Cardiogenic Shock: Mechanical Support Devices

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Director Interventional Cardiology Fellowship Program
Director Advanced Structural Heart Program
Update in Cardiology at KHN

• Kettering Heart and Vascular Part of KPN
  • 48 Cardiologists from 6 groups integrated into one large unified group that will cover 8 hospitals
  • Subspecialized Pods
    • EP
    • CHF
    • Cardio Oncology
    • Interventional
      • CTO
      • Structural heart TAVR, Mitral Clip, Watchman
      • High Risk Intervention
      • Peripheral
    • Imaging
What’s Ahead

• Single Pod at each of outlying hospitals
• More Specialty Specific Care
• Advanced Technologies
• Systematized Network  wide approach.
• Integration with other specialties
  • CTS
  • Vascular Surg
  • Advanced HTN clinic with Nephro and Cardio together
  • Cardio-Onc
  • Pulm Htn with Cardio and Pulm.
Future Growth

• Support the Network approach
• Cardiology Satellite offices at Primary Care Hubs
• Consolidate some offices
• Building out Hybrid Cath lab
• Expand specialty offices into Cancer Annex
CASE 1

- 58 yo lady with a history of PAF, CVA, HTN, HFrEF with EF 25%, DM-2 presented with chest pain and SOB.

- Previous smoker, quit 30 yrs ago. No significant FHx of CAD

- Lotrel (amlodipine/benzepril), atenolol, atrovastatin, coreg, levemir, metformin, lyrica and effexor

- O/E she was AAOx3, BP 190/110 mmHg.

- EKG: sinus tachycardia at 101 bpm, normal axis, poor R-wave progression in the pericordial leads and non-specific ST-T wave changes. Initial torponins level was 2.4
EKG
▪ Stabilized and treated medically for NSTEMI + Hypertensive emergency.

▪ Next morning>> Cath lab.
• Diagnostic LHC revealed: 95% ostial LAD, 90% prox, 75% mid and 95% distal. LCx: dominant vessel with moderate disease distally. RCA: small non-dominant.

• Planned PCI to LAD as not best surgical candidate

• Started with angioplasty and ballooning – Didn’t tolerate Baloon.

• Subsequent Angiography showed 99% occlusion of proximal LAD and 100% distal LAD.

• low BP + pulseless + CPR multiple rounds of Epi >>> impella CP
• On impella she had 1 episode of Vfib – defibrillated + intubated, pulse was regained in <2 mins

• Balloon inflations in LAD causes drop in BP with more rounds of Epinephrine and CPR

• Surgery Present but did not want to take to OR with multiple problems

• ECMO placed > more hemodynamically stable > PCI to LAD with 3 stents
ECMO

- Transferred to UC
- ICU with ECMO for 3 days
- Eventually DC
- EF Improved to 40%
Cardiogenic Shock

• CS occurs in approx 5-8% of Patients hospitalized with ST elevation MI and 2.5% of NSTEMI. 50,000 cases per year in United States.

• MI with LV failure remains the most common cause of CS

• Must exclude complicating factors.
  • VSD, MV Chordal rupture, Free wall rupture, Massive PE, Hemorrhage, Infection or Bowel Ischemia, RV Dysfunction.

• Iatrogenic: BBs and ACE inhib, Diuretics, Volume overload.
Cardiogenic Shock

- CS is a state of end-organ hypoperfusion due to cardiac failure. The definition of CS includes hemodynamic parameters: persisting hypotension (Sys BP < 90 mm Hg or MAP 30 mm Hg lower than baseline) with severe reduction of CI < 1.8L min/M2.
  - Must have adequate or elevated filling pressure (LVEDP > 18)
- Usually presents with cool extremities, decreased UO, and/or altered Mental status.
- Can be mild to profound
- Mortality directly related to severity and time until corrected.
Figure 1. Current concept of CS pathophysiology.
Iatrogenic shock.
Cardiogenic Shock Diagnosis

- Clinical grounds
- PA Catheter
- Doppler Echocardiography
Range of LVEF in studies of heart failure and in the SHOCK trial.


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SIRS

• Recent research has suggested that the peripheral vasculature and neurohormonal and cytokine systems play a role in the pathogenesis and persistence of CS. Increased levels of IL6 TNF-a and rise of Cytokine levels. All impair endothelial function and suppress Myocardial function.

• Mi can cause SIRS and impaired perfusion of intestinal tract increases risk of transmigration of bacteria.

• Sooner tissue perfusion is restored the better prognosis.

