DES Anesthésie-Réanimation/DESC Réanimation médicale Ile de France

Choc cardiogénique de l'infarctus du myocarde

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Historique

- 1942, Stead and Ebert 1ère description des manifestations d'un choc résultat d'une dysfonction cardiaque avec des effets systemique multiples:

"characterized clinically by the signs of a marked decrease in cardiac output and tissue anoxia. . .,"

Stead EA, Ebert RV. Shock syndrome produced by failure of the heart. Arch Intern Med 1942;69:369–83.

Définition

Baisse du débit cardiaque avec hypoxie tissulaire en présence d'une volémie adaptée.

- **PA < 80 mmHg depuis au moins 30 mn**
- **Hypoperfusion périphérique**
- **Index cardiaque < 2,2 L/mn/m^2**
- **Pression capillaire pulmonaire > 18 mmHg**

Braunwald, Heart Disease 5th,Saunders

Définition

- **PAS < 90 mm Hg depuis ≥1 h**
- **Échec remplissage seul**
- **Secondaire à une dysfonction cardiaque ou**
- **Associée à des signes d'hypoperfusion périphérique ou IC<2.2L/min/m^2 et Pcap >18 mm Hg**
- **Ou PAS > 90 mm Hg sous inotrope**

Hasdai, Lancet 2000; 356:749-56

International Consensus Conference, Paris, France, April 2006.

Cardiogenic shock is defined as

- systemic tissue hypoperfusion
- secondary to inadequate cardiac output
- Despite adequate circulatory volume and LV filling pressure.
- Diagnostic hemodynamic criteria
  - Systolic blood pressure <90 mm Hg for >30 min;
  - Drop in mean arterial blood pressure >30 mm Hg below baseline,
  - Cardiac index (CI) <1.8 L/min/m2 without hemodynamic support or <2.2 L/min/m2 with support
- pulmonary capillary wedge pressure (PCWP) >15 mm Hg

Antonelli M,, et al. Intensive Care Med. 2007

IABP SHOCK

• PAS < 90 mm Hg pendant plus de 30 minutes

• ou catécholamines pour maintenir une PAS > 90 mmHg

• Des signes cliniques de surcharge pulmonaires et d'hypoperfusion tissulaire avec au moins l'un des critères :

-Confusion

-Peau et extrémités froides et moites

-Oligurie avec diurèse < 30 mL/h

-Lactates sériques > 2,0 mmol /L

Thiele H, Am H J 2012

Tableau clinique

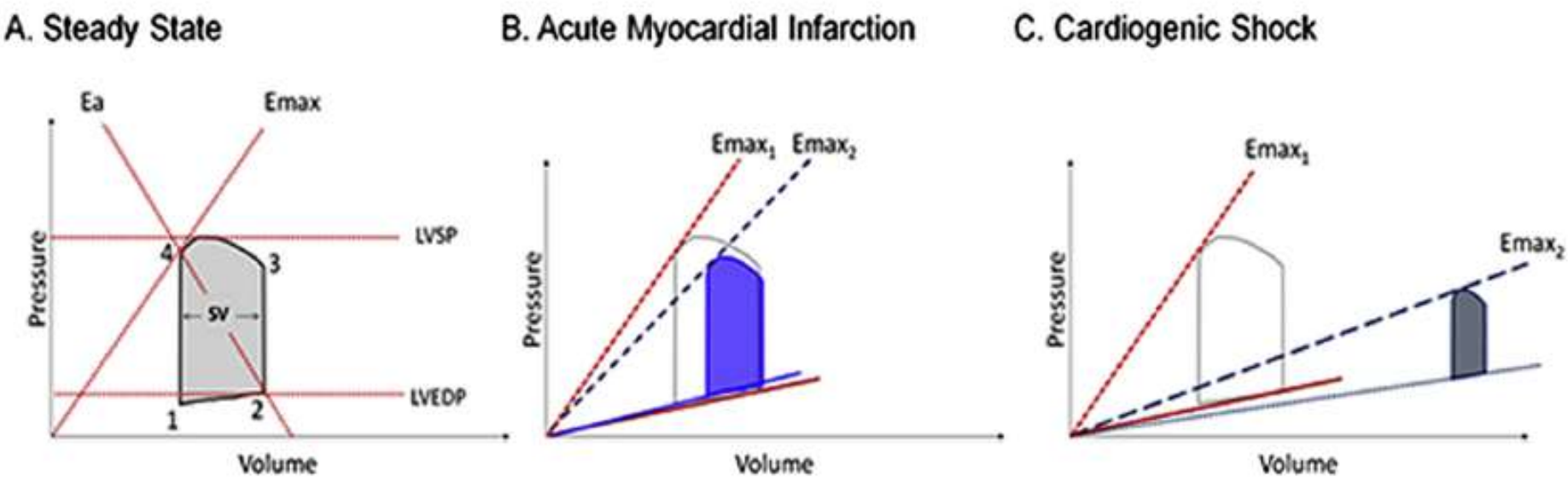
- **Apparition au cours d'un infarctus**
  - ***Soit d'emblée:*** **diagnostic facile, pronostic catastrophique**
  - ***Soit secondaire:*** **le plus fréquent. Le diagnostic est difficile car PA normale souvent pdt 48 heures. Savoir prédire l'apparition d'un choc cardiogénique.**

- **Cas particulier de l'arrêt cardiaque**
  - Choc dans 50% des cas, 6-8 heures après arrêt, mécanisme mixte (cardiaque et vasoplégique)

Dysfonction ventriculaire

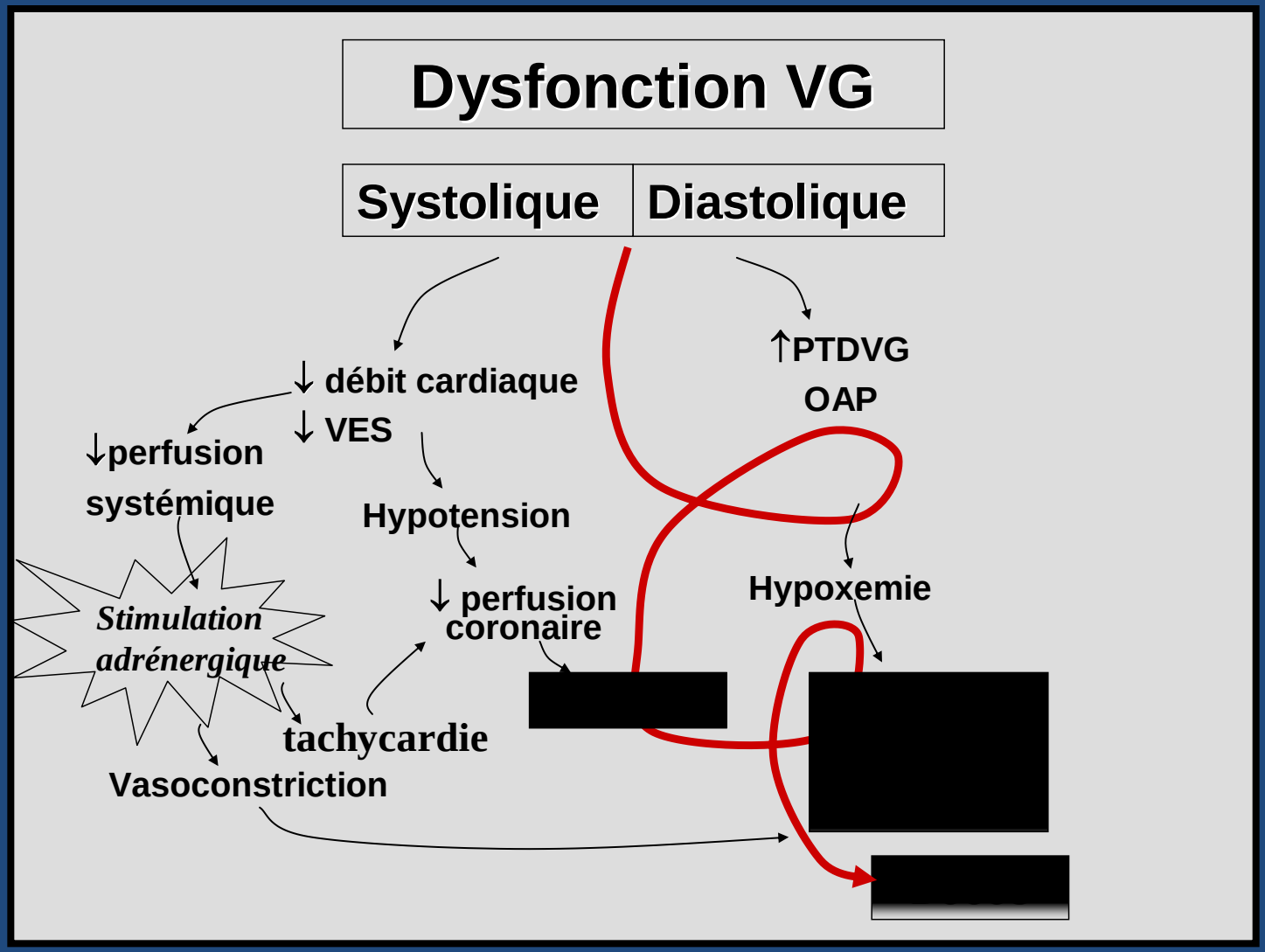
- élévation précharge (incompétence valvulaire, ↑FC)
- diminution contractilité myocardique,
- élévation postcharge,
- asynergie de la contraction
- ± réduction de la fréquence cardiaque (tr conductifs)

FIGURE 1 Normal and Abnormal Pressure Volume Loops



2015 SCAI/ACC/HFSA/STS Clinical Expert Consensus Statement on the Use of Percutaneous Mechanical Circulatory Support Devices in Cardiovascular Care

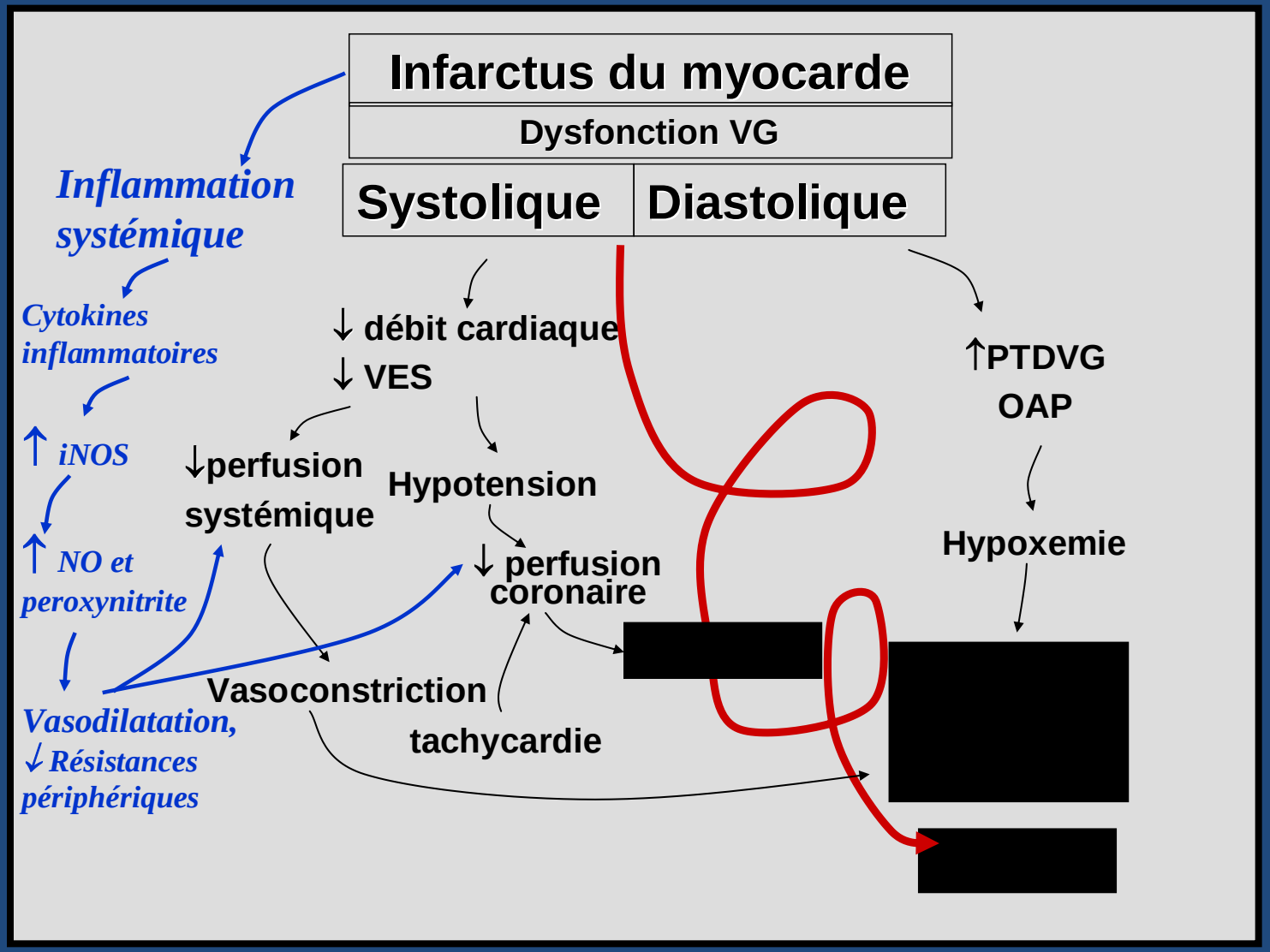
Physiopathologie : effets systémiques.



Remise en cause du « dogme »

- FEVG non effondrée: 30%,
- résistances systémiques non élevées en moyenne,
- choc à PA conservée
- CARDIAC POWER *(Cotter et al)*: IC x PAM
- sd inflammatoire systémique:
  - Cytokines
  - iNOs

Physiopathologie, schéma modifié



D'après SHOCK

Etiologie IDM + choc

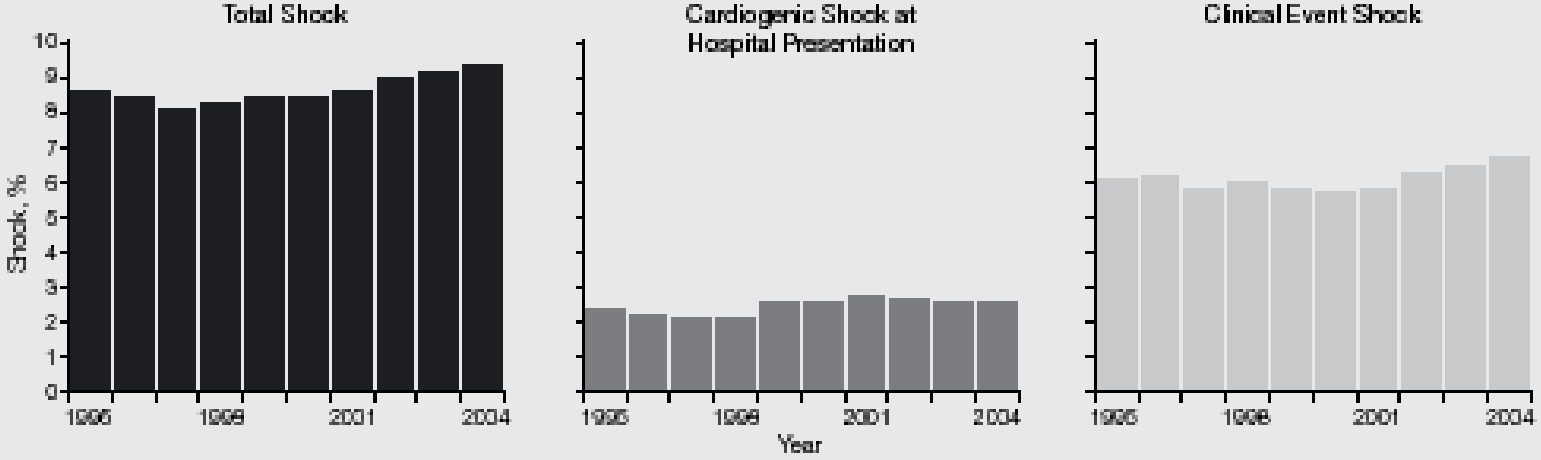
- 40% ATCD d'IDM: réserve myocardique diminuée **40%**
- IDM étendu > 40% du VG *(Killip T. JACC 1989)* **25%**
- IDM étendu au VD **20%**
- IM aiguë (rares actuellement) **6.9%**
- CIV **3.9%**
- IDM limité au VD **2.8%**
- Rupture myocardique **1.4%**

Trends in Management and Outcomes of Patients With Acute Myocardial Infarction Complicated by Cardiogenic Shock.

