PHARMACOLOGICAL MANAGEMENT OF CARDIOGENIC SHOCK

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Shock Categories

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

Shock Registry
Hochman, JACC 36: 1063, 2000
The overall prevalence of post MI cardiogenic shock has remained relatively steady over the past 30 years at about 6-7%.

Despite improvements in management, mortality remains very high (around 50% @ 30d).

However, substantial progress is being made
Swiss registry, about 24,000 patients.
Trends in Management and Outcomes of Patients With Acute Myocardial Infarction Complicated by Cardiogenic Shock

Anvar Babaev; Paul D. Frederick; David J. Pasta; et al.


**Figure 2.** Revascularization Rates in Patients With Cardiogenic Shock at Presentation (n=7356)

**Table 2.** In-Hospital Mortality Rates

<table>
<thead>
<tr>
<th>Year</th>
<th>Total (No.)</th>
<th>&lt;75 y (No.)</th>
<th>&gt;75 y (No.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1995</td>
<td>434 (60.3)</td>
<td>274 (56.8)</td>
<td>160 (69.9)</td>
</tr>
<tr>
<td>1996</td>
<td>510 (59.8)</td>
<td>290 (61.4)</td>
<td>220 (76.1)</td>
</tr>
<tr>
<td>1997</td>
<td>530 (60.7)</td>
<td>313 (53.3)</td>
<td>217 (75.0)</td>
</tr>
<tr>
<td>1998</td>
<td>413 (58.0)</td>
<td>225 (49.2)</td>
<td>188 (73.7)</td>
</tr>
<tr>
<td>1999</td>
<td>554 (55.9)</td>
<td>324 (50.3)</td>
<td>230 (56.3)</td>
</tr>
<tr>
<td>2000</td>
<td>475 (56.6)</td>
<td>258 (47.9)</td>
<td>217 (72.1)</td>
</tr>
<tr>
<td>2001</td>
<td>416 (52.1)</td>
<td>222 (43.9)</td>
<td>194 (56.4)</td>
</tr>
<tr>
<td>2002</td>
<td>399 (49.8)</td>
<td>187 (40.8)</td>
<td>152 (38.5)</td>
</tr>
<tr>
<td>2003</td>
<td>282 (51.3)</td>
<td>162 (44.7)</td>
<td>120 (33.8)</td>
</tr>
<tr>
<td>2004†</td>
<td>163 (47.9)</td>
<td>86 (39.5)</td>
<td>75 (34.1)</td>
</tr>
</tbody>
</table>

*P* value: <.001 <.001 <.001
### Table 7: Outcomes and LOS in EHFS II

<table>
<thead>
<tr>
<th>Variable</th>
<th>All</th>
<th>ADCHF</th>
<th>De novo AHF</th>
<th>Decomp. HF</th>
<th>Pulm. oedema</th>
<th>Cardiog. shock</th>
<th>Hypert. HF</th>
<th>Right HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital mortality (n=)</td>
<td>239/3580</td>
<td>131/2250</td>
<td>108/1329</td>
<td>116/2340</td>
<td>53/581</td>
<td>55/139</td>
<td>6/407</td>
<td>9/113</td>
</tr>
<tr>
<td>LOS days, median (IQR)</td>
<td>9 (6-14)</td>
<td>9 (6-14)</td>
<td>9 (5-15)</td>
<td>9 (6-15)</td>
<td>10 (6-15)</td>
<td>10 (4-17)</td>
<td>8 (6-12)</td>
<td>11 (7-17)</td>
</tr>
<tr>
<td>% staying in ICU/CCU</td>
<td>51.0</td>
<td>46.3</td>
<td>59.0</td>
<td>44.4</td>
<td>76.0</td>
<td>92.7</td>
<td>41.3</td>
<td>43.6</td>
</tr>
<tr>
<td>ICU/CCU stay days, median (IQR)</td>
<td>3 (2-5)</td>
<td>3 (2-5)</td>
<td>3 (2-5)</td>
<td>3 (2-5)</td>
<td>3 (2-5)</td>
<td>4 (2-8)</td>
<td>3 (1-5)</td>
<td>3 (2-5)</td>
</tr>
<tr>
<td>Life-threatening arrhythmias (%)</td>
<td>4.7</td>
<td>3.9</td>
<td>6.2</td>
<td>3.6</td>
<td>7.2</td>
<td>24.5</td>
<td>1.7</td>
<td>2.7</td>
</tr>
</tbody>
</table>

