Supplemental Material:

Supplemental Methods:

Definitions of baseline covariates

Definitions for covariates in ARIC and CHS were similar[1-3]. In ARIC prevalent myocardial infarction (MI) was defined as a physician-reported history of MI, and/or ECG evidence of MI as defined by the Minnesota code[4]. In CHS, prevalent MI was based on self-reported physician diagnosis validated by baseline ECG or medical record review. In ARIC prevalent diabetes mellitus (DM) was defined as a non-fasting glucose level of at least 200 mg/dL, a fasting (≥8 hours) glucose level of ≥126 mg/dL, a self-reported history of DM, or the use of diabetic medications. In CHS DM was defined as a blood glucose ≥126 mg/dL or use of diabetic medications. Both studies assessed medication use in the 2 weeks prior to the baseline examination by medication inventory[5]. Prevalent hypertension was defined as a systolic blood pressure of ≥140 mmHg, a diastolic blood pressure ≥ 90 mmHg, or physician-reported history of hypertension and use of anti-hypertensive medications. Prevalent stroke in ARIC was diagnosed by a stroke and transient ischemic attack diagnostic algorithm, the details of which have been previously reported[6]. Prevalent stroke in CHS was based on a validated self-reported physician diagnosis. Prevalent heart failure (HF) in ARIC was defined as a self-reported use of HF

medication or evidence of symptomatic HF as defined by stage 3 of the Gothenburg criteria[7] which required the presence of specific cardiac and pulmonary symptoms in addition to medical treatment of HF. Prevalent HF in CHS was defined as self-reported physician diagnosis of HF validated by medical record review or use of both diuretics and either digitalis or a vasodilator. Prevalent coronary heart disease (CHD) was defined as a history of prevalent MI (as defined above), a history of angina pectoris, or a history of coronary revascularization (either via coronary artery bypass surgery or percutaneous coronary intervention). In ARIC, prevalent atrial fibrillation (AF) was defined as either a self-reported and validated[8] history of AF or diagnosis of AF on the baseline ECG. In CHS, prevalent AF at baseline was ascertained by the ECG.

Use of anti-hypertensive medications and beta-blockers was self-reported and validated by medication inventory[5]. Physical activity index categorized exercise intensity (in CHS) or leisure activity (in ARIC) as absent, low, moderate, or high. Use of alcohol was quantified as self-reported ethanol intake in grams per week, assuming that a standard drink contains 0.6 fluid ounces or 14 grams of pure alcohol.

Definition of incident non-fatal cardiovascular events

In ARIC, potential non-fatal cardiovascular events were identified during annual follow-up examinations, annual telephone calls, and local hospital surveillance. In CHS, potential non-fatal cardiovascular events were identified during annual follow-up examinations and at 6-month phone contacts up until 1998-99, and thereafter during twice yearly phone contacts. In both studies, medical records were reviewed by a committee of physicians and adjudicated using standardized criteria.

In both ARIC and CHS, incident AF was diagnosed if detected either on 12-lead ECGs

performed during follow-up exams, or on hospital discharge records (*ICD-9* code 427.3). Self-limited AF occurring during a cardiac surgery hospitalization event was not considered an incident AF event[9,10].

In ARIC, incident stroke was defined as a sudden/rapid onset of neurological symptoms lasting more than 24 hours or leading to death in the absence of evidence of a non-stroke etiology. In CHS, incident stroke was defined as a subarachnoid hemorrhage or a neurological deficit of rapid onset lasting more than 24 hours unless death supervened, or, if less than 24 hours, an appropriate lesion to explain the deficit was seen on brain imaging. In both studies, potential incident strokes were identified through review of hospital discharge diagnoses indicative of cerebrovascular disease (*ICD-9* codes 430-438). ARIC additionally identified potential incident strokes through analysis of certain key phrases in hospital documentation and review of imaging studies with cerebrovascular findings. In both studies, potential incident stroke events were then classified using standardized criteria applied by trained physicians and a computer algorithm (in ARIC only). Further details of incident stroke adjudication have been previously reported.[11-13] In both ARIC and CHS, incident stroke included cases that were adjudicated as definite or probable strokes.

In ARIC, incident HF events were diagnosed as first HF hospitalization with *ICD-9* code 428.0-428.9 in any position of the hospital discharge list as well as characteristic signs and symptoms of congestion, characteristic chest X-ray findings, and/or assessment of left ventricular function[14]. In CHS, classification of HF required both a diagnosis from a physician and validation of this diagnosis by either: (i) active treatment for congestive HF; (ii) characteristic chest X-ray; or (iii) characteristic echocardiography or contrast ventriculography findings. Active treatment for HF was defined as a current prescription for both a diuretic and

either digitalis or a vasodilator. Characteristic chest x-ray findings included cardiomegaly or pulmonary edema, while characteristic echocardiography or contrast ventriculography findings included a dilated ventricle and/or global or segmental wall-motion abnormalities with decreased systolic function[15].

In ARIC and CHS, incident MIs were classified on the basis of the following: (i) an evolving diagnostic ECG pattern (as determined by the Minnesota Code); (ii) a diagnostic ECG pattern and abnormal cardiac biomarkers; or (iii) cardiac pain with abnormal cardiac biomarkers and either evolving ST-T wave changes on ECG or an equivocal ECG pattern[16,17]. In ARIC and CHS, CHD was defined as a definite or probable MI, angina, or a coronary revascularization procedure[17].

Measurement of ECG parameters reflecting global electrical heterogeneity

ECG signal processing

Raw digital ECG signals were exported from the GE Magellan research utility (GE Marquette, Milwaukee, WI). Baseline wander was removed using a method based on Empirical Mode Decomposition[18,19]. This is a data driven adaptive technique that decomposes a signal into a series of Intrinsic Mode Functions. The highest-order Intrinsic Mode Functions correspond to the baseline wander frequency which was subtracted from the original signal to obtain a "clean" ECG signal. Then median beat (QRST complex) was constructed and further analyzed using customized Matlab software (MathWorks, Inc, Natick, MA).

A modified inverse Dower transformation matrix[20] was used to convert the 12 standard ECG leads into orthogonal ECG leads (X-, Y-, and Z-leads) and 3-dimensional vectorcardiograms (VCGs). To simplify the visual interpretation of the VCG we assigned

positive values to the anteriorly directed Z-lead. Thus, positive values of all 3 orthogonal X-, Y-, and Z-leads indicated normal position of the anatomical heart axis, directed from the base to apex, with the apex of the heart pointing anterior, inferior, and leftward. The median QRST complex was analyzed as follows:

SAI QRST measurement

Sum absolute QRST integral (SAI QRST) was measured as the arithmetic sum of areas under the entire QRS-T curve (Figure 1A) according to the following equation:

$$SAI\ QRST = \int_{QBeg}^{TEnd} |V_x(t)|\ dt + \int_{QBeg}^{TEnd} \left|V_y(t)\right|\ dt + \int_{QBeg}^{TEnd} |V_z(t)|\ dt$$

where $V_x(t)$, $V_y(t)$, and $V_z(t)$ represent the QRS-T complex in the X-, Y-, and Z- axes respectively, *QBeg* is the onset of the QRS complex, and *TEnd* is the end of the T-wave. Baseline zero value was assigned at the end of T-wave. Trapezoidal Riemann sums (0.5 ms interval size) were used to calculate the area under the QRS-T complex.

Spatial QRS-T angle measurement

The QRS and T vector magnitudes were defined as the magnitude of the QRS and T peak vectors[21]. Spatial mean QRS-T angle was defined as the 3-dimensional angle between the mean QRS vector and the mean T vector (Figure 1B) according to the following equation:

$$Spatial\ QRS-T\ angle = \arccos\left(\frac{\overrightarrow{QRSm}\cdot\overrightarrow{Tm}}{|QRSm||Tm|}\right)$$

where $\overrightarrow{QRSm}$ represents the mean QRS vector and $\overrightarrow{Tm}$ represents the mean T vector which were computed by assessing the vectorial sum of QRS and T vectors over time:

$$\overrightarrow{QRSm} = \int_{QBeg}^{QEnd} \overrightarrow{QRS}(t)\, dt \qquad and \qquad \overrightarrow{Tm} = \int_{TBeg}^{TEnd} \vec{T}(t)\, dt$$

In order to define the origin of the QRS and T vectors, a cross point was assigned as the point located halfway between QRS onset and T offset in 3-dimensional space. The QRS and T peaks were defined as the furthest points on the QRS and T loops from the origin, respectively. The QRS and T vector magnitudes were defined as the magnitude of the QRS and T peak vectors.

Spatial ventricular gradient (SVG) measurement

SVG vector magnitude and direction (Figure 1C) were measured as following: The X-, Y-, and Z- components of the SVG were calculated by integrating the area under entire QRS complex and T wave in the X-, Y-, and Z- axes. Subsequently, the SVG vector ($\overrightarrow{SVG}$) and SVG magnitude ($|SVG|$) were defined as follows:

$$\overrightarrow{SVG} = \left[\int_{QBeg}^{TEnd} V_x(t)\, dt, \qquad \int_{QBeg}^{TEnd} V_y(t)\, dt, \qquad \int_{QBeg}^{TEnd} V_z(t) dt\right]$$

$$|SVG| = \sqrt{\left(\int_{QBeg}^{TEnd} V_x(t)\, dt\right)^2 + \left(\int_{QBeg}^{TEnd} V_y(t)\, dt\right)^2 + \left(\int_{QBeg}^{TEnd} V_z(t) dt\right)^2}$$

In addition, the azimuth and elevation of the SVG vector were defined as following:

$$SVG\ Azimuth = \arctan\left(\frac{\int_{QBeg}^{TEnd} V_z\,(t)dt}{\int_{QBeg}^{TEnd} V_x\,(t)dt}\right) \qquad SVG\ Elevation = \arccos\left(\frac{\overrightarrow{SVG}\cdot\vec{Y}_d}{|SVG|}\right)$$

where $\vec{Y}_d$ is defined as a unit vector pointing inferiorly on the Y axis.

Rationale for inclusion of ventricular paced ECGs in analysis:

Wilson's concept of the "ventricular gradient"[22] suggests that information on myocardial global electrical heterogeneity, as revealed by the SVG, is largely independent of the specific ventricular activation sequence[23]. Information on the degree of electrical heterogeneity obtained from the SVG primarily depends on heterogeneity of action potential morphology across the entire myocardium rather than on the specific sequence in which the myocardium is activated[23-25]. In addition to previous theoretical and experimental studies, the postulate of information encoded within the SVG being independent of the myocardial activation sequence was recently confirmed in a clinical study[26] which demonstrated similar intracardiac QRST integrals in patients with intracardiac cardioverter-defibrillators with and without ventricular pacing

Adjudication of sudden cardiac death outcomes

SCD was similarly adjudicated in ARIC and CHS. Available data from death certificates, informant interviews, physician questionnaires, coroner reports, and hospital discharge summaries were reviewed, in addition to circumstances surrounding the event. For unwitnessed deaths, the participant must have been seen within 24 hours of the arrest in a stable condition and without evidence of a non-cardiac cause of cardiac arrest. Participants with non-fatal cardiac arrest who were successfully resuscitated in the field but who died subsequently

during hospitalization, were appropriately adjudicated as SCD cases. Data on cases of non-fatal cardiac arrest in which participants were resuscitated and survived to hospital discharge were not available, and were not considered as SCD cases for purposes of these analyses.

