The implementation of the guidelines for heart failure in clinical practice
DECONGESTION IN HEART FAILURE WITH LOW BLOOD PRESSURE

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DECLARATION OF INTEREST

No conflict of interest related to this presentation
Clinical profiles of patients with AHF

- **CONGESTION (-)**
  - Pulmonary congestion
  - Orthopnoea/paroxysmal nocturnal dyspnoea
  - Peripheral (bilateral) oedema
  - Jugular venous dilatation
  - Congested hepatomegaly
  - Gut congestion, ascites
  - Hepatojugular reflux

- **HYPOPERFUSION (-)**
  - Cold sweated extremities
  - Oliguria
  - Mental confusion
  - Dizziness
  - Narrow pulse pressure

- **CONGESTION (+)**

- **HYPOPERFUSION (+)**

- **WARM-DRY**

- **WARM-WET**

- **COLD-DRY**

- **COLD-WET**

Hypoperfusion is **not synonymous** with hypotension, but often hypoperfusion is accompanied by hypotension.
Initial management in AHF

Patient with suspected AHF

Urgent phase after first medical contact

1. Cardiogenic shock?
   - Yes
   - Circulatory support
     - pharmacological
     - mechanical
   - No

2. Respiratory failure?
   - Yes
   - Ventilatory support
     - oxygen
     - non-invasive positive pressure ventilation (CPAP, BiPAP)
     - mechanical ventilation
   - No
   - Immediate stabilization and transfer to ICU/CCU

Immediate phase (Initial 60–120 minutes)

Identification of acute aetiology:
- C coronary syndrome
- H Hypertension emergency
- A Arrhythmia
- M acute mechanical cause
- P Pulmonary embolism

- Yes
  - Immediate initiation of specific treatment
  - Follow detailed recommendations in the specific ESC Guidelines

- No
  - Diagnostic work-up to confirm AHF
  - Clinical evaluation to select optimal management
Management of AHF based on clinical profile
CASE 1
Initial Presentation

• Forty-four years old female
• Smoker
• Family history of Coronary Artery Disease (CAD)
• Dyslipidemia, probably familial hypercholesterolemia
CASE 1

Initial Presentation. Examination in ER

- Chest pain started 2 hours before first medical contact during the night, nausea and syncope
- HR: 66bpm     BP: 92/60mmHg     T:37°C
- Sweating, cold hands and feet, pale skin (COLD)
- Heart: S3, 2/6 apical systolic murmur
- Lungs: Rales in lower 1/3 of both lungs (WET)
CASE 1

Initial Presentation. ECG
CASE 1

Initial Presentation. Chest RX
CASE 1
Initial Presentation. Echo

- LVEF=25-30% akinesia of apical segments, hypokinesia of basal and mid anteroseptal segments, hypokinesia of basal and mid anterior segments. LVEDD=44mm, IVS=10mm.
- E=0.59, A=0.70, DT=186msec
- MR=1+/4+
- TR=1+/4+, PASP=45mmHg
- IVC=18mm <50% variation.
CASE 1

Initial Presentation. Echo
CASE 1

Initial Presentation. Blood tests

- ABG: pH: 7.24, pO\(_2\): 63, SaO\(_2\): 88%, pCO\(_2\): 39, HCO\(_3\): 16, Lac: 2.1

- Hgb: 14.9 g/dL, WBC: 25.070, PLT: 293.000

- Creatinine: 0.6 Na: 141 K: 4.0

- Troponin (high sensitive troponin T): 27 - 9528

- CRP: 0.19

- TCHOL: 249, TGL: 74, HDL: 42, LDL: 192
CASE 1
Initial Presentation. Coronary angiography
CASE 1

Initial Presentation. Treatment in ER

• She was given ASA 500mg, ticagrelor 180mg as loading doses and 5,000 IU heparin along with omeprazole 20mg iv.

• Coronary angiogram: LAD proximal total thrombotic occlusion. LCx: Non-significant stenosis. RCA: non-significant stenoses in mid-RCA and mid-PDA.