Median LOS reported as for all patients (including in-hospital deaths). Median LOS in ICU/CCU for patients admitted to these units during index hospitalization.
GOALS OF THERAPY IN CARDIOGENIC SHOCK BEYOND REVASCULARIZATION

• Immediate:
  – Raise cardiac output, tissue perfusion and blood pressure
  – Tissue perfusion more important than blood pressure!
  – Maintain organ function until LV remodeling and resolution of stunning and of the inflammatory state allow weaning of support

• Late: Improve patient outcome – not shown with any pharmacological agent
THERAPEUTIC STRATEGY IN CARDIOGENIC SHOCK

• Early revascularization – the only modality shown to improve outcome

• Mechanical support (mostly IABP)
  Associated with reduced mortality in NRMI-2 and in the SHOCK trial, no randomized data.

• Pharmacological support

• A number of agents used traditionally as well as some newer ones. No placebo controlled trials.

• No comparative studies between IABP and pharmacological support; the substantial increase in coronary and peripheral perfusion without an increase in oxygen demand with IABP make it a more attractive first tool to stabilize the patient
POSITIVE INOTROPIC AGENTS

• Act by increasing intracellular calcium thus promoting myocyte contraction
• Increase oxygen demand, a major drawback in patients with coronary artery disease.
• Hypothetically, positive inotropes might be better tolerated in patients who had adequate revascularization.
## MAJOR CATECHOLAMINES IN USE

<table>
<thead>
<tr>
<th></th>
<th>β1</th>
<th>β2</th>
<th>α</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>++ (main effect at medium dose)</td>
<td>+</td>
<td>++ (effect increases with dose)</td>
<td>+ (only important effect at low dose)</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>++</td>
<td>+</td>
<td>+++</td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td>++</td>
<td>++</td>
<td>++</td>
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</table>
### MAJOR INOTROPIC AGENTS IN USE

<table>
<thead>
<tr>
<th></th>
<th>HR/arrhythmia</th>
<th>SBP</th>
<th>PCW</th>
<th>Afterload</th>
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</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>↑↑</td>
<td>↑↑</td>
<td>↑</td>
<td>↑ (moderate – high dose)</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↓ (mainly at lower doses)</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>↑</td>
<td>↑↑</td>
<td>↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Levosimendan</td>
<td>↑↓</td>
<td>↑↓</td>
<td>↓↓</td>
<td>↓↓</td>
</tr>
<tr>
<td>Milrinone/Enoxime</td>
<td>↑↓</td>
<td>↑</td>
<td>↓↓↓</td>
<td>↓↓</td>
</tr>
</tbody>
</table>
Original Article

Comparison of Dopamine and Norepinephrine in the Treatment of Shock

Daniel De Backer, M.D., Ph.D., Patrick Biston, M.D., Jacques Devriendt, M.D., Christian Madl, M.D., Didier Chochrad, M.D., Cesar Aldecoa, M.D., Alexandre Brasseur, M.D., Pierre Defrance, M.D., Philippe Gottignies, M.D., Jean-Louis Vincent, M.D., Ph.D., for the SOAP II Investigators

N Engl J Med
Volume 362(9):779-789
March 4, 2010
Screening and Enrollment

2011 Patients were assessed for eligibility

332 Were excluded
94 Had arrhythmia
79 Had shock lasting >4 hr
73 Were not enrolled by their physician
38 Had major therapeutic limitation
20 Had been included in the study previously
16 Were <18 yr of age
12 Were brain-dead

