ORIGINAL RESEARCH

Bringing Critical Race Praxis Into the Study of Electrophysiological Substrate of Sudden Cardiac Death: The ARIC Study

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***Background*—**Race is an established risk factor for sudden cardiac death (SCD). We sought to determine whether the association of electrophysiological substrate with SCD varies between black and white individuals.

***Methods and Results*—**Participants from the ARIC (Atherosclerosis Risk in Communities) study with analyzable ECGs (n=14 408; age, 54±6 years; 74% white) were included. Electrophysiological substrate was characterized by ECG metrics. Two competing outcomes were adjudicated: SCD and non-SCD. Interaction of ECG metrics with race was studied in Cox proportional hazards and Fine-Gray competing risk models, adjusted for prevalent cardiovascular disease, risk factors, and incident nonfatal cardiovascular disease. At the baseline visit, adjusted for age, sex, and study center, blacks had larger spatial ventricular gradient magnitude (0.30 mV; 95% CI, 0.25–0.34 mV), sum absolute QRST integral (18.4 mV*ms; 95% CI, 13.7–23.0 mV*ms), and Cornell voltage (0.30 mV; 95% CI, 0.25–0.35 mV) than whites. Over a median follow-up of 24.4 years, SCD incidence was higher in blacks (2.86 per 1000 person-years; 95% CI, 2.50–3.28 per 1000 person-years) than whites (1.37 per 1000 person-years; 95% CI, 1.22–1.53 per 1000 person-years). Blacks with hypertension had the highest rate of SCD: 4.26 (95% CI, 3.66–4.96) per 1000 person-years. Race did not modify an association of ECG variables with SCD, except QRS-T angle. Spatial QRS-T angle was associated with SCD in whites (hazard ratio, 1.38; 95% CI, 1.25–1.53) and hypertension-free blacks (hazard ratio, 1.52; 95% CI, 1.09–2.12), but not in blacks with hypertension (hazard ratio, 1.15; 95% CI, 0.99–1.32) (*P*-interaction=0.004).

***Conclusions*—**Race did not modify associations of electrophysiological substrate with SCD and non-SCD. Electrophysiological substrate does not explain racial disparities in SCD rate. **(*J Am Heart Assoc*. 2020;9:e015012. DOI: 10.1161/JAHA.119.015012.)**

Key Words: ECG • global electrical heterogeneity • race • sudden cardiac death

Sudden cardiac death (SCD) disproportionately affects black individuals, with a nearly 2-fold increased risk of SCD in black compared with white individuals.[1–3] Socioeconomic disparities and the burden of cardiovascular risk have been proposed as factors that may underlie the observed racial differences in SCD.[1] However, adjustment for socioeconomic and behavioral measures of health does not fully explain an excess of SCD risk in black inidividuals.[1,2,4] Moreover, the large prospective community-based cohort, the ARIC (Atherosclerosis Risk in Communities) study, showed that race modifies an association of several major risk factors with SCD. Prevalent coronary heart disease (CHD) and body mass index (BMI) carried greater risk of SCD in white compared with black individuals.[1] In contrast, hypertension carried a significantly larger risk of SCD in black individuals compared with white individuals.[1] The mechanisms behind these observed interactions remain unclear but pose the question of differences in electrophysiological substrate metrics between the 2 racial groups.

A conventional 12-lead ECG characterizes the global electrophysiological substrate of SCD,[5] which can be assessed by traditional ECG metrics, such as heart rate, QRS duration, corrected QT (QTc) interval, and ECG left ventricular hypertrophy (LVH). Previous studies did not find differences in heart rate, QRS duration, and QT (QTc) QTc interval between white and black individuals.[2] Yet, racial differences in ECG LVH are well-known,[6–8] bearing challenges in ECG interpretation.[9] Although racial differences in ECG LVH diagnostic criteria are recognized,[10] it is unknown whether there are racial differences in the strength of association between electrophysiological substrate and SCD.

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Clinical Perspective

What Is New?

- Race does not modify an association between electrophysiological substrate and sudden cardiac death.
- Voltage-based ECG metrics (spatial ventricular gradient magnitude, sum absolute QRST integral, and Cornell voltage) are larger in black than white individuals.
- Voltage-based ECG metrics are associated with increased sudden cardiac death risk in obese black individuals.

What Are the Clinical Implications?

- Applying Critical Race Theory to increased risk of sudden cardiac death in black compared with white individuals emphasizes the need for additional studies investigating the effects of institutionalized racism on health.
- Development of race-specific sudden cardiac death risk stratification tools is needed to incorporate race-specific thresholds of ECG voltage-based metrics.
- Prevention and treatment of obesity in black individuals with increased voltage-based ECG metrics may be potentially lifesaving.

Global electrical heterogeneity (GEH) is a novel ECG measure of global electrophysiological substrate.[11,12] GEH is measured by spatial QRS-T angle, magnitude and direction (elevation and azimuth) of the spatial ventricular gradient (SVG) vector, and its scalar value sum absolute QRST integral (SAI QRST). The addition of GEH to known cardiovascular risk factors improves the reclassification of SCD risk.[13] The SVG vector describes the magnitude and direction of the steepest gradient between the areas of the heart with the longest and the shortest total recovery time.[14–16] The SVG is related to global heterogeneity of action potential duration and morphological characteristics[17,18] through the heart. Although association of GEH with SCD is independent of cardiovascular disease and known cardiovascular risk factors,[13] it remains unknown whether race can modify an association of GEH with SCD.

Self-identified race is a social construct. Recently, the Public Health Critical Race Praxis[19] was developed as a theoretical framework for studies of racial disparities. Critical Race Theory[20] aims to acknowledge inequities in the biomedical field and bring to light health consequences that result from discrimination and inequalities in access to health care. Race is a product of social processes of power. Critical Race Theory defines race not as an inherent characteristic of a person, but as a product of social practices.[20] Our goal was to apply the Public Health Critical Race Praxis[19] approach to the epidemiological study of SCD substrate, to answer the question of whether race modifies the association of electrophysiological substrate with SCD. We hypothesized that (1) there are racial differences in global ECG measures of electrophysiological substrate and (2) self-identified race modifies the association of electrophysiological substrate with SCD.

Methods

The ARIC study data are available through the National Heart, Lung, and Blood Institute's Biological Specimen and Data Repository Information Coordinating Center[21] and the National Center of Biotechnology Information's Database of Genotypes and Phenotypes.[22] Informed consent was obtained from all study participants before enrollment. The study was approved by the Oregon Health and Science University Institutional Review Board.

Study Population

The ARIC study recruited 15 792 participants (age, 45–64 years) in 1987 to 1989. All participants underwent standardized examinations.[23] In this study, we included ARIC study cohort participants with recorded resting 12-lead ECG and measured GEH[13] (n=15 777). We excluded participants who self-identified themselves as nonwhite or nonblack race (n=48), as black at the Washington County and Minneapolis field centers (because of small subgroup size; n=55), and those with missing covariates (n=1220), outcome (n=1), and nonsinus median beat (n=45). The final sample of participants with normal sinus median beat included 14 408 participants (Figure 1).

Exposures of Race and Electrocardiographic GEH

Race was self-reported. We analyzed resting 12-lead ECGs of the first 5 study visits.[24] Visit 1 was conducted in 1987 to 1989, visit 2 in 1990 to 1992, visit 3 in 1993 to 1995, visit 4 in 1996 to 1998, and visit 5 in 2011 to 2013.

Electrophysiological substrate was characterized by traditional and novel global ECG metrics.[24] Traditional ECG metrics (heart rate, QRS duration, Bazett-corrected QTc interval, and R_{aVL} and S_{V3} amplitudes) were measured by the 12 SL algorithm, as implemented in the Magellan ECG Research Workstation V2 (GE Marquette Electronics, Milwaukee, WI); and Cornell voltage was calculated as the sum of R_{aVL} and S_{V3} amplitudes.

GEH was measured by spatial QRS-T angle, SVG magnitude, azimuth, and elevation, and SAI QRST.[25] Both area and peak SVG vectors as well as QRS-T angles were measured.[25] We used normal sinus time-coherent median beat with identified isoelectric heart vector origin point.[26] The open-source MATLAB (MathWorks, Natick, MA) software code for

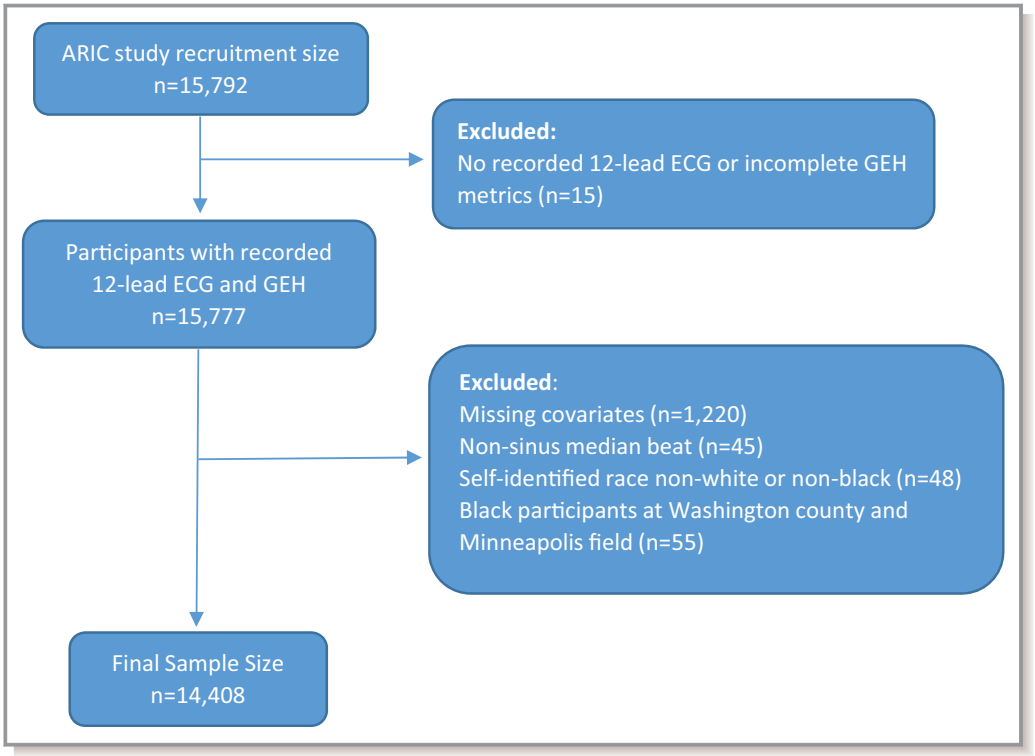

Figure 1. Flowchart of study cohort development. ARIC indicates Atherosclerosis Risk in Communities; GEH, global electrical heterogeneity.

GEH measurement and the heart vector origin definition is provided at https://physionet.org/physiotools/geh and https://github.com/Tereshchenkolab/Origin.

Primary Outcome: SCD

Follow-up of ARIC study participants[27] and determination of SCD have been described in prior reports.[24] SCD, the primary study outcome, was defined as a sudden pulseless condition in a previously stable participant without a noncardiac cause of arrest if it occurred outside of the hospital or in the emergency department. It was classified as definite, probable, or possible, on the basis of physician adjudication.

Competing mortality outcome: non-SCD

Competing non-SCD was defined as an SCD exclusion, a composite of fatal CHD, heart failure (HF) death, death in a participant with baseline HF, or incident hospitalized HF. Fatal CHD cases were adjudicated by the ARIC study Morbidity and Mortality Classification Committee.[27,28] Baseline prevalent HF was based on Gothenburg criteria (stage 3 symptomatic HF with both cardiac and pulmonary symptoms and current medical treatment[29]) or self-reported use of HF medication. Incident HF was defined as the presence of HF codes in a death certificate or an *International Classification of Diseases, Ninth Revision* (*ICD*-9), discharge code, as previously described.[30] All other deaths were included in the noncardiac death outcome.

Baseline Clinical Characteristics

BMI was categorized as underweight (<18.5 kg/m^2), normal weight (18.5–<25.0 kg/m^2), overweight (25.0–<30.0 kg/m^2), or obese (≥30.0 kg/m^2). Hypertension was defined as blood pressure of ≥140/90 mm Hg or self-reported antihypertensive medications at visit 1. Diabetes mellitus was defined as nonfasting blood glucose ≥200 mg/dL, fasting blood glucose ≥126 mg/dL, self-reported physician diagnosis of diabetes mellitus, or self-reported medications for diabetes mellitus or high blood sugar at visit 1. Stages of chronic kidney disease (CKD) were based on estimated glomerular filtration rate (eGFR), calculated using the CKD Epidemiology Collaboration (CKD-EPI) equation.[31] Participants were classified into stage 1 CKD ($eGFR_{CKD\text{-}EPI}$ ≥90 mL/min per 1.73 m^2), stage 2 CKD ($eGFR_{CKD\text{-}EPI}$ 60–<90 mL/min per 1.73 m^2), stage 3 CKD ($eGFR_{CKD\text{-}EPI}$ 30–<60 mL/min per 1.73 m^2), stage 4 CKD ($eGFR_{CKD\text{-}EPI}$ 15–<30 mL/min per 1.73 m^2), stage 5 CKD ($eGFR_{CKD\text{-}EPI}$ <15 mL/min per 1.73 m^2), or established kidney failure. Baseline serum electrolyte concentrations were measured in the central laboratory.

Prevalent stroke was based on a previously reported stroke and transient ischemic attack diagnostic algorithm.[32]

Prevalent CHD included a self-reported physician diagnosis of myocardial infarction, baseline ECG evidence of myocardial infarction by the Minnesota code,[33] or a history of coronary revascularization (via either coronary artery bypass surgery or percutaneous coronary intervention). The use of antiarrhythmic drugs included self-reported and validated by medications inventory use of class I, II (β blockers), III, IV (phenylalkylamines and benzothiazepine calcium channel blockers), or V (digoxin) antiarrhythmic agents.

Socioeconomic Factors

Socioeconomic status was assessed during a home interview at visit 1. Total combined family income for the past 12 months (in 1987–1989 values) was self-reported in one of the following categories: <$5000; $5000 to $7999; $8000 to $11 999; $12 000 to $15 999; $16 000 to $24 999; $25 000 to $34 999; $35 999 to $49 999; >$50 000; or not reported. Lifetime educational level was defined as the highest grade or year of school completed. The most recent occupation was recorded in one of the following categories: (1) managerial and professional specialty occupations; (2) technical, sales, and administrative support occupations; (3) service occupations; (4) farming, forestry, and fishing occupations; (5) precision production, craft, and repair occupations; (6) operators, fabricators, and laborers; (7) homemakers; (8) retired; or (9) others.

Baseline physical activity was measured at work, in sport, and during leisure time, using the modified Baecke questionnaire,[34] which defined semicontinuous indexes ranging from 1 (low) to 5 (high). Current cigarette smoking and consumption of alcoholic beverages were self-reported at visit 1. Self-reported health insurance status was ascertained at visit 1.

Incident Nonfatal Cardiovascular Events

Incident nonfatal cardiovascular events included atrial fibrillation, stroke, CHD, and HF. Incident atrial fibrillation included atrial fibrillation detected on follow-up 12-lead ECG or hospital discharge records (*ICD-9* code 427.3).[35] Physician-adjudicated definite or probable incident strokes were included.[36] Incident CHD was physician adjudicated and included definite or probable myocardial infarction or a coronary revascularization procedure.[27,28] Incident HF was defined above.[30]

Statistical Analyses

Cross-sectional analyses of visit 1 data

To investigate differences in global ECG metrics between black and white individuals, we performed cross-sectional linear regression analyses using visit 1 data. Model 1 was adjusted for age, sex, and study center. To determine whether racial differences in GEH could be explained by racial disparities, including differences in socioeconomic, traditional, and novel clinical risk factors, model 2 was additionally adjusted for prevalent cardiovascular disease (HF, CHD, or stroke), known cardiovascular risk factors (diabetes mellitus, hypertension, current smoking and alcohol intake, work, sport, and leisure physical activity levels, levels of total cholesterol, high-density lipoprotein, and triglycerides, and BMI), use of antihypertensive and antiarrhythmic medications, serum concentrations of sodium, potassium, calcium, magnesium, phosphorus, and uric acid, total protein and albumin, blood urea nitrogen, CKD stage classified by $eGFR_{CKD\text{-}EPI}$, traditional ECG characteristics (mean heart rate, QRS duration, QTc interval, and Cornell voltage), and socioeconomic factors (education level, occupation category, income, and health insurance).

Analysis of circular variables

Spatial QRS-T angle, SVG azimuth, and SVG elevation are circular variables. Unadjusted comparison of circular variables was performed using the Watson U-square statistic and the Kuiper statistics.

By convention, QRS-T and SVG elevation angles can be only positive, ranging from 0° to 180°. Because distributions of QRS-T angle and SVG elevation angle were normal or nearly normal, QRS-T and SVG elevation angles were included in all conventional statistical analyses without transformation. The SVG azimuth angle is expressed as an axial variable, ranging from −180° to 180°. As recommended for circular statistics,[37] we transformed SVG azimuth by doubling its value and then adding 360°. We then analyzed the SVG azimuth using a conventional statistical approach, and for interpretation, transformed it back.

