Heart Failure (HF) is a major public health problem, with a prevalence of more than 5.8 million in the United States and more than 23 million worldwide. In 1997, HF was singled out as an emerging epidemic. An epidemic can reflect increased incidence, increased survival leading to increased prevalence, or both factors combined. Delineating the respective responsibility of each of these factors is essential to understand the determinants of the HF epidemic. The conceptual framework that guides the investigation is illustrated schematically in Figure 1. As shown, progress in the primary prevention of HF would lead to decreasing incidence of the disease while improvement in medical care would result in improved survival, in turn increasing the prevalence of HF. Both incidence and survival in turn play a major role in the genesis of the burden of hospitalization among patients living with HF. An in-depth understanding of the data relevant to this conceptual framework is required to understand the HF epidemic and design policies and strategies to prevent and manage HF. This review uses this conceptual framework to discuss incidence, mortality, and hospitalizations in HF. To identify relevant studies, the MEDLINE database was searched for publications with the subject headings “heart failure, epidemiology prevalence, incidence, trends” between 2005 and present. This review focuses on publications relevant to understanding the HF epidemic and designing policies and strategies to prevent and manage HF.
to epidemiology and population sciences after reviewing the abstracts for relevance to these topics.

Definition and Classifications

Definition

In the American Heart Association (AHA)/American College of Cardiology guidelines, HF is defined as “a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill or eject blood.” The guidelines underscore that “it is largely a clinical diagnosis that is based on a careful history and physical examination.” As HF is a syndrome and not a disease, its diagnosis relies on a clinical examination and can be challenging. To assess the burden of HF in populations and study its epidemiology, standardized criteria that can be used on a large scale for ascertainment from medical records are needed.

Standardized Criteria for HF Diagnosis

Several criteria have been proposed to diagnose HF (Table 1). These include in particular the Framingham criteria,7 the Boston criteria,8 the Gothenburg criteria,9 and the European Society of Cardiology criteria.7 All rely on similar indicators of symptoms and elevated filling pressures and combine data from the medical history, physical examination, and chest radiograph.

The European Society of Cardiology criteria7 require objective evidence of cardiac dysfunction. For population sciences studies, this implies that, to apply these criteria, cardiac function must be uniformly evaluated by appropriate tests. This is not always the case in practice.8–12

When the Boston and Framingham criteria were compared against the masked assessment of a cardiologist,13 their sensitivity was excellent at 100%. The specificity of the Framingham criteria and their positive predictive value were lower than those of the Boston score for definite HF; but it provided greater sensitivity to diagnose possible HF. Altogether, 5 of the 6 scores studied by Mosterd et al13 had a similar performance for the detection of HF, but the sample size was small thereby limiting the ability to detect differences across criteria. The Boston criteria have been recommended compared with other diagnostic criteria in older adults because of their construct validity and improved prediction of adverse outcomes.14

The Cardiovascular Health Study criteria rely on a panel of physicians that assign a diagnosis of HF by reviewing data on history, physical examination, chest radiographs, and medications. The comparison of the Framingham criteria to the Cardiovascular Health Study criteria yielded similar results.15 As the Framingham criteria offer good performance and are unaffected by time and use of diagnostic tests, they are well suited for studies of secular trends. Clinical cases of HF not meeting validation criteria are also important to capture in populations studies as they are captured in Vital Statistics and thus contribute to the epidemic and the use of healthcare resources.

Acute Decompensated HF

HF is a chronic disease characterized by acute exacerbation. Acute decompensated HF has been defined as “gradual or rapid change in heart failure signs and symptoms resulting in a need for urgent therapy”.16 This definition comprises 3 clinical situations: worsening chronic HF, new onset HF, and advanced HF. As acute decompensated HF is treated in the hospital, it constitutes one cause among several causes of hospitalization in patients living with HF. Identifying acute decompensated HF is crucial to accurately measure the burden of hospitalizations truly related to HF versus those related to comorbidity. Studies that rely on hospital dismissal codes may overestimate the true burden of acute decompensated HF by “counting” all cases ever diagnosed as HF.

In this context, 1 important question is whether existing criteria can accurately identify acute decompensated HF. The Atherosclerosis Risk in Communities (ARIC) study developed a classification, relying on manual adjudication to identify acute decompensated HF and compared it with other HF classifications, including the Framingham criteria, the Boston criteria, and the Gothenburg criteria (Table 1). The performance of these comparison criteria to the ARIC approach to identify acute decompensated HF was quite variable, underscoring the importance of considering which set of criteria is applied to validate HF.17 Further work conducted in the ARIC study examined the accuracy of an automated algorithm for the classification of acute decompensated HF.18 Compared with a physician reviewer panel, the automated algorithm was more efficient and less costly but its accuracy was modest. These results delineate a domain where more work is urgently needed because the ability to identify acute decompensated HF accurately is critical to comprehend the burden of HF in populations fully.

Systolic and Diastolic HF

Further classification of HF requires knowledge of the parameters of left ventricular function. The left ventricular ejection fraction (EF) enables classifying HF as preserved or reduced EF (Figure 2).19 Different thresholds for EF have been
The threshold of 55% was recommended in the American Society of Echocardiography guidelines. The Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF) registry used 40% as the cut point, as did the Acute Decompensated Heart Failure National Registry (ADHERE) database. The AHA and American College of Cardiology guidelines recommend 50% as a cutoff, which is used in the Framingham Heart Study and Olmsted County Study. The variations in threshold notwithstanding, EF is preserved in approximately half of HF cases in the community. To further classify subjects with HF and preserved EF, several criteria have been proposed, relying on the direct assessment of diastolic function which can be achieved with catheterization or Doppler echocardiography. Invasive measurements with conductance catheters have historically been considered the gold standard to measure filling pressures. However, it carries risks inherent to invasive studies, is seldom used in practice, and is not feasible for population studies. While MRI is an excellent tool to assess cardiac volumes and mass and is gaining more ground in the evaluation of HF, its use to evaluate diastolic function is presently not established. Echocardiography-Doppler is thus the approach of choice to assess diastolic function in routine practice.

