Mechanical Assist Devices in Cardiogenic Shock

Prof. Dr. J.J. Piek
Conflict of interest disclosure

- Member MAB Abbott Vascular
- Consultant Miracor
Case 1

♀ 61 years

History
Hypertension

Presentation
Acute congestive heart failure after sudden onset chest pain.
Rapid hemodynamic detoriation:
- systolic blood pressure < 80 mmHg, cold extremities, altered mental state.
- Intubation because of respiratory insufficiency

ECG
Suggesting severe 3-vessel disease and/or left main CA occlusion
Case 1

15-DIC-1944 (61 yr)
Female

Vent. rate: 81 BPM
PR Interval: 146 ms
QRS duration: 86 ms
QT/QTc: 356/413 ms
P-R-T axes: +83 -20 195

Referred by:
Confirmed By: NON SUPERVISED

25mm/s 10mV 40Hz 00SE 12SL 233 CID: 15

Academic Medical Center, Amsterdam, The Netherlands
IABP insertion, prior to CAG and PCI, because of hemodynamic detoriation
Subtotal narrowing LMCA (aspect of plaque rupture)
Case 1 - PCI

Successful IABP assisted PCI
direct stenting LM-LAD; fenestration towards RCX; kissing balloons LM-LAD and LM-RCX.
Case 1

Hospital course

Cath. Lab:
After PCI persisting cardiogenic shock / hemodynamic instability:
- Systolic blood pressure < 90 mmHg, despite inotropes and IABP.
- pH 6.9
- Additional insertion of Impella 2.5 (percutaneous LVAD).

ICU:
- Further hemodynamic deterioration, despite inotropics, IABP, Impella 2.5
- Detioriation of renal function, requiring Continuous Veno-Venous Hemofiltration
- 3 days after acute event, surgical insertion of Impella 5.0
- Gradual recovery of LV function
- 12 days after acute event, weaning from Impella 5.0 en removal of device
- Weaning from mechanical ventilation
Mortality in CS

Goldberg et al. NEJM 1999; Hochman et al. NEJM 1999; Sjauw, Henriques et al. NHJ 2012
Zeymer et al. Eurointervention 2011; Thiele et al. ESC Congress 2012
CS: “Vicious circle of deterioration”

Rationale for mechanical assistance

- **STEMI**
  - Myocardial recovery

- **STEMI + CS**
  - Myocardial recovery
  - Organ recovery

**Mechanisms**
- Acceleration recovery of contractility in stunned myocardium by increasing postischemic myocardial (microvascular) blood flow.
- Unloading effect:
  - Peak left ventricular wall stress ↓
  - Myocardial workload ↓
  → Reduced myocardial oxygen consumption.

Sjauw KD, Engström AE, Henriques JPS; Percutaneous Mechanical Cardiac Assist In Myocardial Infarction. Where Are we Now, Where Are We Going? Acute Card Care 2007;9(4):222-30
Currently available devices

- IABP
- TandemHeart
- Impella 2.5
- Impella 3.5 (CVAD)
- Impella 5.0 (surgical insertion)
- Minituarized ECMO
What hemodynamic support do you use at your institution for patients in cardiogenic shock?

a. Only positive inotropic and vasopressor drugs

b. Positive inotropic drugs and IABP

c. Drugs, IABP and/or more effective support (Impella, Tandemheart, LVAD, ECMO)
IABP
IABP - The guidelines

Class 1B  “although no data support a reduction in mortality rates”

Class 1C
Meta-analysis IABP vs. no-IABP in HR-STEMI

Randomized controlled trials

### Meta-analysis Results

<table>
<thead>
<tr>
<th>Trial</th>
<th>IABP</th>
<th>No IABP</th>
<th>30-day Mortality</th>
<th>Risk Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No reperfusion</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O’Rourke</td>
<td>8/14</td>
<td>10/16</td>
<td>0.01 (-0.26 to 0.28)</td>
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<tr>
<td>Flaherty</td>
<td>4/10</td>
<td>3/10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>12/24</td>
<td>13/26</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Thrombolysis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kono</td>
<td>0/23</td>
<td>0/22</td>
<td>-0.06 (-0.21 to 0.08)</td>
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<tr>
<td>TACTICS</td>
<td>10/30</td>
<td>12/27</td>
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<tr>
<td>Overall</td>
<td>10/53</td>
<td>12/49</td>
<td></td>
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<tr>
<td><strong>Primary PCI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Ohman</td>
<td>2/96</td>
<td>2/86</td>
<td>0.01 (-0.02 to 0.04)</td>
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<tr>
<td>PAMI-II</td>
<td>9/211</td>
<td>7/226</td>
<td></td>
<td></td>
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<tr>
<td>van ’t Hof</td>
<td>12/118</td>
<td>9/120</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>23/435</td>
<td>18/432</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Overall</strong></td>
<td>45/502</td>
<td>43/507</td>
<td>0.01 (-0.03 to 0.04)</td>
<td></td>
</tr>
</tbody>
</table>