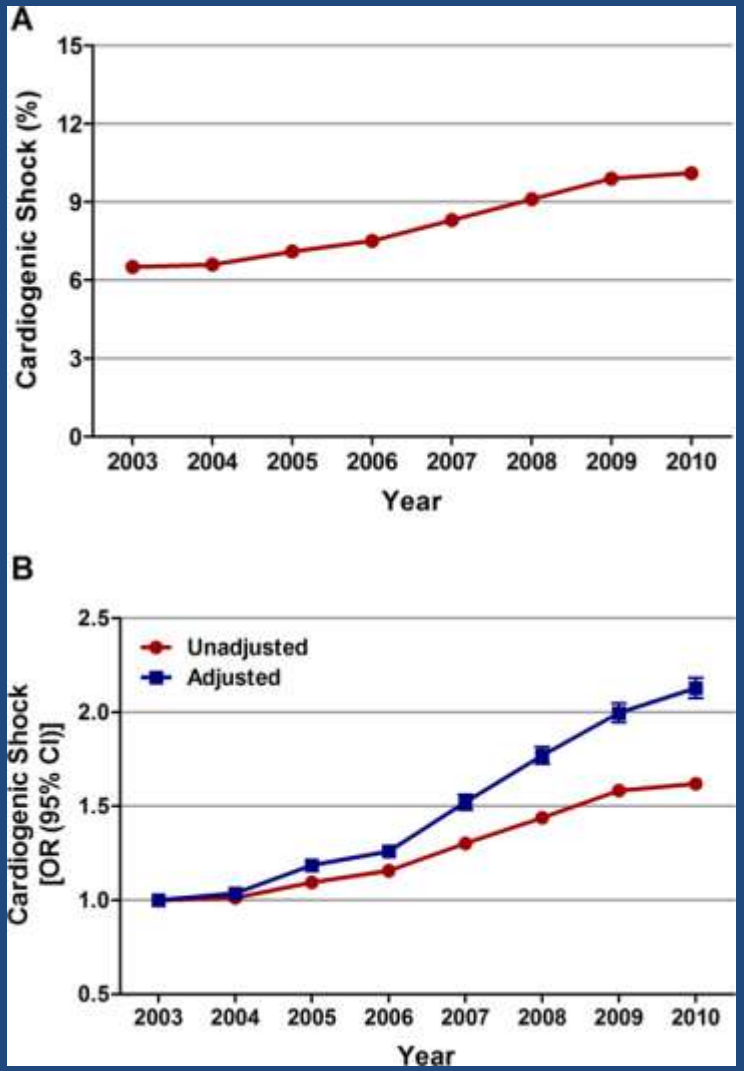
Anvar Babaev et al. JAMA, 2005

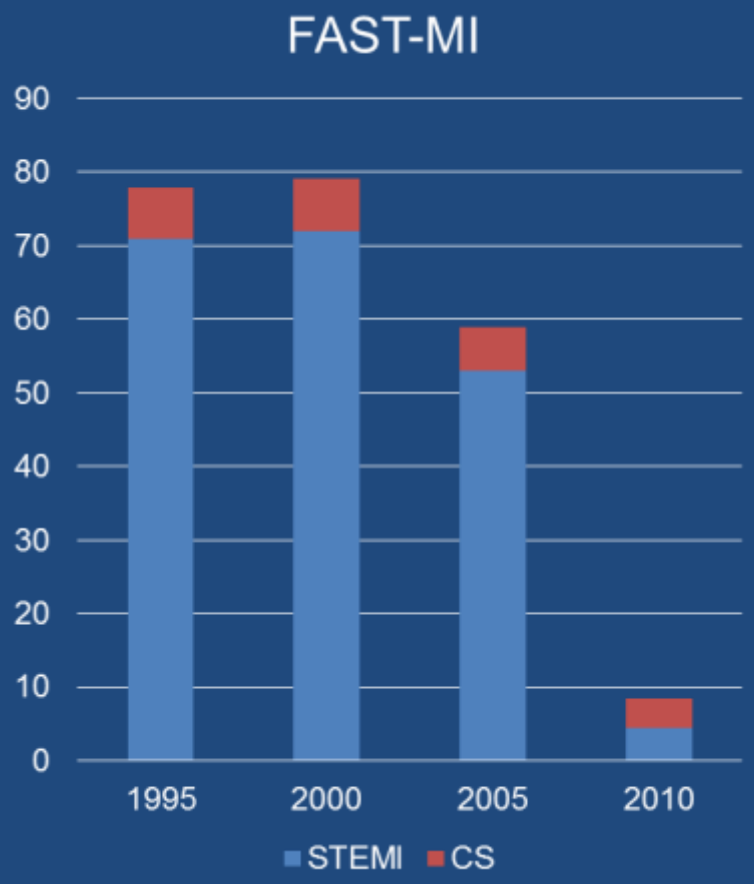
- ***8.6% des IDM***
- ***29% des cas le choc est présent à l'admission, principalement si >75 ans***
- ***71% développe le choc durant l'hospitalisation le plus souvent entre 24 et 48 heures***

Figure 1. Frequency of Cardiogenic Shock Among Patients in the NRMI Registry



Incidence





Kolte JAHA 2014, Aissaoui, ESC 2014

Facteurs prédictifs de la survenue d'un choc

- **Âge,**
- **fréquence cardiaque >75,**
- **Facteurs de risque: diabète,**
- **ATCD d'IDM, de PAC**
- **Classe killip à l'admission,**
- **Localisation IDM antérieur**

Hasdai, JACC 2000

Pronostic

- **Mortalité hospitalière: > 70 % stable entre 1975 et 1990**
- **59 % en 1997, alors que patients plus vieux et plus malades**
- **Plus élevée si absence sus-décalage ST**
  - Plus âgés
  - Lésions plus diffuses
  - Lésions TC plus fréquentes
  - Moins de revascularisation

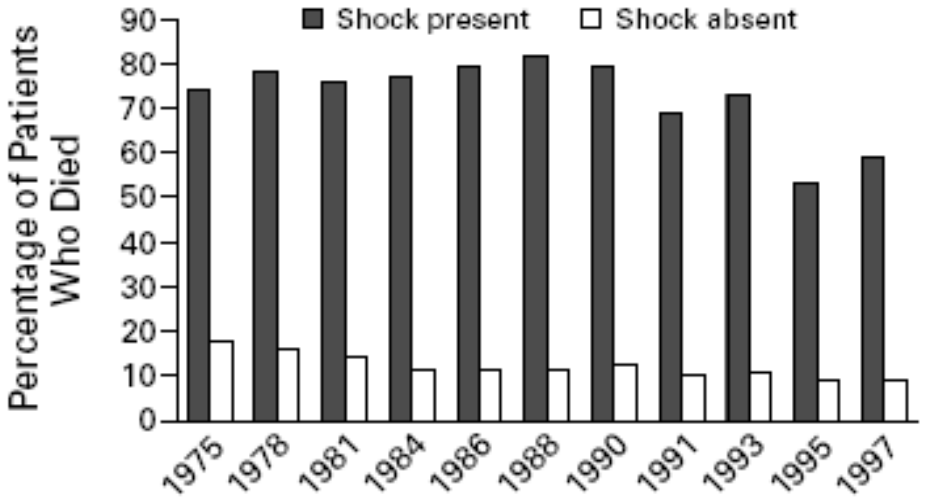
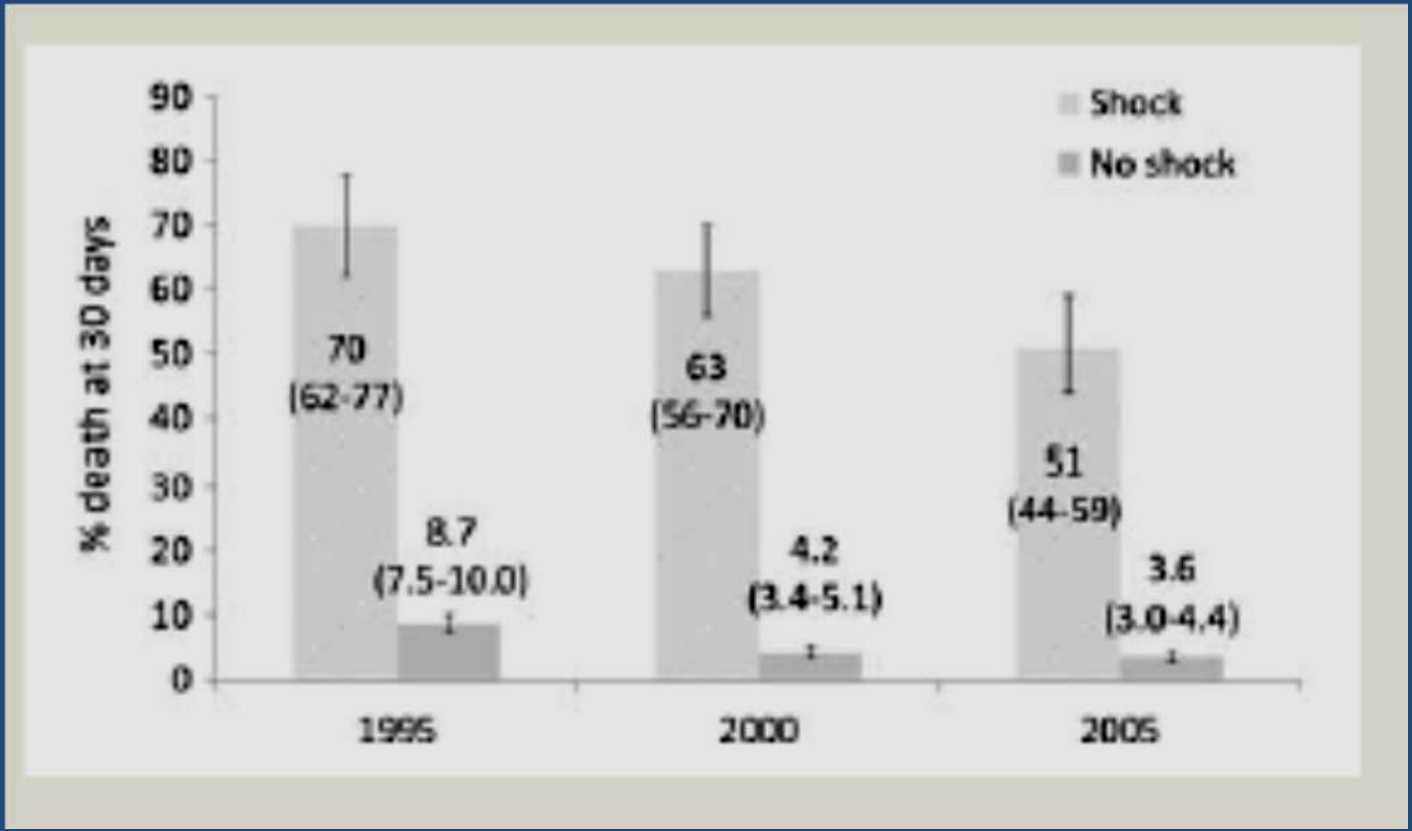


Figure 2. Temporal Trends in In-Hospital Death Rates among Patients with Acute Myocardial Infarction According to the Presence or Absence of Cardiogenic Shock.

Goldberg, NEJM 1999

- **Mortalité ~ 50%** malgré : reperfusion en urgence, ttt medical optimal et IABP

FAST MI



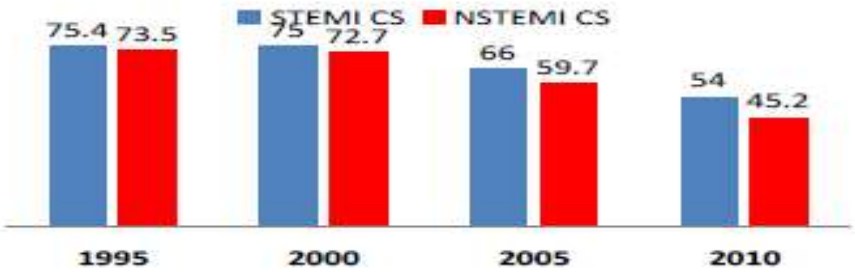
Aissaoui N. Eur Heart J 2012

Evolution of incidence and one-year outcomes of cardiogenic shock in acute myocardial infarction from 1995 to 2010. The FAST-MI programme

- **One-year mortality :**

✓ was considerably higher in CS patients (67% vs 10%);

✓ similar patterns for STEMI and NSTEMI, and across age groups.



- **In CS patients (Cox multivariate analysis)**

✓ Age, diabetes, higher BMI and STEMI, were independent correlates of increased one-year death;

✓ Time period was associated with reduced mortality (HR 2010 vs 1995= 0.57; 95% CI: 0.40-0.83, P=0.003), along with early use of PCI and medications at the acute stage.

Aissaoui, ESC 2014

Pronostic

- IABP SHOCK:
  - index cardiaque seul + biomarqueur de dysfonction pas corrélés à la survie précoce.
- score de sévérité d'atteinte multiorgane : APACHE II et SAPS II)
- biomarqueurs syndrome de réponse inflammatoire systémique (Il-6, récepteur des produits de glycation avancé RAGE)

→ prédisent la mortalité de manière plus fiable que les paramètres hémodynamiques

Facteurs risque mortalité

Etudes TRIUMPH SHOCK et registre SHOCK

- âge,
- lésions cérébrales anoxiques,
- atteintes d’hypoperfusion d’organe,
- volume d’éjection systolique,
- fraction d’éjection du ventricule gauche,
- pression artérielle systolique,
- support vasopresseur
- clairance de la créatinine

Amélioration pronostic

REVASCULARISATION

- **Seul paramètre amélioration survie Réduction:**
  - **Choc admission**
  - **Choc retardé**
- **Amélioration de la survie à 6mois, 6ans**

(SHOCK, ISIS-2, GISSI, GUSTO-I)

Traitements

« mécaniques »

- **Revascularisation**
- **Assistances circulatoires**

« médical »

EARLY REVASCULARIZATION IN ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK

Judith S. Hochman et al. NEJM, 1999.

IDM (↑ ST ou BBG) + choc < 36 h

Revascularisation vs Stabilisation médicale

(ATC ou PAC) (CPBIA/fibrinolyse)

152 150

54.6%ATC (35.7% avec stent)

37.5%PAC

Objectif I: mortalité à 30j

Objectif II: mortalité à 180j

EARLY REVASCULARIZATION IN ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK

Outcome and Subgroup	Revascularization	Medical Therapy
	percent (number in subgroup)	
30-day mortality		
Total	46.7 (152)	56.0 (150)
Age <75 yr	41.4 (128)	56.8 (118)
Age ≥75 yr	75.0 (24)	53.1 (32)*
6-mo mortality‡		
Total	50.3 (151)	63.1 (149)*
Age <75 yr	44.9 (127)	65.0 (117)*
Age ≥75 yr	79.2 (24)	56.3 (32)*

Judith S. Hochman et al. NEJM, 1999.

EARLY REVASCULARIZATION IN ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK

Judith S. Hochman et al. NEJM, 1999.

- Succès ATC Mortalité J30: 38%vs 79% échec ATC

  13 vies sauvées pour 100 patients traités
- Succès :
  - Sténose résiduelle < 50/%.
  - Diminution d'au moins 20% du degré de sténose
  - Flux Timi II ou III.
- Taux de succès élevé (opérateur dépendant)
- Utilisation des stents intracoronaires
- Utilisation des anti GPIIb/IIIa.
- Précocité de la revascularisation (0.9h)

Emergency revascularization in patients with cardiogenic shock on admission: a report from the SHOCK trial and registry

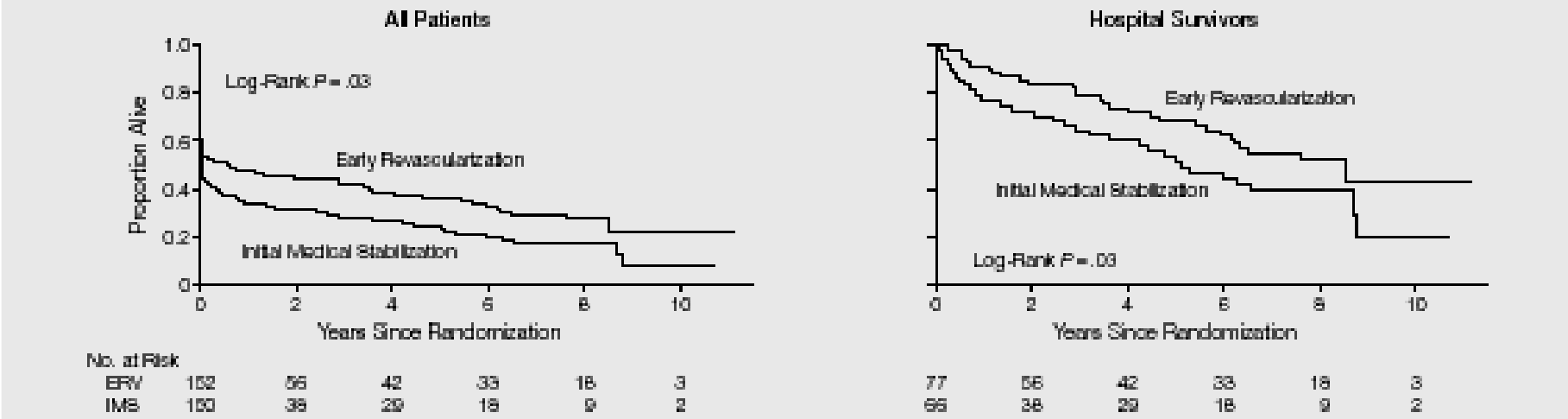
Jeger R et al, European Heart Journal, 2006

- ↑Mortalité hospitalière: 75 vs. 56% (p=0,001)
- Décès plus rapide : mortalité à 24h: 40 vs.17% (p=0,001)
- **La revascularisation en urgence** réduit la mortalité hospitalière tant des chocs à l'admission : 60 vs. 82% (p< 0.001) que des chocs retardés : 46 vs. 62% (p< 0.001) (interaction P = 0.25)
- Choc à l'admission: facteur prédictif indépendant de mortalité hospitalière OR: 1.68 (P = 0.008)

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

Judith S. Hochman et al. JAMA, 2006.

