Cardiogenic shock

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HARVARD MEDICAL SCHOOL
TEACHING HOSPITAL

Disclosures

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Several 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

F Number:

I aVR V1 V4

II aVL V2 V5

III aVF V3 V6

V1

II

V5

96 (Derived)

BWH ECHO
S5-1
49Hz
16cm

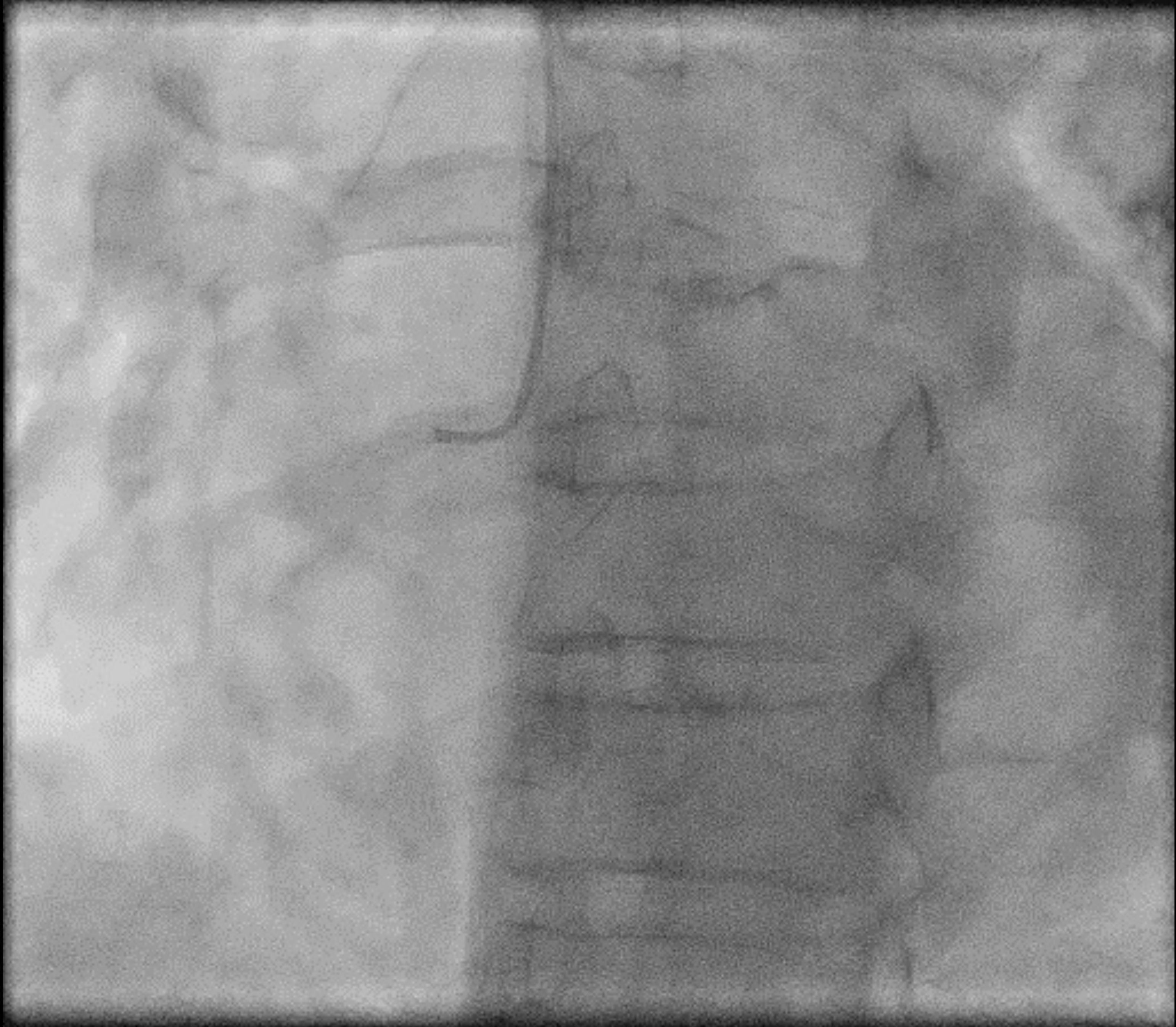
TIS0.6 MI 1.3

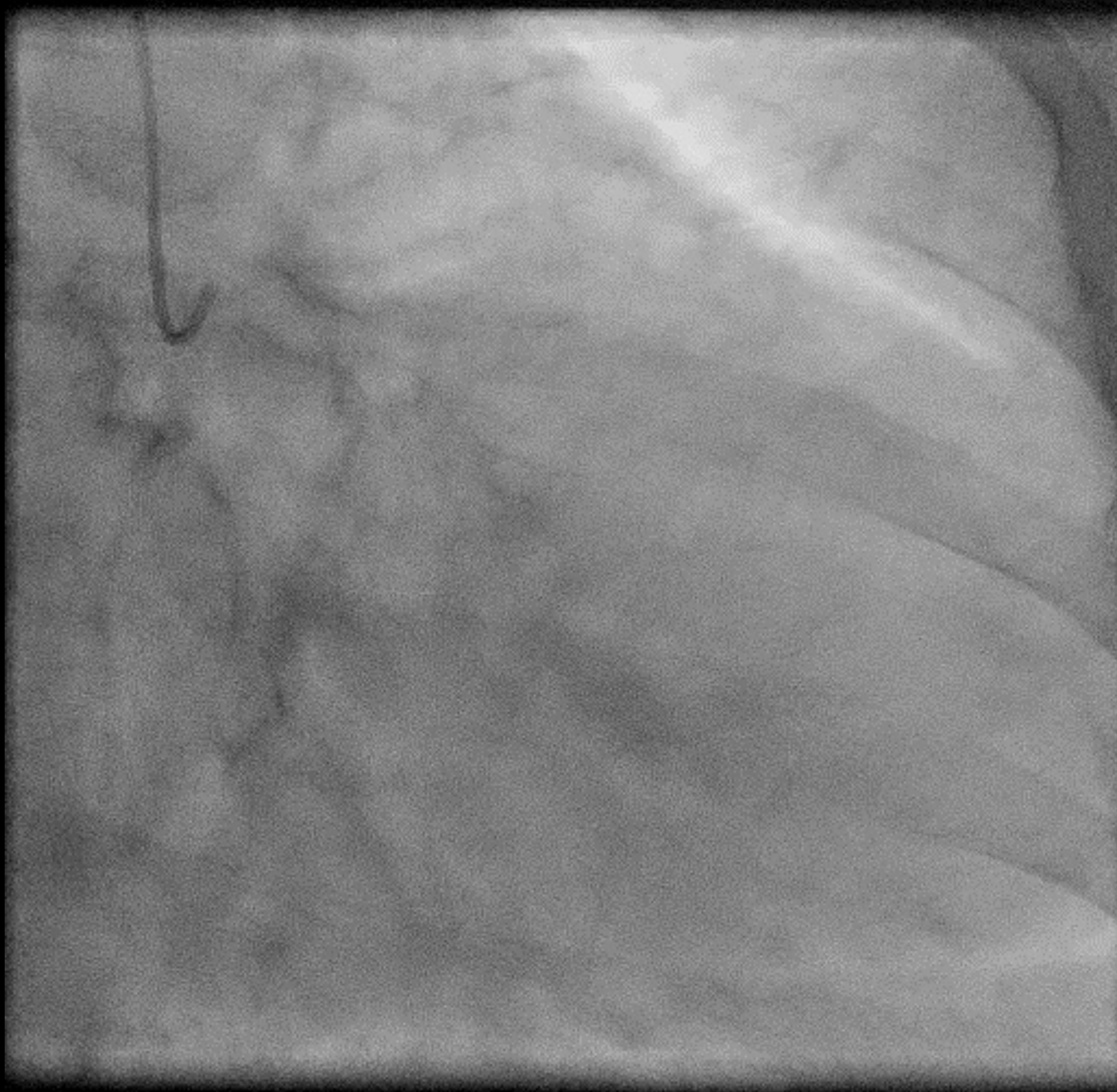
M3

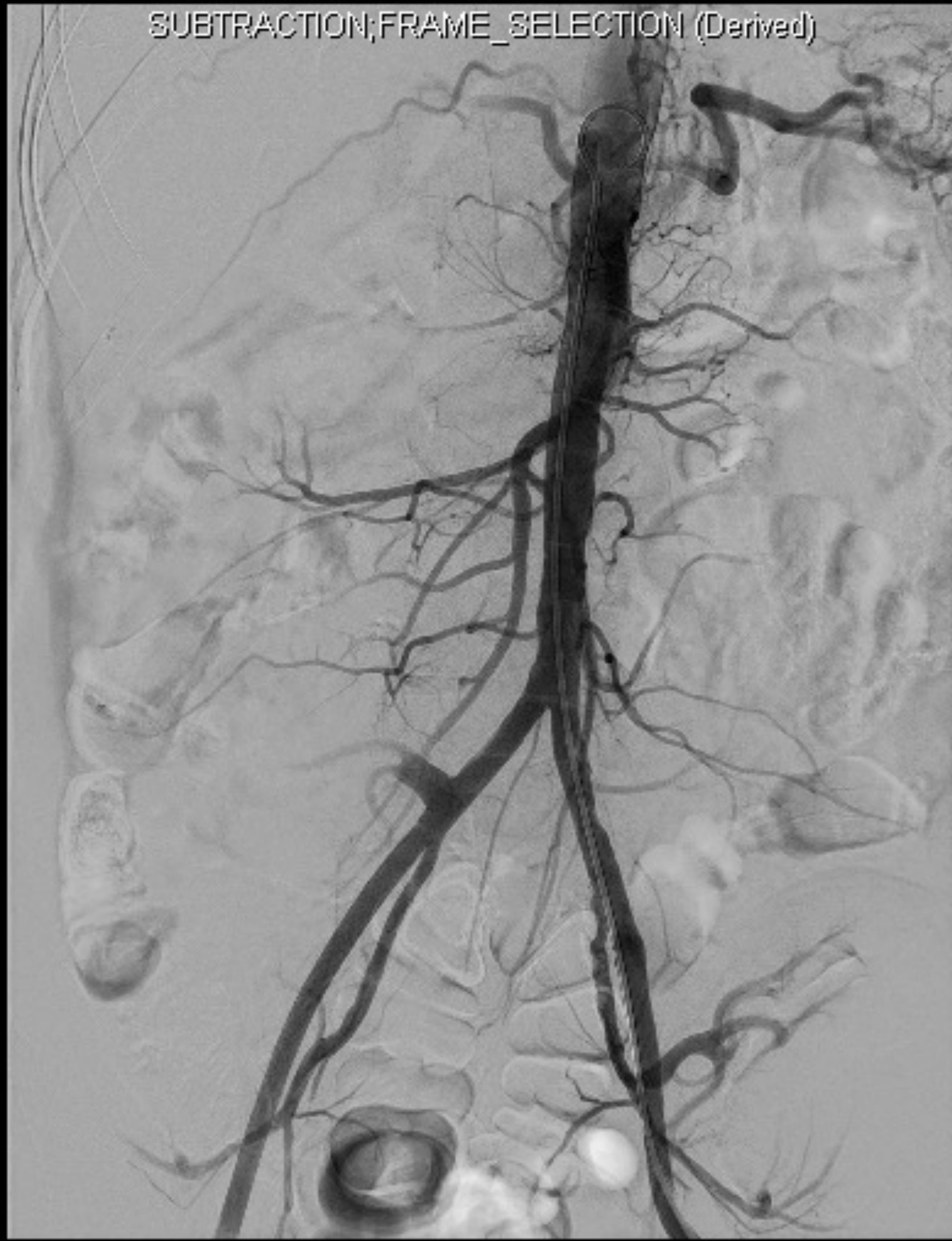
2D
67%
C 50
P Low
HPen

X2

93 bpm



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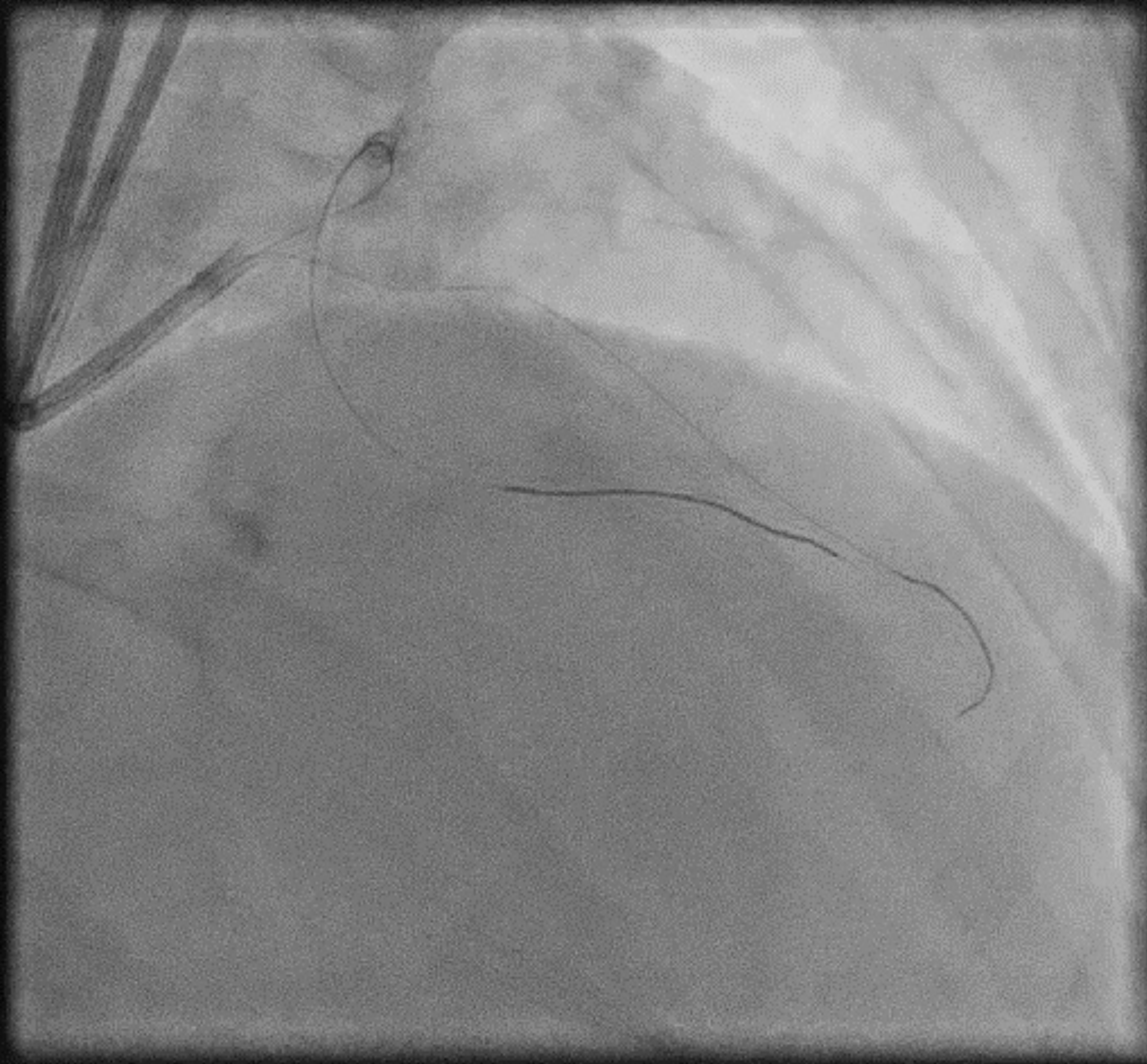
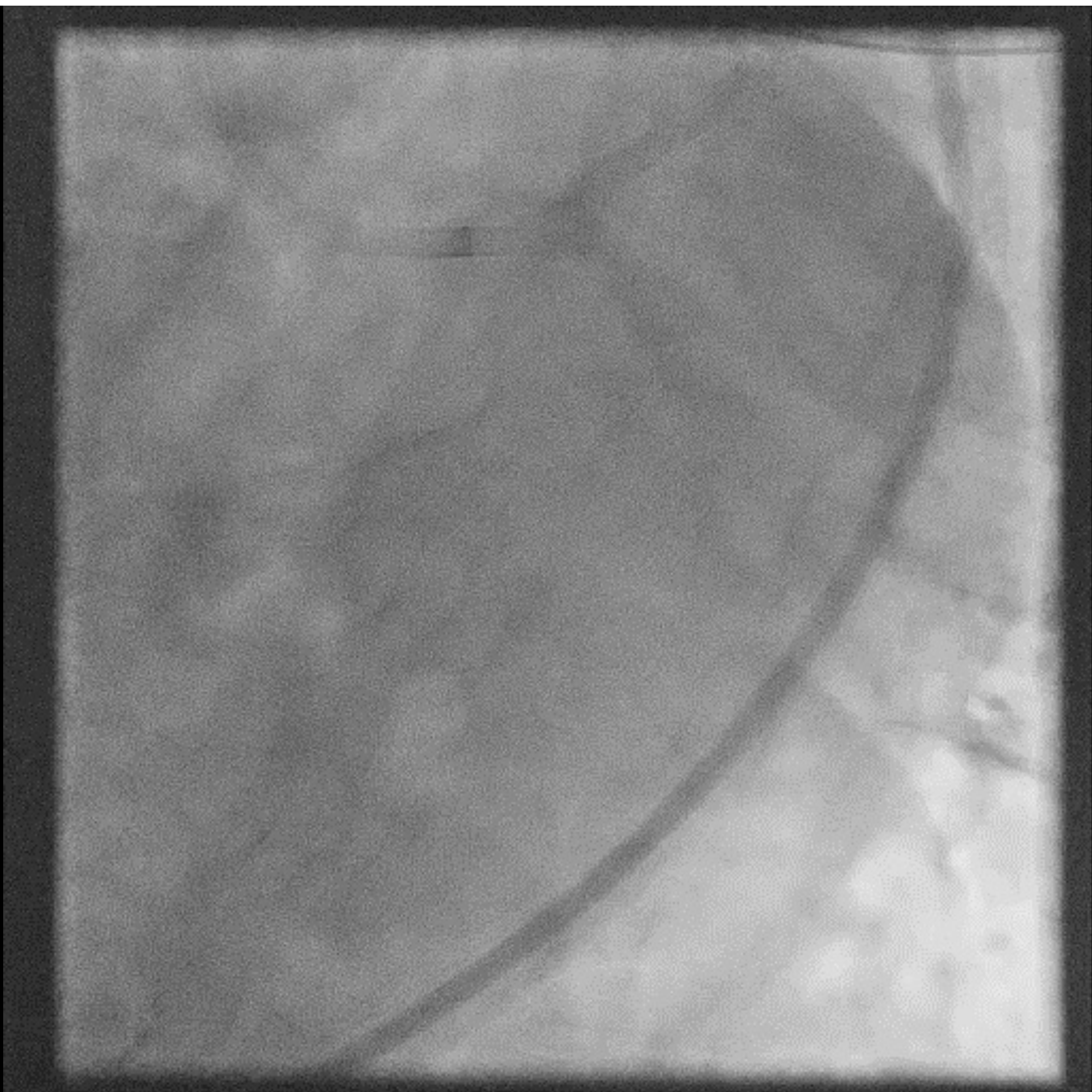




Case

HD 3
ECMO decannulation