Recommendation, all arbitrary in nature and derived from imaging studies with intrinsic variability. The threshold of 55% was recommended in the American Society of Echocardiography guidelines. The Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF) registry used 40% as the cut point, as did the Acute Decompensated Heart Failure National Registry (ADHERE) database. The AHA and American College of Cardiology guidelines recommend 50% as a cutoff, which is used in the Framingham Heart Study and Olmsted County Study. The variations in threshold notwithstanding, EF is preserved in approximately half of HF cases in the community. To further classify subjects with HF and preserved EF, several criteria have been proposed, relying on the direct assessment of diastolic function which can be achieved with catheterization or Doppler echocardiography. Invasive measurements with conductance catheters have historically been considered the gold standard to measure filling pressures. However, it carries risks inherent to invasive studies, is seldom used in practice, and is not feasible for population studies. While MRI is an excellent tool to assess cardiac volumes and mass and is gaining more ground in the evaluation of HF, its use to evaluate diastolic function is presently not established. Echocardiography-Doppler is thus the approach of choice to assess diastolic function in routine practice.

Table 1. Heart Failure Diagnostic Criteria

<table>
<thead>
<tr>
<th>Framingham</th>
<th>Boston</th>
<th>European Society of Cardiology</th>
<th>Gothenburg Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Major criteria</strong></td>
<td>Category I: History</td>
<td>1. Symptoms of heart failure (at rest or during exercise)</td>
<td>Cardiac score</td>
</tr>
<tr>
<td>Paroxysmal nocturnal dyspnea or orthopnea</td>
<td>Rest dyspnea (4 pts)</td>
<td>History of heart disease (1–2 pts)</td>
<td>Self-report</td>
</tr>
<tr>
<td>Neck vein distension</td>
<td>Paroxysmal nocturnal dyspnea (3 pts)</td>
<td>Angina (1–2 pts)</td>
<td>Self-report</td>
</tr>
<tr>
<td>Rales</td>
<td>Dyspnea on walking on level (2 pts)</td>
<td>Edema (1 pt)</td>
<td>Self-report</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>Dyspnea on climbing (1 pt)</td>
<td>Nocturnal dyspnea (1 pt)</td>
<td>Self-report</td>
</tr>
<tr>
<td>Acute pulmonary edema</td>
<td>Category II: Physical examination</td>
<td>2. Objective evidence of cardiac dysfunction (at rest)</td>
<td></td>
</tr>
<tr>
<td>S3 gallop</td>
<td>Heart rate abnormality (1–2 pts)</td>
<td>Rales (1 pt)</td>
<td>Physical examination</td>
</tr>
<tr>
<td>Increased venous pressure ≥16 cm water</td>
<td>Jugular venous pressure elevation (1–2 pts)</td>
<td>Atrial fibrillation (1 pt)</td>
<td>ECG</td>
</tr>
<tr>
<td>Circ. time ≥25 s</td>
<td>Lung crackles (1–2 pts)</td>
<td>Pulmonary score</td>
<td></td>
</tr>
<tr>
<td>Hepatoglycogly reflux</td>
<td>Wheezing (3 pts)</td>
<td>History of chronic bronchitis/asthma (1–2 pts)</td>
<td>Self-report</td>
</tr>
<tr>
<td><strong>Minor criteria</strong></td>
<td>Category III: Chest radiography</td>
<td>Criteria 1 and 2 should be fulfilled in all cases</td>
<td></td>
</tr>
<tr>
<td>Ankle edema</td>
<td>Third heart sound (3 pts)</td>
<td>Cough, phlegm, or wheezing (1 pt)</td>
<td>Self-report</td>
</tr>
<tr>
<td>Night cough</td>
<td></td>
<td>Rhonchi (2 pts)</td>
<td>Physical examination</td>
</tr>
<tr>
<td>Dyspnea on exertion</td>
<td></td>
<td>Cardiac and pulmonary score are calculated and used to differentiate cardiac from pulmonary dyspnea</td>
<td></td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>Alveolar pulmonary edema (4 pts)</td>
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<tr>
<td>Pleural effusion</td>
<td>Interstitial pulmonary edema (3 pts)</td>
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<tr>
<td>Vital capacity decreased 1/3 from maximum</td>
<td>Bilateral pleural effusions (3 pts)</td>
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<tr>
<td>Tachycardia rate of ≥120/min</td>
<td>Cardiothoracic ratio ≥0.50 (3 pts)</td>
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<tr>
<td><strong>Major or minor criterion</strong></td>
<td>Upper zone flow redistribution (2 pts)</td>
<td></td>
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<tr>
<td>Weight loss ≥4.5 kg in 5 d in response to treatment</td>
<td>Definite heart failure 8–12 pts, possible 5–7 pts, unlikely ≤4 pts</td>
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<td></td>
</tr>
</tbody>
</table>

Circ. indicates circulation; and pts, points.

Heart failure present with 2 major or 1 major and 2 minor criteria

The threshold of 55% was recommended in the American Society of Echocardiography guidelines. The Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF) registry used 40% as the cut point, as did the Acute Decompensated Heart Failure National Registry (ADHERE) database. The AHA and American College of Cardiology guidelines recommend 50% as a cutoff, which is used in the Framingham Heart Study and Olmsted County Study. The variations in threshold notwithstanding, EF is preserved in approximately half of HF cases in the community. To further classify subjects with HF and preserved EF, several criteria have been proposed, relying on the direct assessment of diastolic function which can be achieved with catheterization or Doppler echocardiography. Invasive measurements with conductance catheters have historically been considered the gold standard to measure filling pressures. However, it carries risks inherent to invasive studies, is seldom used in practice, and is not feasible for population studies. While MRI is an excellent tool to assess cardiac volumes and mass and is gaining more ground in the evaluation of HF, its use to evaluate diastolic function is presently not established. Echocardiography-Doppler is thus the approach of choice to assess diastolic function in routine practice.