P (heterogeneity) = 0.94
I² = 0%
P (overall effect) = 0.75

Meta-analysis of IABP vs. no IABP in CS

Cohort Studies

A

<table>
<thead>
<tr>
<th>Trial</th>
<th>IABP n/N</th>
<th>no IABP n/N</th>
<th>30-day mortality risk difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>No reperfusion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moloupolos</td>
<td>24/34</td>
<td>15/15</td>
<td></td>
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<tr>
<td>Overall</td>
<td>24/34</td>
<td>15/15</td>
<td>-0.29 (-0.47 to -0.12)</td>
</tr>
<tr>
<td>Thrombolysis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>1415/2878</td>
<td>2890/4320</td>
<td>-0.18 (-0.20 to -0.16)</td>
</tr>
</tbody>
</table>

18% decrease in 30-day mortality

6% increase in 30-day mortality

IABP-SHOCK 2

Results
Primary Study Endpoint (30-Day Mortality)

- Control: 41.3%
- IABP: 39.7%

P = 0.92 by log-rank test
Relative risk 0.96; 95% CI 0.79-1.17; P = 0.69 by Chi²-Test

Time after Randomization (Days)

Mortality (%)
Currently available devices

What about the other available devices

Scientific evidence?
LVAD – The guidelines

“A hemodynamic support device is recommended for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacological therapy (384,424–427). Level of Evidence: IB”
## Scientific evidence & Guidelines

<table>
<thead>
<tr>
<th></th>
<th>IABP</th>
<th>Impella 2.5/3.5</th>
<th>TandemHeart</th>
<th>Mini ECMO</th>
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</thead>
<tbody>
<tr>
<td><strong>Elective HR-PCI</strong></td>
<td>Brigouiri (LM) cohort n=219</td>
<td>± PROTECT I n=20</td>
<td>+ Case series</td>
<td>Case series</td>
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<tr>
<td></td>
<td>Brigouiri (3-VD) cohort n=133</td>
<td>± Europella n=144</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>BCIS-1 RCT n=301</td>
<td>± PROTECT II RCT n=654</td>
<td>±</td>
<td></td>
</tr>
<tr>
<td><strong>STEMI</strong></td>
<td>Sjauw Meta-analysis n=1009</td>
<td>± Sjauw MACH2 n=20</td>
<td>+ Case series</td>
<td>Case series</td>
</tr>
<tr>
<td></td>
<td>CRISP-AMI RCT n=300</td>
<td>± Mini-AMI RCT n=50</td>
<td></td>
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<tr>
<td><strong>STEMI CS</strong></td>
<td>Sjauw Meta-analysis n=10529</td>
<td>± ISAR-Shock RCT n=26</td>
<td>+ Burkhoff RCT n=33</td>
<td>Case series</td>
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<tr>
<td></td>
<td>Prondzinsky RCT n=40</td>
<td>± IMPRESS in severe shock RCT</td>
<td>- Thiele RCT n=40</td>
<td>± Kar n=117</td>
</tr>
<tr>
<td></td>
<td>IABP-SHOCK 2 RCT n=600</td>
<td>±</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Routine IABP for HR-PCI and STEMI not supported by current evidence. In the setting of CS expert opinion (ESC class IC; ACC/AHA class IB)

New devices promising. However, results from RCTs awaited.

Especially in CS indications for device Tx still open for debate/research i.e. bridge to transplant, bridge to LVAD, bridge to decision...

