Acutely Decompensated Heart Failure: Opportunities to Improve Care and Outcomes in the Emergency Department

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Each year about 550,000 new patients are diagnosed as having congestive heart failure, which for acutely symptomatic patients is also referred to as acutely decompensated heart failure. The incidence of congestive heart failure is approximately 10 per 1000 for Americans over the age of 65 years. Men and women are affected in equal numbers, and 5-year mortality has been reported to be as high as 50%. Increased longevity increases the likelihood that heart failure will develop as a consequence of pathophysiologic processes that gradually weaken the myocardium and the vascular system. Patients who present to the emergency department with complaints of shortness of breath, dyspnea on exertion, increasing lower extremity edema, and/or worsening fatigue should have heart failure included in the differential diagnosis. Heart failure patients experiencing symptoms consistent with cardiac ischemia, hypoxia, potentially lethal arrhythmias, marked hypertension, or hypotension should be immediately triaged to a critical care area. The approval of nesiritide by the U.S. Food and Drug Administration in 2001 has stimulated the development of revisions in strategies for the emergency department treatment of acute decompensated heart failure patients. The early use of nesiritide, along with topical nitroglycerin and a loop diuretic, may lead to more rapid resolution of these patients' acute symptoms and hemodynamic dysfunction.
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Key words: Congestive heart failure • Emergency department • Systolic dysfunction • Angiotensin • Natriuretic peptides

Epidemiology of Heart Failure

Approximately 5 million Americans have been given a diagnosis of congestive heart failure (CHF). Each year about 550,000 new patients are diagnosed as having CHF, which for acutely symptomatic patients is also referred to as acutely decompensated heart failure (ADHF). The incidence of CHF is approximately 10 per 1000 for Americans over the age of 65 years. Men and women are affected in equal numbers, and 5-year mortality has been reported to be as high as 50%. In 1999

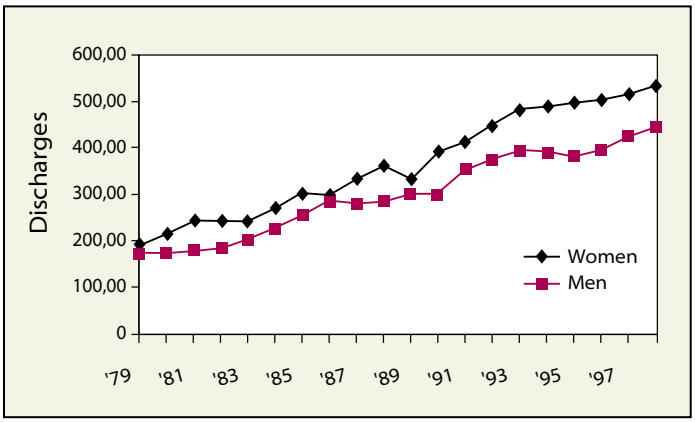


Figure 1. *Heart failure hospitalizations. The number of heart failure hospitalizations is increasing in both men and women.*

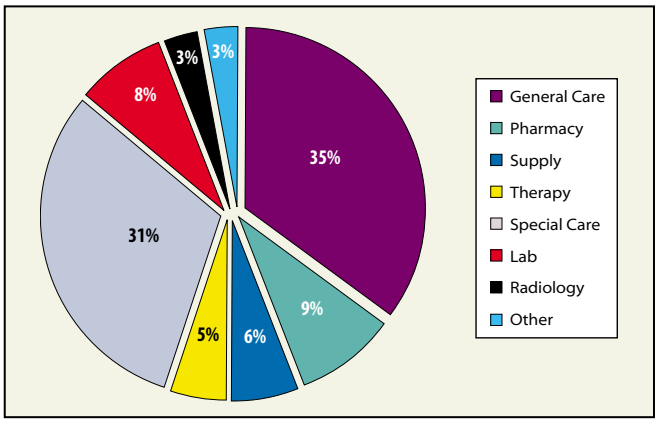


Figure 2. *Distribution of costs by cost center DRG 127 (CHR). Figure provided by Scios, Inc.*

CHF was the primary discharge diagnosis for 962,000 hospitalized patients.[1] This number represents an increase of more than 150% since 1979. The increase in the number of individuals with CHF may be due to the fact that a growing number of Americans are surviving diseases such as coronary artery disease, cancer, and severe infections that were responsible in the past for greater mortality at younger ages than is currently the case. Increased longevity increases the likelihood that heart failure will develop as a consequence of pathophysiologic processes that gradually weaken the cardiovascular system.

