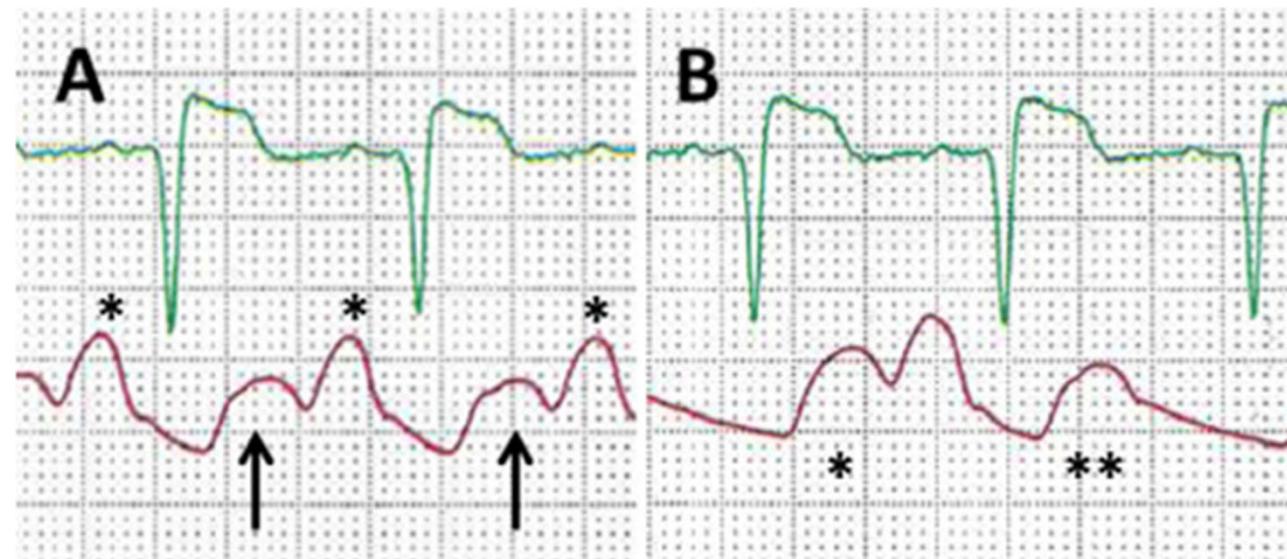


Contre-pulsion intra-aortique (CPIA): ses principes, ses indications

Pr. Edouard Gerbaud
Soins Intensifs Cardiologiques
Plateau de Cardiologie Interventionnelle
Hôpital Cardiologique du Haut Lévêque
CHU de Bordeaux

Définition

- Dispositif temporaire de soutien circulatoire mis en place de manière invasive par voie fémorale, via un désilet 8F, dans l'aorte descendante sous contrôle scopique.
- 1^{ère} application clinique en 1968 pour un diagnostic de choc cardiogénique (Dr Kantrowitz)



Histoire

History:

1962 Animal studies

Moulopoulos et al. Am Heart J 1962;63:669-675

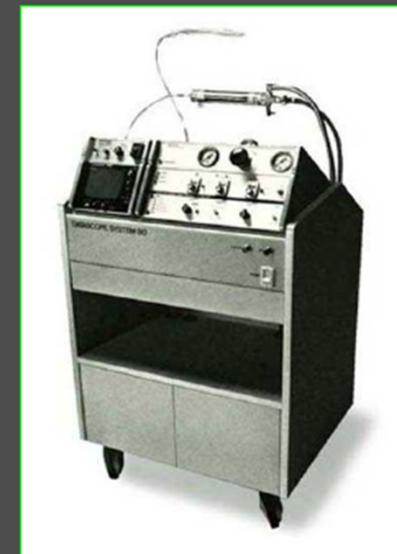


1968 First clinical description in shock

Kantrowitz et al. JAMA 1968;203:135-140

1973 Hemodynamic effects in shock, Mortality unchanged

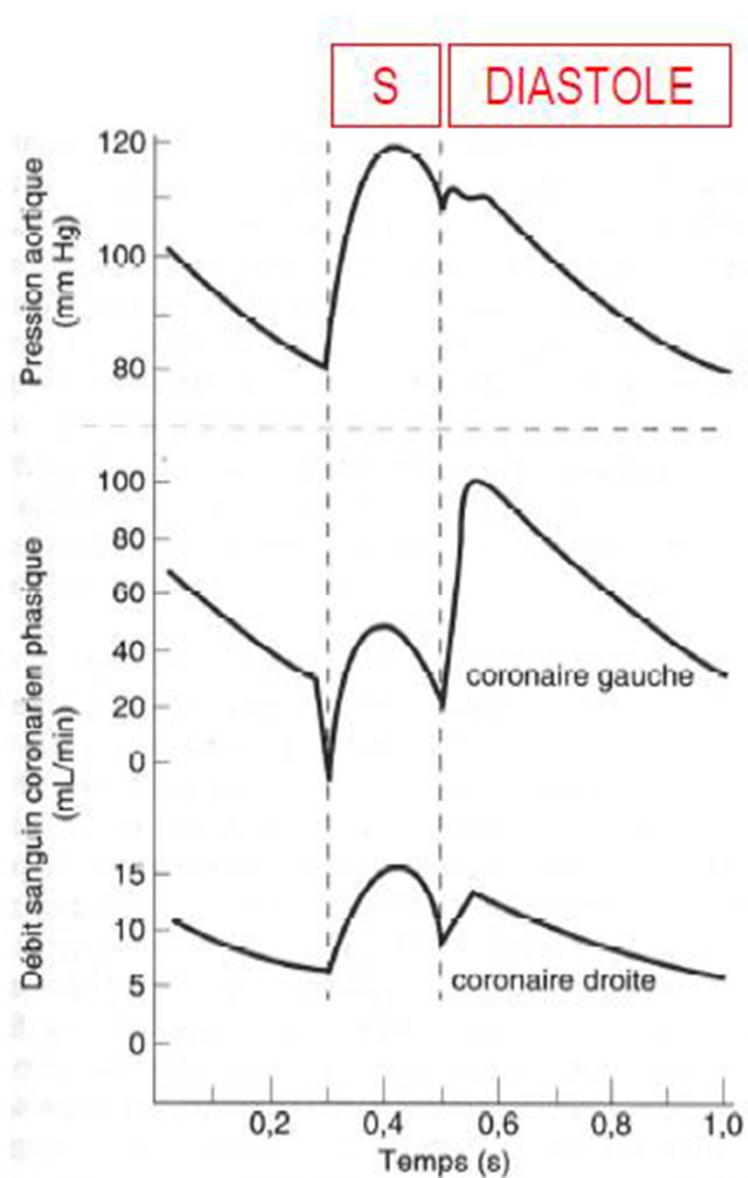
Scheidt et al. NEJM 1973;288:979-984



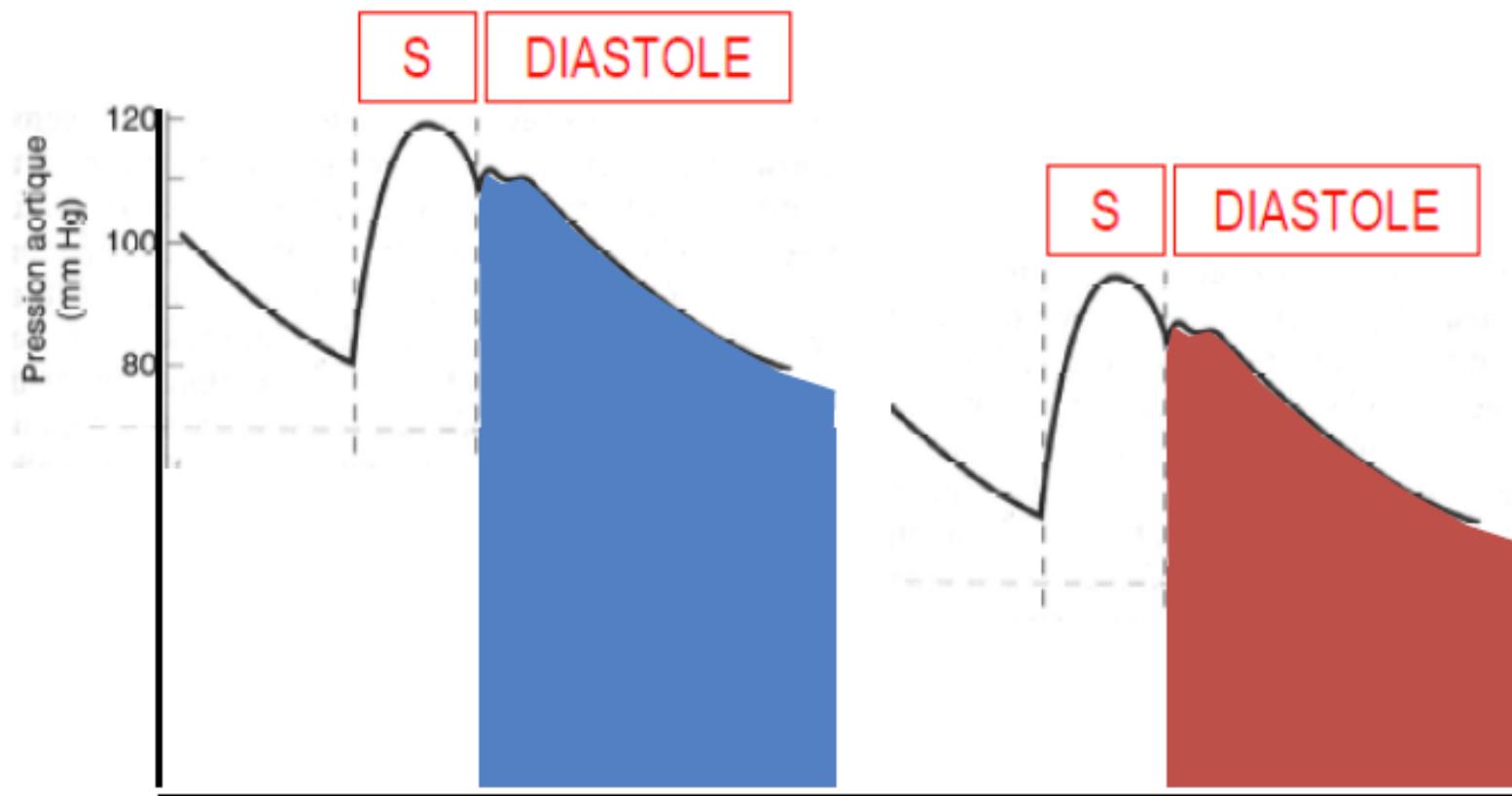
> 40 years > 1 Million patients treated, low complication rate, Benchmark registry

Ferguson et al. JACC 2001;38:1456-1462

- La perfusion d'une coronaire se fait en diastole
et surtout pour l'artère coronaire gauche