Figure 2. Kaplan-Meier Long-term Survival of All Patients and Those Discharged Alive Following Hospitalization



+67% survie à 6ans groupe revascularisation précoce

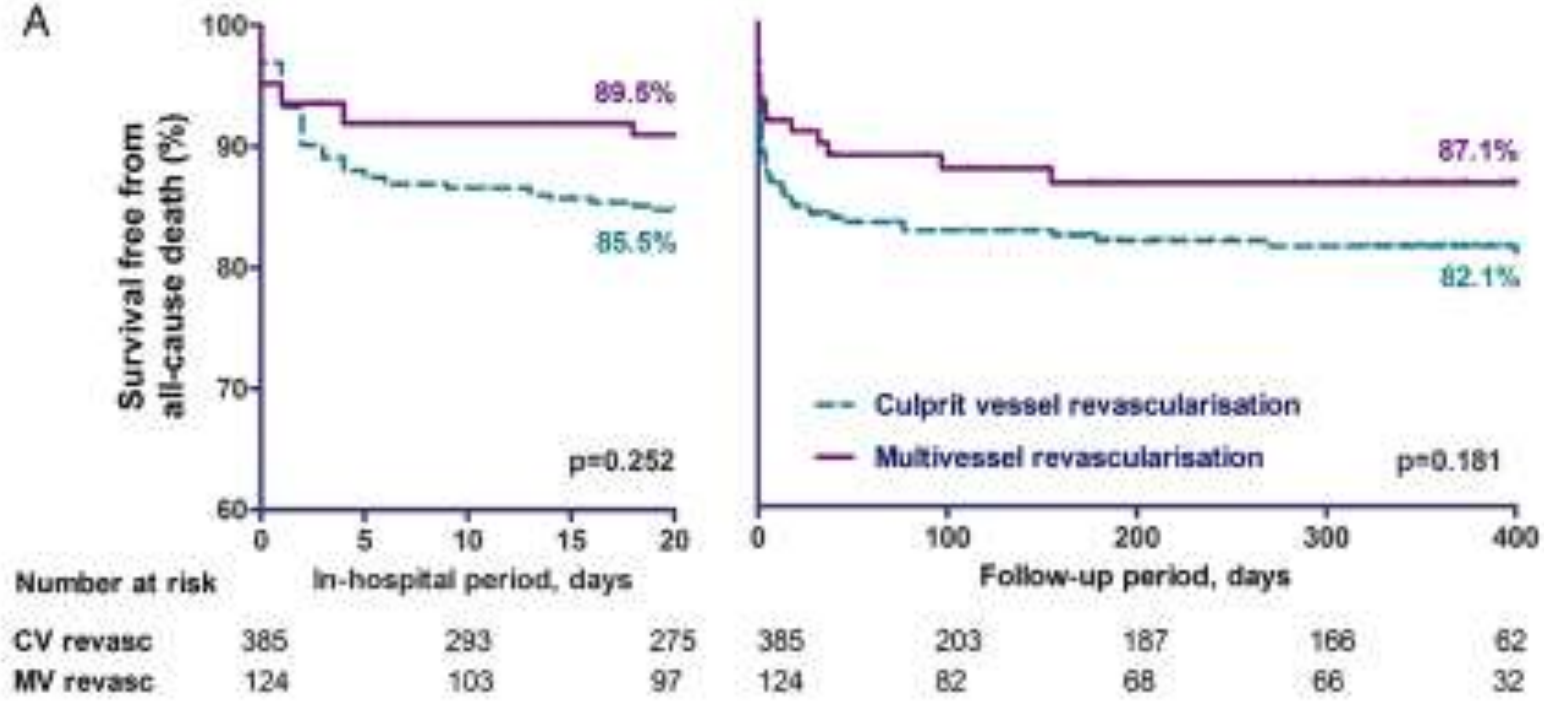
Use and Outcomes of Multivessel Percutaneous Coronary Intervention in Patients With Acute Myocardial Infarction Complicated by Cardiogenic Shock (from the EHS-PCI Registry).

Bauer T, Zeymer U, Hochadel M, Möllmann H, Weidinger F, Zahn R, Nef HM, Hamm CW, Marco J, Gitt AK.

- The value of multivessel percutaneous coronary intervention (MV-PCI) in patients with cardiogenic shock (CS) and multivessel disease (MVD) is still unclear because randomized controlled trials are missing.
- 336 patients with acute myocardial infarction complicated by CS and ≥70% stenoses in ≥2 major epicardial vessels were included in this analysis of the Euro Heart Survey PCI registry.
- Patients undergoing MV-PCI (n = 82, 24%) were compared to those with single-vessel PCI (n = 254, 76%).
- Patients with ventilation were more likely to receive MV-PCI (30% vs 19%, p = 0.05).
- There was a tendency toward a higher hospital mortality in patients with MV-PCI (48.8% vs 37.4%, p = 0.07).
- After adjustment for confounding variables, no significant difference for in-hospital mortality (odd ratio [OR] 1.28, 95% confidence interval [CI] 0.72 to 2.28)
- **MV-PCI is currently used in only 1/4 of patients with CS and MVD. An additional nonculprit PCI was not associated with a survival benefit in these high risk patients.**

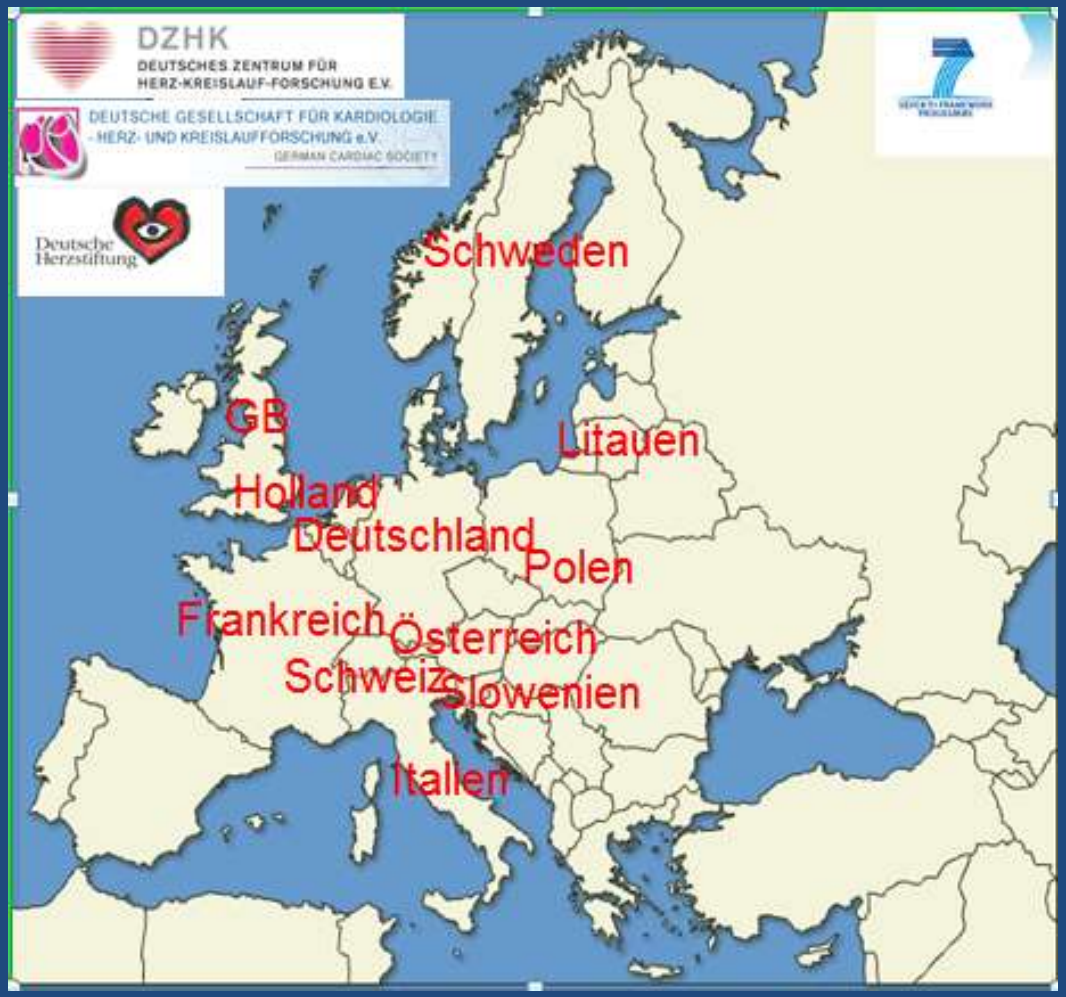
Am J Cardiol. 2012 Jan 9.

Culprit or multivessel revascularisation in ST-elevation myocardial infarction with cardiogenic shock



Park et al. Heart 2015

CULPRIT-SHOCK Study



IHF GmbH 2014

DSMB:
Peter Clemmensen
Ferenc Follath
Karl Wegscheider

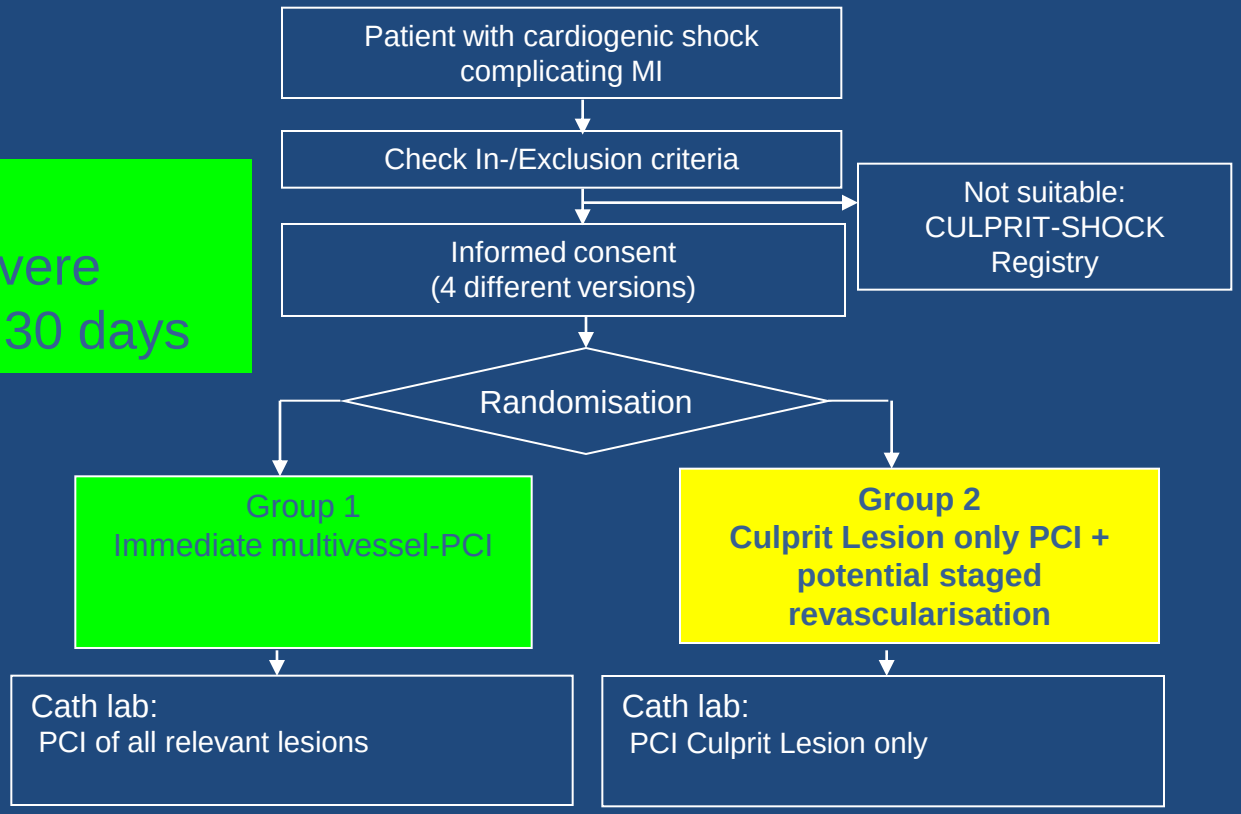
PI + Coordination:
Holger Thiele

Co-PI:
Steffen Desch
Uwe Zeymer

National Coordinators:
Kurt Huber
Gilles Montalescot
Jan Piek
Holger Thiele
Pranas Serpytis
Janina Stepinska
Stefan James
Marko Noc
Keith Oldroyd
Stefan Windecker
Stefano Savonitto

Trial Flow

Primary endpoint:
Mortality and/or severe renal failure within 30 days



Revascularisation chirurgicale

- **Pas d'indication en 1ère intention**
- **Si échec ou impossibilité ATC ???**

- **Petites études: bons résultats**
  - **Décharge ventriculaire due CEC**
  - **Revascularisation complète**
  - **Biais de sélection**

Fibrinolyse

- **Réduit l'incidence des chocs cardiogéniques si efficace.** **GUSTO-I (*JACC 1995*)**
- **Inefficace dans le choc constitué.**
  - GISSI : mortalité inchangée vs PCB (streptokinase) GISSI-I *(Lancet 1986)*
  - Kennedy (J Am Coll Cardiol 1985, 55, 871-877.) : SK intracoronaire. Reperfusion moins bonne chez les patients choqués (43 vs 71%). Par contre , si succès baisse de la mortalité (42 vs 84%).
- **Physiopathologie**
  - Baisse de la PAM et collapsus passif de l'artère $\Rightarrow \Downarrow$ pénétration dans le thrombus
  - Acidose inhibe la conversion du plasminogène en plasmine
  - Efficacité améliorée par les vasopresseurs.

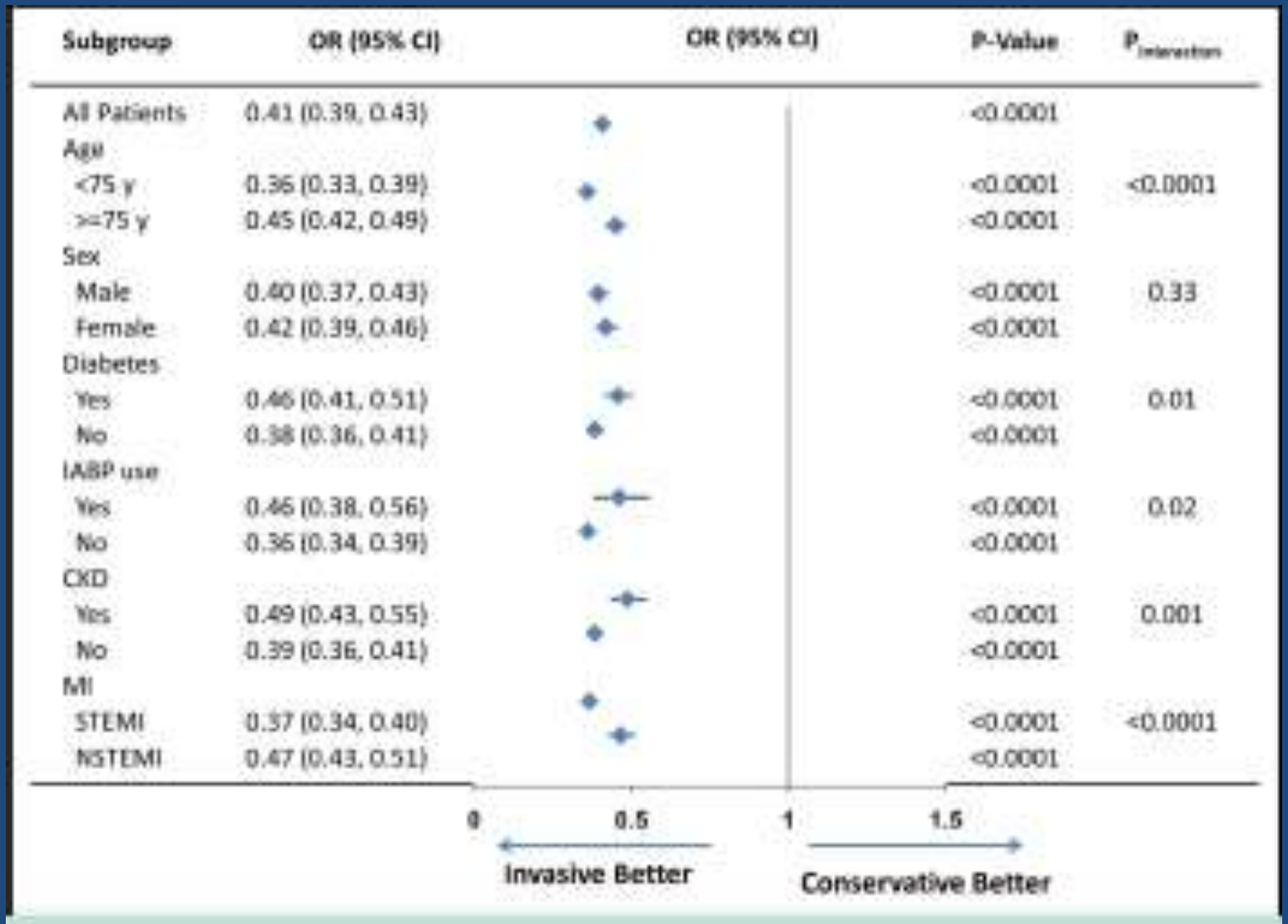


Figure 3 In-hospital mortality among patients with cardiogenic shock managed invasively vs conservatively in the propensity score-matched cohort.

Bangalore et al. AM J Med 2015

Traitement médical

Patient choqué = VIP

Ventilation, Infusion, Pompe

O2, VAC

Remplissage pcap 20-22mmHg

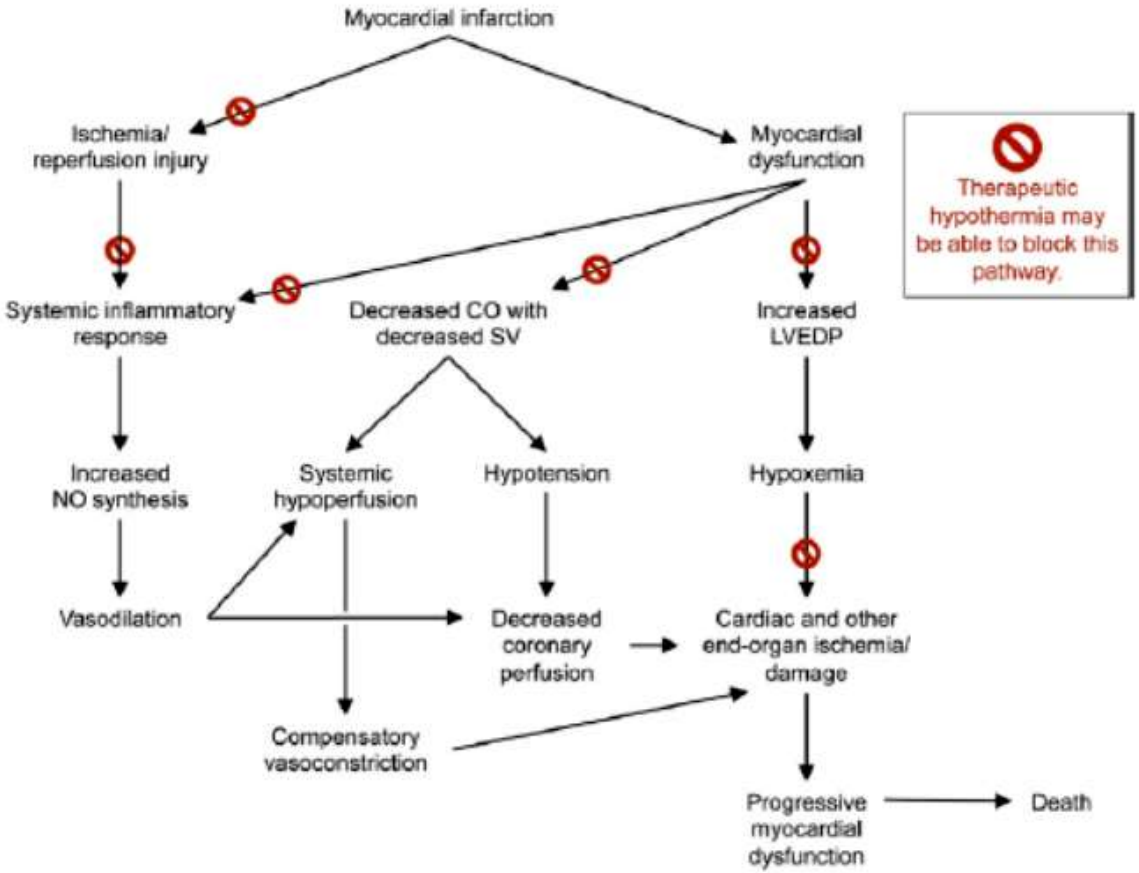
Weil MH, et al. JAMA 1969

Ventilation mécanique

- **Effets sur l'oxygénation.**
- **Effets hémodynamiques.**
  - **↑ pression intrathoracique.**
    - **Baisse de la précharge VG par baisse du retour veineux et du volume sanguin intrathoracique.**
    - **Baisse de la postcharge par baisse de la pression transmurale VG.**

    (pression intracavitaire VG – PIT).

