A Comparison of the Effects of Three Selected Exercise Intensities on the Lactate Recovery Ability of Elite Distance Runners

by

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Abstract

Six elite, male, cross-country runners were assessed on a motorized treadmill for aerobic endurance capacity using blood lactate ([HLa]) recovery ability (ΔLa) in three square-wave endurance exercise tests (SWEET) with repeat intervals below (S1), at (S2), and above (S3) threshold (OBLA). Each SWEET was composed of 6 possible repeat intervals (OVERs) followed by active rest intervals (UNDERs) of the same distance where $\Delta La = [HLa]_{OVER} - [HLa]_{UNDER}$. S1 and S2 interval [HLa] values where stable whereas S3 interval [HLa] values drifted upwards over the 6 repeats. Interval HR values were rising slightly over the SWEET durations. Interval $\dot{V}O_2$s were similar in value over the SWEET durations. ΔLa (mM) values were significantly increased ($p<0.05$) from 0.66 ±0.06 for ΔLa_{S1}, to 1.31 ± 0.45 for ΔLa_{S2}, to a nearly significant increase ($p=0.06$ for N=6) to 1.58 ±0.79 for ΔLa_{S3}. ΔLa_{S1} and ΔLa_{S2} were 40% and 80% of ΔLa_{S3} values. Post hoc analysis revealed the source of statistical significance as all SWEET repeats except 1 and 6. Within SWEET repeat ΔLas were not significantly different. Lactat recovery ability increases were attributed to either the increased utilization of La as a fuel during periods of increased metabolic rate or the augmented flow of La down concentration gradients from lactate producing space to lactate usage and storage space. Implications for runners were that excursions at or above threshold levels were compensated for by increased ΔLa, but that the number of repeat intervals possible during racing and or training may vary for individuals and should be considered.

Introduction

Testing of high performance athletes in sport science laboratories is used not only as an assessment of physiological responses but also as an indicator of future training stimuli for whatever training philosophy and exercise prescription is pursued by the athlete and/or coach.

Aerobic endurance athletes are not as concerned with $\dot{V}O_2max$ scores as with the intensity level that has a high correlation to race performances (Londeree, 1986) and has a high fractional utilization of aerobic power (Costill et al., 1979). Blood lactate concentration ([HLa]) thresholds have been used to assess both maximal steady state (MSS) exercise (Heck et al., 1985) and the point at which lactate begins to accumulate, defined as the onset of blood lactate accumulation (OBLA) (Sjödin and Jacobs, 1981). However, the high correlation for mean running velocity that elicits OBLA versus mean race pace velocity diminishes to moderate and low correlations when a group is fairly homogeneous (Sjödin & Svedenhag, 1985). The individual anaerobic threshold (IAT), which is based on the assumption that HLa removal is in equilibrium with production at the IAT point determined by Stegmann et al.'s model (1981), can discriminate among similar athletes (Stegmann et al., 1981; Stegmann and Kindermann, 1982). This greater discrimination holds promise of a new understanding of aerobic endurance exertion for the examination of individual HLa removal.

Notwithstanding the above, controversy still exists about progressive incremental exercise test results, especially when assessing not only [HLa] inflection

points (Richardson & Gladden, 1984; Beaver et al., 1985; Campbell et al., 1989) but also aerobic endurance capacity from [HLa] (Maasen & Busse, 1989).

Other methods of measuring aerobic endurance capacity include both repeat intervals (Astrand, 1986) or Square-Wave protocols with HLa removal ability measured after each active rest interval (Gimenez et al., 1982a; Gimenez et al., 1982b; Rieu et al., 1989). Therefore the purpose of this study was to describe the effects of three endurance exercise tests (SWEETs) of repeat intervals set at three selected exercise intensities around threshold on HLa recovery ability in elite male distance runners during treadmill running. Changes in the patterns of heart rate (HR), oxygen consumption ($\dot{V}O_2$) and [HLa] responses during the three SWEETs was also of interest.

Methodology

Six, elite, male cross-country runners of the 1991 CIAU championship team volunteered, signed informed consent (see Appendix A), then completed a lactate identification test followed by a series of 3 Square-Wave Endurance Exercise Tests (SWEETs). One test occurred every other day. The 18 SWEETs were randomized in a latin square pattern with one exception, test 17 and 18 were reversed in order (See appendix H for exception and reason).

The La Identification Tests were progressive incremental continuous tests of 3 minute stages starting at a speed of 10 km x h^{-1} with increases of 1 km x h^{-1} till a

[HLa] of 5.5 mM was reached. Fingertip blood samples of whole mixed venous arterial blood was sampled in the last 20 seconds of each stage from the subjects outstretched hand while he continued to run on the treadmill. Workloads that elicited a [HLa] of 2.5, 4.0 and 5.5 mM (see Appendix I1.1.4.4 "Fixed Blood Lactate Concentrations" p. 64) were identified from this test as OVER intervals by interpolating to the nearest 0.5 km x h^{-1} of treadmill velocity for the target [HLa]s. The workloads that preceded the first significant rise in [HLa] were identified as the UNDER intervals. Because the HLa Identification Test was not taken to $\dot{V}O_2max.$ levels, a traditional indicator of intensity, $\%\dot{V}O_2max$, was not availbale to categorize the subjects responses. [HLa] was used as the marker of intensity in the present sudy.

Each of the three SWEETs were composed of six three minute OVER intervals interspersed by equal distance UNDER intervals (See Appendix C Tables C1 and C2). This was done for two reasons: first, the fixing of the distance is traditional in track and field, and second this guarantees a minimum of 3 minutes for La diffussion time for the UNDER interval. Delta La (ΔLa) was determined as the difference between samples taken after an OVER interval (in the first 20 seconds of the UNDER interval after the treadmill slowed and and while the subject continued to run with an outstretched hand) and just before the next OVER interval (in the last 20 seconds of the UNDER interval). Fingertip whole blood was assayed through a micropunture technique requiring 20 µL that was immediately haemolized and diluted for analysis via the Kontron 640 Lactate Analyzer, which has been shown to be both reliable

(±0.05 mM) and valid (±0.1 mM) (Geyssant et al., 1985). Oxygen consumption and carbon dioxide production were assessed throughout the test using the Amtek Electrochemistry Oxygen Analyzer (S-3A1) and the Carbon Dioxide Analyzer (CD-3A). The heart rate was monitored beat to beat by the PE3000 sport tester by Polar Electronics.

All descriptive and inferential statistics were calculated with the Statistical Analysis System (SAS) using a 5% level of significance. A one way ANOVA with three levels of exercise intensity was conducted with 6 repeated measures for ΔLa using the General Linear Model (GLM) procedure of SAS.

Results

Subjects

Six subjects are described anthropometrically in Table 1 and can best be described as "elite" (Sjödin & Svendenhag, 1985) runners possessing high thresholds (velocity at OBLA), $\dot{V}O_2$s, and performance times.

The individual [HLa], HR, and $\dot{V}O_2$ responses are listed in Appendix D with means for each SWEET for repeats by OVER, UNDER and ΔLa ($[HLa]_{OVER}$-$[HLa]_{UNDER}$) in Appendix F. The patterns of individual responses are fully depicted in Figures E1 to E12 in Appendix E. All individual cell correlations for each HR, [HLa], and $\dot{V}O_2$ for each subject by SWEET and by stage are fully listed in Appendix G.

Pooled subject data are summarized in Table 2 with patterns depicted in Table

Table 1

Mean height, weight, age, treadmill running velocity at OBLA from progressive incremental testing, and 10 Km (10 K) personal best running times for 6 elite cross-country runners on seperate but similar courses.

Subject Number	Age (years)	Height (cm)	Weight (kg)	Stage for [HLa_4] (km x h^{-1} - km x h^{-1})	Personal best for any 10 k race (min:sec)
1	22	170	68.4	17-18	30:47
2	23	183	67.5	16-17	31:30
3	26	184	67.0	16-17	35:45
4	21	178	64.7	17-18	31.22
5	21	179	70.0	16-17	30:30
6	24	172	61.0	18-19	27:45
Mean for N=6	22.9	177.7	66.5	16.6-17.6	31:17

1. Figures 1, 2 and 3 are for mean [HLa], HR and $\dot{V}O_2$ responses respectively.

The mean peak [HLa] values for the OVER intervals for the first two SWEETs are consistently close to the target levels of 2.5 and 4.0 mM for the 6 repeats, whereas the mean peak [HLa] values for the highest intensity SWEET (3) shows a pattern of slight increasing values above 5.5 mM for the 6 repeats. All the SWEET pattern mean [HLa]s are distinctly different except SWEET 1 and 2 mean UNDER [HLa] for repeat 6 where the standard error bars overlap.

The pattern of mean HR responses show an upward drift for the three SWEETs. A clear distinction between the SWEET 1 OVER HR pattern versus the SWEET 2 and 3 OVER HR pattern was evident, as depicted in Figure 2. The same differentiation between SWEET 1 OVER values versus SWEET 2 and 3 OVER values is apparent in Figure 3 for the mean $\dot{V}O_2$s except for the third repeat OVER where SWEET 1 and 2 values overlap. In contrast to the HR responses the UNDER oxygen consumptions show no slight upward increases.

The mean ΔLa for SWEET 1, 0.66 mM, is 40% of that found for SWEET 3, 1.58 mM, whereas the ΔLa for SWEET 2, 1.31 mM, is 80% of that found for SWEET 3 (See Table 2). The pattern of ΔLa for the three SWEETs is consistent with the order of the differences between SWEETs for HR and $\dot{V}O_2$ being that SWEET 1 is clearly lower than 2 or 3 (Figure 4) except that the fifth UNDER repeats for SWEET 2 were slightly higher than the fifth UNDER repeats for SWEET 3.

The HR, [HLa], $\dot{V}O_2$ correlations in Table 3 and 4 from data pooled by

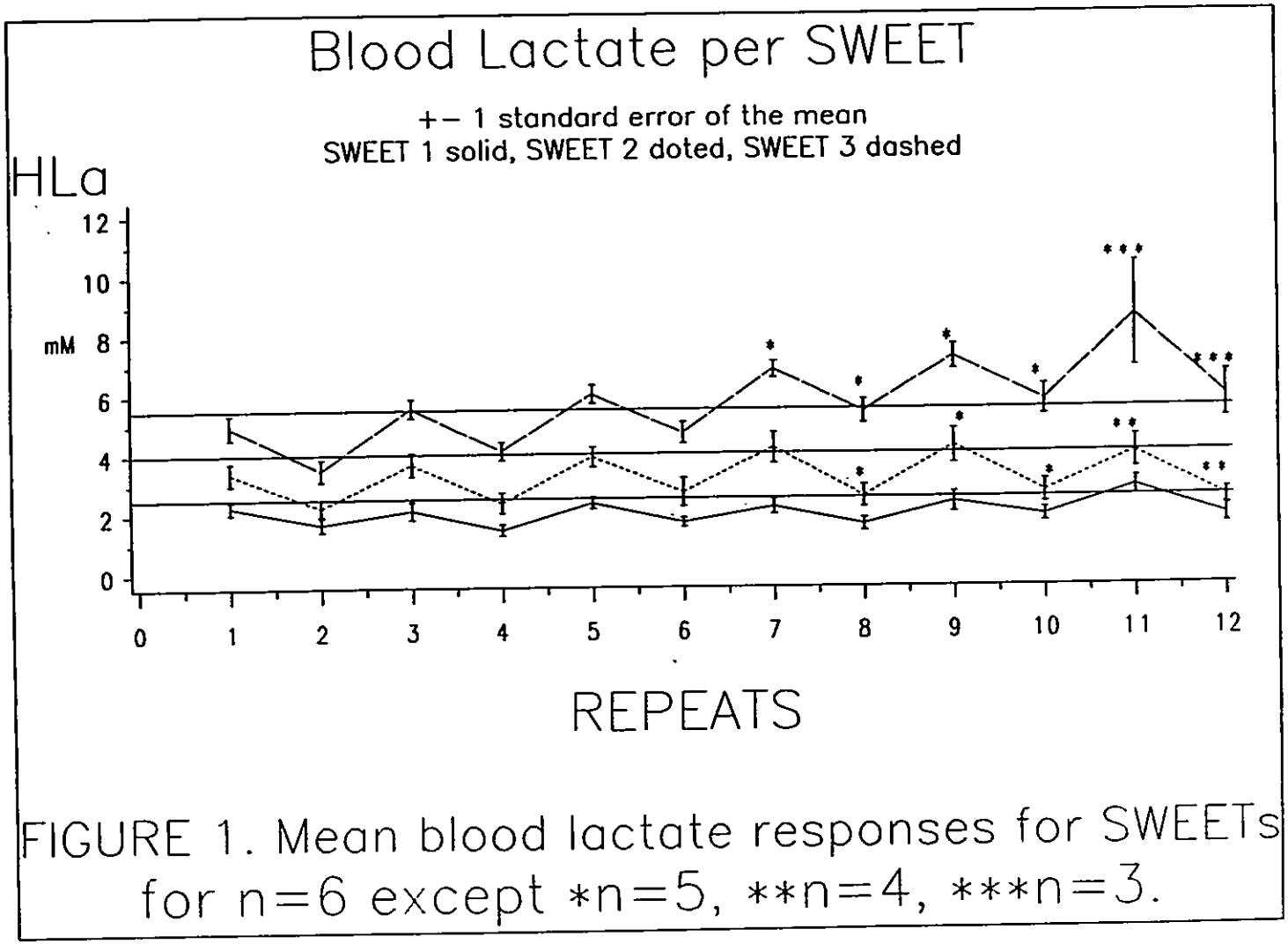

FIGURE 1. Mean blood lactate responses for SWEETs for n=6 except *n=5, **n=4, ***n=3.

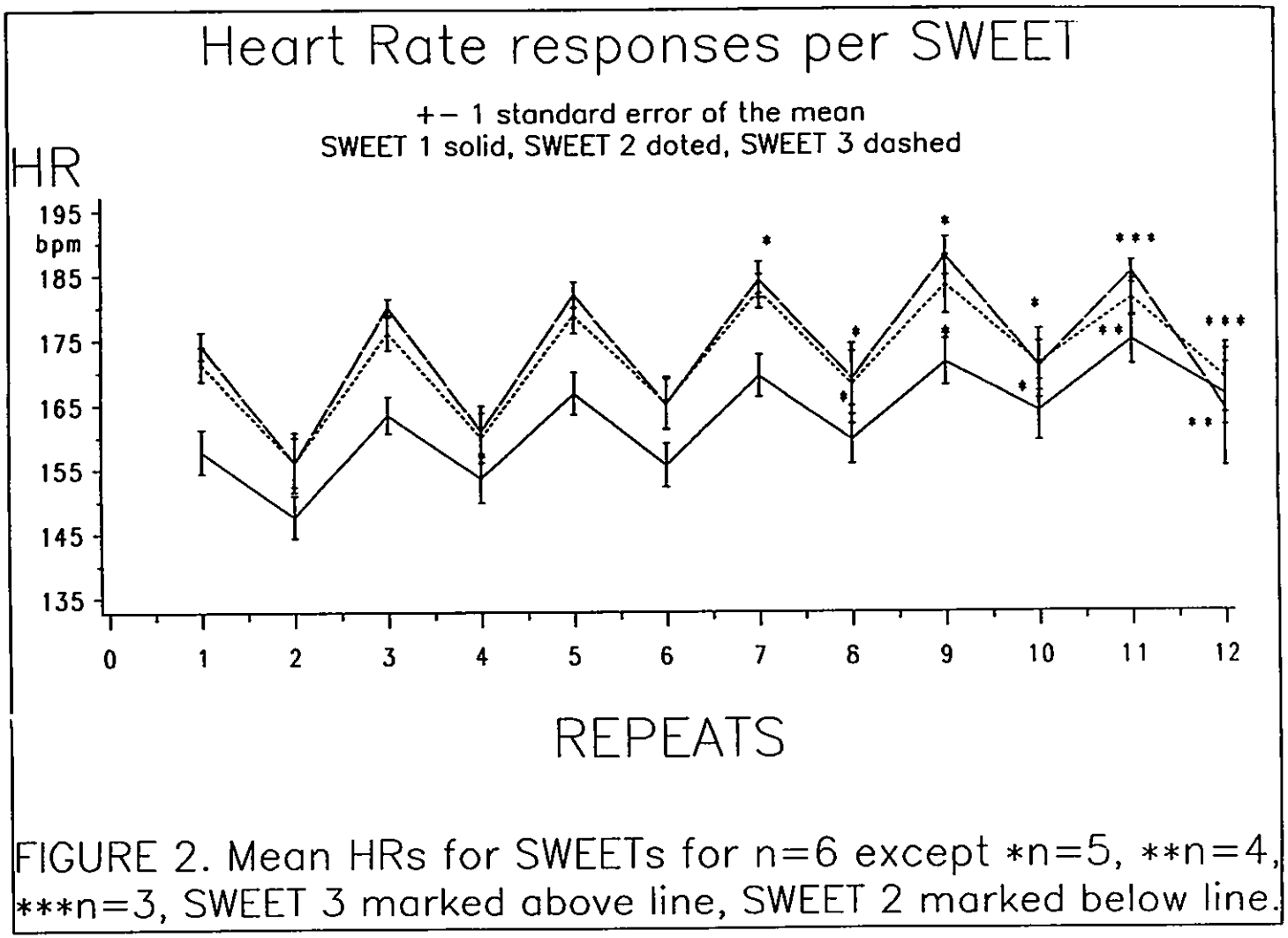

FIGURE 2. Mean HRs for SWEETs for n=6 except *n=5, **n=4, ***n=3, SWEET 3 marked above line, SWEET 2 marked below line.

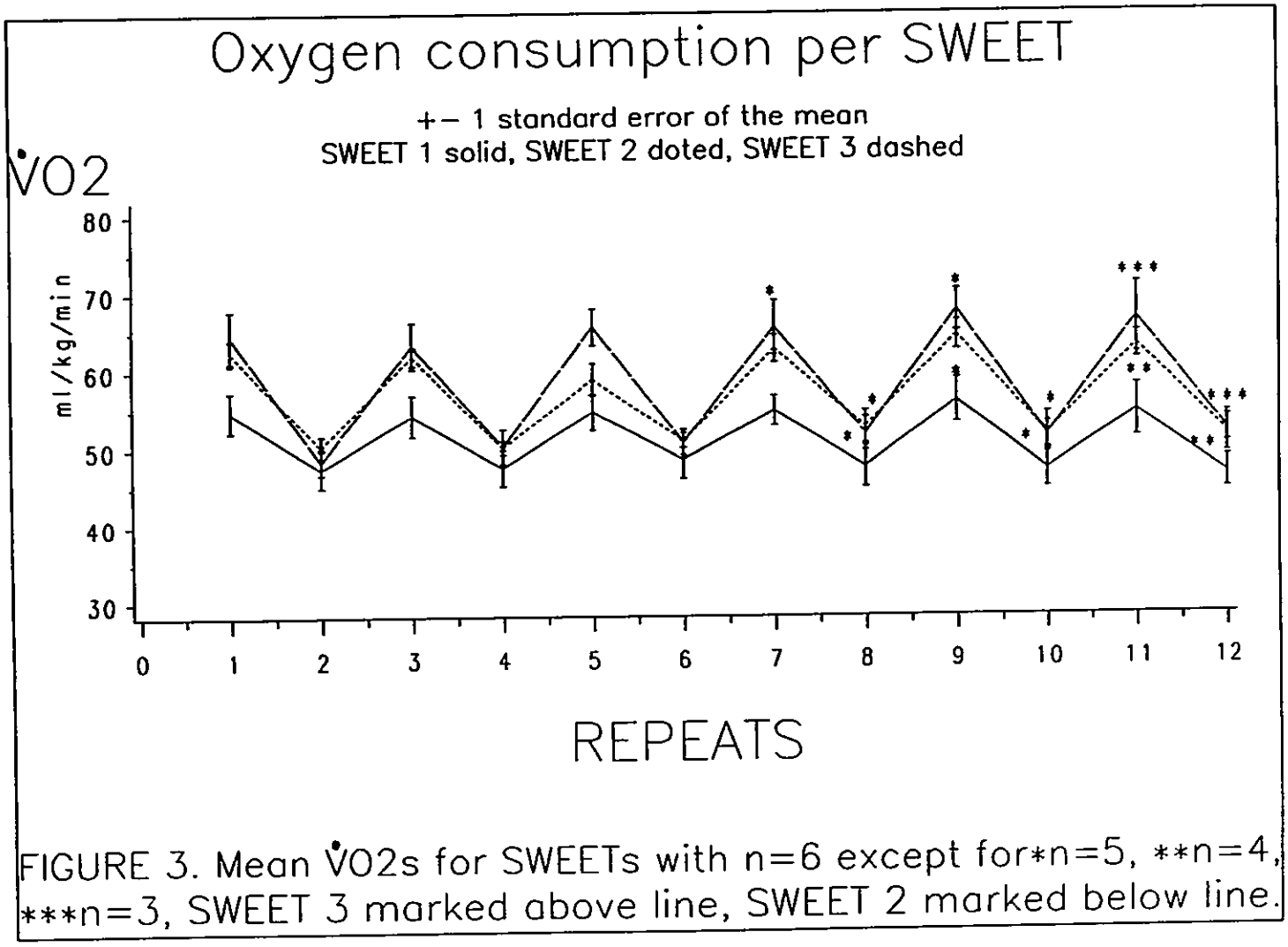

FIGURE 3. Mean V̇O2s for SWEETs with n=6 except for*n=5, **n=4, ***n=3, SWEET 3 marked above line, SWEET 2 marked below line.

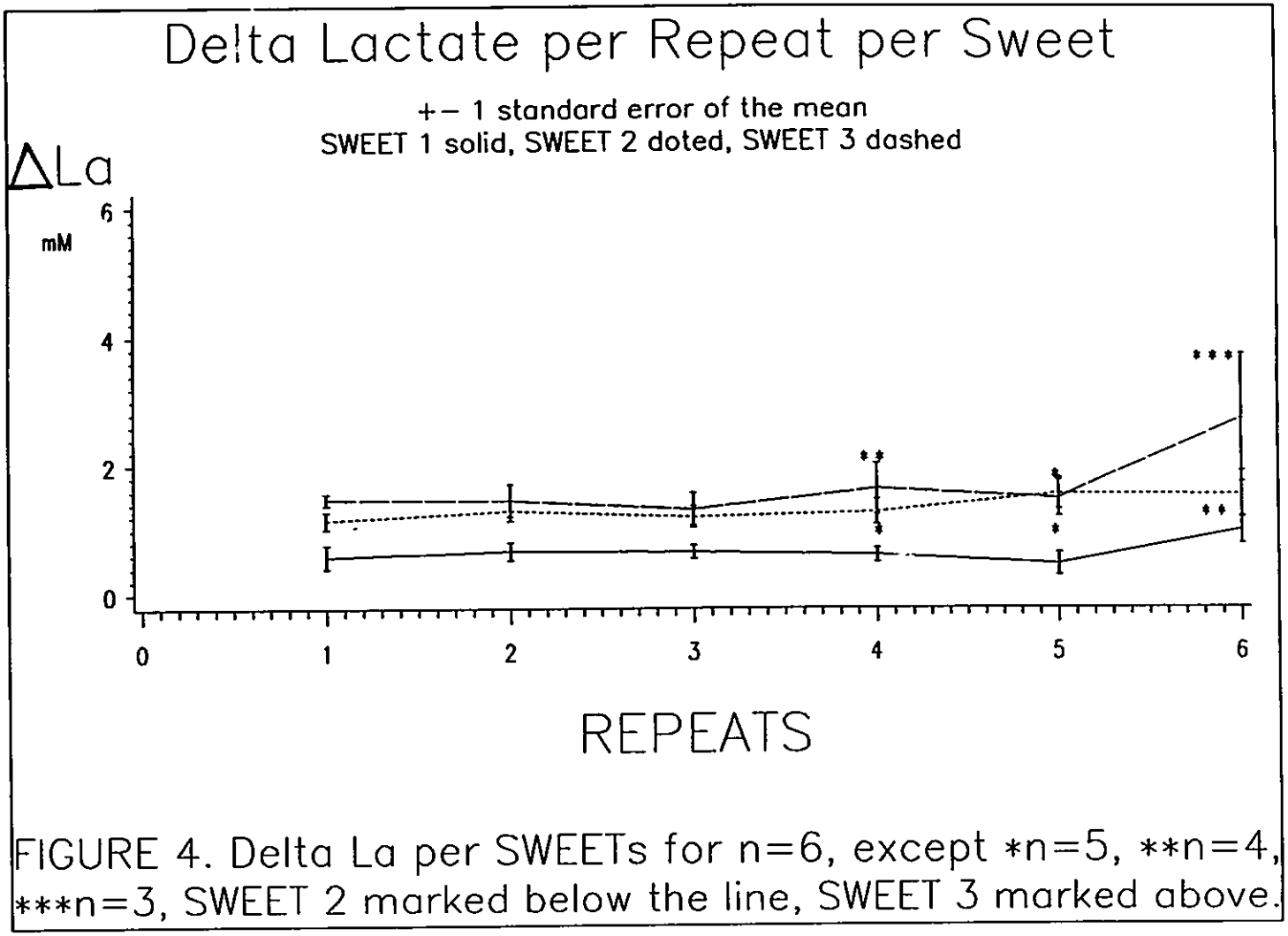

FIGURE 4. Delta La per SWEETs for n=6, except *n=5, **n=4, ***n=3, SWEET 2 marked below the line, SWEET 3 marked above.

Table 2

Descriptive statistics for [HLa], HR, $\dot{V}O_2$ pooled for subject data by SWEETs and stages. Means and SD are listed for each grouping of N.

Variable	(N)	OVER	(N)	UNDER	(N)	Δ(OVER-UNDER)
		Mean ± SD		Mean ± SD		Mean ± SD
HLa	35	2.33 0.66	36	1.69 0.52	36	0.66 0.06
HR	35	167 10	36	158 11		
$\dot{V}O_2$	35	54.8 5.89	36	47.4 5.41		
HLa2	33	3.87 1.02	31	2.52 0.80	32	1.31 0.45
HR2	33	179 7	31	165 11		
$\dot{V}O_2$2	33	62.1 4.13	31	51.2 3.79		
HLa3	28	6.43 1.51	30	4.73 1.23	30	1.58 0.79
HR3	28	182 7	30	164 11		
$\dot{V}O_2$3	28	65.6 6.40	30	50.6 4.24		

Notc: HLa, HR, $\dot{V}O_2$ = SWEET 1; HLa2 HR2, $\dot{V}O_2$2 = SWEET 2; HLa3, HR3, $\dot{V}O_2$3 = SWEET 3

Table 3

Pearson Product-Moment correlation coefficients for three SWEETs

for 6 subjects pooled for SWEETs and stages (OVER + UNDER).

ID=1 N=32				ID=2 N=25		
	LA	HR	$\dot{V}O_2$	LA	HR	$\dot{V}O_2$
LA		0.66361*	0.68548*	LA	0.58117*	0.55430*
HR			0.79157*	HR		0.68545*

ID=3 N=36				ID=4		
	LA	HR	$\dot{V}O_2$		LA	HR	$\dot{V}O_2$
LA		0.69501*	0.58747*	LA	0.76769* 35	0.64931* 33
HR			0.69256*	HR		0.63957* 34

ID=5				ID=6 N=36		
	LA	HR	$\dot{V}O_2$		LA	HR	$\dot{V}O_2$
LA		0.46900* 33	0.16181 33	LA	0.67839*	0.76239*
HR			0.63050* 32	HR		0.90445*

Note: * = significance at $p \leq 0.05$, ID = subject number, N = number of observations.

Table 4

Pearson product moment correlation coefficient for 3 SWEET's for 6 subjects pooled for subjects and stages (OVER + UNDER). r / Probability / N.

	HLa2	HLa3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
HLa	0.73699 0.0001 65	0.64692 0.0001 61	0.66937 0.0001 72	0.30903 0.0123 65	0.41052 0.0010 61	0.17519 0.1439 71	0.32194 0.0095 64	0.31129 0.0155 60
HLa2		0.63349 0.0001 61	0.54064 0.0001 65	0.44691 0.0002 65	0.51577 0.0001 61	0.47014 0.0001 64	0.46028 0.0001 64	0.62311 0.0001 60
HLa3			0.73435 0.0001 61	0.55202 0.0001 61	0.62229 0.0001 60	0.05117 0.6978 60	0.46220 0.0002 60	0.40165 0.0014 59
HR				0.76261 0.0001 65	0.75230 0.0001 61	0.11428 0.3426 71	0.44965 0.0002 64	0.24202 0.0625 60
HR2					0.91227 0.0001 61	0.34071 0.0059 64	0.56471 0.0001 64	0.42935 0.0006 60
HR3						0.22082 0.0900 60	0.58820 0.0001 60	0.47091 0.0001 59
$\dot{V}O_2$							0.64592 0.0001 63	0.77304 0.0001 59
$\dot{V}O_2$2								0.78970 0.0001 59

Note: HLa, HR & $\dot{V}O_2$ = SWEET 1; HLa2, HR2 & $\dot{V}O_2$2 = SWEET 2; HLa, HR3 & $\dot{V}O_2$3= SWEET 3.

subject or by SWEET show moderate positive correlation coefficients (r). Generally the correlation of HR and $\dot{V}O_2$ was greater than that for HLa and $\dot{V}O_2$.

An analysis of variance for intensity with three levels (S1, S2, S3) with six possible repeated measures on each factor (SWEET) revealed a significant main effect due to intensity for ΔLa (See Table 5). Post hoc analysis of this main effect showed the source of the significant difference between the means of SWEET 1 and 2 and 1 and 3, but not quite (p=.06) between SWEET 2 and 3 for ΔLa (See Table 6). The repeated measures analysis showed a significant within subject variance over the six repeats as shown in Table 7. No significant interaction between SWEETs and repeats were found. Post hoc analysis of repeated measures showed significant differences across the SWEETs as presented in Table 8.

Table 5

Summary of ANOVA for general linear model (GLM) analysis with SWEETs as independent variable and ΔLa as the dependent variable.

Source of Variation	df	SS	MS	F value	PR > F
SWEET	2	15.17	7.59	24.50	0.001*
ERROR	95	29.41	0.31		

NOTE:* significance $p < 0.05$.

Table 6

Matrix of GLM least square means post hoc analysis for differences between means.

SWEET	LSMEANS	1	2	3
1	0.66	--	0.0001*	0.0001*
2	1.31		--	0.0603
3	1.58			--

NOTE: * significance $p < 0.05$.

Table 7

Summary of GLM ANOVA with repeated measures for ΔLa repeats and interaction.

Source of Variation	df	SS	MS	Fvalue	PR>F
Repeats	5	5.02	1.00	4.21	0.0032*
Interaction (Repeats*SWEETs)	10	2.89	0.29	1.21	0.31
ERROR	45	10.75			

NOTE: * significance $p < 0.05$.

Table 8

General Linear Model probabilities for least squared means among repeated measures of ΔLa across SWEETs (intensity factor).

Repeats	LSMEANS	SWEET	1	2	3
ΔLa1	0.59	1	--	0.1148	0.0235*
	1.07	2		--	0.2376
	1.53	3			--
ΔLa2	0.67	1	--	0.0391*	0.0178*
	1.22	2		--	0.3772
	1.51	3			--
ΔLa3	0.65	1	--	0.0030*	0.0150*
	1.33	2		--	0.8649
	1.29	3			--
ΔLa4	0.60	1	--	0.0122*	0.0017*
	1.30	2		--	0.1003
	1.85	3			--
ΔLa5	0.46	1	--	0.0066*	0.0285*
	1.34	2		--	0.8733
	1.28	3			--
ΔLa6	0.98	1	--	0.3829	0.0209*
	1.53	2		--	0.0827
	3.11	3	--	--	--

Note: * significance, $p < 0.05$.

Discussion

The effect of SWEET protocols intended to be at below threshold (SWEET 1 = 2.5 mM), at threshold (SWEET 2 = 4.0 mM) and at above threshold (SWEET 3 = 5.5 mM) intensities on ΔLa were assessed for a group of six, male, elite cross-country runners uring treadmill exercise. The pattern of responses for three traditional physiological measures of intensity, namely [HLa], HR, and $\dot{V}O_2$, were also of interest and thus were described through correlations and graphical summaries.

The main finding of this study is discussed in the context of a square-wave intermitent exercise protocol with six repeated measures. The postulated mechanisms suggested as cause of the main effect of this study are given within the constraint of [HLa] values as a balance between HLa production and HLa removal and not a direct measurement of La removal ability. The arguments will focus on La production; changing La kinetics curves at three intensities based on a mathematical model of ΔLa responses, the effects of recovery exercise intensity on ΔLa, and the effects of various sampling sites on [HLa] response values. Following the main discussion are implications for the applied physiology of distance running, runners training prescriptions, future research suggestions and recommended procedures.

The main finding of this study, that ΔLa increased as SWEET intensity increased, cannot be directly linked to any one factor as a single cause. [HLa] is a direct reflection of the balance of La production and influx into the blood volume, minus La removal or utilization via efflux from the blood volume to oxidizing or

storage sites (Brooks, 1985; Åstrand, 1986).

The mean OVER [HLa]s remained elevated but constant at the target OVER levels for the six repeats of SWEET 1 and 2 (Figure 1) and were reflective of submaximal subthreshold to threshold work (Stegmann et al., 1981; Stegmann and Kindermann, 1982; Londeree, 1986). The SWEET 3 OVER [HLa] values appeared to increase slightly over time however this may be due to a decreasing n. The postulated mechanism responsible for the inability of the body to remove sufficient HLa during work and recovery periods can be linked to both metabolic and neural control factors.

The increased OVER [HLa] values for SWEET 3 can be attributed to a larger O_2 debt and consequently a greater amount of early La build up outpacing the removal amount (Cerretelli et al., 1979; Mazzeo et al., 1986). It is believed that mitochondrial ATP production cannot match the increased demand thereby stimulating glycolytic production of ATP. To accomplish this pyruvate is converted to La with NADH + H^+ oxidized to NAD via the lactate dehydrogenase (LDH)-H isoenzyme (Stainsby, 1986). Rieu et al. (1989) suggests decreasing [HLa] for transitions from light square-wave exercise to heavy square-wave exercise in contrast to those from rest to heavy square-wave exercise. This was evident in the patterning of [HLa] in the present study and might explain the lower [HLa]s at SWEET 1 and SWEET 2 OVER intervals.

The postulated mechanism for the increased OVER [HLa]s can be seen in the concurrent working of the neural and metabolic processes. The metabolic consequence of the oxidative capacity for ATP production lagging behind the energy production

needs of skeletal muscle is coupled with a gradual shift in the recruitment pattern of skeletal muscle fibre types along a prefixed order, resulting in augmented [HLa] levels. The fast twitch (FT) fibres recruited to accomplish the work at the higher velocities of running have metabolic profiles which facilitate La production from the LDH-M4 isoenzyme (Lehninger, 1982; Burke and Edgerton, 1975). The fixed order of recruitment proceeds from easy to recruit slow twitch (ST) and fast oyxidative glycolytic (FOG) fibres to more difficult to recruit FT fibres. ST and FOG fibres are characterized as fatigue resistant, innervated by smaller axon neurons needing relatively lower excitatory thresholds signals, and are responsive to lower frequency signals from the motor cortex. FT fibres are characterized as highly fatigable, innervated by larger axon neurons requiring relatively higher excitatory threshold signals, and are responsive to higher frequency signals from the motor cortex. This process of recruitment proceeds from small to large motor units and is referred to as the size principle (Henneman and Olson, 1965).