Approximately 5 million Americans have been given a diagnosis of congestive heart failure.

The estimated cost of treating patients with CHF was $38.1 billion in 1994. This represents 5.4% of total health care expenditures in that year.[2] CHF is the most expensive condition covered by Medicare. Recently the Centers for Medicare and Medicaid Services have provided financial incentives to encourage the care of CHF patients in observation units rather than in hospital wards. Figure 1 shows increases in heart failure hospitalizations and Figure 2 shows the cost of CHF by hospital cost center.

From the emergency department (ED) perspective, patients with CHF often present while they are experiencing symptoms of acute decompensation. In other instances CHF may be noted as a complicating factor in a patient with more than one coexisting disease process who comes to the ED with symptoms unrelated to CHF. Approximately 4 of 5 patients who receive treatment in the ED for ADHF require hospitalization. Of those ADHF patients presenting to the ED, 21% are experiencing their first episode of ADHF, and the remaining 79% have had prior hospital visits for the same condition.

Figure 3 demonstrates the frequency with which ADHF patients are readmitted with the same diagnosis at 1 month and 6 months. Readmission to the hospital for CHF patients may become necessary as a result of worsening of the underlying cause of CHF (Table 1) or as a result of a change in patient behavior with regard to adherence to diet and/or medication use (see Figure 4). Regardless of the cause, disruption of circulatory hemostasis causes the patient to develop the symptoms of ADHF. Increased intake of salt and water or nonadherence to a complicated medication regimen is commonly reported by ED patients with ADHF.

Definition of Heart Failure

Heart failure covers a spectrum of clinical entities defined by the patient's symptoms or by measurements of cardiac function. The clinical spectrum spans patients who have evidence of asymptomatic ventricular dysfunction to patients with severe pulmonary edema and hypotension.

In the normal heart, blood flows in the ventricles at low pressure during diastole and is then pumped out into the arterial system during systole at pressures sufficient to provide adequate perfusion to the organs of the body. Heart failure occurs when the right ventricle, the left ventricle, or both ventricles cannot generate suf-

ficient pressure during contraction to meet the metabolic needs of the body, or when the pressure needed to fill the chambers during diastole must be elevated to achieve adequate precontraction volumes. Heart failure due to systolic dysfunction is associated with abnormal ventricular emptying as would occur when ventricular contractility is impaired. As an example, ventricular contractility becomes impaired as a result of repeated myocardial necrosis. In the presence of systolic dysfunction, elevated left ventricular (LV) filling pressure causes retrograde transmission of elevated pressure to the left atrium and the pulmonary veins and capillaries. When pulmonary capillary wedge pressure exceeds approximately 20 mm Hg, fluid enters the pulmonary interstitium, causing pulmonary congestion. Clinically this pulmonary congestion results in symptoms such as dyspnea on exertion, and findings such as rales when auscultation is performed. Two thirds of CHF patients have systolic dysfunction.

Patients with diastolic dysfunction may exhibit normal ventricular contractility. In these patients diastolic relaxation is impaired, thereby impeding the filling of both ventricles. Under such circumstances pressure rises in the venous system until adequate levels are reached to maintain right ventricular filling. Restrictive cardiomyopathy and asymmetric ventricular hypertrophy are examples of conditions that can cause "stiffness" of the ventricular wall. Elevated pressure in the venous system drives plasma fluid into interstitial spaces, particularly in gravity-dependent parts of the body such as the legs and the sacral area. Highly vascular organs, such as the liver, may also become congested with fluid. Signs and symptoms include peripheral edema, jugular venous distention, and abdominal pain secondary to hepatomegaly. Approximately one third of CHF patients have diastolic dysfunction.

Patients with diastolic dysfunction may exhibit normal ventricular contractility.

