The Spectrum and Management of Cardiogenic Shock

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Cowger Disclosures

• Institutional Grant Support for Clinical Trials
  – Medtronic (Minneapolis, MN)
  – Abbott (Abbott Park, IL)
• Steering Committee: Medtronic and Procyrion (Houston, TX)
• Research-related travel support and speaker: Abbott and Medtronic
Learning Objectives

• Recognize Cardiogenic Shock and the Spectrum of Shock
• Understand the Trends in Incidence of Cardiogenic Shock
• Know the Mortality associated with Cardiogenic Shock
• Know what CPO is and a goal value
• Understand the management of Cardiogenic Shock
Cardiogenic Shock

• “Insufficient CO and end-organ hypoperfusion”
  – Acute myocardial infarction
  – Acute heart failure due to myocarditis
    • Chemo
    • Viral
  – Acute/Progressive exacerbation of chronic heart failure
  – Arrhythmias
Cardiogenic Shock: On the Rise

- National Inpatient Sample
Cardiogenic Shock

STEMI is #1 Cause of Cardiogenic Shock

- $N=1,990,486$

National Inpt Sample

Kolte, JAHA 2014;3:e000590
Cardiogenic Shock

• Reduction in inpatient mortality

Kolte, JAHA 2014;3:e000590
Defining Cardiogenic Shock
Commonly Accepted Definition of Cardiogenic Shock

• Evidence of persistent hypotension without hypovolemia
  – SBP <90 mmHg or MAP <60 mmHg or use of vasopressors to maintain SBP >90 mmHg

• Evidence of reduced cardiac output/index
  – Low cardiac index (<1.8 L/min/m²)
  – Reduced cardiac power output (CPO)
    \[ CPO = \text{MAP} \times \text{CO}/451 \]
  – Reduced PAPi: RV involvement
    \[ \text{PAPi} = \text{PAS}-\text{PAD}/\text{RA} \]

Cowger J, Goldstein D., in Braunwald’s 2nd edition of Mechanical Circulatory Support, 2019 in press
Commonly Accepted Definition of Cardiogenic Shock (cont)

- Evidence of **end-organ hypoperfusion**
  - Cool extremities
  - Elevated lactic acid
  - Renal failure
  - Transaminitis
Cowger J, Goldstein D., in Braunwald’s 2nd edition of Mechanical Circulatory Support, 2019 in press
Cardiogenic Shock in Chronic vs. Acute Heart Failure

- CS presentation may differ between chronic end stage HF with CS and Acute MI (with prior “normal” heart) and CS
- Chronic HF patients “tolerate” shock better
  - Acute MI crash fast, chronic HF may slowly crash
- Acute MI patients are more likely to wean off support if the culprit is treated
  - Acute MI
  - Toxin
  - Chronic HF: “can’t fix broken”
Cardiogenic Shock in Chronic vs. Acute Heart Failure

- Chronic HF with CS
  - ↑RA
  - ↑mPAP
  - ↑Wedge

Chronic HF in CS:
- Lower SVO$_2$
- Less pH derangement
- Better “adapted” to chronic low flow

<table>
<thead>
<tr>
<th>TABLE 3. Oxygen and carbon dioxide measurements</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
</tr>
<tr>
<td>$C_a$O$_2$ (ml O$_2$/dL)</td>
</tr>
<tr>
<td>DO$_2$ (ml O$_2$/L/min)</td>
</tr>
<tr>
<td>$S_v$O$_2$ (%)</td>
</tr>
<tr>
<td>$C_v$O$_2$ (ml O$_2$/dL)</td>
</tr>
<tr>
<td>$\Delta$O$_2$ content (ml O$_2$/dL)</td>
</tr>
<tr>
<td>$O_2$ extraction ratio</td>
</tr>
<tr>
<td>P50 (mm Hg)</td>
</tr>
<tr>
<td>$P_a$CO$_2$ (mm Hg)</td>
</tr>
<tr>
<td>$P_v$CO$_2$ (mm Hg)</td>
</tr>
<tr>
<td>$\Delta$PCO$_2$ (mm Hg)</td>
</tr>
<tr>
<td>$\Delta$PCO$_2$ /$\Delta$O$_2$ content</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 2. Strong ions and acid–base balance</th>
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<tbody>
<tr>
<td></td>
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<tr>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>pH</td>
</tr>
<tr>
<td>Na (mmol/L)</td>
</tr>
<tr>
<td>Cl (mmol/L)</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
</tr>
<tr>
<td>BE</td>
</tr>
</tbody>
</table>

