Extent of possible rehabilitation of service personnel with ischaemic heart disease

Inder Singh, P. K. Khanna, M. C. Srivastava, and R. S. Hoon
From the Directorate General, Armed Forces Medical Services, New Delhi, India

Sixty unselected survivors from ischaemic heart disease, all male service personnel, were treated, within 1 to 6 months of recovery from the acute episode, by promotion of physical activity, correction of coronary risk factors, and routine use of nicoumalone and pencyamine lactate. The results showed that this approach was conducive to more rewarding rehabilitation than was obtained in 89 patients who, in the past, were on conventional treatment based on advice regarding weight, diet, and physical and mental activity. Thus within 1 to 30 months of treatment by this approach, out of 60 patients, 12 were fit for medical category A active service duties in operational areas in any part of the world including altitudes below 10,000 feet, and 17 were fit for medical category C sedentary duties in non-operational areas in India only. One patient, who initially recovered from congestive heart failure, died while in a state of temporary unfitness for service. No patient was released from service on account of ischaemic heart disease. Against this, by the conventional approach, out of 89 patients, 81 were fit for medical category C sedentary duties in non-operational areas in India only, 4 were released from service, and 4 died.

‘Years ago a patient with a heart attack remained in bed for weeks or months, resigned from his job, and lived the remainder of his restricted life with fear and unhappiness (Hurst and Logue, 1966).’ Dr. Paul White is credited with reversing this approach. Goldwater established the first Work Evaluation Unit in 194I to assess the employment potential of cardiac patients. Since then some 50 Work Evaluation Units in the United States of America have shown that the great majority of civilian cardiac patients are capable of returning to productive work.

Rehabilitation of ischaemic heart disease patients in the armed forces, where standards of required physical fitness are tough and exacting, had not been considered free of apprehension regarding safety and doubts about actual performance. However, in India, we have now been able to reverse this tendency. To accommodate personnel who are disabled by illness, the standards of physical fitness in the armed forces in India are defined mainly in three categories, A, B, and C. Personnel in category A are fit for active service duties in operational areas in any part of the world and in any terrain, including high altitudes between 10,000 feet and 18,000 feet. Personnel in category B are fit for all service duties in non-operational communication zones in any part of the world including altitudes below 10,000 feet. Personnel in category C are fit for sedentary service duties in non-operational areas in India only. Those personnel who were temporarily disabled by illness and require sick leave are placed in category D. Those personnel who are not likely to recover sufficiently to return to service duties are placed in category E and are released as unfit for further service, though most of them are still fit for sedentary civilian duties. In the past, the highest category, and that too rarely obtained by any patient after the acute attack, who had been managed by the conventional methods of treatment, was B. With our current methods of management it has been possible to rehabilitate so far 20 per cent of these patients, even in category A. The purpose of this paper is to report the methods used and the results obtained in 60 unselected patients with confirmed ischaemic heart disease who were survivors after admission to hospital during 1966–1968.
Pilot studies
Between 1960 and 1966, pilot studies, which now form the basis of our treatment, were carried out on the feasibility of correction of coronary risk factors, the incidence of intravascular thrombosis, the quantum of physical activity within safe limits, and the assessment of recovery. Evidence had already accumulated and was being continuously substantiated by various workers on the role of smoking, physical inactivity, excessive calorie intake, obesity, hypertension, impaired carbohydrate tolerance, hyperlipidaemia, and hyperuricaemia as coronary risk factors. While proof of efficacy of modification of these factors in the treatment of the disease was lacking, modifications seemed reasonable and desirable. Pilot studies showed that absolute correction of risk factors, short of losing the patient's co-operation, was possible in most cases. The pace for co-operation could be set psychologically by the successful promotion of physical activity under preventive anti-anginal cover, mainly with prenylamine lactate and, sometimes, if this was not adequate, with a combination of prenylamine lactate and propranolol.

Necropsy studies of 34 patients with ischaemic heart disease brought in dead or who died within 6 hours of an acute attack showed that the block in the coronary arteries was a recent thrombus with varying degree, extent and site of atherosclerosis in 29 patients (85%) and atheroma alone in 5 patients (15%). As intravascular thrombosis accounted for 85% of the deaths, we attempted, as far as possible, to overcome this tendency with the routine use of anticoagulants supplemented by the beneficial effect of the correction of coronary risk factors on blood coagulation.

Tests were evolved for assessing the recovery of the disabled patients in relation to their future rehabilitation categories. The practical utility of these tests was established by field trials.