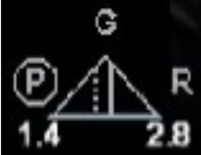
HD 15
RCA PCI

PCI

HD 8
Cr peak: 3.60 mg/dL

HD 22
Discharge

Most recent Cr 1.72 mg/dL


G
R
1.4
2.8

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

Outline

Epidemiology

Definition and diagnosis

Management

- Acute MI
- General supportive measures
- Mechanical circulatory support

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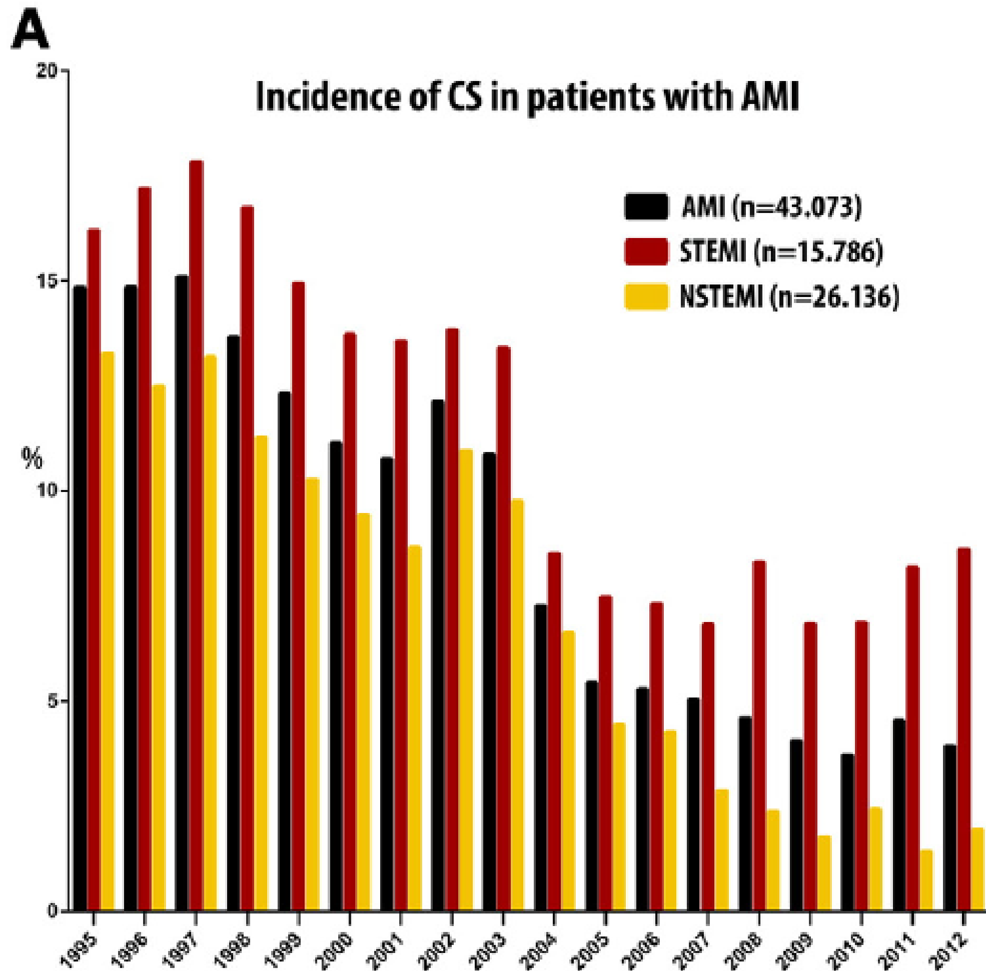
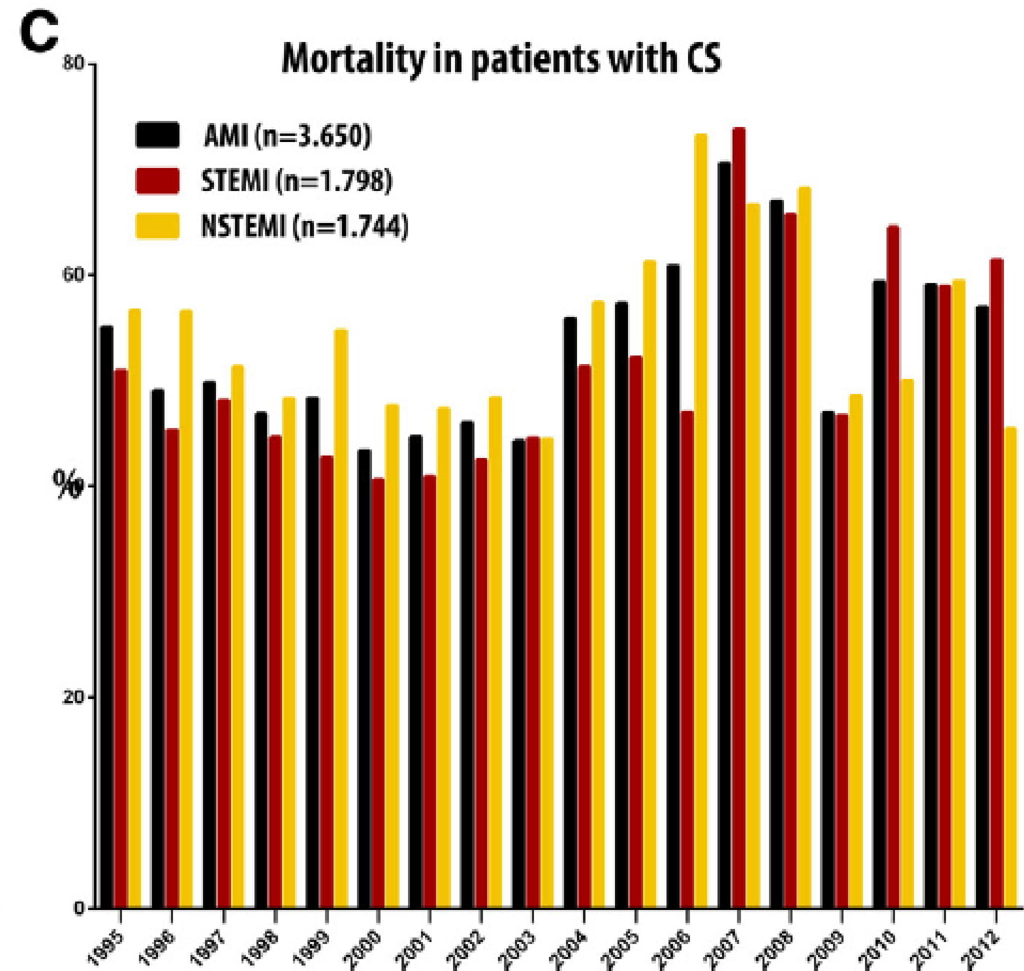
Epidemiology





Patients are older and with more comorbidities

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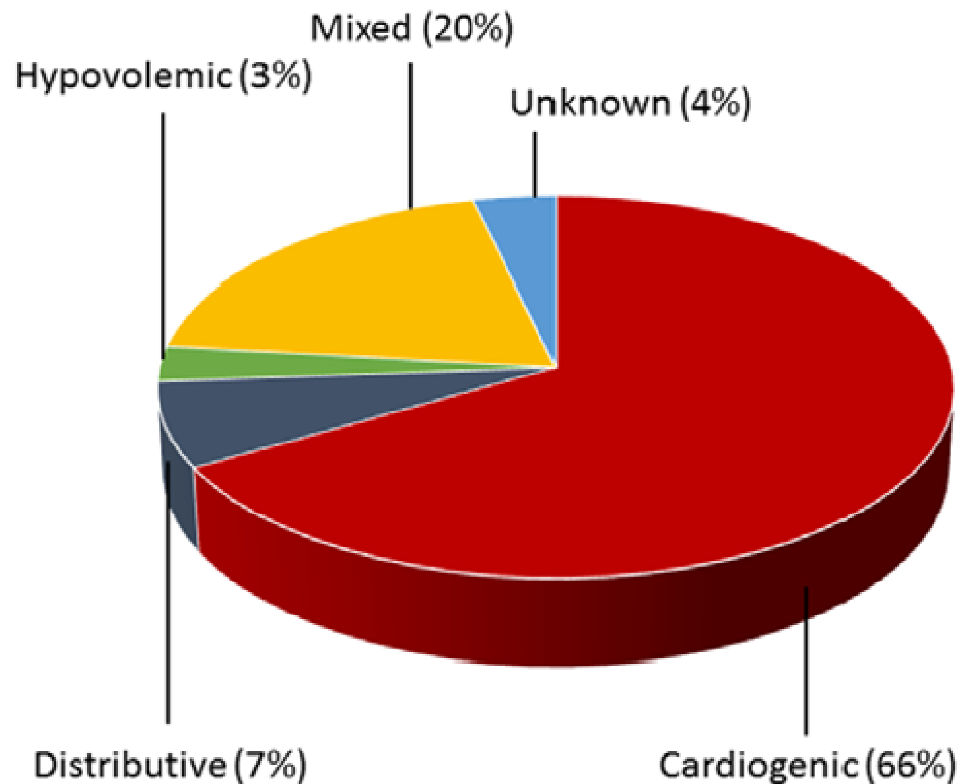
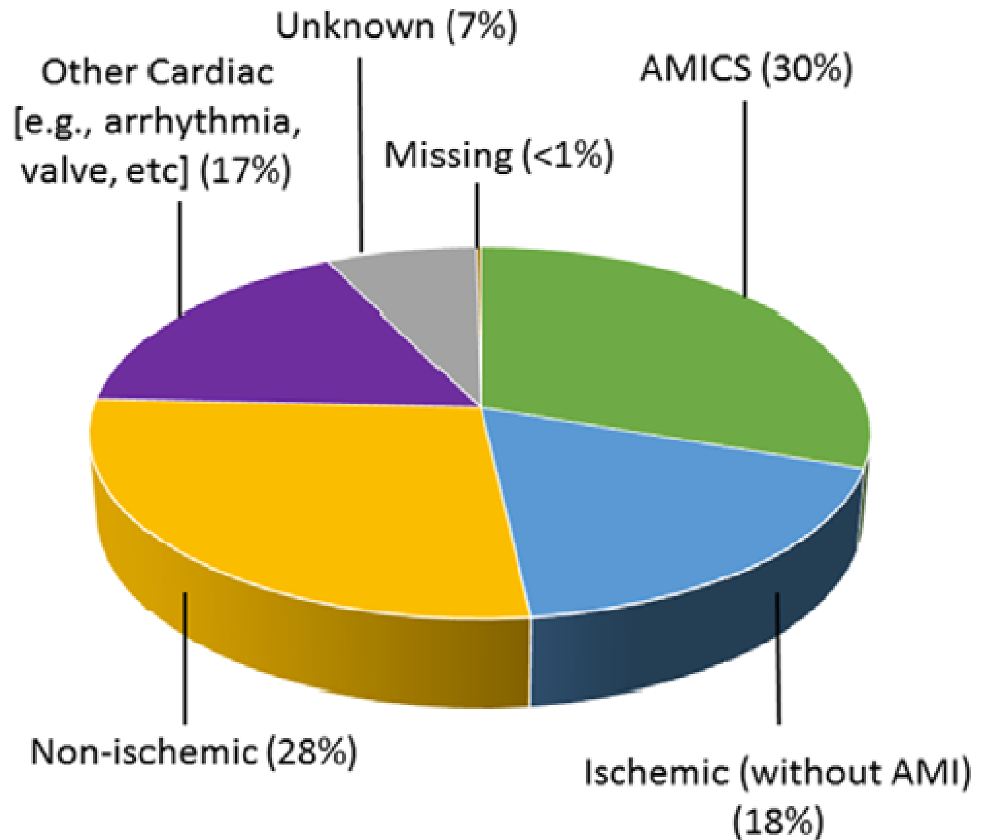
Brigham and Women's Hospital and Harvard Medical School