Survival analyses

We built Cox proportional hazards and Fine-Gray competing risks models. The proportional-hazards assumption was verified using *stcox PH-assumptions* suite of tests implemented in STATA (StataCorp LP, College Station, TX) for all predictors of interest in most models. Exceptions were stated. All ECG variables were represented as their z score to standardize comparisons. To adjust for confounders, we constructed 2 models, performed a statistical test for interaction with race in each model, and constructed race-stratified models for white and black individuals. Relative hazard ratio with 95% CI of SCD risk for black relative to white individuals was reported, assuming hazard ratio for white individuals is a reference.

Model 1 was adjusted for demographic characteristics (age, sex, and study center), prevalent cardiovascular disease (HF, CHD, and stroke), baseline cardiovascular risk factors (diabetes mellitus, hypertension, levels of total cholesterol, high-density

Table 1. Comparison of Baseline Clinical and ECG Characteristics in Black and White Participants

Characteristics	Black Participants (n=3739)	White Participants (n=10 669)	*P* Value
Age, mean (SD), y	53.5 (5.8)	54.4 (5.7)	<0.0001
Women, n (%)	2309 (61.8)	5622 (52.7)	<0.0001
Heart failure, n (%)	257 (6.9)	407 (3.8)	<0.0001
Coronary heart disease, n (%)	148 (4.0)	534 (5.0)	0.009
Stroke, n (%)	106 (2.8)	138 (1.29)	<0.0001
Body mass index, mean (SD), kg/m^2	29.6 (6.2)	27.0 (4.9)	<0.0001
Diabetes mellitus, n (%)	733 (19.6)	965 (9.0)	<0.0001
Hypertension, n (%)	2077 (55.6)	2886 (27.1)	<0.0001
Antihypertensive drugs, n (%)	1644 (44.0)	2731 (25.6)	<0.0001
Current tobacco smoker, n (%)	1126 (30.1)	2643 (24.8)	<0.0001
Current alcohol drinker, n (%)	1189 (31.8)	6894 (64.6)	<0.0001
Leisure physical activity score, mean (SD)	2.1 (0.6)	2.5 (0.5)	<0.0001
Sport physical activity score, mean (SD)	2.2 (0.7)	2.5 (0.8)	<0.0001
Work physical activity score, mean (SD)	2.3 (1.0)	2.1 (0.9)	<0.0001
Family income <$25 000, n (%)	2406 (64.4)	2779 (26.0)	<0.0001
Education less than high school, n (%)	1538 (41.1)	1813 (17.0)	<0.0001
Health insurance, n (%)	2883 (77.1)	10 163 (95.3)	<0.0001
Total cholesterol, mean (SD), mmol/L	5.6 (1.2)	5.6 (1.1)	0.924
HDL cholesterol, mean (SD), mg/dL	55.2 (17.7)	50.4 (16.7)	<0.0001
Triglycerides, mean (SD), mmol/L	1.3 (0.9)	1.6 (1.0)	<0.0001
Chronic kidney disease stage ≤1, n (%)	2901 (77.6)	7039 (66.0)	<0.0001
Use of antiarrhythmic drugs, n (%)	548 (14.7)	1464 (13.7)	0.156
Heart rate, mean (SD), bpm	66.5 (11.0)	66.2 (9.9)	0.132
QRS duration, mean (SD), ms	91.4 (12.6)	92.5 (12.2)	<0.0001
QTc, mean (SD), ms	418 (21)	416 (18)	<0.0001
Cornell voltage, mean (SD), mV	1.5 (0.6)	1.2 (0.5)	<0.0001
Peak QRS-T angle, mean (circular SD), °	47.3 (36.3)	40.6 (30.2)	<0.001
Area QRS-T angle, mean (circular SD), °	60.8 (29.4)	60.4 (27.6)	<0.05
Peak SVG elevation, mean (circular SD), °	66.4 (14.2)	62.4 (15.5)	<0.001
Area SVG elevation, mean (circular SD), °	71.5 (15.9)	66.0 (17.5)	<0.001
Peak SVG azimuth, mean (circular SD), °	6.7 (19.5)	1.2 (21.1)	<0.001
Area SVG azimuth, mean (circular SD), °	25.2 (21.5)	24.6 (21.5)	<0.001
SAI QRST, mean (SD), mV*ms	157 (60)	139 (46)	<0.0001
Peak SVG magnitude, mean (SD), mV	1.8 (0.5)	1.5 (0.4)	<0.0001
SVG magnitude, mean (SD), mV	2.0 (0.5)	1.6 (0.4)	<0.0001

Bpm indicates beats per minute; HDL, high-density lipoprotein; SAI QRST, sum absolute QRST integral; SVG, spatial ventricular gradient; QTc, corrected QT.

lipoprotein, and triglycerides, BMI, use of antihypertensive and antiarrhythmic medications, serum concentrations of sodium, potassium, calcium, magnesium, phosphorus, and uric acid, total protein and albumin, blood urea nitrogen, and CKD stage classified by $eGFR_{CKD\text{-}EPI}$,), and socioeconomic factors measured at visit 1 (smoking and alcohol intake, work, sport, and leisure physical activity levels, education level, occupation category, income, and health insurance). Time-updated model 2 included time-updated ECG predictors (one by one), all baseline covariates included in model 1, and time-varying covariate incident nonfatal cardiovascular events (atrial fibrillation, HF, CHD, and stroke). ECG variables were time varying, updated in

Table 2. Difference in GEH Variables in Black Compared With White Participants

	Model 1		Model 2	
GEH Characteristic	Difference (95% CI)	*P* Value	Difference (95% CI)	*P* Value
Peak QRS-T angle, °	11 (8.6 to 15.0)	<0.0001	5.2 (2.2 to 8.3)	0.001
Area QRS-T angle, °	1.4 (−1.2 to 4.1)	0.284	−2.7 (−5.2 to −0.3)	0.031
Peak SVG elevation, °	4.6 (3.1 to 6.0)	<0.0001	0.8 (−0.5 to 2.2)	0.832
Area SVG elevation, °	6.1 (4.5 to 7.8)	<0.0001	2.3 (0.6 to 3.9)	0.006
Peak SVG azimuth, °	11.0 (5.8 to 15.6)	<0.0001	0.3 (−4.5 to 5.0)	0.907
Area SVG azimuth, °	1.8 (−3.1 to 6.7)	0.475	−11.3 (−15.9 to −6.6)	<0.0001
SAI QRST, mV*ms	18.4 (13.7 to 23.0)	<0.0001	14.0 (9.8 to 18.0)	<0.0001
Peak SVG magnitude, mV	0.26 (0.21 to 0.30)	<0.0001	0.22 (0.18 to 0.26)	<0.0001
SVG magnitude, mV	0.30 (0.25 to 0.34)	<0.0001	0.28 (0.23 to 0.33)	<0.0001
Cornell voltage, mV	0.30 (0.25 to 0.35)	<0.0001	0.22 (0.18 to 0.27)	<0.0001
QRS duration, ms	−0.4 (−1.6 to 0.7)	0.440	−3.3 (−4.4 to −2.2)	<0.0001
QT interval, ms	2.4 (0.5 to 4.2)	0.011	−0.6 (−2.3 to 1.1)	0.517
Heart rate, bpm	−0.8 (−1.8 to 0.2)	0.098	−3.2 (−4.2 to −2.3)	<0.0001

Linear regression model 1 was adjusted for age, sex, and study center. Model 2 was additionally adjusted for prevalent heart failure, coronary heart disease, stroke, diabetes mellitus, hypertension, current smoking and alcohol intake, work, sport, and leisure physical activity levels, levels of total cholesterol, high-density lipoprotein, and triglycerides, body mass index, use of antihypertensive and antiarrhythmic medications, serum concentrations of sodium, potassium, calcium, magnesium, phosphorus, and uric acid, total protein and albumin, blood urea nitrogen, chronic kidney disease stage classified by estimated glomerular filtration rate (calculated using the Chronic Kidney Disease–Epidemiology Collaboration equation), education level, occupation category, income, health insurance, heart rate, QRS, QTc, and Cornell voltage. Bpm indicates beats per minute; °, degrees; GEH, global electrical heterogeneity; SAI QRST, sum absolute QRST integral; SVG, spatial ventricular gradient; QTc, corrected QT.

the exact date of ECG recording. The span of time was either from one ECG recording to another ECG recording or from an ECG recording to the primary outcome, competing death outcome, or the last known follow-up (censored). Race-stratified associations of continuous ECG variables with SCD were also studied using adjusted (model 1) Cox regression models incorporating cubic splines with 4 knots.

To compare competing risks of SCD and non-SCD, we constructed 2 Fine and Gray's competing risk models[38] for SCD and non-SCD outcomes, using the same covariates as in Cox models. We calculated the relative sub–hazard ratio with 95% CI of SCD risk for black relative to white individuals, assuming sub–hazard ratio for the white participants is a reference.

On the basis of a recent study demonstrating that race significantly modified association of hypertension, CHD, and BMI with SCD,[1] we additionally constructed sets of models that included 2-way interactions with race and hypertension, race and CHD, and race and BMI categories. Because of the small size of the underweight BMI category, for 2-by-2 interaction analysis, we lumped together underweight and normal-weight BMI categories and referenced them as normal weight. The white hypertension-free, white CHD-free, and white normal-weight subgroups were the reference subgroups in respective models. The 2-by-2 interaction analysis was performed for global ECG variables that were significantly different by race (voltage-based global ECG metrics), or if race significantly modified their association with SCD (spatial QRS-T angle).

Statistical analyses were performed using STATA MP 16.1 (StataCorp LP, College Station, TX). Considering the many multivariate analyses performed, statistical significance at the 0.05 level should be interpreted cautiously.

Results

Study Population

Black study participants (Table 1) were slightly younger, with a higher prevalence of HF, stroke, and major risk factors (hypertension, diabetes mellitus, and smoking) than white individuals. White participants had a higher prevalence of CHD, had a higher level of triglycerides, were less physically active at work, but were more physically active at leisure and sport than black participants. There were large disparities between races in access to health care, income, education, and occupation.

Differences in ECG Metrics Between Black and White Participants

SVG magnitude, SAI QRST, and Cornell voltage were significantly larger in black than white individuals, even after adjustment for confounders (Table 2 and Figure 2). The

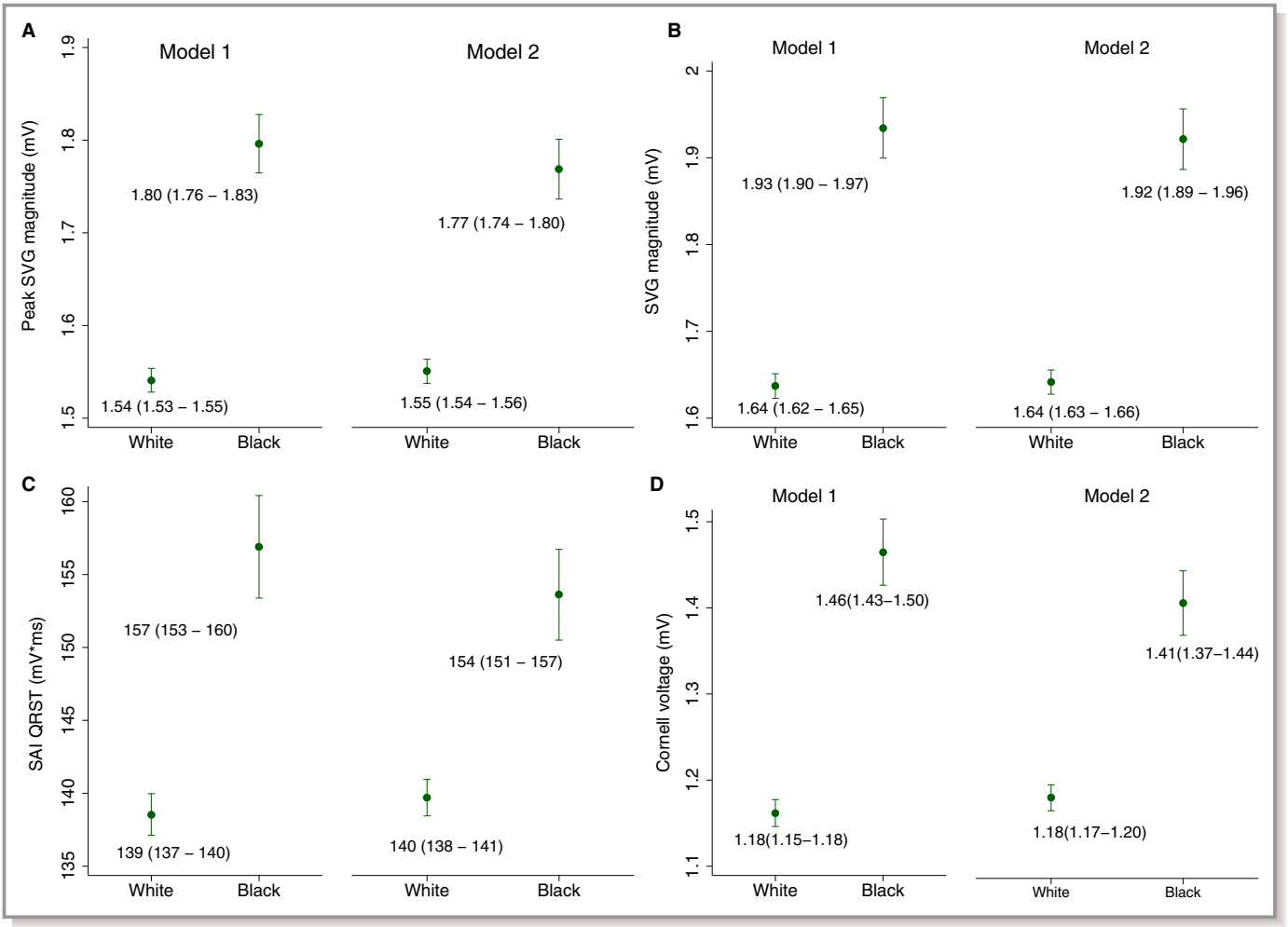

Figure 2. Estimated adjusted marginal (least-squares) means and 95% CIs of peak spatial ventricular gradient (SVG) magnitude (**A**), area SVG magnitude (**B**), sum absolute QRST integral (SAI QRST) (**C**), and Cornell voltage (**D**) for white and black participants. Model 1 was adjusted for age, sex, and study center. Model 2 was additionally adjusted for heart failure, coronary heart disease, stroke, diabetes mellitus, hypertension, current smoking and alcohol intake, work, sport, and leisure physical activity levels, levels of total cholesterol, high-density lipoprotein, and triglycerides, body mass index, use of antihypertensive and antiarrhythmic medications, serum concentrations of sodium, potassium, calcium, magnesium, phosphorus, and uric acid, total protein and albumin, blood urea nitrogen, and chronic kidney disease stage classified by estimated glomerular filtration rate (calculated using the Chronic Kidney Disease–Epidemiology Collaboration equation), heart rate, QRS duration, Bazett-corrected QT interval, Cornell voltage, education level, occupation category, income, and health insurance.

spatial QRS-T angle and direction of SVG, as well as QTc, were similar in white and black participants (Figures S1 and S2).

Over a median follow-up of 24.4 years, there were 522 SCDs (incidence, 1.74 per 1000 person-years; 95% CI, 1.59–1.89 per 1000 person-years) and 2147 non-SCDs (incidence, 7.14 per 1000 person-years; 95% CI, 6.85–7.45 per 1000 person-years). SCD incidence was 2 times higher in black (2.86 per 1000 person-years; 95% CI, 2.50–3.28 per 1000 person-years) than white participants (1.37 per 1000 person-years; 95% CI, 1.22–1.53 per 1000 person-years). Incidence of non-SCD was higher in black (9.60 per 1000 person-years; 95% CI, 8.92–10.33 per 1000 person-years) than white participants (6.33 per 1000 person-years; 95% CI, 6.01–6.67 per 1000 person-years).

Considering hypertension subgroups, the lowest SCD incidence was observed in hypertension-free white individuals (0.95 per 1000 person-years; 95% CI, 0.82–1.11 per 1000 person-years), followed by hypertension-free black individuals (1.31 per 1000 person-years; 95% CI, 0.98–1.75 per 1000 person-years), and then by white individuals with hypertension (2.59 per 1000 person-years; 95% CI, 2.20–3.04 per 1000 person-years). Black individuals with hypertension had the highest rate of SCD: 4.26 per 1000 person-years (95% CI, 3.66–4.96 per 1000 person-years) (Figure 3).

