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ESC Guidelines

Executive summary of the guidelines on the diagnosis and treatment of acute heart failure

The Task Force on Acute Heart Failure of the European Society of Cardiology

Endorsed by the European Society of Intensive Care Medicine (ESICM)

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Introduction

AHF Guidelines:

• provide an approach to diagnosis and treatment
• describe the rationale for therapeutic decisions

The recommendations prepared by the Acute Heart Failure Task Force were approved by the Committee for Practice Guidelines (CPG) of the ESC and the European Society of Intensive Care Medicine (ESICM).
### Classes of Recommendations

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Condition for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective</td>
</tr>
<tr>
<td>II</td>
<td>Condition for which there is conflicting evidence and/or a divergence of opinion about the usefulness / efficacy of a procedure or treatment</td>
</tr>
<tr>
<td>IIa</td>
<td>Weight of evidence /opinion is in favour of usefulness / efficacy</td>
</tr>
<tr>
<td>IIb</td>
<td>Usefulness/efficacy is less well established by evidence / opinion</td>
</tr>
<tr>
<td>III</td>
<td>Evidence or general agreement that the treatment is not useful/effective and in some cases may be harmful</td>
</tr>
</tbody>
</table>

### Levels of Evidence

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Data derived from multiple randomised clinical trials or meta-analyses</td>
</tr>
<tr>
<td>B</td>
<td>Data derived from a single randomised trial or large non-randomised studies</td>
</tr>
<tr>
<td>C</td>
<td>Consensus of opinion of experts and/or small studies, retrospective studies, registries</td>
</tr>
</tbody>
</table>
Definitions and Aetiology

• AHF is defined as the rapid onset of symptoms and signs, secondary to abnormal cardiac function.
• Cardiac dysfunction can be related to systolic or diastolic dysfunction, to abnormalities in cardiac rhythm or to preload and afterload mismatch.
• It is often life threatening and requires urgent treatment.
Definitions and Aetiology

(1) Decompensation of chronic heart failure

(2) Acute coronary syndromes
   (a) AMI / UAP / ischaemic dysfunction,
   (b) mechanical complications of AMI
   (c) right ventricular infarction

(3) Hypertensive crisis

(4) Acute arrhythmia (VT, VF, AF, SVT)
Definitions and Aetiology

(5) Cardiomyopathy and myocarditis

(6) Valvular regurgitation

(7) Aortic stenosis

(8) Acute myocarditis

(9) Cardiac tamponade

(10) Aortic dissection

(11) Post-partum cardiomyopathy
Definitions and Aetiology

(11) Non cardiovascular precipitating factors
   (a) poor compliance for medical treatment
   (b) volume overload
   (c) infections, especially pneumonia, septicaemia
   (d) severe brain injury
   (e) major surgery
   (f) reduced renal function
   (g) asthma
   (h) drug abuse
   (i) alcohol abuse

(12) High output syndromes
   (a) septicaemia
   (b) thyrotoxicosis
   (c) anaemia
   (d) shunt syndromes
Classification

Patients with AHF present with six distinct clinical conditions.
**Clinical Conditions**

**Acute decompensation of CHF**: Signs and symptoms are mild

- Heart rate +/-
- SBP +/-
- CI +/-
- PCWP +
- Diuresis +
- Hypoperfusion +/-

**AHF with pulmonary oedema**: severe respiratory distress with rales over the lungs

- Heart rate +
- SBP +/-
- CI -
- PCWP ++
- Diuresis +
- Hypoperfusion +/-
Clinical Conditions
Cardiogenic shock

Low output syndrome: reduced BP, low urine output, tissue hypoperfusion
- Heart rate +
- SBP -
- CI -
- PCWP +
- Diuresis -
- Hypoperfusion +

Severe Cardiogenic shock: low BP, organ hypoperfusion, anuria
- Heart rate ++
- SBP --
- CI --
- PCWP ++
- Diuresis --
- Hypoperfusion ++
Clinical Conditions

