

NIH Public Access

Author Manuscript

J Electrocardiol. Author manuscript; available in PMC 2013 February 11.

Published in final edited form as:

J Electrocardiol. 2010; 43(5): 385.e1–385.e9. doi:10.1016/j.jelectrocard.2010.02.001.

Prevalence of Electrocardiographic Abnormalities in a Middle-Aged, Biracial Population: Coronary Artery Risk Development in Young Adults (CARDIA) Study

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Abstract

Background—Few studies to date have described the prevalence of electrocardiographic (ECG) abnormalities in a biracial middle-aged cohort.

Methods and Results—Participants underwent measurement of traditional risk factors and 12-lead ECGs coded using both Minnesota Code (MC) and Novacode (NC) criteria. Among 2585 participants, of whom 57% were women and 44% were black (mean age 45 years), the prevalence of major and minor abnormalities were significantly higher (all P<0.001) among black men and women compared to whites. These differences were primarily due to higher QRS voltage and ST/T wave abnormalities among blacks. There was also a higher prevalence of Q waves (MC 1-1, 1-2, 1-3) than described by previous studies. These racial differences remained after multivariate adjustment for traditional cardiovascular (CV) risk factors.

Conclusions—Black men and women have a significantly higher prevalence of ECG abnormalities, independent of traditional CV risk factors, than whites in a contemporary cohort middle-aged participants.

INTRODUCTION

It has been well established that major and minor ECG abnormalities are significant prognostic risk markers for incident cardiovascular disease (CVD) events, independent of traditional risk factors (1–6). The prevalence of ECG abnormalities has been best studied in cohorts of middle-aged and older white men. Data are sparse, however, regarding the prevalence of ECG abnormalities across age, sex, and ethnicity. Moreover, there have been

CONFLICTS OF INTEREST: none

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few studies to date that have described the prevalence of ECG abnormalities in an ethnically diverse population of younger and middle-aged adults. Such data can contribute to the understanding of cardiovascular epidemiology, mechanisms of CVD, and clinical risk assessment.

We sought to describe the prevalence of ECG abnormalities and their association with traditional cardiovascular risk factors among the participants (mean age 45) in the Year 20 examination of the Coronary Artery Risk Development in Young Adults (CARDIA) cohort. The CARDIA cohort is uniquely suited for the purposes of our study in that it is a large, healthy, middle-aged cohort with similar numbers of black and white men and women. Current data reflect recent advances in the primary and secondary prevention of CVD, as well as contemporary exposures to CVD risk factors (ie. the current obesity epidemic). Moreover, the CARDIA cohort has been assessed with high-quality digital 12-lead ECG's coded with Minnesota Code (MC) and Novacode (NC), as well as other routine and sophisticated ECG parameters. A description of the ECG abnormalities from this middle-aged and healthy biracial cohort will expand knowledge of the prevalence of ECG abnormalities, and their association with risk factors in a contemporary cohort.

METHODS

Study Sample

The CARDIA cohort was initiated to investigate the development of heart disease risk factors in young age. Details about the study design have been previously published (7). Briefly, 5,115 men and women aged 18 to 30 years with approximate balance in distribution of sex, race (black and white) and education were enrolled in 1985 and 1986 from 4 field sites: Birmingham, AL (University of Alabama at Birmingham); Chicago, IL (Northwestern University); Minneapolis, MN (University of Minnesota); and Oakland, CA (Kaiser Permanente Northern California). Participants were examined at baseline (Y0) and at follow-up examinations in year 2 (Y2), Y5, Y7, Y10, Y15, and Y20. With the addition of telephone contact with participants who did not attend exams, follow up has been maintained on 91% of all baseline participants. For the present analysis, we sought to describe the prevalence of ECG abnormalities and their association with traditional risk factors of the participants attending the most recent (Y20) examination. All participants signed informed consent at each examination, and all study protocols were approved by the institutional review boards at each site.

Clinical Assessment and Measurements

Demographic, anthropometric, physiologic and hematologic measurements for each participant were obtained according to previously published standardized protocols (7). Current medication data was obtained at the Y20 examination. ECGs were obtained in conjunction with the CARDIA Fitness Study, an ancillary study of the main CARDIA cohort during examination Year 20. Participants who declined or were ineligible to participate in the CARDIA Fitness Study, therefore, did not undergo routine ECG ascertainment. As a result, 2,585 of the 3,548 participants attending the Year 20 examination had codeable ECGs available for analysis.

ECG Analysis

For electrocardiography all participants had standard limb and precordial ECG leads placed, with use of the Heartsquare device (8), to determine appropriate placement of the precordial leads (V_1-V_6) . Each field center utilized the GE MAC1200, the latest data acquisition unit from GE. After acquisition the data was transmitted directly to the EPICARE central processing facility (Wake Forest University) and loaded directly into the Research

Workstation for processing. EPICARE utilizes strict quality control measures for ECG acquisition and processing to ensure precision and accuracy of data collection. The EPICARE Center personnel conducted standardized, centralized training of CARDIA ECG technicians, supervising personnel, clinic coordinators and principal investigators. Senior ECG technicians of EPICARE have been trained to monitor ECG quality and to identify any procedural errors in ECG acquisition. Visual over reading ensures accurate coding by employing a test library of 100 sets of ECGs which have previously been carefully coded and verified by the senior electrocardiographers. In addition, the Coding Supervisor performed a 1% daily coding audit.

