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RELATIONSHIP BETWEEN HEART RATE AND BLOOD LACTATE RESPONSES TO A SPORT SPECIFIC FIELD TEST IN ELITE MALE AND FEMALE BADMINTON PLAYERS

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B.Sc., University of Ottawa, 1987

THESIS

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ABSTRACT

The relationship between heart rate and lactate is often used to measure intensity and recovery but little is known if they yield the same information. The purpose of this study was to establish the relationship between heart rate (HR) and blood lactate (BLa) responses (peak and recovery) to a sport specific field test (4-corner test) in elite male and female badminton players. 10 male and 10 female members of the Canadian badminton team were tested. After having performed the 4-corner test, two variables were measured: first, a 5 minute HR recovery (HRREC) period, during which HR values were taken from post 0 minute to post 5 minute every 30 seconds for the first 2 minutes and then every minute; and second, a 5 minute BLa recovery (BLaREC) period, from post 1 minute to post 5 minute every minute.

No statistically significant difference ($\alpha = 0.05$) between genders for any of the variables was observed, thus the data was pooled. Among all the comparisons of the 30 second intervals within the 5 minute HRREC data, only two time periods were not statistically significant ($\alpha = 0.05$). One was early in recovery (0-30 sec.) and one was late in recovery (90-120 sec. and 120-300 sec. inclusive). Mean BLa went down every minute from 6.91 mMol at post 1 minute to 6.51 mMol at post 5 minute but this difference was not statistically significant ($\alpha = 0.05$). No statistically significant correlations were found to exist between HRREC and BLaREC. However, PEAKBLa following the test was correlated with HRREC measurements such that the following two trends were found.

First, the correlation between PEAKBLa and the absolute HR values obtained at different time intervals during recovery became stronger with increasing time up to the 2nd minute of recovery at which point it plateaued (from $r = -0.24$, $p<0.30$ at post 0 min. to $r = 0.66$, $p<0.002$ at post 2 min.) and second, PEAKBLa was negatively correlated with 5 minute HRREC ($r = -0.80$, $p<0.0001$). When total heart rate recovery was broken down into the two smaller time intervals, reflecting the fast and slow components, PEAKBLa was negatively correlated with the HRREC obtained in the first two minutes of recovery ($r = -0.77$, $p<0.0001$) but this relation then became a positive one with the HRREC obtained between the 2nd and 5th minute of recovery ($r = 0.46$, $p<0.04$). These results suggest that a higher BLa concentration at the end of exercise reflects a body state which is related to a slower HRREC. It cannot be said if the effect of BLa concentration on HR is of direct or indirect nature. The results support the recommendation that if one is to use HRREC measurements to assess the fitness level of an athlete, it would be relevant to also have PEAKBLa values.

INTRODUCTION

Badminton is known as an intermittent sport in the sense that the athlete has repeated brief pauses between short bouts of exercise. For this type of event the exercise physiologist must be able to measure both the intensity of effort and the recovery from work. Two of the relevant trainable physiological parameters that are often utilized in elite athlete testing are heart rate (HR) and blood lactate (BLa). Also, these two parameters are frequently employed in a complementary fashion in order to monitor both intensity of, and recovery from, work. Both are elevated during exercise and decrease during recovery.

A relationship betweeen HR and lactate has been found to exist (Thimm et al., 1984; Saltin et al., 1981; Tibes et al., 1976) during exercise where HR increased with increased lactate. However, the relationship between HR and lactate during recovery is not as clear. Saltin et al. (1981) found that the concentration of lactic acid in the interstitial space remains elevated for several minutes and yet during this time HR was already decreasing. Conversely, Thimm et al. (1984) found that the increase in HR lasted for several minutes although the lactate concentration in perfusion outflow decreased during that time. The authors hypothesized that this prolonged effect on HR may occur because the lactate concentration in interstitial space remains relatively high due to slow back-diffusion from the interstitial space to the capillaries. However, it is also possible, as mentionned by Thimm et al. (1984), that the mechanisms which control the increase in HR are different from those which control its return to base-line. Measuring BLa during recovery, immediately following a sport-specific test, through the fairly new and fast fingertip blood sampling technique may give some insight into the relation between HR and lactate

recovery in elite athletes. The purpose of this study was thus to establish the relationship between HR and BLa responses (peak and recovery) to a sport specific field test (4-corner test) in elite male and female badminton players.

METHODS

The subjects in this study were all members of the Canadian National Badminton team. These included 10 male and 10 female players, all between the ages of 19 and 31 years of age. They performed the 4-corner badminton fitness test (Reed, 1981) which reflects the demands of their sport. All players had been previously required to give their informed consent upon accepting national team status through the respective sport-governing association. Following this, two physiological recovery variables were measured: first a 5 minute HR recovery (HRREC) period, during which HR values were taken from post 0 min. to post 5 min. every 30 sec. for the first 2 minutes and then every minute; and second a 5 min. BLa recovery (BLaREC) period, from post 1 min. to post 5 min. every minute. 5 minute is the rest period allowed between the 2nd and 3rd game in a singles badminton match. HRREC was monitored with a Sport Tester™ PE-3000 (Polar Electro Co.) and lactate samples were drawn using the finger-tip blood sampling technique. BLa concentration was determined using a Kontron Medical 640 Lactate Analyzer™ (Roche Bio-Electronics). A two way analysis of variance (ANOVA) was used to analyze the collected data where the gender and time of recovery were the two independent variables and HR and BLa concentration served as the dependent variables. In order to determine the relationship between HR and BLa (peak and recovery values), a correlational matrix was used. An index was also generated which looked at the ratio HRREC/peak BLa.

RESULTS

Table 1 gives some descriptive results of the players used in this study.

Table 1

Means and Standard Deviation of Some Descriptive Results.

Variable	N	Mean	S.D.	Minimum Value	Maximum Value
		Gender=F			
AGE	10	24.10	4.22	19.31	31.52
HEIGHT (cm)	10	165	4	161	173
WEIGHT (kg)	10	61.8	7.2	51.9	78.0
Sum Of 5 Skinfolds (CSTF protocol)	10	49.0	21.4	23.6	102.0
		Gender=M			
AGE	10	23.48	3.34	19.55	28.65
HEIGHT (cm)	10	181	4	173	190
WEIGHT (kg)	10	76.7	3.0	73.0	81.0
Sum Of 5 Skinfolds (CSTF protocol)	10	38.0	9.41	25.5	60.3

The badminton players in this study finished the 4-corner badminton test with a mean peak HR (HR0) that equals 90% of their predicted maximal HR. This corresponds to the values obtained during an international singles match (Agnevik, 1970; in Astrand, 1986). They recovered 83 ± 12 bpm in the 5 minutes following the test (HRREC5) and 83% of that recovery occured within the first 2 minutes (HRREC2) while 17% occured between the 2nd and 5th minute of recovery (HRREC25), a peak BLa (PEAKBLa) of 7.24 ± 1.49 mMol was obtained and this value droped 6% between the 1st and 5th minute of recovery

(BLaREC15) (Table 2). A listing of all the raw data can be found in Appendix A.

The ANOVA yielded no statistically significant difference (α = 0.05) between genders for any of the variables observed (Table 3), thus the gender data was pooled. HRREC measurements were significantly different (α = 0.05) among all 30 sec. intervals except between post 0 sec. and 30 sec., 90 sec. and 120 sec., and from 120 sec. to 300 sec. inclusive (Table 4). BLa went down every minute from 6.91 mMol at post 1 min. to 6.51 mMol at post 5 min. but this difference was not statistically significant (α = 0.05). Figure 1 shows the heart rate and lactate recovery curves.

Table 5 shows the correlations among the variables. Full correlational matrices can be found in Appendix B. No statistically significant correlations were found to exist between HRREC and BLaREC (see Figure 1 for HR and BLa recovery curves). However, PEAKBLa following the test was correlated with HRREC measurements. The following two trends were found: first, the correlations between PEAKBLa and the absolute HR values obtained at different time intervals during recovery become stronger with increasing time up to the 2nd minute of recovery at which point it plateaus (from $r = -0.24$, $p<0.30$ at HR0 to $r = 0.66$, $p<0.002$ at HR120) and second, PEAKBLa was negatively correlated with HRREC5 ($r = -0.80$, $p<0.0001$). However, when total heart rate recovery was broken down into the two smaller time intervals, reflecting the fast and and slow components, PEAKBLa was negatively correlated with HRREC2 ($r = -0.77$, $p<0.0001$) but this relation then became a positive one with HRREC25 ($r = 0.46$, $p<0.04$).

The index HRREC/PEAKBLa was calculated using three time intervals for HRREC; these were HRREC2, HRREC5 and HRREC25 (see Table 2 for means and definitions). Table 6 shows the correlations among these three indices. The INDEX02 strongly correlated with the INDEX05 ($r= 0.97$, $p<0.0001$). The INDEX02 and the INDEX25 correlated negatively ($r= - 0.44$, $p<0.05$); the INDEX05 and INDEX25 did not correlate significantly ($r= - 0.21$, $p<0.37$).

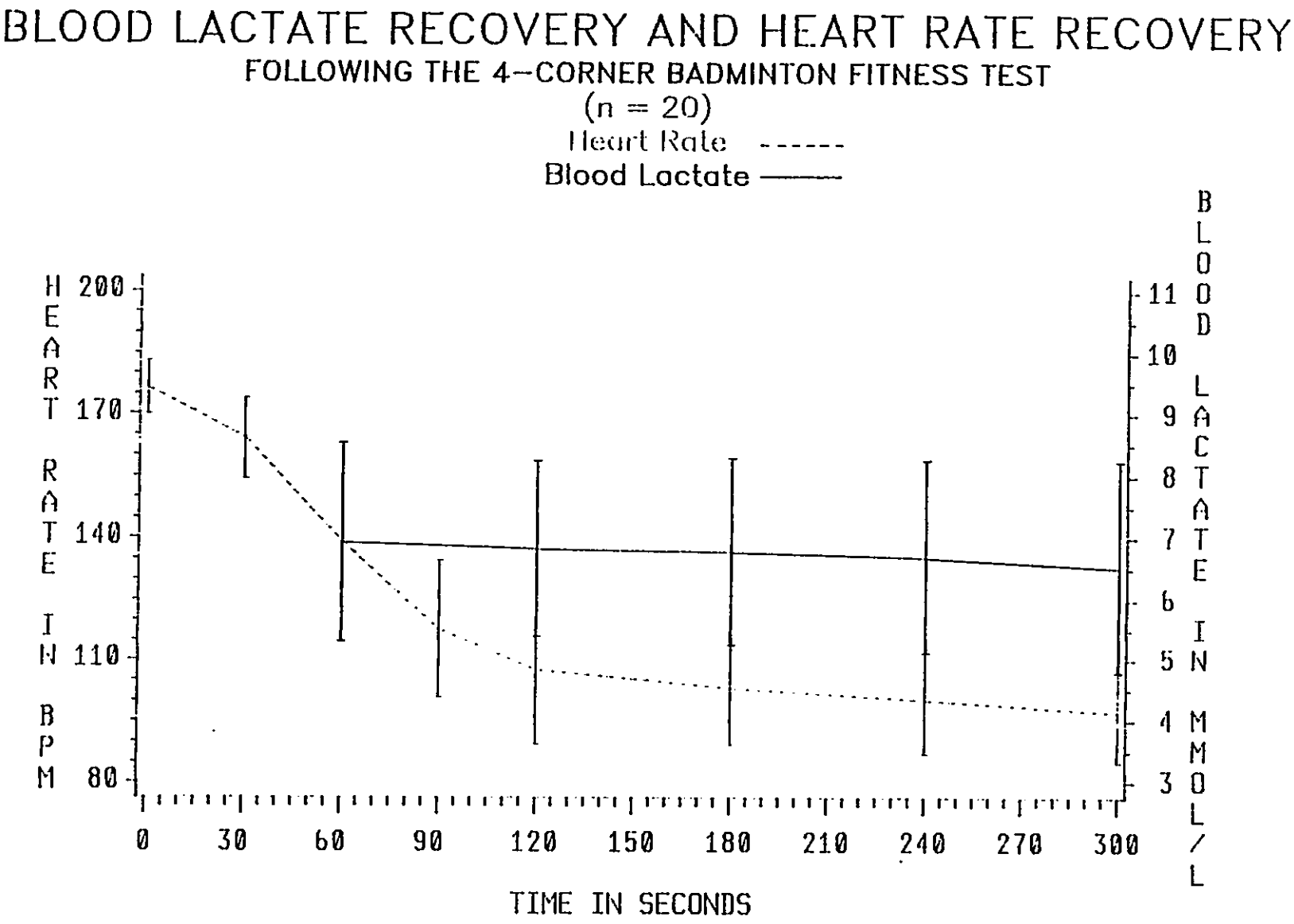
BLOOD LACTATE RECOVERY AND HEART RATE RECOVERY
FOLLOWING THE 4–CORNER BADMINTON FITNESS TEST
(n = 20)
Heart Rate ------
Blood Lactate ——
HEART RATE IN BPM
H 200
170
140
110
80
BLOOD LACTATE IN MMOL/L
11
10
9
8
7
6
5
4
3
0
30
60
90
120
150
180
210
240
270
300
TIME IN SECONDS

Figure 1

Table 2

Means and Standard Deviation (n = 20).

Variable		Mean	S.D.	Minimum Value	Maximum Value
HR0	(bpm)	176	7	163	185
HR30	(bpm)	164	10	147	184
HR60	(bpm)	139	16	109	166
HR90	(bpm)	118	17	94	145
HR120	(bpm)	107	18	79	137
HR180	(bpm)	100	15	75	130
HR240	(bpm)	96	15	71	124
HR300	(bpm)	93	14	73	120
HRREC5	(bpm)	83	12	58	108
HRREC2	(bpm)	69	17	41	104
HRREC15	(bpm)	46	12	32	79
HRREC905	(bpm)	24	9	4	44
HRREC25	(bpm)	14	8	4	29
HRREC35	(bpm)	7	6	-1	19
HRREC45	(bpm)	2	4	-8	11
LACTATE1	(mMol)	6.91	1.61	4.82	10.98
LACTATE2	(mMol)	6.81	1.42	4.72	10.26
LACTATE3	(mMol)	6.77	1.52	4.50	10.58
LACTATE4	(mMol)	6.68	1.57	4.20	10.30
LACTATE5	(mMol)	6.51	1.72	4.08	10.60
PEAKBLa	(mMol)	7.24	1.49	4.82	10.98
LACREC15	(mMol)	0.40	0.91	-1.58	1.92
LACRECPE	(mMol)	1.02	0.44	0.44	1.92
INDEX02		10.27	4.27	3.73	21.58
INDEX05		12.15	3.93	5.28	22.41
INDEX25		1.89	1.03	0.53	4.80

HR0 to HR300 = Heart rate at given recovery time in seconds, HRREC2 = HRREC in the first 2 minutes, HRREC5 = HRREC in 5 minutes, HRREC15 = HRREC between the 1st and 5th min. of recovery, HRREC905 = HRREC between 90sec. and the 5th min. of recovery, HRREC25 = HRREC between the 2nd and 5th min. of recovery, HRREC35 = HRREC between the 3rd and 5th min. of recovery, HRREC45 = HRREC between the 4th and 5th min. of recovery, LACTATE1 to LACTATE5 = BLa at given recovery time in minutes, PEAKBLa = Peak BLa, LACREC15 = LACTATE1 - LACTATE5, LACRECPE = BLaREC from Peak BLa, INDEX02 = HRREC2/PEAKBLa, INDEX05 = HRREC5/PEAKBLa, INDEX25 = HRREC25/PEAKBLa.