In ARIC, each event was adjudicated independently by 2 investigators, and classified as "definite sudden arrhythmic death," "possible sudden arrhythmic death," "definite non-sudden death," or "unclassifiable." If disagreement existed between the first 2 reviewers, a third investigator independently reviewed the event to provide final classification. In CHS, each event was adjudicated by a cardiologist's record review, and classified as "definite sudden cardiac death," "probable sudden cardiac death," and "not sudden cardiac death or unclassifiable." A blinded second physician review of a random sample of 70 of these death records showed an 88% inter-reviewer agreement and κ=0.74 for SCD. Both of these physicians also participated on the ARIC SCD review panel, to ensure consistency of the SCD phenotype across studies.[17,27,28]. For the present analysis, SCD was defined as deaths that were definite or probable sudden deaths.

Statistical methods:

Association between GEH ECG parameters and baseline characteristics

Linear regression (adjusted for age, sex, race, cohort, and study center) was used to determine associations between baseline demographic, clinical, and traditional ECG characteristics and GEH ECG parameters. For continuous baseline characteristics, change in each GEH parameter was assessed per 1 standard deviation (SD) change in the characteristic being evaluated. As SVG azimuth and elevation are circular variables, circular statistics (Watson U-square statistic and Kuiper statistics) were used for their description and unadjusted

comparisons. Square root transformed SVG elevation and root transformed absolute SVG azimuth were used in all subsequent adjusted analyses, both when used as outcomes and as predictors, to improve model fit.

Circular variable statistics:

As SVG azimuth and elevation are circular variables, circular statistics (Watson U-square statistic and Kuiper statistics) were used for their description and unadjusted comparisons. The mean and SD direction of each circular vector variable was determined. The mean vector strength was calculated as the length of the resultant vector divided by the number of observations.

Association between GEH parameters and SCD

Cox proportional hazards analyses were employed to quantify associations between individual GEH ECG parameters and SCD. Models were fit separately on the ARIC and CHS cohorts, and on the merged data from both cohorts. We constructed four Cox proportional hazards models to adjust for potential confounders, treating GEH ECG parameters as continuous variables. Competing risks models with the same variables were also constructed to account for the competing risks of non-cardiac death and non-sudden cardiovascular death.

Model 1 adjusted for demographic characteristics (age, gender, race, study cohort, and study center). Model 2 additionally adjusted for prevalent cardiovascular disease (CHD, HF, stroke, and AF), use of beta-blockers, serum creatinine, and traditional cardiovascular risk factors (body-mass-index, hypertension, anti-hypertensive medications, DM, smoking status, alcohol intake, total cholesterol, high-density lipoprotein cholesterol, triglycerides, and physical

activity index). Model 3 was further adjusted for ECG parameters associated with SCD: heart rate, QTc, QRS duration, sex-specific Cornell product, and bundle branch block (BBB) or intra-ventricular conduction delay (IVCD). Model 4 evaluated whether the association of baseline GEH ECG parameters with SCD remained significant over time and included time-updated GEH ECG parameters, all baseline covariates in Model 3, time-updated ECG measurements (heart rate, QTc, QRS duration, sex-specific Cornell product), and time-updated incident non-fatal cardiovascular events (AF, HF, CHD, and stroke). In Model 4, GEH ECG parameters and other ECG covariates were measured longitudinally at up to 10 CHS visits or 4 ARIC visits and were updated on the date of ECG recording.

Subgroup analysis was performed in Model 3 to determine significant interactions between GEH parameters and clinical characteristics. In addition, associations between GEH parameters and SCD were also evaluated using adjusted Cox regression models incorporating quadratic splines with 4 knots.

Sensitivity analyses were conducted to evaluate the effect of adding baseline left ventricular ejection fraction (LVEF) as assessed by echocardiography into time-updated models. Because baseline LVEF was only measured in the original CHS cohort (recruited in 1989 to 1990), these analyses were restricted to the included 4,954 CHS participants who had baseline LVEF assessed. First Model 4 with time-updated measurements was re-run in only these 4,954 CHS participants; then baseline LVEF was added and the results were compared.

Longitudinal changes in GEH ECG parameters over time and risk of SCD

Mixed effect multilevel models (adjusted by age, sex, and race with participants nested within study center nested within cohort) were constructed to determine whether GEH

parameters changed over time. To investigate whether longitudinal changes in GEH parameters were independently associated with SCD, two analyses were performed.

First, the interaction with time was assessed in time-updated Cox models to test the assumption of proportionality for the hazard of time-updated variables over time. Time-updated Cox models evaluated whether an increase or decrease in GEH parameters over time was associated with a non-proportional increase or decrease in the risk of SCD over time.

In addition, separate Cox proportional hazards models were used to determine if large increases or decreases in GEH parameters between study visits 1-3 were associated with SCD. The relative change in each GEH parameter between visits 1 and 2, and between visits 1 and 3 was calculated. Categorical variables were then constructed to categorize the change in each GEH parameter. For all GEH parameters except QRS-T angle, a change of $< \pm 25\%$ of the baseline value was considered the reference value. Because larger changes in QRS-T angle were observed, a change of $< \pm 50\%$ of the baseline value of QRS-T angle was defined as the reference value for analyzing changes in QRS-T angle. For SAI QRST, SVG magnitude, SVG elevation, and SVG azimuth, categories of decrease of $\geq 33\%$, decrease of 25-32%, increase of 25-32%, increase of 33-49%, and increase of $\geq 50\%$ were compared to the reference category. Because significantly larger relative changes were observed for QRS-T angle, categories of decrease of $\geq 300\%$, decrease of 50-299%, increase of 50-200%, increase of 201-299%, and increase of $\geq 300\%$ were compared to the reference category.

To answer the question as to whether these sudden changes in GEH parameter values were independently associated with SCD, we constructed 4 Cox proportional hazards regression models. Model 1 was adjusted for age, sex, race, study center, and cohort. Model 2 was further adjusted for CHD, HF, stroke, AF, use of beta-blockers, creatinine, traditional cardiovascular

risk factors (BMI, hypertension, anti-hypertensive medications, diabetes, smoking, alcohol intake, total cholesterol, HDL cholesterol, triglycerides, and physical activity index). Model 3 was further adjusted for baseline ECG characteristics: heart rate, QTc, QRS duration, Cornell product, presence of BBB/IVCD, ventricular pacing, and incident non-fatal cardiovascular events (AF, HF, CHD, stroke). Finally, Model 4 was further adjusted for the baseline value of each GEH parameter obtained at the initial study visit.

Schoenfeld residuals confirmed that the proportional hazards assumption was valid in all Cox proportional hazards models.

Risk score development:

We constructed 2 SCD risk scores using Fine and Gray's competing risks model to test the incremental predictive value of adding GEH parameters as continuous variables to clinical characteristics. A combination of clinical guidance and backward selection was used to select covariates for inclusion in the final risk score models. To allow wider applicability of the risk score we initially assessed clinically important covariates from Model 3. Variables initially tested for inclusion included age, gender, race, CHD, stroke, diabetes, hypertension, HF, AF, and use of anti-hypertensive medications. After backwards selection, the final clinical-only SCD risk score included age, gender, race, CHD, stroke, diabetes, and hypertension. The other covariates were removed from the score due to lack of statistical significance.

The combined clinical+GEH score was developed using covariates from the clinical-only score, all 5 GEH parameters, and all significant interaction terms. Backwards selection was then performed with a cut-off p-value of 0.10. The final clinical+GEH model included age, gender, race, CHD, stroke, diabetes, hypertension, SAI QRST, spatial QRS-T angle, SVG elevation, and

interaction terms (SAI QRST*age, QRS-T angle*age, QRS-T angle*race, QRS-T angle*diabetes, QRS-T angle*hypertension, and SVG azimuth*gender). Weighting of each variable's contribution to SCD risk was determined by relative size of effect estimates. A cumulative incidence function was used to assign 10-year SCD risk to each participant based on their individual clinical-only and clinical+GEH risk scores as described in the Supplemental Methods. Cut-off points for "low-" and "high-risk" were selected based on reasonable levels of 10-year SCD risk.

After the clinical-only and clinical+GEH risk scores were generated, 10-year SCD risk was assigned to each participant using the cumulative incidence function (CIF):

$$CIF(t \mid x) = 1 - (1 - CIF(t|0))^{\exp(xbeta)}$$

In this equation, *t* represents time since enrollment, *x* represents covariates included in each risk score model, and *beta* represents coefficient estimates from the risk score models (see Table 3). The final equation for estimated 10-year risk of SCD based on the clinical+GEH risk score was:

1 - 0. 999966^exp[(0.7066*Age) – (0.9851*Female) –(1.0180*White) + (1.1734*DM) + (0.8452*HTN) + (1.1298*CHD) + (0.6525*Stroke) + (0.9126*SAI QRST) + (0.3923*QRS-T) + (0.1199*SVGel) – (0.0566*QRS-T*age) + (0.0908*QRS-T*White) – (0.0534*QRS-T*DM) – (0.0481*QRS-T*HTN) + (0.0882*SVGaz*Female) – (0.1030*SAI QRST*Age)]

Where age is per 10 years; SAI QRST is per 100mV*ms; QRS-T angle is per 10°; SVGel is square root transformed SVG elevation (in degrees); SVGaz is square root transformed absolute value of SVG azimuth (in degrees). An interactive risk score calculator is available in the supplemental online files and at http://www.ecgpredictscd.org/.

Risk score performance:

Discrimination:

C-statistics were computed for each risk score in entirety and over the 2% subinterval of the false positive fraction, as a false positive fraction <2% is considered a useful threshold for screening healthy individuals[29].

Calibration:

We compared how closely the predicted probabilities of 10-year SCD using competing risks models agreed numerically with observed SCD outcomes over 20 equal sized categories.

Goodness of Fit:

Overall model fit for the 2 risk scores was compared using the Akaike Information Criterion (AIC) which takes into account both the statistical goodness of fit and the number of parameters required to achieve this particular degree of fit by imposing a penalty for increasing the number of parameters.

Stratification capacity:

Stratification capacity of the risk score was assessed using a risk stratification table. We defined 10-year SCD risk categories as low (<0.5%), moderate (1-5%), and high (>5%). We additionally evaluated a "high-risk" threshold of 10% and a "low-risk" threshold of 1%. We used the risk stratification table to estimate classification accuracy and reclassification improvement rates as the extent to which participants with SCD events were appropriately or inappropriately assigned/reassigned to high-, intermediate- and low-risk categories with the addition of GEH ECG parameters.

Prediction model internal cross-validation:

Internal 5-fold cross-validation of the clinical+GEH risk score was performed in order to evaluate its ability to fit out-of-sample data. The data were split randomly into 5 partitions. For each partition the entire process creating the prediction model was repeated. The clinical+GEH risk score was fit using 4 of the groups, and the resulting parameters were used to predict the SCD in the remaining group. Metrics used to determine risk score performance were recalculated each time the data were split and re-validated.

