

BRIGHAM HEALTH



BRIGHAM AND
WOMEN'S HOSPITAL



Cardiogenic shock

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HARVARD MEDICAL SCHOOL
TEACHING HOSPITAL



Disclosures

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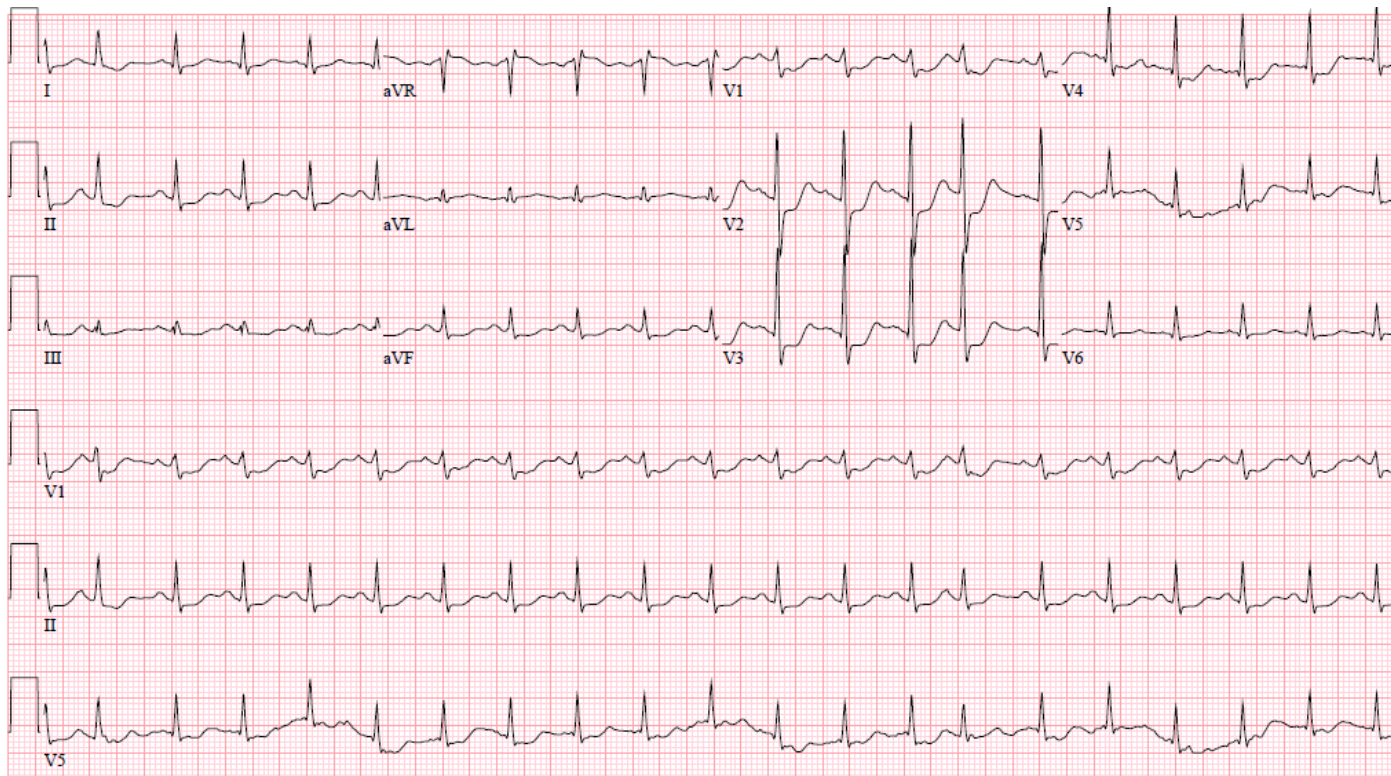
Case

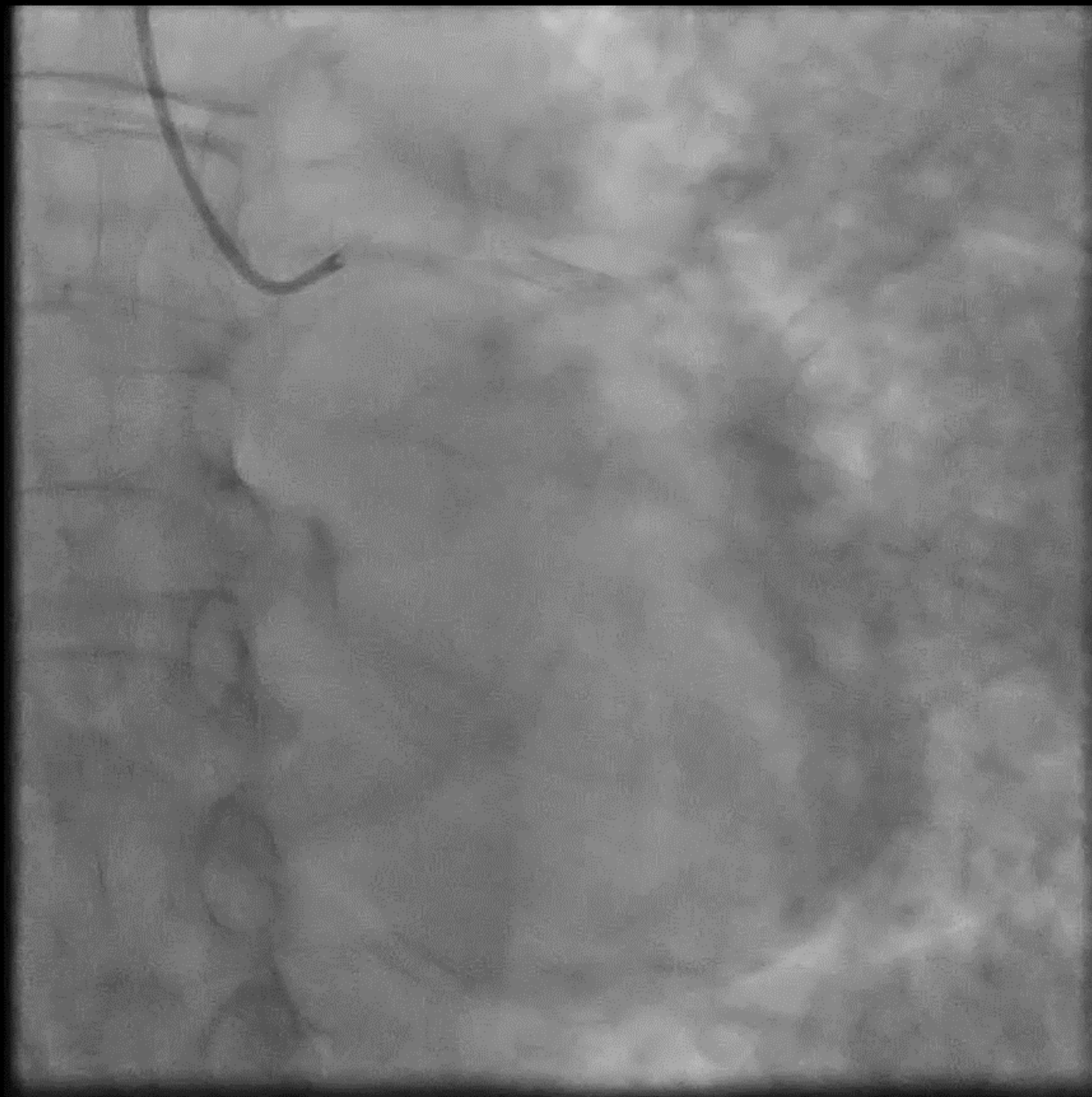
67-year-old man

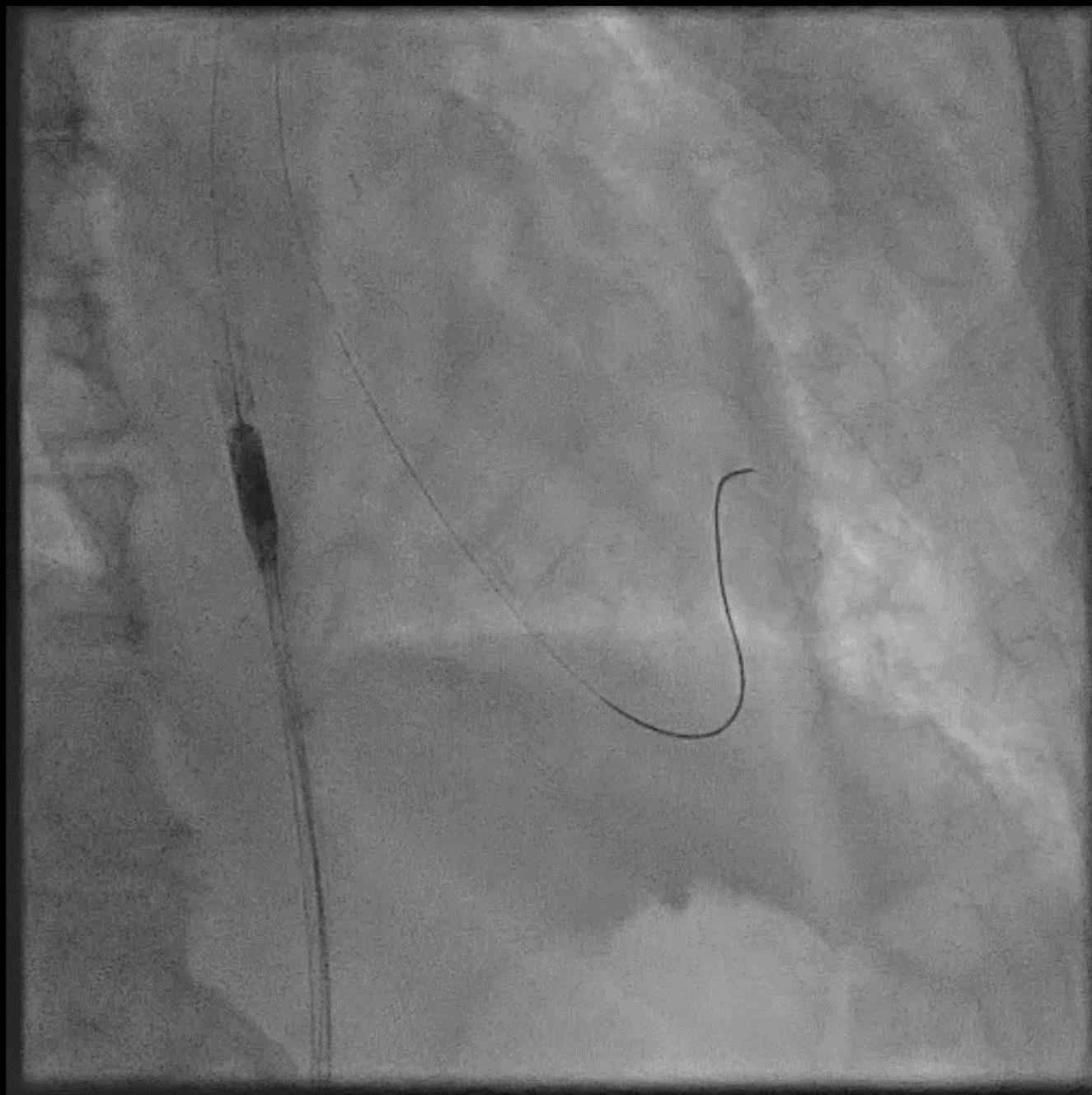
Cardiac arrest, defibrillated

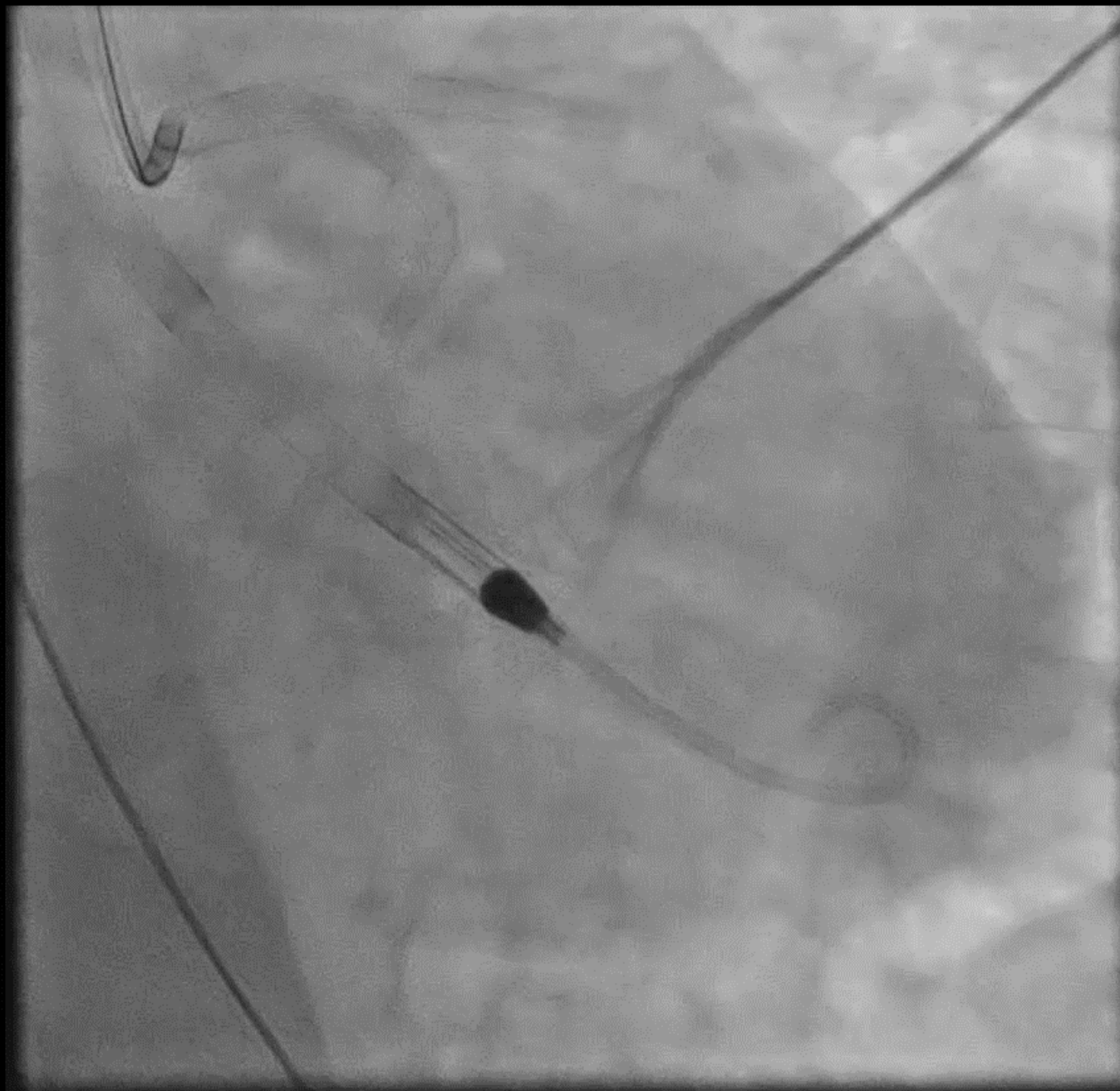
Recurrent episodes of VT

Hypotensive











Outline

Definition and Epidemiology

Management

- General supportive measures
- Etiologies with specific therapies
 - Acute MI
 - PE
- Mechanical circulatory support