Among CHD subgroups, CHD-free white participants had the lowest SCD incidence (1.01 per 1000 person-years; 95% CI, 0.89–1.16 per 1000 person-years), followed by CHD-free

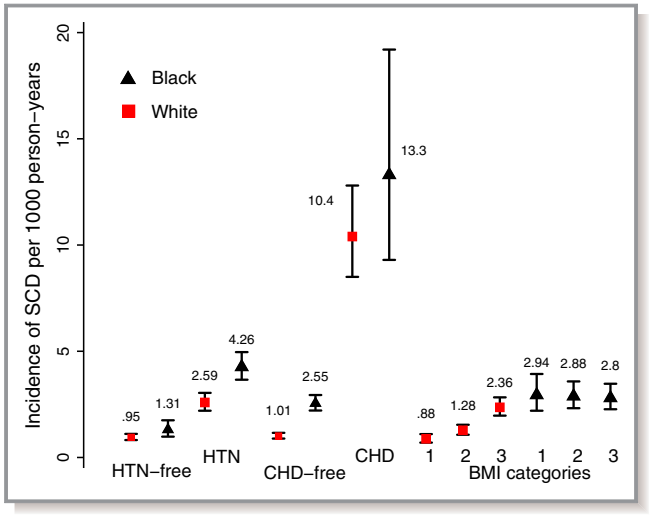

Figure 3. Incidence of sudden cardiac death (SCD) per 1000 person-years in black (black triangles) and white (red quadrants) participants with and without hypertension (HTN) and coronary heart disease (CHD), and by body mass index (BMI) subgroups. Black lines correspond to 95% CI bounds.

black participants (2.55 per 1000 person-years; 95% CI, 2.21–2.94 per 1000 person-years). SCD incidence was dramatically higher in participants with CHD (both white [10.4 per 1000 person-years; 95% CI, 8.5–12.8 per 1000 person-years] and black participants [13.3 per 1000 person-years; 95% CI, 9.3–19.2 per 1000 person-years]).

In BMI categories subgroups, normal-weight white participants had the lowest incidence of SCD (0.88 per 1000 person-years; 95% CI, 0.70–1.10 per 1000 person-years), followed by overweight white individuals (1.28 per 1000 person-years; 95% CI, 1.07–1.54 per 1000 person-years), and then by obese white individuals (2.36 per 1000 person-years; 95% CI, 1.97–2.83 per 1000 person-years). Incidence of SCD in black individuals with any BMI was significantly higher than in white individuals: 2.94 per 1000 person-years (95% CI, 2.20–3.93 per 1000 person-years) in normal-weight individuals, 2.88 per 1000 person-years (95% CI, 2.32–3.58 per 1000 person-years) in overweight individuals, and 2.80 per 1000 person-years (95% CI, 2.27–3.47 per 1000 person-years) in obese black participants (Figure 3).

Association of Electrophysiological Substrate With SCD in Black and White Participants

All traditional ECG metrics, SVG direction, and SAI QRST were associated with a similar risk of SCD in black and white participants, a finding that was consistent in both Cox regression (Tables S1 and S2) and competing risk models (Tables S3 and S4).

Time-updated Cox regression model 2 revealed significant interaction with SVG magnitude (Figure 4). However, competing risk analysis found no effect modification by race of SVG magnitude and SCD association (Figure 4 and Table S3). Continuous hazard functions were similar for white and black individuals (Figure S3), suggesting an absence of meaningful effect modification.

Race did not modify associations of ECG measures with non-SCD (Tables S3 and S4).

Race and Hypertension Modify an Association of Spatial QRS-T Angle With SCD

Race significantly modified an association of spatial QRS-T angle with SCD, in both Cox regression (Figure 4 and Table S1) and competing risk models (Figure 4 and Table S3). The spatial QRS-T angle was associated with ≈20% reduced risk of SCD for black participants compared with white participants. Adjustment for incident nonfatal cardiovascular disease in Cox model 2 attenuated the effect modification (Figure 4). In race-stratified Cox regression (Figure 5 and Table S2) and competing risk models 1 and 2 (Figure 6 and Table S4), spatial QRS-T angle was associated with SCD in white, but not black, participants (Figure 7).

Further analysis of the interaction with both race and hypertension showed that spatial QRS-T angle was associated with SCD similarly in white and hypertension-free black participants (Figure 8 and Table S5), but not in black participants with hypertension. Effect modification by race and hypertension remained significant after adjustment for incident cardiovascular events in both Cox regression (Figure 9 and Table S5) and competing risk models (Table S6).

Two-Way Interaction With Race and Prevalent CHD

In Cox and competing risk models 1, SAI QRST had a stronger association with SCD in white participants with CHD, compared with CHD-free white participants (Tables S5 and S6). In contrast, in black CHD participants, SAI QRST was associated with less SCD risk (Figure 10), but greater incidence of non-SCD (relative sub–hazard ratio, 1.29; 95% CI, 1.13–1.47; $P<0.0001$ in competing non-SCD risk model 2). As expected, after adjustment for incident cardiovascular events, there was no significant 2-way interaction with race-CHD category in association of ECG measures with SCD (Tables S5 and S6).

Race and BMI Modify Associations of Spatial QRS-T Angle and SVG Magnitude With SCD

Analysis of the interaction with both race and BMI showed that spatial QRS-T angle was associated with SCD similarly in white and obese black participants, but not in normal-weight

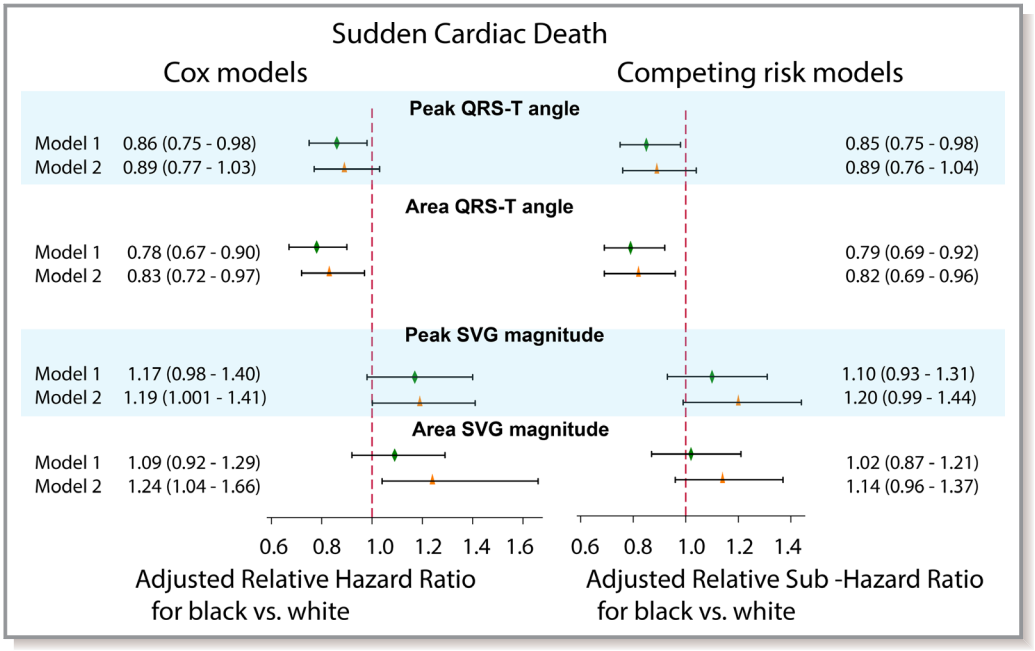

Figure 4. Adjusted Cox proportional relative hazard ratio and competing sudden cardiac death risk relative sub–hazard risk ratio with 95% CI for black compared with white participants, with hazard ratio/sub–hazard ratio for white participants equal 1.0. Models 1 (green diamond) and models 2 (orange triangle) for the QRS-T angle and spatial ventricular gradient (SVG) magnitude are shown. Black lines correspond to 95% CI bounds. Model 1 was adjusted for age, sex, study center, prevalent heart failure (HF), coronary heart disease (CHD), stroke, diabetes mellitus, hypertension, levels of total cholesterol, high-density lipoprotein, and triglycerides, body mass index, use of antihypertensive and antiarrhythmic medications, serum concentrations of sodium, potassium, calcium, magnesium, phosphorus, and uric acid, total protein and albumin, blood urea nitrogen, chronic kidney disease stage classified by estimated glomerular filtration rate (calculated using the Chronic Kidney Disease–Epidemiology Collaboration equation), smoking and alcohol intake, work, sport, and leisure physical activity levels, education level, occupation category, income, and health insurance. Time-updated model 2 included time-updated ECG predictors (one by one), all baseline covariates included in model 1, and time-updated incident nonfatal atrial fibrillation, HF, CHD, and stroke.

or overweight black participants, as shown in both Cox regression and competing risk analyses (Figure 9 and Tables S5 through S8).

After adjustment for incident cardiovascular events, all global voltage-based ECG metrics (SVG magnitude, Cornell voltage, and SAI QRST) had a significantly stronger association with SCD in obese black participants, compared with normal-weight white participants (Figure 10 and Tables S5 through S8).

Discussion

This large, prospective, community-based cohort study of >14 000 participants showed several important findings. Apart from the QRS-T angle, the association of electrophysiological substrate with SCD was similar in black and white individuals. In this study, race did not modify an association of electrophysiological substrate with SCD, supporting the recognition of race as a product of social practices, but not an inherent characteristic of individuals. Similar strength of the association of global ECG metrics with SCD in black and white individuals implies that electrophysiological substrate does not explain racial disparities in SCD rate.

Furthermore, we observed that black individuals with hypertension experienced 4.5-fold higher SCD incidence than hypertension-free white individuals. Black race in the presence of hypertension is associated with high SCD risk regardless of QRS-T angle, which explains the weaker association of QRS-T angle with SCD in black than white individuals. However, relative risk of SCD carried by QRS-T angle did not differ in hypertension-free black or white individuals with or without hypertension. We also observed that black individuals had larger amplitude-based ECG metrics (SAI QRST, SVG magnitude, and Cornell voltage) compared with white individuals. The difference of ≈0.3 mV was robustly observed, even after accounting for socioeconomic status and other confounders. This finding is consistent with previous studies[6,7] and supports use of race-specific thresholds for abnormal SAI QRST,[13] SVG magnitudes,[13] and ECG LVH.[8,10] Moreover, we showed that SAI QRST, SVG

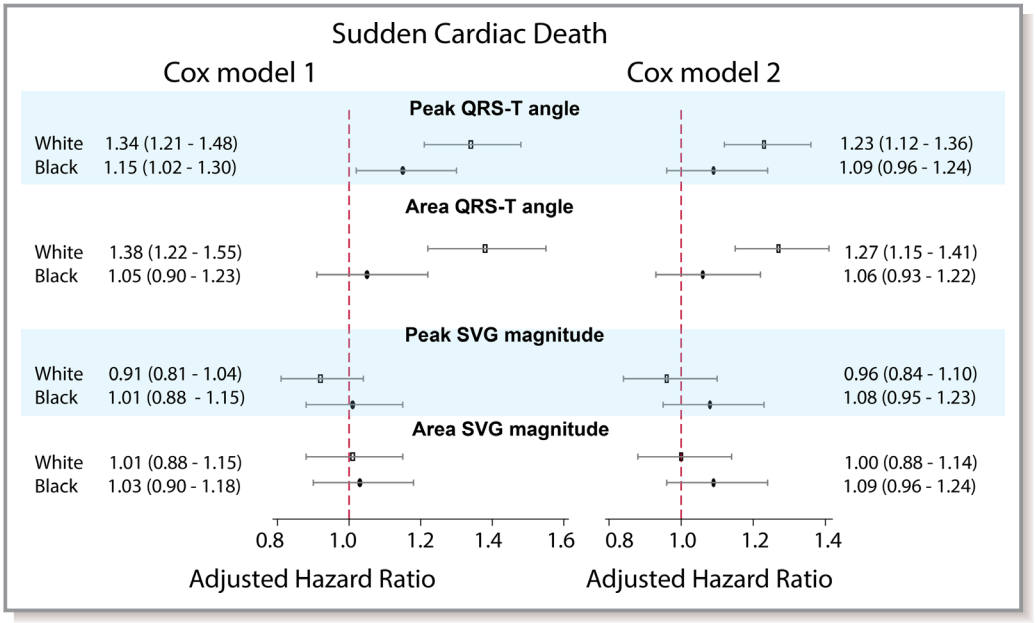

Figure 5. Race-stratified adjusted (models 1 and 2, as described in Figure 4 legend) Cox proportional hazard ratio and 95% CI of sudden cardiac death for QRS-T angle and spatial ventricular gradient (SVG) magnitude in white (hollow rectangle) and black (black oval) individuals. Black lines correspond to 95% CI bounds.

magnitude, and Cornell voltage convey higher risk of SCD in obese black individuals, compared with normal-weight white individuals, by 20% to 50%. Prevention of obesity in black individuals with increased SVG magnitude, SAI QRST, or Cornell voltage can be potentially lifesaving. A randomized clinical trial is warranted to test this hypothesis.

Race Does Not Modify an Association of Electrophysiological Substrate With SCD

Our study found that all traditional ECG metrics (QTc, QRS duration, and Cornell voltage), SVG direction, and SAI QRST were associated with a similar risk of SCD among black and

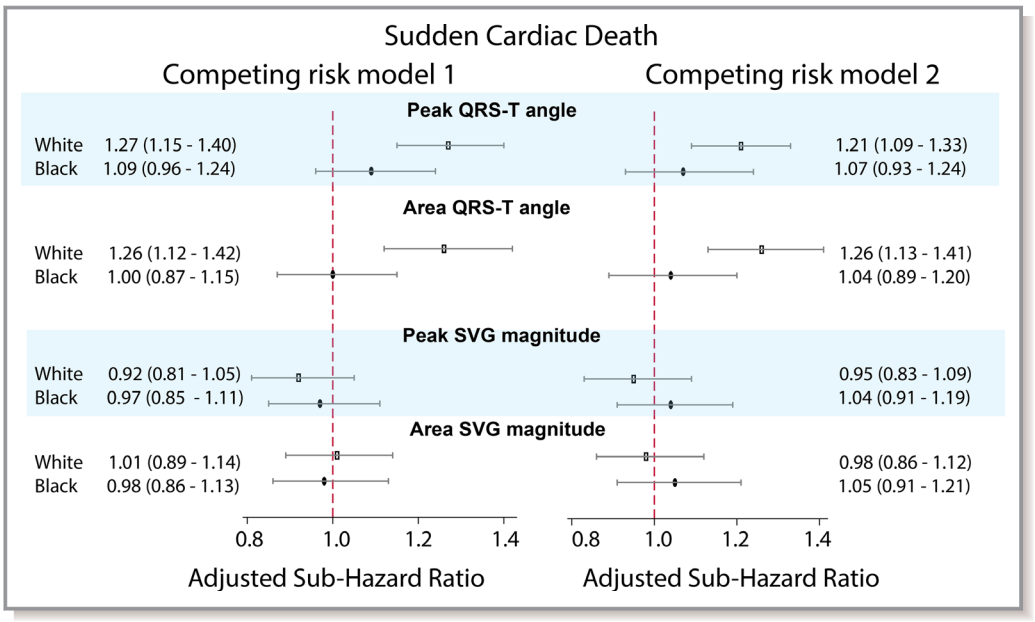

Figure 6. Adjusted (models 1 and 2, as described in Figure 4 legend) competing risk sub–hazard ratio and 95% CI of sudden cardiac death for QRS-T angle and spatial ventricular gradient (SVG) magnitude in white (hollow rectangle) and black (black oval) individuals. Black lines correspond to 95% CI bounds.

