Definition

Low-cardiac-output state resulting in life threatening end-organ hypoperfusion

Criteria:
1. Persistent hypotension (SBP <80 to 90 mm Hg or MAP 30 mm Hg lower than baseline)
2. Severe reduction in cardiac output
3. Adequate or elevated filling pressure
Clinical Findings:
1. Cool extremities
2. Altered mental status
3. ↓ pulses/narrow pulse pressure
4. End-organ dysfunction: oliguria, shock liver, elevated lactate

Confirmatory testing:
1. Pulmonary artery catheterization
2. Echocardiogram
## Must Differentiate Types

<table>
<thead>
<tr>
<th>Type of shock</th>
<th>CO</th>
<th>SVR</th>
<th>PWP</th>
<th>CVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Distributive</td>
<td>↑</td>
<td>↓↓</td>
<td>↓</td>
<td>↓</td>
</tr>
</tbody>
</table>

**CO** = Cardiac Output  
**SVR** = Systemic vascular resistance  
**PWP** = Pulmonary wedge pressure  
**CVP** = Central venous pressure
Classification

Forrester et al.  
Am J Cardiol 1977;39:137
Classification

Clinical Classifications

<table>
<thead>
<tr>
<th>Tissue perfusion</th>
<th>Pulmonary congestion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry and warm</td>
<td>Wet and warm</td>
</tr>
<tr>
<td>Dry and cold</td>
<td>Wet and cold</td>
</tr>
</tbody>
</table>
Causes

Myocardial infarction with subsequent LV dysfunction
Most common cause

Mechanical Defect
1. Acute mitral regurgitation (papillary muscle rupture)
2. Ventricular wall rupture (free wall or septal defect)
3. Tamponade
4. Left ventricular outflow obstruction (HOCM, AS)
5. Left ventricular inflow obstruction (MS, atrial myxoma)
## Causes – continued

**Contractility Defect**
1. Arrhythmias
2. Cardiomyopathy
3. Direct cardiac trauma
4. Sepsis
5. Medications
6. Myocarditis
7. Endocrine disorders
8. Pancreatitis
9. Pulmonary embolus
Myocardial Infarction

Most common cause of cardiogenic shock

Of the more than 1.2 million cases of MI that occur in the United States yearly, up to 8% will be complicated by cardiogenic shock (5-8% of STEMI, 2.5% of NSTEMI)

Babaev, A; JAMA 2005 Jul 27;294(4):448-54
Fox KA; Heart.2007; 93: 177–182
Mechanical complications must be considered in all patients with shock in the setting of MI.

1. Ventricular septal rupture
2. Free wall rupture
3. Papillary muscle rupture
Ventricular Septal Rupture

1. Occurs 2-8 days following MI
2. Loud systolic murmur, often with a thrill
3. 90% of patients die without surgery
4. Occurs more commonly in setting of first MI and/or delayed reperfusion
Free Wall Rupture

1. In-hospital mortality of > 60%
2. Risk factors first MI, advanced age, delayed presentation
3. JVD, pulsus paradoxus, diminished heart sounds
4. Emergent pericardiocentesis while transporting to operating room
Papillary muscle rupture

1. Occurs 2-8 days following MI
2. Loud systolic murmur
3. More common with inferior MI (posteromedial papillary muscle supplied by PDA, anterolateral dual supplied by LAD & LCX)
4. IABP/vasodilators while awaiting surgery
1. The left ventricle simultaneously benefits and suffers from low afterload
   - ↓ coronary flow
   - ↑ cardiac output with low afterload

2. Hypoperfusion causes release of catecholamines
   - ↑ contractility and peripheral blood flow
   - ↑ myocardial oxygen demand = ↑ afterload = myocardial dysfunction
   - ↑ arrhythmias

3. Activation of neurohormonal cascade
   - ↑ salt and water retention = ↑ improve perfusion & ↑ pulmonary edema

Causes of Persistent Shock

1. No–reflow phenomenon
2. Reversible myocardial dysfunction: stunning of LV
3. Vasodilatory pathway of shock:
   - ↑ levels of inflammatory cytokines
   - ↑ activity of the inducible form of nitric oxide synthase (iNOS) in infarcted heart muscle
4. Persistent ischemia in non-infarct related artery
   - More than 2/3rd of patients with MI and CS have multivessel disease
5. Bleeding
CS due to No-Reflow

CS due to No-Reflow
Right Ventricular Failure

Pulmonary Artery Pulsatility Index

\[ \text{PaPi} = \frac{\text{PA Systolic Pressure} - \text{PA Diastolic Pressure}}{\text{Right Atrial Pressure}} \]

