

Current ESC/ESICM and ACCF/AHA guidelines for the diagnosis and management of acute heart failure in adults – are there differences?

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KEY WORDS

acute heart failure,
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inotropes,
vasodilators

ABSTRACT

There are many similarities between the recommendations of the European and American societies (i.e., the European Society of Cardiology, the European Society of Intensive Care Medicine, the North American College of Cardiology, and the American Heart Association) on the management of acute heart failure (AHF). There is a consensus that the symptoms of AHF should be relieved as rapidly as possible, which requires combining oxygen, noninvasive ventilation, vasodilators, and diuretics. There is also a consensus that long-lasting oral treatments such as β-blockers and angiotensin-converting enzyme inhibitors or angiotensin-receptor blockers should be administered to patients unless they are hemodynamically unstable. The diagnosis of AHF is based on clinical signs. However, electrocardiography and chest X-ray should be performed in all AHF patients. Natriuretic peptides should be measured in doubtful cases to either confirm or exclude the diagnosis of AHF. Echocardiography should be promptly performed by cardiologists. Because a unanimous consensus between European and North American societies has now been reached, the next step is to apply the guidelines in all medical units worldwide in order to minimize morbidity and mortality of AHF patients.

Introduction Former European¹ and American² guidelines on acute heart failure (AHF) provided divergent views on important issues such as handling volemia, using vasodilators or positive inotropic agents. Last year, the European Society of Cardiology (ESC) in collaboration with the European Society of Intensive Care Medicine (ESICM) published the “ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008”.³ In the early 2009, the American College of Cardiology (ACC) and the American Heart Association (AHA) published the “2009 focused update: ACCF/AHA guidelines for the diagnosis and management of heart failure in adults”.⁴ For the first time, both the European and American guidelines have an extensive section on AHF. The societies share views on many critical issues including definition, diagnosis and treatment, which may result from the fact that an increasing number of studies and surveys are conducted on both continents. The present paper reviews

the position of the ESC/ESICM on the management of AHF in Europe, which is in many ways similar to ACC/AHA recommendations for the United States.

Definitions AHF is defined as a rapid onset of or deterioration in the signs and symptoms of heart failure (HF), requiring urgent treatment. It may present as new HF (also called *de novo* HF) or worsening HF in the presence of chronic HF.

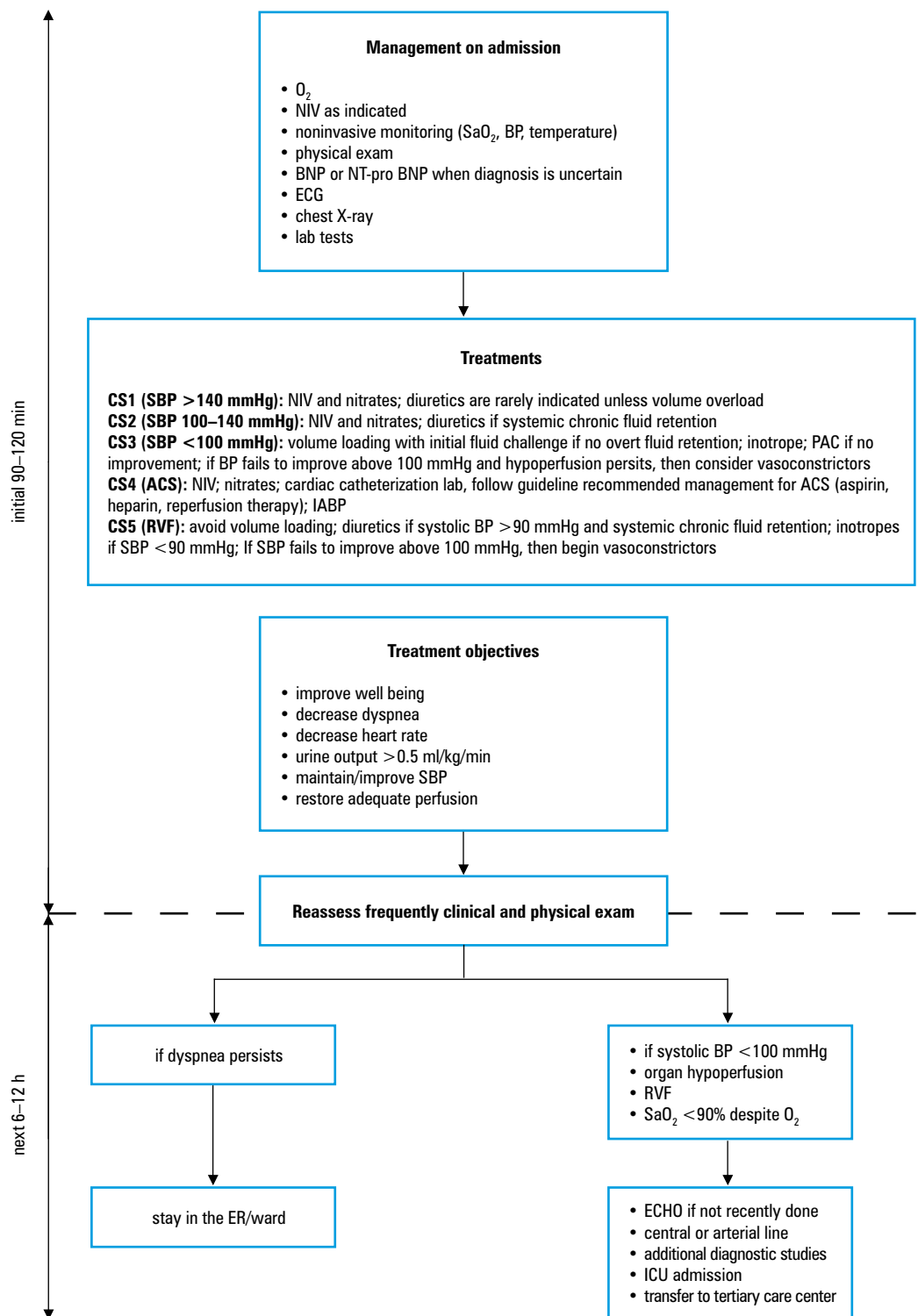
ESC/ESICM guidelines define 6 clinical categories of AHF. Four of them are common:

- 1 worsening or decompensated chronic HF
- 2 pulmonary edema (patients present with severe respiratory distress, tachypnea, orthopnea with rales over the lung fields; arterial oxygen saturation is usually <90% on room air)
- 3 hypertensive HF (signs and symptoms of HF accompanied by high blood pressure [BP] and usually relatively preserved left ventricular [LV] systolic function)

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FIGURE Proposed algorithm for the prehospital and early in-hospital management of patients with acute heart failure syndromes

Abbreviations: ACS – acute coronary syndrome, BNP – B-type natriuretic peptide, BP – blood pressure, CS – clinical scenario, ECG – electrocardiogram, ECHO – echocardiography, ER – emergency room, ICU – intensive care unit, INAB – intrathoracic balloon pump, NIV – noninvasive ventilation, NT-proBNP – N terminal pro-BNP, PAC – pulmonary artery catheter, RVF – right ventricle failure, SaO₂ – arterial oxyhemoglobin saturation, SBP – systolic blood pressure



4 acute coronary syndrome (ACS) and AHF (~15% of patients with ACS have signs and symptoms of AHF that are frequently associated with an arrhythmia).

The two other categories are less common: cardiogenic shock that is characterized by reduced systolic BP (<90 mmHg or a drop of mean arterial pressure >30 mmHg) and absent or low urine output (<0.5 ml/kg/h), and isolated right HF.

Because prehospital and early management of AHF is primarily based on signs and symptoms, European and American experts proposed categorization and an algorithm based on

systolic BP on admission (FIGURE, TABLE).⁵ Indeed, in patients admitted with AHF and systolic BP >140 mmHg, LV systolic function is likely preserved, while at systolic BP of 100–140 mmHg LV systolic function is limited, and many patients with impaired LV systolic function exhibit systolic BP <100 mmHg.

Diagnosis of acute heart failure and monitoring on admission The diagnosis of AHF should be quasi-exclusively based on the symptoms present on admission and clinical findings. The latter involve a search of evidence of nonadherence

TABLE Clinical scenarios in acute heart failure syndrome

Clinical scenario	Characteristics
CS1	SBP > 140 mmHg symptoms develop abruptly predominantly diffuse pulmonary edema minimal systemic edema (patient may be euvolemic or hypovolemic) acute elevation of filling pressure often with preserved LVEF vascular pathophysiology
CS2	SBP 100–140 mmHg symptoms develop gradually, together with a gradual increase in body weight predominantly systemic edema minimal pulmonary edema chronic elevation of filling pressure, including increased venous pressure and elevated pulmonary arterial pressure manifestations of organ dysfunction (renal impairment, liver dysfunction, anemia, hypoalbuminemia)
CS3	SBP < 100 mmHg rapid or gradual onset of symptoms predominantly signs of hypoperfusion minimal systemic and pulmonary edema elevation of filling pressure two subsets: clear hypoperfusion or cardiogenic shock no hypoperfusion/cardiogenic shock
CS4	symptoms and signs of acute heart failure evidence of ACS isolated elevation of cardiac troponin is inadequate for CS4 classification
CS5	rapid or gradual onset no pulmonary edema right ventricular dysfunction signs of systemic venous congestion

Abbreviations: ACS – acute coronary syndrome, LVEF – left ventricular ejection fraction, SBP – systolic blood pressure

to medical advice as well as physical examination assessing peripheral perfusion, skin temperature, systemic congestion, cardiac and pulmonary auscultation.

