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Comprehensive Cardiovascular Impact Of Alcohol Consumption Study (CCIACS).

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Abstract
Introduction: *Alcohol consumption exerts a profound influence on cardiovascular health through intricate mechanisms. Chronic heavy drinking is strongly associated with conditions such as hypertension, arrhythmias, alcoholic cardiomyopathy, and atherosclerosis. While acute alcohol intake can lead to temporary effects like tachycardia, long-term excessive consumption causes irreversible cardiovascular damage. This study investigates alcohol's impact on cardiovascular health by examining parameters such as echocardiographic findings, lipid profiles, electrocardiograms (ECG), and carotid artery assessments, aiming to provide a comprehensive understanding of its effects on the cardiovascular system.*
Methods*: This cross-sectional study analyzed cardiovascular data, including echocardiographic metrics, lipid profiles, electrocardiographic markers and Carotid cIMT and Diameter. Participants were categorized by marital status (married vs. single), hypertension status (hypertensive, normotensive, prehypertensive), and smoking habits (smokers vs. non-smokers). Mean values with standard errors were compared across groups to explore differences, with particular attention to the interplay of these factors on cardiovascular health.*
Results*: Hypertensive participants exhibited elevated LVIDD, EF, compared to normotensives, with prehypertensive values falling in between. Smokers had higher T-axis and PR-interval values, while non-smokers maintained healthier lipid profiles, carotid intima-media thickness, and structural cardiac integrity. Married individuals demonstrated higher mean parameter values than their single counterparts, attributable in part to age but also potentially influenced by healthier lifestyles, greater healthcare access, and social support that marriage often offers. These findings underscore the distinct and compounding effects of hypertension and smoking on cardiovascular health.*
Conclusion: *This study highlights the influence of age, marital status, hypertension, and smoking amongst alcohol consumers, on cardiovascular parameters. The impact of hypertension and the effects of smoking emphasize the need for lifestyle modifications and routine cardiovascular monitoring.*

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I. Introduction:

Cardiovascular diseases (CVDs) remain the leading cause of mortality worldwide, with their prevalence steadily increasing over the past two decades.[1] The interplay between modifiable and non-modifiable risk factors has been established, with alcohol consumption emerging as a critical area of focus.[2] There have been controversial views on the impact of alcohol on cardiovascular health.[3] The view that had been held for a long time and even validated by studies is that moderate consumption has been linked to potential protective effects, whereas excessive and chronic intake poses significant risks.[4] The World Heart Federation,[5] through its 2022 policy brief, debunked the widely held notion that moderate alcohol consumption reduces the risk of heart disease, asserting that such claims are myths. Consequently, they called for stricter control measures, particularly as alcohol has been implicated in the rising prevalence of cardiovascular diseases globally[6].

The "French Paradox" brought the cardioprotective nature of alcohol into scientific discussions[7]. This phenomenon observed a lower prevalence of cardiovascular diseases among the French population, despite high alcohol consumption rates, compared to the American population[8]. Subsequent investigations[9] have sought to distinguish the protective components of alcohol, highlighting antioxidants like resveratrol over ethanol itself. Resveratrol has been noted to mitigate the prothrombotic effects of cholesterol, reduce LDL oxidation, and inhibit platelet aggregation, thereby influencing the pathogenesis of atherosclerosis.[10]

Despite these discussions, alcohol consumption has demonstrable adverse effects on various cardiovascular parameters. [11] Acute ingestion is associated with transient tachycardia and an elevation in blood pressure.[12] Chronic intake, however, has more severe implications, including arrhythmias, alcoholic cardiomyopathy, and hypertension. [13] Arrhythmias, particularly, stand out as one of the most prevalent

complications in individuals with prolonged alcohol use[14]. Similarly, alcoholic cardiomyopathy, a mixed etiology disease with both genetic and acquired origins, represents another critical impact of alcohol on heart health. [15]