Echocardiography-Doppler examination is indicated for the evaluation of HF and categorized as a Class I indication (conditions for which there is evidence and/or general agreement...
that a given procedure or treatment is beneficial…”) in the HF guidelines.2,3 Furthermore, left ventricular function assessment is a core performance measure for HF under the Joint Commission on Accreditation of Health Care Organizations (JCAHO).4 Early Doppler indices for diastolic function have been criticized for their complexity, dependency on loading conditions, and limited reproducibility.3,4,14 Tissue Doppler imaging, combined with mitral inflow measurements, now provides a feasible approach to assess filling pressures.3,4,5 Several algorithms have been proposed, including the algorithm proposed by the American Society of Echocardiography.5 The distinction between the existing sets of criteria should not obscure the fact that the basic measurements are similar such that it is important for the user to select the algorithm that he/she is most comfortable with based on the performance of the laboratory where the measurements are to be performed. Finally, as the field is rapidly evolving, it is likely that new measurements will enable characterizing left ventricular relaxation and be suitable for routine clinical use in the near future.6

Regardless of measurements issues, the mechanistic link between the elevation of filling pressures and the disease process is complex (Figure 2) and remains the subject of debate. Indeed, the causal role of intrinsic diastolic dysfunction (impaired relaxation and increased diastolic stiffness)3 was challenged against that of altered ventricular–vascular coupling.53–55 The altered ventricular–vascular coupling hypothesis needs to be considered cautiously as HF with normal EF is likely itself a heterogeneous entity within the HF syndrome.54,56 Furthermore, evaluating the putative role of other mechanisms requires complex measures that cannot be easily implemented in large-scale epidemiology studies. One additional matter that must be discussed is pulmonary hypertension, which is frequent and often severe in HF with preserved EF. Although pulmonary venous hypertension contributes to pulmonary arterial hypertension, it does not always fully account for its severity, suggesting that a superimposed component of pulmonary arterial hypertension also plays a role.57 Finally, these mechanisms are not exclusive of one another, and measuring diastolic function as can be done by echocardiography–Doppler is an important step toward a better understanding of the HF syndrome.58

Incidence and Prevalence of HF
In the United States, prevalent cases of HF now exceed 5.8 million and each year ≥550 000 new cases are diagnosed.59,60 Selected data on the incidence of HF are tabulated (Table 2) and organized according to the criteria used to ascertain HF. Several estimates are derived from hospital discharges, which are not always validated by standardized criteria, and shifts in hospital discharge diagnoses preferences after the introduction of the Diagnosis-Related Groups payment systems have been documented.62,63 For HF in particular, the potential for upcoding of discharge diagnoses due to reimbursement incentives is well known. Hospitalization statistics are event based, not person based and allow multiple hospitalizations for the same individual to be counted without distinguishing between first and subsequent admission such that incidence cannot be derived from such data. Thus, national statistics and claims data are not well suited to inform on the incidence of HF. Inpatient data may not capture all cases of HF because care is increasingly delivered in the outpatient setting.64 Studies using surveys of physicians or self-report are by design more inclusive in their ascertainment. They reported relatively broad ranges of prevalence without validation. When validation was performed, approaches have ranged from medical record review and adjudication as in the Cardiovascular Health Study to the use of criteria such as the Framingham, Boston, or European Society of Cardiology criteria.4,5,86 Using standardized criteria, the incidence of HF in an earlier study from Framingham was between 1.4 and 2.3 per 1000/y among persons aged 29 to 79 years.4 However, the size of the cohort inherently limits its power to analyze secular trends in this report. Among the studies of secular trends,6,66,67,70,72,73,87 few included outpatient data. Others used hospitalized cases without validation and are thus subject to secular changes in hospitalization practices and coding patterns, which likely confound time trends in incidence. It should not be surprising therefore that their results differ. Croft et al,67 comparing the rates of initial hospitalization for HF using Medicare hospital claims in 1986 and 1993, reported an increase in the initial hospitalization for HF, while acknowledging limitations related to the lack of validation and possible incomplete ascertainment of incidence. Data from the Henry Ford Health system, a managed care organization,71 indicated that the prevalence of HF was increasing over time but did not detect any secular change in incidence or mortality. In the Framingham Heart Study70 and the Olmsted County Study72 which include outpatient HF, the incidence of HF failure remained stable over time72 or even declined in women.70 It should be noted that, although the interpretation and informal comparison of trends across studies is appropriate, as adjustment approaches differ, the absolute numbers cannot be compared. Importantly, the trends noted among the elderly are different and data from the Kaiser Permanente system comparing the incidence of HF in 1970–1974 and 1990–1994 among persons aged ≥65 years indicated that the age-adjusted incidence increased by 14% over time and was greater for older persons and for men.71 The Framingham and Olmsted County studies also reported trends toward increasing HF incidence among older persons, which are concerning given the aging of the population.
Table 2. Selected Studies Reporting on the Incidence and Prevalence of Heart Failure