Table 3

Analysis of Variance for gender.

Source	Sum of Squares	Degrees of Freedom	Mean Square	F value
PEAKBLa	42.29	19	2.24	0.88
LACREC15	15.68	19	0.83	0.87
HR0	824.55	19	45.80	0.00
HRREC2	5788.95	19	312.58	0.52
HRREC5	2915.80	19	160.39	0.18

*F value for $p<0.05$ is 3.18

Table 4

Tukey post-hoc test on heart rate recovery values (n=20).

Time (sec.)	Mean HR (bpm)	Significance
0	176	Sig. dif. from ≥ 60
30	164	Sig. dif. from ≥ 60
60	139	Sig. dif. from all others
90	118	Sig. dif. from 0-60, ≥ 180
120	107	Sig. dif. from 0-90
180	100	"
240	96	"
300	93	"

Table 5

Pearson correlation coefficients and levels of significance for the variables investigated (n = 20).

		PEAKBLa	LACREC15
HR0	(bpm)	r= - 0.24, n.s.	
HR30	(bpm)	r= 0.11, n.s.	
HR60	(bpm)	r= 0.23, n.s.	
HR90	(bpm)	r= 0.53, p<0.02	
HR120	(bpm)	r= 0.66, p<0.002	
HR180	(bpm)	r= 0.53, p<0.02	
HR240	(bpm)	r= 0.55, p<0.01	
HR300	(bpm)	r= 0.60, p<0.005	
HRREC2	(bpm)	r= - 0.77, p<0.0001	r= - 0.01, n.s.
HRREC5	(bpm)	r= - 0.80, p<0.0001	r= - 0.02, n.s.
HRREC15	(bpm)	r= - 0.38, n.s	
HRREC905	(bpm)	r= 0.08, n.s.	
HRREC25	(bpm)	r= 0.45, p<0.04	r= - 0.01, n.s.
HRREC35	(bpm)	r= - 0.03, n.s.	
HRREC45	(bpm)	r= 0.04, n.s.	

Table 6

Pearson correlation coefficients and levels of significance for the three indices generated (n = 20).

	INDEX02	INDEX25
INDEX02	X	
INDEX25	r= - 0.44, p<0.05	X
INDEX05	r= 0.97, p<0.0001	r= - 0.21, n.s.

DISCUSSION

A fast HRREC phase was found to occur up to 2 minutes in recovery following which HR kept decreasing but not in a statistically significant way (slow phase)(Figure 1). These two phases concur in time with those found in previous studies (Pilardeau et al., 1982; Morand et al., 1978). BLa decreased from the first minute to the fifth minute of recovery but the difference was not statistically significant. Some research has been done in relation to lactate removal rates but most studies have looked at lactate removal over a relatively long period of time (20 minutes or more) (Bonen and Belcastro, 1976; Hermansen and Stensvold, 1972). The results obtained in this study support the mention by Evans and Cureton (1983) that a study measuring the effects of conditionning on the peak rate of blood lactate removal is needed. Since the rest period between the second and third games of a badminton match is 5 minutes, it was important to examine the lactate and heart rate recoveries of badminton players within this time period.

Gender had no significant effect (α=0.05) on either HR (peak and recovery) or BLa (peak and recovery) values. Though not significantly, PEAKBLa was higher in men than in women. The homogeneity of this group, the similarity of fiber type distribution between genders and the fact that this was not a test for maximal lactate response may all join to explain why men, with generally larger muscle mass and higher power output, did not produce significantly more lactate than women. In the pooled data, BLaREC did not significantly correlate with HRREC. This may be explained by the different nature of the mechanisms involved since HRREC control mechanisms are more of a neural

nature while BLaREC mechanisms are relatively more of a vascular and metabolic nature.

Unlike BLaREC, PEAKBLa did correlate with HRREC. The correlations between PEAKBLa and HR measurements taken every 30 seconds during recovery show that in the first minute there is no significant relationship between heart rate and PEAKBLa but from 90 seconds to 5 minutes, higher PEAKBLa had a statistically significant tendency (α=0.05) to be related to higher heart rates at any given time whith this tendency becoming strongest at 2 minutes ($r = 0.66$, $p<0.002$). PEAKBLa also correlated negatively with HRREC5 ($r = -0.80$, $p<0.0001$) but the relation changed to a positive one when correlating PEAKBLa to HRREC25 ($r = 0.46$, $p<0.04$). Thus, the conclusion that higher PEAKBLa following the 4-corner test is significantly related to overall lower heart rate recovery in 5 minutes seems justified. Moreover, higher PEAKBLa is significantly related to that part of the 5 minute recovery which occurs from 2 to 5 minutes.

HR during exercise is affected by many physiological factors: 1) central command (Rowell, 1980), 2) humoral drives (Ruddel et al., 1986; Nielsen et al., 1984), 3) neurogenic reflexes from the muscle that may be initiated by the following different factors: mechanical (Kniffki et al., 1981; Kumazawa and Mizumura, 1977), $[K^+]$ (Rybicki et al., 1984; Tibes et al., 1978), $[H^+]$ (Thimm et al., 1984; Thimm and Meier zu Verl, 1984), osmolality (Tibes et al, 1978; Tibes and Groth, 1977), lactate concentration (Thimm et al., 1984; Thimm and Meier zu Verl, 1984), Pi concentration (Tibes et al., 1978; Tibes and Groth, 1977), thermal stimuli (Kniffki et al., 1981; Kumazawa and Mizumura, 1977), or a combination of these parameters.

As previously mentionned, a relationship between HR and lactate (muscle and arterial) had been found to exist during exercise (Thimm et al., 1984; Saltin et al., 1981) where HR increased with increased lactate. However, as mentionned by Thimm et al. (1984), the mechanisms which control the increase in HR may be different from those which control it's return to baseline. The results obtained in this study would support the conclusion that BLa concentration is related to HRREC. In 1942, Johnson et al. (in Degre and Denolin, 1965) examined the first three minutes of HRREC and found that a linear relationship existed between the average HRREC during those three minutes and the total of the HRs added from the whole effort. Thus, the more intense the work, the higher the average recovery HR. Further these findings support the conclusion that the nature of the relationship between BLa and HRREC is such that the higher the PEAKBLa (more intense work), the higher the average recovery HR with the correlation becoming statistically significant at 90 seconds ($r= 0.53$, $p<0.02$).

Again these results seem to justify the conclusion that a higher BLa concentration at the end of exercise is related to a slower HRREC. It cannot be said if the relationship between BLa concentration and HR is of direct or indirect nature. However, it may be recommended that if one is to use HRREC measurements to assess the fitness level of an athlete, it would be relevant for a more accurate interpretation of these results to also have PEAKBLa values. More studies, however, are needed to examine the effect of exercise specificity on the relationship between HRREC and PEAKBLa and to see if the relationship found in this study may be generalised to other athletes. Also, some studies are needed to examine the effects of different types of training on heart rate and blood lactate recoveries and to examine the effect of test specificity on

heart rate and blood lactate recoveries. The relationship found in this study is specific to lactate ranges obtained from this test and don't necessarely reflect relationships obtained from other ranges of lactate. If the relationships found in this study between HRREC and PEAKBLa does show to be true for other intermittent sports then the INDEX02, which correlates very strongly with the INDEX05 ($r= 0.97$, $p<0.0001$), may be of use to monitor training. If for a given intermittent sport, both a good alactic capacity (monitored through low PEAKBLa) and aerobic fitness for recovery (as monitored through HRREC) are needed, then an improvement of any one or both of these parameters following a sport-specific test would increase the INDEX02 and thus show an improvement in the level of specific fitness required for these athletes.

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Appendix A

Descriptive Results

Gender=Female

	Subjects									
Variables	1	2	3	4	5	6	7	8	9	10
AGE(years)	21.3	28.5	23.3	19.3	25.3	19.8	28.5	20.5	22.8	31.5
HEIGHT(cm)	166.0	163.0	161.6	165.0	171.5	162.0	170.5	164.0	161.0	173.0
WEIGHT(kg)	61.4	54.9	61.4	63.2	57.9	51.9	78.0	65.0	58.5	65.7
SUMSKIN(mm)	48.9	23.6	38.1	60.7	42.5	33.0	102.0	53.1	50.1	38.5
VERTJUMP(cm)	43.5	41.0	47.0	39.5	41.0	30.0	40.5	50.0	49.0	41.0
AVSPEED(sec)	7.62	8.75	8.28	8.60	8.19	8.49	8.80	9.97	7.52	8.58
DROPOFF(sec)	0.18	0.18	0.51	0.50	0.43	0.46	0.98	0.74	0.32	0.45
HR0(bpm)	166	168	185	179	184	183	163	178	174	183
HR30(bpm)	158	153	173	158	184	175	154	168	154	166
HR60(bpm)	137	113	145	127	166	152	130	156	121	154
HR90(bpm)	123	94	108	99	127	134	106	144	94	114
HR120(bpm)	116	85	100	99	119	119	104	137	86	79
HR180(bpm)	106	75	101	101	106	109	83	130	86	94
HR240(bpm)	94	71	96	102	100	110	90	124	76	79
HR300(bpm)	93	73	94	95	102	110	82	120	78	75
HRREC30(bpm)	8	15	12	21	0	8	9	10	20	17
HRREC60(bpm)	29	55	40	52	18	31	33	22	53	29
HRREC90(bpm)	43	74	77	80	57	49	57	34	80	69
HRREC2(bpm)	50	83	85	80	65	64	59	41	88	104
HRREC5(bpm)	73	95	91	84	82	73	81	58	96	108
HRREC15(bpm)	44	40	51	32	64	42	48	36	43	79
HRREC905(bpm)	30	21	14	4	25	24	24	24	16	39
HRREC25(bpm)	23	12	6	4	17	9	22	17	8	4
HRREC35(bpm)	13	2	7	6	4	-1	1	10	8	19
HRREC45(bpm)	1	-2	2	7	-2	0	8	4	-2	4
HRRECSP	58.0	66.3	47.1	65.0	27.7	48.4	55.9	53.7	60.2	27.9
LACTATE1*	6.42	6.14	5.10	5.96	6.88	5.10	8.00	10.98	5.76	4.82
LACTATE2*	7.76	6.50	6.54	4.76	6.12	5.76	7.26	10.26	5.54	4.72
LACTATE3*	7.48	6.22	6.60	4.60	6.18	5.68	7.24	10.58	5.52	4.50
LACTATE4*	7.64	6.36	6.60	4.48	6.50	5.32	7.48	10.30	5.26	4.20
LACTATE5*	8.00	6.06	6.36	4.42	6.10	5.10	7.12	10.60	5.22	4.08
PEAKBLa*	8.00	6.50	6.60	5.96	6.88	5.76	8.00	10.98	5.76	4.82
LACREC15*	-1.58	0.08	-1.26	1.54	0.78	0.00	0.88	0.38	0.54	0.74
INDEX02**	6.25	12.77	12.88	13.42	9.45	11.11	7.38	3.73	15.28	21.58
INDEX25**	2.88	1.84	0.91	0.67	2.47	1.56	2.75	1.55	1.39	0.83
INDEX05**	9.12	14.61	13.79	14.09	11.92	12.67	10.12	5.28	16.67	22.41

* mMol
**bpm/mMol

Appendix A (continued)
Descriptive Results

Gender=Male

	Subjects									
Variables	1	2	3	4	5	6	7	8	9	10
AGE(years)	26.7	23.0	19.5	28.6	27.9	21.9	20.8	21.8	19.8	24.7
HEIGHT(cm)	182.0	173.0	179.0	181.0	179.2	182.0	179.0	185.5	190.0	181.5
WEIGHT(kg)	81.7	73.9	76.8	75.2	77.1	80.1	73.1	73.0	79.2	76.9
SUMSKIN(mm)	43.7	35.6	34.0	25.5	34.0	39.1	32.8	42.4	32.9	60.3
VERTJUMP(cm)	49.0	64.5	52.5	51.0	53.0	52.5	63.0	58.0	61.0	61.0
AVSPEED(sec)	8.22	8.41	8.24	7.28	8.98	8.15	8.75	7.96	8.29	8.15
DROPOFF(sec)	0.63	0.44	0.69	0.55	0.59	0.51	0.46	0.29	0.76	0.61
HR0(bpm)	181	180	181	170	181	181	175	172	168	175
HR30(bpm)	174	169	165	147	166	170	161	166	147	172
HR60(bpm)	153	150	139	109	144	144	135	139	112	155
HR90(bpm)	138	133	124	95	113	129	112	124	95	145
HR120(bpm)	123	126	121	89	95	122	92	119	83	132
HR180(bpm)	112	115	113	76	89	115	86	108	81	115
HR240(bpm)	105	106	111	74	90	109	79	110	81	112
HR300(bpm)	94	103	104	74	85	110	87	105	79	106
HRREC30(bpm)	7	11	16	23	15	11	14	6	21	3
HRREC60(bpm)	28	30	42	61	37	37	40	33	56	20
HRREC90(bpm)	43	47	57	75	68	52	63	48	73	30
HRREC2(bpm)	58	54	60	81	86	59	83	53	85	43
HRREC5(bpm)	87	77	77	96	96	71	88	67	89	69
HRREC15(bpm)	59	47	35	35	59	34	48	34	33	49
HRREC905(bpm)	44	30	20	21	28	19	25	19	16	39
HRREC25(bpm)	29	23	17	15	10	12	5	14	4	26
HRREC35(bpm)	18	12	9	2	4	5	-1	3	2	9
HRREC45(bpm)	11	3	7	0	5	-1	-8	5	2	6
HRRECSP	48.3	55.6	70.0	75.3	43.0	62.7	48.2	62.3	65.9	46.5
LACTATE1*	6.04	8.70	6.84	5.42	6.12	8.44	6.60	8.22	7.28	9.48
LACTATE2*	5.68	8.80	6.40	6.34	5.26	7.28	7.14	8.30	7.48	8.40
LACTATE3*	5.56	8.38	5.72	6.32	5.14	8.18	7.60	8.04	7.46	8.36
LACTATE4*	5.52	8.90	5.70	6.06	4.78	7.80	7.42	7.82	7.16	8.32
LACTATE5*	5.32	8.86	4.92	5.74	4.48	8.04	7.28	6.86	6.90	8.84
PEAKBLa*	6.04	8.90	6.84	6.34	6.12	8.44	7.60	8.30	7.48	9.48
LACREC15*	0.72	-0.16	1.92	-0.32	1.64	0.40	-0.68	1.36	0.38	0.64
INDEX02**	9.60	6.07	8.77	12.78	14.05	6.99	10.92	6.39	11.36	4.54
INDEX25**	4.80	2.58	2.48	2.37	1.63	1.42	0.66	1.69	0.53	2.74
INDEX05**	14.40	8.65	11.25	15.14	15.69	8.41	11.57	8.07	11.90	7.28

* mMol
**bpm/mMol

SUMSKIN=Sum of 5 skinfolds (CSTF), VERTJUMP=Vertical jump height, AVSPEED=Average time to complete the 4-corner test pattern, DROPOFF=Slowest pattern - fastest pattern, HRRECSP= (HRREC60/HRREC2)X100, (see Table 2 for definitions of the other variables).