Hypothermie thérapeutique?



Stegman et al. Therapeutic Hypothermia for Cardiogenic Shock JACC Vol. 59, No. 7, 2012

Hypothermie

- Hypoperfusion → dysfonction multi organes → mortalité et morbidité,
- Hypothermie thérapeutique diminue le métabolisme de 5 à 7% par degré de diminution de la température corporelle,
- ↘ consommation en oxygène,
- ↘ production en dioxyde de carbone
- ↘ consommation de glucose
- étude pilote chez l'homme (à moins de 35°C au moment de la reperfusion) *Gotberg M et al. CCI 2010*
  - diminution de la taille de l'infarctus
  - la réduction de l'élévation des biomarqueurs
- Complications: infectieux, coagulopathies, arythmie
- Pas d'aggravation du pronostic chez patients survivant d'arrêt cardiaque
- L'abaissement à 33° ne semble apporter de bénéfice comparé à 36° *Nielsen et al. NEJM 2013*
- La précocité de l'induction , en pré hospitalier ne semble améliorer la survie *Kim et al JAMA 2013*

Catécholamines

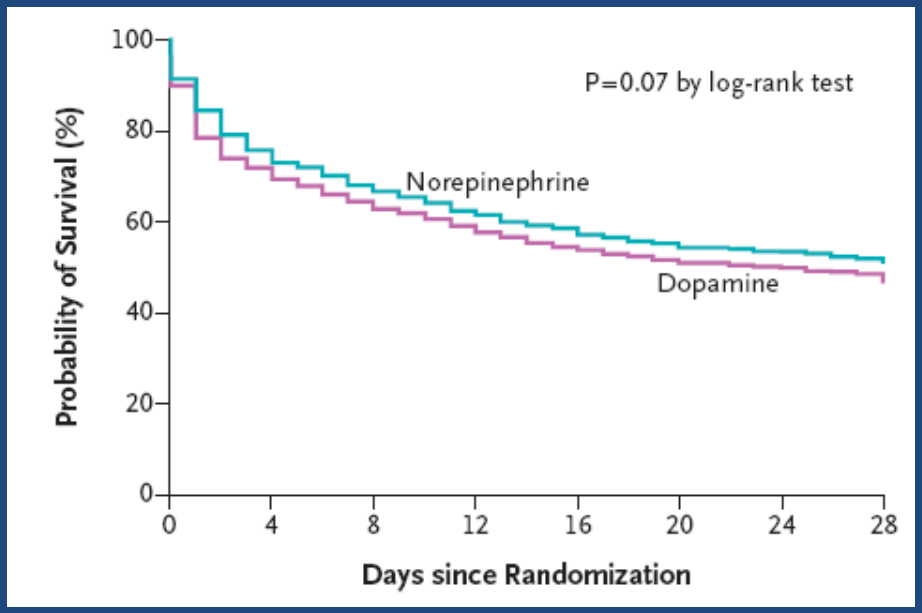
- **Catécholamines inotropes et vasopressives**
  - **Noradrenaline +++**
  - « ***It should be used at the lowest possible dose and titrated until the systolic arterial pressure rises to at least 80 mmHg. Subsequently and because its beta-2-adrenergic effect dobutamine can be given simultaneously to improve contractility.*** » **ESC 2012**
  - **Adrénaline : surtout si nécrose VD**
  - **Dobutamine.**
  - **Diminution de la survie** *(Thackray S, The effectiveness and relative effectiveness of intravenous inotropic drugs acting through the adrenergic pathway in patients with heart failure-a meta-regression analysis. Eur J Heart Fail 2002).*

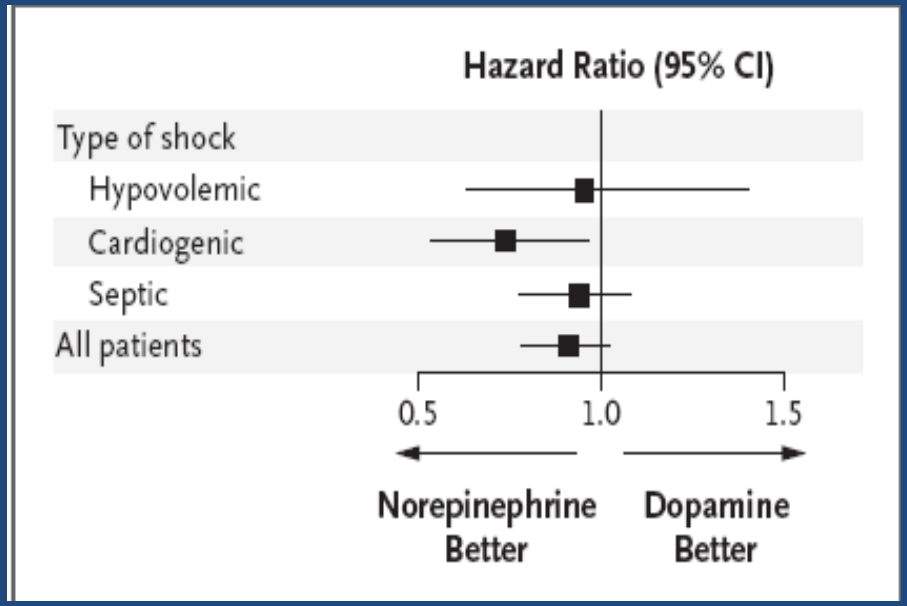
The NEW ENGLAND JOURNAL of MEDICINE

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

Comparison of Dopamine and Norepinephrine in the Treatment of Shock

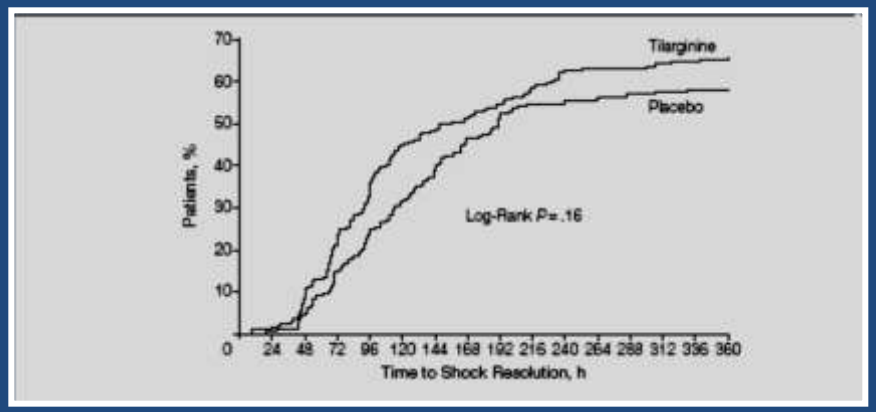
Daniel De Backer, M.D., Ph.D., Patrick Biston, M.D., Jacques Devriendt, M.D., Christian Madl, M.D., Didier Chochrad, M.D., Cesar Aldecoa, M.D., Alexandre Brasseur, M.D., Pierre Defrance, M.D., Philippe Gottignies, M.D., and Jean-Louis Vincent, M.D., Ph.D., for the SOAP II Investigators*

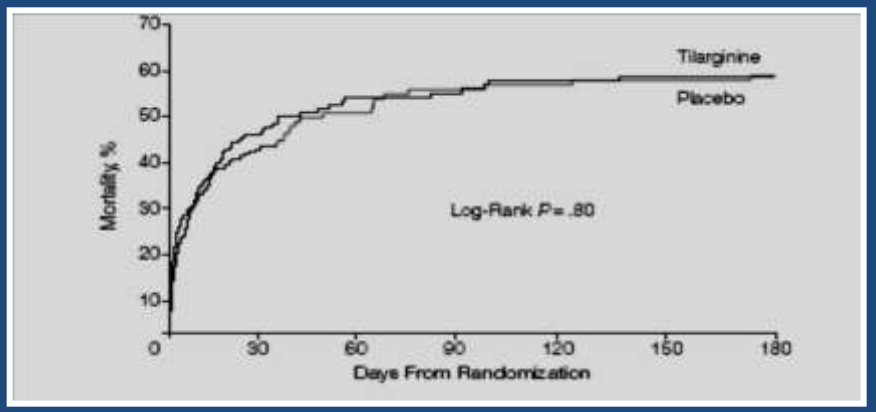




NO?

- Inhibition non sélective iNOS: L-NMMA
- Non randomisée: prometteur *(Cotter G et al Circulation 2000, Eur Heart J 2003)*
- SHOCK-2: pas différence significative *(Dzavik V et al. Eur Heart J 2007)*
- TRIUMPH: Tilarginine dans IDM compliqué de choc et revascularisé: pas de diminution de la mortalité *(Triumph investigators, JAMA,2007)*

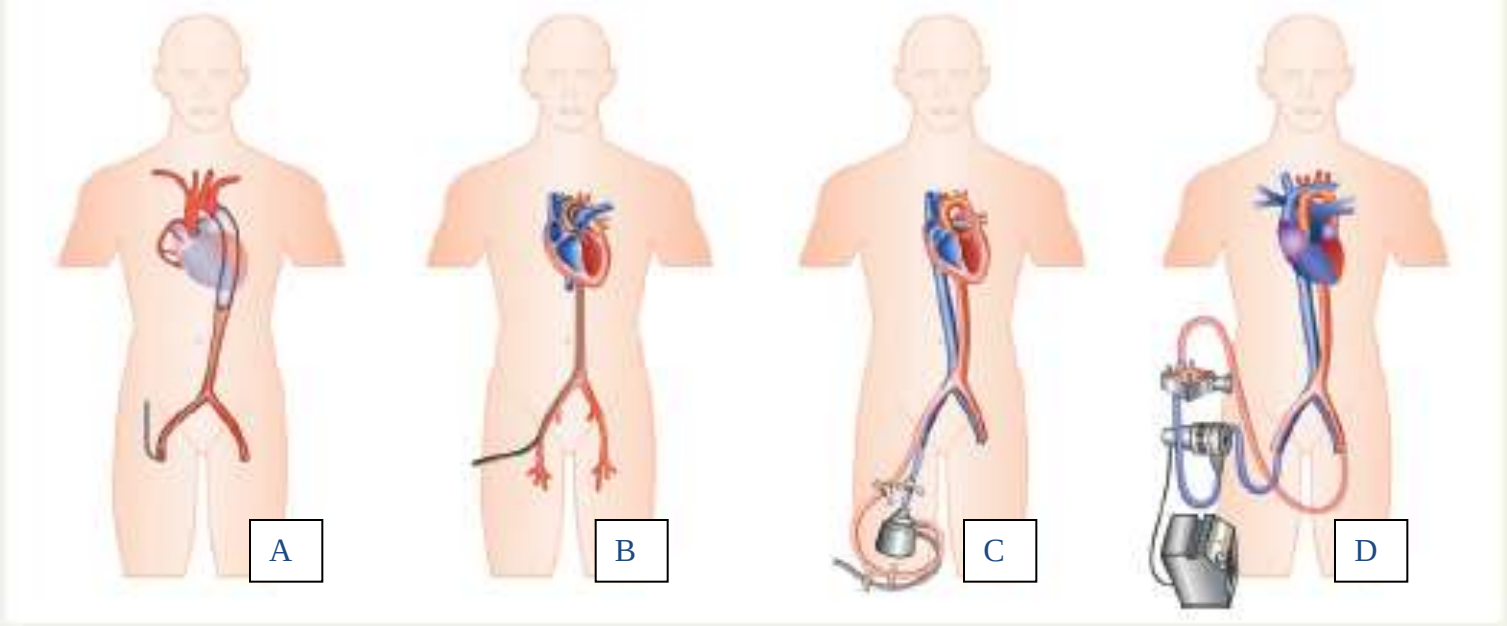




Assistances circulatoires

- **Absence amélioration malgré ATL, ou ATL impossible**
- **Patients jeunes**
- **Quand ?**
  - **Nécessité augmenter doses inotropes**
  - **Avant défaillance rénale, hépatique, hypotension majeure**

Assistances circulatoires

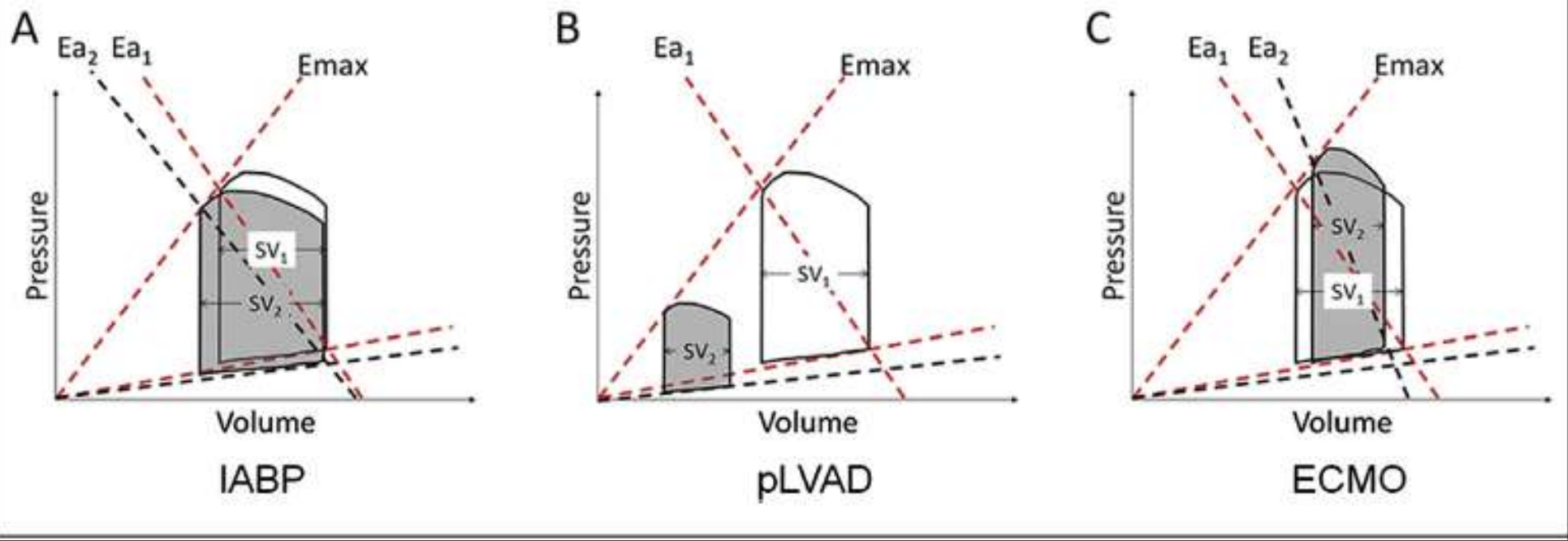


A: CPIAB; B: IMPELLA; C: Tandem Heart; D: ECMO

	CPIAB	IMPELLA	Tandem Heart	ECMO
Mécanisme de la pompe	pneumatique	Rotation axiale	centrifuge	centrifuge
Taille du désilet d'insertion	7-9 French	13 French	21 French canule veineuse ; 15-17 French canule artériel	18-21 French canule veineuse ; 15-22 French canule artériel
Insertion	Aorte descendante par l'artère fémorale	Cat éther 12 French placé par voie rétrogradé au travers de la valve aortique	Canule d'aspiration placée dans l'oreillette gauche par ponction transseptale, réinjection dans l'aorte descendante à la bifurcation iliaque	Canule d'aspiration dans l'oreillette droite par la veine fémorale, réinjection après oxygénation dans l'aorte descendante
Support hémodynamique (L/min)	0,5-1	2,5	4	4,5
Durée d'implantation	+	+	+++	++
Risque d'ischémie de membre	+	+	+++	+++
Anticoagulation	+	+	+++	+++
Hémolyse	+	++	++	++
Complexité d'ablation	+	++	++++	+++
Effet sur la post charge	diminution	diminution	augmentation	augmentation
Effet sur le volume d'éjection VG	légère augmentation	diminution	diminution	diminution
Effet sur la perfusion coronaire	légère augmentation	augmentation	inconnue	inconnue
Effet sur la pré charge VG	légère diminution	diminution	diminution	diminution
Effet sur la pression capillaire	légère diminution	diminution	diminution	diminution
Effet sur la perfusion tissulaire périphérique	pas d'augmentation	augmentation	augmentation	augmentation
Effet sur la demande en oxygène du myocarde	**légère diminution**	**diminution**	**diminution**	**augmentation**

