Cardiogenic Shock: It Is About The Team And The Process

Great things in business are never done by one person. They're done by a team of people.

- Steve Jobs

Bassel Alkhalil

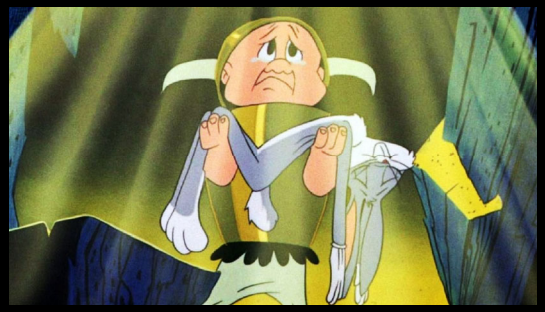
- No Conflict of interest

Goals

- Assess the vasopressor and inotrope use
- Know the devices to use for the right condition, Know how it operates, its limitation and complications
- Define Cardiogenic Shock
- Constant reassessment of the disease trajectory
- Recognize the value of the process and the team
- Cases

Depending on the etiology the outcome can range from:

- Complete LV recovery
- Survival with partial LV recovery
- Survival but with no LV recovery requiring advanced HF therapy



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Inotropes and Vasopressors

		Effects			
	Mechanism	Contractility	Heart Rate	SVR	Venous Tone
Dobutamine	α1 β1 β2	↑↑	↑↑	↓	
Milrinone	PDE-3 Inhibitor	↑↑	↑	↓↓	↓
Epinephrine	α1 α2 β1 β2	↑	↑↑	↑	↑
Norepinephrine	α1 α2 β1 β2	↑	↑	↑↑	↑↑
Phenylephrine	α1			↑↑	
Dopamine	α1 β1 Dopa	↑	↑	↑	
Vasopressin	V1			↑	

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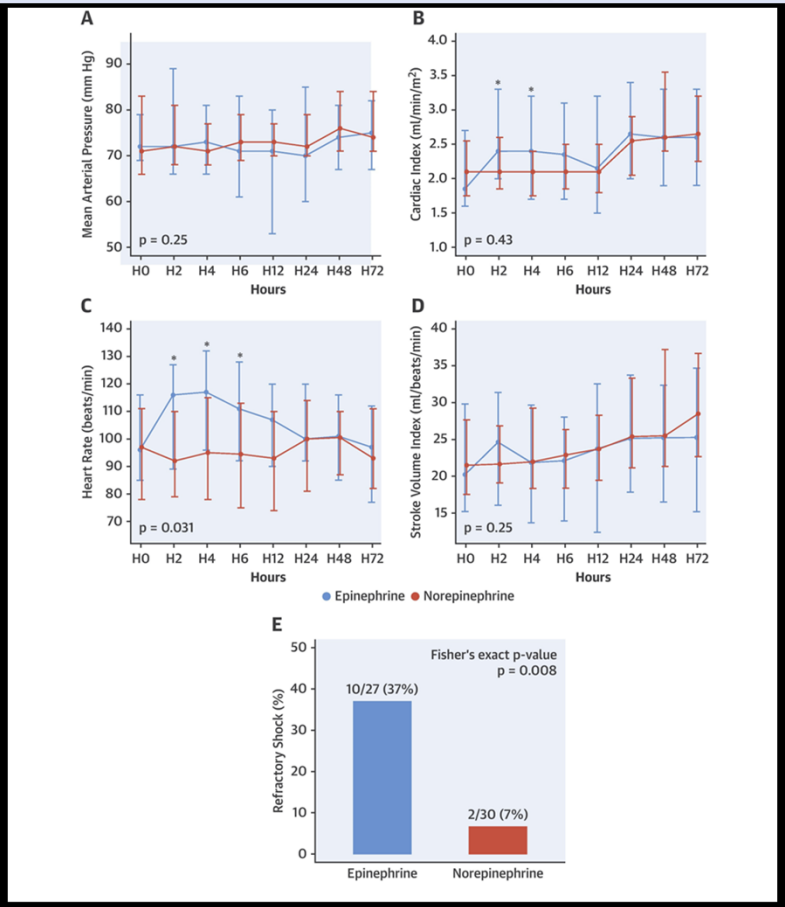
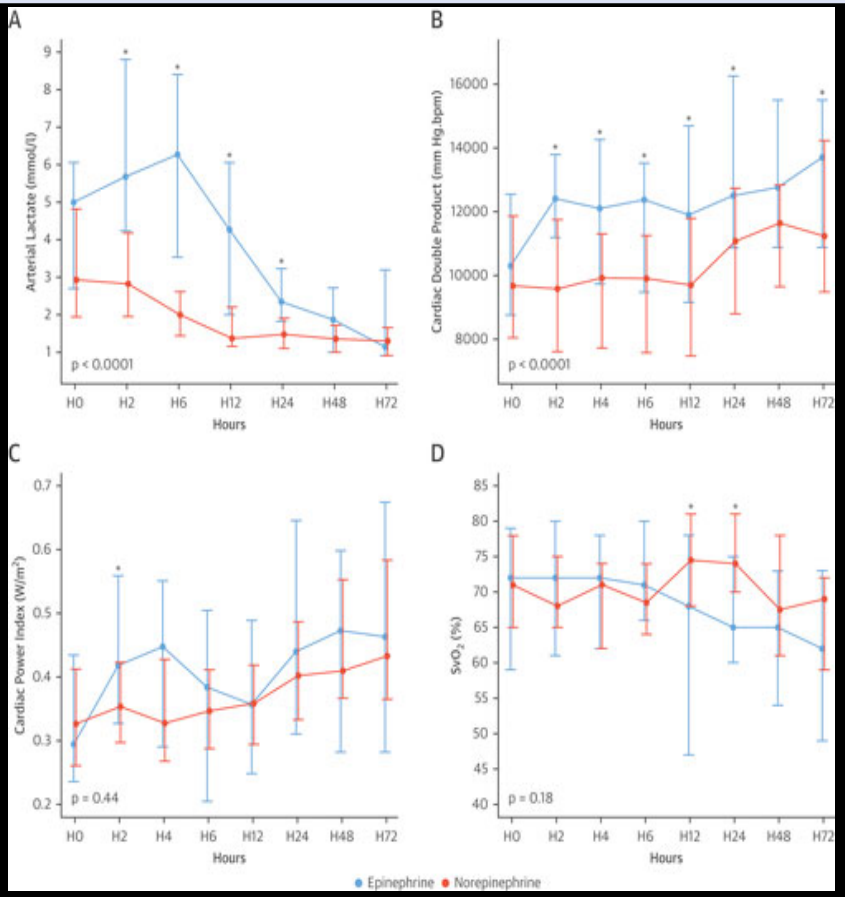
CENTRAL ILLUSTRATION: Epinephrine Versus Norepinephrine in Cardiogenic Shock After Acute Myocardial Infarction





Bruno Levy et al. *J Am Coll Cardiol* 2018; 72:173-182

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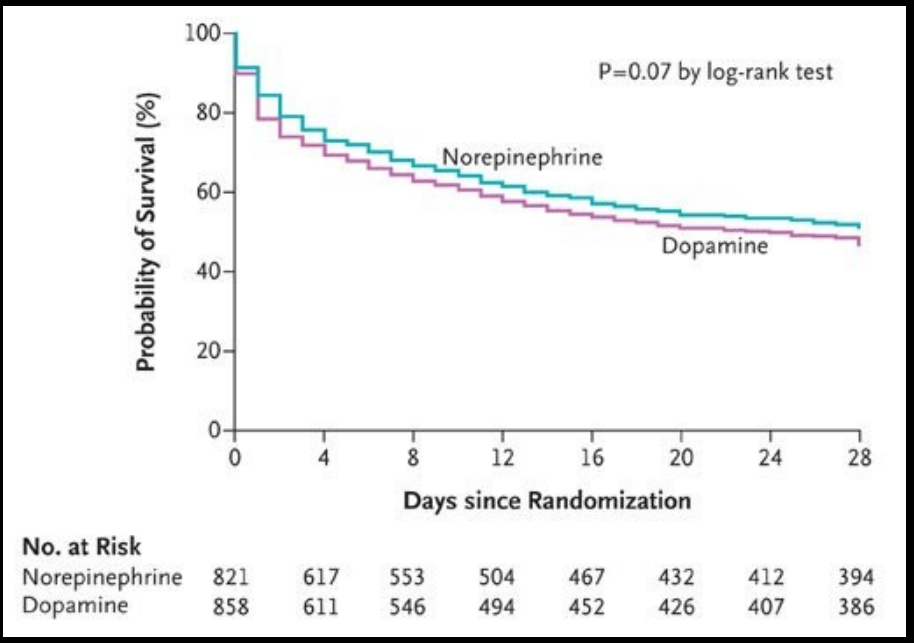
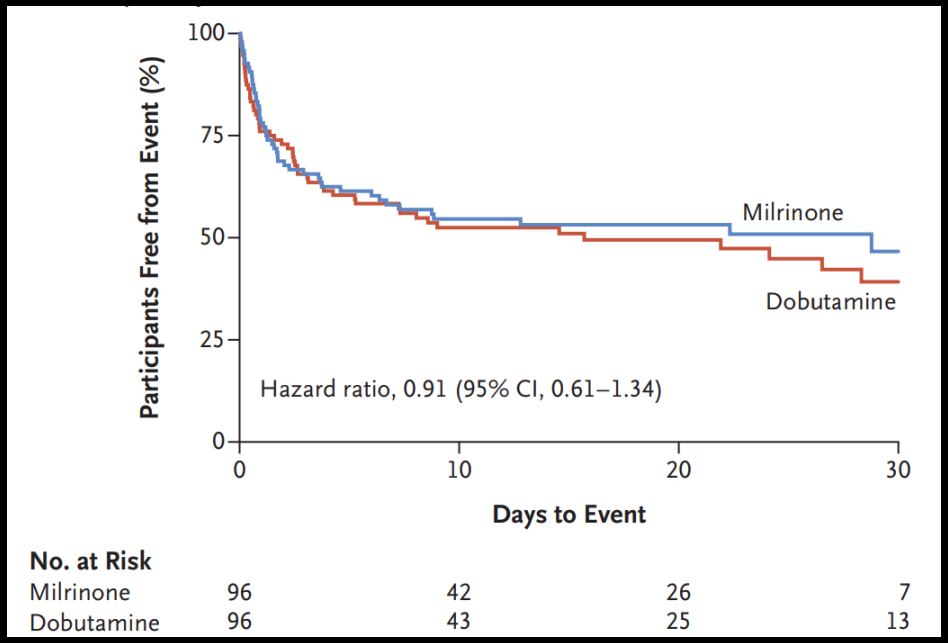


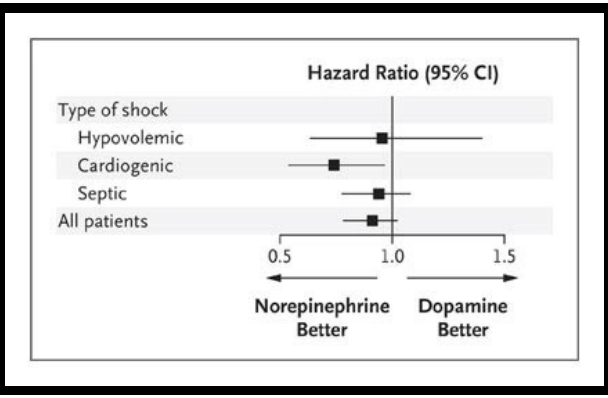
No. at Risk

Norepinephrine	821	617	553	504	467	432	412	394
Dopamine	858	611	546	494	452	426	407	386