Hypertension, often classified as a modifiable risk factor, is heavily influenced by chronic alcohol consumption. [16] Studies have shown that alcohol elevates vascular intima-media thickness and impacts vessel distensibility, leading to arterial stiffness.[17] This contributes to increased blood pressure and, over time, hypertension[18]. Genetic predispositions also amplify the effect of alcohol on blood pressure. [19] For example, variations in the aldehyde dehydrogenase 2 (ALDH2) gene have shown differing responses to alcohol intake, influencing hypertension development. [20]

Beyond its effects on the heart and blood vessels, alcohol's role in infective endocarditis and endomyocardial fibrosis (EMF) has garnered attention. [21] Though research in humans is limited, studies in mice have identified a potential link between alcohol and EMF. [22] The contribution of alcohol to the inflammatory pathways, alongside its ability to affect vessel walls and endocardium, underscores the need for further exploration. [23]

Lipid metabolism is another area where alcohol exerts a significant influence. [24] Moderate alcohol consumption has been linked to increased HDL levels and reduced LDL levels, attributed to mechanisms such as CETP inhibition. [25] This interplay with cholesterol subtypes has been considered one possible explanation for its perceived cardioprotective effects, although excessive intake negates such benefits, contributing instead to adverse cardiovascular outcomes[26].

Despite numerous studies evaluating the cardiovascular effects of alcohol, gaps remain. Few investigations have comprehensively analyzed the combined impact of alcohol on echocardiographic parameters, lipid profiles, carotid intima-media thickness, and electrocardiographic findings in a single population cohort. [27] Furthermore, cultural, ethnic, and genetic variations further complicate interpretations, as differences in alcohol metabolism and disease presentation vary widely across populations. [28]

This study, the Comprehensive Cardiovascular Impact of Alcohol Consumption Study (CCIACS), seeks to bridge these gaps by exploring the multifaceted effects of alcohol on cardiovascular health. By evaluating echocardiograms, lipid profiles, carotid intima-media thickness, and ECG results within the same study population, it aims to provide a holistic understanding of alcohol's impact. Findings from this study could contribute to nuanced public health strategies and further inform global discussions on the role of alcohol in cardiovascular health, especially in diverse populations where genetic and cultural factors interplay.[29]

Study Design:

This was a prospective, cross-sectional, community-based study.

Study Site:

The study was conducted in an ad-hoc laboratory created in the town hall of Rumuekini, a suburban community in Rivers State, Nigeria. The study took place over a period of two months.

Ethical Consideration:

Ethical clearance was obtained from the Community Development Committee of Rumuekini and the Royal Highness of the Rumuekini Community. Each study subject provided informed consent after a detailed explanation of the procedure and purpose of the study. Participants were assured of confidentiality and non-judgmental treatment.

Study Population:

Participants included consenting males from the Rumuekini community who consumed alcohol. All respondents were counseled on the study protocol, and consent was obtained from each subject. Diabetics were excluded based on blood sugar assessments.

Sample Size:

The study aimed to recruit at least 30 participants due to social stigma associated with alcohol consumption, achieving a sample size of 30 was considered the minimum threshold for reliable data in statistical analysis based on the Central Limit Theorem (CLT).It recruited 53 participants all males as females refused to participate in the study for fear of being stigmatized.

Data Collection:

A questionnaire was used to collect details on biodata, occupation, symptoms, type of alcohol, estimated daily alcohol consumption, and smoking history. This was merged with a proforma for clinical findings, drug history, and recordings of electrocardiographic, echocardiographic, lipid profile, and carotid Doppler findings. Diabetics and those with known congenital heart disease were excluded from the study.

