Pathophysiology of Cardiogenic Shock: Triage and Treatment

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Disclosures:
None
1. What is cardiogenic shock?
2. What are the causes of cardiogenic shock?
3. Why is it important to recognize and treat cardiogenic shock?
4. How should we treat cardiogenic shock?
5. Can we prevent cardiogenic shock? If so, how?
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What is Cardiogenic Shock?

Inability of the heart to adequately perfuse other vital organs caused by cardiac (ir)reversible dysfunction
## Cardiogenic Shock: Criteria

### Table 1. Pragmatic and Clinical Trial Definitions of CS

<table>
<thead>
<tr>
<th>Clinical Definition</th>
<th>SHOCK Trial**</th>
<th>IABP-SHOCK II†</th>
<th>ESC HF Guidelines†‡</th>
</tr>
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<tbody>
<tr>
<td>Cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion</td>
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<td><em>Clinical criteria:</em> SBP &lt;90 mm Hg for ≥30 min OR Support to maintain SBP ≥90 mm Hg AND End-organ hypoperfusion (urine output &lt;30 mL/h or cool extremities) Hemodynamic criteria: Cl of ≤2.2 L-min⁻¹.m⁻² AND PCWP ≥15 mm Hg*</td>
<td></td>
<td>Clinical criteria: SBP &lt;90 mm Hg for ≥30 min OR Catecholamines to maintain SBP &gt;90 mm Hg AND Clinical pulmonary congestion AND Impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output &lt;30 mL/h, or lactate &gt;2.0 mmol/L)</td>
<td>SBP &lt;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</td>
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Circulation. 2017;136:e232–e268
What is Cardiogenic Shock?

1) SBP < 90mmHg x 30min or vasopressors required to keep SBP > 90mmHg

2) Cardiac index < 1.8 without support and ~2-2.2 with support

3) Elevated filling pressures (wedge > 18mmHg)

4) Usually accompanied by evidence of hypoperfusion: cool extremities, decreased UOP, altered MS, elevated lactate
What are the causes of Cardiogenic Shock?

1) Acute myocardial infarction
2) AMI with mechanical complications
   (VSD, free wall rupture, acute MR)
3) Acute decompensated heart failure
   (ischemia, valvular disease, volume overload, arrhythmia)
   4) Post-cardiotomy shock
5) Acute rejection status/post transplant
6) RV mediated shock (i.e. massive PE)*
What are the causes of Cardiogenic Shock?

1) Acute myocardial infarction
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Why is it important to recognize and treat Cardiogenic Shock?
Why is it important to recognize and treat Cardiogenic Shock?

It kills people….frequently

For CS due to STEMI, the in-hospital mortality rate has decreased from 60.3% in 1995 to 47.9% in 2004. JAMA. 2005;294(4):448.

Even in more recent studies, the in-hospital mortality rate remains ~30%. J Am Heart Assoc. 2014 Feb; 3(1): e000590.
Pathophysiology of Cardiogenic Shock
Injury

Pathophysiology of Cardiogenic Shock

- Acute myocardial infarction
- LV Dysfunction
  - Systolic
  - Diastolic

- LV Injury

- Inotropes/Vasopressors
- Mechanical support: IABP/LVAD
- Bleeding/Transfusion
- Reperfusion: PCI/CABG

- SIRS
- Cardiac output ↓
  - Stroke volume ↓
- Peripheral perfusion ↓
- Hypotension
- Coronary perfusion ↓

- Ischaemia
  - LVEDP ↑
  - Lung oedema
  - Hypoxaemia

- Vasoconstriction
- Fluid retention

- SVR ↓
  - Pro-inflammation
  - Catecholamine sensitivity ↓
  - Contractility ↓

- Death

- Progressive LV Dysfunction
Pathophysiology of Cardiogenic Shock

Microvascular dysfunction

LV Injury
Pathophysiology of Cardiogenic Shock

NO/Inflammation

Systemic Inflammatory Response Syndrome (SIRS)

Microvascular dysfunction

LV Injury

LV Dysfunction

LV Dysfunction systolic diastolic

Acute myocardial infarction

Cardiac output ↓ Stroke volume ↓

Peripheral perfusion ↓

Coronary perfusion ↓

Ischaemia

LVEDP ↑ Lung oedema

Hypoxaemia

Death

SVR ↓ Pro-inflammation
Catecholamine sensitivity ↓ Contractility ↓
Pathophysiology of Cardiogenic Shock

