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Carotid intima-media thickness and coronary atherosclerosis: weak or strong relations?

We would like to respond to a recent article by Bots et al.1 We agree with the authors’ conclusion that there is a relationship between carotid intima-media thickness (CIMT) and coronary atherosclerosis. However, significant progress has been made in the analysis of IMT and lesions since the time many of these studies were conducted. Furthermore, the predictability of event risk has evolved from large epidemiological prediction to individual prediction.

In the beginning, this group recognized the importance of IMT by itself to predict stroke and myocardial infarction on a population scale.2 Nevertheless, in 2007, the analysis method has clearly evolved since the quoted studies were performed. For example, the authors previously published an article on a related topic3 and the letter to the editor by Barth et al.4 addressed similar issues. The response to that letter by Bots et al. stated that ‘our CIMT measurement predicts future disease in a magnitude similar to that of population based studies that use either manual tracings or automated edge detection tracings.’ A fully automated individualized analysis method is now possible and may, given a long-term sequential database, lead to an individual predictability that was not previously available.

Additionally, the fact that the authors are not dealing with all aspects of carotid ultrasound and coronary angiography and the incomplete use of the literature in their meta-analysis5 may explain, in part, their conclusions. Coronary angiography focuses on the lumen and is generally performed in symptomatic/advanced disease populations, whereas with IMT HeartScan, lesion detection and tissue typing are usually performed in an asymptomatic population. Only considering what is happening in the lumen to assess the disease and not the wall is debatable. Further, the importance of lesion detection as indicated by Spence6 further underscores that, although in large population studies manual or automated edge detection tracings may demonstrate a relationship, it fails to assess lesions or plaque composition, resulting in low confidence of event predictability on an individual basis. Measuring the area of such lesions, particularly when assessing progression, is much more informative than measuring the thickness alone, because plaque progresses along the carotid artery at 2–4 times faster than it thickens.7 In a prospective study,8 a risk score based on age, blood pressure, smoking, and cholesterol predicted only 32% of patients with vascular events over a 5-year period, whereas 77% of events occurred among patients in the top quartile of plaque area.

Finally, the clinical relevance and long-term follow-up in different ethnic and age groups of IMT measurements in combination with plaque formation underscores the importance of current advances in IMT technology.5,6 Our large database can reliably predict on an individual basis the likelihood of a cardiovascular complication within several years if no intervention is performed. The SHAPE report highlights the importance of an initial IMT measurement for clinical follow-up.9 Quantitative IMT in combination with lesion detection and plaque composition assessment is used widely in clinical settings with great predictability on an individual basis for cardiovascular outcomes.

References

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Discrepancy in guidelines for the prevention of thromboembolism in patients with prosthetic heart valves

We have read with interest and became aware of diverse therapeutic approaches in several recently published guidelines, referring to patients with prosthetic heart valves and atrial fibrillation.

In the latest European guidelines on the management of valvular heart disease,1 life-long oral anticoagulation is recommended for all patients with mechanical valves and for those patients with bioprostheses who have additional indications for anticoagulation such as atrial fibrillation, heart failure, or impaired left ventricular function. Indications for the addition of antiplatelet therapy include concomitant arterial disease, in