Coronary artery calcium for the prediction of mortality in young adults <45 years old and elderly adults >75 years old

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Aims
To determine if coronary artery calcium (CAC) scoring is independently predictive of mortality in young adults and in the elderly population and if a young person with high CAC has a higher mortality risk than an older person with less CAC.

Methods and results
We studied a cohort of 44 052 asymptomatic patients referred for CAC scans for cardiovascular risk stratification. All-cause mortality rates (MRs) were calculated after stratifying by age groups (<45, 45–54, 55–64, 65–74, and ≥75) and CAC score (0, 1–100, 100–400, and >400). Multivariable Cox regression models were constructed to assess the independent value of CAC for predicting all-cause mortality in the <45- and ≥75-year-old age groups. The MR increased in both the <45- and ≥75-year-old age groups with an increasing CAC group. After multivariable adjustment, increasing CAC remained independently predictive of increased mortality compared with CAC = 0 [<45 age group, hazard ratio (95% confidence interval): CAC = 1–100, 2.3 (1.2–4.2); CAC = 100–400, 7.4 (3.3–16.6); CAC > 400, 34.6 (15.5–77.4); ≥75 age group: CAC = 1–100, 7.0 (2.4–20.8); CAC = 100–400, 9.2 (3.2–26.5); CAC > 400, 16.1 (5.8–45.1)]. Persons <45 years old with CAC = 100–400 and CAC >400 had 2- and 10-fold increased MRs, respectively, compared with persons ≥75 with no CAC. Individuals ≥75 years old with CAC = 0 had a 5.6-year survival rate of 98%, similar to those in other age groups with CAC = 0 (5.6-year survival, 99%).

Conclusion
The value of CAC for predicting mortality extends to both elderly patients and those <45 years old. Elderly persons with no CAC have a lower MR than younger persons with high CAC.

Keywords
Coronary artery calcium • Ageing • Coronary CT • Coronary heart disease

Introduction
The coronary artery calcium (CAC) score is a robust marker of coronary atherosclerotic burden. Numerous studies have demonstrated that increasing CAC predicts cardiovascular and all-cause mortality.1–3 Both the American Heart Association (AHA) and the European Society of Cardiology have suggested that it is reasonable to perform CAC testing in intermediate risk patients (10–20% 10-year risk), with the AHA extending this recommendation to low-intermediate risk patients (6–10% 10-year risk).
risk). While the clinical utility of CAC in further risk-stratifying patients deemed by traditional risk-scoring systems as intermediate risk has been studied extensively, less is known about its predictive value in young patients, as well as in the elderly. These populations are not typically classified as intermediate risk by traditional coronary heart disease risk scoring algorithms.6,7 Uncertainty remains regarding the use of CAC in young patients. Young patients are likely to be categorized as low cardiovascular risk independent of traditional cardiovascular risk factors, because the Framingham risk score (FRS) heavily weights chronological age. Furthermore, previous research has shown that coronary plaque is less likely to be calcified in young individuals.8 Although there have been few studies, Taylor et al.9 assessed the predictive value of CAC in 1634 asymptomatic 40–50-year-old men and noted that CAC was independently predictive of coronary heart disease (CHD) in those with an FRS more than 5%.

Although clinical and histological studies have confirmed that atherosclerotic calcification increases with age,8,10–12 it is not certain if CAC retains its utility in estimating atherosclerotic risk in the elderly. Two recent studies have validated the utility of CAC in the elderly and have suggested a strong ability of CAC in risk-stratifying elderly patients.13,14

We hypothesized that CAC scoring would be independently predictive of mortality in the elderly as well as in patients <45 years old and that a young person with a high CAC burden would have a higher mortality risk than an older person with less CAC. To this end, we studied the largest available cohort of asymptomatic patients referred for CAC scoring as part of clinical CHD risk assessment.15

**Methods**

The study cohort consisted of 44 052 asymptomatic individuals referred for electron beam computed tomography (EBT) for the assessment of subclinical atherosclerosis. This study incorporated data from three centres during the time period 1991–2004 (Torrance, CA; Columbus, OH; Nashville, TN), all of whom utilized a common scanning protocol.15 Methods of data collection were similar for all centres. The combined population was predominantly Caucasian, estimated at ≏88%, with smaller proportions of Hispanics, African Americans, and Asians.

