Cardiogenic Shock: Mechanical Support Devices

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Director Interventional Cardiology Fellowship Program
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Update in Cardiology at KHN

- Kettering Heart and Vascular Part of KPN
  - 48 Cardiologists from 6 groups integrated into one large unified group that will cover 8 hospitals
  - Subspecialized Pods
    - EP
    - CHF
    - Cardio Oncology
    - Interventional
      - CTO
      - Structural heart TAVR, Mitral Clip, Watchman
      - High Risk Intervention
      - Peripheral
    - Imaging

What's Ahead

- Single Pod at each of outlying hospitals
- More Specialty Specific Care
- Advanced Technologies
- Systematized Network wide approach.
- Integration with other specialties
  - CTS
  - Vascular Surg
  - Advanced HTN clinic with Nephro and Cardio together
  - Cardio-Onc
  - Pulm Htn with Cardio and Pulm.

Future Growth

- Support the Network approach
- Cardiology Satellite offices at Primary Care Hubs
- Consolidate some offices
- Building out Hybrid Cath lab
- Expand specialty offices into Cancer Annex

CASE 1

- 58 yo lady with a history of PAF, CVA, HTN, HFrEF with EF 25%, DM-2 presented with chest pain and SOB.

- Previous smoker, quit 30 yrs ago. No significant FHx of CAD

- Lotrel (amlodipine/benzepril), atenolol, atrovastatin, coreg, levemir, metformin, lyrica and effexor

- O/E she was AAOx3, BP 190/110 mmHg.

- EKG: sinus tachycardia at 101 bpm, normal axis, poor R-wave progression in the pericordial leads and non-specific ST-T wave changes. Initial torponins level was 2.4

EKG


Kettering Health Network - Kettering Medical Center (1-01-73)
Confirmed for: Bequeire, Reginald (MD) 11-Nov-2016 09:53
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Device: KEC503
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10 mm/mV

- Stabilized and treated medically for NSTEMI + Hypertensive emergency.

- Next morning>> Cath lab.

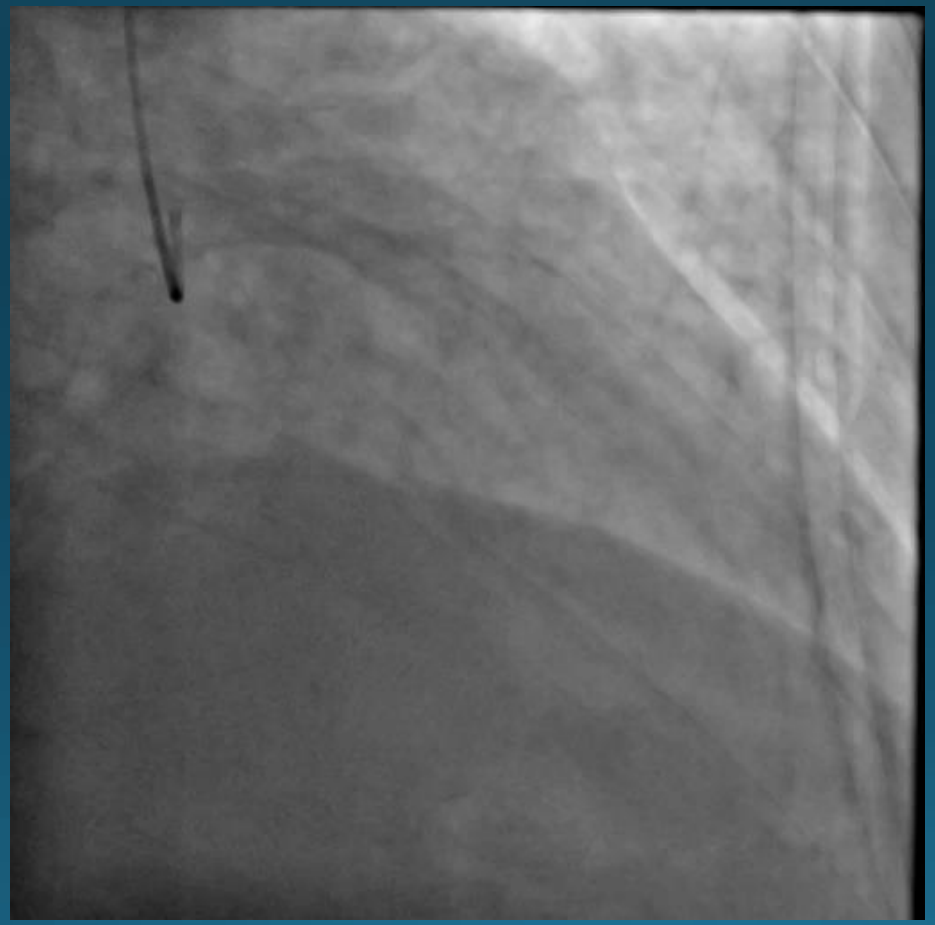
- Diagnostic LHC revealed: 95% ostial LAD, 90% prox, 75% mid and 95% distal. LCx: dominanant vessel with moderate disease distally. RCA: small non-dominant.

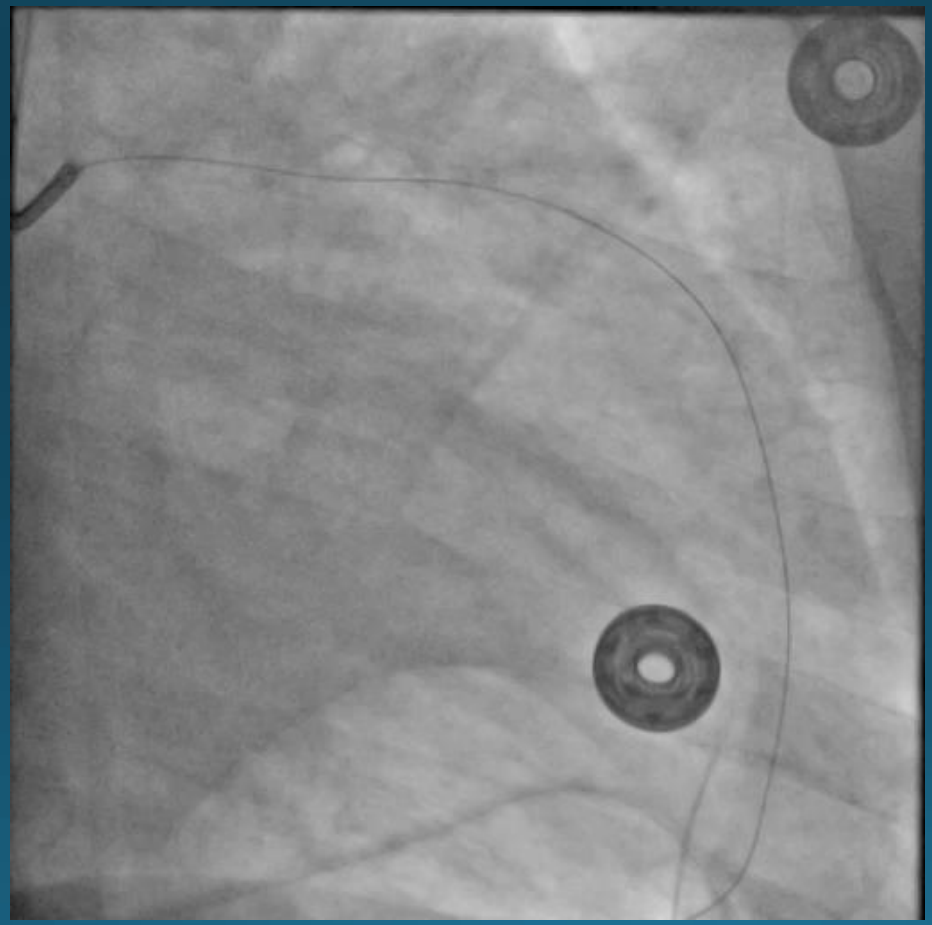
- Planned PCI to LAD as not best surgical candidate

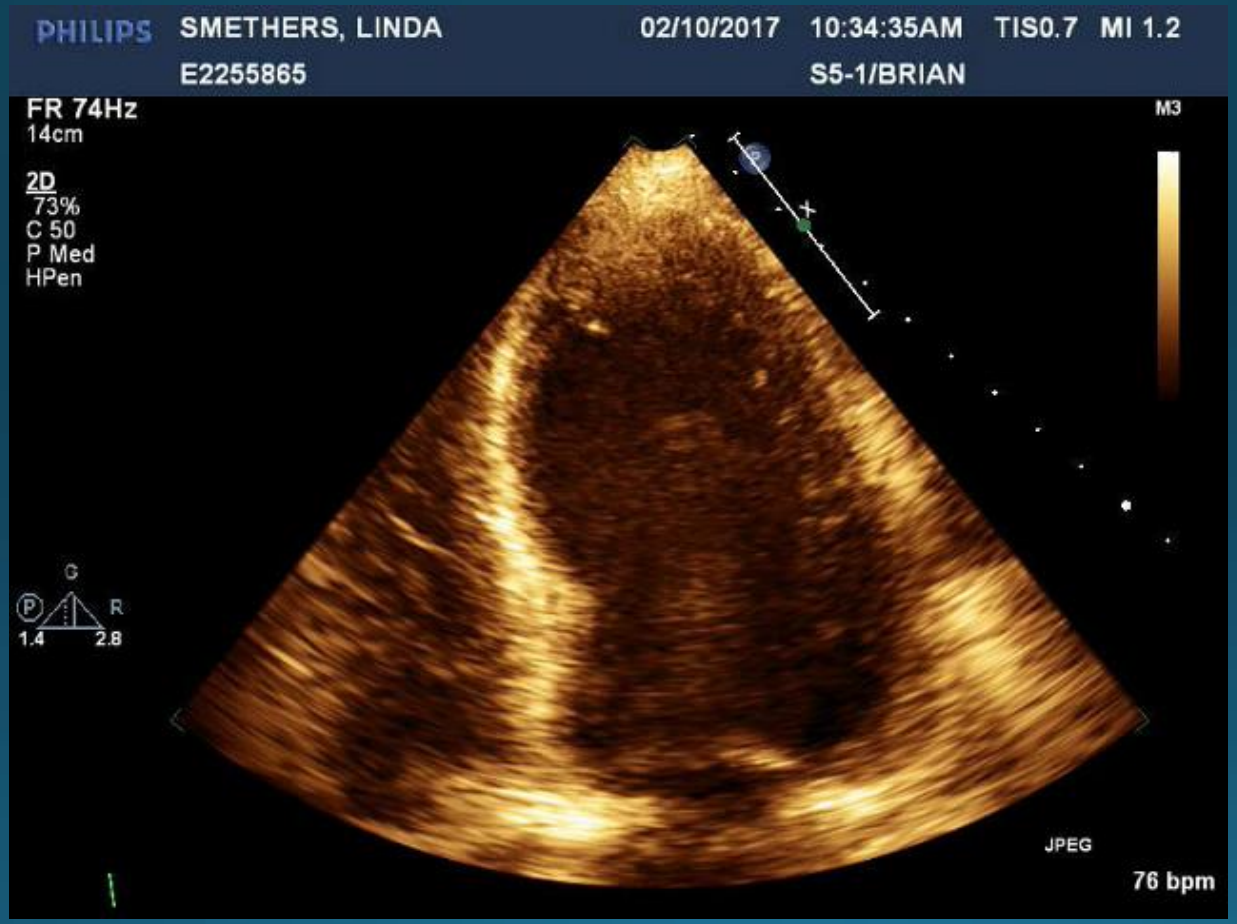
- Started with angioplasty and ballooning – Didn't tolerate Baloon.

- Subsequent Angiography chowed 99% occlusion of proximal LAD and 100% distal LAD.

- low BP + pulseless + CPR multiple rounds of Epi >>> impella CP



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SMETHERS, LINDA
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02/10/2017
10:34:35AM
TIS0.7
MI 1.2
S5-1/BRIAN
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14cm
2D
73%
C 50
P Med
HPen
M3
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R
1.4
2.8
JPEG
76 bpm


PHILIPS
SMETHERS, LINDA
E2255865
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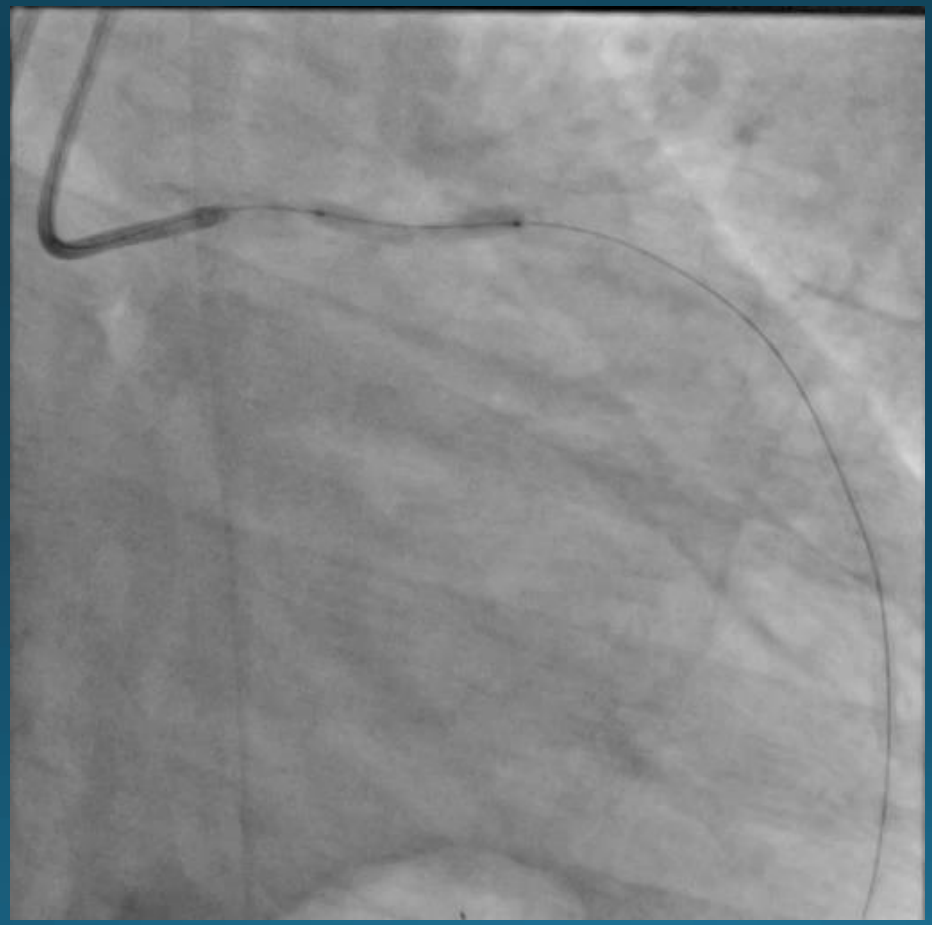
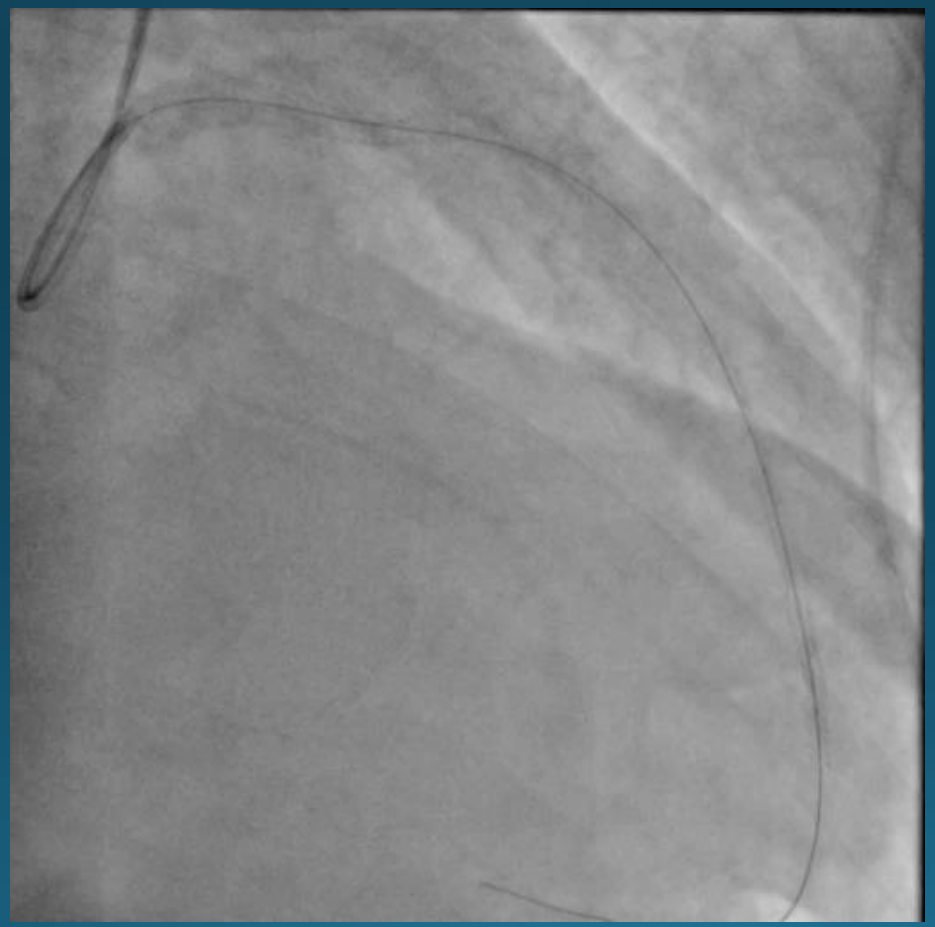
MI 1.4
S5-1/BRIAN
FR 39Hz
26cm
2D
76%
C 50
P Med
HPen
M3
G
P
R
1.4
2.8
JPEG
82 bpm

