RELATIONSHIP BETWEEN TRAINING HEART RATE AND AEROBIC THRESHOLD IN EXERCISING CARDIAC PATIENTS

by

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Abstract

The purpose of this study was to examine the relationship between training heart rate (THR) and the HR occurring at the Aerobic Threshold (AerTHR), and to examine the AerT as an index of training intensity in selected coronary artery disease (CAD), post-myocardial infarction (MI), and post-coronary artery bypass surgery (CABS) patients. Twenty male subjects (age=54.9; wt=73.7 kg; %body fat=25.8) were recruited on the basis of regular participation in a cardiac rehabilitation program (CRP) (3/week at 70 - 85% HRmax) for 6 months; no beta- adrenergic medication; and symptom-free during exercise. Field measurements of THR during the aerobic phase at CRP was carried out by computer-assisted portable telemetry with mean THR computed from each 30 minute value per subject. A maximal treadmill test starting at 2.5 mph at 0% grade with speed increasing 0.5 mph each minute was carried out using a Beckman MMC for 30 second determinations of respiratory gas values. The AerT was determined by visual inspection of the first departure from linearity of Ve and excess CO2 . VO2max was 35.6 ±5.6 $ml/kg/min^{-1}$, with HRmax 166.2 ±11.8 bpm. Paired t-tests were performed; AerTHR was 124.8 ±15.3 bpm with THR 133.7 ±13.4 bpm ($p < .03$). Percent HRmaxAerT was 75.1 ±8.05 and %HRmaxTHR was80.6 ±8.3 ($p < .03$). Mean %VO2maxAerT (54.4 ±6.7) is consistent with other reported data showing lower values in less trained individuals. Stepwise correlations were performed, and a regression equation was produced to predict AerT grom HRmax, height, and weight with a multiple $r = .74$ ($p < .01$). These

data suggest that in this population, THR, as calculated by the relative percentage of maximum method, produces training intensities above the AerT expressed as absolute or relative percents of HRmax. This finding may have implications for optimal body fat reductions, patient compliance to the exercise program, and safety in CRP's.

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I. INTRODUCTION

The influence of aerobic exercise programs on the tertiary prevention and rehabilitation of patients with coronary artery disease (CAD) and post-myocardial infarction (MI) patients received considerable attention over the last decade (Kavanagh et al., 1973; Wilson et al., 1981). Several studies have offered insights into the central and peripheral physiological adaptations which occur in the CAD patient with training (Barnard et al., 1977; Clausen et al., 1970; Sim et al., 1974). Recently, however, researchers have begun to question the accuracy and validity of previously accepted methods of prescribing indecies of exercise intensity for these patients. For CAD patients in cardiac rehabilitation programs (CRP), prescribing exercise intensity involves complex interrelationships among variables not evident in a healthy population; these include ischemic symptoms, decreased stroke volume with a reduction in myocardial contractility, beta-adrenergic medications, musculoskeletal limitations, and psychological problems (Kavanagh et al, 1973; Wilson, 1975; Wilson et al., 1981). Determining the correct exercise intensity for CAD patients is thus necessary if the patient is to achieve full benefits of aerobic exercise programs with minimal risk.

In determining the exercise prescription, most CRPs utilize a combination of Karvonen's (1957) formula, relative percent values of 70-85 % of maximal heart rate (HRmax) and 57-78 % of maximal oxygen consumption (V02max), or 60-70 % of maximum metabolic equivalents (METS) (American College of Sports Medicine, 1980; Fox et al., 1972; Wilson, 1975). These values are derived on the basis of maximal performances on graded exercise treadmill or bicycle ergometer tests (GXT). The types and description of these protocols are described elsewhere (Bruce, 1971; Ellestad, 1975); most exercise prescriptions, however, are calculated from observed maximal HR for age and sex. Froelicher et al (1975) found that the method of predicting V02max from age and maximal treadmill time for the Bruce and Balke protocols was inadequate. Other studies have demonstrated errors in assuming age-predicted maximal HR in CAD patients, (Poweles et al., 1979), and errors in the Bruce protocol in predicting functional capacity (Sullivan et al., 1982).

Smith et al., (1982) found that the Karvonen equation failed to accurately estimate training HR (THR) in CAD patients, while Katch et al., (1978) demonstrated discrepancies in the use of the "relative percent" concept for determination of training intensities. Wilmore et al., (1981) and others (Dressendorfer et al., 1981; Dwyer et al., 1981; Katch, 1978) have recommended the use of the anaerobic threshold (AT) as a more accurate basis for exercise prescription intensity. The AT, as originally defined by Wasserman et al.(1964) identifies a fall in blood pH and bicarbonate (HC03) with non-linear increases in the volume of carbon dioxide (VC02), minute ventilation (Ve) and the respiratory exchange ratio (R) with respect to HR and oxygen consumption, which increases linearly. These breakaway points observed during a GXT indicate a point of transition from predominately aerobic, to more anaerobic pathways in the working muscles.

Weltman et al., (1976) demonstrated that the AT could be utilized as a criterion of submaximal fitness evaluation. Other more recent workers have re-examined the AT and have subsequently suggested alternate terminologies, where the aerobic threshold (AerT) is equivalent to the AT, and the anaerobic Threshold (AnT) describes a second break point, occurring at higher intensities, recruiting more fast oxidative-glycolytic and fast-glycolytic muscle fibers, and resulting in higher blood lactate values (Kinderman et al., 1979; Skinner et al., 1981). For the purpose of this thesis the AerT will be considered synonymous with the AT as defined by Wasserman (et al., 1973; 1975).

McLellan and Skinner (1981), using these criteria found significantly greater improvements in V02max in a group using AerT rather than % VO2max as an index of training intensity. Wilmore (et al., 1981) suggests that patients who participate in CRPs could be exercising significantly below or above their AerT, independent of a "correct" exercise prescription based on a previously determined "relative percent" of HRmax or V02max. While differences between individuals whose HRmax differs is accounted for by the relative percentage of HRmax method, differences between individuals at submaximal work loads is not taken into consideration. Also, there are significant differences between subjects in related stress and training effects if subjects work at the same assigned relative percent of HRmax (Katch, 1978; Powles et al., 1974). It has been suggested that CAD patients should most optimally train at a HR just below the AerT (Wilmore et al., 1981). This would theoretically prevent metabolic acidosis during exercise due to a larger dependence on oxidative rather than glycolytic mechanisms. In the CAD and post-myocardial infarction patient, the deconditioned and damaged myocardium cannot maintain a high stroke volume to increase cardiac output during anaerobic work. In addition, a higher heart rate (occurring above the AerT) in conjunction with severe coronary artery occlusion is undesirable and might be potentially dangerous for certain patients during the transition from aerobic to anaerobic states (Wilmore et al., 1981).

Few studies have dealt with the CAD patient in CRPs with regard to training intensities and the AerT. Although Wasserman's paper in 1964 dealt with AerT detection in its relation to cardiac disease, it is unclear to what extent he was referring to CAD or valvular and congenital diseases. Since intensity is still the most crutial but least understood variable in the exercise prescription with CAD patients in CRPs, the metabolic demands of the exercise prescription will be examined with regard to the AerT.

Hence, the purpose of this study is to determine the relationship between previously prescribed THRs for exercising CAD patients and the AerT. In addition, this study will examine the possibility of using the AerT, HR, and other variables to construct regression equations that can be utilized for precise and safe exercise prescriptions in addition to standard methods of prescribing exercise intensity. It is hoped that these equations will maximize the conditioning effects for the CAD, post surgery, or post-MI patient, without overlooking the need to prevent training from occurring at or above the AerT in CAD and Post-MI patients.

II. METHODS

Twenty male subjects (age 41-63), were recruited voluntarily from local CRPs in the Greater Vancouver area. All subjects had either documented CAD affecting at least one vessel as determined by angiography, one or more MI's as documented by ECG and enzyme changes, or a history of coronary artery bypass surgery (CABS). A description of the subjects is summarized in Table 1. Subjects were made aware of the potential risks involved, and informed consent was obtained (Appendix I). Patients receiving beta-adrenergic blocking medication or with medical contraindications such as pulmonary disease, hypertension, unstable angina, congestive heart failure, or history of ectopic ventricular arrhythmias were not included in the study. All patients were exercising regularly 3 times per week for 6 months to 4 years during the course of the study.

Data collection consisted of two phases. Subjects were initially investigated in the field, during attendance at a CRP, and were individually instructed to carry on with their prescribed walk/jog training session, at the usual intensity or THR while being continually telemetered by a Burdick portable Cardiodyne telemeter during the aerobic phase. A CM5 lead configuration was utilized, and ECG recordings were obtained for 30 minutes on micro cassette tapes. The tape cassettes were replayed into an Avionics 4000 Cardiogard interfaced with a Hewlett-Packard 3052A Data Acquisition System, where HR values at 15, 30, 45 and 60 seconds of each minute, plus the mean HR for each minute were automatically computed and recorded.

Electrocardiogram recordings were simultaneously obtained at intervals of 2 minutes for reliability purposes. The first 5 minutes and last 5 minutes of the recordings were omitted to allow for normal attainment of the steady-state THR and cool-down period, respectively. Mean THR was obtained by averaging all 20 mean minute values, with any signal artifact values being omitted from the calculation (see samples in Appendix D and E). Subjects were told prior to the recording session that the investigation was to observe heart rhythm, so as not to produce anxiety and influence or bias exercise performance. All subjects' THRs had been previously determined by assigning an average of 75% of symptom limited HRmax on the basis of the last previous Bruce GXT. The telemetry procedure was repeated several times to ensure the reliability of the data, with the additional use of a Resperonics Exersentry heart rate monitor to validate THR. Computed THR was also compared to THR as reported by individual subjects in the regular CRP daily exercise log sheets (palpatation of radial pulse; counting for 10 seconds and multiplying by 6).

