# Blood group and its correlation with cardiovascular risk factors: A community survey

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## Abstract

Cardiovascular disease is a leading cause of morbidity and mortality globally. Non-O blood groups have been shown to confer a high risk of coronary heart disease. There is paucity of data on the relationship between blood group and left ventricular hypertrophy, among other cardiovascular risk factors. A community-based study was done to evaluate the blood groups and their associations with conventional cardiovascular risk factors in Ejigbo. Anthropometry, blood pressure and other clinical variables were measured. Blood samples were taken for biochemical analysis for blood group typing, serum cholesterol and triglyceride assay. A 12-lead electrocardiogram was performed. The analysis was done using SPSS version 20 to determine possible relationships between the variables. Two hundred and six cases were recruited and analyzed. The prevalence of the various groups was as follows: A - 23%; B-31.4%; AB-4.4%; O-41.2%; rhesus-positive-92.7% and rhesus-negative-7.3%. Blood group B had similar systolic blood pressure (136.0±23.9 *vs* 137.3±22.3; p=0.726), higher Sokolow-Lyon voltage sum (3.3±1.1 *vs* 2.9±0.9; p=0.025) and serum triglycerides [14(21.9%) vs7(8.4%), p=0.021] than those with blood group O. Those with Rhesus Positive status had longer PR interval (169.3±25.4 *vs* 154.2±19.1; p value=0.055) and QRS duration (83.8±12.8 *vs* 78.4±7.6; p value=0.043) than those with Rhesus negative blood groups. Binary logistic regression revealed blood group B as an independent determinant of left ventricular hypertrophy(LVH) (OR: 3.028; p=0.012; 95% CI:1.275-7.192). Blood group B is a determinant of LVH. Rhesus positive status is associated with delayed electrical conduction through the myocardium.

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## Introduction

Non-O blood groups have been shown to confer a high risk of coronary heart disease , accounting for 6.27% of all cases.[1] A recent meta-analysis has also supported the earlier findings, showing that blood group A has the strongest predisposition to coronary heart disease (CHD) among the non-O blood groups.[2] Arterial strokes, peripheral vascular disease and myocardial infarction have all been shown to be increased in the Non-O blood group population.[3] Another study in the Chinese people showed that the non-O blood group, especially blood groups A and B, were independent predictors of myocardial infarction in those with established CVD.[4]

In the Multi-ethnic study of Atherosclerosis study, the A blood group was found to be associated with an increased prevalence of peripheral arterial disease in African Americans.[5] Also, a study in Senegal showed that blood group A was associated with a higher prevalence of ischaemic stroke and coronary artery disease.[6] However, there appears to be no study in indigenous Africans on the relationship between blood group and left ventricular hypertrophy, among other cardiovascular risk factors.

We therefore set out to evaluate the blood groups and their associations with electrocardiographic left ventricular hypertro-

phy(LVH), alongside other conventional cardiovascular risk factors in Ejigbo.

## Materials and Methods

### Study area

Ejigbo is a rural community in southwest Nigeria. It lies along Latitude 7.54'0N Longitude 4□18'54" E /Latitude 7.90000□ Longitude 4.31500□E. It is about 426 meters above sea level and it receives about 133cm depth of rain annually. The population was counted to be 132,515 in 2006 and is made up of traders and farmers, with teachers, commercial drivers and other professions composing the rest of its inhabitants.[7] The study was conducted at the field adjacent to Baptist Medical Centre, Ejigbo, Osun State. This study was a cross-sectional, descriptive study.

### Sample size determination

Using Leslie Kish formula, a minimum sample size of 185 was estimated for the study. Prevalence of left ventricular hypertrophy (12.5%) in a previous study was used.[8] Tolerable margin of error was set at 5% and a non-response rate of 10% was envisaged among the respondents and corrected for.

### Study population

The study population was made up of 206 people in the community. Subjects older than 18 years and who gave informed consent were included into the study. Females who were pregnant were excluded from the study. High blood pressure was defined according to WHO/ISH guideline: A SBP≥140mmHg and/or DBP ≥ 90 mmHg or being on treatment.[9]

Blood pressure (BP) was taken using the aneroid sphygmomanometer with appropriate cuff sizes. Blood pressure was taken after allowing at least 5 minutes rest and after ensuring that patients had not taken coffee or smoked cigarette within 30 minutes of taking BP. Systolic BP and DBP were taken as Korotkoff's sound I and V (disappearance) respectively. The BP readings were recorded to the nearest 2mmHg. Three BP readings were taken and the average reading was used for statistical analysis

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### Subject assessment

The study made use of a pretested, semi-structured interviewer-administered questionnaire. The WHO-STEPS questionnaire was adopted and the questionnaire, containing questions on demographic, socioeconomic status and habits of the subjects (sex, age, family status and educational levels, smoking and alcohol use etc) was administered under the supervision of the investigator.