APPENDIX B

CORRELATIONS AMONG THE VARIABLES

PEARSON CORRELATION COEFFICIENTS / PROB > |R| UNDER HO:RHO=0 / N = 20

	HR0	HR30	HR60	HR90	HR120	HR180	HR240	HR300	HRREC2	HRREC5	HRREC15	HRREC905	HRREC25	HRREC35	HRREC45
HR0	1.00000 0.0000	0.78024 0.0001	0.68969 0.0008	0.42703 0.0604	0.26809 0.2531	0.51752 0.0194	0.42580 0.0612	0.44947 0.0468	0.10054 0.6732	0.02883 0.9040	0.39969 0.0808	0.10908 0.6471	-0.17946 0.4490	0.30491 0.1931	0.06418 0.7883
HR30	0.78024 0.0001	1.00000 0.0000	0.93557 0.0001	0.76816 0.0001	0.64362 0.0022	0.71752 0.0006	0.65795 0.0016	0.68751 0.0008	-0.37023 0.1081	-0.35440 0.1252	0.45415 0.0443	0.37612 0.1022	0.26604 0.2569	0.26644 0.2562	0.12116 0.6109
HR60	0.68969 0.0008	0.93557 0.0001	1.00000 0.0000	0.84530 0.0001	0.66577 0.0014	0.76637 0.0001	0.67537 0.0011	0.67606 0.0011	-0.42727 0.0602	-0.38974 0.0894	0.55196 0.0116	0.53356 0.0154	0.33768 0.1454	0.42612 0.0610	0.21743 0.3571
HR90	0.42703 0.0604	0.76816 0.0001	0.84530 0.0001	1.00000 0.0000	0.90258 0.0001	0.89247 0.0001	0.84031 0.0001	0.83579 0.0001	-0.77096 0.0001	-0.70815 0.0005	0.16744 0.4804	0.57768 0.0076	0.60148 0.0050	0.37842 0.0999	0.28738 0.2192
HR120	0.26809 0.2531	0.64362 0.0022	0.66577 0.0014	0.90258 0.0001	1.00000 0.0000	0.90362 0.0001	0.92451 0.0001	0.91333 0.0001	-0.93156 0.0001	-0.87943 0.0001	-0.15674 0.5093	0.28508 0.2231	0.68897 0.0008	0.22019 0.3509	0.33565 0.1480
HR180	0.51752 0.0194	0.71752 0.0006	0.76637 0.0001	0.89247 0.0001	0.90362 0.0001	1.00000 0.0000	0.94040 0.0001	0.92928 0.0001	-0.73789 0.0002	-0.76464 0.0001	-0.04229 0.8595	0.26296 0.3020	0.43751 0.0537	0.44156 0.0513	0.34066 0.1416
HR240	0.42580 0.0612	0.65795 0.0016	0.67537 0.0011	0.84031 0.0001	0.92451 0.0001	0.94040 0.0001	1.00000 0.0000	0.95883 0.0001	-0.79407 0.0001	-0.84648 0.0001	-0.19559 0.4086	0.10409 0.6623	0.43325 0.0564	0.20894 0.3767	0.45428 0.0442
HR300	0.44947 0.0468	0.68751 0.0008	0.67606 0.0011	0.83579 0.0001	0.91333 0.0001	0.92928 0.0001	0.95883 0.0001	1.00000 0.0000	-0.77360 0.0001	-0.87997 0.0001	-0.24128 0.3055	0.03465 0.8847	0.33410 0.1500	0.07891 0.7409	0.18259 0.4410
HRREC2	0.10054 0.6732	-0.37023 0.1081	-0.42727 0.0602	-0.77096 0.0001	-0.93156 0.0001	-0.73789 0.0002	-0.79407 0.0001	-0.77360 0.0001	1.00000 0.0000	0.91910 0.0001	0.31272 0.1795	-0.25324 0.2813	-0.77926 0.0001	-0.11233 0.6373	-0.32241 0.1656
HRREC5	0.02883 0.9040	-0.35440 0.1252	-0.38974 0.0894	-0.70815 0.0005	-0.87943 0.0001	-0.76464 0.0001	-0.84648 0.0001	-0.87997 0.0001	0.91910 0.0001	1.00000 0.0000	0.48253 0.0312	0.01923 0.9359	-0.46929 0.0368	0.07385 0.7570	-0.17018 0.4732
HRREC15	0.39969 0.0808	0.45415 0.0443	0.55196 0.0116	0.16744 0.4804	-0.15674 0.5093	-0.04229 0.8595	-0.19559 0.4086	-0.24128 0.3055	0.31272 0.1795	0.48253 0.0312	1.00000 0.0000	0.66350 0.0014	0.06664 0.7801	0.47192 0.0357	0.07971 0.7383
HRREC905	0.10908 0.6471	0.37612 0.1022	0.53356 0.0154	0.57768 0.0076	0.28508 0.2231	0.26296 0.3020	0.10409 0.6623	0.03465 0.8847	-0.25324 0.2813	0.01923 0.9359	0.66350 0.0014	1.00000 0.0000	0.59814 0.0053	0.57151 0.0085	0.25165 0.2845
HRREC25	-0.17946 0.4490	0.26604 0.2569	0.33768 0.1454	0.60148 0.0050	0.68897 0.0008	0.43751 0.0537	0.43325 0.0564	0.33410 0.1500	-0.77926 0.0001	-0.46929 0.0368	0.06664 0.7801	0.59814 0.0053	1.00000 0.0000	0.36921 0.1091	0.45190 0.0455
HRREC35	0.30491 0.1931	0.26644 0.2562	0.42612 0.0610	0.37842 0.0999	0.22019 0.3509	0.44156 0.0513	0.20894 0.3767	0.07891 0.7409	-0.11233 0.6373	0.07385 0.7570	0.47192 0.0357	0.57151 0.0085	0.36921 0.1091	1.00000 0.0000	0.47585 0.0339
HRREC45	0.06418 0.7883	0.12116 0.6109	0.21743 0.3571	0.28738 0.2192	0.33565 0.1480	0.34066 0.1416	0.45428 0.0442	0.18259 0.4410	-0.32241 0.1656	-0.17018 0.4732	0.07973 0.7383	0.25165 0.2845	0.45190 0.0455	0.47585 0.0339	1.00000 0.0000
LACTATE1	-0.17209 0.4681	0.14585 0.5395	0.27143 0.2470	0.55055 0.0119	0.66299 0.0014	0.54769 0.0126	0.59853 0.0053	0.60278 0.0049	-0.74965 0.0001	-0.76601 0.0001	-0.32466 0.1625	0.10600 0.6565	0.46169 0.0404	0.01395 0.9535	0.38128 0.4444
LACTATE2	-0.30931 0.1845	0.03506 0.8833	0.34615 0.1387	0.47281 0.0353	0.58417 0.0068	0.45248 0.0452	0.46139 0.0406	0.51580 0.0199	-0.72003 0.0003	-0.74165 0.0002	-0.39123 0.0883	0.09379 0.6941	0.43405 0.0559	-0.03175 0.8943	-0.02067 0.9311
LACTATE3	-0.26947 0.2506	0.05914 0.8044	0.15775 0.5065	0.46293 0.0398	0.55913 0.0104	0.41842 0.0532	0.44245 0.0508	0.52524 0.0174	-0.67913 0.0010	-0.73104 0.0003	-0.38664 0.0922	0.06177 0.7959	0.35928 0.1198	-0.09262 0.6977	-0.11588 0.6266
LACTATE4	-0.28364 0.2256	0.08314 0.7275	0.17789 0.4531	0.46608 0.0383	0.58256 0.0070	0.41795 0.0534	0.43619 0.0544	0.51266 0.0208	-0.70868 0.0005	-0.72450 0.0003	-0.34588 0.1352	0.08621 0.7178	0.43591 0.0547	-0.06334 0.7908	-0.09740 0.6829
LACTATE5	-0.26087 0.2666	0.08902 0.7090	0.20396 0.3884	0.48749 0.0297	0.57803 0.0076	0.45871 0.0419	0.42613 0.0610	0.50635 0.0227	-0.69541 0.0007	-0.70533 0.0005	-0.30440 0.1919	0.13456 0.5717	0.41666 0.0542	0.00803 0.9732	-0.11311 0.6349
PEAKLACT	-0.24215 0.3037	0.10902 0.6473	0.22772 0.3343	0.53177 0.0158	0.65842 0.0016	0.52653 0.0171	0.55553 0.0110	0.59895 0.0053	-0.77137 0.0001	-0.79899 0.0001	-0.37730 0.1004	0.07748 0.7454	0.45791 0.0423	-0.03387 0.8873	0.04439 0.8526
LACREC15	0.38946 0.4237	0.09007 0.7057	0.09496 0.6905	0.05258 0.8258	0.08042 0.7361	0.10210 0.6684	0.25421 0.2795	0.10951 0.6458	-0.01155 0.9614	-0.02180 0.9273	0.00114 0.9962	-0.06711 0.7786	-0.00878 0.9707	0.00954 0.9681	0.53656 0.0147
INDEX02	0.22077 0.3496	-0.23687 0.3147	-0.26618 0.2566	-0.62936 0.0029	-0.83538 0.0001	-0.60190 0.0050	-0.68510 0.0009	-0.70356 0.0005	0.94605 0.0001	0.90467 0.0001	0.44562 0.0489	-0.09961 0.6761	-0.68331 0.0009	0.08454 0.7231	-0.16484 0.4874
INDEX05	0.21531 0.3620	-0.19059 0.4209	-0.21887 0.3539	-0.56218 0.0099	-0.77178 0.0001	-0.57937 0.0074	-0.67228 0.0012	-0.72402 0.0003	0.87831 0.0001	0.92466 0.0001	0.53109 0.0160	0.05310 0.8241	-0.49769 0.0256	0.19505 0.4099	-0.05628 0.8137
INDEX25	-0.09359 0.6947	0.25499 0.2779	0.26862 0.2521	0.46420 0.0392	0.51836 0.0192	0.28439 0.2243	0.27464 0.2413	0.35344 0.5184	-0.57065 0.0086	-0.22146 0.3481	0.18014 0.4473	0.61684 0.0038	0.92666 0.0001	0.39180 0.0849	0.46949 0.0368

APPENDIX B

CORRELATIONS AMONG THE VARIABLES

PEARSON CORRELATION COEFFICIENTS / PROB > |R| UNDER HO:RHO=0 / N = 20

	LACTATE1	LACTATE2	LACTATE3	LACTATE4	LACTATE5	PEAKLACT	LACREC15	INDEX02	INDEX05	INDEX25
HR0	-0.17209 0.4681	-0.30931 0.1845	-0.26947 0.2506	-0.28364 0.2256	-0.26087 0.2666	-0.24215 0.3037	0.18946 0.4237	0.22077 0.3496	0.21531 0.3620	-0.09359 0.6947
HR30	0.14585 0.5395	0.03506 0.8833	0.05914 0.8044	0.08314 0.7275	0.08902 0.7090	0.10902 0.6473	0.09007 0.7057	-0.23687 0.3147	-0.19059 0.4209	0.25499 0.2779
HR60	0.27143 0.2470	0.14615 0.5387	0.15775 0.5065	0.17789 0.4531	0.20396 0.3884	0.22772 0.3343	0.09496 0.6905	-0.26618 0.2566	-0.21887 0.3539	0.26862 0.2521
HR90	0.55055 0.0119	0.47281 0.0353	0.46293 0.0398	0.46608 0.0383	0.48749 0.0292	0.53177 0.0158	0.05258 0.8258	-0.62936 0.0029	-0.56218 0.0099	0.46420 0.0392
HR120	0.66299 0.0014	0.58417 0.0068	0.55913 0.0104	0.58256 0.0070	0.57803 0.0076	0.65842 0.0016	0.08042 0.7361	-0.83538 0.0001	-0.77178 0.0001	0.51836 0.0192
HR180	0.54769 0.0124	0.45248 0.0452	0.43842 0.0532	0.43795 0.0534	0.45871 0.0419	0.52653 0.0171	0.10210 0.6684	-0.60190 0.0050	-0.57937 0.0074	0.28439 0.2243
HR240	0.59853 0.0053	0.46139 0.0406	0.44245 0.0508	0.43639 0.0544	0.42613 0.0610	0.55553 0.0110	0.25421 0.2795	-0.68510 0.0009	-0.67228 0.0012	0.27464 0.2413
HR300	0.60278 0.0049	0.51580 0.0199	0.52524 0.0174	0.51266 0.0208	0.50635 0.0227	0.59895 0.0053	0.10951 0.6458	-0.70356 0.0005	-0.72402 0.0003	0.15344 0.5184
HRREC2	-0.74965 0.0001	-0.72003 0.0003	-0.67913 0.0010	-0.70868 0.0005	-0.69541 0.0007	-0.77137 0.0001	-0.01155 0.9614	0.94605 0.0001	0.87831 0.0001	-0.57065 0.0086
HRREC5	-0.76601 0.0001	-0.74165 0.0002	-0.73104 0.0003	-0.72450 0.0003	-0.70533 0.0005	-0.79899 0.0001	-0.02180 0.9273	0.90467 0.0001	0.92466 0.0001	-0.22146 0.3481
HRREC15	-0.32466 0.1625	-0.39123 0.0881	-0.38664 0.0922	-0.34588 0.1352	-0.30440 0.1919	-0.37790 0.1004	0.00114 0.9962	0.44562 0.0489	0.53109 0.0160	0.18014 0.4473
HRREC905	0.10600 0.6565	0.09379 0.6941	0.06177 0.7959	0.08621 0.7178	0.13456 0.5717	0.07748 0.7454	-0.06711 0.7786	-0.09961 0.6761	0.05310 0.8241	0.61684 0.0038
HRREC25	0.46169 0.0404	0.43405 0.0559	0.35928 0.1198	0.43591 0.0547	0.43666 0.0542	0.45791 0.0423	-0.00878 0.9707	-0.68131 0.0009	-0.49769 0.0256	0.92666 0.0001
HRREC35	0.01395 0.9535	-0.03175 0.8943	-0.09262 0.6977	-0.06334 0.7908	0.00803 0.9732	-0.03387 0.8873	0.00954 0.9681	0.08454 0.7231	0.19505 0.4099	0.39480 0.0849
HRREC45	0.18128 0.4444	-0.02067 0.9311	-0.11588 0.6266	-0.09740 0.6829	-0.11311 0.6349	0.04439 0.8526	0.53656 0.0147	-0.16484 0.4874	-0.05628 0.8137	0.46949 0.0368
LACTATE1	1.00000 0.0000	0.86863 0.0001	0.87007 0.0001	0.86683 0.0001	0.85383 0.0001	0.94693 0.0001	0.15549 0.5127	-0.81461 0.0001	-0.84614 0.0001	0.14769 0.5343
LACTATE2	0.86863 0.0001	1.00000 0.0000	0.97702 0.0001	0.98117 0.0001	0.95216 0.0001	0.95914 0.0001	-0.26439 0.2600	-0.82730 0.0001	-0.86790 0.0001	0.11719 0.6227
LACTATE3	0.87007 0.0001	0.97702 0.0001	1.00000 0.0000	0.98712 0.0001	0.97294 0.0001	0.96251 0.0001	-0.30127 0.1968	-0.80553 0.0001	-0.86310 0.0001	0.04510 0.8502
LACTATE4	0.86683 0.0001	0.98117 0.0001	0.98712 0.0001	1.00000 0.0000	0.98143 0.0001	0.96363 0.0001	-0.32312 0.1647	-0.83417 0.0001	-0.87442 0.0001	0.12079 0.6120
LACTATE5	0.85383 0.0001	0.95216 0.0001	0.97294 0.0001	0.98143 0.0001	1.00000 0.0000	0.95672 0.0001	-0.38145 0.0970	-0.80508 0.0001	-0.84448 0.0001	0.11447 0.6308
PEAKLACT	0.94693 0.0001	0.95914 0.0001	0.96251 0.0001	0.96363 0.0001	0.95672 0.0001	1.00000 0.0000	-0.13400 0.5733	-0.87188 0.0001	-0.91489 0.0001	0.12262 0.6065
LACREC15	0.15549 0.5127	-0.26439 0.2600	-0.30127 0.1968	-0.32312 0.1647	-0.38145 0.0970	-0.13400 0.5733	1.00000 0.0000	0.08121 0.7336	0.09997 0.6750	0.04504 0.8505
INDEX02	-0.81461 0.0001	-0.82730 0.0001	-0.80553 0.0001	-0.83417 0.0001	-0.80508 0.0001	-0.87188 0.0001	0.08121 0.7336	1.00000 0.0000	0.97194 0.0001	-0.43665 0.0542
INDEX05	-0.84614 0.0001	-0.86790 0.0001	-0.86310 0.0001	-0.87442 0.0001	-0.84448 0.0001	-0.91489 0.0001	0.09997 0.6750	0.97194 0.0001	1.00000 0.0000	-0.21278 0.3678
INDEX25	0.14769 0.5343	0.11719 0.6227	0.04510 0.8502	0.12079 0.6120	0.11447 0.6308	0.12262 0.6065	0.04504 0.8505	-0.43665 0.0542	-0.21278 0.3678	1.00000 0.0000