• She underwent a successful primary PCI with predilation (Boston non-compliant 2x15mm) and DES implantation (Synergy 3X16mm). TIMI III flow was restored post-procedure. There is an intermediate stenosis in mid LAD after D1.
CASE 1

After admission. CCU

• 25/1/2018. The patient was hemodynamically unstable and was given iv inotropes (dobutamine – 2.5μg/kg/min) + vasopressors (noradrenaline – 0.8μg/kg/min).

• She was also given furosemide 20mg BID iv and tirofiban iv for 24h post-procedure.

• She was started with atorvastatin 40mg/ezetimibe 10m QOD.

• HR: 124bpm, BP: 102/73mmHg, SaO2: 96%, Urine: 4100ml (-3050ml)
CASE 1

After admission. CCU

• 26/1/2018. The patient experienced improvement of symptoms and adequate diuresis was restored. Reduction of the doses of inotropes and vasopressors.

She was started eplerenone 25mg QOD and ivabradine 5 ½ BID

HR: 120bpm, BP: 104/68mmHg, SaO2: 96%, Urine output: 4900ml (-2050ml)

• 2/2/2018. The patient was successfully weaned from noradrenaline and inotropes
CASE 1

After admission. CCU

- **3/2/2018.** Furosemide was stopped.
- **5/2/2018.** Acenocoumarol was initiated. Enoxaparin was increased to 60 1x2.
- Echo 5/2/2018: LVEF=25-30% akinesia of apical segments, hypokinesia of basal and mid anteroseptal, basal and mid anterior segments. Apical **thrombus** and spontaneous echo contrast inside LV. LVEDD=44mm, IVS=10mm, MR=1+/4+. TR=1+/4+, PASP=35mmHg, IVC=22mm <50% variation.
CASE 1

After admission. Medical ward

• **Enalapril** 2.5mg QOD and **bisoprolol** 2.5mg were initiated whereas ivabradine was stopped.
• Holter ECG: SR, no pauses, no episodes of VT or NSVT, a few VEB.
• Echo 12/2/2018: LVEF=30% akinesia of apical segments, hypokinesia of basal and mid anteroseptal, basal and mid anterior segments. No apical thrombus was found, however contrast inside LV still exists. LVEDD:52mm, E/e’:10, MR=2-3+/4+. TR=1+/4+, PASP=60mmHg. No pericardial effusion.

• She was discharged on

ASA 100mg QOD, clopidogrel 75mg QOD, acenocoumarol dose according to INR

**Enalapril** 5mg 1/2x1, **Bisoprolol** 5mg 1/2x1, **Eplerenone** 25mg QOD

**Atorvastatin** 40mg QOD, **ezetimibe** 10mg

**Esomeprazole** 40mg QOD
CASE 1

After discharge

• SPECT: Permanent perfusion defects in apical segments, in basal and mid anterior segments, basal and mid anteroseptal segments and basal and mid anterolateral segments.

• She experienced presyncope. Ventricular tachycardia induced during EPS (4/6/2018).

• An ICD was successfully implanted (20/6/2018)
CASE 1
At present

- NYHA II. BP: 80/60mmHg, HR: 54bpm, ECG: SR.
- Echo 20/9/2018: LVEF=35%, LVEDD:48mm, LAV: 39ml/m2, E/e’: 10, MR: 2+/4+ (EROA=0.18cm2), PASP<35mmHg, TR 1+/4+.
- Enalapril 2.5mg BID, bisoprolol 2.5 QOD, furosemide 40mg ½ QOD, eplerenone 25mg QOD, atorvastatin 40mg QOD, ezetimibe 10mg QOD, ASA 100mg QOD, clopidogrel 75mg QOD, esomeprazole 40mg QOD.
CASE 2

Initial Presentation

• Male 75 years old
• Dyspnea and fatigue on mild exertion in the last 2 weeks.
• Progressive deterioration of dyspnea and severe orthopnea.
• HFREF with EF=25%, NYHA III
• CAD, previous MI - PCI in LCX, LAD: 60% stenosis proximally (Last coronary angiography -2010)
• ICD (2010)
CASE 2
Initial Presentation

• PAF
• Hypertension, dyslipidemia
• Current medications without ACEI, MRA, OAC, antiplatelets (allergic to acetylsalicylic acid?)