D'après Manzo-Silberman et Deye, Réanimation 2014

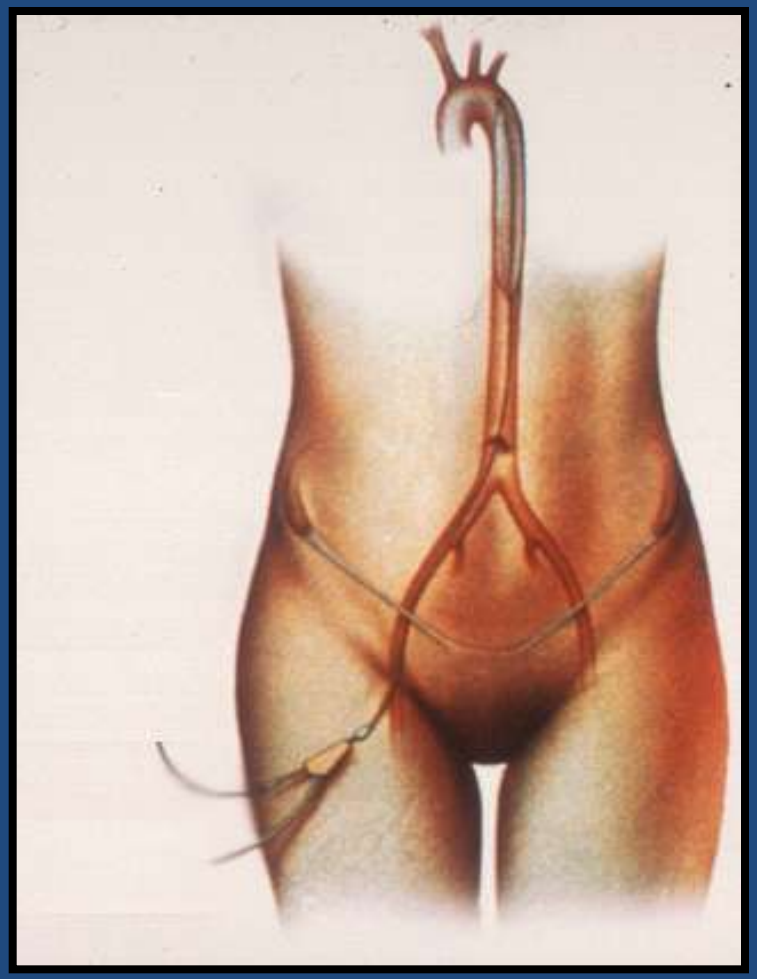
FIGURE 2 Cardiac Effects of Mechanical Support



2015 SCAI/ACC/HFSA/STS Clinical Expert Consensus Statement on the Use of Percutaneous Mechanical Circulatory Support Devices in Cardiovascular Care

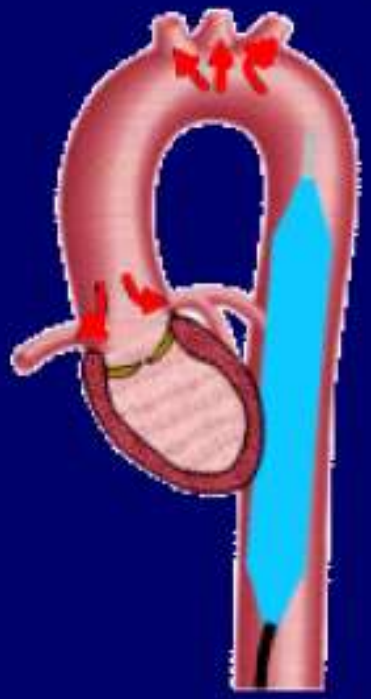
Contre-pulsionpar ballonnet intra-aortique

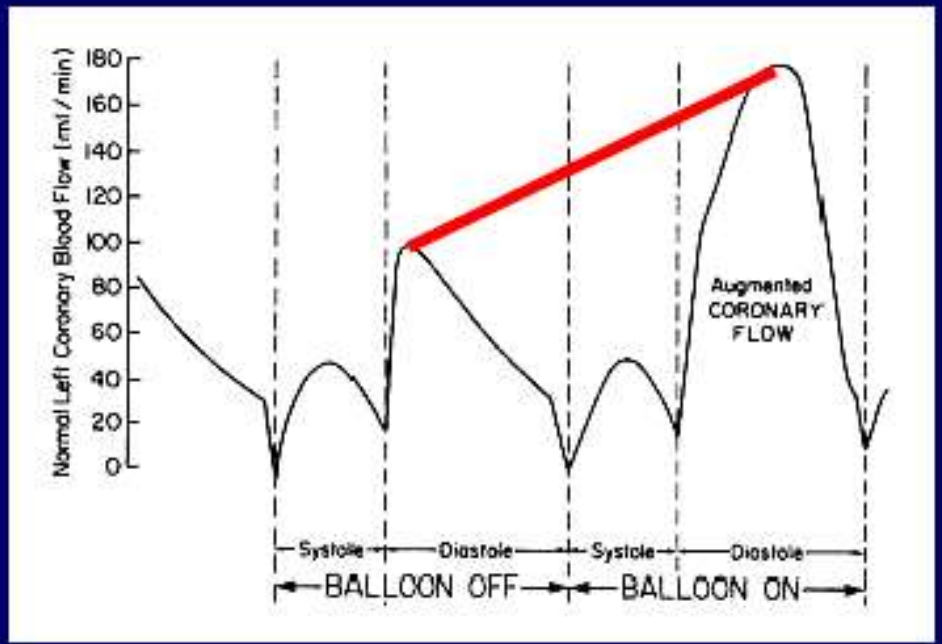
- **Synchronisée à ECG**
- **Inflation fermeture valve Aortique**
- **Déflation pré-systole**

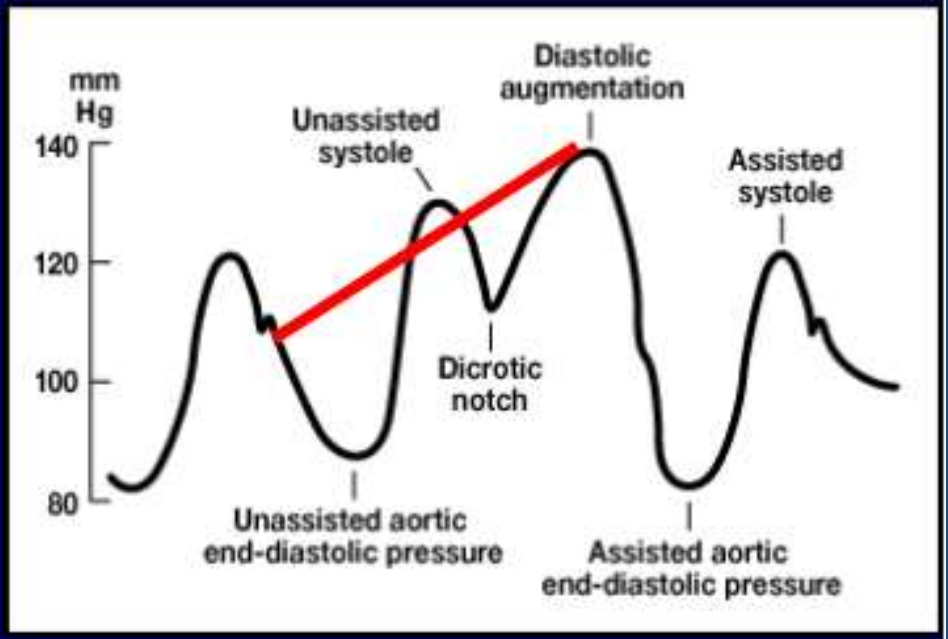


Benefits of IAB inflation

- Increased CBF
- Increased diastolic pressure
- Increased systemic perfusion

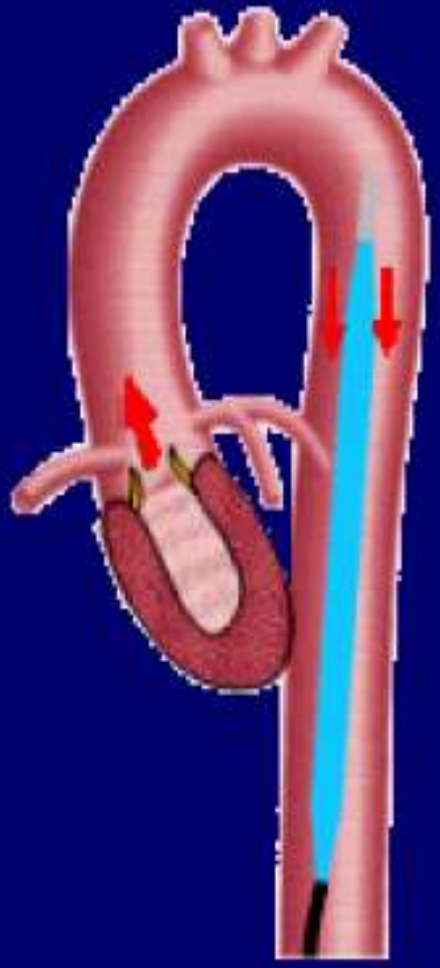






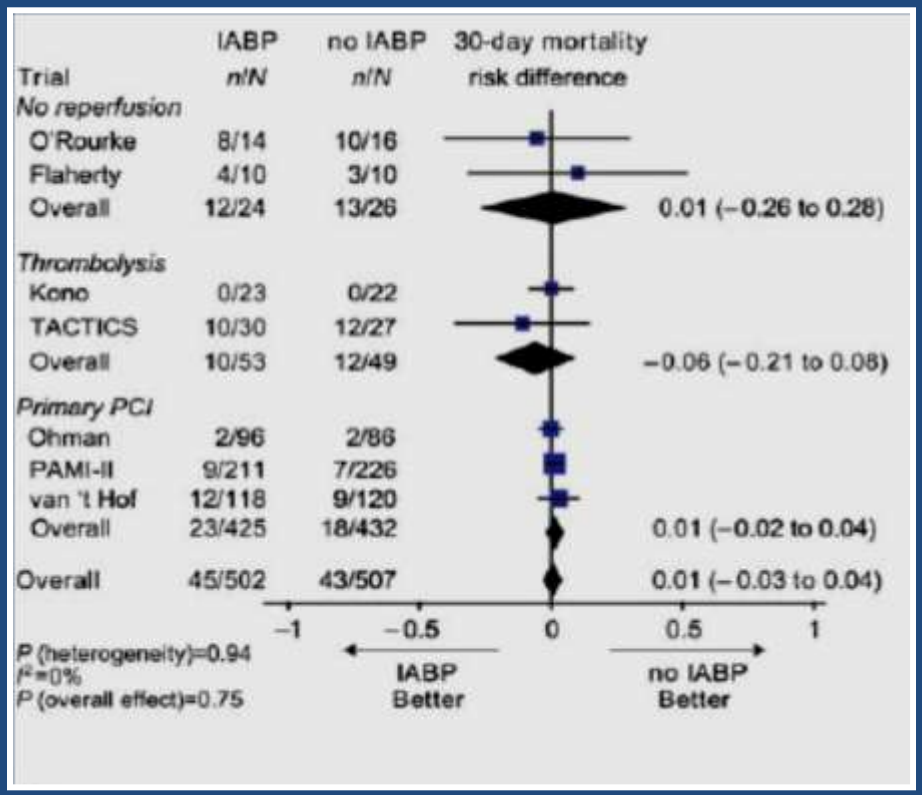
Benefits of IAB deflation

- Decreased Afterload
- Increased Stroke Volume
- Enhanced organ perfusion

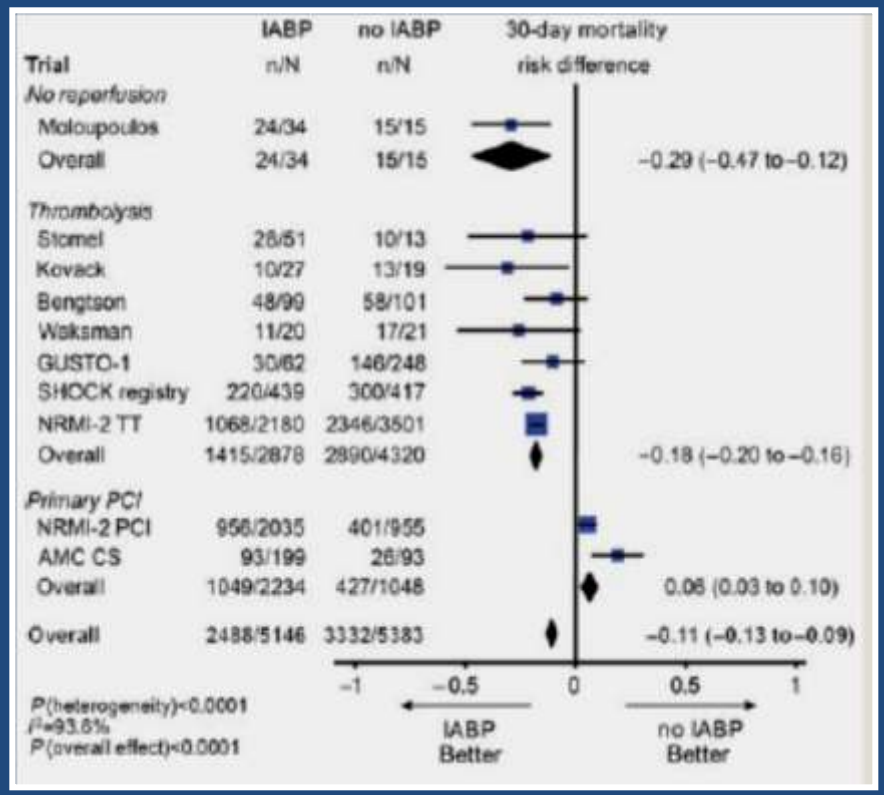


A systematic review and meta-analysis of intra-aortic balloon pump therapy in ST-elevation myocardial infarction: should we change the guidelines?

K. D. Sjauw et al. European Heart Journal (2009)



Meta-analyse d'études randomisées de l'utilisation de la CPIAB dans IDM STEMI



Meta-analyse de cohorte CPIAB dans IDM compliqué de choc cardiogénique

The Prospective, Randomised IABP - Shock-Trial - Hemodynamic Effects of Intraaortic Balloon Counterpulsation in Patients With Acute Myocardial Infarction complicated by Cardiogenic Shock.

Prondzinsky R et al Shock. 2012

- **METHODS:** PCI of IRA (infarct-related artery) was performed in 40 patients with AMI complicated by cardiogenic shock, within 12 h of onset of hemodynamic instability. Serial hemodynamic parameters were determined over the next 4 days and compared in patients receiving medical treatment alone with those treated with additional intraaortic balloon counterpulsation.

- **RESULTS:** There were no significant differences among severity of disease (i.e., APACHE II score) initially and no differences among both groups for disease improvement. We observed significant temporal improvements of CO (4.8±0.5 to 6.0±0.5 l/min), SVR (926+73 to 769±101 dyn·s·cm), and the prognosis-validated cardiac power output (CPO; 0.78±0.06 to 1.01±0.2 W) within IABP group. However, there were no significant differences between the IABP group compared to the medical alone group.

- **CONCLUSIONS:** Additional IABP treatment did not result in a significant hemodynamic improvement compared to medical therapy alone in a randomized prospective trial in patient with cardiogenic shock following PCI. **Therefore, the use and recommendation for IABP-treatment in CS remains unclear.**

IABP-SHOCK 2

Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

Holger Thiele, M.D., Uwe Zeymer, M.D., Franz-Josef Neumann, M.D., Miroslaw Ferenc, M.D., Hans-Georg Olbrich, M.D., Jörg Hausleiter, M.D., Gert Richardt, M.D., Marcus Hennersdorf, M.D., Klaus Empen, M.D., Georg Fuernau, M.D., Steffen Desch, M.D., Ingo Eitel, M.D., Rainer Hambrecht, M.D., Jörg Fuhrmann, M.D., Michael Böhm, M.D., Henning Ebelt, M.D., Steffen Schneider, Ph.D., Gerhard Schuler, M.D., and Karl Werdan, M.D., for the IABP-SHOCK II Trial Investigators*

University of Leipzig–Heart Center, Leipzig

Baseline Characteristics of the Patients.

Table 1. Baseline Characteristics of the Patients.*

Characteristic	IABP (N=301)	Control (N=299)
Age — yr		
Median	70	69
Interquartile range	58–78	58–76
Male sex — no. (%)	202 (67.1)	211 (70.6)
Weight — kg		
Median	80	81
Interquartile range	73–90	73–90
Height — cm		
Median	172	175
Interquartile range	165–178	168–180
Body-mass index†		
Median	27.5	26.9
Interquartile range	24.7–30.1	24.7–29.4
Cardiovascular risk factors — no./total no. (%)		
Current smoking	96/295 (32.5)	108/299 (36.1)
Hypertension	213/296 (72.0)	199/299 (66.6)
Hypercholesterolemia	122/295 (41.4)	105/299 (35.1)
Diabetes mellitus	105/297 (35.4)	90/299 (30.1)
Prior myocardial infarction — no./total no. (%)	71/300 (23.7)	61/299 (20.4)
Prior stroke — no./total no. (%)	24/300 (8.0)	20/299 (6.7)
Known peripheral arterial disease — no./total no. (%)	40/300 (13.3)	33/299 (11.0)
Prior PCI — no./total no. (%)	63/299 (21.1)	52/299 (17.4)
Prior bypass surgery — no./total no. (%)	20/300 (6.7)	12/299 (4.0)

* Patients were randomly assigned to intraaortic balloon counterpulsation (IABP) or no intraaortic balloon counterpulsation (control); all the patients were expected to undergo early revascularization and to receive the best available medical therapy. There were no significant differences between the groups in the baseline characteristics listed here. PCI denotes percutaneous coronary intervention.

† The body-mass index is the weight in kilograms divided by the square of the height in meters.

Thiele H et al. N Engl J Med 2012;367:1287-1296

The NEW ENGLAND JOURNAL of MEDICINE

Clinical Course before Randomization.

