Blood Pressure, Heart Rate, Cardiovascular Reflexes and Electrocardiographic changes in some Hypertensive Nigerians

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Summary: The effects of hypertension on resting and reflex cardiovascular function were investigated in this study. Blood pressure, heart rate and electrocardiogram were recorded in male and female control subjects and hypertensive Nigerian patients. Blood pressure was measured, using the sphygmomanometer/auscultatory method. Heart rate was determined from palpating the radial pulse or from the resting electrocardiograph. The systolic and diastolic blood pressures were high in the hypertensive patient (160.90 ± 2.06 mmHg and 110.8 ± 1.95 mmHg respectively compared with control subjects (119.3 ± 2.05 and 73.58 ± 1.09mmHg; P<0.01). Pulse pressure and mean arterial pressures were also higher in the hypertensive patients. Heart rate was higher in the hypertensive compared to the control groups (86.93 ± 2.83 cf 71 ± 1.35 beats per minute, P<0.01). ECG analysis showed that the intervals were lower in the controls than in the hypertensive group except for PR intervals (0.21 ± 0.01 cf 0.23 ± 0.01 sec). The amplitude of the waves was also lower in the control group than the hypertensive group. Cardiovascular response to exercise assessed from the post-exercise recovery graph showed that the aggregate recovery (6min after) was lower in the hypertensive subjects (22% cf 28%, p<0.05) than in controls. This suggests that the baroreflex sensitivity was higher in the control than in the hypertensive subjects. Results from this study suggest that in hypertension there may be increased heart rate, altered electrocardiograph readings indicating ventricular hypertrophy and delay in ventricular conduction. In hypertension baroreflex sensitivity may be reduced.

Keywords: Blood pressure, Heart Rate, ECG, Cardiovascular reflexes, Hypertension, Nigerian

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INTRODUCTION

Hypertension or high blood pressure is known to be widespread in Nigeria and among Africans. It is a major public health problem as it leads to various complications when not treated or managed appropriately (Reddy and Yusuf 1998, Salako et al 2002). Many reports on the effects of hypertension on various organs of the body have been published (Murray and Lopez 1997; Khalil 2005; Mannino et al 2008).

However, only very few studies have been carried out on the state of the cardiovascular reflex mechanisms among Black Africans. Many reports have shown that various disease states may affect or alter cardiovascular function (Al-Mahroos et al 2000). However very few reports are available on the effects of hypertension on the cardiovascular response to exercise in Nigerian subjects. In this environment there are very few reports on other aspects of cardiovascular function in uncomplicated hypertension as most reports are based on observations in complicated cases often involving multiple organ damage (Murray and Lopez 1997). Therefore this study investigated the resting and reflex cardiovascular function in some hypertensive Nigerians. The blood pressure, heart rate, electrocardiogram and the cardiovascular response to exercise were evaluated.

MATERIALS AND METHODS

Adult male and female volunteers took part in the study. They included healthy adult volunteers (n=39) who served as controls and patients (n=39) attending the hypertension clinic at the cardiology unit Lagos University Teaching Hospital, Idi-Araba, Nigeria.

In this study subjects were divided into two groups. Those with blood pressure above 140/90 mmHg on two different readings were classified as hypertensive (WHO 1999). However patients with any other disease condition were excluded from the study. The informed consent of the subjects was obtained and subjects responded to a standard...
questionnaire. Exclusion criteria were smoking, significant alcohol use and medications affecting cardiovascular function. Ethical and institutional approvals were obtained.

Seated blood pressure was measured using sphygmomanometer/Auscultatory methods. Systolic and diastolic blood pressures were taken at Korotkoff phase I and phase V respectively. Heart rate was determined from the radial pulse or from the ECG. Resting 12 lead Electrocardiogram (ECG) was recorded in the standard method (Araoye, 1996). The machine was calibrated to read 10mm deflection to 1 milivolt.

The cardiovascular response to exercise was assessed by the step-stool test. The subject stepped up and down the exercise stool 20 times a minute for 3 minutes. The heart rate and blood pressure were taken before exercise and immediately after the exercise. Thereafter parameters were obtained at one-minute intervals until the values returned to normal. The slope of the linearized relationship between blood pressure, heart rate and time was used as index of cardiovascular response to exercise.

**Statistical Analysis and Data Presentation:** Results are presented as mean ± standard error of the mean (S.E.M.) Paired t-test was used for comparison within groups while the un-paired t-test was used for comparisons between groups. Significance was taken as p<0.05.

**RESULTS**

**Resting Blood Pressure in Control Subjects**

Resting systolic blood pressure (SBP) was 119.33 ± 2.05 mmHg and 110.8 ± 1.95 mmHg in males and females respectively, while the diastolic blood pressure (DBP) was 73.58 ± 1.09 mmHg and 74.4 ± 1.33 mmHg, in males and females respectively (NS). The pulse pressures (PP) and heart rate (HR) were 45.75 ± 2.32, 36.4 ± 2.19 mmHg and 71 ± 1.35 and 70.13 ± 4.077 beats per minutes (p<0.05) respectively. Figs. 1 and 2.

**Resting blood pressure in hypertensive patients**

As a group (HTW) the SBP and DBP in males and females were 160.96 ± 2.60, 152.93 ± 3.40 and 100.08 ± 1.39 and 97.3 ± 2.10 mmHg respectively (Fig.3). The pulse pressure and mean arterial pressures was 60.67 ± 1.50 and 120.31 ± 1.72 mmHg; and 55.2 ± 1.74 and 116.13 ± 2.48 mmHg in males and females respectively. In male hypertensive subjects the systolic, diastolic, pulse and mean arterial blood pressures were slightly higher than in females (p<0.05). These values are generally higher than those obtained in corresponding controls (p<0.05 - p<0.001). Fig. 4.

Heart rate (HR) was 81.67 ± 2.21 and 86.93 ± 2.86 beats/min. HR was slightly lower in males than in females Heart rate was higher in both male and female hypertensive than in the control subjects (p<0.05 - p<0.01). Fig.2.

**Fig 1:** Resting blood pressure in male and female control subjects. Male subjects appear to have a slightly higher systolic pressure. Values are means ± SEM. n = 37. p < 0.05

**Fig 2:** Bar chart showing heart rate obtained from control and high blood pressure subjects. MC = male control, FC = female control, MHT = male hypertensive, FHT = female hypertensive

**Fig 3:** Resting blood pressure in hypertensive subjects. Values are means ± SEM. Legends as in fig. 2. above.
Resting Electrocardiogram in control subjects
Analysis of the ECG showed that PR intervals were slightly higher in males (0.19±0.01sec) than in females (0.18±0.01sec). The QT, ST and RR intervals were similar (NS). However wave amplitude was slightly higher in males in many leads than in female subjects. (0.87±0.11mV vs 0.58±0.08mV, for QRS complexes (lead III) respectively, p<0.01).

Electrocardiogram in hypertensive patients.
Analysis of ECG showed that PR interval, QRS duration, QT-interval was slightly higher in male hypertensive than in females. RR interval was longer in the males. Amplitude of the QRS and T waves were similar in both groups but with minor variations. Comparison with control group showed that PR interval was longer in control than in the hypertensive. (0.21 ± 0.01 cf 0.23 ± 0.01sec). QRS Duration was shorter in the controls (0.05 ± 0.00 secs cf 0.19 ± 0.00 sec) than hypertensive (Fig.5). QT interval was shorter in the control than in the hypertensive group. RR interval was longer in the control group (p<0.05) while P wave amplitude was lower than in the hypertensive. Amplitude of QRS complex and T wave was lower in the control than in the hypertensive (Fig. 6).

Cardiovascular response to exercise in control subjects and hypertensive patients
In step “up and down” exercise immediate post-exercise (PE) SBP was 159.08 ± 4.32mmHg while DBP fell to 59.75 ±1.48 mmHg and HR was 90.83 ± 4.28 beats per minute. In the hypertensive subjects, PE SBP was 208.58 ± 2.03 while DBP was 122.58 ± 1.98 mmHg and HR 133.67 ± 2.97 beats minute. The increase in SBP was significant (208.6 ± 2.03 cf 160.96 ± 2.60; p<0.001) for DBP (122.58 ± 1.98 cf 100.08±1.39 mmHg; p<0.001). In the control group exercise reduced DBP (59.75 ± 1.48 cf 73.58 ± 1.09 mmHg; p<0.05. In hypertensive subjects’ heart rate increased from 81.6 ± 2.21 beats per minute to 113.67 ± 2.97 beats/min, p<0.05. Return to resting levels was more delayed in the hypertensive subjects than in the normotensive control subjects.

DISCUSSION
This study was designed to investigate the effect of high blood pressure on the electrocardiogram and also to investigate the cardiovascular response to exercise in hypertensive subjects. The hypertensive subjects had elevated systolic and diastolic arterial blood pressures similar to, previous reports (WHO 1999; Pickering, 1996; Staesson et al, 1996; Al-Mahroos et al, 2000). Heart rate was higher in hypertensive subjects than in normotensive controls. This suggests that the setpoint for the chronotropic control of the heart rate may have been reset in the
hypertensive subjects. Previous reports have shown that though hypertension results in an increase in blood pressure it does not cause an increase in heart rate (Tripathi 1994). The observed increase in heart rate cannot be easily explained but it may be due to increased sympathetic activity or reduced baroreflex sensitivity, which contributes to the positive feedback that contributes to the hypertension.

Pulse pressure (PP) was higher in the hypertensive subjects than in the control subjects. The pulse pressures were lower in female subjects than their male counterparts. This suggests that cardiac work is more in the hypertensive persons.

The electrocardiogram (ECG) of the hypertensive of the hypertensive subjects appeared normal except for the slight increase in QRS duration. The increase in QRS duration could be an indication of Bundle branch block or left ventricular hypertrophy (Ganong 2005, Guyton and Hall 2006). ST interval was normal but QT interval was prolonged. The amplitude of the P wave, QRS complex and T waves were higher in the hypertensive subjects suggesting chamber enlargement. In general the wave amplitudes were slightly but not significantly higher in males than females. This is in consonance with previous reports by Araoye et al (1996).

PR interval was shorter in hypertensive individuals. The PR interval is an index of electrical conductivity of the atroventricular (AV) node. The reduced PR interval may be indicative of an increase in conductivity of the atrial muscle and internodal pathways, thus reducing the interval from the onset of atrial activation to the onset of ventricular activation. This may explain the higher heart rates in the hypertensive subjects despite the increased duration of the QRS complex. Adrenaline and Noradrenaline are known to speed up electrical activity of cardiac myocytes. P wave amplitude was lower than in the hypertensive group. The increase in amplitude of the ECG in hypertensive subjects may be associated with increased myocardial tension or enlargement or increased electrical activity. The duration of the QRS complex was increased thus suggesting that conduction through the Bundle of His and Purkinje fibres may be affected. The cardiovascular response to exercise was different in male hypertensive subjects. In the post exercise recovery period, while systolic blood pressure fell in the two groups, diastolic pressure demonstrated a rise towards normal in the control group. There was a progressive drop in the elevated diastolic pressure in the hypertensive subjects. This increase in diastolic pressure in hypertension may be due to the fact that peripheral resistance did not decrease during exercise (Reisin, 1997). The cardiovascular response to exercise is dependent on the integration of neural and local (metabolites) factors (Bevegaard and Shepherd, 1992, Berne and Levy, 1996; Goswami et al 2009). These investigations further demonstrate that electrocardiogram was altered in hypertension, with increases in amplitude and duration of QRS complex in these Nigerian subjects. There was also alteration of cardiovascular response to exercise.

REFERENCES


