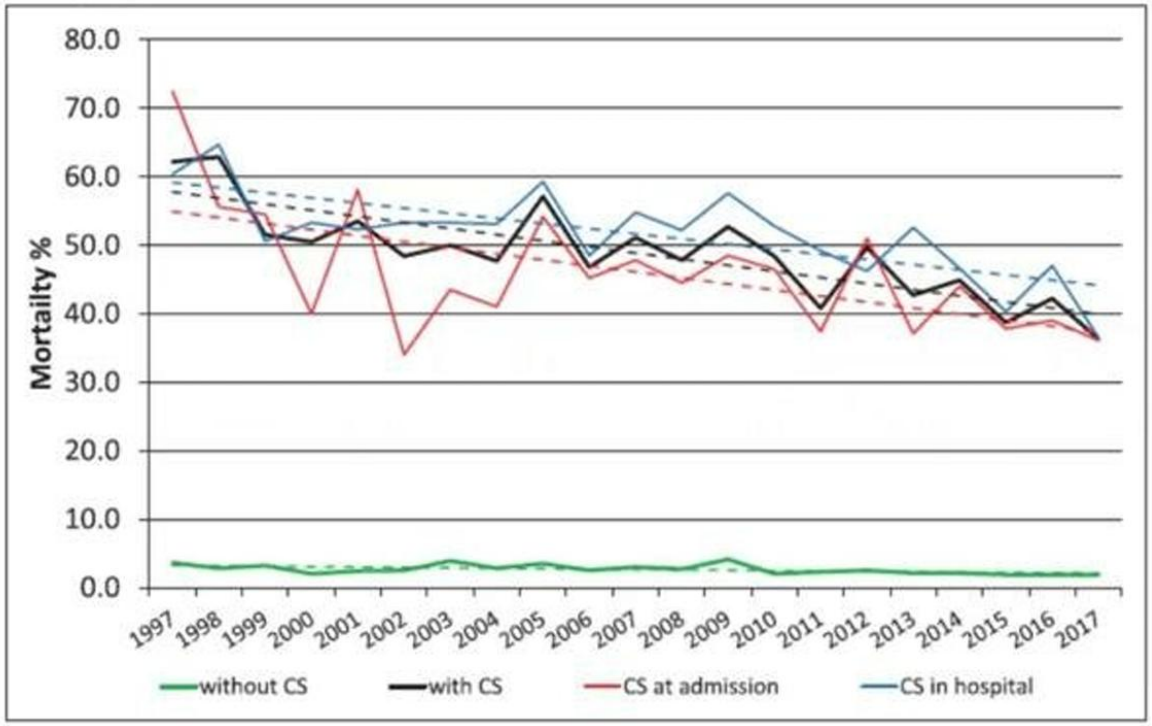
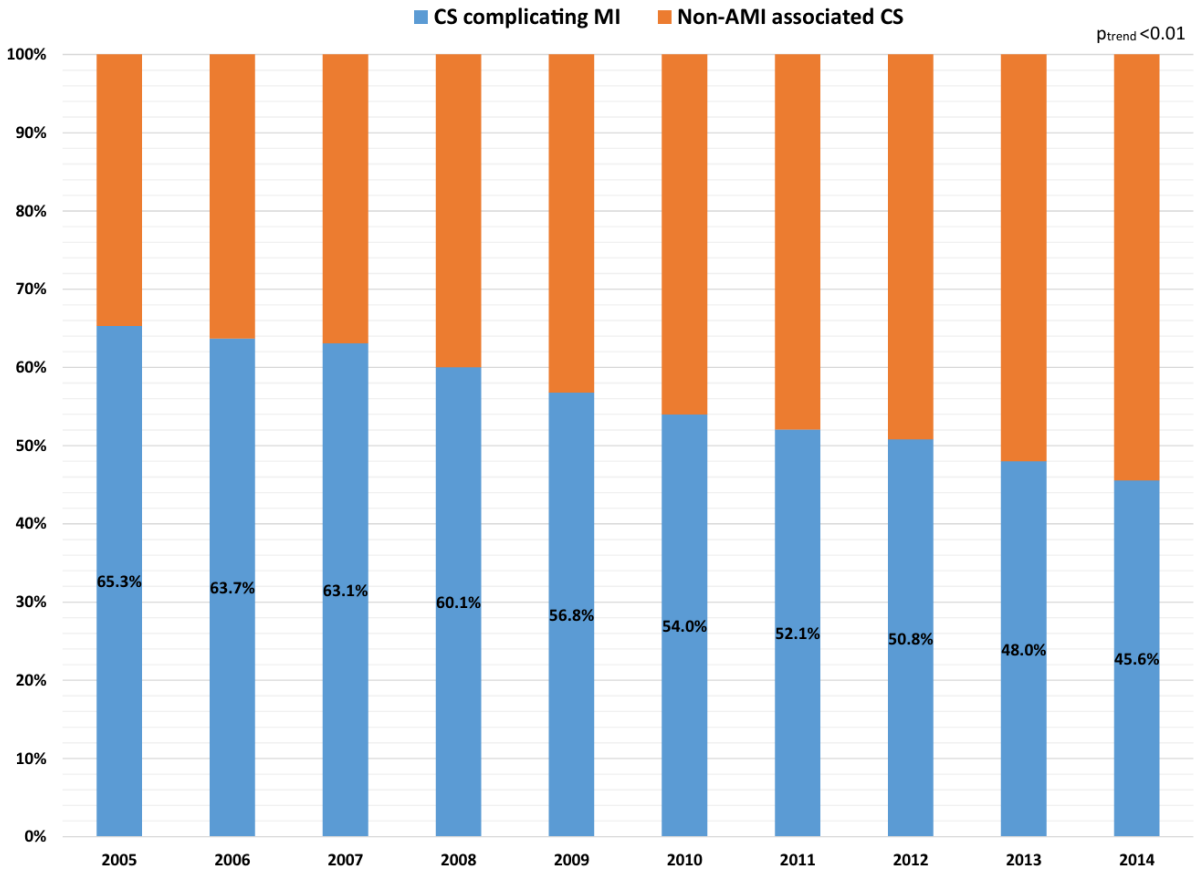


Figure 4. Trends in in-hospital mortality of patients with acute myocardial infarction according to cardiogenic shock (CS). Percentage in the table indicates percentage of overall CS. Dotted lines indicate trendlines.

Épidémiologie du choc cardiogénique



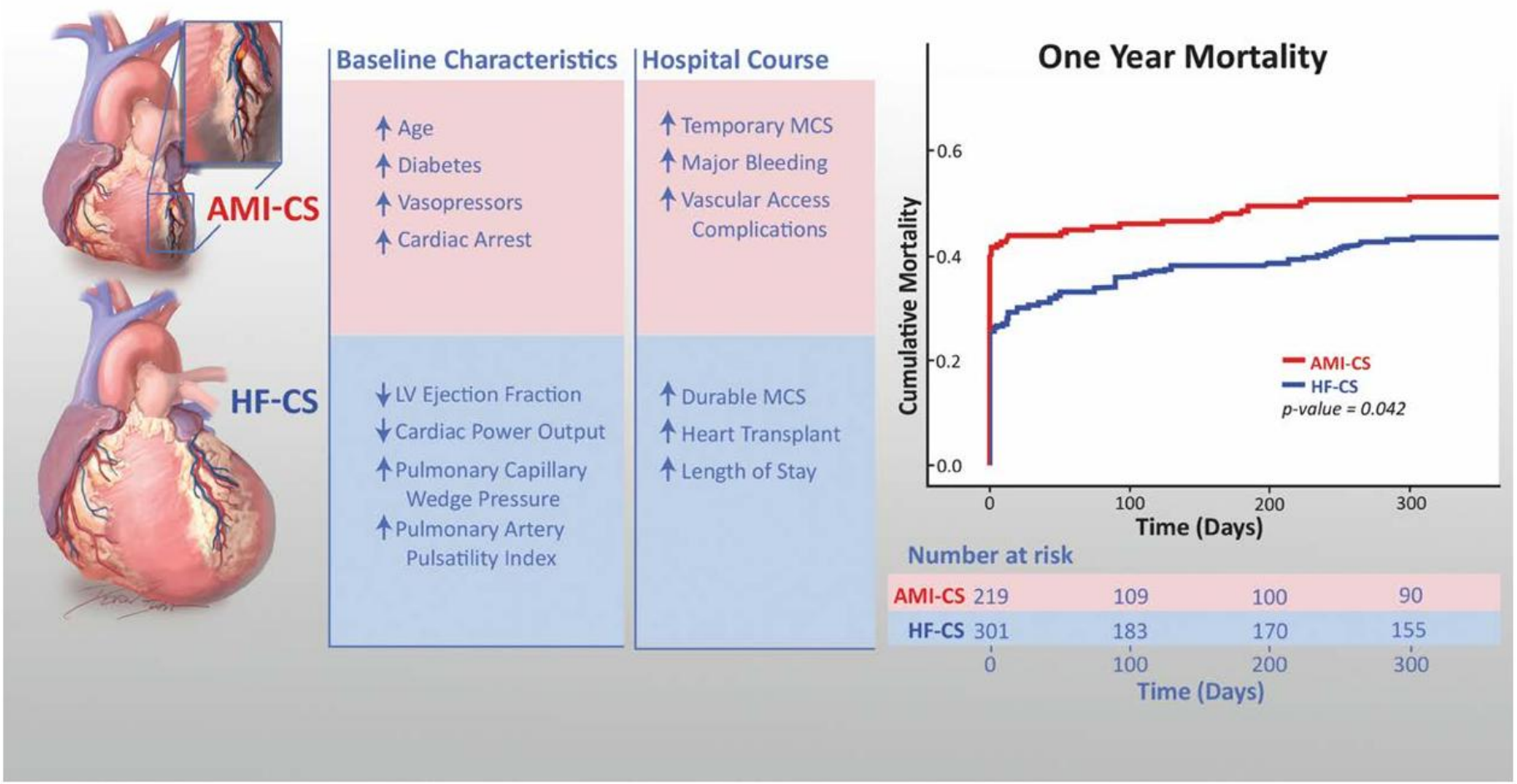
- Ischemic cardiogenic shock:
  - Main etiology
  - Net decrease
- CS from others etiologies:
  - Net increase
  - Miscellaneous

Shah. *Clin Research Cardiol* 2017

Épidémiologie du choc cardiogénique

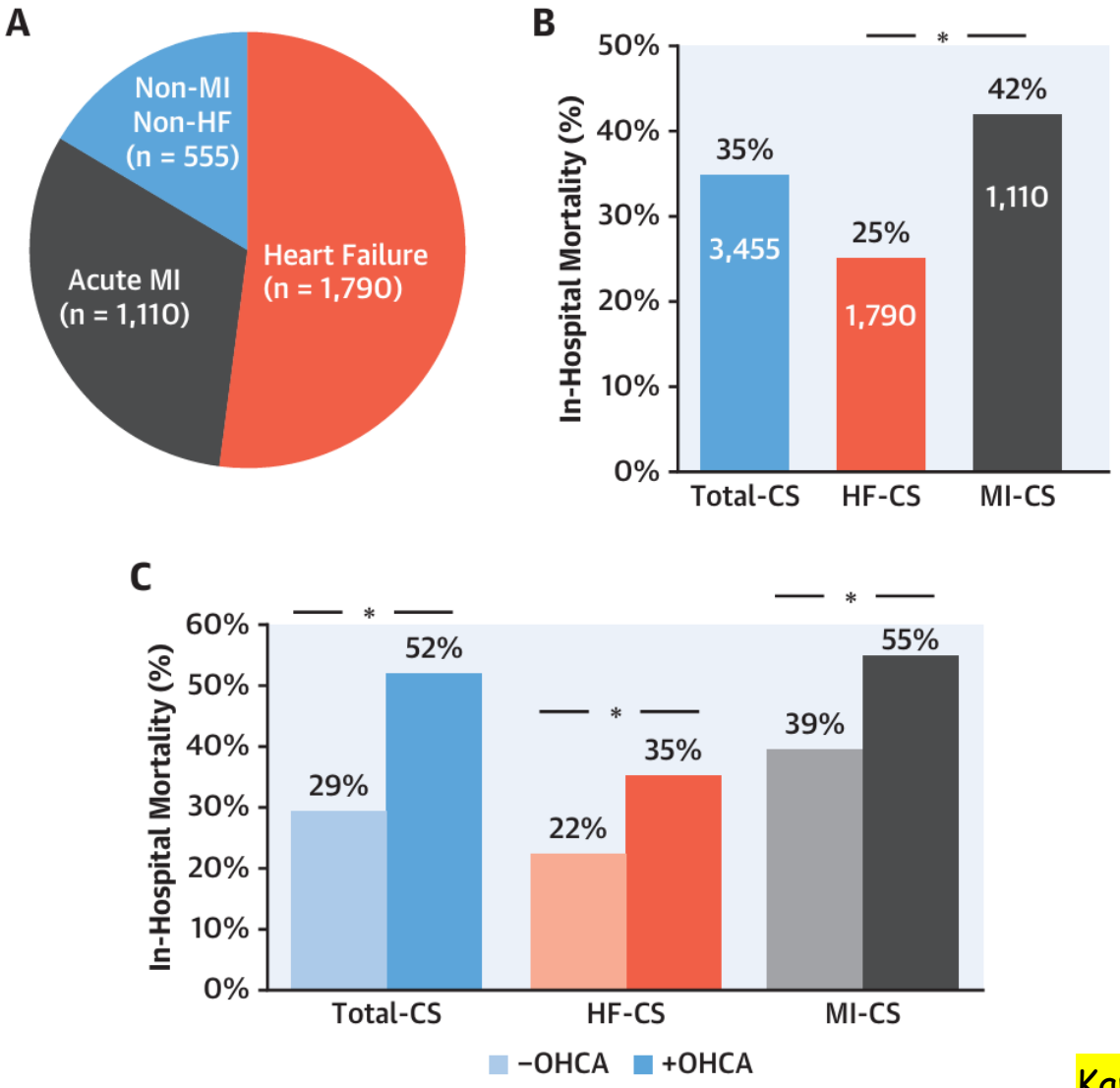
Cardiogenic Shock From Heart Failure Versus Acute Myocardial Infarction: Clinical Characteristics, Hospital Course, and 1-Year Outcomes

Sinha. *Circulation Heart Failure* 2022



Épidémiologie du choc cardiogénique

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



Kapur. JACC. 2022

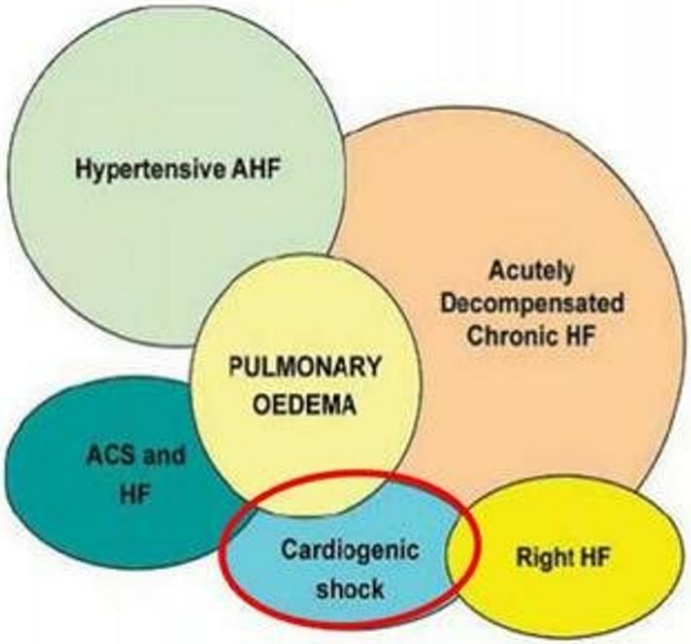
Définition « originelle » du choc cardiogénique

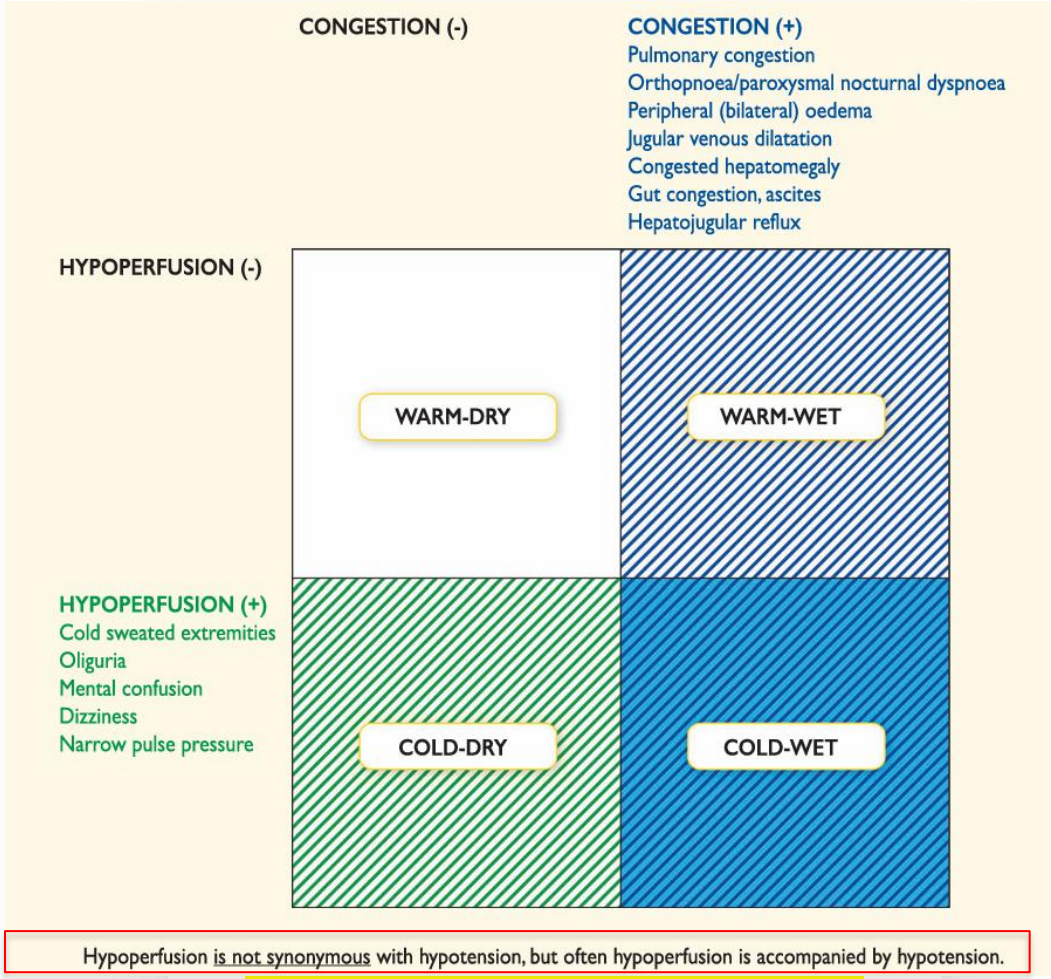
Baisse du débit cardiaque avec hypoxie tissulaire en présence d'une volémie normale (… ou normalisée après optimisation)

- ✓ **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**
- ✓ **SvO_2 < 60%** (Sv_mO_2 … Sv_cO_2 après optimisation volémie)
- ✓ $DAVO_2$ > 5,5 mL O_2 /dL

- ✓ **Dysfonction contraction myocardique** : FE_{VG} <40% / **Augmentation pressions** de remplissage (PTD_{VG})
- ✓ **Hypoperfusion tissulaire** (altération conscience, extrémités ± 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[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·min^{-1}·m^{-2} AND PCWP ≥15 mm Hg	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Catecholamines to maintain SBP >90 mm Hg AND Clinical pulmonary congestion AND Impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output <30 mL/h, or lactate >2.0 mmol/L)	SBP <90 mm Hg with adequate volume and clinical or laboratory signs of hypoperfusion Clinical hypoperfusion: Cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure Laboratory hypoperfusion: Metabolic acidosis, elevated serum lactate, elevated serum creatinine

		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)

Van Diepen. *Circulation*. 2017

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*

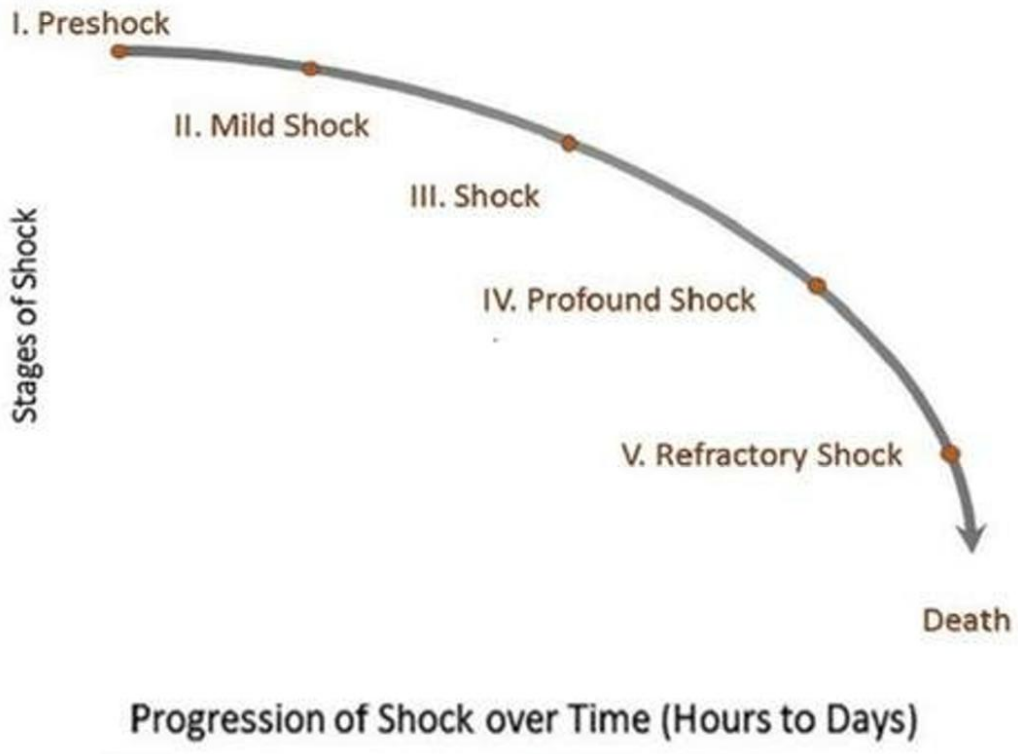
Design et résultats préliminaires de l'étude FRENSHOCK 2016 : une étude nationale prospective multicentrique de cohorte sur le choc cardiogénique toute cause

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 !



I. Preshock-Worsening Heart Failure:
Diuretics/Inodilators

II. Mild Shock-Hypotension:
Inotropes/Vasopressors+/-IABP, Impella CP

III. Shock-Lactic Acidosis, Tissue Hypoperfusion:
Tandem Heart, Impella 5.0, CentriMag, RotaFlow, ECMO

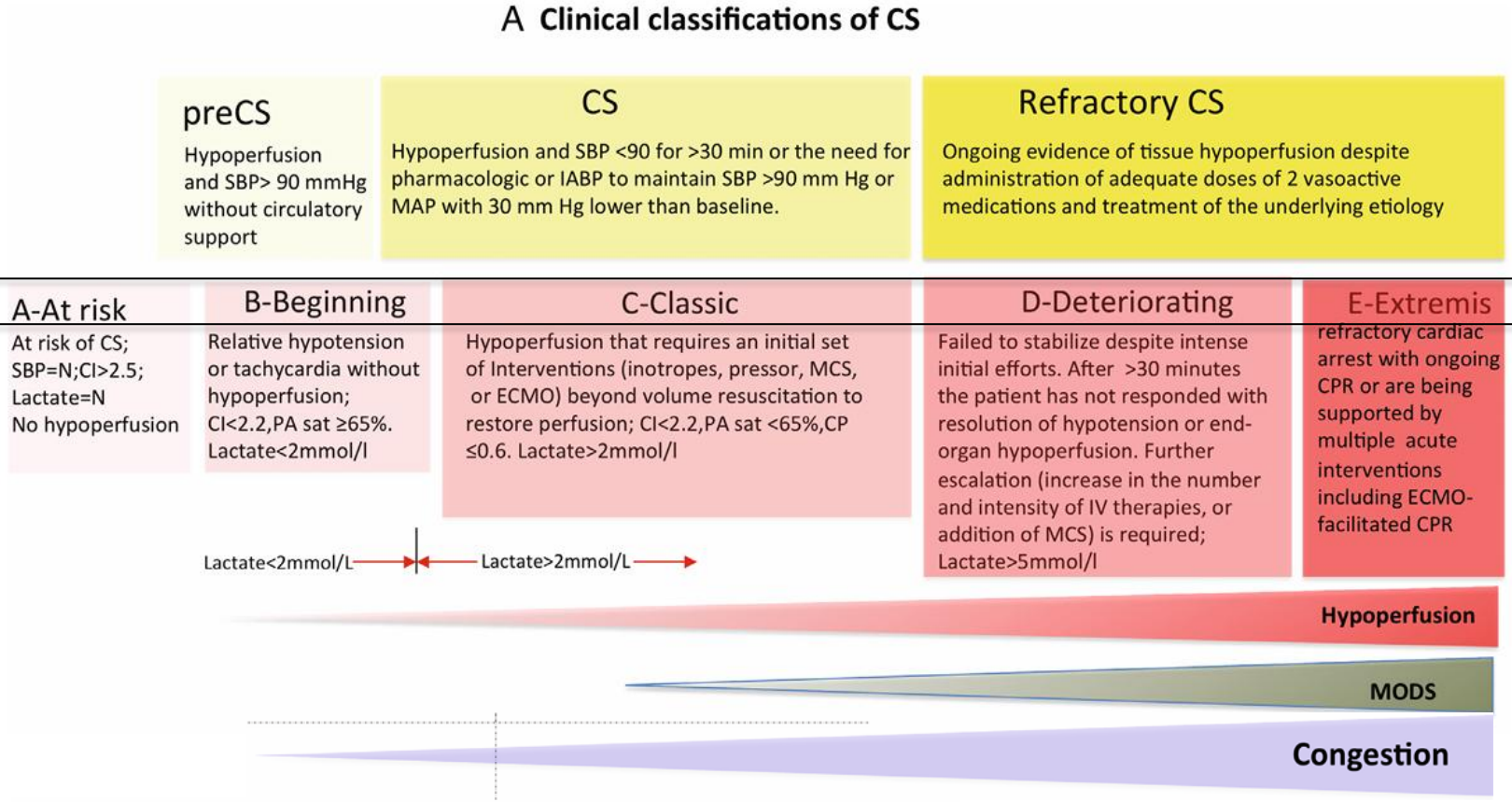
IV. Profound Shock-End Organ Failure:
CentriMag, RotaFlow, ECMO

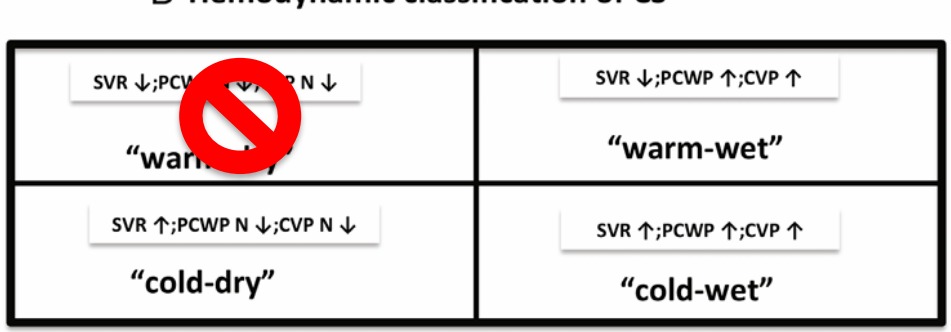
V. Refractory Shock-Vasodilation, Multiorgan Failure, Refractory Hypotension:
Assess for futility of Care

Classification du choc cardiogénique

Baran. *Catheter Cardiovasc Interv* 2019

Classification du choc cardiogénique





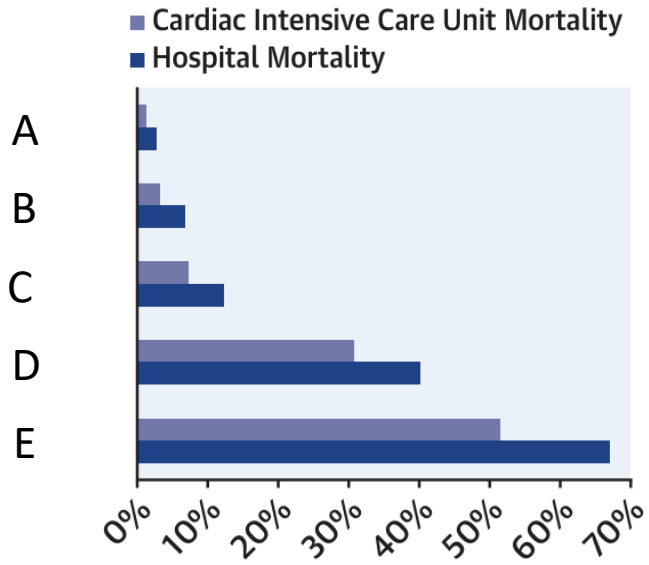
Adapted from: Chioncel. *EHJ Heart Failure* 2020

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

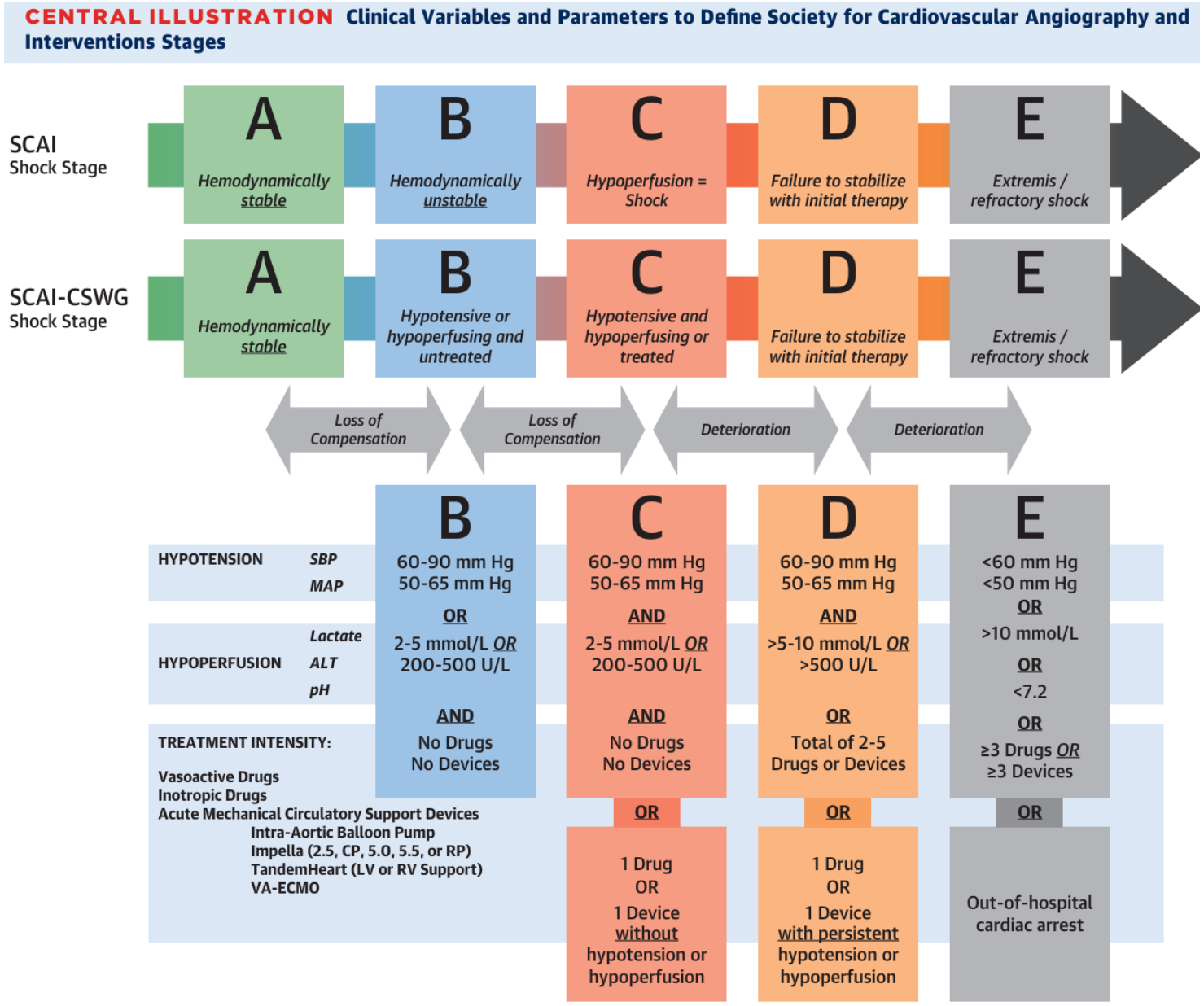
Jentzer. *JACC* 2019

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



Mortalité croissante ds tous les sous-groupes (SCA, insuff. card., ACR, détériorat° tardive)

Classification du choc cardiogénique



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

Kapur. JACC. 2022

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**

Kohsaka. *Arch Intern Med 2005*

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

- ↑ Fréquence cardiaque
- ↑ Résistances vasculaires systémiques
- ↑ Pression télédiastolique du VG
- Mobilisation secteur veineux splanchnique / rétention HS
- ↓ Pression artérielle

