THE MANAGEMENT OF MYOCARDIAL INFARCTION

A CLINICAL AND HAEMODYNAMIC STUDY

BY

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SUMMARY OF A THESIS

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An investigation into the effects of earlier mobilisation of patients who have sustained a myocardial infarction is presented. Two comparable groups of male patients have been studied. One group has been treated with a strict bed rest regime, and has been nursed in bed for twenty-five days, after which gradual mobilisation was undertaken over a period of ten days. The other group was treated for fourteen days in bed during which considerable freedom of activity was allowed and then mobilised over a period of seven days in hospital with discharge after 21 days.

The two groups of patients were comparable in terms of age, sex, duration of severity of illness and previous history of infarction. It has been found that the early mobilisation programme has not been deleterious to the patients in respect of mortality or morbidity or the development of serious arrhythmia or other complications of infarction; nor has this programme increased the incidence of aneurysm formation in the earlier mobilised group. The incidence of neurotic reaction in the two

groups was not significantly different, when this was assessed in hospital and after discharge, nor were significant differences detected when the groups were tested psychologically using the Eysenck Personality Inventory. The earlier mobilised group has been found to return to work more rapidly than the other group, but after six months no significant differences were found in the numbers returning to work between the two groups.

Cardiac outputs have been estimated using a dye dilution technique on three groups of patients in the supine and in the 45° head elevated position, corresponding to the position in which a patient might be nursed sitting in bed. The three groups were -

(a) 10 subjects without evidence of cardiac or respiratory disease.

(b) 11 patients who had sustained a myocardial infarction and in whom there was no evidence of pulmonary oedema.

(c) 10 patients who had sustained a myocardial infarction and in whom there was evidence of pulmonary oedema.

The mean cardiac output of the patients in whom there was evidence of pulmonary oedema has been found to be significantly higher in the seated position than in the supine position, but significant differences were not found in either of the other two groups.

It is concluded from these studies that a regime of earlier mobilisation and greater activity of infarct patients, while being treated in bed, has not been harmful and may assist in the rehabilitation of patients. The cardiac output of patients who have no pulmonary oedema is not significantly different if they are supine or seated, but the cardiac output of patients with pulmonary oedema is higher when the patient is nursed seated. It is concluded that there is no justification in insisting on a strict bed rest regime for patients who have sustained a myocardial infarction.

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Some of the material in this thesis has been incorporated in publications in journals and in papers to learned societies. The following are the relevant references -

Return to Work after Myocardial Infarction	(1967)	Scot. Med. J., 12.297
Radiological Abnormalities after Myocardial Infarction. Possible Relation to Aneurysm Formation. (with W.B. James).	(1968)	Brit. Heart J., 30.236
Management of Myocardial Infarction. The Effects of Early Mobilisation. (with A. Allison and G. Shaw).	(1968)	Scot. Med. J., 12.435
Cardiac Aneurysm. (with I. McDicken and W.B. James).	(1968)	Postgrad. Med. J., 44.775
The Significance of Persistent R-ST. Elevation after Acute Myocardial Infarction. (with W.B. James).	(1969)	Brit. Heart J., 31.334

The Effect of Change of Posture on Cardiac Output after Myocardial Infarction		Submitted
Ventricular Aneurysm after Myocardial Infarction	(1967)	West of Scotland Cardiac Society
Cardiac Aneurysm	(1967)	Scottish Thoracic Society
The Effect of Change of Posture on Cardiac Output after Myocardial Infarction	(1968)	Scottish Society for Experimental Medicine

PREFACE

This thesis describes an investigation into the effects of an earlier mobilisation programme of patients who have sustained a myocardial infarction and of allowing them a regime of increased activity while being nursed in bed. Two comparable groups of men have been studied with regard to mortality, complications, psychological sequelae, radiological abnormalities and return to work.

One group has been allowed considerable freedom while being nursed in bed for fourteen days and mobilised over the next seven days. The other group has been allowed only restricted movement in bed, nursed in bed for twenty-five days and gradually mobilised over the next ten days.

Cardiac output studies have been carried out on three groups of patients in the supine and in the 45° head elevated position corresponding to the position in which a patient might be nursed sitting in bed. The three groups were a) a group of non-cardiac controls, b) a group of infarct patients in whom there was no evidence

of pulmonary oedema and c) a group of patients who had sustained a myocardial infarction and in whom pulmonary oedema was present.

The results indicate that the regime of earlier mobilisation which is described is not detrimental to infarct patients and that cardiac output is significantly higher when patients with pulmonary oedema are nursed sitting in bed than when nursed supine.

THE MANAGEMENT OF MYOCARDIAL INFARCTION

CHAPTER I

INTRODUCTION

The first account of myocardial infarction is believed to be that of the Earl of Clarendon (when he described the death of his father in his autobiography published in 1676). "The pain in his arm seising upon him he fell down dead without least motion of any limb - nor could any physician guess whence the mortal blow proceeded." (Underwood, 1953).

One hundred years elapsed before the first medical description of angina by William Heberden in 1772. He noted how frequently the life of a person ended when the person fell down suddenly and perished almost immediately. Heberden also noticed the predominantly male sex incidence of the disease for of the one hundred cases he described, only three were women. (Heberden, 1802).

In 1776, John Fothergill described the autopsy findings in a case of angina terminating in infarction where the coronary arteries were 'one piece of bone' and where the parts of the heart were paler and harder

than normal. The post-mortem in this case was performed by John Hunter, and two of his pupils, Calet Parry and Edward Jenner, were the first to relate angina and infarction to disease of the coronary arteries. Parry (1799) published a book 'Syncope Anginosa' and in it he correlated a large number of autopsy reports with previous clinical histories. The first case of coronary occlusion correctly diagnosed during life was reported by Hammer in 1876 (Major, 1948).

Until the beginning of this century, it was considered that coronary thrombosis was almost immediately fatal. Herrick (1912) pointed out that this was not so, although it was some years before his views were accepted. He discussed the differential diagnosis and gave a detailed account of the signs and symptoms of myocardial infarction. He indicated that occlusion of even a large coronary artery was not always fatal. McNee's account (1925) of three patients

who did not die immediately after myocardial infarction was the first report of this in the British literature.

Herrick (1912) recognised the value of promoting an adequate collateral circulation and recommended several days of absolute bed rest as being of prime importance in the management of myocardial infarction.

The concept of rest as an integral part of the management of patients with coronary artery disease had been introduced at an early stage (Roberts, 1894) and gradually the concept of prolonged bed rest became accepted almost generally. Parkinson and Bedford (1928) regarded complete bed rest as essential in treatment, and recommended that "all preparations for a serious and lengthy illness should be made at once". They considered that absolute rest in bed for not less than one month was imperative to allow healing of the infarction and to reduce the risk of embolism. Convalescence was to be prolonged and the return to ordinary life postponed as long as possible. It was

also suggested that "if exertion was limited to less than that which induced pain greater capacity might ultimately be obtained". Haynes (1931) recommended four to six weeks in bed and Coneybeare (1932) recommended at least three months. Kilgore (1933) recognised that there was a division of opinion concerning bed rest after myocardial infarction but recommended at least four to six weeks in bed and regarded the practice of treating a patient on clinical grounds as dangerous and indicating an inadequate conception of the disease. He had noted at autopsy how often patients had died when there was only a small area of infarction. Hay (1935) suggested one month in bed as the bare minimum with another month in bed advisable. He was aware however of the psychological problems that such a period of bed rest engendered.

Pardee (1920) recognised an electrocardiographic pattern that was typical of infarction and as a result milder cases of infarction were diagnosed. Gradually

it became to be realised that the prognosis was not necessarily as grave as had previously been thought, and that reasonable functional recovery could occur.

It is not surprising that prolonged bed rest should have been recommended for patients after a myocardial infarction. Rest of the affected organ has always been regarded as a cardinal principle of the treatment of disease. Doctors immobilise fractured bones, rest insulted digestive organs, and for many years it was standard practice to collapse diseased lungs. Although it is not possible to rest the heart completely it has been assumed that maximum rest can be obtained for it by absolute rest in bed.

Few clinical trials of the value of bed rest in myocardial infarction have been conducted. Cooksey (1935) in a rather inconclusive trial claimed that patients kept in bed for six weeks after infarction did not do as well as those confined to bed for shorter periods. Mallory, White and Salcedo-Sagar (1938)

in a post-mortem study of hearts with recent myocardial infarction found that necrotic muscle had been removed after two weeks and that the scar was reasonably sound after three weeks. They insisted that at least three weeks in bed was essential. Bain (1941) recommended bed rest for four to six weeks on the basis of these findings, but felt that this regime could be modified if the patient felt well.

Dr. S.A. Levine considered that the heart could be rested more effectively with the patient seated in a comfortable chair by the bedside, than with the patient confined strictly to bed. In a series of papers (Levine, 1940; Levine, 1944; Levine, 1950; Levine & Lown, 1952) he propounded his arguments against the treatment of cardiac (including coronary) patients with prolonged bed rest. He considered that keeping the patient in bed produced in some cases those conditions which one would generally hope to avoid. He pointed out that venous return is increased

in the recumbent posture, and fluid tends to accumulate in the lungs since in some cases the left ventricle is unable to keep up with the increased work of the right heart. He referred to his own observations and those of Perera and Berliner (1943) which indicated that with recumbency, haemodilution tends to occur and the blood volume increases as the result of the shift of extravascular fluid into the vascular compartment.

McMichael and McGibbon (1939) had shown that there was a decrease in total lung volume of over 300 ml. with the patient recumbent and a decrease in vital capacity of 200 ml. in normal individuals. Levine used these facts as further examples of the deleterious effect of bed rest. He also cited some of the other problems that might be encountered as a result of bed rest. Urinary retention often develops, necessitating catheterisation and its attendant complications including the introduction of infection. Some patients develop hypostatic pneumonia and while a patient is in bed, the

risks of deep vein thrombosis and pulmonary embolism are greatly increased.

Levine had employed the armchair treatment of patients with coronary thrombosis since 1937. As soon as possible after the initial pain of the infarction had settled, the patient was assisted into a comfortable chair. Care was taken that no pressure was exerted on the leg veins. The patient was left in the chair until fatigue was experienced and then helped back into bed. The aim was to have the patient out of bed as much as possible without discomfort. Most patients were out of bed for one or two hours the first day, with increasing periods subsequently. By the end of the first week, most of the day was spent out of bed. The only contra-indications to the use of the chair were a continuing state of shock, marked debility and a concomitant cerebrovascular accident. Pyrexia, pain, pericardial friction, triple rhythm, heart block and arrhythmias, or the need for oxygen therapy were not regarded as

contra-indications. Nearly all the patients fed themselves and were either permitted the use of the bedside commode or granted toilet privileges. They were allowed to take a few steps towards the end of the third week and remained in hospital for about four weeks. The mortality rate for a series of 81 patients treated in this way was 9.9%. Levine stated quite clearly that only selected patients were treated in this way and the only method available of assessing results was to compare the mortality rate with a group of patients in the same hospital treated along conventional lines who had a mortality rate of 13.8%. However, he concluded that the armchair treatment had not increased mortality rates and thought that one of the most encouraging aspects of this type of management was the continued sense of well-being and the high morale that existed in patients treated in the chair. Levine observed that profound psychological changes followed putting a patient to bed for a long period, and that anxiety was

especially harmful to the patient with coronary artery disease since Stead, Warren, Merril and Brannon (1945) had demonstrated that emotion caused a marked rise in cardiac output which almost certainly reflected an increase in cardiac work.

That prolonged bed rest is detrimental to the well-being of patients and that it impairs the speed of convalescence has been demonstrated by other writers. Dock (1944) described some of the sequelae of bed rest in general and in a symposium on "Bed Rest" the undesirability of prolonged bed rest was stressed in cardiovascular disease (Harrison,1944), surgery (Powers, 1944), orthopaedics (Ghormley, 1944), obstetrics (Eastman, 1944) and psychiatry (Menninger, 1944). Keys (1945) stated "the current practice of prescribing almost indiscriminately bed rest and then continuing this until all the signs of the primary disease are gone definitely enhances deconditioning and probably delays real recovery. The physician emerges

as an influential agent in deconditioning but he too often relies on nature and chance for the reconditioning of his patients". He felt that much of the deconditioning could be prevented by allowing the patient to sit up in bed, by the use of simple hand and arm exercises and by allowing toilet privileges.

Dietrick, Whedon and Shorr (1948) investigated the effects of immobilisation on four healthy young male volunteers. After an initial control period of observation in a strictly controlled environment from the metabolic point of view, these four young men were strictly immobilised for three months and then continued to be observed during the recovery period. It was found that immobilisation produced negative nitrogen, calcium, phosphorus, potassium and sodium balance. Muscle strength as measured by ergometric methods fell by 13.3% in the anterior tibial groups and by 20.8% in the gastrocnemius and soleus groups. Muscle girth was decreased significantly (from 2% to 6.3% in different

muscle groups). It required four weeks for muscle strength to return to normal and five to six weeks for girth of muscle to return to normal. It was also found that immobilisation brought about a definite deterioration in the mechanisms essential for the maintenance of vascular tone. Within one week of the time immobilisation was instituted the subjects began to develop a tendency to faint in the erect position during tilt-table tests and towards the end of the immobilisation period all four subjects developed purpuric haemorrhages about the feet on the tilt-tests. Master exercise tolerance tests on the subjects showed decreases in exercise tolerance as a result of immobilisation and all the subjects fatigued more readily.

These workers concluded that there was little danger to the average patient from periods of unrestricted bed rest of two to three weeks, but with longer periods there was the risk of urinary tract

stone formation, of impaired response of the circulation to the upright position and of loss of muscle strength and mass.

Cuthbertson (1929) in a paper from the Glasgow Royal Infirmary and University of Glasgow also demonstrated that prolonged rest in healthy subjects led to a loss of nitrogen, sulphur, phosphorus and calcium which appeared to be due primarily to the non-use of muscles. He investigated eight subjects on a strictly controlled diet. After a pre-rest period which was imposed for base-line studies the subjects were confined to bed; one lower limb was confined in an osteotomy splint, the other was loosely attached to a sand bag. The subjects were confined to bed in a propped up position and asked to limit movement as much as possible.

Levine had insisted throughout that he did not advocate early mobilisation of his coronary patients and that all his efforts were directed at resting the

heart as much as possible during its early healing phase. However this argument and the other work quoted has encouraged physicians to advocate earlier mobilisation for their coronary patients (Irvine & Burgess, 1950; Brummer, Linko & Kasanen, 1956; Brummer, Kallio & Tala, 1966). These studies have not dealt with comparable groups of patients treated simultaneously and therefore are open to criticism on account of selection and because of gradual improvement in supportive therapy for myocardial infarction which has taken place over the years. While many workers are engaged in assessing the results of anticoagulant therapy (M.R.C., 1964), intensive care units (Goble, Sloman & Robinson, 1966; Fluck, Olsen, Pentecost, Thomas, Fillmore, Shillingford & Mounsey, 1967; Lawrie, Goddard, Greenwood, Harvey, Julian & Oliver, 1967), insulin, glucose and potassium regime (Sodi-Pallares, Testelli, Fishleder, Bisteni, Medrano, Friedland, De Micheli, 1962; Mittra, 1965), low

molecular weight dextran (Laangsjoen, Falconer, Sanchez & Lynch, 1963; Borchgrevink & Enger, 1966), it is apparent that agreement has not been reached on some of the basic points of management.