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Cardiogenic Shock

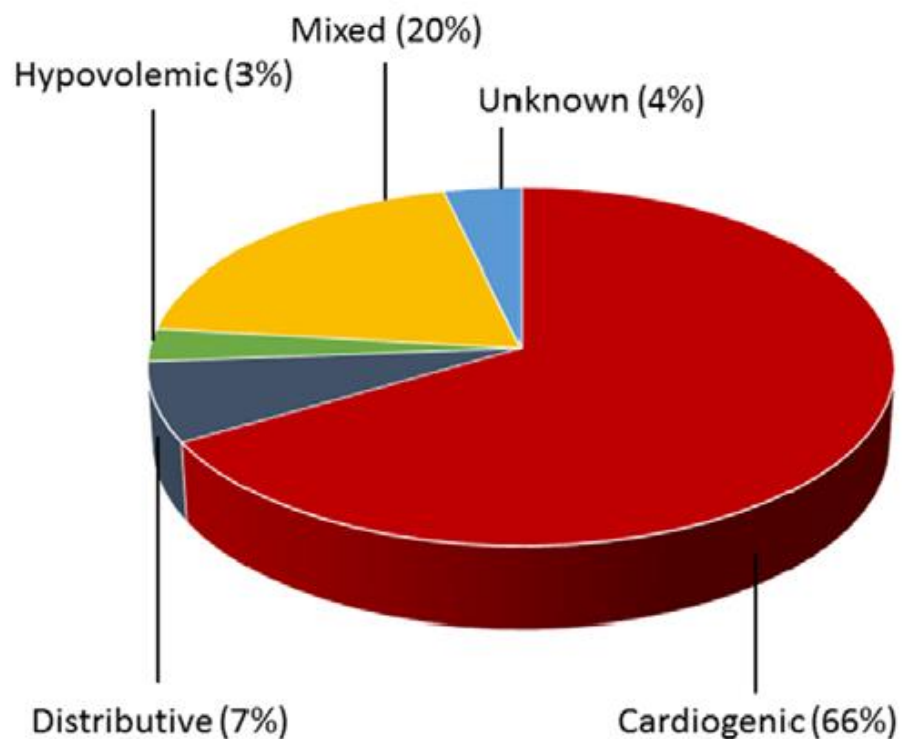
Clinical Definition	SHOCK Trial ^{9*}	IABP-SHOCK II ^{††}	ESC HF Guidelines ¹⁵
Cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Support to maintain SBP ≥90 mm Hg AND End-organ hypoperfusion (urine output <30 mL/h or cool extremities) Hemodynamic criteria: CI of $\leq 2.2 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ AND PCWP $\geq 15 \text{ mm Hg}$	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

1) Blood pressure threshold

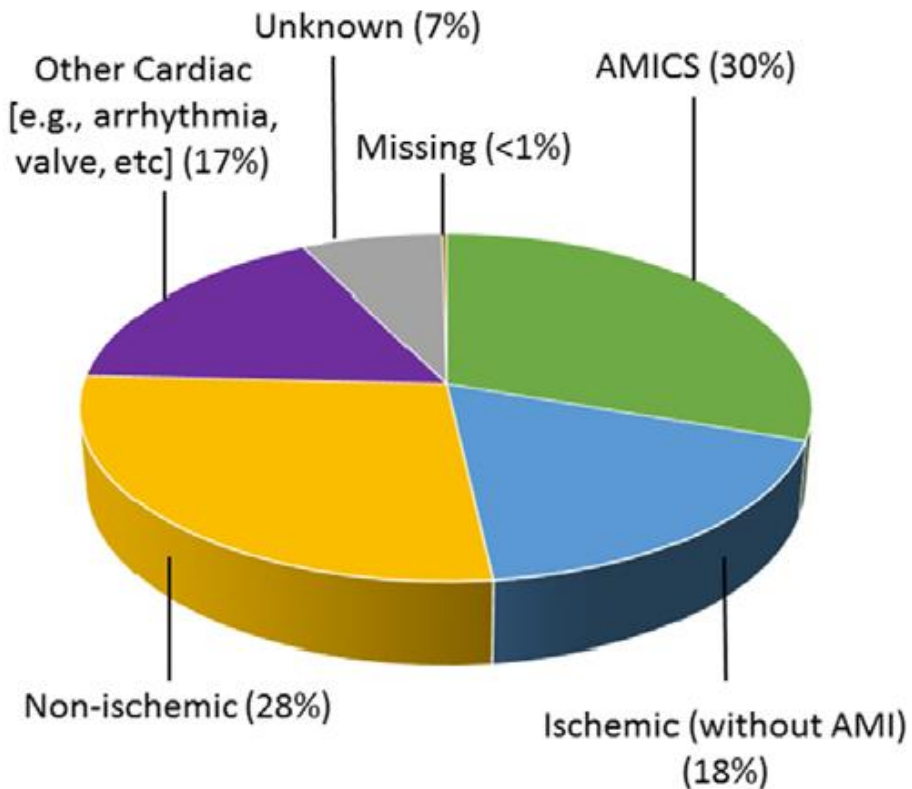
2) Clinical/laboratory evidence of hypoperfusion/congestion

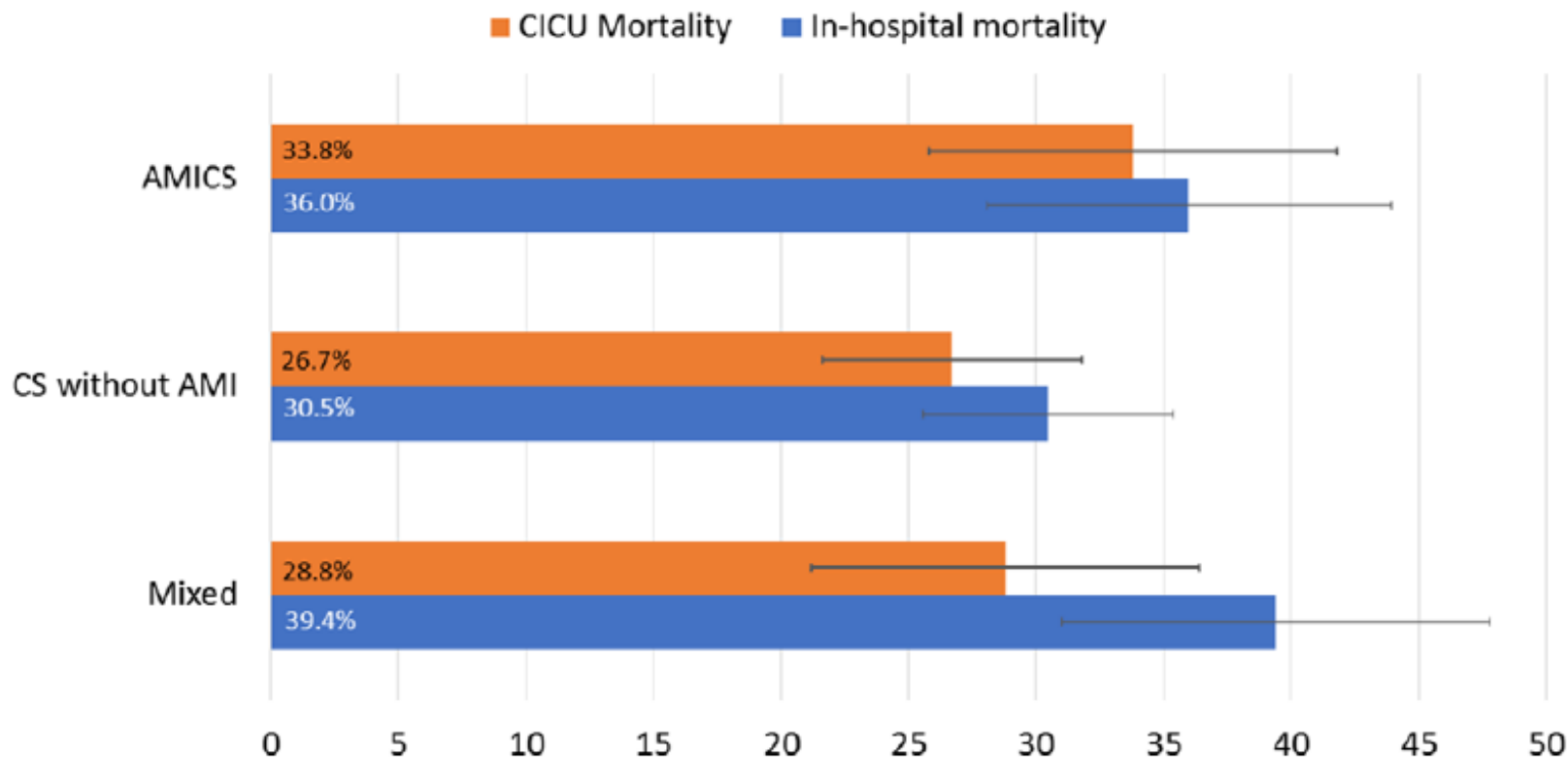
3) +/- Hemodynamic evidence of low flow/congestion

A Etiology of Shock (N=677)



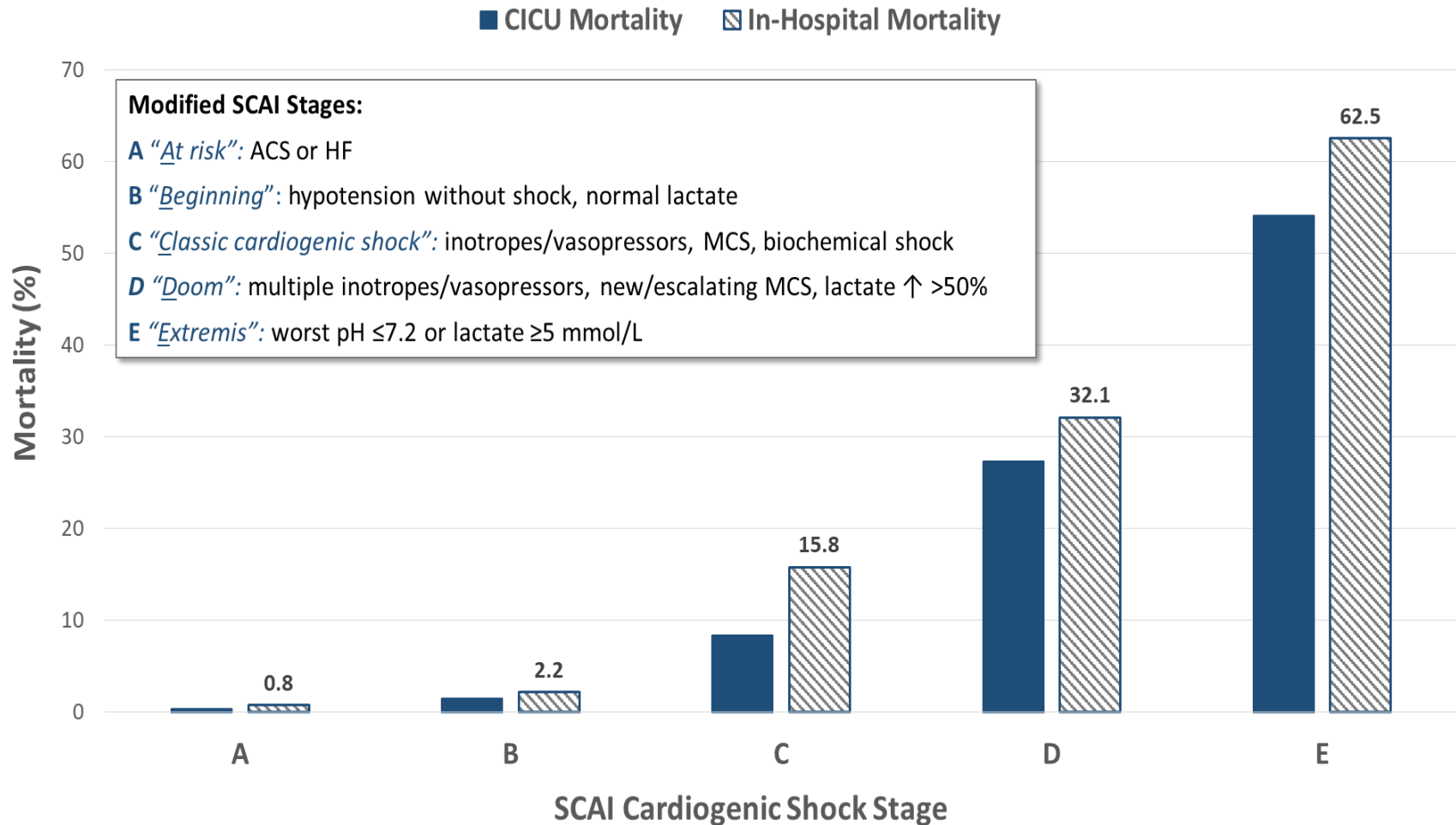
B Cause of Cardiogenic Shock (N=450)