No. at Risk

Milrinone	96	42	26	7
Dobutamine	96	43	25	13



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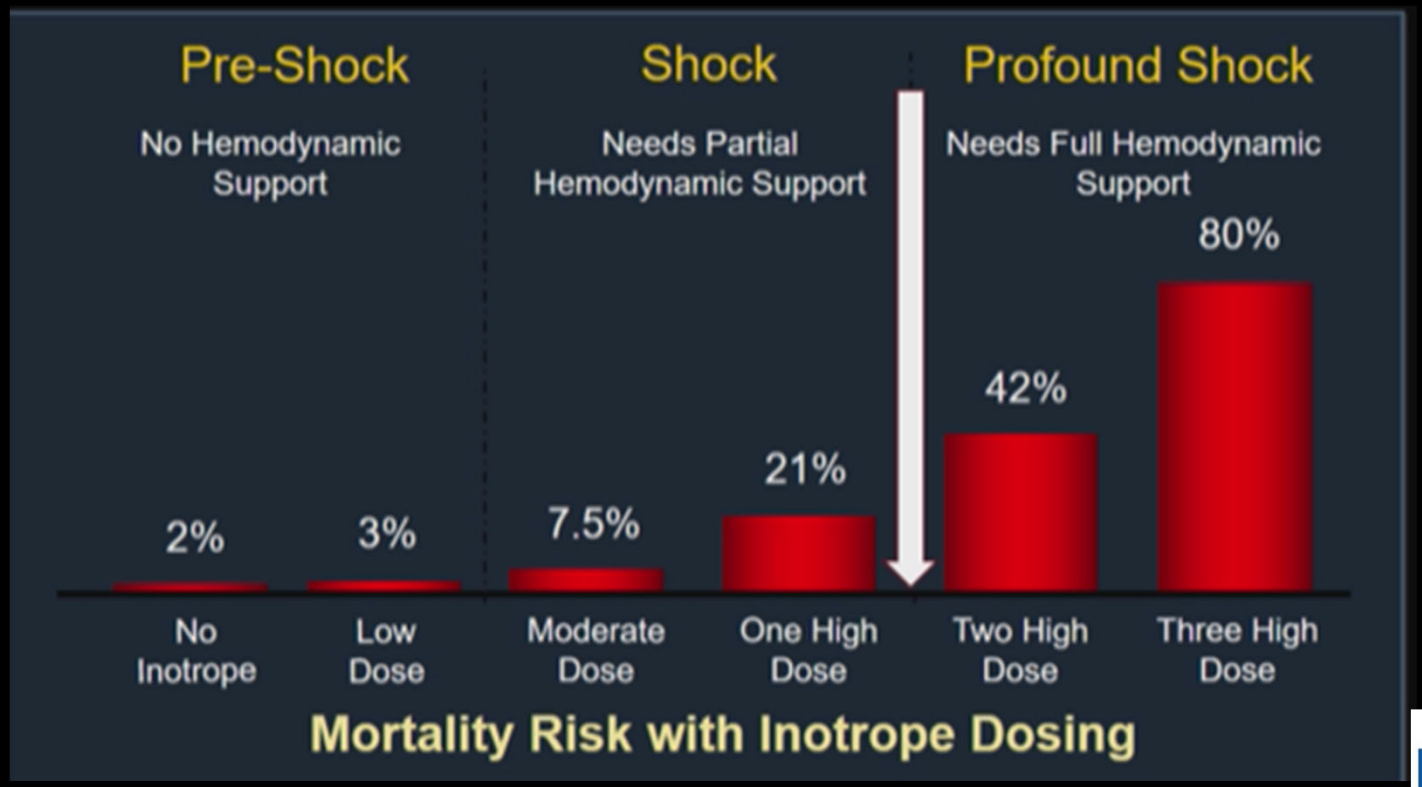
Cardiogenic Shock and adequate Therapy shift AWAY from Vasopressors to EARLY MCS



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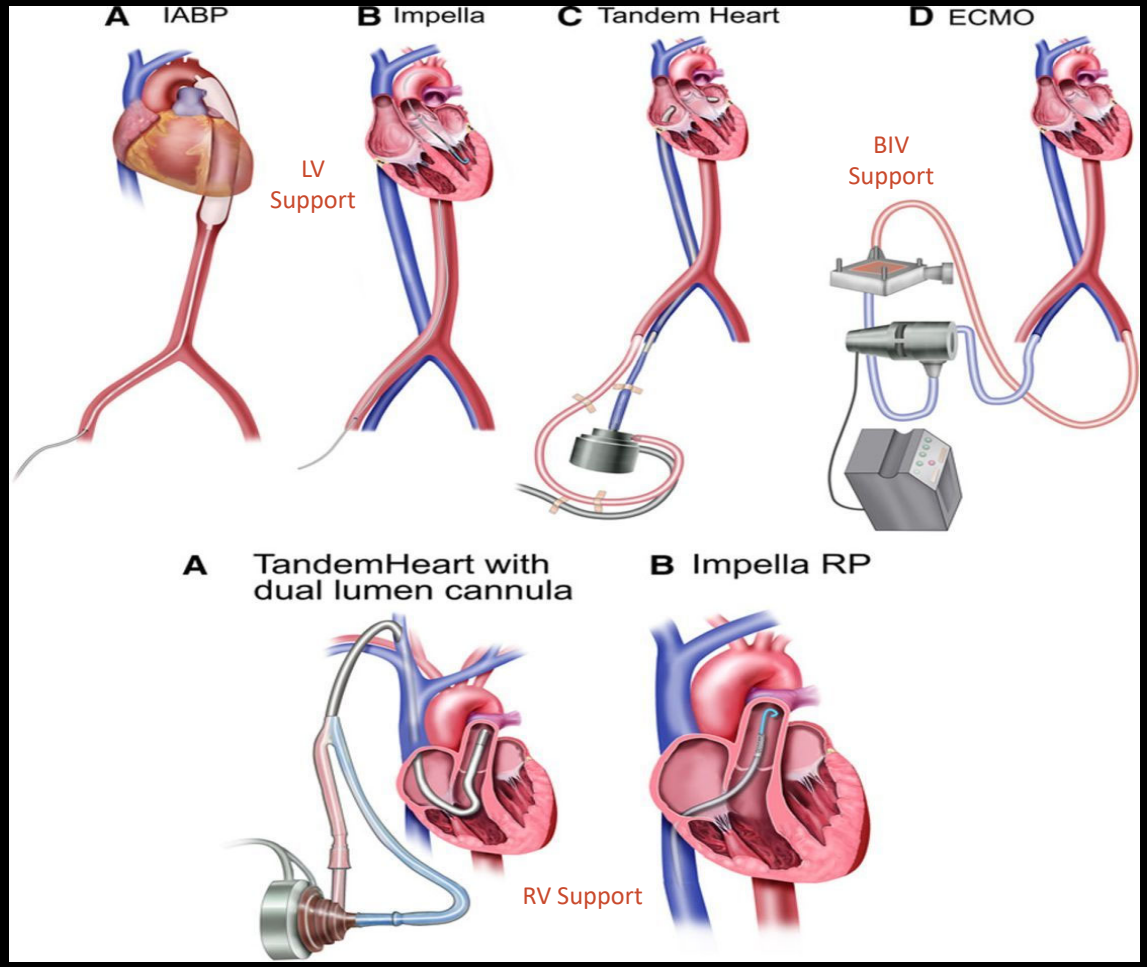

Aditya Mandawat. Circulation: Cardiovascular Interventions. Percutaneous Mechanical Circulatory Support Devices in Cardiogenic Shock, Volume: 10, Issue: 5, DOI: (10.1161/CIRCINTERVENTIONS.116.004337)

Definition of Cardiogenic shock

- Systolic BP<90 mmHg for more than 30 minutes (or support in order to maintain BP)
- Cardiac Index < 2.2 $L \cdot min^{-1} \cdot m^{-2}$
- Pulmonary Capillary Wedge Pressure (PCWP) >15 mmHg
- Markers of end organ perfusion
  - urine output < 30 ml/h
  - altered mental status
  - cool extremities
  - Lactate > 2 mmol/L

Not Mean BP

- Hypotension SBP<90 mmHg only does not define shock, it can be seen in pre-shock or in chronic HF patients
- You can still see shock in "Normotensive Shock" with hypoperfusion findings
- Hypoperfusion and SBP < 90 mmHg

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Case 1

53 yo lady with no previous cardiac hx came with rapidly evolving HF symptoms over few days. She was transferred to Norton

<u>LHC:</u>

Unremarkable coronary angiogram

AO 82/62 (68), 77/64 (69), AO sat 96%

LV 82/32 (31)

<u>RHC numbers</u>

RA 21, PA 30/24 (25), PCWP 23

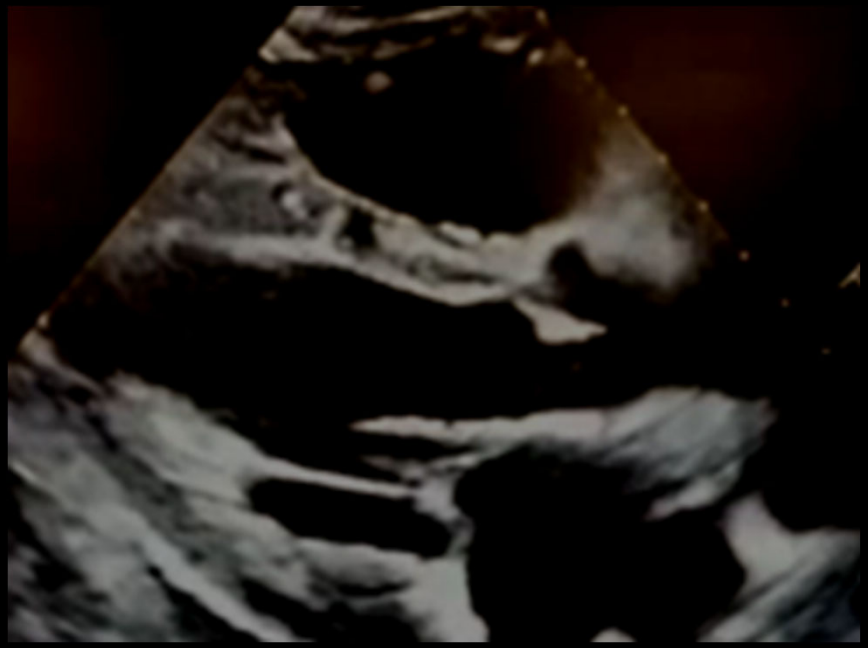
PA sat 35%

Fick CO 2.6 CI 1.3

Thermal CO 1.7 CI 0.9

<u>Intervention:</u>

LV impella placed, shortly after Impella RP was placed.