BE indicates base excess; Cl, chloride; Na, sodium.
Pathophysiology of Cardiogenic Shock
Pathophysiology of CS

Coronary perfusion pressure

- Coronary perfusion pressure (CPP): Driving force for coronary blood flow
  - When vascular resistance is constant
    \[ \text{CPP} \propto \text{coronary flow} \propto \text{CO} \]
  - Need CPP of at least 15 mmHg
  - CPP needs are higher with CAD

Pathophysiology of CS: “Shock Begets Shock”

Coronary perfusion pressure

- Coronary perfusion pressure (CPP): Driving force for coronary blood flow
  - When vascular resistance is constant
    CPP \propto \text{coronary flow}

\[ \text{CPP} = \text{DBP} - \text{LVEDP} \]

DBP = diastolic blood pressure
Pathophysiology of CS

SIRS

- AMI with release of myocardial proteins triggers the “damage associated molecular patterns”
- Interleukin
- Chemokines
- Meant to clean up dead tissue → goes “haywire”
- Vasodilation, ↓SVR, ↑SVO₂ due to poor extraction

Fang J. Geriatric Cardiology 2015;12:305-12
SIRS and Mortality

• Even with normalized CO, mortality high!
Predictors of Death in Cardiogenic Shock
More inotropes, worse outcome: cVAD registry

- N=285 pts with Acute MI with CS supported with an Impella

Figure 2. Inhospital survival rates as a function of inotropic support to MCS implantation.

Delay in MCS associated with Mortality in AMI/CGS

Figure 2: In-Hospital Survival Rates as a Function of Shock Onset to MCS Implantation

66%  37%  26%
1st Tercile (n=43)  2nd Tercile (n=43)  3rd Tercile (n=43)

<75 mins  >4 hrs

Kaplan-Meier Curve for Freedom from Death (to 30 Days) by Device Implanted Pre/Post PCI. The separation of the Kaplan-Meier curves occurs very early post-PCI reinforcing that early MCS is a key determinant in clinical outcomes.


