Οξύ στεφανιαίο σύνδρομο και καρδιογενής καταπληξία. Επεμβατική προσέγγιση

Σωτήριος Πατσιλινάκος
Κωνσταντοπούλειο Γ.Ν. Ν. Ιωνίας
ACUTE HEART FAILURE AND CAD:

- ACS / LV ischaemic dysfunction

- Mechanical complications of AMI
  - Acute MR
  - V.S. rupture
  - Free wall rupture

- Right ventricular infarction

CARDIIOGENIC SHOCK
ΑΙΤΙΟΛΟΓΙΑ ΚΑΡΔΙΟΓΕΝΟΥΣ ΚΑΤΑΠΛΗΞΙΑΣ

- Predominant LV Failure: 74.5%
- Acute Severe MR: 8.3%
- VSD: 4.6%
- Isolated RV Shock: 3.4%
- Tamponade/rupture: 1.7%
- Other: 7.5%
- Other: 7.5%

Shock Registry
JACC 2000 35:1063
Classification of AHF

- **Hypertensive AHF**: High BP, +/- preserved LV systolic function; increased sympathetic tone with ↑HR, vasoconstriction; may be euvolaemic or only mildly hypervolemic, and frequently with signs of pulmonary or systemic congestion.
- **PULMONARY OEDEMA**: Severe respiratory distress, ↑RR, orthopnea, rales. O2 sats <90% RA prior to O2.
- **Acute Ly Decompensated Chronic HF**: Clinical and lab evidence of an ACS; ~15% of patients with an ACS have signs and symptoms of HF. Episodes of AHF are frequently associated with or precipitated by arrhythmia (bradycardia, AF, VT).
- **Cardiogenic shock**: Usually sys BP <90 mmHg or drop in MAP >30 mmHg and absent/low urine output. Organ hypoperfusion and pulmonary congestion develop rapidly.
- **Right HF**: Low output in absence of pulmonary congestion with increased JVP, w/ or w/o HSM, and low LV filling pressures.

Usually a hx of prog. worsening of known chronic HF on Rx, and evidence of systemic/pulmonary congestion.

ESC 2008
Classification of AHF

- high BP, +/- preserved LV systolic func; increased sympathetic tone with ↑HR, vasoconstriction; may be euvolaemic or only mildly hypervolemic, and frequently with signs of pulmonary or systemic congestion

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- low output in absence of pulmonary congestion with increased JVP, w/ or w/out HSM, and low LV filling pressures

usually a hx of prog. worsening of known chronic HF on Rx, and evidence of systemic/pulmonary congestion.

ESC 2008
• Hypotension (>30 min): < 90 mmHg
• Hypoperfusion: oliguria <30 ml/kg, cold extremities, altered mental status)
• Pulmonary congestion (“cold and wet”)
• C.I.: < 2,2 lt/min/m², PCWP >15-18 mmHg
Table II – Clinical and hemodynamic subgroups in acute myocardial infarction

<table>
<thead>
<tr>
<th>Killip Subgroup</th>
<th>Clinical characteristics</th>
<th>Hospital mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No congestion signs</td>
<td>&lt;6%</td>
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<tr>
<td>II</td>
<td>S3, basal rales</td>
<td>&lt;17%</td>
</tr>
<tr>
<td>III</td>
<td>Acute pulmonary edema</td>
<td>38%</td>
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<tr>
<td>IV</td>
<td>Cardiogenic shock</td>
<td>81%</td>
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</table>

<table>
<thead>
<tr>
<th>Forrester subgroup</th>
<th>Hemodynamic characteristics</th>
<th>Hospital mortality</th>
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<tbody>
<tr>
<td>I</td>
<td>PCP &lt;18, IC &gt;2.2</td>
<td>3%</td>
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<tr>
<td>II</td>
<td>PCP &gt;18, IC &gt;2.2</td>
<td>9%</td>
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<tr>
<td>III</td>
<td>PCP &lt;18, IC &lt;2.2</td>
<td>23%</td>
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<tr>
<td>IV</td>
<td>PCP &gt;18, IC &lt;2.2</td>
<td>51%</td>
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</table>

PCP - pulmonary capillary pressure; CI - cardiac index.

