What is Cardiogenic Shock?

2023 HV Conference – State of the Heart

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Disclosures

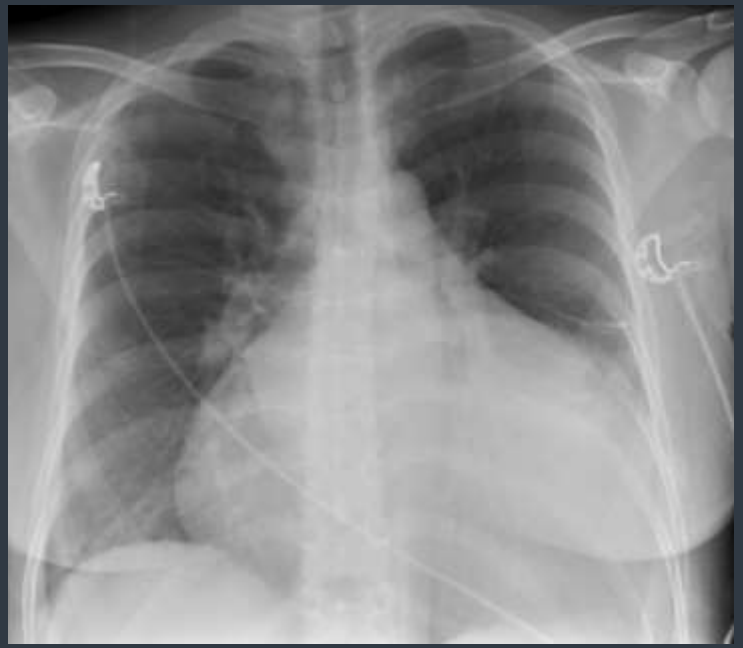
- Speaker's Bureau - Abbott

Objectives

1. Define what cardiogenic shock is
2. Understand how to identify and diagnose CS
3. Describe initial treatment strategies for CS

Case

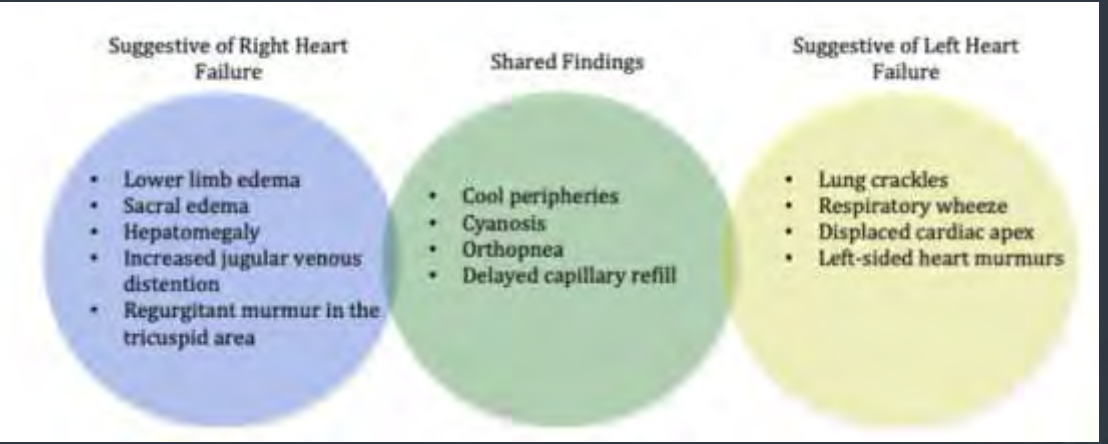
- 32 yo F, 4 days of DOE and fatigue
- Known HFrEF and drug abuse
- On lisinopril, spironolactone, and torsemide
- BP 144/109 HR 121 Temp 36.8 RR 31 O2 sat 99%
- Mild crackles, JVD noted to jaw, mottling of the LEs
- Hgb 13.5, Cr 1.1, AST/ALT 69/66, Lactate 2.6, BNP 2910



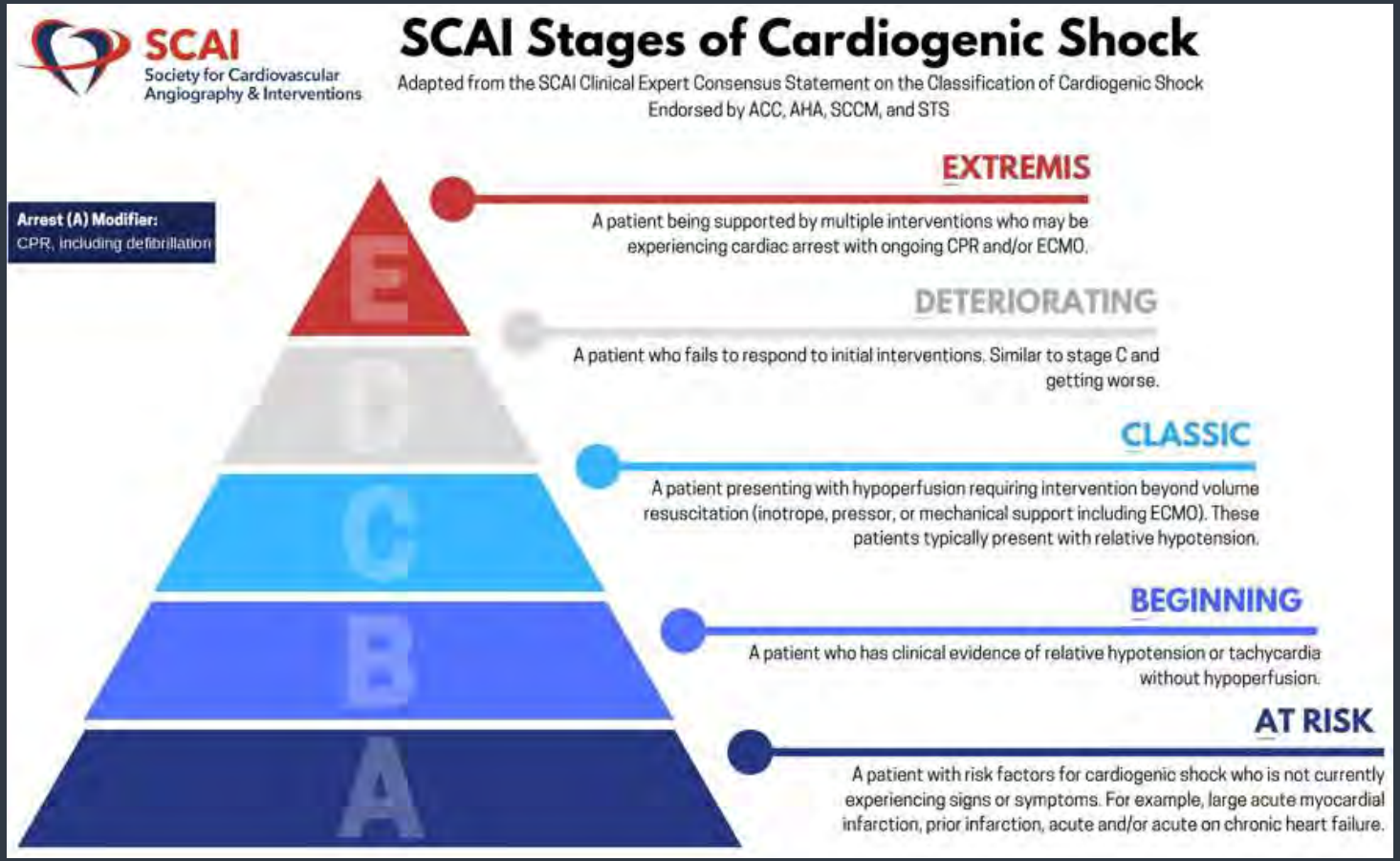
What is cardiogenic shock?

- Reduced cardiac output to the body resulting in poor O2 delivery and inability to meet metabolic demands

- Manifest as:
  - Hypotension
  - Elevated wedge and CI < 2.2 OR
  - Signs of poor perfusion
  - AMS
  - Low UOP
  - Cold, clammy skin

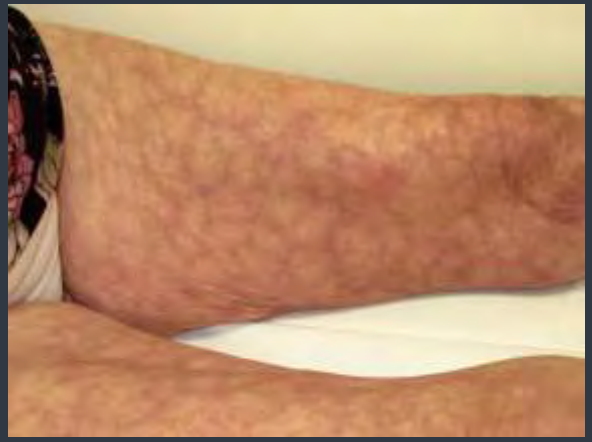


Vahdatpour et al. *JAHA* 2019


SCAI
Society for Cardiovascular Angiography & Interventions
SCAI Stages of Cardiogenic Shock
Adapted from the SCAI Clinical Expert Consensus Statement on the Classification of Cardiogenic Shock
Endorsed by ACC, AHA, SCCM, and STS
Arrest (A) Modifier:
CPR, including defibrillation
E
D
C
B
A
EXTREMIS
A patient being supported by multiple interventions who may be experiencing cardiac arrest with ongoing CPR and/or ECMO.
DETERIORATING
A patient who fails to respond to initial interventions. Similar to stage C and getting worse.
CLASSIC
A patient presenting with hypoperfusion requiring intervention beyond volume resuscitation (inotrope, pressor, or mechanical support including ECMO). These patients typically present with relative hypotension.
BEGINNING
A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.
AT RISK
A patient with risk factors for cardiogenic shock who is not currently experiencing signs or symptoms. For example, large acute myocardial infarction, prior infarction, acute and/or acute on chronic heart failure.

Making the Diagnosis

- Diagnosis is largely clinical
- Hypotension and/or hypoperfusion
  - Often SBP < 90 or MAP < 60
  - Narrow pulse pressure
  - Skin mottling, low UOP, AMS
- Venous Blood Gas
  - Low SvO2 often seen
- Lactic Acid
  - Usually, but not always elevated
- Jugular Venous Distension
  - Frequently not present
- ECG changes possible if 2/2 acute insult
- Cardiomegaly on chest imaging if acute on chronic



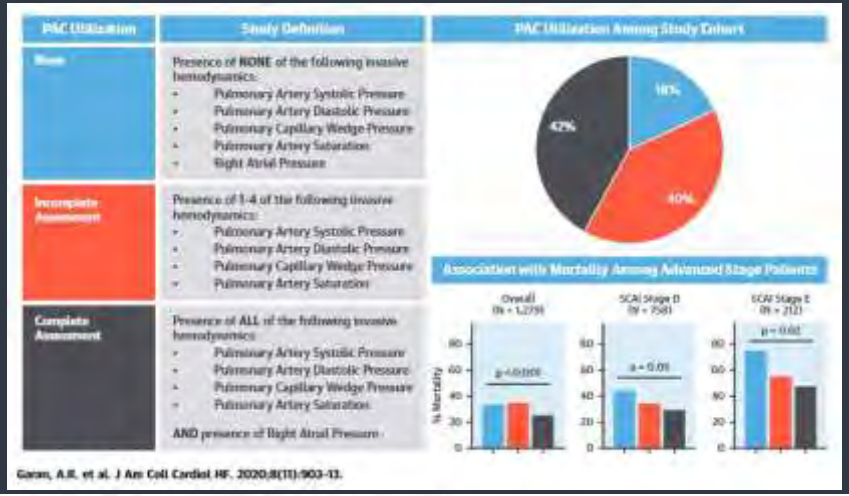
pH Mixed Venous	7.32 - 7.42 Units	7.44 ^	7.44 ^	7.44 ^	7.45 ^
pCO2 Mixed Venous	40 - 50 mmHg	40.0	38.5 v	37.6 v	39.0 v
pO2 Mixed Venous	35 - 40 mmHg	31.9 v	25.7 v	21.8 v	22.6 v
Bicarbonate Mixed Venous/Calc	22 - 30 mmol/L	26.5	25.8	25.3	26.8
Base Excess Mixed Venous	mmol/L	2.6	2.0 OK	1.5 OK	3.1 OK
Comment: Ref: -1 to 3 mmol/L					
O2 Saturation Mixed Ve-	60 - 80 %	49.0 v	33.6 v	24.1 v	26.3 v

SCAI SHOCK STAGE	PHYSICAL EXAM	BIOCHEMICAL MARKERS	HEMODYNAMICS
A	Normal JVP Lung sounds clear Strong distal pulses Normal mentation	Normal renal function Normal lactic acid	Normotensive (SBP≥100 or normal for pt.) If hemodynamics done: • Cardiac index ≥2.5 • CVP <10 • PA Sat ≥65%
B	Elevated JVP Rales in lung fields Strong distal pulses Normal mentation	Normal lactate Minimal renal function impairment Elevated BNP	SBP <90 OR MAP <60 OR >30mmHg drop Pulse ≥100 If hemodynamics done: • Cardiac Index ≥2.2 • PA Sat ≥65%
C	Ashen, mottled, dusky Volume overload Extensive Rales Killip class 3 or 4 BiPap or mechanical ventilation Acute alteration in mental status	Lactate ≥2 Creatinine doubling OR >50% drop in GFR Increased LFTs Elevated BNP Urine Output <30mL/h	Drugs/device used to maintain BP above stage B values. • Cardiac Index <2.2 • PCWP >15 • RAP/PCWP ≥0.8 • PAPI <1.85 • Cardiac Power Output ≤0.6
D	Any of stage C	Any of stage C AND deteriorating	Any of stage C AND Requiring multiple pressors OR addition of mechanical circulatory support devices to maintain perfusion
E	Near pulselessness Cardiac collapse Mechanical ventilation Defibrillator used	Lactate ≥5 pH ≤ 7.2	No SBP without resuscitation PEA or Refractory VT/VF Hypotension despite maximal support

Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock. Catheter Cardiovasc Interv. 2019;1–9. https://doi.org/10.1002/ccd.28329
For more information, please visit: www.scai.org/shockdefinition

RHC/Swan Role in CS

- RHC/Swan help guide tx decisions
- Recent studies show improved outcomes in CS
- Improved survival
- Decreased 30-day readmits, time to readmit, death during readmit



Garan, A.R. et al. J Am Coll Cardiol HF. 2020;8(11):903-13.

Table 2. Index Admission In-Hospital Outcomes and Therapies

Outcomes	Total, N=226 168	Non-RHC, N=213 316	RHC, N=25 840	P value
Death, %	38.0	38.5	25.8	<0.001
Stroke, %	3.9	3.9	3.4	0.016
Need for hemodialysis, %	3.2	2.9	5.6	0.009
Mechanical ventilation, %	48.9	49.0	39.5	<0.001
Length of stay, d	15.3 (16.3)	14.3 (15.1)	22.7 (20.9)	<0.001

RHC indicates right heart catheterization

J Am Heart Assoc. 2021;10:e019843. DOI: 10.1161/JAHA.120.019843

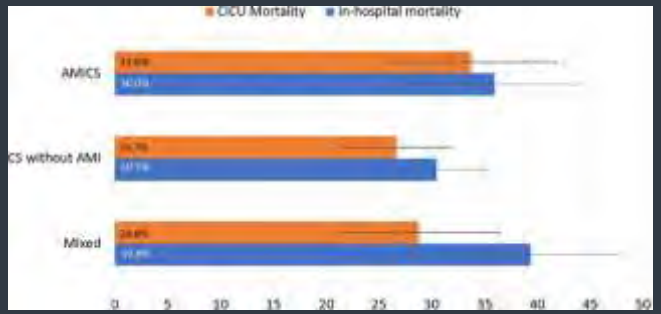
CS Mortality remains high

- Despite advancements,
  CS mortality remains 30-40%

- CS complicates 5-10% AMI
  - Leading cause of death post-MI

- Door-to-support time
  - Earlier MCS support associated with increased survival
  - Inova experience -> every 1hr delay in intensification ↑ mortality by 10%

	2012	2011	P for Trend
Overall MCS	23.9%	20.5%	<0.001
IABP	21.8%	18.8%	<0.001
Nonpercutaneous VAD	0.3%	0.2%	0.02
Percutaneous VAD	1.9%	2.9%	<0.001
ECMO	1.3%	2.0%	0.01
In-Hospital Mortality			
With Acute MI	37.6%	38.1%	0.46
Without Acute MI	38.8%	38.2%	0.53

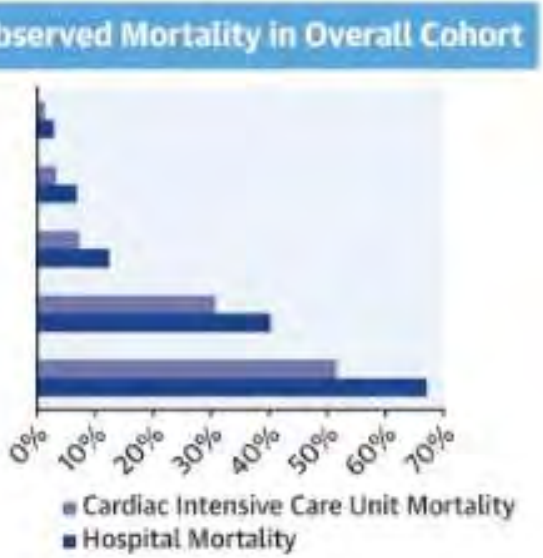


Panhwar et al. *Structural Heart* 2019
Berg et al. Circ: *CV Qual Outcomes* 2019

Cardiogenic Shock Mortality

CENTRAL ILLUSTRATION: Definitions of SCAI Shock Stages A Through E, With Associated Cardiac Intensive Care Unit and Hospital Mortality in Each SCAI Shock Stage

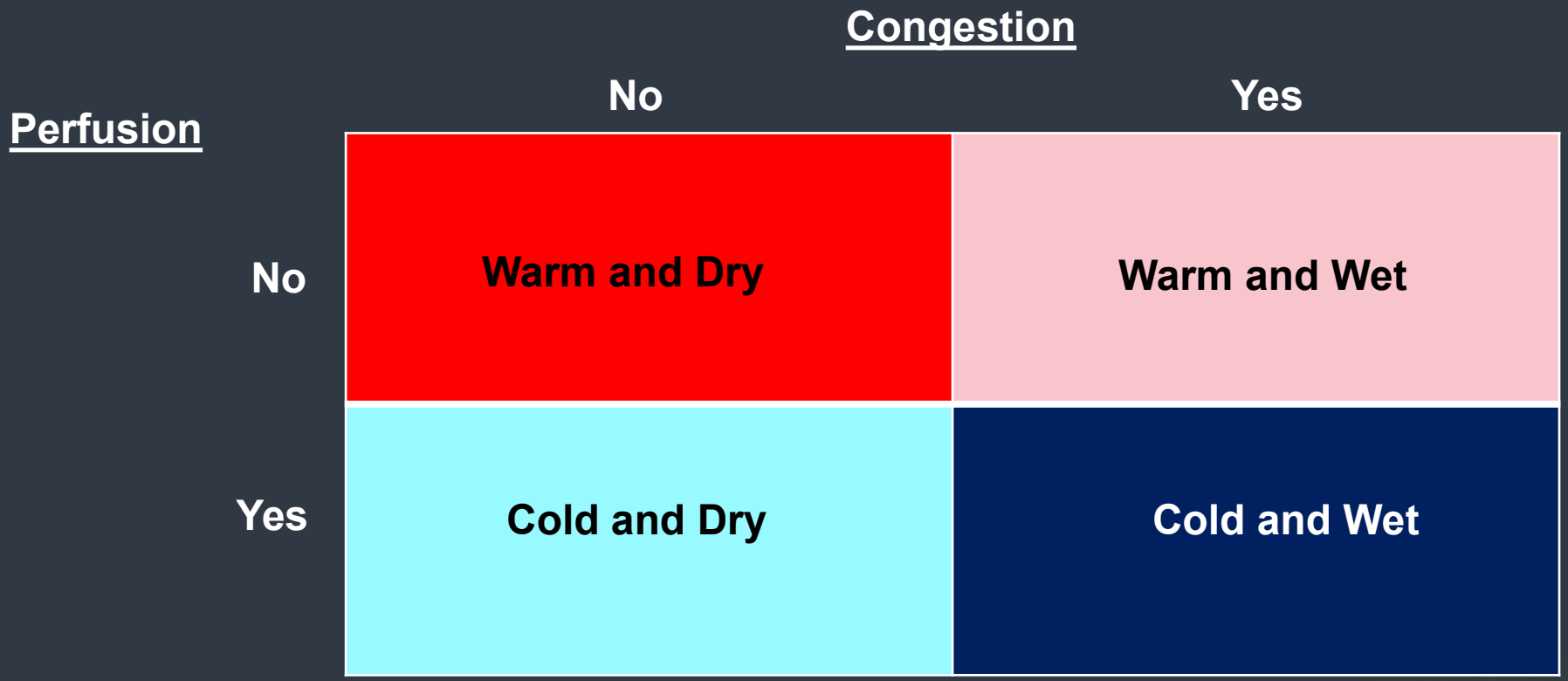
Cardiogenic Shock Stage	Study Definition
Stage A ("At risk")	Neither hypotension/tachycardia nor hypoperfusion
Stage B ("Beginning")	Hypotension/tachycardia WITHOUT hypoperfusion
Stage C ("Classic")	Hypoperfusion WITHOUT deterioration
Stage D ("Deteriorating")	Hypoperfusion WITH deterioration NOT refractory shock
Stage E ("Extremis")	Hypoperfusion WITH deterioration AND refractory shock



Jentzer, J.C. et al. J Am Coll Cardiol. 2019;74(17):2117-28.

Early Management Strategies

Volume and Perfusion Status



Initial Management Strategies

- Warm generally means perfusion is normal
- ≠ cardiogenic shock
- Warm/Dry
  - Optimization of HF GDMT
- Warm/Wet
  - Diuresis
    - Can trial oral initially
    - Most utilize IV if needing admission
    - Bolus vs Continuous drip
  - Optimize GDMT
- Any interaction with HF pt is an opportunity to optimize GDMT

Initial Management Strategies

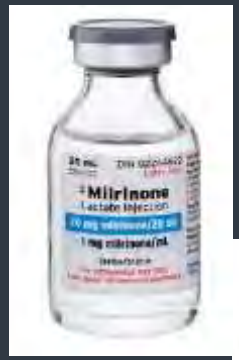
- Cold extremities concerning for low perfusion

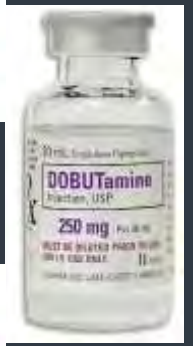
- Due to high afterload and/or low CO/CI
  - Afterload measured by SVR - - - > (MAP-RA)/CO
  - Acute MI shock may have primarily low CO/CI

- Initial therapy depends on primary etiology

Elevated Afterload

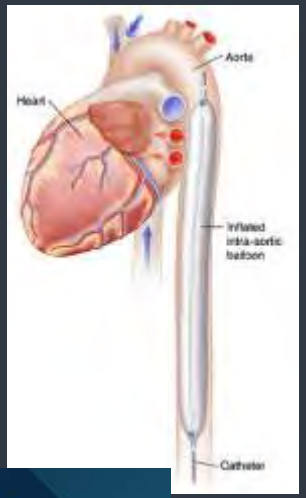
- Oral agents
  - Often not rapidly acting enough to work in CS
- Milrinone
  - PDE-3 inhibitor
  - Induces vasodilation along with cardiac contractility and relaxation
  - Prolonged time to peak onset (hours)
- Dobutamine
  - Beta agonist (primarily β1)
  - Limited β2 -> less impact on vasdilation than milrinone
- Sodium Nitroprusside
  - Used in cath lab to prove improvement in hemodynamics
  - Can be used short-term inpatient

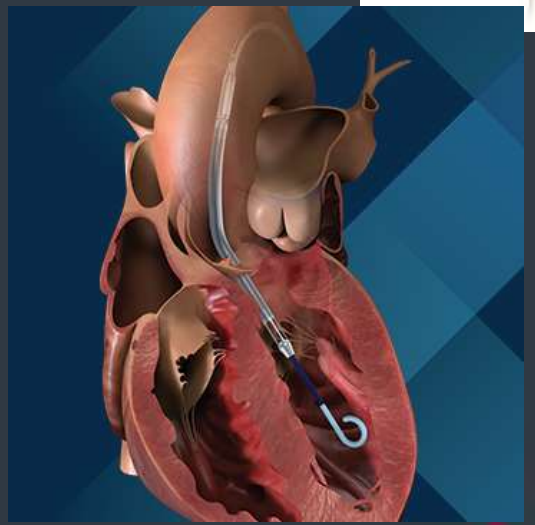




Reduced Cardiac Output

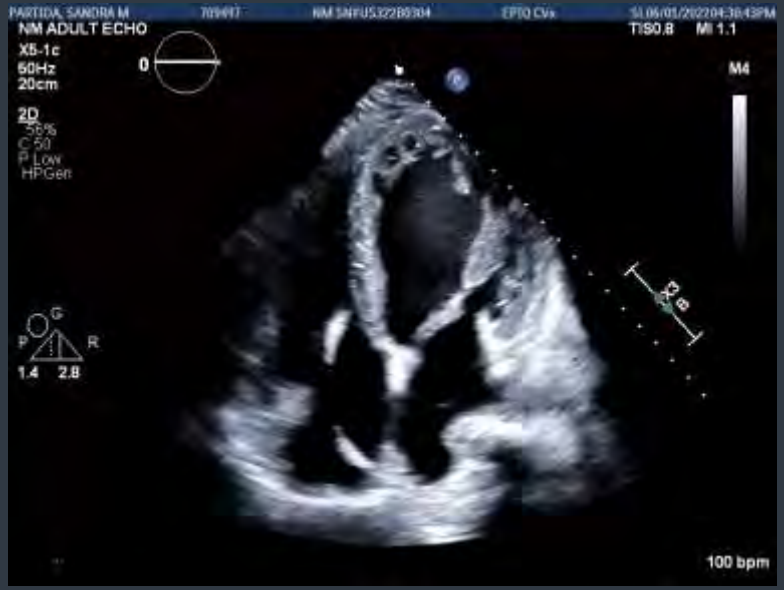
- Inotropic Support
  - Dobutamine – more contractility than vasodilation
  - Epinephrine – low doses inotropic support, higher doses vasoconstriction
- Intra-aortic balloon pump
  - 8F sheath, sits within descending aorta
  - Primary role is still afterload reduction
  - CO only increases by 0.5-1 L/min
  - Probably best for chronic HF with shock
- Microaxillary flow pump (Impella CP)
  - 14F sheath
  - Crosses AoV, direct LV unloading
  - Increases CO, up to 4 L/min
  - Increased risk of complications:
    - Hemolysis, access site complications, limb ischemia, ventricular arrhythmias





Case

- Sent for RHC from ED
  - BP 129/94 (105)
  - RA 23 mmHg
  - PA 67/35 (49) mmHg
  - Wedge 22 mmHg
  - PA sat 22%
  - Fick CO/CI 1.44/0.95
  - SVR 4543 Dynes.sec/cm5
- Started on milrinone + dobutamine
- Oral afterload agents optimized



Wrap Up

- CS generally presents with hypotension and hypoperfusion
- Despite improved diagnosis and tx options, mortality remains high
- SCAI staging system can help assess severity
- RHC/Swan has shown to decrease mortality in CS
- Initial management depends on the case
  - If hypervolemic, diuresis
  - Hypoperfusion -> reduce afterload and improve CO/CI
  - Whether trial inotropes first vs MCS depends on severity of shock
  - ALWAYS TAKE OPPORTUNITY TO OPTIMIZE GDMT

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