- On impella she had 1 episode of Vfib – defibrillated + intubated, pulse was regained in <2 mins
- Balloon inflations in LAD causes drop in BP with more rounds of Epinephrine and CPR
- Surgery Present but did not want to take to OR with multiple problems
- ECMO placed > more hemodynamically stable > PCI to LAD with 3 stents





ECMO

- Transferred to UC
- ICU with ECMO for 3 days
- Eventually DC
- EF Improved to 40%

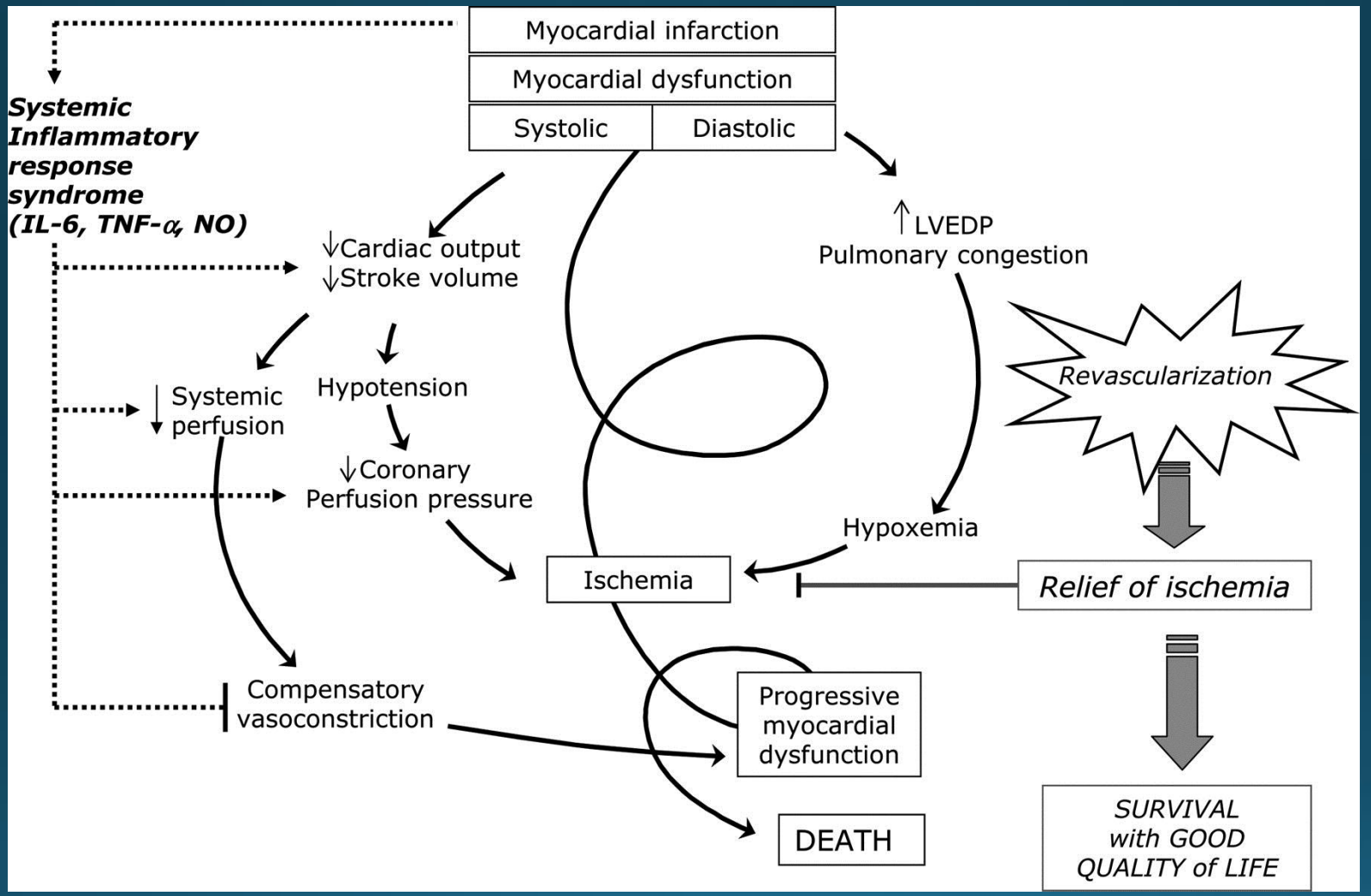
Cardiogenic Shock

- CS occurs in approx 5-8% of Patients hospitalized with ST elevation MI and 2.5% of NSTEMI. 50,000 cases per year in United States.
- MI with LV failure remains the most common cause of CS
- Must exclude complicating factors.
  - VSD, MV Chordal rupture, Free wall rupture, Massive PE, Hemorrhage, Infection or Bowel Ischemia, RV Dysfunction.
- Iatrogenic: BBs and ACE inhib, Diuretics, Volume overload.

Cardiogenic Shock

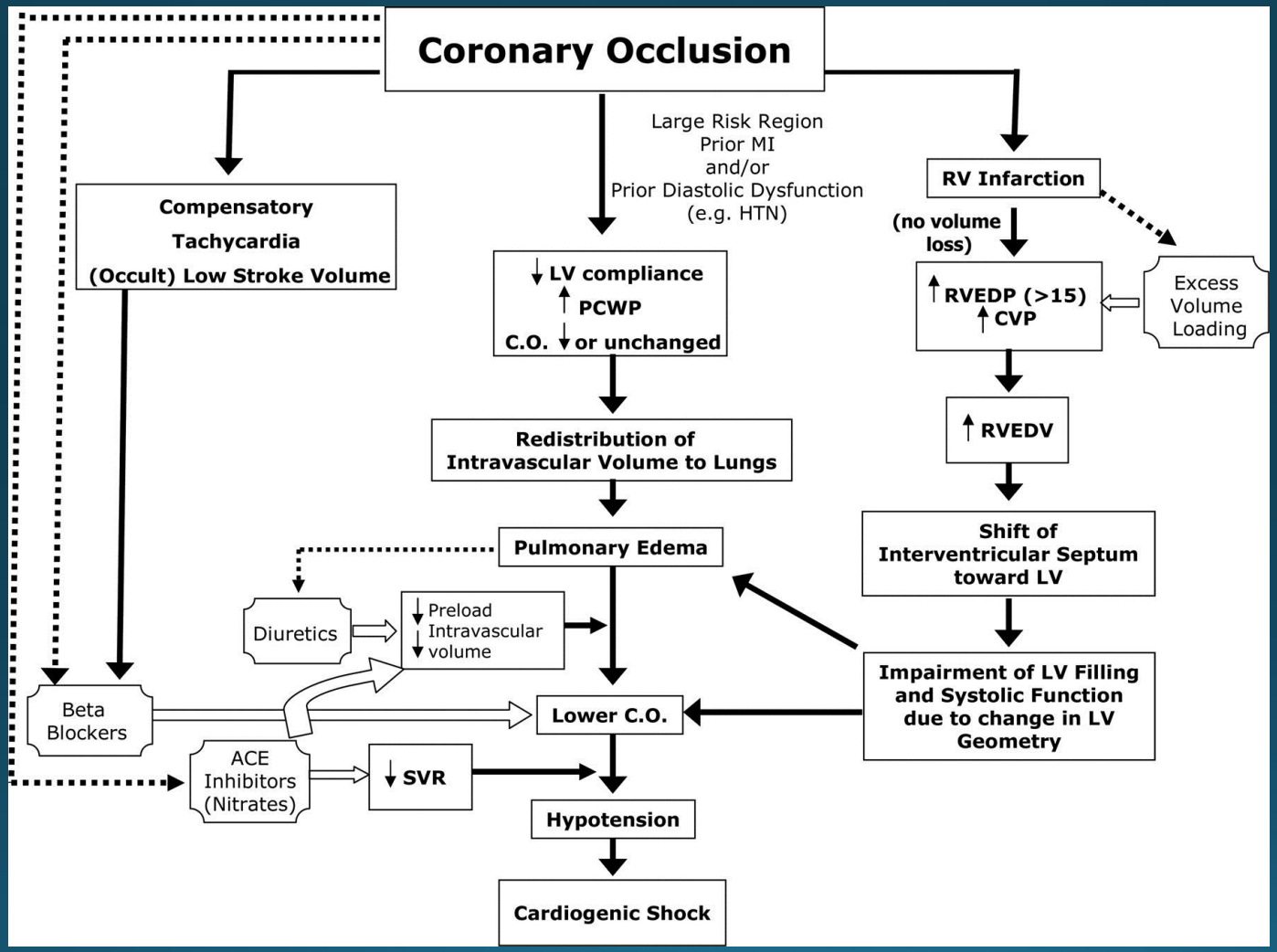
- CS is a state of end-organ hypoperfusion due to cardiac failure. The definition of CS includes hemodynamic parameters: persishypotension (Sys BP <90 mm Hg or MAP 30 mm Hg lower then baseline) with severe reduction of CI <1.8L min/M2.
  - Must have adequate or elevated filling pressure (LVEDP >18)
- Usually presents with cool extremities, decreased UO, and/or altered Mental status.
- Can be mild to profound
- Mortality directly related to severity and time until corrected.

Figure 1. Current concept of CS pathophysiology.



Harmony R. Reynolds, and Judith S. Hochman Circulation. 2008;117:686-697

Iatrogenic shock.

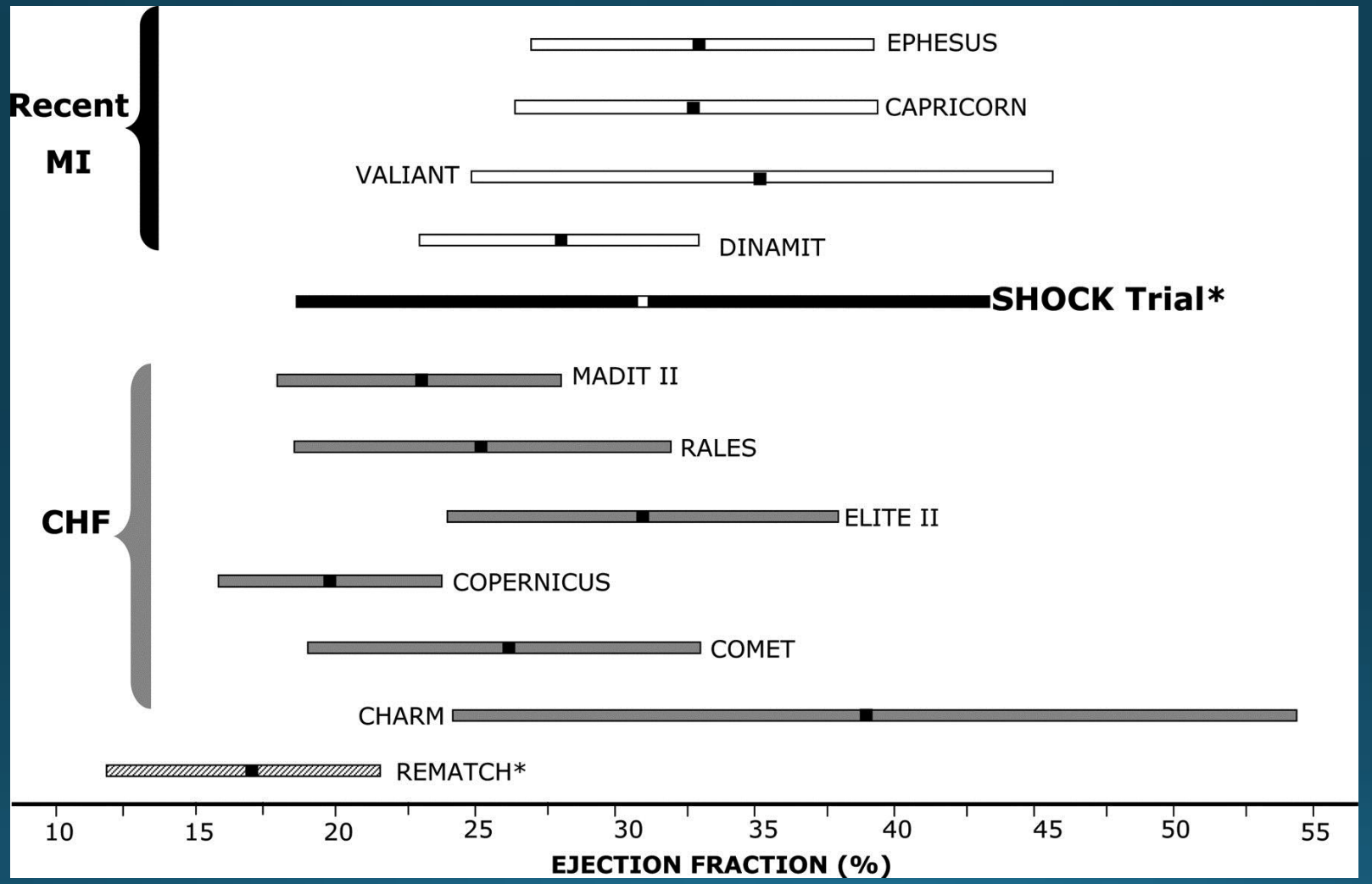


Harmony R. Reynolds, and Judith S. Hochman Circulation. 2008;117:686-697

Cardiogenic Shock Diagnosis

- Clinical grounds
- PA Catheter
- Doppler Echocardiography

Range of LVEF in studies of heart failure and in the SHOCK trial.



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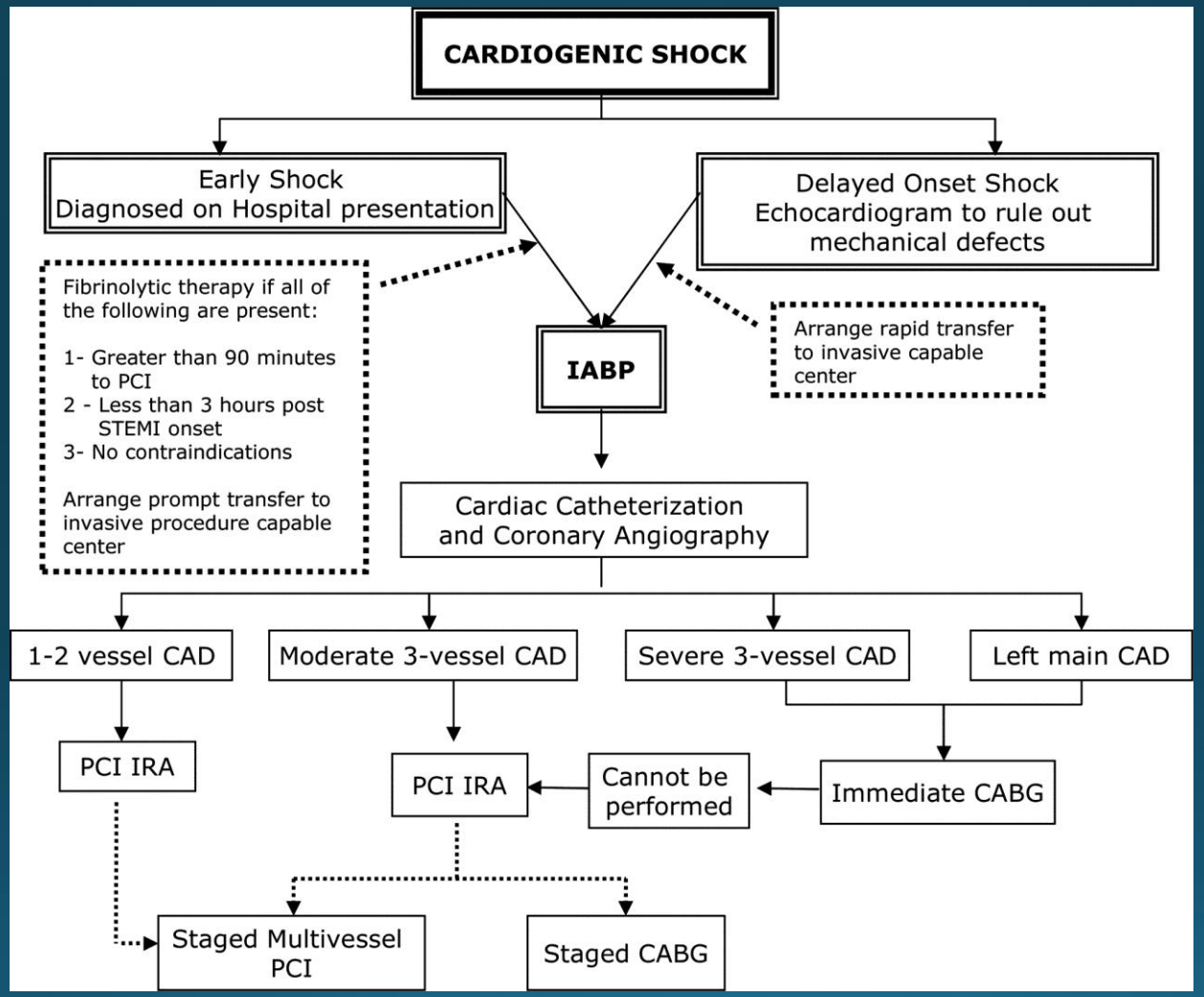
SIRS

- Recent research has suggested that the peripheral vasculature and neurohormonal and cytokine systems play a role in the pathogenesis and persistence of CS. Increased levels of IL6 TNF-a and rise of Cytokine levels. All impair endothelial function and suppress Myocardial function.
- Mi can cause SIRS and impaired perfusion of intestinal tract increases risk of transmigration of bacteria.
- Sooner tissue perfusion is restored the better prognosis.
- Mechanical support devices can help.
- Hospital Survivors have an excellent chance for long term survival and recovery. Including a good QOL.

Treatment

- Early recognition of cause
  - ECG/Echo/Cath/Enzymes and other tests if clinical suspicion.
- Early transfer to Advanced Treatment program
- Pressors and Ionotropes
- Adequate but not excessive volume resusatation.
- Rapid revascularization. PCI or CABG
- Mechanical Support.