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Feasible: +
Inconclusive / debatable: ±
Negative: −
Enrolling: 🧑‍⚕️
AMC Impella experience

The Impella 2.5 and 5.0 devices for ST-elevation myocardial infarction patients presenting with severe and profound cardiogenic shock: The Academic Medical Center intensive care unit experience*

Annemarie E. Engström, MD; Ricardo Cocchieri, MD; Antoine H. Driessen, MD; Krischan D. Sjauw, MD; Marije M. Vis, MD; Jan Baan, MD, PhD; Mark de Jong, RN; Wim K. Lagrand, MD, PhD; Jos A. P. van der Sloot; Jan G. Tijssen; Robbert J. de Winter; Bas A. S. de Mol; Jan J. Piek; José P. J. M. Henriques, MD, PhD

Conclusion

IABP therapy in addition to primary PCI for patients with AMI complicated by cardiogenic shock has no effect on mortality. (IABP-SHOCK 2, Thiele et al.)

The use of more potent (left) ventricular assist devices will be topic of debate in determining the optimal treatment strategy in CS. (Perhaps the newly CE-marked Impella cVAD 3.5 L support device, preferable more support.)

Furthermore the revascularization strategy, i.e. culprit vs. complete revascularization will be an important issue to resolve in upcoming trials.
Thank you for your attention
Back up slides

Extra case
Case 2

♀ 50 years

History
Alcohol abuse

Presentation
Out of hospital cardiac arrest caused by VF → ROSC after 1 shock with AED. Poor hemodynamic state requiring inotropes for systolic blood pressures > 90 mmHg. Intubation.

ECG
Acute anterolateral infarction
Case 2

14-DEC-1960 (50 yr)
Female

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>121 BPM</td>
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<tr>
<td>PR interval</td>
<td>122 ms</td>
</tr>
<tr>
<td>QRS duration</td>
<td>84 ms</td>
</tr>
<tr>
<td>QT/QTc</td>
<td>352/499 ms</td>
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<tr>
<td>P-R-T axes</td>
<td>77 -43 48</td>
</tr>
</tbody>
</table>

Referrer by: [Signature]
Confirmed by: NON SUPERVISSED

Academic Medical Center, Amsterdam, The Netherlands
Case 2 - CAG

Severe ostial LMCA lesion with aspect of thrombus
Distal LAD occlusion with aspect of thrombus embolisation
Case 2

Hospital course

Cath. Lab:
After discussion with cardiac thoracic surgeon emergent CABG. Risk of systemic embolization considered to high with PCI.

Operating room:
- Thrombectomy → macroscopic visible thrombus in LMCA ostium (histology: fresh thrombus).
- Successful CABG: LIMA-LAD, Ao-Mo.
- Insertion of Impella 5.0 because of persisting cardiogenic shock.

ICU:
- Acute kidney injury, requiring Continuous Veno-Venous Hemofiltration.
- Gradual recovery of LV function and kidney function.
- 9 days after acute event weaning from Impella 5.0 en removal of device
STEMI in patient with MVD

Dariusz Dudek, FESC

Institute of Cardiology
Jagiellonian University Medical College
Krakow, Poland
Clinical characteristics

- 39-year old male
- positive family history of CAD – brother
- non-smoker, no additional CAD risk factors
- no other comorbidities

- symptoms from 12 hours
- continuous, however mild chest pain at rest

- HR 100 / min. BP 80/60 mmHg
ECG teletransmission from the field

- ECG: ST-segment elevation in II, III, aVF – suspicion of AMI of the inferior wall, confirmed by ECG teletransmission
- expected delay to cathlab admission 30-35 minutes
ECG during admission to catheterization lab.

- Direct transfer to cathlab bypassing Emergency Room
- ECG: ST-segment elevation in II, III, aVF; ST-segment depression in I, aVL, V1-V6 precordial leads
- Diagnosis: acute myocardial infarction of the inferior wall
Coronary angiography through radial access
Coronary angiography via left radial artery

diffuse disease in prox LAD, Dg and Cx

Thrombus?! / calcification?! in LAD mid
Coronary angiography via left radial artery
Coronary angiography through radial access

RCA – 100%, TIMI 0
(infarct related artery – IRA)
Voting Question

Patient with STEMI and multivessel disease

1. PCI

2. CABG

3. Medical
PCI; guiding catheter 6FJR4.0
problems with BMW wire passage before
intra coronary abciximab (Gp IIb/IIIa)

Distal flow restored and wire passage after
i.c. bolus of abciximab
PCI
Export 6F aspiration catheter