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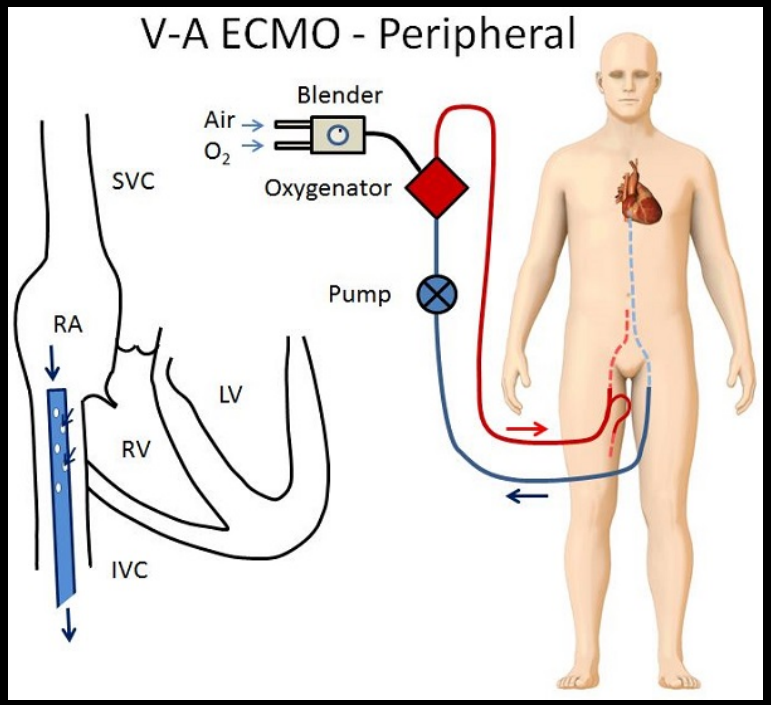
3 R
Reassess, React, Respond

- Patient Continued to deteriorate and was intubated Decision for VA ECMO made
- Had cardiopulmonary arrest right before VA ECMO was placed.
- (RP Impella removed) LV impella left in for LV vent

- She continued on VA ECMO and LV Impella with echocardiographic and hemodynamic improvement of her Biventricular function
- VA ECMO was decannulated first followed by LV impella.



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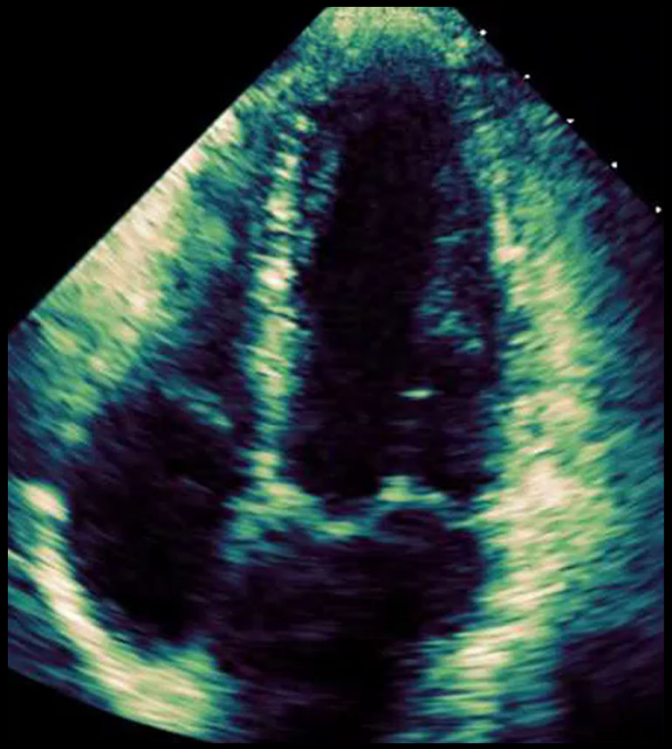
VA ECMO was decannulated first followed by LV impella



Doing well
Enrolled in cardiac rehab

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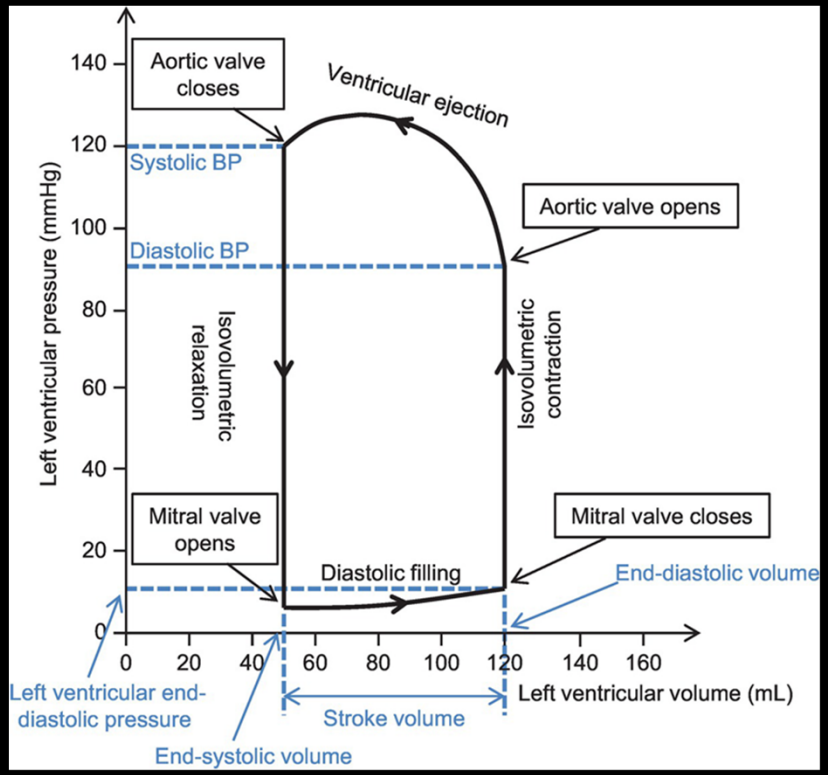
Pressure-Volume Loop



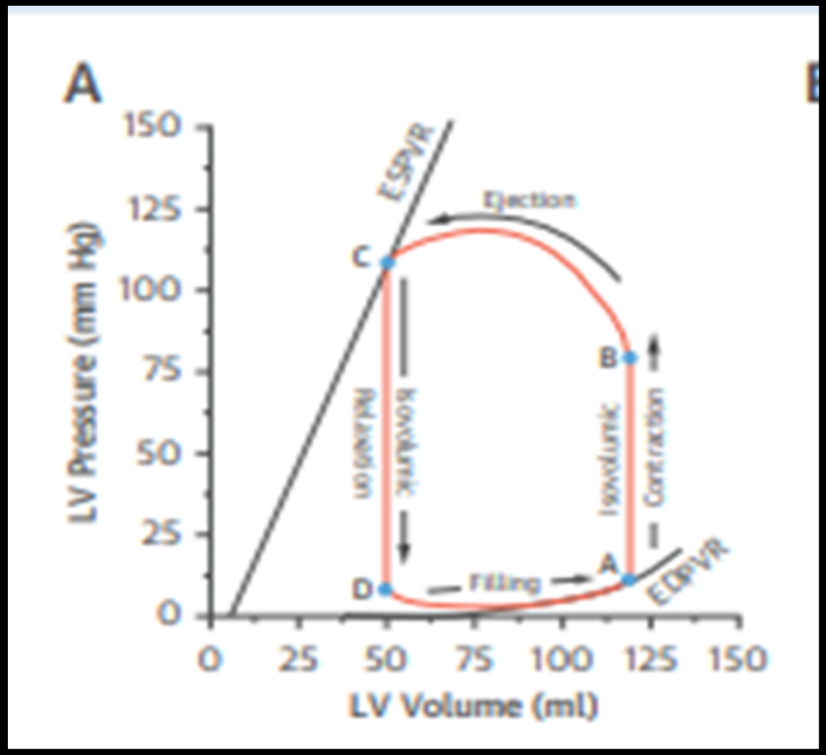


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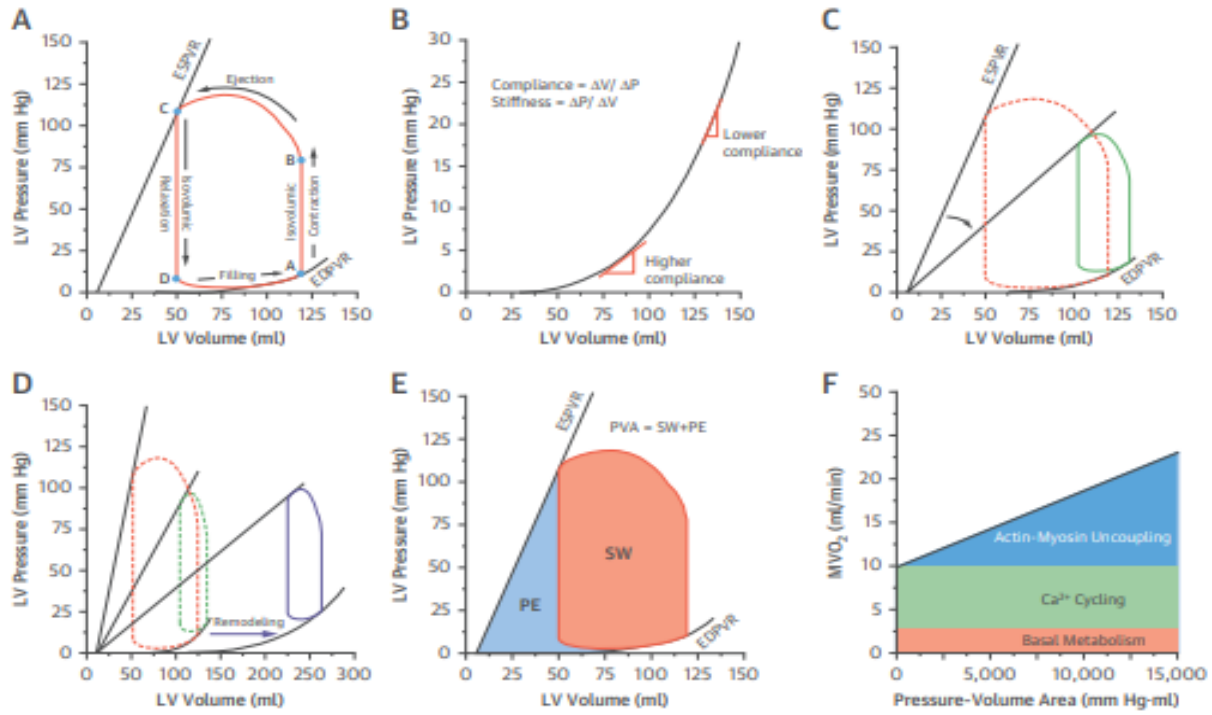
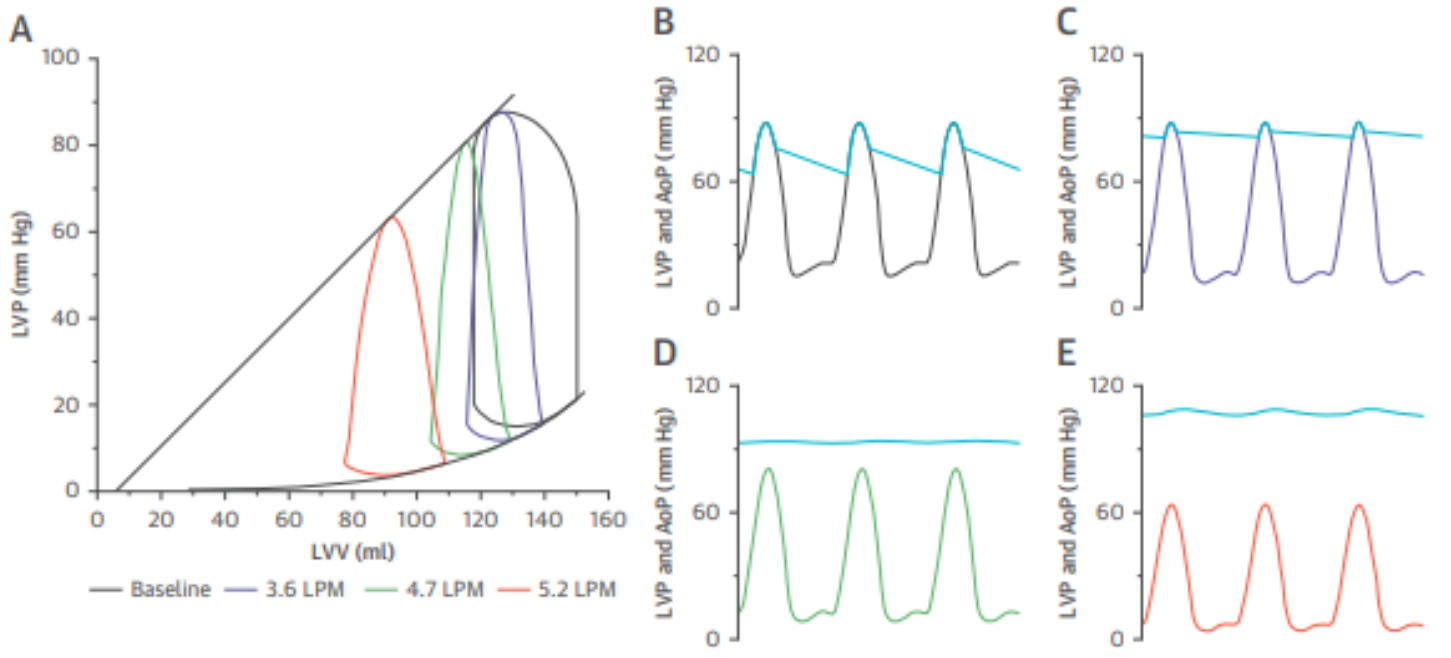
FIGURE 1 **PV Analysis of LV Hemodynamics During AMI and Following LV Unloading**