Table 1
Common Etiologies of Acute Heart Failure

- Uncontrolled hypertension
- Ischemic heart disease
- Sustained cardiac arrhythmias
- Cardiomyopathy
  - EtOH, infiltrative
- Valvular heart disease
- Pericardial disease

Patients with advanced biventricular heart failure (systolic and diastolic dysfunction) will exhibit evidence of both right- and left-sided heart failure. Increased pulmonary capillary wedge pressure will cause pulmonary congestion, and increased peripheral venous pressure will cause peripheral edema. The challenge in managing these patients is maintaining the balance of salt and water hemostasis along with adequate organ perfusion. The New York Heart Association (NYHA) has devised a classification system for heart failure that is based on the degree of the patient's functional impairment (Table 2). The American Heart Association and American College of Cardiology

Figure 3. *Emergency department presentations. Approximately 90% of the emergency department visits for congestive heart failure result in hospitalizations. Figure provided by Scios, Inc.*

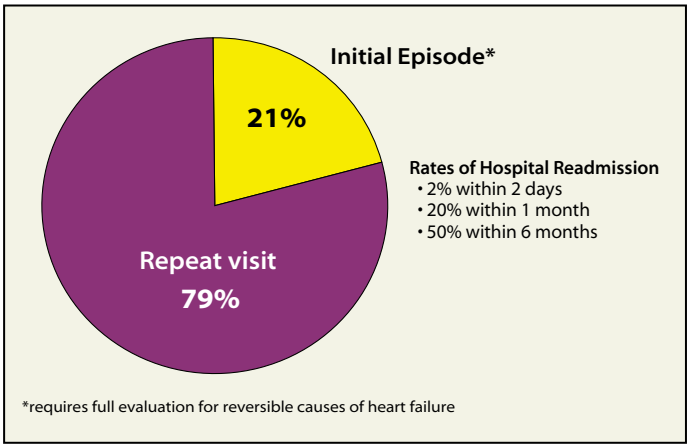
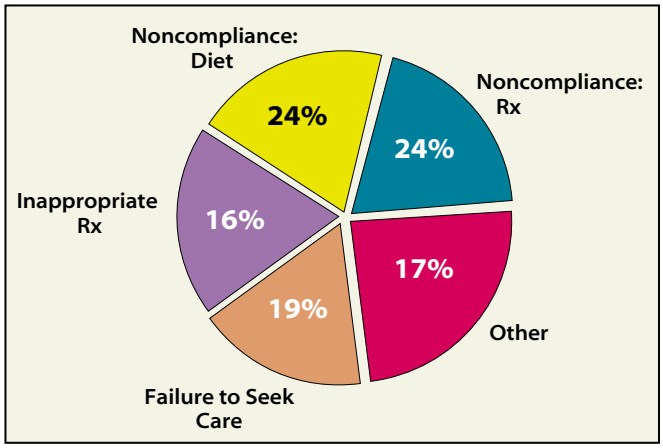


Figure 4. *Causes of hospital readmission for congestive heart failure. Figure provided by Scios, Inc.*



have developed a classification system that focuses on the stages of development of heart failure (Table 3).

Pathophysiology of Congestive Heart Failure

The human body has a series of neurohumoral systems that have evolved to assure hemodynamic stability and salt/water hemostasis. Organ perfusion and cellular metabolism can be maintained in a wide range of environments and survive many of the injuries sustained during hunting or fighting. These mechanisms include the following.

Adrenergic Nervous System

When a fall in cardiac output occurs, baroreceptors in the carotid sinus and aortic arch transit afferent signals via the autonomic nervous system. In response there is increased activity in sympathetic efferent pathways. Heart rate and the force of ventricular contraction are increased by activation of β-receptors. An increase in peripheral vasoconstriction is affected via α-receptor activation. From an evolutionary/survival perspective, this nervous system feedback loop response allows for rapid response to loss of circulatory volume from injury-related bleeding, water deprivation, or dehydration from diarrhea or vomiting. If, however, sympathetic nervous system stimulation via these pathways continues unabated for long periods of time, "downregulation" of the β-adrenergic receptors occurs. "Downregulation" refers to a decrease in sensitivity of the myocardium to the circulating catecholamines released by sympathetic nervous system activation. The result is a reduced inotropic response.

Long-term, catecholamine-mediated increases in ventricular contractility can be harmful because such stimulation may increase the ischemia and necrosis of myocardium served by occluded coronary vessels. As chronic excess of catecholamines increases the strain of ischemic myocardial tissue, fibroblasts migrate into the tissue, depositing collagen and producing myocyte hypertrophy. Fibrosis of the atria and ventricles causes a fall in the LV ejection fraction and eventually symptoms of heart failure. This process is referred to as "cardiac remodeling"; over time, "remodeling" can cause the left ventricle to become thick and stiff or, if accompanied by widespread myonecrosis, distended and unable to contract effectively.