RELATIONSHIP BETWEEN HEART RATE AND BLOOD LACTATE RESPONSES TO A SPORT SPECIFIC FIELD TEST IN ELITE MALE AND FEMALE BADMINTON PLAYERS

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CONTENTS

THE PROBLEM

INTRODUCTION

Kinanthropology can generally be defined as the study of man in motion. One of its applications is to aid athletes in their endeavour to improve their performance. In the more specific area of exercise physiology, kinanthropologists examine the physiological components relative to the sport, and through sport specific testing, help athletes improve their performance with efficient training programs.

One type of sports activity that the exercise physiologist is often confronted with is intermittent in nature. Badminton is one such intermittent sport in the sense that the athlete has repeated brief pauses between short bouts of exercise. This is in contrast to endurance or continuous sports. Mikkelsen (1979) found that elite badminton players rest for an average of 9 seconds and work for an average of 9 seconds. Reed (1981), from the analysis of the 1979 and 1981 Badminton World Championships, found that the players in men's singles rested for an average of 10 seconds and worked for an average of 10 seconds. Both studies concur in their obtained work:rest ratio of 1:1.

For this type of event the exercise physiologist must be able to measure both the intensity of effort and the recovery from work. Two of the relevant trainable physiological parameters that are often utilized in elite athlete testing are heart rate and blood lactate. Also, these two parameters are frequently employed in a complementary fashion in order to monitor both intensity of, and recovery from, work. Both are elevated during exercise and decrease during recovery.

Some research has been done in relation to lactate removal rates (Jorfeldt et al., 1978; Bonen and Belcastro, 1976; Hermansen and Stensvold, 1972) but most

studies have looked at lactate removal over a long period (20 minutes or more) (Bonen and Belcastro, 1976; Hermansen and Stensvold, 1972) and as mentionned by Evans and Cureton (1983), a study of the effects of conditionning on the peak rate of blood lactate removal is clearly needed. Measuring blood lactate during recovery, immediately following a sport-specific test, through the fairly new and fast fingertip blood sampling technique may give some insight into the relation between heart rate and lactate recovery in elite athletes.

A relationship betweeen heart rate and lactate has been found to exist (Thimm et al., 1984; Saltin et al., 1981; Tibes et al., 1976) during exercise. However, the relationship between heart rate and lactate during recovery is not as clear. Saltin et al. (1981) found that the concentration of lactic acid in the interstitial space remains elevated for several minutes and yet during this time heart rate was already decreasing. Conversely, Thimm et al. (1984) found that the increase in heart rate lasted for several minutes although the lactate concentration in perfusion outflow decreased during that time. The authors hypothesized that this prolonged effect on heart rate may occur because the lactate concentration in interstitial space remains relatively high due to slow back-diffusion from the interstitial space to the capillaries. However, it is also possible, as mentionned by Thimm et al. (1984), that the mechanisms which control the increase in heart rate are different from those which control its return to base-line. Some authors (Degre et Denolin, 1965; Margaria et al., 1933; Hill, 1925) also related the heart rate recovery with the O_2 recovery considering that the fast heart rate recovery phase (first two minutes of recovery) corresponds to the alactic O_2 debt and that the slower phase (2nd to the 5th minute) corresponds to the lactic O2 debt. It thus seems important to look at the relationship between heart rate and blood lactate during the early phase of recovery (first 5 minutes).

STATEMENT OF THE PROBLEM

The purpose of the study is to establish the relationship between heart rate and lactate responses (peak and recovery) to a sport specific field test (4-corner test) in elite male and female badminton players.

LIMITATIONS

The subjects tested were badminton players of an elite level. Thus, they were not randomly selected and caution is advised if the results are to be applied to other groups. Again, because of this specificity of subjects, the number of subjects available for testing was a limiting factor. The observed heart rate and lactate responses were produced in relation to a very badminton-specific test (4-corner test). These responses may be different following a different test protocol so discretion is again advised in this respect. Lactate recovery may be different at other intensity levels so the relation between lactate and heart rate recovery may also be different. The whole blood technique for assessing lactate concentration may not correspond exactly to other lactate assessing methods.

ABBREVIATIONS

A	Actin
AHTR	Alactic half-time recovery
CP	Creatine phosphate
CS	Citrate synthase
1,3 DPG	1,3 diphosphoglycerate
EMD	Time interval between the onset of electrical activity and that

	of force production
FAD	Flavine-adenine-dinucleotide
FDP	Fructose diphosphate
FOG	Fast twitch oxidative glycolytic muscle fibre (IIa)
FP	Flavin prosthetic group
F-1, 6-P_2	Fructose-1, 6 diphosphate
F-6-P	Fructose-6-phosphate
FT	Fast twitch glycolytic muscle fibre (IIb)
GP	Glycogen phosphorylase
G-1-P	Glucose-1-phosphate
G-2-P	Glucose-2-phosphate
G-3-P	Glucose-3-phosphate
GS	Glycogen synthase
GTP	Guanosine triphosphate
$HATP^{3-}$	Protonated form of ATP
HK	Hexokinase
HRR2	Heart rate after two minutes of recovery
LDH	Lactate dehydrogenase

M	Myosin
M*	High-energy form of myosin
NAD/NADH	Nicotinamide diphosphate oxidized/reduced
OAA	Oxaloacetate
OSM	Osmolality
PDH	Pyruvate dehydrogenase
PEP	Phosphoenol pyruvate
PFK	Phosphofructokinase
3-PGA	3-phosphoglycerate
PHR	Peak heart rate
Pi	Inorganic phosphate
PPi	Pyrophosphate
PWC170	Physical Work Capacity at HR = 170 bpm
RHR	Resting heart rate
SDH	Succinate dehydrogenase
ST	Slow twitch oxidative muscle fibre (Ia)
succCoA	Succinyl-coenzyme A
UTP	Uridine triphosphate

V_{int}	Interstitial volume
V_E	Ventilation
VL	Vastus lateralis
α-KG	Alpha-ketoglutarate

REVIEW OF LITERATURE

INTRODUCTION

The purpose of this chapter was to review the literature in relation to: 1) intensity of exercise responses, 2) regulation of heart rate during exercise and recovery, 3) heart rate as an indicator of exercise intensity and recovery, 4) regulation of blood lactic acid during exercise and recovery, 5) lactic acid as an indicator of exercise intensity and recovery, 6) relationship between lactic acid and heart rate, 7) the influence of exercise specificity on heart rate and lactic acid responses, 8) physiology of badminton performance.

INTENSITY OF EXERCISE RESPONSES

"The main objective of most organ functions is to maintain the internal equilibrium of the single cell in spite of primary changes or disturbances in the animal's internal or external environment, in accordance with the 'one for all and all for one' concept. A continuous exchange of materials between interstitial fluid and blood plasma is necessary for the normal function of the cell" (Astrand and Rodahl, 1986).

Skeletal muscle is unique in that it can vary its metabolic rate to a greater degree than any other tissue. In fact the working skeletal muscles may increase their oxidative processes to more than 50 times their resting levels. This situation may create serious problems for the working muscle cell, for while the consumption of fuel and O_2 increases 50-fold, the rate of removal of heat, carbon dioxide, water, and waste products must be increased similarly. The cells must continually be 'flushed' by fresh fluid in order to maintain their chemical and physical equilibrium. When muscles are required for vigorous exercise, the ability to maintain their internal equilibrium

necessary to continue the work is entirely dependent on those organs which service the muscle. The circulatory and respiratory organ functions basically strive to keep the muscle cell in indirect instantaneous contact with the surrounding air at all times (Astrand and Rodahl, 1986).

When a person goes from a resting state to a physically active state, many physiological changes occur: increases in heart rate, cardiac output, ventilation, oxygen consumption, systolic blood pressure and blood lactate concentration are some of these changes. All of these will vary in relation to the intensity of exercise. The more intense the exercise, the stronger the response. To understand these varying responses it is important to understand the interplay between the energy systems that furnish ATP which is the basic chemical energy source for contracting muscle. At the onset of exercise an increased need for ATP is created. If the muscle had to rely completely on the limited amount of ATP within its fibres, it would fatigue very quickly. For this reason the ATP must be resynthesized as quickly as it is broken down (Vander et al., 1985). The chemical pathways for regeneration of ATP represent three distinct processes: 1) Creatine Phosphate Splitting; 2) Anaerobic Glycolysis; and 3) Aerobic (Oxidative) Metabolism (Green, 1982).

Depending on the energy demands of the exercise, these three sources will contribute in different proportions to the resynthesis of ATP. For example, when someone walks or jogs, the ATP will be mostly resynthesized through the oxidative energy system whereas if the person runs a 100m sprint, the energy will be mostly derived from the ATP-CP system (Hirvonen et al., 1987). As the immediate end-products from the different energy producing pathways vary (CO_2 and H_2O for aerobic, lactic acid for anaerobic glycolysis), measuring these end-products during and following exercise informs the physiologist about the intensity of the work and the energy systems being utilized. If the work load is relatively light, the aerobic energy

system will support most of the ATP requirements and lactate concentration will remain relatively low since anaerobic glycolysis is not used at a high rate. If, however, the intensity of the workload goes up, the aerobic energy system may not have a fast enough turnover rate for ATP to resynthesize the ATP at the needed rate. Anaerobic glycolysis will then have to assist the aerobic system in furnishing the required ATP to enable the working muscles to sustain the increased workload. Lactate, being the inevitable end-product of anaerobic glycolysis, will be increased in its muscle concentration and subsequently its blood concentration. Thus, blood lactate concentration is a physiological parameter that is often used to monitor intensity of exercise. The more intense the exercise, the higher the production of lactate and the higher its blood concentration. The relation between blood lactate concentration and work intensity is curvilinear (Astrand and Rodahl, 1986).

Heart rate is another physiological parameter that is often used to monitor intensity of exercise. At rest the heart rate is kept down by parasympathetic action via the vagal nerve. At the onset of exercise, or even before, there is an inhibition of the parasympathetic activity and an increased sympathetic impulse traffic. The heart's inhibition is released and it starts to beat faster and with increased force. This increased heart rate, joined with other circulatory changes, helps increase the blood flow to the working muscles. The increased blood flow brings more oxygen to the working muscle while at the same time removing unwanted end-products (Astrand and Rodahl, 1986). In many types of submaximal work, a linear relation is found between increase in heart rate and increase in exercise intensity (Astrand and Rodahl, 1986).

REGULATION OF HEART RATE DURING EXERCISE AND RECOVERY

The sinoatrial (SA) node is a small mass of specialized myocardial cells embedded on the right atrial wall near the entrance of the superior vena cava. The SA

node is also the normal pacemaker for the entire heart. Its rhythmic discharge occurs spontaneously in the absence of nervous or hormonal influences. The inherent autonomous discharge rate of the SA node is approximately 100 beats per minute. However, the SA node is under the constant influence of both nerves and hormones. A large number of parasympathetic and sympathetic fibres terminate on the SA node as well as other areas of the conducting system. The stimulation of the parasympathetic (vagus) nerve causes the slowing down of the heart through local application of acetylcholine. The sympathetic nerves cause just the reverse; their stimulation increases the heart rate through local application of norepinephrine. The sympathetic increases the slope of the pacemaker potential which causes the cell to reach threshold more rapidly and is responsible for the increase in heart rate. The parasympathetic stimulation has the opposite effect, it reduces the slope of the pacemaker potential, threshold is reached more slowly and the heart rate is decreased (Vander et al., 1985). Vander et al. (1985) also state other factors than cardiac nerves that can alter heart rate: epinephrine (liberated by the adrenal medulla, speeds the heart), temperature, plasma electrolyte concentrations, and hormones other than epinephrine. These, however, are generally of lesser importance, and the heart rate is primarily regulated very precisely through the balancing of the parasympathetic and sympathetic autonomic systems, both operating on the SA node. For example, it is known that the heart rate acceleration at the initiation of a muscle contraction is elicited predominantly through a withdrawal of vagal tone (Freyschuss, 1970). However, several sources of afferent information to the cardiovascular system that makes its responses proportional to its functional capacity have been identified.

