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PROGRESSIVE EXERCISE THERAPY

IN

CHRONIC LUNG DISEASE

by

TUDOR HALE

A MASTER'S THESIS

Submitted in partial fulfilment of the
requirements for the award of

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Supervisor: E. J. Hamley, B. A., Ph. D.
Department of Human Sciences.

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ABSTRACT

Physiological responses to work tests before and after a programme of progressive exercise have been measured in 11 subjects aged 51 - 71 years suffering from chronic obstructive lung disease with the object of improving understanding of the effects of training on work tolerance.

The historical development of graded exercise as a therapeutic agent is reviewed, with special emphasis on the rehabilitation of patients with cardio-respiratory dysfunction, and it is concluded that insufficient attention has been paid to the influence of habituation and learning processes on work test responses and that consideration of the 'Hawthorne phenomena' is almost entirely lacking.

A protocol was designed to control these particular problems and a method for the breath by breath analysis of a work test was devised.

Thirty minutes exercise of near maximal intensity a day over a period of about twenty days led to increased working time on both treadmill and cycle ergometer: maximum heart rate fell slightly on the treadmill but was virtually unaltered on the cycle; respiratory rate and minute volumes remained basically unchanged on both exercise devices; sympton-limited oxygen uptake on the treadmill did not change and rose only slightly on the bicycle; carbon dioxide excretion increased on both machines at maximum, mean single breath and standard tidal volume levels; dead space measures were generally lower, and in the few patients who completed pre and post nitrogen washout tests resting ventilatory efficiency improved.

A 'placebo' exercise programme did not significantly affect the work tolerance of control group.

Irrespective of whether objective improvements occurred or not the majority of patients claimed that they felt better able to perform everyday activities, that activities previously avoided were now approached less fearfully and that the exercise programme was beneficial.

The findings are related to other studies and are discussed in relation to:

a) reduced anxiety and increased motivation.

b) enhanced neuromuscular coordination.

c) oxygen reserve.

d) carbon dioxide excretion

e) volume, duration and intensity of training.

The implications of the findings for future physical training programmes is discussed and suggestions are made on the work required to clarify further the role of progressive exercise in the rehabilitation of patients with chronic lung disease.

PART ONE

THE STATEMENT OF THE PROBLEM

CHAPTER ONE

The Context of the Study

Introduction

Disease of the heart and lungs are not only major causes of death in mechanised societies such as ours, but are responsible also for a great deal of chronic disability. Improved medical knowledge and care mean many more people now survive an initial episode, and consequently there is an urgent need to salvage the growing number of individuals suffering from the long-lasting after-effects of these disabling diseases. Recent evidence suggests that many cardio-respiratory cripples lead needlessly restricted lives and would benefit from a more aggressive approach to rehabilitation programmes with progressive exercise regimes being a feature of the therapeutic strategy (Laros & Swierenga 1972).

The practise of advocating exercise, both as a preventive and a therapeutic agent, is rooted in antiquity (Grimby & Hook 1971), and there is a long-held belief that some form of regular physical activity is a prerequisite for optimum physical, mental and social well-being (Fox & Skinner 1964; Fox & Haskell 1966; WHO 1968). Thus it is surprising to find the value of exercise as a palliative measure becoming a neglected feature of clinical strategy in the recent past. (Hellerstein & Ford 1957; Miller, Taylor & Jasper 1962; Hass & Cardon 1969; Petty 1975). Indeed, Groden, Semple & Shaw (1971) go as far as saying that, with the exception of orthopaedics and neurosurgery, exercise-oriented rehabilitation in this country'is an area that is almost neglected by practitioners of medicine, practically ignored by teachers and unknown to students'.

The assessment of exercise regimes for patient groups is admittedly a difficult process, but this fact should stimulate rather than deter research

in the area. It is this view, shared recently and perhaps belatedly by a committee of the Royal College of Physicians (1975), that produced the impetus for the study that follows.

Theoretical considerations

The theoretical basis on which exercise is prescribed stems from the biological principle, first proposed by Roux almost a century ago, that organs are both maintained and developed by functional stress (Mellerowicz 1966). Growing objective support is found in data from cross-sectional and longitudinal studies on ethnic groups (Anderson 1967a; 1967b; Mann 1967; Mann, Schaffer and Rich 1965), occupations (Morris, Heady, Rozzle, Roberts, and Parks 1953; Astrand & Rodahl 1970; Brunner, Manelis, Modan & Levin 1974), participants in leisure pursuits (Astrand & Saltin 1968) and a variety of patient groups suffering numerous disabilities (Rusk 1964).

The result of regular vigorous physical activity can be described, briefly and over-simply, as a greater capacity for physical work (fig. 1). This increase is dependent on complex interactions between body systems,

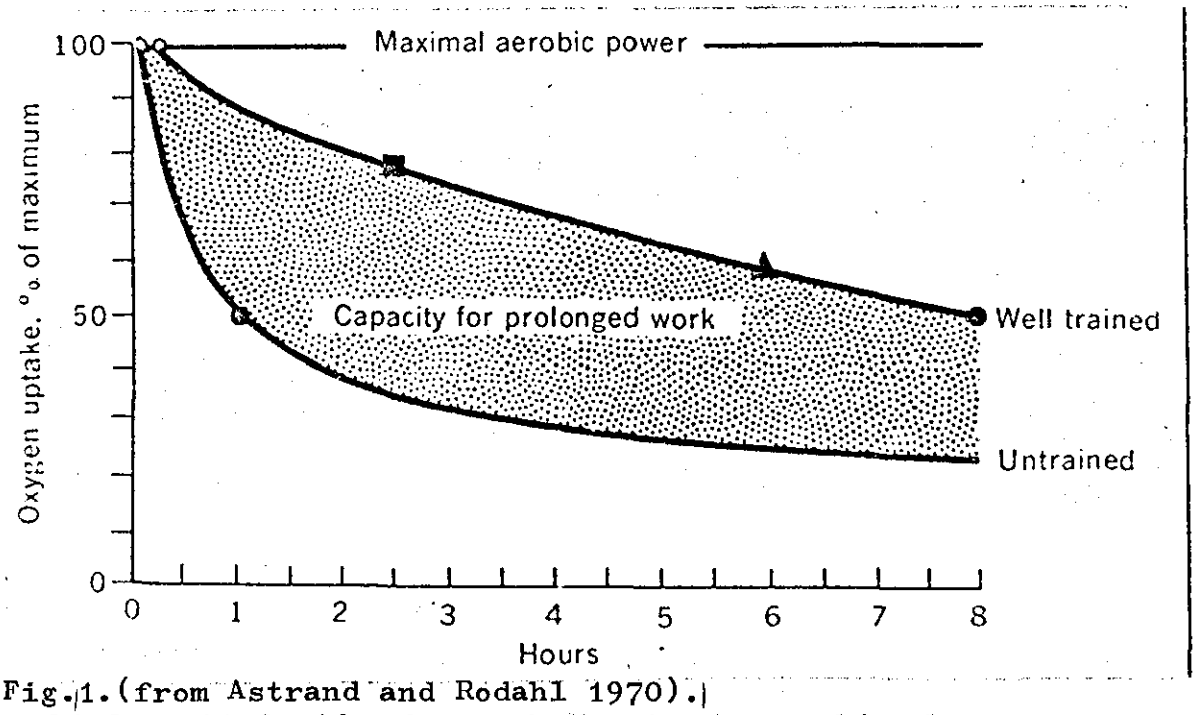


Fig. 1. (from Astrand and Rodahl 1970).

and is brought about by changes in the structure and function of several of these systems. The changes can be summarised under three main headings:

a) Skeletal muscle:

(i) hypertrophy, allowing increased force to be exerted.

(ii) enhanced cellular metabolic resources (increased adenosine triphosphate, creatine phosphate and myoglobin) and increased mitochondrial content together with enhanced oxidative mechanism within the organelle which permit aerobic metabolism at higher work rates and may increase tolerance to exhausting work.

b) Cardiovascular system:

(i) left ventricular hypertrophy, which with a reduced heart rate at rest and sub-maximal work and the subsequent enhanced venous return, produces greater stroke volume, increased cardiac output at maximal work and a more efficient pump.

(ii) enhanced vaso-control whereby blood volume is distributed more effectively during heavy work, and combined with changes in skeletal muscle, results in greater a-$\bar{v}$ 0_2 differences during maximal work.

c) Respiratory system.

(i) lung function measures at the upper end of the normal range.

(ii) improved lung diffusing capacity during rest and work.

(iii) efficient respiration at rest and during work.

The long term significance of these benefits is not yet clear, but in the opinion of Davies, Drysdale and Passmore (1963) exercise is instrumental in promoting health, and many authors argue that reduced physical activity is a major risk factor in ischaemic heart disease (Fox et al 1964; Brunner et al 1974), although the evidence for both assertions is by no means conclusive. In the short term however, cardio-respiratory performance- denoted by aerobic capacity (Taylor, Buskirk & Henschel, 1955; Mitchell, Sproule & Chapman, 1958) is clearly enhanced by regular exercise and results in an increased capacity for prolonged work.

Applicability to patient groups

If Roux's principle is appropriate to the treatment of these people with disabling cardiorespiratory disease, any increase in work capacity could constitute an important opportunity to engage in a wider range of activities thereby improving the quality of life that remains.

It is this concept of improving the quality of life that lies at the heart of the study, for exercise oriented rehabilitation will certainly not reverse permanent structural damage of the type seen in chronic pulmonary disease for example. The purpose is to enable the patient to utilise his limited cardio-respiratory reserve to the full and live a life commensurate with his physical and mental capacity (WHO 1964); the objective is to add life to years rather than years to life.

In spite of growing evidence that many patients suffering the residue of cardio-respiratory disease lead needlessly restricted lives, unnecessarily cautious treatment is still prominent however (Fisher 1969; Groden et al 1971). Rest is prescribed followed by advice to avoid strenuous exercise; the patient, now fearful, interprets the advice as an injunction against all activity and an already reduced cardio-pulmonary reserve becomes progressively diminished; even minimal levels of exertion become increasingly beyond reach and the cardio-respiratory cripple is produced (Laros & Swierenga 1972). In addition, an erroneous notion on the part of the public that physical activity is invariably harmful has not been adequately dispelled by the medical profession (Hellerstein 1959, Groden et al 1971), and has led to invalidism is some patients and a delay in successful rehabilitation in others. (Kellerman, Levy, Feldman & Kariv, 1967).

The rewards of regular exercise are not inconsequential, particularly for those people who have developed a neurotic fear of even minimal exertion, and a planned supervised programme of progressive exercise can produce

physiological, psychological and sociological benefits for many individuals at present living under the sentence of self-imposed inactivity.

The purpose of the study

As stated previously it is generally acknowledged that exercise therapy is difficult to evaluate. So, in spite of persuasive, albeit circumstantial, evidence from many sources, Kavanagh & Shephard (1973) feel satisfactorily controlled experiments with patients have yet to be completed. This study, by including more rigidly controlled conditions, sets out to examine the effects of a short period of physical training on the physiological responses to progressive work tests of a group of men with cardio-respiratory disease and low work tolerance.

Three features distinguish this study from others in the area of cardio-respiratory dysfunction.

a) The people studied are patients suffering from chronic obstructive lung disease, the management of which presents difficult and discouraging problems. Medical care is often not sought until marked ventilatory impairment and significant disability occurs when structural damage to the lungs has advanced to the point where cardio-pulmonary reserve can barely meet physiologic demand. Symptomatic relief can be achieved through bronchodilators, antibiotics and tranquilisers; physiotherapy is commonly advocated, although some workers feel that physical therapy has no established scientific basis and evidence of lasting clinical benefit is lacking. The patient has a prognosis of a shorter life span and it is often felt that improvements are purely psychological, due to tender loving care and at best are transient. It is hardly surprising that rehabilitation of these patients is lagging behind even that of heart disease patients. But is is worth noting the comments by Lefevre & Paterson (1973), in an otherwise dismal editorial; 'exercise training appears to result in definite subjective improvement in

exercise tolerance and in objective indirect improvement', and 'current evidence suggests that training is a useful adjunct to therapy.' Maybe with supportive drug treatment exercise therapy is the only real treatment with good prognosis.

b) An attempt is made to distinguish between general and specific training effects by testing patients on two devices - treadmill and bicycle - following supervised training on the treadmill.

c) The control group is given a training programme of minimal activity in an effort to distinguish between improvements brought about by close attention plus intensive activity and those due largely to greater attention.

Few studies have considered the Hawthorn effect. This phenomenon, demonstrated in an industrial setting in the early part of this century, indicated that workers responded positively to any change in working conditions suggesting that they were susceptible to management expectations. It is possible that patients given a minimal dose of exercise might demonstrate an increase in work capacity merely because it satisfied supervisor's, and their own expectations, and not through changes in physiological function.

CHAPTER TWO

The Historical Review

Introduction

The practical application of Roux's principle to the sick is reviewed in this chapter, and the survey shows:

a) changes in the pattern of disease during this century, with the number of people suffering from cardio-respiratory disease growing thereby making studies into rehabilitation programmes an urgent requirement;

b) exercise as a therapeutic tool is not new, and the present study seeks to refine methods to gain insight into their validity rather than revolutionise established practice;

c) prolonged inactivity produces undesirable sequelae that may be halted or even reversed in certain cases by exercise oriented rehabilitation;

d) exercise therapy is based on sound physiological principles;

e) exercise programmes, suitably modified for ischaemic heart disease patients, produce beneficial effects, and recently have gained acceptance in the U. K. ;

f) exercise studies on patients with chronic obstructive lung disease are less conclusive and a great deal more work needs to be undertaken with this group.

The need for rehabilitation programmes

The improved quality and greater availability of medical care during the past fifty years or so have produced a marked change in life expectancy. The pattern of disaese has changed (Table 1) so that infant

mortality is reduced, and survival curves have changed progressively from the theoretical logarithmic form of 'death independent of age', to the rectangular form of 'death due to senescence' (figure 2). Stone age man rarely exceeded 25 years, (Young 1971), but the average length of life has increased from 49 years in 1900 to close to 70 years today (figure 3).

CAUSE OF DEATH	1900	1966
Heart Disease	2350	4327
Cancer	829	2279
Typhoid	173	0
Scarlet Fever	117	0
Measles	391	2
Tuberculosis	1902	49

TABLE 1 Cause of death in England and Wales
(all ages, per million population)

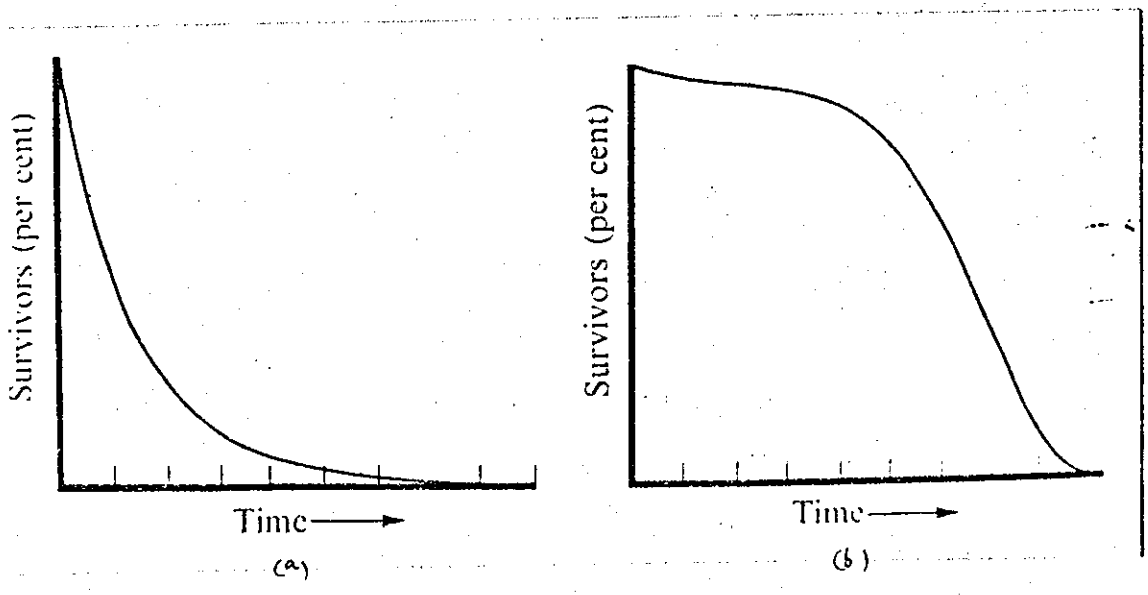


FIGURE 2. a) Survival curve at a constant rate of mortality (50% per unit of time).

b) Survival curve of a population that shows senescence. (Young, 1971).

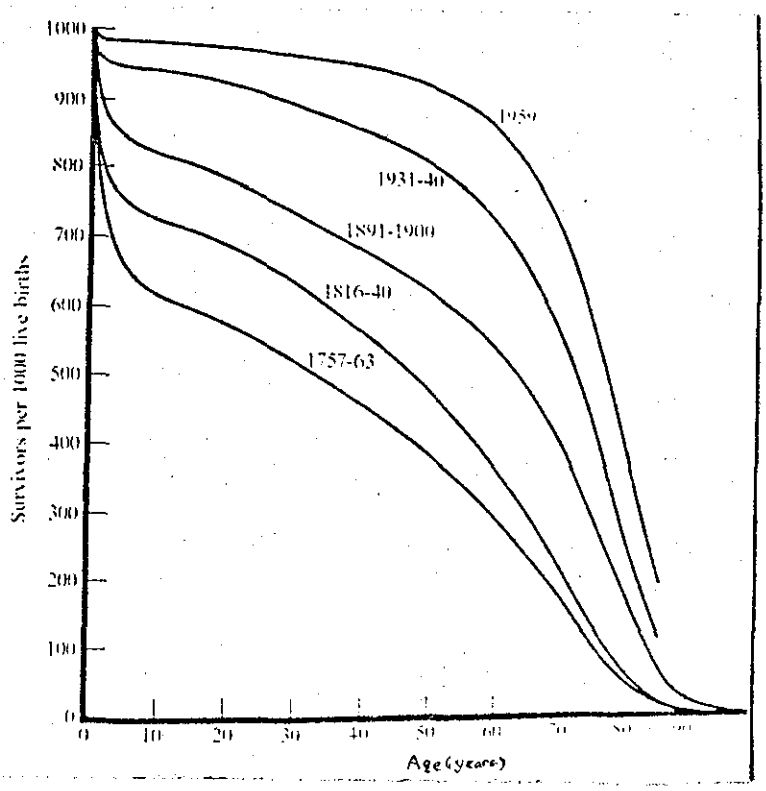


FIGURE 3. The change in shape of the survival curve from the mid-18th Century to the present day - Swedish data (Young 1971).

But man's physiology may be, as yet, poorly equipped to deal with longevity. Modern man has retained many of the biological characteristics of his remote ancestors, and the genes that governed Paleolithic man's physically demanding struggle for survival still regulate man's response to today's highly automated society, in which individuals not only live longer but also are becoming increasingly sedentary (Durnin 1967). Thus, according to Dubos (1968) 'the challenges of today have to be met with biological equipment that is largely anachronistic', and the increasing incidence of cardio-respiratory disease in the United Kingdom - 89,000 hospital admissions in 1957 rising to 178,000 by 1967 whilst total admissions rose 25% - may be a consequence of inadequate ageing physiological equipment.

The increasing incidence of cardio-respiratory disease combined with a high discharge rate of hospitalised patients - 75% of IHD patients, 90. 0% of cold patients (DHSS 1970) - means there are growing numbers of people suffering the residue of chronic disabling disease. According to Benton and Rusk (1953) these people presented a grave medical and social problem over twenty years ago, and the problem is likely to increase rather than diminish. The statistics underline the pressing need to provide appropriate rehabilitation programmes where the aim is not only the restoration of patients to previous capacity but also the optimisation of physical and mental function (Hellerstein 1967; Hass et al. 1969; Laros & Swierenga 1973). Implicit in this aim there is, for many workers, a clear commitment to include progressive exercise therapy in the overall approach to the problem, and the remainder of this review investigates the foundation on which that commitment is based.

The history of exercise therapy

The value of exercise as a part of clinical strategy has been known empirically for some time. Grimby et al (1971) reveal that exercise as a therapeutic measure was recommended by medical practitioners of Ancient China, Egypt and Greece, and in 1553 Mendez produced his 'Book of Bodily Exercise' (Miller et al 1962) extolling the virtues of exercise in medical treatment. Heberden noted in 1772, in probably the first published observation of a beneficial effect of physical activity in coronary artery disease, that one of his patients was 'nearly cured' after a 6 month period of sawing wood daily for 30 minutes, and shortly afterwards (1799) Parry discussed the benefits of moderate exercise in patients with angina (Frick 1968). Medical gymnastics featured in the programme of the Central Gymnastic Institute in Stockholm created in 1813 by P. H. Ling. Stokes introduced graded hill walking for his patients in the mid 19th Century; he said 'the patient must adopt early hours and pursue a system of graduated muscular exercises, and it will often happen that after perseverance in this system the patient will be enabled to take an amount of exercise which at first was totally impossible owing to the difficulty of breathing which followed exertion' (Grimby et al 1971). Hill walking was generally adopted in Sweden towards the end of the 19th Century and the idea transferred to England at King Edward VII Hospital in Midhurst when it was opened as a sanatorium in 1905. A map of graded walks among the South Downs can be found in the entrance hall today.

Exercise as an adjunct to physical therapy was a common feature in the clinical treatment of asthma, emphysema and other chronic pulmonary disease in the 1930's, particularly on the continent, but the treatment in Britain seemed to be biased towards improving breathing technique rather than restoring work

capacity. At this stage exercise prescription was a subjective affair but by 1945 an attempt had been made to quantify therapy. De Lorme (1945) restored skeletal muscle function, diminished by disease, through setting specific goals in terms of a weight to be lifted and the number of times it should be lifted at any one session. New goals were set at appropriate times so that training became controlled and progressive. Remedial gymnastics is now well established in physiotherapy departments throughout the United Kingdom.

In 1948 a graded activity programme was described for a safe return to self-care for heart disease patients (Newman, Andrews, Koblish & Baker, 1948). This was one of the first reports of the beneficial effect of progressige exercise therapy in ischaemic heart disease and led the way for numerous studies perticularly those headed by Hellestein and his co-workers (1957, 1963, 1968). In 1964 the effects of exercise on patients with chronic lung disease is described (Pierce et al, 1964) but unlike IHD, only a handful of follow-up studies have been attempted in the intervening 10 years. These studies will be dealt with later.

The physiological basis for exercise therapy

This can be presented in two ways. Firstly there is a negative view which pinpoints the physiologically undesirable effects of inactivity. These were described over thirty years ago (Harrison 1944; Dock 1945) at a time when bed rest as a therapeutic measure was being abused (Keys 1945). Keys felt that the most frequent cause of deconditioning, even in disease, was simply lack of activity. Dock describes the evil sequelae of extended bed rest as follows:

a) phlebothrombosis in the veins of the lower extremities and pelvis.

b) pulmonary oedema, pulmonary embolism, and hypostatic

pneumonia (conditions which add further burdens to cold sufferers).

c) decreased bowel activity leading to straining and possible damage to the heart.

d) increased cardiac output ($\dot{Q}$) through increased rate (f_H) leading to greater myocardial oxygen consumption ($M\dot{v}_{O2}$) and further embarrassment to IHD patients with already diminished cardiac reserve.

But the presence of illness is not a pre-requisite for loss in function. Reduced exercise tolerance and metabolic disturbances (increased excretion of Ca^{++} and P, and an accumulating negative nitrogen balance) were observed in 4 healthy young men undergoing 6 - 7 weeks immobilisation (Dietrick, Whedon & Shorr 1948) and were supported by a similar study of that period (Taylor, Henschel, Brozek & Keys 1949). More recently, well controlled studies have reported large scale reductions in cardio-respiratory performance following quite short periods of bed rest (20 days) (Saltin, Mitchell, Blomquist, Johnson, Wildenthal & Chapman, 1968; Giese 1969; Blomquist & Mitchell 1971). In the Saltin study aerobic capacity ($\dot{V}o_{2max}$) fell from 3.3 to 2.4 L. min; $\dot{Q}$ during upright exercise at sub-maximal loads fell 15%; mean stroke volume Sv fell 30%; f_H at 600 Kpm. min. increased from 129 to 154 beats, and f_H at $\dot{V}o_2$ 2.0 l. min rose from 145 to 180 b. min. Metabolic changes have been observed after only a few days of weightlessness (Grimby et al 1971), and even the cessation of training results in the disappearance of improved function, beginning after only fourteen days (Roskamm 1967).

Secondly there is the positive view which underlines the advantages that accrue from organised training programmes. The benefits affecting normal people have been described earlier, (page 5), therefore only the responses of patients with CRD are presented here. For ease of presentation IHD and COLD patients are considered separately.

Exercise therapy in ischaemic heart disease (IHD)

Compared with normal people IHD patients tend, at rest, to have reduced SV, lower $\dot{Q}$, greater myocardial a-$\bar{v}O_2$ difference and higher pulmonary and systemic vascular resistances. During exercise these tendencies become more strongly confirmed as the following physiological deficiencies; lower SV, lower $\dot{Q}$, higher f_H at standard work and at a given $\dot{V}o_2$, and higher mean systolic, right ventricular end diastolic and pulmonary artery pressures. Additionally the coronary reserve is seriously reduced. (Case et al, 1955): Gorlin et al 1960; Harvey et al 1962: Malmcrona et al 1963; Malmborg 1965). This reserve has been variously described as:

a) the range within which an adequate myocardial O_2 supply is guaranteed and characterised by the ratio of O_2 supply and demand ($\geq$ 1, supply is adequate, $<$ 1 = ischaemia) (Schimert et al 1966).

b) the difference between the variable as it exists and the maximum up to which increased performance of the heart can be expected and beyond which decreased performance ensues (Katz 1960), and

c) the range between coronary flow during basal conditions and the increased flow required during maximum tolerated work (Freidberg 1966).

The two main reasons for reduction are:

a) pathologically produced vascular obstructions and increased local and peripheral resistances that place an ever-present additional load on the heart, even at rest (Gorlin 1962 & 1963; Katz 1960) and

b) the reduced limits of beneficial compensatory mechanisms of dilatation, hypertrophym increased contractile power and tachycardia (Burton 1959; Freidberg 1966; Gilbert et al 1965; Katz 1960).

Progressive exercise therapy is designed to reduce the oxygen demand of the heart at rest and particularly during exertion, thereby safeguarding the coronary reserve of patients and reducing the incidence of myocardial ischaemia. This is achieved, it is argued, in two possible ways, singly or in combination. First, structural changes within the myocardium which lead to increased coronary flow; second, a more efficient use of the reserve through the altered behaviour of the cardio-vascular system.

The first mechanism is presented schematically in figure 4.

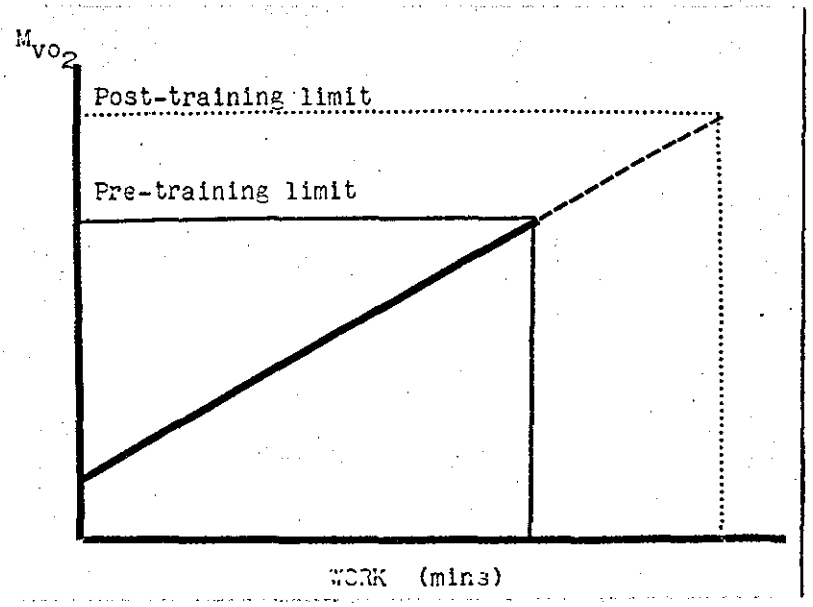


Figure 4. Schematic representation of increased coronary reserve via enhanced collateral circulation.

It is based very largely on animal experiments and the extrapolation to the human is therefore highly speculative; even so some workers find the theory attractive (Kaufman et al 1966; Mann 1967; Stothart et al 1971). It is known from autopsy and angiographic studies that the presence of anoxic conditions within the myocardium - e.g. anaemia, narrow or occluded arteries - produces collateral circulation in both man and animal (Zoll et al 1956; Eckstein 1957; Blumgart et al 1968); the extent

of the anastomoses is closely related to the degree of stenosis (Heinle et al 1968). Following ligation of coronary arteries in experimental animals mild collateral circulation developed; when exercise was administered over a period of time a markedly more extensive development of collateral circulation was observed (Eckstein 1957; Hass et al 1959; Bloor et al 1965; Ravio et al 1968). Some workers feel that such development can be an important factor in survival rates following extensive coronary artery occlusion (Blumgart et al 1968; Ravio et al 1968), and that these collateral vessels, at times, protect the myocardium from serious damage. But the functional significance of increased collateral circulation in man remains obscure (Varnauskas 1971). Certainly coronary blood flow adjustments are crucial in any increase in the heart's performance (Lombardo et al 1956; Case et al 1955, 1963; Horvath 1959; Katz 1960; Messer et al 1962; Gorlin 1971) but other factors may be involved. Raab (1966) found, for example that at markedly reduced coronary flow ECG changes suggestive of ischaemia were absent until catecholamine content of the circulating blood was increased. Furthermore the presence of collateral vessels does not necessarily protect against angina or infarct (Katz 1967; Scott 1967; Heinle et al 1968) and post-training angiographic observations remain equivocal (Kaufmann et al 1966).

More efficient use of the myocardial reserve through post-training changes in cardio-respiratory function seems a more profitable line of enquiry. Several factors contribute to the overall oxygen demand of the heart (figure 5), but it appears that Mv_{02} is set not so much by the work done, as by the manner in which the work is accomplished (Katz 1955;

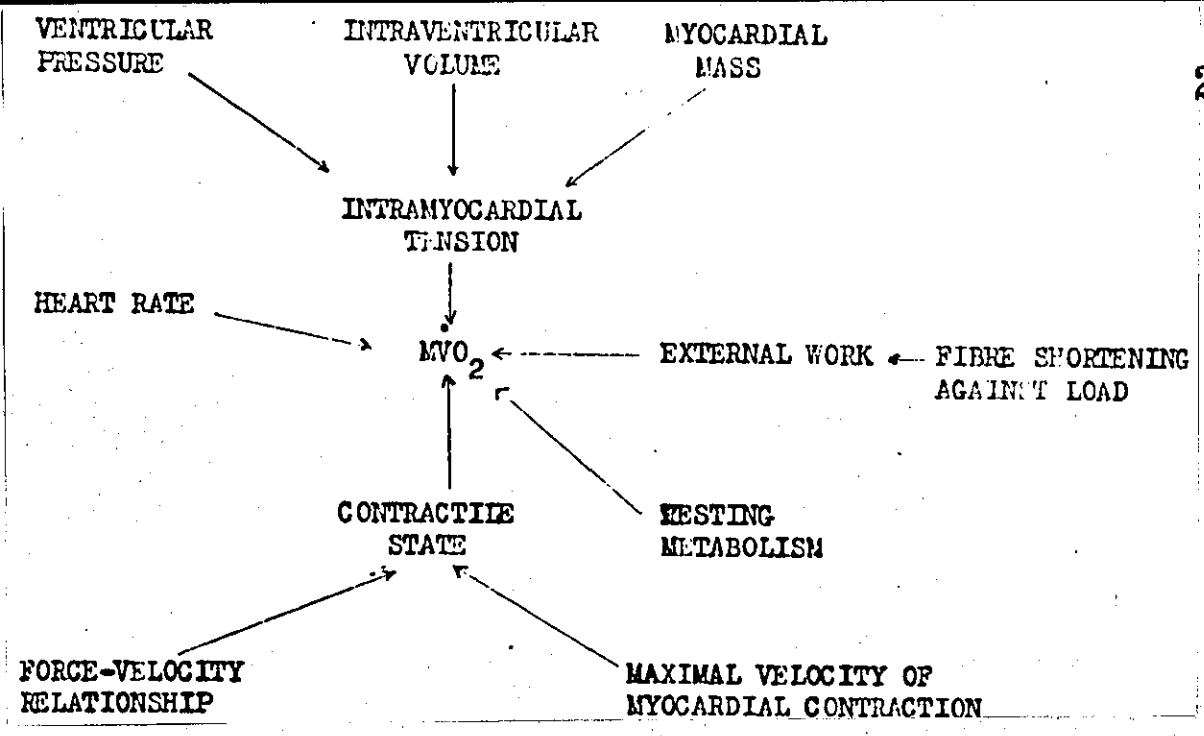


Figure 5. The multi-factorial nature of myocardial Vo2.

Braunwald et al 1958; Sarnoff et al 1958; Burton 1958; Case et al 1963; Schimert et al 1966; Scott 1967; Wang 1969). Improved myocardial efficiency is achieved largely through the agency of relative bradycardia of training, combined with changes in left ventricular power and circulation of blood at the periphery. This is represented schematically in figure 5.

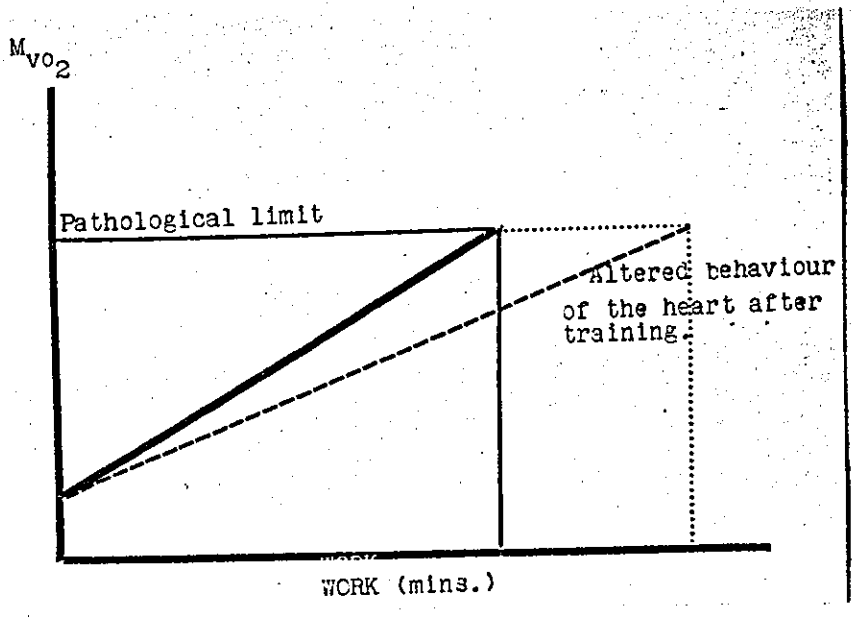


Figure 6. Schematic representation of improved myocardial efficiency after training.

The chief value of bradycardia is metabolic economy (Hall 1963: Katz 1967; Mayhew 1971). Tachycardia is the primary source of increased $\dot{Q}$ during exercise. The resultant increase in Mv_{O_2} is greater at high than

at low f_H for any given left ventricular work (Mitchell 1963; Raab et al 1962); a tripling of cardiac work at constant f_H leads to a 33% increase in Mv_{O_2} whereas tripling f_H at constant work results in Mv_{O_2} increases in the order of 75% (Schimert et al 1966). In addition, although training does not necessarily lessen the magnitude of the pressure generated in the ventricle per beat, bradycardia results in fewer pressure generations per minute and reduces Mv_{O_2} (Frick 1968), and the longer diastole afforded by bradycardia provides more time for the predominantly diastolic coronary blood flow (Jokl et al 1966: Mellerowicz 1966; Raab 1969) this enhancing oxygen transport to the tissue.

The mechanisms by which the relative bradycardia of training occurs are not yet clear, but intra- and extra- cardiac factors are involved. the intra-cardiac influences may be:

a) slowing the atrial pacemaker by increased cholinergic discharge by atrial tissue and/or vagal discharge (Hall, 1963).

b) marked vagal preponderance at rest and low sympathetic drive during exercise (Denison et al 1958: Hall 1963; Jokl et al 1966; Raab 1962).

c) reflex deceleration resulting from presso-receptor activity produced by the greater pulse pressure of increased stroke volume (Raab et al 1962; Frick et al 1968; Clausen et al 1969).

Extra-cardiac influences:

a) hypertrophy of skeletal muscle leading to decreased central nervous discharge and reduced cardio-accelerator activity (Hall 1963).

b) increased relative blood volumes leading to lower sympathetic drive (Raab 1962; Hall 1962).

c) improved peripheral circulation leading to improved cellular oxygenation, reduced anaerobic metabolite accumulation andthus lower

chemoreceptor activity (Varnauskas et al 1966; Holloszy 1967; Clausen et al 1969 & 1971).

As previously stated evidence to support the 'collateral circulation' theory is lacking, but the view that adaptation within the cardiovascular and respiratory systems improve coronary reserve is endorsed by post training evaluation. Objective measures of patient responses to work tolerance tests indicate wide ranging improvements in physiological function and these will now be discussed. For ease of presentation the findings will be divided into four sections.

a) Haemodynamic measures

Two schools of thought have emerged regarding the effect of training on haemodynamic measures. The first group argue that the central pump is the major site of change. Frick et al (1971) compared haemodynamic responses to a single level work test in two groups of patients - an exercising group and a control group. No significant changes occurred in the latter group but in the experimental group f_{H_W} fell 7.5% ($P < 0.02$), SV rose 10% ($P < 0.05$), $\dot{Q}$ rose 2.2% and a-$\bar{v}0_2$ difference fell 3.1%. In a previous study Frick et al (1968) noted changes at two levels of exercise. At the first level f_{H_W} was 13.2% lower ($P < 0.01$), sv was 9.7% higher ($P < 0.05$), $\dot{Q}$ and a-$\bar{v}$ 0_2 difference remaining unchanged; at the second level f_{H_W} was 8.3% lower ($P < 0.02$), sv rose 13% ($P < 0.05$), $\dot{Q}$ rose 7.5%, a-$\bar{v}$ 0_2 difference fell 8.4%. The second group hold that alteration in the peripheral circulatory regulation is the major training effect. Clausen et al (1969) report an 8.0% reduction in f_{H_W}, 14.2% increase in sv, 0.4% lowering of $\dot{Q}$, and a 44.7% reduction in blood lactate.

Varnauskas & Bergman (1966) observed reduced f_{H_W} ranging between 3.7 and 5.9% but sv was also reduced (range 10.6 - 13.4%) producing a fall in cardiac output; however a-$\bar{v}$ 0_2 difference rose (range 11.0 - 16.2%), and left ventricular work was reduced (17.8 - 21.4%) through a slight fall in brachial artery pressure.

b. Cardiovascular function (CVF)

Indirect measurement of cardiovascular function also show adaptive changes. Reductions in f_H at rest, during sub-maximal and maximal work, and during recovery are commonly reported (Rosenbaum et al 1959; Hellerstein etaal 1963 & 1965; Naughton et al 1964 & 1966; Sloman et al 1965; Pedersen 1971). E.C.G. changes during work have occurred viz. augmented R and T waves, T wave amplitude increase at lowered f_H, and reduced S-T segment depression (Cain et al 1961; Mazzarella et al 1965; Salzmann 1969; Mayhew 1971). Amplitude changes however, S-T segment excepted, are less likely to indicate improved function than changes in the time intervals between the P-Q-R-S-T deflections (Thomason et al 1968; Thomason 1972).

Reduced blood pressure (B_p)has been observed at rest and during work (Naughton et al 1964; Raab 1969; Boyer et al 1970). Lowered Mv_{o_2} is claimed on the basis of reduced systolic-time index (STTI) - derived from the product of f_H and systolic-pressure - (Hellerstein et al 1963, 1965 & 1968; Frick et al 1968; Clausen et al 1969), but this indirect measure is rather crude (Katz 1960) and may not be valid since it is the development of tension that is important rather than a time factor (Sonnenblick et al 1968).

c) Respiratory function (RF)

Changes in RF can be considered from two angles; firstly changes in lung volumes and capacities, and secondly the overall effect on oxygen transport. Increases in VC (Hellerstein et al 1963; Frick et al 1968) and MVV (Hellerstein et al 1963) and reduced minute volume during work ($\dot{V}_E$) (Naughton et al 1964; Varnauskas et al 1966, Clausen et al 1969) have been reported, but may be the result of increased motivation and habituation to equipment rather than responses to training. Improved oxygen transport has been claimed (Kellerman et al 1967) on the basis of increased 0_2 extraction (Varnauskas et al 1966), greater oxygen pulse (Gottheiner 1966 & 1968) reduction in RQ at standard work (Clausen et al 1969) and increased $\dot{V}0_2$ max (Mazzarella et al 1965; Kavanagh et al 1970). The first three measures could have resulted from habituation also; the first and third through reduction in f_R, the second by lower f_H through reduced anxiety. Improved $\dot{V}0_2$max is the most useful indicator of enhanced oxygen transport, but even so the changes are small (2.3 - 8.6 ml. Kg. min) (Mazzarella et al. 1965), and the 20% increase reported by Kavanagh et al (1970) is in predicted $\dot{V}0_2$ max. the reliability of which is open to some doubt.

d) Physical work capacity (PWC)

The integration of all the adaptive mechanisms is manifest in an enhanced ability to work. An increase of 34% in maximal work capacity (Clausen et al 1969), of 25% in work at f_H^{150} (Hellerstein et al 1968), and unspecified improvements in work achieved have been reported (Rechnitzer et al 1965; Sloman et al 1965; Sorour et al 1969). But the importance of work capacity per se is probably exaggerated;

PWC max. is susceptible to psychological factors and a five-fold increase reported in one study (Kattus et al 1965) may owe more to reduced anxiety than improved physiology. Even work at a given f_H (usually 150 b. min) is not entirely free from suspicion (Davies et al 1968).

From the patients' point of view however, which mechanism is at work matters little, because the end product of exercise oriented rehabilitation is, for many, a reduced myocardial oxygen demand during exercise, greater exercise tolerance before coronary reserve is compromised and greatly increased confidence and enhanced life-style.

Exercise therapy in chronic obstructive lung disease (COLD)

Claims are also made that exercise programmes are beneficial for patients with chronic pulmonary disease. The evidence on which these claims is based is far from compelling however. Considerably less attention has been paid to exercise therapy in lung disorders than in IHD for example - few than thirty investigations were cited in a recent review (de Coster, Sergysels & Degre 1972) and, it is argued later, many of the studies contained defects in experimental design. To obtain a clearer picture of the problems involved in progressive exercise therapy in COLD a brief resume of the sociological and physiological implications of the disorder precedes the review of work already accomplished.

The two disorders which concern this thesis are chronic bronchitis and emphysema, conditions which are characterised by chronic diffuse irreversible airways obstruction (American Thoracic

Society Committee 1962). The former is defined as a hypersecretion of bronchial mucous, usually accompanied by recurrent productive cough, the latter as an anatomical enlargement of the lungs with abnormal enlargement of the distal air spaces accompanied by destructive changes in the alveolar wall (Thurlbeck - personal communication), Since the conditions are often indistinguishable clinically the term chronic obstructive lung disease (COLD) is used to cover the whole clinical spectrum.

The disorder is a leading cause of death and disability in the United States and the United Kingdom. Reports from the United States during the period 1959 - 1964 state that first visits to physicians increased 189% for bronchitis with emphysema, 81% for chronic bronchitis, and 195% for emphysema (Hass et al 1969; Bass et al 1970), and that in 1970, 265,000 Americans were hospitalised for the treatment of COLD and 100 million dollars were paid in disability benefits (Addington et al 1974). In 1968 the system for recording 'causes for hospitalisation in the U.K.' was changed and therefore figures are less clear cut. However, in 1957 an estimated 47,800 people were hospitalised for bronchitis, reaching a peak of 86,270 in 1063, remaining at approximately 75,000 until 1967. The figures for 1968 show that all forms of bronchitis, together with emphysema, was responsible for over 76,000 hospital admissions, with an average stay of 25.9 days - longer than for acute infarct (20.1 days), and used an estimated 1970 beds daily (DHSS 1971); a death rate of only 10% reveals the number of patients who are faced with some residual disability and who might benefit from exercise oriented rehabilitation.

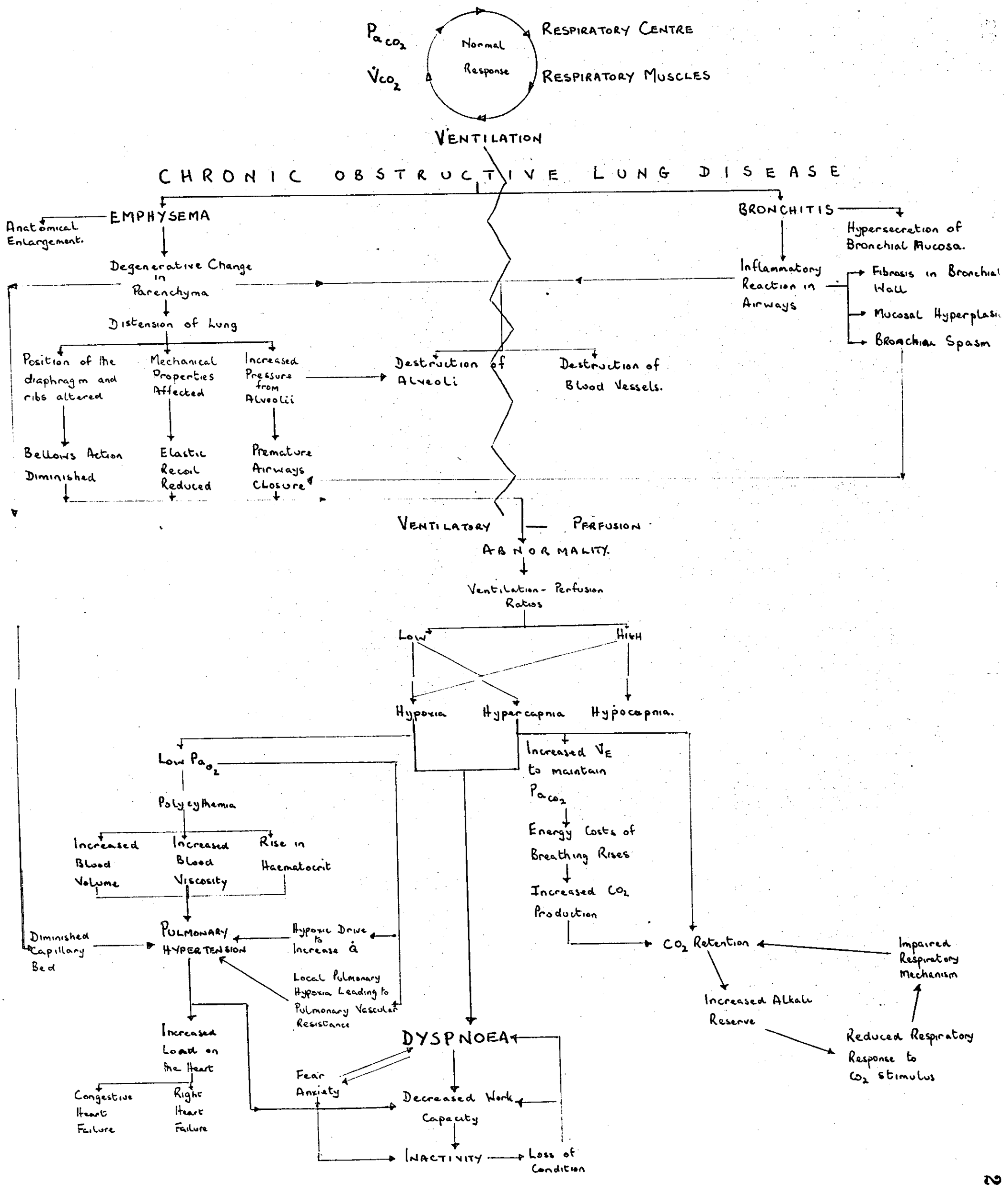


Figure 7. The inter-active nature of COLD.

The disease impairs pulmonary function in two ways;

a) usually there is a reduced ability to breathe; the lung becomes resistant to ventilatory movements by scattered uneven increase in resistance and decreased compliance; ventilation/perfusion disturbances occur and more power is needed to provide adequate ventilation.

b) the ability to arterialise venous blood is reduced and hypoxia results; depending on the state of the disease hypercarbia and hyperacidity may also occur. The interactive nature of these impairments and the consequences are illustrated in figure 7.

The twin physiological disturbances of increased work of breathing together with oxygen lack combine to produce dyspnoea on exertion. In this context dyspnoea is defined as 'the unpleasant awareness of the necessity to breathe, either at rest or at a grade of exertion not causing this awareness in normals' (Laros et al 1972), and is one of the most disabling features of COLD: Impaired exercise tolerance results, and according to numerous sources dyspnoea is a major factor in necessitating reduced activities of daily living.

Strenuous efforts have been made to relieve the underlying causes of dyspnoea (Miller 1952, 1967), the methods being grouped under two main headings:

a) Pharmacologically oriented treatment.

(i) Drug therapy - antibiotics, expectorants, bronchodilators (this area will not be discussed further).

(ii) Low-flow oxygen therapy.

b) Physically oriented treatment.

(i) Physiotherapy - breathing exercises, relaxation exercises, postural drainage.

(ii) Artificial aids - abdominal belts, intermittent positive pressure breathing deviees (IPPB).

Oxygen therapy can be an effective means of improving exercise tolerance in certain patients. Presumably Po_2 in both alveolar gas and the tissues is raised and the chemoreceptor drive to respiration is reduced; increased exercise tolerance is dependent on concentration of oxygen in the inspired air (F_{Io_2}) and the flow rate (figure 8.).

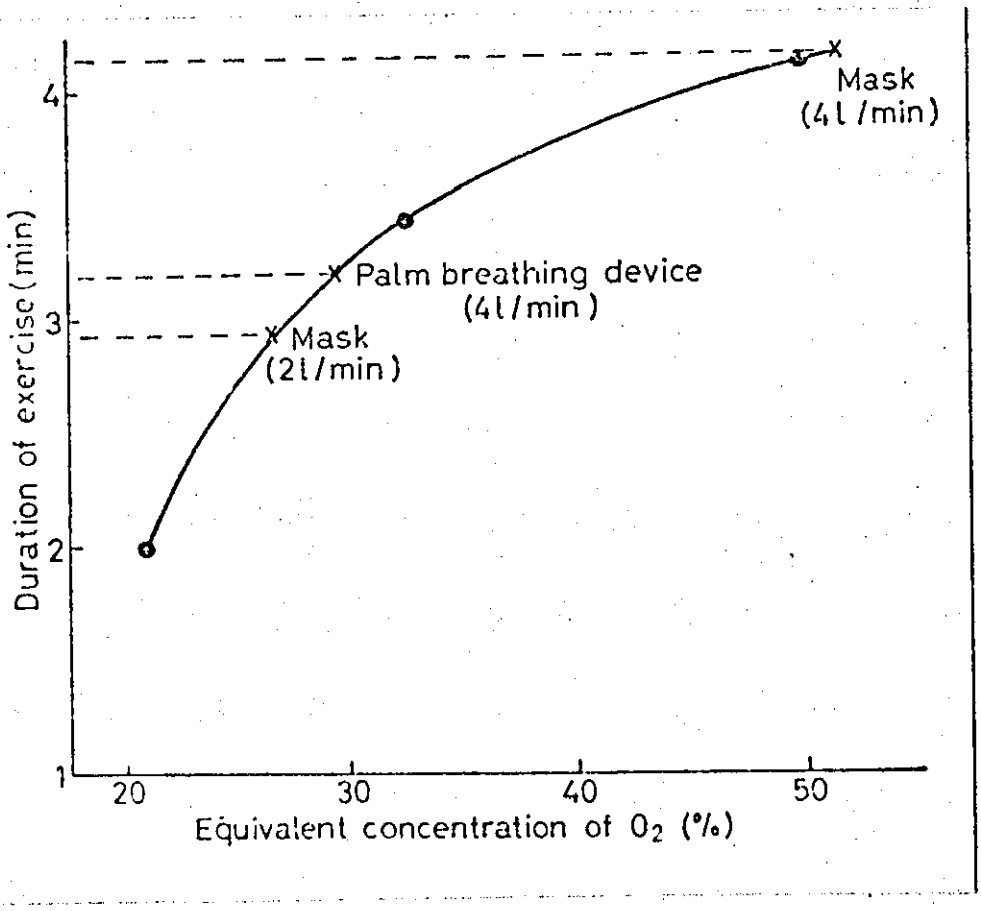


FIGURE 8 Effects of breathing oxygen or exercise tolerance of COLD patients (Cotes 1968).

However, O_2 delivery system are invariably intrusive and socially unacceptable, and satisfactory criteria are not available to determine the concentration, duration of administration or the

type of patient who would benefit from such therapy. Furthermore, O_2 is known to damage the lung through its injurious effects on capillaries and alveolar epithelium (Pierce & Saltzman 1975).

The response to physical therapy is variable. Postural drainage, often accompanied by steam inhalation, a water-mist aerosol or percussion of the chest, can remove secretions in the bronchial tree, but according to Jones (1975) does not appear to influence the long-term outlook of COLD patients. Miller (1952) and Barach (1966) feel that breathing exercises are beneficial, but although Barach reported a 20% reduction in $\dot{V}_E$ 1. min after a period of training, there were no accompanying changes in Sa_{O2} or Pa_{CO2}; Thoman et al (1966) found that pursed-lips breathing improved Pa_{CO2} and the ventilatory rate of the most slowly ventilated component of FRC, but these improvements were due, very largely, to the accompanying drop in f_R and increase in V_T. However, a study by Paul, Eldridge, Mitchell and Fiene (1966) revealed that despite improved function accompanying reduced f_R patients often feel less comfortable than when breathing at their own rate. On the other hand Cotes (1968) claims that there is little objective evidence to support breathing exercise procedures; Degre et al (1974) report no changes at rest or during exercise after 6 weeks breathing training, Jones (1975) feels that the effects on pulmonary gas exchange and the long term benefits have not been established, and Saunders & White (1965) were of the opinion that claims of relief from dyspnoea by an experimental group 'were moved by gratitude rather than fact'.

The fundamental point is that none of the above measures can counteract the underlying problem of cardio-respiratory deconditioning.

Once dyspnoea becomes a deterrent to continued physical activity a vicious cycle of events begins. Inactivity leads to further deconditioning and diminition of muscle tone and efficiency so that fatigue rapidly accompanies slight exertion and breathlessness becomes more intrusive (Miller et al 1962, Bass et al 1970). According to Smodlaka et al (1974) 'severely affected patients have very low pulmonary reserve, low maximal working capacity and poor ability to maintain a steady state sub-maximal, work load. Activities of daily living are a heavy load, near maximal, for them.' They fall easily into oxygen debt with severe dyspnoea and hypercapnia on the slightest exertion. A self imposed limitation of activity by the patient is therefore understandable, but the fact that a similar course of action is frequently recommended for COLD patients is less obviously understood.

Numerous reports repeatedly suggest that patients who remain active are in relatively better health for longer periods than their more sedentary counterparts. Over a decade ago Miller et al (1962) demonstrated the value of exercise therapy in promoting a state of well being and making patients physically independent. Pierce et al (1964) felt that most COLD patients should not only be encouraged to remain physically active to the limits of their tolerance, but should also undertake appropriate physical conditioning programmes, and Hass et al (1969) see beneficial prognostic implications for exercise oriented rehabilitation. According to Woolf et al (1969) programmes can be simple, and Christie (1968) claims that programmes may be run on an outpatient basis with little more supervision than is usual in any clinical practice.

The effects of progressive exercise therapy in COLD were first documented by Pierce et al (1964) , and Woolf et al (1969) feel that there is general agreement that patients are able to increase the amount of tolerated exercise as a result of training programmes. Many workers report marked subjective improvements, with a feeling of general well being and daily activities being carried out with much more ease. Objective measures of training have been cited to substantiate the subjective assessments, and more detailed discussion of these objective measures now follows.

a) Cardiovascular function (CVF)

In common with normals, and IHD patients, reductions in f_H at rest and during sub-maximal work have been observed in COLD patients. Miller et al (1962) report reduced f_{HW} in individual cases; Pierce et al (1964) recorded f_H drop from 137 - 104 b. min. at a given treadmill speed (N=9, $P < 0.05$); Paez et al (1967) and Bass et al (1970) disclose significant reductions in f_{Hr} (83.1 - 78.4, N=8, $P < 0.01$; and 90.0 - 75.9, N = 11, $P < 0.001$ respectively), and the latter group observed slower f_{HW} (120 - 98, $P < 0.01$). Smodlaka et al (1974) report lower individual f_H at standard work, but a study by Vyas et al (1971) found no such change. In no study was a change in f_{Hmax} reported.

Indirectly measured systolic blood pressure fell in certain cases (Smodlaka et al 1974), and Degre et al (1974) describe a 25% drop in mean resting pulmonary artery pressure (N = 11, $P < 0.05$) following 6 weeks training. Changes in the peripheral circulation have been observed by Paez et al (1967), a - $\bar{v}O_2$ difference increasing, producing

a concomitant reduction in $\dot{Q}$, $P < 0.025$ in each case.

b) Respiratory Function (RPF)

Changes in resting lung function variables occurred, but the changes were inconsistent, often small and rarely significant. Ambrus et al (1970) found that $FEV_{1.0}$ improved significantly after 6 weeks exercise ($N = 43$, $P < 0.05$). Blood gases were affected in four studies; Ambrus et al (1970), and Degre et al (1974) observed increased resting Pa_{02} (78.3 - 82.3, $N = 34$, $P < 0.01$; 76 - 82, $N = 11$, $P < 0.05$ respectively); long term improvements are reported by Petty et al (1969) - Pa_{02} and Pa_{C02} were raised (57.2 - 60.1, 40.2 - 43.6 respectively $N = 182$, $p < 0.025$) one year after the initial study. Woolfe et al (1969) report a reduction in 90% desaturation time ($N = 14$, $P < 0.01$), but in no study was Sa_{02} significantly improved.

Improved ability to transport oxygen has been claimed, usually on the basis of increased $\dot{V}_{02\ max.}$ Christie (1968), relating the measure to body area, describes a mean increase of 102.9 ml. sq. m. ($N = 11$, $P < 0.01$); Vyas et al (1971) report increased $\dot{V}_{02max.}$ (0.630 - 0.690 L. 0.5 min, $N = 14$, $P < 0.05$), and in total V_{02} (2.94 - 3.58 L., $P < 0.015$); Degre et al (1974) observed a 10% improvement in 'symptom-limited' oxygen uptake ($\dot{V}_{02sl}$), (1.229 - 1.350 L. min, $N = 11$, $P < 0.05$). Petty et al (1969) report improved mechanical efficiency, and Hass et al (1969) describe lowered $\dot{V}_{02}$ during work and a reduction in oxygen debt with quicker recovery following a mixed training programme. The use of the term $\dot{V}_{02max}$ may be inappropriate in the present context; it is used to signify the $\dot{V}_{02}$ at maximum tolerated work rather than the generally accepted criteria proposed by Taylor et al (1955), and the terms 'breaking-point $\dot{V}_{02}$' (Nicholas et al 1970) and $\dot{V}_{02\ sl}$ of Degre et al (1974) are more meaningful.

c) Physical Work Capacity (PWC)

Increased work tolerance is the consistent feature of all studies reviewed. Bass et al (1970) and Vyas et al (1971) are the only groups to measure PWC_{max} directly and both report increases in mean maximal work rates; (170 - 350 Kg. m. min. (42. 2 - 49 W), N = 14, $P < 0.025$ respectively). These groups calculated work from a step-test or stair climbing; Christie (1968) describes a mean increase of 65. 9 Kg. m. min. (10. 5W), N = 11, $P < 0.01$) in stepping whilst Petty et al (1969) and Guthrie et al (1970) noted an increase in the number of stairs climbed (42 - 70, 42 - 68, $P < 0.001$ respectively) with a resultant increase in work achieved (calculated as 485 - 819 and 582 - 875 Kg. m, $P < 0.001$); however the time taken to complete the work was not given hence changes in work rate are unknown.

Numerous claims of increased work tolerance are made on the basis of continuous walking time, either on a level treatmill or along a corridor, before dyspnoea halts the performance. Since this kind of variable is heavily dependent on patient motivation and is influenced by habituation and other processes, alternative indices of training response are needed to support the claim of improved physiological function.

Indeed, impressive though the objective improvements may be in revealing the trainability of these COLD patients, defects in experimental design are apparent, and some of the claims cannot be supported by the data. Examples are given below:

a) over half the studies reviewed included other forms of treatment in addition to exercise, thus it is impossible to attribute any changes to the exercise regime alone.

b) not all studies report habituation procedures; therefore post training changes may include marked habituation to equipment, personnel and environment rather than changes brought about by exercise. Reduced f_H at a given treadmill load may be nothing more than habituation (Davies et al 1968), and Christie (1968) claims an increase in O_2 extraction rates on the basis of reduced $\dot{V}_E$ (by 3.55 L. min, $N = 11$, $P < 0.05$) at standard work; a similar response could be attributable to reduced anxiety and less hyperventilation. There was an improvement in 0_2 extraction but the exercise may have played no part in bringing about the change.

c) many of the changes have occurred when a single device (usually an ergometer or treadmill) was used to both test and train the patient. In these cases the problem is to distinguish between fundamental changes in gas transport which may enable more work to be done, a learning process where the patient becomes more efficient at performing the exercise and can therefore increase work output without altering gas transport, or quite simply a gradual improvement in confidence which encourages the patient to increase his performance. The problems concerning habituation and learning are dealt with more fully later.

d) statistical validation is absent from a number of the studies. Not one of the studies reviewed satisfied four criteria viz: exercise as the only change in therapy, an habituation process, different devices for testing and training, and statistical validation;

furthermore the mechanisms by which changes occur are not certain and the need for further investigation is clear.

The psychological basis for exercise therapy

Psychological disturbances invariably accompany chronic illness (Katz, Bruce, Plummer & Hellerstein 1958; Biorck 1959; Barry 1966; Hammett 1966; Mulcahy & Hickey 1971). The emotional response of IHD patients particularly are 'prominent in almost every case and often constitute the major disability' (Hellerstein et al 1968). Anxiety, depression and denial of illness are the most frequent reactions (Barry, Daly, Pruett, Steinmetz, Birkhead & Rodahl 1966; Biorck 1964; WHO 1963), and emotional reassurance was required in 71% of patients attending a work evaluation unit before rehabilitation could be effected (Hellerstein 1959). Psychological disablement was a major problem in 30% of unemployed patients in an Australian Centre (Editorial 1964), and varying degrees of psychological symptoms and personality difficulties were apparent in 16 patients still out of work 6 months after infarct (Sharland 1964).

Psychological problems are sometimes aggravated by restriction of activity until an almost neurotic fear of any physical effort develops (WHO 1963, Torkelson 1964, Kellerman, Levy, Feldman & Kariv 1967). As long as activity is restricted there is the likelihood that patients will develop a moderate degree of depression (Newman et al 1948; Hammett 1967; Hellerstein et al 1968; Archibald & Gefter 1970). Hammett believes the occurrence is so frequent that it may be considered a normal reaction, and Hellerstein reports that recovery is often spontaneous particularly when the patient begins self-care. However many workers content that severe depression does occur; behaviour is self-centred, thought processes slow, ideas of guilt or worthlessness appear (Archibald et al

1970); emotional stress accompanies the patient's renouncing of ownership of his own body (Hellerstein et al 1968). According to Hammett this sort of reaction is not self limiting but tends to continue and worsen, and Archibald and Newman with their co-workers report that older individuals are greatly affected even after a few days in bed.

There is agreement among some workers that exercise produces beneficial psychological effects (Eggleston 1940; Newman et al 1948; Katz 1967; Alderman 1967; Kennedy 1969), and Harrison & Reeves (1965) feel that the importance of properly regulated and prescribed exercise in overcoming fear cannot be over-emphasised. Clausen et al (1969) also feel that patients are highly susceptible to the psychotherapeutic aspects of treatment and that increased physical activity is an indispensable condition for improvement. The many reports of subjective improvement in patients general outlook, irrespective of the lack of any objective improvement, indicate that the initial value of any training programme may be the psychological reassurance which develops (Hellerstein et al 1963; Kaufman & Anslow 1966; Paivio 1967; Rechnitzer 1967; Kavanagh, Shephard, Pandit & Doney 1970; McGlynn 1970).

Among the benefits that have been observed are improved self-image, subjective well-being and marital relations, loss of fear of exercise and moderately strenuous activities, and the ability to perform such activities more easily (Kattus & KacAlpine 1965; Sloman, Pitt, Hirsch & Donaldson 1965; Zohman & Tobias 1967; Morgan, Roberts, Brand & Feinerman 1970). Occasionally over-reaction occurs and patients become euphoric to such a degree that limits have to be placed on their physical activity (Kennedy 1969).

The sociological basis for exercise therapy.

There are two important factors to be considered under the

heading of the sociological consequences of progressive exercise therapy on patients with CRD; firstly the recurrence/mortality rates, and secondly the possibility of gainful employment. Studies of these particular aspects are rare, but three reports - 2 on IHD patients, 1 on a COLD group - suggest that exercise can be a favourable influence.

Gottheiner (1966) reviewing five years experience and over 11,000 exercising IHD patients reports a mortality rate of 3.6% compared with 12% for a non-exercising grou. Rechnitzer et al (1971) describe a follow-up study on 68 exercising patients matched with two non-exercising groups (N = 198) for age and year of infarct; 7.4% of the exercising group have died, 2.9% had non-fatal occurrence, compared with 13.3% and 26.0% respectively for the other group. The prognosis for patients with moderate to severe COLD is poor - less than 50% survival rate over 5 years (Simpson 1968), but an exercise oriented rehabilitation programme, (Hass et al 1969), run over 5 years, showed that 22% of an experimental group (N = 252) died of respiratory failure compared with 42% of a non-exercising group (N = 50), whereas the percentage dying from causes unrelated to their disease were similar (8% and 6%).