Participants were referred by their primary physicians on the basis of established cardiovascular risk factors for atherosclerosis. As an inclusion criterion, patients were determined to be free of known CHD based on prior assessment by the referring physician. All participants provided informed consent to undergo EBT and for the use of their blinded data for epidemiological research. The study was conducted in accordance with the Declaration of Helsinki and received approval from the Humans Investigations Committee. Separate Committee approval was also obtained for patient interviews and collection of baseline and follow-up data.

**Risk factor data collection**

Participants completed a questionnaire for the collection of demographic and clinical characteristics, including baseline cardiovascular risk factors. Cigarette smoking was present if a subject was a smoker at the time of scanning. Dyslipidaemia was defined by the presence of a history of high total cholesterol, high low-density lipoprotein cholesterol, low high-density lipoprotein cholesterol, hypertriglyceridaemia, or current use of lipid-lowering therapy. Study subjects were considered to have diabetes mellitus if they reported using oral anti-diabetes medications or insulin. Hypertension was defined as a self-reported history of hypertension or use of antihypertensive medication. Family history of CHD was determined by the presence of a first-degree relative with a history of CHD [male <55 years old/female <65 years old in 36 010 (82% of the study population); <55 years old for both male and female relatives in 8042 participants from the Columbus, OH centre (18% of the study population)].

**Screening protocol**

All subjects underwent EBT with either a C-100 or a C-150 Ultrafast computed tomography scanner (GE-Imatron, South San Francisco, CA, USA). Using a tomographic slice thickness of 3 mm, a total of ~40 sections were obtained from the level of the carina to the diaphragm. Image acquisition was electrocardiographically triggered at 60–80% of the R-R interval, using a 100-ms/slice scanning time. A calcified lesion was defined as ≥3 contiguous pixels with a peak attenuation of at least 130 Hounsfield units. Lesions were scored using the method developed by Agatston et al.16

**Follow-up and mortality ascertainment**

Patients were followed for a mean of 5.6 ± 2.6 years (range 1–13 years). The primary endpoint for the study cohort was all-cause mortality. Ascertainment of mortality was conducted by individuals blinded to baseline historical data and EBT results, and was verified using the Social Security Death Index. The United States Social Security Death Index is a national registry of all deaths that occurred within the USA. Follow-up, therefore, occurred by studying this index for the reporting of the deaths of patients enrolled in the study, allowing for mortality ascertainment in 100% of participants. Follow-up was not obtained by direct contact with patients, either by clinic visits or by telephone.

**Statistical methods**

The baseline characteristics of the study population are presented by age group (<45, 45–54, 55–64, 65–74, and ≥75 years old) and in aggregate for the entire study population. Risk variables are expressed as frequencies, with the mean number of risk factors expressed as the mean ± SD. Risk factor burden was compared across increasing age groups using analysis of variance techniques; χ² analysis was used for proportional frequencies of categorical variables. A P-value of <0.05 was considered statistically significant, and all tests for statistical significance were two-sided.

Annualized all-cause mortality rates (MRs) were estimated by dividing the number of deaths by the number of person-years at risk. Mortality rates were first expressed for each age group and then stratified according to the CAC group. The Kaplan–Meier survival curves were constructed for time to all-cause survival by increasing CAC, stratified by the age group. For each age group, the effect of the CAC group on all-cause mortality was evaluated by calculating hazard ratios (HRs) and 95% confidence intervals (CIs) with Cox proportional hazards regression modelling using CAC = 0 as the reference group. This model was adjusted for gender, hypertension, diabetes mellitus, dyslipidaemia, smoking, and a family history of CHD. We also adjusted for age as a continuous variable in this age group stratified analysis to adjust for residual confounding by age. The interaction between the age group and the CAC group on mortality was also tested. Schoenfeld residuals were calculated and visually interpreted to evaluate the validity of proportional hazards.
assumptions. There were no deviations from the proportional hazards assumption in any multivariable model.

We compared the relative predictive value of the age group and the CAC group on mortality by comparing their respective z-scores from separate fully fitted multivariable Cox models. Further comparison was conducted by constructing two univariable Cox proportional hazards regression models, the first with age only and the second with CAC only, both as continuous variables. The area under the receiver operator curve (ROC) was then calculated and compared for each model using the standard techniques.

All statistical analyses were performed with STATA version 10 (STATA Corp., College Station, TX, USA).