Simplified Forrester & Diamond hemodynamic classification in STEMI

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<thead>
<tr>
<th>Class</th>
<th>Hypotension</th>
<th>Pulmonary congestion</th>
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<tr>
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<td>2</td>
<td>No</td>
<td>Pulmonary</td>
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<tr>
<td>3*</td>
<td>Hypotension</td>
<td>No</td>
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<tr>
<td>4*</td>
<td>Hypotension</td>
<td>Pulmonary</td>
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</table>

* Include RV and Bi-Ventricular MI. ** Cardiogenic shock.
**Figure 12.2** Initial management of a patient with acute heart failure. *Acute mechanical cause: myocardial rupture complicating acute coronary syndrome (free wall rupture, ventricular septal defect, acute mitral regurgitation), chest trauma or cardiac intervention, acute native or prosthetic valve incompetence secondary to endocarditis, aortic dissection or thrombosis, see above.*
ACS AND CARDIOGENIC CAD

INVASIVE TREATMENT

- Revascularization (PCI/CABG)
- Surgical Repair
- Mechanical Circulatory Support
ACS AND CARDIOGENIC CAD

INVASIVE TREATMENT

• Revascularization (PCI/CABG)
• Surgical Repair
ACS AND CARDIOGENIC CAD

INVASIVE TREATMENT

• ACS / LV ischaemic dysfunction

• Mechanical complications of AMI
  ➢ Acute MR
  ➢ V.S. rupture
  ➢ Free wall rupture

• Right ventricular infarction
TREATMENT OF **STEMI AND AHF**

2017 ESC Guidelines

[Diagram showing the treatment strategies for STEMI and AHF, including ECG diagnosis, primary PCI strategy, fibrinolysis strategy, and reperfusion criteria.]
**Immediate invasive strategy (<2h)** in pts with at least one (IC):

- haemodynamic instability or cardiogenic shock
- mechanical complications of MI
- acute heart failure with refractory angina or ST deviation
- recurrent dynamic ST- or T-wave changes, particularly with intermittent ST-elevation
- recurrent or ongoing chest pain refractory to medical treatment
- life-threatening arrhythmias or cardiac arrest
• **Immediate invasive strategy (<2h)** in pts with at least one (IC):
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## Procedural aspects of the PPCI strategy

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Class</th>
<th>Level</th>
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<tr>
<td><strong>IRA strategy</strong></td>
<td></td>
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<tr>
<td>Primary PCI of the IRA is indicated.</td>
<td>I</td>
<td>A</td>
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<td>New coronary angiography with PCI if indicated is recommended in patients with symptoms or signs of recurrent or remaining ischaemia after primary PCI.</td>
<td>I</td>
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<td><strong>IRA technique</strong></td>
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<td>Stenting is recommended (over balloon angioplasty) for primary PCI</td>
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<tr>
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<td>Routine use of thrombus aspiration is not recommended.</td>
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<td>A</td>
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<tr>
<td>Routine use of deferred stenting is not recommended.</td>
<td>III</td>
<td>B</td>
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<tr>
<td><strong>Non-IRA strategy</strong></td>
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<td></td>
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<td>Routine revascularization of non-IRA lesions should be considered in STEMI patients with multivessel disease before hospital discharge.</td>
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ACS AND CARDIOGENIC SHOCK

INVASIVE TREATMENT

• ACS / LV ischaemic dysfunction

• Mechanical complications of AMI
  ➢ Acute MR
  ➢ V.S. rupture
  ➢ Free wall rupture

• Right ventricular infarction
ACCS AND CARDIOGENIC SHOCK
INVASIVE TREATMENT

Mechanical complications of AMI

➢ Acute MR
  • Hemodynamic stabilization (diuretics, vasodilators, IABP - IIa)
  • Catheterization
  • Surgery (MV replacement or repair)

➢ V.S. rupture
  • Hemodynamic stabilization (diuretics, vasodilators, IABP - IIa)
  • Catheterization
  • Surgery (timing depends on hemodynamic stability)

➢ Free wall rupture
  • Pericardiocentesis
  • Hemodynamic stabilization
  • Immediate surgery
ACS AND CARDIOGENIC SHOCK

INVASIVE TREATMENT

• ACS / LV ischaemic dysfunction

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  ➢ Acute MR
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  ➢ Free wall rupture

• Right ventricular infarction
ACS AND CARDIOGENIC SHOCK

INVASIVE TREATMENT

Right ventricular infarction

• Early reperfusion (<2h) with PCI (do not ignore right ventricle branches)