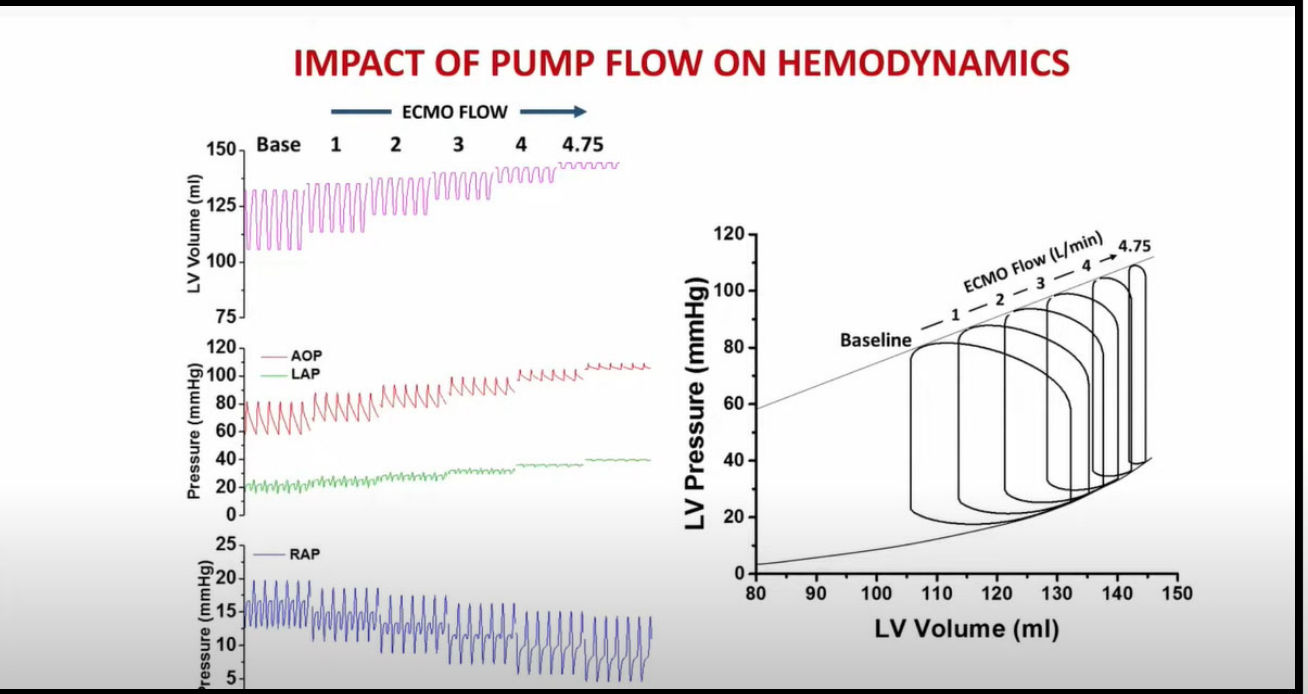
(A) The LV pressure-volume (PV) loop is bounded by the end-systolic pressure volume relationship (ESPVR) and end-diastolic pressure-volume relationship (EDPVR). **Point A** represents mitral valve closure and the onset of isovolumic contraction. **Point B** represents aortic valve opening and the onset of systolic ejection. **Point C** represents aortic valve closure and the onset of isovolumic relaxation. **Point D** represents mitral valve opening and the onset of the diastolic filling period. **(B)** The EDPVR characterizes passive ventricular properties. Ventricular compliance is determined by the slope of EDPVR, and decreases with increased filling pressures. **(C)** Following acute myocardial infarction, cardiac contractility is reduced, and the ESPVR slope is reduced. The PV loop shows a reduction in pressure generation and decrease in stroke volume **(green loop)**. **(D)** Following myocardial injury, ventricular remodeling leads to a rightward shift of the EDPVR, and further downward shifting of the ESPVR, causing LV enlargement and a decrease in LV contractility **(blue loop)**. **(E)** Mechanical work of the LV is composed of stroke work (SW) and potential energy (PE). The pressure-volume area (PVA) is the sum of the SW and PE, and represents the total mechanical work of the heart per beat. **(F)** Myocardial oxygen consumption (MVO2) is composed of basal metabolism, calcium (Ca^{2+}) cycling, and mechanical work. MVO2 is directly correlated with PVA. AMI = acute myocardial infarction; LV = left ventricular; ΔP = change in pressure; ΔV = change in volume.

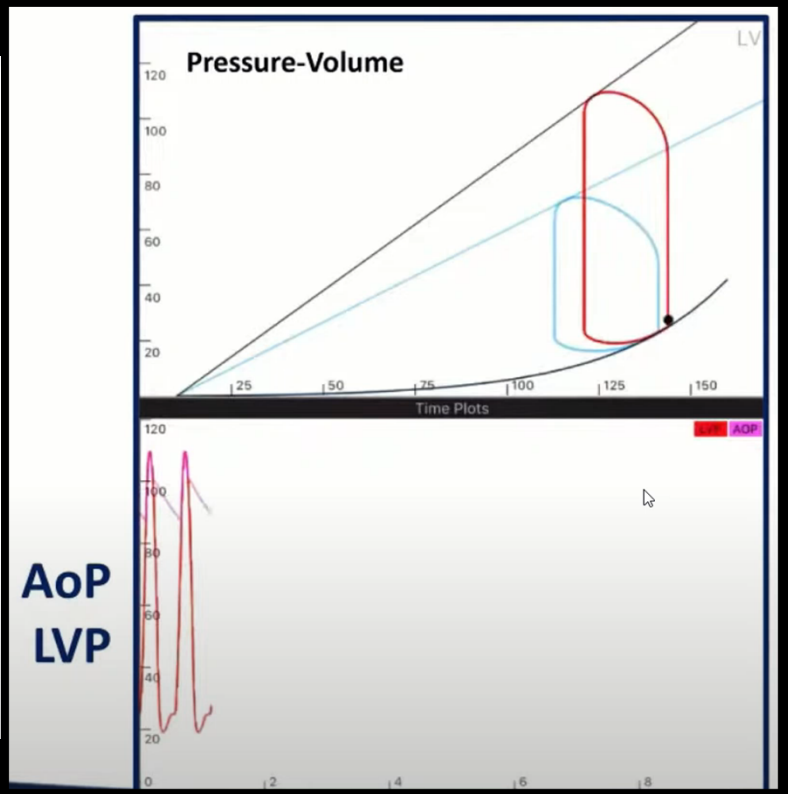
FIGURE 2 Hemodynamic Changes During LV Unloading by Mechanical Circulatory Support



(A) During acute myocardial infarction or in chronic heart failure, the PV loop shifts to the right along the EDPVR, resulting in elevated end-diastolic pressures **(black loop)**. With LV unloading by a continuous-flow left ventricular assist device (LVAD), the isovolumic contraction and relaxation periods are lost, and the PV loop assumes a triangular shape. Increases in LVAD support result in progressive leftward shifting of the PV loop to lower points on the EDPVR curve, with a reduction in LV volume (LVV). **(B to E)** Increases in LVAD flow result in uncoupling of LV pressure (LVP) and aortic pressure (AoP). LPM = liters per minute; other abbreviations as in Figure 1.

Effect of Peripheral VA ECMO on hemodynamics





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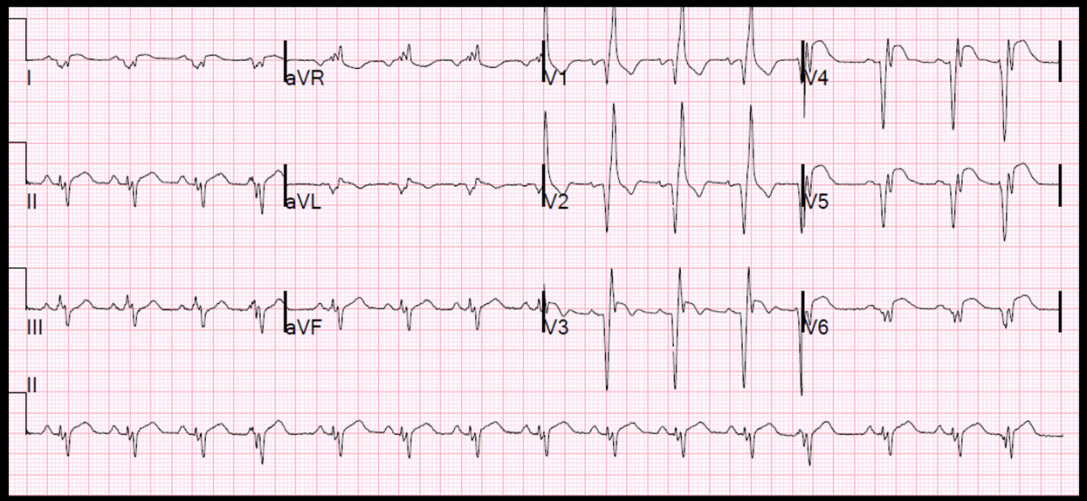
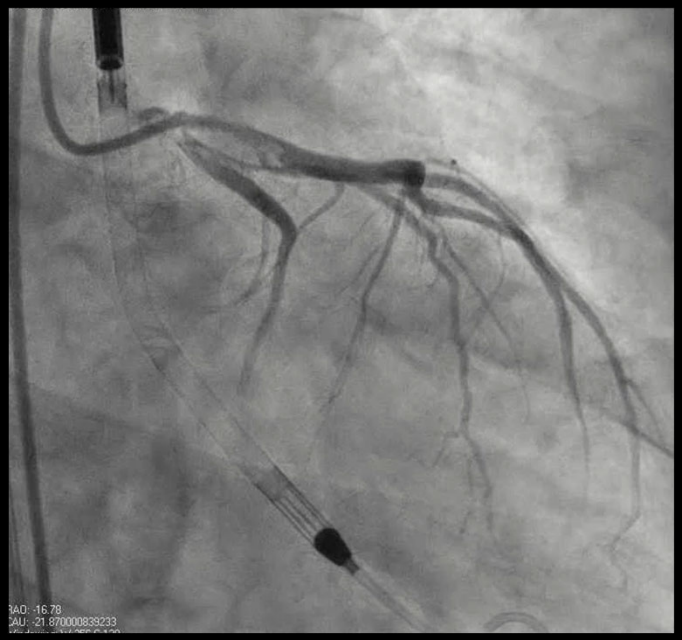
Case 2