The second phase of data collection occurred in the J.F. Buchannan Research and Fitness Centre at the University of British Columbia (see Appendix J). Measurement of body weight, height and estimation of percent body fat with skinfold measurements (Yuhasz, 1978) was followed by a maximum treadmill test (MTT) to determine V02max, HRmax and the AerT. The MTT protocol was performed on the treadmill and was consistent with Wasserman's (et al., 1964) method for determination of the AerT, utilizing 1-minute work increments. Treadmill starting speed was 2.5 mph at 0 % grade (including a 4 minute warm up at the same speed and grade), and increased 0.5 mph every minute until termination. Subjects exercised to maximum with the major consideration of termination being fatigue.

Heart rates were recorded by direct ECG, utilizing a CM5 lead configuration and Avionics 4000 Cardiogard with oscilliscope and ST-segment shift display. Expired gases were continually sampled and analyzed by a Beckman Metabolic Measurement Cart interfaced into a Hewlett-Packard 3052A Data Acquisition system for 15-second determinations of respiratory gas exchange values (see Appendix F,G and H). The velocity of the treadmill at the onset of anaerobic metabolism (Vtam), the percent of maximal oxygen uptake (%Vo2max) and the percent of HRmax (%HRmax) at the AerT were calculated. The AerT was determined by visual examination of first deviation from linearity of the excess C02 curve according to methods described by Volkov et al. (1975) and the Ve curve, as described by Wasserman (et al. 1964, 1973, 1975). Computer-generated curves were examined individually by three investigators. AerT was defined as the Vtam, HR, and corresponding %V02max and %HRmax occurring immediately below these non-linear changes in excess CO2 and Ve.

AerTs were determined for individual subjects. V02max, HRmax, the HR at the AerT (AerT-HR), Vtam, %HRmax at the AerT (%HRmax AerT), %HR max at the AerT (%HRmax AerT), and the difference between THR and the AerT-HR were calculated and recorded.

Paired t-tests was utilized to determine any differences between mean THR and mean AerT-HR, and between %HRmax of THR (%HR-THR) and %HRmax of AerT-HR (%HR-AerT-HR). BMDP P3D program at the University of British Columbia's Computer Science Department for correlated t-tests was utilized for the analyses. These were tested at the .05 level of significance. If there existed significant differences between subjects Vo2max scores, a t-test on V02max was performed to separate subjects of high and low fitness.

A stepwise regresson analysis was subsequently performed utilizing a BMDP P2R program to observe the relation between the variables, and to derive multiple regression equations from these observed data.Two equations were produced. One predicting THR from AerT data (with related variables omitted) and one predicting AerT-HR with its related variables omitted from the equation. Multiple correlation coefficients were analyzed between the variables for validity of the regression equations.

In order to determine the reliability of the measurement of the AerT in these subjects over time, five subjects were brought back to the laboratory a second time. Subjects were chosen on the basis that no changes in their fitness level and progression of their exercise capacity from the previous treadmill test had occurred. The treadmill protocol and measurements were the same as previously outlined. Paired t-tests were performed on AerT-HR1 vs AerT-HR2 with correlation coefficients compared with those from trial 1 for reliability purposes. These were tested at the .05 level of significance (r = .97). For raw data and results, see Appendix C.

III. RESULTS

Mean age for the group was 54.9 ± 5.51 years height was 174.9 ± 7.64 cm, body weight 73.7 ± 9.79 kg and percent body fat was 25.82 ± 4.36. Nine subjects had recieved coronary graft bypass surgery on at least one vessel, while 13 were post-MI patients andtwo had angiographic evidence of CAD.

The physiological data is summarized in Table 1, and individual subjects' data can be found in Appendix B. VO2max ranged from 27.2 to 52.7 $ml/kg/min^{-1}$ and the mean was 35.57 ± 5.57 $ml/kg/min^{-1}$ for the group. The AerT, as determined by visual inspection of the excess CO2 and Ve curves was recorded at a Vtam of 4.55± 0.64 mph. This was equivellent to 54.45 ± 6.77 percent of VO2 max. HRmax was 166.2 ± 11.88bpm. AerT-HR was found to be 124.85 ± 15.3 bpm, and represented 75.1 ± 8.05 percent of HRmax.

The mean THR when measured in the field setting and checked for accuracy against patients' personal daily exercise log sheets was found to be 133.75 ± 13.42 bpm, and represented 80.65 ± 8.26 percent of HRmax.

Mean THR was found to be significantly greater than mean AerT-HR by 8.9 bpm (133.75±13.42 vs 124 ±15.53bpm) ($p < .0304$). A correlation of .30 was found between THR and AerTHR.

When mean %HRmaxTHR and %HRmaxAerTHR were examined , %HRmaxTHR was found to be significantly greater ($p < .0293$) with values of %80.65 ±8.26 and % %75.1 ±8.05, respectively.

Several correlations were subsequently performed on the data to investigate the relationship between the AerT, THR and other recorded physiological data (see Appendix K for corellation matrix). Stepwise regression analysis was performed using first THR as the dependent variable in the first analysis, followed by AerTHR as the dependent variable in the second analysis. Analysis using the dependent variable THR with related variable %HRmaxTHR omitted from the analysis resulted in a negative correlation coeficient of -.11 between VO2max and %VO2maxAerT, while VO2max was highly correlated to Vtam (r = .79). Stepwise regression proceded through 2 steps, terminating after F-levels below 1.5 were attained. Predictors HRmax and age resulted in the following equation for prediction of THR with a standard error of estimate of 12.57:

$$y = 0.434(a) + 0.85(b) + 14.64$$

where (a) is HRmax and (b) is age.

The second stepwise regression equation with AerTHR as the dependent variable, and its related variables omitted (Vtam,%VO2AerT and %HRmaxAerT) was then generated. A moderate correlation of .51 between HRmax and AerTHR was found. The following prediction of AerTHR with the regression equation, utilizing HRmax,weight, and height was produced, yielding a standard error of estimate of 10.91:

$$y = 1.21(a) + 0.68(b) - 1.04(c) - 123.23$$

where (a) is height in cm, (b) is HRmax, and (c) is body weight in kg.

The equation for prediction of AerTHR yielded a multiple correlation of .74, which was statistically significant ($p < .01$). The equation for prediction of THR yielded a multiple correlation of .46 ($p < .05$).

Table I -

Physical Characteristics of Subjects

Subject	Age	Status	Height(cm)	Weight(kg)	%Body Fat
RM	58	MI,CABS	171.6	69.4	29.9
WK	61	MI¤	169.8	81.3	30.3
DG	53	CAD■	170.1	73.7	33.9
BD	57	MI,CABS	174.6	67.2	21.2
JB	63	CAD	166.2	62.1	20.0
PH	52	CABS°	167.4	62.4	20.9
PM	57	MI	167.1	66.6	23.4
LC	55	MI,CABS	182.2	77.9	20.8
RS	53	MI	198.6	102.3	26.2
JD	51	CABS	173.6	72.4	26.7
PD	49	CABS	182.4	78.9	26.9
NK	61	CABS	168.1	74.9	29.5
JH	63	CABS	177.2	85.0	30.0
BS	59	MI	173.5	63.7	23.7
MS	52	MI,CABS	177.7	72.8	26.1
VM	58	MI	173.5	68.6	32.7
FD	41	MI	168.2	78.2	19.2
CK	50	MI	175.1	85.1	27.5
TW	56	MI	183.4	64.9	26.0
SG	49	MI	177.7	67.2	21.6
Mean	54.9		174.9	73.7	25.8
SD	5.51		7.64	9.97	4.36

¤ MI - Myocardial Infarction
° CABS- Coronary Graft Bypass Surgery
■ CAD - Coronary Artery Disease

Table II -

Physiological Data

Aerobic Threshold and Relative Percent Heart Rates

	VO2max (ml/kg/min^{-1})	%VO2maxAerT	Vtam (mph)	HRmax (bpm)
Mean	35.57	54.45	4.55	166.2
SD	5.57	6.77	0.64	11.88

	THR (bpm)	AerTHR (bpm)	%HRmaxAerT	%HRmaxTHR
Mean	133.75¤	124.8	75.1	80.65■
SD	13.42	15.3	8.05	8.26

¤ significantly greater than AerTHR ($p < .03$)

■ significantly greater than %HRmaxAert ($p < .03$)

IV. DISCUSSION

The subjects examined in this investigation represents a homogeneous sample, as indicated by the relatively small standard deviations for variables such as height, weight, age, and percent body fat,and are comparable to variablility found in other studies (Weltman, et al., 1976; Weltman and Katch, 1979). The subjects had been involved in a regular exercise program for at least six months and as the time between the treadmill test and the field evaluation was less than 4 weeks, it is assumed that no significant physiological changes occurred in response to training.

Mean VO2max values of $35ml/kg/min^{-1}$ are consistent with values reported by others for trained CAD or post-MI patients, utilizing a similar aerobic exercise protocol (Kavanagh, et al., 1973; Wilson, et al., 1981). HRmax was within limits of that expected for this age range, although the variability of HRmax observed here agrees with Ryan, et al.(1980) observation of the wide range of HRmax found during maximal GXTs. A low negative correlation of -.20 between age and HRmax found in the present investigation supports this observation.

Mean THR values of 133 bpm, when expressed as a percentage of HRmax was 80.6%. This training intensity is in agreement with others for exercise prescription based on the relative percent method using between 70 and 80% HRmax (Fox et al.,1972; Pollock, 1973; Wilson, 1975; Zohman, et al., 1970).

