Acute Heart Failure
Update 2012

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University of Antwerp
ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012

The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC.

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Acute Heart Failure - Definition

• Acute heart failure (AHF) is defined as a rapid onset or change in the signs and symptoms of heart failure, resulting in the need for urgent therapy.

• AHF may be either new heart failure or worsening of pre-existing chronic heart failure.

• Patients may present as a medical emergency such as acute pulmonary oedema or cardiogenic shock.
# Causes & precipitants of AHF

## Usually leading to rapid deterioration
- Rapid arrhythmia or severe bradycardia/conduction disturbance
- Acute coronary syndrome (ACS)
- Mechanical complication of ACS (e.g. VSR, MV chordal rupture, RV infarction)
- Acute pulmonary embolism
- Hypertensive crisis
- Cardiac tamponade
- Aortic dissection
- Surgery and perioperative problems
- Peripartum cardiomyopathy

## Usually leading to less rapid deterioration
- Infection (including infective endocarditis)
- Exacerbation of COPD/asthma
- Anaemia
- Kidney dysfunction
- Non-adherence to diet/drug therapy
- Iatrogenic causes (e.g. prescription of an NSAID or corticosteroid; drug interactions)
- Arrhythmias, bradycardia, and conduction disturbances not leading to sudden, severe change in heart rate
- Uncontrolled hypertension
- Hypothyroidism or hyperthyroidism
- Alcohol and drug abuse

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Acute Heart Failure Syndromes

De novo HF → No CAD → Preserved EF
Worsening Chronic HF* → No CAD → Preserved EF
Worsening Chronic HF* → CAD† → Reduced EF

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3 important questions to be asked during initial assessment:

- Does the patient have HF or is there an alternative cause for their symptoms and signs (e.g. chronic lung disease, anaemia, kidney failure, or pulmonary embolism)?
- If the patient does have HF, is there a precipitant and does it require immediate treatment or correction (e.g. an arrhythmia or acute coronary syndrome)?
- Is the patient's condition immediately life-threatening because of hypoxaemia or hypotension leading to underperfusion of the vital organs (heart, kidneys, and brain)?
Evaluation of pts. with suspected AHF

History/examination
(including blood pressure and respiratory rate)
- Chest X-ray
- ECG
- Echocardiogram or NP (or both)
- Oxygen saturation
- Blood chemistry
- Full blood count

Simultaneously assess for

Ventilation/systemic oxygenation inadequate
- Oxygen
- NIV
- ETT and invasive ventilation

Life-threatening arrhythmia/bradycardia
- Electrical cardioversion
- Pacing

Blood pressure <85 mmHg or shock
- Inotrope/vasopressor
- Mechanical circulatory support (e.g. IABP)

Acute coronary syndrome
- Coronary reperfusion
- Antithrombotic therapy

Acute mechanical cause/severe valvular disease
- Echocardiography
- Surgical/percutaneous intervention

Diagnosis and treatment are usually carried out in parallel, especially in patients who are particularly unwell, and management must be initiated promptly.

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Evaluation of pts. with suspected AHF

Diagnosis and treatment are usually carried out in parallel, especially in patients who are particularly unwell, and management must be initiated promptly.

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Treatment of acute heart failure: acute coronary syndromes

<table>
<thead>
<tr>
<th>Immediate primary PCI (or CABG in selected cases) is recommended if there is an ST elevation or a new LBBB ACS in order to reduce the extent of myocyte necrosis and reduce the risk of premature death.</th>
<th>I</th>
<th>A</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alternative to PCI or CABG:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intravenous thrombolytic therapy is recommended, if PCI/CABG cannot be performed, if there is ST-segment elevation or new LBBB, to reduce the extent of myocyte necrosis and reduce the risk of premature death.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Early PCI (or CABG in selected patients) is recommended if there is non-ST elevation ACS in order to reduce the risk of recurrent ACS. Urgent revascularization is recommended if the patient is haemodynamically unstable.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>An ACE inhibitor (or ARB) is recommended in patients with an EF ≤40%, after stabilization, to reduce the risk of death, recurrent myocardial infarction, and hospitalization for HF.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>A beta-blocker is recommended in patients with an EF ≤40%, after stabilization, to reduce the risk of death and recurrent myocardial infarction.</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>An i.v. opiate (along with an antiemetic) should be considered in patients with ischaemic chest pain to relieve this symptom (and improve breathlessness). Alertness and ventilatory effort should be monitored frequently after administration because opiates can depress respiration.</td>
<td>IIa</td>
<td>C</td>
</tr>
</tbody>
</table>
Evaluation of pts. with suspected AHF