Table 2. Clinical Course before Randomization.*

Variable	IABP (N = 301)	Control (N = 299)
Sign of impaired organ perfusion — no./total no. (%)		
Altered mental status	215/300 (71.7)	232/299 (77.6)
Cold, clammy skin and extremities	257/300 (85.7)	245/299 (81.9)
Oliguria	90/300 (30.0)	99/299 (33.1)
Serum lactate >2.0 mmol/liter	226/300 (75.3)	218/298 (73.2)
Serum lactate — mmol/liter		
Median	3.6	4.7
Interquartile range	2.1–7.2	2.3–8.2
Fibrinolysis <24 hr before randomization — no. (%)	28 (9.3)	20 (6.7)
Resuscitation before randomization — no. (%)	127 (42.2)	143 (47.8)
Myocardial infarction — no./total no. (%)		
Non–ST-segment elevation	96/300 (32.0)	81/298 (27.2)
ST-segment elevation	200/300 (66.7)	212/298 (71.1)
Anterior	136/298 (45.6)	116/296 (39.2)
Systolic blood pressure — mm Hg		
Median	89	90
Interquartile range	79–107	80–109
Diastolic blood pressure — mm Hg		
Median	55	55
Interquartile range	46–67	45–65
Mean blood pressure — mm Hg†		
Median	69	68
Interquartile range	59–80	59–80
Use of catecholamines at randomization — no./total no. (%)	270/301 (89.7)	268/298 (89.9)
Heart rate — beats/min		
Median	92	92
Interquartile range	72–110	75–110
Creatinine — mg/dl		
Median	1.30	1.26
Interquartile range	1.04–1.67	1.03–1.64
Creatinine clearance — ml/min‡		
Median	60.7	56.8
Interquartile range	43.4–86.6	39.7–78.1
No. of diseased vessels — no./total no. (%)		
1	61/296 (20.6)	65/293 (22.2)
2	81/296 (27.4)	74/293 (25.3)
3	154/296 (52.0)	154/293 (52.6)
Infarct-related artery — no./total no. (%)		
Left anterior descending	132/293 (45.1)	121/293 (41.3)
Left circumflex	55/293 (18.8)	57/293 (19.5)
Right coronary artery	73/293 (24.9)	79/293 (27.0)
Left main	26/293 (8.9)	28/293 (9.6)
Bypass graft	7/293 (2.4)	8/293 (2.7)
Left ventricular ejection fraction — %		
Median	35	35
Interquartile range	25–45	25–45

* There were no significant differences between the groups with respect to any of the variables listed. To convert the values for creatinine to micromoles per liter, multiply by 88.4.
† The mean blood pressure, an approximation of the time-weighted average of blood pressure values in large arteries during the cardiac cycle, is derived from the area under the curve for invasive blood pressure measurements.
‡ Creatinine clearance was calculated with the use of the Cockcroft–Gault formula.

Thiele H et al. N Engl J Med 2012;367:1287-1296

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Clinical Outcomes.

Table 3. Clinical Outcomes.

Outcome	IABP (N=300)	Control (N=298)	P Value	Relative Risk with IABP (95% CI)
	number (percent)			
Primary end point: all-cause mortality at 30 days	119 (39.7)	123 (41.3)	0.69	0.96 (0.79–1.17)
Reinfarction in hospital	9 (3.0)	4 (1.3)	0.16	2.24 (0.70–7.18)
Stent thrombosis in hospital	4 (1.3)	3 (1.0)	0.71	1.32 (0.30–5.87)
Stroke in hospital	2 (0.7)	5 (1.7)	0.28	0.40 (0.08–2.03)
Ischemic	2 (0.7)	4 (1.3)	0.45	0.49 (0.09–2.71)
Hemorrhagic	0	1 (0.3)	0.50	—
Peripheral ischemic complications requiring intervention in hospital	13 (4.3)	10 (3.4)	0.53	1.29 (0.58–2.90)
Bleeding in hospital*				
Life-threatening or severe	10 (3.3)	13 (4.4)	0.51	0.76 (0.34–1.72)
Moderate	52 (17.3)	49 (16.4)	0.77	1.05 (0.74–1.50)
Sepsis in hospital	47 (15.7)	61 (20.5)	0.15	0.77 (0.54–1.08)

* Bleeding during the hospital stay was assessed according to the Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO) criteria.

Thiele H et al. N Engl J Med 2012;367:1287-1296

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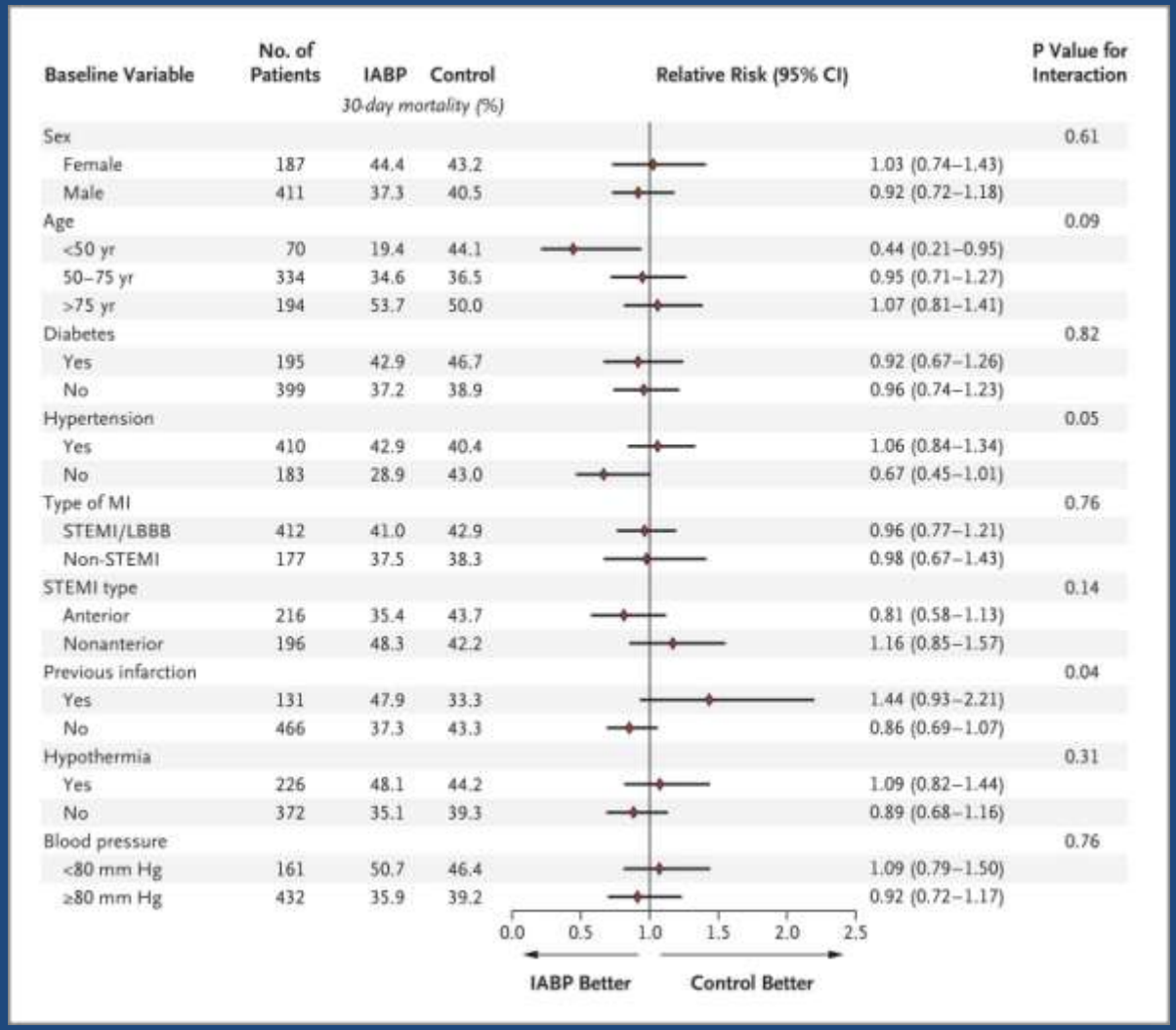
Subgroup Analyses of the Primary End Point.



Baseline Variable	No. of Patients	IABP	Control	Relative Risk (95% CI)	P Value for Interaction
		30-day mortality (%)			
Sex					0.61
Female	187	44.4	43.2	1.03 (0.74–1.43)	
Male	411	37.3	40.5	0.92 (0.72–1.18)	
Age					0.09
<50 yr	70	19.4	44.1	0.44 (0.21–0.95)	
50–75 yr	334	34.6	36.5	0.95 (0.71–1.27)	
>75 yr	194	53.7	50.0	1.07 (0.81–1.41)	
Diabetes					0.82
Yes	195	42.9	46.7	0.92 (0.67–1.26)	
No	399	37.2	38.9	0.96 (0.74–1.23)	
Hypertension					0.05
Yes	410	42.9	40.4	1.06 (0.84–1.34)	
No	183	28.9	43.0	0.67 (0.45–1.01)	
Type of MI					0.76
STEMI/LBBB	412	41.0	42.9	0.96 (0.77–1.21)	
Non-STEMI	177	37.5	38.3	0.98 (0.67–1.43)	
STEMI type					0.14
Anterior	216	35.4	43.7	0.81 (0.58–1.13)	
Nonanterior	196	48.3	42.2	1.16 (0.85–1.57)	
Previous infarction					0.04
Yes	131	47.9	33.3	1.44 (0.93–2.21)	
No	466	37.3	43.3	0.86 (0.69–1.07)	
Hypothermia					0.31
Yes	226	48.1	44.2	1.09 (0.82–1.44)	
No	372	35.1	39.3	0.89 (0.68–1.16)	
Blood pressure					0.76
<80 mm Hg	161	50.7	46.4	1.09 (0.79–1.50)	
≥80 mm Hg	432	35.9	39.2	0.92 (0.72–1.17)	

Thiele H et al. N Engl J Med 2012;367:1287-1296

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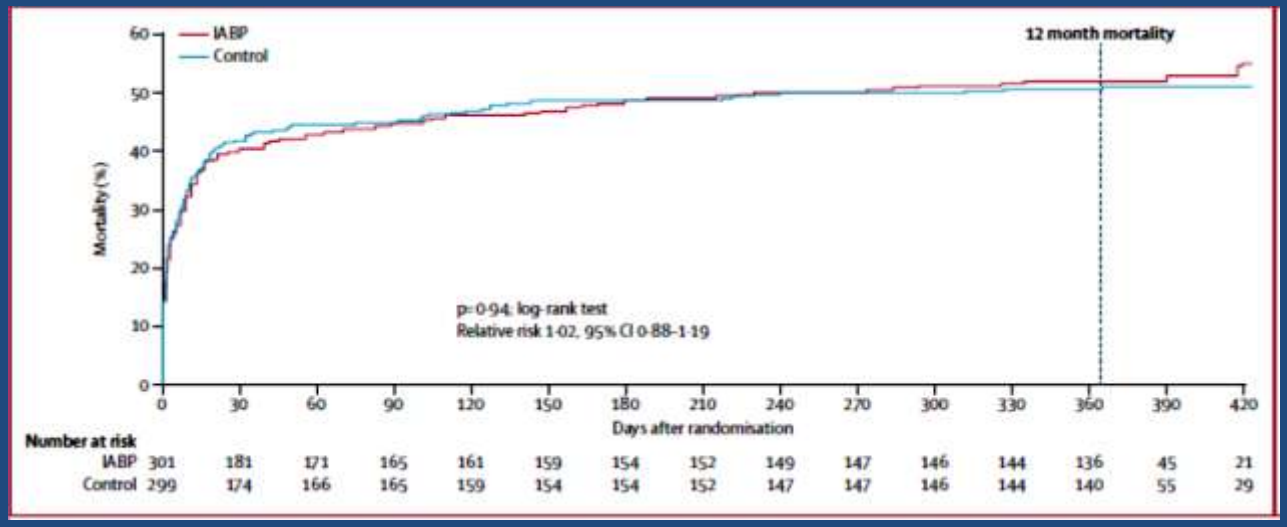
IABP SHOCK à 12 mois

	IABP (n=299)	Control (n=296)	Relative risk (95% CI)	p value
All-cause mortality	155/299 (52%)	152/296 (51%)	1·01 (0·86–1·18)	0·91
Cardiac mortality	150/299 (50%)	148/296 (50%)	1·00 (0·85–1·18)	0·97
Non-cardiac mortality	5/299 (2%)	4/296 (1%)	1·23 (0·34–4·56)	1·00
Events in 1-year survivors				
Reinfarction	13/144 (9%)	5/144 (3%)	2·60 (0·95–7·10)	0·05
Stroke	3/144 (2%)	2/144 (1%)	1·50 (0·25–8·84)	1·00
Recurrent revascularisation	29/144 (20%)	32/144 (22%)	0·91 (0·58–1·41)	0·77
Repeat PCI	22/144 (15%)	25/144 (17%)	0·88 (0·52–1·49)	0·63
Additional CABG	7/144 (5%)	7/144 (5%)	1·00 (0·36–2·78)	1·00
ICD implantation	14/144 (10%)	14/144 (10%)	1·00 (0·49–2·02)	1·00

Data are n/N (%), relative risk (95% CI), or p value. IABP=intra-aortic balloon pump. PCI=percutaneous coronary intervention. CABG=coronary artery bypass grafting. ICD=implantable cardioverter defibrillator.

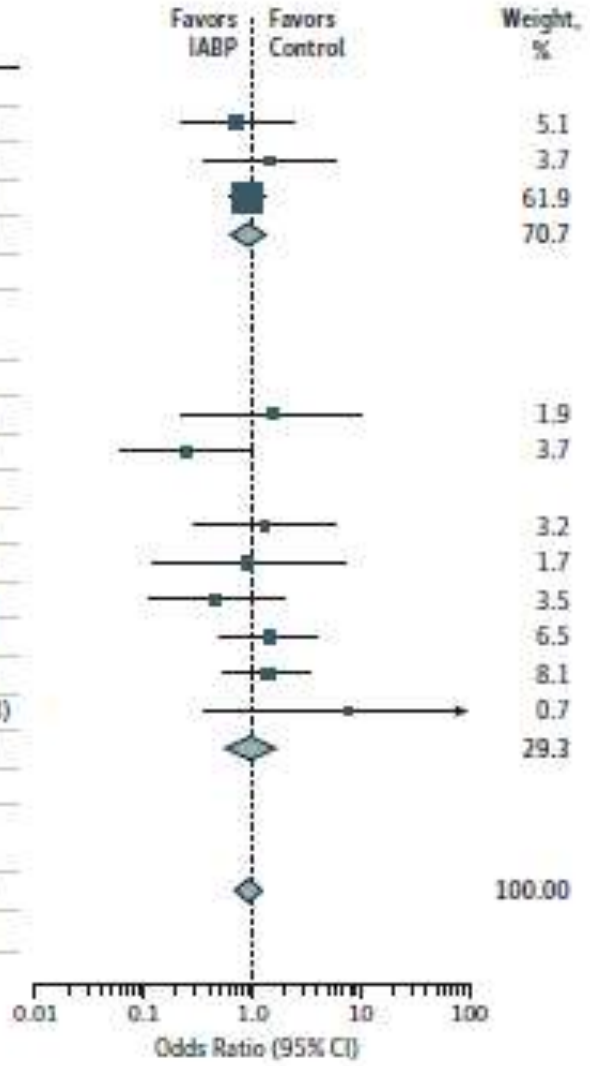
Table 1: **Clinical outcomes at 12 months**



Thiele H, Lancet 2013

Figure 2. Randomized Controlled Trials Comparing Intra-Aortic Balloon Pump (IABP) Therapy With Control for the Outcome of Mortality in Patients With Acute Myocardial Infarction, Stratified by the Presence or Absence of Cardiogenic Shock

Study or Subgroup	IABP Events	IABP Total	Control Events	Control Total	Odds Ratio (95% CI)	Weight, %
Cardiogenic shock						
Ohman et al,[18] 2005	8	30	9	27	0.73 (0.23-2.27)	5.1
Prondzinsky et al,[19] 2010	7	19	6	21	1.46 (0.39-5.51)	3.7
Thiele et al,[2] 2012	119	300	123	298	0.94 (0.67-1.30)	61.9
Subtotal (95% CI)		349		346	0.94 (0.69-1.28)	70.7
Total events	134		138			
Heterogeneity: $\tau^2=0.00$; $\chi^2=0.62$, df = 2 ($P=.73$); $I^2=0\%$						
Test for overall effect: $z=0.39$ ($P=.69$)						
No cardiogenic shock						
Flaherty et al,[20] 1985	4	10	3	10	1.56 (0.24-9.91)	1.9
Gu et al,[32] 2011	3	51	11	55	0.25 (0.07-0.96)	3.7
Kono et al,[22] 1996	0	23	0	22	Not estimable	
O'Rourke et al,[23] 1981	7	14	7	16	1.29 (0.30-5.43)	3.2
Ohman et al,[24] 1994	2	96	2	86	0.89 (0.12-6.48)	1.7
Patel et al,[28] 2011	3	161	7	176	0.46 (0.12-1.80)	3.5
Stone et al,[25] 1997	9	211	7	226	1.39 (0.51-3.81)	6.5
van 't Hof et al,[26] 1999	12	118	9	120	1.40 (0.57-3.45)	8.1
Vijayalakshmi et al,[27] 2007	3	17	0	16	7.97 (0.38-167.53)	0.7
Subtotal (95% CI)		701		727	0.98 (0.57-1.69)	29.3
Total events	43		46			
Heterogeneity: $\tau^2=0.10$; $\chi^2=8.43$, df = 7 ($P=.30$); $I^2=17\%$						
Test for overall effect: $z=0.06$ ($P=.95$)						
Total (95% CI)		1050		1073	0.96 (0.74-1.24)	100.00
Total events	177		184			
Heterogeneity: $\tau^2=0.00$; $\chi^2=9.09$, df = 10 ($P=.52$); $I^2=0\%$						
Test for overall effect: $z=0.33$ ($P=.74$)						
Test for subgroup differences: $\chi^2=0.02$, df = 1 ($P=.89$); $I^2=0\%$						

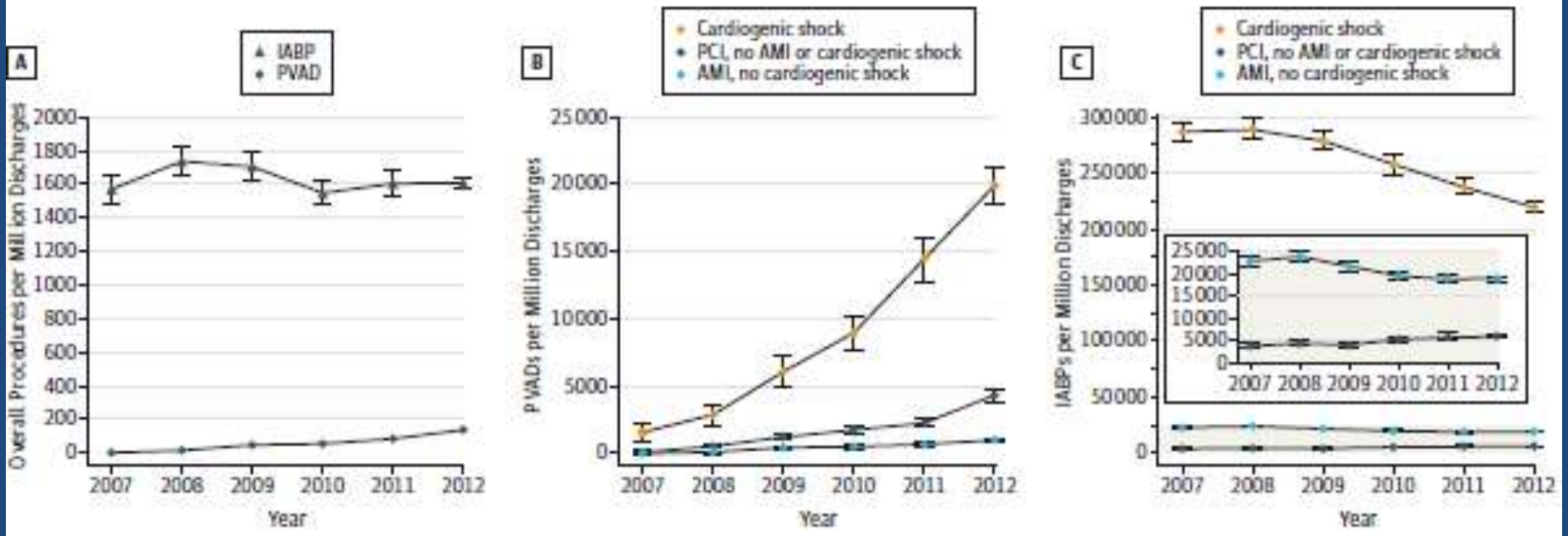


Odds ratios are calculated by random-effects Mantel-Haenszel analysis.