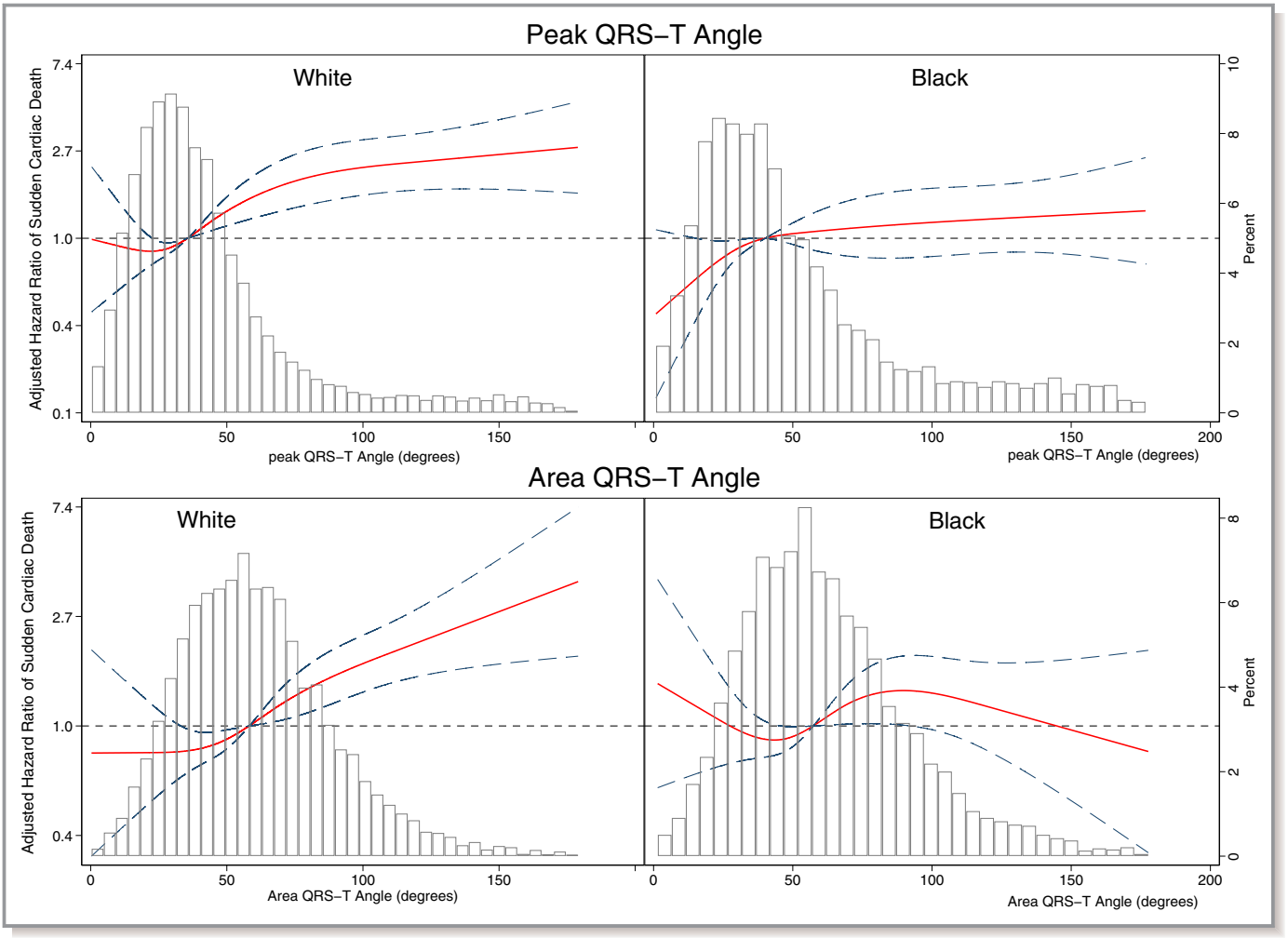

Figure 7. Adjusted (model 1, as described in Figure 4 legend) risk of sudden cardiac death associated with area and peak QRS-T angle in black and white participants. Restricted cubic spline with 95% CI shows a change in the hazard ratio (*y* axis) in response to QRS-T angle change (*x* axis). The 50th percentile of QRS-T angle is selected as a reference. Knots of peak QRS-T angle in white participants are at 10° to 28° to 44° to 118°, and in black participants are at 11° to 31° to 53° to 149°. Knots of area QRS-T angle in white participants are at 22° to 48° to 69° to 112°, and in black participants are at 21° to 48° to 69° to 118°.

white individuals. There is little existing literature studying the association of ECG metrics and the risk of SCD by race, and this study provides new insight into racial differences and similarities. In our prior study on sex differences,[39] we found that Cornell voltage, SVG magnitude, and SAI QRST are associated with a 16% to 24% greater risk of SCD in women compared with men. Sex is biologically determined and therefore may cause differences in electrophysiological substrate in contrast to race, which is a complex social construct.

There has been growing recognition of the Critical Race Theory,[20] which aims to better account for the effects of institutionalized racism on health. The results of our study support the Critical Race Theory postulate. Many previous studies recognized race as an independent and strong risk factor of SCD, even after adjusting for socioeconomic factors but attributed it, at least partially, to underlying biological differences between black and white individuals.[1,2,4] In contrast, our comprehensive study of electrophysiological substrate did not find meaningful effect modification by race. Results of our study call attention to structural racism as an important determinant of the increased SCD rate in black compared with white individuals.[40]

Several studies investigated the effects of racism on physical health. These studies showed that self-reported daily discrimination and stress was associated with increased waist circumference, increased waist/hip ratio, and higher fasting glucose level.[41–43] Not only does daily stress increase potential risk factors for SCD, but long-term negative emotions also increase vulnerability to life-threatening arrhythmias[44] and can potentially contribute to racial disparities in SCD. Consistent with previous studies, we observed 2-by-2

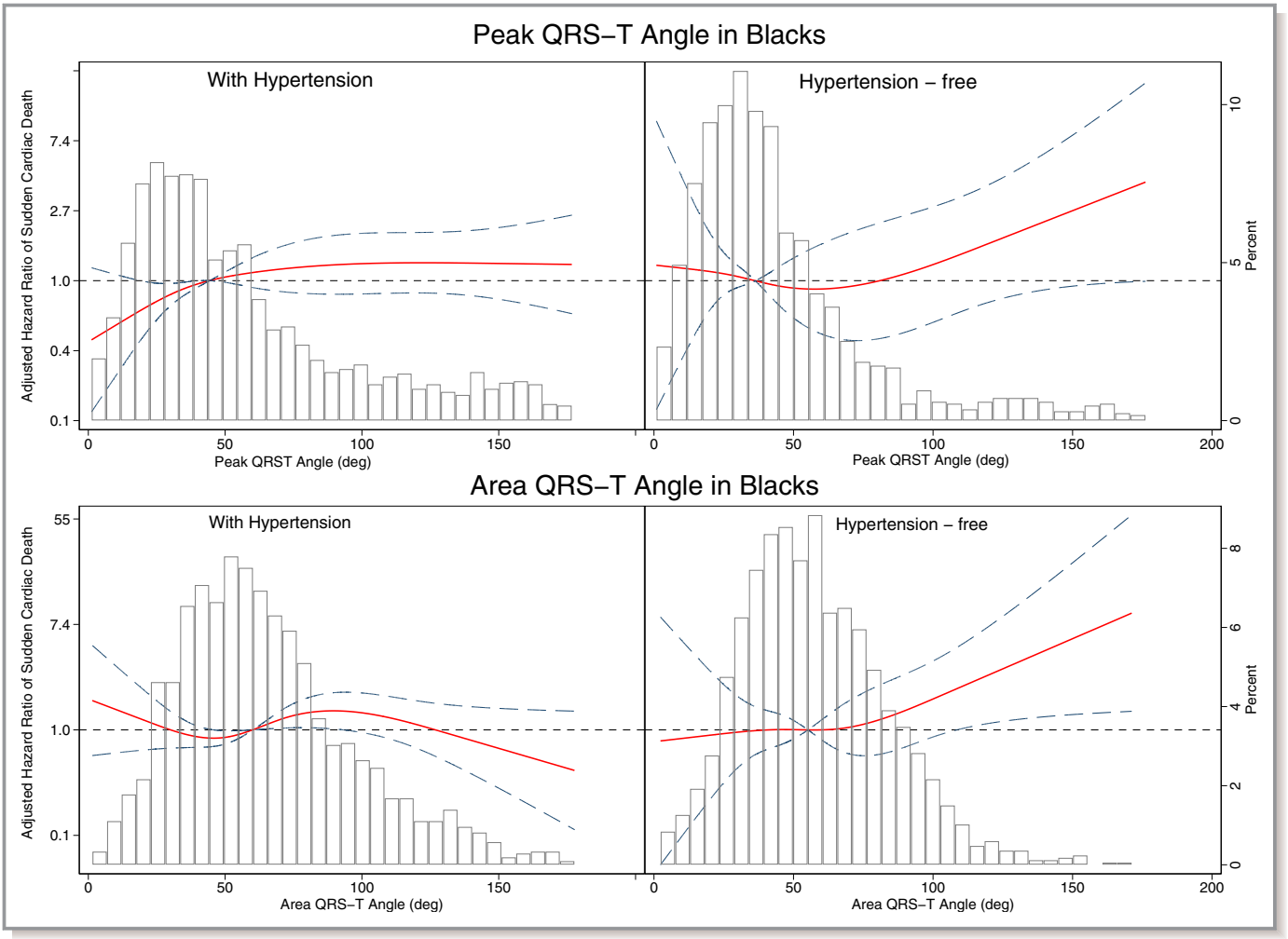

Figure 8. Adjusted (model 1, as described in Figure 4 legend) risk of sudden cardiac death associated with area and peak QRS-T angle in black participants with and without hypertension. Restricted cubic spline with 95% CI shows a change in the hazard ratio (*y* axis) in response to QRS-T angle change (*x* axis). The 50th percentile of QRS-T angle is selected as reference. Knots of peak QRS-T angle are at 10° to 29° to 45° to 120°. Knots of area QRS-T angle in black participants with hypertension are at 21° to 50° to 72° to 130°, and in hypertension-free black participants are at 20° to 46° to 66° to 103°.

interaction with race and BMI. Disproportionally high risk of SCD was associated with all voltage-based ECG metrics (Cornell voltage, SAI QRST, and SVG magnitude) in black obese (but not in black overweight or normal-weight) individuals.

When thinking about race and potential biological differences determining health, it is important to consider that race is not equivalent to genetic ancestry, but as mentioned above is a social construct. In our study, participants self-identified as black or white, but no ancestry information was analyzed. Ancestry analyses of the self-reported racial and ethnic identity in the United States have shown that self-identified black individuals carry up to 24% of European ancestry, and ≈1 in 10 self-identified white individuals in the US South have at least 1% of African ancestry.[45] Association of genetic ancestry with electrophysiological substrate of SCD[4] deserves separate investigation.

Race-Specific Thresholds of ECG Voltage Measurements and Excess of SCD Risk in Obese Black Individuals

Racial differences in ECG voltage and ECG LVH definition have been previously reported.[6–8] This study showed consistent results: voltage-based ECG metrics (SVG magnitude, SAI QRST, and Cornell voltage) were larger in black than white individuals, by 0.2 to 0.3 mV. Comprehensive adjustment for cardiovascular risk factors in this study did not affect the racial differences in SVG magnitude, SAI QRST, and Cornell voltage. Similarly, the DHS (Dallas Heart Study) reported[46] that after adjustment for cardiovascular risk factors and body composition, both black race and African ancestry were associated with ≈0.25-mV larger ECG voltage. A recent genome-wide association study of GEH revealed 10 GEH-

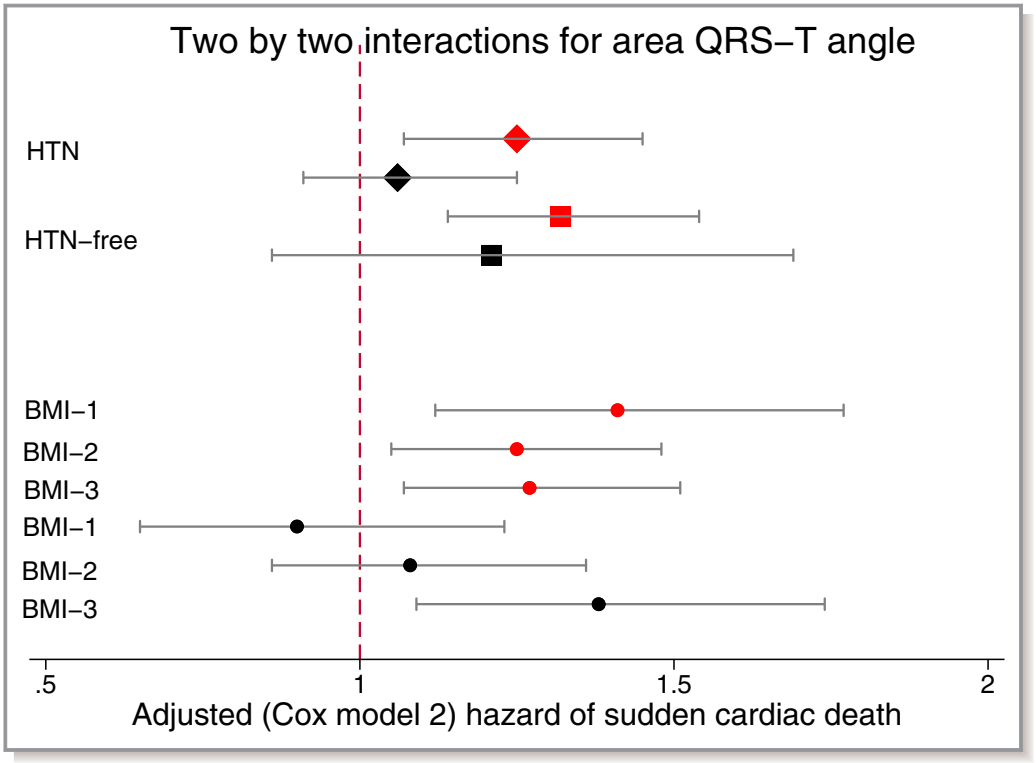

Figure 9. The 2-by-2 interactions for area QRS-T angle. Adjusted (model 2, as described in Figure 4 legend) Cox proportional hazard ratio and 95% CI of sudden cardiac death in white (red) and black (black) individuals with (diamonds) and without (quadrants) hypertension (HTN). Circles indicate body mass index (BMI) subgroups. Black lines correspond to 95% CI bounds.

associated loci.[47] Four loci (11p11.2 cluster, near *ACTB*, *LUZP1-KDM1A*, and *IGF1R*) were associated with increase in both SAI QRST and SVG magnitude. SAI QRST–associated locus near *LUZP1-KDM1A* has higher effect allele frequency[47] in African ancestry (0.92) compared with European ancestry (0.59), which can potentially contribute to larger SAI QRST in a black population. Further studies are needed to test these hypotheses.

More important, we showed that all ECG voltage-based metrics carry excess SCD risk in obese black individuals. Prevention of obesity in black individuals with increased SVG magnitude, SAI QRST, or Cornell voltage can be potentially lifesaving, and should be investigated in randomized clinical trials. Furthermore, development of race-specific SCD risk stratification is needed, to incorporate race-specific thresholds of ECG voltage-based metrics and observed heterogeneity of response.

Two-Way Interaction of SAI QRST With Race and Prevalent CHD

Previous studies showed that in the predominantly white population of patients with HF, larger SAI QRST was associated with increased risk of ventricular tachyarrhythmias,[48] whereas in a mixed population that included black patients with HF, smaller SAI QRST was associated with increased risk of sustained ventricular tachyarrhythmias.[49,50] Results of this study confirmed 2-by-2 interaction of SAI QRST with race and prevalent CHD, showing opposite directions of associations of SAI QRST with SCD in black and white participants with CHD. In white patients with CHD, large SAI QRST conveys increased SCD risk, whereas in black patients with CHD, large SAI QRST indicates risk of competing nonsudden cardiovascular death, rather than SCD. In black individuals, CHD is likely diagnosed later, at more advanced stages, and treated less effectively, compared with white individuals, which can explain observed competing risk differences.

Strengths and Limitations

The strengths of our study included the sizable community-dwelling cohort, the extended follow-up, and the rigorous adjudication of SCD. We were also able to adjust for time-varying ECG measurements and incident nonfatal cardiovascular events and conduct competing risk analyses. However, our study has limitations, as previously acknowledged.[13] Although efforts were made to differentiate SCD from non-

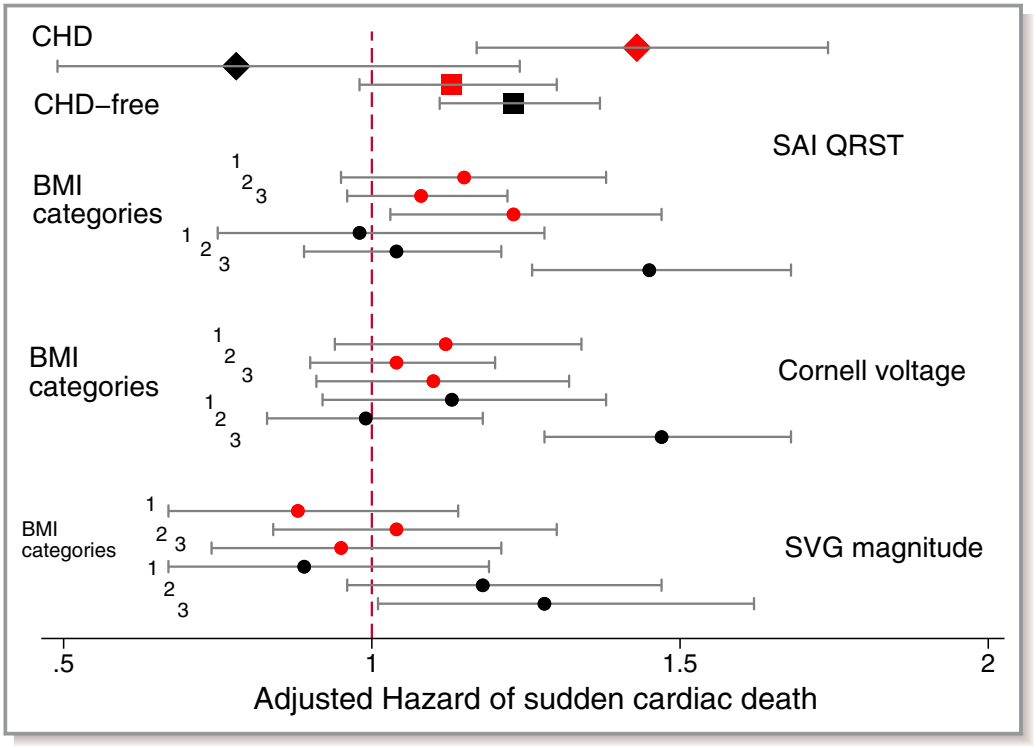

Figure 10. The 2-by-2 interactions for voltage-based ECG metrics (sum absolute QRST integral [SAI QRST], peak spatial ventricular gradient [SVG] magnitude, and Cornell voltage). Adjusted Cox proportional hazard ratio and 95% CI of sudden cardiac death in white (red) and black (black) individuals with (diamonds) and without (quadrants) coronary heart disease (CHD) (Cox model 1). Circles indicate body mass index (BMI) subgroups (Cox model 2). Black lines correspond to 95% CI bounds. Models as described in Figure 4 legend.