Each participant had his/her weight (kg) measured using a standard hospital weighing scale with a weight limit of 120Kg and error margin of 0.4±0.3kg. The privacy of subjects was upheld during all measurements. Each subject was in minimal clothing with shoes off while being weighed. Height (m) was measured against a graduated height scale with patients in erect position and unshod.

Body mass index (BMI) was calculated from the weight and height as follows:

$BMI = weight\ (kg)/Height^2\ (m^2)$.

### Blood sample collection, storage and laboratory analysis

Venous blood was collected in plain universal and lithium heparin bottles and centrifuged at 3,000xg for 5 minutes. The plasma obtained was stored frozen at $-20^0$C before analysis. Plasma from each participant was analyzed in batches using standards and controls for all the biochemical parameters. Blood group determination was done using standard methods.

The enzymatic endpoint was used for lipid profile (Total Cholesterol, HDL-Cholesterol and Triglycerides) parameters except LDL-Cholesterol. Low-density lipoprotein cholesterol was calculated using Friedewald formula.[10] Dyslipidaemia classification for the cut off points are derived from the American Heart Association as follows: Total cholesterol>5.2mmol/L, Triglycerides >1.7mmol/L, Low-density lipoprotein >3.4mmol/L, and High-density lipoprotein <0.9mmol/L.[11] The definition of dyslipidaemia used in this study is an abnormality in at least one of the above domains.

### Electrocardiogram

Electrocardiography (ECG) – 12 lead resting ECG was done on all the subjects (using Nihon Kohden cardiofax Q-9130K and Cardimax FX-3101 devices). Left ventricular hypertrophy was defined using standard criteria.[12–16] Other ECG variables were assessed using standard protocol. Interpretation was done by an experienced cardiologist.

### Data management and analysis

All data obtained were entered into a standard proforma. Continuous variables were expressed as mean ± Standard Deviation [SD] while categorical variables were expressed as count (percentages). Data analysis was done using IBM SPSS Statistics for Windows, Version 20.0.

The Shapiro-Wilks test was used to assess for normality of distribution of variables. The means of continuous variables was compared using the Student's *t*-test for independent groups. For categorical variables, Chi-square and/or Fisher's exact test was applied to test the equality of distributions between the two groups. The demographic, anthropometric, laboratory and electrocardiographic characteristics of subjects were evaluated first, among the four ABO blood groups and between the two rhesus groups, using ANOVA and independent t-test respectively. Variables with significant relationships were entered into a model to evaluate for determinants of LVH. A two-tailed p-value of less than or equal to 0.05 was said to be significant.

## Results

Two hundred and six cases were recruited and analyzed. The prevalence of the various ABO groups was as follows: A -23%; B-31.4%; AB-4.4% and O-41.2%. The prevalence of the Rhesus groups was as follows: rhesus-positive-92.7% and rhesus-negative-7.3%.

### LVH and blood groups

Major electrocardiographic LVH criteria were utilized in the analysis- most did not show significant differences among the groups. Only those that were significant (or showed a trend towards significance) were included in the table. The Sokolow-Lyon criteria was the only one that showed a difference, with blood groups B and A showing prevalence values of 42% and 27% for

LVH (Sokolow-Lyon) respectively. None of the subjects with blood group AB had LVH (0%).

The Estee point score ≥4 in males showed that those with blood group O had a tendency towards having LVH. Only 60 people had ever consumed alcohol in the entire cohort of patients. Among these, only 20 of them consumed alcohol within the last 12 months- 4(A), 8(B), 0(AB) and 5(O) ($p=0.777^F$); out of which only 14 drank alcohol in the previous 30 days: 1(A), 5(B), 0(AB) and 5(O). Nine people also smoked cigarettes currently: 3(A), 1(B), 1(AB) and 4(O). Details are seen in Table 1.