**Hypertensive AHF**: Signs and symptoms of AHF with high BP and preserved LVEF

- Heart rate +
- SBP ++
- CI +/-
- PCWP +
- Diuresis +/-
- Hypoperfusion +/-
**Clinical Conditions**

- **High output failure:**
  - signs of increased cardiac output with elevated heart rate with warm periphery

- **Right heart failure:**
  - low output syndrome with increased JVP, tender hepatomegaly and hypotension

- Heart rate +
- SBP +/-
- CI +
- PCWP +/-
- Diuresis +
- Hypoperfusion +/-

- Heart rate +/-
- SBP -
- CI -
- PCWP -
- Diuresis +/-
- Hypoperfusion +/-
**Killip Classification**

A clinical estimate of the severity of LV dysfunction in the treatment of AMI

**Class I** - No heart failure. No clinical signs of cardiac decompensation.

**Class II** - Heart failure. Diagnostic criteria include rales, S3 gallop and pulmonary venous hypertension. Pulmonary congestion with wet rales up to half of the lung fields.

**Class III** - Severe heart failure. Pulmonary edema with rales in all lung fields.

**Class IV** - Cardiogenic shock. Signs include hypotension (systolic BP < 90 mmHg), and evidence of peripheral vasoconstriction such as oliguria, cyanosis and diaphoresis.
Forrester Classification

Normal

Diuretics vasodilators

Pulmonary oedema

Hypovolemic

Normal blood pressure:
Vasodilators
Reduced blood pressure:
Inotropics or vasopressors

Pulmonary congestion
PCWP: 18 mmHg

Tissue perfusion
Cardiac index: 2.2 l/min/m²

Fluid administration
### Evaluation of acutely decompensated chronic heart failure

#### Congestion lungs

<table>
<thead>
<tr>
<th>Tissue perfusion</th>
<th>Clinical Classifications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry and warm</td>
<td>Wet and warm</td>
</tr>
<tr>
<td>Dry and Cold</td>
<td>Wet and cold</td>
</tr>
</tbody>
</table>
Diagnostic algorithm

- Clinical assessment
- Patient history
- ECG
- X-ray
- $O_2$ saturation
- CRP, electrolytes, creatinine
- BNP/NT-proBNP, troponin
- Echocardiography in all patients as soon as possible
Suspected Acute Heart Failure

Assess symptoms & signs

Heart Diseases? ECG/BNP/X-Ray?
- Abnormal
  - Evaluate cardiac function by echocardiography
    - Abnormal
      - Heart failure
        - Characterize type and severity
          - Selected tests, (angio, haemodynamic monitoring)

- Normal
  - Consider other diagnosis
    - Normal
Assessment of Ventricular Function

Left Ventricular Ejection Fraction (LVEF)

- Reduced LVEF
- <40%
- "Preserved" LVEF

- Systolic LV Dysfunction
  - Transient Systolic Dysfunction
  - Diastolic Dysfunction

- Error in diagnose (no heart failure)
**Laboratory tests**

<table>
<thead>
<tr>
<th>Test</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Routine haematology</td>
<td>always</td>
</tr>
<tr>
<td>Platelet count</td>
<td>always</td>
</tr>
<tr>
<td>Creatinine/urea</td>
<td>always</td>
</tr>
<tr>
<td>Electrolytes</td>
<td>always</td>
</tr>
<tr>
<td>Blood Glucose</td>
<td>always</td>
</tr>
<tr>
<td>Troponin (CKMB)</td>
<td>always</td>
</tr>
<tr>
<td>Arterial blood gases</td>
<td>always</td>
</tr>
<tr>
<td>CRP</td>
<td>always</td>
</tr>
<tr>
<td>D-dimer</td>
<td>always</td>
</tr>
<tr>
<td>Transaminases</td>
<td>consider</td>
</tr>
<tr>
<td>Urinanalysis</td>
<td>consider</td>
</tr>
<tr>
<td>BNP or NT-proBNP</td>
<td>consider</td>
</tr>
<tr>
<td>INR</td>
<td>if anticoagulated or severe HF</td>
</tr>
</tbody>
</table>
Treatment goals

- The goals in the treatment of heart failure are to improve clinical symptoms and outcomes.