Subjects and methods
The patients were 60 Indians, all male service personnel; 29 were between 41 and 50, and 31 were between 51 and 58 years old. They were all unselected survivors from confirmed ischaemic heart disease who had been initially treated in hospital. Of the 60, 14 were moderate smokers and 46 were non-smokers; 13 were 3–10 kg overweight and 47 were of normal weight. In 41 the diastolic blood pressure was 70–80 mm. Hg, in 11 it was 81–90 mm. Hg, and in 8 it was 91–115 mm. Hg. The duration of overt ischaemic heart disease was 1 to 6 months.

An estimate of the effort tolerance, electrocardiographic, and biochemical states of each patient was made initially and subsequently once a month. The tests assessing the effort tolerance and the associated electrocardiographic changes were of a progressive series. The extent to which they could be undertaken depended entirely on the performance of the patients initially and subsequently during treatment. An existing electrocardiographic abnormality needed some care but was not considered to be a bar to initiate the effort tolerance tests.

Effort tolerance The effort tolerance was based on the following. (a) The functional capacity of the patient in terms of Classes I, II, III, and IV as defined by the New York Heart Association (1964). (b) The capacity of the patient to undertake double Master's two-step exercise test. (c) The capacity of the patient to undertake a three- and -half mile walk in one hour. (d) The capacity of the patient to undertake a hill climbing test involving a climb of 1000 feet over a gradient of 1 in 2 within a specific time at an altitude of 15,000 feet. The performance was graded as excellent, good, satisfactory, poor, and unsatisfactory (Table 1). Only excellent, good, and satisfactory grades were acceptable. The equivalent indoor test was a patient's capacity to climb up and down, 1000 feet each way, a flight of 40 steps over a gradient of 1 in 1.5 within a specific time at sea-level. (e) The capacity of the patient to withstand 45 minutes' exposure and then undertake a double Master's two-step exercise test in a decompression chamber at 15,000 feet.

The pretreatment functional capacity was Class I in 16, Class II in 20, Class III in 22, and Class IV in 2 patients.

Electrocardiograms Electrocardiograms were taken at rest, and immediately, 5, 10, and 15 minutes after double Master's two-step exercise test, the three- and -half mile walk, the hill climbing

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**Table 1: Equivalent grades of physical performance at sea-level and at 15,000 feet (hill climbing test)**

<table>
<thead>
<tr>
<th>Test</th>
<th>Grading</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Excellent</td>
</tr>
<tr>
<td><strong>1000 feet climb and descent, gradient 1 in 1.5, at sea-level</strong></td>
<td>Up to 25 minutes</td>
</tr>
<tr>
<td><strong>1000 feet climb, gradient 1 in 2, at 15,000 feet</strong></td>
<td>Up to 35 minutes</td>
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</table>
test, and at rest and after double Master's two-step exercise test in the decompression chamber after 45 minutes exposure at 15,000 feet.

The electrocardiographic changes were graded as those of (1) myocardial infarction with significant Q pattern and ST and T changes, (2) myocardial injury with ST and T changes, and (3) myocardial ischaemia with T, T and ST changes at rest or after double Master's two-step exercise test. Patients with doubtful electrocardiographic changes were not included in this observation. Electrocardiographic recovery was assessed on the basis of criteria as defined under the heading rehabilitation categories.

The pretreatment electrocardiographic changes were those of infarction in 21, injury in 3, ischaemia at rest in 25, and ischaemia on exercise in 11 patients.

**Biochemical parameters.** The biochemical parameters included tests for impaired carbohydrate tolerance, and estimation of blood cholesterol and blood uric acid levels. The serial tests for impaired carbohydrate tolerance were the preprandial and postprandial blood sugar estimations, the 100 g. glucose tolerance test, and if the results of preprandial and postprandial blood sugar estimations and the 100 g. glucose tolerance test were negative, the prednisolone-glucose tolerance test. The norms for these parameters were selected on the basis of those levels at or below which we encounter the lowest incidence of the disease and were as follows. Preprandial blood sugar less than 100 mg. and postprandial less than 140 mg./100 ml.; glucose tolerance test with fasting blood sugar less than 100 mg., peak less than 160 mg., and at 120 minutes less than 120 mg./100 ml.; prednisolone-glucose tolerance test (with prednisolone 10 mg. given 8 hours and 2 hours before the glucose tolerance test) with fasting blood sugar less than 140 mg., peak less than 200 mg., and at 120 minutes less than 140 mg./100 ml. of blood; blood cholesterol less than 180 mg./100 ml. of blood; blood uric acid less than 2.5 mg./100 ml. blood.

The pretreatment preprandial and postprandial blood sugar was abnormal in 28, the glucose tolerance was abnormal in 15, and the prednisolone-glucose tolerance was abnormal in 10 patients—these patients were considered hyperglycaemic; in the remaining 7 patients the prednisolone-glucose tolerance was normal. The pretreatment blood cholesterol level was normal in 8 and abnormal in the remaining 52 patients—these patients were considered hypercholesterolaemic. The pretreatment blood uric acid level was normal in 5 and abnormal in the remaining 55 patients—these patients were considered hyperuricaemic. Forty-eight patients had hypercholesterolaemia plus hyperglycaemia plus hyperuricaemia, 1 patient had hypercholesterolaemia plus hyperglycaemia, 2 patients had hypercholesterolaemia plus hyperuricaemia, 1 patient had hyperglycaemia plus hyperuricaemia, and 1 patient had hypercholesterolaemia, 3 had hyperglycaemia, and 4 had hyperuricaemia alone.

**Treatment** The essentials of treatment were (1) to promote physical activity and achieve maximum physical efficiency, (2) to correct obesity, and to return to normal blood pressure, carbohydrate tolerance, blood cholesterol, and blood uric acid, and (3) to provide continuous anticoagulant cover.

To prevent anginal pain during physical exertion, under anti-anginal cover with prenylamine lactate 180 mg. daily, and if this was not fast enough or adequate enough, with a combination of prenylamine lactate 180 mg. and propranolol 30 mg. daily, all patients were encouraged initially to walk increasing distances, then to take part in recreational games and hobbies involving physical activity, and finally to undertake more strenuous physical activity to enable them to achieve ultimately the standard of physical fitness required for returning them to service duties in rehabilitation categories as defined subsequently.

The norms for weight were —2 to —5 kg. of the minimum as defined in the data for ideal weights for men and women, aged 25 and over, by the Metropolitan Life Insurance Company, Statistical Bureau (1958). Weight was reduced as fast as possible with diets consisting mainly of milk or animal proteins 100 to 125 g., boiled vegetables, salads, and fruits with or without a small helping of carbohydrates. After weight had returned to normal, the individual calorie needs were met with a diet containing, approximately, protein 100—125 g., fat 50—75 g., and carbohydrates to make the rest. Fat was mostly provided as safflower oil or 50—50 mixture of groundnut and safflower oil. Simple sugars were restricted. Whisky and gin were allowed in moderation, but beers and sweet wines, on account of their adverse effects on blood coagulation, were not advised.

The norm for diastolic blood pressure was 90 mm. Hg or less. If raised diastolic blood pressure persisted after weight reduction, judicious use was made of diuretics, diuretics plus guanethidine, or diuretics plus methyldopa.

Abnormalities in carbohydrate tolerance still persisting after weight reduction were corrected to as near normal as possible with time-disintegration phenformin (DBI-TD) 50—150 mg. daily, and, if this was not adequate, with time-disintegration phenformin 50—150 mg. plus tolbutamide 0.5 to 1.5 g. daily. The purpose was to achieve under treatment a normal prednisolone-glucose tolerance, or as the next best a normal glucose tolerance, or at the minimum normal preprandial and postprandial blood sugar levels. Raised blood cholesterol persisting after weight reduction and the prescribed diet was treated with nicotinic acid 1500—3000 mg. daily and, if this was not adequate, with nicotinic acid 1500—3000 mg. plus clofibrate 750—1500 mg. daily. Raised blood uric acid was treated with probenecid 0.5 to 1.5 g. daily.

Nicoumalone 1—4 mg. was used routinely as an anticoagulant on an indefinite long-term basis to keep the prothrombin time and control time ratio between 1·6:1 and 2:1 and the Lee White coagulation time within 20—30 minutes.
Smokers were advised to give up smoking.

Rehabilitation categories  The results of effort tolerance, electrocardiographic recovery, and the corrected metabolic abnormalities were computed as follows for defining a particular rehabilitation category.

Category C  Patient required to be asymptomatic during physical activity; electrocardiogram at rest to be normal or stabilized; weight and metabolism to be normal.

Category B  Patient required to be asymptomatic during physical activity; electrocardiogram at rest to be normal or stabilized; no deterioration after a three- and-half mile walk in one hour and after double Master's two-step exercise test; weight and metabolism to be normal.

Category A  Patient required to be asymptomatic during physical activity; electrocardiogram at rest to be normal or stabilized; no deterioration after a three- and-half mile walk in one hour, after double Master's two-step exercise test, after hill climbing test, and after exposure and exercise in the decompression chamber at 15,000 feet. Weight and metabolism to be normal.

Results  Out of 60 patients treated, 12 returned to category A, 30 to category B, 17 to category C, and one died while still in category D after the acute attack. Against these results, of 89 patients treated in the past by us mainly with conventional advice in connexion with weight, diet, and physical and mental activity as given in standard textbooks, none returned to category A or category B, 81 returned to category C, 4 were released from service (category E), and 4 died of a subsequent attack.

Factors that seem to have favoured the return of patients to category A were age 41-50 years, pretreatment effort tolerance Class I, pretreatment myocardial ischaemia associated with only post-exercise electrocardiographic changes, and duration of treatment over 12 months.

A persisting electrocardiographic abnormality, however, so long as it did not show any deterioration after double Master's two-step exercise test, after the three- and-half mile walk, after the hill climbing test, and at rest and after exercise in the decompression chamber at 15,000 feet, was of no practical significance as far as the patient's capacity for physical performance in category A was concerned. Thus 4 out of 11 patients with pretreatment post-exercise ischaemic changes, 4 out of 25 patients with pretreatment ischaemic changes at rest, and 4 out of 21 patients with pretreatment changes of infarction, qualified for category A (Table 2).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Total no. of cases</th>
<th>Rehabilitation category (no. of cases)</th>
<th>Died</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td><strong>Age (yr.)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>41-50</td>
<td>29</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>51-58</td>
<td>31</td>
<td>3</td>
<td>18</td>
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<tr>
<td><strong>Effort tolerance</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>16</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>Class II</td>
<td>20</td>
<td>4</td>
<td>11</td>
</tr>
<tr>
<td>Class III</td>
<td>22</td>
<td>—</td>
<td>11</td>
</tr>
<tr>
<td>Class IV</td>
<td>2</td>
<td>—</td>
<td>1</td>
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<tr>
<td><strong>Electrocardiogram</strong></td>
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<td></td>
<td></td>
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<tr>
<td>Q, ST, T at rest</td>
<td>21</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>ST, T at rest</td>
<td>3</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>T, ST at rest</td>
<td>25</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>T, ST on exercise</td>
<td>11</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td><strong>Duration of treatment</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>1-6 mth.</td>
<td>27</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>7-12 mth.</td>
<td>12</td>
<td>2</td>
<td>5</td>
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<tr>
<td>13-18 mth.</td>
<td>8</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>19-24 mth.</td>
<td>4</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>25-30 mth.</td>
<td>9</td>
<td>3</td>
<td>6</td>
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Discussion  Blood pressure and serum lipid levels have been considered to constitute together the key factors in assessing the magnitude of the risk of developing ischaemic heart disease. At any level of either factor the risk is greater with the increasing level of the other (Dawber and Kannel, 1968). However, in our series diastolic hypertension did not occupy a significant place as a predisposing factor. Thus out of 60 patients, only 8 had a diastolic blood pressure between 91 and 115 mm. Hg, 11 were between 81 and 90 mm. Hg, and the remaining 41 were between 70 and 80 mm. Hg. Moreover, 48 out of 60 patients had not only hypercholesterolaemia but also associated hyperglycaemia and hyperuricaemia. Moderate smoking, if it affected the disease at all, was a factor only in 14 out of 60 patients. Only 13 out of 60 patients were 3-10 kg. overweight. Before the onset of the disease the subjects were fighting-fit soldiers. Therefore, physical inactivity as a predisposing cause was completely ruled out. However, in 17 out of 60 patients the acute episode had occurred during
the period of rest after severe physical stress.

The rehabilitation results are overwhelmingly in favour of the treatment we have adopted. The extent to which promotion of physical activity, the use of coronary dilators, correction of metabolic abnormalities, and the use of anticoagulants individually contributed to the achievement of these results is difficult to assess with any degree of accuracy. Our patients were motivated by a desire to return to duty in the highest possible rehabilitation category to brighten their future prospects. The fact that they were under the care of a team of medical officers led personally by the senior consultant in medicine may have had an added psychogenic effect on their co-operation and recovery. It is possible that the most important factor that contributed to recovery was the promotion of physical activity. The effect of physical activity in the development of collateral coronary circulation (Eckstein, 1957; Currens and White, 1961) and the consequent improvement in myocardial function and reduction in mortality rate (Kannel et al. quoted by Dawber and Thomas, 1968) seem to be well established. However, other workers have not promoted physical activity to the extent we have found it possible to do. The initial pace for promotion of physical activity can be set if during such activity the patient does not have anginal pain. For this purpose, a protective anti-anginal cover with prenylamine lactate or prenylamine lactate plus propranolol is useful. The more successful one is in this attempt, the better is the co-operation. Prenylamine lactate probably helps in other ways. Thus, though prenylamine causes only a short-lived increase in coronary flow at rest (Rudolph, Meixner, and Kunzig quoted by Grebecker and Palm, 1968), it seems probable that this is more sustained during exercise, especially in healthy subjects.

In the relative absence of diastolic hypertension, smoking, obesity, and physical inactivity as predisposing factors the combination of hypercholesterolaemia, hyperglycaemia, and hyperuricaemia seemed most important, being present in 48 out of 60 patients. The adverse effects of this combination on blood coagulation and the atherosclerotic process are closely interrelated. Thus impaired carbohydrate tolerance associated with raised plasma insulin level or increased insulin resistance predisposes to an increased fatty acid response to glucose load (Soloff and Schwartz, 1966). In diabetics there is a greater incidence of low fibrinolytic activity than there is in controls, with increased platelet stickiness (Bridges et al., 1965; Bray et al., 1967) and increased viscosity of the blood (Skovborg et al., 1966). Ingestion of fats inhibits fibrinolytic activity in atherosclerotic subjects (Gajewski, 1961) and enhances platelet stickiness. Blood platelet counts correlate with blood cholesterol levels (McDonald and Edgill, 1959). Long chain fatty acids from C10 onwards accelerate thrombus formation time (Connor, Hoak, and Warner, 1965). Therefore rapid lipid mobilization can result in thrombosis (Hoak, Poole, and Robinson, 1963). Blood uric acid probably acts as an intimal conditioning agent and thus furthers cholesterol deposition, especially when the serum lipids are unstable. It may also impair glucose tolerance. A return to normal of these metabolic factors was, therefore, considered desirable, as they probably help in the reversal of the atherosclerotic process, as in animal experiments, and minimize the adverse effects of these factors on blood coagulation. Unfortunately the dividing line between the normal and the abnormal is only arbitrary, and constitutes a hurdle in evolving a consensus on the extent to which these metabolic factors should be tackled for the purpose of treatment. We have selected the norms as those levels at or below which we encounter the lowest incidence of the disease. For our patients, by virtue of their occupation, the calorie and protein intake had to be high to be adequate for requirements of energy and maintenance of normal weight. To make the diet anticholesterologenic, we depended on a moderate fat content of 50-75 g. provided mostly as safflower oil in which the polyunsaturated fatty acid and the saturated fatty acid ratio is 10:1. When reduction of blood cholesterol level was not achieved with this diet, recourse was made to nicotinic acid and in addition, if necessary, to clofibrate. Nicotinic acid had the advantage of being cheap but was not complete in itself. It inhibits the mobilization of free fatty acids (Carlson, 1965) and lowers both free and ester cholesterol but does not affect beta-lipoprotein cholesterol. In contrast, clofibrate reduces platelet stickiness, serum cholesterol, low density lipoprotein, and SF fractions 0-12 and 12-400 (Carson et al., 1963). Phenformin was preferred to improve carbohydrate metabolism. In coronary heart disease phenformin reduces the insulin output, and decreases serum cholesterol, serum triglycerides, and serum phospholipids, while free fatty acids vary widely and glucose tolerance remains unchanged (Tzagournis, Seidensticker, and Hamwi, 1968).

We used nicoumalone as an anticoagulant arbitrarily, fully realizing that it has no effect on Factor VIII or on the anti-Willibrand factor which is related to platelet stickiness.
However, the clinical use of anticoagulants now seems to be further justified by the reported increase in the survival rate from the initial infarct and in coronary disease manifested by angina (Royston, 1968). Our results suggest that the therapeutic measures which involve reduction of platelet stickiness, increased fibrinolysis, and reduced coagulation have a better effect than anticoagulants alone, not only on survival but on recovery as well.

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References


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