Upon arrival to EPICARE all ECGs were read using both Minnesota Code (MC) (9) and Novacode (NC) (10) criteria, which have significantly improved standardization of ECG measurements. Processing for the present study utilized the 2001 version of the GE Marquette 12-SL program ('Marquette 12SL ECG Physician' Guide at www.gehealthcare.com). The repeatability of this program for coding is 100% unlike repeatability of visual coding by trained electrocardiographers for MC or NC, which in turn is superior to that of repeat cardiologist reading for lesser ECG abnormalities. Continuous measurement of all waveform duration and voltage for select leads as well as measures of the short-term components of heart rate variability were obtained. In secondary analyses, isolated minor ST and T wave abnormalities, (NSSTTA), minor and major ECG abnormalities were examined as defined by the Pooling Project (11), and other published studies (4) as well as analyses from ARIC (12). Participants with major ECG abnormalities were excluded from the analyses examining isolated minor NSSTTA. Finally, we examined various measures of left ventricular hypertrophy (LVH) including MC, NC, Cornell voltage (13), Cornell voltage product and left ventricular mass (LVM). Sex and race specific models of LVM indexed to weight (14, 15), which have previously been shown to have the closest correlation with echocardiographic LVM, were also employed.

Statistical Analysis

All analyses were performed using SAS version 9.1 (SAS institute, Cary, NC). Baseline characteristics were described, after stratification by race and sex, for the 2,585 participants using means and standard deviations (SD's) or percentages. Race and sex-specific ECG characteristics of the study sample were described by mean values and SD's or percentages. Differences in means or percentages were tested using chi-square tests for categorical variables and t-tests or Wilcoxon rank-sum tests for continuous variables, as appropriate. Multivariable logistic and linear regressions, as appropriate, were used to determine the cross-sectional associations of ECG abnormalities with age, race and traditional cardiovascular risk factors. P-values < 0.05 were considered statistically significant.

RESULTS

Study Sample

The study sample consisted of 2585 participants, of whom 57% were women and 44% were black, with a mean age of 45 years. Characteristics of the study cohort at Exam Y20, stratified by race and sex, are shown in Table 1. Compared to whites, black men and women had higher systolic and diastolic blood pressures, higher rates of diabetes and smoking, and were more likely to be treated for hypertension.

Continuous ECG Variables

As shown in the data supplement (Supplemental Table 1), men had significantly longer QRS duration than women and women had a longer QTc duration than men. Black men and women had significantly larger R wave amplitude in lead aVL compared with white men.

Black men and women also had higher amplitudes at the J-point in lead II, V5 and V6 as compared to their white counterparts.

Minnesota and Novacode Code Variables

The prevalence of Minnesota Code and Novacode variables are presented in Tables 2 and 3, respectively, stratified by race and sex. Compared with black men, white men had significantly higher rates of MC major and minor Q waves as well as MC left axis deviation. High MC R wave voltage was significantly more prevalent in black men and women vs. whites of the same sex. There was also a significantly higher percentage of MC isolated minor NSSTTA, minor and major ST abnormalities in blacks compared with whites, with the majority of this difference coming from T wave abnormalities.

Major and Minor ECG abnormalities

When pooling all ECG abnormalities into groups as shown in Table 4, there is a higher prevalence of both major and minor ECG abnormalities among black men and women compared with whites of the same sex. The majority of these abnormalities come from the large percentage of high voltage R waves and T wave abnormalities among black participants.

Left Ventricular Hypertrophy

Figure 1 displays the proportion of participants with conventional measures of LVH and left ventricular mass index (LVMI) stratified by age and sex. Black men and women had higher rates of LVH compared with their white counterparts when using conventional dichotomous clinical criteria including Cornell voltage, Cornell product and Minnesota Code, but they exhibited similar degrees of LVMI when examined with race and sex specific ECG models for LVMI.

Cross-Sectional Association with CV Risk Factors

The cross-sectional association of selected ECG abnormalities and traditional risk factors is shown in Table 5. Minor and major Q waves were associated with white race and smoking status. Minor STTA were associated with black race (OR 2.21, 1.51–3.22) independent of traditional risk factors including blood pressure. They were also associated with systolic blood pressure (SBP), body mass index and treatment for hypertension. LVH by Minnesota code and Cornell Voltage were associated with black race, SBP and treatment for hypertension while LVMI was associated with white race, male sex, SBP, lower DBP, total cholesterol, HDL, smoking status, and body mass index.