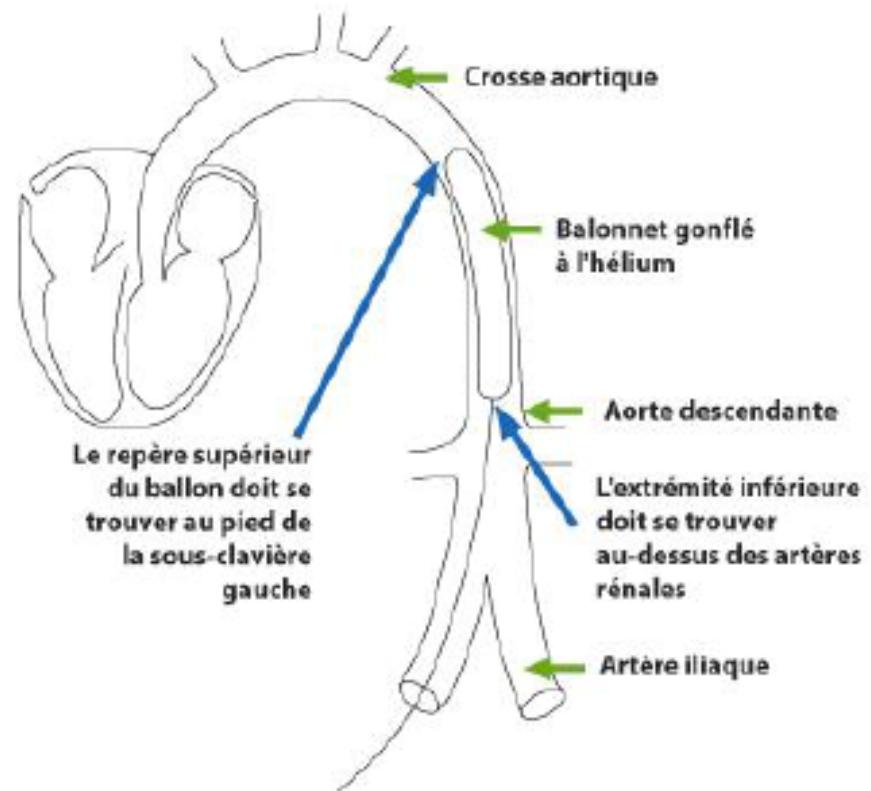


Choc cardiogénique

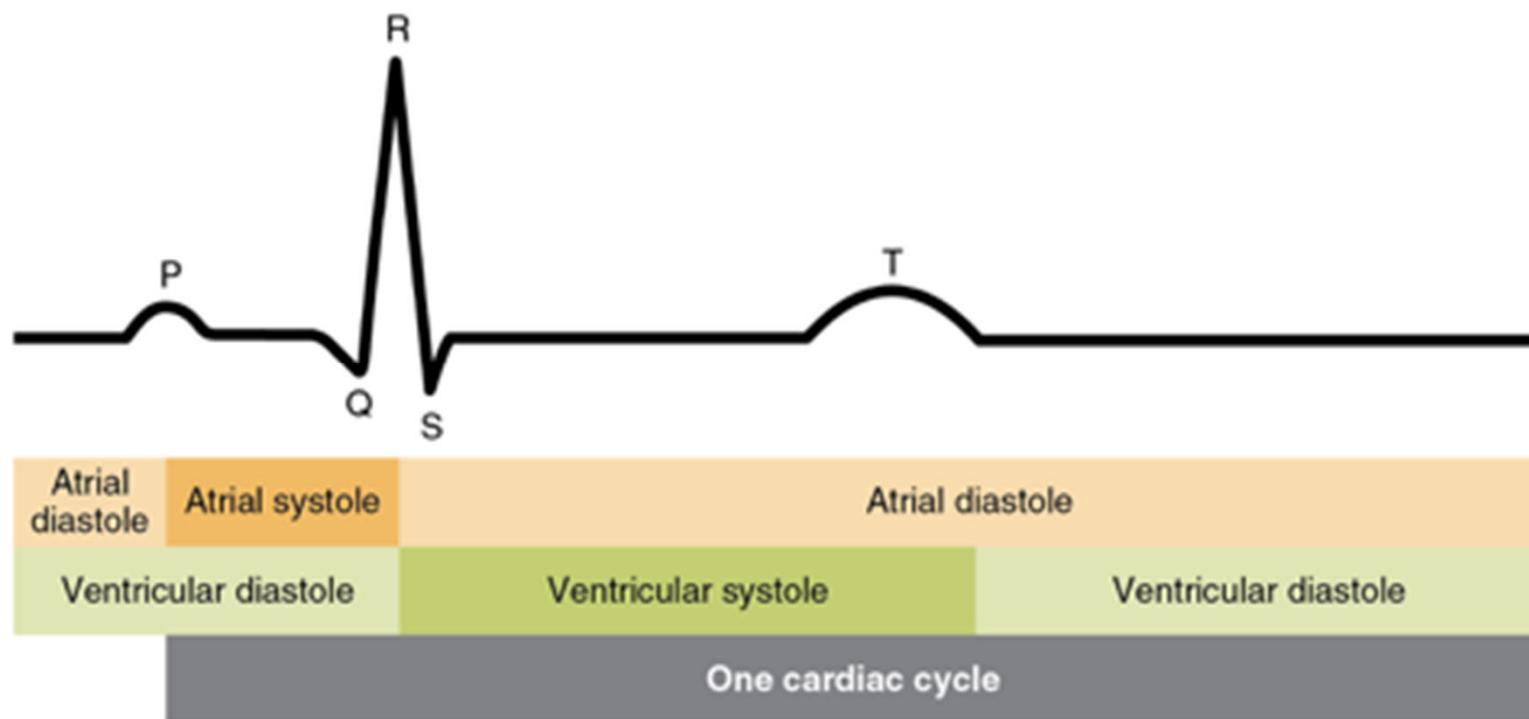


Fonctionnement

- Ballon en Silicone de 30 ou 40 cc (++)
- Hélium
- Synchronisation sur l'ECG pour
 - Se gonfler en début de diastole
 - Se dégonfler (de manière active) avant le début de la systole



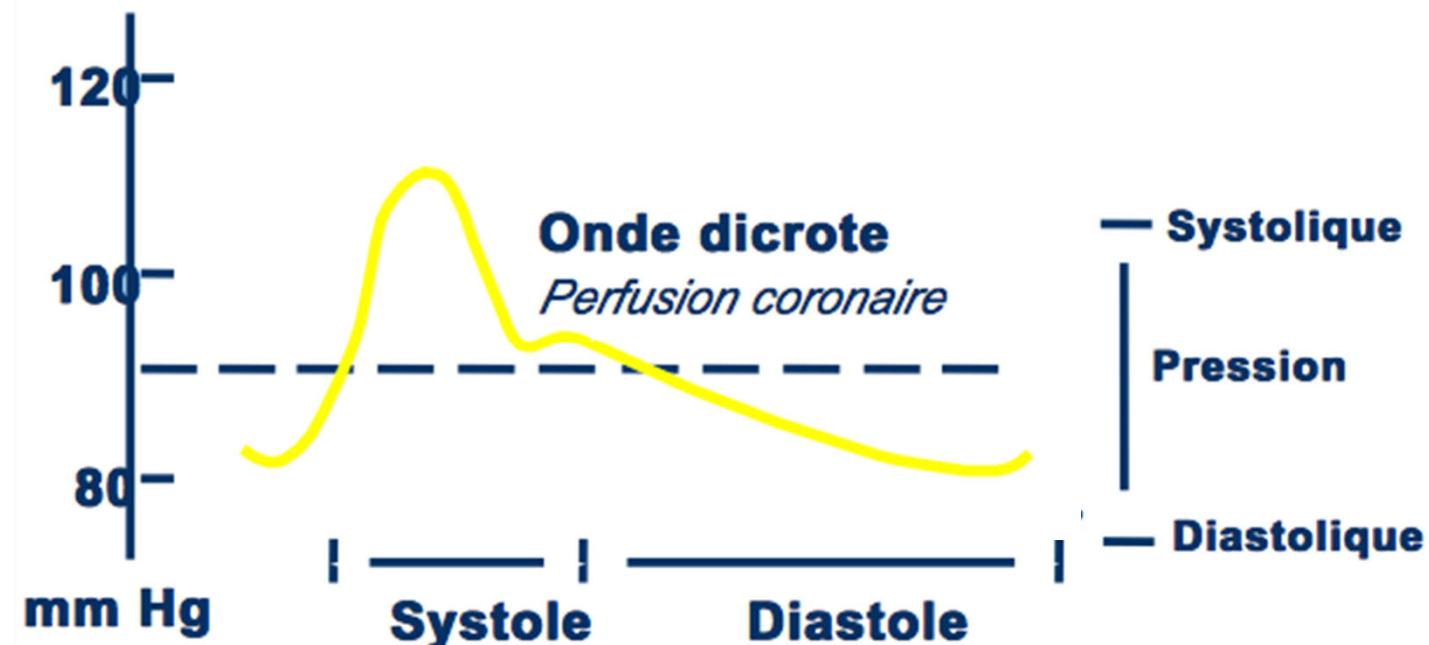
Le cycle cardiaque



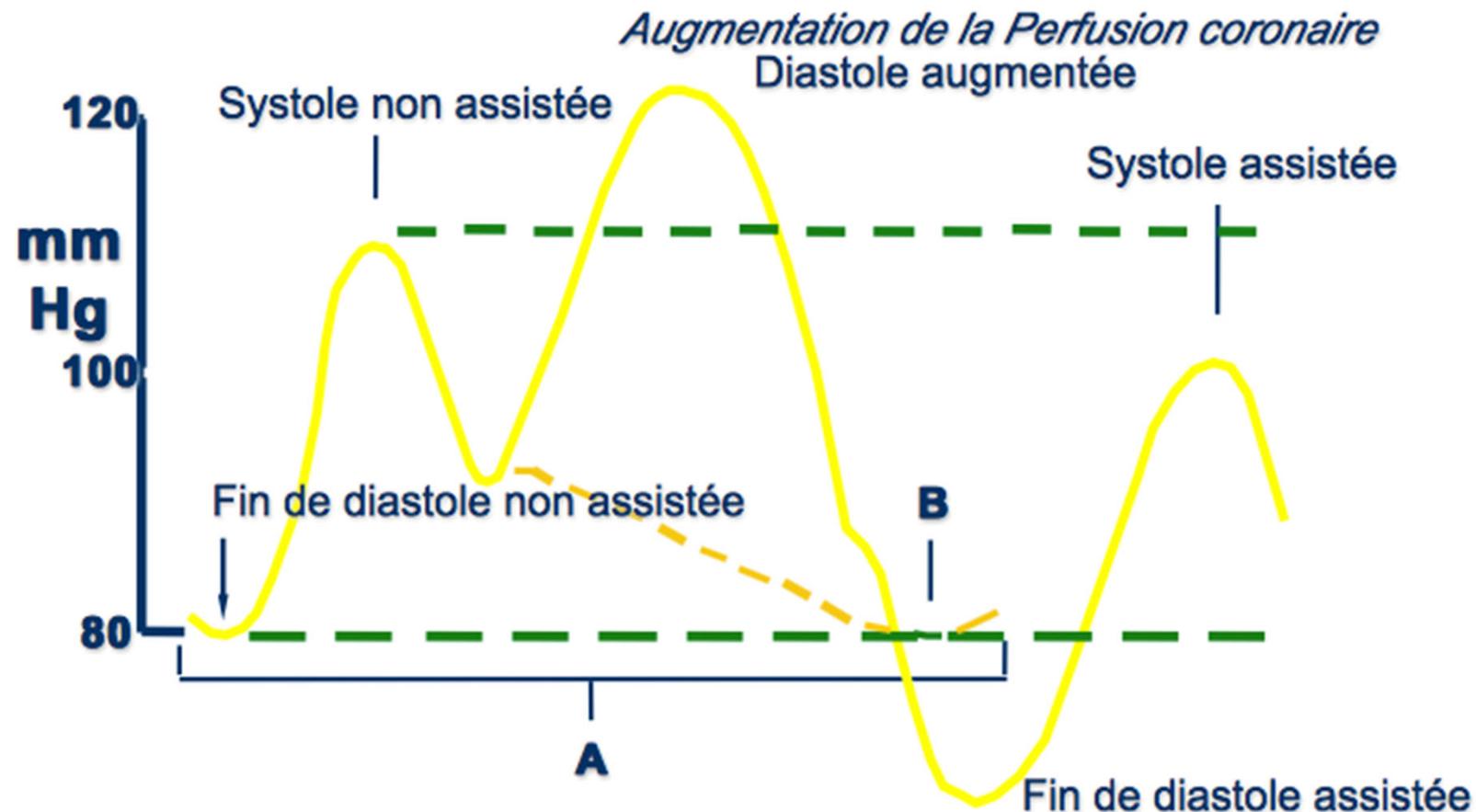
- L'impulsion électrique précède l'activité mécanique

Fonctionnement

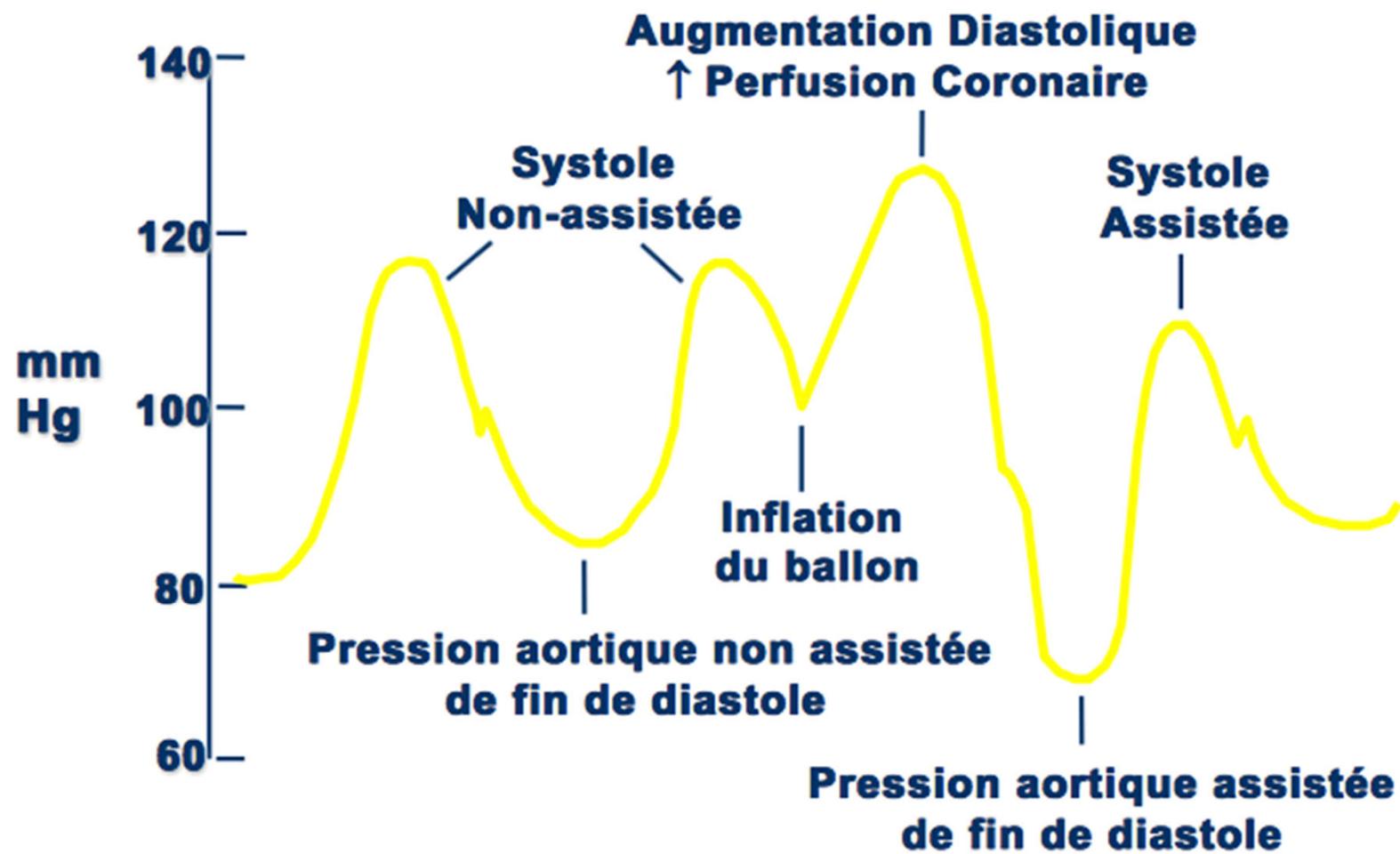
- Pression artérielle moyenne
- Indicateur de la perfusion périphérique
- Améliorée par la CPIA