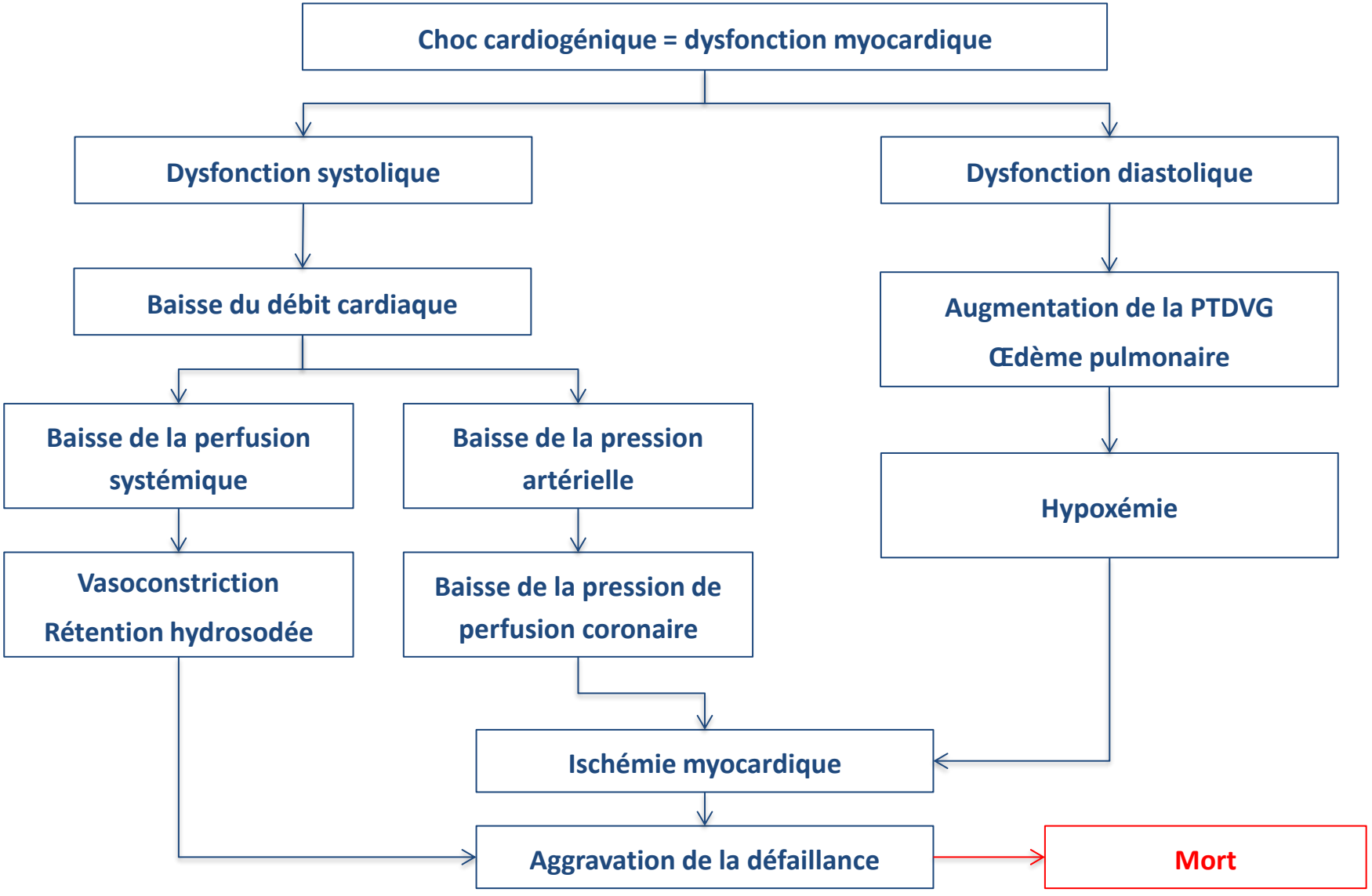
- Hypoperfusion périphérique
- Congestion pulmonaire / hypoxie

- Souffrance de certains organes

- Développement d’une inflammation systémique généralisée - le SIRS

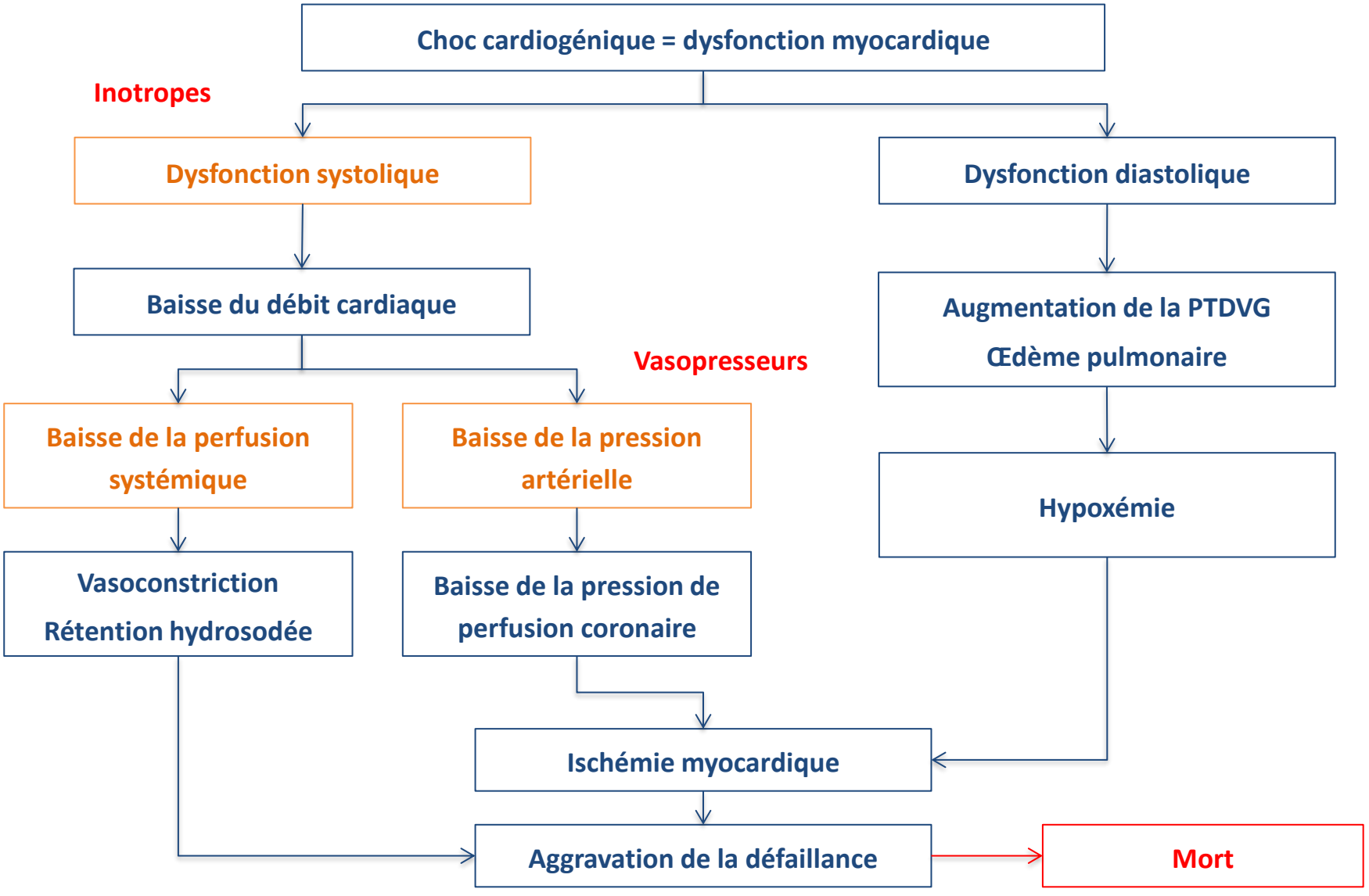
Merci à Eric Bonnefoy. *DIU USIC 2025*

Physiopathologie : paradigme classique



Merci à Adrien Bouglé. DIU TUSAR 2025

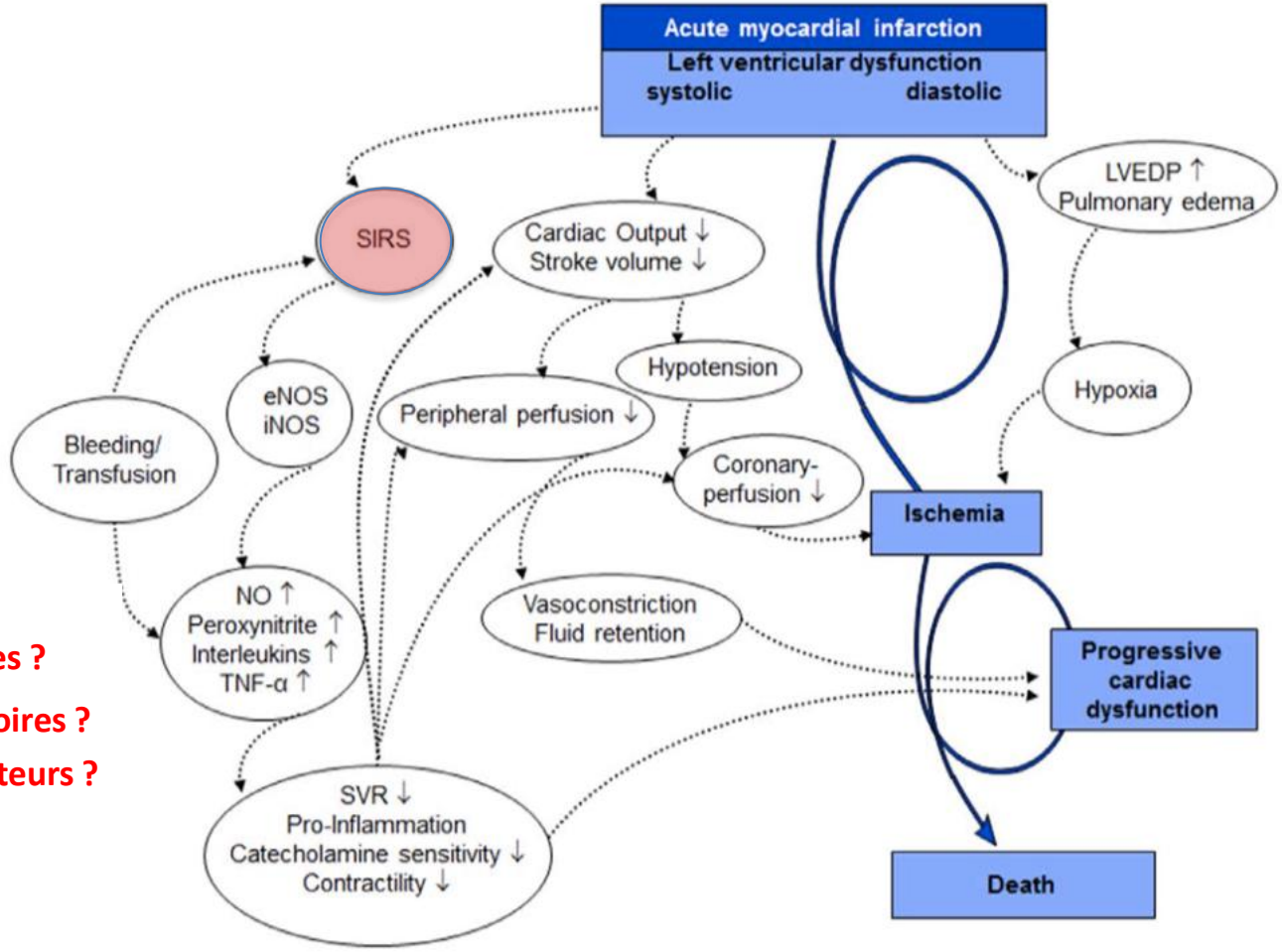
Physiopathologie : paradigme classique



Merci à Adrien Bouglé. DIU TUSAR 2025

Physiopathologie : « nouveau » paradigme

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



Traitements étiologiques

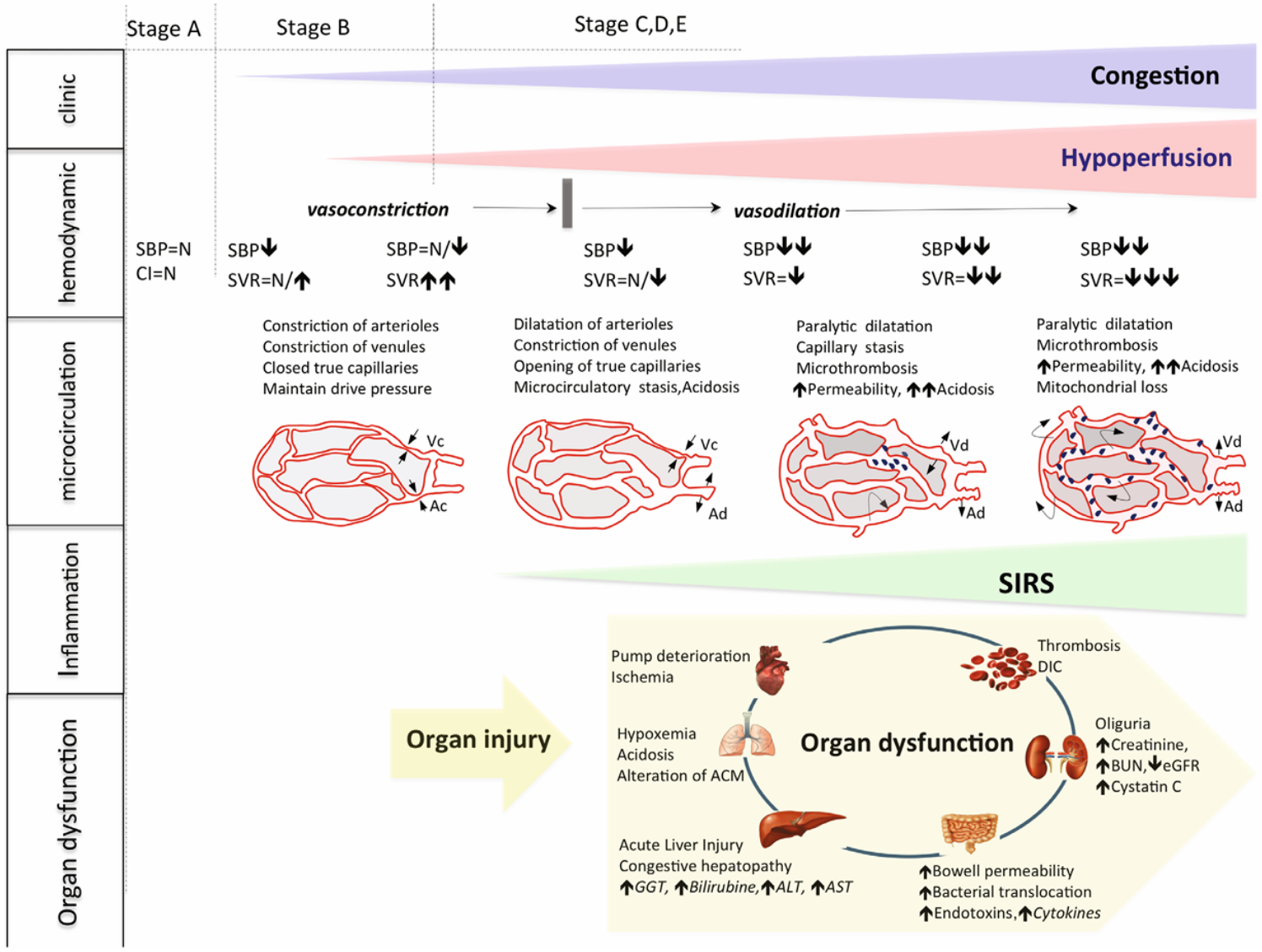
Anti-biotiques ?

Anti-inflammatoires ?

Immunomodulateurs ?

Van Diepen. *Circulation 2017*

Physiopathologie : « nouveau » paradigme



Van Diepen. *Circulation* 2017

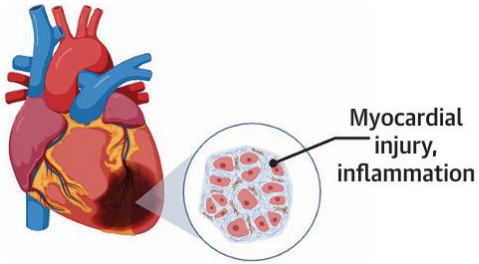
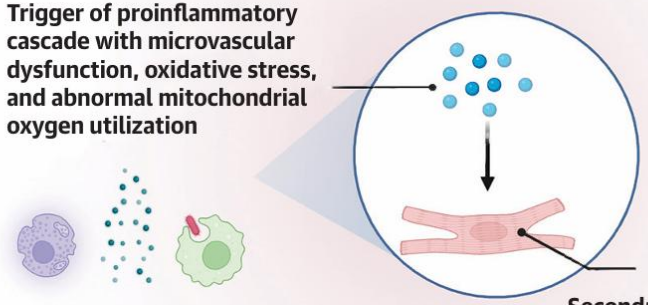
Physiopathologie : « nouveau » paradigme

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

Merci à Eric Bonnefoy. *DIU USIC 2025*

CENTRAL ILLUSTRATION Conceptual Model of Mixed Shock Etiology and Pathogenesis

	Etiology	Hemodynamic Features
Cardiogenic-Vasodilatory Shock	 Primary Cardiac Insult	Prototypical Cardiogenic Shock: Low Cardiac Index → Maladaptive Reduction in SVR
Vasodilatory-Cardiogenic Shock	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
Primary Mixed Shock	Post-cardiotomy; Post-cardiac arrest syndrome → *Ischemia reperfusion injury* Primary Insult Triggering Low Cardiac Output and Vasoplegia	Simultaneous or Rapid Sequential Cardiac Dysfunction and Vasoplegia

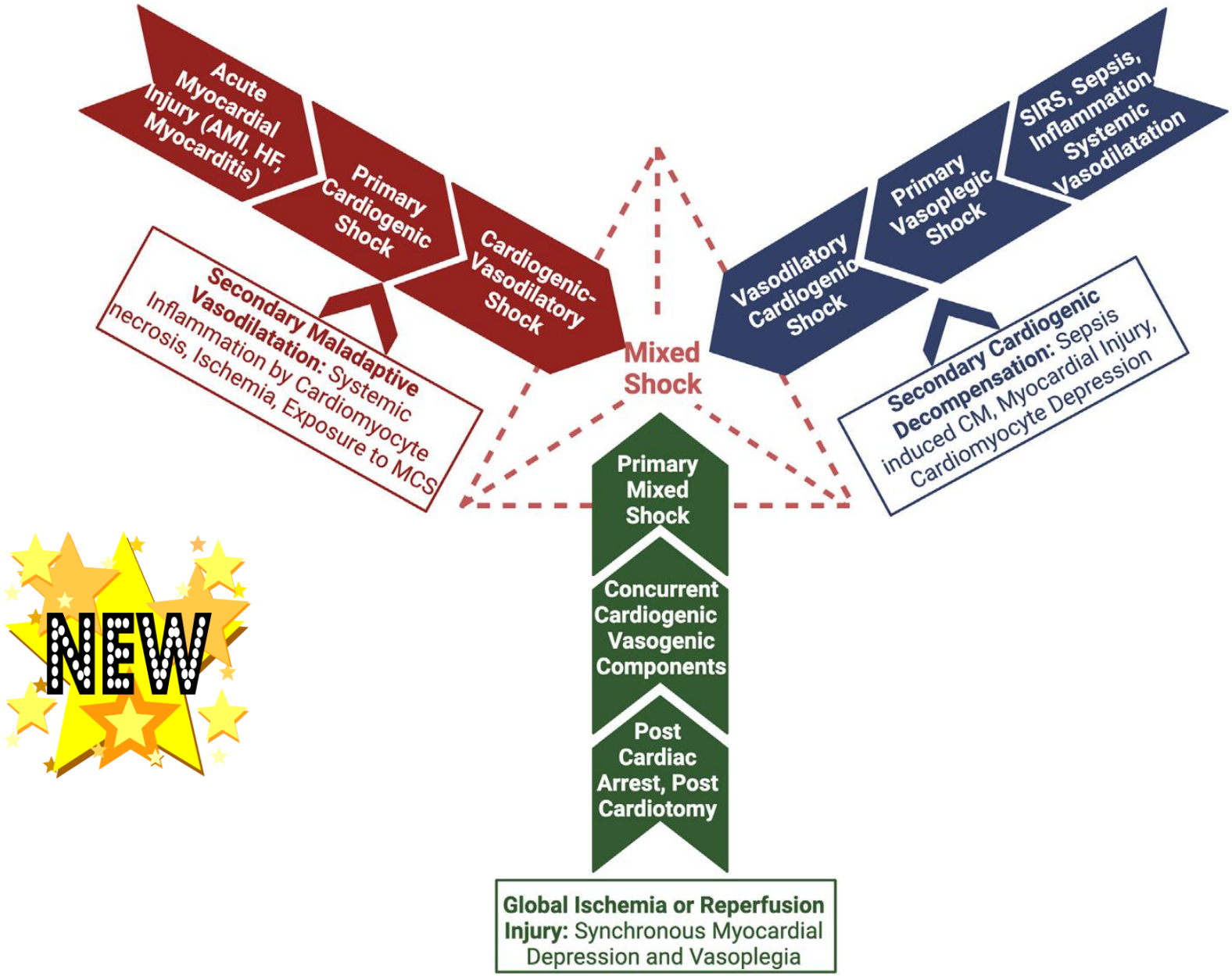
'*Two-Hit*' Pathogenesis Model

'*Single-Hit*' Model

Secondary cardiac stress: *De novo or pre-existing cardiac disease*

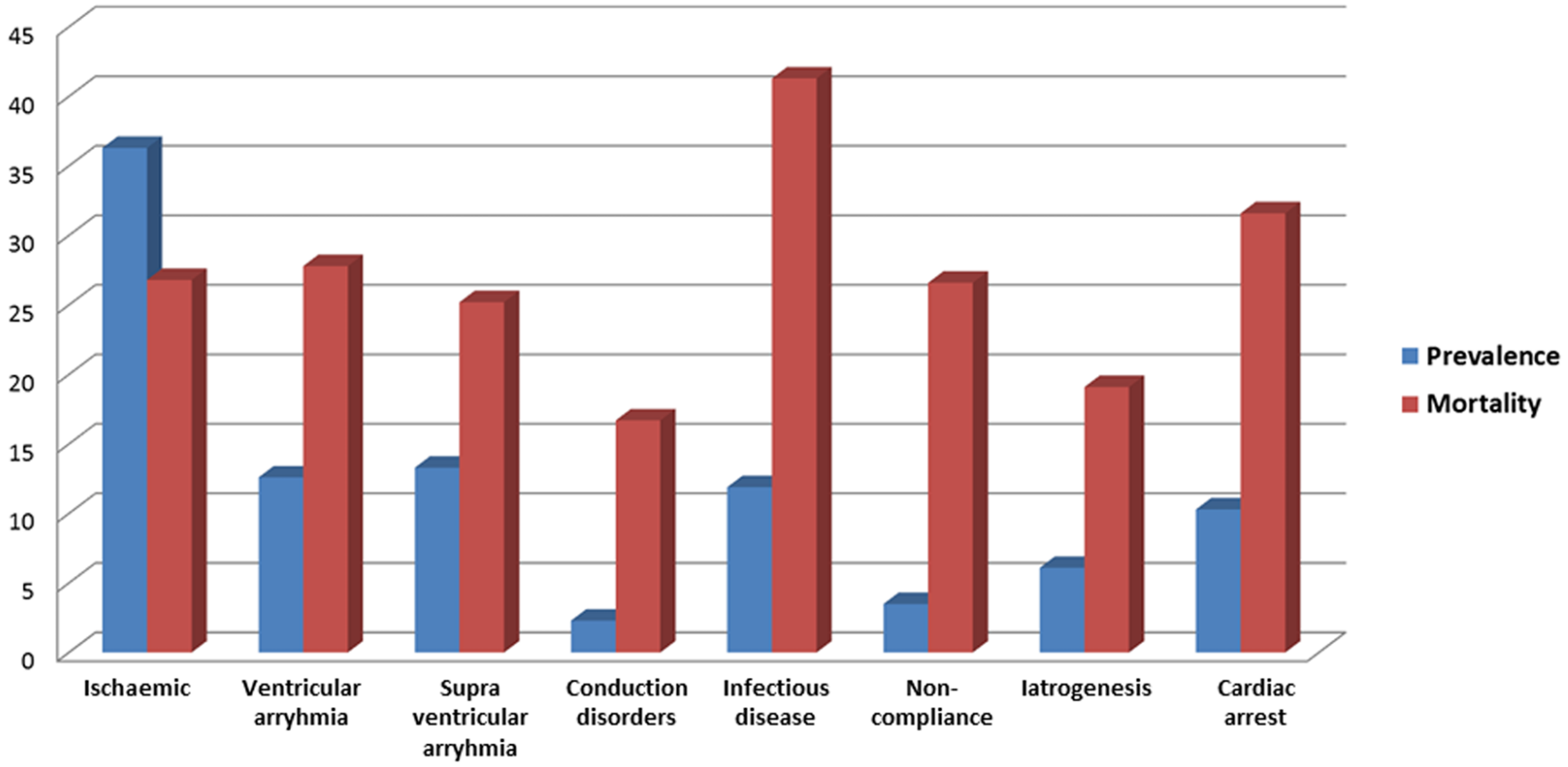
Jentzer. *Mixed Cardiogenic Vasodilatary Shock. JACC 2025*

FIGURE 1 Proposed Classification of Mixed Shock



Jentzer. *Mixed Cardiogenic Vasodilatary Shock. JACC 2025*

Facteurs de gravité = grand danger (FRENSHOCK)



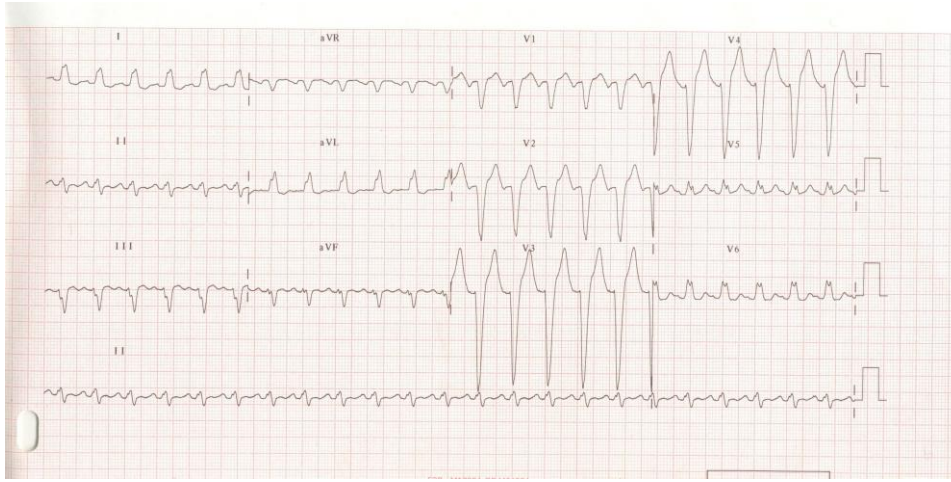
- ✓ 48 centres ; 772 patients
- ✓ Critères hémodynamiques + surcharge droite et/ou gauche + « malperfusion »
- ✓ Choc cardiogénique d'origine ischémique : 36,3% ; non-ischémique 63,7% (jusqu'à 3 « triggers » / patient)
- ✓ Nécessité d'un support d'organe chez 58,3% des patients

Delmas. *ESC Heart Fail 2022*

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

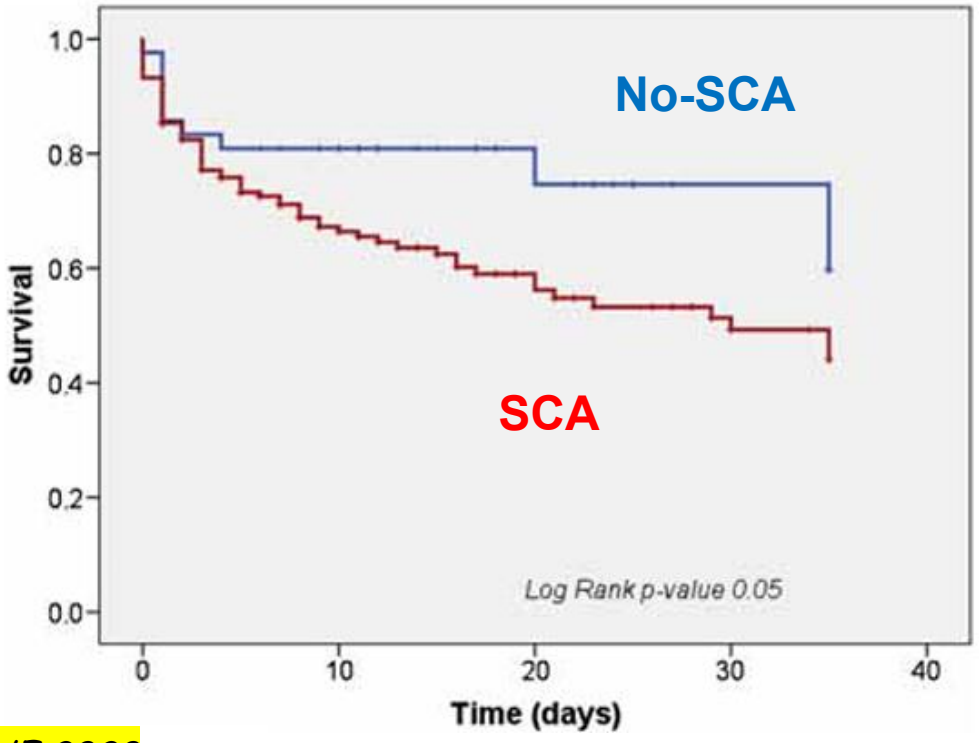
Facteurs de gravité = grand danger (SCA)

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

Veli-Pekka Harjola[1*,†], Johan Lassus[2†], Alessandro Sionis[3], Lars Køber[4], Tuukka Tarvasmäki[5], Jindrich Spinar[6], John Parissis[7], Marek Banaszewski[8], Jose Silva-Cardoso[9], Valentina Carubelli[10], Salvatore Di Somma[11], Heli Tolppanen[2], Uwe Zeymer[12], Holger Thiele[13], Markku S Nieminen[2], and Alexandre Mebazaa[14], 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



EHJ HF 2020

Facteurs de gravité = grand danger (SCA)

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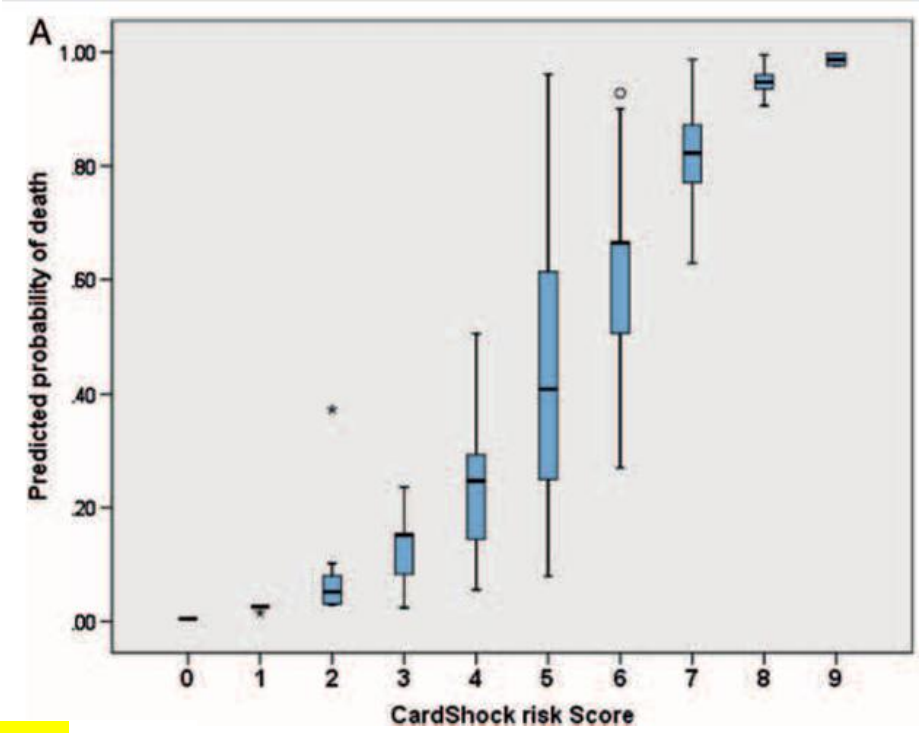
Table 4 The CardShock risk Score for risk prediction of in-hospital mortality in cardiogenic shock

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



EHJ HF 2020

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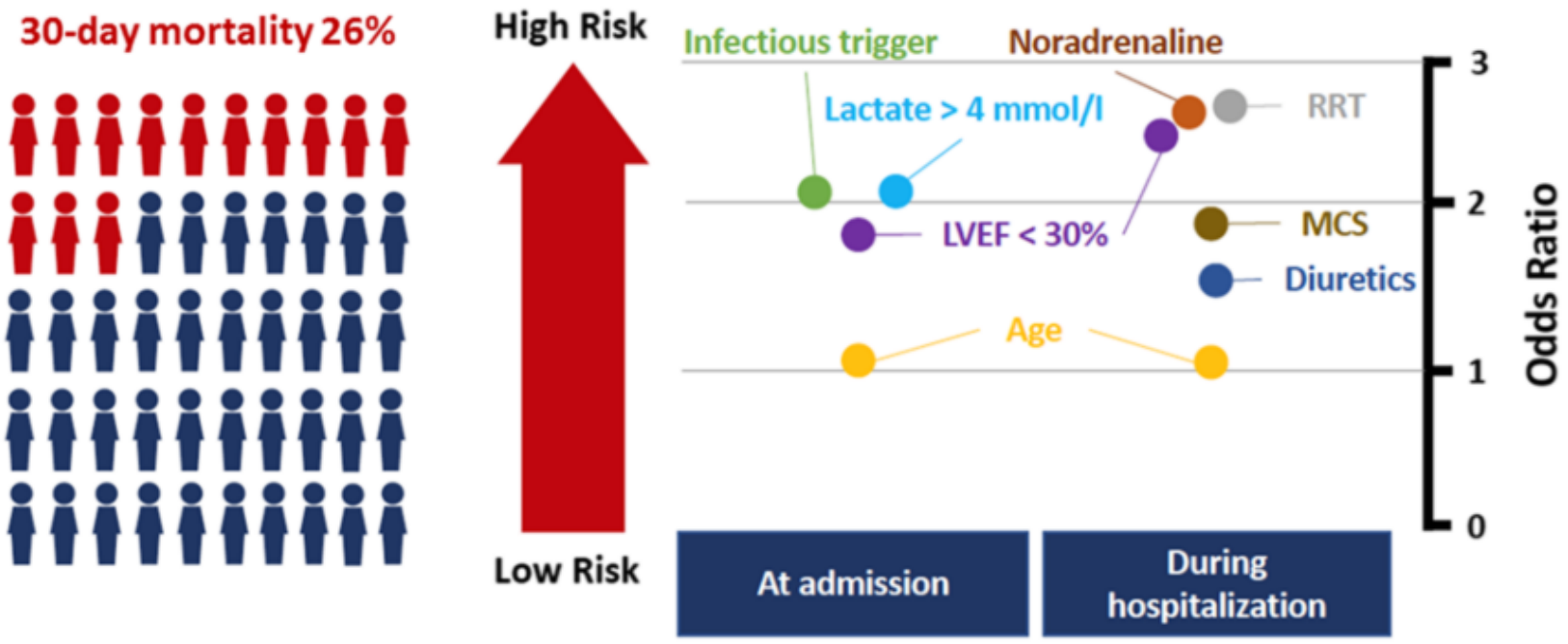

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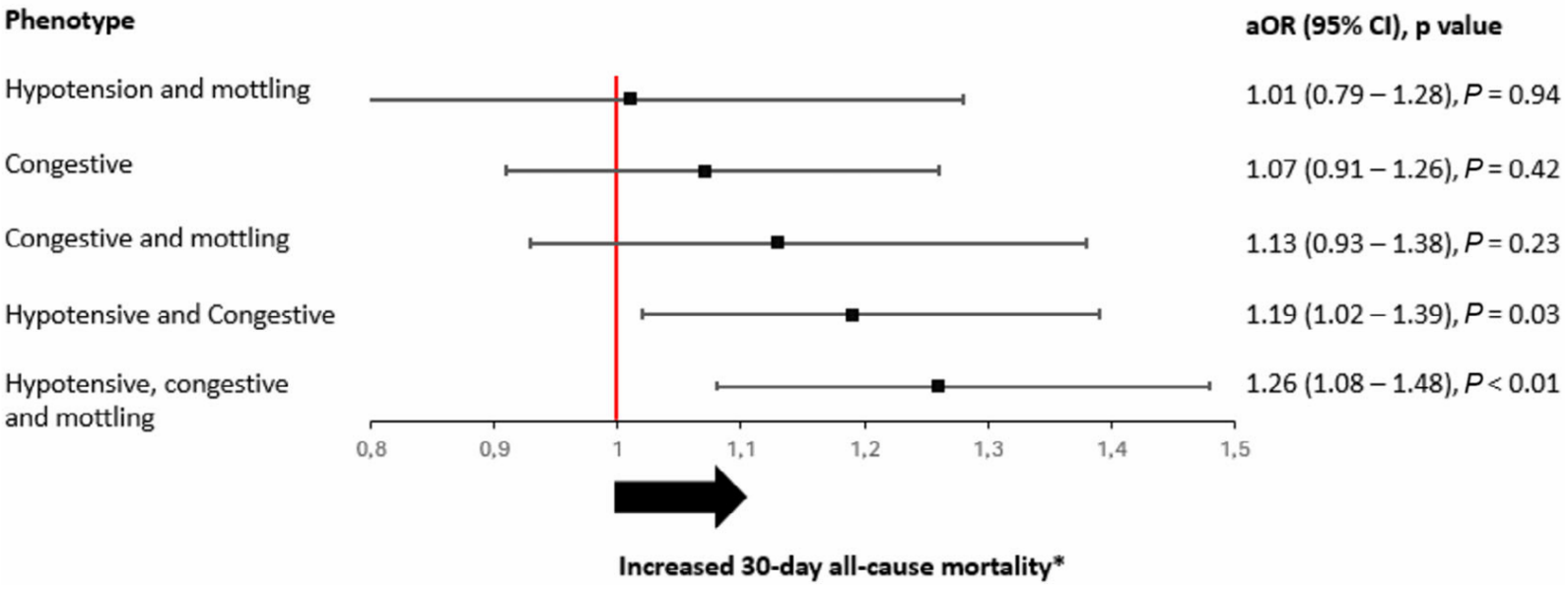
- ✓ Critères hémodynamiques + surcharge droite et/ou gauche + « malperfusion »
- ✓ Choc cardiogénique d'origine ischémique : 36,3% ; non-ischémique 63,7% (jusqu'à 3 « triggers » / patient)
- ✓ Nécessité d'un support d'organe chez 58,3% des patients

Delmas. *ESC Heart Fail 2022*

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



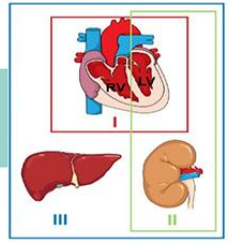
*The reference group was that with hypotension without congestion or mottling. Analysis were adjusted for age, sex, history of cardiomyopathy and ischaemic CS trigger.