Redfors. Int J Cardiol. 2015.

Epidemiology





Berg. Circ Cardiovasc Qual/Outcomes. 2019.

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Definition and diagnosis

Management

- Acute MI
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Recognition

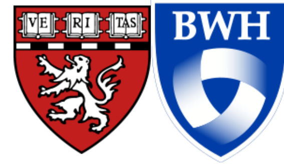

TABLE 21.4 Sensitivity and Specificity of History and Physical Examination (H&P) Components for Diagnosis of Elevated Filling Pressures in Patients with Heart Failure*

H&P FINDING	FREQUENCY	SENSITIVITY	SPECIFICITY	PREDICTIVE VALUE Positive	PREDICTIVE VALUE Negative	LR Positive	LR Negative	OR (95% CI)
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

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

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

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

Outline

Epidemiology

Definition and diagnosis

<u>Management</u>

- 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=11,145)

DICINE

ORIGINAL ARTICLE

Effect of Platelet Inhibition with Cangrelor during PCI on Ischemic Events

N=59,430

N=0 with Cardiogenic Shock

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

Trial	Follow-up	n/N	n/N	Mortality Relative Risk 95% CI	Relative Risk 95% CI
Revascularization (PCI/CABG)					
SHOCK	1-year	81/152	100/150		0.72 (0.54;0.95)
SMASH	30 days	22/32	18/23		0.87 (0.66;1.29)
Total		**103/184**	**118/173**	Early revascularization better / Medical therapy better	**0.82 (0.69;0.97)**
Vasopressors					
SOAP II (CS subgroup)	28 days	64/145	50/135	Norepinephrine better / Dopamine better	0.75 (0.55;0.93)
Inotropes					
Unverzagt et al.	30 days	5/16	10/16	Levosimendan better / Control better	0.33 (0.11;0.97)
Glycoprotein IIb/IIIa-inhibitors					
PRAGUE-7	In-hospital	15/40	13/40	Up-stream abciximab better / Standard treatment better	1.15 (0.59;2.27)
Nitric oxide synthase inhibitors					
TRIUMPH	30 days	97/201	76/180		1.14 (0.91;1.45)
SHOCK-2	30 days	24/59	7/20		1.16 (0.59;2.69)
Cotter et al	30 days	4/15	10/15		0.40 (0.13;1.05)
Total		**125/275**	**93/215**	Nitric oxide synthase inhibition better / Placebo better	**1.05 (0.85;1.29)**
IABP					
IABP-SHOCK I	30 days	7/19	6/21		1.28 (0.45;3.72)
IABP-SHOCK II	30 days	119/300	123/298		0.96 (0.79-1.17)
Total		**126/319**	**129/319**	IABP better / Standard treatment better	**0.98 (0.81;1.18)**
LVAD					
Thiele et al	30 days	9/21	9/20		0.95 (0.48;1.90)
Burkhoff et al	30 days	9/19	5/14		1.33 (0.57-3.10)
Seyfarth et al	30 days	6/13	6/13		1.00 (0.44-2.29)
Total		**24/53**	**20/47**	LVAD better / IABP better	**1.06 (0.68-1.66)**

Axis: 0 0.25 0.5 0.75 1 1.5 2 2.5 3

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*…

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- *Et cetera*…

Acute MI complicated by shock

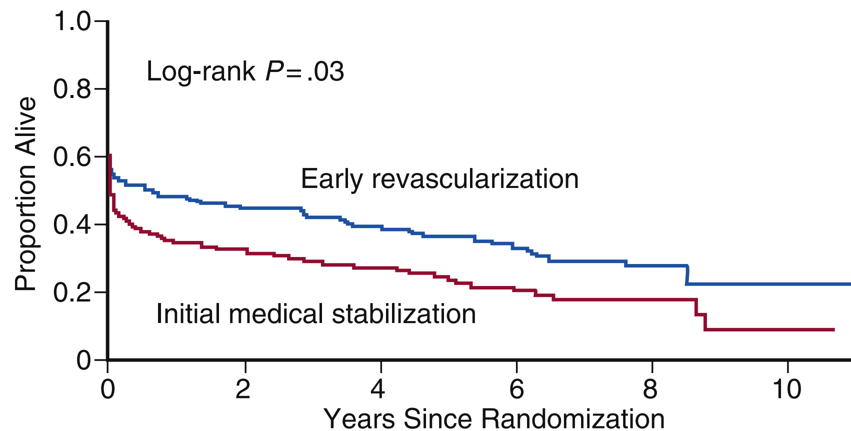
Early revascularization

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

Relative risk

- **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

Non-culprit lesions in STEMI

Table 2 Procedural characteristics of patients with STEMI and CS

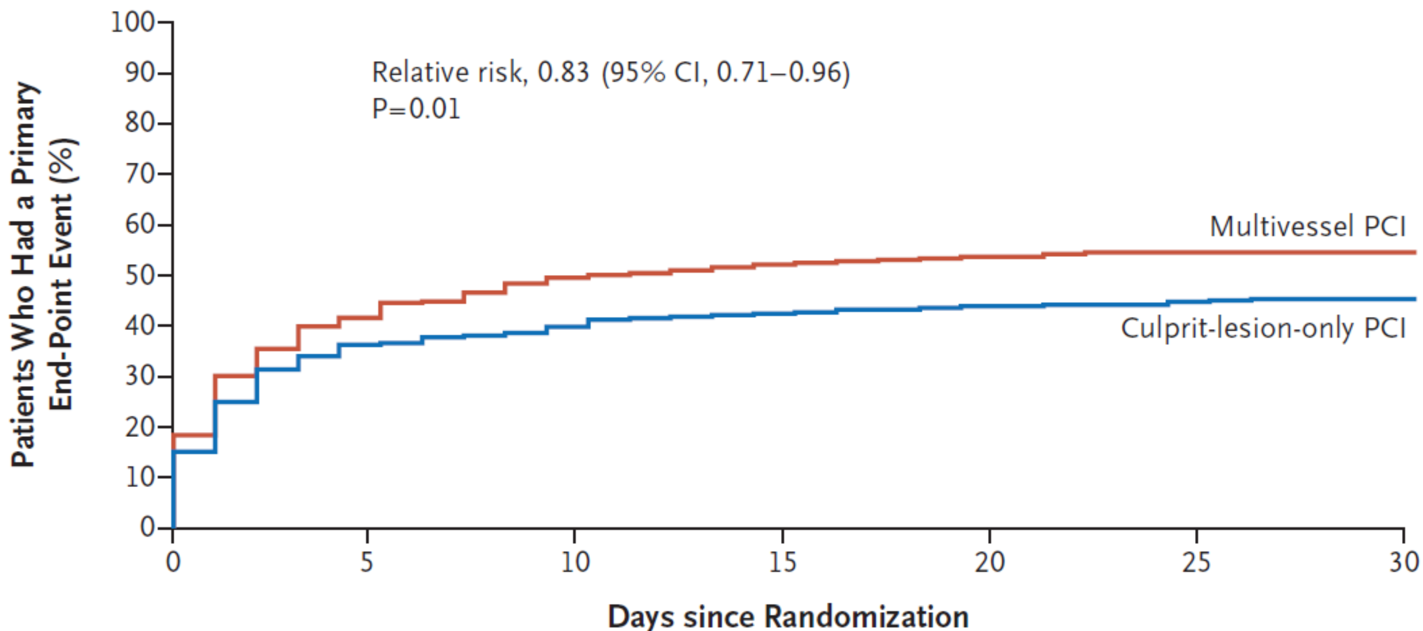
	2006–2009 ($n=302$)	2010–2013 ($n=301$)	2014–2016 ($n=378$)	p value
Left main disease, n (%)	50 (17)	43 (14)	44 (12)	0.118
One-vessel disease, n (%)	77 (26)	93 (31)	117 (31)	0.255
Two-vessel disease, n (%)	89 (29)	84 (28)	116 (31)	0.732
Three-vessel disease, n (%)	133 (44)	124 (41)	145 (38)	0.326

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



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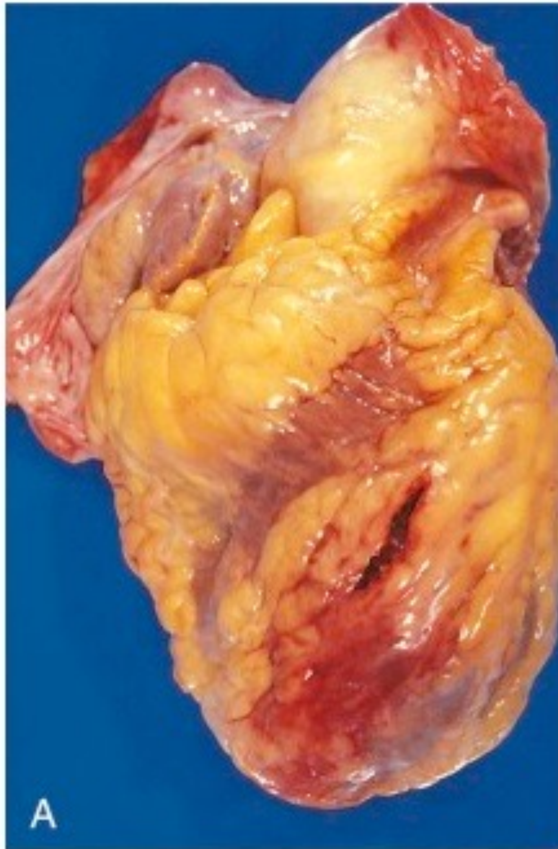
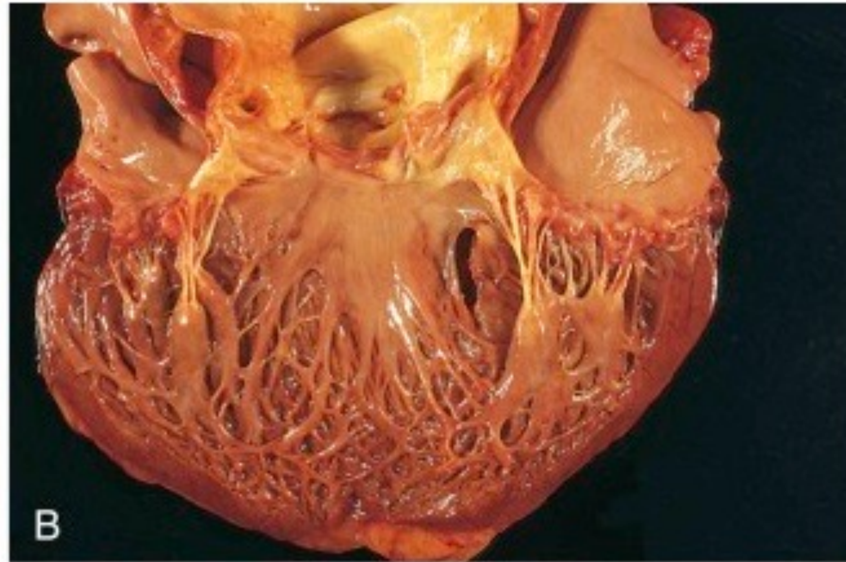
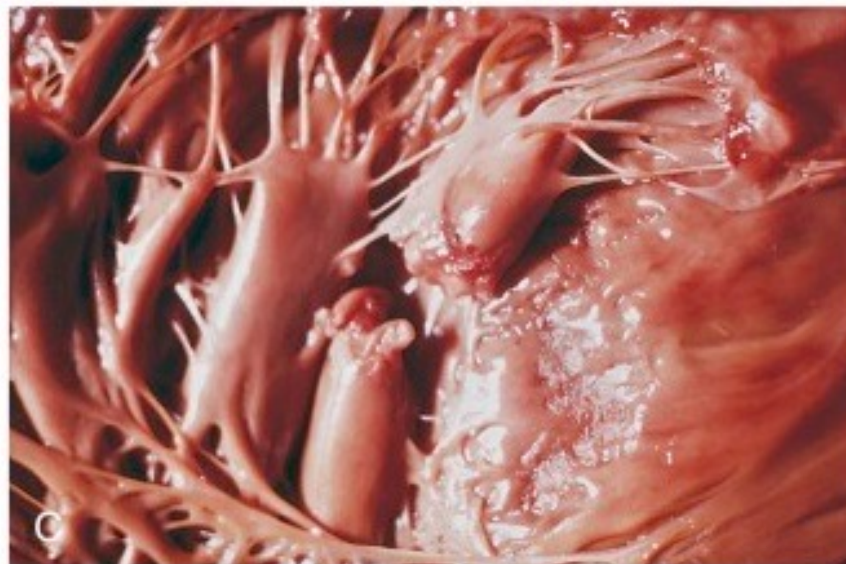
Thiele. NEJM. 2017.