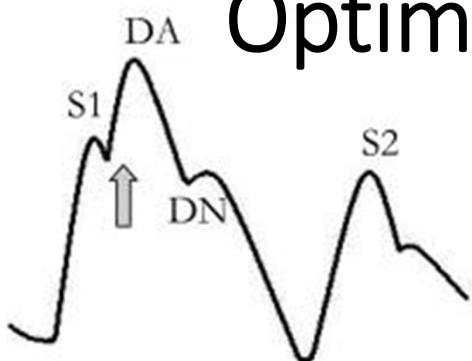
Fonctionnement



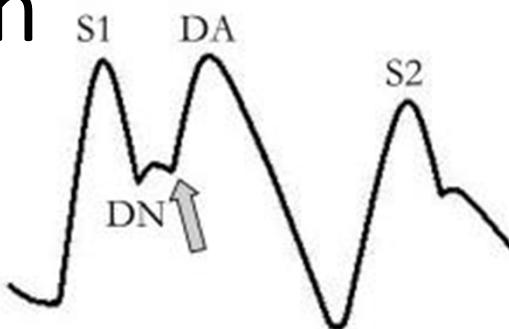
Fonctionnement



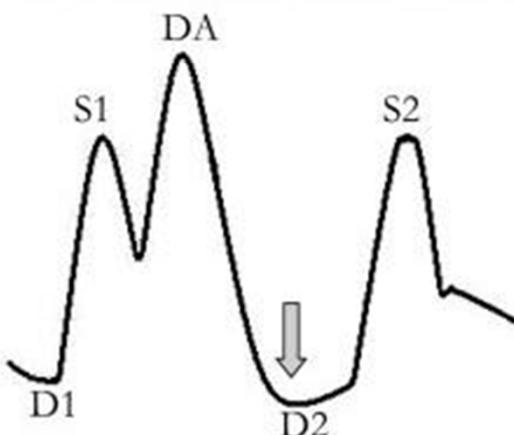
Optimisation



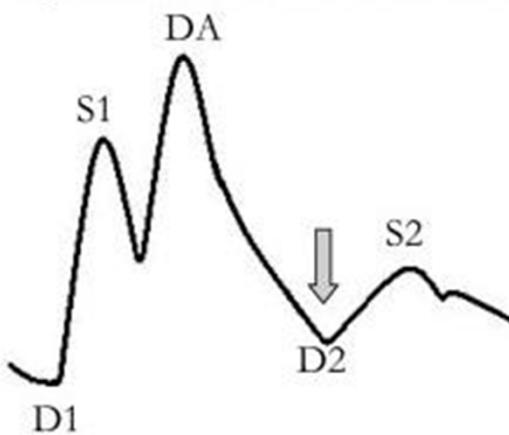
Early Inflation— rapid rise in diastolic pressure with dicrotic notch after IABP deflation; causes increased afterload.



Late inflation— prolonged dip before a decreased diastolic augmentation reduces effectiveness.



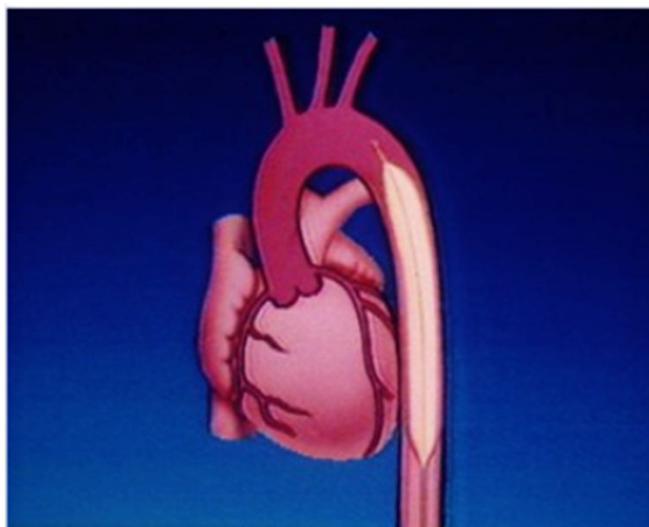
Early deflation— prolonged dip of assisted end-diastolic pressure and no decrease in assisted systolic pressure; no afterload reduction.



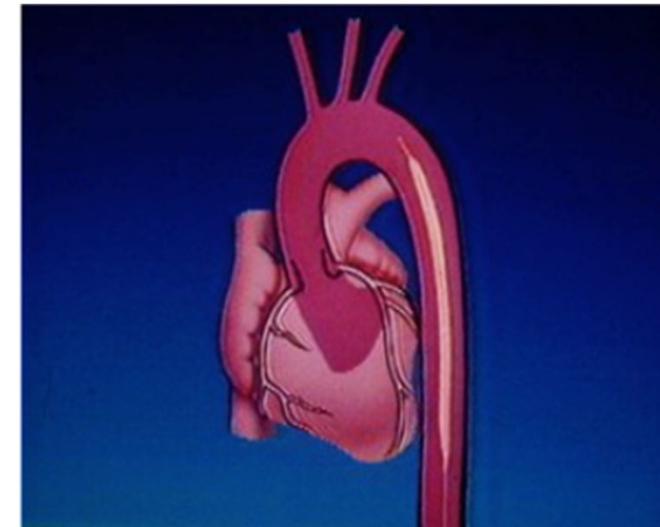
Late deflation— the assisted end-diastolic pressure is higher than the unassisted end-diastolic pressure; causes increased afterload.

Quel Gain?

- Augmentation de la pression aortique diastolique
- Amélioration du débit de perfusion coronaire
- Augmentation de l'apport en O_2
- Diminution de la pression artérielle systolique
- Diminution de la résistance contre lequel le cœur travaille
- Diminution de la consommation en O_2



GONFLAGE du ballon



DEGONFLAGE du ballon

Quel Gain?

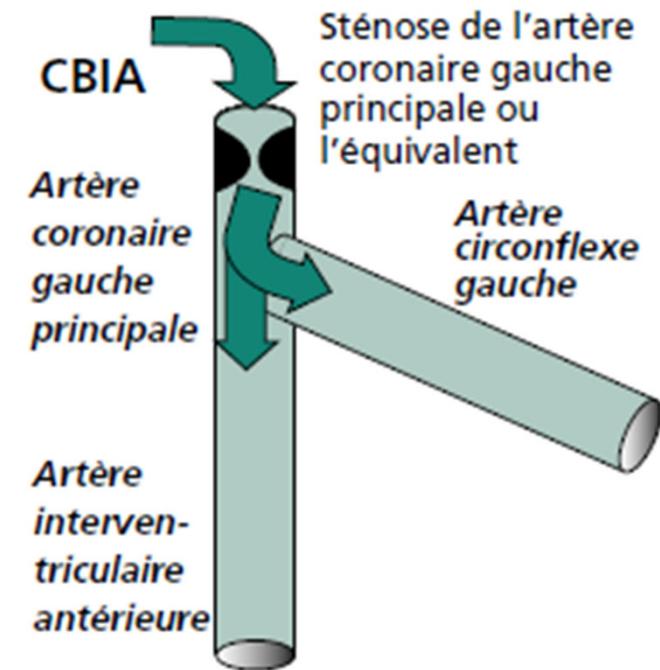
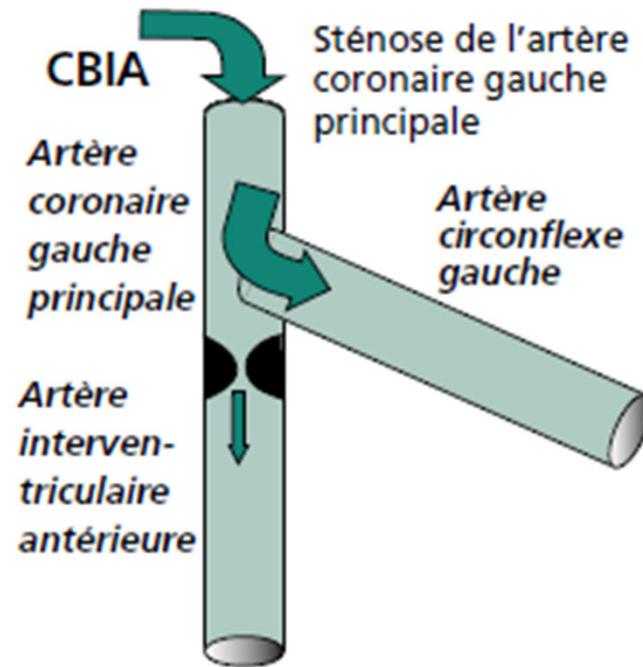
- Cela dépend
 - de la fréquence cardiaque
 - du volume du ballon



- de la sténose coronaire

Le problème de la sténose coronaire

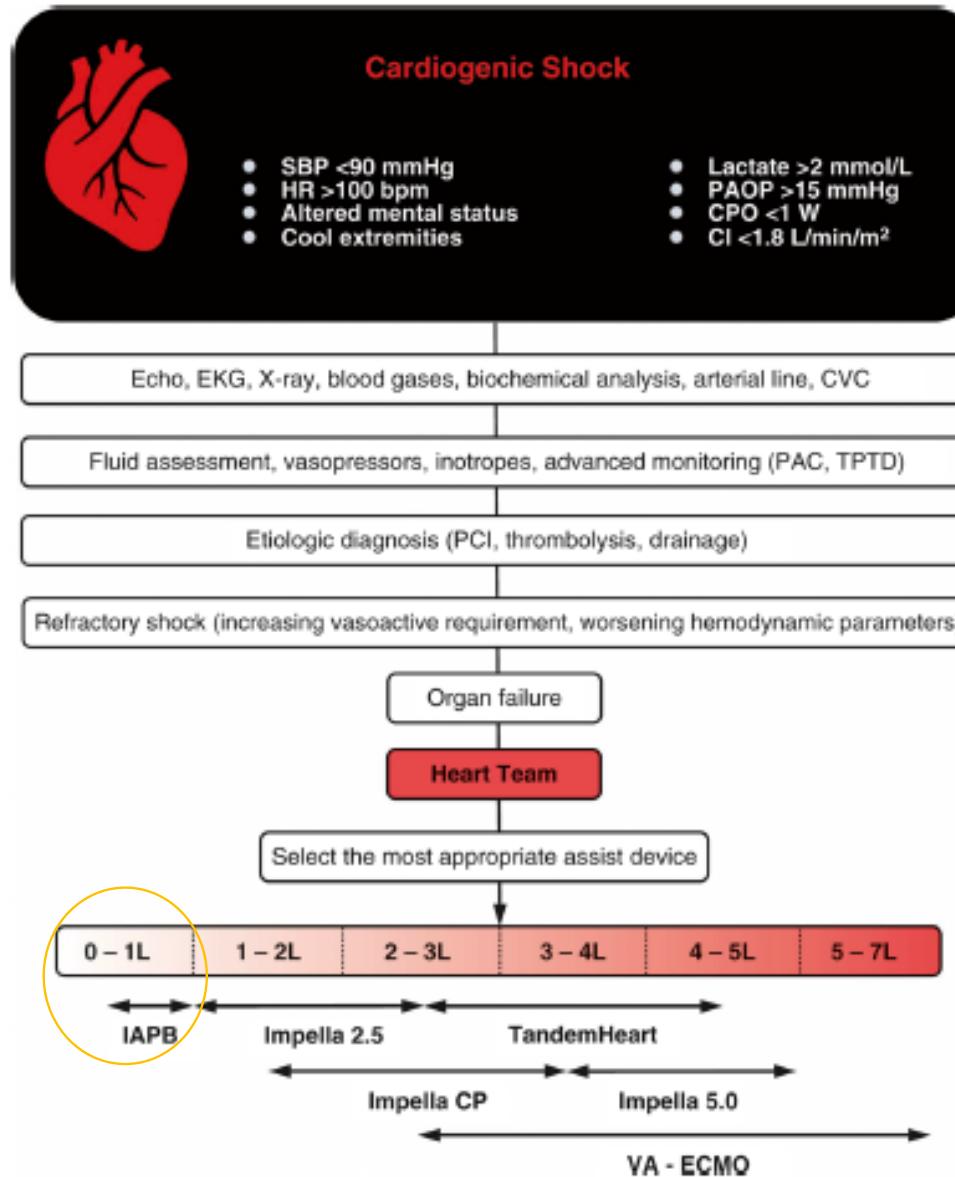
- Il faut l'enlever



- par angioplastie ou par pontage aorto-coronarien

Quel Gain?

Augmentation du
débit cardiaque
d'environ 0.5 L/min



Les indications

- Choc cardiogénique dans un contexte de syndrome coronarien aigu
 - lésion isolée de l'IVA proximale (atteinte mono-tronculaire)
 - « Pré-choc »: patient « limite / borderline » sur le plan hémodynamique avec faible dose concomitante d'amines vasopressives
- Angioplastie protégée / « High risk PCI »
- Infarctus du ventricule droit

Les indications

- Réduire la post-charge du ventricule gauche
- Augmenter le volume d'éjection systolique effectif en diminuant le shunt G>D ou en diminuant le volume régurgitant mitral
- Pré-chirurgie cardiaque
 - pontages aorto-coronariens (tronc commun)
 - insuffisance mitrale
 - communication interventriculaire
- Période « post-opératoire » de chirurgie cardiaque
- Décharge ECMO



ELSEVIER

Archives of Cardiovascular
Diseases

Volume 112, Issue 12, December 2019, Pages 792-798



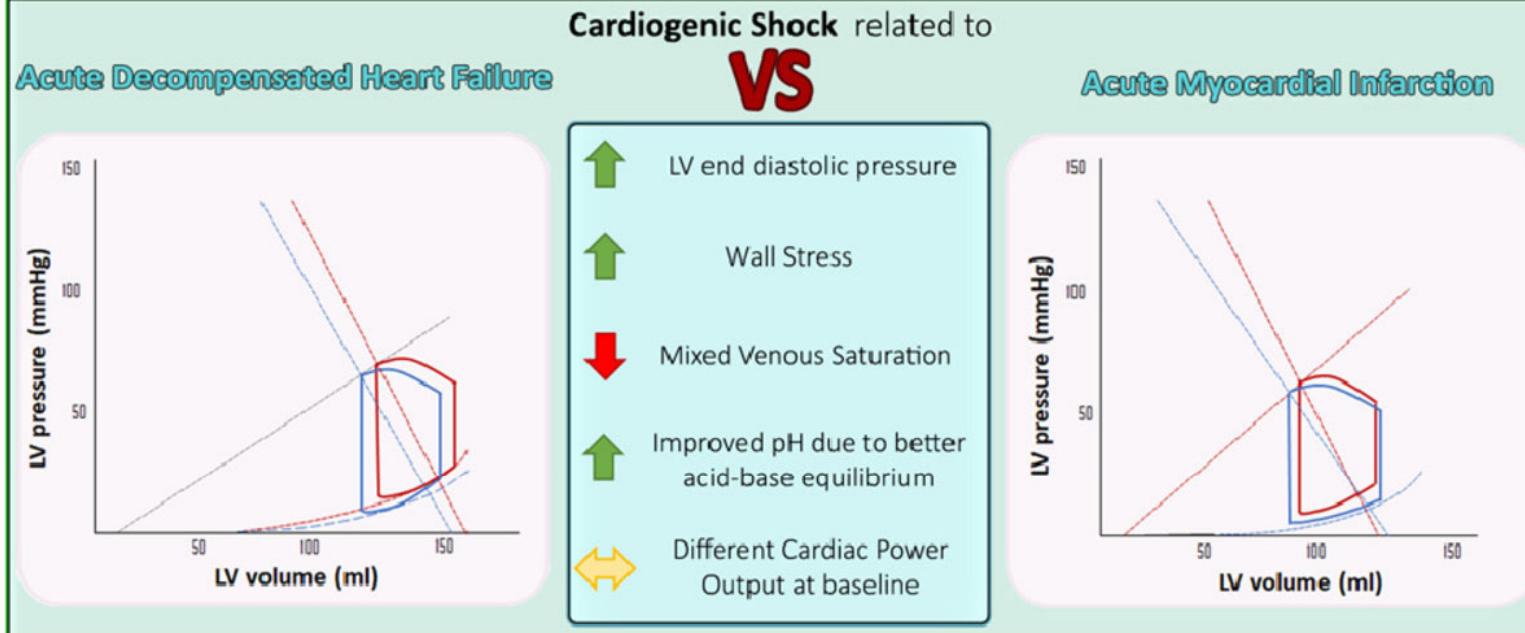
Review

Is there still a role for the intra-aortic balloon pump in the management of cardiogenic shock following acute coronary syndrome?