Some evidence shows that central command (Freyschuss, 1970; Goodwin et al., 1971; Rowell, 1980) and humoral drives (Tibes, 1977; Ruddel et al., 1986; Nielsen et al., 1984) may contribute to exercise response of the cardiovascular and respiratory

systems. However, most of the recent studies point toward the importance of reflexes originating in the muscle (Rowell, 1980). Many factors have been proposed as being able to initiate the reflexes:

1) mechanical : (Kao et al., 1967; Kalia et al., 1972; Hodgson and Matthews, 1967; Kumazawa and Mizumura, 1977; Kniffki et al., 1981),

2) K^+ concentration : (Kumazawa and Mizumura, 1976;Kumazawa and Mizumura, 1977; Tibes et al., 1978; Tibes et al., 1976; Saltin et al., 1981; Hemmer and Tibes, 1977; Tibes and Groth, 1977; Baum et al., 1988; Tibes et al., 1977; Rybicki et al., 1984),

3) H^+ concentration : (Thimm and Meier zu Verl, 1984; Thimm et al., 1984a; Tibes and Groth, 1977; Tibes et al., 1976; Tibes et al., 1978),

4) osmolality : (Tibes and Groth, 1977; Laser et al., 1960; Tibes and Hemmer, 1973; Tibes et al., 1978),

5) lactate concentration : (Tibes and Groth, 1977; Thimm and Meier zu Verl, 1984; Thimm et al., 1984; Tibes et al., 1978; Tibes et al., 1976),

6) Pi concentration : (Tibes and Groth, 1977; Tibes et al., 1978; Tibes et al., 1976),

7) thermal stimuli : (Kumazawa and Mizumura, 1976; Kimazawa and Mizumura, 1977; Kniffki et al., 1981),

or a combination of these parameters. The majority of investigators favour chemical rather than mechanical stimuli as the major sources for the cardiorespiratory drives. However, some controversy still remains about the major candidate. Jose et al. (1970) have shown that intrinsic rate of the normal human heart with sympathetic and

parasympathetic blockade rises 7.1 beats/min per 1 C rise in right atrial temperature. Thus, as explained by Rowell (1974), the much greater increases in HR observed during prolonged moderate exercise cannot be attributed solely to the small rise in body temperature noted by Ekelund et al. (1967). The muscle spindle also did not produce any appreciable stimulation of respiration or HR in the decerebrate cat (Hodgson and Matthews, 1968). Other work has also shown that the circulatory and respiratory reflex responses of muscular origin are preferentially or exclusively mediated by group III (small myelinated fibres) and group IV (unmyelinated fibres) afferents (McCloskey and Mitchell, 1972; Kumazawa and Mizumura, 1976; Tibes, 1977; Hnik et al., 1969; Kniffki et al., 1981) and that their receptors are situated in the interstitial space of the muscle (Baum et al., 1988; Rybicki et al., 1984). Group I and II afferent fibres do not seem to respond to the above stimuli (McCloskey and Mitchell, 1972; Tibes, 1977; Kniffki et al., 1981; Tibes et al., 1978). Liu et al. (1969) found that the K^+-evoked nerve impulses from the muscle are transmitted through the afferent fibres of the somatic nerve to the medulla, stimulating the respiratory and vasomotor centers simultaneously. The circulatory responses after intra-arterial injection of KCl are mediated through the release of catecholamines at the sympathetic nerve endings of the heart and blood vessels. This seems to be supported by Mitchell et al. (1977) who found that the cardiovascular changes are mediated in part by the activation of beta-adrenergic receptors in the heart. They found that heart rate and blood pressure seem to be correlated with catecholamine concentration during submaximal exercise. Nielsen et al. (1984) found that during prolonged exercise, an upward drift of the HR occurs after the first 10 minutes of exercise and this drift was positively correlated to the measured increments in plasma catecholamine concentration, which continued to increase as exercise progressed. Polymodal C-fibre (i.e. group IV fibres) receptors were also found to exist (Rowell, 1980; Kumazawa and

Mizumura, 1977; Kumazawa and Mizumura, 1976; Tibes et al., 1978; Kniffki et al., 1981). The same receptor responds to many stimuli including chemical, mechanical and thermal, though some of these receptors responded more strongly to one of these given stimuli. Kniffki et al. (1981) classify the receptors into two groups: 1) the nociceptors (responsible for the reception of noxious chemicals, mechanical, and thermal stimulis) and 2) the ergoreceptors (responsive to light stretch, contractions, and light local touching). A great specialization seems to exist in these group III and IV afferents. Preliminary results obtained on the innervation of the Achilles tendon have already revealed three characteristic terminal structures of group III afferents and another four types of group IV ones, both in respect to their location and the ultrastructure of the endings and their surrounding tissues (Andres et al., 1980). Some afferent receptors also vary in relation to their activation threshold (low or high threshold). As mentioned by Kniffki et al. (1981), it is difficult to imagine that uniform receptive properties go along with such amazingly great structural diversity.

The V_{int} (interstitial volume) will affect the HR response especially at the beginning of work (30-60 sec) since the shrinking of the V_{int} occurs mainly during that period (Baker and Davis, 1974; Baum et al., 1988). Even a small increase in the V_{int} probably leads to a strong attenuation of the K^+ response. This concurs well with the findings of Tibes et al. (1977) and Tibes and Hemmer (1973) which show K^+ concentration being related to the initial cardiovascular and respiration functions when passing from rest to exercise and that the appropriate K^+ concentration for the activation of these reflexes is that which was observed in the interstitial space of working muscles.

Reflexes from working muscles are now known to affect the cardiovascular and respiratory systems. As mentioned previously, some evidence indicates that central command may contribute to exercise response (Goodwin et al., 1971; Freyschuss,

1970; Rowell, 1980). Freyschuss' (1970) data suggest a contribution that may be in the order of one-half of the response whereas Goodwin et al. (1971), after showing a possible effect from the central command in 1971, qualified it to a much smaller contribution in 1972. Vagal tone has been shown to increase with dynamic endurance training whereas sympathetic activity decreases at identical work loads (Lehmann and Keul, 1986). Lehmann and Keul (1986) found that dynamically and statically trained subjects exhibit a similar training-related control of sympathetic activity. An increased vagal tone, however, occurred in conjunction with only dynamic endurance training.

A training dependent adaptation of the autonomic system can be seen after approximately 4 weeks of intensive endurance training (Winder, 1978). In 1981, Lehmann et al. found that during a graduated exercise lactate and catecholamines show a constant relationship to each other which becomes less exact during higher exercise intensities. An increase in lactate could also be seen during the recovery phase up to the 3rd or 6th min whereas the catecholamines exhibited an earlier decrease; this points to a correlation that is not as close as during the exercise stage. The authors suggest that the differing changes in the lactate and catecholamines during the recovery stage could have circulatory causes or could be the result of differing rates of disappearance.

Tibes and Hemmer (1974) and Tibes (1977) have estimated that peripheral drive from the muscle B- or C-fibres to the centers accounts for the major part of the whole drive on the heart, systemic circulation and ventilation at onset and during steady state of work

Humoral drives also effect cardiovascular responses (Tibes, 1977; Ruddel et al., 1986; Nielsen et al., 1984). Tibes (1977) estimated that the humoral drives accounted for a minor portion of the total cardiovascular and respiratory responses during onset as well as during steady state of work (less than 21%). However,

humoral drives became more effective with increasing duration of work (up to 30% of total drive).

The question asked by Rowell (1974) about the sources of afferent information that make the cardiovascular system respond proportionaly to its functional capacity was probably best answered by himself in a 1980 paper when he states that;

"Multiple, redundant regulatory systems serve to modulate the circulatory system. Acting through cortical and spinal cord motor systems, 'central command' could serve to set basic patterns of effector activity, which in turn are modulated by baroreceptors, muscle mechanoreceptors, and muscle chemoreceptors, as error signals may develop. Under normal conditions of exercise, or perhaps during only mild exercise, a muscle chemoreflex may not be activated. But circulatory delivery of oxygen to working muscle may be so closely matched to muscle oxygen consumption that even a small decrease in muscle blood flow might reduce intramuscular PO_2 below a critical level and cause a reduction in oxygen uptake. The resultant mismatch between oxygen delivery and utilization could then generate a metabolic error signal (i.e., build-up of metabolites in muscle) that is sensed by chemosensitive nerves in muscle. This 'chemoreflex' would correct the imbalance within the limits permitted by a normally sensitive baroreflex. As the severity of exercise is increased toward maximal, the margin for any "flow error" is probably extremely small and may even disappear so that a persistent metabolic error signal acts in concert with the other regulatory systems. In short, the interaction of multiple systems in the exercise response may so far have masked the important role of any given system. We may only see evidence for the participation of a system when all others are inoperative or when extreme conditions such as muscle ischemia call forth a dominant contribution from one system."

As concluded by Ruddel et al. (1986) the cardiovascular response, even during submaximal exercise, appears to be related to complex interactions of hormonal changes and electrolytes but further investigation on the matter is needed.

More details are given later about the interrelation between lactate and HR (see section 'Interrelationship Between Heart Rate and Lactic Acid').

HEART RATE AS AN INDICATOR OF EXERCISE INTENSITY AND RECOVERY

Heart rate is often used in the testing of athletes to monitor the intensity of the work as well as the recovery from this work (Koutedakis and Sharp, 1985; Pilardeau et al., 1982; Degre and Denolin, 1965; Morand et al., 1978; Pilardeau et al., 1983) As mentioned earlier, the heart rate increases in a relatively linear fashion with work intensity (VO_2), thus making it often a good physiological parameter to monitor the intensity of work. Degre and Denolin (1965) stated that the HR recovery curve was composed of two parts, an initial rapid phase followed by a second slower and much longer one. Johnson et al. (1942, in Degre and Denolin) examined especially this fast phase of recovery (1', 2', and 3'). They found that a linear relation existed between the average HR recovery during those three minutes and the total of the HRs added from the whole effort. Thus, the more intense the work, the higher the average recovery HR. However, for a given workload, the fitter the individual, the lower the average HR. Another recovery assessing method using the rapid HR recovery phase is called the alactic half-time recovery. This method involves analysing the HR recovery curve in a semi-logarithmic graph. After this, two linear functions appear from which a 'half-time' constant can be calculated. This half-time constant is supposed to represent the necessary time to recover half of the oxygen debt developped through the anaerobic energy utilization during the work (Margaria et al., 1933,and Hill, 1925, in Degre and Denolin, 1965). These authors related the HR recovery with the O_2

recovery considering that the fast HR recovery phase corresponds to the alactic O_2 debt and that the slower phase corresponds to the lactic O_2 debt.

Degre and Denolin (1965) compared the alactic half-time recovery method with the average HR method to see which of these two methods is better to estimate the fitness of an individual. The average HR method was shown to be more precise than the alactic half-time recovery method but the average HR method was shown to be much less precise in the fitness evaluation than the HR measured during work ($r = -0.70$) (this was in relation to normal subjects). The correlation between the alactic half-time recovery and the PWC 170 was equal to -0.40.

Later, in an attempt to simplify the methods to monitor the training of an athlete, Morand et al. (1978) studied HR responses during graded exercise on a bicycle ergometer and during recovery. They used again the notion of the alactic half-time recovery. They stated that the better an athlete's alactic half-time recovery is, the better is his cardiovascular adaptation to work (no correlation is given). Also, in order to simplify the long calculation process of the AHTR (alactic half-time recovery), they correlated this method with the instant HR after 2 minutes of recovery (HRR2) divided by peak HR (PHR), both less the resting HR (RHR) : (HRR2-RHR)/(PHR-RHR). The correlation ($r = 0.84$) was statistically significant ($p<0.001$). They concluded that the HRR2 method is a simple and correct way of monitoring the fitness level of an athlete and the efficiency of the training (as opposed to measuring VO_2 and VCO_2 which necessitate relatively restrictive and expensive equipment).

Pilardeau et al. (1982) again used the idea of the alactic half-time recovery. However, to simplify the calculations, they adopted another way of calculating the recovery. They used the slope of the plot during the first minute of recovery and the HR after 5 min. of recovery. In this method, they found the intersection of the 1 minute recovery plot with the resting HR axis. The HR recovery is measured

following a sport specific test. Protocols for different sports were given. A table comparing the fitness evaluation of football players (soccer) in relation to the HR recovery method and two other methods (the systolic tension time or S.T.T. and a $VO_{2\ max}$ test) is also shown. However no statistical correlation is given. In 1983, Pilardeau et al. published results on county level swimmers where they used their method of HR recovery to monitor training and fitness throughout a year. A guide to analysing the HR recovery curve to relate the results to the kind of training needed was given. The swimmers' results showed that they improved throughout the year. The HR recovery method seems good enough to classify athletes of this level into different categories of physical fitness : very poor, mediocre, average, good, very good and excellent. As mentioned by the authors, the interest of this testing method lies in its repetition possibility and the ease with which it can be applied under effort conditions. However, more work needs to be done in relation to its validity and its accuracy if it is to be used with national or international level athletes since the accuracy of the alactic half-time recovery, on which this method is based, has been shown by Degre and Denolin (1965) not to be highly correlated to results from the PWC 170 test (-0.40). At the present, it can be said that the HR recovery curve gives some information in relation to the fitness level of the lower level athlete and that it can help to monitor fitness and training.

Rowell et al. (1966) found that the lactate-oxygen debt relationship during exercise is time dependent while oxygen debt is not. However, Brooks and Gaesser (1980) obtained results indicating that the removal of lactate and the slow phase of the postexercise VO_2 are not related in time. This goes against the classical thinking of the 'O_2 debt' theory, where this slow phase of postexercise VO_2 should be causally related to the removal of lactic acid (i.e. the 'lactacid debt') (Margaria et al., 1933 in Degre and Denolin, 1965; Rowell et al., 1966). Brooks and Gaesser (1980) reported a

temporal relationship between the decline in VO_2 and the tissue temperature in rats after exhaustive exercise and concluded that a major portion of the postexercise VO_2 is a consequence of the well known effect of temperature on the rates of biochemical reactions. Brooks and Gaesser (1980) concluded from their work that the previous hypotheses regarding lactic acid metabolism after exercise were too simplistic and that the term O_2 debt is archaic and should not be used. This does seem to affect the validity of the relation between the slow phase of HR recovery and lactate recovery since this relationship is based upon the questioned relationship between lactate removal and the slow phase of postexercise VO_2 recovery.

Thus, it seems that HR recovery gives us some information in relation to the fitness level of the athlete but it may not be linked to lactate recovery since the relation in time between lactate recovery and the slow phase of O_2 recovery (previously linked to the lactic O_2 debt) is in doubt.

REGULATION OF BLOOD LACTATE DURING EXERCISE AND RECOVERY

In this section, knowledge about ATP production in the muscle, lactate production, lactate in blood, relation between muscle fibre types and lactate, and lactate removal will be presented.

ATP production in muscle.