Ahmad, JAMA Int Med 2015

Trends in the Use of Percutaneous Ventricular Assist Devices Analysis of National Inpatient Sample Data, 2007 Through 2012

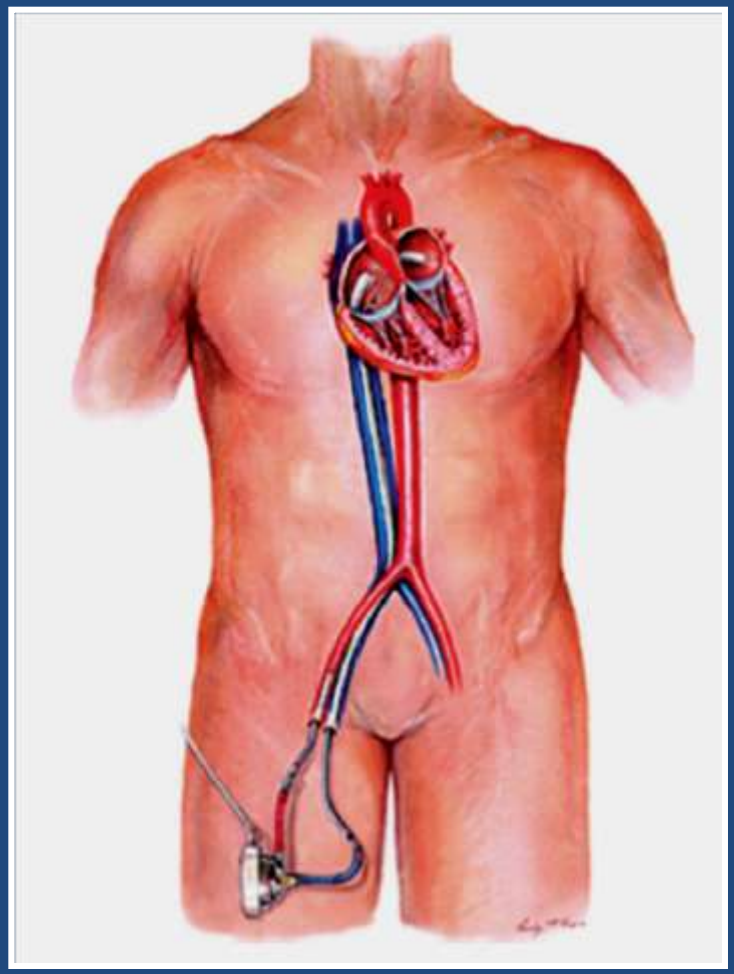
Figure 1. Calendar Year Trends in the Use of Percutaneous Ventricular Assist Devices (PVADs) and Intra-aortic Balloon Pumps (IABPs) in the United States, 2007 Through 2012

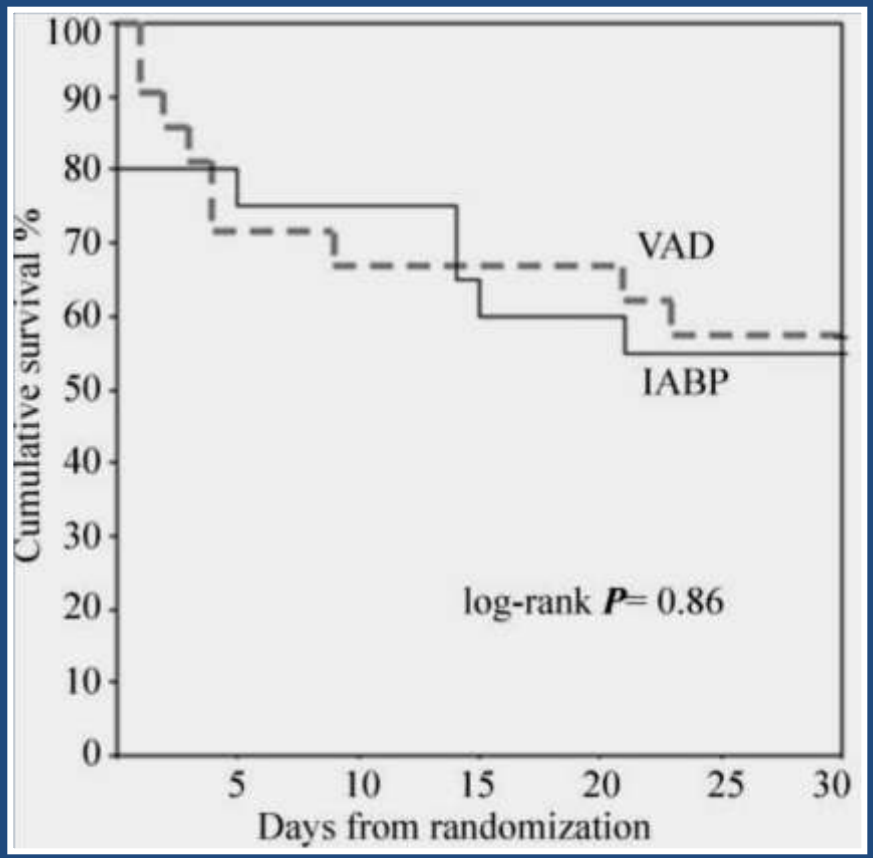


Khera, JAMA Int Med 2015

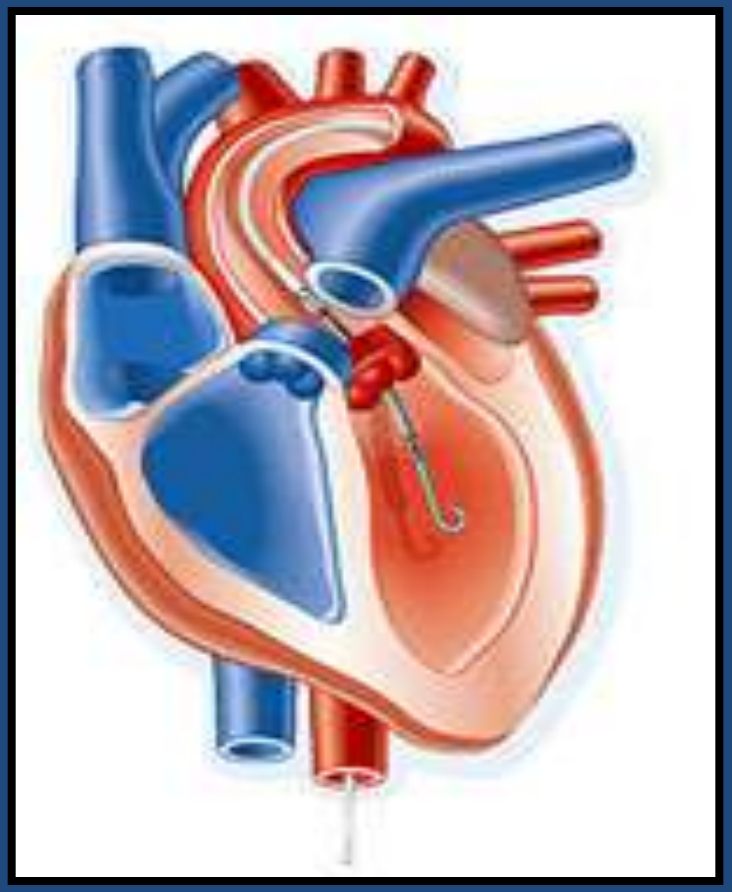
Randomized comparison of intra-aortic balloon support with a percutaneous left ventricular assist device in patients with revascularized acute myocardial infarction complicated by cardiogenic shock

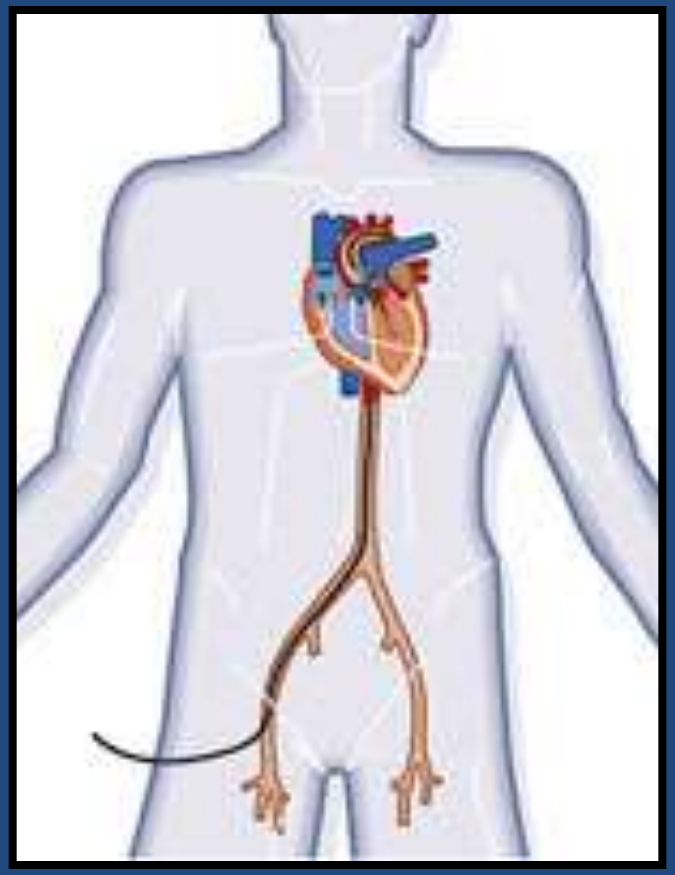
H. Thiele et al. European Heart Journal (2005)





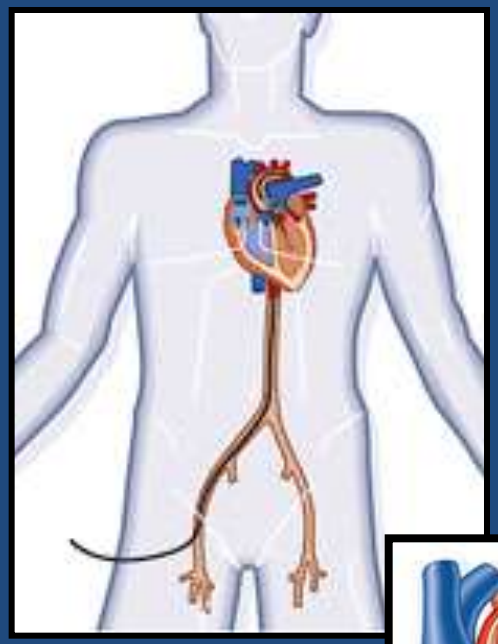
Impella LP2.5

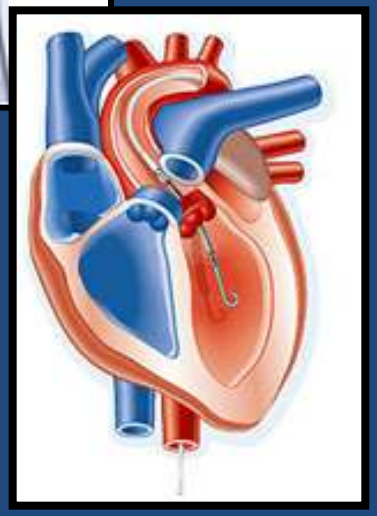




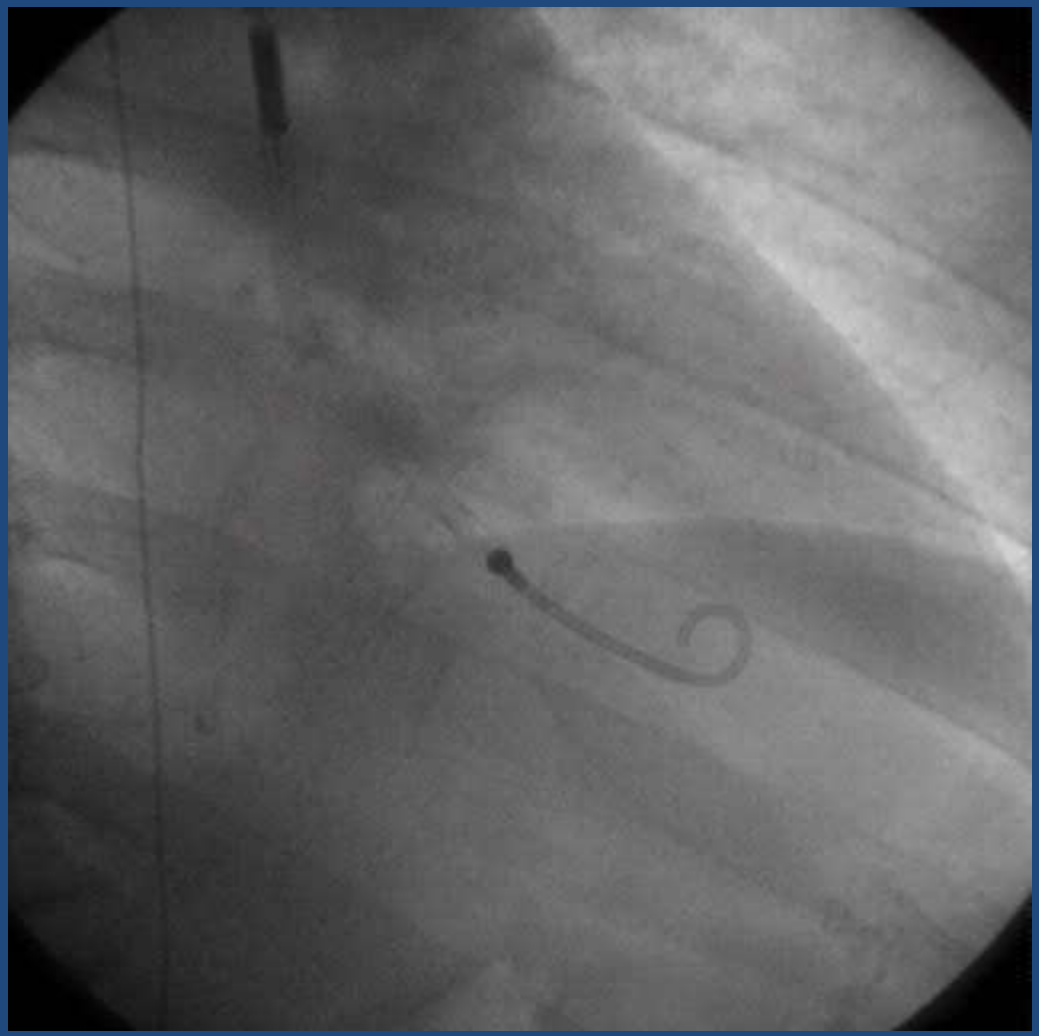
Implantation

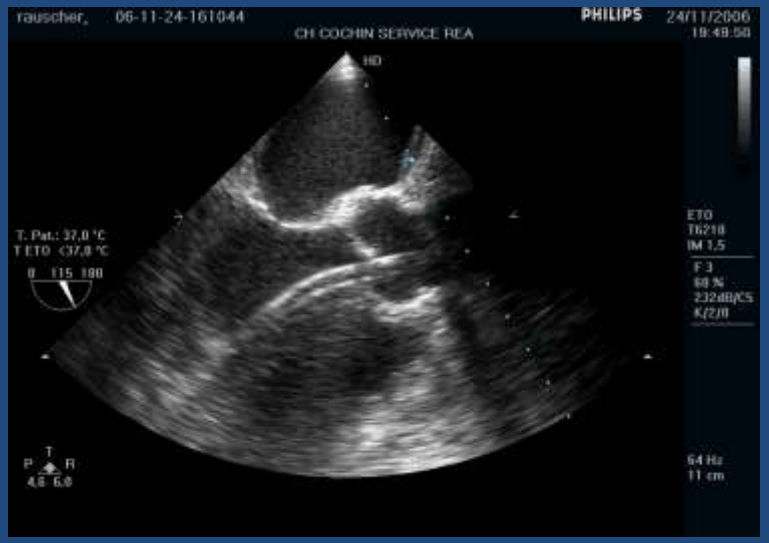
- Percutané : désilet de 13F
- placement de la canule est contrôlé sur la console par la mesure en continu de la pression VG et aortique.
- Sous scopie ou sous ETO
- Le débit d’aspiration-éjection est réglable et peut atteindre 2.5 L/min avec Impella LP2.5.

