Bedside Mechanical Assist Devices for Severe Heart Failure/Cardiogenic Shock: When, Which One and How?

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New York Presbyterian

June 22, 2020
### Interagency Registry of Mechanically Assisted Circulatory Support (INTERMACS)

<table>
<thead>
<tr>
<th>Profile</th>
<th>Description</th>
<th>Temporary circulatory support (TCS)</th>
<th>Arrhythmia (A)</th>
<th>Frequent flyer (FF)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Critical cardiogenic shock</td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>2.</td>
<td>Progressive decline on inotropic support</td>
<td>X</td>
<td></td>
<td>X</td>
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<tr>
<td>3.</td>
<td>Stable but inotrope dependent</td>
<td>X (in hosp)</td>
<td>X</td>
<td>X (if home)</td>
</tr>
<tr>
<td>4.</td>
<td>Resting symptoms home on oral therapy</td>
<td>X</td>
<td></td>
<td>X</td>
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<tr>
<td>5.</td>
<td>Exertion intolerant</td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>6.</td>
<td>Exertion limited</td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>7.</td>
<td>Advanced NYHA Class III symptoms</td>
<td>X</td>
<td></td>
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</table>

NYHA, New York Heart Association Classification.
Traditional Definition of Cardiogenic Shock

Hypotension
- SBP < 90 mmHg for > 30 min
- Need for vasopressors, inotropes, or mechanical circulatory support to maintain SBP > 90 mmHg

Diminished cardiac output
- Cardiac index < 2.0 - 2.2 liters/min/m2

Elevated filling pressures
- Pulmonary capillary wedge pressure > 15 mmHg
- Central venous pressure > 15 mmHg

Evidence of end organ dysfunction
New Pyramidal Scheme of CS

SCAI Stages of Cardiogenic Shock

Adapted from the SCAI Clinical Expert Consensus Statement on the Classification of Cardiogenic Shock. Endorsed by ACC, AHA, SCDM, and BTS.

**EXTREMS**
A patient being supported by multiple interventions who may be experiencing cardiac arrest and/or ECMO.

**DETERIORATING**
A patient who fails to respond to initial interventions. Similar to stage 2a, getting worse.

**CLASSIC**
A patient presenting with hypoperfusion requiring intervention beyond volume resuscitation (e.g., CPR, pressor, or mechanical support including ECMO). These patients typically present with relative hypotension.

**BEGINNING**
A patient who has clinical evidence of relative hypoperfusion without hypotension.

**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 or chronic heart failure.
Performance of the SCAI Classification Scheme

- 10,004 patients who were admitted to the Mayo Clinic CCU from 2007 – 2015 (all comers)
  - 43.1% had acute coronary syndrome
  - 46.1% had heart failure
  - 12.1% with cardiac arrest

- SCAI class was assigned based on data from the hospitalization

- Each higher SCAI shock stage was associated with a step-wise increase in hospital mortality

Jentzer et al. JACC 2019
• Study of 1007 consecutive patients presenting with cardiogenic shock or large MI between 2009 and 2017

• Higher SCAI classification was associated with lower 30-day survival
Goals of Treatment

1. Restore adequate end-organ perfusion
   - Break downward spiral
   - CO, MAP, oxygenation

2. Ventricular unloading
   - Treat/prevent pulmonary edema
   - Minimize myocardial o2 demand
   - Prevent/minimize remodeling

Rihal CS et al, J Am Coll Cardiol. 2015;65:e7-e26
Variable Hemodynamic Response To Therapies

- Degree of LV and/or RV compromise
- Short term recoverability of LV/RV function
- SVR and PVR
- Volume Status
- Background medical therapy
- Duration of cardiomyopathy
Treatment Approaches: Pharmacologic

- Myocardial demand
- Ischemia
- Atrial Arrhythmia
- Ventricular Arrhythmia

- PCWP
- Hypoxia
- RV Afterload
- Peripheral Vasoconstriction
- Splanchnic Vasoconstriction