SCD, it is possible than some SCD events were secondary to sudden noncardiac catastrophe as opposed to ventricular arrhythmias. However, these events likely compromise a small fraction of SCD events and therefore should not significantly affect study results. This study did not include resuscitated out-of-hospital cardiac arrest because of difficulty accurately differentiating its cause. Another important consideration is the inclusion of only black and white individuals, which limits generalizability to multiethnic populations. Further studies in other races and ethnicities are needed to validate our findings. The results of 2-way interaction analyses should be interpreted with caution. As in any observational study, residual confounding is a limitation of the study.

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Disclosures

None.

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SUPPLEMENTAL MATERIAL

Table S1. Race interaction in association of global ECG measures with SCD in Cox models.

	Predictor, per 1 SD	All (n=14,408; 522 SCDs)			
		HR(95%CI)	P-value	RHR for black vs. white pts (95%CI)	$P_{interaction}$
Model 1	Peak QRS-T angle	**1.34(1.22-1.47)**	**<0.0001**	**0.86(0.75-0.98)**	**0.026**
	Area QRS-T angle	**1.35(1.21-1.51)**	**<0.0001**	**0.78(0.67-0.90)**	**0.001**
	Peak SVG elevation	**1.13(1.01-1.26)**	**0.027**	0.92(0.78-1.09)	0.348
	Area SVG elevation	**1.16(1.06-1.28)**	**0.002**	0.88(0.75-1.04)	0.136
	Peak SVG azimuth	**1.11(1.01-1.21)**	**0.026**	1.01(0.88-1.15)	0.940
	Area SVG azimuth	0.99(0.90-1.09)	0.878	1.07(0.92-1.25)	0.389
	Peak SVG magnitude	0.89(0.79-1.01)	0.083	1.17(0.98-1.40)	0.083
	Area SVG magnitude	0.98(0.86-1.11)	0.753	1.09(0.92-1.29)	0.314
	SAI QRST	**1.21(1.08-1.35)**	**0.001**	0.89(0.79-1.01)	0.078
	Heart rate	1.11(0.99-1.24)	0.081	1.01(0.86-1.19)	0.907
	Bazett's QTc	**1.12(1.01-1.24)**	**0.032**	1.01(0.89-1.14)	0.924
	QRS duration	1.07(0.98-1.18)	0.128	0.93(0.81-1.07)	0.304
	Cornell voltage	1.10(0.98-1.24)	0.104	1.01(0.87-1.18)	0.893
Model 2	Peak QRS-T angle	**1.24(1.12-1.36)**	**<0.0001**	0.89(0.77-1.03)	0.121
	Area QRS-T angle	**1.27(1.15-1.40)**	**<0.0001**	**0.83(0.72-0.97)**	**0.020**
	Peak SVG elevation	**1.14(1.03-1.27)**	**0.011**	0.96(0.81-1.15)	0.675
	Area SVG elevation	**1.12(1.01-1.23)**	**0.035**	0.97(0.81-1.16)	0.726
	Peak SVG azimuth	**1.12(1.02-1.22)#**	**0.015**	1.04(0.90-1.21)	0.602
	Area SVG azimuth	1.04(0.95-1.15)	0.387	1.11(0.93-1.32)	0.246
	Peak SVG magnitude	0.98(0.86-1.11)	0.720	**1.19(1.001-1.41)**	**0.048**
	Area SVG magnitude	0.93(0.82-1.07)	0.310	**1.24(1.04-1.66)**	**0.018**
	SAI QRST	**1.10(1.02-1.19)**	**0.013**	1.01(0.91-1.12)	0.865
	Heart rate	1.11(0.99-1.24)	0.078	1.04(0.89-1.23)	0.600
	Bazett's QTc	**1.16(1.06-1.27)#**	**0.002**	1.01(0.88-1.15)	0.916
	QRS duration	**1.18(1.09-1.29)**	**<0.0001**	0.96(0.84-1.10)	0.575
	Cornell voltage	1.06(0.97-1.16)	0.187	1.09(0.97-1.23)	0.135

#Proportionality hazards assumption not met; SVG=spatial ventricular gradient: RHR=relative hazard ratio.

Table S2. Association of GEH with SCD in Cox models for white and black.

	Predictor, per 1 SD	White (n=10,669; 309 SCDs)		Black (n=3,739; 213 SCDs)	
		HR(95%CI)	P-value	HR(95%CI)	P-value
Model 1	Peak QRS-T angle	**1.34(1.21-1.48)**	**<0.0001**	**1.15(1.02-1.30)**	**0.018**
	Area QRS-T angle	**1.38(1.22-1.55)**	**<0.0001**	1.05(0.91-1.22)	0.467
	Peak SVG elevation	**1.13(1.01-1.27)**	**0.032**	1.05(0.90-1.23)	0.523
	Area SVG elevation	**1.17(1.06-1.30)**	**0.002**	1.05(0.90-1.21)	0.555
	Peak SVG azimuth	**1.10(1.003-1.21)**	**0.044**	1.10(0.97-1.26)	0.129
	Area SVG azimuth	0.99(0.90-1.10)	0.901	1.05(0.91-1.22)	0.471
	Peak SVG magnitude	0.92(0.81-1.04)	0.198	1.01(0.88-1.15)	0.915
	Area SVG magnitude	1.01(0.88-1.15)	0.935	1.03(0.90-1.18)	0.625
	SAI QRST	**1.23(1.08-1.39)**	**0.001**	1.07(0.95-1.21)	0.270
	Heart rate	1.11(0.98-1.26)	0.090	**1.16(1.02-1.32)**	**0.029**
	Bazett's QTc	**1.18(1.03-1.29)**	**0.010**	**1.11(1.002-1.23)**	**0.046**
	QRS duration	1.06(0.95-1.18)	0.272	1.04(0.92-1.18)	0.510
	Cornell voltage	1.07(0.95-1.22)	0.256	**1.15(1.02-1.30)**	**0.018**
Model 2	Peak QRS-T angle	**1.23(1.12-1.36)**	**<0.0001**	1.09(0.96-1.24)	0.171
	Area QRS-T angle	**1.27(1.15-1.41)**	**<0.0001**	1.06(0.93-1.22)	0.373
	Peak SVG elevation	**1.16(1.04-1.29)**	**0.006**	1.12(0.96-1.30)	0.147
	Area SVG elevation	**1.13(1.02-1.25)**	**0.021**	1.09(0.94-1.28)	0.251
	Peak SVG azimuth	**1.11(1.01-1.21)**	**0.022**	**1.16(1.02-1.32)**	**0.023**
	Area SVG azimuth	**1.05(0.95-1.16)**	**0.359**	1.15(0.99-1.34) #	0.074
	Peak SVG magnitude	0.96(0.84-1.10)	0.595	1.08(0.95-1.23)	0.236
	Area SVG magnitude	1.00(0.88-1.14)	0.964	1.09(0.96-1.24)	0.183
	SAI QRST	**1.13(1.04-1.22)**	**0.004**	**1.11(1.01-1.21)**	**0.023**
	Heart rate	1.09(0.97-1.23)	0.134	**1.20(1.06-1.36)**	**0.005**
	Bazett's QTc	**1.19(1.09-1.30)**	**<0.0001**	**1.16(1.05-1.29)**	**0.004**
	QRS duration	**1.18(1.08-1.29)**	**<0.0001**	**1.15(1.03-1.30)**	**0.015**
	Cornell voltage	1.08(0.99-1.19)	0.082	**1.16(1.06-1.27)**	**0.001**

#Proportionality hazards assumption not met; SVG=spatial ventricular gradient.

Table S3. Race interaction in association of GEH with SCD and nonSCD in competing risk models.

	Predictor, per 1 SD	SCD (n=14,408; 522 SCDs)				nonSCD (n=14,408; 2,147 nonSCDs)			
		SHR(95%CI)	P-value	RSHR for black vs.white (95%CI)	$P_{interaction}$	SHR(95%CI)	P-value	RSHR for black vs.white (95%CI)	$P_{interaction}$
Model 1	Peak QRS-T angle	**1.28(1.16-1.40)**	**<0.0001**	**0.85(0.75-0.98)**	**0.022**	**1.11(1.05-1.17)**	**<0.0001**	1.01(0.93-1.09)	0.979
	Area QRS-T angle	**1.25(1.13-1.39)**	**<0.0001**	**0.79(0.69-0.92)**	**0.002**	**1.15(1.08-1.22)**	**<0.0001**	1.00(0.91-1.09)	0.914
	Peak SVG elevation	1.11(0.99-1.25)	0.075	0.96(0.80-1.14)	0.630	1.00(0.95-1.06)	0.869	0.930(0.84-1.03)	0.148
	Area SVG elevation	**1.14(1.03-1.25)**	**0.010**	0.94(0.80-1.11)	0.471	1.00(0.94-1.05)	0.904	0.96(0.87-1.05)	0.398
	Peak SVG azimuth	1.09(0.99-1.19)	0.071	1.02(0.89-1.18)	0.755	1.04(0.99-1.09)	0.168	0.99(0.91-1.09)	0.890
	Area SVG azimuth	0.98(0.88-1.08)	0.640	1.07(0.91-1.64)	0.426	**1.07(1.01-1.13)**	**0.017**	0.99(0.90-1.09)	0.837
	Peak SVG magnitude	0.90(0.79-1.02)	0.096	1.10(0.93-1.31)	0.263	0.96(0.90-1.02)	0.170	1.10(0.99-1.21)	0.073
	Area SVG magnitude	0.98(0.87-1.11)	0.727	1.02(0.87-1.21)	0.792	0.98(0.92-1.05)	0.608	1.08(0.98-1.19)	0.114
	SAI QRST	**1.13(1.02-1.26)**	**0.019**	0.88(0.77-1.01)	0.076	1.05(0.98-1.13)	0.192	1.01(0.93-1.10)	0.751
	Heart rate.	1.03(0.92-1.16)	0.576	0.99(0.84-1.17)	0.918	**1.13(1.07-1.20)**	**<0.0001**	1.07(0.98-1.17)	0.128
	Bazett's QTc	1.09(0.98-1.21)	0.124	1.02(0.90-1.17)	0.737	1.05(0.996-1.11)	0.071	1.02(0.94-1.09)	0.673
	QRS duration	1.06(0.95-1.17)	0.293	0.92(0.78-1.07)	0.267	1.05(0.996-1.11)	0.069	0.98(0.90-1.06)	0.608
	Cornell voltage	1.08(0.96-1.22)	0.185	0.98(0.84-1.15)	0.815	**1.12(1.05-1.20)**	**0.001**	0.97(0.89-1.07)	0.543
Model 2	Peak QRS-T angle	**1.21(1.10-1.33)**	**<0.0001**	0.89(0.76-1.04)	0.130	**0.93(0.87-0.98)**	**0.010**	1.02(0.93-1.12)	0.706
	Area QRS-T angle	**1.26(1.13-1.41)**	**<0.0001**	**0.82(0.69-0.96)**	**0.017**	**0.92(0.87-0.97)**	**0.004**	1.02(0.92-1.13)	0.656
	Peak SVG elevation	**1.16(1.04-1.29)**	**0.007**	0.96(0.80-1.15)	0.649	**0.92(0.86-0.98)**	**0.006**	1.07(0.95-1.21)	0.281
	Area SVG elevation	**1.11(1.005-1.24)**	**0.041**	0.98(0.82-1.17)	0.849	**0.93(0.88-0.99)**	**0.031**	1.05(0.93-1.18)	0.418
	Peak SVG azimuth	**1.11(1.01-1.22)**	**0.033**	1.04(0.88-1.23)	0.651	**0.95(0.90-0.999)**	**0.044**	1.01(0.91-1.12)	0.887
	Area SVG azimuth	1.04(0.93-1.16)	0.458	1.10(0.89-1.81)	0.360	0.96(0.91-1.02)	0.171	0.97(0.87-1.07)	0.549
	Peak SVG magnitude	0.91(0.79-1.04)	0.180	1.20(0.99-1.44)	0.058	1.04(0.97-1.12)	0.273	0.94(0.84-1.05)	0.248
	Area SVG magnitude	0.99(0.83-1.08)	0.425	1.14(0.96-1.37)	0.144	1.04(0.97-1.12)	0.273	0.93(0.83-1.03)	0.179
	SAI QRST	**1.10(1.02-1.19)**	**0.011**	0.99(0.87-1.12)	0.831	0.95(0.90-1.01)	0.083	0.97(0.89-1.05)	0.447
	Heart rate.	1.03(0.92-1.15)	0.589	1.05(0.88-1.25)	0.583	**1.11(1.04-1.18)**	**0.001**	0.97(0.88-1.08)	0.590
	Bazett's QTc	**1.13(1.03-1.24)**	**0.009**	1.02(0.89-1.16)	0.792	1.02(0.97-1.09)	0.413	0.96(0.88-1.06)	0.418
	QRS duration	**1.20(1.10-1.30)**	**<0.0001**	0.96(0.83-1.10)	0.530	**0.92(0.87-0.97)**	**0.002**	0.98(0.89-1.08)	0.713
	Cornell voltage	1.05(0.96-1.15)	0.257	1.09(0.96-1.24)	0.204	0.97(0.92-1.02)	0.187	1.00(0.92-1.08)	0.937

RSHR=relative sub-hazard ratio

Table S4. Competing risks of sudden cardiac death and non-sudden cardiovascular death for white and black.

