Atherosclerosis is a chronic immunoinflammatory disease triggered by lipoprotein retention in the subendothelium and produces its most serious manifestations by triggering arterial thrombosis when the plaque ruptures or erodes.1 Atherothrombotic cardiovascular disease remains the leading cause of death in the West and is achieving similar dubious distinction in developing countries.2 Atherosclerosis begins at a young age, but it remains clinically silent for a long time and the majority of clinical manifestations occur later in life. Despite considerable progress, regrettably 50% of life-threatening acute cardiovascular events occur in previously asymptomatic individuals. Identifying individuals at risk for future cardiovascular events affords an opportunity for risk reduction and thus remains a major imperative for healthcare professionals.

In 2006 the SHAPE (Society for Heart Attack Prevention and Education) Task Force introduced an algorithm incorporating noninvasive screening for subclinical atherosclerosis to augment risk factor–based risk stratification in asymptomatic subjects between the ages of 45 to 75 in men and 55 to 75 in women, excluding those at very low risk or at high risk.3 In June 2009, the Texas Governor Rick Perry signed off on the Texas Heart Attack Prevention Bill introduced by Rep Rene Oliveira, mandating health-benefit plans to cover noninvasive screening for subclinical atherosclerosis. This bill grew out of the 2006 SHAPE Task Force guidelines and became effective September 1, 2009; an earlier version of the bill had been rejected in 2007. This generated a mixed reaction from healthcare professionals and others, much of which played out in the media. The critics argued that such an action is inappropriate because: (1) the SHAPE guidelines were created by an ad hoc group and had not been explicitly endorsed by the American College of Cardiology (ACC) or the American Heart Association (AHA), and (2) SHAPE guidelines are not based on randomized clinical trials.4,5

What Is SHAPE?
The SHAPE is a grass roots nonprofit organization with an audacious mission to eradicate heart attacks. It grew from its predecessor, an Association for Eradication of Heart Attack, started by Dr Morteza Naghavi, then on faculty at the Texas Heart Institute. The SHAPE Task Force comprises a diverse group of healthcare professionals from varied backgrounds who volunteer their time and expertise without any compensation; in fact many SHAPE members donated funds to support its mission. The SHAPE Task Force was galvanized into action because of the limitations of the current risk factor–based identification of asymptomatic individuals at risk for an unheralded acute cardiovascular event and that a large body of data supported the incremental prognostic value of subclinical atherosclerosis detection. One could reasonably argue that only the professional societies should develop practice guidelines because they provide the imprimatur of peer-review and conflict-free evidence based process. However, sensible guidelines developed by nonconventional groups such as the SHAPE Task Force should be judged on the merits of evidence base rather than “which group” wrote the guidelines. It would be counterproductive to malign well-meaning volunteers who have assembled the evidence to support the proposed paradigm and are intent on improving cardiovascular prevention. Although some SHAPE Task Force members with imaging expertise could have a conflict of interest, many others, including myself, have no personal axe to grind and no conflict to disclose. Nevertheless, all potential conflict of interest by SHAPE members has been transparently disclosed.6

Scientific Underpinnings Behind the SHAPE Paradigm

Because the lifetime risk of cardiovascular events is quite high, one could consider the whole population at risk and implement preventive measures across the entire population. That makes sense for widespread promotion of a healthy lifestyle; however, at a practical level, that has proven to be an elusive goal for social, economic, and cultural reasons. Similarly, lifelong pharmacotherapy for all to manage dyslipidemia poses its own challenges of cost, accessibility, intolerance, and not to mention suboptimal adherence. If we had a uniformly effective, safe, easily available, and inexpensive intervention that would prevent most coronary heart disease (CHD) events, risk stratification would be unnecessary. Unfortunately, such an intervention does not exist. Furthermore, although the lifetime risk for CHD is quite high, it is far from 100%, and therefore unconditional treatment of all would expose a substantial number of individuals to the costs, inconvenience, and risks of lifelong pharmacotherapy without any benefit. Therefore, risk stratification to target aggressive prevention to those most at risk and avoid it in those with the least amount of risk makes sense.
The Framingham study has provided critical information about risk factors for CHD, many of them modifiable and targets of treatment. The multicountry Interheart study showed that 90% to 95% of population attributable risk of myocardial infarction is related to 9 potentially modifiable risk factors all over the world. Using simple and easily measurable variables, Framingham risk scores can be determined which predict the 10-year risk of CHD for a population, and subjects are divided into arbitrarily constructed low-risk (10-year risk less than 10%), intermediate-risk (10-year risk of 10% to 20%), and high-risk cohorts (10-year risk exceeding 20%). The National Cholesterol Education Program (NCEP) and ATP III guidelines then couple the intensity of treatment, predominantly focused on lipid modification and low-density lipoprotein cholesterol targets, to the magnitude of risk. Although these guidelines have never been validated through randomized trials, their simplicity, low cost, and reasonable prognostic accuracy have made this approach the standard template for prevention. Further improvements in risk assessment could accrue from considering the family history and high-sensitivity C-reactive protein in what has become known as the Reynold’s Risk Score; however, this too has also not been validated in a clinical trial. The shortcomings of the Framingham-NCEP risk assessment-management scheme need to be pointed out: this assessment rarely assigns a high risk score to women under the age of 70, making them ineligible for aggressive lipid modification even though their lifetime risk for CHD is nearly 40%. There is considerable variability in the magnitude of atherosclerotic burden between individuals with similar level of risk factor exposure presumably related to other known or unknown genetic and environmental risk factors, and this is relevant because the atherosclerotic burden impacts clinical outcome. Nasir reported that 79% of young men and women with a significant coronary atherosclerotic burden were not eligible for pharmacotherapy based on current NCEP/ATPIII guidelines. A substantial number (60% to 75% in men and 90% in women) of unheralded cardiovascular events occur in low- and intermediate-risk groups that would not qualify for optimal preventive therapy under the current guidelines. Akosah reported that 75% of previously asymptomatic subjects presenting with their first myocardial infarction had a Framingham risk score low enough that they would not have qualified for lipid lowering therapy before their myocardial infarction.