Many groups have studied the employment patterns of IHD patients, and the findings can be summarised as follows:-

a) There is no evidence that employment adversely influences mortality statistics (Master, & Dack 1940; Sharland 1964; Stein & Altman 1965).

b) 80% or more of surviving patients now return to some form of employment (Katz et al 1958; Crain & Missal 1965; Mulcahy, Hickey & Coghlan 1972).

c) Downgrading as a result of heart disease occurs infrequently, and the majority resume full-time employment at their previous employment (Crain et al 1965).

d) A crucial factor in the delayed return to work of 'heavy workers' was the fear that hard physical work would provoke further attacks (Lund-Johansen 1965).

e) Many unemployed IHD patients are capable of work (Editorial 1964).

f) Rehabilitation-oriented hospital care favourably influences return to work (Gilbert & Auchinloss 1955; Harris, R. 1965; Mulcahy & Hickey 1971).

It is acknowledged that the vocational aim of rehabilitation in COLD patients may not always be realised, but there is evidence that a return to gainful employment is possible in certain cases. Hass et al (1969) compared exercising and non-exercising groups, and found marked differences in the employment patterns of the two groups. In the exercising group, (N = 252), 6% returned to their previous job and were working 5 years after entry to the programme; a further 13% were returned to a new job; 6% were trained for new jobs but were unable to obtain employment. The figures for the non-exercising group (N = 50), were 3%, 0% and 0% respectively. In a two-year study by

Petty et al (1969) and Guthrie et al (1970) 21 patients employed whilst on a rehabilitation programme earned 135, 689 dollars, 35 others participated in some gainful employment and 8 were returned to work after one year's unemployment - (the total number involved in the study was 182).

Although not as dramatic as the IHD pattern, exercise oriented rehabilitation for COLD patients may be a method whereby a significant number of patients, who would otherwise remain totally dependent on family or the state can return to gainful employment, or at least can be returned to self-sufficiency and self-care thus releasing another member of the household for possible employment.

CHAPTER THREE

DESIGNING THE EXPERIMENT

INTRODUCTION

It is generally agreed that the assessment of exercise regimes for patients with CRD is a difficult process and despite numerous reports of the beneficial effects of training Kavanagh & Shephard (1973) feel that adequately controlled studies are lacking. Problems facing the investigators include wide inter and intra- individual variability, (diurnal and seasonal, and involving presence or absence of severe disease, environmental factors, occupation, leisure pursuits, dietary and smoking habits and motivation), criteria for patient selection, assessment of physiological status, exercise prescription and monitoring, control group activity, large numbers necessary and a fairly high drop-out rate. The factors, considered to be of prime importance in designing this experiment, are discussed more fully below.

Evaluation of physiological status

A wide range of techniques exists to assess lung function at rest. Lung capacity and volume, transfer factor, lung compliance, airway resistance, ventilation/perfusion ratio, distribution of inspired gas and ventilatory capacity are all recommended measures (Garbe & McDonnell, 1964, Bass 1966, Bates et al 1964), and are all adequately described by Cotes (1968).

Until the mid-1950's the assessment of a patients physiological capacity for work was preponderantly subjective (Bruce, Rowell, Blackman & Doan 1965; Bruce & Hornsten 1969; Hellerstein et al 1968; Varnauskas 1967). Several criteria-fatigue dyspnoea, pain during work and normal activity (Katz et al 1958; N. Y. Heart Assoc. 1966) were used to determine the range of permissible activities. Such history is helpful but not specific; for example the psychological state of the patient - his work motivation, pain threshold and need to deny pain - and social pressures may help to obscure accurate assessment. In addition little insight is obtained if the patient is overtaxing himself (Rogers & Hurst 1964), and clinical evaluation alone cannot indicate cardio-pulmonary reserve nor quantify the effect of therapy (Denolin & Messin 1966; WHO 1964; Bruce, Sparkman, Levenson & Hurley 1969; Freidberg 1966). Furthermore the reliability of a subjective approach is questionable (Burchell 1968; Cooper 1970; Frasher, Stivelman & Horovitz 1963; Levenson & Sparkman 1961); although good correlations between subjective and objective evaluation can be achieved (Hellerstein 1959), 30 - 35% differences between subjective appraisal and an exercise test have been described (Frasher et al 1963). The inaccuracies reported were fairly evenly divided between those who showed unsuspected abnormalities when none were predicted and those in whom a significantly abnormal response was anticipated but did not occur.

A single method of evaluating response to exercise in patients has yet to be developed however. The early exercise tests were based on the long-standing Master-2 step test (Master & Oppenheimer 1927);

post exercise f_H, blood pressure (BP) and ECG traces only being recorded. Shortly afterwards the same variables were recorded during work and estimates of cardiac output ($\dot{Q}$) and myocardial oxygen consumption (Mv_{O_2}) were obtained from pulse pressure (PP) and tension time index (TTI). Ford and Hellstein (1957) then added respiratory exchanges in measuring the energy expenditure of the Master 2-step test. Although a single level test provides valuable information the standardised load leads to over-stressing of the severely impaired and under-estimation of the reserves of the mildly impaired (Burchell 1968; Skinner, Benson, McDonough & Hames 1966), and did not discriminate between IHD patients in functional classes I and II (New York Heart Assoc. 1966) and normal controls (Bruce et al 1963). Multi-level tests were developed, particularly for IHD patients, the procedures and constraints being adequately described in the WHO technical report (1968).

The purpose of an exercise test is to assess the patient's ability to perform external work over a period of time. High work rates can be maintained over short periods of time ($<$ 1 minute) through anaerobic mechanisms, but the crucial feature of work performance over periods greater than 1 minute (but less than 1 hour) is the patient's ability to transport and process oxygen. The best single indicator of this ability is the $\dot{V}o_{2max}$ test (Taylor et al 1955, Mitchell et al 1958). A maximal test (i. e. close to $f_{H\ max}$) is said to be superior in assessing risk for future IHD, diagnosis of atypical chest syndrome, degree of cardiovascular impairment and evaluation of therapy (Doan, Petersen, Blackman & Bruce 1968; McDonough & Bruce 1969), but the $\dot{V}O_{2\ max}$ test may be unacceptable for elderly patients with lung disease,

and a submaximal work test substituted.

It has been shown that the relationship between steady-state f_H and $\dot{V}_{02}$ during a multi-level sub-maximal work test distinguishes between normals and patients (WHO 1966), trained and untrained normals (Hale et al 1970), and between pre and post training responses of both a normal group (Hale - awaiting publication) and a group of IHD patients (Hellerstein et al 1966). In the case of normal subjects significant differences occur in both the slope and the intercept of the $\dot{V}_{02}/f_H$ regression lines (figure 9). Similar differences might also exist between normal people and COLD patients and an experiment to test this hypothesis was devised.

A brief description and the main conclusions are presented here, but a more thorough coverage of the experiment is given in Appendix A. Twenty-eight males took part, 7 mature students at a college of education and 21 patients attending King Edward VII Hospital Midhurst. The students were designated the normal group (C), the patients were the experimental group (E), and were subsequently sub-divided into group E, (n = 11), E_2 (n = 11), E_3 (n = 6) reasons being given in the appendix. There were no differences in age or weight between groups C and E and the work test administered were similar in concept and differed only in the application of work loads, gas collection procedures, and equipment used.

The most important conclusion was that the test, as administered, did not differentiate between the healthy and the sick in a meaningful manner. The reasons suggested involve problems in sampling expired gases, inter and intra subject variability in low work load situations,

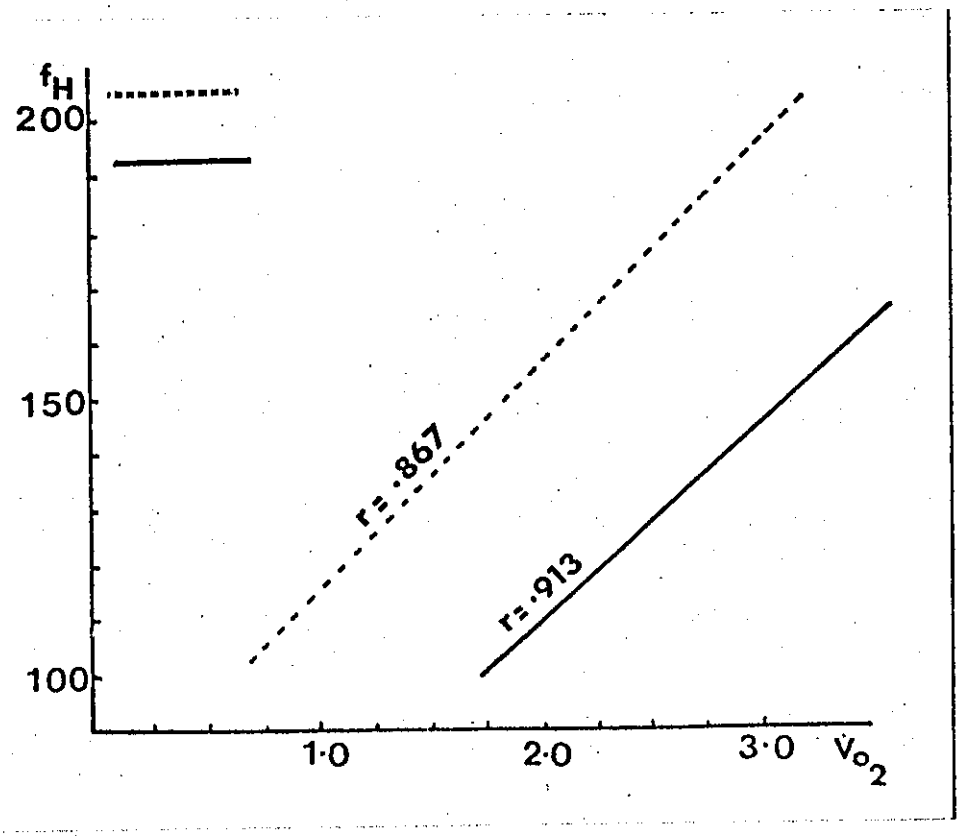


FIGURE 9 Heart rate - oxygen uptake relationship during work of trained (solid line) and untrained (dashed line) groups.

the inability of patients to complete the necessary work loads, and perhaps most crucially the apparent inability of patients to reach steady-state conditions in the time allowed (figure 10).

In addition it is conceivable that the early seconds of increased work contain a great deal of valuable information about a patients response to exercise that is concealed during steady state testing. It was this notion, together with the availability of a respiratory mass spectrometer and gas flow-meter that led to the design of a method for

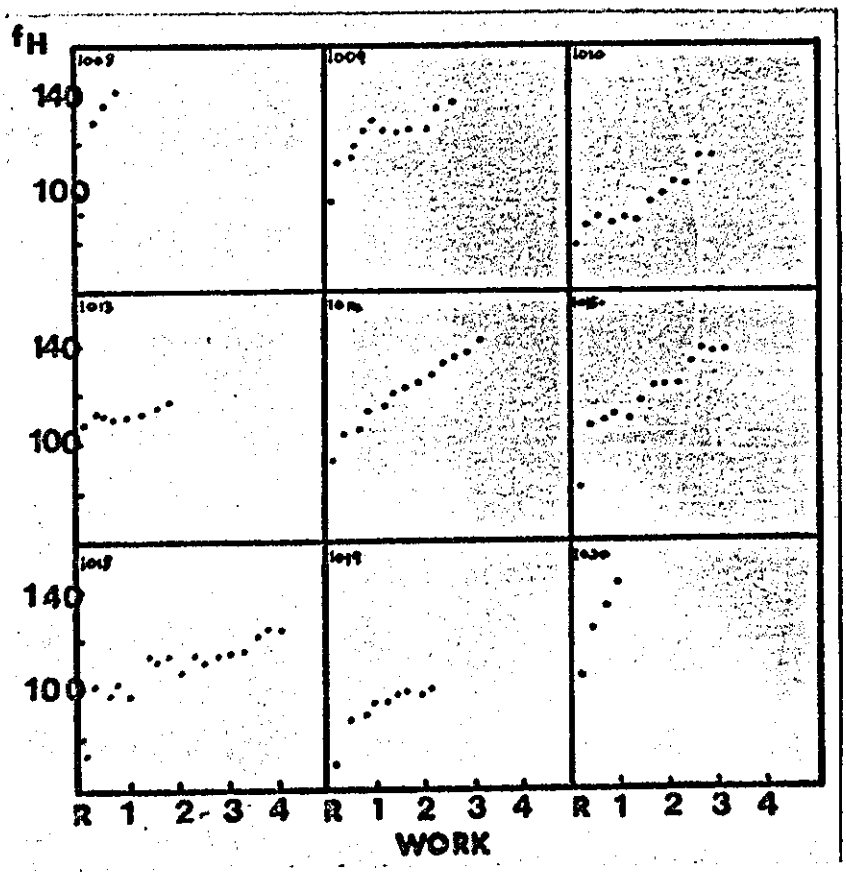


FIGURE 10 Heart rate during steady state work tests; patients of varying ages suffering from non-specific lung disease.

continuously recording cardio-respiratory response to a work test. Methods of calculating expired gas volume and anlysing the content are now described.

The Respiratory Mass Spectrometer (RMS)

Mass spectrometry has been widely used in various fields for a number of years, but application in the field of respiratory physiology has been a comparatively recent advance (Nesrajah 1965). Early RMS were restricted in the choice of gases to be analysed, but in 1957, K. T.

Fowler produced an instrument which satisfied the requirements of clinical respiratory physiologist. The instrument used in this study is a centronics residual gas analyser and brief details of the principles involved and the linearity and accuracy of the device are presented in Appendix B.

According to Nesrajah (1965) gas analysis by RMS has several advantages. It is capable of simultaneous analysis, with reasonable accuracy, of many constitutents in a small sample of gas mixture regardless of the composition of the diluting gases, with a speed, specificity and sensitivity unmatched by any of the other known methods. These virtues allow a thorough examination of the respiratory patterns of cold patients undergoing exercise, and may reveal changes in function resulting from training that are obscured when steady-state measures are being analysed.

The measurement of gas flow

Numerous methods exist for measruing gas flow particularly whilst the subject is at rest. Many of these methods however are less appropriate for an exercising subject because of increasing resistance that develops or because the equipment is not designed to cope with high ventilatory volumes. With the development of sensitive pressure transducers, pneumotachographs can provide accurate measurement of gas flow with very little resistance (10 mm. H_2O at flow rates up to 16 l sec (Cotes 1968). Providing the instrument gives a linear response the signal may be integrated to give volume in unit time ($\dot{V}$).

Errors can occur in gas flow measurement primarily when the viscosity of the gas passing through the pneumotachograph changes. Three factors affect viscosity; first there is the composition - if the concentrations of the various gases alters markedly, viscosity will be affected; secondly, temperature changes - increased gas temperature, unlike fluid, increases viscosity; thirdly, water vapour has a lower viscosity than air and changes in humidity affect the signal output (Smith 1963; Grenvik, Henstrand & Sjogren; Hobbes 1967). Passing expired air through a heated pneumotachograph placed on the expired side of the valve box overcomes these problems adequately, although Bradley (personal communication) found that a cold pneumotachograph gave reliable results during short periods (5 minutes) of normal breathing, and Fry et al (1957) found that although condensation was apparent on the jacket and the screen of a Statham Flowmeter, the calibration factor was unaffected. In a recent study Finnecane et al (1972) revealed that the geometry of the tubing leading to the pneumotachograph and beyond affects the linearity of response. It is essential therefore that the linearity of the pneumotachograph is tested as it is to be used in the work test procedures, and a description of the validation of the pneumotachograph is given in Appendix C.

An example of the electrical output obtained during a work test is shown below (figure 11). and its analysis by computer described in Appendix D.

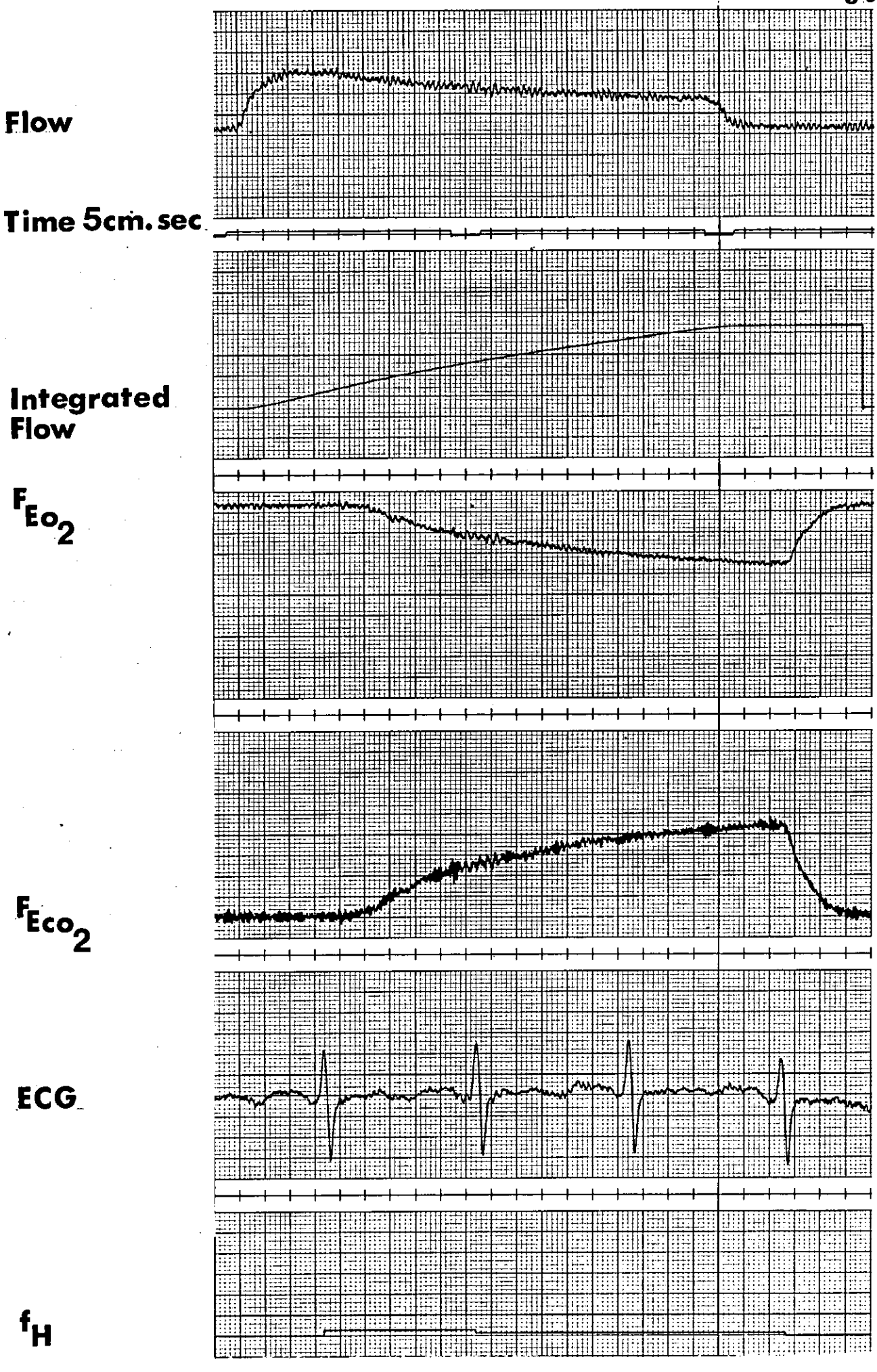


Figure 11. Electrical output recorded during a work test.

Habituation, learning and training

These phenomena, already briefly mentioned in a previous section (page 35), pose particular problems in investigations of the value of training programmes. Changes in physiological responses to work-tests resulting from habituation (chiefly through the agencies of reduced anxiety, familiarity with apparatus, environment, instructions and personnel), and from learning (achieved through regular performance of a specific task), are very similar to and often confused with changes produced by training-reduced f_H at rest and sub-maximal work, lower f_R and decreased $\dot{V}_E$ are three common examples. Habituation is likely to be an even greater problem with elderly patients who may be afraid of physical activity and have an understandable aversion to devices like nose clips and mouth pieces that tend to interfere with an already impaired ventilation. Learning is almost certain to occur if patients are trained on laboratory equipment such as a treadmill.

The problem was studied in a pilot experiment involving a single patient; a description of this experiment can be found in Appendix E, but the main points are given below.

A 49 year old serviceman was admitted to hospital complaining of exertional dyspnoea and inability to cope with his duties. Emphysema was diagnosed and the patient agreed to undergo progressive exercise therapy prior to medical discharge. The procedure followed was briefly

a) work tests on ergometer and treadmill.

b) a 5-day habituation programme to the treadmill only.

c) work tests on ergometer and treadmill repeated.

d) four weeks training - 30 minutes treadmill walking daily, 5 days a week.

e) work tests on ergometer and treadmill repeated.

f) lines of best fit by least squares method were obtained for $\dot{V}_{02}/f_H$ relationships during all work tests; the results are given below.

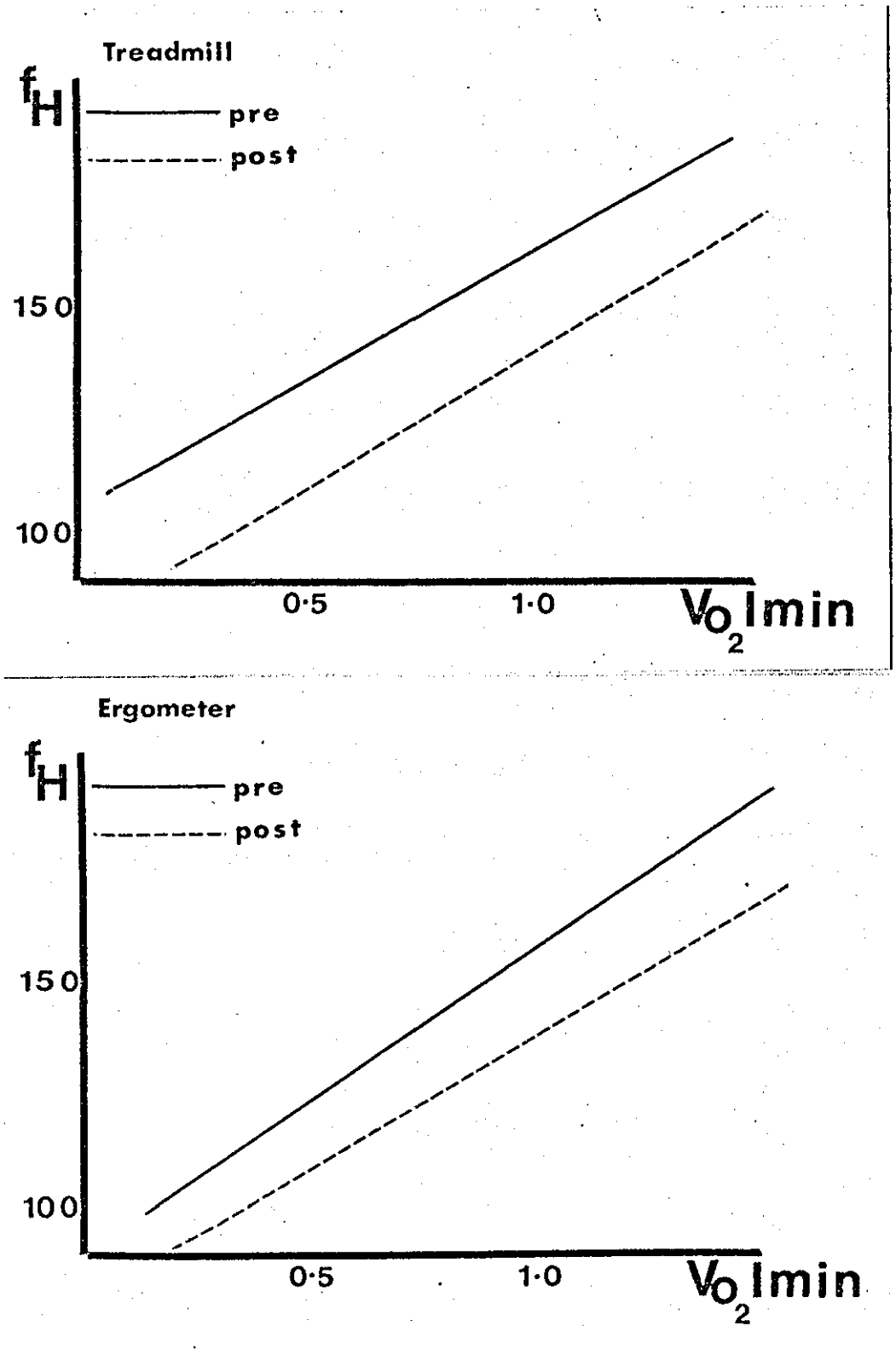


FIGURE 11 Heart rate - oxygen uptake relationship during work tests pre and post habituation.

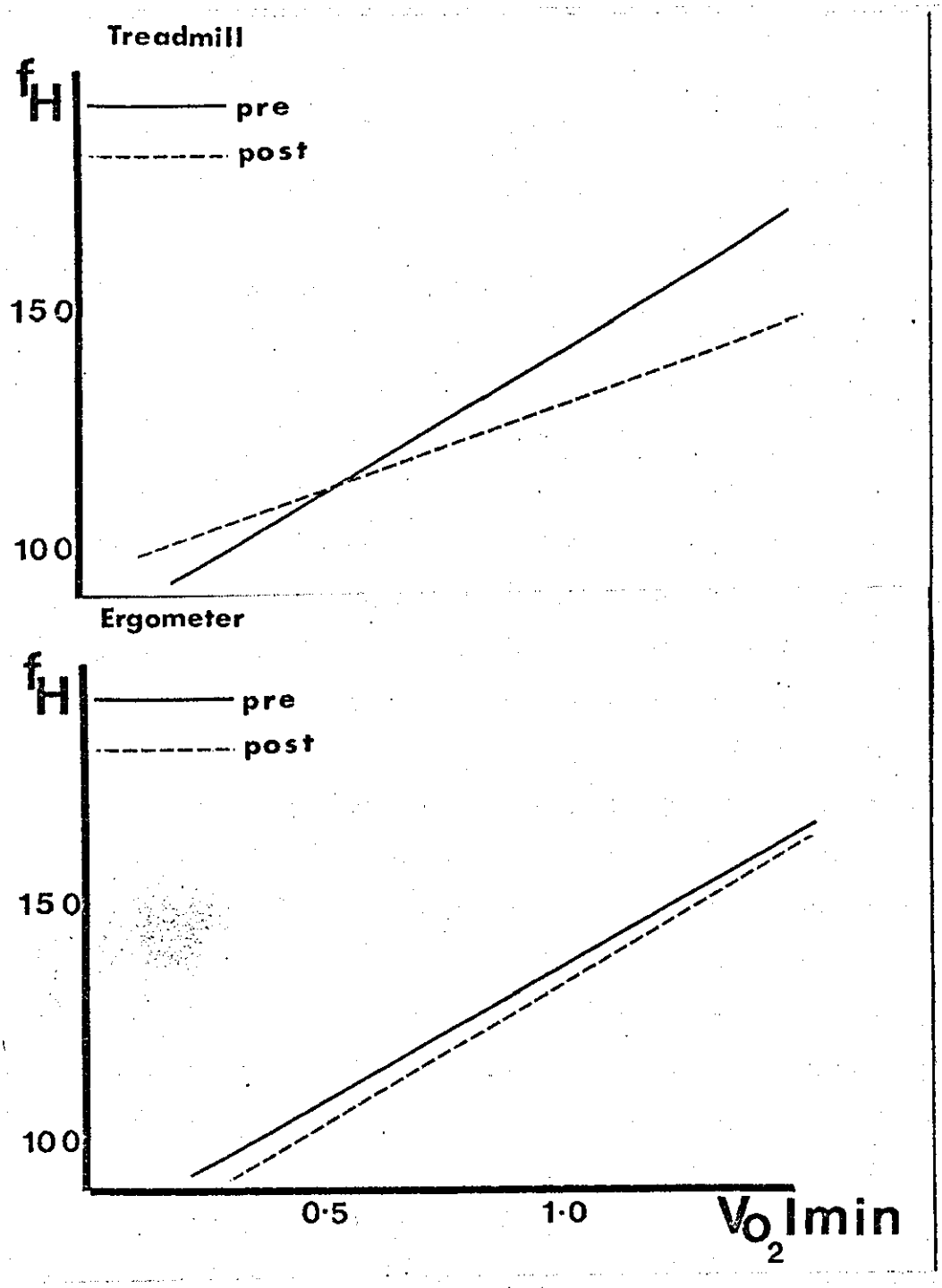


FIGURE 12 Heart rate - oxygen uptake relationship during work tests pre and post-training.

Three things are immediately apparent. First, the treadmill familiarisation programme produced a marked change in the intercepts of the $\dot{V}_{02}/f_H$ regression lines of both treadmill and ergometer during the second work test; this can only reasonably be put down to an habituation response. Second, the treadmill training produced a distinct learning process. Thirdly, the training response to treadmill walking was specific to that activity, since the response to the ergometer test shows little change. As a result, the present study incorporates an habituation procedure and since the bulk of the training takes place on the treadmill,

both treadmill and ergometer work tests were employed to evaluate pre and post training responses.

Exercise prescription

According to Wilmore (1974) the ability to prescribe exercise for both normal people and patients has advanced from an art to a science during the past decade. In prescribing exercise, four factors need to be considered, namely, the volume or frequency of participation, the duration of each session, the intensity of the exercise and the type of activity. Underpinning these factors is the principle, established by Christensen (1931), that work loads should be increased when adaptation to a particular load becomes apparent. Karvonen, Kentola, and Mustala (1957) quantified the minimum training requirements for normal subjects as follows: volume - 4 weeks, five sessions per week; duration - approximately 30 minutes per session; intensity - work sufficiently strenuous to maintain f_H at > 140 b.min. for the duration of the session; activity - whole body movement, i.e. treadmill running.

The most important factor is said to be the intensity at which the individual performs his activity (Wilmore & Haskell 1971; Nordesjö, 1974) and there is the need to establish individual levels of intensity taking such things as age, medical condition and pre-training capacity into account. A level of intensity that is too high, could have serious medical consequences for some, whereas a level that is too low may not produce beneficial results. Roskamm (1967) describes a method whereby the exercise intensity can be determined on an individual basis: the equation is

$$f_{H\ \text{training}} = f_{H\ \text{rest}} + \left[(f_{H\ \text{max}} - f_{H\ \text{rest}}) \times 70/100\right]$$

Individual differences in resting and maximum heart rates are accounted for, and for a normal 20 year old with f_{Hr} of 70 b. min and $f_{H\ max}$ of 200 b. min an f_{Ht} of 161 b. min. is the minimum intensity level. The f_{Ht} of a healthy 60 year old male (f_{Hr} = 60, $f_{H\ max}$ = 160 b. min) is therefore 130 b. min. But a heart rate of 130 b. min is often beyond the maximum capacity of many patients with COLD since intolerable dyspnoea brings work to a halt before such heart rates are reached. A modification to Roskamm's formula, replacing f_{Hmax} by 'breaking point f_H (f_{HBP}) overcomes this problem. Admittedly f_{Ht} is now dependent on the patient's level of motivation and anxiety state, but there is the advantage that the demands of training are within the capabilities of the patients - a possible safeguard against high drop-out rates - and, providing exercise testing is carried out with care, a reasonable degree of $f_{H\ BP}$ repeatability can be achieved.

Finally, the way in which the exercise is administered may be important. Early training schedules consisted of continuous activity, but many patients are unable to maintain even low work loads for the specified 30 minute duration. A study by Christensen, Heldman and Saltin (1960) revealed that quite heavy amounts of work could be achieved, by alternating work with recovery periods, without the physiological stress that frequently accompanies continuous work of comparable amounts. This alternating pattern of activity has been applied successfully to rehabilitation programmes for patients with CRD as well as athletes in training (Smodlaka 1966); Sime & Herbert 1971), and is the basic method employed in this study.

Monitoring training.

Monitoring individual sessions is important for two reasons.

Firstly, if a training threshold has been set (minimum f_{Ht}) it is necessary to know that the work-load is actually above that threshold; a sub-threshold f_H indicates the need for increased work load or working time. Secondly, it is essential to know that a patient is not overtaxing himself, and whereas the dyspnoea of the COLDpatient is sometimes an overt indication of stress the danger signals from IHD patients are frequently observed from the electrocardiogram produced on an oscilloscope.

The simplest approach is to monitor f_H, and improved instrumentation means that monitoring in the laboratory controlled sessions is routine. Developments in miniaturised equipment and bio-medical transmitters also permit evaluation of a wide range of everyday activity in a variety of situations.

Monitoring habitual physical activity (HPA)

Evidence is offered elsewhere (page) that some patients do not utilise the functional capacity to the full because they are frightened. A supervised controlled exercise test often improves the patient's confidence to a marked degree (Kennedy, 1969) which in turn may lead to a dramatic change in HPA. Such changed activity patterns may be great enough to produce a training effect additional to that of the supervised programme, leading to over estimation of the benefits of training programmes. Therefore it is important to obtain some idea of the patients HPA patterns before training begins and although assessment of HPA is not easy, a combination of questionnaire, diary and monitoring of heart rate should provide sufficient information.

The control group

The introduction of a control group into a study raises fundamental

problems in experimental design. Apart from considerations of adequate matching of the groups, a major problem is the activity undertaken by the control group. In the majority of studies the 'controls' are a strangely neglected group, coming into focus only at the beginning and end of the experiment. However, the 'Hawthorn effect' is a well-known phenomena and it seems an unrealistic procedure to give a fair degree of attention to an experimental group, almost ignore a control group and then ascribe all the changes that occur in the experimental group to the training programme alone. It is conceivable, particularly in the case of patients who are susceptible to psychological aspects of treatment (page 36), that the amount of attention received is an important as the exercise undertaken.

If the theory of a minimum training threshold has any validity then it should be possible to give a control group a similar degree of attention (though not necessarily in terms of total time), and prescribe a regime where the number of contacts is similar but where the exercise prescribed is well below the minimum requirements in terms of intensity and duration of session. In this case the only feature which distinguishes between experimental and control groups in the nature of the exercise programme and could lead to more objective evaluation of such a programme.

PART TWO

THE EXPERIMENT

CHAPTER FOUR

The Subjects

Over a period of 18 months 25 patients were referred by consultant physicians for possible inclusion in the study. Eight were R. A. F. Chest Unit; six were private patients; ten had retired, two were unemployed as a result of their condition and one was a woman. Three, including the woman, felt unable to accept the conditions imposed by the design of the study. Five were rejected, two because of E. C. G. abnormalities on exercise (S-T segment depression, T-wave inversion) and the other three because of uncertainty over the irreversibility of their condition. One withdrew because of family and business pressure, another was withdrawn and hospitalised for treatment of acute bronchial infection and pre-infarction illness. One patient took part in the pilot study already mentioned and described more fully in Appendix E.

To ensure group viability the first six patients entering the programme were assigned to the experimental group (E) the next six to the control group (C). Thereafter subjects were assigned alternately. Neither physicians nor patients were aware of this division and therefore selection of patients was not influenced by physician knowledge of the different treatments to be administered. One control subject underwent investigatory surgery during the study and a member of group E completed ten days training only, and so final numbers were E = F and C = 5; all but two (from group E) were resident patients although three did return home for week-ends.

Anthropometric data of both groups are given in table 2.

TABLE 2

Anthropometric data of the two groups (means and SD's)

	Age years	Ht cms	Wt 1 kg	Wt 2 kg (Post training)
Group E				
	60.9	170.0	63.4	63.1
S =	6.29	3.89	8.33	7.27
Group C				
	63.4	171.1	57.8	58.5
S =	8.89	7.66	15.50	17.02

CHAPTER FIVE

METHODS

INTRODUCTION

Patients were studied for a maximum of six weeks; according to Karvonen et al (1955) a minimum of four weeks continuous training is necessary before significant improvements appear, and pre and post training procedures took the remaining two weeks. The ideal pattern of events is given in figure 14, and discussed more fully below.

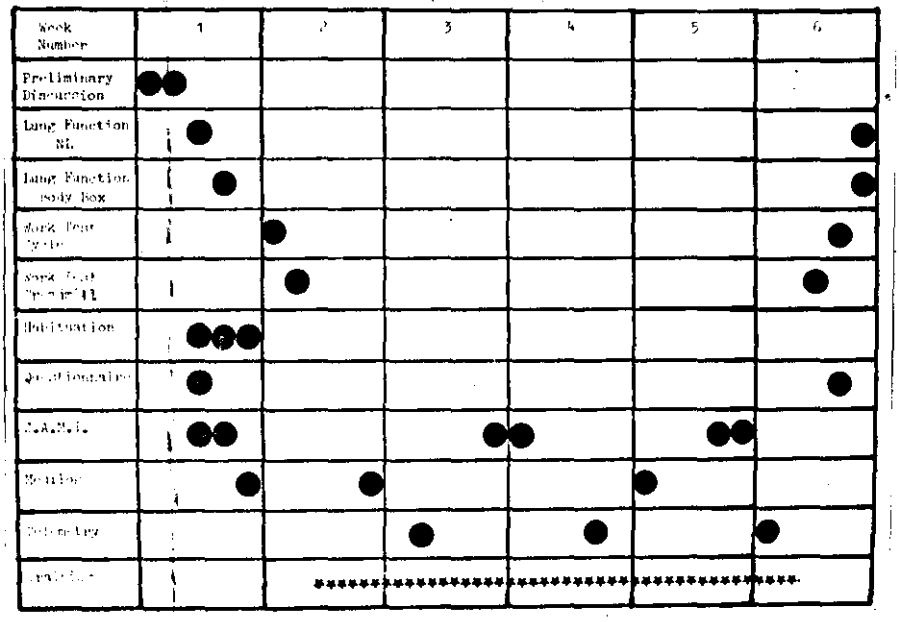


Figure 14. The ideal sequence of events.

Preliminary discussion

During this period patients were introduced to the concept behind the study, and were taken into the exercise physiology laboratory to see the equipment in use; the demands and limitations of exercise therapy were explained and time given for discussion with relatives. After agreeing to enter the study patients were taught, very briefly, to use the mouth-piece, valve box and nose-clip and asked to practise breathing through the apparatus as often as possible before the test procedures. Activity patterns and resting lung function were assessed during this

period also.

Lung function at rest

Two procedures were used to evaluate resting lung function. Firstly, the nitrogen decay curve devised by Cumming (1967) provided information on the efficiency of gas mixing in the human lung, and produces a curve which is readily interpreted and simply expressed by a single number. This single number expresses the percentage efficiency of ventilation, and varies from 70 - 90% in normal subjects to less than 20% in severely impaired patients. Secondly a constant volume plethysmograph (Pulmorex) gave information on lung volume and its sub-divisions, airways resistance and conductance and lung compliance.

Work tests

Patients were tested on two devices - an electronically braked bicycle ergometer (Elema-Schonander) and a variable speed and gradient treadmill (Glieze-Rambae) - on two occasions; the first immediately after the habituation programme, the second immediately post-training. Dummy tests on both devices were administered to the experimental group prior to the habituation programme; work time, rate and heat rate only are reported. Ergometer and treadmill tests were administered on successive days; on the first test occasion the ergometer was always the first device, but for the remaining tests the order was random. Tests occurred at least two hours after a meal with the time of day remaining fairly constant for each patient. A full 12 lead E. C. G. was recorded on the first and penultimate test session.

After work electrodes had been attached (RA-forehead; LA - V_4; RL - V_r4), patients rested for fifteen minutes; the procedures for the two devices were as follows:

Bicycle ergometer

The saddle height and position of the mouth-piece were checked for comfort; this was followed by a rest period. The last minute of rest was recorded on tape (Racal Thermionics), followed immediately by the first work load of 25W. Work remained constant over a wide pedalling range (30 - 120 r.p.m.), but patients were encouraged to pedal at the mechanically advantageous rate of 50 - 60 r.p.m., and checks were made to ensure the work rate was being maintained. Work loads were increased 25W every five minutes until the patient stopped pedalling, heart rate reached a target figure (220 - Age x 80/100), E.C.G. irregularities occurred, dyspnoea became intolerable or the patient failed to communicate by pre-arranged signals, Time was measured initially by a Centisecond timer (Griffin & George), and confirmed by computer analysis.

Treadmill

Patients selected their own walking pace and were allowed to use the hand rails to maintain balance. The tests started at zero gradient, and increased 6° every 5 minutes. In all other respects the treadmill test followed the pattern described for the ergometer test, the procedures being standardised through pre-recorded tape instructions.

Cardio-respiratory function was evaluated as follows:

Cardiac function

The work electrodes were fed into a rate-meter (Hewlett-Packard) which provided a digital output of rate based on 5 R-R intervals; an output from the rate meter to a multi-channel fibre-optic recorder (Cambridge) allowed continuous monitoring of the work electrocardiogram from the oscilloscope. In addition the E. C. G. was recorded for detailed analysis of rate changes and E. C. G. abnormalities.

Respiratory function

Three variables were recorded, namely respiratory rate and expired volume and gas concentrations in expired air. Volume was measured as follows. Patients breathed through a valve-box (Siebe-Gorman) of 45 ml. dead space; connected to the expiratory side was an insulated plastic tube, 3 cms.. bore and 60 cms. in length, leading to a 3 way tap, used for calibrating signals, and a heated pneumotachograph (Fleisch size 3). The pressure drop across the resistive element was detected by a pressure-transducer (Statham PM15) and amplified by a Devices amplification system. The reliability of the system has been described previously (page 47). The output from the Devices amplifier was electronically integrated and compared to a calibration signal to give tidal volume. The calibration signal was obtained as follows; a litre pump (Pulmorex) drew in expired air from a 10 litre.sample bag, and passed the known volume through a heat exchanger set at $37^{o}C$, via. insulated tubing and the three way tap to the pneumotachograph, the output being recorded for reference.

Analysis of expired air was achieved thus; a polythene sample tube, diameter 1 mm. length 3 metres, led from the upper surface of the Siebe-Gorman valve box to a respiratory mass spectrometer (Centronics). The delay time of the RMS was 0.5 sec., with a sample time of 30 m. sec. The device was calibrated as follows - zero gas Nitrogen, span gas atmosphere, check gas 15.5% O_2, 6.51% CO_2 (from Haldane analysis). Calibrations were performed before and after each work test. A block diagram of the entire system is given in figure 15.

The recorded output from the pneumotachograph and RMS were subsequently converted into digital form and processed by computer (Varian) to give V_T, Fe_{O2}, Fe_{CO2}, $Fe_{O2}ET$, $Fe_{CO2}ET$, V_{O2}, and V_{CO2} per breath and f_R, V_E, V_{O2} and V_{CO2} per minute. Details of the computer programme and methods of analysis are given in appendix D.

Habituation programme

Three days were spent on this part of the programme; the patients spent a short time - usually 20 minutes - in intermittent treadmill walking at zero grade. Work lasted for 1 minute, followed by 1 minute rest, and speed remained below that of the work test. Patients were encouraged to practise walking with longer strides, and without holding on to the rails.

Questionnaire

Measures of patient intra and extra version and neuroticism were obtained from Eysenck's personality inventory; form A was completed during the habituation period and form B immediately after training.

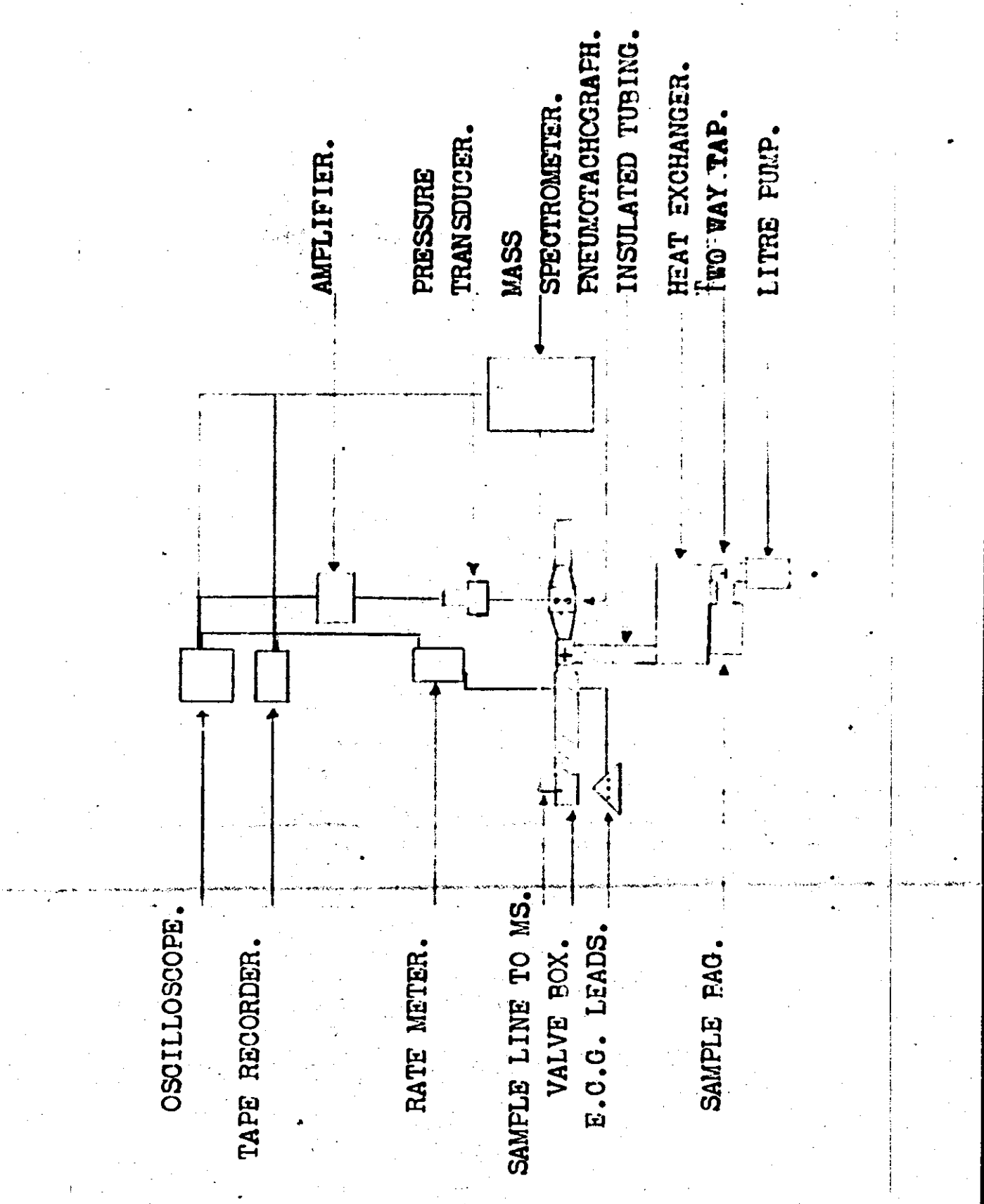


Figure 15. Diagram of work test equipment lay - out.

Habitual Physical Activity

Heart rate recordings were used to evaluate HPA; two devices were used. The first, the SAMI/HR (Tem Engineering), provides f_H over 48 hours. The second, the Medilog miniaturised tape recorder (Oxford Instruments) gives beat-by-beat f_H over 24 hours. At least three f_H recordings were attempted, one covering a week-end when supervised training did not take place.

Training

Both groups of patients took part in training programmes consisting mainly of intermittent treadmill walking but differing in duration and intensity. Training for group E was designed to last for 30 minutes a day, five times a week for about a month. The initial work load set, though sub-maximal was severe enough to raise f_H above the individually calculated minimum threshold levels (see figure 16); work rates were increased as patients tolerance improved, either through increasing speed or gradient or sometimes both together.

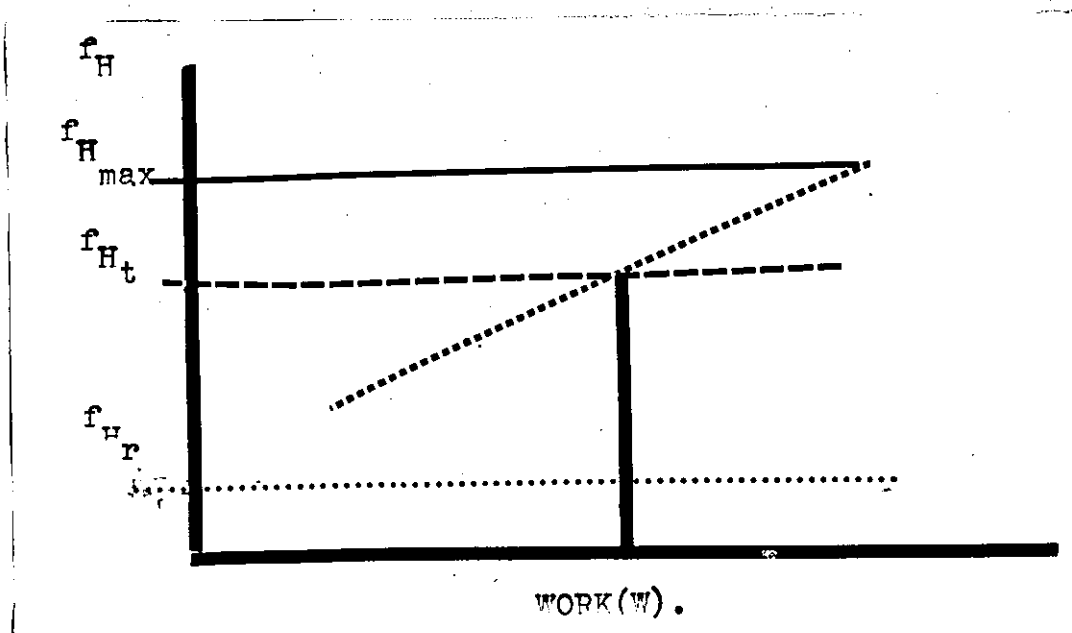


Figure 16. Setting the initial training work load.

Figure 17. Examples of methods used to record training data.

EXERCISE STUDY — TRAINING PROGRAMME

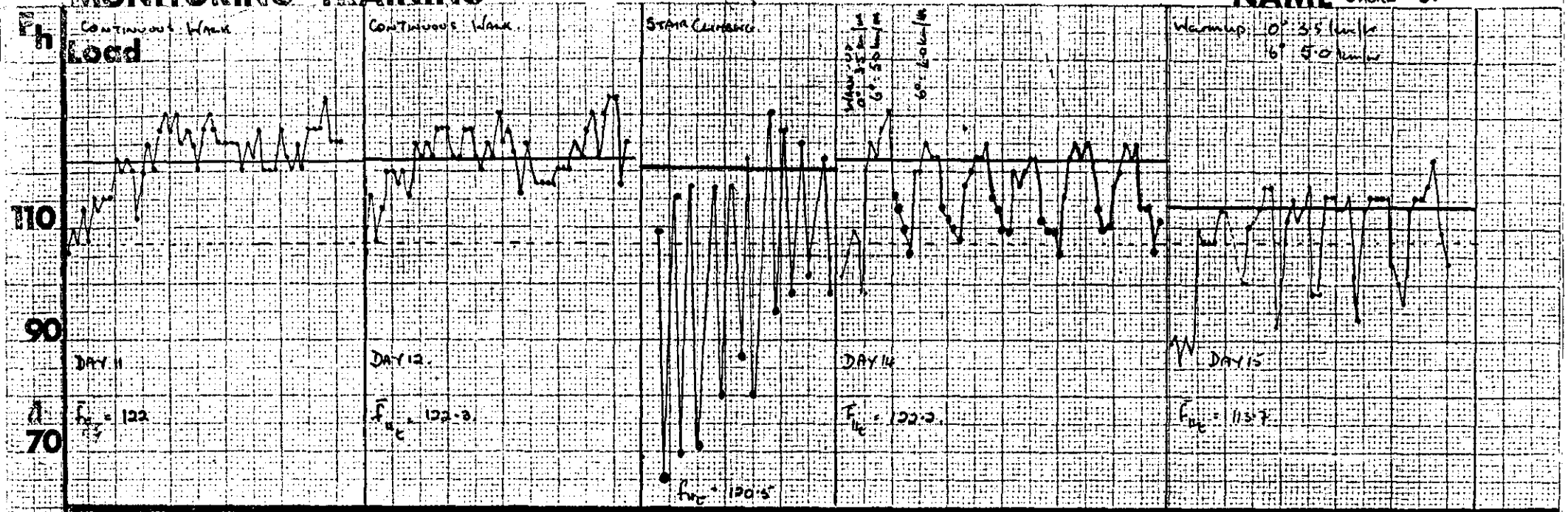
NAME: Bhore D. 1

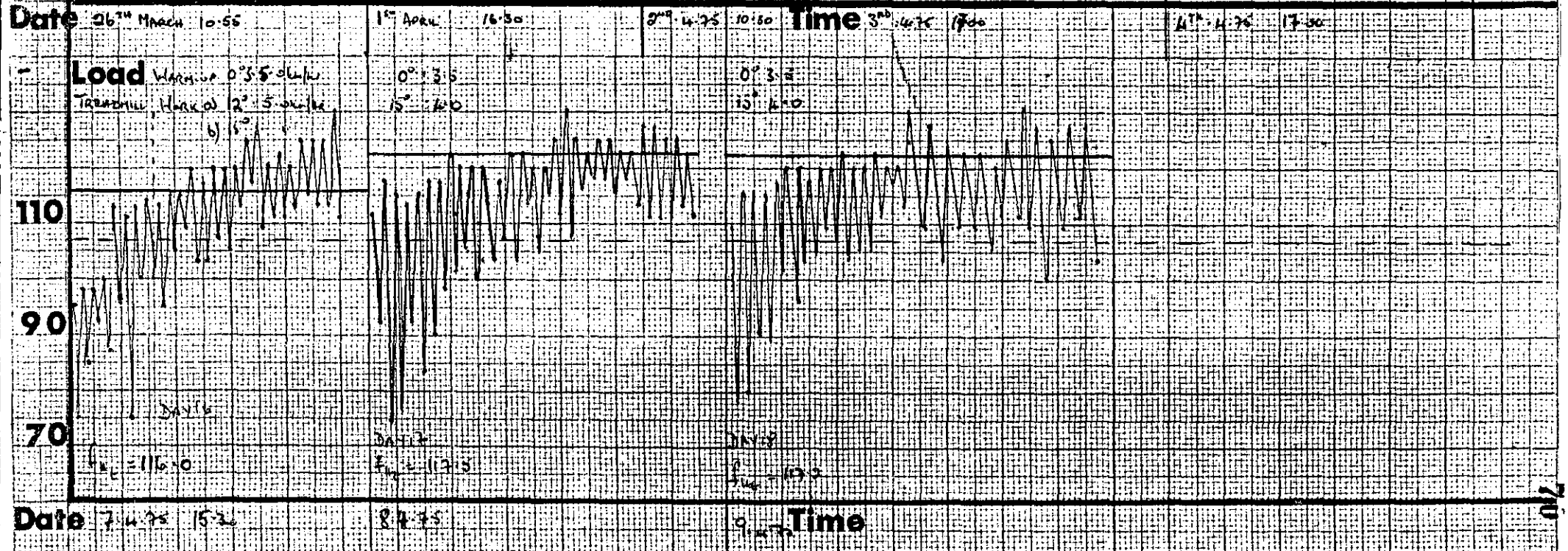
Date	Work			1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	TIME T	$\overline{F_h}$	Distance
12·3·75 10·30	TREAD MILL 6° : 4½ km/hr	a	Time W	5 min	5 min	5 min	6 min	5 min	5 min										31 min	1 88·3 2 100·7 3 103·1 4 104·9 5 108·3 6 110·7 109·7	57·4
		b	Time R	0	4 min	3 min	3 min	4 min	5 min										19 min		
		c	F_h W	6·8 6·7 6·8 6·8 6·8	6·1 5·8 6·1 5·9 5·9	6·1 5·6 6·0 5·8 5·6	5·8, 5·6 5·8, 5·7, 5·7,	5·8 5·3 5·4 5·6 5·6	5·6, 5·3 5·3, 5·4, 6·5,												
13·3·75	TREAD MILL 6° : 5·0 km/hr 6° : 4·0 km/hr 11⅛ min	a		5 min	5 min	5 min	5 min	5 min	5 min	5 min									30 min	2 106 3 111·1 4 111·7 5 114·2 6 113·3 7 113·3 111·7	68·1
		b		4 min	2 min	4 min	5 min	5 min	5 min	5 min									26 min		
		c		Warm up	98·4 105·3 109·1	109·1 109·1 111·1 113·2 113·2	105·3 109·1 113·2 113·2 117·6	109·1 117·6 115·4 111·1 113·6	109·1 113·2 113·2 113·2 113·6	109·1 111·1 115·4 113·6 113·6											
14·3·75	TREADMILL WARM-UP: 0° 6° 3° 5·0 2·0 WORK RECOVERY	a		5 min	5 min	5 min	5 min	5 min	5 min	5 min									30 min	2 109·4 3 112·0 4 113·4 5 113·7 6 110·8 7 112·8 112·0	65·7
		b		0	2 min	4 min	4 min	4 min	4 min	5 min									23 min		
		c	69·0	Warm up	100·0 109·1 109·1 113·2 113·6	109·1 113·2 111·1 111·1 115·4	105·3 115·4 113·2 115·4 117·6	109·1 112·2 113·2 120 113·2	105·3 113·2 107·1 116·4 113·2	109·1 113·2 113·2 113·2 115·4											
17·3·75	Stair climbing	a		5 min	1·45 min	2·0 min	2 min	2 min	2 min	2 min 15 sec	1·30 sec	2 min	2 min						21 min 30 sec	1 107·2 2 113·9	11, 11·, 4, 4·, 11·, 11·, 11, 11, 18½
		b			1 min	93·8 74·4 1·30 sec	98·4 1·0 min	98·4 1·30	101·9 99·4 1·30 sec	96·8 93·2 2 min	98·4 1 min	92·3 95·2 2·0 min							11 min 30 sec		
		c	84·5	Warm up	109·1 105·2	105·3 117·6	111·1 117·6	111·1 122·4	115·4 115·4	115·4 125·0	120·0 115·4	109·1 111·1	115·4 109·1								
18·3·75	3 MILE WALK IN GROUNDS	a		5	5	5	5	5	5	5	5	5	5	5	2				57 min	1 103·6, 12:83·3 2 109·9 3. 115·0 4. 113·2 5. 114·6 6. 112·5 7. 115·5 8 111·7 9 109·2 10 113·3 11 113·7 112·1	3 miles
		b																			
		c	72·3	98·8 104·1 109·1 105·3 111·1 113·2 111·1	109·1 105·3 111·1 113·2 111·1	113·2, 112 117·6 115·4 115·4	110·4 109·1 117·6 109·1 115·4	113·2 111·1 120·0 115·4 113·2	113·2 107·1 115·4 109·1 113·6	115·4 111·1 120·0 113·2 113·6	115·4 109·1 107·1 109·1 113·2	111·1 109·1 103·5 109·1 113·2	111·1 109·1 113·6 113·2 113·4	117·6 109·1 115·4 113·2 113·2	115·4 111·1						
19·3·75	3 MILE WALK IN GROUNDS	a		5	5	5	5	5	5	5	5	5	5	6	1				56 min	1 102·4 2 109·5 3 113·7 4 112·9 5 112·0 6 111·6 7 109·9 8 108·7 9 105·3 10 110·9 11 116·5 12 109·1 110·2	3 miles
		b																			
		c	78·9	87·0 102·5 105·3 111·1 105·3	109·1 109·1 111·1 111·1 107·1	115·4 115·4 113·2 113·2 111·1	109·1 115·4 113·2 113·2 111·1	111·1 113·2 111·1 109·1 115·4	115·4 107·1 109·1 111·1 111·1	113·2 111·1 109·1 109·1 109·1	111·1 109·1 109·1 109·1 109·1	108·3 109·1 107·1 113·2 109·1	105·3 109·1 113·2 113·2 109·1	120·0 109·1 120·0 120·0 113·2	109·1				57		

69

MONITORING TRAINING

NAME SHORE . D.





The duration and intensity of training for group C was less; training was designed to last for less than 10 minutes, including the recovery sessions and the intensity arranged so that minimum training thresholds were seldom exceeded. In addition loads remained constant throughout the programme.

Heart rate was monitored continuously during training sessions; in the laboratory the Hewlett-Packard to Cambridge recorder gave the electrocardiogram, and a telemeter (Parks Electronics) gave audial signals during sessions outside the laboratory. Rate was computed from the time interval of 10 R-R waves during the last 15 seconds of each minute of work and recovery, and recorded in graph form; the mean overall working heart-rate was used as a guide to maintain or increase the load. Figure 17 is an example of the way in which training data were recorded.

Statistical treatment

Because of the smallness of the groups and uncertainty about the distribution of the data, non-parametric methods were used to test the significance of the differences in pre and post training results of group E. The procedure chosen was Wilcoxon's matched-pairs signed rank test, a test which utilises information about both direction and magnitude of change and is said to be almost as powerful (95% efficiency) as Students 't' test for small samples (Siegel 1956).

The procedure is as follows: differences in pre and post training results for each individual are ranked and then the sign of that difference (plus for increase, minus for decrease) is attached. If H_o is true and the training has had no effect the sums of the positive and negative ranks would be similar. H_o is rejected if either of the sums is too small. An example follows in table 3 below.

Changes in working time before and after training

Subject	Pre	Post	d	Rank of d	Sign	Rank with less frequent sign (T)
1	990	1200	210	6	+	
2	354	384	30	2	+	
3	873	997	124	5	+	
4	398	390	8	1	-	1
5	412	486	74	4	+	
6	439	714	275	7	+	
7	470	526	56	3	+	
						T = 1

Table 3 Summary of the Wilcoxon matched-pairs signed-rank test procedure.

When n = 7, T must be equal or less than 2 if $H:_o$ is to be rejected at the 5% level; obviously $H:_o$ is rejected in the above examples It follows that where all subjects have changed in the same direction T = o, and therefore the group differences is significant (at the 2% level where = 7): it also follows that if only two subjects change in the opposite direction to the remainder, no matter how small the change, T must be at least = 3 and the group difference cannot be significant at the generally accepted minimal level of 5%. Thus with small groups the direction of change of each subject reveals at a glance results which are significant are the 2% level, and those which are not significant, leaving only those results where one subject is at odds with the group to be considered further.

CHAPTER SIX

The results - training, resting lung function and personality data

Introduction

Results are presented under four main headings. The first section deals with information concerning the training programme - the number of sessions (supervised and outside the programme), time and nature of exercise and the subjects' heart rate responses; assessment of habitual physical activity is also included. The second section is concerned with assessment of resting lung function, but because of a combination of timing and technical difficulties too few subjects were tested: therefore no meaningful group comparisons can be made, but the results obtained are included for interest. The third section reports very briefly on replies to the Eysenck Personality Inventory questionnaires. The final section presents data on pre and post training responses to the work tests.

Since the exercise test data are of paramount importance a more detailed description of presentation is appropriate and a separate chapter is devoted to these results. Firstly responses on treadmill and bicycle are compared; this is followed by a comparison of the pre-training responses of group E to a normal and then a COLD group studied by other workers. Changes in post-training responses of both groups are presented at the following levels:

a) aggregate level - i. e. sum of the responses of a variable for the whole work period.

b) average values - i. e. aggregate levels over total time.

c) maximal responses - usually achieved during last completed minute of work.

d) mean single breath - mean of all breaths.

e) predicted values - responses at standard tidal volumes 500 ml ($V_t^{0.5\frac{1}{2}}$) and 800 ml. ($V_t^{0.8\frac{1}{2}}$).

f) standard work - group E only; fifth minute at 0^0 grade on treadmill and 25 W on the bicycle.

Values are reported as follows:-

a) individual results are shown graphically within the text (figures 23 - 39) and in full in Appendix H;

b) group means appear in table 10; (pages 91-104) and in appendix H

c) percentage changes are given in the text.

d) gross negative-positive changes are found in table 11 (page 126-127)

Responses during training

Experimental group

Mean values for volume, duration and intensity of supervised training were 19.1 sessions (range 17 - 22), for 30.6 minutes per session (range 27.1 - 37.1 min.) at a heart rate of 111 b. min (range 97 - 127 b. min.). Five of the group exceeded calculated minimum f_{H_t} consistently, but one, E6 had low f_{H_t} during the first week of training which is reflected in a low mean training rate, and another, E4, displayed an unusual heart rate response during the work tests so that a meaningful f_{H_t} was difficult to set. However the mean f_{H_t} of the group exceeded the minimum level by 8 b. min.

Subjects were encouraged to be active outside the supervised programme and asked to record additional activity. Responses to both suggestions were variable. The group mean for this element of the programme was 17 sessions for an average of 16.3 minutes. Two subjects, E1 and E3 regularly spent more than half an hour in brisk walks of 1 - 3 miles in the hospital grounds; f_H recorded during some of these excursions revealed mean levels in excess of minimum f_{H_t}. Two others, E4 and E5 reported regular walks within the close confines of the hospital - corridors, stairs and the

gardens, but these trips were generally less than 15 minutes duration. The remaining three reported no additions to their normal daily activity pattern.

Attempts to monitor habitual physical activity met with mixed success; the level of understanding and reliability required of the subjects was not always available and the HPA data should be viewed accordingly. SAMI recordings of mean heart rate taken at the beginning and about half way through the programme revealed little change in sleeping f_H, mean values 68.7 - 69.6 b. min, but a small increase (7%) in waking f_H from 79.0 - 84.4 b. min. Output from tape recordings (Medilog) was more revealing in that short bursts of activity stood out from the general resting level as quite clear increases in rate, but there is insufficient data available for comparisons with SAMI recordings or for more detailed analysis.

All data is summarised in table 4 and figures 18 and 19 and an example of the Medilog output is given in figure 20.