- Management strategy should be based on clinical, laboratory and haemodynamic findings.
Goals of treatment of the patient with AHF

**Clinical**
- Symptoms (Dyspnoea and/or fatigue)
- Clinical signs
- Body weight
- Diuresis
- Oxygenation

**Haemodynamic**
- PCWP to <18 mmHg
- CO and/or SV

**Outcome**
- Length of stay in the ICU
- Duration of hospitalization
- Time to hospital re-admission
- Mortality

**Laboratory**
- Serum electrolytes normal
- BUN
- Plasma BNP
- Blood glucose normalisation

**Tolerability**
- Low rate of withdrawal from therapy
- Low incidence of adverse effects
Initial management

- Instrumentation and choice of therapy are carried out according to clinical priorities.
- Resuscitative measures may be required with life threatening complications.
- ECG and \textit{SpO2}, an iv line and arterial line can be useful for monitoring.
Initial management

- Correct hypoxia and increase cardiac output, renal perfusion, sodium excretion and urinary output.

- Ultimately ultrafiltration or dialysis may be required.

- Devices may be indicated such as an intravascular balloon pump, assisted ventilation, or a circulatory assist device as temporary measure or as bridge for heart transplantation.
Initial management

- Oxygen by face mask or CPAP (SpO2 target >95%)
- iv morphine (2.5–5 mg prn)
- iv loop diuretic therapy
- Vasodilatation by nitrate or nitroprusside
- Inotropic support with severe AHF or hypotension
- iv fluids if low filling pressure
- Concomitant metabolic conditions treated according to the diagnostic work-up and laboratory status
Initial management

- Patients with ACS or serious mechanical cardiac disorders should proceed rapidly to angiography and catheterisation for therapeutic measures including PCI or surgery.
Steps of care and treatment algorithm in AHF

Acute Heart Failure

- Definitive diagnosis

Diagnosis algorithm

- Definitive treatment

Immediate resuscitation

Patient distressed or in pain

- YES
  - Analgesia or sedation

- NO
  - Arterial oxygen saturation > 95%

- NO
  - Increase FiO₂, Consider CPAP, NIPPV

- YES
  - Pacing, Antiarrhythmics etc...

- NO
  - Normal heart rate and rhythm
Invasive Monitoring with PAC may be required

Mean BP > 70 mmHg

Adequate preload

Adequate CO
reversal of acidosis
SvO₂ > 65%
signs of adequate organ perfusion

Vasodilators, Consider diuresis if volume overload

Fluid challenge

Consider inotropes or further afterload reduction

Reassess frequently
INVASIVE MONITORING

Invasive haemodynamic monitoring may assist in decision making with volume loading, diuretics and/or vasoactive agents in severe AHF.
# Haemodynamic findings

<table>
<thead>
<tr>
<th>CI</th>
<th>Decreased</th>
<th>Decreased</th>
<th>Decreased</th>
<th>Decreased</th>
<th>Maintained</th>
</tr>
</thead>
<tbody>
<tr>
<td>L/min/m²</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCWP</td>
<td>Low</td>
<td>High or Normal</td>
<td>High</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>&gt;85</td>
<td>&lt;85</td>
<td>&gt;85</td>
<td></td>
<td></td>
</tr>
<tr>
<td>mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Outline of therapy**

- **Fluid loading**
  - Vasodilator (nitroprusside, NTG) fluid loading may become necessary
  - Consider inotropic agents (dobutamine, dopamine) and iv diuretics
  - Vasodilators (nitroprusside NTG) and iv diuretics and consider inotrope (dobutamine, levosimendan, PDEI)