60 yo man otherwise healthy and fit presented with anterior STEMI, trop 400 and cardiogenic shock





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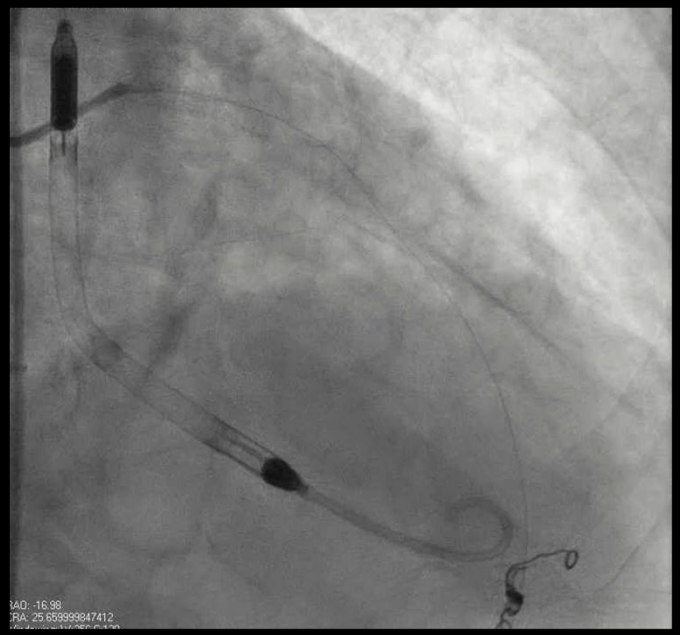

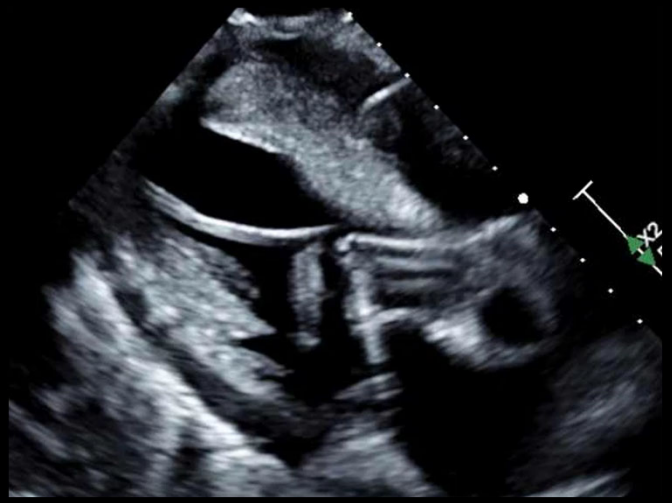

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Case 2 continued

- underwent Impella and PCI to LAD, admitted to ICU
- Unfortunately, he continued to require Vaso, Levo and inotropes.
- He also had hemolysis.

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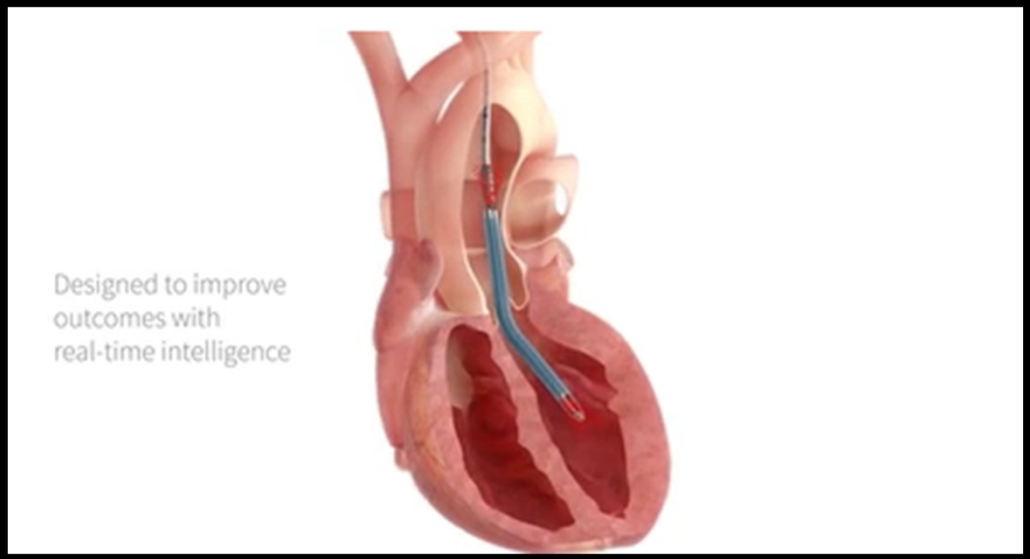
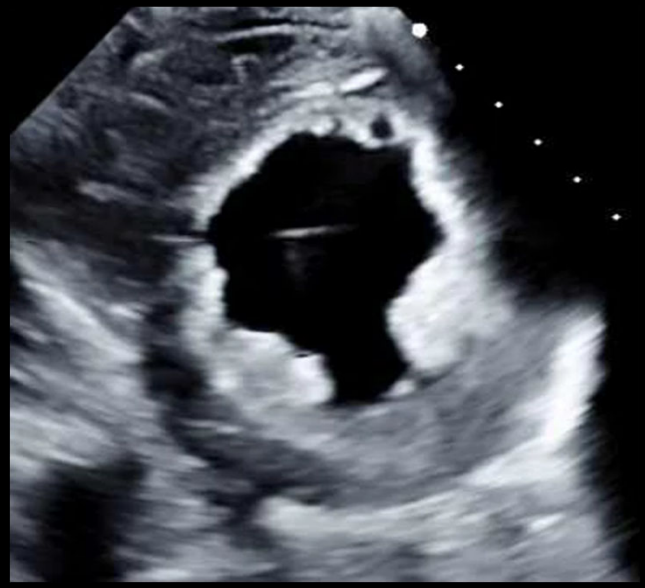
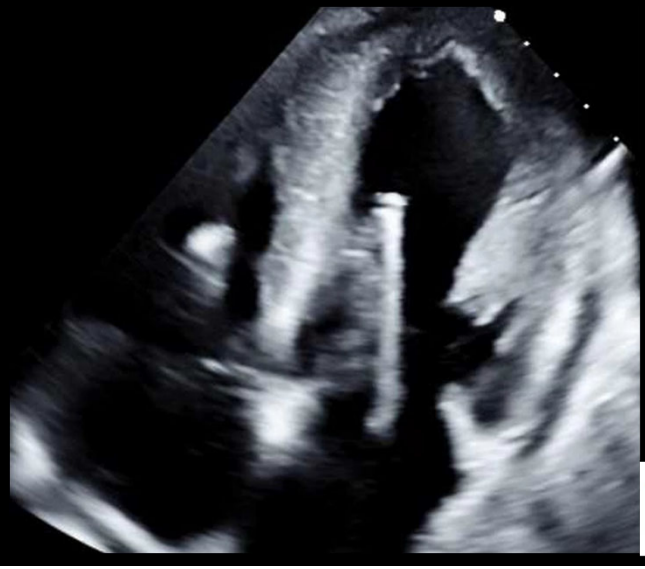




Impella 5.5 was placed→ hemodynamically improved and his Levophed, vasopressin, dobutamine and milrinone were all weaned off successfully over the following 2 weeks.



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Awake Patient = Alive Patient

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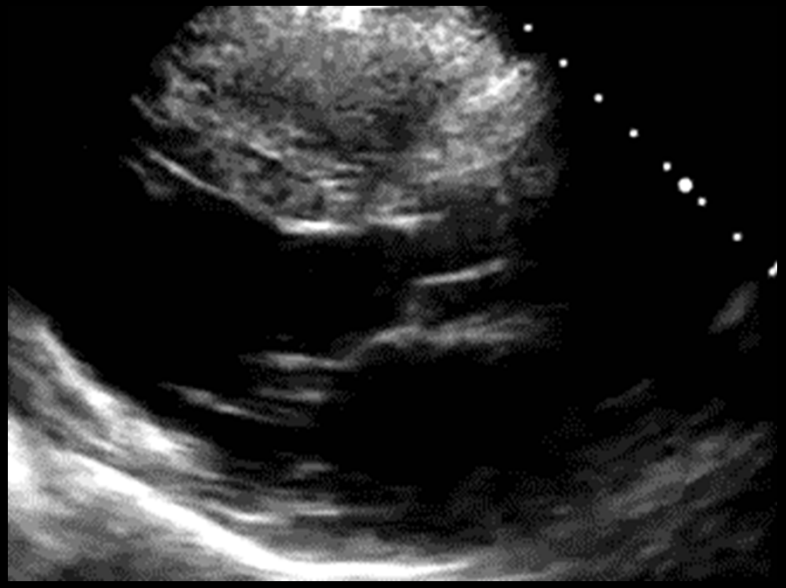
Case 3

- 65 yo lady otherwise healthy was found by her husband gurgling and unresponsive, found to have ventricular fibrillation, she was defibrillated and intubated. Lactic acid was 8.5 at that time
- Transferred to Norton, during her course she was still requiring Epi, Levo and Vaso and remains in shock with lactic acidosis with EF on echo of 15%.
- Cath No significant coronary disease, but severely elevated filling pressures with cardiogenic shock
- Impella and Swan placed ….



Had Impella CP placed, was able to wean off her pressors, lactic acid normalized and shock resolved, Impella was eventually removed and the patient was extubated successfully

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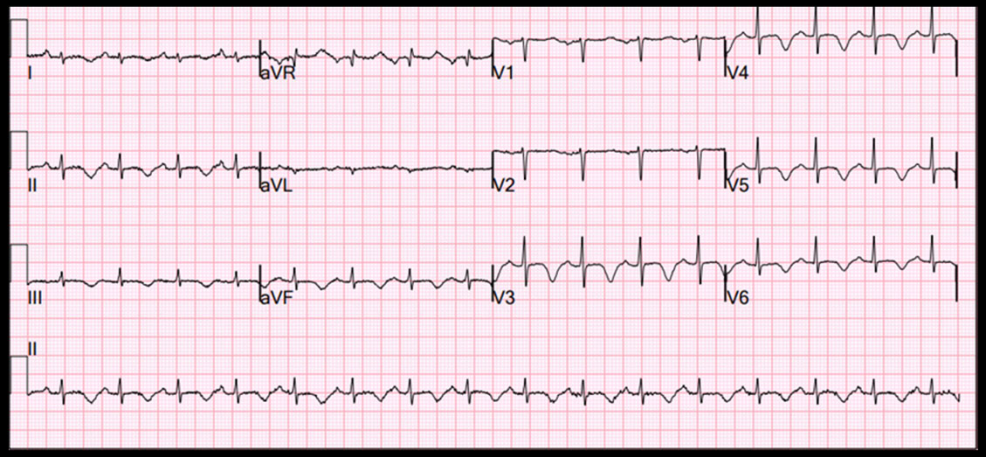
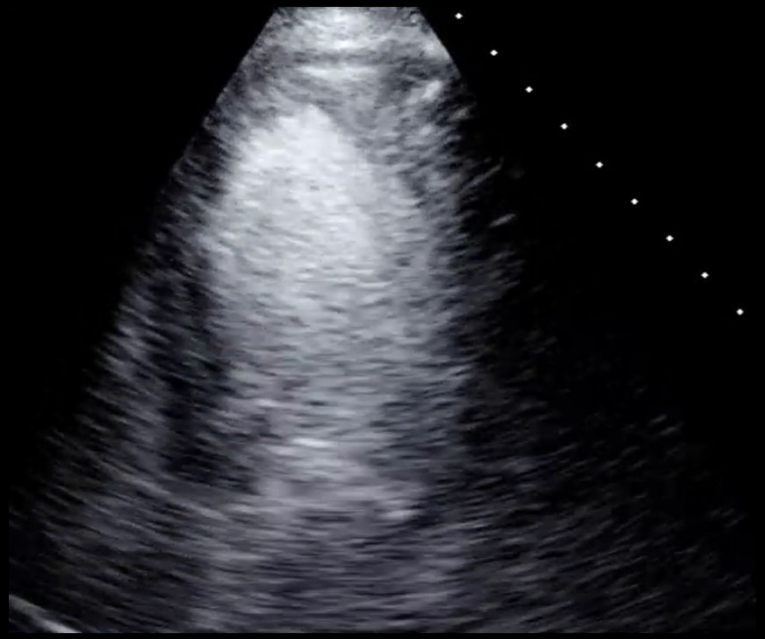
- She had cardiac MRI and endomyocardial biopsy all of which were inconclusive, eventually had an ICD placed for secondary prevention