LV Injury

NO/inflammation

Systemic Inflammatory Response Syndrome (SIRS)

Microvascular dysfunction
Pathophysiology of Cardiogenic Shock

LV Injury

LV Dysfunction
systolic

LV Dysfunction
diastolic

Acute myocardial infarction

Cardiac output ↓
Stroke volume ↓

Hypotension

Peripheral perfusion ↓

Coronary perfusion ↓

Ischaemia

Vasoconstriction
Fluid retention

Microvascular dysfunction

Systemic Inflammatory Response Syndrome (SIRS)

Reperfusion: PCI/CABG

Mechanical support: IABP/LVAD

Bleeding/Transfusion

eNOS
iNOS

NO ↑
Peroxynitrite ↑
IL-6 ↑
TNF-α ↑

SVR ↓
Pro-inflammation
Catecholamine sensitivity ↓
Contractility ↓

LVEDP ↑
Lung oedema

Hypoxaemia

Death

Progressive LV Dysfunction
How should we treat Cardiogenic Shock?
How should we treat Cardiogenic Shock?

Cardiac Output = Stroke Volume x Heart Rate
How should we treat Cardiogenic Shock?

Cardiac Output = \textbf{Stroke Volume} \times \text{Heart Rate}
Stroke Volume (SV): LV dysfunction

Decreased stroke volume (ischemia, rhythm issues, valvular disease, etc)
Stroke Volume (SV): LV dysfunction

- Decreased stroke volume (ischemia, rhythm issues, valvular disease, etc)
- Increased LV pressures (LVEDP)
Decreased stroke volume (ischemia, rhythm issues, valvular disease, etc)

Increased LV pressures (LVEDP)

Increased LA pressure

Stroke Volume (SV): LV dysfunction
Decreased SV
Decreased Pulse
Aortic Pressure
Acute systolic LV dysfunction
Increased LV pressures (LVEDP)
Decreased SV
Increased LA/wedge/ PA diastolic pressure
Decreased SV

Decreased Pulse

Aortic Pressure

Increased LV pressures (LVEDP)

Increased LA/wedge/PA diastolic pressure

Decreased SV

Left ventricular cardiogenic shock
Decreased SV

Significantly Decreased Aortic Pulse Pressure

Increased LV pressures (LVEDP)

Increased LA/wedge/PA diastolic pressure

Decreased SV

Left ventricular cardiogenic shock
Cardiogenic Shock hemodynamics

Wedge/LA/PAD/LVEDP ↔ LV SV ↔ Aortic pulse pressure (SBP-DBP)
Cardiogenic Shock hemodynamics

Wedge/LA/PAD/LVEDP ↔ LV SV ↔ Aortic pulse pressure (SBP-DBP)

Right Ventricle
Cardiogenic Shock hemodynamics

Wedge/LA/PAD/LVEDP  ➔  LV SV  ➔  Aortic pulse pressure (SBP-DBP)

Right Ventricle

RA/CVP  ➔  RV SV  ➔  

RA/CVP  ➔  RV SV  ➔  

LV SV  ➔  LV SV  ➔  

Wedge/LA/PAD/LVEDP  ➔  LV SV  ➔  Aortic pulse pressure (SBP-DBP)
Right Ventricular Failure/Shock

RA/CVP  ↔  RV SV  ↔  Pulm Pulse Pressure (PASP-PAD)

Decreased RV SV

Increased CVP/RA

Decreased Wedge/PAD/LA/LVEDP

Decreased PA pulsatility
Right Ventricular Failure/Shock

RA/CVP  ↔  RV SV  ↔  Pulm Pulse Pressure (PASP-PAD)

Assessment of RV function:

Pulmonary artery pulsatility index (PAPI):

\[(\text{PASP} - \text{PAD})/\text{CVP}\]

Normal ~ 1.8-2
Classification of Cardiogenic Shock

CVP

PCWP
Classification of Cardiogenic Shock

- **High CVP**
  - Low Wedge
- **Low CVP**
  - Low Wedge
  - High Wedge
Classification of Cardiogenic Shock

- **High CVP**
  - Low Wedge
- **Low CVP**
  - High Wedge
- Normal or Hypovolemia
Classification of Cardiogenic Shock