- History/examination (including blood pressure and respiratory rate)
  - Chest X-ray
  - Echocardiogram or NP (or both)
  - Blood chemistry
- ECG
- Oxygen saturation
- Full blood count

Simultaneously assess for
- Ventilation/systemic oxygenation inadequate
- Life-threatening arrhythmia/bradycardia
- Blood pressure <85 mmHg or shock
- Acute coronary syndrome
- Acute mechanical cause/severe valvular disease

Urgent action if present
- Oxygen
- NIV
- ETT and invasive ventilation
- Electrical cardioversion
- Pacing
- Inotrope/vasopressor
- Mechanical circulatory support (e.g. IABP)
- Coronary reperfusion
- Antithrombotic therapy
- Echocardiography
- Surgical/percutaneous intervention

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Evaluation of pts. with suspected AHF

- History/examination (including blood pressure and respiratory rate)
- Chest X-ray
- Echocardiogram or NP (or both)
- Blood chemistry
- ECG
- Oxygen saturation
- Full blood count

Simultaneously assess for:

- Ventilation/systemic oxygenation inadequate?

Urgent action if present:

- Oxygen
- NIV
- ETT and invasive ventilation
- Electrical cardioversion
- Pacing
- Inotrope/vasopressor
- Mechanical circulatory support (e.g. IABP)
- Coronary reperfusion
- Antithrombotic therapy
- Echocardiography
- Surgical/percutaneous intervention

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### Treatment of acute heart failure: pulmonary congestion/oedema without shock

<table>
<thead>
<tr>
<th>Treatment Options</th>
<th>Recommendation Level</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Loop diuretics</strong></td>
<td>B</td>
</tr>
<tr>
<td><strong>High-flow oxygen</strong></td>
<td>C</td>
</tr>
<tr>
<td><strong>Thrombo-embolism prophylaxis</strong></td>
<td>I A</td>
</tr>
<tr>
<td><strong>Non-invasive ventilation</strong></td>
<td>IIa B</td>
</tr>
<tr>
<td><strong>I.V. opiate</strong></td>
<td>IIa C</td>
</tr>
<tr>
<td><strong>I.V. infusion of nitrate</strong></td>
<td>IIa B</td>
</tr>
<tr>
<td><strong>I.V. infusion of sodium nitroprusside</strong></td>
<td>IIb B</td>
</tr>
<tr>
<td><strong>Inotropic agents</strong></td>
<td>III C</td>
</tr>
</tbody>
</table>

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An i.v. loop diuretic is recommended to improve breathlessness and relieve congestion. Symptoms, function, and electrolytes should be monitored regularly during use of i.v. diuretic.

High-flow oxygen is recommended in patients with a capillary oxygen saturation (<90%) to correct hypoxaemia.

Thrombo-embolism prophylaxis (e.g. with LMWH) is recommended in patients with no contraindication to anticoagulation, to reduce the risk of deep venous thrombosis.

Non-invasive ventilation (e.g. CPAP) should be considered in dyspnoea respiratory rate >20 breaths/min to improve breathlessness and may reduce peak inspiratory pressure <85 mmHg (and blood pressure should be monitored regularly afterwards). In opioid-exposed patients to relieve these symptoms and improve breathlessness, the patient should be monitored frequently after administration because opioids can depress ventilation.

An I.V. infusion of nitrate should be considered in patients with pulmonary congestion/oedema and a systolic blood pressure >110 mmHg, who do not respond to further diuretics, to reduce pulmonary capillary wedge pressure and systemic vascular resistance and relieve congestion. Symptoms and blood pressure should be monitored frequently during administration.

An I.V. infusion of sodium nitroprusside may also relieve dyspnoea in these patients and blood pressure should be monitored frequently during administration. It is not recommended in patients with acute myocardial infarction.

Inotropic agents are NOT recommended if the patient is hypotensive (systolic blood pressure <85 mmHg), hypoperfused, or shocked because of safety concerns (atrial and ventricular arrhythmias, myocardial ischaemia, and death).