Vasopressors and mortality

Adj OR mortality = 4.7 with EPI in CS

Leopold, Intensive Care Me 2018;44:847-56.
Cardiac Power Output

- N=541 pt enrolled into SHOCK trial

CPO = MAP x CO / 451

CPO OR mortality = 0.55 [0.41-0.73] per unit increase

Fincke et al JACC 2004;44:340-8
Renal and Liver Dysfunction

- N-59 patient on ECMO

### Table 2. Hemodynamic Parameters and Laboratory Values

<table>
<thead>
<tr>
<th>Variable</th>
<th>Entire Cohort (n = 59)</th>
<th>Survivors to Discharge (n = 25)</th>
<th>In-Hospital Deaths (n = 34)</th>
<th>p Value</th>
<th>OR for Death (95% CI)^b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, beats/min</td>
<td>87 ± 2.6</td>
<td>92 ± 4.1</td>
<td>84 ± 3.5</td>
<td>0.12</td>
<td>...</td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>82 (69, 97)</td>
<td>87.8 (72.3, 99.3)</td>
<td>80.3 (67.3, 95.0)</td>
<td>0.96</td>
<td>...</td>
</tr>
<tr>
<td>Temperature, °C</td>
<td>37.2 (36.5, 37.7)</td>
<td>37.5 (36.8, 37.8)</td>
<td>37.1 (36.5, 37.6)</td>
<td>0.12</td>
<td>...</td>
</tr>
<tr>
<td>RA pressure, mm Hg</td>
<td>17 (14, 23)</td>
<td>16 (14, 21)</td>
<td>19 (14, 26)</td>
<td>0.56</td>
<td>...</td>
</tr>
<tr>
<td>PAP, mm Hg</td>
<td>27 (21, 35)</td>
<td>24 (18, 34)</td>
<td>29 (21, 37)</td>
<td>0.17</td>
<td>...</td>
</tr>
<tr>
<td>Systolic</td>
<td>40 (29, 52)</td>
<td>32 (22, 52)</td>
<td>42 (32, 53)</td>
<td>0.099</td>
<td>...</td>
</tr>
<tr>
<td>Diastolic</td>
<td>20 (16, 27)</td>
<td>20 (16, 26)</td>
<td>21 (15, 31)</td>
<td>0.48</td>
<td>...</td>
</tr>
<tr>
<td>LVAD flow, L/min</td>
<td>4.8 (4.3, 5.2)</td>
<td>4.8 (4.3, 5.2)</td>
<td>4.9 (4.3, 5.1)</td>
<td>0.95</td>
<td>...</td>
</tr>
<tr>
<td>RVAD flow, L/min</td>
<td>4.8 (4.1, 5.2)</td>
<td>5.0 (4.5, 5.3)</td>
<td>4.8 (4.0, 5.2)</td>
<td>0.29</td>
<td>...</td>
</tr>
<tr>
<td>PaO2/FiO2 ratio</td>
<td>122 (74, 177)</td>
<td>131 (80, 188)</td>
<td>105 (62, 172)</td>
<td>0.30</td>
<td>...</td>
</tr>
<tr>
<td>PEEP, cm H2O</td>
<td>5 (5, 8)</td>
<td>5 (5, 8)</td>
<td>5 (5, 8)</td>
<td>0.66</td>
<td>...</td>
</tr>
<tr>
<td>Level of pH</td>
<td>7.40 ± 0.013</td>
<td>7.42 ± 0.016</td>
<td>7.38 ± 0.020</td>
<td>0.14</td>
<td>...</td>
</tr>
<tr>
<td>Urine output, mL/h</td>
<td>670 (225, 1215)</td>
<td>750 (470, 1000)</td>
<td>360 (115, 1600)</td>
<td>0.13</td>
<td>...</td>
</tr>
<tr>
<td>CVVH ≤ 24 h</td>
<td>16 (27)</td>
<td>1 (6)</td>
<td>15 (94)</td>
<td>&lt;0.001</td>
<td>18.2 (2.2–150)</td>
</tr>
<tr>
<td>Inotrope</td>
<td>32 (54)</td>
<td>14 (44)</td>
<td>18 (56)</td>
<td>0.58</td>
<td>...</td>
</tr>
<tr>
<td>Vasopressor</td>
<td>34 (58)</td>
<td>9 (27)</td>
<td>25 (74)</td>
<td>0.024</td>
<td>4.0 (1.3–13)</td>
</tr>
<tr>
<td>Vasodilator</td>
<td>14 (24)</td>
<td>7 (50)</td>
<td>7 (50)</td>
<td>0.36</td>
<td>...</td>
</tr>
<tr>
<td>BUN, mg/dL</td>
<td>23 (16.5, 34)</td>
<td>19 (16, 29)</td>
<td>28 (18, 36)</td>
<td>0.099</td>
<td>...</td>
</tr>
<tr>
<td>Creatinine, mg/dL</td>
<td>1.5 (1, 2.3)</td>
<td>1.1 (0.9, 1.5)</td>
<td>2.1 (1.2, 2.8)</td>
<td>&lt;0.001</td>
<td>5.1 (1.9–14)</td>
</tr>
<tr>
<td>Creatinine &gt; 1.5 mg/dL</td>
<td>28 (48)</td>
<td>5 (18)</td>
<td>23 (82)</td>
<td>&lt;0.001</td>
<td>8.4 (2.5–28)</td>
</tr>
<tr>
<td>Glucose, mg/dL</td>
<td>140 (108.5, 154.5)</td>
<td>123 (106, 142)</td>
<td>147 (113, 174)</td>
<td>0.01</td>
<td>...</td>
</tr>
<tr>
<td>AST, IU/L</td>
<td>390 (140, 1126)</td>
<td>259 (131, 581)</td>
<td>611 (153, 2300)</td>
<td>0.054</td>
<td>...</td>
</tr>
<tr>
<td>ALT, IU/L</td>
<td>83 (48, 270)</td>
<td>73 (49, 115)</td>
<td>113 (48, 819)</td>
<td>0.21</td>
<td>...</td>
</tr>
<tr>
<td>Bilirubin, mg/dL</td>
<td>1.4 (0.9, 3.1)</td>
<td>1.1 (0.8, 1.8)</td>
<td>2.1 (1.2, 4.0)</td>
<td>0.016</td>
<td>1.3 (0.98–1.8)</td>
</tr>
<tr>
<td>INR, sec</td>
<td>1.2 (1.1, 1.5)</td>
<td>1.1 (1.0, 1.3)</td>
<td>1.3 (1.1, 1.7)</td>
<td>0.13</td>
<td>...</td>
</tr>
</tbody>
</table>