Potential Risks

Inotropy/Chronotropy

CO

LV Afterload

SVR
Rising Use of Acute MCS

**TABLE 3** Trends in Short-Term Mechanical Circulatory Support Outcomes

<table>
<thead>
<tr>
<th>Outcome</th>
<th>2004-2007</th>
<th>2008-2011</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length of stay, days</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AMI</td>
<td>18.5 ± 27.3</td>
<td>14.3 ± 19.5</td>
<td>0.09</td>
</tr>
<tr>
<td>CAD</td>
<td>10.3 ± 10.4</td>
<td>7.2 ± 10.4</td>
<td>0.06</td>
</tr>
<tr>
<td>CHF</td>
<td>58.7 ± 65.5</td>
<td>32.3 ± 35.9</td>
<td>0.002</td>
</tr>
<tr>
<td>HVD</td>
<td>11.6 ± 12.7</td>
<td>17.2 ± 22.4</td>
<td>0.44</td>
</tr>
<tr>
<td>Other</td>
<td>25.4 ± 41.2</td>
<td>25.6 ± 31.0</td>
<td>0.81</td>
</tr>
<tr>
<td>Disposition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Routine (home or self-care)</td>
<td>31.7</td>
<td>47.5</td>
<td>0.001</td>
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<tr>
<td>Home health care</td>
<td>23.0</td>
<td>18.4</td>
<td>0.22</td>
</tr>
<tr>
<td>Transfer</td>
<td>45.3</td>
<td>34.1</td>
<td>0.019</td>
</tr>
<tr>
<td>Mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AMI</td>
<td>41.1</td>
<td>33.4</td>
<td>0.027</td>
</tr>
<tr>
<td>CAD</td>
<td>41.4</td>
<td>33.5</td>
<td>0.09</td>
</tr>
<tr>
<td>CHF</td>
<td>37.1</td>
<td>15.4</td>
<td></td>
</tr>
<tr>
<td>HVD</td>
<td>29.6</td>
<td>34.2</td>
<td>0.54</td>
</tr>
<tr>
<td>Other</td>
<td>43.3</td>
<td>55.3</td>
<td></td>
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<tr>
<td>Mortality with cardiogenic shock</td>
<td>51.6</td>
<td>43.1</td>
<td>0.012</td>
</tr>
<tr>
<td>Cost</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AMI</td>
<td>$150,187</td>
<td>$116,851</td>
<td>0.011</td>
</tr>
<tr>
<td>CAD</td>
<td>$142,176</td>
<td>$97,134</td>
<td>0.015</td>
</tr>
<tr>
<td>CHF</td>
<td>$120,699</td>
<td>$66,277</td>
<td>0.015</td>
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<tr>
<td>HVD</td>
<td>$217,144</td>
<td>$190,612</td>
<td>0.010</td>
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<tr>
<td>Other</td>
<td>$143,606</td>
<td>$133,733</td>
<td>0.82</td>
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<tr>
<td>Cost with cardiogenic shock</td>
<td>$157,726</td>
<td>$162,811</td>
<td>0.86</td>
</tr>
</tbody>
</table>

LV Unloading

- LV Unloading is defined as the reduction of total mechanical power expenditure (PVA·HR) of the ventricle which correlates with reductions in myocardial oxygen consumption and hemodynamic forces that lead to ventricular remodeling.

PVA = pressure-volume area
HR = heart rate
Pressure Volume Loop

![Pressure Volume Loop Diagram]

- **LVP (mmHg)**: Left Ventricular Pressure
- **LV Vol (ml)**: Left Ventricular Volume
- **SV**: Stroke Volume
- **ESPVR**: End-Systolic Pressure-Volume Relation
- **EDPVR**: End-Diastolic Pressure-Volume Relation
- **ESV**: End-Systolic Volume
- **EDV**: End-Diastolic Volume
- **Aortic Valve Opening**: Points 1 and 2
- **Aortic Valve Closing**: Points 3
- **Mitral Valve Opening**: Points 4
- **Mitral Valve Closing**: Points 1 and 4
Pressure Volume Loop