The control group

Training for this group was deliberately less; sessions were fewer shorter and of low intensity. The average number of sessions was 14.6 (range 9 - 22) with a mean time of 5.0 min. (range 3.0 - 7.5 min.). Mean minimum f_{H_t} was set at 98 b. min. but the level achieved was only 91 b. min. just 4 b. min greater than the pre-working resting f_H of 87 b. min. One subject, C1, exceeded f_{Ht} but by less than 1 bt. min. and for the least number of training sessions (9).

Activity outside the programme varied widely. The mean number of sessions was 8, with a mean time of 17.0 min. per session. Three of the group reported no additional activity, but the remaining two, C2 and C3, spent more than half an hour in daily walks in the grounds. Unhappily HPA

TABLE 4: GROUP RESPONSES TO TRAINING (means and S.D.)

	HEART RATE (b.min)			SUPERVISED TRAINING	
	f_{H_r}	Min. f_{H_t} -	f_{H_t} ACHIEVED	SESSIONS	TIME (mins)
GROUP E	87.5	103.2	111.2	19.1	30.6
	11.7	7.8	11.0	1.95	3.5
GROUP C	86.9	97.9	90.7	14.6	5.0
	12.3	16.7	14.8	5.1	1.6

TABLE 5: GROUP RESPONSES TO TRAINING (means and S.D.)

	UNSUPERVISED ACTIVITY		HABITUAL PHYSICAL ACTIVITY: MEAN f_H (b.min)					
	SESSIONS	TIME (mins)	PRE-TRAINING	DAY	NIGHT	POST-TRAINING	DAY	NIGHT
GROUP E	17.3	16.3		79.0	68.7		84.4	69.6
				8.7	5.0		6.5	6.0
GROUP C	8.0	17.3						

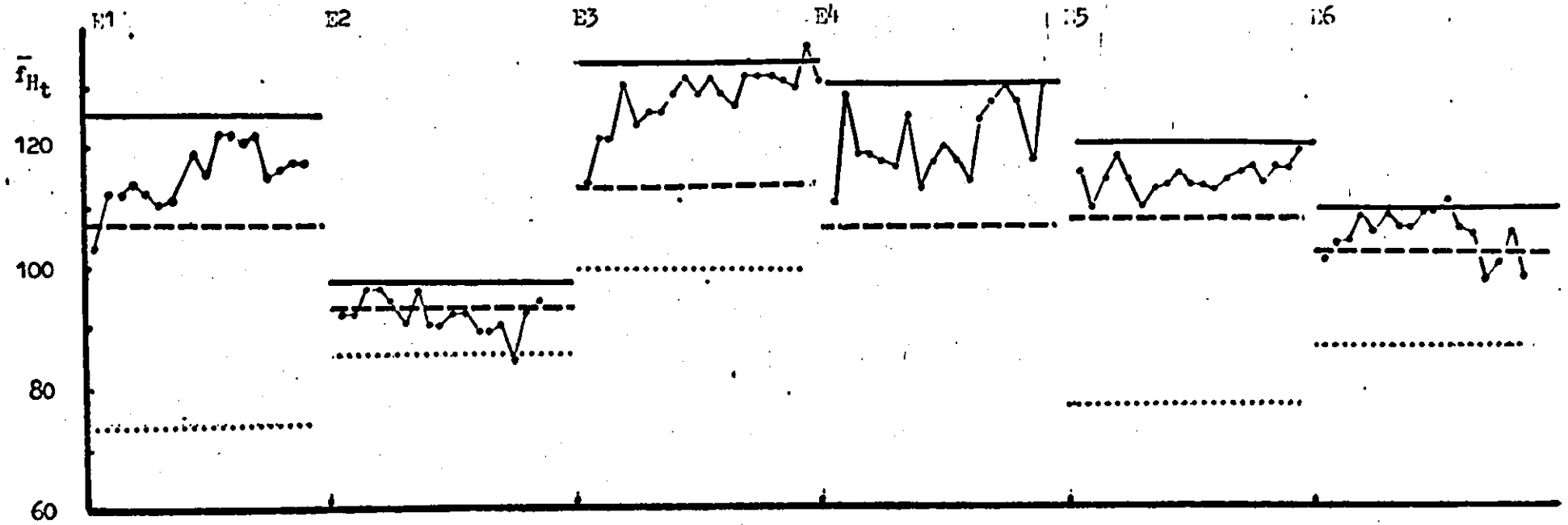


Figure 18. Individual heart rate responses during training (Group E).

(Key as for fig.19)

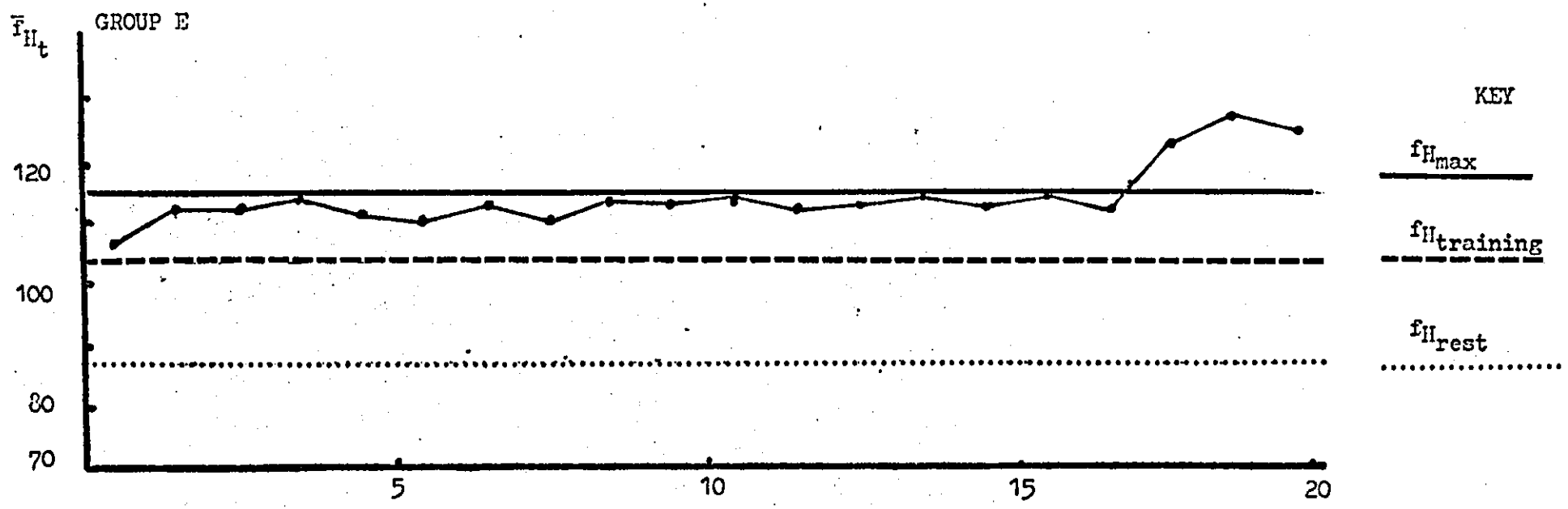
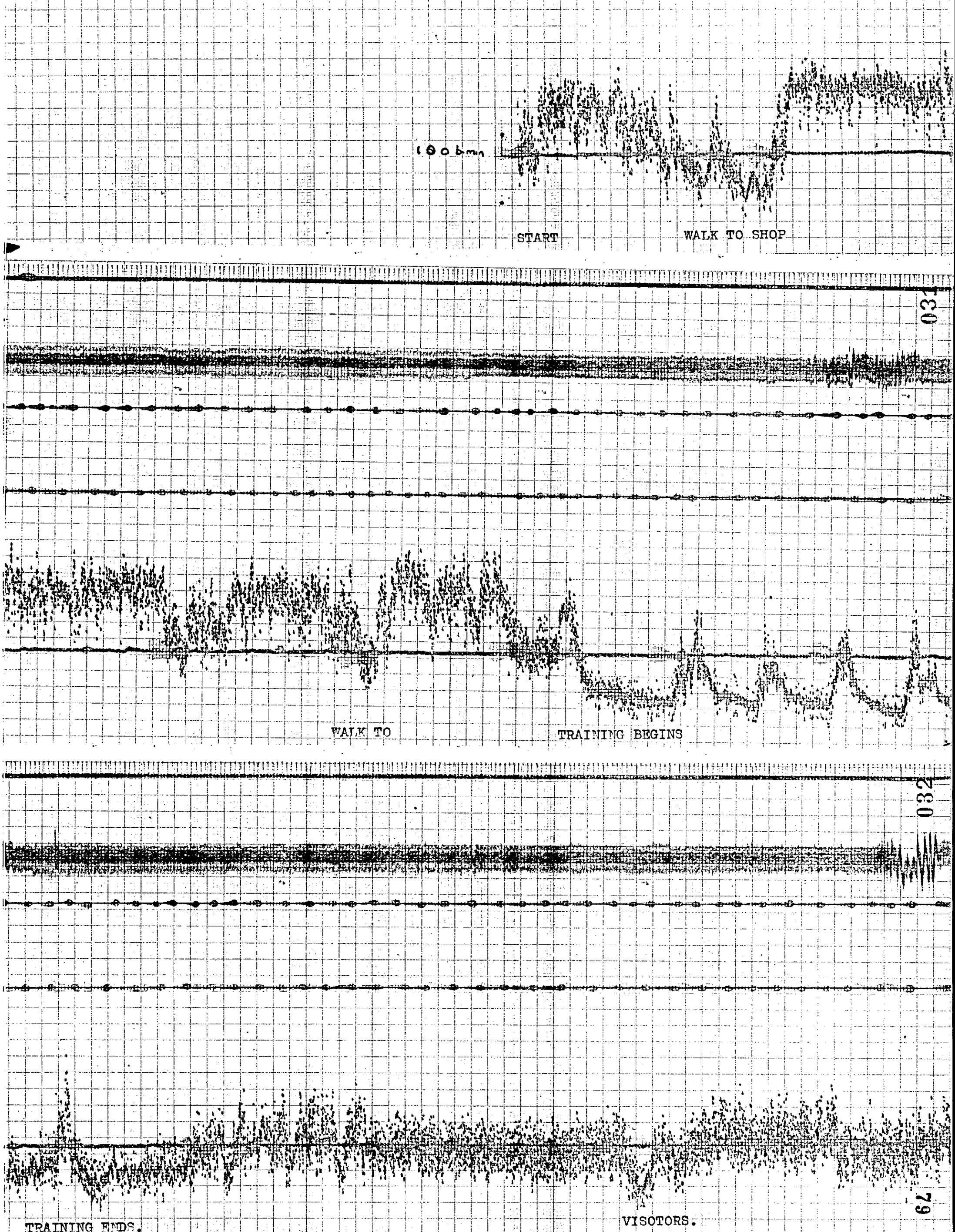


Figure 19 .Group means of heart rate during training (Group E).

Figure 20.


100bmn
START
WALK TO SHOP
034
WALK TO
TRAINING BEGINS
032
TRAINING ENDS.
VISOTORS.
62

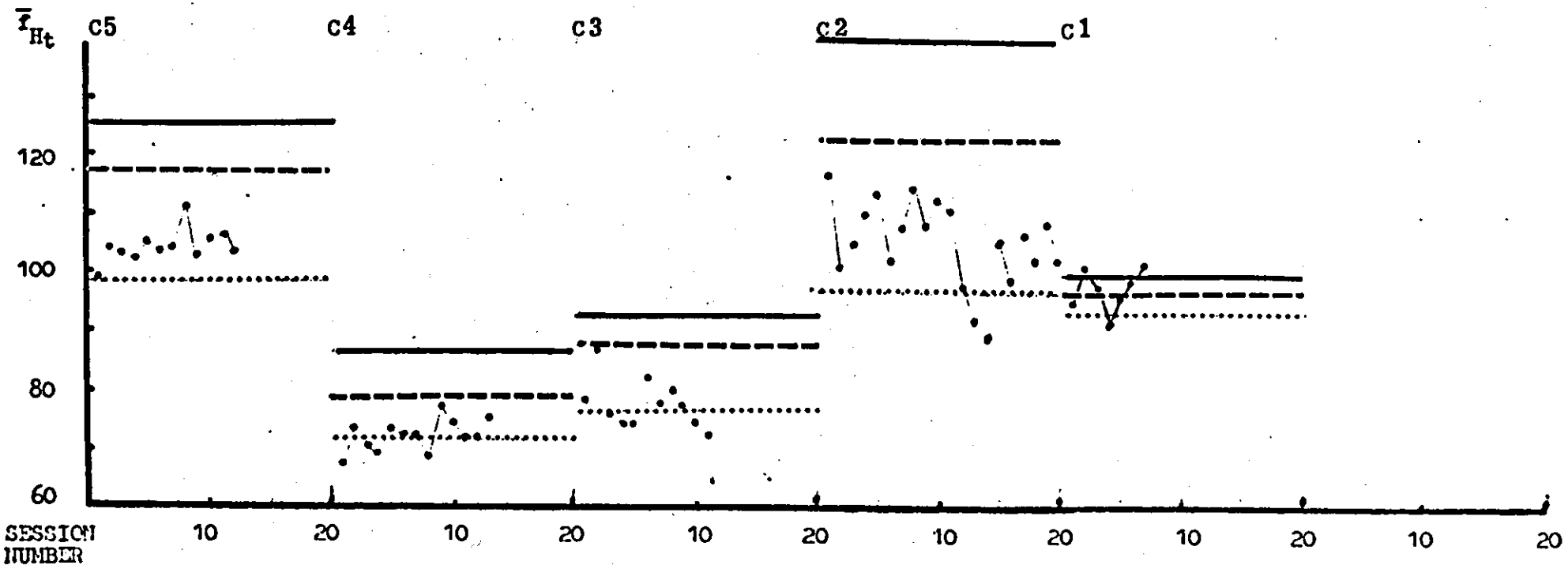


Figure 21. Individual heart rate responses during training (Group C).

(Key as for fig. 19)

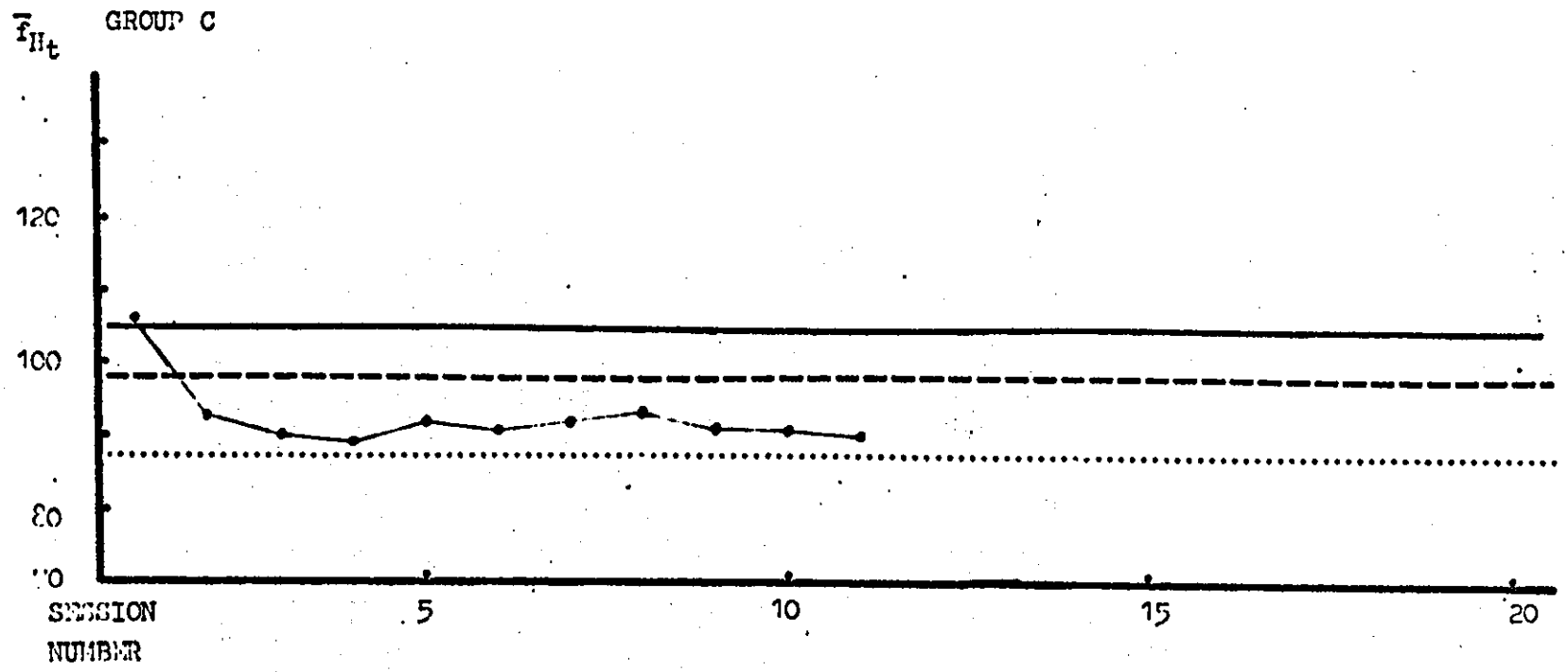


Figure 22. Group means of heart rate during training (Group C).

Table 6. RESTING LUNG FUNCTION OF GROUP E (n=4).

Body plethysmography.

PRE-TRAINING.

V C. L	R V. L	T L C. L	RV/ TLC. %	F R C. L	F E $V_{1.0}$ L	F V C L
3.25	4.93	8.18	59.8	6.31	0.883	3.10
0.54	1.20	1.09	8.8	1.05	0.315	0.33

POST-TRAINING.

3.01	6.01	9.02	66.2	7.07	1.12	2.79
0.87	1.49	0.66	12.3	1.18	0.42	0.69

Table 7 . RESTING LUNG FUNCTION OF PROUP E (n=3).

Nitrogen wash-out

PRE-TRAINING	TLC L	VT ml	VT_{CO2} ml	BOHR D.S.	VE L.min.BTPS	VCO_2 ml. min.	VD ml	VENT.EFFIC
	4.379	596	16.7	167	8.894	242.7	391	33.7
	0.726	175.6	6.51	81.2	1.337	28.9	89.9	4.04
	6.744	599	21.7		9.420	339	326.3	43.3
	1.270	234.0	8.14		1.227	11.3	81.0	7.57

TABLE 8: EYSENCK'S PERSONALITY SCORES (means and S.D.)

	N	E	L
Group E			
Pre-training	7.6	7.1	3.4
	4.08	3.24	2.64
Post-training	8.7	12.0	1.6
	4.34	5.69	1.4
Group C			
Pre-training	10.6	10.0	3.8
	5.77	2.92	2.18
Post-training	10.6	8.6	3.4
	8.14	3.65	1.82

recordings were obtained successfully only on two subjects in the early stages of training and hence no judgement can be made on changes in activity patterns.

Summaries of the data can be found in table 5 and figures 21 and 22.

Resting lung function

As previously mentioned data under this heading is incomplete. Means of the data that are available are presented without comment in tables 6 and 7 and the individual measures included in Appendix F.

Eysenck Personality Inventory

Results for group E showed a slight increase in factor N (neuroticism), mean values changing from 7.6 to 8.7, a marked increase in factor E (the intraextraversion contin) from 7.1 to 12.0 and a reduction in the lie detector score from 3.4 to 1.6. Only the increase in factor E is significant, p 0.05.

The scores for the control group showed no change in factor N, the mean value being 10.6, but unlike group E the intra-extraversion score fell from 10.0 - 8.6, and there was only a minor reduction in the lie detector score from 3.8 - 3.4

Results for both groups are summarised in table 8 and individual results are given in Appendix G.

CHAPTER SEVEN

The results : responses to work tests

Initial responses to work test

Comparison of the pre-training responses of group E on both treadmill and bicycle reveal that, in common with normal subjects values obtained on the treadmill are greater, even though on this occasion the working time is almost the same (a difference of 1.8% in favour of the treadmill) and $f_{H\ max}$ differs by only 1 b. min. Average values on the treadmill are seen to be a 16% higher $\dot{V}_E$ l. min. 14% faster f_R b. min. 28% greater $\dot{V}o_2$ l. min., and a 24% increase in $\dot{V}_{CO2}$ l. min. At maximum levels the treadmill resulted in higher values in $\dot{V}e$ (10%), f_R (8.3%), $\dot{V}o_2$ (12.6%); gas fractions, alveolar ventilation and dead space were also greater. Similar trends were seen at single breath, standard Vt, and standard work; the complete comparison is given in table 9 and the implications are considered in the discussion.

Comparison of group E with other groups

Reduced exercise tolerance is the universal finding of workers studying COLD patients. The degree of disability imposed by the physiological dysfunction is revealed clearly when comparisons are made with a group of normals of about the same age, but care must be taken if comparisons are to be meaningful since maximal responses are influenced by the exercise mode employed, as is revealed in the previous section. In this instance comparison of responses at similar work loads is the approach used, since the ventilatory impairment found in the patient group makes true $Vo_{2\ max}$ impossible and also because a common test device, the bicycle, enables work load to be calculated reliably.

TABLE 9

Comparison between treadmill and ergometer results before and after training

	PRE		POST	
AGGREGATE LEVELS				
TIME WORK	T	1.8	T	8.8
f_H	T	8.0	T	10.0
V_E	T	16.9	O	0.0
f_R	T	20.8	T	6.2
V_A	T	16.7	T	1.6
Vo_2	T	26.0	T	25.0
V_{Co_2}	T	19.4	T	5.4
RELATED TO TIME				
f_H	O	0.0	O	0.0
V_E	T	15.8	E	1.8
f_R	T	13.6	E	2.3
Vo_2	T	27.7	T	22.1
V_{Co_2}	T	24.0	T	7.8
MAXIMUM RESULTS				
GRADE/WORK				
f_H	T	1.3	E	2.6
V_E	T	10.0	E	0.6
f_R	T	8.3	T	4.0
V_T	T	2.1	O	0.0
Vo_2	T	22.5	T	21.0
V_{Co_2}	T	12.6	T	9.6
F_{Eo_2}	T	13.8	T	17.0
F_{ECo_2}	T	7.5	T	10.8
V_A	T	8.0	T	2.5
V_D	T	9.8	E	8.3

KEY

Letter indicates the device which provoked highest value; figures are the % age difference between the two devices.

	PRE				POST			
SINGLE BREATH								
V_T	O	0.0			T	1.6		
Vo_2	T	18.3			T	25.9		
V_{Co_2}	T	6.4			T	11.9		
F_{Eo_2}	T	17.8			T	20.0		
F_{ECo_2}	T	9.8			T	12.3		
V_A	T	1.3			T	3.6		
V_D	O	0.0			E	7.7		
STANDARD VT 0.5, 0.8								
Vo_2	T	5.2	T	8.6	T	18.0	T	15.7
V_{Co_2}	T	6.4	T	2.2	T	4.9	T	7.9
V_A	T	2.1	T	2.6	T	9.2	T	4.9
V_D	E	3.4	T	1.0	E	16.0	E	9.2
F_{Eo_2}	T	8.3	T	4.0	T	20.0	T	12.0
F_{ECo_2}	T	6.0	E	1.7	T	11.3	T	2.1
STANDARD WORK								
f_H	T	1.8			E	1.0		
V_E	T	5.2			E	5.2		
f_R	T	10.4			E	7.0		
V_T	E	2.4			T	3.1		
Vo_2	T	18.8			T	18.0		
V_{Co_2}	T	6.4			T	6.6		
F_{Eo_2}	T	11.3			T	20.0		
F_{ECo_2}	T	4.1			T	10.7		
V_A	T	5.1			T	1.0		
V_D	T	6.1			E	17.3		

The differences revealed at a work load of approximately 50 W (patients = 57.1 W, normals = 49 W (Higgs, Clode, McHardy, Joues & Campbell 1966). are as follows:

a) Ventilation is 19% lower (21.99 - 17.3 l. min BTPS.); f_R is 31% faster (25.7 - 17.8 b. min); consequently V_t is smaller (849 - 1530 ml).

b) Heart rate of 15% higher (115 - 98 b. min) and thus work pulse is lower.

c) $\dot{V}o_2$ is 35% lower (0.725 - 0.980 l. min. STPD), but when related to body weight the difference almost disappears (11.5 - 12.2 ml. kg. min.). Predictably oxygen pulse is lower.

d) $\dot{V}_{co2}$ is less by 27% (0.640 - 0.882 l min. STPD), but again this difference is abolished when body weight is considered, the values being 10.1 and 11.0 ml. kg. min. Even so these values are interesting since a similar CO2 excretion results from lower ventilation.

The important point here is that a minor increase in work output for normal subjects represents the maximal bicycle ergometer work rate for five of the seven patients, and underlines the severe handicap afflicting COLD sufferers.

Further evidence that this group is particularly impaired comes from comparison with a group of 16 COLD patients (mean age 60) studied by Spiro et al (1966). Differences in mean maximal bicycle work are as follows:

a) 21% lower work rate (57.1 - 70.6 W).

b) 20% lower $f_{H\ max}$ (114 - 138 b. min).

c) 9% lower $\dot{V}e$ (21.99 - 25 l. min BTPS)

d) Identical f_R (26. b. min).

e) 18% reduction in Vt (850 - 1035 ml).

f) 15% reduction in $\dot{V}o_2$ (0.725 - 0.839 l. min. STPD)

g) Similar ovygen equivalent (31.5 - 31.2 ml. 0_2 l. min.)

h) Similar RQ. (0.88 - 0.85)

Post-training responses to work tests

The values quoted are percentage changes followed by the actual difference between the means in brackets; individual changes are shown graphically and the group responses are found in the accompanying table 10.

Treadmill Group E

a) Work

Every subject increased treadmill walking time, and three were able to increase the maximum gradient achieved. Aggregate working time increased significantly by more than 28% (163 secs, $p < 0.02$), and the gradient increased by 23% (2.2^{O}).

b) Heart Rate

Except for the total number of heart beats, changes in heart rate were small - in the order of 2.3% (2 - 3 b. min)-, and none were significant. Although there was an increase of 21% (224 b.) in aggregate f_H, and in spite of increased work, average f_H over the total working period, symptom limited $f_{H\ max}$ and f_H at standard work all fell slightly. Individual heart rates either remained identical (4 cases) or fell (the remaining three).

c) Ventilation

The increased work produced generally small changes in ventilatory performance, but results at standard V_t $^{0.5\ l}$ and standard work reveal interesting changes in the effectiveness of ventilation.

Aggregate $\dot{V}e$ rose 5% (9.6 l.) f_R by 4% (8b.) and Vt 1% (11 ml); however when related to total time $\dot{V}e$ and f_R fell 14% (2.8 l. min) and 3% (1 be. min) resulting in a small - 2% - increment in Vt (15 ml). At standard

TABLE 10.

The means of responses to work tests before and after training.

* = $p < 0.05$

** = $p < 0.05$

Means, S.D.'s, percentage changes and levels of significance are given in full in Appendix H, pages 226-261.

GROUP E BICYCLE ERGOMETER.

AGGREGATE VALUES

TIME (Secs.)	WORK (W.)	fH (b.)	VE (L.BTPS.)	VA (L.BTPS.)	fR (b.)	VO_2 (L.STPD)	VCO_2 (L.STPD)
562.3	385.4	960.3	171.4	115.6	196.9	7.155	5.924
671.1*	512.8*	1135.3*	217.40	150.9	243.3*	8.779	7.831

RELATED TO TIME

fH (b.min.)	$\dot{V}E$ (l.min.BTPS)	fR (b. min.)	$\dot{V}O_2$ (l.min.STPD)	$\dot{V}CO_2$ (l.min.STPD)	VEO_2 (ml.100 ml)	$VECO_2$ ml.100 ml)
101.3	16.73	21.5	0.659	0.537	2.66	3.38
100.8	17.35	22.1	0.670	0.611	2.81	2.92

MAXIMAL RESPONSES:

TIME (Secs.)	WORK (W.)	fH (b/m)	fR (br.mi	$\dot{V}E$ (L.BTPS)	VT (ml.BTPS)	$\dot{V}O_2$ (l.min.STPD)	$\dot{V}CO_2$ (l.min.S
562.2	57.1	113.9	25.71	21.99	849.8	0.725	0.640
671	67.9	115.1	26.0	23.39	890.7	0.762	0.697

FE_{O_2} (%)	FE_{CO_2} (%)	FEO_2ET (%)	$FECO_2ET$ (%)	RQ	$\dot{V}A$ (L.min.BTPS)	$\dot{V}D$ (l.min.BTPS)
3.85	3.46	5.90	5.10	0.88	14.91	7.07
3.92	3.64	5.83	5.39	0.95	15.9	7.44

SINGLE BREATH LEVELS (ml ATPS except where shown otherwise)

VT (l)	VO_2	VCO_2	VD	VA	FEO_2 (%)	$FECO_2$ (%)	$PETCO_2$ (mm.Hg)
0.807	31.27	27.00	269.3	537.9	3.67	3.13	40.61
0.802	31.49	27.84	257.6	544.2	3.51	3.37	43.26

PREDICTED VALUES (ml ATPS unless stated otherwise)

$VT^{0.81}$ VO_2	VCO_2	VD	VA	FEO_2 (%)	$FECO_2$ (%)	$VT^{0.81}$ VO_2	VCO_2	VD	VA	FEO_2 (%)	$FECO_2$ (%)
16.63	13.54	192.4	307.5	3.28	2.79	31.54	26.62	267.7	532.3	4.14	3.46
15.47	15.61	182.1	317.9	2.89	2.91	29.61	28.61	255.0	546.2	3.7	3.74

STANDARD WORK (25 W)

fd (b.min)	$\dot{V}E$ (l.min.BTPS)	fR (b.min)	V_T (ml)	$\dot{V}O_2$ (l.min STPD)	$\dot{V}CO_2$ (l.min. STPD)	RQ
95.4	16.772	22.3	760.2	0.520	0.464	0.84
95.6	15.51	21.4	752.7	0.503	0.438	0.92

STANDARD WORK (25 W)

FEO_2 (%)	$FECO_2$ (%)	FEO_2 (%)	$FECO_2ET$ (%)	$\dot{V}D$ (l.min.BTPS)	$\dot{V}A$ (l.min.BTPS)	PET_{CO2} (mm Hg)	$PETO_2$ (mm Hg)
3.69	3.28	5.89	4.93	5.59	11.2	35.37	107.7
3.66	3.43	5.77	5.09	5.03	10.47	35.40	109.5

GROUP E TREADMILL.

AGGREGATE VALUES

TIME (Secs)	fH (bt)	VE (l.BTPS)	VA (l.BTPS)	fR (b)	VO_2 (l.STPD)	VCO_2 (l.STPD)
572.6	1043.9	206.14	138.4	248.9	9.333	7.346
736.1 **	1268.3	215.73	153.3	263.7	10.845	8.334 **

RELATED TO TOTAL TIME

fH (b.min)	VE (l.min.BTPS)	fR (b.min)	VO_2 (l.min.STPD)	VCO_2 (l.min.STPD)	VEO_2 (ml.100 ml)	$VECO_2$ (ml.100 ml)
101.9	19.88	24.9	0.908	0.707	2.21	2.85
99.9	17.05	21.5	0.825	0.662	2.30	2.66

MAXIMAL RESULTS

TIME (Secs)	GRADE (deg.)	fH (b.min)	fR (b.min)	$\dot{V}E$ (l.min BTPS)	VT (ml)	$\dot{V}_{O2}$ (l.min STPD)	$\dot{V}_{CO2}$ (l.min STPD)
572.5	9.4	115.4	28.0	24.14	868.5	0.956	0.752
736.1 **	11.6	112.1	27.1	23.03	888.4	0.928	0.764

MAXIMAL RESULTS

FE_{O2} (%)	FE_{CO2} (%)	FE_{CO2} Max. (%)	FE_{CO2} Max. (%)	RQ	$\dot{V}A$ (l.min STPD)	$\dot{Q}$ (l.min BTPS)
4.70	3.741	7.29	5.57	0.79	16.2	7.83
4.72	4.07	7.02	5.76	0.84	16.2	6.76

MEAN SINGLE BREATH (ml ATPS unless stated otherwise)

VT (l)	VO_2	VCO_2	VD	VA	FE_{O2} (%)	FE_{CO2} (%)	PET_{CO2} (mm Hg)	PET_{O2} (mm Hg)
0.817	38.27	28.83	271.9	545.1	4.50	3.46	44.61	93.5
0.815	40.27	31.61	237.7	577.7	4.65	3.83	46.46	93.4

PREDICTED VALUES (ml ATPS unless otherwise stated)

$VT^{0.51}$

V_{O2}	V_{CO2}	VD	VA	FE_{O2} (%)	FE_{CO2} (%)
18.89	14.46	185.8	314.2	3.84	2.98
20.76	16.43	152.9 *	350.7 *	4.05	3.28

PREDICTED VALUES (ml ATPS unless otherwise stated)

$VT^{0.81}$

V_{O2}	V_{CO2}	VD	VA	FE_{O2} (%)	FE_{CO2} (%)
36.10	27.23	269.7	546.4	4.48	3.40
37.60	31.07	231.7	574.3	4.66	3.84

STANDARD GRADE (0°)

fH (b.min)	fR (b.min)	$\dot{V}E$ (l.min.BTPS)	VT (ml)	$\dot{V}_{O_2}$ (l.min.STPD)	$\dot{V}_{CO_2}$ (l.min.STPD)	RQ
97.2	24.9	17.77	0.742	0.664	0.496	0.77
94.5	19.9 **	14.66	0.777	0.588	0.470	0.78

STANDARD GRADE (0°)

FEO_2 (%)	FE_{CO2} (%)	FEO_2ET (%)	$FE_{CO2}ET$ (%)	$\dot{V}D$ (l.min.BTPS)	$\dot{V}A$ (l.min.BTPS)	PA_{CO2} (mm.Hg)	PAO_2 (mm.Hg)
4.43	3.41	6.97	5.13	5.94	11.8	36.5	102.5
4.79	3.84	6.96	5.34	4.16 **	10.6	38.2	102.1

GROUP C BICYCLE ERGOMETER.

SINGLE BREATH VALUES (ml ATPS unlessotnerwise stated)

V_T (l)	Vo_2	V_{Co_2}	V_D	V_A
0.931	39.26	37.04	268.3	662.4
0.845	33.71	29.45	264.6	579.9

F_{Eo_2}	F_{ECo_2}	$P_{ET_{Co_2}}$	$P_{ET_{o_2}}$
4.07	3.82	46.36	96.8
3.87	3.36	41.73	97.5

PREDICTED VALUES (ml ATPS unless show otherwise)

$V_T^{0.51}$

V_{O_2}	$V_{C_{O_2}}$	V_D	V_A	$F_{E_{O_2}}$ (%)	$F_{E_{C_{O_2}}}$ (%)
18.38	14.31	200.5	295.1	3.07	2.59
16.38	14.24	188.4	311.6	3.28	3.02

$V_T^{0.81}$

V_{O_2}	$V_{C_{O_2}}$	V_D	V_A	$F_{E_{O_2}}$ (%)	$F_{E_{C_{O_2}}}$ (%)
34.68	27.44	259.8	541.1	3.77	3.27
32.54	29.42	250.5	543.5	3.84	3.62

AGGREGATE VALUES

Time (secs)	Work (W)	f_H (b)	$\dot{V}_E$ (L.BTPS)	V_A (L.BTPS)	f_R (b)	V_{O_2} L.STPD	V_{CO_2} L.STPD
398.2	234.3	633.1	122.59	88.924	131.8	5.458	5.155
350.2	192.4	545.1	96.107	79.096	112.8	4.071	3.606

RELATED TO TIME

f_H (b.min)	$\dot{V}_E$ (L.MIN BTPS)	f_R (b.min)	$\dot{V}_{O_2}$ (L.MIN STPD)	$\dot{V}_{CO_2}$ (L.MIN STPD)	$V_{E_{O_2}}$ (ml. 100ml)	$V_{E_{CO_2}}$ (ml. 100ml)
99.3	18.298	20.4	0.750	0.662	2.52	2.88
95.1	15.83	18.9	0.625	0.547	2.66	3.06

MAXIMAL VALUES

Time (secs)	Work (W)	f_H (b.min)	f_R (b.min)	$\dot{V}_E$ (L.min BTPS)	V_T (ml)	$\dot{V}_{O_2}$ (L.min STPD)	$\dot{V}_{CO_2}$ (L.min STPD)
	45	107.2	22.4	20.9	951.5	0.724	0.698
	45	100.7	20.6	17.9	880.4	0.585	0.530

MAXIMAL VALUES

$F_{E_{O_2}}$ (%)	$F_{E_{CO_2}}$ (%)	$F_{E_{O_2ET}}$ (%)	$F_{E_{CO_2ET}}$ (%)	RQ	$\dot{V}_A$ (L.min BTPS)	$\dot{V}_D$ (L.min BTPS)
4.18	4.05	6.02	5.64	0.96	14.9	6.06
3.97	3.56	5.79	5.07	0.90	12.3	5.68

GROUP C TREADMILL.

AGGREGATE VALUES

Time (secs)	f_H (b.)	V_E (L.BTPS)	V_A (L.BTPS)	f_R (b.)	V_{O_2} (L.STPD)	V_{CO_2} (L.STPD)
477	821.1	134.88	89.41	159.7	6.527	5.480
495.7	821.8	154.11	113.08	171.7	6.895	6.616

RELATED TO TIME

f_H (b.min)	V_E (L.min BTPS)	f_R (b.min)	$\dot{V}_{O_2}$ (L.min STPD)	$\dot{V}_{CO_2}$ (L.min STPD)	$V_{E_{O_2}}$ (ml.100 ml)	$V_{E_{CO_2}}$ (ml.100 ml)
95.7	17.012	21.6	0.791	0.656	2.18	2.64
92.1	18.153	21.5	0.791	0.737	2.31	2.51

MAXIMAL VALUES

Time (secs)	Work (W)	f_H (b.min)	f_R (b.min)	$\dot{V}_E$ (L.min BTPS)	V_T (ml)	$\dot{V}_{O_2}$ (L.min STPD)	$\dot{V}_{CO_2}$ (L.min
	45	107.2	22.4	20.9	951.5	0.724	0.698
	45	100.7	20.6	17.9	880.4	0.585	0.530

MAXIMAL VALUES

$F_{E_{CO_2}}$ (%)	$F_{E_{O_2ET}}$ (%)	$F_{E_{CO_2ET}}$ (%)	RQ	$\dot{V}_A$ (L.min BTPS)	$\dot{V}_D$ (L.min BTPS)
4.19	7.09	5.87	0.85	15.7	5.7
4.21	6.23	5.87	0.96	15.4	6.1

SINGLE BREATH VALUES (ml ATPS unless otherwise stated)

V_T (l)	V_{O_2}	V_{CO_2}	V_D	V_A
0.822	39.25	32.84	236.1	586.0
0.863	37.68	36.30	239.2	623.7

SINGLE BREATH VALUES (ml ATPS unless otherwise stated)

F_{EO_2} (%)	F_{ECO_2} (%)	$P_{ET_{CO_2}}$ mm.Hg.	$P_{ET_{O_2}}$ mm.Hg.
4.07	3.82	46.36	96.8
3.87	3.36	41.73	97.5

PREDICTED VALUES (ml ATPS unless otherwise stated)

V_T 0.51

V_{O_2}	V_{CO_2}	V_D	V_A	$F_{E_{O_2}}$ (%)	$F_{E_{CO_2}}$ (%)
19.25	16.64	178.1	321.9	3.78	3.18
17.11	15.91	176.4	323.6	3.46	3.17

PREDICTED VALUES (ml ATPS unless otherwise stated)

V_T 0.8[illegible]

V_{O_2}	V_{CO_2}	V_D	V_A	$F_{E_{O_2}}$ (%)	$F_{E_{CO_2}}$ (%)
38.38	32.24	227.7	571.2	4.92	4.12
33.78	31.41	234.2	565.9	4.10	3.78

FIGURES 23- 30.

Graphical Summary of Responses to

Work Tests Before and After Training

Explanatory note:

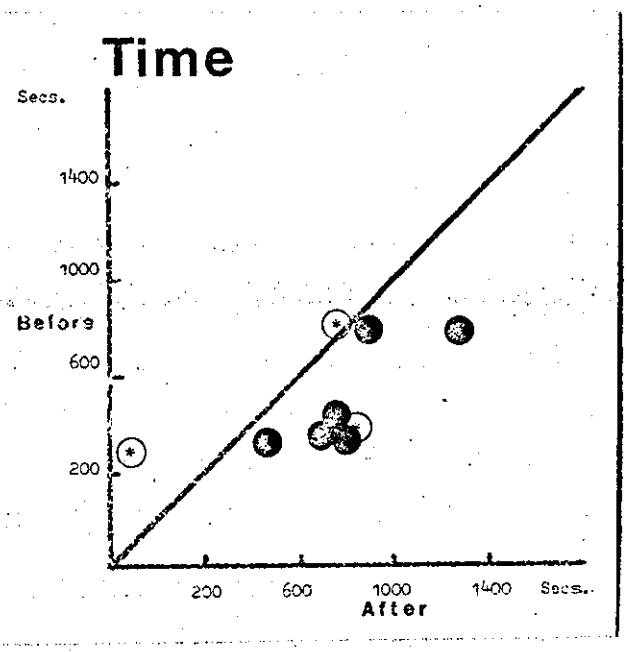
The ordinate represents pre-training data the abscisson post-training data.

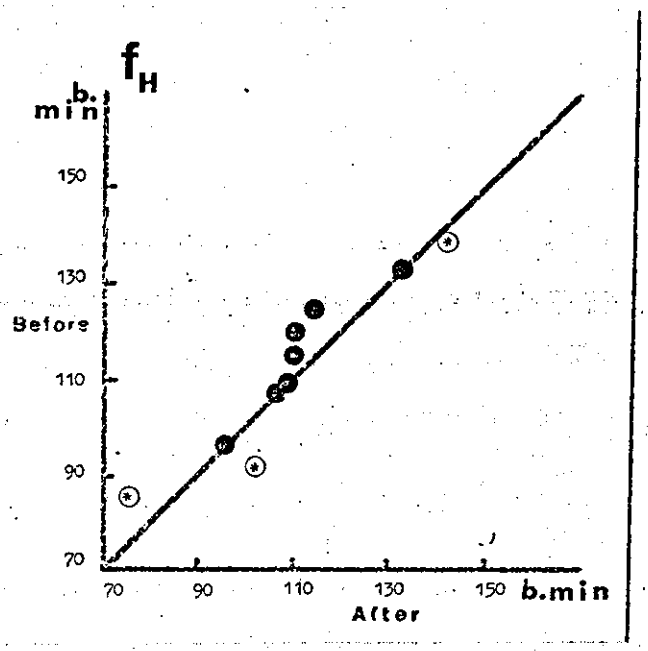
(*) = control group

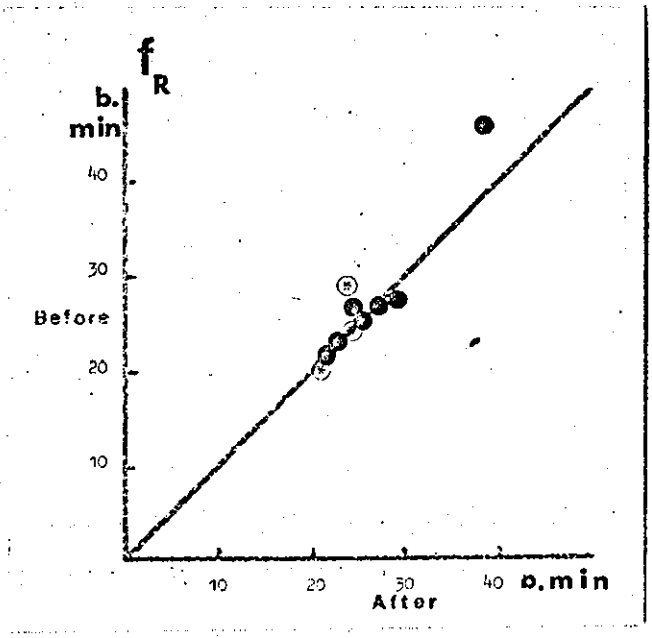
● = experimental group

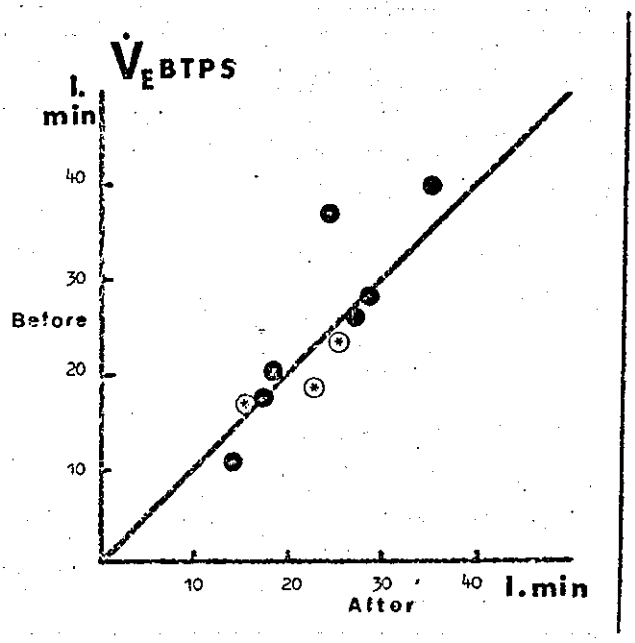
The diagonal represents identical responses before and after training; positions to the left of the diagonal indicate a reduction in post-training response and vice-versa.

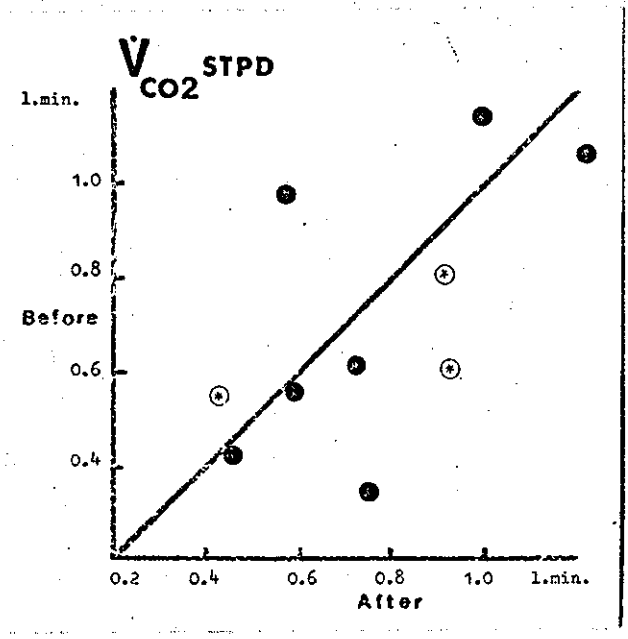
TREADMILL: MAXIMUM VALUES

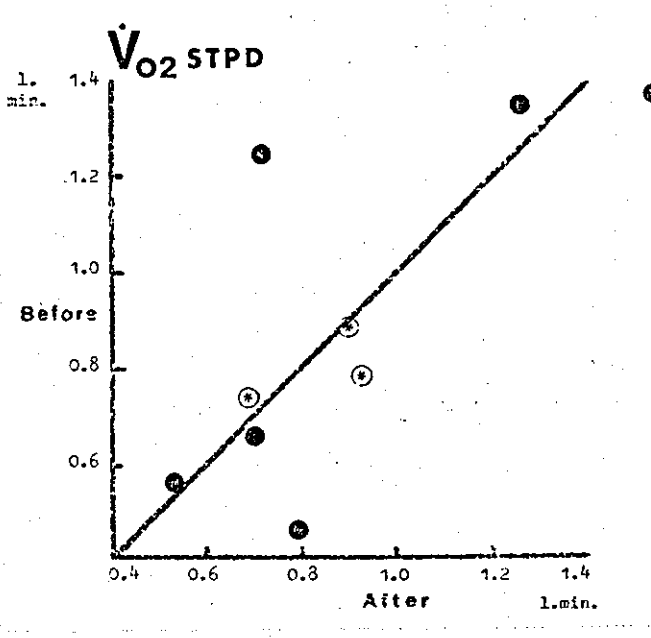

Time
Secs.
1400
1000
Before
600
200
200
600
1000
1400
Secs.
After

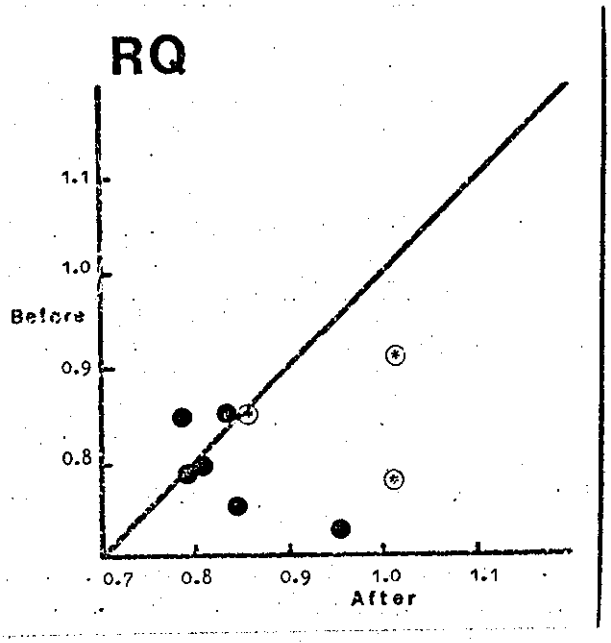

f_H
b.
min
150
130
Before
110
90
70
70
90
110
130
150
b.min
After

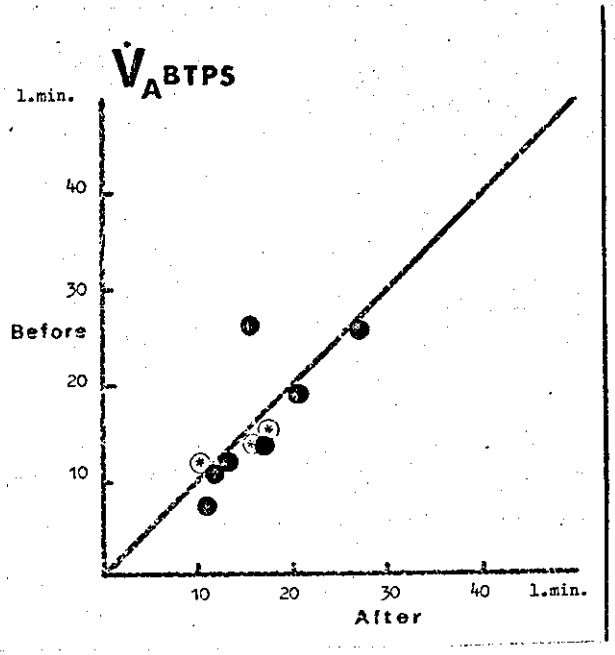

f_R
b.
min
40
30
Before
20
10
10
20
30
40
b.min
After

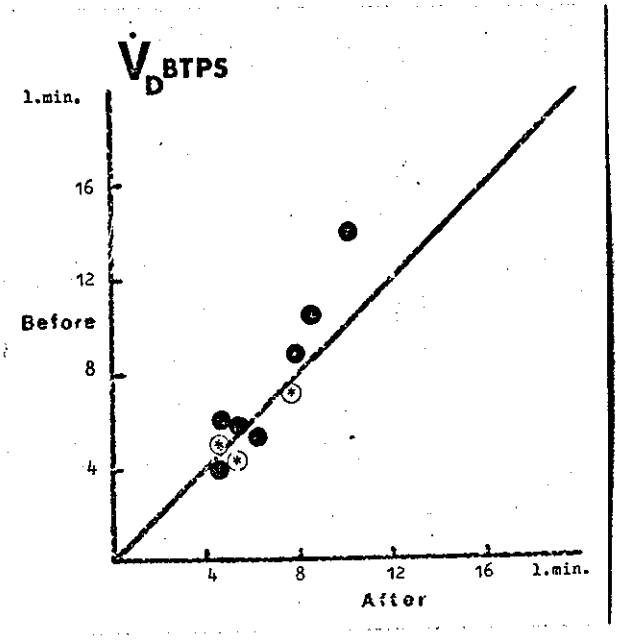

$\dot{V}_E$ BTPS
l.
min
40
30
Before
20
10
10
20
30
40
l.min
After


$\dot{V}_{CO2}$ STPD
l.min.
1.0
0.8
Before
0.6
0.4
0.2
0.4
0.6
0.8
1.0
l.min.
After

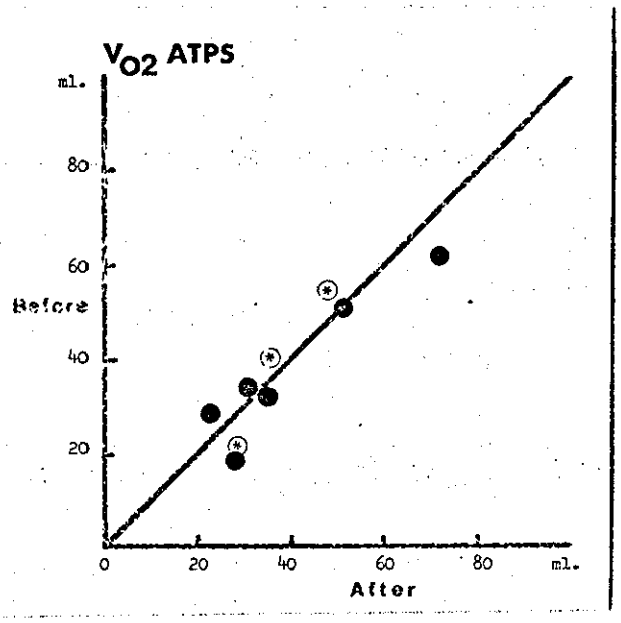

$\dot{V}_{O2}$ STPD
l.
min.
1.4
1.2
1.0
Before
0.8
0.6
0.4
0.6
0.8
1.0
1.2
1.4
l.min.
After

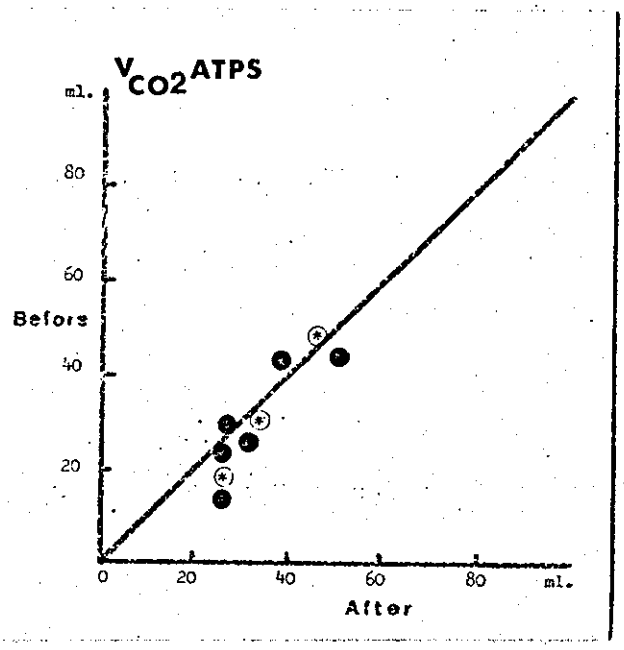

RQ
1.1
1.0
Before
0.9
0.8
0.7
0.8
0.9
1.0
1.1
After

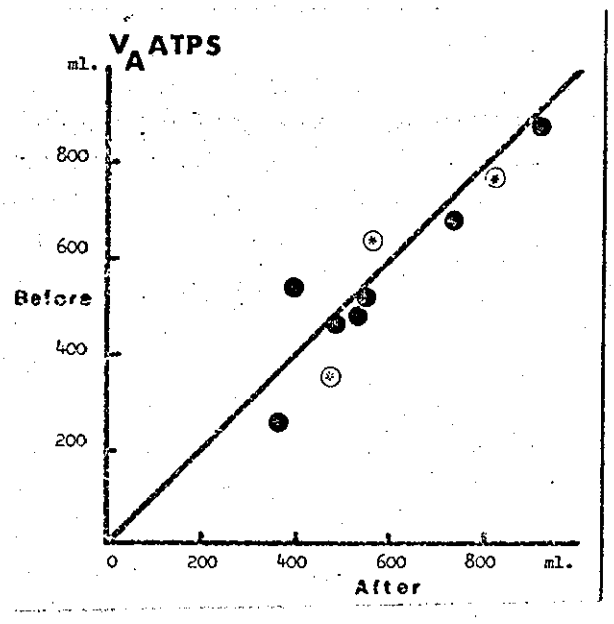

$\dot{V}_A$BTPS
l.min.
40
30
Before
20
10
10
20
30
40
l.min.
After

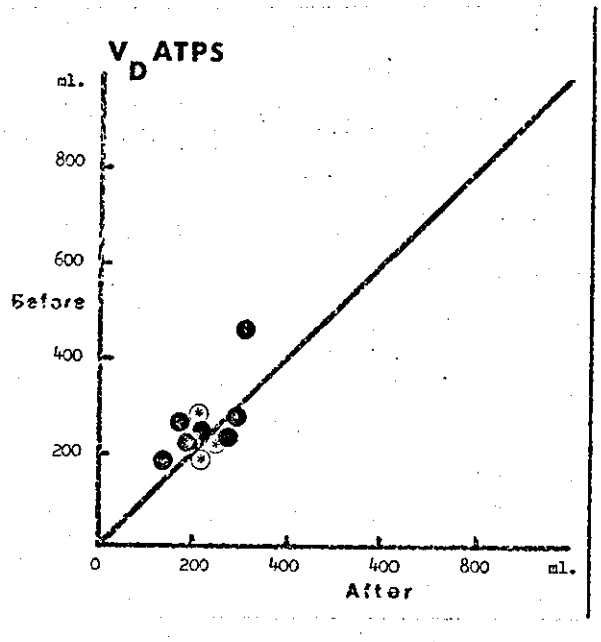

$\dot{V}_D$BTPS
l.min.
16
12
Before
8
4
4
8
12
16
l.min.
After

TREADMILL: SINGLE BREATH VALUES

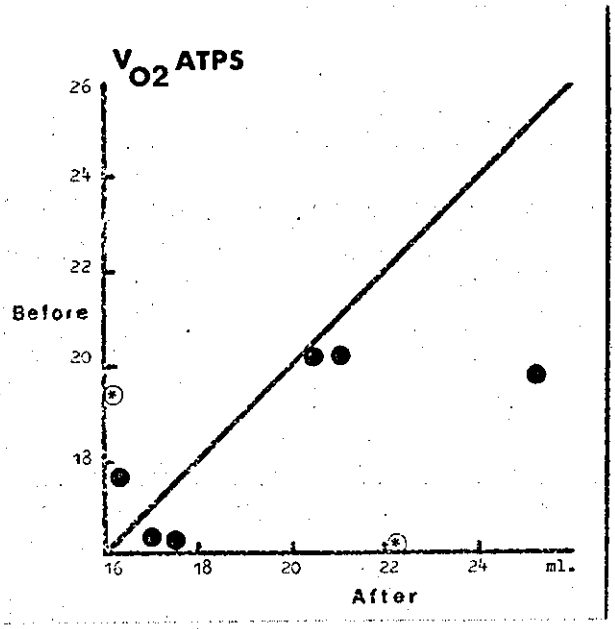

V_{O2} ATPS
ml.
80
60
Before
40
20
0
20
40
60
80
ml.
After

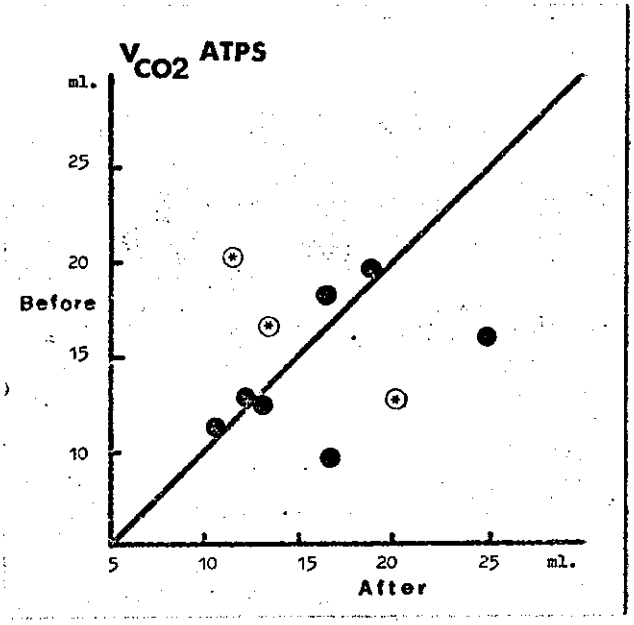

V_{CO2} ATPS
ml.
80
60
Before
40
20
0
20
40
60
80
ml.
After

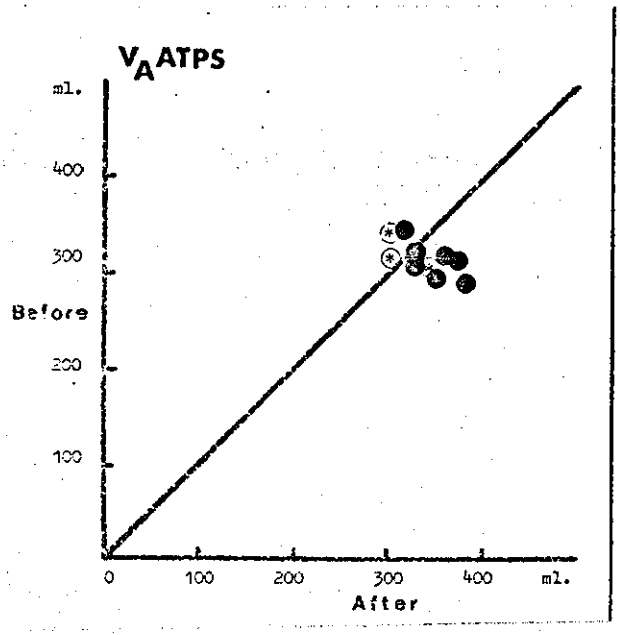

V_A ATPS
ml.
800
600
Before
400
200
0
200
400
600
800
ml.
After

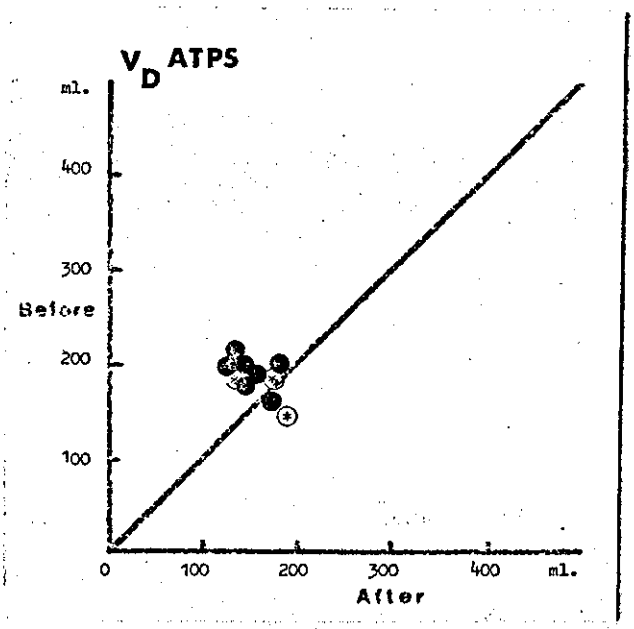

V_D ATPS
ml.
800
600
Before
400
200
0
200
400
400
800
ml.
After

TREADMILL: VALUES AT STANDARD $V_T^{0.51}$

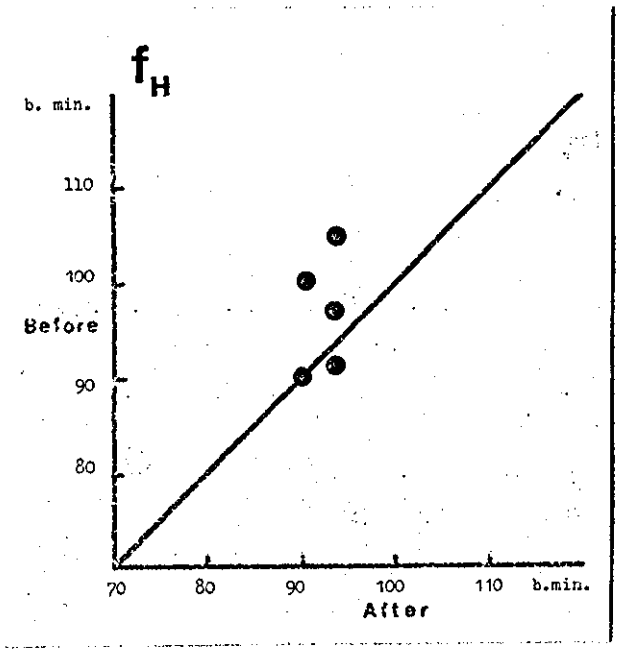

V_{O2} ATPS
26
24
22
Before
20
18
16
18
20
22
24
ml.
After

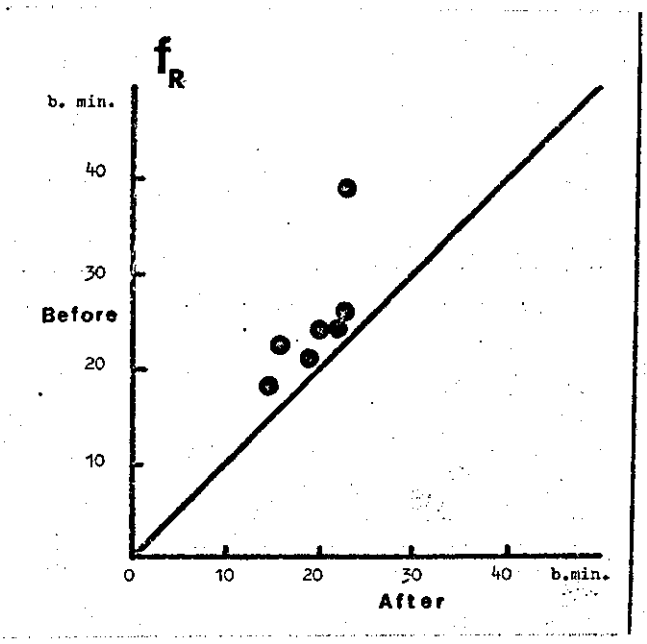

V_{CO2} ATPS
ml.
25
20
Before
15
10
5
10
15
20
25
ml.
After

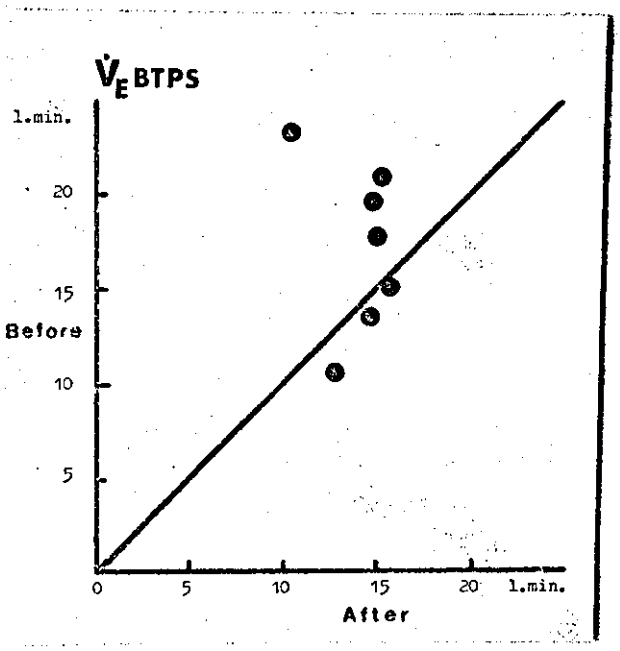

V_A ATPS
ml.
400
300
Before
200
100
0
100
200
300
400
ml.
After

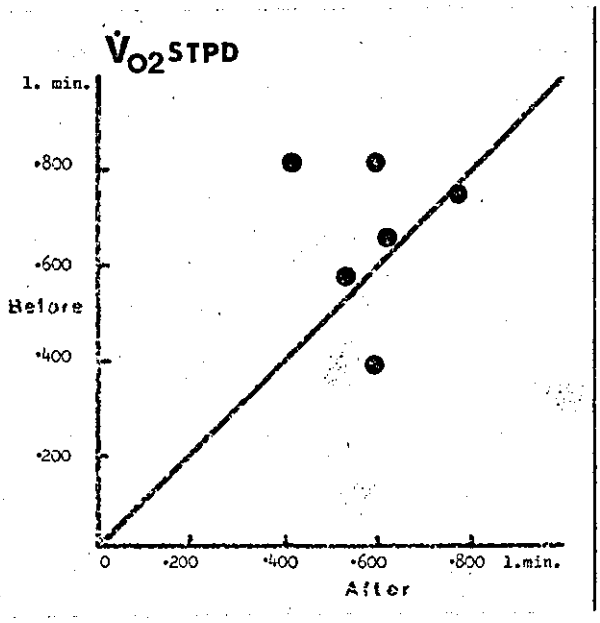

V_D ATPS
ml.
400
300
Before
200
100
0
100
200
300
400
ml.
After

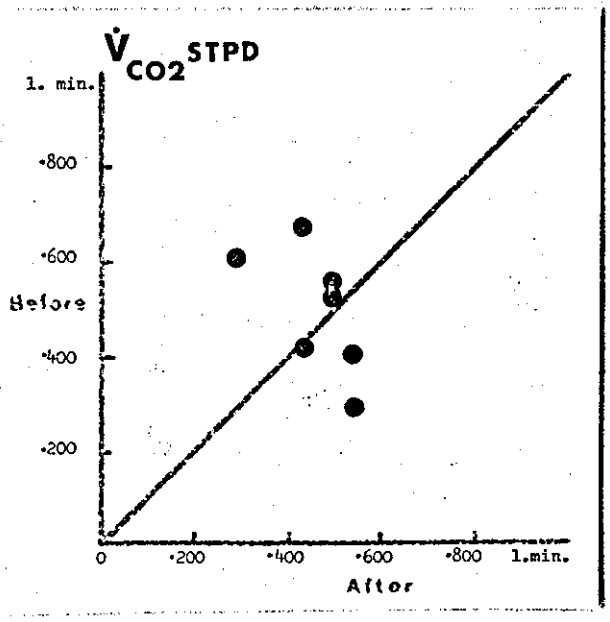
TREADMILL: RESPONSES AT STANDARD GRADIENT

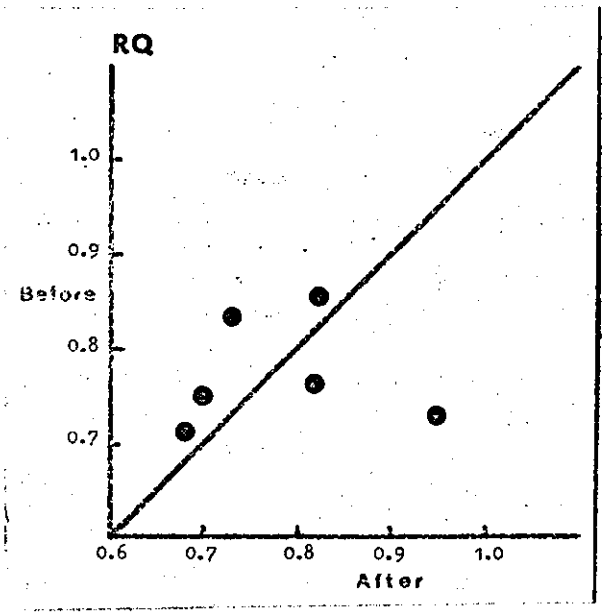

f_H
b. min.
110
100
Before
90
80
70
80
90
100
110
b.min.
After

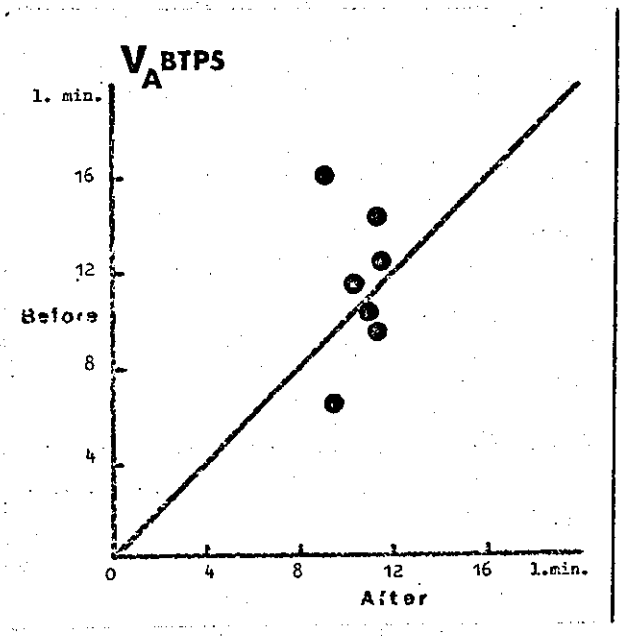

f_R
b. min.
40
30
Before
20
10
0
10
20
30
40
b.min.
After

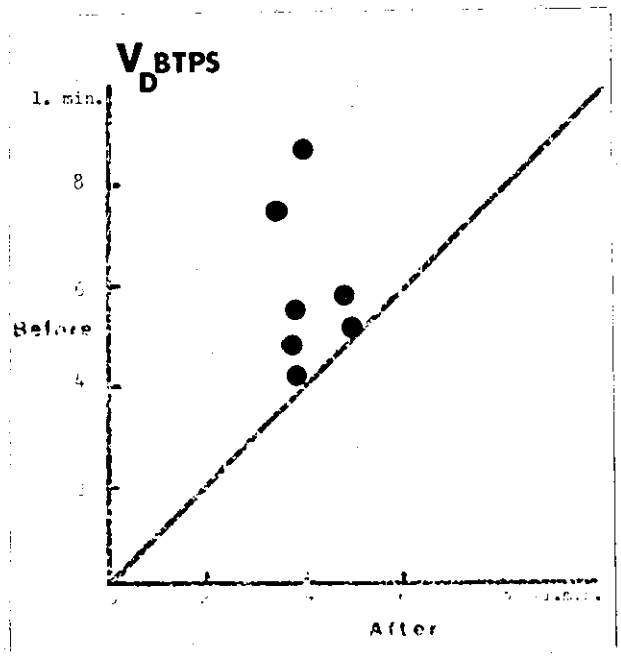

$\dot{V}_E$ BTPS
l.min.
20
15
Before
10
5
0
5
10
15
20
l.min.
After


$\dot{V}_{O2}$ STPD
l. min.
·800
·600
Before
·400
·200
0
·200
·400
·600
·800
l.min.
After