- High CVP
  - High Wedge
- Low CVP
  - High Wedge
- Normal or Hypovolemia
- RV Shock
Classification of Cardiogenic Shock

CVP

PCWP

RV Shock

Normal or Hypovolemia

High CVP
High Wedge

LV shock
Classification of Cardiogenic Shock

- Normal or Hypovolemia
- RV Shock
- Biventricular Shock
- LV shock
Classification of Cardiogenic Shock

- RV Shock
- Biventricular Shock
- Normal or Hypovolemia
- LV shock
Classification of Cardiogenic Shock

CVP

~12mmHg

~18mmHg

PCWP

Normal or Hypovolemia

RV Shock
ECMO
Impella RP
Protek Duo
Meds only

Biventricular Shock

LV shock
Classification of Cardiogenic Shock

CVP

~12mmHg

PCWP

~18mmHg

Normal or Hypovolemia

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Biventricular Shock

LV Shock
ECMO
LVAD
Impella CP
Meds only
Classification of Cardiogenic Shock

CVP

~12mmHg

Normal or Hypovolemia

~18mmHg

PCWP

RV Shock
- ECMO
- Impella RP
- Protek Duo
- Meds only

Biventricular Shock
- ECMO
- Impella CP + Impella RP/Protek Duo
- Impella CP + meds
- Impella RP/Protek Duo + meds
- Meds only

LV Shock
- ECMO
- LVAD
- Impella CP
- Meds only
LV Shock
ECMO
LVAD
Impella CP
Meds only
Left Ventricular Support: Impella

LV Shock
ECMO
LVAD
Impella CP
Meds only
Left Ventricular Support: Impella
Left Ventricular Support: Impella

14Fr system

Provides LV to aortic assist

Archimedes screw pump design

Nonpulsatile, axial flow

Provides 3-4L/min
Left Ventricular Support: Impella

- Femoral or axillary implantation
- Preload dependent
- Requires anticoagulation
- Contraindications: LV thrombus or mechanical AVR
Left Ventricular Support: Impella

Hemodynamic effects:

- Direct LV volume unloading
- Reduces myocardial oxygen consumption, improves MAP, and reduces wedge
**Left Ventricular Support: Impella**

**Benefits:**
- Higher support level vs IABP
- Does not rely on EKG triggering (stable with transient arrhythmias)
- Relatively easy implantation
Left Ventricular Support: Impella

**Drawbacks:**
- Expensive
- Large caliber sheath (vascular complications, bleeding)
- Hemolysis (5-10%)
- Device migration
- Requires preload (good RV function)
Classification of Cardiogenic Shock

CVP

~12 mmHg

Normal or Hypovolemia

RV Shock
ECMO
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Meds only

~18 mmHg

PCWP

Biventricular Shock
ECMO
Impella CP + Impella RP/Protek Duo
Impella CP + meds
Impella RP/Protek Duo + meds
Meds only

LV Shock
ECMO
LVAD
Impella CP
Meds only

Meds only
Right Ventricular Support:
Impella RP

| RV Shock   |
| ECMO      |
| Impella RP|
| Protek Duo|
| Meds only |
Right Ventricular Support: Impella RP
Right Ventricular Support: Protek Duo

- RV Shock
- ECMO
- Impella RP
- Protek Duo
- Meds only
Right Ventricular Support: Protek Duo

Benefits:
- IJ access
- In-line oxygenation
- Lower risk of hemolysis
Classification of Cardiogenic Shock

- RV Shock
  - ECMO
  - Impella RP
  - Protek Duo
  - Meds only

- Normal or Hypovolemia

- Biventricular Shock
  - ECMO
  - Impella CP + Impella RP/Protek Duo
  - Impella CP + meds
  - Impella RP/Protek Duo + meds
  - Meds only

- LV Shock
  - ECMO
  - LVAD
  - Impella CP
  - Meds only

CVP

~12mmHg

~18mmHg

PCWP
Classification of Cardiogenic Shock

- Biventricular Shock
  - ECMO
  - Impella CP + Impella RP/Protek Duo
  - Impella CP + meds
  - Impella RP/Protek Duo + meds
  - Meds only
Biventricular Support: ECMO

