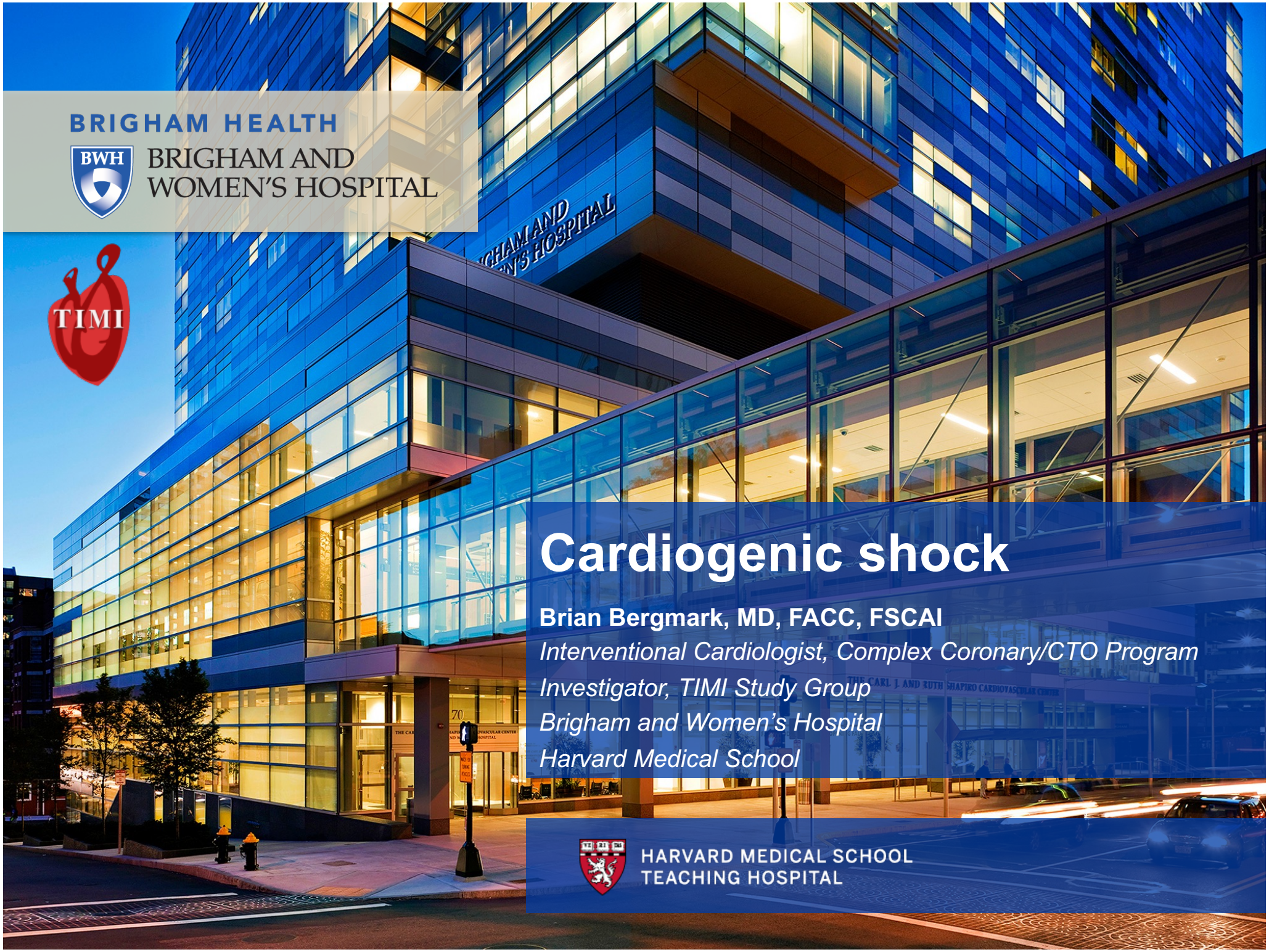
BRIGHAM HEALTH
BWH
BRIGHAM AND
WOMEN'S HOSPITAL
TIMI
Cardiogenic shock
Brian Bergmark, MD, FACC, FSCAI
Interventional Cardiologist, Complex Coronary/CTO Program
Investigator, TIMI Study Group
Brigham and Women's Hospital
Harvard Medical School
HARVARD MEDICAL SCHOOL
TEACHING HOSPITAL

Disclosures

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Numerous slides courtesy of/adapted from Dr. Erin Bohula

Case

55-year-old woman

- Type I diabetes
- Kidney/pancreas transplant in 1999
- Repeat kidney transplant 2010
- Progressive renal insufficiency
- Hypertension
- Asthma

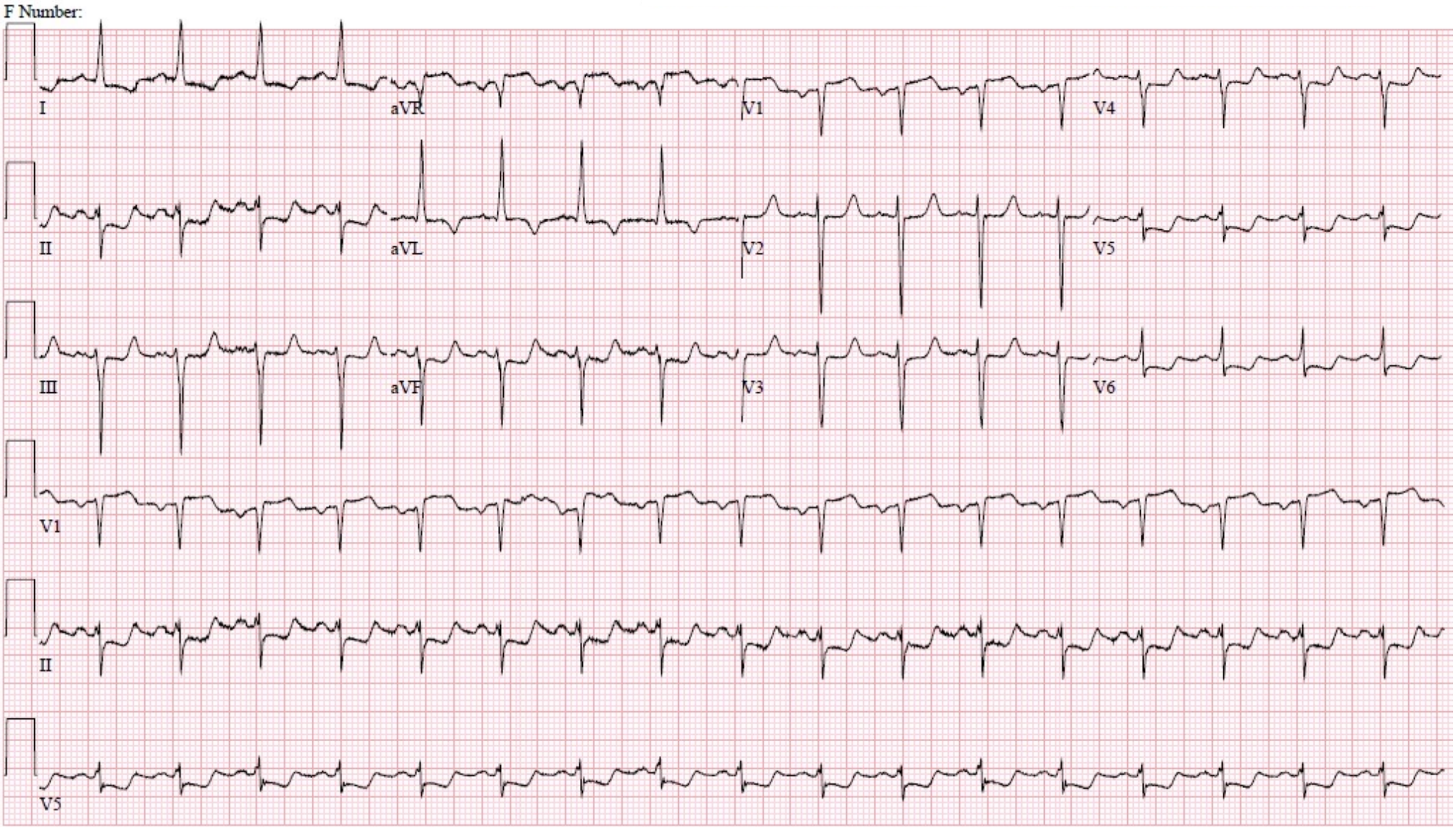
Presented to BWH ED with rest chest pain

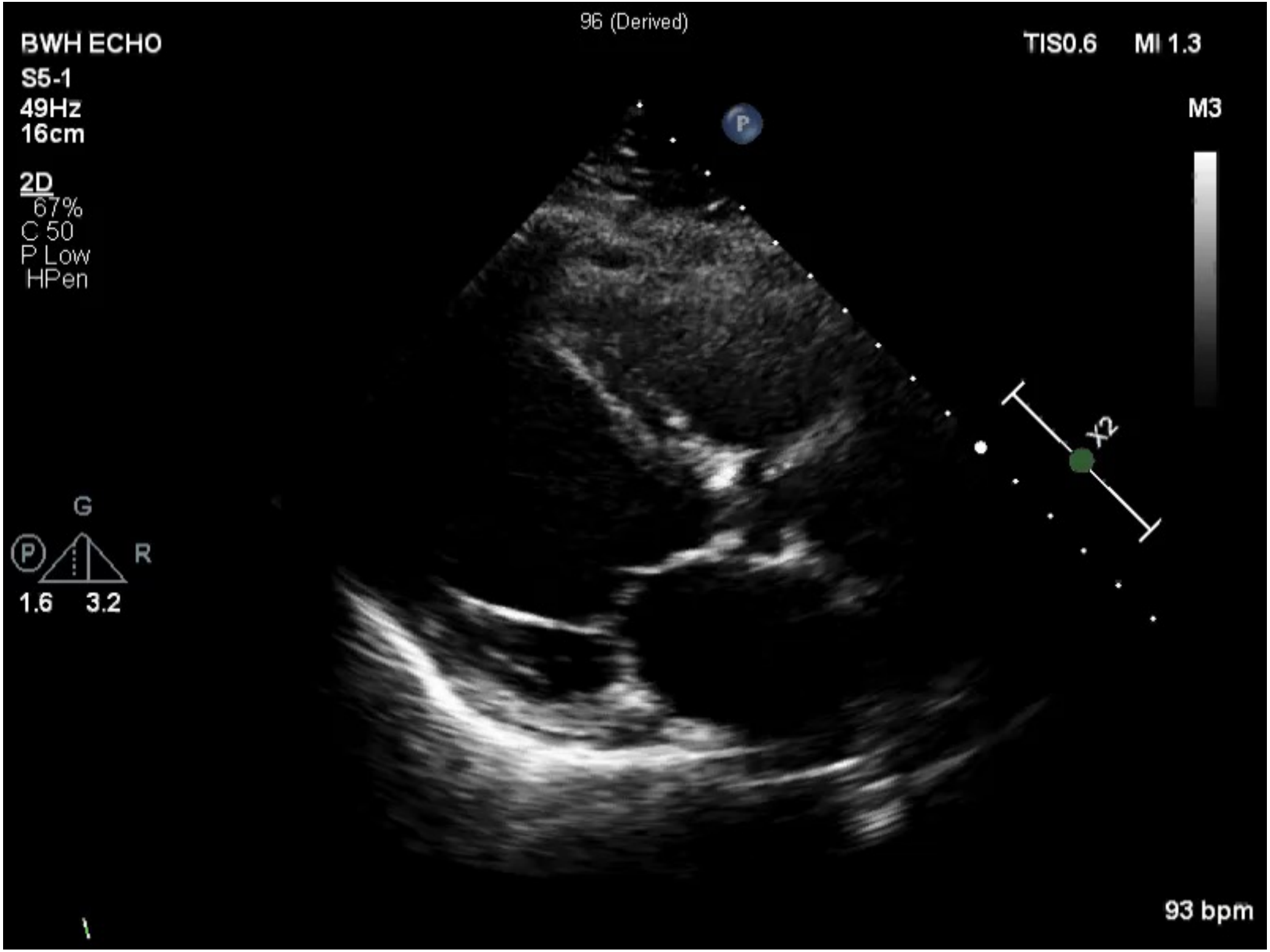
Case

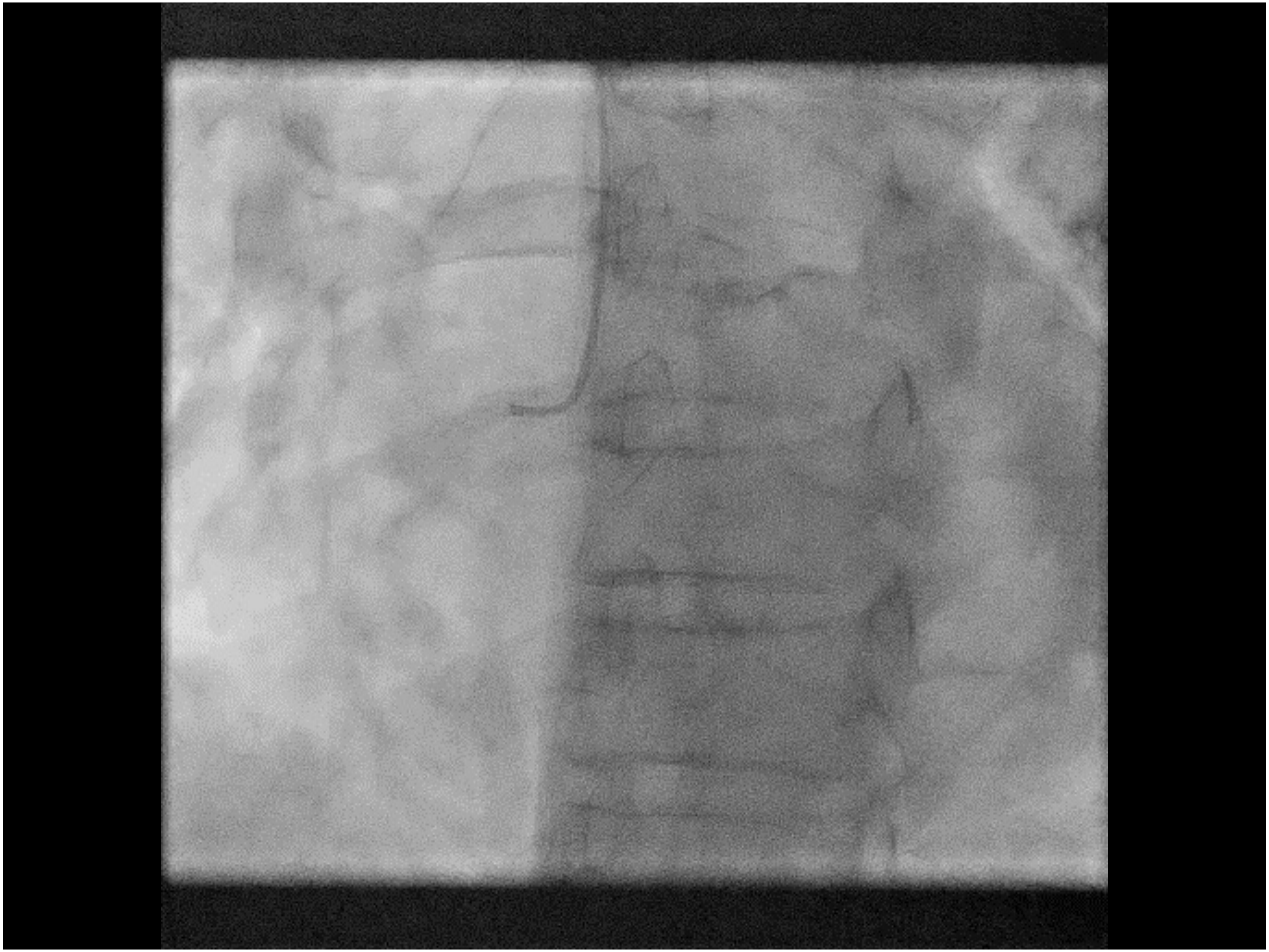
110BPM **80/50**mmHg **24**RPM **96%4LNC**