The mean AerTHR (124 bpm) was significantly lower than mean THR. This was also true when AerTHR was expressed as a percentage of HRmax. This finding is in agreement with Katch et al. (1978), and Dressendorfer, et al. (1981), who state that the relative percent method of prescribing THR does not take into account individual metabolic differences at submaximal workloads.These data indicate that although these subjects are within the 70 - 85% of HRmax zone for training using the popular relative percent method, in fact, thy are exercising above their AerT values as a group. This also comfirms Wilmore's (1981) speculation that in some cases, cardiac patients, when exercising at the prescribed relative percent intensity of HRmax could in fact be significantly above their AerT, and hence exercising more anaerobically than is desired for this population. A low correlation of .30 between THR and AerTHR and .16 for %HRmaxAerTHR and %HRmaxTHR shows a relative lack of relationship between these variables. This dissagrees with the data of Parkhouse and McKenzie, (1982) and Patton et al. (1979) that HR is a good predictor of AerT. This finding, however is consistant with data reported by Wasserman and McIlroy (1964) and more recently Dressendorfer et al. (1981) that HR is a poor predictor of AerT in middle aged males, and as suggested in this investigation, in trained cardiac patients as well. The AerT occurred at 54.4% VO2max in this study, and the individual range in AerT values (42.1 - 72.7 %VO2max) is in agreement with data reported by Davis et al.(1976), McLellan and Skinner (1981) and Weltman and Katch, (1979) who found values ranging from 41 -71 %VO2max. The literature however reports AerT as a percentage of VO2max in populations that are unlike the present sample.

Patton, et al. (1979) and Parkhouse and McKenzie (1982) examined these variables in young healthy subjects and trained athletes. Wasserman's et al. (1964) cardiac subjects were younger, were untrained , and had valvular and congenital diseases. Nevertheless, Wyndham et al., in 1965 found AerT to occur at 45 -50% in patients with cardiomyopathy, with AerT at 50 to 60% in normal middle-aged males. Davis et al. (1979) and Dressendorfer et al. (1981) both utilized middle-aged males in their studies with similar AerT values. Our subjects, although CAD, CABS and post-MI patients, seem to resemble normal or slightly trained middle aged males as far as metabolic performance on a MTT when comparing their AerT to other populations. However, their differences in terms of cardiac disease and the subsequent functional impairment make comparison to other groups unacceptable in this respect.

Stepwise regression analysis produced correlations which reflected the differences between the relative percent concept and the AerT method of exercise prescription. A low negative correlation of -.11 between VO2max and %VO2maxAerT is different than that reported in other studies. Weltman et al.(1979) reported correlation of .69 between VO2max and VO2 at the AerT, and Davis, et al. (1976) reported a slightly smaller value (r = .52). In a later study, Weltman and Katch (1979) reported a correlation of .85 between VO2max and VO2 at the AerT. However, the subjects were young males and the protocol was done using a bicyle ergometer. Our results are similar to that found more recently by McLellan and Skinner (1982) who found a highly significant negative correlation of -.64 between VO2max and %VO2maxAerT. Several explanations were offered to account for this reversal of what would normally be expected from the previous literature. As in McLellan and Skinner's study, this negative correlation could have been a function of the wide range of %VO2maxAerT on either end of the continum, coupled with the observation made earlier by Wasserman et al. (1973) that an absolute lower limit of AerT values exists and is equal to about 3.5 mph at a 0%grade or 13 to 14 $ml/kg/min^{-1}$ for a 70 to 75 kg male. Thus individuals who have lower VO2max values would have relatively higher %VO2AerT scores, thus accounting for the low negative relationship found here.

A significant correlation on the other hand, was found between Vtam and VO2max ($r = .79$), agreeing with Weltman and Katch's finding (1979), but Vtam was only moderately correlated with AerTHR ($r = .46$) although significant ($p < .05$), reflecting Katch et al's. (1978) finding that the high correlation could be spurious when the time element is not removed from the analysis.

The regression equations produced in this investigation are specific only to the population studied here. Namely, middle-aged post-MI, CAD, or post-surgery patients not on beta-adrenergic medication.

The prediction of AerTHR rather than THR seems to be a better index of training intensity, since AerTHR is more specific to the variablitiy in individual response of submaximal work, and thus would be the optimal HR that aerobic training occurs at in these patients, according to the available data. In addition, the multiple r of .74 (although accounting for only 54% of the variance) compared to .46 for prediction of THR, provides better accuracy, and unlike Weltman and Katch's (1979) regression equation for prediction of VO2max, the present equation utilizing height, HRmax and body weight does not depend on metabolic measurment equipment, plus conversion of VO2max into predicted percentages of training VO2max. However, since HRmax is required, a treadmill test would still require a physician in attendance with appropriate rescucitation equipment, which makes this equation impractical in YMCAs, recreation centres and fitness clubs without medical supervision. Thus, a precise intensity index utilizing AerTHR is presented which accounts for individual submaximal metabolic variablility found in this study.

The derivation of AerT using Vtam and subsequent comparison of corresponding metabolic variables deserves discussion. The alinear rise in Ve and excess CO2, heralding the AerT corresponded to a mean Vtam of 4.5 mph. The small variablity of scores as observed in the standard deviation of 0.65 was interesting in that most subjects' Vtam values appeared to occurred at the speed at which running commenced. It was postulated that this initial breakaway of respiratory values could have been a result of the sudden recruitment of muscle mass during the transition from walking to jogging, giving rise to alinear increases in respiratory variables. This could have imitated the first true breakaway point (AerT) as defined by

Skinner and McLellan (1980). In some subjects, a second breakaway point was observed, but corresponded with unrealistically high percentages of VO2max (85 - 90%) to justify this as being the AerT in these subjects (low to moderately trained subjects).

This problem could be a function of the protocol, which we believe might not contain small enough work increments. When dropping a vertical line down from the first alinear rise in Ve and Excess CO2, the speed increments of 0.5mph allow for too much spread in the determination of Vtam within small fractions, which can in reality mean large differences in metabolic activity at various running paces. A work increment of 0.5mph each minute represents a significant increase in running pace. In addition, this protocol might not afford sufficient time per workload for cardiac patients to attain a true steady state.

The change biomechanically from walking to slow jogging may also represent a confounding variable to the interpretation of these curves, and could represent the first visible breakaway point due to the increased muscle mass involvement and resultant inefficiency mentioned above.When one of the present investigators (VO2 max = 65 ml/kg/min) performed this protocol on the treadmill, similar breakawaypoints were also observed at 4.5 mph (initiation of running), with a 'second' breakaway point observed at 9.5 mph. Undoubtedly, this inconsistancy could be avoided by utilizing a bicycle ergometer protocol for smooth work increments, but would seriously reduce the specificity for walking and jogging exercise prescriptions. It is our recommendation that further investigation with samples such as that studied in the present investigation be undertaken. The emphasis should be placed on determining the exact charateristics of respiratory curves in a variety of treadmill protocols utilizing variable speeds (0.25 mph/min), grades(0% to 20%), and increments to uncover whether the first breakoff point is in fact the AerT, or simply increaed metabolic activity. This could be done by incorporating breath-by-breath analysis or lactate studies in a MTT and correlating these to changes in repiratory values, as has been done in elite athletes.

The method of visual inspection of the respiratory variable curves, though more difficult to interpret in less trained subjects (Dunwoody, 1981) have been shown to be as valid compared to computer generated analysis. Orr, et al. (1982) recently compared subjective visual determinations of respiratory AerT with a multi-segmental linear regression computer algorithm, and found a correlation of .94.

Substrate utilization in relation to the AerT is also relevent in this discussion. During predominately aerobic exercise below the AerT, the increased utilization and mobilization of FFA from adipose stores has a significant inhibiting effect on glycolysis (through citrate's inhibition of the Krebs cycle enzyme phosfofructokinase). However, as exercise intensity increases above the AerT, this inhibition is reduced, leading to more glycolysis and less FFA catabolism and lipolysis (Skinner and McLellan, 1981). In addition, adipose tissue lipolysis could be reduced in workloads above the AerT.

Since reduced epinepherine release (which is inhibited during glycolysis) results in less stimulation of adipose cell beta receptors, cyclic AMP production is reduced, resulting in less FFA release into the blood (Issekutz and Miller, 1962). Since reductions in body fat is often an important complimentary goal in CRP's, exercise above the AerT should be avoided, and body fat losses through mainly aerobic means should be encouraged.

Despite these findings however, more needs to be learned about how the AerT can be detected reliably and easily in cardiac patients. Studies with similar groups utilizing blood lactate studies and breath-by-breath analysis (which have already been documented reliably in healthy and athletic samples), needs to be undertaken to further uncover these processes during exercise.

V. CONCLUSIONS

Based on the results of this study, several observations can be made concerning exercise intensity (as measured by HR) and aerobically trained CAD or post-MI patients. Firstly, the relative percent method of prescribing THR does not take into account individual variation in submaximal metabolic variables, and that the use of the AerTHR might be a more accurate and perhaps safer estimation of exercise intensity for this special exercising population. This is especially crutial in terms of long-term patient compliance with the exercise program. In addition, it is possible that exercise above the AerT might reduce FFA mobilization and metabolism, and hence retard the generally desirable body fat losses because of the inhibitory effects of glycolysis on FFA metabolism.

Secondly, the regression equations constructed based on these findings might be useful in conjuction with the relative percent method of predicting appropriate individualized THR in supervised or non-supervised cardiac rehabilitation programs when combined with standard GXTs. However, two drawbacks to this equation exist; 1., although metabolic measurement equipment is not necessary with these equations, a physician with oxygen and resucitation equipment would still be required since a maximum test is performed to obtain HRmax; 2., the equation, though significant, can only account for 54% of the variance, and is thus limited in its use as an independent method of exercise prescription.

Thirdly, more investigation into the treadmill protocol and its applicability and reliability in determining AerT for this population needs to be undertaken, especially with regards to the work increments. Fourthly, more studies investigating the AerT in exercising CAD and post-MI patients needs to be undertaken to fully understand how this index of prescribing exercise intensity for training can be used for these patients.

Finally, because the great majority of exercising CAD, post-MI, and post-surgery patients recieve many forms of beta-adrenergic or calcium agonistic medication which alter age-predicted HR response, investigations into the AerT in these patients needs to be initiated. In these patients, a precise determination of THR based on the AerT would perhaps eliminate substantial errors encountered when utilizing the relative percentage method alone to predict exercise intensity in the face of a multitude of other variables not evident in any other exercising population.

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APPENDIX A - REVIEW OF LITERATURE

Introduction

In exercising anginal and post-myocardial infarction patients, physiological and biochemical variables interact through training to result in increased work capacity (Kavanagh et al., 1973; Kavanagh et al., 1979; Froelicher et al., 1980), reduction of symptoms, and alteration of risk factors (Wilson & Fardy, 1981).