3 passages of thrombectomy catheter with thrombus on the filter

TIMI grade 3 flow after thrombectomy
IVUS assessment to find optimal length of the stent in diffuse disease

IVUS

DES stenting Promus Element 4.0x32 mm, 16 atm

IVUS-guided stenting – concept of “healthy to healthy segment” stenting coverage
PCI of RCA

Final result
Control ECG after PCI of RCA

- patient haemodynamically has improved
- BP 100 / 80 mmHg
- partial ST-segment resolution
Left coronary artery revascularization

diffuse disease in prox LAD, Dg and Cx

Thrombus?! / calcification?! in LAD mid
Left coronary artery revascularization

Patient haemodynamically stable, no chest pain after PCI

OPTIONS

Mode of further revascularization

1. Immediate PCI
2. Staged PCI of LCA
3. CABG
Primary PCI should be limited to the culprit vessel with the exception of cardiogenic shock and persistent ischaemia after PCI of the supposed culprit lesion. (IIa, B)

- There is no current evidence to support emergency intervention in non-IRA lesions.
- The only exceptions, when MVD PCI during acute STEMI is justified, are in patients with cardiogenic shock in the presence of multiple, truly critical (≥90% diameter) stenoses or highly unstable lesions (angiographic signs of possible thrombus or lesion disruption), and if there is persistent ischaemia after PCI of the supposed culprit lesion.
- However, in patients with MVD and cardiogenic shock, non-culprit lesions without critical stenoses should not routinely be stented.
Patient haemodynamically stable, no chest pain after PCI

MY SOLUTION

Revascularization strategy
- one-stage complete revascularization
- one-stage PCI of LAD
- staged revascularization
- staged revascularization after non-invasive tests for ischaemia

Mode of staged revascularization
- PCI
- CABG
In-hospital data

- max CK/MB level – 5184/709
- max. troponin I level – 14.94 ug/l
- EF – 47%
- Patient discharged after 6 days
Heart team discussion before control angiogram

- 1 month after PCI patient without symptoms of angina

Heart team decision

- PCI of left coronary team
Control angiogram – right coronary artery

RCA check:
no in-stent thrombosis and restenosis confirmed by ICUS & OCT

RPD – 80%
Control angiogram – left coronary artery

LAD 80% prox, 80% mid, Cx 80% dyst, Mg 100% ostium
Next steps…

OPTIONS

PCI
- LAD

Or

CABG
- LIMA-LAD
Next steps…

MY SOLUTION

PCI
• LAD

CABG
• LIMA-LAD

Finally patient checked for additional clinical and laboratory assessment, including thrombophilia

He will be discussed during the Heart Team meeting again for the final choice of LAD – revascularization:

PCI LAD vs CABG
STEMI in patient with MVD

Dariusz Dudek, FESC

Institute of Cardiology
Jagiellonian University Medical College
Krakow, Poland
Control angiogram – left coronary artery

LAD 80% prox, 80% mid, Cx 80% dyst, Mg 100% ostium
Patient with postinfarction VSD and cardiogenic shock

Holger Thiele

University of Leipzig – Heart Center
Research Funding:
- Terumo
- Lilly
- Maquet Cardiovascular
- Teleflex Medical

Consulting:
- Maquet Cardiovascular
- Lilly

Speaker Honoraria:
- Lilly
- Astra Zeneca
- Daiichi Sankyo
- Boehringer Ingelheim
- Maquet Cardiovascular
- Medicines Company
STEMI Late Presenter:

13 h after symptom onset PCI
STEMI Late Presenter:

13 h after symptom onset PCI

Post thrombectomy

Final result
New systolic murmur: VSD Complicating AMI 3 days later

Incidence 0.2% after AMI
Surgical Mortality

Single-Center Trials; all patients included?
Surgical Repair of Post MI VSD

Cumulative proportion surviving

Years

 Patients at risk

$189$ $87$ $58$ $30$ $4$

Surgical Mortality

Thirty-day mortality for surgery at different time intervals after VSD diagnosis

Cardiothoracic Surgeon Advice:
Stabilization until surgery in 3-4 weeks

ACC/AHA and ESC-Guidelines:
VSD-Closure immediately irrespective of patient’s hemodynamic status

Question

When do surgeons operate VSD in your institution?