Higher serum triglycerides were found in the non-O as compared to the O group [1.14±0.88 vs 0.91±0.65; p= 0.042]. Also, the P-wave amplitude in V1 [0.02±0.03 vs 0.008±0.02; p=0.018] and lead II [0.073±0.16 *vs* 0.033±0.07; p=0.073] were higher in the non-O than the O group, suggesting larger right atria in the former. There was also a tendency towards having left ventricular hypertrophy in those in the non-O group, largely due to significantly deeper S waves in V1. Details are seen in Table 2.

**Table 1. Showing the distribution of various variables within the ABO blood group system.**

| Variables | Blood Group A, n=44 (%) | Blood Group AB, n=9 (%) | Blood Group B, n=62 (%) | Blood Group O, n=80 (%) | P-value |
|---|---|---|---|---|---|
| Sex | | | | | |
| Male | 18 | 2 | 15 | 17 | 0.163 |
| Female | 26 | 7 | 47 | 63 | |
| Elevated Triglycerides | 5(11.1) | 3(33.3) | 14(21.9) | 7(8.4) | 0.054 |
| Elevated LDL | 22(48.9) | 5(55.6) | 22(34.4) | 27(32.5) | 0.233 |
| Low HDL | 12(26.7) | 2(22.2) | 6(9.4) | 12(14.5) | 0.045* |
| Hypertension | 23(60.5) | 5(55.6) | 32(56.1) | 54(72) | 0.163 |
| Ever consumed alcohol | 20(47.6) | 2(22.2) | 13(57) | 17(22) | 0.017* |
| Vigorous intensity work for 10 minutes | 9(23.7) | 0(0) | 13(22.4) | 13(17.6) | 0.025* |
| Moderate intensity Work for 10 minutes | 10(27) | 2(22.2) | 20(35.7) | 19(25.7) | 0.019* |
| Sokolow Lyon V1+V5 | 10(27) | 0(0) | 21(42) | 11(19.3) | 0.017* |
| Cornell Male (n=57) | 4(23.5) | 2(100) | 0(0) | 0(0) | 0.000* |
| Cornell Volt Product (Male) | 3(21.4) | 1(50) | 0(0) | 0(0) | 0.023* |
| Perugia (Male) | 7(41.2) | 2(100) | 1(7.7) | 4(28.6) | 0.037* |
| Estee Point score≥4(Males) | 5(29.4) | 1(50) | 2(14.3) | 9(64.3) | 0.058 |

$X^2$ of Sokolow Lyon V1V5:12.443; *: P-value ≤ 0.05.

**Tables 2. Shows the clinical, electrocardiographic and laboratory variables between those with Blood groups O *vs* non-O.**

| Variables | O vs Non-O n/n | O group (%) | Non-O group (%) | P-value |
|---|---|---|---|---|
| Age (years) | 80/165 | 59.9±15.8 | 57.8±16.5 | 0.354 |
| Sex | 80/167 | | | |
| Male | | 17 | 46 | 0.288 |
| Female | | 63 | 121 | |
| Serum Triglycerides | 83/133 | 0.9±0.7 | 1.1±0.9 | 0.042* |
| Elevated Triglycerides | 83/133 | 7(8.4) | 25(18.8) | 0.037* |
| Hypertension | 75/142 | 54(72) | 89(62.7) | 0.168 |
| Sokolow-Lyon-Rappaport (V2+V6) | 57/139 | 4(7) | 21(15.1) | 0.159 |
| Normal body weight category | 77/149 | 21(27.3) | 54(36.2) | 0.184 |
| Maximum attained education | 74/144 | | | |
| Primary education(≤6yrs in school) | | 43(58.1) | 64(44.4) | 0.056 |
| Secondary education (≤12yrs in school) | | 63(85.1) | 88(61.1) | 0.000* |
| P amplitude in lead II | 54/136 | 0.03±0.1 | 0.07±0.2 | 0.073 |
| P wave (initial force) in V1 | 51/136 | 0.01±0.02 | 0.02±0.03 | 0.018* |
| P axis | 55/129 | 58.5±25.5 | 49.2±32 | 0.058 |
| $SV_1$ | 56/142 | 1.04±0.59 | 1.22±0.56 | 0.045* |
| $SV_1RV_6$ | 57/141 | 2.31±0.88 | 2.55±0.89 | 0.082 |
| R in lead I | 56/137 | 0.8±±0.4 | 0.9±0.4 | 0.014* |
| T axis | 53/133 | 52.3±42.8 | 40.3±25.1 | 0.019* |

*: P-value ≤ 0.05.

Those with blood group O had lower serum triglycerides and lower voltage sums for LVH as compared to those with blood group B. This was in spite of the fact that those who were diagnosed with hypertension were commoner in the blood group O. See Figures 1 and 2.