	Predictor, per 1 SD	Sudden cardiac death				Non-sudden cardiac death			
		White (n=10,669;309 SCDs)		Black (n=3,739; 213 SCDs)		White (n=10,669;309 SCDs)		Black (n=3,739; 213 SCDs)	
		SHR(95%CI)	P-value	SHR(95%CI)	P-value	SHR(95%CI)	P-value	SHR(95%CI)	P-value
Model 1	Peak QRS-T angle	**1.27(1.15-1.40)**	**<0.0001**	1.09(0.96-1.24)	0.167	**1.10(1.04-1.16)**	**0.002**	**1.15(1.07-1.23)**	**<0.0001**
	Area QRS-T angle	**1.26(1.12-1.42)**	**<0.0001**	1.00(0.87-1.15)	0.975	**1.14(1.07-1.21)**	**<0.0001**	**1.19(1.09-1.29)**	**<0.0001**
	Peak SVG elevation	1.10(0.97-1.26)	0.130	1.09(0.93-1.29)	0.293	0.99(0.93-1.05)	0.783	0.96(0.87-1.06)	0.403
	Area SVG elevation	**1.15(1.04-1.27)**	**0.008**	1.09(0.94-1.27)	0.257	0.98(0.93-1.04)	0.559	1.00(0.92-1.09)	0.959
	Peak SVG azimuth	1.08(0.98-1.19)	0.116	1.12(0.98-1.28)	0.095	1.04(0.99-1.10)	0.137	1.01(0.93-1.11)	0.746
	Area SVG azimuth	0.97(0.87-1.08)	0.594	1.04(0.89-1.22)	0.625	**1.08(1.02-1.14)**	**0.013**	1.05(0.97-1.15)	0.220
	Peak SVG magnitude	0.92(0.81-1.05)	0.229	0.97(0.85-1.11)	0.636	0.97(0.91-1.04)	0.362	1.03(0.96-1.12)	0.344
	Area SVG magnitude	1.01(0.89-1.14)	0.933	0.98(0.86-1.13)	0.823	0.99(0.93-1.07)	0.885	1.06(0.98-1.15)	0.122
	SAI QRST	**1.16(1.03-1.31)**	**0.012**	1.00(0.88-1.15)	0.953	1.04(0.96-1.12)	0.325	**1.11(1.03-1.20)**	**0.010**
	Heart rate.	1.05(0.93-1.19)	0.450	1.04(0.91-1.19)	0.526	**1.14(1.08-1.21)**	**<0.0001**	**1.19(1.10-1.28)**	**<0.0001**
	Bazett's QTc	1.11(0.99-1.25)	0.069	1.10(0.99-1.22)	0.066	**1.06(1.00-1.12)**	**0.037**	1.05(0.98-1.12)	0.180
	QRS duration	1.05(0.94-1.17)	0.420	1.01(0.88-1.16)	0.880	1.03(0.97-1.09)	0.371	1.07(0.99-1.16)	0.085
	Cornell voltage	1.05(0.93-1.19)	0.460	1.09(0.97-1.23)	0.140	**1.12(1.04-1.20)**	**0.002**	**1.11(1.03-1.06)**	**0.006**
Model 2	Peak QRS-T angle	**1.21(1.09-1.33)**	**<0.0001**	1.07(0.93-1.24)	0.340	**0.93(0.87-0.98)**	**0.014**	0.94(0.86-1.02)	0.153
	Area QRS-T angle	**1.26(1.13-1.41)**	**<0.0001**	1.04(0.89-1.20)	0.644	**0.92(0.87-0.98)**	**0.008**	0.92(0.84-1.02)	0.115
	Peak SVG elevation	**1.17(1.05-1.30)**	**0.005**	1.12(0.95-1.32)	0.195	**0.91(0.85-0.97)**	**0.003**	1.01(0.91-1.13)	0.793
	Area SVG elevation	**1.13(1.02-1.26)**	**0.020**	1.10(0.94-1.30)	0.243	**0.93(0.87-0.99)**	**0.032**	1.01(0.91-1.12)	0.812
	Peak SVG azimuth	**1.10(1.004-1.22)**	**0.042**	1.16(0.99-1.35)	0.068	0.95(0.90-1.00)	0.061	0.94(0.85-1.04)	0.254
	Area SVG azimuth	1.04(0.93-1.16)	0.453	1.14(0.95-1.38)	0.169	0.97(0.92-1.02)	0.229	**0.91(0.82-0.998)**	**0.046**
	Peak SVG magnitude	0.95(0.83-1.09)	0.455	1.04(0.91-1.19)	0.541	1.04(0.97-1.23)	0.262	0.96(0.88-1.04)	0.336
	Area SVG magnitude	0.98(0.86-1.12)	0.791	1.05(0.91-1.21)	0.530	1.04(0.97-1.12)	0.245	0.95(0.87-1.03)	0.222
	SAI QRST	**1.11(1.03-1.20)**	**0.007**	1.09(0.97-1.23)	0.148	0.95(0.90-1.01)	0.079	**0.92(0.86-0.99)**	**0.018**
	Heart rate	1.03(0.92-1.16)	0.630	1.12(0.97-1.30)	0.114	**1.12(1.06-1.19)**	**<0.0001**	1.06(0.98-1.16)	0.158
	Bazett's QTc	**1.15(1.05-1.27)**	**0.003**	**1.15(1.03-1.28)**	**0.011**	1.02(0.97-1.08)	0.439	1.02(0.93-1.08)	0.953
	QRS duration	**1.19(1.09-1.30)**	**<0.0001**	**1.16(1.02-1.31)**	**0.019**	**0.91(0.86-0.96)**	**0.001**	0.92(0.84-1.002)	0.056
	Cornell voltage	1.07(0.97-1.17)	0.159	**1.14(1.03-1.27)**	**0.015**	0.97(0.92-1.02)	0.182	0.96(0.89-1.03)	0.224

Table S5. Two-way interactions in association of global ECG measures with SCD in Cox models: race-hypertension; race-coronary heart disease, and race-BMI category.

Predictor, per 1 SD	Subgroup	Cox model 1 RHR(95%CI)	$P_{interaction}$	Cox model 2 RHR (95%CI)	$P_{interaction}$
Peak QRS-T angle	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	0.87(0.66-1.14)	0.304	0.83(0.62-1.12)	0.229
	White HTN(148/2737)	0.94(0.79-1.12)	0.507	0.88(0.74-1.06)	0.170
	Black HTN(167/1910)	**0.82(0.70-0.97)**	**0.021**	**0.82(0.69-0.98)**	**0.029**
Area QRS-T angle	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	1.04(0.71-1.53)	0.681	0.93(0.68-1.26)	0.631
	White HTN(148/2737)	0.93(0.77-1.13)#	0.458	0.92(0.76-1.10)	0.359
	Black HTN(167/1910)	**0.71(0.59-0.86)**	**<0.0001**	**0.76(0.62-0.91)**	**0.004**
Peak SVG magnitude	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	**1.44(1.04-2.01)**	**0.030**	**1.49(1.08-2.05)**	**0.014**
	White HTN(148/2737)	1.26(0.98-1.61)#	0.066	1.18(0.91-1.54)	0.203
	Black HTN(167/1910)	**1.32(1.05-1.65)**	**0.016**	**1.30(1.03-1.63)**	**0.026**
SVG magnitude	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	1.35(0.98-1.85)	0.070	**1.41(1.03-1.92)**	**0.031**
	White HTN(148/2737)	1.28(0.99-1.64)#	0.055	1.17(0.91-1.51)	0.218
	Black HTN(167/1910)	1.25(0.999-1.56)	0.051	1.25(0.999-1.56)	0.051
SAI QRST	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	1.19(0.88-1.61)	0.254	1.19(0.91-1.57)	0.204
	White HTN(148/2737)	1.16(0.95-1.41)	0.140	0.997(0.85-1.17)	0.996
	Black HTN(167/1910)	0.95(0.80-1.14)	0.580	0.98(0.85-1.14)	0.821
Cornell voltage	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	0.95(0.68-1.32)	0.748	0.95(0.69-1.33)	0.781
	White HTN(148/2737)	1.08(0.86-1.36)	0.514	1.02(0.85-1.22)	0.812
	Black HTN(167/1910)	1.04(0.84-1.28)	0.712	1.11(0.95-1.31)	0.200
Peak QRS-T angle	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	0.98(0.84-1.15)	0.823	0.96(0.82-1.13)	0.640
	White CHD(88/534)	1.18(0.97-1.43)	0.100	1.12(0.91-1.40)	0.286
	Black CHD(29/148)	0.98(0.84-1.15)	0.823	0.82(0.57-1.17)	0.270
Area QRS-T angle	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	0.89(0.75-1.07)	0.225	0.88(0.73-1.05)	0.159
	White CHD(88/534)	**1.24(1.01-1.52)**	**0.043**	1.09(0.88-4.35)	0.437
	Black CHD(29/148)	0.82(0.58-1.14)	0.237	0.87(0.62-1.23)	0.434
Peak SVG magnitude	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	**1.22(1.001-1.48)**	**0.049**	1.18(0.97-1.43)	0.100
	White CHD(88/534)	0.95(0.71-1.26)	0.706	0.88(0.63-1.21)	0.423
	Black CHD(29/148)	0.90(0.60-1.35)	0.603	1.36(0.88-2.09)	0.167
SVG magnitude	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	1.19(0.98-1.44)	0.080	1.16(0.96-1.40)	0.128
	White CHD(88/534)	1.06(0.80-1.41)	0.701	0.90(0.67-1.21)	0.501
	Black CHD(29/148)	0.83(0.57-1.22)	0.348	1.10(0.72-1.67)	0.664
SAI QRST	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	1.07(0.91-1.26)	0.404	1.08(0.95-1.23)	0.219
	White CHD(88/534)	**1.26(1.01-1.56)**	**0.038**	1.13(0.94-1.36)	0.207

	Black CHD(29/148)	**0.71(0.51-0.99)**	**0.045**	0.95(0.76-1.19)	0.653
Cornell voltage	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	0.97(0.82-1.16)	0.763	1.09(0.96-1.24)	0.198
	White CHD(88/534)	0.79(0.62-1.003)	0.053	0.94(0.77-1.13)	0.491
	Black CHD(29/148)	0.78(0.57-1.08)	0.130	0.95(0.70-1.30)	0.757
Peak QRS-T angle	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	1.00(0.80-1.24)	0.979	0.93(0.74-1.17)	0.519
	White BMI-3(117/2426)	0.97(0.78-1.21)	0.785	0.88(0.69-1.10)	0.275
	Black BMI-1(46/832)	0.81(0.59-1.10)	0.169	0.83(0.61-1.14)	0.245
	Black BMI-2(82/1397)	0.77(0.60-0.97)	0.028	**0.70(0.54-0.90)**	**0.006**
	Black BMI-3(85/1510)	0.99(0.79-1.24)	0.920	1.02(0.79-1.33)	0.858
Area QRS-T angle	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	0.81(0.63-1.04)	0.098	0.90(0.71-1.14)	0.391
	White BMI-3(117/2426)	0.80(0.63-1.02)	0.077	0.88(0.69-1.12)	0.301
	Black BMI-1(46/832)	**0.57(0.41-0.81)**	**0.002**	**0.71(0.51-0.98)**	**0.040**
	Black BMI-2(82/1397)	**0.65(0.49-0.85)**	**0.002**	**0.67(0.51-0.87)**	**0.003**
	Black BMI-3(85/1510)	**0.73(0.56-0.95)**	**0.018**	0.95(0.72-1.25)	0.703
Peak SVG magnitude	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	1.10(0.80-1.50)	0.566	1.25(0.91-1.72)	0.177
	White BMI-3(117/2426)	1.08(0.79-1.47)	0.646	1.11(0.79-1.55)	0.555
	Black BMI-1(46/832)	1.18(0.85-1.64)	0.321	1.11(0.79-1.56)	0.545
	Black BMI-2(82/1397)	1.14(0.83-1.58)	0.414	**1.38(1.01-1.88)**	**0.043**
	Black BMI-3(85/1510)	1.38(0.995-1.90)	0.054	**1.51(1.10-2.07)**	**0.010**
SVG magnitude	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	1.03(0.74-1.42)	0.867	1.13(0.83-1.54)	0.431
	White BMI-3(117/2426)	1.00(0.72-1.37)	0.981	0.97(0.70-1.36)	0.872
	Black BMI-1(46/832)	1.07(0.78-1.47)	0.668	0.96(0.70-1.33)	0.822
	Black BMI-2(82/1397)	1.02(0.74-1.40)	0.918	1.22(0.90-1.65)	0.191
	Black BMI-3(85/1510)	1.32(0.95-1.84)	0.098	1.35(0.98-1.86)	0.062
SAI QRST	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	1.03(0.81-1.30)	0.825	0.97(0.80-1.18)	0.762
	White BMI-3(117/2426)	1.02(0.80-1.31)	0.864	1.06(0.84-1.33)	0.643
	Black BMI-1(46/832)	0.77(0.58-1.02)	0.069	0.88(0.68-1.15)	0.356
	Black BMI-2(82/1397)	**0.75(0.59-0.95)**	**0.019**	0.87(0.70-1.08)	0.194
	Black BMI-3(85/1510)	1.12(0.91-1.40)	0.288	**1.22(0.998-1.49)**	**0.052**
Cornell voltage	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	1.01(0.75-1.35)	0.968	0.95(0.78-1.17)	0.656
	White BMI-3(117/2426)	1.05(0.78-1.41)	0.749	0.91(0.71-1.15)	0.421
	Black BMI-1(46/832)	1.00(0.74-1.36)	0.995	1.01(0.811-1.27)	0.905
	Black BMI-2(82/1397)	0.96(0.73-1.27)	0.791	0.86(0.68-1.08)	0.200
	Black BMI-3(85/1510)	1.19(0.89-1.60)	0.246	**1.26(1.05-1.52)**	**0.015**

#Proportionality hazards assumption not met; SVG=spatial ventricular gradient: RHR=relative hazard ratio; HTN=hypertension; CHD=coronary heart disease; BMI-1=under- or normal-weight; BMI-2=overweight; BMI-3=obese.

Table S6. Two-way interactions in association of global ECG measures with SCD in competing risk models: race-hypertension; race-coronary heart disease, and race-BMI category.

Predictor, per 1 SD	Subgroup	Competing SCD risk model 1		Competing SCD risk model 2	
		RSHR(95%CI)	$P_{interaction}$	RSHR (95%CI)	$P_{interaction}$
Peak QRS-T angle	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	0.93(0.71-1.23)	0.611	0.87(0.64-1.17)	0.348
	White HTN(148/2737)	0.93(0.78-1.09)	0.368	0.87(0.72-1.04)	0.134
	Black HTN(167/1910)	**0.79(0.67-0.93)**	**0.005**	**0.80(0.67-0.96)**	**0.019**
Area QRS-T angle	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	1.04(0.74-1.44)	0.834	0.97(0.71-1.33)	0.850
	White HTN(148/2737)	0.93(0.77-1.12)	0.448	0.90(0.74-1.10)	0.320
	Black HTN(167/1910)	**0.71(0.59-0.86)**	**<0.0001**	**0.72(0.59-0.89)**	**0.002**
Peak SVG magnitude	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	**1.42(1.07-1.89)**	**0.017**	**1.50(1.07-2.09)**	**0.018**
	White HTN(148/2737)	1.26(0.99-1.60)	0.063	1.15(0.87-1.51)	0.326
	Black HTN(167/1910)	1.19(0.96-1.48)	0.120	1.22(0.97-1.53)	0.090
SVG magnitude	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	**1.32(1.00-1.76)**	**0.050**	**1.44(1.05-1.96)**	**0.023**
	White HTN(148/2737)	**1.27(1.01-1.61)**	**0.043**	1.16(0.90-1.51)	0.255
	Black HTN(167/1910)	1.12(0.90-1.38)	0.314	1.18(0.94-1.48)	0.155
SAI QRST	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	1.22(0.94-1.59)	0.132	1.25(0.96-1.63)	0.102
	White HTN(148/2737)	1.11(0.93-1.33)	0.232	1.01(0.87-1.16)	0.921
	Black HTN(167/1910)	0.90(0.75-1.08)	0.256	0.96(0.82-1.12)	0.626
Cornell voltage	White HTN-free(161/7623)	Reference (1.00)		Reference (1.00)	
	Black HTN-free(46/1616)	1.00(0.73-1.36)	0.985	1.02(0.74-1.39)	0.922
	White HTN(148/2737)	1.09(0.87-1.36)	0.457	1.01(0.86-1.20)	0.864
	Black HTN(167/1910)	1.01(0.82-1.25)	0.899	1.11(0.94-1.32)	0.208
Peak QRS-T angle	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	0.94(0.80-1.09)	0.408	0.93(0.78-1.10)	0.402
	White CHD(88/534)	1.04(0.86-1.24)	0.708	1.04(0.83-1.30)	0.762
	Black CHD(29/148)	**0.71(0.52-0.96)**	**0.027**	0.80(0.54-1.20)	0.282
Area QRS-T angle	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	0.87(0.73-1.03)	0.108	0.85(0.70-1.02)	0.087
	White CHD(88/534)	1.09(0.89-1.33)	0.396	1.04(0.82-1.32)	0.745
	Black CHD(29/148)	0.77(0.56-1.09)	0.107	0.84(0.60-1.18)	0.321
Peak SVG magnitude	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	1.14(0.95-1.38)	0.160	1.17(0.95-1.43)	0.132
	White CHD(88/534)	0.94(0.70-1.26)	0.681	0.86(0.61-1.20)	0.366
	Black CHD(29/148)	0.76(0.51-1.14)	0.182	1.02(0.64-1.62)	0.926
SVG magnitude	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	1.12(0.93-1.35)	0.215	1.14(0.94-1.39)	0.197
	White CHD(88/534)	1.03(0.78-1.37)	0.815	0.89(0.65-1.21)	0.443
	Black CHD(29/148)	0.69(0.47-1.01)	0.059	0.88(0.54-1.42)	0.595
	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	