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

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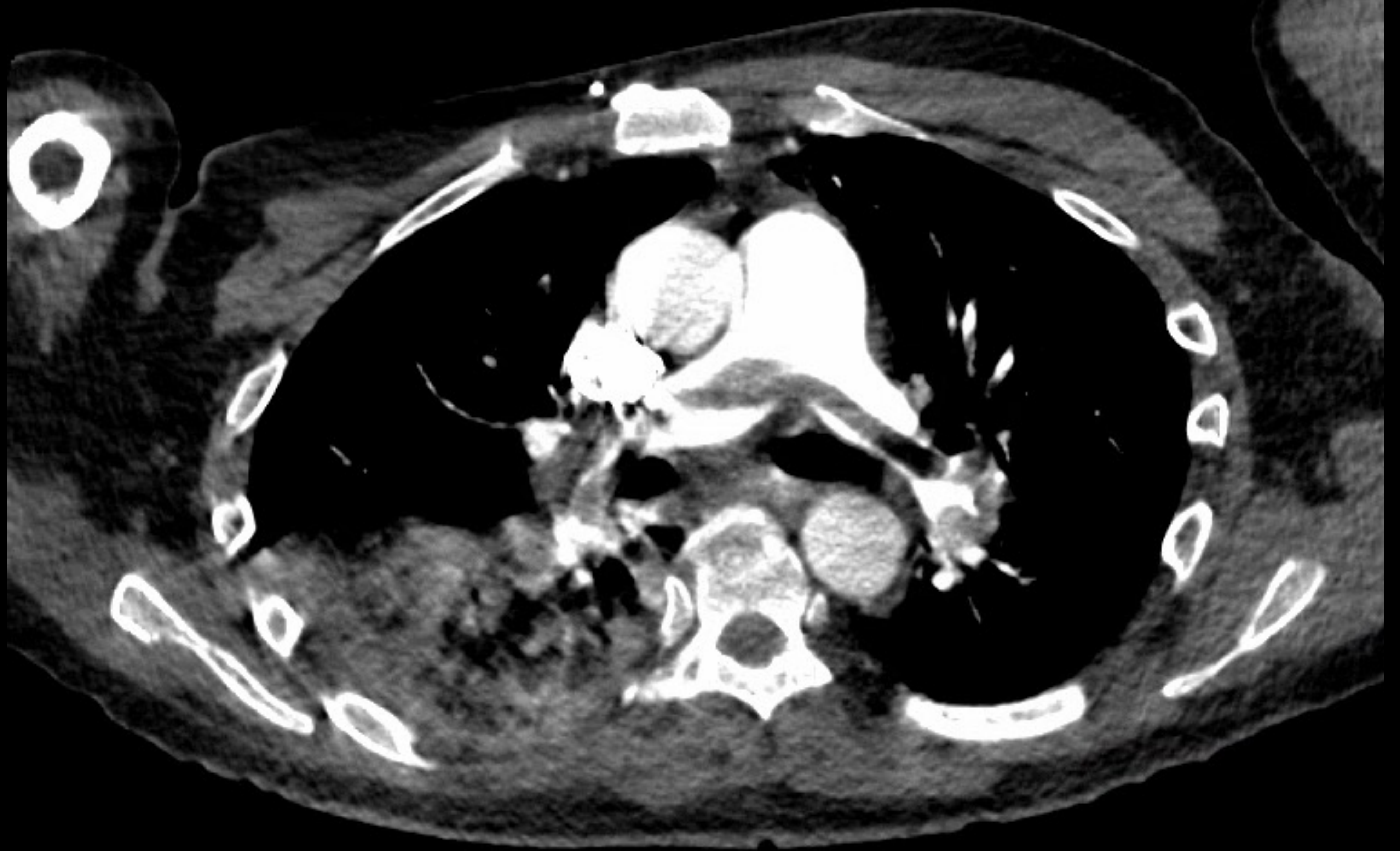