Mortality by SCAI Classification





Care setting

At my hospital:

- A) There is no separate CICU**
- B) There is a CICU and they manage all patient care independently (vent, pressors, etc.)**
- C) There is a CICU but they need help with critically ill patients (Pulm/Crit Care co-manages)**
- D) Other**





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RCTs for P2Y12 inhibition in ACS/PCI

CURE (N=12,562)

The New England Journal of Medicine

EFFECTS OF CLOPIDOGREL IN ADDITION TO ASPIRIN IN PATIENTS WITH ACUTE CORONARY SYNDROMES WITHOUT ST-SEGMENT ELEVATION

PLATO (N=18,624)

The NEW ENGLAND
JOURNAL of MEDICINE

ESTABLISHED IN 1812

SEPTEMBER 10, 2009

VOL. 361 NO. 11

CLARITY-TIMI 28 (N=3,491)

JO

ESTAB

Addition
for M

N=59,430

N=0 with Cardiogenic Shock

(N=11,145)

JOURNAL of MEDICINE

TRITON-TIMI 38 (N=13,608)

The NEW ENGLAND
JOURNAL of MEDICINE

ESTABLISHED IN 1812

NOVEMBER 15, 2007

VOL. 357 NO. 20

Prasugrel versus Clopidogrel in Patients
with Acute Coronary Syndromes

ORIGINAL ARTICLE

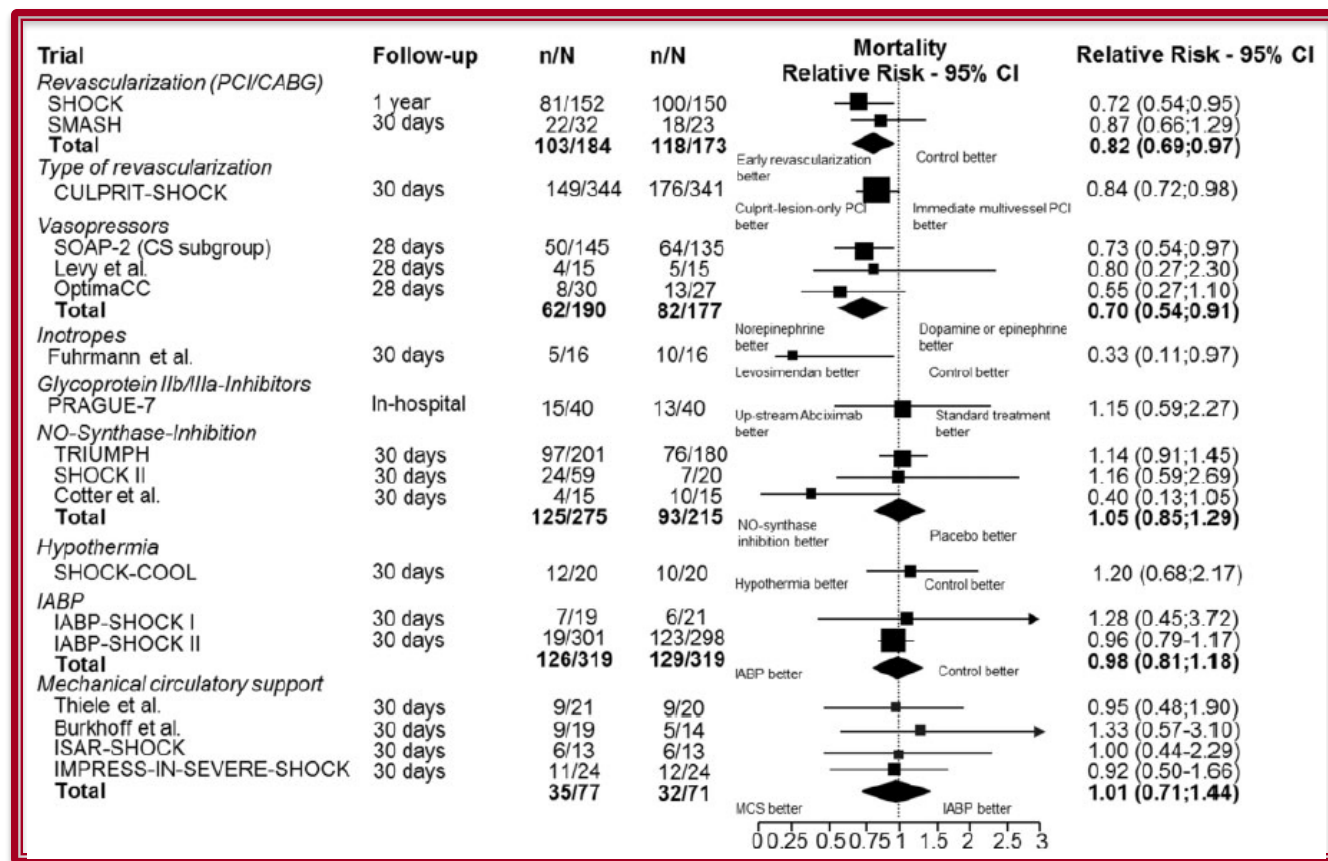
Effect of Platelet Inhibition with Cangrelor
during PCI on Ischemic Events





RCTs in Cardiogenic Shock

Total N~2,000





Etiologies

- Acute MI
- Mechanical complication of MI (VSD, MR, free wall rupture)
- Valvular heart disease
- NICMP with ADHF
- Arrhythmia
- PE
- Tamponade
- Myocarditis
- Congenital heart disease with ADHF
- Pulmonary hypertension
- RV failure
- *Et cetera...*



Uni- or Bi-Ventricular Failure?

Hemodynamic Profiles of Various Forms of Shock

Type of shock	RAP	PCWP	CO	SVR	CPO	PAPi
1° L-sided	nl or ↑	↑	↓	↑	≤0.6	>0.9
1° R-sided	↑	nl or ↓	↓	↑	> or < 0.6	≤0.9
Biventricular	↑	↑	↓	↑	≤0.6	≤0.9

- Cardiac power output (**CPO**) (W) = $\text{MAP} \times \text{CO} / 451$
- Pulmonary artery pulsatility index (**PAPi**) = $(\text{PA systolic} - \text{PA diastolic}) / \text{RA mean}$



For mild to moderate shock

↑ Cardiac output

↓ Resistance

↓ Filling pressures

Inotrope

Vasodilator +
Diuretic



Vasoactive therapies

Pure vasopressors – Incr SVR

Inopressors – Incr CO, Incr SVR

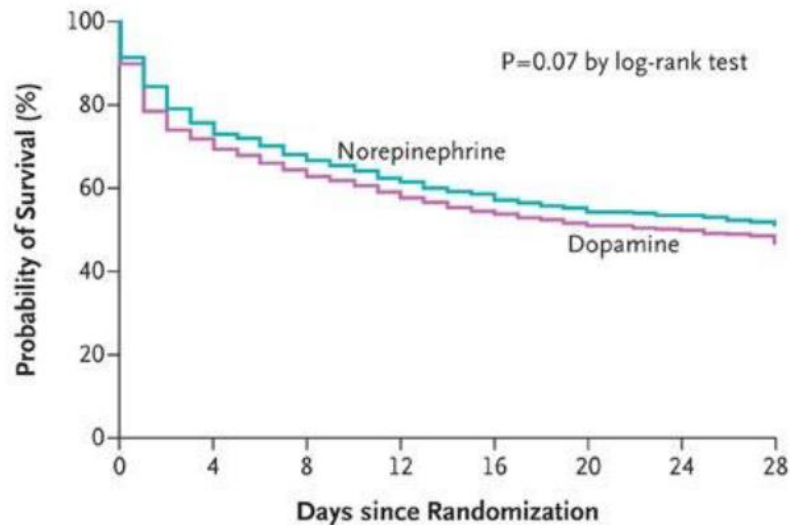
Inodilators – Incr CO, decr SVR

Vasoactive Drugs							
Drug	Receptors	MAP	HR	CO	SVR	PVR	Comment
Pure vasopressors							
Phenylephrine	Pure α_1	↑↑	↓↓ ^a	↓ ^a	↑↑↑	↑↑	
Vasopressin	V_1 & V_2	↑↑	↓↓ ^a	↓ ^a	↑↑↑	↔	Consider if refractory to catechols. Attractive if RV dysfxn or PHT.
Inopressors (relative pressor vs. inotropy depends on drug & dose)							
Norepinephrine	$\alpha \gg \beta_1$	↑↑	↔/↑	↔/↑	↑↑↑	↔/↑	More pressor than inotrope. Fewer tachyarrhythmias than w/ dopa and mortality at least as good if not better.
Epinephrine							
Low-dose	β_1 & $\beta_2 > \alpha$	↑	↑↑	↑↑	↓	↔	Inotrope
High-dose	$\alpha > \beta$	↑↑	↑↑	↑↑	↑↑	↑	Inotrope+pressor
Dopamine ^b							
Low-dose	D	↔	↔/↑	↔/↑	↔/↓	↔	
Medium-dose	$\beta_1 > D, \alpha$	↔/↑	↑	↑↑	↔	↔	
High-dose	$\alpha > \beta_1, D$	↑↑	↑↑	↑	↑↑	↑	
Inodilators							
Dobutamine	$\beta_1 \gg \beta_2, \alpha_1$	↔/↓	↑↑	↑↑	↓	↓	↓ PCWP. Fast onset. Tachyphylaxis.
Milrinone	PDE ₃ inhib	↓↓	↑	↑↑↑	↓↓	↓↓	↓↓ PCWP; ↓ PVR; ∴ attractive if RV dysfxn or PHT. Slow onset. Renally cleared.
Isoproterenol	β_1 & β_2	↓	↑↑↑	↑↑	↓↓	↓	⊕ chronotrope
Pure vasodilators							
Nitroglycerin	NO → sGC	↓	↑	↔	↓	↓	Venodilator >> arteriolar dilator
Nitroprusside ^c	NO → sGC	↓↓↓	↑	↑↑ ^c	↓↓↓	↓↓	Arteriolar dilator ≥ venodilator



SOAP II: Dopamine vs Norepinephrine

1679 patients with shock



No. at Risk								
Norepinephrine	821	617	553	504	467	432	412	394
Dopamine	858	611	546	494	452	426	407	386

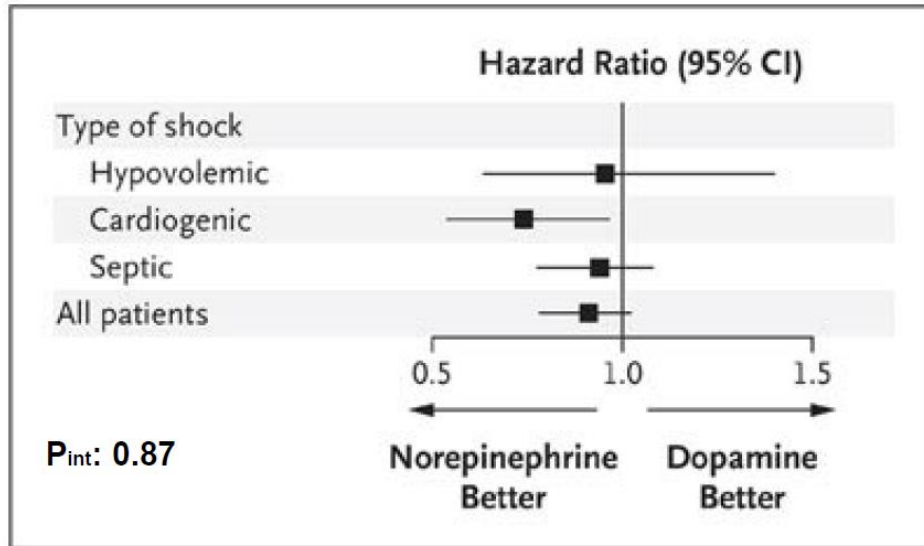
- **28d mortality:**
 - **52.5% for DA vs 48.5% for norepi**
 - **OR 1.17 (0.97-1.42), $p=0.10$**
- **Arrhythmias: 24.1% vs 12.4%**

De Backer et al. NEJM 2010;362:779.



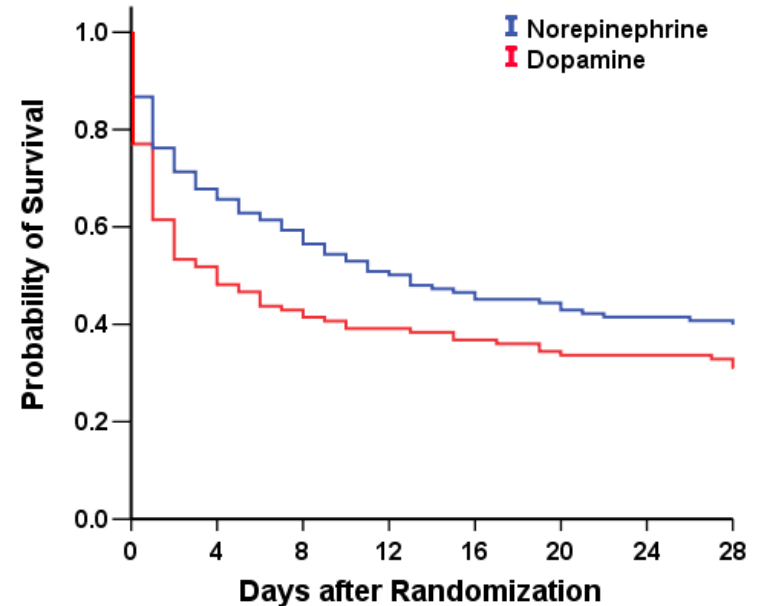


SOAP II: Dopamine vs Norepinephrine



Signal of harm with dopamine?