Carvedilol 3.125 BID
Furosemide 40mg ½ QOD
Atorvastatin 10mg QOD
Amiodarone ½ QOD
Q10
CASE 2

Initial Presentation. Examination in ER

- Dyspnea at rest, tachypnea (26/min)
- HR: 75  BP: 85/50mmHg  T: 36°C
- Sweating cold extremities, pale skin (COLD)
- Marked jugular venous distension
- Heart: S3, 3/6 apical systolic murmur
- Lungs: Rales in lower half of both lungs (WET)
- Legs: bilateral pitting edema
CASE 2

Initial Presentation. ECG
CASE 2

Initial Presentation. Chest RX
CASE 2

Initial Presentation. Echo

• LVEF=15%, akinesia of apical segments, basal and mid anteroseptal segments, basal and mid anterior segments, basal and mid inferolateral segments and hypokinesia of the other segments.

• LVEDD: 68mm. Dilated LA.

• DT=150msec, E/e’=25, MR=1+/4+.

• RVD1: 34, S’RV:6, TR=1+/4+, PASP=45mmHg.

• IVC=14mm <50% variation.
CASE 2

Initial Presentation. Echo
CASE 2

Initial Presentation. Blood tests

- **Hgb:** 14.1 g/dL, **WBC:** 21.910, **PLT:** 154.000
- **Creatinine:** 1.4, **Urea:** 56, **Na:** 144, **K:** 4.0
- **Troponin (high sensitive troponin T):** 19-52
- **CRP:** 0.8
- **TCHOL:** 101, **TGL:** 69, **HDL:** 29, **LDL:** 55
CASE 2

Initial Presentation. Treatment in ER

- Furosemide iv (total dose 200mg, intermittent boluses)
- Non-invasive positive pressure ventilation (BiPAP)

No improvement (ABG: pH:7.07, pO2: 64, SaO2:80%, pCO2: 60, HCO3: 13.5, Lac: 3.3)

- Intubation- mechanical ventilation
CASE 2

Initial Presentation. Treatment in ER

- noradrenaline iv (3μg/kg/min)
- dopamine iv (3μg/kg/min)
- furosemide iv (20mg/h continuous infusion)
- Midazolam iv.
CASE 2

After admission. CCU

• 1/10/2017
  HR: 124bpm, BP: 102/73mmHg, SaO2: 99%, Urine: 1500ml (-500ml)
  Noradrenaline i.v. (3μg/kg/min) dopamine i.v. (3μg/kg/min) furosemide 20mg/h iv and midazolam.
  Tazocin 4.5gr TID and esomeprazole iv were started.

• 2/10/2017
  HR: 132bpm, BP: 112/60mmHg, SaO2: 96%, Urine: 4800ml (-1350ml)
  Spironolactone 25mg QOD and tinzaparin 0.45 QOD were initiated.

• 3/10/2017
  HR: 88bpm, BP: 110/60mmHg, SaO2: 96% Urine: 4200ml (-2160ml)
  Amiodarone iv was started due to episode of AF. Midazolam was stopped, infusion of furosemide reduced to 10mg/h and infusion of noradrenaline iv was reduced as well.
CASE 2

After admission. CCU

• 6/10/2017
  HR: 60bpm, BP: 116/50mmHg, SaO2: 96%, Urine output: 3300ml (-2000ml)

• 8/10/2017 Proteus mirabilis isolated in sputum culture. CRPmax: 13.9

• 10/10/2017 Weaning from mechanical ventilation – Extubation. Weaning from noradrenaline and inotropes.
CASE 2

After admission. Medical ward

Furosemide 40 BID
Spironolactone 25 QOD
Enoxaparin 60 SC 1X1
Meropenem 1gx2
Esomeprazole iv BID
Bisoprolol 1.25 QOD
CASE 2

After discharge

Careful onset of ACEI
SPECT for detection of ischemia and viability
Up-grade of ICD to CRT-D?
Iron deficiency?
Depression
Cachexia
Recommendations for the management of patients with AHF: oxygen therapy and ventilatory support