As an example of the differences of opinion which exist in our local hospitals concerning the management of myocardial infarction, the inquiry of a medical student of each of the medical units of the Glasgow Teaching Hospitals (Shaw, 1967) is of interest. Four units treated their infarct patients in bed for twelve to fourteen days with a further seven to ten days in hospital. Nine units keep patients in bed for twenty-one days or more and one unit kept them in bed for thirty-five days.

Even within one city and often even in one hospital therefore there is a wide variance of opinion on a basic aspect of patient management.

During the past three years a group of one hundred and five male patients with recent myocardial

infarction has been studied in an attempt to compare two regimes of management. With one regime patients were allowed increased activity while they were kept in bed for fourteen days and then allowed home after a period of gradual mobilisation. In the other, patients were strictly limited in respect of the amount of activity permitted in the early stages, were kept in bed for twenty-five days and allowed home after a slower regime of mobilisation.

The results of this investigation are recorded in Chapter II. In Chapter III experimental data obtained while estimating the cardiac output of patients and controls in the two postures, either fully recumbent, or sitting in bed are presented. In Chapter IV, the work is summarised and the conclusions which can be drawn from this study are stated.

THE MANAGEMENT OF MYOCARDIAL INFARCTION

CHAPTER II

THE EFFECTS OF EARLY MOBILISATION

Two comparable groups have been obtained by allocating patients to one of two groups according to the day on which the patient was admitted to hospital. On one receiving day (the day on which new admissions are taken into the ward), patients were admitted to one group, and on the next receiving day, patients were admitted to the other group. The groups have been named Group A and Group B for convenience of discussion. Group A was the more conservatively treated group and Group B the more rapidly mobilised group. Because of the circumstances which prevailed at the time of the study in the Southern General Hospital it was also possible to separate the two groups further by nursing them in separate but adjacent wards which allowed no contact between the patients in the two groups, but made it possible for both groups to be treated medically and nursed by the same people, apart from the consultant in overall charge of the case (Appendix A).

Only male patients with a history of myocardial infarction of less than forty-eight hours duration were admitted to the trial. Group A consisted of fifty-five patients and Group B of fifty patients. The ages ranged from 38 years to 84 years (mean Group A, 59.7; S.D. 9.19; mean Group B, 58.4; S.D. 9.76). The criteria for the diagnosis of myocardial infarction were as follows:-

1) A history of cardiac ischaemic pain or other clinical evidence suggestive of myocardial infarction.
2) Electrocardiographic changes of acute myocardial infarction with Q waves and/or changes in the R-ST segments.
3) A rise of serum glutamic oxalacetic transaminase (SGOT) above 40 units (Dade).
4) A rise in the erythrocyte sedimentation rate (ESR, Westergren).

Generally all four conditions were satisfied but at least two of the four had to be satisfied including either (1) or (2).

Previous history of myocardial infarction.

Twenty-two of the patients had suffered one or more previous episodes of myocardial infarction. Seven of the patients in Group A had one previous infarction and four had two previous infarctions. Eight patients in Group B had one previous infarction, two had sustained two previous infarctions and one had had three previous infarctions.

Condition at time of admission.

Heart failure (left or right), hypotension (B.P. < 100 mm. Hg. systolic) or shock was present on admission in twenty-one patients in Group A and twenty-seven patients in Group B.

The Peel Prognostic Index (Peel, Semple, Wang, Lancaster & Dall, 1962), which has been used to assess the severity of myocardial infarction, was

calculated for the patients in each group. The range in Group A was 1-17, mean 8, and in Group B the range was from 1-21, mean 10.

The data concerning the comparability of the two groups of patients are presented in Table I.

The electrocardiograms were recorded on direct writing Cambridge Mark II or Mark III machines, using the twelve standard leads, on admission, on the third and seventh day after admission and at weekly intervals thereafter until the patient left hospital, or more frequently if required. Tracings were also made at each out-patient visit.

SGOT was measured by the method of Reitman and Frankel (1957) on admission and on the two following days.

ESR was estimated by the Westergren method using sequestrinated blood (Westergren, 1921; Dacie & Lewis, 1963) on admission and on the fifth, sixth or seventh day after admission and weekly thereafter

while the patient remained in hospital.

Management.

The nursing procedure applied to the two groups is detailed in Appendix B. The period of two weeks in bed was considered to be the minimum period of rest which could be safely recommended in view of the time taken for removal of necrosed tissue. The longer period in bed laid down for the other group was felt to be fairly representative of the period of bed rest insisted upon by physicians preferring a conservative regime of treatment.

All patients were given the same information. They were told that they had sustained a 'heart attack' or coronary thrombosis. Reassurance was given at an early stage and every effort was made to allay anxiety. At the time of discharge patients were told to "take things easy for a few weeks" after which they "should gradually increase activity, returning to normal about three months after the incident". They were told to

"avoid sudden or severe exertion and fatigue".

The ward sister was involved in all stages of discussion concerning the project and was responsible for its explanation to junior nurses. The medical staff was the same for the two groups with the exception of the two consultants.

At the stage of mobilisation, chest radiographs were obtained in the postero-anterior and lateral projections and three months after the incident cardiac fluoroscopy was performed using an EMI-Siemen image intensifier television system. Under couch tube films were exposed in deep inspiration in the left lateral, right anterior oblique and left anterior oblique projections of the heart using a tube potential of 125 KV and without a grid. A further PA chest radiograph was obtained at that time. Particular attention was paid to the presence of abnormal contour, abnormal pulsation, calcification or pleuropericardial

adhesion both during fluoroscopy and on reading the radiographs.

In the ward frequent observations were made concerning the patients' physical condition and their psychological state. Patients were reviewed as out-patients ten weeks, six months, twelve months, eighteen months and twenty-four months after the incident, although more frequent consultation occurred if required. Particular attention was paid to pulse rate and rhythm, blood pressure, heart sounds and heart size, lungs and signs of cardiac failure were sought. The presence of angina and dyspnoea and of anxiety, depression and hypochondriasis was recorded.

At the stage of mobilisation all surviving patients and a series of matched controls who had neither clinical nor electro-cardiographic evidence of coronary artery disease were interviewed by a member of the Research Psychosomatic Unit at the Southern General Hospital and psychological testing with the Eysenck Personality

Inventory (Eysenck & Eysenck, 1964) was carried out. This test was repeated approximately one year later (Appendix C).

An electrocardiogram was carried out at each out-patient visit, and careful inquiry was made concerning rehabilitation and return to work. Any problems concerning these were discussed. All patients under 65 years of age have been graded according to the Registrar-General's system of social grading (General Register Office, 1960). Statistical analysis has been carried out according to Hill (1967) unless otherwise specified.

RESULTS:

Mortality Rate. Twelve patients in Group A died and nine patients in Group B. There is no significant difference in the mortality rates of the two groups (Table I). Four of the eleven patients with recurrent myocardial infarction in Group A died and three of the eleven with recurrent infarction

in Group B. When these deaths are excluded from the series there is still no significant difference between the groups (Table II).

The times after the onset of infarction at which the deaths occurred are of interest (Table III). Those between the eighth and fourteenth day merit closer study since this is the time when pulmonary embolisation is a major hazard. However no evidence of this was obtained at post-mortem in any of the patients who died at this time.

Further Pain. Episodes of pain occurring after that of the original myocardial infarction were recorded in fifteen patients in Group A and sixteen patients in Group B. There is no significant difference between the two groups in this respect (Table IV).

Onset of Hypotension, Heart Failure and Shock. The presence of one of these serious complications of myocardial infarction was recorded at the time of the patient's admission to hospital (Table I). Eleven

patients in each group developed one of these complications after admission to hospital (Table IV).

Arrhythmia and Conduction Disturbance. Electronic monitoring was not carried out in all patients. Clinical or electrocardiographic evidence of a rhythm or conduction disturbance was obtained in eighteen patients in Group A and nineteen patients in Group B. These numbers are not statistically significant (Tables IV & V).

Cardiac Rupture. This was not found in any of the sixteen autopsies nor was there clinical suspicion of this complication in any of the five patients who died and in whom autopsy was not performed.

Psychological Disturbance and Testing. Pathological degrees of anxiety or depression were seen in five patients in each group while in hospital. Six patients in Group A developed neurotic symptoms after return home, and one man's neurotic symptoms improved after discharge from hospital. Neurotic symptoms

developed after return home in five patients in Group B, but two patients who had been depressed while in the ward improved after return home (Table VI).

The results of the personality questionnaire revealed no significant differences in the degree of neuroticism or extroversion between the two groups, either at the time of discharge from hospital or at the follow up examination approximately one year later, although a trend is apparent towards lower neuroticism scores in the earlier mobilised group, both on leaving hospital and one year later (Table VII).

Radiological abnormalities. Seventy-five of the survivors have been examined by the radiological techniques described above; thirty-nine of these were in Group A and thirty-six in Group B.

The major abnormalities detected have been grouped under the following headings:-

i) Bulges

ii) Abnormal pulsations

iii) Pleuropericardial adhesion

i) Bulge. This is a localised prominence of the exterior surface of the heart (Plates 1, 2 & 3). They have been found in fifteen patients. Nine of these were in Group A and six were in Group B. Some were obvious, others minimal and seen only in tangential projections. Most were associated with absent or frankly paradoxical pulsation. Twelve of the bulges occurred on the anterior border of the heart, two on the posterior border and one on the postero-inferior border. The position of twelve of these bulges corresponded to the electrocardiographic site of the infarction. Q waves in the E.C.G., indicating transmural infarction, were present in seven patients.

ii) Abnormal pulsations. This is an area of diminished pulsation or one of frankly paradoxical movement when compared with the adjacent heart muscle. This occurred in five patients (all in Group A). Four were

on the anterior border of the heart and the other at the apex of the heart. They corresponded to the electrocardiographic site of the infarction in four cases and Q waves occurred in one patient.

iii) Pleuro-pericardial adhesion. This is a tent shaped opacity with its base contiguous with the heart shadow. An adhesion may obscure a cardiac bulge, but apart from that its significance is debatable, although it seems likely that it represents the end result of the inflammatory reaction that commonly occurs in the pericardium as a result of myocardial infarction (Plates 4 & 5).

Pleuro-pericardial adhesions were seen in eleven patients (six in Group A; five in Group B) and corresponded to the electrocardiographic site of the infarction in eight cases. All occurred on the anterior surface of the heart. Q waves were present in six patients.

Other abnormalities detected.

Left ventricular enlargement was detected in nine patients, coronary artery calcification in eight patients, pulmonary congestion due to heart failure in four patients and areas of pulmonary infarction in two patients.

The results of the radiological investigation are shown in Table VIII.

Return to Work.

Patients who were sixty-five years of age or older and patients who were unfit physically to return to work were excluded from this assessment. Four deaths occurred in this group within a short time of discharge from hospital and have been excluded. The patients were almost entirely from social grades 3, 4, 5 (Table IX).

Twelve of the remaining thirty-one patients in Group A and fourteen of the remaining twenty-six patients in Group B had returned to work three months

after the infarction. The numbers at six months were twenty-two and twenty-three respectively (Table X).

DISCUSSION

Following unproven traditional procedures is a common fault in medicine, and it is important to review periodically the evidence which is available. Many physicians have spoken out in the past against prolonged bed rest for myocardial infarction, and numerous patients have also rejected this method of treatment.

The important advantages ascribed to bed rest have been the avoidance of cerebral anoxia during hypotension, the prevention of sudden death, the preservation of myocardial function, and the prevention of cardiac rupture and aneurysm formation.

The introduction of intensive care units has provided much information concerning the mechanism of hypotension and shock. It is now realised that putting the patient in a head down position might increase

pulmonary congestion and hypoxia and the rationale of this procedure in the management of hypotension especially in the presence of heart failure must be suspected.

The effect of the change of posture on the cardiac output in patients after a myocardial infarction is examined in Chapter III.

Electronic monitoring of patients has revealed the high incidence of arrhythmia following myocardial infarction. Figures as high as 90% have been quoted (Julian, Valentine & Miller, 1964) and it is accepted that 30-40% of patients die an "electrical death" from ventricular fibrillation, (Proceedings of Second Bethesda Conference of the American College of Cardiology, 1966). While this may be precipitated by anoxia and pulmonary congestion, increased mobility and earlier mobilisation would not appear to be important factors.

Evidence is lacking that the heart will heal more soundly if the patient is treated for a long period in bed or that the heart's function will eventually be better in patients treated in this way. Rupture of the heart usually occurs in the first two weeks and is relatively uncommon thereafter (Friedman and White, 1944). It has been shown histologically that much of the necrotic tissue has been removed and replaced by connective tissue after two weeks (Mallory, White & Salcedo-Salgar, 1939). After this time, healing continues and is complete after six to eight weeks, depending on the size of the infarction, since organisation must take place from the periphery, and on the efficiency of the collateral circulation. The report by Jetter and White (1944) concerning the high incidence of rupture of the myocardium in sudden, unexplained deaths of psychotic mental hospital patients cannot be used as evidence in this discussion. These patients did not take even reasonable precautions and this group of

patients was chosen for examination since it was anticipated that both mental and physical excitement would be present during the post-infarction period. In fourteen of the sixteen cases which these authors reported the time of rupture was within two weeks of the time of infarction as determined pathologically.

It has been stated that failure to rest in the early stages after the infarction predisposes to aneurysm production (Parkinson, Bedford & Thomson, 1938; Moyer & Miller, 1951). The work of Sutton and Davis (1931) is frequently quoted. Five dogs, after coronary artery ligation, were exercised on a motor driven treadmill commencing at intervals from 2 - 6 days after the experimentally induced infarction. One of the dogs, exercised severely from the third day showed an aneurysmal area in the heart when it was sacrificed. Sutton and Davis concluded from this single experiment that early exercise favours aneurysm formation and that rest produced a firm scar. Not

only is this report statistically worthless, but the forced exercise to which the dogs were subjected is in excess of anything contemplated in the management of human cases, and yet this report is one of those most frequently quoted in discussion regarding aneurysm production as a result of early mobilisation of infarction patients.

Brummer et al (1956) and Brummer et al (1966) have reported their experiences in the treatment of patients after a myocardial infarction by a scheme of early ambulation. Since 1952, their patients have been allowed to sit in bed about one week after the infarction if the acute symptoms have settled. Bedside toilet privileges are allowed from the beginning. Patients were kept in bed for periods of two weeks although in the latter years of the experiment this was reduced to periods of ten days. Their results indicate that early ambulation does not constitute an increased danger to the patient. No attempt has been made to control these experiments. Levine and Lown (1952) presented their

results of the treatment of 81 patients with acute coronary thrombosis. No details are given concerning the severity of the infarctions, nor was there a control group. The mortality rate of 9.9% for 57 patients treated in hospital and 24 patients treated at home is compared with the general hospital mortality rate of 15%. Beckwith, Kernodle, Le Hew and Wood (1954) reported the results of a controlled trial of a modification of the armchair regime suggested by Levine in which 80 patients were involved. A group of 39 'up patients' were allowed to sit in an armchair for increasing periods, after symptoms of shock and pain had disappeared and the results with regard to mortality and morbidity compared favourably with the observations in the other group of 41 patients treated in bed. These authors also found that psychological disturbance was less in the 'up patients' and rehabilitation proceeded more smoothly. Lauper, Lichtlen and Rossier (1967) have also recently reported their results with a modified form of the

armchair treatment. They kept all patients in bed for a week after infarction and found no significant differences subsequently between two groups one of which was treated out of bed and the other of which was treated with long periods of bed rest before mobilisation.