- ExtraCorporeal Membrane Oxygenation
- Unloads RV and LV
- Provides full cardiopulmonary support (biventricular + oxygenation); >4.5L/min depending on cannula size
- SVC cannula → deoxygenated blood → oxygenated → pumped into femoral artery
- RV, LV, and lungs → 2/3 down → ECMO
Biventricular Support: ECMO

Hemodynamic effects:

- Reduces LV preload but increases afterload, myocardial oxygen demand, and wall stress; often requires LV venting (with Impella) to unload LV.
Biventricular Support: ECMO
Biventricular Support: ECMO
Biventricular Support: ECMO

Veno-arterial (VA) ECMO
supports both heart and lungs

- Oxygenator
- Pump
- Drainage cannula
- Return cannula
Biventricular Support:
ECMO

Veno-arterial (VA) ECMO
supports both heart and lungs
Biventricular Support: ECMO

Veno-arterial (VA) ECMO supports both heart and lungs

Diagram showing the ECMO system with pump, oxygenator, drainage cannula, and return cannula.
Biventricular Support: ECMO
Biventricular Support: ECMO
Biventricular Support: ECMO

Speed and Simplicity.
The TandemLife Kit takes the complexity out of extracorporeal life support and can go anywhere you need to be.

Does not require perfusionist
Biventricular Support: Protek Duo + Impella CP

Does not require perfusionist
Biventricular Support: “Bipella”: Impella RP + Impella CP

Does not require perfusionist
No in-line oxygenation vs Protek Duo
2017 ESC guidelines for STEMI management:

Short-term mechanical support may be considered in patients with refractory shock (IIB/LOE C)

2013 ACC/AHA guidelines for STEMI management:

Alternative LV assist devices may be considered for circulatory support in patients with refractory CS (IIB/LOE C)
Percutaneous approaches for cardiogenic shock

1. There are no class I indications recommending any particular percutaneous device to treat cardiogenic shock

2. There are no RCTs demonstrating any mortality benefit for any percutaneous device used to treat cardiogenic shock
STEMI + Cardiogenic Shock Mortality Rates

Warden K et al, Eur Heart J (2014) 35:156-67
STEMI + Cardiogenic Shock Mortality Rates

IABP no longer recommended for cardiogenic shock
While advanced support devices have not shown mortality benefit, they are typically utilized to temporize hemodynamic instability in order to allow for recovery from cardiac stunning/injury.

Cardiogenic shock patients are diverse in presentation.

Difficult to conduct RCTs.
### Medical Therapy

#### Table 4. Mechanism of Action and Hemodynamic Effects of Common Vasoactive Medications in CS

<table>
<thead>
<tr>
<th>Medication</th>
<th>Usual Infusion Dose</th>
<th>Receptor Binding</th>
<th>Hemodynamic Effects</th>
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<tr>
<td></td>
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</tr>
<tr>
<td>Vasopressor/Inotropes</td>
<td></td>
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<tr>
<td>Dopamine</td>
<td>0.5–2 μg·kg⁻¹·min⁻¹</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>5–10 μg·kg⁻¹·min⁻¹</td>
<td>+</td>
<td>+++</td>
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<td>Norepinephrine</td>
<td>0.05–0.4 μg·kg⁻¹·min⁻¹</td>
<td>+++</td>
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<tr>
<td>Epinephrine</td>
<td>0.01–0.5 μg·kg⁻¹·min⁻¹</td>
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<tr>
<td>Phenylephrine</td>
<td>0.1–10 μg·kg⁻¹·min⁻¹</td>
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<td>Vasopressin</td>
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- Stimulates V₁ receptors in vascular smooth muscle

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<td>Inodilators</td>
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<td>Dobutamine</td>
<td>2.5–20 μg·kg⁻¹·min⁻¹</td>
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<td>Isoproterenol</td>
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<tr>
<td>Milrinone</td>
<td>0.125–0.75 μg·kg⁻¹·min⁻¹</td>
<td>PD-3 inhibitor</td>
<td>↑CO, ↓SVR, ↓PVR</td>
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<td>Levosimendan</td>
<td>0.05–0.2 μg·kg⁻¹·min⁻¹</td>
<td>Myofilament Ca²⁺ sensitizer, PD-3 inhibitor</td>
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CO indicates cardiac output; CS, cardiogenic shock; PD-3, phosphodiesterase-3; PVR, pulmonary vascular resistance; and SVR, systemic vascular resistance.
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CO indicates cardiac output; CS, cardiogenic shock; PD-3, phosphodiesterase-3; PVR, pulmonary vascular resistance; and SVR, systemic vascular resistance.
Cardiac Output = **Stroke Volume** $\times$ Heart Rate
Cardiac Output = Stroke Volume x Heart Rate
Cardiac Output = Stroke Volume x Heart Rate
5000cc/min = 70cc x 71bpm
Cardiac Output = Stroke Volume x Heart Rate
5000cc/min = 70cc x 71bpm