Supplemental Table 1: Associations of baseline clinical and ECG characteristics with SAI QRST, spatial QRS-T angle, and SVG magnitude

Characteristic	SAI QRST, mV*ms Difference (95% CI)	P-Value	QRS-T Angle, ° Difference (95% CI)	P-Value	SVG Magnitude, μV Difference (95% CI)	P-Value
Age, per 1 SD increase	+2.25 (0.94-3.56)	0.001	+3.54(2.82–4.26)	<0.0001	-22.67(-34.49 to -10.84)	<0.0001
Female	-32.34 (-33.83 to -30.85)	<0.0001	-16.04(-16.85 to -15.22)	<0.0001	-62.46(-75.90 to -49.02)	<0.0001
White	-12.47 (-16.66 to -8.28)	<0.0001	-1.44(-3.74 to 0.86)	0.22	-226.31 (-264.05 to -188.56)	<0.0001
Diabetes	-3.19 (-5.42 to -0.96)	0.005	+7.83 (6.61-9.05)	<0.0001	-102.84 (-122.89 to -82.78)	<0.0001
Hypertension	+7.47 (5.87-9.08)	<0.0001	+4.72 (3.85-5.61)	<0.0001	+57.80 (43.36-72.24)	<0.0001
Anti-hypertensive medications	+5.92 (4.31-7.53)	<0.0001	+3.43 (2.55-4.31)	<0.0001	+7.76 (-6.74 to 22.27)	0.29
Coronary heart disease	+16.97 (14.26-19.68)	<0.0001	+15.88 (14.40-17.36)	<0.0001	-37.16 (-61.66 to -12.67)	0.003
Heart failure	+15.00 (11.44-18.56)	<0.0001	+14.23 (12.29-16.18)	<0.0001	-41.43 (-73.56 to -9.30)	0.011
Stroke	+15.60 (10.68-20.52)	<0.0001	+11.14 (8.44-13.84)	<0.0001	+50.08 (5.71 to 94.45)	0.027
Atrial fibrillation	+3.01 (-4.84 to 10.85)	0.45	+28.67 (24.38-32.95)	<0.0001	-31.24 (-101.91 to 39.44)	0.39
Current smoking	+3.94 (1.97-5.91)	<0.0001	+4.17 (3.09-5.25)	<0.0001	-19.61 (-37.38 to -1.85)	0.030
Body mass index, per 1SD increase	-10.54 (-11.29 to -9.79)	<0.0001	-1.00 (-1.42 to -0.59)	<0.0001	-114.24 (-120.91 to -107.56)	<0.0001
Moderate physical activity vs. inactive	-2.94 (-8.08 to 2.20)	0.26	-5.85 (-8.67 to -3.03)	<0.0001	+30.40 (-15.92 to 76.72)	0.20
Total cholesterol, per 1SD increase	-2.38 (-3.13 to -1.63)	<0.0001	-0.21 (-0.62 to 0.20)	0.32	-20.01 (-26.76 to -13.27)	<0.0001
HDL cholesterol, per 1SD increase	+5.39 (4.59-6.20)	<0.0001	-0.46 (-0.90 to -0.02)	0.041	+63.52 (56.32-70.73)	<0.0001
Triglycerides, per 1SD increase	-4.50 (5.24 to –3.76)	<0.0001	+0.81 (0.40-1.22)	<0.0001	-45.51 (-52.20 to -38.81)	<0.0001
Use of β-blockers	+8.37 (5.89-10.86)	<0.0001	-3.99 (-5.35 to -2.63)	<0.0001	+68.49 (46.13-90.86)	<0.0001
Alcohol consumption, per 1SD increase	+0.91 (0.16-1.67)	0.018	+0.05 (-0.37 to 0.46)	0.83	+14.60 (7.80-21.40)	<0.0001
Creatinine, per 1SD increase	+3.01 (2.23-3.78)	<0.0001	+1.46 (1.04-1.89)	<0.0001	+6.50 (-0.49 to 13.48)	0.068
Abnormal LVEF*	+81.79 (72.29-91.29)	<0.0001	+36.73 (31.70-41.75)	<0.0001	+60.84 (-16.72 to 138.41)	0.12
Heart rate, per 1SD increase	-8.79 (-9.53 to -8.05)	<0.0001	+4.31 (3.90-4.71)	<0.0001	-54.83 (-61.52 to -48.14)	<0.0001
QTc, per 1SD increase	+9.71 (8.96-10.46)	<0.0001	+4.98 (4.56-5.39)	<0.0001	-25.62 (-32.48 to -18.76)	<0.0001
QRS duration, per 1SD increase	+25.49 (24.80-26.19)	<0.0001	+10.01 (9.60-10.41)	<0.0001	+11.24 (4.20-18.28)	0.002
BBB/IVCD	+35.42 (33.13-37.71)	<0.0001	+13.39 (12.12-14.66)	<0.0001	-32.78 (-53.87 to -11.68)	0.002
Sex-adjusted Cornell Product, per 1SD	+26.69 (26.03-27.36)	<0.0001	+13.39 (13.02-13.77)	<0.0001	+26.41 (19.55-33.27)	<0.0001
Ventricular Pacing	+160.0 (144.8-175.2)	<0.0001	+66.0 (57.59-74.35)	<0.0001	-236.2 (-374.5 to -97.86)	0.001

Adjusted for age, gender, race, cohort, and location (n=20,177)

*LVEF data only available in CHS (n=4,954)

Abbreviations: HDL:high density lipoprotein; LVEF:left ventricular ejection fraction; BBB:bundle branch block; IVCD:intraventricular conduction delay; SD=standard deviation

Supplemental Table 2: Associations of baseline clinical and ECG characteristics with square root transformed SVG elevation and SVG azimuth, adjusted for demographic characteristics.

Characteristic	SQRT SVG Elevation, °^1/2 Difference (95% CI)	P-Value	SQRT SVG Azimuth, °^1/2 Difference (95% CI)	P-Value
Age, per 1SD increase	+0.21 (0.19-0.24)	<0.0001	+0.07 (0.02-.012)	0.011
Female	-0.30 (-0.32 to -0.27)	<0.0001	-0.06 (-0.11 to 0.002)	0.058
White	-0.38 (0.47 to -0.30)	<0.0001	-0.05 (-0.21 to 0.12)	0.57
Diabetes	+0.15 (0.10-0.19)	<0.0001	+0.32 (0.24-0.41)	<0.0001
Hypertension	+0.21 (0.18-0.24)	<0.0001	+0.21 (0.15-0.27)	<0.0001
Anti-hypertensive medications	+0.19 (0.16-0.23)	<0.0001	+0.26 (0.20-0.32)	<0.0001
Coronary heart disease	+0.20 (0.14-0.25)	<0.0001	+0.89 (0.79-1.00)	<0.0001
Heart failure	+0.18 (0.11-0.25)	<0.0001	+0.75 (0.62-0.89)	<0.0001
Stroke	+0.08 (-0.01 to 0.18)	0.087	+0.52 (0.33-0.71)	<0.0001
Atrial fibrillation	+0.09 (-0.06 to 0.24)	0.26	+1.17 (0.87-1.47)	<0.0001
Current smoking	-0.21 (-0.25 to -0.17)	<0.0001	+0.36 (0.29-0.44)	0.013
Body mass index, per 1SD increase	+0.17 (0.16-0.19)	<0.0001	-0.09 (-0.12 to -0.06)	<0.0001
Moderate physical activity vs. inactive	+0.16 (-0.08 to 0.12)	0.76	-0.21 (-0.41 to -0.01)	0.038
Total cholesterol, per 1SD increase	-0.004 (-0.02 to 0.01)	0.64	-0.04 (-0.07 to -0.01)	0.015
HDL cholesterol, per 1SD increase	-0.08 (-0.10 to -0.07)	<0.0001	+0.01 (-0.02 to 0.04)	0.54
Triglycerides , per 1SD increase	+0.06 (0.05-0.08)	<0.0001	+0.03 (0.003-0.06)	0.030
Use of β-blockers	+0.11 (0.06-0.16)	<0.0001	+0.12 (0.03-0.22)	0.013
Alcohol consumption, per 1SD increase	-0.01 (-0.03 to 0.002)	0.090	+0.03 (-0.003 to 0.06)	0.082
Creatinine, per 1SD increase	+0.02 (-0.0005 to 0.03)	0.057	+0.04 (0.01-0.07)	0.008
Abnormal LVEF*	+0.44 (0.27-0.60)	<0.0001	+1.84 (1.50-2.18)	<0.0001
Heart rate, per 1SD increase	-0.03 (-0.04 to -0.01)	0.001	+0.06 (0.04-0.09)	<0.0001
QTc , per 1SD increase	+0.13 (0.11-0.14)	<0.0001	+0.42 (0.40-0.45)	<0.0001
QRS duration, per 1SD increase	+0.16 (0.15-0.18)	<0.0001	+0.88 (0.85-0.90)	<0.0001
BBB/IVCD	+0.22 (0.17-0.26)	<0.0001	+1.43 (1.34-1.52)	<0.0001
Sex-adjusted Cornell Product, per 1SD	+0.33 (0.32-0.35)	<0.0001	+0.84 (0.81-0.87)	<0.0001
Ventricular pacing	+2.2 (1.9-2.5)	<0.0001	+3.45 (2.86-4.04)	<0.0001

Adjusted for age, gender, race, cohort, and location (n=20,177)

*LVEF data was only available in CHS (n=4,954)

Abbreviations: HDL: high density lipoprotein; LVEF: left ventricular ejection fraction; BBB: bundle branch block; IVCD: intraventricular conduction delay; SD=standard deviation

Supplemental Table 3: Unadjusted associations of baseline clinical and ECG characteristics with spatial ventricular gradient (SVG) azimuth and elevation analyzed as circular statistics

Characteristic	SVG Azimuth, °	P-Value	SVG Elevation, °	P-Value
Age	0.103	<0.0001	0.250	<0.0001
Female vs. Male	25.17 (21.95) vs. 22.64 (26.86)	<0.0001	67.71 (16.94) vs. 72.47 (20.15)	<0.0001
White vs. Black	24.07 (24.61) vs. 24.38 (22.72)	<0.0001	68.84 (18.82) vs. 72.99 (17.30)	<0.0001
Diabetes; no vs. yes	23.77 (23.85) vs. 26.83 (26.22)	<0.0001	69.18 (18.27) vs. 73.94 (20.00)	<0.0001
Hypertension; no vs. yes	23.27 (23.65) vs. 25.45 (24.89)	<0.0001	67.17 (18.02) vs. 73.60 (18.70)	<0.0001
Anti-hypertensive meds; no vs. yes	23.18 (23.52) vs. 26.04 (25.30)	<0.0001	67.91 (18.11) vs. 73.33 (18.91)	<0.0001
CHD; no vs. yes	23.63 (23.28) vs. 31.60 (32.45)	<0.0001	69.10 (18.10) vs. 77.19 (21.66)	<0.0001
Heart failure; no vs. yes	23.81 (23.93) vs. 32.31 (28.20)	<0.0001	69.61 (18.42) vs. 73.69 (21.18)	<0.0001
Stroke; no vs. yes	24.05 (24.02) vs. 28.82 (30.59)	<0.0001	69.66 (18.52) vs. 75.32 (19.80)	<0.0001
Atrial Fibrillation; no vs. yes	24.03 (24.08) vs. 40.13 (30.87)	<0.0001	69.72 (18.51) vs. 77.58 (22.61)	<0.0001
Smoking; never vs. current	23.34 (23.13) vs. 27.59 (24.21)	<0.0001	70.39 (17.93) vs. 66.45 (18.16)	<0.0001
BMI, kg/m^2	0.039	<0.0001	0.159	<0.0001
Moderate physical activity vs. inactive	24.21 (23.73) vs. 23.43 (30.22)	<0.0001	68.86 (18.05) vs. 77.19 (21.28)	<0.0001
Total cholesterol, mg/dL	0.015	0.01	0.032	<0.0001
HDL, mg/dl	0.052	<0.0001	0.086	<0.0001
Triglycerides , mg/dl	0.032	<0.0001	0.060	<0.0001
Beta-blockers; no vs yes	24.01 (24.01) vs. 25.40 (25.71)	<0.0001	69.50 (18.63) vs. 72.40 (17.78)	<0.0001
Alcohol Consumption, g/wk	0.017	0.002	0.010	0.11
Creatinine, mg/dL	0.038	<0.0001	0.062	<0.0001
LVEF; normal vs. abnormal*	21.11 (26.67) vs. 38.98 (34.97)	<0.0001	75.42 (20.08) vs. 82.99 (22.21)	<0.0001
Heart rate, bpm	0.029	<0.0001	0.067	<0.0001
QTc, ms	0.181	<0.0001	0.166	<0.0001
QRS Duration, ms	0.361	<0.0001	0.289	<0.0001
BBB/IVCD; no vs. yes	23.40 (22.07) vs. 35.00 (38.71)	<0.0001	69.21 (17.22) vs. 74.49 (26.50)	<0.0001
Sex-adjusted Cornell Product, mV*ms	0.389	<0.0001	0.338	<0.0001
Ventricular pacing; no vs yes	69.69 (18.36) vs. 125.72 (34.06)	<0.0001	24.13 (24.12) vs. 48.03 (50.03)	<0.0001