Cherbi. *ESC Heart Fail 2025*

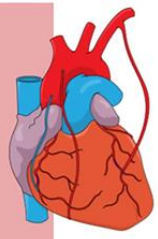
Diagnostic du choc cardiogénique

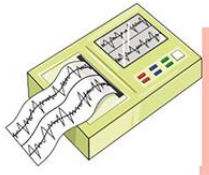
Definition
SBP<90mmHg
End-organ hypoperfusion: cold extremities, confusion, oliguria<30mL/h
Lactate>2mmol/L
CI<2.2L/min/m2

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

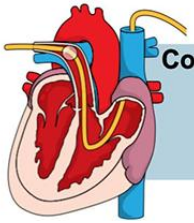


Identify cause of CS
Acute myocardial infarction
Cardiac arrest
Ventricular / Supraventricular arythmia
Conduction disorders
Infectious disease
Iatrogenesis
Non-compliance





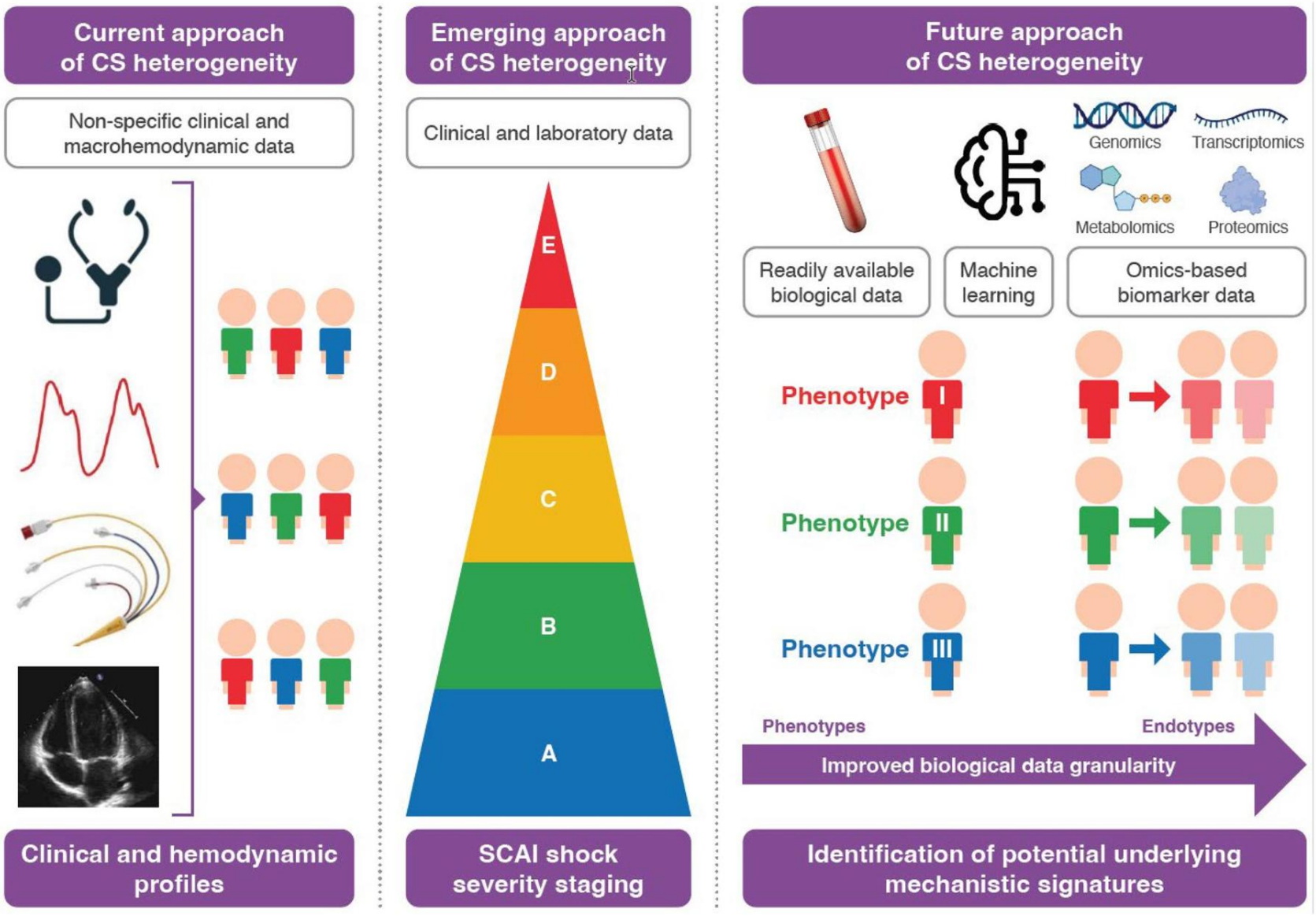
Key investigations for etiological assessment
12-lead ECG, X-Ray, Laboratory tests
Transthoracic +/- transoesophageal echocardiography
Lung and pleural ultrasound
Tomography scan for Aortic syndrome or Pulmonary embolism



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

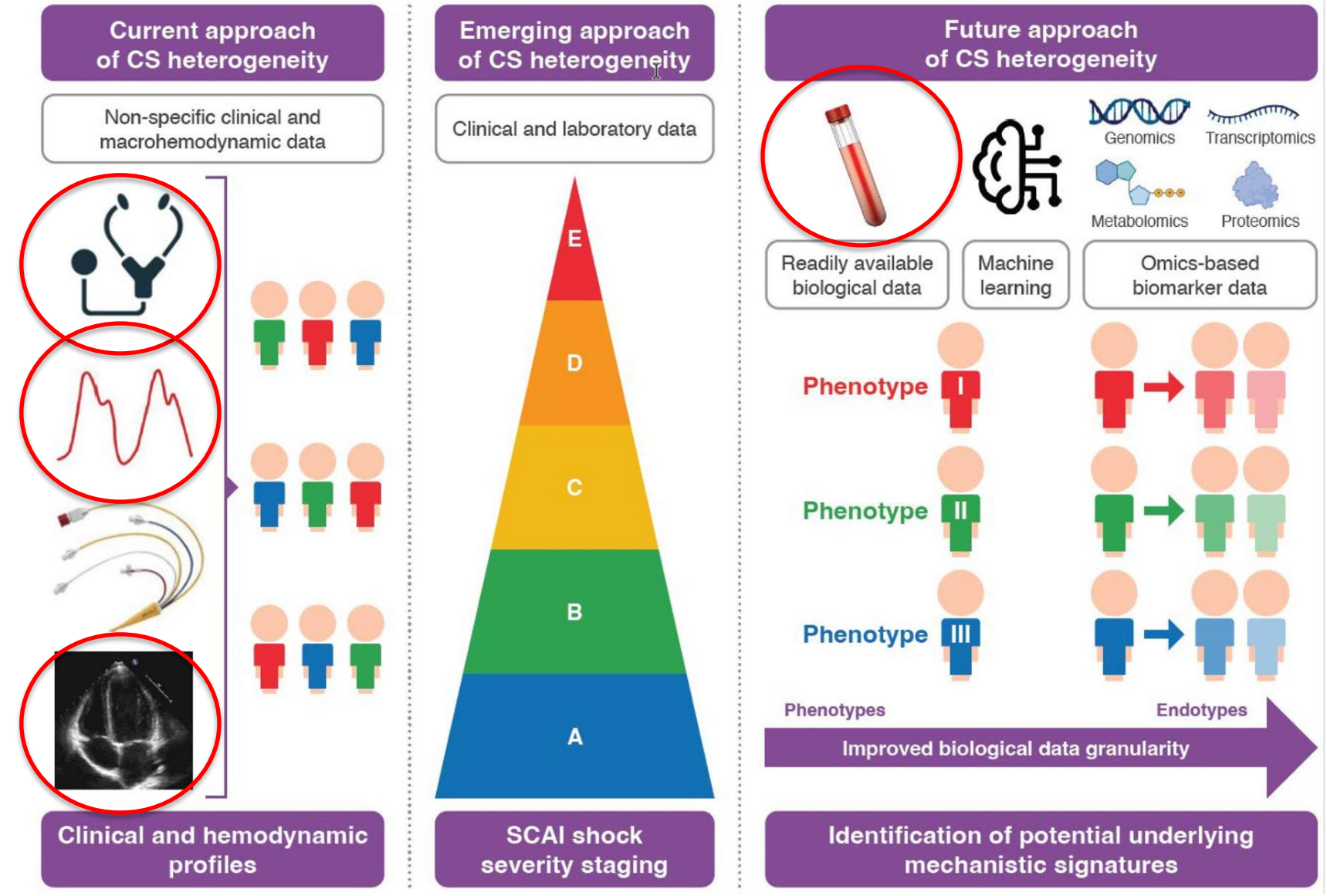
Laghlam. *AIC 2014*

Diagnostic du choc cardiogénique



Jung. Management of cardiogenic shock. *ICM* 2024

Outils du choc cardiogénique (diagnostic/pronostic)

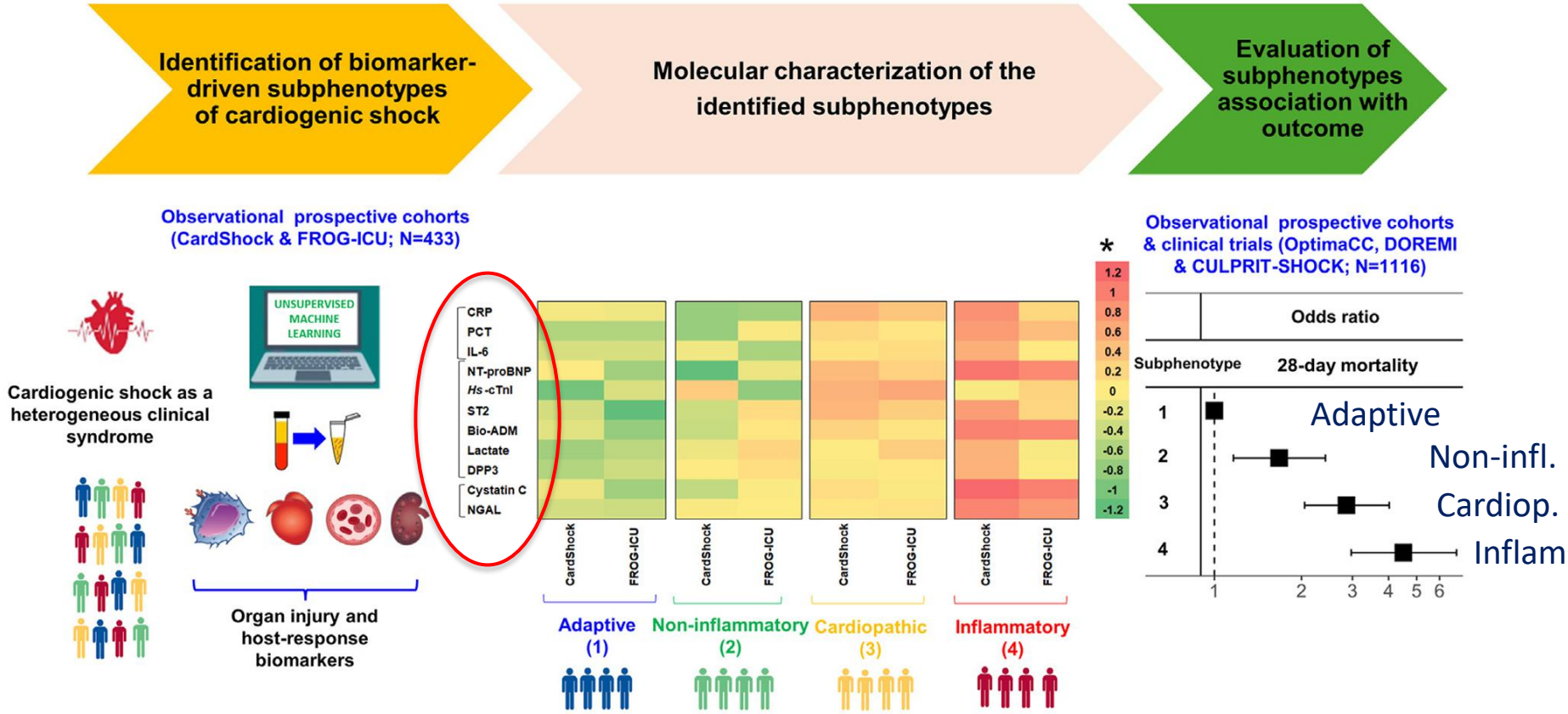


Jung. Management of cardiogenic shock. *ICM* 2024

Outils du choc cardiogénique (diagnostic/pronostic)

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

Sabri. *Lancet* 2025



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

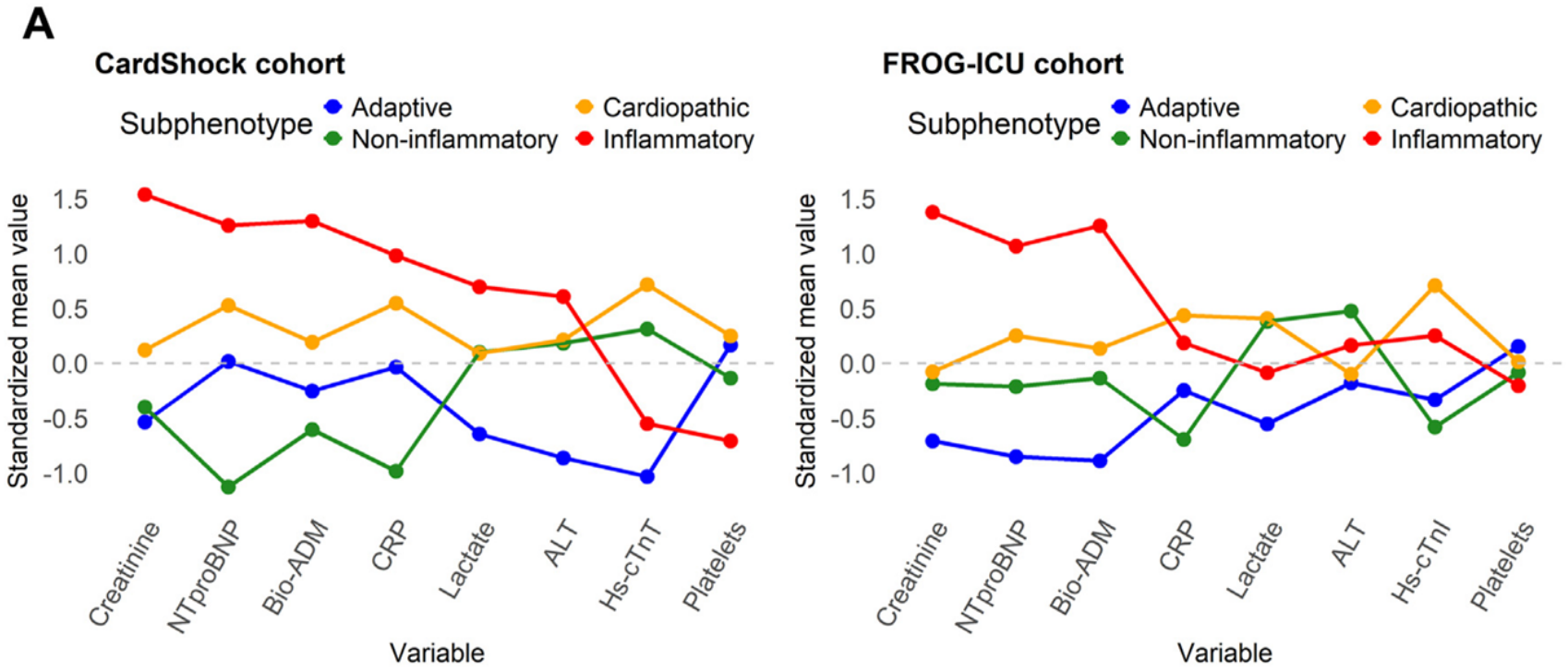
Rénal : créatinine, NGAL, cystatine C

Dysfonction/lésion CV : NT-proBNP, cTnI HS, ST2, bio-ADM, Lactate, DDP3

Outils du choc cardiogénique

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

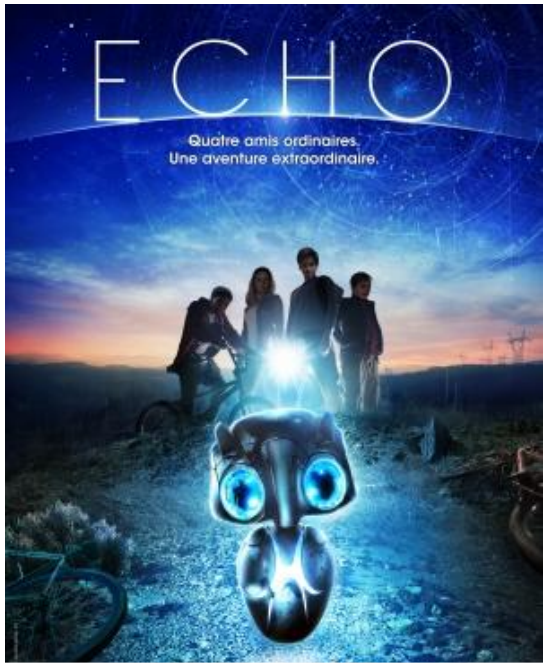
Sabri. *Lancet* 2025



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.

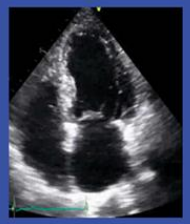
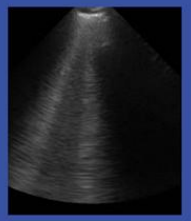
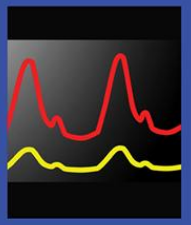
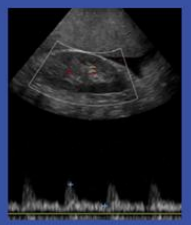
How we use critical care ultrasonography in the management of cardiogenic shock: a strategic game of chess in intensive care

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 +++

CHESS 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.

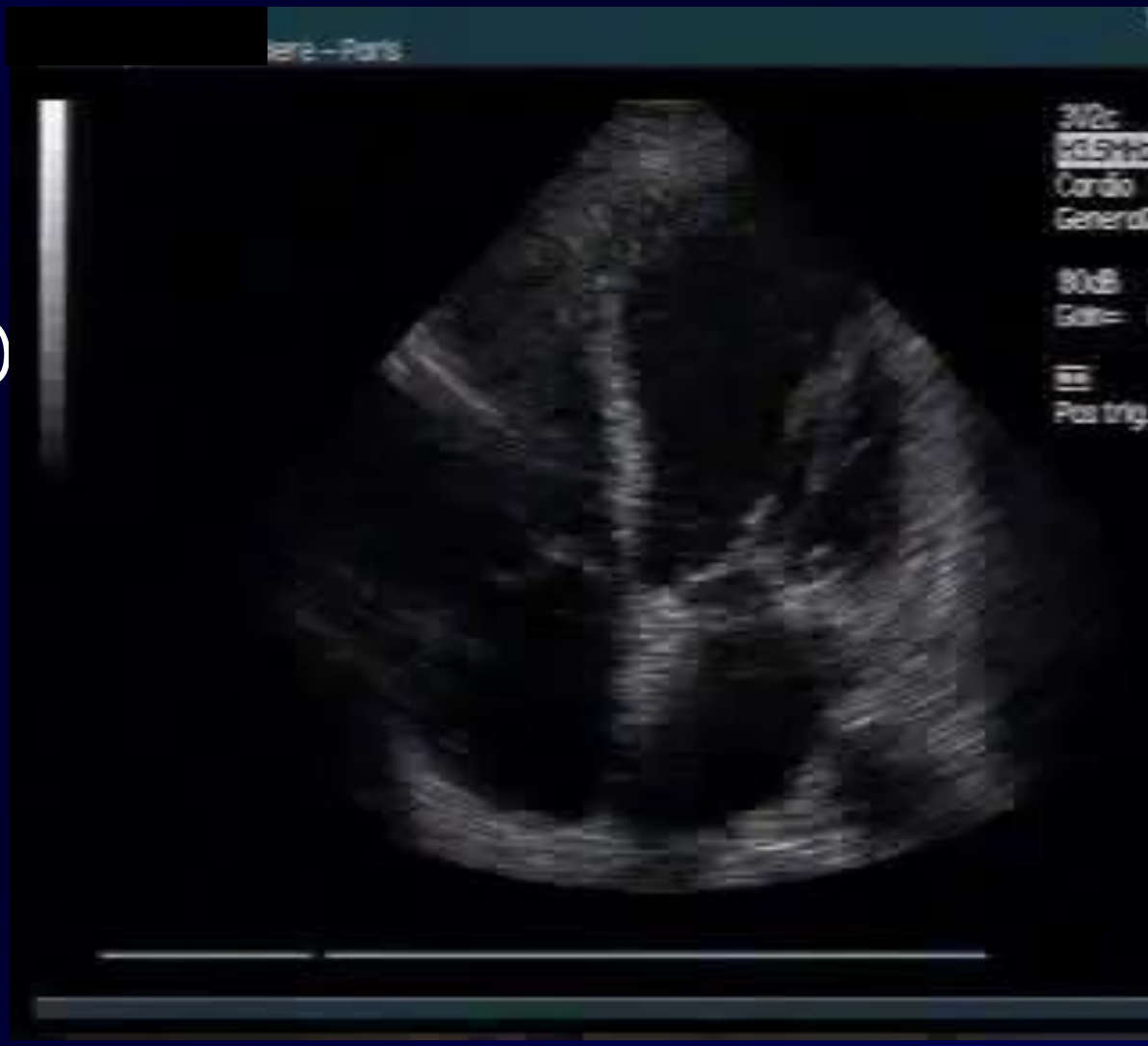
CAS 1 : diagnostic ?

Femme 48 ans

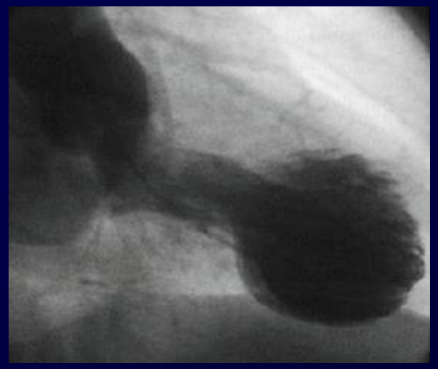
Surdosage aux bêta-bloquants et AA2 + ulcère duodénal (stress)

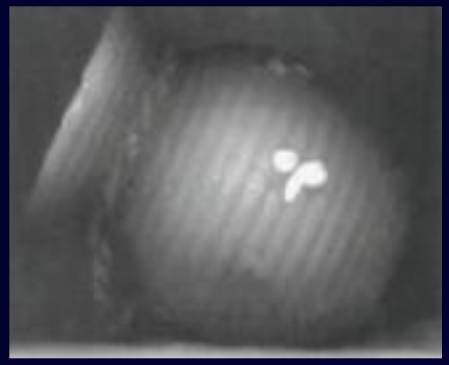
Survenue d'un choc cardiogénique à QRS fins et QT normal

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 dyskinésie apicale ± 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	Adré 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°	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 :

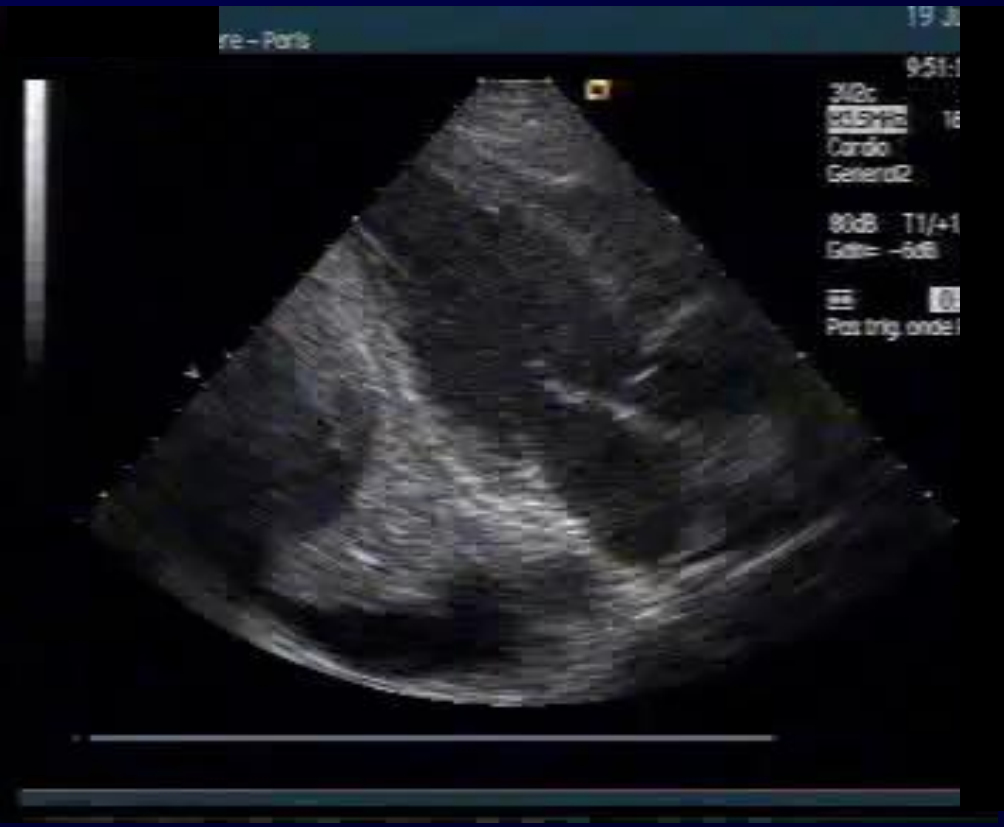
IS (µg/kg/min) = dobutamine + dopamine + 100xAdré + 100xNoradré

ECLS si choc persistant et IS > 60 µg/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

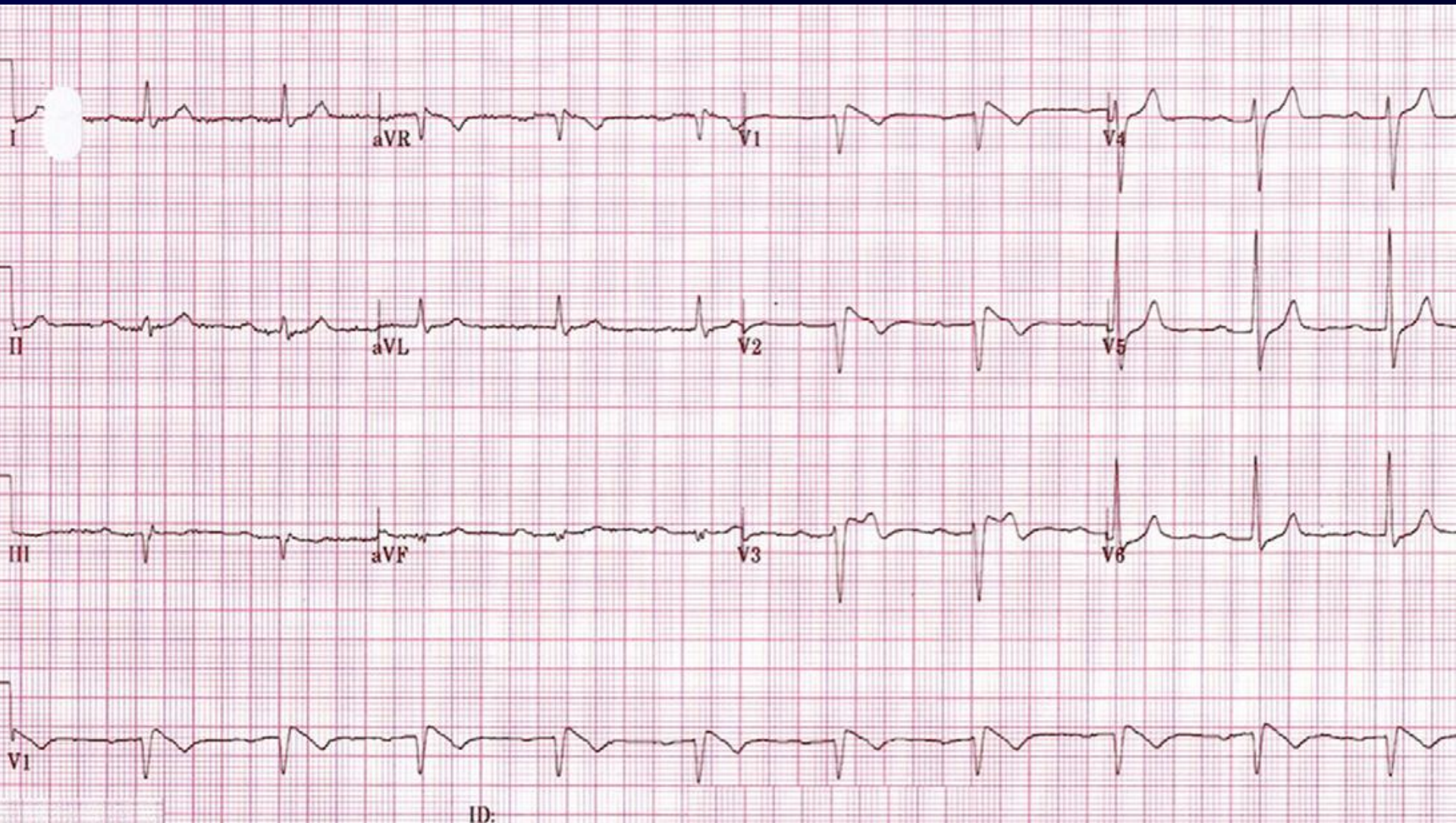


Takotsubo inversé ou reverse
(attention à Takotsubo médian vs myocardite segmentaire)

FE_{VG} 20 %, Qc ≈ 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)

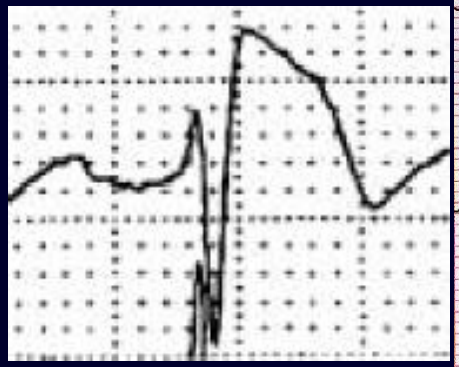
SDB type 1

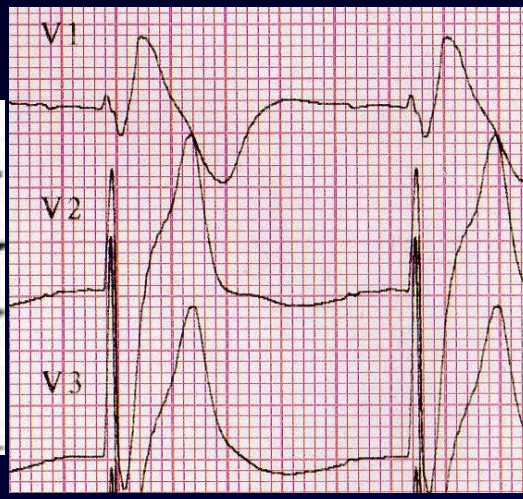
élévation du point J en V1 ≥ 2mm

onde T-

ST en dôme (« coved »)

(BBID)