In the presence of so much conflicting opinion about the treatment of myocardial infarction and in the absence of any controlled trials there would appear to be a need for a controlled study concerning the effects of early mobilisation on immediate mortality rates, complication rates and subsequent psychological re-adjustment and rehabilitation.

Before commencing the study which is reported here, one had had several years of experience in treating patients along the lines laid down for early mobilisation in the trial, and had been impressed with the favourable mortality rate, and the physical and psychological well-being of the patients at all stages but especially after discharge from hospital.

The difficulties in arranging a trial of this type are numerous; these include the attitudes of different physicians, the training of nurses, the attitudes of the patients themselves and of their relatives. The intermingling of patients who are being treated by different regimes would present difficulties.

Due to the staffing arrangements at the Southern General Hospital it was possible to meet some of these problems by segregating the two groups of patients to be studied in two different wards, situated adjacent to each other and yet allowing the patients little opportunity of meeting. At all times the same house officer and registrar (B.M.G.) were in contact with the patient, although the consultant in charge of each ward was different. The same sister and nursing staff was in attendance for the two groups (Appendix A). After discharge from hospital, the only hospital doctor seeing the patients was the registrar (B.M.G.). Due to these ward arrangements and due to an interest in rehabilitation

and return to work, the observations were confined to male patients, although the principles established are now being applied to the treatment of all infarction patients.

Although patients were not allocated to each group according to a strict random selection routine but according to day of admission, that the two groups are comparable is demonstrated by the lack of significant difference between the groups in respect of numbers, ages, Peel Prognostic Index and the presence of major complications at the time of admission; and all the patients were male.

The mortality rates of the two groups of patients while in hospital were almost identical even when allowance is made for the increased hospital stay of patients in Group A and when the deaths occurring in those patients with recurrent infarction are excluded. The mortality rates are lower than is sometimes reported in hospital series of myocardial infarction

(Honey & Truelove, 1957; Richards, 1958; Lovell, 1964). There are several possible reasons to account for this, including case selection, delay in admission to hospital and diagnostic criteria. Grace (1967) has recently submitted a plea that articles referring to mortality rates in myocardial infarction should specifically indicate what type of case is being reported, the complications present on admission and the delay between the onset of infarction and admission to hospital. Groups A and B were comparable in these respects although they may be different from other series reported. It is also possible that the improved mortality rate can be attributed to the greater care which is now being taken in the management of patients who have sustained a myocardial infarction, particularly by those who have a special interest in this field. While coronary care units are not yet general in this country at this time, many of the lessons being learnt in these units are being applied to the management of patients

in the general wards. Hypoxia and pulmonary congestion are being recognised earlier and oxygen, diuretics and anti-arrhythmic drugs are exhibited more frequently.

The incidence of arrhythmias detected clinically is similar to the incidence in other clinical reports (Honey & Truelove, 1957), but falls far short of the numbers which are being detected by continuous monitoring of heart rate and rhythm (Julian et al, 1964; Robinson, Sloman & McRae, 1964; Groden, 1968). Although some of our patients were monitored in this way, facilities were not fully available at the beginning of the trial and the indication for the use of electronic monitoring equipment was, in general, the detection of a potentially serious arrhythmia or conduction disturbance. All that can be concluded from the observations recorded is that obvious rhythm and conduction disturbances occurred in similar numbers of patients in the two groups.

A serious complication such as hypotension or congestive or left sided heart failure developed after

admission in the same number of patients in each of the groups studied. Allowing for the slight disparity in the sizes of the groups these differences are not significant.

From these observations it seems not unreasonable to conclude that the regime of greater freedom and earlier mobilisation which has been followed has not had an adverse effect on the prognosis in the early stages after myocardial infarction.

Radiological Findings

Seventy-five of the survivors of the initial myocardial infarction were examined radiologically to determine the incidence of aneurysm formation in the two groups. In this way it has been possible to observe radiologically the changes which occur after a myocardial infarction. A major concern has always been that inadequate rest and earlier mobilisation might predispose to aneurysm formation. The work of Sutton and Davis (1931) mentioned previously has been regarded as confirmation of this belief. Moyer and

Hiller (1951) state that in the twenty cases of aneurysm after myocardial infarction which they reported only three had an adequate period of bed rest in the early stages after the infarction. These authors were of the opinion that this strengthened the argument that early ambulation following myocardial infarction plays an important role in aneurysm formation. The problem has also been examined recently by Dubnow, Burchell and Titus (1965). These workers grouped the cases of aneurysm which they had detected in an autopsy study into two groups, according to whether the patients had been treated in bed for periods of more than or less than three weeks. They found that this factor was not important. Mitchell, Dealy, Lown and Levine (1954) re-examined 42 of the 56 surviving patients of the original series of 81 chair treated patients with acute myocardial infarction at an average of 26 months after the acute illness. No evidence of ventricular aneurysm was found with careful radiological examination including

fluoroscopy of the heart. However this was an uncontrolled study and the fluoroscopic facilities available at that time would make small abnormalities difficult to detect. It is noteworthy that twelve of their patients were found to have enlargement of the heart which has been said to be suggestive of aneurysm, when it develops after myocardial infarction (Holmes & MacFadyen, 1964).

Of the seventy-five patients in the present series who were examined radiologically by cardiac fluoroscopy more than three months after the infarction, fifteen patients were found to have bulges of the external contour of the heart. Nine of these were in Group A and six in Group B. Areas of abnormal pulsation were seen in five patients in Group A and no patients in Group B. It seems fairly certain that the bulges detected are aneurysms and the areas of abnormal pulsation probably are too. Although it has been stated that pleuro-pericardial adhesions may

conceal aneurysms (Holmes & MacFadyen, 1964) it is not possible to state that the adhesions which were detected represented aneurysms, and while acknowledging that they almost certainly represent the end result of the pericarditis which commonly complicates myocardial infarction, they will not be considered further.

The incidence of bulges is not significantly different in the two groups. This indicates that the increased mobility and earlier mobilisation has not predisposed to aneurysm formation.

These findings are consistent with the studies of Master, Gubner, Dack and Yaffe (1940), Prinzmetal, Schwartz, Corday, Spritzler, Bergman and Kruger (1949), and of Kurtzman and Lofstrom (1963). Master et al (1940) and Kurtzman and Lofstrom (1963) examined groups of patients at different stages after a myocardial infarction and found areas of bulging and abnormal pulsation in large numbers. In the early stages after infarction the incidence almost reached 80% (Kurtzman and Lofstrom, 1963). Prinzmetal et al (1949) ligated

the coronary arteries of dogs and demonstrated ballooning of the infarcted area. Gorlin, Klein and Sullivan (1967) found areas of bulging of the heart in 20% of 100 patients after myocardial infarction, who were undergoing coronary arteriography and in whom left ventriculography was also carried out. It would appear therefore that cardiac aneurysms are much more common after myocardial infarction than is commonly supposed. An examination of the literature reveals a reported incidence of from 3% (Lisa & Ring, 1932) to 38% (Appelbaum & Nicolson, 1935). In a most comprehensive review of the subject, Schlichter, Hellerstein and Katz (1954) indicated that aneurysms were present in 20% of patients who had died with evidence of a previous myocardial infarction. Douglas, Sperazza and Marici (1962) found an incidence of 8.7% in patients dying from myocardial infarction and Abrams, Edelist, Luria and Miller (1963) 12.4%. Dubnow et al (1965) in a review of 2,293 hearts with old or recent

infarction found about 3%. All of these were post-mortem studies in selected material; several authors make the point that the diagnosis is rarely made in life, and clinical reports are few and usually relate to small numbers of patients.

The knowledge that surgery is feasible for the treatment of ventricular aneurysm (Chapman, Amad and Cooley, 1961) has stimulated greater interest in the diagnosis in life and the management of this condition. Holmes and MacFadyen (1964) described six cases diagnosed during life, and Bjork (1966) described his experience with fourteen cases diagnosed by left ventriculography and confirmed at operation. Steinberg (1966) reported eleven cases collected over a period of twenty-seven years in all of whom the diagnosis was confirmed by intravenous angiocardiography. In many of these cases however, the diagnosis had been suspected by simpler radiological methods and since it would appear that these cases which are of functional importance can

be diagnosed by simple chest radiology and careful cardiac fluoroscopy as described by Groden and James (1968, 1969), it is doubtful whether the risks of these more elaborate procedures are justified.

None of the cases in whom we have found evidence of aneurysm has been referred for surgery. The views of Abrams et at (1963) seem reasonable. They suggest that a conservative approach should be adopted in the management of post infarction aneurysm and that surgery is indicated only when the aneurysm is exerting a functional effect and where cardiac failure cannot be treated effectively by medical means; and in those cases where repeated systemic emboli originating in the aneurysm cannot be controlled by anticoagulant therapy.

RETURN TO WORK

There are two main aims in the management of myocardial infarction. The first is the immediate saving of the patient's life; the second is to prepare

him for further living. The latter has often been neglected in a desire to accomplish the first. It has been suspected in the past that not only the length of bed rest but also the rigid routine that is usually practised at home and in hospital are unnecessary (Irvine & Burgess, 1950; Fry, 1967). It has also been found to be difficult to recommend regimes that allow greater freedom or to recommend that patients should return to work since an unfortunate incident or coincidence might affect a physician's reputation.

It was felt that one of the ways in which a man might demonstrate his physical and psychological well-being would be by an early return to work after an infarction. This aspect of the management of the patient with myocardial infarction has not attracted as much attention as other aspects of the disease, although failure to return to work represents a great economic burden to the patient and the community. There is no reference in the literature to the effects of early

mobilisation or armchair treatment on a patient's success in returning to work.

Cole, Singian and Katz (1954) followed up a series of 285 patients who had sustained an initial myocardial infarction between the years 1932 and 1942 until death or the end of the study in 1952. They stressed the importance of recognising a large group of mild cases with a relatively good prognosis, but found that only 59 men had returned to work after the attack (27%). Master, Yaffe, Teich and Brinberg (1954) found that 69.7% of patients from whom they were able to obtain information were working full time or part time, and most had returned to the type of work they had been doing before the incident. They noted that work performance after an infarction is often very satisfactory and that the patients who had resumed work fared as well as those who had retired. The majority of the patients returned to work within three to six months after the infarction although some patients were quicker and others were slower.

Dimond (1961) studied 202 railway operating employees who returned to full time work after a first myocardial infarction. He found that the average duration of survival was eight years, and that the prognosis for those returning to work was significantly better than for those who did not. The improved prognosis was due not so much to freedom from re-infarction as to a decreased incidence of myocardial failure and the ability to survive re-infarction.

It would seem that in general terms the fitter a patient is the more likely he is to resume work, although it is possible that the increased activity in travelling to and from work and actually in performing work might help to improve coronary circulation.

These studies have been conducted in the United States, but the problem has also been studied to some extent in Scandinavia, in Australia and in this country.

Biorck and Wedelin (1964) in a report to the

W.H.O. Expert Committee on Rehabilitation of Cardiacs in Geneva (July, 1963) stated that about 17% of patients previously employed did not return to work after an infarction. Lund-Johansen (1965) found that 75% of patients under retiral age returned to work. The figure for those returning to non-manual work (78%) was slightly greater than for those doing heavy (72%) or light manual work (71%). Seamen in particular had difficulty in getting back to work, although the continuation of long term anticoagulant therapy and the need for frequent blood tests for dosage control may have contributed to the difficulties encountered.

Goble, Adey and Bullen (1963) and Seldon (1963) in early reports from the Work Assessment Centre of the National Heart Foundation of Australia described some of the problems encountered by patients after myocardial infarction in attempting to return to work and discussed some of the methods used in dealing with

these problems.

No similar type of establishment exists in this country but Sharland (1964) working in London followed up the survivors of a myocardial infarction and found that 55% had returned to work three months after the infarction. Although most men had returned to their usual employment, changes in occupation were more common in social grades IV and V (General Register Office, 1960). Wincott and Caird (1966) working in Oxford, England, found similar results. Their failure to find any obvious difference physically or psychologically between those who returned to work and those who did not suggested to them that indefinable or irrational factors were operative, and they considered that the main remedy for the social and psychological problems arising after myocardial infarction lay in detailed attention to them from a very early stage of the illness.

The patients in the present series had every opportunity to discuss their problems on numerous occasions and provided that their physical condition permitted it were positively encouraged to return to work about three months after the infarction.

It has been found that of fifty-seven surviving patients below the age of 65 years twelve of the thirty-one patients in Group A and fourteen of the twenty-six patients in Group B had returned to work three months after the incident. The numbers at six months were 22 and 23 respectively (Table X). Another patient in Group A, a 57 year old bank manager, returned to work seven months after the infarction. The unusually long delay in this man with a sedentary occupation was due to psychological factors.

Twenty-five of these patients were being maintained on long term anticoagulants, for the control of which they attended the hospital anticoagulant clinic at approximately monthly intervals.

There is no statistical difference between the two groups in respect of their final return to work records. A significant difference at 4 months and 5 months after infarction suggests that patients in Group B returned to work more rapidly than those in Group A (Table X).

Forty-three of the patients in this series under the age of 65 ostensibly returned to the same work as they had been doing before the incident. Three patients obtained different employment; a labourer obtained a job as gardener in the Corporation of Glasgow Parks Department; a heavy lorry driver obtained employment with the same company driving a light van; a scaffolder reverted to a previous occupation as a plumber (with considerable loss of income as a result).

Details of the eleven patients who did not return to work are given in Table XI. This group is of particular interest and merits further consideration.

Two of the patients had not worked for several

years prior to the infarction, one because of severe deafness and the other for poorly defined medical reasons but not because of cardiac disability.

Two patients retired. Both were 64 year old men who retired a few months prematurely.

Case V was a shepherd who lived in a remote part of the country. On his return to his croft where he lived with his wife, he found he was no longer able to spend long days with his flocks on the hills and he was unable to find regular alternative employment. He did, however, spend some time during the summer months doing light work on the farms.

Case VI was a labourer who was discharged from his employment while convalescing from his infarction. Although fit and keen to return to work he could not persuade his previous employers to re-employ him nor was he able to obtain employment elsewhere.

Case VII sustained a further myocardial infarction five months after the first.

Case VIII had a severe depressive illness following his infarction. This was originally observed while he was still in the ward and took the form of depression, obsessionalism and hypochondriasis. He had always been an athletic individual who had put great store by his physical condition, and was unable to accept the fact that he had sustained an infarction. The mild angina which he experienced was intolerable and seemed to indicate to him that he was no longer able to lead a normal life. After about six months of outpatient attendance in which he had considerable reassurance and sedation, he was referred for psychiatric outpatient treatment. He reacted unfavourably to anti-depressant drugs. Electro-convulsive therapy might have been used if there had been no history of myocardial infarction, but the psychiatrist was unwilling to use this method of treatment in this man. He was treated at the psychiatric day hospital with only slight benefit and two years after his infarction is still depressed

and apathetic.