Participants without ECGs

Only 2,585 (73%) of the 3,548 Year 20 participants had resting ECGs available for inclusion. In addition, 1,566 participants did not attend the Year 20 examination. This degree of attrition may have biased the internal validity of the sample by selecting for participants that are more likely to pursue follow up. To further examine this possibility we compared the baseline characteristics of participants who did not attend the Year 20 examination with those who attended and did not receive an ECG, and those who attended and did receive an ECG. The participants who did not attend the Year 20 examination were more likely to be male (non-attendees 51%, attendee/no ECG 43% and attendee with ECG 43%, respectively), black (63%, 52% and 45%) and smokers (40%, 30% and 25%). Other cardiovascular risk factors including age, systolic and diastolic blood pressure, body mass index, total cholesterol, LDL and HDL-cholesterol, were similar across the three groups.

DISCUSSION

Principal Findings

The prevalence of ECG abnormalities among blacks in this younger and middle-aged biracial cohort was markedly higher than whites. This difference was primarily due to higher QRS voltage and more frequent ST and T wave abnormalities. Blacks had a significantly higher prevalence of ECG-LVH compared with whites when using dichotomous cut points, but this difference was attenuated to non-significance when using race and sex-specific LVM models. Among white men, there was a significantly higher absolute prevalence of MC major and minor Q wave abnormalities compared with black men. These racial differences were observed after multivariate adjustment for traditional risk factors. Whereas it is unclear whether these racial differences or simply imprecise ECG definitions, these abnormalities are known to be associated with incident CVD and CHD events, independent of race, sex and traditional CV risk factors (1–6).

Current Study in Context

There have been few large studies to date examining the prevalence of ECG abnormalities among middle-aged biracial cohorts. Previous analyses of the Evans County cohort (16) and the Charleston Heart Study (6) have made important contributions by describing racial differences in ECG abnormalities among healthy, middle-aged, and biracial populations, but these studies were conducted several decades prior to present day and were limited by small sample sizes. More recent analysis of healthy men and women from the Atherosclerosis Risk in Communities cohort (12) has been conducted with a larger sample size but was limited by a wider range of ages among the cohort (45–65 years) and a disproportionately white population (2,193 white vs. 503 black participants). These studies all demonstrated a significantly higher prevalence of ECG abnormalities among African Americans, particularly higher R/S amplitude and ST-T wave abnormalities. The present study benefits from a substantially larger sample size, examination of a large number of ECG variables (including both Minnesota and Novacode measures), and automated interpretation of ECG variables with physician over-reading. Moreover, the prevalence estimates in CARDIA reflect a more contemporary birth cohort.

Left Ventricular Hypertrophy

It has been well documented from previous cohorts that blacks have higher R/S amplitudes and a 2- to 6- fold higher prevalence of ECG-LVH as compared with whites (17,18). The validity of dichotomous voltage criteria for the diagnosis of LVH, however, has been shown to correlate poorly with echocardiographic data across race and sex, with particularly poor sensitivity among black men and women (19,20). Moreover, while conventional dichotomous voltage criteria for ECG-LVH have been associated with incident CVD events and mortality (21), the majority of these analyses have been conducted in all white cohorts and have not been replicated in ARIC (22) and the Charleston Heart Study (6). In order to address the shortcomings of ECG-LVH, Rautaharju et al. (14, 15) propose using race and sex specific models for LVM estimation with adjustment for body size difference as an alternative to dichotomous cut-points for the diagnosis of LVH. These methods have been extensively validated in numerous cohorts and have demonstrated both greater sensitivity for echocardiographic LVH, as well as stronger association with incident CVD mortality. Rautaharju (23) has also shown that electrocardiographic estimate of LV mass index can identify a substantially larger fraction of persons at increased risk for cardiovascular mortality than conventional electrocardiographic criteria for LV hypertrophy. In addition, analyses by Kannel (21) and Lanti (24) have shown that cardiovascular events and sudden cardiac death occurred incrementally in relation to left ventricular mass. These data suggest

that conventional dichotomous ECG-LVH criteria may be inappropriate for prevalence reporting and, as shown in CARDIA, vary significantly across race independent of conventional CV risk factors. Continuous measures of LVMI such as the model reported in our data may be more appropriate for prevalence reporting and risk prediction in the future.

ST and T wave abnormalities

Both major and minor ST and T wave abnormalities are well-established risk markers for incident coronary heart disease events (1-6). The prevalence of ST and T wave abnormalities have been extensively studied in white populations, but to date there have been no published longitudinal data examining the prevalence of ST and T wave abnormalities in young and middle-aged blacks. In ARIC (12), the prevalence of combined major ST segment and T wave (MC 4-1, 4-2, 5-1, 5-2) abnormalities among black men and women was 5.3% and 3.2%, respectively. Among whites the prevalence was 0.5% for men and 1.5% for women. Even though the CARDIA cohort is on average younger than ARIC, the prevalence of major ST and T wave abnormalities was higher for both black men and women (7.5% and 5.6%, respectively) as well as white men and women (2.1% and 1.6%, respectively). The prevalence of minor ST abnormalities was less than 1% in all groups within ARIC, but in CARDIA the prevalence was 5.1% and 2.9% among black men and women, respectively and 1.7% and 1.2% among white men and women, respectively. Both minor and major STTA were associated with black race independent of CV risk factors. In addition to black race, minor and major STTA were associated with both SBP and treatment for hypertension, which would suggest the hypothesis that the prognostic utility of these ECG abnormalities might be mediated through hypertension-related subclinical CVD. Further studies are warranted to investigate whether there are mechanisms underlying the racial differences in repolarization abnormalities.

