Οξύ Στεφανιαίο Συνδρόμο και Καρδιογενής Καταπληξία. 
Επεμβατική Προσέγγιση.

Δ. Ρούσος
Επιμ. Ά Γ.Ν. Άργους
Επιστ. Συνεργάτης Γ.Ν.Α.
"Ιπποκράτειο,,"
ΔΗΛΩΣΗ ΣΥΜΦΕΡΟΝΤΩΝ
CARDIOGENIC SHOCK (CS)

• Cardiogenic shock is defined as a clinical condition of systemic tissue hypoperfusion secondary to inadequate cardiac output despite adequate circulatory volume and LV filling pressure.

Rihal et al JACC 2015 SCAI/ACC Expert Consensus Statement on the use of p MCS devices
Shock Categories

Ventricular Septal Rupture 4.6%
"Isolated" RV Shock 3.4%
Tamponade/Rupture 1.7%
Acute Severe MR 8.3%
Other 7.5%
Predominant LV Failure 74.5%

Shock Registry
Hochman, JACC 36: 1063, 2000
ACS and CARDIOGENIC SHOCK (CS)

- **CS complicates:** 5-10% of ACS  
  (6-8% of STEMI)  
  (2% of nonSTEMI)

- **CS occurs:**  
  25% on admission  
  50% 0-6h after onset of symptoms  
  25% 6-24h after onset of symptoms

- **CS mortality rates:** 80-90% pre pPCI era  
  < 50% pPCI era
ACS and CARDIOGENIC SHOCK (CS)

• Despite this high mortality rates, it is important to note that patients with CS who survive to discharge have a long term outcome similar to that of patients without CS with a good functional outcome at 1 year.

Singh M et al JACC 2007
Hochman JS et al JAMA 2006
Decreasing in-hospital mortality with increasing rates of early PCI in patients with cardiogenic shock

STEMI registry Germany

<table>
<thead>
<tr>
<th>Year</th>
<th>Early PCI</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1994-96</td>
<td>72.2</td>
<td>100</td>
</tr>
<tr>
<td>1997-98</td>
<td>67.7</td>
<td>100</td>
</tr>
<tr>
<td>1999-00</td>
<td>56.1</td>
<td>100</td>
</tr>
<tr>
<td>2001-02</td>
<td>46.4</td>
<td>100</td>
</tr>
<tr>
<td>2007-08</td>
<td>38.1</td>
<td>100</td>
</tr>
</tbody>
</table>
# CARDIOGENIC SHOCK CRITERIA

## TABLE 1 Hemodynamic Criteria for Cardiogenic Shock

<table>
<thead>
<tr>
<th>Clinical</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP &lt; 90 mm Hg for 30 min</td>
</tr>
<tr>
<td>Supportive measures needed to maintain SBP &gt; 90 mm Hg</td>
</tr>
<tr>
<td>End-organ hypoperfusion</td>
</tr>
<tr>
<td>Cool extremities</td>
</tr>
<tr>
<td>UOP &lt; 30 ml/h</td>
</tr>
<tr>
<td>HR &gt; 60 beats/min</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hemodynamic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac index &lt; 2.2 ml/min/m²</td>
</tr>
<tr>
<td>PCWP &gt; 15 mm Hg</td>
</tr>
</tbody>
</table>

The SHOCK trial defined cardiogenic shock according to the clinical and hemodynamic criteria listed (11).

HR = heart rate; PCWP = pulmonary capillary wedge pressure; SBP = systolic blood pressure; UOP = urine output.
PATHOPHYSIOLOGY OF CS

Cardiogenic Shock Spiral

**Acute Myocardial Infarction**
- LV-Dysfunction
  - systolic
  - diastolic

**SIRS**
- Cardiac Output Stroke volume down
- Hypotension
- Peripheral Perfusion down
- Coronary-perfusion down

**Ischemia**
- LVEDP up
- Lung edema
- Hypoxia

**Progressive LV-Dysfunction**
- Death

**Inotropes/Vasopressors**
- Mechanical Support: IABP/LVAD
- Bleeding/Transfusion

**NO**
- Peroxynitrite
- IL-6 up
- TNF-α up

**SVR**
- Pro-Inflammation
- Catecholamine sensitivity down
- Contractility down

SIRS: Systemic Inflammatory Response Syndrome
SPECTRUM OF CARDIOGENIC SHOCK

Pre/Early Shock
Clinical
- SBP <100 mm Hg
- HR 70-100 beats/min
- Normal lactate
- Normal mentation
- Cool extremities
Hemodynamic
- CI 2.2
- PCWP <20
- LVEDP <20
- CPO >1 W
Vasoactive medications
- 0 or 1 low dose