Results

The mean age of the study population was 54.4 ± 10.7 years. Males comprised 54% of the study population, 37% of patients had a family history of premature CHD, and 14% were smokers at the time of the study. Diabetes mellitus, hypertension, and dyslipidaemia were present in 5, 24, and 30% of the study population, respectively. The proportion of patients with hypertension, diabetes mellitus, and dyslipidaemia increased significantly with the increasing age group, while the prevalence of tobacco use or a positive family history of CHD decreased with increasing age (Table 1, P < 0.0001 for all risk factors mentioned).

The proportion of participants with no CAC in both genders decreased with the increasing age group, from ~70% in the <45-year-old age group to 16% in the ≥75-year-old age group (Figure 1A, P < 0.0001). Conversely, the percentage prevalence of patients in the CAC = 101–400 and >400 subgroups increased progressively with the increasing age group (Figure 1B; CAC = 101–400: <45 age group 3%, ≥75 age group 25%; CAC > 400: <45 age group 1%, ≥75 age group 36% (P < 0.0001)).

The Kaplan–Meier survival curves demonstrate that within both the <45- and >75-year-old age groups, the increasing CAC group predicted higher mortality (Figure 2A and B). A CAC score of zero predicted a survival of ≥98% over 5.6 years in all age groups studied (Table 2). In the <45-year-old age group, increasing CAC predicted progressively lower survival at mean 5.6-year follow-up (CAC = 0, 99%; CAC = 1–100, 99%; CAC = 101–400, 96%; CAC > 400, 86%). Similarly, in the ≥75-year-old age group, the increasing CAC group predicted a progressively lower survival at mean 5.6-year follow-up (CAC = 0, 98%; CAC = 1–100, 92%; CAC = 101–400, 91%; CAC > 400, 81%).

Within the <45-year-old age group, MRs increased from 0.7 to 6.8 and 27.6 (per 1000 person-years) as CAC increased from 0 to 100–400 and >400 (Figure 3). When compared with the 2007 US Census Data,17 patients <45 years old who had CAC ≤100 had an MR of 67.3/100 000 (CAC = 0) and 161.9/100 000 (CAC = 1–100), which compares favourably with the national average for this age group (184/100 000). In contrast, CAC = 100–400 and CAC > 400 groups had much higher annual MRs of 683 and 2761 (per 100 000).

Other age groups demonstrate a similar, though attenuated, increase in all-cause mortality with increasing CAC. In the ≥75-year-old age group, a CAC score of zero was associated with an MR of 2.8/1000 person-years, which is comparable with that of a young person with mild CAC (<45 age group: CAC = 0, 0.6; CAC = 1–100, 1.6).

Increasing CAC independently predicted an increased risk of all-cause mortality among all age groups (Table 3). In the <45-year-old age group, increasing CAC was associated with a 2–34-fold increased risk of all-cause mortality when compared with CAC = 0 [CAC = 1–100, HR 2.3 (95% CI 1.2–4.2); CAC = 101–400, HR 7.4 (95% CI 3.3–16.6); CAC > 400, HR 34.6 (95% CI 15.5–77.4)]. Similarly, in the ≥75-year-old age group, CAC > 400 was associated with 16 times the mortality risk of CAC = 0 (CAC > 400, HR 16.1 (95% CI 5.8–45.1)). The interaction between the age group and the CAC group on all-cause mortality reached statistical significance (z = −2.48, P = 0.013) and carried a negative value. Thus, the relative risk of death for a given CAC category was slightly lower among the older age groups.

When assessed in separate models, age and CAC categories were responsible for a similar proportion of the overall model variation (z-score: 17.4 for CAC vs. 18.5 for age, P = 0.25). The area