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monitoring of transcutaneous arterial oxygen saturation ($\text{SpO}_2$) is recommended.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Measurement of blood pH and carbon dioxide tension (possibly including lactate) should be considered, especially in patients with acute pulmonary oedema or previous history of COPD using venous blood. In patients with cardiogenic shock arterial blood is preferable.</td>
<td>IIa</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Oxygen therapy is recommended in patients with AHF and $\text{SpO}_2 &lt; 90%$ or $\text{PaO}_2 &lt; 60$ mmHg (8.0 kPa) to correct hypoxaemia.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Non-invasive positive pressure ventilation (CPAP, BiPAP) should be considered in patients with respiratory distress (respiratory rate &gt; 25 breaths/min, $\text{SpO}_2 &lt; 90%$) and started as soon as possible in order to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation. Non-invasive positive pressure ventilation can reduce blood pressure and should be used with caution in hypotensive patients. Blood pressure should be monitored regularly when this treatment is used.</td>
<td>IIa</td>
<td>B</td>
<td>541–545</td>
</tr>
<tr>
<td>Intubation is recommended, if respiratory failure, leading to hypoxaemia ($\text{PaO}_2 &lt; 60$ mmHg (8.0 kPa)), hypercapnia ($\text{PaCO}_2 &gt; 50$ mmHg (6.65 kPa)) and acidosis (pH &lt; 7.35), cannot be managed non-invasively.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Intra-arterial line should be considered in patients with hypotension and persistent symptoms despite treatment.</td>
<td>IIa</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Pulmonary artery catheter may be considered in patients who, despite pharmacological treatment present refractory symptoms (particularly with hypotension and hypoperfusion).</td>
<td>IIb</td>
<td>C</td>
<td></td>
</tr>
</tbody>
</table>
## Recommendations for the management of patients with AHF – ESC 2016

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diuretics</strong></td>
<td></td>
</tr>
<tr>
<td>Intravenous loop diuretics are recommended for all patients with AHF admitted with signs/symptoms of fluid overload to improve symptoms. It is recommended to regularly monitor symptoms, urine output, renal function and electrolytes during use of i.v. diuretics.</td>
<td>I</td>
</tr>
<tr>
<td>In patients with new-onset AHF or those with chronic, decompensated HF not receiving oral diuretics the initial recommended dose should be 20–40 mg i.v. furosemide (or equivalent); for those on chronic diuretic therapy, initial i.v. dose should be at least equivalent to oral dose.</td>
<td>I</td>
</tr>
<tr>
<td>It is recommended to give diuretics either as intermittent boluses or as a continuous infusion, and the dose and duration should be adjusted according to patients’ symptoms and clinical status.</td>
<td>I</td>
</tr>
<tr>
<td>Combination of loop diuretic with either thiazide-type diuretic or spironolactone may be considered in patients with resistant oedema or insufficient symptomatic response.</td>
<td>IIb</td>
</tr>
<tr>
<td><strong>Vasodilators</strong></td>
<td></td>
</tr>
<tr>
<td>i.v. vasodilators should be considered for symptomatic relief in AHF with SBP &gt; 90 mmHg (and without symptomatic hypotension). Symptoms and blood pressure should be monitored frequently during administration of i.v. vasodilators.</td>
<td>IIa</td>
</tr>
<tr>
<td>In patients with hypertensive AHF, i.v. vasodilators should be considered as initial therapy to improve symptoms and reduce congestion.</td>
<td>IIa</td>
</tr>
</tbody>
</table>
### Recommendations for the management of patients with AHF – ESC 2016

**Inotropic agents – dobutamine, dopamine, levosimendan, phosphodiesterase III (PDE III) inhibitors**

- **Short-term, i.v. infusion of inotropic agents** may be considered in patients with hypotension (SBP <90 mmHg) and/or signs/symptoms of hypoperfusion despite adequate filling status, to increase cardiac output, increase blood pressure, improve peripheral perfusion and maintain end-organ function.
- **An intravenous infusion of levosimendan or a PDE III inhibitor** may be considered to reverse the effect of beta-blockade if beta-blockade is thought to be contributing to hypotension with subsequent hypoperfusion.
- Inotropic agents are not recommended unless the patient is symptomatically hypotensive or hypoperfused because of safety concern.