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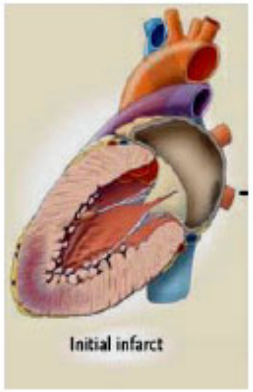
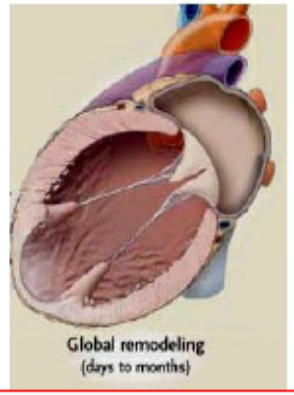
For usual adult 70 kg BSA 1.8 m2
LVEDV 100 ml SV 60 ml EF 60%
HR 80 bpm CO >4L/min and CI >2.2



Acute MI
Cardiogenic Shock

AMI →EF 25%, LVEDV 100 ml SV 25 ml
HR → 150 bpm CO 3.8 L/min CI 2.1



Advanced HF
Cardiogenic Shock

Chronic dilated LV
LVEDV 200 ml EF 25%
SV 50 ml HR 80 bpm CO 4 CI 2.2

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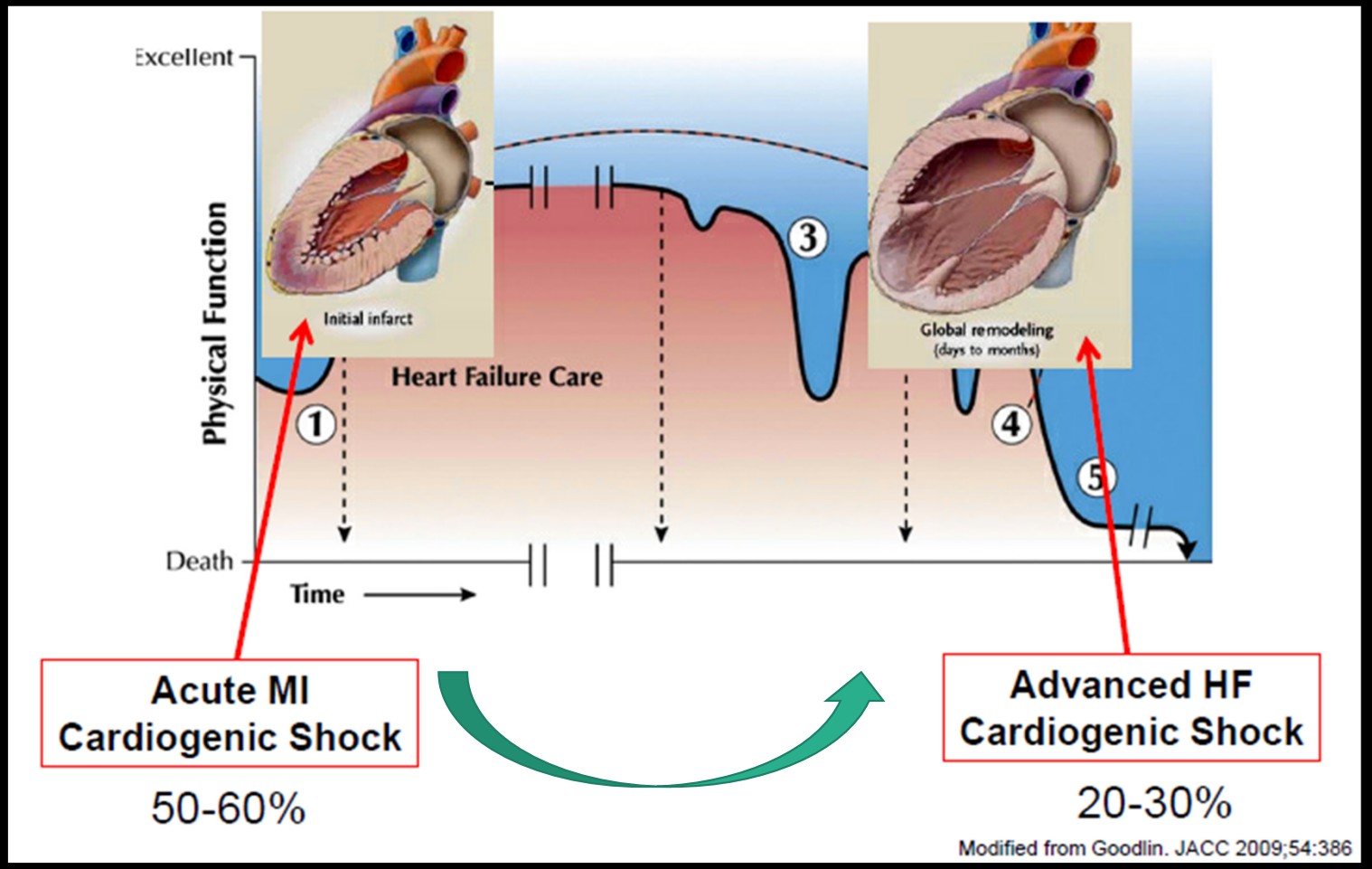
We must centralize and tailor expertise for cardiogenic shock management



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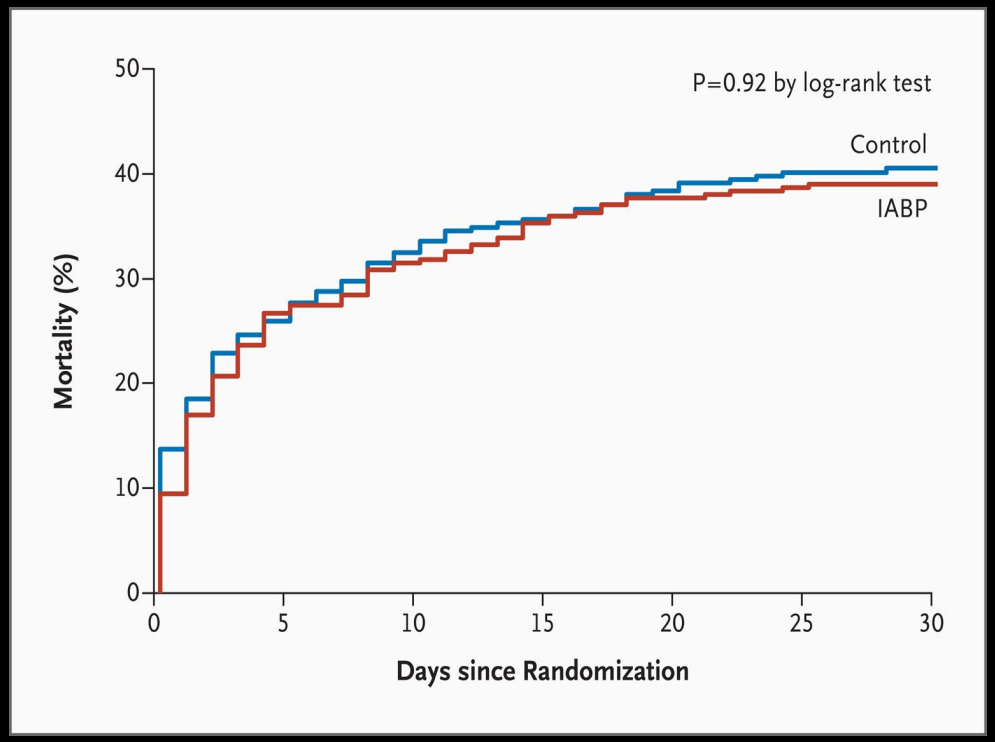
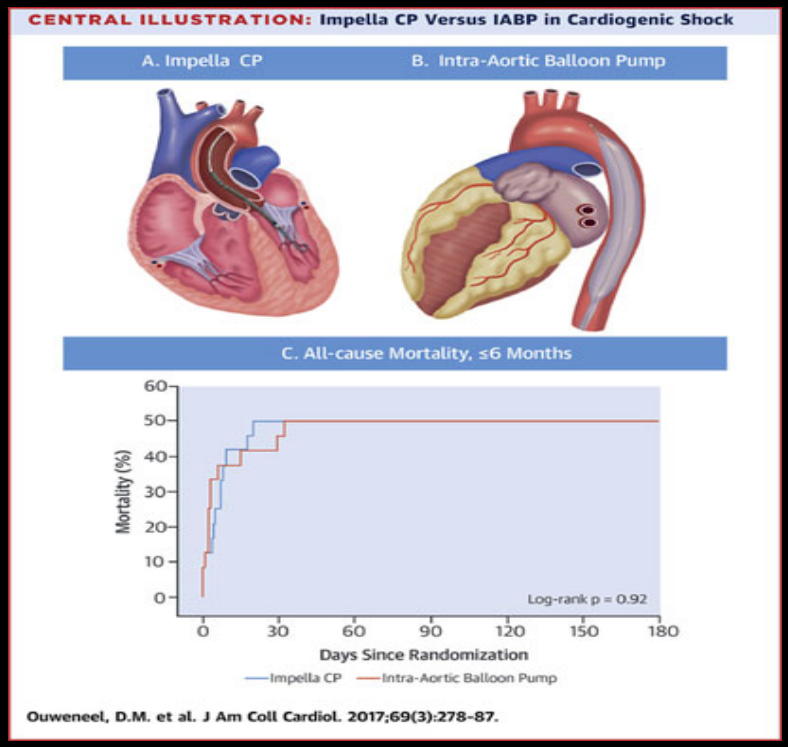
Which Device is better





Thiele H et al. IABP for MI with CS *NEJM 2012*

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It is not about the pump but rather
Process People and Place