Le ballon de contre pulsion intra-aortique a-t-il encore un intérêt dans la prise en charge du choc cardiogénique ?

Guillaume Leurent ^a✉, Vincent Auffret ^a, Camille Pichard ^a, Marc Laine ^b, Laurent Bonello ^b

Leurent G et al. Arch Cardiovasc Dis 2019; 112:792-798

Intra-Aortic balloon pump for acute decompensated heart failure complicated by cardiogenic shock**IABP****Hemodynamic Benefits through:**

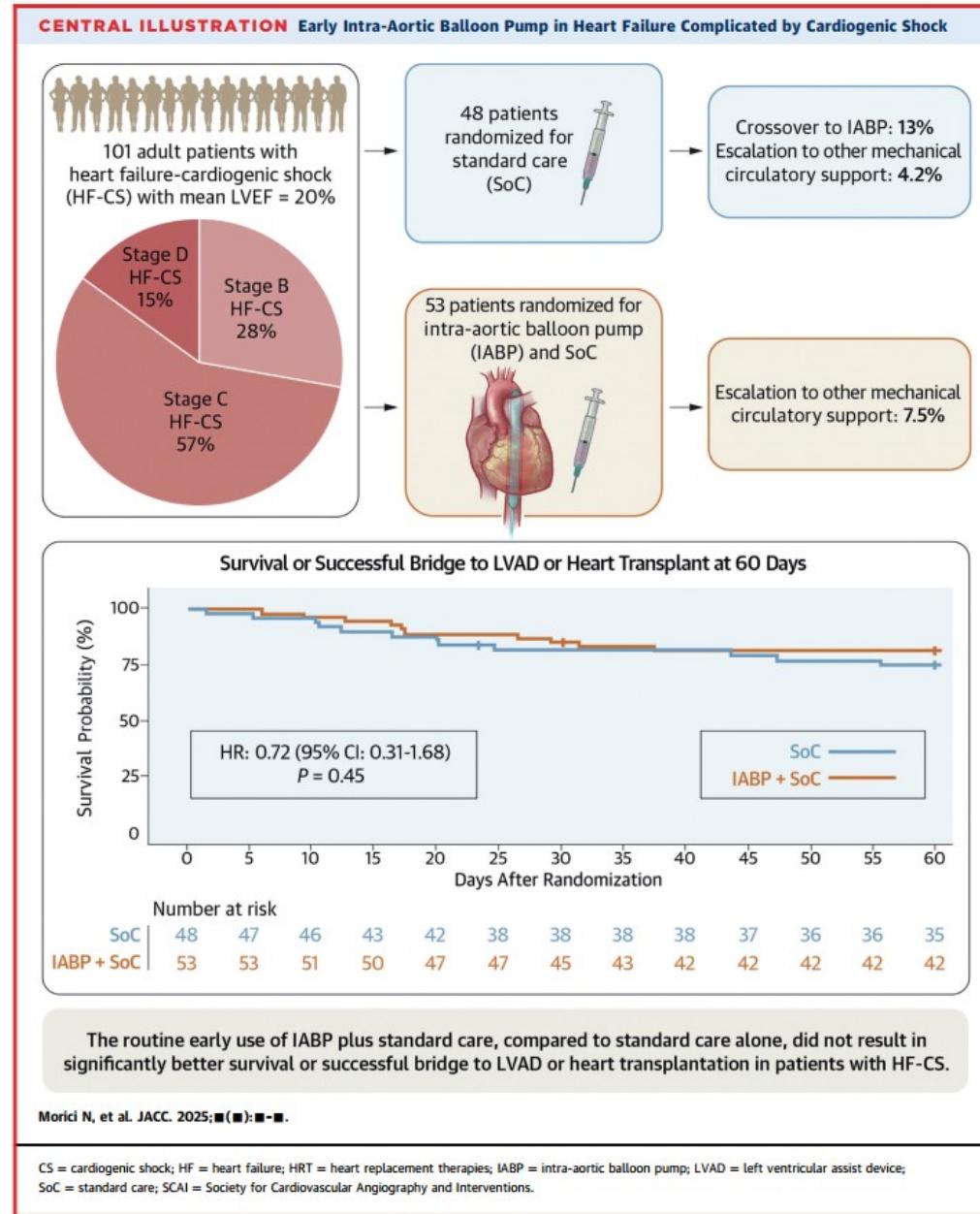
- Increasing diastolic aortocoronary pressure (*diastolic pressure time index, DTI*)
- Reducing myocardial oxygen demand (*tension time index, TTI*)
- Increasing Buckbee index (DTI/TTI)
- Increasing Cardiac Output
- Improving ventriculo-arterial coupling
- Improving Right Ventricular function
- Direct action on Mitral valve anatomy and physiology

Hemodynamic Effects hampered by tachyarrhythmias -> optimize triggering (autopilot mode recommended)

Morici N et al. J Cardiac Failure 2022; 28:1202-1216

Brown MA et al. J Am Heart Assoc. 2021 Aug 3; 10(15): e019376.

Altshock-2 trial was stopped because of futility



Les contre-indications

- Insuffisance aortique
- Syndrome douloureux aortique, dissection, hématome de paroi, anévrysme aortique...
- Accès artériel membre inférieur insuffisant
- « Relative »: Les tachycardies

Les recommandations dans le STEMI

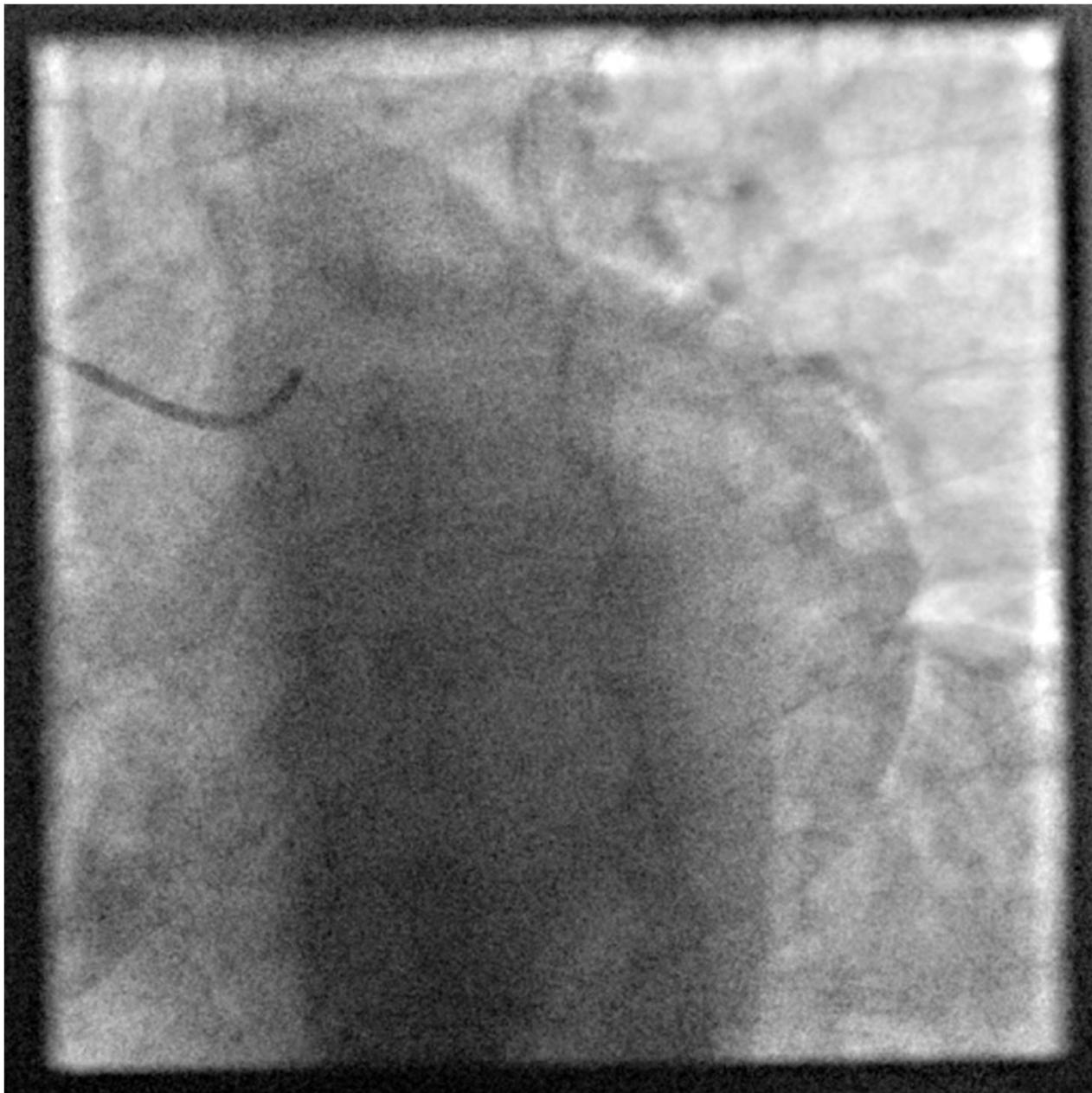
Intra-aortic balloon pumping should be considered in patients with haemodynamic instability cardiogenic shock due to mechanical complications.	IIa	C
Short-term mechanical support ^c may be considered in patients in refractory shock.	IIb	C
Routine intra-aortic balloon pumping is not indicated. ^{177,437}	III	B

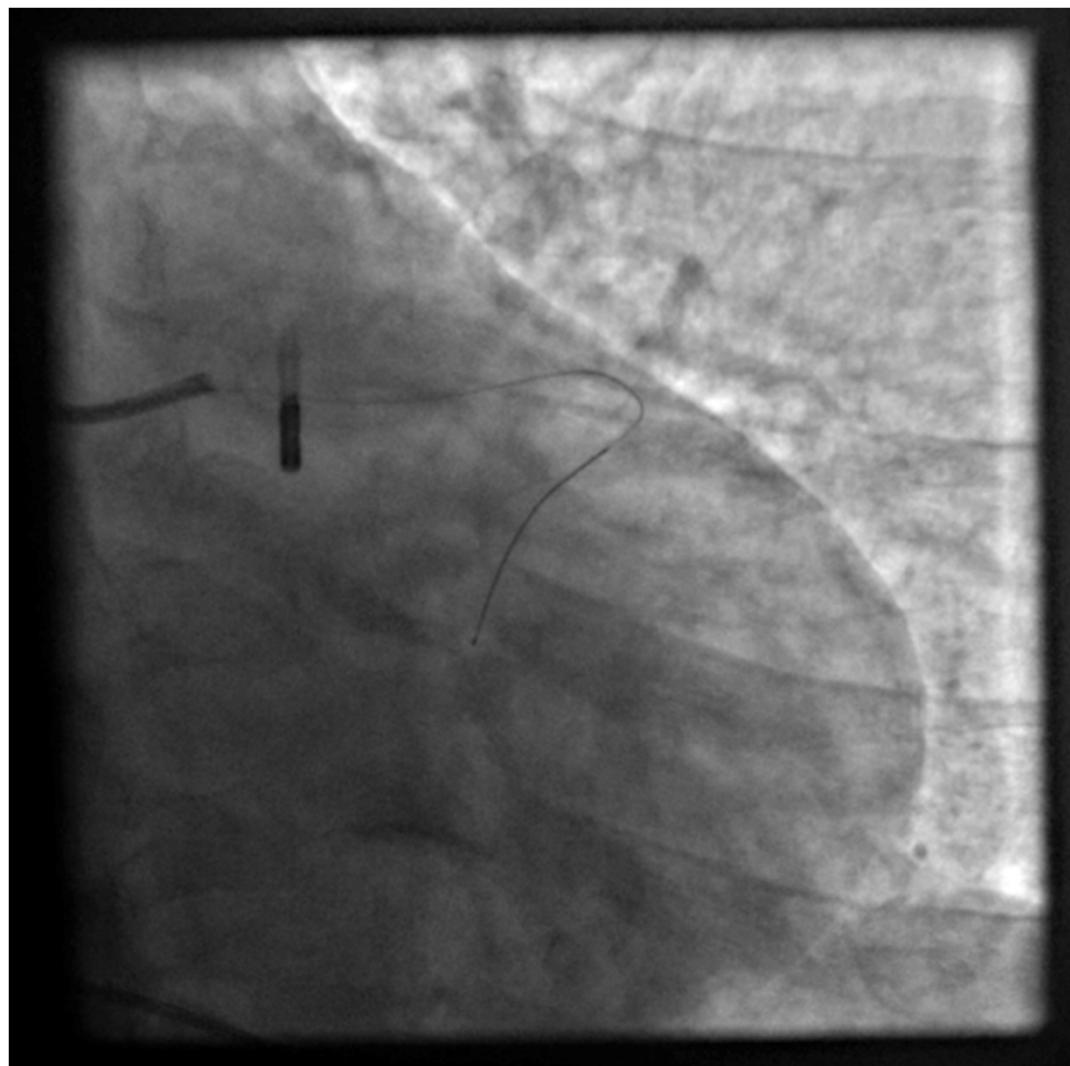
^c Percutaneous cardiac support devices, ECLS, and ECMO