Case IX was a welder. He had a history of epilepsy and chronic bronchitis, and while in hospital with his infarction he developed a frozen shoulder syndrome. This improved with physiotherapy, but he continued to have angina of effort. He had a considerable distance to walk from his home to public transport and this prevented him from returning to work, although he felt that he would have been fit to do the work involved.

Case X was a docker. He was an alcoholic who was subsequently seen on several occasions at the Casualty Department complaining of chest pain. Usually he was intoxicated, but at no time was evidence of further infarction obtained. He strenuously rejected any suggestion that he should return to work.

Case XI was a coppersmith. He had a difficult personality and while in the ward he was truculent, querulous and aggressive. He continued to complain after discharge and insisted that he was unable to return

to work although clinically he was regarded as having made a good recovery.

Cases V, VI and IX might have been helped to obtain alternative employment but local circumstances and the poor national economic situation prevailing prevented this. If an employer has the opportunity of choosing between two men for a vacancy and one has had a previous myocardial infarction, he will tend to choose the other man. The infarction victim is therefore at a greater disadvantage when there is a large pool of unemployed men looking for work.

Case VIII eventually decided to retire. By doing so, he considerably reduced his mental conflict in that he no longer felt guilty about his inability to return to work.

Twenty-five patients in the two groups under study were maintained on long term anticoagulants after discharge from hospital. Some clinicians have feared that the frequent attendance at hospital that is

necessary for the control of anticoagulant therapy might prejudice chances of returning to work and of holding down a job. However all of these twenty-five patients had returned to work within six months of the infarction.

The figures concerning return to work quoted here correspond closely to the previous studies mentioned. This may represent the proportion of patients who can be expected to return to work in the majority of series. While it may be possible to achieve an 80% success rate for return to work in the average group of patients of working age there is a sizeable group who for a variety of reasons do not return to work. Nor is this group confined to the lower social grades, and in the present series there is no preponderance of unskilled patients in the group who did not return to work. Some of the reasons may be irreversible, such as severe cardiac damage leading to continued symptoms and impaired effort tolerance. There will also be external factors over which the doctor may have little control, such as the local labour situation, the general economic

situation, the patient's capacity for retraining and the attitude of employers. But much can be done with regard to the patient's attitude to returning to work, the influence of wives and relatives and the prejudice of employers (Hellerstein & Ford, 1960). Many of these attitudes and those of doctors are relics of the experience people had with myocardial infarction in the years after it was originally described and when many considered that if the patient recovered from the acute attack, useful life would no longer be possible (Parkinson & Bedford, 1928). In the long run, success or failure in rehabilitation will depend on the interest and enthusiasm of the physician (Katz et al, 1956). In this study every effort was made to encourage patients to return to work. A close rapport was established with the patient in most cases while he was in the ward and subsequently as an outpatient. Discussion about the problems of work, the illness and its sequelae was encouraged. It was hoped that in this way many anxieties could be removed.

Those who had to return to heavy occupations were advised not to over-exert themselves, and most reported that they were able to accomplish some re-organisation of their work to follow this advice. Often an unofficial arrangement exists in such occupations whereby younger and fitter men protect older men from the heavier jobs and gradually the patient finds what he can do and what he should avoid. Employers will sometimes co-operate by allowing patients to readjust gradually, perhaps by starting later and finishing earlier (Brit. Med. J., 1964). However, in advising men to avoid heavy lifting and severe exertion, it is important not to cast the seeds of doubt that the individual has not in fact made a good recovery.

Cook et al (1962) suggested that return to work adversely affects longevity. It has been found in this series that only two of the patients who returned to work sustained a further infarction and one of them died. This was a man who had sustained two infarctions prior to the

one which admitted him to the trial. There has been no other death in this group, which has now been followed up for periods of up to three years. Sharland (1964) did not find that return to work affected prognosis, and it would seem more reasonable that fitter men return to work, and that the prognosis for them is better than for those who do not return (Lovell, 1964). It would seem that an optimistic approach is justifiable in the management of ischaemic heart disease (Plotz, 1957). Honey and Truelove (1957) have shown that the chances of a sixty year old man surviving five years after leaving hospital after a myocardial infarction are as good as for the general population. Dubnow et al (1965) quote five year survival rates of 79% for those who survive the original infarction and Sigler (1962) has indicated that longevity is a relatively common occurrence.

Dr. Paul D. White (quoted by Plummer, 1956) stated "I would like to emphasise the beneficial effect of work on mind and soul of any occupation in which it

is possible for a cardiac patient to engage. Idleness breeds unhappiness and is actually bad for the health. It is a rare patient indeed who is fit for nothing. It greatly pays to make every effort to find something vocational or avocational into which to fit the sick man or woman".

Patients still find problems in returning to work and great benefits could result from an organised approach, for example, through Work Assessment Clinics such as have existed in the United States for many years (Ruskin, 1964), and as Goble et al (1963) describe. In this way the individual problems of patients physical and psychological, in relation to past and prospective employment could be evaluated, and the patient could be advised and encouraged. While no attempt would be made to act as an employment agency, employers could be contacted directly and the short and long term working potential of the man discussed. An alternative scheme is proposed in Scot. med. J. (1967). It is suggested that the substantial

rehabilitation and consultative services which already exist are not adequately used and that these might help to produce a solution, together with the active participation of general practitioners.

Either type of approach would be very useful and should substantially increase the prospects of rehabilitation. The day must soon come when no country can neglect this important area and no medical centre will be complete without a full rehabilitation programme, which will reach into the homes of all, rich and poor alike. In this way not only will we make people well, but well and useful members of the community (Katz et al, 1958).

Psychological Disturbance and Personality Testing.

One of the objects of this trial was to determine if a shorter and less strict bed rest regime might be beneficial in reducing the amount of neurotic disturbance which occurs after myocardial infarction. Levine (1951) indicated that patients felt something ominous and fore-

boding, when compelled to lie absolutely quiet in bed for a month or longer and was impressed with the psychological well-being of patients treated by the arm-chair method. The difference in the psychological state of patients was seen readily in those who had a previous attack and were treated on that occasion with a strict bed rest regime. Beckwith et al (1954) also noticed a decrease in anxiety and a feeling of well-being in patients treated out of bed.

In discussion with the earlier ambulated patients in this trial similar conclusions have been reached. Some of these patients had previously sustained infarctions when they were treated with a long period of bed rest and kept in hospital for periods of six to eight weeks. They were unanimous that they preferred the regime of earlier mobilisation and stated that they felt their recovery had been more rapid and that they felt more optimistic.

These are only subjective impressions, but it

is important that this regime was readily accepted by this group of patients and most preferred it to the regime of which they had had experience previously.

When the results are examined in respect of overt psychological disturbance it is seen that this occurred in the same number of patients in each group while in hospital and in closely similar numbers of patients after return home. Most of these upsets were of a minor nature and consisted of feelings of tension, insomnia and hypochondriasis. Two patients developed more severe depressive illness. One of these was in the younger age group and was one of those who did not succeed in returning to work (Case VIII above). The other was an older man who required psychiatric admission and made a fair recovery with small doses of anti-depressant drugs. Taking the two groups as a whole anxiety or depressive symptoms occurred while in hospital in 9.5% of all patients in the trial and in 10.4% of the survivors when those deaths occurring in

the first forty-eight hours after admission have been excluded. During the first year after the infarction eighteen of the eighty-four survivors (21%) were found to have some degree of anxiety or depression. Again there is no statistically valid difference between the two groups. Personality testing using the Eysenck Personality Inventory which measures neuroticism as an index of emotional instability revealed differences between the infarct patients and their controls, but there is no significant difference in the index between those infarct patients in Group A and those in Group B although there is a trend towards lower scores both at the time of discharge and at follow up in the patients in Group B.

The psychological reactions to a myocardial infarction are of the greatest interest and have to be understood to permit adequate handling of the patient (Hellerstein & Ford, 1960). Fear is understandable. The symbolic importance of the heart is great. It is a vital organ, and its damage is a colossal threat to the

individual. Most people now are well aware of the results of myocardial infarction in terms of mortality and their experiences of the disease when they were younger and perhaps even in recent years fills them with foreboding regarding their own recovery and economic independence.

It has been the experience in work classification clinics that the psychological reactions of patients to myocardial infarction are prominent in almost every case, and in many cases constitute the major disability (Rosenbaum and Belknap, 1959). During the period after infarction, the patient requires support and reassurance from all of those around him and if he receives this, severe psychological disturbance might be averted (Caplan, 1959). Although it must be admitted that no particular psychological advantage has resulted from the regime of earlier ambulation, both groups had considerable explanation, reassurance and encouragement. And yet 21% had some degree of psychological disturbance in

the year following the infarction. Fry (1967) has recently discussed this problem from the point of view of the general practitioner and believes that the psychological disturbance which follows myocardial infarction is closely related to the patient's management in hospital. While this is certainly not the entire reason, all doctors must ask themselves what effect is the treatment which a patient experiences in hospital, going to have on a successful readjustment after return home and is everything being done to cause minimum psychological trauma?

THE MANAGEMENT OF MYOCARDIAL INFARCTION

CHAPTER III

THE EFFECT OF CHANGE OF POSTURE ON CARDIAC OUTPUT AFTER MYOCARDIAL INFARCTION

As part of the investigation into the effects of earlier mobilisation, the cardiac output changes involved in sitting a patient up after a myocardial infarction have been examined.

The estimation of cardiac output in i il patients has presented numerous problems. The technique should be capable of being carried out frequently and at the bedside.

A convenient method is the photo-electric earpiece technique for the recording of dye dilution curves. This eliminates the necessity for cardiac catheterisation and accurate estimation of oxygen consumption which is required by methods which depend on the Fick Principle, and obviates the necessity for repeated arterial puncture which has previously been required for dye dilution techniques. Stewart (1897) first showed that it was possible to estimate cardiac output by injecting an indicator substance into a vein and by repeated and rapid sampling of its concentration

in the arterial circulation. Hamilton (1932) demonstrated the successful application of the method in man and subsequently showed that there was a fair correlation between indicator dilution methods and Fick Principle methods (Moore, Kinsman, Hamilton and Spurling, 1929; Hamilton, Riley, Attyah, Cournand, Fowell, Himmelstein, Noble, Remington, Richards, Wheeler, Witham, 1948) and this has been confirmed more recently by Miller, Gleason and McIntosh (1962) who found an average difference of only 4%.

The introduction of what appeared to be a stable non-toxic substance (Coomassie Blue, I.C.I.) which does not cause staining of the skin and which can be easily estimated in plasma (Taylor and Shillingford, 1959; Taylor and Thorpe, 1959) made it possible to carry out repeated estimations with a minimum of disturbance to the patient when combined with the photo-electric earpiece method (Gabe and Shillingford, 1961; Bruce and Shillingford, 1962; Gabe, Tuckman and

Shillingford, 1962) and it is this technique which is described here and which has been used in the experiments to be described.

Gabe and Shillingford (1961) have demonstrated the excellent reproducibility of successive cardiac output estimations using this technique and the close correlation between results obtained with the photo-electric earpiece and the arterial cuvette (Table XII). Problems with the stability of Coomassie Blue have been encountered by some workers and the dye is no longer commercially available, but these do not affect the validity of the results reported here.

MATERIALS AND METHODS.

Three groups of subjects have been examined.

Group 1 - Ten patients without evidence of cardiac disease (normal controls). These were patients who were in hospital for non-cardiac reasons, normotensive without evidence of valvular disease of the heart

and without electrocardiographic or radiological evidence of heart or chest disease. Their ages ranged from 20 to 68 years (mean 35.4, SD = 16.2). There were eight males and two females in the group.

Group 2 - Eleven patients who had sustained a myocardial infarction within the three weeks prior to the experiment, but in whom there was neither clinical nor radiographic evidence of pulmonary oedema. These patients were all male and their ages ranged from 38 to 65 years (mean 55.4, SD = 9.6).

Group 3 - Ten patients who had sustained a myocardial infarction and in whom there was clinical and/or radiographic evidence of pulmonary oedema. Their ages ranged from 47 to 67 years (mean 55.5, SD = 8.1) and all were male.

The diagnosis of myocardial infarction was made on the basis of clinical history, electrocardiographic and biochemical changes as described previously (Chapter II).

All patients were examined supine (with one pillow as a head rest) and propped up at an angle of 45° in their own beds with legs horizontal. Alternate subjects were examined in the supine position first, or in the 45° head elevated position first. Ten subjects (normal controls) were examined on a tilting table, supine and in the 45° head elevated position with legs horizontal. The procedure adopted is shown in Figs. 1a and 1b. All observations were carried out more than two hours after a meal and with the patient rested but not sedated. Five minutes were allowed to elapse after a change of position to allow haemodynamic equilibrium to occur. Cardiac output was measured by the dye dilution technique (Gabe and Shillingford, 1961; Thomas, Malmcrona and Shillingford, 1965)

using a selenium photo-electric earpiece (Cambridge Instrument Coy.) and Coomassie blue dye (I.C.I.). A polythene catheter (Intracath. 24" long, 14G) was inserted percutaneously into a median antecubital vein and advanced with the object of leaving the tip as near the great veins as possible. Between dye injections the patency of the catheter was maintained by a slow infusion of dextrose water with 1000 u. heparin added per 540 ml. 40 mg. of dye (2 ml.) were injected as a bolus through the catheter from a plastic insulin syringe and this was followed immediately by a flushing dose of 10 ml. dextrose in water to clear the catheter. Duplicate or triplicate measurements were made in each position. Dye curves were drawn from a steady baseline on a Cambridge Mark II Dye Recorder using the Cambridge photo-electric earpiece (Fig. 2). The first dye curve (Fig. 3) was calibrated by the tail height method. The tail height was measured three minutes after the injection and at this time a blood sample was removed

for estimation of plasma dye concentration and estimation of packed cell volume, which was performed using a Hawksley micro-haematocrit in duplicate (Dacie and Lewis, 1963). Coomassie Blue was measured by the method suggested by the manufacturers of the dye. Proteins are precipitated and the density of the filtrate compared against a known control at 585 μ. in a spectrophotometer (Unicam S.P. 600).

The area of the curve was measured by planimetry after semi-logarithmic plotting and extrapolation of the down slope to the base line and replotting on a linear scale. Relative cardiac outputs were calculated from the reciprocal of the areas of the extrapolated dye curves. Cardiac output was calculated from the formula

$$C.O. = \frac{60i}{A}$$

where C.O. = cardiac output in litres per min., i = dose of dye injected (mg.) and A = area of the curve. Heart rate was derived from the dye curve by using slightly

incomplete pulse rejection on the dye recorder. Statistical analysis has been carried out according to Hill (1967).

RESULTS

The results obtained are tabulated in Tables XIII - XXII.

In general, the range of cardiac output estimations agrees with the estimations of other workers, both for the infarct patients and for the normals (Gilbert, Goldberg, Griffin, 1954; Murphy, Glick, Schreiner, Yu, 1963; Nager, Thomas & Shillingford, 1967).

NORMAL CONTROLS (10 Subjects) - GROUP I

Cardiac outputs in this group ranged from 4.2 to 10.3 litres per min. (mean 6.8, S.D. = 2.4) in the supine position and from 4.5 to 11.2 litres per min. (mean 7.2, S.D. = 2.4) when propped up at 45° as shown. Pulse rates varied from 60 - 96 per min. (mean 71.4, S.D. = 13.3) supine and from 60 - 96 per min. (mean 71.6, S.D. = 12.7) when propped up. Stroke volume ranged from 51 to 160 ml. (mean 96.3, S.D. = 31.7) when supine and 51 to 148 ml. (mean 100.7, S.D. = 27.3)

when propped up.