a) Immediately as guidelines recommend

b) Delayed for 3-4 weeks
Patients with VSD (n=87)

Cardiogenic shock (n=55)
- Surgery (n=23)
  - Mortality (n=20, 87%)
- No Surgery (n=22)
  - Mortality (n=22, 100%)

No cardiogenic shock (n=32)
- Surgery (n=28)
  - Mortality (n=12, 43%)
- No Surgery (n=4)
  - Mortality (n=4, 100%)
VSD Complicating AMI 3 days later

Hemodynamic situation:
Blood pressure: 80/50 mmHg, Heart rate: 102/min

Serum lactate: 5.2 mmol/l

-> cardiogenic shock
-> IABP insertion
-> Decision for interventional closure
# Initial IABP Effects

<table>
<thead>
<tr>
<th></th>
<th>pre IABP</th>
<th>post IABP</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eff. Cardiac output (l/min)</td>
<td>3.6 ± 1.3</td>
<td>4.2 ± 1.3</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Eff. Cardiac Index (l/min/qm)</td>
<td>1.9 ± 0.7</td>
<td>2.2 ± 0.7</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Qp:Qs</td>
<td>3.1 ± 0.9</td>
<td>2.5 ± 0.7</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Shunt volume (l/min)</td>
<td>7.3 ± 3.5</td>
<td>5.6 ± 3.3</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Mean blood pressure (mmHg)</td>
<td>66 ± 16</td>
<td>75 ± 14</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>PAP mean (mmHg)</td>
<td>33 ± 5</td>
<td>28 ± 5</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>20 ± 7</td>
<td>16 ± 6</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>17 ± 4</td>
<td>14 ± 4</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>108 ± 20</td>
<td>102 ± 18</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Lactate (mmol/l)</td>
<td>2.2 ± 1.2</td>
<td>1.8 ± 1.1</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>BE (mmol/l)</td>
<td>-2.8 ± 4.9</td>
<td>-2.0 ± 4.3</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>pH</td>
<td>7.39 ± 0.1</td>
<td>7.42 ± 0.1</td>
<td>p &lt; 0.005</td>
</tr>
</tbody>
</table>

N=23

Thiele et al. Am J Cardiol 2003, 92:450-454
Surgical Repair Postinfarction VSD

Cardiogenic Shock

Mortality (%)

- GUSTO-1: 81%
- SHOCK-Registry: 100%

Interventional Closure VSD

Thiele et al, Eur Heart J 2009;30:81-88
Is percutaneous VSD repair performed at your institution?

a) Yes

b) No
Interventional Closure I
Interventional Closure II
Interventional Closure III
Closure Devices

ASD / Septal - Occluder 1,5 mm (4-54 mm)

Muscular VSD - Occluder 7 mm (4-16 mm)

Muscular VSD - Occluder P.I. 10 mm (16-24 mm)
MRI Post-VSD Closure

Thiele et al, Eur Heart J 2006; 27:1136
### Interventional Closure VSD – Literature Overview

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Acute phase (N)</th>
<th>Subacute/chronic phase (N)</th>
<th>Mean time AMI – closure in days (range)</th>
<th>Primary/secondary VSD closure (N)</th>
<th>Success rate (%)</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holzer et al(^46)</td>
<td>18</td>
<td>6</td>
<td>12</td>
<td>25 (2-95)</td>
<td>8/10</td>
<td>89</td>
<td>39</td>
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<tr>
<td>Goldstein et al(^30)</td>
<td>4</td>
<td>0</td>
<td>4</td>
<td>58 (15-108)</td>
<td>0/4</td>
<td>75</td>
<td>25</td>
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<tr>
<td>Szkutnik et al(^33)</td>
<td>7</td>
<td>0</td>
<td>7</td>
<td>54 (14-70)</td>
<td>6/1</td>
<td>71</td>
<td>20</td>
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<tr>
<td>Chessa et al(^40)</td>
<td>12</td>
<td>3</td>
<td>9</td>
<td>-</td>
<td>7/5</td>
<td>83</td>
<td>40</td>
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<tr>
<td>Martinez et al(^47)</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>6 (1-16)</td>
<td>3/2</td>
<td>100</td>
<td>20</td>
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<tr>
<td>Bialkowski et al(^29)</td>
<td>19</td>
<td>1</td>
<td>18</td>
<td>62 (14-336)</td>
<td>17/2</td>
<td>95</td>
<td>31</td>
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<tr>
<td>Leipzig experience</td>
<td>22</td>
<td>19</td>
<td>3</td>
<td>6 (1-26)</td>
<td>22/0</td>
<td>78</td>
<td>68</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>87</td>
<td>32</td>
<td>55</td>
<td>35 (1-336)</td>
<td>63/24</td>
<td>84</td>
<td>35</td>
</tr>
</tbody>
</table>
Mean time AMI-VSD-Closure 5 ± 2 days

