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among Stroke Patients in Sub-Saharan Africa**



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Inter-Racial Effect on Electrocardiographic Abnormalities among Stroke Patients in Sub-Saharan Africa

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Abstract

Purpose: The study aimed to determine the prevalence of electrocardiographic abnormalities in black stroke patients and to find out if the black race predisposes to increased electrocardiographic abnormalities as well as specific electrocardiographic abnormalities among the study population.

Methodology: This was a cross-sectional analytical study carried out at the University of Benin Teaching Hospital Benin. The electrocardiographic abnormalities of one hundred and twenty (120) admitted black stroke patients in this study were compared with Goldstein study on

Caucasians in the United States of America. The data were analyzed using the IBM SPSS statistics version 22. A $p < 0.05$ was considered significant.

Results: The study demonstrated the prevalence of electrocardiographic abnormalities among black stroke patients to be 76.6% with the associated presence of left axis deviation (52.17% vs 15.22%, $p < 0.001$), left atrial enlargement (17.39% vs 5.80%, $p = 0.005$) and ST segment depression (43.50% vs 21.74%, $p = 0.001$). On the contrary, stroke patients of the Caucasian race were linked to sinus tachycardia (30.44 % vs 13.04%, $p = 0.002$), prolonged QT (49.28% vs 8.70%, $p < 0.001$) and premature ventricular complex (13.04% vs 4.35%, $p = 0.049$) with a concomitant absence of bi-atrial enlargement, low limb lead voltage and non-specific intraventricular block. They had a prevalence of 92%.

Unique Contribution to Theory, Policy and Practice: The prevalence of electrocardiographic abnormalities is lower among the black stroke individuals compared to the Caucasians even though the former tend to present with features of structural abnormalities compared to the latter who had more electrical abnormalities. Therefore, in resource-poor settings where ECG cannot readily be carried out, the race of the patient may guide the clinician in suspecting the probable cardiac changes in stroke patients.

Keywords: *Inter-Racial, Black race, Electrocardiogram, Stroke, Sub-Saharan Africa*

Introduction

Stroke, according to the World Health Organization (WHO) is an acute neurological deficit of cerebrovascular origin that persists beyond twenty-four hours or is interrupted by death within twenty-four hours. It has been projected that stroke could soon be the most common cause of death worldwide as it is currently the second leading cause of death in the world, ranking after heart disease.¹⁻⁹

Globally there is an increasing trend in the burden of non-communicable diseases especially cardiovascular and cerebrovascular diseases, particularly in low and middle-income countries of Africa including Nigeria. This transition imposes more constraints in dealing with the double burden of communicable and non-communicable diseases in a poor economy characterized by inadequate health systems. This trend has been attributed to the increasing use of tobacco, westernized lifestyle and urbanization with reduced physical activity, increased caloric consumption and psychosocial stress. These act synergistically to cause increased cardiovascular and cerebrovascular risk via weight gain, hypertension, dyslipidemia, dysglycemia and hyperuricemia.

About eight hundred thousand people in the United States have strokes each year, and one hundred and thirty thousand of them die each year. One American dies from stroke every four minutes on average. The cost of stroke care in the United States is estimated to be \$36.5 billion

each year.^{10,11} The incidence of stroke increases exponentially from thirty years of age, and the etiology varies with age. Advanced age is one of the most significant stroke risk factors. Ninety-five percent of strokes occur in people aged forty-five and above, and two-thirds occur in those over sixty-five.^{3,12}

Disability affects seventy-five percent of stroke survivors enough to decrease their employability and stroke can affect patients physically, mentally, emotionally, or its combination. The result of stroke varies widely depending on size and location of the lesion. Thirty to fifty percent of stroke survivors suffer post-stroke depression, which is characterized by lethargy, irritability, sleep disturbance, lowered self-esteem and withdrawal while up to ten percent of all stroke patients develop seizures most commonly in the weeks after the stroke event and the severity of the stroke increases the likelihood of a seizure.^{8, 13-15}

