CARDIOGENIC SHOCK

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Definition

- HYPOTENSION REFRACTORY TO VOLUME RESUSCITATION WITH FEATURES OF END-ORGAN HYPOPERFUSION REQUIRING PHARMACOLOGICAL OR MECHANICAL INTERVENTION.
  ACUTE MYOCARDIAL INFARCTION (MI) ACCOUNTS FOR 81% OF PATIENT IN CS
  CARDIOGENIC SHOCK (CS) IS A COMMON CAUSE OF MORTALITY, AND MANAGEMENT REMAINS CHALLENGING DESPITE ADVANCES IN THERAPEUTIC OPTIONS.

CS IS CAUSED BY SEVERE IMPAIRMENT OF MYOCARDIAL PERFORMANCE THAT RESULTS IN DIMINISHED CARDIAC OUTPUT, END-ORGAN HYPOPERFUSION, AND HYPOXIA.

Cardiogenic shock

- **CS COMPLICATES 5% TO 10% OF CASES OF ACUTE MI AND IS THE LEADING CAUSE OF DEATH AFTER MI.**

  ST-SEGMENT–ELEVATION MYOCARDIAL INFARCTION (STEMI) IS ASSOCIATED WITH A 2-FOLD INCREASED RISK FOR DEVELOPMENT OF CS COMPARED WITH NON–ST-SEGMENT–ELEVATION MYOCARDIAL INFARCTION (NSTEMI).

  PATIENTS WITH NSTEMI-ASSOCIATED CS ARE LESS LIKELY TO UNDERGO EARLY CARDIAC CATHETERIZATION, DELAYING PCI AND/OR CORONARY ARTERY BYPASS GRAFT AND INCREASING THE RISK OF MORTALITY COMPARED WITH PATIENTS WITH STEMI-ASSOCIATED

Cardiogenic shock

HIGHER INCIDENCES OF CS ARE OBSERVED IN WOMEN, ASIAN/PACIFIC, AND PATIENTS AGED >75 YEARS.

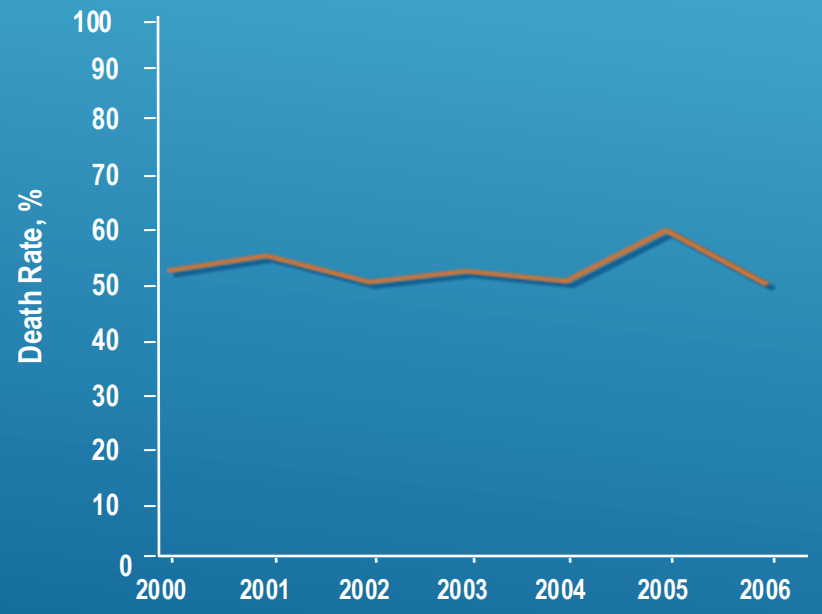
THE INCIDENCE OF CS HAS INCREASED IN RECENT YEARS, DUE TO IMPROVED DIAGNOSIS AND BETTER ACCESS TO CARE .WHILE THE IN-HOSPITAL MORTALITY HAS IMPROVED, THE 6- TO 12-MONTH MORTALITY IN CARDIOGENIC SHOCK HAS REMAINED UNCHANGED AT ≈50% OVER THE PAST 2 DECADES.

Cardiogenic shock

CARDIOGENIC SHOCK REMAINS LEADING CAUSE OF MORTALITY IN AMI

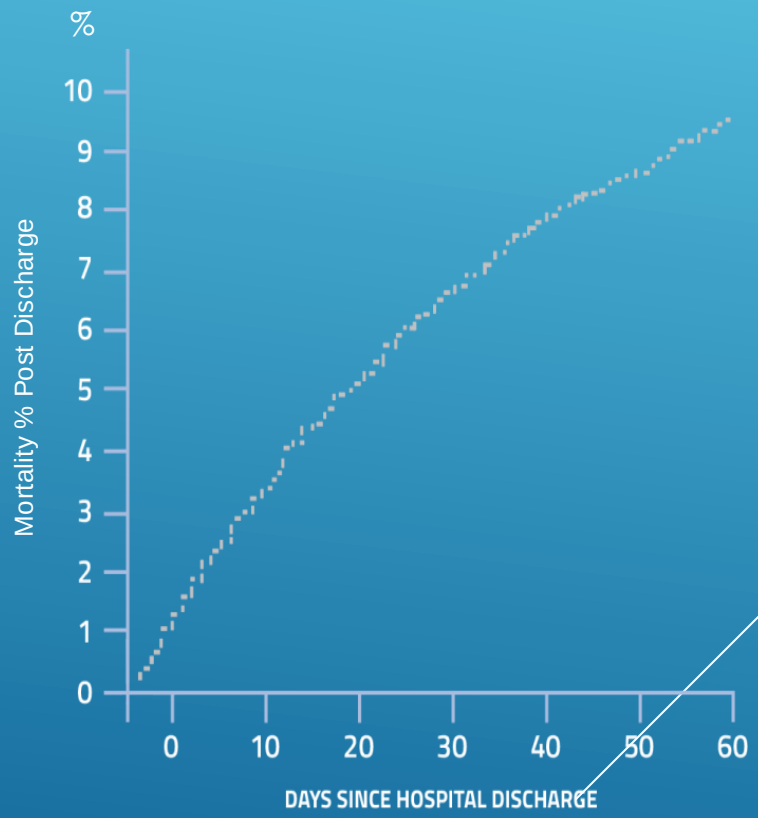
High In-Hospital Mortality During AMI Cardiogenic Shock[1]