CS: N=280

1679 Underwent randomization

838 Were assigned to receive dopamine
838 Were included in intention-to-treat analysis

821 Were assigned to receive norepinephrine
821 Were included in intention-to-treat analysis

Kaplan-Meier Curves for 28-Day Survival in the Intention-to-Treat Population

LEVOSIMENDAN

- Calcium sensitizer, leads to a positive inotropic effect and peripheral vasodilatation
- May cause severe hypotension
- Not shown to be superior to dobutamine in ADHF (SURVIVE).
- Does it have a role in cardiogenic shock?
• 25 patients with CS persisting > 24h post revascularization, IABP in 13. Levosimendan added.
LEVOSIMENDAN IN CARDIOGENIC SHOCK

Patients with acute myocardial infarction (STEMI/NSTEMI) and refractory CS following preliminary haemodynamic support (dobutamine +/- norepinephrine) and primary PCI of the infarct-related artery

Additional levosimendan infusion (bolus 12 µg/kg, then 0.1 µg/kg for 24 h) (n=10)

Additional IABP insertion (n=12)

Haemodynamic monitoring for 48 hours; continuous haemodynamic support as required (e.g. PAOP 18 mmHg, MAP > 65 mmHg)

Cardiac Index (CI, l/min/m²)

Pre-treatment (0-) 3 h 24 h 48 h

(a)

Cardiac Power Index (CI x RRI mean x 0.0022)

Pre-treatment (0-) 3 h 24 h 48 h

(b)

Pulmonary artery occlusion pressure (PAOP)

Pre-treatment (0-) 3 h 24 h 48 h

(c)

SOFA score

Day 1 Day 2 Day 3

(d)

Christoph et al. Ac. Cardiac Care 2008;10:49
Levosimendan is superior to enoximone in refractory cardiogenic shock complicating acute myocardial infarction*

- A randomized trial of 32 patients with refractory CS, all had revascularization and IABP

A randomized trial of 22 patients with CS, 12 months follow up
LEVOSIMENDAN IN CARDIOGENIC SHOCK

- Preliminary data seem to indicate that levosimendan may have a role in cardiogenic shock. However, since this agent may exacerbate hypotension, much larger studies are needed to assess its efficacy and safety and to identify eligible patients.
Phosphodiesterase inhibitors: Milrinone

- Increases CO, peripheral vasodilator
- May be arrhythmogenic.
- May increase mortality in severe heart failure
- Despite extensive experience in severe heart failure and in surgical patients, no studies in post MI cardiogenic shock.
## USE OF PHARMACOLOGICAL AGENTS IN EUROPE