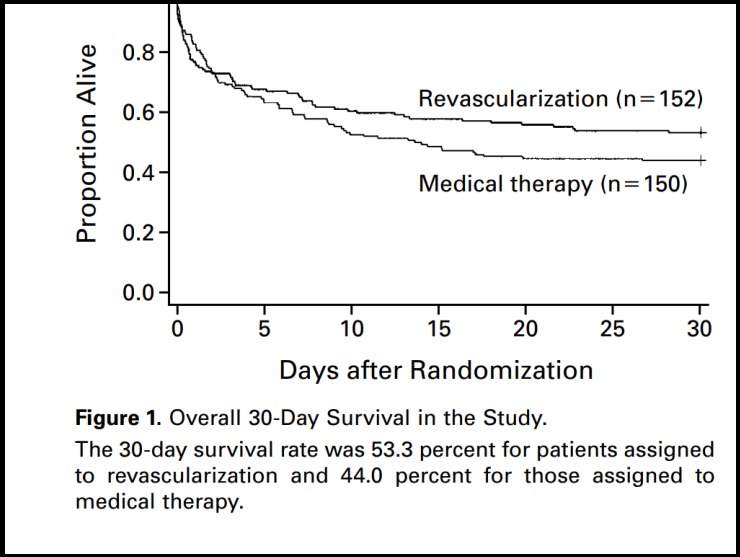
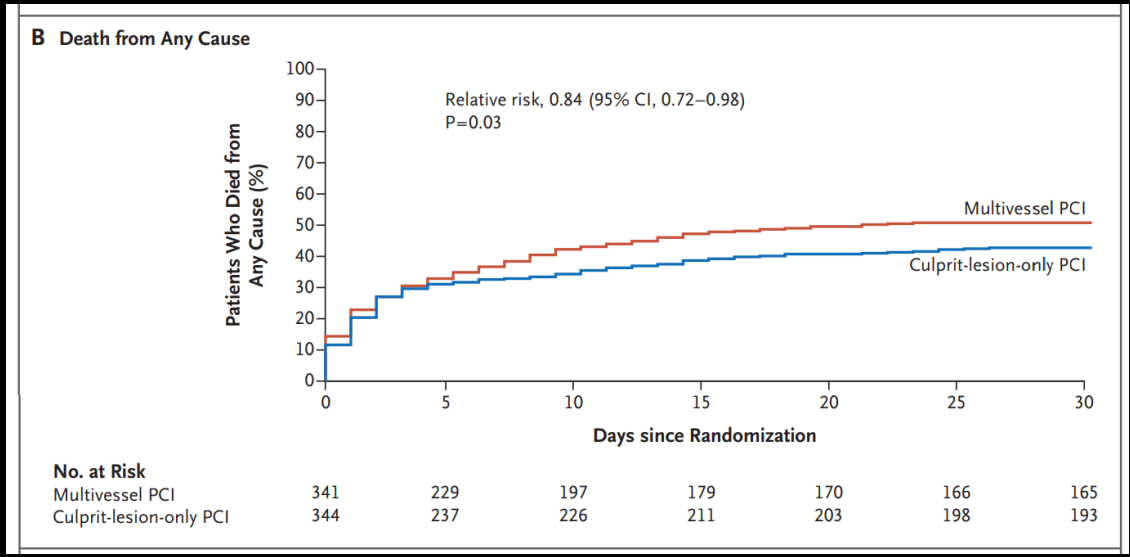


Figure 1. Overall 30-Day Survival in the Study.
The 30-day survival rate was 53.3 percent for patients assigned to revascularization and 44.0 percent for those assigned to medical therapy.



Except for culprit revascularization in MI shock, no specific therapy has improved outcome but rather the rapid protocol driven escalation that improves outcomes

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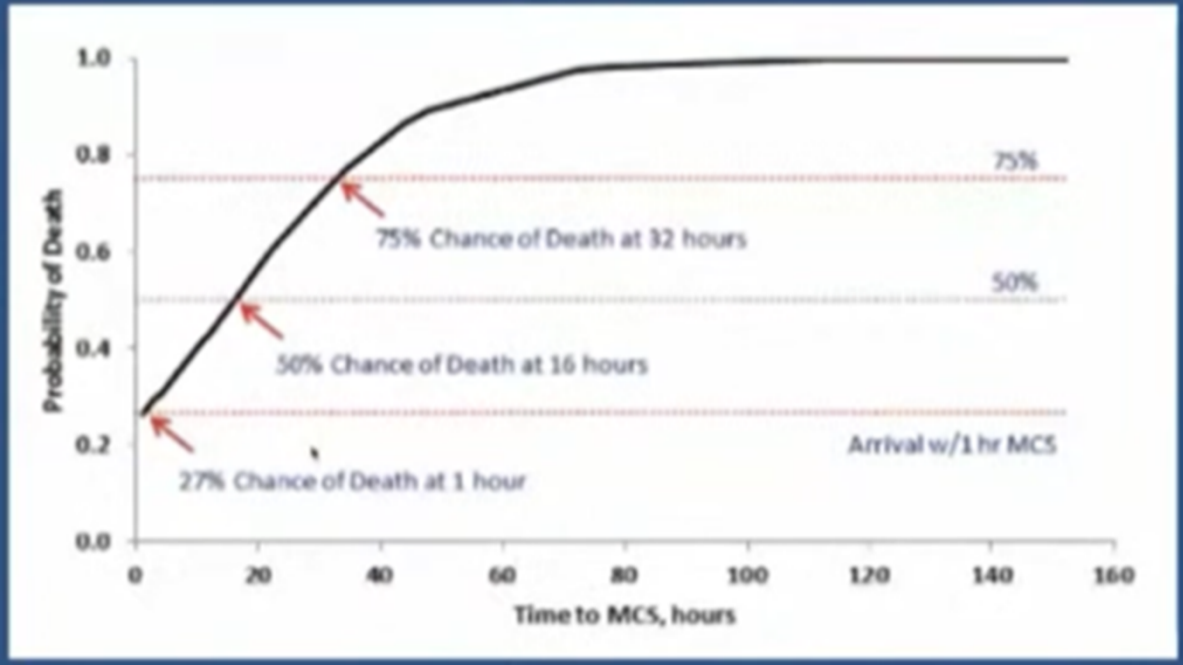
Time factor



Tehrani et al. Standardized Team-Based Care for Cardiogenic Shock. J Am Coll Cardiol. 2019 Apr 9;73(13):1659-1669. doi: 10.1016/j.jacc.2018.12.084.

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In AMI CS: Pump First, PCI Second

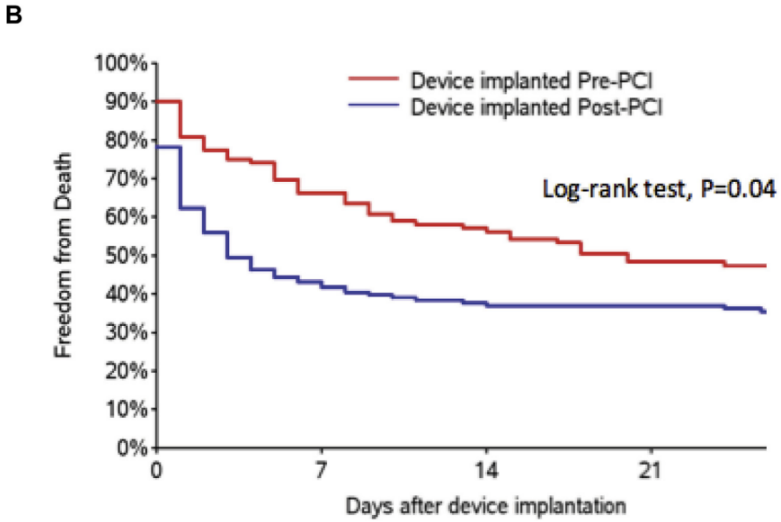

Figure 1. *(A)* Hemodynamic and clinical effects of Impella demonstrated in previous studies. Use of MCS results in numerous beneficial effects including increasing cardiac power output, unloading of the left ventricle, and increasing coronary flow. *(B)* The Kaplan-Meier curve for Freedom from Death (to 30 days) by device implanted pre/post-PCI. The separation of the Kaplan-Meier curves occurs very early post-PCI reinforcing that early hemodynamic support is a key determinant in clinical outcomes.

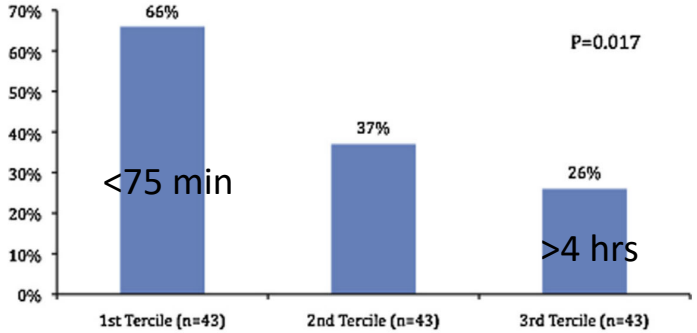

Figure 3. Inhospital survival rates as a function of shock onset to MCS implantation.

