Cardiogenic Shock

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Definition

• Cardiogenic shock (CS) is a clinical condition of inadequate tissue (end organ) perfusion due to cardiac dysfunction

• Hypotension (SBP < 80-90 mmHg) or MAP 30 mmHg below baseline

• Reduced cardiac index (<1.8 L/min per m2) < 2.0-2.2 L/min per m2 with support

• Adequate or elevated filling pressures
Differentiating types of Shock

<table>
<thead>
<tr>
<th>Etiology</th>
<th>PCWP</th>
<th>CO</th>
<th>SVR</th>
<th>SVO2</th>
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</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td>v</td>
<td>v</td>
<td>^</td>
<td>v</td>
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<tr>
<td>Cardiogenic</td>
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<td>v</td>
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<td>v</td>
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<tr>
<td>Septic</td>
<td>v or =</td>
<td>^</td>
<td>v</td>
<td>^</td>
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Prevalence

- Occurs in 5-8% of STEMI and 2.5% of NonSTEMI
- 40,000-50,000 cases annually in the US
Mortality

- Current registries suggest a mortality rate of about 50%
- Historically rates quoted as high as 80-90%
- With early revascularization, newer modalities and aggressive treatment rates improving
Mortality Risks

- Older age
- Female sex
- Anterior wall STEMI
- Hypertension
- Type II Diabetes
- Multivessel Coronary Artery Disease
- Prior STEMI or Angina
- STEMI with new LBBB
- History of Heart Failure
Etiology

- Acute myocardial infarction with left ventricular failure (or rv failure)
- Acute mitral regurgitation
- Ventricular septal or free wall rupture
- Any other cause of acute right or left ventricular dysfunction
other etiologies

- Severe valvular heart disease (AS, AI, MS or MR)
- Post cardiotomy
- Acute fulminant myocarditis
- End stage cardiomyopathy
- Hypertrophic cardiomyopathy with severe LVOT obstruction
- Aortic dissection with acute severe AI or Tamponade
- Pulmonary Embolus
- Incessant refractory or prolonged tachyarrhythmias
- post cardiac arrest
Cardiogenic Shock
Who is at risk?

• Older age
• Female sex
• Anterior wall STEMI
• Hypertension
• Type II Diabetes
• Multivessel Coronary Artery Disease
• Prior STEMI or Angina
• STEMI with new LBBB
• History of Heart Failure
Signs and Symptoms

- Hypotension
- Absence of hypovolemia
- Tachycardia
- Clinical signs of poor perfusion (i.e., oliguria, cyanosis, cool extremities, altered mentation)
Physical Exam

- Skin - Ashen or cyanotic and cool, mottled
- Peripheral pulses rapid and faint, irregular
- JVD, Rales, Edema may be present
- Heart sounds distant, S3 or S4
- Pulse pressure low
- Decrease CO, Increased SVR, Decreased SvO2
Figure 1. Current concept of CS pathophysiology.

Systemic Inflammatory response syndrome (IL-6, TNF-α, NO)

↓Cardiac output
↓Stroke volume

↓Systemic perfusion

↓Hypotension

↓Coronary Perfusion pressure

Compensatory vasoconstriction

Myocardial infarction
Myocardial dysfunction
Systolic
Diastolic

LVEDP
Pulmonary congestion

Hypoxemia

Ischemia

Relief of ischemia

Progressive myocardial dysfunction

DEATH

Revascularization

SURVIVAL with GOOD QUALITY of LIFE

Hypoperfusion

• Increased catecholamine
  - peripheral arterial constriction
  - maintain perfusion to vital organs

• Vasopressin and Angiotensin II levels increase
  - increases coronary and peripheral perfusion
  - cost of increased LV afterload leading to further LV impairment

• Activation of neurohormonal cascade
  - promotes salt and water retention
  - improving perfusion at the cost of worsening pulmonary edema

• Reflex mechanisms to increase SVR not fully effective
What about RV dysfunction?

• 5% of CS cases from MI
• Typically pts have High RVEDP > 20mmHg
• Limited LV filling
decreased cardiac output (preload)
ventricular interdependance

  think pericardium and intraventricular septum

• Treatment is to assure adequate right sided filling pressure and adequate LV preload
More on CS due to RV dysfunction..

- Increases in RVEDP lead to RV dilation, septum bulging into LV, increases LA pressure and impaired LV systolic function
- Low RVEDP will affect LV preload and CO
- Optimal RVEDP range 10-15 mmHg
- Hemodynamic monitoring likely to be beneficial
- Mortality risk for CS due to primary RV dysfunction nearly as high as LV dysfunction
- Early Revascularization carries same survival benefits
Iatrogenic

Are we contributing?

• Beta Blockers, Ace Inhibitors, MSo4 decrease bp, slow heart rate and decrease in cardiac contractility may cause CS in certain High Risk patients

• Diuretics
  - in some patients pulmonary edema due to decreased LV compliance and net redistribution of intravascular volume to the lungs
  - further decline in circulating intravascular volume with diuretic use could lead to hypotension and shock
  - (lv compliance first to go in MI)
  - Treat with low dose diuretics, nitrates, seated position to reduce preload

• Excess volume loading in RV infarct

(Swan-Ganz ?)
Figure 3. Iatrogenic shock.

Systemic inflammatory response

- Inappropriate vasodilation
- Impaired perfusion of GI tract
- Transmigration of bacteria
- Sepsis
- Risk for SIRS increases with duration of shock
Initial Treatment and Evaluation

• EKG, ABG, Lytes, CBC, Troponin
• Urgent Transthoracic Echocardiogram
• ABC’s
• Circulation
  Treat arrhythmias
  Art Line
  Urinary Catheter
  Central venous (Swan-Ganz) catheter
Treatment cont.

• Aspirin and Heparin

• Dual antiplatelet therapy for PCI (not before)

• Maintain SaO2 and pH

• Aggressive insulin tx for hyperglycemia

• Low threshold for mech ventilation  
  (PEEP reduces preload and afterload) & (reduces work of breathing)

• REVASCULARIZE!
Hemodynamic Monitoring

• Swan-Ganz (PA catheter)
  - monitor cardiac output
  - monitor filling pressures (pap, pcw, rvedp)
  - continuous-

• ECHO Assessment
  - Estimated peak pap
  - Estimate pcw (MV early diastolic deceleration time)
    (<140 msec = pcwp > 20 mmHg)
  - onetime or intermittent-
Pharmacologic Support

- Norepinephrine (Levophed)
- Dopamine
- Dobutamine (Dobutrex)
- Phenylephrine
- Isoproterenol (Isuprel)
- IV Fluids

- Avoid negative inotropes and vasodilators
  (use lowest doses possible)
Receptor Activity

• **Alpha-1 receptor**
  - smooth muscle contraction
  - vasoconstriction
  - increased SVR

• **Beta-1**
  - Increased heart rate
  - increased myocardial contractility

• **Beta-2**
  - Smooth muscle relaxation
  - Decreased SVR

• **Dopamine**
  - Vasodilation (low dose)
  - Increased cardiac output
  - Vasoconstriction (high dose)
# Vasoactive Medication Receptor Activity and Clinical Effects

<table>
<thead>
<tr>
<th>Drug</th>
<th>Receptor activity</th>
<th>Predominant clinical effects</th>
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<tr>
<td></td>
<td>Alpha-1</td>
<td>Beta-1</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>+++</td>
<td>0</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>+++</td>
<td>++</td>
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<tr>
<td>Epinephrine</td>
<td>+++</td>
<td>+++</td>
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<table>
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<tr>
<th>Dopamine (mcg/kg/min)*</th>
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<tr>
<td>0.5 to 2.</td>
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<tr>
<td>5. to 10.</td>
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<tr>
<td>10. to 20.</td>
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<tr>
<td>Dobutamine</td>
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<tr>
<td>Isoproterenol</td>
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+++ : Very strong effect; ++ : Moderate effect; + : Weak effect; 0 : No effect.

* Doses between 2. and 5. mcg/kg/min have variable effects.
Vasoactive medication

- Vasopressors always required
  - use lowest doses possible
  - higher doses associated with poorer survival

- Inotropes treat for contractile failure
  - increases myocardial ATP uses
  - increases myocardial oxygen demand
  - Dobutamine may be useful if BP adequate

- AHA recommends norepinephrine first
  - 8-12 mcg/min and titrate

- Dopamine has inotropic properties

- Isoproterenol to treat shock due to bradycardia

- Phenylephrine to increase afterload in hocm or certain cases of Tako-Tsubo
Mechanical Support

- IABP
  - improve coronary and peripheral perfusion
  - initiate as quickly as possible
  - higher rates of survival in high use centers

- Newer devices
  - LV, RV or BiV assist devices
  - impella, tandem heart, extracorporeal life support (ecls)
  - Trials have shown hemodynamic improvement but no survival benefit thus far
IABP Shock II Trial

- 600 patients all treated with ERV and optimal medical therapy then randomized to IABP(300) or no IABP(298).
- Morality rate at 30 days: 39.7% IABP and 41.3% no IABP (p=0.69)
- 86.6% placed after PCI
- 10% of no IABP group crossed over (AHA Class IIa recommendation)
Danish Cardiogenic Shock Trial (DanShock)

- Conventional therapy vs Impella LVAD
- began enrollment in 2012
Reperfusion

• Percutaneous coronary intervention (PCI)
• Coronary Artery Bypass Grafting (CABG)
• Thrombolysis for patients not receiving PCI or CABG
Shock Trial
(Should we emergently revascularize Occluded Coronary arteries for shock)

• 302 patients randomized to either emergency revascularization (erv) or initial medical stabilization (ims)

• ERV- revascularization (PCI or CABG) within 6 hours of randomization

• IMS- could undergo delayed revascularization a minimum of 54 hours post randomization
Shock Trail

• Primary cause of Cardiogenic Shock
  74.5%   left ventricular failure
  8.3%    severe mitral insufficiency
  4.6%    ventricular septal rupture
  3.4%    isolated right ventricular failure
  1.7%    tamponade or cardiac rupture
  8%      other causes
Figure 5. Long-term follow-up of the SHOCK trial cohort. Early revascularization (ERV) is associated with sustained benefit.

Log-Rank $P=.03$

<table>
<thead>
<tr>
<th>No. at Risk</th>
<th>ERV</th>
<th>IMS</th>
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<tbody>
<tr>
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Shock Trial

- 13% increased 1 year survival with early revascularization (nnt = 8)
- 37% of early revascularization pts had CABG
- Rate of CABG in community < 10%
- 87% of pts in Shock Trial had multivessel disease
Quality of Life
as assessed by NYHA functional class

• Revascularized -75.9% NYHA class 1-11 2 weeks post DC

• Control group -62.5% NYHA class 1-11 2 weeks post DC

• 55% of those class 111-1V pts at 2 weeks that survived to 1 year improved to NYHA class 1-11
Figure 6. Functional status in the SHOCK trial. The majority of patients who survived 2 weeks after discharge had good functional status (and quality of life) at that time point.

Figure 4. 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.

**CARDIOGENIC SHOCK**

- **Early Shock**
  - Diagnosed on Hospital presentation
  - Fibrinolytic therapy if all of the following are present:
    1. Greater than 90 minutes to PCI
    2. Less than 3 hours post STEMI onset
    3. No contraindications
  - Arrange prompt transfer to invasive procedure capable center
- **Delayed Onset Shock**
  - Echocardiogram to rule out mechanical defects
  - Arrange rapid transfer to invasive capable center

**IABP**

- Cardiac Catheterization and Coronary Angiography

- 1-2 vessel CAD
  - PCI IRA
  - Staged Multivessel PCI

- Moderate 3-vessel CAD
  - PCI IRA

- Severe 3-vessel CAD
  - Cannot be performed
  - Immediate CABG
  - Staged CABG

- Left main CAD
  - Immediate CABG

Mechanical Complications

- Ventricular septal, free wall or papillary muscle rupture (12% of CS cases)
- Ventricular septal rupture mortality 87%
- Women and elderly at higher risk
- Acute MR from papillary or chordal rupture, or LV dilation and failure of leaflets to coapt appropriately
- Papillary muscle rupture more common with inferior MI
- Timely repair critical for survival
Special conditions

• LVOT obstruction/HOCM
  - No diuretic
  - No Inotropes
  - Use Beta Blocker
  - Use pure alpha agonist (phenylephrine)
    increases afterload, increases cavity size, decreases obstruction

• Tako-Tsubo
  - may see lvot obstruction
  - IABP
  - alpha agonist
  - No beta blocker
  - if no LVOT obstruction could use inotropes
AHA Guidelines

• Class I
  1. Early revascularization (PCI or CABG)
  2. Fibrinolysis in candidates unsuitable for ERV with no contraindications

• Class IIa
  1. Use of IABP can be useful in patients with CS who don’t quickly stabilize with pharmacologic therapy

• Class IIb
  Alternative LV assist device may be considered in patients with refractory CS
Conclusions

• Reduce Cardiogenic Shock cases
  Early recognition of the signs/sxs of MI
  Early repurfusion < 2 hours from sx onset

• CS is treatable with a chance for full recovery

• Early REVASCULARIZATION can improve short and long term survival and can result in excellent quality of life

• Aggressive early care even in highly unstable patients
Figure 2. Range of LVEF in studies of heart failure and in the SHOCK trial.

Recent MI

- VALIANT
- DINAMIT
- CAPRICORN
- EPHESUS

SHOCK Trial*

CHF

- MADIT II
- RALES
- ELITE II
- COPERNICUS
- COMET
- CHARM
- REMATCH*

Class I Recommendations for ICD Defibrillators

The Class I recommendations for ICD defibrillators\textsuperscript{1-3} are listed below. ICD therapy is indicated in patients:\textsuperscript{*}

\textit{Level of Evidence – A}
\begin{itemize}
  \item With LVEF \leq 35\% due to prior MI who are at least 40 days post-MI and are in NYHA Functional Class II or III
  \item With LV dysfunction due to prior MI who are at least 40 days post-MI, have an LVEF \leq 30\%, and are in NYHA Functional Class I
  \item Who are survivors of sudden cardiac arrest due to ventricular fibrillation (VF) after evaluation to define the cause of the event and to exclude any completely reversible causes
\end{itemize}

\textit{Level of Evidence – B}
\begin{itemize}
  \item With nonischemic DCM who have an LVEF \leq 35\% and who are in NYHA Functional Class II or III
  \item With nonsustained ventricular tachycardia (VT) due to prior MI, LVEF < 40\%, and inducible ventricular fibrillation or sustained ventricular tachycardia at electrophysiological study
  \item With structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable
  \item With syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study
\end{itemize}
\textsuperscript{*} Assuming patients are on chronic, optimal medical therapy and have a reasonable expectation of survival with good functional status for > 1 year.