Although most of the population attributable risk for myocardial infarction is related to several modifiable risk factors, their specificity is low because their prevalence is also high in those who never get CHD. Intuitively, therefore, it makes sense to detect subclinical atherosclerosis, anatomic substrate for all but few of CHD events, because its detection provides an integrated view of the cumulative exposure to known and unknown risk modifiers. The potential advantage of such an approach is that those without atherosclerosis could be spared pharmacotherapy (ie, lipid modification, etc), whereas those with atherosclerosis could be recommended more aggressive risk factor modification including pharmacotherapy. Subclinical atherosclerosis can be identified by noncontrast CT imaging of coronary arteries for calcification using the coronary calcium score (CCS) and B-mode ultrasound to measure carotid intima-media thickness (cIMT) and detect carotid plaque. CCS serves as a noninvasive measure of plaque burden in both genders and multiple ethnic groups. The prognostic value of CCS, independent of and incremental to that of Framingham risk and high-sensitivity C-reactive protein, has been demonstrated in multiple studies. More importantly, substantial reclassification of Framingham risk has been demonstrated after incorporation of CCS in the Framingham Offspring Cohort and by application of SHAPE algorithm to the Dallas Heart Study Cohort. Furthermore, zero CCS observed in 40% to 45% of asymptomatic subjects identifies a very low–risk cohort who can be spared aggressive pharmacotherapy (specifically lipid modifying therapy) and additional downstream testing for CHD.

Although a zero CCS does not absolutely rule out coronary atherosclerosis, it identifies no or very low atherosclerotic burden. The major drawback of CCS is radiation exposure. The actual average radiation exposure with coronary calcium scanning is 0.6 to 1.0 mSv for electron beam computed tomography and 0.9 to 2 mSv for multislice/multidetector computed tomography, although it can vary 10-fold depending on technical factors which can be optimized to keep the radiation exposure below 1.0 mSv. A recent model suggested that a median radiation exposure of 2.3 mSv could slightly increase lifetime risk of cancer; however, the estimated risk could be higher or lower by a factor of 2 depending on assumptions used.

B-mode ultrasound to measure cIMT or detect plaque has the advantage of being risk free and could be more accessible for screening but special technical expertise in performance and interpretation are required. Prospective studies show that cIMT >75th percentile based on age, gender and race or presence of plaque predict increased future risk of myocardial infarction, stroke and death that is, generally, incremental to traditional risk factors. However, comparative studies in asymptomatic subjects have shown that CCS provides a relatively greater incremental prognostic information compared to cIMT.

Subclinical atherosclerosis detection has not been tested in randomized trials. However, observational data generally indicate that knowledge of subclinical atherosclerosis could increase the use of and adherence with risk modifying interventions which could be reasonably expected to improve outcomes; a premise also of Framingham risks assessment and NCEP/ATP III guidelines which have also not been tested in randomized trials. Several observational studies have reported a significant association between the evidence of subclinical atherosclerosis and increased use of risk modifying behavior and pharmacotherapy among asymptomatic subjects. The only randomized trial reported to date did however fail to show an impact of CCS assessment on altered Framingham risk 1 year later; however, this study comprised a small low-risk cohort with only 66 subjects with abnormal CCS. Thus, the totality of evidence from observational and prospective studies support a significant incremental prognostic value of subclinical atherosclerosis detection (in particular using CCS) and potential favorable impact on the use and compliance with risk modifying interventions making
such testing a useful adjunct to Framingham risk assessment. The significant reclassification of risk categories especially in the intermediate Framingham risk cohort afforded by CCS further suggest practical utility of such testing in selected subsets. These conclusions were supported by the endorsement of 2007 ACCF/AHA Consensus Panel. The major thrust of the SHAPE Task Force guidelines is thus largely consistent with this position. 

Ideally, legislative advocacy should not be the domain of ad hoc groups but is best achieved through the broader constituency of our professional organizations. The Texas Bill was not a SHAPE initiative, and SHAPE was not involved in lobbying efforts; on the contrary, it was Rep Oliveira who, after suffering a heart attack, reached out to SHAPE. Our professional organizations have been slow in incorporating the wealth of data supporting the incremental value of imaging guided risk assessment in specific subsets of patients. This fact coupled with the reality of at-risk but yet unidentified and untreated subjects continuing to experience unheralded major cardiovascular events and reluctance of insurers to cover this service provided an impetus for the SHAPE group to support the efforts of Rep Oliveira. Regrettably, some have chosen to ignore the evidentiary support and instead misconstrued the intentions of SHAPE. TheSHAPE paradigm is the collective effort of many dedicated volunteers motivated by the best interests of patients in mind.

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Disclosures

None.

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