The gradual transition from predominantly aerobic work to anaerobic work was described in english by Heck et al. (1985) from the German work of Mader (1976)

> The aerobic-anaerobic threshold is crossed like all other biological processes gradually and not abruptly. The rise in lactic acid concentration to 4 mM in peripheral blood during gradual increases in workloads can be considered as the criteria for the establishment of the aerobic-anaerobic threshold in spiroergometric testing (p. 117).

For several minutes during each OVER interval for SWEET 3 there was an onset of HLa accumulation in the present study. The gradual nature of this process of shifting

from steady state work to work where HLa accumulation occurs is believed to be responsible for the gradual accumulations of SWEET 3 OVER [HLa]s.

The balance of HLa production and removal has been termed turnover (Brooks & Fahey, 1986). Tracer studies using continuous or bolus injections of labelled La have monitored La activity in the blood stream. Katz (1986) defines turnover as the ratio of the area under the curve of the specific activity of the tracer (at the sampling site) to the quantity of the tracer (the dose) injected as a bolus. Mazzeo et al. (1986) defines the La irreversible disposal rate in the same manner as turnover but warns that the values do not take into consideration the recirculated labelled La. For continuous infusion of the tracer the specific activity of La labelled carbons attain a plateau. The rate of turnover is then defined as equal to the ratio of the infused dose to the plateau of specific activity. Mazzeo et al. (1986) using a Na^{+}-L(+)-[1-^{13}C]lactate bolus found that La was continually turning over in the blood. They found that oxidation was the major fate of HLa during exercise, with oxidation rates linearly related to metabolic rate. Also found was that for any [HLa] the rate of La production and removal is several times greater during exercise than during rest. With this the authors concluded, in concurrence with Brooks (1985), Davis (1985), and Gladden (1989), that a single [HLa] value is not directly related to La production or removal but indirectly related to the balance of the two processes. Therefore changes in [HLa] cannot be directly linked to a single event like production or removal.

Careful control of intervening variables that influence La production like diet

(Maassen and Bussec, 1989), relative workload intensity (Bassett et al., 1991), workload duration, and relative recovery intensity (McLelland and Skinner, 1982; Hermansen and Stensvold, 1972), justifies attributing changes in [HLa] to La recovery aspects of aerobic capacity (Heck et al., 1985). Within this context it is the effect of the mechanisms of La removal that are indirectly reflected in [HLa] changes during work and recovery periods and thus provides a new physiological definition of aerobic endurance capacity. An examination of the physiological mechanisms that might be responsible for the ΔLa results demonstrated in this study follow below.

One plausible explanation for ΔLa changes, applying the work of Freund et al. (1984) is that La kinetics are changing from SWEET 1 to SWEET 3 suchthat a more rapid blood lactate recovery is occurring at progressively higher SWEETs. These La kinetics changes are seen as [HLa] dependent and time dependent in that a shorter recovery times are associated with higher concentrations in submaximal SWEET protocols.

The postulated mechanisms for the ΔLa values observed have both a rapid and a slow component. The rapid effect is due to La or lactic acid equilibrating or sinking down relatively larger concentration gradients. La moves from producing compartments to storage or utilization compartments where La uptake can be either active oxidation or a passive sink (Freund et al., 1984; Åstrand, 1986; Gladden, 1989). The longer duration effect is due to continuing metabolic clearance, via oxidation, of La by aerobic tissues like heart, liver, and active oxidative skeletal muscle (Åstrand,

1986).

The model of Freund et al. (1978) represents the most complete mathematical model of [HLa] recovery kinetics explaining 99% of the variance based on two components of recovery or a biexponential function. This function describes arterial [HLa] during recovery

$$La(t) = La(0) + A_1(1 - e^{-\gamma_1 t}) + A_2(1 - e^{-\gamma_2 t})$$

where γ_1 and γ_2 are the velocity constants of the decreasing exponential functions, and A_1 and A_2 are the amplitudes of this function. La(t) and La(0) are the arterial [HLa]s at time t and at the onset of recovery respectively. The γ_1 velocity constant represents both mathematically, the increasing [HLa] curve, and functionally, the ability of the muscle to exchange La between active muscle groups and venous or arterial blood. Whereas the γ_2 velocity constant represents both mathematically, the decreasing [HLa] curve, and functionally the ability of the system to remove La via metabolism (Freund et al., 1989).

ΔLa increased significantly from SWEET 1 to SWEET 2, but the SWEET 2 to SWEET 3 increase fell just short ($p=0.06$) of the criterion ($\alpha=0.05$). Taking into account the small sample size ($n=6$), judgement must be reserved regarding the trend of mean ΔLas increasing with SWEET intensity. Figure 4 shows a trend for the Mean ΔLa values, in absolute terms, to increase with increasing SWEET intensity (100% increase from SWEET 1 to SWEET 2 intensity levels, but only a 50% increase from SWEET 2 to SWEET 3 intensity levels). The relative slowing of the ΔLa ability when

comparing the SWEET 2 to SWEET 3 ΔLas might be attributed to the limitations of the transport of La or lactic acid across tissue membranes. This slowing of the metabolic clearance of L^- and H^+ ions has been found in other studies. Gladden (1989) reviews lactate uptake by skeletal muscle and cites evidence of a carrier mediated transport system that limits lactate recovery ability in the first 0-5 minutes of a La stress. This short time period represents the individual active recovery period (UNDER) during the three SWEETs that were used in this study.

The contribution of the carrier mediated transport is estimated at 50-75% of the total lactate uptake. There are four postulated mechanisms for La transport of which three are carrier mediated. They are one, La ionic (anion) diffusion that occurs mainly through membrane pores, two, a bicarbonate linked anion exchange of lactate for bicarbonate, and three, a H^+-symport carrier mediated proton linked cotransport of H^+ and L^- (Mainwood et al., 1987; Gladden, 1989). Lactate ionic diffusion, a passive process most likely affected by H^+ ions, was not likely during La uptake because La would have to be actively transported against its electrochemical gradient. Antiport bicarbonate exchange has been discounted as a factor by Mainwood et al. (1975) because La efflux has been shown to be independent of external buffers. The H^+ symport carrier system is considered to be indistinguishable from an OH^- antiport lactate carrier because cotransport of H^+ and La cannot be distinguished from countertransport of La and OH^- in opposite directions.

Jorfeldt et al. (1978) examined muscle La levels in association with arterial-

venous differences (a-v) across the femoral vasculature. They found a linear increase in La versus La_{a-v} until 4 mM/kg wet weight after which a-v plateauing resulted in a La release ceiling of 4-5 mM/min. The saturation of the translocation protein mechanism was cited as the cause of the levelling off of the La release.

The limitations of La release due to saturation of the membrane carrier can be responsible for slowing both the release and uptake of La, that is La produced in FT fibres and utilized in ST fibres. Membranes crossed before oxidation of HLa include: one, those between the vascular volume to the interstitial space of ST (oxidizing tissue) fibre; and two, the muscle membrane and the mitochondrial membrane to oxidation sites (Gladden, 1989). After considering membrane transport the rate of La uptake, as mentioned above, has been attributed to both a passive sinking of La into tissue fluids as well as an oxidation of La as a fuel.

Continued increases in mean ΔLa from SWEET 1 to SWEET 2 to SWEET 3 responses in this study might be due to a continuously rising La irreversible disposal rate (Mazzeo et al., 1986) or La turnover rate (Katz et al., 1986). La oxidation was determined during these same tracer studies as the principle removal pathway of La (Brooks, 1986). Disappearance has also been attributed to a continued passive La sink or equilibriation of [La]s from higher blood concentrations to lower tissue concentrations by Freund et al. (1989). La sink was due to the concentration gradient that remained high and continued to drive the translocation mechanism of La, most probably the free diffusion of undissociated lactic acid (Gladden, 1989).

Freund and Zouloumian (1981) found that γ_1 and γ_2 were lower after maximal than submaximal exercise performed on a bicycle. Freund et al. (1989) examined the mean velocity constants γ_1 and γ_2 after 3 versus 6 minutes of cycling and after 3 versus 60 minutes of continuous cycling. Additionally nonsignificant but lower velocity constants at easy exercise (39-50% $\dot{V}O_2max$) and moderate exercise (52-67%$\dot{V}O_2max$) versus hard exercise (76-82%$\dot{V}O_2max$) were found for the longer workload experiment. When the workload duration was increased as in the Freund et al. (1989) study decreases of 25 to 30% for γ_1 were observed. This represents a decreased functional ability of the tissues to exchange lactate between previously exercised muscles and the arterial blood and/or the remainder of the space where La is distributed. Decreases of 15 to 19% for γ_2 were also reported for workload duration increases. This represents the functional ability of the body to remove La via metabolism. In the Freund et al (1990) study increases in workloaad duration to 60 minutes showed a significant slowing of the γ_2 component, 28% versus a nonsignificant slowing of the γ_1 component, 10%. In contrast, no decrease in ΔLa was seen with repeat 3 minute bouts over the time course of this experiment.

The following are studies that show the relationship between increased ΔLa values and increased metabolic rate. Mazzeo et al. (1986) studied La irreversible disposal rates and oxidation rates during cycling and found both oxidation rates and irreversible disposal rates were significantly increased from rest to easy exercise (50% $\dot{V}O_2max$) but not significantly increased from easy to hard exercise (75% $\dot{V}O_2max$).

This concurs with the pattern of ΔLa increases in this study. The rates of irreversible disposal and oxidation were both linearly related to the metabolic rate in the Mazzeo et al. (1986) study.

The continuously increasing oxidation rate of La might be attributed to increasing HLa removal as a fuel for ST and FOG fibres that are recruited at SWEET 1 and SWEET 2 intensities. Brooks (1986) sees this process of "shuttling" of oxidizable substrate (La) from areas of high glycolytic activity to areas of high cellular respiration as an efficient coordination of tissues during exercise. The relative overproduction of La by small and medium FT fibres at the beginning of exercise is believed to be compensated for by the relatively increased utilization of La by oxidative tissues such as ST and FOG skeletal muscle fibres, liver, heart, kidney and others (Mazzeo et al., 1986; Brooks, 1985; Brooks, 1986). Åstrand (1986) attributes 10% of La removal during rest recovery to liver, 40% to oxidizing skeletal muscle, with the remainder (50%) stored in muscle tissue as glycogen. Brooks (1986) cites the percentage of turnover due to oxidation as 43% during rest and as increasing to 74% during execise.

The mechanism of La oxidation is controlled by the A4 or H form of the lactate dehydrogenase isoenzyme. This enzyme is active in oxidative tissues converting La to pyruvate for further aerobic catabolism via the pyruvate dehydrogenase enzyme complex in the mitochondrial membrane and the Kreb's cycle (Lehninger, 1982). In contrast to the biochemical control of La utilization the control

of the relative workload in the UNDER interval also affected La utilization in this study.

The contrasting of ΔLa both within and between SWEETs was based on one vital assumption in the experimental design, that recovery intensities (UNDERs) were relatively similar and represented net La removal workloads. The relationship between HLa recovery and exercise intensity or metabolic rate has been investigated with the purposes of setting an optimal recovery pace and to examine La oxidation. In this context several authors have found slightly varying results.

Belcastro and Bonen (1975) found the highest metabolic rate for optimal La removal at 17-49% $\dot{V}O_2$max for relatively lower fit subjects while cycling. Stamford et al. (1981) corrected for baseline [HLa] values and found when plotted semilogrithmically 40% and 70% anaerobic threshold recovery intensities produced parallel decreasing lines representing equal La removal rates.

In contrast to cycling exercise Hermansen and Stensvold (1972) tested high fit runners on a treadmill and found optimal HLa removal at 63% $\dot{V}O_2$max with a 2 mM per min. rate of decline for [HLa] after three one minute bouts of maximal running. These findings were greater than the mean ΔLa found in this study for SWEET 3 at 1.58 mM yet the findings of Hermansen and Stensvold (1972) were the only values reported in the La removal literature for very high fit subjects similar to the subjects of this study. None of the athletes achieved a ΔLa that when calculated as a rate equalled the HLa removal rate of Hermansen's study. It appears [HLa] removal amounts may

be linked to concentrations.

Hermansen and Stensvold (1972) also found that during incremental exercise no rises in HLa were reported until 63% $\dot{V}O_2$max was reached. This corresponds to McLelland and Skinner's (1982) findings that optimal [HLa] removal was found to occur just below the aerobic threshold when it is defined as the first rise of [HLa] above baseline. Gladden (1989) confirms that any drop in endogenous La promotes La utilization by allowing entry and oxidation of exogenous La. The fact that the recovery intensities were low enough in this study to allow net La uptake and oxidation was evident in the patterning of the [HLa] values where clear drops in La occurred for every UNDER interval. Blood lactate recovery ability did not decrease for repeated intervals in contrast to other studies.

Peripheral [HLa] analysis of mixed venous-arterial whole blood, as a measure of intensity, can be compared and contrasted to other methods cited in the literature. [HLa] was chosen as the key dependent variable because of its high correlation to peripheral fatigue. This is due in part to the effects of the hydrogen ion (H^+) of the dissociated acid acting on glycolysis, the contractile process itself, and certain key equilibrium reactions in the energy transduction pathway of skeletal muscle (McLaren et al., 1989). Also because La^- and H^+ ions are consumed in equimolar concentrations, La recovery can be an indirect measure of decreasing [H^+] (Gladden, 1989). The observed effects will be above and beyond the La^- and H^+ ions produced that are buffered by inorganic phosphate, protein-bound histidine residues, the dipeptide

carnosine, bicarbonate and creatine phosphate (Parkhouse and McKenzie 1984).

The limitations upon [HLa] analysis must be placed in context with other intrusive methodologies. Direct measurement of muscle [La] was inappropriate for 12 samples per test, therefore peripheral blood samples were used as a quick, minimally intrusive and relatively inexpensive method for determination of [HLa] (Jacobs, 1986) that was considered both reliable (±0.05 mM) and valid (±0.1 mM) (Geyssant et al., 1985). The limitations imposed by the frequency, the sites of sampling, the number and the timing of the sampling procedure limit investigators to peripheral [HLa] analysis during SWEET protocol both because of the minimal volumes that can be extracted and the previously mentioned ease.

Within this context arterialized-venous bloods contain a higher [La] than deep venous bloods for 3 minutes of constant load work (Orok et al., 1989), yet less than muscle [La] (Green et al., 1983). Jacobs and Kaiser (1982) found a muscle [La] to [HLa] ratio at OBLA intensities of 2.3. Oyono-Enguelle et al. (1989) examined arterial, arterialized-venous and venous La kinetics to compare each to a previously constructed model (Freund et al., 1986) that explains 99% of the variance in La kinetics during recovery. They found the most sensitive method for [HLa] determination used arterial La samples with arterialized venous samples not quite as sensitive.

HRs tend to drift upwards during the SWEETs for both OVER and UNDER intervals of work. In contrast the $\dot{V}O_2$s are stable at OVER and UNDER intervals.

[HLa] patterns as previously described are stable for SWEET 1 and SWEET 2 repeat intervals, but are slightly increased for SWEET 3 OVER and UNDER intervals. Test termination is usually preceded by sharp rises in [HLa] versus plateauing of $\dot{V}O_2$ or HR values in this study. Although body temperautre was not measured in the present study one explanation for the upward drift in heart rate over time was an accumulating heat stress.

The moderate positive correlation coefficients of HR, [HLa], and $\dot{V}O_2$ make HR and $\dot{V}O_2$ inadequate predictors of [HLa] (see Appendix G). Although [HLa]s, like HRs and $\dot{V}O_2$s, are generally related to intensity, in response to progressive incremental exercise intensities [HLa] values rise at first curvilinearly and then exponentially. Increasing [HLa] values do not show the same linear relationship to increased exercise intensity that HR and $\dot{V}O_2$ values show (Astrand, 1986). [HLa] value increases remain relatively lower longer for progressive incremental exercise tests before rising rapidly for elite athletes versus average or untrained subjects (Heck et al., 1985; Sjödin and Svendenhag, 1985; Macdougall et al., 1991).

The implications of ΔLa increasing with SWEET intensity are that as [HLa] increased from below threshold to threshold levels, with increased treadmill speed, so did the althletes ability to remove HLa. This trend continued for further increases in speed from threshold levels to suprathreshold levels although increased [HLa] were only partially compensated for by increased ΔLa rates. This is in contrast to little change in HR and $\dot{V}O_2$ values for increases in exercise intensity from SWEET 2 to

SWEET 3 levels. Runners compensated for increased intensity by increasing ΔLa. Within the six subjects in the present study considerable differences exist as to how many repeats were possible at SWEET 3, a running pace above their OBLA threshold. Racing strategies for speed surges or difficult climbs would have to take this information into account for each individual. This is where differentiation of homogeneous groups of athletes can best be seen.

The athlete with the best performance time for a 10 kilometre race had the most stable [HLa] responses for SWEET 3 OVER intervals. Subject six's ten kilometre personal best running time qualify him as a world class runner. His running speeds were considerably higher at the three SWEET intensities (See appendix B). As well this subject maintained a stable level of OVER (approximately 6 mM) and UNDER (approximately 4 mM) [HLa] for the six SWEET 3 repeats. In contrast other subjects, not all of whom finished six repeats, had slightly rising [HLa] values during the SWEET 3 protocol. Four subjects with similar performance scores had slightly increasing [HLa] values from approximately 3.5 to 8.0 mM during the SWEET 3 protocol. The subject with the lowest performance score had a sharp sixth repeat [HLa] value increase to 12 mM. Further study of both the responses of world class runners to SWEET testing and the effects of moderate [HLa] intensities around OBLA threshold on ΔLa is warranted.

Other implications for athletes involve training prescriptions for moderate intensity intervals at [HLa]s of 2.5, 4.0 or 5.5 mM. If an athlete can only complete

three 5.5 mM level intervals without fatigue then an extended active rest period corresponding to half recovery times may be in order to allow [HLa] to equilibrate with muscle [La] but most importantly for [La] levels in general to decrease. This is possible because circulating HLa is now seen as a fuel that can be used by active skeletal muscle during exercise (Brooks, 1986; Brooks, 1990). Thus premature fatigue attributable only to metabolic accumulation may be avoided and a greater training volume could be accomplished. Alternate training strategies may be to extend rest periods between more intense intervals if La tolerance is not the goal of the training. If the overload stress is that of HLa removal then sufficient time should be given to diminish this before the next stress is administered.

Workloads identified from the progressive incremental tests were not easily translated into 3 SWEET intensities elicited by treadmill speeds of no less than 1 km x h^{-1} differences. Appendix B contains the projected versus the actual speeds used. Progressive incremental tests of 3 minute durations do not accurately predict SWEET [HLa] on an individual basis -- although with slight adjustments in protocol speeds the means [HLa] values were within ±1 SE of the target intensities.

Rieu et al. (1989) has identified an intermittent progressive incremental test of 4 minute duration followed by 1 minute rest periods, similar to Gimenez et al.'s (1982) original SWEET protocol. The authors found [HLa] responses from progressive incremental tests did equate to isolated constant load tests of the same intensity and duration. This procedure may better identify target SWEET relative intensities for

fixed [HLa]s.

SWEET testing appears to be a more sport specific aerobic endurance capacity test for cross-country than progressive incremental tests or one speed threshold (OBLA) tests. This can be attributed to not only a closer simulation of speed or interval training workout patterns of elite runners but also to a closer simulation of the varying intensities of certain hilly cross-country or road courses. Thus Square-Wave Endurance Exercise Test protocols are recommended for elite athlete aerobic endurance capacity testing.

The findings of this study were that as SWEET intensity increased from below threshold to OBLA threshold intensity ΔLa increased significantly for all repeats, whereas as SWEET intensity increased above OBLA threshold ΔLa trended upwards for five of the six repeats but were not significantly increased. The patterning of the three traditional physiological markers of intensity, being HR, $\dot{V}O_2$, and [HLa] when correlated were moderately positive yet were inadequate when used singly as predictors of the other markers -- thus [HLa] was a unique indicator of intensity.

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Appendix A

Thesis Research Project Consent Form

for Robert A. Séguin, B.P.E., B.Ed., C.F.A.
Phone: home (613-741-4223), office (613-564-5946)
Office Montpetit 302
Supervisor Dr. Alfred Reed,
School of human Kinetics
(613-564-9123)

CONSENT FORM

For the 'Lactate Verification Test' and three repeats of the 'Square-Wave Endurance Exercise Test'.

Studies involving human subjects require written informed consent of the participants. I _____, authorize Robert Séguin (741-4223) of the School of Human Kinetics to conduct a Lactate Verification Test and three repeats at three different intensities of the "Square-Wave Endurance Exercise Test (SWEET)".

THE PURPOSE OF THIS STUDY

The purpose of this study is to measure the total time of cumulative exercise as well as three physiological variables, namely heart rate, oxygen consumption and blood lactate concentration, over three SWEET applications at three different treadmill speeds.

Tests will be carried out in laboratories on the third floor of Montpetit Hall.

When you run on the treadmill the amount of oxygen that you utilize can be calculated by measuring the air you inhale and exhale. For gas analysis a low resistance tubing will be attached to you via a mouth piece, a one way valve, and a nose clip. Heart rate will also be recorded from the light weight chest halter and wrist receiver that you will wear. There should be minimum discomfort during the above two procedures. Finally blood samples will be taken from your outstretched hand approximately every three minutes to determine the level of metabolic fatigues you will experience during the tests. This procedure involves the pressing of a precision micropuncture lancet against your outstretched fingertip as it is held firmly in place by a trained technician. Approximately two drops of blood will be drawn into a capillary tube followed by the application of a nonlint sterile wipe. 3M surgical tape will keep the finger clean between and after sampling. There will be a slight discomfort associated with this minimally intrusive fingertip micropunture technique.

Although you will be undergoing exercise that might lead to temporary exhaustion, there is little risk involved if you are a normal healthy well trained runner. The treadmill will start at a slow seed and increase in speed gradually. If you stumble or fall, the treadmill will be stopped immediately and the consequences will be similar to the ones experienced if you had fallen on the track.

Day one will be for the "Lactate Verification Test". Day three, five and seven will be for the "SWEET" tests. Testing will occur every other day, always with one day off between tests.

All the tests will involve a pretest stretching period, a 9 minute gradual warm-up, and 3 minute intervals of running followed by approximately 3 minutes of active recovery for a total test time not exceeding 60 minutes. The intensities are similar to those experienced weekly in your training workouts.

I, _____, acknowledge by my signature below that I have been informed of, and had the opportunity to ask questions about the "Lactate Verification Test" and the "SWEET" tests. I understand that I am expected to participate in activities that involve moderate levels of physical discomfort, and that may cause headache, nausea, dizziness, fatigue, metabolic disruption, muscle stiffness and soreness, and, in rare cases vascular-centred problems. I am a healthy well-trained runner who has had an annual medical examination. I further understand that it is my responsibility to inform the laboratory personnel of any illness, infection, or other condition, which would prevent me from fully participating in this session. I also understand that I may withdraw at any time for any reason if I so choose without any adverse consequences to my team standing, to my academic standing, or adverse consequences of any kind.

NOTE: Results are confidential and are reported back to the subjects by individual reports.

_____, _____

(signature) (date)

_____

(witness)

Appendix B

Table B1. Projected workloads from the progressive incremental tests versus actual workloads.

Subject #	Speed of 1st rise in [HLa] with [HLa]	Speed interpolated from progressive test ($km \cdot hr^{-1}$) / Speed used ($km \cdot hr^{-1}$)		
1	14/1.5	16.5/17	17.5/18	18/19*
2	14/1.4	15/16	16.5/17	17/18*
3	12/1.8	14.5/15	16/16	16.5/17*
4	14/1.4	16.5/16	17.5/17	18/18*
5	14/1.22	15/15	16.5/16.5	17/19**
6	15/1.26	17/17	18/19.5	19/21**

Note: *Speed adjusted so that SWEETs at least 1 kmh apart in intensities.

**Speed adjusted when responses too low for target intensities from progressive test.

Appendix C

Calculation tables of (1), treadmill speed, time and distance covered in 3 minutes, and (2), time and distance covered in OVER and coresponding UNDER intervals for all SWEETs.

Sweet protocol displayed graphically.

Table C1. A calculation table for treadmill speed, millivolt readings, and distance covered in 3 minute intervals (OVERs) used to calculate equal distance running times at recovery interval speeds (UNDERs), such that OVER and UNDER distances were equal.

Treadmill Speed Settings.							
Speed km·hr^{-1}	milli-volts mV	Speed m/min	3 min distance (m)	Time for equivalent distance at (km·hr^{-1}) 12	13	14	15
10	3.33	166.67	500.01				
11	3.66	183.34	550.02				
12	4.00	200.01	600.03				
13	4.33	216.67	650.01	3'15"			
14	4.66	233.34	700.02	3'30"	3'14"		
15	5.00	250.01	750.03	3'45"	3'28"	3'13"	
15.5	5.17	258.33	725.00	3'53"	3'35"	3'19"	
16	5.33	266.67	800.01	4'00"	3'41"	3'26"	
16.5	5.40	274.67	825.00	4'04"	3'48"	3'32"	
17	5.66	283.34	850.02	4'15"	3'55"	3'38"	3'24"
18	6.00	300.0	900.0	4'30"	4'09"	3'52"	
19	6.33	316.67	950.01	4'45"	4'23"	4'04"	
19.5	6.50	324.97	975.0	4'53"	4'30"	4'11"	3'54"
20	6.66	333.34	1000.02	5'00"	4'37"	4'17"	
21	7.00	350.01	1050.03	5'15"	4'51"	4'30"	4'12"
22	7.33	366.67	1100.01	5'30"	5'05"	4'43"	

Table C2. Time and distance covered during SWEET OVER and UNDER intervals to demonstrate equality of distance control. The 3 minute distance run in the OVER interval is also run in the UNDER interval but taking a longer time: see the final column Time in minutes for the UNDER interval time.

Subject #	SWEET #	OVER Velocity m/min / km/hr		OVER 3 minute distance in metres	UNDER Velocity m/min / km/hr		UNDER Time min.
1	1	283.3	17	850.0	233.3	14	3'38"
	2	300.0	18	900.0	233.3	14	3'52"
	3	316.7	19	950.0	233.3	14	4'04"
2	1	266.7	16	800.0	233.3	14	3'26"
	2	283.3	17	850.0	233.3	14	3"38"
	3	300.0	18	900.0	233.3	14	3'52"
3	1	250.0	15	750.0	200.0	12	3'45"
	2	267.7	16	800.0	200.0	12	4'00"
	3	283.3	17	850.0	200.0	12	4'15"
4	1	266.7	16	800.0	233.3	14	3'26"
	2	283.3	17	850.0	233.3	14	3"38"
	3	300.0	18	900.0	233.3	14	3'52"
5	1	250.0	15	750.0	233.3	14	3'13"
	2	274.7	16.5	825.0	233.3	14	3'32"
	3	316.7	19	950.0	233.3	14	4'04"
6	1	283.3	17	850.0	250.0	15	3'24"
	2	325.0	19.5	975.0	250.0	15	3'54"
	3	350.0	21	1050.0	250.0	15	4'12"

Appendix D

Table D1. Raw data.

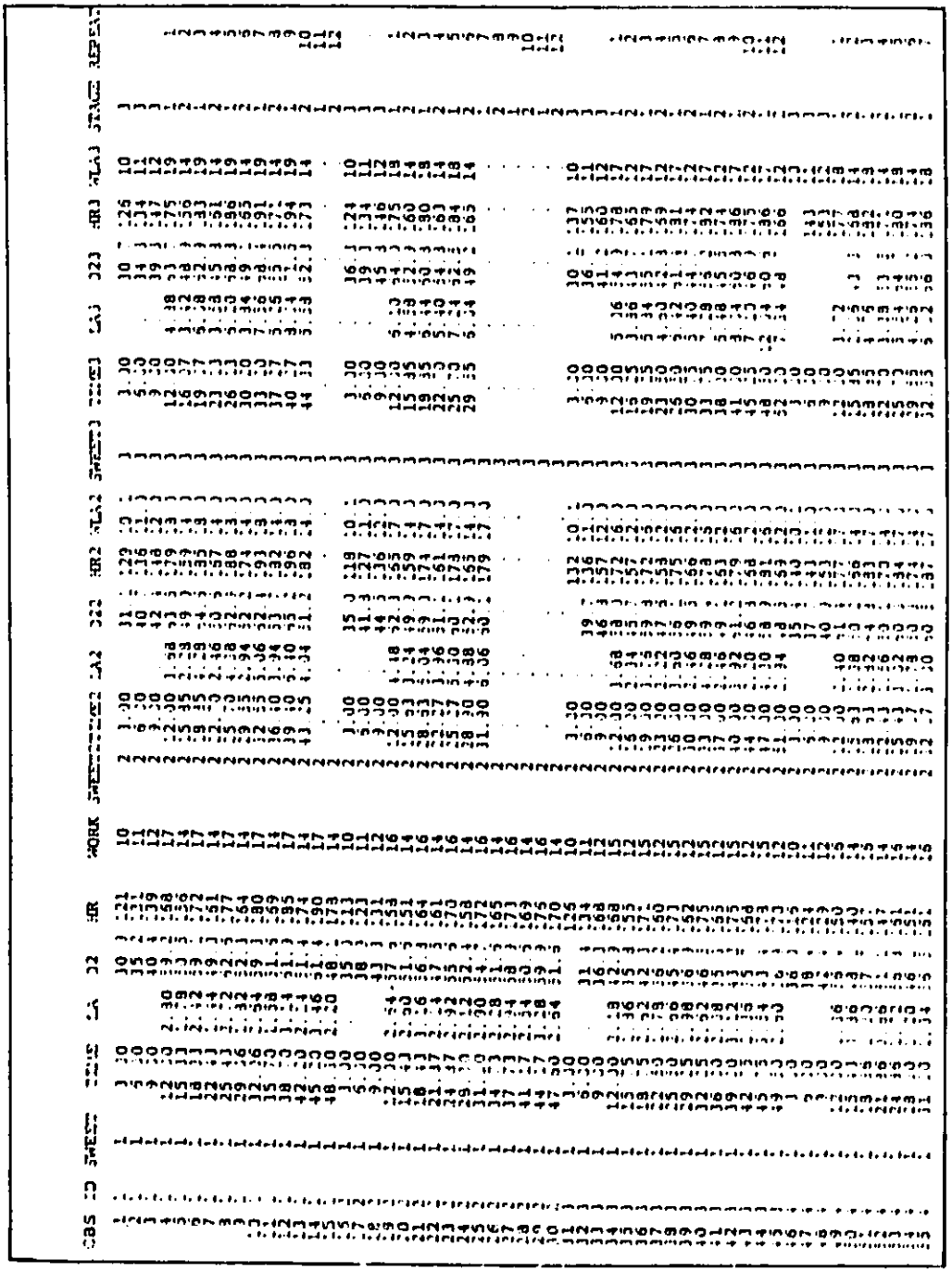
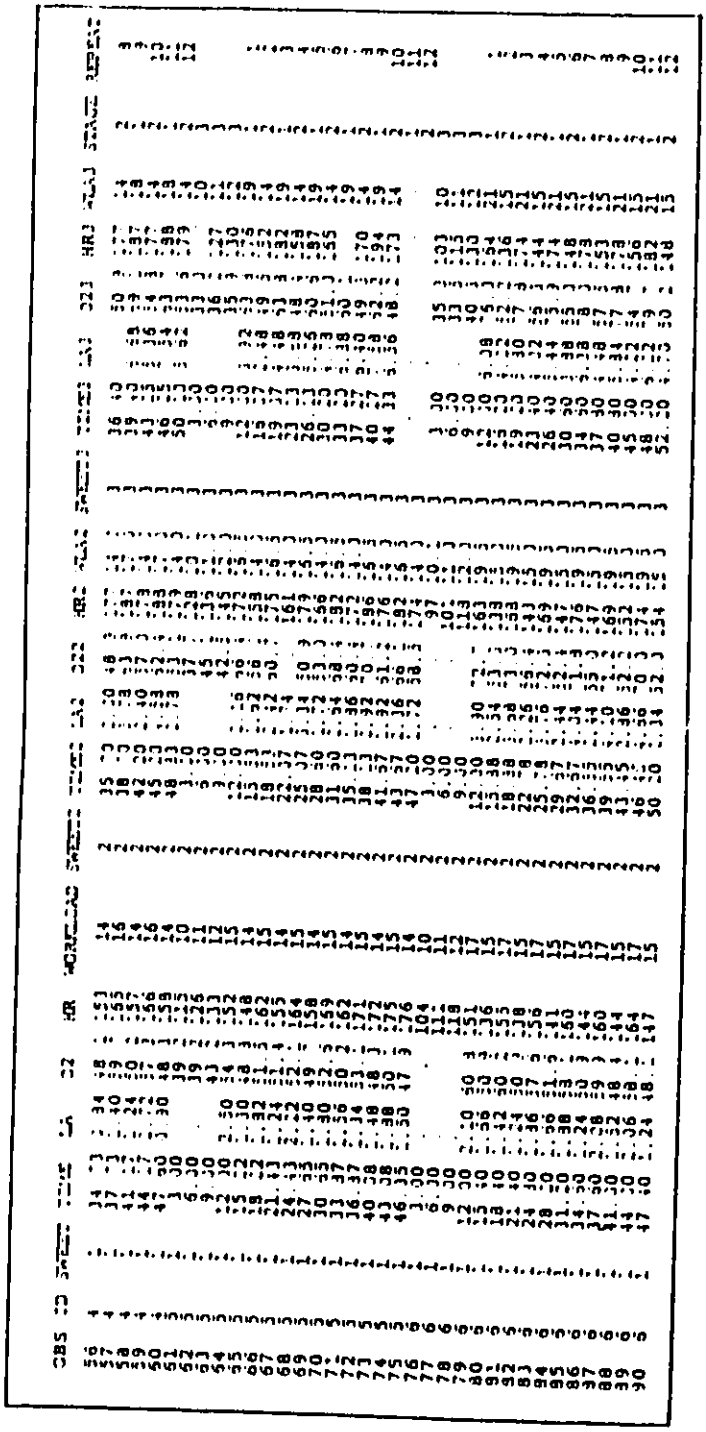


Table D2. Raw data continued.



Appendix E

Graphical summaries of the pattern of heart rate, oxygen consumption, blood lactate, and Delta La (ΔLa) during three Square-Wave Endurance Exercise Tests (SWEETs) set such that OVER workloads of three minutes should produce blood lactate concentrations of 2.5, 4.0 and 5.5 mM whereas the UNDER workloads are of equal distance run. (Subjects 1 to 6).