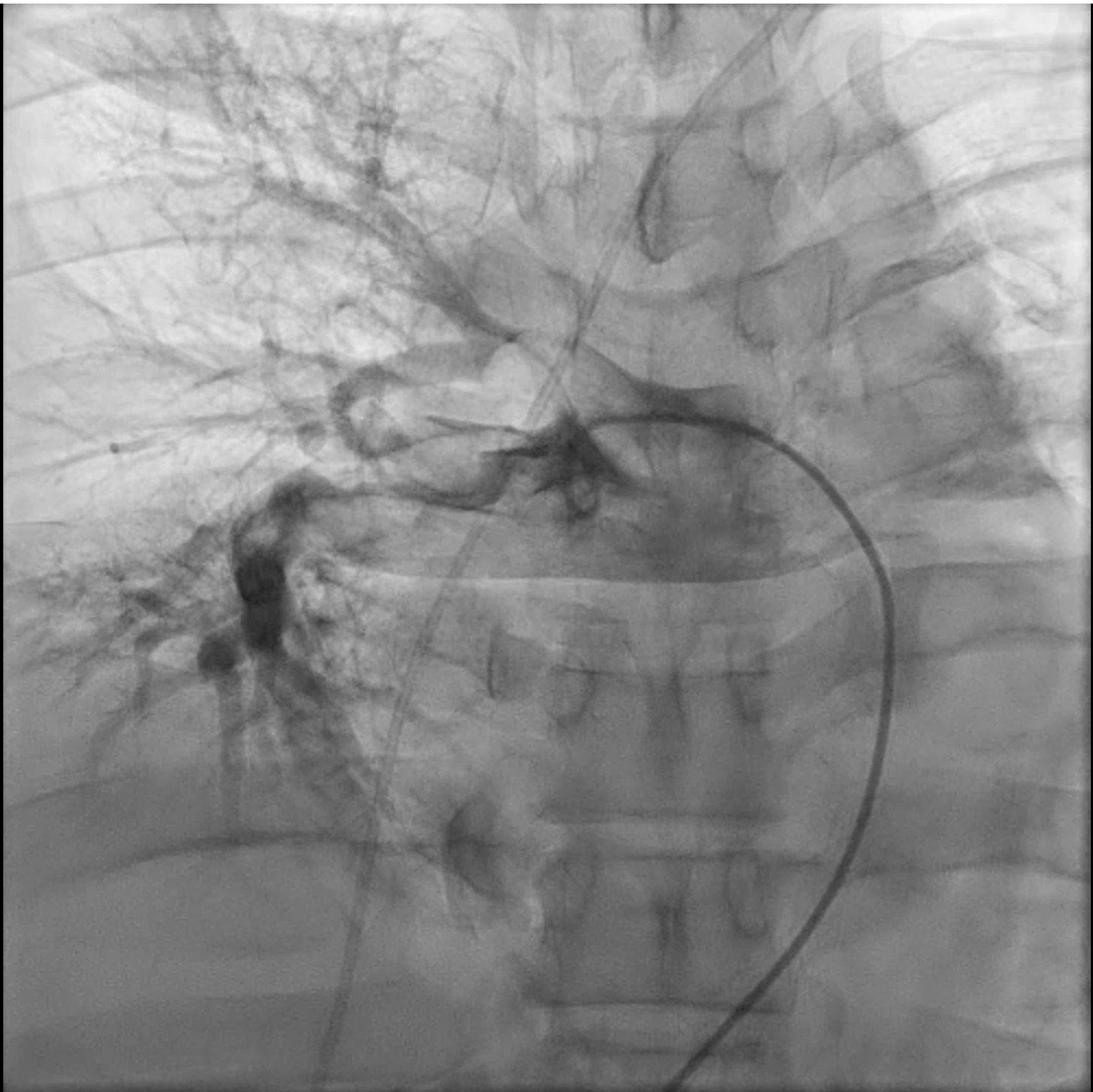

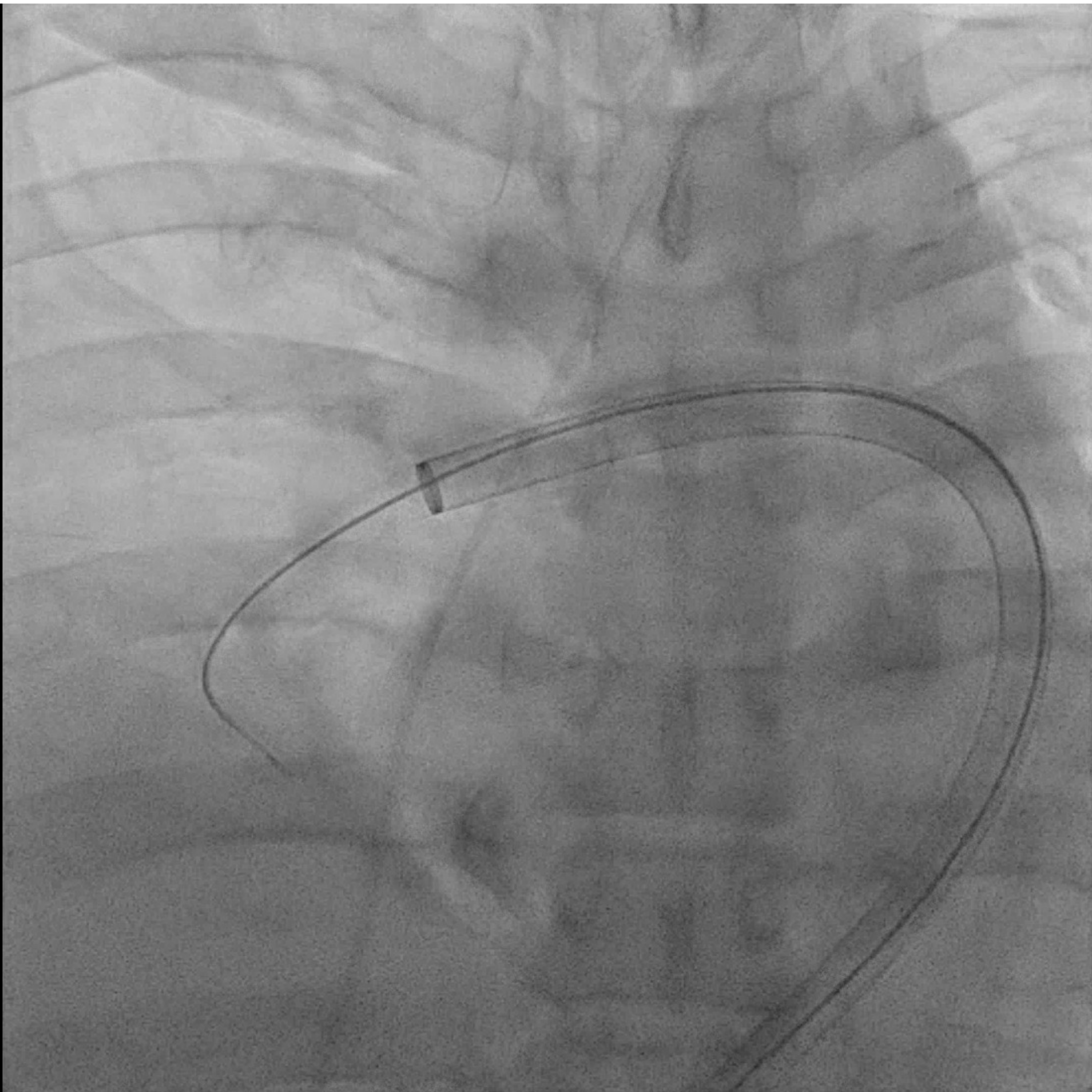

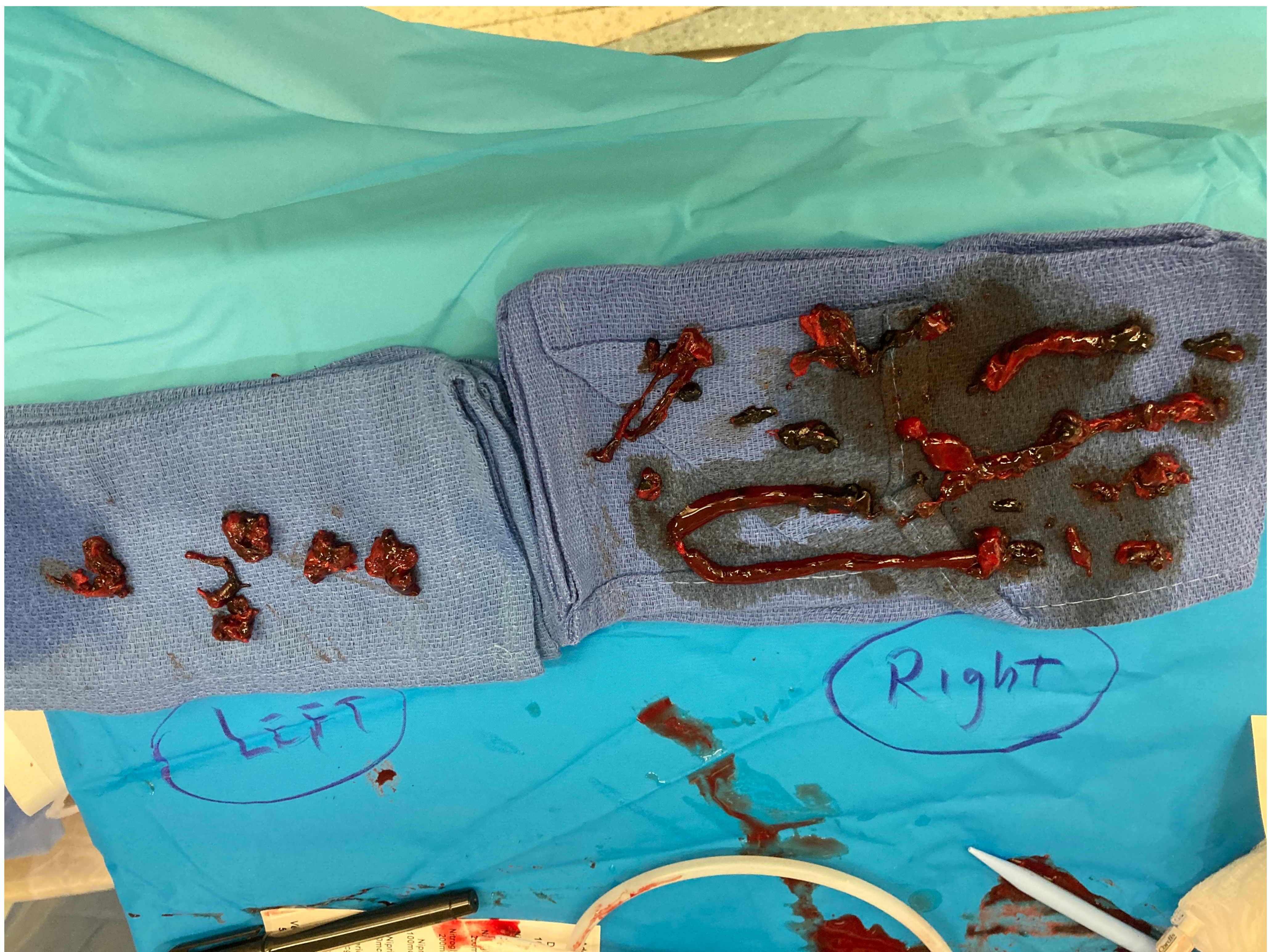
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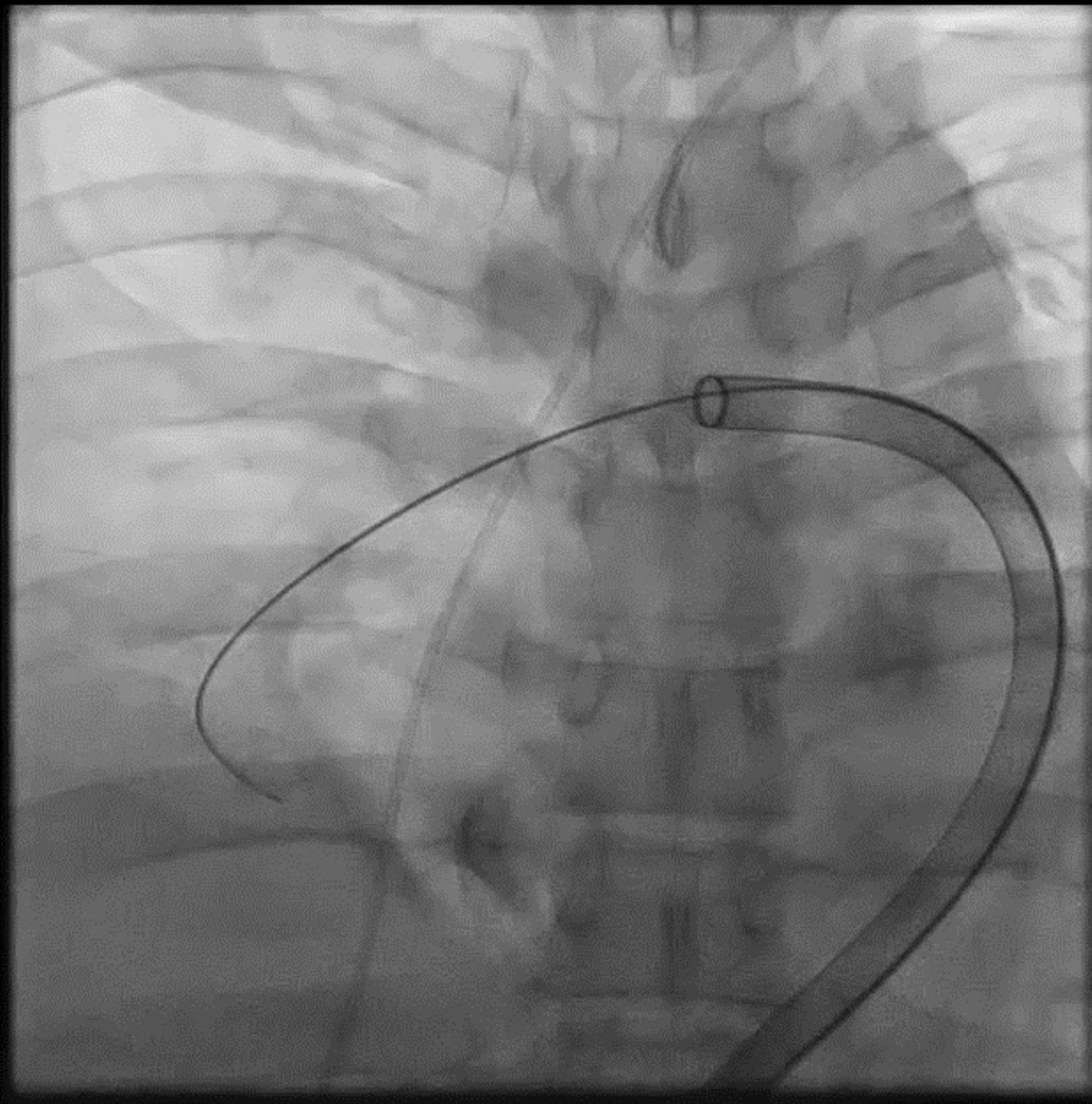

Outline

Epidemiology

Definition and diagnosis

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- Acute MI
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- Mechanical circulatory support

For mild to moderate shock

↑ Cardiac output

↓ **Resistance**

↓ **Filling pressures**

Inotrope

Vasodilator + Diuretic

Vasoactive therapies

Pure vasopressors – Incr SVR

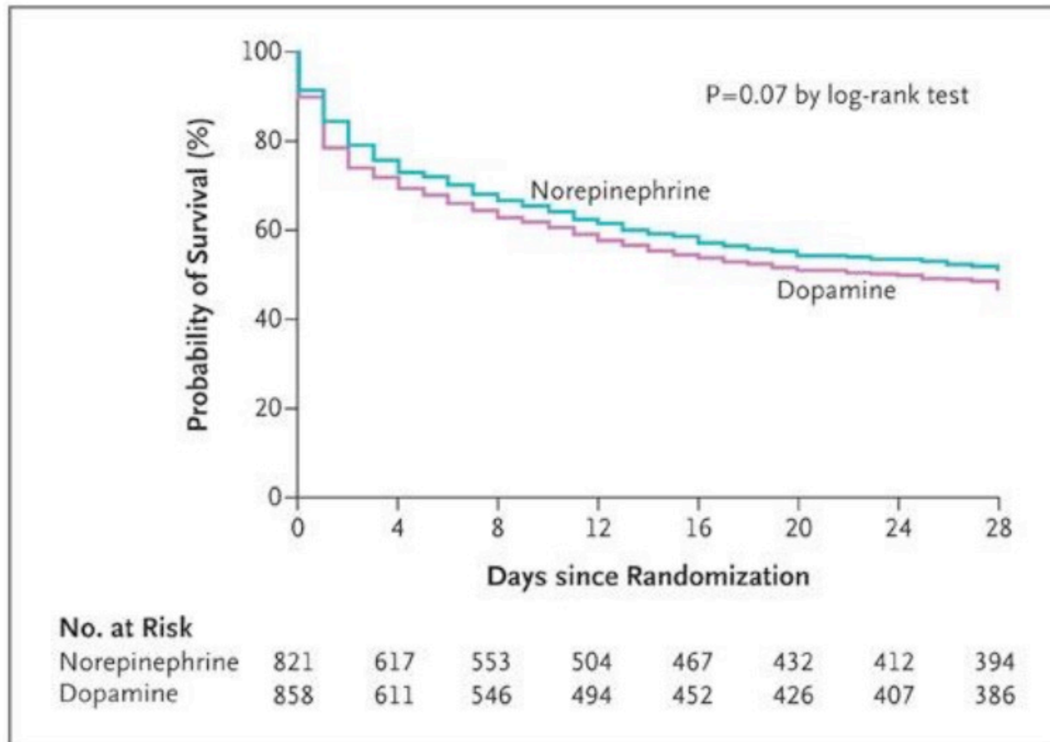
Inopressors – Incr CO, Incr SVR

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

Slide courtesy of Dr. Erin Bohula

SOAP II: Dopamine vs Norepinephrine

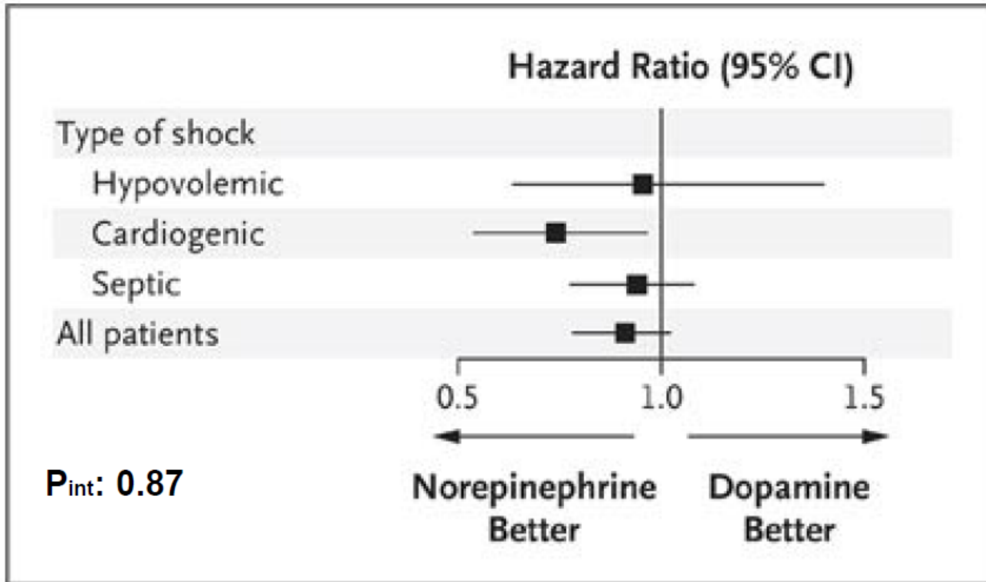


De Backer et al. NEJM 2010;362:779.

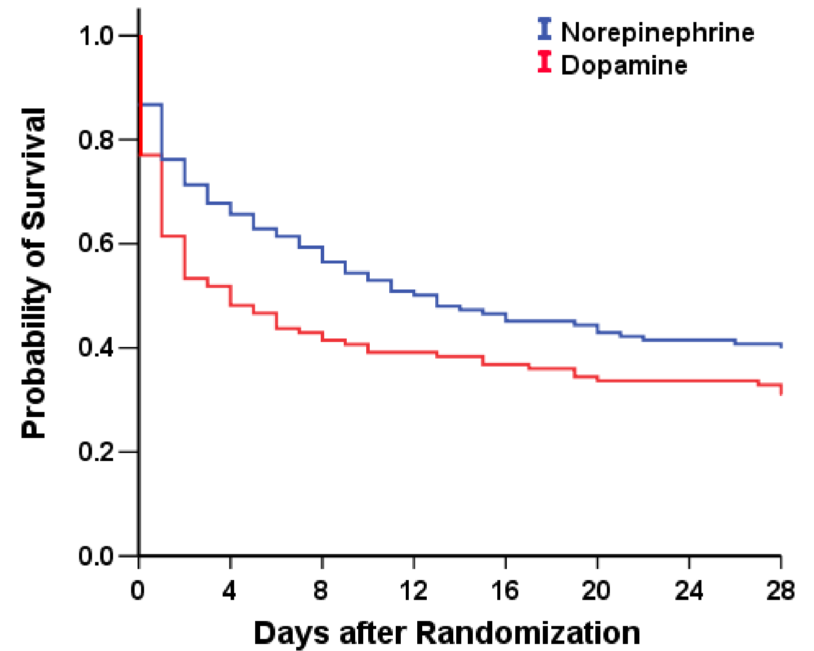
- ***28d mortality:***
  - ***52.5% for DA vs 48.5% for norepi***
  - ***OR 1.17 (0.97-1.42), p=0.10***
- ***Arrhythmias: 24.1% vs 12.4%***

Slide courtesy of Dr. Erin Bohula

SOAP II: Dopamine vs Norepinephrine



Cardiogenic Shock (N=280)



Signal of harm with dopamine?

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

Slide courtesy of Dr. Erin Bohula

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 (often norepinephrine)**
- **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

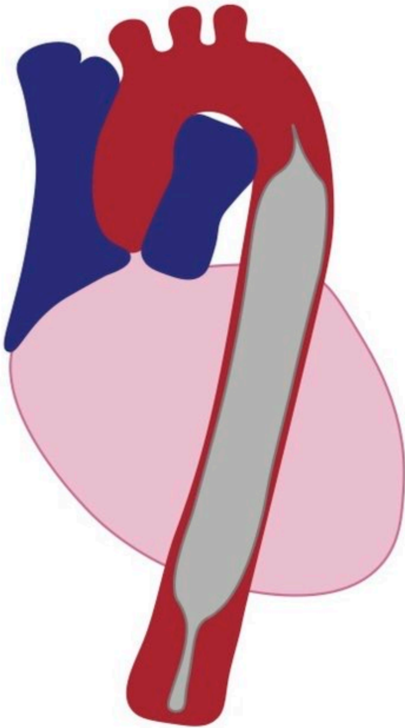
Complex Decisions

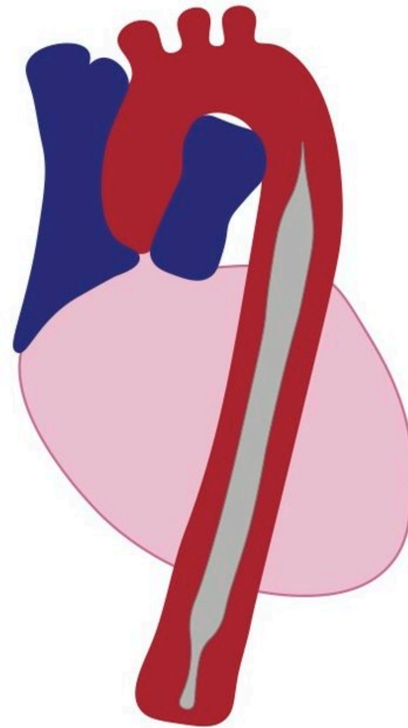
Shock Team

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)



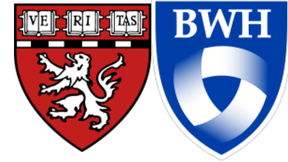


(+)

Rapid placement
Lower profile than other MCS options
Axillary possible

(-)

Minimal support



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Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