As previously mentioned, ATP is the energy source for contracting muscles. As explained by McGrail et al. (1977), without replenishment of the ATP used at the contractile site, muscle is capable of only a few contractions (<1 sec). Intramuscular stored creatine phosphate (CP) is the most immediate contributor to the resynthesis of ATP. In the presence of the enzyme creatine kinase, CP and ADP yield ATP and creatine. However CP is in limited supply and diminishes severely after 10-15 seconds of activity. Continuing intense activity is then supported by a larger energy pool:

glycogen. This substrate is synthesised from glucose and is stored in the liver and muscle. During exercise, glycolysis results in the release of ATP from specific reactions (i.e. 1,3 DPG+ADP --> 3 PGA+ATP and PEP+ADP -->Pyruvate+ATP). Glucose can be metabolised, via glycolysis, from glucose-6-phosphate without having been stored as glycogen. The relative contribution of glycogen and glucose to the support of muscular activity varies with the nature and duration of the work being performed , and is considerably affected by the hormonal milieu during exercise. Another major contributor to the ongoing supply of ATP is intramuscular triglycerides and the triglycerides which are stored as body fat. In adipocytes, in the presence of the enzyme lipase, triglycerides are 'split' and fatty acids are therefore liberated and transported, via the blood, to the muscle. The acetyl-CoA produced by the degradation of the fatty acids combines with carnitine to form acyl-carnitine, which is transported across the mitochondrial membrane via a carrier, carnitine-acyl-transferase. Once inside the mitochondria acetyl Co-A is once more formed and then oxidized in the Krebs cycle to ATP. Intramuscular triglycerides provide energy in much the same manner.

It is from these sources (CP, glucose, glycogen, and free fatty acids) that ATP is derived for muscular contraction. Protein catabolism is thought to be of minimal significance when compared to the energy supply of the other fuels (McGrail et al., 1977).

Lactate Production.

Glycogen can be metabolized in two ways, aerobically (in the presence of O_2) or anaerobically (in the absence of O_2). Regardless of which route it follows, the glycolitic pathway is common to both processes. In glycolysis, glucose is reduced to 2 pyruvate molecules which results in the net production of 2 ATP molecules. This process, however, is dependent on the supply of NAD^+ (nicotinamide-adenine

dinucleotide). Once the supply is depleted, the conversion of G-3-P to 1,3-DPG would be prevented thus inhibiting further chemical reactions. It is therefore necessary for NADH to be oxidized to NAD^+ elsewhere in order to again become available to support the conversion of G-3-P to 1,3-DPG (McGrail et al., 1977). It seems that in the presence of oxygen (aerobic metabolism) pyruvate is transferred from the cytoplasm into the mitochondria, converted into acetyl-CoA via the lipoic acid cycle, and completely oxidized in the Krebs cycle. When metabolised in this way, and using the malate-aspartate shuttle, pyruvate yields an additional 36 molecules of ATP demonstrating the large energy potential of carbohydrates when metabolised oxidatively (34 additional molecules of ATP when the glycerol phosphate shuttle is used instead of the malate-aspartate shuttle). At several points along this pathway, the reformation of ATP from ADP occurs, resulting in the transfer of hydrogen ions to O_2, forming H_2O, and oxidizing NAD to NAD^+. The operation of the Krebs cycle and the electron transport chain facilitates the regeneration of NAD^+ via the malate-aspartate shuttle which operates across the mitochondrial membrane. Hence, the NAD^+ becomes available to support the G-3-P —> 1,3-DPG reaction in the sarcoplasm. Under some circumstances, as in the onset of exercise or during periods of strenuous exercise, the NAD^+ demands of glycolysis surpass the amount that can be regenerated via the malate shuttle, thus necessitating the oxidation of NADH elsewhere. In the presence of the enzyme lactic dehydrohenase (LDH), additional NAD^+ is made by means of the following reaction in which pyruvate is reduced to lactic acid:

$$\text{PYRUVATE} + \text{NADH} \longrightarrow \text{LACTATE} + NAD^+$$

The production of lactate is thought to be accelerated during exercise by a combination of increased concentrations of NADH and pyruvate, as well as an

increase in LDH activity (McGrail et al., 1977). However, Hermansen (1971) found that the two-fold increase of pyruvate shown by Karlsson (1971) seems to explain only a fraction of the total lactate produced, even in submaximal exercise. Thus the concentration of NAD^+ may be the single most important factor involved in regulating the production of lactate (McGrail et al., 1977). Therefore it would seem important to establish the effect of physical training on the enzyme levels of the NADH shuttle? Schantz (1986) found that with endurance training, the malate-aspartate shuttle enzyme levels can increase while no alteration is seen in the alpha-glycerophosphate shuttle enzymes. Type I fibres are, compared to type II fibres, characterized by higher levels of malate-aspartate shuttle enzymes but lower levels probably contribute to the shift from anaerobic to aerobic glycolysis at submaximal exercise intensities. Increased malate-aspartate shuttle enzyme levels can also facilitate oxidation of lactate into CO_2 and H_2O, a pathway possibly utilized at low exercise rates following a burst of high intensity exercise.

Numerous factors influence glycolysis in skeletal muscle. The conversion of glycosyl residues to G-1-P catalyzed by the phosphorylase system is activated by the contraction stimulus, possibly by liberation of Ca^{++}, by anoxia and by cyclic 3'.5' AMP, the intracellular messenger of increased sympathetic activity. Only small amounts of extracellular glucose are transferred to G-6-P, since hexokinase activity in skeletal muscle is small (Hirche et al., 1973) and because hexokinase is inhibited by G-6-P which is synthesized very rapidly from glycogen. Another rate controlling step in glycolysis is the conversion of F6P to FDP which is catalyzed by PFK. PFK is activated by high concentrations of ADP, 5' AMP, Pi, F-6-P, FDP, and cyclic 3'.5' AMP. The majority of factors mentioned here are known to activate the Embden-Meyerhof pathway of glycogenolysis considerably in FT muscle fibres during severe exercise. PFK is strongly inhibited by high concentrations of CP, ATP, H^+ and

citrate. Citrate easily leaves the mitochondria, thus adapting the rate of glycolysis to the rate of respiration. As mentioned previously, during glycolysis pyruvate is reduced to lactic acid while NADH is oxidized to NAD. This reaction is catalyzed by LDH. The balance within the cell is far to the side of the reduced partner, so that LA/pyruvate ratios attain values of more than 10 in muscle cells (Hirche et al., 1973).

Although extramitochondrial NADH itself cannot penetrate the mitochondrial membrane, electrons derived from it can enter the mitochondrial electron-transport chain by indirect routes called shuttles (Lehninger, 1982). In FT muscle fibres the most important is the glycerol phosphate shuttle (Hirche et al., 1973), in which the cytoplasmic NADH first reacts with cytoplasmic dihydroxyacetone phosphate, an intermediate of glycolysis, to form L-glycerol 3-phosphate in a reaction catalyzed by the cytoplasmic NAD-linked glycerol phosphate dehydrogenase:

Dihydroxyacetone phosphate + NADH + H^+ <—> L-glycerol 3-phosphate + NAD^+

Glycerol 3-phosphate can penetrate mitochondrial membranes very rapidly. Within the mitochondria another glycerol 3-phosphate dehydrogenase oxidizes glycerol 3-phosphate back to dihydroxyacetone phosphate, and its flavin prosthetic group (FP) becomes reduced:

Glycerol 3-phosphate + FP —> Dihydroxyacetone phosphate + FPH2

Under aerobic conditions the cytoplasmic NADH formed by triose oxidation is reoxidized, not by pyruvate, but by the shuttle system and the respiratory chain. Both have a much higher affinity for NADH than LDH and therefore win in the competition for cytoplasmic NADH. For this reason pyruvate is normally not reduced to LA during respiration but is oxidized directly to acetyl-coenzyme-A and CO_2. Sahlin et al. (1976) found that even after exhaustive exercise of 5-10 min. which resulted in high

muscle lactate content, pyruvate content was only slightly higher than at rest. During recovery lactate content of muscle decreased exponentially in respect to time, whereas pyruvate content increased.

Although FT muscle fibres have a lower cellular content of mitochondria, these mitochondria contain a specifically higher activity of glycerolphosphate dehydrogenase. With regard to the function of this enzyme in the transfer of extramitochondrial H^+ to the respiratory chain, this higher glycerolphosphate dehydrogenase activity was interpreted in terms of a 'compensation phenomena' which may help to prevent muscle metabolism from aerobic glycolysis. Sahlin et al. (1976) found that NADH/NAD was highly increased after exercise and that it had not returned to the basal value after 20 minutes of recovery.

A hormonal mechanism also seems to play a role in lactate production. During exercise, plasma epinephrine concentration rises. In the dog, plasma epinephrine is the only hormone known to increase the rate of glycogenolysis in skeletal muscle. The effect could be described as beta2-stimulation, via beta2-receptors in the muscle, and could be blocked by beta-blockers such as propranolol (Issekutz, 1984). Issekutz (1984) found that beta-blockade had two striking effects on carbohydrate metabolism in exercising dogs: inhibition of lactate production and increase of glucose turnover. The inhibition of lactate production was much more profound when the blocker was infused in the steadily running dog than when it was administered in a single injection prior to exercise. The explanation given by the author for this difference is that the course of propranolol concentration in the plasma and in the fact that there is a competitive antagonism between epinephrine and the beta-blocker. Issekutz (1984), concluded that the rate of glycogenolysis is under a dual control. There is an intracellular metabolic control (Pi accumulation and increased Ca^{++} concentration which was shown to activate phosphorylase b) and there is a hormonal control that involves epinephrine

and the beta2-receptors in the muscle. Beta-blockade inhibits the exercise-induced muscle glycogenolysis.

The relatively large ATP potential of pyruvate, when oxidized aerobically, is not lost as a result of its anaerobic conversion to lactate. The pyruvate-lactate reaction is reversible in the presence of LDH-H, and thus, under some conditions, lactate may serve as a significant source of energy (McGrail et al., 1977).

Lactate in blood.

Lactic acid has a pK value of 3.86 and is therefore almost completely ionized in the body. Undissociated lactate is lipid insoluble. Lactic acid therefore can permeate across cell membranes only in an ionized form (lactate) and thus the permeation rate of the lactate anion as well as the H^+ ion can become rate limiting (Hirche et al., 1973). The lactate produced during anaerobic glycolysis diffuses from the muscle and accumulates in the blood, where, if measured , it can serve as an indicator of the extent to which anaerobic processes are activated during a workout. Blood lactate concentrations can exceed 180 mg/100 ml as a result of supramaximal continuous exercise, and 270 mg/100 ml after supramaximal, intermittent exercise (McGrail et al., 1977). Jorfeldt et al. (1978) measured the release of lactate in relation to the muscle lactate concentration. The release of lactate rose approximately linearly with the muscle lactate concentration up to about 4-5 mmol/min but then the relationship clearly levelled off. Their results indicate a maximal level for the lactate release from the exercising muscles with a translocation hindrance for lactate within the muscle. Peak blood lactate levels may become evident anywhere from 1-10 minutes after the cessation of activity (McGrail et al., 1977).

It has been demonstrated that lactate is produced during aerobic submaximal exercise. It has been reported that exercise greater than 82% of VO_2 max, which lasts 30 min. in duration, elicits a continuous accumulation of lactate in the blood

(Nagle et al., 1970). During moderate exercise (60-80% $VO_{2\ max}$), lactate concentrations in the blood usually rise for 10-15 minutes, depending on the severity of the work, to a value of approximately 18-50 mg/100 ml. Thereafter, lactate concentration reaches a plateau or begin to decrease slowly. It is at this time that the oxidative contribution may be thought of as having caught up to the total energy demand and lactate removal surpasses lactate production. Light to moderate work (less than 60% $VO_{2\ max}$), is considered to be aerobic in nature as it can be supported almost entirely by oxidative processes, and thus little or no lactate accumulation occurs (McGrail et al., 1977). Hermansen and Stensvold (1972) stated from their results that a production of lactate is possible even with no increase in blood lactate since human skeletal muscle possesses a pronounced capacity to oxidize lactate.

At equivalent, absolute levels of O_2 uptake (L/min), blood lactate concentrations are greater during arm work when compared to leg work. Similarly, blood lactate concentration are higher during bicycle ergometer exercise than during treadmill running. This can be explained by the notion that, for a given work load, as the muscle mass decreases the anaerobic contribution to the total work output must increase.

In 1971, Hermansen (in McGrail, 1977) had reported that during work periods of varying duration (20 sec - 10 mins), similar blood lactate levels (X=149 mg/100ml) were seen at exhaustion. Lactate levels were considerably lower (X=73 mg/100ml) at the point of exhaustion during exercise bouts of extended duration (16 mins). This led to the suggestion that, if lactate levels limited short-duration exercise (<10 mins), then other factors limited extended exercise.

An increase in the muscle and blood lactate concentration leads to a decrease in pH, which may interfere with the enzymatic activity of several glycolytic enzymes and subsequently decrease the glycolytically derived supply of ATP within the muscle

(Karlsson et al. 1974; Keul et al. 1972). There is also a possibility that an increased hydrogen ion concentration within the muscle directly affects the actin-myosin interaction with the contractile process (Katz , 1970) Therefore, while lowered blood pH has been shown to be well tolerated during maximal exercise , the more severe decrease in intramuscular pH may serve as a limiting function in exercise. In 1985, Spriet et al. examined the metabolism and performance of a perfused rat hindquarter preparation during heavy exercise in three conditions: control (C), metabolic acidosis (MA, decreased HCO_3 concentration), and respiratory acidosis (RA, increased CO_2 tension). Initial isometric tensions were similar in all conditions, but the rate of tension decay was largest in acidosis. Lactate release was greatest in C, least in MA, intermediate in RA. This was in accordance with the finding of Hirche et al. (1973) that showed that during metabolic alkalosis, induced by infusion of bicarbonate, LA output was much higher than during metabolic acidosis, caused by the infusion of hydrochloric acid. During alkalosis permeation rates between 3-4 umoles/g/min were reached while during acidosis values of 1 umole/g/min were not exceeded. This observation supports the view that muscle cell membrane is the main barrier to LA diffusion out of muscle tissue (Hirche et al, 1973). Similar results were obtained by Mainwood et al. (1972) who found that lactate efflux from frog sartorius muscles stimulated to fatigue was impaired at low external bicarbonate concentration (1 mmol/l). Sahlin et al. (1978) found that after exhaustion, the intracellular pH and bicarbonate concentration of human muscle had decreased. Intracellular pH decreased from the normal value at rest of 7.00 to about 6.4 after exercise. HCO_3^- concentration decreased from 10.2 + 1.2 mmol/l at rest to about 3 mmol/l after exercise. Later, Sahlin and Henriksson (1984) obtained results that indicated that skeletal muscle buffer capacity can be increased in man by anaerobic training. Spriet et al. (1985) also found that acidosis resulted in less muscle glycogen utilization and

lactate accumulation than during control. Muscle CP utilization and ATP levels were unaffected by acidosis. Acidosis decreased the muscle's ability to generate isometric tension and depressed both aerobic and anaerobic metabolism. During stimulation in this model, lactate left the muscle mainly as a function of the production rate, although a low plasma bicarbonate concentration at pH 7.15 depressed muscle lactate release.