POST-INFARCTION GROUP (No pulmonary oedema, 11 Subjects) - GROUP 2

The cardiac outputs of this group ranged from 3.7 to 8.4 litres per min. (mean 6.1, S.D. = 1.5) when supine and from 3.7 to 8.6 litres per min. with head elevated (mean 5.8, S.D. = 1.3). Pulse rate (supine) ranged from 60 to 88 per minute (mean 72.1, S.D. = 8.6) and with elevation ranged from 60 to 90 per minute (mean 73.1, S.D. = 9.9). Stroke volume (supine) was from 46 ml. to 120 ml. (mean 85.3, S.D. = 22.8) and stroke volume with head elevated was from 45 to 114 ml. (mean 80.5, S.D. = 19.3).

POST-INFARCTION PATIENTS (With Pulmonary oedema, 10 Subjects) - GROUP 3

The cardiac output (supine) of this group ranged from 2.2 to 6.4 litres per min. (mean 5.0, S.D. = 1.2) and with head elevated ranged from 2.7 to 8.5 litres per min. (mean 6.0, S.D. = 1.2). Pulse rate in this group ranged from 68 to 92 per min. supine (mean 77.6,

S.D. = 7.9) and from 70 to 96 in the elevated position (mean 79.6, S.D. = 9.4). Stroke volume ranged from 28 to 86 ml. supine (mean 64.3, S.D. = 17.1) and from 33 to 115 ml. in the 45° elevated position (mean 77, S.D. = 23.7).

DISCUSSION

Gabe and Shillingford (1961) (Table XII) have demonstrated the reproducibility of successive cardiac output estimations, using the technique upon which the technique described in this work has been modelled. Two injections of dye were made in each position in each subject, but in two subjects three injections were made in each position. Tables XIII - XV show the planimetry readings for each curve and the other relevant data necessary for the calculation of the cardiac output. The planimetry readings are also expressed as proportions of the initial estimation in a single position (Tables XVI - XVIII). The mean difference between the second and the first estimation is -3%. 95% of second observations will be within the

range -17% and +11% of the first observation (t_{65} at 5% probability level = 2).

It can be concluded from this analysis that the technique provides reproducible results and that the changes which have been detected and which are significant are the result of the positional change and not due to instability of the technique.

A further safeguard has been that alternate subjects were examined in the supine or in the head elevated position first.

The Law of the Heart (Starling, 1915) states that, within physiological limits, an increase in diastolic volume results in a greater energy of contraction with a greater amount of chemical change at each contraction.

The cardiac output has been related to venous pressure (Markwelder and Starling, 1914; Patterson, Piper and Starling, 1914; Patterson and Starling, 1914; McMichael and Sharpey-Shafer, 1944), and has been

shown to be less in the erect position than in the recumbent (McMichael, 1937; McMichael and Sharpey-Schafer, 1944; Weissler, Leonard and Warren, 1957; Wang, Marshall and Shepherd, 1960). Starr and Rawson (1941) using the technique of ballistocardiography showed that the response to tilting in normal subjects is variable and demonstrated that a similar variability could be found in many of the previously published reports. Starr and Rawson (1941) estimated cardiac outputs between one and two and a half minutes after putting their subjects into the vertical position, but most of the other reports do not record the time interval which elapsed between the change of position and the output estimation.

McMichael and Sharpey-Schafer (1944), in one of the first large catheterisation studies in this country, showed that the arterio-venous oxygen difference of 40 patients increased from 4.1 to 6.1 vols. per cent with the change from the supine to the erect position, and calculated, using Fick Principle methods, that

cardiac output must have fallen an average of 1.5 litres per min. with the change.

Stead, Warren, Merril and Brennan (1945) studied the effect of passive tilting from the recumbent to the 70° head-up position. This produced a decrease of cardiac output in each of six subjects, the mean fall being 0.8 litres/min./sq. metre of body surface. Nowy, Kikodse and Zollner (1957) using a dye dilution method found a mean fall in cardiac output of 30% (range 11-49) with the change from lying to standing. Similar findings have been recorded using the dye dilution technique by Chapman, Fisher and Sproule (1960), Wang, Marshall and Shepherd (1960) and Reeves, Grover, Blount and Filley (1961). Levine (1940, 1944, 1950) suggested that the work of the heart might be reduced if patients suffering from cardiac failure were propped up in bed or treated out of bed in an armchair. Donald, Wade and Bishop (1952) using Fick Principle methods studied the nursing position of

thirty-six patients and found that changes in position resulted in a significant change in cardiac output but concluded that these changes were unlikely to be of much benefit to any patient. Coe (1954) using a dye dilution technique showed that cardiac work was less while patients were seated by the bedside (mean decrease of 23%) and similar results were obtained by Atuk, Beckwith and Wood (1959).

It would appear therefore that changes in the cardiac output can be produced by changes in position and that the load on the heart after myocardial infarction can be reduced by sitting patients in bed.

Howarth, McMichael and Sharpey-Schafer (1946) demonstrated that in cardiac failure with low cardiac output, the output of the heart was increased by venesection and that calculation of cardiac work showed an increase in cardiac work in all congested cases. One might anticipate therefore that if the venous return is reduced in patients with failing hearts, the heart might

respond with an increase in cardiac output as happened in Starling's original experiments on the heart lung preparation. The clinical benefit of allowing patients with heart failure to sit up is accepted generally even by those who prefer their non-congested patients to be nursed lying flat.

There has recently been a renewal of interest in the effect of posture on cardiac output. This has been in part due to Richards' (1955) doubts as to the validity of Starling's law of the heart in the intact animal and in man and Sarnoff's concept of a series of left ventricular function curves of the Starling type (Sarnoff and Berglund, 1954; Sarnoff, 1955).

In the present investigation, it has been found that there were small but insignificant differences in sitting and supine cardiac outputs in the group of normal controls and in the group of patients who had sustained a myocardial infarction and in whom there was no evidence of cardiac decompensation. There was almost no change

in pulse rate associated with the postural change and little change in stroke volume. It would appear that the postural change involved is less than when the whole individual is tilted to the 45° head up position which has often been reported to produce a fall in cardiac output.

In the group of normal controls, it was found that in six subjects, cardiac output was greater in the propped up position, less in two patients and in two patients there was no change. The mean change was 0.4 litres/min. but this change was not significant at the 0.05 level.

The group of infarct patients, who did not show evidence of pulmonary oedema, demonstrated a small difference in supine and propped up cardiac output (mean difference 0.3 litre/min.). This change falls just short of the 0.05 level of significance. Seven of these patients had a smaller cardiac output in the seated position than in the supine, two had a larger

output, and two showed no change.

Ten patients were examined after myocardial infarction at a time when they showed evidence of pulmonary oedema. The cardiac output in nine of these patients was greater in the seated position than in the supine and the tenth patient showed a fall in output. The mean difference was 1.00 litres/min. and this was statistically significant ($p<.01$).

The rise in output is the result of a rise in stroke volume (mean change of 15.8 ml.) and according to Starling's theory, is due to decreasing the load on the heart by putting the patient into a sitting posture and thus reducing venous return. Nager, Thomas and Shillingford (1967) state that stroke volume is a more representative measurement of the ability of ventricles to contract than cardiac output and it would appear that ventricular contractility has been improved by placing the patient in the 45° head elevated position.

The mean cardiac outputs and mean stroke volumes of the normal controls were higher than those of the infarct patients although this might be accounted for by the lower ages of the normal controls (Wade & Bishop, 1962). The mean cardiac outputs and mean stroke volumes of the infarct patients with pulmonary oedema were lower than those of the patients who did not have pulmonary oedema. These differences observed are not significant at $p = 0.05$.

These observations indicate that the cardiac output of normal subjects and of patients who have sustained a myocardial infarction but who do not show evidence of pulmonary oedema, is only slightly influenced by the change in posture involved in moving from the supine to the sitting position. The changes induced are not statistically significant and are unlikely to be of importance in the management of the individual patient. Significant changes are seen in the group of patients who have sustained a myocardial infarction and who have

radiological evidence of pulmonary oedema. The mean cardiac output of this group is significantly greater when sitting in bed than when lying supine. Nine of the ten patients in this group had a greater cardiac output when sitting than when supine. The increase in cardiac output is considered to arise in the way that Starling (1915) has suggested for the denervated heart by a reduction in venous return associated with the change of posture. The increase in output might be of clinical significance in promoting clearing of pulmonary oedema and improving tissue perfusion.

THE MANAGEMENT OF MYOCARDIAL INFARCTION

CHAPTER IV

SUMMARY AND CONCLUSIONS

An investigation into the effects of earlier mobilisation of patients who have sustained a myocardial infarction is presented. Two comparable groups of male patients have been studied. One group has been treated with a strict bed rest regime, and has been nursed in bed for twenty-five days, after which gradual mobilisation was undertaken over a period of ten days. The other group was treated for fourteen days in bed during which considerable freedom of activity was allowed and then mobilised over a period of seven days in hospital with discharge after 21 days.

The two groups of patients were comparable in terms of age, sex, duration and severity of illness and previous history of infarction. It has been found that the early mobilisation programme has not been deleterious to the patients in respect of mortality or morbidity or the development of serious arrhythmia or other complications of infarction; nor has this programme increased the incidence of aneurysm formation in the earlier mobilised group. The incidence of neurotic reaction in the two

groups was not significantly different, when this was assessed in hospital and after discharge, nor were significant differences detected when the groups were tested psychologically using the Eysenck Personality Inventory. The earlier mobilised group has been found to return to work more rapidly than the other group, but after six months no significant differences were found in the numbers returning to work between the two groups.

Cardiac outputs have been estimated using a dye dilution technique on three groups of patients in the supine and in the 45° head elevated position, corresponding to the position in which a patient might be nursed sitting in bed. The three groups were -

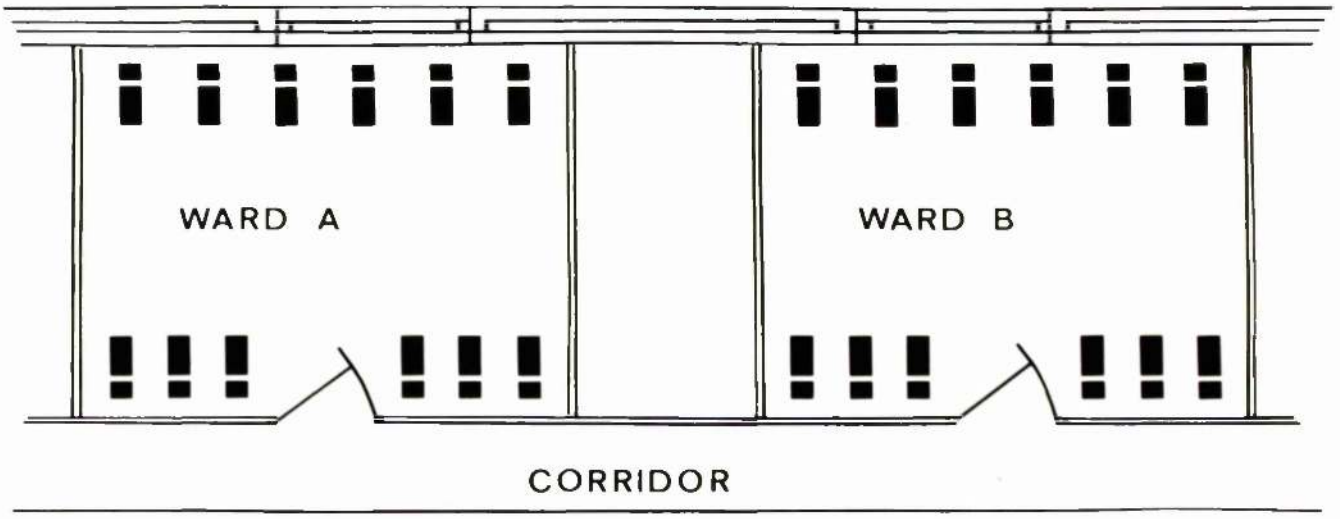
(a) 10 subjects without evidence of cardiac or respiratory disease.

(b) 11 patients who had sustained a myocardial infarction and in whom there was no evidence of pulmonary oedema.

(c) 10 patients who had sustained a myocardial infarction and in whom there was evidence of pulmonary oedema.

The mean cardiac output of the patients in whom there was evidence of pulmonary oedema has been found to be significantly higher in the seated position than in the supine position, but significant differences were not found in either of the other two groups.

It is concluded from these studies that a regime of earlier mobilisation and greater activity of infarct patients, while being treated in bed, has not been harmful and may assist in the rehabilitation of patients. The cardiac output of patients who have no pulmonary oedema is not significantly different if they are supine or seated, but the cardiac output of patients with pulmonary oedema is higher when the patient is nursed seated. It is concluded that there is no justification in insisting on a strict bed rest regime for patients who have sustained a myocardial infarction.



STAFF

Resident Physician

Registrar

Sister

Staff Nurse

Student Nurses

Auxiliary Nurses

Consultant Physician A Consultant Physician B

WARD AND STAFFING ARRANGEMENTS, WARD 20

SOUTHERN GENERAL HOSPITAL

Appendix A:

Management

	GROUP A	GROUP B
1st week	Total bed rest. Allowed one pillow. Fed and washed by a nurse (for 3 days). Bed pan (or commode).	Assumes comfortable position in bed. Feeds and washes himself. Bed pan (or commode).
2nd week	As above. Allowed two pillows.	As above.
3rd week	Assumes comfortable position in bed. As above.	Allowed to swing legs, gradually mobilised and granted toilet privileges.

Appendix B: Nursing procedure.

4th week	As above. Mobilisation commences on 25th day.	Home on 22nd day.
5th)) weeks 6th)	Progressive mobilisation, toilet privileges and allowed home on 36th day.	

Notes.

1. One patient in Group A and eight patients in Group B had some minor modification of the procedure because of persisting chest pain, recurrence of pain, hypotension or heart failure.
2. Conventional treatment with oxygen, diuretics, digoxin and anti-arrhythmic drugs were given as indicated.

Appendix B: Nursing procedure (contd.)

3. 34 patients in Group A and 31 patients in Group B received anticoagulant therapy (commencing with heparin and continuing with warfarin sodium) while in hospital. This was continued after discharge in 15 patients in Group A and 11 patients in Group B for periods up to two years (M.R.C., 1964).

Anticoagulant therapy was withheld from patients with a history of dyspepsia, renal failure, alcoholism or other haemorrhagic risk.

Appendix B: Nursing procedure (contd.)

EYSENCK PERSONALITY INVENTORY

by H. J. Eysenck and Sybil B. G. Eysenck

PERSONALITY QUESTIONNAIRE

FORM A

NAME..... AGE.....

OCCUPATION..... SEX.....

N= ☐ E= ☐ L= ☐

Instructions

Here are some questions regarding the way you behave, feel and act. After each question is a space for answering "YES" or "NO".

Try to decide whether "YES" or "NO" represents your usual way of acting or feeling. Then put a cross in the circle under the column headed "YES" or "NO". Work quickly, and don't spend too much time over any question; we want your first reaction, not a long-drawn out thought process. The whole questionnaire shouldn't take more than a few minutes. Be sure not to omit any questions.

Now turn the page over and go ahead. Work quickly, and remember to answer every question. There are no right or wrong answers, and this isn't a test of intelligence or ability, but simply a measure of the way you behave.

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E ○ N ○ L ○ | | | | | | | | | | |

FORM A

		YES	NO
1.	Do you often long for excitement?	○	○
2.	Do you often need understanding friends to cheer you up?	○	○
3.	Are you usually carefree?	○	○
4.	Do you find it very hard to take no for an answer?	○	○
5.	Do you stop and think things over before doing anything?	○	○
6.	If you say you will do something do you always keep your promise, no matter how inconvenient it might be to do so?	○	○
7.	Does your mood often go up and down?	○	○
8.	Do you generally do and say things quickly without stopping to think?	○	○
9.	Do you ever feel "just miserable" for no good reason?	○	○
10.	Would you do almost anything for a dare?	○	○
11.	Do you suddenly feel shy when you want to talk to an attractive stranger?	○	○
12.	Once in a while do you lose your temper and get angry?	○	○
13.	Do you often do things on the spur of the moment?	○	○
14.	Do you often worry about things you should not have done or said?	○	○
15.	Generally, do you prefer reading to meeting people?	○	○
16.	Are your feelings rather easily hurt?	○	○
17.	Do you like going out a lot?	○	○
18.	Do you occasionally have thoughts and ideas that you would not like other people to know about?	○	○
19.	Are you sometimes bubbling over with energy and sometimes very sluggish?	○	○
20.	Do you prefer to have few but special friends?	○	○
21.	Do you daydream a lot?	○	○
22.	When people shout at you, do you shout back?	○	○
23.	Are you often troubled about feelings of guilt?	○	○
24.	Are *all* your habits good and desirable ones?	○	○
25.	Can you usually let yourself go and enjoy yourself a lot at a gay party?	○	○
26.	Would you call yourself tense or "highly-strung"?	○	○
27.	Do other people think of you as being very lively?	○	○

		YES	NO
28.	After you have done something important, do you often come away feeling you could have done better?	○	○
29.	Are you mostly quiet when you are with other people?	○	○
30.	Do you sometimes gossip?	○	○
31.	Do ideas run through your head so that you cannot sleep?	○	○
32.	If there is something you want to know about, would you rather look it up in a book than talk to someone about it?	○	○
33.	Do you get palpitations or thumping in your heart?	○	○
34.	Do you like the kind of work that you need to pay close attention to?	○	○
35.	Do you get attacks of shaking or trembling?	○	○
36.	Would you always declare *everything* at the customs, even if you knew that you could never be found out?	○	○
37.	Do you hate being with a crowd who play jokes on one another?	○	○
38.	Are you an irritable person?	○	○
39.	Do you like doing things in which you have to act quickly?	○	○
40.	Do you worry about awful things that might happen?	○	○
41.	Are you slow and unhurried in the way you move?	○	○
42.	Have you ever been late for an appointment or work?	○	○
43.	Do you have many nightmares?	○	○
44.	Do you like talking to people so much that you never miss a chance of talking to a stranger?	○	○
45.	Are you troubled by aches and pains?	○	○
46.	Would you be very unhappy if you could not see lots of people most of the time?	○	○
47.	Would you call yourself a nervous person?	○	○
48.	Of all the people you know, are there some whom you definitely do not like?	○	○
49.	Would you say that you were fairly self-confident?	○	○
50.	Are you easily hurt when people find fault with you or your work?	○	○
51.	Do you find it hard to really enjoy yourself at a lively party?	○	○
52.	Are you troubled with feelings of inferiority?	○	○
53.	Can you easily get some life into a rather dull party?	○	○
54.	Do you sometimes talk about things you know nothing about?	○	○
55.	Do you worry about your health?	○	○
56.	Do you like playing pranks on others?	○	○
57.	Do you suffer from sleeplessness?	○	○

PLEASE CHECK TO SEE THAT YOU HAVE ANSWERED *ALL* THE QUESTIONS

		Group A		Group B		Simple X^2 Test	p<	
Number		55		50				
Mean age (years)		59.7		58.4				
		S.D. 9.19		S.D. 9.76				
Deaths		12 (22%)		9 (18%)		0.239	.50	NS
Condition on admission								
Hypotension		5)		7)				
Heart failure		11)	21	15)	27			
Shock		5)		5)				
History of previous	x 1	7		8				
infarction	x 2	4		2				
	x 3	0		1				
Peel Prognostic Index								
mean		8		10		*D = 0.06		NS

Table I Comparison of the two groups in respect of number of patients, mortality rate, previous record of infarction, condition on admission.

* Kolmogorov-Smirnov Test (Siegel, 1956). To be significant at .05, D would have to be greater than 0.43.

NS = not significant.

	1st Infarction	Deaths	Recurrent infarctions	Deaths
Group A	44	8	11	4
Group B	39	6	11	3

Table II Details concerning mortality and primary or recurrent infarction.

There is no significant difference in deaths occurring in the two groups A & B ($p<0.5$). There is a trend towards significance when deaths occurring in patients with a history of previous infarction are compared with deaths occurring in patients without a previous history of infarction ($p<.10$).

	Number of Deaths	
Time after admission	Group A	Group B
Within 24 hours	3	4
24 - 48 hours	0	1
48 hours - 7 days	1	0
8 - 14 days	6	0
15 - 21 days	0	1
22 - 35 days	2	3

Table III The times at which the deaths occurred in the two groups.

Note: Patients with protracted chest pain, prolonged hypotension or shock or in failure were kept in bed and in hospital for longer periods. This explains deaths occurring in Group B more than three weeks after admission. See text.

	Group A	Group B	Simple X^2	p<
Number in group	55	50		
Further pain	15	16	0.281	.50
Heart failure, hypotension or shock developing after admission	11	11	0.063	.80
Arrhythmia	18	19	0.319	.50
Possible aneurysms (Bulges & abnormal pulsation)	14 (39 examined)	6 (36 examined)	3.540	.20

Table IV Complications occurring in patients in each group.

No significant differences are detected.

		Group A	Group B
Extrasystoles	Ventricular	6	7
	Nodal	1	1
Sinus tachycardia		3	2
Atrial fibrillation		2	4
Sinus bradycardia		1	0
Heart Block			
Potential a-v block		1	2
Partial a-v block		1	0
Complete heart block		1	1
Bundle branch block (L)		1	2
Electrical alternans		1	0
		18	19

Table V Details of arrhythmias detected clinically and electrocardiographically.

	Group A	Group B	Simple X^2	p<
At hospital	5	5	.025	.90
At home	10	8	.012	.90
	(43 assessed)	(36 assessed)		

Table VI **Incidence of psychological disturbance occurring in the two groups in hospital and after returning home.**

No significant differences are found.

	Group A				Group B			
	Hospital		1 year later		Hospital		1 year later	
	mean	S.D.	mean	S.D.	mean	S.D.	mean	S.D.
N EPI	9.87	4.51	11.04	4.88	7.60	5.07	9.33	3.95
E EPI	11.52	4.17	10.74	3.96	13.20	4.00	11.73	4.44

Table VII **Results of psychological testing.**

No significant differences are found.

(Mann Whitney "U" test (Siegel, 1956)).

	Group A	Group B	Total
Bulges	9	6	15
Abnormal pulsation	5	0	5
Pleuro-pericardial adhesion	6	5	11
L.V. Enlargement[1]			9
Coronary calcification[2]			8
Pulmonary oedema[3]			4
Pulmonary infarction[4]			2
Total examined	39	36	75

Table VIII **Radiological abnormalities detected.**

1, 2, 3, 4 not divided into groups because of small numbers.

Social Grade	1	2	3	4	5
No.	0	5	28	12	14

Table IX **Social Grading of patients in series below the age of 65.**

2 patients not included since they had not worked for long periods before infarction.

Grading according to General Register Office Classification of Occupations, 1960.

Time after infarction	Group A	Group B	Simple X^2	p<	
3 months	12	14	1.306	.30	NS
4 months	16	21	5.278	.05	S
5 months	16	21	5.278	.05	S
6 months	22	23	2.604	.20	NS

Table X **Number of patients who had returned to work at different stages after the infarction.**

The results indicate a significant difference 4 and 5 months after the infarction and suggest that patients in Group B tended to return to work more rapidly than patients in Group A. Significance is lost at six months.

S = Significant. NS = Not significant.

Case No.	Age	Previous Occupation	Reason for non-return
Case I	53	Had not worked for many years	Deaf
Case II	57	Had not worked for two years before infarction	
Case III	64	Shipbuilder	Retired
Case IV	64	Welder	Retired
Case V	60	Shepherd	Unable to find alternative employment
Case VI	54	Labourer	
Case VII	44	Welder	Re-infarction (5 months)
Case VIII	63	Fitter	Depression
Case IX	54	Welder	Unable to cope with travelling involved
Case X	45	Docker	Personality
Case XI	62	Coppersmith	Personality

Table XI Details of patients under age of 65 who did not return to work.

Subject	Order of Estimation			
	1	2	3	4
J.B.	1.00	1.04	1.01	1.02
A.B.	1.00	1.08	0.98	1.10
F.G.	1.00	1.12	1.03	1.05
H.A.	1.00	0.99	1.00	1.01

Table XII **Successive estimations of Cardiac output in normal resting subjects, expressed as proportions of the initial determinations.** Extracted from Gabe and Shillingford (1961).

Subject	Planimetry readings: Supine 1	Supine 2	45° 1	45° 2	Coomassie blue (mg./litre plasma)	Tail height (3 min.)	PCV (mean of two estimations)
J.R.	231	216	200	200	15.6	10	41
A.S.	134	133	133	136	9.9	11	43
E.B.	195	191	200	190	15.4	6	42
J.J.	239	251	205	202	18.4	3	39
E.K.	267	228	210	237	19.7	10	47
J.B.	198	163	162	187	12.4	10	46
I.C.	312	277	289	285	17.8	12	42
J.P.	197	172	185	151	16.6	13	47
J.D.	200	168	198	190	12.2	10	45
A.W.	266	258	218	222	13.7	6	44

Table XIII Planimetry readings, 3 min. Coomassie blue levels, tail height and packed cell volumes.

Subjects without evidence of cardiovascular disease.

Subject	Planimetry readings: Supine 1	Supine 2	45° 1	45° 2	Coomassie blue (mg./litre plasma)	Tail height (3 min.)	PCV (mean of two estimations)
C.K.	188	193	201	175	12.5	8	40
G.A.	127	127	122	124	14.5	4	39
G.B.	206	242	210	223	21.0	7	45
A.C.	320	324	304	283	20.1	11	52
A.V.	148	152	164	154	13.0	7	42
A.F.	403	395	320	288	16.7	11	46
A.T.	266	236	250	234	16.4	10	47
J.G.	243	244	254	248	17.0	10	45
A. McG.	395	374	454	430	13.7	12	45
W.S.	327	299	341	323	8.6	9	44
G.R.	297	264	287	310	7.9	5	42

Table XIV Planimetry readings, 3 min. Coomassie blue level, tail height and packed cell volumes.

Subjects after myocardial infarction, no evidence of pulmonary oedema.

Subject	Planimetry readings: Supine 1	Supine 2	Supine 3	45° 1	45° 2	45° 3	Coomassie blue (mg./litre plasma)	Tail height (3 min.)	PCV (mean of two estimations)
J.R.	314	361		208	227		15.6	10	41
A.F.	385	344		334	313		11.5	10	42
R. McI.	190	217	189	166	180	171	13.0	8	37
W.E.	272	243		280	215		13.2	9	47
R.S.	342	348		370	360		16.9	12	45
J.P.	175	183		174	173		16.6	6	45
A.M.	308	242		181	187		14.9	14	41
D.C.	287	265		234	229		18.6	5	34
J.S.	215	200	184	163	120	134	13.8	6	45
A.E.	527	507		414	430		14.3	13	45

Table XV Planimetry readings, 3 min. Coomassie blue levels, tail height and packed cell volume. Subjects after myocardial infarction with evidence of pulmonary oedema.

	Supine			45°		
J.R.	231	216	.94	200	200	1.00
A.S.	134	133	.99	133	136	1.02
E.B.	195	191	.98	200	190	.95
J.J.	239	251	1.05	205	202	.99
E.K.	267	228	.85	210	237	1.12
J.B.	198	163	.82	162	187	1.15
I.C.	312	277	.89	289	285	.99
J.P.	197	172	.87	185	151	.82
J.D.	200	168	.84	198	190	.96
A.W.	266	258	.97	218	222	1.02
		mean	.92		mean	1.00
		S.D. =	.08		S.D. =	.09

Table XVI Successive estimations of cardiac output. Planimetry readings expressed as proportion of the initial determination. Subjects without evidence of cardiovascular or respiratory disease.

	Supine			45°		
C.K.	188	193	1.03	201	175	.87
G.A.	127	127	1.00	122	124	1.02
G.B.	206	242	1.17	210	223	1.06
A.C.	320	324	1.01	304	283	.93
A.V.	148	152	1.03	164	154	.94
A.F.	403	395	.98	320	288	.90
A.T.	266	236	.89	250	234	.94
J.G.	243	244	1.00	254	248	.98
A. McG.	395	374	.95	454	430	.95
W.S.	327	299	.91	341	323	.95
G.R.	297	264	.89	287	310	1.08
		mean	.99		mean	.97
		S.D.	.08		S.D.	.07

Table XVII Successive estimations of cardiac output. Planimetry readings expressed as proportion of the initial determination. Subjects after myocardial infarction without pulmonary oedema.

	Supine			45°		
J.R.	314	361	1.15	208	227	1.09
A.F.	385	344	.89	334	313	.94
R. McI.	190	217	1.14	166	180	1.08
	190	189	1.00	166	171	1.03
W.E.	272	243	.89	280	215	.77
R.S.	342	348	1.02	370	360	.97
J.P.	175	183	1.05	174	173	1.00
A.M.	308	242	.79	181	187	1.03
D.C.	287	265	.92	234	229	.98
J.S.	215	200	.97	163	120	.74
	215	184	.86	163	134	.82
A.E.	527	507	.96	414	430	1.04
		mean	.97		mean	.96
		S.D.	.11		S.D.	.12

Table XVIII Successive estimations of cardiac output. Planimetry readings expressed as proportion of the initial determination. Subjects after a myocardial infarction, with pulmonary oedema.

			Supine Observations			Observations at 45°		
Subject	Age	Sex	C.O. Litres/min.	Pulse /min.	Stroke Volume ml.	C.O. Litres/min.	Pulse /min.	Stroke Volume ml.
J.R.	21	M	6.5	60	108	6.3	60	105
A.S.	68	F	6.8	62	110	6.8	62	110
E.B.	22	F	4.5	88	51	4.5	88	51
J.J.	47	M	4.2	68	62	5.1	66	77
E.K.	26	M	4.9	66	74	5.5	66	83
J.B.	36	M	10.2	86	119	10.5	84	125
I.C.	25	M	5.0	64	78	5.2	66	79
J.P.	20	M	10.2	96	106	11.2	96	117
J.D.	39	M	10.3	64	160	9.8	66	148
A.W.	50	M	5.7	60	95	6.8	62	110
Mean	35.4		6.8	71.4	96.3	7.2	71.6	100.7
S.D. $\pm$	16.2		2.4	13.3	31.7	2.4	12.7	27.3

Table XIX **Results of Observations in ten subjects without evidence of cardiovascular disease.**

			Supine Observations			Observations at 45°		
Subject	Age	Sex	C.O. Litres/min.	Pulse /min.	Stroke Volume ml.	C.O. Litres/min.	Pulse /min.	Stroke Volume ml.
C.K.	38	M	5.8	78	74	5.7	78	73
G.A.	63	M	4.6	80	56	4.6	76	60
G.B.	56	M	3.7	80	46	3.7	82	45
A.C.	63	M	8.4	88	95	8.6	90	96
A.V.	63	M	7.9	66	120	7.5	66	114
A.F.	65	M	7.0	68	103	5.4	72	75
A.T.	56	M	5.7	75	76	5.9	84	70
J.G.	42	M	5.6	66	85	5.4	62	87
A. McG.	58	M	5.6	66	85	5.4	62	87
W.S.	62	M	7.6	66	115	7.2	72	100
G.R.	44	M	5.0	60	83	4.7	60	78
Mean	55.4		6.1	72.1	85.3	5.8	73.1	80.5
S.D. $\pm$	9.6		1.5	8.6	22.8	1.3	9.9	19.3

Table XX Results of observations in eleven subjects, without evidence of pulmonary oedema, who had sustained a myocardial infarction within the previous three weeks.

			Supine Observations			Observations at 45°		
Subject	Age	Sex	C.O. Litres/min.	Pulse /min.	Stroke Volume ml.	C.O. Litres/min.	Pulse /min.	Stroke Volume ml.
J.R.	65	M	4.4	76	58	6.5	76	86
A.F.	65	M	5.0	72	69	5.7	78	73
R. McI.	50	M	6.4	76	84	7.5	76	99
W.E.	44	M	6.2	72	86	6.6	74	89
R.S.	56	M	4.8	70	68	4.5	70	64
J.P.	47	M	5.5	88	63	5.7	90	63
A.M.	56	M	5.1	68	75	7.6	66	115
D.C.	67	M	2.2	78	28	2.7	82	33
A.E.	48	M	4.0	84	48	4.9	88	56
J.S.	57	M	5.9	92	64	8.5	96	89
Mean	55.5		5.0	77.6	64.3	6.0	79.6	77
S.D. $\pm$	8.1		1.2	7.9	17.1	1.2	9.4	23.7

Table XXI **Results of observations in ten patients with pulmonary oedema after myocardial infarction.**

		Supine			Elevated at 45°		
		Cardiac Output	Pulse Rate	Stroke Volume	Cardiac Output	Pulse Rate	Stroke Volume
Normal Controls (10 subjects)	Mean	6.8	71.4	96.3	7.2	71.6	100.7
	S.D.	2.4	13.3	31.7	2.4	12.7	27.3
Infarct Patients (no pulmonary oedema) (11 subjects)	Mean	6.1	72.1	85.3	5.8	73.1	80.5
	S.D.	1.5	8.6	22.8	1.3	9.9	19.3
Infarct Patients (with pulmonary oedema) (10 subjects)	Mean	5.0	77.6	64.3	6.0	79.6	77.0
	S.D.	1.2	7.9	17.1	1.2	9.4	23.7

Table XXII Mean results and standard deviations of cardiac output, pulse rate, stroke volume for three groups of subjects.

Significant differences are found between mean cardiac output of infarct patients with pulmonary oedema in supine and elevated position ($t = 3.7$, $p = <.01$) and in mean stroke volume in this group ($t = 2.85$, $p = <.05$). (paired t test, Hill 1967.)

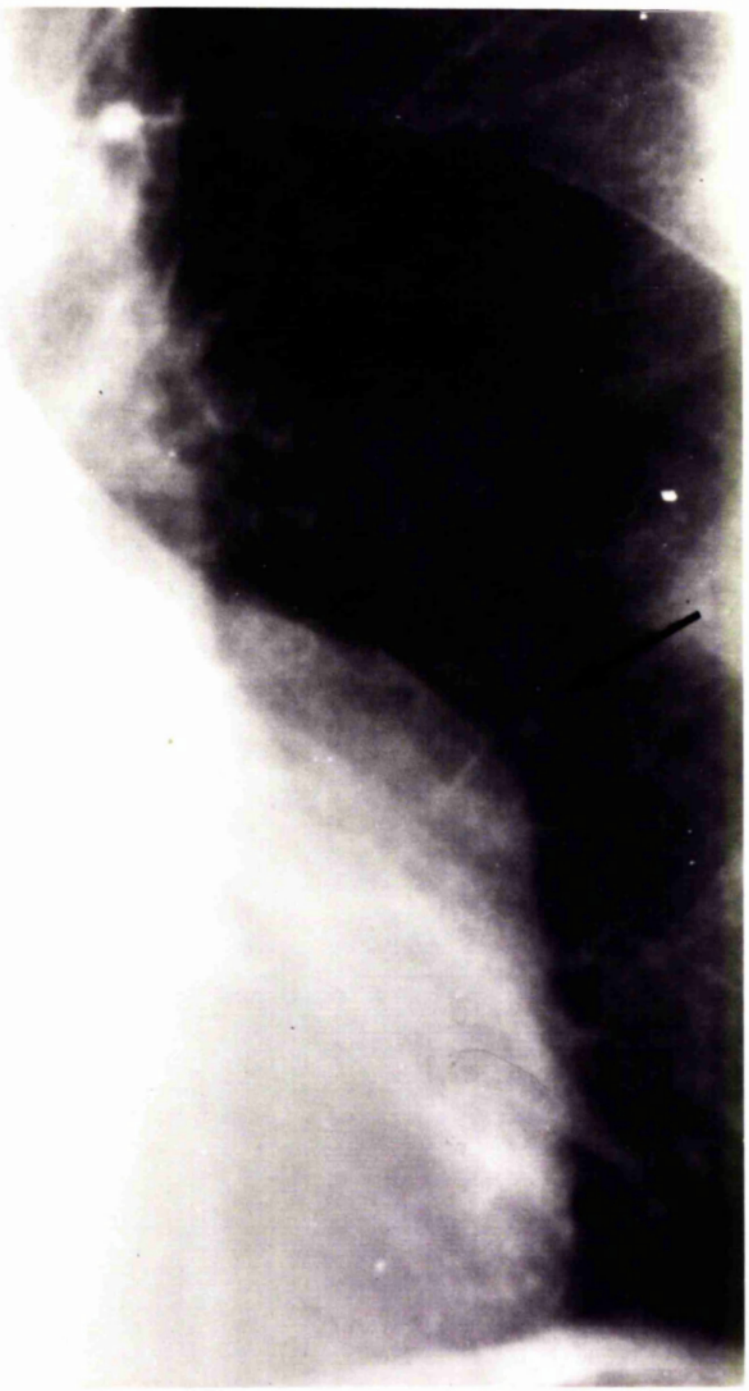


Plate I: W. O'H. - 11.5.67.

Medium sized bulge on anterior surface of heart after anterior infarction. R.A.O. projection.

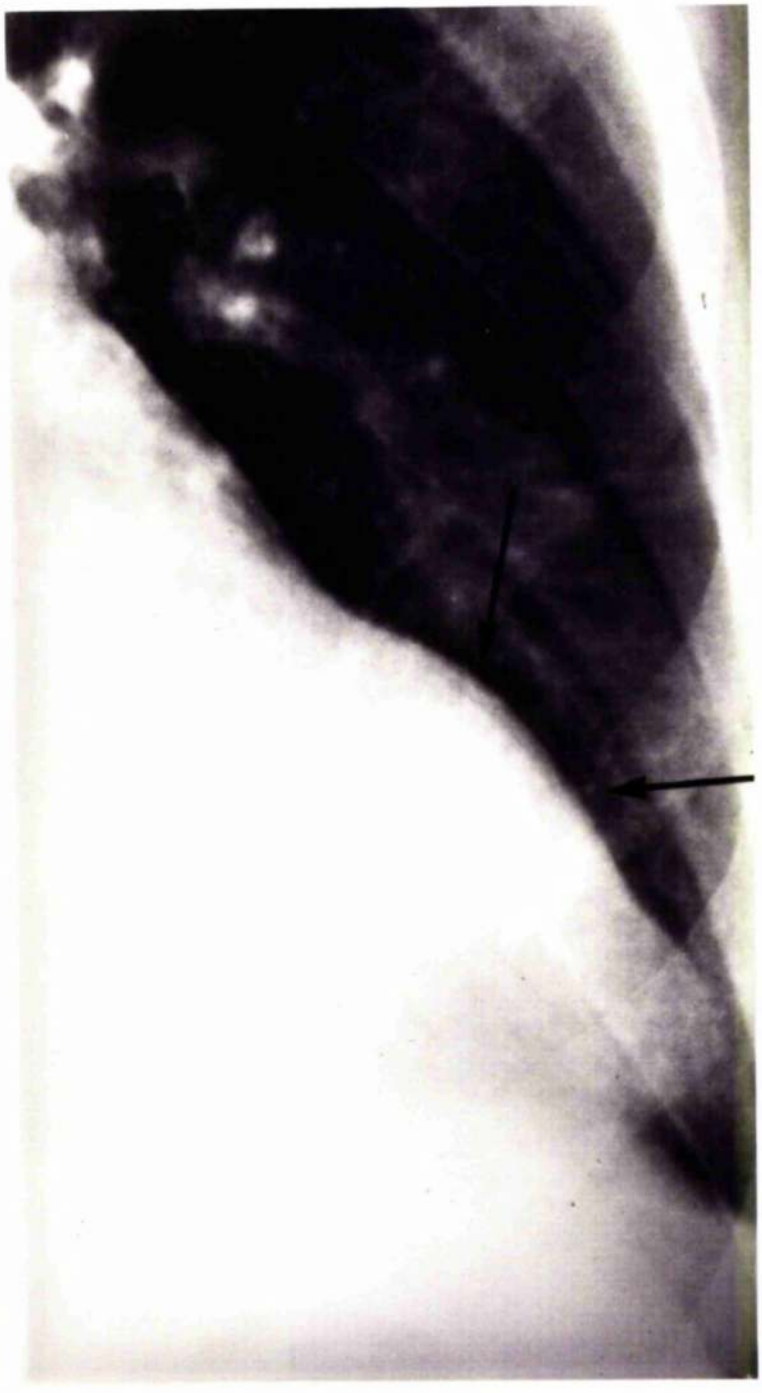


Plate 2: D. McD. - 20.7.66.

Medium sized bulge seen on anterior surface of heart after anterior infarction. R.A.O. projection.

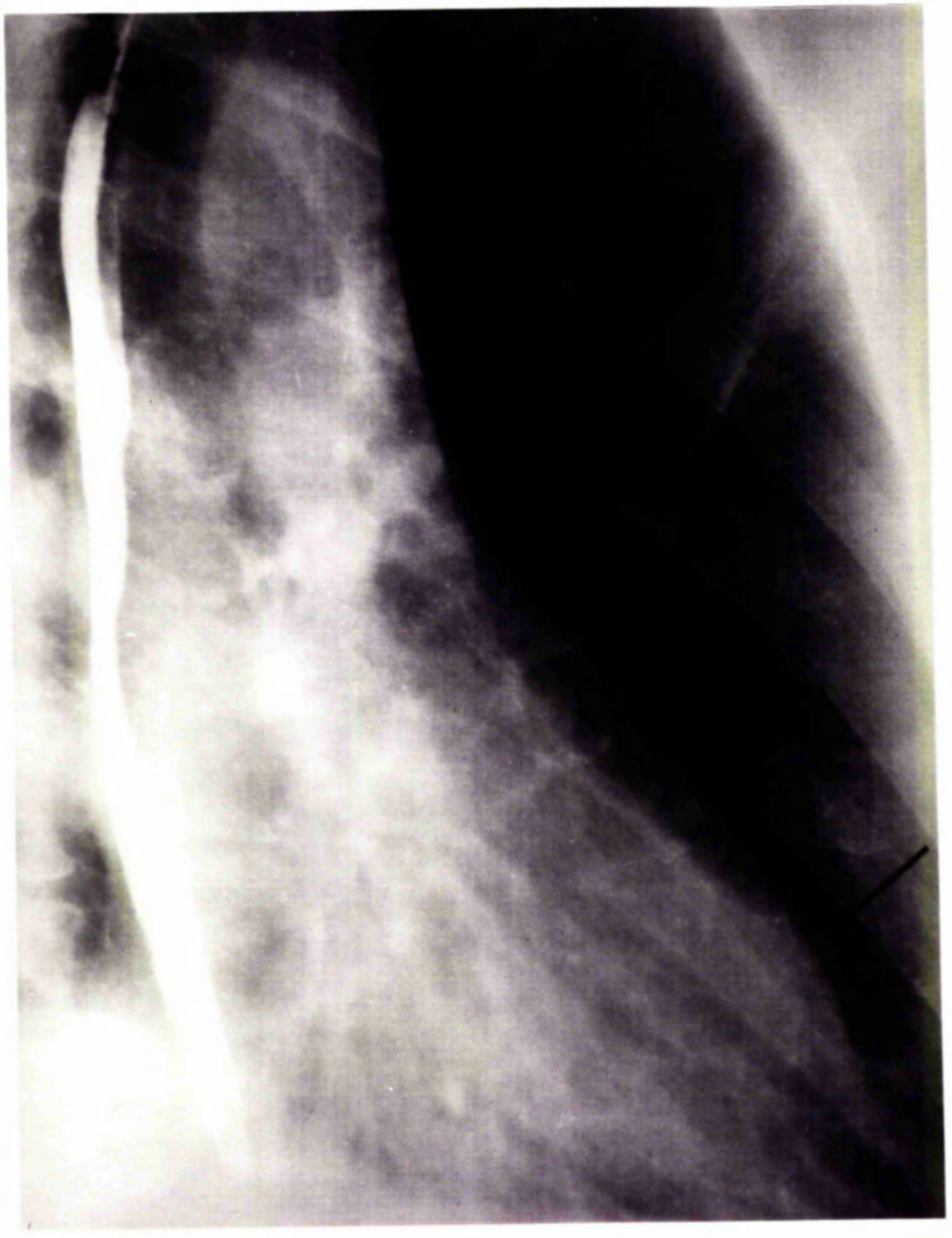


Plate 3: T.M. - 11.2.66.

Small bulge on anterior surface of heart following myocardial infarction. R.A.O. projection.

