Congestive Heart Failure

Michael C. Scott, MD[a,b], Michael E. Winters, MD[a,*]

KEYWORDS

- Congestive heart failure • Acute decompensated heart failure
- Noninvasive positive pressure ventilation • Nitrates
- Angiotensin converting enzyme inhibitors • Diuretics • Inotropes

KEY POINTS

- Many patients with acute decompensated heart failure do not have intravascular volume overload.
- The foundation of emergency department management of acute decompensated heart failure is the use of noninvasive positive pressure ventilation and nitrate medications.
- Diuretics should not be used until optimal preload and afterload reduction has been achieved.
- Morphine, nesiritide, β-blockers, and intraaortic balloon pump should not routinely be used in the management of patients with acute heart failure in the emergency department.

INTRODUCTION

Congestive heart failure (CHF) remains the most common reason for hospitalization in the United States for people aged 65 years and older, with more than 1 million patients admitted for this condition each year.[1,2] Despite improvements in diagnosis and treatment, approximately 300,000 deaths each year can be attributed to CHF. Furthermore, the in-hospital mortality rate can be as high as 12% for patients admitted with an acute exacerbation of CHF.[2] Patients with acute decompensated heart failure (ADHF) frequently present to the emergency department (ED) for evaluation; many of

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[a] Emergency Medicine/Internal Medicine/Critical Care Program, Department of Emergency Medicine, University of Maryland Medical Center, 110 South Paca Street, 6th Floor, Suite 200, Baltimore, MD 21201, USA; [b] Department of Medicine, University of Maryland Medical Center, 22 South Greene Street, Baltimore, MD 21201, USA
* Corresponding author. Department of Emergency Medicine, University of Maryland Medical Center, 110 South Paca Street, 6th Floor, Suite 200, Baltimore, MD 21201.
E-mail address: mwinters@umem.org

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them are critically ill, requiring immediate treatment. In 2007, CHF accounted for 6.3% of ED patients requiring hospital admission.[3] It is imperative that the emergency physician be expert in the rapid assessment and treatment of patients with ADHF. The article focuses on the management of the ED patient with ADHF, with attention to therapies that improve patient symptoms, morbidity, and mortality.

PATHOPHYSIOLOGY

Recent reports have improved our understanding of the pathophysiology of ADHF. Although some patients present with the textbook description of preexisting systolic dysfunction and progressive volume overload, most ED patients with ADHF have preserved systolic function and are not overloaded in terms of total body volume. In fact, most acutely ill ED patients with ADHF harbor processes much more complex than an excess of intravascular volume. These processes can be divided into 2 categories: cardiac failure and vascular failure.[4,5]

Patients with cardiac failure represent the textbook description of ADHF, that is, a patient with chronic heart failure who develops intravascular volume overload through a variety of mechanisms (eg, medication noncompliance, dietary noncompliance, acute kidney injury). Quite simply, their damaged heart cannot tolerate increases in intravascular volume, so edema develops in the lungs and peripheral tissues. In general, symptoms develop gradually in the patient with this type of ADHF.

Patients with vascular failure have an abrupt increase in vasoconstriction and afterload, resulting in acute decompensation. This sharp increase in afterload is believed to occur through neurohumoral pathways involving the sympathetic and renin–angiotensin–aldosterone axes.[4,5] Patients with vascular failure typically present with an acute onset of symptoms, often lack the classic signs of peripheral edema, and generally have preserved cardiac function. In addition, these patients are frequently hypertensive upon ED presentation and can be either euvolemic or hypovolemic.

CLASSIFICATION

The European Society of Cardiology has classified acute heart failure into distinct clinical syndromes (**Box 1**).[6,7] Some patients present with overlapping features, but their appropriate classification is important, because each requires a specific therapeutic approach.

Box 1
Classification of acute heart failure

- Acute decompensated heart failure
- Hypertensive acute heart failure
- Pulmonary edema
- Cardiogenic shock
- Right heart failure
- Acute coronary syndrome complicated by acute heart failure

Adapted from Dickstein K, Cohen-Solal A, Filippatos G, et al. ESC guidelines for the diagnosis and treatment of acute heart failure 2008. Eur J Heart Fail 2008;10:968.