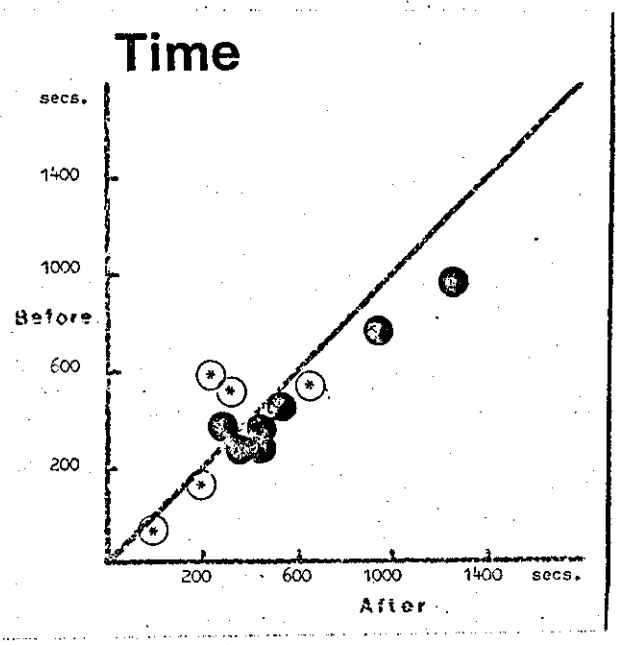

$\dot{V}_{CO2}$ STPD
l. min.
·800
·600
Before
·400
·200
0
·200
·400
·600
·800
l.min.
After

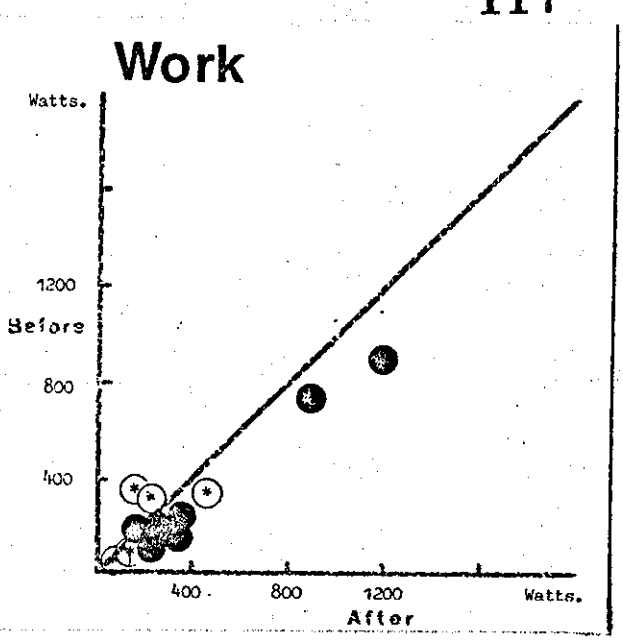

RQ
1.0
0.9
Before
0.8
0.7
0.6
0.7
0.8
0.9
1.0
After

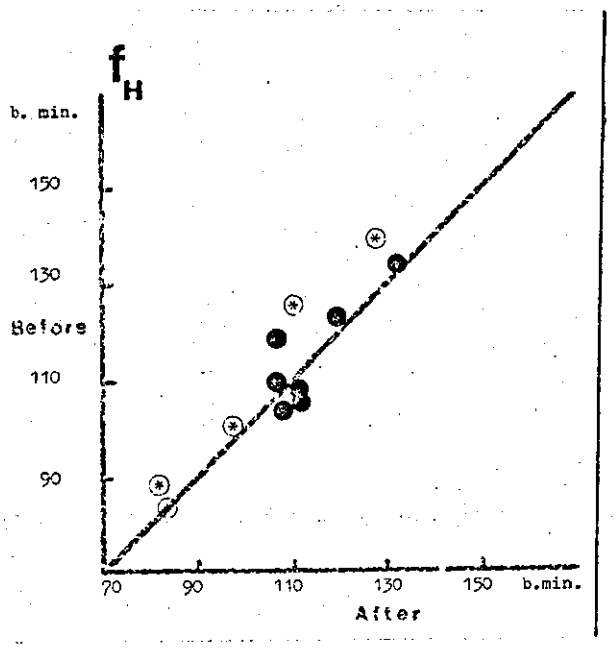

V_ABTPS
l. min.
16
12
Before
8
4
0
4
8
12
16
l.min.
After

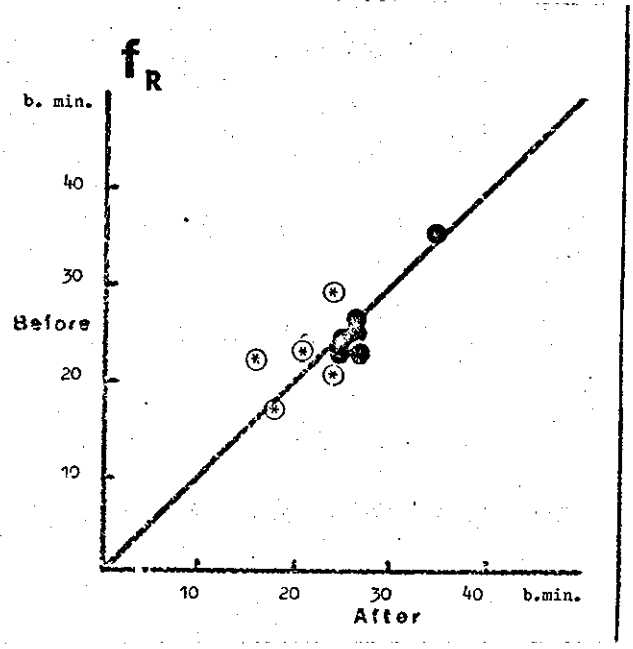

V_DBTPS
l. min.
8
Before
4
After

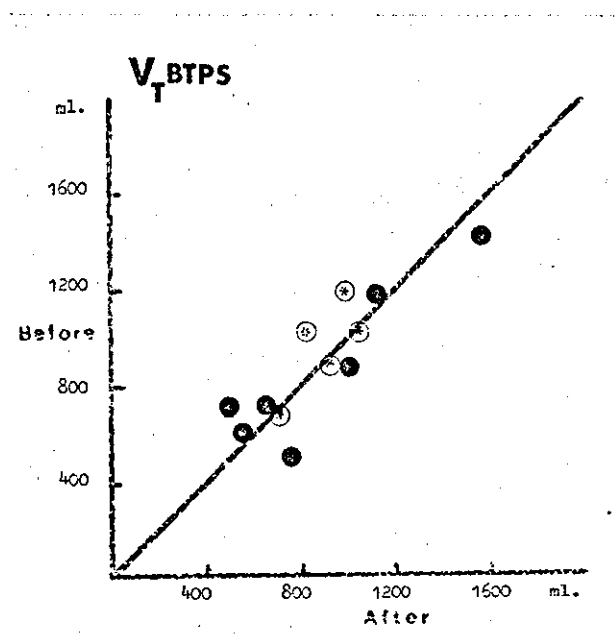
BICYCLE: MAXIMUM VALUES

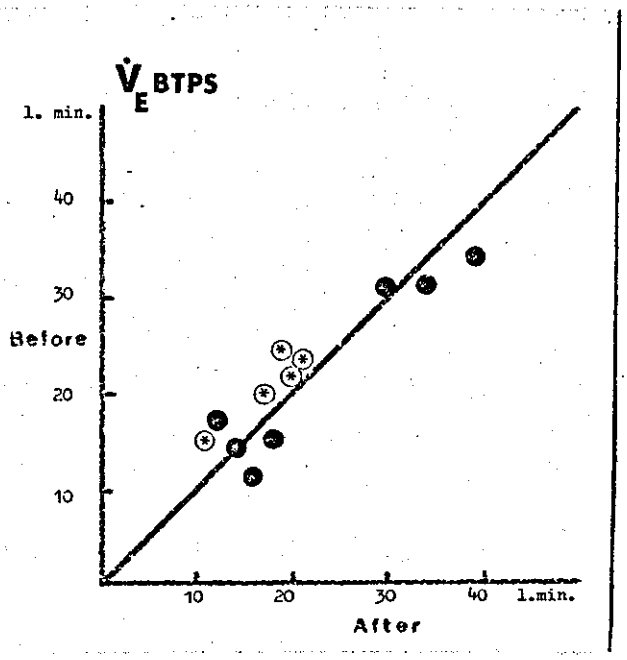

Time
secs.
1400
1000
Before
600
200
200
600
1000
1400
secs.
After

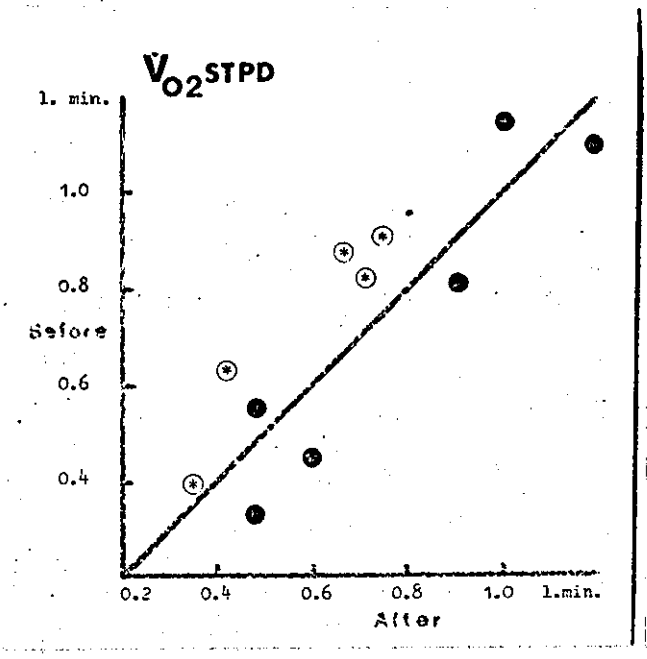

Work
Watts.
1200
Before
800
400
400
800
1200
Watts.
After

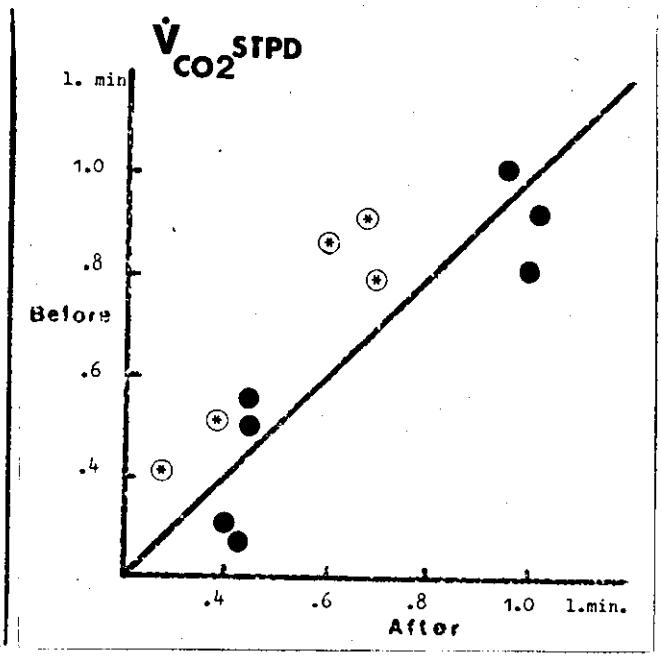

f_H
b. min.
150
130
Before
110
90
70
90
110
130
150
b.min.
After

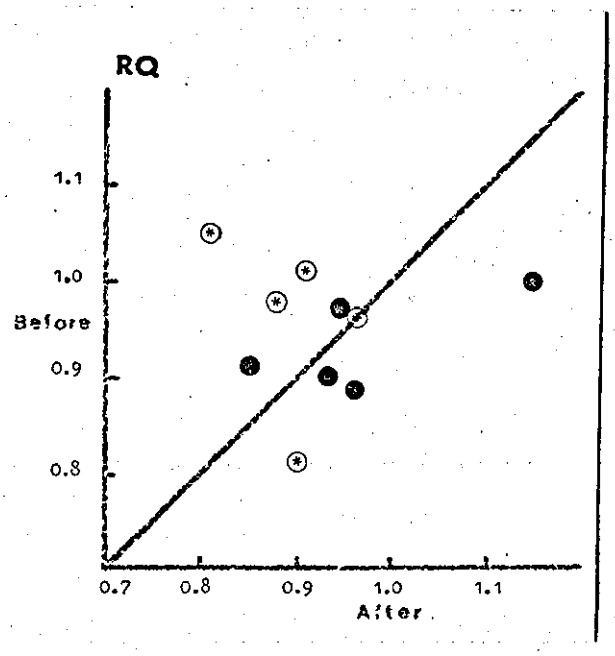

f_R
b. min.
40
30
Before
20
10
10
20
30
40
b.min.
After

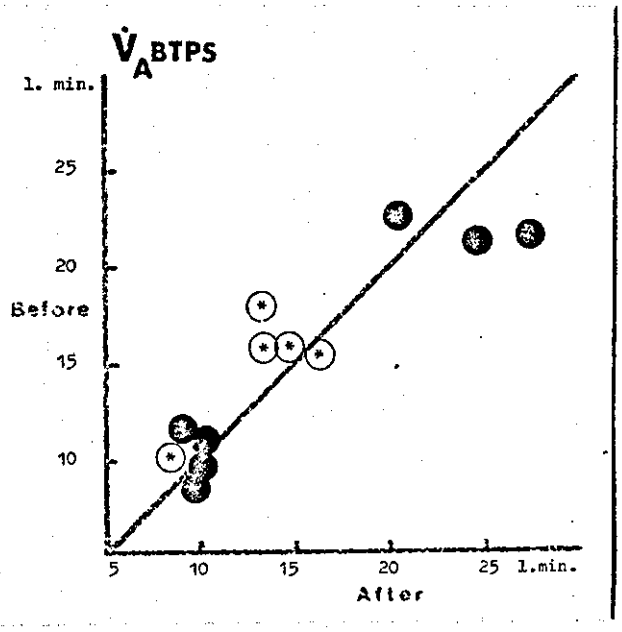

V_T BTPS
ml.
1600
1200
Before
800
400
400
800
1200
1600
ml.
After

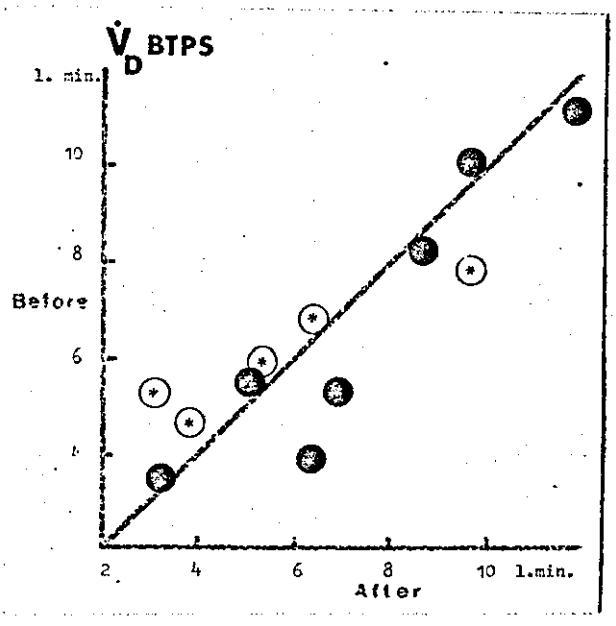

$\dot{V}_E$ BTPS
l. min.
40
30
Before
20
10
10
20
30
40
l.min.
After


$\dot{V}_{O_2}$STPD
l. min.
1.0
0.8
Before
0.6
0.4
0.2
0.4
0.6
0.8
1.0
l.min.
After

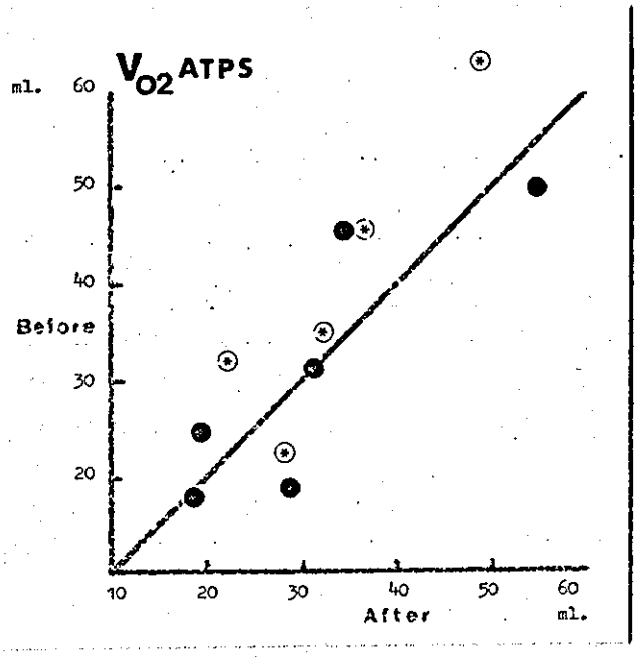

$\dot{V}_{CO_2}$STPD
l. min
1.0
.8
Before
.6
.4
.4
.6
.8
1.0
l.min.
After

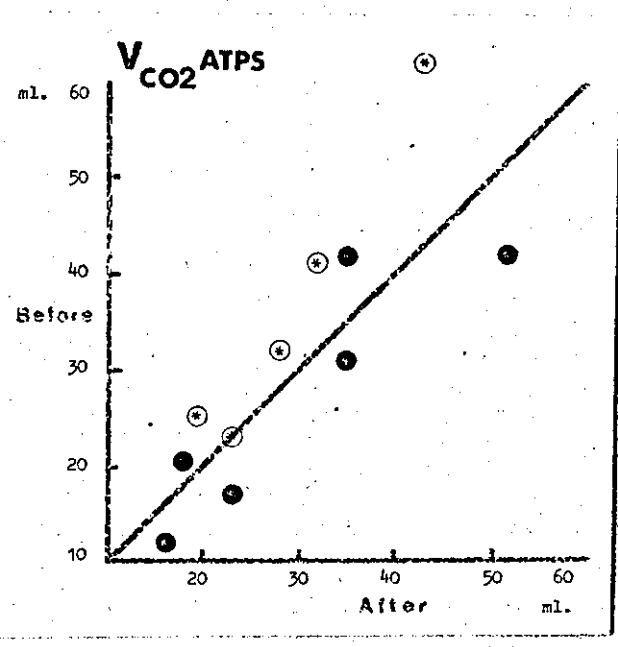

RQ
1.1
1.0
Before
0.9
0.8
0.7
0.8
0.9
1.0
1.1
After

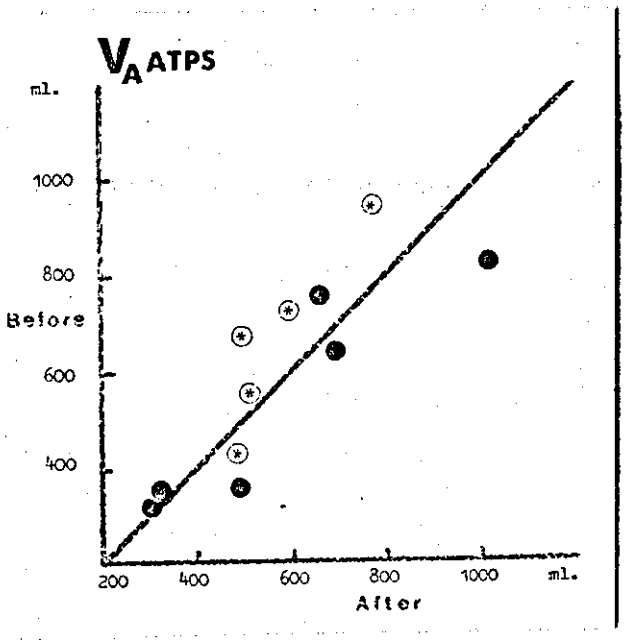

$\dot{V}_A$BTPS
l. min.
25
20
Before
15
10
5
10
15
20
25
l.min.
After

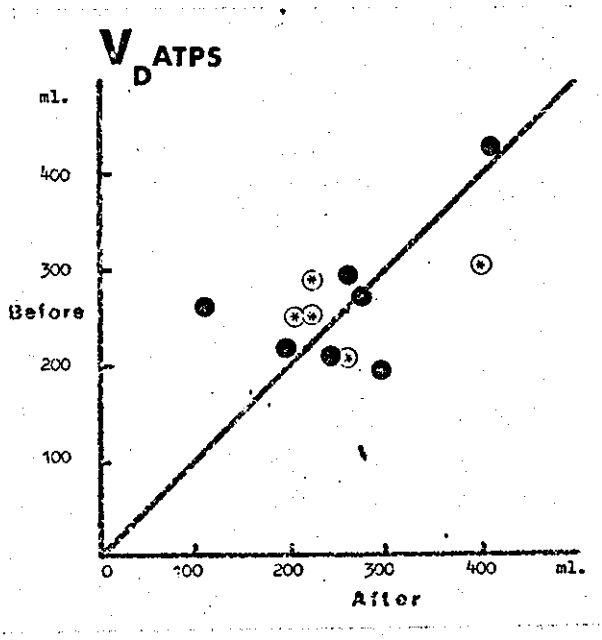

$\dot{V}_D$BTPS
l. min.
10
8
Before
6
2
4
6
8
10
l.min.
After

BICYCLE - SINGLE BREATH VALUES

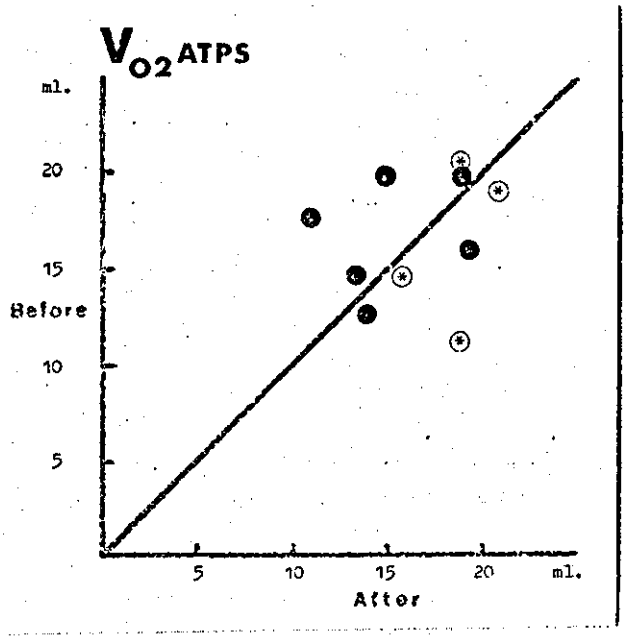

V_{O2} ATPS
ml.
60
50
40
Before
30
20
10
20
30
40
50
60
ml.
After

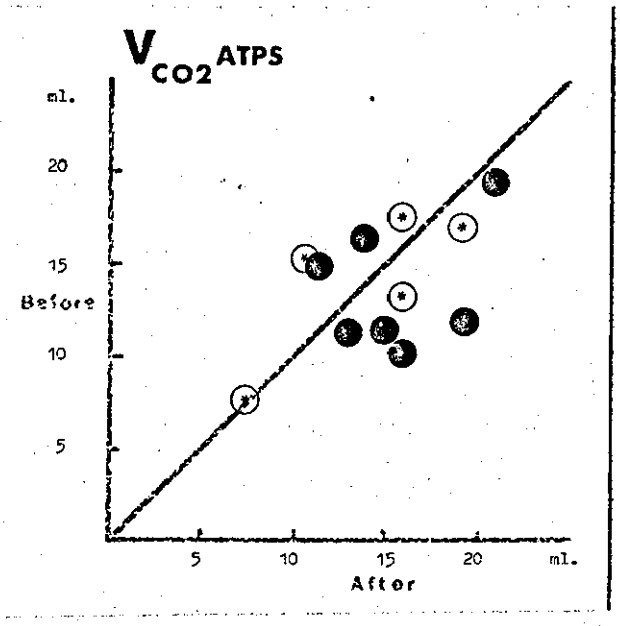

V_{CO2} ATPS
ml.
60
50
40
Before
30
20
10
20
30
40
50
60
ml.
After

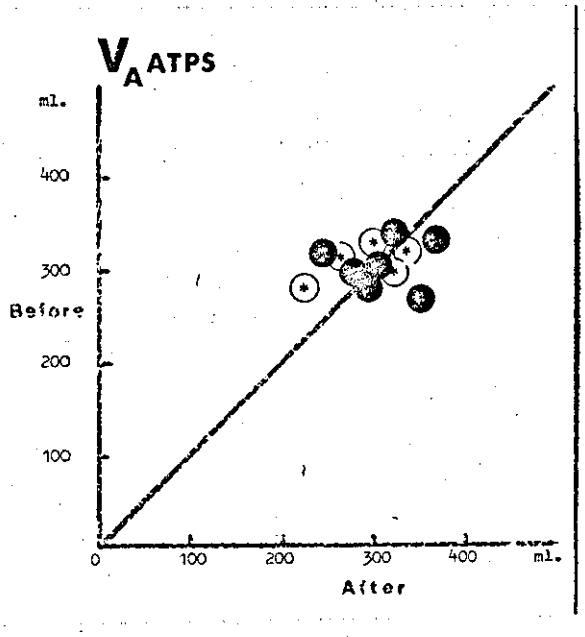

V_A ATPS
ml.
1000
800
Before
600
400
200
400
600
800
1000
ml.
After

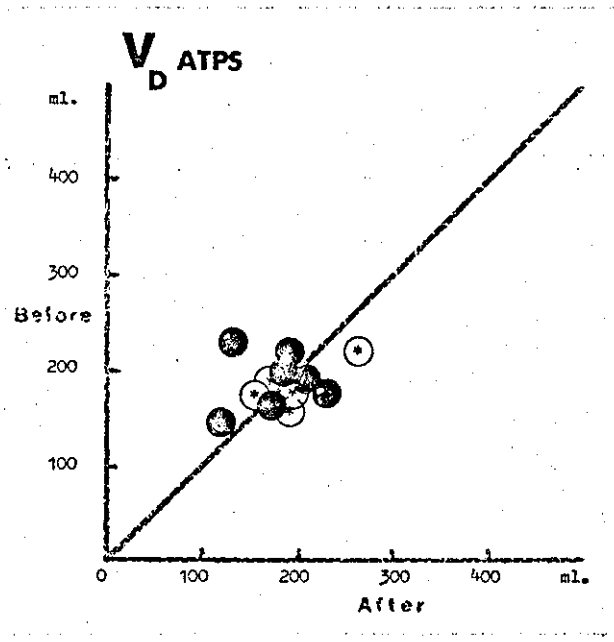

V_D ATPS
ml.
400
300
Before
200
100
0
100
200
300
400
ml.
After

BICYCLE: VALUES AT STANDARD $V_T^{0.51}$.

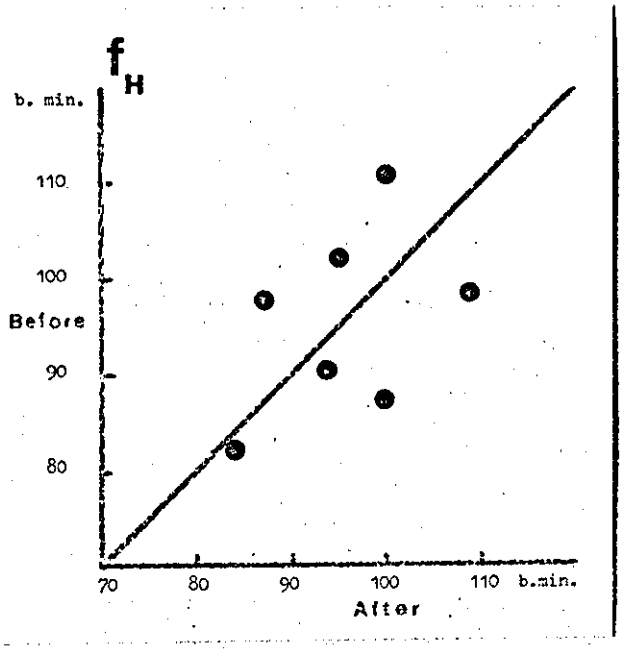

V_{O2} ATPS
ml.
20
15
Before
10
5
5
10
15
20
ml.
After

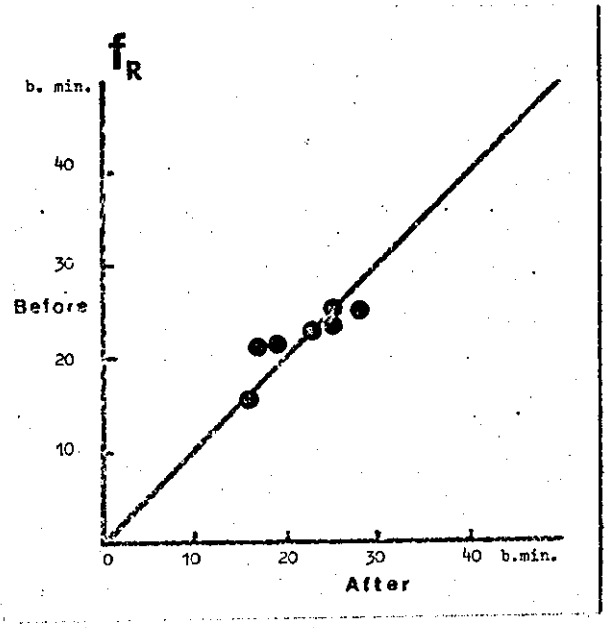

V_{CO2} ATPS
ml.
20
15
Before
10
5
5
10
15
20
ml.
After

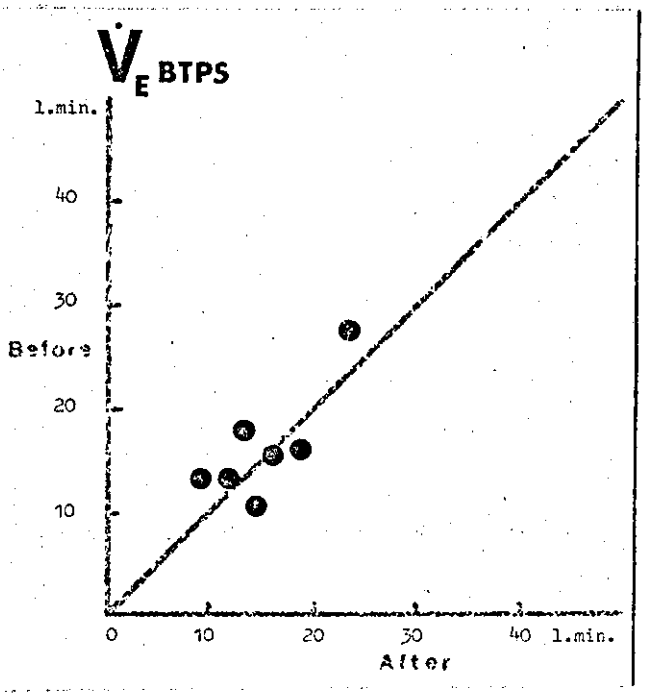

V_A ATPS
ml.
400
300
Before
200
100
0
100
200
300
400
ml.
After

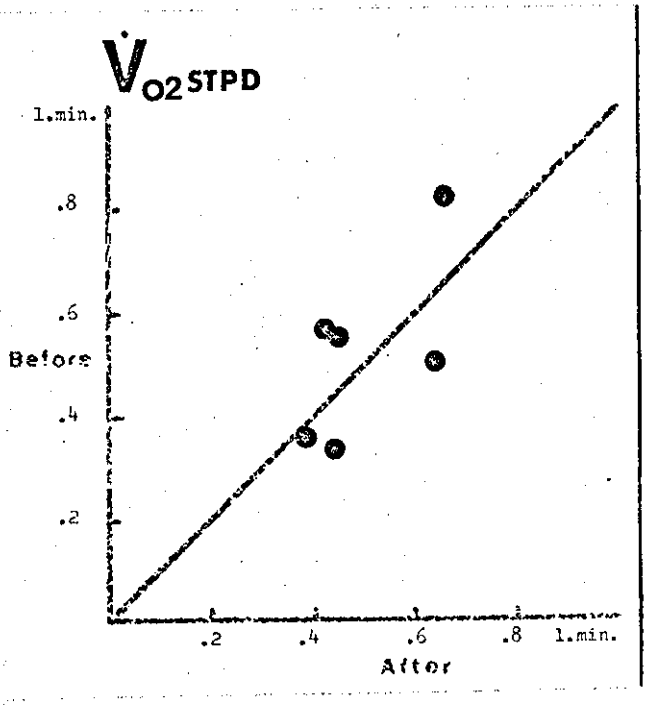

V_D ATPS
ml.
400
300
Before
200
100
0
100
200
300
400
ml.
After

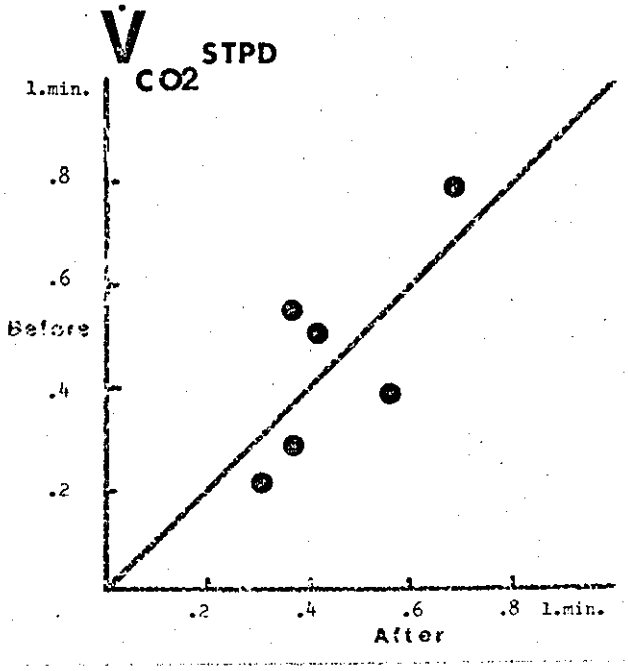
BICYCLE: RESPONSES AT STANDARD WORK (25W)

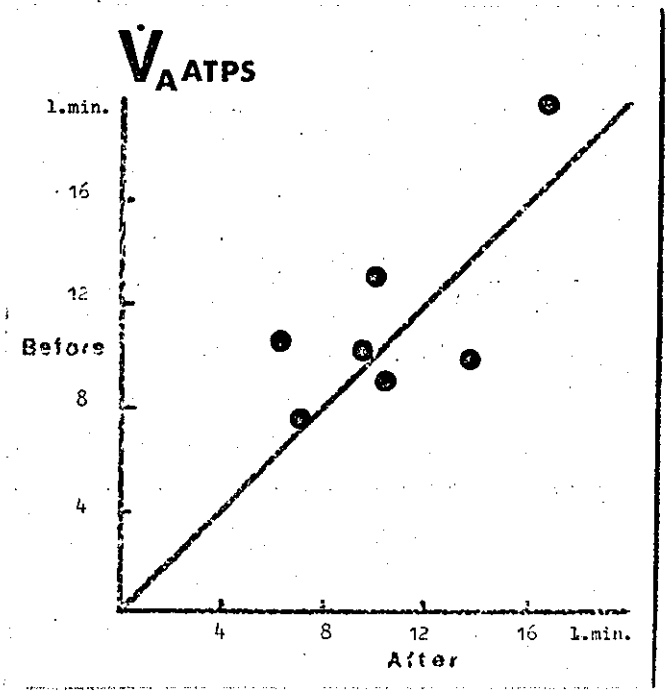

f_H
b. min.
110
100
Before
90
80
70
80
90
100
110 b.min.
After

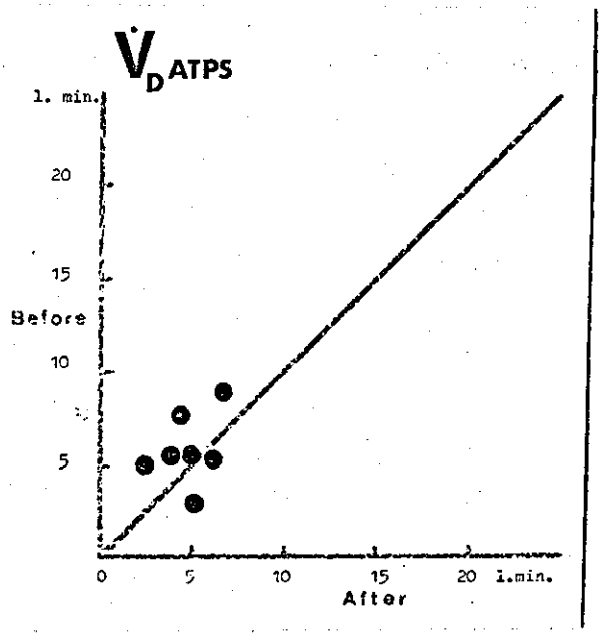

f_R
b. min.
40
30
Before
20
10
0
10
20
30
40 b.min.
After


$\dot{V}_E$ BTPS
l.min.
40
30
Before
20
10
0
10
20
30
40 l.min.
After


$\dot{V}_{O2}$ STPD
l.min.
.8
.6
Before
.4
.2
.2
.4
.6
.8 l.min.
After


$\dot{V}_{CO2}$ STPD
l.min.
.8
.6
Before
.4
.2
.2
.4
.6
.8 l.min.
After


$\dot{V}_A$ ATPS
l.min.
16
12
Before
8
4
4
8
12
16
l.min.
After


$\dot{V}_D$ ATPS
l. min.
20
15
Before
10
5
0
5
10
15
20
l.min.
After

BLE 11

Gross changes in work test responses before and after training

	GROUP E		GROUP C	
AGGREGATE LEVELS	TREADMILL	CYCLE	TREADMILL	CYCLE
TIME	+++**	++*	o	—
WORK		+++*		—
f_H	+++	++*	o	—
V_E	o	+++	++	—
f_R	o	+++*	+	-
V_A	++	+++	+++	—
Vo_2	++	++	+	—
Vco_2	++	+++	+++	—
RELATED TO TIME				
f_H	o	o	o	o
V_E	—	o	+	—
f_R	—	o	o	-
Vo_2	-	o	o	—
Vco_2	-	++	++	—
MAXIMUM RESULTS				
GRADE/WORK	+++	++	o	o
f_H	o	o	o	-
V_E	o	+	++	—
f_R	o	o	-	-
V_T	o	o	++	—
Vo_2	o	+	o	—
V_{Co2}	o	+	++	—
F_{Eo2}	o	o	—	-
F_{ECo_2}	+	o	o	—
V_A	o	+	++	—
V_D	—	o	+	-

KEY

<5% = o

10% = +

20% = ++

>20% = +++

* = $p<0.05$

** = $p<0.02$

	TREADMILL		CYCLE		TREADMILL		CYCLE	
SINGLE BREATH								
V_T	o		o		+		−	
V_{O_2}	+		o		o		−−	
V_{CO_2}	+		o		++		−−−	
F_{EO_2}	o		o		−		−	
F_{ECO_2}	++		+		o		−−	
V_A	+		o		+		−−	
V_D	−		o		o		o	
STANDARD VT 0.5, 0.8								
V_{O_2}	++	o	o	o	−−	−−		
V_{CO_2}	++	++	++	+	o	o	o	−
V_A	++*	+	o	o	o	o	−	o
V_D	−−*	−−	−	o	o	o	+	o
F_{EO_2}	+	o	−	o	−	−−	−	o
F_{ECO_2}	++	++	o	+	o	−	−−	−
STANDARD WORK								
f_H	o		o					
V_E	−−		−					
f_R	−−**		o					
V_T	o		o					
V_{O_2}	−−		o					
V_{CO_2}	−		−					
F_{EO_2}	+		o					
F_{ECO_2}	++		o					
V_A	−−		−					
V_D	−−**		−					

work the changes are more marked; $\dot{V}_E$ fell 17% (3 l. min.) and f_R by 20% (5. b. min), this last reduction being significant at the 2% level.;

Alveolar ventilation (V_A) increased in aggregate value 11% (14. 9 l), at single breath level by 6% (31 ml) and at standard V_T by 12% (36 ml, $p < 0.05$) at 5% (28 ml) respectively, but remained unchanged at maximum work and fell 10% (1. 2 l. min BTPS) at standard work. Ventilatory dead space (V_D) decreased consistently; at maximum work $\dot{V}_D$ fell almost 8% (5. 3 l. min), single breath level by 13% (34 ml) and at standard V_T by 18% (33 ml. $p < 0.05$) and 14% (38 ml). The greatest reduction, 30% (1. 7 l. min) occurred at standard work; this is equivalent to a mean reduction of 85 ml. b. and was significant at the 2% level.

Gas exchange

The slower slightly deeper breathing pattern produced small but consistent increases in oxygen and carbon dioxide extraction rates. Mixed expired $F_{\bar{E}O2}$ rose very slightly, 0. 4% (0. 02%) at maximum work, 3% (0. 15%) at single breath level, approximately 4% at standard V_T (0. 17% and 0. 18% respectively) and by 8% (0. 36%) at standard work. $F_{\bar{E}CO2}$ increases were generally greater, and at the levels just described were: maximum 9% (0. 33%), single breath 11% (0. 37%), 10% (0. 30%) and 13% (0. 44%) and standard V_T and 13% (0. 41%) at standard work.

In keeping with longer working time aggregate $\dot{V}o_2$ rose 16% (1. 5 l), but fell 10% (0. 91 l. min) when related to time, remained virtually unchanged at maximum level, but was more than 11% (0. 07 l. min) lower at standard work. Oxygen delivery per breath improved slightly - 5% (2 ml.) at mean single breath, and by 10% and 5% (2 ml in each case) at standard V_T.

Carbon dioxide excretion increased significantly in aggregate terms by 13% (1. 98 l, $p < 0.02$), $\dot{V}_{CO2}$ increased at maximum work 4%

(0. 03 l. min), by 10% (4 ml) at single breath level, and by about 14% (2 and 4 ml) at standard V_T, but fell 10% (0. 45 l. min) when aggregate value was related to time and by 5% (0. 03 l. min) at standard work.

Treadmill: Group C

The responses of the control group now follow, but as only three of the five were able to complete the post training treadmill work test some caution is required in interpretation. Furthermore comparisons at standard work are not possible as only two subjects were capable of working for the required 5 minutes.

a) Work

Individual responses varied greatly, but mean working time increased slightly by 3% (20 secs) and the maximum grade achieved remained unchanged.

b) Heart rate

Changes in f_H were negligible; total number of beats rose by only 1, but average f_H over the whole period fell by 4% (4 b. min). Symptom limited $f_{H\ max}$ remained virtually unchanged, increasing by a mere 1% (1 b. min).

c) Ventilation

Although the increase in working time was small, marked changes occurred in some measures of ventilation. Aggregate V_E rose 14% (19. 2 l), f_R increased 7% (12 b.) and accordingly V_T rose 6% (500 ml); even when related to time $\dot{V}_E$ rose 7% (1. 4 l. min) but f_R remained unchanged. At maximum $\dot{V}_E$ was 10% higher (2. 1 l. min), and with a 6% drop in f_R (1. 4 b. min) V_T increased 13% (105 ml).

As a result V_A increased at all levels except standard V_T. At maximum the rise was 12% (1. 7 l. min) and at single breath it was 6% (38 ml) higher; changes at standard V_T were less than 1% (+ 2 ml, - 5 ml.

respectively). $\dot{V}_D$ increased 7% (0.40 l. min) at maximum, fell 3% (8 ml) at single breath level, remained almost unchanged (- 2 ml) at V_T 0.5 l and rose 3% (7 ml) at V_T 0.8 l.

Gas exchange

The increased alveolar ventilation produced consistently higher expired oxygen concentrations; $F_{\bar{E}O2}$ fell 11% (0.56%) at maximum work, 6% (0.28%) at single breath level, and 8% (0.32%) and 17% (0.82%) at standard V_T. Changes in carbon dioxide concentrations were more variable; there was a negligible increase - less than 1% (0.02%) at maximum work, an increase of 4% (0.15%) at mean single breath, no change at V_T 0.5 l, and an 8% (0.34%) fall at V_T 0.8 l.

Aggregate oxygen uptake rose 5% (0.36 l) but remained unchanged when aggregate values were related to time, at maximum value and at single breath level, and fell 12% (2 and 4 ml. respectively) at standard V_T. Carbon dioxide excretion increased as a result of higher ventilation; aggregate levels rose 21% (1.14 l), average levels by 12% (0.08 l. min); at maximum the increase was 16% (0.10 l. min), and was 10% (3 ml) at single breath level. However at standard V_T C02 excretion fell slightly - 4% (1 ml. in both areas).

Bicycle ergometer: Group E

a) Work

An increase in working time was observed on the bicycle. Although one subject, E4, failed to increase work time, three were able to maintain the same work load longer and the remaining three increased maximum rate by 25 W. The results of these changes were a rise of 19% (109 secs) in work time and a 33% increase (128 W) in aggregate work, both results being significant at the 5% level and a rise of 19% (11 W) in maximum work rate.

b) Heart rate

Not surprisingly the total number of beats rose by 18% (175 b. $p < 0.05$), but when related to time f_H remained unchanged, and increased by merely 1 b. min. at maximum work. Relating work to f_H (work-pulse) a 16% (0.8W) increase in work done per 10 beats was observed.

c) Ventilation

Greater work output led to a 27% (46 .1) increase in aggregate V_E, and a 24% (46 b) rise in f_R which was significant, $p < 0.05$. However when related to time the increases were modest - $\dot{V}_E$ rose 4% (0.60 l. min), and f_R remained virtually unchanged (lb. min). At maximum levels too increases were small; $\dot{V}_E$ was 5% (1.2 l. min) higher, f_R rose by less than 1 breath per minute and V_T was 4% (33 ml) greater. At mean single breath V_T remained practically unaltered (+5 ml), but at standard work measures tended to decrease, but again reductions were generally small - $\dot{V}_E$ fell 7% (1.2 l. min), f_R by less than 1 breath and V_T by only 1% (7 ml).

Except at standard work, where a 6% (0.07 l. min) fall was observed, V_A increases slightly. The greatest rise, at maximum work, was only 6% (0.90 l. min): at single breath level the increase is barely nitoceable - 1% (6 ml.), and at standard V_T it amounted to no more than 3% (11 and 14 ml. respectively). $\dot{V}_D$ rose at maximum work by 4% (031 l. min), but fell at single breath, 4% (11 ml.), standard V_T, 5% (10 and 13 ml. respectively) and standard work, 10% (0.60 l. min).

d) Gas exchange

Changes in gas concentrations in the expired air reflected the increased ventilation. F_{E02} was unchanged at single breath level, but was reduced 3% (0.13%) at maximum work, 8% (0.28%) and 5% (0.20%) at standard V_T, and 2% (0.10%) at standard work. F_{EC02} rose consistently;

5% (0.17%) at maximum work, 8% (0.24) at single breath level, 4% (0.12%) and 9% (0.30%) at standard V_T, and 5% (0.16%) at standard work.

Oxygen uptake increased in aggregate terms by 23% (1.62 l) but when this value is related to time the increase was less than 2% (0.01 l. min). Symptom limited $\dot{V}_{02}$ improved 5% (0.37 l. min) but V_{02} delivered per breath remained virtually unchanged, (1 ml increase) and actually fell at standard V_T (1 and 2 ml) and by 3% (0.02 l. min) at standard work. V_{C02} increased at all levels except standard work where the value fell 5% (0.06 l. min); the increases were 32% (1.91 l) in aggregate terms, 14% (0.07 l. min) when related to time, 8% (0.05 l. min) at maximum work, by only 3% (1 ml) at single breath level but by 15% (3 ml) and 8% (2 ml) at standard V_T.

Bicycle ergometer: Group C

All of the controls completed post-training bicycle tests, but again comparison of responses at standard work is impossible because too few were able to complete the initial 25 W work load.

a) Work

Responses here were very variable. Three subjects increased working time, one C3, by 18%, but as C4 reduced his time by 50% the overall result is a decrease of 12% (48 secs) in total work time and a reduction of 18% (42 W) in aggregate work with no change in the maximum work rate.

4) Heart rate

The reduced work output is reflected in decreases in mean f_H; aggregate f_H fell 14% (88 b.), and when related to time by 4% (4 b. min) Maximum f_H also fell, 6%, (7 b. min), but work done per beat increased 17% (0.8 W. 10 b.)

c) Ventilation

Consistently lower measures of ventilation resulted from decreased work. Aggregate V_E fell 21% (26.3 l) and by 14% (2.5 l. min). when related to time; maximum value also fell 14% (3.0 l. min). Respiratory rate fell consistently; 7% (19 b) in aggregate terms, 7% (1.5 b. min) when related to time, and 8% (1.8 b. min) at maximum work. Consequently V_T fell 11% (107 ml) at maximum and 9% (86 ml) at mean single breath.

Not surprisingly V_A was diminished at all levels. Reductions were 17% (2.6 l. min) at maximum work, 12% (82 ml) at single breath level, but only 6%(17 ml) and 1% (9 ml) at standard V_T. Ventilatory dead space fell 6% (0.58 l. min) at maximum, remained virtually unchanged, less than 1% at mean single breath, but increased slightly - 6% (12 ml) and 4% (20 ml) at standard V_T.

d) Gas exchange.

The gas fractions of both oxygen and carbon dioxide were consistently smaller. The changes in $F_{\bar{E}O2}$ were generally slight; reductions of 5% at maximum work (0.21%) and single breath level (0.20%), 6% (0.21%) and 2% (0.07%) at standard V_T were noted. $F_{\bar{E}CO2}$ changes were greater; at maximum work and at single breath level there was a 12% fall (0.49% and 0.47% respectively), and at standard V_T the reductions were 14% (0.43%) and 10% (0.35%).

Decreasing ventilation and smaller gas fractions combined to produce lower oxygen uptake and carbon dioxide excretion. Aggregate V_{O2} fell 25% (1.39 l), and, when related to time, by 17% (0.125 l. min); at maximum work the value fell 19% (0.14 l. min) and at mean single breath there was a 14% (5 ml) reduction. The decreases in V_{CO2} were even

greater; aggregate levels fell 30% (1.55 l), 17% (0.11 l. min.) when related to time; at maximum work CO_2 excretion fell 24% (0.17 l. min), and a 23% (8 ml.) reduction occurred at single breath level; mean value remained practically unchanged (1%) at V_T 0.5 l, but fell 7% (2 ml) at V_T 0.8 l.

Means and all individual responses are given in Appendix H.

PART THREE

DISCUSSION

CHAPTER EIGHT

Discussion : Introduction

In common with previous investigations into the effects of physical training on COLD patients, significant increases in exercise tolerance and markedly enhanced confidence, are observed in this study. The actual increases in working time - 2.5 minutes, on the treadmill and 1.75 minutes on the bicycle - may seem small, but in percentage terms they do represent quite striking increases over initial pre-habituation values; the post-habituation improvements, though more modest (28% and 19% respectively), were not considered trivial by the patients, who saw them as tangible rewards for the strenuous activity of the training programme.

More important perhaps than the increase in work capacity was the widely expressed view that the programme produced enhanced confidence, activities of everyday living were accomplished more easily and activities previously avoided were approached in a less fearful, more realistic fashion. This subjective assessment by the patient may be the most important consequence of the training programme since it may be the precursor of a reorganised life style which could lead to an improvement in the quality of life.

The underlying causes for both objective and subjective improvement have been matters for debate in most studies, but it seems reasonable to conclude that the changes reflect psychological as well as physiological adjustments to activity.

Three studies cite quite specific psychological processes to explain improved performances. Paez et al (1967) and Brundin (1974) claim that increased neuromuscular coordination is chiefly responsible for greater exercise tolerance on the treadmill. Brundin (1974) also suggests that

better tolerance of hypoxenia is an important factor, and similarly Agle et al (1973) see frequent exposures to progressive exercise leading to a decrease in the unrealistic fear of activity and breathlessness.

The majority of investigators seek physiological answers however, and these appear under four general headings. First there are those who feel that changed respiratory mechanics are important (Ambrus et al (1967); Bass et al (1970); Laros et al (1972) and Alpert et al (1974). Secondly there is a group indicating rearranged ventilation-perfusion as a possible source of greater oxygen saturation (Christie (1968); Woolf et al (1969), Degre et al (1974)). There is a third suggestion from Paez et al (1967), Woolf et al (1969), Guthrie et al (1970) and Degre et al (1974) that peripheral oxygen extraction is a major contributory factor, and finally Pierce et al (1964) and Vyas et al (1971) believe that increased symptom-limited oxygen uptake is the process chiefly responsible for enhanced performance.

This study was designed to throw light on to some of these questions, and the next chapter is devoted to closer consideration of the possible causes underlying the subjective and objective improvements that have been noted.

CHAPTER NINE

Discussion: Psycho-physiological aspects of improved performance

Psychological aspects

There are two reasons for placing this area at the head of the discussion. Firstly it is accepted that a variety of psychological mechanisms are involved when an individual, patient or international athlete, is working near his maximum. Simple performance tests have shown that demands for all-out effort can be influenced by improved skill, greater confidence, hypnosis, incentives of various kinds - spectator reaction; encouragement and placebos. Secondly many workers place prime importance on the psycho-therapeutic value of exercise. They believe that reduced work tolerance is principally the product of anxiety, depression and feelings of worthlessness (see page 58); thus they argue an immediate consequence of any carefully controlled and supervised activity session is some relief from the symptoms, leading to rapid improvement in performance. The implication is clear - changes in physiology are unnecessary; reduced anxiety, or greater motivation (or both) will suffice.

Although the quantification of psychological adaptation to training is still not reliable, there is objective evidence elsewhere to support the particular theory just mentioned. Many studies report increased exercise tolerance without any measurable physiological improvement and Kavanagh et al (1970) observed an increased work output of cardiac patients following a course of hypnotherapy without specific physical training.

The present study offers conflicting evidence on the theory. Following exposure to two work tests and a short habituation programme of low intensity the mean working time of the experimental group rose 27% on the treadmill ($n = 6$) and 9% on the bicycle. It seems reasonable to suggest that increases

of such magnitude following rather minimal exposure to exercise could only arise from a combination of habituation - to the treadmill, nose-clip and mouth-piece, the personnel and to the procedure generally - and, since pedalling time also improved, to an overall reduction in anxiety. Small reductions in mean pre-work test f_H of group E (86.5 - 83.2 b.min. on the treadmill 83,2 - 81.8 on the bicycle) and the controls (86.1 - 85.0 on the bicycle) are suggestive of reduced anxiety.

It is clear that for many patients anxiety adversly influences work tolerance, but it is equally apparent that the benefits occuring from reduced anxiety are limited, and after the initial response working time is hardly affected by daily training of minimal intensity. Comparison of post-habituation and post-training responses from the control group reveal only a small increase in treadmill time (3 - 4% but note n = 3 only), and a reduction in bicycle work; even if the marked reduction in C4's time is ignored the mean pedalling time of the remainder is only 4.5% greater. Thus increases in work tolerance in excess of about 5% are dependent on factors other than reduced anxiety.

Increased motivation, extrensic as well as intrensic may be a factor. Although it was impossible to disguise the relative importance of the final tests positive attempts were made to reduce extrensic motivation before and during the tests. Patients were unaware of their pre-training results and vocal encouragement was not employed by the supervising staff. Nevertheless it is impossible to say how much the patient's need for success influenced his working time, but it is worth noting that mean $f_{H\ max}$ on both devices for both groups remained virtually unchanged or even marginally reduced on the final tests; if greater motivation had been a dominant influence in the work test situation an increased $f_{H\ max}$ would not have been surprising.

The psycho-physiological 'desensitisation' theory of Agle et al (1973) and Brundin (1074), based on the reciprocal inhibition methods employed by behaviour therapists to extinguish phobias, is another plausible possibility. Bearing in mind the combined effects of frequent and uncomfortable reminders of this low work tolerance together with the rather negative advice proffered by the physician to avoid unnecessarily strenuous activity, it is not difficult to imagine patients, particularly severely impaired ones, developing some kind of exercise-phobia. According to Eysenck (1965) a conditioned sympathetic response to an unpleasant experience - in this case exertional dyspnoea - is set up; the fear that develops is roughly proportional to the proximity of the stimulus in question but by gradual progressive and controlled exposures to the source of the phobia the sympathetic response can be extinguished or replaced by a conditioned para-sympathetic response. In the present study the supervised progressive exercise regime is seen as the sympathetic deconditioner and would explain, in part, two heart rate observations. Firstly there is the reduction in maximal working heart rate, and secondly the relatively unchanged group f_{Ht} recorded during training sessions. This latter feature is taken-up in another context later in the discussion but it is worth noting here that although speed and gradients were increased over the period of training the mean daily f_{Ht} remained remarkably constant, varying as little as 5 b. min. after the first day of training. This, together with the reduced fH max on the treadmill could be interpreted as a reduction in the sympathetic response to exercise phobia.

It is also possible that a second psycho-physiological phenomenon, namely greater neuro-muscular coordination (Paez et al 1967) is largely responsible for improved performance. The suggestion is based on the notion that practice of a motor skill is likely, up to a point, to result in better technique; extraneous body movements are eliminated, the activity is performed more effectively and there is a concomitant reduction in energy demand. There is evidence to support this contention. Reduction in $\dot{V}_E$ and $\dot{V}_{02}$ of more than 20% have been reported during standard treadmill walking (0°, 60 m. min) following practice or injunctions to walk in a relaxed fashion (Cotes 1968). Reduced oxygen demand at standard work on a bicycle was the only significant change in the responses of a group of students undergoing a recreative training scheme which included pedalling on a fixed bicycle (Hole - unpublished material). During class experiments with female physical education students it has been observed that lower oxygen uptake (related to body weight) at standard work is frequently displayed by those students who cycle to College in comparison with those who employ other means of transport.