SVG elevation and azimuth data are presented as mean (circular SD) for subgroups comparison. Circular-linear correlation coefficient r is reported for continuous variables.
P-value per Watson U-square statistic and Kuiper statistics (both were in agreement)
*Data on LVEF was only available in the Cardiovascular Health Study (n=4,953)

Abbreviations: CHD = coronary heart disease; BMI = body mass index; HDL = high density lipoprotein; LVEF = left ventricular ejection fraction; BBB = bundle branch block; IVCD = intraventricular conduction delay

Supplemental Table 4: Associations of measures of global electrical heterogeneity with SCD, per 1 SD of predictor in competing risks models

Merged	**SAI QRST**		**QRS-T Angle**		**SVG Magnitude**		**SVG Elevation**		**SVG Azimuth**	
	SHR (95%CI)	**P value**	**SHR (95%CI)**	**P value**	**SHR (95%CI)**	**P value**	**SHR (95%CI)**	**P value**	**SHR (95%CI)**	**P value**
Model 1	1.22 (1.16-1.28)	<0.0001	1.46 (1.34-1.60)	<0.0001	1.02 (0.94-1.12)	0.625	1.24 (1.13-1.35)	<0.0001	1.25 (1.14-1.36)	<0.0001
Model 2	1.17 (1.11-1.24)	<0.0001	1.23 (1.13-1.34)	<0.0001	1.07 (0.98-1.17)	0.114	1.18 (1.07-1.28)	<0.0001	1.11 (1.02-1.21)	0.016
Model 3	1.12 (1.04-1.20)	0.004	1.17 (1.06-1.29)	0.002	1.06 (0.98-1.16)	0.164	1.12 (1.02-1.22)	0.018	1.01 (0.91-1.11)	0.885
Model 4	1.13 (1.04-1.23)	0.003	1.26 (1.13-1.40)	<0.0001	1.11 (1.02-1.22)	0.020	1.03 (0.94-1.12)	0.52	1.05 (0.94-1.16)	0.39
ARIC										
Model 1	1.31 (1.21-1.41)	<0.0001	1.64 (1.49-1.81)	<0.0001	0.99 (0.87-1.11)	0.824	1.30 (1.17-1.45)	<0.0001	1.27 (1.12-1.44)	<0.0001
Model 2	1.20 (1.09-1.33)	<0.0001	1.32 (1.20-1.46)	<0.0001	1.05 (0.93-1.19)	0.393	1.19 (1.06-1.33)	0.004	1.09 (0.97-1.23)	0.126
Model 3	1.14 (1.02-1.27)	0.020	1.25 (1.11-1.41)	<0.0001	1.02 (0.90-1.15)	0.744	1.12 (0.99-1.26)	0.061	0.98 (0.86-1.12)	0.776
Model 4	1.06 (0.97-1.17)	0.204	1.35 (1.19-1.53)	<0.0001	1.05 (0.93-1.18)	0.464	1.06 (0.95-1.19)	0.279	1.07 (0.94-1.23)	0.304
CHS										
Model 1	1.21 (1.12-1.32)	<0.0001	1.29 (1.10-1.51)	0.001	1.09 (0.96-1.24)	0.190	1.17 (1.01-1.35)	0.035	1.27 (1.11-1.45)	0.001
Model 2	1.17 (1.07-1.28)	0.001	1.12 (0.96-1.32)	0.154	1.11 (0.97-1.26)	0.133	1.14 (0.99-1.32)	0.063	1.14 (1.00-1.31)	0.051
Model 3	1.09 (0.95-1.25)	0.216	1.03 (0.85-1.23)	0.785	1.12 (0.98-1.27)	0.103	1.07 (0.93-1.23)	0.343	1.01 (0.86-1.19)	0.920
Model 4	1.21 (1.06-1.38)	0.004	1.08 (0.90-1.30)	0.427	1.19 (1.04-1.36)	0.012	0.98 (0.87-1.11)	0.803	0.97 (0.83-1.14)	0.693
Sensitivity Analysis for LVEF in CHS Participants*										
No LVEF*	1.22 (1.05-1.40)	0.005	1.07 (0.88-1.30)	0.495	1.19 (1.04-1.36)	0.013	1.00 (0.88-1.14)	0.971	0.96 (0.81-1.13)	0.622
with LVEF†	1.21 (1.05-1.39)	0.009	1.05 (0.88-1.28)	0.593	1.19 (1.04-1.37)	0.014	1.00 (0.88-1.14)	0.966	0.97 (0.83-1.14)	0.729

Model 1-adjusted for age, sex, race, study center, and cohort.
Model 2-further adjusted for CHD, HF, stroke, AF, use of beta-blockers, creatinine, traditional cardiovascular risk factors (BMI, hypertension, anti-hypertensive medications, diabetes, smoking, alcohol intake, total cholesterol, HDL cholesterol, triglycerides, and physical activity index).
Model 3-further adjusted for ECG characteristics: heart rate, corrected QT interval, QRS duration, Cornell product, and bundle branch block or intraventricular conduction delay
Model 4-incorporated time-updated ECG measurements and time-updated incident non-fatal cardiovascular events (AF, HF, CHD, stroke)
Sensitivity analysis is restricted to 4,954 patients in CHS with baseline LVEF data available

SD=standard deviation; CHD=coronary heart disease; HF=heart failure; AF=atrial fibrillation; LVEF=left ventricular ejection fraction

Supplemental Table 5: Association of time with GEH ECG parameters in mixed effect models

GEH ECG Parameter	β-coefficient (95% CI)	P-value
SAI QRST, mV*ms	+0.63 (0.51 to 0.76)	<0.0001
Spatial QRS-T angle, degrees	+0.36 (0.29 to 0.42)	<0.0001
SVG Magnitude, μV	-4.02 (-5.05 to -3.00)	<0.0001
Square root of SVG elevation	+0.035 (0.032 to 0.037)	<0.0001
Square root of absolute SVG Azimuth	+0.008 (0.003 to 0.012)	0.001

All models are mixed effect models (participant nested within study center nested within cohort), adjusted by age, sex, and race.

β-coefficient shows change in corresponding GEH ECG parameter over each time interval of median 2.8 (interquartile range 1.0-3.2) years of follow-up.

Supplemental Table 6A: Association of sudden increase in GEH ECG parameters (SAI QRST, SVG Magnitude, SVG Elevation, and SVG Azimuth) at visits 2 or 3, as compared to baseline study visit, with sudden cardiac death.

		SAI QRST			SVG Magnitude			SVG Elevation			SVG Azimuth		
	% Increase	**N**	**HR (95%CI)**	**P value**	**N**	**HR (95%CI)**	**P value**	**N**	**HR (95%CI)**	**P value**	**N**	**HR (95%CI)**	**P value**
Model 1	**↓ ≥33%**	425	1.14 (0.62-2.10)	0.66	452	0.85 (0.43-1.65)	0.62	103	1.20 (0.39-3.76)	0.75	4,899	0.48 (0.35-0.65)	<0.0001
	↓ 25-32%	1,160	0.80 (0.50-1.28)	0.35	780	1.00 (0.62-1.62)	0.99	152	1.41 (0.59-3.41)	0.45	1,132	0.72 (0.45-1.15)	0.17
	↑↓ 0-24%	14,384	Reference	--	15,646	Reference	--	17,011	Reference	--	5,133	Reference	--
	↑ 25-32%	977	1.54 (1.04-2.29)	0.037	647	1.27 (0.79-2.05)	0.32	518	0.77 (0.38-1.54)	0.46	766	0.56 (0.30-1.04)	0.065
	↑ 33-49%	902	1.92 (1.34-2.76)	0.001	533	2.88 (2.01-4.15)	<0.0001	395	1.02 (0.51-2.06)	0.95	1,116	0.85 (0.56-1.30)	0.46
	↑ ≥50%	2,329	3.85 (3.12-4.75)	<0.0001	2,119	3.09 (2.48-3.86)	<0.0001	1,998	3.27 (2.63-4.07)	<0.0001	7,131	1.26 (1.01-1.56)	0.042
Model 2	**↓ ≥33%**	425	1.10 (0.60-2.02)	0.76	452	0.75 (0.38-1.46)	0.39	103	1.01 (0.32-3.18)	0.98	4,899	0.61 (0.45-0.82)	0.001
	↓ 25-32%	1,160	0.80 (0.50-1.28)	0.35	780	0.89 (0.55-1.43)	0.62	152	1.31 (0.54-3.19)	0.55	1,132	0.87 (0.54-1.39)	0.56
	↑↓ 0-24%	14,384	Reference	--	15,646	Reference	--	17,011	Reference	--	5,133	Reference	--
	↑ 25-32%	977	1.40 (0.94-2.08)	0.10	647	1.14 (0.71-1.84)	0.59	518	0.72 (0.36-1.45)	0.36	766	0.61 (0.63-1.48)	0.11
	↑ 33-49%	902	1.64 (1.14-2.37)	0.008	533	2.38 (1.65-3.43)	<0.0001	395	0.95 (0.47-1.92)	0.89	1,116	0.97 (0.63-1.48)	0.88
	↑ ≥50%	2,329	2.97 (2.39-3.70)	<0.0001	2,119	2.62 (2.09-3.27)	<0.0001	1,998	2.75 (2.20-3.43)	<0.0001	7,131	1.37 (1.10-1.70)	0.005
Model 3	**↓ ≥33%**	425	1.23 (0.67-2.27)	0.50	452	0.73 (0.36-1.48)	0.38	103	1.34 (0.43-4.19)	0.62	4,899	0.75 (0.55-1.04)	0.085
	↓ 25-32%	1,160	0.76 (0.45-1.26)	0.28	780	0.88 (0.53-1.47)	0.63	152	1.08 (0.40-2.90)	0.88	1,132	1.09 (0.68-1.77)	0.71
	↑↓ 0-24%	14,384	Reference	--	15,646	Reference	--	17,011	Reference	--	5,133	Reference	--
	↑ 25-32%	977	1.37 (0.89-2.10)	0.15	647	1.02 (0.61-1.70)	0.94	518	0.50 (0.21-1.21)	0.13	766	0.60 (0.31-1.19)	0.15
	↑ 33-49%	902	1.57 (1.07-2.31)	0.021	533	2.45 (1.67-3.59)	<0.0001	395	1.06 (0.52-2.14)	0.87	1,116	1.04 (0.66-1.65)	0.87
	↑ ≥50%	2,329	2.61 (2.07-3.31)	<0.0001	2,119	2.36 (1.86-3.01)	<0.0001	1,998	2.43 (1.91-3.08)	<0.0001	7,131	1.66 (1.30-2.11)	<0.0001
Model 4	**↓ ≥33%**	425	1.04 (0.56-1.93)	0.90	452	0.72 (0.35-1.47)	0.37	103	1.42 (0.45-4.45)	0.55	4,899	0.80 (0.57-1.11)	0.18
	↓ 25-32%	1,160	0.69 (0.42-1.16)	0.16	780	0.88 (0.53-1.46)	0.61	152	1.06 (0.39-2.86)	0.90	1,132	1.10 (0.68-1.78)	0.70
	↑↓ 0-24%	14,384	Reference	--	15,646	Reference	--	17,011	Reference	--	5,133	Reference	--
	↑ 25-32%	977	1.42 (0.93-2.19)	0.11	647	1.09 (0.66-1.83)	0.73	518	0.54 (0.22-1.30)	0.17	766	0.62 (0.31-1.23)	0.17
	↑ 33-49%	902	1.65 (1.12-2.43)	0.011	533	2.74 (1.86-4.05)	<0.0001	395	1.14 (0.56-2.32)	0.71	1,116	1.08 (0.68-1.72)	0.74
	↑ ≥50%	2,329	2.64 (2.09-3.34)	<0.0001	2,119	2.49 (1.95-3.17)	<0.0001	1,998	2.50 (1.97-3.17)	<0.0001	7,131	1.79 (1.38-2.34)	<0.0001

Supplemental Table 6B: Association of sudden increase in GEH ECG parameters (QRS-T Angle) at visits 2 or 3, as compared to baseline study visit, with sudden cardiac death.