Routine basic observations of temperature, respiratory rate, heart rate, BP, oxygenation, urine output, and the electrocardiogram (ECG) are mandatory and should be started as soon as possible after the patient's arrival to the emergency unit. A pulse oximeter should be used on admission to capture initial arterial oxygen saturation and is required later only in patients treated with oxygen. Invasive monitoring is required in a small number of patients and involves arterial line in case of hemodynamic instability, central venous line to measure central venous pressure and administer drugs, and/or pulmonary artery catheter in hemodynamically unstable patients who are not sufficiently responding to traditional treatments.

The guidelines considered ECG and chest X-ray as appropriate procedures in all patients with AHF.

Concerning laboratory tests, the initial diagnostic evaluation of patients with AHF includes full blood count, sodium, potassium, urea, creatinine, glucose, albumin, hepatic enzymes, troponins, and international normalized ratio. However, arterial blood analysis is not required in most patients with AHF as arterial oxygen saturation is widely used. Natriuretic peptides are more and more commonly measured in the initial evaluation of patients admitted with acute dyspnea. This helps to confirm or exclude the diagnosis of AHF.

Evaluation of heart and coronary functions If possible, cardiologists should evaluate heart and coronary functions. This, however, depends on the facilities of individual institutions.

ECHO/Doppler imaging should be used to evaluate and monitor regional and global left and right ventricular (RV) systolic function, diastolic function, valvular structure and function, pericardial pathology, mechanical complications of acute myocardial infarction, and evidence of dyssynchrony. ECHO/Doppler findings may influence treatment strategy.

In cases of AHF and evidence of myocardial ischemia, such as unstable angina or ACS, coronary angiography is recommended in patients without strong contraindications. Successful reperfusion treatment followed by aggressive medical treatment (IIb/IIIa glycoprotein antagonists, oral antiplatelet agents and statins) has been shown to improve prognosis.

Organization of AHF treatment As presented in the [FIGURE](#), immediate goals of AHF treatment are to improve symptoms and to stabilize hemodynamic condition in the first 90–120 min after admission. When dyspnea is relieved and hemodynamic condition secured, most AHF patients will require to continue (in case of worsening chronic HF) or to introduce (in case of *de novo* HF) long-term treatment of chronic HF.

Management in the first 90–120 min after admission

Guidelines, mostly based on expert opinions, recommend the following:

1 to administer oxygen as early as possible to achieve an arterial oxygen saturation $\geq 95\%$ ($>90\%$ in patients with chronic obstructive pulmonary disease). Patients with serious obstructive airway disease should be under special care to avoid hypercapnia.

2 Noninvasive ventilation (NIV) refers to all modalities that assist ventilation without the use of an endotracheal tube but rather with a sealed face mask. NIV with positive end-expiratory pressure (PEEP 5–10 cm H₂O) should be considered in every patient with acute cardiogenic pulmonary edema and hypertensive acute HF as early as possible, because it improves clinical parameters including respiratory distress. NIV with PEEP improves LV function by reducing LV afterload. NIV

should be used with caution in cardiogenic shock and RV failure.

3 IV boluses of morphine 2.5–5.0 mg should be considered in the early stage of treatment of patients admitted with severe AHF, especially if they present with restlessness, dyspnea, anxiety or chest pain. Morphine relieves dyspnea and other symptoms in patients with AHF and may improve cooperation for the application of NIV.

4 An IV bolus of furosemide 20–40 mg (0.5–1.0 mg of bumetanide; 10–20 mg of torasemide) is recommended on admission. Patients should be assessed frequently in the initial phase to observe urine output. In patients with evidence of volume overload, an IV dose of furosemide may be increased according to renal function and a history of chronic oral diuretic use. In such patients, continuous infusion may also be considered after the initial starting dose. The total furosemide dose should remain <100 mg in the first 6 h and 250 mg in the first 24 h.

5 Nitroglycerin spray of 400 µg (2 puffs) every 5–10 minutes, buccally (isosorbide dinitrate 1 or 3 mg), is recommended in the early phase of AHF while preparing a continuous infusion of 10–20 µg/min, increased in increments of 5–10 µg/min every 3–5 minutes as needed. Those agents are recommended in patients without symptomatic hypotension, systolic BP <90 mmHg or serious obstructive valvular disease. They decrease systolic BP, decrease left and right heart filling pressures and systemic vascular resistance, and improve dyspnea.