Stroke can be classified into ischaemic Stroke and hemorrhagic stroke. Ischaemic stroke occurs as a result of an obstruction within a blood vessel supplying blood to the brain. It accounts for about eighty-seven percent of all stroke cases. The underlying condition for this type of obstruction is the development of fatty deposits lining the vessel wall. This condition is called atherosclerosis. Cerebral thrombosis refers to a thrombus that develops at the clogged part of the vessel and cerebral embolism occurs as a result of a blood clot in the cerebrovascular system from another location in the circulatory system, usually, the heart and large arteries of the upper chest and neck, and these tend to be associated with atrial fibrillation and other heart diseases. Systemic hypoperfusion is a decrease in blood supply while venous thrombosis leads to stroke due to locally increased venous pressures which exceed the pressure generated by the arteries. These infarcts are more likely to undergo haemorrhagic transformation than other types of ischaemic stroke. Cryptogenic stroke, which is a stroke of unknown origin, constitutes thirty to forty percent of all ischaemic stroke.^{9, 16-19} Stroke can also be classified into Total Anterior Circulation Infarct (TACI), Partial Anterior Circulation Infarct (PACI), Lacunar infarct (LAC) and Posterior Circulation Infarct (POCI). These four entities predict the extent of the stroke, the area of the brain affected, the underlying cause and the prognosis.^{20,21} Hemorrhagic stroke arises from bleeding within the brain parenchyma or intraventricular spaces. They constitute about fifteen percent of strokes. They result in tissue injury by causing compression of tissue from expanding hematomas or hematomas. This can distort and injure tissues. In addition, the pressure may lead to a loss of blood supply to the affected tissues with resulting infarction, and the blood released by brain hemorrhage appears to have direct toxic effects on brain tissue and vasculature. Inflammation also contributes to secondary brain injury after a hemorrhage.^{19, 22, 23}

Black Americans have twice the risk of stroke compared to Caucasians. Post-stroke recurrence among black is 5-14% in one year, twenty-four percent among females and forty percent among males in five years even though stroke, recurrence and TIA can be prevented through lifestyle changes, medications, surgery or a combination of all three methods.²⁴ Blacks and Asians

hospitalised for ischaemic strokes are less likely to die than whites. However, black men between the ages of forty-five and fifty-four are three to four times more likely to die than whites. Hospitalization for stroke in blacks is associated with a longer stay, higher cost but lower thirty-day mortality and reperfusion therapy than whites. Blacks tend to have more disabilities after stroke than white. There are prominent race differences in ECG characteristics and a differential association with mortality.²⁵⁻²⁸ These interracial differences and their impact on the ECG findings have not been adequately documented in the African subregion. Therefore, this study aimed to determine the prevalence of ECG abnormalities in black stroke patients, if black stroke patients are associated with increased and specific ECG abnormalities.

Materials and Method

This cross-sectional observational study was carried out at the University of Benin Teaching Hospital (UBTH) Nigeria which offers secondary and tertiary care to patients in Edo and neighbouring states. The population of the study was stroke presenting at the hospital. A simple non-randomized sampling method was used in selecting the stroke patients recruited for this study. One hundred and twenty patients who met the inclusion criteria were recruited from the stroke unit and all patients were computerized tomography confirmed to have a bleed or an infarct. They had a detailed history and physical examination finding entered into the data acquisition sheet. ECG was performed on the stroke patients within the first twenty-four hours of presentation. Inclusion criteria include patients who are 18 years and above and have first-ever occurrence of stroke. Stroke patients that have two or more occurrences of stroke (recurrent stroke), less than eighteen (18) years of age, resolved within twenty-four (24) hours, as evidenced by resolution of presenting complaints, patients that died within 7 days of presentation, HIV positive patient, patients with malignancies, immunosuppressive therapy and electrolyte abnormalities were excluded from the study.

A conventional resting 12 lead ECG was performed. Lead II was used as the rhythm strip. The recommendation of the Society for Cardiological Science & Technology (SCST) concerning the standardization of leads and specification for instruments was followed. Each Electrocardiogram was assessed for (a) Rhythm: Sinus rhythm and rhythm abnormalities (including Atrial fibrillation or Atrial flutter, premature ventricular complexes, ventricular tachyarrhythmia) was noted (b) Conduction abnormalities e.g. Atrioventricular blocks, Bundle branch blocks, bifascicular and Trifascicular blocks (c) Chamber sizes; Right atrial enlargement, left atrial enlargements, Bi atrial enlargement, Left ventricular hypertrophy, Right Ventricular Hypertrophy. (d) Others; QT Interval, QTc Interval, QRS Duration, QRS Axis.

Anthropometric measurement and data collected using the preformat were collated and analyzed using the International Business Machines Statistical Product and Service Solutions (IBMSPSS) version 22. Data were presented using tables and charts. Frequencies and percentages were used to present categorical data while continuous data were expressed as mean (Standard Deviation). Frequencies were compared using the Pearson's Chi-square test while

means were compared using the independent t-test. A $p < 0.05$ was considered significant for all statistical comparisons.