Cardiogenic Shock (N=280)





Epinephrine vs Norepinephrine

57 pts with CS due to AMI s/p PCI and with PA line in place

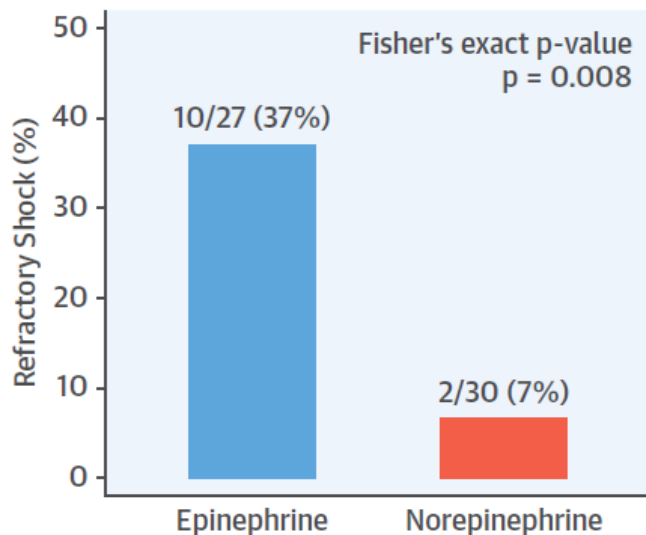


TABLE 2 Serious Adverse Events and Outcomes

	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

Values are n (%) unless otherwise indicated. Odds ratios were expressed by using the norepinephrine group as reference. *p value from the Fisher exact test. †p value from the Wald test.

ECLS = extracorporeal life support.

*Refractory Shock: Sustained hypotension,
end-organ hypoperf, incr LA, high inotrope or
vasopressor doses*



Milrinone vs Dobutamine

SCAI B,C,D, or E

PEP: In-hospital death,
resuscitated cardiac
arrest, cardiac
transplant/MCS, MI,
TIA/stroke, or RRT

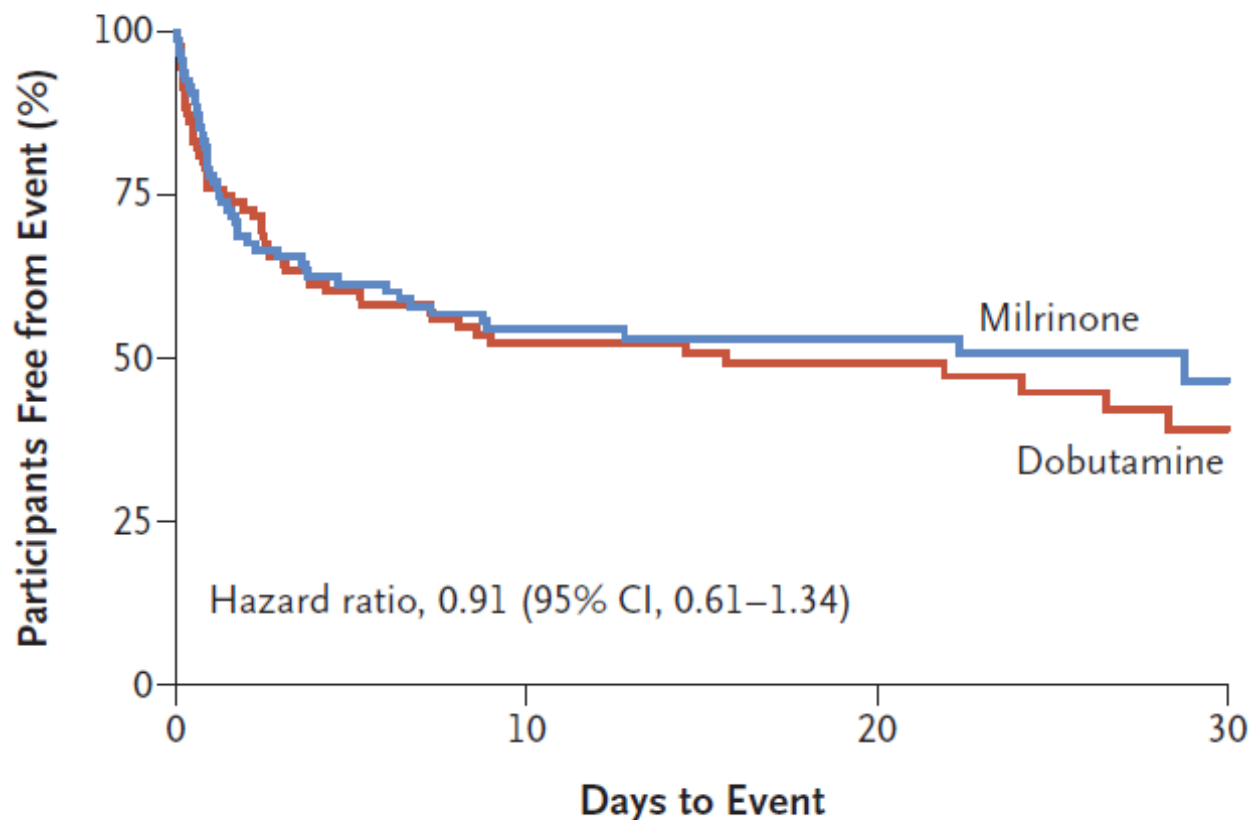
Table 1. Baseline Characteristics of the Participants.*

Characteristic	Milrinone (N = 96)	Dobutamine (N = 96)
Age — yr	68.9±13.8	72.0±11.3
Female sex — no. (%)	36 (38)	34 (35)
Median body-mass index (IQR) †	26.4 (23.7–31.0)	26.0 (22.5–30.5)
Race — no. (%) ‡		
White	86 (90)	79 (82)
Non-White	10 (10)	17 (18)
Left ventricular function		
Median left ventricular ejection fraction (IQR) — %	25 (20–40)	25 (20–40)
Cause of ventricular dysfunction — no. (%)		
Ischemic	66 (69)	62 (65)
Nonischemic	30 (31)	33 (34)
Coexisting conditions — no. (%)		
Previous myocardial infarction	39 (41)	29 (30)
Previous percutaneous coronary intervention	30 (31)	19 (20)
Previous coronary-artery bypass grafting	20 (21)	19 (20)
Previous stroke or transient ischemic attack	13 (14)	15 (16)
Atrial fibrillation	49 (51)	46 (48)
Chronic kidney disease §	38 (40)	40 (42)
Chronic liver disease	6 (6)	7 (7)
Chronic obstructive pulmonary disease	11 (11)	14 (15)
SCAI cardiogenic shock class — no. (%) ¶		
A	0	0
B	6 (6)	5 (5)
C	77 (80)	78 (81)
D	10 (10)	12 (12)
E	3 (3)	1 (1)
Time from admission to the cardiac ICU to randomization — hr	23.4±92.6	17.9±50.6



Milrinone vs Dobutamine

A Primary Composite Outcome



No. at Risk

Milrinone	96	42	26	7
Dobutamine	96	43	25	13





Vasopressor summary

- **Limited evidence base**
- **Catecholamines have not demonstrated improved survival**
- **But, data suggest norepinephrine may be better than dopamine or epinephrine**





Step-Wise Approach to CS Management

- **Correct hypotension (MAP goal ≥ 65 mmHg), typically with inopressor initially (often norepinephrine)**
- **Assess degree of congestion (preload) & adequacy of perfusion (CO)**
- **Assess and treat reversible causes of cardiogenic shock:**
 - Acute ischemia, etc
 - Other potential contributors: dysrhythmias, acid/base disturbances, negative inotropes (bB, CCB) and antihypertensives
- **Optimize hemodynamics, often with PAC to guide therapy**



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Etiologies

- Acute MI
- Mechanical complication of MI (VSD, MR, free wall rupture)
- Valvular heart disease
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- Arrhythmia
- PE
- Tamponade
- Myocarditis
- Congenital heart disease with ADHF
- Pulmonary hypertension
- RV failure
- *Et cetera...*



Acute MI complicated by shock

Early revascularization

General supportive measures

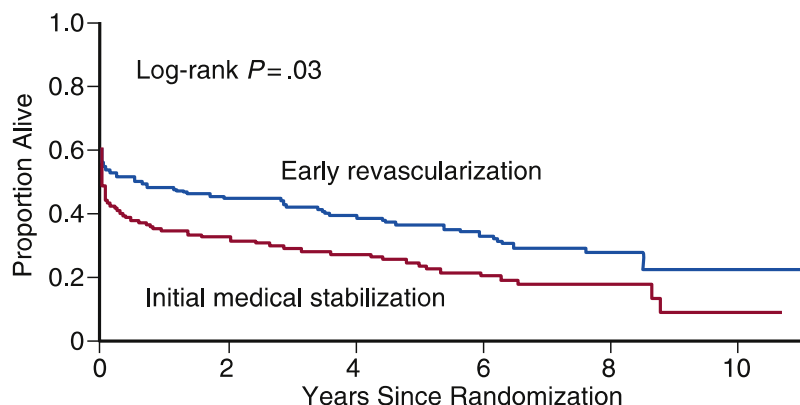
Mechanical circulatory support as needed

Recognition and mgmt of mechanical complications





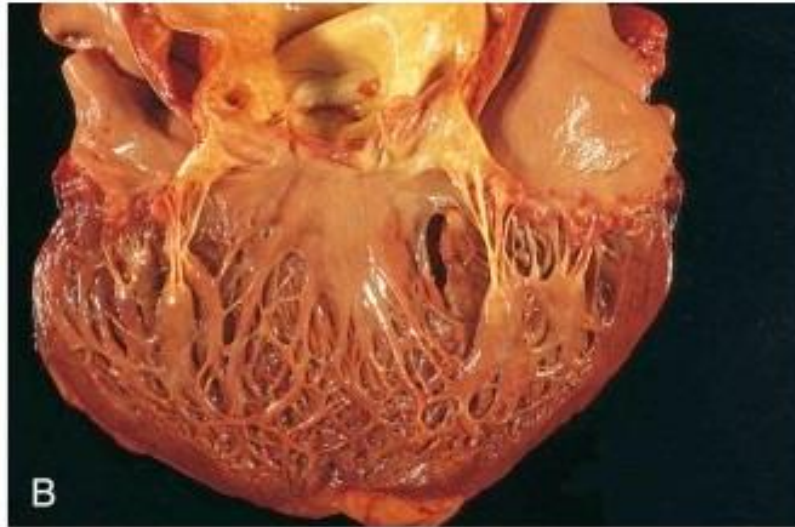
Mortality Benefit with Early Revascularization



No. at risk						
Early revascularization	152	56	42	33	18	3
Initial medical stabilization	150	38	29	18	9	2

- 302 pts with STEMI and CS
- Early revasc w/in 6 hrs vs med Rx followed by prn revasc
- Survival
 - 30 d: 53.3% vs 44.0% ($p=0.11$)
 - 1 yr: 46.7% vs 33.6% ($p<0.03$)
 - 6 yr: 32.8% vs 19.6% ($p=0.03$)

Mechanical Complications





Mechanical Complications

CHARACTERISTIC	VENTRICULAR SEPTAL RUPTURE	RUPTURE OF THE VENTRICULAR FREE WALL	PAPILLARY MUSCLE RUPTURE
Incidence	1-3% without reperfusion therapy, 0.2-0.34% with fibrinolytic therapy, 3.9% in patients with cardiogenic shock	0.8-6.2%; fibrinolytic therapy does not reduce risk; primary PTCA seems to reduce risk	≈1% (the posteromedial more frequent than the anterolateral papillary muscle)
Time course	Bimodal peak; within 24 hr and 3-5 days; range, 1-14 days	Bimodal peak; within 24 hr and 3-5 days; range, 1-14 days	Bimodal peak; within 24 hr and 3-5 days; range, 1-14 days
Clinical manifestations	Chest pain, shortness of breath, hypotension	Anginal, pleuritic, or pericardial chest pain; syncope; hypotension; arrhythmia; nausea; restlessness; hypotension; sudden death	Abrupt onset of shortness of breath and pulmonary edema; hypotension
Physical findings	Harsh holosystolic murmur, thrill (+), S ₃ , accentuated second heart sound, pulmonary edema, RV and LV failure, cardiogenic shock	Jugular venous distention (29% of patients), pulsus paradoxus (47%), electromechanical dissociation, cardiogenic shock	A soft murmur in some cases, no thrill, variable signs of RV overload, severe pulmonary edema, cardiogenic shock
Echocardiographic findings	Ventricular septal rupture, left-to-right shunt on color flow Doppler echocardiography through the ventricular septum, pattern of RV overload	>5 mm pericardial effusion not visualized in all cases; layered, high-acoustic echoes within the pericardium (blood clot); direct visualization of tear; signs of tamponade	Hypercontractile LV, torn papillary muscle or chordae tendineae, flail leaflet, severe mitral regurgitation on color flow Doppler echocardiography
Right-heart catheterization	Increase in oxygen saturation from the RA to RV, large v waves	Ventriculography insensitive, classic signs of tamponade not always present (equalization of diastolic pressures in the cardiac chambers)	No increase in oxygen saturation from the RA to RV, large v waves, * very high pulmonary capillary wedge pressure

Acute shock after MI:

- Think of mechanical complications
- They can happen whenever they want to
- Immediate ultrasound
- Typically a surgical emergency





