AN EXPERIMENTAL STUDY
OF THE ROLE OF AN EXERCISE PROGRAMME
IN THE TREATMENT OF ALCOHOLISM

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The Role of Exercise in the treatment of Alcoholism

M.Sc Thesis

presented by

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I, Brett Adams, declare that the work on which this thesis is based is original (except where acknowledgements indicate otherwise) and that neither the whole work nor any part thereof has been, is being, or will be submitted for another degree in this or any other University.

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Part of this study was presented at the First South African International Sports Medicine Congress, Rand Afrikaans University in March 1985.

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Brett Adams

Cape Town

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ABSTRACT

The aim of the present study was to investigate the effect of participation in an exercise training programme on the outcome of treatment in a group of alcoholic patients admitted for three weeks of inpatient treatment. Furthermore, the psychological changes observed after three weeks and again after three months in the exercise group were compared to those in a control group. The study also hoped to determine the state of fitness in this group and whether or not their response to exercise and to training was normal. Other objectives were to determine the incidence of cardiomyopathy in the population group studied and to monitor changes in blood lipid concentrations during the course of the study.

The study was conducted at the William Slater Hospital for Alcohol Rehabilitation, where patients are admitted for a three-week inpatient treatment regime of conventional therapy. All subjects for the study were male volunteers less than 45 years of age (mean age 31,7 years) with an average alcohol consumption of approximately 300 g/day one month prior to admission.

A two group pre/post study design was used in which control data were gathered prior to the intervention of the exercise programme. All subjects were tested at admission, after three weeks and again after three months. The tests performed included anthropometric measurements, a maximal exercise test, blood lipid

measurements and psychometric testing.

The exercise training programme consisted of an initial warm-up period of stretching and walking, followed by twenty to thirty minutes of calisthenic exercises and cycling, followed by a cool-down period of stretching. Intensity was between sixty and eighty percent of maximal heart rate. The exercise sessions were held three times per week for the initial three week period. Thereafter the exercise programme was upgraded to suit each subject's individual needs.

The results of the study show a significant difference in abstinence rating after three months in the exercise group compared to controls (66% and 25% respectively, $p < 0,05$). After three months, the exercise group showed significant changes in all the physiological measures of fitness ($p < 0,05$). Peak workload and maximal oxygen consumption increased and blood lactate, heart rate and oxygen consumption at each workload decreased after training, indicating that the subjects had undergone a normal training response. The group as a whole showed a normal cardiorespiratory and metabolic response to exercise, although their initial state of fitness was poor.

Very significant differences between groups were also evident in the psychological parameters measured. Already after three weeks, significant improvements in Tension, Depression and Self-

confidence scores were found in the exercise group ($p < 0,02$) whereas no significant changes were observed in the control group. After three months, the exercise group showed a significant increase in the Vigour ($p < 0,02$), Self-confidence and Self-control ($p < 0,05$) scores. Again there were no significant changes observed in the control group.

No evidence for cardiomyopathy nor hypertension was found in any of the subjects and resting heart rates were normal. Blood lipid changes after three weeks of abstinence suggest that excessive alcohol consumption lowers blood total cholesterol concentration, triglyceride concentration, low density lipoprotein cholesterol concentration and elevates high density lipoprotein (HDL) cholesterol concentration, particularly the HDL2 subfraction. After three months of exercise training, similar changes in blood lipid concentrations were observed.

In conclusion, the exercise training programme greatly improved abstinence after three months and induced positive psychological changes during treatment as well as after three months. The study showed that alcoholic patients have a normal response to exercise and can undergo normal physiological adaptations to training. Should further research substantiate these findings, particularly after longterm follow-up, exercise could provide an effective, low-cost intervention accessible to large numbers of alcoholic sufferers.

ABBREVIATIONS

ACL	-	Adjective Check List
ADH	-	Alcohol Dehydrogenase and Aldehyde Dehydrogenase
ALT	-	Alanine Aminotransferase
AST	-	Aspartate Aminotransferase
ATP	-	Adenosinr Triphosphate
BP	-	Blood Pressure
bpm	-	beats per minute
C	-	Celsius
CaSO4	-	Calcium/Sulphate
Chol	-	Cholesterol
cm	-	centimeter
ECG	-	Electrocardiogram
g	-	gram
GGT	-	Gamma Glutamyltransferase
HDL-chol	-	High Density Lipoprotein Cholesterol
HR	-	Heart Rate
kg	-	kilogram
Lact	-	Lactate
LDL-chol	-	Low Density Lipoprotein Cholesterol
m	-	meter
min	-	minute
ml	-	millilitre
ml/kg/min	-	millilitre per kilogram per minute
mm	-	millimeter

mmHg	-	millimeters Mercury
mmol/l	-	millimole per litre
NAD	-	Nicotinamide Adenine Dinucleotide
NADH	-	Nicotinamide Adenine Dinucleotide Reduced
OESS	-	Overall Emotional Stability Score
PCA	-	Perchloric acid
POMS	-	Profile Of Mood States
Trig	-	Triglyceride
VCO2	-	Carbon dioxide consumption
VO2	-	Oxygen Consumption
VO2 max	-	Maximal Oxygen Consumption
%	-	percentage

CHAPTER I

RATIONALE AND SCOPE OF THE THESIS

INTRODUCTION

The abuse of alcohol is a major health problem and has far reaching medical and socio-economic implications. Alcohol is a known aetiological factor for certain respiratory, gastrointestinal and liver cancers, cirrhosis of the liver, gastritis, pancreatitis and cardiovascular disease (Marsden, 1977). Alcohol abuse is linked to premature deaths due especially to cirrhosis of the liver, which is the fifth leading cause of death in Western Society (Schimdt, 1977), but also to road accidents, crime and suicide. Unemployment and absenteeism due to alcohol abuse also have astronomical cost implications. However, the extent of damage resulting from the abuse of alcohol cannot be viewed in terms of monetary costs alone. Many involved in the treatment of alcoholism believe that the social damages caused by alcohol abuse, far outweigh the economic costs thereof (Paton et al., 1981).

Of all the problems associated with alcoholism, probably the most disconcerting and controversial is the poor success rate that treatment of this condition has (Smith, 1981b; Kendell, 1979). Only a small percentage of those with an alcohol problem actually seek help and of these, only a very select few are admitted for treatment. This plus the poor success rate of current treatment programmes casts considerable doubt on the efficacy of the treatment of alcoholism.

STATEMENT OF THE PROBLEM

The cost of alcoholism and the extent of damage caused as a direct result of the abuse of alcohol, are difficult to determine. However, the British Office of Home Economics estimates that in Btitain, 10 000 premature deaths per annum are alcohol-related (Smith, 1981a). The estimated costs of working hours lost as a result of these premature deaths are of the order of 1 000 million pounds per annum (Paton et al., 1981). The British Office of Home Economics also estimates that the cost of absenteeism in government departments, as a result of alcohol abuse, cost the state approximately 200 million pounds per annum (Smith, 1981a). Similarly, the United States Post Office estimates that the cost of absenteeism due to misuse of alcohol among their 700 000 employees is $ 37 million per annum (Von Weigand, 1972).

In Britain (1980), an estimated 130 000 convictions for drunken behaviour and 70 000 for drunken driving were made and more than 10% of the total psychiatric admissions (some 20 000 cases) were alcohol-related (Paton et al., 1981). In the United States, at least 14 000 deaths per annum are caused by cirrhosis of the liver and alcohol abuse is considered to be the contributing factor in more than half the road accident fatalities, 70% of drownings and 30% of deaths from suicide (Desmond, 1987).

These statistics, although not very scientific, do give an indication of the extent of damage caused by the abuse of

alcohol. Although very little information is known about the extent of the problem in this country, it could confidently be stated that the same alarming percentages hold true. As in the United States, between 57% and 63% of fatal road accidents in South Africa are believed to be alcohol-related (Pieterse, 1984). Furthermore, it is estimated that the medical, legal, administrative and personal costs involved for these accidents alone exceed R 1 600 million per annum (Pieterse, 1984).

Other major socio-economic implications of alcohol abuse are unemployment and crimes of violence such as assault, rape and murder. Morbid jealousy is common amongst excessive drinkers. One study found that 10 of 46 murderers were heavy drinkers and very often it is the spouse of the alcoholic who is murdered (Marsden, 1977).

The social damages of alcoholism however, are most crippling. Unlike cigarette smoking, the abuse of alcohol contributes more to morbidity than mortality (Smith, 1981a). The effects of alcoholism are felt by all family members. Social isolation and shame experienced by the spouse and children of the alcoholic are likely to cause further behavioural problems (Orford, 1977). Children of a parent with a drinking problem are prone to develop a similar problem early in their lives (O'Connor, 1976). A recent survey in the United States, shows that 25% of families report a problem with alcohol in the family (Desmond, 1987).

TREATMENT OF ALCOHOLISM

A controversial issue of alcoholism today is the poor success rate of the treatment regimes employed (Smith, 1981b; Kendell, 1979). Invariably, esoteric and expensive treatment regimes have proved no more effective than simple, low cost therapies. A three week inpatient treatment programme seems as effective as a three month programme (Willems, 1973) and outpatient treatment as effective as inpatient treatment (Edwards and Guthrie, 1967). More thorough and recent studies show similar equivocal findings and some even suggest that inpatient treatment is as ineffective as no treatment at all (Edwards et al., 1977). In the longitudinal study that has the longest follow-up, Vaillant et al. (1983) showed that self-help methods and "natural healing" are more effective in achieving abstinence in problem drinkers than professional treatment. A review by Emrick (1975) found similar outcomes of treatment in groups of alcoholics who received either no treatment or minimal treatment. However, some of the these findings have methodological weaknesses such as (a) different levels of alcohol dependency and psychosocial stability in subjects used for the various studies, (b) different measures used to determine outcome and (c) different follow-up periods.

With regard to the South African experiance, success rates of between 16 and 25 % have been reported (Gillis and Keet, 1969), which compares favourably with those reported elsewhere (Emrick, 1975).

These findings justify the statement by Vaillant et al. (1983) that, in order to understand the true aetiology of alcoholism and its treatment, more attention should be directed towards understanding the natural history of alcoholism. He also recommends that more effective, low-cost treatment regimes be developed which cater for the large numbers of those suffering from the effects of alcohol abuse.

The pessimism associated with the poor outcome of treatment is illustrated by the following quote (Kendell, 1979) : "The medical treatment of alcoholism is of limited efficacy, as is the treatment of most of its secondary consequences. Above all, there is no realistic prospect of any of the caring professions, individually or corporatively, being able to cope effectively with the disability and suffering caused by alcohol abuse in the forseeable future, even if the human and material resources available to them were to be greatly increased." He concludes that alcoholism should be viewed as a political problem rather than a medical one and suggests that until this approach is taken, the problem will never be dealt with effectively. The only effective way to curb alcoholism and its related ills, is to reduce by law the average alcohol consumption of the population.

Nevertheless, medical care and treatment of patients who have suffered extensive physical and psychological illness as a result of the abuse of alcohol is still necessary. But considering the above finding that intensive treatment may be no more effective than minimal treatment, more attention should be directed towards

developing effective low cost treatment regimes which could serve a larger portion of those suffering the ill effects of alcoholism.

THE POSSIBLE ROLE OF EXERCISE IN THE TREATMENT OF ALCOHOLISM

The alcohol-dependent person is characterized by a poor self-image, low self-esteem, lack of self-confidence, depression and anxiety (Conner, 1962; Vanderpool, 1969; Chafetz, 1979). The problem drinker often has a dependent personality and uses alcohol either to satisfy dependency needs or as a mechanism to cope with emotional problems. The subsequent abuse of alcohol causes shame, guilt, resentment and further depression which stimulates more drinking. Hence dependency on alcohol becomes a vicious circle and what may have begun as purely a psychological dependency soon becomes a physical addiction. Apart from the psychological disorders present in alcoholic patients, these patients are often malnourished , inactive and usually smoke heavily (Visocan, 1983). This state of ill-health leads to a poor body-image which contributes further to poor self-image and depression.

Regular exercise on the other hand, is known to improve psychological well-being and can provide a positive coping mechanism for stress (Folkins and Sime, 1981; Young, 1979; Michael, 1976). It also improves body-image, self-esteem and self-confidence (Collingwood, 1972; Young and Ismail, 1976; Maloney et al., 1986). A single bout of physical exercise has been associated with a sense of well-being often described as

tranquil and euphoric (De Vries, 1981). It also relieves depression and anxiety (Morgan and Horsman, 1976), so much so that some authors have considered it to be as effective as a mild tranquiliser (De Vries, 1981; Harber and Sutton, 1984). Exercise has also been associated with increased happiness with general personality changes evident after long-term training (Carter, 1977; Noakes, 1985).

Paradoxically, people who tend to exercise regularly might also have addictive personalities. Noakes (1985) descibes the "addictive dependent personality" as a possible psychological explanation for the so-called "addiction" to running. He also suggests that exercise might serve as a substitute dependency for addictive personality types as seen in alcohol-dependent persons. The major benefit of exercise as a substitute dependency is that it is supportive of health whereas alcohol destroys health. Other benefits of exercise include relaxation, stress management, physical health and psychological well-being (Noakes, 1985).

Lastly, exercise seems to satisfy the recommendations made by Vaillant et al. (1983) for potential interventions which might maximise the natural healing process of alcoholism, namely (a) finding a substitute dependency for alcohol, (b) discovering a fresh source of hope and self-esteem and (c) obtaining new social support structures.

HYPOTHESIS

The hypothesis of the present study is that regular exercise included in an inpatient treatment regime for alcohol-dependent persons, will enhance fitness and induce beneficial psychological adaptations thereby improving abstinence.

SCOPE FOR THE STUDY

Considering the above literature, it would seem most likely that a regular exercise programme might benefit the alcoholic patient. Quite surprisingly, a review of the literature reveals very few studies of this topic. A few studies investigated the effects of exercise on psychological well-being in alcoholic patients (Gary and Guthrie, 1972; Frankel and Murphy, 1974; Tsukue and Shohoji, 1981; Palmer et al., 1988) and their findings are equivocal. Only one study evaluated the effects of an exercise training programme on abstinence (Sinyor et al., 1982). This study showed a significant difference in abstinence rates measured after eighteen months in those alcoholics who participated in a 6-week exercise programme as opposed to a non-exercising, control group (69% versus 38% respectively).

In addition, there are only a few studies that investigate the physical fitness of alcoholic patients and the effects of exercise training (Gary and Guthrie, 1972; Frankel and Murphy, 1974; Piorkowski and Axtell, 1976; Tsukue and Shohoji, 1981; Sinyor et al., 1982; Palmer et al., 1988). All these studies have important limitations particularly with respect to their

measurements of fitness and the duration of the exercise programmes adopted. Although the study by Sinyor et al. (1982) is by far the most sophisticated study to date, they failed to measure directly such important variables as the maximal oxygen uptake.

These and other studies reveal equivocal findings on resting heart rates of alcoholics, and the prevalence of hypertension and cardiomyopathy in these patients (Pader, 1973; Mckelvy et al., 1980; Saunders et al., 1981; Schweitzer and Mark, 1979).

Although all the above studies support the general hypothesis that physical exercise positively influences both psychological well-being and physical fitness of alcoholic patients, they have several shortcomings in their experimental design. The present study was designed to address these shortcomings and to investigate the following:

(a) the effect of participation in an exercise training programme on the psychological status of alcoholic patients after three weeks of inpatient treatment and again after three months of exercise training;
(b) whether such a programme would improve abstinence after three months;
(c) the general state of fitness of alcoholic patients compared to non-alcoholics;
(d) the physiological adaptations to regular exercise after three months, and

(e) the incidence of cardiomyopathy, elevated resting heart rate and hypertension in the group studied.

The study also investigated blood lipid profiles of alcoholic patients and the changes that occur with (a) abstinence and (b) exercise training. Much of the current research is focused on cardiovascular disease and in particular, the significance of blood lipid concentrations as a predictor of coronary heart didease risk. Increased serum cholesterol concentrations are associated with increased risk of coronary heart disease (Witzum and Schonfeld, 1979). Recent studies show an increase in high density lipoprotein cholesterol (HDL-chol) concentrations with exercise training as well as with alcohol consumption (Klatsky et al., 1981; Ernst et al., 1980; Cowan, 1983; Wood et al., 1983), raising the possibility that alcohol consumption might reduce the risk of coronary heart disease. More recently, attention has shifted to the protective role of the different subfractions of HDL-chol and the results are conflicting (Eisenberg, 1984). This is a most interesting area of research and the present study provides a unique working model to investigate the effect of chronic alcohol abuse on the various blood lipid concentrations as evidenced after three weeks of abstinence, and the effects of exercise training on blood lipid concentrations.

CHAPTER II

LITERATURE REVIEW

1. A DEFINITION OF ALCOHOLISM

It is extremely difficult to define alcoholism. The most commonly used definition is that of physical dependence. Persons who consume large quantities of alcohol on a daily basis for three weeks or more, are likely to develop a tolerance for alcohol, and if the consumption of alcohol is suddenly stopped, withdrawal symptoms often develop indicating physical dependence (Fraser, 1981). However, it is not easy to identify an alcoholic on the basis of physical dependence. The typical "skid-row" alcoholic comprises only 4% of alcoholics in South Africa (Fraser, 1981). The majority of alcoholics are respectable people with families and responsible jobs and the only way they can be identified is via social and/or occupational difficulties as a result of the abuse of alcohol. Thus social and/or occupational impairment as a direct result of alcohol abuse must be included in the definition of alcoholism.

2. IDENTIFYING THE ALCOHOL-DEPENDENT PERSON

The alcohol-dependent person is broadly classified into one of two groups on the basis of their drinking pattern (Ben-Arie, 1985). The first is the "loss of control" drinker who is easily identified as they are frequently intoxicated and as a result often present with social and legal problems. The second is the "inability to abstain" drinker who is not as easily diagnosed as they seldom get intoxicated. These are often referred to as "hidden alcoholics" and may well be respectable members of the community with adequate financial resources to protect them from

the negative effects of their drinking problem. The fact that these individuals are dependent on alcohol, is often only revealed once abstinence is unavoidable, for example during hospitalization. It has been found that in South Africa, 15-25% of all general medical and surgical patients admitted to hospital are alcohol-dependent (Fraser, 1981).

Only about 10% of alcohol-dependent persons are medically diagnosed (Fraser, 1981). It is difficult to diagnose a person as alcohol-dependent, as these idividuals characteristically use denial and minimisation as defence mechanisms to hide their drinking problem. Often this is reinforced by the family or deluded employer. Projection and rationalization are additional defence mechanisms that are commonly used. For instance the "loss of control" types will rationalize that they could stop drinking if they so wished, as they abstain between bouts of excessive drinking. Similarly, the "inability to abstain" types will say they are never drunk.

Alcohol-dependent persons are most likely to be identified by their general practioner. It is therefore important for the family doctor to be aware of the signs of alcohol dependency so that an early diagnoses can be made. The doctor may provide a certificate of leave for a mild gastric disturbance, when in fact the reason for illness is alcohol-abuse. The following criteria by George (1981) are used to diagnose alcohol dependence :

(a) PHYSICAL INDICATORS

* Increased serum gamma-glutamyl transpeptidase (GGT) activity and increased red cell mean corpuscular volume (MCV).
* Medical conditions associated with alcohol abuse include gastritis, pancreatitis, alcoholic hepatitis and hepatic cirrhosis.
* Frequent blackouts (amnesia) caused by the rapid intake of large amounts of alcohol. Although this can occur in persons without a drinking problem, when it occurs frequently, alcohol-dependent drinking should be suspected.
* Frequent bumps to the head.
* Epileptic fits and transient aural or visual hallucinations (or both) are associated with alcohol withdrawal.
* In the chronic state, delirium tremens frequently accompanies alcohol withdrawal.

(b) BEHAVIOURAL INDICATORS

* Impotence, irritability, unduly aggressive behaviour and drinking before a social or stressful event.
* Frequent job changes and absenteeism, particularly on a Monday.
* Early morning drinking
* Frequent legal offences such as drunken driving, motor vehicle accidents and assault are most likely to be alcohol-related.
* Marital and family problems - commonly anxiety or depression in the spouse; behavioural problems in the children and separation or divorce, are often the result of an alcohol problem.

3. TREATMENT OF ALCOHOL DEPENDENCE

The treatment of acute symptoms of physical dependence on alcohol is relatively straightforward, and simple. The difficulty is in treating the long-term psychological aspect of alcohol dependence. In the acute stages, anxiety and depression might require medication. However, drugs generally have a small role in the treatment of alcohol dependence. The only medication used extensively is disulfiram (Antabuse) and vitamin-B supplementation. Disulfiram ensures abstinence during treatment and assists the patient in behaviour modification during rehabilitation. When disulfiram interacts with alcohol, it causes unpleasant physical reactions such as dyspnoea, sweating, palpitations and skin flushing, nausea, vomiting and headaches (Marsden, 1977). The biochemical basis for this reaction is that disulfiram neutralises the enzyme aldehyde dehydrogenase (ADH) through competitive binding with the active portion of the enzyme, nicotinamide adenine dinucleotide (NAD). The accumulation of acetaldehyde as a result of this reaction, is thought to evoke these physical reactions (Marsden, 1977).

The primary mode of treatment for alcoholism is psychotherapy and counselling which aim to stengthen the alcoholic's ability to curb his drinking habits and improve psycho-social adjustment. This demands recognition by the patient of their dependency on alcohol, and guidance by the therapist toward insight and motivation to stop drinking. Psychotherapy takes the form of (a) individual counselling and (b) group therapy.

Individual counselling is essential to develop a good relationship between therapist and patient. The therapist's objectives are to improve the self-esteem of the patient and develop trust in the relationship. Here the patient can express his or her emotional feelings and allow the therapist to clarify their attitude toward their drinking problem. The prognosis for individual psychotherapy is better if the patient is well motivated, is able to express themselves, attends regularly and acknowledges the fact that they have a drinking problem.

Group therapy provides a closer and deeper understanding of each member's inter-personal relationships and social skills. The duration and depth of the sessions depend on the goals and ability of the therapist and the emotional and intellectual abilities of the patients. Loeberstein (1981) distinguishes two types of group setting; (a) Supportive - where patients have limited inter-personal resources and (b) Therapeutic - where more vigorous therapy is applied and the psychological problems of the patient are exposed more clearly. This type of treatment requires that the patients are more psychologically stable.

Self-help groups such as Alcoholics Anonymous (AA) are also a form of group therapy but in the absence of professional control. The fact that support, confrontation or both is provided by other alcoholics rather than by a therapist, is thought to make AA a more effective treatment modality. Members are generally well educated and psychologically stable.

Family therapy is also an integral part of psychotherapy and a form of group therapy. The family of an alcoholic needs help in their own right and their reactions to the patient are also influential in treatment. Most emotional disorders stem from the alcohol problem rather than the contrary. Two organisations in addition to AA offer the family of the alcoholic advice and guidance. They are AL- ANON (for spouses) and ALATEEN (for children).

Apart from AA and other welfare organisations, two other types of facilities are available for the treatment of alcoholism. These are: (a) Treatment units which are usually attached to a hospital or similar medical institution, which offer an out-patient service for detoxification and medical treatment, as well as in-patient treatment. The most important function of such units is to provide a network of long term treatment for the alcoholic patient. (b) Detoxification centres where the more skid-row type of alcoholic is treated. Admission is with minimal formality and the sole purpose of treatment is to manage withdrawal symptoms.

Alcoholic treatment units incorporate psychotherapy, educational techniques, disulfiram, vitamin-B supplementation and encouragement to join AA once discharged. However, the services offered by such units are limited and simply cannot provide for the large number of alcoholic sufferers in need of attention.

In South Africa most of these facilities are available, but on a

very limited scale. The South African National Council for Alcohol and Drug Dependence (SANCA) offers treatment for alcoholics as well as support to their families. Its primary function however, is to educate the public of the dangers of alcoholism. Various alcoholic rehabilitation clinics exist in association with State Hospitals in the major centres. In the Western Cape there are two treatment units run by the Department of Psychiatry, Groote Schuur Hospital. One of these is the William Slater Hospital for Alcohol Rehabilitation, where the present study was performed.

Treatment at the William Slater Hospital consists of a three week inpatient treatment programme limited to 16 patients, as well as an extensive outpatient care programme. On admission the patient is detoxified and clinically assessed. On the basis of a personal interview, patients are allocated to either inpatient or outpatient treatment regimes. Inpatient treatment includes individual and group psychotherapy, family therapy and occupational therapy. Disulfarim is used during treatment and for the initial period after discharge. Vitamin supplementation and where necessary, drug therapy such as tranquilisers are precribed during detoxification. The outcome of treatment for this unit has been reported by Gillis and Keet (1969). These authors found that showed 16% of 797 patients achieved complete abstinence after treatment and a further 23% returned to controlled drinking.

4. THE OUTCOME OF TREATMENT

The outcome of treatment for alcohol dependence is very disappointing. The general consensus is that although most alcoholics improve after treatment, long term follow-up indicates no significant difference in outcome. Various studies have shown inpatient treatment to be no more effective than outpatient care (Emrick, 1975; Costello, 1975; Edwards and Guthrie, 1967; Statton, 1966; Ritson, 1968). Neither was there any difference in outcome derived from long-stay treatment of 80 or more days as opposed to short-stay treatment of approximately 20 days (Willems et al., 1973). These findings are confounded by three major methodological difficulties encountered when doing comparitive studies such as the above. Nevertheless, they indicate a rather pessimistic outlook on the treatment programmes for alcoholics.

The following methodological problems are exposed when evaluating the various studies:

(a) the different definitions of what constitutes improvement,
(b) the different and inadequate follow-up periods used and
(c) the different premorbid characteristics of the patient.

Whether or not total abstinence should be the measure of successful treatment is debatable. It is generally accepted however, that abstention should not be the only goal of treatment. Improvement in other spheres such as social and occupational stability may occur without permanent abstention (Pattison et al., 1969). Return to controlled drinking is felt to be a better goal for the following reasons:

i) drinking in moderation is the socially accepted norm,

ii) most alcoholics admitted for treatment have expressed the desire to be able to return to normal drinking,

iii) considering the above, a treatment regime in which complete abstinence is the goal may evoke a defiant attitude in the patient,

iv) most alcoholics do not become permanently abstinent and

v) perhaps the fact that patients are told that it is impossible to return to controlled drinking, may explain this very phenomenon.

Vaillant et al. (1983) illustrates the shortcoming of inadequate follow-up in the assessment of treatment success. In this study with an eight year follow-up period, 95% of the 107 patients had a relapse during the time and 57% were abstinent for periods longer than 6 months. Thus if evaluated at any point in time, the outcome of treatment might have depicted either failure or success. Such ambiguity disappeared with substantial long term follow-up. Vaillant emphasises the need to study the natural aetiology of alcoholism in order to assess accurately the effect of treatment on outcome. Very few such studies are found in the literature.

It is difficult to compare the outcome of treatment from various studies as improved outcome might be attributed to better premorbid characteristics of the patient rather than the treatment intervention. Therefore it is essential to control for the effects of prognostic attributes on the outcome of

treatement. These are a stable, social and occupational environent (Straus and Bacon, 1951), stable residency, avoidance of crude spirits, preservation of economic resources, absence of criminal convictions, no history of delirium tremens, first admission and weekend binger as oppposed to constant drinking (Marsden, 1977). Gillis and Keet (1969) and Ben-Arie et al. (1983) have shown that a favourable prognosis before treatment correlates well with improvement in drinking behaviour after treatment.

The most significant studies on the outcome of treatment in alcoholics, are those which have attempted to follow-up patients over a long period of time.

Probably the most methodologically sound longitudinal study on the natural history of alcoholism is by Vaillant et al. (1983). A concerted effort was made to follow-up 106 patients treated for 5 days at a detoxification centre, every 18 months for 8 years. Contact of 90% of these patients was achieved after 8 years. The sample used was biased towards severity and chronicity of alcoholism.

The study showed a decline in the prevalence of alcoholism after the age of 45 years. Stable remission was found in 34% of the patients and 29% had died after 8 years. After 1 year, 81% were still alcohol dependent, this had dropped to 26% after 8 years. During the 8 year period, 95% had a relapse but 58% were abstinent for longer than 6 months. This ambiguity might explain

the discrepancies in outcome of treatment in various studies. Of those who died 10% were younger than 40 and 50% were older than 50 years of age. The breakdown of the major causes of death were as follows: 4 died of cirrhoses of the liver, 5 from coronary thrombosis, 4 from cancer, 8 homocides and accidents and only 1 suicide.

Important conclusions from this study were:

(a) alcoholic patients recover not so much because of treatment but because they help themselves,

(b) stable remissions were closely correlated with AA attendances, independent of pre-morbid social stability,

(c) a good pre-morbid psycho-social adjustment favourably influenced short term prognosis, but was less effective in the long term and

(d) stable psycho-social adjustment was synonymous with complete abstinence.

This study certainly exposed the limitations and efficacy of treatment in alcoholism. He concludes that future research should focus on interventions which maximise the natural healing process, are cost effective and incorporate large numbers. He concludes that the best form of treatment would be to encourage patients to attend AA meetings and to seek help at an early stage of the disorder.

In conclusion, the disease concept of alcoholism has proved to be unjustifiable in that the only difference between alcoholics and

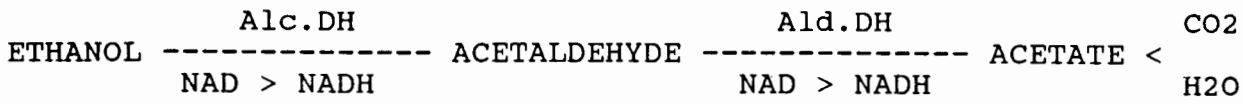
normal individuals is the amount of alcohol consumed and not the result of metabolic or psychological abnormalities as was previously thought. Viewing alcoholism as a disease provides formidable obstacles to its in effective treatment. These are that the public and government bodies view alcoholism as a medical problem to be solved by the medical profession whereas alcoholics themselves believe their problem to be the result of some abnormality which is to be medically treated and is not their own fault. Although contemporary medical treatment is necessary, it is limited and unable to deal effectively with the problem of alcohol abuse and its consequences. Historical evidence shows the extent of alcoholism to be closely related to alcohol consumption per capita, which is sensitive to legislation (Kendell, 1979). Hence it would seem that society and in particular legislators should stop regarding alcoholism essentially as a medical problem and tackle it effectively through legislation (Kendell, 1979).

5. THE METABOLIC EFFECTS OF ALCOHOL

Ingested alcohol is absorbed mainly from the stomach and small intestine (Horn, 1985). Absorption is rapid and is influenced by (a) the volume and dilution of alcohol consumed; (b) the period over which it is ingested and (c) the presence of food in the stomach at the time of ingestion (Horn, 1985). Absorption is even more rapid from the small intestine (Bode, 1978).

Alcohol cannot be stored in the body and must therefore be oxidized. Almost 90% of the circulating alcohol is metabolized in

the cytoplasm of the liver in a two-step reaction (Lieber, 1975):



The primary step is the conversion of ethanol to acetaldehyde, a reaction catalyzed by the cytoplasmic enzyme alcohol-dehydrogenase (Alc.DH). In this reaction, the co-factor nicotinamide adenine dinucleotide (NAD) is reduced. The acetaldehyde is then oxidized by the enzyme aldehyde-dehydrogenase (Ald.DH) to form acetate. The acetate is then further oxidized via the mitochondrial citric acid cycle in liver and muscle to carbon dioxide and water. Fifty to 80% of acetate is metabolised outside the liver.

The metabolic clearance rate of ethanol is relatively constant and does not vary widely, nor is it influenced by blood alcohol levels. This clearance is roughly proportional to body weight. Approximately 10 ml of alcohol is cleared per hour by the average adult (Horn, 1985). Thus the clearance of alcohol is relatively slow and it may take 2,5 - 3 hours to metabolize the alcohol present in 540 ml of beer, or 60 ml of whisky (Horn 1985). The rate limiting step in the oxidation of ethanol, is the recycling of NAD. This is done by shuttling the H ion of NADH from the cytosoplasm to the mitochondria via the malate/oxalo-acetate; lactate/pyruvate and B-hydroxybutyrate/acetoacetate redox pairs.

This ethanol-induced increase in the cytoplasmic NAD:NADH ratio has several potentially deleterious effects on cytoplasmic and

mitochondrial functions. The increased rate of cytoplasmic reduction of pyruvate to lactate inhibits gluconeogenesis as pyruvate is an essential intermediate for the gluconeogenic pathway (Lieber, 1975). If liver glycogen levels are low due to an inadequate carbohydrate intake, as is usually the case in the alcohol-dependent person, hypoglycaemia is induced with the eventual risk of brain damage (Lieber, 1975). Ironically, excessive alcohol consumption is also able to induce hyperglycaemia. Acetate competes with glucose for oxidation, particularly in the muscle tissue, thereby inhibiting glucose uptake from the blood (Lieber, 1975).

The reduced cytoplasmic redox state also interferes with mitochondrial metabolism. The additional hydrogen ion created by the oxidation of ethanol provides an alternative fuel for mitochondrial oxidative metabolism, thereby inhibiting the oxidation of free fatty acids. This causes an accumulation of free fatty acids and low-density lipoproteins. Furthermore, the cytoplasmic to mitochondrial hydrogen shuttles are also precursors for the synthesis of triglycerides. In combination these two pathways explain why lipid accumulates in the liver of alcoholic persons (Lieber, 1975).

Other complications include hyperuricemia, which may exaccerbate gout in susceptible individuals (Lieber, 1975; Faller and Fox, 1982), and repeated depletion of NAD could reduce the production of testosterone in the testis which might explain sexual impotence in male alcoholics (Lieber, 1975).

5.1 THE EFFECT OF ALCOHOL ON THE LIVER

Epidemiological evidence suggests that the alcohol consumption is causally related to the number of deaths due to cirrhosis of the liver (Williams and Davis, 1977). Survey statistics show that up to 65% of reported cases of liver cirrhosis can be attributed to heavy drinking (Hodgson and Thompson, 1976). However, only one-third of heavy drinkers are likely to develop alcoholic hepatitis, while only 10% will develop cirrhosis (Galambos, 1974).

The abnormalities in liver function due to the abuse of alcohol, have been directly associated with the metabolism of alcohol (Lieber, 1975). As described above, the metabolism of alcohol occurs mainly in the liver where it interferes with the metabolism of fat. This results in a fatty liver which may lead to acute hepatitis, decreased liver cell function and obstructed blood flow. Ultimately this condition could result in cirrhosis of the liver.

Animal models have also conclusively demonstrated the association between excessive alcohol consumption and liver disease (Regan et al., 1974). The reason why this exogenous product has such a dramatic effect on a single body organ is because the metabolism of ethanol is organ specific. Furthermore, in addition to the metabolic effects already described, ethanol and particularly acetaldehyde have toxic effects of their own. Chronic alcohol abuse appears to produce persistent mitochondrial changes,

independent of the acute effects described above. These changes include increased mitochondrial fragility and impaired integrity of the inner mitochondrial membrane (Cederbaum and Rubin, 1974).

A sensitive indicator of liver dysfunction is raised plasma gamma glutamyltranspeptidase (GGT) activity, presumably due either to death of the hepatocytes or to increased cell permeability. Elevated serum GGT activity can however occur in a variety of conditions, and its use in the detection of alcohol abuse has been overrated (Horn, 1985). Nevertheless, plasma GGT levels are of considerable value in monitoring alcoholic patients during treatment. Chronic liver damage is strongly indicated if elevated GGT levels persist in the alcoholic patient who is definately abstinent (Horn, 1985).

Other serum enzyme activities that are used to indicate liver status are aspartate aminotransferase (AST) and alanine aminotransferase (ALT). An increase in AST activity without a comparable increase in ALT activity suggests chronic liver disease, while grossly elevated activities for both enzymes indicate acute hepatitis (Kirsch, 1983). Elevated serum AST and ALT activities however, are short-lived. Thus their serum activities must be determined at a very early stage of the disease if they are to be of value. The serum enzyme activities only indicate liver dysfunction and are not indices specifically for alcohol-induced liver damage (Horn, 1985).

Clinically, patients with fatty infiltration of the liver are often asymptomatic, but vague malaise and ill-defined right upper

quadrant pain may be present. Hepatomegaly is invariably present and there are usually mild abnormalities in liver function tests. Uncomplicated fatty liver is considered to be a benign condition, reversible with abstinence from alcohol (Williams and Davis, 1977). Alcoholic hepatitis is characterized by hepatocyte necrosis and patients present clinically with fever, malaise, upper abdominal pains and frequently become jaundiced. Hepatomegaly is found on clinical examination. Biochemical tests of liver function are invariably abnormal. However, the degree of clinical illness varies widely and some patients may be asymptomatic yet display histological evidence of alcoholic hepatitis.

Cirrhosis of the liver due to alcohol is usually micronodular in type. However, as the disease progresses, liver cell hyperplasia makes it become macronodular which makes it difficult to determine the cause of the disease. A further feature of alcoholic cirrhosis is iron overload which can be seen histologically from a liver biopsy (Williams and Davis, 1977).

5.2 ALCOHOL AND MALNUTRITION

There is controversy concerning the relative roles of alcohol and associated nutritional deficiency as the cause of alcoholic liver disease. Studies have demonstrated that certain dietary deficiencies cause hepatic lesions (Davis, 1977). However, these lesions differ in a number of ways from those produced by alcohol. Hence dietary factors alone cannot be the primary cause

of alcoholic liver diseas.

Heavy alcohol consumption has been associated with certain vitamin and mineral deficiences (Visocan, 1983). This could be the result of decreased ingestion of nutrients; malabsorption as a result of a direct toxic effect of alcohol on the gastrointestinal mucosa (folic acid, thiamine, cyanocobalamin and ascorbic acid), and to pancreatic insufficiency causing malabsorption of the fat soluble vitamins; interference of ethanol with the metabolism of certain vitamins (retinol, thiamine, riboflavin and pyridoxine), and ethanol-induced hyperexcretion of zinc, calcium and magnesium (Visocan, 1983).

5.3 THE EFFECT OF ALCOHOL ON THE PANCREAS

It has long been recognized that alcohol ingestion may cause both acute and chronic inflammation of the pancreas (Horn, 1985). The incidence of alcoholic pancreatitis varies from about 10% of total cases in some countries to 90% in some states of America (Horn, 1985). In South Africa, the only reported incidence is 59% (Louw et al., 1967). Mortality rate in alcoholics in the United States due to acute pancreatitis is 9.1% (Horn, 1985). The hypothesized pathogenesis is that duodenal inflammation caused by alcohol may split the papilla of Vater , causing a reflux of duodenal content into the pancreatic ducts. The duodenal enzyme enterokinase is thought to activate proteolytic pancreatic enzymes which enter the interstitial tissue giving rise to pancreatitis (Horn, 1985).

5.4 THE EFFECT OF ALCOHOL ON THE HEART

Although cardiac disease has been associated with excess alcohol consumption since the nineteenth century, few data exist on the actual incidence of clinically significant heart disease in the alcoholic population.

Originally heart disease associated with alcoholism was attributed to the malnutrition found in alcohol-dependent persons; hence it was assumed that the form of heart disease found in alcoholics was Beri-beri Heart Disease. Subsequently however, it was shown that heart disease was unresponsive to thiamine treatment in well nourished alcoholics. This entity is known as alcoholic cardiomyopathy and is manifest by arrythmias, palpitations, conduction defects, dyspnoea and fatigue. During the early stages of this condition, patients complain of vague chest pain and attacks of arrhythmias. Upon physical examination, gallop rythmn, cardiomegaly and holo systolic apical murmurs are heard (Pader, 1973). After 2-4 years, the heart is enlarged and develops biventricular failure with ventricular dysrhythmias.

Despite the widespread acceptance that cardiomyopathy may develop even in well-nourished alcoholics, there are few data on the prevalence of electrocardiographic (ECG) changes suggestive of cardiomyopathy in alcoholics. The most recent and thorough study by Pader (1973) found one of the 100 patients randomly selected to have an ECG abnormality, while 3 presented with clinical heart disease. The later subjects were all men, between the age of 45

and 55 years.

A few studies have demonstrated functional impairment of the heart without clinical evidence of heart disease, that is latent left-ventricular dysfunction, in alcoholics. This observation comes from exercise-induced haemodynamic studies which show an increased left-ventricular end-diastolic pressure (Gould et al., 1969) and prolongation of the pre-ejection phase of left-ventricular contraction (Spodick et al., 1972; Wu et al., 1976) in alcoholic patients.

However, these haemodynamic changes are not the only factors influencing cardiac function; hence it has been possible for some alcoholic patients to achieve similar exercise performances as normal individuals despite these documented abnormalities in myocardial function.

Schweitzer and Mark (1979) showed that alcoholic patients achieved similar workloads and durations of exercise as did controls, despite a prolonged pre-ejection period (PEP) and increased PEP : left ventricular ejection time ratio. However, a lower maximum heart rate and systolic blood pressure response to exercise was found in the alcoholic group. A possible explanation for this discrepancy is the higher stroke volume needed to achieve a similar cardiac output during exercise in the alcoholic group (Schweitzer and Mark, 1979).

A number of studies show that alcohol-dependent persons have

higher resting heart rates (HR) compared to the normal population (Frederickson and Hed, 1958; Gary and Guthrie, 1972; Pader, 1973; Frankel and Murphy, 1974; McKelvy et al., 1980; Sinyor et al., 1982). For example, Frederickson and Hed (1958) and Pader (1973), found that 30% and 14% respectively of alcoholic patients had resting heart rates above 95 beats per minute (bpm) compared to 72 bpm in normal subjects. McKelvy et al. (1980) on the other hand found a mean resting HR of 77 bpm in the 48 subjects tested; only 6% had a resting HR above 95 bpm. This discrepancy may be due to the age differences of the subjects tested in the respective studies. The mean age in McKelvy et al.'s study (1980) was 23 years, compared to 46 years in the other studies.

Several clinical and epidemiological studies have shown a positive association between resting blood pressure (BP) and alcohol consumption (Beevers, 1977; Matthews, 1976; Saunders et al., 1981). For instance Saunders et al. (1981) found that 46% of 132 alcoholic patients tested had a systolic BP greater than 140 mmHg (normal 120 mmHg), and 34% a diastolic BP higher than 90 mmHg (normal 80 mmHg). In this group there were significant correlations between blood pressure and mean daily amount of alcohol consumed over the previous three months, serum GGT activity and mean corpuscular volume. Furthermore, the study showed that with abstention, elevated BP returned to normal but rose again once heavy drinking resumed, indicating a causal relationship between BP and alcohol consumption. Other studies have not however shown such a direct relationship (Frankel and Murphy, 1974).

Mortality studies on alcoholics indicate that heavy drinking is associated with increased mortality from coronary artery disease (CAD) (Pell and D'Alonzo, 1973; Dyer et al., 1980). In contrast, various studies show an inverse relationship between small to moderate amounts of alcohol consumption and CAD (Yano et al., 1977; La Porte et al., 1980; Klatsky et al., 1981; Marmotte et al., 1981). The reasons for these anomalous findings are not clear.

Earlier studies found no significant differences in the degree of coronary occlusion between alcoholics and moderate or non-drinkers (Hirst et al., 1965; Sackett et al., 1968), while more recent studies show a significant inverse relationship between alcohol consumption and the extent of CAD (Moore et al., 1975; Hennekens, 1983). These latter findings are supported from cross-sectional studies in which coronary angiography showed a significant inverse association between the level of alcohol consumption and the degree of coronary artery occlusion (Barboriak, 1977; Barboriak et al., 1979).

It has been suggested that the negative association between alcohol consumption and CAD, is due to the effect of alcohol on serum HDL-cholesterol concentrations (Castelli et al., 1977; Ernst et al., 1980). The negative association between serum HDL-cholesterol concentration and the risk of CAD was first suggested by Barr et al. (1951), but the current interest in this association was only aroused again by the work of Miller and Miller (1975). Since then, several epidemiological studies have

supported this negative association which suggests that elevated serum HDL concentrations have a protective role against the development of CAD (Rhoades et al., 1976; Castelli et al., 1977; Gordon et al., 1977; Miller et al., 1977; Roussouw et al., 1985).

The proposed mechanism for this protective role of increased serum HDL concentration on CAD, is that this lipid component is involved in the nett removal of cellular cholesterol from arterial smooth muscle cells (Stein et al., 1977). However, the complex nature of reverse cholesterol transport, the role of HDL in this process, and the complex structure of the HDL molecule has been stressed (Berger, 1984; Eisenberg, 1984). It has been suggested that HDL-cholesterol should be interpreted in the light of the total lipid profile in order to provide a better measure of cardiovascular risk (Castelli et al., 1983; Berger, 1984). Total blood cholesterol concentration as well as low density lipoprotein cholesterol concentration are the two other measures considered to be independent, positive risk factors for coronary artery disease (Ross and Harker, 1976; Witzum and schonfeld, 1979).

The serum HDL molecule has been further subdivided into two subfractions on the basis of different densities, namely HDL2 and HDL3 (Anderson, 1978; Eisenberg 1984). It has been suggested that the HDL2 subfraction is the active component of HDL while HDL3 is relatively inactive (Gofman et al., 1966; Witzum and Schonfeld, 1979). This postulate remains a controversial issue (Eisenberg, 1984).

The most common environmental factors known to influence HDL-cholesterol concentrations, are alcohol and exercise. Although the earlier studies were cross-sectional in design, recent prospective studies support the finding that alcohol and exercise respectively, increase HDL-cholesterol concentrations independent of confounding variables (Hulley and Gordon, 1981; Haskell et al., 1984; Haffner et al., 1985). Further evidence for the effect of alcohol on HDL-cholesterol concentrations, come from studies performed on alcoholic patients (LaPorte et al., 1981; Devenyi et al., 1981; Ekman et al., 1981; Taskinen et al., 1982; Duhamel et al., 1984). All these studies show that HDL-cholesterol concentration is higher in the alcoholic group. This alcohol-induced increase in HDL-cholesterol concentration, is reversible, as the levels return to normal within 10 to 14 days of abstinence. It has been postulated that part of the mechanism involved in the alcohol-induced increase in HDL-cholesterol concentration is the increased lipoprotein lipase activity associated with the metabolism of alcohol (Taskinen et al., 1985).

The studies of alcoholic subjects show that the increase in HDL-cholesterol concentration is mainly due to an increase in the HDL2 subfraction (LaPorte et al., 1981; Taskinen et al., 1982; Duhamel et al., 1984). Other studies using moderate levels of alcohol administered to non-alcoholics, show an increase in the HDL3 rather than HDL2 subfraction (Haskell et al., 1984; Haffner et al., 1984). A further study administered alcohol to a healthy population, but in large doses, showed an increase in the HDL2 rather than HDL3 subfraction (Taskinen et al, 1982).

Recent studies have suggested an interesting possibility that HDL-cholesterol subfraction concentrations be used to differentiate stages of liver disease in alcoholic patients (Denvyi et al., 1981; Duhamel et al., 1984; Sabesin and Weidman, 1984). Measurements of HDL2 and HDL3-cholesterol concentration and the respective apoprotein fractions of each (Apo A-1 and A-2), correlated well with the progression of liver disease (Duhamel et al., 1984). A significant finding showed that patients with severe liver disease failed to show a rise in HDL2-cholesterol concentration after acute ethanol ingestion (Denvyi et al., 1981).

The few prospective studies of exercise and serum HDL-cholesterol concentrations, provide strong evidence for the positive association between physical fitness and serum HDL-cholesterol concentrations (Keins et al., 1980; Cowan, 1983; Wood, 1983). The major criticism of these findings is that weight-loss accompanying physical training rather than the training per se, is responsible for the changes observed in HDL-cholesterol concentration (Lipson, 1978; Wood, 1983). However, some studies did control for weight losses and the results were nevertheless significant (Keins et al., 1980; Huttunen, 1982; Nakamura et al., 1983). The postulated mechanism for this observed exercise-induced change in HDL-cholesterol concentration, is an increased lipoprotein lipase activity with training in skeletal muscle as well as adipose tissue (Nikkila et al., 1978). The exercise-induced changes in HDL-cholesterol concentration are manifest mainly in the HDL2 subfraction (Kuusi et al., 1982). This is

compatible with the hypothesis that HDL2, rather than HDL3 is actively involved in the protective role of HDL in coronary artery disease.

5.5 ALCOHOL-INDUCED NEUROMUSCULAR DISORDERS

The neuromuscular complications of alcohol abuse are widespread. These complications include acute and chronic alcoholic myopathy, generalized peripheral neuropathy, epilepsy, chronic cerebellar syndrome, central pontine myelinolysis, amblyopia, subdural haematoma, Wernicke-Korsakoff syndrome and dementia (Marsden, 1977).

Acute alcoholic myopathy is a rare disease characterized by aching tender muscles with oedema and associated weakness which follows severe alcohol abuse (Carlson et al., 1969). Myoglobinuria and hyperkalaemia are common and renal failure may occur. It is hypothesized that the acute muscle necrosis results from alcohol-induced inhibition of muscle phosphorylase (Marsden, 1977). The condition is reversible with alcohol withdrawal. Chronic alcoholic myopathy is characterized by gradually progressive weakness of the proximal muscles of the lower limbs in particular. Whether or not this is a true myopathy is debatable, as the electromyographic and histological changes are similar to those that result from peripheral nerve degeneration (Marsden, 1977).

Peripheral neuropathy seems to develop in only 10% of alcoholics.

The severity varies from asymptomatic neuropathy, manifest as absent ankle jerks, to severe neuropathy. In the majority of cases the legs alone are affected, and the autonomic and cranial nerves are usually spared. Symptoms are pain and weakness in the legs. Also common is parasthesia or "burning feet". The pathogenesis is unclear. The direct effect of alcohol itself is unlikely to be the critical factor as restoration of a normal diet despite continued drinking, improves the neuropathy. There is a strong rsemblance between alcohol-related neuropathy and nutritional neuropathies such as beri-beri and pellagra. The general concept is that these neuropathies are a result of multiple vitamin deficiencies, particularly of the B-complex vitamins. Hence, alcoholic neuropathy responds to dietary and vitamin therapy, albeit very slowly.

Central nervous system complications include the well known Wernicke-Korsakoff syndrome. Only recently has the relationship between Wernicke's encephalopathy (punctuate haemorrhages in the grey matter around the third and fourth ventricles and the aqueduct of the cerebellum) and Korsakoff's psychosis (amnesic syndrome related to alcoholic polyneuropathy) been recognised beyond doubt as manifestations of a single disease (Marsden, 1977). Again, this disorder is related to vitamin deficiency, specifically thiamine (vitamin B-1). If corrective therapy is introduced at an early stage of the illness, recovery may be complete. However, once the disorder has progressed to Korsakoff's amnesic syndrome (chronic stage), the patient will not respond to vitamin supplementation. Hence the prophylactic

therapy of vitamin B-complex in established alcoholics.

Other disorders include epilepsy, cerebellar syndrome (destructive lessions in the cerebellar cortex which are most commonly revealed cliniocally as gait ataxia), central pontine degeneration (demyelination of the nerve tracts from the pons, characterized by pseudobulbar palsy and quadriplegia) and Marchiafava-Prignami disease (similar to the latter in which demyelination of the corpus callosum occurs, causing progressive dementia often with fits). These are rare disorders and are only diagnosed at autopsy. Most of these disorders are related primarily to malnutrition as a result of alcoholism rather than the toxic effects of alcohol per se.

6. THE POSSIBLE ROLE OF EXERCISE IN THE TREATMENT OF ALCOHOLISM

The alcohol-dependent person is characterized by a poor psychological well-being, which includes poor self concept, low self-esteem, lack of self-confidence, anxiety and depression (Conner, 1962; Vanderpool, 1969; George, 1981; Cooper, 1983). Often they have a dependent personality and use alcohol either to satisfy dependency needs or as a mechanism to avoid stress. The subsequent abuse of alcohol causes shame, guilt, resentment and depression which stimulates further drinking. Hence dependency on alcohol becomes a vicious circle impairing psychological health.

Furthermore, these patients are often malnourished , inactive and usually smoke heavily (Visocan, 1983). This state of ill-health

causes poor body image, which further contributes to the poor self-image and overall psychological condition of the alcohol-dependent person.

Regular physical activity, on the other hand, is known to improve psychological well-being and provides a positive mechanism for coping with stress (Michael, 1976; Young, 1979; Folkins and Sime, 1981; Blumenthal et al., 1982). Not only does regular exercise enhance body image but it can also improve self-esteem, relieve tension and depression (Folkins, 1970; Collingwood, 1972; Young and Ismail, 1976; Morgan and Horseman, 1976).

A single bout of physical exercise has been associated with a sense of well-being often described as tranquil and euphoric (DeVries, 1981). It also relieves depression and anxiety. The current hypothesis for this phenomenon is that the endogenous opiate beta-endorphin is released during physical exercise (DeVries, 1981). However, this has not been conclusively established (Harber and Sutton, 1984)

The physiological benefits of regular exercise training are better substantiated. Training induces a change in body composition and significant physiologial and biochemical changes at cellular level (McArdle et al., 1981; Holloszy and Coyle, 1984). Percentage body fat is decreased with training, accompanied by a reduction in body mass or an increase in lean body mass, or both (McArdle et al., 1981). The physiological adaptations include thickening of the left ventricular wall of

the heart muscle which increases stroke volume and subsequently cardiac output which potentially enhances oxygen consumption (McArdle et al., 1981). This results in a decreased heart rate and percentage of maximum oxygen consumption corresponding to a particular workload after training. The resting heart rate and blood pressure during rest and submaximal exercise are decreased. At a cellular level, there is an increased capacity of skeletal muscle for the generation of adenosine triphosphate (ATP) via oxidative phophorylation by (a) an increase in the number and size of the mitochondria, (b) an increased enzyme activity to mobilize and oxidize fats and (c) an increased skeletal muscle myoglobin content McArdle et al., 1981).

Considering the above findings, it seems obvious that an exercise programme might increase the athletic fitness of the alcoholic patient. Further, a review of the literature reveals that there are very few articles reporting the use of exercise in the treatment of alcoholism (Gary and Guthrie, 1972; Frankel and Murphy, 1974; Piorkowski and Axtell, 1976; Tsukue and Shohoji, 1981; Sinyor et al., 1982; Palmer et al., 1988). Some of these studies were concerned solely with the assessment of physical fitness in alcoholic patients and did not study the potential psychological benefits associated with improved fitness.

To date, there are only three documentated studies of the effect of physical fitness on psychological changes in alcoholics (Gary and Guthrie 1972; Frankel and Murphy 1974; Palmer et al., 1988). The findings of Gary and Guthrie (1972) indicate very little

correlation between physical fitness and psychological improvement. However self-esteem and sleep patterns improved in the exercise group. The study was statistically unsound and the measures of physical fitness were inadequate. Furthermore, the exercise training used was unlikely to result in any physiological adaptation as the subjects only ran 1,6 km per day for 20 days.

Frankel and Murphy (1974) employed a more adequate physical training programme. Their subjects trained for an hour five times per week for a period of twelve weeks. The measures of physical fitness used (resting heart rate, blood pressure and sub-maximal step test) are outdated. However, the investigators did show a significant reduction in anxiety and depression with exercise. The authors emphasized the importance of an exercise programme in increasing self-esteem of the alcoholic person.

Palmer et al. (1988), showed a significant reduction in state and trait anxiety and depression but no change in self-concept or aerobic capacity. Although the exercise programme complied to the American College of Sports Medicine's minimum requirements for exercise training, the programme lasted for only 28 days.

In 1976, Piorkowski and Axtell studied the effect of a circuit training exercise programme on the physical fitness of alcoholic patients. They were solely concerned with the type of exercise training and did not look at psychological changes accompanying the physiological adaptations. But again, the measure of physical

fitness used was inadequate, and furthermore, the duration of the exercise programme was only three weeks. Nevertheless the study showed that circuit training was an effective means of training for the alcoholic patient.

An interesting study of the premise that exercise would enhance the self-concept of the alcoholic person, was that of Tsukue and Shohoji (1981). They introduced basketball, played for an hour three times per week, into the treatment of alcoholic patients. Their main concern was a type of movement therapy that would ameliorate the neurological defects of the patients and improve their state of physical health. The physical condition of the subjects was not stated and no measure of physical fitness was made. However,it was concluded that the movement therapy improved the physical condition of the patients, most noticeably their co-ordination, strength, agility, skill and flexibility. No measures of personality or psychological changes were made.

The most recent and sophisticated study on the use of exercise in the treatment of alcoholics is that of Sinyor et al. (1982). The physical training programme used in this study was adequate and comprised five one hour sessions per week, for six weeks. The measure of physical fitness used was also more sophisticated than that of the previous studies; yet only an indirect estimate of maximum oxygen consumption was used. This is not an useful measure of athletic fitness. The main objective of the study was to determine the effect of training on subsequent abstinence rates. The results showed a positive correlation between

participation in an exercise programme and sustained abstinence after three months. Although this is the average follow-up period used in most studies, in view of the findings by Vaillant et al. (1983), this is considered short-term. Nevertheless, Sinyor et al. found the abstinence rate after three months to improve from 36,9 % before the introduction of the exercise programme to 69,3 % afterwards. They also showed that the adaptations to an exercise programme in alcoholics was similar to that found in normal persons.

CHAPTER III

MATERIALS AND METHODS

1. INTRODUCTION

The study was designed to investigate the effect of an exercise training programme on the outcome of alcoholic patients undergoing an initial three week inpatient treatment programme at the William Slater Hospital for Alcohol Rehabilitation in Rondebosch. The William Slater Hospital offers an intensive three week inpatient treatment programme as well as an outpatient service. Alcohol or drug dependent persons are self-admitted or referred to the hospital. These patients are screened and subsequenty allocated to either outpatient care or to inpatient treatment. All discharged inpatients automatically become outpatients. Only twelve to fifteen patients can be accommodated as inpatients at the hospital. Hence admission is done on a rotational basis with only four to five new patients admitted each week.

Due to the rotational system of admission, subjects for the study had to be sequentially recruited. Random selection of subjects to two groups would not have protected the internal validity of the study due to the small sample size, and interaction between study groups. In addition, it was deemed unethical to offer exercise treatment to randomly selected patients only. Therefore a two-group pre/post design was used. Data were gathered on control subjects prior to the implementation of the exercise training programme, when subjects were recruited and tested for the exercise group.

2. SUBJECTS

Volunteer subjects were recruited from patients admitted to the William Slater Hospital for inpatient treatment. All admitted patients who met with the following criteria, were approached and invited to participate in the study:

(a) a history of problem drinking exceeding four years,

(b) no history of cardiovascular disease or any other disability which would prohibit the patient from exercising,

(c) male, under 45 years of age, and

(d) resident in the immediate viciniy of Cape Town.

All subjects were therefore volunteers and gave their informed consent.

Twelve consecutively recruited patients served as controls in the study (average age 32,2 years). Three of these subjects failed to complete the follow-up test. Once data had been gathered for the control group, recruitment for the exercise group began. Nine subjects were tested initially (average age 31,3 years), but only five completed the follow-up test.

3. EXPERIMENTAL PROCEDURE

The experimental procedure involved a series of tests performed on all subjects on three different occassions:

(a) Test 1.

All subjects were tested on admission to the hospital. The following tests were performed: (i) anthropometric measurements, (ii) estimated alcohol consumption, (iii) psychometric testing,

(iv) blood tests and (v) a twelve lead electrocardiogram (ECG) stress test.

(b) Test 2.

All subjects were tested immediately prior to discharge (ie. after three weeks of inpatient treatment). The following tests were performed: (i) psychometric testing, (ii) blood tests and (iii) an incremental workload exercise test to exhaustion on the cycle ergometer.

(c) Test 3.

Approximately three months after discharge, subjects were followed-up and the following tests performed: (i) anthropometric measurements, (ii) estimated alcohol consumption, (ii) psychometric testing, (iv) blood tests and (v) an incremental workload exercise test to exhaustion on the cycle ergometer.

All physiological and psychometric testing was done at the MRC/UCT Bioenergetics of Exercise Research Unit, Department of Physiology, University of Cape Town Medical School. Full blood count and blood lipid assays were kindly performed at the Department of Chemical Pathology, Red Cross War Memorial's Children Hospital. Serum enzyme activities were determined by the Department of Chemical Pathology, Groote Schuur Hospital. The details of all these tests are described in the following sections.

4. ANTHROPOMETRIC MEASUREMENTS

The subject stood in bare feet with the heels, buttocks, back and occiput flattened against a wall. The vertical height was then measured from the ground to the top of the cranium and recorded to the nearest centimetre. Body mass was measured using an electronic Seca 770 Alpha Personal Scale (Vogel and Halke, Hamburg). Subjects were weighed dressed in shorts only and the mass recorded in kilograms to the nearest 100g.

Skinfold thickness was measured using a Holtain skinfold caliper (Holtain Ltd, Crosswell, UK) by the method of Durnin and Rahman (1967). Measurements were taken from the right side of the body in a relaxed standing position, at the following four sites : (a) Biceps - over the midpoint of the muscle belly with the arm hanging loosely at the subject's side; (b) Triceps - midway between the olecranon and tip of the acromion; (c) Subscapular - immediately below the tip of the inferior angle of the scapula at approximately 45 degrees to the vertical, and (d) Suprailiac - immediately above the iliac crest in the anterior axillary line. All skinfold measurements were performed by the author on both occasions in order to ensure consistency of technique. Percentage body fat was computed using the formula listed in Appendix A.

5. ALCOHOL CONSUMPTION AND THE OUTCOME OF TREATMENT

A history of each subject's drinking problem was obtained on admission by the attending psychiatrist who personally interviewed each subject and assessed the extent of his drinking

problem. A rating score of the subject's drinking pattern and estimate of the amount of alcohol consumed one month prior to admission, was ascertained. An improvised questionnaire of habitual alcohol use (Mehrabian and Russel, 1978) was used to assist the psychiatrist in estimating the amount of alcohol consumed (in grams per day) one month prior to admission. The four-point rating scale of drinking pattern (Gillis and Keet, 1969), was used to rate each subject's drinking pattern before admission and again three months after hospital discharge. Subjects who failed to complete the follow-up tests were nevertheless given a rating based on written reports taken by the community sister. These ratings were also performed by the attending psychiatrist.

The following scale for drinking pattern was used:

DRINKING PATTERN	SCORE
Complete abstinence	1
Abstinent with occasional breaks	2
Intermittent drinking	3
Continuous drinking	4

6. PSYCHOMETRIC TESTING

The Profile Of Mood States (POMS) questionnaire and the Californian Adjective Check List (ACL) questionnaire were used to gauge the psychological status of the subjects during the course of the study. These tests have been shown to be reliable and valid measures of emotions such as tension, depression, anger,

confusion, self-confidence, self-control and defensiveness (McNair et al., 1971; Gough et al., 1965).

The POMS measures six different mood states, defined as Tension, Depression, Anger, Vigour, Fatigue and Confusion. Subjects were asked to rate how they had felt over the past three days, for each of the 65 adjectives listed in the questionnaire. Scores are rated on a five-point scale (0 to 4) in which 0 indicates a rating of "not at all" and 4 "extremely". Raw scores for the various mood states were calculated as the sum of rating scores for the specific adjectives listed under each particular mood state. All raw scores obtained were converted to standard T-score values using the prescribed tables (McNair et al.,1971). A seventh score defined as the Overall Emotional Stability Score (OESS) was determined by the sum of the T-scores obtained for the five negative mood states (Tension, Depression, Anger, Fatigue and Confusion), less the T-score for Vigour and the total then divided by six (the total number of mood states). This score gave an indication of the subject's overall emotional stability.

The standard T-scores obtained relate to a normal distribution with a score of 50 corresponding to the median. Hence scores on either side of the median indicate either an increased or decreased mood state. For instance a high score in the Tension factor would imply the subject was very tense at the time of testing whereas a low score would imply the opposite. The median for the OESS would be 33 and a score greater than this would imply decreased emotional stability whereas a score less than 33

would indicate increased emotional stability.

The ACL questionnaire comprises a total of 200 adjectives. In this test the subject is requested to circle only those adjectives which describe the way he feels. From the adjectives indicated by the subject, raw scores are obtained for various psychological traits such as Defensiveness, Self-confidence, Self-control and Personal Adjustment (adaptability and trust). A further score for the Number of Adjectives answered was obtained. This score indicates drive and confidence. As with the POMS, all raw scores were converted to standard T-scores. Thus scores greater than 50 meant an accentuation of a particular trait whereas a low score indicated a decrease in the trait concerned.

7. BLOOD ANALYSES

Blood samples were drawn on the morning of each testing occasion after the subjects had fasted for at least 10 hours. Plasma or serum was obtained by spinning the respective vacutainer test tubes in a refrigerated Beckman centrifuge (model TJ-6) for 20 minutes at 2000 rpm and stored frozen for later analysis. Plasma was analysed to determine the concentrations of total plasma cholesterol, triglyceride, high density lipoprotein cholesterol (HDL-chol) and low density lipoprotein cholesterol (LDL-chol), and of the HDL-chol subfractions, HDL2 and HDL3. Whole blood samples were used for full blood count (FBC) analysis.

The following assay methods were used:

(a) Total plasma cholesterol concentration was assayed using the enzymatic colorimetric method of Boehringer Mannheim : Monotest Cholesterol CHOD:PAP method (cat.no. 236691) with modifications so that the Multistat III Instrumentation Laboratory, Micro Centrifugal analyser system could be used. The standard used was Precilip Boehringer Mannheim Lyophilised control serum based on human serum (cat.no 125067, lots 3-372 and 2-376). Controls used were LA, LB and LC from the Centre For Disease Control, Atlanta Georgia, USA. Bichromatic analysis was performed and the of total cholesterol concentration determined.

(b) HDL-cholesterol concentration was assayed manually after LDL was precipitated using heparin/manganese. The supernatant was assayed using the same method as above but with Preciset Cholesterol (cat.no. 125512) as standard and inhouse pooled plasma aliquoted and stored at -70 degrees C, as controls. The instrumentation used for this assay was the Varian DMS 100 UV/Visible Spectrophotometer.

(c) HDL3-cholesterol concentration was assayed manually after dextran/sulphate precipitation of the supernatant from the heparin/manganese precipitation above. The same method was used as for HDL-cholesterol assay. The numeric difference between the total HDL-cholesterol concentration measured and the HDL3-cholesterol concentration measured, was considered to be the HDL2-cholesterol concentration.

(d) Plasma triglyceride concentrations were assayed using the indirect colorimetric Enzymatic Trinder Method (Carlo Erba, code 543181). This assay measures the glycerol released from

triglyceride after hydrolysis by lipoproteinlipase (LPL). Precilip Boehringer Mannheim as above, was used as standard and the Multistat III plus Instumentation Laboratory Micro Centrifugal Analyser System used for bichromatic analysis. The controls used were LA, LB and LC as above.

(e) LDL-cholesterol concentration was calculated as follows:

[LDL-chol] = [Total Chol] - ([Triglyceride]/2,18 - [HDL-chol])

(all measures in mmol/l).

All these assays were performed in the Department of Chemical Pathology at the Red Cross War Memorial Childrens Hospital.

Serum samples were used for determination of the activities of gamma glutamyltranspeptidase (GGT), aspartate (AST) and alanine (ALT) aminotransferase, and for creatinine concentration. These assays were performed using an automated technique (Technicon SMAC II, Tarrytown, New York, USA.) in the Department of Chemical Pathology at Groote Schuur Hospital.

8. ELECTROCARDIOGRAPHIC STRESS TEST

An electrocardiographic (ECG) stress test was performed on all subjects upon admission. This was to identify any signs of cardiomyopathy, cardiovascular disease or hypertension which may have resulted from alcohol abuse. This test also cleared the patients for participation in the exercise programme and provided the baseline necessary for exercise prescription.

The standard 12-lead ECG was recorded at rest and at regular intervals during exercise with a Marquette Electrocardiograph Case system (Marquette Electronics Inc., Mikwaukee, USA). Heart rate (HR) and blood pressure (BP) were also monitored during the test. Subjects walked on a treadmill according to the Balke protocol of incremental workload. The test was terminated when the patient either (a) developed any adverse signs or symptoms, (b) had achieved at least 80 % of his age predicted maximum heart rate or (c) felt that he could no longer continue the test.

9. MAXIMAL EXERCISE TEST

A maximal exercise test was performed on all subjects in the study, at the time of hospital discharge and again after three months. Heart rate, oxygen consumption and blood lactate concentrations were measured each minute during a progressive incremental exercise test on an electronically-loaded bicycle ergometer (Godart NV, Bilthoven, Holland). The exercise testing protocol was as follows :

After resting measurements had been made, the subject began pedalling at a workload of 15 watts, after which the workload was increased progressively by 15 watts every minute until the subject voluntarily terminated the test. Subjects were required to pedal at 60 revolutions per second throughout the test. The workload (watts) at which the test was terminated was labelled the peak workload.

Heart rate was measured using three pellet electrodes applied to

the subject's anterior chest wall and connected to a Life Trace electrocardiograph monitor (Albury Instruments Ltd., London, UK). Resting heart rate and minute heart rate readings at the end of each workload were recorded in beats per minute (bpm). The heart rate recorded at peak workload will be refered to as the peak heart rate.

Oxygen consumption was measured using open-circuit calorimetry. Subjects cycled with a counterbalanced headgear to support a mouthpiece with two one-way valves (Hans Rudolph, model no. 2700, Kansas City, USA), connected to 35mm clear-bore tubing on both the inspiratory and expiratory ends. Room air was inspired through the open inspiratory end via a Morgan Ventilation Monitor Mark 2 (PK Morgan Ltd.,Gillingham, UK). The latter was calibrated regularly using a Collins chain-compensated gasometer (Collins Inc., Braintree, USA). Minute inspiratory volume (l/min) and respiratory rate (breaths/min) were recorded at the end of each minute. A nose clip prevented nasal breathing. The exhaled air was passed via 1,5 m tubing to a 15 litre perspex mixing chamber with baffles. This mixed-expired air was continuously sampled through anhydrous CaSO4 ("Drierite", Vacumed Inc., Ventura, USA) to the pick-up heads of a Beckman OM-11 Oxygen Analyser (model 242B) and a Beckman LB-2 Carbon dioxide Analyser (model 240M), both from Beckman Instruments (Illinois, USA). The outputs from these two analysers were recorded on a Beckman Respiratory Recorder (model RR-2). Both analysers were calibrated before and after each test , using previously calibrated gases of known composition. Fractional expiratory concentrations for oxygen and

carbon dioxide at each workload were extrapolated from the recorder trace after the test. Rates of oxygen uptake (VO2) and carbon dioxide production (VCO2) were calculated using conventional equations (Jones, 1982). The VO2 (ml/kg/min) measured at the peak workload is referred to as the peak VO2, and the percentage VO2 at each workload was calculated as a percentage of this value.

Blood lactate concentrations (mmol/l) were measured from blood sampled every minute via a 22G Jelco teflon intravenous catheter (Critikon, Florida, USA) inserted into a forearm vein. The catheter was connected to 150 cm polythene tubing (internal diameter 0,86 mm, Portex Ltd., Kent, UK) which was looped around the rollers of an Eyela Microtube Pump (model MP-3, Tokyo Rikakikai Co., Tokyo, Japan). The speed of the pump was set so that sampling occured 60 seconds after the blood left the forearm vein. Heparin was flushed through the tubing before the test to prevent blood clotting during sampling. Blood samples of approximately 1 ml were collected every minute during the test as well as for the first four minutes after the cessation of the exercise test. Each blood sample was collected in a pre-weighed plastic test tube containing 2 ml of ice cold 70% perchloric acid solution (PCA). The blood and PCA was thoroughly mixed using a Heidolph Whirlimixer and the deproteinised sample stored on ice. Subsequently, the samples were re-weighed and spun in a refrigerated Beckman Centrifuge (model TJ-6) for 10 minutes at 2000 rpm. The supernatant was removed and stored frozen for later analysis of lactate concentration.

The following variables pertaining to blood lactate concentration were calculated :

(a) The lactate turnpoint, defined as the workload (watts) corresponding to a clearly visible increase in the blood lactate concentration (Matter et al., 1987). It is now appreciated that the lactate turnpoint is an imprecise definition as blood lactate concentrations increase progressively during exercise rather than as a threshold phenomenon (Hughson et al., 1987). Nevertheless, as defined, the "lactate turnpoint" can be used as a measure of athletic ability and adaptation to training (Noakes et al., 1990).

(b) The peak blood lactate concentration, which is the blood lactate concentration corresponding to the peak workload achieved in the maximum exercise test.

(c) The highest or maximum blood lactate concentration measured either during the maximum exercise test or during recovery.

10. THE EXERCISE TRAINING PROGRAMME

The exercise training programme for those subjects in the exercise group consisted of two phases: (a) the initial three week period of inpatient treatment at the William Slater Hospital and (b) a three month period after discharge from the hospital.

During the initial three week period of inpatient treatment at the William Slater Hospital, three supervised exercise sessions of one hour each per week were included in the subject's treatment regime. These sessions took place at the hospital in the late afternoon and were open to all patients at the hospital.

Three cycle ergometers and a number of free-standing weights were made available for patients to use for the duration of the study. The exercise programme was designed to meet the minimum requirements for frequency, duration and intensity as recommended by the American College of Sports Medicine (1980). The supervised exercise sessions consisted of:

(a) 10 minutes of stretching exercises,

(b) 10 minutes of slow walking or jogging,

(c) 20 minutes of calisthenic and srengthening exercises,

(d) 10 minutes of cycling and

(e) 10 minutes of stretching (cool down).

The exercise sessions were neither strenuous nor rushed. The object was to make the exercise sessions enjoyable, relaxed and pleasant. The 10 minutes of stretching and 10 minutes of walking or jogging was treated as a warm-up period. The following 20 minutes of calisthenic exercise and 10 minutes of cycling, comprised the actual duration of exercise at which an intensity level of between 60 and 75% of the maximum heart rate was maintained. This range was calculate for each subject based on their maximum heart rate achieved during the initial stress test performed. Each subject was shown how to monitor his own heart rate to ensure that this intensity was maintained. The calisthenic exercises were done in circuit fashion with each exercise station lasting 30 seconds. The following exercises were performed in sequence and the circuit repeated three times: star jumps, sit ups, press-ups, lateral arm raises, upright rowing, burpees, arm curls and rope skipping (see appendix B for

illustrations).

During this period all subjects were educated on aspects of fitness and the principles of successful training. This was essential as the next phase of training was not officially supervised. After hospital discharge, a centralised exercise programme under supervision proved to be impractical. An individual home-programme was prescribed and monitored for each subject. Here the emphasis was on aerobic activities such as walking, running and cycling for a minimum duration of 20 minutes per day, four days per week. Intensity was monitored as before. Subjects were issued with Logbooks to record details of their training and their progress was monitored regularly. The duration and intensity of the exercise programme was progressively increased according to each subject's goals and ability.

11. STATISTICAL METHODS USED

For descriptive statistical purposes, all result are expressed as means and standard deviations. The unpaired Student's t-test for independent samples was used to determine any statistically significant differences between sample means obtained for the two study groups. The paired Student's t-test was used to determine any statistically significant differences observed over time within each of the two groups. The two-tailed level of significance was used for most of the variables measured, as it was accepted in the null hypothesis that no difference between

groups existed. As this was not true for the physiological changes observed with training, a one-tailed level of significance was used in the analysis of these variables. A two-way analysis of variance was used to find any significant change in a particular variable measured over the three month time period, within a single study group, for example, psychological changes.

The confidence limits accepted in the study were probabilities less than 5 or 1 %, and in some cases probabilities of less than 0,5 and 0,1 % were found. This meant a p value of $< 0,05$ or 0,01 and $< 0,005$ and 0,001 respectively.

CHAPTER IV

EXPERIMENTAL RESULTS

1. ANTHROPOMETRIC DATA OF SUBJECTS PARTICIPATING IN THE STUDY

The anthropometric data of all 21 subjects who participated in the study are presented in Table 1. The mean age of the subjects was 31,7 years and the mean height 176,2 cm. The mean body mass was 73,8 kg, the mean percentage body fat 14,8 percent, and he mean lean body mass 63,6 kg.

Table 1. Anthropometric data of all 21 subjects.

Age (years)	31,7 ± 5,8
Height (cm)	176,2 ± 13,6
Body mass (kg)	74,5 ± 8,8
% Body fat	14,8 ± 3,8
Lean body mass (kg)	63,6 ± 6,7

Results are expressed as means and standard deviations.

Anthropometric data of subjects studied in the exercise and control groups respectively, are presented in Table 2. No significant differences between the two groups were found. The following changes were observed within each group during the three months of observation: Percentage body fat was significantly reduced in both groups studied ($p < 0,02$), and lean body mass increased significantly only in the exercise group ($p < 0,02$). Note that body mass increased slightly in the exercise group and decreased in the control group , but neither change was significant.

Table 2. Anthropometric data for exercise and control groups at admission and after three months.

	Exercise group		Control group	
Variable	Admission (n=9)	3 Months (n=5)	Admission (n=12)	3 Months (n=9)
ge (years)	32,2 +_6,0		31,3 +_5,8	
Height (cm)	175,9 ± 14,1		176,1 ± 13,9	
Body mass (kg)	72,0 ± 9,1	73,5 ± 8,0	76,4 ± 8,2	74,8 ± 9,2
% Body fat	13,1 ± 4,6	10,5 ± 4,2 **	15,8 ± 6,4	13,2 ± 6,2 **
Lean body mass (kg)	62,6 ± 5,8	65,8 ± 6,3 **	64,3 ± 6,2	64,9 ± 6,5

Results are expressed as means and standard deviations.

No statistical significance between groups was found.

** $p < 0,02$ for unpaired data (3 Months vs Admission).

2. ALCOHOL CONSUMPTION PRIOR TO ADMISSION

All subjects had a history of alcohol abuse for at least five years. The mean estimated daily alcohol consumption one month prior to admission was approximately 300 grams of alcohol per day. Mean values for the exercise and control groups were very similar (see Table 3).

Table 3. Estimated alcohol consumption one month prior to admission.

	Exercise group (n=9)	Control group (n=12)
Alcohol consumption (g/day)	311 ± 82	313 ± 77

Results are expressed as means and standard deviations.

No statistical significant difference between groups was found.

3. THE OUTCOME OF TREATMENT

The best objective measure of the outcome of treatment was the rating score for drinking pattern given to all subjects participating in the study, after three months. The percentage of scores achieved in each study group at admission and after three months for each of the four ratings, are presented in Table 4.

Table 4. Percentage drinking pattern scores at admission and after three months in the exercise and control groups.

Drinking Pattern	Exercise group Admission (n=9)	Exercise group 3 months (n=9)	Control group Admission (n=12)	Control group 3 months (n=12)
Complete abstinence	0	67 *	0	25
Occasional breaks	0	0	0	16
Intermittent drinking	44	22	42	16
Continuous drinking	56	11	58	43

Results are expressed as percentages.

* $p < 0,05$ for Exercise vs Control groups (Kolmogorov-Smirnov "goodness of fit" test).

The results show that the exercise group had significantly better dinking patterns after three months ($p < 0,05$).

4. THE INCIDENCE OF CARDIOMYOPATHY

No evidence for cardiomyopathy was found in any of the 21 subjects studied. No abnormalities were seen on the electrocadiographic traces either at rest or during exercise, nor were any clinical signs of this condition detected by the examining physician. Mean resting heart rate (HR) and blood pressure (BP) measurements determined at rest appear in Table 5. The results show that the group studied was normotensive with normal heart rates for untrained persons. No significant difference between groups was observed.

Table 5. Mean resting heart rate and blood pressure readings recorded in the exercise and control groups.

Variable	Exercise group (n=9)	Control group (n=12)
Resting HR (bpm)	77 ± 10	79 ± 8
Systolic BP (mmHg)	123 ± 12	134 ± 12
Diastolic BP (mmHg)	82 ± 10	85 ± 10

Results are expressed as means and standard deviations.

No statistically significant difference between groups was found.

5. MAXIMAL EXERCISE TEST RESULTS

The results of the physiological variables measured during the initial maximal exercise test performed on 21 alcoholic subjects are listed in Table 6. The physiological response to the graded maximal exercise test is depicted in Figure 1. Results are expressed as means and standard deviations.

Table 6. Initial maximal exercise test results for all subjects.

Variable Measured	Value (n=21)
Resting HR (bpm)	76,7 ± 10.9
Peak HR (bpm)	181,1 ± 10,9
Peak VO2 (ml/kg/min)	43,8 ± 8,0
Peak Workload (watts)	216,7 ± 30,1
Blood Lactate Measurement	**Value (n=10)**
Peak [Lactate] (mmol/l)	7,1 ± 1,5
Highest [Lactate] (mmol/l)	10,6 ± 1,5
Lactate Turnpoint:	
(% VO2 max)	49,3 ± 4,1
(% max workload)	51,1 ± 3,9

HR = Heart rate

VO2 = Oxygen consumption

Results are expressed as means and standard deviations.

The mean resting heart rate and peak heart rate for the group were normal. The peak heart rate achieved was a little below the age predicted maximum. The peak workload achieved and peak oxygen

consumption were also normal, but relatively low. This was to be expected as these subjects were untrained and of poor health. The lactate measurements were also normal.

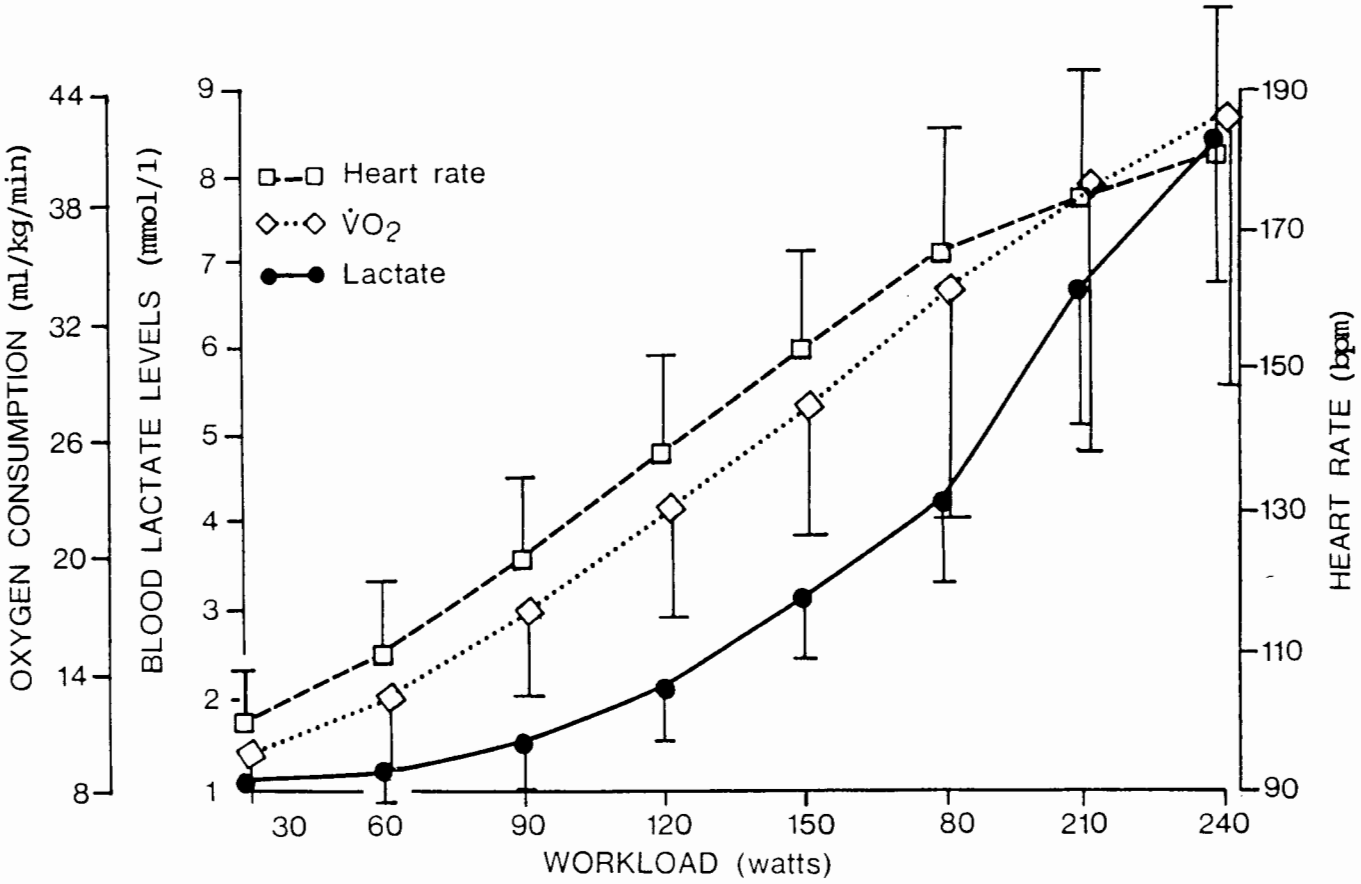


Figure 1. The mean oxygen consumption, blood lactate concentration and heart rate response to increasing workloads during a graded maximal exercise test in the 21 subjects.

Figure 1 shows a linear increase in heart rate and oxygen consumption with increasing workload as found in normal persons. The mean heart rate response near maximal workload depicts a plateau phenomenon.

Blood lactate concentrations increase as a curvilinear response to increasing workload. Overall, the physiological response to exercise in these subjects appears to be quite normal, which is surprising.

Table 7 lists the physiological variables measured during the initial maximal exercise test in the exercise and control groups respectively.

Table 7. Initial maximal exercise test results for the exercise and control groups.

Variable Measured	Exercise group (n=9)	Control group (n=12)
Resting HR (bpm)	76,7 ± 10.9	79,3 ± 7,6
Peak HR (bpm)	181,1 ± 10,9	178,4 ± 9,6
Peak VO2 (ml/kg/min)	43,8 ± 8,0	34,9 ± 4,2 **
Peak Workload (watts)	216,7 ± 30,1	200,0 ± 24,9
Blood lactate Measurement	**Exercise group (n=4)**	**Control group (n=6)**
Peak [lactate] (mmol/l)	7,1 ± 1,5	6,5 ± 1,7
Highest [lactate] (mmol/l)	10,6 ± 1,5	9,6 ± 2,9
Lactate turnpoint:		
(% VO2 max)	49,8 ± 3,6	48,9 ± 3,8
(% max workload)	51,9 ± 4,1	50,8 ± 3,5

HR = Heart rate

VO2 = Oxygen consumption

Results are expressed as means and standard deviations.

** $p < 0,02$ for Exercise vs Control group.

The only significant difference between the two groups at the initial test was the significantly higher peak oxygen consumption in the exercise group (43.8 and 34,9 ml/kg/min for the exercise and control groups respectively, $p < 0,02$). The mean resting heart rate (HR) for the exercise and control groups was 76,7 and 79,3 beats per min (bpm) respectively. No statistical significant difference between the groups in any other variable was found.

Table 8 lists the changes observed in the physiological variables measured in the five subjects who continued to exercise for three months and the nine control subjects who were followed up after three months respectively. The physiological response to the maximal exercise test in these subjects as well as the change observed after three months, are depicted in Figures 2 and 3.

The results in table 8 show that no significant changes occured over the three month period in the control group whereas every variable measured changed significantly in the exercise group after three months of training ($p < 0,05$). The mean resting heart rate for the exercise group was significantly lower after three months of training (from 78,0 bpm to 65,4 bpm, $p < 0,05$). The control group showed no change in mean peak heart rate after three months, whereas the exercise group showed a significant fall in mean peak heart rate after training (181,1 to 176,6 bpm, $p < 0,05$). The three month follow-up test showed a significant increase in the mean peak VO2 of the exercise group (41,0 to 46,0 ml/kg/min, $p < 0,05$), while no change was observed in the control group after three months. The peak lactate concentration,

highest lactate concentration and lactate turnpoint were all significantly different after three months in the exercise group ($p < 0,02$). as was the case with the other variables, no change was evident in the control group. All measurements after three months were significantly different in the exercise versus control groups ($p < 0,05$).

Figure 2 shows the mean heart rate response to the corresponding workload in the exercise (n=5) and control (n=9) groups respectively. Results are expressed as means and standard deviations. The figure shows that the mean heart rate at each workload during exercise up to 180 watts fell significantly in the exercise group after three months of training ($p < 0,05$). Surprisingly, the mean heart rate at each workload increased significantly after three months in the control group ($p < 0,02$).

Figure 3 shows the mean percentage maximal oxygen uptake at the corresponding workloads in the exercise (n=5) and control (n=9) groups. Results are expressed as means and standard deviations. This figure shows that the percentage maximal oxygen consumption at each workload was significantly lower in the exercise group after three months of training ($p < 0,02$), whereas no change was observed in the control group.

Table 8. Maximal exercise test results before and after three months in the exercise and control groups.

Variable Measured	Exercise group (n=5) Admission	Exercise group (n=5) 3 Months	Control group (n=9) Admission	Control group (n=9) 3 Months
Resting HR (bpm)	78,0 ± 9,8	65,4 ± 6,4 *	79,3 ± 7,6	81,4 ± 8,9 #
Peak HR (bpm)	182.8 ± 4,7	176,6 ± 3,8 *	178,4 ± 9,6	182,4 ± 9,2 #
Peak VO2 (ml/kg/min)	41,0 ± 7,2	46,0 ± 7,7 *	34,9 ± 4,2	35,3 ± 4,5 ##
Peak workload (watts)	225,0 ± 31,8	255,0 ± 42,4*	200,0 ± 24,9	201,7 ± 17,0 #
Blood lactate Measurement	**Exercise group (n=4)**		**Control group (n=6)**	
Peak [Lactate] (mmol/l)	7,9 ± 1,6	5,8 ± 0,8 **	6,5 ± 1,7	6,3 ± 2,3
Highest [Lactate] (mmol/l)	9,7 ± 1,1	11,3 ± 1,0 **	9,6 ± 2.9	9,6 ± 0,8
Lactate Turnpoint: (% VO2 max)	49,8 ± 3,6	65,3 ± 6,2 **	48,9 ± 3,8	47,8 ± 3,6 ##
(% max workload)	51.9 ± 4,1	67,2 ± 6,9 **	50,8 ± 3,5	50,1 ± 3,8 ##

HR = Heart rate

VO2 = Oxygen consumption

Results are expressed as means and standard deviations.

* $p < 0,05$ and ** $p < 0,02$ for paired data (3 Months vs Admission)

# $p < 0,05$ and ## $p < 0,02$ for Exercise vs Control groups.

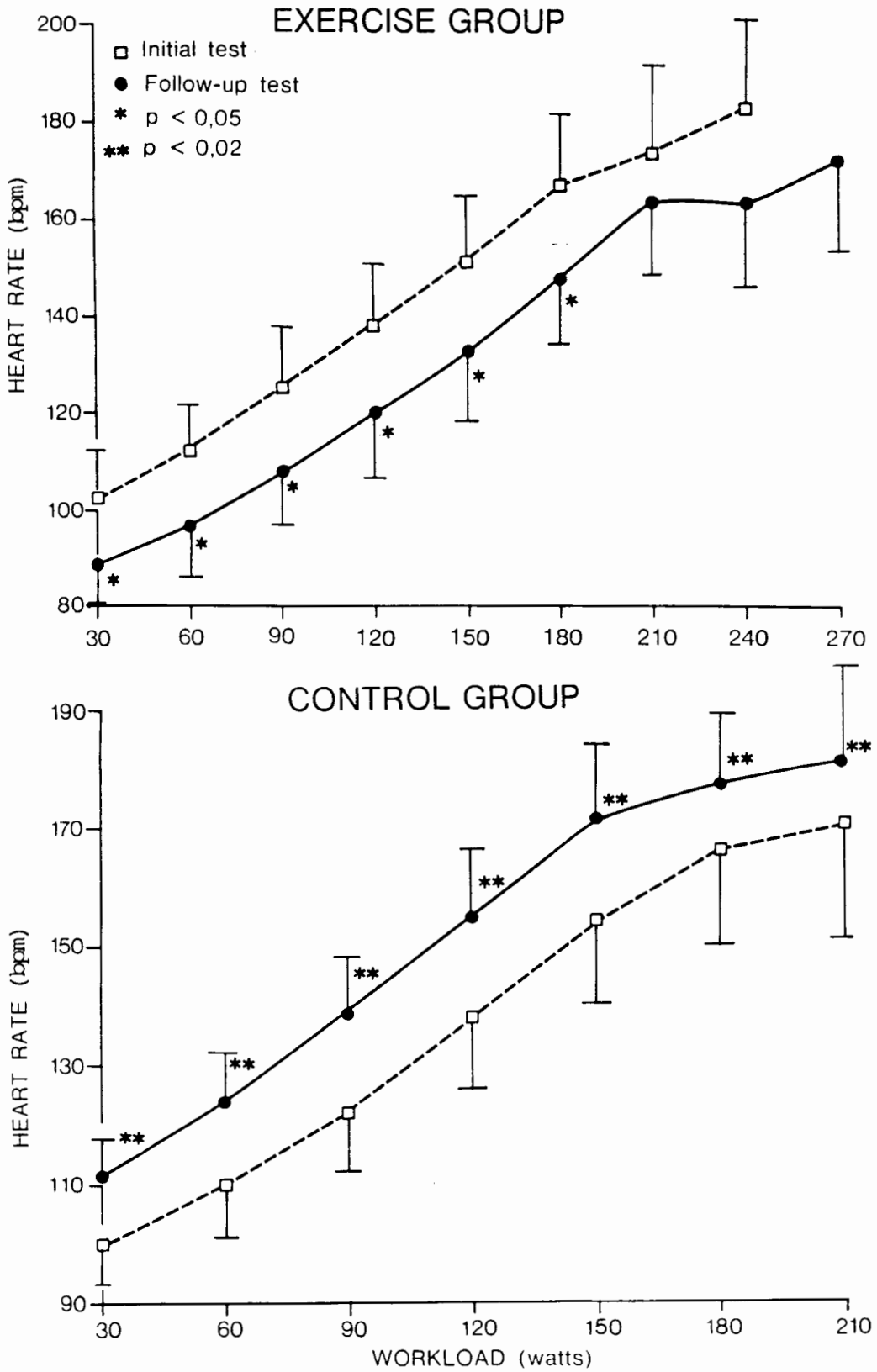


Figure 2. The mean heart rate response to increasing workload during a graded maximal exercise test at admission and after three months in the exercise and control groups.

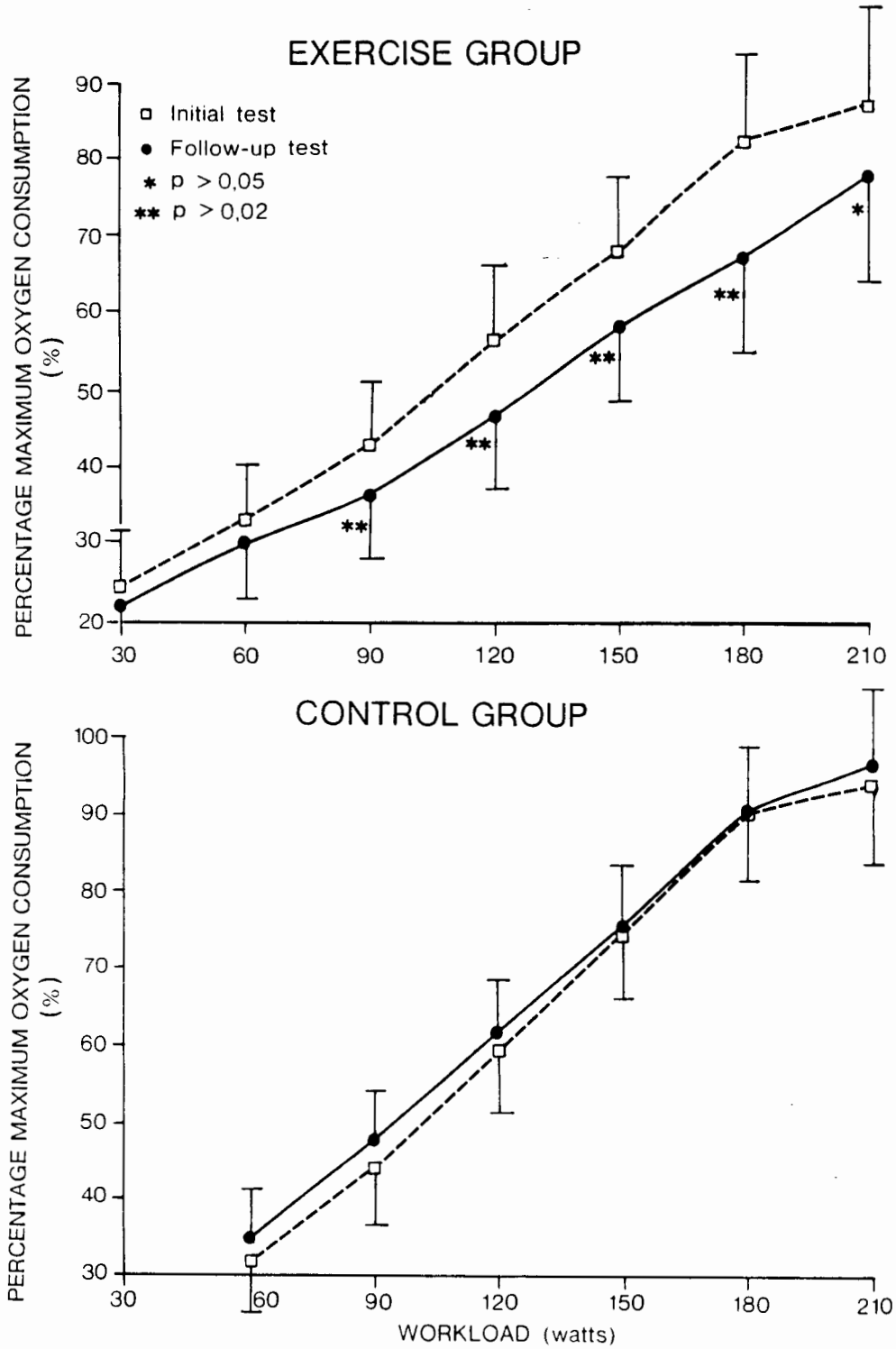


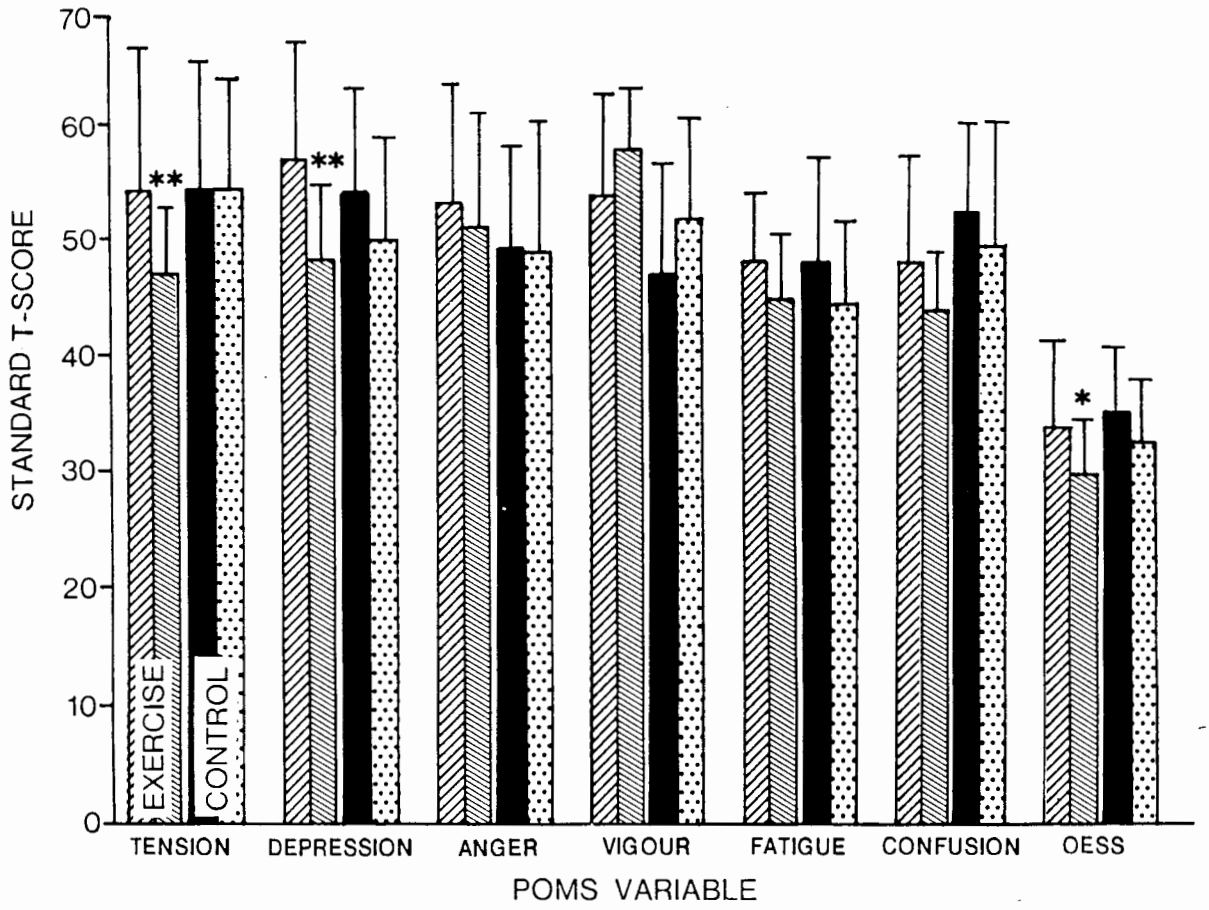
Figure 3. The percentage maximal oxygen consumption at different workloads during a graded maximal exercise test at admission and after three months in the exercise and control groups.

All the above results show conclusively that subjects tested in the exercise group depicted the classical training response to three months of exercise. In particular the maximum oxygen consumption and peak workload increased significantly and the blood lactate concentration, heart rate and oxygen consumption at each workload decreased significantly.

6. PSYCHOMETRIC TEST RESULTS

Results of the two questionaires completed by all subjects at admission, after three weeks and again after three months, are presented in the figures below.

Figure 4 shows the mean standard T-scores obtained for the variables measured in the Profile Of Mood States questionnaire at admission and after three weeks in the exercise (n=9) and control (n=12) groups. No significant differences between groups was observed neither at admission nor after three weeks. Note that the seemingly higher score for Vigour observed in the exercise group was not statistically significant. Very interestingly, all the scores for all subjects were around the median for the average population. The changes observed after three weeks of inpatient treatment showed very similar trends in the two study groups . No significant changes were found in the control group whereas the exercise group showed significant changes in the Tension and Depression scores ($p < 0.02$), as well as the Overall Emotional Stability Score ($p < 0.05$).



Exercise group at admission =

Exercise group after three weeks =

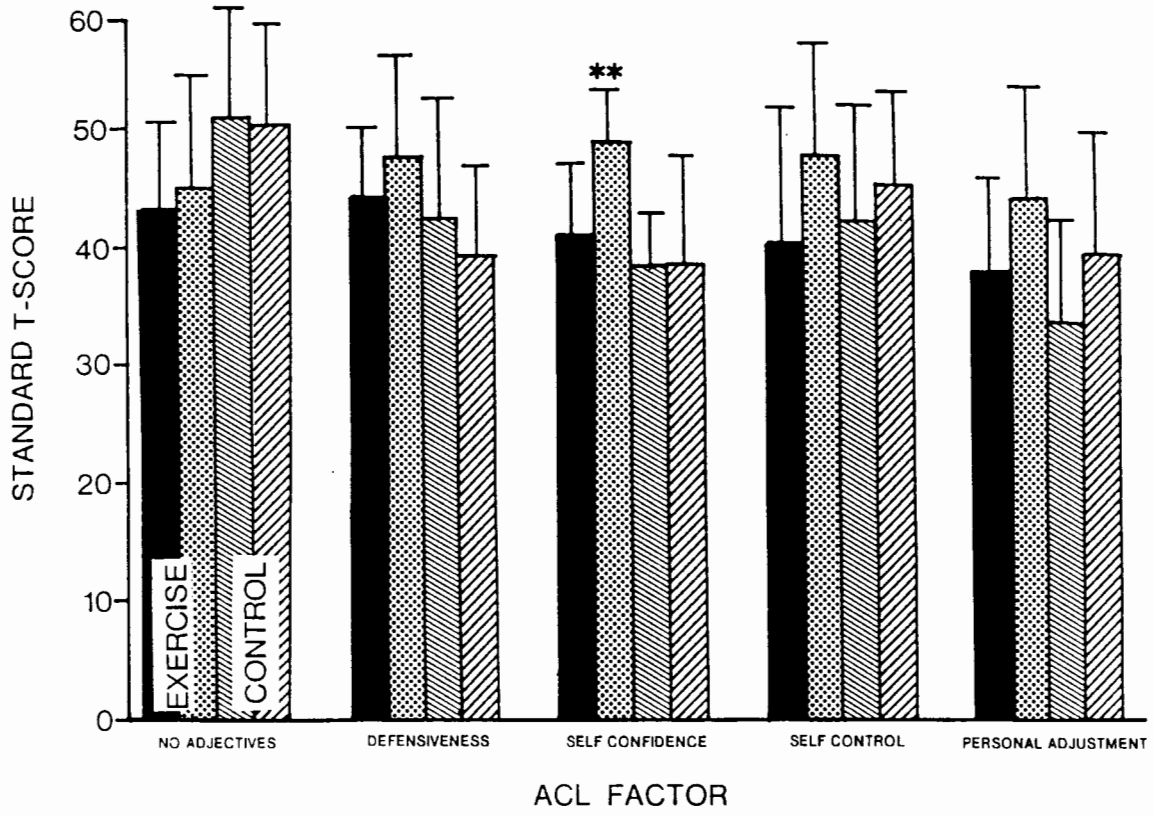
Control group at admission =

Control group after three weeks =

* $p < 0,05$ and ** $p < 0,02$ for paired data (3 Weeks vs Admission)

No statistical significance between groups was found.

Figure 4. Mean standard T-scores for the Profile Of Mood States variables measured at admission and after three weeks in the exercise and control groups.



Exercise group at admission =

Exercise group after three weeks =

Control group at admission =

Control group after three weeks =

** p < 0,02 for paired data (3 Weeks vs Admission).

No statistical significance between groups was found.

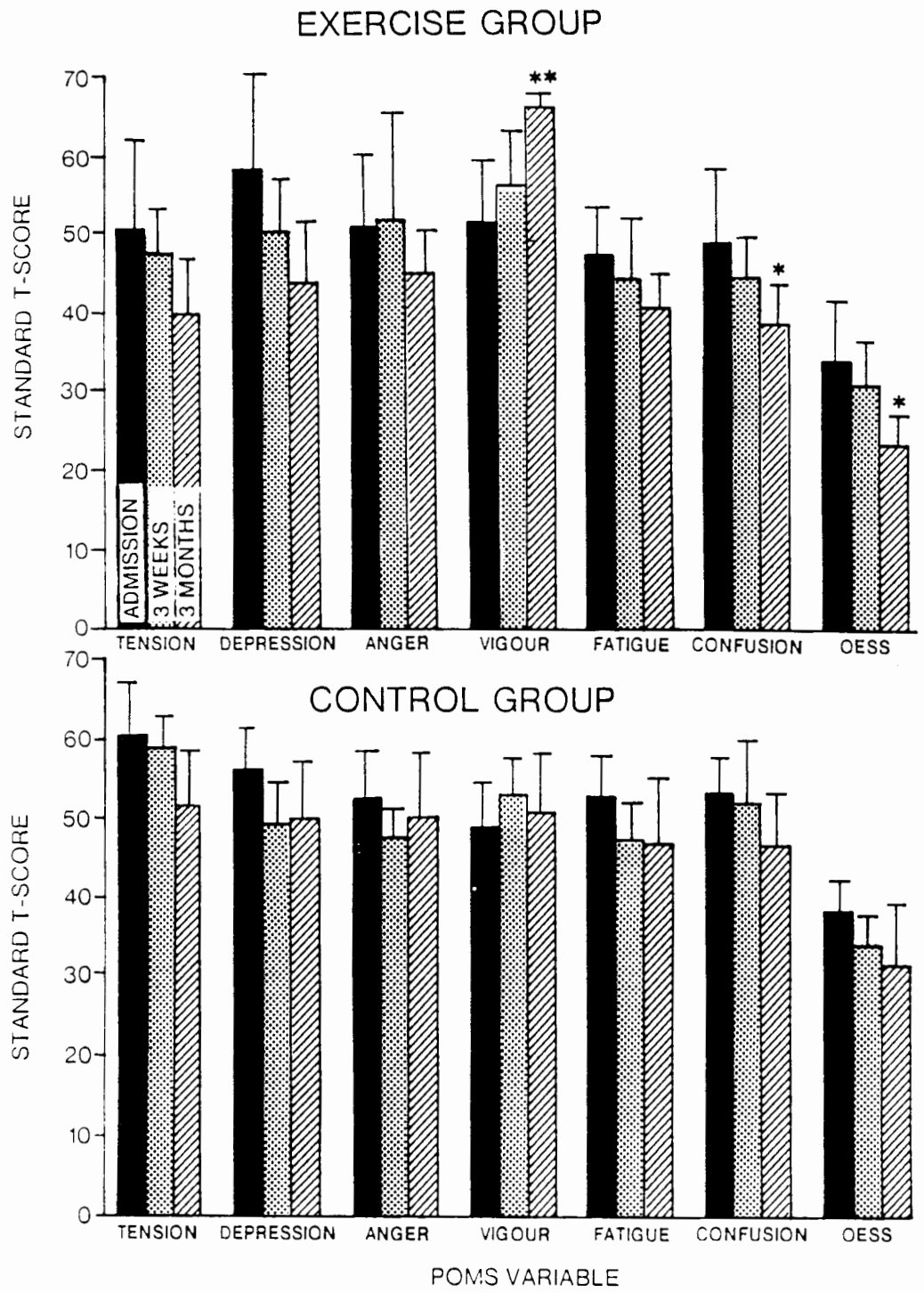
Figure 5. Mean standard T-scores for the Adjective Check List variables measured at admission and after three weeks in the exercise and control groups.

Figure 5 also shows the mean standard T-scores obtained for the variables measured in the Adjective Check List at admission and after three weeks in the exercise (n=9) and control (n=12) groups. Again, no significant differences were found between the two study groups either at admission or after three weeks. However, most standard T-scores for both groups were below the median, in particular the scores for Self-confidence, Self-control and Personal Adjustment. Although the Number of Adjectives score was lower in the exercise group than the controls, this was not statistically significant. Very little change was observed in the control group after three weeks whereas the exercise group showed an improvement in all five variables. The only statistically significant change in the exercise group after three weeks was the score in Self-confidence ($p < 0.02$).

Figure 6 shows the mean standard T-scores obtained for the variables measured in the Profile Of Mood States questionnaire at admission, after three weeks and after three months in the exercise (n=5) and control (n=7) groups respectively. After three months, a definite trend towards improved scores is evident in all variables measured in the exercise group. A decrease in Tension, Depression, Anger and Fatigue scores was observed although none were statistically significant. The increase in the Vigour score ($p < 0,02$) and the decrease in the Confusion and Overall Emotional Stability scores, were however statistically significant ($p < 0,05$). No definate changes were observed in the control group after three months and no statistically significant

changes were found. No statistical significance between groups was found, although the Vigour and OESS in the exercise group were markedly increased in comparison with the control group.

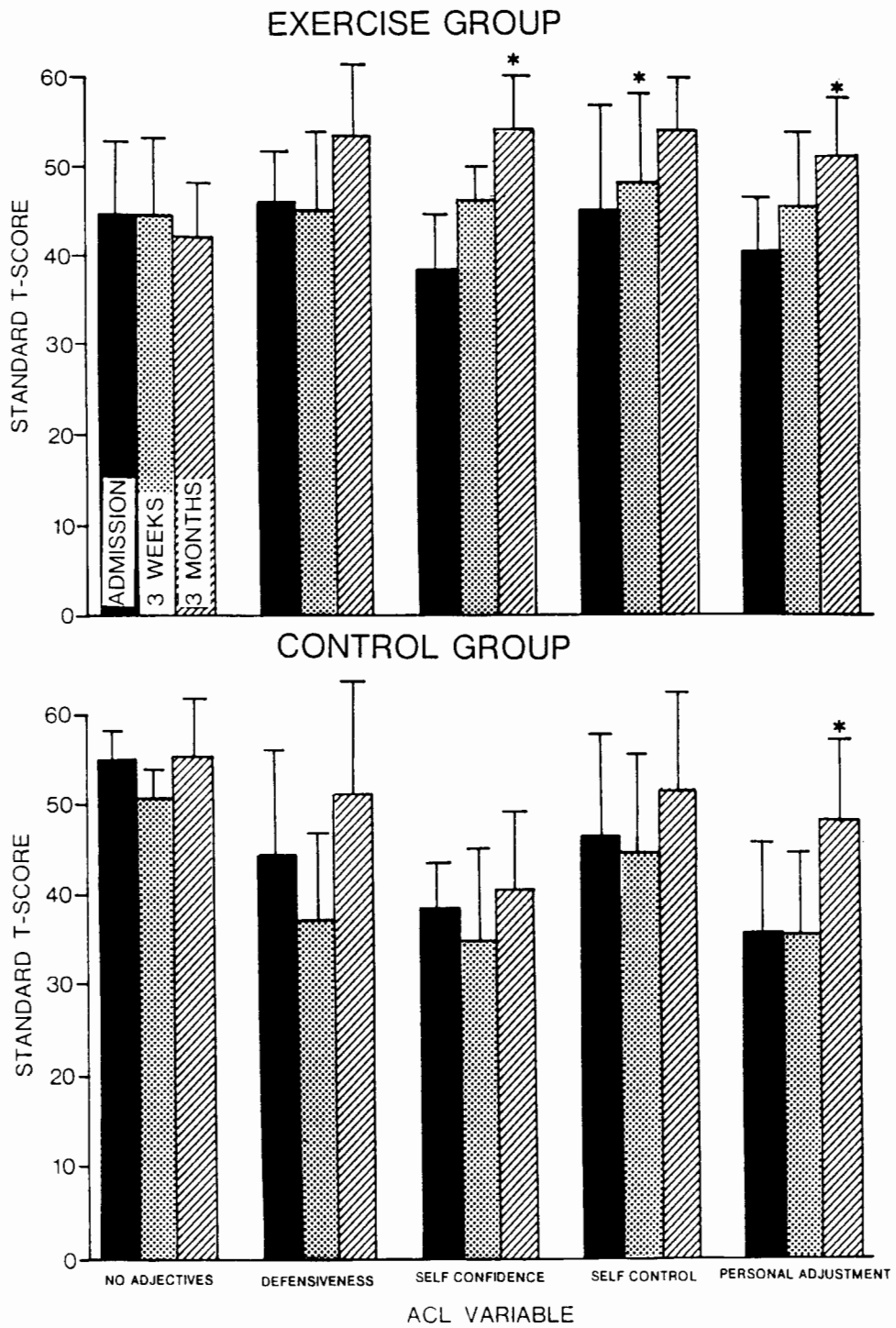
Figure 7 shows the mean standard T-scores obtained for the variables measured in the Adjective Check List at admission, after three weeks and after three months in the exercise (n=5) and control (n=7) groups respectively. An improvement in the Defensiveness, Self-confidence, Self-control and Personal Adjustment scores is evident in the exercise group after three months. The changes in the Self-confidence, Self-control and Personal Adjustment scores were statistically significant ($p < 0,05$). An improvement was also shown in the scores for Defensiveness, Self-control and Personal Adjustment in the control group after three months, however only the latter was statistically significant ($p < 0,05$). No statistical significance between groups was found but the Self-confidence score in the exercise group improved markedly in comparison with the control group.



* p < 0,05 and ** p < 0,02 for paired data (3 Weeks vs Admission)

No statistical significance between groups was found.

Figure 6. Mean standard T-scores for the Profile Of Mood States variables measured at admission, after three weeks and after three months in the exercise and control groups respectively.



* p < 0,05 for paired data (3 Weeks vs Admission)

No statistical significance between groups was found.

Figure 7. Mean standard T-scores for the Adjective Check List variables measured at admission, after three weeks and after three months in the exercise and control groups respectively.

7. BLOOD LIPID CONCENTRATIONS

Blood lipid concentrations were monitored during the course of the study to determine (a) the effect of a three week period of abstinence during the inpatient treatment programme and (b) the effect of three months of exercise training on the different blood lipid concentrations.

Table 9. Blood lipid concentrations at admission and after three weeks in the 21 subjects.

Measured Parameter	Admission	After 3 weeks
[Total cholesterol]	5,29 ± 0,8	5,62 ± 1,0 *
[Triglyceride]	1,60 ± 0,8	1,72 ± 0,8
[LDL-cholesterol]	3,05 ± 0,8	3,65 ± 1,0 **
[HDL-cholesterol]	1,41 ± 0,5	1,19 ± 0,3 **
[HDL2-cholesterol]	0,37 ± 0,3	0,27 ± 0,2 *
[HDL3-cholesterol]	1,04 ± 0,3	0,92 ± 0,2 **
Total:HDL-chol Ratio	4,07 ± 1,0	4,91 ± 1,2 **
LDL:HDL-chol Ratio	2,39 ± 0,9	3,21 ± 1,0 **
HDL2:HDL3-chol Ratio	0,34 ± 0,1	0,30 ± 0,1

Results are expressed as means and standard deviations.
Values are expressed as concentrations in mmol/l (except for ratios).
* $p < 0,01$ and ** $p < 0,005$ for paired data (3 weeks vs admission).

Table 9 shows the changes in blood lipid concentrations after three weeks of abstinence in the 21 subjects studied. Very interesting and significant changes occured after three weeks of abstinence. These include the following:

(a) total cholesterol concentration increased significantly ($p < 0,01$); (b) triglyceride concentration increased although not statistically significantly; (c) LDL-cholesterol concentration also increased and HDL-cholesterol concentration decreased significantly ($p < 0,005$), and (d) HDL2-cholesterol concentration ($p < 0,01$) and HDL3-cholesterol concentration ($p < 0,005$) both decreased. The above significant changes caused had a resultant significant increase in the cholesterol ratios ($p < 0,005$).

Table 10 lists the blood lipid concentrations in the exercise (n=5) and control (n=9) groups on admission to hospital and again after three months. There were no significant changes in any of the concentrations measured in either group after three months. However, interesting changes were evident.

The lipid concentrations measured at admission are quite similar in the two groups studied except perhaps for HDL2-cholesterol concentrations which were lower in the exercise group.

After three weeks, the changes observed in table 9 are evident in both groups. A striking exception is that the triglyeride concentration fell after three weeks in the exercise group whereas it increased in the controls. Also, the HDL-cholesterol

Table 10. Blood lipid concentrations measured at admission, after three weeks and after three months in the exercise and control groups.

Measured Parameter	Exercisegroup Admission	At 3 weeks	At 3 months
[Total cholesterol]	5,39 ± 0,7	6,21 ± 0,5	5,06 ± 1,0
[Triglyceride]	1,46 ± 0,7	1,35 ± 0,5	1,01 ± 0,4
[LDL-cholesterol]	3,49 ± 0,6	4,34 ± 0,4	3,21 ± 0,7
[HDL-cholesterol]	1,23 ± 0,3	1,26 ± 0,3	1,25 ± 0,2
[HDL2-cholesterol]	0,28 ± 0,1	0,29 ± 0,1	0,20 ± 0,1
[HDL3-cholesterol]	0,94 ± 0,2	0,97 ± 0,2	1,05 ± 0,2
Total:HDL-chol Ratio	4,53 ± 0,1	5,03 ± 0,7	4,14 ± 0,9
LDL:HDL-chol Ratio	2,97 ± 0,9	3,53 ± 0,7	2,58 ± 0,5
HDL2:HDL3-chol Ratio	0,30 ± 0,1	0,33 ± 0,2	0,19 ± 0.1
Measured Parameter	Control group Admission	At 3 weeks	At 3 months
[Total cholesterol]	5,46 ± ,07	5,82 ± 1,2	5,36 ± 1,7
[Triglyceride]	1,44 ± 0,6	1,78 ± 0,8	1,62 ± 1,0
[LDL-cholesterol]	3,11 ± 0,9	3,76 ± 1,2	3,25 ± 1,7
[HDL-cholesterol]	1,46 ± 0,6	1,25 ± 0,4	1,41 ± 0,4
[HDL2-cholesterol]	0,42 ± 0,4	0,34 ± 0,2	0,45 ± 0,3
[HDL3-cholesterol]	1,04 ± 0,3	0,92 ± 0,2	0,96 ± 0,1
Total:HDL-chol Ratio	4,04 ± 1,1	4,98 ± 1,7	4,18 ± 1,7
LDL:HDL-chol Ratio	2,37 ± 0,9	3,28 ± 1,4	2,38 ± 1,0
HDL2:HDL3-chol Ratio	0,37 ± 0,2	0,36 ± 0,1	0,45 ± 0.3

Results are expressed as means and standard deviations.

Values are expressed as concentrations in mmol/l (except for ratios).

concentration and the HDL2 subfraction remained unchanged in the exercise group whereas the both fell in the control group.

After three months, all variables seemed to have reverted to the original values measured at admission. This might suggest that the effects of alcohol abstention on blood lipid concentrations were reversed. This is particularly the case for the control group. In the exercise group, however, some subtle differences were observed.

In the exercise group, total cholesterol and triglyceride concentrations were greatly reduced but were not statistically significant. LDL-cholesterol concentration was also greatly reduced whereas HDL-cholesterol concentration remained unchanged. The HDL2 subfraction fell whereas it increased in the control group and the resultant HDL2 : HDL3 ratio was markedly lower in the exercise group compared to the control group (0,19 vs 0.45 respectively).

CHAPTER V

DISCUSSION OF RESULTS

The results of the present study show a marked improvement in abstinence after three months in the exercise group compared to the control group. The rates of abstinence for both the exercise and control groups respectively, was very similar to those shown in the study by Sinyor et al. (1982). The abstinence rate measured in the control group confirms the poor outcome of inpatient treatment as shown by Emrick (1975) and compares favourably to that of a previous study performed at the William Slater Hospital (Gilles and Keet, 1969). The very high rate of abstinence found in the exercise group is encouraging. However in view of the study by Vaillant et al. (1983), the follow-up period used in the present study as well as that of Sinyor et al.(1982), is relatively short and further research incorporating long-term follow-up is required.

The premise of the present study was that participation in an exercise programme would improve the negative psychological traits characteristically present in alcoholic patients. The results of the study show conclusively that participation in the exercise programme had a very significant effect on the psychological variables measured. As early as three weeks, significant changes in the measured psychological variables were found in the exercise group whereas no changes were evident in the control group. Increased scores were evident in each variable measured in the exercise group and those that were statistically significant were the Tension, Depression, Self-confidence and Overall Emotional Stability scores. These findings support those of Frankel and Murphy (1974) and Palmer et al. (1988).

After three months the findings were even more encouraging. Again, each score in the exercise group showed a definite change toward an increased score whereas very little change was evident in the control group. Scores that were statistically different after three months in the exercise group, were Vigour , Confusion, Overall Emotional Stability, Self-confidence, Self-control and Personal Adjustment (motivation). The only significant change observed in the control group was that of Personal adjustment. A possible criticism against the significant findings observed in the exercise group, could be the fact that drop-outs were not accounted for. Intricate statistical tests could not be used due to the small sample size used. However, the drop-out rate was not significantly different from that of controls. Hence conclusions from the significant findings should be valid.

An unexpected yet important finding in the present study with regard to psychological status, was that the standard T-score obtained for most of the variables measured was around the fifty percentile for the normal population. A possible explanation for this finding might be that the POMS test is not standardised for the population group under study. It did however, serve as a good measure of changes observed within the group over the three month period.

The physiological variables measured during a maximal exercise test before and after three months showed a definite training effect in the exercising group. The heart rate, percentage

maximal oxygen consumption and lactate concentration at each workload decreased after training indicating the classical training response in these variables. Peak workload achieved, maximal oxygen consumption and lactate turnpoint also increased significantly after three months of training. After three months training, these variables were all significantly different from the control group.

Another very significant change was the reduction in percentage body fat accompanied by an increase in lean body mass in the exercise group after three months of training. This finding is also considered to be a result of training (McArdle et al., 1981). The percentage body fat was also significantly reduced in the control group after three months, but lean body mass remained unchanged while overall body mass was also reduced. A possible explanation might be that the subjects in the control group were again becoming malnourished possibly as a result of their return to drinking.

These findings support previous studies showing that alcoholic subjects can undergo the normal physiological adaptations to exercise training (Frankel and Murphy, 19874; Tsukue and Shohoji, 1981; Sinyor et al., 1982; Palmer et al, 1988). However, the present study employed the most sophisticated measures of fitness yet used in such studies and extended the findings of these previous authors.

This study also set out to determine the state of fitness in the

alcoholic subjects and whether or not their response to a maximal exercise test was normal. Quite surprisingly, the results showed a perfectly normal response to exercise with heart rate and oxygen consumption increasing linearly in response to increasing workloads and with normal values for peak workload achieved, peak lactate concentration, lactate turnpoint and maximal oxygen consumption. No plateau in oxygen consumption was reached during the maximal exercise test, which indicates that the subject's performance was not limited by the cardiovascular limitations in oxygen delivery to muscle . More likely, they probably terminated the exercise test as a result of muscle fatigue (Noakes, 1988).

Contrary to the findings of Sinyor et al. (1982) and Pader (1983), the present study found no evidence for cardiomyopathy in the 21 subjects studied. The group also appeared to be normotensive and their resting heart rates were also normal. The subjects for the present study were not older than 40 years of age. Possibly the exceptionally high resting heart rates and blood pressure readings recorded by Sinyor at al. (1982) and Pader (1983), could be attributed to the older age of the subjects they tested.

Blood lipid concentrations changed in an interesting way with abstinence and with exercise training. After three weeks of abstinence, very significant changes were observed in most of the lipid variables measured. Total cholesterol concentration and LDL-cholesterol concentration increased and HDL-cholesterol concentration decreased, accompanied by a decrease in the HDL2

subfraction. These findings support those of LaPorte et al. (1981), Taskinen et al. (1982) and Duhamel et al. (1984) which suggest that excessive alcohol consumption alters the serum lipid profile to one associated with reduced risk of coronary artery disease and that the HDL2 subfraction is actively involved. The significant change demonstrated in the HDL2-subfraction supports the notion that this measure may be used to identify alcohol abuse (Duhamel et al., 1984). A possible variable confounding this interpretation, is the fact that the subjects tested were well fed during the three weeks of inpatient care.

No statistically significant differences were found in the exercise group compared with controls. However, certain differences although not significant suggest that exercise training over three months elicited a possibly beneficial change in the lipid profile. Triglyceride, total cholesterol and LDL-cholesterol concentrations were dramatically reduced and HDL-cholesterol concentration and HDL2 subfraction remained unchanged. These changes occurred with a resultant increase in body mass, possibly supporting the suggestion that the blood lipid changes due to exercise are not the result of weight loss (Huttenen, 1982; Nakamura et al., 1983).

It appears that exercise and alcohol consumption alter blood lipid concentrations in very similar ways. Both reduce the risk of coronary artery disease through changing blood lipid concentrations, in particular by decreasing triglyceride, LDL and total cholesterol concentrations, and increasing HDL-cholesterol

concentration particularly the HDL2 subfraction.

A major problem with the present study is that without random selection of subjects into two study groups, selective factors could confound the significant findings. This is particularly so when subjects were asked to volunteer to participate in the exercise programme. This showed up with a significant difference in maximal oxygen consumption observed between the exercise and control groups studied. However, the two groups were homogenous in all other variables measured and this isolated finding is within the statistical confidence limits accepted in the study. Peak workload achieved, heart rate and blood lactate measures were all very similar between groups. Likewise there were no significant differences in their psychological variables or their alcohol consumption prior to admission. The fact that those subjects in the exercise group had a significantly higher maximal oxygen consumption but achieved the same peak workloads during exercise, indicates only that they were less mechanically efficient during exercise (Noakes, 1988).

Another problem with the study design was the sequential recruitment of volunteer subjects in a pre/post intervention study method. This procedure was extremely time consuming and was the main reason for the small sample size used in the study. Ideally, a larger sample size is necessary in order to obtain more powerful statistical analyses. The small sample size used in the present study, therefore detracts from the significant findings.

Nevertheless, the results of the present study are such that they cannot be ignored and further research is warranted. This study would be improved with a larger sample size in which subjects are randomly allocated to either an exercise or control group. An ideal location for such a study would be a large self-help group such as Alcoholics Anonymous, where large numbers of subjects from different socio-economic classes could be recruited. The follow-up period should be extended to at least one year with the exercise programme lasting initially for three months. During this period, controls should be given an alternative activity to participate in, such as a routine of co-ordination exercises for example. This would greatly decrease the possibility of a placebo effect. Variables studied could be limited to the psychological questionnaires used in the present study and a maximal exercise test performed before and after three months and again after a year. Obviously, subjects should be homogenous with regard to the history of their drinking habits and the daily amount of alcohol consumed.

CHAPTER VI

SUMMARY AND CONCLUSION

A significant improvement in abstinence was evident in the exercise group compared to controls after three months. This finding warrants further research as the sample size was limited. Nevertheless, the observed changes were in the desired direction.

Surprisingly, the group of alcoholics studied showed a normal response to exercise as well as normal physiological adaptations to exercise training. The significant changes in physiological measures after three months of training suggest that the apparently favourable changes observed in the psychological parameters measured in the exercise group might be attributed to the exercise training programme and the enhanced physical fitness of the trained subjects. However, a placebo effect was not controlled for and the small sample size once again, detracts from the significant findings.

The increase in lean body mass and reduction of percentage body fat observed in the exercise group after three months, would also likely have improved body and self-image.

A very interesting finding was the significant psychological changes observed in the exercising group already after only three weeks of training. It was hypothesised that these changes might benefit the patient's inpatient treatment programme and consequently contribute to the improved outcome of treatment measured in the exercise group.

No evidence for cardiomyopathy, hypertension and elevated resting

heart rate in alcoholic patients was found in the present study. Changes in blood lipid concentrations with abstinence and after exercise, were inconclusive. However, findings of the present study indicate that excessive alcohol consumption might reduce risk of coronary artery disease by reducing total cholesterol concentration and elevating HDL-cholesterol concentration, particularly the HDL2 subfraction. It is suggested that malnutrition might confound these findings. The protective effect of exercise on blood lipid concentrations is also evident and in particular the HDL2 subfraction seems to play a dominant role. The present study design was not sufficiently rigorous to show any conclusive findings with regard to the blood lipid concentrations and more specific research on this topic is recommended.

The major criticism of the present study is the relatively short follow-up period used. In order to make these findings more credible, a longterm study should be conducted on a larger group of randomly selected, paired subjects.

Nevertheless, the findings of this study show a very positive benefit of exercise in the treatment of alcoholism, albeit in the short term. Should further research support these findings, exercise might provide an effective, low-cost treatment intervention accessible to large numbers of alcoholic patients.

APPENDIX A

CALCULATION TO DETERMINE PERCENTAGE BODY FAT

Body density (D) = 1,1533 - ((0,0643 X L)

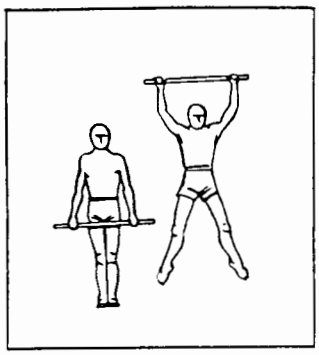
where L = log X the sum of the skinfolds (mm)

(ie. tricep, bicep, subscapular and supra-iliac measurements)

Then % body fat = 100 X (4,57/D - 4,142).

APPENDIX B

ILLUSTRATIONS OF EXERCISES



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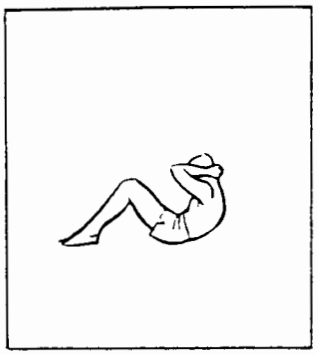

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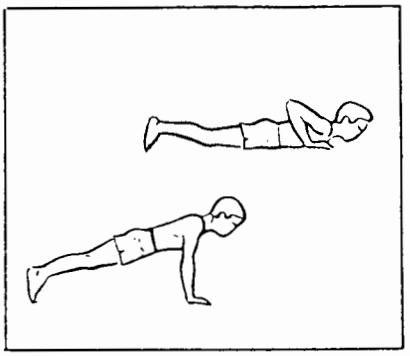

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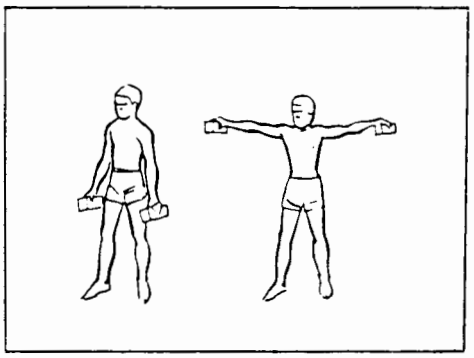
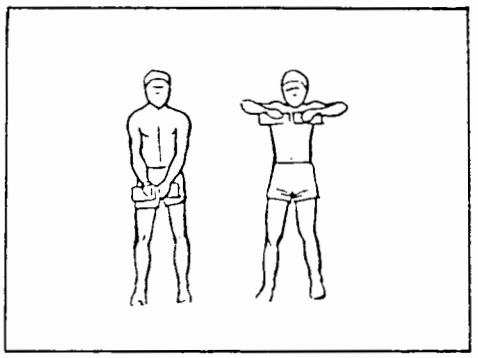
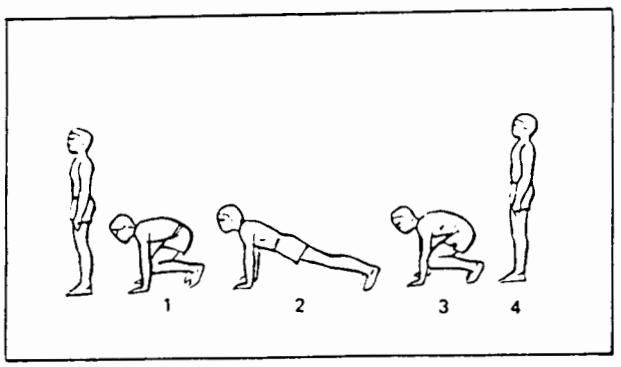


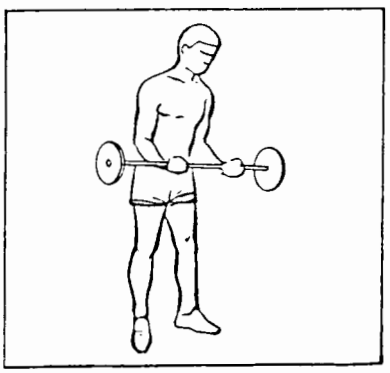
LATERAL ARM RAISES



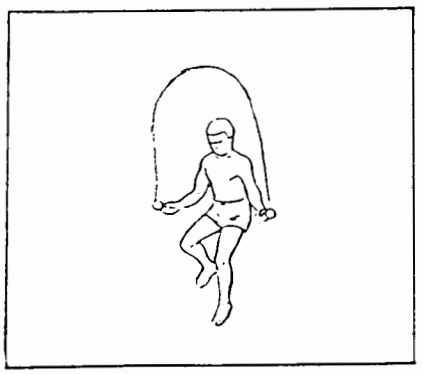
UPRIGHT ROWING



BURPEES



ARM CURLS



ROPE SKIPPING

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