Cr	2.47 mg/dL
hsTnI	631 ng/L

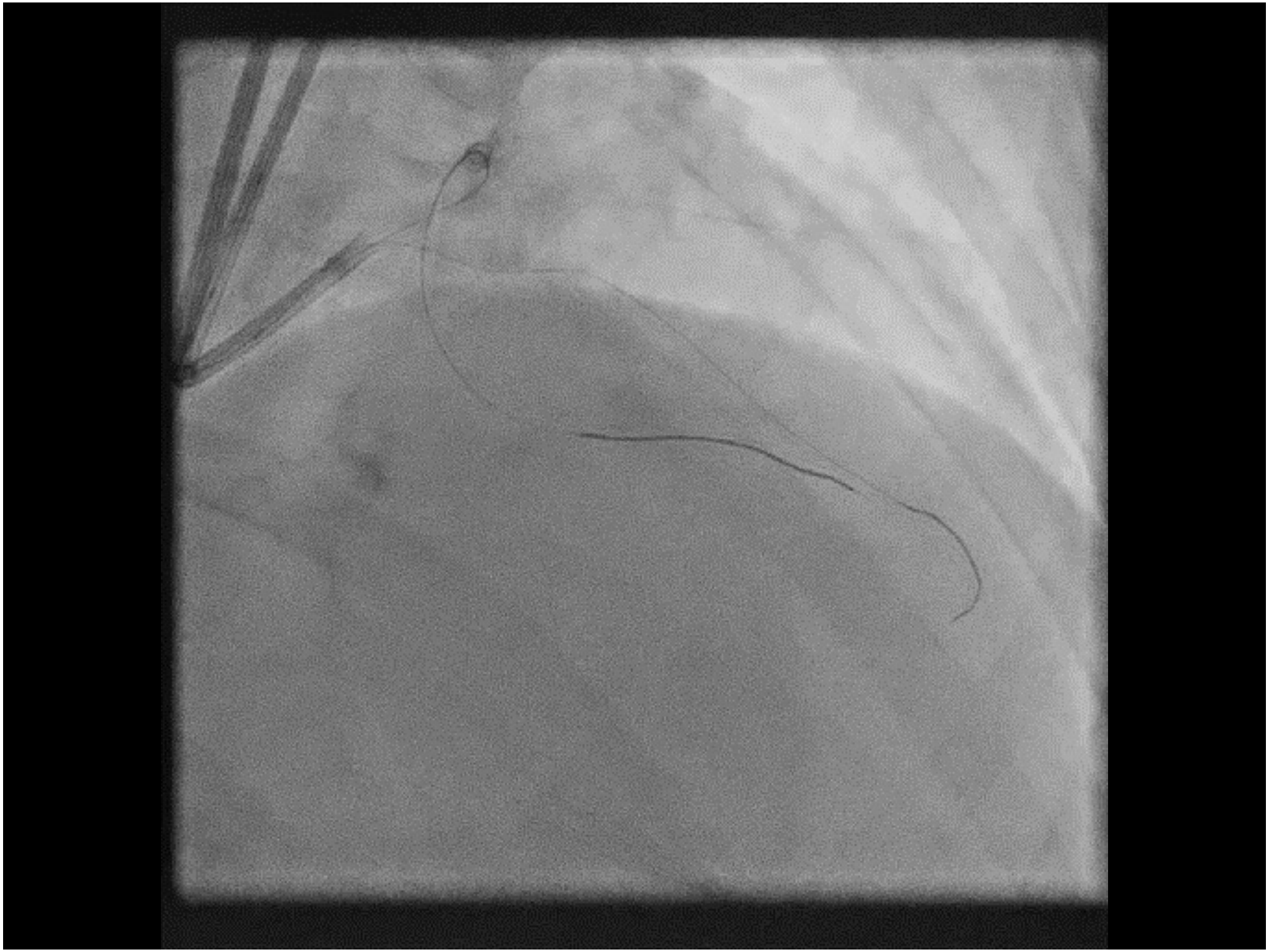
Case

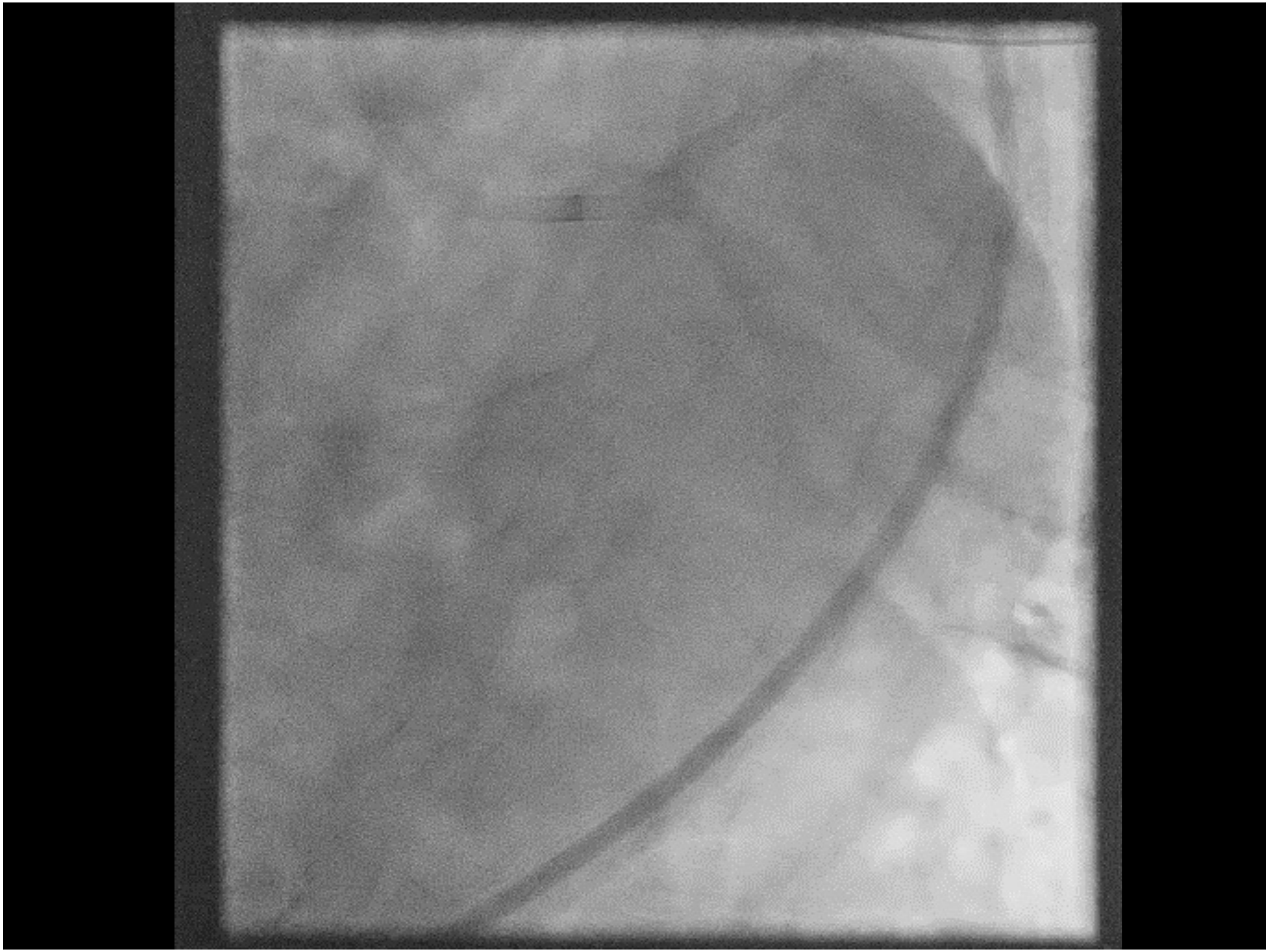



96 (Derived)
BWH ECHO
S5-1
49Hz
16cm
2D
67%
C 50
P Low
HPen
TIS0.6
MI 1.3
M3
P
X2
G
P
R
1.6
3.2
93 bpm

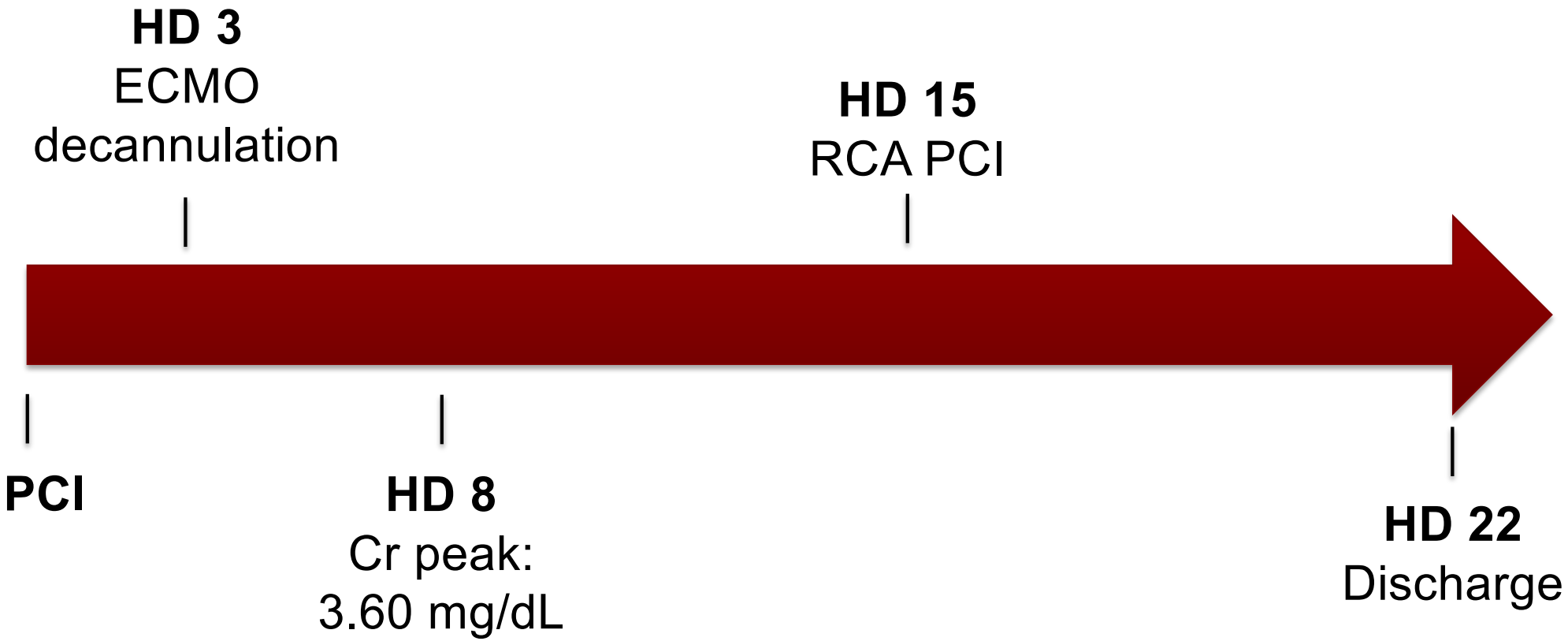



SUBTRACTION;FRAME_SELECTION (Derived)





Case



Most recent Cr 1.72 mg/dL


FR 50Hz
15cm
2D
44%
C 50
P Med
HPen
M3
G
P
R
1.4
2.8
JPEG
76 bpm

Outline

Epidemiology

Definition and diagnosis

Management

- Acute MI
- General supportive measures
- Mechanical circulatory support

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Epidemiology

Definition and diagnosis

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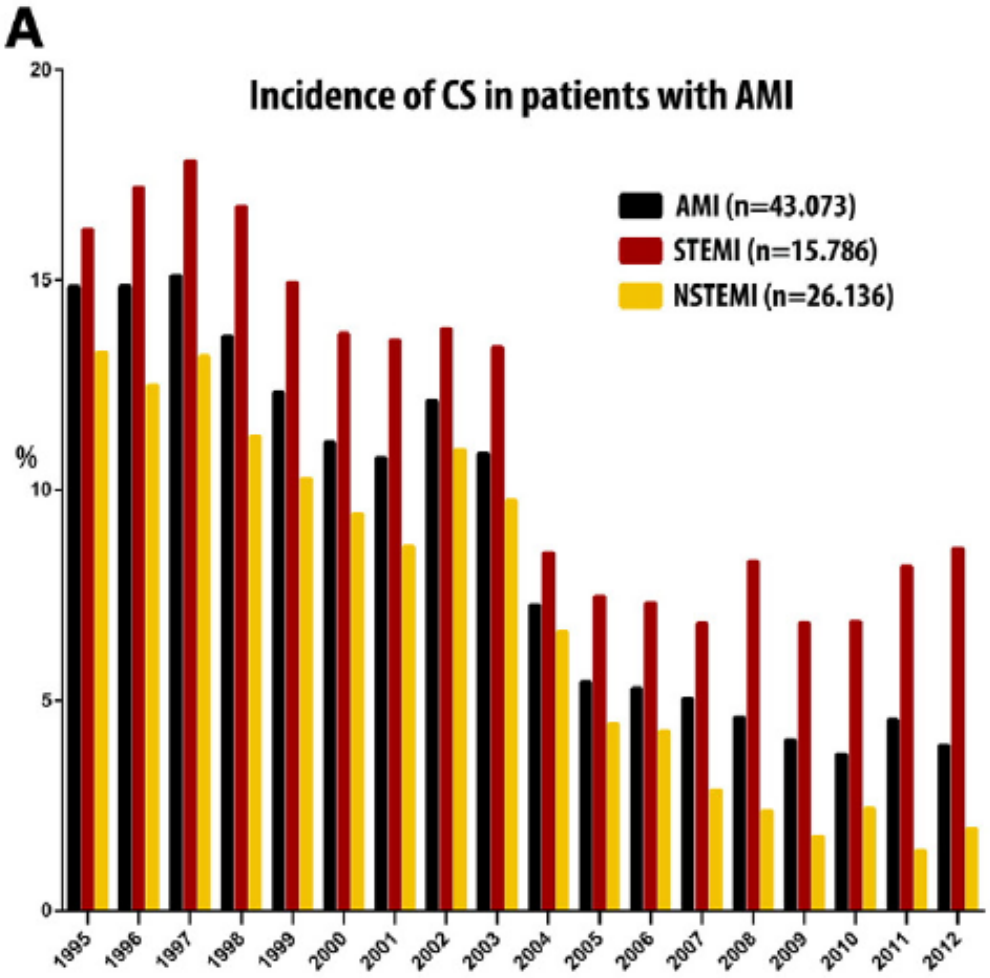
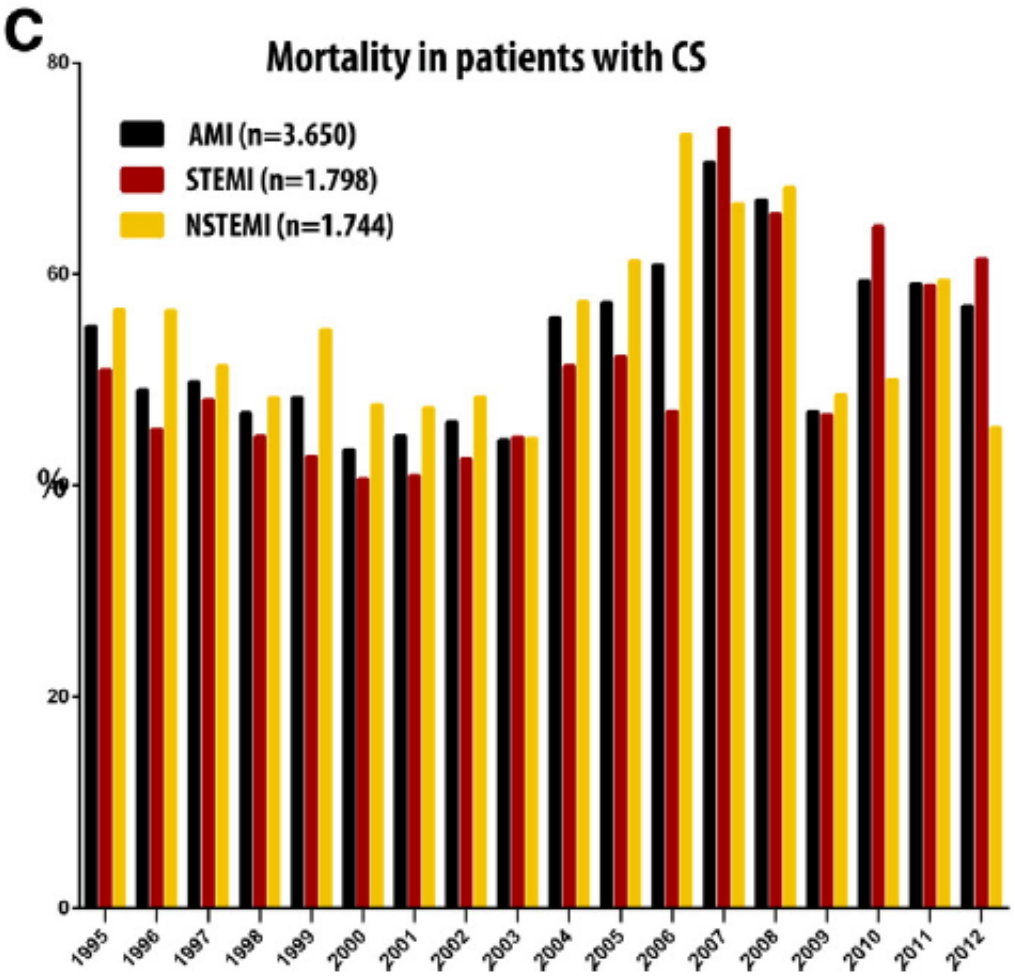
Epidemiology

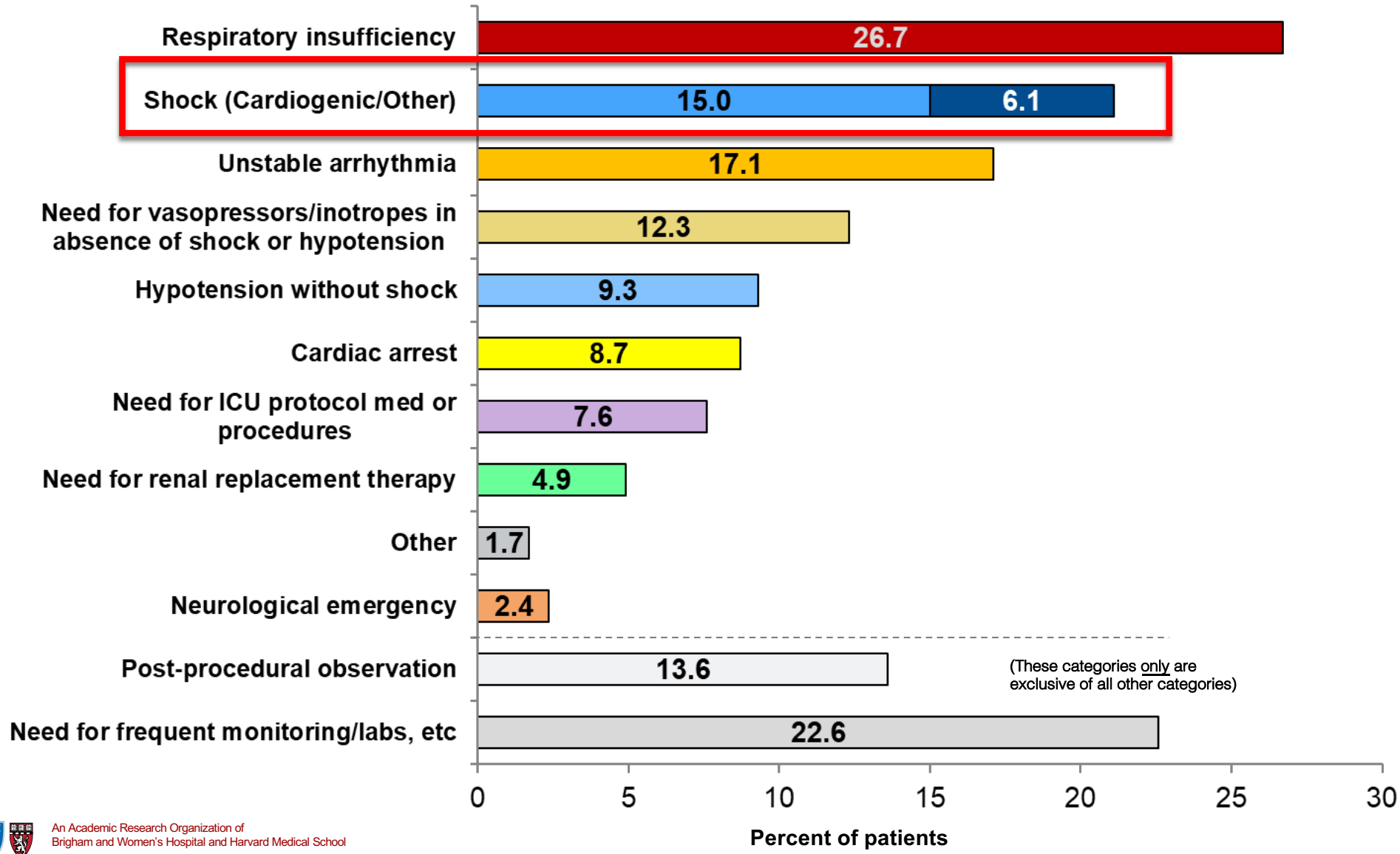




Patients are older and with more comorbidities

Redfors. Int J Cardiol. 2015.