• Hemodynamic stabilization (balance between fluid repletion and inotropic agents)

• Improve concomittant LV systolic / diastolic dysfunction

• Treatment of cardiac arrhythmias

• Consider rescue therapies such as mechanical circulatory support in medically refractory cases of reversible RVF
ACS AND CARDIOGENIC CAD

INVASIVE TREATMENT

- Revascularization (PCI/CABG)
- Surgical Repair

Mechanical Circulatory Support
Mechanical circulatory support in cardiogenic shock

Karl Werdan¹*, Stephan Gielen¹, Henning Ebelt¹, and Judith S. Hochman²

A  IABP  B  Impella  C  Tandem Heart  D  ECMO
The Spectrum of AMCS Devices in 2017

Left Ventricle

Pulsatile

- IABP

Continuous Flow Pumps

Axial-Flow

- Impella CP
- PHP *

Centrifugal Flow

- TandemHeart
- VA-ECMO

Right Ventricle

Intracorporeal

- Axial Flow
- Impella RP
- VA-ECMO

Centrifugal Flow

- Tandem pRVAD
- Protek Oxy-RVAD
IABP in Shock
How and Why It Should Work

<table>
<thead>
<tr>
<th>Net Effects</th>
<th>Benefits</th>
</tr>
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<tbody>
<tr>
<td>~20% increase in CO</td>
<td>Enhanced coronary blood flow</td>
</tr>
<tr>
<td>~20% decrease in SBP</td>
<td>Improved myocardial energetics</td>
</tr>
<tr>
<td>~30% increase in DBP</td>
<td>Low vascular complications (2-6%)</td>
</tr>
<tr>
<td>≤ 20% decrease in HR</td>
<td>Should help in acute ischemic states</td>
</tr>
<tr>
<td>≤ 20% decrease in PAWP</td>
<td></td>
</tr>
</tbody>
</table>
2008 ESC STEMI Guidelines

## Treatment of shock (Killip class IV)

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Class</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Mechanical ventilatory support according to blood gasses</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Haemodynamic assessment with balloon floating catheter</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>Inotropic agents: dopamine and dobutamine</td>
<td>IIA</td>
<td>C</td>
</tr>
<tr>
<td>Intra-aortic balloon pump</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>LV assist devices</td>
<td>IIA</td>
<td>C</td>
</tr>
<tr>
<td>Early revascularization</td>
<td>I</td>
<td>B</td>
</tr>
</tbody>
</table>

### 2004 ACC/AHA STEMI Guidelines

**Class I**

1. Intra-aortic balloon counterpulsation should be used in STEMI patients with hypotension (systolic blood pressure less than 90 mm Hg or 30 mm Hg below baseline mean arterial pressure) who do not respond to other interventions, unless further support is futile because of the patient’s wishes or contraindications/unsuitability for further invasive care. See Section 7.6.2. *(Level of Evidence: B)*

2. Intra-aortic balloon counterpulsation is recommended for STEMI patients with low-output state. See Section 7.6.3. *(Level of Evidence: B)*

3. Intra-aortic balloon counterpulsation is recommended for STEMI patients when cardiogenic shock is not quickly reversed with pharmacological therapy. IABP is a stabilizing measure for angiography and prompt revascularization. See Section 7.6.5. *(Level of Evidence: B)*

4. Intra-aortic balloon counterpulsation should be used in addition to medical therapy for STEMI patients with recurrent ischemic-type chest discomfort and signs of hemodynamic instability, poor LV function, or a large area of myocardium at risk. Such patients should be referred urgently for cardiac catheterization and should undergo revascularization as needed. See Section 7.8.2. *(Level of Evidence: C)*
HEMODYNAMIC EFFECTS OF INTRA-AORTIC BALLOON COUNTERPULSATION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK: THE PROSPECTIVE, RANDOMIZED IABP SHOCK TRIAL

n = 40

Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial

P = 0.92 by log-rank test

IABP (N = 300)  Control (N = 298)

A Prospective, Randomized Evaluation of Prophylactic Intraaortic Balloon Counterpulsation in High Risk Patients With Acute Myocardial Infarction Treated With Primary Angioplasty

PAMI II trial

Use and impact of intra-aortic balloon pump on mortality in patients with acute myocardial infarction complicated by cardiogenic shock: results of the Euro Heart Survey on PCI