Other etiologies of cardiogenic shock requiring specific therapy

Pulmonary embolism

Valvular disease

Arrhythmia

Tamponade

Myocarditis

Pulmonary hypertension





Other etiologies of cardiogenic shock requiring specific therapy

Pulmonary embolism

Valvular disease

Arrhythmia

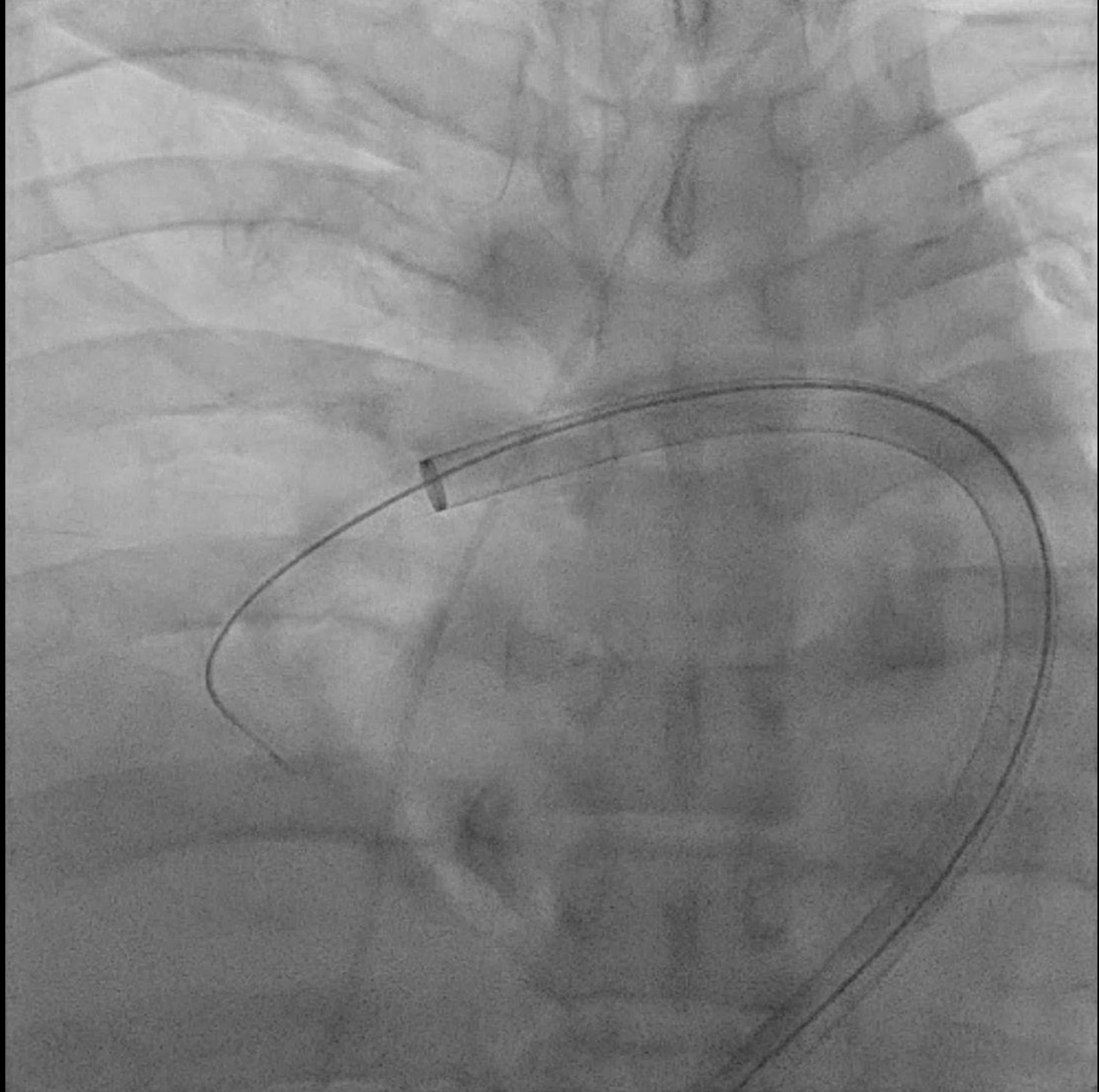
Tamponade

Myocarditis

Pulmonary hypertension



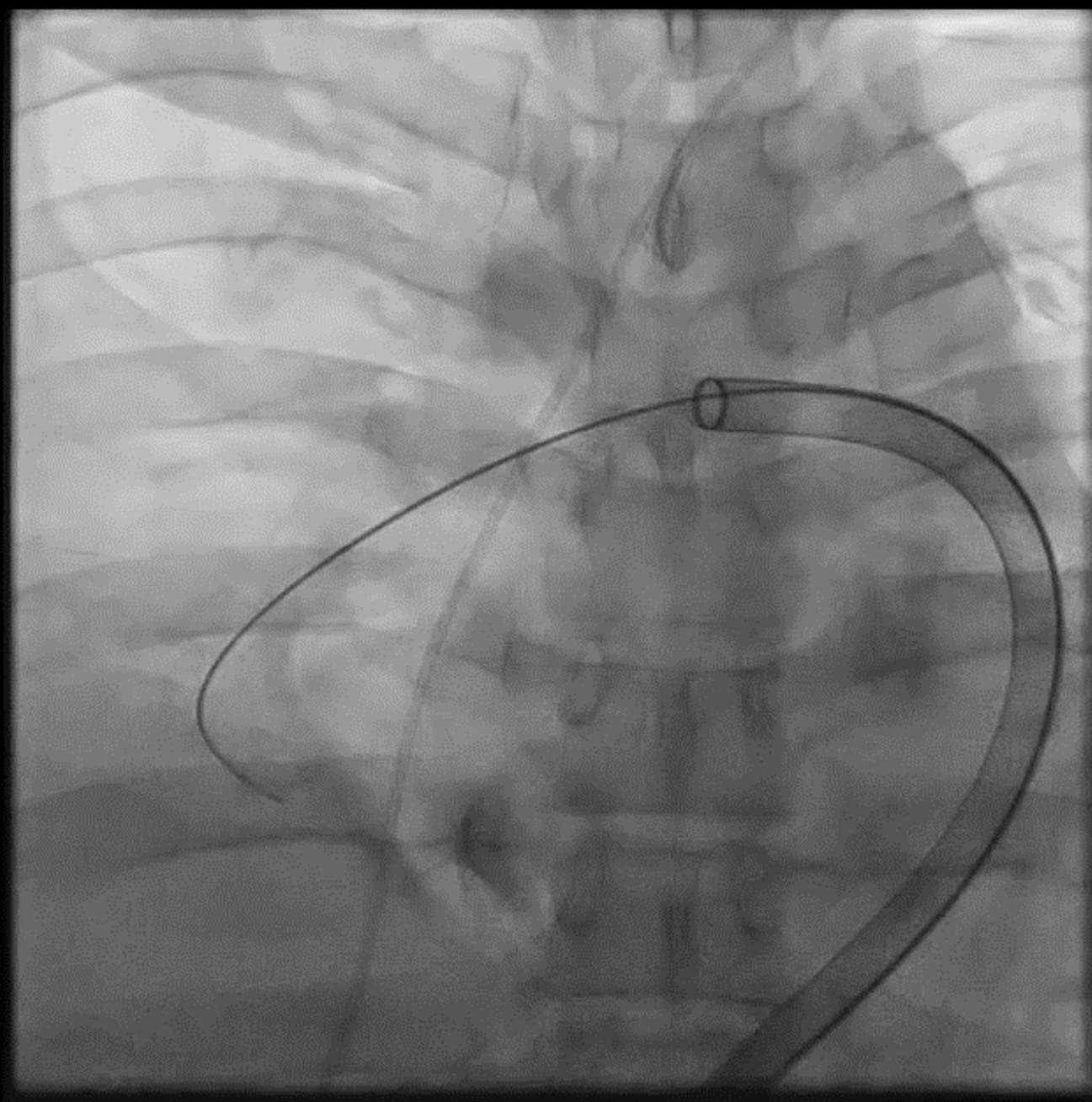






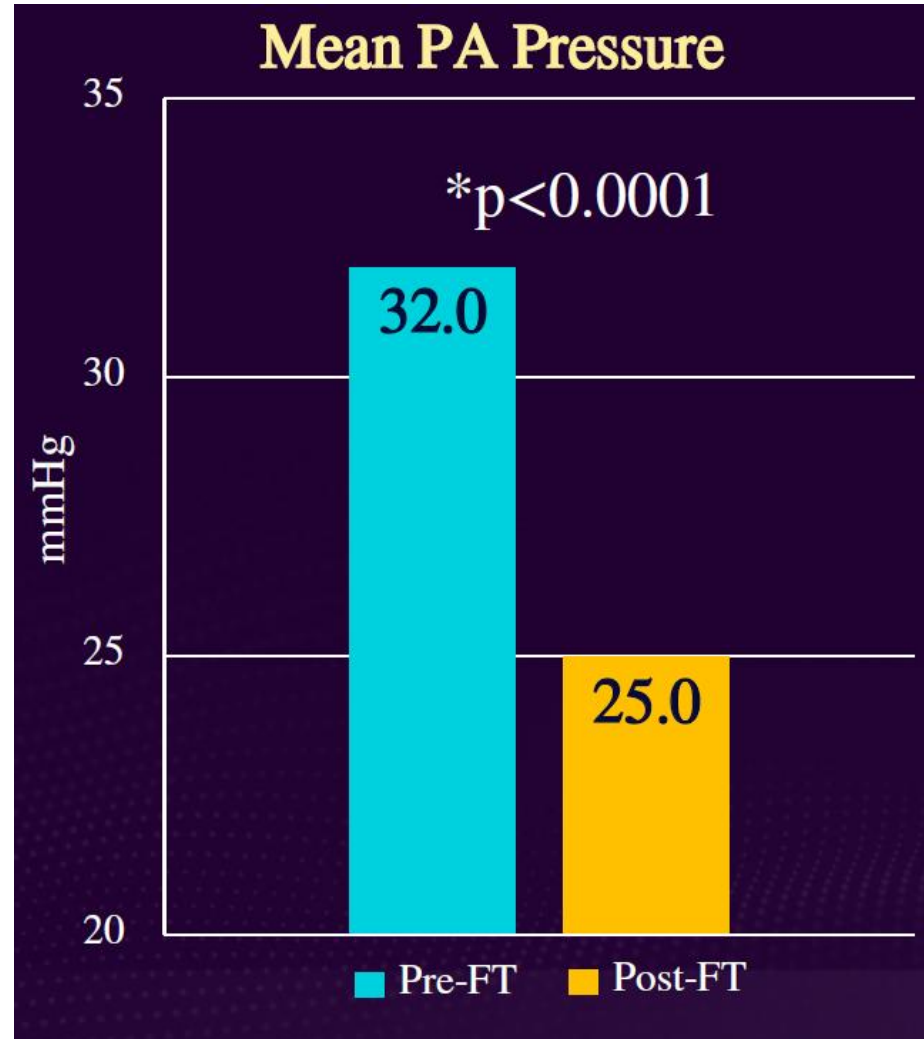
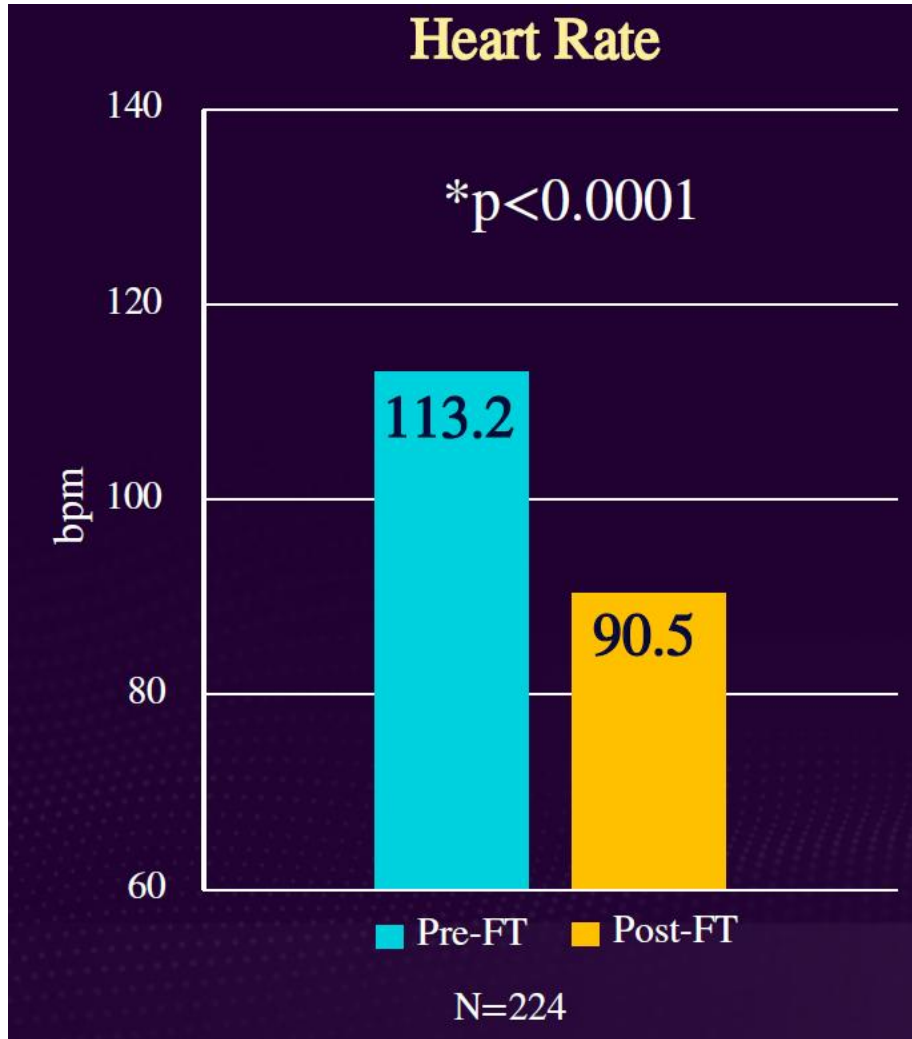
Left

Right





FLASH Registry





PEERLESS Trial

PLACEHOLDER – TO BE PRESENTED AT TCT ON OCTOBER 28TH

N=550 patients

Hemodynamically stable PE

Randomized 1:1 to thrombectomy with
FlowTrieve vs catheter-directed thrombolysis

PEP: Win ratio:

- (1) all-cause mortality
- (2) intracranial hemorrhage
- (3) major bleeding,
- (4) clinical deterioration and/or escalation to bailout
- (5) intensive care unit admission and length of stay





PEERLESS Trial

**PLACEHOLDER – TO BE PRESENTED AT TCT ON
OCTOBER 28TH**





PE Revascularization

At my hospital:

- A) There are no percutaneous or surgical options for PE revascularization**
- B) There are percutaneous revascularization options only**
- C) There is surgical revascularization only**
- D) There are both percutaneous and surgical options**
- E) Other**



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Complex Decisions

Shock Team

Chambers needing support (LV, RV, both)

Degree of support needed

Need for gas exchange

Vascular access considerations

Other anatomic considerations

Timing

Candidacy for long term therapies (VAD, transplant)





LV Support





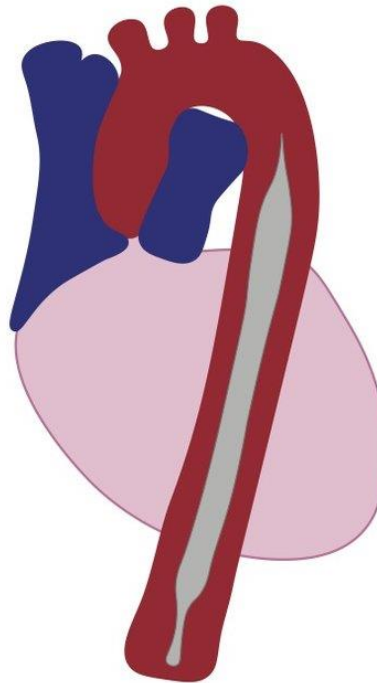
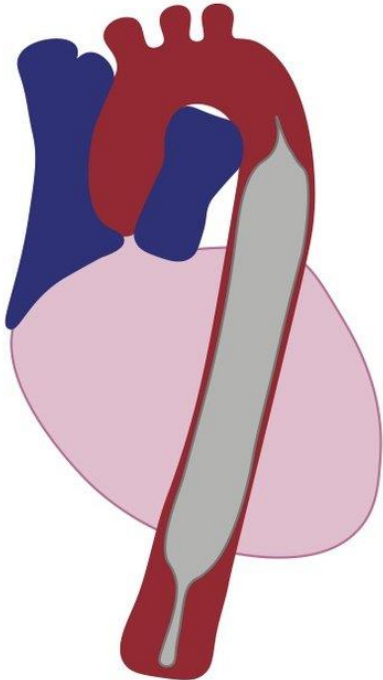
LV Support

For a patient with SCAI C/D cardiogenic shock from LV failure, the typical first line MCS at my hospital is:

- A) There are no MCS options**
- B) IABP**
- C) Impella CP**
- D) TandemHeart**
- E) ECMO**
- F) Other**



Intra-aortic balloon pump (IABP)

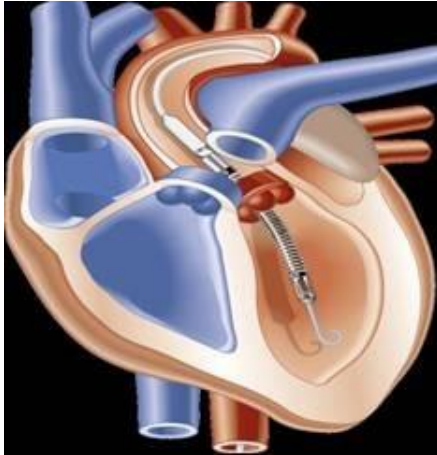


(+)
Rapid placement
Lower profile than
other MCS options
Axillary possible

(-)
Minimal support



Impella CP



(+)

Good support (3.5 L/min)

Typically rapid placement

Unloads LV

Axillary/transcaval possible

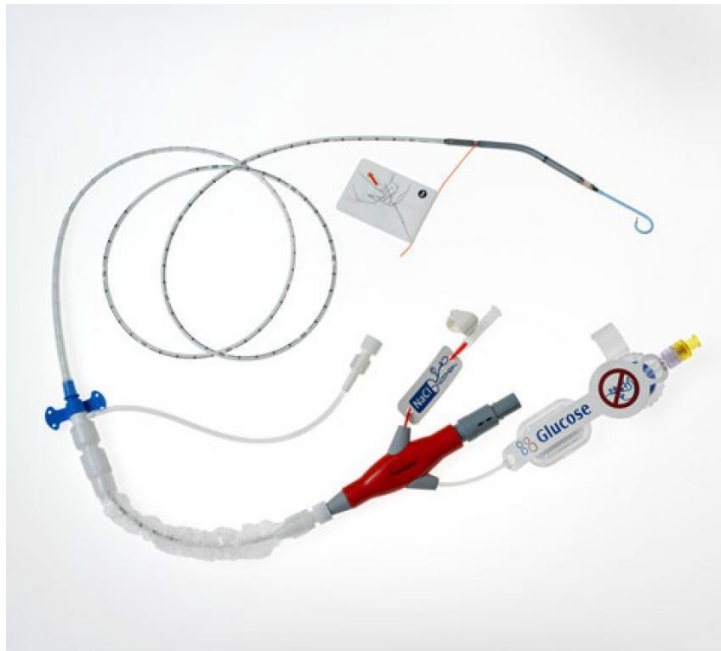
(-)

Migrates

Thrombocytopenia/hemolysis

Vascular injury

Note: Impella 5.5 also available (ax/transAo)





ORIGINAL ARTICLE

Microaxial Flow Pump or Standard Care in Infarct-Related Cardiogenic Shock

J.E. Møller, T. Engstrøm, L.O. Jensen, H. Eiskjær, N. Mangner, A. Polzin, P.C. Schulze, C. Skurk, P. Nordbeck, P. Clemmensen, V. Panoulas, S. Zimmer, A. Schäfer, N. Werner, M. Frydland, L. Holmvang, J. Kjærgaard, R. Sørensen, J. Lønborg, M.G. Lindholm, N.L.J. Udesen, A. Junker, H. Schmidt, C.J. Terkelsen, S. Christensen, E.H. Christiansen, A. Linke, F.J. Woitek, R. Westenfeld, S. Möbius-Winkler, K. Wachtell, H.B. Ravn, J.F. Lassen, S. Boesgaard, O. Gerke, and C. Hassager, for the DanGer Shock Investigators*



DanGer Shock

N=360 patients with STEMI complicated by shock

Randomized 1:1 to Impella CP vs standard care

PEP: Death from any cause at 180 days

A couple important points:

- Exclusions for: comatose after OHCA; overt RV failure
- Rando occurred before or after PCI
- Impella to be placed immediately after rando
- Impella at highest possible performance level for 48 hours





DanGer Shock

Table 1. Demographic and Clinical Characteristics of the Patients at Baseline and Timing of Randomization.*

Characteristic	Microaxial Flow Pump plus Standard Care (N = 179)	Standard Care Alone (N = 176)
Median age (IQR) — yr	67 (58–76)	69 (61–76)
Male sex — no. (%)	142 (79.3)	139 (79.0)
Median systolic blood pressure (IQR) — mm Hg	84 (72–91)	82 (72–91)
Median of the mean arterial blood pressure (IQR) — mm Hg	63 (55–72)	64 (55–73)
Median heart rate (IQR) — beats/min	94 (77–110)	95 (76–111)
Median arterial lactate level (IQR) — mmol/liter	4.6 (3.4–7.1)	4.5 (3.2–6.9)
Median left ventricular ejection fraction (IQR) — %	25 (20–31)	25 (15–30)
Resuscitation before randomization — no. (%)	39 (21.8)	33 (18.8)
Intubation before randomization — no. (%)	35 (19.6)	28 (15.9)
Transfer from outside hospital — no. (%)	51 (28.5)	48 (27.3)
Anterior myocardial infarction — no. (%)	126 (70.4)	129 (73.3)
SCAI-CSWG stage at admission — no. (%)†		
C	100 (55.9)	97 (55.1)
D	51 (28.5)	50 (28.4)
E	28 (15.6)	29 (16.5)



DanGer Shock

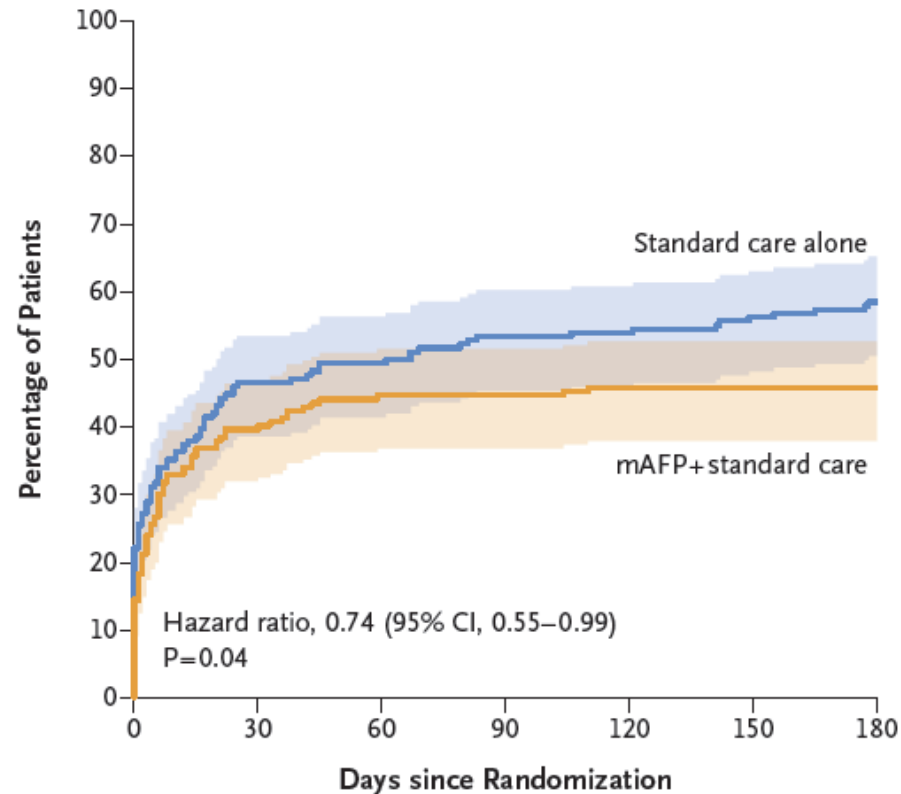
Table 2. In-Hospital Management of Cardiogenic Shock.*

Management	Microaxial Flow Pump plus Standard Care (N = 179)	Standard Care Alone (N = 176)
Mechanical circulatory support		
Placement of Impella CP device — no. (%)†	170 (95.0)	3 (1.7)
Randomization occurred before PCI and microaxial flow pump placed before PCI — no./total no. (%)	84/99 (84.8)	3/3 (100)
Median time from randomization to placement of microaxial flow pump (IQR) — min	14 (8–29)	15 (8–31)
Median duration of microaxial flow pump support (IQR) — hr	59 (30–87)	60 (31–92)
Mechanical hemolysis — no./total no. (%)	21/170 (12.4)	1/3 (33.3)
Device malfunction — no./total no. (%)‡	2/170 (1.2)	1/3 (33.3)
Successful weaning from microaxial flow pump — no./ total no. (%)	138/170 (81.2)	1/3 (33.3)
Escalation to additional mechanical circulatory support		
Placement of Impella 5.0 device — no. (%)	7 (3.9)	5 (2.8)
Placement of Impella CP for venting during venoarterial ECMO therapy — no. (%)	0	4 (2.3)
Placement of Impella 2.5 device — no. (%)	0	1 (0.6)
Placement of Impella RP device — no. (%)	0	0
Venoarterial ECMO — no. (%)	21 (11.7)	33 (18.8)
Median time from randomization to placement of venoarterial ECMO (IQR) — hr	14 (4–54)	2 (1–5)
Placement of permanent LVAD — no. (%)	10 (5.6)	4 (2.3)
Any escalation to additional mechanical circulatory support — no. (%)	28 (15.6)§	37 (21.0)¶



DanGer Shock

A Death from Any Cause



No. at Risk

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

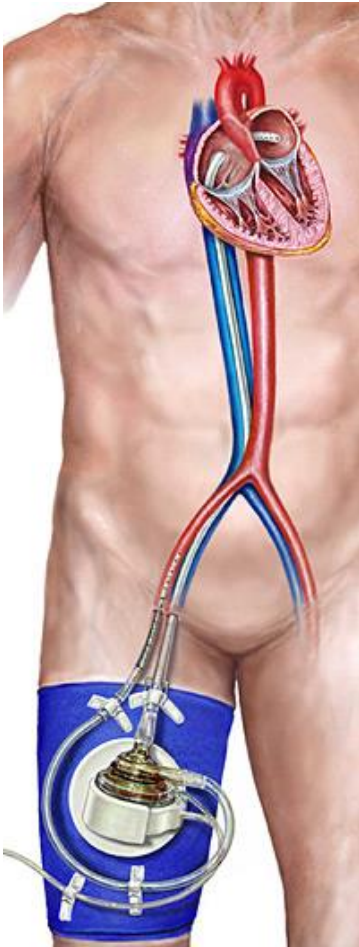


DanGer Shock

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)

TandemHeart



(+)

Robust support (4-5 L/min)

Possible to add gas exchange to circuit

Migration is unusual

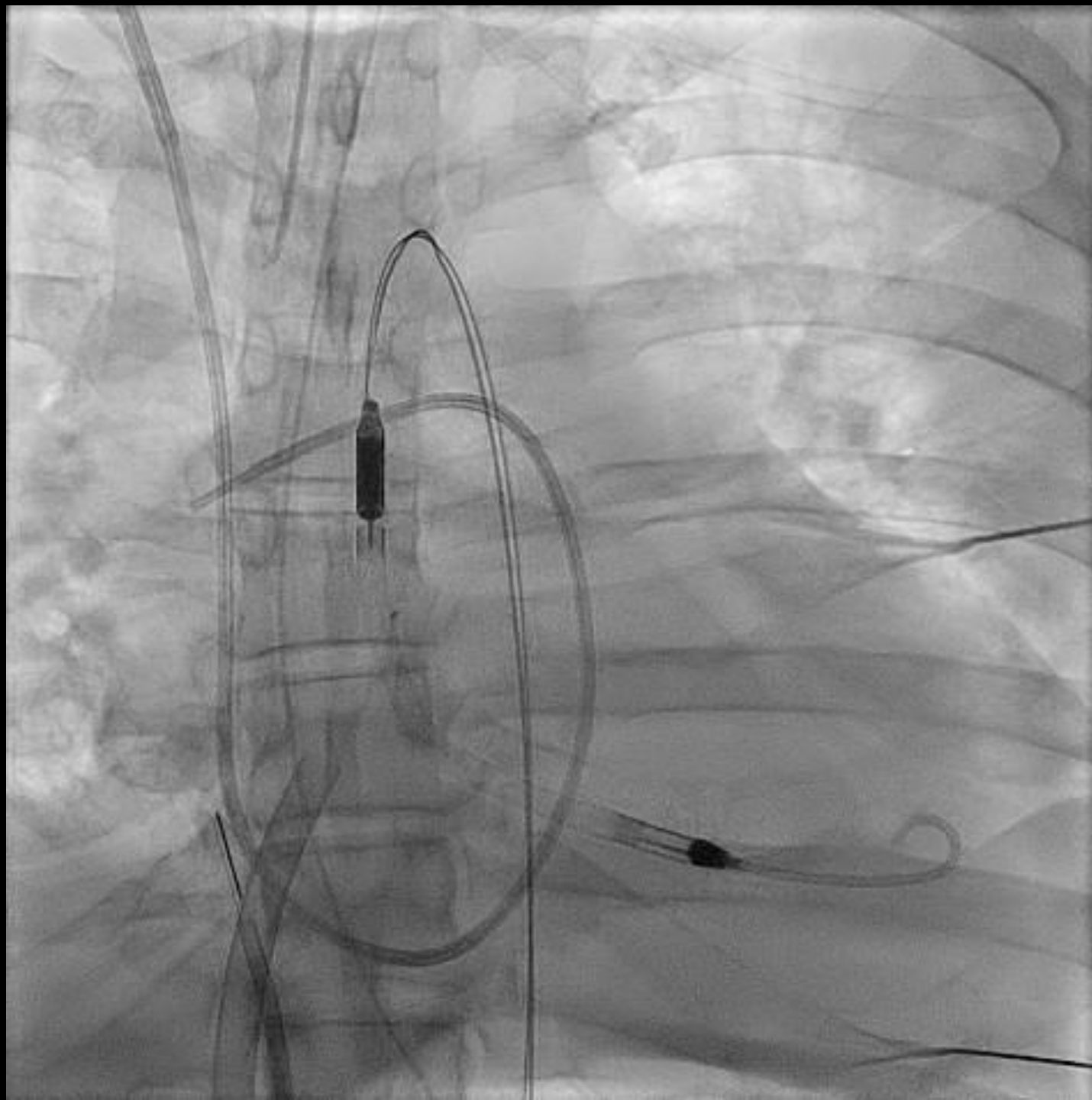
(-)

Limited availability

Requires transeptal puncture

Imperfect LV unloading

Vascular injury





RV Support





Impella RP Flex

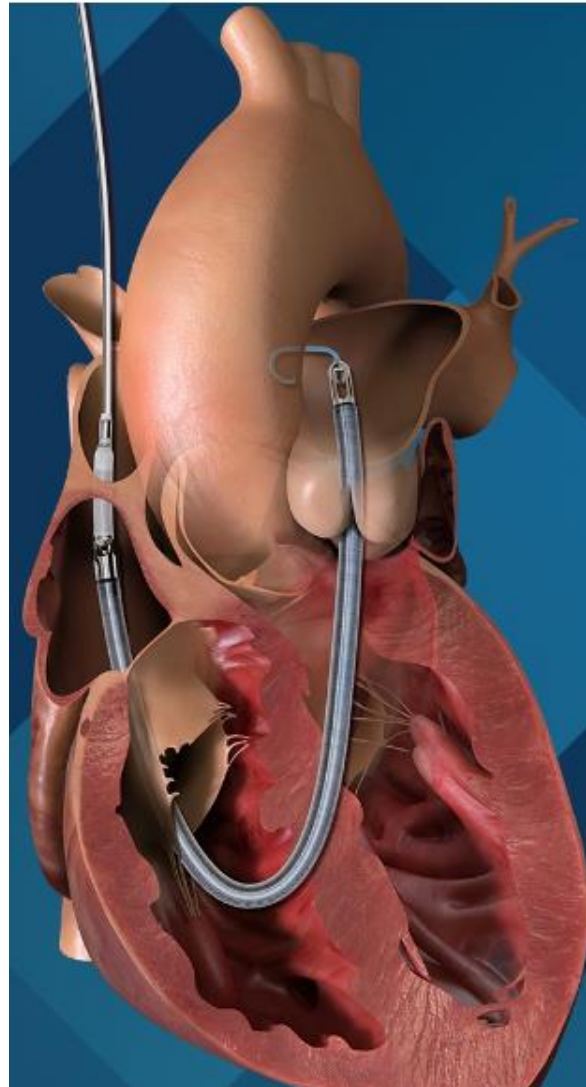
(+)

4 L/min

Typically fast
placement

(-)

Migrates
Thrombocytopenia
/hemolysis



RAO 1°
CAUD 0°
FD 17.0 inch



0:53
16:24:29

12
9



Tandem RVAD

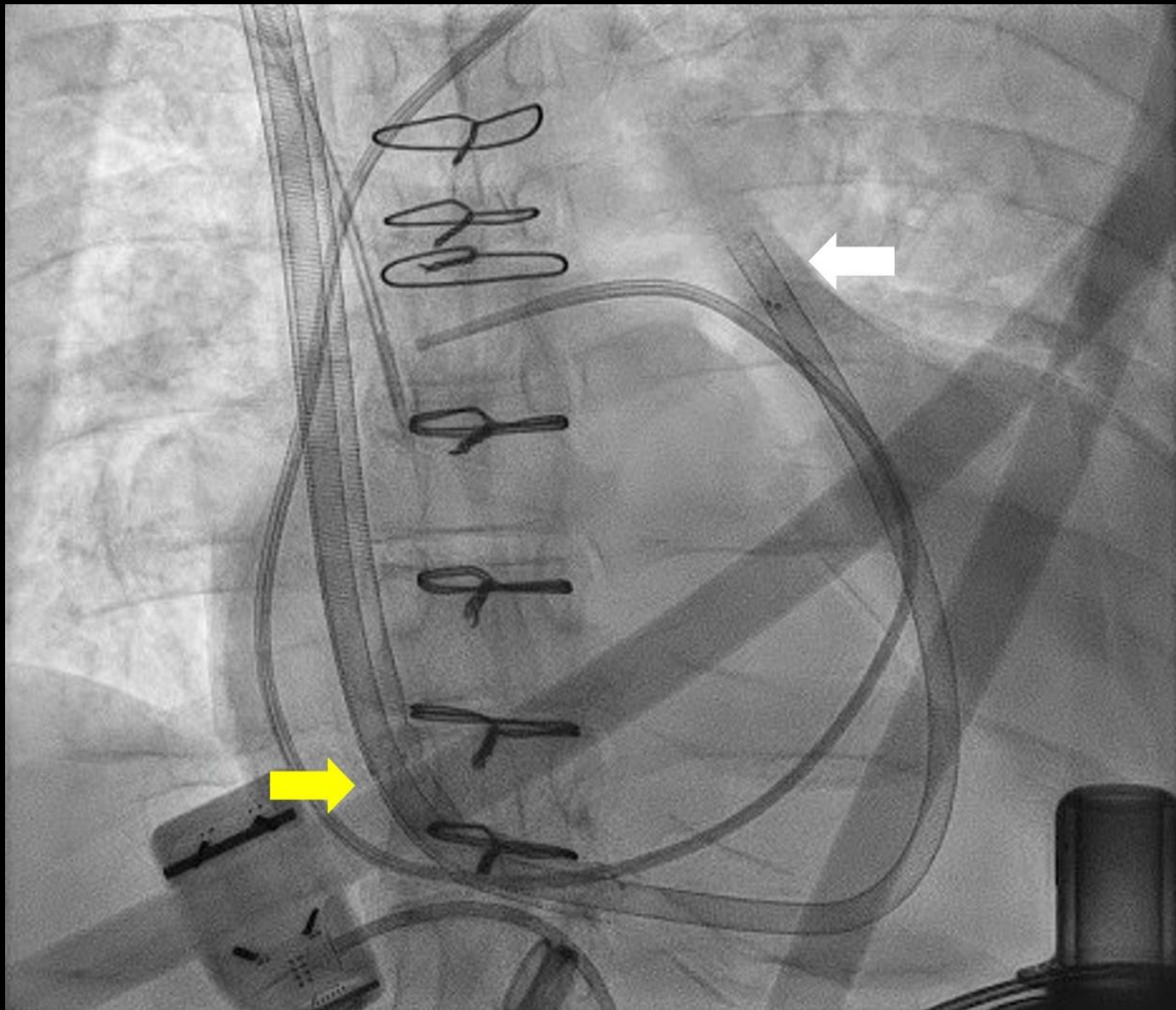
(+)

- 5+ L/min
- Typically fast placement
- Can add oxygenator
- If pair with TandemHeart LVAD and gas exchanger, have full ECLS in place
- Flexible access

(-)

- Larger access (28-31 Fr)
- Need to de-air circuit







Biventricular Support

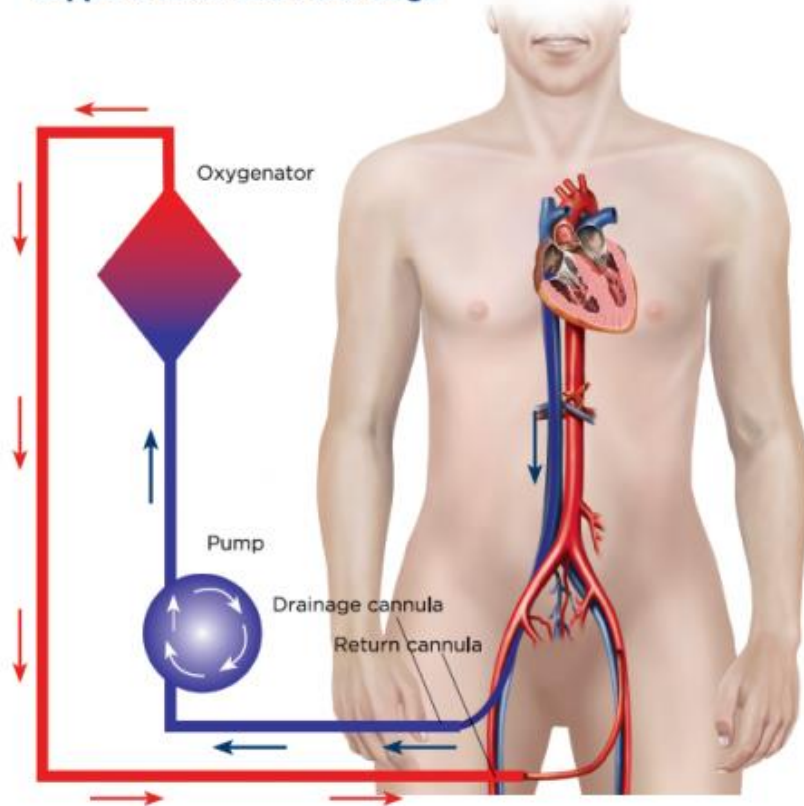




Extracorporeal Membrane Oxygenation (ECMO)

Veno-arterial (VA) ECMO

supports both heart and lungs



(+)

Full cardiopulmonary bypass
(Up to 6 L/min)

RV support

VT/VF tolerated

(-)

May require LV vent

Vascular injury

Limited availability



ECMO in ACS c/b shock

- N=420 patients
- Acute MI w cardiogenic shock
- Randomized to early ECMO vs standard care
- PEP: Death through 30d

Table 1. Characteristics of the Patients at Baseline.*

Characteristic	ECLS (N=209)	Control (N=208)
Median age (IQR) — yr	62 (56–69)	63 (57–71)
Signs of impaired organ perfusion — no. (%)		
Altered mental status	200 (95.7)	198 (95.2)
Cold, clammy skin and limbs	202 (96.7)	204 (98.1)
Oliguria	150 (71.8)	150 (72.1)
Resuscitation before randomization — no. (%)	162 (77.5)	162 (77.9)
Median time until return of spontaneous circulation during longest continuous resuscitation (IQR) — min	20 (10–25)	20 (12–28)
No. of diseased vessels — no./total no. (%)		
1	71/203 (35.0)	63/200 (31.5)
2	71/203 (35.0)	53/200 (26.5)
3	61/203 (30.0)	84/200 (42.0)
Infarct-related artery — no./total no. (%)		
Left anterior descending	95/203 (46.8)	97/200 (48.5)
Left circumflex	36/203 (17.7)	35/200 (17.5)
Right coronary	52/203 (25.6)	48/200 (24.0)
Left main	20/203 (9.9)	20/200 (10.0)
Median left ventricular ejection fraction (IQR) — %	30 (20–35)	30 (20–40)
Laboratory values on admission		
Median pH (IQR)	7.2 (7.1–7.3)	7.2 (7.1–7.3)
Median lactate (IQR) — mmol/liter	6.8 (4.5–9.6)	6.9 (4.6–10.0)
Median creatinine (IQR) — mg/dl	1.2 (1.0–1.5)	1.3 (1.1–1.6)
Median high-sensitivity cardiac troponin T (IQR) — ng/liter	1540 (232–6630)	987 (173–5700)