N = 23,696

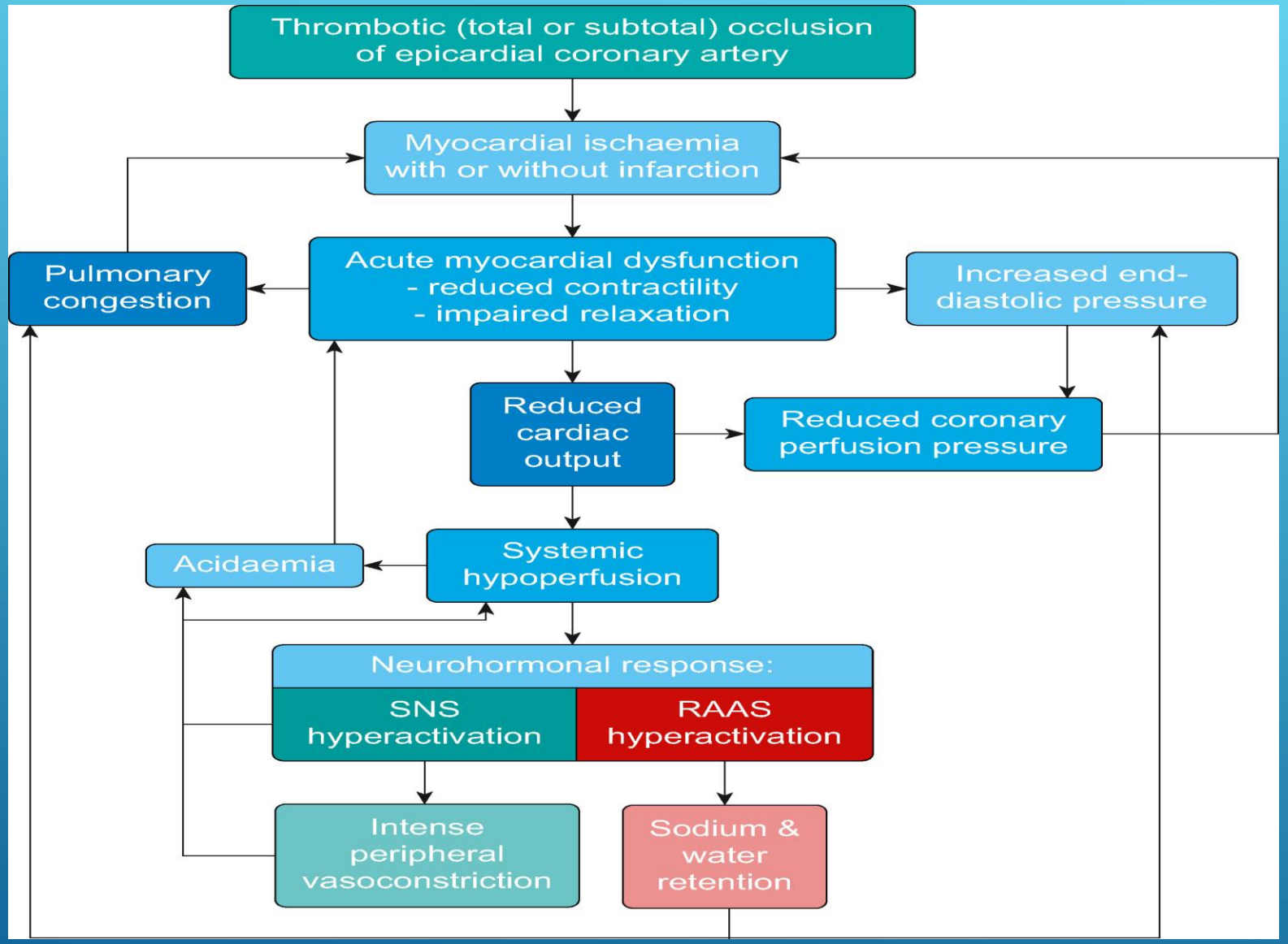


… and Ongoing Hazard Post Discharge after AMI Cardiogenic Shock[2]

N = 112,668



1. Jeger, et al. Ann Intern Med. 2008
2. Shah, et al. JACC 2016 NCDR Registry


Thrombotic (total or subtotal) occlusion of epicardial coronary artery
Myocardial ischaemia with or without infarction
Pulmonary congestion
Acute myocardial dysfunction - reduced contractility - impaired relaxation
Increased end-diastolic pressure
Reduced cardiac output
Reduced coronary perfusion pressure
Acidaemia
Systemic hypoperfusion
Neurohormonal response:
SNS hyperactivation
RAAS hyperactivation
Intense peripheral vasoconstriction
Sodium & water retention

Shock Clinical Criteria*

SBP <90 mm Hg for >30 min:
a. Or mean BP <60 mm Hg for >30 min
b. Or requirement of vasopressors to maintain systolic BP ≥90 mm Hg or mean BP ≥60 mm Hg
Hypoperfusion defined by:
c. Decreased mentation
d. Cold extremities, livedo reticularis
e. Urine output <30 mL/h
f. Lactate >2 mmol/L

Suggested Shock Hemodynamic Criteria*

1. SBP <90 mm Hg or mean BP <60 mm Hg
2. Cardiac index <2.2 L/min/m^2
3. Pulmonary capillary wedge pressure >15 mm Hg
4. Other hemodynamic considerations
a. Cardiac power output ([CO x MAP]/451) <0.6 W
b. Shock index (HR/systolic BP) >1.0
c. RV shock
i. Pulmonary artery pulse index [(PASP-PADP)/CVP] <1.0
i. CVP >15 mm Hg
i. CVP-PCW >0.6

BP indicates blood pressure; CO, cardiac output; CVP, central venous pressure; HR, heart rate; MAP, mean arterial pressure; PADP, pulmonary artery diastolic pressure; PASP, pulmonary artery systolic pressure; PCW, pulmonary capillary wedge; RV, right ventricular; and SBP, systolic blood pressure.