• Mechanical support devices can help.

• Hospital Survivors have an excellent chance for long term survival and recovery. Including a good QOL.
Treatment

• Early recognition of cause
  • ECG/Echo/Cath/Enzymes and other tests if clinical suspicion.
• Early transfer to Advanced Treatment program
• Pressors and Ionotropes
• Adequate but not excessive volume resuscitation.
• Rapid revascularization. PCI or CABG
• Mechanical Support.
Algorithm for revascularization strategy in cardiogenic shock, from ACC/AHA guidelines. Whether shock onset occurs early or late after MI, rapid IABP placement and angiography are recommended.

Long-term follow-up of the SHOCK trial cohort. Early revascularization (ERV) is associated with sustained benefit.


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The intra aortic balloon pump (IABP) acutely improves systemic hemodynamics, augments coronary flow, increases coronary patency, and reduces myocardial oxygen demand.

Physiological and clinical data, led to a widespread use of the IABP in cardiogenic shock, refractory angina, primary PCI, high risk PCI, and complex CABG in the last decades.

Kern et al. Circulation. 1993;87:500–511
Ohman Circulation. 1994;90:792–799
Rihal et al. JACC. 2015; 65: e7-26
IABP

- >1 Million patients treated, low complication rate, Benchmark registry\(^1\)
- IABP therapy is the most widely used means of circulatory support for patients with hemodynamic instability resulting from LV Failure\(^2\)
- Well known effects of IABP include decrease in afterload that leads to an increase in stroke volume and cardiac output\(^2\)

\(^1\) Ferguson et al. JACC 2001;38:1456-1462
IABP PROs

• Small ateriotomy (7.5-8.0 F)
• Can be used in combination with other devices
• Short term outcome similar to other technologies
• Easy antithrombotic management
IABP CONs

• Minimum of cardiac function and a competent aortic valve required
• Modest ventricular unloading
• No outcome improvement in some studies
• Practice guidelines downgraded recommendations or even discouraged the use of IABP
BISIS-1: Elective IABP improves long-term survival after high-risk PCI

301 patients, high-risk PCI (EF<30% and extensive myocardium at risk) 12/2005 and 01/2009, 17 clinical sites UK

50% no planned IABP before PCI (N=151)

12% required bailout IABP (N=18)

50% planned IABP before PCI (N=150)

IABP-SHOCK II Trial

600 patients with cardiogenic shock (clinical assessment) were enrolled between June 2009 and March 2012 in 37 clinical sites in Germany.

- 301 randomized to IABP
  - 13 did not receive IABP
  - 287 primary PCI
  - 11 no revascularisation

- 299 randomized to control
  - 30 cross-over to IABP
  - 288 primary PCI
  - 8 no revascularisation

298 with 30 d follow-up

Primary endpoint: 30 d all-cause mortality

IABP-SHOCK II Trial

**Strength**

- Largest randomized shock trial
- 600 patients included within 32 month
- Contemporary CS treatment (>95 % revasc.)
- Follow-up: 99.2%

**Limitations**

- Underpowered for the primary endpoint
- No hemodynamic shock assessment
- 10% cross-over to IABP
- 83% of pts. received IABP post PCI
Impact of IABP-Timing in CS

Design


- OBJECTIVE: To evaluate the impact of IABP timing (before or after PCI) in STEMI complicated by cardiogenic shock.

- ENDPOINTS: Total mortality, MACCE, renal failure

Impact of IABP-Timing in CS
Clinical outcomes at 30 days

<table>
<thead>
<tr>
<th></th>
<th>IABP before PCI (n=49)</th>
<th>IABP after PCI (n=53)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>12 (25%)</td>
<td>29 (55%)</td>
<td>0.002</td>
</tr>
<tr>
<td>Emergency CABG</td>
<td>0 (0%)</td>
<td>5 (9%)</td>
<td>0.027</td>
</tr>
<tr>
<td>Cerebrovascular Events</td>
<td>4 (8%)</td>
<td>4 (8%)</td>
<td>0.908</td>
</tr>
<tr>
<td>MACCE</td>
<td>15 (31%)</td>
<td>32 (60%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Bleeding</td>
<td>12 (25%)</td>
<td>14 (26%)</td>
<td>0.824</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>9 (18%)</td>
<td>14 (26%)</td>
<td>0.331</td>
</tr>
</tbody>
</table>

Conclusions

• IABP can improve short term outcome in cardiogenic shock patients, as long as it is preceding PCI.