Algorithm for revascularization strategy in cardiogenic shock, from ACC/AHA guidelines.42,44 Whether shock onset occurs early or late after MI, rapid IABP placement and angiography are recommended.



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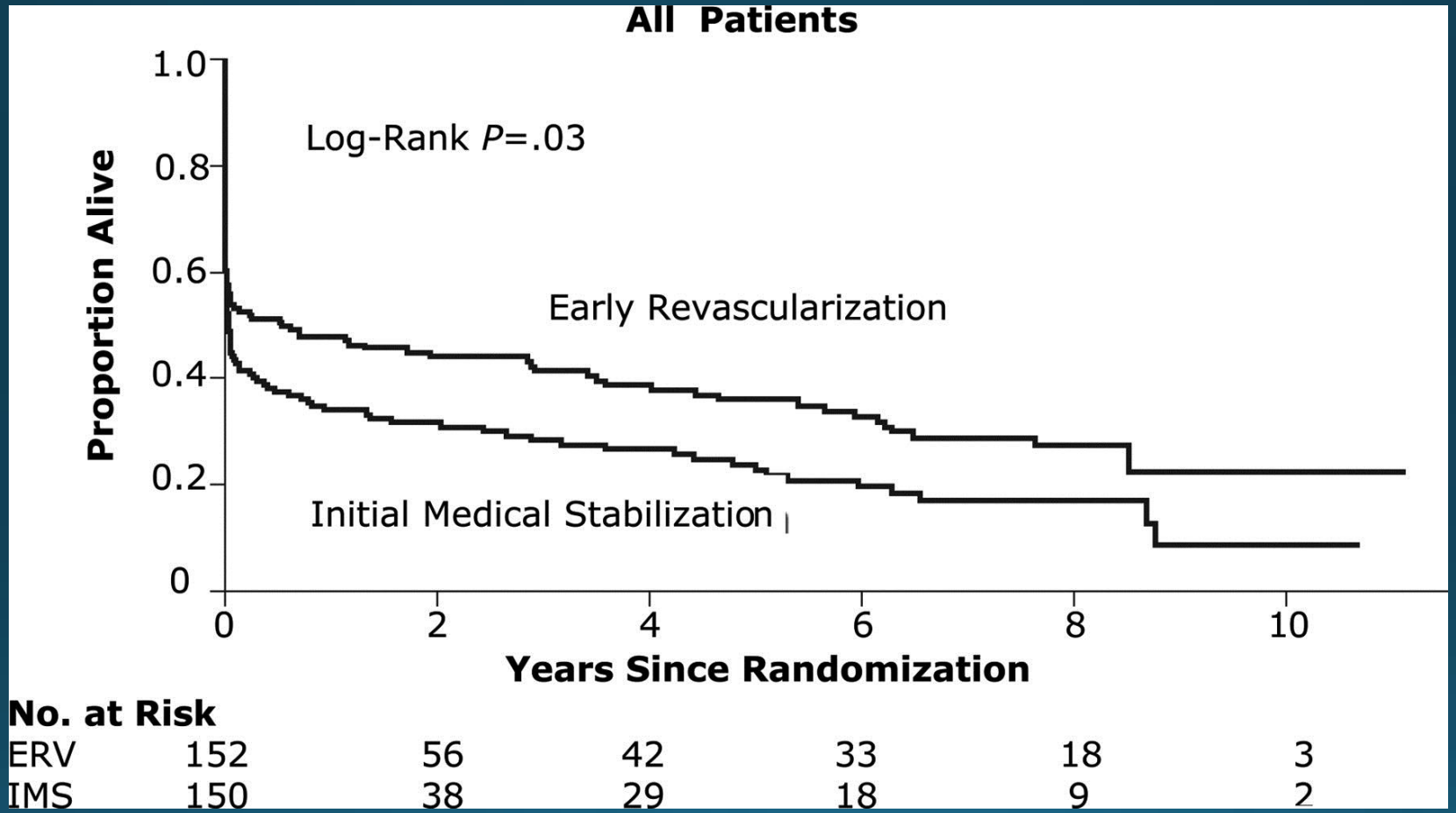
Long-term follow-up of the SHOCK trial cohort.55 Early revascularization (ERV) is associated with sustained benefit.



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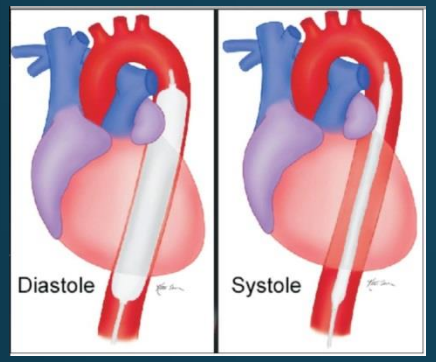
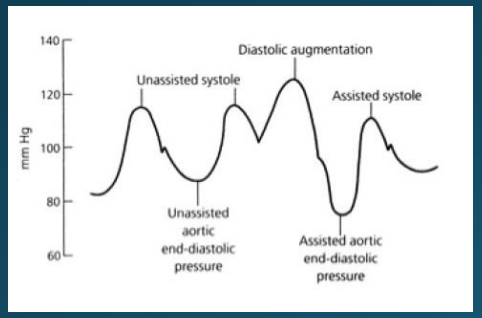
IABP





- The intra aortic balloon pump (IABP) acutely improves systemic hemodynamics, augments coronary flow, increases coronary patency, and reduces myocardial oxygen demand.
- Physiological and clinical data, led to a widespread use of the IABP in cardiogenic shock, refractory angina, primary PCI, high risk PCI, and complex CABG in the last decades.

Kantrowitz et al. JAMA. 1968;203:113–118
Williams et al. Circulation. 1982;66:593–597
Kern et al. Circulation. 1993;87:500–511
Ohman Circulation. 1994;90:792–799
Rihal et al. JACC. 2015; 65: e7-26

IABP

- >1 Million patients treated, low complication rate, Benchmark registry[1]
- IABP therapy is the most widely used means of circulatory support for patients with hemodynamic instability resulting from LV Failure[2]
- Well known effects of IABP include decrease in afterload that leads to an increase in stroke volume and cardiac output[2]

[1] Ferguson et al. JACC 2001;38:1456-1462
[2] Prognostic impact of IABP before vs. after cardiac surgical intervention. A S Dhaliwal et al. AmJSurg 2009; 198: 628-632

IABP PROs

- Small ateriotomy (7.5-8.0 F)
- Can be used in combination with other devices
- Short term outcome similar to other technologies
- Easy antithrombotic management

IABP CONs

- Minimum of cardiac function and a competent aortic valve required
- Modest ventricular unloading
- No outcome improvement in some studies
- Practice guidelines downgraded recommendations or even discouraged the use of IABP

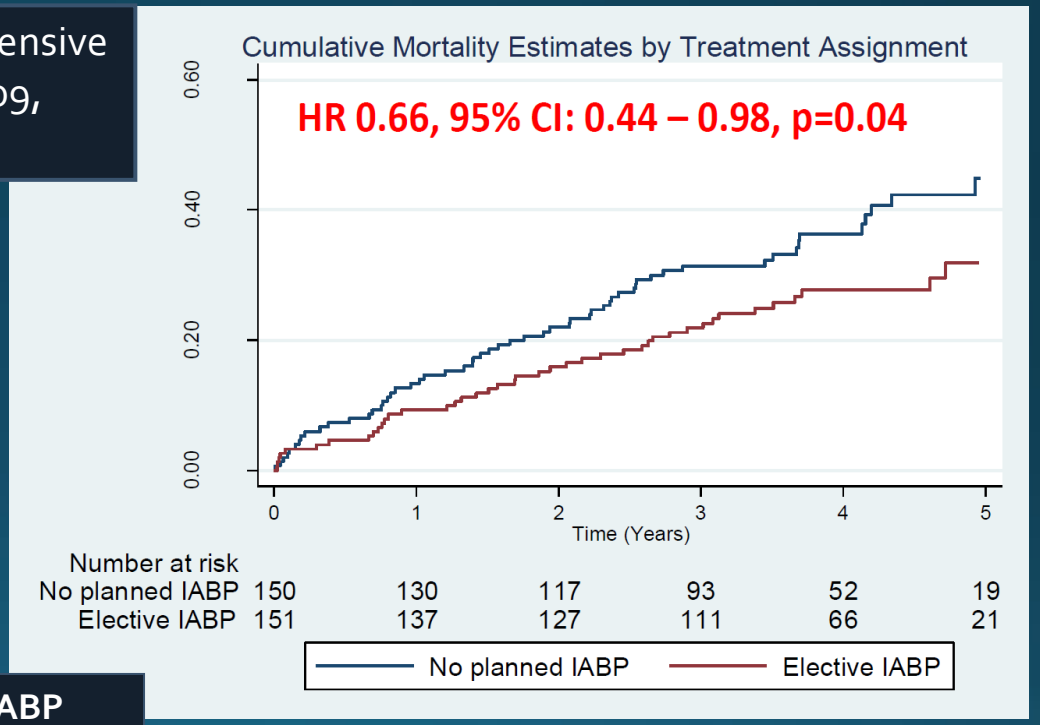
BISIS-1: Elective IABP improves long-term survival after high-risk PCI

301 patients, high-risk PCI (EF<30% and extensive myocardium at risk) 12/2005 and 01/2009, 17 clinical sites UK

50% no planned IABP before PCI (N=151)

12% required bailout IABP (N=18)

50% planned IABP before PCI (N=150)



Perera et al. Circulation 2013; 127: 207-212

IABP-SHOCK II Trial

600 patients with cardiogenic shock (clinical assessment) were enrolled between June 2009 and March 2012 in 37 clinical sites in Germany.

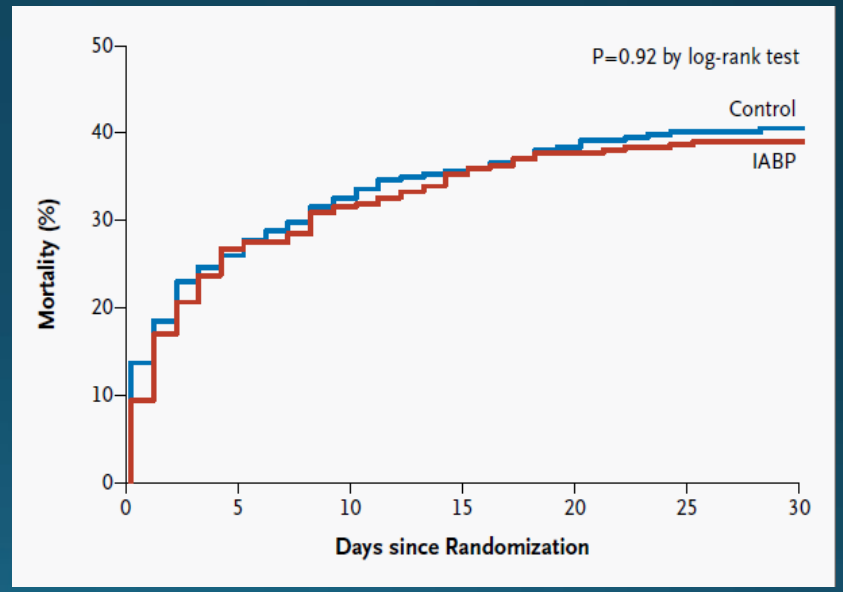
301 randomized to IABP
- 13 did not receive IABP
- 287 primary PCI
- 11 no revascularisation

299 randomized to control
- 30 cross-over to IABP
- 288 primary PCI
- 8 no revascularisation

300 with 30 d follow-up

298 with 30 d follow-up

Primary endpoint: 30 d all-cause mortality



Thiele et al. N Engl J Med 2012;367:1287-96.

IABP-SHOCK II Trial

Strength

- Largest randomized shock trial
- 600 patients included within 32 month
- Contemporary CS treatment (>95 % revasc.)
- Follow-up: 99.2%

Limitations

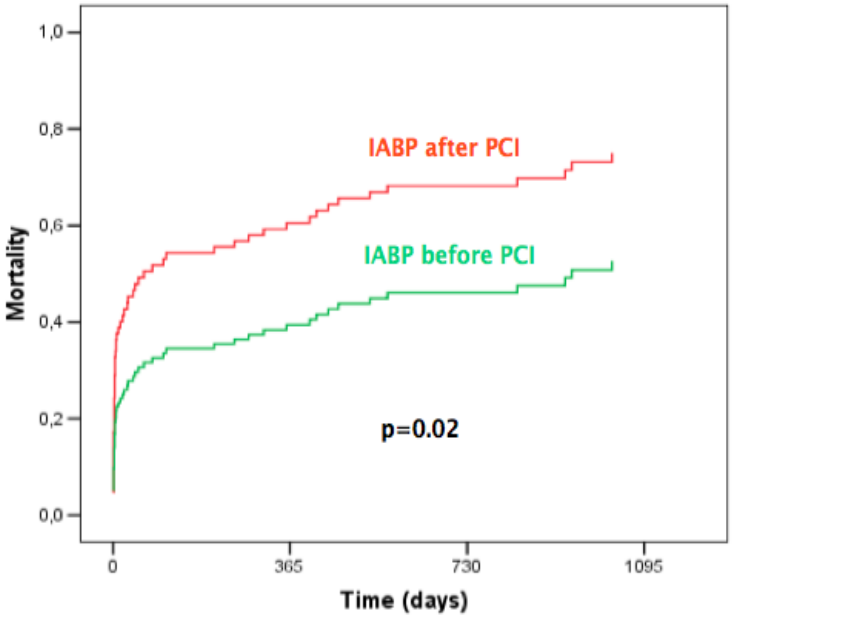
- Underpowered for the primary endpoint
- No hemodynamic shock assessment
- 10% cross-over to IABP
- 83% of pts. received IABP post PCI

Impact of IABP-Timing in CS

Design

- DESIGN: Single center observational study in 102 patients (Jan. 2005-Dez. 2010).
- OBJECTIVE: To evaluate the impact of IABP timing (before or after PCI) in STEMI complicated by cardiogenic shock.
- ENDPOINTS: Total mortality, MACCE, renal failure

Total mortality



*Adjusted for age, smoking, AF, MV disease, prev. CABG, CPR before PCI, vasopressors before PCI, pre-existing renal failure

Schwarz et al. Med Klin Intensivmed Notfmed (2015)

Impact of IABP-Timing in CS

Clinical outcomes at 30 days

	IABP before PCI (n=49)	IABP after PCI (n=53)	p-value
Mortality	12 (25%)	29 (55%)	0.002
Emergency CABG	0 (0%)	5 (9%)	0.027
Cerebrovascular Events	4 (8%)	4 (8%)	0.908
MACCE	15 (31%)	32 (60%)	< 0.001
Bleeding	12 (25%)	14 (26%)	0.824
Acute renal failure	9 (18%)	14 (26%)	0.331

Schwarz et al. Med Klin Intensivmed Notfmed (2015)

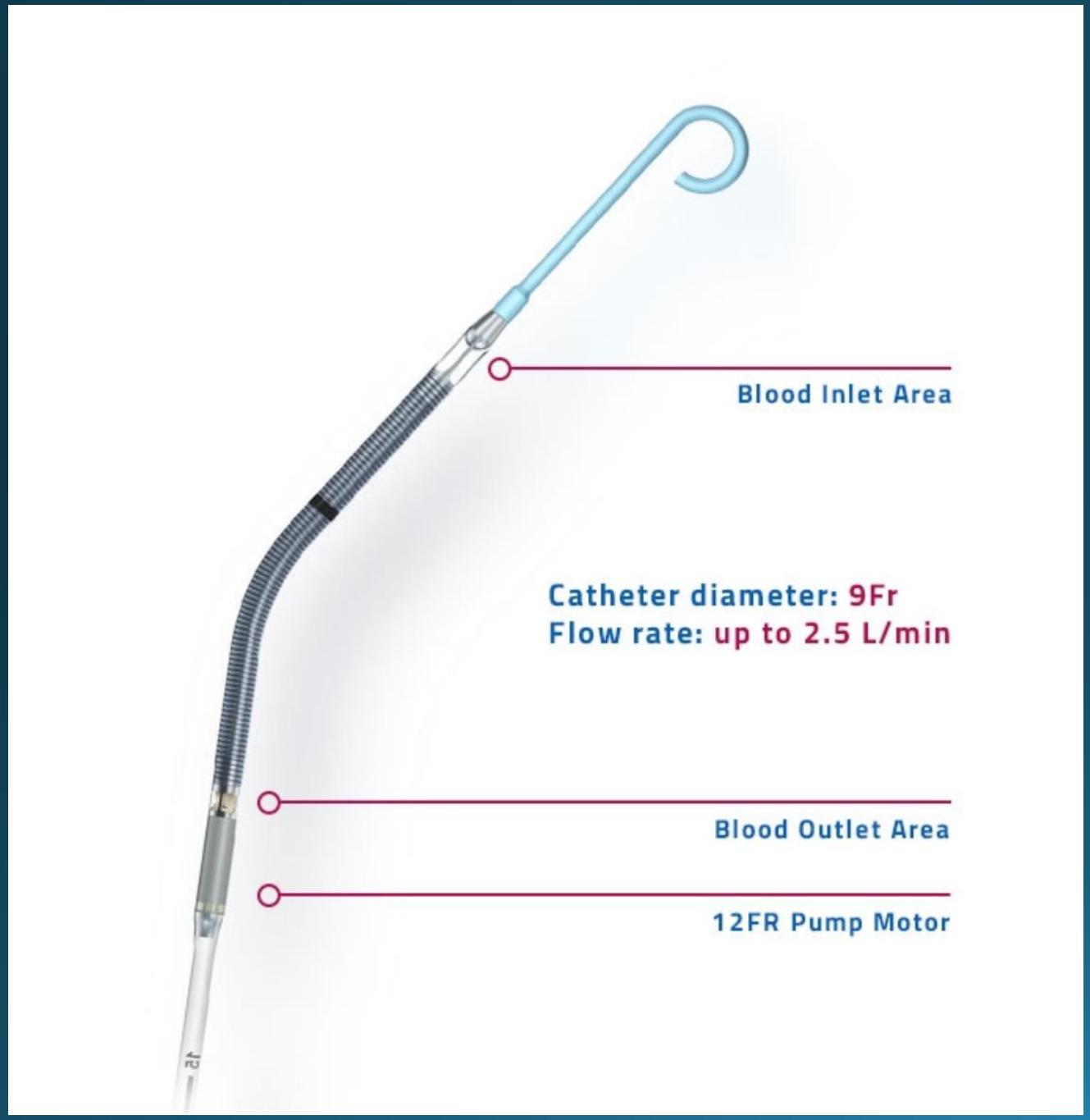
Conclusions

- IABP can improve short term outcome in cardiogenic shock patients, as long as it is preceding PCI.