**Vasopressors**

- A **vasopressor (norepinephrine preferably)** may be considered in patients who have cardiogenic shock, despite treatment with another inotrope, to increase blood pressure and vital organ perfusion.
- It is recommended to monitor ECG and blood pressure when using inotropic agents and vasopressors, as they can cause arrhythmia, myocardial ischaemia, and in the case of levosimendan and PDE III inhibitors also hypotension.
- In such cases intra-arterial blood pressure measurement may be considered.
Recommendations for the management of patients with cardiogenic shock – ESC 2016

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>In all patients with suspected cardiogenic shock, immediate ECG and echocardiography are recommended.</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>All patients with cardiogenic shock should be rapidly transferred to a tertiary care center which has a 24/7 service of cardiac catheterization, and a dedicated ICU/CCU with availability of short-term mechanical circulatory support.</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>In patients with cardiogenic shock complicating ACS an immediate coronary angiography is recommended (within 2 hours from hospital admission) with an intent to perform coronary revascularization.</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Continuous ECG and blood pressure monitoring are recommended.</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Invasive monitoring with <strong>arterial line</strong> is recommended.</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Fluid challenge (saline or Ringer’s lactate, &gt;200 ml/15–30 min) is recommended as the first-line treatment if there is no sign of overt fluid overload.</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Intravenous <strong>inotropic agents (dobutamine)</strong> may be considered to increase cardiac output.</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td><strong>Vaspressors (norepinephrine preferable over dopamine)</strong> may be considered if there is a need to maintain SBP in the presence of persistent hypoperfusion.</td>
<td>IIb</td>
<td>B</td>
</tr>
<tr>
<td>IABP is not routinely recommended in cardiogenic shock.</td>
<td>III</td>
<td>B</td>
</tr>
<tr>
<td><strong>Short-term mechanical circulatory support</strong> may be considered in refractory cardiogenic shock depending on patient age, comorbidities and neurological function.</td>
<td>IIb</td>
<td>C</td>
</tr>
</tbody>
</table>
# Cardshock risk score

## Prediction of in-hospital mortality in Cardiogenic shock

<table>
<thead>
<tr>
<th>Factor</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 75yrs</td>
<td>1</td>
</tr>
<tr>
<td>Confusion</td>
<td>1</td>
</tr>
<tr>
<td>Previous MI or CABG</td>
<td>1</td>
</tr>
<tr>
<td>ACS etiology</td>
<td>1</td>
</tr>
<tr>
<td>LVEF &lt; 40%</td>
<td>1</td>
</tr>
<tr>
<td>Lactate 2-4</td>
<td>1</td>
</tr>
<tr>
<td>Lactate &gt; 4</td>
<td>2</td>
</tr>
<tr>
<td>eGFR: 30-60</td>
<td>1</td>
</tr>
<tr>
<td>eGFR &lt; 30</td>
<td>2</td>
</tr>
<tr>
<td><strong>Maximum points</strong></td>
<td><strong>9</strong></td>
</tr>
</tbody>
</table>
Mechanism of Action and Hemodynamic Effects of Common Vasoactive Medications in CS

<table>
<thead>
<tr>
<th>Medication</th>
<th>Usual Infusion Dose</th>
<th>Receptor Binding</th>
<th>Hemodynamic Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>α₁</td>
<td>β₁</td>
</tr>
<tr>
<td>Vasopressor/inotropes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dopamine</td>
<td>0.5–2 μg·kg⁻¹·min⁻¹</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>5–10 μg·kg⁻¹·min⁻¹</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td></td>
<td>10–20 μg·kg⁻¹·min⁻¹</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>0.05–0.4 μg·kg⁻¹·min⁻¹</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>0.01–0.5 μg·kg⁻¹·min⁻¹</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>0.1–10 μg·kg⁻¹·min⁻¹</td>
<td>+++</td>
<td>−</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>0.02–0.04 U/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stimulates V₁ receptors in vascular smooth muscle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inodilators</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>2.5–20 μg·kg⁻¹·min⁻¹</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>2.0–20 μg/min</td>
<td>−</td>
<td>+++</td>
</tr>
<tr>
<td>Milrinone</td>
<td>0.125–0.75 μg·kg⁻¹·min⁻¹</td>
<td>PD-3 inhibitor</td>
<td>↑CO, ↓SVR, ↓PVR</td>
</tr>
<tr>
<td>Enoximone</td>
<td>2–10 μg·kg⁻¹·min⁻¹</td>
<td>PD-3 inhibitor</td>
<td>↑CO, ↓SVR, ↓PVR</td>
</tr>
<tr>
<td>Levosimendan</td>
<td>0.05–0.2 μg·kg⁻¹·min⁻¹</td>
<td>Myofilament Ca²⁺ sensitizer, PD-3 inhibitor</td>
<td>↑CO, ↓SVR, ↓PVR</td>
</tr>
</tbody>
</table>