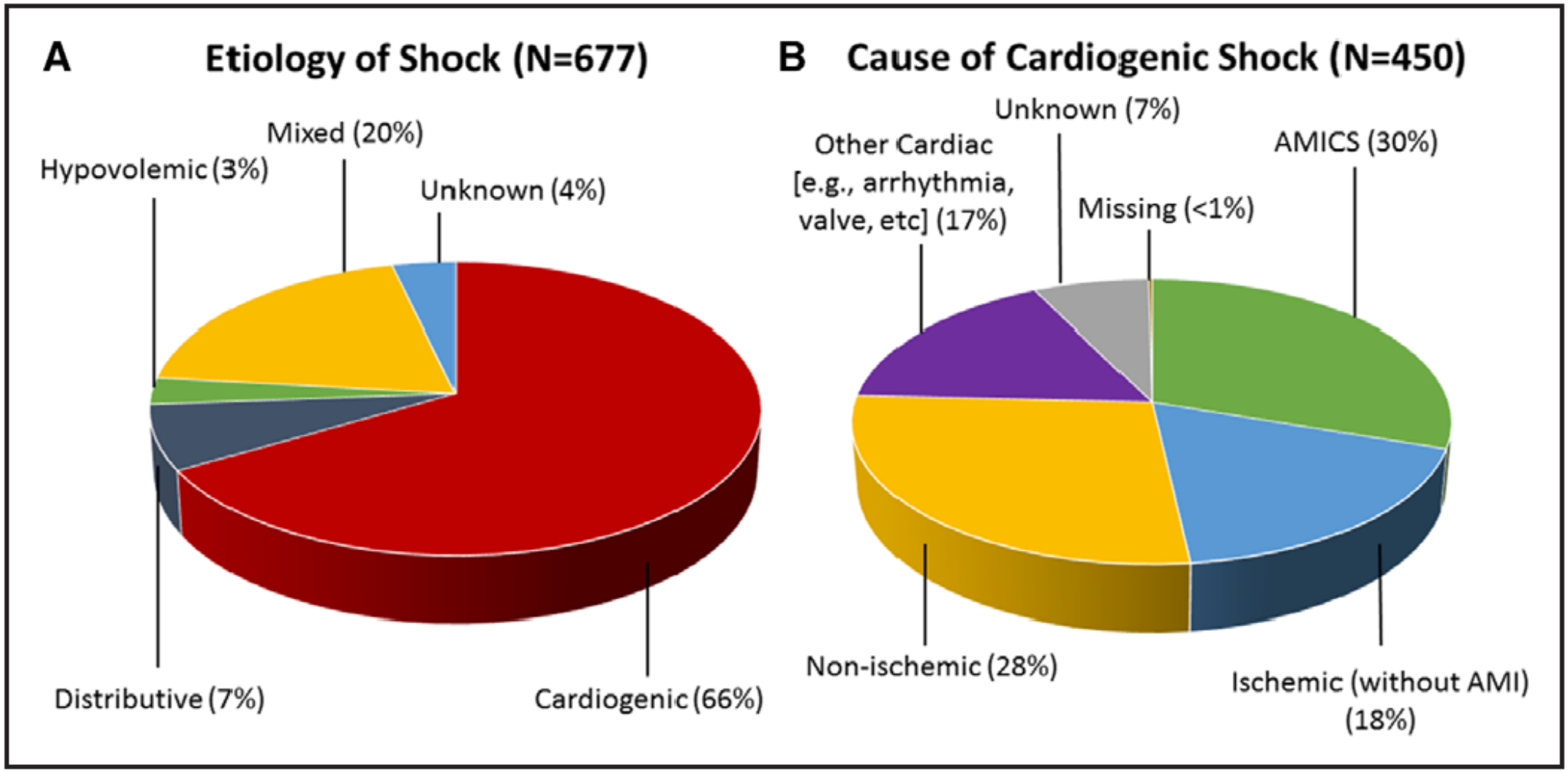
Indications for Intensive Care



An Academic Research Organization of
Brigham and Women's Hospital and Harvard Medical School

Bohula et al. JAMA Cardiology. 2019

Epidemiology



Berg. Circ Cardiovasc Qual/Outcomes. 2019.

Outline

Epidemiology

Definition and diagnosis

Management

- Acute MI
- General supportive measures
- Mechanical circulatory support

Shock

		Volume Status	
		Wet	**Dry**
Peripheral Circulation	**Cold**	Classic Cardiogenic Shock (↓CI; ↑SVRI; ↑PCWP)	Euvolemic Cardiogenic Shock (↓CI; ↑SVRI; ↔PCWP)
	Warm	Vasodilatory Cardiogenic Shock or Mixed Shock (↓CI; ↓/↔SVRI; ↑PCWP)	Vasodilatory Shock (Not Cardiogenic Shock) (↑CI; ↓SVRI; ↓PCWP)

Van Diepen. Circulation. 2017.

Cardiogenic Shock

Clinical Definition	SHOCK Trial[9]*	IABP-SHOCK II[1]†	ESC HF Guidelines[15]
Cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Support to maintain SBP ≥90 mm Hg AND End-organ hypoperfusion (urine output <30 mL/h or cool extremities) Hemodynamic criteria: CI of ≤2.2 $L \cdot min^{-1} \cdot m^{-2}$ AND PCWP ≥15 mm Hg	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Catecholamines to maintain SBP >90 mm Hg AND Clinical pulmonary congestion AND Impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output <30 mL/h, or lactate >2.0 mmol/L)	SBP <90 mm Hg with adequate volume and clinical or laboratory signs of hypoperfusion Clinical hypoperfusion: Cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure Laboratory hypoperfusion: Metabolic acidosis, elevated serum lactate, elevated serum creatinine

1) Blood pressure threshold

2) Clinical/laboratory evidence of hypoperfusion/congestion

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

Van Diepen. Circulation. 2017.

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 ↓	↓	↑	>0.6	≤0.9
Biventricular	↑	↑	↓	↑	≤0.6	≤0.9

- Cardiac power output (CPO) (W) = MAP × CO/451
- Pulmonary artery pulsatility index (PAPi) = (PA systolic - PA diastolic) / RA mean

Slide courtesy of Dr. Erin Bohula

Recognition

TABLE 21.4 Sensitivity and Specificity of History and Physical Examination (H&P) Components for Diagnosis of Elevated Filling Pressures in Patients with Heart Failure*

H&P FINDING	FREQUENCY	SENSITIVITY	SPECIFICITY	PREDICTIVE VALUE		LR		OR (95% CI)
				Positive	Negative	Positive	Negative	
Rales (≥1/3 lung fields)	26/192	15	89	69	38	1.32	1.04	1.4 (0.6, 3.4)
S3	123/192	62	32	61	33	0.92	0.85	0.8 (0.4, 1.5)
Ascites (moderate/massive)	31/192	21	92	81	40	2.44	1.15	2.8 (1.1, 7.3)
Edema (≥2+)	73/192	41	66	67	40	1.20	1.11	1.3 (0.7, 2.5)
Orthopnea (≥2 pillows)	157/192	86	25	66	51	1.15	1.80	2.1 (1, 4.4)
Hepatomegaly (>4 fingerbreadths)	23/191	15	93	78	39	2.13	1.09	2.3 (0.8, 6.6)
Hepatojugular reflux	147/186	83	27	65	49	1.13	1.54	1.7 (0.9, 3.5)
JVP ≥12 mm Hg	101/186	65	64	75	52	1.79	1.82	3.3 (1.8, 6.1)
JVP <8 mm Hg	18/186	4.3	81	28	33	0.23	0.85	0.2

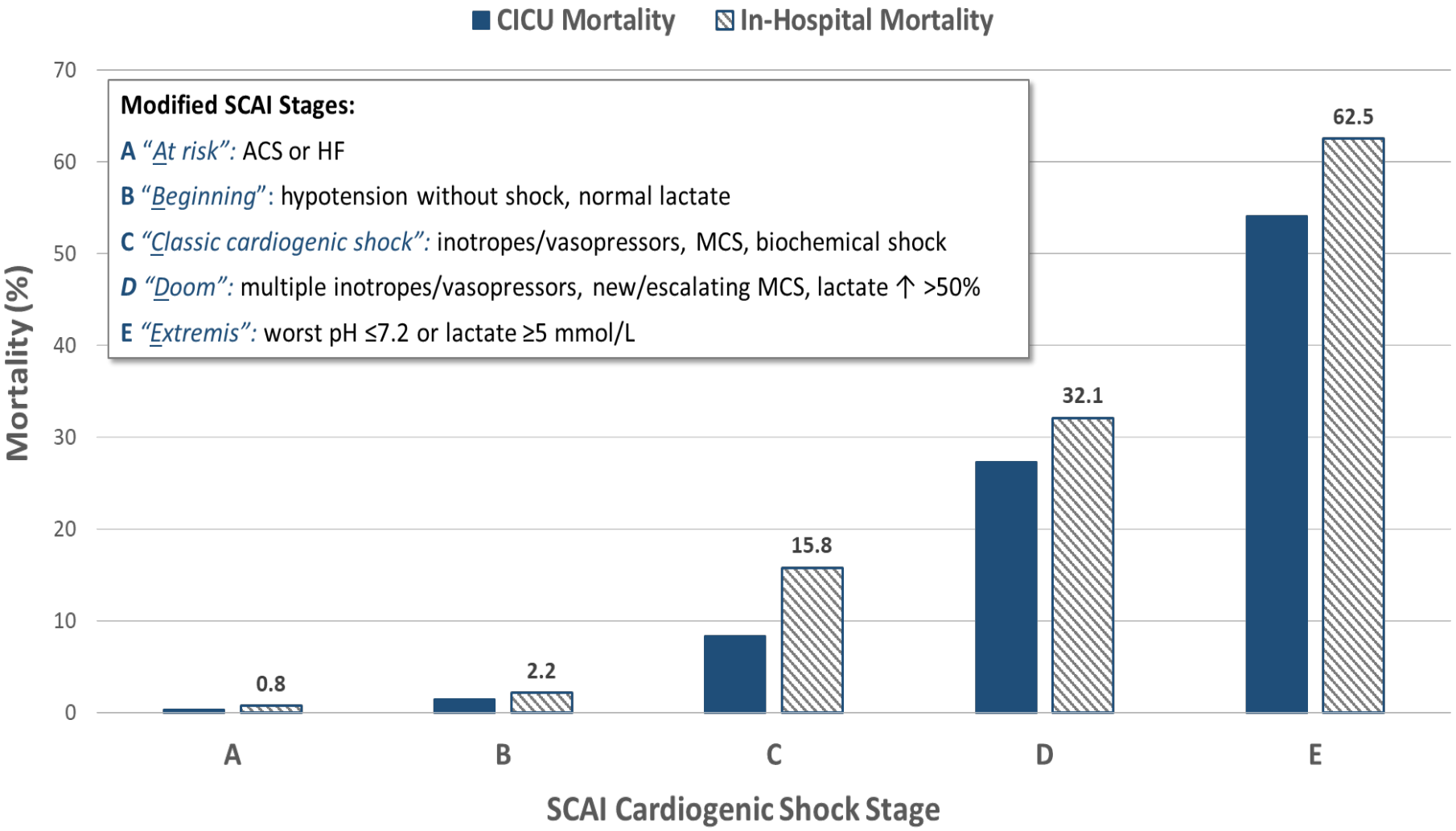
Braunwald's Heart Disease. 11th Ed. Chpt 21.

Cardiogenic Shock Staging

SCAI Shock Stages (*Cath CVI* 2019;94:29)					
Stage	**Description**	**BP**	**Exam**	**Labs**	**Hemodyn.**
At risk	MI, ADHF	nl	nl	nl	nl
Beginning	Relative HoTN; tachycardia; w/o hypoperfusion	SBP <90, MAP <60, or >30 mmHg ↓ from baseline	↑ JVP, crackles, extrem warm	nl	↑ PCWP CI ≥2.2
Classic	Hypoperfusion that requires intervention	SBP <90, MAP <60, or requiring drugs/device to maintain BP	↑ JVP, crackles, extrem cool & mottled, ↓ UOP	↑ Cr lactate ≥2 ↑ LFTs	↑ PCWP CI <2.2 CPO ≤0.6
Deteriorating	Failing to respond			↑↑ Cr, lactate, & LFTs	
Extremis	Near or in cardiac arrest	HoTN despite max support		lactate ≥5 pH <7.2	CI <<2.2 CPO <<0.6

Slide courtesy of Dr. Erin Bohula

Mortality by SCAI Classification



Slide courtesy of Dr. Erin Bohula

Lawler et al. Critical Care Med. 2021

Outline

Epidemiology

Definition and diagnosis

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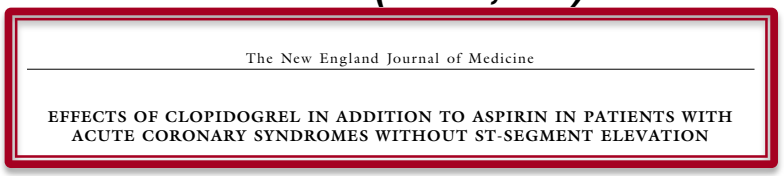
- Acute MI
- General supportive measures
- Mechanical circulatory support

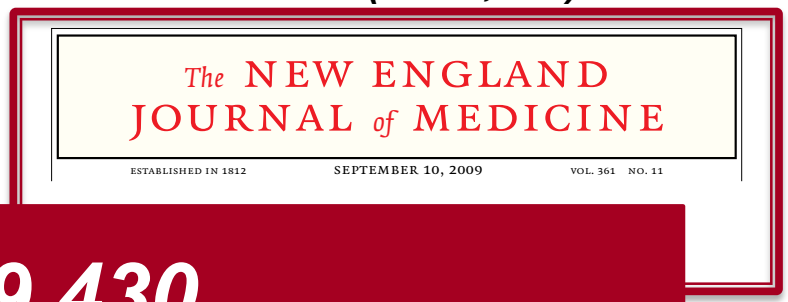
RCTs for P2Y12 inhibition in ACS/PCI

CURE (N=12,562)


The New England Journal of Medicine

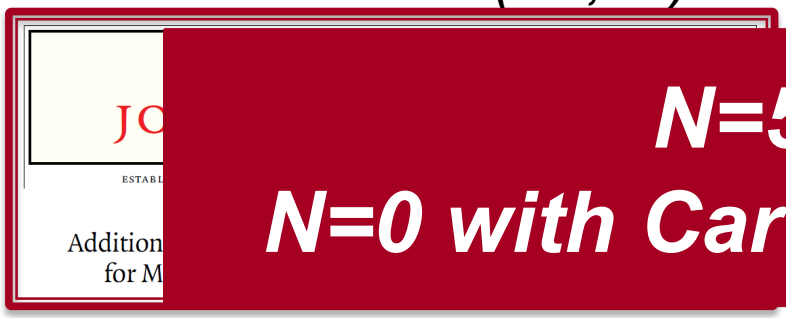
EFFECTS OF CLOPIDOGREL IN ADDITION TO ASPIRIN IN PATIENTS WITH ACUTE CORONARY SYNDROMES WITHOUT ST-SEGMENT ELEVATION

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

ESTABL

Addition
for M

N=59,430
N=0 with Cardiogenic Shock

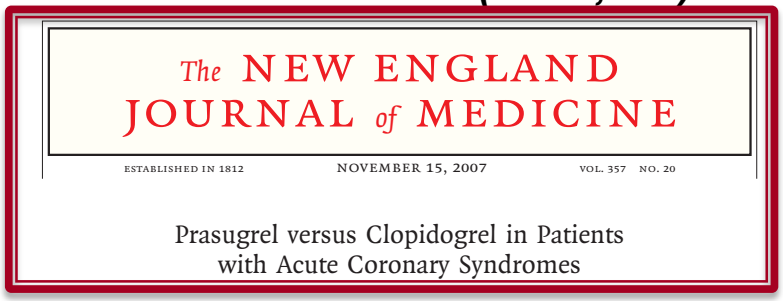
(N=11,145)


DICINE

ORIGINAL ARTICLE

Effect of Platelet Inhibition with Cangrelor during PCI on Ischemic Events

TRITON-TIMI 38 (N=13,608)