*Diagnosis of shock requires ≥1 criteria to be present along with cardiac index <2.0 L/min/m^2 and SBP <90 mm Hg.

Society for Cardiovascular Angiography and Interventions (SCAI) Cardiogenic Shock Criteria

Stage	Bedside Findings	Selected Laboratory Markers	Hemodynamics
A: At risk --Normotensive --Normal perfusion --Cause for risk for shock such as large myocardial infarction or HF	--Normal venous pressure --Clear lungs --Warm extremities --Strong palpable pulses --Normal mentation	--Normal renal function --Normal lactate	--SBP >100 mm Hg --Hemodynamics: Normal

Society for Cardiovascular Angiography and Interventions (SCAI) Cardiogenic Shock Criteria (con't.)

B: Beginning shock ("pre-shock") --Hypotension --Normal perfusion	--Elevated venous pressure --Rales present --Warm extremities --Strong pulses --Normal mentation	--Preserved renal function --Normal lactate --Elevated BNP	a) SBP <90 mm Hg b) MAP <60 mm Hg or c) >30 mm Hg decrease from baseline SBP --HR >100 bpm --Hemodynamics: CI ≥2.2 L/min/m^2

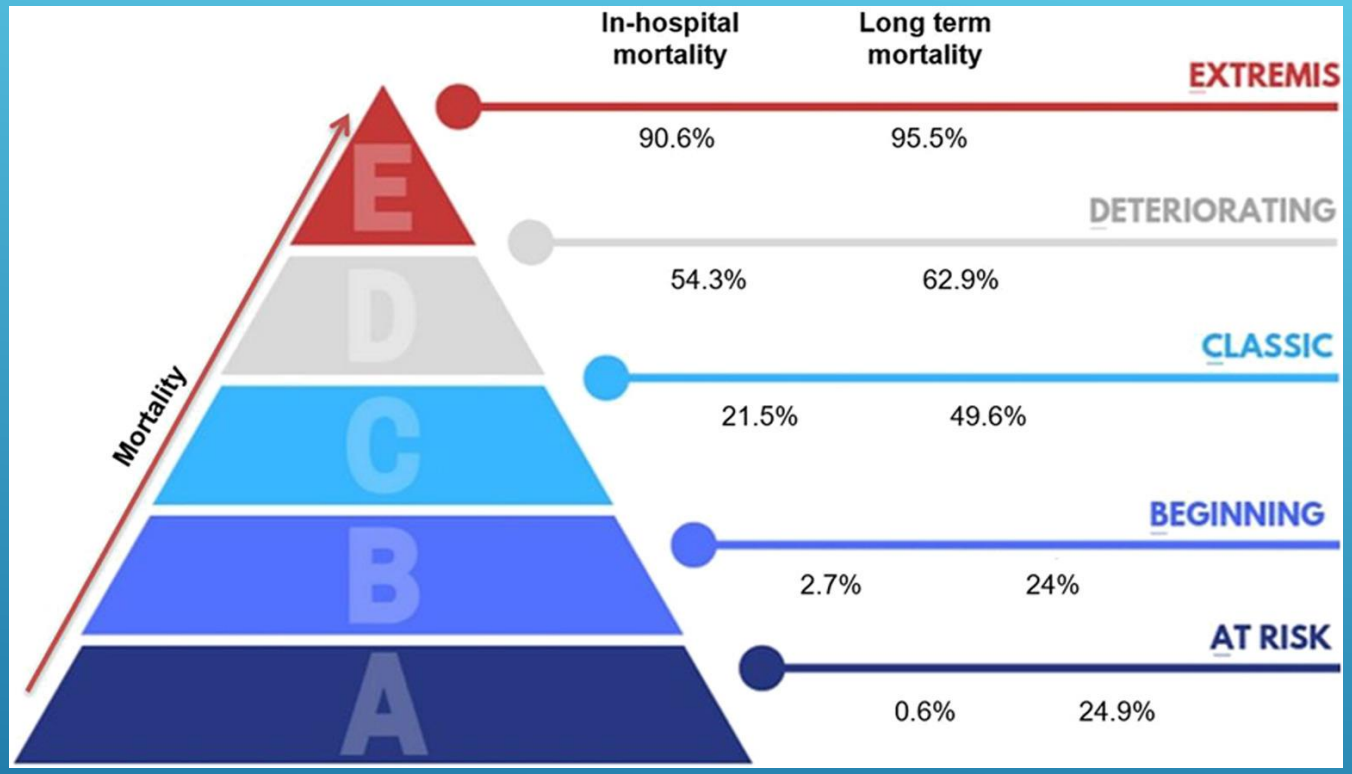
Society for Cardiovascular Angiography and Interventions (SCAI) Cardiogenic Shock Criteria (con't.)