Cardiogenic Shock
5000cc/min = 35cc x
Cardiac Output = Stroke Volume x Heart Rate

5000cc/min = 70cc x 71bpm

Cardiogenic Shock

5000cc/min = 35cc x 142bpm
Cardiogenic Shock

\[ 5000 \text{cc/min} = 35 \text{cc} \times 142 \text{bpm} \]
When the SV is very low and the pressures of the RA, LA, or both are very high, what kind of tachycardic rhythm would you expect?

Cardiogenic Shock

$5000 \text{cc/min} = 35 \text{cc} \times 142 \text{bpm}$

- RV Shock
- Biventricular Shock
- Normal or Hypovolemia
- LV shock

CVP

$\sim 12 \text{mmHg}$

PCWP

$\sim 18 \text{mmHg}$
Atrial fibrillation with a rapid ventricular rate is not unexpected.

A rapid heart rate is how the body tries to sustain cardiac output when SV is very low.

Cardioversion or rate control can be detrimental.
<table>
<thead>
<tr>
<th>State</th>
<th>Flow (cc/min)</th>
<th>Volume (cc)</th>
<th>Heart Rate (bpm)</th>
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<tbody>
<tr>
<td>Normal</td>
<td>5000</td>
<td>70</td>
<td>71</td>
</tr>
<tr>
<td>Compensated</td>
<td>5000</td>
<td>35</td>
<td>142</td>
</tr>
<tr>
<td>Uncompensated</td>
<td>2275</td>
<td>35</td>
<td>65</td>
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</table>

AF + RVR (VR ~200)
Diastology

EF

HR

Mitral in-flow

A. Normal
B. Delayed relaxation (DD degree I)
C. Pseudonormal (DD degree II)
D. Restrictive pattern (DD degrees III and IV)
Diastology

EF

20%

HR

Mitral in-flow
Diastology

Rate control for AF/RVR beneficial

EF

20%

Mitral in-flow

HR
Diastology

Rate control for AF/RVR → improved LV filling

Mitral in-flow

EF

20%

HR
Diastology

Rate control for AF/RVR → improved LV filling

EF

Rate control for AF/RVR detrimental

Mitral in-flow

20%
Diastology

Rate control for AF/RVR → improved LV filling

Rate control for AF/RVR → no change in LV filling

Mitral in-flow

EF

20%
Diastology

Rate control for AF/RVR → improved LV filling

20%

EF

HR

Mitral in-flow

LV filling impaired

Tachycardia is trying to sustain CO

SV × HR = CO
Atrial fibrillation with a rapid ventricular rate is not unexpected

A rapid heart rate is how the body tries to sustain cardiac output when SV is very low

Cardioversion or rate control can be detrimental

Only holds for CS patients (i.e. very elevated filling pressures with very compromised SV)
Putting it all together
Putting it all together

1. Understand why and what
   - CAD, valvular disease?
   - RV shock, LV shock, or biventricular shock
Putting it all together

- RV Shock
- Biventricular Shock
- Normal or Hypovolemic
- LV Shock

CVP

~12mmHg

~18mmHg

PCWP
Putting it all together

1. Understand why and what
   - CAD, valvular disease?
   - RV shock, LV shock, or biventricular shock
2. Treat shock
PCWP

CVP

~12mmHg

~18mmHg

Normal or Hypovolemia

RV Shock
ECMO
Impella RP
Protek Duo
Meds only

Biventricular Shock
ECMO
Impella CP + Impella RP/Protek Duo
Impella CP + meds
Impella RP/Protek Duo + meds
Meds only