| Table 1 | Clinical characteristics of subjects according to age groups |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Risk factor     | <45 (n = 8143)  | 45–54 (n = 15 628) | 55–64 (n = 12 439) | 65–74 (n = 6036) | ≥75 (n = 1663) | P-value |
| Mean age        | 40              | 50              | 59              | 69              | 79              |        |
| Follow-up duration (years) | 5.8 (2.7) | 5.7 (2.6) | 5.6 (2.6) | 5.5 (2.6) | 4.8 (2.1) | <0.0001 |
| Female gender   | 3778 (46.4)    | 7234 (46.3)    | 5796 (46.6)    | 2709 (44.9)    | 819 (46.1)    | 0.27   |
| Diabetes mellitus | 217 (2.7) | 712 (4.6) | 770 (6.2) | 509 (8.4) | 180 (10.0) | <0.0001 |
| Smoking         | 1142 (14.0)    | 2299 (14.7)    | 1685 (13.6)    | 753 (12.5)     | 141 (7.8)     | <0.0001 |
| Hypertension    | 1272 (15.6)    | 3330 (21.3)    | 3382 (27.2)    | 1932 (32.0)    | 650 (35.6)    | <0.0001 |
| Dyslipidaemia   | 1926 (23.7)    | 4597 (29.4)    | 3923 (31.5)    | 2018 (33.4)    | 562 (31.1)    | <0.0001 |
| Family history  | 3224 (39.6)    | 5951 (38.1)    | 4509 (36.3)    | 2128 (35.3)    | 590 (32.7)    | <0.0001 |
| Total number of risk factors | 0 (%) | 1 (%) | 2 (%) | ≥3 (%) |        |
| 47              | 24              | 17              | 12              | 44              | 22              | 20              | 16              | 38              | 23              | 22              | 16              | 32              | <0.0001 |
under the ROC for CAC (continuous) was higher than the area under the ROC curve for age (continuous) in univariate models fit separately for CAC and age (0.773 vs. 0.745, $P=0.002$).

**Discussion**

Our study demonstrates that in a large, asymptomatic population, CAC remained an independent predictor of all-cause mortality in both the <45- and ≥75-year-old age groups. Previous radiologic-al$^{8}$ and autopsy$^{18}$ studies have established that CAC increases with age. Whether this finding occurs as a result of longer exposure to known cardiovascular risk factors or is the consequence of the cellular and structural changes intrinsic to the ageing process itself is unclear. This has led to some uncertainty in the applicability of CAC scoring for determining the intensity of risk modification therapies, specifically in patients at the extremes of chronological age.$^{4,5}$

**Previous studies of coronary artery calcium in the elderly population**

Our results corroborate the findings of other large studies validating the prognostic utility of CAC scoring in the elderly.$^{13,14,19}$ Vliegenthart et al.$^{13}$ studied a subset of elderly patients in the Rotterdam Coronary Calcification Study, a prospective population-based study designed to evaluate the determinants and consequences of coronary calcification. This study demonstrated...
a 2-fold increase in mortality in 1795 patients >70 years old who had CAC > 100, when compared with CAC ≤ 100. Raggi et al. reported a 6–11-fold higher mortality risk among elderly individuals with increasing CAC burden.

LaMonte et al. reviewed the incidence of CHD death or non-fatal myocardial infarction in an asymptomatic population which had CAC scoring performed as part of cardiovascular risk assessment. Among patients >65 years old, CAC > 400 was associated with a 9-fold increased incidence of events compared with those with no CAC (CAC = 0, 0.9 events/1000 person-years; CAC > 400, 8.2 events/1000 person-years). Our study emulated this trend with the finding that in individuals ≥75 years old, increasing CAC predicted a significantly increased MR when compared with CAC = 0, independent of known cardiovascular risk factors (Table 3).

The utility of coronary artery calcium = 0 in the elderly

Blaha et al., in a previous analysis of our cohort, observed that the absence of CAC predicted excellent 10-year survival rates and suggested that a CAC score of zero might be used as a rationale to forego repeated imaging studies. Rozanski et al. evaluated this ‘gate-keeper’ approach to CAC scoring by randomizing asymptomatic individuals with cardiovascular risk factors to either a ‘scan’ or a ‘no-scan’ protocol. Knowledge of a CAC score of zero lead to less invasive procedures and a more frugal healthcare utilization approach than the ‘no-scan’ group. We have added to the present knowledge of a CAC score of zero, by demonstrating that individuals ≥75 years old with a CAC score of zero had an excellent mean 5.6-year survival rate of 98%. The prevalence of a CAC score of zero in individuals ≥75 years old was 16% (285 of 1806), whereas 39% had CAC ≤ 100 (706 of 1806). The fact that a significant fraction of the elderly population had no CAC and that those patients could potentially be reclassified as lower risk is of immediate clinical impact with regard to downstream healthcare utilization. Overall, our results suggest that the broader use of CAC scoring in elderly patients may be clinically useful.