One of the primary reasons of using mild to moderate aerobic exercise such as walking and jogging in cardiac rehabilitation programs (CRP) is to enable an individual to perform greater physical activity with reductions in the metabolic costs to the myocardium (Barnard et al., 1977; Clausen & Trap-Jensen, 1970). Sim and Neill in 1974 showed increases in the anginal threshold in 9 patients with coronary artery disease (CAD) after training 3 days per week for 9 - 11 weeks by walking or jogging. These improvements were significant ($P<.05$) either by work level attained, or by duration of time exercising ($P<.005$), although no direct improvements in myocardial oxygen uptake were found.

Bannister and Taunton (1971) found that after 14 male CAD and post-MI patients were divided into 3 training groups, continuous cycle training resulted in higher physical working capacity than interval or calisthenic-walk/jogging methods. All groups demonstrated reductions in working HR ($p < .01$), diastolic and systolic blood pressure, cholesterol ($p < .05$), and triglycerides after the training program. Detry et al. (1971) attributed significant improvements in 12 CAD patients' maximal oxygen uptake (VO2max) ($P<.001$) of 22.5% after training to increases in arteriovenous 02 differences (A-V02). These changes were thought to be a result of increased peripheral 02 extraction, increased arterial 02 content, or increased hemoglobin post-training.

To a large extent, the beneficial effects of submaximal aerobic training are a result of specific skeletal muscle mitochondrial oxidative capacity improvements (Gollnick & King, 1969; Holloszy, 1973). These changes include increases in slow-twitch (Type I) muscle fiber mitochondrial enzymes (SDH, citrate synthatase), increased size and number of mitochondria per muscle fiber (Holloszy, 1973), improvements in A-VO2 differences (Detry et al., 1971), increases in the capillary to fiber ratio in the exercised muscles (Brodal et al., 1976), increased myoglobin content, and the improved shunting capacity of the circulation from non-exercising tissues to the working muscles (Astrand and Rodahl, 1977). In addition to the peripheral effects, changes in left ventricular perfusion, structure and function as measured in recent radionuclide studies contribute to a central training effect. Froelicher et al., (1980) found slight but non-significant improvements in ventricular function after cycle, treadmill and arm ergometer training 3 times a week at 60%-85% V02max in CAD patients. Jensen et al. (1980) found significant improvements after step climbing, rowing, arm cranking, cycle training, and treadmill running 3 times a week at 65% - 85% V02max in submaximal ejection fraction, maximal work load, V02max, and maximal rate-pressure product, but non-significant increases in maximal ejection fraction.

Body fat reductions have been documented in exercising middle-aged men (Pollock, 1977) and trained post-myocardial infarction (MI) patients (Kavanagh et al., 1973). Favorable changes in blood lipid profiles, particularly, increases in plasma high-density lipoprotein cholesterol (HDL-C) have been demonstrated in exercising CAD patients (Streja and Mymin, 1979). They found significant (P<.01) increases in HDL-C in 32 CRP participants after 13 weeks of 3/week aerobic exercise. High levels of HDL-C have been shown to be associated with lower risk for CAD, and positively correlated with lean individuals and increased aerobic fitness. HDL-C is reduced with CAD, obesity, inactivity, in middle-aged males, and is associated with increased levels of plasma low-density lipoprotein cholesterol (LDL-C) (Wood and Haskell, 1979).

Improved psychological parameters have been reported following training in post-MI patients (Noble, 1977). Kavanagh et al. (1977) found reductions in depression as recorded on the Minnissota Multiple Personality Inventory scale in highly trained post-MI runners compared to a control group.

Cardiac rehabilitation programs emphasizing aerobic exercise training have, in the majority of studies, not statistically shown a reduction in the incidence of subsequent MIs or an increase in the longevity of CAD patients. Despite several large multicentre trials utilizing large sample sizes, no changes in longevity after participation in CRPs were noted (Kalio et al., 1979; Rechnitzer, 1981; Shaw et al., 1981). These studies, however, have lead to further in-depth investigation into the prognostic implications of cardiac rehabilitation programs. Kavanagh and associates (1979) and Shephard et al. (1981) have linked compliance with the exercise prescription as favorably influencing long term survival and the chance of future fatal and non-fatal infarctions.

Admission to a CRP is usually preceded or followed by an initial medical screening process and graded exercise test (GXT). The purpose of this test is to determine the functional status, readiness and safety for exercise therapy (Bruce, 1971; Ellestad, 1975; Hellerstein, 1973), and to assist in the formulation of the exercise prescription (Zohman and Tobis, 1970). In addition, regular assessments throughout a CRP serve to provide additional patient motivation, quantify improvement of functional capacity, and determine if any modification of the exercise prescription is required. Numerous studies have already focused on the applications, sensitivity, reliability, electrocardiographic essentials and procedures of the test protocols available today (Bruce, 1971; Ellestad, 1975; Froelicher et al., 1975).

The exercise prescription is often described in terms of four variables; intensity, duration, frequency, and mode (Fox et al., 1972), with consideration of the interactions and limitations imposed by age, medications, myocardial status, musculoskeletal problems and program design (Wilson et al., 1981). Of the four variables, intensity is the most important, but least understood and agreed upon component of the exercise prescription.

Heart Rate and the Exercise Prescription

An early study to determine the effects of different training intensities, and to form the basic principles and implications for optimizing training programs was by Karvonen et al. (1957). They studied 6 young male subjects who were trained for 30 minutes, 4-5 days per week for 4 weeks on a horizontal treadmill. It was found that if training was performed at greater than 60% of "available range of pulse rates" this would result in an eventual slowing of the heart rate at rest and for any given submaximal workload. The most significant aspect of this study was the introduction of a formula to derive an index of training intensity as measured by heart rate (THR).

$$THR = (HRmax - HRrest) \times (60\% - 70\%) + HRrest$$

This formula has been used extensively in the past and continues to be used as the basis of exercise prescription in many exercise and rehabilitation programs (American College of Sports Medicine, 1980). Although the authors used young adults in their study, the equation remains contemporary and valid since they took into account the age-related maximal HR which has been shown to decline with age (Astrand & Rodahl, 1977).

Investigators have examined these principles in terms of exercise stress testing in a hospital setting, mainly to diagnose the presence of CAD and to define the patient's physical capacity. Sheffield and Reeves (1965) encorporated the principle of "predicted" percent HRmax to arrive at their suggestion of 90% as termination point for most GXTs. The concept of a predicted maximal HR was investigated further by Lester et al., (1968) where normal but athletically trained subjects were found to have slightly lower maximal HRs than untrained subjects. It was found, however, that HRmax had a standard deviation of ±12 beats per minute (bpm), and that there was a wide spread of HRs at a given workload occurring around the regression line, thus showing large variability in HR responses to exercise between individuals. This was substantiated by Ryan in 1980 who noted that 67% of those tested in a GXT will deviate from the predicted rate by ±10 bpm.

Extensive research throughout the 1970's, producing excellent correlations between V02max and HR was carried out by Astrand (Astrand & Rodahl, 1977) which resulted in important principles of training intensity. Based on the linear relationship between V02 and HR at any given workload, THR (as an index of training intensity) was believed to be 70% to 85% of maximum age-related HRmax, or 57% to 78% of V02max for normal healthy young adult, up to middle-aged adults. They subsequently produced nomograms for easy determination of predicted V02max from submaximal bicycle ergometry HR plotted against workload. These nomograms included age, body weight, and an age correction factor to predict V02max, and still used extensively today.

Much of the literature agrees with the basic prerequisites of utilizing these relative percentages as indices of intensity (Fox et al., 1972; Pollock, 1973; Hellerstein et al., 1973). As the field of cardiac rehabilitation developed in later years, these same principles were applied to the exercise prescriptions of post-MI and CAD patients. Zohman and Tobis (1970) suggested training cardiac patients at 75% to 85% of HRmax or 57% to 78% of V02max. Kavanagh et al. (1973) used these guidelines in aerobically trained post-MI patients enrolled in a CRP and found significant improvements in physical working capacity, V02max and ischemic symptoms.

The American College of Sports Medicine outlines several methods of prescribing exercise intensity (ACSM, 1980). They recommend that exercise intensity is best expressed as a percentage of functional capacity, and that intensities not exceed 90% but not be lower than 60% of functional capacity, although with cardiac patients, initial intensities of 40% to 60% should be prescribed. Method 1 utilized prescription by METS. One MET is equal to a resting 02 consumption taken in a sitting position and is approximately 3.5 $ml/kg/min^{-1}$.

<u>Exercise Prescription Utilizing METS</u>

Average Conditioning Intensity = .70 x (METSmax)

Peak Conditioning Intensity = .90 x (METSmax)

Method 2 involves prescribing exercise intensity by heart rate. Heart rate is in one method, plotted against METS and V02 based on upper and lower percentages of V02max or METSmax (60% or 90%). Alternate methods include use of the Karvonen equation previously outlined, or by calculating a given percentage of HRmax to determine THR:

THR = (HRmax x Percent (60%-90%) of maximum HR on a GXT)

This last method of using heart rates to determine exercise intensity however underestimates THR for a given MET level by 15%, and must be corrected by adding 15% to the calculated THR (ACSM, 1980).

Hellerstein et al., (1973) in a review article agreed that cardiac patients could benefit from the same principles of exercise prescription as athletes and healthy normal middle-aged males. They illustrate thatfor CAD patients there exists a good relationship between %V02max and %HRmax. However, they point out some severe flaws in Karvonen's formula, which demand attention when writing exercise prescriptions for those on beta-blocking medications, or symptom-limited maximal HRs. They recommend intensities of 57% to 75% V02max and 70% to 85% of symptom-limited HRmax for CAD and post-MI patients, with symptoms and ECG signs (maximum 4-5mm ST segment depression) taken into account.