Comparison of Dopamine and Norepinephrine in the Treatment of Shock

Figure 2. Kaplan–Meier Curves for 28-Day Survival in the Intention-to-Treat Population.

Current real-life use of vasopressors and inotropes in cardiogenic shock mortality

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**Critical Care (2016) 20:208**
Epinephrine and short-term survival in cardiogenic shock meta-analysis of 2583 patients

<table>
<thead>
<tr>
<th>Study</th>
<th>No. of patients</th>
<th>No. of patients receiving epinephrine</th>
<th>OR for short-term mortality [95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adler, 2012</td>
<td>40</td>
<td>10</td>
<td>4.00 [0.87 - 18.45]</td>
</tr>
<tr>
<td>Adler, unpublished</td>
<td>47</td>
<td>0</td>
<td>4.27 [0.60 - 20.67]</td>
</tr>
<tr>
<td>AHEAD, 2011</td>
<td>674</td>
<td>304</td>
<td>5.10 [9.08 - 25.05]</td>
</tr>
<tr>
<td>ALARM, 2011</td>
<td>520</td>
<td>86</td>
<td>2.14 [1.94 - 3.42]</td>
</tr>
<tr>
<td>Chua, 2011</td>
<td>105</td>
<td>80</td>
<td>0.99 [0.40 - 2.45]</td>
</tr>
<tr>
<td>CARDSHOCK, 2018</td>
<td>210</td>
<td>46</td>
<td>6.84 [3.22 - 13.71]</td>
</tr>
<tr>
<td>Champion, 2014</td>
<td>192</td>
<td>130</td>
<td>7.27 [2.85 - 18.54]</td>
</tr>
<tr>
<td>EFICA, 2008</td>
<td>158</td>
<td>75</td>
<td>3.10 [1.61 - 5.98]</td>
</tr>
<tr>
<td>Gautard, 2015</td>
<td>40</td>
<td>11</td>
<td>3.15 [0.75 - 13.29]</td>
</tr>
<tr>
<td>IMPRESS in Severe Shock, 2017</td>
<td>48</td>
<td>14</td>
<td>12.55 [2.38 - 66.01]</td>
</tr>
<tr>
<td>CPT/IMA CC, 2018</td>
<td>57</td>
<td>27</td>
<td>2.55 [0.84 - 7.72]</td>
</tr>
<tr>
<td>Basir, unpublished</td>
<td>45</td>
<td>8</td>
<td>0.96 [0.16 - 5.73]</td>
</tr>
<tr>
<td>Popovic, 2011</td>
<td>86</td>
<td>47</td>
<td>1.11 [0.47 - 2.63]</td>
</tr>
<tr>
<td>Simonis, 2012</td>
<td>39</td>
<td>25</td>
<td>1.37 [0.53 - 3.55]</td>
</tr>
<tr>
<td>SMASHI, 1990</td>
<td>111</td>
<td>41</td>
<td>0.02 [0.20 - 1.47]</td>
</tr>
<tr>
<td>Valente, 2011</td>
<td>152</td>
<td>34</td>
<td>2.40 [0.38 - 14.96]</td>
</tr>
</tbody>
</table>

All studies: 2583 patients, 947 events. OR: 3.33 [2.81 - 3.94]