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IABP-SHOCK II

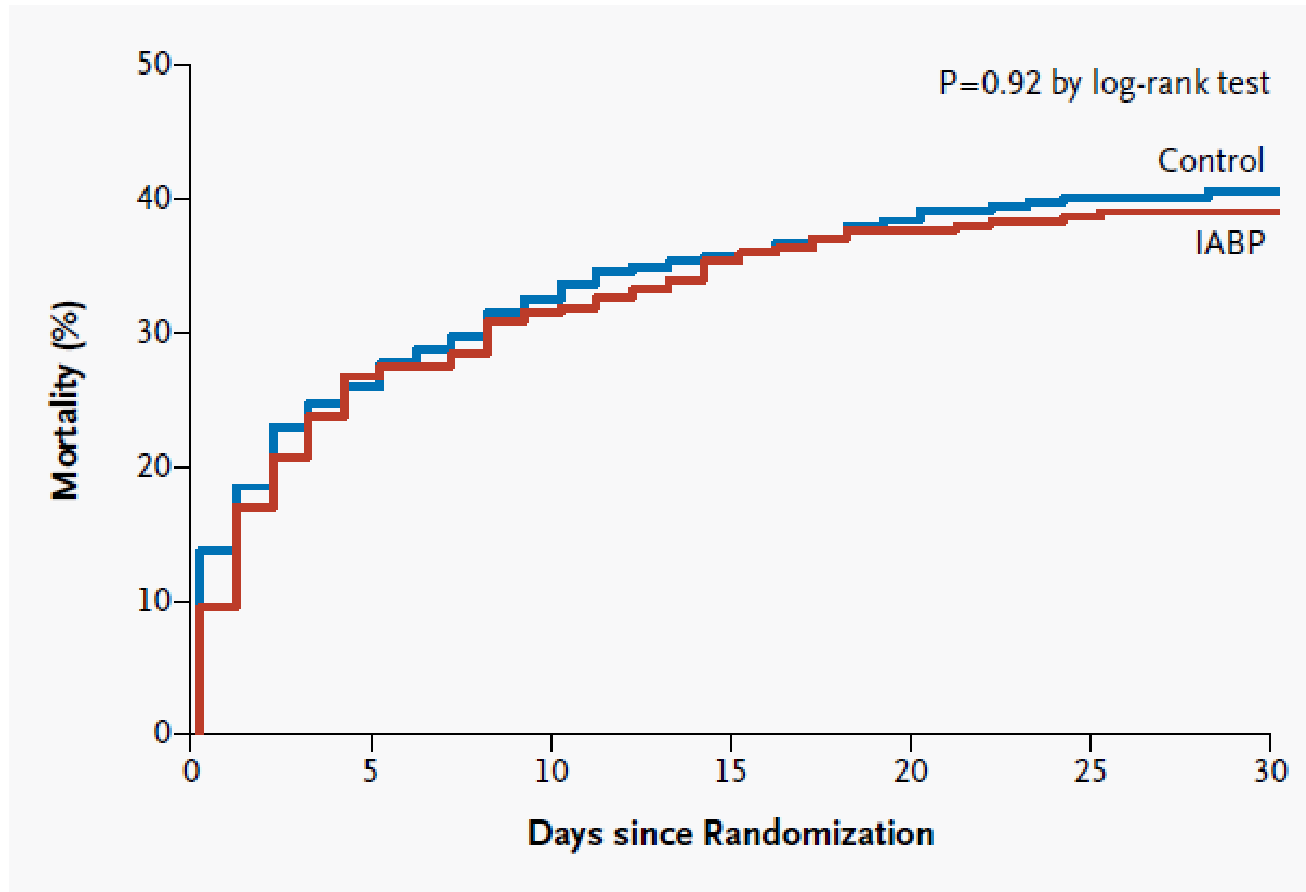
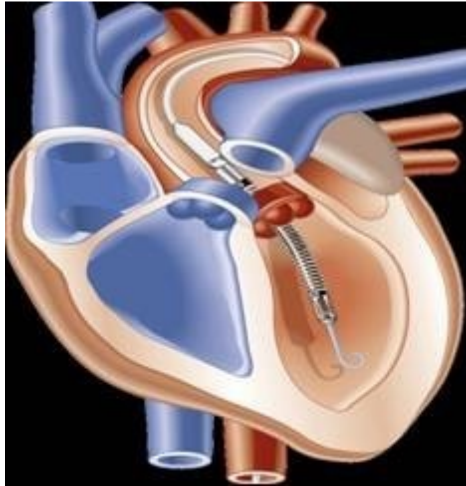
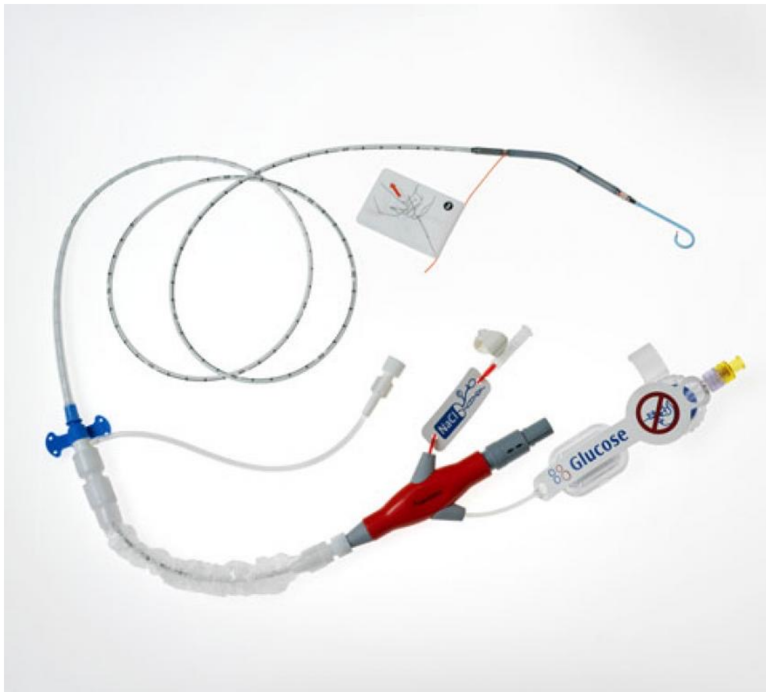


Figure 1. **Time-to-Event Curves for the Primary End Point.**
Time-to-event curves are shown through 30 days after randomization for the primary end point of all-cause mortality. Event rates represent Kaplan–Meier estimates.

Thiele. NEJM. 2012.

Impella CP





(+)

Good support (3.5 L/min)

Typically rapid placement

Unloads LV

Axillary possible

(-)

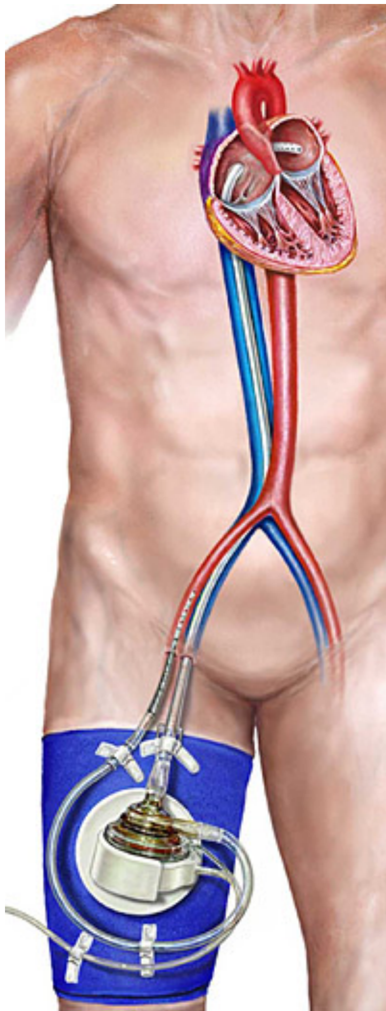
Migrates

Thrombocytopenia/hemolysis

Vascular injury

Note: Impella 5.5 also available (ax/transAo)

TandemHeart



(+)

Robust support (4-5 L/min)

Possible to add gas exchange to circuit

Migration is unusual

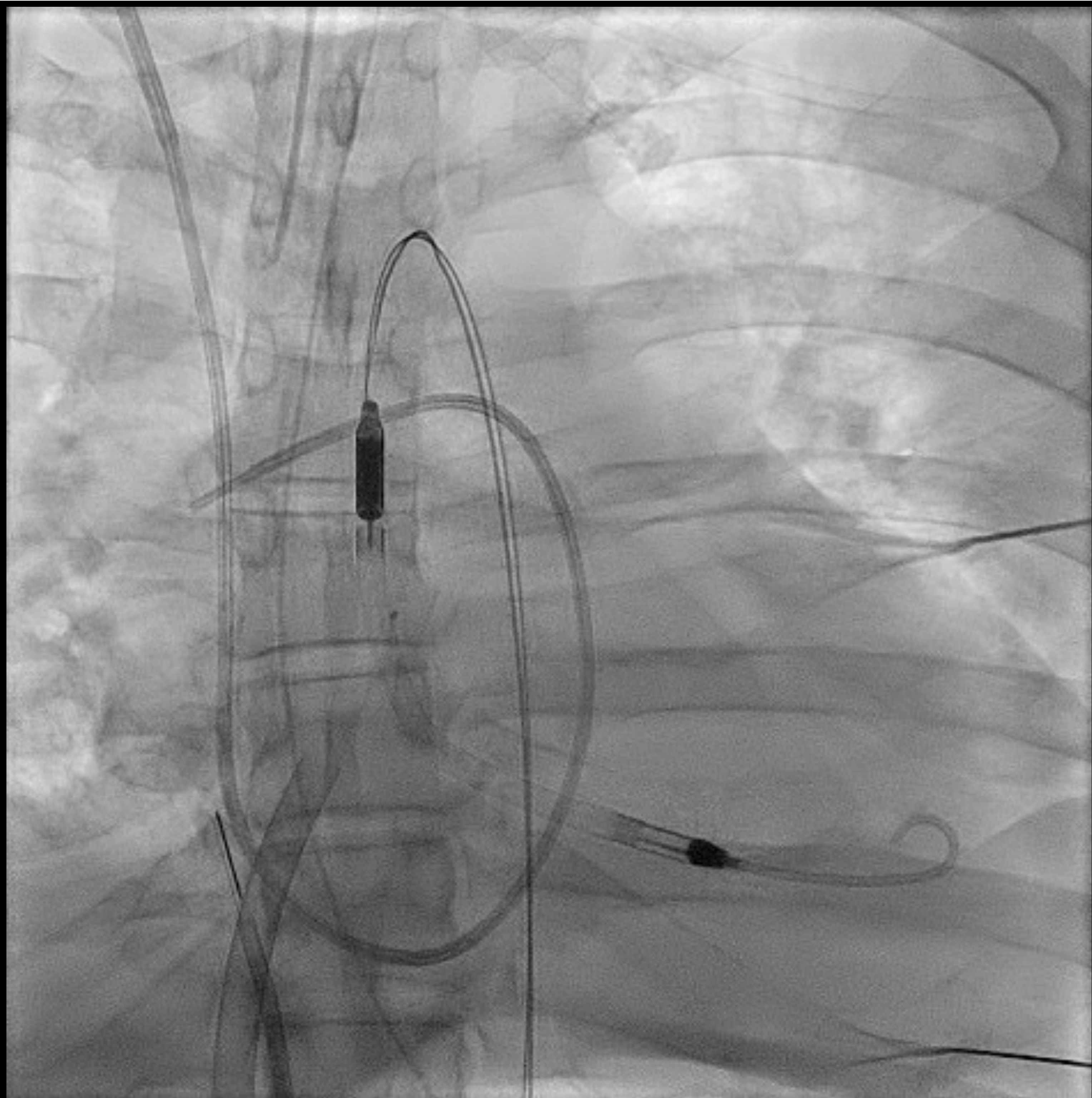
(-)