Ethical clearance was obtained from the Research and Ethics Committee of the University of Benin Teaching Hospital, Benin City, Edo State. Informed consent was obtained from patients before participation in the study. Respect for respondents and confidentiality were maintained throughout the process of extracting the data.

Result

Table 1: Sociodemographic Characteristics of Black vs Caucasian stroke patients

Sociodemographic Variables	Study Population		<i>P</i> value
	Black Race (n=120)	Caucasian Race(n=150)	
Age, yrs (Mean±SD)	60.67±7.91	66.40±8.55	0.585
MALE SEX, n(%)	56(46.67)	72(48.00)	0.827
FEMALE SEX, n(%)	64(53.33)	78(52.00)	0.827

Table 2: ECG abnormalities of the study population

ECG abnormality	Study Population, n(%)	P value
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	Black Race (n=120)	Caucasian (n=150)	Race
ABNORMALITY	92(76.67)	138(92.00)	<0.001*
NO ABNORMALITY	28 (23.33)	12(8.00)	<0.001*

**P<0.05 indicating significant*

Table 3: Structural changes of black vs caucasian stroke patients

Structural ECG changes	Study Population, n(%)		P value
	Black Race, n=120	Caucasian, n=150	
LEFT AXIS DEVIATION	48(52.17)	21(15.22)	<0.001*
LEFT ATRIAL ENLARGEMENT	16(17.39)	8(5.80)	0.005*
BI ATRIA ENLARGEMENT	4(4.35)	0(0)	0.050
T WAVE INVERSION	4(4.35)	10(7.25)	0.368

ST DEPRESSION	40(43.50)	30(21,74)	0.001*
LEFT VENTRICULAR HYPERTROPHY	28(30.44)	39(28.26)	0.722
LEFT ANTERIOR FASCICULAR BLOCK	8(8.70)	10(7.25)	0.689
LEFT BUNDLE BRANCH BLOCK	0(0)	3(2.17)	0.406
LOW LIMB LEAD VOLTAGE	4(4.35)	0(0)	0.050
NON-SPECIFIC INTRAVENTRICULAR BLOCK	4(4.35)	0(0)	0.050

**P<0.05 indicating significant*

Table 4: Electrical changes of black vs caucasian stroke patients

Electrical changes	Study population, n(%)		p value
	Black Race, n=120	Caucasian, n=150	
SINUS TACHYCARDIA	12(13.04)	42(30.44)	0.002*
ATRIAL FIBRILLATION	12(13.04)	21(15.22)	0.645

PROLONG QT	8(8.70)	68(49.28)	<0.001*
PREMATURE ATRIAL COMPLEX	4(4.35)	10(7.25)	0.368
PREMATURE VENTRICULAR COMPLEX	4(4.35)	18(13.04)	0.049*

* $P < 0.05$ indicating significant

Sociodemographic Characteristics of black vs caucasian stroke patients

Table 1 showed that 120 black patients, and 150 caucasian patients were compared. The mean age of black and caucasian patients, and sex difference were not significantly different.

ECG abnormality of black vs caucasian stroke patients

There were no ECG abnormalities in 28 (23.33%) black stroke patients and 12 (8.00%) caucasian stroke patients. However ECG abnormalities were found in 92(76.67%) and 138(92.00%) of black and caucasian stroke patients respectively. This was found to be statistically significant, $p < 0.001$.

Structural Changes of black vs caucasian stroke patients

Table 2 showed Forty-eight (52.17%) Black cases had a Left axis deviation while 21(15.22%) Caucasian cases had a Left axis deviation. There was a significant difference of $p < 0.001$. The p-wave duration ranges from 78-134 msec for blacks and none was documented for the Goldstein study. The mean p-wave duration was 108.01 ± 12.12 msec for blacks and none for the Goldstein study. 4 (4.35%) black and 10 (11.91%) Caucasian had this ECG abnormality. There was however no statistically significant difference, $p = 0.365$. 40(43.50%), 30(35.72%) black and Caucasian respectively had these ECG abnormalities but there was no significant difference, $p = 0.001$. In addition, left atrial enlargement was present in 16 (17.38%) and 18 (5.80%) of black and Caucasians respectively; and it was found to be significant, $p = 0.005$. 4(4.35%) of black had biatrial enlargement compared to the Caucasians who had none ($p=0.05$). Left ventricular hypertrophy was present in 28(30.44%) of the black and 9.26% of Caucasians ($p=0.722$). The left anterior fascicular block was present in eight (8.70%) Black stroke patients' ECG and ten (7.25%) of caucasian. This difference was not significant $p = 0.689$. The left

bundle branch block was absent in blacks and present in 3(2.37%) of Caucasians ($p=0.406$).