Those with blood group A have taller voltage sums (Sokolow Lyon and Gubner-Ungerleider) for LVH, longer PR intervals and a tendency to have wider pulse pressures than those with blood group AB. See Figure 3.

The subgroup study of electrocardiographic LVH among the ABO blood groups using the Bonferroni adjustments in ANOVA showed that the Sokolow-Lyon voltage sum was significantly different specifically between blood groups B and AB. Therefore, a T-test was done to evaluate the same variables between blood groups B and AB. In addition to having higher Sokolow-Lyon LVH voltages, the systolic blood pressure and pulse pressure were also higher in those with B group while diastolic blood pressure was not. However, the heart rate of those with the AB group was higher. Also, LDL-C values and total cholesterol-HDL ratios were higher in those with AB than those with B. See Figure 4.

Those who were rhesus positive had greater incidence of left atrial enlargement, longer PR interval and QRS duration than those who were rhesus negative. The waist and hip circumferences showed a trend towards being higher in those with a rhesus positive status. However, the Rhesus negative group had higher LDL cholesterol levels. Details are seen in Table 3.

Subjects with blood group AB had lower blood pressures [121.7±13.9 *vs* 137.3±22.3;p=0.043], better pulse pressures [43.9±10.3 *vs* 56±15.1;p=0.022], a tendency towards having less sum of RV5+SV1, (criterion used for LVH) but paradoxically worse dyslipidaemia (all AB subjects had dyslipidaemia in one domain or the other) than those with blood group O[ 9(100%) *vs* 45(54.2%);p=0.009]. Those with blood group AB also had a tendency to complain of chest pain more than those with blood group O [5(55.6%) *vs* 20(26.3%); p=0.116]. There also seemed to be an inverse relationship between education and hypertension- those

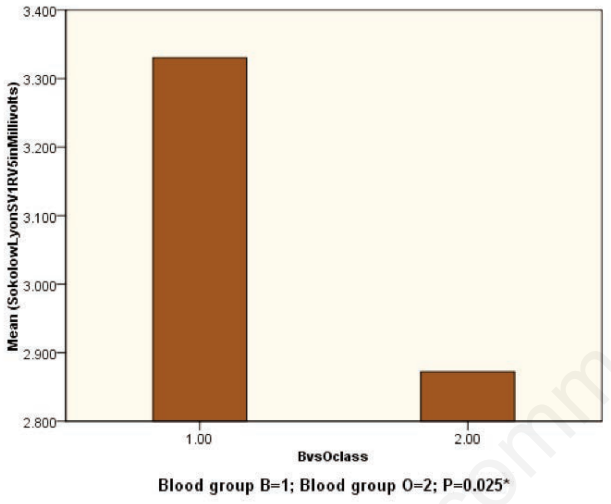


**Figure 1. Showing left ventricular hypertrophy between those with blood groups B and O. *:P ≤ 0.05.**

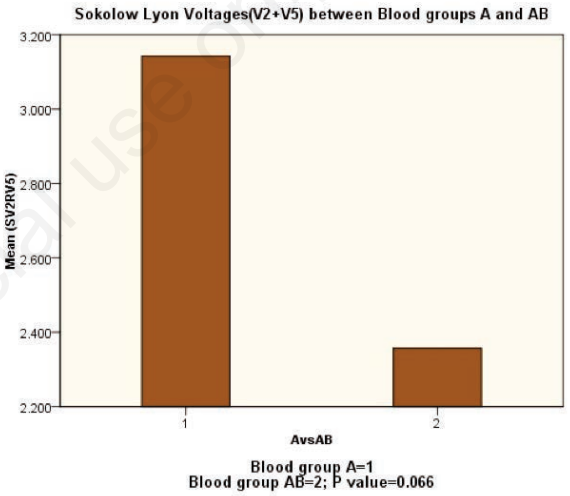


**Figure 3. Shows the mean voltages for left ventricular hypertrophy (Sokolow-Lyon-Rappaport) between blood groups A and AB.**

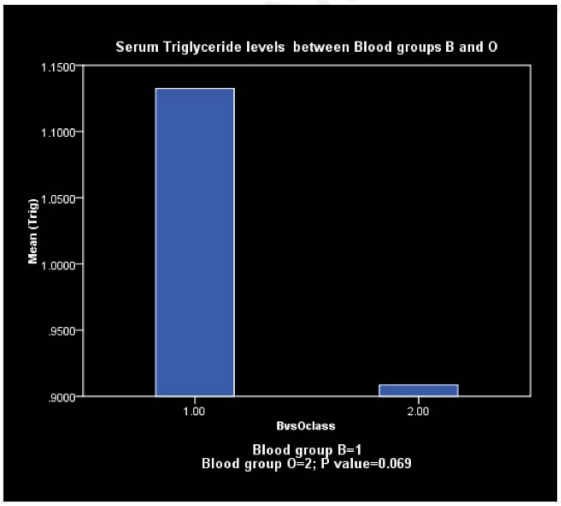


**Figure 2. Shows the mean Triglyceride levels between blood groups B and O.**

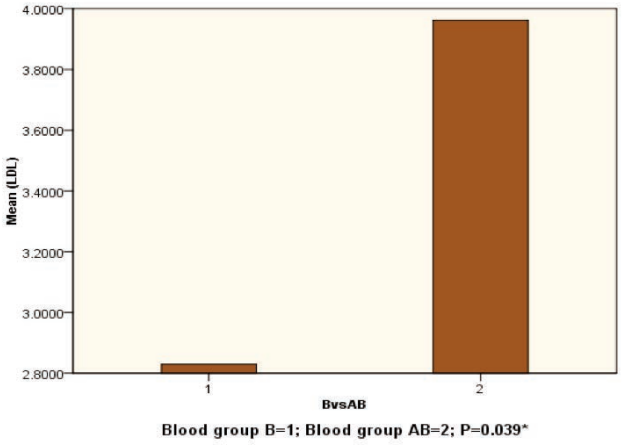


**Figure 4. Shows the mean low-density lipoprotein (LDL) levels between blood groups B and AB *:P ≤0.05.**

who were more educated (blood group AB) were less hypertensive [5(55.6%) *vs* 63(85.1%); p=0.052].

When binary logistic regression was done to assess for the blood group determinants of LVH, only blood group B was an independent determinant of LVH, accounting for between 8.0 and 11.5% of the LVH seen among the study subjects. See Table 4.

## Discussion

In this study, in the ABO blood group, those with non-O blood groups had less secondary education 88(61.1%) *vs* 63(85.1%)p=0.000, had higher serum triglyceride levels [1.14±0.88 *vs* 0.91±0.65;p=0.042], taller P wave amplitudes (in leads V1) 0.02±0.03 *vs* 0.008±0.02; p=0.018and a tendency to having higher electrocardiographic 'left ventricular hypertrophy' voltages than those with O blood group [2.55±0.89 *vs* 2.31±0.88; p=0.082]. Elevated serum triglyceride levels has been shown to increase the odds of developing LVH in the general populace.[17] Those with blood group B had significantly higher incidence of LVH, with 3-fold the odds of developing LVH than those with blood group O. This was in spite of the fact that those with O blood group had a tendency to be more hypertensive, though the measured blood pressure values were essentially same.

The N-acetylgalactoseamine and D-galactose side chains that define the A and B blood groups respectively interact with von Willebrand factor to promote platelet aggregation and thrombus formation.[18]These antigenic determinants interact with endothelium-derived adhesion molecules to cause cardiovascular diseases.[19] Interestingly, the ABO 'blood group' antigens are not specific to blood cells, but are found in salivary glands, respiratory epithelium and the gastrointestinal epithelium and have been found to be determinants of non-cardiovascular diseases and malignancies.[20] These blood group antigens have been shown to exhibit wide functional diversity, being involved with transcellular transport, serving as structural proteins, enzymes and adhesion molecules, among others.[21]

In post-menopausal women and hypertensive adults with cere-

**Table 3. Showing the clinical, laboratory and electrocardiographic parameters between those with Rhesus positive and Rhesus Negative blood groups.**

