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EFFECT OF CHRONIC HEAVY ALCOHOL INTAKE ON THE HEART INVESTIGATION BY ELECTROCARDIOGRAPHY MAINLY. ^

BY

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- D E C L A R A T I O N -

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

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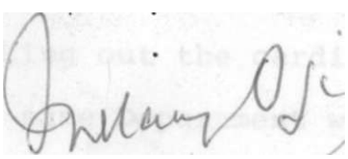

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SUMMARY

Fourty three male subjects without any cardiac symptoms or signs, who had been drinking for a mean of 12.5 years range (5-25 years), their ages ranged from 20-55 years. These subjects, and thirty seven non drinking controls from the same environment, and matched for age; had their cardiac functions studied using electrocardiography, some very few subjects were also studied by echocardiography and chest x-rays. There were no abnormalities found either on electrocardiography or on the few echocardiographs and chest x-rays. The negative results in this study may mean that subjects had marked alcohol tolerance, possibly due to their general physical fitness, or the myocardial abnormalities could not be picked up by the methods used, or a possible sampling bias.

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INTRODUCATION AND LITERATURE REVIEW

Association between alcohol and heart disease has been recognised since late 19th Century. Steell[1] wrote about it in 1893, and Mckenzie[2] recognised and described the association while working in China in 1902.

Brigden[3], Evans[1'], Walsh and Burch[5] and other workers who have studied alcohol associated cardiomyopathy, clinically and pathologically over long periods have found non-specific changes, not very different from other dilated cardiomyopathies. The term alcoholic cardiomyopathy is applied in dilated cardiomyopathies associated'with heavy intake of alcohol, and which on withdrawal of alcohol there is clinical as well as microscopic improvement, and reoccurs on resumption of alcoholic intake.

Between 1906 and 1931 beri-beri was discovered to cause heart failure and most literature during this period is mainly about association of congestive heart failure and thiamine deficiency. Some of these patients with beri-beri heart disease were also alcoholic, but the beri-beri theory was so easy to explain that even those that did not respond to thiamine were just labelled resistant; the contribution of alcohol per se in these resistant patients was glossed over.

Between 1931 and 1939, Jones and Bramwel[6] studied alcohol associated heart disease and found that whereas one group responded to thiamine, another group did not. This study-revived interest in alcohol per se as a cause of cardiomyopathy as distinct from thiamine deficiency cardiomyopathy. Since then, a lot of work has been done on

the subject. As already noted, alcoholic cardiomyopathy has been a diagnosis by exclusion of all other possible causes of congestive heart failure. There are very few studies that have been done among heavy drinkers to find out what early changes by non-invasive methods can be detected prior to the onset of failure. The following is a summary of the work that has been done on the subject of alcoholic cardiomyopathy and some on beri-beri disease or.both. Beri-beri heart disease is included because, for a long time, both- beri-beri and alcoholic heart disease were thought to be the same disease entity.

I. Wenckebach[7] 1928. Delivered a classic lecture on beri-beri heart disease. After collating his own data and those of several Dutch workers in the Far East, he concluded that beri-beri heart failure was characterised by cardiac enlargement of both right and left chambers with primarily right sided failure. In their series there were no significant electrocardiagraphic changes, except some degree of increased voltage amplitude and a right axis preponderance. In all these cases, there was no response to cardiac glycosides or diuretics, but they responded to thiamine.

II. Blankehorn et al[8] in 1946, reported 12 cases of beri-ber type heart failure. They used criteria earlier described by Blankehorn that included:

i) there was insufficient evidence for other aetiology;

ii) three or four months of thiamine free diet;

iii) signs of neuritis or pellagra;

iv) enlarged heart;

v) dependant oedema;

vi) elevated -venous pressure;

vii) minor ECG changes and

viii) recovery with decrease in heart size after thiamine administration.

Eleven of the twelve patients were alcoholics and it is stipulated in the paper that alcohol contributed to their poor diet, but -they failed to recognize possible direct effect of alcohol on the heart. In their summary they concluded that beri-beri was a heart failure with fast circulation. Alcohol was not considered as a cause of heart failure of those who did not respond to thiamine,

III. Brigden (A) in 1957 examined causes of cardiac failure in 50 patients due to uncommon causes. Of these, he found that alcohol accounted for 13 cases, the biggest single group in the study. The rest included congenital, association with inflammation, puerperal myocarditis, acromegally, amyloidosis and collagen diseases. In these, most patients had cardiomegaly radiologically and some had non specific ECG changes including T wave changes, low amplitudes and various bundle branch blocks in a few.

IV. Walsh and Burch 1963 reviewed cases of cardiomegaly of no definite aetiology other than association with prolonged high alcohol intake. On withdrawal of alcohol and bed rest, the cardiac size became normal again.