The NEW ENGLAND JOURNAL of MEDICINE

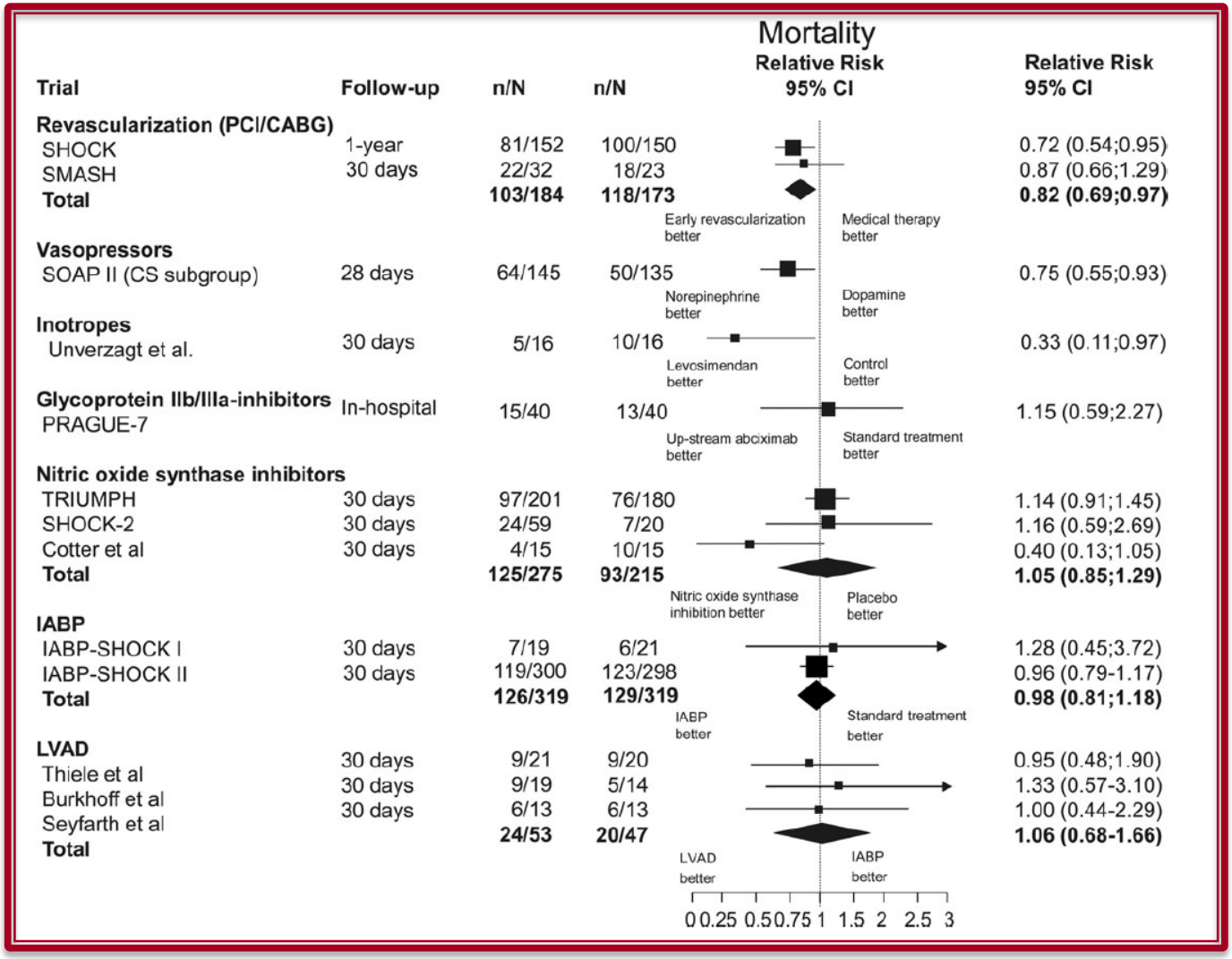
ESTABLISHED IN 1812 NOVEMBER 15, 2007 VOL. 357 NO. 20

Prasugrel versus Clopidogrel in Patients with Acute Coronary Syndromes

Slide courtesy of Dr. Erin Bohula

RCTs in Cardiogenic Shock

Total N~2,000



Slide courtesy of Dr. Erin Bohula

Thiele et al. EHJ. 2015

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*…

Etiologies

- Acute MI
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- Arrhythmia
- PE
- Tamponade
- Myocarditis
- Congenital heart disease with ADHF
- Pulmonary hypertension
- RV failure
- *Et cetera*…

Acute MI complicated by shock

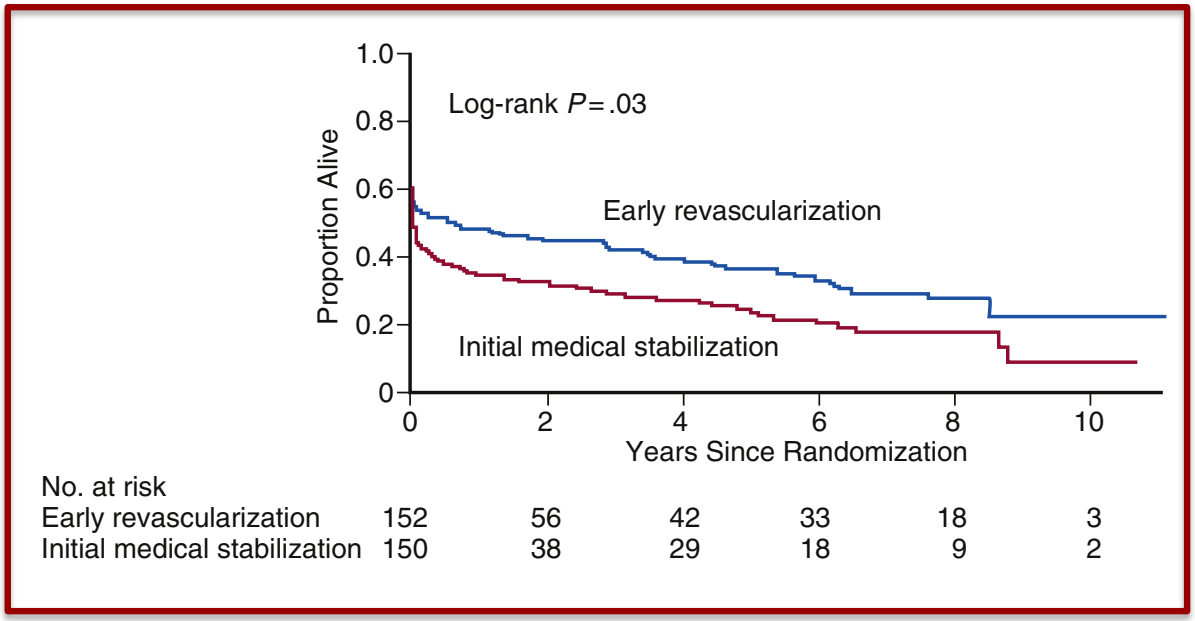
Early revascularization

General supportive measures

Mechanical circulatory support as needed

Recognition and mgmt of mechanical complications

Mortality Benefit with Early Revascularization



- **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)

Hochman JS, Sleeper LA, Webb JG, et al: JAMA *295(21):2511, 2006*

Emergency Revascularization

STEMI + **symptom duration** <12 hours

STEMI with symptom duration 12-24 hours + **ongoing evidence of ischemia**

STEMI + **shock or severe HF** irrespective of symptom duration

NSTEMI + **shock/dangerous arrhythmia/refractory symptoms/etc.**

Non-culprit lesions in STEMI

Table 2 Procedural characteristics of patients with STEMI and CS

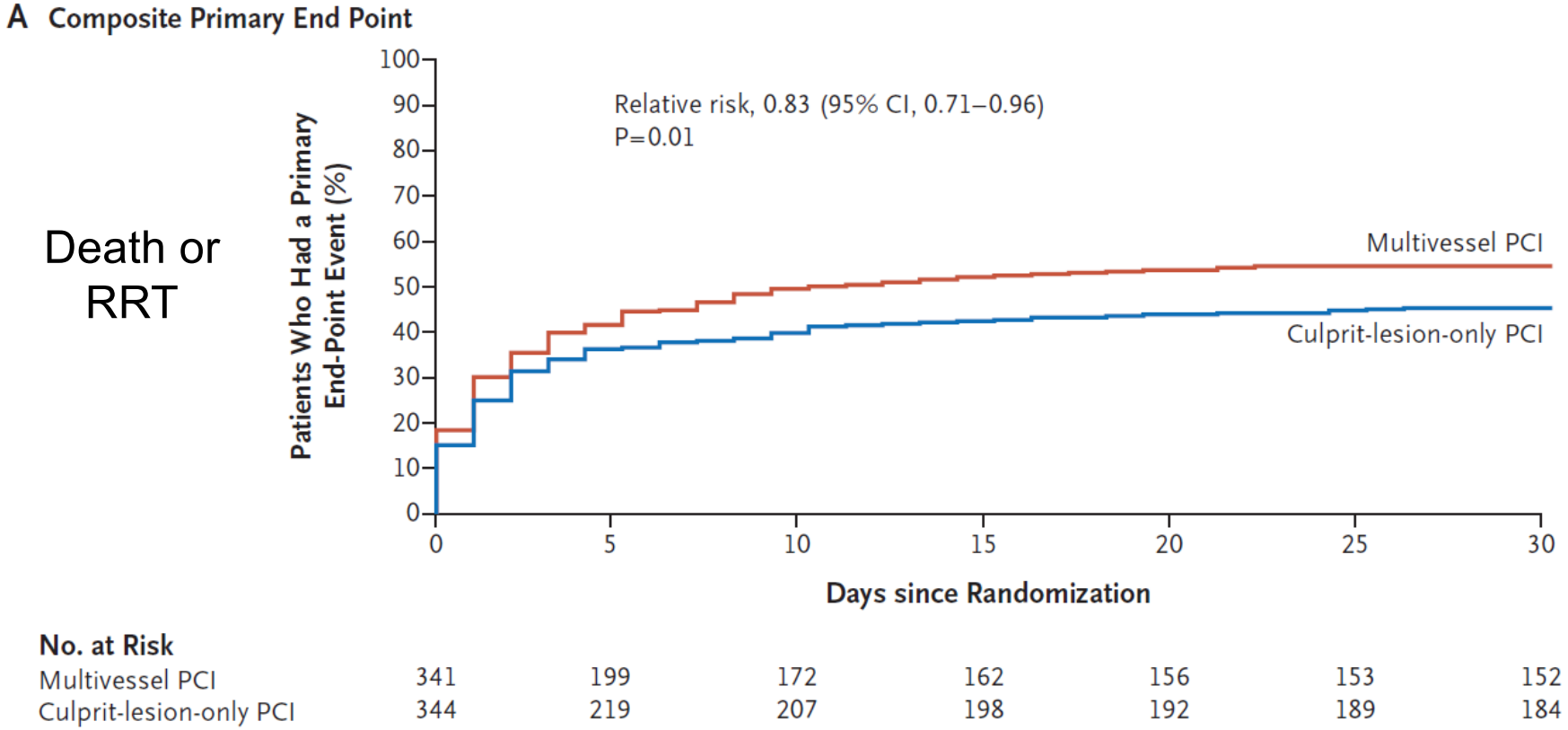
	2006–2009 ($n=302$)	2010–2013 ($n=301$)	2014–2016 ($n=378$)	p value
Left main disease, n (%)	50 (17)	43 (14)	44 (12)	0.118
One-vessel disease, n (%)	77 (26)	93 (31)	117 (31)	0.255
Two-vessel disease, n (%)	89 (29)	84 (28)	116 (31)	0.732
Three-vessel disease, n (%)	133 (44)	124 (41)	145 (38)	0.326

Backhaus. Clin Res Cardiol. 2018.

CULPRIT-SHOCK

706 pts with CS due to AMI (61% STEMI/39% NSTEMI) and MVD
Rx: Immediate MV PCI vs Culprit-Only +/- Staged PCI

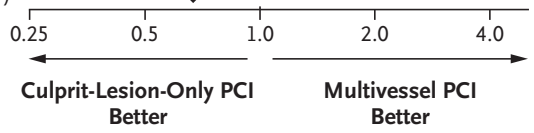
Death or RRT



Thiele. NEJM. 2017.

CULPRIT-SHOCK

Subgroup	Primary End-Point Event: Multivessel PCI	Primary End-Point Event: Culprit-Lesion-Only PCI	Relative Risk (95% CI)	P Value for Interaction
	no. of patients with event/total no. (%)			
Sex				0.11
Male	148/266 (55.6)	109/257 (42.4)	0.76 (0.64–0.91)	
Female	41/75 (54.7)	48/86 (55.8)	1.02 (0.77–1.35)	
Age				0.24
<50 yr	3/16 (18.8)	6/17 (35.3)	1.88 (0.56–6.29)	
50–75 yr	114/226 (50.4)	82/212 (38.7)	0.77 (0.62–0.95)	
>75 yr	72/99 (72.7)	70/115 (60.1)	0.84 (0.69–1.01)	
Diabetes				0.08
No	116/218 (53.2)	93/235 (39.6)	0.74 (0.61–0.91)	
Yes	66/116 (56.9)	59/102 (57.8)	1.02 (0.81–1.28)	
Hypertension				0.47
No	68/129 (52.7)	65/139 (46.8)	0.89 (0.70–1.13)	
Yes	114/205 (55.6)	88/200 (44.0)	0.79 (0.65–0.97)	
Type of infarction				0.96
NSTEMI	54/97 (55.7)	45/98 (45.9)	0.82 (0.62–1.09)	
STEMI	128/233 (54.9)	108/237 (45.6)	0.83 (0.69–0.99)	
Location of STEMI				0.07
Anterior	59/113 (52.2)	57/108 (52.8)	1.01 (0.79–1.30)	
Nonanterior	48/92 (52.2)	34/97 (35.1)	0.67 (0.48–0.94)	
Previous infarction				0.83
No	154/281 (54.8)	128/279 (45.9)	0.84 (0.71–0.99)	
Yes	28/53 (52.8)	25/60 (41.7)	0.79 (0.53–1.17)	
No. of affected vessels				0.56
2	64/124 (51.6)	48/122 (39.3)	0.76 (0.58–1.01)	
3	124/215 (57.7)	109/218 (50.0)	0.87 (0.73–1.03)	
Chronic total occlusion				0.26
No	146/259 (56.4)	131/267 (49.1)	0.87 (0.74–1.02)	
Yes	43/82 (52.4)	27/77 (35.1)	0.67 (0.46–0.97)	



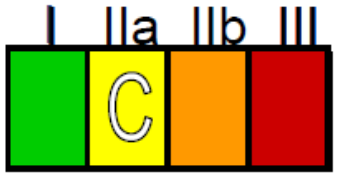
Thiele H et al. NEJM 2017;377(25).

Multi-vessel PCI in Shock

ESC STEMI Guidelines 2017 → Revascularization Guidelines 2018

STEMI (NSTEMI), Cardiogenic Shock

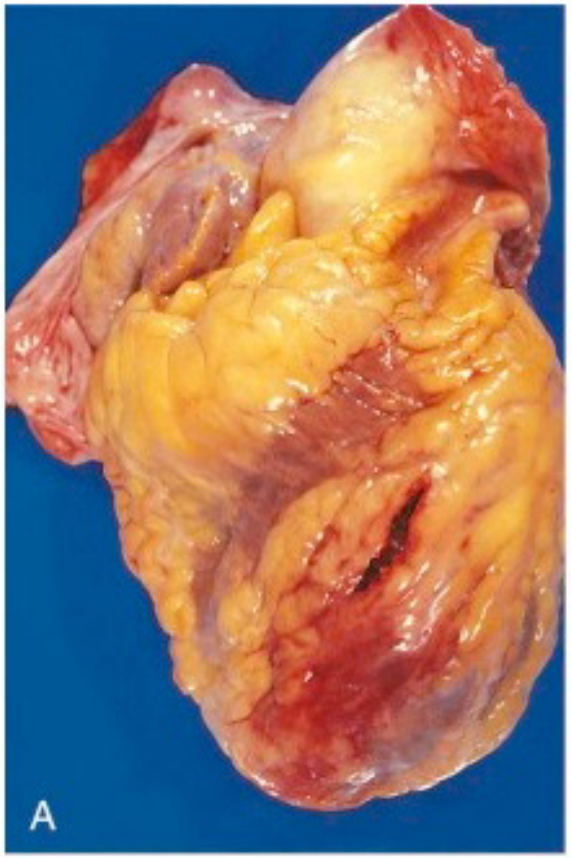
2017

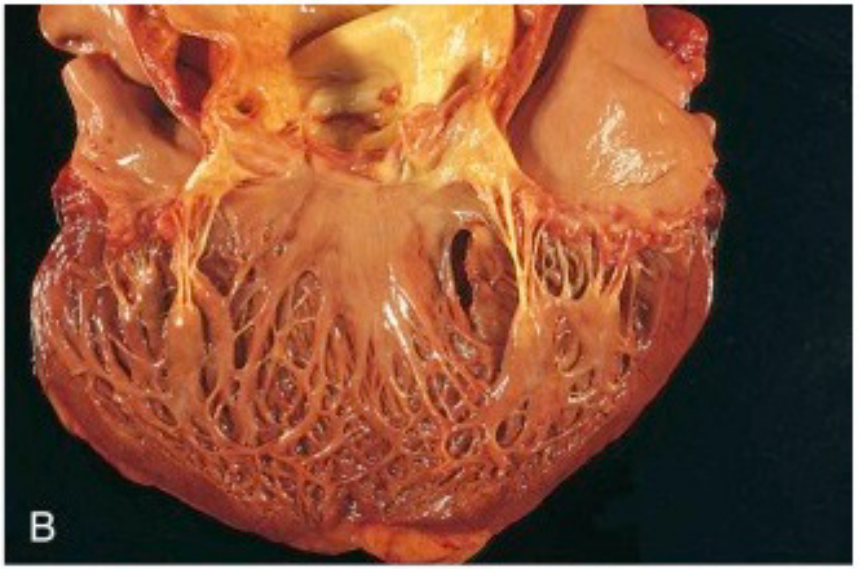


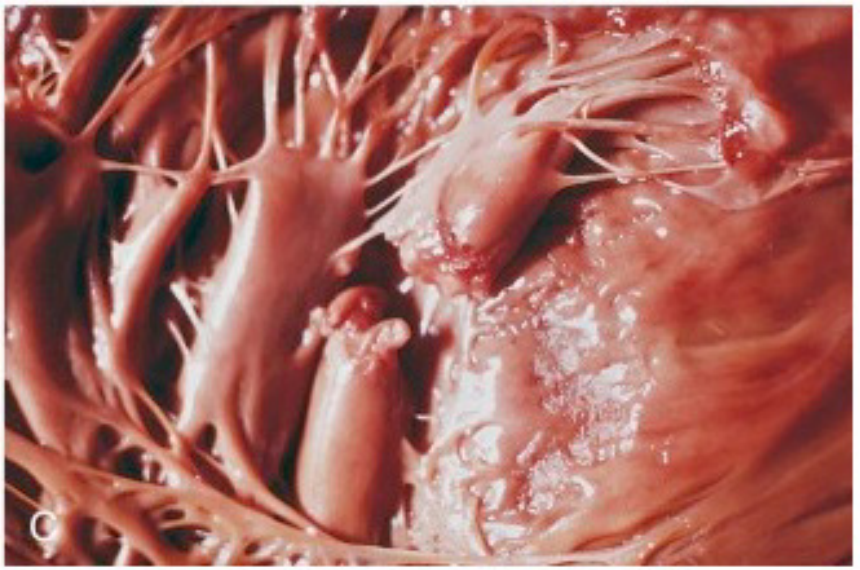
2018

Slide courtesy of Dr. Erin Bohula

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
- 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