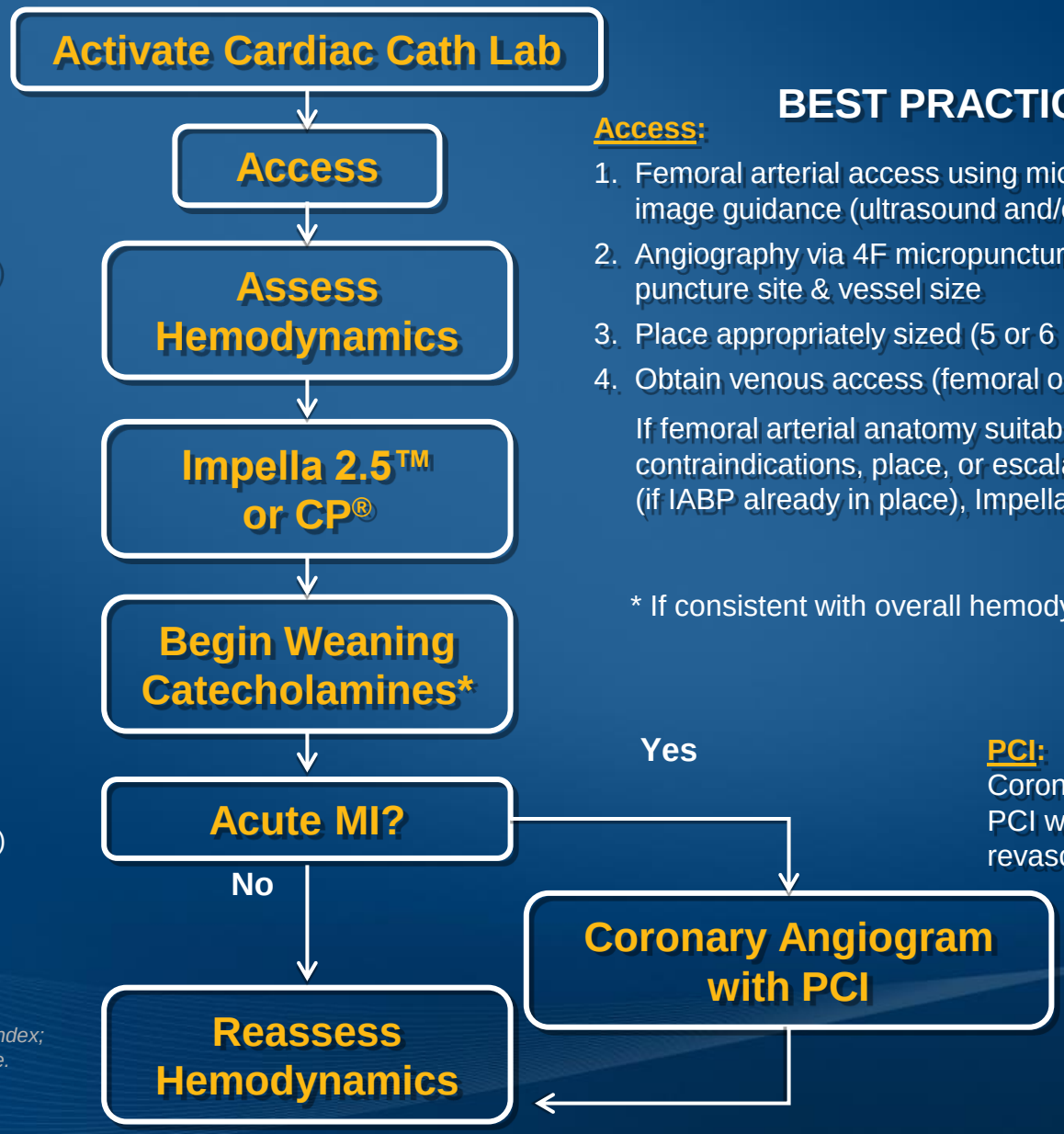
- IABP improves long term survival in high-risk PCI - possibly related to more stable procedural hemodynamics with more complete revascularisation.

Percutaneous LVAds

- Impella


Blood Inlet Area
Catheter diameter: 9Fr
Flow rate: up to 2.5 L/min
Blood Outlet Area
12FR Pump Motor

Stabilize Early and Complete Revascularization



BEST PRACTICE

Assess Hemodynamics: LVEDP or PAC

- If sustained hypotension (SBP < 90 mmHg) for > 30 min

  Or

- CI < 2.2 with LVEDP or PCWP >18 mmHg, consider mechanical circulatory support

Reassess Hemodynamics: PAC (if not done initially)

1. CPO = MAP × CO/451 W
2. PAPi = sPAP-dPAP/RA

CO, cardiac output; CPO, cardiac power output; dPAP, diastolic pulmonary arterial pressure; MAP, mean arterial pressure; PAC, pulmonary arterial catheter; PAPi, pulmonary artery pulsatility index; RA, right arterial pressure; sPAP, systolic pulmonary arterial pressure.

BEST PRACTICE

Access:

1. Femoral arterial access using micropuncture with image guidance (ultrasound and/or fluoroscopy)[1]
2. Angiography via 4F micropuncture dilator to confirm puncture site & vessel size
3. Place appropriately sized (5 or 6 Fr) arterial sheath
4. Obtain venous access (femoral or internal jugular)

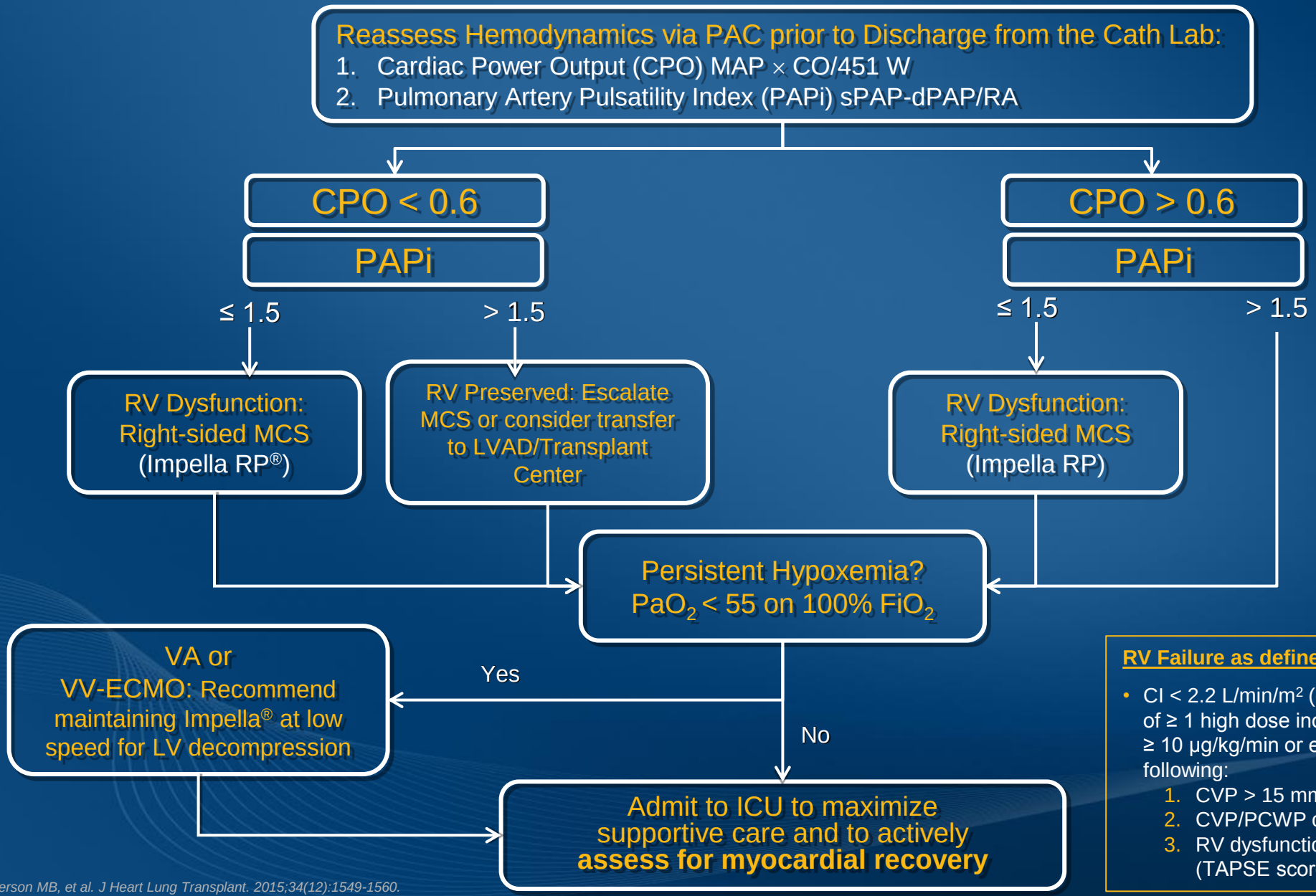
If femoral arterial anatomy suitable and no contraindications, place, or escalate to (if IABP already in place), Impella 2.5 or Impella CP

* If consistent with overall hemodynamic management

PCI:
Coronary angiography and PCI with goal of complete revascularization

Soverow J, Lee MS. J Invasive Cardiol. 2014;26(12):659-667.

Reassess Prior to Discharge From Cath Lab



RV Failure as defined by Recover Right[1]:

- CI < 2.2 L/min/m^2 (despite continuous infusion of ≥ 1 high dose inotrope, ie, da/dobutamine ≥ 10 µg/kg/min or equivalent) and any of the following:
  1. CVP > 15 mmHg, *or*
  2. CVP/PCWP or LAP ratio >0.63, *or*
  3. RV dysfunction on TTE (TAPSE score ≤14 mm)

Anderson MB, et al. J Heart Lung Transplant. 2015;34(12):1549-1560.

Escalation, Weaning, and Transfer

Assess for Myocardial Recovery
(At least every 12 hours)

Improving
Clinical, Echocardiographic & Hemodynamic parameters (concordant):
- ↑ Cardiac output
- ↑ CPO
- ↑ Urine output
- ↓ Lactate
- Inotropes low dose/discontinued
- Adequate Ramp test

→ **Myocardial Recovery**

→ Wean & Explant Impella®
(After a minimum of 48h)

Mixed picture
Clinical, Echocardiographic & Hemodynamic parameters (discordant):
- Some parameters are improving
- Pressors lowered but not discontinued
- Fails "ramp test"

→ **Inadequate Recovery**

→ Continue Impella® support & frequent clinical reassessment

Failure to recover within 48-72 h, consider escalation or durable VAD/transplant

Worsening
Clinical, Echocardiographic & Hemodynamic parameters (concordant):
- ↓ Cardiac output
- ↓ CPO
- ↓ Urine output
- ↑ Lactate
- Inotrope dependent
- Absent pulsatility

→ **No Recovery**
Escalate (& Ambulate)
or Transfer

→ See Escalate or Transfer Protocol

No Recovery: Escalate or Transfer

No Recovery
Escalate (& Ambulate) or Transfer
Guidance by RHC Is Critical

Worsening / not improving clinical, echocardiographic & hemodynamic parameters (concordant):

- ↓ Cardiac output
- ↓ CPO
- ↓ Urine output
- ↑ Lactate
- Inotrope dependent
- Absent pulsatility

RV Failure as defined by Recover Right[1]:

- CI < 2.2 L/min/m^2 (despite continuous infusion of ≥ 1 high dose inotrope, ie, da/dobutamine ≥ 10 µg/kg/min or equivalent) and any of the following:
  1. CVP > 15 mmHg, *or*
  2. CVP/PCWP or LAP ratio >0.63, *or*
  3. RV dysfunction on TTE (TAPSE score ≤14 mm)

CPO remains < 0.6 After 24 hours

PAPi ≤ 0.9 (RV Failure)	0.9 < PAPi ≤ 1.5 (RV dysfunction)	PAPi > 1.5 (acceptable RV function)
Biventricular support with Impella RP® on the right side (transfer if not available)	Consider supporting the right side with Impella RP (transfer if not available)	Consider left-side escalation with Impella 5.0™ (transfer if not available)

CPO = (MAP × CO)/451.
PAPi = sPAP - dPAP/RA.

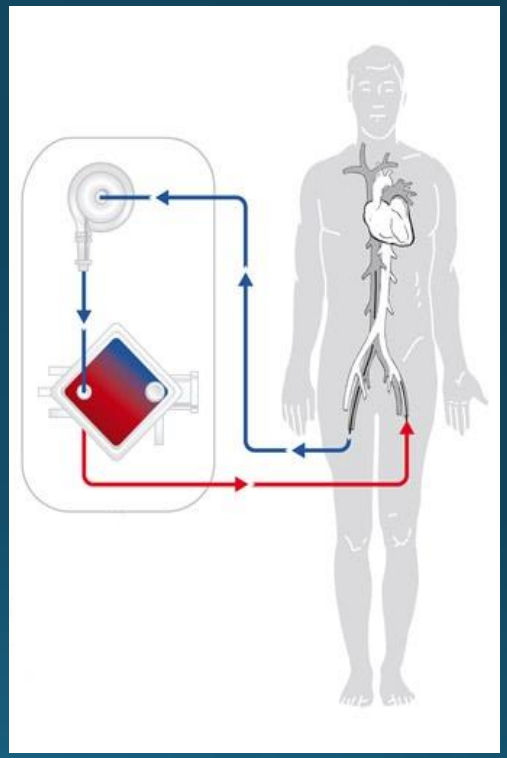
Anderson MB, et al. J Heart Lung Transplant. 2015;34(12):1549-1560.

ECMO

MCV00019555 REVB

CENTRIFUGAL PUMP AND OXYGENATOR

- Guidelines specific for H1N1 pandemic
  - Use extracorporeal circuit for total support including diffusion membrane oxygenator and centrifugal pump
  - Heat exchanger to control blood and patient temperature at a specific level
  - Utilize patient monitoring for continuous inlet/outlet pressures, blood gas, saturation and S_VO_2

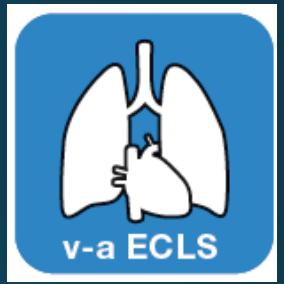


ECLS SUPPORT

Partial Support

Full Support

WHEN FAILURE IS NOT AN OPTION… THE GOAL OF HEMODYNAMIC STABILITY



- Provide adequate flow to every patient (>2.5 liters)
- Decrease work of the heart and stabilize MVO_2
- Unload and or decompress the entire heart
- Support end organ perfusion
- Provide adequate oxygen supply to entire body
- Optimize hemodynamic stability
- Temperature regulation

ECLS FOR ASSISTED PCI IN PROFOUND CARDIOGENIC SHOCK[1]

- Study comparing shock with profound shock
  - Control Group: 120/920 (13.0%) STEMI
  - Pulmonary Edema, SBP<90mmHg
- Persistent hypotension with cardiac output
  - No response to fluid, requiring vasopressors
- Profound Shock
  - SBP<75mmHg on inotropes and IABP
  - Altered mental status and/or respiratory failure

Clinical Investigations

Early extracorporeal membrane oxygenator-assisted primary percutaneous coronary intervention improved 30-day clinical outcomes in patients with ST-segment elevation myocardial infarction complicated with profound cardiogenic shock

Jiunn-Jye Sheu, MD; Tzu-Hsien Tsai, MD; Fan-Yen Lee, MD; Hsiu-Yu Fang, MD; Cheuk-Kwan Sun, MD, PhD; Steve Leu, PhD; Cheng-Hsu Yang, MD; Shyh-Ming Chen, MD; Chi-Ling Hang, MD; Yuan-Kai Hsieh, MD; Chien-Jen Chen, MD; Chiung-Jen Wu, MD; Hon-Kan Yip, MD

Objectives: This study tested the hypothesis that early extracorporeal membrane oxygenator offered additional benefits in improving 30-day outcomes in patients with acute ST-segment elevation myocardial infarction complicated with profound cardiogenic shock undergoing primary percutaneous coronary intervention.

Methods: Between May 1993 and July 2002, 920 patients with acute ST-segment elevation myocardial infarction underwent primary percutaneous coronary intervention. Of these patients, 12.5% (115) with cardiogenic shock were enrolled in this study (group 1). Between August 2002 and December 2009, 1650 patients with acute ST-segment elevation myocardial infarction underwent primary percutaneous coronary intervention. Of these patients, 13.3% (219) complicated with cardiogenic shock were enrolled (group 2).