Brigden in 1964'', reported on 50 patients seen between 1952 and 1963 who had a history of prolonged heavy alcohol intake, 13 of the patients had no other cause for being in heart failure, except for heavy drinking. Of the 50, some had died, and autopsy revealed no coronary disease. Five of these patients had beri-beri. A second group presented v/ith various arrhythmias especially atrial fibrillation and other forms of arrythmias. A third group had hyperkinetic failure with ECG evidence of severe myocardial disease.
Sakaran Asoken[10] et al in 1972 in America, studied possible early signs in heavy drinkers. They found that most of their patients had mild cardiovascular signs such as exertional dyspnoea in a few, vague chest pains and some very few had 3rd heart sounds. ECGs and chest x-rays were normal. Cardiac catheterization however, showed lower cardiac output in heavy drinkers, average 2.56 L/Minute as compared to 3-19L/minute in controls. Catheter studies also revealed left ventricular end-diastolic volume was normal- in 7 out of 9 but left ventricular end diastolic pressure increased in all but two. Their study showed that there is some impairement of left ventricular end diastolic volume and left ventricular end diastolic pressure, and decrease in cardiac output.
John Demakis et al[11] 1974. This group studied 51 patients admitted into hospital in congestive cardiac failure with no other cause other than history of high alcohol. intake. The study took an average of 40.5 months. In the period, 15 patients improved -

group A; 12 patients' condition remained stable - group B; and 30 patients deteriorated - group C. It is notable that 73% of group A abstained after diagnosis as compared with 14% in group C and 25% from group B. They found that 40 patients had abnormal * T waves, 17 patients had low QRS complexes with or without abnormal T. waves. Four patients had pathological Q waves. Thirteen patients, had conduction defects: 4 in left bundle branch block and 2 in right bundle branch block. The rest had non specific conduction defects. Six patients had arrhythmias. Those who reached autopsy just showed enlargement of all four chambers. Histology showed myocardial hypertrophy and fibrosis with some patches of interstitial oedema.

Levi et al[12] in 1977 studied 43 subjects who had a high alcohol intake for 5 years or more. They calculated pre-ejection period and left ventricular ejection time from ECG, 'phonocardiogram and carotid pulse waves. Their study showed prolonged pre-ejection period and pre-ejection period index, and shortening of the left ventricular ejection time, consistent with .impaired myocardial function in the absence of any abnormalities in ECG and phonocardiogram.

In April, 1977, Goodwin[13] delivered a very comprehensive paper on alcoholic cardiomyopathy to the Fourth Conference of the European Association of Internal Medicine. He reviewed at length work done to that time and aptly differentiated beri-beri heart disease

from alcoholic heart disease; beri-beri being a high output failure while alcohol is a low output failure. He discussed, among other things, the pathology of the condition, the clinical feature, diagnosis and management.

Ettinger and Wu[14] in 1978 reviewed 24 patients who presented to hospital with various dysrrythmias after heavy bouts of drinking. In their cases, the only history common to all these patients was heavy alcoholic intake prior to start of the dysrrythmias. All of them were controlled within a short time in hospital, mostly with bed rest, very few needed drugs. On abstinence from alcohol, the symptoms did not reccur.

A British Medical Journal[15] on 29th April, 1982 reviewed work done on alcoholic cardiomyopathy since 1946. They reported among others, work done by Burch et al on rats where they demonstrated microscopic myocardial damage after prolonged drinking, irrespective of type or strength of alcohol, they used beer, wine and spirits. The lesions however, were not pathognomonic of alcohol and had been described in heart muscles diseases of different aetiologies.

An editorial in the Lancet of May, 1980[16] summarizes alcohol heart disease, and reviews work done to date. It states that in pre—clinical alcoholic cardiomyopathy, alcoholics have abnormal systolic time intervals, raised left ventricular end-diastolic pressures and depressed cardiac output. It goes on to state that once diagnosed in congestive cardiac failure, most

alcoholic cardiomyopathies progress inexorably to death within 3 to 4 years.

XIII. Goodwin[17] in a review of cardiomyopathy in general in his "Frontiers of Cardiomyopathy" said that pre-failure signs of gallop rhythm, cardiomegally, * impaired left ventricular frunction detectable on echocardiography and non specific electrocardiographic changes are hallmarks of early dilated cardiomyopathy of which alcoholic cardiomyopathy -is one. He also postulates the virus theory causing cardiac muscle damage leading to cardiomyopathy. In our setup with a plethora of vectors, the virus theory; possibly setting early cardiac damage which needs a trigrger like alcohol to full cardiomyopathy, would be very attractive

XIV. Rees and Chukwumeka[18] studied 135 "pombe" (local alcoholic drink) drinking subjects. In their study, they found 38 normal ECG tracings, 49 tracings of drinkers with minor ECG abnormalities and 48 tracings with major ECG abnormalities. Minor abnormalities included sinus bradycardia, ST segment elevation, tall precordial T waves, voltage of left ventricular • hypertrophy, partial right bundle branch block and left atrial strain.

The major abnormalities included low, flat or inverted T waves in left ventricular leads, low voltage, left axis deviation, frequent ventricular extrasystoles, complete right bundle branch block, first degree heart block, right ventricular hypertrophy, sinus tachycardia and ST segment depression. They

noted these changes but admitted that due to lack of standards for African population in Kenya, it was difficult to interpret the results with certainty. The ECG abnormalities however, were consistent with changes in alcoholic cardiomyopathy noted by other workers.

Because Rees and Chukwumeka investigated subjects whose alcohol^consumption and duration of drinking were not recorded; I decided to make a controlled study of drinkers whose quantity of alcohol consumption could be ascertained. They were compared to non drinking controls living in the same environment, with similar dietary habits, and whose physical excercises and stresses would be fairly comparable.

As already noted in the literature review most work done on alcohol associated cardiomyopathy has been done on subjects already in stage of cardiac decompensation and in most cases there were no controls. The aim in this particular study is to find out what preclinical changes would be noted among drinkers compared with controls from the same environment. Positive findings would possibly form a basis for caution 'and advise to heavy drinkers.

SUBJECT AND METHODS

The subjects examined (both drinkers and non-drinkers) were drawn from the country's Armed Forces (Kenya Army, Kenya Air Force and Kenya Navy). Armed Forces personnel are always recruited on a vigorous medical and physical fitness. All of them, therefore, begin their careers with normal cardiac functions. Their nutritional status is above average. Their working environment is comparable. They are generally better physically fit than equivalent general population.

A preliminary survey was done in which about five hundred soldiers were interviewed, and their medical records examined. Subject with histories suggestive of heart disease, chronic chest disease and hypertension were excluded from the study. Only subjects who were apparently normal but had a history of heavy drinking, were included in the study. The controls were from the same source, they had a history of never having drunk alcohol all their lives. Many of the subjects were not available for final examination and electrocardiographic recordings because of the nature of their duties, which took them out of their barracks, at the time of the final examinations, and ECG recordings.

History of the exact amount of alcohol taken was difficult to elicit. Many people do not keep a record of their intake of alcohol, and many more hid their intake, fearing some kind of official repercussion. Corroboration of estimated amount was taken from platoon and section commanders and their mess colleagues. Heavy drinkers are readily recognized by their commanders and their colleagues.

In this study, heavy drinkers are described as having an average daily intake of six half litre beers or an equivalent in spirits, for periods ranging from 5 to 25 years (120 gm of alcohol daily). Practically all the drinkers were beer drinkers. Controls were total abstainers for all* their lives. Eighty subjects were finally examined and had electrocardiographic recording (43 drinkers and 37 controls). A history was first taken from the subjects, concentrating mainly on cardiovascular system, to elicit symptoms of reduced heart function

palpitations, exertional dyspnoea, paroxysmal nocturnal dyspnoea, chest pains and oedema. The subjects were then examined, particular attention being paid to their cardiovascular systems. Pulse, blood pressure, apex beat, the jugular venous pressure and heart sounds were examined. Three subjects had diastolic blood pressures over 95 mmHg but their medical records showed no history of hypertension and they were included in the study. All other parameters were normal. The electrocardiograph machine used was a Hewlett-Packard 1500A, direct writing machine. The machine was callibrated so that 1 millivolt deflected the stylus 10 millimetres in height. The machine was regularly checked for technical faults. The recording speed used throughout was 25 millimetres per second.

Each subject had his ECG recording made when relaxed, lying supine on a couch. The recordings were made at least 12 hours after the last alcoholic drink and at least half hour after smoking the last cigarette. Care was taken in skin prepration with electrode jelly to ensure effective

contact. The standard twelve lead two plane (frontal and horizontal) scalar electrocardiography was recorded.

Because of shortage of echocardiographic papers, every fifth subject was selected for echocardiography. The echocardiographs were recorded from a Siemens Echopan K S, M Mode machine. The recording speed was 25 millimetres per second, as in electrocardiography, care was taken in skin preparation with electrode jelly to ensure effective contact. Attention was paid to the right and left ventricles and the septum to determine chamber sizes and wall thickness. Attention was' also paid to wall movements to elicit any reduction in wall mobility that would suggest reduced cardiac output due to reduced ventricular contractions. The same subjects chosen for electrocardiography were subjected to chest x-ray. The x-ray machine used was Phillips fitted with a chest stand placed 36 inches from the tube. Subjects were carefully centralised before pictures were taken, to enable a fair assessment of cardiothoracic ratios. Postero-anterior chest x-rays were taken in full inspiration.

The parameters analysed from the electrocardiographic recordings included:

1. Rate and rhythm, both atrial and ventricular rates;
2. PR interval, measured from the beginning of the P wave to the beginning of QRS complex;

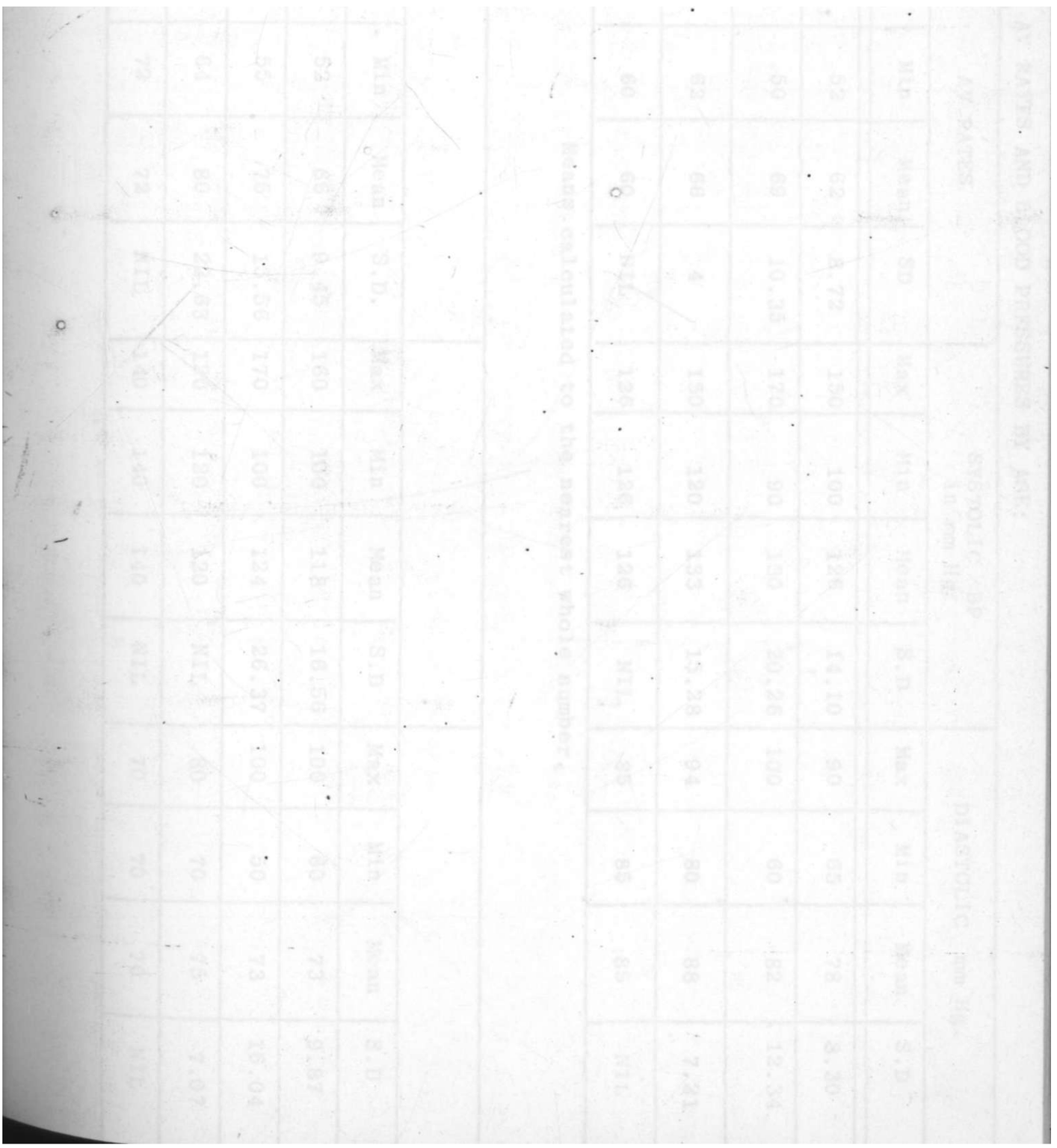
. The QRS complex, duration, direction and amplitude of the s and r waves and $s^{v}1+r^{v}5$. The QRS frontal axis using total voltages (s+r) in standard leads I and III.

The ST segment elevation or depression.

5. QT interval corrected for rate.

6. T wave direction and amplitude u wave presence or absence.

RESULTS - pages 13 to 16.



AV PATES. AND BLOOD PRESSURES BY AGE:

AGES	NO SUBJ.	AV PATES				SYSTOLIC BP in mm Hp:				DIASTOLIC mm Hg.			
		Max	Min	Mean	SD	Max	Min	Mean	S.D	Max	Min	Mean	S.D
20-30	13	80	52	62	8.72	150	100	126	14.10	90	65	78	8.30
31-40	26	100	50	69	10.35	170	90	130	20.26	100	60	82	12.34
41-50	3	70	62	66	4	150	120	133	15.28	94	80	88	7.21
51 +	1	60	60	60	NIL	126	126	126	NIL	85	85	85	NIL

Means calculated to the nearest whole number.

AGES	NO SUBJ												
		Max	Min	Mean	S.D.	Max	Min	Mean	S.D	Max	Min	Mean	S.D
20-30	14	82	52	66	9.45	160	100	118	16.56	100	60	73	9.87
31-40	20	98	55	75	15.56	170	100	124	26.37	100	50	73	16.04
41-50	2	96	64	80	22.63	120	120	120	NIL	80	70	75	7.07
51+	1	72	72	72	NIL	140	140	140	NIL	70	70	70	NIL

Table 1 and 2;

Table 1 shows the maximum, minimum and the mean for atrioventricular rates, systolic and diastolic blood pressures divided into age groups, for drinkers.

Table 2 shows the same for non drinking controls. A statistical analysis showed no statistically significant difference between the two groups in all parameters presented on these tables. Results discussed later.

TOTAL AMPLITUDE BY AGE (s^v1+r^v5)

DRINKERS

TABLE 3

AGES	NO SUBJ	s^v1				r^v5				(s^v1+r^v5)			
		Max	Min	Mean	S.D	Max	Min	Mean	S.D	Max	Min	Mean	S.D
20-30	13	28		11.5	5.94	37	11	20.9	6.44	52	21	32.5	9.01
31-40	26	25		13.7	4.81	29		18.7	5.62	49	15	32.4	8.56
41-50		16	10	12.3	3.21	32	13	21.3	9.71	42	29	3 7	7.23
51*			’0	9	0.0	17	17	17	0.00	26 -	26	26	0.00

Means expressed to the .nearest one decimal place.

NON DRINKERS

TABLE 4

AGES	NO SUBJ	s^v1				r^v5				(s^v1+r^v5)			
		Max	Min	Me an	S.D	Max	Min	Mean	S.D.	Max	Min	Mean	S.D
20-30	14	26		15.9	7.67	26	8	18.4	5.80	48	16	34.3	10.65
31-40	20	15		9.7	4.39	17	12	13.9	1.86	30	18	23.6	4.58
41-50		8		6.5	2.12	24		16.5	10.61	32	14	23.0	12.73
51+	1	14	14	14	0.0	12	12	12	0.0	26	26	26	0.0

to

Tables 3 and 4:

These tables show maximum, minimum and mean for s in $^{v}1$, r in $^{v}5$ and total amplitudes $s^{v}1+r^{v}5$. Table 3 for drinkers and Table 4 for non drinking controls. The tables are divided into age groups of subjects. An analysis of the two tables showed no statistically significant difference in total amplitudes between the two groups. Results discussed later.

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PR INTERVAL, QRS DURATION AND QRS AXIS BY AGE

A

t

DRINKERS

TABLE 5

AGES	NO SUBJ	PR INTERVAL				QRS DURATION				QRS AXIS			
		Max	Min	Mean	S.D	Max	Min	Mean	S.D	Max	Min	Mean	S.D
20-30	13	0.22	0.14	0.18	0.03	0.08	0.04	0.06	0.02	85	30	47	
31-40	26	0.20	0.14	0.17	0.02	0.08	0.04	0.06	0.02	90	15	56	
41-50	3	0.20	0.16	0.19	0.02	0.08	0.04	0.07	0.01	88	30	54	
51+	1	0.16	0.16	0.16	0.00	0.06	0.06	0.06	0.00	60	60	60	

NON DRINKERS

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AGES	NO SUBJ	PR INTERVAL				QRS DURATION				QRS AXIS			
		Max	Min	Mean	S.D	Max	Min	Mean	S.D	Max	Min	Mean	S.D
20-30	14	0.20	0.16	0.17	0.02	0.08	0.04	0.05	0.02	90	5	55	
31-40	20	0.24	0.16	0.18	0.03	0.08	0.04	0.05	0.02	75	5	34	
41-50	2	0.16	0.16	0.16	0.00	0.06	0.04	0.05	0.01	45	45	45	
151+	1	0.16	0.16	0.16	0.0	0.06	0.06	0.06	0.01	30.	30	30	0.0

Tables 5 and 6:

These tables show the PR intervals, QRS duration and QRS axis, table 5 for drinkers and table 6 for non-drinking subjects. An analysis of the two tables showed no statistically significant difference between the two groups on all the parameters analysed in the tables. All the QRS axes are within normal limits. Results discussed later.

ECHOCARDIOGRAPH AND CHEST X-RAY RESULTS

DRINKERS

SERIAL NO	AGE	LVC LVID	RVC RVID	SW	CTR
1	40	4.5	2.0	1	11.5:29 (1:2.52)
2	30	4.2	2.0	1	13.2.31 (1:2.42)
3	34	5.5	2.5	1.2	13.33.2 (1:2.55)
4	32	3.8	1.5	1.5	13.5:29 (1:2.15)
5	27	4	2	1.2	13:30 1:2.30
6	35	5	1.0	1.5	13.2:30 (1:2 27)
7	37	4	1.2	1.2	15:31.5 1 ;2.10

• *4* •

NON-DRINKERS

SERIAL NO	AGE	LVC LVID	RVC RVID	SW	CTR
1	29	5	1.5	1	13:29.5 1:2.27
2	43	4.8	•2.5	1.2	13.5:28 1:2.07
3	34	3	2.5	1.5	11.5:30 1:2.61
4	45	5.2	2	1.6	indistinct

LVID - Left ventricular internal dimension

RVID - Right ventricular internal dimension

XTR - Cardio-thoracic ratios

SW - Septal wall

Measurements are in centimentres.

Tables 7 and 8:

These two tables show the echocardiographic and chest x-ray findings. There are 11 subjects - 7 drinkers and 4 non drinking controls. Each subject has his age, left ventricular internal dimension in diastole, right ventricular internal dimension in diastole, septal wall thickness and. cardiothoracic ratio in chest x-ray.. One subject, seria.l no 4 in table 8 had poor chest x-ray results and therefore no CTR. The results show 4 subjects, 2 drinkers and 2 controls, with septal wall thickness above normal, at 1.5 cm. Otherwise, all results are within normal limits. Results discussed under echocardiography and chest x-rays later./

DISCUSSION

The latest classification of cardiomyopathies divides them into congestive or dilated, hypertrophic and restrictive. They are defined as a group of heart muscle diseases of no known aetiology. The other group of heart muscle diseases are called rare specific heart muscle disease. Their aetiologies are known usually associated with a generalised systemic disease such as amyloid heart disease. Alcohol cardiomyopathy has been defined by Goodwin in 1977 "as syndrome of congestive cardiomyopathy (cardiomegally, dilated ventricles and poor contractile function) associated with high and prolonged intake of ethyl alcohol with a tendency to remission when alcohol is discontinued". Although there is aetiologic association with alcohol clinical presentation and pathologic features are similar to other dilated cardiomyopathies. There is enough evidence that alcohol alters the metabolism of the heart muscle acutely, but there is great variability in development of cardiomyopathy among alcoholics. No specific triggering mechanism has been identified nor has any reason been given why, whereas in the acute stage alcohol depresses cardiac function in all drinkers and yet some chronic alcoholics develop cardiomyopathy while others do not.

In 1969 Regan[19] working with volunteers did cardiac catherization studies on drinking subjects and showed that acutely, after drinking, there was increased potassium and phosphate in coronary bloou. This shows that there is scute injury with leakage of intracellular potassium and phosphate into extracellular space. Continuous assault in

this manner over a long period would probably cause permanent damage.

Ettinger[20] and others, working with dogs, found that after prolonged alcoholic intake in large quantities, the dogs developed evidence of myocardial dysfunction as shown by ECG changes such as prolonged PR interval, widened QRS complexes and various bundle blocks.

Alcoholic cardiomyopathy, as all other dilated cardiomyopathies, has been characterized by non specific conduction defects, and enlarged ventricular chambers. These changes have been seen in patients who have been admitted to hospital in congestive cardiac railure or in acute dysrrhthmias after a heavy bout of alcohol as in Ettinger's study. Levi et al have shown that alcoholics have reduced left ventricular function. Regan's work also showed similar findings. It has been stipulated that chronic minor cellular damage results, after a long time into permanent damage. Brigden noted that the period before development of cardiomyopathy was at least ten years. In this study, electrocardiographic recordings of 43 heavy drinkers over a period ranging from four years to thirty years with a mean of 12.2 years have been analysed. A few echocardiographs and chest x-rays have also been analysed. They are compared with 37 teetotal controls. All subjects denied any history referable to the cardiovascular system, and their physical examinations were normal. An analysis of their electrocardiographs follows.

Rhythm:

All subjects, alcoholics and controls, were in sinus rhythm. Work already quoted have reported various dysrrhythmias

mainly in proven cardiomyopathy, usually in patients in hospital.

Rate:

The mean rate for alcoholics is 66 beats per minute (max100 min 50) and the mean rate for non drinkers is 70 beats per minute (max 98 min 52).

Twelve out of 43 (27.9%) of drinkers had sinus bradycardia, defined as heart rates of 60 beats per minute and below, and 33.3% of controls had bradycardia.

Statistical analysis of the heart rates between the two groups shows no significance (p 0.1) the mean rates' are within the accepted normal limits, but the higher incidence of. sinus bradycardia of about 30% (26 out of 80) compared with Okwera's[21] study in which only 2 out of 295 males is most likely to be a reflection of physical fitness of the soldiers in general. Previous studies have not shown any specific tendency either to bradycardia or tachycardia among drinkers until they reach stage of decompensation. Rees and others, found sinus bradycardia in 16 out of 135 (11.8%) in a study of pombe drinkers in Machakos Kenya. It is difficult to ascertain the significance of this finding as, there were no controls.

P Wave;

Wave of atrial depolarisation. The p wave duration is generally accepted as being not more than 0.11 seconds and not more than 3mm in amplitude.

In this study, all the p waves were within normal limits Brigden found notched or biphasic p waves, suggestive of left atrial hypertrophy in cardiomyophathies, his subjects were in congestive cardiac failure.

PR Interval:

Is the time the impulse takes to travel from sino-atrial node to the ventricular muscle fibres. Normal range is between 0.12 seconds and 0.22 seconds. The interval varies with body build and heart rate. Bigger bodies and slower * heart rates tending to have longer PR intervals. In this study a mean PR interval of 0.18 seconds was found among alcoholics (max 0.24 min 0.16) and a mean of 0.18 seconds (max 0.24 and min 0.16) among controls. There is no statistical difference between the two groups. One 34 year old individual in the control group had a PR interval of 0.24 seconds. The individual had a big body. He had no other cardiac signs. Rees and others in their study of pombe drinkers, found abnormal PR prolongation; described as first degree heart block among 6 out of their 135 subjects McDonald[22] and co workers found PR prolongation in 19 out of 46 patients with alcoholic cardiomyopathy. Other workers have also described evidence of atrioventricular conduction defects among patients with alcoholic and indeed other cardiomyopathies. Ettinger and co-workers working with dogs, found prolongation of PR interval in chronic alcohol drinking experimental dogs. It is significant to note that most PR abnormalities were seen among patients with proven cardiomyopathy. Brigden and subsequent workers have noted this in all kinds of cardiomyopathy. The findings would be expected in the pathology of cardiomyopathy which include cell injury and fibrosis.

In this study of 43 alcoholics and 37 controls, PR intervals were within normal .limits. It is normal even among° those

subjects who have been drinking heavily for over fifteen years.

QRS Complex:

Is the time the depolarisation impulse takes to travel through the total ventricular mass. In this study, the mean QRS duration among 43 alcoholics is 0.06 (max 0.08 min 0.04) and among controls was 0.05 (max 0.08 min 0.04.- Normal between 0.04 to 0.11, shorter or longer durations are indicative of intraventricular conduction defects. In this study both groups had durations within normal limits and there is no statistically significant difference between the two.
Rees and others found evidence of partial right bundle branch block in 1 out of 13, and complete right bundle branch block in 2 subjects. Hollister and Goodwin[23] and other workers who have studied alcoholic cardiomyopathy have found various evidence of various bundle branch blocks mostly among patients admitted with congestive cardiac failure (CCF). Ettinger found, among chronic drinking experimental dogs, evidence of bundle branch blocks, mainly left bundle branch block. It is reasonable to stipulate that by time these changes occur, definite damage has been done to the myocardium and cardiomyopathy has set in. In this study, chronic alcoholics had no evidence of intraventricular conduction defects.

QRS Amplitude:

Normal QRS amplitudes have a wide variation; r or s waves should normally be not less than 5 mm in standard leads I-III, and not less than 7mm in chest leads V_2 and V_5, and 5mm in V_1 and V_6. Scott's criteria for left ventricular hypertrophy

(LVH), the sum of s in V1 and r in V5 should not be more than 30mm, and not less than 12mm. The upper limit is taken in Kenya as 35mm. The amplitudes are affected by various factors such as body build, presence of fluid or air in the chest wall and height of individuals. In this study, the mean total amplitude s 1 + r 5) in drinkers was 32.35mm (max 52mm min 15mm) and 29.54 among controls (max 47mm min 14mm). Among the drinkers, 12 out of 43 had ECG hypervoltage, of these, three had diastolic pressures of over 95mm Hg but no medical record of hypertension, four out of the 12 had diastolic pressures of 80 mmHg or below, the rest are between 81 and 95. Among the controls 9 out of 37 (24.3) had ECGs of left ventricular hypertrophy. of all these controls, non had diastolic pressure above 95 mm Hg, the highest had a diastolic of 90mm Hg and the lowest a diastolic of 60mm Hg. There was no statistical difference between the two groups. In Okwera's study of 567 normal subjects 6% showed ECG evidence of left ventricular hypertrophy in normal healthy Kenyans, with no evidence of hypertension or heart disease. In this study, there is hypervoltage or ECG of LVH among 27.9% of drinkers and 24.3% among controls who had no clinical hypertension. This finding would suggest possible higher values for total amplitude among Africans than given in Scotts criteria. ReeS and others did not record this hypervoltage phenomenon. ECG recordings of left ventricular hypertrophy have been recorded by other workers studying cardiomyopathies. It is however, common in hypertrophic cardiomyophathies than in dilated cardiomyopathies where recordings tend to show low total

amplitudes. In this study, there is no evidence of right ventricular hypertrophy.

ST Segment:

Defined as the isoelectric phase between ventricular depolarisation represented by QRS and repolarisation, represented by T wave. It has been noted by workers among Africans, Asians and other races that the ST segment pattern in these groups tend to differ from those of Caucasians without evidence of any disease. In the African group, the ST Segment has been found to be elevated or mildly depressed in normal healthy individuals. In this study, 7 out of 43 drinkers (16.3%) had ST elevation of over 1mm but not more than 3mm. Six out of 37 non drinkers (16.2%) had similar changes. Workers on ECG changes in cardiomyopathies have generally reported non specific ST Segment changes.

T and U waves:

The T wave represents the duration of ventricular repolarisation. The significance of u wave is not known. It appears in states of hypokalaemia with loss of T wave, but its significance when it appears after normal T in normal tracings is uncertain. The "Bantu" T wave pattern is peculiar to Africans, they are characteristically broad based, peaked, tall T waves in precordial leads with no pathological significance. In this study more than 60% of both groups had this kind of T wave. T wave inversion is more significant and usually signifies ventricular strain and or damage. Brigden, Ettinger, Goodwin among others studying ECGs of cardiomyopathies have described various T wave changes mainly in patients in failure. Rees and

others, doing a similar study noted T wave changes (low, flat or inverted) in 19 out of 135 (about 15%). In this study, only one control had inverted T wave but with all other parameters normal. All others had normal T waves. U waves were noted in three drinkers, all of them in precordial waves V2 - V4 and in one control over the same area. As said earlier, no significance can be attached to them in the absence of any other abnormalities.

Echocardiographs:

Every fifth subject was randomly chosen for echocardiography. The same subjects had chest x-rays. A total of 11 subjects, seven alcoholics and four controls, had echocardiography. In echocardiography, attention was given to the ventricular chambers, taken at their broadest points.

Alcoholic cardiomyopathy, being a dilated cardiomyopathy, early dilation of the ventricles would be expected, even with normal ECG and chest x-rays. The upper limit of normal for left ventricular internal dimension in diastole as measured from echocardiograms is 5.7 cm," and the upper limit of normal for right ventricle is 2.5 cm. As can be seen from tables 7 and 8, both drinkers and controls had normal ventricular internal dimensions. One drinker and two non-drinkers had right ventricular internal dimensions on the upper limit of normal. There has not been study among the local population to give an average chamber size, and the standards quoted are from American and European studies. On the strength of those normals, these figures are within normal limits.

The upper limit for septal wall thickness in normal subjects is 1.3 cm (European standards). From tables 7 and 8, there

were two drinkers and two controls with septal wall thickening all of them at 1.5 cm. None of them have any other echocardiographic or electrocardiographic abnormality. There are no standardised normal among the local population, therefore, it is difficult to say whether the thickenings * are significant or not. The th.ickining is not confined to any one group - drinkers or controls. The sample chosen for echocardiography is also too small to make much inferences from. 1

The left ventricular posterior wall should not be more than 1.2 cm, (European standards). In this study, all subjects- had left ventricular posterior wall measurements within normal limits - (Tables 7 and 8). Among abnormalities noted by Demakis and other workers, were thickening of ventricular wall especially left ventricular walls. These were noted on those patients who died of alcoholic cardiomyopathy and had autopsies done. Since all of these studies were on c

' patients who already had cardiomyopathy it is not known at what stage ventricular wall thickening occurs.

The posterior ventricular walls and septal movements were normal and adequate. There was no reduction in ventricular wall movements. The mitral valves were all normal. The pericardia were also normal.

The echocardiographs of the 11 subjects can be summarized to have been, all within normal limits.

Chest x-rays:

Cardiothoracic rations were worked on the ratio of cardiac shadow to chest width, at the level of the diaphragmatic dome. The ratio of cardiac shadow to thoracic width should not be

more than 1:2. The cardiothoracic ratios in the 10 subjects, 7 alcoholics and 3 controls, were within normal limits. Of the 11 subjects chosen, one 45 year old control with a big fat chest had a poor chest radiograph, most likely due to his size and was excluded from the CTR study. He needed a-more penetrated x-ray to make a good study.

These 11 subjects therefore, had no cardiac enlargements detectable neither by echocardiography nor by chest x-ray.

These findings were similar to findings by workers such as Sakaran Asoken and others, and by Levi and others. They studied pre-clinical alcoholics and found no detectable cardiac signs by chest x-ray or ECG. On cardiac catheterisation studies, they, however, found evidence of reduced left ventricular function among most of the drinkers with no clinical signs.

CONCLUSION:

Fourty three heavy drinkers over a long period, were studied with thirty seven controls to find evidence of cardiac damage by alcohol over long periods. History from the subjects revealed no abnormal symptoms compared to controls. Both groups performed fairly comparable exercises with no evidence of cardiac decompensation among drinkers. Their physical examinations were equally normal and an analysis of their ECGS, echocardiographs and chest x-rays were all within normal limits. There is no difference between drinkers and controls. From these results it would be tempting to conclude that there is no cardiac impairement among our chronic heavy drinkers. Cardiac catheterization studies have shown that in apparently normal hearts of heavy drinkers, there is already left ventricular subnormality, usually raised left ventricular end diastolic pressure. These pre-clinical subjects were, however, not followed up to find out what percentage actually developed cardiomyopathy. It is possible that the subjects in this study already have left ventricular abnormalities, just as it is quite possible they do not. More sensitive or elaborate investigation, preferably non invasive, need to be done to measure their ventricular functions. With the more modern echocardiographic machines, this is possible. ECG, chest radiograph and echocardiographic studies need not be abnormal in hearts with early damage.

Not very much is known about cardiomyopathy, and although the association between alcohol and cardiomyopathy is known, no reasons for the very wide variability in

development of overt cardiac damage in some heavy drinkers and not in others.

What is the triggering mechanism? It does not appear to be the type of drink - it is as common among drinkers of spirits, wines and beer; but only a small percentage of these people develop cardiomyopathy. We would like to find out what role genetics, race, nutritional status and general physical fitness play in the causation of cardiomyopathy. The normal tracings in this study was possibly because these subjects are generally more physically fit and have possibly better cardiac functions. It would also be expedient to find out the role the viruses play. Dilated cardiomyopathy is a common enough problem among cardiac patients in Kenya, and the history of alcohol has been vague in the few that admitted it. To determine with authority what proportion of these cardiomyopathies are due to alcohol, we need a large prospective study of chronic heavy alcohol drinkers over a long period. Alongside this, research should be intensified to determine the triggering mechanism that makes some heavy drinkers develop cardiomyopathy while others do not. Is dilated cardiomyopathy more common among drinkers than non drinking population in Kenya?

In this study, the 43 chronic heavy alcohol drinkers have normal cardiac functions as compared to 37 controls from similar environment. The methods used might not have been sensitive enough to detect early changes of cardiac function.

I would recommend that: one, a large group of heavy alcohol drinkers be followed up over a long period to find

out the natural history of heavy alcohol intake over a long period in this country. I would suggest a follow up peri0cj of at least 10 years to find out what proportion of these people developed cardiac signs, what proportion developed cirarhosis, dementia, or died from road accidents. For this type of study, a stable population that can easily be

± •wed up wil] be necessary. Secondly, I would recommend

- 1:V3, i'V;r we get local echocardiograph standards from a no-.'marl herniation to compare our findings .with. At present we con pare our echocardiograph findings with results obtained else.v.'u re and these may not represent the true local pattern of c<' <i rdiographs.

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