Outpatient Emergencies

Acute Heart Failure

Malgorzata Mysliwiec, MD, Raphael E. Bonita, MD, ScM*

KEYWORDS

• Acute heart failure • Cardiomyopathy • BNP • Heart failure hospitalization
• Volume overload

KEY POINTS

- Heart failure is a growing health care crisis given the aging population.
- Volume overload manifested by symptoms of congestion is the most common clinical presentation of acute heart failure.
- Consider a broad differential diagnosis when evaluating patients with shortness of breath.
- Treatment of acute heart failure must address precipitating factors, identification of etiology, and should be tailored to the degree of volume overload and adequacy of end-organ perfusion.

INTRODUCTION AND DEFINITIONS

Heart failure is a major health care problem in the United States and internationally. For Americans, the lifetime risk of developing heart failure is 20% after the age of 40 years.[1] The prevalence of heart failure has been well documented to increase with age, and is higher in men and African American individuals.[2] The diagnosis of heart failure is associated with a significant degree of overall morbidity, recurrent hospitalizations leading to tremendous health care expenditures, and readmissions, and is marked with a strikingly high rate of mortality estimated to be 50% at 5 years.[3] Heart failure is a complex syndrome in which patients manifest symptoms from either impaired contractility of the heart to eject blood forward to vital tissues or in the setting of normal cardiac function at the expense of elevated filling pressures. Symptomatic heart failure results from volume overload due to reduced left ventricular systolic function or secondary to preserved cardiac contractility with impaired relaxation and increased diastolic stiffness. The left ventricular ejection fraction (EF) is used to classify the type of heart failure. According to the 2013 American College of Cardiology (ACC)/American Heart Association

Disclosure Statement: Authors have nothing to disclose.
Department of Medicine, Jefferson Heart Institute, Sidney Kimmel Medical College of Thomas Jefferson University, 925 Chestnut Street, Suite 323A, Philadelphia, PA 19107, USA
* Corresponding author.
E-mail address: Raphael.Bonita@jefferson.edu

Med Clin N Am ■ (2017) ■–■
http://dx.doi.org/10.1016/j.mcna.2016.12.010 **medical.theclinics.com**

(AHA) Heart Failure Guideline, heart failure with reduced EF (HFrEF) is defined by an EF $\leq$40% with heart failure with preserved EF (HFpEF) defined by an EF $\geq$50%.[4]

PATHOPHYSIOLOGY

The pathophysiology of heart failure is a highly complex process that can involve a multitude of factors. Some are extrinsic to the myocardium, others result from cardiac structural abnormalities that activate well-defined neurohormonal pathways in the circulatory system that become dysregulated, culminating in reduced pump function or a stiffened left ventricle with impaired relaxation[5] (**Fig. 1**). The neurohormonal model of renin-angiotensin-aldosterone and sympathetic nervous system activation of heart failure play a paramount role in the progressive cardiac remodeling that occurs with heart failure and is the target of current guideline-directed medical therapy (ie, angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers, aldosterone antagonists, and beta-blockade, respectively).

The natriuretic peptides (NPs) are a well-known family of hormones that play a major role in the body's response to fluid and sodium retention. Atrial natriuretic peptide (ANP) and B-type (BNP) are secreted from atrial and ventricular tissue, respectively, in response to stretch secondary to volume or pressure overload.[6] The mechanisms of action of these hormones include natriuresis, diuresis, and vasodilation via activation of guanylyl cyclase causing increase in intracellular cyclic guanosine monophosphate.[7] NPs are degraded and cleared from the circulation by the neutral endopeptidase neprilysin.[8] Neprilysin inhibition therefore prevents the breakdown of the NPs, thereby promoting a favorable hemodynamic response of vasodilatation and maintenance of fluid homeostasis. Entresto, a combination drug composed of the neprilysin inhibitor, sacubitril, and angiotensin receptor blocker, valsartan, were

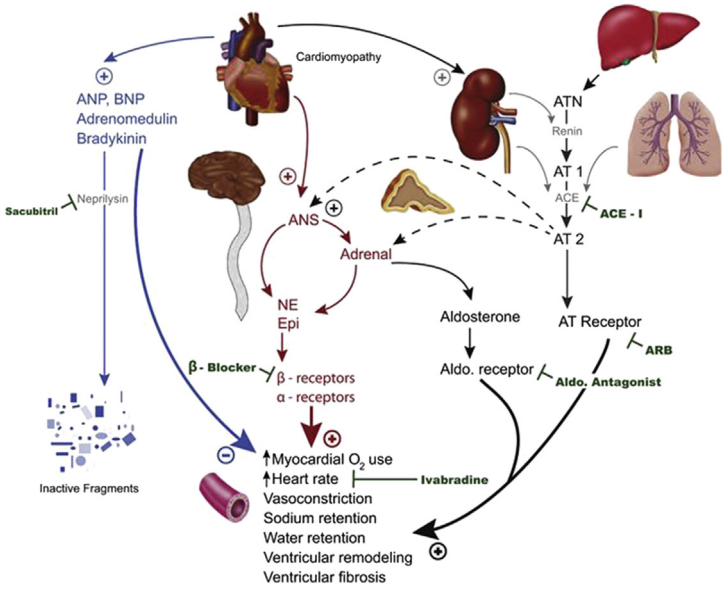

Fig. 1. Major neurohormonal pathways in heart failure and corresponding treatment targets. ANS, autonomic nervous system; AT, angiotensin 1; ATN, angiotensinogen; Epi, epinephrine; NE, norepinephrine. (*From* Gordin J, Fonarow G. New medications for heart failure. Trends Cardiovasc Med 2016;26:489; with permission.)