Wilson (1975) introduced the concept of the "talk test" for an additional subjective cue for self-determination of correct exercise intensity, apart from the 70%-85% HRmax equation. If one can carry on a conversation while exercising without excessive difficulty, (i.e. moderate ventilation) then the training pace is assumed to be appropriate. Fardy (1977) pointed out that when training at about 70% of V02max, a peak of 82% gain in training adaptation responses will occur for the CAD patient. However, intensities above 70% result in a plateau, and an eventual diminishing response due to fatigue. Hence, the exercise prescription must consider a multitude of factors, yet be specific enough to allow for individual variation (Hellerstein et al., 1972).

When cardiac patients and those at high risk are given a GXT, the purpose, apart from determining the presence and/or severity of significant disease, is to estimate the maximal working capacity and hence be able to prescribe a safe and effective index of exercise intensity (Wilson et al., 1981). Bruce (1971) showed that exercising intensity expressed as a percentage of HRmax or V02max reflected not only functional capacity, but more importantly, myocardial oxygen demand. Thus exercise intensity is related to myocardial function and perfusion status. The Bruce stress test protocol is extensively used in the clinical setting for diagnosis of coronary artery disease as well as an aid in the formulation of the exercise prescription. It consists of continuous 3-minute stages of simultaneously increasing treadmill grade and speed.

Bruce Treadmill GXT

Treadmill Speed (mph)	% Grade	Estimated VO2max (ml/kg/min|$^{-1}$)	METS
1.2	0	8.0	2.3
1.7	5	15.0	4.3
1.7	10	17.5	4.6-5.7
2.5	12	24.5	6.6-7.4
3.4	14	34.3	8.6-10.6
4.2	16	43.8	11.7-14.0
5.0	18	55.5	15.1-16.6
5.5	20	58.0	16.6 +

This protocol and nomogram, which estimates V02max from the stage attained has been criticised. Froelicher et al. (1975) demonstrated an inadequate relationship for predicting V02max from maximal treadmill treadmill time and age. Bruce's Functional Aerobic Impairment index (FAI) has also continued to be regarded as an alternate measurement or expression of a patient's aerobic capacity or cardiovascular impairment:

Bruce Functional Aerobic Impairment Index

FAI = Predicted-Observed V02max / Predicted VO2max x 100

Values in the FAI rating range from normal, which equals a value of 0 indicating 100% of normal aerobic capacity, to values below 0, indicating better than average fitness. Values on the positive side, indicate varying levels of aerobic impairment.

More recently, investigators have begun to question these previous methods of prescribing exercise intensity for CAD patients based on physiological principles such as maximal age-related HRmax from a GXT. Mazzeo et al., (1982) telemetered 16 CAD patients for a 24 hour period which included participation in a CRP (10 minutes warm-up), 20 minutes aerobic, 15 minutes cool-down). THR during the aerobic portion were calculated to be at 54.2% HRmax. Although the patients remained in this THR zone for only 10.3 minutes of the 20 minute aerobic session, a significant improvement ($P<.05$) in functional capacity over a 4.7 month period was found (7.18 METS to 8.19 METS). This improvement, equivalent to 3.46 $ml/kg/min^{-1}$., represented a 20% to 30% relative improvement in functional capacity in these patients. Mean HR at any given submaximal workload decreased after training, enabling patients to achieve higher workloads before the onset of angina symptoms. The authors concluded that lower than accepted intensities of 40% to 60% HRmax reserve can elicit aerobic training effects in selected CAD patients enrolled in a CRP.

Doll et al., (1982) found that in a sample of 150 CAD patients, HRmax increased after 6 months of training in a CRP 3 times per week at 80% to 90% HRmax. It was not indicated whether this increase was significant. The significance of this finding, the authors concluded, was that age-predicted HRmax from data based on normals over-estimates maximal rates and thus exercise HR prescriptions for exercising CAD patients. Sullivan and McKirnan (1982) measured V02 in 12 normal and 12 CAD patients during a Bruce protocol GXT. They found that actual V02max for patients for the 3 stages attained ranged from 1.8 to 7.3 ml/kg/min. lower than would be extrapolated by the Bruce protocol. It was not indicated whether this data was statistically significant. The authors suggested that in CAD or post-MI patients, myocardial damage may alter or slow oxygen "kinetics" and result in lower actual V02 during treadmill GXTs, and that the Bruce protocol might not take this into account. A recent study investigated the Karvonen equation for determination of training intensity. Smith et al., (1982) studied 42 male CAD patients, and compared THR, as determined by Karvonen's equation to the %V02 method THR, and HR during a trial treadmill run. The treadmill run consisted of a 20 minute steady state run at 60% of V02max. The authors found mean trial HR to be 104±16 bpm, mean Karvonen THR of 114±17 bpm, and a mean V02 method THR of 103±16 bpm. Karvonen's THR was significantly higher than actual steady state trial HR ($P<.005$). THRs from the %V02 method was not significantly different from the trial HR. Karvonen's method was accurate within ±6 bpm in 42% of the patients tested compared to 57% of those tested by the %V02 method. The authors concluded that the Karvonen equation overestimates THR, and should be used with caution in exercise prescriptions for cardiac patients.

A survey of the literature thus reveals conflicting findings on the accuracy and reliability of current methods of deriving exercise prescription intensity on the basis of maximum performance on GXTs. Heart rate, as the easiest variable or index of exercise intensity can differ, depending on the method utilized to calculate it. Current methods used to calculate THR include the Karvonen equation, which incorporates HRrest, HRmax and a sliding percentage of HRmax; 70% to 85% HRmax attained on a GXT; or 70% to 90% of maximal METS attained on a GXT.

Anaerobic Metabolism and the Anaerobic Threshold

Lactic acid has for quite some time been identified as a major contributor to fatigue during exercise. The in-depth study into its relationship to exercise continues to be a major focus of investigation.

As lactate is produced in the working muscles as a result of anaerobic glycolysis, it diffuses out of the cells, and into the circulating venous blood. There are many fates of lactate, some of which include reconversion to pyruvate and then complete oxidation in the liver, heart and other organs, or as a nutrient substance in well oxygenated skeletal muscle during rest (Guyton, 1976).

Turrell and Robinson in 1942 studied the biochemical processes of lactic acid production and acid-base balance during anaerobic conditions. They illustrated the increase in lactic acid with decreasing bicarbonate (BHCO3), and provided the model and equation where BHCO3 buffers lactate: (HLa):

$$HLa + BHCO_3 = B(La) + H_2CO_3 = CO_2 + H_2O$$

Carbonic acid, a weak acid, easily dissociates to CO_2 and H_2O. The CO_2 is exhaled, accounting for the increase in the volume of expired CO_2 (VCO_2). The increase in minute ventilation (Ve) is also augmented by the produced H^+ which stimulate medullary centres, carotid and aortic bodies, thus increasing breathing rate (Guyton, 1976). The authors also demonstrated that the rise in HLa concentration equalled the CO_2 capacity of the blood. Isselrutz and Rodahl (1961) introduced the concept of the respiratory exchange ratio, or R, defined by the equation:

$$R = VCO_2/VO_2$$

and found that it not only reflected changing acid-base balance, fuel utilization and anaerobic metabolism, but to also to increase with greater workloads. This relationship was however earlier enlightened by a study by Balke et al. (1954), where he stress tested sedentary blood donors before and after donation and found that VCO_2 initially increased proportionately to the VO_2 increase. However after increasing workloads, VCO_2 exceeded VO_2. When R was greater than 1.0, it was assumed that the limits of aerobic metabolism had been exceeded. They also observed a sudden decline in alveolar pCO_2 with a rise in Ve alinearly to VO_2. In a later study, Wells and co-workers(1957) examined La production in terms of exercise intensity and equated La changes with changes in R. Further studies looked exclusively at exercise intensities, and methods to pinpoint the onset of anaerobic metabolism. Using 102 subjects, Issekutz and Rodahl (1961) found a correlation of .92 between the change in La and excess CO_2 during bicycle ergometry. They hypothesized that the excess CO_2 (derived from the buffering of HLa by HCO_3) was a more useful indicator of anaerobiosis than HLa release, thus demonstrating its reliability and validity for use in determining anaerobiosis. This lead to the equation for calculation of excess, or non-metabolic CO_2:

$$\text{Excess } CO_2 = VCO_2 - (\text{Resting } R \times VO_2)$$

Wasserman and McIlroy (1964) suggested that the onset of anaerobic metabolism, or the anaerobic threshold (AT) occurred at the workload just below the point of non-linear change in Ve and VCO_2 in exercising cardiac patients. They noted that although the onset of anaerobic metabolism could be measured by an increase in HLa concentrations, a decrease in arterial blood pH and HCO_3, and a rise in R, the non-invasive methods of determination utilizing respiratory parameters were more attractive. Later studies by Wasserman et al., (1973, 1975) using breath-by-breath analysis of respiratory exchange variables found good correlations between excess CO_2 and HCO_3 ($r = .98$).

The measurement of blood lactate itself in determination of the AT has been studied extensively, but with less agreement upon its validity. Graham (1978) argued that HLa diffusion out of muscles can be delayed. As well, muscle La and blood La are not equal due to the variable characteristics of blood sampling time, blood flow, diffusion rate and fibre type. An early study to describe the relationship between AT and VO_2max was carried out by Wyndham et al., (1965). They examined the sudden concentration of HLa as the onset of anaerobic metabolism and found that the AT occurred at 50 - 60% of V02max in normal men, and 45 - 50% in hospital patients with cardiomyopathy. Wasserman in 1975 studied the occurrence of the AT in incremental exercise tests using breath by breath gas analysis. He illustrated that the advantages of using a 1-minute as opposed to a 4-minute increment to detect the AT in GXTs are that the 1-minute increment allows discrimination between the AT and other possible causes of non-anaerobiotic increases in R such as hyperventilation, anxiety, or hypoxia. The physiological basis of this advantage, according to the author is that end-tidal P02 increases with no changes in end-tidal PC02, contrary to a 4-minute increment, where end-tidal P02 increases but end-tidal PC02 decreases at the AT. In addition, the shorter increment allows for a shorter total GXT duration, subjects recover more rapidly after the test, and it yields a better plateauing of V02 with progressive work increments (Wasserman, 1975). Wasserman et al., (1973) found the AT detection utilizing breath by breath methods using the 1-minute work increment to be consistently reproducible over 1 hour, 4 hours, 1 week and 9 months. It was also explained however that though detection of the AT in normal subjects or in patients with circulatory insufficiencies is valuable, it has limited use in patients with respiratory impairments.