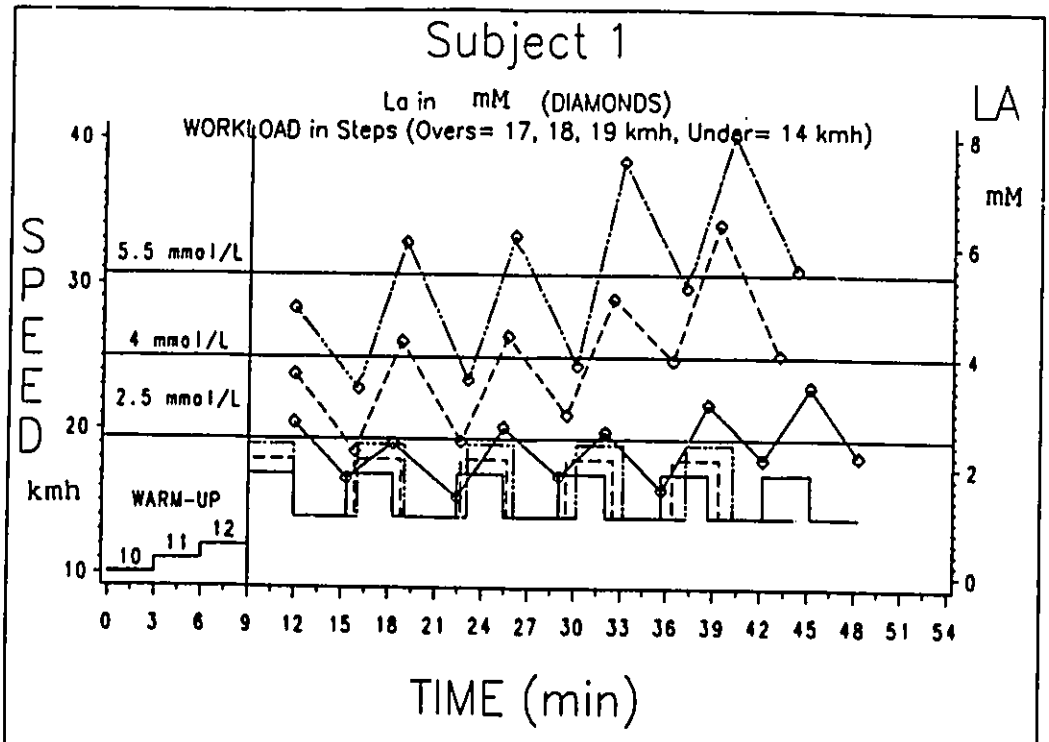


FIGURE 1. La for SWEET 1 (solid), 2 (dashed), & 3 (mixed).

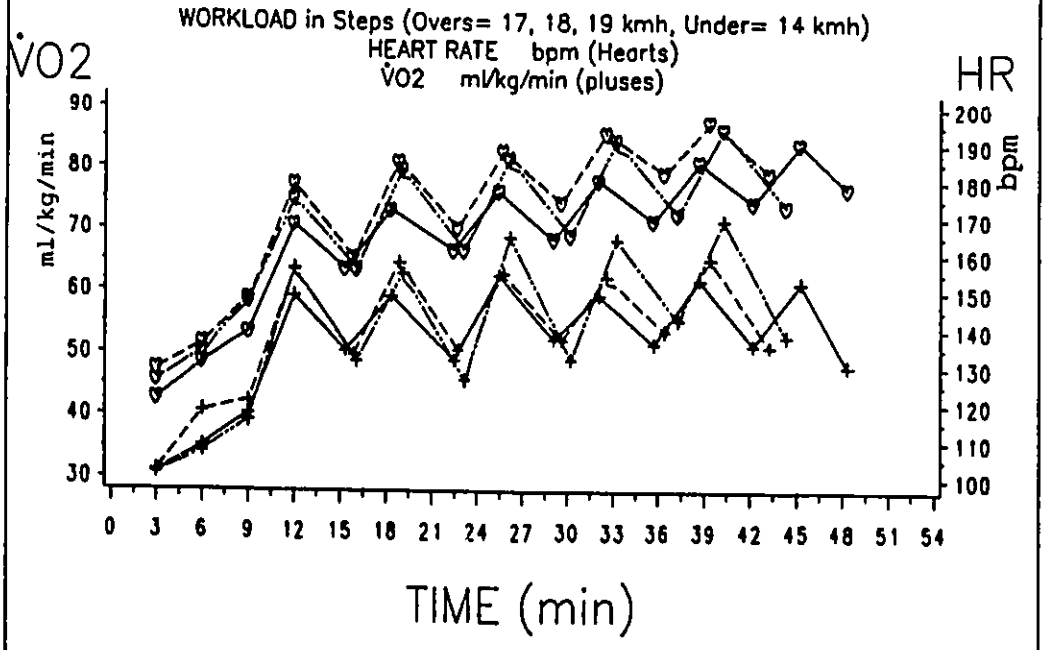


FIGURE 2. HRs and V̇O2s for 3 SWEETS.

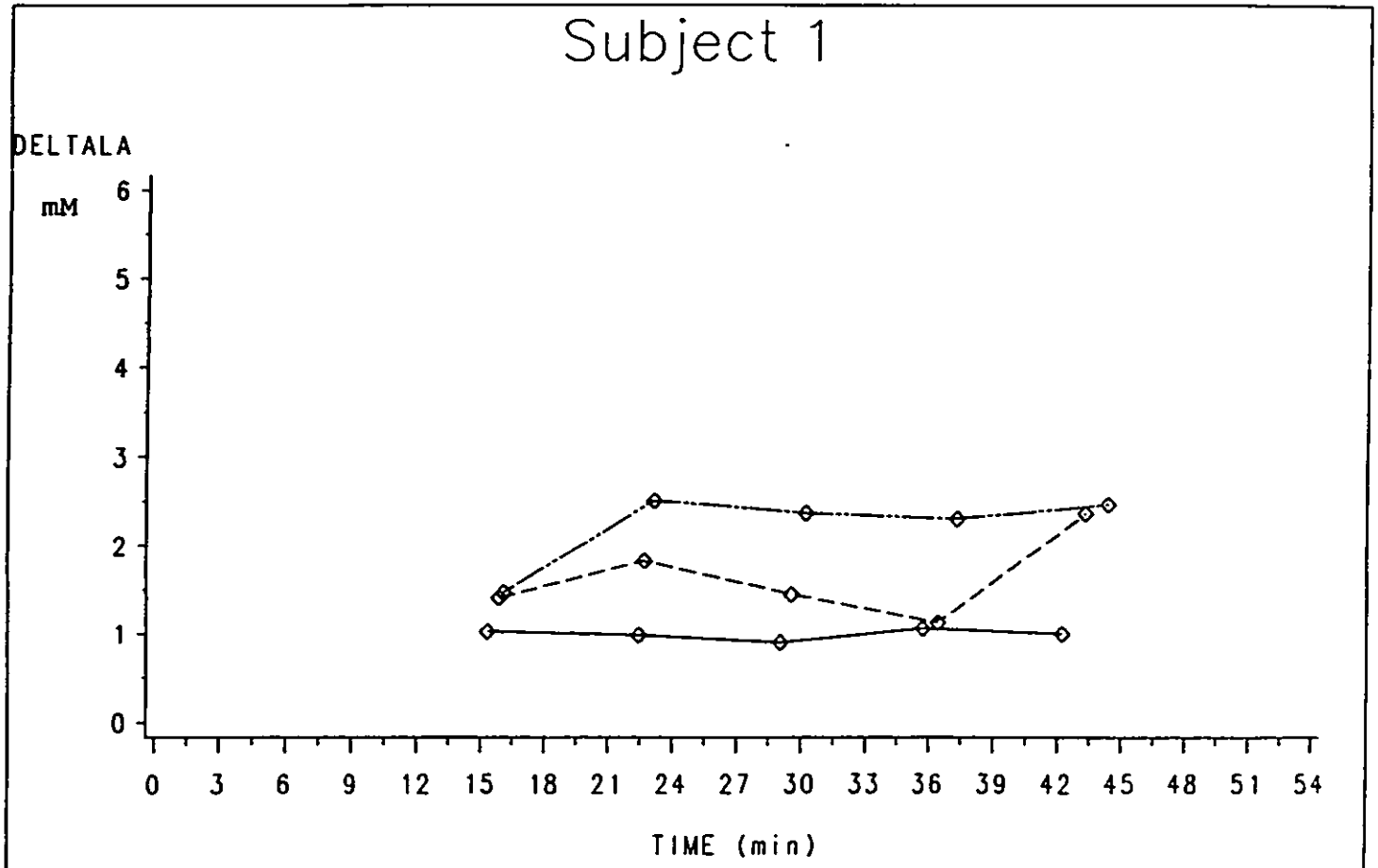


FIGURE 3. DeltaLa for SWEETs 1(solid), 2 (dashed), 3 (mixed)

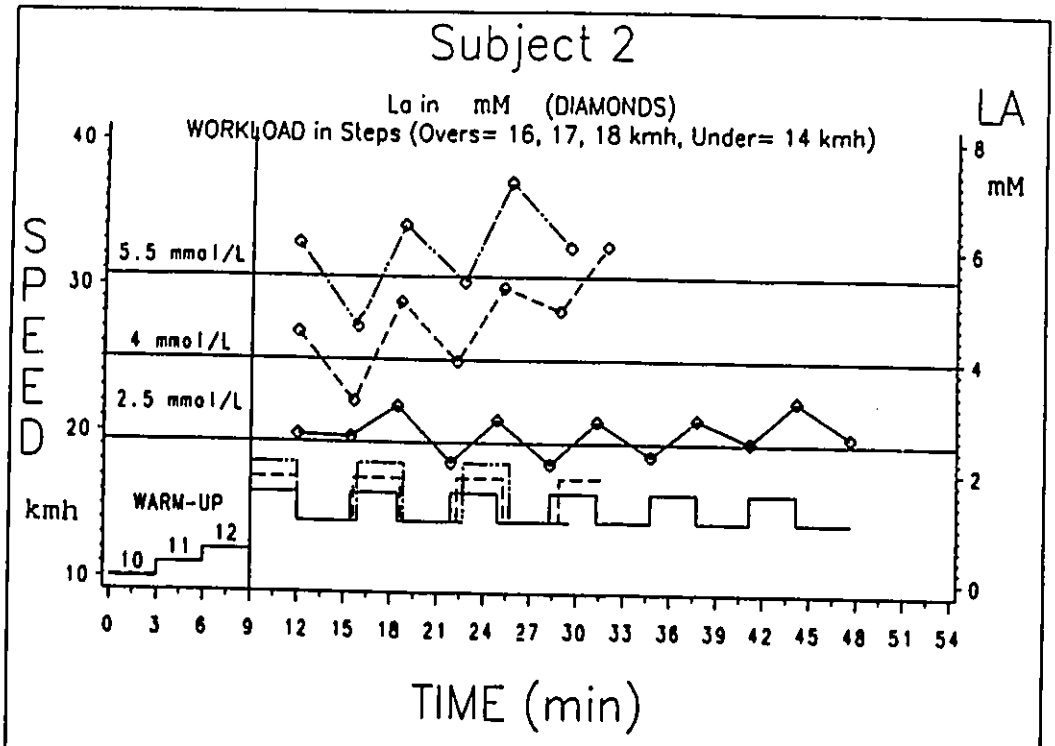


Figure 4. La for SWEET 1 (solid), 2 (dashed), & 3 (mixed).

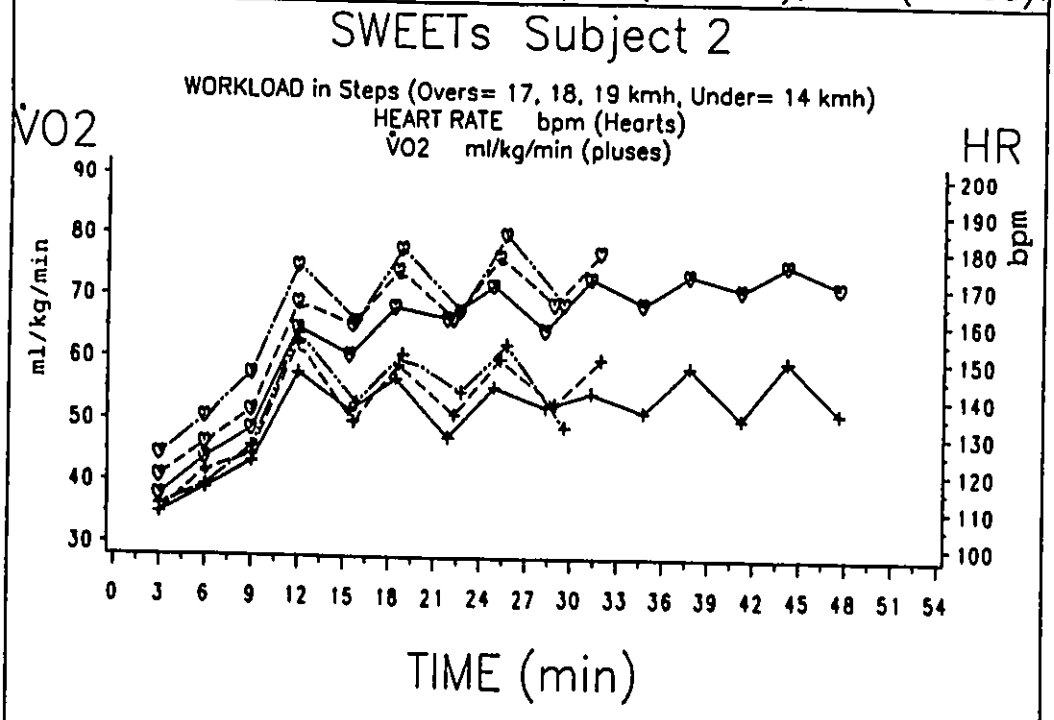


Figure 5. HRs and V̇O2s for 3 SWEETS.

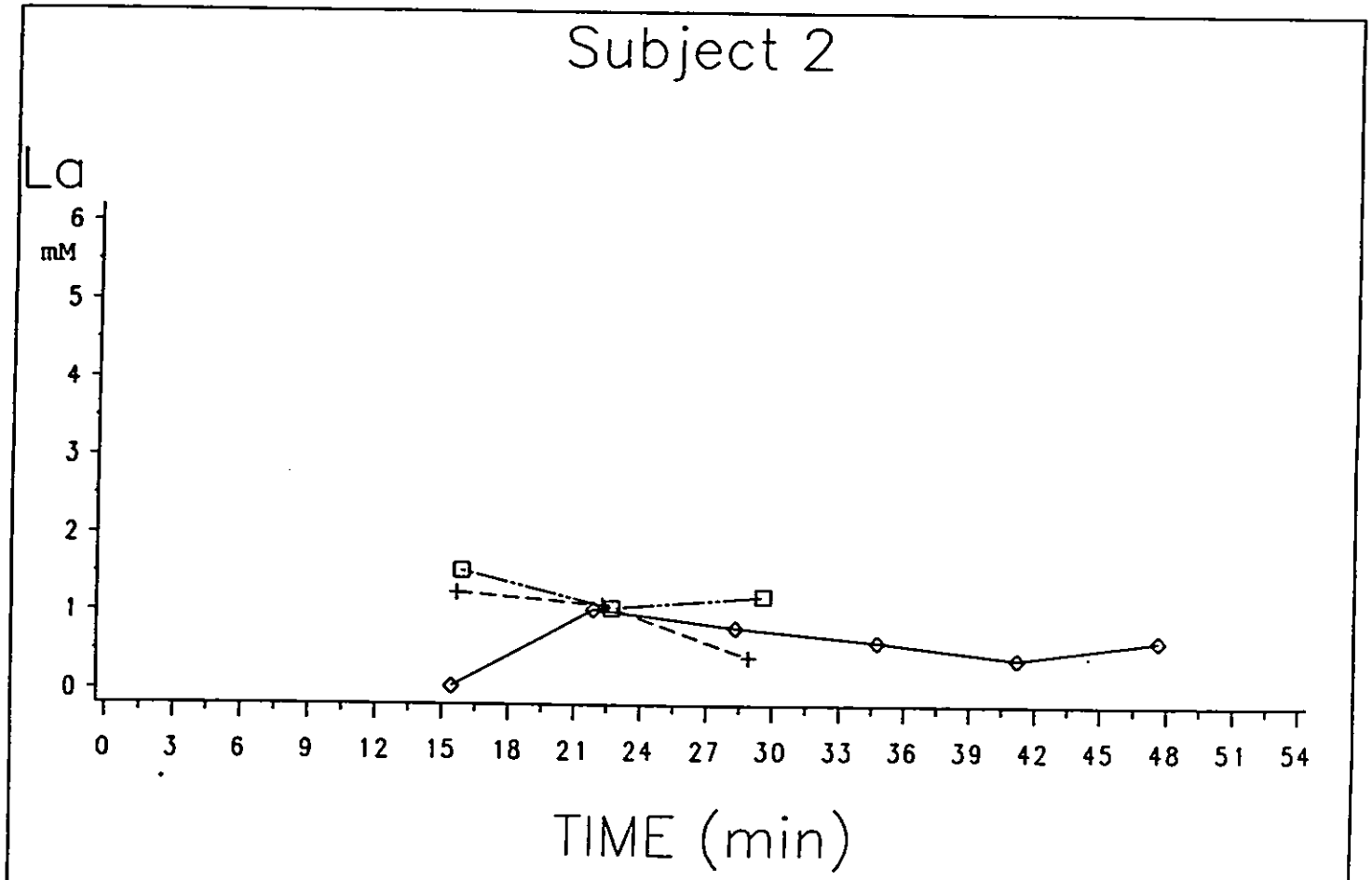


Figure 6. DeltaLa for SWEETs 1(solid), 2 (dashed), 3 (mixed)

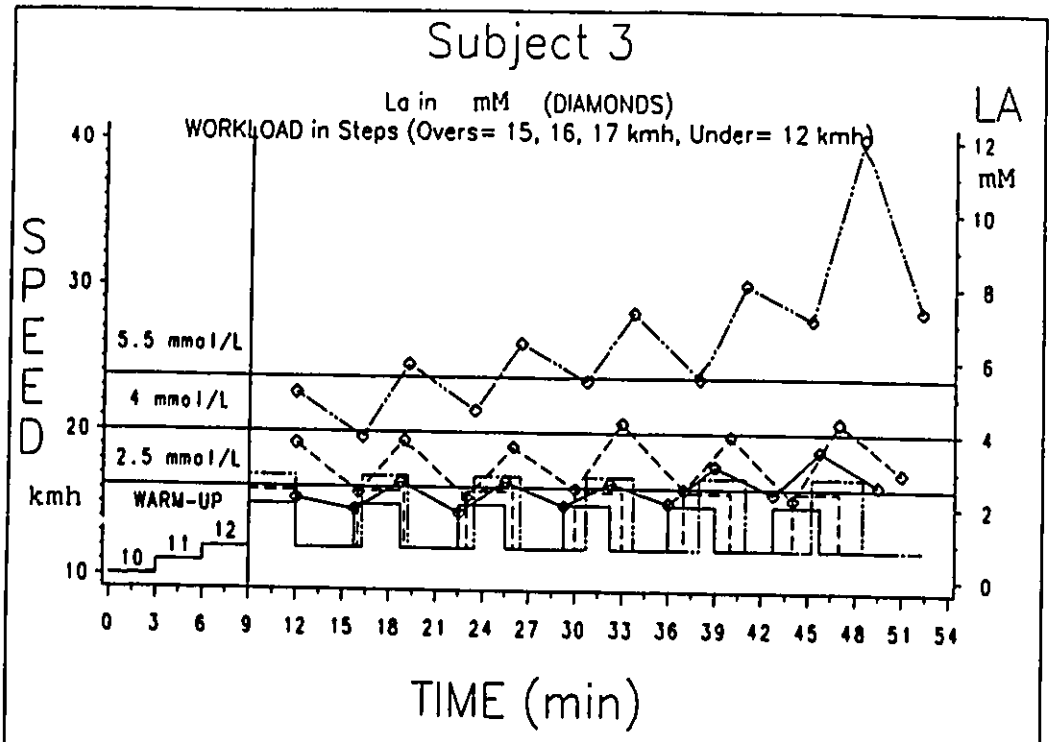


Figure 7. La for SWEET 1 (solid), 2 (dashed), & 3 (mixed).

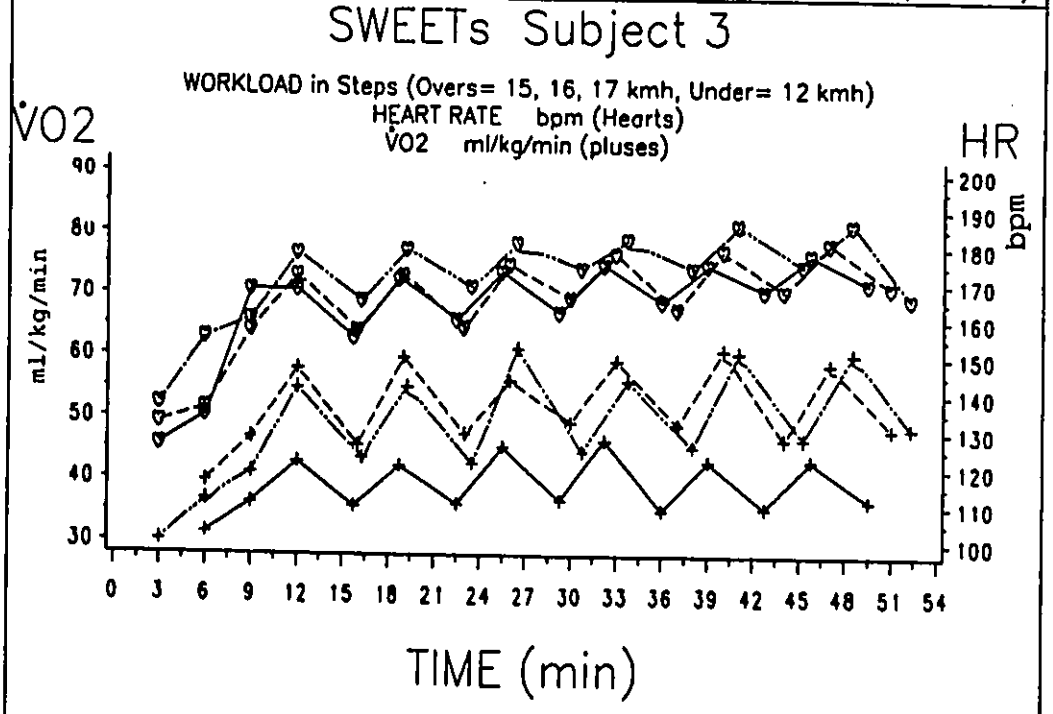


Figure 8. HRs and $\dot{V}O_2$s for 3 SWEETS.

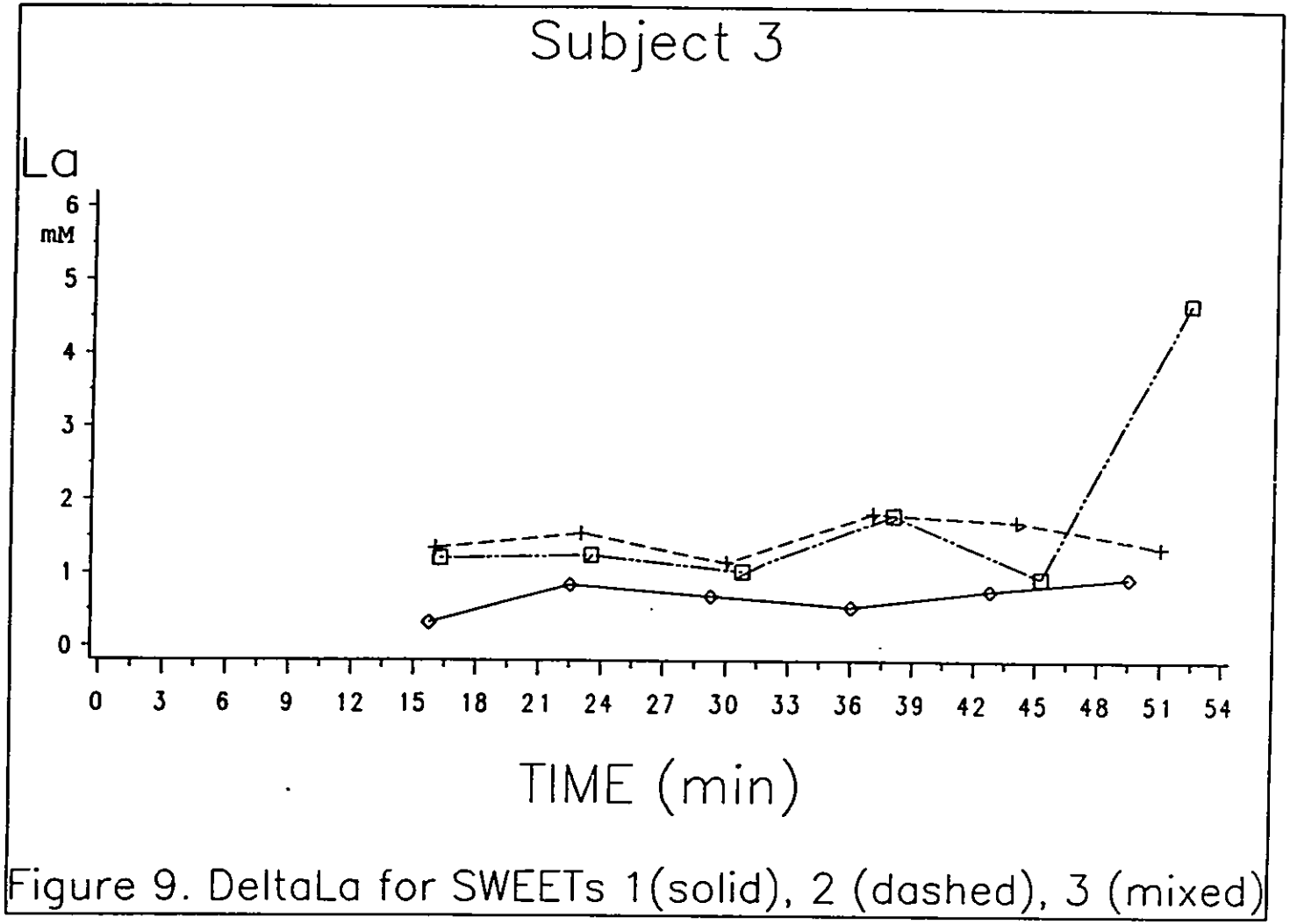


Figure 9. DeltaLa for SWEETs 1(solid), 2 (dashed), 3 (mixed)

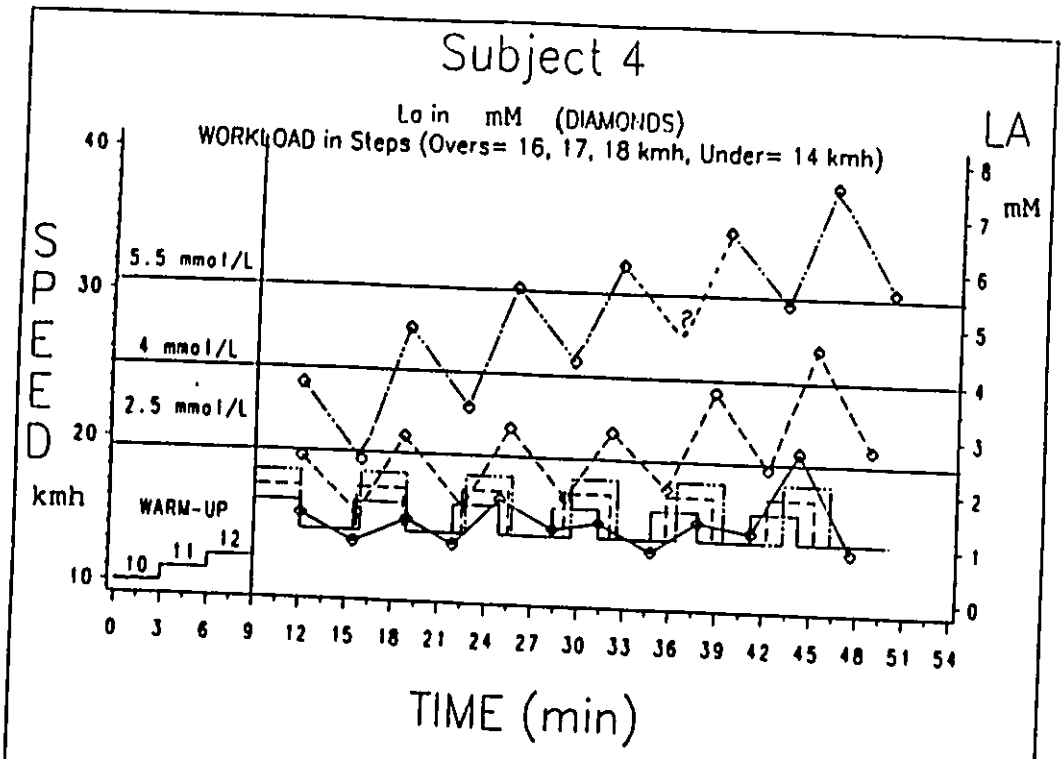


Figure 10. La for SWEET 1 (solid), 2 (dashed), & 3 (mixed).

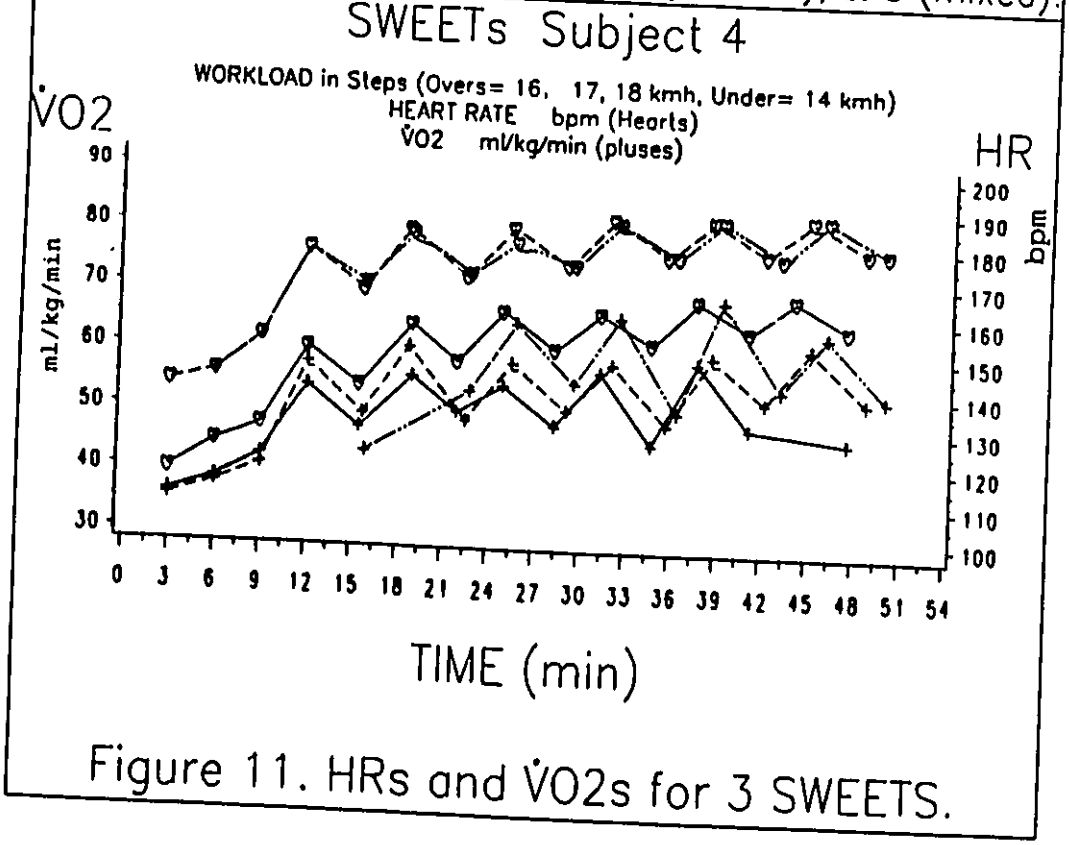


Figure 11. HRs and V̇O2s for 3 SWEETS.