Results: The incidence of profound shock (defined as systolic blood pressure remaining ≤75 mm Hg after intra-aortic balloon pump and inotropic agent supports) was similar in both groups (21.7% vs. 21.0%, $p > .5$). Extracorporeal membrane oxygenator support, which was available only for patients in group 2, was performed in the catheterization room. The results demonstrated that final thrombolysis in myocardial infarction grade 3 flow in infarct-related artery was similar between the two groups ($p = .678$). However, total 30-day mortality and the mortality of patients with profound shock were lower in group 2 than in group 1 (all $p < .04$). Additionally, the hospital survival time was remarkably longer in patients in group 2 than in patients in group 1 ($p = .0005$). Furthermore, multivariate analysis demonstrated that unsuccessful reperfusion, presence of advanced congestive heart failure, profound shock, and age were independent predictors of 30-day mortality (all $p < .02$).

Conclusion: Early extracorporeal membrane oxygenator-assisted primary percutaneous coronary intervention improved 30-day outcomes in patients with ST-segment elevation myocardial infarction with complicated with profound cardiogenic shock. (Crit Care Med 2010; 38:1810–1817)

Key Words: acute myocardial infarction; profound cardiogenic shock; extracorporeal membrane oxygenator

Cardiogenic shock is the leading cause of death in patients hospitalized for acute myocardial infarction (AMI) (1–3). Conservative therapy has been reported to yield disappointing results with a hospital mortality rate exceeding 80% in some clinical observational studies (4–6). Although reperfusion therapy using thrombolysis is one of the gold standards in the treatment of AMI (7, 8), no definite benefit of this therapeutic option has been reported in patients with AMI complicated with cardiogenic shock (9).

Accumulating evidence has demonstrated that the poor prognostic outcomes (1–3) of AMI complicated with cardiogenic shock can be improved by primary percutaneous coronary angioplasty (PCI) (1, 2, 9–11). Besides, stent-supported primary PCI has been shown to be superior to primary PCI in terms of immediate angiographic results and reduction of recurrent ischemia or reinfarction (6). However, even primary PCI was performed along with intra-aortic balloon pump (IABP) support for the patients with cardiogenic shock, both short-term (2) and long-term (11) mortality of these patients remains >45% (2, 11). Therefore, the treatment of the patients with AMI complicated with cardiogenic shock remains an unsolved problem that has vexed cardiologists for more than several decades. Interventional cardiologists are still searching for a satisfactory strategy in the management of this problem (11–13).

Our previous study (2) demonstrated that patients with AMI complicated with cardiogenic shock can be clearly categorized into profound shock and nonprofound shock subgroups, with the former having a substantially higher 30-day mortality rate as compared with the latter when undergoing primary PCI (71.4% vs. 22.1%, $p = .001$). An effective means of

From the Division of Cardiovascular Surgery (J-JS, F-YL), the Division of General Surgery (KS), Department of Surgery, the Division of Cardiology (T-HT, H-YF, SL, C-HY, S-MC, C-LH, Y-KH, C-JC), and the Department of Internal Medicine, Chang Gung Memorial Hospital-Kaohsiung Medical Center, Chang Gung University College of Medicine, Kaohsiung, Taiwan, Republic of China.

F-YL contributed equally to this study compared with the first author; C-JW contributed equally to this study compared with the corresponding author.

The authors have not disclosed any potential conflicts of interest.

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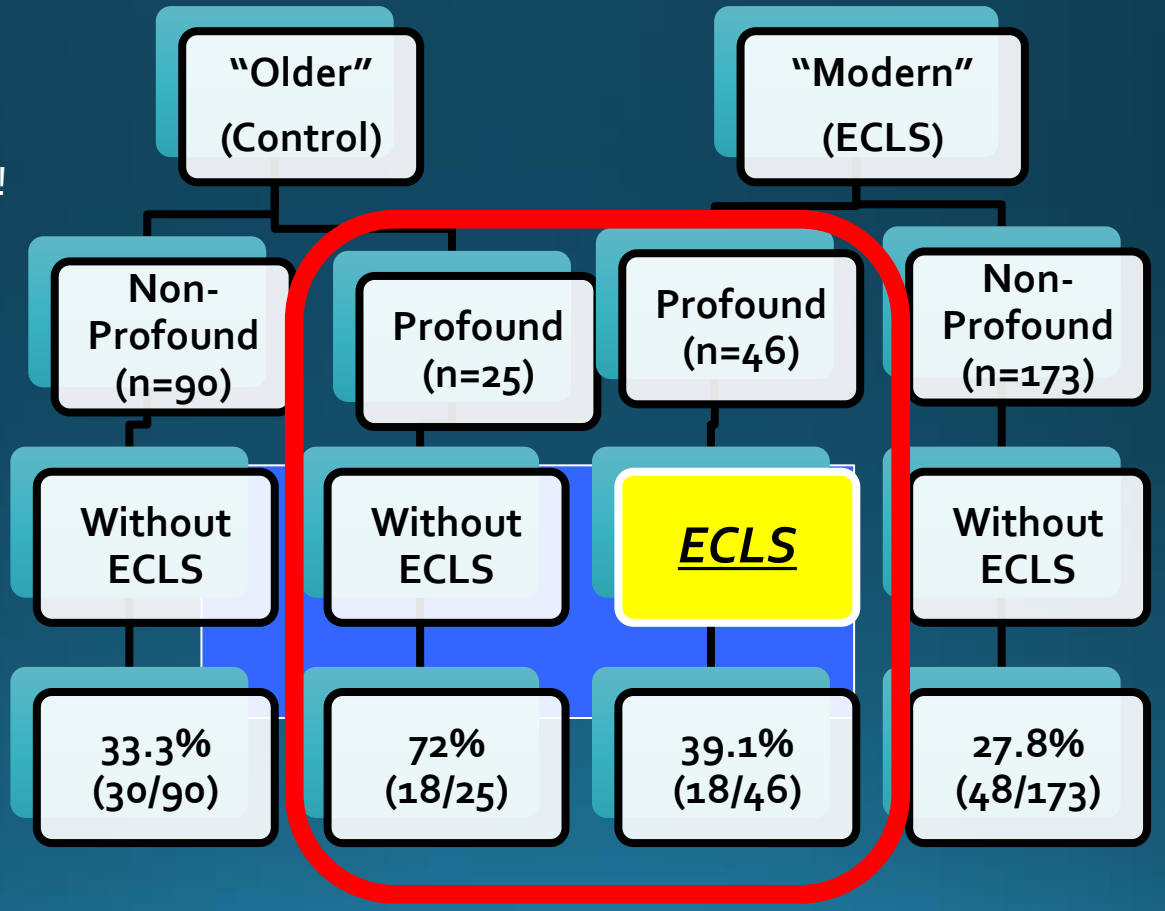
DOI: 10.1097/CCM.0b013e3181e8acf7

[1] Sheu et al. Crit Care Med 2010; 38;1810-1817

ECLS FOR ASSISTED PCI IN PROFOUND CARDIOGENIC SHOCK[1]

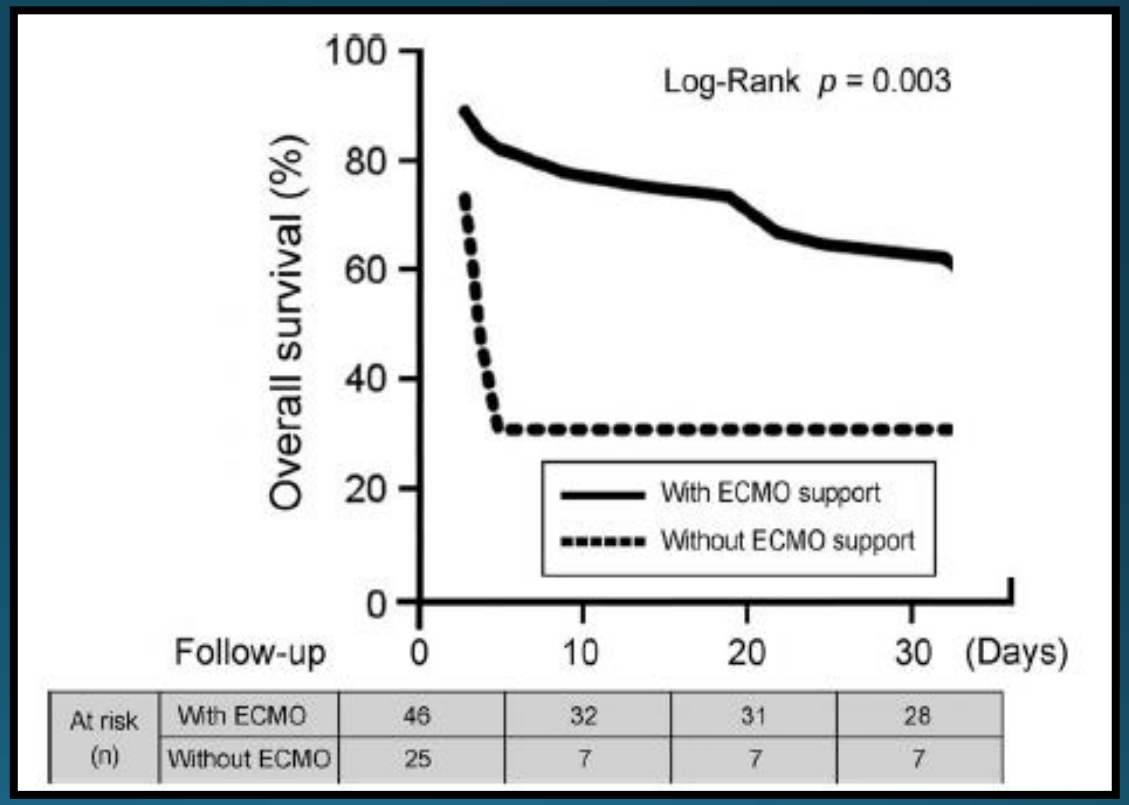
- 30 day Mortality
- 2x increase in survival rate!



[1] Sheu et al. Crit Care Med 2010; 38;1810-1817

ECLS FOR ASSISTED PCI IN PROFOUND CARDIOGENIC SHOCK[1]

- 30-day mortality was notably reduced in patients with ECMO support from what was seen for those without ECC



At risk (n)	With ECMO	46	32	31	28
	Without ECMO	25	7	7	7

[1] Sheu et al. Crit Care Med 2010; 38;1810-1817

ECLS FOR ASSISTED PCI IN PROFOUND CARDIOGENIC SHOCK[1]

- Implications:
  - ECLS offered "great benefit" in reducing 30 day mortality in patients with profound shock
  - Patients without ECLS tended to die early post-AMI
  - Can serve as a bridge to additional therapy as advanced heart failure was the strongest predictor of death in all groups

[1] Sheu et al. Crit Care Med 2010; 38;1810-1817

ECLS FOR ASSISTED PCI IN PROFOUND CARDIOGENIC SHOCK[1]

- "ECLS support played the key role in maintaining hemodynamic stability, which, in turn, allowed primary PCI to be continued until final procedure success"

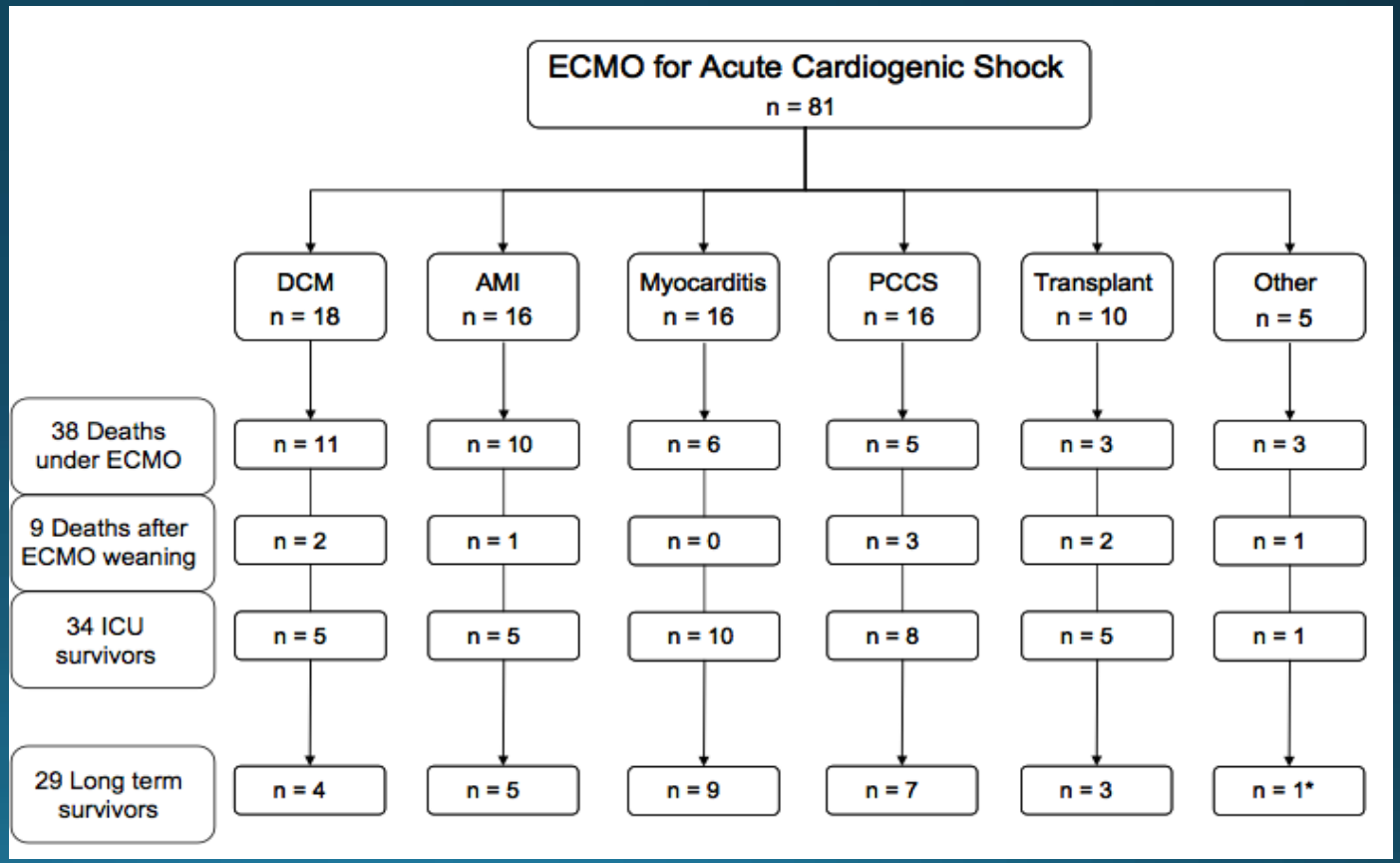
[1] Sheu et al. Crit Care Med 2010; 38;1810-1817

OUTCOMES AND LONG-TERM QUALITY-OF-LIFE OF PATIENTS SUPPORTED BY EXTRACORPOREAL MEMBRANE OXYGENATION FOR REFRACTORY CARDIOGENIC SHOCK[1]

- ECMO support can rescue 40% of otherwise fatal cardiogenic shock patients

42%

36%



[1] Combes et al. Crit Care Med. 2008 May;36(5):1404-11. doi:10.1097/CCM.0b013e31816f7cf7

CARDIOPULMONARY RESUSCITATION WITH ASSISTED EXTRACORPOREAL LIFE-SUPPORT VERSUS CONVENTIONAL CARDIOPULMONARY RESUSCITATION IN ADULTS WITH IN-HOSPITAL CARDIAC ARREST: AN OBSERVATIONAL STUDY AND PROPENSITY ANALYSIS[1]

- In hospital arrest
- 975 patients with IHCA
- 113 received CPR
- 59 had ECLS+CPR

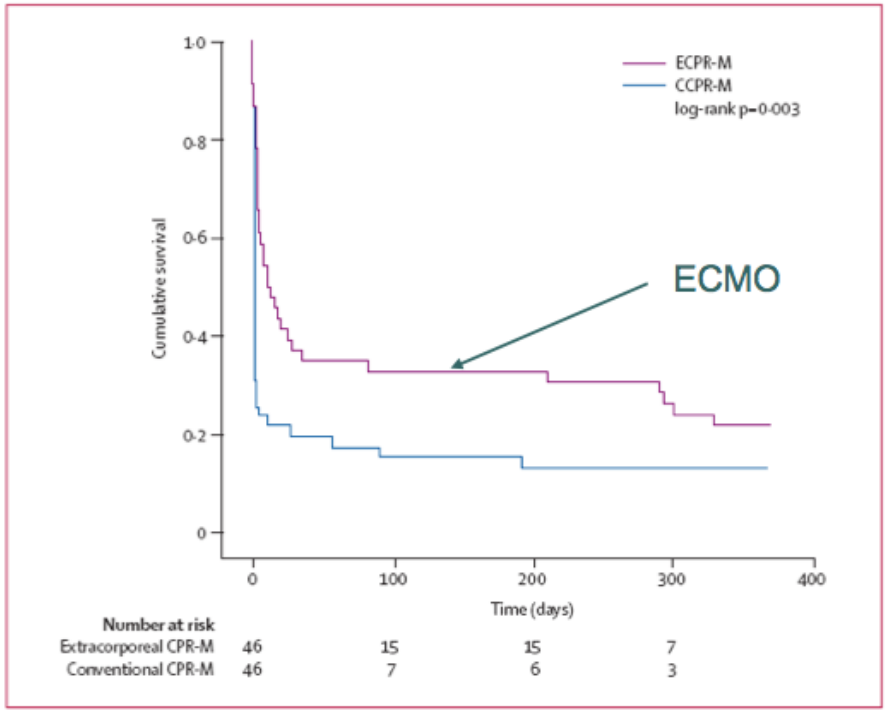


Figure 3: Kaplan-Meier plot of the survival curves in the extracorporeal CPR-M and conventional CPR-M groups for 1 year