SAI QRST	Black CHD-free(184/3591)	1.02(0.87-1.18)	0.842	1.06(0.92-1.22)	0.395
	White CHD(88/534)	1.07(0.87-1.31)	0.506	1.05(0.86-1.27)	0.631
	Black CHD(29/148)	**0.64(0.47-0.88)**	**0.007**	0.86(0.64-1.17)	0.337
Cornell voltage	White CHD-free(221/10135)	Reference (1.00)		Reference (1.00)	
	Black CHD-free(184/3591)	0.96(0.81-1.13)	0.052	1.09(0.95-1.25)	0.219
	White CHD(88/534)	**0.76(0.60-0.98)**	**0.037**	0.90(0.75-1.08)	0.256
	Black CHD(29/148)	0.72(0.81-1.13)	0.615	0.91(0.54-1.53)	0.729
Peak QRS-T angle	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	0.94(0.76-1.15)	0.546	0.87(0.69-1.09)	0.232
	White BMI-3(117/2426)	0.91(0.74-1.12)	0.362	0.83(0.65-1.05)	0.112
	Black BMI-1(46/832)	**0.73(0.54-0.99)**	**0.043**	0.78(0.57-1.07)	0.125
	Black BMI-2(82/1397)	**0.72(0.57-0.92)**	**0.008**	**0.68(0.52-0.88)**	**0.004**
	Black BMI-3(85/1510)	0.94(0.75-1.17)	0.562	0.96(0.73-1.26)	0.755
Area QRS-T angle	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	0.82(0.65-1.04)	0.098	0.86(0.67-1.11)	0.255
	White BMI-3(117/2426)	**0.78(0.62-0.97)**	**0.029**	0.82(0.63-1.06)	0.136
	Black BMI-1(46/832)	**0.57(0.41-0.78)**	**0.001**	**0.65(0.46-0.90)**	**0.009**
	Black BMI-2(82/1397)	**0.64(0.49-0.83)**	**0.001**	**0.65(0.49-0.87)**	**0.004**
	Black BMI-3(85/1510)	**0.75(0.58-0.96)**	**0.024**	0.86(0.64-1.17)	0.336
Peak SVG magnitude	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	1.16(0.86-1.57)	0.328	1.28(0.93-1.76)	0.132
	White BMI-3(117/2426)	1.13(0.85-1.49)	0.411	1.15(0.81-1.64)	0.438
	Black BMI-1(46/832)	1.14(0.87-1.51)	0.343	1.11(0.79-1.55)	0.555
	Black BMI-2(82/1397)	1.14(0.84-1.54)	0.403	**1.43(1.02-2.01)**	**0.036**
	Black BMI-3(85/1510)	1.25(0.94-1.67)	0.130	**1.44(1.07-1.94)**	**0.017**
SVG magnitude	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	1.13(0.84-1.52)	0.423	1.15(0.84-1.58)	0.377
	White BMI-3(117/2426)	1.10(0.82-1.47)	0.527	1.04(0.73-1.47)	0.847
	Black BMI-1(46/832)	0.99(0.74-1.32)	0.922	0.96(0.69-1.34)	0.800
	Black BMI-2(82/1397)	0.99(0.73-1.34)	0.956	1.27(0.90-1.79)	0.178
	Black BMI-3(85/1510)	1.16(0.86-1.57)	0.328	1.27(0.92-1.75)	0.152
SAI QRST	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	1.02(0.84-1.25)	0.813	0.99(0.83-1.19)	0.959
	White BMI-3(117/2426)	0.99(0.80-1.23)	0.923	1.04(0.83-1.29)	0.754
	Black BMI-1(46/832)	0.76(0.57-1.02)	0.070	0.86(0.62-1.20)	0.373
	Black BMI-2(82/1397)	**0.76(0.63-0.93)**	**0.006**	0.86(0.70-1.06)	0.148
	Black BMI-3(85/1510)	1.06(0.84-1.33)	0.637	**1.24(1.01-1.51)**	**0.037**
Cornell voltage	White BMI-1(75/3951)	Reference (1.00)		Reference (1.00)	
	White BMI-2(117/4292)	1.03(0.79-1.33)	0.643	0.95(0.78-1.15)	0.591
	White BMI-3(117/2426)	1.02(0.76-1.37)	0.897	0.87(0.68-1.12)	0.287
	Black BMI-1(46/832)	0.97(0.73-1.28)	0.812	1.00(0.78-1.27)	0.992
	Black BMI-2(82/1397)	0.96(0.74-1.24)	0.739	0.83(0.64-1.07)	0.150
	Black BMI-3(85/1510)	1.14(0.85-1.52)	0.375	**1.31(1.10-1.56)**	**0.003**

#Proportionality hazards assumption not met; SVG=spatial ventricular gradient: RHR=relative hazard ratio; HTN=hypertension; CHD=coronary heart disease; BMI-1=under- or normal-weight; BMI-2=overweight; BMI-3=obese.

Table S7. Stratified association of global ECG measures with SCD in Cox models: race-hypertension; race-coronary heart disease, and race-BMI category subgroups.

Predictor, per 1 SD	Subgroup	Cox model 1		Cox model 2	
		HR(95%CI)	P	HR (95%CI)	P
Peak QRS-T angle	White HTN-free(161/7623)	**1.34(1.17-1.54)**	**<0.0001**	**1.30(1.13-1.49)**	**<0.0001**
	Black HTN-free(46/1616)	1.30(0.97-1.73)	0.078	1.00(0.72-1.41)	0.974
	White HTN(148/2737)	**1.37(1.21-1.57)**	**<0.0001**	**1.17(1.01-1.35)**	**0.031**
	Black HTN(167/1910)	**1.27(1.12-1.43)**	**<0.0001**	1.13(0.98-1.30)	0.087
Area QRS-T angle	White HTN-free(161/7623)	**1.36(1.17-1.58)#**	**<0.0001**	**1.32(1.14-1.54)**	**<0.0001**
	Black HTN-free(46/1616)	**1.52(1.09-2.11)**	**0.013**	1.21(0.86-1.69)	0.276
	White HTN(148/2737)	**1.41(1.22-1.63)**	**<0.0001**	**1.25(1.07-1.45)**	**0.004**
	Black HTN(167/1910)	1.15(0.99-1.32)	0.060	1.06(0.91-1.25)	0.430
Peak SVG magnitude	White HTN-free(161/7623)	**0.83(0.69-0.99)**	**0.041**	0.89(0.73-1.07)	0.203
	Black HTN-free(46/1616)	1.04(0.76-1.41)	0.826	1.14(0.85-1.51)	0.387
	White HTN(148/2737)	0.97(0.81-1.17)#	0.780	1.04(0.86-1.25)	0.703
	Black HTN(167/1910)	1.01(0.87-1.17)	0.882	1.06(0.91-1.22)	0.462
SVG magnitude	White HTN-free(161/7623)	0.90(0.75-1.08)	0.247	0.91(0.76-1.10)	0.342
	Black HTN-free(46/1616)	1.03(0.76-1.39)	0.863	1.14(0.86-1.52)	0.372
	White HTN(148/2737)	1.08(0.90-1.30)#	0.402	1.08(0.90-1.29)	0.420
	Black HTN(167/1910)	1.06(0.92-1.23)	0.422	1.08(0.94-1.26)	0.282
SAI QRST	White HTN-free(161/7623)	1.09(0.92-1.29)	0.312	1.10(0.95-1.27)	0.195
	Black HTN-free(46/1616)	1.27(0.93-1.75)	0.136	1.27(0.92-1.75)	0.153
	White HTN(148/2737)	**1.36(1.18-1.56)**	**<0.0001**	**1.15(1.03-1.28)**	**0.010**
	Black HTN(167/1910)	**1.14(1.02-1.27)**	**0.020**	**1.12(1.02-1.23)**	**0.024**
Cornell voltage	White HTN-free(161/7623)	1.04(0.86-1.25)	0.707	1.07(0.93-1.24)	0.347
	Black HTN-free(46/1616)	1.02(0.72-1.44)	0.925	0.98(0.67-1.46)	0.944
	White HTN(148/2737)	**1.23(1.05-1.44)**	**0.012**	1.12(0.99-1.27)	0.065
	Black HTN(167/1910)	**1.22(1.08-1.39)**	**0.002**	**1.19(1.08-1.31)**	**<0.0001**
Peak QRS-T angle	White CHD-free(221/10135)	**1.28(1.14-1.44)**	**<0.0001**	**1.20(1.06-1.35)**	**0.004**
	Black CHD-free(184/3591)	**1.27(1.13-1.43)**	**<0.0001**	1.10(0.96-1.26)	0.161
	White CHD(88/534)	**1.53(1.28-1.83)**	**<0.0001**	**1.35(1.10-1.65)**	**0.004**
	Black CHD(29/148)	1.24(0.81-1.88)	0.323	1.00(0.64-1.54)	0.991
Area QRS-T angle	White CHD-free(221/10135)	**1.24(1.09-1.41)**	**0.001**	**1.24(1.09-1.40)**	**0.001**
	Black CHD-free(184/3591)	**1.18(1.03-1.36)**	**0.018**	1.05(0.90-1.22)	0.545
	White CHD(88/534)	**1.67(1.38-2.01)**	**<0.0001**	**1.42(1.16-1.75)**	**0.001**
	Black CHD(29/148)	1.17(0.72-1.89)	0.535	1.53(0.97-2.44)	0.070
Peak SVG magnitude	White CHD-free(221/10135)	0.91(0.78-1.06)	0.222	0.99(0.85-1.15)	0.898
	Black CHD-free(184/3591)	1.07(0.93-1.23)	0.359	1.07(0.93-1.22)	0.353
	White CHD(88/534)	0.87(0.67-1.12)	0.279	0.90(0.66-1.23)	0.502
	Black CHD(29/148)	1.28(0.77-2.11)	0.345	1.40(0.83-1.36)	0.208
SVG magnitude	White CHD-free(221/10135)	0.98(0.84-1.15)	0.828	1.04(0.89-1.21)	0.623
	Black CHD-free(184/3591)	1.12(0.98-1.29)	0.107	1.10(0.96-1.25)	0.172
	White CHD(88/534)	1.01(0.79-1.29)	0.930	0.90(0.68-1.19)	0.467
	Black CHD(29/148)	0.99(0.59-1.65)	0.961	1.14(0.66-1.97)	0.650
SAI QRST	White CHD-free(221/10135)	1.13(0.98-1.30)	0.088	1.10(0.99-1.22)	0.064
	Black CHD-free(184/3591)	**1.23(1.11-1.37)**	**<0.0001**	**1.13(1.03-1.25)**	**0.012**
	White CHD(88/534)	**1.43(1.17-1.74)**	**<0.0001**	**1.30(1.07-1.59)**	**0.008**

	Black CHD(29/148)	0.78(0.49-1.24)	0.298	1.14(0.85-1.52)	0.370
Cornell voltage	White CHD-free(221/10135)	**1.20(1.04-1.39)**	**0.015**	**1.13(1.01-1.26)**	**0.032**
	Black CHD-free(184/3591)	**1.23(1.08-1.39)**	**0.001**	**1.17(1.07-1.29)**	**0.001**
	White CHD(88/534)	0.97(0.78-1.20)	0.756	1.05(0.87-1.27)	0.621
	Black CHD(29/148)	1.07(0.68-1.70)	0.767	1.34(0.96-1.87)	0.089
Peak QRS-T angle	White BMI-1(75/3951)	**1.24(1.01-1.51)**	**0.042**	**1.28(1.02-1.60)**	**0.029**
	White BMI-2(117/4292)	**1.40(1.20-1.64)**	**<0.0001**	**1.25(1.06-1.47)**	**0.007**
	White BMI-3(117/2426)	**1.44(1.24-1.67)**	**<0.0001**	**1.19(1.01-1.40)**	**0.038**
	Black BMI-1(46/832)	1.18(0.89-1.57)	0.242	1.05(0.78-1.42)	0.750
	Black BMI-2(82/1397)	1.12(0.93-1.34)	0.243	1.03(0.83-1.27)	0.812
	Black BMI-3(85/1510)	**1.43(1.20-1.69)**	**<0.0001**	**1.35(1.09-1.68)**	**0.006**
Area QRS-T angle	White BMI-1(75/3951)	**1.52(1.23-1.89)**	**<0.0001**	**1.41(1.12-1.77)#**	**0.004**
	White BMI-2(117/4292)	**1.31(1.10-1.56)**	**0.002**	**1.25(1.05-1.48)**	**0.012**
	White BMI-3(117/2426)	**1.39(1.17-1.65)**	**<0.0001**	**1.27(1.07-1.51)**	**0.006**
	Black BMI-1(46/832)	0.99(0.73-1.36)	0.970	0.90(0.65-1.23)	0.500
	Black BMI-2(82/1397)	1.12(0.91-1.39)	0.288	1.08(0.86-1.36)	0.506
	Black BMI-3(85/1510)	**1.31(1.07-1.59)**	**0.008**	**1.38(1.09-1.74)**	**0.007**
Peak SVG magnitude	White BMI-1(75/3951)	0.88(0.68-1.14)	0.343	0.88(0.67-1.14)	0.327
	White BMI-2(117/4292)	0.95(0.77-1.17)	0.622	1.04(0.84-1.30)	0.712
	White BMI-3(117/2426)	0.88(0.71-1.08)	0.222	0.95(0.74-1.21)	0.658
	Black BMI-1(46/832)	0.87(0.67-1.12)	0.278	0.89(0.67-1.19)	0.436
	Black BMI-2(82/1397)	0.95(0.76-1.21)	0.697	1.18(0.96-1.47)	0.121
	Black BMI-3(85/1510)	1.18(0.94-1.48)	0.154	**1.28(1.01-1.62)**	**0.040**
SVG magnitude	White BMI-1(75/3951)	0.98(0.76-1.26)	0.860	0.99(0.77-1.27)	0.911
	White BMI-2(117/4292)	1.05(0.85-1.30)	0.656	1.06(0.86-1.31)	0.566
	White BMI-3(117/2426)	0.94(0.75-1.18)	0.606	0.94(0.73-1.19)	0.591
	Black BMI-1(46/832)	0.86(0.68-1.10)	0.231	0.87(0.66-1.14)	0.317
	Black BMI-2(82/1397)	0.96(0.76-1.22)	0.748	1.23(0.996-1.52)	0.055
	Black BMI-3(85/1510)	**1.36(1.07-1.72)**	**0.012**	**1.28(1.01-1.63)**	**0.041**
SAI QRST	White BMI-1(75/3951)	1.17(0.96-1.43)	0.109	1.15(0.95-1.38)	0.147
	White BMI-2(117/4292)	**1.24(1.05-1.47)#**	**0.013**	1.08(0.96-1.22)	0.201
	White BMI-3(117/2426)	**1.24(1.02-1.50)**	**0.027**	**1.23(1.03-1.47)**	**0.026**
	Black BMI-1(46/832)	0.89(0.68-1.18)	0.426	0.98(0.75-1.28)	0.875
	Black BMI-2(82/1397)	0.94(0.78-1.14)	0.537	1.04(0.89-1.21)	0.620
	Black BMI-3(85/1510)	**1.50(1.30-1.73)**	**<0.0001**	**1.45(1.26-1.68)**	**<0.0001**
Cornell voltage	White BMI-1(75/3951)	1.03(0.81-1.31)	0.807	1.12(0.94-1.34)	0.210
	White BMI-2(117/4292)	1.14(0.94-1.39)	0.195	1.04(0.90-1.20)	0.582
	White BMI-3(117/2426)	1.15(0.94-1.42)	0.184	1.10(0.91-1.32)	0.322
	Black BMI-1(46/832)	1.12(0.89-1.40)	0.334	1.13(0.92-1.38)	0.254
	Black BMI-2(82/1397)	1.09(0.90-1.31)	0.384	0.99(0.83-1.18)	0.889
	Black BMI-3(85/1510)	**1.37(1.10-1.70)**	**0.005**	**1.47(1.28-1.68)**	**<0.0001**

#Proportionality hazards assumption not met; SVG=spatial ventricular gradient: RHR=relative hazard ratio; HTN=hypertension; CHD=coronary heart disease; BMI-1=under- or normal-weight; BMI-2=overweight; BMI-3=obese.

Table S8. Competing risk of sudden cardiac death in race-hypertension, race-coronary heart disease, and race-BMI category subgroups.