Haft, Cowger-Matthews ATS 2009;88:711-18
### Predictors of Survival at 12-24 hours (N=75)

**CARDIAC POWER OUTPUT**

<table>
<thead>
<tr>
<th>LACTATE</th>
<th>&gt; 0.6</th>
<th>≤ 0.6</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥4</td>
<td>63% Survival (n=5/8)</td>
<td>30% Survival (n=3/10)</td>
</tr>
<tr>
<td>&lt;4</td>
<td>96% Survival (n=45/47)</td>
<td>80% Survival (n=8/10)</td>
</tr>
</tbody>
</table>
Management
LV unloading and Flow

Cannula size determines flow!

<table>
<thead>
<tr>
<th></th>
<th>iVAC 2L®</th>
<th>TandemHeart™</th>
<th>Impella® 5.0</th>
<th>Impella® 2.5</th>
<th>Impella® CP</th>
<th>ECLS (multiple systems)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catheter size (F)</td>
<td>11 (expandable)</td>
<td>—</td>
<td>9</td>
<td>9</td>
<td>9</td>
<td>17–21 venous</td>
</tr>
<tr>
<td>Cannula size (F)</td>
<td>17</td>
<td>21 venous</td>
<td>21</td>
<td>12</td>
<td>9</td>
<td>16–19 arterial</td>
</tr>
<tr>
<td>Flow (L/min)</td>
<td>Max 2.8</td>
<td>Max. 4.0</td>
<td>Max. 5.0</td>
<td>Max. 2.5</td>
<td>3.7–4.0</td>
<td>Max. 7.0</td>
</tr>
<tr>
<td>Pump speed (rpm)</td>
<td>Pulsatile, 40 mL/beat</td>
<td>Max. 7500</td>
<td>Max. 33 000</td>
<td>Max. 51 000</td>
<td>Max. 51 000</td>
<td>Max. 5000</td>
</tr>
<tr>
<td>Insertion/Placement</td>
<td>Percutaneous (femoral artery)</td>
<td>Percutaneous (femoral artery + vein for left atrium)</td>
<td>Peripheral surgical (femoral artery)</td>
<td>Percutaneous (femoral artery)</td>
<td>Percutaneous (femoral artery + vein)</td>
<td></td>
</tr>
<tr>
<td>LV unloading</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>+</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>Anticoagulation</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Recommended duration of use</td>
<td>— 21 days</td>
<td>— 14 days</td>
<td>10 days</td>
<td>10 days</td>
<td>10 days</td>
<td>— 7 days</td>
</tr>
<tr>
<td>CE-certification</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>FDA</td>
<td>—</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Relative costs</td>
<td>++</td>
<td>++++++++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+ (++)</td>
</tr>
</tbody>
</table>

Thiel. European Heart J 2015;36:1223-1230
Mortality

SHOCK-II Trial: 600 pts with AMI with CS. Randomized to IABP or not during PCI

- No difference in survival (51% and 52% 1Y survival); no difference in renal function, lactate, LOS, catechol dose

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Delay in MCS associated with Mortality in AMI/CGS

Figure 2: In-Hospital Survival Rates as a Function of Shock Onset to MCS Implantation

66% <75 mins
37% 2nd Tercile (n=43)
26% 3rd Tercile (n=43)

Log-rank test, P=0.04

Kaplan-Meier Curve for Freedom from Death (to 30 Days) by Device Implanted Pre/Post PCI. The separation of the Kaplan-Meier curves occurs very early post-PCI reinforcing that early MCS is a key determinant in clinical outcomes.


Need More Randomized Clinical Trials

- How do you match patients in shock?: Shock is on a spectrum of illness
- Is it ethical to without support?
- Is it ethical to put pts on support and risk limb ischemia and death without good data?
Temporary Circ Support (TCS) Outcomes

Death

Cardiogenic shock with TCS

Failure to Wean

Improvement with TCS Wean!

Transplant

Days, weeks, months
LVAD Therapy

- **FDA Approved**
  - HeartMate II
    - Thoratec
    - Abbott

- **FDA approved for Short Term Support only**
  - HeartMate 3
    - Thoratec
    - Abbott

- **FDA Approved**
  - HVAD
    - HeartWare
    - Medtronic
MOMENUMT3 Clinical Trial: HeartMate 3 vs HeartMate II LVAD

• 1:1 Randomized trial of HM3 vs HMII LVAD

• Inclusion:
  – LVEF ≤25%
  – NYHA III or IV
  – Inotrope dependent or CI <2.2 off
HeartMate 3 Endpoints

Disability Stroke 2Y
7% HM3
8% HMII

Reop for clot/malfunction
3% HM3
25% HMII

Survival

HR = 0.71 (95% CI: 0.44 - 1.15)
P = 0.16 by log-rank test

Mehra et al. NEJM 2017; 376:440. DOI: 10.1056/NEJMoa1610426
LVAD

- Patients in shock prior to VAD have worse survival
- Early referral is KEY!
TCS for “Bridge to Decision”

- N=58 patients
- Impella 5 for “Bridge to Decision”
- Need to get them to a TXP/VAD center by day 3-4 of TCS if not improving

Hall et al. JHLT 2018;37:100-06.
Take Home Points

- Incidence of CS is increasing
- Mortality from CS is decreasing
- Increased use of Temp Circ Support in US but benefit is not fully clear
  - Probably beneficial in carefully selected and well managed patients
- **EARLY** referral of recalcitrant CS is key to long-term survival
  - Day 3-4: if not better → Refer out
  - Think about LONG TERM survival not just “this stay”
Jennifer Cowger, MD, MS
(734) 546 4911 (call or text)
jcowger1@hfhs.org
Good References


Case 1

• 32 yo F presents to ER with CP and anterior STEMI
• SCAD
• Coded → Impella → CCU
Scenario 1

- Day 1
  - Flows 3.9L on p8
  - BP 92/78
  - 99% sat on 1L
  - RA 8, PA 35/16, WP 14, SVO2 68%, Hg 9, CO 4.4
  - No gtts

- Day 3
  - Flows 1.7 L on P2
  - BP 110/70(83)
  - 99% sat on RA
  - RA 8, PA 32/14, Wp 10, SVO2 64%, CO 4.2
Scenario 2

- **Day 1**
  - Flows 3.6 L on P8
  - BP 88/83
  - 99% sat on 1L
  - RA 6, PA 40/20, WP 20, SVO2 65%, Hg 9, CO 3.9
  - No gtts

- **Day 3**
  - Flows 1.7 L on P2
  - BP 82/73(76)
  - 99% sat on 4L
  - RA 7, PA 45/30, Wp 29, SVO2 43%, CO 2.8
  - No gtts
Scenario 3

Day 1
- Flows 3.9L on p8
- BP 92/78, HR 102, temp 37
- 99% sat on 1L
- RA 8, PA 35/16, WP 14, SVO2 68%, Hg 9, CO 4.3
- No gtts

Day 3
- Flows 2.0 L on P5
- BP 83/79, HR 115, temp 39
- Vaso, levo
- 99% sat on RA
- RA 8, PA 32/14, Wp 10, SVO2 78%, CO 5.3
- Cr doubled
Scenario 4

Day 1
- Flows 3.9L on p8
- BP 92/78, HR 102, temp 37
- 99% sat on 1L
- RA 12, PA 41/16, WP 14, SVO2 68%, Hg 9, CO 4.3
- No gtts

Day 3
- Flows 2.0 L on P4
- BP 90/70, HR 110
- 99% sat on RA
- RA 16, PA 32/14, Wp 10, SVO2 58%, CO 3.8
- Cr doubled
Mixed results: ?negative study or unfair study design?

<table>
<thead>
<tr>
<th>Selected Device Trials</th>
<th>Trial Type</th>
<th>Indication</th>
<th>Sample Size</th>
<th>Primary End Point</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>IABP</td>
<td>Randomized Elective IABP vs medical</td>
<td>High-risk PCI</td>
<td>N = 301</td>
<td>MACCE&lt;sup&gt;a&lt;/sup&gt;</td>
<td>No difference (15.2% IABP vs 16% medical; P = .85)</td>
</tr>
<tr>
<td>Shock II trial&lt;sup&gt;5&lt;/sup&gt;</td>
<td>Randomized IABP vs medical</td>
<td>AMI with cardiogenic shock</td>
<td>N = 600</td>
<td>30-d all-cause mortality</td>
<td>No difference (39.7% IABP vs 41.3% medical; P = .69)</td>
</tr>
<tr>
<td>Impella</td>
<td>Randomized Impella 2.5 vs IABP</td>
<td>Cardiogenic shock</td>
<td>N = 25</td>
<td>Change of CI from baseline to 30 min</td>
<td>Difference seen (Impella CI change = 0.49 ± 0.46 L/min/m²; IABP change CI = 0.11 ± 0.31 L/min/m²; P = .02)</td>
</tr>
<tr>
<td>Protect II trial&lt;sup&gt;7&lt;/sup&gt;</td>
<td>Randomized Impella 2.5 vs IABP</td>
<td>High-risk PCI&lt;sup&gt;b&lt;/sup&gt;</td>
<td>N = 452</td>
<td>30-d incidence of major adverse events</td>
<td>No difference (35.1% for Impella vs 40.1% for IABP; P = .22)</td>
</tr>
<tr>
<td>RECOVER-I&lt;sup&gt;8&lt;/sup&gt;</td>
<td>Nonrandomized prospective single arm (Impella 5.0)</td>
<td>Postcardiotomy shock</td>
<td>N = 16</td>
<td>30-d death or stroke survival to implantation of next therapy</td>
<td>13% stroke or death, 7% bridge to other therapy, and survival to 30 d was 94%</td>
</tr>
<tr>
<td>Recover Right trial&lt;sup&gt;9&lt;/sup&gt;</td>
<td>Nonrandomized prospective single arm (Impella RP)</td>
<td>RV failure within 48 h post-LVAD, postcardiotomy, or MI shock</td>
<td>N = 30</td>
<td>30-d survival, hospital discharge, or bridge to next therapy</td>
<td>Difference seen 73% for Impella RP vs 58.3% benchmark reference&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>TandemHeart</td>
<td>Randomized TandemHeart vs IABP</td>
<td>AMI with cardiogenic shock</td>
<td>N = 41</td>
<td>Cardiac power index within 2 h after device placement</td>
<td>Difference seen (Tandem Heart CPI change from 0.22 to 0.37 W/m² compared with IABP from 0.22 to 28 W/m²; P = .004 intergroup comparison)</td>
</tr>
<tr>
<td>TandemHeart vs IABP&lt;sup&gt;11&lt;/sup&gt;</td>
<td>Randomized TandemHeart vs IABP</td>
<td>Cardiogenic Shock</td>
<td>N = 42</td>
<td>Superior hemodynamic benefit</td>
<td>Difference seen greater decrease in PCWP and increase in CI and mean BP (TandemHeart vs IABP)</td>
</tr>
</tbody>
</table>