Thrombolysis Plus Aortic Counterpulsation: Improved Survival in Patients Who Present to Community Hospitals With Cardiogenic Shock

Total patients with discharge codes for cardiogenic shock + MI: 335

Confirmed cardiogenic shock: 255

Met criteria for MI: 191

Received thrombolytic therapy: 65
No thrombolytic therapy: 126
Community hospital survival 5%

Within 12 h: 46
Community hospital survival 70%
Beyond 12 h: 19

Received IABP: 27
Community hospital survival 93%
No IABP: 19
Community hospital survival 37%

No transfer: 4
30-day survival 50%
Transfer to tertiary care: 23
30-day survival 70%
Transfer to tertiary care: 7
30-day survival 86%
No transfer: 12
30-day survival 0%

Revascularization procedure: 27
30-day survival 74%

Thrombolysis and Counterpulsation to Improve Survival in Myocardial Infarction Complicated by Hypotension and Suspected Cardiogenic Shock or Heart Failure: Results of the TACTICS Trial

- TACTICS (n=57; P=0.23*)
  - Randomized: IABP + lytic 43%, Lytic alone 34%
  - 6 months

- Kovack\textsuperscript{16} (n=46; P=0.02)
  - Observational: IABP + lytic 33%, Lytic alone 33%
  - 1 year

- GUSTO-I\textsuperscript{17} (n=310; P=0.04)
  - IABP + lytic 57%, Lytic alone 68%
  - 1 year

- NRMI\textsuperscript{18} (n=23,180; P<0.05)
  - IABP + lytic 67%, Lytic alone 67%
  - in-hospital

Time to Reperfusion Remains Crucial
IABP: Time may be the key

Ischemic Insult

Shock

Performance Parameter

Time

Reperfusion

IABP

IABP

IABP
IABP: Time may be the key

- Non-IABP devices
- “time to effective unloading”
Impella
Percutaneous Bi-Ventricular Support in the Cath Lab

Impella CP (LV support)

Impella RP (RV support)
Randomized Trial of Impella vs. IABP in AMI with Shock (ISAR-SHOCK)

Change in Cardiac Index (primary endpoint)

- IABP: 0.11
- Impella: 0.49 (p=0.02)

Cardiac Power Index

- Impella (native heart CPI)
- Impella (pump contribution)
- IABP

Pre-Support vs. Post-Support

N=25

Adapted from Seyfarth et al., JACC 2008
In-Hospital Survival by Timing of Support

Survival to Discharge By timing of PCI

- All Pts: 50.9%
- Pre-PCI: 65.1%
- Post-PCI: 40.7%

P = 0.003

STEMI
- Pre-PCI: 60.0% (N=35)
- Post-PCI: 40.0% (N=80)

NSTEMI
- Pre-PCI: 71.4% (N=28)
- Post-PCI: 45.5% (N=11)

Impella Support Initiation
A Prospective, Randomized Clinical Trial of Hemodynamic Support With Impella 2.5 Versus Intra-Aortic Balloon Pump in Patients Undergoing High-Risk Percutaneous Coronary Intervention

The PROTECT II Study

452 Symptomatic Patients

Intent-to-treat population.

Per protocol population.

TandemHeart

- Pumps blood from the LA to the femoral artery via a trans-septally placed LA cannula
- With TandemHeart, both the LV and the pump contribute flow to the aorta simultaneously thereby working in parallel, or tandem
Hemodynamic Effects of TandemHeart in Patients with Cardiogenic Shock

Cardiac Index

PCWP

p=0.017 Pre vs During Support

p=0.013 Pre vs During Support
ECMO

- Percutaneous heart-lung machine
- Centrifugal pump
- Hemodynamic support >4.5 l/min
- Can increase preload and afterload
- No randomized control trials or large cohorts yet

Solving the Hemodynamic Support Equation For Patients Referred for HR-PCI or Shock

<table>
<thead>
<tr>
<th>Circulatory Support Systemic Perfusion</th>
<th>Ventricular Support LV/RV Unloading</th>
<th>Coronary Perfusion</th>
<th>Renal &amp; Hepatic Unloading</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Arterial Pressure</td>
<td>LV-ESP &amp; EDP Aortic Pulse Pressure</td>
<td>MAP - LVEDP</td>
<td>RA-PA Hemodynamics</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>IABP</th>
<th>VA-ECMO</th>
<th>TandemHeart</th>
<th>Impella</th>
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<tbody>
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Shock Team Treatment Algorithm