• IABP improves long term survival in high-risk PCI - possibly related to more stable procedural hemodynamics with more complete revascularisation.
Percutaneous LVADs

- Impella
Catheter diameter: 9Fr
Flow rate: up to 2.5 L/min
Activate Cardiac Cath Lab

Access

Assess Hemodynamics

Impella 2.5™ or CP®

Begin Weaning Catecholamines*

Acute MI?

Reassess Hemodynamics

Yes

Coronary Angiogram with PCI

No

BEST PRACTICE

Access:
1. Femoral arterial access using micropuncture with image guidance (ultrasound and/or fluoroscopy)
2. Angiography via 4F micropuncture dilator to confirm puncture site & vessel size
3. Place appropriately sized (5 or 6 Fr) arterial sheath
4. Obtain venous access (femoral or internal jugular)

If femoral arterial anatomy suitable and no contraindications, place, or escalate to (if IABP already in place), Impella 2.5 or Impella CP

* If consistent with overall hemodynamic management

Assess Hemodynamics: LVEDP or PAC
- If sustained hypotension (SBP < 90 mmHg) for > 30 min
  - Or
- CI < 2.2 with LVEDP or PCWP > 18 mmHg, consider mechanical circulatory support

Reassess Hemodynamics: PAC (if not done initially)
1. CPO = MAP × CO/451 W
2. PAPI = sPAP - dPAP/RA

CO, cardiac output; CPO, cardiac power output; dPAP, diastolic pulmonary arterial pressure; MAP, mean arterial pressure; PAC, pulmonary arterial catheter; PAPI, pulmonary artery pulsatility index; RA, right arterial pressure; sPAP, systolic pulmonary arterial pressure.


IMP-127-16
Reassess Hemodynamics via PAC prior to Discharge from the Cath Lab:
1. Cardiac Power Output (CPO) MAP × CO/451 W
2. Pulmonary Artery Pulsatility Index (PAPi) sPAP–dPAP/RA

- **CPO < 0.6**
  - PAPi ≤ 1.5
    - RV Dysfunction: Right-sided MCS (Impella RP®)
  - PAPi > 1.5
    - RV Preserved: Escalate MCS or consider transfer to LVAD/Transplant Center

- **CPO > 0.6**
  - PAPi ≤ 1.5
    - RV Dysfunction: Right-sided MCS (Impella RP®)
  - PAPi > 1.5
    - RV Preserved: Escalate MCS or consider transfer to LVAD/Transplant Center

**Persistent Hypoxemia?**
- PaO₂ < 55 on 100% FiO₂
  - VA or VV-ECMO: Recommend maintaining Impella® at low speed for LV decompression
  - Yes
  - No

- Admit to ICU to maximize supportive care and to actively **assess for myocardial recovery**

**RV Failure as defined by Recover Right¹:**
- CI < 2.2 L/min/m² (despite continuous infusion of ≥ 1 high dose inotrope, ie, da/dobutamine ≥ 10 µg/kg/min or equivalent) and any of the following:
  1. CVP > 15 mmHg, or
  2. CVP/PCWP or LAP ratio >0.63, or
  3. RV dysfunction on TTE (TAPSE score ≤14 mm)

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Assess for Myocardial Recovery
(At least every 12 hours)

**Improving**
Clinical, Echocardiographic & Hemodynamic parameters (concordant):
- ↑ Cardiac output
- ↑ CPO
- ↑ Urine output
- ↓ Lactate
- Inotropes low dose/discontinued
- Adequate Ramp test

Myocardial Recovery
Wean & Explant Impella® (After a minimum of 48h)

**Mixed picture**
Clinical, Echocardiographic & Hemodynamic parameters (discordant):
- Some parameters are improving
- Pressors lowered but not discontinued
- Fails “ramp test”

Inadequate Recovery
Continue Impella® support & frequent clinical reassessment
Failure to recover within 48-72 h, consider escalation or durable VAD/transplant

**Worsening**
Clinical, Echocardiographic & Hemodynamic parameters (concordant):
- ↓ Cardiac output
- ↓ CPO
- ↓ Urine output
- ↑ Lactate
- Inotrope dependent
- Absent pulsatility

No Recovery
Escalate (& Ambulate) or Transfer
See Escalate or Transfer Protocol

Worsening Clinical, Echocardiographic & Hemodynamic parameters (concordant):
- ↓ Cardiac output
- ↓ CPO
- ↓ Urine output
- ↑ Lactate
- Inotrope dependent
- Absent pulsatility