6 Inotropic agents are recommended *only* in patients with low output states, with signs of hypoperfusion or congestion despite the use of vasodilators and/or diuretics. BP and heart rate should always be monitored. Dobutamine is usually initiated with a 2–3 µg/kg/min infusion rate without a loading dose. The infusion rate may then be progressively modified according to symptoms, diuretic response or clinical status. Its hemodynamic actions are dose-related, which can be increased to 15 µg/kg/min. Dopamine at low doses (\leq 2–3 µg/kg/min) has been shown to have limited effects on diuresis; it should be used with caution in patients with heart rate >100 bpm. Milrinone and enoximone are administered by a continuous infusion possibly preceded by a bolus dose in patients with stable BP. Phosphodiesterase inhibitor should be administered with caution in patients with coronary artery disease, because it may increase medium-term mortality. Levosimendan may be effective in patients with decompensated chronic HF and represents an alternative for patients on β -blocker therapy. Levosimendan may be administered as a bolus dose (3–12 µg/kg) during 10 min followed by a continuous infusion (0.05–2.0 µg/kg/min for 24 h). Vasopressors (noradrenaline) are not recommended as first-line agents and are only indicated in cardiogenic shock when the combination of an inotropic agent and fluid challenge fails to restore systolic BP >90 mmHg with inadequate

organ perfusion, despite an improvement in cardiac output.

Management of patients with acutely decompensated chronic HF treated with β -blockers and ACE inhibitors/ARBs

Ideally, β -blockers and ACE inhibitors/ARBs should be maintained in AHF patients already treated with these agents, unless patients are hemodynamically unstable. Specifically, the dose of β -blocker may need to be temporarily reduced or omitted in the presence of contraindications (e.g., bradycardia, advanced atrioventricular block, severe reactive airways disease or cardiogenic shock) or in cases of severe AHF and an inadequate response to initial therapy.

In AHF, with no former treatment with ACE inhibitor/ARB or β -blockers, all those medications should be initiated before discharge: β -blockers are started when the patient has been stabilized on an ACE inhibitor or ARB.

REFERENCES

- 1 Nieminen MS, Bohm M, Cowie MR, et al. 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. *Eur Heart J*. 2005; 26: 384–416.
- 2 Hunt SA, Abraham WT, Chin MH, et al. ACC/AHA 2005 Guideline update for the diagnosis and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure): developed in collaboration with the American College of Chest Physicians and the International Society for Heart and Lung Transplantation: endorsed by the Heart Rhythm Society. *Circulation*. 2005; 112: e154–e235.
- 3 Dickstein K, Cohen-Solal A, Filippatos G, et al. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008: the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care Medicine (ESICM). *Eur Heart J*. 2008; 29: 2388–2442.
- 4 Jessup M, Abraham WT, Casey DE, et al. 2009 focused update: ACCF/AHA guidelines for the diagnosis and management of heart failure in adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: developed in collaboration with the International Society for Heart and Lung Transplantation. *Circulation*. 2009; 119: 1977–2016.
- 5 Mebazaa A, Gheorghide M, Piña IL, et al. Practical recommendations for prehospital and early in-hospital management of patients presenting with acute heart failure syndromes. *Crit Care Med*. 2008; 36 (1 Suppl): S129–S139.

Aktualne wytyczne ESC/ESICM i ACCF/AHA dotyczące rozpoznawania i leczenia ostrej niewydolności serca u dorosłych – czy się różnią?

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naczynia, ostra
niewydolność serca

STRESZCZENIE

Jest wiele podobieństw w postępowaniu z chorymi z ostrą niewydolnością serca (ONS) między wytycznymi europejskimi (European Society of Cardiology i European Society of Intensive Care Medicine oraz północnoamerykańskimi (American College of Cardiology i American Heart Association). Istnieje zgodność stanowisk, że należy jak najszybciej dążyć do zniesienia objawów podmiotowych ONS, co wymaga zastosowania tlenu, wentylacji nieinwazyjnej, leków rozszerzających naczynia i diuretyków. Wytyczne zgodne są także co do leczenia przewlekłego β-blokerem i inhibitorem konwertazy angiotensyny lub blokerem receptora angiotensynowego, jeśli nie ma objawów niestabilności hemodynamicznej. ONS rozpoznaje się na podstawie objawów klinicznych, jednak u wszystkich chorych należy wykonać EKG i RTG klatki piersiowej. W razie wątpliwości dotyczących rozpoznania ONS należy oznaczyć stężenie peptydów natriuretycznych w celu jej potwierdzenia lub wykluczenia. Jeśli jest to możliwe, badanie echokardiograficzne powinien wykonać kardiolog. Wobec znacznej zgodności wytycznych europejskich i północnoamerykańskich, następnym krokiem powinno być ich wdrożenie na każdym oddziale na całym świecie w celu zminimalizowania chorobowości i śmiertelności u chorych z ONS.

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