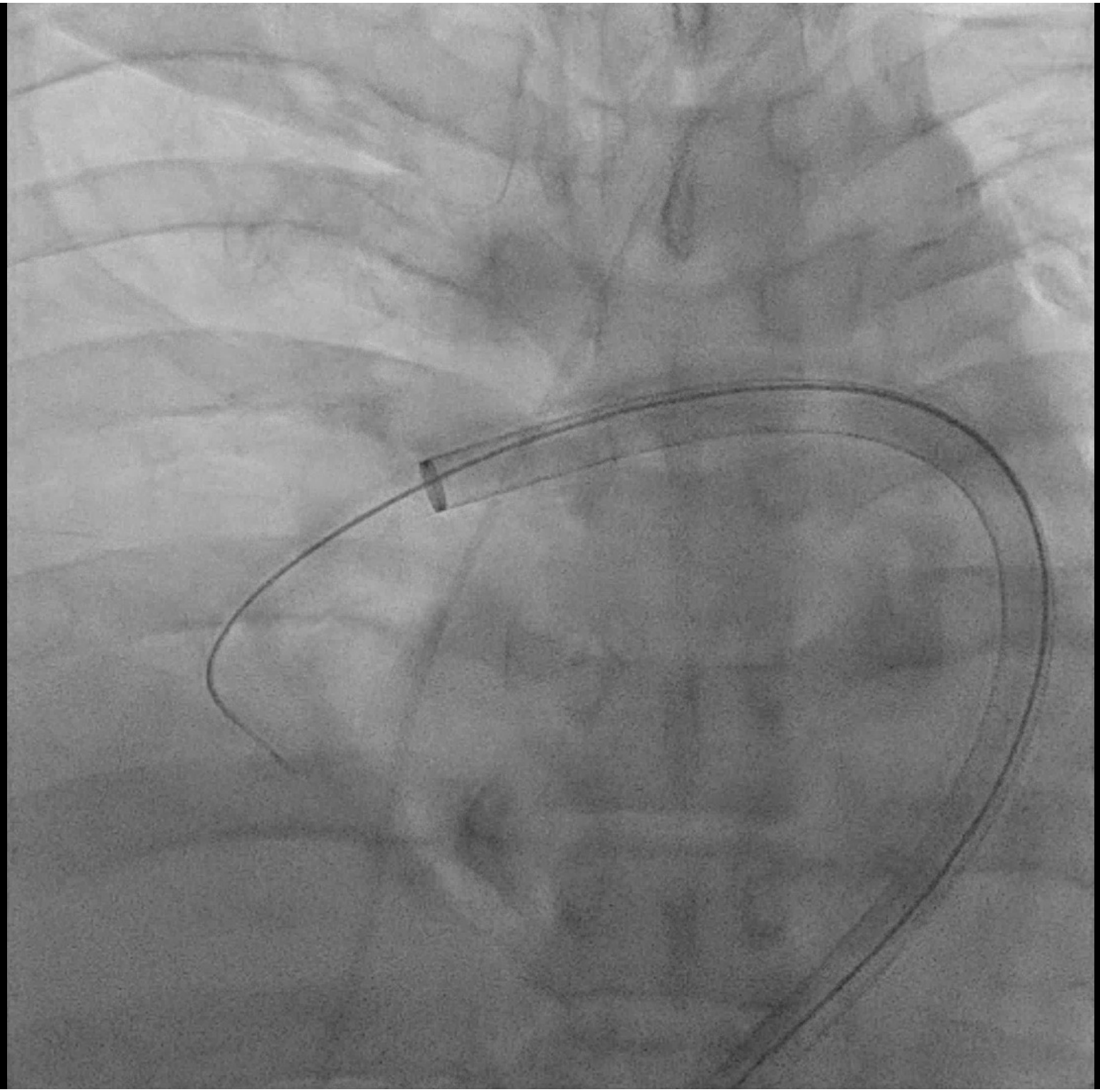
Valvular disease

Arrhythmia

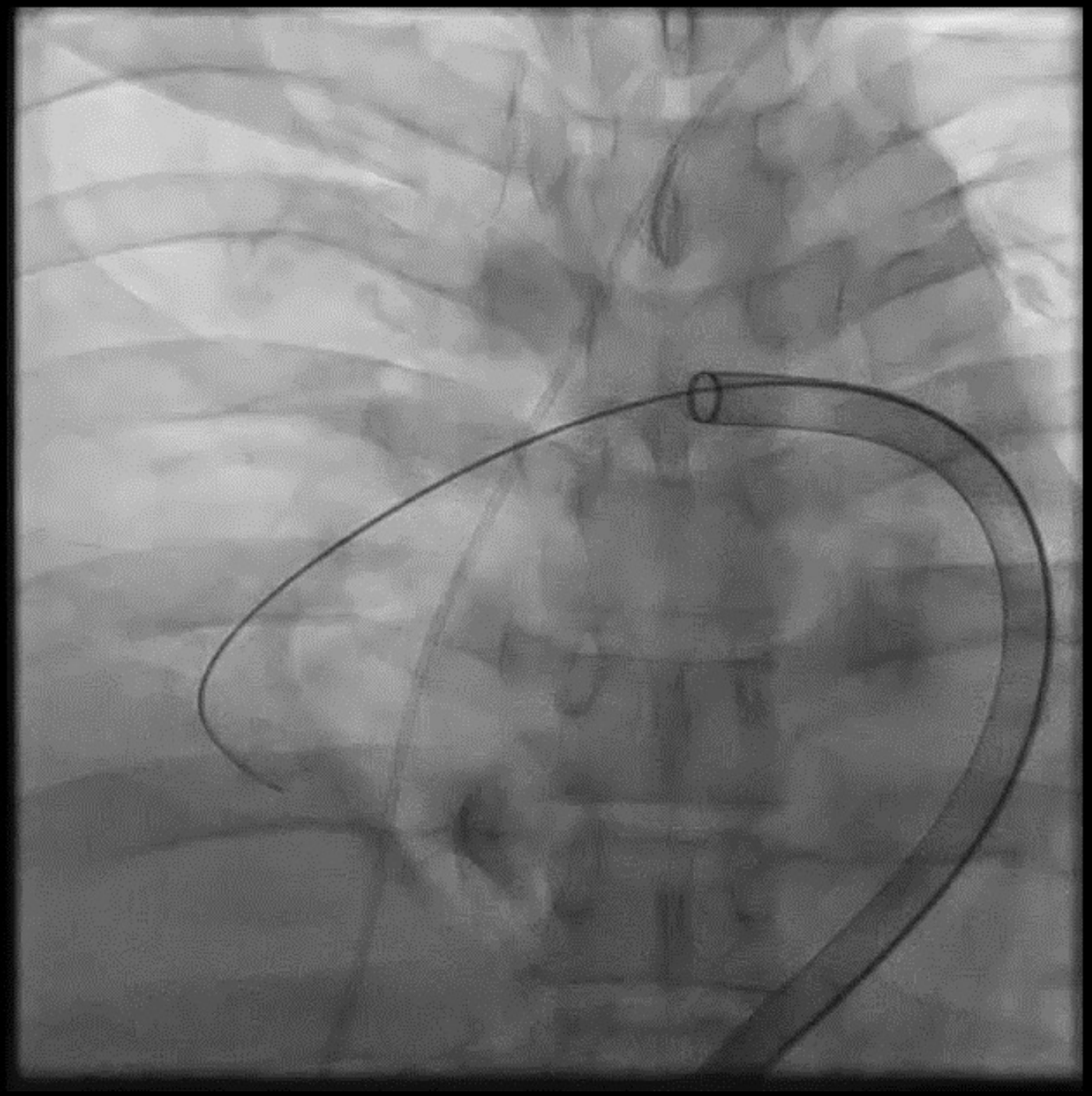
Tamponade

Myocarditis

Pulmonary hypertension



LEFT
Right



Outline

Epidemiology

Definition and diagnosis

Management

- Acute MI
- General supportive measures
- Mechanical circulatory support

For mild to moderate shock

↑ Cardiac output

↓ **Resistance**

↓ **Filling pressures**

Inotrope

Vasodilator + Diuretic

Vasoactive therapies

Pure vasopressors – Incr SVR

Inopressors – Incr CO, Incr SVR

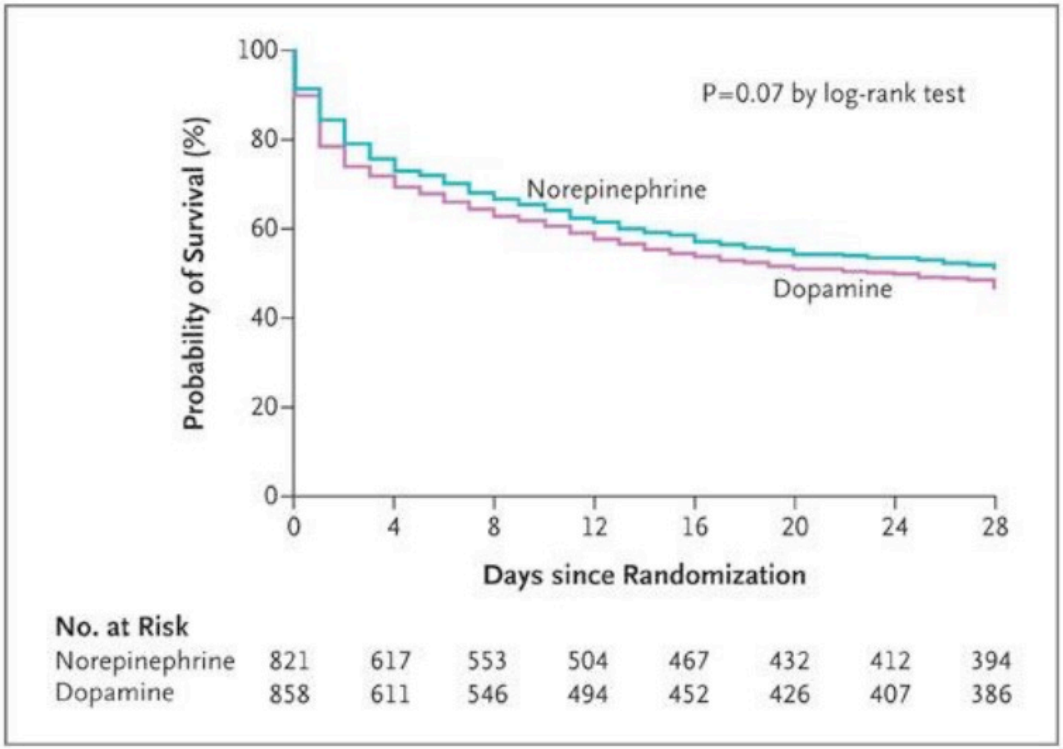
Inodilators – Incr CO, decr SVR

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	$\alpha >> \beta_1$	↑↑	↔/↑	↔/↑	↑↑↑	↔/↑	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	β_1 > D, α	↔/↑	↑	↑↑	↔	↔	
High-dose	$\alpha > \beta_1$, D	↑↑	↑↑	↑	↑↑	↑	
Inodilators							
Dobutamine	$\beta_1 >>> \beta_2, \alpha_1$	↔/↓	↑↑	↑↑	↓	↓	↓ 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

An Academic Research Organization of
Brigham and Women's Hospital and Harvard Medical School

Slide courtesy of Dr. Erin Bohula

SOAP II: Dopamine vs Norepinephrine

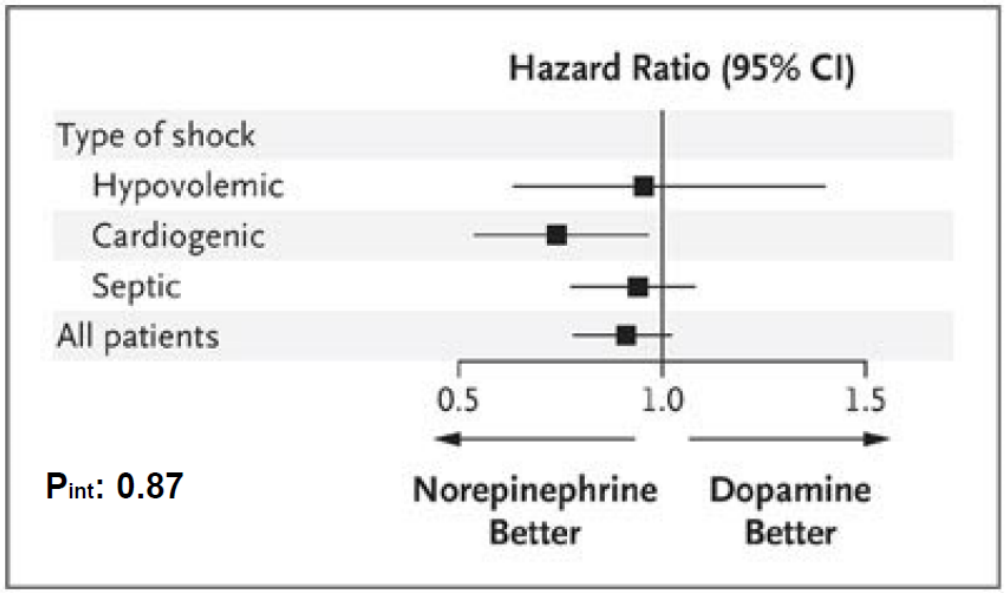


De Backer et al. NEJM 2010;362:779.

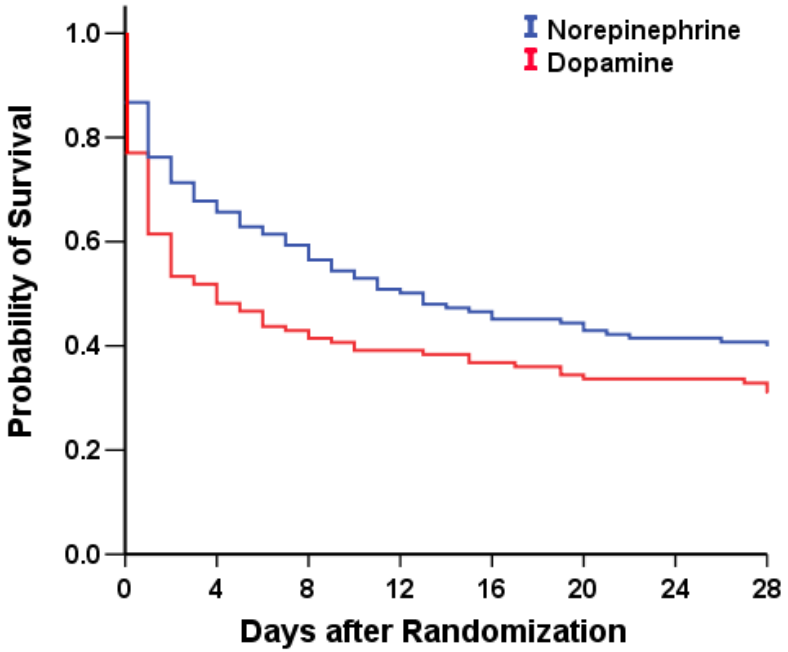
- ***28d mortality:***
  - ***52.5% for DA vs 48.5% for norepi***
  - ***OR 1.17 (0.97-1.42), p=0.10***
- ***Arrhythmias: 24.1% vs 12.4%***

Slide courtesy of Dr. Erin Bohula

SOAP II: Dopamine vs Norepinephrine



Cardiogenic Shock (N=280)