QRS-T Angle				
	% Increase	**N**	**HR (95%CI)**	**P value**
Model 1	**↓≥300%**	344	1.50 (0.77-2.91)	0.23
	↓ 50-299%	795	0.82 (0.46-1.46)	0.50
	↑↓ 0-49%	14,202	Reference	--
	↑50-200%	1,944	1.13 (0.81-1.58)	0.47
	↑201-299%	764	1.89 (1.26-2.82)	0.002
	↑≥300%	2,128	3.57 (2.86-4.45)	<0.0001
Model 2	**↓≥300%**	344	1.30 (0.67-2.53)	0.44
	↓ 50-299%	795	0.81-0.46-1.45)	0.49
	↑↓ 0-49%	14,202	Reference	--
	↑50-200%	1,944	1.01 (0.72-1.41)	0.95
	↑201-299%	764	1.62 (1.08-2.43)	0.021
	↑≥300%	2,128	2.89 (2.31-3.62)	<0.0001
Model 3	**↓≥300%**	344	0.86 (0.38-1.96)	0.72
	↓ 50-299%	795	0.97 (0.53-1.68)	0.84
	↑↓ 0-49%	14,202	Reference	--
	↑50-200%	1,944	1.07 (0.76-1.51)	0.71
	↑201-299%	764	1.78 (1.17-2.70)	0.007
	↑≥300%	2,128	2.72 (2.13-3.45)	<0.0001
0Model 4	**↓≥300%**	344	0.82 (0.63-1.87)	0.64
	↓ 50-299%	795	0.91 (0.51-1.64)	0.76
	↑↓ 0-49%	14,202	Reference	--
	↑50-200%	1,944	1.32 (0.92-1.89)	0.13
	↑201-299%	764	2.53 (1.63-3.93)	<0.0001
	↑≥300%	2,128	3.05 (2.39-3.88)	<0.0001

All models are Cox regression models.

Model 1-adjusted for age, sex, race, study center, and cohort.
Model 2-further adjusted for CHD, HF, stroke, AF, use of beta-blockers, creatinine, traditional cardiovascular risk factors (BMI, hypertension, anti-hypertensive medications, diabetes, smoking, alcohol intake, total cholesterol, HDL cholesterol, triglycerides, and physical activity index).
Model 3-further adjusted for ECG characteristics: heart rate, corrected QT interval, QRS duration, Cornell product, presence of bundle branch block or intraventricular conduction delay, ventricular pacing, and incident non-fatal cardiovascular events (AF, HF, CHD, stroke)
Model 4 further adjusted for baseline (at visit 1) value of predictor

Supplemental Table 7: Interactions of GEH ECG parameters with SCD, per 1 SD of predictor

	SAI QRST		QRS-T Angle		SVG Magnitude		Sqrt SVG Elevation		Sqrt SVG Azimuth	
	HR (95%CI)	$P_{Interaction}$	HR (95%CI)	$P_{Interaction}$	HR (95%CI)	$P_{Interaction}$	HR (95%CI)	$P_{Interaction}$	HR (95%CI)	$P_{Interaction}$
Model 3 (All)	**1.16 (1.07-1.25)**		**1.21 (1.10-1.32)**		**1.09 (1.00-1.19)**		**1.11 (1.02-1.22)**		**1.01 (0.92-1.11)**	
Age, year	0.992 (0.986-0.997)	0.003	0.983 (0.975-0.990)	<0.0001	1.00 (1.00-1.01)	0.473	0.994 (0.99-1.00)	0.142	0.992 (0.985-1.000)	0.054
Race	--	0.064	--	0.039	--	0.605	--	0.856	--	0.996
White (n= 15,590)	1.24 (1.13-1.37)		1.29 (1.15-1.44)		1.14 (1.02-1.28)		1.11 (1.01-1.23)		1.00 (0.89-1.11)	
Black (n= 4,587)	1.03 (0.90-1.18)		0.96 (0.81-1.14)		1.02 (0.89-1.18)		1.17 (0.98-1.39)		1.02 (0.85-1.23)	
Gender	--	0.024	--	0.400	--	0.108	--	0.260	--	0.019
Male (n= 8,903)	**1.13 (1.03-1.24)**		1.23 (1.09-1.38)		1.05 (0.94-1.17)		1.12 (1.01-1.25)		0.97 (0.86-1.09)	
Female (n= 11,274)	1.25 (1.08-1.45)		1.15 (0.98-1.35)		1.19 (1.02-1.39)		1.09 (0.93-1.27)		1.14 (0.97-1.35)	
Diabetes	--	0.168	--	0.049	--	0.339	--	0.674	--	0.083
Yes (n=2,613)	1.12 (0.98-1.30)		1.13 (0.96-1.33)		1.00 (0.85-1.18)		1.23 (1.06-1.43)		0.96 (0.81-1.13)	
No (n= 17,564)	1.19 (1.08-1.31)		1.23 (1.10-1.38)		1.14 (1.03-1.27)		1.05 (0.94-1.17)		1.06 (0.94-1.19)	
HTN	--	0.078	--	0.009	--	0.935	--	0.932	--	0.332
Yes (n= 8,250)	1.11 (1.01-1.22)		1.13 (1.00-1.26)		1.07 (0.96-1.19)		1.12 (1.00-1.25)		0.99 (0.88-1.11)	
No (n= 11,927)	1.34 (1.14-1.57)		1.33 (1.13-1.56)		1.15 (0.98-1.35)		1.09 (0.95-1.26)		1.04 (0.89-1.23)	
CHD	--	0.135	--	0.092	--	0.060	--	0.145	--	0.595
Yes (n=1,762)	1.20 (1.07-1.34)		1.41 (1.23-1.62)		1.05 (0.91-1.23)		1.04 (0.90-1.19)		1.06 (0.91-1.23)	
No (n=18,415)	1.21 (1.07-1.36)		1.02 (0.90-1.16)		1.18 (1.05-1.32)		1.10(0.99-1.24)		0.96 (0.85-1.09)	
Heart Failure	--	0.97	--	0.942	--	0.239	--	0.506	--	0.210
Yes (n = 914)	1.03 (0.82-1.30)		1.08 (0.83-1.40)		1.01 (0.76-1.35)		1.11 (0.84-1.45)		1.11 (0.83-1.49)	
No (n=19,263)	1.17 (1.08-1.27)		1.22 (1.10-1.35)		1.11 (1.01-1.22)		1.13 (1.03-1.24)		1.00 (0.91-1.11)	
BMI, kg/m^2	1.014 (1.002-1.027)	0.018	0.999 (0.984-1.013)	0.829	1.007 (0.991-1.023)	0.400	0.997 (0.980-1.013)	0.697	1.001 (0.985-1.017)	0.928
HTN Meds	--	0.003	--	0.231	--	0.031	--	0.261	--	0.379
Yes (n= 7,030)	1.14 (1.03-1.26)		1.24 (1.11-1.39)		1.02 (0.91-1.15)		1.09 (0.97-1.22)		1.02 (0.90-1.16)	
No (n= 13,147)	1.21 (1.07-1.38)		1.12 (0.96-1.32)		1.22 (1.07-1.40)		1.15 (1.00-1.32)		0.99 (0.85-1.15)	
QRS Duration	0.998 (0.996-0.999)	0.009	0.997 (0.994-1.000)	0.084	0.999 (0.996-1.002)	0.678	0.997 (0.994-0.999)	0.035	0.997 (0.993-1.000)	0.068
Cornell Product	0.99996 (0.99993-0.99999)	0.010	0.99990 (0.99985-0.99997)	0.004	0.999998 (0.99995-1.00004)	0.987	0.99990 (0.99982-0.99998)	0.016	0.99992 (0.99984-0.99999)	0.036
BBB/IVCD		0.078		0.502		0.788		0.306		0.259
Yes (n= 2,328)	1.15(1.01-1.31)		1.13(0.93-1.37)		1.12(0.92-1.35)		1.07(0.90-1.26)		0.96(0.78-1.20)	
No (n= 17,849)	1.20(1.08-1.34)		1.23(1.10-1.37)		1.09(0.98-1.20)		1.16(1.04-1.29)		1.04(0.94-1.17)	
Ventricular Pacing	--	0.327	--	0.079	--	0.054	--	0.521	--	0.236
Yes* (n=47)	0.92(0.60-1.41)		0.81(0.44-1.48)		2.03(0.85-4.84)		1.17(0.57-2.40)		0.78(0.34-1.79)	
No* (n=20,130)	1.25 (1.19-1.32)		1.55 (1.43-1.68)		1.03 (0.94-1.12)		1.24 (1.14-1.35)		1.30 (1.19-1.41)	

Adjusted for Model 3 variables: age, sex, race, study center, cohort, CHD, heart failure, stroke, atrial fibrillation, use of beta-blockers, creatinine, BMI, HTN, anti-hypertensive medications, diabetes, smoking, alcohol intake, total cholesterol, HDL cholesterol, triglycerides, physical activity index, heart rate, corrected QT interval, QRS duration, Cornell product, and bundle branch block or intraventricular conduction delay

* Minimally adjusted model (by age, sex, race, cohort, and study center) due to small size of VP subgroup

Abbreviations: HTN: hypertension; CHD: coronary heart disease; BMI: body-mass-index; SD: standard deviation

Supplemental Table 8: Optimal cut-off points for GEH ECG parameters stratified by sex and race

GEH ECG Parameter	WM (N=7,155)	WF (N=8,435)	BM (N=1,748)	BF (N=2,839)
SAI QRST, mV*ms	≥174.1	≥178.3	≥166.1	≥181.5
Spatial QRS-T angle, °	≥89.6	≥70.3	≥89.3	≥57.7
SVG Magnitude, µV	≥858.5	≥2022.7	≥1725.1	≥2335.7
Square root of absolute SVG azimuth, degree$^{1/2}$	≥6.8	≥6.1	≥5.8	≥6.4
Square root of SVG elevation, degree$^{1/2}$	≥8.6	≥8.6	≥8.2	≥8.5

M=males; F=females; W=white; B=black.

