"Evidence of Ischaemic Heart Disease in patients with chest pain as seen at Kenyatta National Hospital."

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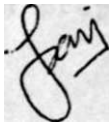
DECLARATION

This dissertation is the original work and has not been presented for a degree in any other University or any publication in any Journal or institution by the author.



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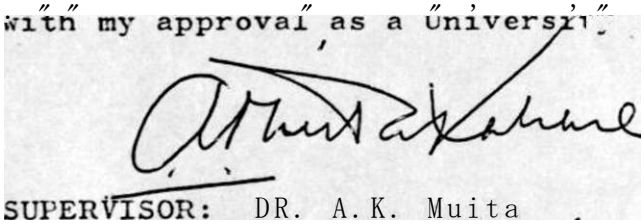
This dissertation has been submitted for examination with my approval as a University supervisor.



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ABBREVIATIONS USED IN THIS DISSERTATION

I.H.D.	-	Ischaemic Heart Disease.
M.I.	-	Myocardial Infarction.
E.C.G.	-	Electro Cardiogram.
E.S.T.	-	Exercise Stress Test.
B.P.	-	Blood Pressure.

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SUMMARY

The study comprised 52 patients vho presented at Kenyatta National Hospital with anterior chest pain which was. nontraumatic and noinfective in origin. It was cither suggestive of cardiac origin or was atypical in r.;:tnre i.e. of nonspecific origin.

The patients included in this study were all abovr the age of 30 years Kith an average age of year>.

There were 36 male and 16 female patients. Male; *i*:· the sixth and seventh decade of life were found to hr-vc a higher incidence of ischaemic heart disease as compared +0 females and younger males.

Sedentary life, hypertension and diabetes mellitus vcre not observed to be particularly associated with Ī':!.' in this f study. Cigarette smoking was also present in only 2??' of the patients with IHD. Hypercholesterolemia wrs not sought for because of the lack of facilities.

Six patients vere diagnosed to have M.I. either on the strength of ECG changes (four) or with significantly elevated cardiac enzymes (two)* One patient gave history typical of unstable angina and also had ECG evidence of it. One patient had a positive EST but had normal resting ECG. So, out of the total 52 patients included in this study, eight patients were diagnosed to have IHD.

REVIEW OF LITERATURE

·Ischaemia' refers to oxygen deprivation resulting from reduced perfusion. The term 'ischaemic heart disease' defines a disease spectrum of diverse etiology with the common factor being an imbalance between myocardial oxygen supply and demand.

Ischaemic Heart Disease is an old killer. The first evidence of this disease is available from the postmortem findings of an exhumed body, 2100 years ago in China. (1). William Harve (1600) was the first one to describe coronary circulation (2). About 150 years later, William Heberden (1768) described angina pectoris and noted that patients with symptoms of IHD may die suddenly (3). In mid 18th century, Morgagni noted deposition of calcium in the coronary arteries of men (4). Perry and Janner related this deposition to the symptoms of angina (5). Authorities like Quain and Rokitansky maintained that myocardium is only a seat of primary inflammation and degenerative changes, thus recognition of myocardial ischaemia remained submerged for decades (k).

In 18*》0, Williams noted a pallid, yellowish appearance of the heart and postulated that it is due to an altered state of nutrition, perhaps due to partial obstruction of coronary arteries (7)* Hall (1842) also supported this view (k). Carl Weigrt, at the turn of the 19th Century, recorded a complete pathological description of coronary artery disease and M.I. and identified their cause and effect relationship (3). Adam Hammer (1878) was the first clinician to diagnose coronary thrombosis antemortem which was later confirmed at autopsy (3). Leyde again mentioned frequent postmortem findings of coronary artery narrowing and/or thrombosis in patients with chest pain but his studies were largely ignored (11). Dock (1896) described clinical features of coronary occlusion followed by Baumgarten who published a book on 'infarction of heart'(12, 13). Physiologic processes involved in coronary atherosclerosis were described by Poster et al in 190*1. Despite all the information available then, Mathews and Miller (1909) maintained that 'coronary occlusion could only lead to sudden death and the diagnosis was possible only at postmortem* CO. James Harrick (1912) formulated the concept of coronary thrombosis and M.I. He also described clinical symptoms accurately. Smith, Harrick and Pardee (1918-1920) described T wave changes on ECG in cases of IHD.

Farkinson and Bedford (1928) described the complications of coronary occlusion (k). Following the recognition of IHD, various modes of management were introduced and mobile coronary care units were established in 1936 (6, 7, 8, 9, 10, 11).

As far as Africa is concerned, Akinkugbe (1972) mentioned that 1,100 postmortems were carried out in Africans in 193ˆ and no atherosclerosis was found in the thoracic or abdominal aorta. The same conclusion was drawn after 1,000 necropies in 1938 (12). Baldachin (1961) did not observe any case of M.I. in 70 consecutive Africans above 60 years of age (13). The first case of M.I. in a Kenyan African was reported by Ojiambo (1968), (1ˆi), followed by 8 more cases reported recently (15).

Ischaemic Heart Disease does not have an equal global distribution and it does not affect all ages and sexes equally.

The Western world is affected the most. In 1964, the male population of age group 45-5ˆ years had a death rate of 442/100,000 population from IHD in Finland, followed by USA and Scotland with death rates of 359/100,000 and 354/100,000 respectively. United Kingdom had a death rate of 254/100,000 in the same year followed by Canada, Australia and North Ireland.

Japan had the lowest death rate of 51/100.000 in the same study (16). The same trend in the Western world wns noted by Akinkugbe in 1970 (12). Long (19B0) found that every year, 650,000 patients die of IHD in USA and about 1,000,000 new or recurrent cases of M.I. are reported every year (17). Mathur et al reviewed the available literature on incidence of IHD in India. It was reported to be less than 1/1000,000 population / year (13).

Ischaemic heart disease is a disease of the middle aged and elderly people. The incidence is highest in the sixlh decade of life followed by the fifth and seventh decades and then the ftnrrth and eighth decades of life (lo).

Enos et al found gross evidence of coronary artery disease in young American soldiers killed in the Korean war (19) Progression of atheroslerosis is fast in pre-disposed males in the third decade and so IHD becomes quite common in middle age and thereafter.

IHD mainly affects males and post-menopausal females (18). In the age group 15 to 39 years, males have more extensive coronary atherosclerotic lesions and they progress faster than in young females (20).

In Kenya, Shaper has found that IHD is a major cause of death in people of Indian origin. It was found to be almost non-existent in the African population (20). Asians in East Africa were found to have poor fibrinolytic activity as compared to the Africans. This may lead to enhancement of atherosclerosis and thrombus formation.

Among the Kenyans, the Masai have been known to have very low incidence of HID. They can suppress endogenous cholesterol synthesis by $0% in response to the large dietary intake. This may be regarded as a long term biological adaptation. It may be a genetically transmissible trait unique to the Masai (20).

Myocardial ischaetnia occurs because of an imbalance between oxygen supply and demand. Oxygen supply to the myocardium is decreased due to coronary atherosclerosis in *90%* of cases. Inadequate collaterals, reflex spasm or a fall in B.P. can also do the same. Oxygen demand is increased in exercise and excitement. It also increases by an increase in the work of heart or due to an increased oxygen consumption.

As atherosclerosis is the most important cause of IHD, risk factors related to it have been studied.

The major risk factors are hypercholesterolaemia, hypertension, cigarette smoking and diabetes mellitus. The Framingham study has shown that cigarette smoking, cholesterol level of more than 250 mgˆ’ and a systolic B.P. greater than 160mm of Hg, all combined, increase the frequency of IHD to 5》35 times that of expected while the presence of any 2 of them increases it by 2.03 times and only one factor will increase it by 1.02 times (21). There are several other studies supporting the above findings (17, 20, 22, 23, 2*》).

Hypertensive patients develop coronary artery sclero-sis at a younger age.

They have a 2 to 3 fold **increase in the risk** of developing IHD according to **some workers (17,** 25)· Hypertension causes endothelial **damage** and increases filteration of lipids from plasma, thereby enhancing atheroma formation. Good control of B.P. decreases the risk of IHD (16).

Diabetes mellitus increases the incidence of precocious atherosclerosis. Angina and M.I. occur at a younger age in the diabetic patients. 50% of diabetics die of IHD (22) The control of blood sugar has no effect on the course of IHD (16, 22, 25, 26).

Cigarette smoking increases the risk of IHD 3 fold (13). It also causes vasconstriction by increasing release of epinephrine and norepinephrine. Risk is dose related. Stopping smoking reduces the risk of developing IHD. Smoking increases the risk of sudden death 5 fold (17, 26).

Other minor risks factors that have been described for IHD are old age, male sex, positive family history of M.I. or sudden death, type A personality, hyperuricaemia, overweight and sedentary life (2, 13, 16, 17, 22, 26, 27). Apart from atherosclerosis coronary artery spasm may also be an important contributory factor in the causes of angina pectoris and M.I.

Clinical diagnosis of IHD is the first step, followed by investigations to support it. Resting 12 lead ECG is very important in diagnosing acute or past M.I. It may be entirely normal in 51° 6 cases of angiographically proven coronary artery disease (28).

In cases of doubt, an EST should be carried out to increase myocardial demand for oxygen. ECG tracings should be done during and immediately after exercise. The specificity and sensitivity of EST have been estimated to be about 86-97y£and 5ˆ-80ˆ. respectively; its predictive value was found to be 87-96% by Fortuinet [a]I (29). Positive EST has been defined as 'Horizontal or downslopping ST segment depression of more than 1mm 0.08 sec. after J point during or immediately after exercise* (30)》 There are many false positive and false negative responses depending upon the prevalence of the disease in the population, sex of the patient, drug intake, electrolyte imbalance and presence of cardiac abnormalities (31, 32). Other methods of diagnosis are radionuclide scanning with Thalium 201 and technicium - 99 imaging, radionuclide ventriculography and cardiac catheterisation. Cardiac enzymes 1.e. LDH isomers, CPK MB, SGOT are also helpful in diagnosing acute M.I., if taken serially.

AIM'

This study was carried out to detect and describe some cases of ischaemic heart disease among the patients presenting with chest pain at Kenyatta National Hospital.

MATERIALS AND METHODS

Materials

This prospective study comprised 52 patients, THey presented to Kenyatta National Hospital (K.N.H.) during the years 190ˆ and 19?5 with anterior chest pain. These patients were referred upon request, to-'the cardiology department. They were reassessed by the author and appropriate patients were selected for the study using the following inclusion criteria.

1) The patient should be above 30 years of age.

2) The chest pain should either be suggestive of M.I., angina pectoris or it should be of non specific origin i.e. excluding the patients with chest pain of infective or traumatic origin or when the etiological factor like dyspepsia was suggested on careful history taking.

Methods

A proforma (Appendix 1) was filled, which comprised the personal data, characteristics of chest pain, past medical history, family history, social history and brief psychiatric history. After taking a thorough history, patients had a full physical examination and then they were investigated by some available, non invasive methods to identify the existence of IHD.

All the patients had a posteroanterior chest x-ray. Cardiothoracic ratio was measured to detect cardiomegaly. Other abnormalities, if present, were also recorded. A 12 lead resting ECG in supine position was done in all the patients. A Q wave which was slurred, broader than 0.0*1 sec. and deeper than 25?£ of the R wave on the same lead was taken as an evidence of acute or old M.I. ST segment elevation with reciprocal ST segment depression in opposite leads was taken as an evidence of acute M.I., in the presence or absence of associated Q wave.

An. EST was done on a treadmill using the Bruce Protocol (Appendix II). t was done in all the patients with chest pain unless there was a specific contra-indicntion. It wa's not done in patients with evidence of acute M.I., overt congestive cardiac failure, diastolic D.P. more than 110mm Hg and multiple premature ventricular beats. Patients who refused to give consent were also excluded from the EST. An ECG tracing was recorded initially with the patient standing and hyperventilating afVer careful placing and fixation of electrodes.

Vˆ vas used for constant monitoring and recording of ECG during and after the exercise. Patients were explained about the test and were told to report all the symptoms - chest pain, fatigue, breathlessness, dizziness, pain in the legs etc. occurring during the exercise.

Before starting the exercise, a verbal consent vas taken and it was ensured that the patient had not taken any B-blocker on the day of the test. All the ESTS were done in the author's presence and resuscitation equipment were kept ready during the test. The heart rate and B.P. were recorded before the test. The EST was stopped if the patient complained of the chest pain, fatigue, pain in legs, breathlessness, dizziness or if ST segment depresion was seen. Positive EST was taken as a downward sloping or*horizontally displaced ST segment of 1mm at .OP sec. from the J point on ECG. Tracing showing isolated ST segment depression or ST segment elevation were taken as responses of doubtful significance. Any other abnormality, if present, was recorded.

Lipid profile and cardiac enzymes were not done in any of the patients at Kenyatta National Hospital due to lack of facilities but three patients with M.I. got them estimated elsewhere. Random blood sugar was checked in all the patients with IHD to find out the possibility of co-existing diabetes mellitus. It was not done in one patient with M.I. vho died on admission.

RESULTS

Ase

The selected patients were all above the age of 30 years. Average age of the whole study group was ˆ7.2 years, the range being thirty to seventy two years. Patients with M.I. had an average age of sixty two years, the range being fifty four to seventy two years.

Table - 1

Age in Years	Total No. of patients and per-centages (?.')	Patients with M. I.
31-40	20 (h 2 %)	-
41-50	13 (25%) ·	-
51-60	12 (2 2 %)	3
61-70	6 (11%)	0
> 70	1 (2≪0	1

Sex

36 patients were males, six of them being patients of M.I. Sixteen patients were females.

Table - 10

Occupation	Total No. of patients.	Patients with M.I.
Manager	1	-
Civil Engineer	1	-
Businessmen	7	
Teachers	5	-
Clerical Officers	8	1
Priest	a	.
Laboratory Technician	2	-
Student	1	
Nurse	1	

First three categories of people were from high income group while the rest were middle class people.

Table - 10

*

Occupation	Total No. of patients	Patients with M.I.
House wives	5	-
Cleaner Messenger	7	
Labourers		
Vegetable sellers	3	-
Drivers	2	2
Farmers		f
Fireman	1	1

All the above people are from the low income group doing manual work.

Table-10

Site of radiation of pain	Total No. of patients; percentages (%)	Patients with M.I.
Left arm	[illegible] (950	3
Dack	C (17%)	-
Right side of chest	6 (13%)	1
Abdomen	5 (9%)	-
Neck	1 (2%)	-
Jaw	1 (2%)	-
No Radiation	7 (15%)	o
More than one site	20 [illegible]	-

Table - 5

Charac ter of pain	Total No. of patients· percentages (%]	Patients with M.I.
Crushing or constricting	20 (39%)	’l
Burning	Ik (28%)	-
Tiercing or sharp	5 (9%)	-
Dull	5 (9%)	-
Cutting	2 (k%)	1
Throbbing	1 (2%)	-
Not specific	6 (13%)	1’

Table - 10

Precipitating factors.	Tet.il number ®f pp.ti cnts/pcrcen-tagcs(‴)	Patients with M. I.
Exercise	30 (62%)	3
Rest	7 (1M0	1
No thing particular	9 (17%)	2
Worry, anxiety	5 (9?0	-
Food	1 (2%)	-

Table - 10

Relieving fac tors.	Total No. of patients, Per-centages (?')	Patients with M.I.
Rest	[illegible]	
Analgesics	12 (23?0	-
Nitroglycerin	2 (h%)	
Not specific	11 (22%)	
None	6 (12*)	6

Table - 10

Frequency of pain	Total No. of patients;perccn-t.-es (?')	Patients with M.I.
Daily	18 (35%)	1
Weekly	5 (9%)	-
Non specific	20 [illegible]	o
Infrequent	7 (1W)	1
First episode	2 [illegible]	2

Table - 10

Intensity of pain	Total No. of patient ^, percent ages	Patients with M.I.
Severe	31 (61?0	6
Moderate	10 (35?0	-
Mild	2	-
Vague		-

Table - 10

Dura tion of pain	Total No. of patients/percen-taces (‴)	Patients with M.I.
up to 60 tnins.	28 (5 W	6
up to zt hrs.	15 (2 9 %)	-
Not specific	9 (1750	-

Table - 11

Total duration of illness	Total No. of patients/per-ccntnrcs (?Z)	Patients with M.I.
1 year	29 (57%)	
1-12 months	19 (38%)	k
weeks	3 (6%)	
under one week	2 (k%)	2

Table 12

Complaints	Total No. of patients	Patients with K.I.
Palpitations	6	0
Sweating	5	1
Headache	2	-
Weakness		-
Dyspnoea on exertion	3	-
Cough		-
Nausea, Vomiting	5	
Body ache	3	-
Loss of appetite	2	-
Insomnia	2	-
Pain in knee joint		-
Ankle swelling	2	-
Intermittent claudication	1	-
Mix complaints	11	3

Post Medical History

Two patients with M.I. had positive ECG evidence of M.I. in the past, while one patient gave a history suggestive of M.I. but no ECG evidence was available in him.

38.5% out of these 52 patients were known hypertensives on treatment. One of them had pre-eclampsia but had no hypertension at the time of this study. These twenty patients included three patients with M.I.

Three patients were known cases of diabetes mellitus, one of them had M.I. and one had features of unstable angina. All of them were on treatment with chlorpropamide

Family History

None of the patients gave a positive family history of IHD and / or sudden death.

Four patients gave history of diabetes mellitus in their first degree relatives; three had a diabetic father, one had a diabetic brother. Only one of these four was a known diabetic.

Eight patients gave a history of hypertension in their first degree relatives but only -j $_{G}$f >tbese eic was a hypertensive patient.

Table - 1? Social history.

No. of Cigarettes	Tot.nl No. of patients, .·percenta-	Patients with M.I.
<10	8 (15%)	1
11 - 20	7 (13° /»)	1
y 20	1 (2 %)	-
Uncertain	3 (6 %)	-

Six patients including two M.I. patients were still smoking at the time of study. Thirteen patients had stopped smoking prior to the study. Six of these thirteen patients had stopped smoking for less than one year while seven other patients had stopped for more than a year. Fifteen out of nineteen patients who were either smokers or exsmokers, smoked for more than one year.

Physical Examination

13.5% patients were obese. They had 20% excess over their ideal weight as per 1983 metropolitan height and weight chart. Two of these seven patients had M.I. and one had features of unstable angina. Rest of the patients were of average height and weight.

Cardiovascullar system

All the patients had a pulse rate ranging between 60 to 120 beats/minute. None of them had an apex pulse deficit. The blood pressure w^s found to be elevated in the range of 130/100mm of Hg.to220/160mm of Hg at the time of first examination in 15 patients 3 of them being M.I. patients. One patient with M.I. had a B.P. of 90/60mm of Hg on admission. The rest of the patients had a normal B.P. with an average of 125/82mm of Hg.

Two patients had a pansystolic murmur at the apex, one of them being an M.I. patient.

Two patients had features of congestive cardiac failure.

fo* t《ro anterior Cheat X-ray	Total No. of Patients, percentage (·')	Patients vith M.I.
Ktrul	40	1
C《r4loa*ply	10	3
Not don*	2	

Tebl· - 15

P».tlnf 12 lead ECG	Total No. of patients. per-centages (?")	Patients vith M.I.
Abntmal	18 (35?0	6
KOHMI	3'i (68?i)	

Table - 16

Abnormalities found in ECG.	Total No. of patients.	Paitnets with M.I.
P wave	-	I
Pathological Q wave	2	2
ST segment elevation	0	2
Left bundle branch block	1	·
Complete atrioventricular block	1	-
Multiple premature ventricular contractions (PVCs)	o	1
Left ventricular hypertrophy	2	
T wave inversion	₂	1
Bradyc ardia	1	-
Mixed changes	⁾	

Exercise Stress Test:-

Twenty eight patients were exercised.

Table - 17

Reasons for not performing EST	No. of patients
Myocardial infarction (proven or suspected)	9
Diastolic pressure ^ 110mm Hg.	3
Overt congestive cardiac failure.	2
Multiple ventricular extrasystole .	2
Refusal to give consent .	3
Did not turn up for follow-UP.	2
Died before EST.	3

Only two patients reached stage five of the Bruce protocol. Eight patients stopped in stage four and eight patients stopped in stage three. Ten patients could not do more exercise than stage one only.

Only three patients achieved the target heart rate i.e. 220-age in years. Twenty patients achieved 80% of the target heart rate. Five patients could not achieve even 80% of the target heart rate. All the patients had a rise in B.P.

The range was 150 to 2*》0 mm Hg systolic and 70 to 110mm Hg diastolic, with an average of l'iO/P-7 mm Hg . None of the patients had a fall in B.P. during or immediately after exercise.

The double product i.e. achieved heart rate x achieved systolic D.P. was found to be in the range of 16,000 to 38,ˆ00 in all the patients exercised. The patient with a positive EST achieved double product of 30,000. Five patients with chest pain but no ST segment depression achieved double products of above 16,000.

Table - 18

Reasons for stopping EST before target hpˆirt rate was achieved!-

	No. of patients
Fatigue in legs	14 (27%)
Chest pain	6 (12%)
Pain in the legs	3 (6%)
Dizziness	1 (2%)
Palpitations	1 (2%)

Only one patient developed an isolated ventricular ectopic beat in the second resting minute after stopping exercise bccausc of fatigue and hotness in the chest.

One patient showed a horizontally depressed ST segment of 3mm which was 0.08 sec. from J point along with chest pain, denoting a positive response.

One female patient showed some ST segment depression in the third resting minute, most likely a false positive response. One patient showed ST upsloping along with chest pain during exercise. No ST segment changes were seen in the remaining twenty five ESTS.

No Q waves were seen before or following EST in any of the patients.

Five patients developed chest pain during EST without any associated ST segment changes. The pain had characteristics of angina in all of them.

Random blood sugar was found to be normal in five patients with M.I. The patient with a positive; EST also had a normal random blood sugar. The patient with unstable angina was a known diabetic on treatment.

Lipid **profile revealed normal Fredrickson typing in** 3 patients **with suspected M.I.**

Cardiac enzymes (lactic dehydrogenase, creatinine phosphokinase and amino-transferase) were raised in two patients with M.I. find they vere normal in one patient with M.I.

From the above results it is evident that there were eight patients with IHD (according to the criteria used in this study) out of fifty two selected patients. Six of them had ncutf M.I. and one patient had a positive EST on ECG criteria. One patient had a history suggestive of unstable angina and also had ECG evidence of it.

DISCUSSION

IHD is mainly a disease of the Western World. This study was undertaken to identify and describe some cases of IHD seen at Kenyatta National Hospital. No prospective study has been carried out so far to document evidence for IHD in Kenya. So the results of this study are not compared with any other study done Kenya.

IHD has a broad spectrum of symptoms ranging from asymptomatic heart disease to sudden death. This study comprised fifty two patients who presented to Kenyatta National Hospital with non-traumatic and non-infective chest pain.

The mean age of patients with M.I. was sixty two years. The patient who showed a positive EST response was fifty seven years old. These findings are in keeping with findings of other series where IHD was found to be a disease of middle aged and elderly people (18, 19).

All the patients with IHD were males which is in keeping with the sex incidence of IHD.

Premenopausal females are relatively at a low risk because of the hormonal influence and a protective effect of high levels of high density lipoproteins. (18, 20, 22). Three patients with M.I. were doing sedentary work while three were manual labourers. Sedentary work is a known minor risk factor for IHD. People who do exercise are generally physically fit with good coronary callateral circulation and enlarged coronary blood vessles. (20, 22). But in this study even manual labourers formed 50% of the patients with M.I. As Kenyatta National Hospital usually serves people with low income, this findings can be explained on that basis. Jaggi O.P. also found a similar trend in India (27).

Characteristics of chest pain:

A myocardial Infarction

The pain of M.I. has been described classically as heavy, squeezing, crushing, or deep visceral pain in the central part of anterior chest and / or epigastrium; In this study, all the patients with M.I. described the pain to be retrosternal or substernal in origin.

Four of them had classical crushing or constricting pain, one had cutting pain while the remaining patient was non specific about the character of pain.

Sixteen other non M.I. patients also described the pain as crushing or constricting in nature and so, although this nature of pain is suggestive of the diagnosis, it is not pathognomonic of M.I.

Patients with M.I. described the pain radiating to the left arm, back, right side of the chest or nowhere, lˆi other patients also described the pain radiating to the left arm and so, it should be remembered that 'all chest pains radiating to the left arm do not originate in the heart.» (33)·

«

The pain of M.I. is one of the most severe pains and was described to be so by all patients of M.I. in this study.

Table - 8 shows the frequency of chest pain in patients with M.I. It can be the firsˆ manifestation of existing IHD or it may be preceded by several attacks of angina pectoris before the actual infarction occurs. The frequency of chest pain in M.I. can be any where between 'never before' to ˆdaily pain', which was found to be true in this study also.

As shown in table - 10 the patients with M.I. had pain lasting for up to sixty minutes in all cases and this is in keeping with the classical duration of M.I. pain.

M.I. can occur after exercise or even at rest. Evans et al found that 48% of M.I. occurred during natural activity including walking followed by 27% at rest, only 2% of M.I. occurred after severe exertion (18). In this study, one patient with M.I. described pain occurring at rest. Five other patients with M.I. described exercise as a usual precipitating factor in the previous episodes of chest pain, suggesting that these patients may have had angina pectoris.

These findings arc difficult to interpret as some patients were mixing up past episodes of chest pain with the present one in answering questions about precipitating factors.

As shown in Table - 7; all the patients with M.I. had no specific relieving factors. Three patients with M.I. described rest and/or nitroglycerin as relieving factors in their past episodes of chest pain. The pain of M.I. usually requires very powerful analgesics e.g. narcotics to get relief and allay the associated anxiety.

M.I. is known to be associated with symptoms like nausea, vomiting, dizziness, breathlessness, excessive sweating, sense of impending doom, confusional state, syncope, and profound weakness. These symptoms may occur in the absence of classical chest pain also. In this study, as shown in table 12, all the patients with M.I. had one or more of the above listed symptoms along with anterior chest pain.

D Angina pectoris

The chest pain of angina pectoris has been described as discomfort, pressure, heaviness, smothering, tightness, choking or a squeezing sensation in the anterior chest wall which is aggravated or precipitated by exertion or emotion, coming on during physical or emotional stress, anger, fright, hurrying or sexual activity. It may depend upon the time of day, being more common in the morning. It occurs with unaccustomed tasks rather than routine work requiring equal amount of effort.

It may radiate to the neck, jaw, throat, back, shoulder, arm or abdomen. It is relieved typically by rest or nitroglycerin.

In this study, two patients with T wave inversion on resting ECG described the chest pain which was typical of angina pectoris as described above. Pain was

radiating to the left arm, crushing or burning in nature, precipitated by exertion and relieved promptly by rest. It was described as frequent, almost daily pain with moderate intensity and lasting for a few minutes to an hour.

One patient with T wave inversion on the resting ECG gave a history suggestive of unstable angina. He had anterior chest pain on minimal exertion or even at rest, radiating to the left arm, constricting in nature, not relieved promptly by rest. It was recurrent in nature for a duration of one month.

The patient with a normal ECG but with ST depression on EST, described the chest pain radiating to the left arm, sharp in character, occurring even at rest with no specific relieving factors. It was moderate in intensity, lasting for two to three minutes and occurring intermittently for the last twenty years.

Five patients developed chest pain on EST. They gave history suggestive of angina pectoris with crushing or non-specific character of chest pain, radiating to neck, back or left arm.

Precipitating factor was exertion or anxiety in all these patients with rest as a relieving factor. It was described as a pain of moderate severity lasting for a few minutes. Total duration of the pain was ranging between two months to four years with frequenc; described as often. These patients may be suffering from IIID.

All the patients with evidence of IHD i.e. M.I. and angina pectoris, gave a history of chcst pain suggestive of cardiac origin, thus history taking is very important to establish a clinical diagnosis of IIID.

Atypical chest paiii

It is a pain of non specific origin. It has been described as 'stabbing in character,'accompanied by exhaustion, dyspnoea, deep sighing respirations or dyspepsia'. It lasts for only a few seconds and is not relieved instantly by rest or nitroglycerin. These patients may not be suffering from IEID.

From the remaining forty four, patients with anterior chcst pain but no evidence of IHD twenty five patients gave a history suggestive of angina but did not have all the classical features of it. As can be seen from the tables of results (tabic - 3 to table !1) these patients had varying types of pair, with varied radiation, precipitating and relieving factors,

intensity, duration and frequency. It is difficult to reach a conclusion about the etiology of their chest pain on history alone as this can be very varied and subjective in nature. But this pain is most likely non-cardiac in origin. These patients may be suffering from anxiety neurosis.

A patient with a past medical history suggestive of M.I. or angina pectoris is obviously at a greater risk of developing an acute M.I., as the underlying pathology, i.e. atherosclerosis, becomes worse with time.

In this study, only two patients with M.I. had previous, proven attacks of M.I. So, although posit past medical history of angina is helpful, it mcynot be available in all cases of IHD.

Risk factors

Family history, personal history and social history were taken in detail to identify the presence of hypertension, diabetes mellitus, cigarette smoking and history of IHD or sudden death in the family and in the past.

Hypertension is said to 'run in families'. In this study only 12.5° 》 of patients with positive family

history of hypertension were found to be hypertensive. Out of total twenty hypertensive patients, only 25ˆ had an evidence of IHD.

Diabetes mellitus has a multifactorial inheritance. Genetic predisposition is aggravated by environmental factors for decompensation of carbohydrate tolerance. It is known to cause precocious atherosclerosis and juvenile onset diabetics develop coronary insufficiency at a younger age than the general population. (16, 27》 30). M.T. can unmask latent diabetes also (61). In this study, four patients gave a history of diabetes mallitus in their first degree relatives but only one of these four had diabetes.

None of the patients with M.I. had abnormal levels of random blood sugar. But the patient with unstable angina was a known diabetic patient. So, although diabetes mellitus was not seen as an important risk factor, no conclusion can be drawn from this because of the small number of the patients involved.

The other major risk factor is cigarette smoking. Heavy smokers for a long period of time are at a greater risk for IHD than non smokers (2*1, 30). In this study, only one patient was smoking about twenty cigarettes/day since last ten years but he did not have any evidence of IHD by the non-invasive

methods used in this study. As shown in table - 13 only two patients with M.I. were smokers and smoking may be a contributory factor in enhanced atheroselerosi and IHD in them, but they were not heavy smokers.

Lipid profile studies were done in only three patients with suspected M.I. which showed normal Fredrickson typing. An increase in the Low Density Lipoprotein (LDL) cholesterol level or a decrease in High Density Lipoprotein (HDL) level is a very important risk factor for IHD. High levels of HDL are protective against IHD (63, 65)· Several workers have established normal lipid levels for Kenyans. (3ˆi 35, 36) Lore W. et al have also shown an increase in total lipid triglycerides in adult hypertensive Kenyans (37). Because of lack of data, it is not possible to comment upon the 'role of hypercholestero-lemia in patients with IHD in this study.

A family history of IHD is also an important risk factor. It has been found that sudden death due to IHD in the first degree relative increases the risk of IHD three fold for males and five fold for females under the age of fifty five and sixty five years respectively, as compared to the general population (30). In this study, all the patients denied history of sudden death or IHD in their family members. This may be because of low prevalence of HID.

It may also be explained by lack of diagnostic facilities available to the indigenous black population, staying in rural areas. Lack of knowledge about IHD in the general population and low index of suspicion on the part of medical workers may also contribute to it.

Type A personality has been associated with increased incidence of IHD (17). M.I. patients in this study did not reveal this type of personality.

Patients with anxiety neurosis may complain of chest pain along with a host of other complaints. 75% of patients included in this study, gave history suggestive of anxiety neurosis. In one study it was found that 10-14% of patients seen by a cardiologist every year suffer from anxiety neurosis (39). Since the symptoms frequently focus on the cardiovascular system, it is referred to as Neurocirculatory or Vasoregulatory asthenia. (24, 40). In another study, of patients suffering from l《ft inframammary pain were found to have no cardiovascular disease while only 22% had some form of cardiovascular pathology. Thus Backer Concluded that, a patient suffering from this type of pain is more likely to be normal as far as heart condition is concerned (41).

Obesity is a **known** risk factor for **IHD** with its associated sedentary habits, lack of physical exercise and diet rich in cholesterol (16, 1?). Obesity increases the work load on the heart. It also leads to generalized fat deposition which may in turn lead to enhanced atherosclerosis in the coronary arteries. Carlson et al found that the weight/height index did not have any effect on the occurrence of HID. He suggested that the relationship between hypertriglyceridaemia and IHD was not an effect of obesity *(k2)*. In this study, two patients with M.I. were obese. As no local figures are available, the American standards have been used to define obesity. Obesity with its associated sedentary living habits may be a contributory factor towards M.I. in these patients.

The cardiovascular system examination in cases of IHD may be normal. It may show features of etiological factors of angina pectoris e.g. aortic stenosis. Hypertension can be detected as well. In this study, out of the twenty hypertensives, five were shown to have IHD. Shearing forces due to uncontrolled hypertension lead to endothelial damage and thereby enhance atheroma formation.

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The patients with M.I. may have varied signs depending upon its complications. As has already been shown in the results, patients with M.I. developed signs of congestive cardiac failure, hypotension and mitral regurgitation in keeping with the usual complications of M.I.

Postcro anterior chest x-raj- was done in 50 patients to rule out chest infections and abnormalitie of the spine. I11 this study, 40 patient/G had normal chest x-ray which included 3 patients with M.I. Hence normal ch est x-ray does not exclude the diagnosis of M.I. Three M.I. patients showed cardiomegaly which may be due to the left ventricular hypertrophy secondary to hypertension. Seven other hypertensive patients also had cardiomegaly. Two patients with suspected M.I. died on admission so no x-rays were taken.

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Posting 12 lead ECG is an important investigation in detecting an old M.I. as it may show pathological Q waves with or without associated ST - T wave changcs. ST segment displacement returns to the baseline after an acute injury has resolved, and so it may not be seen in patients with old M.I.

In a case of acute M.I., the ST segment is elevated in the leads facing the infarcted area and is depressed in the opposite leads.

In this study, four patients were diagnosed as having M.I. on the strength of a resting 12 lead ECG. It showed the abnormalities listed in Table - 15 of the results.

Ischaemia is manifested by changes in T wave but they are difficult to interpret as inversion of T wave may also occur in myocarditis and pericarditis which also present with chest pain. In this study, nine patients had T wave inversion on resting 12 lead ECG. Six of them were suspected to have M.I. on the basis of history, but they did not have ST elevation or pathological Q wave. Ischaemia was suspected in three patients on the basis of a typical history of anginal pain along with T wave inversion on the resting ECG.

As outlined in table - 13 some other abnormalities were also seen on ECG which were suggestive of complications of M.I. e.g. left bundle branch block or complete heart block. But as the ST segment elevation and/or Q wave suggestive of M.I. were not present they were not accepted as cases of M.I.

ECG may be normal in about 51% of cases with angiographically proven IHD (28). It has been found that there may be 50 to 70% occlusion of the coronary arteries without any symptoms or ECG changes of myocardial ischaemia (43). So, a normal ECG does not rule out the existence of HID. In this study, one patient with a normal resting ECG had a positive EST indicating the presence of IHD.

An ECG at rest can be normal but cardiac decompensation occurs with exercise and an ECG taken during or immediately after exercise may show features of ischaemia.

An EST is a relatively safe procedure. No mortality and morbidity requiring hospitalisation was observed in the patients studied. Fortuin et alhave reported 0.01% mortality and 0.2% morbidity on EST. (29). In another large study of 102,000 stress tests of all types, only 7 M.I. leading to four deaths have been reported (kh).

Indications for EST in this study were limited. It was done in the patients with atypical chest pain or history suggestive of angina of effort.

In some ccntres, patients ≫-ith M.I. are exercised after nn average of twelve days following the event, ('15). They use a modified protocol to document the functional capacity of the heart. In this study, no EST was done in the pr.tien ts with M.I. as the study is directed towards identifying cases of IHD.

Contraindications of EST were strictly observed, as shown in Table - lfi making EST a more safe procedure.

It is important to record ECG conti:-.uosly during exercise as significant changes may occur during exercise only.

To prevent baseline fluctuations and artefacts, electrodes were carefully placed and fixed. Tachycardia with pronounced Ta wave, muscle action currents and respiratory variations were difficult to avoid. They led to technical difficulties and problems with interpretation of ST segment changes.

In this study V_5 was used during EST as it can detect upto R9/u of abnormalities. But anterior or inferior wall ischaenia may have been missed with the use of V_5 only. So, it has been recommended that at least six different leads should be used to identify all the ST segment abnormalities during exercise. (17≫ , 'i6)

There are different opinions about the usefulness of EST in predicting IHD. Goldchalger et al found it to be 93% specific and 6sensitive as compared to angiographically proven coronary heart disease Ci?). Sokolow found the specificity and sensitivity of EST to be 70%. (30)# Fortuin et al showed sensitivity to be 5'iXto 80%, specificity 86ˆto 97% vith a predictive value of 87%to 96% (29).

The predictive value of any test depends upon the prevalence of the disease in the study population. If EST is done in subjects with highly suspected IHD, its predictive value is high. If it is done in the population at low risk, its predictive value is limited. Important prognostic information nay be derived from its judicious use in the symptomatic individuals, as it was done in this study. It is not a cost effective, reliable measure for screening the asymptomatic people for coronary artery disease (ˆ8, 49, 50). Diagnostic accuracy of EST is increase when prior probabilities manifested in the history, resting ECG and symptoms while walking on the tread-mill are taken into account (29). Normal EST does not exclude IHD as 25ˆto *»0% patients may develop IHD inspite of normal EST (29). It is not possible to comment upon this observation, as in this study coronary angiography was not performed on any of the patients.

Stress test is being used since the last fifty years. Golhammer and Schert introduced EST for diagnosis of coronary insufficiency in 1932. Master made the test very popular in his articles published in 1935 and 1950 (51). There are two ty- of exercises - isometric and dynamic. It can be submaximal or maximal and discontinuous or contir.u' The treadmill test is a graded type of dynamic, cor.r nuous, maximal or submaximal exercise. It can be applied in a large group of patients and the inert: in oxygen demand is produced in a safe and reprodu: ble fashion, so, it is a very useful test in ider.-.i ing presence of IHD in symptomatic individuals.

The normal ECG response to exercise is decreased PR interval, increased amplitude of P wave, no cha- in the QRS complex, progressive right axis deviati'. dcreased amplitude from the left to the right precordial leads.

ST changes occurring during EST represent changes :- metabolic stress-strain relationship of the tr.yocsr ST segment depression begins with a rise in left atrial pressure, a fall in myocardial extraction o:' lactate and a fall in intracellular $_{p}H$ with a mark'-. efflux of K^{+}. $_{A}$ positive EST was defined as folic-..-.

The criterion used in this study i.e. ST depression of 1mm more than 0.08 sec. from the J point is taken from Sokolow et al (30). A fifty seven year old male teacher who was complaining of chest pain suggestive of angina pectoris but with a normal resing 12 lead ECG, showed ST segment depression on EST in the second minute post exercise. He also had associated chest pain in the second minute of stage three^ so, he did total of seven minutes of exercise before stopping. Veiner has mentioned that if only ST segment depression is taken as a criterion for positive test, 20 to 65% false negative responses are obtained with considerable loss of sensitivity (52). Goldschlager et al found that the downsloping ST segment and the horizontal, depressed ST segment had a specificity of 99% and'93% respectively (47). Fortuin et al and Willerson et al have suggested the horizontal depression of 1.0mm or down slopping ST segment as a positive criterion. They claim that if it appears on maximal or near maximal test, it has the greatest specificity with least loss of sensitivity (29, 40).

A normal person can reach stage three or stage four of the Bruce protocol (53).

If ST segment depression occurs after more than nine minutes of exercise there is 'zero' mortality associated with it (48). Ellestad et al also four, that if ST depression occurs after seven minutes of excrcisc, the mortality rate is slightly higher than the normal population. If it occurs after fi minutes of exercise, it carries worse prognosis. Changes occurring after only three minutes of exerci connote worst prognosis (5ˆ). This view is al supported by Kattuset al (55)* Taking all these v: into account, it can be said that the ST depressicr occurring in the patient in this study is most like a true positive test. As it occurred after sever, minutes of exercise, it is associated with a progr.'. vhich is worse than the general population. This of ST segment depression is indicative of main coronary artery or double or triple vessel disease If coronary angiogram were possible, it would have been very helpful in establishing a firm diagnosis.

The patients who give typical history of angina pectoris and who also have ischaemic ECG response EST are shown to have an occlusion on coronary anc: gram if it is done. One patient showed an upslopi: ST segment with chest pain on the EST. According - the criterion used in this study, this is a normal t.

Goldschlager et ol also found that a slow risir.r ST segment has ?6% sensitivity but 32% false positives are obtained C》7). In a population at a low risk of IHD, like Kenyans, there are more chances of obtaining false positive responses according to the Dayes' Theorem *C19)*. Stuart found that an upsloping pattern of ST and horizontally depressed ST had a 90% incidence of IHD with a 75% and 60% incidence of major two or three vessel disease respectively (56). But by the general view, an upsloirinr * ST pattern is a false positive response and was taker, to be so in this study. One obese postmenopausal female patient showed ST segment depression in the third resting minute post exercise. She had done exercise for 5- minutes, when it was stopped because of dizziness and frtigue. This may be a false positiv response. Post exertional ST segment depression ir. middle aged women is cuite common, 25% false positive have been reported. It may be due to the lower level of hematocrit, a low circulating red cell mass and estrogens which have similar chemical structure as digitalis (29). Sketch et al found that in women, a positive test has little value in indentifying IHD but a negative test is cuite helpful in ruling out significant IHD. Ialse positive is about 67%*in middl aged females (31). De Costa's syndrome (vasoregulator asthenia) has been suggested to be a common cause of false positive in women.

It is characterised by ST - T abnormality on a resting ECG, which increases on standing upright, with hyperventilation and after exercise. ST segment changes are seen early during exercise and improve with more severe exertion. It can be differentiated from a true positive by heart rate lability with inappropriate tachycardia on standing or with mild exercise (53). Other causes of false positives are drug intake e.g. digitalis, propranolol and potassium depleting drugs (29, 53i 56). In this study, it was ensured that none of the patients was on these drugs prior to an EST. Subjects with preexisting repolarization abnormalities, left bundle branch block, Volf-Parkinson-Vhite syndrome and short P-R interval also show false positive response (29, 32). The patient with a positive EST in this study had a normal resting 12 lead ECG and so all the above abnormalities were excluded. Cardiomyopathy, ventricular hypertroph> with strain and valvular heart disease may also give a similar response to exercise (29, 53) but none of these were present in the patient with a positive EST. A false negative response can occur in patients who are on B Blockers due to the blunting of ionotropic effects of exercise.

Bruce has found that if the criteria of ST segment depression is too strict, then of the patients with angina may give a false negative response i.e. ST depression <Jmm (57). Apart from ST segment depression, various other workers have used chest pain, fall in systolic B.P. and lov achieved heart rate as positive evidence of IHD on EST.

In this study, six patients developed chest pain during the EST. Only one of them had ST segment depression on ECG while one patient had an upsloping ST segment. The other four patients did not have any ECG changes. The EST was stopped on occurrence of the chest pain which was relieve by rest in all the six patients. Classical chest pain of angina pectoris occurring during an EST is claimed to have 90% predictive value as compared to 85,t with ECG changes (52). Some other workers have also found chest pain occurrir. during EST to be one of the most important criterion of IHD (53t 55). All these five patients who developed chest pain on EST without ST segment depression on ECG may be suffering from IHD but according to the criterion used in this study, these patients were not considered to have positive EST.

A fall in the systolic B.P. is a very important criterion of coronary artery insufficiency. The ionotropic reserve of the heart is measured by maximum achieved B.P.

Failure of systolic 13.1'. to rise above 130 mm. $_{0}$f has been said to be a better predictor of coronary events than ST changes in asymptomatic individuals (17). Attenuated D.T. response is associated with poor prognosis Cf0). In this study, none of the patients developed fall in **Br.** Achieved heart rtt-: measures the chronotropic reserve of the heart. it depends upon the habitual physical activity of t'-. individual. So, the value of using target heart Te- as on end point has been seriously questioned (>7;. Usually there is a linear heart rate-work load relationship, so it provides a rough index of performance capacity (k0). Subjects who achieve significantly lower heart rate have twice the mortality as compare to the subjects who achieve maximum heart rate (>7J- In this study, three patients achieved target heart rate, twenty patients achieved at least 80% of targe- heart rate. Five patients did not achieve even c0¥ of target heart rate when test was stoppped because of fatigue, chest pain or hotness in the chest.

Double product is defined ns the maximum achieved heart rate x peak systolic B.P. If patients with angina are exercised, they develop chest pain at the same double product every time.

In this study, patients were not exercised more than once, but most of them achieved high double product. Ideally the patient should be exercised until a target heart rate or at least 80% - 90% of it is achieved (58). But sometimes the test has to be stopped with submaximal exercise because of various limiting symptoms. In study done by Bruce prominent limiting symptoms in the healthy men were weakness of legs in 71% and fatigue in 25%. Hypertensive patients complained of dyspnoea, 5ˆ% and weakness 50% before finishing maximal exerciseˆ 61% of patients with a history of angina complained of tightness of chest and *»6% complained of weakness (57). In this study, as shown in Table 18, fatigue was the most prominent symptom followed by chest pain.

This differs from the study cited above. Predominance of fatigue as a limiting symptom may be explained by poor physical fitness, poor motivation, malingering or anxiety neurosis.

Thallium - 201 radionuclide imaging and cardiac catheterization are also important and sensitive diagnostic methods for identifying the presence of IHD but they were not done in any of the patients included in this study.

Cardiac enxymes are useful in confirming the dir.;. - diagnosis of M.I. Lactic dehydrogenase (kDH)}LDK, with an increase in quantity of both or with a rela: or an absolute decrease in LDII^ LDIb and LDH^j occurs in transmural and subendocardial infarctions. It is an excellent test with high specificity and simplicity (59). Creatinine phosphokinase - MD is c specific enzyme from cardiac muscles and it increase within 6 hours of infarction (r/y) Aminotransferase increases in 96 to 97% of M.I. but it does not inert in subendocardial infarcts, Because of lack of facili* cardiac enzymes were not estimated in all the patie:.- with M.I. Only three patients were investigated or. J two of them showed a significant rise in the levels of LDH, CPK and aminotransferase. These patients wer suspected to have acute M.I. on history and were diagnosed on the strength of elevated cardiac enzy-.—: ECGs of these two patients showed low voltageswi t. inverted T waves in left precordial leads and rr.ultij.". premature ventricular contractions. These ECGs may have been taken during evolution of M.I. accounting for absence of ST elevation and/or C. waves. In one- patient with pathological Q waves on ECG, cardiac enzymes were estimated after two weeks of hospitalisa- tion and were found to be normal.

CONCLUSION

CIinical ischoemic heart disease is still vcrv rare in Kenya and most African patients with chest pain as seen at Kenyatta National Hospital do not have coronary heart disease even if the pair, may have some of the characteristics of angina pectoris. It is suggested that most of then; r.ay be si:ffcri:;- from anxiety neurosis.

P EC OMMCNDATTONS

Tatients vith chest pains should be fully evaluated to establish cause of cheat pain especially in middle aged males and those with predisposing factors.

Detailed history should be taker, in all the cases as this might help exclude the patients suffering froc anxiety neurosis.

Although rare in Kenya, physicians should have a high index of suspicion for HID as it does occur.'

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APPENDIX 1;

Personal Data

1. Name

2. Date of Birth

3. Sex: M F

k. Marital status: Single Married

5. Occupation:

Type of work:

Job group and approximate income:

6. Address:

History:

1. History of present illness:

a) Site of pain

b) Radiation

c) Character

d) Precipitating and aggravating factors

e) Relieving factors

f) Frequency

g) Intensity and duration

h) Total duration of illness

i) Other symptoms

2. Past Medical History:

a) Proved myocardial infarction:

ECG - single

- Serial

Cardiac enzymes - single

serial

b) Hypertension Yes/No

Duration:

Drugs used:

No treatment:

c) Diabetes Mellitus Yes/No

Duration:

Drugs Used:

No treatment:

d) Any other drug ingestion Yes/No

3. Family History:

a) Ischaemic heart disease in family . Yes/No

b) Sudden death in family Yes/No

c) Diabetes Mellitus Yes/No

d) Hypertension Yes/No

k. Social History:

Cigarette smoking Yes/No

No. of cigarettes/day

duration

if stopped, when:

5. Brief psychiatry history:

Physical Examination:

1. Height

2. Weight

3. Cardiovascular system:

   Pulse:

   Heart Rate:

   B.P.

   Any murmur: Yes/No

   Site of the murmur

k. Abnormality in any other system.

(-

INVESTIGATIONS

Plain postero Anterior chest y-rny

Cardiothoracic ratio

Any other abnormalities

2. Resting 12 lead ECG

Any abnormalities Yes/No

3. EST

a) Symptoms

b) Heart rate - Initial

- Achieved

c) 13.P. - Initial

- Achieved

d) ST - T wave abnormalities - Yes/No

e) Q-Wave -

f) Ectopics -. Yes/No

Initial -

developed

g) Conduction abnormalities

Fasting blood sugar-done / Not done

5. Lipid profile-done/Not done

6. Cardiac enzymes-done/Not done

LDH

CPK

SCOT

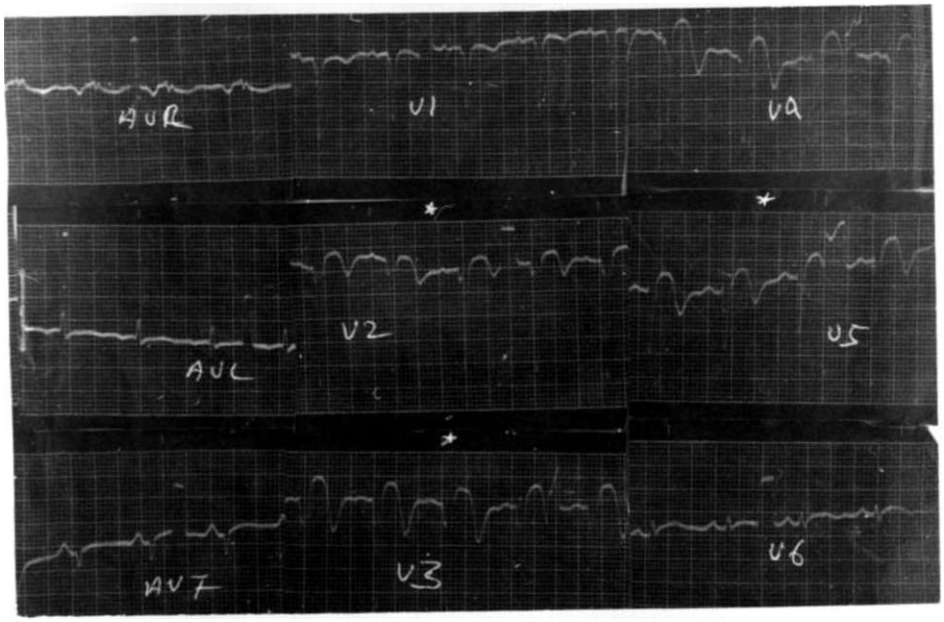
Appendix II:

Bruce Protocol:

Stage	Gradiant	Speed	Time
1	10%	1.7mph	3 mins.
2	12%	2.5mph	3 mins.
3	14%	3.4mpy	3 mins.
4	16%	4.2mph	3 mins.
5	18%	5.0 mph	3 mins.
6	20%	5.5 mph	3 mins

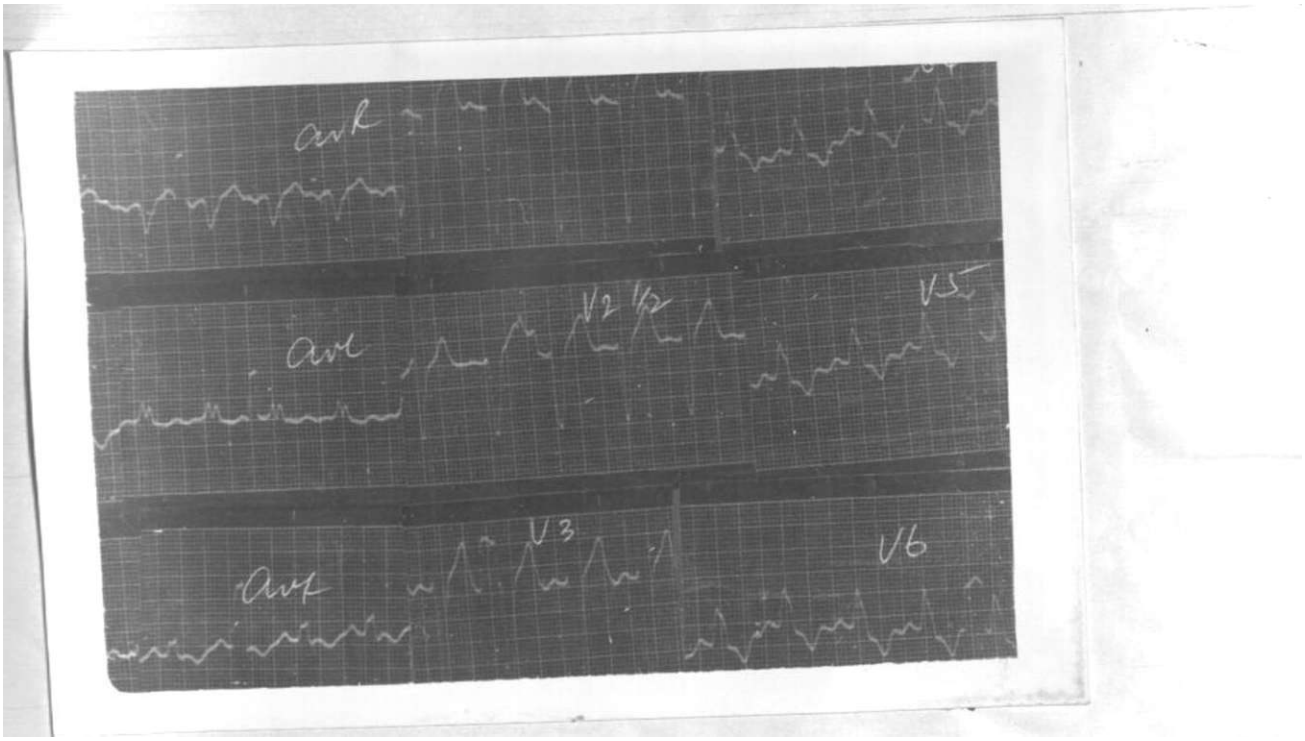
PHOTOGRAPH

1. ECG of the patient with M.I. showing Q waves* ST elevation and T wave inversion in all preccrdinl leads excejt i s, Jg£estive of anterior M.I.



PHOTOGRAPH

ECG of the patient showing left bundle branch block, with suspected M.I. from the history.



PHOTOGRAPH

ECG (V_5) of the patient with positive EST showing ST depression of .5 mm.