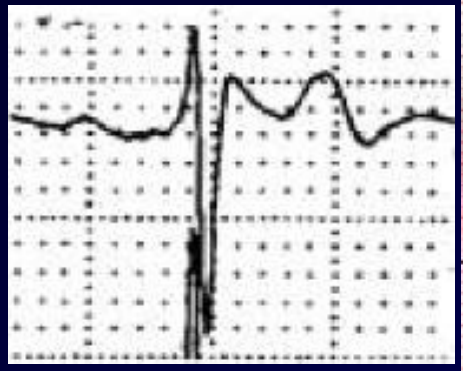
SDB type 2 ou 3

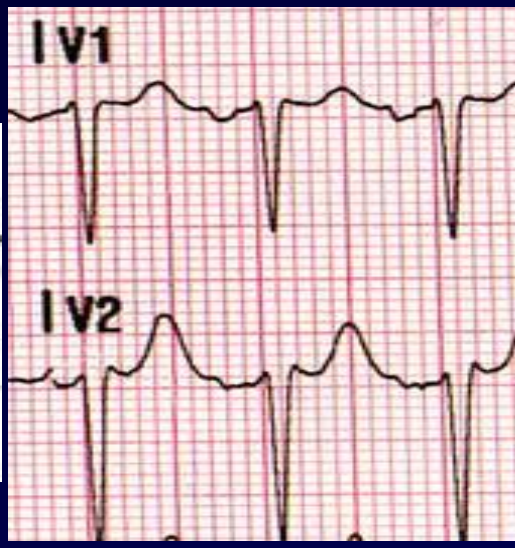
élévation du point J en V1 ≥ 2mm

onde T+

ST en selle (« saddleback »)

(± sus-ST)

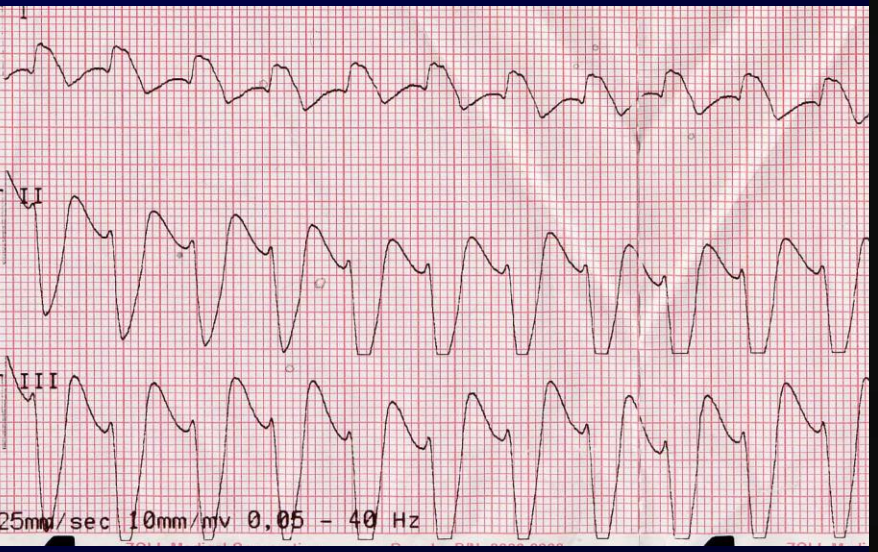


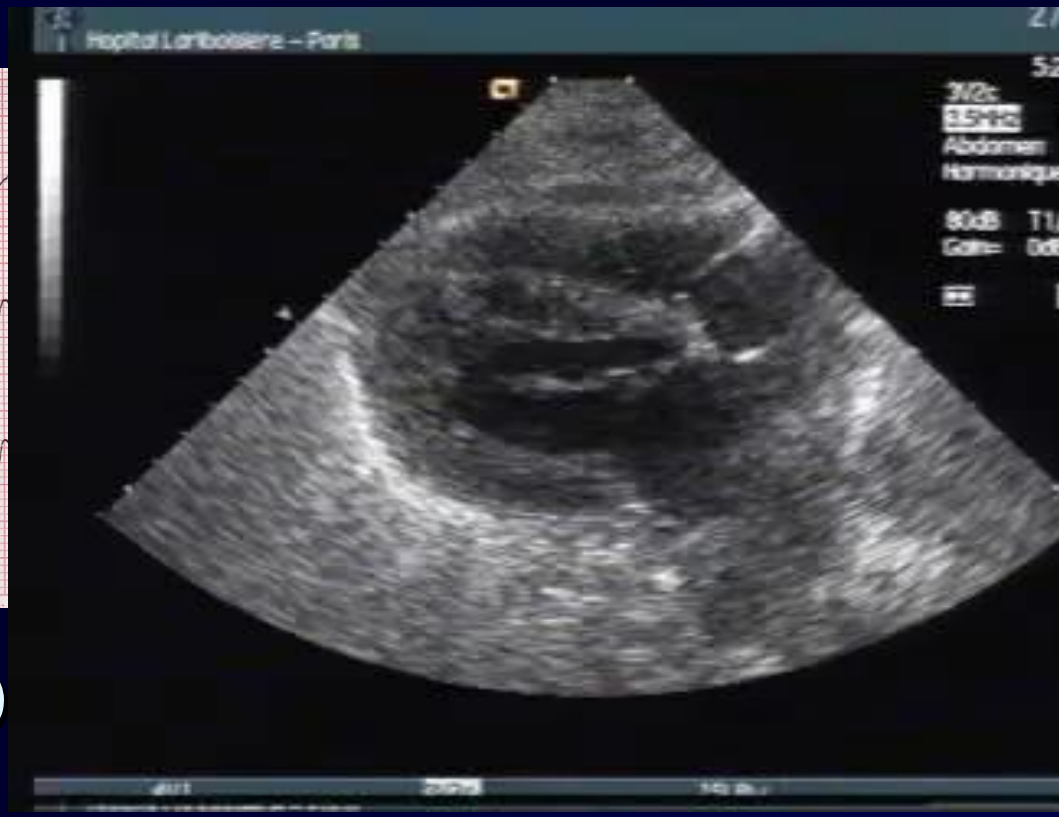


CHOCS CARDIOGÉNIQUES RÉFRACTAIRES TOXIQUES

EN BAS DEBIT 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…).

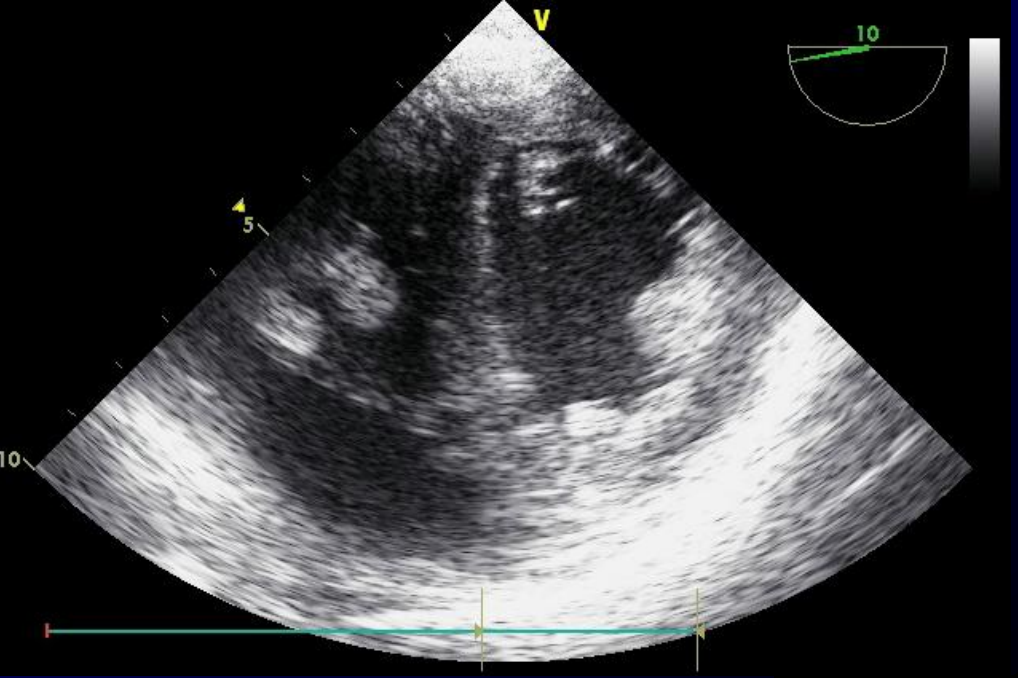
Conférence d'Experts SRLF/SFAR 2006-2020

Quels patients doit-on assister ?

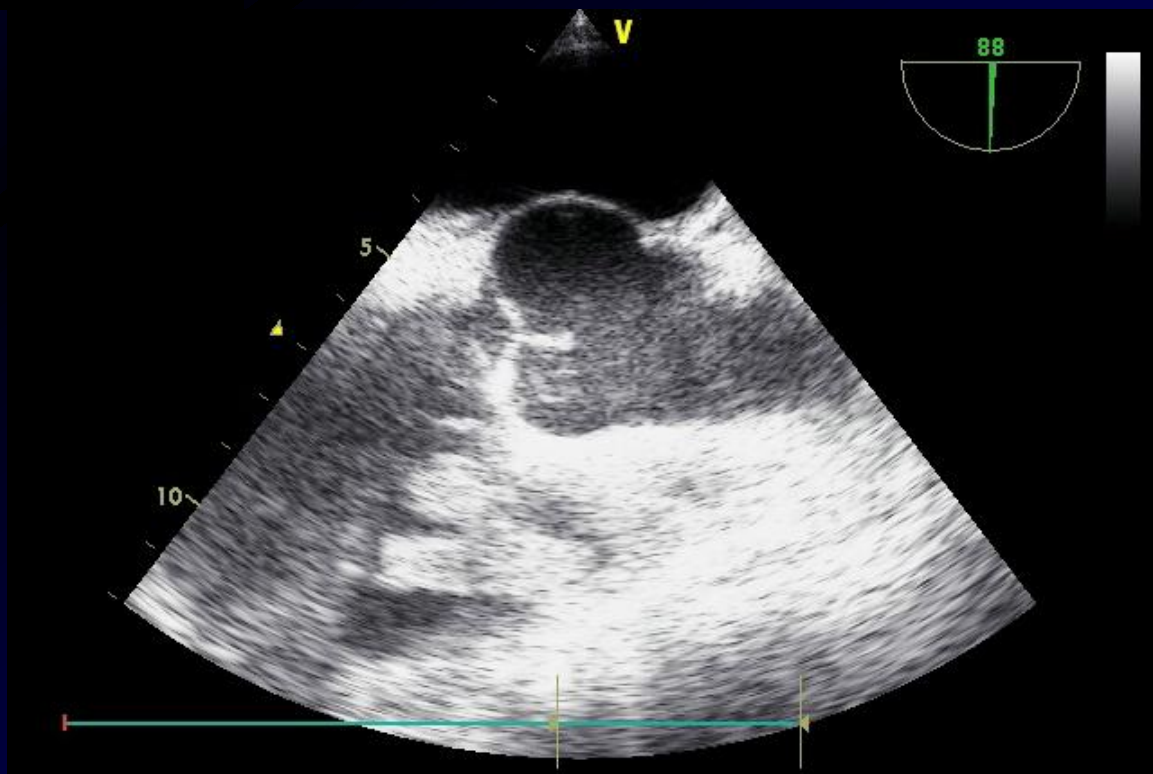
Ni trop tardif (AC) : risque d'encéphalopathie anoxique ou de défaillance multiviscérale

Ni trop facile (choc non réfractaire) : patients qui auraient guéri avec un traitement médical bien conduit = choc cardiogénique prouvé (IC ≤ 2,5 l/min/m^2) 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

Lancet 2007; 369: 1900

Case Report

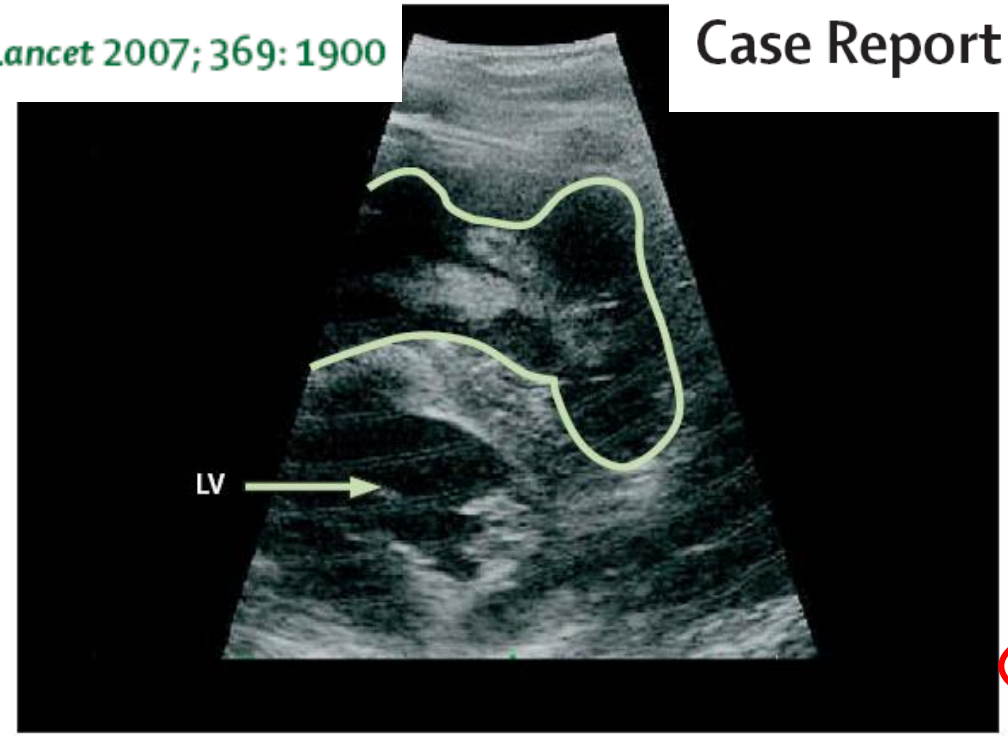


Figure: Echocardiography, showing AFE
The grossly enlarged right ventricle is outlined. At least three intracardiac masses are visible. The left ventricle (LV) is also visible.

3 précédentes grossesses : RAS

Déclenchement W (Misoprostol)

Rupture des membranes artificielle

Min 15 : coma, arrêt respiratoire

FV : CEE, MCE

CIVD ⇒ 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

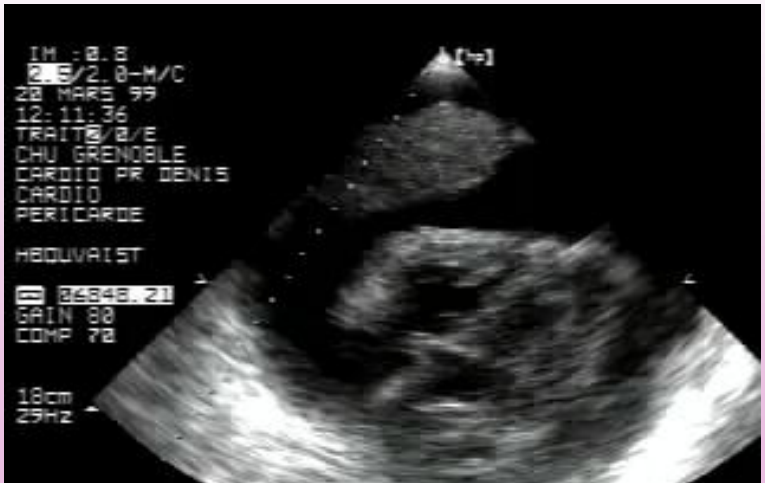
Choc réfractaire (FE_{VG} < 15%) : ECLS à H8

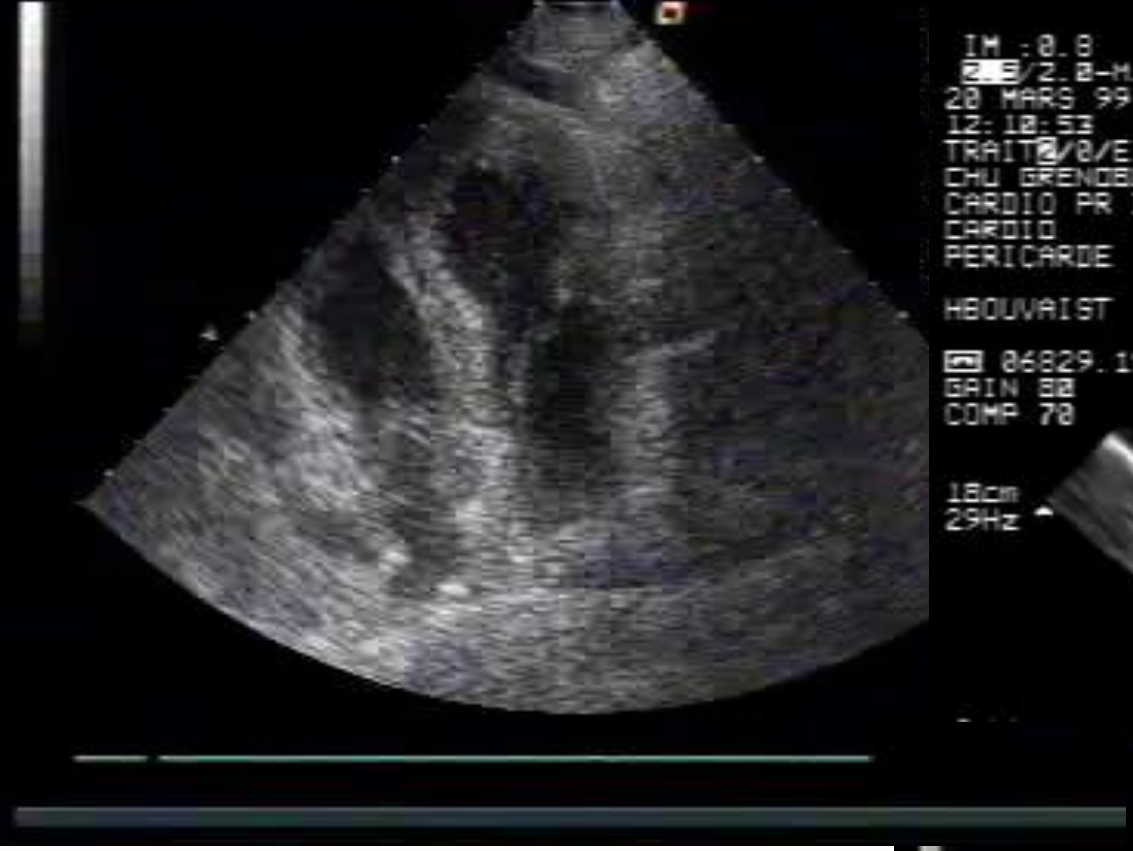
SIRS : corticoïdes (J12)

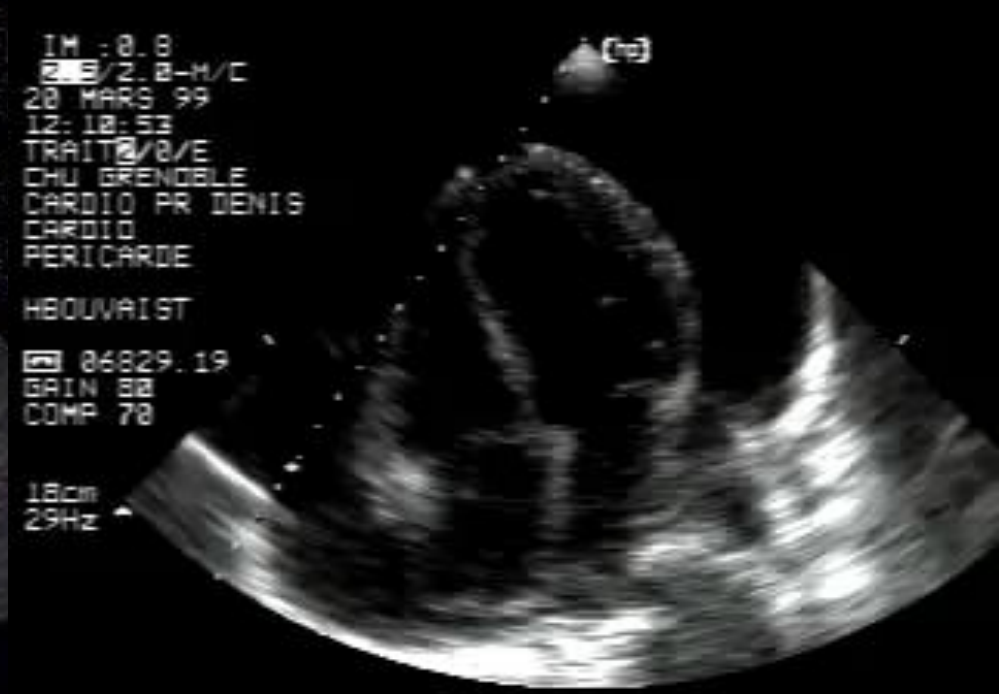
Décanulation J20, extubation J26

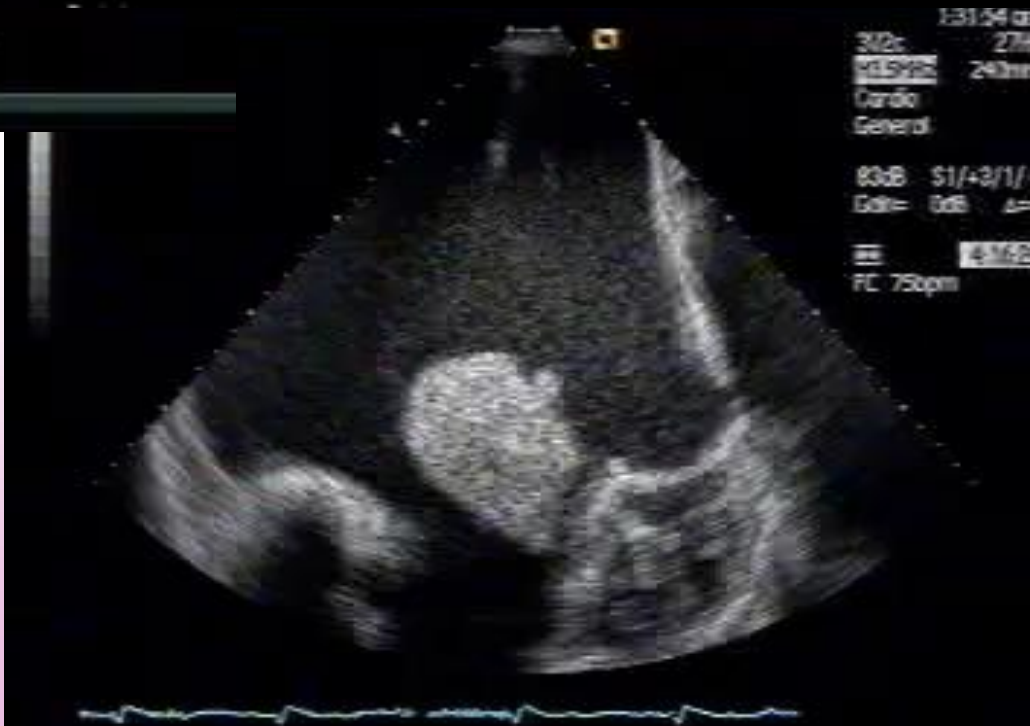
Sortie J40 (pas de séquelle)

Tamponnade


IM :0.8
2.5/2.0-M/C
20 MARS 99
12:11:36
TRAIT 0/E
CHU GRENOBLE
CARDIO PR DENIS
CARDIO
PERICARDE
HBOUVAIST
06848.21
GAIN 80
COMP 70
18cm
29Hz

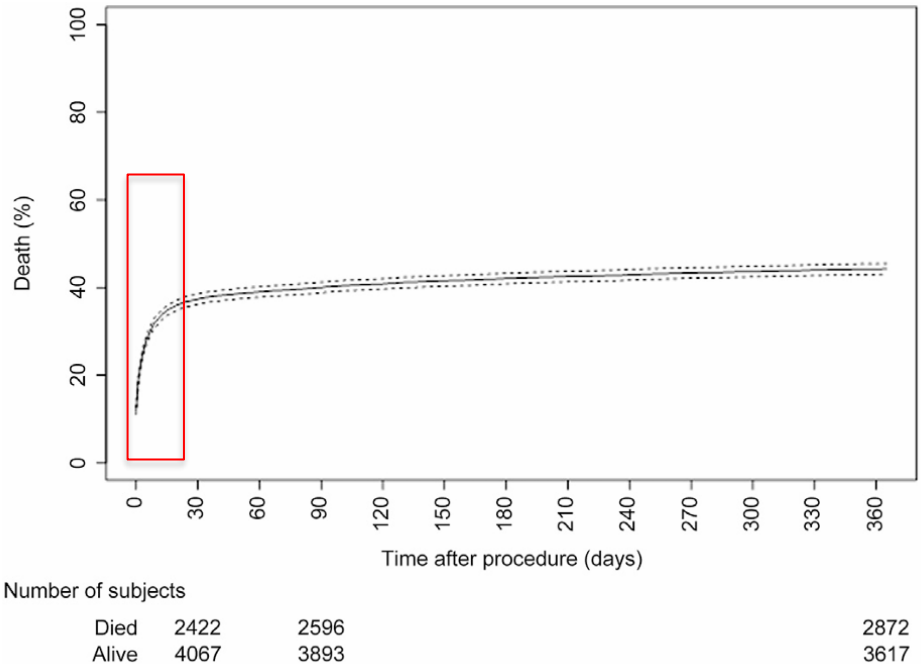



IM :0.8
2.5/2.0-M/C
20 MARS 99
12:10:53
TRAIT 0/E
CHU GRENOBLE
CARDIO PR DENIS
CARDIO
PERICARDE
HBOUVAIST
06829.19
GAIN 80
COMP 70
18cm
29Hz


Cardio
General
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

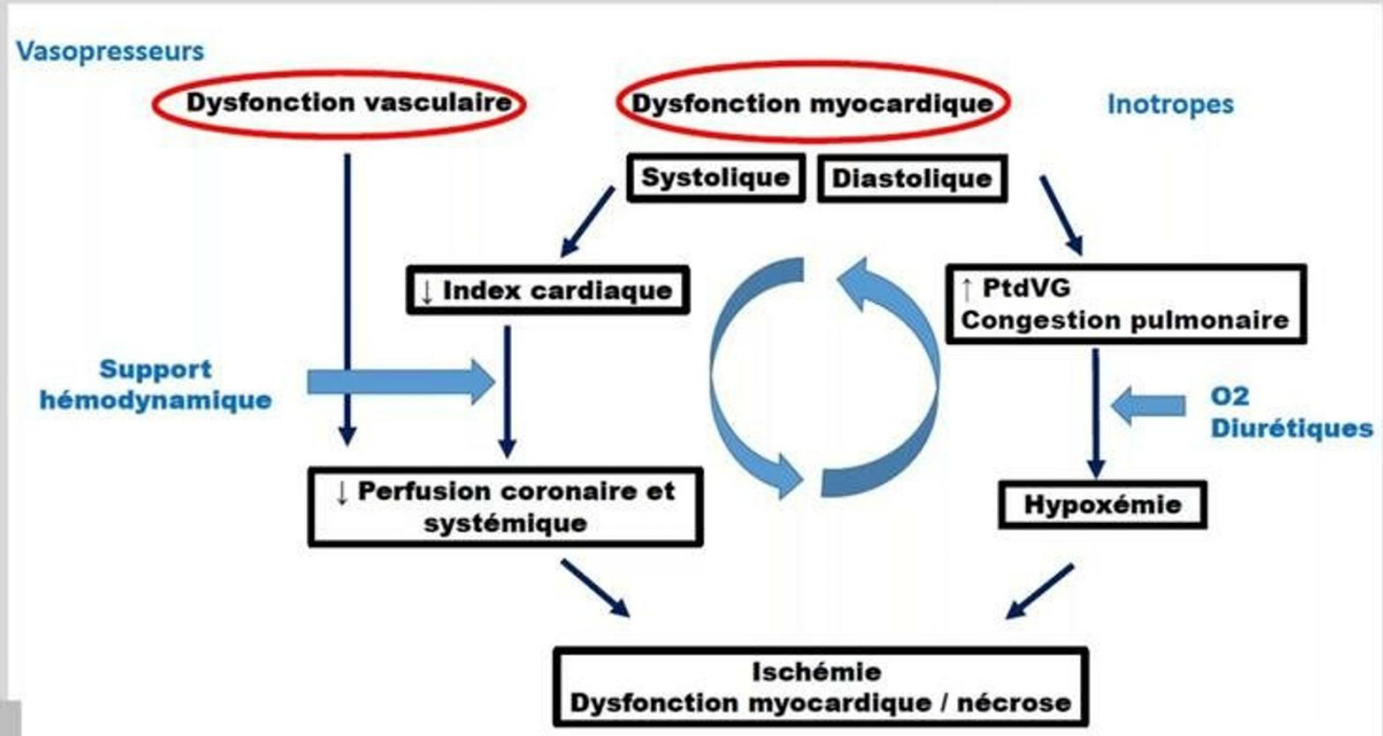
ESC 2014 = Classe IB
ESC 2016 = Classe IB
ESC 2017 = Classe IB

2. Inotrops
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



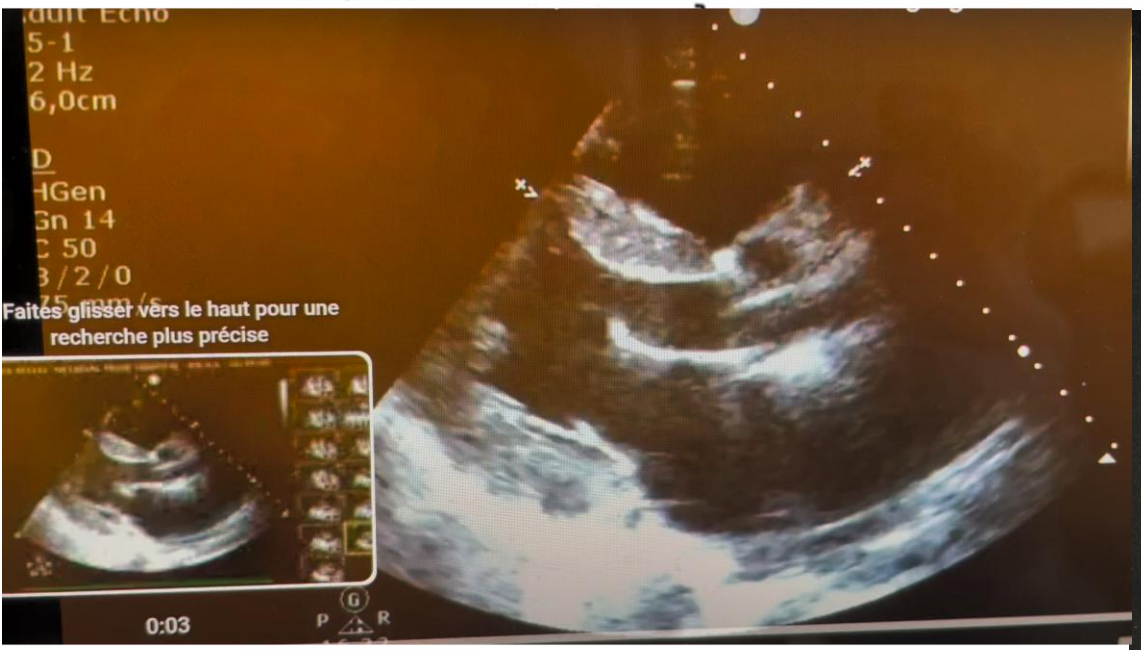
Delmas C et al, MIR 2017

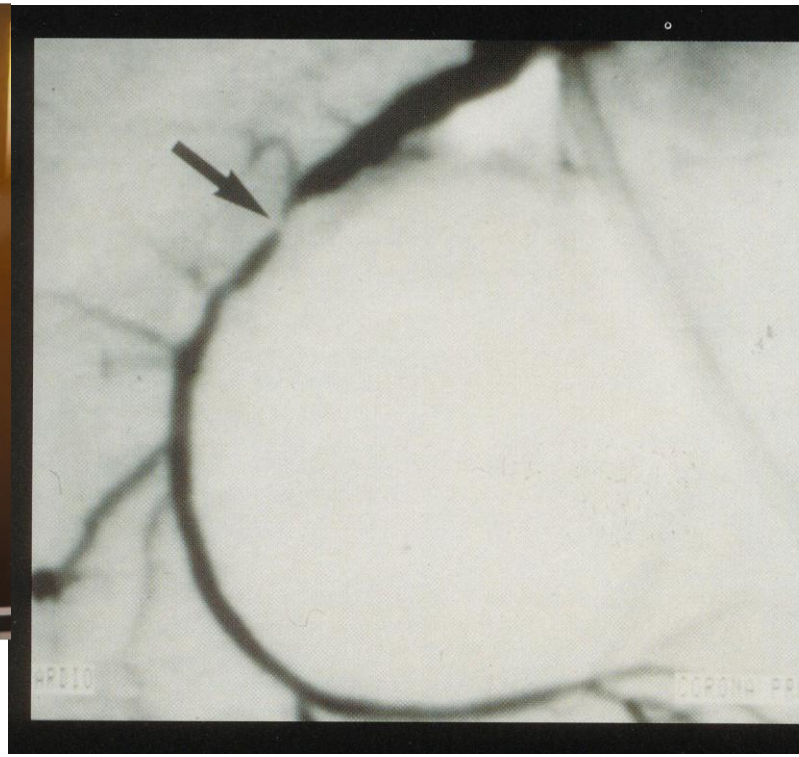
From C. Delmas (CHU de Toulouse)

Prise en charge immédiate

TRAITEMENT ETIOLOGIQUE +++

Causes of CS	Organ dysfunction	Immediate actions
ECG → ACS? +/- troponin		Cath lab
Echo: mechanical complications		Operating room





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

REVASCULARISATION CORON

Mebazaa et al. Acute heart failure and cardiogenic shock. *ICM 2016*

Prise en charge : revascularisation

Étude SHOCK (N=302) = shock +STEMI

Groupe revascularisation

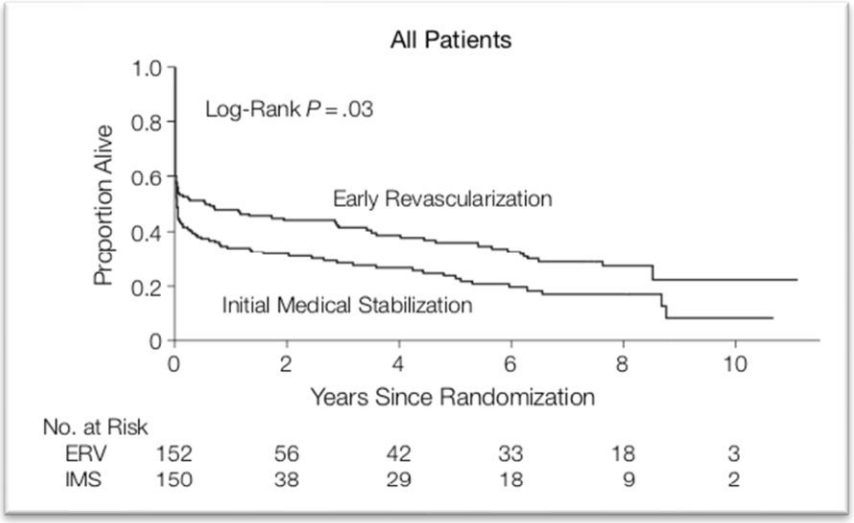
- ✓ Revascularisation < 6h
- ✓ Pontages ou angioplastie
- ✓ Ballon de contre-pulsion recommandé