LV Shock
ECMO
LVAD
Impella CP
Meds only
Putting it all together

1. Understand why and what
   -CAD, valvular disease?
   -RV shock, LV shock, or biventricular shock
2. Treat shock
3. Decompression via fluid removal:
   -Bumex or Lasix gtt
   -CVVHD
4. Mechanical ventilation
5. Maintain organ perfusion
   -MAP 65-70mmHg with pressors, acid-base
6. Cardiac Power Output >0.53 watts; goal CVP <8mmHg, wedge <18mmHg
Cardiac Power Output

Cardiac Power Output

\[ \text{CPO} = \text{CO} \times \frac{\text{MAP}}{451} \]

- \( \text{CPO} \leq 0.53 \text{ W} = 58\% \text{ in-hospital mortality} \)
- \( \text{CPO} > 0.53 \text{ W} = 29\% \text{ in-hospital mortality} \)

What are causes of cardiogenic shock?

1) Acute myocardial infarction
2) AMI with mechanical complications
   (VSD, free wall rupture, acute MR)
3) Acute decompensated heart failure
   (ischemia, valvular disease, volume overload, arrhythmia)
   4) Post-cardiotomy shock
5) Acute rejection status/post transplant
6) RV mediated shock (i.e. massive PE)*
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Cardiogenic shock complicates 6% of AMIs annually
~60,000 cases/yr
Paradigm shift for STEMI: From door to balloon to door to unload?

Preclinical studies suggest that LV unloading for 30 min prior to PCI for acute myocardial infarction (without shock) is associated with benefits:
Impella for STEMI with Cardiogenic Shock

![Graph showing freedom from death over days after device implantation with log-rank test, P=0.04](image)

Door to unload: Decreased infarct size

**Primary Reperfusion**

**Primary Unloading + Reperfusion**

Door to unload: Less cellular perturbation

Door to unload:

Paradigm shift for STEMI: From door to balloon to door to unload?
STEMI + Cardiogenic Shock

42M with history of CAD s/p mid LAD 2011 presents with anterior STEMI and BP 80s.
STEMI + Cardiogenic Shock
STEMI + Cardiogenic Shock
STEMI + Cardiogenic Shock

Unload x 30 min before opening the artery?
Unload x 30 min then PCI versus immediate PCI?
Unload x 30 min then PCI versus immediate PCI?

Acute Mechanical LV Unloading in Ischemia Reperfusion Injury

Be Prepared*

Kiyotake Ishikawa, MD,a Bart Meyns, MD, PhDb
Cases
Case 1

70M previously healthy develops acute onset chest pain

EMS on scene, inferior STEMI $\rightarrow$ cath lab activated
Case 1
Case 1
Case 1
Case 1

Develops hypotension with BP 70s-80s and bradycardia

Swan ganz catheter inserted:

CVP/RA 20

PA 39/21

Wedge 17
Case 1

Swan ganz catheter inserted:

CVP/RA 20

PA 39/21

Wedge 17

- \(~12\text{mmHg}\)
- \(~18\text{mmHg}\)

- RV Shock
- Biventricular Shock
- Normal or Hypovolemia
- LV Shock
Swan ganz catheter inserted:

CVP/RA 20

PA 39/21

Wedge 17

PA pulsatility index = 39-21/20 = 0.8

RV Shock

Normal or Hypovolemia

Biventricular Shock

LV Shock

~12mmHg

~18mmHg

PCWP
Case 1: Inferior STEMI with RV shock

- **CVP ~12mmHg**
  - **RV Shock**
  - ECMO
  - Impella RP
  - Protek Duo
  - Meds only

- **CVP Normal or Hypovolemia**

- **CVP ~18mmHg**
  - **Biventricular Shock**
  - ECMO
  - Impella CP + Impella RP/Protek Duo
  - Impella CP + meds
  - Impella RP/Protek Duo + meds
  - Meds only

- **PCWP**
  - **LV Shock**
  - ECMO
  - LVAD
  - Impella CP
  - Meds only
What next?

A). RV support device
B). ECMO
C). Fluid bolus
D). Pressors
E). Milrinone
F). Lasix
G). D, E, and F
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B). ECMO
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G). D, E, and F
Case 1: Inferior STEMI with RV shock

Started on epinephrine to increase HR and help with BP

Started on milrinone for RV inotropy

Lasix 40mg IV administered with brisk UOP

Develops AF with RVR in lab

What should we do now?