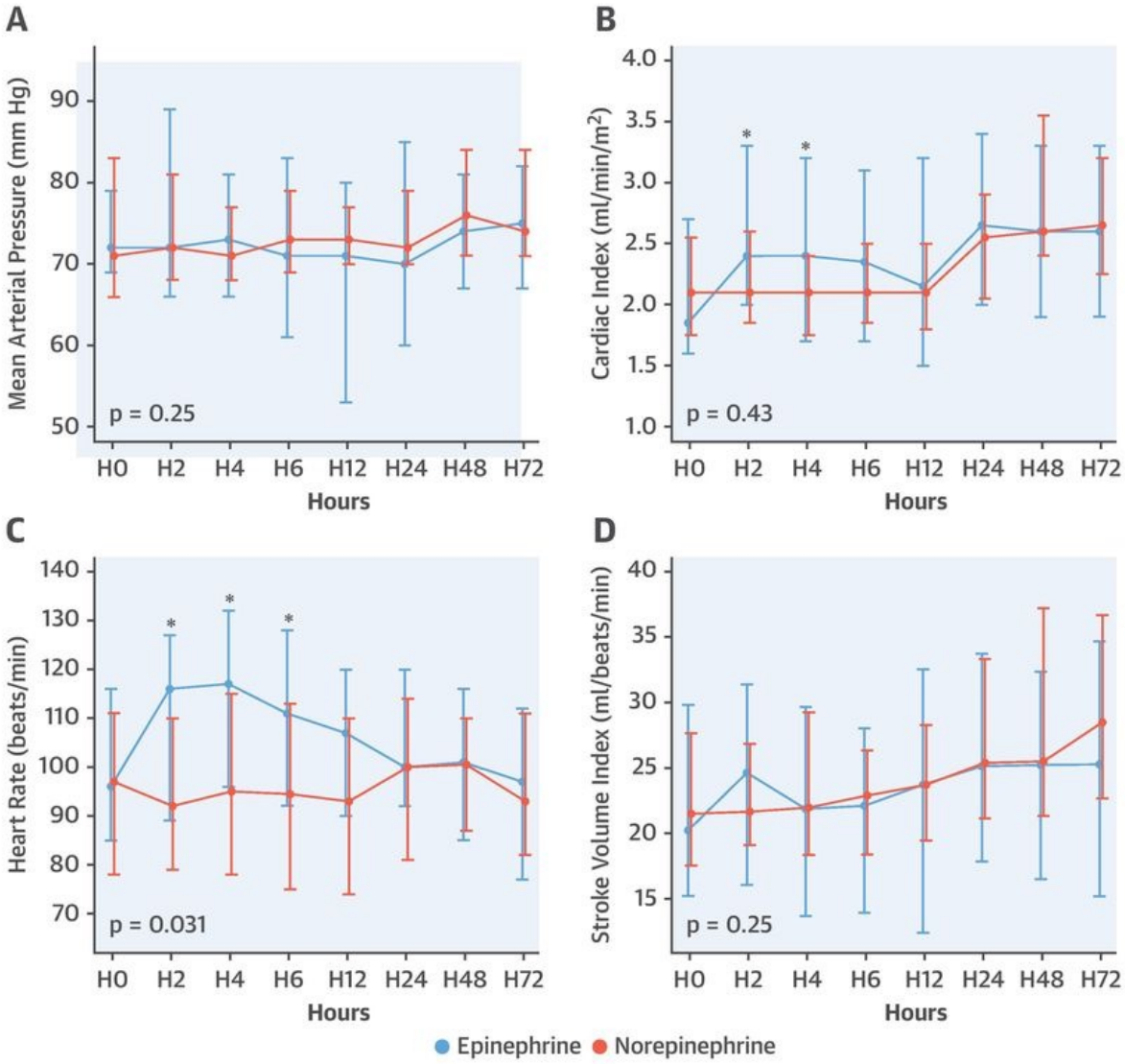


Signal of harm with dopamine?

Slide courtesy of Dr. Erin Bohula

Epinephrine vs Norepinephrine

57 pts with CS due to AMI s/p PCI with PA Line



Levy et al. JACC 2018;72:173-82.

Epinephrine vs Norepinephrine

57 pts with CS due to AMI s/p PCI with PA Line

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*

Levy et al. JACC 2018;72:173-82.

Vasopressor summary

- Catecholamines have not demonstrated improved survival
- But, limited 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**
- **Assess degree of congestion (preload) & adequacy of perfusion (CO)**
- **Assess and treat reversible causes of cardiogenic shock:**
  - Acute ischemia
  - Address other potential contributors: dysrhythmias, acid/base disturbances, negative inotropes (bB, CCB) and antihypertensives
- **Optimize hemodynamics, often with PAC to guide therapy**

Outline

Epidemiology

Definition and diagnosis

Management

- Acute MI
- General supportive measures
- Mechanical circulatory support

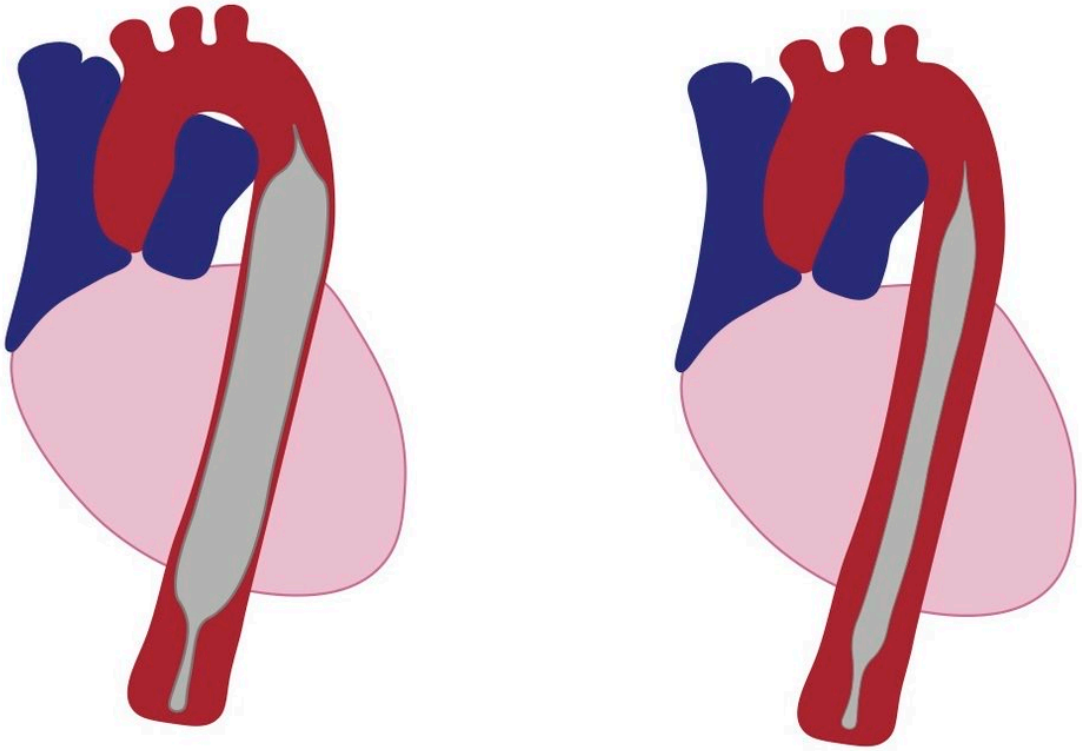
Complex Decisions

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, trxplant)

LV Support

Intra-aortic balloon pump (IABP)



(+)
Rapid placement
Lower profile than other MCS options

(-)
Minimal support
AI

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 OCTOBER 4, 2012 VOL. 367 NO. 14

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*

IABP-SHOCK II

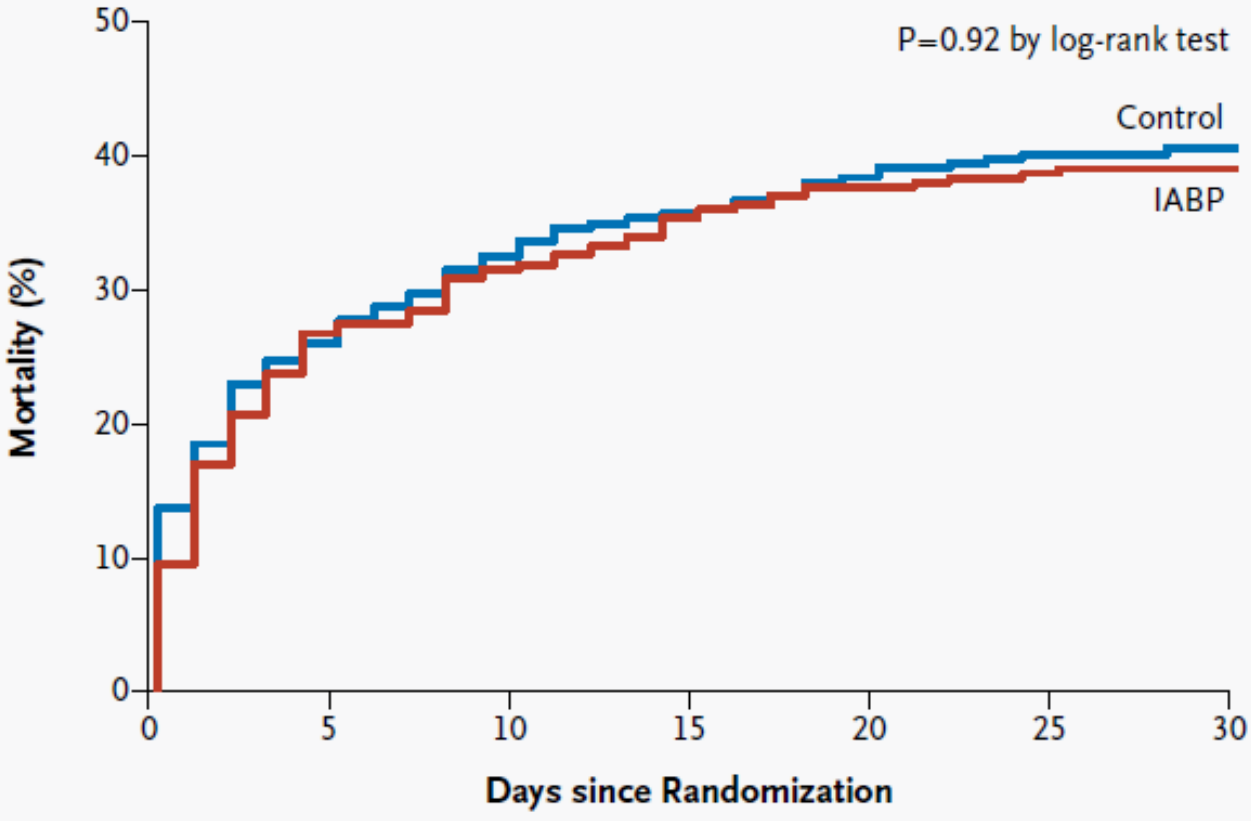
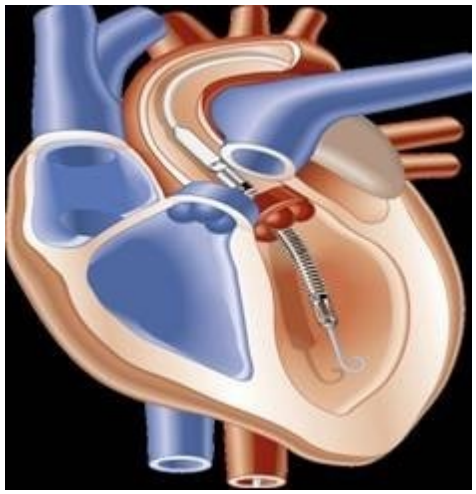


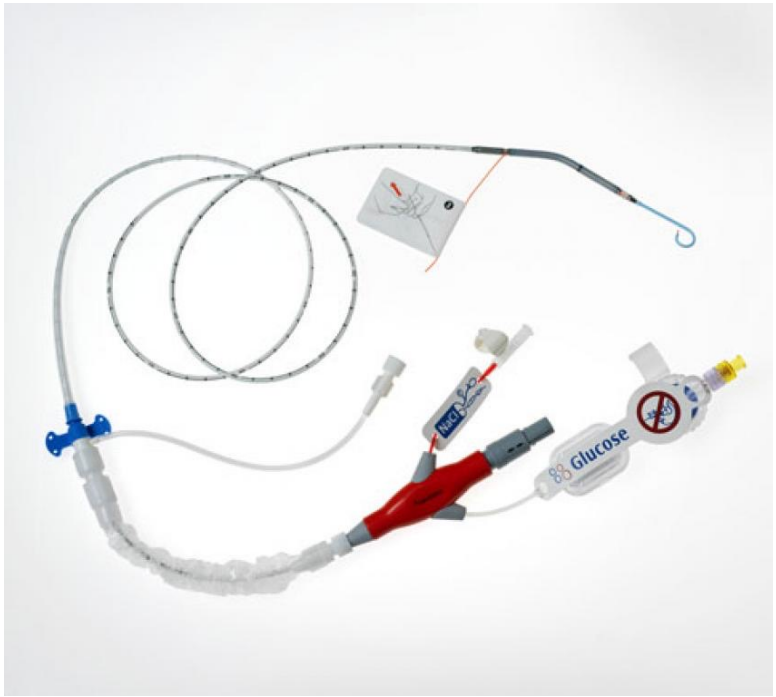
Figure 1. Time-to-Event Curves for the Primary End Point.

Time-to-event curves are shown through 30 days after randomization for the primary end point of all-cause mortality. Event rates represent Kaplan–Meier estimates.

Thiele. NEJM. 2012.

Impella CP





(+)

Good support (3.5 L/min)

Typically rapid placement

Unloads LV

(-)

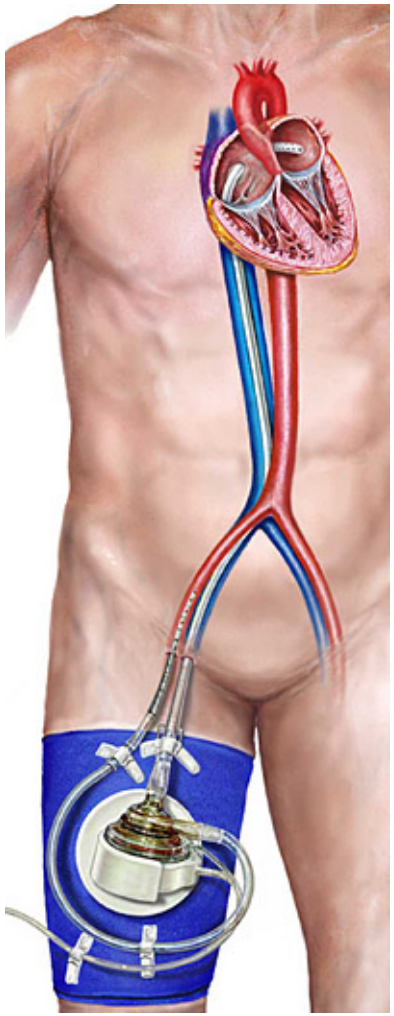
Migrates

Thrombocytopenia/hemol

Vascular injury

Note: Impella 5.0 (and now 5.5) also available

TandemHeart



(+)

Robust support (4-5 L/min)

Possible to add gas exchange to circuit

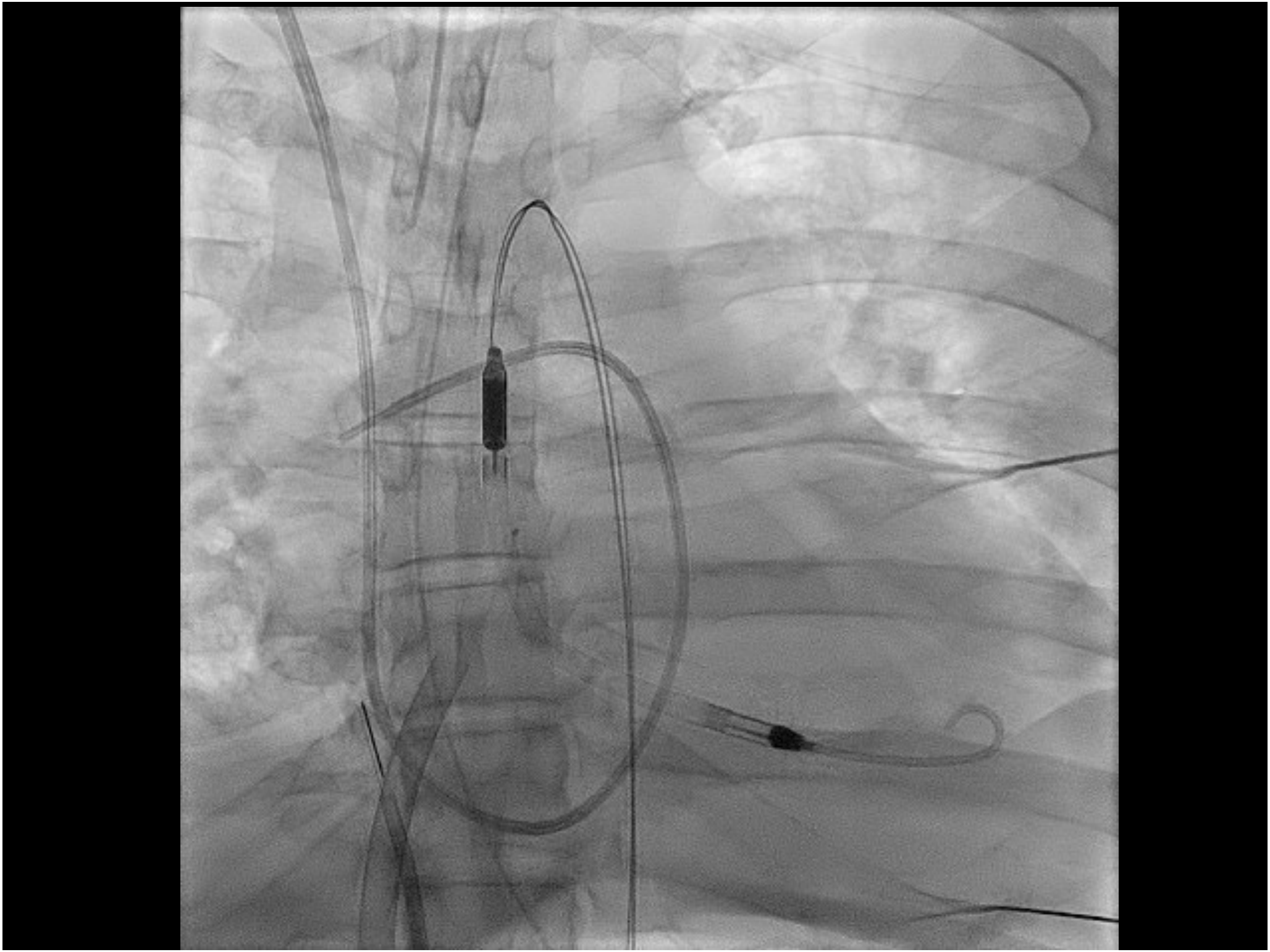
(-)