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



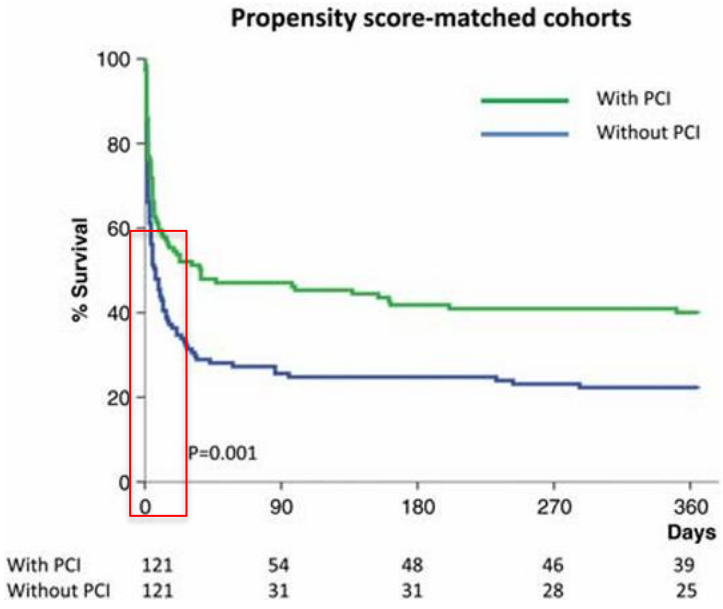
The NEW ENGLAND JOURNAL of MEDICINE

Hochman. *N Engl J Med 1999*
Hochman. JAMA 2006

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

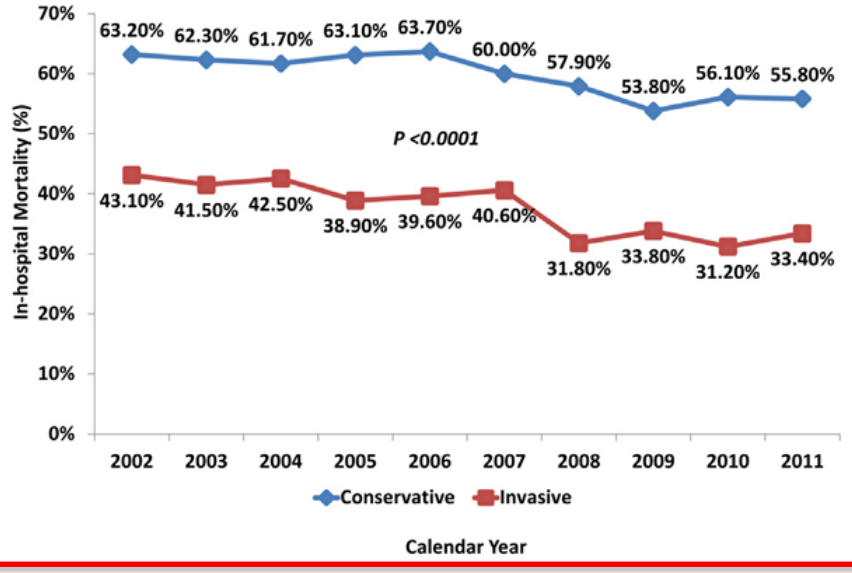
Improved outcome of cardiogenic shock at the acute stage of myocardial infarction: a report from the USIK 1995, USIC 2000, and FAST-MI French Nationwide Registrie

Assaoui. EHJ. 2012



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

Bangalore. Am J Med. 2015

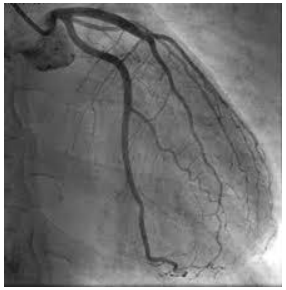


Recommendations	Class[a]	Level[b]
Immediate PCI is indicated for patients with cardiogenic shock if coronary anatomy is suitable. If coronary anatomy is not suitable for PCI, or PCI has failed, emergency CABG is recommended.[248]	I	B
Fibrinolysis should be considered in patients presenting with cardiogenic 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

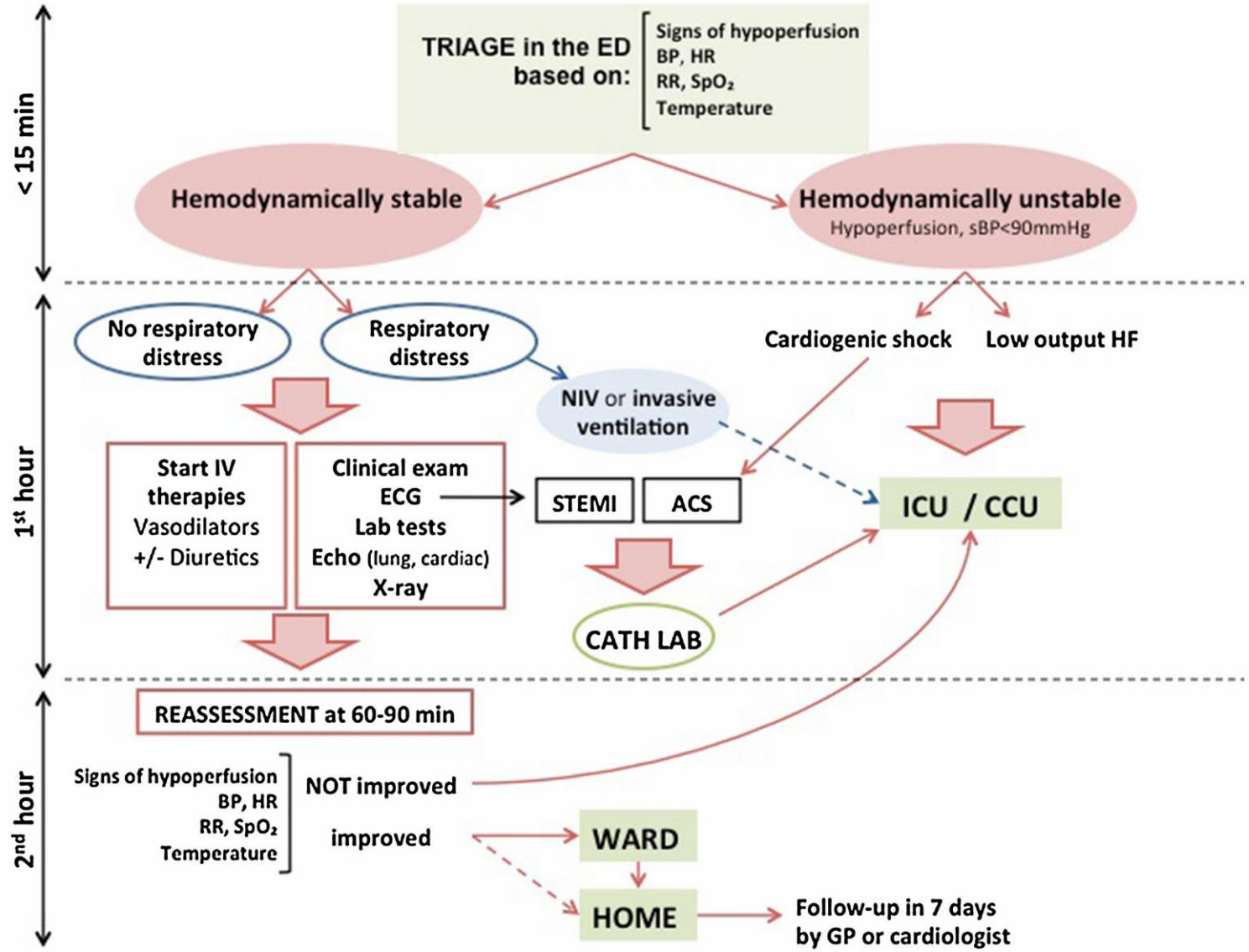
En cas de lésions multiples : traiter l'artère coupable



- ✓ 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%

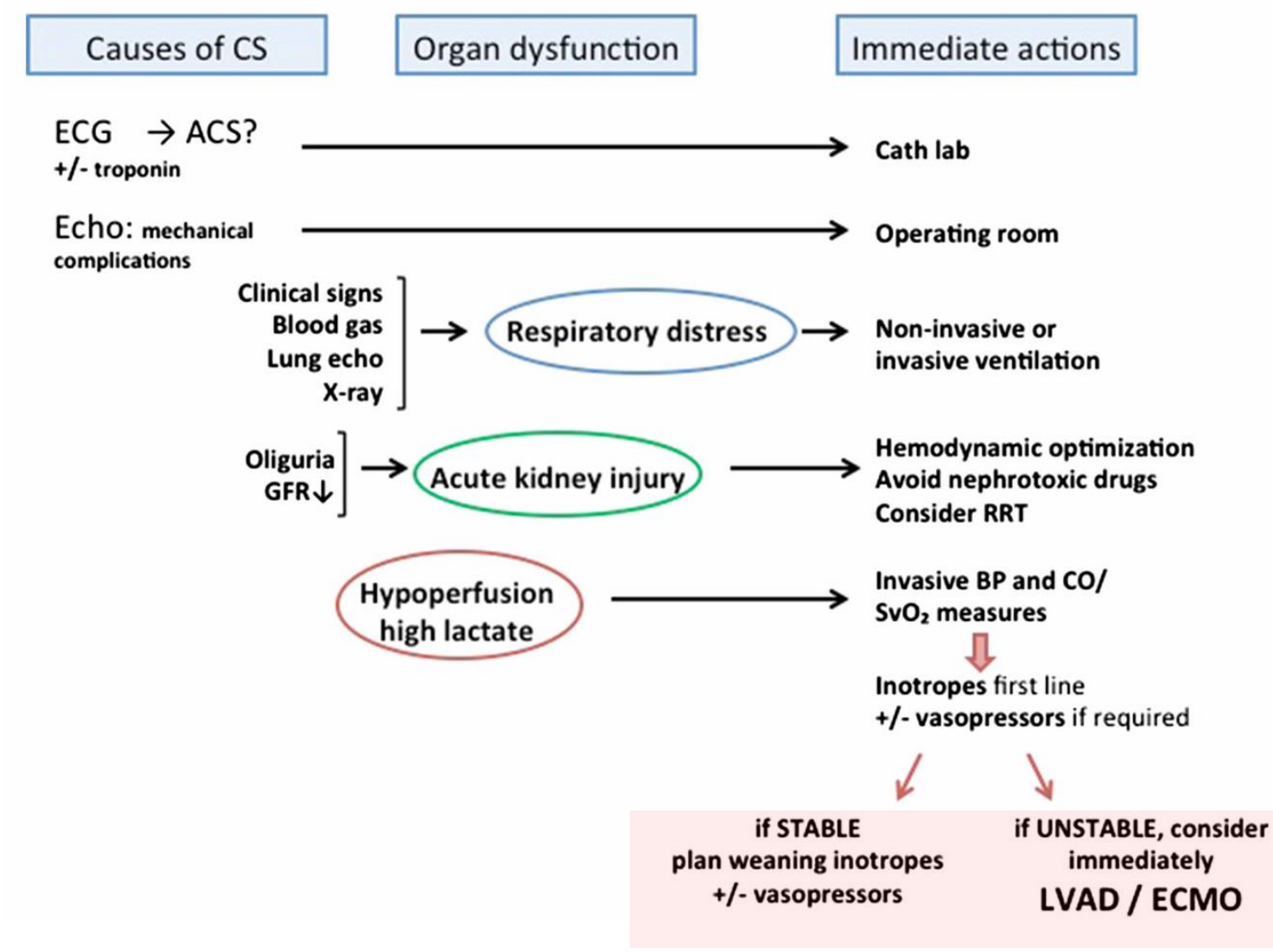
Thiele *N Engl J Med 2017*

Prise en charge immédiate



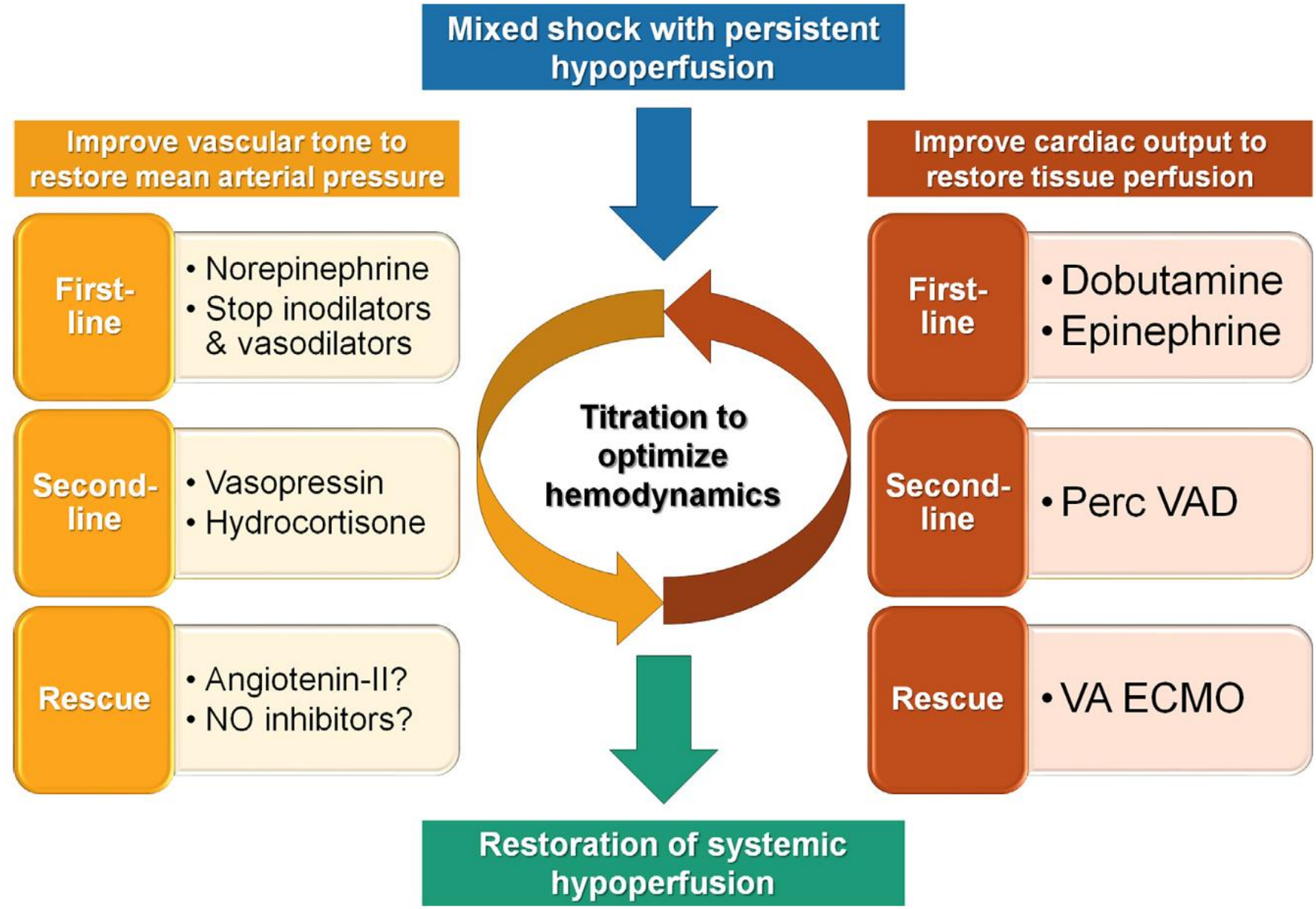
Mebazaa et al. Acute heart failure and cardiogenic shock. *ICM 2016*

Prise en charge immédiate



Mebazaa et al. Acute heart failure and cardiogenic shock. *ICM 2016*

FIGURE 3 Overview of Hemodynamic Support in Mixed Shock



Jentzer. *Mixed Cardiogenic Vasodilatary Shock. JACC 2025*

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	[illegible]
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	[illegible]
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 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

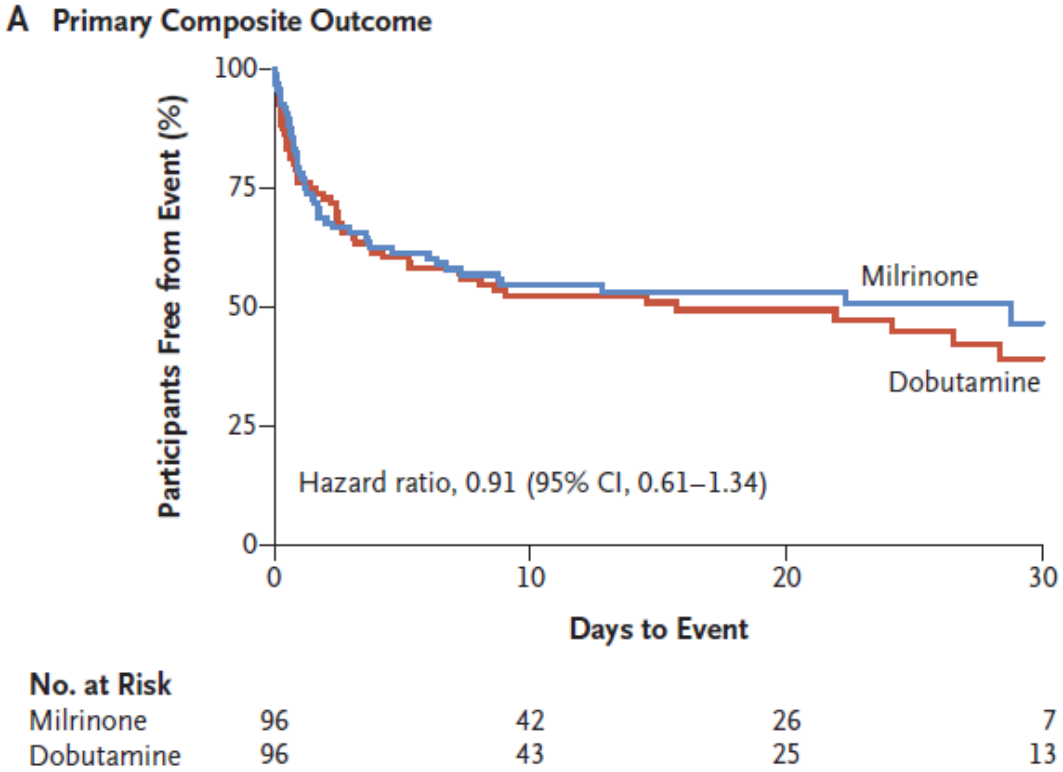
Inotrope idéal ?

- ✓ 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

Fellahi JL et al. *Anesthesiology*. 2013

Dobutamine versus milrinone



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

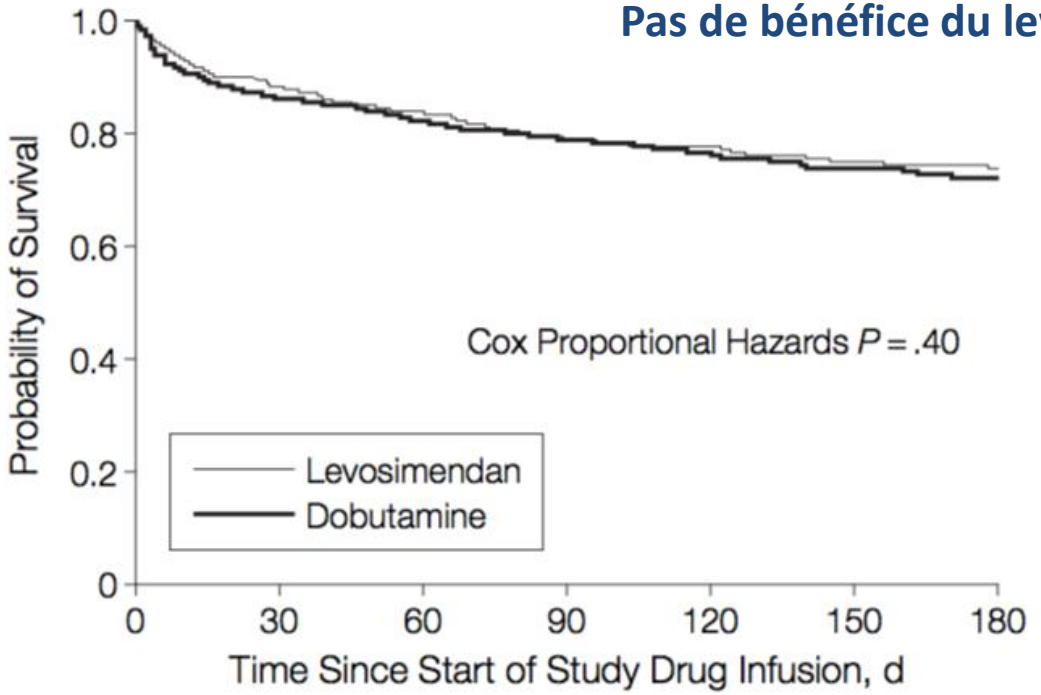
Pas de différence sur diurèse, lactate…

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

Mathew. *N Engl J Med*. 2021

Levosimendan versus Dobutamine

Pas de bénéfice du levosimendan

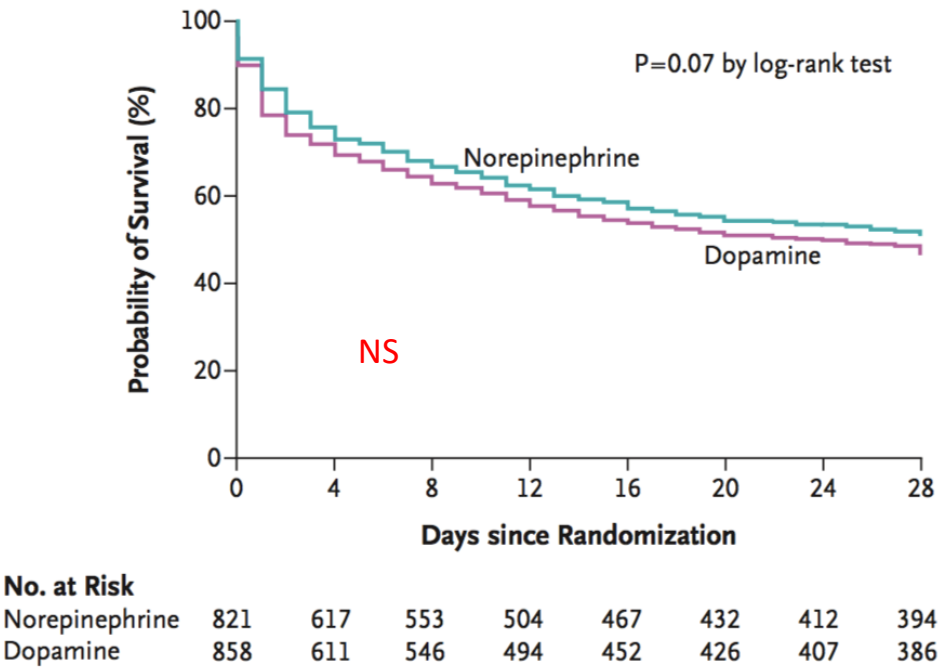


No. at Risk					
Levosimendan	664	608	586	525	462
Dobutamine	663	596	568	519	454

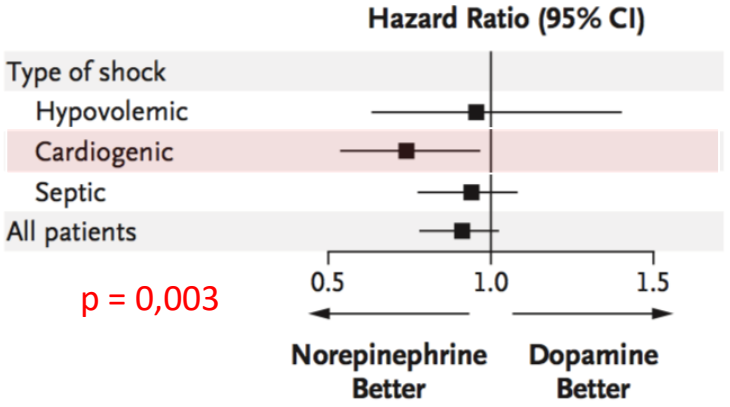
- ✓ Étude prospective multicentrique randomisée
- ✓ Dobutamine (N=663) versus levosimendan (N=664)

Mebazaa. JAMA. 2007

Quel vasopresseur ?



Vasopresseur = noradrénaline



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

De Backer D et al, *N Engl J Med* 2010

Noradrénaline versus Adrénaline

Étude OptimaCC

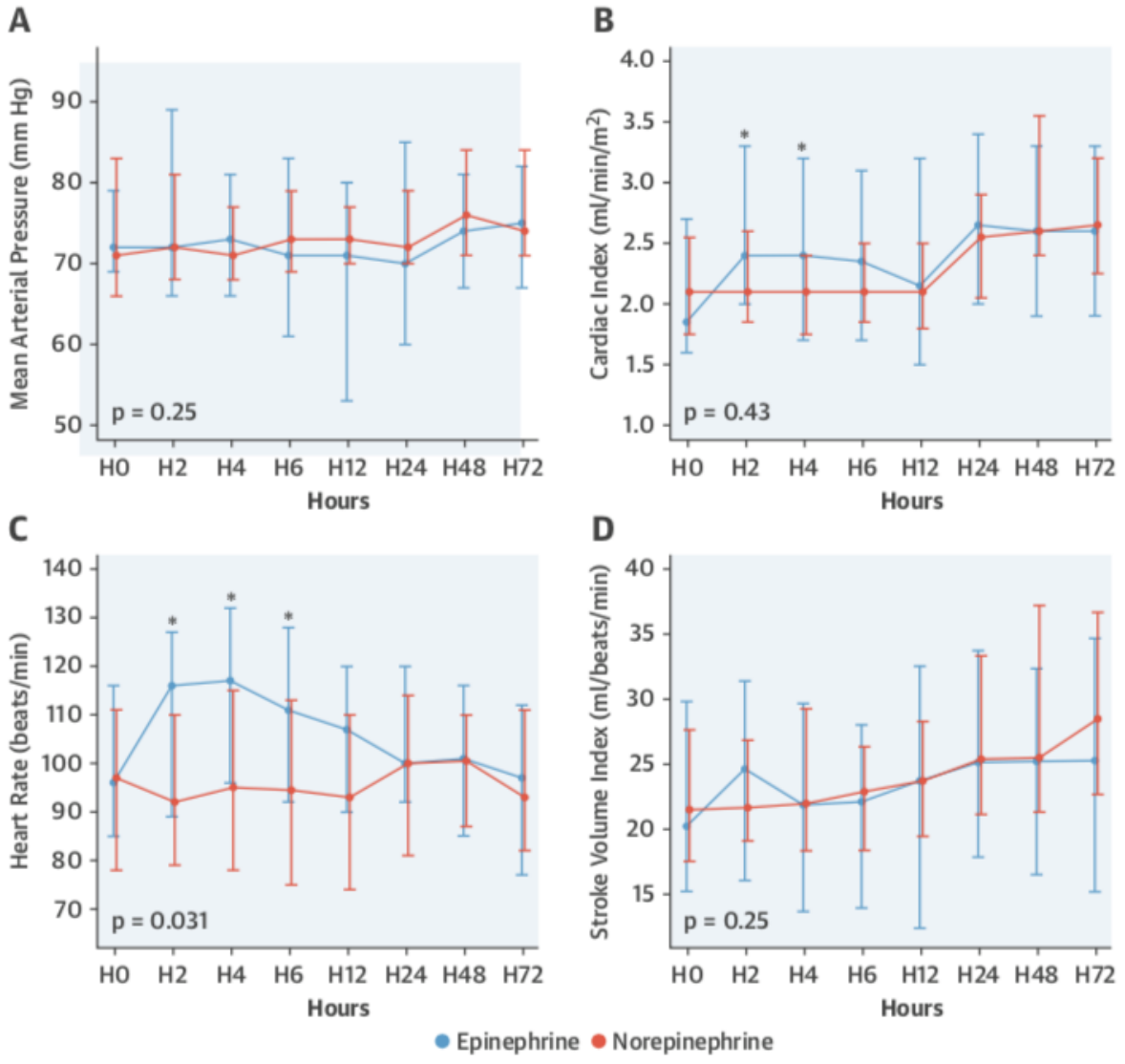
(N=57 CS + STEMI)

↓

Adrénaline (N=27)

Noradrénaline (N=20)

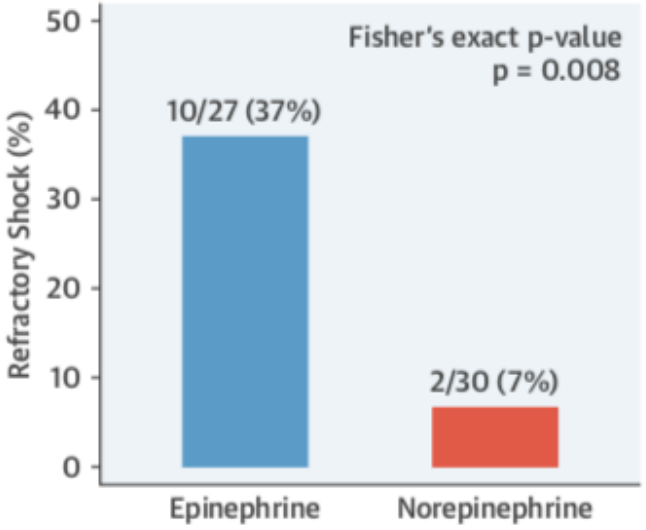
Critère de jugement principal : index cardiaque



Lévy B et al. *JACC* 2018

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

Lévy B et al. *JACC* 2018

Quelles catécholamines ?

REVIEW Open Access

CrossMark

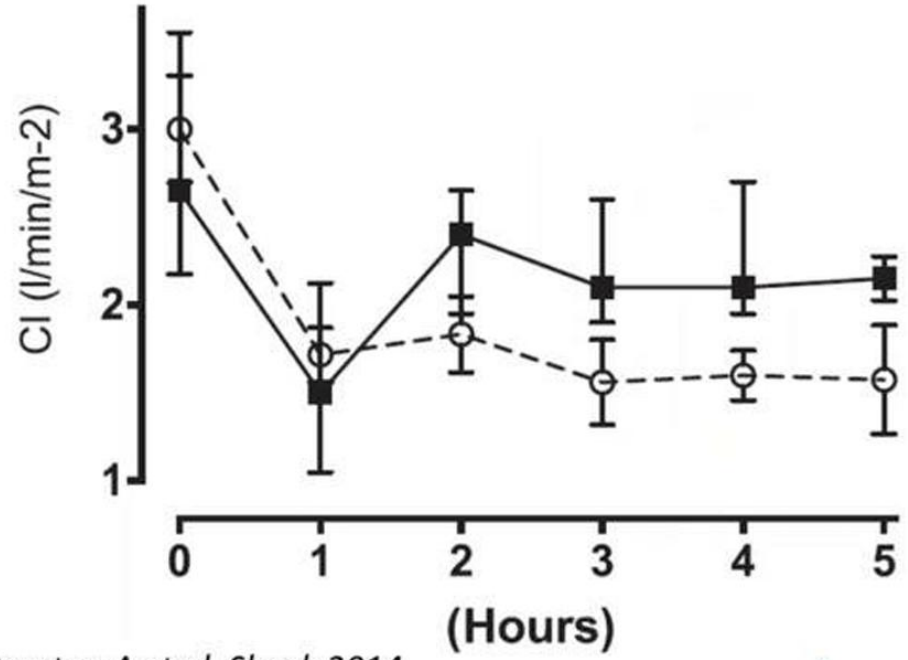
Experts' recommendations for the management of adult patients with cardiogenic shock

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**

Levy. AIC. 2015

Quelles catécholamines ?

In case of hypotension: Norepinephrine first !