Korabathina. Cath and Cardiovas Intervent 2012; 80:593-600
CS is Sometimes iatrogenic
ClOpidogrel and Metoprolol in Myocardial Infarction Trial

- 45,852 admitted to 1250 hospitals within 24 hours of AMI
- Randomly allocated to IV metoprolol then 200mg metoprolol versus placebo
Management

1. Anti-thrombotics
2. Anti-platelets
3. Revascularization/Reperfusion
4. PA catheterization to guide therapy
5. Mechanical circulatory support
Management

WHIP THE HORSE
UNLOAD THE WAGON
SLOW THE HORSE
GET A NEW HORSE
GET A TRACTOR
HEAL THE HORSE
Invasive Monitoring

Flow-directed catheter

<table>
<thead>
<tr>
<th>Pressure</th>
<th>Right atrium</th>
<th>Right ventricle</th>
<th>Pulmonary artery</th>
<th>Pulmonary artery wedge (PAOP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Impact of PA catheter

- Patients in ATTEND registry: n = 4842
  - Insufficient data collection: n = 43
  - Excluded in this analysis (IABP, PCPS, LVAD, CHDF): n = 365
- Patients in ATTEND registry: n = 4477
  - Propensity score matched patients: n = 1004
    - Control group: n = 502
    - PAC group: n = 502

Kaplan-Meier Estimates for Time to All-cause Death

Logrank test: P = 0.003

60 yo with hx CAD c/b ICM w/ EF 35% p/w respiratory failure. SBP 90/50, HR 90. Intubated, oliguric, crackles at bases.

**Medical therapy:**
- Nitroprusside/afterload reduction
- IV diuresis

**Consider:**
RV mechanical support
LV mechanical support

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA</td>
<td>10 mmHg</td>
</tr>
<tr>
<td>PA</td>
<td>37/23 mmHg</td>
</tr>
<tr>
<td>PCWP</td>
<td>16 mmHg</td>
</tr>
<tr>
<td>C.I.</td>
<td>1.7 mmHg</td>
</tr>
<tr>
<td>SVR</td>
<td>1200 mmHg</td>
</tr>
<tr>
<td>SVO2</td>
<td>45%</td>
</tr>
</tbody>
</table>
EARLY REVASCULARIZATION IN ACUTE MYOCARDIAL INFARCTION
COMPlicated BY CARDIOGENIC SHOCK

JUDITH S. HOCHMAN, M.D., LYNN A. SLEEPER, Sc.D., JOHN G. WEBB, M.D., TIMOTHY A. SANBORN, M.D.,
HARVEY D. WHITE, D.Sc., J. DAVID TALLEY, M.D., CHRISTOPHER E. BULLER, M.D., ALICE K. JACOBS, M.D.,
JAMES N. SLATER, M.D., JACQUES COL, M.D., SONJA M. MCKINLAY, Ph.D., AND THIERRY H. LEJEMTEL, M.D.,
FOR THE SHOCK INVESTIGATORS*
## SHOCK Trial

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Revascularization (N=152)</th>
<th>Medical Therapy (N=150)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPR, VT, or VF before randomization (%)*</td>
<td>32.7</td>
<td>23.9</td>
</tr>
<tr>
<td>Thrombolytic therapy (%)</td>
<td>49.3</td>
<td>63.3</td>
</tr>
<tr>
<td>Inotropes or vasopressors (%)</td>
<td>99.3</td>
<td>98.6</td>
</tr>
<tr>
<td>Intraaortic balloon counterpulsation (%)</td>
<td>86.2</td>
<td>86.0</td>
</tr>
<tr>
<td>Pulmonary-artery catheterization (%)</td>
<td>93.4</td>
<td>96.0</td>
</tr>
<tr>
<td>Left ventricular assist device (%)†</td>
<td>3.6</td>
<td>0.9</td>
</tr>
<tr>
<td>Heart transplantation (%)</td>
<td>2.0</td>
<td>0.7</td>
</tr>
<tr>
<td>Coronary angiography (%)</td>
<td>96.7</td>
<td>66.7</td>
</tr>
<tr>
<td>Angioplasty (%)</td>
<td>54.6</td>
<td>14.0</td>
</tr>
<tr>
<td>Stent placed‡</td>
<td>35.7</td>
<td>52.3</td>
</tr>
<tr>
<td>Platelet glycoprotein IIb/IIIa receptor antagonist§</td>
<td>41.7</td>
<td>25.0</td>
</tr>
<tr>
<td>Coronary-artery bypass grafting (%)</td>
<td>37.5</td>
<td>11.3</td>
</tr>
<tr>
<td>Angioplasty or coronary-artery bypass grafting (%)</td>
<td>86.8</td>
<td>25.3</td>
</tr>
<tr>
<td>Median time from randomization to revascularization (hr)¶</td>
<td>(0.6–2.8)</td>
<td>(79.0–162.0)</td>
</tr>
</tbody>
</table>