- **iv diuretics**
  - If SBP low, vasoconstrictive inotropes
Specific Pharmacological Treatment

Requires an understanding of the pharmacodynamics and pharmacokinetics of each drug and its potential interactions, side-effects, and toxicity.
Diuretics

- Patients will usually require diuretics to treat pulmonary and peripheral congestion.
- Agents should usually be administered iv in the acute phase.
- Resistance to diuretics is a common problem.
## Diuretic treatment

<table>
<thead>
<tr>
<th>Severity of fluid retention</th>
<th>Diuretics</th>
<th>Dose (mg)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate</td>
<td>Furosemide, or</td>
<td>20-40</td>
<td>Oral or iv according to clinical symptoms</td>
</tr>
<tr>
<td></td>
<td>Bumetanide, or</td>
<td>0.5-1.0</td>
<td>Titrate dose according to clinical response</td>
</tr>
<tr>
<td></td>
<td>Torasemide</td>
<td>10-20</td>
<td>Monitor Na+, K+, creatinine and blood pressure</td>
</tr>
<tr>
<td>Severe</td>
<td>Furosemide, or</td>
<td>40-100</td>
<td>iv</td>
</tr>
<tr>
<td></td>
<td>Furosemide infusion</td>
<td>5-40 mg/h</td>
<td>Better than very high bolus doses</td>
</tr>
<tr>
<td></td>
<td>Bumetanide, or</td>
<td>1-4</td>
<td>Orally or iv</td>
</tr>
<tr>
<td></td>
<td>Torasemide</td>
<td>20-100</td>
<td>orally</td>
</tr>
<tr>
<td>Severity of fluid retention</td>
<td>Diuretics</td>
<td>Dose (mg)</td>
<td>Comments</td>
</tr>
<tr>
<td>----------------------------</td>
<td>-----------</td>
<td>-----------</td>
<td>----------</td>
</tr>
<tr>
<td>Refractory to loop diuretics</td>
<td>Add HCTZ, or</td>
<td>25-30 twice daily</td>
<td>Combination with loop diuretic better than very high dose of loop diuretic alone</td>
</tr>
<tr>
<td></td>
<td>Metolazone, or</td>
<td>2.5-10 once daily</td>
<td>Metolazone more potent if creatinine clearance &lt; 30 ml/min</td>
</tr>
<tr>
<td></td>
<td>Spironolactone</td>
<td>25-30 once daily</td>
<td>Spironolactone best choice if patient not in renal failure and normal or low serum K+</td>
</tr>
<tr>
<td>In case of alkalosis</td>
<td>Acetazolamide</td>
<td>0.5</td>
<td>iv</td>
</tr>
<tr>
<td>Refractory to loop diuretics and thiazides</td>
<td>Add dopamine for renal vasodilation, or dobutamine as an inotropic agent</td>
<td></td>
<td>Consider ultrafiltration or haemodialysis of co-existing renal failure and adequate BP</td>
</tr>
</tbody>
</table>
Managing resistance to diuretics

- Restrict Na+/water intake and follow electrolytes
- Volume repletion in cases of hypovolaemia
- Increase dose and/or frequency of administration of diuretics
- Use iv administration as bolus, or iv infusion

- Combine diuretics
  - loop diuretic + HCTZ
  - loop diuretic + spironolactone
  - loop diuretic + metolazone

- Combine diuretic therapy with dopamine, or dobutamine
- Reduce the dose of ACE-inhibitor or ARB or use very low doses
- Consider ultrafiltration
AHF with systolic dysfunction

Oxygen/CPAP
furosemide +/- vasodilator
Clinical evaluation

SBP > 100 mmHg
Vasodilator
(NTG, nitroprusside, nesiritide)
Good response
Oral therapy – furosemide, ACE-I

SBP 85-100 mmHg
Vasodilator and/or inotropic
(dobutamine, PDEI, or levosimendan)
No response
– inotropic agents