Beurton.A et al, Shock 2014

Effets vasculaires α > action β1

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 analyzed patients	OR for short-term mortality [95% CI]
Age	<45 yo	170	3.48 [1.72 - 7.02]
	45-60 yo	558	3.09 [2.14 - 4.47]
	60-70 yo	664	4.13 [2.95 - 5.78]
	70-80 yo	719	3.67 [2.65 - 5.08]
	>80 yo	415	2.58 [1.66 - 4.02]
	Female	1217	3.86 [3.01 - 4.96]
	Male	1313	2.88 [2.28 - 3.63]
	non-ACS	779	2.91 [2.14 - 3.95]
	ACS	1563	3.65 [2.93 - 4.56]
HR	<90 bpm	812	4.95 [3.61 - 6.79]
	90-110 bpm	547	4.02 [2.75 - 5.87]
	>110 bpm	640	2.96 [2.11 - 4.16]
	High BP	1263	4.27 [3.33 - 5.48]
	Low BP	979	2.83 [2.16 - 3.72]
	High EF	1010	4.56 [3.43 - 6.06]
	Low EF	729	3.28 [2.39 - 4.51]
	Low NP	262	6.04 [3.31 - 11.02]
	High NP	258	2.31 [1.34 - 4.00]
	Low Trop	290	4.70 [2.61 - 8.45]
	High Trop	287	2.40 [1.45 - 3.96]
	Low MR	1466	1.90 [1.51 - 2.41]
	High MR	1117	4.79 [3.65 - 6.29]
	No cardiac arrest	524	2.05 [1.34 - 3.13]
	Cardiac arrest	464	1.78 [1.21 - 2.59]
eGFR	<60 mL/min/1.73 m²	795	1.84 [1.38 - 2.45]
	>60 mL/min/1.73 m²	1030	4.29 [3.24 - 5.70]
	No mechanical support	675	2.78 [1.94 - 3.98]
	Mechanical support	124	1.09 [0.52 - 2.27]

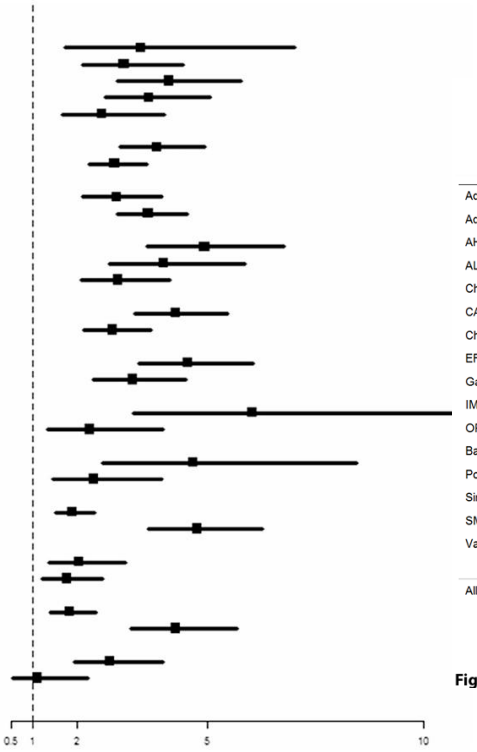


Fig. 5 Forest plot of the meta-analysis of short-term mortality, with subgroup analysis. ACS acute coronary syndrome, HR heart rate, BP blood pressure (low BP, MAP < 65 mmHg or SBP < 90 mmHg), EF ejection fraction, NP natriuretic peptide, Trop troponin, MR mortality rate. Low/high cutoff was defined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

	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 - 13.29]
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]
Simonis, 2012	89	25	1.37 [0.53 - 3.55]
SMASH, 1998	111	41	0.62 [0.26 - 1.47]
Valente, 2011	152	34	2.40 [0.38 - 14.96]
All studies	2583	947	3.33 [2.81 - 3.94]

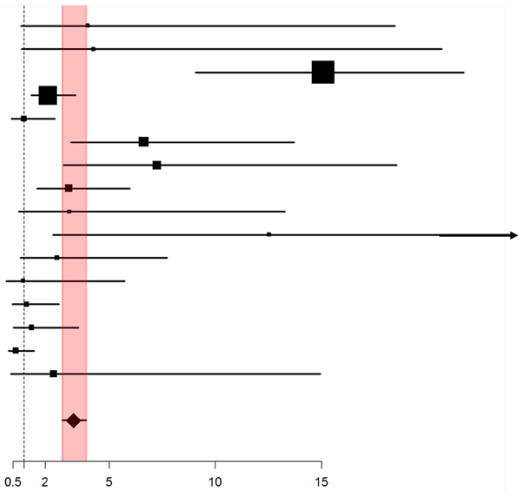
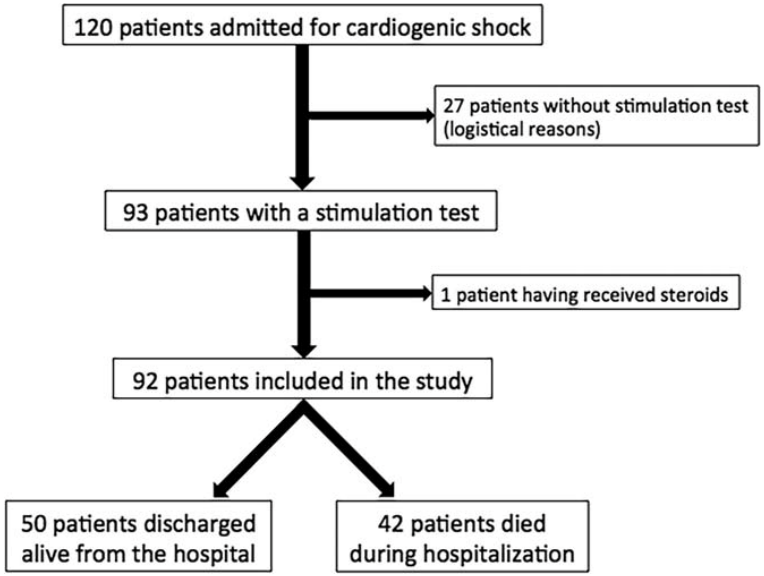
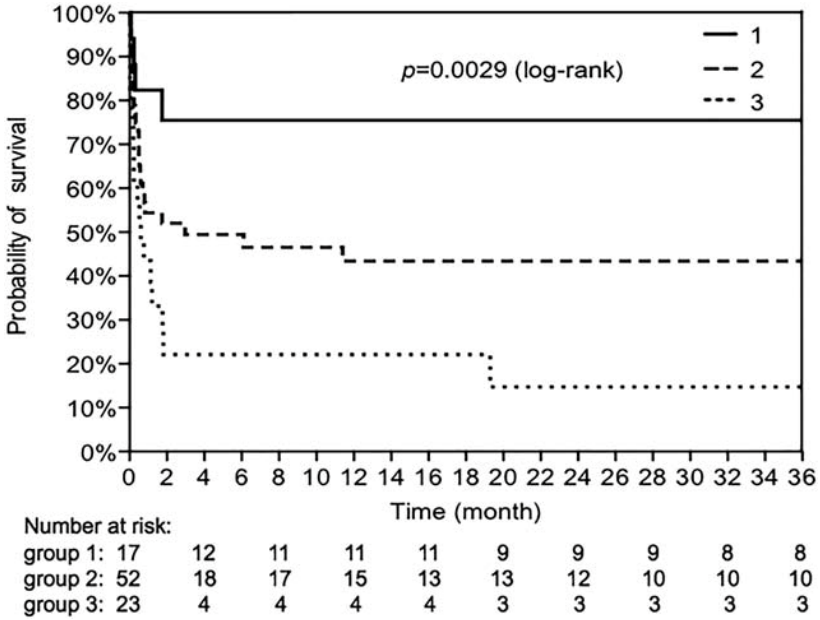


Fig. 3 Forest plot of the meta-analysis of short-term mortality

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

Bagate F. *Shock*. 2017

Insuffisance surrénale : supplémentation ?

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

	Hazard ratio (95% confidence interval); *P*	
Variables	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 Δmax; Group 2, lower T0 and lower Δmax or higher T0 and higher Δmax; Group 3, higher T0 and lower Δmax; 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

Bagate F. *Shock*. 2017

Prise en charge non médicamenteuse : O2 / VM

Place for Oxygen supplementation +++

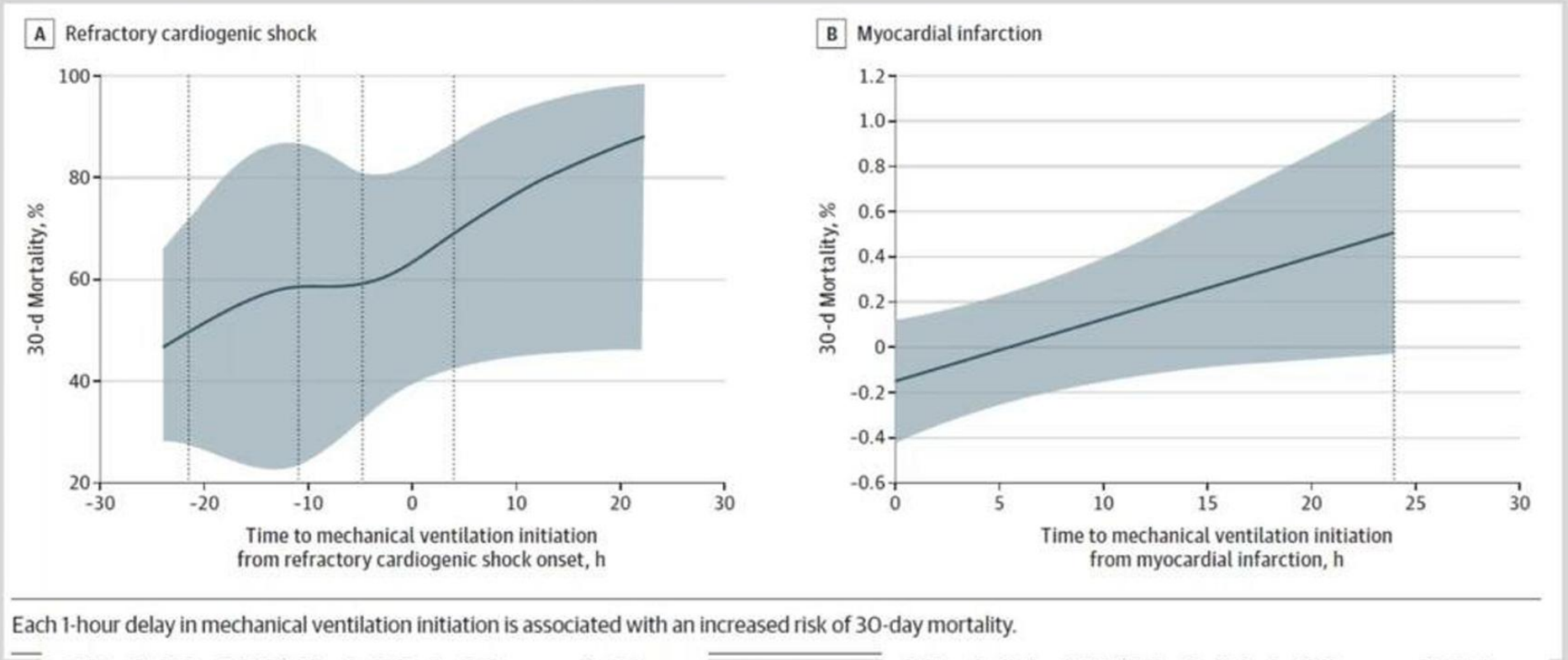
Oxygen and ventilatory support		
Oxygen is recommended in patients with SpO_2 <90% or PaO_2 <60 mmHg to correct hypoxaemia.	I	C
Intubation is recommended for progressive respiratory failure persisting in spite of oxygen administration or non-invasive ventilation.[448]	I	C
Non-invasive positive pressure ventilation should be considered in patients with respiratory distress (respiratory rate >25 breaths/min, SpO_2 <90%) and started as soon as possible in order to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation.[448]	IIa	B

Mc Donagh. *EHJ. 2021*

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

Prise en charge non médicamenteuse : O2 / VM

What timing to initiate MV in CS?



Van Diepen. *JAMA Cardiol. 2020*

ETUDE TRIUMPH : 65% sous VM

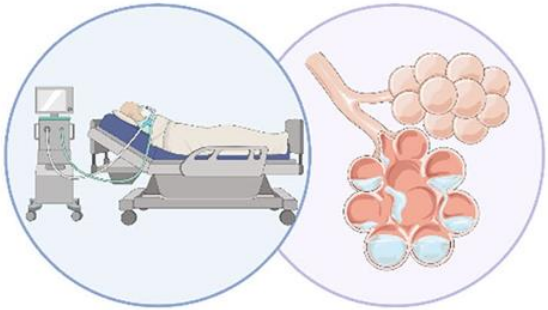
Chaque heure de délai de ventilation mécanique est associée au risque de mortalité à J30

Prise en charge non médicamenteuse : O2 / VM

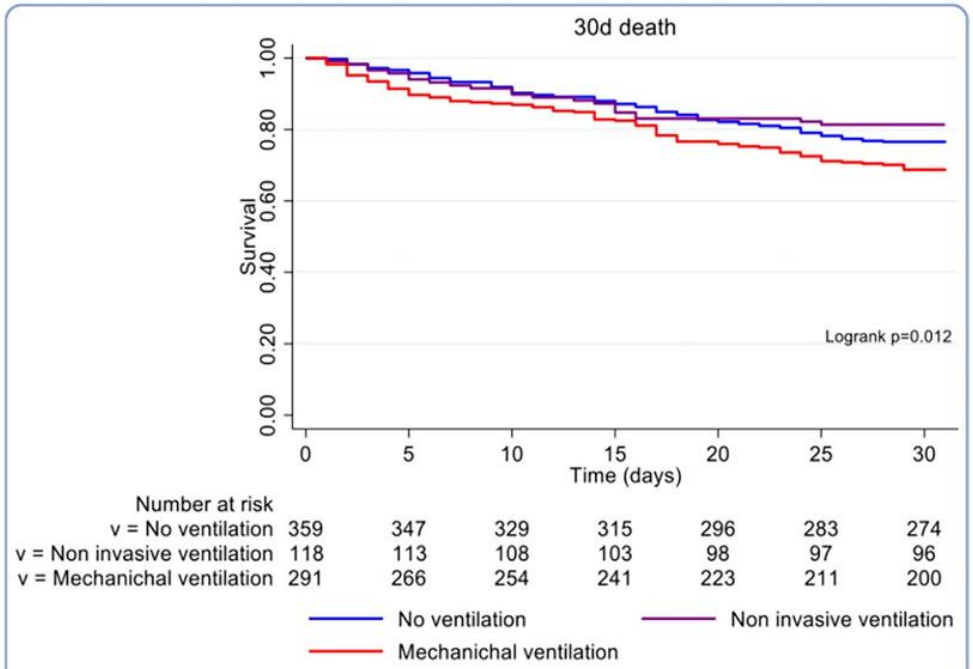
Ventilation strategies in cardiogenic shock: insights from the FRENSHOCK observational registry

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 ≥ 2,2 - 2,5L/Min/m2

- Amélioration perfusion d'organes
- Amélioration perfusion myocarde

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

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

From C. Delmas (CHU de Toulouse)

SURVEILLANCE : A TOUT INSTANT

Overload | **Malperfusion** | **Pain/Anxiety**

Monitoring +++

Arterial line, Central venous catheter, urinary tube

+

Telemetry, arterial BP, pulse oxymetry, respiratory rate

I C

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

IIb B

Clinical

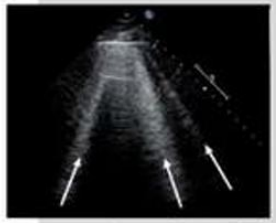
- Left and right overload signs
- Diuresis +++
- Marbles
- Confusion,…

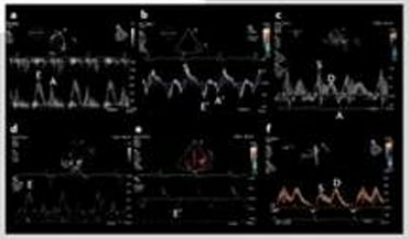
Biological

- 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
- …



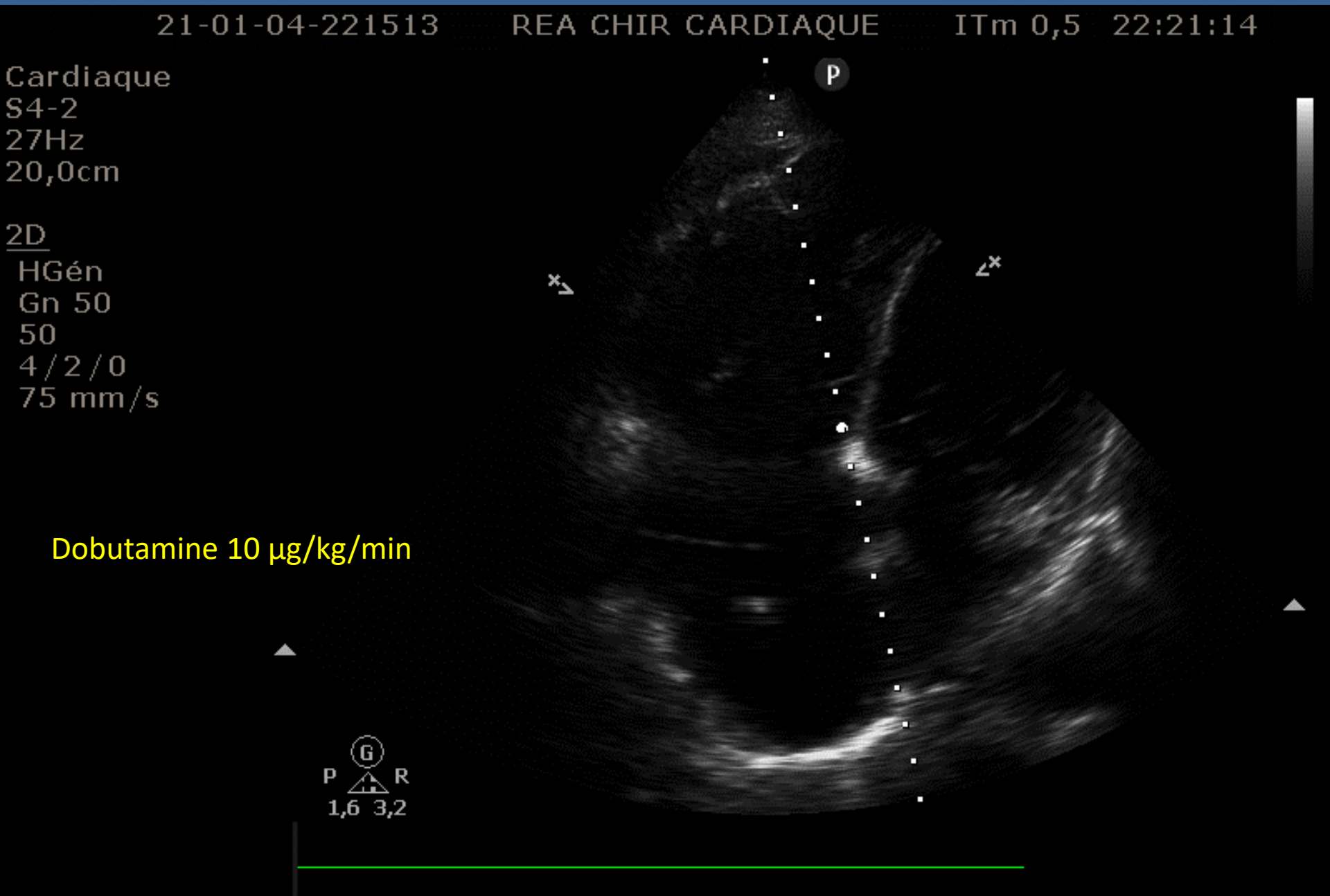


I C

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

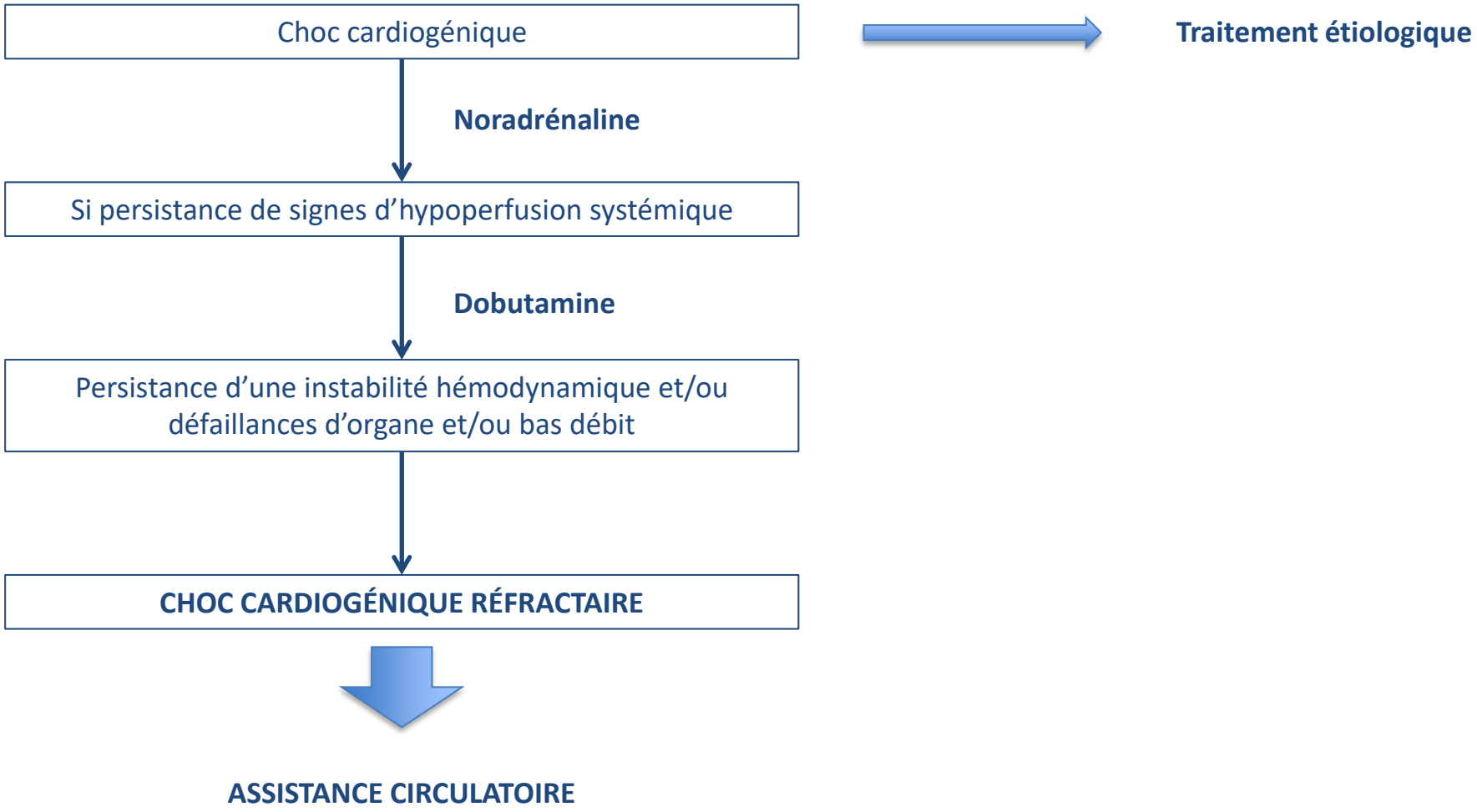
From C. Delmas (CHU de Toulouse)

Prise en charge médicamenteuse = insuffisante



Dobutamine 10 µg/kg/min

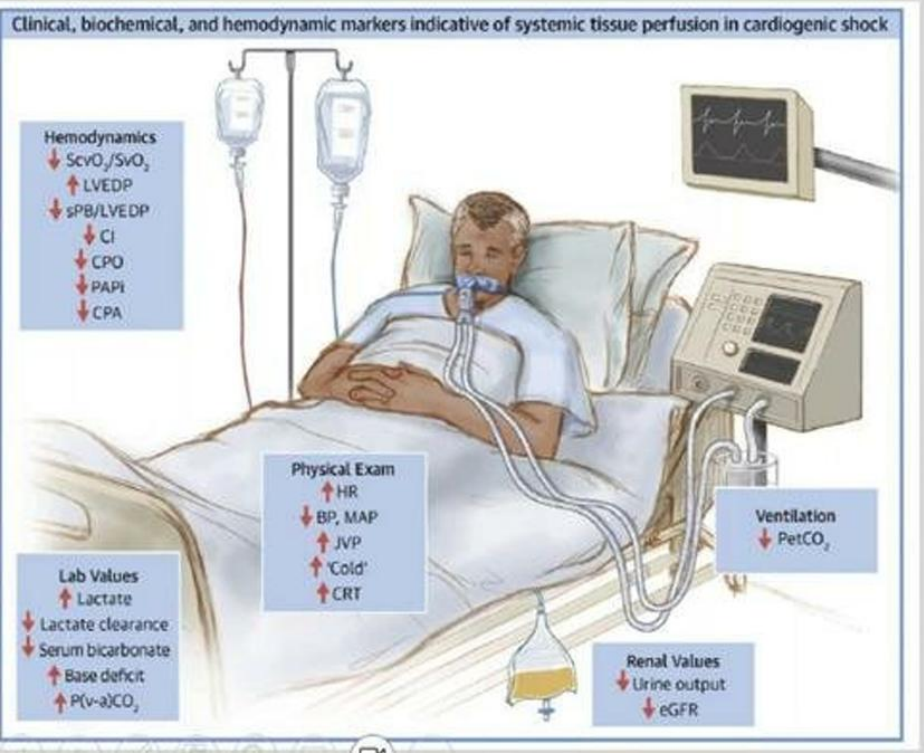
Prise en charge non médicamenteuse



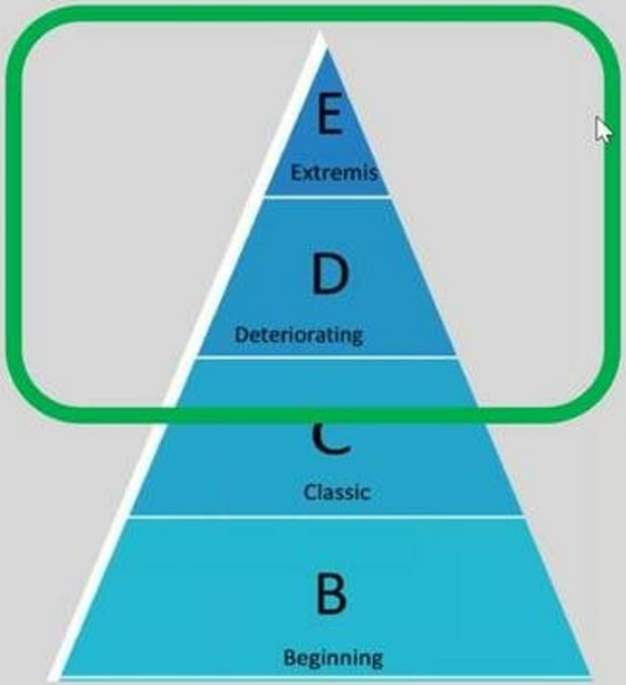
Prise en charge non médicamenteuse

When to think about aMCS ?