In 1975, Volkov et al. examined the excess C02 concept in determination of the AT. In conjunction with its alinear rise at a specific treadmill velocity (Vtam), the AT could be confidently determined. The author found Vtam values to be high in trained subjects, with excess C02 remaining constant at submaximal, or sub-AT speeds.

In a study involving middle-aged males, Davis et al. (1976) produced high correlations between the AT determined by HLa and gas exchange variable (r=.95). However, R was not considered a reliable criterion of the AT since it did not discriminate between true anaerobiosis and hyperventilation at higher workloads, thus disagreeing with Wasserman's (1973) findings that the non-linear increase in R is a reliable variable characterizing the AT.

Weltman and Katch (1979) showed the close relationship between the V02 at the AT and V02max (r=.85). The authors concluded that aerobically trained individuals could work at higher percentages of their V02max before they produce lactate as a result of anaerobiosis. AT has been studied in relation to its use in other exercise evaluation protocols. Davis et al. (1976) compared three exercise modes (bicycle, walk, run) and found that V02max and %V02max at AT were similar for each task, and demonstrated the reproducibility of the AT for most protocols. A later study (Davis et al., 1979) demonstrated significant improvements in the AT of up to 44% in untrained middle-aged men after cycling 45 min./day, 4 days a week, for 9 weeks. This result was in agreement with MacDougall (1977) who showed that the AT is trainable and can be increased by a balance of long duration submaximal training plus specific anaerobic high-intensity interval training. He sited various factors including increased muscle capillary density, myoglobin, mitochondrial size and enzyme activity, and increased shunting of pyruvate to the alanine cycle to account for this improved utilization of V02max. LaFontine et al. (1982) demonstrated that an intensity threshhold must be achieved during training, for increases in the aerT to take place independently of increases in V02max. The authors found that subjects who trained at the medium and high intensities (AerT-HR and AnT-HR, respectively) improved the AerT, whereas training at the low intensity (AerT-HR-20 bpm) produced no changes in AerT.

The relationship of aerobic fitness levels and the onset of the AT by examination of Blood La was examined by Costill (1970) and it was shown that elite distance runners (V02max = 73 $ml/kg/min^{-1}$) demonstrated extremely low La levels after a marathon run and at below 70% of V02max during a treadmill run. In a later study, Costill et al. (1973) found a highly significant relationship between %V02max and distance running performance ($r = .94$) on a treadmill run at 10 mph. It was concluded that %V02max at the AT will vary between individuals of different fitness levels and there is an optimal pace dependent on lactate production and subsequent metabolism.

This data is significant for non-athletic populations and those training for health, fitness and rehabilitation. Some investigators have shown that not only does the AT determine the capability to perform aerobic exercise, but can also determine which substrates the individual is utilizing. MacDougall (1977) and Katch et al. (1978) suggested that fat metabolism is reduced when exercising above the AT. Training above the AT will tend to promote glycogen utilization and accelerate muscle glycogen depletion, especially with prolonged activity.

Research by Kinderman et al. (1979) has attempted to take all the previous data and reclassify anaerobic metabolism during exercise (the AT as previously defined by Wasserman) into three transitional phases, reflecting changes in blood lactate concentrations. The "aerobic threshold" occurs at about 2 mmol/L^{-1} La with prolonged exercise maintained for 4 hours. The "aerobic/anaerobic transition" occurs between 2 and 4 mmol/L^{-1} and activity is possible for an hour. The "anaerobic threshhold" is characterized by extreme lactate values in excess of 4 mmol/L^{-1}, where activity can only be maintained for substantially under 1 hour. This constitutes the final phase. The authors, using trained cross country skiiers found that 4 mmol/L^{-1} La occurred at above 80% of maximal treadmill speed with HRs between 169 and 180 bpm. They concluded that optimal work intensities should occur in the aerobic/anaerobic transition zone (2-4 mmol/L^{-1}) for optimal improvement of oxidative pathways and hence endurance.

This terminology was expanded by Skinner and McLellan in their 1980 review. They also suggested an aerobic to anaerobic transitional process with an aerobic phase 1, where 02 uptake satisfies ATP demand, hence minimizing La production. They redefined Wasserman's et al. (1973) anaerobic threshold as the "aerobic threshold" (AerT) preceding phase 2, where La can reach 2-4 mmol/L^{-1} and which corresponds to a point between 40% and

60% of V02max. The third phase, corresponding to 65% to 90% V02max with steeply rising La above 4 $mmol/L^{-1}$ occurs at maximal workloads, and depicts an even greater non-linear breakaway of Ve and VC02 to compensate for the metabolic acidosis. Hyperventilation becomes evident by a drop in FEC02 and a rise in FE02. Phase 3, following the anaerobic threshold is characterized by:

1. reduction/occlusion of muscle blood flow.
2. recruitment transition from SO to FOG and FG muscle fibers
3. decreased FFA utilization.
4. increased glycogen utilization

Exercise Prescription and the Aerobic/Anaerobic Threshold

In 1976, Weltman et al. studied 28 moderately trained students and determined The AerT using Ve, VC02, and FE02 changes. They showed that those subjects matched for V02max did not necessarily show similar V02 at AT values, indicating that at submaximal workloads, there are metabolic differences that V02max does not take into account. A later study by Weltman and Katch et al. (1978) using stepwise correlations of performance and respiratory data indicated that "heart rate attained on a test, or HR at the point of metabolic acidosis shows little relationship with the other variables", thus agreeing with Wasserman's et al. (1973) observation that HR response is a poor predictor of metabolic acidosis. They also presented a method of regression for prediction of V02max from V02 at AT (V02maxAT), which the authors suggested "is a possible method of determining functional capacity in a clinical setting where exercise through metabolic acidosis is unadvised".

The use of the aerobic threshold as a basis for training was recently examined by McLellan and Skinner (1981). Fourteen male subjects were trained at either a relative percent of V02max or percent of aerobic threshold (AerT) for 30 - 45 minutes/day, 3/week, for 8 weeks. Significant improvements in V02max between pre and post-training (23.8% AerT vs. 18.3% %V02) were found, but there were no increases in AerT values after training as expressed as %V02max. The authors concluded that using the AerT as an index of intensity, equal improvements in aerobic fitness are possible.

Poweles et al. (1979) studied 39 middle-aged post-infarction males and found 18 who were 2 standard deviations below age predicted HRmax. Despite the pathological reasons behind this poor HR response, the authors felt that this was a case where the prescribed target HR would have been grossly overestimated and unsafe. The anaerobic threshhold (AerT) was determined by observing an alinear Ve response and detected in 82% of the subjects, with mean post exercise La of 7.5 mmol/L^{-1}. They concluded their study with strong recommendations that in these types of patients, determination of the AT can safely determine the cardiovascular limitations to exercise.

Katch et al.(1978) found a correlation of .97 between V02-max and HRmax in 31 male subjects. However, when the author removed the time element from the equation, this value dropped to .17. Katch evaluated the use of the "relative percent" concept in prescribing training HRs. He questioned the use of HR as the sole index of training intensity, as significant differences between subjects were found in the responses in training intensities when individuals were working at the same relative percentage of HRmax. At 80% HRmax, 17 subjects were training at or above the AT, with 14 training below it. Hence, the author advised that since the relative percent concept might be invalid for equating training intensities, the AT could be used to do so more reliably, thus accounting for individual variation of HR and submaximal metabolic responses.

In a study by Dwyer and Bybee, (1981), utilizing low to moderately trained females, HRs between 60% and 80% of V02max resulted in inconsistent work stress among the subjects. and is was concluded that the AT was a better basis for exercise prescriptions than an arbitrarily determined %HRmax or %V02max.

Dressendorfer et al. (1981), studying untrained healthy middle-aged men agreed, reported ATs occurring well above the upper zone of THR recommended for safe training . This result was statistically significant (P<.05).

Parkhouse and McKenzie (1982) studied untrained, trained, and highly trained young adults, and, contrary to Katch'set al. (1978) findings, concluded that HR was in fact an adequate predictor of AT. All three groups had different ATs; those highly trained having increased ATs. Both absolute and relative HRs at AT were essentially the same for all groups, but it was cautioned that this could be a result of the large intragroup variability. These results were also in agreement with data by Patton et al., (1980).

Wilmore et al., (1981) assimilate this information with regard to the exercising cardiac patient. They outline a rationale for cardiac rehabilitation participants to be exercising at a %HRmax which is just below the aerobic threshold of Skinner and McLellan (1980). They suggest that the aerobic threshold should be used in a GXT along with %HRmax target HR to further individualize the exercise prescription. Since an impaired myocardium cannot achieve high stroke volumes, and a tachycardia at low workloads accommodates for this, HRs do not reflect the true metabolic requirement. It was also noted that cardiac output is limited by HRmax, which can vary in cardiac patients, agreeing with observatons by Poweles et al., (1979).

Other variables such as psychological, medication, musculoskeletal limitations and compliance with the exercise program exist, hence extra caution must be incorporated into the formulation of the exercise prescription's level of intensity (Kavanagh et al., 1973). The exercise program must incorporate and fulfil two requirements; it must provide the desired physiological adaptive effects, and be consistently enjoyable and tolerable (Wilson et al., 1981).

Kavanagh et al. (1979) found that compliance with the exercise protocol was the most important single determinant of prognosis. The risk ratio for fatal and non-fatal reinfarctions was 23.6 times higher for poor compliers. Further analysis of these results in subsequent studies suggested that 22% of the non-compliers compared to 4.4% of compliers had a combined fatal and non-fatal recurrence rate (Shephard et al., 1981). George et al. (1981) also found that patients who sensed considerable fatigue during the exercise session had greater dropout rates.