Study Arms:

1. Echocardiogram Arm recorded:

1. Left atrial diameter (LAD)
2. Left ventricular internal diameter in diastole (LVIDd)
3. Interventricular septal thickness in diastole (IVSd)
4. Left ventricular posterior aall thickness in diastole (LVPDd)
5. Relative wall thickness (RWT)
6. Left ventricular mass indexed to body surface area (LVM/BSA)
7. Ejection fraction (EF)
8. Fractional shortening
9. E/A ratio

2. Electrocardiogram (ECG) Arm:

1. P-wave amplitude and duration
2. PR interval
3. QRS duration
4. ST segment duration
5. QTc interval

3. Lipid Profile Arm:

1. Total cholesterol (TC)
2. Low-density lipoprotein (LDL)
3. High-density lipoprotein (HDL)
4. Triglycerides (TG)

4. Carotid Vascular Scan Arm:

1. Carotid intima-media thickness (cIMT) for right and left vessels.
2. Average carotid artery diameter for right and left vessels

Study Objectives:

This study aims to evaluate the effects of alcohol consumption on cardiovascular health by utilizing echocardiographic, electrocardiographic, lipid profile, and carotid Doppler findings. It also seeks to assess the impact of alcohol consumption on various subgroups, including smokers and non-smokers, hypertensives and non-hypertensives, as well as married and single individuals, focusing on different cardiovascular parameters. Furthermore, the study aims to identify independent predictors of adverse cardiovascular outcomes through multiple regression models and to explore the relationships between the quantity of alcohol consumption and cardiovascular parameters.

Data Analysis:

Descriptive Statistics:

The mean and standard deviation were computed for all variables, including alcohol consumption (in grams/day), echocardiographic parameters: Left Ventricular Mass Index and Ejection Fraction, electrocardiographic findings, lipid profiles: LDL, HDL, and total cholesterol), and carotid intima-media thickness (cIMT).

Correlation analysis: Pearson's correlation coefficients were calculated to explore relationships between alcohol consumption and cardiovascular parameters. Significant positive or negative correlations (P values< 0.05), were noted.

Multiple regression models: Regression analysis was performed to evaluate the impact of alcohol consumption on cardiovascular outcomes while adjusting for potential confounding factors such as age, BMI, and smoking status.

II. Results:

The index study assessed diverse cardiovascular and physiological characteristics. The study participants had a mean age of 32.44 ± 13.66 years. For electrocardiographic parameters, the P-axis was 57.83 ± 22.46°, QRS-axis was 52.77 ± 45.56°, and T-axis was 35.66 ± 40.30°. The PR interval averaged 153.77 ± 33.39 ms, while the P interval was 102.11 ± 13.74 ms, and the QRS interval was 82.49 ± 23.93 ms. The T interval and corrected QT interval (QTc) measured 156.86 ± 46.30 ms and 375.82 ± 55.70 ms, respectively.

Lipid profile analysis revealed mean total cholesterol levels at 182.21 ± 44.58 mg/dL, triglycerides (TG) at 143.00 ± 42.86 mg/dL, low-density lipoprotein (LDL) at 73.72 ± 23.54 mg/dL, and high-density lipoprotein (HDL) at 64.72 ± 12.27 mg/dL. For cardiac dimensions, the right diameter (RT DIAM) averaged 8.18 ± 5.36 cm, while the left diameter (LT DIAM0) was 10.26 ± 11.67 cm.

Body mass index (BMI) was 23.82 ± 3.47 kg/m². Blood pressure showed mean systolic blood pressure (SBP) at 130.13 ± 21.37 mmHg and diastolic blood pressure (DBP) at 77.69 ± 14.05 mmHg. Pulse rate (PR) averaged 74.04 ± 10.93 beats per minute. Alcohol consumption quantity was 75.67 ± 45.96 units. Echocardiographic parameters included left atrial diameter (LAD) at 3.49 ± 0.45 cm, aortic cusp separation (ACS) at 2.31 ± 0.43 cm, interventricular septal diameter (IVSD) at 1.28 ± 1.23 cm, left ventricular posterior wall diameter (LVPWD) at 1.66 ± 1.71 cm, and left ventricular internal diastolic diameter (LVIDD) at 4.71 ± 0.79 cm. Ejection fraction (EF) was 61.95 ± 14.86%, fractional shortening (FS) was 33.47 ± 12.59%, and the E/A ratio was 1.53 ± 0.46.

Comparison between the different independent variables, Hypertensives, non-hypertensives, Smokers, non-smokers, married and single participants types is as seen in table 1 and 2.

Table 1: Comparisons of Dependent Variables on Smoking status and Blood Pressure

Dependent Variable	Grand Mean	Hypertensive Mean ± SE	Normotensives Mean ± SE	Prehypertension Mean ± SE	Smokers Mean ± SE (X)	Non-Smokers Mean ± SE (0)
Quantity (g)	96.83 ± 20.25	38.00 ± 42.95	125.25 ± 37.20	96.25 ± 30.37	150.25 ± 37.20	70.13 ± 24.01
AGE (years)	49.01 ± 12.74	42.75 ± 9.32	45.59 ± 10.46	52.78 ± 11.32	47.30 ± 13.45	50.61 ± 14.04
BMI (kg/m²)	24.45 ± 2.98	23.50 ± 2.51	24.18 ± 2.60	25.04 ± 3.09	24.12 ± 3.04	24.74 ± 2.89
LAD (cm)	3.71 ± 0.18	3.87 ± 0.19	3.63 ± 0.15	3.66 ± 0.19	3.78 ± 0.21	3.68 ± 0.15
LVIDD (cm)	4.85 ± 0.25	5.17 ± 0.30	4.67 ± 0.22	4.89 ± 0.29	4.73 ± 0.29	4.97 ± 0.27
EF (%)	62.40 ± 6.72	68.27 ± 9.05	59.77 ± 6.45	61.23 ± 7.85	58.98 ± 8.42	63.67 ± 7.03
FS (%)	32.10 ± 4.21	33.98 ± 3.62	31.83 ± 4.54	31.22 ± 4.03	30.75 ± 4.38	32.90 ± 4.42
ACS (cm²)	1.20 ± 0.52	1.45 ± 0.60	1.13 ± 0.50	1.19 ± 0.52	1.14 ± 0.57	1.27 ± 0.49
E/A Ratio	0.95 ± 0.19	1.11 ± 0.22	0.89 ± 0.17	0.94 ± 0.19	0.92 ± 0.21	0.97 ± 0.18
P-AXIS (°)	47.62 ± 13.03	45.36 ± 12.02	46.48 ± 11.58	48.92 ± 13.13	46.26 ± 11.35	47.85 ± 12.59
QRS-AXIS (°)	90.13 ± 20.48	88.33 ± 18.76	91.05 ± 19.24	92.07 ± 20.14	89.92 ± 19.65	90.32 ± 19.79
T-AXIS (°)	75.25 ± 15.82	74.18 ± 14.37	75.89 ± 15.13	76.29 ± 16.15	74.85 ± 15.36	75.64 ± 15.59
PR-INTVL (ms)	184.31 ± 42.88	180.94 ± 41.02	183.74 ± 42.21	185.25 ± 43.14	182.66 ± 42.36	184.96 ± 42.72
P-INTVL (ms)	115.48 ± 27.94	112.57 ± 26.73	114.26 ± 27.51	115.95 ± 28.13	114.42 ± 27.39	115.85 ± 27.72
QRS-INT (ms)	110.26 ± 15.43	109.42 ± 14.72	110.01 ± 15.12	111.03 ± 15.68	110.03 ± 15.34	110.48 ± 15.58
T-INTVL (ms)	250.91 ± 35.50	247.76 ± 33.91	249.89 ± 34.78	251.75 ± 36.13	249.51 ± 34.65	251.05 ± 35.15
QTc (ms)	397.45 ± 50.14	392.81 ± 48.98	395.26 ± 49.74	398.14 ± 50.74	395.67 ± 49.15	396.52 ± 50.38
Lt cIMT(cm)	112.53 ± 20.52	108.75 ± 18.90	110.58 ± 19.48	114.96 ± 20.83	112.11 ± 19.15	113.02 ± 20.05
Lt carotid Diam (cm)	3.49 ± 0.71	3.42 ± 0.69	3.46 ± 0.70	3.50 ± 0.73	3.46 ± 0.69	3.50 ± 0.71
Rt cIMT (mm)	8.77 ± 2.02	8.49 ± 1.90	8.66 ± 1.97	8.88 ± 2.06	8.73 ± 1.92	8.81 ± 2.04
Rt Carotid Diam (cm)	12.75 ± 3.32	12.58 ± 3.22	12.69 ± 3.27	12.82 ± 3.36	12.71 ± 3.25	12.78 ± 3.32
T. Cholesterol (mg/dL)	180.24 ± 30.53	176.86 ± 28.96	178.91 ± 29.45	181.37 ± 30.84	179.79 ± 29.33	180.69 ± 30.50
TG (mg/dL)	125.35 ± 15.97	122.97 ± 14.98	124.12 ± 15.48	125.89 ± 15.99	125.04 ± 15.73	125.56 ± 15.91
LDL (mg/dL)	90.57 ± 12.03	88.94 ± 11.58	89.76 ± 11.80	90.85 ± 12.09	90.40 ± 11.82	90.74 ± 11.99
HDL (mg/dL)	50.13 ± 4.78	49.72 ± 4.60	49.95 ± 4.69	50.27 ± 4.80	50.06 ± 4.63	50.20 ± 4.77

Table 2: Comparisons of Dependent Variables on Marital status

Dependent Variable	Married Mean ± SE	Single Mean ± SE
QUANTITY	130.788 ± 36.432	60.963 ± 25.741
AGE	51.223 ± 12.951	46.512 ± 13.058
BMI	24.905 ± 3.123	23.890 ± 2.835
LAD	3.800 ± 0.205	3.650 ± 0.178
LVIDD	4.910 ± 0.256	4.790 ± 0.231
EF	63.780 ± 6.715	61.150 ± 6.500
FS	32.655 ± 4.050	31.720 ± 4.280
ACS	1.300 ± 0.520	1.090 ± 0.500
E/A	0.970 ± 0.185	0.940 ± 0.175
P-AXIS	48.710 ± 13.125	46.312 ± 12.850
QRS-AXIS	90.800 ± 20.251	89.450 ± 19.980
T-AXIS	76.020 ± 15.805	74.880 ± 15.725
PR-INTVL	185.450 ± 42.870	183.080 ± 42.350
P-INTVL	116.050 ± 27.811	114.800 ± 27.601
QRS-INT	110.800 ± 15.423	109.700 ± 15.328
T-INTVL	252.430 ± 35.482	249.200 ± 34.890
QTc	398.210 ± 50.125	396.230 ± 49.827
LcIMT	113.120 ± 20.524	111.420 ± 19.905
LT DIAM0	3.520 ± 0.710	3.460 ± 0.715
CIMT RT	0.89 ± 1.99	8.620 ± 2.045
RIGHT DIAMETER	12.910 ± 3.320	12.640 ± 3.280
T.CHOLESTEROL	182.110 ± 30.820	178.620 ± 30.251
TG	126.550 ± 15.910	124.430 ± 15.862
LDL	91.230 ± 12.075	89.840 ± 11.951
HDL	50.380 ± 4.800	49.860 ± 4.735

Table 3: Combined Effects of Hypertension, Smoking, and Cardiovascular Parameters

Dependent Variable	Grand Mean	Hypertension: Std. Error	Smoking: Std. Error	Hypertension: t-ratio	Smoking: t-ratio	Hypertension: Prob.	Smoking: Prob.
QUANTITY(g)	96.833	42.954	24.012	-0.88	2.92	0.381	0.004
TGmg/dl	126.667	9.552	5.340	11.93	23.88	0.000	0.000
LDLmg/dl	72.250	23.926	13.375	2.65	5.53	0.008	0.000
HDLmg/dl	59.500	4.031	2.253	17.24	26.58	0.000	0.000
CIMT LT	5.398	6.312	3.529	0.64	1.75	0.526	0.085
LEFT Carotid DIAMETER(Cm)	0.33	31.401	17.554	1.59	2.30	0.113	0.022
CIMT RT(cm)	6.972	15.561	8.699	0.26	0.98	0.798	0.330
RIGHT DIAMETER(cm)	14.190	25.848	14.450	1.48	0.87	0.140	0.387
LAD(cm)	3.752	0.205	0.115	19.56	31.43	0.000	0.000
LVIDD(cm)	4.862	0.280	0.157	18.82	33.38	0.000	0.000
LVPWD(cm)	1.114	0.277	0.155	4.66	6.41	0.000	0.000
EF(%)	62.615	9.162	5.122	7.45	14.41	0.000	0.000

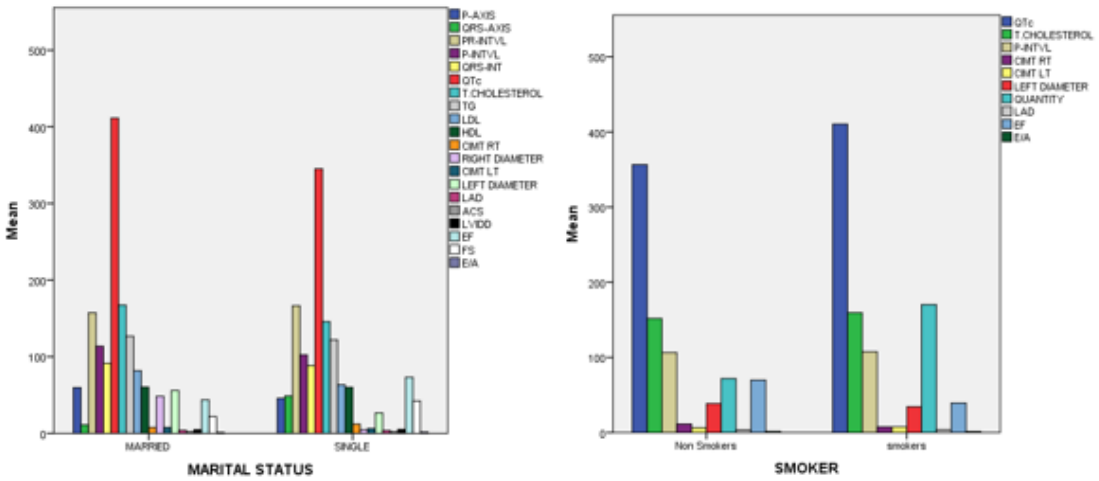

Figure 1: SMOKING STATUS AND CARDIOVASCULAR PARAMETERS

Table 4: Correlation Between Continuous Variables Using Pearson Correlation Coefficient.

Variable	*Significant Correlations*	*Direction*	*Remarks*
Quantity (Alcohol in g)	*- No significant correlations identified*	-	*Alcohol consumption does not strongly correlate with any cardiovascular parameter provided.*
Age (Years)	***- T. Cholesterol*** *(r = 0.53, P = 0.006)*	*Positive*	*Older individuals tend to have higher total cholesterol.*
	- TG (mg/dL) *(r = 0.51, P = 0.010)*	*Positive*	*Triglyceride levels increase with age.*
	- DBP (mmHg) *(r = 0.47, P < 0.001)*	*Positive*	*Older individuals also exhibit higher diastolic blood pressure.*
	- Right Diameter (cm) *(r = 0.589, P < 0.001)*	*Positive*	*Larger right diameters are associated with older age.*
	- Left Diameter (cm) *(r = 0.524, P = 0.001)*	*Positive*	*Older age is significantly associated with larger left diameters.*
P-Axis (°)	***- DBP (mmHg)*** *(r = 0.37, P = 0.028)*	*Positive*	*Diastolic blood pressure rises with changes in P-axis values.*
QRS-Axis (°)	***- QRS Interval (ms)*** *(r = -0.62, P < 0.001)*	*Negative*	*Longer QRS durations align with lower QRS-axis values.*
	- T-Interval (ms) *(r = -0.55, P = 0.001)*	*Negative*	*Prolonged T-interval correlates with lower QRS-axis values.*
T-Axis (°)	***- QRS Interval (ms)*** *(r = 0.41, P = 0.014)*	*Positive*	*Higher T-axis values associate with longer QRS intervals.*
PR-Interval (ms)	***- QRS Interval (ms)*** *(r = 0.52, P = 0.001)*	*Positive*	*Prolonged PR intervals align with longer QRS intervals.*
BMI (kg/m²)	***- DBP (mmHg)*** *(r = 0.323, P = 0.021)*	*Positive*	*Higher BMI is linked to elevated diastolic blood pressure.*
	- SBP (mmHg) *(r = 0.323, P = 0.021)*	*Positive*	*Increased BMI correlates with elevated systolic blood pressure.*
Right carotid Diameter (cm)	***- CIMT RT (mm)*** *(r = 0.409, P = 0.022)*	*Positive*	*Increased carotid intima-media thickness correlates with larger right diameters.*
	- Left Diameter (cm) *(r = 0.543, P = 0.002)*	*Positive*	*Strong positive correlation between right and left diameters.*
LT carotid diameter (cm)	***- BMI*** *(r = -0.355, P = 0.043)*	*Negative*	*Higher BMIs show an inverse relationship with transverse diameter (LT DIAM0).*
Lt carotid Diameter (cm)	***- Right Diameter (cm)*** *(r = 0.543, P = 0.002)*	*Positive*	*Larger left diameters align with larger right diameters.*
SBP (mmHg)	***- Age*** *(r = 0.343, P = 0.011)*	*Positive*	*Older age is significantly associated with higher systolic blood pressure.*
LAD (cm)	***- SBP (mmHg)*** *(r = 0.337, P = 0.021)*	*Positive*	*Left atrial diameter increases with elevated systolic blood pressure.*
	- Age *(r = 0.295, P = 0.037)*	*Positive*	*Larger left atrial diameters are linked to older age.*

Variable	*Significant Correlations*	*Direction*	*Remarks*
EF (%)	***- FS*** *(r = 0.922, P < 0.001)*	*Positive*	*Strong positive association between ejection fraction and fractional shortening.*
FS (%)	***- EF*** *(r = 0.922, P < 0.001)*	*Positive*	*Fractional shortening correlates positively with ejection fraction.*

III. Discussion:

Rumuekini Community is a suburban community in Rivers State, very few people responded because of the social stigmata associated with alcohol and the fear of knowing the complication, women did not avail themselves of the opportunity. Women are known to have better health seeking behaviours in Nigeria from local studies, but the social stigmata associated with alcohol ingestion affected the turn-out the married population accounted for 24.07%.

The observation that married individuals generally exhibit higher parameter values can be partly explained by age, as they are typically older and age is positively correlated with many health and physiological factors, such as blood pressure, cholesterol levels, and cardiovascular dimensions like left ventricular diameter and wall thickness. Additionally, lifestyle and social structures associated with marriage, such as healthier diets, increased healthcare utilization, and social support, may contribute to these elevated values. The protective or buffering effects of marriage on stress could also impact health positively, while cultural expectations may encourage healthier habits. However, it's crucial to acknowledge that not all married individuals experience these benefits uniformly, as marital stress or discord could negate these advantages.

Focusing on the updated table for hypertension, notable differences emerge between hypertensive, normotensive, and prehypertensive individuals. For example, hypertensive individuals show higher values for parameters such as left ventricular internal diameter in diastole (LVIDD) and systolic function markers like ejection fraction (EF), reflecting structural and functional changes associated with high blood pressure. Prehypertension demonstrates intermediate values, suggesting progressive changes compared to normotensives. Normotensive individuals generally exhibit lower values, highlighting healthier cardiovascular profiles. These distinctions underscore the escalating impact of blood pressure on cardiovascular health across different hypertension stages.