Images de BPCIA

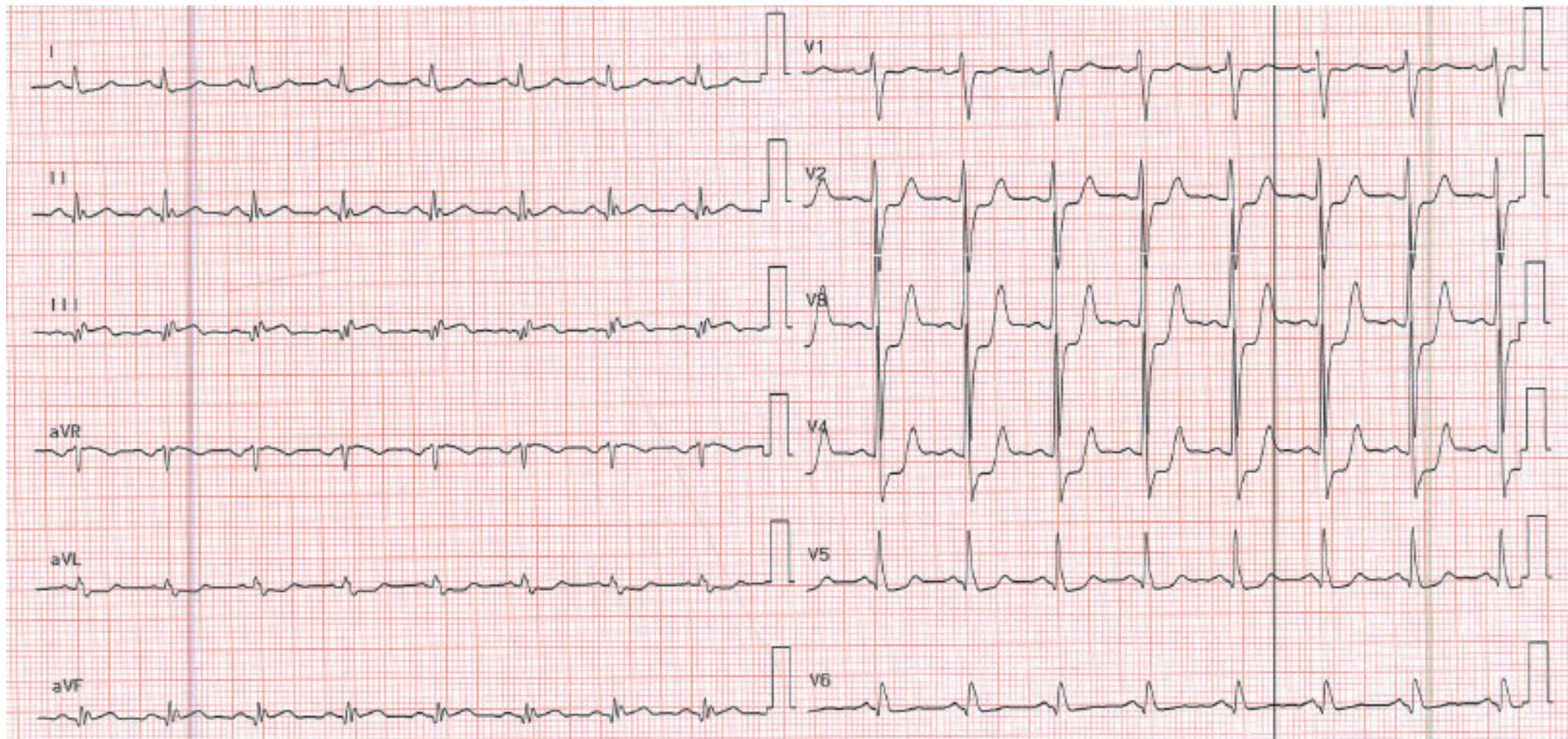


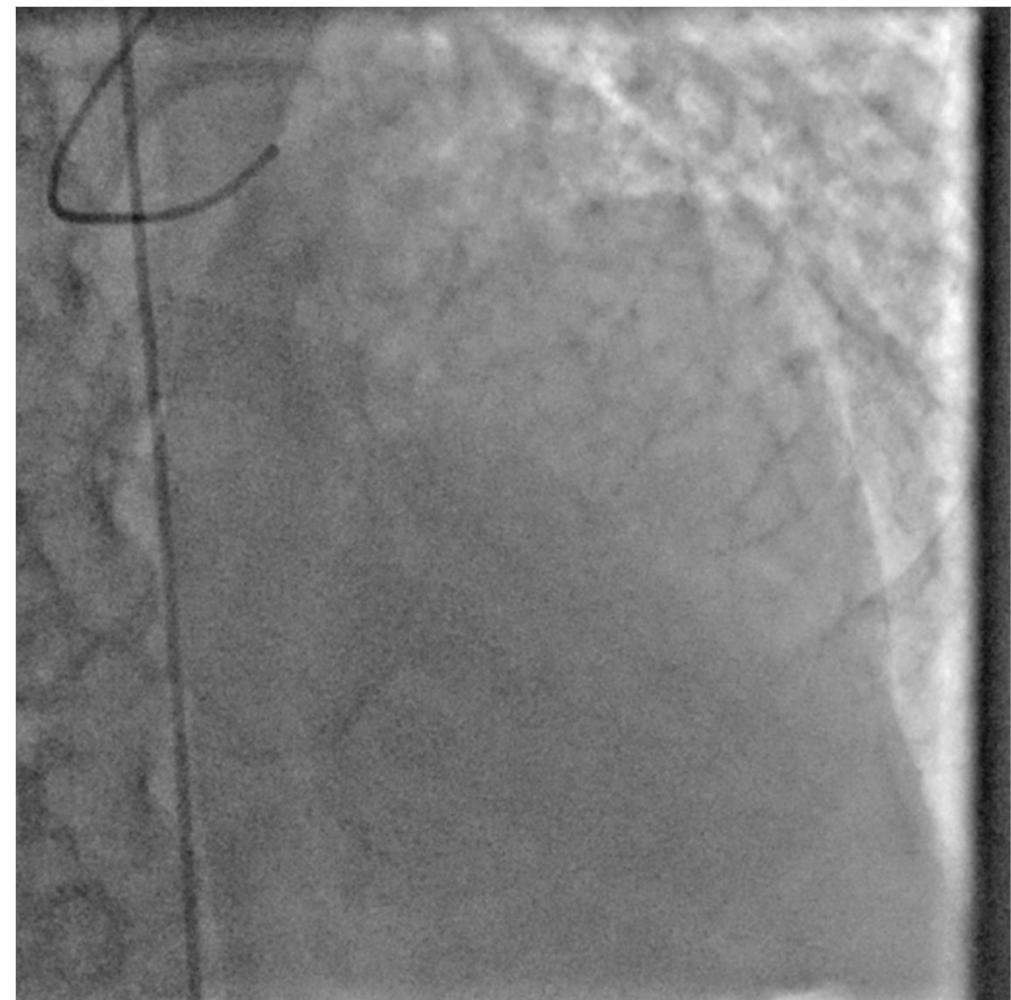
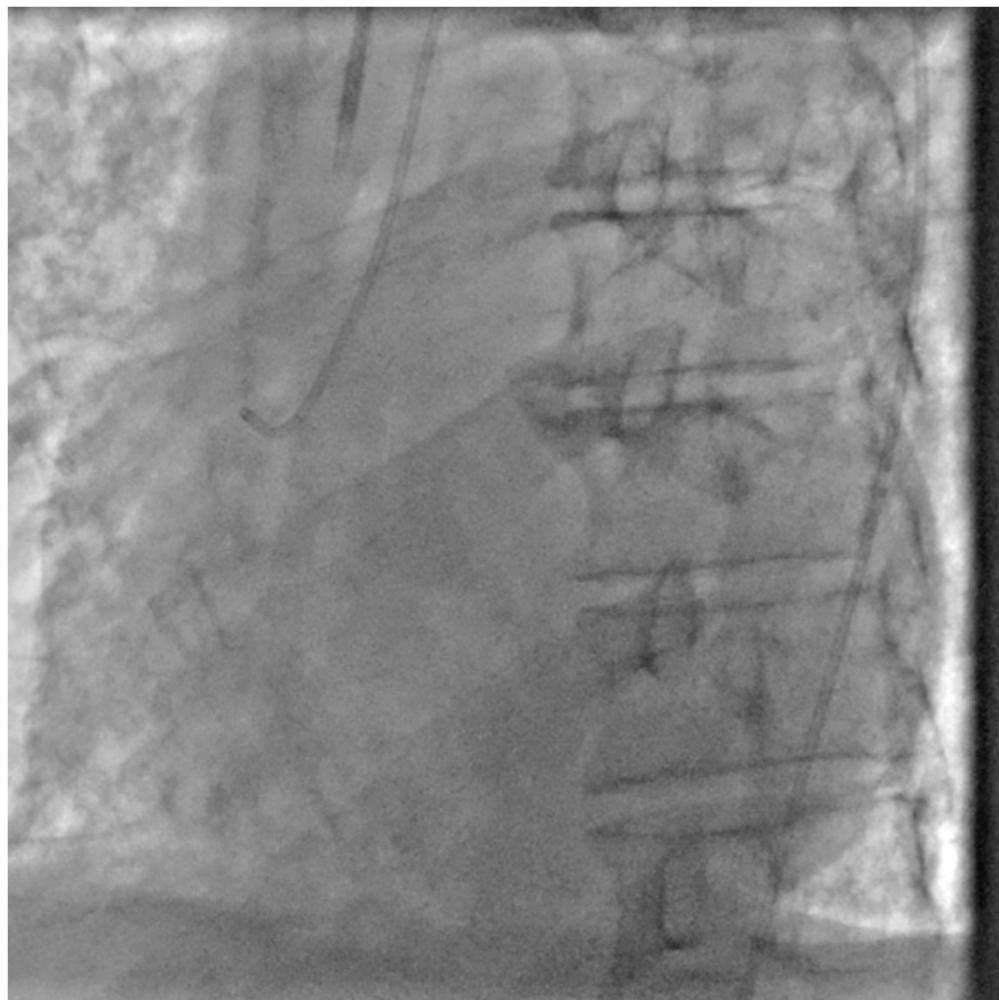
Héparinothérapie IVSE durant l'utilisation avec un objectif d'héparinémie / Activité anti Xa (HNF): 0,3-0,4 UI/mL