Q waves

Q waves are significant risk markers for both incident and latent CVD (25). Previous analyses of both all white and biracial middle-aged cohorts have estimated that the prevalence of combined major (MC 1-1, 1-2) and minor (MC 1-3) Q waves was < 3–4% (25). In our analysis of CARDIA the prevalence of major and minor Q waves was significantly higher, with 9.0% and 5.3% of white men and women, respectively, and 4.9% and 6.6% of black men and women, respectively, demonstrating MC major or minor Q waves. It is unclear whether this observed increased prevalence of Q waves represents true occult infarction or inaccurate coding. Another possible explanation is that the CARDIA ECG tracings were read using the EPICARE electronic algorithm, rather than being visually coded (as in previous studies), which may have led to greater precision in calculating the duration and depth of Q waves. However, in light of the significant prognostic implications of both major and minor Q waves, with estimated hazards ratios for incident CHD and CVD ranging from 2 to 19 in other epidemiologic cohorts (25), the high prevalence of Q waves in the CARDIA cohort warrants further longitudinal assessment.

Limitations

Despite representing a large, middle-aged, and biracial cohort with high quality automated ECGs, only 2,585 (73%) of the 3,549 Year 20 participants underwent ECGs available for inclusion. In light of the already high prevalence of major and minor ECG findings among our sample, inclusion of the participants who were ineligible or declined ECG testing for Year 20 would likely have resulted in an even higher number of abnormal ECG findings and presumably a higher prevalence. Direct prevalence comparisons to previous studies (ARIC etc.) are somewhat limited by the varying methodologies used for coding and quality control issues. For example, while ECG's in both ARIC and CARDIA were recorded and transmitted to the EPICARE center in the same fashion, they were coded using two different

computing programs. Despite these differences, CARDIA benefitted from a more updated version of coding software (the GE Marquette 12-SL program) and the repeatability of the current coding programs at EPICARE is 100% unlike visual duplicate coding that has a repeatability of about 90% for Q waves and 80+ % for STT waves.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Work on this manuscript was supported (or partially supported) by contracts: University of Alabama at Birmingham, Coordinating Center, N01-HC-95095; University of Alabama at Birmingham, Field Center, N01-HC-48047; University of Minnesota, Field Center and Diet Reading Center (Year 20 Exam), N01-HC-48048; Northwestern University, Field Center, N01-HC-48049; and Kaiser Foundation Research Institute, N01-HC-48050; from the National Heart, Lung and Blood Institute.

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Figure 1.

Prevalence of Left Ventricular Hypertrophy (Minnesota Code* or Minnesota Code 3-1 [Panel A], Cornell Voltage† or Cornell voltage product \ddagger [Panel B]) and Left Ventricular Mass Index§ [Panel C] by race and sex. (n=2585) \parallel

* Minnesota Code voltage criteria: 3.1 plus repolarization abnormalities: 4.1 or 4.2 or 4.3 or 5.1 or 5.2 or 5.3

Women

[†] Cornell voltage (CV) > 2200μ V for women; > 2800μ V for men

‡ Cornell voltage product > 244 microvolt seconds = CV*QRS duration

Men

§ A method developed by Rautaharju et al...

White Women	ECG LVM (g) = $0.02 \times CV + (1.12 \times BW^*) + 36.2$
Black Women	ECG LVM (g) = $0.023 \times CV + (0.87 \times BW) + 37.6$
White Men	ECG LVM (g) = $0.026 \times CV + (1.25 \times BW) + 34.4$
Black Men	ECG LVM (g) = $0.024 \times CV + (1.18 \times BW) + 34.8$

* BW = weight in Kg and CV = Cornell Voltage

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Characteristics of CARDIA Participants by Race and Sex at Year 20