Renin–Angiotensin–Aldosterone System

The renin–angiotensin–aldosterone system (RAAS) is critical to regulation of salt and water homeostasis by affecting renal perfusion and renal tubular sodium reabsorption. In healthy individuals, the RAAS allows the body to regulate renal blood flow during changes in body position and varying degrees of muscle exertion. Renin is produced and released from the juxtaglomerular cells lining the distal tubules in response to a decrease in renal artery perfusion. Renin converts angiotensinogen produced in the liver to angiotensin I. Angiotensin I is then converted to angiotension II by angiotensin–converting enzyme (ACE). Angiotensin II causes constriction of renal and systemic arterioles and reabsorption of sodium in renal tubules. Angiotensin II also stimulates the adrenal cortex to secrete aldosterone. Aldosterone promotes the reabsorption of sodium in exchange for potassium in the renal tubular system, in the colon, and in salivary and sweat glands.[3]

The consequence of myocardial pump dysfunction, which may

Table 2
New York Heart Association Functional Classification

Functional Class	Patient Limitations
Class I	• None • Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain • Often were previously symptomatic but are now in a well-compensated state
Class II	• Slight • Patient is comfortable at rest • Ordinary physical activity results in fatigue, shortness of breath, palpitations, or angina
Class III	• Marked • Patient is comfortable at rest • Less than ordinary activity leads to symptoms
Class IV	• Severe • Inability to carry on physical activity without discomfort • Patient is symptomatic at rest • Any physical activity increases symptoms and discomfort

Data from The Criteria Committee of the New York Heart Association. *Diseases of the Heart and Blood Vessels: Nomenclature and Criteria for Diagnosis*. 6th Edition. Boston, MA: Little Brown, 1964.

Table 3
American Heart Association and American College of Cardiology Stages of Heart Failure

Stage	Description
A	Patients at high risk of developing heart failure as a result of the presence of conditions that are strongly associated with the development of heart failure. These patients do not have any identified structural or functional abnormalities of the pericardium, myocardium, or cardiac valves and have never shown signs or symptoms of heart failure.
B	Patients who have developed structural heart disease that is strongly associated with the development of heart failure but who have never shown signs or symptoms of heart failure.
C	Patients who have current or prior systems of heart failure associated with underlying structural heart disease.
D	Patients with advanced structural heart disease and marked symptoms of heart failure at rest despite maximal medical therapy and who require specialized interventions.

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occur during acute myocardial ischemia, is a decrease in cardiac output and organ perfusion. The RAAS system becomes activated and the intravascular volume is expanded as a means of maintaining cardiac output and therefore organ perfusion.

Natriuretic Peptides

The RAAS system is counterbalanced by the release of natriuretic peptides from atrial and ventricular tissue. These peptides are released in response to volume expansion and rising pressure within the cardiac chambers. The natriuretic peptides produce arterial and venous vasodilation as well as diuresis as a result of inhibition of sodium reabsorption. The result is a drop in ventricular filling pressures and more effective cardiac function for patients experiencing ADHF.

During periods of acute decompensation, increased stretch of myocardial tissue stimulates the release of stored endogenous natriuretic peptide and the production of additional peptide. The quantity of endogenous natriuretic peptides released is not sufficient in ADHF to counteract the effects of the RAAS fully. Therefore, infusion of exogenous natriuretic peptide, commercially available as nesiritide (Scios Inc., Sunnyvale, CA), can be used to overcome fluid retention and vasoconstriction encouraged by the RAAS.

Endothelium Receptor System

Endothelin is another substance produced by the vascular endothelium, which contributes to the regulation of vascular tone and peripheral vascular resistance. Endothelium-regulated contraction of vascular smooth muscle causes vasoconstriction, especially in the kidney, where reduced blood flow reduces urine production. Research into the role of other neurohumoral factors produced by various tissues in the body (tissue necrosis factor, for example) in response to changes in circulatory volume, vascular pressure, water/salt homeostasis, and inflammation or organ ischemia is likely to result in the development of future pharmacologic breakthroughs.

Identifying the Patient with Acutely Decompensated Heart Failure in the Emergency Department

Patients who present to the ED with complaints of shortness of breath, dyspnea on exertion, increasing lower extremity edema, and/or worsening fatigue should have heart failure included in the differential diagnosis. Patients experiencing symptoms consistent with cardiac ischemia, hypoxia, potentially lethal arrhythmias, marked hypertension, or hypotension should be immediately triaged to a critical care area. Within the ED, ADHF patients are at high risk for sudden deterioration or death.[4] Monitoring of vital signs and oxygen saturation is essential while additional information is obtained about the patient's past medical history, current symptoms, and medications.