Table 5: Acute cardiac care by clinical class

<table>
<thead>
<tr>
<th>Treatment % performed</th>
<th>Total</th>
<th>Decomp. HF</th>
<th>Pulmonary oedema</th>
<th>Cardiogenic shock</th>
<th>Hypert. HF</th>
<th>Right HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venlatory support&lt;sup&gt;a&lt;/sup&gt;</td>
<td>13.9</td>
<td>8.1</td>
<td>31.5</td>
<td>56.1</td>
<td>7.4</td>
<td>14.2</td>
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<tr>
<td>Invasive mechanical ventilation</td>
<td>5.1</td>
<td>2.3</td>
<td>11.0</td>
<td>36.7</td>
<td>1.7</td>
<td>4.4</td>
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<tr>
<td>Diuretic</td>
<td>92.9</td>
<td>94.6</td>
<td>97.6</td>
<td>77.5</td>
<td>82.8</td>
<td>88.5</td>
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<tr>
<td>Oral</td>
<td>8.6</td>
<td>10.3</td>
<td>3.6</td>
<td>0.0</td>
<td>8.7</td>
<td>8.0</td>
</tr>
<tr>
<td>IV bolus</td>
<td>72.1</td>
<td>71.7</td>
<td>81.9</td>
<td>58.7</td>
<td>68.6</td>
<td>58.4</td>
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<tr>
<td>Infusion</td>
<td>12.3</td>
<td>12.6</td>
<td>12.1</td>
<td>18.8</td>
<td>5.5</td>
<td>22.1</td>
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<tr>
<td>Beta-blocker</td>
<td>10.1</td>
<td>10.4</td>
<td>8.3</td>
<td>9.4</td>
<td>11.1</td>
<td>10.6</td>
</tr>
<tr>
<td>Opioids</td>
<td>19.4</td>
<td>13.5</td>
<td>38.3</td>
<td>49.3</td>
<td>18.2</td>
<td>10.7</td>
</tr>
<tr>
<td>IV nitrate</td>
<td>37.8</td>
<td>30.4</td>
<td>70.6</td>
<td>36.5</td>
<td>39.7</td>
<td>8.6</td>
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<tr>
<td>IV nitroprusside</td>
<td>0.9</td>
<td>0.5</td>
<td>2.1</td>
<td>2.2</td>
<td>1.2</td>
<td>0.0</td>
</tr>
<tr>
<td>IV inotrope</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Adrenaline</td>
<td>1.8</td>
<td>1.2</td>
<td>2.6</td>
<td>15.8</td>
<td>0.0</td>
<td>1.8</td>
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<tr>
<td>Dobutamine</td>
<td>10.2</td>
<td>8.6</td>
<td>13.3</td>
<td>44.6</td>
<td>2.0</td>
<td>14.2</td>
</tr>
<tr>
<td>Dopamine</td>
<td>11.3</td>
<td>8.5</td>
<td>15.8</td>
<td>65.5</td>
<td>2.2</td>
<td>12.4</td>
</tr>
<tr>
<td>Levosimendan</td>
<td>3.9</td>
<td>4.4</td>
<td>3.8</td>
<td>7.9</td>
<td>0.2</td>
<td>0.9</td>
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<td>Noradrenaline</td>
<td>2.6</td>
<td>1.2</td>
<td>4.5</td>
<td>24.5</td>
<td>0.7</td>
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<tr>
<td>Amiodarone</td>
<td>17.5</td>
<td>16.8</td>
<td>18.8</td>
<td>32.1</td>
<td>14.8</td>
<td>16.8</td>
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<td>Heparin (UFH)</td>
<td>18.7</td>
<td>17.8</td>
<td>18.8</td>
<td>45.7</td>
<td>15.9</td>
<td>15.2</td>
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<td>LMWH</td>
<td>41.0</td>
<td>38.3</td>
<td>54.6</td>
<td>37.0</td>
<td>37.8</td>
<td>43.8</td>
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<tr>
<td>Blood transfusion</td>
<td>5.9</td>
<td>5.2</td>
<td>7.9</td>
<td>10.1</td>
<td>3.5</td>
<td>12.4</td>
</tr>
<tr>
<td>PCI</td>
<td>8.4</td>
<td>6.4</td>
<td>10.2</td>
<td>40.6</td>
<td>7.0</td>
<td>7.1</td>
</tr>
<tr>
<td>CABG</td>
<td>1.8</td>
<td>1.6</td>
<td>3.1</td>
<td>4.3</td>
<td>0.5</td>
<td>0.9</td>
</tr>
<tr>
<td>IABP</td>
<td>2.2</td>
<td>1.2</td>
<td>1.4</td>
<td>30.9</td>
<td>0.5</td>
<td>0.0</td>
</tr>
<tr>
<td>Pacemaker</td>
<td>2.7</td>
<td>2.4</td>
<td>3.8</td>
<td>5.0</td>
<td>1.7</td>
<td>3.5</td>
</tr>
<tr>
<td>ICD</td>
<td>1.2</td>
<td>1.4</td>
<td>0.9</td>
<td>2.9</td>
<td>0.5</td>
<td>0.0</td>
</tr>
</tbody>
</table>

<sup>a</sup>Ventilatory support = continuous positive airway pressure/non-invasive positive pressure ventilation/invasive mechanical ventilation (intubated).

UFH, unfractionated heparin; LMWH, low-molecular-weight heparin; ICD, implantable cardioverter/defibrillator.
Despite younger age, lower rate of anterior MI, prevalence of single vessel disease, and similar benefit from revascularization, mortality was similar in RV and LV shock.