No Recovery
Escalate (& Ambulate) or Transfer
See Escalate or Transfer Protocol
No Recovery: Escalate or Transfer

Guidance by RHC Is Critical

CPO remains < 0.6 After 24 hours

PAPi ≤ 0.9 (RV Failure)
- Biventricular support with Impella RP® on the right side (transfer if not available)

0.9 < PAPi ≤ 1.5 (RV dysfunction)
- Consider supporting the right side with Impella RP (transfer if not available)

PAPi > 1.5 (acceptable RV function)
- Consider left-side escalation with Impella 5.0™ (transfer if not available)

Worsening / not improving clinical, echocardiographic & hemodynamic parameters (concordant):
- ↓ Cardiac output
- ↓ CPO
- ↓ Urine output
- ↑ Lactate
- Inotrope dependent
- Absent pulsatility

RV Failure as defined by Recover Right¹:
- CI < 2.2 L/min/m² (despite continuous infusion of ≥ 1 high dose inotrope, ie, da/dobutamine ≥ 10 µg/kg/min or equivalent) and any of the following:
  1. CVP > 15 mmHg, or
  2. CVP/PCWP or LAP ratio > 0.63, or
  3. RV dysfunction on TTE (TAPSE score ≤ 14 mm)

PAPi = sPAP - dPAP/RA.

IMP-127-16

CPO = (MAP × CO)/451.

ECMO
CENTRIFUGAL PUMP AND OXYGENATOR

- Guidelines specific for H1N1 pandemic
  - Use extracorporeal circuit for total support including diffusion membrane oxygenator and centrifugal pump
  - Heat exchanger to control blood and patient temperature at a specific level
  - Utilize patient monitoring for continuous inlet/outlet pressures, blood gas, saturation and $S\sqrt{O_2}$
ECLS SUPPORT
Partial Support

Full Support
WHEN FAILURE IS NOT AN OPTION...
THE GOAL OF HEMODYNAMIC STABILITY

• Provide adequate flow to every patient (>2.5 liters)
• Decrease work of the heart and stabilize MVO₂
• Unload and or decompress the entire heart
• Support end organ perfusion
• Provide adequate oxygen supply to entire body
• Optimize hemodynamic stability
• Temperature regulation
CARDIOHELP: SAFE, TRANSPORTABLE, RAPIDLY DEPLOYED

ECLS FOR ASSISTED PCI IN PROFOUND CARDIOGENIC SHOCK

- Study comparing shock with profound shock
  - Control Group: 120/920 (13.0%) STEMI
  - Pulmonary Edema, SBP<90mmHg
- Persistent hypotension with cardiac output
  - No response to fluid, requiring vasopressors
- Profound Shock
  - SBP<75mmHg on inotropes and IABP
  - Altered mental status and/or respiratory failure

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1 Sheu et al. Crit Care Med 2010; 38;1810-1817
CARDIOHELP: SAFE, TRANSPORTABLE, RAPIDLY DEPLOYED

ECLS FOR ASSISTED PCI IN PROFOUND CARDIOGENIC SHOCK¹

- 30 day Mortality
- 2x increase in survival rate!

Sheu et al. Crit Care Med 2010; 38;1810-1817
CARDIOHELP: SAFE, TRANSPORTABLE, RAPIDLY DEPLOYED

ECLS FOR ASSISTED PCI IN PROFOUND CARDIODYNAMIC SHOCK¹

- 30-day mortality was notably reduced in patients with ECMO support from what was seen for those without ECC

¹ Sheu et al. Crit Care Med 2010; 38;1810-1817
ECLS FOR ASSISTED PCI IN PROFOUND CARDIOGENIC SHOCK¹

• Implications:
  • ECLS offered “great benefit” in reducing 30 day mortality in patients with profound shock
  • Patients without ECLS tended to die early post-AMI
  • Can serve as a bridge to additional therapy as advanced heart failure was the strongest predictor of death in all groups

¹ Sheu et al. Crit Care Med 2010; 38;1810-1817
ECLS FOR ASSISTED PCI IN PROFOUND CARDIOGENIC SHOCK\(^1\)

• “ECLS support played the key role in maintaining hemodynamic stability, which, in turn, allowed primary PCI to be continued until final procedure success”

\(^1\) Sheu et al. Crit Care Med 2010; 38;1810-1817
OUTCOMES AND LONG-TERM QUALITY-OF-LIFE OF PATIENTS SUPPORTED BY EXTRACORPOREAL MEMBRANE OXYGENATION FOR REFRACTORY CARDIOGENIC SHOCK