ESPVR – End Systolic Pressure Volume Relationship

(A) Normal pressure-volume loop (PVL), is bounded by the end-systolic pressure-volume relationship (ESPVR) and end-diastolic pressure-volume relationship (EDPVR). ESPVR is approximately linear with slope end-systolic elastance (Ees) and volume-axis intercept (Vo). Effective arterial elastance (Ea) is the slope of the line extending from the end-diastolic volume (EDV) point on the volume axis through the end-systolic pressure-volume point of the loop.

(B) Slope of the Ea line depends on total peripheral resistance (TPR) and heart rate (HR), and its position depends on EDV. (C) The ESPVR shifts with changes in ventricular contractility, which can be a combination of changes in Ees and Vo. Changes in contractility can be indexed by V120, the volume at which the ESPVR intersects 120 mm Hg. ESV = end-systolic volume; LV = left ventricular.
Following initiation of MCS. Both components of device effects will be discussed later.

Finally, use of the theories of ventricular mechanics detailed earlier within the context of a comprehensive cardiovascular simulation (9,10) facilitates illustration and comparison of the hemodynamic effects of different forms of MCS. The simulation we used has been detailed, can be used to understand the physiology of MCS, and has been validated to a certain degree pre-clinically (15). Other aspects of validation and limitations of the simulation have also been detailed previously (15–17). Note that the response of a given patient to MCS must account for baseline pre-load, afterload LV contractility, and the flow rate of the MCS pump. For simplicity, subsequent comparisons keep these factors constant. Importantly, the basic principles to be discussed apply across a wide range of conditions.

**RA-TO-ARTERIAL CIRCULATORY SUPPORT.**

Extra-corporeal venoarterial membrane oxygenation (ECMO), also referred to as extracorporeal life support, utilizes a pump with the capacity to assume responsibility for the entire cardiac output and a gas exchange unit for normalizing pCO₂, pO₂, and pH. However, strictly on a hemodynamic basis, the use of this circuit configuration can cause significant increases in LV pre-load and, in some cases, pulmonary edema. This is illustrated in Figure 4A, which depicts PVLs in a case of cardiogenic shock due to profound, irreversible LV dysfunction. Baseline cardiogenic shock conditions (PVL in black) have a high LV EDP, low pressure generation, low SV, and low ejection fraction. As ECMO flow is initiated and increased stepwise from 1.5 to 3.0 to 4.5 l/min, the primary hemodynamic effect is increased LV afterload pressure and effective Ea. If TPR and LV contractility are fixed, the only way for the LV to overcome the increased afterload is via the Starling mechanism, and blood accumulates in the LV. Consequently, LV EDP, LA pressure, and pulmonary capillary wedge pressure (PCWP) increase, and the PVL becomes increasingly narrow (decreased native LV SV) and taller (increased afterload pressure), and shifts rightward and upward along the EDPVR. Because the EDPVR is nonlinear, large increases in LV EDP may cause only subtle increases in LV EDV. An echocardiogram showing a persistently closed aortic valve during ECMO would also signify a state of maximal LV loading and high PCWP. These increases in LV pre-load and PCWP are detrimental to blood oxygen saturation coming from the lung and markedly increase myocardial oxygen demand (increased PVA), which can worsen LV function, especially in the setting of acute myocardial ischemia or infarction.

These responses to ECMO can be modulated by secondary regulatory factors that influence either TPR or LV contractility. TPR can be reduced naturally by the baroreceptors, pharmacologically (e.g., nitroprusside), or mechanically (e.g., by intra-aortic balloon pumping). As illustrated in Figure 4B, a 50% reduction in TPR during ECMO markedly blunts the rise in LV EDP. Short-term improvements in LV function can also modulate the rise in PCWP. LV function can be improved during ECMO due to increased central aortic pressure, the improved coronary perfusion, normalization of blood oxygen content (improved oxygen delivery to the myocardium), and normalization of oxygen for:

**FIGURE 3** Myocardial Energetics Assessed on the Pressure–Volume Diagram

(A) Pressure–volume area (PVA) is the sum of the stroke work (SW) and potential energy (PE). (B) Myocardial oxygen consumption (MVO₂) is linearly correlated with PVA and is divided into 3 major components, as indicated in the figure. LV = left ventricular.
Most Common Devices Used in CS

- There are several devices for use in cardiogenic shock, each with a different risk profile and potential benefit to the patient.