C: Classic cardiogenic shock --Hypotension --Hypoperfusion	--Elevated venous pressure --Rales present --Cold, ashen, livedo --Weak or nonpalpable pulses --Altered mentation --Decreased urine output --Respiratory distress	--Impaired renal function --Increased lactate --Elevated BNP --Increased LFTs --Acidosis	--SBP <90 mm Hg; MAP <60 mm Hg; >30 mm Hg from baseline SBP despite drugs and temporary MCS --HR >100 bpm --Hemodynamics: CI ≤2.2 L/min/m^2; PCW >15 mm Hg; CPO <0.6 W; PAPi <2.0; CVP-PCW >1.0

Society for Cardiovascular Angiography and Interventions (SCAI) Cardiogenic Shock Criteria (con't.)

D: Deteriorating --Worsening hypotension --Worsening hypoperfusion	Same as stage C	--Persistent or worsening values of stage C	Escalating use of pressors or MCS to maintain SBP and end-organ perfusion in setting of stage C hemodynamics
E: Extremis --Refractory hypotension --Refractory hypoperfusion	--Cardiac arrest --CPR	--Worsening values of stage C laboratories	--SBP only with resuscitation --PEA --Recurrent VT/VF

BNP indicates brain natriuretic peptide; CI, cardiac index; CPO, cardiac power output; CPR, cardiopulmonary resuscitation; CVP, central venous pressure; HR, heart rate; LFT, liver function test; MAP, mean arterial blood pressure; MCS, mechanical circulatory support; PAPi, pulmonary artery pulsatility index; PCW, pulmonary capillary wedge pressures; PEA, pulseless electrical activity; SBP, systolic blood pressure; VF, ventricular fibrillation; and VT, ventricular tachycardia.


In-hospital mortality
Long term mortality
EXTREMIS
90.6%
95.5%
DETERIORATING
54.3%
62.9%
CLASSIC
21.5%
49.6%
BEGINNING
2.7%
24%
AT RISK
0.6%
24.9%
Mortality
E
D
C
B
A

Intravenous Inotropic Agents Used in the Management of HF

Inotropic Agent	Dose (mcg/kg)		Drug Kinetics and Metabolism	Effects				Adverse Effects	Special Considerations
	Bolus	Infusion (/min)		CO	HR	SVR	PVR		
Adrenergic agonists									
Dopamine	NA	5–10	$t_{1/2}$: 2–20 min	↑	↑	↔	↔	T, HA, N, tissue necrosis	Caution: MAO-I
	NA	10–15	R, H, P	↑	↑	↑	↔		
Dobutamine	NA	2.5–20	$t_{1/2}$: 2–3 min H	↑	↑	↔	↔	↑/↓BP, HA, T, N, F, hypersensitivity	Caution: MAO-I; CI: sulfite allergy

Intravenous Inotropic Agents Used in the Management of HF (con't.)

Vasopressors									
Epinephrine	NR	5–15 mcg/min	$t_{1/2}$: 2–3 min	↑	↑	↑ (↓)	↔	HA, T	Caution: MAO-I
		15–20 mcg/min	$t_{1/2}$: 2–3 min	↑	↑↑	↑↑	↔	HA, T,	Caution: MAO-I
Norepinephrine	NR	0.5–30 mcg/min	$t_{1/2}$: 2.5 min	↔	↑	↑↑	↔	↓ HR, tissue necrosis	Caution: MAO-I

BP indicates blood pressure; CI, contraindication; CO, cardiac output; F, fever; H, hepatic; HA, headache; HF, heart failure; HR, heart rate; LFT, liver function test; MAO-I, monoamine oxidase inhibitor; N, nausea; NA, not applicable; NR, not recommended; P, plasma; PDE, phosphodiesterase; PVR, pulmonary vascular resistance; R, renal; SVR, systemic vascular resistance; T, tachyarrhythmias; and t1/2, elimination half-life.

Up arrow means increase.
Side arrow means no change.
Down arrow means decrease.
Up/down arrow means either increase or decrease.

Intravenous Inotropic Agents Used in the Management of HF (con't.)

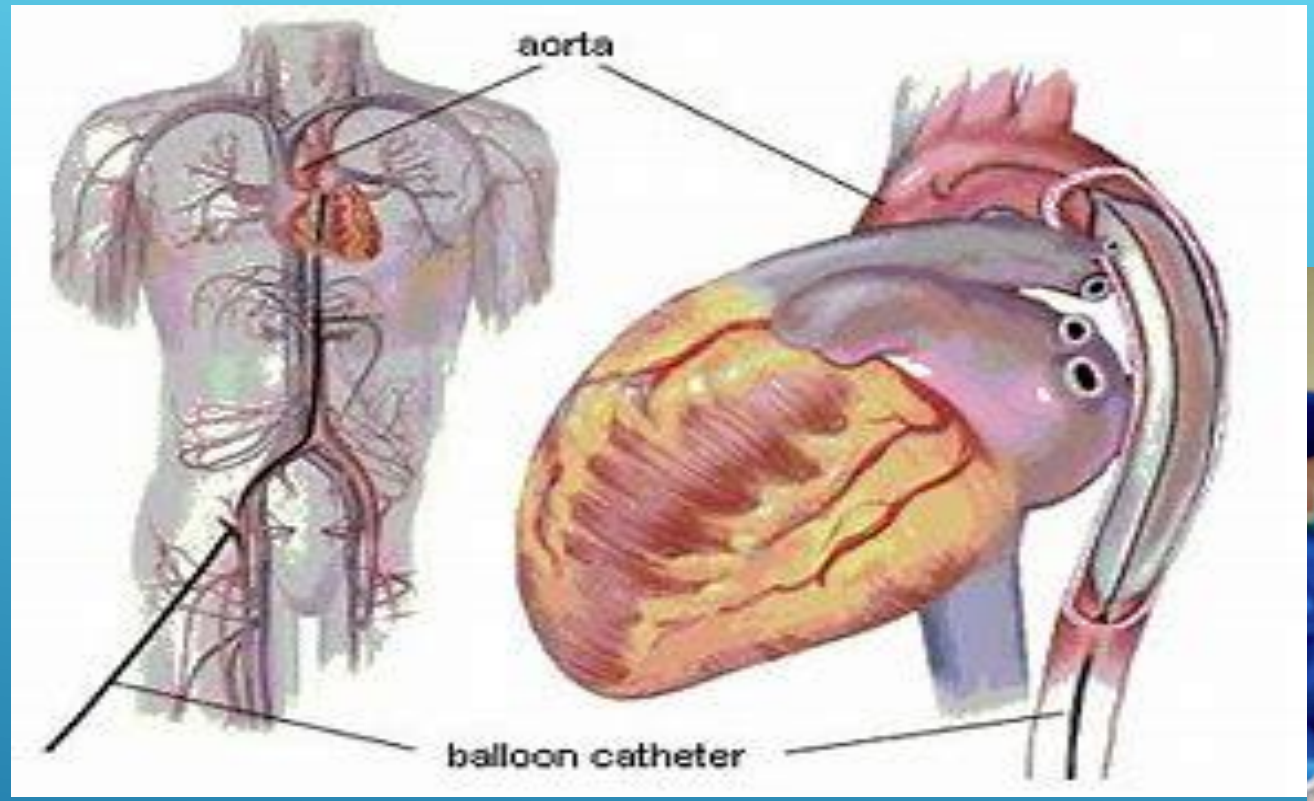
PDE 3 inhibitor									
Milrinone	NR	0.125–0.75	$t_{1/2}$: 2.5 h H	↑	↑	↓	↓	T, ↓BP	Accumulation may occur in setting of renal failure; monitor kidney function and LFTs