Shock
Clinical
- SBP <90 mm Hg
- HR >100 beats/min
- Lactate >2
- AMS
- Cool extremities
Hemodynamic
- CI 1.5-2.0
- PCWP >20
- LVEDP >20
- CPO <1 W
Vasoactive medications
- 1 moderate to high dose

Severe shock
Clinical
- SBP <90 mm Hg
- HR >120 beats/min
- Lactate >4
- Obtundned
- Cool extremities
Hemodynamic
- CI <1.5
- PCWP >30
- LVEDP >30
- CPO <0.6 W
Vasoactive medications
- 2 or more
ACS and CARDIOGENIC SHOCK (CS) 
Reperfusion strategy

• Early revascularization or medical therapy?

• Multivessel or Culprit only PCI?

• Radial or femoral?
Early revascularization or Medical therapy?

The SHOCK Trial has been the most important study for management guidelines in patients with cardiogenic shock.

The New England Journal of Medicine

VOLUME 341
AUGUST 26, 1999
NUMBER 9

EARLY REVASCULARIZATION IN ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDILOGENIC 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*

www.escardio.org/ACCA

JS Hochman et al.
N Engl J Med 1999;341:625-34

70 ΧΡΟΝΙΑ ΚΑΡΔΙΟΛΟΓΙΑΣ (ΕΚΕ)
70 YEARS OF CARDIOLOGY (HSC)
ΠΑΝΕΛΛΗΝΙΟ ΚΑΡΔΙΟΛΟΓΙΚΟ ΣΥΝΕΔΡΙΟ
PANHELLENIC CONGRESS OF CARDIOLOGY
WWW.HCS.GR
Early revascularization or Medical therapy?

Early Revascularization and 1 Year Survival-SHOCK trial

- 13% absolute increase in 1 year survival in patients assigned to early revascularization
- number needed to treat of <8 patients to save 1 life

J5 Hochman et al.
JAMA. 2001;285:190-192

www.escardio.org/ACCA
Early revascularization or Medical therapy?

**SHOCK Trial**

- **30 days (n=302)**: 47% (ERV), 56% (IMS), p=0.11
- **6 months (n=301)**: 50% (ERV), 63% (IMS), p=0.03
- **12 months (n=299)**: 53% (ERV), 66% (IMS), p=0.03
- **6 years**: 67% (ERV), 80% (IMS), p=0.02

**NNT ~ 8**
### Table 1: Mortality for multivessel vs. culprit lesion only PCI in cardiogenic shock in registries

<table>
<thead>
<tr>
<th>Trial</th>
<th>N</th>
<th>Mortality multivessel PCI, %</th>
<th>Mortality culprit lesion only PCI, %</th>
<th>Adjusted odds ratio or hazard ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Webb et al.</td>
<td>74</td>
<td>55</td>
<td>20</td>
<td>2.75 (1.05–7.25)</td>
</tr>
<tr>
<td>Van der Schaaf et al.</td>
<td>161</td>
<td>60</td>
<td>53</td>
<td>Not reported (P = 0.05)</td>
</tr>
<tr>
<td>Cavender et al.</td>
<td>3087</td>
<td>36.5</td>
<td>27.8</td>
<td>1.5 (1.22–1.95)</td>
</tr>
<tr>
<td>Bauer et al.</td>
<td>336</td>
<td>48.8</td>
<td>37.4</td>
<td>1.28 (0.72–2.28)</td>
</tr>
<tr>
<td>Zeymer et al.</td>
<td>735</td>
<td>46.8</td>
<td>35.8</td>
<td>1.5 (1.15–1.84)</td>
</tr>
<tr>
<td>Yang et al.</td>
<td>338</td>
<td>35.0</td>
<td>30.6</td>
<td>1.06 (0.61–1.86)</td>
</tr>
<tr>
<td>Mylotte et al.</td>
<td>266</td>
<td>20.4</td>
<td>43.9</td>
<td>0.57 (0.38–0.84)</td>
</tr>
</tbody>
</table>