<table>
<thead>
<tr>
<th>Diagnostic Criteria</th>
<th>Author</th>
<th>Years</th>
<th>Incidence</th>
<th>Prevalence</th>
<th>Population Source</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nonvalidated criteria</strong></td>
<td></td>
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<tr>
<td>Self-report</td>
<td>Schocken et al61</td>
<td>1971–1975</td>
<td>...</td>
<td>1%–2%</td>
<td>NHANES I</td>
<td>At 10 y: 43%</td>
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<tr>
<td></td>
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<td></td>
<td>Ages 1–74 y, not adjusted</td>
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<td></td>
<td></td>
<td></td>
<td>Age ≥20 y</td>
</tr>
<tr>
<td>First hospitalization for heart failure</td>
<td>Croft et al63</td>
<td>1986</td>
<td>White: 22.4/1000 person-years</td>
<td>...</td>
<td>Medicare beneficiaries (age ≥65 y)</td>
<td>In hospital</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Black: 22.4/1000 person-years</td>
<td></td>
<td>Age adjusted</td>
<td>1986: White: 13%</td>
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<td>Black: 11%</td>
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<td>1993: White: 10%</td>
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<td></td>
<td>Black: 9%</td>
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</tr>
<tr>
<td>First hospitalization for heart failure</td>
<td>Jhund et al64</td>
<td>1986</td>
<td>Men: 1.2/1000 persons</td>
<td>...</td>
<td>Scotland</td>
<td>1-y age adjusted</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Women: 1.3/1000 persons</td>
<td></td>
<td>Age adjusted</td>
<td>1986: Men: 33%</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>Women: 31%</td>
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<td></td>
<td>2003: Men: 28%</td>
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<td></td>
<td></td>
<td></td>
<td>Women: 27%</td>
</tr>
<tr>
<td>Hospital discharge or death certificate with ICD code for HF</td>
<td>Loehr et al65</td>
<td>1987–2002</td>
<td>White women: 3.4/1000 person-years</td>
<td>...</td>
<td>Atherosclerosis Risk in Communities Study</td>
<td>Overall 1-y mortality: 22% (similar for blacks and whites)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>White men: 6.0/1000 person-years</td>
<td></td>
<td>Age adjusted</td>
<td>2003: Men: 28%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Black women: 8.1/1000 person-years</td>
<td></td>
<td></td>
<td>Women: 27%</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Black men: 9.1/1000 person-years</td>
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<tr>
<td>Hospital discharge diagnosis</td>
<td>Stewart et al66,67</td>
<td>1990–1996</td>
<td>Women: 1.3–1.9/1000 persons</td>
<td>...</td>
<td>Scotland</td>
<td>At 1 y</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Men: 1.3–2.2/1000 persons</td>
<td></td>
<td>All ages, not adjusted</td>
<td>1990: ~40%</td>
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<tr>
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<td>1996: ~36%</td>
</tr>
<tr>
<td>Administrative database: CMS</td>
<td>Curtis et al68</td>
<td>1994</td>
<td>32/1000 person-years</td>
<td>9%</td>
<td>Medicare beneficiaries (age ≥65 y)</td>
<td>1-y risk adjusted</td>
</tr>
<tr>
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<td></td>
<td>Age adjusted</td>
<td>1994: 29%</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td>2002: 28%</td>
</tr>
<tr>
<td>Administrative database</td>
<td>Yeung et al69</td>
<td>1997</td>
<td>4.5/1000 persons</td>
<td>...</td>
<td>Ontario, Canada</td>
<td>1-y risk adjusted</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Age and sex standardized</td>
<td>1997: Outpatients: 18%</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Inpatients: 36%</td>
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<td>2007: Outpatients: 16%</td>
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<td></td>
<td></td>
<td></td>
<td>Inpatients: 34%</td>
</tr>
<tr>
<td>Standardized criteria</td>
<td>Levy et al70</td>
<td>1950–1999</td>
<td>~5/1000 person-years</td>
<td>...</td>
<td>Framingham Heart Study</td>
<td>At 1 y age adjusted</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>All ages, age adjusted, mostly whites</td>
<td>1950–1969: Men: 30%</td>
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<td></td>
<td></td>
<td></td>
<td>Women: 28%</td>
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<td>1990–1999: Men: 28%</td>
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<td></td>
<td></td>
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<td></td>
<td>Women: 24%</td>
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</tbody>
</table>