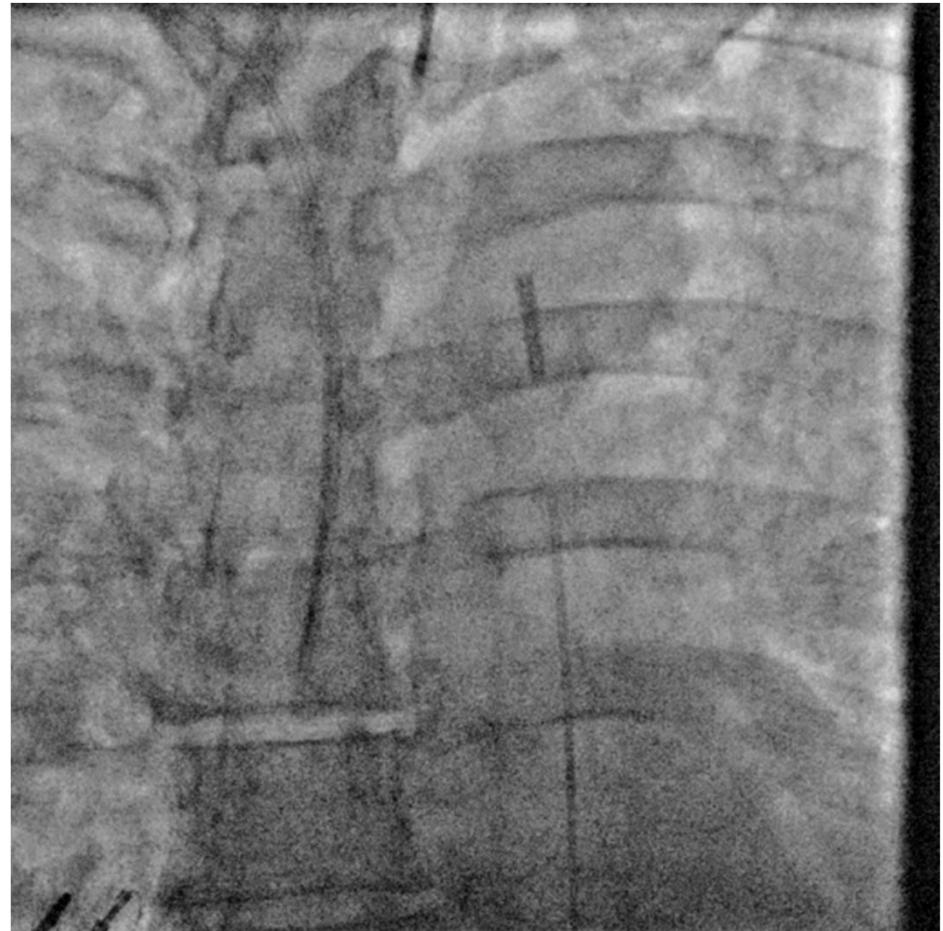
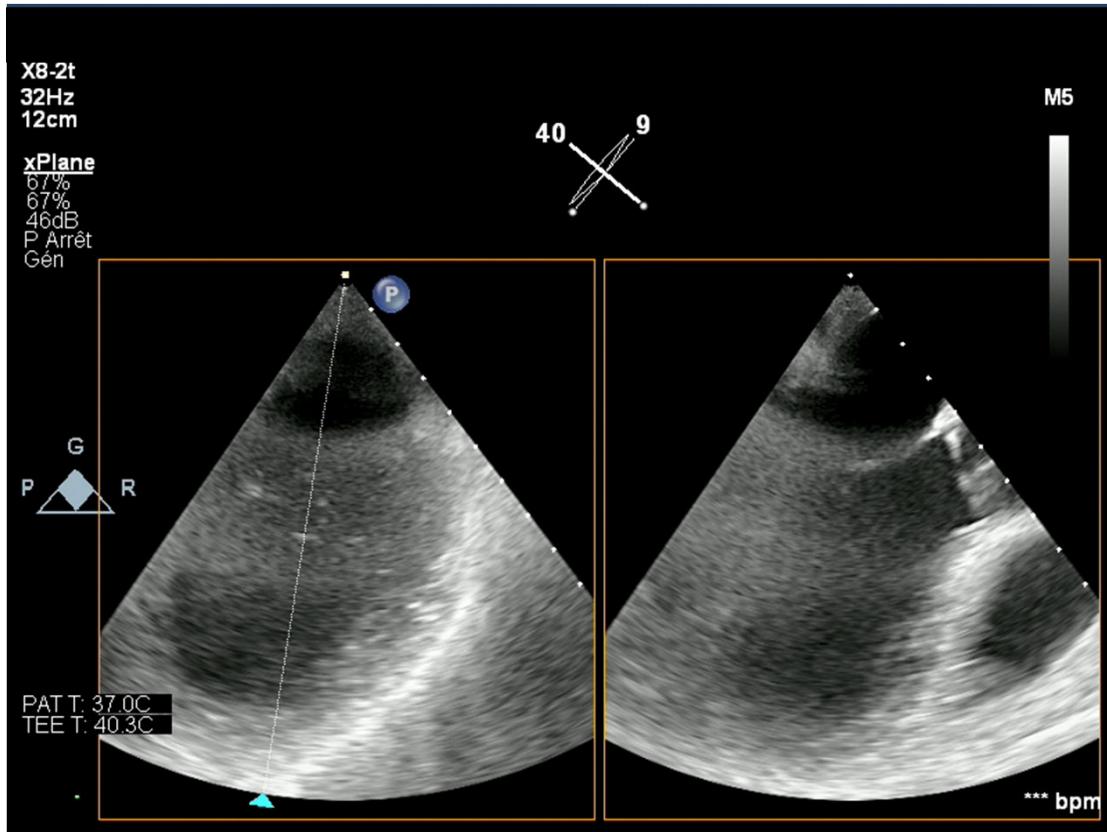




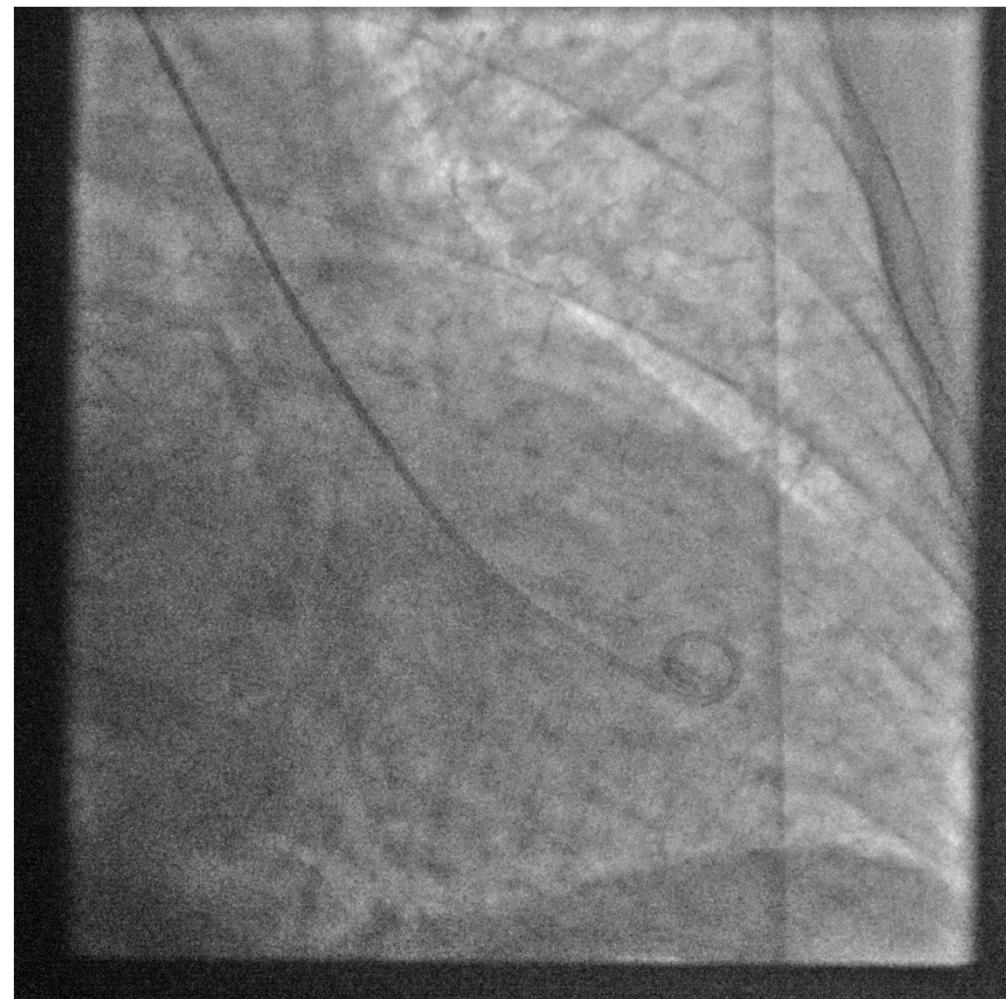
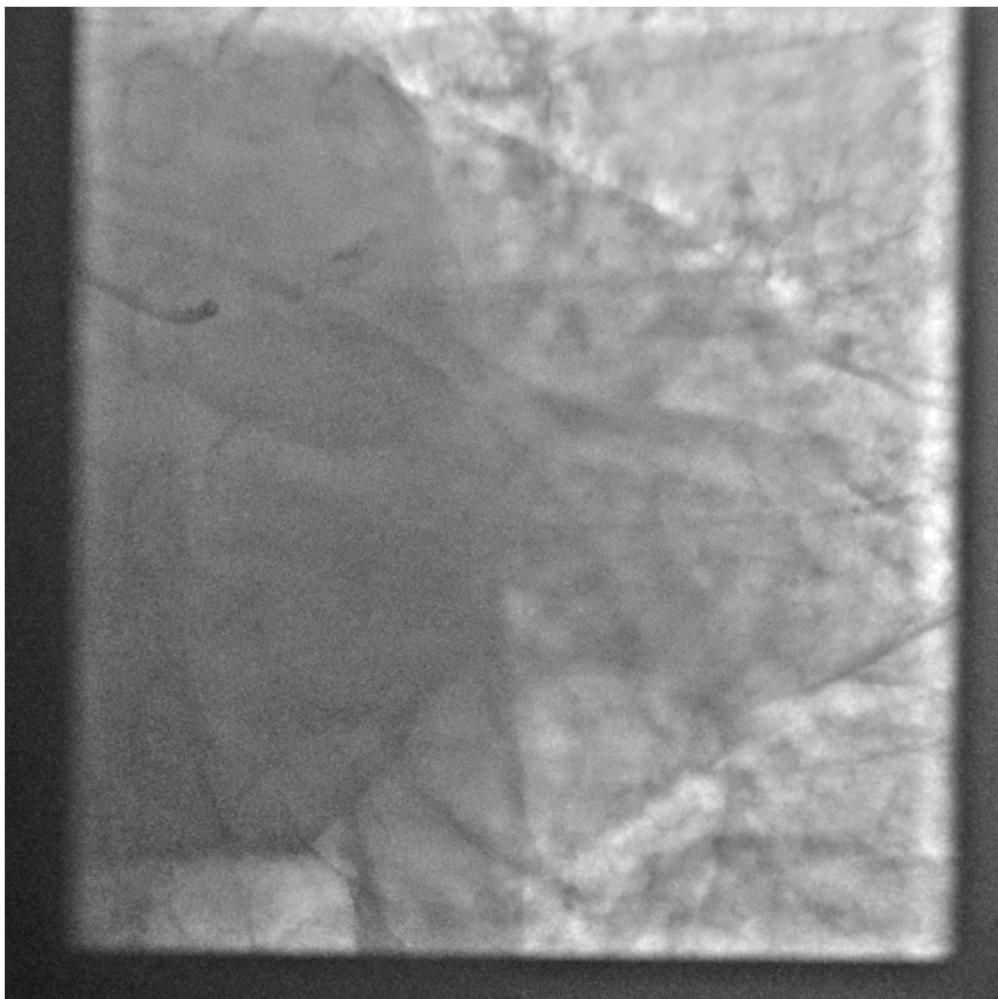
Infarctus inférieur tardif

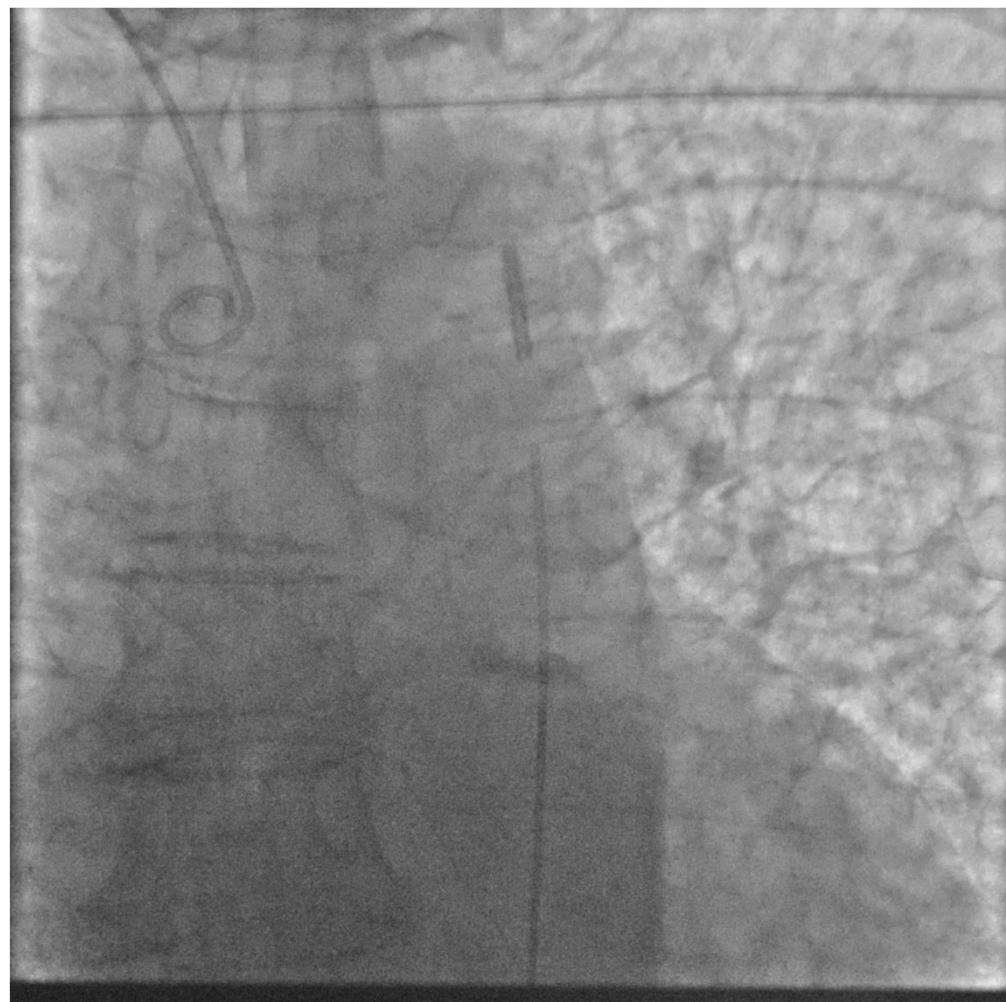
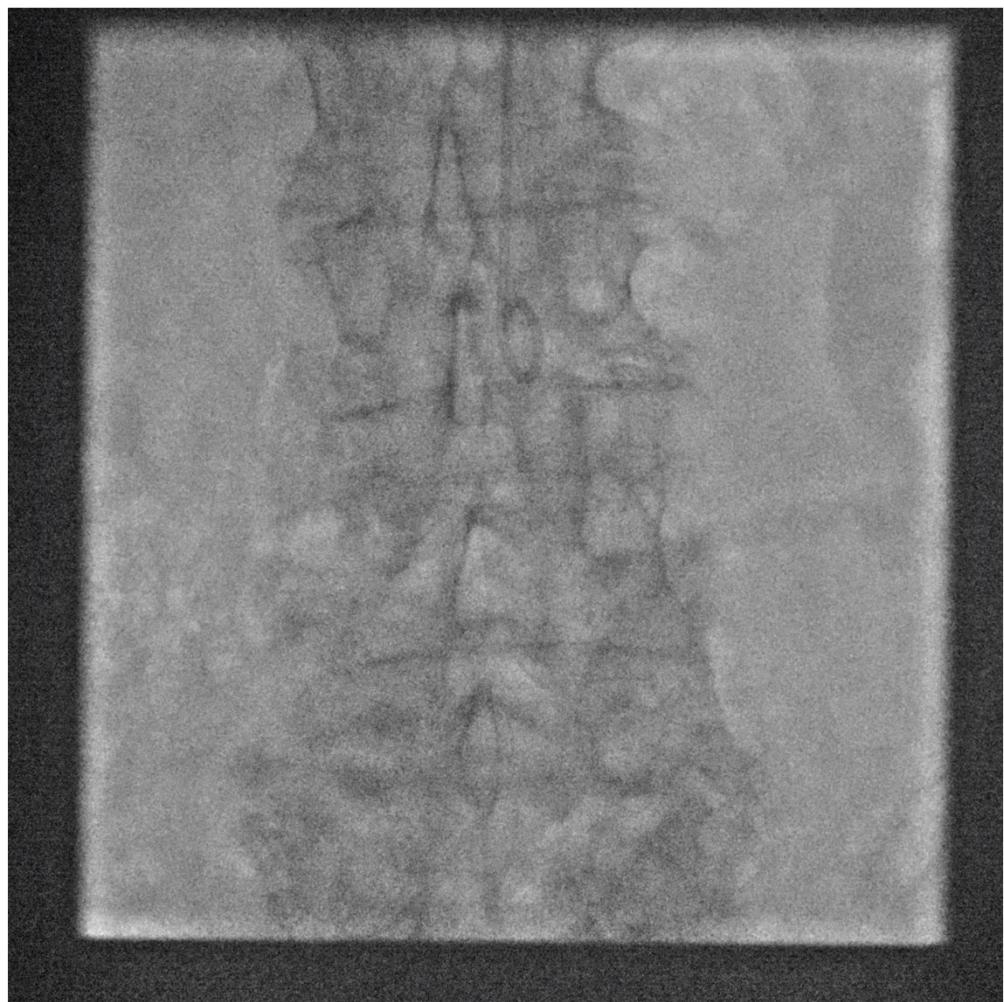