SBP < 85 mmHg
Volume loading?
Inotrope and/or
dopamine >5 µ/kg/min
and/or norepinephrine
## Vasodilator: Glyceryl Trinitrate, 5-mononitrate

<table>
<thead>
<tr>
<th>Indication</th>
<th>AHF, when blood pressure adequate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dosing</td>
<td>Start 20 µg/min, increase to 200 µg/min</td>
</tr>
<tr>
<td>Main side-effects</td>
<td>Hypotension, headache</td>
</tr>
<tr>
<td>Other</td>
<td>Tolerance on continuous use</td>
</tr>
</tbody>
</table>

## Vasodilator: Isosorbide dinitrate

<table>
<thead>
<tr>
<th>Indication</th>
<th>AHF, when blood pressure adequate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dosing</td>
<td>Start with 1mg/h increase to 10mg/h</td>
</tr>
<tr>
<td>Main side-effects</td>
<td>Hypotension, headache</td>
</tr>
<tr>
<td>Other</td>
<td>Tolerance on continuous use</td>
</tr>
<tr>
<td>Drug</td>
<td>Vasodilator</td>
</tr>
<tr>
<td>------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Indication</td>
<td>Hypertensive crisis, cardiogenic shock combined with inotropes</td>
</tr>
<tr>
<td>Dosing</td>
<td>0.03 µg/kg/min</td>
</tr>
<tr>
<td>Main side-effects</td>
<td>Hypotension, isocyanate toxicity</td>
</tr>
<tr>
<td>Other</td>
<td>Drug is light sensitive</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Drug</th>
<th>Vasodilator</th>
<th>Nesiritide (not approved by EMEA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication</td>
<td>Acute decompensated heart failure,</td>
<td></td>
</tr>
<tr>
<td>Dosing</td>
<td>Bolus 2 µg/kg + infusion 0.015–0.03 µg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Main side-effects</td>
<td>Hypotension</td>
<td></td>
</tr>
</tbody>
</table>
Inotropic Agents

• Are often required in patients with moderate or severe heart failure and hypotension.

• Tachycardia and vasoconstriction are frequently observed.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Bolus</th>
<th>Infusion rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine</td>
<td>No</td>
<td>2–20 µg/kg/min β+</td>
</tr>
<tr>
<td>Dopamine</td>
<td>No</td>
<td>&lt; 3 µg/kg/min : renal effect (δ+) 3–5 µg/kg/min : inotropic (β+) &gt; 5µg/kg/min (β+), vasopressor (α+)</td>
</tr>
<tr>
<td>Milrinone</td>
<td>25–75µg/kg over 10–20 min</td>
<td>0.375–0.75 µg/kg/min</td>
</tr>
<tr>
<td>Enoximone</td>
<td>0.25–0.75 mg/kg</td>
<td>1.25–7.5 µg/kg/min</td>
</tr>
<tr>
<td>Levosimendan</td>
<td>12–24 µg/kg over 10 min</td>
<td>0.1 µg/kg/min which can be decreased to 0.05 or increased to 0.2 µg/kg/min</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>NO</td>
<td>0.2–1.0 µg/kg/min</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>1 mg can be given iv at resuscitation, may be repeated after 3–5 min, endotracheal route is not favoured</td>
<td>0.05–0.5 µg/kg/min</td>
</tr>
</tbody>
</table>
Cardiac disorders and AHF requiring surgical treatment

- Cardiogenic shock after AMI patients with multi vessel CAD
- Post-infarction VSR
- Free wall rupture
- Acute decompensation with valve disease
- Aortic aneurysm or aortic dissection into the pericardial sac
• **Acute mitral regurgitation from:**
  - Ischaemic papillary muscle rupture
  - Ischaemic papillary muscle dysfunction
  - **Myxomatous chordal rupture**
  - **Endocarditis**
  - Trauma