(Continued)
Table 2. Continued

<table>
<thead>
<tr>
<th>Diagnostic Criteria</th>
<th>Author</th>
<th>Years</th>
<th>Incidence</th>
<th>Prevalence</th>
<th>Population Source</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Framingham criteria</td>
<td>Barker et al[7]</td>
<td>1970–1974</td>
<td>Women: 8.6/1000 person-years</td>
<td>...</td>
<td>Kaiser Permanente</td>
<td>1- year age-adjusted mortality</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Men: 11.7/1000 person-years</td>
<td></td>
<td>Age adjusted, mostly whites</td>
<td>1970–1974: Women: 27%</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>Men: 47%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Men: 12.7/1000 person-years</td>
<td></td>
<td></td>
<td>Women: 33%</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Women: 28%</td>
</tr>
<tr>
<td>Framingham criteria</td>
<td>Roger et al[72]</td>
<td>1979–2000</td>
<td>≈3/1000 persons</td>
<td>...</td>
<td>Olmsted County</td>
<td>At 1 y (75-year-olds)</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td>Age adjusted, mostly whites</td>
<td>1979–1984: Men: 30%</td>
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<td></td>
<td></td>
<td></td>
<td>Women: 20%</td>
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<td>1996–2000: Men: 21%</td>
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<td></td>
<td></td>
<td></td>
<td>Women: 17%</td>
</tr>
<tr>
<td>Framingham criteria</td>
<td>McCullough et al[7]</td>
<td>1989–1999</td>
<td>Women: 3.7–4.2/1000 persons</td>
<td>Women: 0.4%–1.4%</td>
<td>REACH Study</td>
<td>Per year: 17%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Men: 4.0–3.7/1000 persons</td>
<td>Men: 0.4%–1.5%</td>
<td>Henry Ford Health System</td>
<td>50% whites; age adjusted</td>
</tr>
<tr>
<td>Framingham criteria</td>
<td>Goldberg et al[74]</td>
<td>2000</td>
<td>≈2/1000 persons</td>
<td>...</td>
<td>Worcester, MA hospitals</td>
<td>Hospital case fatality rate: 5%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Not age adjusted</td>
<td></td>
</tr>
<tr>
<td>Boston criteria</td>
<td>Remes et al[75]</td>
<td>1986–1988</td>
<td>Women: 1.0/1000 persons</td>
<td>...</td>
<td>Eastern Finland</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Men: 4.0/1000 persons</td>
<td></td>
<td>In and out patient national registries</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Age adjusted</td>
<td></td>
</tr>
<tr>
<td>Boston criteria</td>
<td>Nielsen et al[76]</td>
<td>1993–1995</td>
<td>...</td>
<td>0.5%–12%</td>
<td>Denmark</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>General practice population Ages ≥50 y, not adjusted</td>
<td></td>
</tr>
<tr>
<td>European Society of</td>
<td>Bleumink et al[77]</td>
<td>1989–2000</td>
<td>Women: 12.5/1000 person-years</td>
<td>1998: 7%</td>
<td>The Rotterdam Study</td>
<td>At 1 y: 37%</td>
</tr>
<tr>
<td>Cardiology criteria</td>
<td></td>
<td></td>
<td>Men: 17.6/1000 person-years</td>
<td></td>
<td>Not age adjusted</td>
<td></td>
</tr>
<tr>
<td>European Society of</td>
<td>Cowie et al[78]</td>
<td>1995–1996</td>
<td>Women: 1.2/1000 persons/y</td>
<td>...</td>
<td>Geographically defined in United Kingdom</td>
<td></td>
</tr>
<tr>
<td>Cardiology criteria</td>
<td></td>
<td></td>
<td>Men: 1.4/1000 persons/y</td>
<td></td>
<td>In- and outpatient</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>All ages, not adjusted</td>
<td></td>
</tr>
<tr>
<td>European Society of</td>
<td>Davies et al[79]</td>
<td>1995–1999</td>
<td>...</td>
<td>2%–3%</td>
<td>UK population</td>
<td></td>
</tr>
<tr>
<td>Cardiology criteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Random sample</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ages ≥45 y, not adjusted</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular Health</td>
<td>Gottdiener et al[80]</td>
<td>1990–1996</td>
<td>Nonblack: 19/1000 person-years</td>
<td></td>
<td>Cardiovascular Health Study</td>
<td></td>
</tr>
<tr>
<td>Study criteria</td>
<td></td>
<td></td>
<td>Black: 19/1000 person-years</td>
<td></td>
<td>Age 65–100 y, age adjusted</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Women: 15/1000 person-years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Men: 26/1000 person-years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multi-Ethnic Study of</td>
<td>Bahrami et al[81]</td>
<td>Enrolled from 2000 to 2002; Median follow-up 4.0 y</td>
<td>Blacks: 4.6/1000 person-years</td>
<td>...</td>
<td>Multi-Ethnic Study of Atherosclerosis</td>
<td>Not age adjusted</td>
</tr>
</tbody>
</table>
The data discussed up to this point reflect largely the US experience. In Scotland, Stewart et al suggested that trends in HF hospitalization in the 1990s had “leveled off.” These results are limited by the lack of validation and sole use of inpatient data but prompt the question of whether the stabilization of the HF hospitalization rates could be offset by increasing outpatient care practice. Data on temporal trends in incidence of HF from Ontario 69 and Scotland 64 are informative in this regard as they indicate that the incidence of HF started to decline since the late 1990s. This finding is important, as it further highlights the fact that the continuing burden of HF hospitalizations reflects persisting difficulties in managing existing disease, rather than an increasing number of new cases developing HF.

Most of the aforementioned studies pertain to white subjects, and data on the burden of HF in diverse populations are scarce. In ARIC and in the Multi-Ethnic Study of Atherosclerosis (MESA), HF incidence was higher in blacks than in whites. In both studies, the difference between blacks and whites was attenuated after adjustment and overall the greater HF incidence in blacks was related to their greater burden of atherosclerotic risk factors as well as to socioeconomic status. 65, 61 This underscores the imperative for continued community surveillance of cardiovascular disease in diverse populations. 89, 90 Data on the incidence and prevalence of HF according to EF and how it may have changed over time are very limited. The available evidence suggests that the prevalence of HF with preserved EF increased over time. 34

Measures of lifetime risk are anchored in a robust methodological framework, provide more complete information than shorter term risk, and are useful to identify patients at risk and communicate with them for the purpose of risk modification. 91 For HF, the reported lifetime risk of developing the disease ranged from 20% to 33% in predominantly white cohorts. 92 Recently, lifetime risks for developing HF were reported among a diverse large group of 39,578 participants in several cohorts, including the Chicago Heart Association Detection Project in Industry, the ARIC study, and the Cardiovascular Health Study. 93 At age 45 years, lifetime risks for HF through age 75 or 95 years were 30% to 42% in white men, 20% to 29% in black men, 32% to 39% in white women, and 24% to 46% in black women. Higher blood pressure and body mass index at all ages in both blacks and whites led to higher lifetime risks.

In the Rotterdam Heart Study, the lifetime risk of HF at the age of 55 years was 33% for men and 29% for women. 77 These numbers are commensurate with the data from the United States.

In summary, the overall prevalence of HF ranges from 1% to 12% based on available data from the United States and Europe. The incidence of HF varies across studies largely reflecting differences in ascertainment and adjustment approaches. These methodological differences, however, do not affect the interpretation of secular trends in incidence where the focus is on the evolution over time. Temporal trends are congruent across studies and quite informative for the investigation of the HF epidemic because they indicate that the incidence of HF is stable or perhaps even decreasing over time. Available data indicate that lifetime risks are high regardless of sex, race, and geography, underscoring the importance of population-wide efforts to contain the burden of HF.