Persistant malperfusion despite initial management

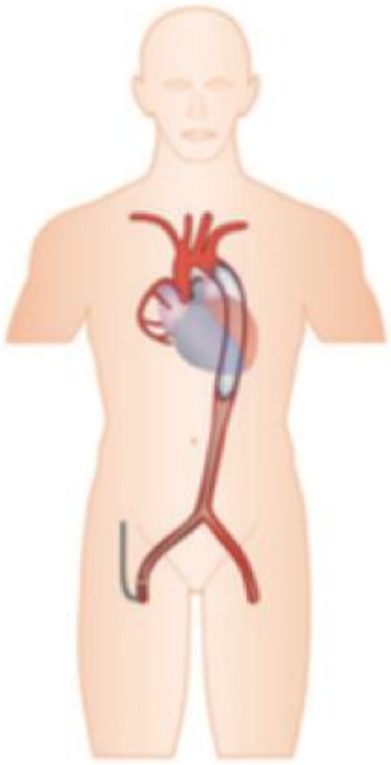


Degradation or Severe/profound initial CS presentation

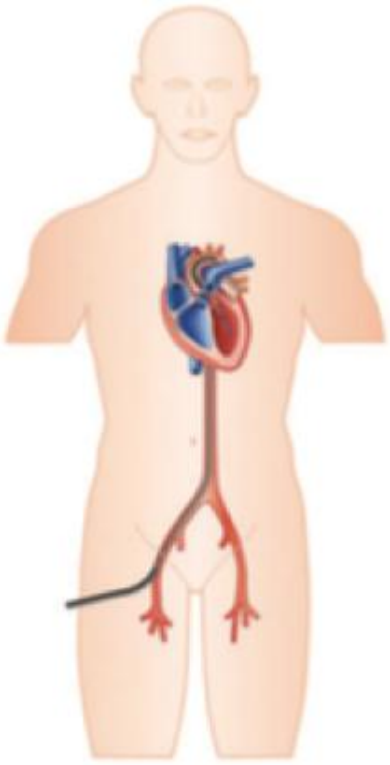


Mathew. JACC Advances. 2022

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

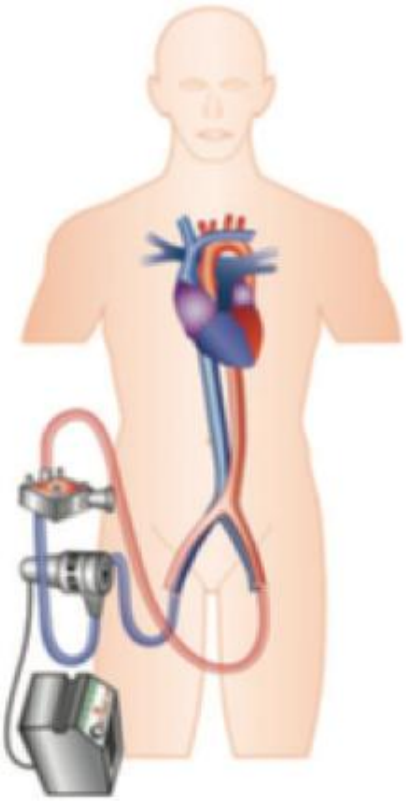


Ballon de contre-pulsion intra aortique



Impella

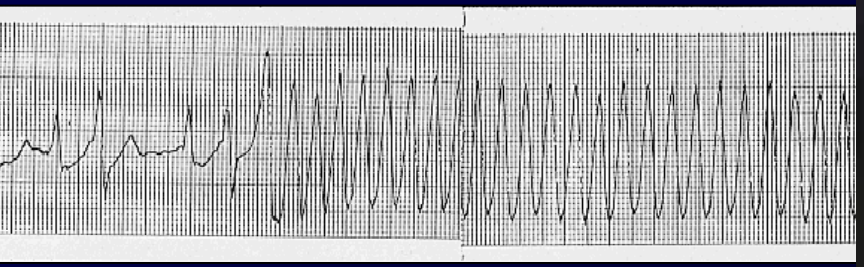
TandemHeart

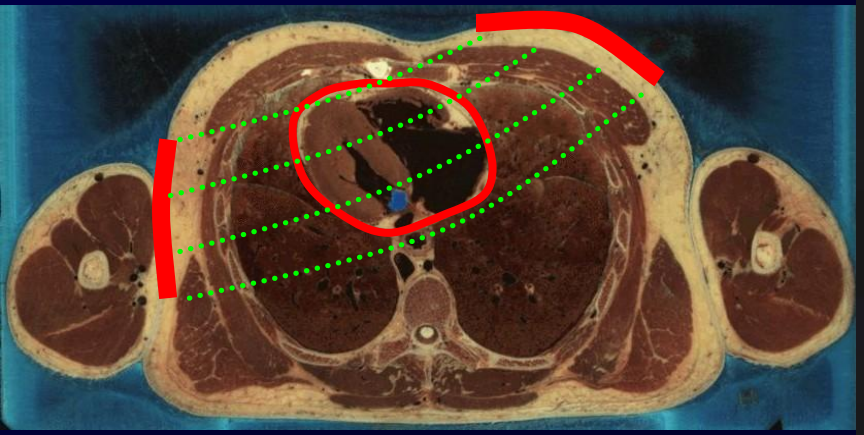


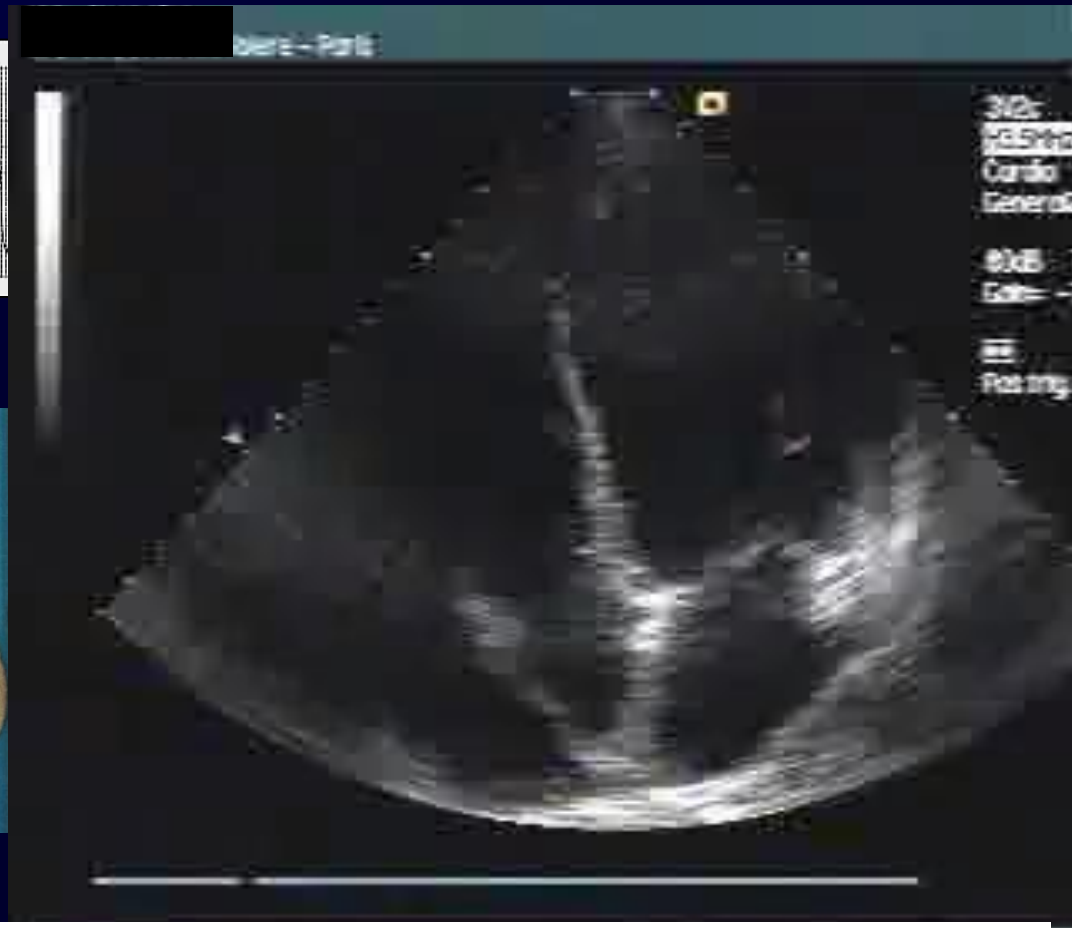
ECMO veino-artérielle

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

Resuscitation (2007) 73, 309–313

ELSEVIER

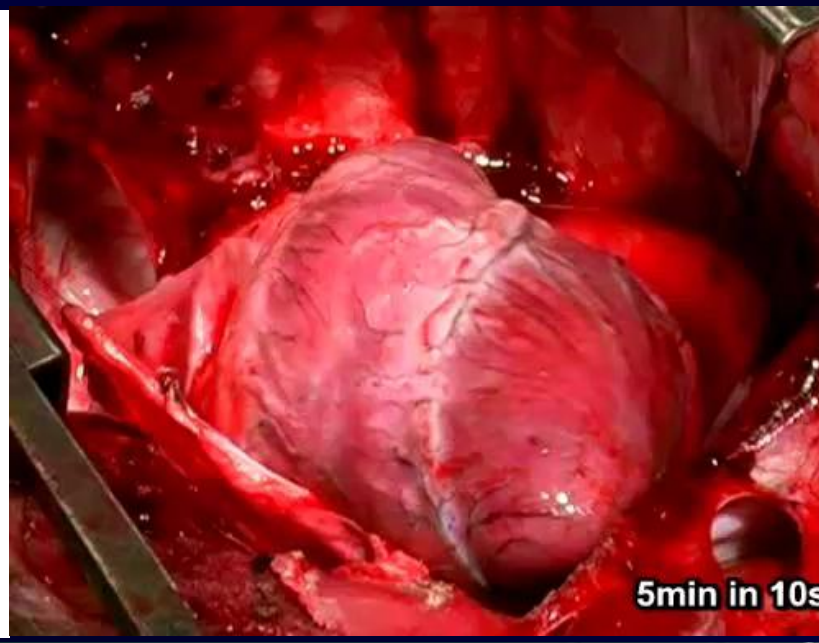
RESUSCITATION

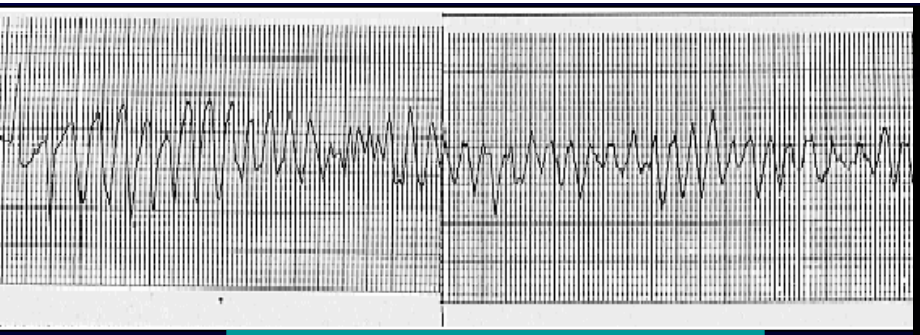
www.elsevier.com/locate/resuscitation

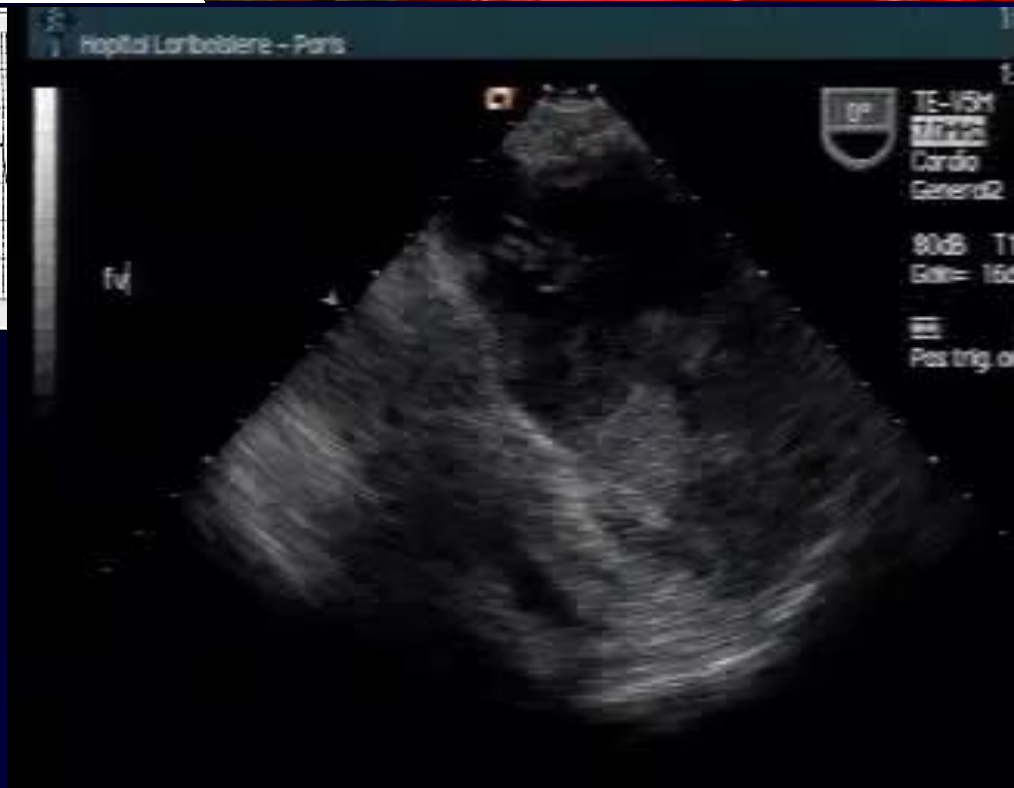
CASE REPORT

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

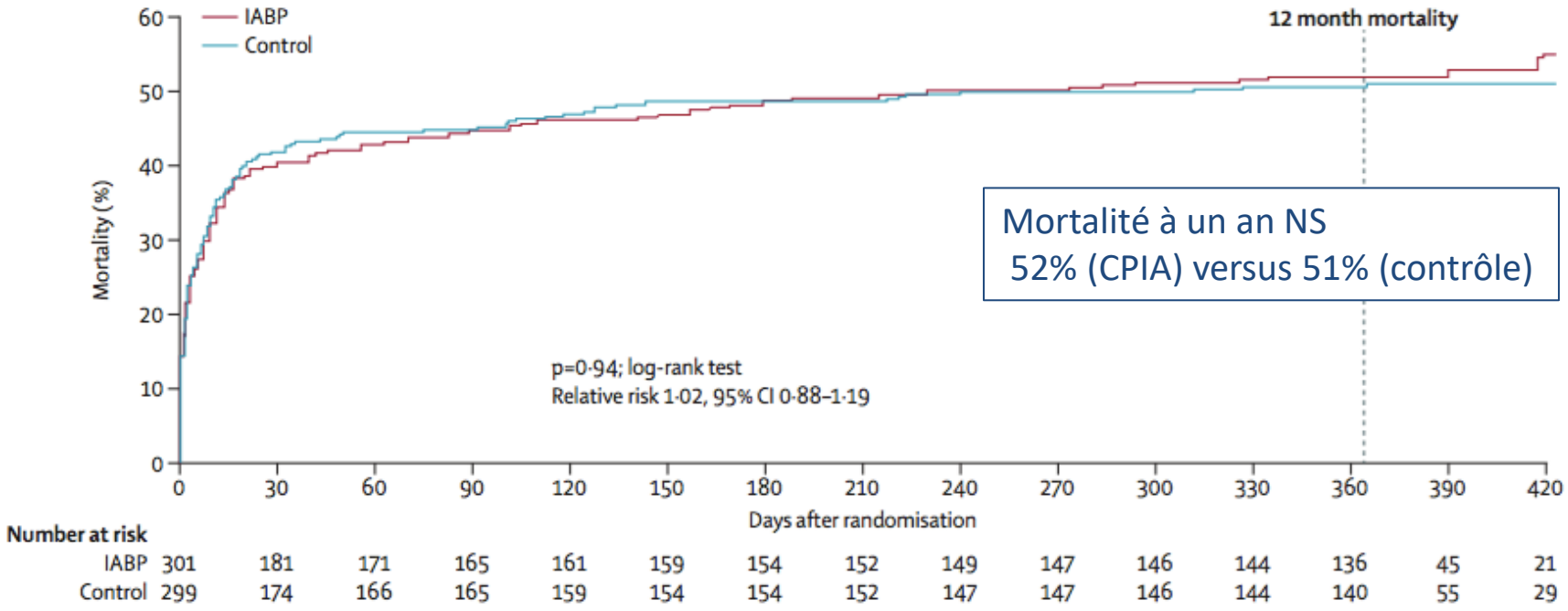
Jae-Seung Shin[a], Sung-Woo Lee[b,*], Gap-Su Han[b], Won-Min Jo[a], Sung-Hyuk Choi[b], Yun-Sik Hong[b]







Ballon de contre-pulsion intra aortique



Mortalité à un an NS
52% (CPIA) versus 51% (contrôle)

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

Thiele H et al, *Lancet* 2013;382(9905):1638-45

Ballon de contre-pulsion intra aortique

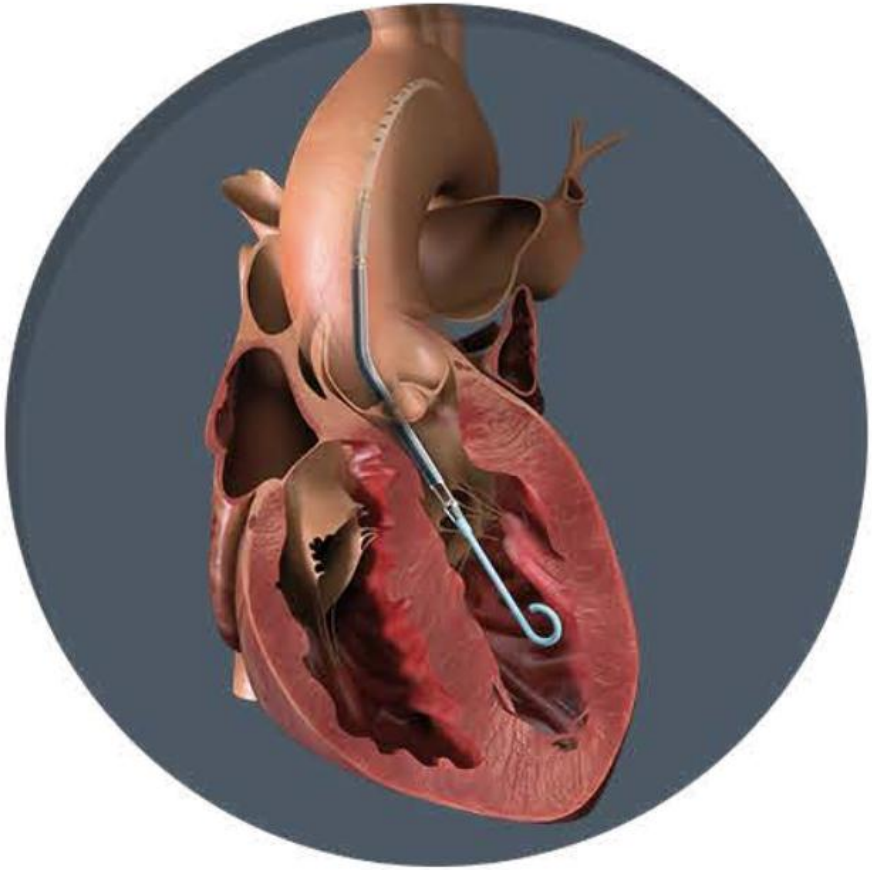
Recommendations	Class [a]	Level [b]	Ref [c]
In all patients with suspected cardiogenic shock, immediate ECG and echocardiography are recommended.	I	C	
All patients with cardiogenic shock should be rapidly transferred to a tertiary care center which has a 24/7 service of cardiac catheterization, and a 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	
Continous 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

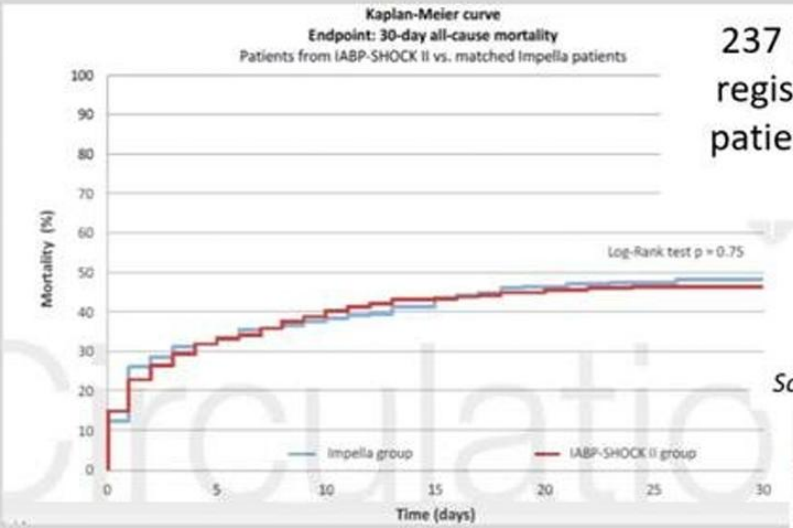
- ✓ Implantation endovasculaire
- ✓ Position transvalvulaire aortique
- ✓ Aspire le sang du VG pour le réinjecter dans l'aorte



- ✓ **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

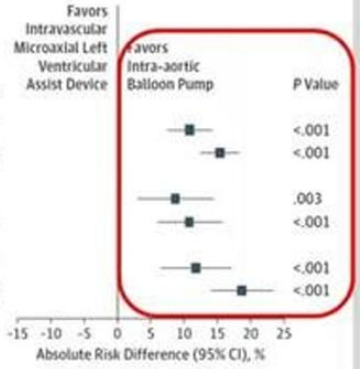
Schrage.B et al, Circulation 2018

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

We need RCT !

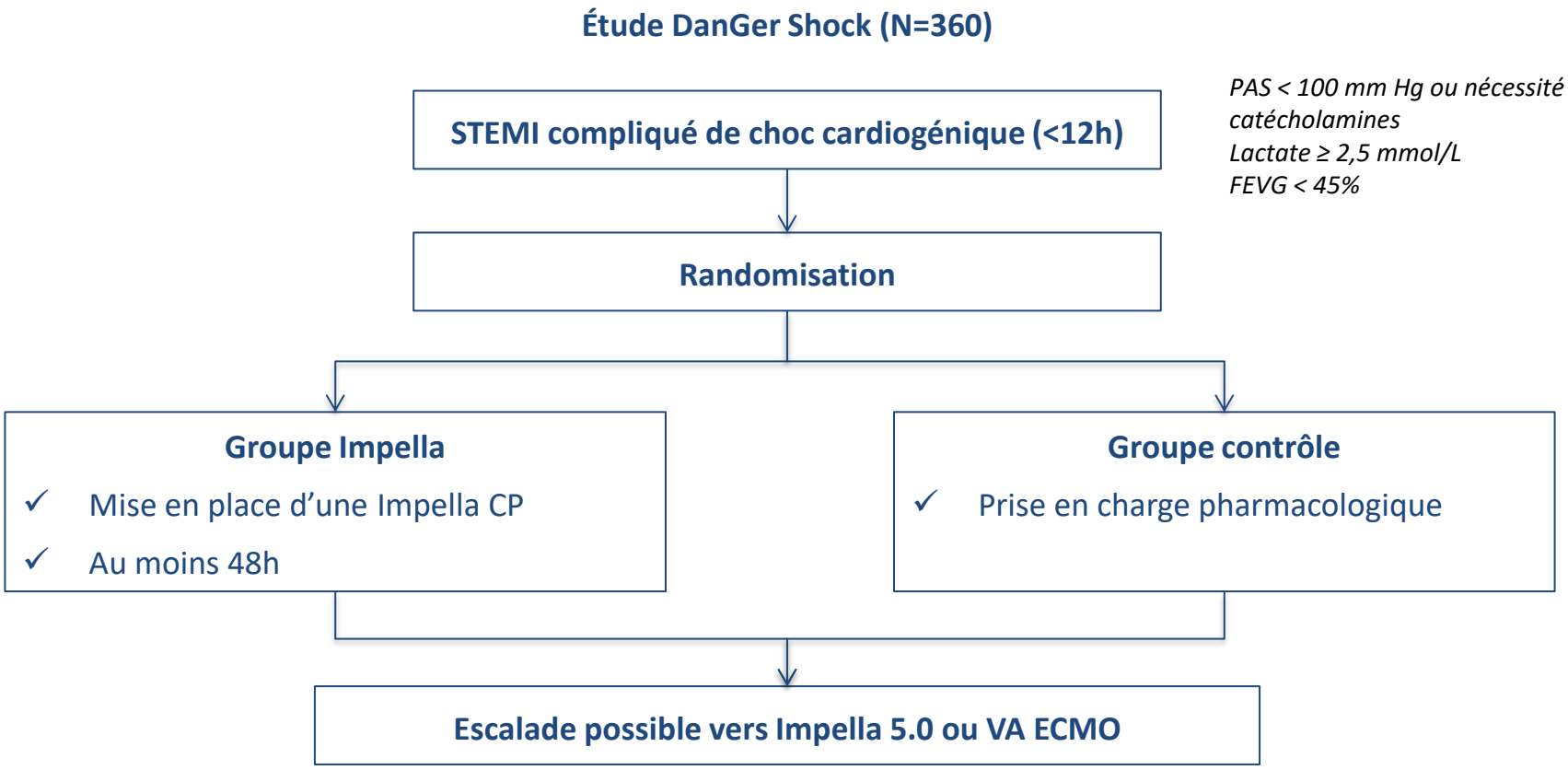
Figure 2. In-Hospital Outcomes Among Propensity-Matched Patients With Acute Myocardial Infarction Complicated by Cardiogenic Shock Undergoing Percutaneous Coronary Intervention With Intravascular Microaxial Left Ventricular Assist Device vs Intra-aortic Balloon Pump

	Intravascular Microaxial Left Ventricular Assist Device		Intra-aortic Balloon Pump		Absolute Risk Difference (95% CI), %
	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)
Major bleeding	526	31.3	268	16.0	15.4 (12.5-18.2)
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)
Major bleeding	157	27.4	95	16.6	10.8 (6.1-15.6)
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)
Major bleeding	228	34.4	104	15.7	18.7 (14.2-23.3)



Miller.PE JAMA Intern Med

Impella (IABP)



Critère de jugement principal : mortalité à 180 jours

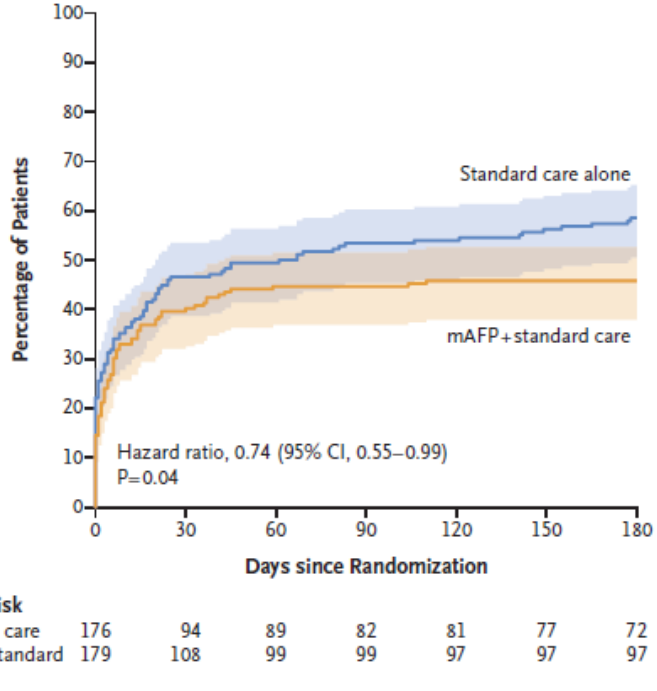
Critère de jugement secondaire : escalade MCS ; mortalité ; HTx

The NEW ENGLAND JOURNAL of MEDICINE

Møller JE et al. *N Engl J Med.* 2024

Impella (IABP)

Étude DanGer Shock (N=360)



No. at Risk

Standard care	176	94	89	82	81	77	72
mAFP+standard care	179	108	99	99	97	97	97

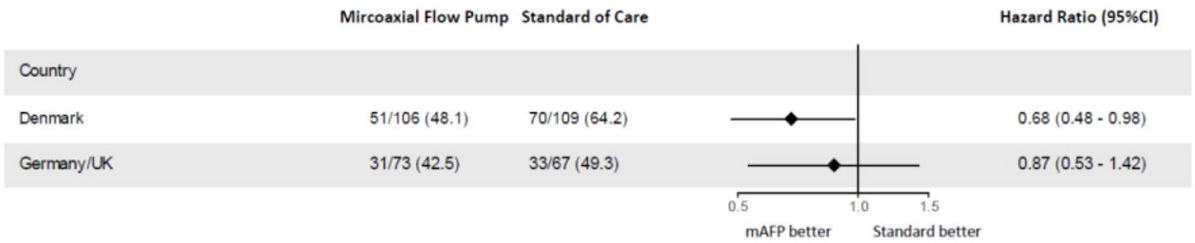
Réduction de la mortalité à 180 jours

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)

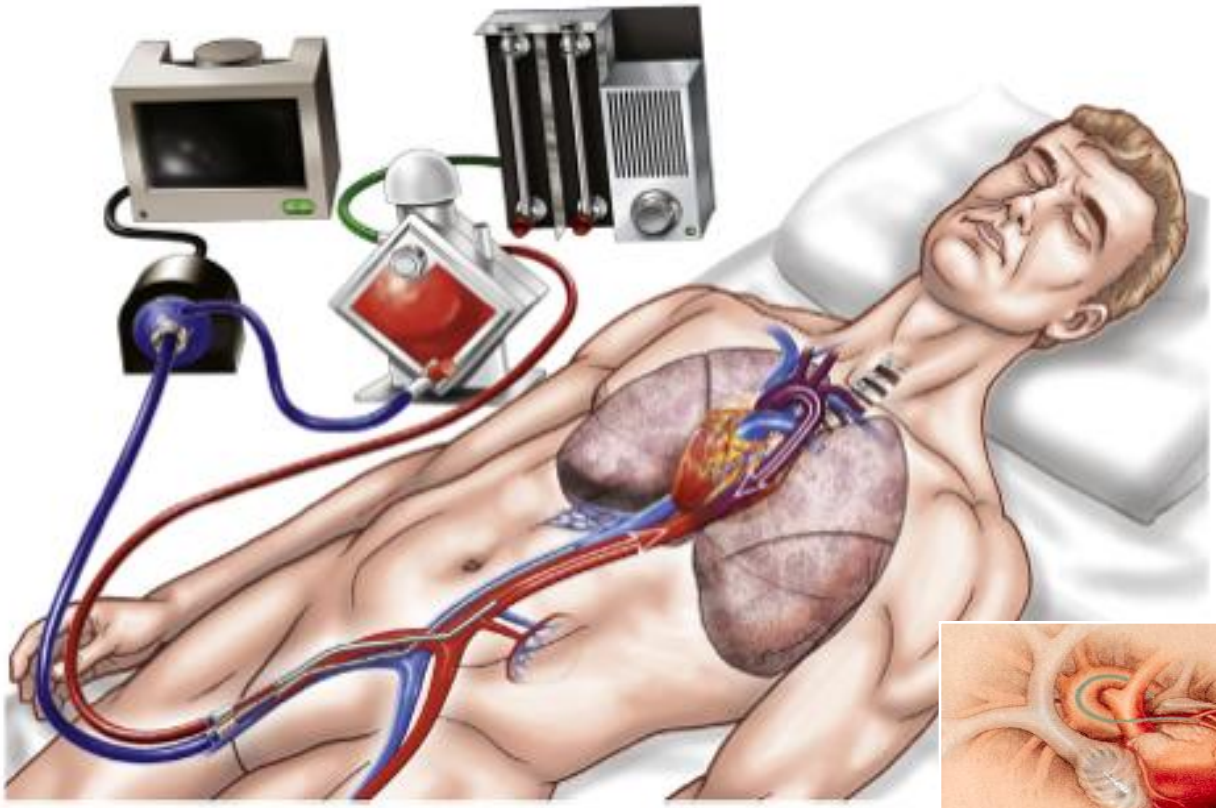
Subgroup Analaysis of Death from Any Cause at 180 Days According to Country of Enrollment

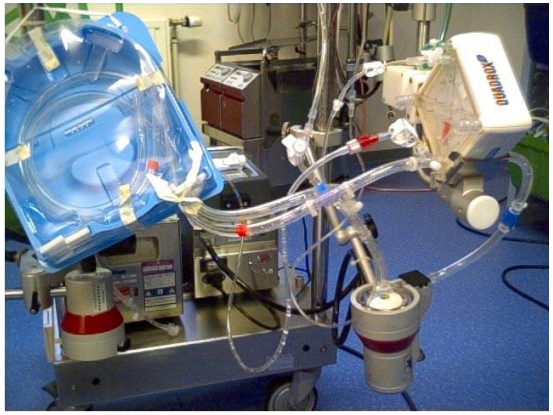


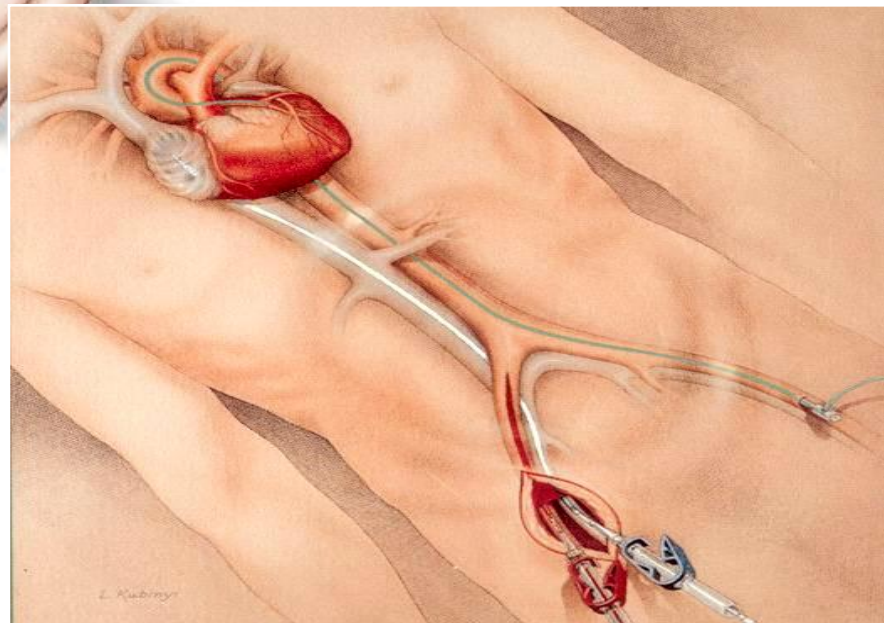
The NEW ENGLAND JOURNAL of MEDICINE

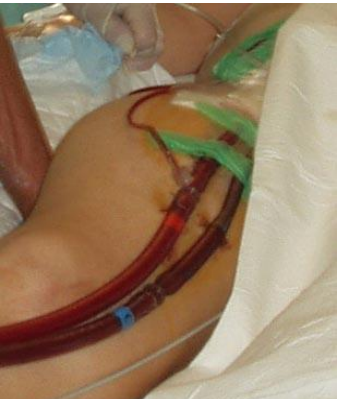
Møller JE et al. *N Engl J Med.* 2024

Extracorporeal Membrane Oxygenation (ECMO, ECLS)



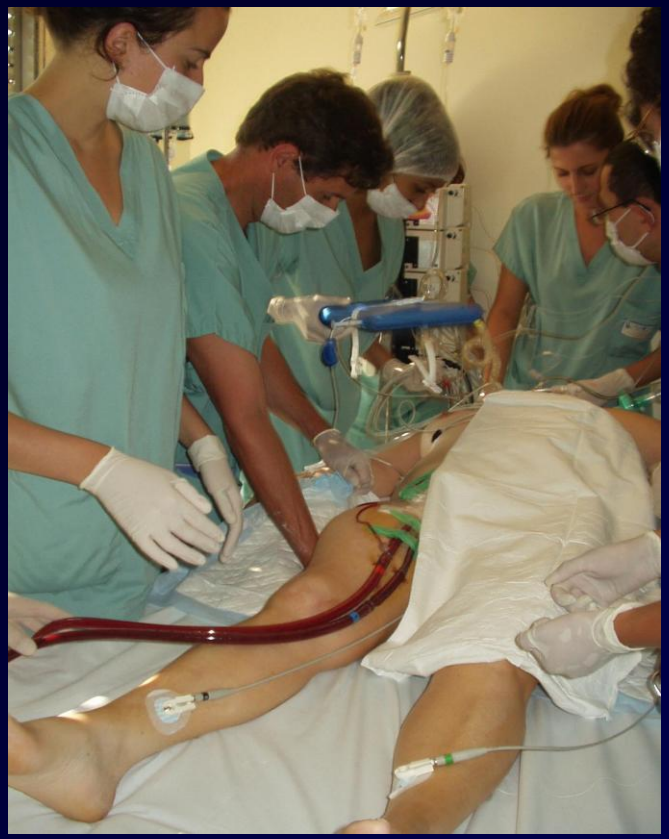






INDICATIONS DE L'ECLS ?

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



Resuscitation (2005) 67, 213–247

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 these[323] 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)

Indication possible

Intoxication
Hypothermie (≤ 32°C)

Signes de vie
per RCP

Mouvements spontanés
Absence de mydriase
Réactivité pupillaire
Gasps inspiratoires

$ETCO_2$ ≥ 10 mmHg
ET Low-flow ≤ 100 min

Lactate/pH?

Incertitude

AC réfractaire

Évaluation de la durée de No-flow

0-5 min

Évaluation du rythme

TV, TdP, FV

Evaluation de la durée de Low-flow

Pas d'indication

Cormorbidités

> 5 min ou sans témoin

Asystole
Rythme agonique

$ETCO_2$ < 10 mmHg
OU Low-flow > 100 min

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

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

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

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, hyperkaliemia, 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. Feistritzer, J. Pöss, 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. Eberl, 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 Scquizzato[1] | Alessandra Bonaccorso[1] | Justyna Swol[2] | Lorenzo Gamberini[3] | Anna Mara Scandroglio[1] | 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)

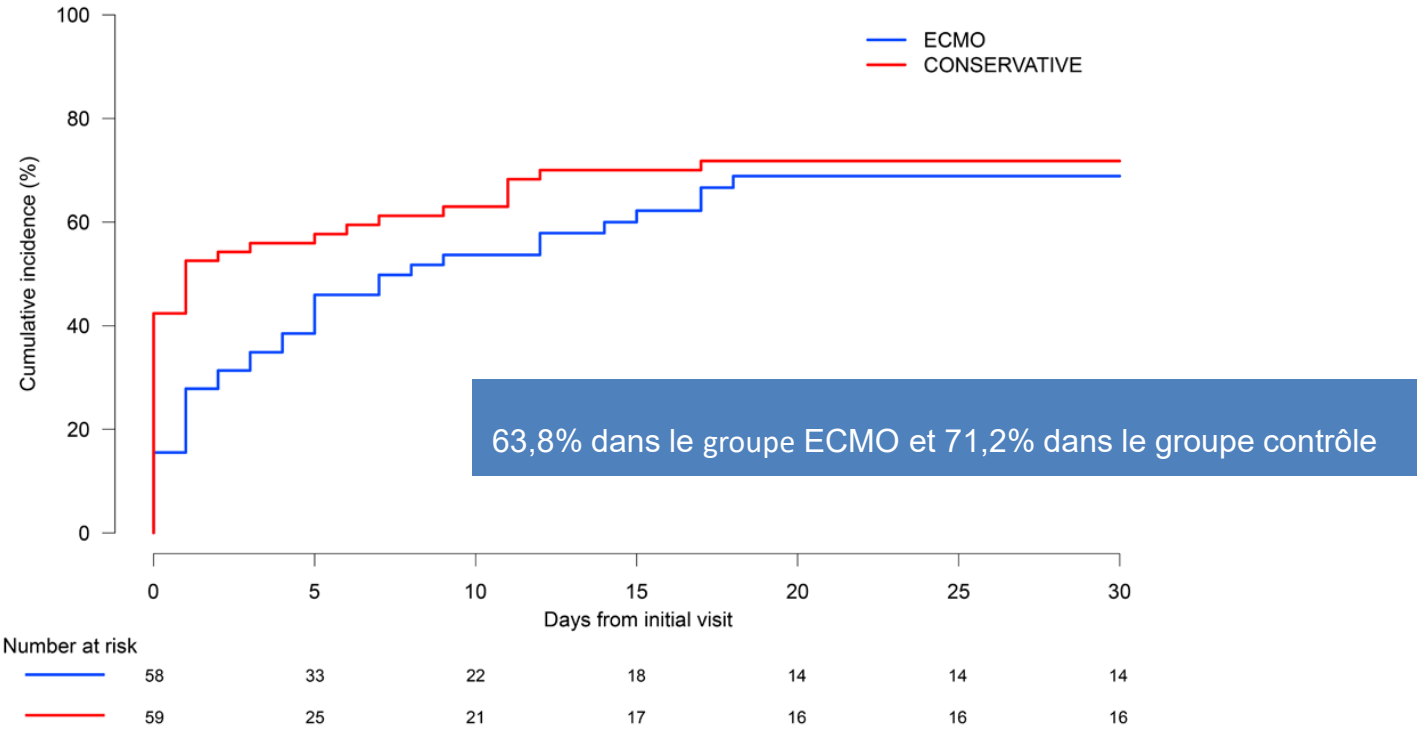
Étude ECMO-CS (N=117)

- ✓ 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

Ostadal P et al. *Circulation* 2023

RCT de l'ECMO VA (SCA / CMD)



63,8% dans le groupe ECMO et 71,2% dans le groupe contrôle

- ✓ 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

Ostadal P et al. *Circulation* 2023

RCT de l'ECMO VA (STEMI + CS)

Étude ECLS-SHOCK (N=420)

Choc cardiogénique sur STEMI

- ✓ PAS < 90 mm Hg ou nécessité de catécholamines
- ✓ Lactate ≥ 3 mmol/L
- ✓ Hypoperfusion d'organes : confusion, marbrures, oligurie

↓

Randomisation

↓

ECMO VA

Prise en charge médicale

↓

ECMO VA si dégradation hémodynamique

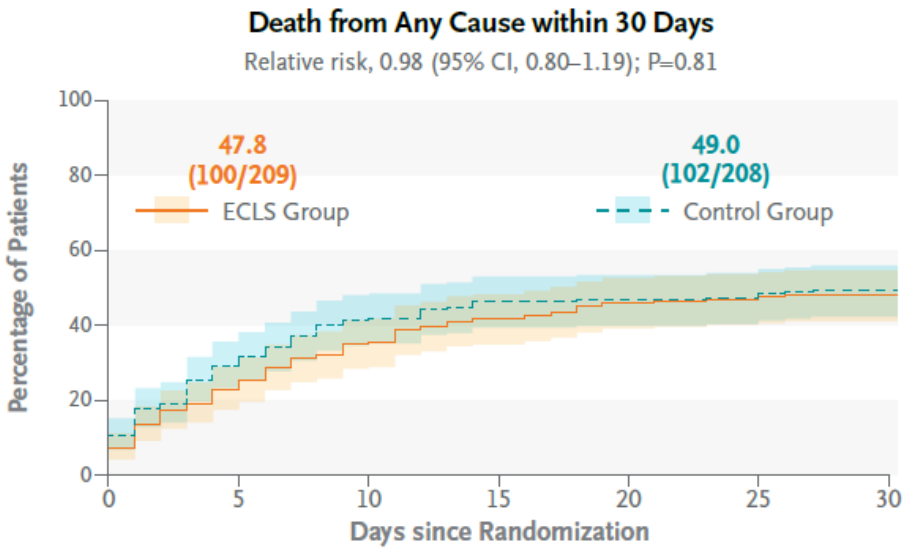
Critère de jugement principal : mortalité à 30 jours