Electrical changes of Black vs Caucasian stroke patients

Table 3 showed that 12 (13.04%) of Black cases and 42(30.60%) of Caucasian cases had sinus tachycardia and this was found to be statistically significant $p = 0.002$. 12(13.04%) black cases and 21 (15.22%) Caucasian cases had atrial fibrillation but there was no statistically significant difference($p = 0.645$). The QRS duration ranges from 78 to 140 msec with a mean of 101.85 ± 18.78 msec for blacks. The QRS duration range for the Goldstein study was not documented.

The mean PR interval was 144.04 ± 19.18 msec for the blacks ranging between 114 to 194msec. The PR interval range was not also documented for the Goldstein study. 8(8.70%) of black and 68(49.28%) of caucasian had a prolonged QT interval(< 0.001).

Premature atrial complex, premature ventricular complex, low limb lead voltage and nonspecific intra-ventricular block were present in 4 (4.35%) each of the black cases and 10(7.35%), 18(13.04%), 0(0%) and none of the caucasian respectively ($p > 0.05$).

Discussion

Race and ethnicity are known risk factors for stroke, being more common in people of color than amongst whites. The prevalence of electrocardiographic abnormalities in black stroke patients was 76.67% with a tendency towards structural and ischemic changes on ECG. On the contrary, Caucasians had a preponderance of electrical changes on ECG with an overall higher percentage of 92%.

The structural abnormalities among the black race included left axis deviation, left atrial enlargement and ST segment depression. This further buttresses the fact that hypertension, which is the most common risk factor for stroke in sub-Saharan Africa, is probably responsible for cardiac changes. In addition, it has been reported in several literature that blacks have a higher burden of hypertensive heart disease. The Caucasian race, on the other hand, predisposes to sinus tachycardia, prolonged QT and premature ventricular complex. These findings suggest that rhythm disorders are less common among black individuals, whereas it is the major disorder among Caucasians.

This study showed that electrical abnormalities were predominant in the whites compared to the blacks, this is consistent with findings from Prineas et al [1] which reported increased QRS duration in whites compared to the blacks in patients within the age group of 45-65 years, they also noted that bundle branch block (BBB) was common in elderly white men compared to blacks. However, it was seen that major Q waves were prevalent in elderly white men compared to blacks, but were more common in blacks within the younger age group [1]. In the same vein, elderly blacks had a lower prevalence of atrial fibrillation compared to whites, but the reverse

was the case in the younger black population, as they were more likely to have atrial fibrillation compared to whites [1].

Left ventricular hypertrophy was seen to be more common in blacks in the study done by Prineas et al [30], this finding was also consistent with that of Pinto-Filho et al who reported that black men and women had an increased prevalence of left ventricular hypertrophy and ST-T abnormalities [31]. Although in our study we found the prevalence of left ventricular hypertrophy similar when compared to that of Goldstein's study [5]. However, we observed in our study that blacks had more structural abnormalities compared to whites, with an increase in the prevalence of left axis deviation, left atrial enlargement and ST segment depression which is suggestive of a higher burden of hypertensive heart disease. Ishikawa et al reported that left ventricular hypertrophy on ECG was linked to an increased risk of ischemic stroke in the general population [32], this finding is useful in highlighting the increased risk of ischemic stroke within the black population. In contrast to our study, Prineas et al noted more prevalence of atrial premature beats in blacks compared to whites [30], as we noted more of these electrical abnormalities in whites compared to blacks.

Studies have shown that the middle-aged black population in the US had significantly more ECG abnormalities compared to whites [30,33], Strogatz et al also showed that ECG abnormalities were more common in blacks compared to whites [34]. Sutherland et al revealed that blacks had more ECG abnormalities compared to the whites after Minnesota code was performed on the men [35], Riley et al reported differences in the ECG abnormalities between the black and white populations [36]. Pinto-Filho et al noted a higher prevalence of ECG abnormalities in the black population when compared to other races, reiterated that these ECG abnormalities increased with age, and the black race was an isolated risk factor for the presence of major cardiovascular abnormalities [31].

