Cardiogenic Shock: Updates and New Treatment Strategies

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Disclosures

S. Emani:
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- Abbott
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- CHF Solutions
- Cardionomics
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R. Magorien:
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This presentation will include off-label therapies
Cardiogenic Shock (CS) Outcomes

- 81% mortality with untreated/inadequately treated CS
- 27-51% mortality with appropriately treated CS
- **Acute MI** is the most common cause of CS
- Complex presentations and causes make treatment challenging
- Underscores need for improved shock strategies

What is Shock?

A clinical scenario with signs of end-organ hypoperfusion

Related to multiple factors:

- Perfusion gradients
- Tissue oxygen supply and demand
- Neurohormonal imbalances

Cardiogenic Shock

- Shock arising from a primary cardiac cause
- Major manifestation is low cardiac output

- No universal cut-off for CO/CI
  - <1.8
  - <2
  - <2.2

  Shock has been observed with all ranges

- No EF cut-off for cardiogenic shock
Cardiac Power Output

- Analysis from SHOCK trial showed correlation to mortality
- Better than other hemodynamic parameters
- CPO ≤ 0.53 W had sensitivity and specificity of 0.66
- Clinically, CPO < 0.6 used as a cut point

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


OSU Working Definition of Cardiogenic Shock

**HYPOTENSIVE CARDIOGENIC SHOCK:**
SBP < 90 mmHg for > 30 minutes and evidence of decreased organ perfusion or use of catecholamines to maintain SBP > 90 mmHg
  - Especially if >1 inotrope or vasopressor

**NON-HYPOTENSIVE SHOCK:**
Low cardiac index in setting of normal systemic pressures & high SVR
Defining Cardiogenic Shock

Definition of CS is not dependent on invasive hemodynamics

However – use of invasive hemodynamics is helpful:

- Characterize hemodynamic profile
- Allows better selection of initial therapies
- Allows monitoring of response to therapies

Use of a PA catheter is encouraged!

Decline in use of PA catheters after ESCAPE

Mortality Trends with PA Catheter

Timing is Everything

Early recognition & implementation of therapy improves outcomes

Factors associated with increased survival:

- **Recognition** within 12 hours of CS
- Use of **invasive hemodynamics**
- **Early therapies** to stabilize hemodynamics
- Utilization of “**shock networks**” to escalate care
Timing is Everything

Learning from the Past

- Prior to 2006 Ohio State had no specific STEMI protocol
- “STEMI” pager carried by interventional fellows (initiated in 2004)
Key strategies associated with reduced D2B times

<table>
<thead>
<tr>
<th>STRATEGIES</th>
<th>MINUTES SAVED</th>
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<tbody>
<tr>
<td>Activate lab with EM Physicians (23% do this)</td>
<td>8.2 minutes</td>
</tr>
<tr>
<td>Activate w/ single call from ED to operator (14%)</td>
<td>13.8 minutes</td>
</tr>
<tr>
<td>Activate on pre-hospital ECG while patient enroute (9%)</td>
<td>15.4 minutes</td>
</tr>
<tr>
<td>Expectation that cath team arrive 20 - 30 minutes (13%)</td>
<td>19.3 minutes</td>
</tr>
<tr>
<td>Provide real time feedback to ED/lab (42%)</td>
<td>8.6 minutes</td>
</tr>
<tr>
<td>Attending cardiologist always on site (4%)</td>
<td>14.6 minutes</td>
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Bradley et al, NEJM, 2006

Ohio State Experience

In 2006, Developed *in-house* Algorithm for STEMI Care
- Dedicated STEMI Hotline
- Overhead “STEMI Alert”
- Allows ED physician to activate cath lab
- In-house team assembles in cardiac catheterization lab immediately (MD’s, RN’s, RT’s, Pharmacy)
- Interventional Cardiology Team assembled within 20-25 minutes
“OSU STEMI Alert” 366-8111

Red Phone, OSU Transfer Center

- ED or EMS Activation of the OSU Cath Lab
- 3-way call ED (or EMS), OSU, MedFlight
- In-House cath lab team immediately available
- Interventional Cardiology team arrives within 20 minutes during off hours and procedure starts upon arrival
- D2B average for August 2019 was 27.5 minutes, including off hour cases

Ohio State….Where we are:

- Activate Lab with ED Physicians
- Field Activations (EMS)
- Activate Lab with Single Phone Call
- Activate based on Pre-Hospital EKG
- Expect Team to arrive in 20-30 min
- Provide real-time feedback
- Attending Cardiologist on site
- D2B 40.64 min average FY19
- D2D2B 101.82 min average FY19
- EMS Alerts D2B 22.4 min average FY19
- Education Efforts
  - EMS
  - Nursing
  - MD/CME
- Quality Assurance
  - Immediate feedback
  - D2B committee
Pathophysiology of Cardiogenic Shock
A Cardio-Metabolic Cascade

Continuum of Cardiogenic Shock

Opportunity for meaningful intervention
Salvage Therapy
Treating Cardiogenic Shock

SCAI Classification of Shock

- **Stage A: At Risk**. A patient who is not currently experiencing signs or symptoms of cardiogenic shock, but is at risk for its development. These patients may include those with acute myocardial infarction, acute and/or acute on chronic heart failure symptoms.