PCI, percutaneous coronary intervention; CI, confidence interval.
PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock

CULPRIT SHOCK TRIAL

A. Composite Primary End Point

- Relative risk, 0.83 (95% CI, 0.71–0.96)
- P=0.01

No. at Risk
- Multivessel PCI: 341
- Culprit-lesion-only PCI: 344

Days since Randomization

B. Death from Any Cause

- Relative risk, 0.84 (95% CI, 0.72–0.94)
- P=0.03

No. at Risk
- Multivessel PCI: 341
- Culprit-lesion-only PCI: 344

C. Renal-Replacement Therapy

- Relative risk, 0.71 (95% CI, 0.49–1.03)
- P=0.07

No. at Risk
- Multivessel PCI: 341
- Culprit-lesion-only PCI: 344
The prognostic impact of revascularization strategy in acute myocardial infarction and cardiogenic shock: insights from the British Columbia Cardiac Registry

Andrew McNeice¹,², Imad J. Nadra¹,², Simon D. Robinson¹,², Eric Fretz¹,², Lillian Ding³, Anthony Fung⁴, Eve Aymong⁵, Albert W. Chan⁶, Steven Hodge⁷, John Webb⁵, Sanjit Jolly, Shamir Mehta, Anthony Della Siega¹,², David A. Wood⁵, M. Bilal Iqbal¹,² on behalf of the British Columbia Cardiac Registry Investigators

¹ Victoria Heart Institute Foundation, Victoria, BC, Canada. 2 Royal Jubilee Hospital, Victoria, BC, Canada. 3 Provincial Health Services Authority, Vancouver, BC, Canada. 4 Vancouver General Hospital, Vancouver, BC, Canada. 5 St. Paul's Hospital, Vancouver, BC, Canada. 6 Royal Columbian Hospital, Vancouver, BC, Canada. 7 Kelowna General Hospital, Kelowna, BC, Canada.
Are there deleterious effects of immediate MVI?

The early separation of the Kaplan-Meier curves in our study and in CULPRIT-SHOCK trial would suggest that the negative impact associated with immediate MVI is acute and transient, and occurs early during the period of shock physiology.

2018 ESC/EACTS Guidelines on myocardial revascularization

**Recommendations for invasive evaluation and revascularization in non-ST-elevation acute coronary syndrome**

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Level&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>In cardiogenic shock, routine revascularization of non-IRA lesions is not recommended during primary PCI.&lt;sup&gt;190&lt;/sup&gt;</td>
<td>III</td>
<td>B</td>
</tr>
</tbody>
</table>

Primary percutaneous coronary intervention for myocardial reperfusion in ST-elevation myocardial infarction: procedural aspects (strategy and technique)

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Level&lt;sup&gt;b&lt;/sup&gt;</th>
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<td>In cardiogenic shock, routine revascularization of non-IRA lesions is not recommended during primary PCI.&lt;sup&gt;190&lt;/sup&gt;</td>
<td>III</td>
<td>B</td>
</tr>
</tbody>
</table>
RADIAL or FEMORAL access in CARDIOGENIC SHOCK?
RADIAL vs FEMORAL in CS

Arterial access site utilization in cardiogenic shock in the United Kingdom: Is radial access feasible?


Utilization Radial and Femoral Access in Patients with Cardiogenic Shock (January 2006-December 2012)

Am Heart J 2014;167:900-908
RADIAL vs FEMORAL in CS

Arterial access site utilization in cardiogenic shock in the United Kingdom: Is radial access feasible?