- The biggest factors in choosing a device for patients is how much circulatory support is required and whether LV, RV or BIV failure is present.
Intraaortic Balloon Pump
Impact of IABP in CGS

Pressure-Volume

LVP and AoP

Small ↓ PCWP
Small ↑ CO
Lack of Hemodynamic Benefit of IABP in Acute MI with Cardiogenic Shock (IABP-Shock Trial)

Prondzinsky R et al. Shock 2012
Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

$P = 0.92$ by log-rank test.

Mortality (%) vs Days since Randomization

V-A ECMO
Impact of RA→Ao MCS (ECMO) on Hemodynamics and Energetics

- ↑ Afterload
- ↑ Preload
- ↑ AoP
- ↑ LVP
Impact of RA→Ao MCS (ECMO) on Hemodynamics and Energetics

↑ Afterload
↑ Preload

↑ PVA
↑ MVO2
ECMO – The Advantages

- Easily deployed (at bedside if needed)
- Significant hemodynamic support with flows of 5+L possible
- Biventricular support
  - Circulatory support is maintained independent of ventricular function
  - In extreme examples, flow is maintained even in VF
- Acute MCS device which supports gas exchange
- Less hemolysis
Inadequate ventricular unloading

- ECMO maintains end organ perfusion but may actually increase cardiac work
- This may cause pressure overload which can lead to LV distention and pulmonary edema

Large Cannula size (V=23F, A=15-17F)

- Bleeding is common
- Ischemic complications cause major morbidity

Resource intensive
Percutaneous Pumps

Transvalvular

Impella Family of Devices
2.5/4.0/5.0/RP

Transseptal

TandemHeart LA→Ao Pump

Thoratec PHP, Tranvalvular Pump with Expandable Cage (Experimental)
Impact of LA→Ao MCS in CGS

↑BP
↑Total CO
↓PCWP
TandemHeart pVAD in refractory cardiogenic shock

- Baseline patient characteristics:
  - 117 severe cardiogenic shock patients from 2003-08
  - All patients refractory to IABP and multiple vasopressors
  - 48% of patients undergoing CPR at time of implant
  - 68% ischemic, 32% non-ischemic cardiomyopathy

Kar B, et al. JACC 2011
Impact of LV→Ao MCS on Hemodynamics and Energetics

- ↓ Peak LVP
- ↓ Preload
- ↑ AoP
- ↓ LVP
- LV-Ao Uncoupling
Impact of LV→Ao MCS on Hemodynamics and Energetics

- **Peak LVP**
- **Preload**
- **PVA**
- **MVO2**
LVAD Unloading and Decoupling

A

B

C

D

E

LVP (mmHg)

Pressure (mmHg)

Pressure (mmHg)