Patients with PIVSD (n=29)

Shock (n=15)
Mortality 14/15 (93%)

Total (n=29)
Mortality 19/29 (65%)

No Shock (n=14)
Mortality 5/14 (36%)

Thiele et al, Eur Heart J 2009;30:81-88
### Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Shock (N=15)</th>
<th>Non-Shock (N=13)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>72±8</td>
<td>71±9</td>
<td>0.76</td>
</tr>
<tr>
<td><strong>Male/Female</strong></td>
<td>9/6</td>
<td>7/6</td>
<td>0.71</td>
</tr>
<tr>
<td><strong>Hypertension (n; %)</strong></td>
<td>13 (87)</td>
<td>10 (77)</td>
<td>0.86</td>
</tr>
<tr>
<td><strong>HLP (n; %)</strong></td>
<td>9 (60)</td>
<td>5 (38)</td>
<td>0.45</td>
</tr>
<tr>
<td><strong>Smoking (n; %)</strong></td>
<td>4 (27)</td>
<td>3 (23)</td>
<td>1.0</td>
</tr>
<tr>
<td><strong>Diabetes mellitus (n; %)</strong></td>
<td>8 (53)</td>
<td>6 (46)</td>
<td>0.93</td>
</tr>
<tr>
<td><strong>Occurrence VSD (days)</strong></td>
<td>1.5±1.2</td>
<td>4.0±2.8</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>VSD – Closure (days)</strong></td>
<td>1.1±1.5</td>
<td>5.8±5.4</td>
<td>0.02</td>
</tr>
<tr>
<td><strong>Extent CAD (1/2/3)</strong></td>
<td>8/8/6</td>
<td>5/4/4</td>
<td>0.85</td>
</tr>
<tr>
<td><strong>Anterior MI (n; %)</strong></td>
<td>7 (47)</td>
<td>7 (54)</td>
<td>1.0</td>
</tr>
<tr>
<td><strong>Qp:Qs</strong></td>
<td>3.5±1.2</td>
<td>2.5±1.1</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>Lactate</strong></td>
<td>4.5±3.2</td>
<td>1.8±1.2</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Thiele et al, Eur Heart J 2009;30:81-88
Overall-Mortality Interventional VSD Closure

Cumulative proportion surviving

Thiele et al, Eur Heart J 2009;30:81-88
30 Day Mortality Interventional VSD Closure

Cumulative Survival (%)

No cardiogenic shock

Cardiogenic Shock

P<0.001

Total implantations n = 29

Successful implantation (n=25)
- Dislocation of device (n=3)
- Qp:Qs >1.5:1 (n=4)
- AV III° (n=1)

Unsuccessful implantation (n=4)
- VSD too large for device (n=1)
- Dislocation of device (n=1)
- LV-rupture (n=1)

LV-rupture (n=2)
- Surgery (n=2)

Surgery (n=2)

Surgery (n=1)
TTE: Device Embolization into RV

Cobra-like Deformation

Persistent Shunt after Closure

Limitations of Current Devices

- Size of devices (Complex structure of VSD)
- Removal of guidewire
- Rigid delivery sheath
- Kinking of delivery sheath
- No immediate closure (device design)

Bridge by LVAD?
Summary and Conclusions

- IABP as bridge to closure in VSD (Hemodynamic effects positive, outcome?)
- Interventional VSD closure feasible
- Very demanding + difficult procedure
- High mortality (cardiogenic shock)
- In many patients futile situation! (Surgery and Interventional approach!)
- Interventional closure may allow immediate closure directly after initial diagnosis + might serve as definite treatment and/or bridge-to-surgery, if surgery is not performed immediately
Thank you for your attention

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Patient with postinfarction VSD and cardiogenic shock

Holger Thiele

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