Mamas A. Mamas, MA, DPhil, BMBCh, MRCP, a,b, h Simon G. Anderson, a,b, h Karim Ratib c,h Helen Routledge, d,h Ludwig Neyses, b,h Douglas G. Fraser, a,h Iain Buchan, c,h Mark A. de Belder, f,h Peter Ludman, e,h and Jim Nolan, c,h Manchester, Stoke-on-Trent, Worcester, Middlesbrough, and Birmingham, United Kingdom

Am Heart J 2014;167:900-908
**Radial versus femoral approach comparison in percutaneous coronary intervention with intraaortic balloon pump support: The RADIAL PUMP UP Registry**

Enrico Romagnoli, MD, PhD, Maria De Vita, MD, Francesco Burzotta, MD, PhD, Bernardo Cortese, MD, Francesco Biondi-Zoccai, MD, Francesco Summario, MD, Roberto Patrizi, MD, Chiara Lanzillo, MD, Valerio Lucci, MD, PhD, Caterina Cavalessa, MD, Fabio Tarantino, MD, Giuseppe M. Sangiorgi, MD, Ernesto Lioy, MD, Filippo Crea, MD, Sunil V. Rao, MD, and Carlo Trani, MD. Rome, Avezzano, Forli, Milan, and Latina, Italy; and Durham, NC

<table>
<thead>
<tr>
<th></th>
<th>Femoral (n=209)</th>
<th>Radial (n=112)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NACE</td>
<td>57.4%</td>
<td>36.6%</td>
<td>0.001</td>
</tr>
<tr>
<td>MACCE</td>
<td>38.3%</td>
<td>25.9%</td>
<td>0.027</td>
</tr>
<tr>
<td>Death</td>
<td>34.9%</td>
<td>19.6%</td>
<td>0.004</td>
</tr>
<tr>
<td>Bleeding</td>
<td>33.5%</td>
<td>16.1%</td>
<td>0.001</td>
</tr>
<tr>
<td>Access related</td>
<td>18.7%</td>
<td>6.3%</td>
<td>0.002</td>
</tr>
<tr>
<td>Non-access related</td>
<td>14.8%</td>
<td>9.8%</td>
<td>0.204</td>
</tr>
<tr>
<td>Transfusions</td>
<td>14.8%</td>
<td>9.8%</td>
<td>0.023</td>
</tr>
</tbody>
</table>
PUMP UP REGISTRY

* Unadjusted model and adjusted including propensity score.
** Adjusted without propensity score.

Adjusted model is on age, gender, previous peripheral vascular disease, need for IABP, GRACE score (and CRUSADE score for bleeding).
RADIAL vs FEMORAL in CS

Impact of access site choice on outcomes of patients with cardiogenic shock undergoing percutaneous coronary intervention: A systematic review and meta-analysis

Samir B. Pancholy, MD, FACP, FACC, FSCAI, a Ghanshyam Palamander Subhash Shantha, MD, a, b Enrico Romagnoli, MD, PhD, a
Sasko Kedev, MD, PhD, a Ivo Bernat, MD, PhD, a Sunil V. Rao, MD, FACC, FSCAI, a,b Sanjti Jolly, MD, FRCP (C), a
Olivier F. Bertrand, MD, PhD, FSCAI, a,b and Tejas M. Patel, MD, DM, FESC, FACC, FSCAI a Scranton, PA; Viterbo, Italy; Skopje, Macedonia; Pilsen, Czech Republic; Durham, NC; Ontario, Quebec, Canada; and Ahmedabad, India

8 studies - 8,131 patients with CS undergoing PCI
TRA: 2,321 – TFA: 5,810 patients