Pressure (mmHg)
TABLE 1 Baseline Characteristics

<table>
<thead>
<tr>
<th></th>
<th>pMCS (n = 24)</th>
<th>IABP (n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>58 ± 9</td>
<td>59 ± 11</td>
</tr>
<tr>
<td>Male</td>
<td>18/24 (75)</td>
<td>20/24 (83)</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>25 (23–26)</td>
<td>26 (25–27)</td>
</tr>
<tr>
<td>Hemodynamic variables before randomization</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>81 ± 21</td>
<td>83 ± 28</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
<td>66 ± 15</td>
<td>66 ± 15</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>81 ± 17</td>
<td>84 ± 19</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>58 ± 22</td>
<td>57 ± 13</td>
</tr>
<tr>
<td>Medical therapy before randomization</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Catecholamines or inotropes</td>
<td>24/24 (100)</td>
<td>22/24 (92)</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>24/24 (100)</td>
<td>24/24 (100)</td>
</tr>
<tr>
<td>Cardiac arrest before randomization</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Witnessed arrest</td>
<td>22/24 (92)</td>
<td>17/20 (85)</td>
</tr>
<tr>
<td>First rhythm VT/VF</td>
<td>22/24 (92)</td>
<td>17/20 (85)</td>
</tr>
<tr>
<td>Time till return of spontaneous circulation, min</td>
<td>21 (15–46)</td>
<td>27 (15–52)</td>
</tr>
<tr>
<td>Traumatic injuries at admission</td>
<td>5/24 (21)</td>
<td>2/24 (8)</td>
</tr>
<tr>
<td>Blood values on admission*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactate, mmol/l</td>
<td>7.5 ± 3.2</td>
<td>8.9 ± 6.6</td>
</tr>
</tbody>
</table>
TABLE 4 Clinical and Functional Outcomes

<table>
<thead>
<tr>
<th></th>
<th>pMCS (n = 24)</th>
<th>IABP (n = 24)</th>
<th>p Value</th>
<th>Hazard Ratio With pMCS (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30-day all-cause mortality</td>
<td>11 (46)</td>
<td>12 (50)</td>
<td>0.32</td>
<td>0.96 (0.42-2.18)</td>
</tr>
<tr>
<td>6-month all-cause mortality</td>
<td>12 (50)</td>
<td>12 (50)</td>
<td>0.32</td>
<td>1.04 (0.40-2.62)</td>
</tr>
</tbody>
</table>

* Mortality is shown as Kaplan-Meier estimates.

Percutaneous Mechanical Circulatory Support Versus Intra-Aortic Balloon Pump in Cardiogenic Shock After Acute Myocardial Infarction - IMPRESS

RV Support – Impella RP

Anderson MB et al. JHLT 2015
Impella Complications
Impella Complications

The Evolving Landscape of Impella® Use in the United States Among Patients Undergoing Percutaneous Coronary Intervention with Mechanical Circulatory Support


Originally published 17 Nov 2019 | https://doi.org/10.1161/CIRCULATIONAHA.119.044007 | Circulation. 0: null

Impella Support for Acute Myocardial Infarction Complicated by Cardiogenic Shock

Matched-Pair IABP-SHOCK II Trial 30-Day Mortality Analysis

Benedikt Schrage, Karim Ibrahim, Tobias Loehn, Nikos Werner, Jan-Matte Sinning, Federico Pappalardo, Marina Pieri, Carsten Skurk, Alexander Lauten, Ulf Landmesser, Ralf Westenfeld, Patrick Horn, Matthias Paechsinger, Dennis Eckner, Raphael Twerenbold, Peter Nordbeck, Tim Salinger, Peter Abel, Klaus Empen, Mathias C. Busch, Stephan B. Felix, ... Show all Authors

Originally published 5 Dec 2018 | https://doi.org/10.1161/CIRCULATIONAHA.118.036614 | Circulation. 2019;139;1249–1258

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Impella (n = 237)</th>
<th>IABP-SHOCK II (n = 237)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-Day Mortality</td>
<td>48.5%</td>
<td>46.4%</td>
<td>0.64</td>
</tr>
<tr>
<td>Severe or Life-threating Bleeding</td>
<td>8.5%</td>
<td>3.0%</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Peripheral Vascular Complications</td>
<td>9.8%</td>
<td>3.8%</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Figure 3. Association of Impella versus IABP use with clinical outcomes.
Percutaneous LVAD vs. IABP: Hemodynamic Effects

RA → Ao MCS + LV → Ao MCS (ECPELLA)