Fig. 3 Forest plot of the meta-analysis of short-term mortality

Epinephrine Versus Norepinephrine for Cardiogenic Shock After AMI

J AmColl Cardiol 2018;72:173–82
ROSE AHF

Key findings from ROSE AHF

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>Low-dose dopamine</th>
<th>Low-dose nesiritide</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>72-hour cumulative urine output</td>
<td>8.5 L</td>
<td>8.6 L</td>
<td>8.3 L</td>
</tr>
<tr>
<td>72-hour change in cystatin C level</td>
<td>0.12 mg/L</td>
<td>0.07 mg/L</td>
<td>0.11 mg/L</td>
</tr>
<tr>
<td>Persistent or worsening heart failure within 72 hours</td>
<td>9%</td>
<td>5%</td>
<td>4%</td>
</tr>
<tr>
<td>60-day mortality</td>
<td>9%</td>
<td>7%</td>
<td>10%</td>
</tr>
<tr>
<td>180-day mortality</td>
<td>19.7%</td>
<td>19.1%</td>
<td>21.1%</td>
</tr>
</tbody>
</table>

Note: The study involved 360 patients hospitalized with acute heart failure and renal dysfunction. There were no significant between-group differences in any endpoints.

Source: Dr. Chen

Frontline Medical News
How to use inotropes

• Skin, mental status, blood gases, lactate, urine output

Perfusion targets
• Lactate<2
• Alertness, skin, diuresis
• SvO₂>60%

Haemodynamic targets
CI>2.2
MAP (55)-65-75, Fluid challenge, Noradrenaline if needed
Indications and practical approach to non-invasive ventilation in AHF

<table>
<thead>
<tr>
<th>Main characteristics</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>Main indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPAP</td>
<td>Continuous positive intra-thoracic pressure</td>
<td>Very simple use&lt;br&gt;Does not require a ventilator&lt;br&gt;Improves oxygenation</td>
<td>Does not provide ventilatory help on inspiration</td>
</tr>
<tr>
<td>HFNC</td>
<td>High humidified flow (up to 60–80 L/min) through nasal cannula, producing: • Low level of PEEP&lt;br&gt;• Decreased upper airway resistance&lt;br&gt;• Tracheal air washout</td>
<td>Simple use&lt;br&gt;Does not require a ventilator&lt;br&gt;Good adaptation&lt;br&gt;Improves oxygenation</td>
<td>Does not provide ventilatory help on inspiration</td>
</tr>
<tr>
<td>NIPSV</td>
<td>Inspiration: Decelerated flow to maintain a target pressure (pressure support) triggered by patient’s effort. Expiration: PEEP</td>
<td>Provides ventilatory support&lt;br&gt;Results as a continuous positive pressure plus a help on inspiration</td>
<td>Needs expertise and appropriate device.&lt;br&gt;May produce overassistance when patients increase inspiratory effort</td>
</tr>
<tr>
<td>PAV</td>
<td>Adjusts ventilator assistance to the activity of respiratory muscles estimated by an algorithm, proportionally to the patient’s effort</td>
<td>Provides ventilatory support&lt;br&gt;Better adaptation than NIPSV&lt;br&gt;May prevent overassistance</td>
<td>Mismatching in complex respiratory pattern</td>
</tr>
</tbody>
</table>

Use of noninvasive and invasive mechanical ventilation in cardiogenic shock

IABP-SHOCK II trial

Lancet 2013; 382: 1638–45
Percutaneous short-term active mechanical support devices in cardiogenic shock

### Table A

<table>
<thead>
<tr>
<th></th>
<th>MCS</th>
<th>IABP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Events Total</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thelie et al.</td>
<td>9</td>
<td>20</td>
</tr>
<tr>
<td>Burkhoff et al.</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>ISAR-SHOCK</td>
<td>6</td>
<td>13</td>
</tr>
<tr>
<td>IMPRESS in Severe Shock</td>
<td>11</td>
<td>24</td>
</tr>
<tr>
<td>Overall</td>
<td>35</td>
<td>77</td>
</tr>
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</table>