	White Men (n=667)	Black Men (n=455)	White Women (n=768)	Black Women (n=695)
Age, years *	45.7 ± 3.3	44.6 ± 3.5	45.8 ± 3.3	44.6 ± 3.8
Systolic BP, mm Hg	117 ± 12	123 ± 14	109 ± 13	119 ± 17
Diastolic BP, mm Hg	72 ± 10	76 ± 11	68 ± 10	76 ± 12
Body mass index, kg/m ²	29 ± 6	29 ± 6	27 ± 6	32 ± 8
Weight, lbs.	199 ± 37	204 ± 47	162 ± 7	191 ± 46
Height, cm	178 ± 8	178 ± 7	165 ± 7	164 ± 7
Total cholesterol, mg/dL	188 ± 37	184 ± 35	187 ± 32	185 ± 34
HDL-cholesterol, mg/dL	46 ± 13	50 ± 16	62 ± 17	58 ± 16
LDL-cholesterol, mg/dL	115 ± 33	112 ± 34	106 ± 29	110 ± 31
Triglycerides, mg/dL	141 ± 102	110 ± 72	97 ± 61	88 ± 50
Diabetic [†]	5.9 %	9.7%	7.2%	13.5%
Current Smoker	13.8%	27.5%	12.9%	19.9%
Treated for Hypertension	12.6%	17.1%	8.1%	28.5%
Treated for Hyperlipidemia ^{\ddagger}	15.2%	7.9%	5%	7.5%
Creatinine (mg/dl)	0.98 ± 0.14	1.11 ± 0.71	0.78 ± 0.14	0.82 ± 0.33

Abbreviations:

BP = Blood Pressure, HDL = High density lipoprotein, LDL = Low density lipoprotein

* Means \pm Standard deviation unless otherwise indicated

 $^{\not\!\!\!\!\!\!\!\!\!\!}$ Defined as self-reported "yes" or fasting glucose >126 mg/dL

 ‡ Any lipid lowering medication

Minnesota Code by Race and Sex in the CARDIA Cohort at Year 20 (%)

ECG Variable	М	en	Wo	men
	White N=667 % (cases)	Black N=455 % (cases)	White N=768 % (cases)	Black N=695 % (cases)
Q and QS patterns (MC 1)				
• Major and Minor Q wave (1-1, 1-2, 1-3, all leads) ^{\pm}	9 (60)	4.9 (22)	5.3 (40)	6.6 (45)
• Major Q wave (1-1, 1-2, except 1-2-6, 1-2-8)	3.5 (23)	1.8 (8)	3.0 (23)	2.3 (16)
QRS Axis Deviation (MC 2)				
• Left Axis Deviation $(2-1)^{\pm}$	2.3 (15)	1.4 (6)	0.7 (5)	1.5 (10)
• Right Axis Deviation $(2-2, 2-3)^{\pm}$	1.1 (7)	0.9 (4)	1.4 (10)	0.6 (4)
High Amplitude R Waves (MC 3)				
• High R wave voltage $(3-1, 3-3)^{\pounds}$	6.2 (41)	23.3 (103)	1.5 (11)	11 (75)
ST Junction (J) and Segment Depression (MC 4)				
• Isolated Minor ST Junction Depression $(4-3, 4-4)^{\pm}$	0.9 (6)	2.4 (11)	0.7 (5)	0.9 (6)
• Any Minor ST Junction Depression $(4-3, 4-4)^{\pounds}$	1.7 (11)	5.1 (23)	1.2 (9)	2.9 (20)
• Any Major ST Junction Depression (4-1, 4-2)	1.1 (7)	1.5 (7)	0.4 (3)	1.2 (8)
T-wave Items (MC 5)				
• <i>Isolated</i> Minor T wave abnormalities $(5-3, 5-4)^{\pounds}$	1.4 (9)	8.1 (37)	3.5 (27)	8.5 (59)
• Any Minor T wave abnormalities $(5-3, 5-4)^{\pounds}$	2.1 (14)	13.0 (59)	4.0 (31)	11.0 (76)
• Any Major T wave abnormalities $(5-1, 5-2)^{\pounds}$	1.7 (11)	6.6 (30)	1.3 (10)	5.0 (35)
Combined ST and T wave Abnormalities				
• Isolated Minor STTA (MC 4-3, 4-4, 5-3, 5-4) $^{\pounds}$	2.1 (14)	9.9 (45)	3.8 (29)	9.1 (63)
• Any Minor STTA (MC 4-3, 4-4, 5-3, 5-4) ${}^{\pounds}$	3.5 (23)	15.2 (69)	4.7 (36)	12.7 (88)
• Any Major STTA (MC 4-1, 4-2, 5-1, 5-2) \pounds	2.1 (14)	7.5 (34)	1.6 (12)	5.6 (39)
A-V Conduction Defect (MC 6)				
• First Degree AV Block (6-3) €	1.2 (8)	2.6 (12)	0.1 (1)	1.9 (13)
• Wolff-Parkinson-White Pattern (WPW) (6-4)	0	0	0	0
• Short P-R interval $(6-5)^{\pounds}$	0.9 (6)	0.9 (4)	4.9 (38)	1.6 (11)
Ventricular Conduction Defect (MC 7)				
• Left bundle branch block (LBBB) (7-1-1)	0	0	0	0
• Right bundle branch block (RBBB) $(7-2)^{\pounds}$	0.3 (2)	0.2 (1)	0.4 (3)	0.9 (6)
• Incomplete RBBB $(7-3)^{\pounds}$	3.7(24)	1.1 (5)	2 (15)	0.4 (3)
• Intraventricular block $(7-4)^{\pounds}$	0.8 (5)	0.5 (2)	0.2 (2)	0
• Incomplete LBBB $(7-6)^{\pounds}$	24.3 (159)	17.4 (77)	6.5 (49)	4.2 (29)
• Left anterior hemiblock (LAH) $(7-7)^{\pounds}$	0.3 (2)	0	0	0.1 (1)
Arrhythmias (MC 8)				
• Frequent atrial or junctional premature beats (8-1-1)	1.6 (11)	3.3 (15)	1.8 (14)	0.6 (4)