Elements of the ED evaluation of patients with ADHF should include the patient's history, an examination, an electrocardiogram, laboratory tests, and a chest x-ray if pulmonary findings or symptoms are present. A B-type natriuretic peptide (BNP) level may be helpful if the patients' respiratory symptoms are also consistent with pneumonia, bronchospastic disease, or an acute exacerbation of chronic obstructive pulmonary disease. The value of BNP levels in narrowing the differential diagnosis will be discussed in the article by Dr. Maisel in this supplement.

Managing the Patient with Acutely Decompensated Heart Failure in the Emergency Department

Until recently, the traditional management of ADHF involved oxygen, morphine, a loop diuretic, a vasodilator such as nitroglycerin, and inotropic agents (dobutamine, dopamine, and milrinone, for example) if the patient was hypotensive. If the cause of the decompensation can be identified, cardiac ischemia for example, treatment directed at that cause has been and remains important. The approval of nesiritide by the U.S. Food and Drug Administration in 2001 has stimulated the development of revisions in strategies for the ED treatment of ADHF patients.[5] According to results from recently published studies, the early use of nesiritide, along with topical nitroglycerin and a loop diuretic, may lead to more rapid resolution of an ADHF patient's acute symptoms and hemodynamic function.[6] It is hoped that the addition of nesiritide to the medications available to treat ADHF will lead to a decrease in the number of patients requiring intensive care unit hospitalization, a decrease in the number of patients requiring intubation for ADHF, and possibly more patients being discharged from the ED observation unit of a hospital.

Other changes in treatment that should be considered, based on recently published studies, include tighter restrictions on the use of inotropic agents. One study has noted an increase in the occurrence of ventricular arrhythmias in ADHF patients receiving dobutamine versus nesiritide treatment.[7] Another study has shown that short-term intravenous milrinone treatment for ADHF is of no benefit.[8] In the past, β-blocker therapy was discouraged for heart failure patients. New evidence suggests that β-blocker use for heart failure patients may reduce morbidity and mortality in NYHA class II–IV patients.[9] When patients on β-blockers present to the ED with ADHF, β-blocker drugs should be continued in the absence of a contraindication. The patient may receive nesiritide during the infusion. ACE inhibitor drugs may also be continued in patients receiving nesiritide if not contraindicated.

In summary, the management of ADHF in the ED has been expanded significantly in the past year. Better patient outcomes can be expected if physicians and nurses involved in the care of ADHF take the time to develop treatment protocols for ED and subsequent management of these patients. Once again the scope of emergency medical practice has been expanded through the research and development of new pharmaceutical products. The following four articles in this supplement will provide further information about

Main Points

- Congestive heart failure (CHF) as the primary discharge diagnosis increased by more than 150% between 1979 and 1999, perhaps because more Americans are surviving diseases such as coronary artery disease, cancer, and severe infections.
- CHF may be a complicating factor in patients with more than one coexisting disease process who present at the emergency department with symptoms unrelated to CHF.
- Long-term catecholamine-mediated increases in ventricular contractility can be harmful because such stimulation may increase the ischemia and necrosis of myocardium served by occluded coronary vessels.
- The renin-angiotensin-aldosterone system (RAAS) is critical to regulation of salt and water homeostasis by affecting renal perfusion and renal tubular sodium reabsorption.
- RAAS is counterbalanced by the release of natriuretic peptides from atrial and ventricular tissue in response to volume expansion and rising pressure within the cardiac chambers; the natriuretic peptides produce arterial and venous vasodilation as well as diuresis as a result of inhibition of sodium reabsorption.
- Infusion of exogenous natriuretic peptide, commercially available as nesiritide, can be used to overcome fluid retention and vasoconstriction encouraged by the RAAS.
- Patients who present with complaints of shortness of breath, dyspnea on exertion, increasing lower extremity edema, and/or worsening fatigue should have heart failure included in the differential diagnosis.
- Patients experiencing symptoms consistent with cardiac ischemia, hypoxia, potentially lethal arrhythmias, marked hypertension, or hypotension should be immediately triaged to a critical care area.
- Early use of nesiritide, along with topical nitroglycerin and a loop diuretic, may lead to more rapid resolution of an ADHF patient's acute symptoms and hemodynamic function.

developments in the management of ADHF. ■

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