MECHANICAL CIRCULATORY SUPPORT DEVICES
## FIGURE 1 Comparison of MCS Devices

<table>
<thead>
<tr>
<th></th>
<th>IABP</th>
<th>IMPELLA</th>
<th>TANDEMHEART</th>
<th>VA-ECMO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac Flow</strong></td>
<td>0.3-0.5 L/min</td>
<td>1-5 L/min (Impella 2.5, Impella CP, Impella 5)</td>
<td>2.5-5 L/min</td>
<td>3-7 L/min</td>
</tr>
<tr>
<td><strong>Mechanism</strong></td>
<td>Aorta</td>
<td>LV → AO</td>
<td>LA → AO</td>
<td>RA → AO</td>
</tr>
<tr>
<td><strong>Maximum implant days</strong></td>
<td>Weeks</td>
<td>7 days</td>
<td>14 days</td>
<td>Weeks</td>
</tr>
<tr>
<td><strong>Sheath size</strong></td>
<td>7-8 Fr</td>
<td>13-14 Fr</td>
<td>15-17 Fr Arterial 21 Fr Venous</td>
<td>14-16 Fr Arterial 18-21 Fr Venous</td>
</tr>
<tr>
<td><strong>Femoral Artery Size</strong></td>
<td>&gt;4 mm</td>
<td>Impella 2.5 &amp; CP - 5-5.5 mm Impella 5 - 8 mm</td>
<td>8 mm</td>
<td>8 mm</td>
</tr>
<tr>
<td><strong>Cardiac synchrony or stable rhythm</strong></td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td><strong>Afterload</strong></td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑↑↑</td>
</tr>
<tr>
<td><strong>MAP</strong></td>
<td>↑</td>
<td>↑↑</td>
<td>↑↑</td>
<td>↑↑↑</td>
</tr>
<tr>
<td><strong>Cardiac Flow</strong></td>
<td>↑</td>
<td>↑↑</td>
<td>↑↑</td>
<td>↑↑↑</td>
</tr>
<tr>
<td><strong>Cardiac Power</strong></td>
<td>↑</td>
<td>↑↑</td>
<td>↑↑</td>
<td>↑↑↑</td>
</tr>
<tr>
<td><strong>LVEDP</strong></td>
<td>↓</td>
<td>↓↓</td>
<td>↓↓</td>
<td>↓↓</td>
</tr>
<tr>
<td><strong>PCWP</strong></td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td><strong>LV Preload</strong></td>
<td>---</td>
<td>↓↓</td>
<td>↓↓</td>
<td>↓</td>
</tr>
<tr>
<td><strong>Coronary Perfusion</strong></td>
<td>↑</td>
<td>↑</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Myocardial oxygen demand</strong></td>
<td>↓</td>
<td>↓↓</td>
<td>←→</td>
<td>←</td>
</tr>
</tbody>
</table>
Four main families of devices exist for percutaneous MCS, which includes IABP, Impella (Abiomed Inc., Danvers, Massachusetts), TandemHeart (CardiacAssist, Inc., Pittsburgh, Pennsylvania), and VA-ECMO. Each device provides a different level of cardiac flow and device selection should be tailored to the level of support needed. Abbreviations as in Figure 1.
<table>
<thead>
<tr>
<th></th>
<th>IABP</th>
<th>Impella</th>
<th>TandemHeart</th>
<th>VA-ECMO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Contraindications</strong></td>
<td>Moderate to severe AR</td>
<td>LV thrombus</td>
<td>Severe PAD</td>
<td>Contraindications to anticoagulation</td>
</tr>
<tr>
<td></td>
<td>Severe PAD</td>
<td>Mechanical aortic valve</td>
<td>HIT</td>
<td>Moderate to severe AR</td>
</tr>
<tr>
<td></td>
<td>Aortic disease</td>
<td>Aortic stenosis with AVA &lt; 0.6</td>
<td>DIC</td>
<td>Severe PAD</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Contraindications to anticoagulation</td>
<td></td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td>Stroke</td>
<td>Device migration</td>
<td>Air embolism</td>
<td>Bleeding</td>
</tr>
<tr>
<td></td>
<td>Limb ischemia</td>
<td>Device thrombosis</td>
<td>Thromboembolism</td>
<td>Vascular trauma</td>
</tr>
<tr>
<td></td>
<td>Vascular trauma</td>
<td>Limb ischemia</td>
<td>Device Dislodgement</td>
<td>Limb ischemia</td>
</tr>
<tr>
<td></td>
<td>Balloon rupture</td>
<td>Vascular trauma</td>
<td>Cardiac tamponade</td>
<td>Acute kidney injury</td>
</tr>
<tr>
<td></td>
<td>Thrombocytopenia</td>
<td>Hemolysis</td>
<td>Limb ischemia</td>
<td>Hemolysis</td>
</tr>
<tr>
<td></td>
<td>Acute kidney injury</td>
<td>Infection</td>
<td>Vascular trauma</td>
<td>Thromboembolism</td>
</tr>
<tr>
<td></td>
<td>Bowel ischemia</td>
<td>Stroke</td>
<td>Hemolysis</td>
<td>Air embolism</td>
</tr>
<tr>
<td></td>
<td>Infection</td>
<td></td>
<td>Infection</td>
<td>Infection</td>
</tr>
</tbody>
</table>

| Bleeding/hemolysis   | +                              | ++                             | ++                             | ++                             |
| Vascular complications| +                              | ++                             | +++                            | +++                             |

Contraindications and complications must be reviewed prior to MCS device use in all patients and can vary according to device.