| Variables | Rhesus neg/positive n/n | Rhesus Negative | Rhesus Positive | P-value |
|---|---|---|---|---|
| Sex | 15/182 | 6 | 46 | 0.214 |
| Male | | 9 | 136 | |
| Female | | | | |
| Moderate intense work for 10min | 13/165 | | | |
| Yes | | 1 | 52 | 0.112 |
| No | | 12 | 113 | |
| Occupation | 15/178 | | | 0.054 |
| Government employee | | 0 | 14 | |
| Non-Government employee | | 0 | 14 | |
| Self employed | | 13 | 94 | |
| Student | | 1 | 4 | |
| Home maker | | 0 | 1 | |
| Retired | | 0 | 41 | |
| Unemployed-able to work | | 0 | 7 | |
| Unemployed-unable to work | | 1 | 3 | |
| Left atrial enlargement | 10/128 | | | |
| Yes | | 2 | 68 | 0.053 |
| No | | 8 | 60 | |
| Abnormal LDL class | 15/188 | | | |
| Yes | | 9 | 68 | 0.067 |
| No | | 6 | 120 | |
| Total cholesterol (mmol/L) | 15/188 | 5.9±2.6 | 5.2±1.7 | 0.127 |
| Low density lipoprotein (mmol/L) | 15/188 | 3.8±1.9 | 3.0±1.5 | 0.059 |
| PR interval (msec) | 11/140 | 154.2±19.1 | 169.3±25.4 | 0.055 |
| QRS duration[#] (msec) | 12/140 | 78.4±7.6 | 83.8±12.8 | 0.043* |
| Waist circumference (cm) | 14/171 | 83.9±12 | 89.4±12.3 | 0.110 |
| Hip circumference (cm) | 14/170 | 94.7±11.1 | 100.9±11.9 | 0.064 |
| Body Surface area ($m^2$) | 15/169 | 1.6±0.2 | 1.7±0.2 | 0.148 |

[#]Not normally distributed; *: P-value ≤ 0.05.

**Table 4. Binary Logistic regression showing the impact of blood group on electrocardiographic LVH(V1+V5) using blood group O as reference.**

| Variables | Odds ratio | P value | Confidence Interval |
|---|---|---|---|
| Blood group A | 1.549 | 0.381 | 0.582-4.124 |
| Blood group B | 3.028 | 0.012* | 1.275-7.192 |
| Blood group AB | 0.000 | 0.999 | 0.000 |

*: P-value ≤ 0.05; R2: Cox and Snell-0.080; Nagelkerke: 0.115.

brovascular and cardiovascular disease, the A blood group was found by Wiggins *et al.*[22] to confer increased odds of having ventricular tachycardia (OR:1.79; CI: 1.41-2.26) and myocardial infarction (OR:1.23; CI:1.05-1.44, as compared to O) while the B blood group had higher odds of both ventricular tachycardia (OR:1.82; CI:1.29-2.57) and ischaemic stroke (OR:1.59; CI:1.17-2.17).[22] However, their study was among those with established CVD, not a general population-based research. Also, in those with familial hypercholesterolaemia, patients with the non-O blood group had >2-fold prevalence of cardiovascular disease as compared to the blood group O patients.[23] The non-O blood group has also been found to have higher odds of having non-alcoholic fatty liver disease, especially blood groups A and B.[24]

The mean pulse pressure of those with the AB groups in this study was less than those with blood group O, with associated reduced electrocardiographic LVH. Increased pulse pressure has been found to be a risk factor for myocardial infarction, increased LV mass and to be predictive of increased cardiovascular mortality.[25,26]

One would think that the presence of both antigenic epitopes on the blood cells in the AB population would confer a higher cardiovascular risk profile on them but that seems not to be the case. Instead, there seem to be a dousing effect when both are present, reducing the cardiovascular risk as compared to those with either A or B blood groups. This is in agreement with the findings of Biswas et al.[27] Most available literature implicates either blood groups A or B as the main culprit in different population groups.[2,28,29] Interestingly, the total cholesterol and LDL-cholesterol in this study were higher in the AB populace than those with blood group B while the systolic blood pressure and left ventricular hypertrophy indices were higher in the latter.

Those who were rhesus positive had more left atrial enlargement, longer PR interval and QRS duration than those who were rhesus negative (it seems that the rhesus antigen delays the conduction of action potentials across the entire myocardium). The waist and hip circumferences showed a trend towards being more in those with a rhesus positive status. However, the Rhesus negative group had higher LDL cholesterol levels.

## Conclusions

The Non-O blood groups, especially blood group B had higher blood pressure values, left ventricular hypertrophy and serum triglycerides than those with blood group O. The AB populace tend to have higher total cholesterol and LDL cholesterol than blood group B (which seems to have the highest cardiovascular risk profile due to LVH). These suggest that the various ABO blood group types have their peculiarities and requesting for blood group with a view to assessing cardiovascular risk should be given a strong consideration.

Rhesus positive status was associated with delayed electrical conduction through the myocardium, especially from the atrioventricular node to the ventricular cells. This may necessitate an adjustment of the normal PR and QRS durations based the Rhesus status of people in the general populace.

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