Cardiovert, rate control, or nothing?
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Case 1: Inferior STEMI with RV shock

Patient transferred to Y3

Swan ganz guided therapy, continued diuresis to unload RV

Wean milrinone as RV function/PAPI/TAPSE recovers

Patient reverted to NSR once CVP down to reasonable level

Patient discharged home one week later

In follow up, no issues
Case 1: Inferior STEMI with RV shock

Patient transferred to Y3

Swan-ganz-guided therapy, continued diuresis to unload RV

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In follow-up, no issues

- **Atrial fibrillation with a rapid ventricular rate** is not unexpected
- A rapid heart rate is how the body tries to sustain cardiac output when SV is very low
- Cardioversion or rate control can be detrimental and likely unsuccessful before heart is unloaded

**Cardiogenic shock is typically not treated with IVF**
Case 2

- 52F with moderate aortic stenosis and ESRD now hypotensive following HD session (BP 90s on dopamine)

- Coronary angiography no change from prior study

- Stat TTE with EF 65%, severe, calcific aortic stenosis (AVA 0.8cm², max vel 4.3m/s, mean gradient 44mmHg), no AI, mild MS, no significant MR, mild TR, RV TAPSE = 18
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After urgent, multidisciplinary discussion, referred for emergent balloon aortic valvuloplasty

Temporary pacemaker tested, PEA arrest, CPR x 20 min

ROSC, systolic BP 60-70s
PEA arrest

Coded x 40 min

ROSC, sys BP 60

Impella CP device placed
Impella weaned and removed in 2 days

Patient extubated but required re-intubation several days later

Family withdrew care 2 weeks later
Impella weaned and removed in 2 days

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Family withdrew care 2 weeks later

You can’t win them all

But I did learn something…..
The next case of severe AS + Cardiogenic Shock.....
1. What is cardiogenic shock?
2. What are the causes cardiogenic shock?
3. Why is it important to recognize and treat cardiogenic shock?
4. How should we treat cardiogenic shock?
5. Can we prevent cardiogenic shock? If so, how?
Pathophysiology of cardiogenic shock

Systemic Inflammatory Response Syndrome (SIRS)

Microvascular dysfunction

NO/Inflammation

LV Injury

LV Dysfunction
systolic
diastolic

Acute myocardial infarction

Cardiac output ↓
Stroke volume ↓

Hypotension

Peripheral perfusion ↓

Coronary perfusion ↓

Ischaemia

Progressive LV Dysfunction

LVEDP ↑
Lung oedema

Hypoxaemia

Death

SVR ↓
Pro-inflammation
Catecholamine sensitivity ↓
Contractility ↓

NO ↑
Peroxyxynitrite ↑
IL-6 ↑
TNF-α ↑

Inotropes/ Vasopressors
Mechanical support: IABP/LVAD
Reperfusion: PCI/CABG

SIRS

eNOS
iNOS

Blood/Transfusion

Vasoconstriction
Fluid retention
Pathophysiology of cardiogenic shock

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LV Dysfunction

SIRS

Cardiac output ↓
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NO/ inflammation
Low Cardiac Output Syndrome

Stabilization/Recovery

Cardiogenic Shock
Low Cardiac Output Syndrome

Elevated filling pressures
Marginal systemic perfusion

Fatigue
Malaise
Poor appetite/nausea
Decreasing urine output

Low blood pressure***
IVF do not help patient
Resting tachycardia***
Cool to touch

Caution when EF<30
Extreme caution when EF<20

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Stabilization/Recovery
Low Cardiac Output Syndrome

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Check a lactate, CMP (Cr/LFTs), ABG
Consider holding rate-controlling agents
Increase diuretics
Refer for admission re: impending cardiogenic shock
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Low blood pressure***
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The answer is NOT fluids
The answer is NOT rate control
The answer is iontropes and diuresis

Refer for admission for impending cardiogenic shock

Stabilization/Recovery
Low Cardiac Output Syndrome

- Elevated filling pressures
- Marginal systemic perfusion
- Fatigue
- Malaise
- Poor appetite
- Nausea
- Decreasing urine output
- Low blood pressure

Cardiogenic Shock

Stabilization/Recovery

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- Consider holding rate-controlling agents
- Increase diuretics
- Refer for admission re: impending cardiogenic shock

The answer is NOT fluids

The answer is NOT rate control

The answer is ionotropes and diuresis
Thank you!

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