• **Acute aortic regurgitation from:**
  - **Endocarditis**
  - Aortic dissection
  - Closed chest trauma

• Ruptured aneurysm of the sinus of Valsalva

• Acute decompensation of chronic cardiomyopathy requiring support by mechanical assist devices.
Treatment of rhythm disturbances

• Rhythm disturbances may frequently precipitate or aggravate episodes of decompensation and should be treated aggressively.
Treatment of arrhythmias in acute heart failure

- **VF or pulseless VT:**
  Defibrillate with 200-300J. If refractory, inject epinephrine 1 mg or vasopressin 40 IU and/or amiodarone 150-300 mg.

- **VT:**
  If unstable cardiovert. If stable, amiodarone or lidocaine may achieve medical cardioversion.
• **Sinus tachycardia or SVT:**
  Use $\beta$-blocking agent when haemodynamically stable: metoprolol 5 mg iv as a slow bolus. Adenosine may be used to slow AV conduction or to cardiovert re-entry tachycardia.

• **Atrial fibrillation or flutter:**
  Cardiovert if possible. Digoxin 0.5-1.0 mg iv, $\beta$-blocking agent, or iv amiodarone (300 mg/30 min followed 50-100 mg/h), may be used to slow AV conduction. Amiodarone may induce medical cardioversion without compromising haemodynamics. Patients should be anticoagulated.
• **Bradycardia:**
  Atropine 0.25-0.5 mg iv to total of 1-2 mg.
  Isoproterenol iv from 2-12 µg/min.
  If bradycardia persists, transcatheter or transvenous pacing.
  Theophylline may be used with atropine-resistant bradycardia with bolus of 0.25-0.5 mg/kg and infusion at 0.2-0.4 mg/kg/h.
Specific conditions may require surgical management. These cardiac disorders must be detected promptly. The indications for IAPB, LVAD or cardiac transplantation are discussed in the executive summary of these guidelines.
Echocardiography

Low EF
No signs of mechanical complications

Diagnosis: cardiogenic shock from loss of ventricular myocardium

Medical therapy : Consider
• IABP
• Mechanical ventilation
• PCI or CABG
• VAD
• Heart transplant
Echocardiography

Echo signs of acute severe MR
+/- visualisation of ruptured papillary muscle

If diagnosis uncertain:
Consider TEE
If TEE non diagnosis:
Consider PAC
- To exclude VSR

Diagnosis
Acute mitral regurgitation

If diagnosis uncertain:
Consider TEE
If TEE non diagnosis:
Consider PAC
- To exclude VSR

Medical therapy

Unstable patient: consider
- IABP
- Mechanical ventilation
- PAC

Stable patient

Coronary angiography

Coronary angiography

Urgent surgical therapy

Immediate surgical correction

ESC Guidelines on the Diagnosis and Treatment of Acute Heart Failure
Echocardiography

- Pericardial effusion
- Echo densities in the effusion
- Echo signs of tamponade

Diagnosis: Free wall rupture

Pericardiocentesis
- Fluids
- Inotropes
- Consider IABP

Immediate surgical correction
Echocardiography

Ventricular Septal rupture
- Site
- Size
- Qp:Qs

Diagnosis: VSR

Medical Therapy
- Stable patient

Coronary Angiography
- Urgent surgical correction

Diagnosis uncertain
- PAC
- Oxymetry
- O₂ Step up >5% RA-RV

Unstable patient consider
- IABP
- Mechanical ventilation
- PAC

Coronary Angiography

Immediate surgical correction
• The patient with AHF may recover to their clinical status or deteriorate depending on the management, aetiology and precipitating mechanisms.

• Appropriate management of chronic HF is required after stabilisation. Adequate follow-up strategy should be planned.

• Treatment should be performed according to the principles introduced in these guidelines and in the ESC task force guidelines for the diagnosis and treatment of chronic heart failure [European Heart Journal, 2005;26:1115-1140].