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Acute pulmonary oedema/congestion

1. Intravenous fluids of loop diuretics

2. Hypoaemia

   Yes → Oxygen

   No

3. Severe anxiety/distress

   Yes → Consider IV, opiates

   No

4. Measure systolic blood pressure

   SAP <65 mmHg or shock

   Yes → Add non vasodilating inotrope

   No → No additional therapy until response assessed

   SAP 65–110 mmHg

   No additional therapy until response assessed

   SAP >110 mmHg

   Consider vasodilator (e.g., NTG)

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Diuretic Strategies in Patients with Acute Decompensated Heart Failure: DOSE trial

Patients’ Global Assessment of Symptoms during the 72-Hour Study-Treatment Period.

A  Bolus vs. Continuous Infusion

- AUC with bolus infusions, 4236±1440
- AUC with continuous infusion, 4373±1404
- P=0.47

B  Low-Dose vs. High-Dose Strategy

- AUC with low-dose strategy, 4171±1436
- AUC with high-dose strategy, 4430±1401
- P=0.06

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GM Felker et al.
Diuretic Strategies in Patients with Acute Decompensated Heart Failure: DOSE trial

Mean Change in Serum Creatinine Level.

- Bolus: Change in Creatinine = 0.05, P = 0.45
- Continuous: Change in Creatinine = 0.07
- Low Dose: Change in Creatinine = 0.04, P = 0.21
- High Dose: Change in Creatinine = 0.08


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What are the implications of the DOSE trial?

• Since the DOSE trial demonstrated:
  – No benefit with a continuous infusion than with a regimen of intermittent boluses of diuretics.
  – A high dose of loop diuretics, as compared with a low dose, does not substantially worsen renal function.

• Therefore:
  – Since a high-dose regimen may relieve dyspnea more quickly without adverse effects on renal function, that regimen is preferable to a low-dose regimen.
  – Administration of boluses may be more convenient than continuous infusion and is equally effective.
In patients with acute pulmonary edema, NIV induces a more rapid improvement in respiratory distress and metabolic disturbance than does standard oxygen therapy but has no effect on short-term mortality. Therefore, NIV can be useful as adjunctive therapy.

Acute pulmonary oedema/congestion

Adequate response to treatment?  
- Yes  
  - Continue present treatment
- No
  - Re-evaluation of patient’s clinical status

SBP < 85 mmHg
- Yes  
  - Stop vasodilator
  - Stop beta-blocker if hypoperfused
  - Consider non-vasodilating inotrope or vasopressor
  - Consider right-heart catheterization
  - Consider mechanical circulatory support
- No

SpO₂ < 90%
- Yes  
  - Oxygen
  - Consider NIV
  - Consider ETT and invasive ventilation
- No

Urine output < 20 mL/h
- Yes
  - Bladder catheterization to confirm
  - Increase dose of diuretic or use combination of diuretics
  - Consider low-dose dopamine
  - Consider right-heart catheterization
  - Consider ultrafiltration
- No

CPAP = continuous positive airway pressure; ETT = endotracheal tube; i.v. = intravenous; NIPPV = non-invasive positive pressure ventilation; NIV = non-invasive ventilation; NTG = nitroglycerine; PaO₂ = partial pressure of oxygen; SBP = systolic blood pressure; SpO₂ = saturation of peripheral oxygen.

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Evaluation of pts. with suspected AHF

History/examination (including blood pressure and respiratory rate)
- Chest X-ray
- Echocardiogram or NP (or both)
- Blood chemistry

ECG
- Oxygen saturation
- Full blood count

Simultaneously assess for

Urgent action if present
- Ventilation/systemic oxygenation inadequate
  - Oxygen
  - NIV
  - ETT and invasive ventilation
- Life-threatening arrhythmia/bradycardia
  - Electrical cardioversion
  - Pacing
- Blood pressure <85 mmHg or shock
  - Inotrope/vasopressor
  - Mechanical circulatory support (e.g. IABP)
- Acute coronary syndrome
  - Coronary reperfusion
  - Antithrombotic therapy
- Acute mechanical cause/severe valvular disease
  - Echocardiography
  - Surgical/percutaneous intervention
### Treatment of acute heart failure: atrial fibrillation or bradycardia

#### Patients with AF and a rapid ventricular rate

Patients should be fully anticoagulated (e.g. with i.v. heparin), if not already anticoagulated and with no contraindication to anticoagulation, as soon as AF is detected to reduce the risk of systemic arterial embolism and stroke.