• ECMO support can rescue 40% of otherwise fatal cardiogenic shock patients

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CARDIOPULMONARY RESUSCITATION WITH ASSISTED EXTRACORPOREAL LIFE-SUPPORT VERSUS CONVENTIONAL CARDIOPULMONARY RESUSCITATION IN ADULTS WITH IN-HOSPITAL CARDIAC ARREST: AN OBSERVATIONAL STUDY AND PROPENSITY ANALYSIS

- In hospital arrest
- 975 patients with IHCA
- 113 received CPR
- 59 had ECLS+CPR

CARDIOHELP: SAFE, TRANSPORTABLE, RAPIDLY DEPLOYED

CARDIOPULMONARY RESUSCITATION WITH ASSISTED EXTRACORPOREAL LIFE-SUPPORT VERSUS CONVENTIONAL CARDIOPULMONARY RESUSCITATION IN ADULTS WITH IN-HOSPITAL CARDIAC ARREST: AN OBSERVATIONAL STUDY AND PROPENSITY ANALYSIS

Figure 1: Relation between CPR duration and the survival rate to discharge
ECPR=extracorporeal CPR. CCFR=conventional CPR.

POSSIBLE APPLICATIONS\(^1\):

- Cardiogenic Shock
- High-Risk PCI
- Valvular Interventions
- Mechanical bridge to other assist device
- Ventricular Tachycardia Ablation

PERCUTANEOUS CARDIOPULMONARY BYPASS FOR CARDIAC EMERGENCIES

Table 1 Indications and contraindications for PCPS

<table>
<thead>
<tr>
<th>Reported indications</th>
<th>Contraindications</th>
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<tbody>
<tr>
<td>Resuscitation</td>
<td>Absolute</td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>Unwitnessed cardiac arrest</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>Aortic regurgitation</td>
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<tr>
<td>Cardiac trauma</td>
<td>Aortic dissection</td>
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<tr>
<td>Pulmonary insufficiency</td>
<td></td>
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<tr>
<td>Status asthmaticus</td>
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<tr>
<td>Smoke inhalation</td>
<td></td>
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<tr>
<td>Hyperalveolar proteinosis</td>
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<tr>
<td>Drug overdose</td>
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<tr>
<td>Pulmonary edema</td>
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<tr>
<td>Massive pulmonary embolism</td>
<td></td>
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<tr>
<td>Hypothermia</td>
<td></td>
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<tr>
<td><strong>Procedural support</strong></td>
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<tr>
<td>Assisted angioplasty</td>
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<tr>
<td>Pulmonary embolectomy</td>
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<tr>
<td>Port access coronary artery bypass</td>
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<tr>
<td>Resection of cerebral</td>
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<tr>
<td>arteriovenous malformation</td>
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<tr>
<td>Donor heart preservation</td>
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<tr>
<td>Abdominal aortic</td>
<td></td>
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<tr>
<td>graft replacement</td>
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<tr>
<td>Tracheal reconstruction</td>
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</tbody>
</table>

ESC / EACTS GUIDELINES: TREATMENT ALGORITHM FOR CIRCULATORY SUPPORT

WHY CONSIDER ECLS?

• Patients in profound refractory CS have dismal survival rates
• ECLS is a form of Cardiopulmonary Bypass that can be accomplished percutaneously
• ELSO registry data supports good outcomes in CS patients
  • >50% in pediatrics
  • 35% in adults
• Provides circulatory support up to 7 liters
• Provides oxygenation
• Affords temperature regulation (hypothermia)
CONSIDERING CARDIOHELP

- Special software application suitable for the OR, Cath lab and transportation purposes
- Connection cable for internal sensors:
  - 3 x pressures
  - 1 x arterial temperature
- 1 external pressure sensor
- Venous probe head for measurement of:
  - Venous oxygen saturation
  - Hemoglobin
  - Hematocrit
  - Venous temperature
HLS CATHETER VASCULAR ACCESS

- Select appropriately sized cannulae to provide the desired extracorporeal blood flow
- The flow through a single MAQUET HLS cannulae at various pressure drops