Supplemental Table 9: Calibration of SCD risk score: Predicted and observed rate of SCD in 20 equally sized subgroups of participants

Predicted cumulative incidence of SCD			Total Participants, n	Observed SCD	
Mean, %	Min, %	Max, %		SCD, n	SCD rate, %
0.16	0.05	0.22	1009	1	0.099
0.26	0.22	0.30	1009	2	0.198
0.33	0.30	0.37	1009	1	0.099
0.40	0.35	0.44	1009	5	0.496
0.47	0.44	0.51	1009	7	0.694
0.55	0.51	0.59	1008	5	0.496
0.64	0.60	0.68	1009	8	0.793
0.73	0.68	0.78	1009	9	0.892
0.83	0.78	0.89	1009	8	0.793
0.95	0.89	1.01	1009	16	1.586
1.07	1.01	1.13	1008	16	1.587
1.20	1.13	1.28	1009	18	1.784
1.37	1.29	1.47	1009	21	2.081
1.58	1.47	1.71	1009	28	2.775
1.85	1.71	2.01	1009	22	2.180
2.20	2.01	2.39	1008	32	3.175
2.64	2.39	2.94	1009	33	3.271
3.40	2.94	3.98	1009	43	4.262
4.93	3.98	6.24	1009	75	7.433
10.89	6.25	55.51	1009	136	13.479

Supplemental Table 10: Ten-year risk for sudden cardiac death as predicted by the clinical-only and clinical+GEH risk scores using competing risks models, a low-risk cutoff of 1% and a high-risk cutoff of 10%

	10-year risk from **Clinical+GEH** risk score			
	<1%	**1-10%**	**>10%**	**Total**
10-year risk from **Clinical-only** risk score				
<1%				
Participants in this category (% total study cohort)	7,956 (39.43%)	1,158 (5.74%)	4 (0.02%)	9,118 (45.19%)
SCD events (% of all SCD events)	37 (7.61%)	16 (3.29%)	0 (0.00%)	53 (10.91%)
Nonevents (non-SCD) (% all non-SCDs)	7,919 (40.22%)	1,142 (5.80%)	4 (0.02%)	9,065 (46.04%)
Proportion of all deaths due to SCD in this category	3.37%	7.77%	0.00%	4.06%
Non-sudden fatal CHD (% all deaths in category)	222 (20.22%)	63 (30.58%)	0 (0.00%)	285 (21.86%)
Non-CHD death (% all deaths in category)	839 (76.41%)	127 (61.65%)	0 (0.00%)	966 (74.08%)
Proportion of all deaths not due to SCD in this category	94.63%	92.23%	0.00%	95.94%
All-cause death (% total participants in this category)	1,098 (13.80%)	206 (17.79%)	0 (0.00%)	1,304 (14.30%)
1-10%				
Participants in this category (% total study cohort)	2,125 (10.53%)	8,429 (41.78%)	161 (0.80%)	10,715 (53.11%)
SCD events (% of all SCD events)	24 (4.94%)	319 (65.64%)	28 (5.76%)	371 (76.34%)
Nonevents (non-SCD) (% all non-SCDs)	2,101 (10.67%)	8,110 (41.19%)	133 (0.68%)	10,344 (52.53%)
Proportion of all deaths due to SCD in this category	4.49%	7.62%	22.95%	7.66%
Non-sudden fatal CHD (% all deaths in category)	107 (20.00%)	1,418 (33.85%)	54 (44.26%)	1,579 (32.58%)
Non-CHD death (% all deaths in category)	404 (75.51%)	2,452 (58.53%)	40 (32.79%)	2,896 (59.76%)
Proportion of all deaths not due to SCD in this category	95.51%	92.38%	77.05%	92.34%
All-cause death (% total participants in this category)	535 (25.18%)	4,189 (49.70%)	122 (75.78%)	4,846 (45.23%)
>10%				
Participants in this category (% total study cohort)	0 (0.0%)	84 (0.42%)	260 (1.29%)	344 (1.70%)
SCD events (% of all SCD events)	0 (0.0%)	9 (1.85%)	53 (10.91%)	62 (12.76%)
Nonevents (non-SCD) (% all non-SCDs)	0 (0.0%)	75 (0.38%)	207 (1.05%)	282 (1.43%)
Proportion of all deaths due to SCD in this category	0.0%	14.75%	24.42%	22.30%
Non-sudden fatal CHD (% all deaths in category)	0 (0.0%)	25 (40.98%)	88 (40.55%)	113 (40.65%)
Non-CHD death (% all deaths in category)	0 (0.0%)	27 (44.26%)	76 (35.02%)	103 (37.05%)
Proportion of all deaths not due to SCD in this category	0.0%	85.25%	75.58%	77.70%
All-cause death (% total participants in this category)	0 (0.0%)	61 (72.62%)	217 (83.46%)	278 (80.58%)
Total				
Participants in this category (% total study cohort)	10,081 (49.96%)	9,671 (47.93%)	425 (2.11%)	20,177 (100.00%)
SCD events (% of all SCD events)	61 (12.55%)	344 (70.78%)	81 (16.67%)	486 (100.00%)
Nonevents (non-SCD) (% all non-SCDs)	10,020 (50.89%)	9,327 (47.37%)	344 (1.75%)	19,691(100.00%)
Proportion of all deaths due to SCD in this category	3.74%	7.72%	23.89%	7.56%
Non-sudden fatal CHD (% all deaths in category)	329 (20.15%)	1,506 (33.80%)	142 (41.89%)	1,977 (30.76%)
Non-CHD death (% all deaths in category)	1,243 (76.12%)	2,606 (58.48%)	116 (34.22%)	3,965 (61.68%)
Proportion of all deaths not due to SCD in this category	96.26%	92.28%	76.11%	92.44%
All-cause death (% total participants in this category)	1,633 (16.20%)	4,456 (46.08%)	339 (79.76%)	6,428 (31.86%)

Green background: appropriate reclassification; Red background: inappropriate reclassification

Supplemental Table 11: Ten-year risk for sudden cardiac death as predicted by the clinical-only and clinical+GEH risk scores using competing risks models, a low-risk cutoff of 1% and a high-risk cutoff of 5%

	10-year risk from **Clinical+GEH** risk score			
	<1%	**1-5%**	**>5%**	**Total**
10-year risk from **Clinical-only** risk score				
<1%				
Participants in this category (% total study cohort)	7,956 (39.43%)	1,134 (5.62%)	28 (0.14%)	9,118 (45.19%)
SCD events (% of all SCD events)	37 (7.61%)	15 (3.09%)	1 (0.21%)	53 (10.91%)
Nonevents (non-SCD) (% all non-SCDs)	7,919 (40.22%)	1,119 (5.68%)	27 (0.14%)	9,065 (46.04%)
Proportion of all deaths due to SCD in this category	3.37%	7.54%	14.29%	4.06%
Non-sudden fatal CHD (% all deaths in category)	222 (20.22%)	61 (30.65%)	2 (28.57%)	285 (21.86%)
Non-CHD death (% all deaths in category)	839 (76.41%)	123 (61.81%)	4 (57.14%)	966 (74.08%)
Proportion of all deaths not due to SCD in this category	94.63%	92.46%	85.71%	95.94%
All-cause death (% total participants in this category)	1,098 (13.80%)	199 (17.55%)	7 (25.00%)	1,304 (14.30%)
1-5%				
Participants in this category (% total study cohort)	2,125 (10.53%)	7,317 (36.26%)	377 (1.87%)	9,819 (48.66%)
SCD events (% of all SCD events)	24 (4.94%)	228 (46.91%)	48 (9.88%)	300 (61.73%)
Nonevents (non-SCD) (% all non-SCDs)	2,101 (10.67%)	7,089 (36.00%)	329 (1.67%)	9,519 (48.34%)
Proportion of all deaths due to SCD in this category	4.49%	6.65%	18.60%	7.11%
Non-sudden fatal CHD (% all deaths in category)	107 (20.00%)	1,106 (32.28%)	106 (41.09%)	1,319 (31.26%)
Non-CHD death (% all deaths in category)	404 (75.51%)	2,092 (61.06%)	104 (40.31%)	2,600 (61.63%)
Proportion of all deaths not due to SCD in this category	95.51%	93.35%	81.40%	92.89%
All-cause death (% total participants in this category)	535 (25.18%)	3,426 (46.82%)	258 (68.44%)	4,219 (42.97%)
>5%				
Participants in this category (% total study cohort)	0 (0.0%)	201 (1.00%)	1,039 (5.15%)	1,240 (6.15%)
SCD events (% of all SCD events)	0 (0.0%)	8 (1.65%)	125 (25.72%)	133 (27.37%)
Nonevents (non-SCD) (% all non-SCDs)	0 (0.0%)	193 (0.98%)	914 (4.64%)	1,107 (5.62%)
Proportion of all deaths due to SCD in this category	0.0%	7.27%	15.72%	14.70%
Non-sudden fatal CHD (% all deaths in category)	0 (0.0%)	47 (42.73%)	326 (41.01%)	373 (41.22%)
Non-CHD death (% all deaths in category)	0 (0.0%)	55 (50.00%)	344 (43.27%)	399 (44.09%)
Proportion of all deaths not due to SCD in this category	0.0%	92.73%	84.28%	85.30%
All-cause death (% total participants in this category)	0 (0.0%)	110 (54.73%)	795 (76.52%)	905 (72.98%)
Total				
Participants in this category (% total study cohort)	10,081 (49.96%)	8,652 (42.88%)	1,444 (7.16%)	20,177 (100.00%)
SCD events (% of all SCD events)	61 (12.55%)	251 (51.65%)	174 (35.80%)	486 (100.00%)
Nonevents (non-SCD) (% all non-SCDs)	10,020 (50.89%)	8,401 (42.66%)	1,270 (6.45%)	19,691(100.00%)
Proportion of all deaths due to SCD in this category	3.74%	6.72%	16.42%	7.56%
Non-sudden fatal CHD (% all deaths in category)	329 (20.15%)	1,214 (32.50%)	434 (40.94%)	1,977 (30.76%)
Non-CHD death (% all deaths in category)	1,243 (76.12%)	2,270 (60.78%)	452 (42.64%)	3,965 (61.68%)
Proportion of all deaths not due to SCD in this category	96.26%	93.28%	83.58%	92.44%
All-cause death (% total participants in this category)	1,633 (16.20%)	3,735 (43.17%)	1,060 (73.41%)	6,428 (31.86%)

Supplemental Table 12: Ten-year risk for SCD as predicted by the clinical-only and clinical+GEH risk scores using competing risks models, a low-risk cutoff of 0.5% and a high-risk cutoff of 5% in a subgroup of participants with BBB/IVCD