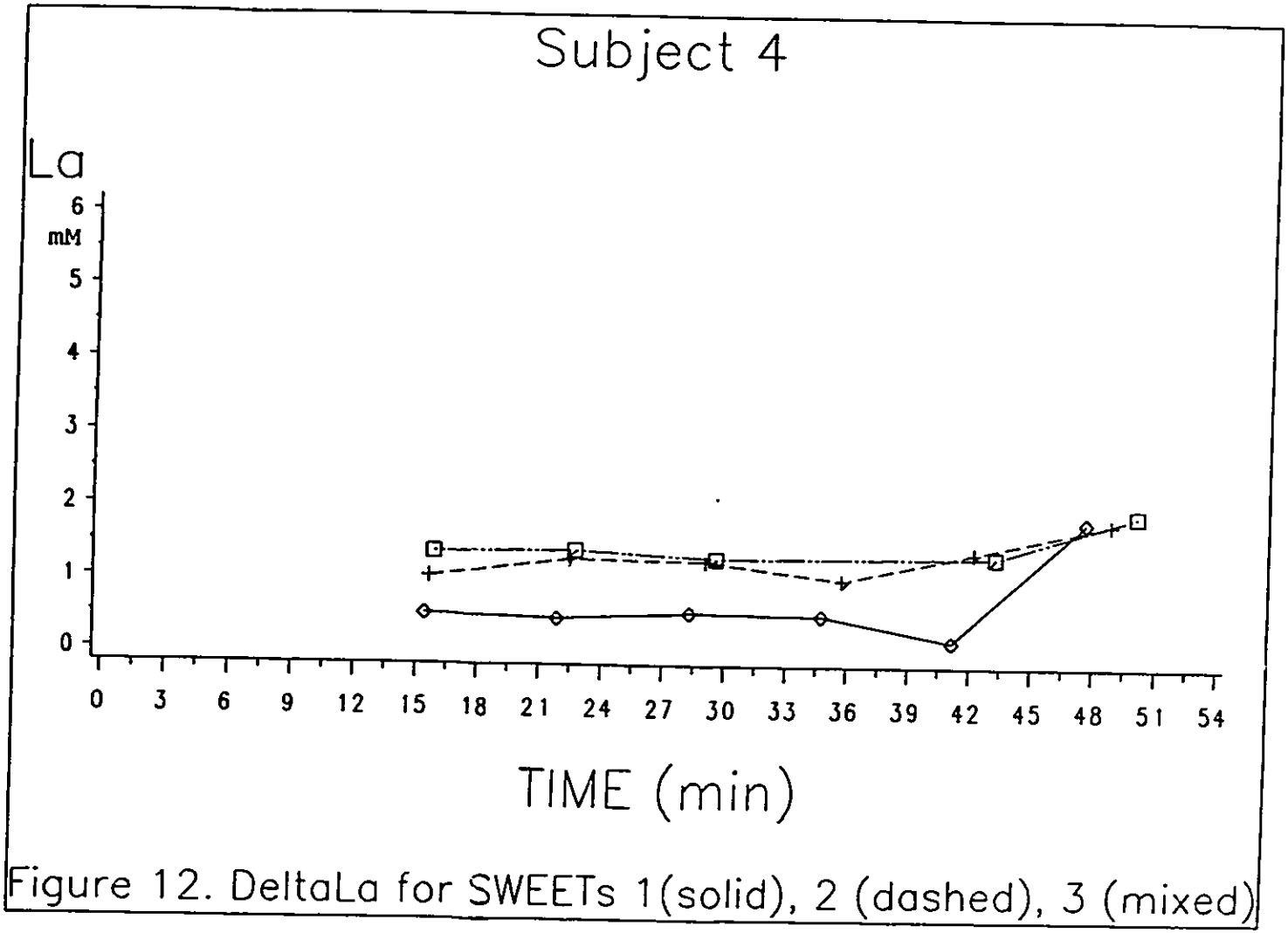


Figure 12. DeltaLa for SWEETs 1(solid), 2 (dashed), 3 (mixed)

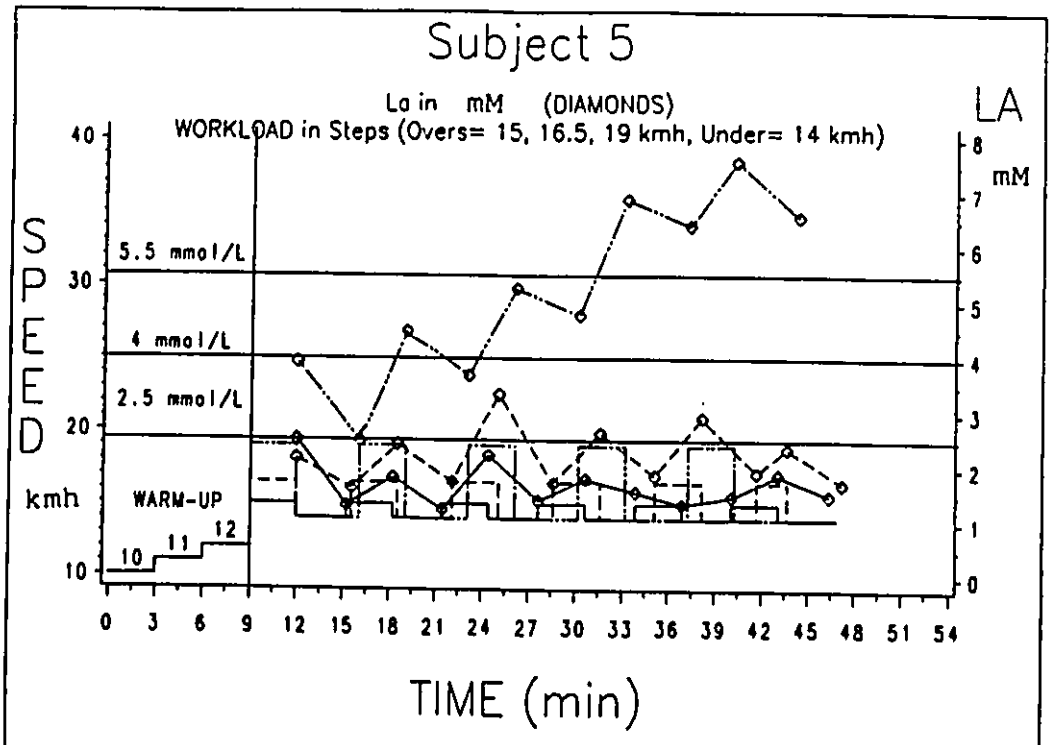


Figure 13. La for SWEET 1 (solid), 2 (dashed), & 3 (mixed).

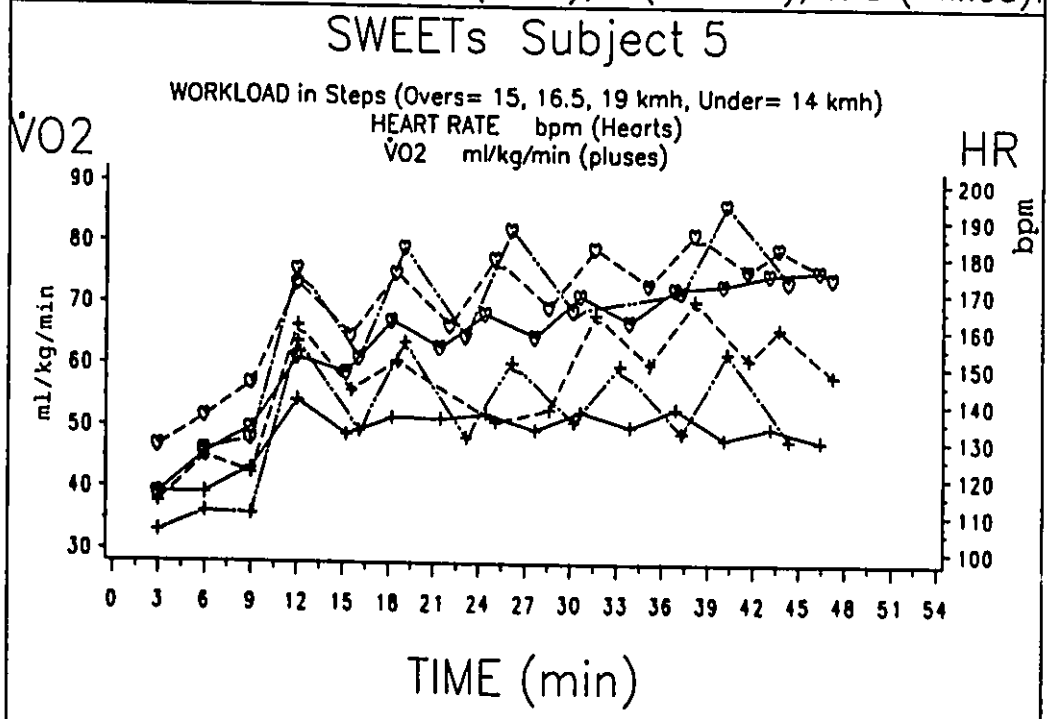


Figure 14. HRs and V̇O2s for 3 SWEETS.

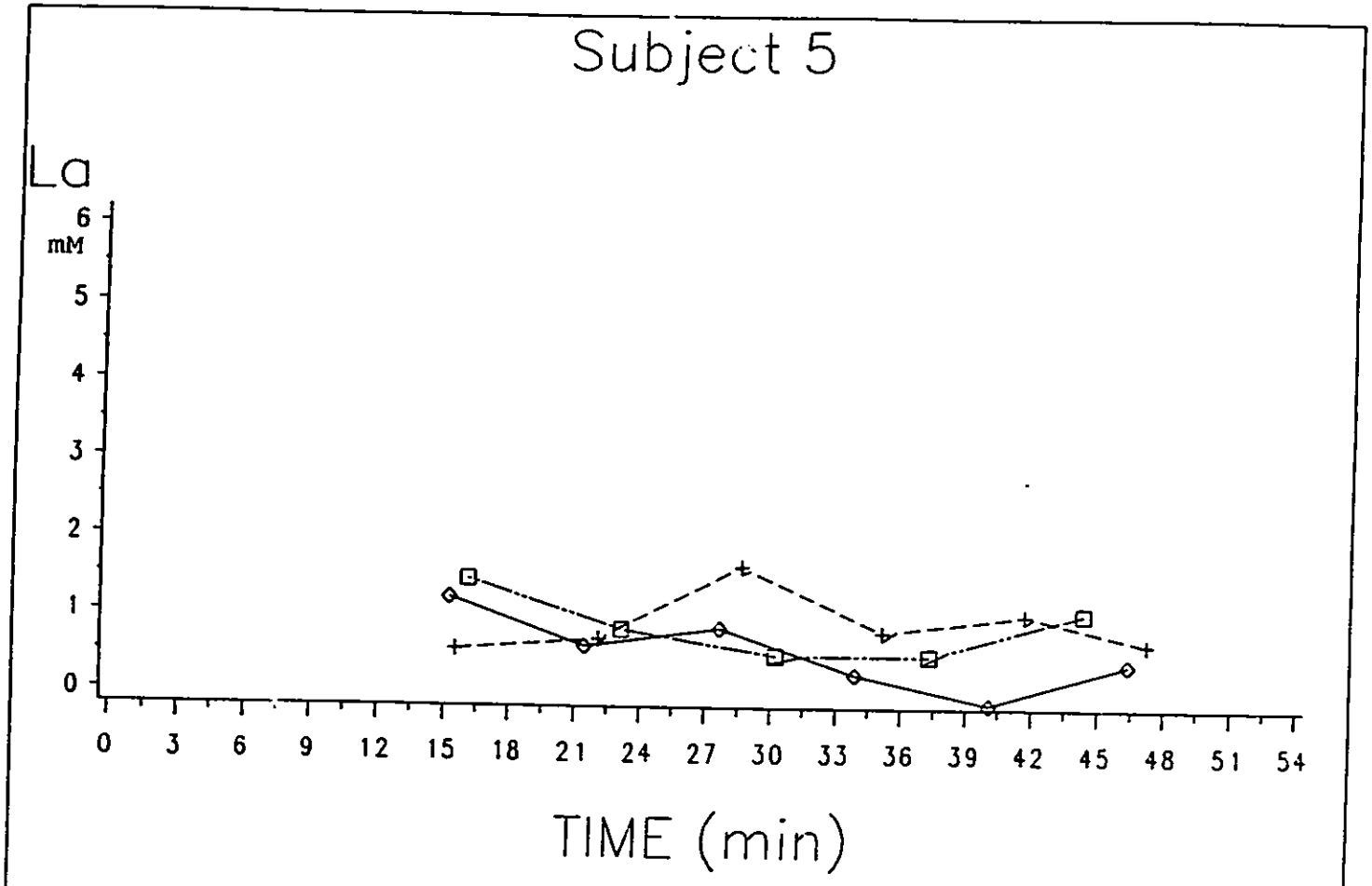


Figure 15. DeltaLa for SWEETs 1(solid), 2 (dashed), 3 (mixed)

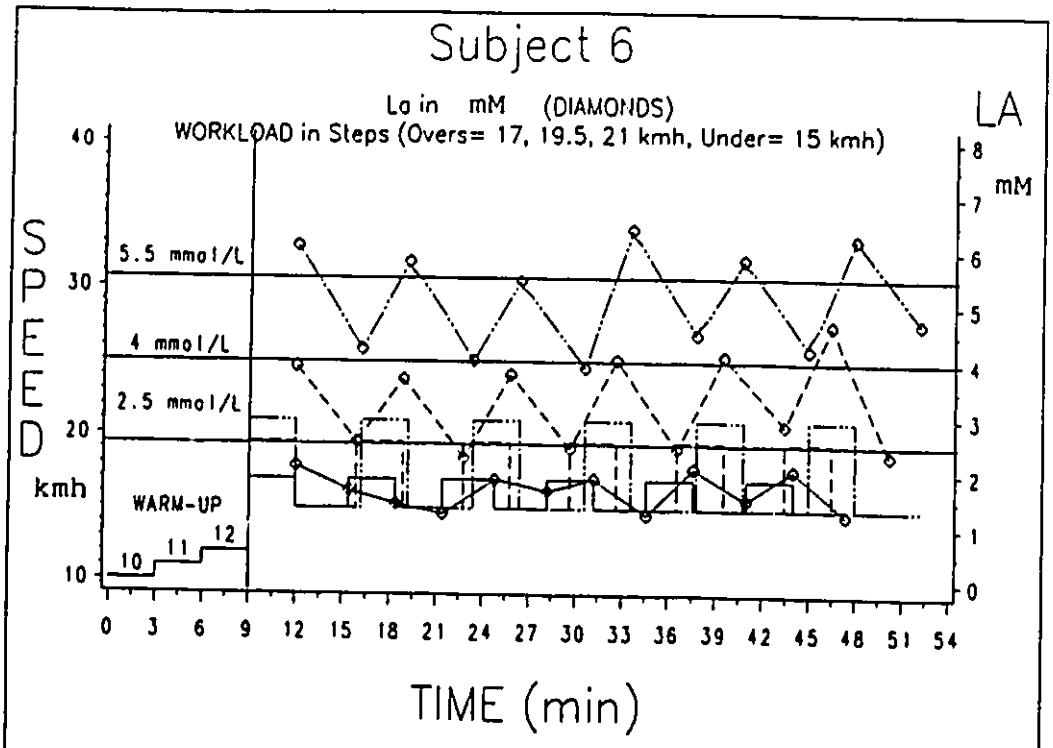


Figure 16. La for SWEET 1 (solid), 2 (dashed), & 3 (mixed).

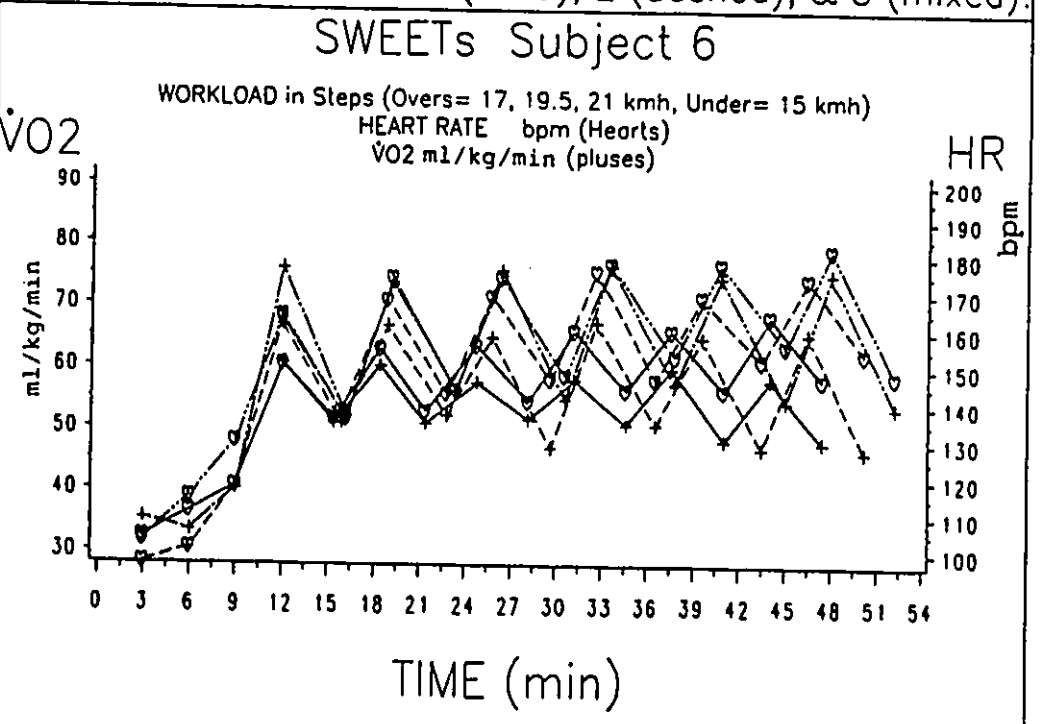


Figure 17. HRs and V̇O2s for 3 SWEETS.

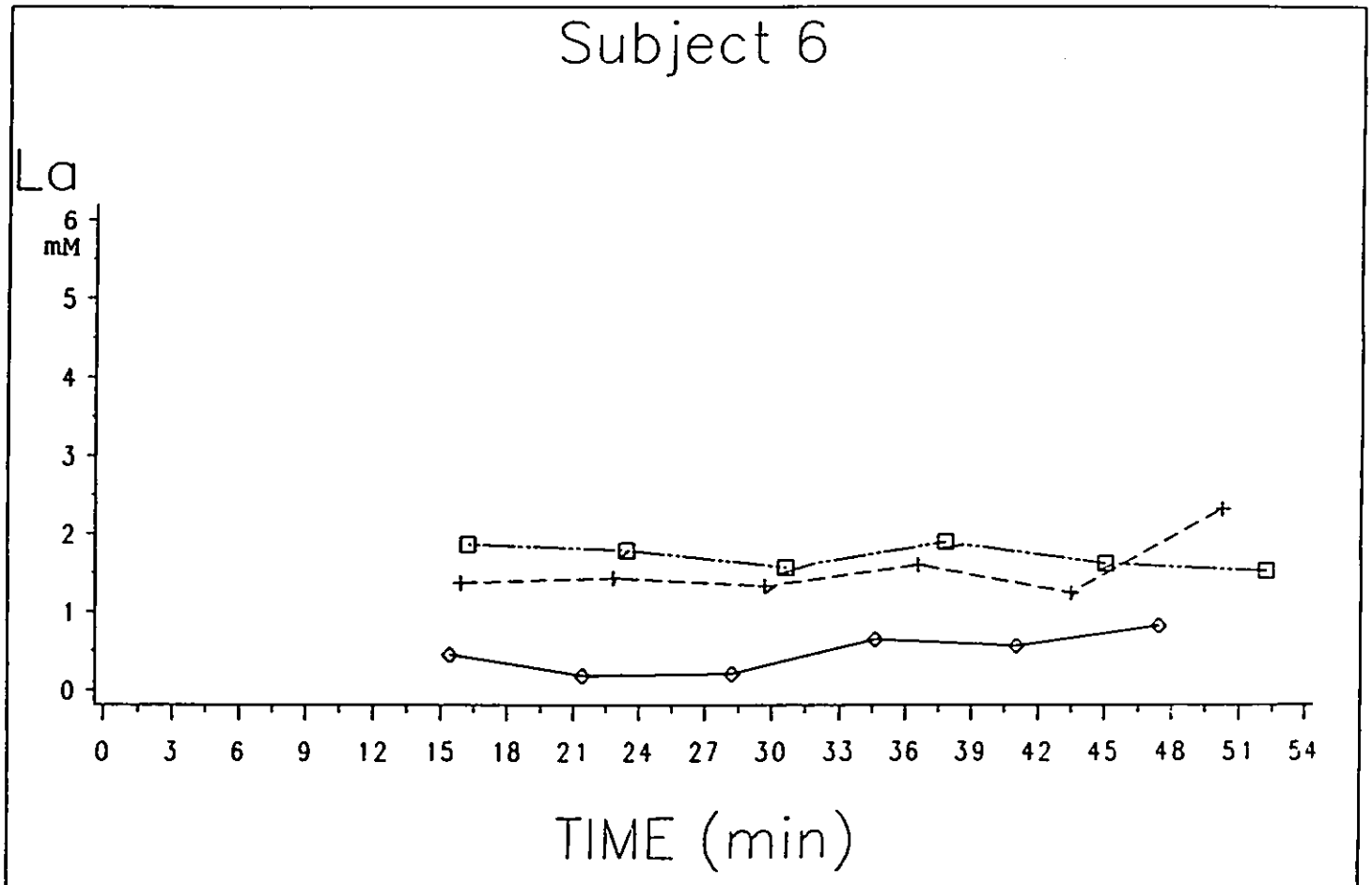


Figure 18. DeltaLa for SWEETs 1(solid), 2 (dashed), 3 (mixed)

Appendix F

Individual cells means for HLa, HR, and $\dot{V}O_2$ for each subject by stage (OVER and UNDER) and by SWEET.

Table F1. Statistics for [HLa] (mM) for 6 subjects per SWEETs per stages.

ID	Stage	N	SWEET 1	N	SWEET 2	N	SWEET 3
			($\bar{X}$, Max./SD Min)		($\bar{X}$, Max./SD Min)		($\bar{X}$, Max./SD Min)
1	Over	6	2.86 3.46	5	4.76 6.40	5	6.55 8.04
			0.37 2.42		1.04 3.68		1.26 4.88
	Under	6	1.83 2.20	5	3.13 4.04	5	4.34 5.58
			0.30 1.44		0.82 2.28		1.01 3.42
Delta La		6	1.04 1.26	5	1.63 2.36	5	2.22 2.50
(Over - Under)			0.12 0.90		0.43 1.12		0.43 1.46
2	Over	6	2.97 3.28	4	5.22 6.06	3	6.59 7.24
			0.22 2.64		0.66 4.48		0.59 6.10
	Under	6	2.38 2.64	4	4.02 4.88	3	5.34 6.04
			0.24 2.12		0.82 3.24		0.73 4.58
Delta La		6	0.59 1.02	3	0.91 1.24	3	1.25 1.52
(Over - Under)			0.34 0.04		0.43 0.42		0.24 1.04
3	Over	6	2.79 3.54	6	3.92 4.30	6	7.44 12.04
			0.47 2.18		0.30 3.60		2.48 5.06
	Under	6	2.11 2.60	6	2.44 2.94	6	5.63 7.34
			0.31 1.78		0.27 2.20		1.37 3.86
Delta La		6	0.68 0.94	6	1.49 1.82	6	1.82 4.70
(Over - Under)			0.22 0.32		0.26 1.14		1.44 0.94
4	Over	6	1.64 2.72	6	3.27 4.58	6	5.71 7.54
			0.55 1.30		0.78 2.40		1.36 3.72
	Under	6	0.98 1.24	6	1.98 2.78	6	4.20 5.62
			0.18 0.84		0.53 1.38		1.36 2.36
Delta La		6	0.66 1.82	6	1.28 1.80	5	1.45 1.92
(Over - Under)			0.59 0.16		0.29 1.00		0.26 1.28
5	Over	6	1.92 2.50	6	2.64 3.34	5	5.63 7.58
			0.40 1.34		0.43 2.16		1.56 3.92
	Under	6	1.41 1.56	6	1.77 1.92	5	4.78 6.56
			0.12 1.24		0.11 1.62		1.75 2.48
Delta La		6	0.51 1.20	6	0.87 1.62	5	0.84 1.44
(Over - Under)			0.47 -.14		0.40 0.54		0.40 0.48
6	Over	6	1.90 2.10	6	4.02 4.66	6	5.96 6.38
			0.26 1.42		0.35 3.68		0.33 5.44
	Under	6	1.42 1.66	6	2.48 2.86	6	4.25 4.70
			0.21 1.24		0.21 2.26		0.30 3.88
Delta La		6	0.47 0.82	6	1.54 2.32	6	1.76 1.90
(Over - Under)			0.25 0.18		0.4 1.24		0.16 1.52

Table F2. Statistics of heart rates (bpm) for 6 subject per SWEET per stages.

ID	Stage	N	SWEET 1	N	SWEET 2	N	SWEET 3
			($\bar{X}$, Max./SD Min)		($\bar{X}$, Max./SD Min)		($\bar{X}$, Max./SD Min)
1	Over	6	179 190	5	188 196	5	186 194
			8 168		7 179		7 175
	Under	6	167 178	5	172 182	5	165 173
			8 156		9 159		7 156
Delta HR		6	12 13	5	15 20	5	21 22
(Over - Under)			1 11		4 11		1 19
2	Over	6	169 176	4	174 179	3	179 184
			7 158		6 165		5 175
	Under	6	162 170	3	162 165	3	163 165
			7 151		3 159		3 160
Delta HR		6	6 12	3	11 13	3	17 19
(Over - Under)			3 3		4 6		2 15
3	Over	6	173 178	6	176 181	5	182 186
			4 168		4 172		4 178
	Under	6	163 170	6	164 169	6	171 175
			6 155		5 157		4 165
Delta HR		6	10 13	6	12 15	6	12 20
(Over - Under)			2 7		2 9		5 7
4	Over	6	179 190	5	188 196	5	186 194
			8 168		7 179		7 175
	Under	6	167 178	5	172 182	5	165 173
			8 156		9 159		7 156
Delta HR		6	12 13	5	15 20	5	21 22
(Over - Under)			1 11		4 11		1 19
5	Over	6	169 176	4	174 179	3	179 184
			7 158		6 165		5 175
	Under	6	162 170	3	162 165	3	163 165
			7 151		3 159		3 160
Delta HR		6	6 12	3	11 133		17 19
(Over - Under)			3 3		4 6		2 15
6	Over	6	173 178	6	176 181	5	182 186
			4 168		4 172		4 178
	Under	6	163 170	6	164 169	6	171 175
			6 155		5 157		4 165
Delta HR		6	10 13	6	12 15	6	12 20
(Over - Under)			2 7		2 9		5 7

Table F3. Statistics of $\dot{V}O_2$ (ml/kg/min) for 6 subjects per SWEET per stage.

ID	Stage	N	SWEET 1 (X, Max./SD, MIn)	N	SWEET 2 (X, Max./SD, MIn)	N	SWEET3 (X, Max./SD, MIn)
1	Over	6	60.6 62.6 1.56 59.1	5	63.7 71.5 1.16 62.4	5	67.1 71.5 3.64 62.9
	Under	6	50.5 52.3 1.63 48.1	5	51.5 53.7 1.48 49.9	5	50.4 55.5 3.77 45.8
Delta O_2 (Over - Under)		6	10.1 13.3 1.95 7.7	5	12.2 14.0 2.4 8.7	5	16.6 19.7 2.8 12.9
2	Over	6	57.3 59.9 1.96 54.7	4	60.4 62.3 1.38 59.0	3	62.4 64.0 1.60 60.8
	Under	6	50.9 52.4 1.75 47.54	3	51.3 52.9 1.51 49.9	3	52.3 54.8 2.85 49.2
Delta O_2 (Over - Under)		6	6.4 9.3 1.95 7.7	3	9.2 12.4 2.8 7.2	3	10.1 13.3 3.7 6.0
3	Over	6	44.0 46.5 1.63 42.3	6	58.9 61.2 1.73 56.1	6	58.2 61.4 3.07 54.9
	Under	6	36.1 36.9 0.66 35.3	6	47.9 49.5 1.45 45.7	6	45.4 48.7 2.16 42.8
Delta O_2 (Over - Under)		6	7.8 11.2 1.9 6.1	3	11.0 14.4 2.6 6.6	6	12.8 16.7 2.2 10.7
4	Over	5	56.0 58.9 2.05 53.8	6	59.4 61.6 1.57 57.5	4	65.9 69.1 2.35 63.7
	Under	6	47.6 50.0 1.59 45.5	6	50.7 53.1 1.92 48.8	6	51.9 55.2 4.47 43.3
Delta O_2 (Over - Under)		5	8.8 10.7 1.4 6.6	6	10.6 14.7 2.6 6.9	6	12.3 15.1 2.9 9.5
5	Over	6	52.3 54.3 1.43 50.1	6	63.9 70.7 7.17 50.9	5	62.1 63.8 1.73 60.1
	Under	6	49.4 51.4 1.31 47.9	5	57.8 61.2 3.36 53.0	5	49.3 51.0 1.11 48.2
Delta O_2 (Over - Under)		6	2.9 5.5 2.0 0.1	5	6.7 10.4 5.0 -2.1	5	12.8 15.4 2.5 9.6
6	Over	6	59.1 60.8 1.34 57.6	6	66.1 67.4 1.04 64.9	6	75.3 76.5 1.03 73.6
	Under	6	50.1 51.6 1.85 52.2	6	49.2 52.3 2.49 2.26	6	54.9 57.5 1.85 3.88
Delta O_2 (Over - Under)		6	9.0 11.5 2.0 6.0	6	18.28 19.9 1.38 16.6	6	23.2 29.5 3.3 20.5

Appendix G

Individual cell means correlated with Pearson Product-Moment correlation coefficients for [HLa] (LA), HR, $\dot{V}O_2$ for each subject (ID) by stage (OVER or UNDER) and by SWEET (1,2 or 3). NOTE LA, HR, $\dot{V}O_2$ = SWEET 1; LA2, HR2, $\dot{V}O_2$2= SWEET 2; LA3, HR3, $\dot{V}O_2$3 = SWEET 3.

Table G1

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=1 ID=1

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.66113 0.2244 5	0.41611 0.4859 5	0.79165 0.0606 6	0.44331 0.4546 5	0.38634 0.5206 5	0.54357 0.2650 6	0.36385 0.5472 5	0.71426 0.1753 5
LA2		0.93549 0.0195 5	0.94470 0.0155 5	0.92751 0.0232 5	0.90870 0.0327 5	0.45738 0.4386 5	0.44560 0.4520 5	0.83476 0.0786 5
LA3			0.96228 0.0087 5	0.98972 0.0012 5	0.98424 0.0024 5	0.34789 0.5662 5	0.18921 0.7605 5	0.81119 0.0956 5
HR				0.98653 0.0019 5	0.97712 0.0041 5	0.58390 0.2237 6	0.17605 0.7770 5	0.93352 0.0204 5
HR2					0.99701 0.0002 5	0.46855 0.4260 5	0.14702 0.8135 5	0.87147 0.0542 5
HR3						0.47236 0.4218 5	0.14567 0.8152 5	0.85012 0.0681 5
$\dot{V}O_2$							0.12544 0.8407 5	0.74891 0.1452 5
$\dot{V}O_2$2								0.02673 0.9660 5

Table G2

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=1 ID=2

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.35181 0.6482 4	0.33264 0.7841 3	0.60285 0.2053 6	0.59520 0.4048 4	0.59079 0.5976 3	0.37932 0.4583 6	-0.97848 0.0215 4	-0.99677 0.0512 3
LA2		0.90600 0.2782 3	0.93483 0.0652 4	0.89049 0.1095 4	0.98964 0.0917 3	-0.97180 0.0282 4	-0.52477 0.4752 4	-0.64092 0.5571 3
LA3			0.97392 0.1457 3	0.90158 0.2848 3	0.95740 0.1865 3	-0.99352 0.0725 3	-0.46607 0.6913 3	-0.25577 0.8353 3
HR				0.97524 0.0248 4	0.99795 0.0408 3	0.21830 0.6777 6	-0.58640 0.4136 4	-0.46845 0.6896 3
HR2					0.98810 0.0983 3	-0.91768 0.0823 4	-0.74773 0.2523 4	-0.64883 0.5505 3
HR3						-0.98402 0.1140 3	-0.70170 0.5048 3	-0.52404 0.6488 3
$\dot{V}O_2$							0.45991 0.5401 4	0.36401 0.7628 3
$\dot{V}O_2$2								0.97453 0.1440 3

Table G3

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=1 ID=3 (N=6)

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.61242 0.1962	0.94242 0.0049	0.90081 0.0143	0.85957 0.0282	0.91888 0.0096	-0.09363 0.8600	0.35014 0.4963	0.70463 0.1180
LA2		0.75579 0.0822	0.77856 0.0681	0.79517 0.0586	0.63533 0.1752	0.30788 0.5528	0.43726 0.3859	0.09800 0.8535
LA3			0.89639 0.0155	0.88649 0.0186	0.85492 0.0300	0.01344 0.9798	0.22598 0.6668	0.58893 0.2187
HR				0.95076 0.0036	0.90538 0.0130	0.32918 0.5241	0.27364 0.5998	0.66809 0.1470
HR2					0.95169 0.0034	0.30794 0.5527	0.32826 0.5253	0.66086 0.1530
HR3						0.09003 0.8653	0.43735 0.3858	0.75069 0.0855
$\dot{V}O_2$							-0.35760 0.4865	0.19350 0.7134
$\dot{V}O_2$2								-0.14273 0.7874

Table G4

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=1 ID=4

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.81149 0.0500 6	0.67156 0.1441 6	0.55021 0.2580 6	0.44508 0.3765 6	0.44276 0.3793 6	-0.07684 0.8850 6	-0.09786 0.8537 6	-0.72350 0.2765 4
LA2		0.93854 0.0055 6	0.86761 0.0251 6	0.78180 0.0662 6	0.83030 0.0408 6	0.45036 0.3701 6	0.22108 0.6738 6	-0.17876 0.8212 4
LA3			0.96930 0.0014 6	0.93652 0.0059 6	0.91998 0.0093 6	0.42700 0.3984 6	0.07467 0.8882 6	-0.10052 0.8995 4
HR				0.96056 0.0023 6	0.88245 0.0199 6	0.51303 0.2980 6	0.11710 0.8252 6	0.10935 0.8907 4
HR2					0.93495 0.0062 6	0.41833 0.4091 6	0.10058 0.8496 6	0.11069 0.8893 4
HR3						0.50286 0.3093 6	0.26381 0.6135 6	0.23413 0.7659 4
$\dot{V}O_2$							0.72168 0.1054 6	0.80327 0.1967 4
$\dot{V}O_2$2								0.58914 0.4109 4

Table G5

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=1 ID=5

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	-0.23129 0.6592 6	-0.81950 0.0895 5	-0.73950 0.0930 6	-0.80709 0.0522 6	-0.82196 0.1780 4	0.27040 0.6043 6	-0.47087 0.3459 6	0.17815 0.7744 5
LA2		0.47855 0.4148 5	0.29089 0.5760 6	0.48614 0.3282 6	0.74589 0.2541 4	-0.02727 0.9591 6	-0.53886 0.2699 6	-0.69909 0.1889 5
LA3			0.91971 0.0270 5	0.98165 0.0030 5	0.95840 0.0416 4	-0.07201 0.9084 5	0.46686 0.4279 5	-0.61032 0.2743 5
HR				0.89412 0.0162 6	0.97534 0.0247 4	-0.67716 0.1395 6	0.24407 0.6412 6	-0.64139 0.2434 5
HR2					0.98813 0.0119 4	-0.29620 0.5687 6	0.34340 0.5051 6	-0.62416 0.2604 5
HR3						-0.29743 0.7026 4	0.10498 0.8950 4	-0.62723 0.3728 4
$\dot{V}O_2$							0.23599 0.6526 6	0.24649 0.6894 5
$\dot{V}O_2$2								0.18179 0.7698 5

Table G6

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=1 ID=6

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.59061 0.2171	0.30726 0.5536	0.23731 0.6507	-0.01010 0.9849	-0.01010 0.9849	-0.07196 0.8923	-0.57228 0.2353	0.00877 0.9868
LA2		0.57604 0.2315	0.79918 0.0564	0.54124 0.2674	0.62035 0.1888	-0.33389 0.5178	-0.95477 0.0030	0.77823 0.0683
LA3			0.36691 0.4743	0.47208 0.3445	0.20008 0.7039	0.01415 0.9788	-0.39749 0.4352	0.56398 0.2437
HR				0.85470 0.0301	0.96134 0.0022	-0.60341 0.2047	-0.90128 0.0141	0.84935 0.0323
HR2					0.86333 0.0267	-0.75459 0.0829	-0.61796 0.1911	0.70213 0.1199
HR3						-0.62673 0.1830	-0.76413 0.0769	0.81374 0.0488
$\dot{V}O_2$							0.46167 0.3567	-0.23996 0.6470
$\dot{V}O_2$2								-0.75775 0.0809

Table G7

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=2 ID=1

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.44245 0.4556 5	0.46690 0.4279 5	0.70002 0.1215 6	0.33043 0.5871 5	0.41119 0.4916 5	-0.12985 0.8063 6	-0.13395 0.8300 5	0.33878 0.5770 5
LA2		0.98635 0.0019 5	0.95867 0.0100 5	0.95746 0.0105 5	0.97620 0.0044 5	0.56832 0.3175 5	0.68649 0.2005 5	0.88253 0.0475 5
LA3			0.94809 0.0141 5	0.90881 0.0326 5	0.94708 0.0145 5	0.46459 0.4305 5	0.57913 0.3062 5	0.86857 0.0561 5
HR				0.95846 0.0101 5	0.98673 0.0018 5	-0.21291 0.6855 6	0.56604 0.3199 5	0.71467 0.1749 5
HR2					0.99088 0.0010 5	0.57975 0.3056 5	0.77450 0.1241 5	0.76629 0.1308 5
HR3						0.54275 0.3446 5	0.69204 0.1954 5	0.77209 0.1260 5
$\dot{V}O_2$							0.62603 0.2586 5	0.64130 0.2435 5
$\dot{V}O_2$2								0.72144 0.1689 5

Table G8

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=2 ID=2

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	-0.84882 0.3546 3	-0.91483 0.2646 3	0.20626 0.6950 6	-0.77953 0.4309 3	-0.93167 0.2367 3	0.31271 0.5462 6	-0.82436 0.3831 3	0.21855 0.8597 3
LA2		0.99003 0.0900 3	0.62900 0.5669 3	0.99283 0.0763 3	0.98289 0.1179 3	0.22121 0.8580 3	0.99900 0.0284 3	-0.70142 0.5051 3
LA3			0.73225 0.4769 3	0.96608 0.1663 3	0.99904 0.0279 3	0.08161 0.9480 3	0.98275 0.1184 3	-0.59400 0.5951 3
HR				0.53155 0.6432 3	0.76143 0.4490 3	-0.07392 0.8893 6	0.59366 0.5954 3	0.11291 0.9280 3
HR2					0.95382 0.1942 3	0.33621 0.7817 3	0.99718 0.0479 3	-0.78159 0.4288 3
HR3						0.03779 0.9759 3	0.97368 0.1464 3	-0.55813 0.6230 3
$\dot{V}O_2$							0.26453 0.8296 3	-0.85025 0.3529 3
$\dot{V}O_2$2								-0.73254 0.4767 3

Table G9

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=2 ID=3 (N=6)

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.65790 0.1555	0.94282 0.0048	0.89718 0.0153	0.87087 0.0239	0.03321 0.9502	0.24568 0.6389	0.18664 0.7233	0.99372 0.0001
LA2		0.48495 0.3296	0.50829 0.3032	0.51803 0.2925	-0.38631 0.4494	0.56528 0.2424	0.46151 0.3569	0.69087 0.1284
LA3			0.97011 0.0013	0.93924 0.0054	0.29371 0.5721	0.25372 0.6276	0.30926 0.5509	0.92693 0.0078
HR				0.90005 0.0145	0.35400 0.4912	0.17829 0.7354	0.45337 0.3665	0.89165 0.0170
HR2					0.41382 0.4147	0.38149 0.4555	0.48016 0.3351	0.88225 0.0200
HR						-0.26792 0.6077	0.45958 0.3592	0.06401 0.9041
$\dot{V}O_2$							0.39298 0.4409	0.23240 0.6577
$\dot{V}O_2$2								0.24607 0.6383

Table G10

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=2 ID=4

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.24755 0.6363 6	0.50035 0.3906 5	0.40568 0.4249 6	0.35235 0.4933 6	0.26760 0.6082 6	-0.17199 0.7446 6	0.62476 0.1848 6	0.56334 0.2444 6
LA2		0.95151 0.0127 5	0.92651 0.0079 6	0.90804 0.0123 6	0.92231 0.0088 6	0.03664 0.9451 6	0.65259 0.1601 6	0.54186 0.2668 6
LA3			0.99727 0.0002 5	0.99936 0.0001 5	0.99544 0.0004 5	0.01439 0.9817 5	0.74370 0.1496 5	0.75761 0.1379 5
HR				0.98596 0.0003 6	0.97516 0.0009 6	0.03365 0.9496 6	0.58667 0.2210 6	0.76942 0.0736 6
HR2					0.99482 0.0001 6	-0.05767 0.9136 6	0.50168 0.3106 6	0.69828 0.1228 6
HR3						-0.07577 0.8866 6	0.45665 0.3626 6	0.65405 0.1588 6
$\dot{V}O_2$							0.36884 0.4718 6	0.11866 0.8228 6
$\dot{V}O_2$2								0.39379 0.4399 6

Table G11

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=2 ID=5

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.62247 0.1869 6	0.88645 0.0451 5	0.72507 0.1030 6	0.88479 0.0191 6	0.85543 0.0645 5	-0.45579 0.3637 6	0.69075 0.1966 5	0.06159 0.9216 5
LA2		0.94906 0.0137 5	0.57763 0.2299 6	0.77092 0.0727 6	0.93335 0.0204 5	-0.00517 0.9922 6	0.75341 0.1414 5	-0.47821 0.4152 5
LA3			0.92578 0.0240 5	0.98166 0.0030 5	0.99167 0.0009 5	-0.17390 0.7797 5	0.66510 0.3349 4	-0.18310 0.7682 5
HR				0.93262 0.0067 6	0.94630 0.0148 5	-0.56262 0.2451 6	0.58616 0.2989 5	-0.36923 0.5408 5
HR2					0.98309 0.0026 5	-0.48430 0.3303 6	0.70728 0.1815 5	-0.24193 0.6950 5
HR3						-0.21574 0.7275 5	0.59350 0.4065 4	-0.13664 0.8266 5
$\dot{V}O_2$							-0.23546 0.7030 5	-0.03181 0.9595 5
$\dot{V}O_2$2								-0.91211 0.0879 4

Table G12

Pearson Product-Moment correlation coefficients for each cell for La, HR, and $\dot{V}O_2$ by subject (ID) and stage (OVER or UNDER).