Mortality of HF

After the diagnosis of HF, survival estimates are 50% and 10% at 5 and 10 years, respectively, 94–96 and left ventricular dysfunction is associated with an increase in the risk of sudden death. 97 Improvement in the survival of hospitalized HF among the Scottish population was reported 64 with notable age and sex differences in the magnitude of the secular trends. These data may reflect in part the effectiveness of angiotensin-converting enzyme inhibitors. However, the median survival improved relatively modestly from 1.2 to 1.6 years such that, while the large sample size (66,547 patients) results in high statistical significance, the clinical significance of this improvement in survival is more modest. Furthermore, the analyses relied solely on hospitalized cases, not validated, such that the improvement in outcome may be confounded by trends in coding practice and shifting of hospitalization thresholds. Regardless, these data resonate with clinical trials that indicated that angiotensin-converting enzyme inhibitors, while associated with large reductions in the relative risk of mortality, resulted in more modest absolute event-rate difference. 89 Administrative data, however, convey somewhat of a different message. In the Henry Ford Health system, which include outpatient encounters, the median survival was 4.2 years without any discernible improvement over time. 73 Similar, among >2 million elderly Medicare beneficiaries, early- and long-term mortality remained quite high (115% at 30 days and 37% at 1 year). 68

These discrepancies in survival estimates underscore the challenges in investigating the HF epidemic and help delineate key requirements for such evaluation. This investigation should include all cases of HF in a geographically defined population and use standardized validation criteria to generate valid longitudinal trends. The analyses should examine trends in hospital admission as an additional outcome as high hospital admission rates after diagnosis provide insights into the outcome of HF, independently of disease severity, 99–103 and are an important component of its public health burden. Data from Framingham 70 and Olmsted County 72 underscored the persistently high mortality of HF in these populations, despite improvements over time; indeed, after age adjustment, estimated 5-year mortality rates were 59% in men and 45% in women during the time period 1990–1999 in Framingham and 50% in men and 46% in women during the time period 1990–2000 in Olmsted County. Improvements in survival were noted more specifically within an elderly population as shown by data from the Kaiser Permanente system. Indeed, during the 2 decades between the mid-1970s and mid-1990s after adjustment for age and comorbidities, survival after the diagnosis of HF improved by 33% in men and 24% in women. 71 Importantly, in the Kaiser Permanente study, improvement in survival was primarily associated with β-blocker treatment. Data from Ontario 66 and Scotland 64 also support the observation that although survival after HF diagnosis remains quite poor, improvements have been detected since the late 1990s. Altogether, these trends in mortality coincide temporally with major changes in the treatment of HF and thus suggest that HF treatment is effective in the community but that much
progress remains to be accomplished. As the proportion of HF with preserved EF for which there is no specific treatment is increasing over time, its prevalence will likely increase, underscoring the urgent need for new therapeutic approaches of this entity.24

The causes of death in HF can be challenging to ascertain. In the community, cardiovascular deaths are less frequent among subjects with preserved EF. Indeed, in Olmsted County, MN, among 1063 persons with HF, the leading cause of death in subjects with preserved EF was noncardiovascular (49%) versus coronary disease (43%) for subjects with reduced EF. The proportion of cardiovascular deaths decreased from 69% in 1979–1984 to 40% in 1997–2002 (P=0.007) among subjects with preserved EF contrasting with a modest change among those with reduced EF (77%–64%, P=0.08).104 The shift in the distribution of the causes of death toward less cardiovascular causes is congruent with the major burden of comorbid conditions in HF and is of crucial importance for the management of HF and the interpretation of its outcomes.

In summary, survival after the diagnosis of HF remains quite poor but has improved substantially over time. The results are consistent across studies and, combined with the aforementioned trends in incidence, indicated that the epidemic of HF is an epidemic of hospitalizations among survivors who now live longer with the disease.

Hospitalizations in HF

As the incidence of HF has remained stable during the past 2 decades although survival has improved,20,72,73 the HF epidemic is a large chronic disease epidemic reflecting an increase in the prevalence of HF in an aging population and the improved survival of patients with HF.73 HF is characterized by periodic exacerbations that require treatment intensification most often in the hospital and is the single most frequent cause of hospitalization in persons aged ≥65 years. Nearly 1 million hospitalizations for HF occur each year with rates of hospitalization continuing to rise. This trend, coupled with the forecasting of a major increase in the prevalence of HF by the AHA,105 underscores the persisting severity of the burden that HF creates on healthcare systems and the need for continued surveillance of HF trends to delineate strategies for management. Importantly, such strategies can be expected to change over time as the case mix of the disease evolves. Examples of the importance of such population surveillance can be found in several recent publications. Using data from the Centers for Medicare and Medicaid services (CMS) data, Chen et al106 reported an encouraging decline in admission rates for HF between 1998 and 2007. This decline seemed largely related to a reduction in the number of unique individuals hospitalized for HF. During the same time period, however CMS data indicate that readmission rates after an index admission for HF have remained unchanged or have even increased.107 A subsequent analysis of CMS data indicates that after an initial hospitalization, 25% of HF patients are readmitted within 30 days with 35% of readmissions also attributed to HF.108 Data from the Veteran’s Affairs Health Care System also support the notion that as mortality was decreasing, readmission rates have in fact increased over time.109 Taken collectively, these important reports suggest that that threshold for admitting patients with HF to the hospital might be evolving. However, once patients have been hospitalized with HF, their risk of readmission is not decreasing over time but they will be readmitted rather infrequently because of HF. Hospital readmissions are now a quality indicator under the Hospital Readmissions Reduction Program of the Patient Protection and Affordable Care Act with downward adjustment of Medicare payment for hospitals with excess 30-day readmission rates.110 This program has been controversial, specifically raising concerns for HF that the driver of readmission is not the disease per se but rather the associated comorbidity burden poorly addressed by disease-specific disease management programs. Hence, the need to inform policy by an understanding of the root causes of hospitalizations is urgent.111 Doing so effectively requires consideration of several methodological issues, particularly related to data sources.112 Administrative datasets provide extensive population coverage but hospital admission data are most often event based, counting multiple hospitalizations for the same individual. Diagnoses are not validated and discharge diagnoses choices are sensitive to changes in payment systems.82,83,89 Clinical data are lacking, which limits case mix adjustment. Clinical registries provide rich clinical information on HF and are critical to gain insight into real-life clinical practice. Several large-scale registries are specifically dedicated to HF. The OPTIMIZE-HF registry includes 259 hospitals that have enrolled >50,000 hospitalized patients with HF.114 Initially supported by industry, OPTIMIZE-HF is now integrated to the AHA Get with the Guidelines program, which includes 558 hospitals and >530,000 hospitalized patients with HF.114 The ADHERE, sponsored by industry (Scios Inc California), enrolled >150,000 patients from 300 community and academic centers to evaluate characteristics, management, and outcomes of patients hospitalized with acute decompensated HF.115 Participation to registries is voluntary, creating an unavoidable selection bias, and registries are seldom positioned to ascertain the incident status of HF such that the ensuing incidence prevalence bias also limits validity. Registries typically include only inpatient data on clinical presentation, care, and outcomes. These limitations are important to consider while interpreting registry data.