Acute Cardiopulmonary Failure

- Biventricular
  - Impella (Bilateral)
  - V-A ECMO
  - Surgical Bi-VAD
  - Yes
    - V-A ECMO
  - No
    - LV (also MR, VSD)
      - Poor oxygenation likely to persist
        - Yes
          - Impella (LV)
          - Tandem
          - V-A ECMO
        - No
          - RV
            - Poor oxygenation likely to persist
              - Yes
                - V-V ECMO
              - No
                - Pulmonary
                  - V-V ECMO

*NOTE: If an unstable patient cannot make to procedural area, V-A ECMO at bedside can be considered in all scenarios
<table>
<thead>
<tr>
<th>European Guidelines</th>
<th>American Guidelines</th>
<th>German–Austrian Guidelines</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IIb/B</strong> Intraaortic balloon pumping may be considered in patients with cardiogenic shock (Killip class IV)</td>
<td><strong>IIa/B</strong> Haemodynamic support for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacological therapy</td>
<td>↑ In patients undergoing fibrinolysis ↔ In patients undergoing PCI ↑ In patients with mechanical complications</td>
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<td><strong>IIb/C</strong> LV assist devices may be considered for circulatory support in refractory shock in patients with cardiogenic shock (Killip class IV)</td>
<td><strong>IIb/C</strong> Alternative left ventricular (LV) assist devices for circulatory support may be considered in patients with refractory cardiogenic shock.</td>
<td>Routine use not recommended</td>
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<tr>
<th>Recommendation</th>
<th>Grade</th>
<th>Level</th>
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<tr>
<td>IABP insertion should be considered in patients with haemodynamic instability/cardiogenic shock due to mechanical complications.</td>
<td>IIa</td>
<td>C</td>
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<tr>
<td>Patients with mechanical complication after acute myocardial infarction require immediate discussion by the Heart Team.</td>
<td>I</td>
<td>C</td>
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<tr>
<td>Short-term mechanical circulatory support in ACS patients with cardiogenic shock may be considered.</td>
<td>IIb</td>
<td>C</td>
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<tr>
<td>Percutaneous repair of VSD may be considered after discussion by the Heart Team.</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>Routine use of IABP in patients with cardiogenic shock is not recommended.</td>
<td>III</td>
<td>A</td>
</tr>
</tbody>
</table>
CASE PRESENTATION

• On arrival the patient was unresponsive with no spontaneous breathing

• Probable time of arrest before admission: 30-40 min

• The presenting rhythm was ventricular fibrillation

• After 4 cycles of CPR and defibrillation we managed to restore a perfusing rhythm
Hypothermia and PCI
CONCLUSIONS

• **Early** diagnosis and **early** treatment are crucial in cardiogenic shock caused by CAD

• The assist device (if used) should be performed before PCI

• Treating properly ACS syndromes, cardiogenic shock could be **prevented**
Despite a Successful Operation the Patient Died
Reasons for Failure:

1. Failure to Revascularize: Risk Aversion
2. Failure to Salvage
   - Late presentation
   - No Reflow
   - Incomplete Revascularization
Keys to success:

1. Revascularization
2. Hemodynamic monitoring
3. Early Support
4. Creation of a SHOCK network
Reasons for Failure:

1. Failure to Revascularize: Risk Aversion
2. Failure to Salvage
3. Failure to Characterize
4. Failure to Support
5. Failure to Transfer
Cardiogenic Shock

- Myocardial contractile dysfunction resulting in:
  - low cardiac output (CI < 2.2 L/min/M²)
  - *despite* normal or elevated pre-load (PCWP greater > 15-18 mmHg).
- Systolic blood pressure < 90 mmHg
  - for > 30 min or require catecholamines to maintain SBP > 90 mmHg.
- Clinical signs of peripheral tissue hypoperfusion.
  - decreased urine output.
  - altered mental status and/or cold extremities.
- Signs of pulmonary congestion and impaired organ perfusion with:
  - altered mental status; cold, clammy skin and extremities.
  - oliguria with urine output < 30 mL/H; abnormal LFTs.
  - serum lactate > 2.0 mmol/L.