STAGE=2 ID=6 (N=6)

	LA2	LA3	HR	HR2	HR3	$\dot{V}O_2$	$\dot{V}O_2$2	$\dot{V}O_2$3
LA	0.52025 0.2900	-0.57170 0.2359	-0.46090 0.3576	-0.35605 0.4885	-0.30210 0.5606	0.28889 0.5787	-0.51506 0.2957	-0.17466 0.7407
LA2		-0.06390 0.9043	0.12486 0.8137	0.20590 0.6955	0.40730 0.4228	-0.37647 0.4620	-0.50991 0.3014	0.00142 0.9979
LA3			0.66822 0.1469	0.47990 0.3354	0.20918 0.6908	-0.64278 0.1686	-0.20524 0.6965	-0.44243 0.3797
HR				0.94971 0.0037	0.77756 0.0687	-0.69137 0.1282	-0.44342 0.3785	-0.16874 0.7493
HR2					0.79514 0.0587	-0.75635 0.0818	-0.51431 0.2966	-0.20096 0.7026
HR3						-0.42635 0.3992	-0.19088 0.7172	0.38557 0.4503
$\dot{V}O_2$							0.52230 0.2878	0.55294 0.2551
$\dot{V}O_2$2								0.66390 0.1505

Appendix H

Table H1. Latin square order of treatments projected versus actual test sequence.

Subject	Projected order/ actual order		
1	2/2	3/1*	1/3*
2	3/3	1/1	2/2
3	2/2	1/1	3/3
4	3/3	2/2	1/1
5	1/1	2/2	3/3
6	1/1	3/3	2/2

* = Athlete not able to do difficult workout on day two.

Table H2. N for ΔLa per SWEET per repeat.

SWEET	Repeat					
	1	2	3	4	5	6
1	6	6	6	6	6	6
2	6	6	6	5	5	3
3	6	6	6	4	5	3

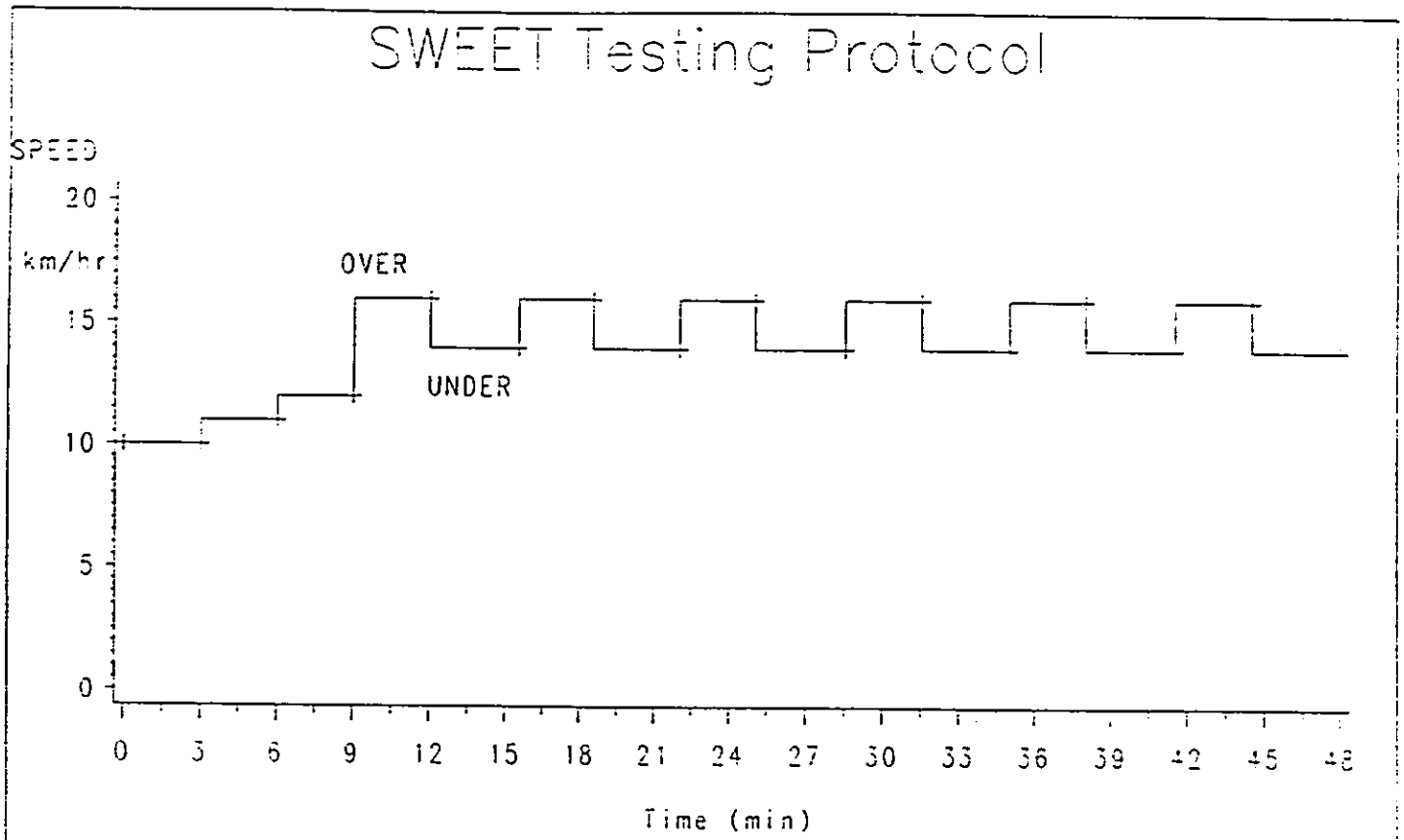


FIGURE H1. Square-Wave Endurance Exercise Test profile with the OVER and UNDER speeds set individually from the HLa ID Test.

APPENDIX I

Three Chapters: A Proposal

A Comparison of the Effects of Three Selected Exercise Intensities on the Lactate Recovery Ability of Elite Distance Runners

by

Robert A. Séguin, B.P.E., B.Ed.

First Three Chapters

A thesis proposal presented to the University of Ottawa
School of Human Kinetics in partial fulfilment
of the thesis requirement for the degree of
Masters of Science in Kinanthropology.

Blood Lactate Recovery at Three Intensities Around Threshold in Elite Runners

by

Robert A. Séguin, B.P.E., B.Ed.

An article presented to the University of Ottawa
School of Human Kinetics in partial fulfilment
of the thesis requirement for the degree of
Masters of Science in Kinanthropology.

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CHAPTER ONE

I.1 Introduction

In the field of exercise physiology sport scientists have been researching the physiological responses to aerobic endurance events. Cross country running and skiing are examples of two sport groups traditionally tested in high performance laboratories.

There has been a trend in this research to assess the athletes physiological responses that are better indicators and/or predictors of prolonged performance (Costill, Thomason & Roberts, 1973; Farrell, Wilmore, Coyle, Billings & Costill, 1979; Sjodin & Jacobs, 1982); are relevant to performance modes, intensities and durations (Whithers, Sherman, Miller & Costill, 1981; Åstrand, 1984; Heck, Mader, Hess, Muller & Hollman, 1985); and show greater interindividual differences in homogeneous groups (Coyle, 1988; Sjodin & Svedenhag, 1985).

This progression is reflected in the movement from investigation of maximal aerobic power (MAP) (Åstrand, 1984; Shepherd, 1984); to the anaerobic threshold (AT) (Davis, 1985; Brooks, 1985; Caiozzo, Davis, Ellis, Azus, Vandagriff, Prietto & McMaster, 1982); to blood lactate threshold (LT) and related variables (Kinderman, Simon & Keul, 1979; Stegmann & Kindermann, 1982; Londeree, 1986); to testing blood lactate recovery kinetics and reclassifying the results relative to various different lactate thresholds or lactate baselines (McLelland & Skinner, 1982; Kumagai, Tanaka,

Matsuura, Matsuzaka, Hirakoba, & Asano, 1982; Stark, 1989).

There have been numerous methodologies for fixing the lactate inflection points with just as many criticisms (Richardson & Gladden, 1984: Beaver, Wasserman & Whipp, 1985; Hughson, Weisiger & Swanson, 1987). Recently Campbell, Hughson and Green (1989) have shown the lactate inflection point to be an artifact of our methodology. In contrast to previous models Campbell et al. (1989) see blood lactate accumulation from incremental testing as a smooth continuous increasing exponential function that depicts no inflection point when plotted. Although we may conclude from this that there may not be a discrete physiological threshold; this does not preclude a performance threshold beyond which aerobic endurance is sharply curtailed by early fatigue due to metabolic acidosis. In the laboratory this is measured as total work time in either a continuous or discontinuous pattern of exercise until subject-determined exhaustion. This exhaustion point is preceded by an accumulation of blood lactate beyond the 4 mM point to those levels associated with maximal aerobic power which corresponds to a blood lactate level of 8 to 10 mM (Heck et al., 1985: Astrand, 1986).

With doubt being cast on the lactate inflection point as a discrete physiological threshold (Campbell et al., 1989), other performance thresholds at fixed blood lactate concentrations like the mean steady state (MSS=2.2 mM) (Stegmann and Kindermann & Schnabel, 1981; Lafontaine, Londeree & Spath, 1981), the onset of blood lactic acid accumulation (OBLA) (Heck et al., 1985; Sjodin & Jacobs, 1981) and the individual

anaerobic threshold (IAT) (Stegmann & Kinderman, 1982; Stegmann et al., 1981) may better explain the large variance of an elite athletes' aerobic endurance performance (Londeree, 1986).

The OBLA level of exertion has been targeted as the exercise level most highly correlated with distance race times (Sjödin & Jacobs, 1984; Heck et al., 1985; Jacobs, 1986; Londeree, 1986). However this high correlation for OBLA related variables versus race pace in general diminishes to moderate and low correlations when the groups tested become more and more homogeneous (Sjödin & Svedenhag, 1985). A new understanding of aerobic endurance is needed to further discriminate among elite athletes.

I.1.1 The Problem

The previously cited work of Stegmann et al. (1981) has shown large individual differences for a lactate threshold concept like the individual anaerobic threshold (IAT). Thus the area of blood lactate kinetics, unlike maximal aerobic power or lactate threshold determinations, holds promise of greater sensitivity to individual differences among well-trained elite aerobic endurance athletes.

Differences in lactate kinetics are apparent in incremental exercise testing in that highly trained athletes have their lactate response curves right-shifted compared to normal subjects (Heck et al., 1985). Hence elite, fit groups are capable of higher power outputs before they experience the inevitable rise in lactate concentrations

associated with incremental tests to maximal levels of effort (Sjödin & Svedenhag, 1985). Gimenez, Servera and Salinas (1982) have shown the same effects for endurance tests. In contrast to progressive incremental work testing the area of blood lactate recovery ability during endurance tests has not been as well studied. The Square-Wave Endurance Exercise Tests (SWEET) of Gimenez et al. (1982) are examples of endurance tests that are composed of intervals of work at greater than 70% maximal tolerated power followed by active recovery exercise at less than aerobic threshold (as defined by Kinderman et al. 1979). These authors suggest intratest blood lactate testing for peak to baseline work (Gimenez et al. 1982b). They further suggest an unexplained physiological phenomena or "transition phase" (p. 362) after 15 minutes of SWEET testing that creates a change in perceived exertion similar to the "second wind" (p. 362) concept.

If blood lactate concentration is a balance between production and removal (Brooks 1985), one or both of these elements is keeping blood lactate concentrations down in high fit subjects during initial work loads. Donovan and Brooks (1983) have suggested that endurance training enhances lactate clearance not lactate production. More recently Brooks (1990) has suggested a very slight decrease in La production as a result of training. Freund, Oyono- Enguelle, Heitz, Marbach, Otti and Gartner (1989) and Freund, Zouloumian, Oyono-Enguelle and Lampert (1984) have confirmed Donovan and Brooks' findings of trained subjects displaying enhanced La clearance. Also confirmed by Freund et al., (1984, 1989) were the findings of Belcastro and

Bonen (1975) that submaximal exercise intensities enhanced lactate clearance from blood (recovery) better than maximal exercise levels.

In a limited series of repeat bouts of exercise intervals lactate recovery ability was found to vary with the time course of exercise, whereas oxygen consumption repeatedly achieved the same level (Rieu, Duvallet, Scharapan, Thieulart & Ferry, 1988). This suggests that lactate recovery ability varies with time and may not be as O_2 dependent as suggested by Davis (1985).

Heck et al. (1985) critiqued the various LT testing procedures because they found that endurance capacity, work load intensity, and duration of exercise were all factors that affected levels of lactate in the blood. Therefore if work load intensity and duration of exercise are set then the variance in HLa concentration should be determinants of endurance capacity. Fixed blood lactate concentrations (FBLC) would equate relative intensities. Åstrand (1984) lauds repeat intervals of work as promising for fitness assessment.

SWEETs have been used previously to assess endurance capacity in a controlled interval protocol. Repeat intervals of work at physiological response levels that are highly correlated to race pace like the OBLA point, along with intervals at other lactate thresholds both above and below OBLA should provide a survey of lactate stress conditions in which to assess lactate recovery ability and endurance performance capacity. Using a three zone model (Stark 1989) the upper, lower, and middle points of the aerobic-anaerobic transition zone would result in lactate recovery

abilities being contrasted for predominantly aerobic work, aerobic to anaerobic transition work, and predominantly anaerobic work intervals. As with incremental exercise protocols repeat bouts of exercise for high fit athletes should elicit sharp accumulation values later for higher fit subjects than in lower fit subjects.

Hermansen and Stensvold (1977) have placed optimal lactate recovery ability at 63% $\dot{V}O_2max$ for high fit runners on a treadmill. Skinner and McLelland (1982) suggest the first significant rise in blood lactate as the optimal individual lactate recovery intensity.

Lactate recovery ability can be assessed as an indicator of aerobic endurance capacity if controls are in place for exercise duration and intensity (Heck, et al. 1985) and athletes' diet (Yoshida, 1986). If different values of blood lactate concentration are taken for an individual on the same lactate response curve then right or left shifting of that curve by high or low carbohydrate diets will be controlled for and differences in lactate recovery ability could be attributed to aerobic endurance capacity.

I.1.2 The Purpose of the Study

The purpose of this study was to describe the effects of three selected exercise intensities on blood lactate recovery ability in male distance runners during treadmill running exercise. Changes in the patterns of heart rate, oxygen consumption and blood lactate responses at the three selected intensities was also of interest.

I.1.3 Null Hypothesis

There will be no effect of the three selected exercise intensities on blood lactate recovery ability.

I.1.4 Limitations

The finding of this study can only be generalized to male well trained varsity cross-country runners who are tested on a motor driven treadmill using a similar SWEET protocol.

I.1.5 Definitions and Abbreviations

The onset of diminished lactate recovery occurred when the difference in the blood lactate concentration between the end of the set work interval and the end of the set active recovery interval was significantly decreased from the first repeat to a subsequent trial.

The reversal of lactate recovery occurred when the difference in the blood lactate concentration value between the end of the work interval and the end of the active recovery interval was negative following a previous positive difference.

Optimal lactate recovery is the point where not only the lactate level from both the over and under stages are minimal, compared to all other stages, but also the difference from the over stage blood lactate concentration value to the corresponding under stage blood lactate concentration value (delta lactate) is minimal for the testing

session.

IAT - individual anaerobic threshold

MSS - maximal steady state of blood lactate concentration

LT - lactate threshold

FBLC - fixed blood lactate concentration(s)

OBLA - onset of blood lactate accumulation

OPLA - onset of plasma lactate accumulation

AT - also AnT or AT_c - anaerobic threshold

AT_n - AT determined noninvasively through gas exchange

AT_i - AT determined invasively through blood sampling

AerT - aerobic threshold

RCT - respiratory compensation threshold

TDMA - threshold of decompensated metabolic acidosis

LSM - lactate slope method

CLW - constant load work

IW - incremental work

MAP - maximal aerobic power

$\dot{V}O_2max$ - maximal oxygen consumption per one minute

LA - undissociated lactic acid

La - dissociated lactic acid as the anionic salt lactate

[La] - lactate concentration

[HLa] - blood lactate concentration

ATP - adenosine triphosphate

CP - creatine phosphate

Pi - inorganic phosphate

Cr - creatine

FT - fast twitch muscle fibre

ST - slow twitch muscle fibre

SO - slow oxidative fibre

FOG - fast oxidative glycolytic fibre

FG - fast glycolytic

NAD - nicotinamide adenine dinucleotide

NADH + H^+ - reduced form nicotinamide adenine dinucleotide

H^+ - hydrogen ion

LDH - lactate dehydrogenase

PDH - pyruvate dehydrogenase

LDH-H - lactate dehydrogenase (heart form) isoenzyme

LDH-M - lactate dehydrogenase (muscle form) isoenzyme

NaLa - Sodium lactate

PFK -- phosphofructokinase (e.c. 2.7.1.11)

CHAPTER TWO

II.1 Review of Literature

II.1.1 Introduction

In studying the trend in fitness evaluation from traditional MAP tests to newer LT response classifications the following general areas were examined: a general review of the physiology of aerobic exercise; various tests of aerobic power and capacity; and reclassifying [HLa] response values relative to a La baseline. The review of the physiology of aerobic exercise included muscle structure and function; the ATP molecule; the three pathways of energy production; LDH activity; metabolic control; exercise induced fatigue; and neural recruitment. The review of tests of aerobic power and capacity included $\dot{V}O_2max$ tests; the relationship between running performance and submaximal physiological variables; indirect and direct lactate threshold tests; fixed [HLa] determination; and square-wave endurance exercise tests. The review of reclassifying [HLa] responses relative to baseline values encompassed production, membrane transport, and removal of HLa. The HLa removal studies reviewed expressed La recovery values in both absolute values and relative values using baseline La values as the reference points.

II.1.2 The Physiology of Aerobic Exercise

A brief review of the physiology of aerobic endurance exercise involves muscle structure and function; the adenosine triphosphate molecule; the three metabolic pathways of the aerobic energy system of the body; the lactate dehydrogenase

molecule; the metabolic and neural control of metabolism; exercise induced fatigue; and neural recruitment.

II.1.2.1 Muscle structure and function

Structurally muscle is made up of four proteins that are responsible for muscle contractions. Troponin, which has a calcium binding site, has a globular structural unit which is intermixed with other globular units of actin to make a two strand, fibrous, twisted rope-like strand. Overlaid on the actin filaments is the tropomyosin helical strand. The shifting of this strand, caused by the binding of calcium to troponin, causes the actin binding sites to be unmasked. The thick rod-like myosin molecules are interdigitated with actin whereby cross bridges of myosin are in close proximity to actin binding sites. The myosin cross bridges have projections called myosin heads which contain actin binding sites and ATP binding sites. Two speeds of actin myosin binding is regulated structurally by the activity of myosin ATPase enzyme found at different myosin heads. FT fibres have a predominance of myosin heads higher in ATPase activity than ST fibres (Lehninger, 1982; Brooks & Fahey, 1984).

Functionally muscle is activated by a neural release of acetylcholine (ACH) at the alpha motor neurons neuromuscular junction. Binding of ACH to the muscular membrane evokes a depolarization which is propagated along the membrane and down the transverse tubules to cause the lateral sacs of the sarcoplasmic reticulum to release calcium ion (Ca^{2+}) into the cytoplasm. It is this release and eventual uptake which

triggers and releases actin-myosin binding by the tropomyosin shift (Brooks & Fahey, 1984).

The release of the myosin cross bridges and the uptake of calcium by the sarcoplasmic reticulum are both endergonic reactions. Energy for these procedures is ultimately provided by the hydrolysis of the adenosine triphosphate molecule (ATP) (Brooks & Fahey, 1984).

II.1.2.2 The ATP Molecule.

Exergonic reactions of membrane pumping against an osmotic gradient or the release of bound molecules, as exemplified above in the sliding muscle filament action, is mainly supplied by the energy released by the hydrolysis of covalent bonds of phosphate to adenine. As such adenosine triphosphate is the major intermediary between exergonic and endergonic reactions. It is composed of a nitrogenous base (adenine), a five carbon sugar (ribose), and three phosphates. This molecule with one covalently bond phosphate is called adenosine monophosphate (AMP), with two phosphates bond, adenosine diphosphate (ADP), and with three phosphates bond, adenosine triphosphate (ATP). The ATP bond conserves about 7.3 kcal of energy when a third phosphate molecule is covalently bound to ADP to make ATP.

The terminal group of ATP is negatively charged such that they are usually associated with a magnesium ion (Mg^{2+}). As such, magnesium is a cofactor for ATP reactions (Brooks & Fahey, 1984).

II.1.2.3 Three Metabolic Pathway Systems

The "common chemical intermediate" (Brooks & Fahey, 1984, p. 60) ATP is supplied by three distinct energy systems. They are called the phosphogen system, the glycolytic system, and the oxidative system after the major steps in the respective pathways that result in ATP production.

II.1.2.3.1 The Phosphogen System.

The phosphogen system is composed of muscular stores of ATP and creatine phosphate (CP). CP can transfer its phosphate group to ADP via the *creatine kinase* (E.C. 2.7.3.2) reaction.

$$CP + ADP \xrightarrow[creatine\ kinase]{MG^{2+}} ATP + Creatine$$

There are about 3 to 4 times as much CP in muscle as ATP, therefore the first energy responses for replenishing hydrolysed ATP by rephosphorylating ADP with a free phosphate is the reversible *creatine kinase* reaction. A second step that releases a phosphate group and provides energy is the *adenylate kinase* reaction below.

$$ADP + ADP \xrightarrow[adenylate\ kinase]{MG^{2+}} ATP + AMP$$

In muscle this enzyme is called *myokinase* (E.C. 3.6.1.3). This enzymatic reaction is also an energy reserve which can be tapped to resynthesis ATP for muscular use. The time course of this system as a predominate energy source is

limited to about 10 to 15 seconds when muscles are contracting at full intensity (Green in MacDougal, Wenger & Green, 1982). The total energy capacity available out of this system in continuous maximal muscle use is about 30 seconds (Edington & Edgerton, 1976 cited in Brooks and Fahey 1984). As such this energy system which shuttles phosphates is called the "phosphogen system" (Lehninger, 1982, p. 384).

This energy system has also been termed the alactic energy system because La production is not a consequence of ATP production. This system can be trained. Karlsson (1971) has shown that for the trained (T) and the untrained (U) marked CP depletion can be seen in submaximal exercise but that ATP depletion could only be observed with maximum or near maximum work loads. Yet there was a marked difference in the two groups when comparisons where made in absolute terms. Relative work loads at 75 and 100% maximum showed the same degree of CP depletion for the two groups. As well at 90 to 100 %$\dot{V}O_2$max phosphogens were depleted to maximum levels after 2 minutes whether the subjects were exhausted or not (Karlsson 1971). Karlsson (1971) has shown that phosphogens were depleted to about 4 to 5 mM/kg wet muscle before [La] increased beyond baseline rest values. The existence of La accumulation in working muscle when phosphogens were not fully depleted suggested concomitant energy production from phosphogens and the glycolytic pathway.

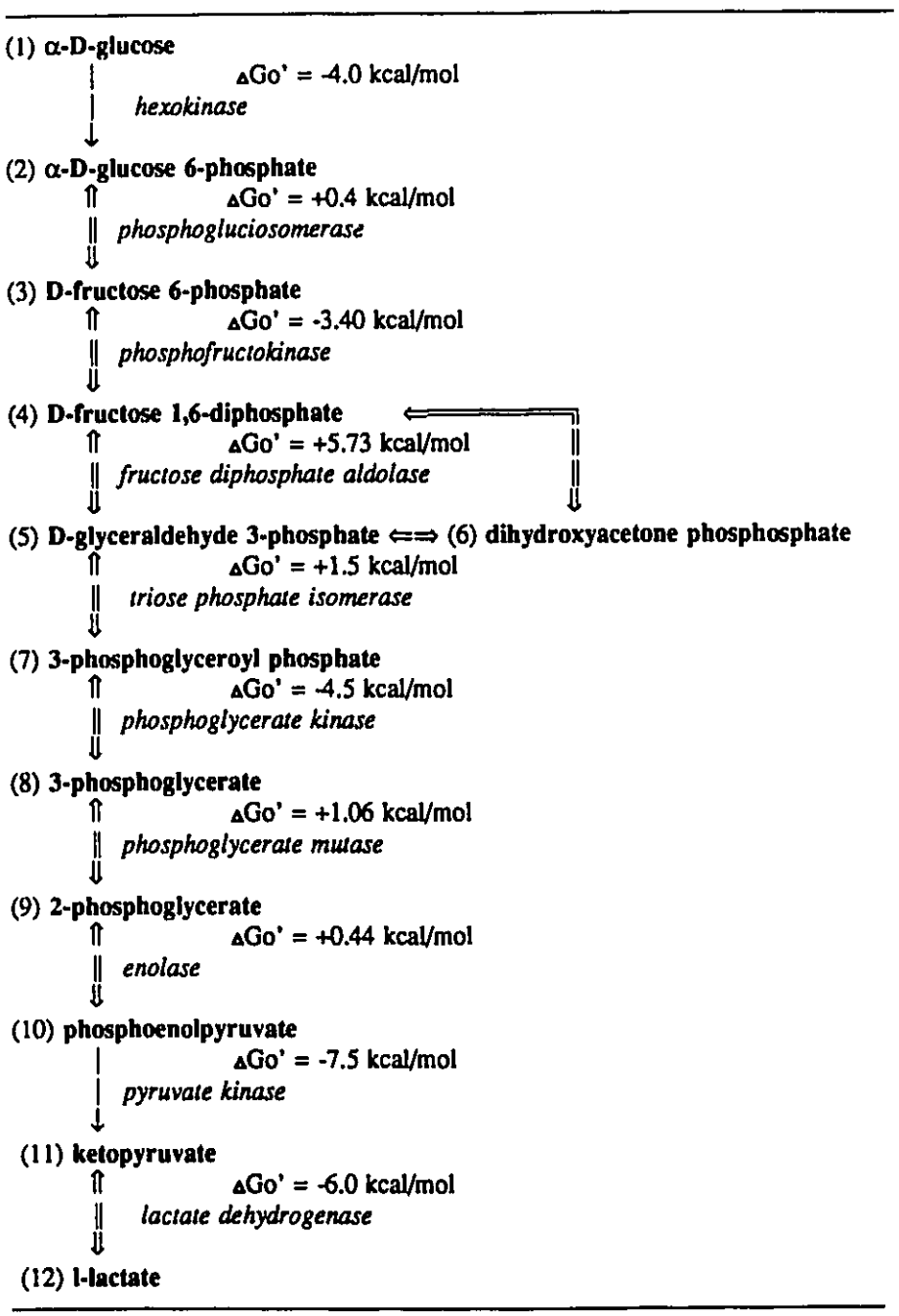
II.1.2.3.2 The Glycolytic System.

The glycolytic energy pathway provides energy through the two possible routes of substrate phosphorylation or oxidative phosphorylation. In glycolysis a six carbon sugar is irreversibly transported across the cell membrane, phosphorylated twice and broken down into two identical three carbon sugars as depicted in Figure I1 (p. 16). The rate limiting step (3) is catalysed by *phosphfructokinase* (E.C. 2.7.1.11) (PFK) in an endergonic, irreversible reaction ($\Delta GO' = -3.40$ kcal/mol). PFK is considered to be one of the most complex regulatory enzymes known (Lehninger, 1982) with its sensitivity due in part to the fact that it is situated at a substrate cycle and partly to the fact that it has multiple internal and external regulator on the substrate flux (Newsholme, 1988).

In steps 5 to 7 NAD^+ is reduced to NADH + H^+. As NADH + H^+ is created a crucial limit can be placed on the glycolytic flux. NAD^+ occurs in limited concentrations in the cytosol and is a necessary substrate for further carbon flow through this step 5. Thus a crucial limit to glycolytic flow is created by NAD availability. Step 11 to 12 may reverse this limiting process by oxidizing NADH + H^+ to yield NAD^+. If the rate of NAD^+ needed exceeds that being returned to it via the mitochondria glycolysis will slow.

Step 11 proceeds for two main reasons. One, if oxygen is limited to the mitochondria and electrons cannot be transferred from NADH + H^+ to molecular oxygen to reform the necessary cytosolic NAD^+. Two, if the glycolytic sequence is

Figure 11. The 12 steps of glycolysis with **intermediates**, energy equations and *catalysing enzymes* modified from Lehninger (1982), Principles of Biochemistry.



fluxing at a rate which cannot be matched by the mitochondria's output of NAD^+ or intake of pyruvate, then pyruvate will be dehydrogenated to yield La and the necessary NAD^+. The final control of the glycolytic sequence by the regulatory enzyme *lactate dehydrogenase* (E.C. 1.1.1.27) (LDH) is crucial as to whether La is produced or removed.

II.1.3.3 The Lactate dehydrogenase molecule.

There are four basic subunits of the *lactate dehydrogenase* molecule (LDH). These subunits combine to produce five different isoenzymes forms, which differ in their V_{max} and Km values for pyruvate. Each subunit contains different amounts of two kinds of polypeptide, one termed H (or A) because it predominates in heart tissue, and the second termed M (or B) for it predominates in skeletal muscle. The five forms of LDH molecule in the body are A4, A3B, A2B2, AB3, and B4.

The LDH isozyme A4, heart type (H4), has a "low Km for pyruvate and is strongly inhibited by pyruvate, whereas the muscle isoenzyme (designated M4) has a higher Km for pyruvate, is not inhibited by pyruvate, and is more active catalytically" (Lehninger, 1982, p. 413).

There are two possible outcomes for a six carbon glucose molecule in the glycolytic sequence. One is the two molecules of La, two hydrogen ions (H^+) two molecules of ATP, and two water molecules, as depicted in this summary equation.

$$Glucose + 2P + 2ADP \xrightarrow{LDH} 2\ La^- + 2H^+ + 2ATP + 2H_2O$$

The second possible outcome is two molecules of pyruvate, two molecules of ATP, two reduced hydrogen carriers (NADH + H^+), and two water molecules, as depicted in (4) from Lehninger (1982, p. 414).

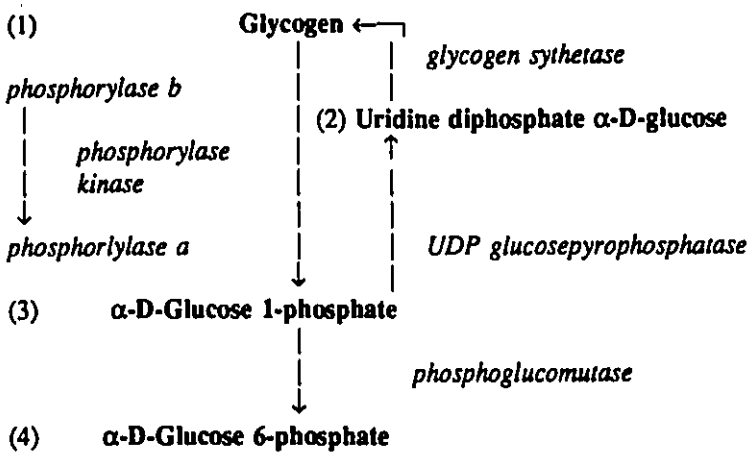
$$Glucose + 2P + 2ADP \xrightarrow{LDH} 2\ Py^- + 2NADH + 2H^+ + 2ATP + 2H_2O$$

II.1.2.3.4 Glycogenolysis.

Entry into the glycolytic sequence can also occur from stored chains of 6 carbon sugars called glycogen, see Figure I2.

Figure I2.

Glycogenolysis with **intermediates** and *catalyzing enzymes.*



The glycolgenolytic sequence does not require the ATP used by the glycolysis sequence to transport glucose across the membrane. Therefore less energy is

The glycolgenolytic sequence does not require the ATP used by the glycolysis sequence to transport glucose across the membrane. Therefore less energy is consumed when using stored muscle sugar versus blood sugar.

The second outcome of glucose is the precursor to the aerobic phase of catabolism, called respiration which occurs inside the mitochondria. To further metabolize pyruvate, it must gain access to the mitochondria where further substrate and oxidative phosphorylation can occur. For each carbon atom left in pyruvate a CO_2 molecule must be produced, with the hydrogens attached to these same carbons atoms of pyruvate transported to the sites of respiration. The entrance of glycogen into the glycolytic sequence is modulated by a transformation of *phosphorylase b* to *phosphorylase a.* Calcium released by the sacroplasmic reticulum as a result of neural activation links fuel availability with sliding filament action. (Brooks & Fahey, 1984).

A series of 5 enzymatic steps decarboxylates and dehydrogenates pyruvate yielding CO_2, NADH + H^+ and acetate. Acetate combines with a cofactor, coenzyme A (CoA) to form acetyl-CoA, which is what enters the Kreb's cycle. But before acetyl-CoA enters the mitochondria two reducing equivalents are passed to NAD, via flavin adenine dinucleotide (FAD). The *pyruvate dehydrogenase enzyme* complex is located in the mitochondrial membrane and consists of three enzymes: *pyruvate dehydrogenase*, *dihydrolipoyl transacetylase*, and *dihydrolipoyl dehydrogenase.* The following is a summary equation.

$$\textbf{Pyruvate} + \textbf{NAD}^+ + \textbf{CoA-SH} \rightarrow \textbf{acetyl-CoA} + \textbf{NADH} + \textbf{CO}_2$$

II.1.2.3.5 Kreb's Cycle.

The Kreb's cycle continues to catabolize carbon skeletons of pyruvate but unlike the linear glycolytic sequence, it progresses circularly, recycling its intermediates in an 9 step process, see Figure 3 (p, 18).

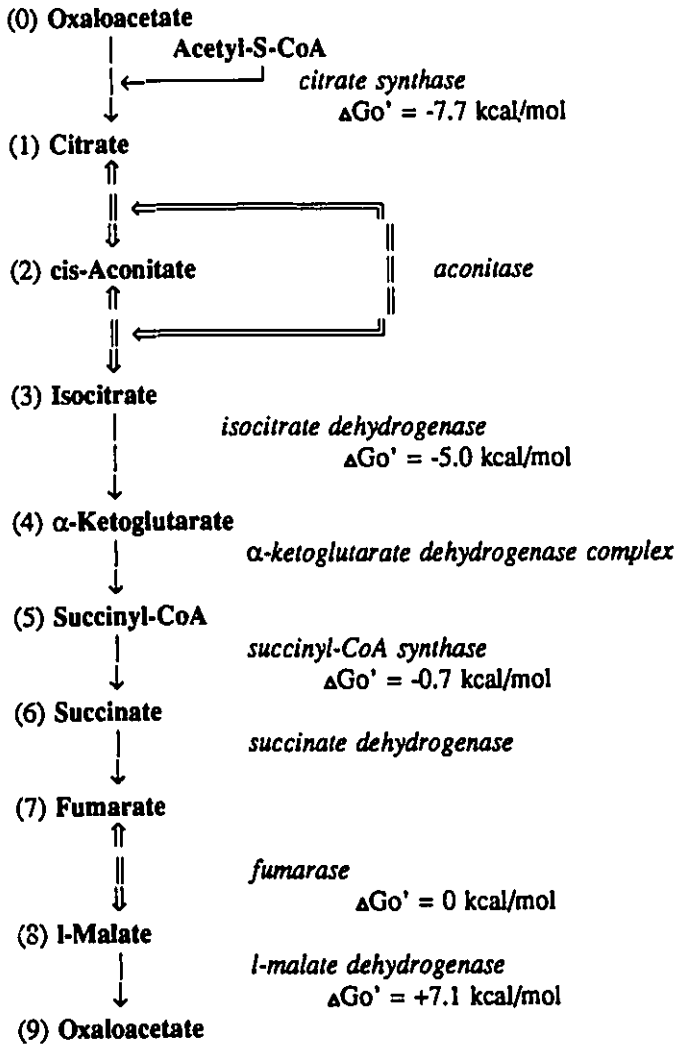
Once acetyl-CoA is available to the cycle each turn of the cycle releases two carbon molecules as CO_2. The hydrogens bond to the carbons are passed to the electron transport chain via reducing equivalents, three NADH's and one FADH for each 2 carbon acyl-CoA entry into the cycle.

II.1.2.3.6 Electron Transport Chain.

The final common step to aerobic metabolism is the oxidative phosphorylation that takes place in the electron transport chain (ETC). Several carriers of electrons like NADH, FADH, NADPH, carry in single electron equivalents, named reducing equivalents, to a path that leads eventually to reactions with molecular oxygen. As electrons pass from reducing equivalents to conjugate redox pairs, they are flowing from relatively electronegative (E'o = -0.32 mV) electron donators like NADH to relatively electropositive (E'o = +0.82) electron acceptors pairs like H_2O-O_2.

As two pairs of electron pass along the ETC from NADH to O_2, 52.6 kcal are released. As with substrate phosphorylation, oxidative phosphorylation conserves the released energy by phosphorylating ADP to ATP. As each ATP synthesized requires 7.3 kcal of energy the three active sites of energy conservation of the ETC can

Figure I3. Kreb's Cycle pathway intermediates with energy equations and *catalyzing enzymes*. The 9 steps of the citric acid cycle with descriptive equations of the reactions and the free energy changes involved modified from Lehninger (1982), Principles of Biochemistry.



therefore conserve 3 X 7.3 or 21.9 kcal of energy in the form of ATP molecules. Yet some reducing equivalents like FADH only enter the ETC after the first energy conserving site, and therefore only synthesis 2 ATP molecules for each reducing equivalent.

The current mechanism thought responsible for energy conservation is the chemiostatic hypothesis (Lehninger, 1982). In brief it proposes that an intact inner mitochondrial membrane pumps out H^+ from the matrix to the outer aqueous solution. The resulting osmotic gradient supposedly drives the endergonic synthesis of ATP (Lehninger, 1982).

Associated with the glycolytic sequence, Kreb's cycle, and the ETC are feeder and shuttle systems. Two systems that transport reducing equivalents into the mitochondria are the glycerol-phosphate shuttle and the malate-aspartate shuttle. The "glycerol-phosphate shuttle is the main mechanism in skeletal muscle", whereas "the malate-aspartate shuttle is the main mechanism in the heart" (Brooks & Fahey, 1984, p. 80).

The sequence of the malate-aspartate shuttle starts in the cytosol and is oxaloacetate (OAA) to aspartate, which crosses the membrane and reverses back to OAA, to malate, which crosses back across the membrane, to OAA. As OAA proceeds to go to malate inside the mitochondria in a reversible reactions NADH is oxidized to NAD^+. This process is reversed in the cytosol.

The sequence for the glycerol-phosphate shuttle is dihydroxyacetone phosphate

Figure I4.

Beta oxidation with **intermediates** and *catalyzing enzymes*. Acyl-CoA is reused with FADH and NADH producing 2 and 3 ATP each. The last step of beta oxidation is the rate limiting step (Brooks & Fahey, 1984).

---

(1) saturated fatty acyl-Coa

↓ *fatty acyl dehydrogenase*

(2) α-ß-Unsaturated fatty acyl-CoA

↓ *enol hydrase*

(3) ß-hydroxyacl-CoA

↓ *hydroxyacyl dehydrogenase*

(4) ß-ketoacyl-CoA

↓ *ß-kethothiolase*

(5) saturated fatty acyl-CoA

---

to glycerol phosphate in a one way reaction via cytoplasmic *glycerol phosphate dehydrogenase*, which also yields NAD^+ from NADH + H^+ in a one way reaction. Glycerol phosphate crosses the membrane and mitochondrial glycerol phosphate dehydrogenase brings glycerol phosphate to dihydroxyacetone phosphate, with FAD reduced to FADH.

As mentioned earlier FADH + H^+ only produces two ATP in the ETC, whereas NADH + H^+ produces three ATP.

II.1.2.3.7 Beta Oxidation.

Another feeder pathways into the Kreb's cycle is beta oxidation, a fat catabolism pathway. The enzymes for this pathway are located in the mitochondrial matrix and are summarized in Figure I4 (p. 23). The second step is a rate limiting step of transportation across the mitochondrial membrane via the actions of *carnitine transferase* (carnitine palmitoyltransferase E.C. 2.3.1.21). Once inside the mitochondria two carbon atoms are cleaved off the chain with each flux of the pathway such that products are acetyl-CoA, FADH and NADH.

II.1.2.4 Metabolic Control

The regulation of glycolytic flux occurs in three points along the pathway. One the entry of glucose into the cell is allostericly controlled by the *hexokinase* (E.C. 2.7.1.1) enzyme. Hexokinase is an enzyme which is regulated allostericly (internal regulation) by its reaction product glucose-6-phosphate (Newsholme 1980). The second point of entry control in the glycolytic sequence is *phosphorylase* (E.C. 2.4.1.1). External regulators affect the substrate cycling at *PFK* and *phosphorylase* either facilitating or inhibiting the carbon flux. Stored glycogen enters the glycolytic pathway through the action of *phosphorylase a*, the catalytically active form of the enzyme; whereas, *phosphorylase b* is the dephosphorylated inactive form of the enzyme. The main effectors of these enzyme forms are cyclic AMP (cAMP), the intracellular hormone, and intracellular calcium (Ca^{2+}). The Ca^{2+} ion has the quickest

effect on glyconeolytic control by modulating *phosphorylase b kinase* to *phosphorylase a*. Epinephrine and norepinephrine binding to the cell membrane causes a cascade of reactions that result in cAMP action on phosphorylase b to convert it to phosphorylase a.

Other points of metabolic control of glycolysis include the most complex, that of the *phosphofructokinase (PFK)* reaction, the *pyruvate kinase* reaction, as well as the *glyceraldehyde 3-phosphate dehydrogenase* reaction. The rate limiting step catalysed by PFK is controlled by allosteric modulation via high or low concentrations of ATP, ADP, AMP, Pi, fructose 1,6 diphosphate, Mg^{2+}, and citrate. Mg^{2+} and citrate are the two most important inhibiters whereas AMP and fructose 1,6 diphosphate are the most active stimulatory modulators. No satisfactory explanation in any hypothesis can resolve the magnitude by which glycolysis increases 1000-2000 fold during maximal muscular contraction. Thus the glycolytic rate, which is sometimes seen as synonymous with flux through PFK, is linked to the contractile process without sufficient explanation (Katz & Sahlin, 1990)

The *pyruvate kinase* reaction is inhibited by ATP creating a low affinity for phosphoeneolpyruvate in the reaction. Other inhibiters are acetyl-CoA and long chain fatty acids. As such these fuels for the citric acid cycle slow glycolytic flux if these fuels are in high concentrations (Lehninger, 1982). Trained athletes reduce glycolytic flow by FFA inhibition of glycolysis and hence suppress La production (Holloszy & Coyle, 1984).

Holloszy and Coyle (1984) cite greater mitochondrial enzymes density especially in the malate-aspartate shuttle and a complete conversion of type IIb fibres to type IIa as training adaptations in highly fit athletes. This greater shuttling ability plus less La production in FT fibres is accompanied by shifting isomerase patterns of the LDH molecule towards more H type -- all leading to an increased ability of the mitochondria to "compete with lactate dehydrogenase for pyruvate" (p. 836). Also reported was a slower utilization of glycogen with a greater oxidation of fat.

The citric acid cycle is regulated by entry of substrates into its sequence similar to the glycolytic sequence. The *pyruvate dehydrogenase* (E.C. 1.2.7.1) complex (PDC) is regulated through allosteric modulation. When ATP is in high concentrations *pyruvate dehydrogenase kinase* phosphorylates pyruvate dehydrogenase to yield its inactive form pyruvate dehydrogenase phosphate. This response is also stimulated by Ca^{2+} rise inside the cell which provides a second immediate input by Ca^{2+} on catabolic pathways. This couples the signal for muscle contraction with fuel supply for ATP resynthesis through substrate or oxidative phosphorylation. Katz and Sahlin (1990) find no "convincing evidence that Ca^{2+} is the agent that is directly responsible for contraction-mediated glycolysis" (p. 10).

Other inhibiters of the PDC are acetyl-CoA a product inhibition and NADH. When long chain fatty acids are present pyruvate oxidation inhibition is enhanced by an extra supply of acetyl-CoA units (Lehninger, 1982).

In the citric acid cycle the *citrate synthase* catalysed reaction is the rate-

limiting step. The most crucial factor modulating citrate formation is the concentration of its substrate oxaloacetate (OXA), which is usually in low concentrations. Any small absolute change in OXA change thus has a large change in the rate of the forward reaction. Succinyl CoA provides product inhibition if its levels rise above normal. Citrate and NADH are also inhibiters in some cells (Lehninger, 1982).

The electron transport chain (ETC) is regulated through acceptor availability of ADP and partial pressure of oxygen. If sufficient quantities of either are not present respiration will not take place. Two measures of ADP have been suggested as control factors.

The first was the mass action rate of the ATP systems, represented by the ratio of ATP concentrations to the product of inorganic phosphate and ADP concentrations.

$$\frac{[ATP]}{[ADP]\ [P]} \qquad (6)$$

ATP is formed only when the ratio declines and ADP levels are up. The rate of the ETC fluxes accordingly, rephosphorylating ATP to drive the ratio up again. This ratio is also referred to as the phosphorylation potential (Gollnick, Moore, Riedy &

Quintinskie, 1984).

The second measure was called the energy charge. It is a measure "of the extent to which the total adenine nucleotide system" (ATP, ADP, AMP) is filled with "high-energy phosphate groups" as depicted below in equation (7) (Lehninger, 1982).

$$ENERGY\ CHARGE = \frac{[ATP] + \frac{1}{2}[ADP]}{[ATP] + [ADP] + [AMP]} \qquad (7)$$

Another control mechanism suggested in the metabolic pathway is the concentration ratios of NADH and NAD^+ both inside and outside the mitochondria. In the glycolytic sequence a high redox potential is reflected by a high ratio of [NADH]/[NAD]. Because NAD is in limited quantities in the cytosol, insufficient NAD produced by the malate-aspartate shuttle from the mitochondria results in the formation of La from pyruvate with the concomitant oxidation of NADH + H^+ to NAD. Thus the *glyceraldehyde 3-phosphate dehydrogenase* reaction can continue with a fresh supply of NAD. (See Figure 11, p. 16, step 6). This mass action effect on the LDH reaction is one explanation for the pyruvate to La step.

The general principles of enzyme kinetics also explains the greater capacity shown by trained subjects. Substrate molecules have a greater chance of random encounters with the catalysing enzymes to form enzyme substrate complexes when the density of mitochondria in skeletal muscle fibres is elevated due to training adaptations. This increases the velocity of the reactions for the same substrate

concentrations (Gollnick et al., 1984). Hence less La is supposed to be produced for a given submaximal load in the trained versus the untrained state. This has been controversial for a number of years but recently Brooks (1990) has conceded this possibility (Donovan & Brooks, 1983).

II.1.2.5 Exercise Induced Fatigue

In identifying causes of fatigue Brooks and Fahey (1984) recognize that it is not a simplistic process. The difficulty lies in separating "causality from concurrent appearance" (p. 70).

Peripheral fatigue will be examined first. Brooks and Fahey (1984) report two phases to CP depletion, one rapid, immediately at the onset of exercise, and two, slightly later till the end of the exercise bout. The extent of the initial and final drop in the ATP, CP levels are a result of the "relative work intensity" (p. 702). They report that at the point of fatigue both CP and ATP levels are relatively depleted. Yet Fitts and Metzger (1988) have shown fatigue not totally related to ATP CP depletion with a different time course for ATP and CP depletion and repletion versus peak tension for isolated muscle studies.

Shephard (1984) distinguishes between a central component as cause of fatigue and a peripheral component as site of fatigue. At $\dot{V}O_2max$ levels of exertion the limiting factors are maximal cardiac output limiting the O_2 transport from the lungs to contracting skeletal muscle (Brooks and Fahey, 1984; Åstrand, 1984). There is thus a

reliance on glycolytic ATP production with its consequent metabolic acidosis. At the site of fatigue loss of muscle action is attributed to a pH-modulated inhibition of *phosphorylase* and *PFK* that halts substrate "transfer of energy from glycogen through phosphogen to the actin-myosin cross-bridges" (p. 104).

The acidic form of Pi (H_2PO_4) has been implicated as one factor in the decline of force production (Fitts et al. 1988). The H^+ ion affect and Ca^{2+} ion affect can be summarized below from Fitts et al. (1988) as

> ... direct inhibition of actomyosin ATPase activity and ATP hydrolysis,...and a build up of H^+ ion could induce fatigue by:(1) inhibiting phosphofructokinase and thus the glycolytic rate; (2) competitive inhibition of Ca^{2+} binding to troponin C reducing cross-bridge activation, and inhibiting the SR-ATPase reducing Ca^{2+} reuptake and subsequently Ca^{2+} release (p. 221).

As such the effect on calcium cycling is to increase the transit time of calcium ion in all its flux steps.

Shephard (1984) cites three main causes of the increased acidosis due to enhanced La accumulation. One the electron transport chain of the mitochondria was receiving insufficient O_2. Secondly, a slow La efflux from muscle fibres causes accumulation. Thirdly a delayed malate-aspartate shuttle transport of La from the cytosol into the mitochondria did not decrease the accumulated La.

In contrasting central to peripheral fatigue Shephard (1984) found that exercise employing a large muscle mass, like running or skiing, elicits impending circulatory failure at $\dot{V}O_2max$ efforts. Whether this comes about from a deficiency of venous return or an inadequate stroke volume in light of rising peripheral resistance is not

clear. Brooks and Fahey (1984) cited heat accumulation and the shunting of blood to subcutaneous tissue as diverting some of the cardiac output from working musculature as a possible central fatigue cause.

[HLa] accumulation is diminished by buffering. For example lactic acid (LA) and sodium bicarbonate ($NaHCO_3$) form sodium lactate (NaLa) and carbonic acid (H_2CO_3).

$$HLa + NaHCO_3 \rightarrow NaLa + H_2CO_3 \qquad (8)$$

In the body carbonic acid is a weak acid that does not give up many H^+ ions. It dissolves into water and carbon dioxide. Thus a summary equation for the buffering of lactic acid is presented in (9) below.

$$HLa + NaHCO_3 \rightarrow NaLa + H_2CO_3 \longleftrightarrow NaLa + H_2O + CO_2 \qquad (9)$$

When lactic acid or La and H^+ influx into the blood outpacing the bicarbonate buffering system's ability to compensate for the metabolic acidosis, then the Ph falls.

Beaver, Wasserman and Whipp (1986) have studied the pattern of decrease in arterial bicarbonate concentration ($[HCO_3^-]$ during progressive incremental exercise testing. The authors have compared in detail the drop in arterial blood HCO_3^- with the increase La concentration ($[La^-]$) both to determine the degree of buffering of lactic acid and to determine if other buffering mechanism may be present.

Using the HLa inflection point method which is based on an intersecting point for two regression lines of the log of $\dot{V}O_2$ versus the log of $[La^-]$(Beaver, Wasserman

& Whipp, 1985), Beaver et al. (1986) found "close reciprocity" between the decrease in bicarbonate and the increase in La (p. 476). This validated their presumption that the major buffering mechanism in extracellular fluids and plasma is the bicarbonate system. Thus bicarbonate functions to stabilize pH, since HCO_3^- continually disappears due to ventilatory excretion -- expiration of the CO_2 that immediately formed from dissociating H_2CO_3. The function of graphing the log of HCO_3^- with the log of $\dot{V}O_2$, a log-log transformation of the data, showed a small lack of fit. This was suggested to be a function of the quantitatively small intracellular and extracellular non-bicarbonate buffering system.

The log-log transformation method of plotting [La] recently has been shown to have a greater error than a method of plotting where [La] is depicted as a continuous increasing exponential function (Campbell, Hughson & Green; 1989). This has two implications. First that the small lack of fit between the decrease in HCO_3^- (it was right shifted by a constant) and the increase in La that was conceptualized as another buffer system may not hold. What was considered to be other buffering agents may not affect bicarbonate buffering. Secondly there may not be a distinct physiological threshold for HLa increases. This will be further delineated in the next section on tests of power and capacity. Thus most buffering of lactic acid in the body is accomplished by the bicarbonate system.

Mainwood, Renaud and Mason (1986) have conducted fatigue studies on in vitro frog sartorius muscle. They indicate that following prolonged activity the

contractile response of the skeletal muscle is generally reduced, La efflux slows, and intracellular and extracellular acidosis increases. By studying the relationship between pH, lactic acid in associated or dissociated form and contractile activity, Mainwood et al. (1986) have found that lowering extracellular pH prolongs the suppression of contraction and slows the rate of reduction of intracellular La. The slowing of the La efflux appears to be due to the fact that extracellular pH controls the rate of proton-linked La efflux across the cell membrane. The flux rate is diffusion limited where "there is facilitated diffusion of protons by an adequate buffer system in the extracellular compartment" (Mainwood et al. 1986, p. 654). This proton linked facilitated diffusion membrane transport system works in conjunction with lipid bilayer efflux of undissociated lactic acid and an ionic flow of La and hydrogen ions across the membrane. Only the first two systems' flow rates are a function of the extracellular buffer concentrations and hence only they slow La efflux if extracellular $[H^+]$ is high.

Intracellular and slightly later extracellular acidosis from La production are two of the 'concurrent appearances' that are associated with reduced muscle isometric tension development in these in vitro experiments. Loss of isometric tension as a measure of fatigue has been affected by high extracellular pH. A slight but not statistically significant tendency to decrease fatigue along with a faster recovery from fatigue are consequences of an elevated extracellular pH (8.0). Conversely Mainwood et al. (1986) found that low pH values (6.2 to 6.4) suppresses isometric contractile

force slightly and slow recovery. Therefore the recovery progress is not due to the extent of the local fatigue but to the external pH.

Low external pH affects the membrane characteristics directly by prolonging membrane depolarization by 10-12 mV, increasing transmembrane resistance, and prolonging action potentials. Even though low extracellular pH affects the membrane directly and limits La efflux, the resulting intracellular acidosis cannot account for more than 20 - 30% of the 80% suppression of isometric contraction as shown in Mainwood et al. (1986) in vitro muscle preparations experiments. The authors have shown a direct and reversible effect on the rate of relaxation. They further speculate that both acidosis and depleted ATP diminish Ca^{2+} uptake reducing the available Ca^{2+} pool that can be released.

In contrast to in vitro muscle preparations Cady, Jones, Lynn and Newham (1989) examined the relationship between intracellular metabolites and the generation of force during fatigue in situ using the first interposes muscle. They contrasted normal subjects with myophosphorylase deficiency (MPD) subjects over repeated trials. With MPD subjects, force recovered to a greater extent, raising the possibility that the continued acidosis in the normal subjects may have depressed force recovery. Cady, Jones, Lynn and Newham (1989) cite hydrogen ion as being widely reported as a depressant of force production in both skinned and intact preparations. In their experiments normal subjects followed the pattern of a loss of force matched by an increase in $[H^+]$. The subject with MPD showed a force loss that was independent of

H^+ accumulation as pH rose during her exercise. The authors reported that in MPD muscle there was clearly a mechanism causing a loss of force that was independent of H^+ accumulation. When the blood supply was restored to the fatigued muscles, PCr was synthesized and force recovered despite the continued low pH during the first 60 seconds. But in normals there was also a pH dependent mechanism that showed for a given level of intracellular PCr a force was generated during recovery that was less when pH was low. This lead the authors to conclude that there are two possible fatigue mechanisms one pH dependent and one not that lead to a loss of force along several concurrent metabolic changes.

Bountra and Vaughan-Jones (1989) confirmed this same pattern of low intracellular pH inhibiting contraction tensions in in vitro cardiac tissues. This result was consistently elicited with low extracellular pH. Twitch tension was found to be a function of a constant k and the intracellular hydrogen ion concentration to the negative power of x. This decreasing exponential function is described in equation (10).

$$\textit{twitch tension} = k\,[H^+]_i^{-x} \qquad (10)$$

The mean value for x was 1.45 ± 0.33 for a pH of 6.4 from 7.4.

A second finding was that intracellular sodium activity (a_{Na}^i) acts to modulate the contractile response of cardiac muscle to acidosis. They found a_{Na}^i rises rapidly at low pHi (intracellular pH) but that "the rise of a_{Na}^i is markedly attenuated or absent if pHo (extracellular pH) is also reduced " (p. 177). Thus they conclude that

> mammalian ventricular muscle acid-base disturbances produce changes of contraction that depend on changes in pHi. Providing simultaneous changes in a_{Na}^{i} are minimized, twitch tension declines roughly exponentially with a decrease on pHi,

such that tension is proportionate to intracellular hydrogen ion activity ($[a_H^i]^{-x}$) (p. 177).

Bountra and Vaughan-Jones (1989) also saw twitch tension as a function of a_{Na}^{i}, with t proportionate to $(a_{Na}^{i})m$, where m is a constant of approximately 3 or 4. Combining the two effects produces an equation where tension can be predicted following changes in aNai and aHi.

$$t_2 = t_1 \frac{[(a^i_{Na})2]^m}{[(a^i_{Na})1]} \frac{[(a^i_H)2]^{-x}}{[(a^i_H)1]} \qquad (11)$$

where 1 and 2 are the initial and final values of tension. This area of examination may not be valid when generalized to in situ human muscle during various levels of exercise.

II.1.2.6 Neural Recruitment

Fatigue test in which glycogen depletion patterns have been measured can indirectly implicate neural recruitment patterns in vivo. The pattern of fibre types that are depleted represent the fibres that are recruited under the specific exercise

conditions.

It was found that in isometric contractions of the medial quadriceps femoris slow twitch (ST) fibres are recruited up to tensions of 20% maximal voluntary contractions (MVC) as evidenced by selective glycogen depletion in these same fibres. At intensities above 20% MVC fast twitch (FT) fibres were recruited, again as shown by selective glycogen depletion of FT (Gollnick, Karlsson, Piehl & Saltin; 1973). The reason offered for this pattern is restricted blood flow reducing oxygen availability at forces greater than 20% MVC. It should be noted that muscle and [HLa] significantly increased at isometric contractions of 25% MVC or greater. This is consistent with a ST to FT recruitment pattern. However isometric contractions differ from dynamic exercise.

Armstrong, Saubert, Sembrowich, Shepherd and Gollnick (1974) examined rat skeletal muscle glycogen depletion patterns after various exercise dynamic intensities and durations to exhaustion. With initially low running speeds and during prolonged swimming it was found that oxidative fibres, namely fast oxidative glycolytic (FOG) and slow oxidative (SO) support most of the work. When either oxidative fibres are depleted or the intensity of work increases fast glycolytic (FG) fibres make a major contribution to work. [HLa] was found to be "directly proportional to the percentage of FG fibres showing a decline in glycogen" (Armstrong et al., 1974, p. 254).

In examining the same issue in human subjects Gollnick, Armstrong, Saubert, Sembrowch, Shepherd and Saltin (1973) concurred with a preferential pattern of fibre

recruitment for prolonged and or low intensity exercise commencing with ST fibres and then followed by FT fibres. Unfortunately mixed results for FOG fibres depletion rates and a low n could not posit the FOG fibre position in the pattern of recruitment. The general pattern discovered in rat skeletal muscle was confirmed for human skeletal muscle -- being ST first, then FT after ST depletion or significant intensity increments.

In contrast to prolonged work at approximately 67% $\dot{V}O_2$max high intensity exercise at approximately 150% $\dot{V}O_2$max completed in one minute sprints followed by 10 minute rests showed an early recruitment of FT fibres (Gollnick, Armstrong, Sembrowich, Shepherd & Saltin; 1973). The FT fibre recruitment was attributed to either the speed or intensity of the work. So in intense exercise FT are recruited almost exclusively.

To qualify which factor of speed or intensity of exercise was responsible for the differential fibre type recruitment Gollnick, Piehl and Saltin (1974) varied pedalling speed and metabolic intensity of exercise in a whole series of fibre depletion experiments. Changes in speed had an effect on foot force production on the pedal. Pedalling rates varied from 30-120 rev/min while metabolic intensity is cited as ranging from 30-150% of $\dot{V}O_2$max equivalent power levels. They found that at no time did varying the speed of movement by increasing the pedal rate have any effect on the glycogen depletion patterns and hence indirectly on fibre recruitment patterns. Yet the recruitment of ST fibres primarily during submaximal endurance exercise

could be altered by increasing the metabolic intensity above 100% $\dot{V}O_2max$ or extending exercise duration past the point of ST glycogen depletion to recruit FT fibres to continue. This experiment only produced forces of 35% MVC in the slower cadences. Therefore the question of whether or not higher percentages of MVC would effect fibre recruitment patterns in dynamic cycling exercise has not been answered.

Edgerton et al. (1982) predicates time to exhaustion on the percentage recruitment, which in turn depends on the type of motor neurons that are recruited. Increases in EMG signals for animals running on a treadmill that have increased in speed have been attributed to a "greater percentage of motor neurons being recruited or an increase in firing frequency of the motor units already recruited" (p. 42).

II.1.3 Summary

In summary the metabolic processes whereby ATP are produced are complex and have many stimulatory and inhibitory factors. La production and associated metabolic acidosis is a major consequence of pathway flux as it affects pH which in turn affects both the contractile strength and the endurance capability of repetitions. The absolute cause of fatigue at muscle sites is still controversial and varied as many concurrent occurrences can be mistaken for causation factors. Neural recruitment patterns have implications on La production and accumulation as well as metabolic control factors and fatigue.

II.1.4 Tests of Aerobic Power and Capacity

There are many tests and just as many test variations for assessment of aerobic power and capacity. The focus of this section will be on evolution of tests from $\dot{V}O_2$max tests, to lactate threshold tests, to square-wave endurance tests of repeated bursts of exercise. After examining the anaerobic threshold briefly the various lactate thresholds will be delineated. A criticism of the threshold concept which refutes any notion of an inflection point in [HLa] curves will preface the concept of fixed [HLa] tests. HLa indicators of exercise intensities along a continuum from easy to hard aerobic work will be delineated. This section will be followed by a section on reclassifying [HLa] and will include all the concepts that relate to La recovery.

II.1.4.1 $\dot{V}O_2$max Tests

The term maximal aerobic power (MAP) is misleading when equated to a power output completed on a progressive incremental test. The definition of MAP found in The Physiological Testing of the Elite Athlete, chapter four "Testing Aerobic Power" (Thoden, MacDougall & Wilson, 1982) is impossible to isolate as theoretically delineated, but the practical protocol suggested is quite clear. The theory states that:

> Maximal aerobic power may therefore be considered as being the maximal rate at which energy can be released from the oxidative processes exclusively (Thoden et al., p. 39).

The exclusiveness of the power output of the mitochondrial ATP production

due to oxidative fluxes as previously outlined in Figures I2 and I3 (pgs. 16,18) are impossible to measure if we use external muscle power as a parameter. In the theoretical definition in the manual cited above, muscular contractions result from not only high energy phosphate hydrolysis that comes from stored phosphate reserves in the form of ATP and CP, but also through the substrate phosphorylation of glucose or glycogen in the glycolytic pathway. Thus muscular power output is a cumulative effect of ATP breakdown providing energy to the contracting muscles in various proportions via the three energy systems.

Nevertheless the quantitative representation of the maximal amount of oxygen that can be consumed for a period of time in a progressive incremental test and the power level that corresponds to that consumption is close to, and an overestimation of MAP which is noted as $\dot{V}O_2max$.

There are many variations of the protocols and many practical details involved in maximal oxygen tests (McConnell, 1988; McConnell & Clark, 1988; Shephard 1984). The basic assumptions of these tests are that a large muscle mass is recruited for more than 3 minutes. The suggested time period of the test is 8 to 15 minutes with progressively incremental stages of 3 minutes duration. Speed is usually set at a comfortable pace where the heart rate response is between 140 and 160 beats per minute (bpm). Then the grade is increased by 2%, that is 2 cm rise for every 100 cm run in the treadmill belt length (Thoden et al.,, 1983).

The criterion for subject completion of an MAP test are two fold according to

Åstrand (1984), (1) an oxygen plateau and (2) a [HLa] that exceeds 8 to 9 mM. Shephard (1984) defines an oxygen plateau as a rise no less than 0.15 L/min or 2 ml/kg·min with further increase in power output.

A criticism of progressive incremental protocols is apparent from the work of the kinetics of oxygen uptake by Roston, Whipp, Davis, Cunningham, Effros and Wasserman (1987). The terminal $\dot{V}O_2$ for 15 minute submaximal rides exceeded the $\dot{V}O_2$max determined in previous incremental tests. This was attributed to a slower phase of oxygen uptake that takes longer to rise than the standard 3 minute stage of a max test. The O_2 uptake was significantly correlated to the continually rising [HLa]s in these experiments. Thus O_2 values from 15 minute suprathreshold (anaerobic threshold -- approximately 50% $\dot{V}O_2$max) but submaximal work rate tests are probably more representative of the true $\dot{V}O_2$max than incremental protocols.

Foster, Costill, Daniels and Fink (1978) examined the relationship between muscle enzyme activity, fibre composition, $\dot{V}O_2$max and performance. Their purpose was to extend the relationship found between $\dot{V}O_2$max and performance to include a parameter descriptive of the physiological characteristics of the elite runner's working musculature. Unfortunately the relationship between muscle succinate dehydrogenase activity and performance showed little relationship whereas the muscle fibre composition was only moderately correlated at $p < 0.05$ and $p < 0.01$.

II.1.4.2 Running Performance and Laboratory Tests

The physiological responses to middle and long distance running seems to be better described and predicted by submaximal intensities. Many researchers have shown higher correlations between performance times or mean performance velocities and variables associated with the various physiological thresholds posited in laboratory testing of distance running. For an exhaustive list see Londeree (1986) and Sjodin and Svedenhag (1985).

Various tests have been suggested to evaluate runners potential. They include $\dot{V}O_2max$ testing for relative oxygen consumption values in ml/kg·min; body composition testing for percentage body fat and ideal weight; 10 minute steady state runs to determine efficiency or oxygen cost in ml/kg·min at common speeds of 177, 196, 215, 241, 268, 298 m/min.; [HLa] testing for aerobic-anaerobic thresholds to determine the corresponding oxygen uptake, heart rate and velocity. The common HLa parameters for these tests are the onset of plasma lactate accumulation (OPLA) represented by the first rise in plasma La; maximal steady state which is found at approximately the 2 mM point; and the onset of blood lactate accumulation (OBLA) found at approximately the 4 mM point (Londeree, 1986). A more detailed list will follow in the next section.

Londeree (1986) has reviewed the relationship between performance and $\dot{V}O_2max$. In homogeneous groups of elite runners with high $\dot{V}O_2max$ values the correlation of running performance to $\dot{V}O_2max$ was very low. He cites plateaus in

$\dot{V}O_2$max values after several months of training with performance improvements continuing as the cause of low correlations. This has been proven in other studies.

Daniels, Yarbrough and Foster (1978) studied $\dot{V}O_2$max and performance changes for untrained healthy and trained groups over 4 and 8 weeks. They found a 9% $\dot{V}O_2$max improvement at the onset of training (after 4 weeks) along with a 9% performance improvement in the untrained group. But the continued 3% running performance (after 8 weeks) improvement in the 805 and 3218 m running events did not coincide with any continued improvements in $\dot{V}O_2$max. In contrast the well trained group improved their performance by 4% without any $\dot{V}O_2$max changes.

Running economy or the oxygen consumption at a given speed was examined but showed no significant difference for any group. Such that other physiological factors were indicated as responsible for improvements in running performance. Daniels et al. (1978) concluded that changes in $\dot{V}O_2$max are not good indicators of training adaptation after the initial start up of running training.

Several markers of HLa responses to incremental exercise were cited to be high correlates to running performance, namely, the maximal steady state (MSS); aerobic threshold (AerT); anaerobic threshold (AT); onset of plasma lactate accumulation (OPLA) and onset of blood lactate accumulation (OBLA). Training apparently affects these parameters " after $\dot{V}O_2$max plateaus" (Londeree, 1986, p. 203).

A combination of key limiting factors affect running physiology. Londeree (1986) estimates running potential in terms of $\dot{V}O_2$max, the anaerobic contribution to

energy production for shorter races, and the runner's amount of, use of, or sparing of, glycogen stores for longer races.

In an overview of the literature that cites high correlations between aerobic-anaerobic thresholds Londeree (1986) attempts to average out speed differences amongst the various markers of physiology. He lists the methods of determination as:

> the last workload prior to the first break point in blood lactate, the first workload in which blood lactate increased, the workload corresponding to a blood lactate level of 2.0 mM, the workload corresponding to the second break point in blood lactate level of 4.0 mM and changes in one or more gas exchange variables (Londeree, 1986, p. 209).

The first HLa inflection point on average occurred at 1.70 to 1.75 mM and usually was called the aerobic threshold (AerT). The [HLa] of 2.0 mM elicited a $\dot{V}O_2$ of 2 to 10% higher and on average a running speed of 25 m/min faster, within a range of 19 to 31 m/min faster. The 4.0 mM or OBLA point occurred at speeds on average of 30 m/min faster, within a range of 20 to 50 m/min faster.

Kumagai, Nishizumi and Tanaka (1987) also cite $\dot{V}O_2$max tests as low correlates to performance times. Added to this list of low correlates to performance was running economy which is "defined as oxygen uptake per body weight at a standardized submaximal treadmill speed" (Kumagai et al. 1987, p. 129). The authors correlations between 5 km, 10 Km and 10 mile race performances and AT-$\dot{V}O_2$ are listed in Table I1 (p. 47). $\dot{V}O_2$max values did not correlate as highly with performance times as did AT-$\dot{V}O_2$ values.

In assessing marathon running performance Sjödin and Svedenhag (1985)

found three factors could almost entirely explain the variation in race times. They were $\dot{V}O_2max$, $\dot{V}O_2$ at 15 kmh, and $\%\dot{V}O_{2ma}$ (marathon pace $\dot{V}O_2$ over $\dot{V}O_2max$ values X 100). Within the context of these laboratory tests predicting mean marathon performance the authors have found that with increasingly homogeneous groups correlations to marathon performance decreases, see Table I2 (p. 48). This was shown as well for $\dot{V}O_2max$ and running economy at 15 kmh as well. Shorter distances like 5 and 10 Km performances have shown high significant correlations (r=0.79 to 0.83) for running economy for runners within a narrow range of $\dot{V}O_2max$ assessments but the marathon data of Sjödin and Svedenhag (1985) contrasts these findings by showing a wide variation for marathon runners with "similar performance capacities" (p. 87). The fractional utilization of $\dot{V}O_2max$ at 15 kmh shows the same pattern of decreasing correlations for more and more homogeneous groups, see Table I2 (p. 48). This variable is thought to combine the effects of $\dot{V}O_2max$ and running economy. These components are thought to relate to performance differently.

The percentage (%) $\dot{V}O_2max$ at the marathon race pace ($\%\dot{V}O_2max$ X V_{ma}) determined on the treadmill ranged from 60% in slow runners (SR) to 87% in elite runners (ER), with the top two groups, elite runner plus good runners (ER + GR) at 80%. The three hour duration for SR may explain the non-significance of their results for this measure.

Table I1.

Correlations between 5 km, 10 km, and 10 mile races for $\dot{V}O_2$max and AT-$\dot{V}O_2$ where AT is defined as the systematic increase above resting levels of [HLa] values, at $p < 0.01$ modified from Kumagia et al. (1982).

Intensity	5 km	10 Km	10 mile
$\dot{V}O_2$max (ml/kg/min)	-0.645	-0.674	-0.574
AT-$\dot{V}O_2$	-0.945*	-0.839*	-0.835*

*significance, $p < 0.01$.

Table I2.

Correlations for three groups of runners: being elite runners (ER) $\dot{V}O_2max$ 71.8 ± 1.2 (62.9 - 77.9), good runners (GR) $\dot{V}O_2max$ 65.6 ± 1.2 (55.8 - 72.8), and slow runners (SR) $\dot{V}O_2max$ 58.7 ± 1.9 (53.4 - 67.4) and pooled groups (ER + GR) and (ER + GR + SR), with physiological data correlated to mean marathon performance times Vma from numerous scatter grams in Sjödin and Svedenhag (1985).

GROUP	$\dot{V}O_2max$	$\dot{V}O_{2\text{-}15km/h}$	$\%\dot{V}O_{2\text{-}15km/h}$ X $\dot{V}O_2max$	$\%\dot{V}O_{2max}$ X $\dot{V}O_2max$
ER	0.01	-0.49	-0.66	0.55
(ER + GR)	0.67*	-0.51*	-0.92*	0.09
(ER+GR+SR)	0.78*	-0.55*	0.94*	0.70*

GROUP	V_{HLa4}	Training distance per week (km)
ER	0.63**	0.57**
(ER + GR)	0.91*	0.67*
(ER + GR + SR)	0.96*	0.85*

significance, * $p < 0.001$, ** $p < 0.05$

Sjödin and Svedenhag (1985) reviewed the range of high correlations between submaximal exercise metabolic parameters and endurance exercise capacities. In five studies reviewed with a combined n of 139, correlations ranged from r=0.94 to 0.98. The authors concluded "there seems to be a rather close relationship between the 'thresholds' and performance" (p. 91). The thresholds are the above mentioned AT ventilatory parameters and the plasma and HLa values of 2 and 4 mM. Sjödin and Svedenhag (1985) found r=0.96 for V_{HLa4} and mean marathon velocity V_{ma} for n=35. In this case there was significance in all subgroups for OBLA velocities. An ANOVA for the three groups of runners found that the best single predictor of performance in long distance running is the 'threshold' value. This value is seen as dependent on all the previously listed factors of $\dot{V}O_2max$, running economy, as well as the best fractional utilization of $\dot{V}O_2max$ (p. 92). Finally the training mileage for the two months pre marathon was the highest correlated training parameter variable to marathon performance.

In summary performance in distance running has a very strong predictive power in treadmill velocity at a HLa value of 4 mM. This corresponding velocity will be significant and highly correlated to mean marathon velocity for all homogeneous subgroups as well as large heterogeneous groups. The best grouping of variables to predict marathon performance was $\dot{V}O_2max$, running economy at 15 km/h and percentage of $\dot{V}O_2max$ at marathon pace $\dot{V}O_2$ -- explaining 98% of marathon performance. The single most predictive variable was the anaerobic threshold

explaining 93% of marathon performance.

II.1.4.3 Lactate Thresholds

The indirect measurement of lactate threshold occurs through noninvasive methods of assessing metabolic acidosis by monitoring ventilation and analyzing the gases contained in expired air. Direct methods include blood and muscle sampling for La and other substances.

II.1.4.3.1 Indirect Methods: AT.

The concept of the anaerobic threshold (AT) is still controversial (Anderson & Rhodes, 1989). The most common and current noninvasive marker of the AT is an increase in the ventilatory equivalent of O_2, $\dot{V}E/\dot{V}O_2$ without a concomitant increase in the ventilatory equivalent for CO_2, $\dot{V}E/\dot{V}CO_2$. The triphasic pattern of $\dot{V}E/\dot{V}CO_2$ when plotted against time has been described as a decreasing function, a flat region, and a slight increasing function. This pattern has been demonstrated with a fair degree of certainty (Whithers, Sherman, Mill & Costill, 1981; Caizzo et al., 1982; Jones & Ehrsam, 1983).

The causation of AT by either neurogenic or humeral affects have not been totally isolated (Jones & Ehrsam, 1982; Farrell & Ivy 1982). A criticism of the oxygen insufficiency theory of AT for normoxia and free flow conditions is that the logic to support the theory can be considered circular or inverted for assumptions and

conclusions. For example both Davis (1985) and Katz and Sahlin (1990, 1988, 1987) represent the supposed effects of O_2 insufficiency on metabolism as explanations of anaerobisism by definition. This does not help clarify the cause of increased La production. If indeed neurogenic factors like a shift in motor units recruitment to FT fibres at higher power outputs is the primary cause of the increased La production and exercise hypernia (Jones & Ehrsam, 1982), then O_2 insufficiency theorists have twice erred on associating concurrent appearance with causation. Once for associating La production with O_2 insufficiency instead of glycolytic overproduction of pyruvate outpacing oxidative processes of removal and twice for linking the VT and the LT as cause and effect.

Green, Hughson, Orr and Ranney (1983) evaluated systematically the relationship between the AT as detected by respiratory and HLa criteria as well as selected glycolytic intermediates. They found that muscle and HLa accumulations differed in progressive exercise. Muscle Las were found to increase before HLas. AT determined by gas exchange did not coincide with AT determined by the alteration in [HLa]; and the increase in muscle glycolysis occurred before increases in [La] in muscle and blood occurred.

The cause of both increased La production and eventually increased $\dot{V}E/\dot{V}O_2$ has not been irrefutably isolated. Theories contrasted are: (1) consequence of neural recruitment progression from FT motor units with increasing power outputs (Brooks 1985) or fatiguing ST motor units (Gollnick et al., 1973) and (2) oxygen insufficiency

(Davis 1985; Katz & Sahlin, 1987, 1988, 1990). The recent work of Stainsby et al. (1989) has shown no lack of O_2 at cytochrome oxidase by using a direct intramitochondrial fibre optic assessment technique. This may put the O_2 insufficiency theory to rest if Stainby's methods are reproducible and validated by other laboratories. Farrell and Ivy (1987) have shown that blood pH is not responsible for increased $\dot{V}E/\dot{V}O_2$. This last study contains speculation that a nonhumeral "feed forward mechanism with neural signals originating from regions of the brain and/or the active musculature" (Farrell & Ivy, 1987, p. 1555) are responsible for respiratory control during exercise. Simon, Gutin, Blood and Case (1983) have also shown La accumulation uncoupled temporally from exercise hyperventilation in cycling, thus separating VT from LT.

II.1.4.3.2 Direct Methods: LT.

A survey of Lactate threshold concepts will be covered. Some differences exist between various researchers as to which LT correlates best to distance performances of 5, 10 k, 10 mile and marathon performances distances. All authors agree that LT and related variables correlates better to performance than $\dot{V}O_2max$. Some authors created an exercise continuum based on the various threshold markers being sequentially displayed in subjects in progressive incremental exercise.

To cite actual values for these phases of exercise the work of Kindermann et al. (1979) is reported here for 7 cross country skiers running on a treadmill, as representatives of well-trained aerobic endurance athletes. They found the aerobic threshold at an oxygen consumption of 47 ml/min/kg body weight with a [HLa] of 2 mM and a speed of 13 km/h at a 5% grade. At this point the arterial [HLa] displayed a first significant rise. At 15.3 km/h and at a 5% grade the [HLa] value of 4 mM was reached with a corresponding oxygen consumption of 54.9 ml/min/kg body weight representing 84% of MAP (64.6).

Sjödin and Jacobs (1981) found similar results for OBLA values for 18 marathon runners tested on a treadmill. In contrast Tanaka, Matsuura, Kumagai, Matsuzaka, Hirakoba and Asano (1983) found AT at 69.0% $\dot{V}O_2$max using cycle ergometry in 11 nonendurance trained active males adults. OBLA was found at 84.8% of $\dot{V}O_2$max. In contrast to other authors AT variables correlated better to performance than OBLA variables, which was not surprising since the subjects used in that study were relatively low fit and were tested on a cycle ergometer. The mean AT-LT value for [HLa] was 1.86 mM with a range of 1.46 to 2.34 or 195 watts to 240 watts.

Table I3.

Lactate threshold determination numbered for various authors with a description of possible breakpoints or zone demarcations.

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1. Christiansen et al. (1914) and Owles et al. (1930) - LT - cited in Anderson and Rhodes (1989), LT as the critical intensity above which there is an increase in HLa.
2. Wells et al. (1957) - a 3 zone model - plotting HLa versus $\dot{V}O_2$ creates 3 distinct slopes the beginning and end points of which delineate the 3 zones. Each region is broken into two parts. The 3 slopes are identified as (1) an "almost flat part... within the normal limits of resting values", (2) as an increasing slope of lactic acid to about twice resting values, and (3) a sharply increased slope. Work that elicits these HLa responses were classified as light, heavy and severe. A further breakdown of light work into either (a) mild or (b) moderate phases were marked by (a) a HLa at normal levels (resting) or (b) within normal limits and HRs of (a) < 100 or (b) < 120. Heavy work was broken down into (i) optimal or (ii) strenuous, with HLas of (i) 1.5 Xs rest (normal) or (ii) 2 Xs rest; and HRs of (i) < 140 or (ii) < 160. Severe work was broken down into either (1) maximal or (2) exhausting, with HLas of (1) 5-6 Xs rest or (2) > 6 Xs rest; and HRs of (1) < 180 or (2) > 180.
3. Wasserman and McIroy (1964) and Wasserman et al. (1973) - AT - in progressive

incremental work the stage before a critical work intensity beyond which an elevation of La in the blood occurs and which represents the onset or threshold of anaerobic metabolism.

4. Londeree and Ames (1975) - MSS - 2.2 mM as a threshold of maximal steady state metabolism where inflection of La into blood equals elimination.

5. Kindermann et al. (1979) - AerT, AT - a three zone model with 2 break points. Redefined Wasserman's and McIlroy (1964, 1973) AT as AerT at a FBLC of 2 mM and redefined Mader et al.'s (1976) FBLC of 4 mM as true AT.

6. Farrell et al. (1979) - OPLA - exponential and linear increases in HLa after the OPLA point which is marked at the FBLC of 4 mM.

7. Skinner and McLelland (1980) - AerT, AT - a 3 zone model with breakpoints at a FBLC of 2 mM and at a FBLC of 4 mM. Aerobic-anaerobic transition zone between FBLCs of 2 and 4 mM.

8. Rusko et al. (1980) - AT - increase in HLa above normal resting levels in young female cross country skiers. AT by gas exchange methods verified by 4 mM FBLC.

9. Ivy et al. (1980) - LT - HLa plotted versus $\%\dot{V}O_2max$ with the LT just before the onset of blood La accumulation (not OBLA but less than 1 mM).

10. Sjodin and Jacobs (1981) - OBLA- point at which HLa begins to increase exponentially, citing Mader et al.'s (1976) FBLC of 4 mM.

11. Stegmann and Kindermann (1981) - IAT - La accumulation and diffusion model

where an X axis parallel, at the level of the last stages [HLa] value, on a plot of [HLa] vs. Watts, intersects the HLa recovery curve. This point is the origin of a tangent to the HLa accumulation curve. The point of the tangent is the IAT workload...where a steady state between efflux into the blood compartment and maximal elimination from the blood and muscle compartments occurs.

12. Stegmann and Kindermann (1982) - IAT, AT_c, MSS - thresholds for MSS found through a 50 minute endurance test at the AT_c (the OBLA point) and IAT points that were determined from incremental testing.

13. Caiozzo et al. (1982) - LT - systematic increase in HLa above baseline warm-up values at an average HLa of 1.85 mM for average fit subjects.

14. Simon et al. (1983) - AT - the highest $\dot{V}O_2$ beyond which arterial HLa begins to accumulate causing metabolic acidosis.

15. Kumagai et al. (1983) - AT - LT as AT determined by an increase in "lactic acid above resting levels" (p. 16) at a mean of 1.79 mM. HLa results were reclassified according to the AT with stages as integer multiples of 3 min stages either before or after AT. A second [HLa] breakpoint was recorded at a mean of 3.49 mM but was not labelled or defined (assumed it was visibly interpreted from graphs).

16. Simon et al. (1983) - AT_n, AT_i, RCT or TDMA, CLW - the work threshold where HLa plateaus or returns to baseline resting levels. Using a CLW test

workloads at just below and just above two gas exchange thresholds and one plasma La threshold, noted as AT_n, RCT or TDMA, and AT_i respectively, where used to verify each thresholds distinctiveness. AT_n (noninvasive) was marked by the average or midpoint of the workloads stages between which a nonlinear increase in $\dot{V}E$ occurred and a marked consistent increase in $\dot{V}E/\dot{V}O_2$ and expired O_2 fraction (F_{EO2}) occurs without a marked increase in $\dot{V}E/\dot{V}CO_2$ occurs. The lower and higher workloads of the average were the below and above power settings of AT_n. The RCT or TDMA, as well as the below and above power settings, were assessed as above the marked rise in the ventilatory equivalent for $\dot{V}CO_2$ ($\dot{V}E/\dot{V}CO_2$). The AT_i was determined by the marked increase in plasma La (plotted Δ[La]). The lower and higher power settings of AT_i were the stages just before a marked increase and the next stage after the increase in plasma La. The CLW test verified through direct plasma [La] testing which power setting elicited a "marked plasma lactate accumulation" (p. 15) using an ANOVA with an alpha of $p < 0.05$.

17. Green et al. (1983) - LT - 1.25 as the initial breakpoint from linearity (baseline value of 0.97 mM) or LT determined objectively via a multisegmental linear regression technique using "either a two- or three- line regression model [which] was fitted to the data based on the smallest pooled residual sum of squares...($p<0.05$)" (p. 1033). Some subjects only displayed "a gradual increase which begins almost at the onset of work" (p. 1036).

18. Yeh et al. (1983) - AT - a smooth rise in arterial La -- near exponential. A 1.5 minute delay for venous to arterial equilibrium of La levels during exertion but no delay in recovery.

19. Hagberg and Coyle (1983) - LT - as a 1 mM rise above baseline values of 1.4 mM (therefore LT at 2.4 mM)

20. Brooks (1985) - LT - as an exponential rise in HLa.

21. Davis (1985) - AT - HLa progressively rises through the exercise period.

22. Heck et al. (1985) - OBLA, MSS, LSM - OBLA at a FBLC of 4 mM confirmed as the average threshold value for intervals in progressive incremental testing. MSS defined as La levels that do not increase more than 1 mM during the last 20 minutes of a 25 min. constant load test. The LSM uses a tangent to the La curve at 51° for the workload at LT (citing Keul et al. 1979). Also used was a tangent at 45° to the La curve for the workload at LT (citing Simon et al. 1981). The LSM was reported to be an alternative to the OBLA value for high fit subjects who have right shifted their La response curves due to training and have greater difficulty holding a workload that elicits an OBLA level HLa than lower fit subjects. Heck criticised Stegmann et al.'s IAT model because workloads were too short (2') (in Stegmann, 1981) in incremental tests to allow for muscle to blood La to equilibrium. Also Heck et al. justifies the 4 mM OBLA point in that 50 min of

exercise in Stegmann's experiment had a mean test completion HLa value of 4 mM.

23. Buchanan and Weltman (1985) - LT, OBLA - LT as the last workload that did not result in an elevation of HLa levels above baseline values. FBLC of 2 and 4 mM are used for LT and OBLA.

24. Beaver et al. (1985) - LT - supposedly objective model that transforms response data by plotting the log of the La versus the log of the $\dot{V}O_2$s in two linear segments that are obtained through linear regression analysis. The midpoint of the two lines is determined "visually as the point where the steep portion of the curve begins" (p. 1937) and therefore is still subjective. The log of the La response values are fairly well represented as a straight line.

25. Allen et al. (1985) - LT - LT at a FBLC of 2.5 mM cited in Hagberg and Coyle (1983). (actually Hagberg and Coyle cite 2.4 mM).

26. Rusko et al. (1986) - AerT, A_nT - AerT at a mean value of 1.84 ± 0.15 mM determined visually by two investigators as [HLa] "increased above levels observed at rest or at low power outputs" (p. 182). AnT at a mean value of power of 3.26 ± 0.31 mM and represented the power output where "linearity ... markedly disappeared for the second time and blood lactate accumulation began to accelerate (around 4 mM)" (p. 182) authors brackets.

27. Hughson et al. (1987) - LT - HLa increases as a continuous function plus constant in progressive exercise with a mean square error 3.5 times less for

plot fitting of the La response curve than Beaver et al. (1985).

28. Kumagai et al. (1987) - AT - systematic increase above resting HLa base-line values.

29. Campbell et al. (1989) - LT - confirmation of La increases as a continuous function for 8, 15 and 50 Watt progressive incremental ramp tests.

30. Chwalbinska-Moneta et al. (1989) - LT, OBLA, IAT - LT as a sudden increase in HLa, at a FBLC of 4 mM or the IAT method.

31. McLellan and Gas (1989) - AT, AT+1/3, AT+2/3 - Hughson's et al. (1987) exponential plus constant model "ineffective for 5 subjects so AT defined easily" as the metabolic rate that represents the onset of a progressively increasing" [HLa]. A "log-log transformation of the lactate-power output relationship" (p. 192) was used to standardize AT determination because La values decreased for some subjects from the start of the test making Hughson's model ineffective. AT+1/3 and AT+2/3 are AT plus 1/3 $\Delta\dot{V}O_2$max-AT and AT plus 2/3 $\Delta\dot{V}O_2$max-AT which are used to express exercise intensities relative to the individual AT (in this case @ 51% $\dot{V}O_2$max it probably represents AerT of Skinner and McLelland (1980) or a first rise in HLa.

Whithers et al. (1981) has shown that cycle ergometry and treadmill ergometry results in different values for runners and cyclists, especially with runners

achieving significantly higher $\dot{V}O_2$'s from treadmill running. The Tanaka et al. (1983) study lacks specificity in its analysis of runners by testing them on cycle ergometers and then comparing the results to distance running. They also used untrained men so comparisons to trained or well-trained runners would be an over-generalization error. As well the performance test involved was only 1500 m.

Stegmann and Kindermann (1982) tested 19 rowers on bicycles in 2 minute incremental tests to find OBLA and IAT. Then they endurance tested these same rowers to see which LT indicator was the best MSS parameter. In the majority of rowers the [HLa] was lower as a result of the IAT workloads with 50 minute exertions because the IAT occurred at a lower work rate. A criticism of this protocol and hence the findings was that a two minute workload may not allow for muscle to blood equilibrium during the incremental portion of the test. Therefore the OBLA workload would be underestimated, whereas IAT is based on enough time for La transport into the blood volume during recovery. Again the comparison of a group of athletes, in this case rowers, by testing on a nonspecific ergometer may jeopardize the findings validity. The IAT mean $\dot{V}O_2$ was 64.8% of $\dot{V}O_2$max. The OBLA mean $\dot{V}O_2$ was 77.0% of $\dot{V}O_2$max.

In 1985 Beaver, Wasserman and Whipp improved the detection of the LT by transforming the arterial [La] and oxygen consumption data by plotting the log of each data point. Their purpose was to get straight intersecting lines as a result of linear regression on two segments of the data points. Unfortunately the centre point of the

two segments that are to be linearly regressed is determined visually as the steep portion of the curves -- a subjective analysis which Yeh, Gardener, Adams, Yanowitz and Crapo (1983) rejected. Yeh et al.'s (1983) criticism is that no matter how much better the points reportedly fit the plot there is subjective bias with substantial interobserver and intraobserver lack of reliability. This criticism was also applied by Yeh et al. (1983) to the plotting of ventilation points for determination of the AT. Yeh et al. (1983) reported that some AT plots based on ventilation could not be determined. There is no indication that this was not the same for the method of Beaver et al. (1985).

In 1987 Hughson, Weisiger and Swanson tried to better the log-log model of lactate threshold determination using an continuous exponential plus constant model.

$$La^{-} = a + b[\exp(c\dot{V}O_2)]$$

The mean square error was reportedly 3.5 times larger in the log-log model of Beaver et al. (1985). One of the strengths of this model was that it did not assume a baseline value for La of zero. This matches well with reported aerobic La production in dog muscle under fully aerobic conditions (Connett, Gayeski & Hoinig;, 1985). The 1 minute sampling frequency might also be responsible for a different pattern than previously seen with two, three or four minute stages.

Supporting this contention that lactate thresholds are artifacts of our measuring procedures Hughson et al. (1987) threw noise at their curves and arrived at plots that resembled the data of Beaver et al., (1985). Yet the continuous function plus constant

still fitted the points with a lesser mean square error. The Campbell et al. (1989) report also verified this relationship with different ramp slopes for progressive incremental cycle protocols using 8, 15 and 50 watt increments.

McLelland and Gas (1989) showed that AT as defined by the log-log model of Beaver et al. (1985) was not the limit of steady state metabolism. By expressing exercise intensities relative to both $\dot{V}O_2$max and AT as described in #23 in Table I3. For the two groups, one with low AT values and one with high AT values, the authors found marked differences in ventilation, HLa and acid-base balance. This suggests that common reference points for calculating exercise intensities could not be $\dot{V}O_2$max or AT values. They suggest "that another metabolic rate above AT and below $\dot{V}O_2$max exists for each individual that defines a maximal cardiorespiratory and metabolic steady state of exercise.

In summary it can be said that La accumulation has been proven to be a continuous function best described by an exponential plotting. As well both OBLA values for groups and IAT values for individuals represent a slight overestimate and a mean steady state La respectively, where La production and removal are equal after an initial rise at the onset of exercise. Endurance times at MSS can be 50 minutes with a < 1 mM increase in the last 20 minutes of exercise. The progression of the thresholds in increasing intensity were first, the initial rise in La above baseline or AT around 1.0 to 2.0 mM; aerobic threshold (AerT) or approximately 2.0 to 2.5 mM (also called mean state); a 1 mM increase above baseline; and alternately IAT and OBLA. These

last two markers are specific to the individuals and the ergometers used with differences amongst values shown for tests on ergometers that do not represent the sports experience of the athlete. i.e. cyclists tested running on a treadmill.

II.1.4.4 Fixed Blood Lactate Concentrations

An alternate to threshold determinations is fixed blood lactate concentrations (FBLC). Some FBLC are marked at 2.0, 2.5, 4.0 and 5.5 mM (Weltman, Snead, Stein, Seip, Schurrer, Rutt & Weltman, 1989; Stark, 1989). Factors that affect FBLC are free fatty acids in the blood; glycogen content in the muscle; acid-base status in the blood; the intake of a carbohydrate-rich diet; a hypoxic condition; and the luteal or follicular phase of the menstrual cycle (Yoshida, 1987).

At the University of Ottawa a three zone model uses (Stark, 1989) traditional measures of exercise physiology namely heart rate, oxygen consumption, and [HLa], and reclassifies these values relative to the OBLA point. Three demarcation points are then generated by an objective statistical analysis of the first statistically significant rise in [HLa] level and every other significant rise in La concentrations. Three zones of exertion are conceptualized as predominantly aerobic, at HLa levels of less than 2.5 mM; as a transition from predominantly aerobic to predominantly anaerobic, at HLa levels between 2.5 and 5.5 mM; and as predominantly anaerobic above HLa levels of 5.5 mM. The relationship between La and O_2 showed a moderate negative correlation below the first rise (2.5 mM); a moderate high correlation above the aerobic-anaerobic

transition zone (2.5 to 5.5 mM); and a correlation increasing from moderate negative values to moderate positive values in the transition zone. The high negative correlation that occurred in the aerobic zone associated high oxygen consumption with low La responses. A low order correlation occurred between oxygen consumption and [HLa] at the midpoint of the aerobic to anaerobic transition range, being the 4 mM point, and represented no relationship between O_2 and HLa at this point. A high positive correlation occurred in the anaerobic range with high oxygen consumption associated with high HLa levels (Stark, 1989).

II.1.4.5 SWEET Tests

A test of power and endurance capacity is represented by the "Square-Wave Endurance Exercise Test" (SWEET) (Gimenez, Cereceda & Teculescu, 1982; Gimenez, Servera, Saunier & Lacoste, 1982; Gimenez, Serera & Salinas, 1982). Its purpose is to mimic the training mode of intermittent exercise with one minute peaks followed by 4 minutes of active recovery just below the aerobic-anaerobic threshold of Kinderman et al. (1979) which also corresponded to less than 65% $\dot{V}O_2$max prescribed by Hermansen and Stensvold (1972). The key factors are the intensity and duration of interval exercises above the baseline value, the work to active rest ratio, and the intensity and duration of the active recovery baseline values. It was found that elite athletes were able to complete SWEETs at > 70% maximal tolerated power (1 min) (Gimenez et al., 1982).

A traditional measurement of the cardiovascular system during exercise testing is the heart rate response. In the square-wave testing heart rates were contrasted for differences between peaks and active recovery values. Of the three groups tested the untrained had the "more marked" (p. 363) differences with a range of 20 to 30 beats/min versus trained subjects who had a 10 to 20 beats differences versus the "least difference" (p. 363) for elite sportsmen and trained athletes who only had a range of 4 to 10 beats/min. At the end of the test HR was 98% of that observed during a $\dot{V}O_2$max test.

The change in heart rate over the entire 45 minute test did not correlate significantly with the change in [HLa]. The HLa levels were seen as significantly higher in elite subjects at the end of the sweets.

Mongnoni, Sirtori, Lorenzelli and Cerretelli (1990) found poor predictions between progressive incremental exercise tests fixed [HLa] like the OBLA point and steady state exercise tests La values. Thus prolonged tests appear to be better indicators of endurance time or total work time to exhaustion than any incremental test response. As well HLa responses during endurance tests appear to be better for profiling endurance test HLa responses than progressive incremental protocols.

II.1.5 Summary

In summary $\dot{V}O_2$max tests are sluggish in that they do not reflect continued changes in the trained state of elite endurance athletes. In contrast many physiological variables like the AT, the LT, OBLA, IAT, and FBLC have higher correlations to race

performance intensities. Indirect methods of LT determination has many criticisms because the La causation factor has been invalidated and temporally removed from its ventilatory markers. Since the best state a theory can have is to never have been disproved (Christensen, 1988) AT determination remains at best shaky. Direct LT measurement has been criticized when used to assess a discreet inflection point subjectively. A smooth rising exponential curve has now been shown to best reflect the [HLa] accumulation in progressive incremental exercise. IAT has proven to better reflect MSS than OBLA but OBLA better mirrors prolonged exertion with its creeping HLa values. FBLC have marked transition thresholds at the 2 and 4 mM [HLa] levels on average. A model of three FBLCs at 2.5, 4.0 and 5.5 mM for transition thresholds has shown a triphasic response when correlating HLa and O_2 consumption. The three zone model's objective method of determination holds promise. Finally SWEET protocols have been pursued to evaluate trained and untrained subjects with endurance tests involving repeat intervals for a better indicator of endurance times and endurance [HLa] responses than any progressive incremental test protocol.

II.1.6 Reclassifying HLa Recovery

Blood lactate concentrations ([HLa]) are a result of both La output and uptake by skeletal muscle and other tissues. To assess and reclassify the HLa recovery ability the aspects affecting La production should first be examined. Following this the metabolism of La will be mapped for the possible efflux of the 3 carbon fuel from

muscle to interstitial space, to vascular circulation, and finally back through these two barriers to an oxidative or gluconeogenic fate. The balance of La efflux and influx will be described by a model of La accumulation and La recovery.

II.1.6.1 Lactate Production

Stainsby (1986) is recognized as having listed all the key factors responsible for metabolic control of La production (Stainsby & Brooks, 1990). In examining the inherent characteristics of the muscle itself, devoid of blood flow and hormonal affects in situ muscle, Stainsby (1986) found that inadequate O_2 in the mitochondria cause the electron transport chain (ETC) and oxidative phosphorylation to be slow. Thus the redox state of the mitochondria ([NAD] / [NADH]) and the phosphorylation potential ($[ATP]/[ADP_f]x[Pi]$) of the mitochondria fall. Because these stimulators of glycolysis occur when NADH cannot be effectively removed from the cytosol, pyruvate proceeds to La resulting in NADH oxidation -- allowing glycolysis to continue.

Karlsson (1971) cites the two conditions where O_2 would be insufficient and cause La production. The first occurs at the onset of exercise before a steady state O_2 uptake is reached. The second occurs at supramaximal workloads. Stainsby (1986) gives 1 to 2 minutes as the time course at the onset of exercise for this process of ETC activation to become adequate to reoxidize sufficient NADH to NAD. Once fully activated the ETC facilitates glycolytic continuation without concomitant La

production. As a result of the ETC activity lagging glycolytic activity La production rises to maximum amounts in 2 to 7 minutes, then drops back to 0 in 10 to 20 minutes (Karlsson 1971). At supramaximal intensities the plateaus of maximal oxygen consumption has already occurred at a lower power level thus creating an oxygen insufficiency for any workload above $\dot{V}O_2$max levels.

Brooks and Fahey (1984) acknowledge two other factors related to increased La production. These factors are associated with increased neural drive to produce higher power outputs from muscle tissues in response to the demands of the situation, for example progressive incremental exercise protocols.

The first factor is a function of shifting neural recruitment patterns of homogeneous motor units to accomplish higher levels of muscle power output in exercise. As the intensity of exercise increases more slow oxidative (type I or slow twitch --'SO') fibers are dropped in favour of the fast oxidative glycolytic (type IIa one of two groups of fast twitch --'FOG') or eventually fast glycolytic (type IIb or the second grouping of fast twitch --'FG') fibers in a pattern described by the size principle (Enoka & Stuart, 1984).

The size principle states that "frequency of motor unit recruitment is directly related to the size and ease of triggering an action potential in the soma" (Brooks, 1984, p. 354). SO fibers have smaller somas, smaller axons diameters, lower activation threshold frequencies and slower neural conduction velocities. They are used first and more frequently in slow or low power movements of less than 20-25% MVC. FG

and FOG fibers have larger somas, larger axons diameters, higher activation threshold frequencies and quicker neural conduction velocities. They are used almost exclusively in fast reflexive or ballistic movements compared to combined involvement with ST fibers in high to moderate power movements of greater than 25% MVC.

The shift in neural recruitment as monitored by electromyographic (emg) devices occurs at the same time as [La] in muscles increase (Brook and Fahey, 1984). Helal, Guezennee and Goubel (1987) examined the aerobic-anaerobic transition and the threshold phenomena and found difficulty in correlating the variance in interindividual EMG data with mean values of HLa. However an indication of fiber type recruitment in incremental exercise was found and described as a "progressively enhanced number of fast MUs (motor units) activated rather than a sudden activation of them all" (Helal et al., 1987, p. 648).

The second factor cited by Brooks and Fahey (1984) affecting La production is a concomitant sympathetic autonomic nervous stimulation of the adrenal medulla and the α cells in the pancreas, releasing epinephrine and glucagon. Glucagon and epinephrine enhance glycolysis in muscle and liver (Brooks and Fahey, 1984). Both hormones result in an overproduction of pyruvate not matched by the ETC's ability to utilize pyruvate, with excesses converted to La or alanine. Whether the responses of the mitochondria are slowed (1) because of a lack of O_2, (2) because of a supramaximal workload requiring more O_2 than can be delivered

by the circulatory system, or (3) because of a more rapid activation of the glycolytic sequence, the result is increased muscle La production. Yano (1985) measured the time constant of oxygen kinetics, heart rate, stroke volume and cardiac output and found these values were: 40, 7, 23, and 14 seconds respectively. Thus the delay for O_2 is the greatest of these four parameters, explaining the initial rises in [La] in muscle and then blood. Morton and Gass (1987) found a time constant delay of 14 sec for untrained subjects versus trained subjects for the fast part of the $\dot{V}O_2$ increasing curve at the onset of exercise. Thus the La responses at the onset of exercise are exaggerated for the unfit subjects.

Karlsson (1971) tested trained (T) and untrained (U) groups using muscle biopsies of the vastus lateralis to ascertain muscle [La] with concomitant [HLa] sampling. It was found that there was a baseline resting [La] in muscle. Yet muscle [La] accumulation did not occur till work exceeded 50-60% $\dot{V}O_2$max. It was also found that muscle [La] did not increase till phosphogens were depleted by 4-5 mM/kg wet muscle. At 75% $\dot{V}O_2$max T and U had the same muscle [La] but T reached a higher maximum, 22.7 versus 16.9 mM/kg wet muscle in U. The most probable cause of the increased [La] during exercise, according to Karlsson (1971), was the increased NADH since muscle pyruvate concentrations had negligible increases.

The increase in [HLa] after repeat maximal bouts was found to lag behind muscle [La]. This was suggested as a translocation hindrance where free diffusion was not occurring as evidenced by the lag time. Nevertheless post exercise muscle La

values were found to be plateaued at the same values if time was allowed for equilibrium. Jacobs (1986) found that muscle and blood [La] were closest at the 4 mM mark. This suggests that the translocation mechanism is saturated to work at optimal intensity before further increases limit transport. This follows the general model of enzyme kinetics thus demonstrating a carrier mechanism.

Other findings of Karlsson (1971) were that maximum muscle [La] were found for 2-3 minute bouts of exercise as well as 7 minute bouts, "but were lower when exhaustion time amounted to about 10-20 minutes' (p. 40).

Paradoxically [La] increases result from delays in $\dot{V}O_2$ reaching steady state levels yet [La] increases eventually push O_2 levels to maximum. La can push the upper values of oxygen consumption past $\dot{V}O_2$max levels that are determined via progressive incremental protocols. Increases in HLa accumulation, which result from La production and influx into blood outpacing La removal and efflux from blood, has been linked to a slow upward drift of oxygen consumption above the LT as defined as the AT marker of Cazzeo et al. (1982). Roston, Whipp, Davis, Cunningham, Effros and Wasserman (1987) found that $\dot{V}O_2$ increased to a steady state within 3 minutes for LT or less intensities. Supra-LT intensities have shown $\dot{V}O_2$ climbing till the sixth minute of a cycle test. Further the maximal oxygen consumption elicited from this slow component "commonly exceeded" (Roston et al., 1987; p. 1083) the $\dot{V}O_2$max of the incremental tests.

Tesch, Daniels and Sharp (1982) tested muscle and HLa values and found that they did not co-vary after four minutes of cycling at OBLA work levels previously determined in a progressive incremental test. They found evidence of a translocation hindrance in that muscle [La] rose continuously after [HLa] plateaued.

Jacobs and Kaiser (1982) also measured muscle and blood [La]. They also assessed differences in FT or ST fibers [La] after cycling at OBLA work ($OBLA_w$) intensities. The primary finding was a strong positive relationship between muscle and [HLa] at the work intensity closest to $OBLA_w$ ($r=0.89$, $p < 0.001$). A muscle/[HLa] ratio of 2.3 was found. No significant difference was found between ST and FT fibers at the closest work level to OBLA.

Maasen and Busse (1989) investigated the relationship between workload (WL) and [HLa] under various diet conditions and in two training states being trained and untrained. The relationship between WL and a trained state was that the inevitable increase in [La] occurred at a higher WL or a higher % max WL. CHO deficiency or complete replenishment (supercompensation) has also shown a capacity to shift the [La] work load association curve. Although low CHO diets have shown a lower [HLa] response to a work load, suggesting an increased endurance performance time, endurance tests demonstrated longer performance times and higher maximal HLa values for athletes with a high CHO diet. The implication for submaximal [HLa] of 2 and 4 mM is that a low CHO diet will cause these values to occur at a higher power output, but this will not translate to the relative performance enhancement

demonstrated which was associated with a high CHO diet. Therefore endurance performance is best assessed through endurance performance test parameters not affected by low or high CHO diets or by endurance performance tests themselves.

Yoshida (1986) reviewed many of the findings of the effects of diet on HLa levels. He reported a high carbohydrate (CHO) diet causing higher [HLa] at a given $\dot{V}O_2$ when compared with a low or normal CHO diet. Nevertheless the difference between [HLa] and resting values during the three types of dietary modifications were not statistically different at $p < 0.01$. OBLA's 4.0 mM power level changed with diet modifications (at $p < 0.01$) as compared to the LT power level when LT was measured as the first rise in La above baseline values. This change was attributed to the elevated contribution of CHO metabolism during graded exercise. The transport mechanism for La, which is most commonly cited as a facilitated transport, may be CHO dependent and modifications in diet may affect it more than glycolytic flux. Although Yoshida has failed to account for all the basic variables involved in [HLa] determination, such as the balance between production and removal, he has provided a key control element for eliminating diet modified [HLa] responses. Differences between two points on a La response curve controls for diet variations on that curve. Also the LT, as measured by Yoshida breaks from a higher or lower baseline value due to diet modifications.

Yoshida (1986) showed a delta La value was not affected by low or high carbohydrate diets because delta La values are a difference between baseline values

and stage values both of which may have been shifted by diet -- but the difference between the two points on the curve will remain unaffected. Heck et al. (1985) has shown the same right shifting of the La response curve for running surface.

Glycogen depletion did not cause variances in the physiological determinants of running performance like $\dot{V}O_2max$, LT or VT and consequently no change in 10 K running time was affected. Yoshida concludes that if [HLa] is used for endurance performance determination the OBLA value will be affected by dietary modifications. The AT La inflection point is not affected in spite of altered [HLa] levels. This would mean that if delta La's were taken from either the 2.5, 4.0 or 5.5 mM mark to just below the individual AT the differences would not be altered but the absolute power output that these markers reflected would be different with different diet conditions. An active recovery posited below the AT inflection point would not be difficult to find inspite of any diet modifications.

II.1.6.2 Fixed Blood Lactate Concentrations

The HLa value associated with a 2 mM value and a 4 mM value was cited by Maasen and Busse (1989) in cyclists as depicted in Table I4 (p. 77).

Table I4 (p.77) shows that there was no significant difference between trained and untrained cyclists after two days off and a high carbohydrate diet for FBLC of 2 and 4 mM. There was also no significant difference in FBLCs of 2 and 4 mM and the IAT between cyclist before and after a 13 day training camp (Maassen & Busse, 1989). Yet all subjects had higher work load maximums (WL_{max}) and higher [La] at

the end of exercise. The IAT was higher in some, lower in some, unchanged in some, and undeterminable in two of six.

A final group was tested before and after 3 days LCD while three of the subjects were in their recovery season. In this case the WL_{rel} and WL_{abs} were higher after LCD diets but WL_{max} were higher along with on average 26 minute longer endurance tests. Thus the implication of enhanced endurance by lower relative and absolute [HLa]s is not substantiated with endurance tests at 65% of maximum performance until exhaustion.

The implication of these studies were that the only way to measure endurance capacity is to use an endurance test and parameters that account for possible diet al.,tered values.

Gollnick, Bayley and Hodgson (1986) cite various factors like diet, state of physical fitness, type and duration of exercise as factor affecting La accumulation due to production. The normal resting values are cited as 1 mM/kg wet muscle or litre of blood. Describing normal La accumulation patterns that generally start to increase around 40% and then later become exponential, the authors cite the ability to use an emergency energy supply to complement aerobic energy production as the reason for a La baseline value. However patterns of increase that return to normal resting values after an initial rise are cited as more difficult to interpret, however they have been explained above as aspects of a delay in aerobic utilization of pyruvate.

The normal resting values are cited as 1 mM/kg wet muscle or litre of blood.

Table I4.

Selected values for 2 and 4 mM fixed HLa concentrations, OBLA and IAT where % WLmax is percentage of work load maximal, UT is untrained, T is trained, CY N are cyclist before a training camp (13 days), and CY TC are cyclist after a training camp tested both groups tested in a progressive incremental test, modified from Maassen and Busse (1989).

Group (n)	WLmax (W.kg-1)	2 mM (%WLmax)	4 mM (%WLmax)(n)	IAT (%WLmax)(n)
UT(9)	4.0±0.6*	58±6	75±4	72±6(7)**
T(8)	5.5±0.5*	63±4	78±2(7)	81±4(6)**
CY N	5.3±0.4	65±5	80±7(7)	81±4
CY TC	5.4±0.3	67±4	82±3	81±4

*significance, p<0.001, ** p<0.002

3 Day Low Carbohydrate Diet

Before			After		
Work	WL_{rel}%	[La]	Work	WL_{rel}%	[La]
292±48 W	75	2	244±31 W	59	2
340±41 W	87	4	319±24 W	77	4

Describing normal La accumulation patterns that generally start to increase around 40% and then later become exponential, the authors cite the ability to use an emergency energy supply to complement aerobic energy production as the reason for a La baseline value. Patterns of increases that return to normal resting values after an initial rise are cited as more difficult to interpret, however the authors have been explaining the above as aspects of a delay in aerobic utilization of pyruvate Gollnick et al. (1986) review three factors of La production, namely; intensity of exercise, duration of exercise, as well as training adaptations. These factors have not already been dealt with yet as factors of La production.

Intensity seems to elicit a [La] increase at 50 -60% MAP for untrained and at higher levels for trained. The maximal rate of La production is reported in Gollnick et al., (1986) as 0.5 mM/gram wet weight for muscle composed primarily of type II fibers, and about half that value for type I fibers.

Durations of less than 8 minutes for time to exhaustion is reported by Gollnick et al., (1986) to result in similar muscle and HLa levels. Yet durations of one to two hours see initial rises in [HLa] that return to resting levels.

Endurance training produced intraindividual differences where untrained subjects had a higher [HLa] at the same submaximal work load as trained subjects. Also interindividual studies with two leg exercise shows endurance trained limbs show a marked net uptake of La while untrained limbs show a net release of La.

II.1.6.3 Lactate Recovery

La accumulation inside the muscle cell provides the necessary gradient for La to efflux down a concentration gradient and be removed. The first areas of removal are SO and FOG fibers parallel to FT fibers where La equilibrates across the fiber types (Brooks 1985).

Gladden (1989) reviews the flux of La across the cell membrane to the interstitial space that can be accomplished by ionic (anion) diffusion or transport, antiport bicarbonate linked anion exchange, symport proton linked cotransport, free diffusion of undissociated lactic acid and or any combination of the above. The H^+-symport lactate carrier and the OH^--antiport lactate carrier are indistinguishable because no discrimination can be made between cotransport and countertransport. Ionic transport is thought to occur only for release due to a favourable electrochemical gradient for the La ion. Whereas the La anion diffusion or uptake would require active transport to counter the electrochemical gradient. When external pH is high proton linked and undissociated acid are the predominant flux methods, whereas a "slow pH independent transmembrane leak of La ions follows the electrochemical gradient" (Mainwood, Renaud & Mason, 1986, p. 653).

The free diffusion of LA transport is carrier independent since membrane lipid bilayers are "permeable to undissociated lactic acid" (LA) (p. 653). When La enters the muscle cells proton-linked fluxes predominate "because of the membrane potential contribution to the electrochemical gradient" (p. 653). When net La efflux occurs the

favoured form is free-ion flux because the electrical charge. When proton-linked fluxes occur the buffer concentration juxtaposed to the sites of entry must facilitate diffusion of proton away in the intracellular compartment. Failure results in diffusion limited transport across the cell membrane.

Therefore lowering pH can slow muscle membrane La efflux by inhibiting the H^+ dependent carrier but not the undissociated lipid diffusion rates. This confirms Karlsson's (1971) findings of post exercise equilibrium between muscle and blood given enough diffusion time for equilibrium. The converse influx rate of La uptake also shows H^+ dependent carriers slowing as reported by Karlsson (1971). The importance of blood flow on La efflux is seen in the crucial proton diffusion rate at the site of La exit from membrane tissue and hence there is proton diffusion limited transport.

Proton linked and ionic transmembrane fluxes use a protein carrier and thus show characteristics of mediated diffusion such as saturation (Spence & Mason, 1982).

La release from muscle was examined by Jorfeldt, Juhlin-Dannfelt and Karlsson (1978) via femoral vein and artery differences (v-a) in four physical education students. A linear increase in muscle La versus v-a for La was observed until 4 mM/kg wet weight after which v-a plateauing, resulted in La release ceiling of 4-5 mM/min. The saturation of the translocation mechanism was cited as the cause of the levelling off. Muscle La was then seen to continue increasing.

Hermansen and Vaage (1977) found conflicting La release values after a 3

minute exhaustive ride. In contrast to other studies the recovery activity was passive and hence glycogen syntheses in muscle was the only logical alternative for La disappearance since efflux was recorded as very low. The generalizability of Hermansen and Vaage's 1977 results of only 10% La efflux from muscle is suspect for active recovery running on a treadmill or in the field.

Hermansen and Stensvold (1972) studied HLa recovery of well-trained subjects at rest, 30, 60, 70, and 80% $\dot{V}O_2$max after three one minute bouts of maximal running on a treadmill. They found a maximal rate of HLa removal of 8 mg/100 ml x min-1 at a mean of 63% $\dot{V}O_2$max active recovery intensity. This corresponds to 2 mM per min as a net La recovery rate. The failure of any significant increase in [HLa] up until 62.9% $\dot{V}O_2$max in progressive incremental exercise coupled with an optimal La recovery rate of 63% are the main findings of this study. Although the authors reported these two findings concurrently and did not formally link these findings their juxtaposition seems justified. Liver La removal is discounted as the major site of removal (85% cited previously by Cori) in favour of skeletal muscle as a significant La remover. This 1972 study is the most relevant to the target population of elite runners in this proposed study both because of the fitness levels of Hermansen and Stensvold's subjects, who displayed delayed La accumulation, and because of the specificity of the ergometer used, a treadmill. Hence Hermansen and Stensvold displayed sport specificity in their study in contrast to the dearth of other studies that used cycle ergometry to evaluate runners skiers and rowers.

Belcastro and Bonen's 1975 study is an example of the above point in that they studied both controlled recovery at 30, 45, 60, and 80% $\dot{V}O_2$max and uncontrolled recovery (subject variable load) recovery following 6 minutes of 90% $\dot{V}O_2$max -- while cycling. The relatively low optimal La recovery rate of 17-49% $\dot{V}O_2$max for cycling was attributed to a smaller active muscle mass than treadmill running and the relatively lower fitness state of the subjects. Therefore the generalizability of these findings to elite runners is suspect.

Stamford, Moffat, Weltman, Maldonado and Curtis (1978) examined previously exercised leg muscle and muscle mass factors in cycle ergometry to ascertain La recovery ability. No fatigue affects of one previously exercised muscle versus a fresh limb was observed for La recovery. Percentage $\dot{V}O_2$max was not as important with one and two leg exercise during recovery as compared to whether or not baseline La values were elevated or not. Work rates where intensities are low and baseline blood La accumulation values are minimized are suggested as optimal La recovery intensities over possibly higher % $\dot{V}O_2$max intensities where baseline La values are elevated. Unfortunately this study cannot be evaluated with runners as one leg treadmill running is difficult at best.

Bonen, Campbell, Kirby and Belcastro (1979) examined La recovery and found a multiple regression model of blood La removal that explains 41, 22, and 19 percentage of the variation in La recovery; based on [La] at time zero, % ST fibers, and the recovery intensity respectively. In comparison Tesch and Wright (1983) found

multiple regression analysis revealed that "95% of the variance in recovery from fatigue could be explained by fiber type distribution, capillary density, fatigue and peak torque" (p. 100). This study involved isokinetic (Cybex II) induced fatigue of 50 MVCs at 180° per s., and peak torque at 30, 180, and 300°/sec during knee extension. Here recovery from fatigue was a performance indicator of 5 MVCs after a 40 sec rest from the 50 MVCs. The authors suggest capillary density reflects the metabolic profile of the muscle, and parallels the mitochondria content and regulatory enzymes. Muscle force was found to recover quicker than La elimination proceeded.

Stamford, Weltman, Moffat and Sady (1981) tested HLa recovery ability with baseline corrected [La] values for sub and supra AT intensities, at 40% and 70% $\dot{V}O_2$max respectively in cycling. They found that when baseline values of 0.9, 1.3 and 3.5 mM were used to evaluate single exponential decreasing functions of [HLa] not only did half time HLa recovery decrease at 40% and 70% AT recovery exercise intensities but also the data points were highly correlated to the plot lines, r=.984. When plotted semilogarithmically 40% and 70% AT recovery conditions produced parallel decreasing HLa lines. Thus no real difference existed in La removal rates after correcting for different baselines for the 40 minute recoveries. This seems to indicate a maximum of La recovery ability especially when the increase of a predetermined level of exercise is accounted for. No one has reported delta [La] values for La baselines- interval differences that diminish during square-wave endurance exercise tests (SWEET).

Åstrand, Hultman, Juhlin-Dannfelt and Reynolds (1986) also studied La recovery from combined arm and leg exercise. They found that during a 1 hour recovery hepatic uptake of La, as measured directly, increased up to 18 fold at the 4 minute peak post exercise [HLa] value. Based on critical assumptions of total body water space Åstrand et al. (1986) calculated that of the 830 mM of La produced only 80 mM were taken up by the liver (10%). Approximately 40% was oxidized by skeletal muscle and the remainder was left stored in body water. As well the dominant substrate for replacing muscle glycogen was La not glucose as "50% of the formed La is synthesized into glycogen in muscle" (p. 342). This complements the findings of Brooks (1990) who describes La as an oxidizable substrate instead of the previously defined fatigue agent.

Mazzeo, Brooks, Schoeller and Budinger (1986) studied La irreversible disposal rates and oxidation rates with a stable radioactive tracer [U-^{14}C]lactate. Inspite of criticism of tracer site injections not representing all La entry points into the blood system when only one vein is used as entry port for tracer (Katz, 1986) the percentage of La removed through oxidation was cited as significantly increased from rest to easy exercise but not significantly increased from easy to hard exercise. Oxidation at rest was seen as a major fate of HLa with 49.3% disposed of this way which is close to Åstrand et al. (1986) findings. This value may be overestimated since labelled carbons are exchangeable with other substrates like pyruvate (Katz, 1986) hence real values would approach the 40% reported by Åstrand et al. (1986).

Notwithstanding the overestimation the % oxidation values for La were 87.0% for 2 hours of easy exercise and 79% for hard exercise. This lends support to the claim of Brooks (1990) that La is a major source of fuel during exercise and recovery.

In support of the concept of [HLa] as a balance between production and removal (made by Brooks (1985) and many others, Mazzeo et al. (1986) found La was "continuously produced even at rest" (p. 237) with about half the La carbons being turned over and expelled as CO_2. This validates the view that a [HLa] value alone is "inadequate for the estimation of La production" (Mazzeo et al.1986). Turnover can be 2 to 4 times greater than rest for any given [La]. Finally La disposal and oxidation were significantly correlated to the metabolic rate.

Freund, Oyono-Enguelle, Heitz, Marbach, Ott and Gartner (1989) used a two term exponential function that described arterial [La] concentrations during recovery as described in equation (12) and a two compartment model (Freund and Gendry, 1978; Freund and Zouloumian 1981 cited in Freund, Zouloumian, Oyono Enguelle & Lampert; 1984) to assess the effects of exercise duration and intensity on La recovery ability.

$$La(t) = La(0) + A_1(1 - e^{-\gamma_1 t}) + A_2(1 - e^{-\gamma_2 t})$$

where y_1 and y_2 are the velocity constants of the exponential functions, and A_1 and A_2 are the amplitudes of the exponential functions, La(t) and La(0) are the arterial [HLa]s

at time t and at the onset of recovery respectively.

Freund and Zouloumian (1981) found that y_1 and y_2 were lower after "maximal than submaximal exercise performed on a bicycle" (in Freund et al. 1984). Therefore HLa recovery is dependent on previous intensity. In contrast Freund et al., (1989) examined the mean velocity constants y_1 and y_2 after three versus six minutes of cycling. The lower velocity constant at easy exercise and hard exercise was reconfirmed. When duration was increased decreases of 25 to 30% for y_1 and 15 to 19% for y_2 were reported for 6 minutes versus 3 minute cycling. Yet the goodness of fit of equation (12) was still 99% for the plotted points. This model of Freund et al. represents the most complete mathematical modelling yet reviewed for La recovery kinetics and avoids the criticism of Morgan (1990) that others like Campbell et al.(1989) and Hughson et al. (1987) have not paid sufficient critical attention to their mathematical models.

The y_1 velocity constant represents the ability of the muscle to exchange La between active muscle groups and venous or arterial blood. Whereas the y_2 velocity constant represents the ability of the system to remove La via metabolism.

With the decreased y_1 and y_2 values observed after 6 minutes of exercise in contradistinction to 3 minutes demonstrates a reduction in the bodies ability to exchange and remove La. A direct and inverse relationship between time and La removal is suggested. This supports a "time varying ability of the tissues to remove lactate" (Freund et al. 1989) as a concept. It is suggested that with increased duration

a

> progressive decline with time during the exercise of the ability to utilize lactate, and hence of the lactate clearance rate, when even though lactate production remains constant or even decreases (Freund et al., 1989, p. 539).

The authors also suggest this diminishing ability to remove La may be the cause of the secondary rise in La, which they suggest is observed during "prolonged submaximal exercise" (p. 539). It is the onset of a diminished La recovery or the reversal of La recovery to accumulation values that will be developed in this proposed study. Freund et al.(1978, 1981, and 1989) have not evaluated repeated 3 minute exercise bouts for cycling or for running. Finally the ability to exchange with and clear La from the blood as marked by y_1 and y_2 in Freund et al.'s model is higher for fit subjects therefore was shown to be trainable.

The sole methodological concern is that minimally intrusive fingertip blood samples are the choice method of determining [HLa] in elite exercising runners on a treadmill in a continuous protocol. Onyono-Enguelle, Gartner, Marbach, Heitz, Ott and Freund (1989) have validated the use of arterialized venous blood as comparable to arterial blood in applying their model and blood La recovery equation (12). In contrast venous blood was only considered reliable for La disappearance (y_2) and not for the La exchange parameter y_1.

II.1.7 Summary

In summary La recovery is a complex factor to consider. The factors that

control muscle [La] production like diet; O_2; training; intensity and duration of exercise; and recruitment patterns of different fiber types have not always been reflected in [HLa] values. Transmembrane flux rates have been shown to be from ionic protein carriers, proton-cotransport with a protein carrier, and undissociated bilipid diffusion. As a mediated diffusion process two La efflux or influx pathways show saturation kinetics limiting La release to 5 mM/min. Similarly La uptake has been show as limited (Karlsson 1971). But given sufficient time HLa values will equilibrate as shown in repeated work and rest intervals of a supramaximal nature. Assessing HLa recovery as a delta value controls for variations in HLa values of a day to day nature due to diet conditions of the subjects, which has been shown to right or left shift HLa responses curves. Because La exchange and recovery has been shown to be optimal at submaximal levels this should hold true as well for submaximal repeat efforts. The best mathematical model of La kinetics shows increases in both intensity and duration of exercise eventually lowers La exchange and disappearance velocities as marked by velocity constants in a two part exponential equation. In then follows that extended submaximal exercise should result in a diminished La removal ability. The equation of Freund et al., (1989) has a 99% goodness of fit. The evaluation of repeat intervals of work should show an onset of diminished La recovery or a reversal of La recovery to accumulation values as a result of the diminishment of the ability of the removal system regardless of the La production levels. Thus there is a critical work time at which at which failure of La recovery should precede a performance end point.

This can be evaluated in a SWEET protocol with baseline active rest exercise posited at an intensity that precedes that power level associated with the first increase in [HLa] levels. Interval intensities at three transition zones markers of FBLC of 2.5, 4 and 5.5 mM will survey the differing La and O_2 related conditions for the SWEET exercises.

CHAPTER THREE

III.1 Methodology

This chapter describes the subjects, apparatus, and four protocols developed to assess the effects of three selected exercise intensities on blood lactate recovery ability and the patterns of [HLa], heart rate and oxygen consumption.

III.1.1 Subjects

Twelve elite varsity, provincial or national calibre distance runners under the age of 30, volunteered and signed informed consent after having had opportunity to ask questions about the requirements of the four tests. University of Ottawa Ethics Committee approval was acquired for all aspects of the study.

Anthropometric tests for height and weight were performed to provide descriptive information on the subjects. As well data on personal best times for a 10 K cross country race, years of training, age, as well as weekly training volume were recorded.

III.1.2 Apparatus

Standard blood lactate, heart rate and oxygen consumption sampling techniques and equipment were used, the reliability and validity of which have been well documented and reported in the literature and which are described below.

III.1.2.1 Blood Lactate Determination

Aliquotes of 100 microliters of hemolised whole blood were analyzed with the Kontron Medical Lactate Analyzer 640 (Kontron Medical Equipment Inc., Switzerland) which is an electrochemical-enzymatic sensor. Cytochrome b_2 catalyses the oxidation of lactate to pyruvate producing an electrical signal which is a linear function of the enzymatic activity. The electrical signal is displayed digitaly yielding a quantitative measurement in mM of whole blood lactate concentration. Reproducibility has been shown to be ± 0.1 mM for La analyzed with the Kontron versus other methods (Geyssant, Dormois, Barthelemy & Lacour, 1985). The error of the complete sampling method was demonstrated to ± 0.05 mM (Geyssant et al., 1985).

III.1.2.2 Heart Rate Determination

Heart rate was recorded using a PE3000 (Polar Electric) monitor and microcomputer wrist receiver with an inherent error of 1/2 beat per minute based on the accuracy of the a microcomputer quartz oscillator (Karvonen, Chwalbinska-Moneta & Saynajakangas, 1984). The PE3000 sport tester microcomputer measured beat to beat responses for the first four beats, averages the values and then digitally displays the results on the wrist receiver. The subsequent displays are based on previous averages weighted to 1/2 and the most recent having the weighting of 1/16. Leger and Thivierge (1988) have found that the Sport Tester equivalent AMF Quantium XL to be highly reliable when tested against ECG with validity cited at r=.99 and the standard

error of the estimate at < 4%.

III.1.2.3 Gas Analysis.

An open circuit method was used to determine gas exchanges with the lab environment. Oxygen and carbon dioxide fractions of expired air and minute ventilation were assessed for each 30 s period of the tests. The Amtek Electrochemistry Carbon Dioxide Analyzer CD-3A and the Amtek Electrochemistry Oxygen analyzer S-3A1 were used to determine the fractions of O_2 and CO_2 in .45 L of expired air by continuous sampling during the 30 s period. The air was drawn from a mixing box and dried via a Dry Rite system. One and a half inch low resistance tubing; a two-way, nonrebreathing, T-shaped valve; and a nose clip were used to capture expired gases. Minute ventilation was measured using a Morgan Ventilation Monitor turbine on the inspiratory side of the one way valve. The above equipment was part of a Roxon Metabolic Cart System. Cross validation of ventilation was performed using a Collins 110 L Tissot tank sampling during for the last 30 seconds of each 3 minutes of each stage. Tissot elevation values were corrected for barometric pressure, temperature and gas analyzer sampling volume. The overall gas sampling process resulted in less than 5% error which is considered acceptable (Thoden et al., 1982). Stark (1989) has reported the "in-house laboratory testing" of the entire "gas measuring system to be ± 2.0 ml/kg/min." (p. 41) for MAP-LT tests for elite cross-country skiers.

III.1.2.4 Ergometer

A modified Quinton motor driven treadmill was used for running at a 2% grade. Speeds were determined using a digital output in millivolt for km/h and grade. Appendix 1 lists selective millivolt readings with the corresponding m/min (km/h) velocities for 3 minute durations.

III.1.3 Procedures

Subjects orientation and treadmill familiarization occurred on the first of four testing days. Running protocols included one La Verification Test and three Square-Wave Endurance Exercise Tests (SWEET). Day one of the testing week started with the La Verification Test. The remaining three tests were randomized using a Latin Square design to vary the order of treatments (SWEETs).

Pretest equipment calibration included the Kontron analyzer, the Roxon Metabolic Cart, and the Quinton treadmill. Calibration of the Roxon Cart for % O_2 and %CO_2 was completed using a standard gas at 17.00% O_2 and 4.00 % CO_2 before and after each test. Minute ventilation calibration was completed using a 1 Litre pump and 4 pumps at different speeds over three trials. The Calibration of the Kontron sensor preceded the La Verification Test so that La samples could be analyzed immediately during the test. La sensor calibration procedures used standard solution concentrations of 5 or 10 mM as reference values.

During-test procedures included fingertip micropuncture performed on an

outstretched hand while the subject continued to run. 70% isopropyl alcohol whipes were used to cleanse the fingertip before a precision autolancet punctured the fingertip and a drop of blood was immediately captured in a heprinized capillary tube. Approximately 20 microliters of mixed venous-arterial whole blood was drawn within the first 30 seconds of the next stage. Nonlint whipes and 3M skin tape dried and protected the micropuncture between sampling. An Eppendorf pipettewas used to transfer 20 microliters of sample into 380 microliters of hemolizing solution to bring final values to 400 ml. Surgical gloves were worn by the technician to minimize blood contagion for the tester and subject.

III.1.3.1 The Lactate Verification Test

The La Verification Test was used to quantify the three selected exercise intensities that represented the independent variable. The objective of this test was to determine the first treadmill velocity that elicited a [HLa] slightly greater than 2.5, 4.0 and 5.5 mM. These HLa concentrations were used to mark the independent variable of three selected exercise intensities for the work intervals of the SWEET tests. The three exercise intensities were selected based on Stark's 1989 three zone markers for cross-country skiers. This test was a progressive incremental test of three minute stages (Kumagai et al., 1982; Thoden et al., 1982; Kumagai et al. 1987; Stark 1989; Weltman et al., 1990) increasing by 1 km/h every stage and terminating when the subject's [HLa] reaches a value > 5.5 mM. The treadmill velocity was monitored to

the nearest mV on the modified Quinton treadmill control panel. The grade was kept constant in all tests at 2%. A three minute warm-up at 10, 11 and 12 km/h preceded every test. The velocities associated with the stage before the first rise in [HLa] (AT of Wasserman et al. 1964, 1973, McLellan and Skinner, 1982) was used as the active recovery baseline velocity for SWEET.

III.1.3.2 The Square-Wave Endurance Exercise Tests(SWEET)

The SWEET (Gimenez et al., 1982a; Gimenez et al., 1982b; Gimenez et al., 1982c) consisted of 6 three minute work intervals interspersed with 6 equal work active recovery intervals. The velocity associated with the three HLa concentrations interpolated to the nearest 1/2 km/h was set as the work interval for each of the three SWEETs. Work was controlled for by equating work and rest on a 1:1 basis such that total treadmill belt distance ran for both work and active-rest conditions were equal. See Appendix C for a table of corresponding distances and times covered in three minutes at various recovery speeds for varying work rates. Every other day after a SWEET was given as a day off to allow for full glycogen repletion between tests (Piehl, 1974). A three minute warm-up at 10, 11 and 12 km/h levels always preceded each SWEET work interval that started the test.

Weltman, Snead, Stein, Seip, Schurrer, Rutt and Weltman (1989) cite test-retest reliability coefficients for treadmill velocities at 2.5 and 4.0 mM FBLC as r=.95. Interinvestigator and intrainvestigator reliability coefficients reported by

Weltman et al.(1989) were high at r=.91 and r=.99. FBLC determination for treadmill running intensities of 2.0, 2.5, and 4.0 mM were shown by Weltman et al., (1989) to be reliable and valid with a test-retest reliability coefficient of r = 0.89, 0.91, and 0.95 for the three velocities associated with each FBLC. Duggan and Tebbutt (1989) had shown a reliability of r = 0.93 or greater for both V_{OBLA} and La_{12} ([HLa] at 12 km/h) for test-retests with no significant differences between the tests. The standard error of measurement for velocity settings is reported as ± 10.0 m/min.

III.1.3.3 Statistical Methods

Descriptive statistics of means and standard deviations were calculated for height, weight, age, years of experience, personal best 10 K time (this fall), and average weekly training volume. A one way ANOVA with repeated measures was calculated for the effects of the three exercise levels on the blood lactate recovery ability as measued by the peak in [HLa] following the over (work) periods and the delta La. The level of significance was $p < 0.05$. The pattern of heart rate, oxygen consumption and [HLa] were plotted against work time and stage.

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