Rupture partielle de pilier





Surveillance

- Surveillance

- clinique: point de ponction, membre inférieur, transit, diurèse
- biologique: anticoagulation, hémoglobine, plaquettes, lactates artériels
- radiologique: radiographie pulmonaire de face

- Les Traitements

- anticoagulation
- anxiolytiques

- La console

- pas d'arrêt prolongé
- optimisation de l'inflation
- présence de fuites
- niveau d'hélium

Effets secondaires

- Thrombopénie
- Thrombose artérielle
- Hypoperfusion rénale, mésentérique, ou du membre inférieur

 douleur abdominale chez le (la) patient(e) porteur d'une CPIA

Sevrage et retrait

⚠ Dispositif transitoire! A enlever dès que possible!

- Sevrage progressif
(1 inflation tous les 2 cycles cardiaques puis 1 inflation tous les 4 cycles cardiaques)
- Arrêt de l'héparinothérapie 1 heure avant
- Soit préclosing à la pose
- Soit compression manuelle fémorale (30 minutes) sous couvert d'antalgiques

**Randomized comparison of
intraaortic balloon counterpulsation
versus
optimal medical therapy in addition to early
revascularization in acute myocardial infarction
complicated by cardiogenic shock**

Holger Thiele, MD

Uwe Zeymer, MD; Franz-Josef Neumann, MD; Miroslaw Ferenc,
MD; Hans-Georg Olbrich, MD; Jörg Hausleiter, MD; Gert Richardt, MD;
Marcus Hennersdorf, MD; Klaus Empen, MD; Georg Fuernau, MD; Steffen Desch, MD;
Ingo Eitel, MD; Rainer Hambrecht, MD; Jörg Fuhrmann, MD; Michael Böhm, MD;
Henning Ebelt, MD; Steffen Schneider, PhD;
Gerhard Schuler, MD; Karl Werdan, MD

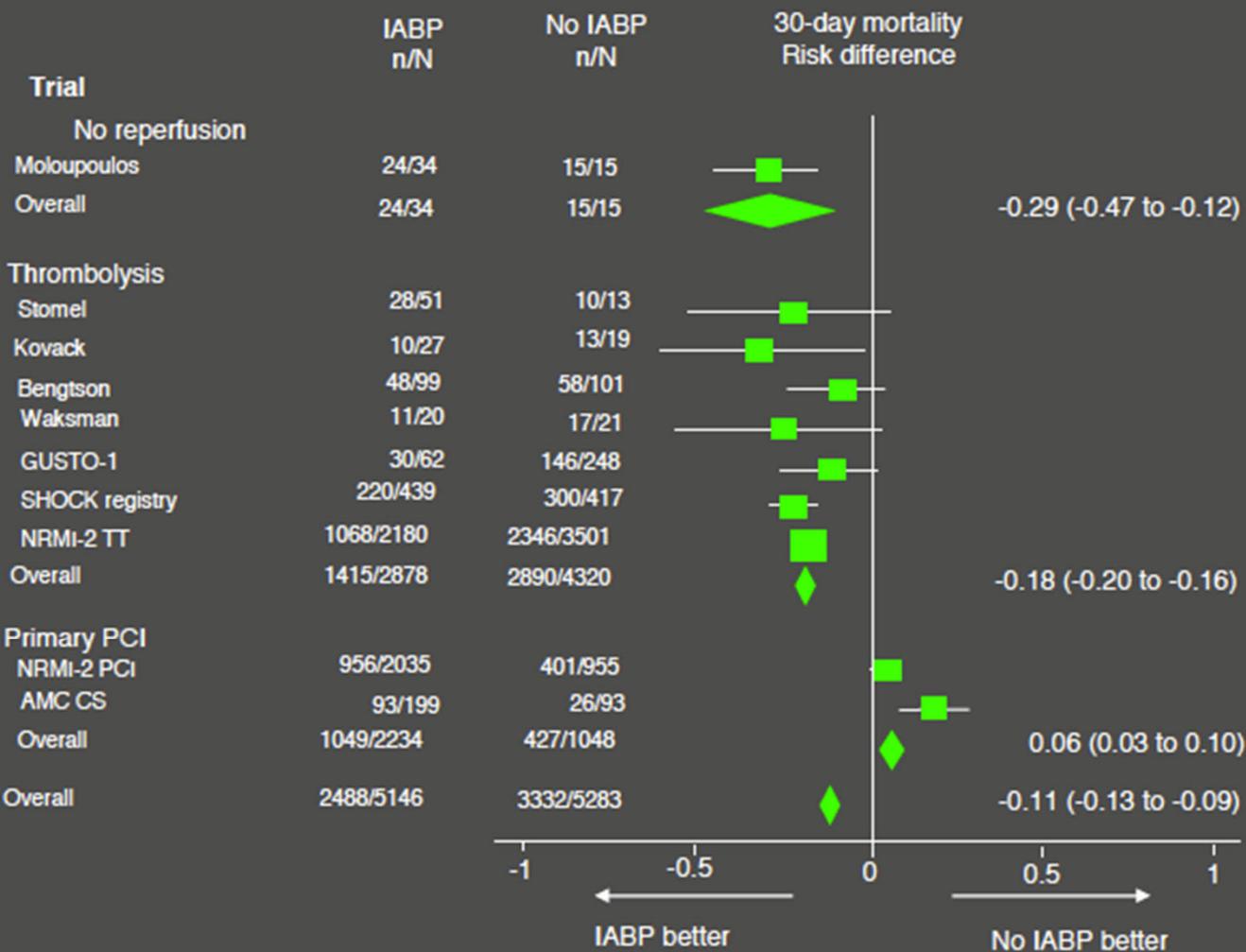
on behalf of the **IABP-SHOCK II Trial** Investigators

University of Leipzig – Heart Center

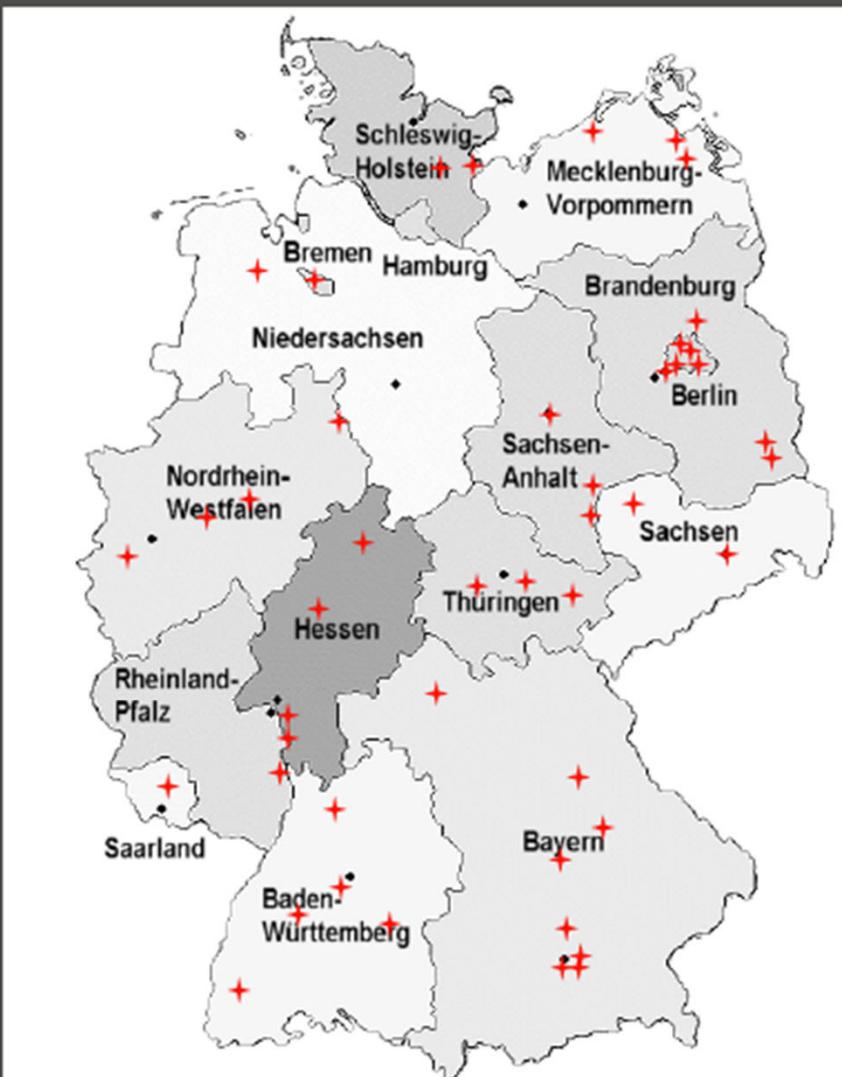
Background

Mortality IABP vs no IABP - Metaanalysis

IABP SHOCK II
Randomized Clinical Trial



Study Sites and Organisation



DSMB:

Kurt Huber
Ferenc Follath
Bernhard Maisch
Johannes Haerting

Steering committee:

Holger Thiele
Karl Werdan
Uwe Zeymer
Gerhard Schuler

Support + Patronage:



Sample Size

- Estimated 12% absolute difference in survival rates
- Sequential statistical design with 2 interim analyses (33% and 66% of patients)
- Significance level 0.0005 at 1st or 0.014 at 2nd interim analysis.
Final analysis at α -level 0.044 → 564 patients
- To compensate losses in follow-up and putative center effect → 600 patients

Primary Study Endpoint:

30-day all-cause mortality

Secondary Study Endpoints:

- Hemodynamic parameters (mean BP, heart rate pre and post revascularization)
- Serum-lactate (every 8 h for 48 h)
- SAPS-2 Score
- Serial creatinine-level and creatinine-clearance (Cockcroft-Gault-formula)
- Inflammatory reaction (CRP)

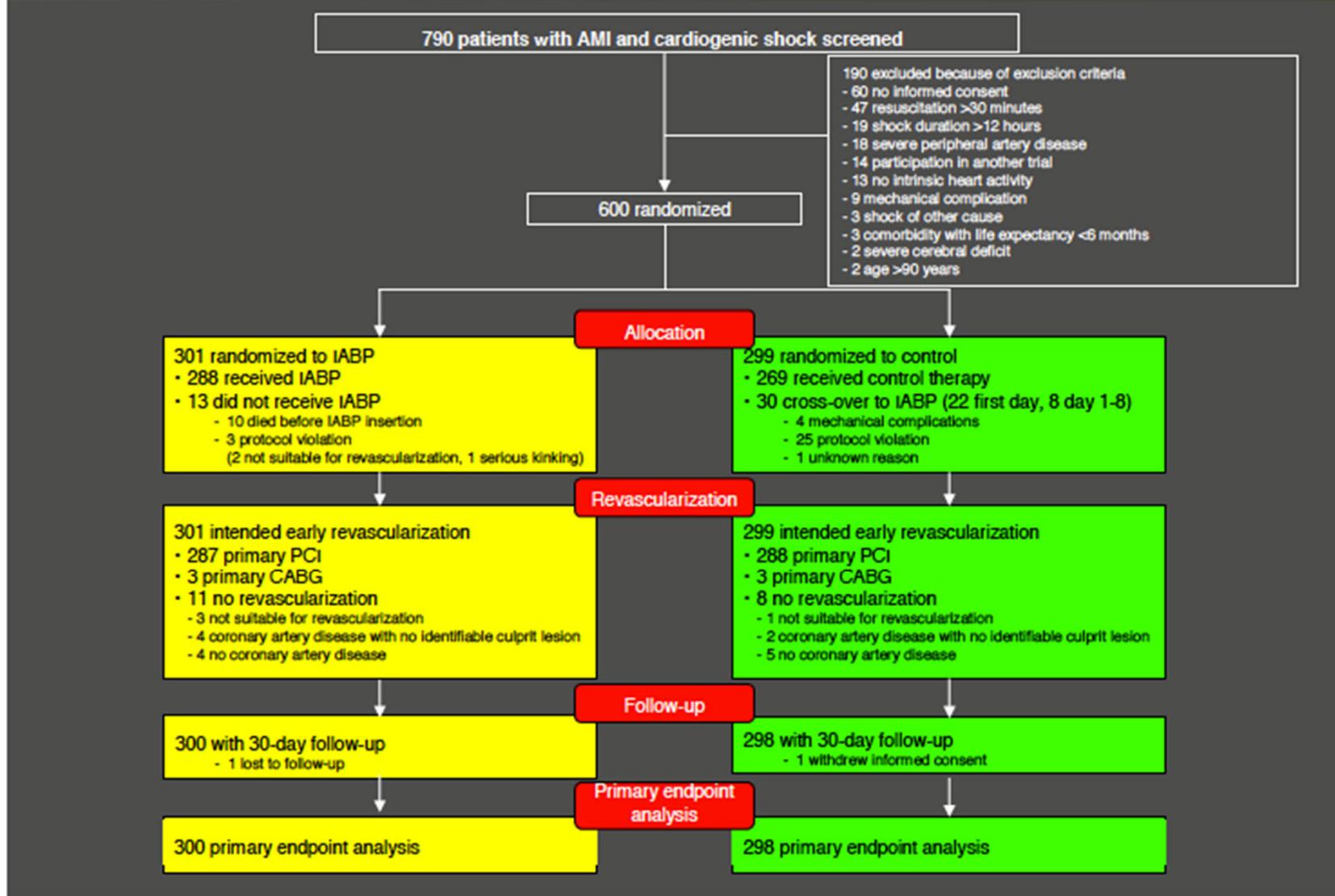
Process of care

- Time until hemodynamic stabilization
- Catecholamine dose and duration
- Requirement for LVAD-implantation or HTx
- Requirement for renal replacement therapy
- Length of ICU-stay
- Length of mechanical ventilation
- Mortality after 6 and 12 months