Three previous studies on patient groups also contain evidence of reduced energy demand following practice. Pierce et al (1964) reported large reductions in f_R (42%), $\dot{V}_E$ (40%), $\dot{V}_{02}$ (28%) and $\dot{V}_{C02}$ (27%); Hass et al (1969) noted reduced oxygen demand in a variety of activities (loop weaving, floor loom weaving, stair climbing), and Vyas et al (1971) observed a small (7%) but significant fall in half minute ventilation and an even smaller drop in $\dot{V}_{02}$ (3%) during standard bicycle work.

Both subjective and objective evidence in support of the general theory of greater mechanical efficiency is also available in the present study. The patients' first attempts at treadmill walking were characterised

by unnatural posture, usually in the form of leaning forward, heavy dependence on the hand rails for support, uneven gait, short steps and exaggerated knee lift. By the end of the habituation programme the worst of the excesses had been eliminated, but the position of the mouth-piece and valve box prevented natural head movements and patients were encouraged to use the rails as stabilisers during the work tests. As the training programme progressed most of the subjects appeared to walk more easily - longer strides, natural arm movements - and many commented on the fact that walking on the treatmill seemed much easier than a comparable gradient outside.

The post-training treadmill results reveal the extent of the metabolic economy. Although aggregate values show consistent increase, when related to time the values are lower, a tendency which is repeated at maximal levels. But it is at standard grade and speed that the most striking changes occur, with marked reduction in respiratory rate, minute volume, and oxygen uptake. Not unexpectedly the economics are rather specific. According to learning theory before transfer of training can take place effectively the activity practised has to be similar to the activity tested, which is obviously the case in the treadmill results; although there are reductions in some variables at standard bicycle work they are minor changes. Thus, mechanisms other than enhanced neuro-muscular coordination are responsible for the greater bicycle work output observed in the present study.

Summarising this section on the psychological aspect the following points can be made:-

a) anxiety almost certainly affects initial work performance, but a pre-study habituation programme appears to reduce the importance of this particular variable and permits the evaluation of training effects to start

from a more reliable base line; substantial improvements in post-habituation exercise tolerance are likely to occur as a result of factors other than continued reduction in anxiety.

b) increased entrinsic motivation might influence final work output, but in the absence of objective evidence in the form of increased respiratory or heart rate during maximum effort, such consideration is speculative.

c) if desensitisation therapy is a factor then exposure to exercise per se will not suffice; the activity needs to be progressive and close to the maximum tolerable by the subjects.

d) much of the improvement noted in studies where a single device is employed to test and train the subjects is probably due to enhanced neuro-muscular coordination which is not transferable to general activities.

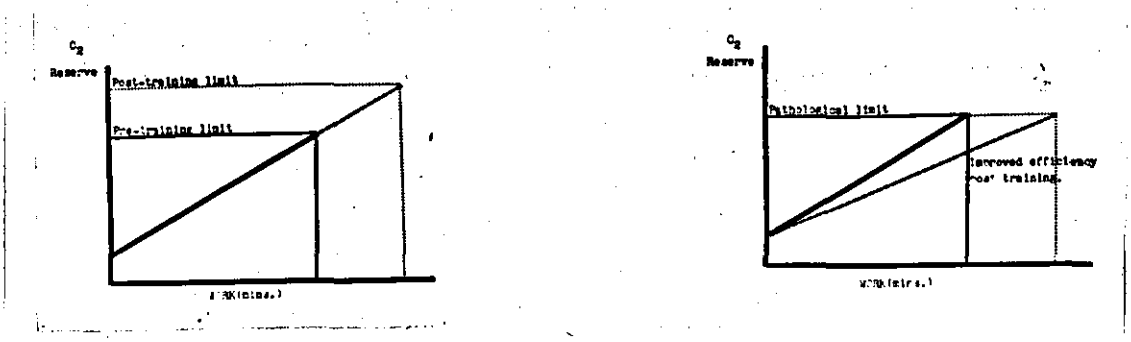
e) providing realistic experimental design precautions are taken psychological factors alone, important though they may be, are unlikely to provide sufficiently satisfying answers to the problems of evaluating physical training programmes for COLD patients.

The other factors implied in the final comment above are physiological in origin and are the concern of the following section.

Physiological aspects

Oxygen availability is the physiological phenomenon which, in COLD patient studies, is examined to the virtual conclusion of all others. Such concern seems logical since arterial hypoxemia is considered to be the principle cause of the commonly observed reduced exercise tolerance, a point confirmed by increases in work noted when COLD patients are given oxygen during exercise (Cotes 1968). Thus it is reasonable to speculate that the increased work output seen in this present study is brought about by improved oxygen availability; further speculation leads to the suggestion

that such improvement is achieved in one of two ways. Either there is an increase in the total amount of oxygen available or better use is made of the existing reserve; the two mechanisms are presented diagrammatically in the accompanying figure.



Increased Vo_{2SE}.

More efficient use of existing 02 reserve.

Figure 31 Possible mechanisms by which exercise tolerance is improved by training.

The reserve available for exercise can be adequately described as the difference between resting arterial and venous blood oxygen content. In normals under standard conditions ($P_{A\ 02}$ = 100 mm. Hg.; Pa_{C02} = 40 mm. Hg. Hb. = 15 gm. %; pH = 7.4, t_B = 38°C) this difference is approximately 15.5 ml. 0_2 100 ml. blood. But the picture is more complicated; in patients conditions are seldom standard - Pa_{02} is low, P_{C02} is often increased, Hb values may be higher and pH may decrease - and the blood oxygen content may be reduced. Furthermore cardiac output, peripheral circulation and the tissue utilisation capacity will affect the amount of oxygen taken up by the working muscle.

Oxygen availability then is dependent on a series of chain reactions involving ventilatory, cardiovascular and tissue activity embracing the pick-up, transportation and final delivery of the oxygen to the mitochondrion. A weak link in any part of the chain effectively diminishes the overall

performance and evidence of improvement in gas exchange at the lung, cardiac performance and oxygen utilisation at tissue level is necessary before the oxygen reserve can be optimally employed. Each link in the chain is now considered more fully.

Oxygen exchange at the lung

Two factors are involved here; they are firstly the gas mixing efficiency in the lung and secondly the pattern of blood supply to the lungs. Cumming (1966) believes that variations in inspired air transit time distributions, whether caused by the anatomical asymmetry of the bronchial tree or by non-uniform transpulmonary pressures, and incomplete diffusion from approximately first-order alveolar-duct level results in regional and stratified inhomogeneity, unequal distribution of inspired gas and impaired gas mixing efficiency. It is also suggested that stratified inhomogeneity (i. e. incomplete alveolar diffusion) is the principal form of mixing inefficiency in the lungs of COLD patients.

There are indications that post-training gas mixing efficiency may have improved. Firstly, although results from only three subjects are available alveolar efficiency at rest, determined by Cummings nitrogen decay curve, increased in all three individuals (four if the pilot study results are considered) the mean increase being 28%. Secondly dead space measures during exercise tended to diminish; this was particularly apparent at single breath and standard tidal volume levels.

However, dead space calculations presented here are based on Bohr's equation and is made up of anatomic dead space - the conducting tubes ; plus gas coming from alveolii with high ventilation-perfusion ratios. At rest the anatomic dead space is fairly constant (West 1970), but on exercise the bronchi, under sympathetic control, widen to facilitate the flow of air and thus enlarges the volume of the anatomic portion of the dead

space. Reductions in sympathetic activity following training may be responsible for diminished dead-space, but according to Cumming (personal communication) the decrease in volume is unlikely to be much greater than 20 ml. is difficult to measure and is best ignored. Slower respiratory frequency would also reduce dead space and this situation is apparent at standard work on the treadmill, but the overall breathing pattern on both treadmill and cycle remained virtually unaltered and other mechanisms need to be proposed.

According to West (1970) alveolar dead space - i. e. the portion of the dead space volume not occupied by the conducting tubes - is caused by inequality of blood flow to the lung. Recent work by Cumming and D'Amato (1976 - awaiting publication) goes further and suggest that alveolar dead space can be divided into oxygen, carbon dioxide and nitrogen compartments and that the compartments can be seen as measures of inequality of ventilation, perfusion and gas exchange distribution respectively. If changes in anatomic dead space brought about by training are ignored it may be inferred that training rearranges the inequality of blood flow in some way thereby reducing dead space. The question of perfusion is considered further in a later section.

The new work by Cumming and D'Amato may illuminate changes in ventilation during exercise. Their method requires detailed analysis of a breath by breath record of a subject breathing air; expired gas volumes are plotted against tidal volume the intercept of the regression line on the V_T coordinate being the volume of anatomic dead space and the slope being used to calculate alveolar dead space.

The findings were discovered too late for inclusion in the design of this experiment but a similar, though less precise approach is contained within the computer programme. Regression equations for a variety of variables were derived for each individual (Appendix I);

Although the methods are not strictly comparable - these subjects were exercising not resting, computer sampling rate was slower (0.1 sec. compared to 0.03 sec) - changes in the slope of the V_T/V_{02} line giving greater oxygen uptake per breath for some subjects may indicate improved lung function. More confident assertions must await more precise analysis however.

Another method of assessing changes in lung efficiency during exercise makes use of analysis of the changing gas concentrations during the course of a breath. The plot of F_{E02} against V_T of a single breath produces the familiar 'S' shaped curve; no change in $F_{E\,02}$ as the anatomical dead space is washed out, rapid increase as the mixed expired air is expelled followed by a plateau as the alveolar air is reached. The slope of the mixed expired gas is taken as an indication of the uneven nature of gas flow in the human lung and is present in the normal healthy adult. It is the relationship between this slope and the end-tidal gas fraction which can be used to show changes in gas mixing efficiency. The method is summarised in the accompanying diagram:

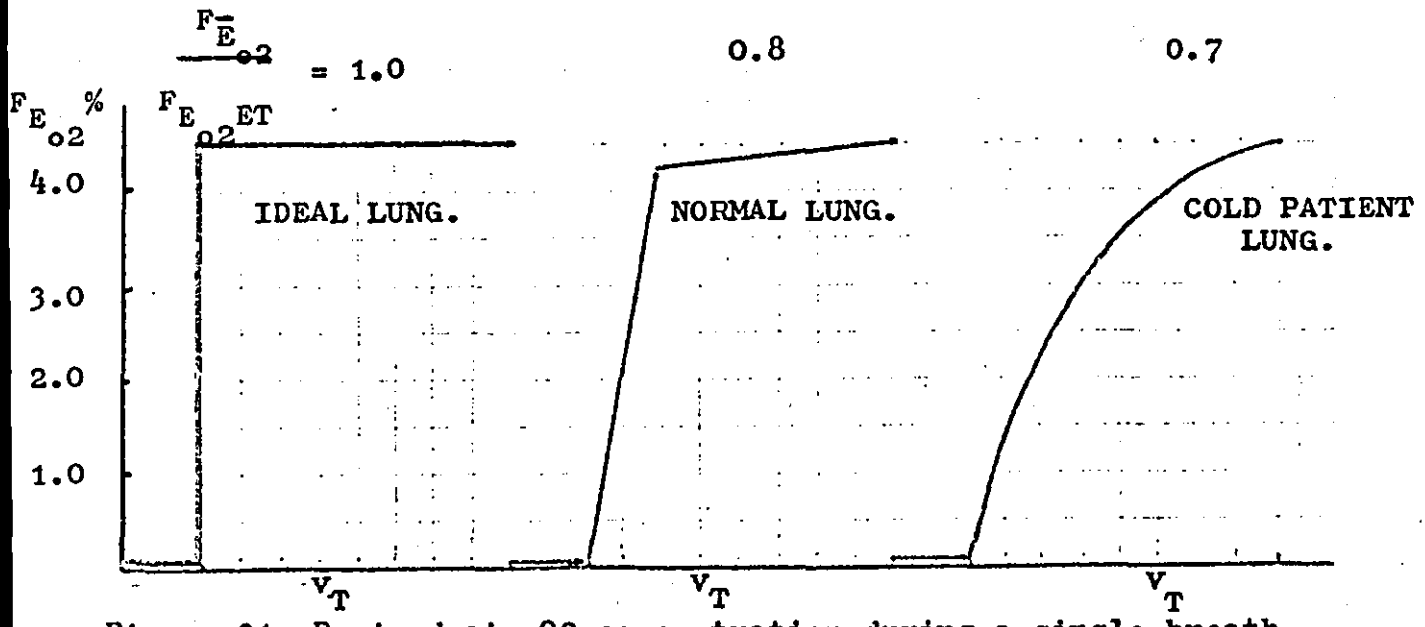


Figure 31. Expired air 02 concentration during a single breath.

The three traces are stylised representations of breaths from an ideal lung where transit time variations do not exist, a normal lung and a lung of a COLD patient. If the highest concentration achieved is measured i. e. $F_{E02\ ET}$ and divided into the mean of the remaining concentration measures i. e. F_{E02} 1 the ratio is unity in the ideal lung becoming progressively less as the lung becomes less ideal. Theoretically, providing V_T and time remained constant any increase in the patient's $F_{\bar{E}02}/F_{E02\ ET}$ ratio would imply a move towards more normal, i. e. more efficient, gas mi xing characteristics. Casual observation of breathing patterns during a work test indicate that neither time nore volume is fixed, but if results over a standard time, e. g. one minute, are used then the time factor becomes fixed, and if the mean V_T changes little then the actual conditions come close to the theoretical criteria and the ratio may be a useful indicator.

In the event the post-training ratio of mean mixed expired $F_{\bar{E}02}$ to mean end-tidal F_{E02} increased during maximal work on the treadmill from 0. 64 to 0. 67 (V_T rose by less than 2%) and from 0. 65 to 0. 67 on the bicycle (V_T increasing by less than 5%); values also increased at standard work from 0. 63 to 0. 69 on the treadmill (V_T again less than 5% higher) and from 0. 63 to 0. 65 on the bicycle where V_T actually fell very slightly (1%).

The ventilatory equivalent for oxygen (V_{E02}) is considered by some workers (Armstrong et al 1966, Kao 1974) to be an important physiological variable, and it too can be used to detect enhanced ventilatory mechanisms. It is the ratio between total ventilation and oxygen consumption (V/V_{02}) and, under normal resting or moderately active conditions, has a value ranging from 22 - 25, but under maximal

work may increase to 30 - 35. Any reduction in the value, it is argued indicates an improved ability to extract oxygen; maximal results in this study show a slight increase in the value on the bicycle (30.3 - 30.6), but a moderate reduction on the treadmill (25.8 - 24.8). If V_A is substituted in the equation, and this seems a legitimate change, a more accurate indication of change will result. Obviously the value itself is smaller, but interestingly when applied to the results indicates an improvement in favour of the bicycle - the value fell slightly from 20.9 - 20.6, but rose from 17.3 - 17.5 on the treadmill.

The changes described here may be indicative of improved ventilatory function, but unless the ventilation can be matched with perfusion the potential benefit is lost. As stated West (1970) and many others believe that inequality of blood flow causes increased dead space, the generally reduced dead space values reported at various levels on both bicycle and treadmill suggests that lung perfusion characteristics are affected by training. Cumming and D'Amato feel that carbon dioxide excretion holds the key to information on perfusion but because this particular section is devoted to oxygen consideration the $C0_2$-perfusion relationship will be covered later.

The next link in the oxygen-transport chain is the performance of the heart and this is considered below.

Cardiac performance

There are three reasons for suggesting that cardiac performance is improved, if only slightly. Firstly $f_{H\ max}$ was fundamentally unchanged on both bicycle (1.2 b. min. higher) and treadmill (3.3. b. min lower) in spite of the 19% increase in work rate observed on the bicycle and the 23% increase in the treadmill gradient. The mean work-pulse on the bicycle rose

by 20% from 0.5 W b. to 0.6 W b. as a result.

Secondly, oxygen pulse at maximum work rose very slightly on treadmill (2%) and bicycle (4.6%). Wasserman and Whipp (1975) argue that since $\dot{V}_{02} = \dot{Q}. (Ca_{02} - Cv_{02})$ and $\dot{Q} = f_H . SV$, $\dot{V}o_2/f_H$ (oxygen pulse) $= SV (Ca_{02} - C_{V_{02}})$ and is therefore a useful index of cardiac performance.

Thirdly, mean daily training heart rate remained fairly steady as mentioned earlier; the variation is generally less than 5 beads over a great part of the training programme. But the average speed and gradient of the treadmill during the last week of training was markedly greater; mean speed had increased by 36.7% and gradient had almost doubled from 4.2^{o} to 8.0^{o}. Mean f_{Ht} at the same periods were 112 b. min during week 1, 114 b. min during week 4, a small increase when related to the greater work load imposed.

Peripheral oxygen extraction

The non-invasive nature of the study means that no data are available on the effects of training on oxygen estraction rate at tissue level, but it is worth noting that preferential distribution of blood flow to working muscle is a well-documented finding (Astraud & Rodahl 1970), and Holloszy (1967, 1971) and Gollnick et al (1972) report that in man and animal the capacity of mitochondrial fractions from trained muscle (skeletal and cardiac) to oxidise pyruvate is increased. The resultant increase in $a\text{-}V_{02}$ difference is a common finding in normals (Andrew et al 1966) has been observed in trained cardiac patients (Varnauslcas et al 1966) and more significantly in COLD patient studies. In the opinion of Guthrie et al (1970), Degre et al (1974) and Paez et al (1967) increased $a\text{-}\bar{V}_{02}$ difference was entirely responsible for the increased work observed. The last named study is particularly interesting; it is the only other study in which both treadmill and bicycle were employed to train

and test the patients and the length of the study was similar, and Paez and his co-workers report that greater oxygen extraction at muscle level appears to be the principle non-specific transferable effect of training. A similar situation may apply to the experimental group in the present study.

The ultimate result of these changes should be an increase in the symptom limited $\dot{V}_{02}$. Pierce et al (1964) showed an average increase of 22.5% in post-training $\dot{V}_{02\ SL}$; a similar improvement was reported by Christie (1968). More modest gains, averaging 10% were observed by Vyas et al (1971) and Degre et al (1974). On the other hand Woolf et al (1969) noted a 7% reduction in $\dot{V}_{02\ SL}$ after training; Nicholas et al (1970) reported no change after training, but did reveal an increase following an habituation process; Paez and his colleagues describe a modest increase - 1.5% in $\dot{V}_{02\ SL}$ on the bicycle following treadmill training. Casual inspection of previous studies reveal inter-patient variability in $\dot{V}_{02\ SL}$ responses to training, and Degre and his co-workers comment that range of improvement is quite wide from 0 - 35%.

In the present study post-training changes in $\dot{V}_{02\ SL}$ were very similar to those reported by Paez et al (1969); mean treadmill value was virtually unchanged, the difference amounting to a fall of less than 1%, and although individual improvements on the bicycle ranged from 4 - 15%, two subjects produced lower values and the mean increase was 5.1%.

A small change in symptom limited $\dot{V}_{02}$ is a common finding in training studies on both normal and patient groups. Claims for improved oxygen delivery during studies on IHD patients have been made frequently on the basis of indirect measures; for example increased oxygen pulse (Gottheiner 1966, 1968), reduction in RQ at standard work

(Clausen et al 1970), and predicted $\dot{V}_{02\ max}$ (Kavanagh et al 1970). But Mazzarella et al (1965) measuring $\dot{V}_{02}$ directly, report changes ranging from 2.3 ml. - 8.6 ml. kg. min. only. Furthermore in the majority of studies, (Paez being an exception), training has generally extended over a period longer than four weeks provided in this study, and it is possible that a longer period is necessary for the changes in the oxygen-chain to become optimally employed.

Therefore the variability in individual post-training $\dot{V}_{02SL}$ in the face of almost unanimous improvement in work on both treadmill and bicycle leads to the suggestion of more efficient use of a pathologically fixed oxygen reserve. There is evidence, particularly in the treadmill results, to support the theory. Maximal values are basically unchanged, although $\dot{V}_E$ fell 4% due to slower f_R, and $\dot{V}_{02SL}$ fell marginally, but when aggregate values are related to time both ventilation and oxygen uptake fell 14% and 10% respectively. At standard values instances of greater efficiency are even more striking; at standard work f_R fell 20%, $\dot{V}_E$ 17% and $\dot{V}_{02}$ 11%, and although V_A also fell (10%) there was compensation in the form of a 30% reduction in V_{0_2}; at standard V_T 0.5 l, V_A increases 12%, V_D fell 18% and with increased mixed expired F_{E02} (5%) V_{02} per breath increased 10%. Even at V_T 0.8 l., i.e. similar to the mean maximum V_T, V_A increased 5%, V_D fell 14% and oxygen delivery per breath rose 4%.

Evidence of greater efficiency during ergometer pedalling is available but it is less impressive, and restricted to levels observed during standard work. Here oxygen demand was reduced slightly (3%) and $\dot{V}_E$, $\dot{V}_A$ and $\dot{V}_D$ all fell between 5 and 10%.

The principal cause of reduced metabolic demand during

treadmill walking is almost certainly the improved neuro-muscular coordination already described earlier. An additional stimulus, and one which would apply to the results at standard bicycle work, is a reduction in oxygen demand through a fall in ventilation. Estimates of the oxygen cost of breathing vary from 0.5% (Kao 1974) to 1.5% (Cotes 1968) of resting oxygen consumption; increased ventilation raises the cost - 3% of total $\dot{V}_{02}$ at a $\dot{V}_E$ of 40 l. min according to Cotes. At high rates of ventilation the oxygen cost increases disproportionately until a situation arises where cost is greater than supply. All of this relates to normal subjects; with increased respiratory impedance the cost is much higher than normal at rest, and according to Kao over 200 ml. of oxygen is used by respiratory muscles of emphysema patients at a rate of 20 l. min.

Thus any reduction in ventilation cost, either by lower rate or by an increase in respiratory muscle efficiency, could influence work performance. Where increased work is accompanied by lower ventilation it is tempting to suggest that the oxygen released from its burden of paying for breathing is available for the increased demand of the work - a situation which might apply to the treadmill. But equally where higher work rate is accompanied by increased ventilation - the bicycle situation in this case - any increase in V_{02} must include oxygen to pay for that greater ventilation; the possibility that the increased $\dot{V}_{02SL}$ noted on the bicycle is largely taken up by the increased cost of ventilation must at least be considered.

The logic of the concern with oxygen availability is clear, but in the face of the modest changes seen in this and other studies it seems reasonable to examine other variables which affect exercise tolerance. One issue which, surprisingly, has received little attention in any study is consideration of changes in C02 handling characteristics of trained people. The following

section deals with this topic.

Enhanced carbon dioxide excretion as a factor in increased exercise tolerance

It is known that in normals exercise tolerance is reduced when Pa_{C02} is raised through addition of C02 to inspired gas, and if hypoxemia is one of the problems facing COLD patients hypercapnia is frequently another. Exercise exacerbates the situation; incomplete respiratory compensation for metabolic acidosis leads to inadequate clearance of C02 and thence to raised Pa_{C02}. An improvement in C02 excretion may positively influence exercise tolerance therefore.

Evidence of increased post-training C02 elearance is to be found in earlier investigations; Pierce et al (1964) describe a 28% increase in $\dot{V}_{C02\ max}$ without further comment; Paez et al (1967) do not report $\dot{V}_{C02}$ measures, but a 4% reduction in Pa_{C02} accompanied by a decrease in $\dot{V}_E$ suggests enhanced C02 excretion; Vyas et al (1971) report increases of 10% in $\dot{V}_{C02\ max}$ and 22% in aggregate terms but add no comment.

In the present investigation improved C02 clearance on both treadmill and bicycle is evident. Treadmill results showed increases in aggregate values (13.4%) maximal values (4.4%) and mean single breath (10%); on the bicycle increases at the same levels were 32.2%, 8% and 3% respectively.

To seek out possible mechanisms by which this consistent improvement occurs we need to return to the relationship between ventilation and perfusion. Woolf et al (1969) and Degre et al (1964) believe that improved ventilation - perfusion mechanics are partly responsible for the increased work tolerance reported in their studies. The former group believe that increased ventilation occurs in alveolii that are poorly perfused-giving rise to increased dead space - and also in alveolii which were poorly

ventilated but had retained good perfusion; as a result A-a gradients are improved and venous admmixture is reduced.

In the present study evidence of reductions in V_D at various levels, particularly the large reductions at standard work when steady state conditions are most likely to exist, suggests that lung perfusion characteristics may have been affected by training. The increased C02 excretion observed can support this suggestion; the arguement is as follows:-

a) V_{C02} can be increased if perfusion characteristics remain unchanged providing V_E is increased; this would reduce Pa_{C02}. No direct data are available on Pa_{C02} levels because of the non-invasive nature of the study, but indirect evidence in the form of end-tidal F_{EC02} tentatively points to a slight increase in Pa_{C02}. Furthermore V_{C02} seemed independent of changes in ventilation, since values were higher whether V_E rose, as at maximal bicycle work, fell, as at maximal treadmill work, or, more significantly, remained constant as at standard V_T on the two machines.

b) V_{C02} may be increased if perfusion characteristics remained unchanged V_E remained constant but f_R slowed sufficiently to allow greater diffusion time, but f_{Rmax} remained fundamentally unaltered - 28.0 - 27.1 b. min on the treadmill 25.7 - 26.0 on the bicycle.

c) V_{C02} would increase if perfusion characteristics remained unchanged, f_R and V_E were constant but Pa_{C02} increased. Such an increase could result from two sources. Firstly increased C02 derived from greater aerobic activity, but there is scant evidence that aerobic capacity was materially altered. Secondly through anaerobic mechanism if buffering of lactic acid occurs via the bicarbonate system as in the schematized reaction:

$$Na^+HC03^- + H^+ \text{ lactate} \longrightarrow Na^+ \text{ lactate} + H_2C0_3 \rightarrow C0_2 + H_20$$

This second mechanism would account for the tentative suggestion already made that Pa_{C02} may have increased and would therefore explain the increases in V_{C02} (Increased anaerobic metabolism would also be a factor in increased working time of course but without direct measures of blood lactate levels the suggestion is speculative).

Increased Pa_{C02} would be reflected in increases in the slope of the V_T/V_{C02} regression line, but such an increase could also hide improved perfusion characteristics. Using a crude approximation of Cumming and D'Amato's method for determining alveolar ventilation and efficiency, i. e.

$$\frac{1}{F_{EC02\,er}} - \frac{1}{F_{\bar{E}C02}} \times V_{C02} = \text{C02 dead space per breath}$$

Alveolar ventilation - C02 dead space /alveolar ventilation = alveolar efficiency , alveolar efficiency tended to improve; if the assertion that C02 dead space reflects inequality of perfusion is correct improved alveolar efficiency may suggest changes in lung perfusion characteristicas. However, the method employed was so imprecise and the improvements so small that no great confidence can be placed in the results obtained, but greater precision in analysis may provide more reliable information and reorganisation of the programme is taking place at the present time.

The mecnahism by which perfusion may be altered concerns broncho-vaso control. Carbon dioxide is a known vasodilator, and Cotes (1968) describes evidence of the broncho-constrictor effect of hypocapnia as follows; 'initially fewer molecules of carbon dioxide pass from the alveolar capillaries into the alveoli of the affected region; the tension of carbon dioxide in the gas leaving such alveoli is therefore reduced below

that in the walls of the bronchi through which the gas is flowing. Carbon dioxide then passes outwards from the tissue of the bronchi into the lumen so that the tension of carbon dioxide in the vicinity of bronchial muscle fibres decreases. This change increases the tone of the muscles which, in turn, reduces the diameter of the lumen'. Add to this the dilatory effects of exercise produced catecholamines and a plausible proposition emerges. Stated baldly it says that the combined effects of the relative hypercapnia and enhanced catecholamine production of exercise induces both vaso and broncho-dilation, which leads to improved perfusion of already ventilated alveoli, increased C02 excretion and a greater exercise tolerance.

The lack of significant increase in V_{02} does not diminish the proposition. The argument is in two parts. Firstly Sa_{02} is not significantly altered by improving perfusion to alveoli with already adequate ventilation; the oxygen content of blood perfusion poorly ventilated alveoli is markedly reduced; but blood draining alveoli with high ventilation-perfusion ratios is already heavily loaded with oxygen and any more restoring V_A/Q towards normal will not influence Sa_{02} greatly (West 1970), Secondly even if Sa_{02} increased as a result of enhanced VA/Q ratios V_{02} would not necessarily increase because cellular metabolic activity is also required to process the additional oxygen.

Summarising this section on physiological aspects the following points can be made:-

a) Increased aerobic capacity seemed to play no part in the improved exercise tolerance seen on the treadmill, but may have been a factor in the greater work output observed on the bicycle.

b) Reduced metabolic demand, brought about by improved

technique, leading to physiological efficiency seems to be important in enhancing treadmill performance, but the effect is restricted largely to the one machine.

c) Anaerobic mechanisms may have played a part in improved performance, but there is no direct evidence to support this, and in the absence of markedly increased post-training work test $f_{H\ max}$ the question must remain unresolved.

d) Consistently increased C02 excretion, irrespective of changes in minute ventilation, respiratory rate or oxygen uptake, is the common feature of post training work-tests on both devices; increased Pa_{C02} is the most likely cause of the increase but improved perfusion of adequately ventilated areas of the lung is another possibility which needs further investigation.

CHAPTER TEN

Discussion: The control group

As stated previously, adequately controlled studies of the importance of physical activity in the rehabilitation of patients with cardio-respiratory disease are generally lacking. Kavanagh & Shephard (1973), reviewing post-coronary rehabilitation studies, state that comparisons between active and non-active 'favour the exercised groups, but it remains uncertain how far the observed benefits can be attributed to exercise per se.' Among other possible factors the authors list support of an interested group, the control of smoking and obesity in those who undertake regular exercise, the presence or absence of severe disease and an unfavourable life style in those who do not exercise. An addition to this list, as far as COLD patients are concerned, is exercise-phobia; as a result of repeated attacks of exertional dyspnoea a conditioned avoidance of exercise is almost certainly developed over a period of time, and it seemed possible that mere exposure to supervised activity might produce improved performance. A straightforward comparison between the active experimental group and a non-exercising control group seemed inappropriate, even though the procedure is commonly adopted - in the majority of patient studies.

The alternative approach used in this study was to give the control group an ineffective dose of exercise - in other words an exercise-placebo. The purpose was to investigate the influence that removal of anxiety had on exercise tolerance, the hypothesis being that it is the volume, duration and intensity of the exercise rather than the exercise itself which produces

increased work. The fact that this kind of approach to the control group is rarely employed justifies the inclusion of this short section in the discussion.

The attempt to treat the control group more realistically was only partially successful. The group was smaller in number, lighter by an average of 6 kg. and slightly taller than the experimental group; more importantly work tolerance was almost 30% lower at the beginning of the experiment and the mean number of training exposures was 14. 6 sessions compared to 19. 1 for group E. No attempt was made to match the groups for age, weight habits, occupations, social background or severity of incapacity; but as described in the methods section allocation to the two groups was a matter of chance, and since each group acted as its own control the experiment may be seen as two separate studies, one investigating the effects of intense physical training, the other investigating the effects of an exercise-placebo.

Two problems were envisaged. Firstly in view of the low initial work tolerance commonly associated with COLD it is difficult to devise a programme which will not improve general fitness; secondly the patient may be stimulated to increase his normal activity pattern to such an extent that the higher levels of HPA could be a training stimulus and over-ride the placebo effects of treadmill walking at low intensity and duration.

In the first case the minimum training heart rate concept, originally quantified by Karvonen et al (1957) and later refined by Roshamm (1967), was used to determine individual minimum f_{Ht} ; the work was then set to ensure f_{Ht} rarely rose above the threshold level. This was achieved through very short sessions - no more than 30 seconds - of level grade walking at a speed no greater than the test speed, followed by unnecessarily long recovery periods, varying between 1 and 1. 5 minutes occupied by general conversation. So, although exposure each session to the laboratory environment and the

supervising staff was almost equal for the two groups the actual working time was not. The experimental group completed 110 training sessions, with a mean training time of 30.6 minutes per session; with one exception (E2), f_{Ht} exceeded threshold levels on all but four occasions. Training sessions for the controls were fewer (73), and shorter - mean time 5.0 minutes, but more importantly f_{Ht} for this group was below threshold on all but four occasions. The calculated minimum f_{Ht} for group C was 98 b.min, but the mean level achieved was only 91 b.min; only on the first training session did mean f_{Ht} exceed the threshold and then by less than 5 b.minute. One subject (C2) reported that he was capable of much more work during training but was willing to continue when told that the objective was to establish the minimum training requirements.

Monitoring the level of HPA outside the programmed was eased by the fact that all the subjects were inpatients for the duration of the programme and any marked change in activity pattern would have been noted by the nursing staff; secondly only two subjects (C2 and C3) made positive efforts to change this general pattern of activity, subjectively confirmed by nursing staff; one C2, kept detailed records of these activities whilst C3 made a ritual of a daily 30 minute walk in the grounds.

The results of these two subjects are interesting. In spite of having the greatest number of treadmill sessions, 22, the longest mean training time - 7.5 minutes per session, and the greatest increase in general activity - 26 sessions ; the post-placebo work times of subject C2 actually fell 10% on the ergometer and 6% on the treadmill. On the other hand C3, with only 11 treadmill sessions, a mean training time of 3.0 minutes per session and a daily 30 minute walk, increased ergometer work time by 12% and treadmill time by 74% (419 - 730 secs).;

Two other problems blur the issue further. Firstly only three subjects were able to complete the final treadmill tests, and secondly the post-placebo working time for one subject fell 50% on the ergometer and 69% on the treadmill. Therefore pre and post programme treadmill results cannot be compared meaningfully, and also the large decrease in ergometer working time, and hence in other variables, shown by C4 has an over-bearing influence on the group mean and tends to weaken the general view that the placebo had little effect on work tolerance.

However, if the results of C4's tests are ignored the evidence still favours the hypothesis that the placebo training does not change fundamental physiology in any marked way. Aggregate values now show a slight increase in bicycle time (4. 5%) and work achieved (7. 6%), a 4% drop in the total number of heart beats and a 6% fall in minute ventilation; these changes are reminiscent of typical training responses, but when they are linked with a 10% increase in f_R and reductions of 11% in V_{02} and 19% in V_{C02} the similarity disappears. Apart from the increase in work time, which amounts to no more than 15 seconds, maximal values are still consistently lower following the placebo training. At single breath level V_T, V_{02}, V_{C02} and V_A are all reduced by more than 10% and V_D increased 4% - undoubtedly the faster breathing rate is a factor here. Values at standard V_T are also generally lower.

Clearly even the minimal increase in control group activity combined with the greater degree of daily attention the programme entailed was sufficient to produce increases in pedalling time in three cases. But except for subject C4 the increases are small (10 seconds for C1, 33 secs. for C5) and more significantly are not accompanied by increases in V_{02} or V_{C02}. They are linked with reduced $f_{H\ max}$ and $f_{R\ max}$ however and this might

indicate a reduction of anxiety, greater relaxation and a resultant increase in work time. In other words the placebo programme seems to act in a similar fashion to the habituation procedure and does not alter the physiological function of the patient.

CHAPTER ELEVEN

Discussion: Implications of this study for future programmes

This study, in common with many others, confirms the view expressed by Petty (1975) that some potential for physiological adaptation is retained by many COLD patients. The majority of the experimental group felt that the programme was worthwhile and was instrumental in producing enhanced feelings of well-being. Two subjects, E1 and E3 showed willingness to undertake regular 3 mile walks in addition to their daily dose of treadmill walking even though it was exertional dyspnoea during walking which first led them to seek medical help. Reduced anxiety about crossing the road and renewed enthusiasm for gardening was expressed by one patient (E7); a desire to return for regular 'topping-up' sessions was declared by two subjects (E5 and E6), and even E4 (who failed to improve his ergometer work) was convinced that the programme was generally beneficial. One subject (E5) felt less depressed as a result of the programme, and E2, the most impaired of the experimental group, was prevented from taking part in a shopping expedition only by the concern shown by his partner. A euphoric over-reaction by E7 resulted in an acute episode of bronchial infection requiring a short spell of hospital care; this occurred after the end of the training programme but did little to diminish the mans convinction that exercise and well-being are closely related. It did serve to remind him of the need to temper enthuriasm with common sense however.

On the basis of such comments, combined with the need to confirm the physiological trends demonstrated here, suggestions which may improve the effectiveness of future programmes can be put forward.

In my view the most important suggestion relates to the actively involved in the training programme. There is evidence from this study which confirms the report from Paez et al (1967), that training can result in the development of largely non-transferable improvements in neuro-muscular coordination. This is seen particularly in treadmill walking, but there are suggestions in the data of Baso et al (1970) and Vyas et al (1971) that a similar process, perhaps less marked, may occur during training on a bicycle ergometer; personal experience also confirms this latter adaptation.

Two issues develop from this: the first is that COLD patients may be able to perform specific repetitive actions more efficiently if practice is provided. Of course the potential improvement is finite, but the application of the procedure to employment, occupational-therapy and everyday activities may go some way to improve the life style of certain patients. The second is that a variety of exercise situations is more likely to produce a general improvement in physiological function than is possible on either treadmill or bicycle alone. It is worth noting that the largest increase in post-training V_{02SL} - over 20% - was reported by Christie (1968) who employed a variety of activities within his programme, and it is also worth recalling the comments of the more impaired members of the group when comparing treadmill training with walking outside or upstairs; invariably they felt that treadmill walking had become much easier as the programme progressed but in walking up slopes outside the laboratory a similar degree of improvement was not detectable. Thus mixed activity programmes like those described by Christie (1968) and more particularly by Hass et al (1969) are worthy of further investigation, and continued study of responses to bicycle or

treadmill training alone are liable to be counter-productive.

The next suggestion refers to the intensity of the exercise programme. The results from the placebo programme imply that although enhanced attention and loving care may be a feature in improving work tolerance the effect is rather limited. The activity patterns must be physically demanding and therefore supervised and monitored adequately. Without supervision and monitoring the level of patient motivation is likely to fluctuate markedly, the safety of the patient is not secured and the progressive quality of the programme is difficult to maintain. An important step will be to monitor the intensity of everyday activities so that a useful mixed programme can be devised for each patient.

The third suggestion concerns the minimum time given to supervised training. It is possible that one month or twenty sessions is not sufficient time for the individual potential to be fully realised, and this study is certainly one of the shortest in terms of the number of training sessions. The assertion by Smodlaka et al (1974) that three months is the minimal time needed for training effects to emerge is worth adopting.

Fourthly, much closer attention must be given to test procedures. The comparison of pre-training treadmill and bicycle work tests, described at the beginning of chapter seven, underlines the need for caution in describing maximum capacity of COLD patients when less than whole body movement is involved. Maximum bicycle work responses considerably under-estimated ventilatory capacities in at least three important variables, namely $\dot{V}_E$, $\dot{V}_{02}$, and $\dot{V}_{C02}$; thus if work tests are to become meaningful diagnostic tools comparison of a group or an individual against population values is only realistic if the exercise mode is the same. The implications are that at least three sets of normal values are necessary -

one for each device, treadmill bicycle and steps. In addition the information obtained from a breath by breath analysis of a work test is more likely to permit clearer understanding of the effects of training; in this study changes at mean single breath and standard tidal volume were useful indicators of changed function particularly when gross changes were small. A great deal of work still needs to be done in this particular field however.

Finally, if a 'de-sensitisation' process is at work there is a need, in common with other protective mechanisms such as immunisation and vaccination, to administer regular 'booster' doses of exercise. This could be achieved by the patient himself maintaining an appropriate level of habitual physical activity with a daily period of time set aside specifically for walking or stain climbing. The evidence from tape recordings and telemeter data indicates that a walk in the hospital grounds was often sufficient to raise heart rate above the training threshold, was sufficiently stressful in dyspnoeic terms to cause patients to stop in order to recover and is therefore a potent force in the desensitisation process, and is an activity which patients see as being relevant to improved life style. Not all patients are sufficiently well-motivated to do this on their own on a regular basis; therefore a twice-yearly visit for a 'booster' dose may be required, and on the evidence of some subjects welcomed.

On the basis of these five suggestions it is impossible to agree with the view of others (Christie 1968, Woolf et al 1969) that programmes may be run on an outpatient basis with little more supervision than is usual in any clinical practice, or that programmes can be simple. The exercises may be simple but the supervising and monitoring is demanding and time consuming and would add a considerable burden to any outpatient clinic.

CONCLUSIONS

This investigation in common with many others demonstrates that a controlled progressive exercise regime leads to a significant improvement in the exercise tolerance of patients with chronic obstructive lung disease. The mechanisms by which such improvements occur are not yet clear but greater tolerance to the distress of dyspnoea, more efficient use of the xoygen reserve and increased anaerobic activity are three mechanisms which may be involved. On the evidence of this study enhanced symptom limited oxygen uptake appeared to be of little consequence but the marked increase in carbon dioxide excretion observed may be important and is worthy of further study.

It is clear that a short habituation programme reduces initial anxiety, and also that a supervised programme of light exercise can induce a feeling of enhanced well being irrespective of accompanying improvement in physiological function; but it is equally clear that increased work capacity can only be achieved by quite strenuous efforts - often near maximal - on the part of the patient and this needs to be clearly understood by the physician, the patient and the patient's family.

The trainability of COLD patients is confirmed by this investigation but the value of a training regime employing a single exercise mode is open to question; the purpose of exercise as a therapeutic agent is to improve the quality of life and this aim is more likely to be achieved if exercise regimes employ a wider range of activities than merely pedalling an ergometer or treadmill walking.

The patient is unlikely to be over-concerned with the mechanisms

by which his improvements arise, but for exercise therapy to be employed effectively some important questions still need to be investigated. These questions concern methods of assessing pulmonary function during work - attempts to study nitrogen wash-out during exercise are being made -, more precise definition and greater reliability in the measurement of psychological variables, greater accuracy in monitoring habitual physical activity patterns before, during and after training programmes, evaluation of longer and more imaginative exercise regimes and the effects of exercise oriented therapy on recurrence of illness, employment patterns, and mortality rates.

These are formidable tasks but in essence are similar to the problems facing the advocates of exercise therapy in ischaemic heart disease in the late 1940's. Almost thirty years later hardly an argument is raised against the principle of exercise-based rehabilitation for certain categories of heart disease patient, even though enthusiastic application of the principle throughout the United Kingdom has yet to be seen. Since the first study on COLD patients was conducted over thirteen years ago we may now be half-way to achieving a similar acceptance of exercise therapy in these patients.

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PART FOUR

APPENDICES

APPENDIX

A : Responses of patients of varying ages with non-specific lung disease to progressive work test.

APPENDIX A

SUBJECTS

28 males took part; 7 mature students at a college of education (C) and 21 patients attending King Edward VII Hospital, Midhurst (E). The latter group was subsequently sub-divided into E1 (n = 11), E2 (n = 11) E3 (n = 6) reasons for which are given later.

TABLE A1 ANTHROPOMETRIC DATA (Mean, S.D.)

	Age (years)	Weight (kg)
Group C	39.6	83.5
	5.21	7.58
Group E	37.5	74.2
	13.8	10.17

METHODS

Three methods were employed; all were sub-maximal tests but differed in work loading, gas collection and equipment used.

Method (1): Group C. Progressive test, discontinuous loads, measures taken during the 5th minute of each load.

Subjects pedalled on a bicycle ergometer (Monark) for 5 minutes at four increasing work loads (nominally 50, 100, 150 and 200 W) with a 5 minute rest period between each load. During the 5th minute of work heart rate (f_H) from chest leads, and respiratory rate (f_R) from a respiratory thermocouple inserted into the valve box were recorded (Ediswan Polygraph), work rate computed from the load and wheel revolutions, and expired air collected in a douglas bag (Plysu). Volume of the expired air was measured by passing the contents of the bag through a dry gas meter (Parkinson-Cowan) and analysed for P_{02} (Beckman D25).

Oxygen uptake (V_{02}STPD) was then calculated. From the V_{02} and f_H recordings at each work load correlation coefficients (Pearson product-moment) and regression lines (least squares method) were derived for each subject.

Results

Maximum levels achieved (mean $\pm$ S.D.) and predicted values are given below:

TABLE A2 MAXIMUM PHYSIOLOGICAL RESPONSES TO WORK TEST (N = 7)

	W w	$f_{H\ max}$ b.min	V_E L.min BTPS	f_R b. min	V_{O_2} L.min STPD	V_{O_2} ml.kg.mm STPD
MEAN	1313.9	168	72.958	28.24	2.066	25.1
S.D.	208.90	13.9	16.647	8.426	0.443	5.16

TABLE A3 PREDICTED VALUES

	'r'	V_{O_2} f_H 130 L.min STPD	f_H V_{O_2} 1.5 b.min
MEAN	+0.950	1.408	108.5
S.D.	0.056	0.388	21.95

These predicted results are similar to those of a comparable group studied by Groszez (Figure A4.) The $\dot{V}_{02}/f_H$ intercept is approximately the same, and although the slope of the regression line of Group C is less steep, the difference is small (1.2%) and suggests that for the purposes of this experiment Group C is a typical group against which the patient group could be compared.

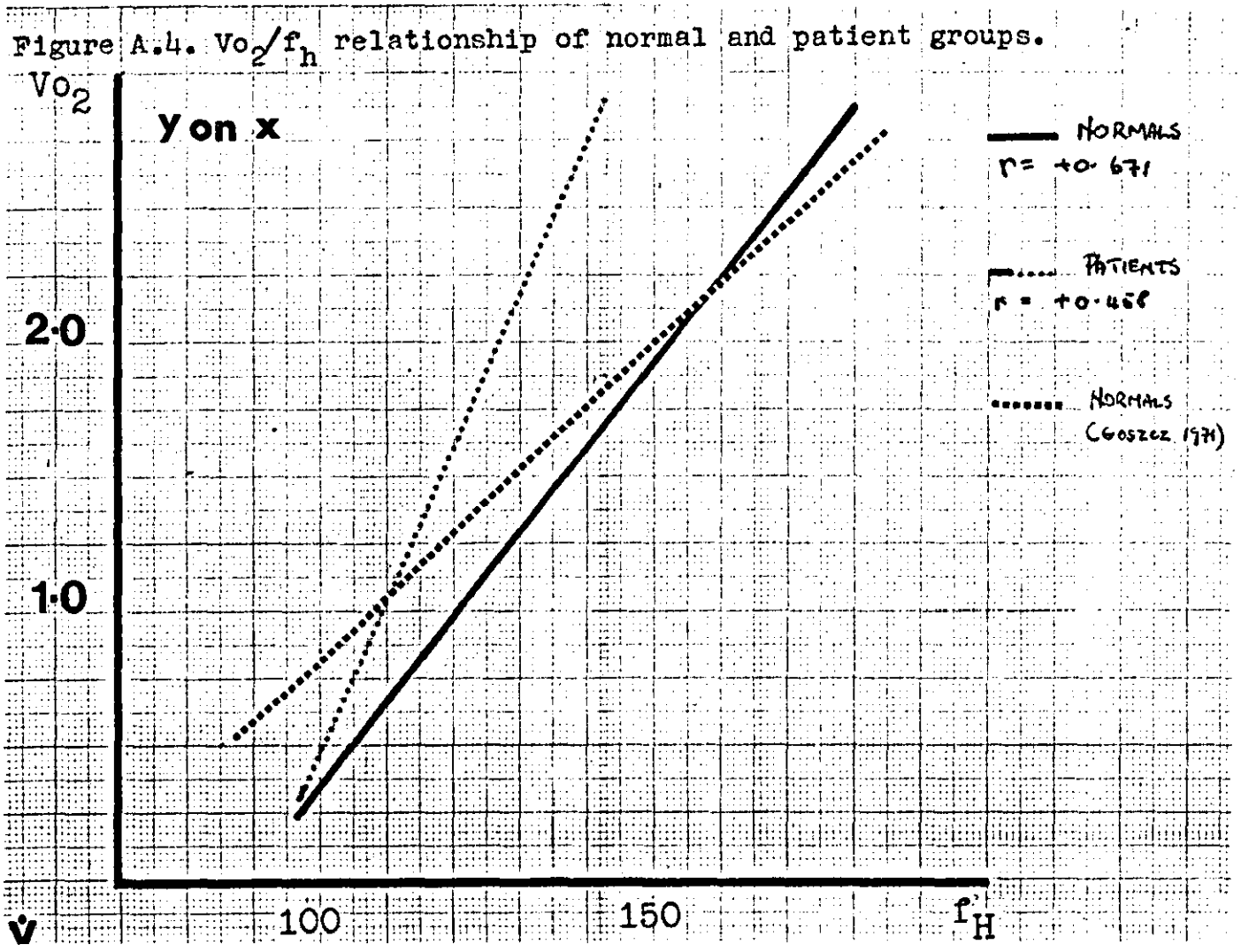
This method, with work rates suitably reduced, was used with two elderly patients. The first stopped work after 3.5 minutes pedalling before gas collections were made; the second completed one work load but was unable to continue. The following method was devised to counter the possible waste of data if patients were unable to complete work loads.

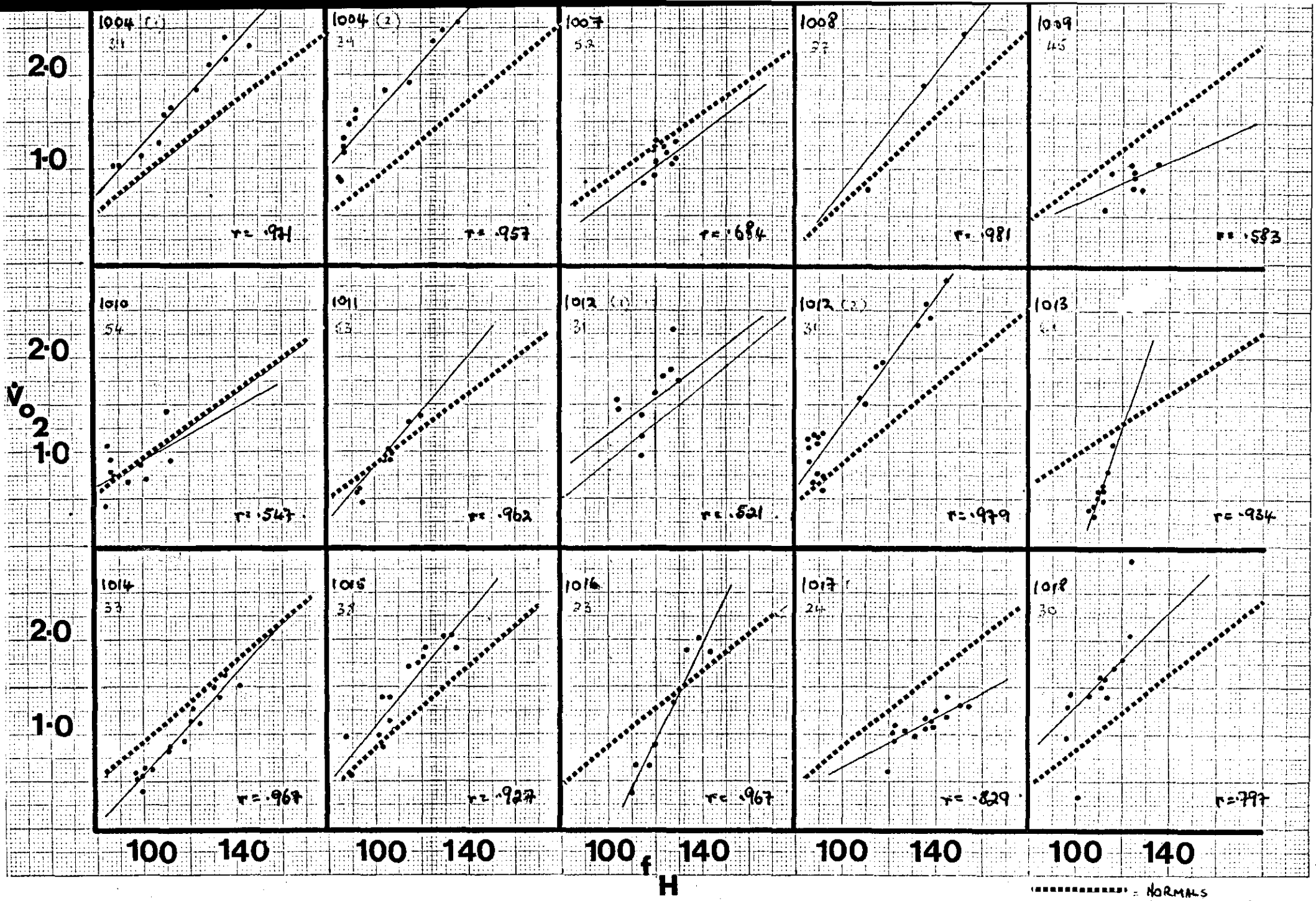
Method (2): Similar to (1), but measures were taken during the last 30 seconds of each minute of each load.

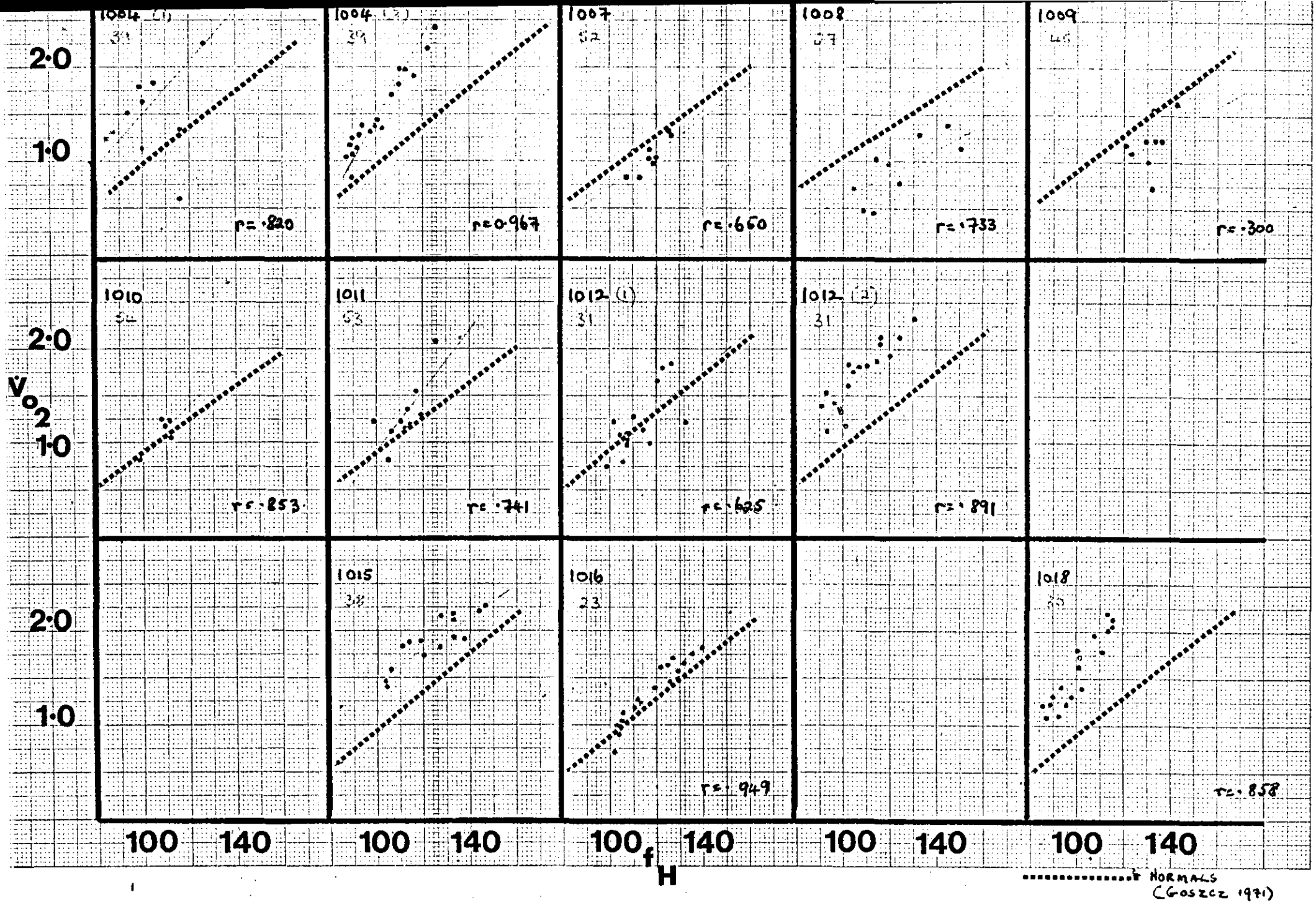
Heart rate and E.C.G. tracings were monitored continuously (Hewlett-Packard and Medelec). During the last 30 seconds of each minute f_H and f_R were recorded (Medelec) and expired air passed directly through a gas-meter (Parkinson Cowan CD4), a 60 ml. sample being drawn into a gas syringe from a part on the inlet side of the meter. Samples were later analysed by a mass spectrometer (Centronics) previously calibrated with a gas of known concentrations. Correlation coefficients and regression equations were obtained.

Subjects 1002 - 1912 (E1) were tested by this method, each completing two work tests - one on the bicycle, the second on the treadmill. Marked intra-variability was evident (Figure A2.) Low correlation coefficients were derived - mean + 0.75 $\pm$ 0.213, and the mean standard error of the estimate too large (3.94 l.) for the results to be treated with

Figure A.4. Vo_2/f_h relationship of normal and patient groups.




1004 (1)
34
r = .971
1004 (2)
34
r = .957
1007
52
r = .684
1008
27
r = .981
1009
45
r = .583
1010
54
r = .547
1011
43
r = .962
1012 (1)
31
r = .521
1012 (2)
31
r = .979
1013
r = .934
1014
33
r = .968
1015
37
r = .927
1016
23
r = .967
1017
24
r = .829
1018
30
r = .797
2·0
1·0
$\dot{V}_{O_2}$
100
140
f H
= NORMALS (Goszcz 1971)


1004 (1)
31
r=.820
1004 (2)
39
r=0.967
1007
52
r=.660
1008
47
r=.733
1009
46
r=.300
1010
54
r=.853
1011
53
r=.741
1012 (1)
31
r=.625
1012 (2)
31
r=.891
1015
38
1016
23
r=.949
1018
33
r=.858
2·0
1·0
$\dot{V}_{O_2}$
100
140
f_H
NORMALS
(Goszcz 1971)

confidence. Three reasons may account for this unreliability:

a) incomplete mixing of expired air leading to variable F_{E02} at similar f_H and work.

b) the rest interval, usually 5 minutes, may not have been long enough for some patients.

c) Anxiety, plus the short recovery period, may have produced higher than usual $f_H : V_{02}$ ratios, particularly at the low work rates employed.

Method (3): Progressive test, stepwise loading every four minutes without rest, measures taken during the last 30 seconds of each minute.

Work loads were increased stepwise every four minutes until f_H 150 ECG changes occurred, dyspnoea became intolerable, or the subject chose to stop working. A mixing chamber was attached to the outlet side of the meter, samples being drawn during the last 30 seconds of each minute, when f_H and f_R were also recorded.

Subjects 1013 - 1021 (E2) were tested by this method. Correlation coefficients improved, the mean coefficient $+0.934 \pm 0.042$ being significantly higher ($P < 0.05$) than for group E1.

The responses of groups C and E2 were compared Age and height of the groups were similar. Although maximum levels achieved during the work tests revealed significant differences in work rates ($P < 0.001$), f_{HW} ($P < 0.001$) and $\dot{V}_E$ ($P < 0.05$) - higher levels being achieved by group C in all cases - no differences were found in $\dot{V}_{02}$ (absolute or when related to body weight) nor in predicted values $\dot{V}_{02}$ fH^{150} $^{-1}$min. and f_H $V02$ 1.5 $^{-1}$ min. As higher f_{HW} and $\dot{V}_E$ would naturally occur because of the higher work rates achieved and work rate is not a reliable measure

of work capacity, the significant differences are meaningless and the regression equation appears incapable of differentiating between the sick and the healthy.

Four possible causes may exist.

a) Uneven sampling of the expired air may have resulted in inadequately mixed gases being analysed.

b) Inter and intra-individual variability in minute-to-minute response to work cannot be meaningfully compared to steady state measures.

c) Four work loads are considered necessary for reliable regression equations to be derived. Less than half of the patients completed four loads; in addition the early work loads were invariably light (< 25 watts) when V_{02} and f_H relationships are fairly unreliable, and anxiety can produce increased f_H.

d) Even the patients who completed four work loads (E3) may not have reached a true steady state in the time permitted (i.e. 4 minutes) before the next load was applied. Examination of work/f_H relationships reveal that only one subject (1018) reached an f_H plateau during the higher work rates (Figure A3).

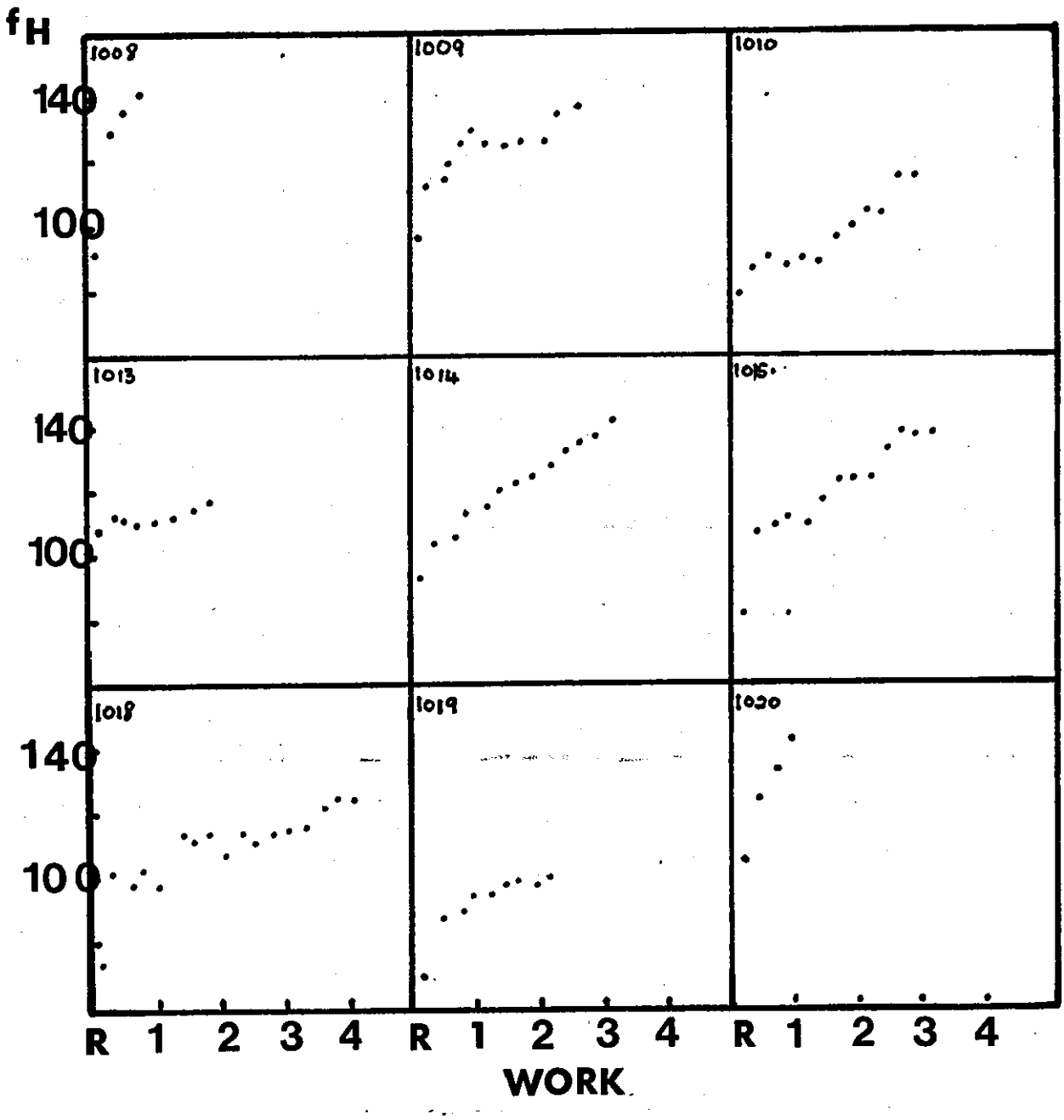


Figure A.5. Individual heart rate responses to increases in work load.

APPENDIX B A BRIEF DESCRIPTION OF THE RESPIRATORY MASS SPECTROMETER (from Nesarajah 1965)

The principles of the respiratory mass spectrometer (RMS) are as follows: A mixture of gases is ionised; gas molecules enter an ionisation chamber and are bombarded by a stream of electrons from a heated tungsten or thenium filament. The molecules give up an electron and become positively charged. The ions are then accelerated and pass through a narrow slit in an accelerator plate emerging as a narrow beam which is deflected along circular paths by a permanent magnet. For a given accelerating voltage and magnetic field, ions of different mass have trajectories with different radii of curvature. (Figure B1.)