	10-year risk from **Clinical+GEH** risk score			
	<0.5%	**0.5-5%**	**>5%**	**Total**
10-year risk from **Clinical-only** risk score				
<1%				
Participants in this category (% total study cohort)	141 (6.06%)	5 (2.41%)	4 (0.17%)	201 (8.63%)
SCD events (% of all SCD events)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Nonevents (non-SCD) (% all non-SCDs)	141 (6.31%)	56 (2.50%)	4 (0.18%)	201 (8.99%)
Proportion of all deaths due to SCD in this category	0.0%	0.0%	0.0%	0.0%
Non-sudden fatal CHD (% all deaths in category)	2 (50.00%)	0 (0.0%)	0 (0.0%)	2 (28.57%)
Non-CHD death (% all deaths in category)	2 (50.00%)	3 (100.00%)	0 (0.0%)	5 (71.43%)
Proportion of all deaths not due to SCD in this category	100.00%	100.00%	0.0%	100.00%
All-cause death (% total participants in this category)	4 (2.84%)	3 (5.36%)	0 (0.0%)	7 (3.48%)
0.5-5%				
Participants in this category (% total study cohort)	244 (10.48%)	1,439 (61.81%)	159 (6.83%)	1,842 (79.12%)
SCD events (% of all SCD events)	0 (0.0%)	33 (35.87%)	27 (29.35%)	60 (65.22%)
Nonevents (non-SCD) (% all non-SCDs)	244 (10.91%)	1,406 (62.88%)	132 (5.90%)	1,782 (79.70%)
Proportion of all deaths due to SCD in this category	0.0%	6.42%	24.55%	9.23%
Non-sudden fatal CHD (% all deaths in category)	6 (23.08%)	160 (31.13%)	45 (40.91%)	211 (32.46%)
Non-CHD death (% all deaths in category)	20 (76.92%)	321 (62.45%)	38 (34.55%)	379 (58.31%)
Proportion of all deaths not due to SCD in this category	100.00%	93.58%	75.45%	90.77%
All-cause death (% total participants in this category)	26 (10.66%)	514 (35.72%)	110 (69.18%)	650 (35.29%)
>5%				
Participants in this category (% total study cohort)	0 (0.0%)	36 (1.55%)	249 (10.70%)	285 (12.24%)
SCD events (% of all SCD events)	0 (0.0%)	0 (0.0%)	32 (34.78%)	32 (34.78%)
Nonevents (non-SCD) (% all non-SCDs)	0 (0.0%)	36 (1.61%)	217 (9.70%)	253 (11.31%)
Proportion of all deaths due to SCD in this category	0.0%	0%	15.38%	13.73%
Non-sudden fatal CHD (% all deaths in category)	0 (0.0%)	15 (60.00%)	91 (43.75%)	106 (45.49%)
Non-CHD death (% all deaths in category)	0 (0.0%)	10 (40.00%)	85 (40.9%)	95 (40.77%)
Proportion of all deaths not due to SCD in this category	0.0%	100.00%	84.62%	86.27%
All-cause death (% total participants in this category)	0 (0.0%)	25 (69.44%)	208 (83.53%)	233 (81.75%)
Total				
Participants in this category (% total study cohort)	385 (16.54%)	1,531 (65.76%)	412 (17.70%)	2,328 (1000.0%)
SCD events (% of all SCD events)	0 (0.0%)	33 (35.87%)	59 (64.13%)	92 (100.00%)
Nonevents (non-SCD) (% all non-SCDs)	385 (17.22%)	1,498 (66.99%)	353 (15.79%)	2,236 (100.00%)
Proportion of all deaths due to SCD in this category	0.0%	6.09%	18.55%	10.34%
Non-sudden fatal CHD (% all deaths in category)	8 (26.67%)	175 (32.29%)	136 (42.77%)	319 (35.84%)
Non-CHD death (% all deaths in category)	22 (73.33%)	334 (61.62%)	123 (38.68%)	479 (53.82%)
Proportion of all deaths not due to SCD in this category	100.00%	93.91%	81.45%	89.66%
All-cause death (% total participants in this category)	30 (7.79%)	542 (35.40%)	318 (77.18%)	890 (38.23%)

Supplemental Table 13: Ten-year risk for SCD as predicted by the clinical-only and clinical+GEH risk scores using competing risks models, a low-risk of 0.5% and a high-risk of 5% in a subgroup with implanted pacemakers and ventricular-paced ECGs.

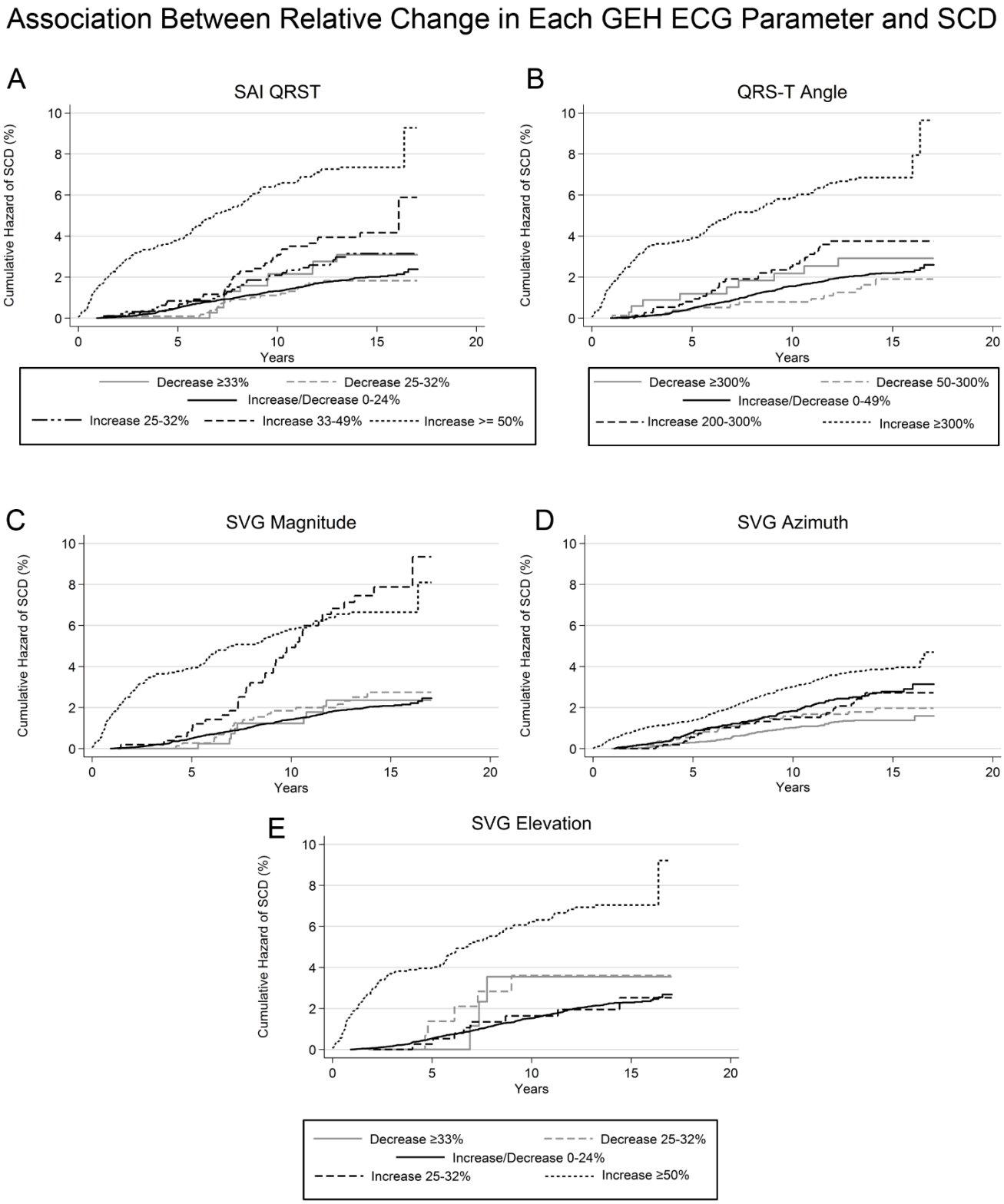
	10-year risk from **Clinical+GEH** risk score			
	<0.5%	**0.5-5%**	**>5%**	**Total**
10-year risk from **Clinical-only** risk score				
<1%				
Participants in this category (% total study cohort)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
SCD events (% of all SCD events)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Nonevents (non-SCD) (% all non-SCDs)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Proportion of all deaths due to SCD in this category	0.0%	0.0%	0.0%	0.0%
Non-sudden fatal CHD (% all deaths in category)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Non-CHD death (% all deaths in category)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Proportion of all deaths not due to SCD in this category	0.0%	0.0%	0.0%	0.0%
All-cause death (% total participants in this category)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
1-5%				
Participants in this category (% total study cohort)	0 (0.0%)	16 (34.04%)	10 (21.28%)	26 (55.32%)
SCD events (% of all SCD events)	0 (0.0%)	1 (16.67%)	2 (33.33%)	3 (50.00%)
Nonevents (non-SCD) (% all non-SCDs)	0 (0.0%)	15 (36.59%)	8 (19.51%)	23 (56.10%)
Proportion of all deaths due to SCD in this category	0.0%	6.67%	20.00%	12.00%
Non-sudden fatal CHD (% all deaths in category)	0 (0.0%)	8 (53.33%)	5 (50.00%)	13 (52.00%)
Non-CHD death (% all deaths in category)	0 (0.0%)	6 (40.00%)	3 (30.00%)	9 (36.00%)
Proportion of all deaths not due to SCD in this category	0.0%	93.33%	80.00%	88.00%
All-cause death (% total participants in this category)	0 (0.0%)	15 (93.75%)	10(100.00%)	25 (96.15%)
>5%				
Participants in this category (% total study cohort)	0 (0.0%)	3(6.38%)	18 (38.30%)	21 (44.68%)
SCD events (% of all SCD events)	0 (0.0%)	0 (0.0%)	3 (50.00%)	3 (50.00%)
Nonevents (non-SCD) (% all non-SCDs)	0 (0.0%)	3 (7.32%)	15 (36.59%)	18 (43.90%)
Proportion of all deaths due to SCD in this category	0.0%	0.0%	16.67%	15.00%
Non-sudden fatal CHD (% all deaths in category)	0 (0.0%)	2 (100.0.%)	7 (38.89%)	9 (45.00%)
Non-CHD death (% all deaths in category)	0 (0.0%)	0 (0.0%)	8 (44.44%)	8 (40.00%)
Proportion of all deaths not due to SCD in this category	0.0%	100.00%	83.33%	85.00%
All-cause death (% total participants in this category)	0 (0.0%)	2 (66.67%)	18 (100.00%)	20 (95.24%)
Total				
Participants in this category (% total study cohort)	0 (0.0%)	19 (40.43%)	28 (59.57%)	47 (100.00%)
SCD events (% of all SCD events)	0 (0.0%)	1 (16.67%)	5 (83.33%)	6 (100.00%)
Nonevents (non-SCD) (% all non-SCDs)	0 (0.0%)	18 (43.90%)	23(56.10%)	41(100.00%)
Proportion of all deaths due to SCD in this category	0.0%	5.88%	17.86%	13.33%
Non-sudden fatal CHD (% all deaths in category)	0 (0.0%)	10 (58.82%)	12 (42.86%)	22 (48.89%)
Non-CHD death (% all deaths in category)	0 (0.0%)	6 (35.29%)	11 (39.29%)	17 (37.78%)
Proportion of all deaths not due to SCD in this category	0.0%	94.12%	82.14%	86.67%
All-cause death (% total participants in this category)	0 (0.0%)	17 (89.47%)	28 (100%)	45 (95.74%)

Supplemental Figure Legend:

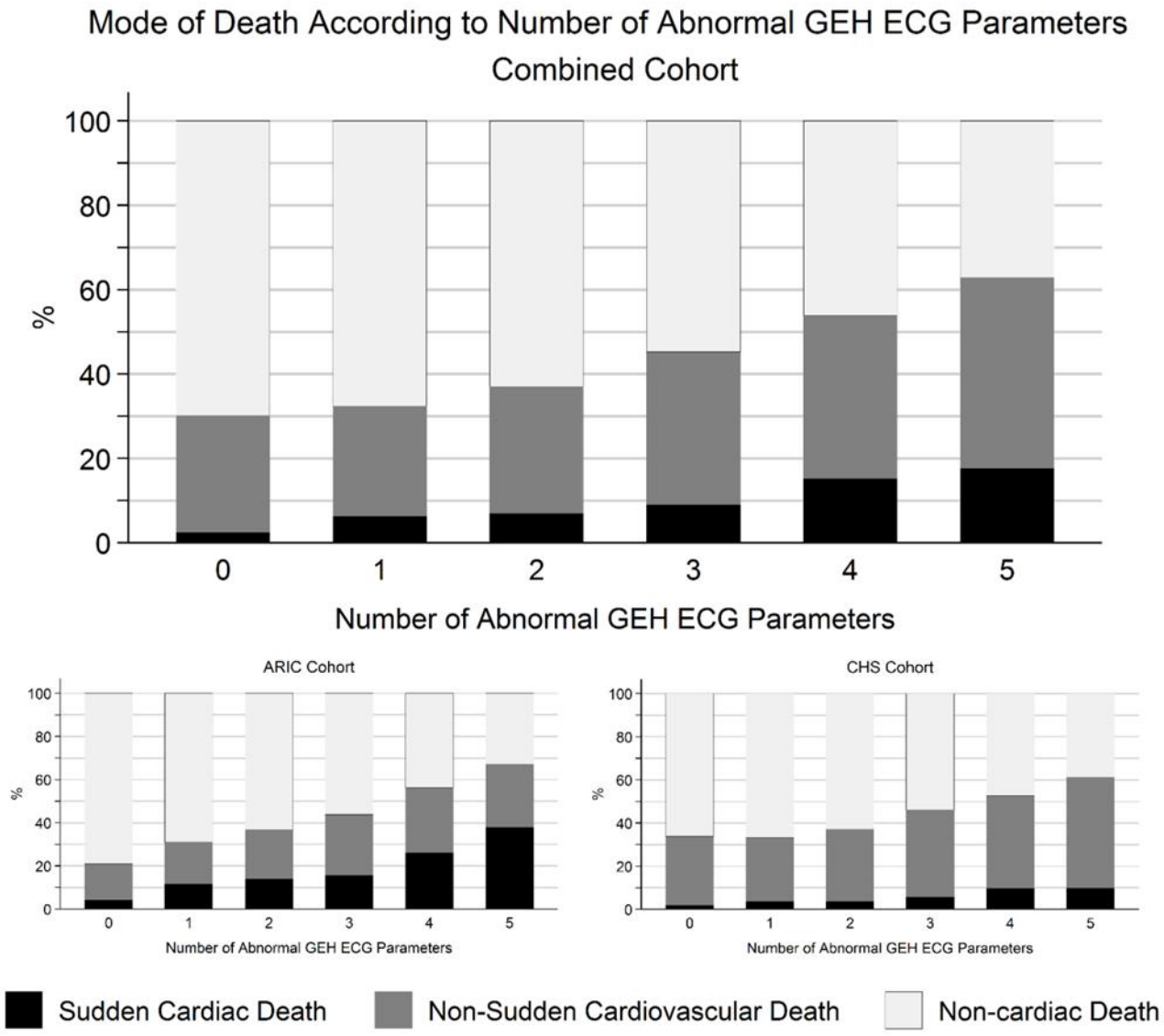
Supplemental Figure 1: Unadjusted Kaplan-Meier curves for the probability of SCD in study participants who experienced significant increases in (**A**) sum absolute QRST integral (SAI QRST), (**B**) spatial QRS-T angle, (**C**) spatial ventricular gradient (SVG) magnitude, (**D**) SVG azimuth, and (**E**) SVG elevation, at study follow-up visits 2 or 3, as compared to baseline study visit.

Supplemental Figure 2. Increase in the proportion of all deaths due to SCD with an increasing number of abnormal GEH ECG parameters. Each GEH ECG parameter was given equal weight in this figure. **A:** Merged ARIC-CHS cohort, **B**: ARIC cohort, **C:** CHS cohort.

Supplemental Figure 1:



Supplemental Figure 2:



References:

1. Fried LP, Borhani NO, Enright P, Furberg CD, Gardin JM, Kronmal RA, Kuller LH, Manolio TA, Mittelmark MB, Newman A and et al. The Cardiovascular Health Study: design and rationale. *Ann Epidemiol*. 1991;1:263-76.

2. The ARIC Investigators. The Atherosclerosis Risk in Community (ARIC) Study: Design and Objectives. *Am J Epidemiol*. 1989;129:687-702.

3. Mittelmark MB, Psaty BM, Rautaharju PM, Fried LP, Borhani NO, Tracy RP, Gardin JM and O'Leary DH. Prevalence of cardiovascular diseases among older adults. The Cardiovascular Health Study. *Am J Epidemiol*. 1993;137:311-7.

4. Blackburn H, KEYS A, Simonson E, Rautaharju P and PUNSAR S. The electrocardiogram in population studies. A classification system. *Circulation*. 1960;21:1160-1175.

5. Psaty BM, Lee M, Savage PJ, Rutan GH, German PS and Lyles M. Assessing the use of medications in the elderly: methods and initial experience in the Cardiovascular Health Study. The Cardiovascular Health Study Collaborative Research Group. *J Clin Epidemiol*. 1992;45:683-92.

6. Toole JF, Chambless LE, Heiss G, Tyroler HA and Paton CC. Prevalence of stroke and transient ischemic attacks in the atherosclerosis risk in communities (ARIC) study. *Annals of Epidemiology*. 1993;3:500-503.

7. Eriksson H, CAIDAUL K, LARSSON B, OHLSON LO, WELIN L, Wilhelmsen L and SV+äRDSUDD K. Cardiac and pulmonary causes of dyspnoeaΓÇövalidation of a scoring test for

clinical-epidemiological use: The Study of Men Born in 1913. *European Heart Journal*. 1987;8:1007-1014.

8. Psaty BM, Kuller LH, Bild D, Burke GL, Kittner SJ, Mittelmark M, Price TR, Rautaharju PM and Robbins J. Methods of assessing prevalent cardiovascular disease in the Cardiovascular Health Study. *Ann Epidemiol*. 1995;5:270-7.

9. Alonso A, Agarwal SK, Soliman EZ, Ambrose M, Chamberlain AM, Prineas RJ and Folsom AR. Incidence of atrial fibrillation in whites and African-Americans: the Atherosclerosis Risk in Communities (ARIC) study. *AmHeart J*. 2009;158:111-117.

10. Mozaffarian D, Psaty BM, Rimm EB, Lemaitre RN, Burke GL, Lyles MF, Lefkowitz D and Siscovick DS. Fish intake and risk of incident atrial fibrillation. *Circulation*. 2004;110:368-73.

11. Rosamond WD, Folsom AR, Chambless LE, Wang CH, McGovern PG, Howard G, Copper LS and Shahar E. Stroke incidence and survival among middle-aged adults: 9-year follow-up of the Atherosclerosis Risk in Communities (ARIC) cohort. *Stroke*. 1999;30:736-743.

12. Rathore SS, Hinn AR, Cooper LS, Tyroler HA and Rosamond WD. Characterization of incident stroke signs and symptoms: findings from the atherosclerosis risk in communities study. *Stroke*. 2002;33:2718-21.

13. Manolio TA, Kronmal RA, Burke GL, O'Leary DH and Price TR. Short-term predictors of incident stroke in older adults. The Cardiovascular Health Study. *Stroke*. 1996;27:1479-86.

14. Matsushita K, Blecker S, Pazin-Filho A, Bertoni A, Chang PP, Coresh J and Selvin E. The association of hemoglobin a1c with incident heart failure among people without diabetes: the atherosclerosis risk in communities study. *Diabetes*. 2010;59:2020-2026.

15. Del Gobbo LC, Kalantarian S, Imamura F, Lemaitre R, Siscovick DS, Psaty BM and Mozaffarian D. Contribution of Major Lifestyle Risk Factors for Incident Heart Failure in Older Adults: The Cardiovascular Health Study. *JACC Heart Fail*. 2015;3:520-8.

16. Soliman EZ, Lopez F, O'Neal WT, Chen LY, Bengtson L, Zhang ZM, Loehr L, Cushman M and Alonso A. Atrial Fibrillation and Risk of ST-Segment-Elevation Versus Non-ST-Segment-Elevation Myocardial Infarction: The Atherosclerosis Risk in Communities (ARIC) Study. *Circulation*. 2015;131:1843-50.

17. Ives DG, Fitzpatrick AL, Bild DE, Psaty BM, Kuller LH, Crowley PM, Cruise RG and Theroux S. Surveillance and ascertainment of cardiovascular events. The Cardiovascular Health Study. *Ann Epidemiol*. 1995;5:278-85.

18. Blanco-Velasco M, Weng B and Barner KE. ECG signal denoising and baseline wander correction based on the empirical mode decomposition. *Comput Biol Med*. 2008;38:1-13.

19. Zhao Z and Liu J. Baseline Wander Removal of ECG Signals Using Empirical Mode Decomposition and Adaptive Filter. *Bioinformatics and Biomedical Engineering (iCBBE), 2010 4th International Conference on*. 2010:1-3.

20. Edenbrandt L and Pahlm O. Vectorcardiogram synthesized from a 12-lead ECG: superiority of the inverse Dower matrix. *JElectrocardiol*. 1988;21:361-367.

21. Sur S, Han L and Tereshchenko LG. Comparison of sum absolute QRST integral, and temporal variability in depolarization and repolarization, measured by dynamic vectorcardiography approach, in healthy men and women. *PLoS One*. 2013;8:e57175.

22. WILSON FN, Macleod AG and Barker PS. The distribution of the action currents produced by heart muscle and other excitable tissues immersed in extensive conducting media. *JGenPhysiol*. 1933;16:423-456.

23. WILSON FN, Macleod AG, Barker PS and JOHNSTON FD. The determination and the significance of the areas of the ventricular deflections of the electrocardiogram. *Am Heart J*. 1934;10:46-61. doi:10.1016/S0002-8703(34)90303-3

24. Gardberg M and ROSEN IL. Monophasic curve analysis and the ventricular gradient in the electrogram of strips of turtle ventricle. *CircRes*. 1959;7:870-875.

25. Abildskov JA, Urie P, Lux R, Burgess MJ and Wyatt R. Body surface distribution of QRST area. *AdvCardiol*. 1978;21:59-64.

26. Tereshchenko LG, Ghanem RN, Abeyratne A and Swerdlow CD. Intracardiac QT integral on far-field ICD electrogram predicts sustained ventricular tachyarrhythmias in ICD patients. *Heart Rhythm*. 2011;8:1889-1894.

27. White AD, Folsom AR, Chambless LE, Sharret AR, Yang K, Conwill D, Higgins M, Williams OD and Tyroler HA. Community surveillance of coronary heart disease in the Atherosclerosis Risk in Communities (ARIC) Study: methods and initial two years' experience. *J Clin Epidemiol*. 1996;49:223-233.

28. Waks JW, Soliman EZ, Henrikson CA, Sotoodehnia N, Han L, Agarwal SK, Arking DE, Siscovick DS, Solomon SD, Post WS, Josephson ME, Coresh J and Tereshchenko LG. Beat-to-beat spatiotemporal variability in the T vector is associated with sudden cardiac death in participants without left ventricular hypertrophy: the Atherosclerosis Risk in Communities (ARIC) Study. *J Am Heart Assoc*. 2015;4:e001357.

29. Baker SG. Identifying combinations of cancer markers for further study as triggers of early intervention. *Biometrics*. 2000;56:1082-7.