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Electrical cardioversion is recommended in patients haemodynamically compromised by AF and in whom urgent restoration of sinus rhythm is required to improve the patient’s clinical condition rapidly.

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Electrical cardioversion or pharmacological cardioversion with amiodarone should be considered in patients when a decision is made to restore sinus rhythm non-urgently (‘rhythm control’ strategy). This strategy should only be employed in patients with a first episode of AF of <48 h duration (or in patients with no evidence of left atrial appendage thrombus on TOE).

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**Intravenous administration of a cardiac glycoside should be considered for rapid control of the ventricular rate.**

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Dronedarone is not recommended because of safety concerns (increased risk of hospital admission for cardiovascular causes and an increased risk of premature death), particularly in patients with an EF ≤40%.

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<td>III</td>
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Class I antiarrhythmic agents are not recommended because of safety concerns (increased risk of premature death), particularly in patients with LV systolic dysfunction.

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<td>III</td>
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#### Patients with severe bradycardia or heart block

Pacing is recommended in patients haemodynamically compromised by severe bradycardia or heart block to improve the patient’s clinical condition.

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</table>
Should we SHIFT our thinking about digoxin? Observations on ivabradine and heart rate reduction in heart failure

Davide Castagno, Mark C. Petrie, Brian Claggett, and John McMurray

1Division of Cardiology, Department of Internal Medicine, University of Turin, Turin, Italy; 2Advanced Heart Failure Service, Golden Jubilee National Hospital, Clydebank, Glasgow, UK; 3Department of Biostatistics, Harvard School of Public Health, Boston, MA, USA; and 4BHF Cardiovascular Research Centre, University of Glasgow, Glasgow G12 8QQ, UK

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Effect of digoxin in high risk HF in the DIG trial

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Evaluation of pts. with suspected AHF

- History/examination (including blood pressure and respiratory rate)
- Chest X-ray
- Echocardiogram or NP (or both)
- Blood chemistry
- ECG
- Oxygen saturation
- Full blood count

Simultaneously assess for:
- Ventilation/systemic oxygenation inadequate
- Life-threatening arrhythmia/bradycardia
- Blood pressure <85 mmHg or shock
- Acute coronary syndrome
- Acute mechanical cause/severe valvular disease

Urgent action if present:
- Oxygen
- NIV
- ETT and invasive ventilation
- Electrical cardioversion
- Pacing
- Inotrope/vasopressor
- Mechanical circulatory support (e.g. IABP)
- Coronary reperfusion
- Antithrombotic therapy
- Echocardiography
- Surgical/percutaneous intervention

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Acute Cardiac Care
ESC Working Group
European Society of Cardiology
### Treatment of acute heart failure: hypotension, hypoperfusion or shock

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Level</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrical cardioversion is recommended if an atrial or ventricular arrhythmia is thought to be contributing to the patient's haemodynamic compromise in order to restore sinus rhythm and improve the patient's clinical condition.</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>An i.v. infusion of an inotrope (e.g. dobutamine) should be considered in patients with hypotension (systolic blood pressure &lt;85 mmHg) and/or hypoperfusion to increase cardiac output, increase blood pressure, and improve peripheral perfusion. The ECG should be monitored continuously because inotropic agents can cause arrhythmias and myocardial ischaemia.</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>Short-term mechanical circulatory support should be considered (as a 'bridge to recovery') in patients remaining severely hypoperfused despite inotropic therapy and with a potentially reversible cause (e.g. viral myocarditis) or a potentially surgically correctable cause (e.g. acute interventricular septal rupture).</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>An i.v. infusion of levosimendan (or a phosphodiesterase inhibitor) may be considered to reverse the effect of beta-blockade if beta-blockade is thought to be contributing to hypoperfusion. The ECG should be monitored continuously because inotropic agents can cause arrhythmias and myocardial ischaemia, and, as these agents are also vasodilators, blood pressure should be monitored carefully.</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>A vasopressor (e.g. dopamine or norepinephrine) may be considered in patients who have cardiogenic shock, despite treatment with an inotrope, to increase blood pressure and vital organ perfusion. The ECG should be monitored as these agents can cause arrhythmias and/or myocardial ischaemia. Intra-arterial blood pressure measurement should be considered.</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>Short-term mechanical circulatory support may be considered (as a 'bridge to decision') in patients deteriorating rapidly before a full diagnostic and clinical evaluation can be made.</td>
<td>IIb</td>
<td>C</td>
</tr>
</tbody>
</table>
Intra-aortic balloon pump