AR = aortic regurgitation; AVA = aortic valve area; DIC = disseminated intravascular coagulation; HIT = heparin-induced thrombocytopenia; LA = left atrium; LV = left ventricle; PAD = peripheral arterial disease; VSD = ventricular septal defect; other abbreviations as in Table 4.
History:

1962  Animal studies

1968  First clinical description in shock
Kantrowitz et al. JAMA 1968;203:135-140

1973  Hemodynamic effects in shock,
Mortality unchanged
Scheidt et al. NEJM 1973;288:979-984

> 40 years  > 1 Million patients treated, low complication rate,
Benchmark registry
Ferguson et al. JACC 2001;38:1456-1462
<table>
<thead>
<tr>
<th>Trial/First Author (Ref. #)</th>
<th>Indication</th>
<th>Definition</th>
<th>N</th>
<th>Control or No IABP Survival</th>
<th>Prophylactic or IABP Survival</th>
<th>Routine Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>IABP-SHOCK-II (3)</td>
<td>AMI and CS</td>
<td>SBP &lt;90 mm Hg for &gt;30 min or vasoactive medications needed to maintain SBP &gt;90, pulmonary edema, end-organ dysfunction (AMS, cool extremities, UOP &lt;30 ml/h, lactate &gt;2)</td>
<td>600</td>
<td>41.3%</td>
<td>39.7%</td>
<td>No difference in survival</td>
</tr>
<tr>
<td>TACTICS (59)</td>
<td>AMI and CS</td>
<td>s/p fibrinolysis</td>
<td>57</td>
<td>67% at 30 days Killip III/IV; 20% at 6 months</td>
<td>73% at 30 days Killip III/IV; 61% at 6 months</td>
<td>No significant difference except in Killip III/IV patients who received IABP</td>
</tr>
<tr>
<td>Waksman et al. (58)</td>
<td>AMI and CS</td>
<td>s/p fibrinolysis</td>
<td>45</td>
<td>19%</td>
<td>46%</td>
<td>In-hospital survival improved with IABP use in patients s/p fibrinolysis</td>
</tr>
<tr>
<td>NRMI (81)</td>
<td>AMI and CS</td>
<td>Observational study: IABP compared to no IABP among patients given fibrinolysis or primary angioplasty</td>
<td>IABP = 7,268 No IABP = 15,912</td>
<td>Lytics: 67% in-hospital mortality</td>
<td>PTCA: 42% in-hospital mortality</td>
<td>IABP provided substantial benefit in patients with AMI and CS who received fibrinolysis</td>
</tr>
<tr>
<td>CRISP-AMI (5)</td>
<td>Anterior MI with planned PCI</td>
<td>Prophylactic IABP</td>
<td>337</td>
<td>No difference in survival</td>
<td>No difference in survival</td>
<td>No reduction in infarct size</td>
</tr>
<tr>
<td>NCDR (82)</td>
<td>High risk including STEMI and CS</td>
<td>UPLMN, CS, severely depressed EF (&lt;30%), or STEMI</td>
<td>181,599</td>
<td>No difference in survival</td>
<td>No difference in mortality</td>
<td>Increase minor bleeding in IABP arm Decreased periprocedural complications in IABP (decreased hypotension) Elective IABP at 5 yrs associated with RRR 34% for all-cause mortality</td>
</tr>
<tr>
<td>BCIS-1 (4)</td>
<td>HR-PCI</td>
<td>EF &lt;30%, severe CAD: jeopardy score ≥8, no shock or STEMI</td>
<td>301</td>
<td>No difference in survival</td>
<td>No difference in survival</td>
<td></td>
</tr>
</tbody>
</table>

A nonexhaustive review of the literature for IABP use in cardiogenic shock and HR-PCI.

