Commentaries

Sixty Years of Preventive Cardiology: A Framingham Perspective

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I was pleased to receive the 2011 Joseph Stokes Award for excellence in preventive cardiology from the board of the American Society for Preventive Cardiology. Joseph Stokes and I were close colleagues for most of our careers as faculty at Harvard University, Boston University, and the Framingham Study. We shared a long and abiding interest in preventive medicine in general and preventive cardiology in particular. We were fortunate to have participated in the evolution of preventive cardiology from its inception by identifying correctable predisposing cardiovascular risk factors using population data from the pioneering Framingham Study. It is gratifying to see how this has stimulated cardiovascular drug development, trials to demonstrate their efficacy, and public health measures to alter cardiovascular disease (CVD)-promoting behavior in the population.

It is satisfying to reflect on the many important lessons gleaned from our epidemiological investigation over the past 6 decades. Clinical misconceptions were corrected. The impact of overt and subclinical atherosclerotic CVD was revealed. The importance of the principle of multivariable risk factor influences on CVD, with no single essential and sufficient cause, was established. Population-based incidence of nonfatal cardiovascular events was provided to enhance mortality statistics. Useful multivariable cardiovascular risk assessment profiles were developed that minimize possibilities of falsely reassuring or needlessly alarming many potential CVD candidates.[1]

We provided clues to the pathogenesis of atherosclerotic cardiovascular for further investigation. Familial and genetic influences are being studied in second and third generations of our study participants. We are now able to document the lifetime risks of CVD events and its predisposing risk factors from 6 decades of long-term investigation.[2] This provides a more compelling depiction of CVD risk for both patients and physicians than current 10-year risk assessments.

Major contributions of observational studies to preventive cardiology also include: proposal and fine-tuning of the multivariable risk factor concept of the etiology of atherosclerotic CVD and its use for risk assessment and gaining pathogenic insights, arousal of interest in preventive cardiology with epidemiology as its fundamental tenet, and redefinition of acceptable levels for predisposing risk factors from usual as normal to optimal for avoiding CVD.[3] The complete gamut of cardiovascular events not captured from clinical data, including those immediately fatal and those silent or unrecognized, were revealed. Pathogenic insights provided include the fact that all the major risk factors cluster, suggesting underlying metabolic linkage; that silent or unrecognized cardiovascular events are not benign; that atrial fibrillation is a direct embolic threat, rather than a derivative of underlying cardiac disease, as formerly believed; that obesity promotes multiple atherogenic risk factors that comprise a metabolic syndrome; and that lipid ratio profiles provide insights into atherogenesis and their CVD hazard.[3,4]

Other lessons include the observation that lifetime risk assessment better reflects the hazard of cardiovascular risk factors than short-term assessment, and that age and positive family history can be regarded as modifiable risk factors.[5,6] Population-based multivariable risk instruments have enabled assessment of the net, joint, and interactive influence of risk factors and estimation of multivariable CVD risk.[7] Further lessons gleaned about preventive cardiology include: the fact that all major identified risk factors cluster, implying metabolic linkage; that cigarette smoking is a major cardiovascular as well as pulmonary risk; that silent or unrecognized cardiovascular events are common and far from benign; that obesity is an important promoter of major risk factors; that most of the cardiovascular hazard of type 2 diabetes derives from its accompanying risk factors; and that lipid ratio profiles provide best estimates of lipid atherogenesis and their cardiovascular hazard.[8]

It was shown that benign essential hypertension was far from benign or essential and that the J-curve phenomenon of hypertension risk may derive from an increased pulse pressure. The view that most of the adverse consequences of hypertension derive from the diastolic pressure was dispelled by demonstration of the dominant influence of the systolic and pulse pressure.[9]

Useful multivariable risk assessment profiles were developed for coronary disease, stroke, peripheral artery disease, and cardiac failure. We also crafted a total CVD multivariable risk assessment tool that encompasses all the forgoing, which can also be used to predict occurrence of each of its component events.[7] Further elaboration of multivariable risk algorithms is needed because 40% to 50% of people having cardiovascular events are not considered high-risk candidates by most risk profiles in use. These events occur in people deemed at intermediate risk. We need to better define risk within this large population segment. Areas under consideration for improved risk assessment are biomarkers, genetic markers, and vascular imaging. Costly biomarkers may not significantly enhance the Framingham

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study multivariable risk score, but may be useful for assessing benefits of therapy. Imaging requires scanning technology to find already existing vascular disease. It stratifies intermediate cardiovascular risk best, but requires a primary care referral.

Challenges for the future include postrecession funding, finding better ways to stimulate greater use of multivariable risk assessment in clinical primary care, determining appropriate us of technological advances in molecular medicine, imaging, ecological forces, and new interventional tools in population research.

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