Conventional indications:

- before surgical correction of specific acute mechanical problems (VSR, acute MR)
- severe acute myocarditis
- in selected patients with acute myocardial ischaemia or infarction before, during, and after PCI or CABG.
IABP therapy in STEMI complicated by cardiogenic shock.

IABP therapy adjunctive to thrombolysis: benefit?  
IABP therapy adjunctive to PPCI: no benefit

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Mechanical circulatory support (MCS)

- Temporary MCS may be used in selected patients with AHF, including intra-aortic balloon counterpulsation, other percutaneous cardiac support, and ECMO.
- MCS, particularly ECMO, can be used as a ‘bridge to decision (BTD)’ in patients with acute and rapidly deteriorating HF where full evaluation has not been possible and in whom death will occur without MCS.
- However, the difficult decision to withdraw MCS may need to be made if the patient is not eligible for conventional corrective surgery or longer term MCS.

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Treating Acute HF: The Belgian Way

Diuretics  Inotropes  Vasoconstrictors  IABP

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Treating Acute HF: The Dutch Way

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Percutaneous ventricular assist devices (PVADs)
## IABP vs. currently available pVAD

<table>
<thead>
<tr>
<th></th>
<th>IABP</th>
<th>TandemHeart pVAD</th>
<th>Impella 2.5 Recover system</th>
<th>ECMO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pump mechanism</strong></td>
<td>Pneumatic</td>
<td>Centrifugal</td>
<td>Axial flow</td>
<td>Centrifugal flow</td>
</tr>
<tr>
<td><strong>Insertion</strong></td>
<td>Retrograde 7-9F balloon catheter into descending aorta via femoral artery</td>
<td>21F inflow cannula into left atrium via femoral vein and transseptal puncture and 15/17F outflow cannula into femoral artery</td>
<td>12F catheter (13F sheath) placed retrograde across the aortic valve via femoral artery</td>
<td>18–31F inflow cannula into the right atrium via femoral vein and 15-22F outflow cannula into descending aorta via femoral artery</td>
</tr>
<tr>
<td><strong>Difficulty of insertion</strong></td>
<td>+</td>
<td>+++</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td><strong>Degree of support</strong></td>
<td>+</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
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<tr>
<td></td>
<td>(↑ CO by 0.5 l/min)</td>
<td>(↑ CO by 3.5–4 l/min)</td>
<td>(↑ CO by 2.5 l/min)</td>
<td>(↑ CO to &gt;4.5 l/min)</td>
</tr>
<tr>
<td><strong>Cardiac power output</strong></td>
<td>+</td>
<td>+ +</td>
<td>+ +</td>
<td>+ +</td>
</tr>
<tr>
<td><strong>Time for implantation</strong></td>
<td>10 min</td>
<td>25–65 min</td>
<td>11–25 min</td>
<td>10–15 min</td>
</tr>
<tr>
<td><strong>Limb ischemia</strong></td>
<td>+</td>
<td>+++</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td><strong>Hemolysis</strong></td>
<td>0</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
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<tr>
<td><strong>Bleeding risks</strong></td>
<td>+</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td><strong>Contraindications</strong></td>
<td>Moderate–severe AI/aortic stenosis, coagulopathy, severe sepsis</td>
<td>Peripheral arterial disease (may be placed with antegrade sideport for limb perfusion), RV failure</td>
<td>LV thrombus, ventricular septal defect, severe aortic stenosis, RV failure, peripheral arterial disease</td>
<td>Contraindication to anticoagulation, irreversible brain injury, terminal disease</td>
</tr>
</tbody>
</table>

<sup>a</sup> www.escardio.org/acutecc

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Paradigm shift in treatment cardiogenic shock