VASOPRESSOR/INOTRPES

- While inotropic agents are used widely, mortality is higher with an increased number of prescribed inotropes/vasopressors. Furthermore, catecholamine therapy is associated with significant limitations including arrhythmias, increased myocardial oxygen consumption, and inadequate circulatory support

MCS

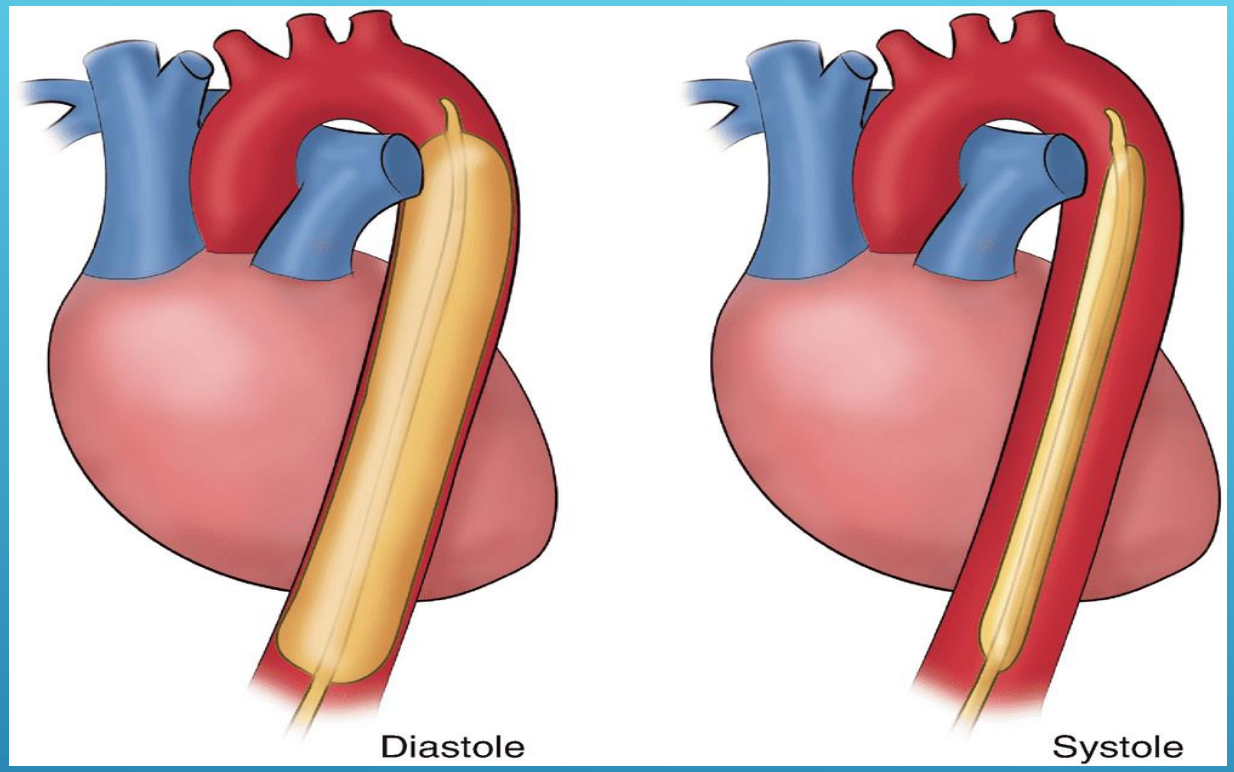
- MCS devices offer significant advantages over vasopressor therapy including substantial cardiovascular support without increased risk of myocardial ischemia and possible decreased myocardial oxygen demand.
- Thus, early use of support devices is an important therapeutic intervention. Options for acute percutaneous MCS include the intra-aortic balloon pump (IABP), axial flow pumps (Impella LP 2.5, Impella CP), left atrial-to-femoral arterial ventricular assist devices (Tandem Heart) and venous-arterial extracorporeal membrane oxygenation (ECMO).