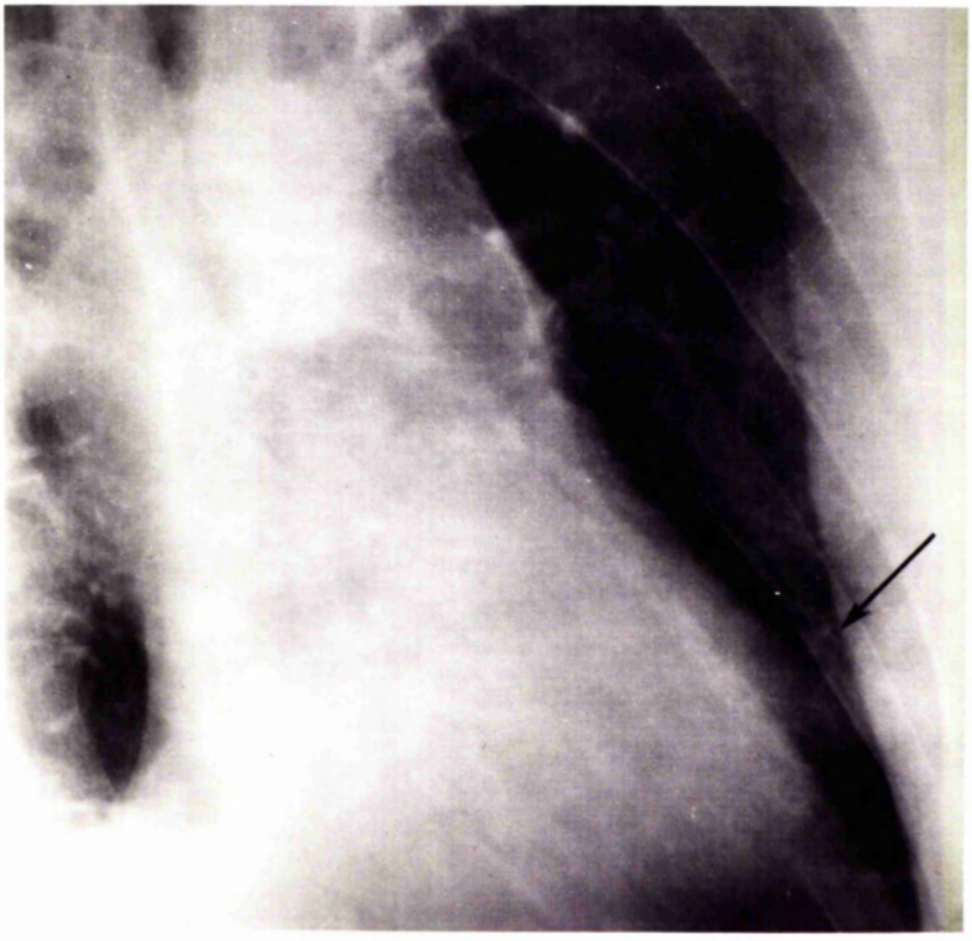


Plate 4: A.S. - 16.10.66.

Anterior pleuro-pericardial adhesion after an anterior myocardial infarction. R.A.O. projection.

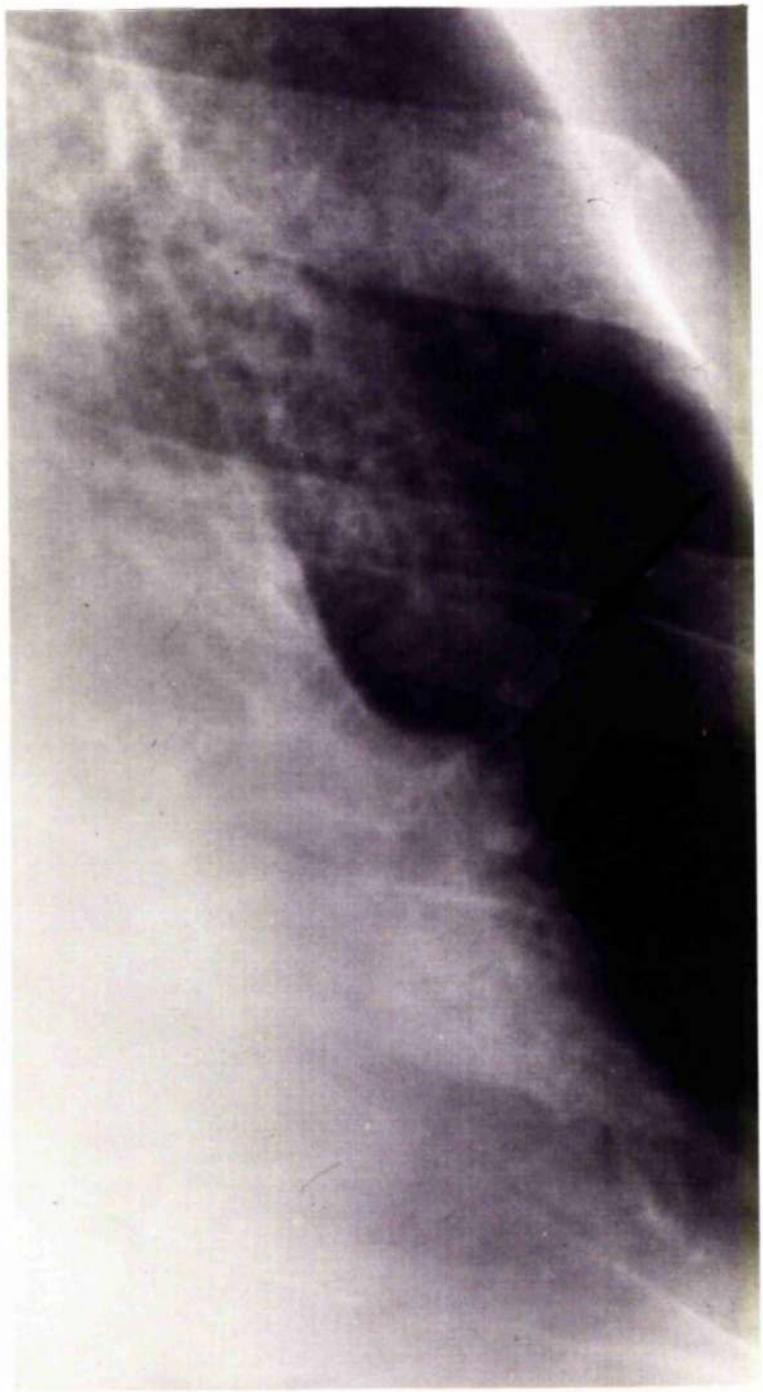
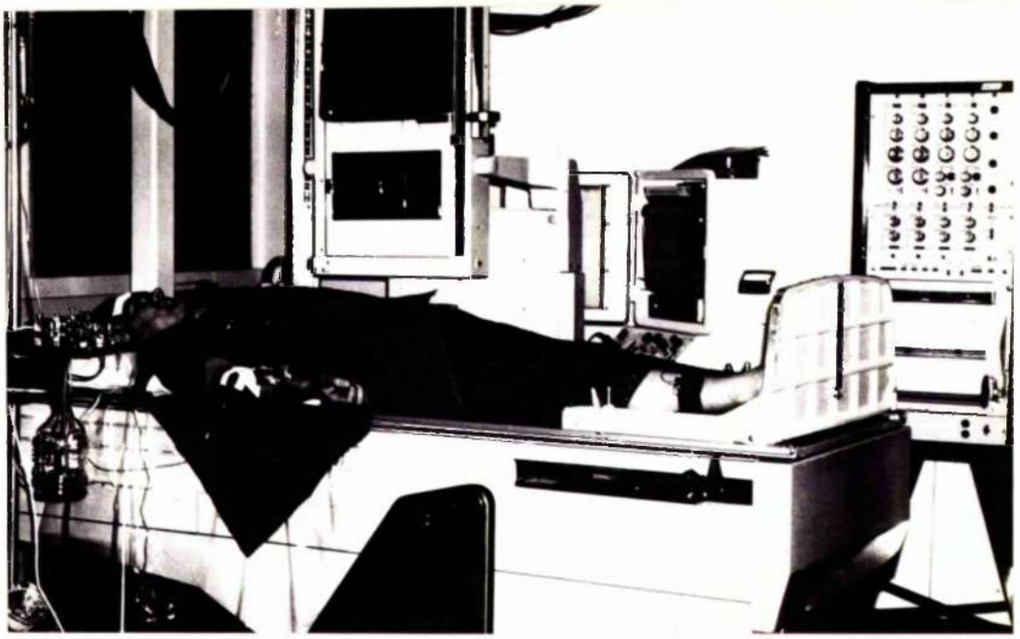
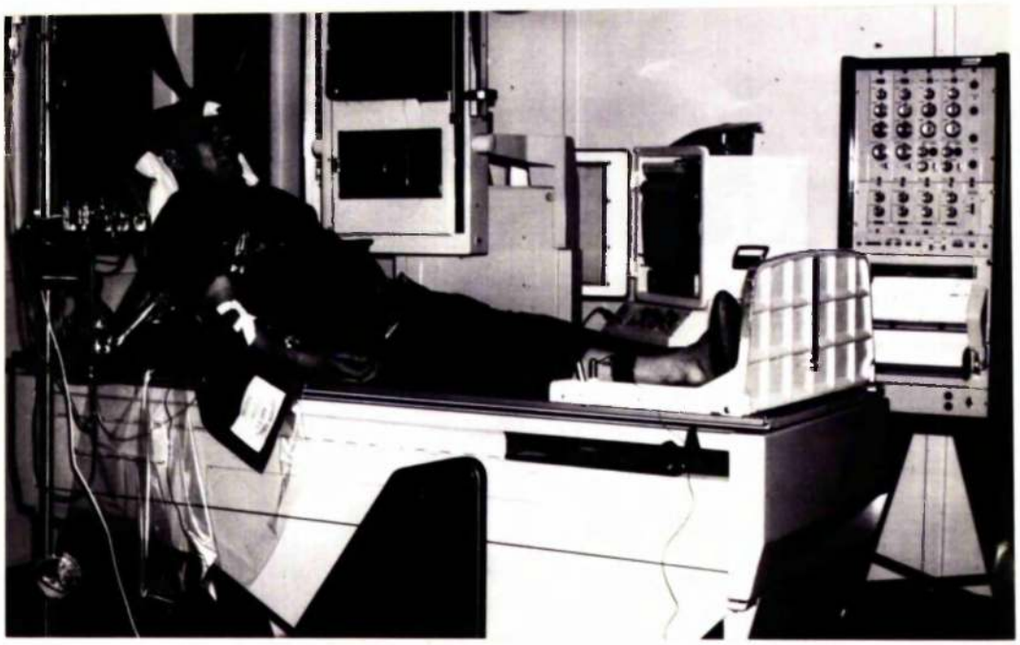


Plate 5: W.T. - 27.4.66.

Anterior pleuro-pericardial adhesion following anterior infarction. R.A.O. projection.



a)



b)

Figure 1: **Cardiac output studies:**

a) above - patient supine

b) below - patient in 45° head elevated position with legs horizontal.

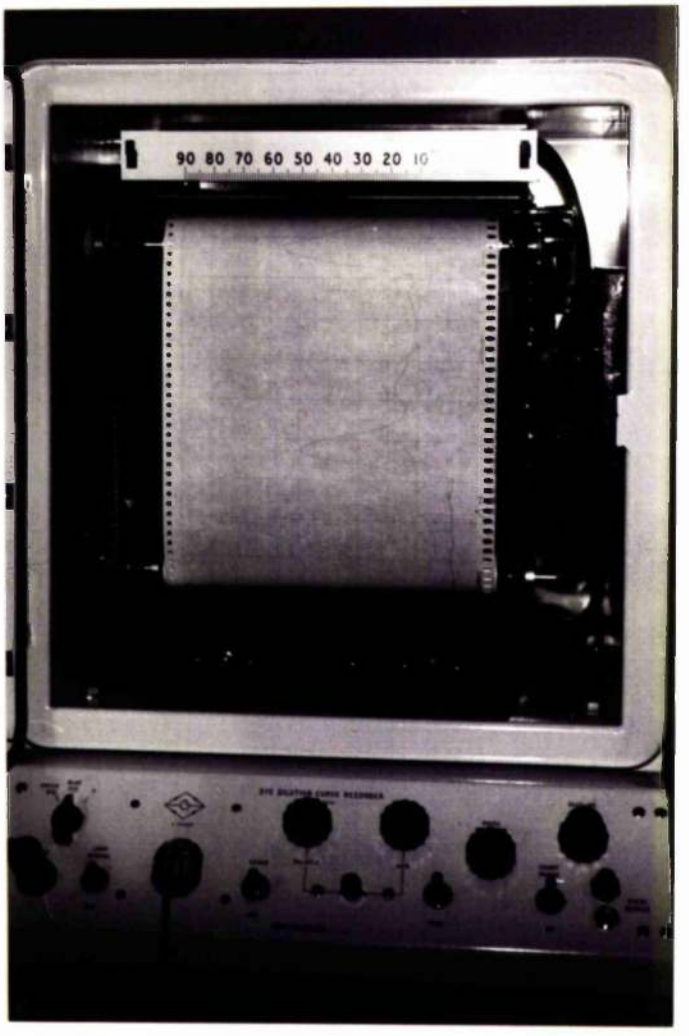


Figure 2: Cambridge Mk.II dye recorder.

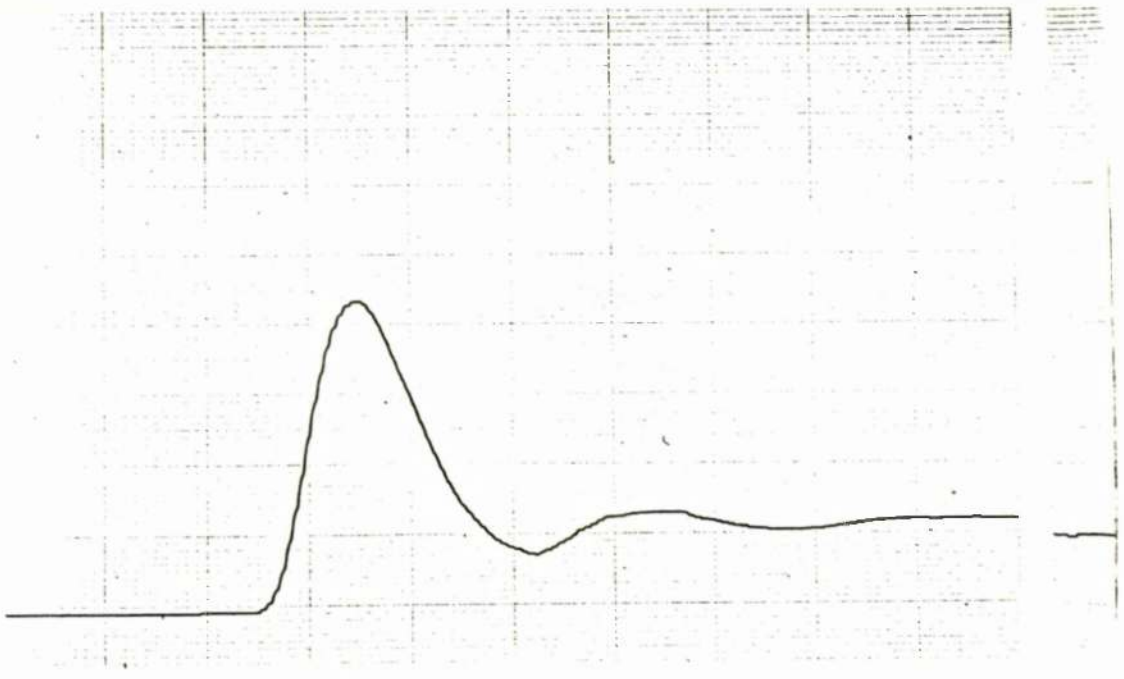


Figure 3: Dye curve drawn on Cambridge Mk. II dye recorder using a photo-electric earpiece after injection of 40mg. Coomassie Blue. The tail height is shown on the extreme right.

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