<table>
<thead>
<tr>
<th>Cannulae caliber (Fr)</th>
<th>100mmHg/H$_2$O</th>
<th>Flow (l/min)</th>
<th>Arterial cannula (15cm length)</th>
<th>Arterial cannula (23cm length)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>2.9</td>
<td>2.6</td>
<td></td>
<td></td>
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<tr>
<td>17</td>
<td>4.0</td>
<td>3.5</td>
<td></td>
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<tr>
<td>19</td>
<td>5.0</td>
<td>4.5</td>
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<tr>
<td>21</td>
<td>6.4</td>
<td>5.8</td>
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<tr>
<td>Cannulae caliber (Fr)</td>
<td>150mmHg/H$_2$O</td>
<td>3.3</td>
<td>2.7</td>
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<tr>
<td>17</td>
<td>4.3</td>
<td>3.8</td>
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<tr>
<td>19</td>
<td>5.5</td>
<td>5.0</td>
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<tr>
<td>21</td>
<td>7.0</td>
<td>6.4</td>
<td></td>
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</tbody>
</table>
CASE 2

- 52 yo gentleman with CAD s/p PCI to prox and distal RCA in 2006, and tobacco use presented with 2 days chest pain and SOB.

- Has not seen a physician in 15 yrs and stopped all his meds despite Hx of cardiac stents

- O/E AAOx3, in moderate distress, diaphoeritic & SOB

- BP 60/40

- EKG showed ST elevation in the anterolateral leads.

- Emergent cardiac cath
• Diagnostic LHC revealed: 99% proximal LM and 50% distally, 95% prox LAD, 75% mid. LCx: 100% in the mid segment, OM1 has 30%, RCA 40% prox and 100% distal.

• Immediately Stabalize CO with Impella while ECMO Team assembeled.

• PCI to LM, LAD prox and mid as well as OM1
Out Come

• Hemodynamics Stabilized in our lab on ECMO
• PCI Completed of LAD and CX
• Transferred to UC
• Kept on ECMO 6 days
• By Day three he was extubated awake and alert
• Scheduled to Have Cannula Removed on Monday at noon
• Hemorrhagic Stroke at 10 am
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.

Identify: Minimize Duration of Shock

**Suspect**
Cardiogenic Shock
See A

**Confirm**
CGS Diagnosis
reassess every 1-2 hours if criteria not initially met
See B

**Activate**
Heart Recovery Team/
Cardiac Cath Lab

---

**Suspect Shock**
Consider any of these criteria:
- Cool, clammy, pale skin
- Confusion/anxiety
- Rapid, shallow breathing
- SBP < 90 mmHg > 30 min
- Inotrope/vasopressor and/or IABP to maintain SBP > 90 mmHg
- Decrease in urine output (<0.5 cc/kg/h)
- Serum lactate level > 2 mmol/L

**Diagnose CGS**
- STEMI/Non-STEMI
- ECG ST segment abnormalities
- Troponin
- ECHO (assess cardiac function)

If PA Catheter (PAC) available:
- Cardiac Index (CI) < 2.2 L/min/m²
- pulmonary capillary wedge pressure (PCWP) > 18 mmHg
- Cardiac power output (CPO) < 0.6 watts
ECMO

• Expensive Bailout providing maximal life support.
• Multidisciplinary team
• 24 hr perfusionists in house
• Advanced Care Unit with trained nursing

• What is the end point?
  • Rest and recovery of Myocardium
  • LVAD
  • Transplant
Future Therapies
LVADs
Thank You
CONSIDERING CARDIOHELP

- Provide circulatory and pulmonary support
- High flow percutaneous CPB in the cath lab to unload the entire heart and support end organ perfusion
- Rapidly deployable, efficient, time-tested fem-fem perfusion provides complete CPB up to 7 liters per minute
- Integrated patient monitoring with ability to auto-regulate
- Transportable throughout the hospital
Impella® for AMI Cardiogenic Shock

**Identify**
- SBP < 90 mmHg or on inotropes/pressors
- Cold, clammy, tachycardia
- Lactate elevated > 2 mmol/L

**Stabilize Early**
- Impella Support pre-PCI
- Reduce Inotropes/Pressors

**Complete Revascularization**
- Per Guidelines

**Assess for Myocardial Recovery**
- (Weaning and Transfer Protocols)
  - ↑ Cardiac Output
  - ↑ Cardiac Power Output
  - ↑ Urine Output
  - ↓ Lactate
  - ↓ Inotropes

**No Recovery**
- Escalate (and Ambulate) or Transfer
- Ongoing Left Heart Failure
- Assess for Right Heart Failure

Cardiogenic etiology evaluation
- EKG (STEMI / NSTEMI)
- Echocardiography
- If available, PA catheter, cardiac output, CPO, CI, PCWP, SvO₂

References: