ELECTROCARDIOGRAPHIC (ECG) CHANGES IN SYSTEMIC HYPERTENSION IN PATIENTS ATTENDING KENYATTA NATIONAL HOSPITAL, NAIROBI, KENYA.

BY

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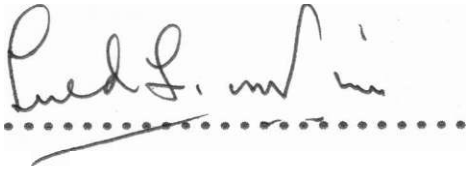
A DISSERTATION PRESENTED IN PART-FULFILMENT FOR THE DEGREE OF MASTER OF MEDICINE (M.MED.) IN INTERNAL MEDICINE OF THE UNIVERSITY OF NAIROBI.

DECLARATION

This dissertation is my original work and has not been presented for a degree in any other University.

Signature:  .....

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CONTENTS

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LIST OF TABLES AND FIGURES

LIST OF TABLES AND FIGURES

PAGE

" SUMMARY

A prospective study of electrocardiograms (ECG's) in 102 hypertensive African patients was carried out at the Kenyatta National Hospital. Mean age for these patients was 45.2 years. All the patients were in normal sinus rhythm. Forty seven percent of the patients had bradycardia, although this could be attributed to the drugs the patients were taking. The P wave duration increased with the severity of hypertension,. Twenty five percent of the patients had evidence of first degree A-V block. This was expected as most of the patients were taking B-adrenergic receptor blocking durgs for their hypertension. Two patients had LBBB and none had RBBB. There were 13 patients with evidence of left axis deviation (LAD) and the incidence increased with the severity of hypertension. S-T, T changes were noted in 12.8% and 29% of patients respectively. Q-Tc duration did not increase with severity of hypertension. U wave incidence was 35.3% in this study. Left Ventricular hypertrophy was noted in .27.5% of the patients using the criteria devised by Romhi&t et al (34)<,

INTRODUCTION

Dr. Albert Cook (1) who worked at the Church Missionary Society* s hospital at Mengo, Uganda from 1897 - 1901 published his "Notes on the diseases met with in Uganda, Central Africa", and noted that "... high tension pulses were not met with ...."© Other workers after him (2,3) also thought that hypertension was rare in the African population as opposed to the caucasian. However, it is common knowledge now that hypertension is indeed very common in the African (4 - 15).

Systemic hypertension may be accompanied by electrocardiographic (ECG) changes. Most of the studies, on E.C.G changes in hypertension, have been carried out in the developed countries. In black Africa, there have been studies in Nigeria (4), Uganda (5,7), Tanzania (9) and in Zambia (8). All the above studies show that there are ECG changes in hypertensive African patients. The common changes found are those consistent with left ventricular hypertrophy (LVH), various types of conduction defects, S-T, T changes and non-specific U waves.

Although some work has been done on hypertensive patients in Kenya (3,16 - 22), none has included ECG changes. Foster (23), in Mombasa, while studying the possible role of hypertension in unexplained forms of heart failure, showed some ECG changes in these patients.

He however studied three patients, two of whom showed features of bundle branch block and the third LVH. Rees et al (24) in an electrocardiographic survey of "pombe" drinkers found that three of the seven subjects with diastolic pressures greater than 90 mmHg (presumably hypertensives) showed normal tracings. There was however no mention of the ECG findings in the other four subjects.

Apart from the work of Foster (23) and Rees (24), no work has been done on the electrocardiographic changes in systemic hypertension in Kenya, hence the purpose of this study.

MATERIALS & METHODS

Patients attending Kenyatta National Hospital (KNH) for hypertension were the subject of this study. Patients were either seen in the medical outpatient clinic (MOPC) or in the medical wards. The patients were therefore not consecutive patients attending KNH for hypertension, since some patients would go to the other departments of the hospital. They were however consecutive patients seen by the author.

A total of 102 patients were included in the study. In each case, a detailed history was taken and physical examination was carried out in order to exclude patients with significant respiratory or valvular heart disease which might be expected to alter certain diagnostic criteria on the ECG. Those patients who appeared grossly obese clinically and those with spinal anatomical deformity were excluded.

The patients were being followed up for what was presumed to be essential hypertension although no attempt was made in delineating the cause of the hypertension. This however would not make any difference since the effect of a raised systemic pressure on the heart would give the same ECG changes whatever the cause (8).

The only criterion for inclusion in this study was the finding of a raised diastolic blood pressure reading of over 100 mmHg. While it is known that for a patient to be labelled hypertensive his diastolic blood pressure must be persistently above 90 mm Hg, in this study only one reading was taken in

patients who are already known to be hypertensive, are being followed up and most were on treatment. The choice of 100 mmHg was to make sure the subjects actually had elevated diastolic blood pressure at the time of the study and is based on already published work (8)0

The blood pressure was measured by indirect auscultation using a mercury sphygmomanometer and a stethoscope (25)„ The sphygmomanometer was constantly checked before each blood pressure measurement to ensure that:-

(a) The mercury level was at zero.
(b) The connections fitted without leakage.

The cuff size used in the study was of 12x23 cm (26).

The following conditions were fulfilled for creating standard conditions of indirect blood pressure recording (27):-

(i) The arm was not constricted by clothing.
(ii) There was no exertion, eating, smoking or exposure to cold for H hour before procedure,,
(iii) No change of posture was made for 5 minutes before recording,*

Procedure for recording blood pressure:

(a) The pressure cuff was snuggly applied to either arm, its lower border 2 - 3 cm above the antecubital fossa. The cuff was applied early and left in place during the interview (27),

(b) The blood pressure was taken when the patient was sitting with the arm in a roughly horizontal position at heart level, supported on a desk (27,28).

(c) The cuff was rapidly inflated to a level above the radial palpatory pressure (27)„

(d) Run down of the mercury column was started promptly, at a rate of fall of 2 mm per second (27).

(e) The systolic pressure level was determined by the first perception of Korotkoff sounds, phase 1 (27).

(f) The diastolic pressure level was determined by the perception of disappearance of Korotkoff sounds, phase V (28 - 31).

The electrocardiogram machine used in this study was the SIEMENS CARDIOSTAT 701, direct writing machine. The machine was calibrated so that a current of one millivolt deflected the stylus 10 mm in height. It was regularly checked for any technical faults (eg. damping, interference etc ...). The recordding speed used for each tracing was 25 mm per second on standard paper (1 mv = 10 mm).

Each patient was recorded relaxed lying supine on a couch, at least 30 minutes after any cigarette smoking and after at least 15 minutes of rest (without physical exertion). In each case, care was taken in skin preparation and electrode jelly application so that effective skin contact was obtainedo

The standard 12 - lead, two plane (frontal and horizontal) scalar electrocardiogram was then recorded. All the recordings were done by one observer.

Standard interpratation of the ECG except for evidence of LVH was done using the method of Grant (32), Goldman (33) and the help of a cardiologist when necessary.

She parameters assessed during analysis of each ECG tracing were:-

ia) Rhythm

(b) Rate:-

(i) Ventricular

(ii) Atrial

(c) P Wave

(i) Maximum duration in standard leads (1,11,III)

(ii) Maximum amplitude (height) in standard leads

(d) P-R Interval

The longest P-R interval measured from the beginning of the P wave to the first deflection of the next QRS complex in any of the 12 leads,

(e) QRS Complex; -

(i) The longest duration in any lead

(ii) Amplitudes of R&S waves in V_x or V_2, V_g or , I, II, III (for use as criterion in ECG diagnosis of LVH).

(iv) QRS frontal axis using voltage amplitudes in leads 1 & II (also for use as ECG criterion in the diagnosis of LVH)„

(v) R-R interval (for Q-T interval correction).

(f) S-T segment depression in $V_{c_{b_t}b}$ »

(g) T wave inversion in $V._{D_t b}$

(h) Q - T Interval

(i) Measured value - from the beginning of the QRS complex to the end of the following T wave, using the lead with the tallest:- T wave for clarity of defining the end of the T wave,

(ii) Corrected value - using the Bazert formula

$$Q\text{-}Tc \text{ » } \frac{Q \quad T \text{ Measured value}}{v/R - R \text{ Interva1}}$$

(iii) U wave:- Presence or absence in any lead.

There are to date over 30 different ECG criteria for diagnosing LVH described in the literature (see appendix I). Those criteria with a high degree of sensitivity generally have a low degree of specificity and the criteria with a high degree of specificity generally have a low degree of sensitivity (34) The point-score system for the ECG diagnosis of LVH as proposed by Romhilt et al (34) is therefore used in this study (see appendix II).

RESULTS

AGE AND SEX

The age and sex distribution of the patients in the study is shown in Figure 1. There were 43 males (42%) and 59 females (58%). The age ranged between 15 and 65 years (mean = 45.2 years).

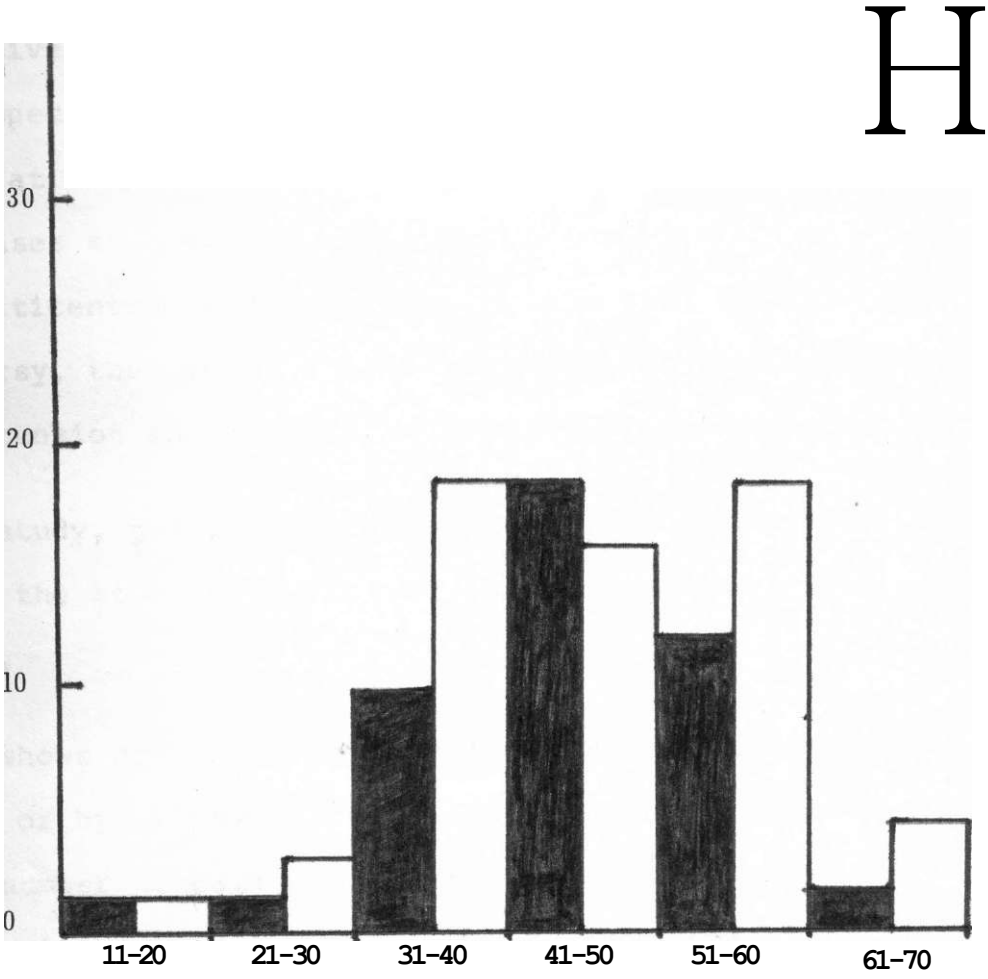

FIG 1: AGE AND SEX DISTRIBUTION OF THE 102 PATIENTS WITH SYSTEMIC HYPERTENSION STUDIED.

v ov * * * * *

SEVERITY OF HYPERTENSION

The Keith-Wagener-Barker (35) grading of the severity of hypertension has been in use since its inception in 1939« However, Akinkugbe (4), Williams (36) and Gelfand (37) noted that one of the oddities of hypertension in the African is the rarity of retinal changes in the presence of severe hypertensive disease. Locally, Foster and Jan Mohamed (22) reported 46,2% incidence of hypertensive retinopathy in 67 hypertensive Kenyan African subjects of the Coast Province. In a prospective hospital study of 100 hypertensive Kenyan Africans at KNH, Awan, Ojiambo and Ogada (38) found 75% of the cases studied had hypertensive retinopathy. Unitl large multicentre studies are carried out to sort out the controversy, the use of retinal changes for grading the severity of hypertension in the African should not be encouraged.

In this study, grading of the severity of hypertension was based on the study of Levitt et al (8) as already alluded to above.

Table 1 shows grading of diastolic pressure according to severity of hypertension in the 102 patients. It shows the largest number of patients having grade 1 and the least number having grades 4 and 5. The above differences are expected as most of the patients are on one form of treatment or the other.

RHYTHM

All the patients in the study showed normal sinus rhythm in their ECG. Sinus arrhythmia which is manifested by alternating periods of slower and more rapid heart rates related to respiration, was not looked for as it does not indicate a disease state. It is a normal finding especially in the young C33)•

RATE

There were 24 patients (47%) with atrioventricular rates less than 60 beats per minute and no patient had a rate greater than 100 beats per minute.

QRS AXIS

This is also one of the criterion for diagnosing LVH (34), Table 5 shows the distribution of the frontal plane QRS axis.

There were 13 patients with evidence of left axis deviation, making an overall incidence of 12»7%. No patient had right axis deviation,,

S-T_t T CHANGES

In this study, S-T, T depression or inversion respectively were specifically looked for and were used in the criteria for the diagnosis of LVH.

Thirteen patients (12.8%) had evidence of S-T depression and 29 (28.4%) had T wave inversion. This shows that in som# cases there would be T wave inversion without necessarily having S-T depression.

WAVE

There were 30 patients whose ECG had a P wave duration of more than 0.11 seconds (see Table 2). The P wave duration ranged between 0.08 to 0.12 seconds. There was a significant increase in P wave duration with the degree of hypertension ($p<0.01$) for grades 1 & 5 diastolic hypertension.

All the patients had a normal P wave amplitude i.e. less than 3 millimetres*

P-R INTERVAL

The P-R interval ranged between 0.14 seconds and 0.34 seconds (see Table 3). Twenty five patients (24.5%) had evidence of

first degree atrioventricular block (P-R interval>0.20 seconds). No patient had second or third degree block.

QRS DURATION

The range of the QRS duration was 0.04 to 0.14 seconds (see Table 4). There was no significant increase in QRS duration with the severity of hypertension ($p>0.05$ for grades 1 & 5)„

Two patients had evidence of left bundle branch block (LBBB).

None had Right bundle branch block (RBBB)$_0$ There were however 4 patients whose ECG showed prolonged QRS duration with no specific features of either Left or Right bundle branch block.

QRS amplitude was measured in V_X or V_2 and V_5 or V^; I, II, III and is used as one of the criteria for diagnosing LVH (34)$_0$

Q-Tc DURATION AND U WAVE INCIDENCE

Table 6 shows the correlation of Q-Tc interval and U wave incidence to the degree of hypertension.
The maximum Q-Tc interval recorded was 0.55 seconds and the minimum recorded was 0.48 seconds$_n$

There was no increase of Q-Tc duration with the severity of diastolic blood pressure (p"^ 0.05) for grades 1 and 5.

There were 36 patients (35.3%) whose ECG showed presence of U waves* There was no increase or decrease in the incidence of U waves with severity of diastolic hypertension.

» <

LVH

There were 20 patients whose ECG showed definite evidence of LVH and 8 patients had ECG showing probable LVH. When patients with probable LVH are included together with those having definite LVH, then the overall incidence of LVH would be 27.5%.

Among the patients with definite LVH, 8 patients (40%) had left axis deviation, 9 patients (45%) had S-T depression, 14 patients (70%) had T wave inversion and 11 patients (55%) had both S-T depression and T wave inversion,,

TABLE 1; DIASTOLIC PRESSURE GRADING SHOWING SEVERITY OF SYSTEMIC HYPERTENSION IN THE 102 PATIENTS.

GRADE OF HYPERTENSION	NUMBER OF PATIENTS		
	MALES	FEMALES	TOTAL
1	22	31	53
" 2 '	12	13	25
3	5	5	10
4	1	6	7
5	3	4	7
TOTAL	43	59	102

GRADING BASED ON LEVITT et al (8)

Grade 1:- diastolic reading 100 - 110 mmHg

Grade 2:- « " 111 - 120 M

Grade 3:- M " 121 - 130 "

Grade 4:- " " 131 - 140

Grade 5:- " " 140 "

TABLE 2: CORRELATION OF P WAVE DURATION AND AMPLITUDE TO SEVERITY OF SYSTEMIC HYPERTENSION.

DEGREE OF HYPERTENSION	NO. OF PATIENTS	DURATION IN SECONDS				AMPLITUDE IN MILLIMETRES			
		MAX	MIN	MEAN	SD	MAX	MIN	MEAN	SD
GRADE 1	53	0.12	0.08	0.11	0.01	2	0.05	1.17	0.55
GRADE 2	25	0.12	0.8	0.11	OoO1	2	0.05	1.27	0.79
GRADE 3	10	0.12	0.08	0.11	0.01	2	0.05	0.01	0.70
GRADE 4	7	0.12	0.10	0.10	0.01	2	1.00	1.57	0.53
GRADE 5	7	0.12	0.10	0.11	0.01	2	1.00	1.64	0.48

TABLE 3: CORRELATION OF P-R INTERVAL AND INCIDENCE OF A - V BLOCK TO THE SEVERITY OF SYSTEMIC HYPERTENSION.

DEGREE OF HYPERTENSION	NO. OF PATIENTS	P-R INTERVAL IN SECONDS			ATRIOVENTRICULAR BLOCK			
						1° BLOCK	2°BLOCK	3°BLOCK
		MAX	MIN	MEAN	SD	NO. OF PTS.	NO. OF PTS.	NO. OF PTS.
GRADE 1	53	0.31	1.16	0.20	0.041	13	NIL	NIL
GRADE 2	25	0.34	1.16	0.20	0.03	7	NIL	NIL
GRADE 3	10	0.24	0.14	0.20	0.03	3	NIL	NIL
GRADE 4	7	0.22	0.16	3.19	0.021	1	NIL	NIL
GRADE 5	7	0.24	0.16	3.19	0.03	1	NIL	NIL

TABLE 4: CORRELATION OF QRS DURATION AND INCIDENCE OF BUNDLE BRANCH BLOCK TO THE SEVERITY OF SYSTEMIC HYPERTENSION.

DEGREE OF HYPERTENSION	QRS DURATION IN SECONDS				NO. OF PATIENT WITH BUNDLE BRANCH BLOCK		QRS 0.10-0.11 WITH NO. SPECIFIC FEATURES OF L or RBBB
Km'^{1}	MAX	MIN	MEAN	SD	LBBB	RBBB	
GRADE 1	0.14	0.04	Oo 08	0.03	1	NIL	2
GRADE 2	0.14	0.04	0.09	0.02	1	NIL	2
GRADE 3	0.10	0.04	0.08	0.02	NIL	NIL	NIL
GRADE 4	0.12	0.06	0.08	0.02	NIL	NIL	NIL
GRADE 5	0.12	0.08	0.10	0.01	NIL	NIL	NIL

TABLE 5: DISTRIBUTION OF FRONTAL PLANE QRS AXIS IN PATIENTS WITH SYSTEMIC HYPERTENSION.

GRADE OF HYPERTENSION	NO. OF PATIENTS	PATIENTS WITH NORMAL AXIS		PATIENTS WITH LEFT AXIS		PATIENT WITH RIGHT AXIS	
		NO.	%	NO.	%	NO.	%
GRADE 1	53	48	94.1	3	5.9	NIL	NIL
GRADE 2	25	21	77.8	6	22.2	NIL	NIL
GRADE 3	10	10	100	NIL	NIL	NIL	NIL
GRADE 4	7	5	71.4	2	28.6	NIL	NIL
GRADE 5	7	5	71.4	2	28.6	NIL	NIL

TABLE 6: CORRELATION OF Q-Tc INTERVAL AND U WAVE INCIDENCE TO THE SEVERITY OF SYSTEMIC HYPERTENSION.

DEGREE OF HYPERTENSION	NO. OF PATIENTS	3-Tc INTERVAL IN SECONDS				U WAVE INCIDENCE	
» t		MAX	MIN.	MEAN	SD	NO. OF PATIENTS	%
GRADE 1	53	0.53	0.35	0.42	0.04	17	33o3
GRADE 2	25	0.52	0.35	0.43	0.04	12	44.4
GRADE 3	10	0.55	0.38	0.44	0.05	3	30.0
GRADE 4	7	0.48	0.41	0.44	0.03	2	28.6
GRADE 5	7	0.50	0.42	0.45	0.03	2	28.6

TABLE 7; INCIDENCE OF LVH IN PATIENTS WITH VARIOUS GRADES OF SYSTEMIC HYPERTENSION.

GRADES OF HYPERTENSION	NO. OF PATIENTS	NO. OF PATIENTS WITH LVH	%	NO. OF PATIENTS WITH PROBABLE LVH	%
GRADE 1	53		9*8	NIL	NIL
GRADE 2	25		25 <>9	4	14.8
GRADE 3	10		20.0	2	20.0
GRADE 4			28.6	NIL	NIL
GRADE 5			57.1	2	28o6
TOTAL	102	20	19o6	8	7.8

DISCUSSION

RHYTHM

All the patients in the study showed sinus rhythm in their ECG. This finding agrees with the study of Levitt et al (8). D'Arbella et al (7) in a study of 222 patients who were sent for ECG because they were hypertensive, found 3 patients with atrial fibrillation. Although hypertensive heart disease may cause atrial fibrillation, in the latter study patients were not screened for any valvular heart lesions. There is therefore the possibility that some patients with valvular heart disease could have been included in the study.,

Three patients out of 102 patients in this study had Premature ventricular contractions (PVCs) in their ECG„ The PVC's were unifocal* Levitt et al (8) found PVC*s in 5 out of 164 patients* D'Arbella et al (7) found PVC's in 8 out of 222 hypertensive subjects© It would therefore appear that there is no significant difference in the finding of PVC's in hypertensive subjects in this study with that of the above studies*

RATE

By definition a person having a heart rate of less than 60 beats per minute has bradycardia and one having more than 100 beats per minute has tachycardia (32, 33). However, well trained athletes may exhibit bradycardia, and this is said to be a sign of excellent

physical training* In this study, there were 24 patients (47%) who had bradycardia and no *patient* had tachycardia*.

All the patients who had bradycardia however were on B-Adreno-receptor blocking drugs (B-blockers)„ None of the patients admitted to being an athlete® It is therefore difficult to compare these findings with those of studies elsewhere as the types of drugs and their dosages being given elsewhere and in this study differ® It also became difficult to correlate the V*

atrioventricular rate to the degree of hypertension.

P WAVE

This is the deflection produced by atrial depolarisation.

(a) Duration: The P wave duration is said not to exceed 0.11 seconds (32). The number of patients with P wave duration 0.11 seconds on their ECG increased with the severity of hypertension*

D'Arbella et al (5, 7) found left atrial P wave (P mitrale) in 88 out of 222 hypertensive Ugandan Africans (39#6%). While it is possible that the P mitrale found in these patients could have been due to increased afterload, their figures would appear to be on the higher side. This could be attributed to the fact that

the patients studied in Uganda were not screened to exclude valvular and other heart diseases.

(b) Amplitude: The P wave amplitude is said not to exceed 3 millimetres (32, 33). All the patients in this study had a normal P wave amplitude.

P-R INTERVAL

This measures the atrioventricular conduction time. The normal value is in the range of 0.12 to 0„20 seconds (32,33). The P-R interval varies with the heart rate, the slower the heart rate the longer the P-R interval and vice versa (32, 33)«

In this study 25 patients (24.5%) had evidence of first degree atrioventricular block. No patients had either second or third degree heart block. Lodha and Makene (9) found 2 patients out of 145 hypertensive patients whose ECG showed first degree A-V block and none had second or third degree block. D'Arbella et al (5,7) found a first degree AV block in 14 out of 222 hyertensive patients*

First degree A-V block however may occur in the absence of any evidence of organic heart disease. It may also be a feature of ischaemia, rheumatic fever, digitalis therapy, other drugs and old age (32,33).

Since the P-R interval is dependent on so many factors, comparison of the above studies and this one is not possible.

QRS DURATION

This is the measurement of total ventricular depolarisation time$_0$ It is measured from the onset of the Q wave (or R if no Q is visible) to the termination of the S wave. The upper limit of normal is 0.01 seconds (32, 33).
1

In this study 2 patients (2.0%) had evidence of LBBB, no patients had ECG evidence of RBBB and 4 patients (3.9%) whose ECG showed QRS duration of 0.10 - 0.11 seconds with no specific features of R or LBBB.

LBBB may occur in almost any form of heart disease including the following (32,33):-

(i) Coronary artery disease
(ii) Any of the diseases which produce LVH (eg hypertension and aortic valvular disease).
(iii) Congenital or sometimes acquired lesions involving the septum.

It is said to be rare in an individual with no clinical evidence of organic heart disease. Most of the above conditions were specifically looked for in the selection of patients for this study (see materials and methods)»

D'Arbella et al (5,7) in their study observed that there were 3 patients (1.4%) whose ECG showed evidence of RBBB, 6 patients (2.7%) with evidence of LBBB and 27 patients (12o2%) whose ECG showed QRS duration of 0.10 - 0.11 seconds with no specific features of either R or LBBB.

Comparison of this study and the latter is rather difficult as no screening of patients was done in the latter study.

It is noteworthy that in this study the patients whose ECG showed LBBB and those with QRS duration of 0.10 - 0.11 seconds with no specific features of R or LBBB all had diastolic pressures grades 1 and 2.

QRS AXIS

Important factors that influence the QRS axis include body build (especially height), posture (lying on either side or sitting up alters the heart* s electrical axis and transitional zone), forceful respiration and mechanical factors (emphysema, ascites, pregnancy and large intraabdominal tumours)«, These factors were looked for in the selection of patients for this study*

In this study 13 patients (12.7%) had left axis deviation and none had right axis deviation. The incidence tended to increase with the severity of hypertension.

D'Arbella (5,7) found evidence of Left axis deviation in 6 out of 222 hypertensive patients (2.7%). Lodha and Makene (9) found left axis deviation in 10 out of 141 hypertensive patients (7.0%). It is not immediately clear as to why there should be so much difference in the three studies above.

In the study of Romhilt et al (34) in which they devised the point-score system for the ECG diagnosis of LVH, left axis deviation scored 2 points (appendix II). It had a sensitivity of 24.4% and a specificity of 13.0%. The results of this study would therefore be in general agreement with the latter study.

S-T, T CHANGES

S-T interval: This is the duration of the RS-T segment. It is that portion of the ECG tracing from the J point to the onset of the T wave (32, 33). This segment is usually isoelectric but may vary from -0.5 to 2.0 mm in praecordial leads. It is elevated or depressed in comparison with that portion of the base line between the termination of the T wave and the beginning of the P wave.

T WAVE: This is the deflection produced by ventricular repolarisation (32, 33).

S-T depression may occur in many situations, for example, patients having angina pectoris, digitalis intoxication, myocardial infarction anxiety state or tobacco smoking, (32, 33).

T wave inversion may occur in myocardial infarction, ischaemic heart disease and perimyocarditis (32, 33).

It has also been observed by a number of workers that there are non-specific S-T, T changes in healthy Africans. Somers and Rankin (39) found S-T elevation in 44% of healthy Nilotics. Powel (40) found 37% incidence of S-T segment changes and 39% incidence of T wave changes in non-cardiac patients. Wessenburger (41) noted frank inversion of the T *Sd*ave in 11% of 131 adult negro men. Wanene (42) and Okwera (43) recently in Nairobi noted similar changes as above.

With the controversy regarding the S-T, T changes in normal Africans still raging, it would be futile to try and draw conclusions when studying EC©s in hypertensive patientso However, since S-T, T changes (with typical pattern of Left ventricular strain with the S-T, T vectors shifted in direction opposite to the mean QRS vector in leads V,. and Vg) is included as a criterion in the ECG diagnosis of LVH (34), the incidence of S-T depression and T wave inversion has been studied*,

Thirteen patients (12.8%) had evidence of S-T depression and 29 patients ($28_0$4%) had T wave inversion. There were patients whose ECG showed T wave inversion without necessarily having S-T depression. This is in agreement with the above studies (39 - 43),,

Q-T DURATION

This is measured from the onset of the Q wave to the end of the T wave. It measures the duration of electrical systole (32). The Q-T interval varies with the heart rate and ideally should be corrected (Q-Tc). This is done by using the Bazzart formula. The normal Q-Tc duration should not exceed 0.42 seconds in men and 0.43 seconds in women.

Prolonged Q-Tc interval may occur in the following conditions:

(i) Almost every known acute infectious disease which involves the myocardium.

(ii) Refrigeration during surgery.

(iii) Quinidine toxicity.

(iv) Hypocalcaemia.

As noted in the section dealing with materials and methods, some of the above conditions were excluded in the selection of patients for this study. Clinically there was no patient with features of hypocalcaemia although a serum calcium level would have been conclusive.

There were 45 patients (44%) whose ECG showed prolonged Q-Tc interval. There were 17 patients (33.3%) in grade 1, 15 patients (55.6%) in grade 2, 5 patients (50.0%) in grade 3, 3 patients (42.9%) in grade 4 and 5 patients (71.4%) in grade 5. The number of patients having grade 5 hypertension was small and thus no conclusions can be drawn from the above findings.

U WAVE

This is the deflection (usually positive) seen following the T wave and preceding the next P wave. The exact cause of of this wave is unknown. It is currently thought to be the result of the slow repolarisation of the intraventricular conduction system (33).

For some unknown reason, prominent U waves occur in v*_ hypokalaemia, not increased incidence (33). This is freguently superimposed upon the T wave and therefore produces "apparent prolonged Q-T interval".

In this study 36 patients (35„3%) had U waves in their ECG. This would seem to disagree with what D'Arbella et al (5,7) found. -In the latter study 60% of the patients showed U waves in their ECG_0 Somers and Rankin (39) found U waves in 47% of healthy Bantu men and 24% of Nilotic men_0

Recently, Okwera (43) found U waves in 16% of healthy male Africans and 15% of healthy female African patients. It is also known that many ECG recordings may not show a distinct U wave and this may cause an apparent low incidence that has been reported in many studies.

However, since nobody at the moment knows the exact origin of the U wave, it becomes difficult to draw conclusions from the above studies.

LVH

LVH commonly results from the following clinical states (32,33)

(i) Hypertension:- essential, renal or hormonal.

(ii) Aortic valvular disease*

(iii) Rheumatic mitral insufficiency*,

(iv) Long - standing coronary artery disease,

(v) Nutritional and idiopathic hypertrophies:- beriberi heart disease, and the chronic myocarditis and myocardiopathie

(vi) *Congenital heart disease:- Patent Ductus Arteriosus, coarctation of the aorta and tricus pid atresia.

In the selection of the patients for this study, the above conditions were excluded.

An ECG diagnosis of LVH can be made very commonly when LVH is anatomically present. In fact, the ECG may be diagnostic before any roentgenographic evidence is present (34). It therefore follows that in LVH secondary to hypertension, the ECG may improve or revert to normal following successful medical or surgical treatment of hypertension.

The ECG criteria developed over the years for the diagnosis of LVH (appendix 1) have been found to be either non-specific or insensitive (34).

Lodha and Makene (9) using the point-score system found definite LVH in 37% of patients and 41% of the patients had probable LVH. However, the criteria they have used (appendix III) are not the same with those as originally devised by Romhilt (appendix II). For reasons not apparently clear, Lodha and Makene included criteria which were not in the original work by Romhilt et al and thus increased their chances of diagnosing LVH, hence their higher figures.

Somers and Rankin (39) in their study of the ECG in 209 normal East Africans employed as their voltage criteria for LVH the sum of Sv_x or Sv_2 + Rv_5 or Rv_g which is 35 mm or greater. They found 28.7% of these people have LVH. The obsession which clinicians in KNH have of diagnosing LVH using the sum of Sv.^ or Sv_2 + Rv_5 or Rv_g should now be discarded.

RECOMMENDATIONS

A larger study than that of Awan, Ojiambo and Ogada (38), preferably multicentre, should be carried out to determine once and for all the suitability of the use of the Keith-Wagener-Barker grading of the severity of hypertension in the African,

Criteria devised by Romhilt (34) for the diagnosis of LVH is recommended to clinicians and researchers because most of the other criteria are either insensitive or non specific. Besides, some of the criteria recommended by the other workers can be found in normal people,.

Presently, echocardiography is said to be more sensitive in the diagnosis of LVH. It is recommended that a prospective comparative echocardiographic and electrocardiagraphic study be carried out.

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APPENDIX 1

CRITERIA FOR DIAGNOSIS OF LVH

(ADOPTED FROM ROMHILT et al, CIRCULATION; VOL XI: 185. 1969).

PRAECORDIAL LEAD CRITERIA

1. (a) R + S >40 mm

   (b) R + S>45 mm

   (The greatest R in any praecordial lead plus the greatest S in any praecordial lead)

2. (a) Sv_1 or Sv_2 + Rv_5 > 40 mm

   (b) R + S > 35 mm

   (The sum of the R wave plus the S wave in any single praecordial lead)

   (a) Sv_2 + RV_5 or RV_6 > 35 mm

   (b) Rv_5 or Rv_6 > 26 mm

   (a) Sv_x + RVg or RVg>30 mm

   (b) Sv_2 + Rv_4 or Rv_5>35 mm

   Sv_x + Rv_5 > 30 mm

   (a) Sv_1 or Sv_2 + Rv_5 or Rv_6 ≥ 35 mm

   (b) Rv_5 or Rv_6 ≥ 20 mm

APPENDIX 1 CONT.

.(a) *SVj* + Rv^>33 mm for *females,*

Sv^ + 36 mm for males,

(b) Rv_6 > 20 mm

Rv_6 Rv,.: Evaluated separately with QRS transition between and QRS transition in any praecordial lead.

Y„

Sv_2 + Rv_5 or V_6 ≥ 45 mm

Sv_x ^ 24 mm

Onset of intrinsicoid deflection in V_5 or V_g ^0.05 seconds.

(a) Sv_x or Sv_2 + RV_5 or Rv_g > 35 mm

(b) Sv_2 + Rv_5 > 35 mm

LIMB LEAD CRITERIA

(a) R_x + S_3 > 25 mm

(b) R_1 > 15 mm

R^ 13 mm

RaVL *y 7.5* mm

4. (a) SaVR > 14 mm

   (b) RaVL > 12 mm

   (c) RaVF > 19 mm

5. (a) RaVL > 13 mm

   (b) RaVF > 20 mm

6. (a) RaVL > 11 mm

   *(b) RaVF > 20 mm

7. RaVL > 11 mm

8. Lewis index + 17 or greater.

9. Left axis deviation - 30° to 90°.

APPENDIX II

CRITERIA FOR THE POINT SCORE

SYSTEM USED IN DIAGNOSING LVH

1. **AMPLITUDE** . . . . . 3 POINTS

   Any of the following:-

   (a) Largest R or S wave in the limb leads 20 mm

   (b) S wave in V_1 or V_2 30 mm

2. ST-T,T segment changes (typical pattern of Left ventricular strain)with S-T,T segment vector shifted in direction opposite to the mean QRS)•

   Without digitalis . . . . . 3 POINTS

   With digitalis . . . . . 1 POINT

3. Left atrial involvement . . . . . 3 POINTS

   Terminal negativity of the P wave in V.^ is 1 mm or more in depth with a duration of 0.04 seconds or more.

4. Left axis deviation -.30° or more . . . . . 2 POINTS

5. QRS duration 0.09 seconds . . . . . 1 POINT

   id deflection in V^ 6 > 0>05 seconds . . . . x POINT

LVH -2 5 POINTS; Probable LVH - 4 POINTS

APPENDIX III

POINT SCORE SYSTEM FOR LMH AS USED BY LQDHA and MAKENE (9).

QRS amplitude (three points).

R a V L > 1 1

R V_5 - V_6 ≥ 25

S V_1 - V_3 ≥ 25

$(R_X + S_3) - (S_1 + R) \wedge 18$

$Sv_x + RVj. - {}_6 \geq 35$

(Each criteria: two points, maximum score 3 points).

S-T segment and T wave changes in absence of digitali (three points).

(a) T changes - two points

R/T in V_5 ^ 10

T inversion V_5 - V_6

(b) S-T depression more than 0.5 mm in two points

V - two points.

5 6»

(c) T inversion more than 5 mm and/or S, S-T depression more than 2 mm in above leads - one extra point (maximum score 3 points).

QRS duration more than 0.09 seconds - one point.

APPENDIX III Cont.

Intrinsicoid deflection more than 0.05 seconds in V_3 _ g one point.

QRS axis deviation more than - 30° in frontal plane two points.

Left atrial abnormality in absence of mitral stenosi (three points).

Total	**13**	**points**
LVH	**5**	**Points or more**
Probable LVH	**4**	**points.**