Limited availability

Requires transeptal puncture

Imperfect LV unloading

Vascular injury

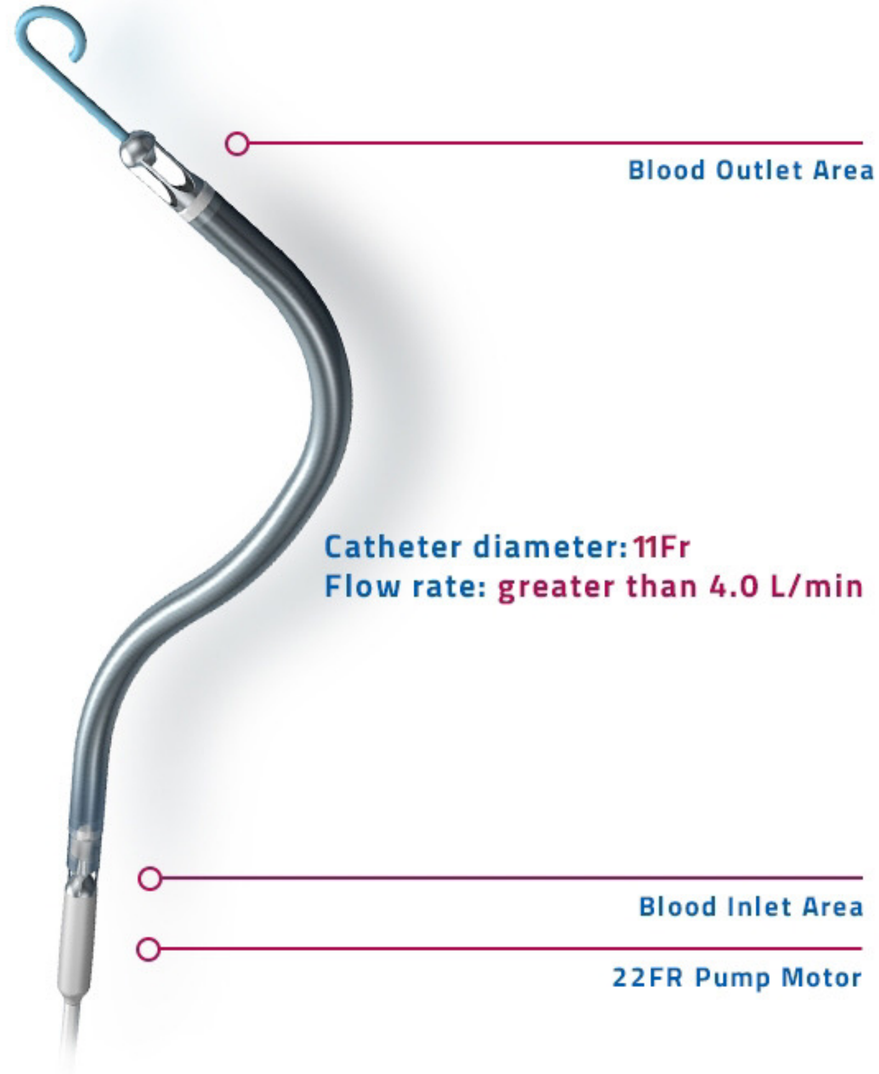


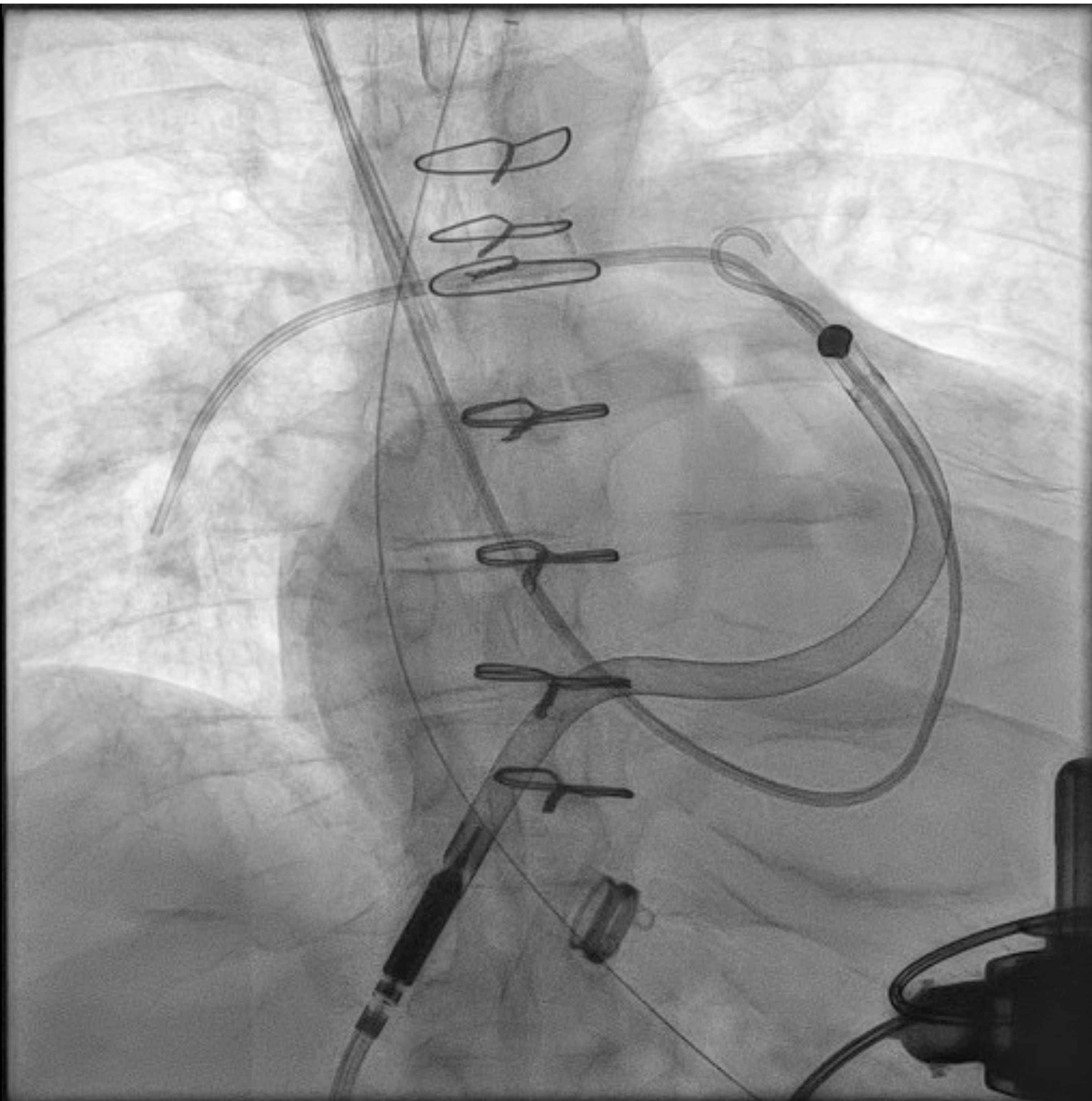
RV Support

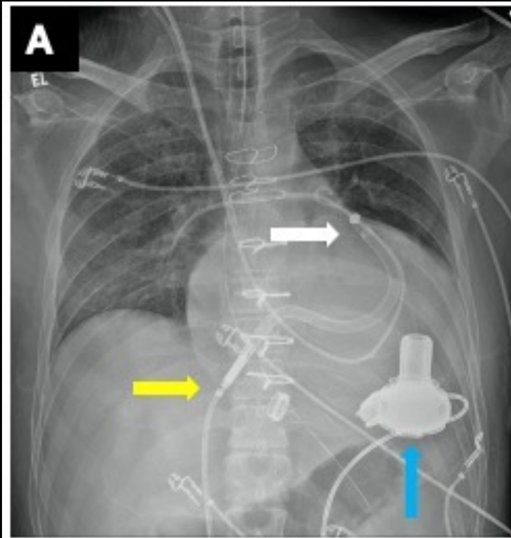
Impella RP

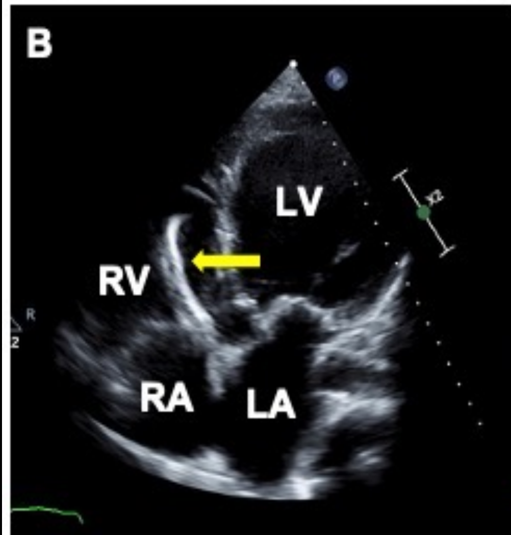
(+)
4 L/min
Typically fast placement

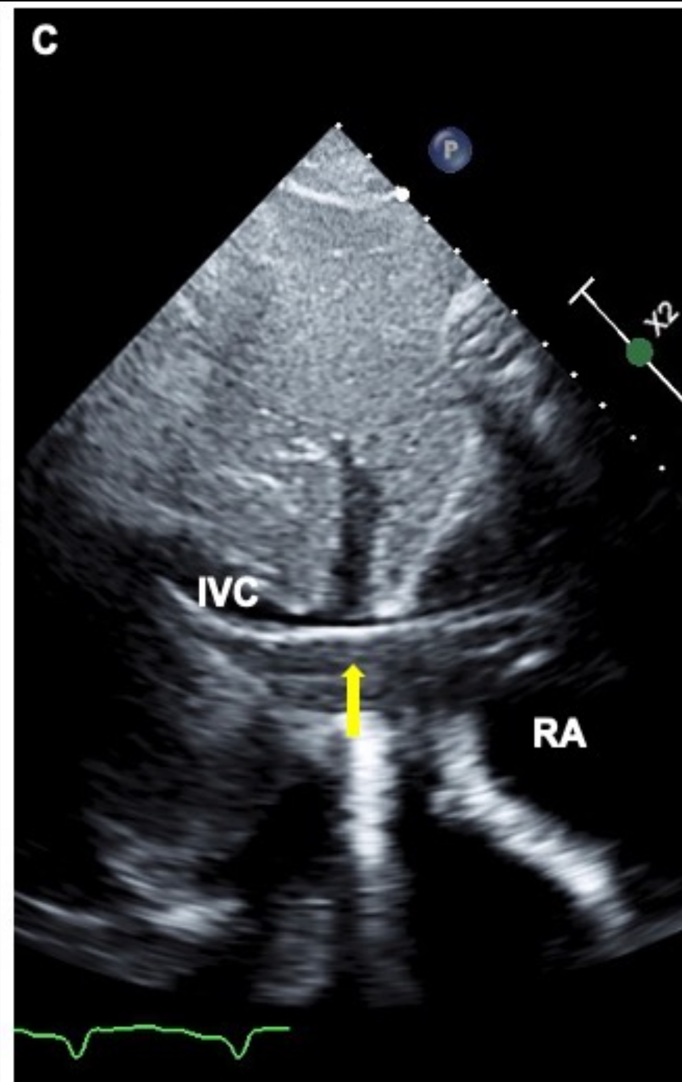
(-)
Femoral only
Migrates
Thrombocytopenia /hemolysis

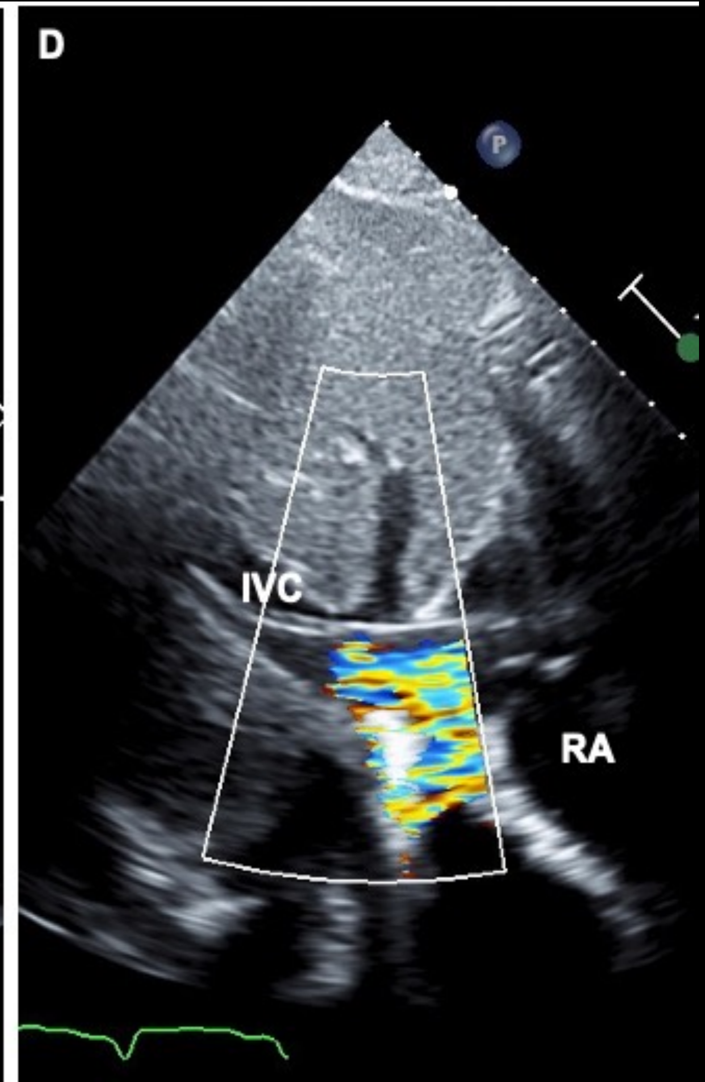










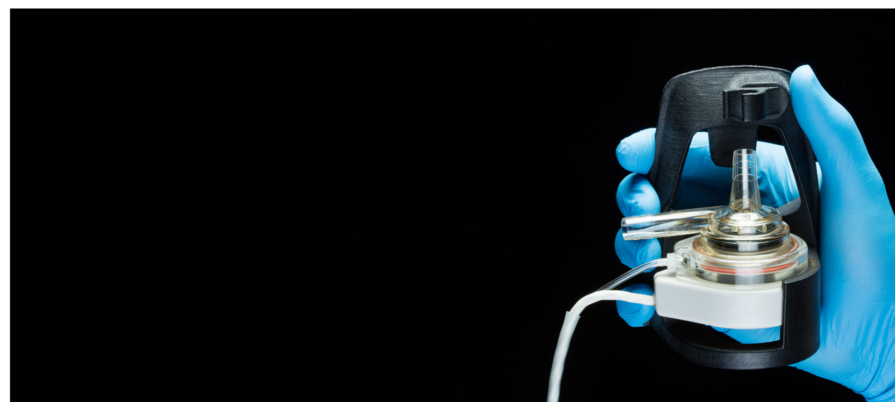


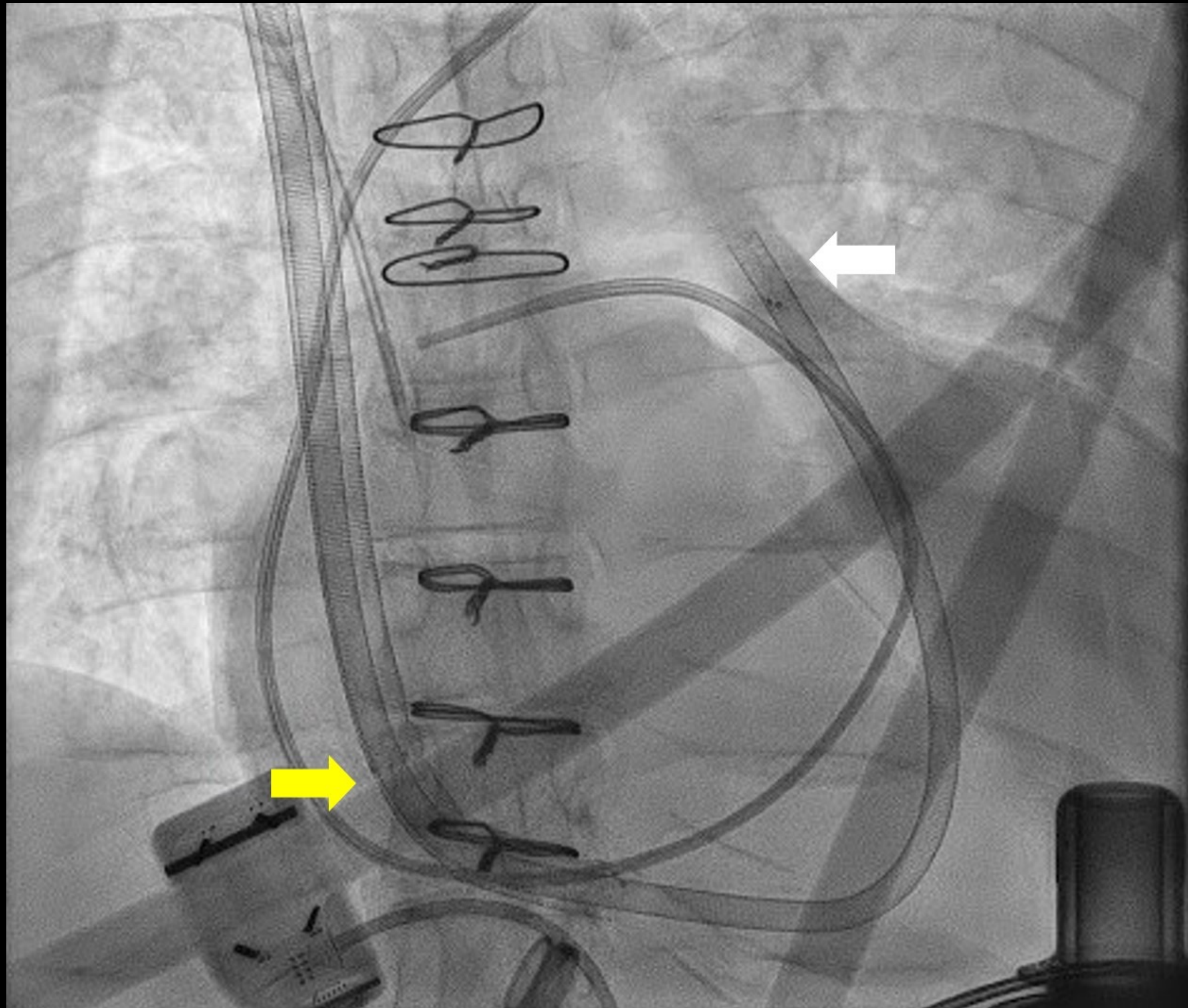
Bergmark and Morrow. Mechanical Support for the Right Ventricle. In Press.