Conclusion

The study showed that black stroke patients presented with lesser ECG abnormalities compared to Caucasian stroke patients. While the latter is more associated with arrhythmia, the former is linked with the remodelling of the heart muscles. Racial differences may therefore account for the ECG findings of patients with stroke. We recommend that amongst black patients with stroke, clinicians should have a suspicion for cardiac arrhythmias where there are changes in heart muscle especially in the resource-poor locality where ECG are not readily available.

References

1. World Health Organization. Cerebrovascular Disorder Geneva: World Health Organization.1978.24-6

2. Boutayeb A and Boutayeb S. The burden of non-communicable disease in developing countries. *Int. J. Equity Health*. 2005; 4: 2-6.
3. Ellekjaer H, Holmen J, Indredavik B, et al. Epidemiology of stroke in Innherred, Norway, 1994 to 1996: Incidence and a 30 – Day case fatality rate. *Stroke* .1997; 28: 2180 -2184.
4. Kocan M J. Cerebrovascular effects of acute stroke. *Prog Cardiovascular Nurs*.1999; 1:61—7.
5. Goldstein D S. The electrocardiogram in stroke: relationship to pathophysiological type and comparism with prior tracings. *Stroke*. 1979; 10: 253-9.
6. Familoni O.B. The pattern and prognostic features of QT intervals and dispersion in patients with acute ischaemic stroke. *J Natl Med. Assoc*. 2006; 98: 1758-62.
7. Tokgozoglu S.L, Batur M.K, Topcuoglu M.A, et al. Effects of Stroke localization on Cardiac Autonomic Balance and Sudden Death. *Stroke*. 1999; 30:1307-11.
8. World Health Organization. The World Health Report 2004. Annex Table 2: Deaths by cause, sex and mortality stratum in WHO regions, estimates 2002. Geneva. World Health Organization.2004.
9. Donnan G A, fisher M, Macleod M, et al. *Stroke*. *Lancet*. 2008; 371: 1612 -15.
10. Go A.S, Mozaffarian D, Roger V .L et al. Heart and stroke statistics 2013update report from the American Heart Association. *Circulation*. 2013; 2: 241-6.
11. WHO. The top 10 causes of death. Geneva. World Health Organization. 2017. Available from www.who.int/en/news-room/factsheets/detail/the-top-10-causes-of-death (Accessed on 16th May 2018).
12. Senelick R, Rossi C, Peter W, et al. *Living with stroke: A Guide for facilities*. Chicago. Contemporary books. 1994.10-6
13. Coffey C, Edward C. Jeffery L, et al. *Stroke* .The American psychiatric press textbook of Geriatric Neuropsychiatry . Washington DC: American Psychiatric press.2000.2 edition. 601 – 617.
14. Lisa D. Sandra E. Fuqiand G et al. Correlating lesion size and location to deficits after ischaemic stroke the influence of accounting for altered peri-necrotic tissue and incidental silent infarcts. *Behav Brain Funct*. 2010; 6: 6-10. Available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2823642>. Accessed 12th September, 2019
15. Reith J, Jorgensen H. S, Hakayama H. et al. Seizures in acute stroke: predictors and prognostic significance. *The Copenhagen stroke study*.1997; 28: 1585-9.
16. Ocarroll C.B, and Barrette K.M. Cardioembolic Stroke. *Continuum Lifelong Learning in Neurology*.2017; 23:111-132.