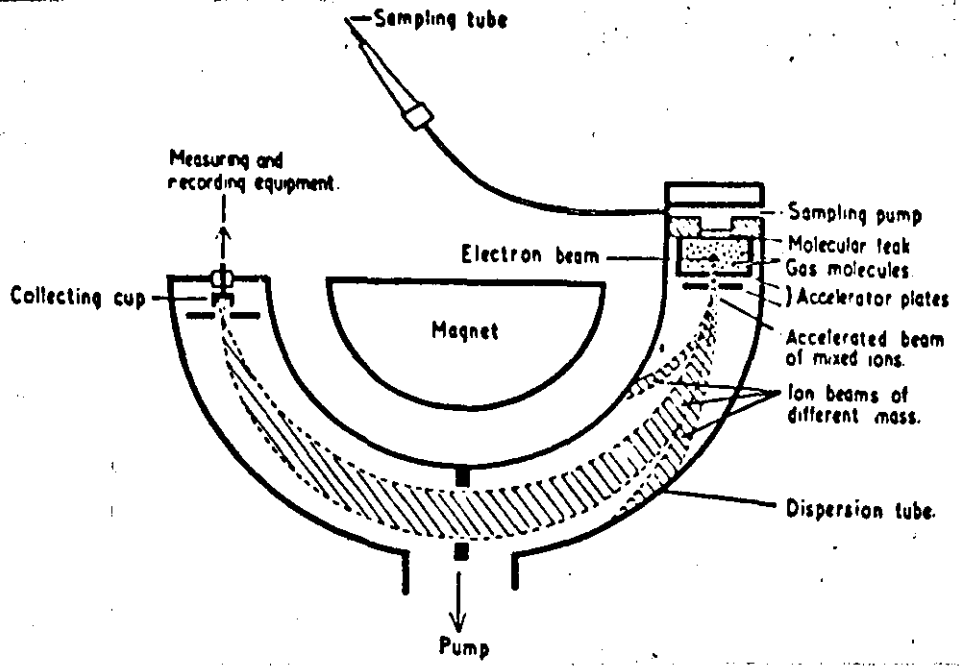


FIGURE B1.

By changing accelerator voltage rapidly and repetitively all trajectories may be focussed on a single fixed collector plate, and the current production indicates the abundance of ions of a given mass charge ratio. A series of peaks proportional to the abundance of ions in each beam is displayed on a cathode ray oscilloscope (Fig. B2.) The position of a peak along the horizontal axis indicates the molecular weight of the gas and the peak height is a measure of its partial pressure.

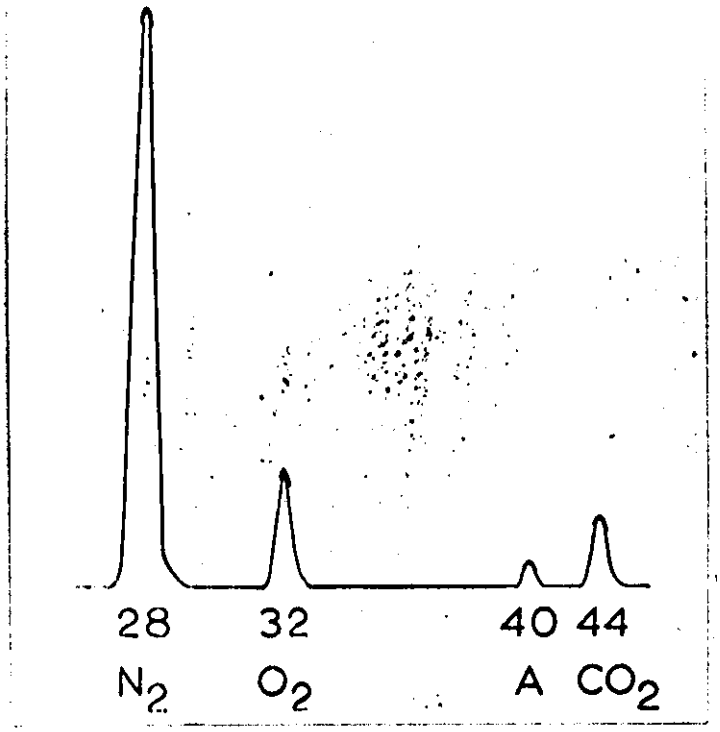


FIGURE B2

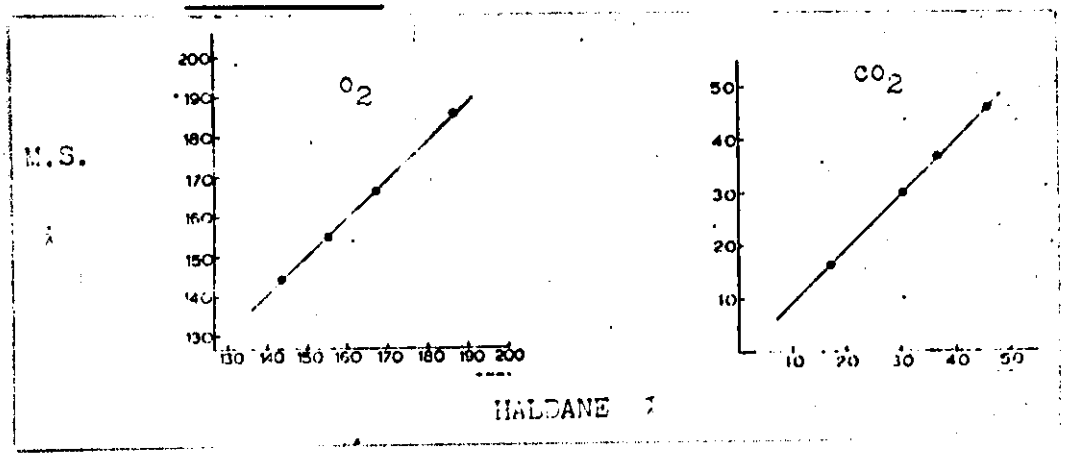


FIGURE B3

The linearity and accuracy of thw RMS used in this study was checked before and after each work test as part of the calibration procedure. Nitrogen is used as a zero calibration for oxygen and carbon dioxide. Air is used as the upper calibrating point for oxygen, a gas blend containing 5% carbon dioxide and 10% oxygen is used to provide the upper calibrating point for carbon dioxide and the lower point for oxygen. Carbon dioxide and onygen concentrations measured by Lloyd-Haldane method plotted against values obtained from the RMS are shown in fig. B3.

APPENDIX C

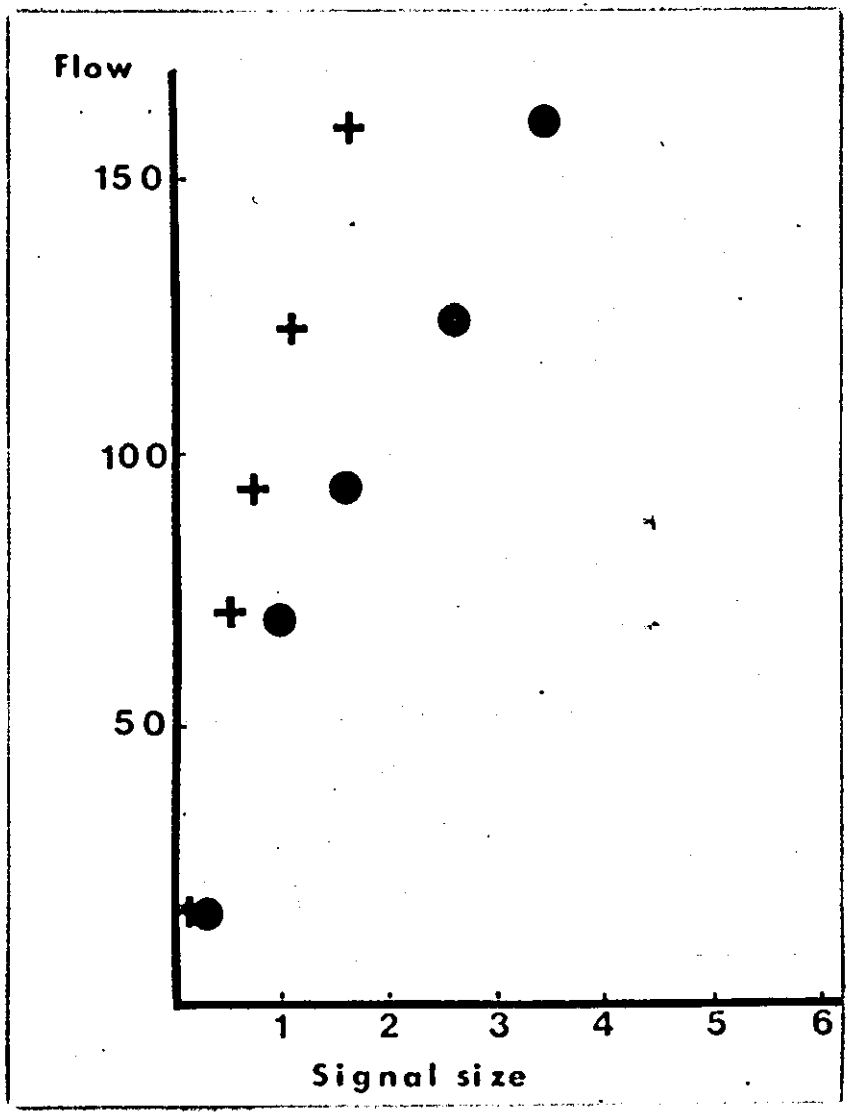
Validation of flow measuring procedure

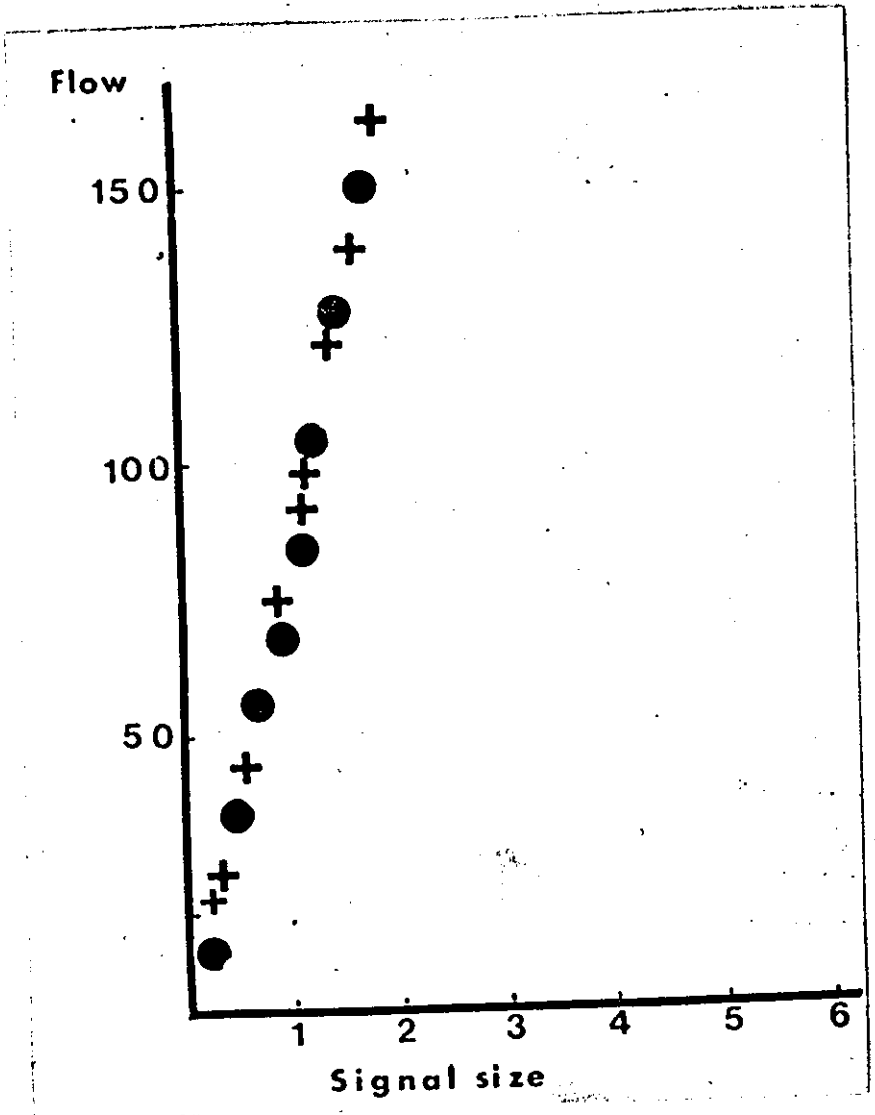
The accuracy of gas flow measurement was tested thus. A variable voltage transformer was connected to a vacuum cleaner to produce different flow-rates. Actual flow rates were determined from repeated readings of gas meter volume (Parkinson Cowan C. D. U.) in unit time. The observed flow rate was passed through a heated Fleisch pneumotachograph (a) without connective tubing, (b) complete with valve box and connective tubing (3 cm. bore 50 cms. length) and (c) as in (b) but with additional tubing on the outflow side of the Fleisch (Finucani et al 1972).

The pressure drop across the resistive element (concentric cylinders) was detected by a Statham PM15 transducer, amplified by a Devices amplifier and recorded on a Cambridge Instruments fibre-optic recorder. Varying flow rates were employed and the entire procedure was repeated on a second occasion. The results are shown in the accompanying figure C1.

The reliability and validity of the set up was tested in two ways. First a series of 1 litre calibration signals were passed through the system at varying speeds and the flow signal integrated; the uniform height of the intervals seen in figure C2 indicates that volume remained constant at varying rates of flow. This was confirmed by the passage of fixed volumes from a ventilator pump at varying rates; figure C3 again shows negligible variation in integral height.

Finally expired air was passed through the pneumotachograph and then on through a gas meter. Observed gas meter volumes were then compared with calculated volumes, the results appearing in figure C4. Variability may be due to additional resistance provided by the gas meter and from temperature change as the gas passed from the Fleisch to the gas meter.





Linearity of Fleisch pneumotachograph

A = Fleisch alone

= Fleisch plus valve box and respiratory tubing.

B. Fleisch plus valve box and respiratory tubing plus tubing on outflow side

= Day 1

= Day 2

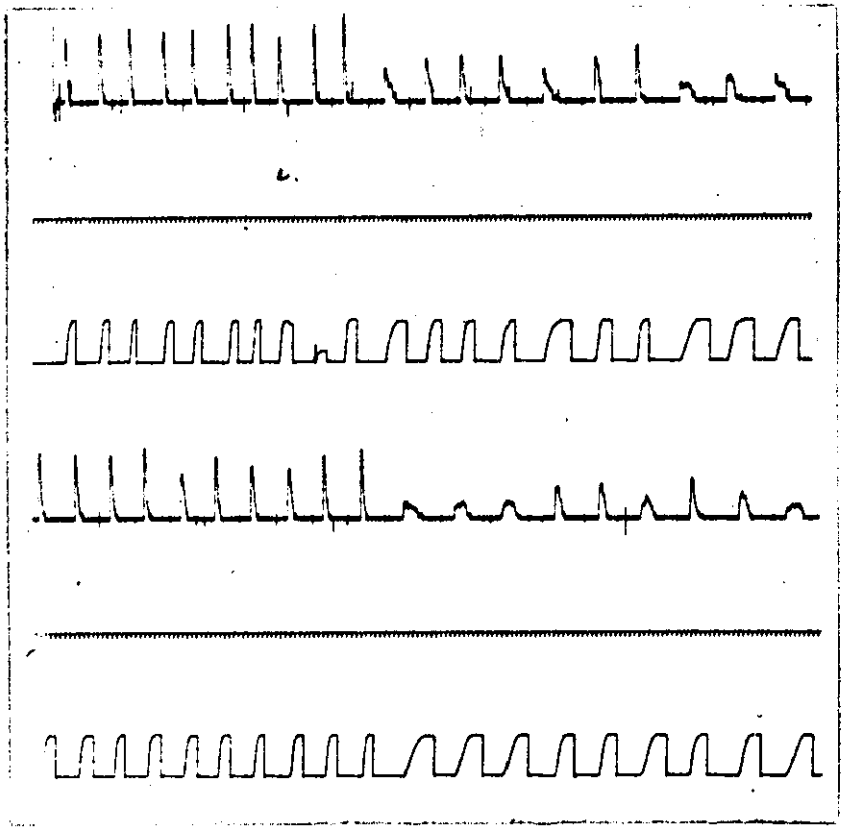


FIGURE C2: One litre signals at varying flow rates

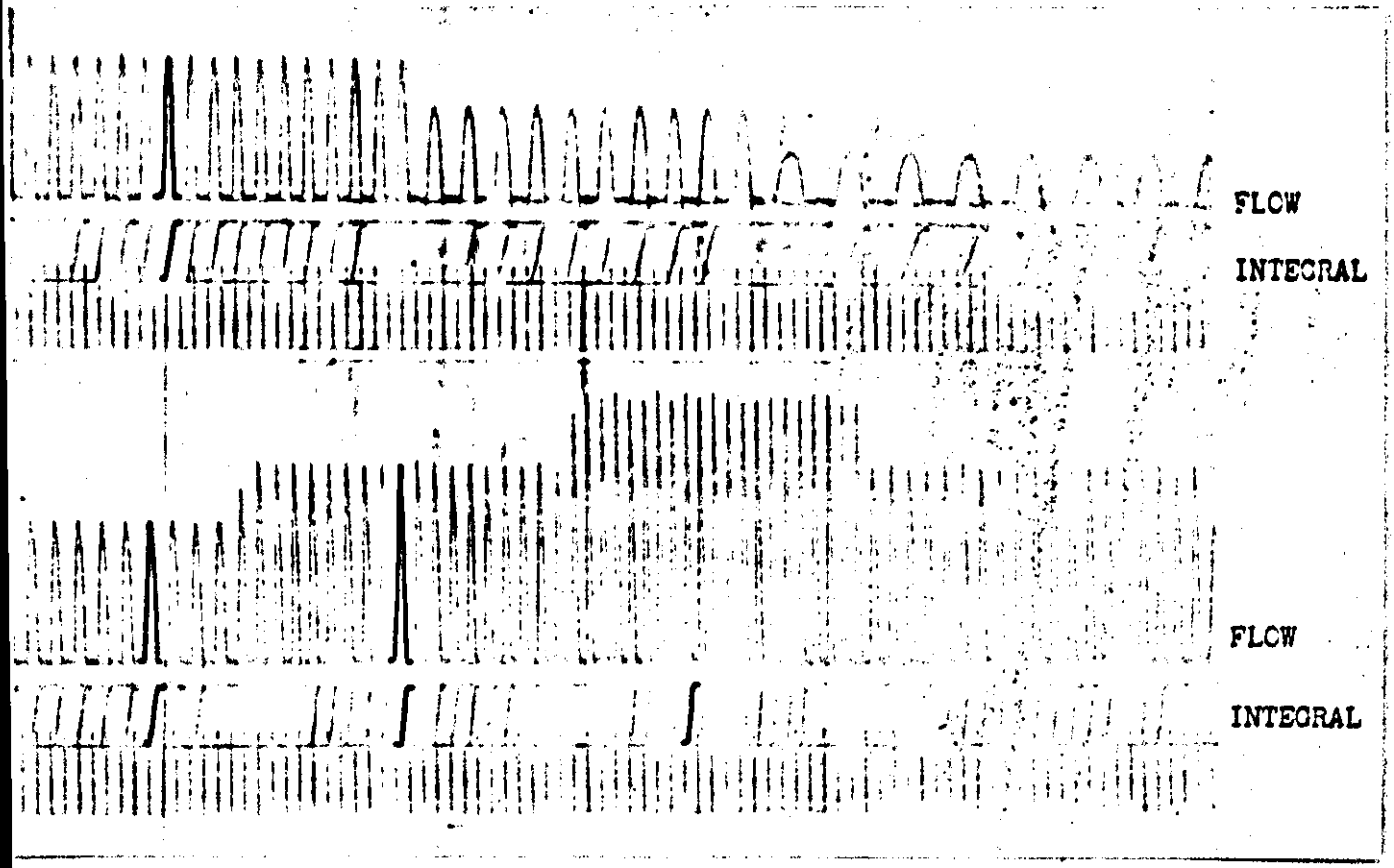


FIGURE C3: Fixed volume, varying flow rates.

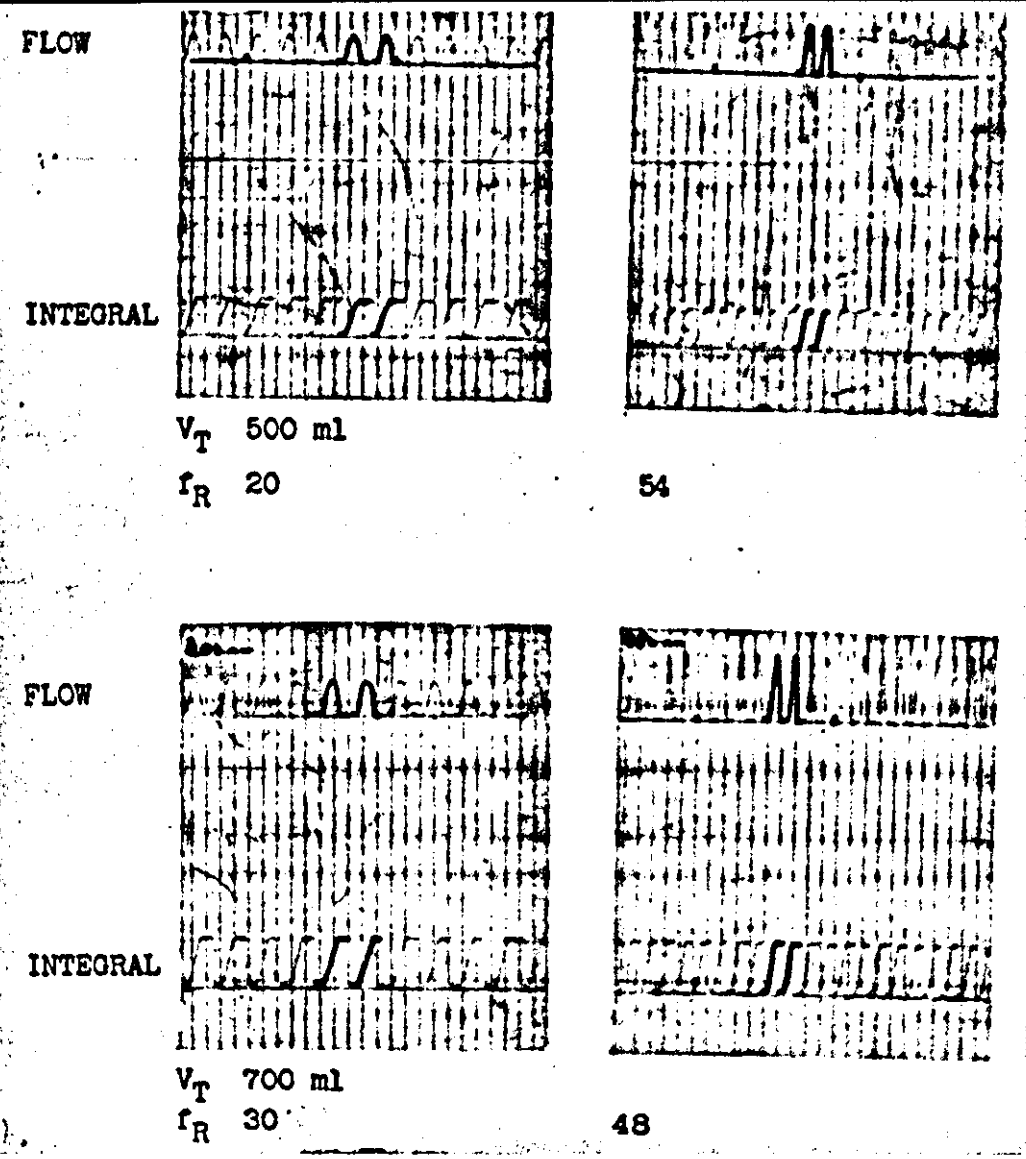


FIGURE C3 continued

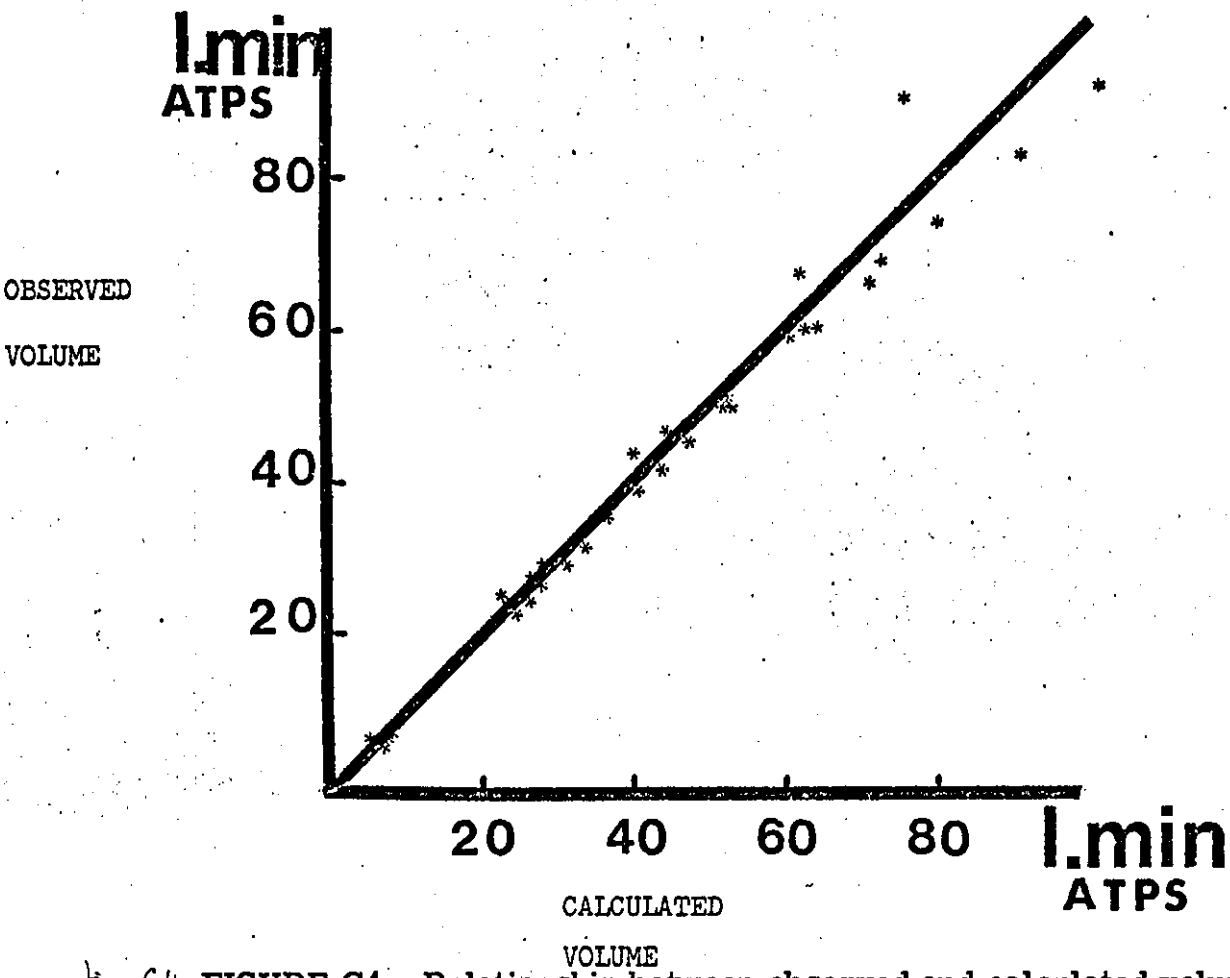


Fig. C4 FIGURE C4: Relationship between observed and calculated volumes at varying flow rates.

APPENDIX D

COMPUTER ANALYSIS PROCEDURE

The analysis of work test data was achieved by computer programme. An example of the raw data collected on tape during a work test is shown in figure D1. The recorded signals were passed through variable frequency filters (KEMO) and converted into digital form by the analogue-digital module of a Varian computer; an example of the digital output from a single breath is shown in Table D1.

The computer programme employed to convert the digital output to meaningful physiological data is enclosed (Table D2) and an example of the print-out is given in Table D3.

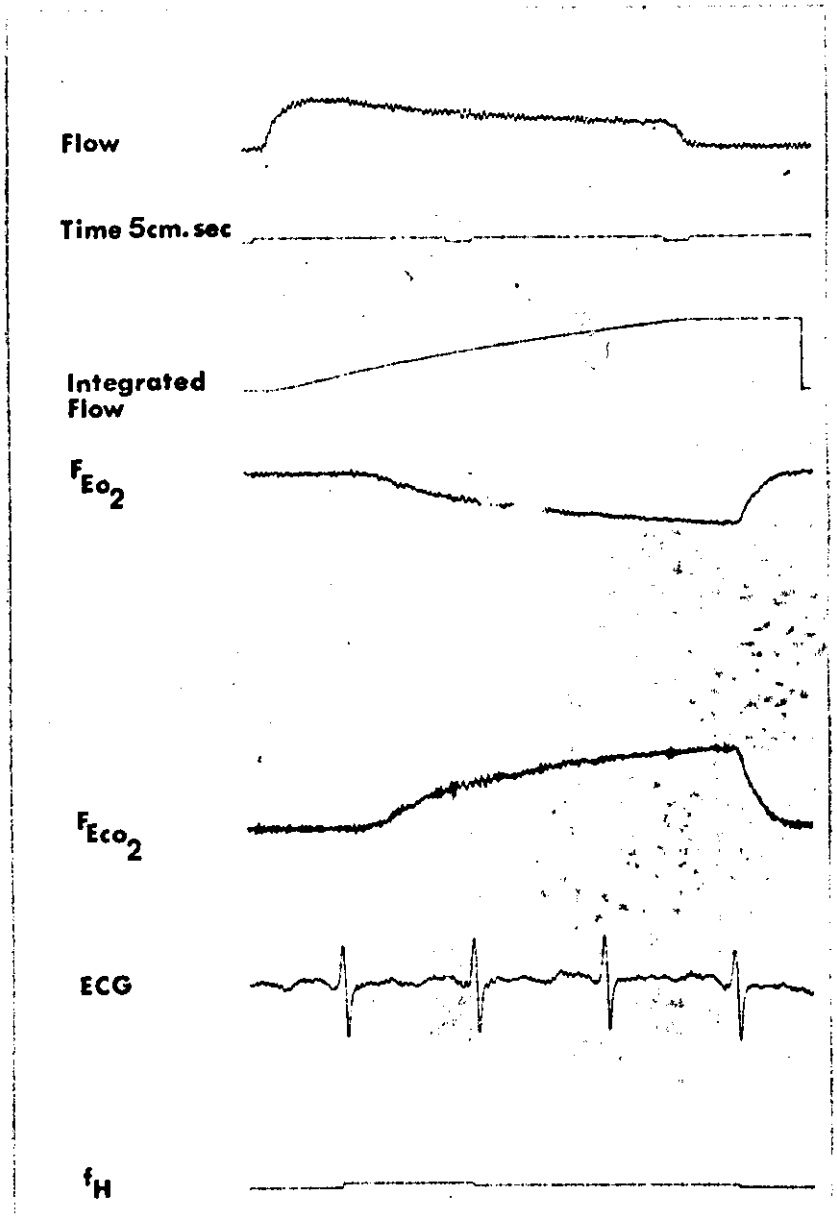


FIGURE D1 - Electrical output from tape recorder

FL	O2	CO2
-196	3984	559
561	3978	544
616	3951	600
445	3393	1397
340	3068	1853
229	2888	2099
-189	2764	2250
-200	3961	593
-203	3976	541
771	4000	553
549	3700	958
429	3243	1619
343	3002	1942
99	2851	2141
-198	3529	1159
-192	3986	555
837	3976	553
631	3877	731
459	3312	1540
358	3030	1903
233	2899	2092
-190	2893	2073
-206	3963	545
693	3985	549
639	3961	596
488	3344	1481
368	3070	1866
270	2897	2092
-192	2791	2206
-197	3979	566
671	3983	546
519	3876	696
396	3211	1655
311	2947	2022
-172	2767	2225
-196	3961	585
-197	3986	548
820	3986	550
586	3542	1205
462	3208	1691
366	2996	1937
270	2863	2139
-192	3169	1703
-199	3982	546
782	3990	553

FL	O2	CO2
-191	3992	557
739	3987	553
564	3828	778
423	3315	1531
316	3023	1926
177	2847	2135
-199	2811	2150
-201	3984	558
-187	3994	555
223	3986	558
524	3553	1180
421	3184	1727
304	2962	2013
-176	2815	2162
-197	3885	669
-190	3990	551
774	4001	552
565	3650	1035
453	3230	1652
327	2982	1965
143	2864	2125
-197	3563	1118
-198	3978	547
804	4009	555
585	3828	786
459	3251	1609
340	3033	1921
184	2865	2106
-202	3419	1314
-201	3968	552
698	3976	555
476	3681	986
379	3132	1784
301	2904	2070
-202	2730	2256
-189	3991	558
-138	3978	547
710	3969	552
566	3426	1377
441	3148	1780
347	2961	1993
42	2839	2173
-189	3719	915
-198	3974	546
740	3988	551

FL	O2	CO2
-193	3998	552
702	3982	546
521	3651	1044
400	3204	1694
277	2959	2001
79	2809	2178
-192	3442	1305
-204	3979	549
410	3992	553
645	3976	573
496	3410	1380
390	3119	1822
265	2921	2053
-197	2798	2186
-202	3957	564
-175	3988	544
698	3985	553
524	3515	1243
427	3160	1747
291	2954	2002
-160	2840	2175
-199	3899	665
-192	3986	551
777	3992	548
531	3618	1079
432	3205	1698
328	2989	1972
-131	2853	2156
-201	3789	803
-191	3992	552
644	4003	563
472	3487	1278
347	3042	1891
264	2881	2110
-200	3364	1406
-196	3976	548
785	3982	544
620	3927	646
530	3310	1533
411	3083	1836
322	2927	2050
-160	2799	2203
-192	3941	610
-107	3924	548
645	3988	574

FL	O2	CO2
-154	3986	553
663	3979	563
488	3538	1216
374	3123	1781
249	2922	2046
-178	2804	2206
-196	3839	740
-196	3979	549
782	3995	553
586	3916	673
464	3307	1528
373	3073	1867
227	2890	2088
-195	2885	2063
-196	3974	554
638	3984	550
679	3961	595
494	3397	1427
403	3110	1836
263	2923	2045
-188	2800	2199
-198	3958	568
-94	3983	548
706	3997	565
502	3458	1337
397	3134	1784
297	2953	2023
-189	2796	2204
-196	3951	611
217	3993	554
580	3953	586
430	3342	1479
327	2987	1974
-39	2798	2179
-200	3816	746
-197	3983	551
880	3989	547
598	3711	954
493	3254	1627
401	3034	1907
292	2880	2084
-187	2774	2229
-198	3978	560
730	3994	548
616	3904	667

Table D1: Digital output of analogue input

TABLE D1. Digital output of analogue input

Table D2 : **TABLE D2**

```
C       A BREATH BY BREATH ANALYSIS OF AN
C      EXERCISE TOLERANCE TEST
C      THIS FIRST SECTION SETS UP THE
C      FILES AND STORAGE SPACE
       COMMON IDATA(960),ISAVE,IREC,ISAVE2,IREC2,
      1IDUM(5),IDELAY,JFCB(13),IBLK(41)
       DIMENSION IFCB(13),FLOW(248),PCO2(240),PO2(240),B(40)
       COMMON /REGRES /SUMT
       DIMENSION SUMT(16,6)
       EQUIVALENCE(SDSC,IBLK(1)),(SDSO,IBLK(3)),(IBLK(5),OBTH)
       DATA IFCB(3)/2H  /
       DATA IFCB(8),IFCB(9),IFCB(10)/2HDA,2HTA,2H  /
       JFCB(3)=IFCB(3)
       JFCB(8)=IFCB(8)
       JFCB(9)=IFCB(9)
       JFCB(10)=IFCB(10)
C      INITIALISE STORES AND CALL FOR CALIBRATION GAS
       REWIND 14
       WRITE(24,500)
500    FORMAT(4X,23HCO2 AND O2 CALIBRATION )
       READ(24,600)CCO2,OO2,PB
600    FORMAT(2F5.2,F5.1)
5      WRITE(24,1000)
1000   FORMAT(4X,25HDELAY TIME NO OF SAMPLES?)
       READ(24,1100)IDELAY
1100   FORMAT(I1)
       IF(IDELAY.LT.1.OR.IDELAY.GT.8)GOTO5
C       THERE ARE NOW TWO LINES AVAILABLE FOR HEADINGS
       READ(17,100)B
       WRITE(14,100)B
100    FORMAT(20A4/20A4)
       WRITE(15,200)B
200    FORMAT(1H ,20X,20A4/20X,20A4/)
C      WE NOW CALL FOR THE ASSEMBLY LANGUAGE
C     PROGRAMME FOR THE A-D CONVERSION
       CALL ADDVS (20000,5)
       CALL V$OPEN(2,53,JFCB,0)
C      SET THE BASE LINE AND SENSITVITY
       IAFL=0
       BFL=1.
C      CALIBRATE FLOW SIGNAL
       CALL LDDAT(FLOW,PCO2,PO2,IAFL,BFL,ID)
C      INITIALSE FLOW AND GAS STORES
       SF=0.
       SO=0.
       SC=0.
C      TAKE TEN SAMPLES
       J=IDELAY+9
       DO 91 I=IDELAY,J
       SF=SF+FLOW(I)
       SO=SO+PO2(I)
91     SC=SC+PCO2(I)
C      THIS GIVES US THE BASELINE OF THE
C      CALIBRATION SIGNALS
C      CALCULATE THE MEAN ZERO AND
C      SET DEAD BAND ABOVE THIS
       AO2=SO/10.
       ACO2=SC/10.
```

```
   59          SFL=AFL+50.0
   60          IAFL=AFL+0.5
   61          SF=0.
   62          DO 92 I=J,240
   63          SF=SF+(FLOW(I)-AFL)
   64          IF(FLOW(I).LT.AFL.AND.I.GT.70) GO TO 93
   65   92     CONTINUE
   66   93     BFL=1000./(SF*0.1)
   67          WRITE(24,85)
   68   85     FORMAT(4X,'FLOW CAL ENDS')
   69   C      NOW CALCULATE 02 CALIBRATION
   70   96     CONTINUE
   71          DO 94 J=I,240
   72          IF(PO2(J).LE.+200..AND.J.LE.225)GO TO 95
   73   94     CONTINUE
   74          CALL LDDAT(FLOW,PCO2,PO2,IAFL,BFL,ID)
   75          I=1
   76          GO TO 96
   77   95     JE=J+14
   78          J=J+5
   79          SO=0.
   80          DO 97 I=J,JE
   81          SO=SO+PO2(I)
   82   97     CONTINUE
   83          BO2=0.209/(AO2-SO/10.)
   84          BPO2=1000.0*BO2
   85          WRITE(24,86)
   86   86     FORMAT(4X,'02 CAL ENDS')
   87   C      CALCULATE CO2 CALIBRATION
   88   991    DO 98 J=I,240
   89          IF(PCO2(J).GT.+1000..AND.J.LE.225)GO TO 99
   90   98     CONTINUE
   91          CALL LDDAT(FLOW,PCO2,PO2,IAFL,BFL,ID)
   92          I=1
   93          GO TO 991
   94   C      ALSO CHECK 02 CALIBRATION
   95   99     SC=0.
   96          SO=0.
   97          JE=J+14
   98          J=J+5
   99          DO 992 I=J,JE
  100          SC=SC+PCO2(I)
  101   992     SO=SO+PO2(I)
  102          BCO2=CCO2/(100.0*(SC/10.-ACO2))
  103          B202=002/(100.0*(AO2-SO/10.))
  104          BPCO2=1000.0*BCO2
  105          BB202=B202*1000.
  106          WRITE(24,87)
  107   87     FORMAT(4X,'CO2 CAL ENDS')
  108          WRITE(15,700)AFL,BFL,AO2,BPO2,ACO2,BPCO2
  109          WRITE(24,700)AFL,BFL,AO2,BPO2,ACO2,BPCO2
  110   700    FORMAT(1X,'VOL CAL,0=',F7.2,'VOL SENS/LITRE',F6.3,/,
  111         1'02CAL 0=',F8.3,'02SENS*1000 FOR 21%02=',F7.4,/,
  112         2'CO2 CAL,0=',F8.3,'CO2 SENS*1000 FOR 7%=',F7.4)
  113   C      CONTINUE? 1=YES,0 OR LESS =NO
  114          WRITE (24,800)
  115   800    FORMAT(11H CONTINUE ?)
  116          READ(24,900)A
```

```
117  900   FORMAT(F10.1)
118        IF(A)2000,2000,995
119  C     NOW PICK UP THE FIRST BREATH FOR ANALYSIS
120  995   JFCB(4)=IREC
121        CALL LDDAT(FLOW,PCO2,PO2,IAFL,BFL,ID)
122        J=(ISAVE/4)+1
123  994   ID=J
124  C      NOW INITIALISE STORAGE AREAS
125        VTM=0.
126        VO2M=0.
127        VCO2M=0.
128        SAFEO2=0.
129        SAFCO2=0.
130        SBRTH=0.
131        SVD=0.
132        SVA=0.
133        SFEO2M=0.
134        SFCO2M=0.
135        TVEO2=0.
136        ATV=0.
137        RQM=0.
138        S3=0.
139        TVD=0.
140        TIME=0.
141        TVA=0.
142        VV=0.
143        WW=0.
144        XX=0.
145        YY=0.
146        RR=0.
147        ISW=1
148        ITIM=0
149        OBTH=0
150        WRITE(15,350)
151  350   FORMAT(1X,'TIM',2X,'BRN',4X,'VT',7X,'MFEO2',5X,'FEO2M',6X,'VO2
152       1,6X,'MFCO2',5X,'FCO2M',6X,'VCO2',4X,'BRQ',3X,'BTM',5X,
153       2'TEXP',3X,'VDB',3X,'VAB',3X,'VEO2B',2X,'PACO2',2X,'PAO2')
154  C     SEARCH FOR START OF EXPIRATION
155  2     IF(ID.GT.240)CALL LDDAT(FLOW,PCO2,PO2,IAFL,BFL,ID)
156        IF(FLOW(ID).GT.SFL.AND.FLOW(ID+1).GT.SFL.AND.
157       1FLOW(ID+2).GT.SFL)GO TO 1
158        ID=ID+1
159        ITIM=ITIM+1
160        GO TO 2
161  C     TEST FOR FIRST CYCLE.IF NOT PRINT RESULTS
162  1     IF(ISW.LT.2) GO TO 11
163        TBRTH =FLOAT(ITIM)*0.1
164        SBRTH=SBRTH+TBRTH
165        TIME=SBRTH
166        FCO2M=FCO2M*100.
167        SFCO2M=SFCO2M+FCO2M
168        FEO2M=FEO2M*100.
169        SFEO2M=SFEO2M+FEO2M
170        WRITE(15,400)TIME,OBTH,VT,AFEO2,FEO2M,VO2,AFCO2,FCO2M,VCO2,BRQ,
171       1TBRTH,TEXP,VD,VA,VEO2B,PACO2,PAO2
172  400   FORMAT(F5.1,F4.0,F8.2,6F10.4,2F7.3,F7.2,F6.1,3F7.1,F6.1)
173  C     CALCULATE GAS FLOW AND VOLUMES
174  11    ISW=ISW+1
```

```
175          ITIM=0
176          IF(ID.LT.3)GO TO 12
177          AO2=(PO2(ID-1)+PO2(ID-2))/2.
178          ACO2=(PCO2(ID-1)+PCO2(ID-2))/2.0
179          GO TO 13
180    12    AO2=PO2(ID)
181          ACO2=PCO2(ID)
182    13    VT=0.
183          VO2=0.
184          VCO2=0.
185          FEO2M=0.
186          FCO2M=0.
187          SFEO2=0.
188          SFCO2=0.
189          BFEO2=0.
190          BFCO2=0.
191          AFEO2=0.
192          AFCO2=0.
193          S1=0.
194          S2=0.
195          VD=0.
196          VA=0.
197          PACO2=0.
198          PAO2=0.
199          VEO2B=0.
200          RAFCO2=0.
201          RAFEO2=0.
202          RVA=0.
203          RVD=0.
204          RVCO2=0.
205          RVO2=0.
206          RVT=0.
207    3     FL=FLOW(ID)*0.1
208          VT=VT+FL
209          FEO2=(AO2-PO2(ID))*BO2
210          BFEO2=(AO2-PO2(ID))*BO2*100.
211          SFEO2=SFEO2+BFEO2
212          S1=S1+1.
213          FCO2=(PCO2(ID)-ACO2)*BCO2
214          BFCO2=(PCO2(ID)-ACO2)*BCO2*100.
215          SFCO2=SFCO2+BFCO2
216          S2=S2+1.
217          IF(FEO2M.GT.FEO2) GO TO 6
218          FEO2M=FEO2
219          PO3=AO2-(FEO2M/BO2)
220    6     IF(FCO2M.GT.FCO2) GO TO 7
221          FCO2M=FCO2
222    7     CONTINUE
223    22    ID=ID+1
224          ITIM=ITIM+1
225          IF(ID.GT.240)CALL LDDAT(FLOW,PCO2,PO2,IAFL,BFL,ID)
226          IF(FLOW(ID).GE.SFL.OR.FLOW(ID+1).GE.SFL.OR.FLOW(ID+2).GE.SFL)
227         1GO TO 3
228          RVT=VT
229          AFEO2=SFEO2/S1
230          CALL SUM (SUMT(5,6),VT,AFEO2)
231          RAFEO2=AFEO2
232          CALL SUM (SUMT(15,6),RVT,RAFEO2)
```

```
PAGE    5  16/09/75  HALE       VORTXII FTN IV                1554 HOURS

 233          SAFE02=SAFE02+AFE02
 234          AFC02=SFC02/S2
 235          CALL SUM (SUMT(6,6),VT,AFC02)
 236          RAFC02=AFC02
 237          CALL SUM (SUMT(16,6),RVT,RAFC02)
 238          SAFC02=SAFC02+AFC02
 239          VD=(((FC02M*100.)-AFC02)/((FC02M*100.)-0.03))*VT
 240          CALL SUM (SUMT(3,6),VT,VD)
 241          RVD=VD
 242          CALL SUM (SUMT(13,6),RVT,RVD)
 243          SVD=SVD+VD
 244          VA=VT-VD
 245          CALL SUM (SUMT(4,6),VT,VA)
 246          RVA=VA
 247          CALL SUM (SUMT(14,6),RVT,RVA)
 248          SVA=SVA+VA
 249          V02=VT*AFE02/100.
 250          CALL SUM (SUMT(1,6),VT,V02)
 251          RV02=V02
 252          CALL SUM (SUMT(11,6),RVT,RV02)
 253          VC02=VT*AFC02/100.
 254          CALL SUM (SUMT(2,6),VT,VC02)
 255          RVC02=VC02
 256          CALL SUM (SUMT(12,6),RVT,RVC02)
 257          VE02B=VT/V02
 258          PAC02=((1.136*PB*VC02)/VA)+0.03
 259          CALL SUM (SUMT(7,6),VT,PAC02)
 260          CALL SUM (SUMT(10,6),AFC02,PAC02)
 261          PA02=((PB-47.*.2093)-(PAC02/BRQ))+(PAC02*.2093*((1.0-BRQ/BRQ)))
 262          CALL SUM (SUMT(8,6),VT,PA02)
 263          CALL SUM (SUMT(9,6),AFE02,PA02)
 264          V02M=V02M+V02
 265          VC02M=VC02M+VC02
 266          VTM=VTM+VT
 267          S3=S3+1.
 268          OBTH=OBTH+1.
 269          TEXP=FLOAT(ITIM)*0.1
 270          BRQ= VC02/V02
 271   C      EACH MINUTE THE VE,V02 AND VC02 ARE COMPUTED
 272          RR=SBRTH-60.
 273          IF(RR.GE.0.)CALL PRINT(VTM,SBRTH,VC02M,V02M,CF,RQM,S3,ATV,TVD,
 274         2TVA,TVE02,RR,SAFE02,SFE02M,SAFC02,SFC02M,PB)
 275          GO TO 2
 276    2000 CALL EXIT
 277          END
0 ERRORS COMPILATION COMPLETE
```

```
PAGE    1  16/09/75  HALE       VORTXII FTN IV              1556 HOURS

    1          SUBROUTINE LDDAT(FLOW,PCO2,PO2,IAFL,BFL,ID)
    2  C       LOADING AND SCALING OF RAW DATA
    3          COMMON IDATA(960),ISAVE,IREC,ISAVE2,IREC2,IDUM(5),
    4         1IDELAY,JFCB(13),IBLK(41)
    5          COMMON /REGRES /SUMT
    6          DIMENSION FLOW (248),PCO2(240),PO2(240)
    7          DIMENSION SUMT(16,6),X(6),Y(6),P(6),Q(6),R(6),S(6),T(6),U(6),
    8         1V(6),W(6)
    9          EQUIVALENCE(IBLK(1),SDSC),(IBLK(3),SDSO),(IBLK(5),OBTH)
   10          DO1 I=1,960,120
   11          IE=I+119
   12          READ(2)(IDATA(K),K=I,IE)
   13          IF (IOCHK(IST))5,1,4
   14     1    CONTINUE
   15          DO 2 I=1,IDELAY
   16     2    FLOW(I)=FLOW(I+240)
   17          IFL =IDELAY
   18          IPC=1
   19          DO3  I=1,960,4
   20          FLOW (IFL)=FLOAT(IDATA(I)-IAFL)*BFL
   21          PO2(IPC)=IDATA(I+1)
   22          PCO2(IPC)=IDATA(I+2)
   23          IFL =IFL+1
   24  3       IPC = IPC+1
   25          ID=1
   26          RETURN
   27     5    WRITE(15,200)
   28     200  FORMAT(1H ,14HI/O DISC ERROR)
   29     4    AA=-9999.
   30          BB=0.
   31          CALL REGRES (SUM(1),X)
   32          WRITE(15,9000)(X(L),L=1,10)
   33  9000    FORMAT(1H,//'REGRESSION FORMULAE',10F12.3)
   34          CALL REGRES (SUM(2),Y)
   35          WRITE(15,9000)(Y(L),L=1,10)
   36          CALL REGRES (SUM(3),P)
   37          WRITE(15,9000)(P(L),L=1,10)
   38          CALL REGRES (SUM(4),Q)
   39          WRITE(15,9000)(Q(L),L=1,10)
   40          CALL REGRES (SUM(5),R)
   41          WRITE(15,9000)(R(L),L=1,10)
   42          CALL REGRES (SUM(6),S)
   43          WRITE(15,9000)(S(L),L=1,10)
   44          CALL REGRES (SUM(7),T)
   45          WRITE(15,9000)(T(L),L=1,10)
   46          CALL REGRES (SUM(8),U)
   47          WRITE(15,9000)(U(L),L=1,10)
   48          CALL REGRES (SUM(9),V)
   49          WRITE(15,9000)(V(L),L=1,10)
   50          CALL REGRES (SUM(10),W)
   51          WRITE(15,9000)(W(L),L=1,10)
   52          WRITE(14,100)AA,(BB,I=1,6)
   53          WRITE(15,100)AA,(BB,I=1,6)
   54          SDSC=SDSC/FLOAT(OBTH)
   55          SDSO=SDSO/FLOAT(OBTH)
   56          WRITE(15,300)SDSC,SDSO
   57    300   FORMAT(1H0,14HMEAN CO2 D.S.=,F7.2,13HMEAN O2 D.S.=,F7.2)
   58   100    FORMAT(2F6.0,5F6.2)
```

```
   59          CALLV$CLOS(2,0)
   60          ENDFILE 14
   61          CALL EXIT
   62          END
  0 ERRORS COMPILATION COMPLETE
```

```
GE    1  19/09/75  HALE       VORTXII FTN IV                1459 HOURS

 1          SUBROUTINE PRINT(VTM,SBRTH,VCO2M,VO2M,CF,RQM,S3,ATV,TVD,TVA,TVEO2,
 2         1RR,SAFEO2,SFEO2M,SAFCO2,SFCO2M,PB,SUM11,SUM12,SUM13,SUM14,SUM15,
 3         2SUM16)
 4  C       CALCULATE MINUTE VALUES.
 5          DIMENSIONSUM11(6),SUM12(6),SUM13(6),SUM14(6),SUM15(6),SUM16(6),
 6         1AR(6),BR(6),CR(6),D(6),E(6),F(6)
 7          WRITE(24,8008)
 8  8008    FORMAT(4X,'ONE')
 9          CF=(273./310.)*((PB-47.)/760.)
10          VTM=((VTM/SBRTH)*60.0)/1000.
11          VO2M=((VO2M/SBRTH)*60.*CF)/1000.
12          VCO2M=(VCO2M/SBRTH*(60.*CF))/1000.
13          RQM= VCO2M/VO2M
14          WRITE(24,6666)
15  6666    FORMAT(1H,'TWO')
16          VV=SAFEO2/S3
17          WW=SFEO2M/S3
18          XX=SAFCO2/S3
19          YY=SFCO2M/S3
20          TVD=((YY-XX)/(YY-0.03))*VTM
21          TVA=VTM-TVD
22          TVEO2=VTM/VO2M
23          ATV=VTM/S3*1000.
24          WRITE(24,6667)
25  6667    FORMAT(1H,'THREE')
26          PACO2M=((1.136*PB*VCO2M)/TVA)+0.3
27          PAO2M=(((PB-47.)*.2093)-(PACO2M/RQM))+(PACO2M*.2093*((1.0-RQM)/RQ
28         1M))
29          WRITE(15,77)
30  77      FORMAT(1H,//,'MINUTE VALUES',/,2X,'VE   FR   MVT    VO2  O2F  MO2
31         1  VCO2  CO2F MCOF  MRQ   TVD  MVA   MVEQ   MPACO2    MPAO2')
32          WRITE(15,78)VTM,S3,ATV,VO2M,VV,WW,VCO2M,XX,YY,RQM,TVD,TVA,TVEO2,
33         1PACO2M,PAO2M
34  78      FORMAT(2F5.1,1X,F6.1,F6.3,2F5.2,F6.3,F5.2,F6.2,F6.2,3F6.1,2F10.1)
35          WRITE(15,3500)
36  3500    FORMAT(1H,1X,'TIM',2X,'BRN',4X,'VT',7X,'MFEO2',5X,'FEO2M',6X,'VO2
37         1,6X,'MFCO2',5X,'FCO2M',6X,'VCO2',4X,'BRQ',3X,'BTM',5X,
38         2'TEXP',3X,'VDB',3X,'VAB',3X,'VEO2B',2X,'PACO2',2X,'PAO2')
39          VTM=0.
40          VCO2M=0.
41          VO2M=0.
42          RQM = 0.
43          SAFEO2=0.
44          SAFCO2=0.
45          SBRTH=0.
46          S3=0.
47          SFCO2M=0.
48          SFEO2M=0.
49          TVD=0.
50          TVA=0.
51          TVEO2=0.
52          ATV=0.
53          RR=0.
54          VV=0.
55          WW=0.
56          XX=0.
57          YY=0.
58          RETURN
```

```
PAGE    1  16/09/75  HALE       VORTXII FTN IV                1556 HOURS

    1          SUBROUTINE REGRES (A,C)
    2  C RETURNS LINEAR REGRESSION FORMULAE
    3          DIMENSION A(6),B(5),C(10)
    4          B(2)=A(5)-(A(2)**2/A(1))
    5          B(1)=A(3)-(A(4)**2/A(1))
    6          B(3)=A(6)-(A(2)*A(4)/A(1))
    7          IF(A(2).NE.0.0) GO TO 1
    8          A(2)=0.000001
    9        1 C(1)=A(2)/A(1)
   10          IF(A(4).NE.0.0)GO TO 2
   11          A(4)=0.000001
   12        2 C(2)=A(4)/A(1)
   13          IF(B(3).NE.0.0)GO TO 3
   14          B(3)=0.000001
   15        3 IF(B(1).NE.0.0)GO TO 4
   16          B(1)=0.000001
   17        4 C(3)=B(3)/B(1)
   18          C(4)=C(2)-C(3)*C(1)
   19          C(5)=B(3)/SQRT(B(1)*B(2))
   20          C(6)=SQRT(B(2))*SQRT(1.0-C(5))
   21          C(7) =SQRT(B(1)/A(1)-1.0)
   22          C(8) =SQRT(B(2)/A(1)-1.0)
   23          C(9)=C(7)/SQRT(A(1))
   24          C(10)=C(8)/SQRT(A(1))
   25          RETURN
   26          END
  0 ERRORS COMPILATION COMPLETE
 /FMAIN
```

```
PAGE    1  16/09/75  HALE       VORTXII FTN IV                    1556 HOURS

    1          SUBROUTINE SUM(A,Z,Y)
    2          DIMENSION A(6)
    3          X=Z/1000.0
    4          A(1)=A(1)+1.0
    5          A(2)=A(2)+X
    6          IF(X.EQ.0.0) GO TO 1
    7          A(3)=A(3)+X**2
    8        1 A(4)=A(4)+Y
    9          IF(Y.EQ.0.0) GO TO 2
   10          A(5)=A(5)+Y**2
   11          IF(X.EQ.0.0) GO TO 2
   12          A(6)=A(6)+X*Y
   13        2 RETURN
   14          END
  0 ERRORS COMPILATION COMPLETE
```

VOL CAL,0= -87.90VOL SENS/LITRE .600
2CAL 0=3316.40202SENS*100) FOR 21%02= .0442
02 CAL,0= 506.700CO2 SENS*1000 FOR 7%= .0302

TIM	BRN	VT	MFE02	FE02M	VO2	MFC02	FC02M	VC02	BRG	BTM	TEXP	VDB	VAB	VE02B	PAC02	PA02
2.0	1.	358.63	1.73	2.65	6.38	1.75	2.57	6.28	.984	2.000	1.40	115.3	242.8	56.2	22.0	0.0
4.3	2.	390.31	3 33	5.76	12.94	3.33	5.83	13.00	1.004	2.300	1.60	168.3	222.0	30.2	49.8	96.3
7.1	3.	579.32	3 27	5.55	18.94	3.39	5.58	19.64	1.037	2.300	2.00	228.7	350.6	30.6	47.6	99.3
10.2	4.	695.12	2.97	5.18	20.62	3.14	5.31	21.84	1.060	3.100	2.30	285.0	410.1	33.7	45.3	102.7
12.7	5.	696.84	2 73	4.85	18.28	2.94	4.98	19.31	1.056	2.500	1.90	271.0	385.8	35.9	42.6	106.1
15.7	6.	787.28	3 27	5.35	23.82	3.42	5.40	24.85	1.043	3.000	2.20	268.8	458.5	30.5	46.1	102.6
18.6	7.	671.90	3 03	5.04	20.33	3.16	5.17	21.22	1.044	2.900	1.90	263.1	408.8	33.1	44.2	104.0
21.4	8.	810.81	3.46	5.14	28.03	3.66	5.29	29.66	1.058	2.800	2.00	251.5	559.3	28.9	45.1	103.1
24.0	9.	576.14	2 93	5.18	17.17	3.10	5.28	17.84	1.039	2.600	1.30	239.9	336.2	33.6	45.1	103.6
27.3	10.	686.42	3 21	5.33	22.04	3.37	5.48	23.15	1.050	3.300	2.40	265.3	421.1	31.1	46.7	101.4
30.9	11.	848.01	3.58	5.68	29.86	3.72	5.80	31.59	1.058	3.800	2.70	305.2	542.8	28.4	49.5	99.1
33.7	12.	749.24	2 97	5.09	22.24	3.14	5.23	23.55	1.059	2.300	2.10	300.9	448.4	33.7	44.7	104.0
36.8	13.	834.33	3 63	5.79	30.31	3.75	5.83	31.27	1.032	3.100	2.40	299.6	534.7	27.5	49.7	99.2
39.1	14.	583.64	2 49	4.71	13.02	2.66	4.91	13.93	1.070	2.300	1.50	241.2	282.4	40.2	42.0	105.8
43.0	15.	720.50	3.54	5.40	25.50	3.72	5.59	26.78	1.050	3.900	2.40	242.9	477.6	28.3	47.7	101.5
46.2	16.	908.97	3 68	5.77	33.42	3.89	5.84	35.39	1.059	3.200	2.60	304.7	604.2	27.2	49.8	98.8
49.1	17.	726.74	3.73	5.76	27.51	3.96	5.81	28.79	1.047	2.900	2.20	232.7	494.0	26.4	49.6	99.4
52.0	18.	703.82	3.13	5.59	22.41	3.32	5.65	23.35	1.042	2.900	2.10	292.2	411.6	31.4	48.3	100.2
54.8	19.	781.34	3.13	5.85	22.55	3.30	5.37	23.83	1.057	2.300	2.10	279.2	442.1	32.0	45.8	102.4
57.4	20.	699.98	3.09	5.83	21.61	3.25	5.37	22.73	1.052	2.600	1.30	278.0	422.0	32.4	45.8	102.9
60.8	21.	829.71	3.69	5.74	30.60	3.92	5.83	32.54	1.063	3.400	2.30	272.7	557.0	27.1	49.7	99.0

MINUTE VALLES

VE	FR	MVT	VO2	C2F	M02F	VC02	CO2F	MCCF	MRQ	TVD	MVA	MVEQ	MPAC02	MPA02
14.9	22.0	678.9	.393	3.18	5 00	.413	3.33	5.10	1.05	5.2	9.7	38.0	36.3	111.7

TIM	BRN	VT	MFE02	FE02M	VO2	MFC02	FC02M	VC02	ERQ	BTM	TEXP	VDB	VAB	VE02B	PAC02	PAO
2.7	22.	715.58	3 20	5.07	22.87	3.37	5.26	24.11	1.054	2.700	1.30	258.7	456.9	31.3	44.9	104.0
5.3	23.	744.68	3.17	5.27	23.61	3.35	5.43	24.93	1.056	2.600	1.90	286.8	457.8	31.5	46.3	102.3
7.8	24.	642.62	3.30	4.94	21.21	3.50	5.18	22.52	1.062	2.500	1.50	209.2	433.5	30.3	44.2	104.4
10.4	25.	740.18	3.13	5.36	23.54	3.39	5.61	25.12	1.067	2.600	1.90	294.3	445.9	31.4	47.9	101.0
13.4	26.	743.36	3.97	5.67	29.49	4.13	5.84	30.71	1.041	3.000	2.00	218.7	524.7	25.2	49.8	99.4
16.1	27.	689.42	3.23	5.20	22.23	3.44	5.46	23.72	1.067	2.700	1.90	256.0	433.5	31.0	46.5	101.7
18.5	28.	581.36	3.09	5.17	17.97	3.25	5.39	18.88	1.051	2.400	1.50	232.4	348.9	32.4	46.0	103.0
21.1	29.	748.34	3 33	5.29	25.31	3.56	5.45	26.64	1.052	2.600	1.30	261.0	487.3	29.6	46.5	102.0
23.4	30.	619.34	3.16	5.11	19.57	3.33	5.31	20.65	1.056	2.300	1.50	231.7	387.6	31.7	45.3	103.2
25.7	31.	706.52	3.37	5.57	23.81	3.56	5.81	25.13	1.055	2.300	1.70	275.0	431.5	29.7	49.5	99.3
27.9	32.	530.66	2.73	4.73	14.45	2.95	5.05	15.67	1.084	2.200	1.30	221.9	308.8	36.7	43.1	105.4
30.1	33.	742.76	3.35	5.48	24.85	3.52	5.72	26.14	1.052	2.200	1.70	286.9	455.8	29.9	48.8	101.0
32.2	34.	695.60	3.36	5.74	23.37	3.56	5.98	24.77	1.060	2.100	1.60	282.5	413.1	29.8	51.0	97.7

REGRESSION FORMULAE

	MEANX	MEANY		SLOPE	INTERCEPT	R	SEE	SDX	SDY	SEMX	SEMY
Vt	.674	21.905	Vo2	43.320	-7.287	.957	1.265	.003	1.045	.004	.177
	.674	23.055	Vco2	45.972	-7.924	.961	1.281	.003	1.109	.004	.187
	.674	251.719	Vd	362.867	40.836	.872	17.368	.003	8.317	.004	1.406
	.674	422.154	Va	687.148	-40.896	.969	16.933	.003	16.437	.004	2.778
	.674	3.194	Feo2	2.146	1.748	.717	.215	.003	.069	.004	.012
	.674	3.360	Feco2	2.337	1.735	.738	.219	.003	.073	.004	.012
	.674	45.774	Petco2	35.051	28.893	.660	2.987	.003	.879	.004	.149
	.674	99.110	Peto2	41.268	71.301	.319	14.453	.003	3.003	.004	.508
	.003	99.110		21484.578	30.485	.496	12.429	.000	3.003	.000	.508
	.003	45.774		10512.562	10.454	.878	1.790	.000	.879	.000	.149

9999 0. 0.00 0 00 0.00 0.00 0.00

TABLE D3 **Computer print out of part of a work-test.**

APPENDIX E

PHYSICAL TRAINING IN COLD: A CASE STUDY

A 49 year old serviceman was referred to the R. A. F. Chest Unit, Midhurst complaining of exertional dyspnoea and inability to cope with service duties. Clinical investigation resulted in a diagnosis of emphysema. Work tolerance tests on cycle and treadmill were undertaken and the results are shown below:-

	TREADMILL	CYCLE
TIME (mins)	4.0	7.0
f_{Hr} (b. min.)	97	100
f_{H} max (b. min.)	140	139
V_{02} (l. min)	0.630	0.723
V_{02} $f_{H}130$ (l. min)	0.457	0.616
$f_{HV02}1.5$ (b. min.)	159.8	153.4

TABLE E1. Responses to work tests shortly after hospitalisation.

Subsequently the man agreed to undergo supervised progressive exercise training in an effort to improve his work tolerance prior to medical discharge. A treadmill familiarisation programme was completed; this consisted of five days of twice daily sessions of walking for short periods, usually less than 3 minutes duration, at varying speeds. The work tests were then repeated, results appearing below.

	TREADMILL	Δ %	CYCLE	Δ %
TIME (min)	9.0	+125	7.0	0
f_{Hr} (b. min.)	100	+3	92	-8
$f_{H\ max}$ (b. min)	132	-5.7	140	+1
V_{02} (l. min.)	0.854	+35.6	0.991	+37.1
$V_{02}f_H^{130}$ (l. min)	0.828	+81.2	0.866	+40
$f_H V_{02}^{1.5\,l}$ (b. min).	138.6	-13.3	137.2	-10.6

TABLE E2. Responses to work tests after 1 week familiarisation programme.

The training consisted of intermittent work on the treadmill designed to raise f_{Ht} above 125 b. min. There were 31 training sessions; 23 on the treadmill, 5 sessions in the hospital grounds and 3 at home during a long week-end leave; the mean time of training sessions was 28.2 minutes.

The work tests were then repeated giving the following results:-

	TREADMILL	Δ %	CYCLE	Δ %
TIME (min.)	11.0	+22.2	7.0	0
f_{Hr} (b. min.)	96	-4	85	-7.6
$f_{H\ max}$ (b. min)	134	+1.5	140	0
V_{02} (l. min)	1.053	+23.3	1.150	+16.0
$V_{02\ fH}^{130}$ (l. min.)	0.993	+19.9	0.923	+6.6
$f_{H\ V02}^{1.5\,l}$ (b. min.)	128.9	-7.0	133.9	-2.4

TABLE E3. Changes in responce to work tests following 6 weeks training.

low working $f_{H\ max}$ demonstrated by many elderly patients is likely to blur issues rather than clarify them, and ventilation will be used as the variable against which changes in other variables will be measured.

Pearson product-moment correlation coefficients and regression equations determined by least squares method were obtained for V_{02}/f_H relationships during work. Results are seen in Figure E1.

It is clear from the lines of least fit that even the pre-training familiarisation programme improved V_{02}/f_H relationships. The very low level of work involved in the pre-training programme is unlikely to produce fundamental changes in any link of the oxygen chain, and thus the improvements in V_{02} at standard f_H and in f_H at standard V_{02} are more likely to be an indication of reduced anxiety. The large changes in intercepts with only minor changes in slope suggest an habituation process; since changes occurred during both cycle and treadmill and familiarisation occurred on the treadmill only, habituation appears to be general to the whole test procedure.

The post-training work tests reveal two things. Firstly a small general improvement in oxygen uptake, shown by the change in the bicycle intercept and, secondly, specific adaptation to treadmill walking indicated by changes in both slope and intercept. Improved technique of treadmill walking resulted in a lowered oxygen cost at a given load and was accompanied by lower f_H slower f_R and reduced V_E.

Clearly if training programmes are to be evaluated meaningfully the twin problems of habituation and learning of a particular motor-skill have to be resolved. Experiments which fail to include familiarisation procedures or use a single device to test and train subjects (in some studies both weaknesses are apparent) are likely to give an exaggerated view of the benefits of exercise therapy. Furthermore in patients who are anxious the V_{02}/f_H relationship may not be a reliable guide to changes in post-training cardio-respiratory function. Anxiety combined with the

APPENDIX F

Individual responses to resting lung function tests.

INDIVIDUAL RESTING LUNG FUNCTION OF GROUP E (n = 3)

Nitrogen wash-out

PRE-TRAINING	TLC L	VT ml	VT_{CO2} ml	BOHR D.S.	VE L.min.BTPS	VCO_2 ml. min.	VD ml	VENT.EFFIC.
E1	4.501	796	23	261	9.445	274	494	38
E2	5.037	525	17	124	7.370	237	351	33
E7	3.600	467	10	117	9.867	217	328	30
$\bar{X}$	4.379	596	16.7	167	8.894	242.7	391	33.7
S	0.726	175.6	6.51	81.2	1.337	28.9	89.9	4.04
POST-TRAINING								
E1	5.404	853	31		9.370	345	406	52
E2	7.929	392	16	119	8.219	326	244	38
E7	6.900	553	18	132	10.673	346	329	40
$\bar{X}$	6.744	599	21.7		9.420	339	326.3	43.3
S	1.270	234.0	8.14		1.227	11.3	81.0	7.57

INDIVIDUAL RESTING LUNG FUNCTION OF GROUP E (n = 4)

Body Plethysmography

	VC	RV	TLC	RV/TLC %	FRC	$FEV_{1.0}$	FVC	$FEV_{1.0}/FVC$ %	$FEV_{1.0}/VC$ %	RAW
Pre-training										
E3	3.93	4.97	8.90	56	6.46	1.15	3.15	37	29	2.80
E4	2.67	5.47	8.14	67	7.00	0.69	3.0	23	26	3.94
E5	3.00	6.03	9.03	67	7.00	0.54	2.73	20	18	3.68
E6	3.41	3.24	6.65	49	4.78	1.15	3.52	33	34	1.74
Post-training										
E3	3.40 -	5.50 +	8.90 o	62 +	6.52 +	1.54 +	2.64 -	58.5 +	45 +	3.17
E4	2.40 -	7.42 +	9.80 +	76 +	8.05 +	0.75 +	2.29 -	33.0 +	31 +	2.72
E5	2.20 -	6.97 +	9.17 +	76 +	8.05 +	0.75 +	2.42 -	31.0 +	34 +	2.70
E6	4.06 +	4.15 +	8.21 +	51 +	5.67 +	1.42 +	3.80 +	37.4 +	35 +	3.72

APPENDIX G

Individual responses to Eysenck's Personality Inventory

INDIVIDUAL RESPONSES TO EYSENCK'S PERSONALITY INVENTORY (Means S.D.)

[illegible] TRAINING

GROUP E	N	E	L
E1	3	5	5
E2	13	5	3
E3	7	13	1
E4	11	6	0
E5	2	4	6
E6	7	10	7
E7	10	7	2
	7.6	7.1	3.4
S =	4.08	3.24	2.64

GROUP C	N	E	L
C1	15	9	5
C2	18	14	1
C3	9	7	6
C4	7	12	5
C5	4	8	2
	10.6	10.0	3.8
S =	5.77	2.92	2.18

[illegible] TRAINING

E1	4+	4-	1-
E2	12-	16+	3o
E3	12+	18+	0-
E4	13+	10+	0o
E5	7+	5+	3-
E6	2-	15+	3-
E7	11+	16+	1-
	8.7	12.0	1.6
S =	4.34	5.69	1.4

C1	20+	8-	3-
C2	17-	13-	1o
C3	11+	9+	3-
C4	4-	10-	4-
C5	1-	3-	6+
	10.6	8.6	3.4
S =	8.14	3.65	1.82

L + 14.5 + 69.0 - 53

28.3 - 14.0 - 10.5

RESPONSES OF EXPERIMENTAL GROUP TO ~~TREADMILL~~ ERGOMETER

WORK TESTS BEFORE AND AFTER TRAINING

Individual responses, group means & S.D, percentage changes and level of significance (n = 7 unless stated otherwise).

AGGREGATE VALUES

	TIME (Secs.)	WORK (W.)	fH (b.)	VE (L.BTPS.)	VA (L.BTPS.)	fR (b.)	VO_2 (L.STPD)	VCO_2 (L.STPD)
E1	990	900.0	1576.1	364.2	240.6	290	14.516	12.060
E2	354	169.75	589.9	61.9	38.7	111	2.546	2.191
E3	873	711.2	1724.1	324.4	233.0	309	14.036	13.058
E4	398	206.6	636.7	70.4	45.7	127	2.480	2.157
E5	412	218.0	679.6	87.9	51.3	164	2.942	1.803
E6	439	225.0	764.2	184.6	128.7	202	6.410	6.240
E7	470	267	751.6	106.7	71.2	175	5.354*	3.957
	562.3	385.4	960.3	171.4	115.6	196.9	7.155	5.924
S_1 =	257.0	293.6	479.9	125.2	88.0	76.5	5.708	4.789
E1	1200 +	1250.0 +	2032.7 +	527.7 +	376.5 +	368 +	20.270 +	18.999 +
E2	384 +	195.4 +	685.3 +	92.7 +	54.7 +	158 +	3.070 +	2.781 +
E3	997 +	880.8 +	1735.8 +	325.1 +	231.3 −	350 +	12.407 −	12.203 −
E4	390 −	199.8 −	612.5 −	90.2 +	56.3 +	114 −	3.167 +	2.689 +
E5	486 +	229.7 +	746.4 +	93.47 +	56.5 +	184 +	3.558 +	2.981 +
E6	714 +	525.0 +	1243.3 +	310.08 +	221.1 +	320 +	10.202 +	11.730 +
E7	526	308.6 +	891.1 +	82.56 −	59.7 −	209 +		3.432 −
	671.1	512.8	1135.3	217.40	150.9	243.3	8.779	7.831
n =							6	
S =	317.4	408.2	557.7	174.0	127.6	101.2	6.908	6.505
%Δ	+ 19.3	+ 33.0	+ 18.2	+ 26.8	+ 30.6	+ 23.6	+ 22.7	+ 32.2
p <	0.05	0.05	0.05	NS	NS	0.05	NS	NS

	fH (b.min.)	$\dot{V}E$ (l.min.BTPS)	fR (b. min.)	$\dot{V}O_2$ (l.min.STPD)	$\dot{V}CO_2$ (l.min.STPD)	VEO_2 (ml.100 ml)	$VECO_2$ ml.100 ml)
E1	95.5	22.1	17.6	0.880	0.731	2.51	3.02
E2	100.0	10.5	18.8	0.431	0.371	2.44	2.83
E3	118.5	22.3	21.2	0.765	0.897	2.31	2.49
E4	96.0	10.6	19.1	0.374	0.325	2.83	3.26
E5	99.0	12.8	23.9	0.428	0.263	2.99	4.87
E6	104.4	25.2	27.6	0.876	0.852	2.88	2.96
E7	95.9	13.62	22.3	0.683*	0.321	1.99*	4.24
	101.3	16.73	21.5	0.659	0.537	2.66	3.38
S_1 =	8.2	6.23	3.46	0.274	0.277	0.28	0.85
E1	101.6 +	26.4 +	18.4 +	1.013 +	0.950 +	2.61 +	2.78
E2	107.0 +	14.5 +	24.5 +	0.480 +	0.434 +	3.02 +	3.34
E3	104.5 -	19.6 -	21.1 -	0.747 -	0.734 -	2.62 +	2.67
E4	94.2 -	13.9 +	17.5 +	0.487 +	0.414 +	2.85 +	3.44
E5	92.1 -	11.5 -	22.7 -	0.439 +	0.368 +	2.62 -	3.13
E6	104.5 +	26.1 +	26.9 -	0.858 -	0.984 +	3.14 +	2.65
E7	101.6 +	9.42 -	23.8 +		0.391 +		- 2.41
	100.8	17.35	22.1	0.670	0.611	2.81	2.92
n =				6			
S =	5.6	6.84	3.36	0.237	0.273	0.23	0.39
% Δ	- 0.8	+ 3.7	+ 2.8	+ 1.7	+ 13.6	+ 5.3	- 13.6
p <	NS	NS	NS	NS	NS	NS	

	TIME (Secs.)	WORK (W.)	fH (b/m)	fR (br.m)	$\dot{V}E$ (l.m.BTPS)	VT (ml.BTPS)	$\dot{V}O_2$ (l.min.STPD)	$\dot{V}CO_2$ (l.min.STPD)
E1	990	75	122	24	34.1	1422.5	1.046	0.919
E2	354	50	103	23	15.6	680.3	0.554	0.498
E3	873	75	133	26	30.8	1186.3	1.149	1.112
E4	398	50	107	24	11.5	479.7	0.337	0.308
E5	412	50	109	24	14.5	604.4	0.453	0.278
E6	439	50	118	35	30.6	873.5	0.813	0.814
E7	470	50	105	24	16.8	701.7	0.726*	0.548
	562.2	57.1	113.9	25.71	21.99	849.8	0.725	0.640
S =		12.2	10.9	4.19	9.42	339.0	0.330	0.316
E1	1200	100 +	120 -	25 +	39.5 +	1580.1 +	1.200 +	1.152 +
E2	384	50 o	109 +	26 +	17.1 +	658.9 -	0.480 -	0.447 -
E3	997	100 +	133 o	26 o	29.6 -	1136.6 -	1.014 -	0.954 -
E4	390	50 o	111 +	21 -	16.2 +	769.2 +	0.471 +	0.399 +
E5	486	50 o	107 -	25 +	14.5 +	578.5 -	0.500 +	0.433 +
E6	714	75 +	115 -	34 -	34.3 +	1009.5 +	0.910 +	1.042 +
E7	526	50 o	111 +	25 +	12.5 -	502.0 -		0.454 -
	671	67.9	115.1	26.0	23.39	890.7	0.762	0.697
n =							6	
S =		23.78	8.9	3.92	10.85	380.0	0.319	0.334
% Δ	+ 19.4	+ 18.7	+ 1.0	+ 1.2	+ 5.4	+ 4.8	+ 5.1	+ 7.8
p <		NS	NS	NS	NS	NS	NS	NS

	$F_{E_{O_2}}$ (%)	$F_{E_{CO_2}}$ (%)	FEO_2ET (%)	$FECO_2ET$ (%)	RQ	$\dot{V}A$ (L.min.BTPS)	$\dot{V}D$ (l.min.BTPS)
E1	3.80	3.34	5.77	4.93	0.88	23.0	11.1
E2	4.33	3.89	6.28	5.88	0.90	10.3	5.3
E3	4.56	4.41	6.46	6.04	0.97	22.5	8.3
E4	3.44	3.14	5.56	4.72	0.91	7.6	3.9
E5	3.79	2.32	6.45	3.68	0.61	9.1	5.4
E6	3.18	3.18	4.86	4.74	1.00	20.4	10.1
E7	5.22*	3.94	7.79*	5.74	0.76	11.5	5.37
	3.85	3.46	5.90	5.10	0.88	14.91	7.07
S =	0.52	0.68	0.63	0.84	0.14	6.75	2.76
E1	3.71 -	3.56 -	5.48 -	5.08 +	0.96 +	27.6 +	11.9 +
E2	3.38 -	3.15 -	5.92 -	5.26 -	0.93 +	10.2 -	6.9 +
E3	4.21 -	3.96 -	6.12 -	5.58 -	0.94 -	20.9 -	8.6
E4	3.55 +	3.01 -	6.11 +	4.98 +	0.85 -	9.7 +	6.4 +
E5	4.19 +	3.64 +	6.78 +	5.70 +	0.87 +	9.2 +	5.3 -
E6	3.19 +	3.66 +	4.60 -	5.08 +	1.14 +	24.6 +	9.7 -
E7	5.23*	4.50 +		6.06 +		9.3 -	3.3
	3.92	3.64	5.83	5.39	0.95	15.9	7.44
n =	6		6		6		
S =	0.69	0.50	0.74	0.40	0.10	8.13	2.87
% Δ	1.8	+ 4.9	- 1.2	+ 5.6	+ 7.9	+ 6.0	+ 4.4
p <	NS	NS	NS	NS	NS	NS	NS

	VT (l)	VO_2	VCO_2	VD	VA	FEO_2 (%)	$FECO_2$ (%)	$PETCO_2$ (mm.Hg)
E1	1.256	50.06	41.59	426.5	829.8	3.98	3.28	42.15
E2	0.558	22.94	19.74	209.4	348.6	3.77	3.20	43.83
E3	1.050	45.43	42.26	295.1	754.5	4.22	3.91	46.61
E4	0.554	19.52	16.98	194.5	360.0	3.39	2.96	39.29
E5	0.536	17.94	10.94	223.2	312.9	3.25	1.98	29.41
E6	0.914	31.73	30.89	276.4	637.3	3.44	3.34	41.42
E7	0.782	31.21*	26.50	259.8	522.0	3.87*	3.21	41.55
	0.807	31.27	27.00	269.3	537.9	3.67	3.13	40.61
S =	0.280	13.70	12.1	78.3	208.3	0.38	0.58	5.44
E1	1.434 +	55.08 +	51.63 +	410.1 -	1023.4 +	3.79 -	3.53 +	42.32 +
E2	0.587 +	19.43 -	17.60 -	241.3 +	345.9 -	3.12 -	2.81 -	41.64 -
E3	0.929 -	35.45 -	34.87 -	267.1 -	661.5 -	3.71 -	3.64 -	43.70 -
E4	0.791 +	27.78 +	23.59 +	296.9 +	494.5 +	3.45 +	2.95 -	40.66 +
E5	0.508 -	19.34 +	16.20 +	200.6 -	307.1 -	3.70 +	3.08 +	44.08 +
E6	0.969 +	31.88 +	34.60 +	278.1 +	691.0 +	3.28 -	3.55 +	43.17 +
E7	0.395 -	20.04 -	16.42 -	109.2 -	285.7 -	4.97	4.04 +	47.27 +
	0.802	31.49	27.84	257.6	544.2	3.51	3.37	43.26
n =		6				6		
S =	0.352	13.26	13.23	92.1	267.6	0.27	0.44	2.13
%Δ =	- 0.6	+ 0.7	+ 3.1	- 4.3	+ 1.1	- 4.3	+ 7.7	+ 6.5
p <	NS	NS	NS	NS	NS	NS	NS	NS

	$VT^{0.81}$						$VT^{0.81}$					
	VO_2	VCO_2	VD	VA	FEO_2 (%)	$FECO_2$ (%)	VO_2	VCO_2	VD	VA	FEO_2 (%)	$FECO_2$ (%)
E1	19.98	10.86	230.4	269.5	3.83	2.64	31.91	23.05	308.21	491.8	3.89	2.90
E2	19.70	16.73	195.7	303.9	3.49	2.91	36.60	32.32	265.21	534.8	4.97	4.40
E3	14.50	11.95	168.6	331.3	3.34	2.90	31.65	28.50	237.71	562.3	3.82	3.45
E4	17.05	14.93	176.9	323.0	3.17	2.79	30.69	26.26	273.5	526.5	4.40	3.74
E5	15.97	9.75	214.9	285.0	3.08	1.88	31.92	19.71	283.2	516.8	4.49	2.73
E6	12.57	11.66	197.6	302.4	2.80	2.63	26.47	25.61	254.7	545.3	3.27	3.15
E7	24.91*	18.91	162.8	337.2	4.94*	3.77	41.75*	30.86	251.2	548.7	5.28*	3.88
	16.63	13.54	192.4	307.5	3.28	2.79	31.54	26.62	267.7	532.3	4.14	3.46
S =	2.91	3.38	24.7	24.7	0.70	0.56	3.23	4.39	23.3	23.3	0.60	0.59
E1	19.16 −	15.04 +	134.2 −	365.8 +	3.07 −	2.59 −	30.70 −	25.89 +	222.9 −	577.1 +	3.30 −	2.89 −
E2	15.41 −	13.87 −	216.9 +	283.1 −	2.81 −	2.50 −	29.23 −	26.69 −	300.8 +	499.2 −	3.88 −	3.56 −
E3	13.54 −	13.26 +	170.8 −	329.2 −	2.86 −	2.73 −	28.88 −	28.39 −	238.2 +	561.8 −	3.46 −	3.37 −
E4	11.32 −	10.89 −	231.8 +	268.3 −	1.85 −	2.05 −	28.26 −	23.96 −	306.8 +	501.2 −	3.50 −	2.97 −
E5	18.92 +	15.83 +	198.5 −	301.5 +	3.67 +	3.05 +	34.94 +	30.10 +	280.2 −	519.8 +	4.84 +	4.23 +
E6	14.50 +	18.22 +	192.6 −	307.5 +	3.08 +	2.97 +	25.62 −	26.76 +	247.3 −	552.7 +	3.21 −	3.34 +
E7		22.15	129.8	370.2	5.37*	4.50	44.40*	38.51 +	188.54	611.6	6.50*	5.83 +
	15.47	15.61	182.1	317.9	2.89	2.91	29.61	28.61	255.0	546.2	3.7	3.74
n =	6				6		6				6	
S =	3.08	3.67	39.2	39.2	0.59	0.77	3.10	4.77	43.2	41.7	0.60	1.02
% Δ	− 7.0	+15.3	− 5.2	+ 3.4	−11.9	−4.3	−3.9	+7.5	− 4.7	+ 2.6	−10.6	+8.7
p <	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

	f_R (b.min)	V_E (l.min.BTPS)	f_R (b.min)	V_T (ml)	$\dot{V}O_2$ (l.min STPD)	$\dot{V}O_2$ (l.min. STPD)	R_Q
E1	82	15.3	15	1022.7	0.499	0.391	0.78
E2	98	15.6	23	680.3	0.554	0.498	0.90
E3	102	18.8	21	894.4	0.559	0.553	0.92
E4	87	10.8	21	513.0	0.334	0.291	0.87
E5	98	13.4	25	534.7	0.357	0.221	0.62
E6	111	27.9	28	998.1	0.820	0.801	0.98
E7	90.0	15.6	23	678.2	0.652*	0.491	0.75*
	95.4	16.772	22.3	760.2	0.520	0.464	0.84
S =	9.83	5.46	4.0	211.6	0.176	0.190	0.13
E1	84 +	19.2 +	16.0 +	1197.9 +	0.642 +	0.573 +	0.89 +
E2	109 +	15.9 +	23.0 +	691.7 +	0.449 -	0.417 -	0.93 -
E3	95 -	13.3 -	17.0 -	783.1 -	0.430 -	0.367 -	0.91 -
E4	100 +	14.9 +	19.0 -	785.7 -	0.451 +	0.383 +	0.85 -
E5	87 -	12.2 -	25.0 o	487.3 -	0.387 +	0.323 +	0.84 +
E6	100 -	24.0 -	25.0 -	959.2 -	0.657 -	0.712 -	1.08 +
E7	94.0 +	9.1 -	25.0 +	364.1 -		0.292 -	0.82*
	95.6	15.51	21.4	752.7	0.503	0.438	0.92
n =					6		6
S =	8.46	4.88	4.0	279.3	0.116	0.151	0.09
%Δ	0	- 7.5	- 4.0	- 1.0	- 3.3	- 5.4	+ 9.5
p <	NS	NS	NS	NS	NS	NS	NS

	FEO_2 (%)	$FECO_2$ (%)	FEO_2 (%)	$FECO_{2ET}$ (%)	$\dot{V}D$ (l.min.BTPS)	$\dot{V}A$ (l.min.BTPS)	PET_{CO2} (mm Hg)	$PETO_2$ (mm Hg)
E1	4.00	3.13	6.52	4.96	5.7	9.6	34.5	103.7
E2	4.33	3.89	6.28	5.88	5.3	10.3	41.1	101.2
E3	3.86	3.56	5.78	5.09	5.7	13.1	36.3	108.6
E4	3.51	3.09	5.80	4.71	3.7	7.0	35.8	108.6
E5	3.16	1.96	5.82	3.36	5.6	7.7	24.8	111.4
E6	3.56	3.47	5.15	4.87	8.1	19.9	35.0	113.0
E7	5.08*	3.83	7.69*	5.61	5.0	10.6	40.1	97.9*
	3.69	3.28	5.89	4.93	5.59	11.2	35.37	107.7
S =	0.41	0.66	0.47	0.81	1.31	4.34	5.30	4.5
E1	4.09 +	3.65 +	5.91	5.01 +	5.2 -	13.9 -	29.9 -	108.9 -
E2	3.43 -	3.19 -	5.91	5.21 -	6.2 +	9.7 -	37.2 -	108.8 +
E3	3.59 -	3.28 -	5.57 -	4.76 -	4.2 -	9.1 -	34.4 -	109.8 +
E4	3.67 +	3.11 +	6.00 +	4.89 +	5.5 +	9.5 +	35.0 -	108.5 -
E5	3.88 +	3.23 +	6.45 +	5.20 +	4.6 -	7.5 -	37.1 +	105.6 -
E6	3.30 -	3.58 +	4.78 -	5.01 +	6.9 -	17.1 -	36.3 +	115.4 +
E7	4.88*	4.00 +	6.97*	5.55 -	2.6 -	6.5 -	37.9 -	100.5* +
	3.66	3.43	5.77	5.09	5.03	10.47	35.40	109.5
n =	6		6					6
S =	0.29	0.32	0.56	0.25	1.41	3.73	2.73	3.2
% Δ	- 0.8	+ 4.9	- 2.0	+ 3.2	- 9.9	- 6.3	0	+ 1.7
p <	NS	NS	NS	NS	NS	NS	NS	NS

RESPONSES OF THE EXPERIMENTAL GROUP TO ~~BICYCLE~~ TREADMILL ERGOMETER WORK TESTS BEFORE AND AFTER TRAINING

Individual Responses, group means & S.D, percentage changes and level of significance (n = 7 unless otherwise stated).

	TIME (Secs)	fH (bt)	VE (l.BTPS)	VA (l.BTPS)	fR (b)	VO_2 (l.STPD)	VCO_2 (l.STPD)
E1	694	1343.7	318.42	206.8	235	14.501	10.306
E2	404	671.4	103.80	68.6	142	4.756	4.131
E3	723	1388.8	273.78	193.7	284	14.912	12.276
E4	377	615.5	88.42	60.9	119	4.007	2.920
E5	421	658.7*	79.20	47.3	180	3.401	2.513
E6	724	1299.9	361.62	250.4	492	14.423	11.806
E7	665	944.0	217.79	141.2	290	10.595*	7.472
	572.6	1043.9	206.14	138.4	248.9	9.333	7.346
S =	162.5	348.6	116.86	81.1	126.0	5.80	4.208
E1	772 -	1332.8 -	281.12 -	209.0 +	228 -	16.318 +	11.592 +
E2	644 +	1054.2 +	164.91 +	113.2 +	230 +	8.166 +	6.737 +
E3	1200 +	2170.7 +	416.52 +	297.3 +	404 +	21.092 +	15.924 +
E4	440 +	694.5 +	115.37 +	76.0 +	139 +	4.300 +	3.583 +
E5	664 +		147.56 +	104.4 +	280 +	7.720 +	7.436 +
E6	767 +	1287.8 -	204.41 -	138.2 -	314 -	7.475 -	5.567 +
E7		1069.8 +	180.22 -	134.7 -	251 -		7.946 +
	736.1	1268.3	215.73	153.3	263.7	10.845	8.334
n =	6	6				6	
S =	232.2	496.9	102.70	75.7	82.76	6.42	4.183
%Δ	+ 28.6	+ 21.5	+ 4.6	+ 10.8	+ 3.9	+ 16.2	+ 13.4
p<	0.05	NS	NS	NS	NS	NS	0.02

	fH (b.min)	VE (l.min.BTPS)	fR (b.min)	VO_2 (l.min.STPD)	VCO_2 (l.min.STPD)	VEO_2 (ml.100 ml)	$VECO_2$ (ml.100 ml)
E1	116.2	27.52	20.3	1.254	0.890	2.19	3.09
E1	99.6	15.41	21.1	0.706	0.613	2.18	2.51
E3	115.2	22.72	23.6	1.237	1.019	1.84	2.23
E4	87.7	12.60	17.0	0.571	0.416	2.21	3.03
E5	93.9*	11.29	25.6	0.485	0.358	2.33	3.15
E6	107.7	29.97	40.8	1.195	0.978	2.51	3.06
E7	85.2	19.65	26.2	0.956	0.674	2.06	2.91
	101.9	19.88	24.9	0.908	0.707	2.21	2.85
S =	13.42	7.246	7.69	0.359	0.265	0.220	0.348
E1	103.6 -	21.84 -	17.7 -	1.268 -	0.901 +	1.72 -	2.42 -
E2	95.5 -	15.36 -	21.4 +	0.760 +	0.628 +	2.81 +	2.44 -
E3	108.5 -	20.86 -	20.2 -	1.054 -	0.796 -	1.98 +	2.62 +
E4	94.7 +	15.73 +	18.9 +	0.586 +	0.489 +	2.68 +	3.22 +
E5		13.33 +	25.3 -	0.698 +	0.671 +	1.91 -	1.99 -
E6	100.7 -	15.99 -	24.6 -	0.585 -	0.435 -	2.73 +	3.68 +
E7	96.4 +	16.23 -	22.6 -		0.716 +		2.27 -
	99.9	17.05	21.5	0.825	0.662	2.30	2.66
n =	6			6		6	
S =	5.42	3.101	2.83	0.277	0.164	0.486	0.586
%Δ	- 2.0	-14.2	-13.6	- 9.1	-6.4	+ 4.1	- 6.7
p<	NS	NS	NS	NS	NS	NS	NS

	TIME (Secs)	GRADE (deg.)	fH (b.min)	fR (b.min)	$\dot{V}E$ (l.min BTPS)	VT (ml)	$\dot{V}_{O2}$ (l.min STPD)	$\dot{V}_{CO2}$ (l.min STPD)
E1	694	12	125	27	39.9	1478.2	1.360	1.059
E2	404	6	97	23	17.2	746.1	0.651	0.552
E3	723	12	133	25	28.4	1134.1	1.348	1.150
E4	377	6	115	22	15.7	746.0	0.559	0.422
E5	421	6	109	27	11.0	405.9	0.462	0.339
E6	724	12	120	46	36.9	802.7	1.239	0.985
E7	665	12	109	26	19.9	766.4	0.863*	0.617
	572.5	9.4	115.4	28.0	24.14	868.5	0.936	0.732
S =		3.21	11.86	8.16	11.1	341.8	0.422	0.327
E1	772 +	12 o	115 -	29 +	36.0 -	1242.1 -	1.547 +	1.217 +
E2	644 +	12 +	97 o	23 o	17.3 +	754.0 +	0.705 +	0.594 +
E3	1200 +	15 +	133 o	25 o	29.2 +	1167.6 -	1.259 -	0.998 -
E4	440 +	6 o	111 -	22 o	17.4 +	791.3 +	0.543 -	0.463 +
E5	664 +	12 +	109 o	27 o	17.2 +	637.9 +	0.796 +	0.761 +
E6	767 +	12 o	111 -	39 -	24.9 -	829.7 +	0.716 -	0.584 -
E7	666 +	12 o	109 o	25 -	19.2 -	796.0 +		0.731 +
	736.1	11.6	112.1	27.1	23.03	888.4	0.928	0.764
S =		2.70	10.76	5.73	7.34	225.5	0.388	0.262
% Δ	+ 28.6	+ 23.4	- 2.0	- 3.3	- 4.6	+ 1.7	- 0.1	+ 4.4
p <	0.02	NS	NS	NS	NS	NS	NS	NS

	FE_{O2} (%)	FE_{CO2} (%)	FE_{CO2} Max. (%)	FE_{CO2} Max. (%)	RQ	$\dot{V}A$ (l.min BTPS)	$\dot{V}D$ (l.min BTPS)
E1	4.18	3.25	6.11	4.99	0.78	25.9	14.0
E2	4.61	3.92	7.53	5.91	0.85	11.3	5.8
E3	5.89	5.02	8.83	7.30	0.85	19.5	8.9
E4	4.40	3.33	7.42	5.03	0.75	10.1	5.2
E5	5.08	3.73	8.06	5.84	0.73	7.0	4.0
E6	4.02	3.19	5.78	4.47	0.79	26.3	10.6
E7	5.24*	3.75	7.82*	5.48	0.72*	13.6	6.3
	4.70	3.741	7.29	5.57	0.79	16.2	7.83
S =	0.692	0.630	1.159	0.915	0.05	7.74	3.53
E1	5.25 +	4.12 +	7.39 +	5.58 +	0.79 +	26.6 +	9.4 -
E2	4.80 -	4.13 +	7.49 -	6.10 +	0.84 -	11.7 +	5.6 -
E3	5.30 -	4.20 -	7.73 -	5.84 -	0.79 -	20.9 +	8.2 -
E4	3.69 -	3.13 -	6.02 -	4.88 -	0.85 +	11.1 +	6.3 +
E5	5.67 +	5.43 +	8.03 -	7.53 +	0.96 +	12.4 +	4.8 +
E6	3.48 -	2.83 -	5.44 -	4.30 -	0.81 +	16.3 -	8.5 -
E7		4.65 +		6.06 +		14.7 +	4.5 -
	4.72	4.07	7.02	5.76	0.84	16.2	6.76
S =	0.911	0.878	1.037	1.023	0.06	5.67	1.94
% Δ	+ 0.4	+ 8.8	- 3.7	+ 3.4	+ 6.3	0	- 13.7
p <	NS	NS	NS	NS	NS	NS	NS

	VT (l)	VO_2	VCO_2	VD	VA	FE_{O2} (%)	FE_{CO2} (%)	PET_{CO2} (mm Hg)	PET_{O2} (mm Hg)
E1	1.355	61.70	43.85	474.6	880.3	4.54	3.21	42.29	90.8
E2	0.731	33.50	28.09	248.5	482.6	4.51	3.80	49.06	89.9
E3	0.964	52.51	43.23	281.4	682.4	5.31	4.36	52.02	
E4	0.743	33.67	24.54	231.5	511.6	4.43	3.24	40.65	94.7
E5	0.440	18.89	13.96	176.9	263.2	4.29	3.16	45.48	89.9
E6	0.735	29.32	22.53	226.3	509.0	3.92	3.91	37.86	102.4
E7	0.751	36.53*	25.77	264.4	486.5	4.87*	3.43	44.89	88.4*
	0.817	38.27	28.83	271.9	545.1	4.50	3.46	44.61	93.5
S =	0.282	15.8	10.97	95.3	191.7	0.46	0.47	4.87	5.3
E1	1.233	71.57 +	50.84 +	316.8 -	916.7 +	5.78 +	4.10 +	47.23 +	84.7 -
E2	0.717	35.51 +	29.29 +	225.1 -	492.2 +	4.86 +	4.00 +	50.37 +	89.4 -
E3	1.031	52.21 -	39.42 -	295.0 +	936.2 +	4.90 -	3.69 -	44.14 -	91.1*
E4	0.830	30.94 -	25.78 +	282.1 +	547.2 +	3.67 -	3.06 -	39.87 -	102.0 +
E5	0.527	27.57 +	26.56 +	153.3 -	373.9 +	5.18 +	5.00 +	60.01 +	85.0 -
E6	0.651	23.81	17.73 -	210.8 -	440.5 -	3.53 -	2.60 -	33.63 -	105.7 +
E7	0.718		31.66 +	180.7 -	537.0 +		4.37 +	49.94 +	
	0.815	40.27	31.61	237.7	577.7	4.65	3.83	46.46	93.4
n =		6				6			5
S =	0.242	18.25	10.70	61.6	187.2	0.88	0.80	8.42	9.84
% Δ	- 0.2	+ 5.2	+ 9.6	- 12.6	+ 5.7	+ 3.3	+ 10.7	+ 4.3	- 3.5
p <	NS	NS	NS	NS	NS	NS	NS	NS	NS

	$\frac{VT^{0.51}}{V_{O2}}$	V_{CO2}	VD	VA	FE_{O2} (%)	FE_{CO2} (%)
E1	19.98	9.78	210.9	289.1	4.19	2.61
E2	21.61	18.75	160.2	339.8	4.10	3.54
E3	16.22	12.14	182.8	317.2	3.73	2.89
E4	17.47	13.48	187.3	312.7	3.26	2.55
E5	21.62	16.00	197.8	302.2	4.31	3.19
E6	16.44	12.46	180.2	319.8	3.48	2.64
E7	26.79*	18.68	181.2	318.8	4.94*	3.46
	18.89	14.46	185.8	314.2	3.84	2.98
S =	2.50	3.45	15.77	15.77	0.42	0.41
E1	25.63 +	15.29 +	114.1 -	385.9 +	5.23 +	3.40 +
E2	22.09 +	18.11 -	167.5 +	332.5 -	4.15 +	3.40 -
E3	17.49 +	13.01 +	156.2 -	368.7 +	3.61 -	2.68 -
E4	16.31 -	13.16 -	162.1 -	337.9 +	2.99 -	2.47 -
E5	25.98 +	25.08 +	145.6 -	354.4 +	5.14 +	4.99 +
E6	17.09 +	12.29 -	173.7 -	326.3 +	3.16 -	2.25 -
E7		18.02 -	150.9 -	349.1 +		3.78 +
	20.76	16.43	152.9	350.7	4.05	3.28
n =	6				6	
S =	4.40	4.49	19.59	21.09	0.97	0.94
% Δ	+ 9.9	+ 13.5	+ 17.7	+ 11.6	+ 5.5	+ 10.1
p <	NS	NS	0.05	0.05	NS	NS

	$V_T^{0.01}$ V_{O2}	V_{CO2}	VD	VA	FE_{O2} (%)	FE_{CO2} (%)
E1	34.05	21.70	303.4	609.9	4.31	2.82
E2	37.05	30.88	274.9	525.1	4.64	3.88
E3	39.69	32.25	246.6	553.4	4.75	3.84
E4	37.64	27.15	241.5	558.5	4.71	3.41
E5	35.31	26.21	302.1	497.9	4.46	3.33
E6	32.85	25.30	239.0	561.0	4.04	3.11
E7	38.45*	27.15	280.7	519.3	4.86*	3.43
	36.10	27.23	269.7	546.4	4.48	3.40
S =	2.52	3.52	27.7	35.51	0.27	0.38
E1	44.42 +	29.83 +	197.0 -	603.0 -	5.46 +	3.68 +
E2	40.6 +	33.54 +	247.0 -	552.9 +	5.12 +	4.23 +
E3	37.10 -	28.29 -	234.6 -	605.4 +	4.34 -	3.25 -
E4	29.63 -	24.64 -	273.6 +	528.6 -	3.61 -	3.00 -
E5	43.47 +	41.32 +	230.2 -	569.8 +	5.52 +	5.21 +
E6	30.40 -	23.06 -	247.3 +	552.7 -	3.89 -	2.94 -
E7		36.81 +	192.0 -	607.9 +		4.60 +
	37.60	31.07	231.7	574.3	4.66	3.84
n =	6				6	
S =	6.41	6.56	28.94	31.50	0.82	0.87
% Δ	+ 4.2	+ 14.1	- 14.1	+ 5.1	+ 4.0	+ 12.9
p <	NS	NS	NS	NS	NS	NS

	fH (b.min)	fR (b.min)	VE (l.min.BTPS)	VT (ml)	$\dot{V}_{O2}$ (l.min.STPD)	$\dot{V}_{CO2}$ (l.min.STPD)	RQ
E1	91	18	20.8	1.156	0.755	0.533	0.71
E2	97	24	17.3	0.720	0.650	0.552	0.85
E3	105	22	19.8	0.898	0.812	0.673	0.83
E4	100	21	15.2	0.746	0.558	0.421	0.76
E5	95*	26	10.7	0.411	0.392	0.287	0.73
E6	100	39	25.7	0.659	0.815	0.608	0.75
E7	90	24	14.4	0.601	0.568*	0.396	0.70*
	97.2	24.9	17.77	0.742	0.664	0.496	0.77
S =	5.78	6.74	4.86	0.235	0.166	0.134	0.06
E1	94 +	15 -	15.6 -	1.040 -	0.762 +	0.514 -	0.68 -
E2	94 -	21 -	15.3 -	0.729 +	0.624 -	0.514 -	0.82 -
E3	94 -	16 -	15.1 -	0.942 +	0.597 -	0.436 -	0.73 -
E4	104 +	19 -	16.0 +	0.842 +	0.535 -	0.439 +	0.82 +
E5		23 -	13.6 +	0.590 +	0.589 +	0.558 +	0.95 +
E6	91 -	23 -	12.0 -	0.610 -	0.419 -	0.295 -	0.70 -
E7	90 o	22 -	15.0 +	0.683 +		0.535 +	
	94.5	19.9	14.66	0.777	0.588	0.470	0.78
n =	6				6		6
S =	4.97	3.29	1.39	0.170	0.112	0.090	0.10
% Δ	- 2.8	-20.1	-17.2	+4.7	-11.4	-5.2	+1.3
p <	NS	0.02	NS	NS	NS	NS	NS

	FEO_2 (%)	FE_{CO2} (%)	FEO_2ET (%)	$FE_{CO2}ET$ (%)	$\dot{V}D$ (l.min.BTPS)	$\dot{V}A$ (l.min.BTPS)	PA_{CO2} (mm.Hg)	PAO_2 (mm.Hg)
E1	4.43	3.11	6.62	4.84	7.5	13.3	34.4	101.6
E2	4.55	3.88	7.39	5.81	5.8	11.5	41.0	100.1
E3	5.09	4.22	7.31	5.85	5.5	14.2	40.2	98.5
E4	4.33	3.27	7.27	4.85	5.1	10.5	34.6	104.8
E5	4.44	3.26	7.42	5.37	4.2	6.5	38.6	98.9
E6	3.76	2.81	5.82	4.23	8.7	17.0	31.3	109.8
E7	4.77*	3.33	7.29*	4.97	4.8	9.6	35.6	100.6*
	4.43	3.41	6.97	5.13	5.94	11.8	36.5	102.3
S =	0.43	0.48	0.64	0.58	1.60	3.41	3.52	4.33
E1	5.93 +	4.00 +	8.25 +	5.33 +	3.9 -	11.7 -	37.9 +	95.5 -
E2	4.94 +	4.07 +	7.45 +	5.91 +	4.8 -	12.5 -	42.4 +	99.2 -
E3	4.72 -	3.45 -	6.62 -	4.59 -	3.8 -	11.3 -	33.2 -	104.0 +
E4	3.93 -	3.23 -	6.03 -	4.70 -	5.0 -	11.0 +	34.8 +	108.1 +
E5	5.33 +	5.04 +	7.62 +	7.05 +	3.9 -	9.7 +	49.3 +	95.4 -
E6	3.90 +	2.75 -	5.79 -	3.94 -	3.9 -	8.9 -	29.0 -	110.7 +
E7		4.35 +		5.83 +	3.8 -	11.2 +	41.0 +	
	4.79	3.84	6.96	5.34	4.16	10.6	38.2	102.1
n =	6		6					6
S =	0.79	0.76	0.97	1.03	0.51	0.99	6.71	6.55
%Δ	+ 8.1	+ 12.6	- 0.1	+ 4.1	- 30.0	- 10.2	+ 4.7	- 0.2
p<	NS	NS	NS	NS	0.02	NS	NS	NS

APPENDIX H

RESPONSES OF CONTROL GROUP TO TREADMILL WORK TESTS BEFORE AND AFTER TRAINING

Individual values, group means and S.D., and percentage changes (n = 3 unless stated otherwise; subjects marked with * are not included in group analysis)

AGGREGATE VALUES

Time (secs)	f_H (b.)	V_E (L.BTPS)	V_A (L.BTPS)	f_R (b.)	V_{O_2} (L.STPD)	V_{CO_2} (L.STPD)
245*	382.8	59.67	40.97	85	2.110	2.066
702	1470.4	183.46	140.73	183	10.091	8.941
419	598.9	148.00	102.08	160	6.501	4.976
310	394.0	73.17	25.43	136	2.990	2.523
196*	312.6	71.63	47.88	74	2.344	1.866
477	821.1	134.88	89.41	159.7	6.527	5.480
202.3	571.3	56.3	58.68	23.5	3.55	3.239
660	1240.8	218.60	165.80	200	9.742	9.466
730	1073.7	216.61	155.12	277	9.851	9.452
97	150.9	27.13	18.32	38	1.093	0.929
495.7	821.8	154.11	113.08	171.7	6.895	6.616
347.0	587.0	110.0	82.24	122.0	5.025	4.925
% +3.4	0	+14.3	+26.5	+7.5	+5.6	+20.7

RELATED TO TIME

f_H	V_E	f_R	$\dot{V}_{O_2}$	$\dot{V}_{CO_2}$	$V_{E_{O_2}}$	$V_{E_{CO_2}}$
(b.min)	(L.min BTPS)	(b.min)	(L.min STPD)	(L.min STPD)	(ml.100 ml)	(ml.100 ml)
93.7	14.613	20.8	0.517	0.506	2.83	2.89
22.5	15.680	15.6	0.862	0.764	1.82	2.05
85.6	21.193	22.9	0.931	0.716	2.28	2.96
78.9	14.162	26.3	0.579	0.488	2.45	2.90
04.2	21.928	22.7	0.718	0.571	3.05	3.84
95.7	17.012	21.6	0.791	0.656	2.18	2.64
23.5	3.70	5.47	0.186	0.147	0.32	0.51
12.8	19.873	18.2	0.886	0.860	2.24	2.31
88.2	17.804	22.8	0.810	0.777	2.20	2.29
75.4	16.781	23.5	0.676	0.575	2.48	2.92
92.1	18.153	21.5	0.791	0.737	2.31	2.51
19.0	1.575	2.879	0.106	0.147	0.15	0.358
-3.9	+6.7	-0.5	0	+12.3	+6.0	+5.2

MAXIMAL VALUES

Time (secs)	Work (W)	f_H (b.min)	f_R (b.min)	$\dot{V}_E$ (L.min BTPS)	V_T (ml)	$\dot{V}_{O_2}$ (L.min STPD)	$\dot{V}_{CO_2}$ (L.min STPD)
90	25	100	22	14.9	678.9	0.393	0.413
525	50	139	17	20.2	1189.0	0.898	0.910
589	50	84	21	21.5	1025.6	0.822	0.789
603	75	88	29	24.5	845.8	0.877	0.864
184	25	125	23	23.4	1018.0	0.631	0.513
	45	107.2	22.4	20.9	951.5	0.724	0.698
	20.9	23.9	4.3	3.74	194.8	0.213	0.221
100	25 o	98.4 -	16 -	11.1 -	696.3 -	0.352 -	0.284 -
474	50 o	127.7 -	18 +	17.6 -	977.1 -	0.751 -	0.682 -
660	75 +	84.5 +	24 +	20.0 -	835.2 -	0.725 -	0.696 -
300	50 -	82.0 -	24 -	19.5 -	868.4 +	0.675 -	0.597 -
217	25 o	111.1 -	21 -	21.5 -	1025.8 +	0.420 -	0.390 -
	45	100.7	20.6	17.9	880.4	0.585	0.530
	20.9	19.1	3.6	4.07	129.2	0.185	0.184
	0	-6.1	-8.1	-14.3	- 11.3	-19.2	-24.1

MAXIMAL VALUES

	$F_{E_{CO_2}}$ (%)	$F_{E_{O_2ET}}$ (%)	$F_{E_{CO_2ET}}$ (%)	RQ	$\dot{V}_A$ (L.min BTPS)	$\dot{V}_D$ (L.min BTPS)
1*	3.53	5.30	5.14	0.99	10.7	4.9
2	5.41	8.17	7.10	0.91	14.1	4.4
3	3.30	6.45	4.95	0.78	15.0	7.6
4	3.86	6.64	5.55	0.85	11.9	5.2
5*	2.67	5.07	3.97	0.81	15.7	7.7
	4.19	7.09	5.87	0.85	13.7	5.7
=	1.09	0.94	1.11	0.06	1.59	1.67
2	4.94	6.73	6.55	1.02	17.2	5.7
3	4.43	6.44	6.34	1.01	18.1	7.8
4	3.26	5.53	4.72	0.86	10.9	4.9
	4.21	6.23	5.87	0.96	15.4	6.1
=	0.86	0.63	1.0	0.09	3.92	1.50
%	+0.5	-12.1	0	+12.9	+12.4	+7.0

SINGLE BREATH VALUES (ml ATPS unless otherwise stated)

	V_T (l)	V_{O_2}	V_{CO_2}	V_D	V_A
C1 *	0.708	24.82	24.30	226.7	481.8
C2	1.003	55.14	48.86	234.2	768.7
C3	0.925	40.63	31.10	287.3	637.8
C4	0.538	21.98	18.55	186.8	351.5
C5 *	0.968	31.67	25.22	320.8	646.7
	0.822	39.25	32.84	236.1	586.0
S =	0.249	16.62	15.23	50.28	213.4
C2	1.093	48.71	47.33	263.7	828.9
C3	0.782	35.56	34.12	222.1	559.8
C4	0.714	28.76	27.44	231.7	482.4
	0.863	37.68	36.30	239.2	623.7
S =	0.202	10.14	10.12	21.78	181.9
Δ %	+5.0	-4.5	+10.5	+1.1	+6.4

SINGLE BREATH VALUES (ml ATPS unless otherwise stated)

	$F_{E}O_2$ (%)	$F_{E}CO_2$ (%)	$P_{ET}CO_2$ mm.Hg.	$P_{ET}O_2$ mm.Hg.
C1	3.19	3.36	45.77	99.1
C2	5.01	4.93	54.78	90.1*
C3	4.56	4.18	48.87	95.0
C4	4.29	4.07	48.90	96.2
C5	3.29	2.60	33.46	106.2*
	4.07	3.82	46.36	96.8
S =	0.80	0.89	7.91	2.11
C1	3.83 +	3.11 -	38.85	98.0
C2	4.72 -	4.21 -	47.02	
C3	4.40 -	3.91 -	45.58	96.0
C4	4.03 -	3.50 -	44.91	98.6
C5	2.35 -	2.05 -	32.28	
	3.87	3.36	41.73	97.5
n =				3
S =	0.91	9.84	6.13	1.36
Δ %	-4.9	-12.3	-10.0	+0.7

PREDICTED VALUES (ml ATPS unless otherwise stated)

	V_T 0.5l V_{O_2}	V_{CO_2}	V_D	V_A	$F_{E_{O_2}}$ (%)	$F_{E_{CO_2}}$ (%)
C1 *	13.71	13.56	186.3	313.7	2.85	2.82
C2	22.27	20.92	154.5	345.5	3.91	3.56
C3	16.11	12.63	198.2	301.8	3.84	2.95
C4	19.38	16.36	181.5	318.5	3.60	3.04
C5 *	10.53	8.37	215.1	248.8	2.38	1.89
	19.25	16.64	178.1	321.9	3.78	3.18
S =	3.08	4.15	22.1	22.1	0.16	0.33
2	13.01	13.42	191.7	308.2	2.80	2.72
3	22.26	20.34	143.7	356.3	4.34	3.97
4	16.06	13.97	193.7	306.3	3.24	2.81
	17.11	15.91	176.4	323.6	3.46	3.17
=	4.71	3.85	28.3	28.3	0.79	0.70
%	-11.7	-4.4	-0.1	+0.5	-8.5	-0.3

PREDICTED VALUES (ml ATPS unless otherwise stated)

	V_T 0.8L/min V_{O_2}	V_{CO_2}	V_D	V_A	$F_{E_{O_2}}$ (%)	$F_{E_{CO_2}}$ (%)
C1 *	29.70	29.01	244.5	555.5	3.67	3.58
C2	41.88	37.58	202.1	597.9	4.78	4.28
C3	33.51	25.66	261.1	538.9	4.20	3.22
C4	39.75	33.47	219.9	576.9	5.79	4.86
C5 *	24.10	19.18	282.9	517.1	2.89	2.30
	38.38	32.24	227.7	571.2	4.92	4.12
S =	4.35	6.05	30.3	29.9	0.80	0.83
C2	31.08	30.59	228.2	571.8	3.58	3.47
C3	36.41	35.00	227.6	572.8	4.50	4.28
C4	33.86	28.64	246.9	553.1	4.23	3.58
	33.78	31.41	234.2	565.9	4.10	3.78
S =	2.67	3.26	11.0	11.10	0.47	0.44
%	-12.0	-2.3	+2.8	-0.9	-16.7	-8.2

APPENDIX H

INDIVIDUAL RESPONSES OF THE CONTROL GROUP TO BICYCLE ERGOMETER WORK TESTS BEFORE AND AFTER TRAINING

Individual values, group means, S.D., and percentage changes
(n=5 unless otherwise stated)

AGGREGATE VALUES

Time (secs)	Work (W)	f_H (b)	V_E (L.BTPS)	V_A (L.BTPS)	f_R (b)	V_{O_2} L.STPD	V_{CO_2} L.STPD
90	37.5	198.4	22.916	14.351	34	0.745	0.784
525	312.5	1089.2	144.144	110.097	117	7.256	7.256
589	365.8	779.1	183.112	135.172	188	8.453	7.771
603	377.4	738.7	196.850	140.716	254	8.597	8.217
184	78.3	360.2	65.93	44.286	66	2.141	1.746
398.2	234.3	633.1	122.59	88.924	131.8	5.458	5.155
= 242.5	163.5	354.9	75.477	56.62	89.7	3.730	3.583
100 +	41.7 +	195.2 -	18.642 -	12.818 -	26 -	0.736 -	0.597 -
474 -	269.9 -	906.0 -	115.596 -	89.034 -	114 -	5.589 -	4.986 -
660 +	450.0 +	840.1 +	192.918 +	142.700 +	237 +	8.711 +	7.804 +
300 -	120.0 -	389.8 -	89.010 -	115.000 -	115 -	3.737 -	3.251 -
217 +	80.4 +	394.3 +	65.088 -	35.93 -	72 +	1.582 -	1.393 -
350.2	192.4	545.1	96.107	79.096	112.8	4.071	3.606
= 220.3	168.0	310.9	64.472	54.059	78.5	3.212	2.898
% - 12.0	- 17.9	- 13.9	- 21.5	- 11.0	- 6.8	-25.4	-30.0

RELATED TO TIME

f_H (b.min)	$\dot{V}_E$ (L.MIN STPS)	f_R (b.min)	$\dot{V}_{O_2}$ (L.MIN STPD)	$\dot{V}_{CO_2}$ (L.MIN STPD)	$V_{E_{O_2}}$ (ml. 100ml)	$V_{E_{CO_2}}$ (ml. 100ml)
99.2	15.277	22.7	0.496	0.523	3.09	2.92
124.5	16.474	13.4	0.841	0.829	1.99	2.08
79.4	18.653	19.1	0.861	0.792	2.17	2.35
73.5	19.587	25.3	0.855	0.599	2.29	3.27
120.0	21.499	21.5	0.698	0.569	3.08	3.78
99.3	18.298	20.4	0.750	0.662	2.52	2.88
23.0	2.474	4.51	0.157	0.138	0.523	0.69
97.6 -	11.185 -	15.6 -	0.442 -	0.358 -	2.53 -	3.12 +
114.6 -	14.632 -	14.4 -	0.707 -	0.631 -	2.07 +	2.32 +
76.4 -	17.538 -	21.5 +	0.792 -	0.709 -	2.21 -	2.47 +
78.0 -	17.802 -	23.0 -	0.747 -	0.650 +	2.38 +	2.74 -
109.0 -	17.997 -	19.9 -	0.437 -	0.385 -	4.12 +	4.67 +
95.1	15.83	18.9	0.625	0.547	2.66	3.06
17.5	2.937	3.73	0.172	0.163	0.833	0.948
% - 4.2	-13.5	- 7.4	-16.7	-17.4	+ 5.6	+ 6.2

MAXIMAL VALUES

Time (secs)	Work (W)	f_H (b.min)	f_R (b.min)	$\dot{V}_E$ (L.min BTPS)	V_T (ml)	$\dot{V}_{O_2}$ (L.min STPD)	$\dot{V}_{CO_2}$ (L.min STPD)
90	25	100	22	14.9	678.9	0.393	0.413
525	50	139	17	20.2	1189.0	0.898	0.910
589	50	84	21	21.5	1025.6	0.822	0.789
503	75	88	29	24.5	845.8	0.877	0.864
184	25	125	23	23.4	1018.0	0.631	0.513
	45	107.2	22.4	20.9	951.5	0.724	0.698
	20.9	23.9	4.3	3.74	194.8	0.213	0.221
100	25 o	98.4 -	16 -	11.1 -	696.3 -	0.352 -	0.284 -
474	50 o	127.7 -	18 +	17.6 -	977.1 -	0.751 -	0.682 -
660	75 +	84.5 +	24 +	20.0 -	835.2 -	0.725 -	0.696 -
300	50 -	82.0 -	24 -	19.5 -	868.4 +	0.675 -	0.597 -
217	25 o	111.1 -	21 -	21.5 -	1025.8 +	0.420 -	0.390 -
	45	100.7	20.6	17.9	880.4	0.585	0.530
	20.9	19.1	3.6	4.07	129.2	0.185	0.184
	0	-6.1	-8.1	-14.3	- 11.3	-19.2	-24.1

MAXIMAL VALUES

$F_{E_{O_2}}$ (%)	$F_{E_{C_{O_2}}}$ (%)	$F_{E_{O_2ET}}$ (%)	$F_{E_{C_{O_2ET}}}$ (%)	RQ	$\dot{V}_A$ (L.min BTPS)	$\dot{V}_D$ (L.min BTPS)
3.18	3.33	5.00	5.10	1.05	9.7	5.2
5.46	5.54	7.39	7.14	1.01	15.7	4.6
4.64	4.46	6.54	6.12	0.96	15.7	5.9
4.35	4.28	6.16	5.90	0.98	17.8	6.8
3.25	2.64	5.03	3.96	0.81	15.6	7.8
4.18	4.05	6.02	5.64	0.96	14.9	6.06
0.97	1.11	1.02	1.19	0.09	3.05	1.27
3.69 +	2.97 -	5.22 +	4.11 -	0.81 -	8.0 -	3.1 -
5.20 -	4.73 -	7.01 -	6.07 -	0.91 -	13.7 -	3.9 -
4.43 -	4.25 -	6.10 -	5.76 -	0.96 o	14.8 -	5.3 -
4.17 -	3.69 -	6.21 +	5.47 -	0.88 -	13.1 -	6.4 -
2.34 -	2.18 -	4.39 -	3.94 -	0.93 +	11.9 -	9.7 +
3.97	3.56	5.79	5.07	0.90	12.3	5.68
1.06	1.01	1.01	0.98	0.06	2.62	2.58
-5.0	-12.1	-3.8	-10.1	-6.2	-17.4	-6.3

SINGLE BREATH VALUES (ml ATPS unlessotherwise stated)

	V_T (l)	Vo_2	Vco_2	V_D	V_A
C1	0.674	21.91	23.06	251.7	422.1
C2	1.232	63.12	62.02	286.6	945.1
C3	0.974	44.96	41.33	255.0	719.3
C4	0.775	33.85	32.35	220.8	554.1
C5	0.999	32.44	26.45	327.6	671.3
	0.931	39.26	37.04	268.3	662.4
S=	0.216	15.64	15.59	40.5	195.4
C1	0.717 +	28.31 +	22.97 -	223.0 -	493.7 +
C2	1.014 -	49.03 -	43.74 -	232.1 -	781.9 -
C3	0.814 -	36.76 -	32.93 -	212.3 -	601.6 -
C4	0.774 -	32.49 -	28.27 -	251.2 +	523.0 -
C5	0.904 -	21.98 -	19.35 -	404.5 +	499.5 -
	0.845	33.71	29.45	264.6	579.9
n =					
S =	0.117	10.15	9.51	79.49	120.8
Δ %	-9.2	-14.1	-23.19	+0.5	-12.4

SINGLE BREATH VALUES (ml ATPS unless otherwise stated)

	$F_{E}O_2$ (%)	$F_{E}CO_2$ (%)	$P_{ET}CO_2$ mm.Hg.	$P_{ET}O_2$ mm.Hg.
C1	3.19	3.36	45.77	99.1
C2	5.01	4.93	54.78	90.1*
C3	4.56	4.18	48.87	95.0
C4	4.29	4.07	48.90	96.2
C5	3.29	2.60	33.46	106.2*
	4.07	3.82	46.36	96.8
S =	0.80	0.89	7.91	2.11
C1	3.83 +	3.11 -	38.85	98.0
C2	4.72 -	4.21 -	47.02	
C3	4.40 -	3.91 -	45.58	96.0
C4	4.03 -	3.50 -	44.91	98.6
C5	2.35 -	2.05 -	32.28	
	3.87	3.36	41.73	97.5
n =				3
S =	0.91	9.84	6.13	1.36
Δ %	-4.9	-12.3	-10.0	+0.7

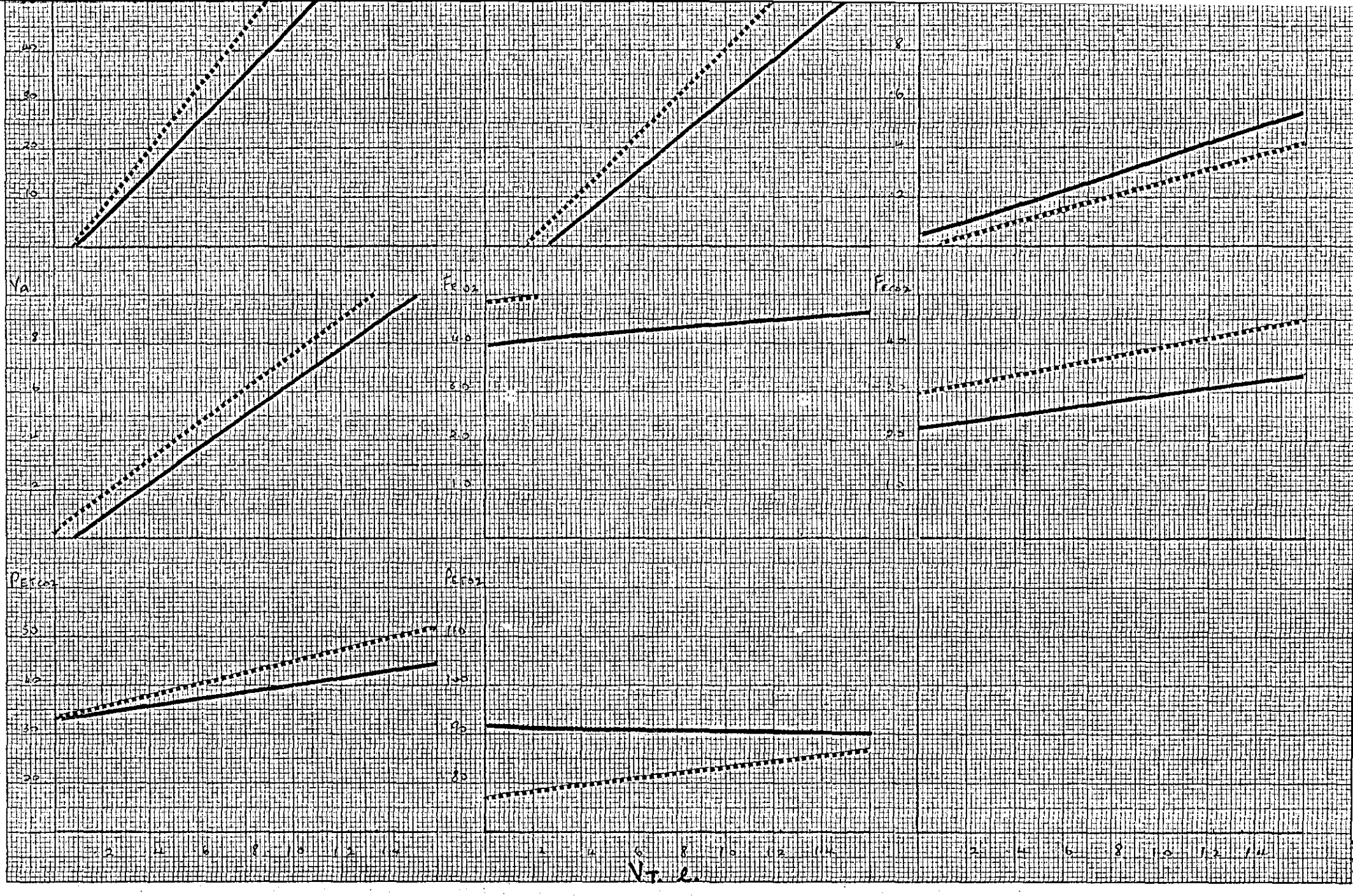
	V_T						V_T					
	V_{O_2}	V_{CO_2}	V_D	V_A	$F_{E_{O_2}}$ (%)	$F_{E_{CO_2}}$ (%)	V_{O_2}	V_{CO_2}	V_D	V_A	$F_{E_{O_2}}$ (%)	$F_{E_{CO_2}}$ (%)
C1	14.37	15.06	197.3	302.7	2.82	2.95	27.37	28.85	291.2	508.8	3.46	3.65
C2	11.05	13.12	181.4	318.6	3.26	3.31	32.40	33.17	224.5	575.5	3.98	3.97
C3	18.93	16.72	178.0	322.0	3.96	3.53	35.39	32.38	226.7	573.3	4.34	3.94
C4	21.19	18.24	166.2	333.8	3.67	3.20	35.00	33.64	225.8	574.2	4.35	4.15
C5		7.88	218.9	281.0	2.69	2.10		19.05	284.3	515.7	3.05	2.40
	16.38	14.24	188.4	311.6	3.28	3.02	32.54	29.42	250.5	549.5	3.84	3.62
S =	4.55	4.02	20.4	20.4	0.54	0.55	3.69	6.09	34.1	34.1	0.57	0.71
C1	16.26 +	13.11 -	175.1 -	324.9 +	3.33 +	2.50 -	32.94 +	26.76 -	241.4 -	558.6 +	4.11 +	3.34 -
C2	17.70 +	16.52 +	208.4 +	266.6 -	2.97 -	2.72 -	35.98 +	32.41 -	222.2 -	577.7 +	3.99 +	3.60 -
C3	20.74 +	17.94 +	156.8 -	343.2 +	4.01 +	3.46 -	36.04 +	32.26 -	209.9 -	595.1 +	4.38 +	3.89 -
C4	18.83 -	16.24 -	194.9 +	305.1 -	3.12 -	2.68 -	33.78 -	29.40 -	256.5 +	543.5 -	4.14 -	3.58 -
C5	9.47 *	7.74 -	267.4 +	235.6 -	1.90 -	1.57 -	18.75 *	16.36 -	369.2 +	430.8 -	2.23 -	1.93 -
	18.38	14.31	200.5	295.1	3.07	2.59	34.68	27.44	259.8	541.1	3.77	3.27
n =	4						4					
S =	1.89	4.07	42.2	43.7	0.76	0.68	1.57	6.61	63.7	64.7	0.87	0.77
Δ %	+12.2	+0.5	+6.4	-5.6	-6.4	-14.2	+6.6	-6.7	+3.7	+1.5	-1.8	-9.7

APPENDIX I

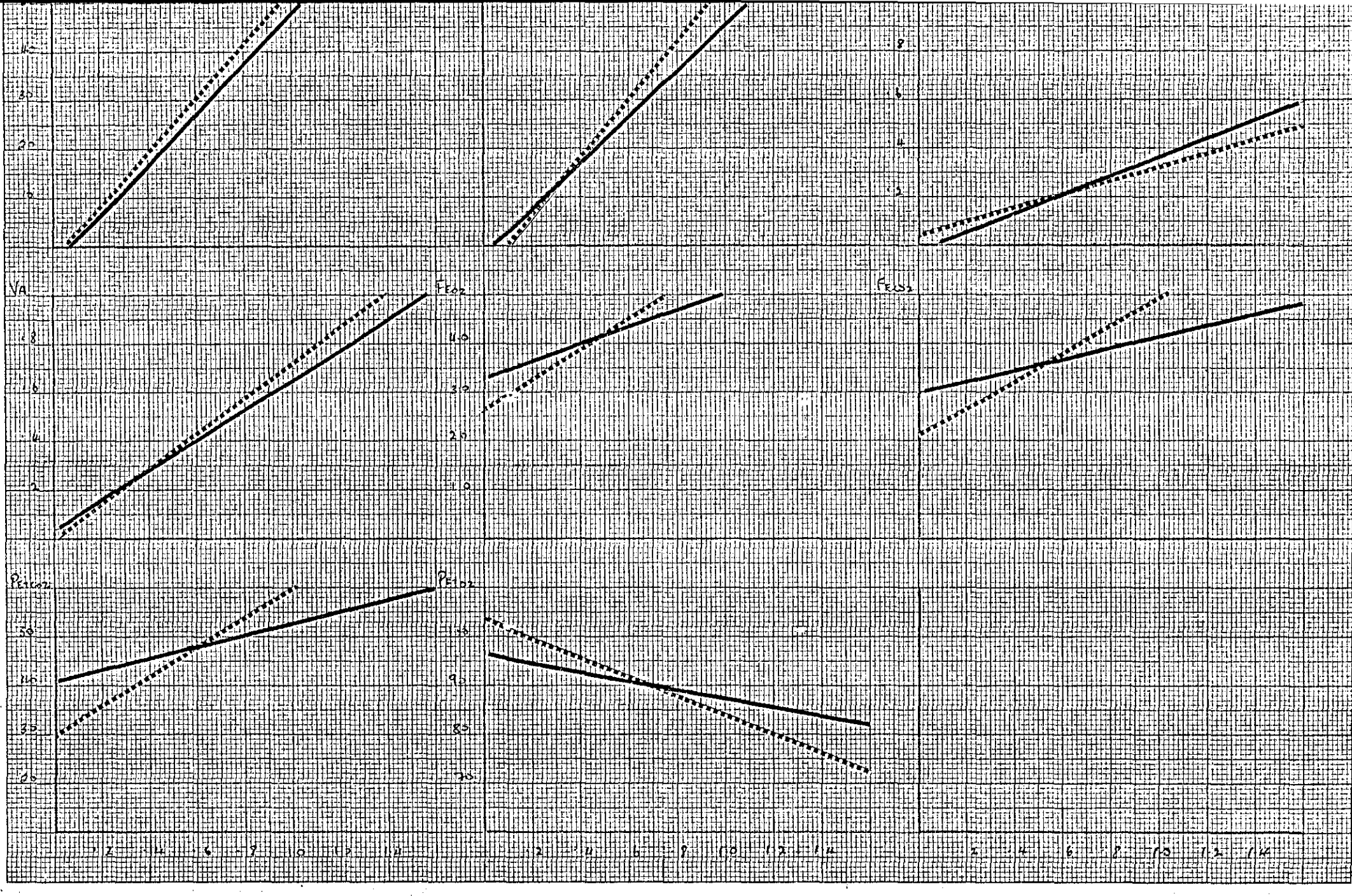
Individual regression line of best fit before and after training.