CAD = coronary artery disease; lytics = fibrinolysis; PTCA = percutaneous transluminal coronary angioplasty; RRR = relative risk reduction; s/p = status post; other abbreviations as in Table 4.
Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

Holger Thiele, M.D., Uwe Zeymer, M.D., Franz-Josef Neumann, M.D., Miroslaw Ferenc, M.D., Hans-Georg Olbrich, M.D., Jörg Hausleiter, M.D., Gert Richardt, M.D., Marcus Hennersdorf, M.D., Klaus Empen, M.D., Georg Fuernau, M.D., Steffen Desch, M.D., Ingo Eitel, M.D., Rainer Hambrecht, M.D., Jörg Fuhrmann, M.D., Michael Böhm, M.D., Henning Ebelt, M.D., Steffen Schneider, Ph.D., Gerhard Schuler, M.D., and Karl Wedan, M.D., for the IABP-SHOCK II Trial Investigators*
IABP-SHOCK II trial
### Recommendations for the management of patients with cardiogenic shock

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>In selected patients with ACS and cardiogenic shock, short-term mechanical circulatory support may be considered, depending on patient age, comorbidities, neurological function, and the prospects for long-term survival and predicted quality of life.</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>Routine use of IABPs in patients with cardiogenic shock due to ACS is not recommended.</td>
<td>III</td>
<td>B</td>
</tr>
</tbody>
</table>

---

2018 ESC/EACTS Guidelines on myocardial revascularization
Impella vs IABP

*ISAR-SHOCK Study*
Randomized 26 pts with cardiogenic shock following an acute MI to Impella 2.5 or IABP

- Cardiac Power Index (W/m²)
  - Pts. with Impella
  - Pts. with IABP

- Survival Probability
  - Log-rank P=0.97

Better hemodynamic support with Impella, but no difference in mortality

*Seyfarth M, et al, J Am Coll Cardiol 2008*
TandemHeart vs IABP

TandemHeart Shock Study

Cardiac Index

Pre
\[ p = 0.4 \]

Post
\[ p = 0.005 \]

30-day Mortality

30-day Mortality (%)

Transfusion

Required Transfusion (%)

\[ p = 0.002 \]

Limb Ischemia

Limb ischemia (%)

\[ p = 0.009 \]

Tiele H et al, Eur Heart J 2005
IABP Prior to PCI vs IABP After PCI

Mortality at 3 years according to IABP-timing (unadjusted)

Bad Segeberger Shock Registry

\[ P = .01 \]

IABP after PCI: 72.3%
IABP before PCI: 51.1%

No. at Risk

<table>
<thead>
<tr>
<th>Time, d</th>
<th>0</th>
<th>365</th>
<th>730</th>
<th>1095</th>
</tr>
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<tbody>
<tr>
<td>53</td>
<td>21</td>
<td>13</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>49</td>
<td>25</td>
<td>23</td>
<td>15</td>
<td></td>
</tr>
</tbody>
</table>

Data courtesy of Gert Richardt

The Current Use of Impella 2.5 in Acute Myocardial Infarction Complicated by Cardiogenic Shock: Results from the USPella Registry

USPella: STEMI and Shock Subset Analysis
Pre- vs Post-PCI Impella Activation

<table>
<thead>
<tr>
<th></th>
<th>Impella Pre-PCI</th>
<th>Impella Post-PCI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 63</td>
<td>N = 91</td>
</tr>
<tr>
<td><strong>MAP, mmHg</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre Support</td>
<td>67.9±20.7 (59)</td>
<td>59.1±17.3 (84)</td>
</tr>
<tr>
<td>On Support</td>
<td>94.5±21.3 (59)</td>
<td>94.4±24.4 (84)</td>
</tr>
<tr>
<td>p value</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>PCWP, mmHg</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre Support</td>
<td>30.8±7.8 (11)</td>
<td>32.7±13.4 (14)</td>
</tr>
<tr>
<td>On Support</td>
<td>19.7±7.9 (11)</td>
<td>18.9±11.1 (14)</td>
</tr>
<tr>
<td>p value</td>
<td>0.004</td>
<td>0.004</td>
</tr>
<tr>
<td><strong>Cardiac Index, L/min/m²</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre Support</td>
<td>1.9±0.9 (7)</td>
<td>1.9±0.6 (16)</td>
</tr>
<tr>
<td>On Support</td>
<td>2.3±0.8 (7)</td>
<td>2.9±0.6 (16)</td>
</tr>
<tr>
<td>p value</td>
<td>0.055</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Cardiac Power Output, Watt</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre Support</td>
<td>0.54±0.2 (7)</td>
<td>0.46±0.1 (16)</td>
</tr>
<tr>
<td>On Support</td>
<td>0.83±0.4 (7)</td>
<td>1.2±0.5 (16)</td>
</tr>
<tr>
<td>p value</td>
<td>0.035</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Log-Rank, p=0.004