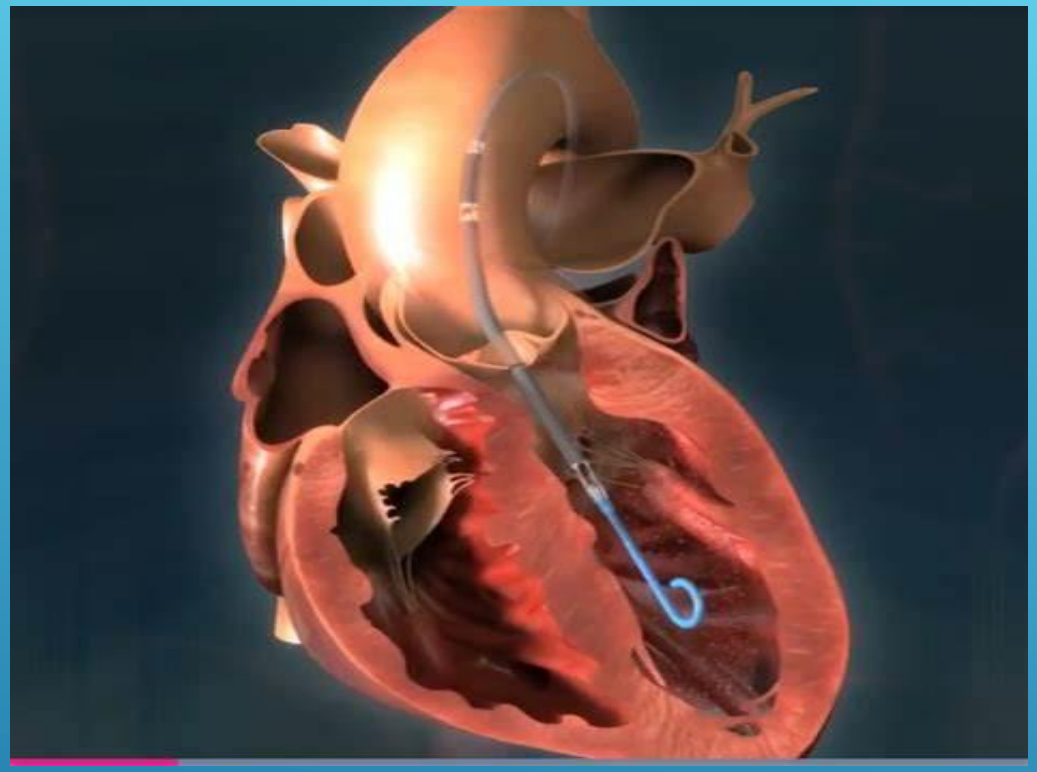

aorta
balloon catheter

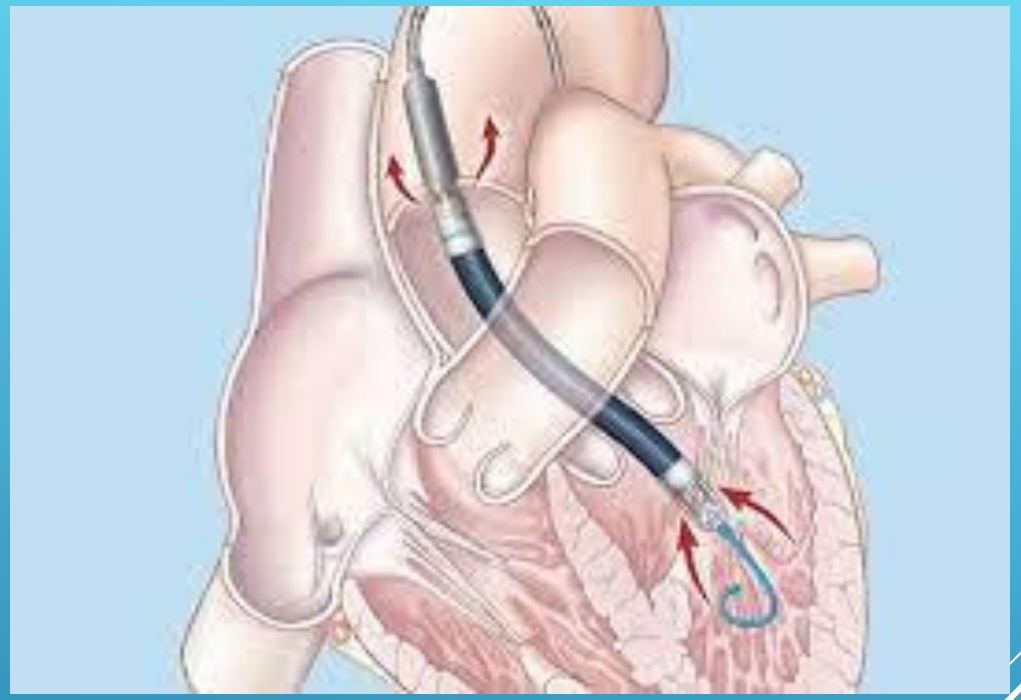

Pressure
84
59
32
68
88
Normal
Direct
Auto
On
Semi Auto

IABP

IABP

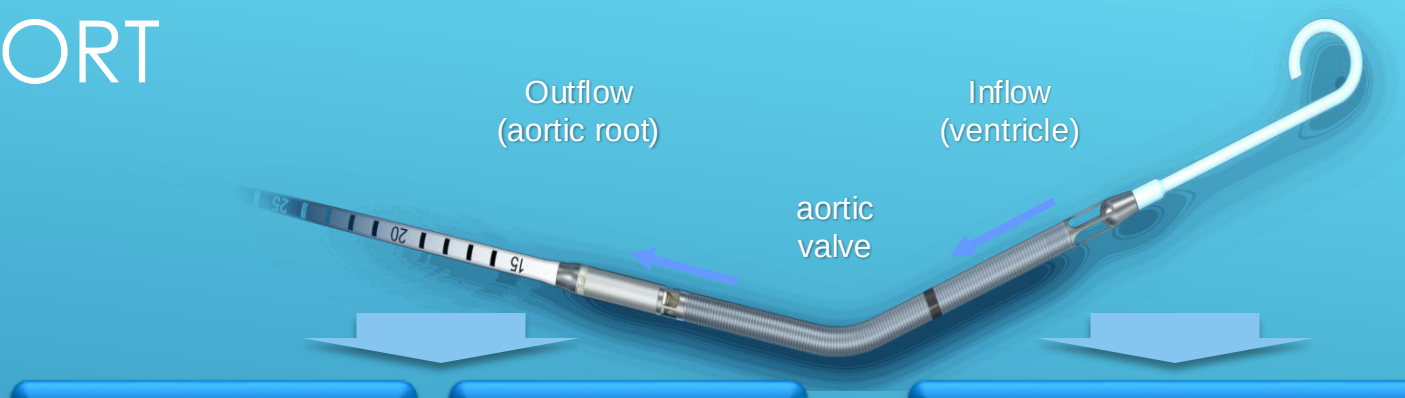






IMPELLA

HEMODYNAMIC EFFECTS OF IMPELLA® SUPPORT



Flow

MAP

LVEDP and LVEDV

Wall Tension

Mechanical Work

Microvascular Resistance

Coronary Perfusion

Cardiac Power Output

End Organ Perfusion

O_2 Supply

O_2 Demand

Unloading to Myocardial Recovery

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Burkhoff D. et al. Am J Physiol Heart Circ 2005
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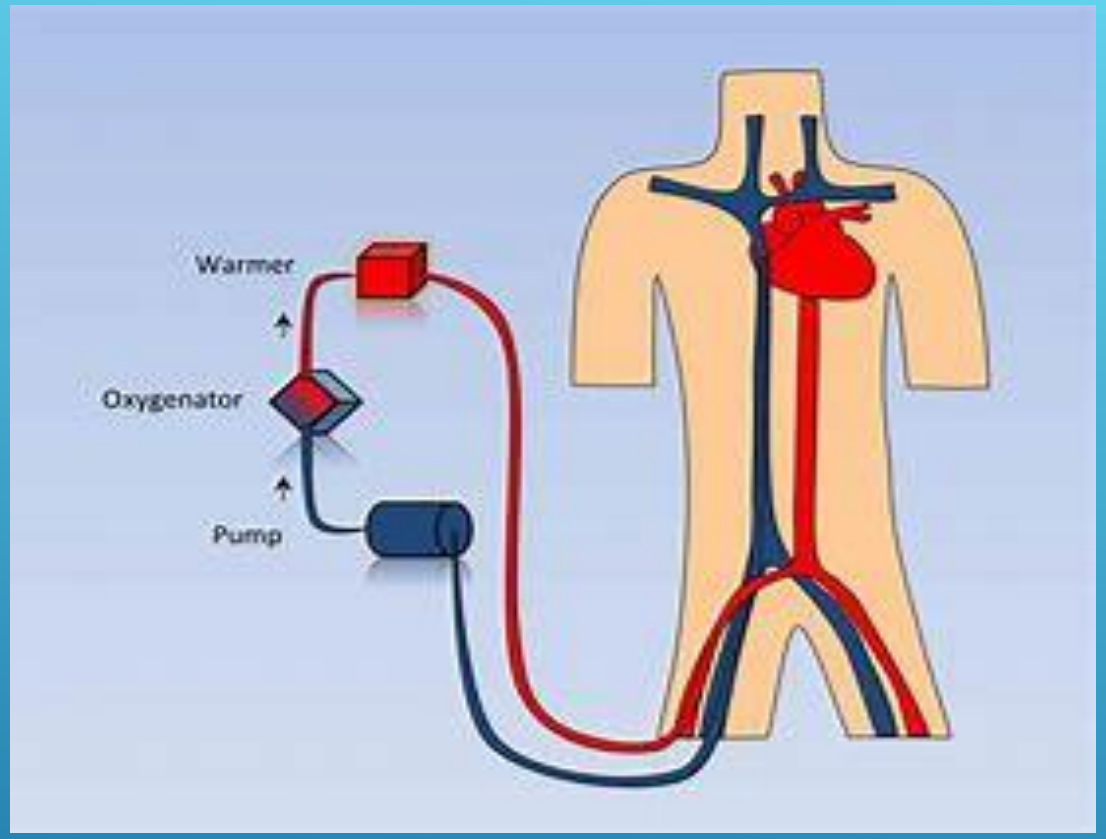
Naidu S. et al. Novel Circulation.2011
Weber DM, et al. Cardiac Interventions Today Supplement Aug/Sep 2009

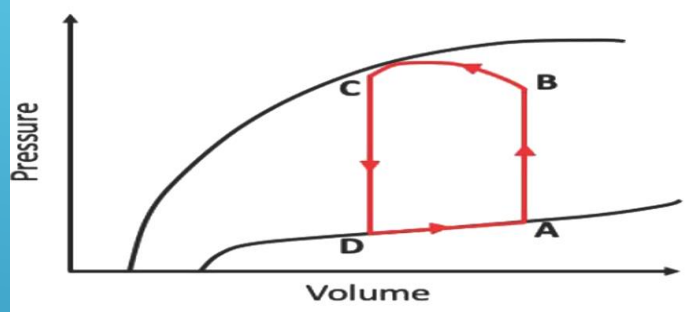
TANDEM HEART

Temporary Devices: Tandem Heart pVAD

- Continuous-flow centrifugal assist device placed extracorporeally
- Cannula in femoral vein through intraatrial septum into LA
- Pump withdraws oxygenated blood from the left atrium, propels it by a magnetically driven impeller through the outflow port
- Blood returns into femoral artery via arterial cannula

