A Lifetime of Prevention
The Case of Heart Failure
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It is widely acknowledged that heart failure (HF) is a growing public health and clinical problem and that, once diagnosed, the prognosis is grim. In this issue of Circulation, Lloyd-Jones and colleagues1 provide a lifetime perspective of HF from the Framingham Heart Study. Several messages are notable. First, the lifetime risk of developing HF at all ages is ~20 percent in both men and women. This contrasts with the fact that short-term risks are substantially lower in younger individuals compared with older individuals (eg, 0.5% 5-year risk versus 21.0% lifelong risk, or a 40-fold difference in those 40 years of age; 1% versus 20%, or a 20-fold difference in those 60 years of age; and 8% versus 20%, or a 2.5-fold difference in those 80 years of age). This implies that for preventive strategies to be fully effective against HF, they may have to be implemented throughout the lifetime of an individual.

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Second, the analysis by Lloyd-Jones et al confirms the importance of hypertension and myocardial infarction, which together account for about three quarters of the population-attributable risk of HF. Both myocardial infarction2 and hypertension3 are largely preventable with currently known and available strategies (Table). The importance of hypertension is exemplified by the 2-fold increase in risk of HF among those with systolic blood pressure (BP) >160 and diastolic BP >90 mm Hg compared with those with both systolic BP <140 and diastolic BP <90. This risk is likely to be continuous so that even lower rates of HF can be expected among those with systolic BP <120 mm Hg. Hypertension can be prevented by modest weight loss, moderate exercise, reduction in sodium consumption, and decreased alcohol consumption.4 All of the above approaches, when used together, could potentially lead to an important drop in the mean BP levels of entire populations. A 5-mm drop in population systolic BP could in itself reduce the age-specific rates of HF by at least about one quarter. In those with hypertension (prevalence of ~20%), a 10-mm reduction in systolic BP has been demonstrated to reduce the incidence of congestive HF by 50%.5 Therefore, a combined strategy of treatment of hypertension along with public health approaches to lower the mean BP of the population should reduce the age-specific rates of HF in the population by more than one third and by as much as one half.

The second major antecedent cause of HF is myocardial infarction. The risk of myocardial infarction can be reduced by avoidance of smoking and modification of the classical risk factors (eg, lowering LDL cholesterol, lowering BP). These risk factors can be favorably altered through population-level lifestyle changes, such as weight reduction, and through strong antitobacco campaigns and legislation. The importance of obesity in causing HF has recently been emphasized by a separate report from the Framingham Heart Study.6 In this regard, obesity should be considered a proximate risk factor for HF and may exert its influence both directly and indirectly, by promoting high BP, diabetes, dyslipidemia, and atherosclerosis. Therefore, primordial prevention efforts (ie, prevention of the development of risk factors through avoidance of obesity) are likely to have an impact on a number of manifestations of cardiovascular disease, including HF.

To be successful in primordial prevention, a broad-ranging effort is needed, including urban planning, changing the nature of most kinds of work to promote physical activity, legislation that promotes consumption of healthy foods and discourages consumption of unhealthy foods (through positive and negative financial incentives), and education about healthy lifestyles throughout life. A multitiered approach at societal, community, and family levels, complemented by aggressive control of risk factors in high-risk individuals, is likely to yield the greatest impact.

Thus far, research aimed at preventing HF in high-risk individuals has been relatively modest in comparison with the extensive efforts at discovering new treatments for patients after HF has developed. Apart from the BP-lowering trials3 and the Prevention trial of the Study of Left Ventricular Dysfunction (SOLVD), which used ACE inhibitors in those with low ejection fractions,5 there have been no other efforts that have primarily targeted the prevention of HF. By contrast, studies involving treatment of patients with clinical HF are numerous.6 These worthwhile efforts have established the roles of ACE inhibitors,7 β-blockers,8 and spironolactone9 in reducing mortality and morbidity and that of digoxin in preventing worsening HF, but further progress has been slow. Several years of treatment with all of these drugs could potentially prolong average survival by ~3 to 4 years in HF patients (extrapolating from the SOLVD 12-year experience).10 At least a similar improvement in survival should be expected from preventive efforts (such as reduced obesity, BP lowering) in high-risk patients. However, because preventive
efforts are likely to be applicable to much larger numbers of individuals, such efforts could lead to greater population-level benefits. At the other extreme, it is likely that an average survival extension of only a few months can be achieved by devices (eg, implantable cardiac defibrillator or left ventricular assist devices), and more significant improvements can be achieved in highly selected individuals who undergo heart transplantation. Although these efforts certainly benefit very ill patients, the population-level impact is likely to be extremely small because it can only be applied to a very small group of patients.

It is therefore important that we develop a population-level strategy of prevention of HF that applies to the large number of “at-risk” individuals. Such a strategy would complement our current approaches that are aimed at intensive management of patients with manifest HF. Implementation of our current knowledge of both prevention and treatment of hypertension, obesity, and atherosclerotic vascular disease has the potential to have a large impact on the age-specific incidence and mortality from HF.

<table>
<thead>
<tr>
<th>Type of Subject</th>
<th>Percentage of Those in the Population &gt;40 Years of Age Affected</th>
<th>Type of Intervention</th>
<th>Population-Level Impact</th>
</tr>
</thead>
<tbody>
<tr>
<td>(A) Class IV HF + low ejection fraction</td>
<td>&lt;0.2</td>
<td>Transplantation; left ventricular assist device, implantable cardiac defibrillator</td>
<td>Tiny</td>
</tr>
<tr>
<td>(B) Any congestive HF</td>
<td>&lt;2</td>
<td>ACE inhibitors, β-blockers, spironolactone</td>
<td>Modest</td>
</tr>
<tr>
<td>(C) High-risk individuals (eg, those with hypertension or who have had a myocardial infarction)</td>
<td>&lt;20</td>
<td>Antihypertensive therapy; drugs to lower cholesterol, ACE inhibitors, smoking cessation</td>
<td>Moderate</td>
</tr>
<tr>
<td>(D) Obese or overweight individual (eg, body mass index &gt;25), plus those in above category</td>
<td>&gt;40</td>
<td>Weight loss, plus above measures</td>
<td>Large</td>
</tr>
</tbody>
</table>

References


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