Cumulative survival

Days from initiation of Impella 2.5 support

O’Neill et al. J Interven Cardiol 2013:9999.1-11
ΣΥΜΠΕΡΑΣΜΑΤΑ

• ΕΓΡΗΓΟΡΣΗ-ΤΑΧΕΙΑ ΔΡΑΣΗ-ΓΝΩΣΗ-ΕΚΠΑΙΔΕΥΣΗ

• Η ταχεία επαναιμάτωση αποτελεί τον ακρογωνιαίο λίθο στην αντιμετώπιση του ΟΕΜ με καρδιογενές shock.

• Καλό είναι να μην υποβάλουμε τους ασθενείς με καρδιογενές shock σε μακροχρόνιες επεμβάσεις.

• Η χρήση συσκευών υποβοήθησης της κυκλοφορίας ενδέχεται να βοηθά (ειδικά τους ασθενείς με βαρειά καταπληξία και υψηλού κινδύνου αγγειοπλαστική).
ΕΥΧΑΡΙΣΤΩ ΓΙΑ ΤΗΝ ΠΡΟΣΟΧΗ ΣΑΣ
Illustrations of PV loops after activation of device therapy (gray loops). (A) Intra-aortic balloon pump (IABP) counterpulsation reduces both peak LV systolic and diastolic pressures and increases LV stroke volume. The net effect is a reduced slope of arterial elastance ($E_{a2}$). (B) Percutaneous LV assist devices (pLVAD: Impella and TandemHeart) significantly reduce LV pressures, LV volumes, and LV stroke volume. The net effect is a significant reduction in cardiac workload. (C) Veno-arterial extra-corporeal membrane oxygenation (VA-ECMO) without a LV venting strategy increases LV systolic and diastolic pressure, while reducing LV stroke volume. The net effect is an increase in arterial elastance ($E_{a3}$).
Each pressure volume (PV) loop represents one cardiac cycle (A). Beginning at the end of isovolumic relaxation (Point 1), LV volume increases during diastole (Phase 1 to 2). At end-diastole (Point 2), LV volume is maximal and isovolumic contraction (Phase 2 to 3) begins. At the peak of isovolumic contraction, LV pressure exceeds aortic pressure and blood begins to eject from the LV into the aorta (Point 3). During this systolic ejection phase, LV volume decreases until aortic pressure exceeds LV pressure and the aortic valve closes, which is known as the end-systolic pressure-volume point (ESPV) (Point 4). Stroke volume (SV) is represented by the width of the PV loop as the volume difference between end-systolic and end-diastolic volumes (Points 1 and 2). The shaded area within the loop represents stroke work. Load-independent LV contractility, also known as $E_{\text{max}}$, is defined as the maximal slope of the ESPV point under various loading conditions, known as the ESPV relationship (ESPVR). Effective arterial elastance ($E_a$) is a component of LV after-load and is defined as the ratio of end-systolic pressure and stroke volume. Under steady state conditions, optimal LV pump efficiency occurs when the ratio of $E_a$/$E_{\text{max}}$ approaches 1. (B) Representative PV loop in AMI (blue loop). LV contractility ($E_{\text{max}}$) is reduced; LV pressure, SV, and LV stroke work may be unchanged or reduced; and LVEDP is increased. (C) Representative PV loop in cardiogenic shock (gray loop). $E_{\text{max}}$ is severely reduced; LVEDV and LVEDP are increased; and SV is reduced.
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Figure 6 Algorithm for the management of patients with cardiogenic shock.