↑ Afterload
↑ Preload

AoP
LVP

Pressure-Volume

Time Plots
Centrimag BiVAD
<table>
<thead>
<tr>
<th>Survival</th>
<th>Overall</th>
<th>FMM*</th>
<th>PCS†</th>
<th>GF‡</th>
<th>RVF</th>
<th>ILVAD§</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>143</td>
<td>71</td>
<td>37</td>
<td>22</td>
<td>13</td>
<td>53.8</td>
</tr>
<tr>
<td>30-Day Survival (%)</td>
<td>61.5</td>
<td>69.0</td>
<td>43.2</td>
<td>72.7</td>
<td>53.8</td>
<td></td>
</tr>
<tr>
<td>Survival to Discharge (%)</td>
<td>53.9</td>
<td>62.0</td>
<td>35.1</td>
<td>63.6</td>
<td>46.2</td>
<td></td>
</tr>
<tr>
<td>1-Year Survival (%)</td>
<td>45.8</td>
<td>55.9</td>
<td>31.0</td>
<td>40.0</td>
<td>40.0</td>
<td></td>
</tr>
</tbody>
</table>

**Destination**

| Myocardial Recovery (%)         | 31.5    | 25.4 | 32.4 | 45.5| 38.5|
| Implantable VAD (%)             | 16.8    | 23.9 | 10.8 | 16.7| 7.7 |
| Heart Transplant (%)            | 16.1    | 23.9 | 2.7  | 18.2| 7.7 |

*Failure of Medical Management  §Right Ventricular Failure
†Postcardiomyopathy Shock       post
‡Graft Failure                   implantable left ventricular assist device
Minimally invasive CentriMag integrated with ECMO
Insertion, flexible configuration, combination with an oxygenator, connected to the CentriMag system. These features allow easy in-rotational speed of 5500 rpm. Various types of cannulas can be used. BiVAD: biventricular assist device; Ec-VAD: extracorporeal ventricular assist device; LVAD: left ventricular assist device.

**Early Outcome**

<table>
<thead>
<tr>
<th>Table 3: Early outcomes and next destinations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BiVAD</strong> (n = 90)</td>
</tr>
<tr>
<td>---------------------</td>
</tr>
<tr>
<td><strong>Support duration, days</strong></td>
</tr>
<tr>
<td><strong>Major morbidity during support, n (%)</strong></td>
</tr>
<tr>
<td>Major bleeding</td>
</tr>
<tr>
<td>Delayed sternal closure</td>
</tr>
<tr>
<td>Stroke</td>
</tr>
<tr>
<td>Mortality on device, n (%)</td>
</tr>
<tr>
<td>Multi-organ failure/sepsis</td>
</tr>
<tr>
<td>Stroke</td>
</tr>
<tr>
<td>Other</td>
</tr>
<tr>
<td><strong>Thirty-day mortality, n (%)</strong></td>
</tr>
<tr>
<td><strong>Destination, n (%)</strong></td>
</tr>
<tr>
<td>Recovery (weaned off device)</td>
</tr>
<tr>
<td>Heart transplant</td>
</tr>
<tr>
<td>Durable VAD</td>
</tr>
<tr>
<td>Pulsatile LVAD</td>
</tr>
<tr>
<td>Continuous-flow LVAD</td>
</tr>
<tr>
<td>Continuous-flow BiVAD</td>
</tr>
</tbody>
</table>

BiVAD: biventricular assist device; Ec-VAD: extracorporeal ventricular assist device; LVAD: left ventricular assist device; VAD: ventricular assist device.
TEAM – BASED is not limited to the choice of device!

Figure 3. Inhospital survival rates as a function of shock onset to MCS implantation.
Conclusions

- Mortality from CS remains unacceptably high
- A new classification scheme for CS may provide important insights and a framework for effective communication of severity of CS
- The PA catheter remains an important tool in assessing and monitoring CS patients
- Acute MCS now forms the cornerstone of treatment of severe CS
- The “Shock Team” approach is important to rapid recognition, triage and treatment of CS patients and recent evidence supports its implementation can improve outcomes in CS
Thank you