Thus, improper exercise intensities, as far as very recent research suggests, could be one factor in these non-compliance and reinfarction data (Wilmore et al., 1981).

The study of the onset of anaerobic metabolism in both normal and cardiac patients suggests that "relative percent" methods of prescribing exercise intensity might not account for differences in individual variations in metabolism at submaximal workloads. Using the Aerobic Threshold in addition to other standard methods might optimize the conditioning effects of the subject. This has great value in the testing and training of exercising CAD patients, whose exercise prescription intensities must be individually based.

APPENDIX B - INDIVIDUAL SUBJECTS PHYSIOLOGICAL DATA

VO2max (ml/kg/min)	%VO2mAerT	Vtam (mph)	HRmax (bpm)	THR (bpm)	AerTHR (bpm)	%HRmaxAerT	%HRmaxT
42.91	52.50	5.00	157	126.0	112	71.0	80.0
34.90	43.40	4.50	152	128.2	108	71.0	84.3
33.30	55.90	5.00	186	132.0	151	81.1	71.1
35.46	61.73	5.50	166	137.3	148	89.1	82.7
32.20	58.20	4.50	168	137.3	140	83.3	82.0
40.87	47.90	5.00	161	134.0	125	77.6	83.2
35.97	53.90	4.50	155	124.0	117	75.4	80.0
34.43	54.00	5.00	160	144.3	141	88.1	90.1
39.20,	50.40	4.50	186	160.0	130	69.8	86.0
39.20	55.24	4.50	159	123.0	118	74.2	77.3
52.70	58.97	6.50	178	125.0	137	76.9	70.2
37.00	55.20	4.50	167	144.0	123	73.6	86.2
33.12	49.20	4.00	184	146.0	128	69.5	79.3
32.76	42.10	4.00	175	104.6	107	61.1	59.7
30.57	72.70	4.00	160	125.3	108	67.5	78.3
32.03	51.60	4.00	164	115.7	117	71.3	70.5
36.09	56.10	4.00	184	156.0	135	73.3	84.7
27.23	61.40	4.00	157	136.6	118	75.1	87.0
30.60	50.80	4.00	150	147.0	94	62.6	98.0
30.97	57.80	4.00	155	128.0	140	90.3	82.5

APPENDIX C - RELIABILITY STUDY

Paired t-tests were performed on AerT(1) and AerT(2) using BMD program p3d. Results showed a test/re-test reliability of r = .967 between the five subjects.

Raw Data

Subjects	AerTHR 1 (bpm)	AerTHR 2 (bpm)
1	140	146
2	151	157
3	130	135
4	118	128
5	112	108