Data on the cause of hospitalizations among HF patients suggest that HF-specific hospitalizations may be noticeably less frequent than all-cause hospitalizations. This observation is critically important as intense treatment efforts (medication, device, and disease management based) are intrinsically disease centric and directed at reducing HF exacerbation. Thus, HF-specific hospitalizations are a key indicator of the effectiveness of HF-specific treatments but disease-specific interventions cannot be expected to reduce all hospitalizations appreciably among persons living with HF, given the high prevalence of comorbidity in these patients. National Hospital Discharge Survey data from 1979 to 2004 indicate that although HF was the first-listed diagnosis for 30% to 35% of these hospitalizations, the proportion with respiratory diseases and noncardiovascular, nonrespiratory diseases as the first-listed diagnoses increased over time.116

In the community of Olmsted County, among incident HF cases diagnosed between 1987 and 2006, hospitalizations were common after HF diagnosis, with 83% of the patients...
hospitalized at least once but the reason for hospitalization was HF in only 17% of hospitalizations, whereas 62% were attributed to noncardiovascular causes. Using the Nationwide Inpatient Sample, Bleker et al. reported on trends in HF hospitalizations between 2001 and 2009. Primary HF hospitalizations declined, but hospitalizations with a secondary diagnosis of HF remained stable.

In summary, these data underscore the major role of comorbidity in HF and that, to reduce the burden of hospitalizations in HF, strategies must consider both cardiac disease and noncardiac conditions. While initial hospitalizations are seemingly decreasing, readmissions after an initial hospitalization are not declining such that, with the increased survival of patients living with HF, the overall burden of hospitalizations in HF remains large.

Cause of HF: An Evolving Picture

Assigning a cause to HF should be envisioned while focusing on clinically ascertained risk factors and acknowledging that multiple causes for HF often coexist and interact in a given patient. From a public health and prevention perspective, the determination of the prevalence of each respective cause as ascertained clinically is important because of the public health implications. To this end, the prevalence of a given risk factor combined to the risk of HF that it confers enables computing the attributable risk of a given factor for HF. This in turn provides an indication of what proportion of the cases of HF would be avoided if the risk factor in questions was eliminated.

Such analyses also have mechanistic implications. For example, demonstrating an increase in the attributable risk of diabetes mellitus independently of clinical coronary disease would then prompt investigations about the mechanisms whereby diabetes mellitus leads to HF in the absence of overt coronary disease. Such mechanisms may include occult coronary disease, but within the appropriate analytic framework, this would be distinct from clinically established coronary disease. The importance of defining the respective contribution of these 2 entities contrasts with the lack of knowledge in this regard. Moreover, the reported data are conflicting and secular trends have infrequently been examined. Yet, the population burden of putative risk factors for HF is changing in the population, such that the attributable risk of these factors for HF may be evolving as well. Examples of the prevalence and attributable risk of selected factors are presented in Figures 3 and 4 using data from the Framingham and Olmsted County studies, selected as the similarities of the presentation of the results enabled creating these plots (Figures 3 and 4). These illustrate that, for example, for hypertension, although the prevalence is high exceeding 50% in all groups, the attributable risk is lower and varies across groups reflecting differences in the relative risk of HF associated with hypertension.

For coronary disease, estimates of the prevalence among patients with HF vary considerably across studies. Fox et al., using angiography, concluded that coronary disease was causal in 52% of new HF cases in patients aged <75 years in a geographically defined population and that clinical assessment without angiography underestimates the contribution of coronary disease to HF. However, few patients were aged >75 years and only 73% underwent angiography, reflecting substantial selection bias. Reviewing randomized trial data, Gheorghiade and Bonow concluded the prevalence of coronary disease in HF was >68%. However, important methodological considerations limit the inference that can be drawn from these data. Indeed, the limitations in external validity inherent to clinical trials may be even more apparent in HF trials, which typically include younger patients and more men than the general population of HF. Furthermore, entry criteria in HF trials are heterogeneous and seldom validated. Finally, HF trials often require systolic left ventricular dysfunction, thereby excluding a substantial proportion of HF cases. An observational report of patients with HF suggest that the prevalence of coronary disease in HF is >50% whereas a population-based study in England reported that coronary disease was the cause of HF in 36% of the cases. This is commensurate with what was noted among men in the Olmsted County study but higher than that reported in the Framingham Heart Study (Figure 3). These large discrepancies likely reflect differences in populations, study design, and ascertainment approaches. They also underscore our limited knowledge regarding the cause of HF, which hinders prevention. In the first National Health and Nutrition Examination Survey (NHANES I), coronary disease had the largest population attributable risk for HF at 62% compared with the other risk factors analyzed (hypertension, obesity, diabetes mellitus, and smoking). The attributable risk of hypertension was 10% and that of diabetes was 3% attributable to its low prevalence. This likely underestimates, as acknowledged by the authors, the role of diabetes mellitus that was ascertained by self-report among patients enrolled >20 years ago with the incidence of diabetes mellitus increasing over time. In the Cardiovascular

\[\text{AR} = \frac{\text{Prevalence of risk factor} \times \text{Relative risk}}{1} \]

Figure 3. Prevalence of risk factors in heart failure. CHD indicates coronary heart disease; and HTN, hypertension.