### Table B

**B**

```

<table>
<thead>
<tr>
<th>Number at risk</th>
<th>0</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>20</th>
<th>25</th>
<th>30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time after randomization (days)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IABP</td>
<td>71</td>
<td>53</td>
<td>48</td>
<td>43</td>
<td>40</td>
<td>39</td>
<td>39</td>
</tr>
<tr>
<td>MCS</td>
<td>77</td>
<td>52</td>
<td>50</td>
<td>47</td>
<td>47</td>
<td>45</td>
<td>42</td>
</tr>
</tbody>
</table>
```

**European Heart Journal (2017) 38, 3523–3531**
## Initial Vasoactive Management Considerations in Types of CS

<table>
<thead>
<tr>
<th>Cause or Presentation of CS</th>
<th>Vasoactive Management Considerations</th>
<th>Hemodynamic Rationale</th>
</tr>
</thead>
</table>
| Classic wet and cold        | Norepinephrine or dopamine<sup>144</sup>  
Inotropic agent<sup>210,211*</sup> | This subtype has low CI and high SVR. Consider hemodynamic stabilization with norepinephrine (preferred in tachycardia or arrhythmias) or dopamine (∆HR preferred but associated with higher risk of arrhythmias).
Consider addition of inotropic agent when stabilized and after revascularization (MI only) |
| Euvolemic cold and dry      | Norepinephrine or dopamine<sup>144</sup>  
Inotropic agent<sup>210,211</sup>  
Small fluid boluses | Consider hemodynamic stabilization with norepinephrine (preferred in tachycardia or arrhythmias) or dopamine (∆HR preferred but associated with higher risk of arrhythmias)  
Consider addition of inotropic agent when stabilized and after revascularization (MI only)  
LVEDP may be low, and patients may tolerate fluid boluses |
| Vasodilatory warm and wet or mixed cardiogenic and vasodilatory | Norepinephrine  
Consider hemodynamics-guided therapy | This subtype has low SVR |
| RV shock                    | Fluid boluses<sup>144,145</sup>  
Norepinephrine, dopamine, or vasopressin<sup>144,212,213</sup>  
Inotropic agents<sup>144*</sup>  
Inhaled pulmonary vasodilators<sup>2,14</sup> | Hemodynamic goals include maintaining preload, lowering RV afterload (PVR), treating absolute or relative bradycardias, and maintaining atrioventricular synchrony  
Dopamine (∆HR preferred but associated with arrhythmia risk)  
Vasopressin may raise SVR and have neutral effect on PVR  
Consider adding or transitioning to inotrope after initial hemodynamic stabilization and revascularization |
| Normotensive shock          | Inotropic agent or vasopressor | Initial inotropic therapy may be appropriate given that this subtype has SBP >90 mm Hg and relatively high SVR |

---

Goals of treatment in AHF - ESC 2016

**Immediate (ED/ICU/CCU)**
- Improve haemodynamics and organ perfusion.
- Restore oxygenation.
- Alleviate symptoms.
- Limit cardiac and renal damage.
- Prevent thrombo-embolism.
- Minimize ICU length of stay.

**Intermediate (in hospital)**
- Identify aetiology and relevant co-morbidities.
- Titrate therapy to control symptoms and congestion and optimize blood pressure.
- **Initiate and up-titrate disease-modifying pharmacological therapy.**
- Consider device therapy in appropriate patients.

**Pre-discharge and long-term management**
- Develop a careplan that provides:
  - A schedule for up-titration and monitoring of pharmacological therapy.
  - Need and timing for review for device therapy.
  - Who will see the patient for follow-up and when.
- Enrol in disease management programme, educate, and initiate appropriate lifestyle adjustments.
- Prevent early readmission.
- Improve symptoms, quality of life, and survival.
TAKE HOME MESSAGES

• Identify cause, immediate echo
• Stabilize patients, ensure vital functions, treat cause
Keep MAP >60-65mmHg
• Temporarily stop/avoid BB, ACEI
• Coronary angiogram in ACS
• Continuously monitor perfusion: skin, mental status, blood gases, lactate, urine output
• NIV if possible to avoid intubation
• PAC in complicated cases
• Only after hemodynamic status has been stabilized, treat with furosemide and MRA
• Do not rush with BB and ACEI
THANK YOU