The incidence of side effects from high-dose statins and excessive bleeding from aspirin may be increased in the elderly. Orthostatic hypotension is more common in elderly hypertensive patients and is associated with an increased risk of falls and a 64% increase in age-adjusted mortality. These factors may be significant for those elderly persons with a CAC score of zero, a subgroup found to have a very low likelihood of a cardiovascular event (Table 4, 98% survival over 5.6 years). Indeed, the

### Table 2

<table>
<thead>
<tr>
<th>Age group</th>
<th>CAC group</th>
<th>Absolute endpoints</th>
<th>Estimated survival at mean follow-up (5.6 years) (%)</th>
<th>Estimated 10-year survival (%)</th>
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<td>99.6</td>
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<tr>
<td></td>
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<td>94.6</td>
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<tr>
<td></td>
<td>&gt;400</td>
<td>10/75</td>
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<td>86.1</td>
</tr>
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</tr>
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<td>91.0</td>
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<td>92.1</td>
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<td></td>
<td>&gt;400</td>
<td>108/650</td>
<td>81.1</td>
<td>57.6</td>
</tr>
</tbody>
</table>

*Survival estimates derived from the Kaplan–Meier estimates of time to all-cause mortality.

Insufficient data points to derive a reliable estimate.

Figure 2 (A and B) The Kaplan–Meier survival curves in the <45-year-old and >75-year-old age groups based on the coronary artery calcium group.
Coronary artery calcium in younger individuals

Younger patients have a lower burden of CAC, leading to the suggestion that a CAC score would be of lesser value in this population. Indeed, in our study, only 4% of patients in the <45-year-old age group were noted to have CAC > 100. In comparison, the Coronary Artery Risk Development in Young Adults (CARDIA) study, a population-based study of young individuals, demonstrated a prevalence of CAC > 100 of 1.6% in patients aged 33–45, whereas the PACC study, which enrolled participants age 40–45, showed nearly 7% of patients with CAC ≥ 45, although men were overrepresented in this study.

Coronary artery calcium > 100 in this age group accounted for an alarming and disproportionately high MR when compared with CAC = 0 (Figure 3). Additionally, the presence of even mild CAC (1–100) was noted in 29.8% of the <45-year-old age group and was associated with a 2-fold increased risk of mortality. This suggests that the number needed to scan to detect one individual <45 years old with any CAC is ~4.

A more exaggerated increase in MR was observed with CAC > 400 in the <45-year-old age group than was expected (Figure 2: 27.6/1000 person-years, compared with 0.7/1000 person-years for CAC = 0). It is possible that young persons with advanced CAC have a more aggressive form of atherosclerosis making them particularly vulnerable to adverse outcomes. Another postulated mechanism is the development of coronary artery collateral vessels, which has been shown in a recent meta-analysis to have a 36% lower mortality risk compared with low collateralization. Collateralization is directly related to the duration of angina and CHD, and hence may be less likely to be developed in younger individuals. Additionally, there are fewer competing cardiovascular risk factors in individuals <45 years old to account for this disparity (Table 1), thereby highlighting the attractiveness of CAC scanning to identify high-risk individuals in this otherwise low-risk age group.

Arterial age vs. chronological age

Our study reveals that CAC independently predicts mortality regardless of the age group. Our results highlight the concept of ‘arterial age’ rather than biological age as a cardiovascular risk factor. McClelland et al. conceptualized ‘arterial age’ as a function of a given CAC score. In a model for incident CHD risk controlling for biological age and arterial age, only the arterial age construct was found to be significant, suggesting that biological age does not provide additional information after controlling for a calculated arterial age.

Our study findings are consistent with prior reports with regard to the prognostic utility of CAC in the elderly population and add

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**Table 3** The hazard ratio for all-cause mortality for increasing coronary artery calcium within each age group

<table>
<thead>
<tr>
<th>Age group</th>
<th>CAC</th>
<th>0–100</th>
<th>101–400</th>
<th>&gt;400</th>
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<tr>
<td>&lt;45</td>
<td>1 (ref)</td>
<td>2.3 (1.2–4.2)</td>
<td>7.4 (3.3–16.6)</td>
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<td>45–54</td>
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<td>1.6 (1.0–2.6)</td>
<td>3.3 (1.9–5.7)</td>
<td>6.0 (3.4–10.6)</td>
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<tr>
<td>55–64</td>
<td>1 (ref)</td>
<td>3.2 (2.1–5.0)</td>
<td>3.9 (2.5–6.2)</td>
<td>7.3 (4.7–11.4)</td>
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<tr>
<td>65–74</td>
<td>1 (ref)</td>
<td>2.7 (1.6–4.5)</td>
<td>3.8 (2.2–6.3)</td>
<td>7.0 (4.2–11.4)</td>
</tr>
<tr>
<td>75–84</td>
<td>1 (ref)</td>
<td>7.0 (2.4–20.8)</td>
<td>9.2 (3.2–26.5)</td>
<td>16.1 (5.8–45.1)</td>
</tr>
</tbody>
</table>