Many researchers have tried to explain muscle fatigue and, what is more relevant to this study, tried to link it at least in part to lactic acid accumulation in muscle (Sahlin et al., 1981; Bolitho-Donaldson and Hermansen, 1978; Tesch, 1980; Donaldson in Knuttgen, 1983; Sahlin in Knuttgen, 1983; Belcastro et al. in Knuttgen, 1983; Renaud and Mainwood, 1985; Taylor et al., 1986).

Capillary and blood pH decreased during maximal work of short duration, mainly due to lactic acid formation (Hermansen and Osnes, 1972; Karlsson and Saltin, 1970). Resting values for capillary blood and muscle pH were 7.42 and 6.93, respectively. Capillary blood pH decreased to 7.11 and 6.94 after continuous and after intermittent maximal exercise, respectively. The muscle pH decreased to about 6.40 in both intermittent and continuous exercise. The authors suggested that the pH of the muscle might be a limiting factor during maximal exercise of short duration. Sahlin et al. (1976) found similar results in relation to muscle pH (7.08 at rest to 6.60 at exhaustion). The difference in absolute values with the results of Hermansen and Osnes (1972), as explained by Sahlin et al. (1976), is most probably due to the differences in analytical technique. Hermansen and Osnes (1972), based on work from Katz (1970) with cardiac muscle, suggested that an increased hydrogen ion concentration reduces the binding capacity for Ca^{++} through an inactivation of the fibrillar protein, troponin, which as previously explained would inhibit muscle contraction. In another study, Nakamura and Schwartz (1972) found that a decrease in pH increases the Ca^{++} binding capacity of the sarcoplacmic reticulum. In 1976,

Fitts and Holloszy examined the relationship between lactic acid concentration and twitch tension in electrically stimulated frog sartorius muscle. In muscles stimulated under anaerobic conditions, contractile force decreased to 36% of the initial value in 15 minutes. The correlation between the increase in lactate and the decrease in contractile force was significant ($r = -0.99$, $P < 0.000001$). The recovery occurred in two phases. A rapid increase in contractile force, which represented 20% of the total recovery, took place during the first 15 seconds and occurred concomitantly with an increase in ATP from 3.9 to 4.6 umol/g. Lactate concentration did not change significantly during this period. The second phase of recovery of the contractile force was complete in 50 min. Lactate concentration and contractile force were significantly correlated during recovery ($r = -0.92$, $P < 0.00001$). However, the recovery of contractile force lagged behind the decrease in lactate. Their conclusion is that, through the interference of Ca^{++} binding to troponin, increased Ca^{++} binding to the sarcoplasmic reticulum and, as shown by Trivedi and Danforth (1966) and as mentioned previously, an inhibition of phosphofructokinase activity, the lowered muscle pH interfered with contractile function. However, they strongly indicate that the relationship is not a simple one in which a given concentration of lactate is associated with a fixed reduction in contractile force and that more work is needed to understand the different mechanisms with which lactate can influence muscle contraction. Fabiato and Fabiato (1978) obtained results supporting the results of Nakamaru and Schwartz (1970). They found that a decrease of pH decreased the frequency of the cyclic contractions and that acidosis decreases the rate of Ca^{++} release by the sarcoplasmic reticulum in the cardiac muscle. Gonzalez-Serratos et al. (1974) provided evidence that a decrease of 0.70 units of the intracellular pH in frog skeletal muscle resulted in a depression of the Ca^{++} transport by the sarcoplasmic r ticulum. Bolitho-Donaldson and Hermansen (1978) found a depressed maximum

tension in soleus, cardiac, and adductor magnus muscles of rabbits at a pH of 6.5 in relation to a starting pH of 7.0. Nassar-Gentina et al. (1978) examined fatigue in single fibres of the frog simitendinosus muscle. Two kinds of fatigue were produced in relation to stimulus duration. A rapidly reversed fatigue occurred with stimulation for under 40 s and was evidenced by a decline in tetanic tension that could be overcome by 1 s of rest. A prolonged fatigue was caused by stimulation for 100-150 s. Fiber phosphocreatine (PCr) fell from a mean of 121 +/- 8 nmol/mg protein to 10% of this value after 150 s of stimulation. Stimulation for 150 s did not significantly affect fibre glycogen and reduced fibre ATP by at most 15%. It is suggested that the prolonged fatigue was due to an accumulation of H^+ in fatigued fibres so as to interfere with the action of Ca^{++} in the coupling process. Sahlin et al. (1981) found that in the rat extensor digitorum longus muscle, tension decline is closely related to the decrease in muscle pH and increase in ADP but not to ATP content per se and that slowing of relaxation rate is closely related to decrease in muscle pH but not to muscle content of ATP or creatine phosphate. Fitts and Holloszy (1976) reported a correlation coefficient of 0.82 between contractile force and ATP concentration. It was suggested that the decrement in force production with fatigue may be due to a limited availability of ATP to myofibrillar ATPase enzyme. It was found that the Ca^{++} transport ability of fast-twitch muscle from exhausted animals is altered (Belcastro et al., 1983). Belcastro et al. (1983) later found that exhaustive exercise actually results in an elevated myofibril ATPase activity which is related to an enhanced Ca^{++} sensitivity and no alteration in the potential for rate of cross-bridge cycling.

In his 1983 paper Sahlin focuses on the effect of acidosis on production of ATP and on the generation of force. Both ATP and ADP will have their ionic composition changed by H^+ion accumulation. The protonated form of ATP ($HATP^{3-}$) is known to be a very potent inhibitor of PFK. The large relative increase in $HATP^{3-}$ during

acidosis will form the basis of the extreme pH sensitivity of PFK. Inorganic phosphate and glycogen are substrates for phosphorylase. It has been shown by Kavinski and Meyer (1977) for rabbit muscle and by Chasiotis for human muscle (personal communication with Sahlin, 1983) that HPO_2^{2-} is the true substrate for phosphorylase. A decrease in pH from 7.0 to 6.5 transfers about 50% of the available substrate (HPO_2^{2-}) to the unreactive form ($H_2PO_4^-$). In contracting muscle, where lactic acid is formed, the store of creatine phosphate is utilized and Pi is liberated. It has been shown that the activity of phosphorylase is critically dependent upon the concentration of Pi (Sahlin, 1983). The liberation of Pi from creatine phosphate and the effect of H^+ accumulation on the active form will thus be important for the regulation of glycogenolytic activity (Sahlin, 1983). As mentioned previously the PFK reaction, which seems to be the main regulatory step for glycolysis, shows a marked pH dependence and is almost completely inactive at a pH of 6.4. This would prevent a continuous high rate of glycolysis which would eventually result in whole-body acidosis and could affect the homeostasis and the function of acid-labile components in muscle and other more sensitive and critical tissues (i.e. heart and brain). The above shows why prevention of excessive lactic acid production is therefore necessary (Sahlin, 1983). Accumulation of fructose 6-P which is a substrate for PFK can overcome the pH inhibition of PFK and necessitates a control point higher up in glycolysis. It had been demonstrated in vitro that the transformation of phosphorylase b to a is slowed during acidosis. Later it was shown by Chasiotis et al. (unpublished data, in Sahlin, 1983) that in vivo during acidosis cAMP formation is inhibited and transformation of phosphorylase b to a in response to adrenaline is slowed down. The latter effect is at least partly due to the lower cAMP content in muscle but phosphorylase b kinase also seems to be affected by acidosis. As mentioned previously, ATP content as such does not seem to be limiting for the contractile machinery.

However, as shown, accumulation of ADP, Pi, and H^+ which occurs in fatigued muscle will decrease the energy yield for the ATP splitting process and a too low energy yield in some critical step utilizing ATP might occur (Sahlin, 1983). In 1983 , Donaldson also found that acidotic depression of contractile force generation should contribute to skeletal fibre fatigue even if it is not a primary causative factor. Donaldson also showed that mammalian skeletal fibres display type-specific acidotic depression of maximum force generation that correlates with their variable fatiguability. Slow-twitch fibres lost only 12% of maximum force, fast-twitch oxidative fibres lost 25% of their maximum force and fast-twitch glycolytic fibres declined 44% in maximum force with the same decrease in pH (from pH 7.0 to pH 6.5). Another interesting finding was that FT fibres are the least sensitive to Ca^{++} at pH 7.0 and their force generation at subsaturating Ca^{++} levels is greatly depressed by acidosis. However for the FOG fibres, their force generation at both pHs was intermediate relative to the other fibre types and all effects of acidosis were completely reversible (Donaldson, 1983). This is in agreement with findings from Tesch (1980) that found a positive correlation between %FT and decline in peak torque. Yates et al. (1983) also found that intense dynamic leg exercise which elevated blood lactate caused a 25% reduction in static arm endurance. The authors explained this reduction by the decreased blood pH and HCO_3^- which slowed the rate of lactic acid or H^+ ion efflux from the arm during the static holding task. A decreased rate of H^+ ion efflux from the arm could result in a more rapid decrease in intramuscular pH during the holding task and thereby a shorter endurance time.

Renaud and Mainwood (1985) found that the ionic conductance of the sarcolemmal membrane (a delicate sheath surrounding the muscle fibre) of frog sartorius muscle in situ at the resting potential and during an action potential are modified by fatigue and that these changes are modulated by extracellular pH. When

muscles were exposed to an extracellular pH of 8.0 the membrane conductance at the resting potential increased during fatigue by about 20% and returned to prefatigue level in about 20 minutes. The membrane conductance of fibres exposed to a pH of 6.4 was about three times less than that of pH 8.0 and decreased further during fatigue. Also, the recovery of a normal membrane conductance was slow at pH 6.4.

In 1987, Horita and Ishiko found that the changes in the frequency components of the EMG and in the contractile property of the muscle during short term intense exercise correlated with lactate accumulation in the muscle, and that the decrease in efficiency of the electrical activity in the muscle suggested peripheral fatigue. Komi and Tesch (1979) found that the higher percentage of FT fibres in the working muscle displayed the greater decline in the mean power frequency of EMG during contraction. Rapid decrease in firing frequency and number of active FT motor units during maximum voluntary effort has been shown elsewhere (Gydikov and Kosarov, 1974; Grimby et al., 1981). Tesch (1980) again showed that a general decrease in mean power frequency was observed during repeated muscle contractions. This shift was due to a decrease in the high frequency bandwith (associated with FT motor units). In individuals characterized by a high percentage of FT fibres, mean power frequency decreased to a higher extent as compared to individuals with a high percentage of ST fibres. The increase in the lower frequency components of the EMG during fatigue has been considered to be due to a decrease in the action potential conduction velocity along the muscle fibre as a result of lowered intramuscular pH associated with muscle lactate accumulation due to ischemia during contraction (Horita and Ishiko, 1987). The authors also found that the EMD (time interval between the onset of electrical activity and that of force production) correlated with the metabolic state of the muscle. The fatigue seems to be of peripheral nature as opposed to central nature. It also seems to involve the impairment of neuromuscular transmission, failure of muscle

action potentials and impaired excitation-contraction (E-C) coupling (Edwards, 1981). Duchateau et al. (1987) also found that muscles containing proportionally more FT fibres do not resist as well as the ones containing proportionally more ST fibres. This concurs well with the fact that FT produce a greater amount of lactate than ST. The authors found the recovery in tetanic tension, rate of tension development and time to half relaxation correlated well with the reduction in venous lactate concentration ($r = -0.95$, -0.93, and 0.96 respectively). Again the results of this study suggest that the reduction in muscle pH resulting from the accumulation of lactate essentially controls mechanical failure by changing the intracellular processes associated with tension development and subsequent relaxation (Duchateau et al., 1987).

Muscle fibre types and lactate.

At least two distinct fibre types have been found to exist within human skeletal muscle. These are referred to as fast-twitch (FT), which is glycolytic in nature, and slow-twitch (ST), which has a high oxidative capacity (McGrail et al., 1977; Essen-Gustavsson and Henriksson, 1983). Edgerton et al. in 1975 had identified a third fibre type, fast-twitch oxidative glycolytic (FOG). Human muscle is heterogeneous with respect to fibre type distribution. Within an individual, varying proportions of FT and ST fibres exist between single muscles and muscle groups (McGrail et al., 1977). It seems, therefore, that individual muscles and muscle groups are predisposed to function optimally either aerobically or anaerobically. Baldwin et al. found in 1977 that the elevations in blood lactate of rodents primarily result from lactate production in the FT fibre type. At a speed of 48 m/min lactate concentration was 7.19, 7.92, and 24.2 umol/g in soleus, FOG vastus, and FT vastus, respectively.

Among many other differentiating characteristics between FT and ST muscle fibres they are rather distinct with regard to the enzyme LDH. LDH exists in five isoenzyme forms , ranging in function from M-LDH (LDH-4, LDH-5), which strongly

favours the formation of lactate from pyruvate, to H-LDH (LDH-1, LDH-2), which conversely resists the formation of lactate and facilitates the reformation of pyruvate from lactate. In normal conditions, the M-LDH isozymes are mainly associated with membranes, such as the sarcoplasmic reticulum (near the Z band of the myofibril). However, H-LDH fractions have been found in the inner membrane of the mitochondria (Sjodin, 1976). Although a considerable portion of the LDH spectrum is present in all muscle fibres, M-LDH is relatively more abundant in FT while ST has a predominance of H-LDH (Sjodin, 1976; Tesch et al., 1978). Total LDH activity was also found to be higher in FT fibres than in ST fibres. Endurance athletes have a higher activity of LDH-1 and LDH-2 compared to normal subjects (Sjodin et al., 1976). Anaerobic training did not change the LDH profile of the muscle and Sjodin et al. suggested that the improvement with the training may be due to better lactate release. This would be in accordance with an increased buffering capacity. Long distance running (at 70%-80% of max HR), however, increased the H-LDH/M-LDH ratio. Sjodin et al. (1976) theorized that the LDH enzymes may be involved in a transport mechanism because of where they are attached. Essen and Haggmark (1975) found a higher concentration of lactate in FT as compared to ST muscle fibres. Tesch et al. (1978) also found a positive correlation between the percentage of FT fibres in the muscle and the lactate concentration in the muscle ($r=0.74$, $p<0.01$) while Sahlin and Henriksson (1984) found a correlation of $r=0.65$ ($p<0.01$) between the same parameters. Tesch et al. (1978) found positive correlations between lactate concentration in muscle and total LDH and LDH-M activity ($r=0.66$, $p<0.05$, and $r=0.70$, $p<0.02$ respectively). M-LDH activity and muscle fibre type distribution were positively correlated ($r=0.98$, $p<0.001$) and so was the relation between lactate concentration in FT fibres and muscle fibre distribution ($r=0.91$, $p<0.001$). Lactate concentration in ST fibres was not correlated with muscle fibre type distribution

(r=-0.04, n.s.). As concluded by Tesch et al. (1978), a high proportion of M-LDH and a greater total LDH make FT fibres better at forming lactate than ST fibres. This is in accordance with Edgerton et al. (1983) who reported that FT and FOG fibres are used when high level of force and power is needed for a given work bout. Tesch in 1978 found a correlation between %ST and $VO_{2\ max}$ ($r= 0.80$, $p < 0.05$) in subjects having just performed a bicycle exercise leading to exhaustion after 1.67 min in average. FT muscle fibres again had a significantly higher concentration of lactate when compared to ST fibres. This is in accordance with the different metabolic profile of the two muscle fibre types (Tesch, 1978). Lactate concentration in the whole muscle was related to the %FT fibres ($r=0.85$, $p<0.05$). A relationship between lactate concentration and performance time was demonstrated ($r=0.76$, $p<0.05$). In addition, Tesch noted that the subjects with muscles rich in FT fibres had higher performance capacity in the anaerobic bicycle test. These findings, as mentioned by Tesch, seem to indicate higher anaerobic power and capacity in subjects rich in FT fibres.