Figure 4. Attributable risk (AR) of select risk factors for heart failure. CHD indicates coronary heart disease; and HTN, hypertension.
Health Study, the attributable risk of coronary disease for HF was similar to that of hypertension, around 12%, with a notable attributable risk of 8% for diabetes mellitus.137 The Framingham Heart Study historically underscored a large contribution of hypertension to HF120,130–132 (Figures 3 and 4). Over time, however, it suggested a 41% increase in the prevalence of coronary disease and a 10% decrease in that of hypertension in HF.133 Whether the results of Framingham are generalizable to larger populations, thereby suggesting that the cause of HF shifted from hypertension to coronary disease remains to be determined, particularly given the unfavorable hypertension trends in the United States and in Olmsted County discussed below. To this end, when the contribution of coronary disease to HF and its hypothetical change over time is examined by analyzing population trends in coronary disease, the data are difficult to reconcile with the aforementioned hypothesis of an increasing contribution of coronary disease to HF. Secular trends in the incidence of myocardial infarction (MI) indicating that the epidemiology of MI is changing that, the burden of incident hospitalized MI, while displaced toward older age groups, is decreasing and that the severity of MI is decreasing.134–136 These findings indicate that the incidence of HF after MI can be expected to be declining over time. There have been few community-based or population investigations of the long-term trends in the incidence of HF after MI. Among residents of Olmsted County with an incident MI and no previous history of HF, a decline in the incidence of HF post-MI was observed and the relative risk of HF post-MI in 1994 versus 1979 was 0.72 (95% confidence interval, 0.55–0.93).27 However, data from the Framingham Heart Study pertaining to 676 participants who experienced a first MI between 1970 and 1999 indicate that the 30-day incidence of HF after MI rose from 10% in 1970–1979 to 23.1% in 1990–1999 as 30-day mortality after MI declined over the same period.137 According to the more recent data from the national Swedish hospital discharge and death registries, among 175 216 patients with a first MI between 1993 and 2004, decreasing trends in the incidence of HF post-MI were observed with a 4% per year decrease in the adjusted risk of HF.138 In the Worcester Heart Attack Study, the incidence of HF post-MI decreased over time between 1986 and 2005.139 The discrepancies across studies likely reflect the different periods under observation during a time of profound changes in the epidemiology of MI. This in turn underscores the importance of continued surveillance of HF post-MI and of the evolving causes of HF. Finally, while it is conceivable that more chronic forms of coronary disease could lead to HF without MI, the role of chronic coronary disease in the genesis of HF is not defined.

With regards to hypertension, conversely, unfavorable trends in awareness, treatment, and control of hypertension have been documented.140,141 Thus, coronary disease and hypertension trends in population studies both suggest that the attributable risk of hypertension for HF should remain high. To this end, in Olmsted County, there was no evidence for a temporal change until 2002 in the population attributable risk for HF of coronary disease, diabetes mellitus, and smoking. By contrast, the population attributable risk of hypertension increased from 15% (1979–1984) to 29% (1979–2002), and that of obesity from 8% (1979–1984) to 17% (1997–2002).121

Finally, the rising tide of diabetes mellitus142 and obesity143 raise the concern of an increasing role of these 2 entities in the genesis of HF. Notwithstanding uncertainties with regards to the exact cellular and molecular mechanisms by which obesity and diabetes mellitus impact both systolic and diastolic left ventricular function, there is mounting evidence for their causal link to HF independently of clinical coronary disease and hypertension.144–146 To this end, the population burden of HF attributable to obesity and diabetes mellitus was recently examined in the ARIC study.149,150 For obesity, while complete elimination of obesity/overweight could prevent almost one third (28%) of new HF cases, a more realistic 30% reduction in obesity/overweight could prevent 8.5% of incident HF cases.150 For diabetes mellitus, a relatively modest 5% reduction in its prevalence would lead to approximately 53 and 33 fewer incident HF hospitalizations per 100 000 person-years in blacks and whites, respectively.140 These results indicate that even modest modification of these risk factors would favorably impact the burden of HF.

Conclusions
HF is a staggering clinical and public health problem. The study of the epidemiology of HF demonstrated that although HF is associated with significant mortality, morbidity, and healthcare expenditures, particularly among those aged ≥65 years, this burden is not related to an increase in the incidence of the disease.

Rather, it reflects the chronic clinical course of patients living with HF, whereby progress in reducing HF-related mortality translates to nearly 1 million hospitalizations for HF occurring each year with frequent readmissions. To improve outcomes for patients and prevent hospitalizations, an in-depth understanding of the causes of hospitalizations in patients living with HF is imperative.

Over time, the case mix of HF is changing with a growing proportion of cases presenting with preserved EF and the causes of HF are evolving. These secular trends underscore the importance of continued disease surveillance to plan prevention and care programs.

Despite progress in reducing HF-related mortality, hospitalizations for HF remain very frequent and rates of readmissions continue to rise. To prevent hospitalizations, a comprehensive characterization of predictors of readmission in patients with HF is imperative and must integrate the impact of multimorbidity related to coexisting conditions.

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None.

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