Limited availability

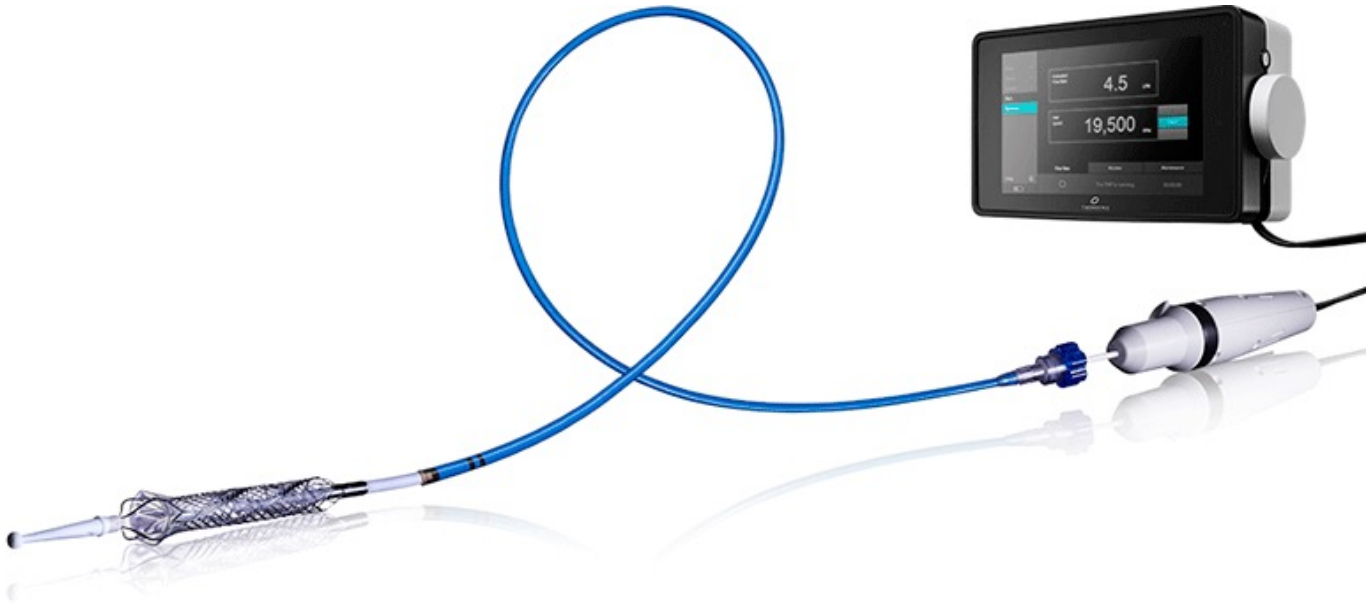
Requires transeptal puncture

Imperfect LV unloading

Vascular injury



Abbott Percutaneous Heart Pump?



BUSINESS

Abbott Labs halts use of HeartMate PHP blood pump after death, malfunctions

St. Jude Medical acquired the device in a 2015 deal and predicted it would be a key growth driver within five years.

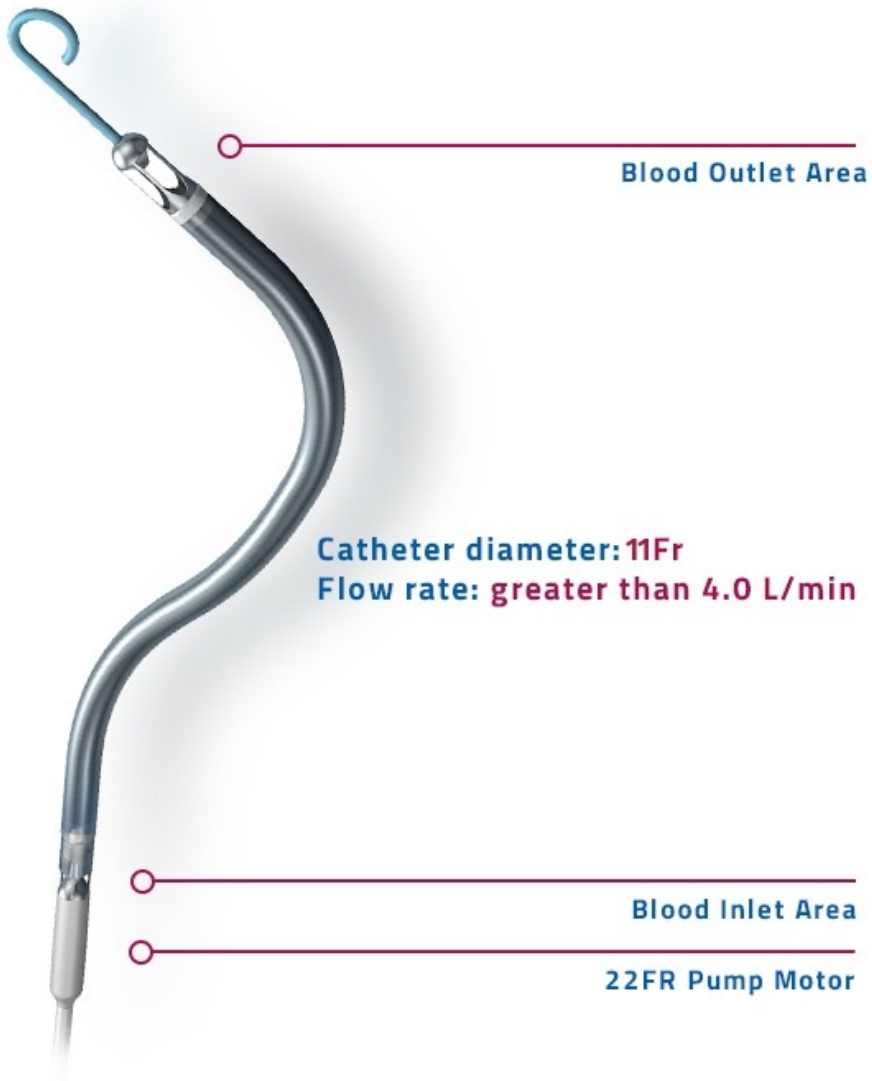
By Joe Carlson Star Tribune | FEBRUARY 9, 2017 — 9:17PM

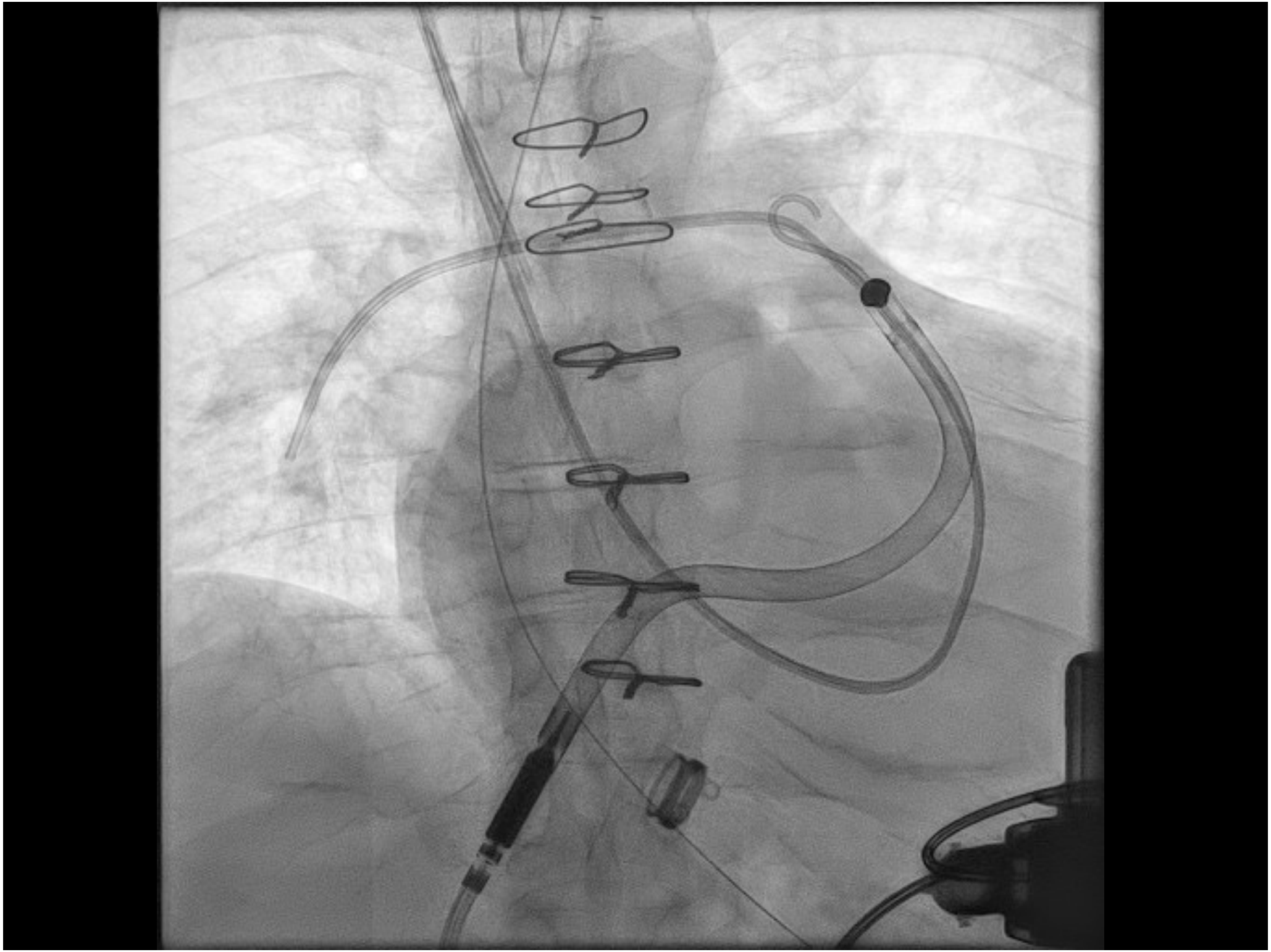
RV Support

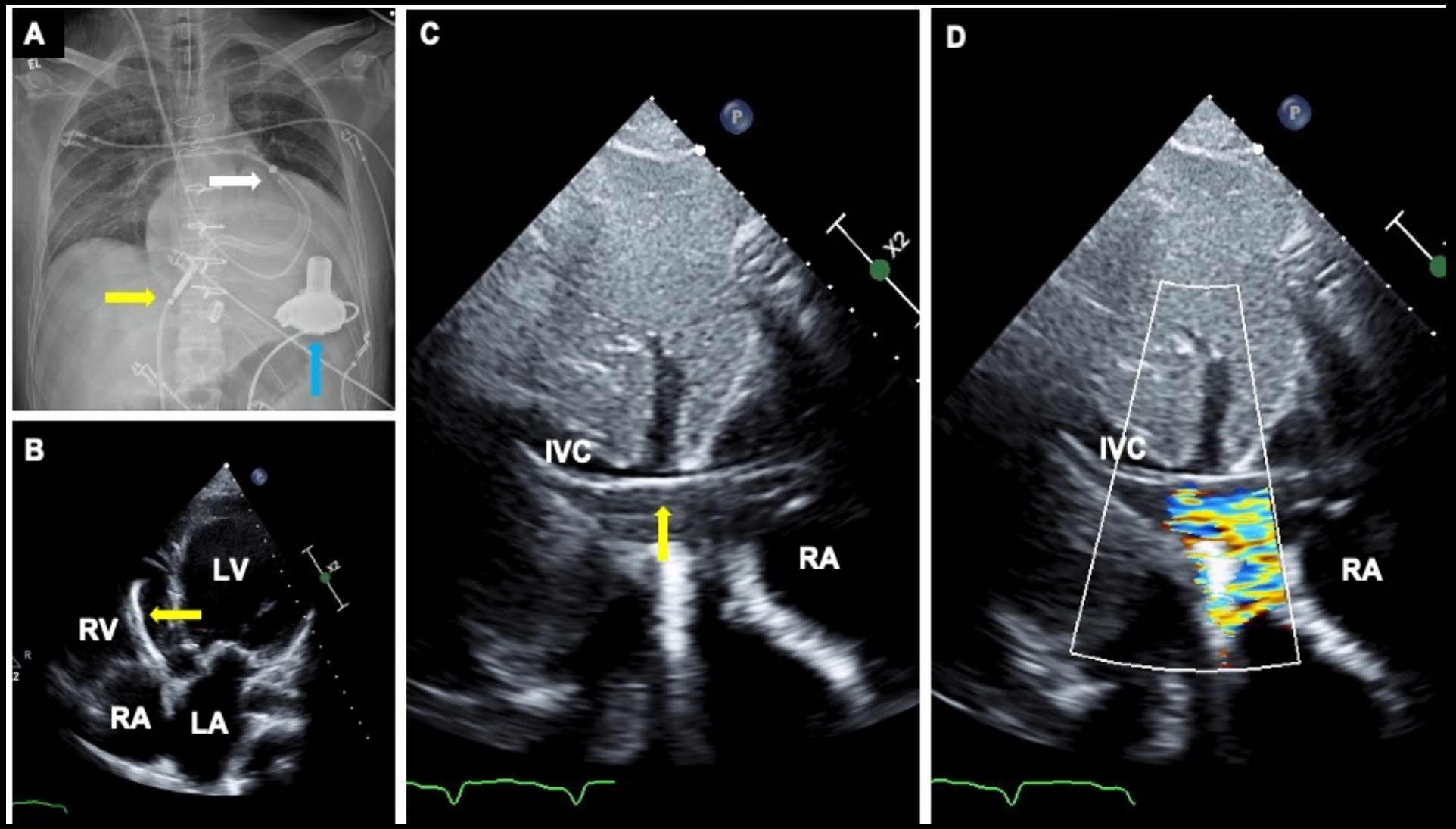
Impella RP

(+)
4 L/min
Typically fast placement

(-)
Femoral only
Migrates
Thrombocytopenia /hemolysis





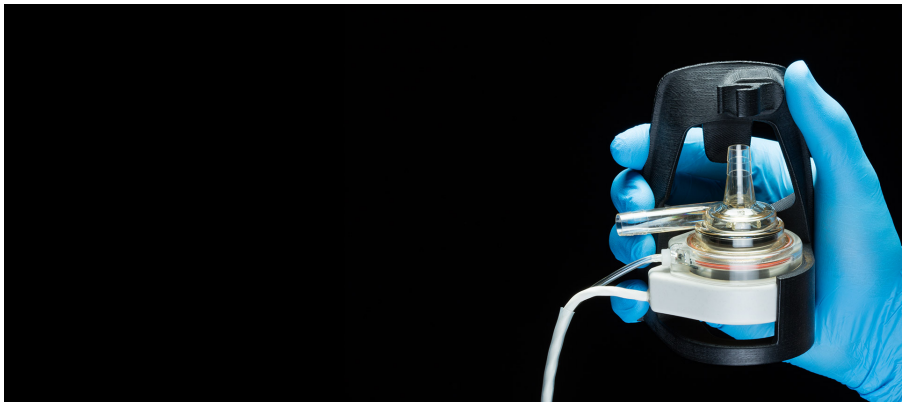


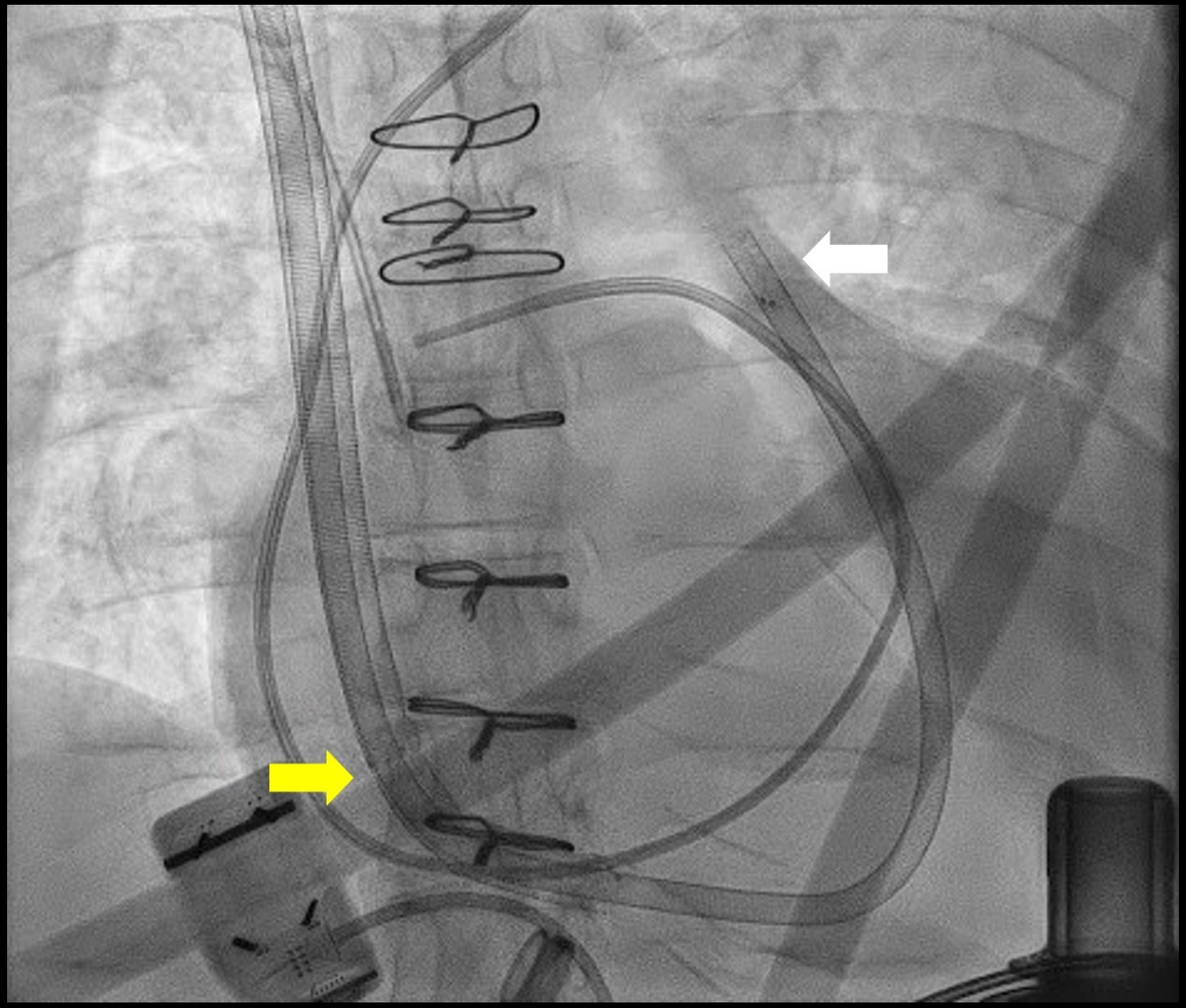
Bergmark and Morrow. Mechanical Support for the Right Ventricle. In Press.