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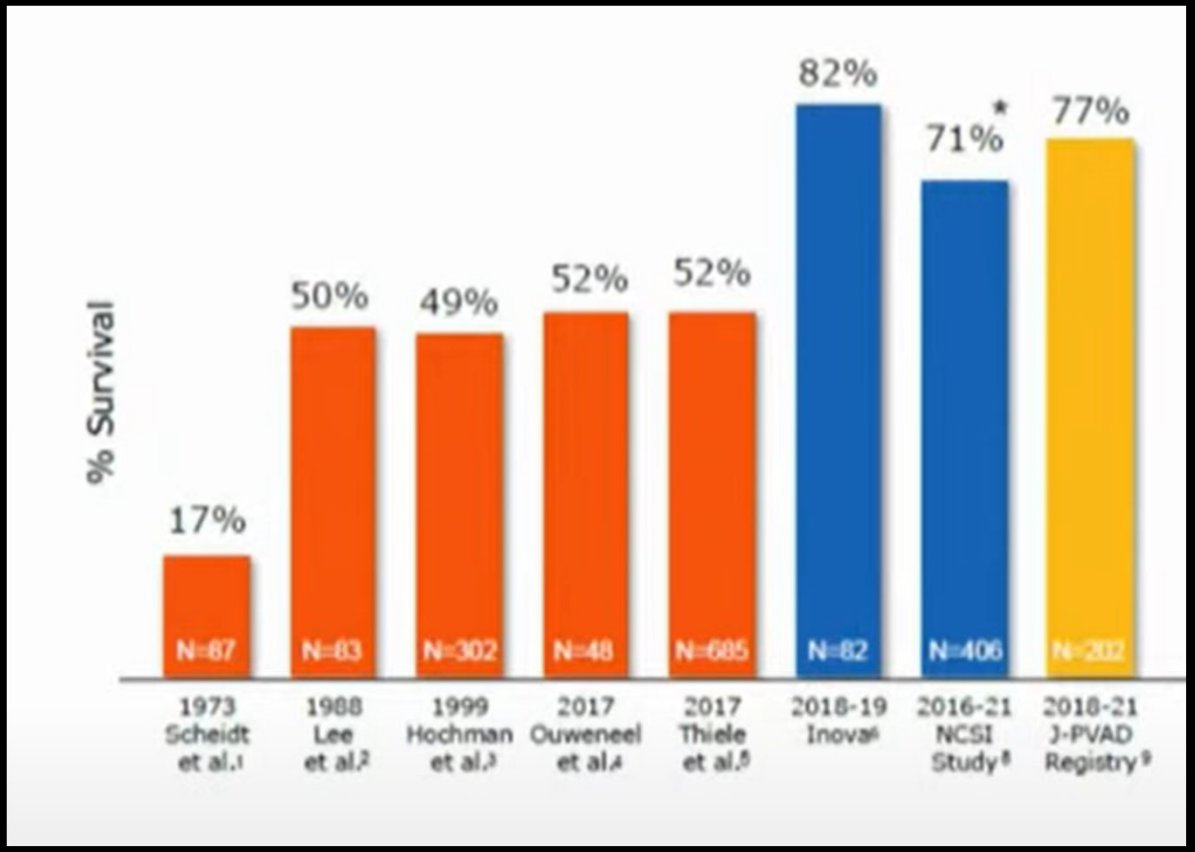
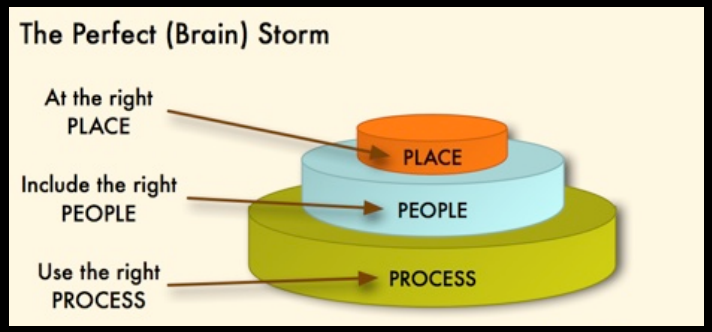
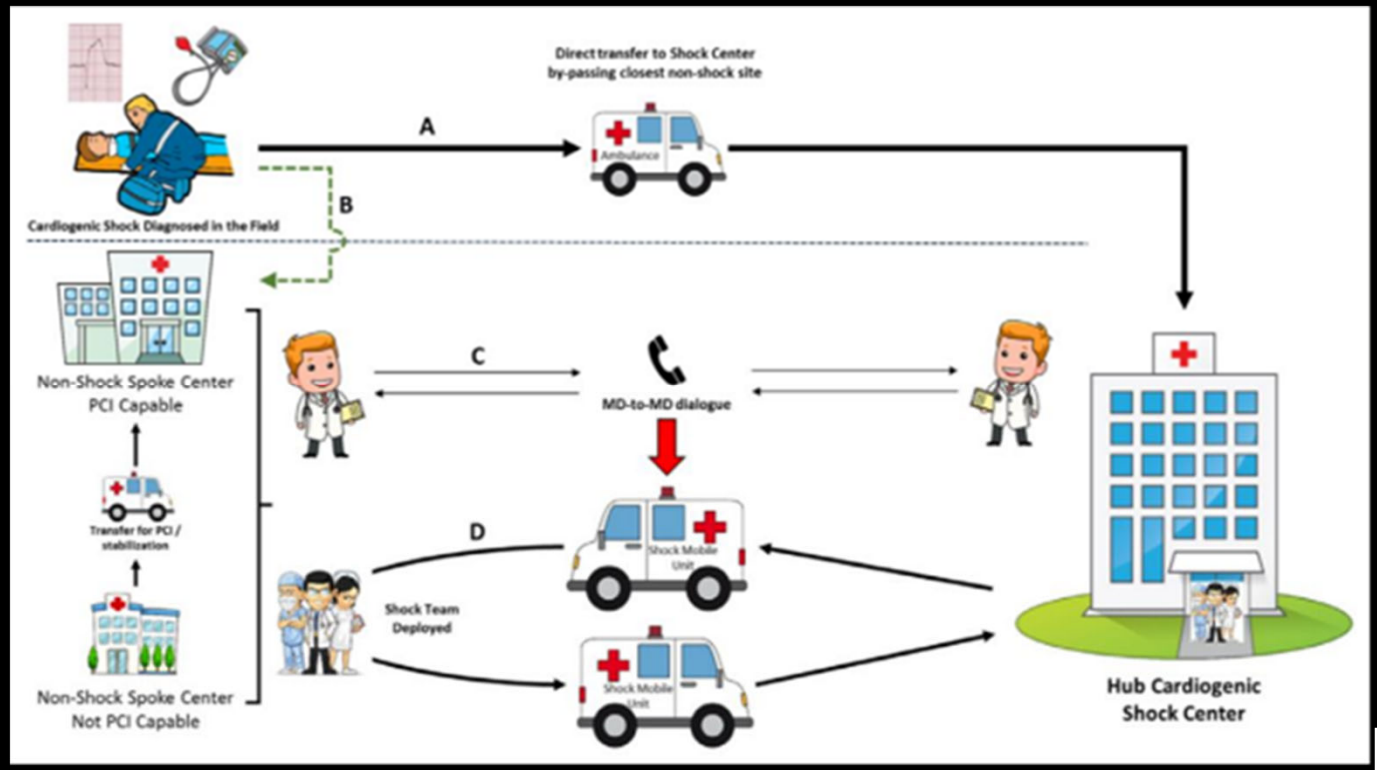
Best Practice Protocol Includes



- Identify cardiogenic shock early and Impella Pre-PCI < 90 min
- Aggressive down titration of inotropes and pressors
- Systematic use of RHC
- Identify RV failure early and plan support appropriately
- Identify inadequate LV support and escalate



Outcome Is As Good As Everyone On The Team Is



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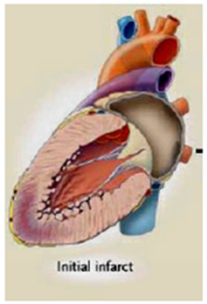
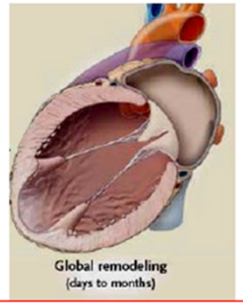
Cardiogenic shock = TEAM APPROACH

Who do you want on *your* Shock Team?



Emergency providers

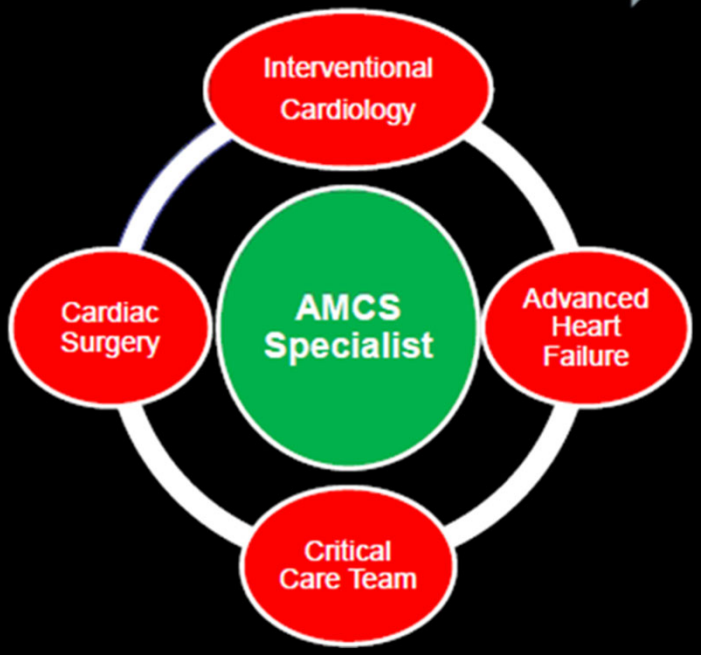


Acute MI Cardiogenic Shock

1. Interventional Cardiologist
2. Cardiac Surgeon
3. Critical Care / Intensivist (MD)
4. Advanced HF Specialist
5. Critical Care Nursing Team
6. Perfusion Team
7. Respiratory Specialists
8. Physical and Occupational Therapy
9. Palliative Care

Advanced HF Cardiogenic Shock

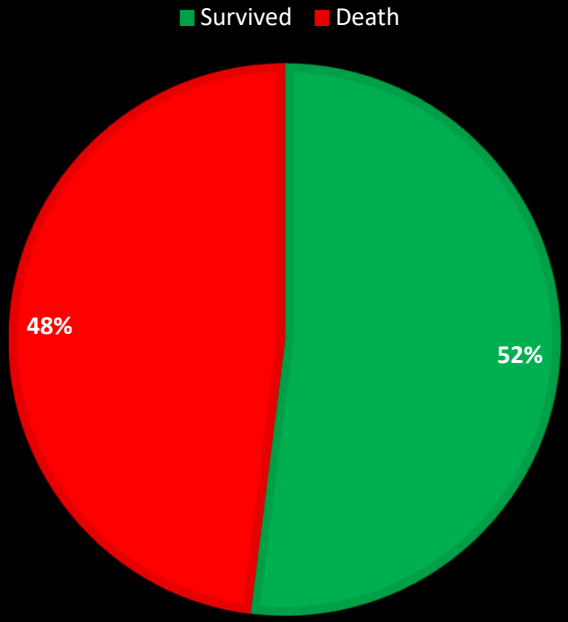
1. Advanced HF Specialist
2. Interventional Cardiologist
3. Cardiac Surgeon
4. Critical Care / Intensivist (MD)
5. Critical Care Nursing Team
6. Palliative Care
7. Perfusion Team
8. Respiratory Specialists
9. Physical and Occupational Therapy



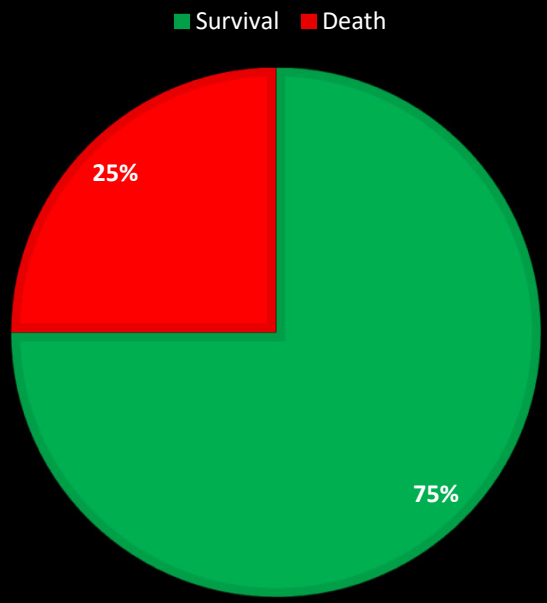
NORTON HEALTHCARE

Norton Healthcare Cardiogenic Shock

SHOCK PRIOR TO PROTOCOL JAN 20'-AUG 2022



SHOCK AFTER IMPLEMENTATION OF PROTOCOL AUG 22'-JUN 23'



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Challenges

Fractured care

- No formal process for multidisciplinary evaluation of patients

- Suboptimal interdisciplinary communication

Late detection

- Patients often too sick/too late

to optimally benefit from

therapies

Impaired access to care

- Delays in transfer of patients into system

- Late recognition of CS in patients

Variations in care

- Inconsistent timing and employment of therapies

- Variable monitoring and reassessment strategies

NORTON HEALTHCARE

Success Factors

- Early recognition
- Frequent hemodynamic, and clinical assessment
- Organ reperfusion and Coronary revascularization
- Ownership (leadership), culture shift toward team mentality

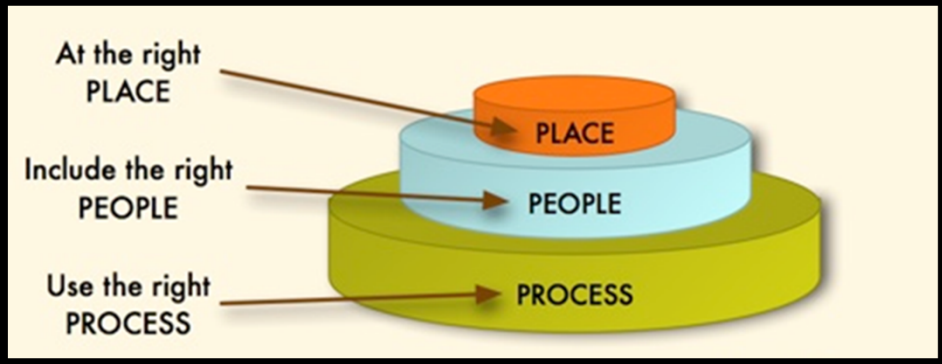
NCSI Best practices

- Mechanical Support (MCS) prior to escalating doses of inotropes
- Use of RHC to guide clinical decision making
- MCS pre-PCI
- Door to Support <90 minutes

NORTON HEALTHCARE

- *Nothing in life is more wonderful than faith—the one great moving force which we can neither weigh in the balance nor test in the crucible*

Sir William Osler



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END