Mortality risk vs. inotrope dosing

- Mild shock
- Profound shock
  - IABP/IMPELLA 2.5
- Severe refractory shock
  - TandemHeart/ECMO

<table>
<thead>
<tr>
<th>inotrope dosing</th>
<th>Mild shock</th>
<th>Profound shock</th>
<th>Severe refractory shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>no inotrope</td>
<td>2</td>
<td>2</td>
<td>42</td>
</tr>
<tr>
<td>low dose</td>
<td>3</td>
<td>7.5</td>
<td>42</td>
</tr>
<tr>
<td>moderate dose</td>
<td>7.5</td>
<td>21</td>
<td>42</td>
</tr>
<tr>
<td>one high-dose</td>
<td>21</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>two high-dose</td>
<td>42</td>
<td>42</td>
<td>42</td>
</tr>
<tr>
<td>three high-dose</td>
<td>80</td>
<td>80</td>
<td>80</td>
</tr>
</tbody>
</table>

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Saving lives is our mission

B Kar et al.
Circulation. 2012;125:1809-1817
ISAR SHOCK: Impella 2.5 vs. IABP in STEMI with shock

A

Cardiac Power Index (W/m²)

Time after Implantation (hours)

B

Serum-Lactate (mmol/L)

Time after Implantation (hours)

C

Survival Probability

Days After Randomization

25 patients

M Seyfarth et al.
J Am Coll Cardiol 2008;52:1584–8

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• 34 STEMI patients
• Improved survival in patients who received immediate Impella 5.0 treatment, as well as in patients who were upgraded from 2.5 to 5.0 support,
• Low survival in patients who received only Impella 2.5 support.
pVAD new developments

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Saving lives is our mission
117 patients with SRCS implanted with TandemHeart pVAD

Table 4
Safety and Efficacy of Use of Tandem Heart Percutaneous Ventricular Assist Device: Complication Rate in Our Center

<table>
<thead>
<tr>
<th>Adverse Event</th>
<th>Frequency</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groin hematoma</td>
<td>6/117</td>
<td>5.12</td>
</tr>
<tr>
<td>Limb ischemia</td>
<td>4/117</td>
<td>3.41</td>
</tr>
<tr>
<td>Bleeding around cannula site</td>
<td>34/117</td>
<td>29.05</td>
</tr>
<tr>
<td>Femoral artery dissection</td>
<td>1/117</td>
<td>0.85</td>
</tr>
<tr>
<td>Atrial perforation</td>
<td>1/117</td>
<td>0.85</td>
</tr>
<tr>
<td>Sepsis</td>
<td>35/117</td>
<td>29.90</td>
</tr>
<tr>
<td>Coagulopathy</td>
<td>13/117</td>
<td>11.00</td>
</tr>
<tr>
<td>Stroke</td>
<td>8/117</td>
<td>6.83</td>
</tr>
<tr>
<td>Blood transfusions</td>
<td>70/117</td>
<td>59.80</td>
</tr>
<tr>
<td>Gastrointestinal bleed</td>
<td>23/117</td>
<td>19.65</td>
</tr>
</tbody>
</table>

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Saving lives is our mission
pVAD in Severe Cardiogenic Shock

117 patients with SRCS implanted with TandemHeart pVAD

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LVAD as destination therapy

Figure 1. Survival Rates in Two Trials of Left Ventricular Assist Devices (LVADs) as Destination Therapy.

The curves labeled 2009 are those reported by Slaughter and colleagues in this issue of the Journal; those labeled 2001 were reported for the REMATCH trial.
LVAD new developments

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LVAD new developments

A Pulsatile-Flow LVAD

B Continuous-Flow LVAD

Fully magnetic levitated rotor

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LVAD new developments

Partial support for patients in class IIIb?

- Inflow cannula transseptally positioned in the left atrium by the interventional cardiologist
- Outflow graft sutured to the subclavian artery by the surgeon

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Conclusions

- Always look for precipitating causes
  - ACS: early revascularization
  - Atrial fib.: rapid rate control with digitalis
- Use LMNOP with IV bolus of loop diuretics
- Noninvasive ventilation: helpful as adjunctive therapy
- Consider temporary use of mechanical support systems in patients unresponsive to inotropics and potential reversible cause
  - Bridge to recovery
  - Bridge to destination therapy

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Thank You!