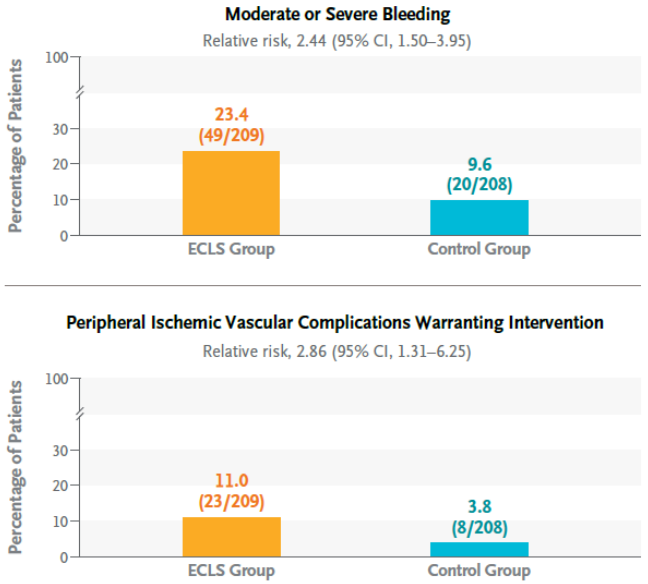
The NEW ENGLAND JOURNAL of MEDICINE

Thiele H et al. *N Engl J Med.* 2023

Place de l'ECMO VA (STEMI + CS)

Étude ECLS-SHOCK (N=420)





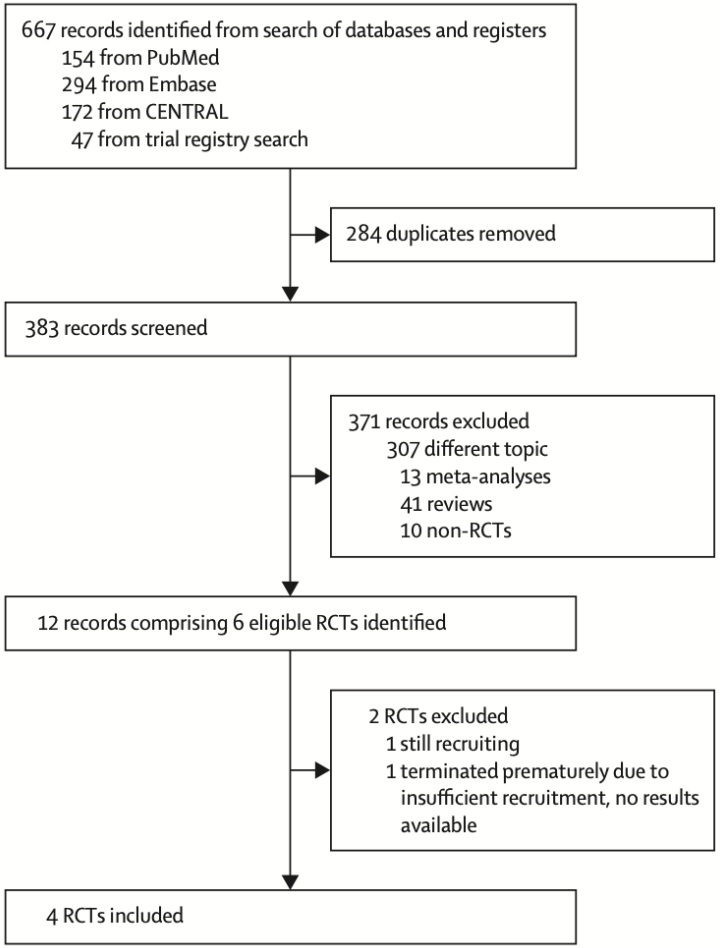
- ✓ N = 417 patients inclus entre 2019 et 2022 (44 centres)
- ✓ 77% des patients ont présenté un arrêt cardiaque récupéré (RCP < 45 minutes)
- ✓ Groupe contrôle : 26 patients implantés d'une ECMO (12,5%) et 28 d'une autre assistance
- ✓ Durée d'ECMO 2,7 jours

The NEW ENGLAND JOURNAL of MEDICINE

Thiele H et al. *N Engl J Med.* 2023

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)



Zeymer U et al. *Lancet*. 2023

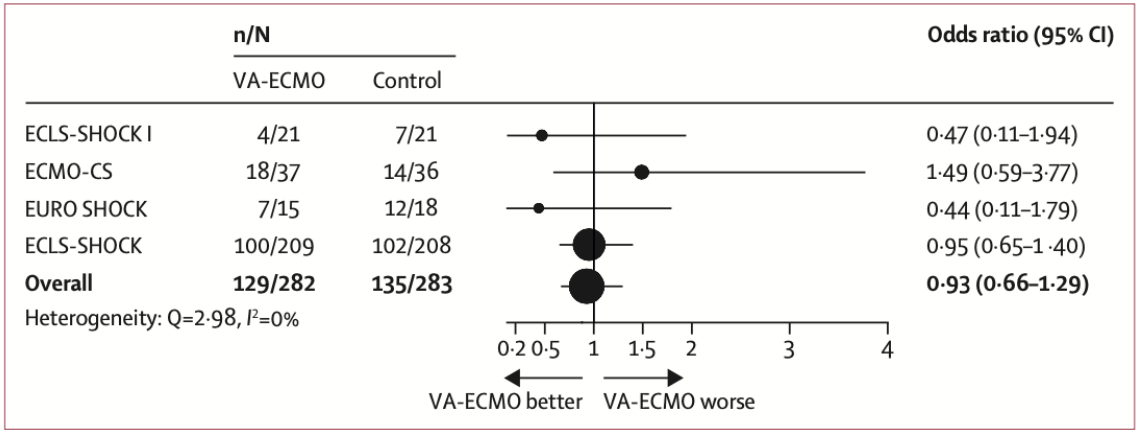
ECMO VA & choc cardiogénique ischémique : review

	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)**



Zeymer U et al. *Lancet*. 2023

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

Zeymer U et al. *Lancet*. 2023

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 ≤ 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

American Heart Association.

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 ?

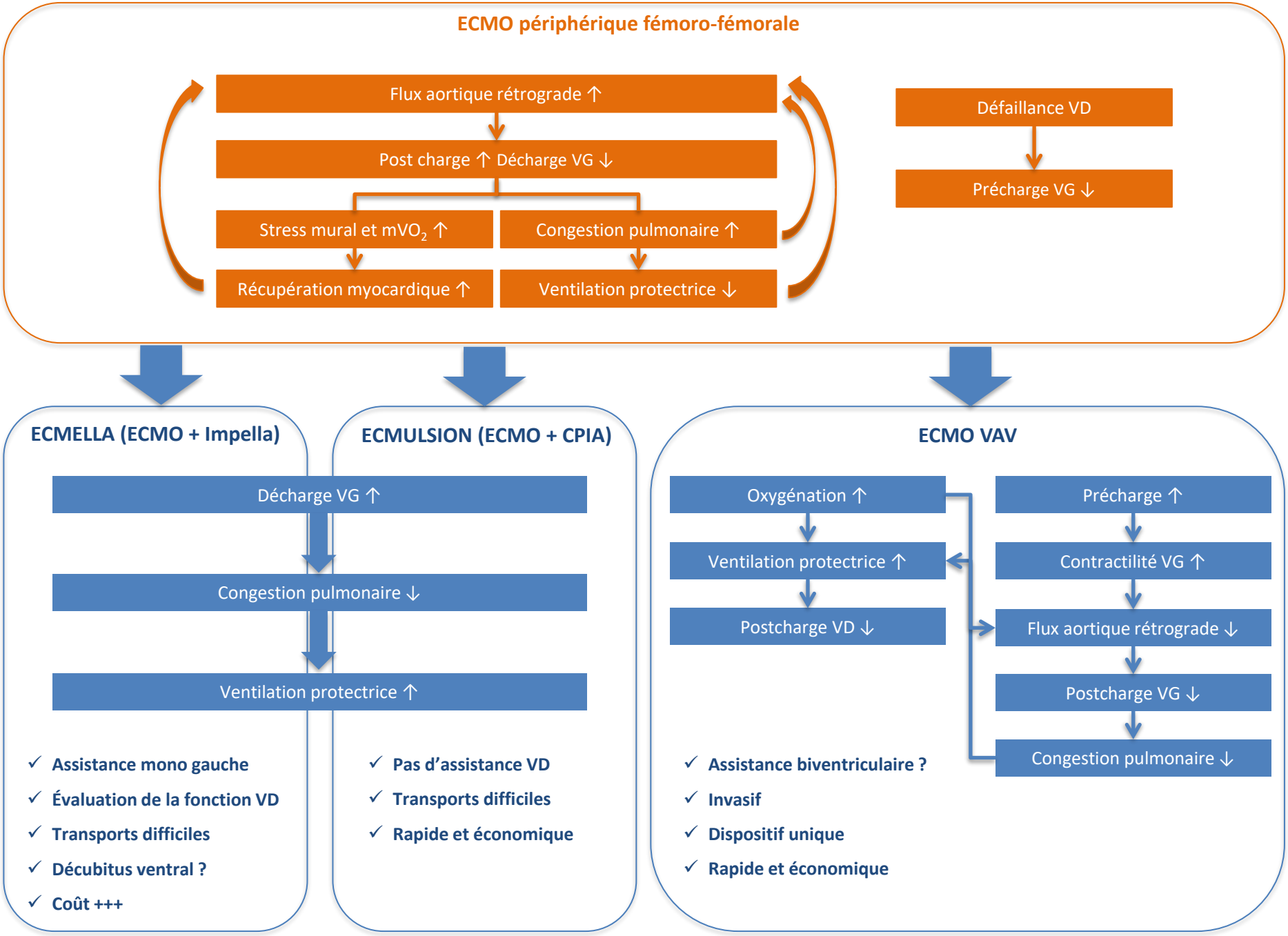
	Survival				
	Hospital (%)		5-Year (%)	HR (95% CI)	5-Year Conditional to Hospital Discharge (%)
PGF	>50%	73.3	57.3	0.30 (0.22-0.42)*	78.2
DCM		53.2	45.3	0.60 (0.45-0.80)*	85.2
Drug overdose		58.6	54.0	0.63 (0.32-1.24)	92.2
Arrhythmic storm		51.6	50.0	0.73 (0.40-1.31)	96.9
Massive PE	30-50%	46.8	38.3	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	<30%	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		25.7	22.8	1.64 (1.25-2.14)*	88.7

Da ial et al, J Am Coll Cardiol 2023

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

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

5-year survival is generaly stable after hospital survival


ECMO périphérique fémoro-fémorale
Flux aortique rétrograde ↑
Post charge ↑ Décharge VG ↓
Stress mural et mVO2 ↑
Congestion pulmonaire ↑
Récupération myocardique ↑
Ventilation protectrice ↓
Défaillance VD
Précharge VG ↓
ECMELLA (ECMO + Impella)
Décharge VG ↑
Congestion pulmonaire ↓
Ventilation protectrice ↑
✓ 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
Oxygénation ↑
Ventilation protectrice ↑
Postcharge VD ↓
Précharge ↑
Contractilité VG ↑
Flux aortique rétrograde ↓
Postcharge VG ↓
Congestion pulmonaire ↓
✓ 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

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]

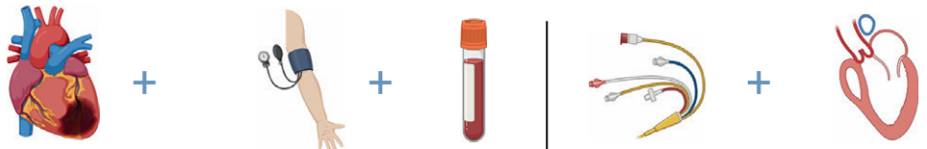
FIGURE 1 Guideline Changes for Use of MCS in MI With CS

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			

DanGer Shock

Inclusion Criteria



after revascularization) need

Exclusion Criteria

GCS <8 after ROSC | precluding mAFP | expectancy <1 year | complications of AMI | Severe aortic abnormalities, LV thrombus

VA-ECMO: NO BENEFIT TO EARLY USE

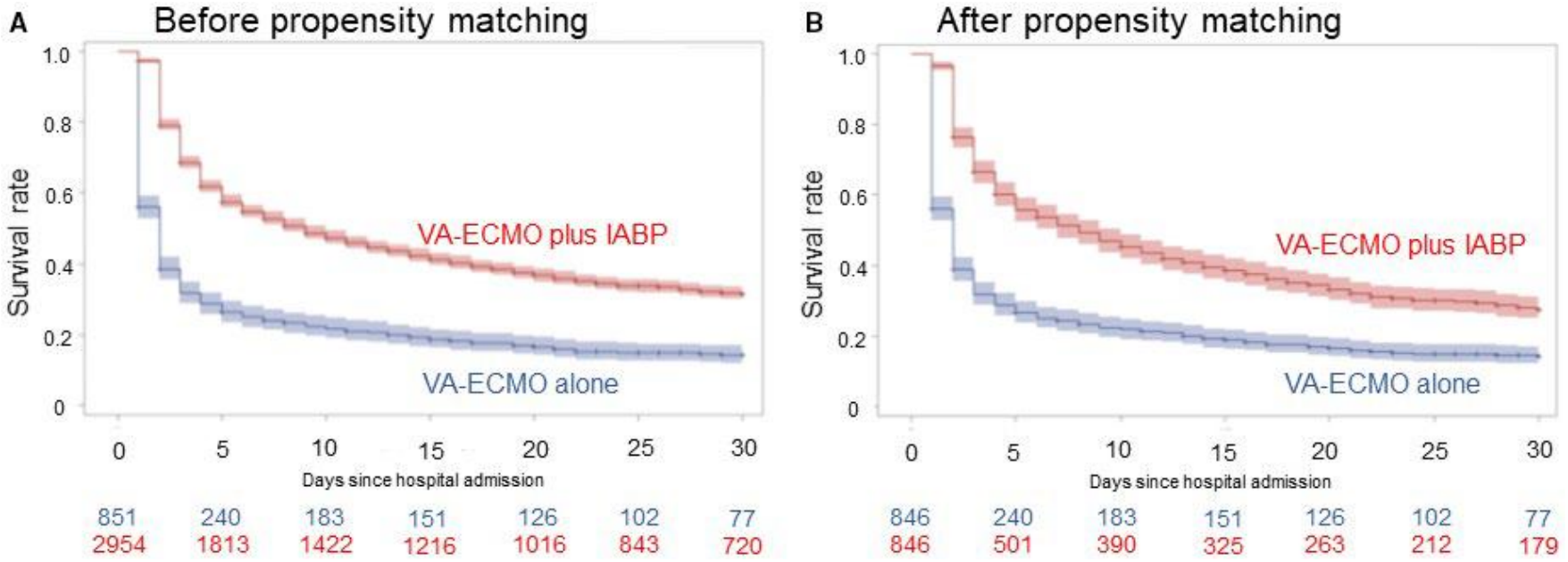
mAFP: UPGRADE IN SELECTED PATIENTS WITH STEMI

ACC/AHA guide

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.

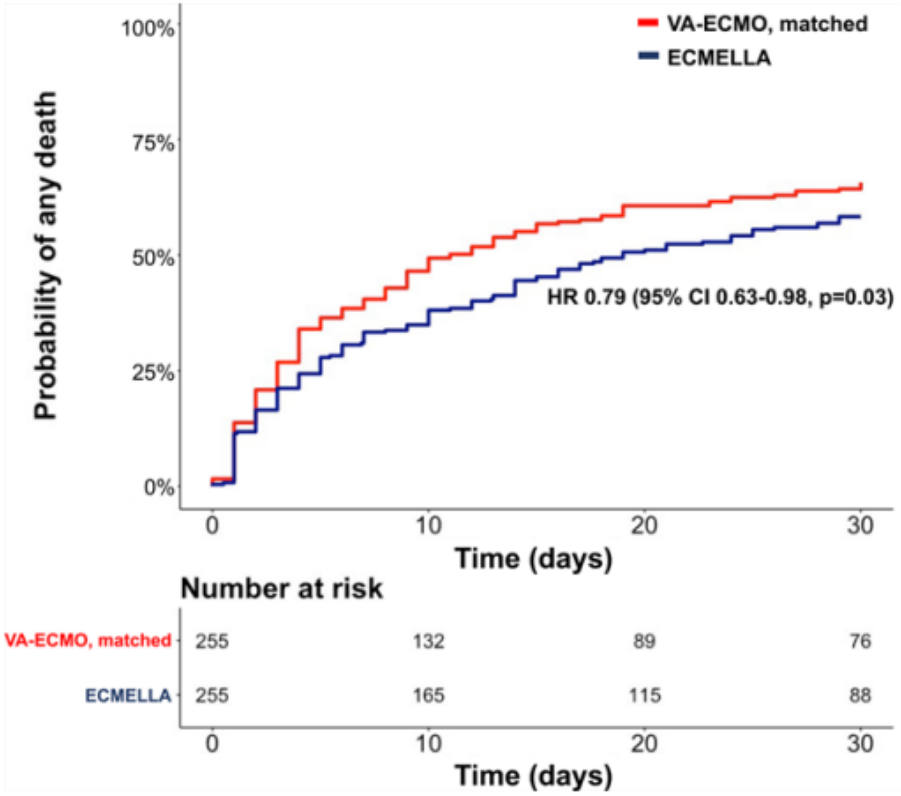
ECMO + ballon de contre-pulsion intra aortique



- ✓ 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

Nishi T et al. *J Am Heart Assoc*. 2022 Apr 5;11(7):e023713

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

Schrage B et al. *Circulation* 2020 Dec;142(22):2095-2106

Choc cardiogénique et régulation : centres CC

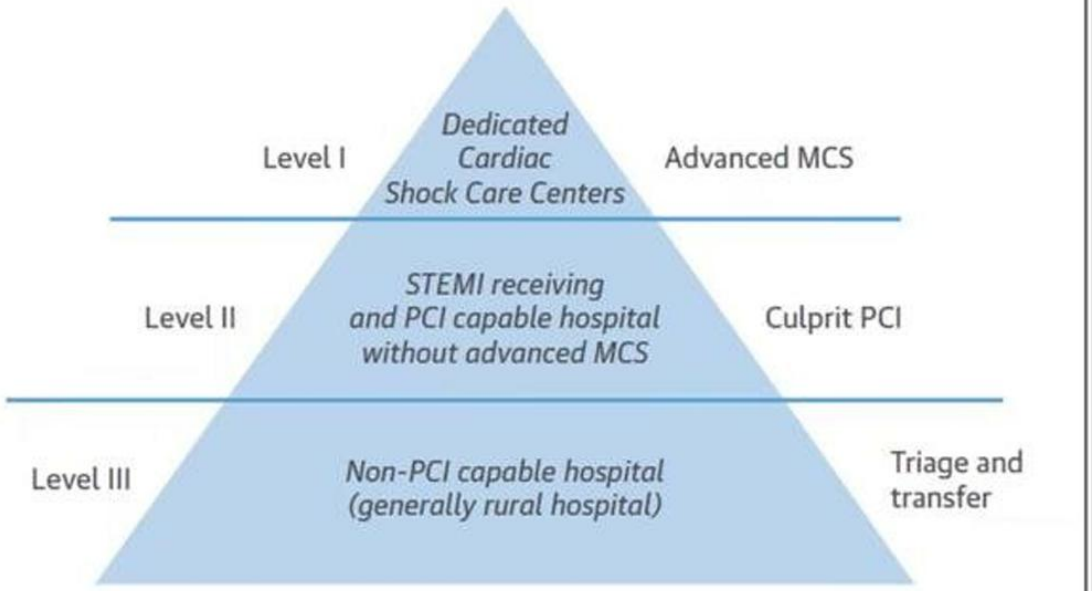
Cardiac Shock Care Centers

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]

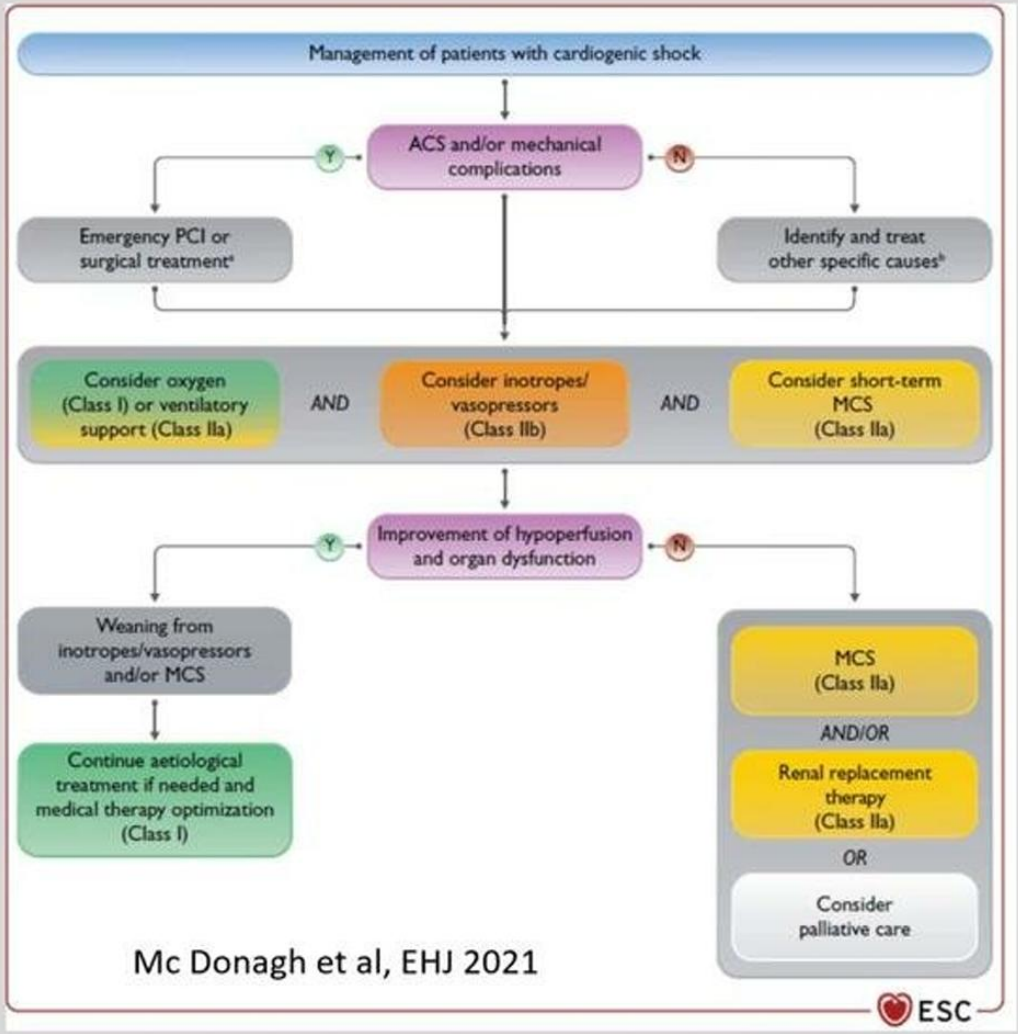
FIGURE 2 Levels of Cardiac Shock Care



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

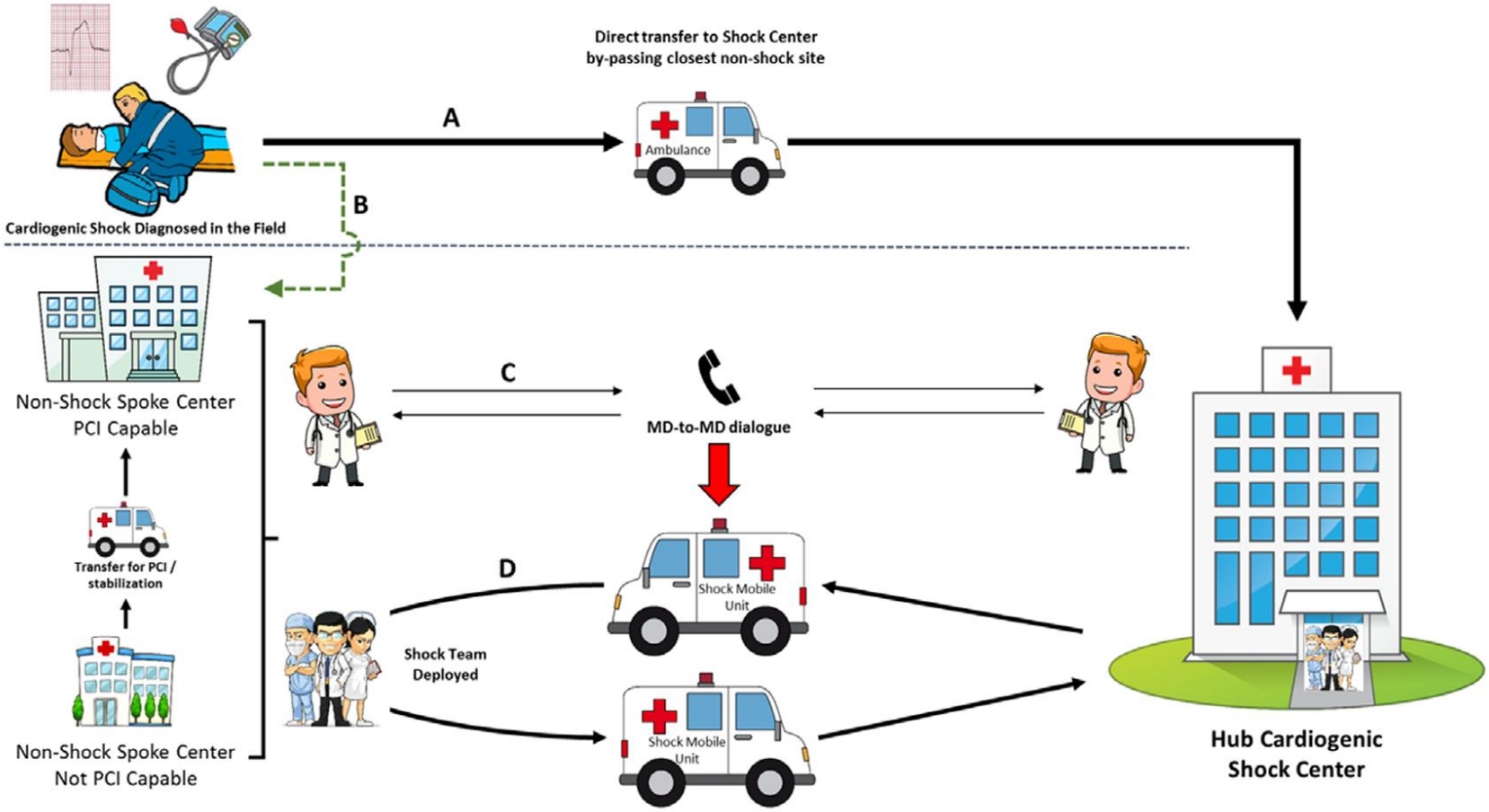
Available 24h/24 and 7days/7

Choc cardiogénique et régulation : protocole



CS management

- **Need a rapid and multiparametric diagnosis (clinic +++)**
- **Management:**
  - Early etiological management
  - Fluid management (diuretics vs fluid challenge)
  - Inotrops (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.

Van Diepen. *Circulation.* 2017

Chioncel.
EHJ Heart Failure
2020

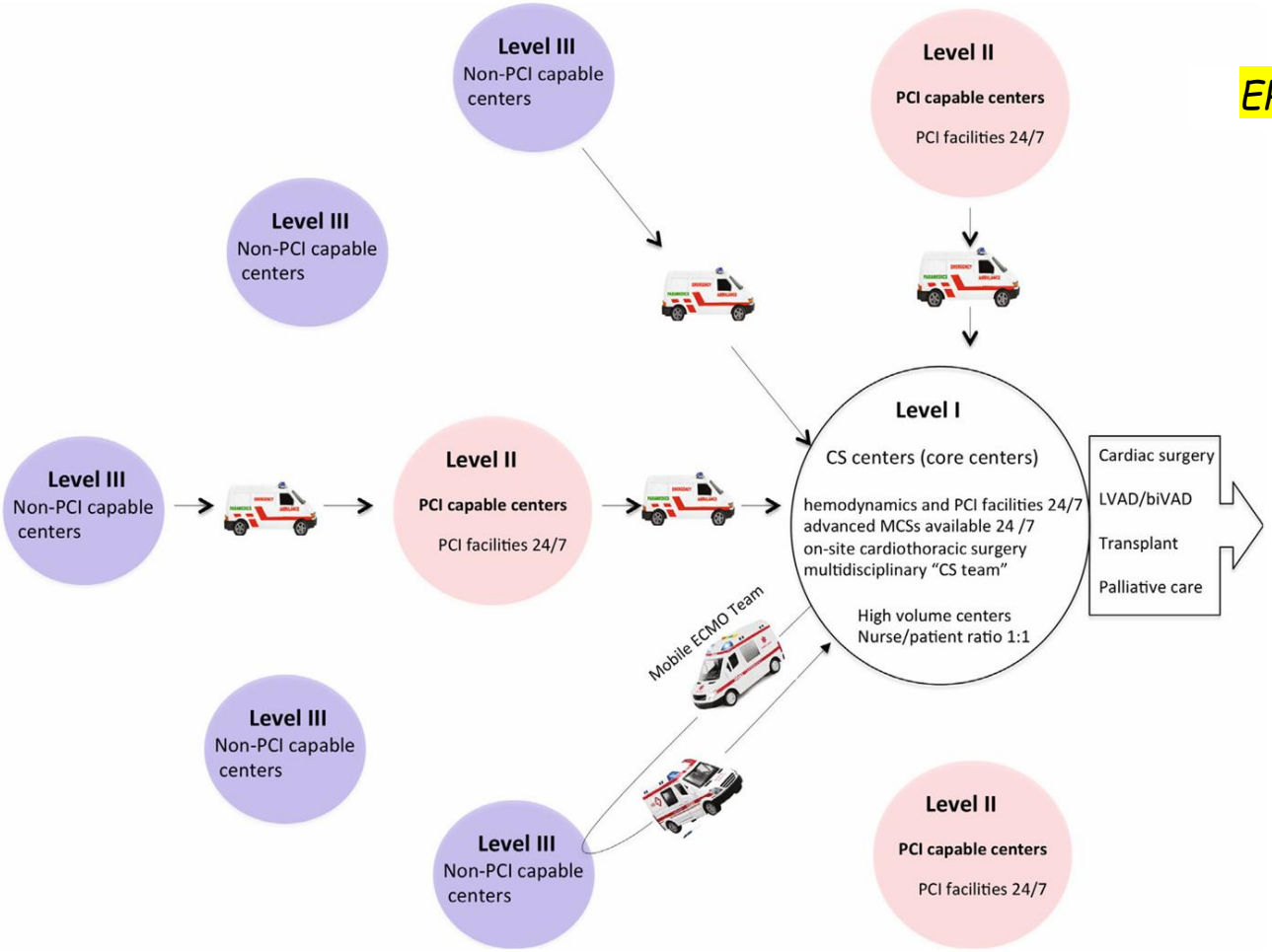
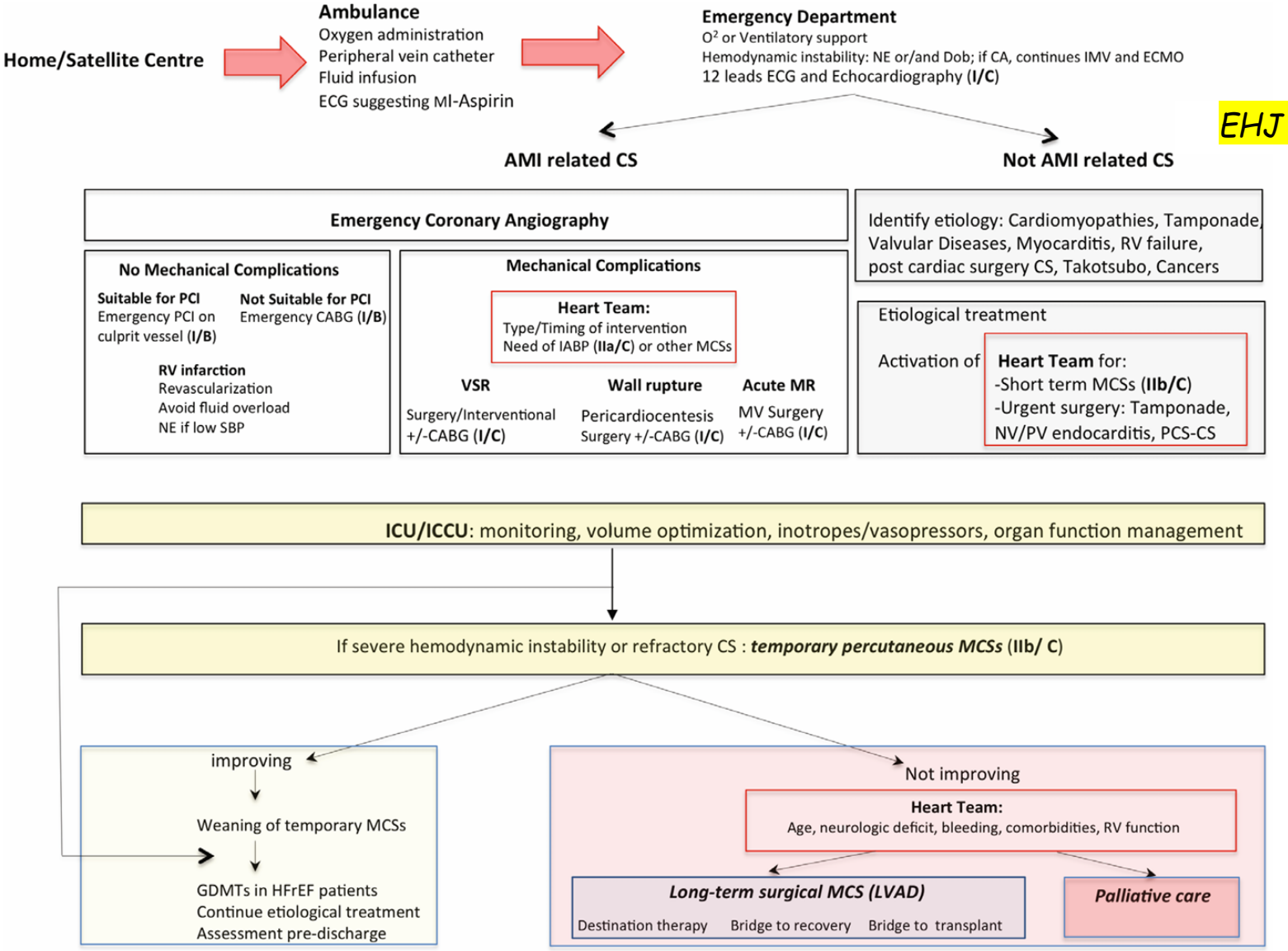


Figure 4 The systems of care for patients with cardiogenic shock (CS). A model for minimizing time delays and optimizing care has recently been proposed by the American Heart Association, where a network between several satellite centres and a central 'CS centre' exists to facilitate optimal care coordination. 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 seconnd 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.



Chioncel.
EHJ Heart Failure
2020

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; ICCU, intensive cardiac care unit; ICU, 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