- **Stage B: Beginning**. Cardiogenic Shock. A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.

- **Stage C: Classic**. Cardiogenic Shock. A patient that manifests with hypoperfusion that requires intervention (inotropes, pressor, or mechanical support, ECMO) beyond bolus resuscitation to reestablish perfusion. These patients typically present with relative hypotension.

- **Stage D: Deteriorating or Down**. A patient that is similar to category C but is getting worse. They have failure to respond to initial interventions.

- **Stage E: Extremis**. A patient with circulatory collapse, frequently (but not always) in refractory cardiac arrest with ongoing cardiopulmonary resuscitation (CPR) or are being supported by multiple simultaneous acute interventions including ECMO-facilitated CPR. These are patients with multiple clinicians at bedside laboring to address multiple simultaneous issues related to the lack of clinical stability of the patient.

Treating Cardiogenic Shock

- Treat acute/reversible causes

Escalating Support

- Inotropes
- IABP
- Impella CP
- Impella 5.0 Tandem Heart
- CentriMag
- ECMO ±RV Support
- ±LV Vent

Levels of Flow Support

Escalating Support

- IABP [0.5-1.5 L/min] (CO ↑ 20-30%)
- Impella CP [~3 L/min] Impella 5.0 [~5 L/min]
- Tandem Heart [~5 L/min]
- CentriMag [10 L/min]
- ECMO [7-10 L/min]
IABP

- "Traditional" tMCS solution with mixed evidence

- IABP-SHOCK-II Trial
  - IABP use in AMI
  - No improvement in 30 day or 1 year mortality vs medical therapy
  - High cross-over rate, no hemodynamics, no prescriptive algorithm on IABP use

- Clinically, IABP still felt to be useful in some situations


IABP

Timing May Be Everything

Follow up study showed improvement in 30 day mortality with IABP if inserted <1 hour of shock onset

- Single-center, retrospective study
- No difference between ACS and non-ACS patients

Shift to metabolic shock profile when used late?

IABP Benefits

- Relatively easy to insert
  - Including alternative access options
- Relatively easy to manage / Lots of experience
- Anticoagulation may be less challenging to manage
  - Including option for minimal/no anticoagulation
- Relatively cheaper than other devices
- By fair comparison, no strong mortality benefit with other devices

IABP Drawbacks

- Limited hemodynamic support
  - Better for SVR reduction?
- Limited by heart rate and rhythm
- Especially with HR >120
- Univentricular support
  - No “RV” IABP
- Aortic valve pathology can complicate/limit utility
Considerations with other tMCS options

- Presence of LV thrombus & AS limits pVAD (Impella)
- TandemHeart requires trans-septal approach (in LV bypass configuration)
- Sheath/cannulae sizes increase with more flow support
  - May require surgical insertion/removal
- Hemolysis is a concern with many devices
- Anticoagulation is more critical, and more difficult to maintain
- FDA labeling for most tMCS is short term (i.e. <6 hours)

Choices, choices

- Minimal data to suggest superiority of a single strategy
- Evaluation of etiology, concurrent therapies, and end-goals are key
- Use of hemodynamics can be useful
- Several published algorithms available
OSU Cardiogenic Shock Algorithm

Shock Suspected

Notify Shock Team

Massive Pulmonary Embolism

1. OSU Cardiogenic Shock Algorithm
2. Hypovolemia (RA < 15 mmHg or PCWP or LVEDP < 18)
3. Predominantly RV Failure (RA > 15 mmHg, PCWP or LVEDP < 18)
4. Predominantly LV Failure (RA ≤ 15 mmHg, PCWP or LVEDP > 18)
5. Biventricular Failure (RA > 15 mmHg, PCWP or LVEDP > 18)
6. Temporary left-sided support
7. Temporary right-sided support

Testing

- Echocardiogram – evaluate function/structure
- PA catheter – leave in (calculate CI)
- Coronary angiogram ± revascularization and LVEDP
- Labs: CBC, electrolytes, creatinine, LFT, ABG with lactate, cardiac troponin level as appropriate
- Chest X-ray

CI > 2.2 consider non-cardiac etiology and intra-cardiac shunt

CI = 2.27

Hypovolemia and/or persistent VT/VF

Ultrafiltration and/or LV ECMO

Hypoxemia

CI ≤ 1.5

Predominantly LV Failure (RA ≤ 15 mmHg, PCWP or LVEDP > 18)

CI = PaPi > 1.57

Temporary left-sided support

For evaluation hemodynamic status in 20 minutes

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Improving Outcomes in Cardiogenic Shock

Ways to improve outcomes:
- Use algorithmic approach
- Form a Cardiogenic Shock Team

Single Center Experience (Fairfax, VA)
- 2016 – 47% survival (30-day)
- 2017 – 58% survival
- 2018 – 77% survival
Summary

- Cardiogenic Shock can lead to poor outcomes
- Cardiogenic Shock is a complex, dynamic process

- Early recognition is paramount
  - Hemodynamic profiles and shock stages can assist in characterization
  - Delayed recognition results in undesirable/unrecoverable situations

- Many therapeutic choices
  - Multiple factors can govern appropriate therapy

- Shock Teams and Shock Networks can improve outcomes
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Thank You

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