ECG Variable	М	en	Wo	men
	White N=667 % (cases)	Black N=455 % (cases)	White N=768 % (cases)	Black N=695 % (cases)
• Frequent ventricular premature beats (8-1-2)	0.9 (6)	1.5 (7)	0.8 (6)	1 (7)
Atrial fibrillation / flutter	0.1 (1)	0.2 (1)	0	0.1 (1)
• Supraventricular rhythm persistent (8-4-1)	0.3 (2)	0	0.5 (4)	0.1 (1)
• Sinus tachycardia (8-7)	0.4 (3)	0	0	0.1 (1)
• Sinus bradycardia $(8-8)^{\pm}$	5.1 (34)	5.9 (27)	4.0 (31)	2.2 (15)
Minnesota Code 9-2: ST Elevation				
• ST elevation: (9-2, all leads) $^{\pounds}$	2.7 (18)	12.2 (54)	0.7 (5)	2.2 (15)
• ST elevation (leads I, aVL, V6) $^{\pounds}$	0.3 (2)	2.3 (10)	0	0.9 (6)
• ST elevation: (leads II, III, aVF) [£]	1.2 (8)	2.7 (12)	0.3 (2)	0.4 (3)
• ST elevation (Leads V1, V2, V3, V4, AND V5) $^{\pounds}$	1.8 (12)	10.1 (45)	0.4 (3)	1 (7)
Miscellaneous Items				
High T wave amplitude $(9-5)^{\pounds}$	2.1 (14)	2.9 (13)	0	0.1 (1)
Minnesota Code MI	3.9 (26)	1.3 (6)	2 (15)	1.4 (10)

 $\overset{\pm}{p}$ < 0.05 for comparison between all four groups

 \mathcal{E} p < 0.01 for comparison between all four groups

f p < 0.0001 for comparison between all four group

Novacode by Race and Sex in the CARDIA cohort at Year 20.

ECG Variable	M	EN	WO	MEN
	White N=667 % (cases)	Black N=455 % (cases)	White N=768 % (cases)	Black N=695 % (cases)
Arrhythmias (NC 1)				
• Wandering atrial pacemaker (1.1)	0.1(1)	0	0.1 (1)	0.3 (2)
• Junctional Rhythm (1.2.1)	0	0	0.1 (1)	0
• Ectopic atrial rhythm (1.3.1)	0.3 (2)	0	0.4 (3)	0.1 (1)
Atrial Fibrillation (1.5.3)	0.1 (1)	0.2 (1)	0	0.1 (1)
Atrioventricular Conduction abnormalities (NC 2)				
• First Degree AV Block $(2.1)^{\pounds}$	1.2 (8)	3.3 (12)	0.1 (1)	1.9 (13)
Prolonged Ventricular Excitation (NC 3)				
• Left Bundle Branch Block $(3.1)^{\pounds}$	0	0	0.1 (1)	0
• Right Bundle Branch Block $(3.2)^{\pounds}$	0.3 (2)	0	0.4 (3)	0.9 (6)
• Indeterminate Ventricular Conduction Delay $(3.3)^{\pounds}$	0.8 (5)	0.7 (3)	0.1 (1)	0
• Borderline delay of right ventricular excitation $(3.4.1)^{\pounds}$	0.5 (3)	0	0.1 (1)	0
• Borderline delay of left ventricular excitation $(3.4.2)^{\pounds}$	1.2 (8)	0.4 (2)	0.1 (1)	0
Prolonged Ventricular Repolarization (NC 4)				
Marginal prolongation of ventricular repolarization (4.1.1)	0.5 (3)	0.9 (4)	0.9 (7)	1.2 (8)
• Definite prolongation of ventricular repolarization (4.1.2)	0.3 (2)	0.2 (1)	0	0.1 (1)
Prevalent Myocardial Infarction/Ischemia (NC 5)				
• Q wave MI with major Q waves $(5.1)^{\pounds}$	0.3 (2)	0.1 (1)	0.1 (1)	0.1 (1)
• Q wave MI with moderate Q waves and with ST-T wave abnormalities (5.2) ${}^{\pounds}$	0	0	0.4 (3)	0
- Possible Q wave MI with moderate Q waves and without ST-T abnormalities $(5.3)^{\pounds}$	0.9 (6)	0.1 (1)	0.5 (4)	0.7 (5)
• Possible Q wave MI with minor Q waves and ST-T abnormalities (5.4) ${}^{\pounds}$	0.5 (3)	0.9 (4)	0.1 (1)	0.4 (3)
• ST abnormalities without Q waves $(5.5)^{\pounds}$	1.4 (9)	2.9 (13)	0.4 (3)	1.8 (12)
• T wave abnormalities without Q waves $(5.6)^{\pounds}$	0.3 (2)	4.8 (21)	0.4 (3)	3 (21)
• Minor Q waves without ST-T wave abnormalities $(5.7)^{\pounds}$	10.1 (67)	5 (22)	3.7 (28)	3 (21)
• Minor ST-T abnormalities $(5.8)^{\pounds}$	9.4 (62)	17.6 (78)	10.8 (81)	16.2 (110)
Left Ventricular Hypertrophy (NC 6)				
• LVH with ST-T abnormalities $(6.1.1)^{\mathcal{L}}$	0.6 (4)	1.6 (7)	0	0.4 (3)
Left Atrial Enlargement (NC 7)				
• Left atrial enlargement $(7.1)^{\pounds}$	12.9 (86)	16.9 (75)	9.1 (69)	16.2 (111)
Right Ventricular Hypertrophy (NC 8)				
• Right Ventricular hypertrophy (8.1)	0.5 (3)	0.4 (2)	0.3 (2)	0.1 (1)
Right Atrial Enlargement (NC 9)				
• Right Atrial Enlargement (9.1)	0.1 (1)	0.2 (1)	0.1 (1)	0.4 (3)
Fascicular Blocks (NC 10)				