[1] Chen et al. Lancet 2008; 372: 554–61

CARDIOPULMONARY RESUSCITATION WITH ASSISTED EXTRACORPOREAL LIFE-SUPPORT VERSUS CONVENTIONAL CARDIOPULMONARY RESUSCITATION IN ADULTS WITH IN-HOSPITAL CARDIAC ARREST: AN OBSERVATIONAL STUDY AND PROPENSITY ANALYSIS[1]

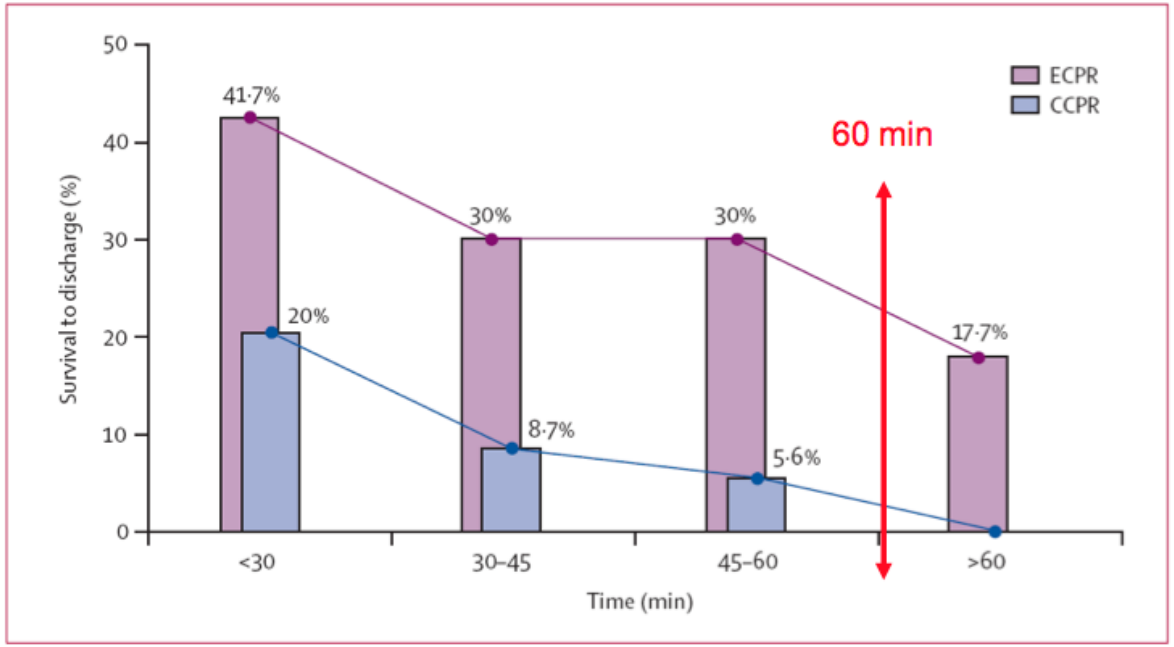
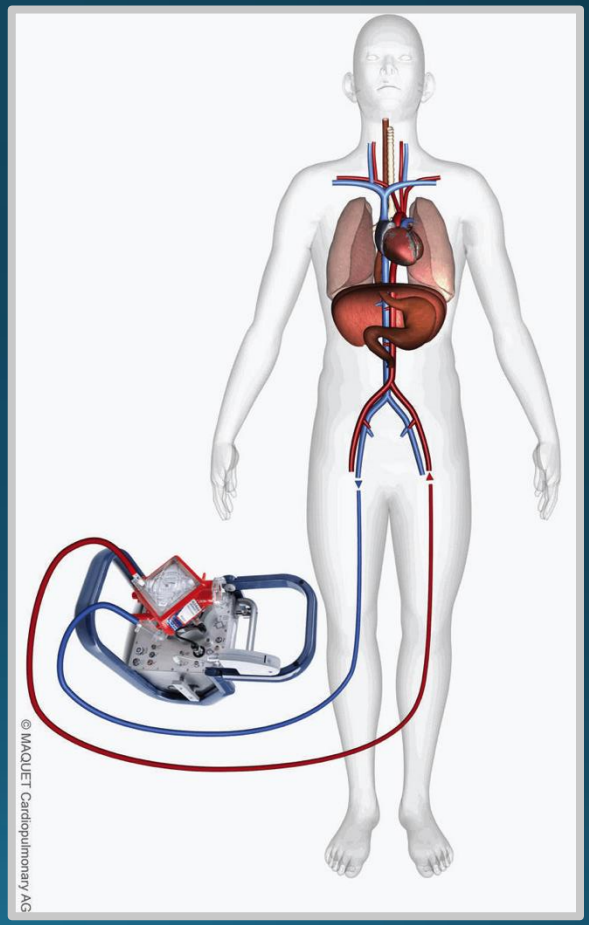


Figure 1: **Relation between CPR duration and the survival rate to discharge**
ECPR=extracorporeal CPR. CCPR=conventional CPR.

[1] Chen et al. Lancet 2008; 372: 554–61

POSSIBLE APPLICATIONS[1]:

- Cardiogenic Shock
- High-Risk PCI
- Valvular Interventions
- Mechanical bridge to other assist device
- Ventricular Tachycardia Ablation



[1] Diego Arroyo and Stephane Cook Percutaneous Ventricular Assist Devices: *New Deus Ex Machina*? Minimally Invasive Surgery ;Volume 2011; 1-10.

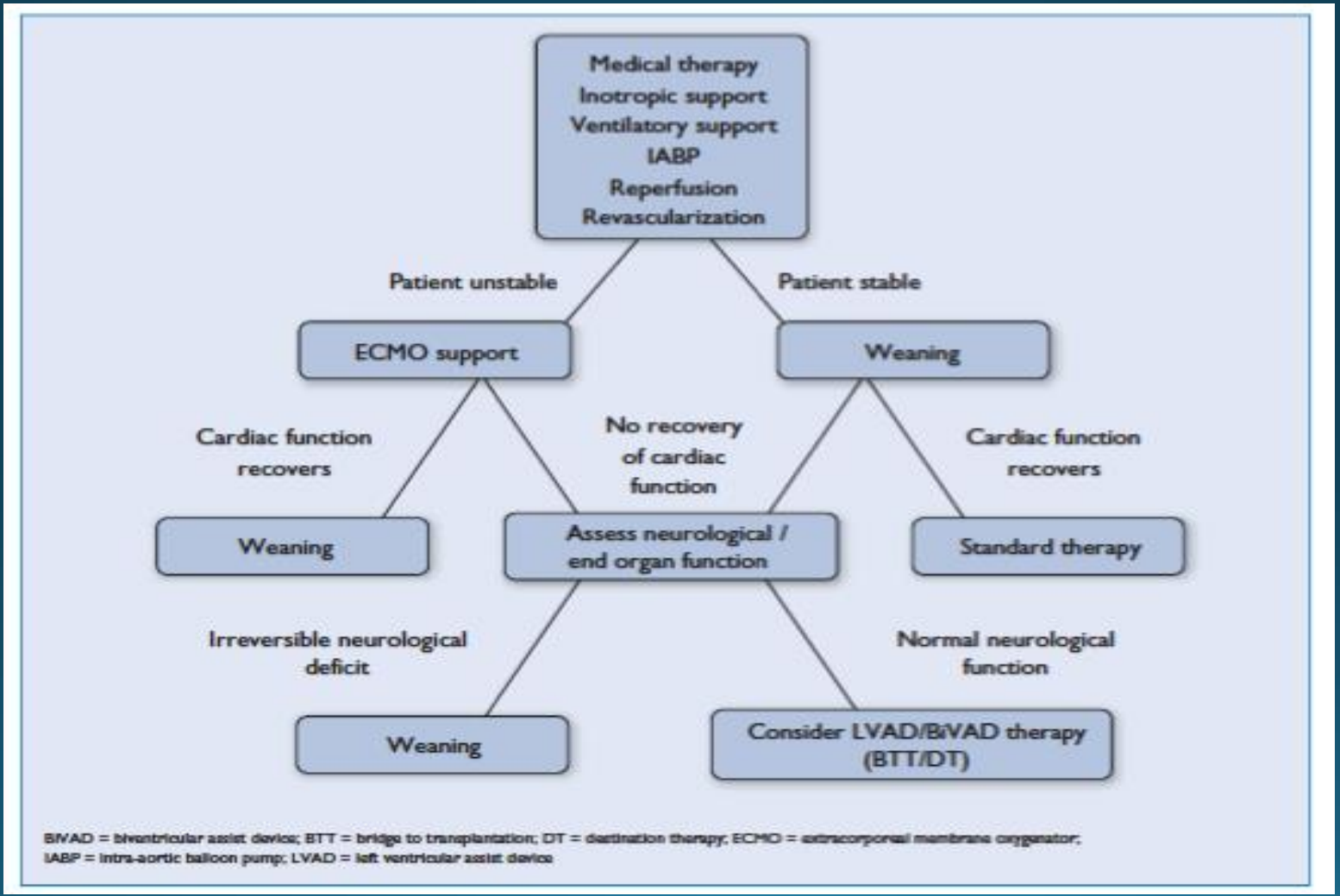
PERCUTANEOUS CARDIOPULMONARY BYPASS FOR CARDIAC EMERGENCIES[1]

Table 1 Indications and contraindications for PCPS

Reported indications	*Contraindications*
Resuscitation	*Absolute*
Cardiac arrest	Unwitnessed cardiac arrest
Cardiogenic shock	Aortic regurgitation
Cardiac trauma	Aortic dissection
Pulmonary insufficiency	
Status asthmaticus	*Relative*
Smoke inhalation	Cardiac arrest >30 min
Hyperalveolar proteinosis	No correctable anatomic defect
Drug overdose	Terminal illness
Pulmonary edema	Diabetes mellitus
Massive pulmonary embolism	Peripheral vascular disease
Hypothermia	Recent cerebrovascular accident
Procedural support	
Assisted angioplasty	
Pulmonary embolectomy	
Port access coronary artery bypass	
Resection of cerebral arteriovenous malformation	
Donor heart preservation	
Abdominal aortic graft replacement	
Tracheal reconstruction	

[1] Kurusz M, Zwischenberger J. Percutaneous cardiopulmonary bypass for cardiac emergencies. *Perfusion.* 2002;17:269-277.

ESC / EACTS GUIDELINES: TREATMENT ALGORITHM FOR CIRCULATORY SUPPORT

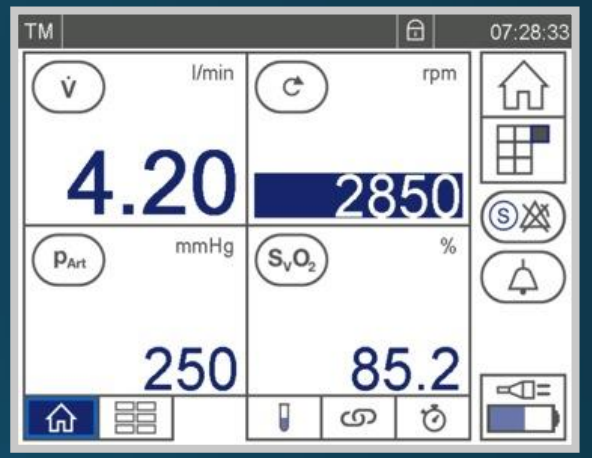


Source: ESC/EACTS Guidelines on Myocardial Revascularization; European Heart Journal (2010) 31, 2501–2555; doi:10.1093/eurheartj/ehq277

WHY CONSIDER ECLS?

- Patients in profound refractory CS have dismal survival rates
- ECLS is a form of Cardiopulmonary Bypass that can be accomplished percutaneously
- ELSO registry data supports good outcomes in CS patients
  - >50% in pediatrics
  - 35% in adults
- Provides circulatory support up to 7 liters
- Provides oxygenation
- Affords temperature regulation (hypothermia)

CONSIDERING CARDIOHELP

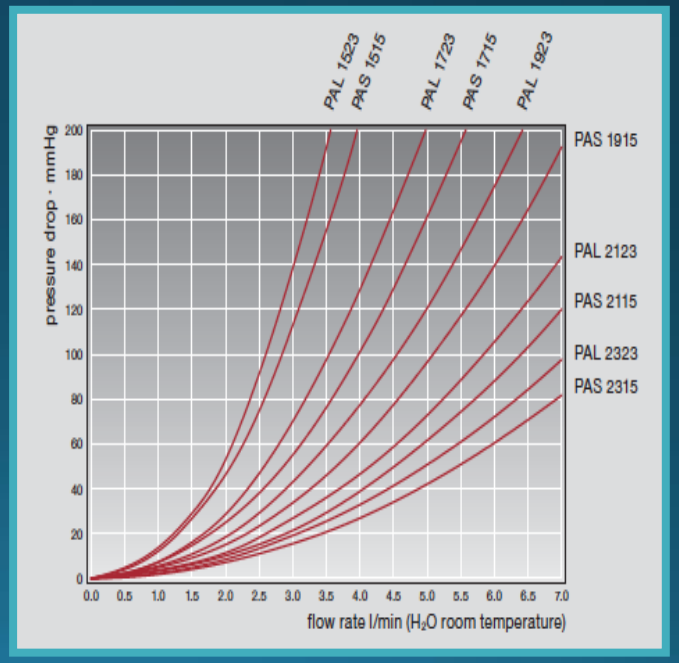


- Special software application suitable for the OR, Cath lab and transportation purposes
- Connection cable for internal sensors:
  - 3 x pressures
  - 1 x arterial temperature
- 1 external pressure sensor
- Venous probe head for measurement of:
  - Venous oxygen saturation
  - Hemoglobin
  - Hematocrit
  - Venous temperature

HLS CATHETER VASCULAR ACCESS

- Select appropriately sized cannulae to provide the desired extracorporeal blood flow
- The flow through a single MAQUET HLS cannulae at various pressure drops

		Flow (l/min)	
		Arterial cannula (15cm length)	Arterial cannula (23cm length)
Cannulae caliber (Fr) 100mmHg/H_20	15	2.9	2.6
	17	4.0	3.5
	19	5.0	4.5
	21	6.4	5.8
Cannulae caliber (Fr) 150mmHg/H_20	15	3.3	2.7
	17	4.3	3.8
	19	5.5	5.0
	21	7.0	6.4



CASE 2

- 52 yo gentleman with CAD s/p PCI to prox and distal RCA in 2006, and tobacco use presented with 2 days chest pain and SOB.

- Has not seen a physician in 15 yrs and stopped all his meds despite Hx of cardiac stents

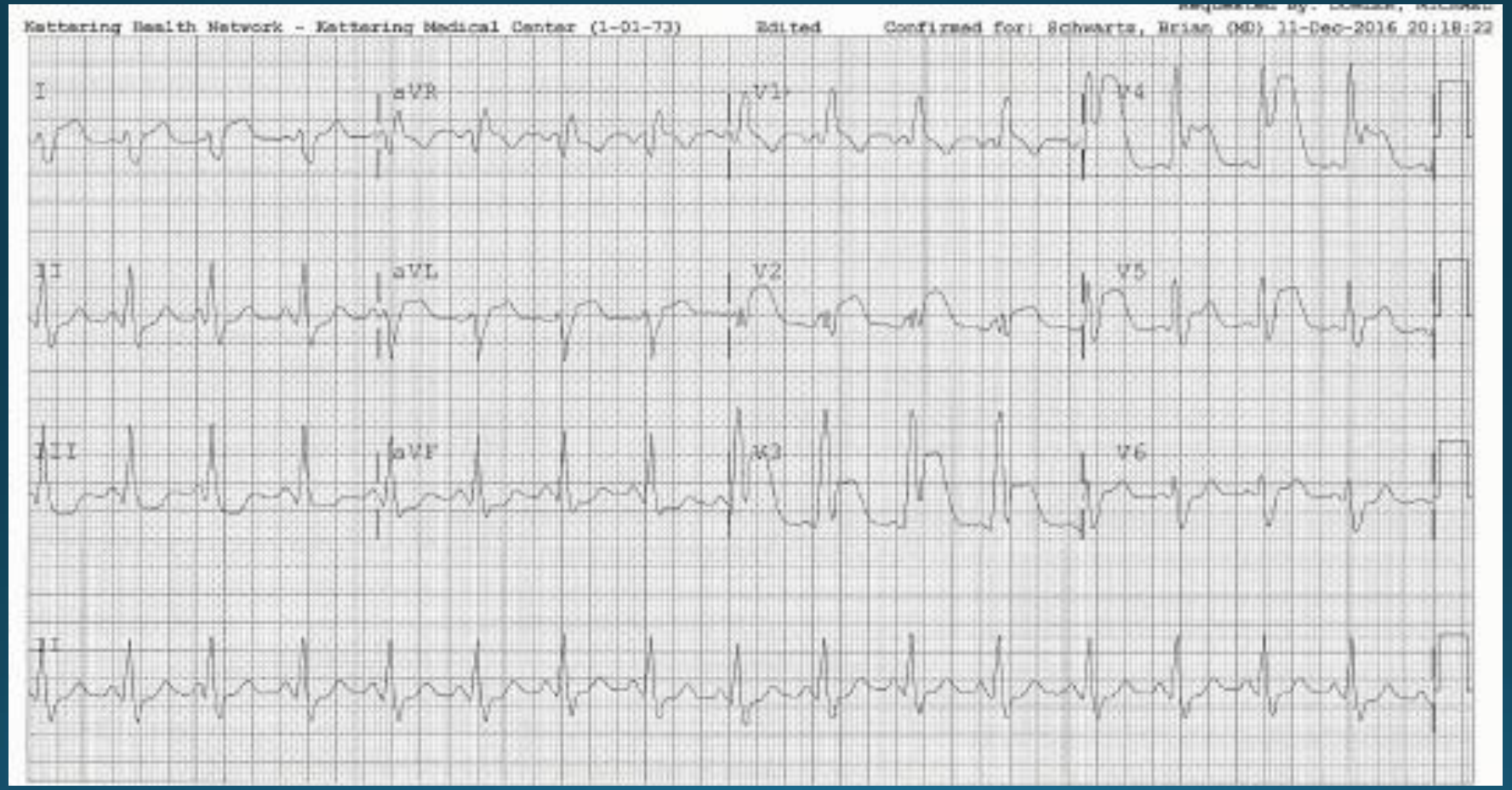
- O/E AAOx3, in moderate distess, diaphoeritc & SOB

- BP 60/40

- EKG showed ST elevation in the anterolateral leads.