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

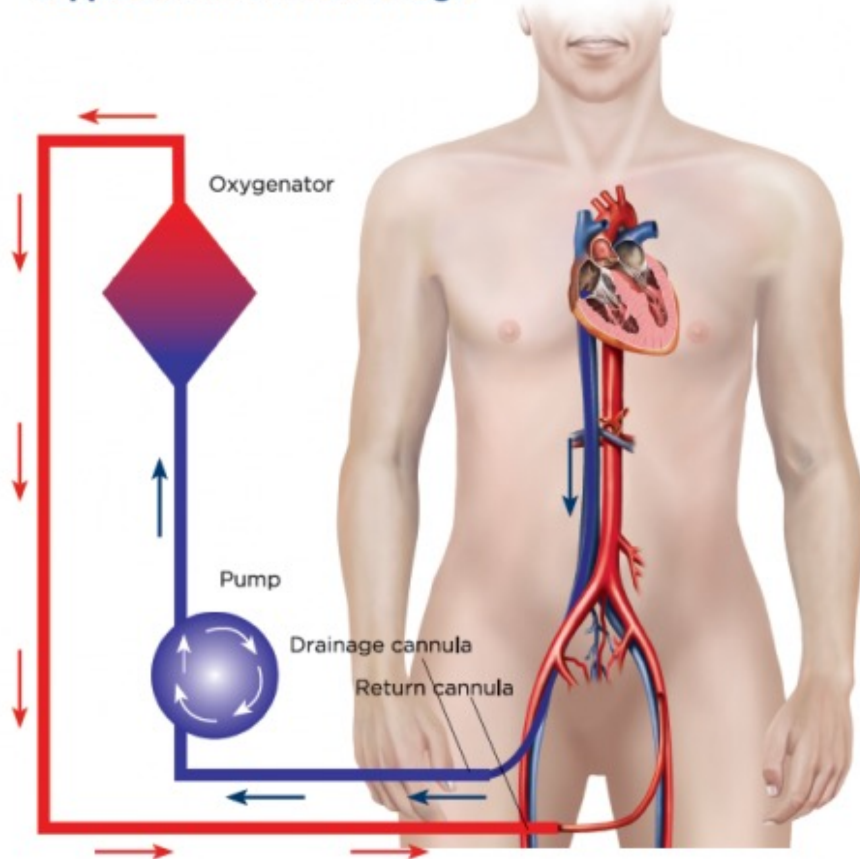




Bergmark and Morrow. Mechanical Support for the Right Ventricle. In Press.

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…)

VA-ECMO, matched
ECMELLA

Probability of any death

100%
75%
50%
25%
0%

HR 0.79 (95% CI 0.63-0.98, p=0.03)

Circulation

Time (days)

Number at risk

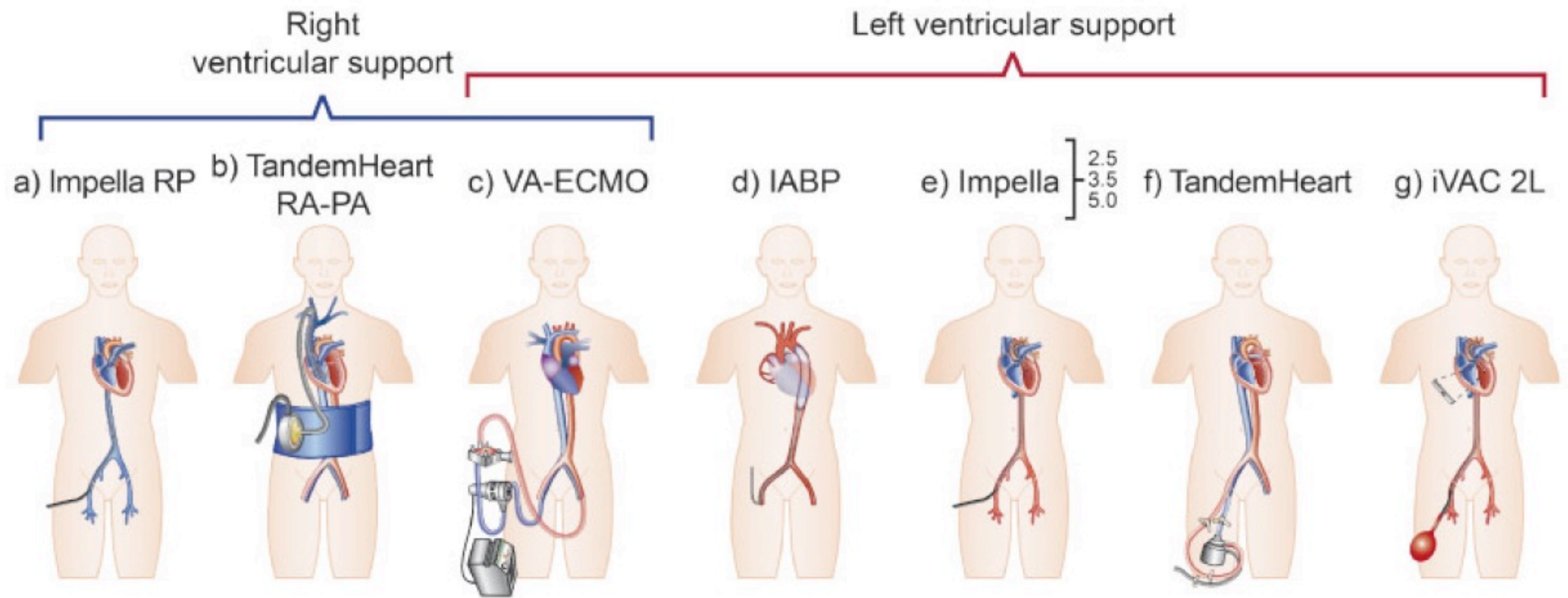
	0	10	20	30
VA-ECMO, matched	255	132	89	76
ECMELLA	255	165	115	88

Time (days)

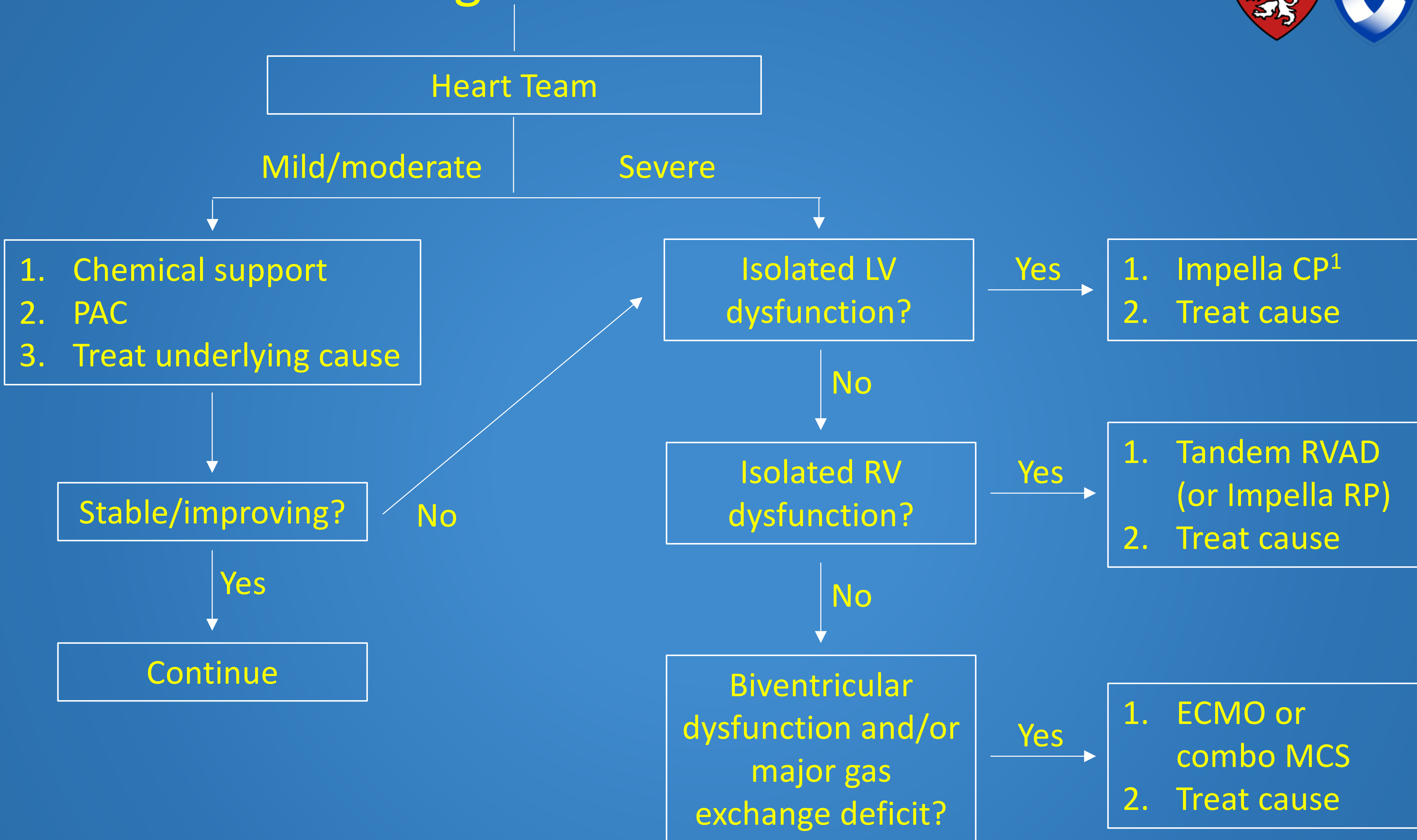
Downloaded from http://ahajournals.org by on October 20, 2020

- 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

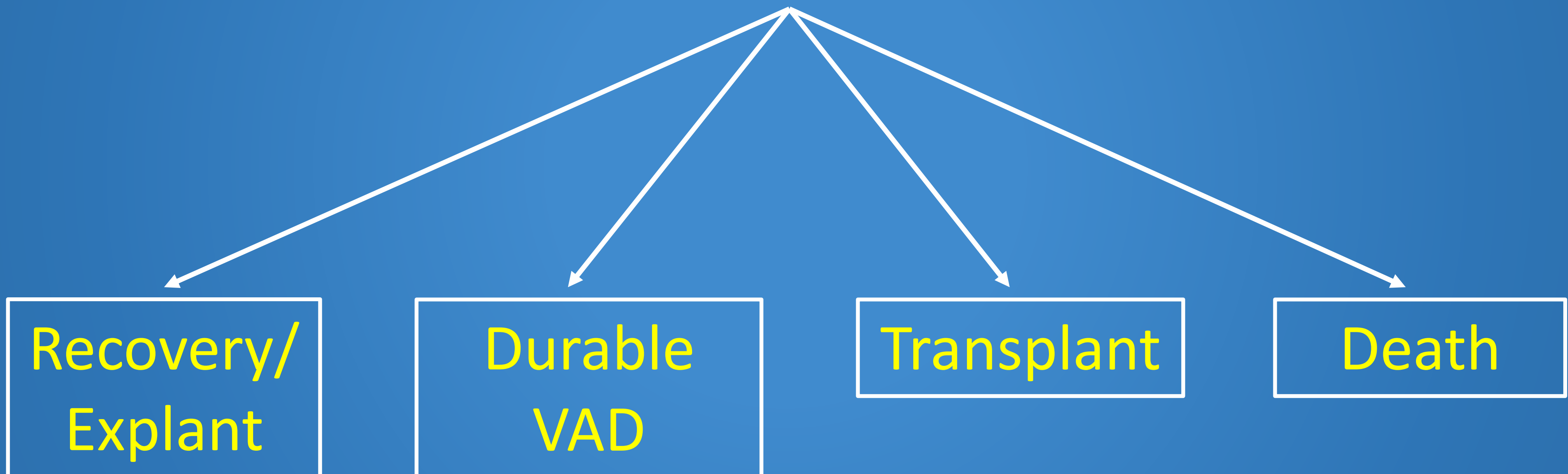


	a) Impella RP	b) TandemHeart RA-PA	c) VA-ECMO	d) IABP	e) Impella	f) TandemHeart	g) iVAC 2L
Flow:	max. 4.0 L	max. 4.0 L	max. 7.0 L		2.5-5.0 L	max. 4.0 L	max. 2.8 L
Pump speed:	33.000 rpm	max. 7.500 rpm	max. 5000 rpm		max. 51.000 rpm	max. 7.500 rpm	40 ml/beat
Cannula size:	22 F	29 F	14-19 F arterial 17-21F 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	+	+	++	-	-	-	-



[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
Recovery/ Explant
Durable VAD
Transplant
Death
BWH

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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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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TIMI