ECMO



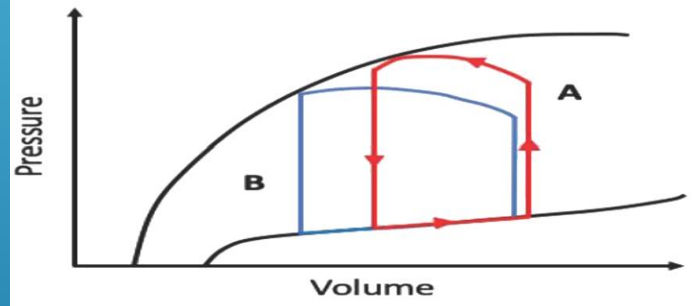


Work = Pressure x Volume

Ventricular "Work" = Area of PV Loop; proportional to O2 demand

Unloading Work = Reducing Area of PV Loop

A = End diastole (mitral valve closure)
B = Aortic valve opening
C = End systole (aortic valve closure)
D = Mitral valve opening

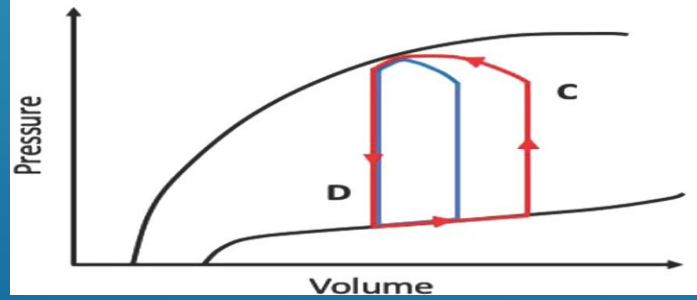


IABP

- **Reduces systolic aortic pressure**
- **Increases stroke volume**

Effect on Cardiac Work = Stroke Volume increase offsets pressure reduction

A = Baseline PV loop
B = After IABP



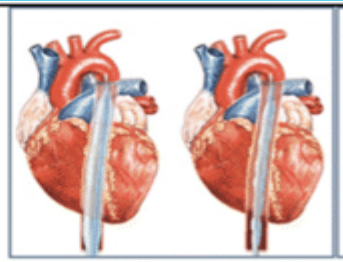
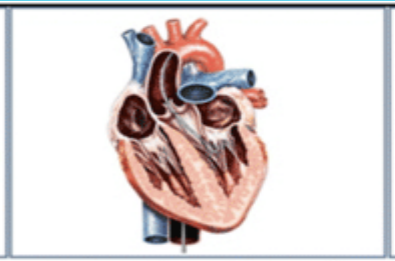
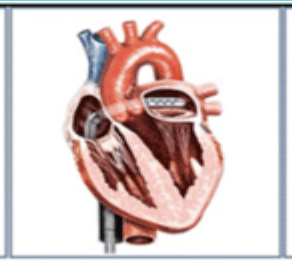
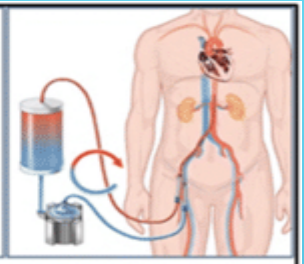
Impella

- **Unloads left ventricle**
- **Reduces diastolic volume**

Effect on Cardiac Work = Volume reduction reduces PV loop area and cardiac work

C = Baseline PV loop
D = After Impella

MCS

	IABP	IMPELLA	TANDEMHEART	VA-ECMO
Cardiac Flow	0.3-0.5 L/ min	1-5L/ min (Impella 2.5, Impella CP, Impella 5)	2.5-5 L/ min	3-7 L-min
Mechanism	Aorta	LV → AO	LA → AO	RA → AO
Maximum implant days	Weeks	7 days	14 days	Weeks
Sheath size	7-8 Fr	13-14 Fr Impella 5.0 - 21 Fr	15-17 Fr Arterial 21 Fr Venous	14-16 Fr Arterial 18-21 Fr Venous
Femoral Artery Size	>4 mm	Impella 2.5 & CP - 5-5.5 mm Impella 5 - 8 mm	8 mm	8 mm
Cardiac synchrony or stable rhythm	Yes	No	No	No
Afterload	↓	↓	↑	↑↑↑
MAP	↑	↑↑	↑↑	↑↑
Cardiac Flow	↑	↑↑	↑↑	↑↑
Cardiac Power	↑	↑↑	↑↑	↑↑
LVEDP	↓	↓↓	↓↓	↔
PCWP	↓	↓↓	↓↓	↔
LV Preload	---	↓↓	↓↓	↓
Coronary Perfusion	↑	↑	---	---
Myocardial oxygen demand	↓	↓↓	↔↓	↔

Evaluation and Management of Cardiogenic Shock

Recommendations for Evaluation and Management of Cardiogenic Shock Referenced studies that support the recommendations are summarized in the Online Data Supplements.		
COR	**LOE**	**Recommendations**
1	**B-NR**	**1. In patients with cardiogenic shock, intravenous inotropic support should be used to maintain systemic perfusion and preserve end-organ performance.**
2a	**B-NR**	**2. In patients with cardiogenic shock, temporary MCS is reasonable when end-organ function cannot be maintained by pharmacologic means to support cardiac function.**

Evaluation and Management of Cardiogenic Shock (con't.)

2a	B-NR	3. In patients with cardiogenic shock, management by a multidisciplinary team experienced in shock in reasonable.
2b	B-NR	4. In patients presenting with cardiogenic shock, placement of a PA line may be considered to define hemodynamic subsets and appropriate management strategies.
2b	C-LD	5. For patients who are not rapidly responding to initial shock measures, triage to centers that can provide temporary MCS may be considered to optimize management.

CONCLUSION

- Cardiogenic Shock remains lethal
- Early Revascularization improves survival
- Mechanical Circulatory Support is redefining the treatment paradigm
- Protocol Driven Approaches are promising