Jacobs, JACC 41: 1273, 2003
Hemodynamic Effects of Inhaled Nitric Oxide in Right Ventricular Myocardial Infarction and Cardiogenic Shock

Ignacio Inglessis, MD,* Jordan T. Shin, MD,*† John J. Lepore, MD,* Igor F. Palacios, MD, FACC,* Warren M. Zapol, MD;‡ Kenneth D. Bloch, MD,*† Marc J. Semigran, MD*
HEMODYNAMIC OBSERVATIONS FROM THE SHOCK TRIAL

- Average LVEF is only around 30\%, with a wide range, similar to many stable CHF patients.
- Average SVR on pressors is not elevated (around 1350-1400 dynes-sec-cm\(^{-5}\)) with a very wide range
- A SIRS like syndrome is frequent

Hochman J. Circulation 2003;107:2998
A systemic inflammatory response syndrome (SIRS) may be triggered by a large MI, with elevation of inflammatory cytokines (e.g., IL-6), as seen in septic shock.

High cytokine levels are associated with the development of cardiogenic shock.

Inflammatory cytokines increase expression of inducible nitric oxide synthase (iNOS) leading to high levels of nitric oxide (NO).
Experimental data demonstrate that high levels of nitric oxide:

- Reduce contractility
- Reduce catecholamine responsivity
- Induce inappropriate systemic vasodilation

Excess NO may play a role in the genesis and persistence of cardiogenic shock
• **30 patients with refractory CS randomized to an iNOS inhibitor (L-NAME) or supportive care**

• **Single center, no placebo control**

• **Baseline urine output: 75-122 cc/h (thus true shock questionable)**
LINCS: Mortality @ 7 & 30 days

Control
L-NMMA

% death

P=0.008

LINCS- L-NAME IN CARDIOGENIC SHOCK
The Tilarginine Acetate Injection in a Randomized International Study in Unstable Acute Myocardial Infarction Patients with Cardiogenic Shock (TRIUMPH)

Judith S. Hochman
on behalf of TRIUMPH Investigators

TRIUMPH was supported by ArgiNOx Pharmaceuticals, Inc.
New York University received a research grant from ArgiNOx for TRIUMPH
Judith S. Hochman, MD served as a consultant to Datascope

TRIUMPH report can be found at jama.ama-assn.org
TRIUMPH Eligibility

Myocardial Infarction + Refractory Shock + Persistent after PCI

Ischemic symptoms $\geq 30$ minutes with:
- Cardiac markers or
- ST-segment elevation or left bundle branch block

Peripheral signs of tissue hypoperfusion and
- SBP $< 100$ mmHg despite
- Vasopressor Rx
  - Dopamine $\geq 7$ mcg/kg/min
  - Norepinephrine $\geq 0.15$ mcg/kg/min
  - Epinephrine $\geq 0.15$ mcg/kg/min

and
- Clinical or hemodynamic evidence of $\uparrow$ LV EDP

- Patency of the infarct artery ($<70\%$ stenosis)
- LVEF $<40\%$
- Shock persisting $\geq 1$ hour after infarct artery patency

Hemodynamics and requirement for vasopressor treatment were reconfirmed just prior to study drug administration to exclude patients with rapidly resolving shock.
1611 Patients
Acute MI with
Cardiogenic Shock
entered in screening
log

398 Eligible, enrolled
≥1hr after IRA patency
documented

206 Tilarginine

201 (97.6%) received study drug
SBP no longer qualifying (n=5)

197 (98.0%) 30-d follow-up complete
Lost to 30-d follow-up (n=4)

186 (94.4%) 6-m follow-up complete
Lost to 6-m follow-up (n=11)

190 Placebo

180 (94.7%) received study drug
SBP no longer qualifying (n=10)

178 (98.9%) 30-d follow-up complete
Lost to 30-d follow-up (n=2)

162 (91.0%) 6-m follow-up complete
Lost to 6-m follow-up (n=16)

Treatment Assignment unknown (n=2) 30 days: 1 died/1 survived
# In-Hospital Procedures

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Tilarginine (n=206)</th>
<th>Placebo (n=190)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCI, %</td>
<td>97</td>
<td>95</td>
<td>0.50</td>
</tr>
<tr>
<td>CABG, %</td>
<td>8.3</td>
<td>10.5</td>
<td>0.44</td>
</tr>
<tr>
<td>IABP, %</td>
<td>89</td>
<td>91</td>
<td>0.56</td>
</tr>
<tr>
<td>LV Assist Device, %</td>
<td>11.2</td>
<td>12.6</td>
<td>0.65</td>
</tr>
<tr>
<td>Cardiac Transplantation, %</td>
<td>0.5</td>
<td>0.6</td>
<td>&gt;0.99</td>
</tr>
</tbody>
</table>
Systolic Blood Pressure change from baseline to 2 hours

ALL  
< 75 years  
≥ 75 years

Interaction p=0.02

P=0.001

* All blood pressures recorded on support measures
Primary Endpoint
30-day Mortality

Mortality

Days from randomization

Tilarginine: Placebo
RR: 1.14
95% CI: 0.92-1.41
P=0.24

Tilarginine
Placebo

48%
42%
6-month Mortality

Mortality

Days from randomization

No. at risk:
Tilarginine: 204 104 89 86 84
Placebo: 188 106 82 76 73

P=0.80
Conclusions

- Tilarginine significantly increased arterial blood pressure. However, this did not correlate with nor translate into improved outcome.

- There may be no adequate surrogate outcome or marker for mortality in cardiogenic shock complicating MI and new agents must be evaluated for their effect on mortality.
## ESC Guidelines for the Management of Cardiogenic Shock

<table>
<thead>
<tr>
<th>Intervention</th>
<th>CLASS</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early revascularization</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>IABP</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Dopamine</td>
<td>IIb</td>
<td>B</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>PA catheter</td>
<td>IIb</td>
<td>C</td>
</tr>
</tbody>
</table>

**ESC 2008 STEMI Guidelines**
Emergency Management of Complicated STEMI

Clinical signs: Shock, hypoperfusion, congestive heart failure, acute pulmonary edema
Most likely major underlying disturbance?

Acute Pulmonary Edema

Administer
- Furosemide IV 0.5 to 1.0 mg/kg
- Morphine IV 2 to 4 mg
- Oxygen/intubation as needed
- Nitroglycerin SL, then 10 to 20 mcg/min IV if SBP greater than 100 mm Hg
- Dopamine 5 to 15 mcg/kg per minute IV if SBP 70 to 100 mm Hg and signs/symptoms of shock present
- Dobutamine 2 to 20 mcg/kg per minute IV if SBP 70 to 100 mm Hg and no signs/symptoms of shock

Hypovolemia

Administer
- Fluids
- Blood transfusions
- Cause-specific interventions
Consider vasopressors

Check Blood Pressure

Low Output - Cardiogenic Shock

Systolic BP Greater than 100 mm Hg
Nitroglycerin 10 to 20 mcg/min IV

Systolic BP 70 to 100 mm Hg
NO signs/symptoms of shock
Dobutamine 2 to 20 mcg/kg per minute IV

Systolic BP 70 to 100 mm Hg
Signs/symptoms of shock
Dopamine 5 to 15 mcg/kg per minute IV

Systolic BP less than 70 mm Hg
Signs/symptoms of shock
Norepinephrine 0.5 to 30 mcg/min IV

Arrhythmia

Bradycardia
Tachycardia

See Section 7.7 in the ACC/AHA Guidelines for Patients With ST-Elevation Myocardial Infarction

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Shock, hypoperfusion, congestive heart failure, acute pulmonary edema

Most likely major underlying disturbance?

Check Blood Pressure

Systolic BP Greater than 100 mm Hg and not less than 30 mm Hg below baseline
ACE Inhibitors
Short-acting agent such as captopril (1 to 6.25 mg)

Further diagnostic/therapeutic considerations (should be considered in nonhypovolemic shock)

Diagnostic
- Pulmonary artery catheter
- Echocardiography
- Angiography for MI/ischemia
- Additional diagnostic studies

Therapeutic
- Intra-aortic balloon pump
- Reperfusion/revascularization

WHICH AGENT FOR CARDIOGENIC SHOCK?

- In the absence of mortality data no definitive recommendation can be made for any agent.
- Catecholamines: not convincingly shown to improve survival. Noradrenaline may be better than dopamine.
- Levosimendan: limited data in CS, not proven better than catecholamines. No reference in recent ESC guidelines in post MI CS
- No data on milrinone in CS
- In the meantime: use the IABP, and then use the minimal effective dose of an inotropic agent