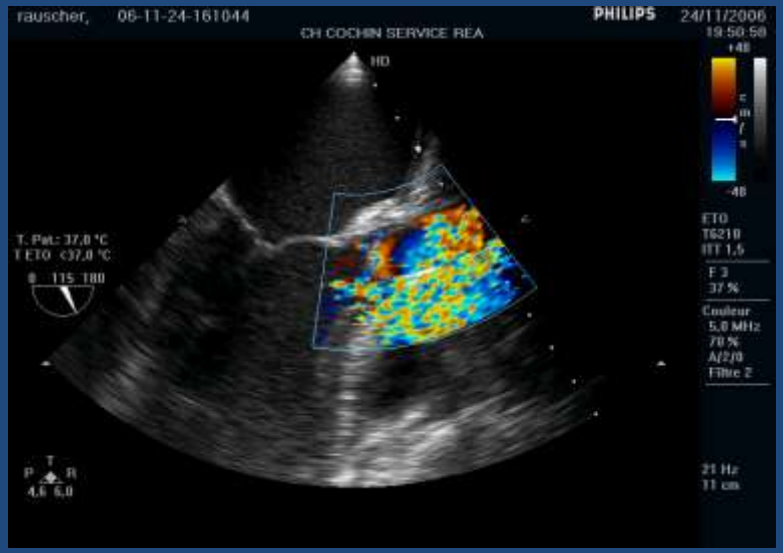




Contrôle du positionnement







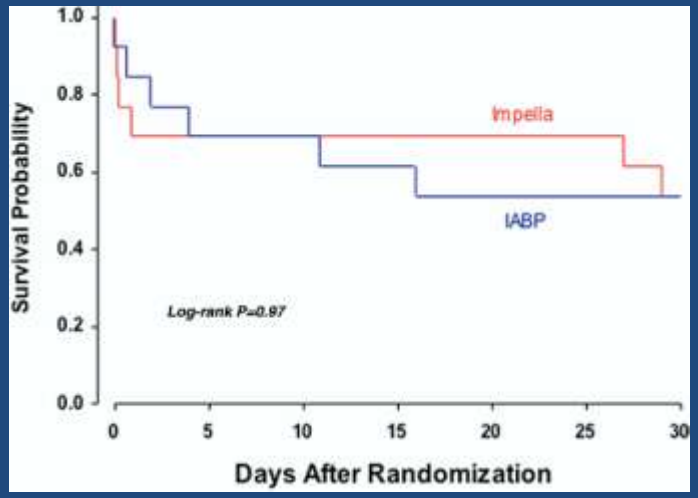
A Randomized Clinical Trial to Evaluate the Safety and Efficacy of a Percutaneous Left Ventricular Assist Device Versus Intra-Aortic Balloon Pumping for Treatment of Cardiogenic Shock Caused by Myocardial Infarction

M. Seyfarth et al. JACC 2008

- Etude prospective IMPELA LP2.5 vs IABP
- 26 patients
- Amélioration hémodynamique à 30 min
- Majoration Index cardiaque :

  Impella 0.49 ±0.46 l/min/m²

  IABP 0.11 ± 0.31 l/min/m²; p 0.02
- Mortalité à 30j: 46%

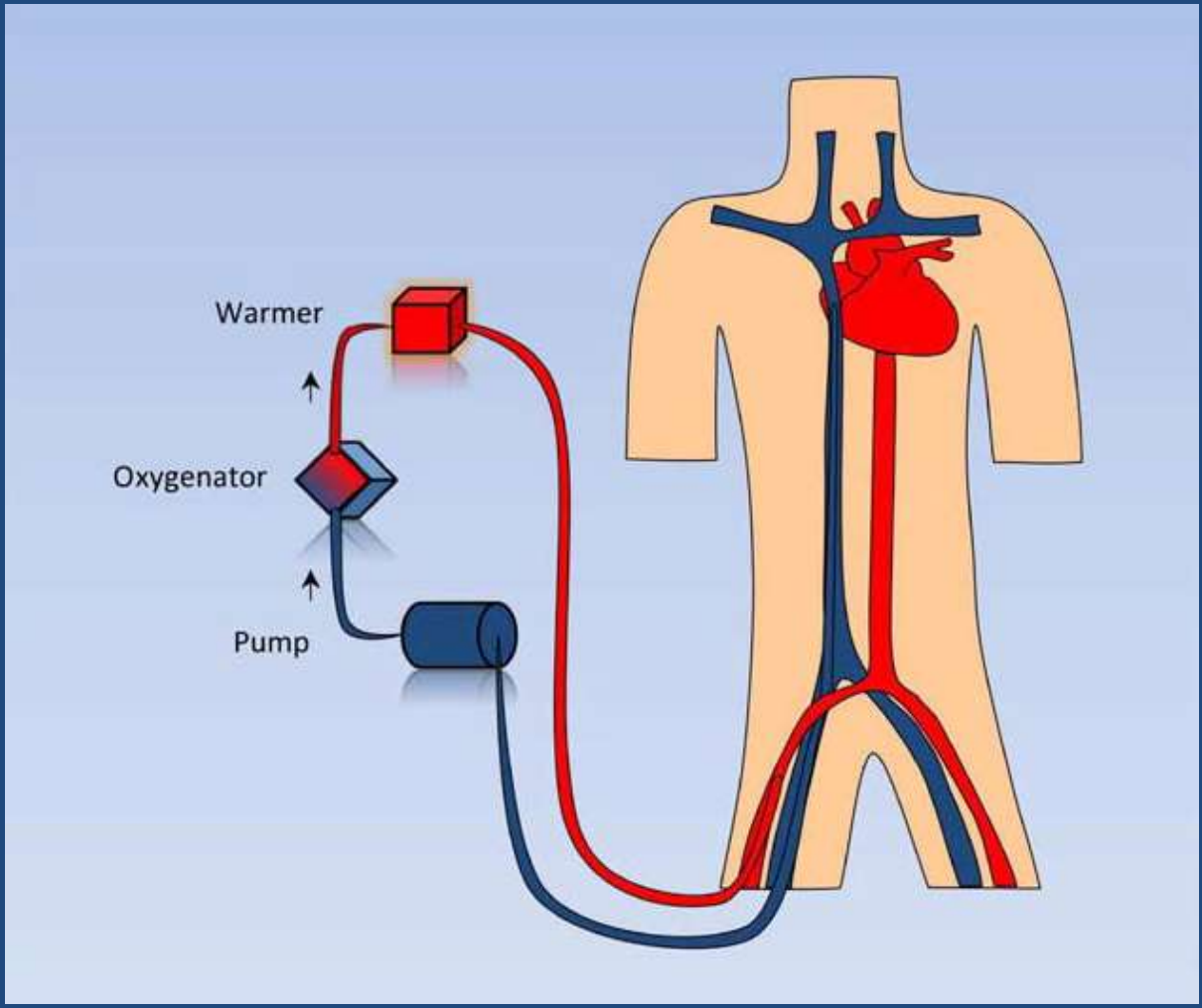


Neurological Outcomes

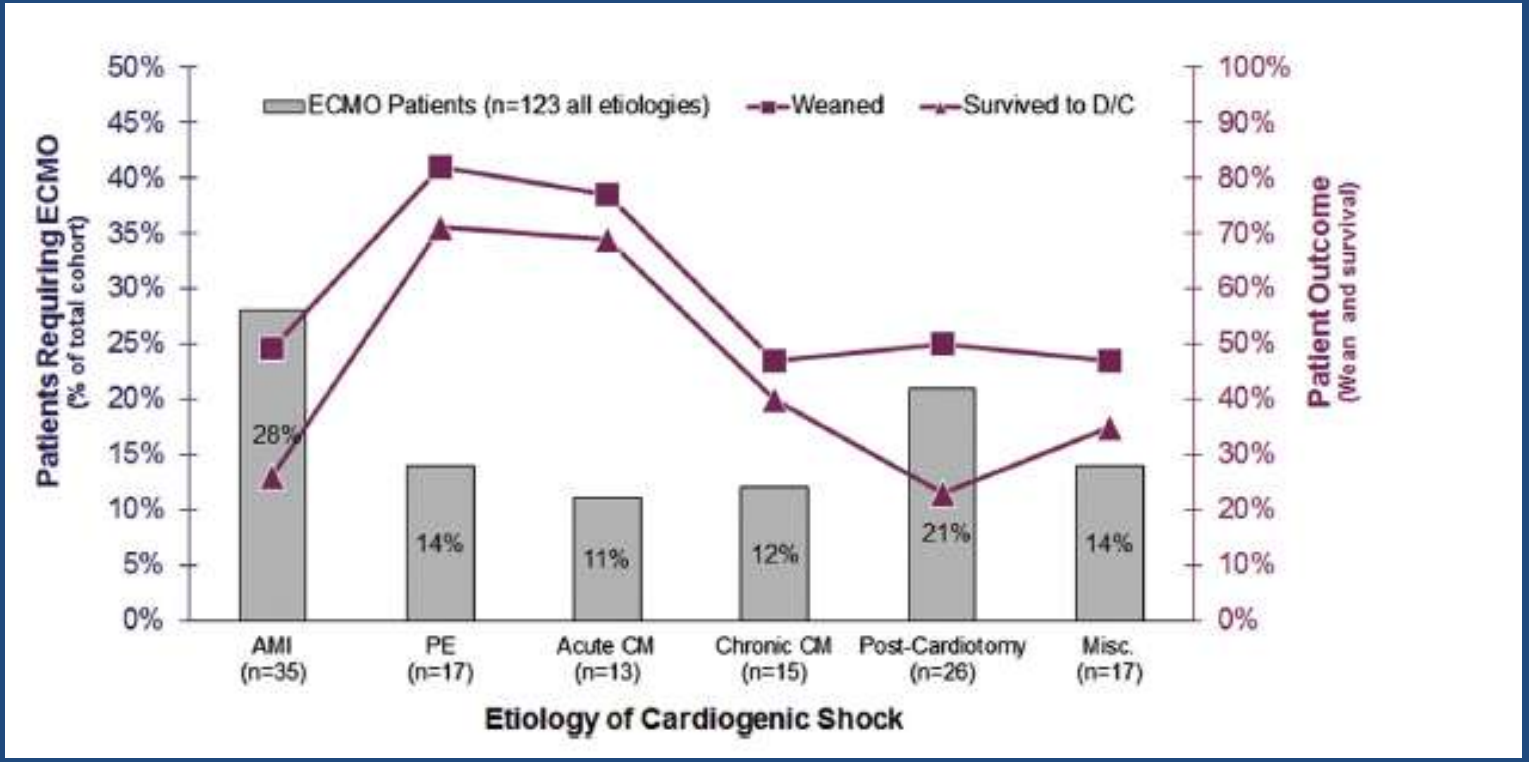
	CPC 1 on day 3 (%)	CPC 3-4 on day 3 (%)	Alive CPC 1 day 28 (%)	death from irreversible encephalopathy (%)
IABP	43	57	29,5	37,2
IMPELLA	75	25	23	8,6

Manzo-Silberman, Fichet et al. Resuscitation 2012

ECMO



Clinical Features and Outcomes in Adults With Cardiogenic Shock Supported by Extracorporeal Membrane Oxygenation



Carroll et al. Am J Cardiol 2015

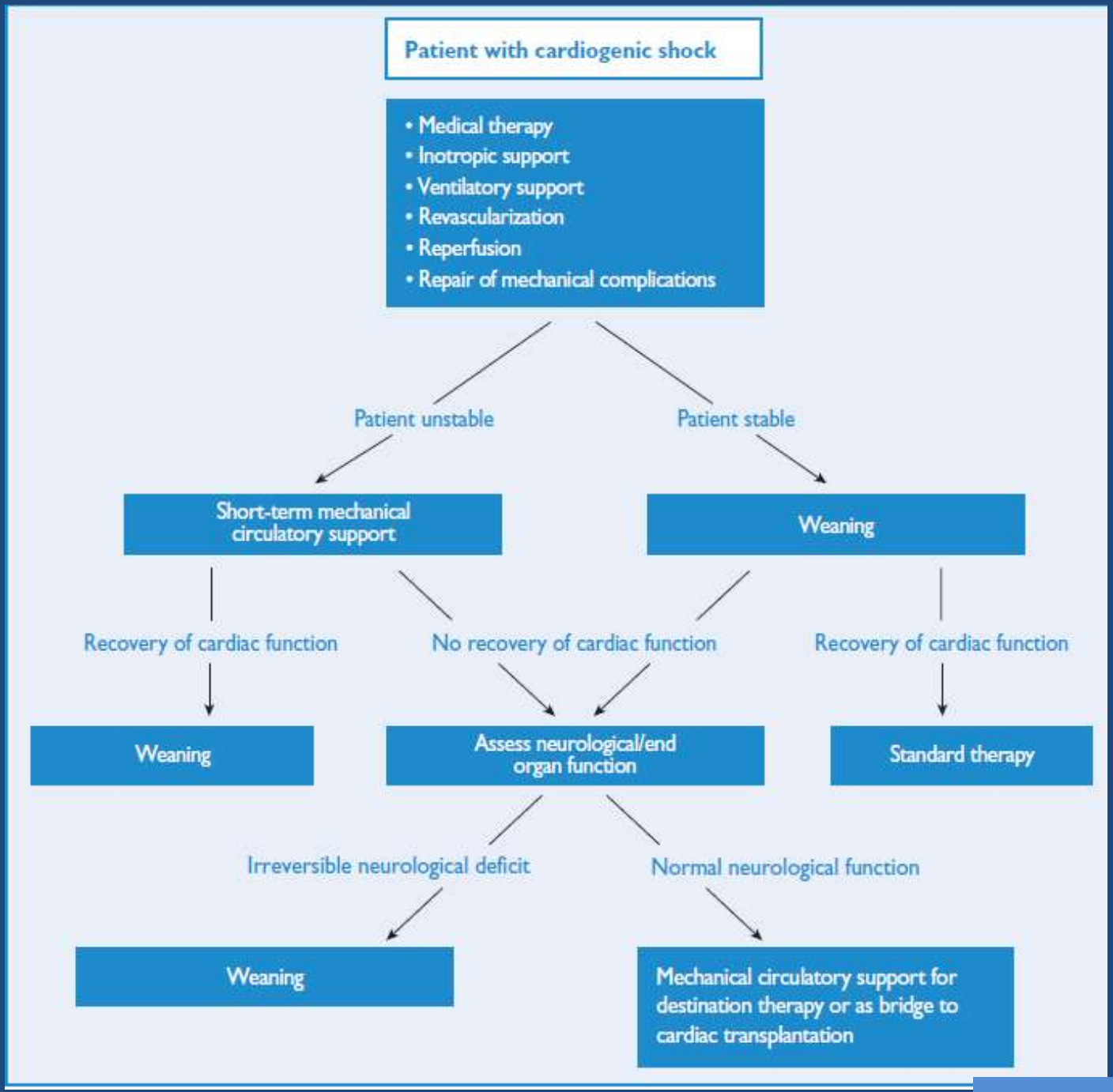
- **No time limit should be set between onset of symptoms and invasive diagnosis and revascularization in patients with cardiogenic shock, whether or not they previously received fibrinolytic treatment.**

- **In these patients, complete revascularization has been recommended, with PCI performed in all critically stenosed large epicardial coronary arteries**

European Heart Journal
doi:10.1093/eurheartj/ehs215

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

Treatment of cardiogenic shock (Killip class IV)			
Oxygen/mechanical respiratory support is indicated according to blood gasses.	I	C	-
Urgent echocardiography/Doppler must be performed to detect mechanical complications, assess systolic function and loading conditions.	I	C	-
High-risk patients must be transferred early to tertiary centres.	I	C	-
Emergency revascularization with either PCI or CABG in suitable patients must be considered.	I	B	100
Fibrinolysis should be considered if revascularization is unavailable.	IIa	C	-
Intra-aortic balloon pumping may be considered.	IIb	B	1, 98, 305
LV assist devices may be considered for circulatory support in patients in refractory shock.	IIb	C	-
Haemodynamic assessment with balloon floating catheter may be considered.	IIb	B	316
Inotropic/vasopressor agents should be considered: • Dopamine	IIa	C	-
• Dobutamine	IIa	C	-
• Norepinephrine (preferred over dopamine when blood pressure is low).	IIb	B	300, 317


Patient with cardiogenic shock
• Medical therapy
• Inotropic support
• Ventilatory support
• Revascularization
• Reperfusion
• Repair of mechanical complications
Patient unstable
Patient stable
Short-term mechanical circulatory support
Weaning
Recovery of cardiac function
No recovery of cardiac function
Recovery of cardiac function
Weaning
Assess neurological/end organ function
Standard therapy
Irreversible neurological deficit
Normal neurological function
Weaning
Mechanical circulatory support for destination therapy or as bridge to cardiac transplantation

Recommendations	Class[a]	Level[b]	Ref[c]
Emergency echocardiography is indicated to assess LV and valvular function and exclude mechanical complications.	I	C	
Emergency invasive evaluation is indicated in patients with acute heart failure or cardiogenic shock complicating ACS.	I	B	180,201, 221,331
Emergency PCI is indicated for patients with cardiogenic shock due to STEMI or NSTE-ACS if coronary anatomy is amenable.	I	B	221
Emergency CABG is recommended for patients with cardiogenic shock if the coronary anatomy is not amenable to PCI.	I	B	221
Emergency surgery for mechanical complications of acute myocardial infarction is indicated in case of haemodynamic instability.	I	C	
IABP insertion should be considered in patients with haemodynamic instability/cardiogenic shock due to mechanical complications.	IIa	C	
Patients with mechanical complication after acute myocardial infarction require immediate discussion by the Heart Team.	I	C	
Short-term mechanical circulatory support in ACS patients with cardiogenic shock may be considered.	IIb	C	
Percutaneous repair of VSD may be considered after discussion by the Heart Team.	IIb	C	
Routine use of IABP in patients with cardiogenic shock is not recommended.	III	A	332,333

Eur Heart J 2014

The NEW ENGLAND JOURNAL of MEDICINE

Circulatory Shock

Jean-Louis Vincent, M.D., Ph.D., and Daniel De Backer, M.D., Ph.D.

CONCLUSIONS

Diminuer l'incidence des complications

- **Identifier les patients à risque, stratifier le risque**
- **Surveillance étroite**
- **Intervention précoce**
- **Augmenter les taux de reperfusion précoce**

Diminuer leur mortalité

- **Reconnaître précocement les signes précurseurs du choc dans l'IDM**
- **Prise en charge accélérée et agressive dès la phase initiale du choc**

EN PRATIQUE

- IDM étendu
- Incomplètement reperfusé
  - Low/No reflow
  - TIMI <3
- Tachycarde
- TAS limite
- PTDVG > 20 mmHg

→ PENSER ASSISTANCE

Merci de votre attention!