Predictor, per 1 SD	Subgroup	Competing risk model 1		Competing risk model 2	
		SHR (95%CI)	P	SHR (95%CI)	P
Peak QRS-T angle	White HTN-free(161/7784)	**1.30(1.13-1.50)**	**<0.0001**	**1.26(1.10-1.44)**	**0.001**
	Black HTN-free(46/1662)	1.23(0.86-1.75)	0.258	1.14(0.77-1.68)	0.512
	White HTN(148/2885)	**1.25(1.08-1.44)**	**0.002**	**1.17(1.00-1.36)**	**0.045**
	Black HTN(167/2077)	1.10(0.96-1.27)	0.177	1.11(0.95-1.30)	0.200
Area QRS-T angle	White HTN-free(161/7784)	**1.31(1.09-1.56)**	**0.003**	**1.31(1.10-1.54)**	**0.002**
	Black HTN-free(46/1662)	**1.51(1.01-2.25)**	**0.044**	1.37(0.95-1.97)	0.092
	White HTN(148/2885)	**1.25(1.07-1.47)**	**0.005**	**1.25(1.08-1.47)**	**0.004**
	Black HTN(167/2077)	0.96(0.82-1.12)	0.595	1.03(0.87-1.23)	0.739
Peak SVG magnitude	White HTN-free(161/7784)	0.85(0.72-1.02)	0.078	0.89(0.74-1.08)	0.229
	Black HTN-free(46/1662)	1.09(0.79-1.50)	0.603	1.16(0.85-1.58)	0.347
	White HTN(148/2885)	0.97(0.81-1.18)	0.793	0.99(0.81-1.23)	0.991
	Black HTN(167/2077)	0.94(0.80-1.10)	0.410	1.01(0.87-1.18)	0.889
SVG magnitude	White HTN-free(161/7784)	0.92(0.77-1.09)	0.339	0.90(0.75-1.09)	0.282
	Black HTN-free(46/1662)	1.04(0.73-1.46)	0.837	1.15(0.82-1.61)	0.413
	White HTN(148/2885)	1.07(0.89-1.28)	0.476	1.05(0.87-1.27)	0.628
	Black HTN(167/2077)	0.97(0.82-1.15)	0.739	1.03(0.87-1.21)	0.753
SAI QRST	White HTN-free(161/7784)	1.10(0.91-1.33)	0.312	1.08(0.95-1.23)	0.232
	Black HTN-free(46/1662)	1.25(0.84-1.87)	0.270	1.33(0.92-1.92)	0.127
	White HTN(148/2885)	1.19(1.02-1.39)	0.030	**1.16(1.04-1.30)**	**0.008**
	Black HTN(167/2077)	0.99(0.85-1.16)	0.909	1.11(0.98-1.27)	0.110
Cornell voltage	White HTN-free(161/7784)	1.00(0.80-1.24)	0.976	1.06(0.92-1.23)	0.400
	Black HTN-free(46/1662)	1.00(0.71-1.40)	0.985	1.13(0.78-1.63)	0.520
	White HTN(148/2885)	1.11(0.96-1.29)	0.166	1.12(0.99-1.27)	0.065
	Black HTN(167/2077)	1.12(0.98-1.27)	0.095	**1.18(1.05-1.33)**	**0.005**
Peak QRS-T angle	White CHD-free(221/10135)	**1.21(1.07-1.38)**	**0.002**	**1.18(1.04-1.33)**	**0.009**
	Black CHD-free(184/3591)	1.11(0.97-1.28)	0.132	1.07(0.93-1.24)	0.344
	White CHD(88/534)	**1.28(1.09-1.52)**	**0.004**	**1.23(1.001-1.51)**	**0.048**
	Black CHD(29/148)	1.21(0.68-2.15)	0.516	**54.12(3.5-832.2)**	**0.004**
Area QRS-T angle	White CHD-free(221/10135)	1.16(0.996-1.34)	0.056	**1.23(1.07-1.41)**	**0.003**
	Black CHD-free(184/3591)	0.96(0.82-1.14)	0.672	1.02(0.87-1.20)	0.787
	White CHD(88/534)	**1.36(1.10-1.67)**	**0.004**	**1.36(1.09-1.69)**	**0.005**
	Black CHD(29/148)	1.05(0.55-2.01)	0.877	1.33(0.82-2.16)	0.244
Peak SVG magnitude	White CHD-free(221/10135)	0.93(0.80-1.08)	0.346	0.98(0.84-1.15)	0.824
	Black CHD-free(184/3591)	1.02(0.89-1.18)	0.770	1.06(0.92-1.21)	0.412
	White CHD(88/534)	0.89(0.69-1.16)	0.407	0.83(0.62-1.13)	0.240
	Black CHD(29/148)	0.97(0.50-1.88)	0.919	1.04(0.62-1.73)	0.895
SVG magnitude	White CHD-free(221/10135)	1.00(0.87-1.16)	0.963	1.00(0.87-1.16)	0.963
	Black CHD-free(184/3591)	1.08(0.93-1.25)	0.337	1.08(0.93-1.25)	0.337
	White CHD(88/534)	1.06(0.83-1.34)	0.659	1.06(0.83-1.34)	0.659
	Black CHD(29/148)	0.78(0.39-1.55)	0.473	0.78(0.39-1.55)	0.473
SAI QRST	White CHD-free(221/10135)	1.13(0.97-1.31)	0.116	1.10(0.999-1.21)	0.051
	Black CHD-free(184/3591)	1.14(0.99-1.32)	0.062	**1.14(1.01-1.29)**	**0.034**
	White CHD(88/534)	1.23(0.99-1.54)	0.067	1.22(0.99-1.51)	0.064

	Black CHD(29/148)	0.59(0.32-1.10)	0.095	2.61(0.20-33.58)	0.461
Cornell voltage	White CHD-free(221/10135)	**1.21(1.04-1.42)**	**0.014**	**1.12(1.00-1.24)**	**0.043**
	Black CHD-free(184/3591)	**1.14(1.01-1.29)**	**0.034**	**1.16(1.04-1.29)**	**0.007**
	White CHD(88/534)	0.81(0.63-1.04)	0.094	0.97(0.82-1.16)	0.755
	Black CHD(29/148)	1.06(0.53-2.13)	0.873	1.30(0.79-2.14)	0.298
Peak QRS-T angle	White BMI-1(75/3951)	**1.27(1.01-1.60)**	**0.039**	**1.35(1.08-1.67)**	**0.007**
	White BMI-2(117/4292)	**1.30(0.11-1.51)**	**0.001**	**1.21(1.02-1.43)**	**0.026**
	White BMI-3(117/2426)	**1.25(1.05-1.49)**	**0.011**	1.13(0.95-1.33)	0.161
	Black BMI-1(46/832)	0.89(0.60-1.33)	0.581	1.04(0.76-1.43)	0.792
	Black BMI-2(82/1397)	1.02(0.83-1.27)	0.833	1.03(0.81-1.31)	0.829
	Black BMI-3(85/1510)	**1.24(1.02-1.51)**	**0.031**	1.27(0.99-1.63)	0.064
Area QRS-T angle	White BMI-1(75/3951)	**1.65(1.26-2.15)**	**<0.0001**	**1.48(1.16-1.90)**	**0.002**
	White BMI-2(117/4292)	**1.22(1.01-1.48)**	**0.043**	**1.24(1.03-1.50)**	**0.024**
	White BMI-3(117/2426)	1.18(0.96-1.44)	0.112	**1.23(1.01-1.48)**	**0.036**
	Black BMI-1(46/832)	0.74(0.51-1.09)	0.128	0.87(0.64-1.19)	0.383
	Black BMI-2(82/1397)	1.01(0.80-1.28)	0.917	1.10(0.85-1.43)	0.476
	Black BMI-3(85/1510)	1.10(0.88-1.37)	0.403	1.25(0.97-1.61)	0.090
Peak SVG magnitude	White BMI-1(75/3951)	0.87(0.69-1.09)	0.216	0.84(0.64-1.12)	0.234
	White BMI-2(117/4292)	0.96(0.76-1.20)	0.697	1.04(0.83-1.29)	0.740
	White BMI-3(117/2426)	0.92(0.75-1.14)	0.464	0.93(0.72-1.21)	0.601
	Black BMI-1(46/832)	0.90(0.70-1.15)	0.411	0.87(0.61-1.22)	0.414
	Black BMI-2(82/1397)	0.94(0.72-1.22)	0.637	1.18(0.90-1.55)	0.226
	Black BMI-3(85/1510)	0.97(0.76-1.24)	0.812	1.13(0.90-1.41)	0.295
SVG magnitude	White BMI-1(75/3951)	0.96(0.76-1.21)	0.708	0.94(0.72-1.24)	0.668
	White BMI-2(117/4292)	1.05(0.85-1.30)	0.654	1.04(0.85-1.28)	0.684
	White BMI-3(117/2426)	0.99(0.80-1.23)	0.946	0.92(0.72-1.18)	0.512
	Black BMI-1(46/832)	0.87(0.68-1.11)	0.263	0.84(0.61-1.15)	0.270
	Black BMI-2(82/1397)	0.95(0.72-1.24)	0.689	1.21(0.92-1.59)	0.183
	Black BMI-3(85/1510)	1.07(0.83-1.38)	0.623	1.14(0.88-1.48)	0.323
SAI QRST	White BMI-1(75/3951)	1.16(0.88-1.52)	0.286	1.14(0.96-1.37)	0.139
	White BMI-2(117/4292)	1.17(0.97-1.40)	0.101	1.09(0.97-1.22)	0.139
	White BMI-3(117/2426)	1.09(0.89-1.34)	0.410	**1.19(1.01-1.43)**	**0.041**
	Black BMI-1(46/832)	0.81(0.54-1.21)	0.305	0.97(0.65-1.46)	0.891
	Black BMI-2(82/1397)	0.89(0.72-1.09)	0.255	1.03(0.88-1.21)	0.693
	Black BMI-3(85/1510)	**1.23(1.01-1.50)**	**0.038**	**1.47(1.22-1.77)**	**<0.0001**
Cornell voltage	White BMI-1(75/3951)	0.99(0.78-1.26)	0.937	1.16(0.98-1.37)	0.083
	White BMI-2(117/4292)	1.07(0.88-1.30)	0.505	1.05(0.92-1.21)	0.474
	White BMI-3(117/2426)	1.03(0.81-1.30)	0.809	1.02(0.85-1.23)	0.815
	Black BMI-1(46/832)	1.07(0.80-1.43)	0.642	1.12(0.89-1.40)	0.353
	Black BMI-2(82/1397)	1.07(0.89-1.29)	0.477	0.97(0.80-1.18)	0.769
	Black BMI-3(85/1510)	1.15(0.90-1.47)	0.268	**1.51(1.31-1.75)**	**<0.0001**

#Proportionality hazards assumption not met; SVG=spatial ventricular gradient: RHR=relative hazard ratio; HTN=hypertension; CHD=coronary heart disease; BMI-1=under- or normal-weight; BMI-2=overweight; BMI-3=obese.

Figure S1. Estimated adjusted marginal (least-squares) means and 95% CI of (A) peak QRS-T angle, (B) area QRS-T angle, (C) peak SVG azimuth, and (D) area SVG azimuth for white and black participants.

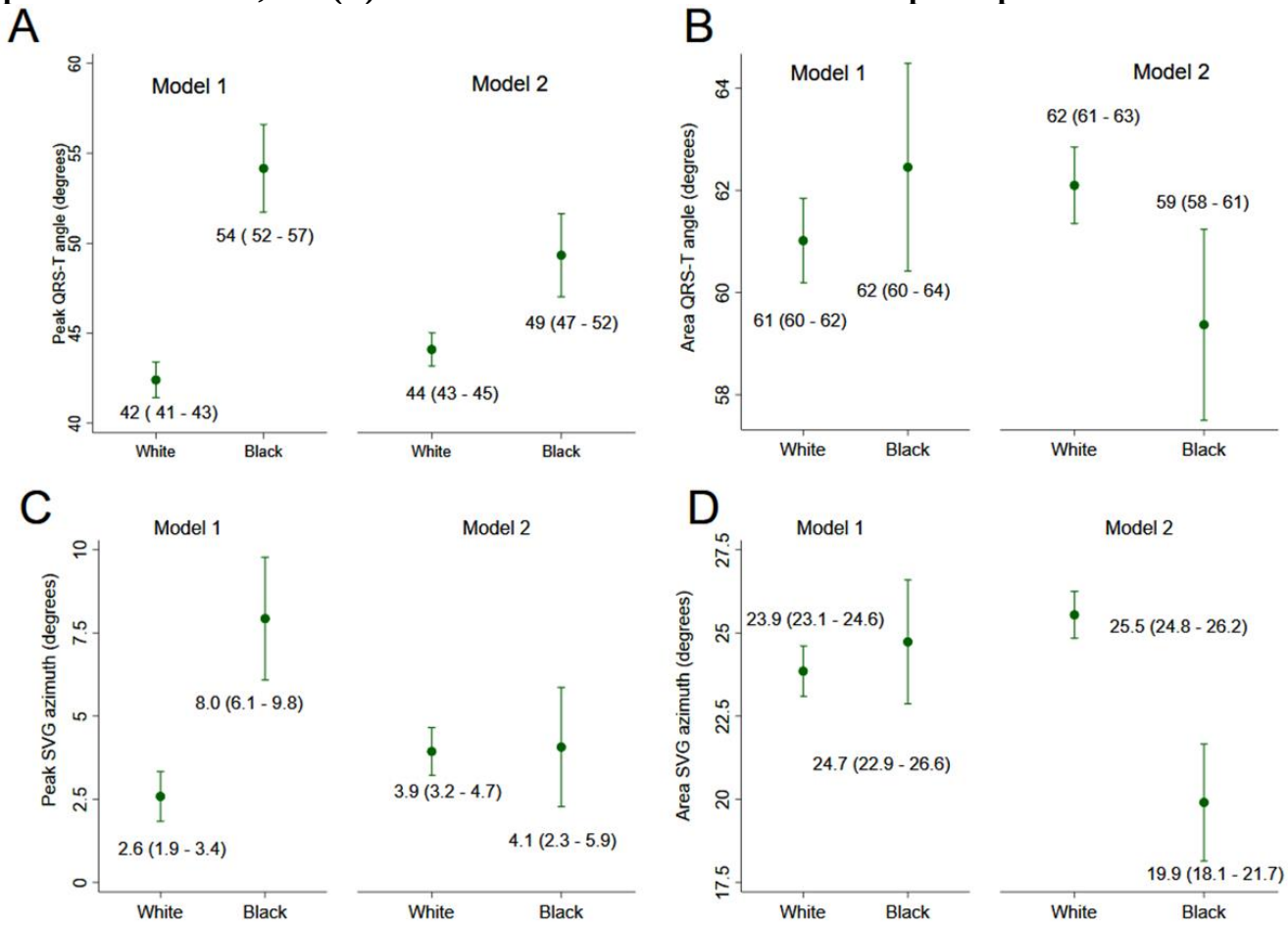

Figure S2. Estimated adjusted marginal (least-squares) means and 95% CI of (A) peak SVG elevation, (B) heart rate, (C) QRS duration, and (D) QTc for white and black participants.

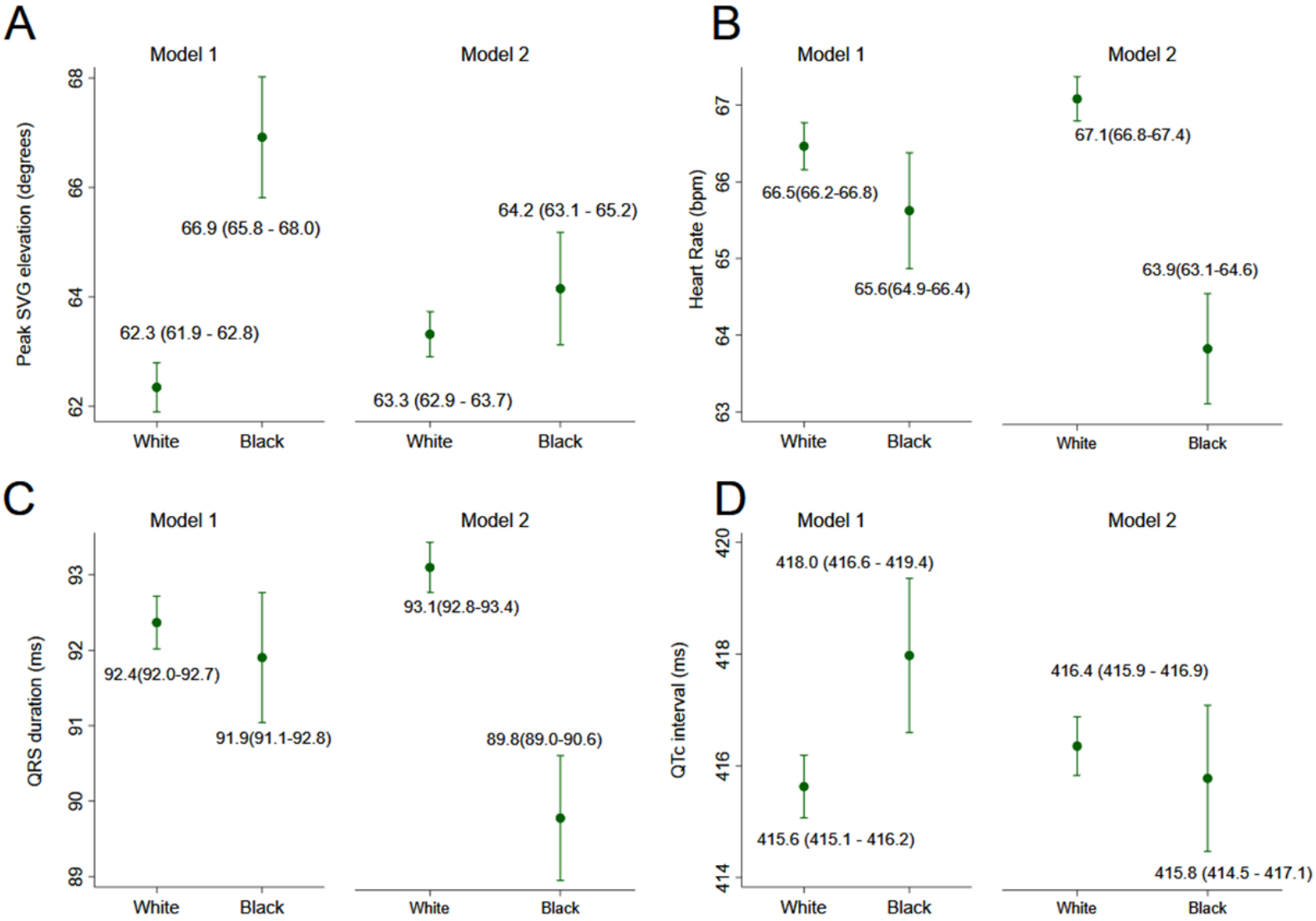

Figure S3. Adjusted (model 1) risk of SCD associated with an area and peak SVG magnitude in black and white participants. Restricted cubic spline with 95% CI shows change in hazard ratio (Y-axis) in response to SVG magnitude change (X-axis). 50th percentile of SVG magnitude is selected as reference. Knots of area SVG magnitude in black participants are at 1.2 – 1.7 – 2.1 – 2.9 mV, and in white participants are at 1.0 – 1.4 – 1.8 – 2.4 mV. Knots of peak SVG magnitude in black participants are at 1.1 – 1.6 – 2.0 – 2.6 mV, and in white participants are at 0.9 – 1.4 – 1.7 – 2.2 mV.