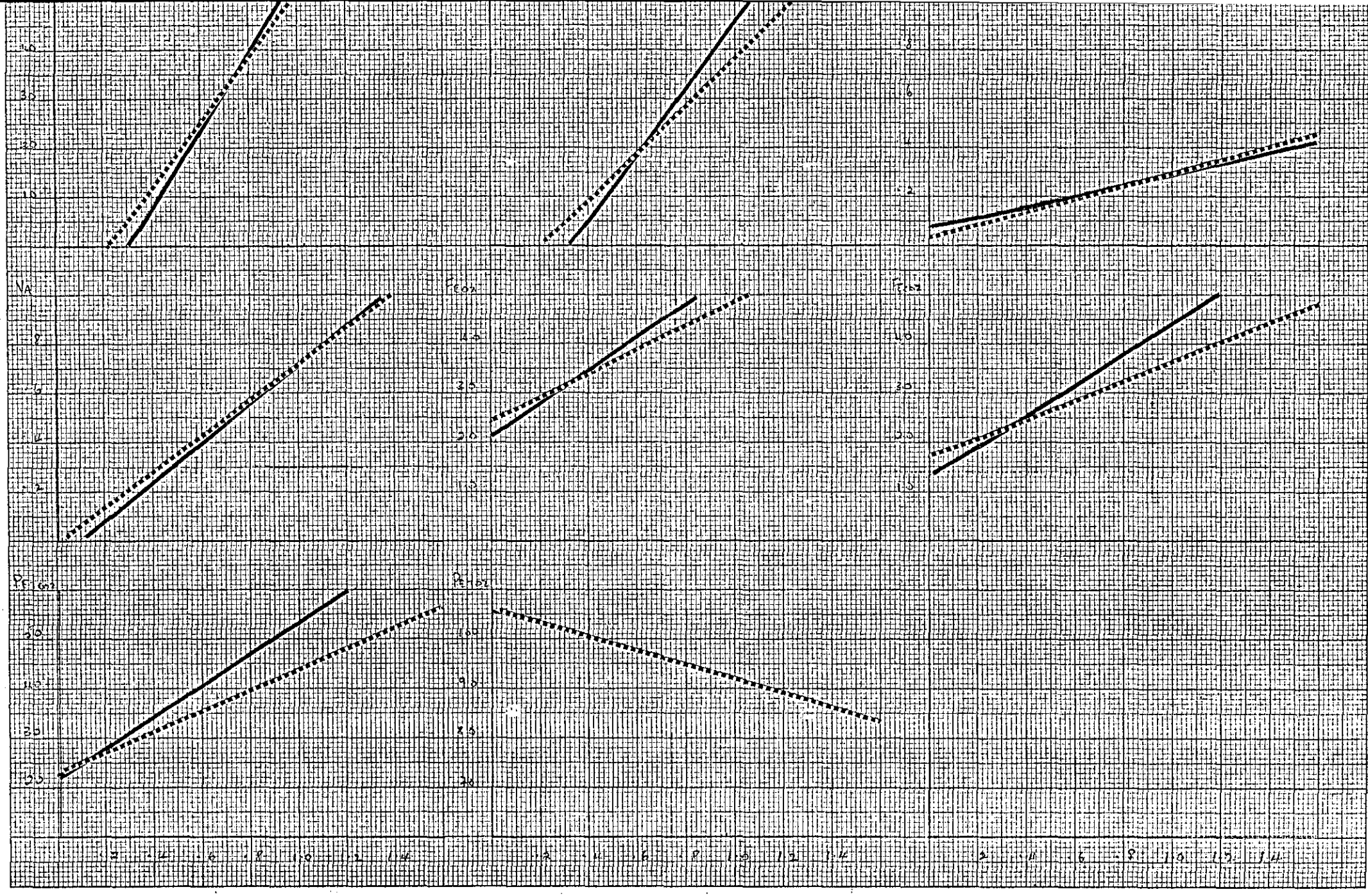
_____ Before.

..... After.

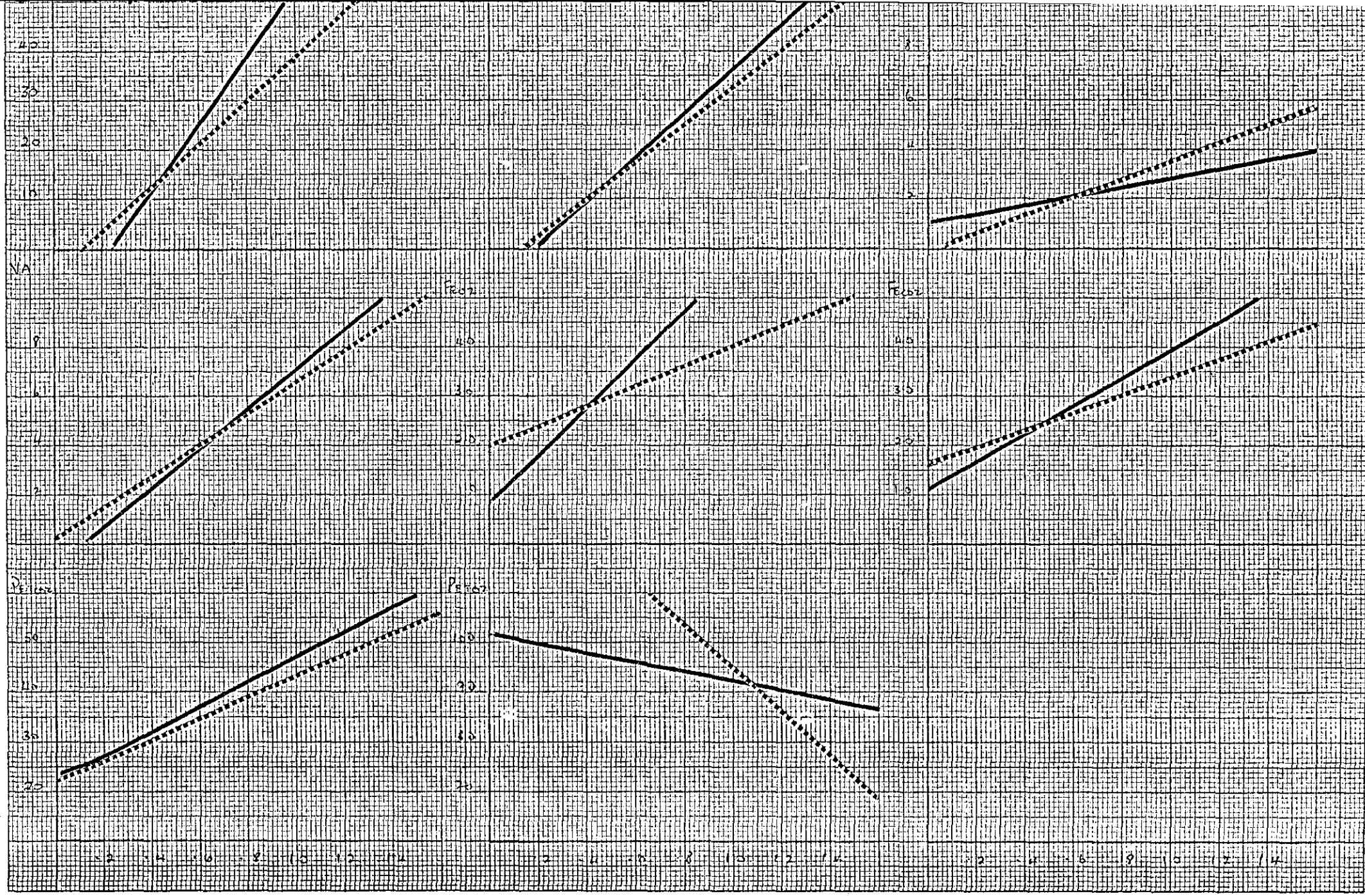


Va
FeCO2
PeCO2
VT. l.

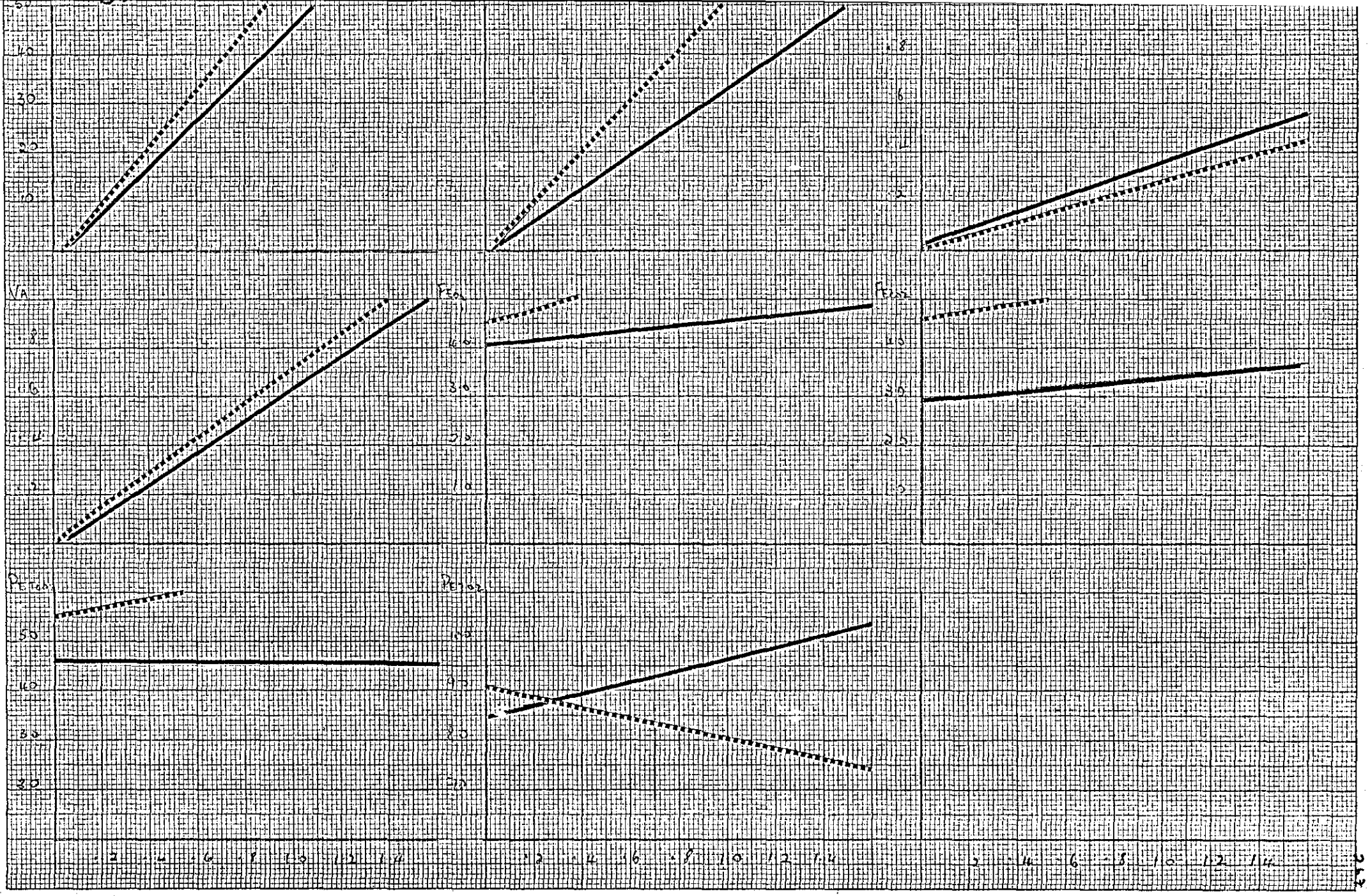

VA
FEO2
FEO2
PETCO2
PETO2

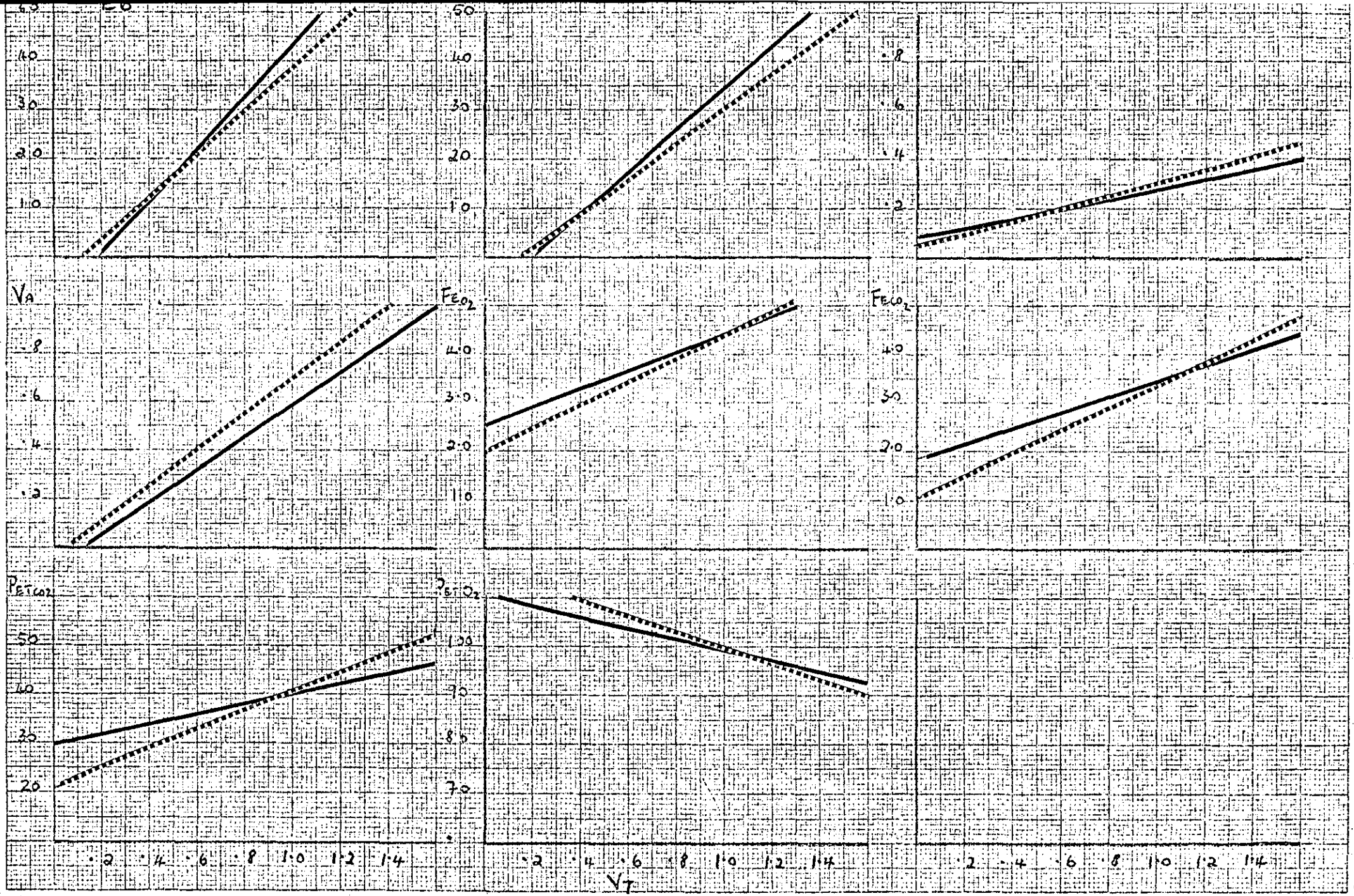


V_A
P_{EO_2}
P_{EO_2}
P_{EO_2}
P_{EO_2}

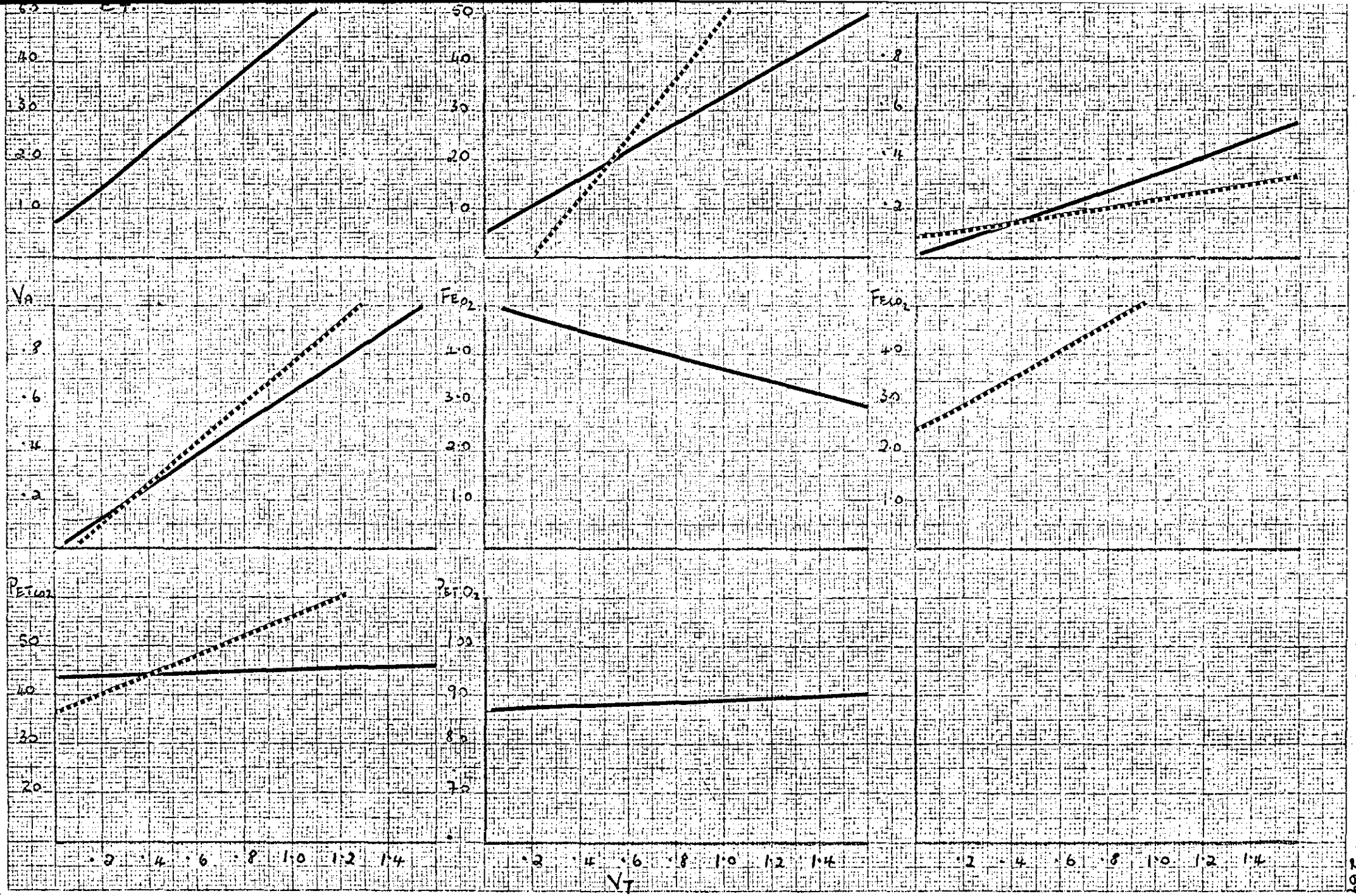


VA
FeO2
FeCO2
PeCO2
PeCO2





V_A
F_{EO_2}
F_{ECO_2}
P_{ETCO_2}
P_{ETO_2}
V_T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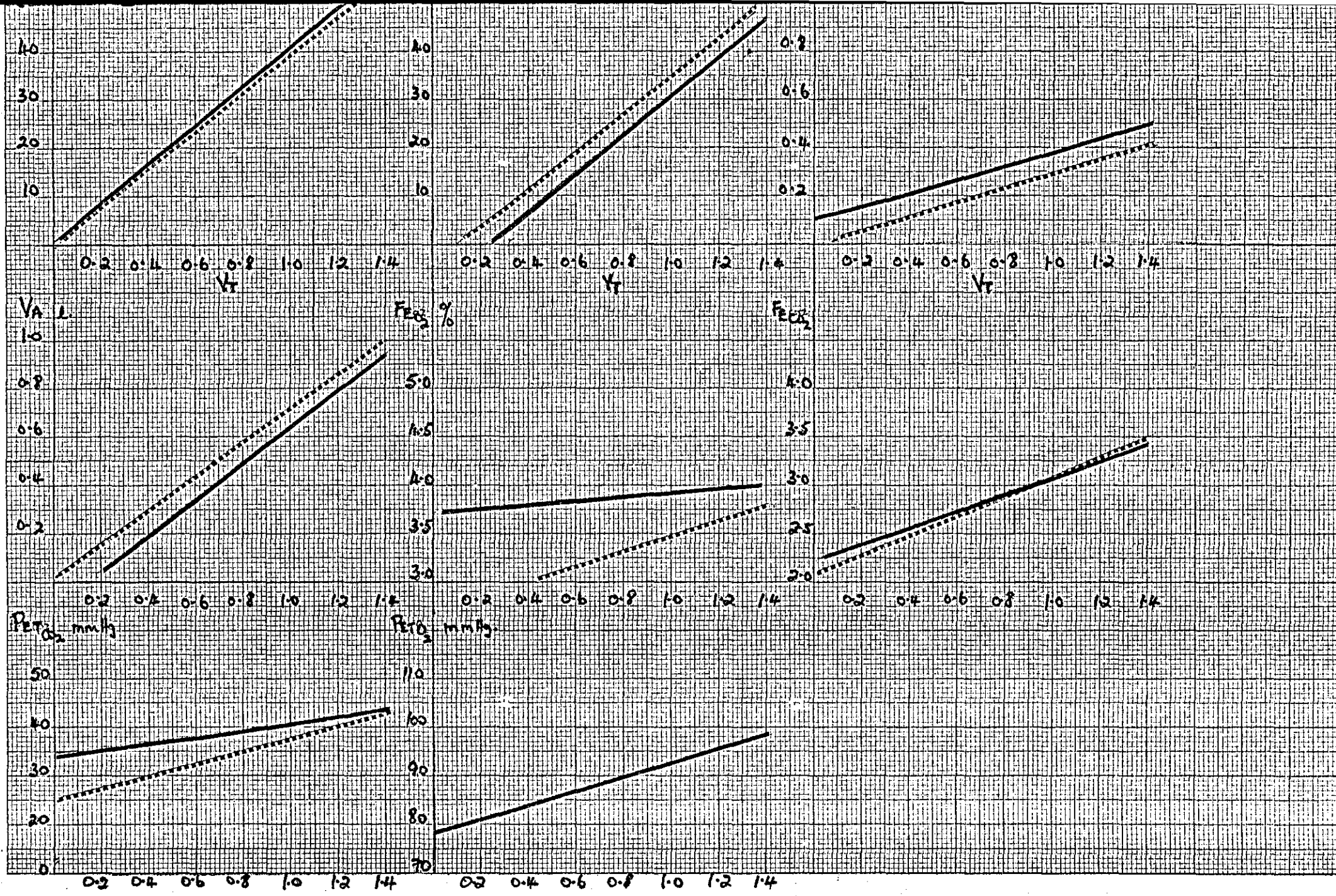
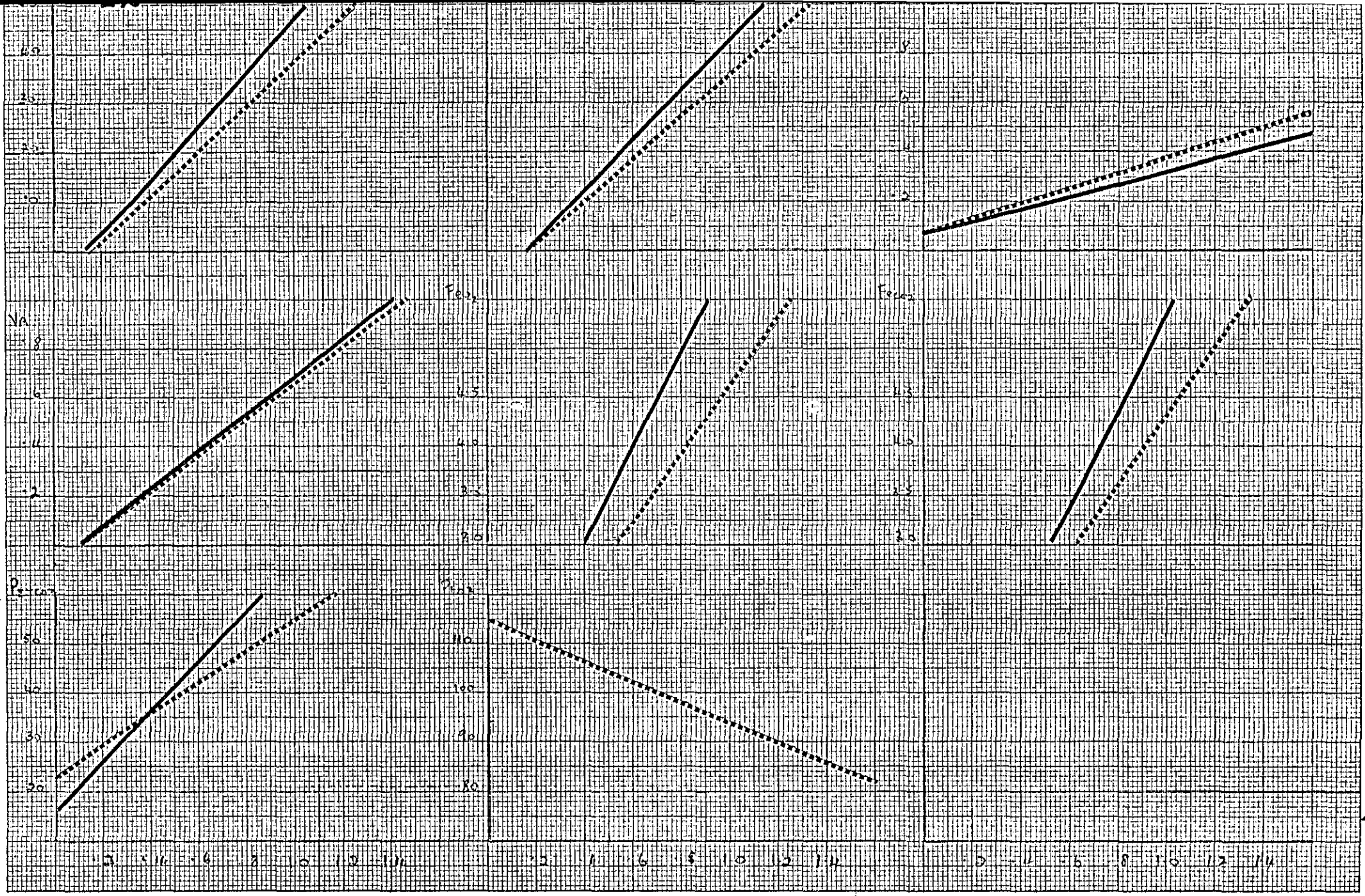
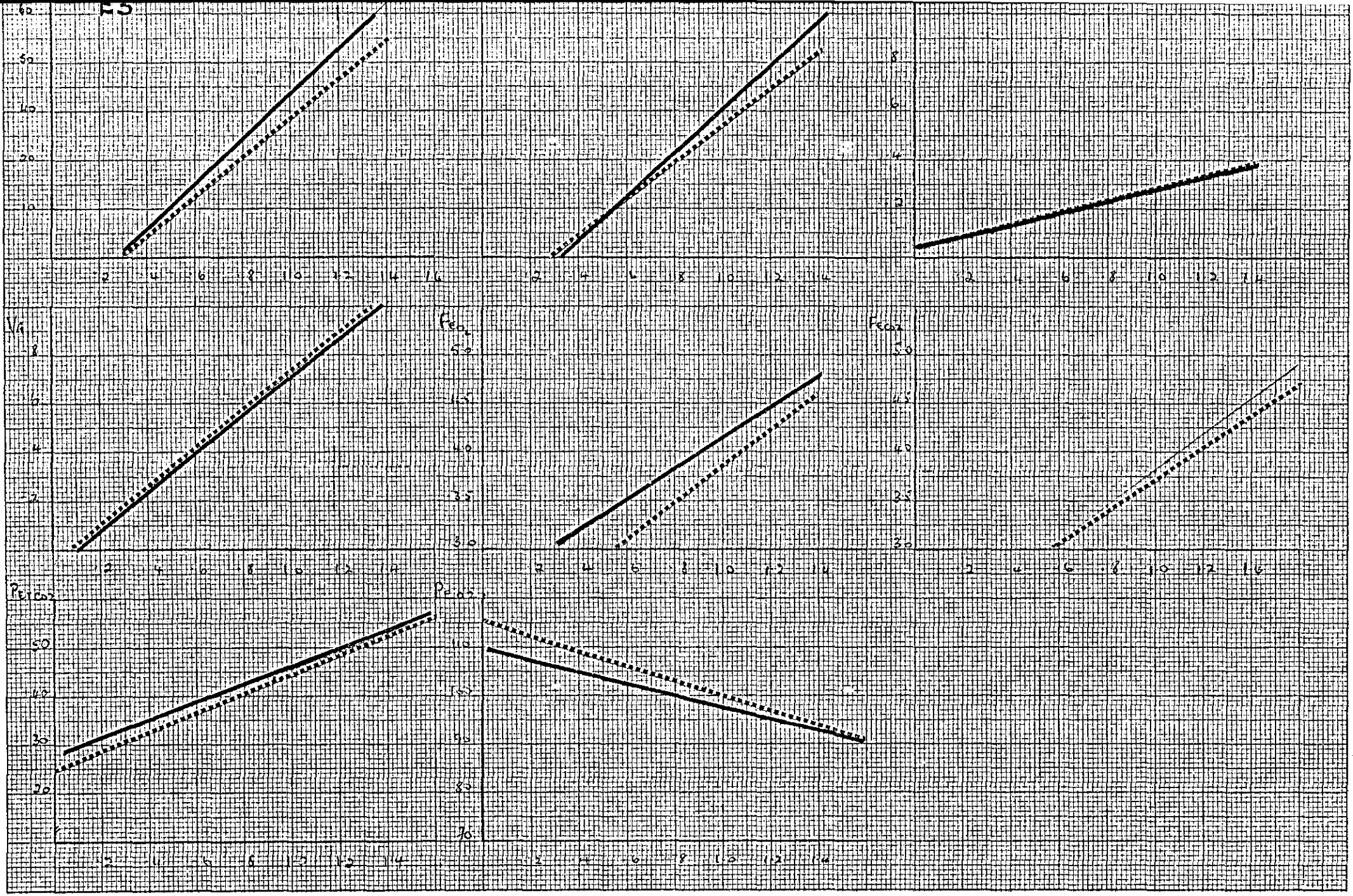
FE_{IO_2}
P_{FEIO_2}
$P_{ET.O_2}$
V_T



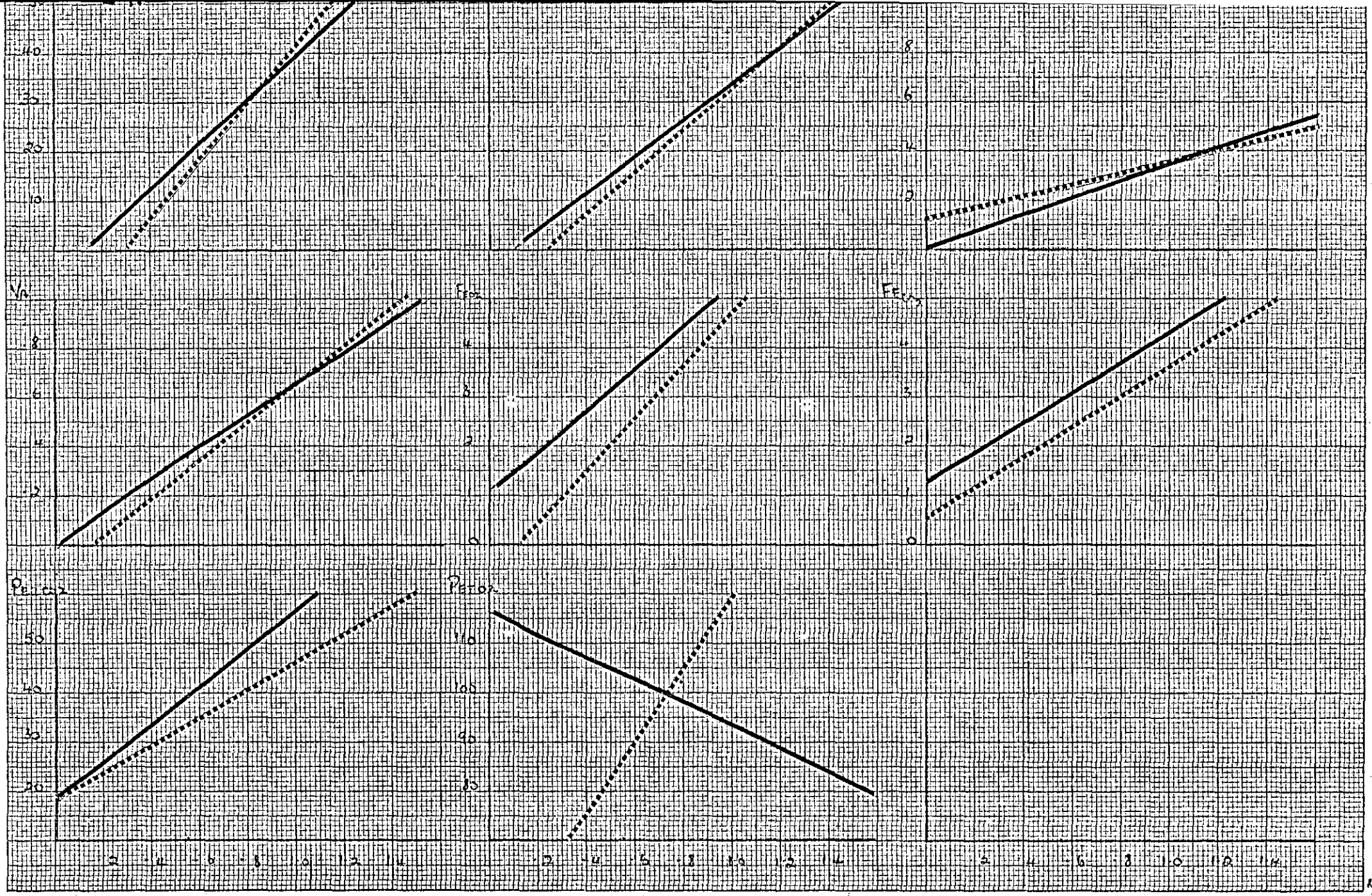
40
30
20
10
0·2 0·4 0·6 0·8 1·0 1·2 1·4
V_T
40
30
20
10
0·2 0·4 0·6 0·8 1·0 1·2 1·4
V_T
0·8
0·6
0·4
0·2
0·2 0·4 0·6 0·8 1·0 1·2 1·4
V_T
V_A l.
1·0
0·8
0·6
0·4
0·2
0·2 0·4 0·6 0·8 1·0 1·2 1·4
$F_{E\,CO_2}$ %
5·0
4·5
4·0
3·5
3·0
0·2 0·4 0·6 0·8 1·0 1·2 1·4
$F_{E\,CO_2}$
4·0
3·5
3·0
2·5
2·0
0·2 0·4 0·6 0·8 1·0 1·2 1·4
$P_{ET\,CO_2}$ mmHg
50
40
30
20
0
0·2 0·4 0·6 0·8 1·0 1·2 1·4
$P_{ET\,O_2}$ mmHg.
110
100
90
80
70
0·2 0·4 0·6 0·8 1·0 1·2 1·4



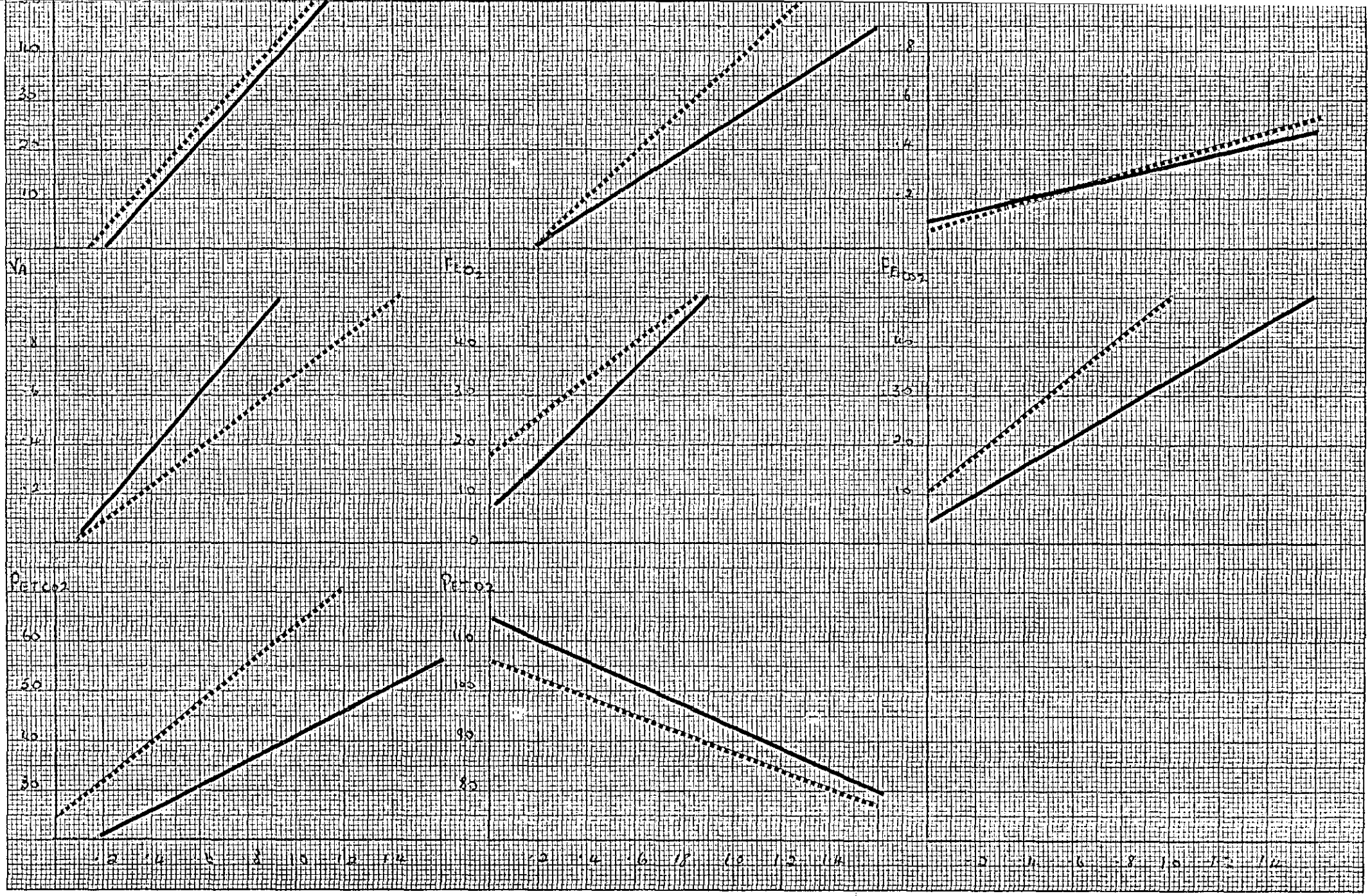
Na
Fe
Fe
Pe
Pe

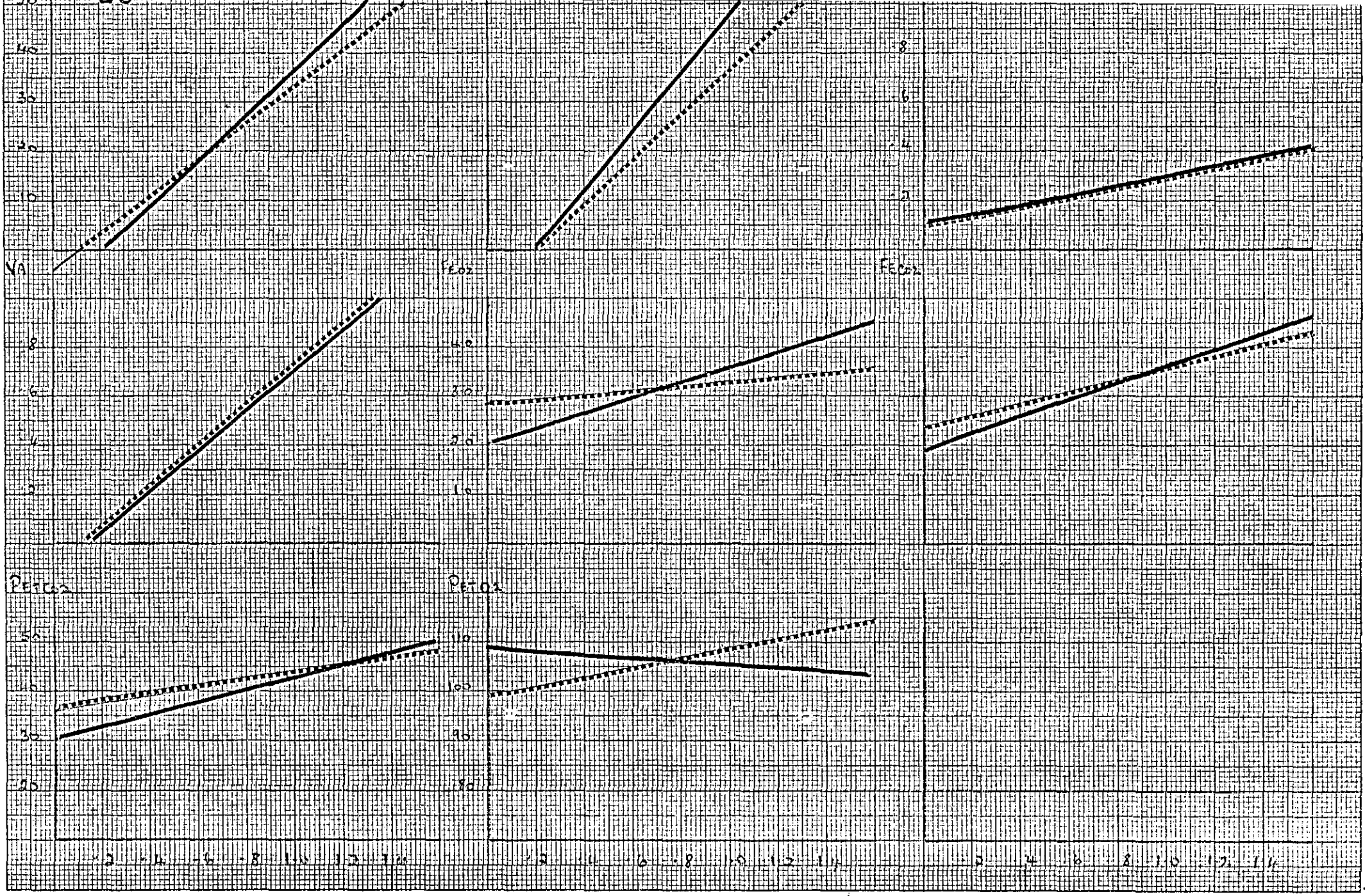


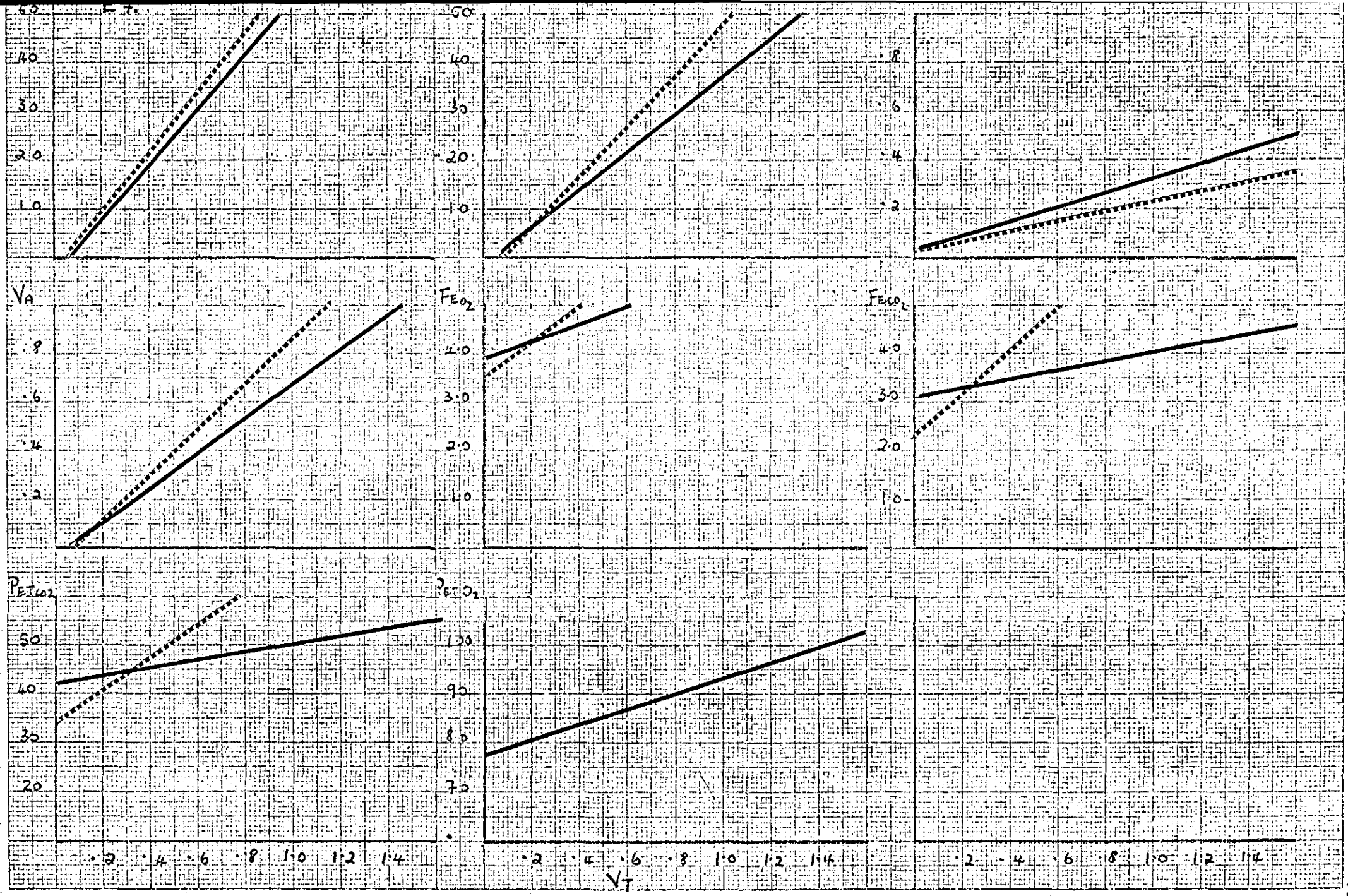
Va
FeO2
FeCO2
PeTCO2
PeO2

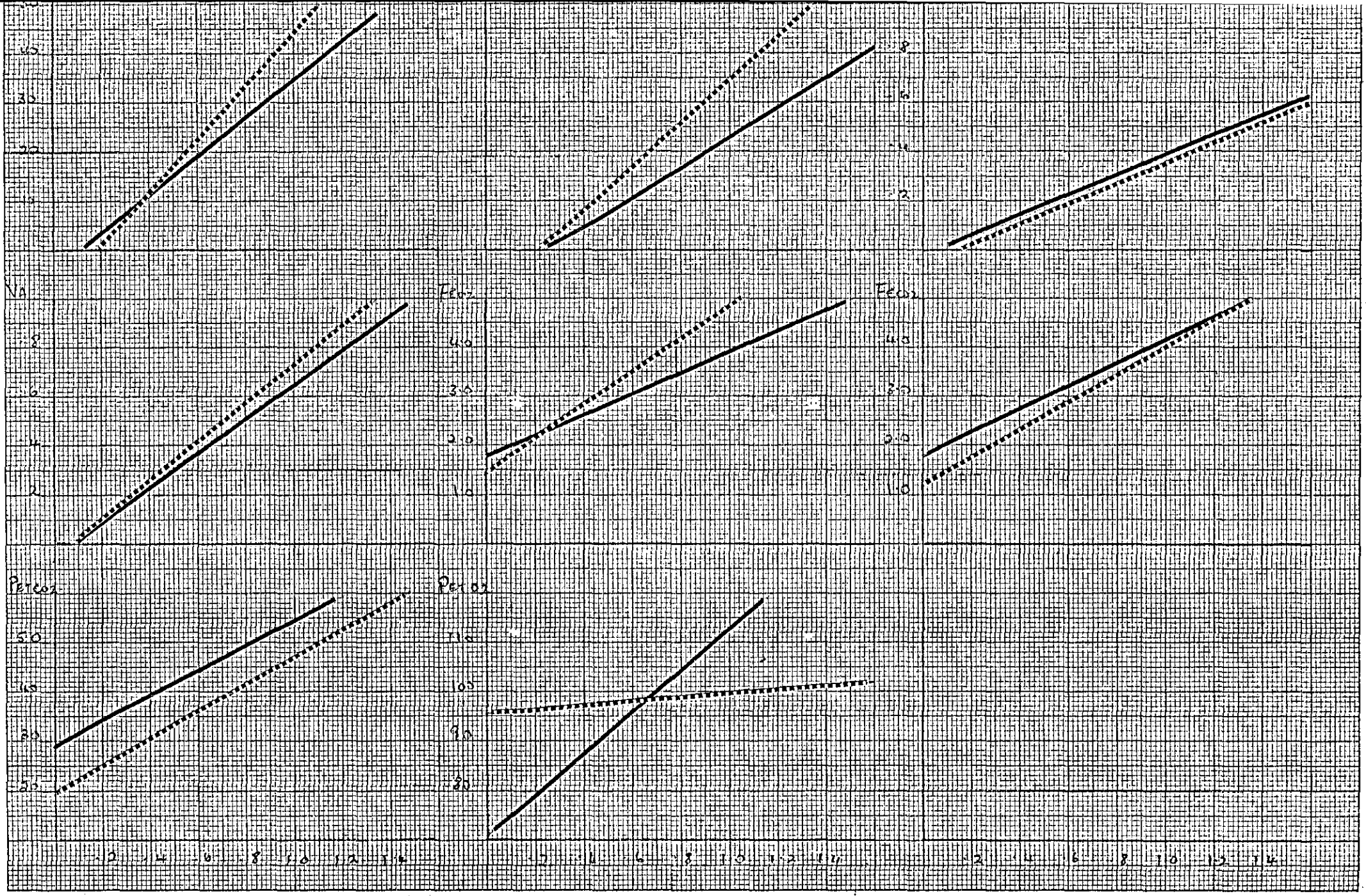


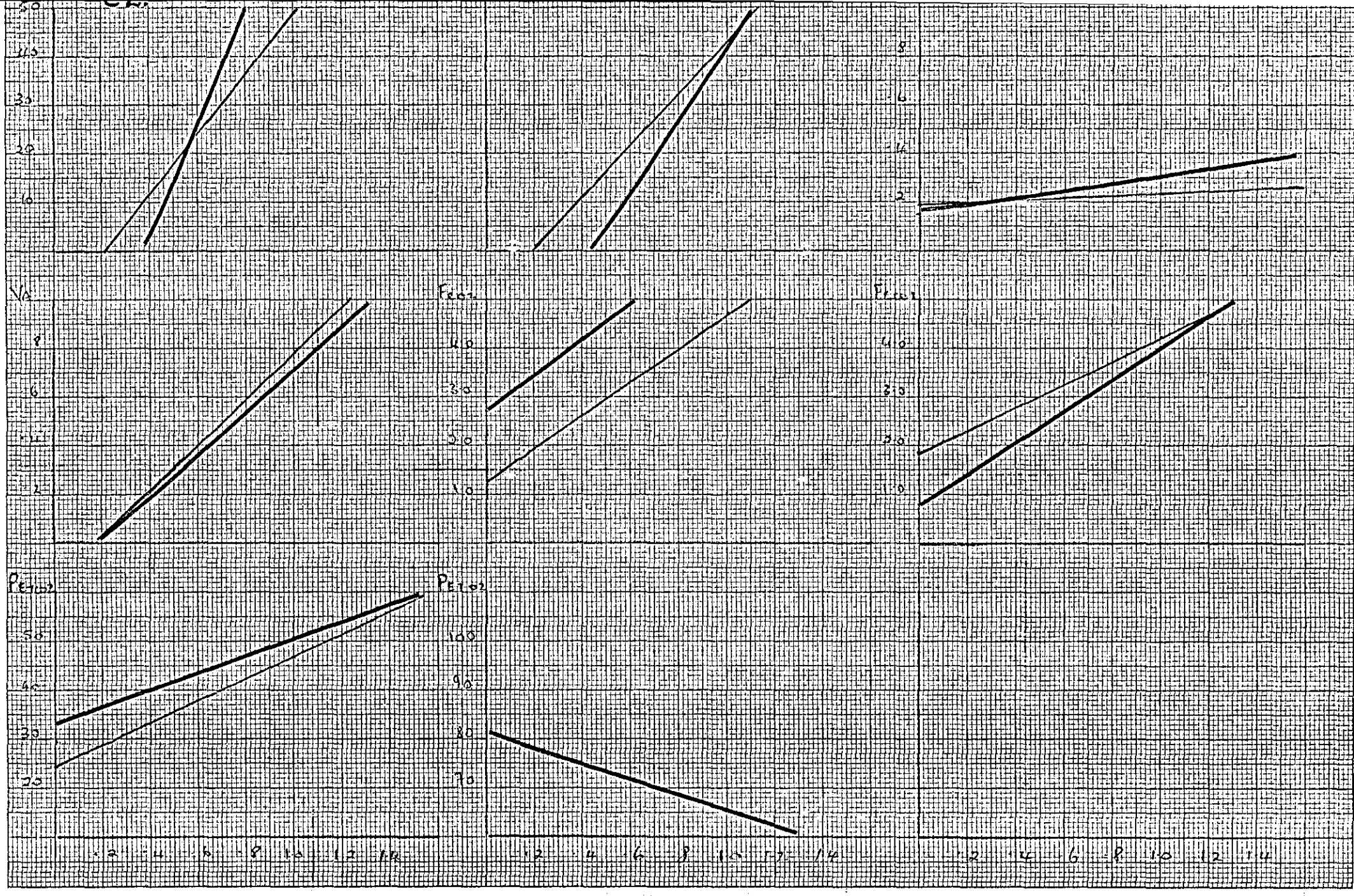
40
30
20
10
8
6
4
2
Va
.8
.6
.4
.2
Feo
4
3
2
1
0
Feo
4
3
2
1
0
Pe.coz
50
40
30
20
Peto
110
100
90
80
.2
.4
.6
.8
1.0
1.2
1.4


VA
PETO2
PETCO2
PETO2
PETCO2

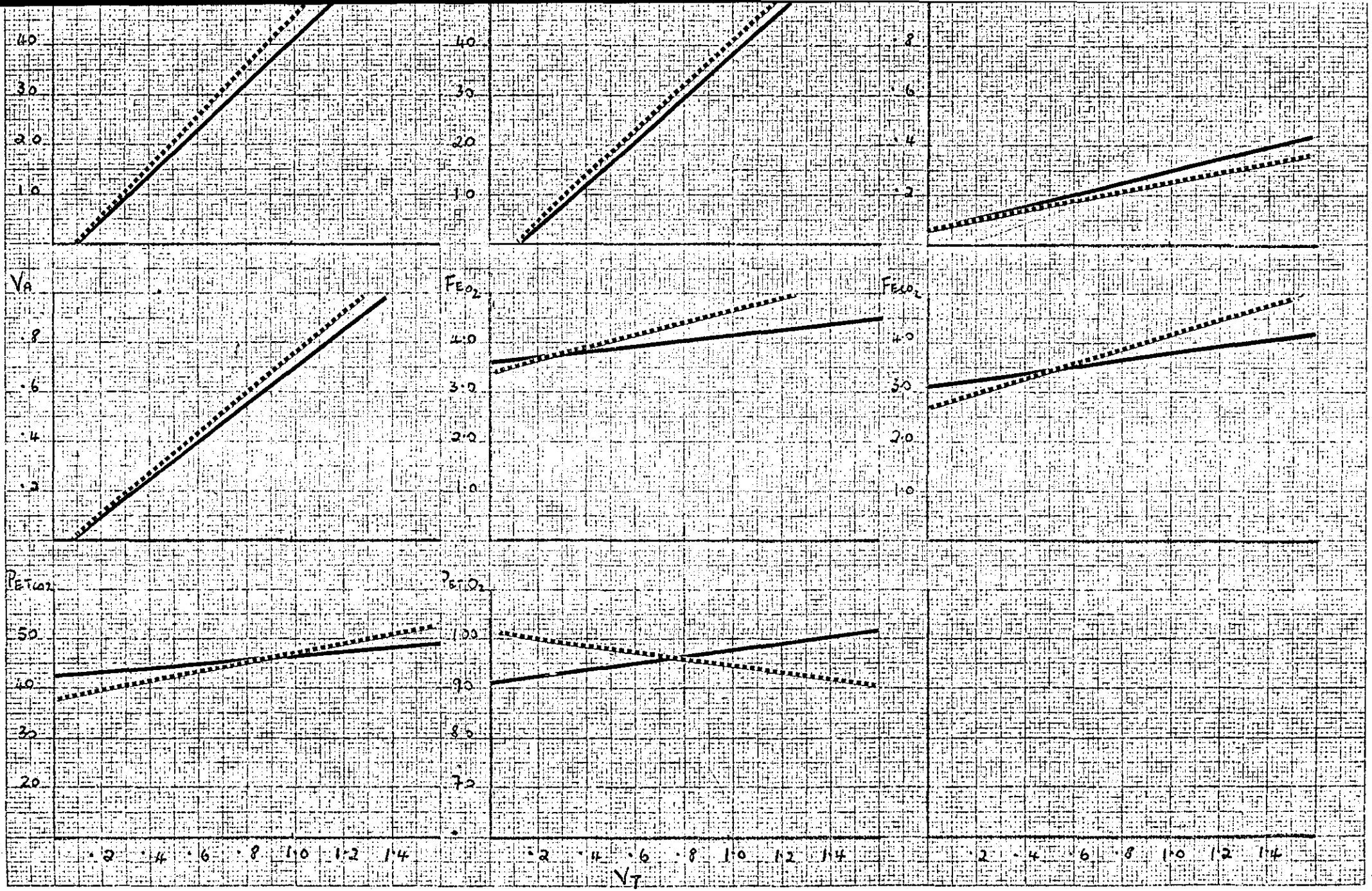

VA
FEO2
FECO2
PETCO2
PETO2

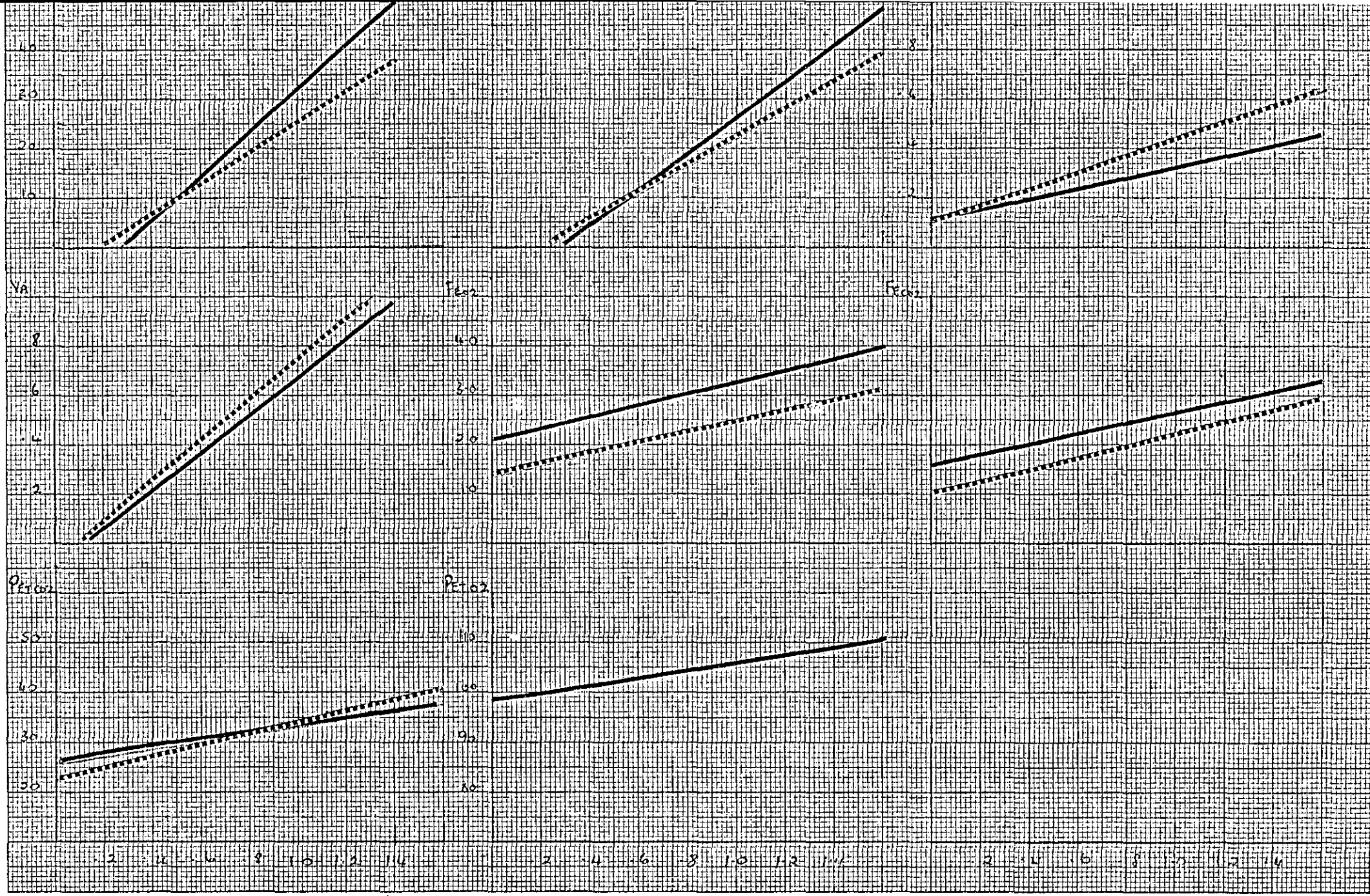

V_A
$F_{E_{O_2}}$
$F_{E_{CO_2}}$
$P_{ET_{O_2}}$
$P_{ET_{CO_2}}$
V_T


VA
Fe_2O_3
Fe_2O_3
P_2O_5
Period
Period



278


40
30
20
10
40
30
20
10
.8
.6
.4
.2
V_A
FE_{O_2}
FE_{CO_2}
.8
.6
.4
.2
4·0
3·0
2·0
1·0
4·0
3·0
2·0
1·0
$P_{ET}O_2$
$P_{ET}O_2$
50
40
30
20
100
90
80
70
·2 ·4 ·6 ·8 1·0 1·2 1·4
·2 ·4 ·6 ·8 1·0 1·2 1·4
2 ·4 ·6 ·8 1·0 1·2 1·4
V_T


VA
$P_{ET\,CO_2}$
$P_{ET\,CO_2}$
$P_{ET\,CO_2}$
$P_{ET\,CO_2}$