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# Pattern of arrhythmias among Nigerians with congestive heart failure



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**Background:** In patients with heart failure, death is often sudden due to life-threatening arrhythmias. This work was carried out to evaluate the pattern of arrhythmias in Nigerians with heart failure.

**Materials and methods:** Thirty subjects with congestive heart failure (CHF), 30 subjects with hypertensive heart disease, and 15 normal subjects with no obvious features of heart disease were evaluated with resting and 24-hour electrocardiographic monitoring and transthoracic echocardiography. Data were analyzed with one-way analysis of variance with post hoc Duncan's analysis, Fisher's exact test, and linear regression analysis using SPSS version 16.

**Results:** CHF subjects had more instances of supraventricular tachycardia ($P$=0.005), ventricular extrasystoles ($P$<0.001), bigeminy ($P$<0.001), trigeminy ($P$<0.001), couplets ($P$<0.001), triplets ($P$<0.001), and nonsustained ventricular tachycardia (VT) ($P$=0.003) than the other two control groups. They also showed a significantly longer VT duration (4.6±5.6 seconds) compared with the other groups ($P$<0.001). Linear regression analysis showed a significant direct relationship between VT and the maximum number of ventricular extrasystoles per hour ($P$=0.001).

**Conclusion:** Cardiac arrhythmias are common in subjects with CHF and are more frequent when compared with patients with hypertensive heart disease and normal subjects.

**Keywords:** arrhythmias, heart failure, hypertensive heart disease, Nigerians

## Introduction

Heart failure (HF) is a cause of considerable morbidity and mortality worldwide. The prevalence continues to rise because of increasing cardiovascular risks and increasing age.[1] The prevalence of HF is expected to increase exponentially in sub-Saharan Africa because of the adoption of western lifestyles and other consequences of urbanization.[2,3] The causes of HF in sub-Saharan Africa and Nigeria are largely hypertension, cardiomyopathy, and rheumatic valvular heart disease.[2,4–7] Despite considerable advances in the management of this condition, prognosis still remains grave in patients with HF. Poor prognostic indices include black race, structural heart disease, high New York Heart Association class (III and IV), electrolyte abnormalities, systolic/diastolic dysfunction, and arrhythmias, among many others.[8] The presence of arrhythmias portends a poorer prognosis because they are independent risk factors for the progression of HF and sudden cardiac death.[8] Basic tools to identify these arrhythmias include resting electrocardiogram (ECG) with impreciseness, and 24-hour Holter ECG monitoring for precise diagnosis. Studies using Holter monitoring for the precise diagnosis of arrhythmias have been carried out in Nigeria, but none to the best of our knowledge have studied its importance in patients with HF[9–11] in comparison with hypertensive heart disease

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(HHD) and apparently healthy individuals. It is the aim of this study to ascertain, with the aid of Holter monitoring, how frequent arrhythmias are, their pattern, and the factors that predict their occurrence in Nigerian HF patients.

## Materials and methods

Subjects and age-matched controls were recruited after informed consent and ethical clearance were obtained. In all, 75 individuals were evaluated using history taking, physical examination, resting and 24-hour electrocardiographic monitoring, and transthoracic echocardiography. Thirty subjects with congestive HF (CHF) secondary to hypertension and dilated cardiomyopathy, 30 subjects with HHD, and 15 normal subjects with no features of heart disease were recruited serially and evaluated.

HHD was the cause of CHF in 21 subjects while in nine subjects, dilated cardiomyopathy was the cause of their CHF. According to the New York Heart Association classification, ten subjects were stage II, 12 subjects were stage III, and eight subjects were stage IV. Also, 18 subjects had low ejection fraction (EF) CHF while EF was normal in 12 subjects. Normal controls were recruited serially among those referred for possible cardiac complaints but without heart disease.

Hypertension was diagnosed according to the eighth report of the Joint National Committee on the detection, evaluation, and treatment of high blood pressure.[12] Subjects with HHD were those with structural cardiac damage resulting from hypertension with no features of CHF,[13] while HF was diagnosed using Framingham's criteria[14,15] and confirmed by cardiac imaging (chest X-ray and echocardiography). Subjects with CHF were receiving frusemide, angiotensin-converting enzyme inhibitors (mainly lisinopril), spironolactone, an anticoagulant (warfarin), and digoxin as indicated.[16] None of the HF subjects were receiving a beta blocker, amiodarone, or any other antiarrhythmic drug. The hypertensive subjects were mainly receiving angiotensin receptor inhibitors/blockers with thiazide diuretics or a calcium channel blocker (dihydropyridine) with thiazide diuretics. None of the subjects were using any beta blocker in combination with their antihypertensives.

Echocardiography was performed using a Philips HP sonos 4500 ultrasound machine. M-mode, two-dimensional and pulsed, continuous wave and color flow Doppler modalities were performed in accordance with the American Society of Echocardiography recommendations.[17–19] Cardiac dimensions and left ventricular mass and mass index were calculated using the Devereux cube formula and corrected for body surface area.[20]

Diastolic functions were measured with pulsed wave Doppler, with the sample volume at the tip of the mitral valve leaflets in the apical four-chamber view. The early and peak atrial velocities and their ratio (E/A), as well as isovolumic relaxation time were measured.[21,22]

Ambulatory ECG (AECG) was recorded using a three-channel Schiller MT-103, in compliance with the American College of Cardiology/American Heart Association guidelines for AECG.[23] The subjects were encouraged to undertake their usual daily activities, but to avoid bathing or bodily contact of water with the electrodes. The 24-hour AECG data were analyzed by Schiller AECG software (serial no-300.04177 and 300.04178) and analysis was confirmed by two physicians. The analysis included the following:

1. Supraventricular extrasystoles (SVES) and supraventricular tachycardia (SVT).
2. Ventricular extrasystoles (VES), including the maximum number of VES in 1 hour (MAXVESPERHR), ventricular couplets, ventricular triplets, ventricular bigeminy or trigeminy, ventricular tachycardia (VT), and R-on-T phenomenon.
3. Sinus tachycardia, sinus bradycardia.
4. Average heart rate.

Ethical approval was obtained from the Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Osun State, Nigeria.

### Statistical analysis

Data analysis was performed by SPSS software (version 16; IBM Corporation, Armonk, NY, USA). Data are expressed as the mean ± standard deviation, or as numbers and proportions. One-way analysis of variance with post hoc Duncan's tests were used to test for the mean difference between normal controls, HHD subjects, and CHF subjects. Chi-square and Fisher's exact tests were used for tests of association. Linear regression analysis was used to determine the predictors of VT (as a quantitative variable). A $P$-value $<0.05$ was taken as statistically significant.

## Results

The clinical and ECG characteristics of subjects are shown in Table 1. Seventy-five subjects comprising 30 CHF patients, 30 HHD patients, and 15 subjects with no heart disease were evaluated. In total, 53 (70.7%) were males and 22 (29.3%) were females, with no significant sex difference in the three groups. Their ages ranged from 21–82 years with similar mean age of 54.4±11.9 years in CHF patients, 55.3±9.5 years in HHD patients and 50.3±15.8 years in normal

**Table 1** Clinical and echocardiographic characteristics of the subjects

| Variable | CHF (n=30) | HHD (n=30) | Normal (n=15) | *P*-value |
|---|---|---|---|---|
| Age (years) | 54.4±11.9 | 55.3±9.5 | 50.3±15.8 | 0.268 |
| Sex | 19 males, 11 females | 22 males, 8 females | 12 males, 3 females | 0.513[c] |
| BMI (kg/m$^2$) | 23.8±5.9[a] | 29.99±2.9[b] | 28.06±3.8[b] | 0.001* |
| LVMI (g/m$^2$) | 182.6±78.7 | 132.5±50.3 | 145.3±137.8 | 0.160 |
| DD (mm) | 56.2±14.4 | 51.7±8.9 | 46.6±6.6 | 0.132 |
| SD (mm) | 47.5±15.0[a] | 31.5±6.1[b] | 33.7±3.7[b] | <0.001* |
| EF (%) | 35.5±19.0[a] | 61.9±12.2[b] | 55.5±8.9[b] | <0.001* |
| FS (%) | 16.72±10.7[a] | 34.43±9.1[b] | 27.19±7.9[b] | <0.001* |
| RVD (mm) | 26.7±10.6 | 21.5±5.9 | 27.3±7.4 | 0.143 |
| LAD (mm) | 47.8±6.0[a] | 37.4±6.1[b] | 37.1±2.6[b] | <0.001* |
| AO (mm) | 30.2±3.5 | 29.5±2.9 | 28.8±3.6 | 0.632 |

**Notes:** Values are expressed as the mean ± standard deviation, or as proportions. *$P<0.05$; [a,b]using Duncan's post hoc test, the figures marked with[a] are significantly different from the figures marked with[b]; [c]Fisher's exact test.

**Abbreviations:** CHF, congestive heart failure; n, number; HHD, hypertensive heart disease; BMI, body mass index; LVMI, left ventricular mass index; DD, left ventricular internal dimension in diastole; SD, left ventricular internal dimension in systole; EF, ejection fraction; FS, fractional shortening; RVD, right ventricular dimension; LAD, left atrial diameter; AO, aortic root diameter.

subjects ($P$=0.268). Subjects with CHF had a significantly lower body mass index (BMI) of 23.8±5.9 kg/m$^2$ compared with 29.9±2.9 kg/m$^2$ in HHD subjects and 28.1±3.8 kg/m$^2$ in normal subjects ($P$=0.001). CHF subjects also showed a significantly lower EF of 35.5%±19.0% compared with 61.9%±12.2% in the HHD subjects and 55.5%±8.9% in the normal subjects ($P<0.001$). The left atrial diameter in CHF subjects was significantly larger at 47.8±6.0 mm than in the HHD and normal subjects with a left atrial diameter of 37.4±6.1 and 37.1±2.6, respectively ($P<0.001$). No difference was found in terms of left ventricular mass index, left ventricular internal dimension in diastole, and right ventricular diameter in the three groups.

The Holter parameters of subjects are shown in Table 2. The average heart rate of 89.4±27.1 bpm was significantly higher among the CHF subjects when compared to the other groups ($P$=0.012). All CHF subjects (100%) had VES ($P<0.001$), with a significantly higher number of VES per hour ($P$=0.011). Also, CHF subjects showed significantly higher amounts of advanced grades of VES in the form of bigeminy ($P<0.001$), trigeminy ($P<0.001$), couplets ($P<0.001$), and triplets ($P<0.001$). There was a significantly higher amount of nonsustained VT in CHF subjects (number [n] =13; 43.3%) when compared with three (10%) in the HHD subjects, and only one (6.7%) in normal subjects ($P$=0.003). CHF subjects also showed a significantly longer VT duration (4.6±5.6 seconds) compared with the other groups ($P<0.001$), but no difference was seen in the VT rate between CHF and HHD subjects. No difference was seen in the number of SVES in the three groups, but CHF subjects showed a higher

**Table 2** Holter parameters of subjects

| Variable | CHF (n=30) | HHD (n=30) | Normal (n=15) | *P*-value |
|---|---|---|---|---|
| RT (hours) | 23.4±1.5 | 23.6±1.1 | 23.4±2.3 | 0.929 |
| Total beats | 167,000.0±2,301.9 | 104,000.0±218.2 | 183,000.0±3,001.0 | 0.210 |
| AVHR (bpm) | 89.4±27.1[a] | 73.13±19.6[b] | 75.40±8.8[b] | 0.012* |
| MAXHR (bpm) | 198.7±76.7[a] | 139.9±42.4[b] | 124.5±20.2[b] | <0.001* |
| MINSHR (bpm) | 56.17±18.2 | 48.5±7.7 | 50.0±9.6 | 0.075 |
| MAXSHR (bpm) | 138.63±52.6 | 122.80±30.3 | 112.60±31.6 | 0.108 |
| VES | 30 (100%)[a] | 29 (46.7%)[b] | 14 (30%)[b] | <0.001*[,d] |
| MAXVESPERHR | 583.9±958.8[a] | 170.1±406.5[b] | 6.0±5.2[b] | 0.011* |
| Bigeminy | 24 (80%)[a] | 5 (16.7%)[b] | 0 (0%)[b] | <0.001*[,d] |
| Trigeminy | 22 (73.3%)[a] | 8 (26.7%)[b] | 0 (0%)[c] | <0.001*[,d] |
| Couplet | 28 (93.3%)[a] | 14 (46.7%)[b] | 4 (13.3%)[b] | <0.001*[,d] |
| Triplet | 23 (76.7%)[a] | 6 (20%)[b] | 1 (6.7%)[b] | <0.001*[,d] |
| VT | 13 (43.3%)[a] | 3 (10%)[b] | 1 (6.7%)[b] | 0.003*[,d] |
| VTD (seconds) | 4.6±5.6[a] | 0.3±0.8[b] | 0.1±0.5[b] | <0.001* |
| VTR (bpm) | 202.2±37.8[a] | 207.00±7.1[a] | 140±0.0[b] | 0.005* |
| SVES | 30 (100%) | 30 (100%) | 14 (93.33%) | 0.200[d] |
| MAXSVESPERHR | 344.3±475.9 | 177.6±580.7 | 18.9±57.3 | 0.093 |
| SVT | 16 (53.3%)[a] | 8 (26.7%)[b] | 1 (6.7%)[b] | 0.005*[,d] |

**Notes:** Values are expressed as the mean ± standard deviation or as proportions. *$P<0.05$; [a–c]using Duncan's post hoc test; [d]Fisher's exact test, the figures marked with[a] are significantly different from the figures marked with[b], and the figures marked with[b] are significantly different from the figures marked with[c].

**Abbreviations:** bpm, beats per minute; CHF, congestive heart failure; n, number; HHD, hypertensive heart disease; RT, recording time; AVHR, average heart rate; MAXHR, maximum heart rate; MINSHR, minimum sinus heart rate; MAXSHR, maximum sinus heart rate; VES, ventricular extrasystoles; MAXVESPERHR, maximum number of ventricular extrasystoles per hour; VT, nonsustained ventricular tachycardia; VTD, duration of ventricular tachycardia; VTR, heart rate of ventricular tachycardia; SVES, supraventricular extrasystoles; MAXSVESPERHR, maximum number of supraventricular extrasystoles per hour; SVT, supraventricular tachycardia.

number of SVT (n=16; 53.3%) compared with HHD and normal subjects ($P$=0.005). The linear regression analysis of the predictors of VT is shown in Table 3. While age ($P$=0.660) and septal wall thickness ($P$=0.440) showed a nonsignificant inverse relationship with VT, BMI ($P$=0.732), EF ($P$=0.575), left ventricular posterior wall thickness ($P$=0.257), and left ventricular internal dimension in diastole ($P$=0.112) showed a nonsignificant and direct relationship with VT. However, the maximum number of VE per hour (MAXVESPERHR) showed a significant direct relationship with VT ($P$=0.001) in that for every MAXVESPERHR in these CHF subjects, the risk of developing VT increases by 0.023.

## Discussion

This study showed that common arrhythmias in HF subjects included SVES, SVT, VES (including its different forms), and VT. It also showed that these cardiac arrhythmias are more commonly found in HF subjects when compared with HHD and normal subjects. This is similar to findings of the greater risk of ventricular arrhythmias in HF subjects when compared with normal controls by Lasisi et al.[24] This has been attributed to a multiplicity of mechanisms, the exact nature of which is important to unravel in order to be able to better risk stratify these patients to avoid sudden death.[25] These mechanisms include early autonomic and repolarization abnormalities, afterdepolarizations, re-entry, and enhanced automaticity.[25,26] In addition to these, other factors that play modifying roles to reduce the threshold for arrhythmogenesis in HF include electrolyte abnormalities, scar and fibrosis formation, valvular disease, complications of medical management (eg, digoxin toxicity and abnormal electrophysiologic predispositions [QT abnormalities and impaired vagal tone]).[17,26,27] Higher grades of VES (couplets, triplets, bigeminy, and trigeminy) were found to be significantly higher in our HF subjects compared to the HHD subjects and normal controls. They are important because they are considered to predate the onset of ventricular fibrillation (VF) in those with structural heart disease.[28]

Forty-three percent of CHF subjects in this study had nonsustained VTs, while 10% of those with HHD also showed nonsustained VTs. This contrasts with a very low number of nonsustained VTs (0.9%) in subjects in the study by Omotoso et al.[29] In their study, they examined the resting ECGs of 2,017 subjects and AECG was not done. This difference shows that AECG is better than resting ECG in the evaluation of arrhythmias in HF and HHD subjects because it is more sensitive and can be used to evaluate the rate, rhythm, and variability of asymptomatic and symptomatic events.[30]

This study also showed higher rates per hour and second of VES and nonsustained VT, respectively, in HF subjects. The roles of the frequency and rate of VES and VT in predicting sudden death have been established. It has been postulated that the increased duration and rate of VT or VES may hasten degeneration to sustained VT and VF, and then to sudden death.[31–33] Mechanisms such as ischemia, adrenergic stimulation, and increase in blood pressure, particularly in those with extrasystoles, mechanoelectrical dissociation, and abnormal loading have been identified in the literature as predictors of nonsustained VT.[34] These factors do so by acting as the drivers of hypertrophy or dilation of the heart.[34] In this study, positive correlations between surrogates of hypertrophy and dilation were seen, though they did not reach statistical significance. This may be due to the relatively small sample size of this study. This study, however, showed that the frequency of VES per hour is a predictor of VT. This is in keeping with studies that have reported a link between this frequency and sudden death, the cause of which is usually sustained VT and VF.[31–33,35] Also, the presence of ten or more VES per hour has been shown to identify patients at an increased risk for VT or sudden death.[36]

**Table 3** Linear regression analysis of predictors of ventricular tachycardia

| Variable | Exp (B) | *P*-value | CI |
|---|---|---|---|
| Age (years) | –0.19 | 0.660 | –1.29 to 0.89 |
| BMI (kg/m$^2$) | 0.28 | 0.732 | –1.75 to 2.32 |
| SWT (mm) | –1.37 | 0.440 | –5.59 to 2.84 |
| PWT (mm) | 2.10 | 0.257 | –2.13 to 6.33 |
| DD (mm) | 0.62 | 0.112 | –0.21 to 1.44 |
| EF (%) | 0.19 | 0.575 | –0.65 to 1.04 |
| MAXVESPERHR | 0.02 | 0.001* | 0.01–0.03 |

**Note:** *$P$<0.05.

**Abbreviations:** Exp (B), odds ratio; CI, confidence interval; BMI, body mass index; SWT, septal wall thickness; PWT, Left ventricular posterior wall thickness; DD, left ventricular internal dimension in diastole; EF, ejection fraction; MAXVESPERHR, maximum number of ventricular extrasystoles per hour.

## Conclusion

Cardiac arrhythmias are common in subjects with CHF and are more frequent when compared with HHD and normal subjects. These arrhythmias include nonsustained VT, which can degenerate into life-threatening forms – namely, sustained VT and VF – causing sudden death. Therefore, routine use of AECG to risk stratify and aid in management is essential in all CHF subjects. In this study, HF subjects on antiarrhythmic drugs were excluded. A further study to assess the impact of these drugs (beta blockers and amiodarone) on morbidity and mortality is being carried out.

## Limitations

The results of this study may be biased by the relatively small sample size.

## Disclosure

The authors report no conflicts of interest in this work.

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