Similarly, smokers displayed distinct patterns compared to non-smokers in various parameters. Non-smokers often show better outcomes for cardiovascular markers like cholesterol levels, intima-media thickness, and diameters of key cardiac structures, indicating less exposure to the harmful effects of smoking. Meanwhile, smokers tend to exhibit higher T-axis and PR-interval values, potentially reflecting electrophysiological changes induced by smoking. These findings emphasize the dual influence of hypertension and smoking on cardiovascular health, showcasing specific trends that could guide interventions to mitigate risk in these populations

Smoking has significant detrimental effects on the cardiovascular system, contributing to conditions such as atherosclerosis, elevated blood pressure, reduced oxygen supply, increased blood clot formation, and damage to blood vessel walls. Nicotine raises blood pressure and heart rate, while carbon monoxide in smoke reduces the oxygen-carrying capacity of blood, both of which strain the heart. Smoking also promotes clotting, reduces "good" HDL cholesterol, and damages arterial linings, increasing the risk of coronary artery disease and peripheral artery disease. These effects align closely with the data from the study above, where smokers exhibited higher carotid intima-media thickness (CIMT), larger left atrial diameter, and a depressed left ventricular ejection fraction (EF). These findings reflect structural and functional cardiovascular damage associated with smoking. In contrast, the lower alcohol intake among hypertensives and non-smokers might indicate an attempt at healthier lifestyle changes. The elevated triglyceride and LDL cholesterol levels in hypertensives are consistent with the well-documented links between hypertension, lipid abnormalities, and cardiovascular risks. This study underscores the compounded cardiovascular impact of smoking, alcohol consumption, and hypertension, highlighting the importance of lifestyle modifications.

The correlation analysis using Pearson Correlation Coefficient did not show any correlation of quantity of alcohol consumed with any cardiovascular parameters , however it revealed significant relationships between age and several cardiovascular parameters. Older individuals showed higher right and left arterial diameters, indicating potential arterial remodeling with aging. Additionally, age positively correlated with total cholesterol and triglyceride levels, highlighting the increased risk of metabolic changes and lipid accumulation in older populations. Age was also strongly associated with elevated systolic and diastolic blood pressure, reinforcing its role as a key risk factor for cardiovascular alterations.

Body Mass Index (BMI) emerged as another important factor, positively correlating with both systolic and diastolic blood pressure. This finding underscores the influence of excess body weight on cardiovascular strain, particularly on arterial pressure. Furthermore, BMI showed an inverse relationship with transverse left carotid diameter, indicating potential structural alterations associated with higher body weight. These results emphasize the critical role of weight management in mitigating cardiovascular risks.

The interaction between various cardiac structures was also notable. A strong positive correlation was observed between ejection fraction (EF) and fractional shortening (FS), highlighting their interdependence as measures of cardiac function. Additionally, right and left diameters showed a positive association, suggesting concurrent structural changes in cardiac chambers. These findings collectively highlight how cardiovascular parameters interrelate, providing insights into potential mechanisms behind cardiovascular remodeling and functional changes.

In conclusion, alcohol consumption exhibits a dual and dose-dependent influence on cardiovascular health, with moderate intake offering potential cardioprotective benefits, such as improved lipid profiles, while chronic and excessive consumption leads to hypertension, arrhythmias, atherosclerosis, and structural changes like increased arterial stiffness. These adverse effects are amplified in the presence of hypertension and smoking, which together accelerate cardiovascular damage through mechanisms such as oxidative stress, vascular inflammation, and electrophysiological disruptions. Smokers exhibit higher T-axis and PR-interval values, while hypertensive individuals display structural cardiac remodeling and progressive deterioration, emphasizing the compounded risks.

Interestingly, the study highlights the added nuance of marital status in the interplay of alcohol consumption and cardiovascular health. Married individuals, while consuming more alcohol on average, appear to be partly protected from adverse outcomes through other mitigating factors. These include enhanced social support systems, healthier lifestyle choices, greater access to healthcare, and the psychosocial benefits of reduced stress in stable relationships. This dynamic underscores the complex and multifactorial nature of alcohol's impact, where demographic and lifestyle factors significantly influence outcomes.

Overall, this investigation reinforces the importance of targeted public health strategies aimed at modifiable risks. Interventions should encourage moderation in alcohol consumption, address the risks associated with smoking and hypertension, and consider the protective social and lifestyle factors associated with marriage. Future research should explore these interactions further to inform policies and interventions that promote cardiovascular health across diverse populations.

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