ECG Variable	M	EN	WO	MEN
	White N=667 % (cases)	Black N=455 % (cases)	White N=768 % (cases)	Black N=695 % (cases)
Left anterior fascicular block (LAFB) (10.1)	0	0	0	0.1 (1)
• Left posterior fascicular block (LPFB) (10.2)	0.3 (2)	0	0.4 (5)	0.4 (3)

 $\overset{\pounds}{p} < 0.05$ for comparison between all four groups

 ϵ p < 0.01 for comparison between all four groups

 $\mathcal{L}_{p < 0.0001}$ for comparison between all four groups

Major and Minor ECG abnormalities by Race and Sex in the CARDIA cohort at Year 20

		M	EN	WO	MEN
	Minnesota Code	White N=667 % (cases)	Black N=455 % (cases)	White N=768 % (cases)	Black N=695 % (cases)
Major ECG abnormalities \pounds_{11}		6.1 (41)	13.2 (60)	5.5 (42)	8.3 (58)
Pathologic Q waves	1-1, 1-2	3.4 (23)	1.8 (8)	3.0 (23)	2.3 (16)
Major / ischemic ST-depression	4-1, 4-2	1.1 (7)	1.5 (7)	0.4 (3)	1.2 (8)
Major / ischemic T-wave abnormality \pounds	5-1, 5-2	1.7 (11)	6.6 (30)	1.3 (10)	5.0 (35)
Atrioventricular Block	6-1, 6-2	0	0	0	0
Wolff-Parkinson-White Pattern	6-4	0	0	0	0
Complete Left bundle branch block	7-1	0	0	0	0
Complete Right bundle branch $block^{\pounds}$	7-2	0.3 (2)	0.2 (1)	0.4 (3)	0.9 (6)
Intraventricular Block^{\pounds}	7-4	0.7 (5)	0.4 (2)	0.3 (2)	0
Frequent ectopic beats	8-1	2.8 (19)	4.8 (22)	2.7 (21)	2 (14)
Ventricular Rhythm	8-2	0	0	0	0
Atrial Fibrillation/Flutter	8-3	0.1 (1)	0.2 (1)	0	0.1 (1)
Supraventricular Tachycardia	8-4	0.3 (2)	0	0.5 (4)	0.1 (1)
		N=628	N=395	N=730	N=638
Minor ECG abnormalities \pounds^{11}		9.2 (58)	24 (95)	7.9 (58)	17.1 (109)
Borderline Q wave ^{\pm}	1-3	5.1 (34)	3.1 (14)	2.2 (17)	3.9 (27)
Axis Deviation ^{\pm}	2-1, 2-2	2.4 (15)	1.8 (7)	0.8 (6)	1.6 (10)
Non-specific ST depression \pounds	4-3, 4-4	1.7 (11)	5.1 (23)	1.2 (9)	2.9 (20)
Non-specific T-wave abnormality \pounds	5-3, 5-4	2.1 (14)	13.0 (59)	4.0 (31)	11.0 (76)
Atrioventricular Block $ e $	6-3	1.2 (8)	2.6 (12)	0.1 (1)	1.9 (13)
Low or high voltage QRS Complex ${}^{\pounds}$	9-1, 3-1, 3-2	6.7 (42)	26 (104)	1.5 (11)	11.8 (75)
Major ECG Abnormalities (ARIC criteria) $\pounds{12}$	See reference	6.2 (41)	8.8 (40)	3.3 (25)	7.5 (52)
Minor ECG Abnormalities (ARIC criteria)£12	See reference	42.3 (282)	46.8 (213)	24.5 (188)	30.5 (212)