Reference group: CAC = 0 (HR = 1). Multivariable Cox regression analysis: adjusted for age (as a continuous variable for residual confounding within each age group), gender, hypertension, diabetes mellitus, dyslipidaemia, family history of CHD, tobacco use.

*All-cause mortality risk: HR (95% CIs).*
to the current literature by describing the interaction across the entire spectrum of ageing and a CAC score for the prediction of all-cause mortality. Additionally, our study highlights the finding that all-cause mortality is more strongly associated with CAC burden rather than chronological age. In those with a CAC score of zero, although increasing age was associated with slightly higher relative risk of mortality, the absolute risk remained very low even among elderly patients. The absence of CAC can be used to identify elderly individuals at an extremely low risk, among whom lifestyle interventions may be advocated with the avoidance of invasive procedures and repeat imaging. Such a strategy could enable us to focus on individuals with actual disease even if they are much younger, as these patients are most likely to benefit from more aggressive preventive therapies. Conversely, young individuals (<45 years) with elevated CAC burden had a much higher mortality risk than elderly individuals (>75 years) with a CAC score of zero. We believe that our study findings will stimulate discussion with the pertinent stakeholders to consider whether this paradigm shift in risk assessment with a focus on the detection of subclinical atherosclerosis in young adults is a feasible approach in aiming to reduce overall economic healthcare costs.

**Limitations**

There is a definite potential for referral bias, given that patients were referred for CAC scanning based on their cardiovascular risk factors and perceived increased cardiovascular risk by their referring physicians. The all-cause MR for the <45-year-old age group in our study did not vary from that of the general population. Although our results regarding CAC in the elderly mirror those of the Rotterdam Coronary Calcification study, the potential for a referral bias must be considered when interpreting our results.

The questionnaire format for data collection instead of the direct measurement of risk factors lends itself to two limitations. First, there is the potential to miss cases of undiagnosed risk factors for which the patient would have reported no history on the questionnaire. Participants were screened for cardiovascular risk factors and enrolled for CAC scanning by their referring physicians. Although Hoff et al. has shown a good reliability of self-reported histories of CHD risk factors in self-referred individuals for EBT scanning, because the CHD risk factors were self-reported, the potential ‘residual confounding’ cannot be ruled out, thus possibly diminishing the strength of association of risk factors with mortality. Nevertheless, in spite of the fact that risk factors may be under reported, the absence of CAC was still associated with favourable prognosis across all levels of risk factors. Because of the aforementioned limitations, our findings need to be verified in cohorts with well-measured risk factors. Another limitation with questionnaire use is recall bias, the causes of which are multifactorial. The time elapsed since a particular exposure, the duration, the social desirability of events (tobacco use is likely to be under-reported), and differential recall in cases compared with controls (exposures may be more vivid or meaningful to cases) all interplay in recall bias. Despite this, it has also been validated as an acceptable method of assessing risk factor data and emulates risk factor collection in the everyday clinical environment.

Our model did not assess whether the cause of death was cardiovascular and did not stratify the attributable risk of specific cardiovascular events including stroke and myocardial infarction.

**Conclusion**

Contemporary cardiovascular risk-scoring algorithms disproportionately weigh chronological age as a risk factor. We have
demonstrated that CAC scoring is a powerful prognostic tool in both young and elderly patients, who are less likely to be designated as intermediate risk due to chronological age. A CAC score of zero in the elderly imparts an excellent prognosis and has the potential to positively impact healthcare expenditure. In contrast, CAC scoring has the potential to identify young patients at a greatly increased risk of death, who would benefit from aggressive risk reduction management. Further research in an unselected population-based cohort is required to corroborate our findings.

Conflict of interest: M. J. B. is on the speaker’s bureau for General Electric. No other potential conflicts of interest relevant to this study were reported.

References