Results

Trial Flow and Treatment

IABP
SHOCK II
PAN-AMERICAN CLINICAL TRIAL



Results

Patient Characteristics

IABP SHOCK II
Randomized Clinical Trial

	IABP (n=301)	Control (n=299)
Age (years); median (IQR)	70 (58-78)	69 (58-76)
Male sex; n (%)	202 (67.1)	211 (70.6)
Current Smoking; n/total (%)	96/295 (32.5)	108/299 (36.1)
Hypertension; n/total (%)	213/296 (72.0)	199/299 (66.6)
Hypercholesterolemia; n/total (%)	122/295 (41.4)	105/299 (35.1)
Diabetes mellitus; n/total (%)	105/297 (35.4)	90/299 (30.1)
Body mass index (kg/m ²); median (IQR)	27.5 (24.7-30.1)	26.9 (24.7-29.4)
Prior myocardial infarction; n/total n (%)	71/300 (23.7)	61/299 (20.4)
Prior PCI; n/total n (%)	63/299 (21.1)	52/299 (17.4)
Prior CABG; n/total (%)	20/300 (6.7)	12/299 (4.0)
Fibrinolysis < 24 h before randomization; n/total (%)	28/301 (9.3)	20/299 (6.7)
STEMI/LBBB; n/total (%)	200/300 (66.7)	212/298 (71.1)
NSTEMI; n/total (%)	96/300 (32.0)	81/298 (27.2)
Resuscitation before randomization; n/total (%)	V 127/301 (42.2%)	143/299 (47.8)
Signs of impaired organ perfusion; n/total (%)		
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/l	226/300 (75.3)	218/298 (73.2)
Creatinine clearance (ml/min); median (IQR)	60.7 (43.4-86.6)	56.8 (39.7-78.1)
Infarct related artery; n/total (%)		
LAD	132/293 (45.1)	121/293 (41.3)
LCX	55/293 (18.8)	57/293 (19.5)
RCA	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)
Multivessel disease; n/total (%)	235/296 (79.4)	228/293 (77.9)
Left ventricular ejection fraction (%); median (IQR)	V 35 (25-45)	35 (25-45)

Results**Treatment + Process of Care Outcomes**

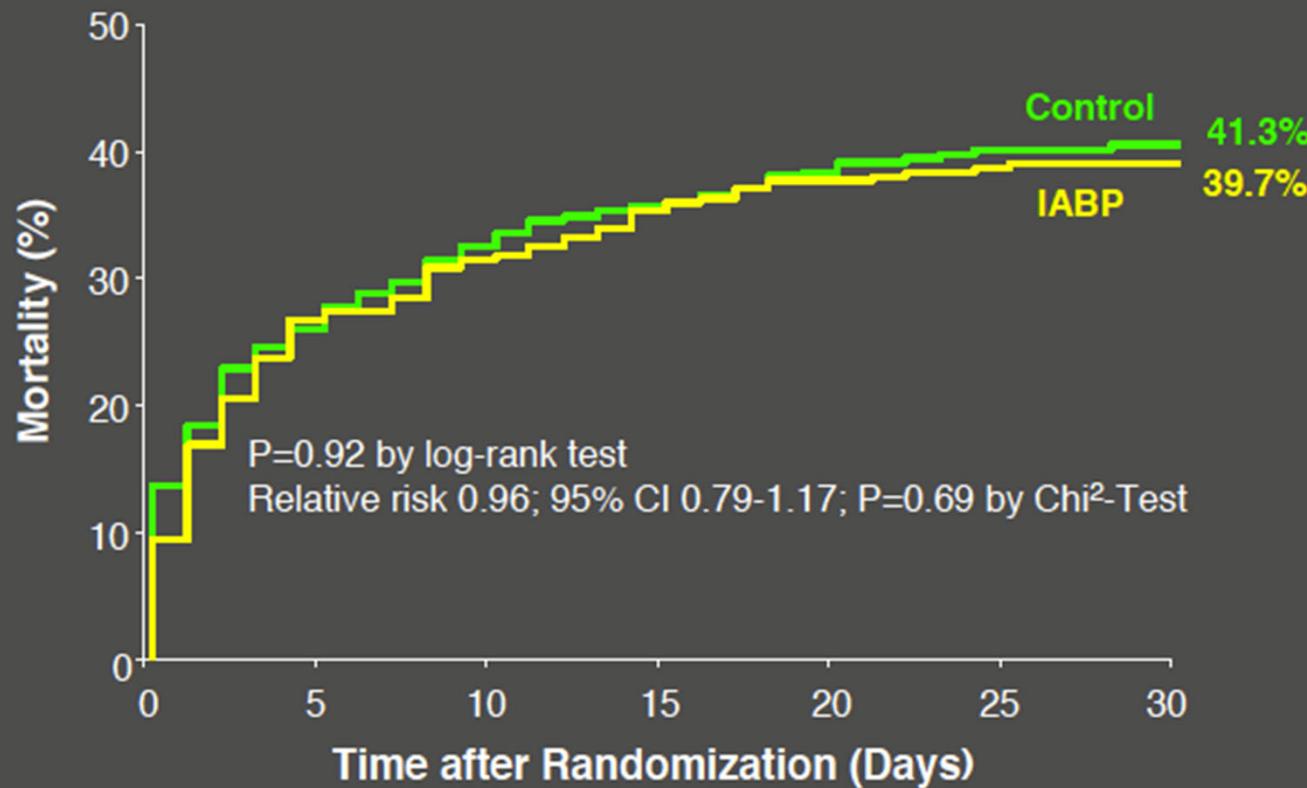
**IABP
SHOCK II**
PANDEMIC CLINICAL TRIAL

Variable	IABP (n=301)	Control (n=299)	p
Primary PCI; n/total (%)	287/301 (95.3)	288/299 (96.3)	0.55
Stent implanted; n/total (%)	273/301 (90.7)	266/299 (89.0)	0.48
Drug-eluting stent; n/total (%)	126/301 (41.9)	123/299 (41.1)	0.86
Immediate PCI of non-culprit lesions; n/total (%)	90/301 (29.9)	81/299 (27.1)	0.45
Immediate bypass surgery; n/total (%)	8/301 (2.7)	10/299 (3.3)	0.62
Staged bypass surgery; n/total (%)	3/301 (1.0)	4/299 (1.3)	0.72
Active left ventricular assist device; n/total (%)	11/301 (3.7)	22/299 (7.4)	0.053
Mild hypothermia; n/total (%)	106/301 (35.2)	120/299 (40.1)	0.21
Mechanical ventilation; n/total (%)	240/301 (79.7)	252/299 (84.3)	0.15
Duration of mechanical ventilation (days); median (IQR)	3.0 (1.0-8.0)	3.0 (1.0-8.0)	0.44
Duration of intensive care treatment (days); median (IQR)	6.0 (3.0-12.0)	6.0 (3.0-13.0)	0.34
Renal replacement therapy; n/total (%)	62/301 (20.6)	47/299 (15.7)	0.12
Antiplatelets and anticoagulation; n/total (%)	293/299 (98.0)	284/298 (95.3)	0.07
Aspirin	216/299 (72.2)	206/298 (69.1)	0.40
Clopidogrel	80/299 (26.8)	76/298 (25.5)	0.73
Prasugrel	19/234 (8.1)	15/228 (6.6)	0.52
Ticagrelor*	138/299 (46.2)	143/298 (48.0)	0.63
Glycoprotein IIb/IIIa-inhibitors	288/299 (96.3)	275/298 (92.3)	0.03
Unfractionated heparin	60/299 (20.1)	59/298 (19.8)	0.94
Low molecular weight heparin	29/299 (9.7)	36/298 (12.1)	0.34
Bivalirudin			
Catecholamines (µg/kg per minute); median (IQR)			
Dopamine	4.1 (2.9-7.7)	4.2 (3.6-8.3)	0.76
Norepinephrine	0.3 (0.1-1.2)	0.4 (0.1-1.1)	0.73
Epinephrine	0.3 (0.1-1.3)	0.3 (0.2-1.4)	0.59
Dobutamine	10.2 (4.9-20.6)	9.0 (4.8-17.6)	0.25
Duration of catecholamines (days), median (IQR)	3.0 (1.0-5.0)	3.0 (1.0-6.0)	0.81
Time to hemodynamic stabilization (days); median (IQR)	3.0 (1.0-5.0)	3.0 (1.0-6.0)	0.50

Results

Primary Study Endpoint (30-Day Mortality)

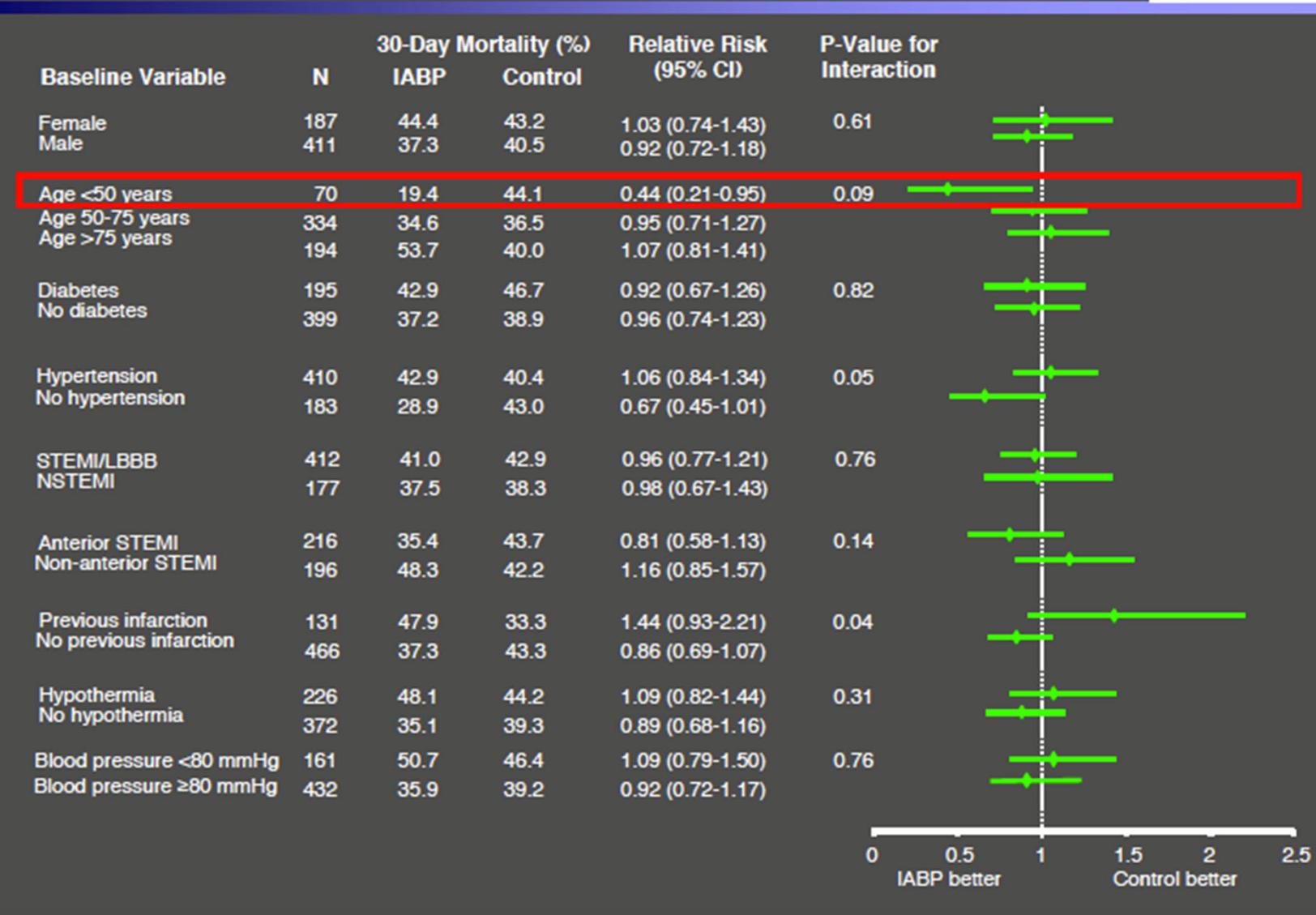
IABP
SHOCK II
PACEMAKER CLINICAL TRIAL



Results

Subgroups (30-Day Mortality)

IABP SHOCK II
Randomized Clinical Trial



Summary + Conclusions



- IABP support in cardiogenic shock is safe without significant inherent complications.
- However, IABP support did not reduce 30-day mortality in this large, randomized, multicenter trial in cardiogenic shock patients complicating myocardial infarction undergoing early revascularization.
- The primary study endpoint results are supported by a lack of benefit in secondary endpoints.

Conclusion

- Encore largement utilisé quand pas d'autre système disponible !
- Garde un « potentiel intérêt » dans certaines indications
 - Patient jeune, IVA proximale, « pré-choc »
 - Complications mécaniques de l'infarctus
 - Pré ou post- chirurgie cardiaque