 $^{\pm}$ p < 0.05 for comparison between all four groups

€ p < 0.01 for comparison between all four groups

f p < 0.0001 for comparison between all four groups

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Multivariate-Adjusted Cross-Sectional Association of Selected Electrocardiographic Abnormalities with Traditional Cardiovascular Risk Factors at CARDIA Year 20

	Major and Mi Q waves (Mt	nor C)	Minor STTA (MC)	Major STTA (1	MC)	LVH (Minnesota Co	de)	LVH (Cornell Volta	age)	(IIWAI) LVH	
	N = 138		N = 169		N = 79		N = 24		N = 55			
	Odds Ratio (95% CI)	p- value	Odds Ratio (95% CI)	p- value	Odds Ratio (95% CI)	p- value	Odds Ratio (95% CI)	p- value	Odds Ratio (95% CI)	p- value	Parameter Estimate (95 % CI)	p-value
Black Race	$0.54\ (0.36, 0.82)$	< 0.01	2.21 (1.51, 3.22)	< 0.01	2.51 (1.47, 4.29)	< 0.01	3.70 (1.21, 11.31)	< 0.02	2.23 (1.04, 4.77)	< 0.04	-3.69 (-4.27, -3.11)	< 0.01
Male Sex	1.06(0.70, 1.59)	NS	0.84 (0.56, 1.26)	NS	1.63 (0.97, 2.75)	NS	3.92 (1.30, 11.79)	< 0.02	0.57 (0.27, 1.21)	NS	2.93 (2.29, 3.57)	< 0.01
Systolic BP, Per 20 mm Hg	1.06(0.70, 1.60)	NS	1.43 (1.03,1.98)	< 0.03	1.63 (1.07, 2.47)	< 0.02	2.73 (1.46, 5.12)	< 0.01	1.98 (1.24, 3.17)	< 0.01	1.35 (0.72, 1.97)	< 0.01
Diastolic BP, Per 10 mm Hg	1.03 (0.80, 1.36)	NS	1.14 (0.89, 1.44)	NS	0.84 (0.61, 1.16)	NS	0.57(0.32,1.01)	NS	1.01 (0.69, 1.47)	NS	$-0.68 \ (-1.10, \ -0.25)$	< 0.01
Total cholesterol, Per 40 mg/dL	1.1 (0.90, 1.34)	NS	0.99 (0.81, 1.20)	NS	1.04 (0.79, 1.36)	NS	0.75(0.42,1.32)	NS	$0.82\ (0.56,1.18)$	NS	$-0.34 \ (-0.65, -0.03)$	< 0.03
HDL-cholesterol, Per 10 mg/dL	1.05 (0.93, 1.20)	NS	1.02 (0.91, 1.14)	NS	1.14 (1.0, 1.31)	NS	$1.09\ (0.80,\ 1.47)$	NS	1.02 (0.83, 1.24)	NS	$0.57 \ (0.39, \ 0.74)$	< 0.01
Diabetic $\dot{ au}$	1.11 (0.59,2.10)	NS	1.14 (0.67, 1.93)	NS	1.72 (0.88, 3.38)	NS	0.60 (0.12, 3.07)	NS	1.67 (0.75, 3.70)	NS	$-0.98\ (-1.97,\ 0.01)$	< 0.05
Current Smoker	1.64 (1.07, 2.5)	< 0.02	0.81 (0.52, 1.27)	NS	1.15 (0.65, 2.05)	NS	0.85 (0.25, 2.82)	NS	1.47 (0.72, 2.99)	NS	0.99 (0.29, 1.70)	< 0.01
Body mass index, Per 4 kg/m^2	1.05 (0.95, 1.17)	NS	1.11 (1.01, 1.22)	< 0.03	1.12 (1.0, 1.26)	NS	1.05 (0.82, 1.36)	NS	1.06(0.89,1.27)	NS	4.85 (4.67, 5.03)	< 0.01
Treated for Hypertension	1.40 (0.85, 2.30)	NS	1.77 (1.18, 2.64)	< 0.01	1.88 (1.07, 3.31)	< 0.03	3.25 (1.15, 9.15)	< 0.03	2.23 (1.11, 4.49)	< 0.03	-0.60 (1.39, 0.19)	NS
Treated for Hyperlipidemia \ddagger	1.25 (0.69, 2.28)	NS	0.75 (0.41, 1.38)	NS	0.74 (0.32, 1.69)	NS	N/a	NS	1.97 (0.85, 4.58)	NS	1.10 (0.10, 2.09)	<0.03

MC = Minnesota Code, STTA = ST and T wave abnormalities, LVH = Left ventricular Hypertrophy, LVMI – left ventricular mass index, CI = confidence interval, BP = Blood Pressure, HDL = High density lipoprotein, NS = Not significant (p > 0.05)

 $^{\prime}$ Defined as self-reported "yes" or fasting glucose >126 mg/dL

J Electrocardiol. Author manuscript; available in PMC 2013 February 11.

 \sharp Any lipid lowering medication