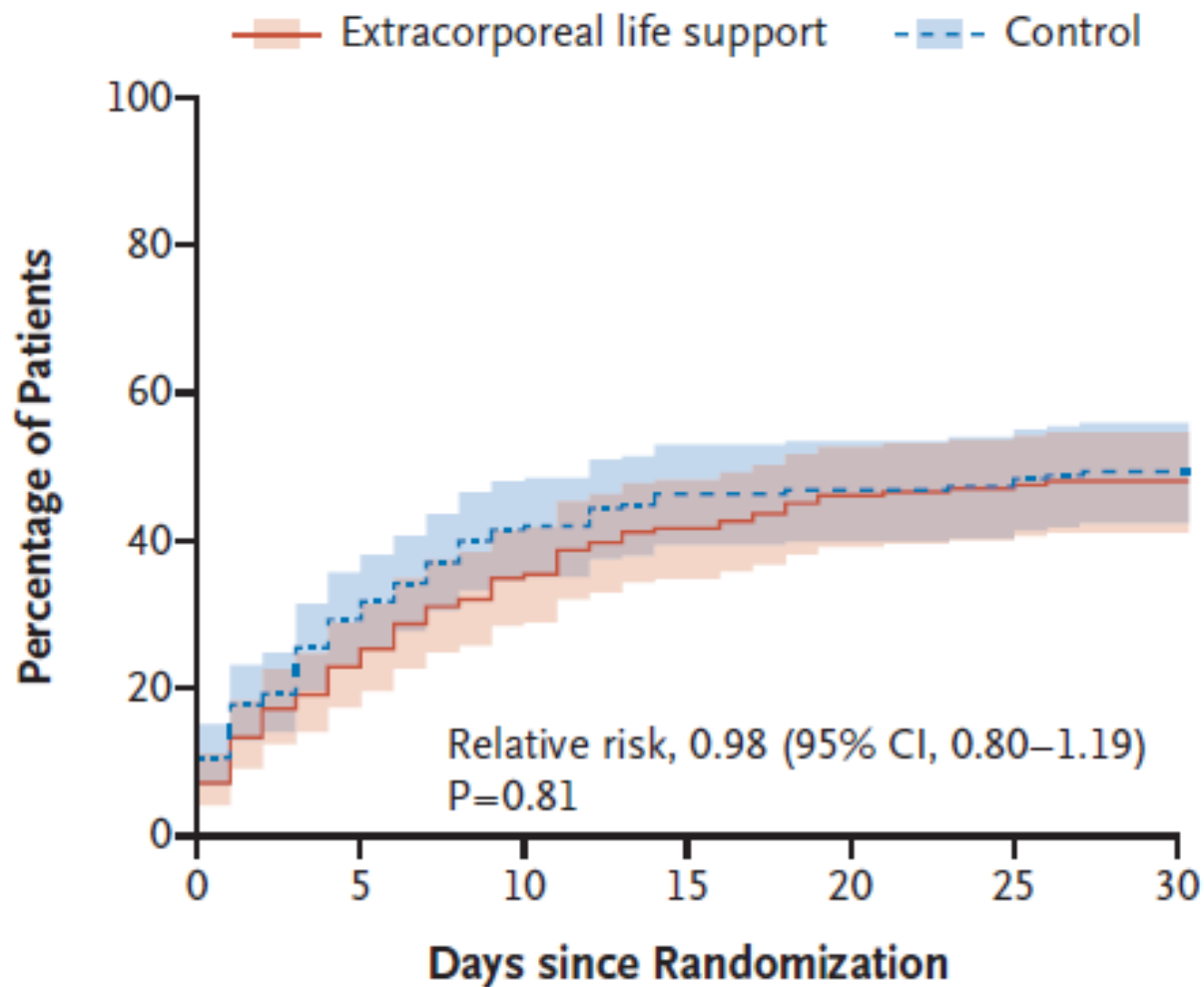
ECMO in ACS c/b shock

Table 2. Treatment.*

Characteristic	ECLS (N=209)	Control (N=208)
ECLS therapy — no. (%)	192 (91.9)	26 (12.5)
Initiation in catheterization laboratory		
Before revascularization	42/192 (21.9)	4/26 (15.4)
During revascularization	50/192 (26.0)	8/26 (30.8)
After revascularization	100/192 (52.1)	7/26 (26.9)
Initiation after catheterization laboratory		
<24 hr	0/192	3/26 (11.5)
≥24 hr	0/192	4/26 (15.4)
Median duration of ECLS therapy (IQR) — days	2.7 (1.5–4.8)	2.7 (2.2–3.8)
Peripheral antegrade perfusion sheath during ECLS therapy — no./total no. (%)	183/192 (95.3)	16/19 (84.2)
Median diameter of arterial cannula (IQR) — French size	17 (15–18)	17 (15–17)
Active left ventricular unloading during ECLS therapy — no./total no. (%)	11/191 (5.8)	6/19 (31.6)
Other mechanical circulatory support in patients without ECLS — no./total no. (%)	0/17	28/182 (15.4)
Intraaortic balloon pump	—	1/28 (3.6)
Impella 2.5	—	1/28 (3.6)
Impella CP	—	24/28 (85.7)
Impella 5.0	—	1/28 (3.6)
Impella 5.5	—	1/28 (3.6)
Permanent left ventricular assist device — no./total no. (%)	1 (0.5)	1 (0.5)
Target temperature management — no./total no. (%)	82/209 (39.2)	109/208 (52.4)



ECMO in ACS c/b shock





ECMO in ACS c/b shock

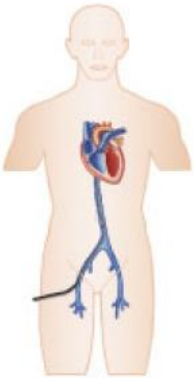


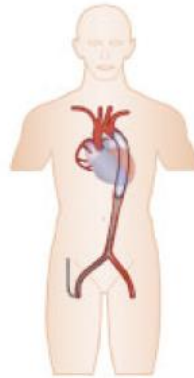
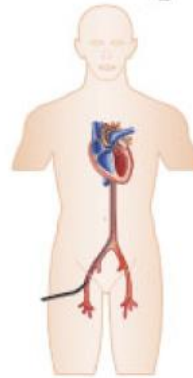


Table 3. Clinical Outcomes at 30 Days.

Outcome	ECLS (N=209)	Control (N=208)	Effect Size (95% CI)*
Safety outcomes			
Peripheral ischemic vascular complications warranting surgical or interventional therapy — no. (%)	23 (11.0)	8 (3.8)	Relative risk, 2.86 (1.31 to 6.25)
Stroke or systemic embolization — no. (%)	8 (3.8)	6 (2.9)	Relative risk, 1.33 (0.47 to 3.76)
Moderate or severe bleeding — no. (%)§	49 (23.4)	20 (9.6)	Relative risk, 2.44 (1.50 to 3.95)

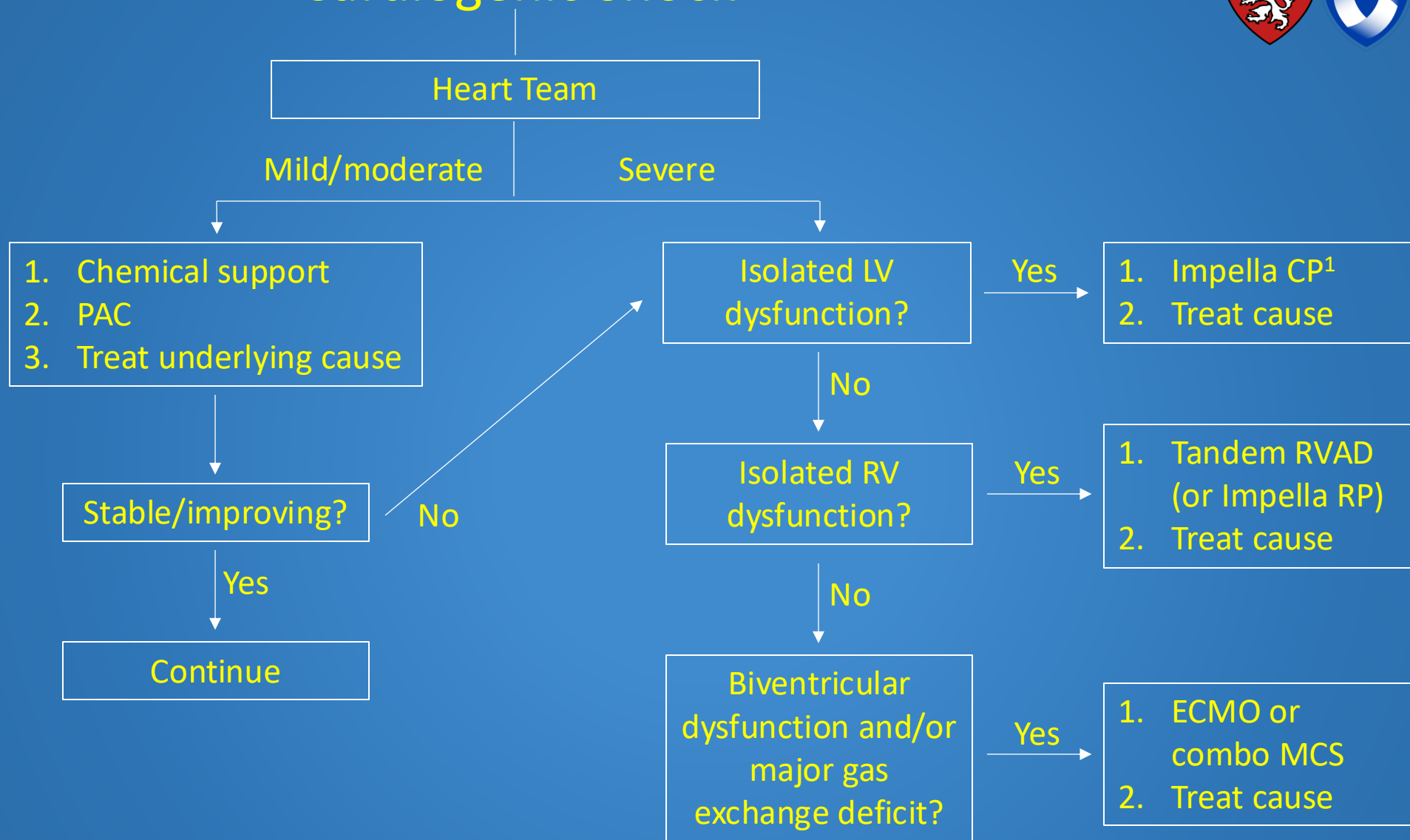
ECLS-SHOCK Take Home Points

- Succeeded in enrolling sick patients (3/4 with cardiac arrest, median lactate ~ 7)
- Hard population to study → parachutes (>25% in control arm got MCS)
- No mortality benefit to routine early ECMO in pts with clinical equipoise
- MCS comes at a cost

MCS Overview

	Right ventricular support			Left ventricular support			
	a) Impella RP	b) TandemHeart RA-PA	c) VA-ECMO	d) IABP	e) Impella	f) TandemHeart	g) iVAC 2L
							
Flow:	max. 4.0 L	max. 4.0 L	max. 7.0 L		2.5-5.0 L	max. 4.0 L	max. 2.8 L
Pump speed:	33.000 rpm	max. 7.500 rpm	max. 5000 rpm		max. 51.000 rpm	max. 7.500 rpm	40 ml/beat
Cannula size:	22 F	29 F	14-19 F arterial 17-21F venous	7-8 F	12-14 F	12-19 F arterial 21F venous	17 F
Insertion/ Placement	Femoral vein	Internal jugular vein	Femoral artery Femoral vein	Femoral artery	Femoral artery	Femoral artery Femoral vein for LA access	Femoral artery
LV Unloading	-	-	-	(+)	+-++	++	+
RV Unloading	+	+	++	-	-	-	-

Cardiogenic Shock



¹ECMO or TandemHeart if contraindication to Impella such as mechanical aortic valve or if Impella CP inadequate (may consider Impella 5.5)

Where are we going with this?

Temporary MCS

Recovery/
Explant

Durable
VAD

Transplant

Death



Boards-Style Question

A 67-year-old woman presented with anterior STEMI 18 hours after symptom onset. Given ongoing chest discomfort and resuscitated VT in the Emergency Department she underwent emergent LAD PCI with TIMI 2 flow at the end of the procedure. On day 3 she develops acute chest pain, hypotension, and dyspnea. Physical exam reveals tachypnea and cool extremities as well as a harsh systolic murmur which was not previously present.

What is the next best step in this patient's care?

- A) Place pulmonary artery catheter to measure RA and RV SpO₂
- B) Emergent coronary angiography for suspected stent thrombosis
- C) Emergent transthoracic echocardiogram with simultaneous consultation of Cardiac Surgery and Cardiac Catheterization Laboratory
- D) CT-PE





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- D) CT-PE





Take Home Points

Cardiogenic shock is associated with high mortality

Recognizing and classifying cardiogenic shock can be challenging, but is essential

Prompt revascularization is the critical therapy for acute MI with shock

Diverse causes of cardiogenic shock exist beyond acute MI, but are much less studied





Take Home Points

For cardiogenic shock caused by a treatable etiology, prompt etiology-specific therapy is essential

Supportive measures include inotropes, vasodilators, diuretics and mechanical circulatory support

Multidisciplinary decision-making facilitates rapid and appropriate initiation of directed supportive therapy





Thank you

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