Hochman. NEJM 1999; 341:625-634
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.
Increasing Lactate & Mortality

High Dose Pressors & Mortality

Survival in Cardiogenic Shock


[Graph showing survival rates in cardiogenic shock from 1997 to 2006, with data points for overall death, death on admission, and death during hospitalization. Each point is marked with a corresponding symbol and line style, indicating significant improvements in survival over the years.]

- Death: cardiogenic shock overall ($P = 0.010$)
- Death: cardiogenic shock on admission ($P = 0.009$)
- Death: cardiogenic shock during hospitalization ($P = 0.094$)
Cardiac Power Is the Strongest Hemodynamic Correlate of Mortality in Cardiogenic Shock: A Report From the SHOCK Trial Registry

Rupert Fincke, MD,* Judith S. Hochman, MD, FACC,† April M. Lowe, MS,‡
Venu Menon, MD, FACC,§ James N. Slater, MD, FACC,† John G. Webb, MD, FACC,¶
Thierry H. LeJemtel, MD, FACC,¶ Gad Cotter, MD, FACC,# for the SHOCK Investigators

Power = Pressure * Flow

or

Cardiac Power = MAP * CO

451
IABP-SHOCK II Trial

- IABP did not reduce 30-day mortality
- Post-hoc analysis showed no mortality benefit of IABP among patients with SBP < 80 mmHg
1. 21F inflow cannula in left atrium via femoral venous puncture
2. 17F arterial cannula
3. 4L/min
ExtraCorporeal Membrane Oxygenation

VA-ECMO

Femoral Artery

Internal Jugular Vein

Returning Oxygenated Blood

De-oxygenated Blood

VV-ECMO
### Summary of Treatment Studies

#### Mortality

<table>
<thead>
<tr>
<th>Trial</th>
<th>Follow-up</th>
<th>n/N</th>
<th>n/N</th>
<th>Relative Risk 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Revascularization (PCI/CABG)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SHOCK</td>
<td>1-year</td>
<td>81/152</td>
<td>100/150</td>
<td>0.72 (0.54;0.95)</td>
</tr>
<tr>
<td>SMASH</td>
<td>30 days</td>
<td>22/32</td>
<td>18/23</td>
<td>0.87 (0.66;1.29)</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>103/184</td>
<td>118/173</td>
<td>0.82 (0.69;0.97)</td>
</tr>
<tr>
<td>Vasopressors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SOAP II (CS subgroup)</td>
<td>28 days</td>
<td>64/145</td>
<td>50/135</td>
<td>0.75 (0.55;0.93)</td>
</tr>
<tr>
<td>Inotropes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unverzagt et al.</td>
<td>30 days</td>
<td>5/16</td>
<td>10/16</td>
<td>0.33 (0.11;0.97)</td>
</tr>
<tr>
<td>Glycoprotein IIb/Illa-inhibitors</td>
<td>In-hospital</td>
<td>15/40</td>
<td>13/40</td>
<td>1.15 (0.59;2.27)</td>
</tr>
<tr>
<td>PRAGUE-7</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitric oxide synthase inhibitors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TRIUMPH</td>
<td>30 days</td>
<td>97/201</td>
<td>76/180</td>
<td>1.14 (0.91;1.45)</td>
</tr>
<tr>
<td>SHOCK-2</td>
<td>30 days</td>
<td>24/59</td>
<td>7/20</td>
<td>1.16 (0.59;2.69)</td>
</tr>
<tr>
<td>Cotter et al</td>
<td>30 days</td>
<td>4/15</td>
<td>10/15</td>
<td>0.40 (0.13;1.05)</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>125/275</td>
<td>93/215</td>
<td>1.05 (0.85;1.29)</td>
</tr>
<tr>
<td>IABP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IABP-SHOCK I</td>
<td>30 days</td>
<td>7/19</td>
<td>6/21</td>
<td>1.28 (0.45;3.72)</td>
</tr>
<tr>
<td>IABP-SHOCK II</td>
<td>30 days</td>
<td>119/300</td>
<td>123/298</td>
<td>0.96 (0.79;1.17)</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>126/319</td>
<td>129/319</td>
<td>0.98 (0.81;1.18)</td>
</tr>
<tr>
<td>LVAD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thiele et al</td>
<td>30 days</td>
<td>9/21</td>
<td>9/20</td>
<td>0.95 (0.48;1.90)</td>
</tr>
<tr>
<td>Burkhoff et al</td>
<td>30 days</td>
<td>9/19</td>
<td>5/14</td>
<td>1.33 (0.57;3.10)</td>
</tr>
<tr>
<td>Seyfarth et al</td>
<td>30 days</td>
<td>6/13</td>
<td>6/13</td>
<td>1.00 (0.44;2.29)</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>24/53</td>
<td>20/47</td>
<td>1.06 (0.68;1.66)</td>
</tr>
</tbody>
</table>
Summary of Treatment Studies