17. Shuaib A, Hachinski V. C, "Mechanisms and management of stroke in the elderly". CMAJ. 1991; 145: 433 - 43.
18. Stam J. Thrombosis of the cerebral veins and sinuses. The New England Journal of Medicine. 2005; 352:1791 – 8.
19. National Institute of Neurological Disorders and Stroke (NINDS). 'Stroke Hope Through Research'. National Institute of Health.1999.112-5
20. Bamford J. Classification and natural history of clinically identifiable subtypes of cerebral infarction. Lancet.1991; 337:1521 – 6.
21. Bamford J. M. 'The role of the clinical examination in the subclassification of stroke'. Cerebrovas. Dis.2000; 10:2 – 4
22. Wang J. 'Preclinical and clinical research on inflammation after intracerebral haemorrhage'. Prog. Neurobiol. 2010; 92: 463–77.
23. Adeloje D. An estimate of the prevalence of hypertension in Nigeria: a systematic review and meta-analysis. J. Hypertension. 2015; 2: 260 – 262.
24. Ezenwa C, and Gutierrez J. Secondary stroke prevention: challenges and solutions. Vasc. Health Risk Manag, 2015; 11: 437-450.
- 25 kumar N, Khera R, Pandey A et al. Racial Differences in Outcomes after Acute Ischaemic Stroke Hospitalization in the United State.J strokecerebrovasdis.2016;25:1970-77.
- 26 Howard VJ. Reasons for Underlying Racial Differences in Stroke Incidence and Mortality. Stroke.2013;44:126-8
- 27 Albright K C, Huang L, Blackburn J, et al. Racial differences in recurrent ischaemic stroke risk and recurrent stroke case fatality. Neurology. 2018;91: 1741-50
- 28 Burke J F, Freedman VA, Lisabeth L D, et al.Racial difference in disability after stroke; results from a nationwide study. Nuerology.2014;83:390-7
- 29 Santhanakrishnan R, Magnani M J, Vasan R S, et al. Racial Difference in Electrocardiographic characteristics and prognostic significance in Whites Versus Asians. J. Am Heart Association.2016;5:2956
- 30 Prineas, R. J., Le, A., Soliman, E. Z., Zhang, Z. M., Howard, V. J., Ostchega, Y., Howard, G., & Reasons for Geographic and Racial Differences in Stroke (REGARDS) Investigators (2012). United States national prevalence of electrocardiographic abnormalities in black and white middle-age (45- to 64-Year) and older (≥ 65 -Year) adults (from the Reasons for Geographic and Racial Differences in Stroke Study). The American journal of cardiology, 109(8), 1223–1228. <https://doi.org/10.1016/j.amjcard.2011.11.061>

- 31** Pinto-Filho, M. M., Brant, L. C. C., Foppa, M., Garcia-Silva, K. B., Mendes de Oliveira, R. A., de Jesus Mendes da Fonseca, M., Alvim, S., Lotufo, P. A., Mill, J. G., Barreto, S. M., Macfarlane, P. W., & Ribeiro, A. L. P. (2017). Major Electrocardiographic Abnormalities According to the Minnesota Coding System Among Brazilian Adults (from the ELSA-Brasil Cohort Study). *The American journal of cardiology*, 119(12), 2081–2087. <https://doi.org/10.1016/j.amjcard.2017.03.043>
- 32** Ishikawa, J., Ishikawa, S., Kabutoya, T., Gotoh, T., Kayaba, K., Schwartz, J. E., Pickering, T. G., Shimada, K., Kario, K., & Jichi Medical School Cohort Study Investigators Group (2009). Cornell product left ventricular hypertrophy in electrocardiogram and the risk of stroke in a general population. *Hypertension (Dallas, Tex. : 1979)*, 53(1), 28–34. <https://doi.org/10.1161/HYPERTENSIONAHA.108.118026>
- 33** Walsh, J. A., 3rd, Prineas, R., Daviglus, M. L., Ning, H., Liu, K., Lewis, C. E., Sidney, S., Schreiner, P. J., Iribarren, C., & Lloyd-Jones, D. M. (2010). Prevalence of electrocardiographic abnormalities in a middle-aged, biracial population: Coronary Artery Risk Development in Young Adults study. *Journal of electrocardiology*, 43(5), 385.e1–385.e3859. <https://doi.org/10.1016/j.jelectrocard.2010.02.001>
- 34** Strogatz, D. S., Tyroler, H. A., Watkins, L. O., & Hames, C. G. (1987). Electrocardiographic abnormalities and mortality among middle-aged black men and white men of Evans County, Georgia. *Journal of chronic diseases*, 40(2), 149–155. [https://doi.org/10.1016/0021-9681\(87\)90066-x](https://doi.org/10.1016/0021-9681(87)90066-x)
- 35** Sutherland, S. E., Gazes, P. C., Keil, J. E., Gilbert, G. E., & Knapp, R. G. (1993). Electrocardiographic abnormalities and 30-year mortality among white and black men of the Charleston Heart Study. *Circulation*, 88(6), 2685–2692. <https://doi.org/10.1161/01.cir.88.6.2685>
- 36** Riley, C. P., Oberman, A., Hurst, D. C., & Peacock, P. B. (1973). Electrocardiographic findings in a biracial, urban population. The Birmingham Stroke Study. *The Alabama journal of medical sciences*, 10(2), 160–170.