- Emergent cardiac cath

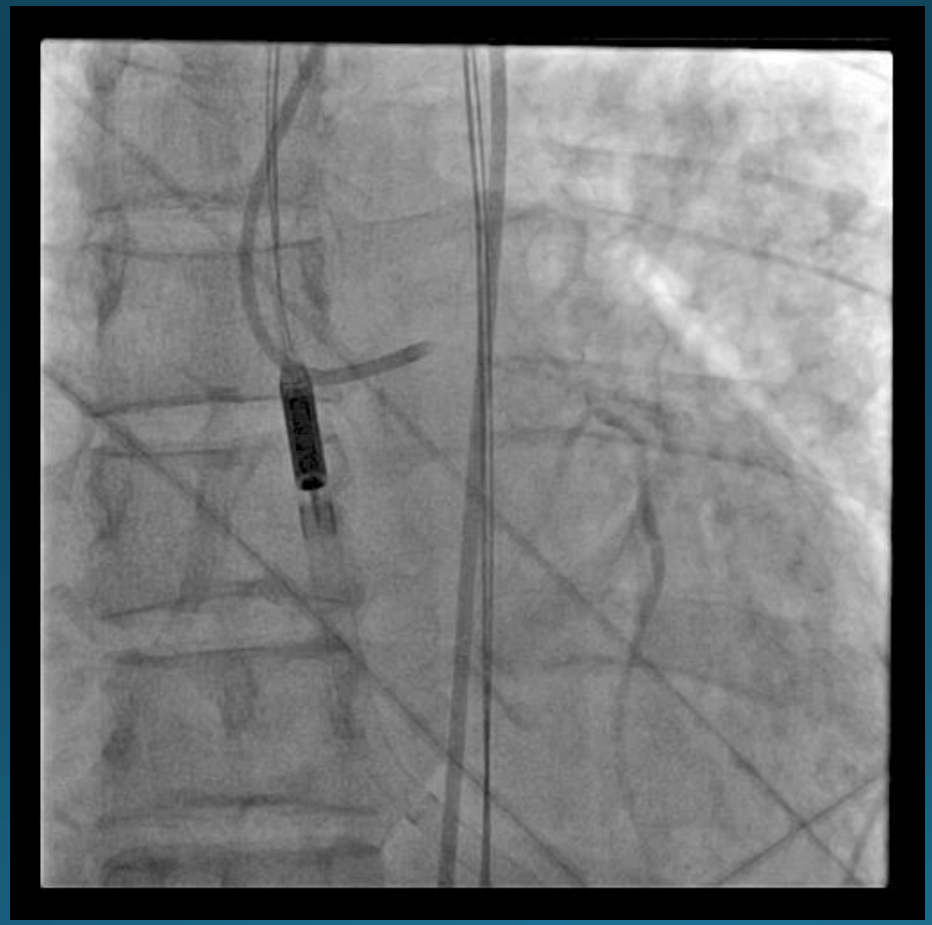
EKG

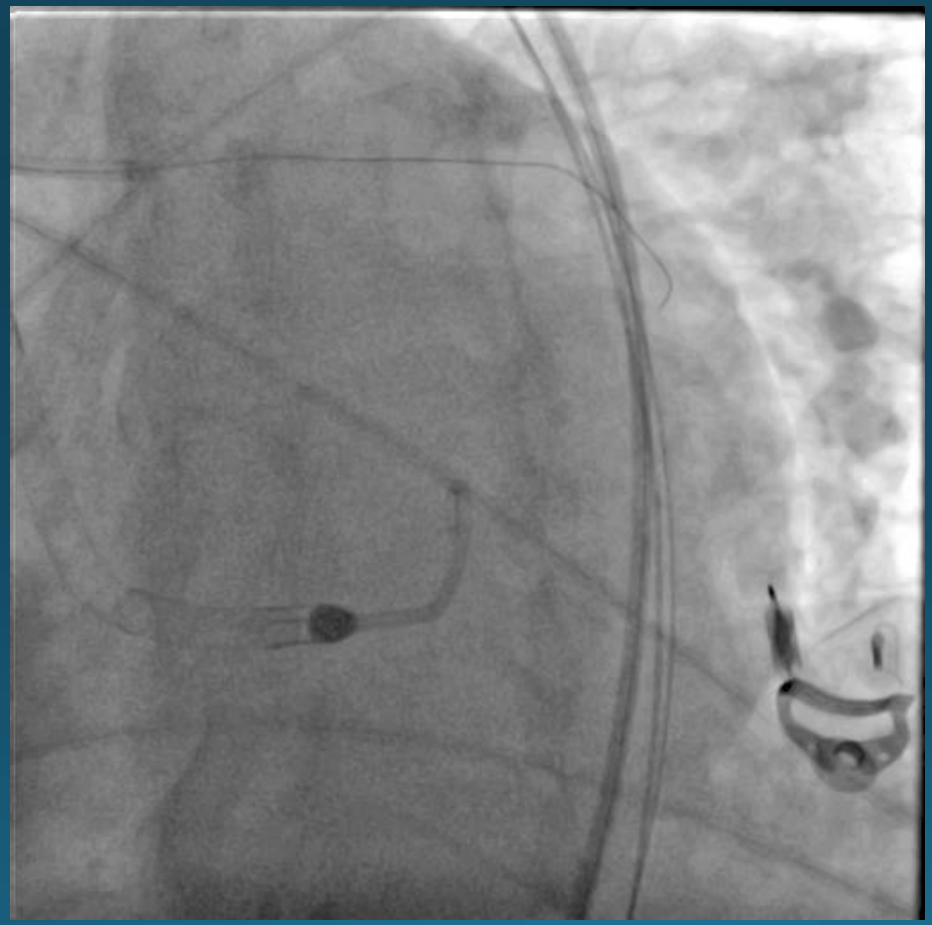

Kettering Health Network - Kettering Medical Center (1-01-73) Edited Confirmed for: Schwartz, Brian (MD) 11-Dec-2016 20:18:22
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

- Diagnostic LHC revealed: 99% proximal LM and 50% distally, 95% prox LAD, 75% mid. LCx: 100% in the mid segment, OM1 has 30%, RCA 40% prox and 100% distaly.

- Immediately Stabalize CO with Impella while ECMO Team assembeled.

- PCI to LM , LAD prox and mid as well as OM1






PHILIPS
CATH, PATIENT
18291120161211
12/11/2016 11:42:54AM TIS0.1 MI 0.5
X7-2t/KMC TEE
FR 50Hz
13cm
M4
2D
69%
C 50
P Low
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P R
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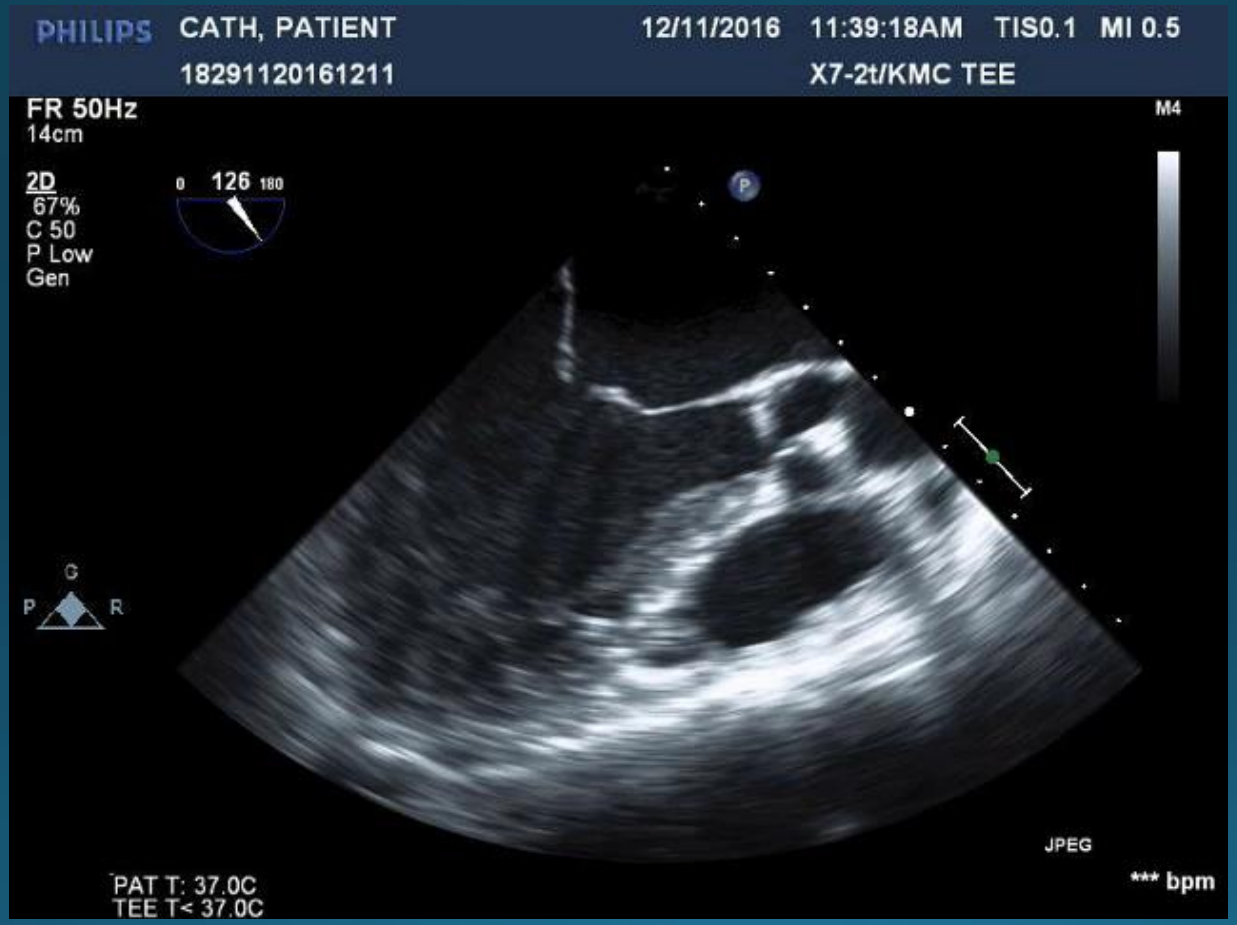
PAT T: 37.0C
TEE T: 37.6C


PHILIPS
CATH, PATIENT
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12/11/2016 11:39:18AM TIS0.1 MI 0.5
X7-2t/KMC TEE
FR 50Hz
14cm
M4
2D
67%
C 50
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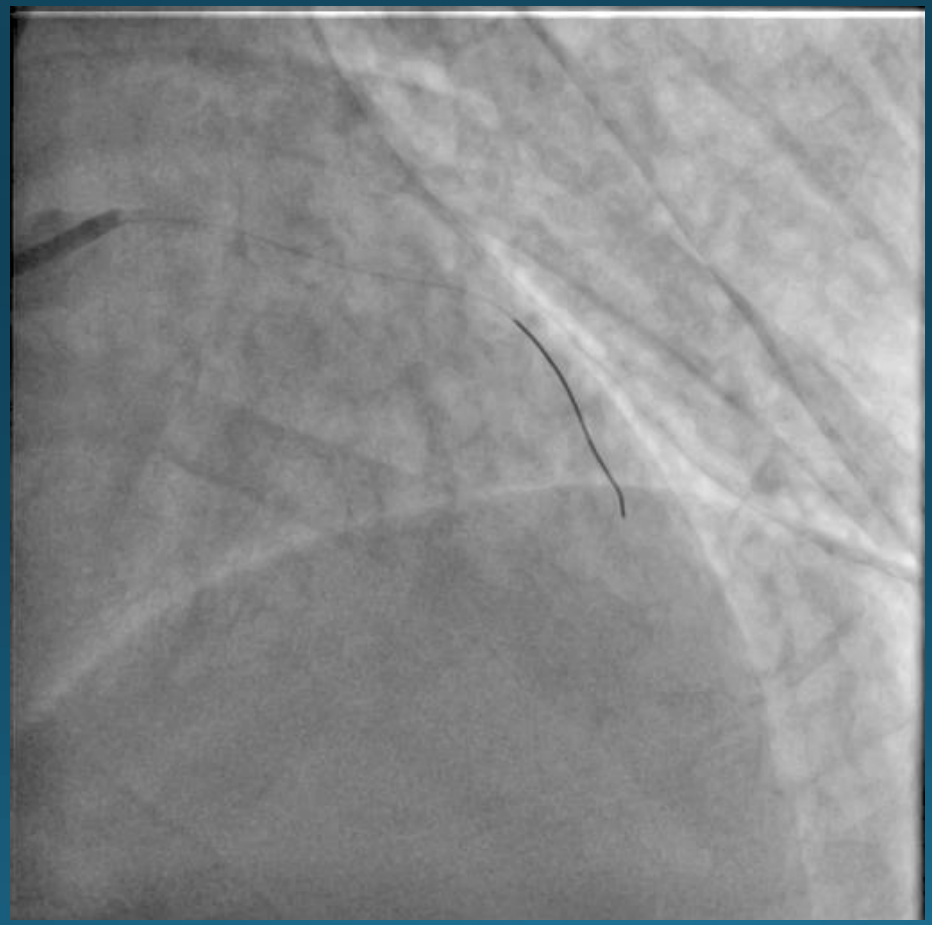
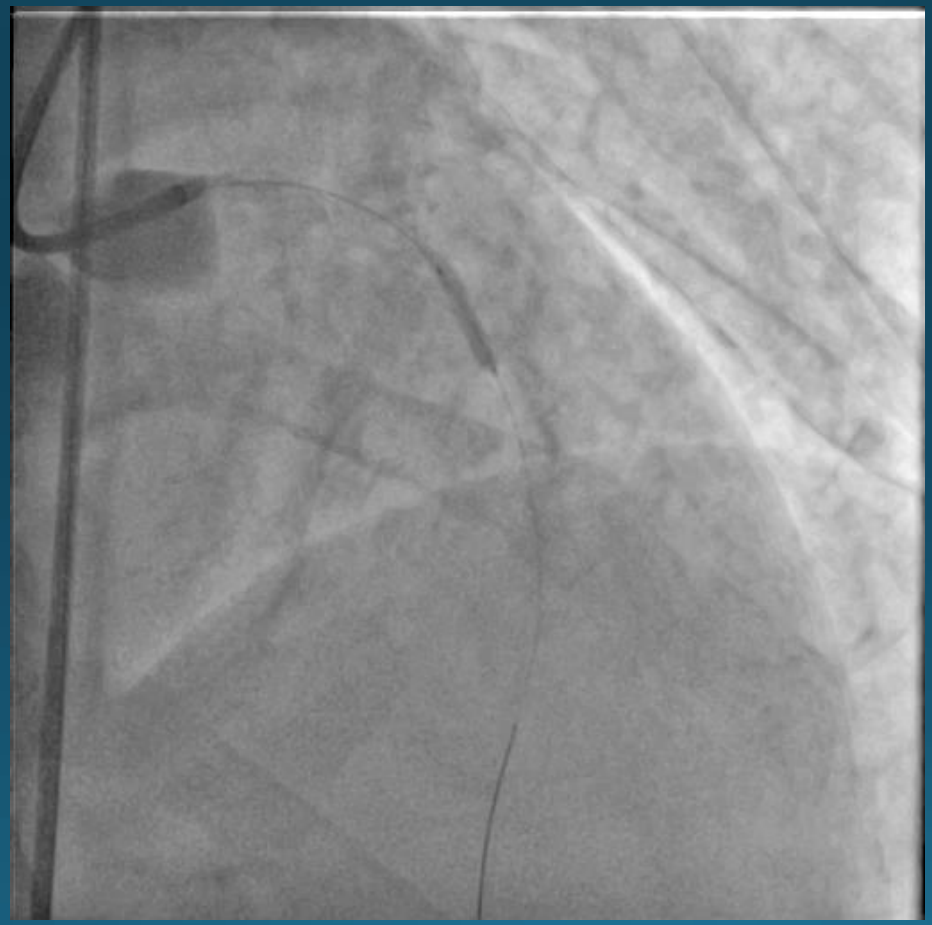
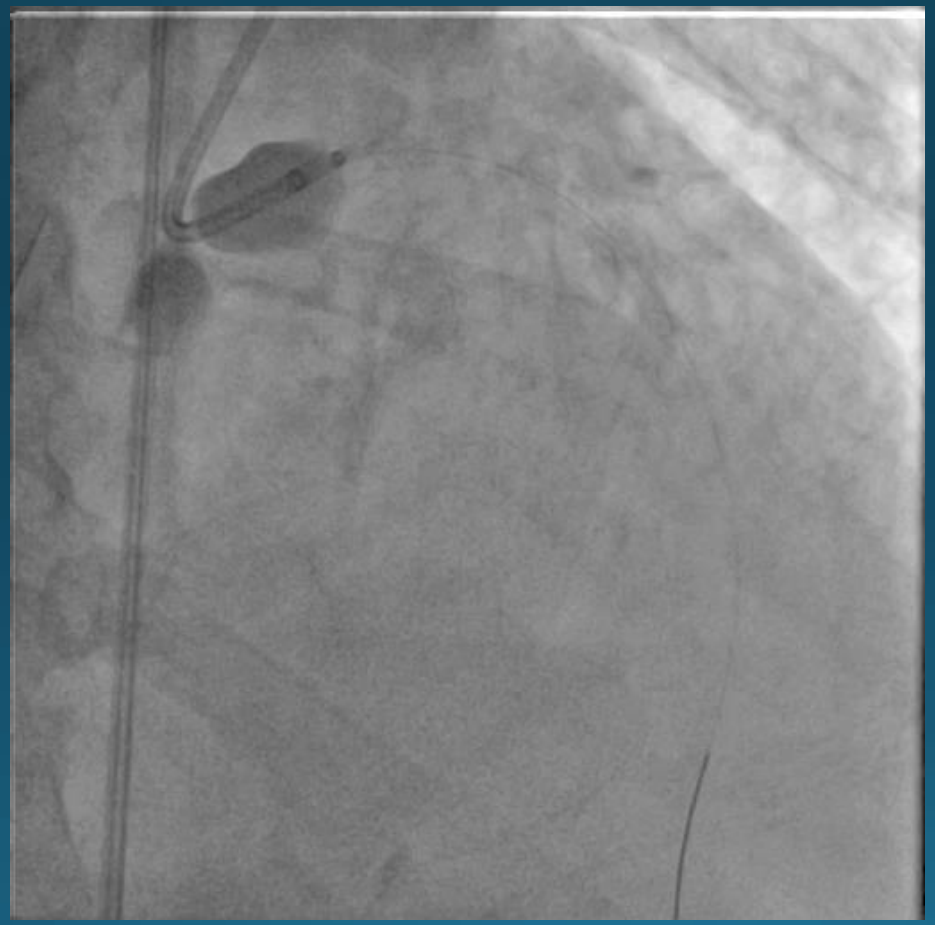
PAT T: 37.0C
TEE T< 37.0C



