What If We Could Prevent Heart Failure?*

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At 40 years of age, American men and women have a 1 in 5 lifetime risk of the development of heart failure (HF); by the latest estimates, there are 5,700,000 people in the United States who already have a diagnosis of the disease. Moreover, by the year 2030, it is predicted that an additional 3 million Americans will have HF, representing an astounding 25% increase from 2010. Many of these individuals will be older than 65 years of age (1). Clearly, there is an urgent need to identify patients at risk of the development of HF and to initiate effective preventive measures.

What can be done to prevent HF and the resulting personal and societal costs? A first step toward recognizing the importance of risk factors leading to HF came with a shift in classification of HF. Instead of focusing solely on the time-honored New York Heart Association functional classification scheme—a useful method to quantify the degree of functional limitation of the patient with established HF—the American Heart Association (AHA) and American College of Cardiology (ACC) endorsed the use of a staging system focused on both pathophysiology and symptoms (2). The staging system and recommended treatment according to stage are presented in Figure 1 (3). Notably, 2 of the 4 categories, stages A and B, identify patients who are asymptomatic, in an effort to emphasize the need to intervene with proven therapies even before patients become symptomatic with more advanced stages of HF. Stage A represents patients with risk factors for HF, including traditional risk factors for coronary artery disease (CAD) and CAD itself. Stage B represents patients with structural heart disease, including left ventricular hypertrophy or systolic dysfunction, but who remain without HF symptoms. It is still a matter of debate whether asymptomatic diastolic dysfunction is stage A or B. In any case, according to a community-based cohort in Olmsted County, Minnesota, ~56% of adults older than 45 years of age were classified as stage A (22%) or B (34%). Thus, HF was likely to develop in at least half of this randomly selected Olmsted County population and could be identified before the onset of HF symptoms by medical record abstraction, physical examination, self-administered Goldman SAS questionnaire, echocardiography, and electrocardiography (4).

In this issue of JACC, Leening et al. (5) suggest an important additional component of the algorithm to predict HF by adding to what is already known about the relationship between coronary artery calcium (CAC) and HF (6,7). In their prospective, cohort study of 1,897 asymptomatic Dutch subjects older than 55 years of age, the authors found a graded association between the extent of CAC, as measured by electron-beam computed tomography, and the risk of incident HF, over a follow-up period of almost 7 years. Remarkably, when corrected for age, sex, and standard cardiovascular risk factors and censoring subjects with incident nonfatal coronary heart disease, a 3-fold increased risk of HF remained, suggesting an association between CAC and HF separate from overt coronary disease in this population (5). Importantly, the Rotterdam cohort was older, so that this study’s findings should not be generalized to a younger population or necessarily to one that is more racially

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Heart Failure

**Stage A**
- At high risk for HF but without structural heart disease or symptoms of HF.
- Patients with prior MI, LV remodeling including UH and low EF, asymptomatic valvular disease.

**Stage B**
- Structural heart disease but without signs or symptoms of HF.
- Patients with previous MI.

**Stage C**
- Structural heart disease with prior or current symptoms of HF.
- Patients with known structural heart disease and shortness of breath and fatigue, reduced exercise tolerance.

**Stage D**
- Refractory HF at rest despite maximal medical therapy (e.g., those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions).
- Patients who have marked symptoms at rest.

**Goals**
- All measures under stage A and B.
- Refractory measures under stage D.

**Drugs**
- ACEI or ARB in appropriate patients (see text) for vascular disease or diabetes.
- Angiotensin receptor blocker; EF = ejection fraction; FHx CM = family history of cardiomyopathy; HF = heart failure; LV = left ventricular; LVH = left ventricular hypertrophy; MI = myocardial infarction. Reprinted, with permission, from Hunt et al. (3).

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**Figure 1. Stages in the Development of Heart Failure/Recommended Therapy by Stage**

ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; EF = ejection fraction; FHx CM = family history of cardiomyopathy; HF = heart failure; LV = left ventricular; LVH = left ventricular hypertrophy; MI = myocardial infarction. Reprinted, with permission, from Hunt et al. (3).

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**At Risk for Heart Failure**

- At high risk for HF but without structural heart disease or symptoms of HF.
- Patients with hypertension, atherosclerotic disease, diabetes, obesity, metabolic syndrome, or Family history of cardiomyopathy.

**Structural heart disease**

- LV angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker.

**Development of symptoms of HF**

- HF therapy.

**Refractory symptoms of HF at rest**

- Patients with severe structural heart disease and shortness of breath and fatigue, reduced exercise tolerance.

**Therapy**

- All measures under stage A and B.
- Refractory measures under stage D.

**Drugs**

- ACEI or ARB in appropriate patients (see text) for vascular disease or diabetes.

**Devices in Selected Patients**

- Implantable defibrillators.

**Therapy**

- All measures under stages A and B.
- Dietary salt restrictions.

**Drugs for Routine Use**

- Diuretics for fluid retention.
- ACEI.
- Beta-blockers.
- Drugs in Selected Patients.
- Aldosterone antagonist.
- ARBs.
- Digoxin.
- Hydralazine/nitrates.

**Devices in Selected Patients**

- Biventricular pacing.
- Implantable defibrillators.

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The larger question to be answered in future studies is how do we lessen this risk, above and beyond treatment of standard cardiovascular risk factors? Will our interventions depend on the type of HF (systolic or diastolic) that is involved? Similar to the emerging story with biomarkers, where natriuretic peptides have shown incremental value in predicting the risk of incident HF (17), we need not only an accurate model for prediction, but also proven methods to mitigate the hazard, with a widespread application of prevention measures to our diverse U.S. population.

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REFERENCES