r = .97

APPENDIX D - SAMPLE OF COMPUTER GENERATED MEAN TRAINING HEART RATE

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********************************************
*        HEART RATE MONITOR PROGRAM        *
*J.M.BUCHANAN FITNESS AND RESEARCH CENTER*
*         SCHOOL OF PHYSICAL EDUCATION     *
*           DEPT OF SPORT SCIENCES         *
*            UNIVERSITY OF B.C.            *
*              VANCOUVER, B.C.             *
********************************************
```

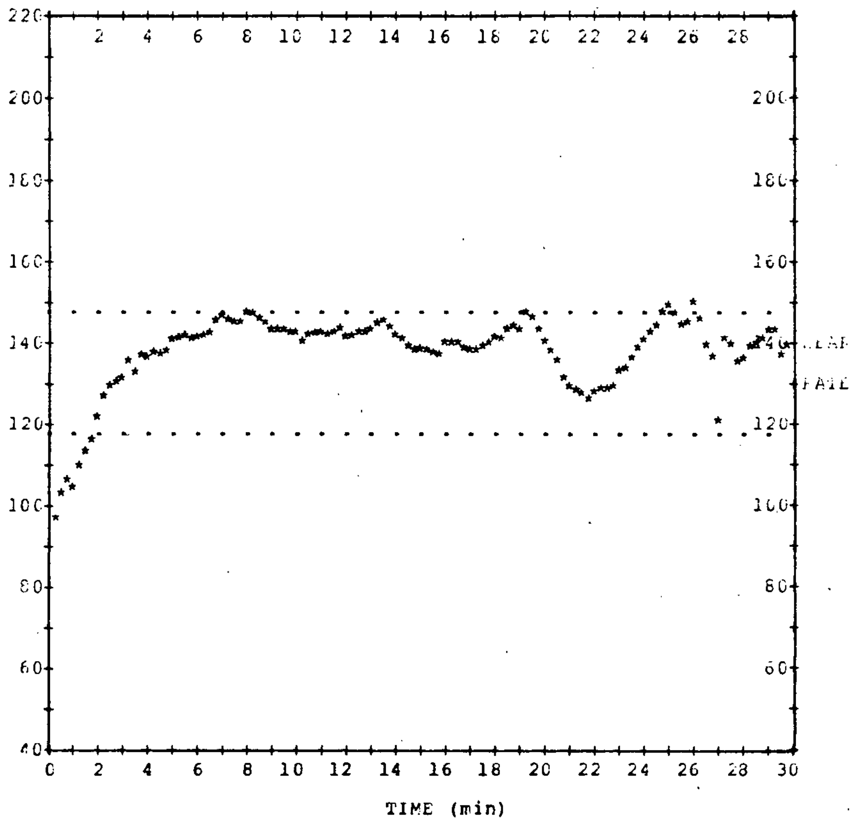
TIME min	SECONDS 00	15	30	45	MEAN HEART RATE
0		97	103	106	102
1	104	110	113	116	111
2	122	127	129	130	127
3	131	135	133	137	134
4	136	137	137	138	137
5	140	141	141	141	141
6	141	141	142	145	142
7	146	145	145	145	145
8	147	147	146	145	146
9	143	143	143	142	143
10	142	140	142	142	142
11	142	142	142	143	142
12	141	141	142	142	142
13	143	145	145	144	144
14	142	141	139	138	140
15	138	138	137	137	138
16	140	140	140	139	140
17	138	138	139	140	139
18	141	141	143	144	142
19	143	147	146	143	145
20	140	138	135	132	136
21	129	128	128	126	128
22	128	129	129	129	129
23	133	134	136	139	135
24	141	142	144	147	144
25	149	147	144	145	146
26	150	146	139	136	143
27	121	141	139	135	134
28	136	139	139	141	139
29	143	143	137	139	141

VOLTS x 55 = HR

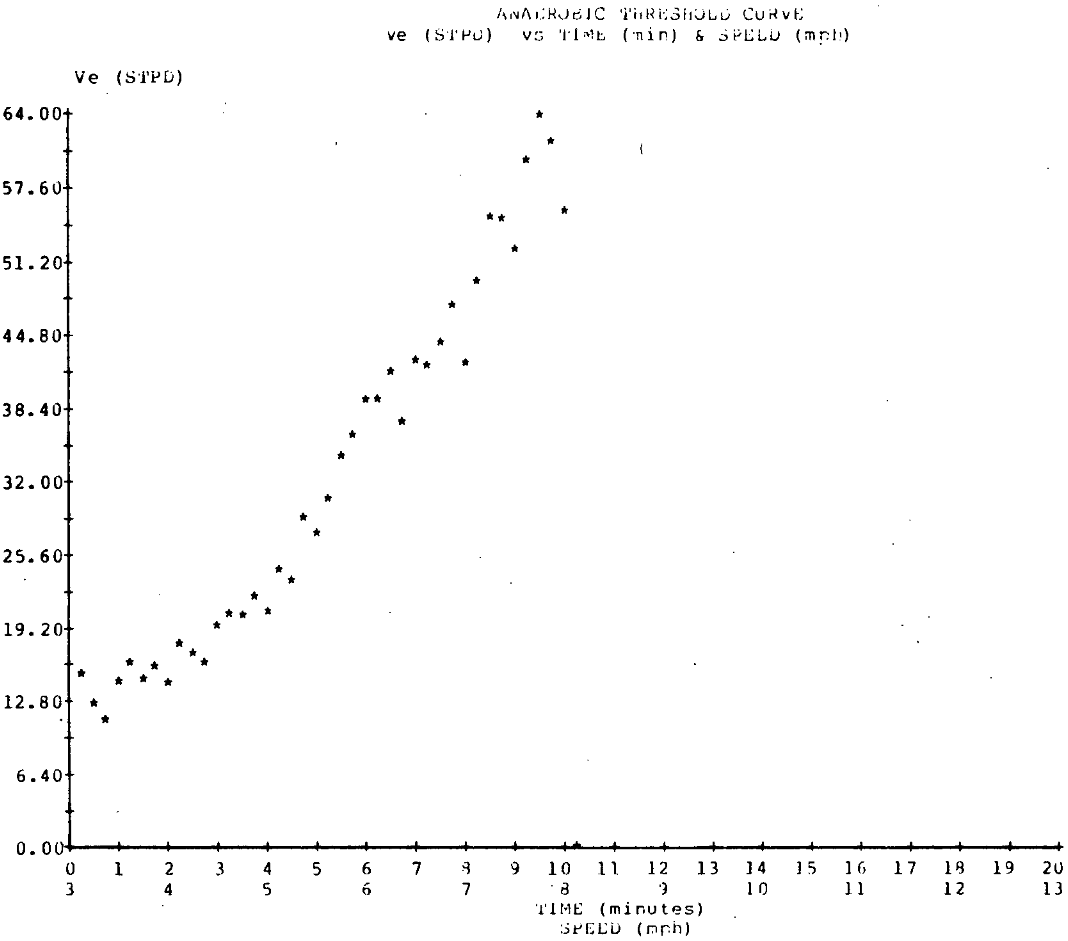
APPENDIX E - SAMPLE OF COMPUTER GENERATED PLOT OF TRAINING HEART RATE

NAME:	
AGE:	53 YEARS
SPORT:	Cardiac Rehab
DATE:	JUNE 27,1982
UPPER LIMIT:	200-AGE+147
LOWER LIMIT:	170-AGE+117

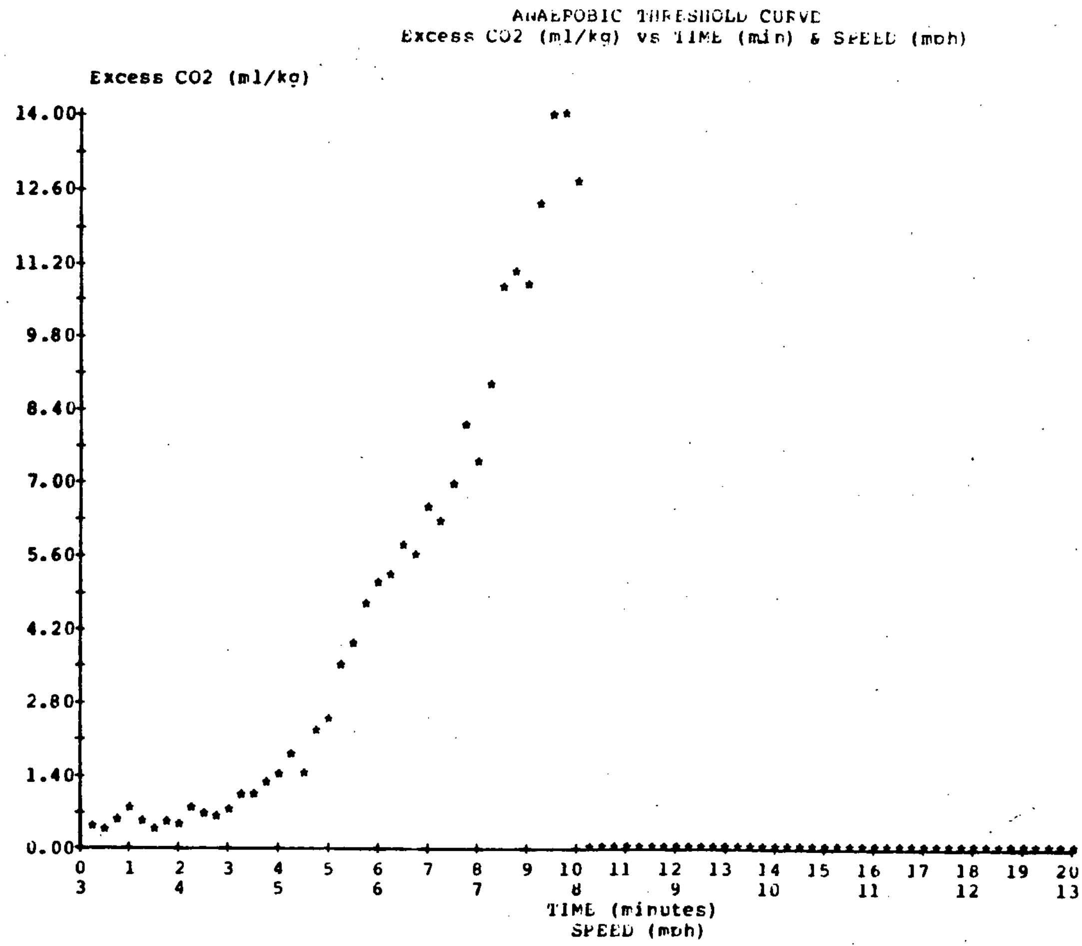
HEART RATE vs TIME GRAPH



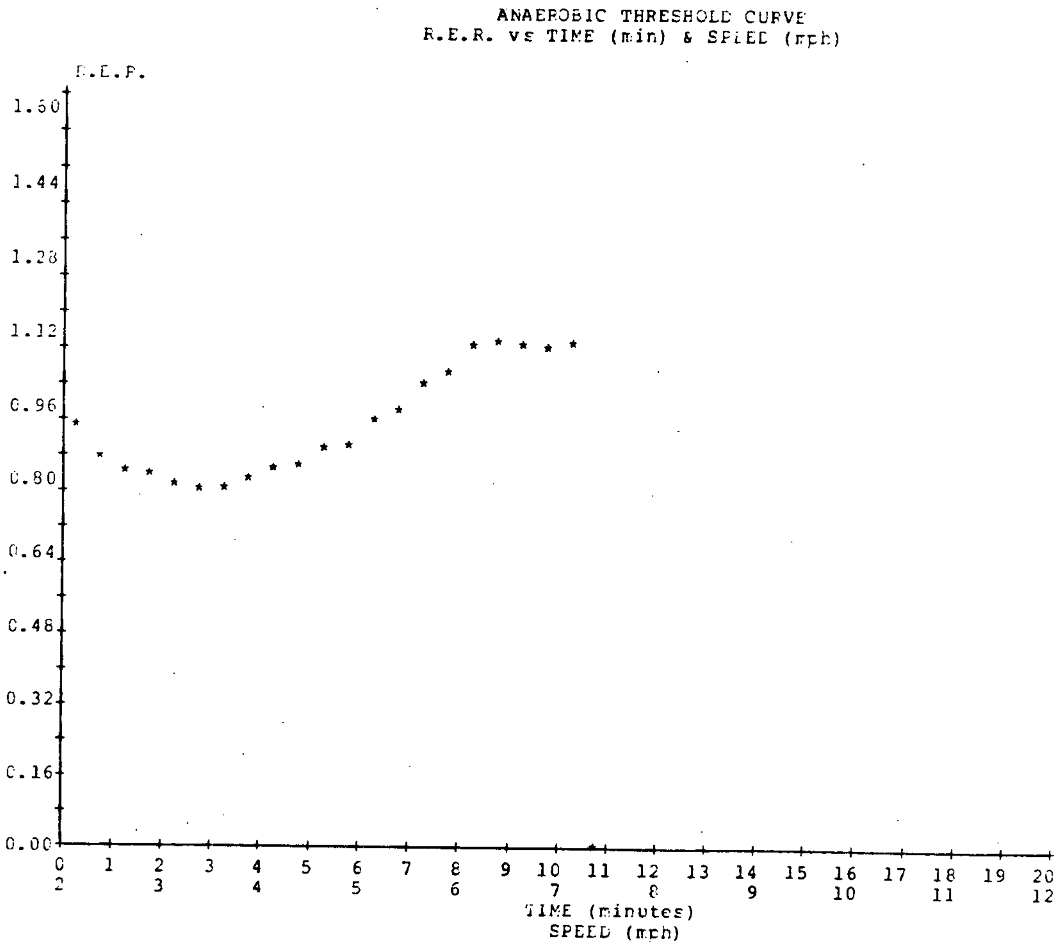
APPENDIX F - SAMPLE OF COMPUTER GENERATED PLOT OF MINUTE VENTILATION



APPENDIX G - SAMPLE OF COMPUTER GENERATED PLOT OF EXCESS CARBON DIOXIDE



APPENDIX H - SAMPLE OF COMPUTER GENERATED PLOT OF RESPIRATORY EXCHANGE RATIO



APPENDIX I - SAMPLE OF INFORMED CONSENT FORM

PROTOCOL AND CONSENT FORM

TITLE: HEART RATE AND ANAEROBIC THRESHCOLD IN EXERCISING CARDIAC PATIENTS

THIS STUDY INVOLVES TWO PROCEDURES WHICH WILL BE DONE ON TWO SEPARATE OCCASIONS. THE INITIAL TEST WILL BE DONE AS PART OF YOUR USUAL EXERCISE PROGRAM DURING THE REGULAR CARDIAC REHABILITATION EXERCISE SESSION. YOUR HEART RATE WILL BE MONITORED BY ATTACHING A SERIES OF ELECTRODES (3) TO YOUR CHEST AND HAVING YOU WEAR A SMALL BATTERY OPERATED RECORDING DEVICE. THIS WILL TAPE YOUR HEART RATE RESPONSE TO YOUR STANDARD EXERCISE ROUTINE. THERE IS NO DISCOMFORT AND YOU WILL NOT EVEN BE AWARE OF THE UNIT ONCE YOU ARE EXERCISING.

THE TREADMILL EVALUATION WILL TAKE PLACE IN THE J.M. BUCHANAN FITNESS AND RESEARCH CENTRE ON THE U.B.C. CAMPUS. YOU WILL BE ASKED TO RUN ON A TREADMILL WITH A PROGRESSIVE INCREASE IN SPEED UNTIL YOU ARE UNABLE TO CONTINUE. YOUR HEART RATE WILL BE CONTINUOUSLY MONITORED AND YOU WILL BE ASKED TO BREATHE THROUGH A MOUTHPIECE SO THAT YOUR EXPIRED AIR CAN BE COLLECTED AND ANALYZED. FROM THIS INFORMATION WE CAN CALCULATE YOUR ANAEROBIC THRESHOLD.

THE RISKS OF THE TREADMILL RUN ARE MINIMAL, BUT IF ANY PROBLEMS DO ARISE THE LABORATORY DOES HAVE ALL NECESSARY MEDICAL EQUIPMENT FOR TREATMENT OF EMERGENCIES AND THE TEST IS MONITORED BY A PHYSICIAN.

ALL DATA WILL BE TREATED IN CONFIDENCE. IN REPORTING THE RESULTS, NAMES OF THE SUBJECTS WILL NOT BE USED. WE WILL BE HAPPY TO ANSWER ANY ENQUIRIES CONCERNING THE PROCEDURES OR THE STUDY IN GENERAL

I CONSENT TO PARTICIPATE IN THIS RESEARCH PROJECT.
I UNDERSTAND THAT I MAY WITHDRAW FROM THE STUDY AT ANY TIME WITHOUT PREJUDICE TO FUTURE CARE.

____
(Signature)

____________________ __________
(Witness) (Date)

Investigators: D.C. McKenzie
L. Goodman

APPENDIX K - STEPWISE CORELLATION MATRIX

	%VO2maxAerT	Vtam	age	%HRmaxAerTHR	AerTHR
VO2max	-.11	.79¤			
HRmax			-.20		.51
THR					.30
%HRmaxTHR				.16	
Vtam					.46■

¤ ($P < .01$)
■ ($P < .05$)