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

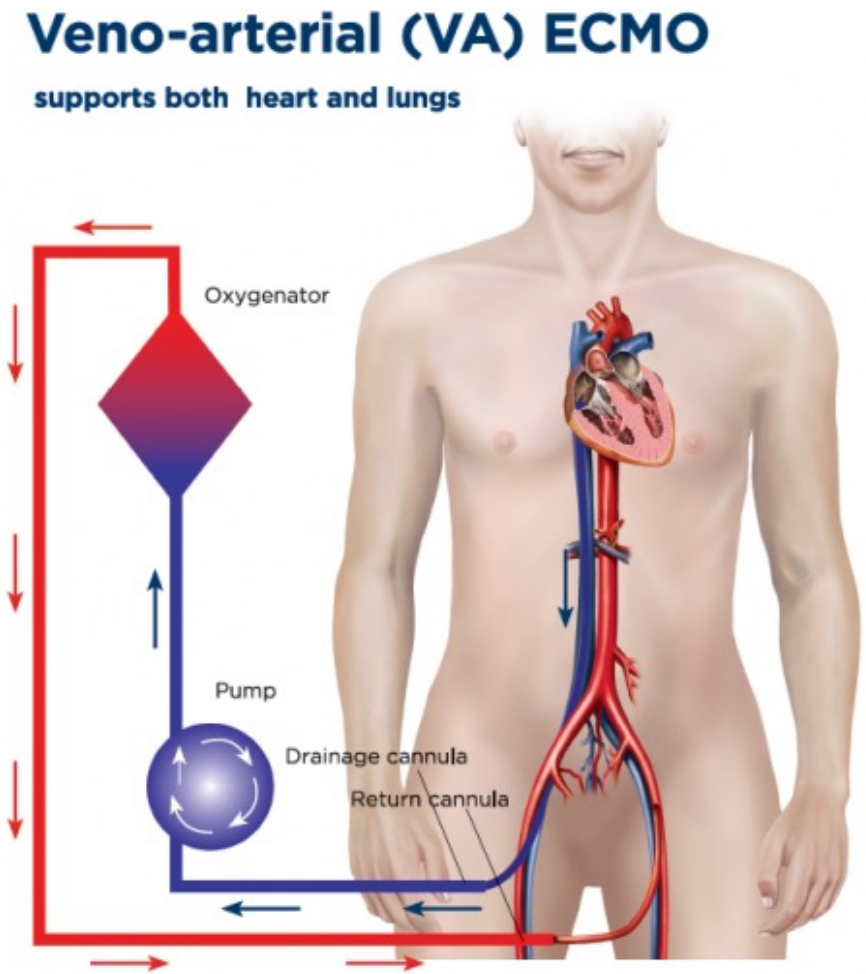




Bergmark and Morrow. Mechanical Support for the Right Ventricle. In Press.

Biventricular Support

Extracorporeal Membrane Oxygenation (ECMO)



(+)

Full cardiopulmonary bypass
(Up to 6 L/min)

RV support

VT/VF tolerated

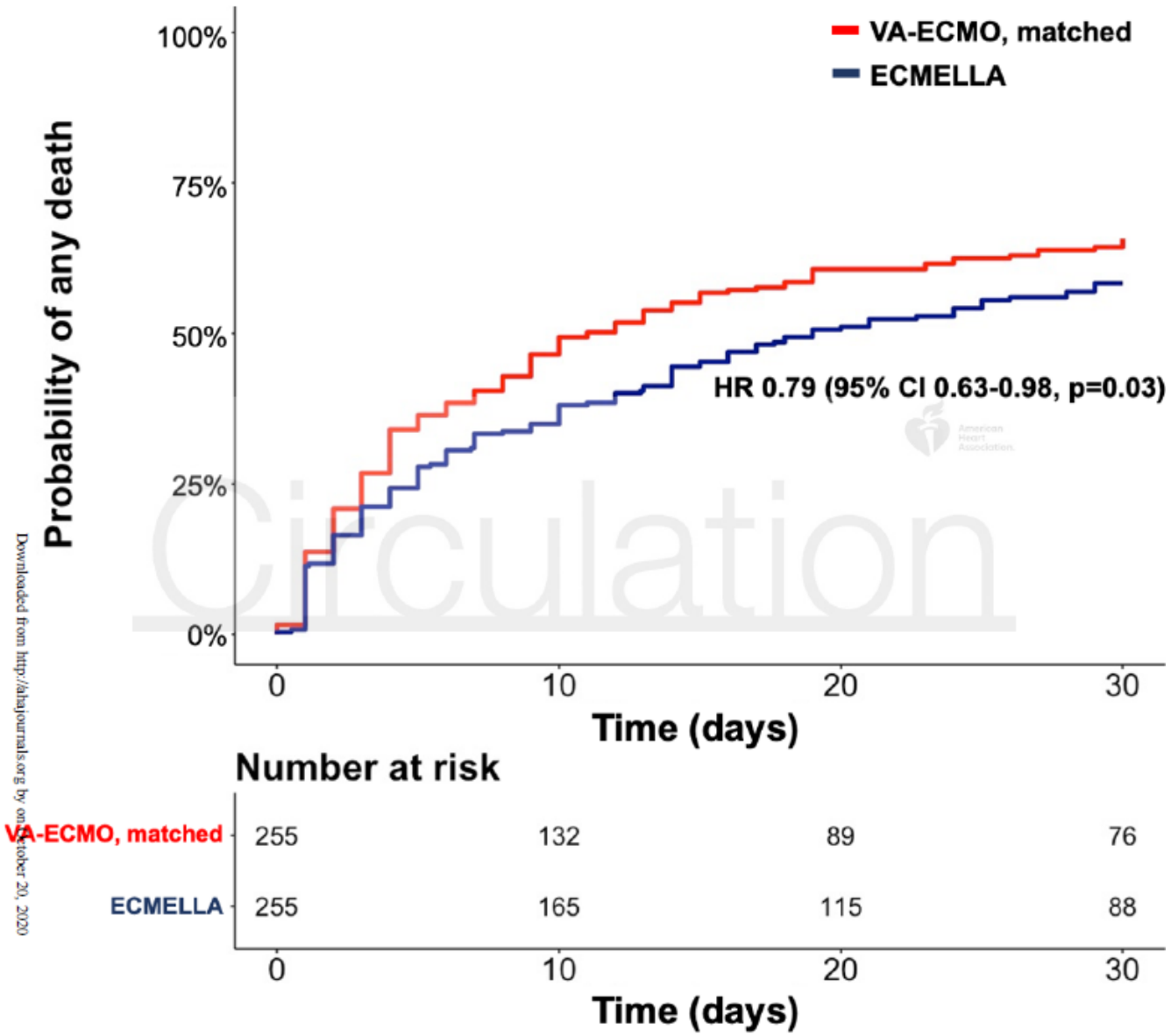
(-)

May require LV vent

Vascular injury

Limited availability

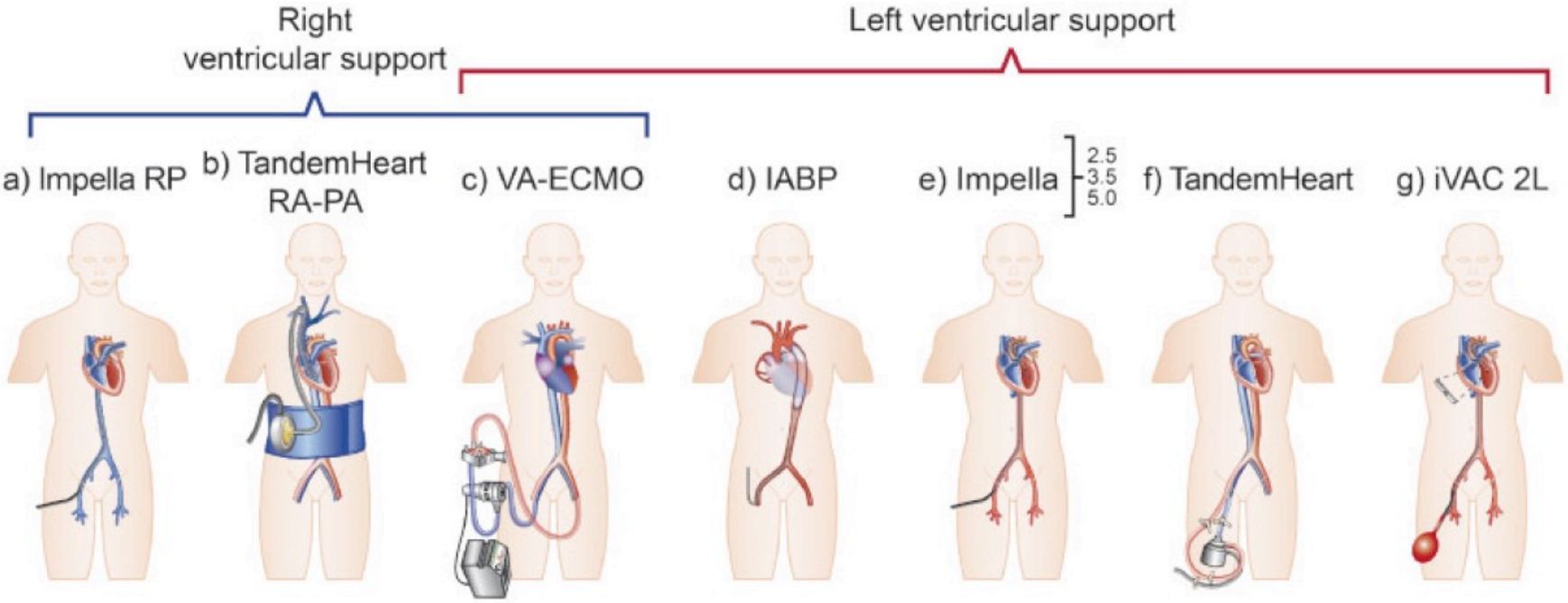
LV Unloading? (trending on twitter…)



Downloaded from http://ahajournals.org by on October 20, 2020

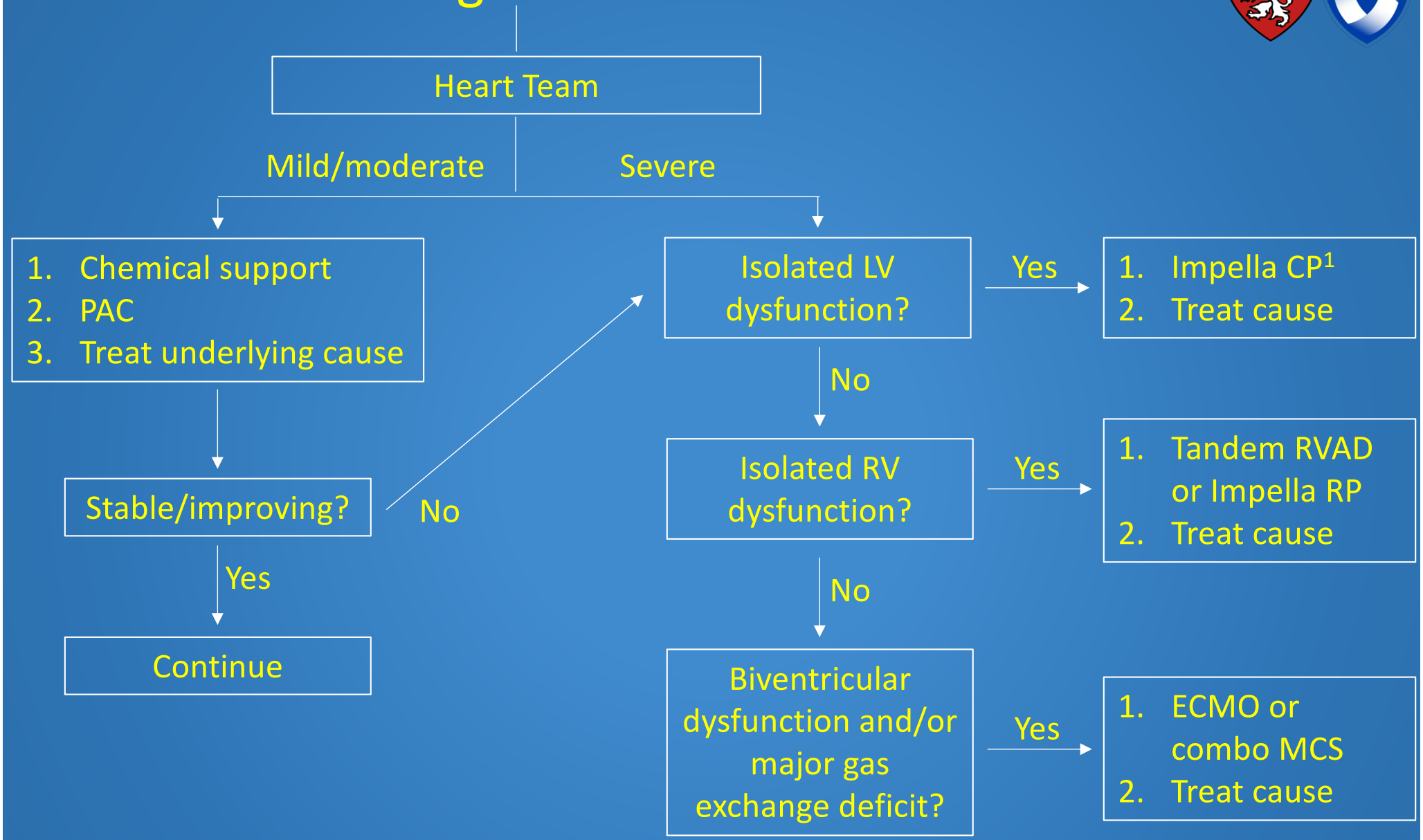
- 16 centers in 4 countries
- 686 consecutive patients
- NOT RANDOMIZED
- 1:1 PSM
- Patients with CS on ECMO in whom team decided to place Impella:
  - Had more complications
  - Had lower rates of death at 30 days
- Hypothesis-generating, needs RCT

MCS Overview



	a) Impella RP	b) TandemHeart RA-PA	c) VA-ECMO	d) IABP	e) Impella 2.5 / 3.5 / 5.0	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	+	+	++	-	-	-	-

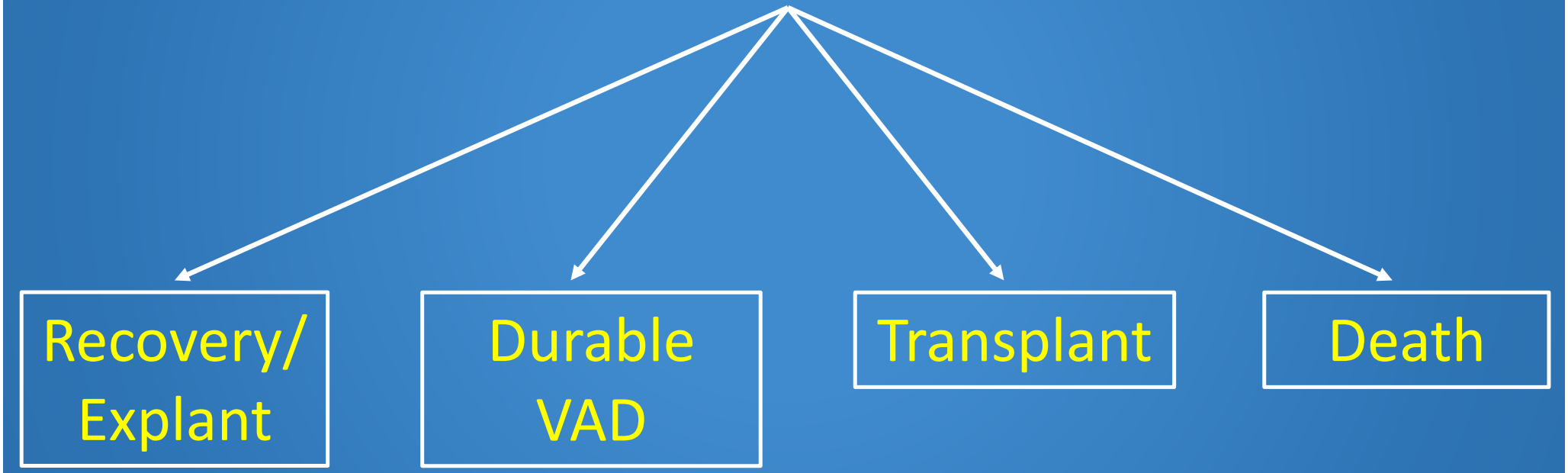
Cardiogenic Shock



[1]ECMO 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



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 developed acute chest pain, hypotension, and dyspnea. Physical exam revealed 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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C) **Emergent transthoracic echocardiogram with simultaneous consultation of Cardiac Surgery and Cardiac Catheterization Laboratory**
D) CT-PE

Take Home Points

Cardiogenic shock is associated with high mortality

Recognizing cardiogenic shock can be challenging, particularly in patients with chronic heart failure

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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