- **SHOCK**: 302 patients
- **TRIUMPH**: 398 patients
- **SMASH**: 55 patients
- **PRAGUE-7**: 80 patients
- **TACTICS**: 57 patients
- **IABP-SHOCK I**: 45 patients
- **IABP-SHOCK II**: 600 patients
- **CULPRIT-SHOCK**: 706 patients

Legend:
- Stop - no effect
- Stop - slow recruitment
- Potentially underpowered
- Stop - slow recruitment
- Surrogate endpoint
Feeling of Futility
Cardiogenic Shock Survivors

Cardiogenic Shock Survivors

Case history:

• 53 yo gentleman with no PMHx was found down by wife after searching for triathlons online
• Defibrillated in field by EMS
• Coded for 40 mins at ER
• Anterolateral STEMI on EKG – tx’d SLH
• Brought to cath lab with recurrent VT
Cardiogenic Shock Survivors
Cardiogenic Shock Survivors

Case history:

- Shocked multiple times in lab
- Stents placed in LAD and LCx
- Glycoprotein IIb/IIIa given during procedure after fluoroscopy showed undigested ticagrelor pills in stomach
- Blood noted to be coming from ETT near conclusion of procedure
Cardiogenic Shock Survivors

Case history:

- Severe hypoxia due to hemothorax – SpO2 in 30’s
- Independent lung ventilation strategy pursued
- IR for bronchial artery embolization attempted
- Prolonged hospital course with acidosis, AKI, c.diff colitis, multiple transfusions, encephalopathy
Cardiogenic Shock Survivors
1 Year Later...
Shock Center

1. 24/7 PCI with available use of hemodynamic support

2. Available consultants
   - Advanced heart failure/transplant specialists
   - EP with expertise in complex VT ablation
   - Echocardiography 24/7
   - In-house intensivists
   - Neurology
   - Palliative care
   - CT surgery
Regional System of Care

A. Direct transfer to Shock Center by-passing closest non-shock site

B. Cardiogenic Shock Diagnosed in the Field

C. MD-to-MD dialogue

D. Shock Team Deployed

Non-Shock Spoke Center PCI Capable
Non-Shock Spoke Center Not PCI Capable

Hub Cardiogenic Shock Center
Saint Luke’s Experience

**Cardiogenic Shock?**

<table>
<thead>
<tr>
<th>Inclusion Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>STEMI or unstable NSTE-ACS OR Rapid Response/Code Blue called for suspected cardiogenic shock/ACS OR SBP &lt; 90, MAP &lt; 60, HR &gt; 100 or Considering and/or initiating vasopressors/inotropes or End-organ dysfunction*</td>
</tr>
</tbody>
</table>

---

**Acute Coronary Syndrome?**

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV Lab</td>
<td></td>
</tr>
<tr>
<td>TO CICU</td>
<td></td>
</tr>
</tbody>
</table>

**TO CICU**

- Hemodynamically guided therapy to include:
  - Afterload reduction in setting of high SVR
  - Diuretic therapy for elevated PCWP
  - Consider transfer to SLH CICU if at regional ICU
  - Consider coronary angiography to rule out ischemia as indicated

<table>
<thead>
<tr>
<th>CV Lab</th>
</tr>
</thead>
<tbody>
<tr>
<td>If LVEDP &gt; 20mmHg or CI &lt; 2.0, place mechanical support if anatomy suitable</td>
</tr>
<tr>
<td>Complete revascularization</td>
</tr>
<tr>
<td>TIMI III flow</td>
</tr>
<tr>
<td>Transfer to SLH CICU after any mechanical support placement</td>
</tr>
</tbody>
</table>

---

**Hemodynamic Goals Met?**

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Begin mechanical support wean once all pressors off</td>
<td></td>
</tr>
<tr>
<td>Ongoing hemodynamic monitoring/reassessment</td>
<td></td>
</tr>
<tr>
<td>Continue guideline directed optimal medical therapy</td>
<td></td>
</tr>
</tbody>
</table>

| Prompt consultation with HF/Transplant to consider if multi-disciplinary evaluation and treatment in conjunction with CT Surgery & Cardiac Anesthesia is appropriate |
| Prompt discussion with SLH CICU attending re: transfer |

---

**Best Practices**

- Arterial line monitoring
- PA catheter placement
- Bedside echocardiogram
- Immediate CO/Ci/SvO2/lactate upon arrival in CICU and q2 hours thereafter
- Prompt critical care consultation

**Hemodynamic Goals:***

- $CFO = (MAP * CO) / 451 > 0.6 \text{ W}$
- $\text{PAP} = (sPAP - dPAP) / RA > 1.85$
- Cardiac Index > 2
- SBP > 90 or MAP > 55
- $\text{SVO2} > 60$
- Decreased lactate
- Able to wean off pressors
- No ventricular arrhythmias
- Improving end-organ dysfunction

---

*Signs of end-organ dysfunction may include the following, as acute/new findings:
1. lactate > 2
2. altered mental status
3. cold extremities/mottled skin
4. oliguria (< 30 mL/hr)
5. AST/ALT > 10x the ULN
6. troponin > 1

---

**†Hemodynamic Goals:**

**Cardiogenic Shock?**

**Inclusion Criteria:**
- STEMI or unstable NSTE-ACS
- Rapid Response/Code Blue called for suspected cardiogenic shock/ACS
- SBP < 90, MAP < 60, HR > 100
- Considering and/or initiating vasopressors/inotropes
- End-organ dysfunction*

**Acute Coronary Syndrome?**

- No
  - **To CICU**
    - Hemodynamically guided therapy to include:
      - Afterload reduction in setting of high SVR
      - Diuretic therapy for elevated PCWP
      - Consider transfer to SLH CICU if at regional ICU
      - Consider coronary angiography to rule out ischemia as indicated

- Yes
  - **To CV Lab**
    - If LVEDP > 20mmHg or CI < 2.0, place mechanical support if anatomy suitable
    - Complete revascularization
    - TIMI III flow
    - Transfer to SLH CICU after any mechanical support placement
**Hemodynamic Goals Met?**

No

- Prompt consultation with HF/Transplant to consider if multi-disciplinary evaluation and treatment in conjunction with CT Surgery & Cardiac Anesthesia is appropriate
- If at OSH ICU, prompt discussion with SLH CICU attending re: transfer

Yes

- Begin mechanical support wean once all pressors off
- Ongoing hemodynamic monitoring/reassessment
- Continue guideline directed optimal medical therapy

---

*Signs of end-organ dysfunction may include the following, as acute/new findings:*
1. lactate > 2
2. altered mental status
3. cold extremities/mottled skin
4. oliguria (< 30 mL/hr)
5. AST/ALT > 10x the ULN
6. troponin > 1

**Best Practices**
- Arterial line monitoring
- PA catheter placement
- Bedside echocardiogram
- Immediate CO/CI/SvO2/lactate upon arrival in CICU and q2 hours thereafter
- Prompt critical care consultation

**Hemodynamic Goals:**
- $CPO = (MAP*CO)/451 > 0.6 W$
- $PAPI = (sPAP-dPAP)/RA > 1.85$
- Cardiac Index > 2
- $SBP > 90$ or $MAP > 55$
- $SVO2 > 60$
- Decreased lactate
- Able to wean off pressors
- No ventricular arrhythmias
- Improving end-organ dysfunction
“A momentary pause in death”

“There is a golden hour between life and death. If you are critically injured you have less than 60 minutes to survive. You might not die right then; it may be 3 days or 2 weeks later but something has happened to your body that is irreparable.”

Recovery  Time in Cardiogenic Shock  Death

Hemodynamic Problem  Hemo-Metabolic Problem

Adapted from Kapur, N. Quote from R Adams Cowley
Conclusions

1. Cardiogenic shock carries great risk of mortality, but great potential for recovery
2. Reversible causes must considered and ruled out
3. Pathophysiology is very complex with several potential iatrogenic causes.
4. Revascularization remains the cornerstone of therapy
5. Advanced circulatory support and transfer to tertiary referral center should be considered in patients not responsive to revascularization.
Thank You