Correspondence

Letters to the Editor must not exceed 400 words in length and must be limited to three authors and five references. They should not have tables or figures and should relate solely to an article published in Circulation within the preceding 12 weeks. Authors of letters selected for publication will receive prepublication proofs, and authors of the article cited in the letter will be invited to reply. Replies must be signed by all authors listed in the original publication. Please submit three typewritten, double-spaced copies of the letter to Herbert L. Fred, MD, % the Circulation Editorial Office. Letters will not be returned.

Coronary Artery Calcium and Cardiac Events

To the Editor:

In the article on electron-beam tomography in the May 27 issue of Circulation, Kondos et al report on calcium scores and events in a self-referred population who presumably paid out of pocket for their scans. Unfortunately, despite the large number of patients in this study, the data were not gathered in a manner that helps us to better understand the role of coronary calcification in risk prediction for cardiovascular events.

Coronary calcium is a risk marker, not a risk factor, a distinction well described in the recent statement from Centers for Disease Control and Prevention and the American Heart Association. A risk factor is a trait that when present elevates the risk of coronary disease and, most importantly, can be treated with resulting reduction of risk. A risk marker is merely a finding that has been associated with increased coronary risk. Risk markers have not been shown to be independently causative of coronary artery disease and, most importantly, lowering the risk marker does not necessarily lower the risk of coronary artery disease; this is true for coronary calcium as well as C-reactive protein.

As Weintraub points out in his editorial, the most important and reliable risk-prediction algorithm is the Framingham risk score. This inexpensive, office-based risk factor assessment has been validated, and, when each of these risk factors has been shown to be elevated, there is an available intervention, such as treating hypertension or hypercholesterolemia. If coronary calcium is high, there is no treatment that lowers coronary calcium. Rather, in the asymptomatic patient, we are still going back to treating the risk factors that make up the Framingham risk score. There is an extraordinarily high rate of revascularizations done in asymptomatic persons after electron-beam tomography (with a relative risk as high as 124 for revascularizations in asymptomatic men with the highest quartile of calcium scores), although there has been no benefit shown to revascularization in asymptomatic persons.

Finally, the one study that analyzed the incremental value of coronary calcium over Framingham risk score found no incremental value of coronary calcium to risk prediction. Unfortunately, the Kondos et al study did not gather the data necessary for Framingham risk assessment and so could not shed any further light on this point. Before we adopt any new and expensive technology, it is essential that we require evidence of its benefit over office-based risk assessment.

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Response

We appreciate the comments from Dr Redberg regarding our recently published article and agree that coronary artery calcium (CAC) may be more properly characterized as a risk marker rather than a risk factor. CAC as measured by electron-beam tomography (EBT) is one of a few noninvasive markers for coronary atherosclerosis. However, although there is no direct treatment for CAC, it has been reported that the extent to which the volume of EBT CAC decreases, stabilizes, or increases is directly related to treatment with 3-hydroxy-3-methylglutaryl–coenzyme A (HMG-CoA) reductase inhibitors and the resulting serum LDL cholesterol levels. Thus, CAC may be subject to modification as the result of interventions on risk factors.

In our study, all participants were initially asymptomatic, and all reported events were verified. To ensure that revascularization procedures were not triggered solely by positive EBT results, medical record documentation of unstable angina or a positive stress test was a required component of the end point definition, and revascularization procedures (coronary artery bypass grafting or catheter-based interventions) not meeting the requirements were excluded from the analysis. Our main finding that presence and degree of CAC was a significant predictor of subsequent coronary events—particularly of soft events—indicates the soundness of utilizing EBT as a screening tool for the identification of individuals with subclinical disease who can benefit from additional testing and interventions. Further, although our data do not permit the computation of Framingham risk scores, our multivariate results suggest that the CAC score provides incremental prognostic information beyond that provided by assessment of established coronary risk factors.

Given the weight placed on age in the Framingham equation and the high correlation of age with CAC score, it is perhaps not surprising that a study conducted in high–coronary-risk subjects whose average age was 66 years reported no incremental value of CAC determination over the Framingham risk score. Indeed, the substitution of CAC score for age in risk assessment equations has been proposed. In our study, average age was ~51 years, and participants were not at high risk for CHD.

We do not suggest that established methods of coronary risk estimation be replaced by EBT screening for CAC, but we do advocate continued research to determine what role EBT screening should play in assessing CHD risk and whether or not, as our findings suggest, measurement of CAC can be viewed as a tool providing additional prognostic information beyond assessment of risk using traditional CHD risk factors.

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