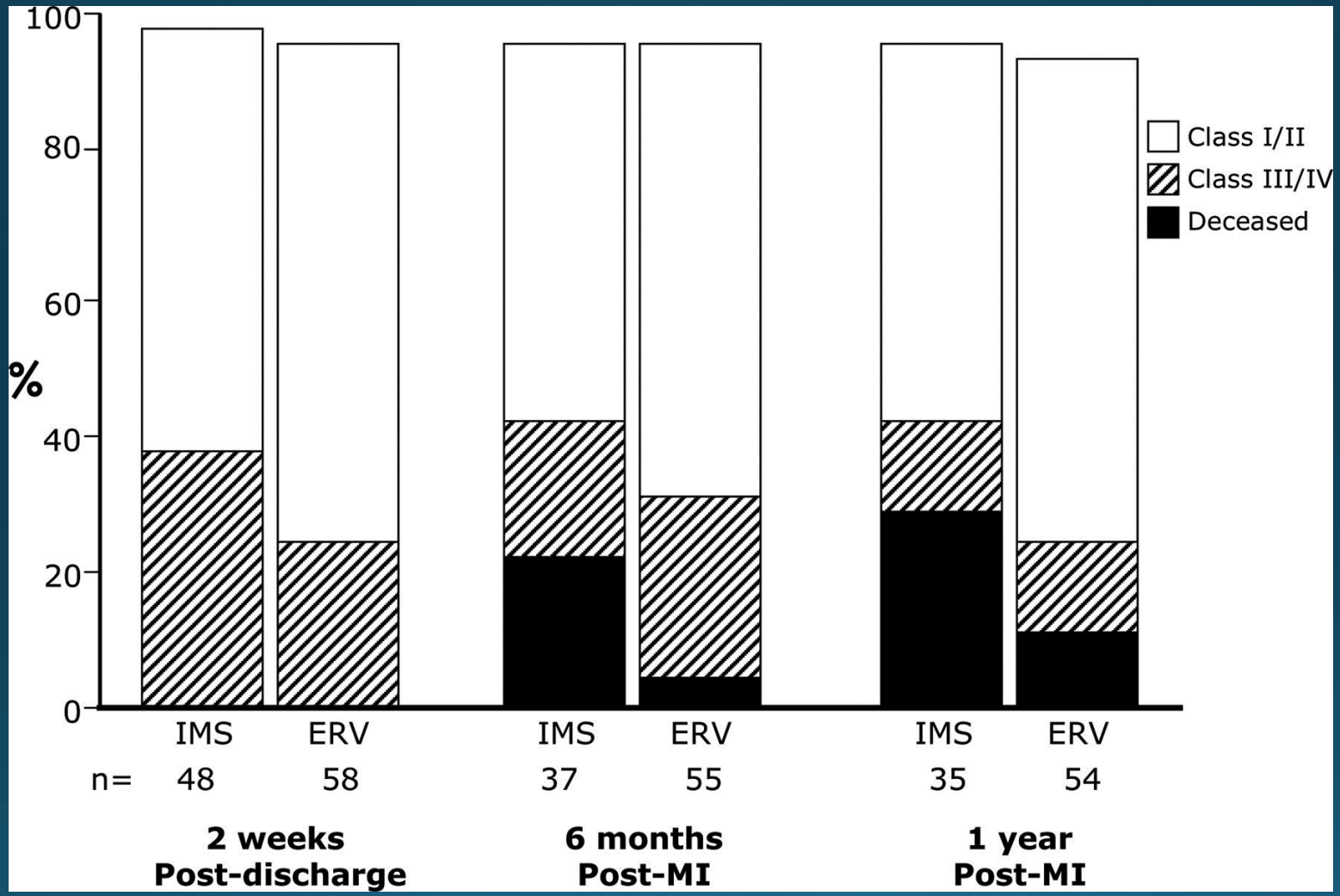




Out Come

- Hemodynamics Stabilized in our lab on ECMO
- PCI Completed of LAD and CX
- Transferred to UC
- Kept on ECMO 6 days
- By Day three he was extubated awake and alert
- Scheduled to Have Cannula Removed on Monday at noon
- Hemmhoragic Stroke at 10 am

Functional status in the SHOCK trial.60 The majority of patients who survived 2 weeks after discharge had good functional status (and quality of life) at that time point.



Harmony R. Reynolds, and Judith S. Hochman Circulation. 2008;117:686-697

Identify: Minimize Duration of Shock

Suspect
Cardiogenic Shock
See A

Confirm
CGS Diagnosis
reassess every 1-2 hours if criteria not initially met
See B

Activate
Heart Recovery Team/
Cardiac Cath Lab

Suspect Shock A

Consider any of these criteria:

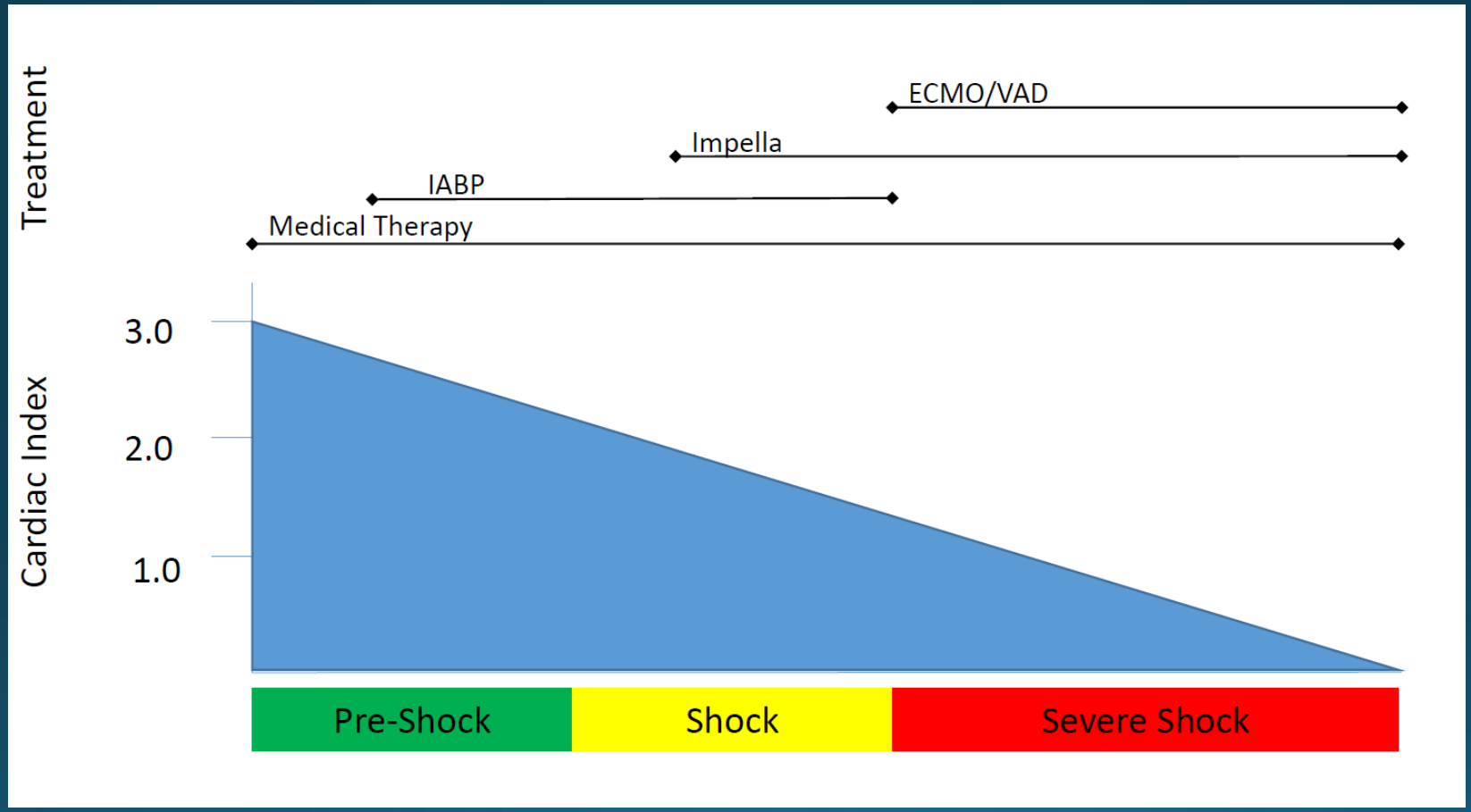
- Cool, clammy, pale skin
- Confusion/anxiety
- Rapid, shallow breathing
- SBP < 90 mmHg > 30 min
- Inotrope/vasopressor and/or IABP to maintain SBP > 90 mmHg
- Decrease in urine output (<0.5 cc/kg/h)
- Serum lactate level > 2 mmol/L

Diagnose CGS B

- STEMI/Non-STEMI
- ECG ST segment abnormalities
- Troponin
- ECHO (assess cardiac function)

If PA Catheter (PAC) available:

- Cardiac Index (CI) < 2.2 L/min/m^2
- pulmonary capillary wedge pressure (PCWP) > 18 mmHg
- Cardiac power output (CPO) < 0.6 watts

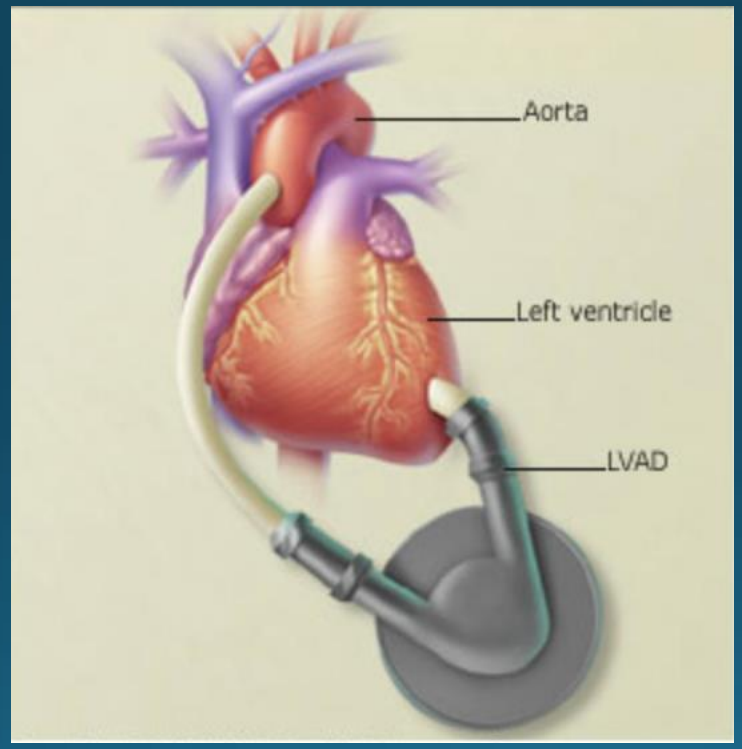

Treatment
ECMO/VAD
Impella
IABP
Medical Therapy
Cardiac Index
3.0
2.0
1.0
Pre-Shock
Shock
Severe Shock

ECMO

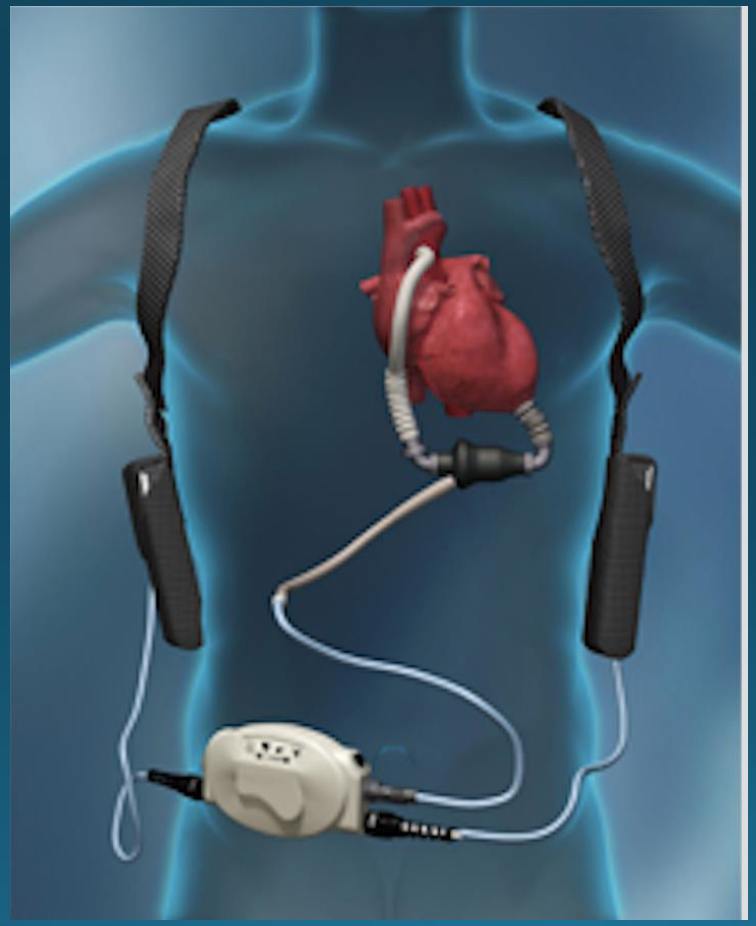
- Expensive Bailout providing maximal life support.
- Multidisciplinary team
- 24 hr perfusionists in house
- Advanced Care Unit with trained nursing

- What is the end point?
  - Rest and recovery of Myocardium
  - LVAD
  - Transplant

Future Therapies
LVADs



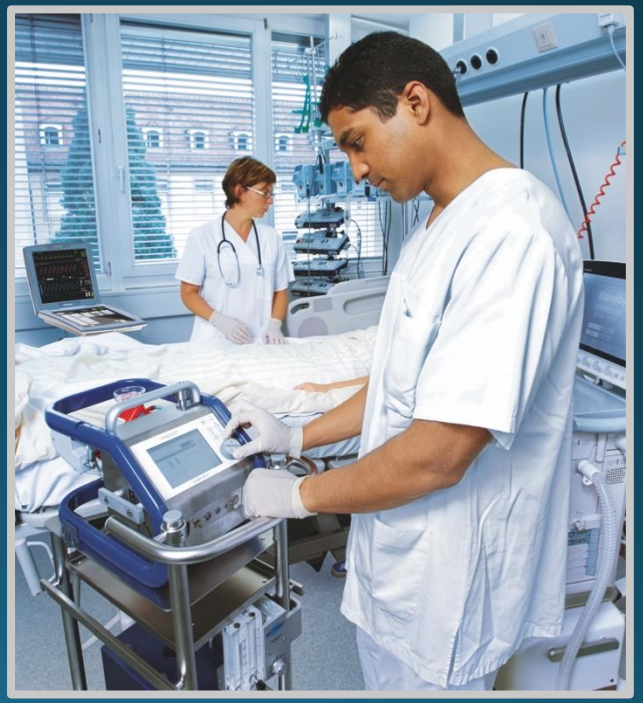
AMI



Thank You

CONSIDERING CARDIOHELP

- Provide circulatory and pulmonary support
- High flow percutaneous CPB in the cath lab to unload the entire heart and support end organ perfusion
- Rapidly deployable, efficient, time-tested fem-fem perfusion provides complete CPB up to 7 liters per minute
- Integrated patient monitoring with ability to auto-regulate
- Transportable throughout the hospital



Impella® for AMI Cardiogenic Shock

Identify[1-3]

- SBP < 90 mmHg or on inotropes/pressors
- Cold, clammy, tachycardia
- Lactate elevated > 2 mmol/L

Cardiogenic etiology evaluation

- EKG (STEMI / NSTEMI)
- Echocardiography[4]
- If available, PA catheter, cardiac output, CPO, CI, PCWP, SvO_2[5-7]

Stabilize Early

- Impella Support pre-PCI[8-10]
- Reduce Inotropes/Pressors[11,12]

Complete Revascularization

- Per Guidelines[13,14]

Assess for Myocardial Recovery
(Weaning and Transfer Protocols)

- ↑ Cardiac Output
- ↑ Cardiac Power Output
- ↑ Urine Output
- ↓ Lactate
- ↓ Inotropes

Myocardial Recovery[15,16]

No Recovery Escalate (and Ambulate) or Transfer[17]

- Ongoing Left Heart Failure
- Assess for Right Heart Failure

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