Medbo and Sejersted (1985) later found that sprint trained athletes obtained a more pronounced lactic acidosis and a lower pH as compared to endurance trained athletes. Tesch again found in 1980 a correlation between lactate concentration and %FT fibre. For cycling exercise, isokinetic exercise and downhill skiing, the correlations were respectively: $r=0.85$, $P<0.01$; $r=0.74$, $P<0.01$; $r=0.93$, $P<0.001$. Correlation coefficients between LDHtot as well as M-LDH activity and individual muscle fibre type distribution (%FT) were 0.96 ($p<0.0001$) and 0.98 ($p<0.001$), respectively. Individual differences in muscle lactate accumulation after exercise were found to be related to individual variations in LDHtot ($r=0.66$, $p<0.05$) and M-LDH ($r=0.70$, $P<0.05$) activity (Tesch, 1980). An effective anaerobic ATP production (helped by an effective anaerobic glycolysis) would permit the muscle fibre to produce and maintain this elevated force and power output. Tesch and Karlsson (1984) also

measured higher lactate concentrations in FT muscle fibres than in ST muscle fibres following leg presses at 50% isometric contraction and at maximal isometric contraction. They also noticed that the difference tended to increase with exhaustive, isometric training. Skeletal muscle then, with three fibre types and the full range of LDH isoenzymes, is capable of simultaneous production and utilization of lactate (McGrail et al., 1977; Tesch et al., 1978). Consequently, blood lactate concentrations during submaximal exercise is thus dependent upon the intensity of the work and perhaps the ratio of FT:ST muscle fibres (McGrail et al., 1977).

The FT:ST ratio, for most muscle, varies among individuals and in part may explain the frequently reported differences in lactate production and removal rates between muscle groups and individuals. An increased utilization of ST fibres, as compared to FT fibres, may not only minimize the production of lactate, but may also accommodate the oxidative metabolism of lactate after its conversion to pyruvate (McGrail et al., 1977).

During exercise, the formation, and possibly the removal of lactate, are also influenced by the state of training of an individual. Training does not affect the ST:FT ratios within muscle, however the enzymatic profile of rat and human muscle can be significantly altered by training (McGrail et al., 1977). The increases reported in the oxidative enzyme activities, in particular citrate synthase and succinate dehydrogenase (Essen-Gustavsson and Henriksson, 1983; Holloszy et al., 1971), which accompany aerobic training, greatly facilitate the aerobic metabolism of substrates (i.e. glycogen, glucose, fatty acids), hence reducing the dependence upon the contribution made by the formation of lactate. As a possible result of these enzymatic adaptations, it has often been observed that less lactate is produced at a standardized submaximal workload as a result of aerobic training (McGrail et al., 1977). This is in contradiction with the findings of Donovan and Brooks (1983) which showed that in rats, training

improved the lactate removal but did not effect the lactate production. Young and Maughan (1983) found that the improvements from aerobic training seem to suggest that the increase in physical exercise capacity is brought about primarily by changes occurring within the trained muscles. The changes in cardiovascular dimensions which take place during the training period did not appear to cause an increase in VO_2 max or in exercise performance if the exercise involves muscle groups other than those involved in the training program. Rowell (1974) outlines in his paper that 50% of the increase in VO_2 max with physical conditioning in very sedentary or normally active young men is due to the rise in stroke volume. It accounts for all of the increase in VO_2 max in subjects who are already trained and all of the decrease in VO_2 max during total bed rest (Rowell, 1974). This is in agreement with the results obtained by Henriksson and Reitman (1977) which found that, in healthy men, after 8-10 weeks of endurance training, VO_2 max increased 19%, SDH activity 32%, and cytochrome oxidase activity 35%. However, at 6 weeks post-training VO_2 max was still 16% above pre-training level, and not significantly different from the level at the end of training. But SDH and cytochrome oxidase had returned to pre-training levels (cytochrome oxidase within 2 weeks). Thus, the enhancement of the oxidative potential in skeletal muscle is not a necessity for a high VO_2 max. In relation to Rowell's paper, an improved stroke volume would be a necessity. However, as mentioned by Vander et al. (1985), how much of the increase in maximal stroke volume is due to changes in the heart itself, as opposed to changes in skeletal muscle vasculature (which would facilitate venous return), remains unclear. Gollnick et al. (1973) also found that after an intensive 5-month training program (75% VO_2 max at the beginning to 85% of VO_2 max at the end) VO_2 max increased 13%. This small increase in VO_2 max contrasted with the large change in oxidative potential of the skeletal muscle (SDH activity increased 95%, PFK increased 117%) supports the

theory that the aerobic capacity of muscle does not limit total body VO_2 max. Holloszy et al. (1970) suggested that these changes in oxidative capacity are probably more important during submaximal than maximal work. They may contribute to a greater oxidation of fat and the lower lactate production seen during exercise after training. Sjodin et al. (1982) then found that a steady state training intensity which approximates V_{OBLA} (running intensity that elicits 4 mmol/l blood lactate) will increase V_{OBLA}, and will result in measurable local metabolic adaptations in the active skeletal muscles of well-trained runners without a significant change in maximal aerobic power. The PFK/CS ratio was decreased and the H-LDH isozyme increased. This would support the idea of a relatively slower rate of glycogenolysis and an increased potential to oxidize pyruvate and/or lactate. Skilleter (1972) previously demonstrated that H-LDH bound to the inner membrane of the mitochondrion will facilitate an oxidative translocation reaction of lactate in the presence of NAD. The net result should be lower muscle and/or blood lactate accumulation at a given absolute and relative exercise intensity, as has been previously demonstrated (Karlsson et al., 1972), and consequently an increase in V_{OBLA}. Daniels et al. (1978) suggest that factors representing local muscular metabolic characteristics should be considered when attempting to evaluate the adaptation to training in trained athletes. This is in the light of their findings that, after training well-trained runners at an intensity higher than their normal training program, performance improvements were noted without any significant alteration in the runners' VO_2 max. This, as explained by Sjodin et al. (1982), may be due in part to the inverse relationship existing between initial physical training status and the increase observed in maximal aerobic power following a training program.

An individual's capacity to produce lactate on short duration maximal exercise has been demonstrated to be increased as a result of anaerobic training. This could

probably be attributed to the increased activity of PFK (McGrail et al., 1977). A similar increase on LDH-M activity has not been demonstrated (Sjodin et al., 1976; McGrail et al., 1977). However, one must not overlook the psychological adaptation to training, which may enhance one's capacity for extended maximal exertion.

Lactate removal.

The process of lactate removal, and the relative contributions, made by the different removal sites of the body have been the topic of much study and appear to be dependent upon many factors. Lactate which accumulates as a result of exercise may be removed at two major sites: skeletal muscle and liver (McGrail et al., 1977; Donovan and Brooks, 1983). The heart is responsible for about 10% of the lactate removed during exercise (Newman et al., 1937, in McGrail et al., 1977), whereas the contributions made by the kidneys and the brain are generally accepted to be negligible when compared to skeletal muscle and the liver.

In the past, the liver had been considered to be the primary site of lactate removal during both rest and exercise (Rowell et al., 1966). Cori and Cori (1929, in McGrail et al., 1977), were the first to describe the reconversion of lactate to glucose in the liver (Cori cycle). This also occurs, although to a much lower extent, in the kidney, for both organs possess the key gluconeogenic enzymes (phosphoenol pyruvate carboxylase, pyruvate carboxylase, fructose-1,6-diphosphatase and glucose-6-phosphatase) which are necessary to reverse the glycolytic pathway. Himwich et al. (1939) proposed that hepatic glyconeogenesis from lactate could also occur, and later this was experimentally demonstrated in animals (Drury and Wick, 1956). Further animal research, however, has found this to be negligible (Brooks et al., 1973). In spite of this, human hepatic glyconeogenesis has recently been presumed to occur (Ahlborg et al., 1975). The amount of lactate removed by the liver has been shown to increase with exercise (Rowell et al., 1966), but not necessarily

proportionally with exercise intensity. Splanchnic lactate removal has been calculated to dispose of as much as 50% of the total body lactate, primarily via gluconeogenesis (Rowell et al., 1966). Turnover studies have revealed that 12-20% and about 25% of blood lactate is converted to glucose during rest and exercise respectively. This process may account for 10-13% of the total blood glucose during exercise (McGrail et al., 1977). Brooks and Gaesser (1980) also found that lactate plays a role in the postexercise gluconeogenic processes but their results show that the majority of lactate is oxidized directly and not converted to glucose prior to oxidation.

During resting recovery after exercise, lactate is taken up in both exercised and non-exercised skeletal muscle. When exercise is performed after blood lactate has been elevated, the working (McGrail et al., 1977) and non-working muscles (Ahlborg et al., 1975) show an increased lactate uptake. By monitoring the formation of $^{14}CO_2$ from infused ^{14}C-lactate, it has been concluded that lactate is metabolised in skeletal muscle (Brooks and Spurgeon, 1983) even during exercise (McGrail et al., 1977). Estimations predict that during exercise as much as 74-75% of the lactate is metabolised in muscle, while this is estimated to be 50-58% at rest in dogs (McGrail et al., 1977). Even though the enzymes necessary for gluconeogenesis have been found to have a higher activity in FT than in ST (Bass et al., 1969) this activity is relatively low and lactate must have been metabolised via the Krebs cycle. Direct support has been shown for this in man, since it was found that lactate removal occurred more rapidly during continuous exercise than during intermittent exercise (McGrail et al., 1977). The results of Hermansen and Stensvold (1973) in relation to lactate recovery during exercise indicated that skeletal muscle, rather than the liver, may be regarded as the main site for lactate removal during exercise. During rest, approximately 40 to 50% of the lactate produced is removed through oxidation (Brooks, 1986). With the use of tracer studies, Brooks et al. (1986) were able to find that during sustained exercise,

muscle glycogen is the source of most carbohydrate derived fuels combusted. Again, during sustained exercise, the turnover rates of lactate exceed those of glucose. Brooks (1986) also states that lactate production during both rest and exercise is not necessarily associated with muscle anaerobiosis. This statement has however been questionned by Katz and Sahlin (1988) who feel that data underlying the conclusion that lactate production during exercise is not O_2 dependent were found to be 1) questionable, or 2) interpretable in an alternative manner.

In 1978, McGrail et al. found a high correlation between VO_2 cost and lactate recovery ($r = 0.92$) and concluded that, in man, lactate removal from the blood during recovery exercise occurs primarily in skeletal muscle.

In 1980, Brooks and Gaesser found (using [U-^{14}C]lactate) that a quantitatively significant amount of glucose and lactate was incorporated into a number of amino acid pools, including alanine, glutamine, glutamate, and aspartate after 15 minutes of recovery from exhaustive exercise. This represented a previously unrecognized and quantitavely important pathway for lactate metabolism. Why lactate and glucose are converted into amino acids in muscle or other tissues is not entirely clear at present. It may provide a means for reducing the concentration of free ammonia produced in skeletal muscle during exercise and provide a nontoxic means for the transport of ammonia from muscle to liver (Brooks and Gaesser, 1980).

Brooks and Gaesser (1980) were also the first to demonstrate incorporation of [U-^{14}C]lactate into glycogen after exercise. Although exercise does stimulate glycogen synthesis, analyses of all major glycogen-containing tissues indicated that less than 20% of the lactate present at the end of exercise is reconverted to glycogen in the postexercise period. Lactate is also buffered by HCO_3^-. Sharp et al. (1986) found that muscle buffer capacity is increased with highly intense sprint training but they found no evidence to suggest that muscle buffer capacity is affected by endurance

training. This supports the findings of Sahlin and Henriksson (1984) described previously. Oxydation was found to be the primary postexercise fate of lactate by Brooks and Gaesser (1980). After 4 hours, synthesis of glycogen could account for only minor fractions of the metabolism of [U-^{14}C]lactate after exhaustive exercise of continuous or intermittent nature (the studies were performed on female Wistar rats). This however comes in contradiction with the findings of Hermansen and Vaage (1977) who concluded that after exercise, synthesis to glycogen in muscle is the primary fate of lactate produced in muscle during exercise. Brooks and Gaesser (1980) explained this discrepency in the results by the questionable method utilised by Hermansen and Vaage (1977) in their measurement of blood flow and the absence of direct (^{14}C tracer) determinations of lactate removal. Also, Brooks and Gaesser criticized the authors for lack of consideration of the total body lactate pool. The calculations of lactate-glycogen conversions presented by Hermansen and Vaage pertain only to the leg muscles. The larger remainder in the lactate space was unaccounted for (Brooks and Gaesser, 1980). Hermansen and Vaage (1979) had found that less than 15% of the lactate disappearing in muscle during recovery apeared to be oxidized to carbon dioxide and water.

Brooks (1986) summarized in his paper that carbon flux through the lactate pool appears to be an important means by which the anabolic functions in some tissues (e.g., liver gluconeogenesis and glycogenesis) are coordinated to catabolic processes in other tissues (e.g., glycogenolysis, glycolysis, and carbohydrate oxidation) to produce an overall integrated metabolic response to stress.

Although it has become apparent that skeletal muscle possesses gluconeogenic enzymes, especially in FT muscle fibres (Bass et al., 1969), Brooks and Gaesser (1980) found that gluconeogenesis from lactate in skeletal muscle after exhausting exercise did not appear to be a major pathway of lactate removal. Their results suggest that a

majority of the glycogen repleted in skeletal muscle resulted from gluconeogenesis in liver and kidney. However, during exercise, lactate removal during gluconeogenesis is reduced to approximately 20% since the relative blood flow to the gluconeogenic areas of the gut is decreased in favor of the working muscle (Donovan and Brooks, 1983). Brooks and Gaesser (1980) also found that a large and rapid incorporation of label from ^{14}C-lactate into glucose occured in the liver and kidney. Muscle radiochromatograms revealed that incorporation of label into muscle glycogen was more rapid following ^{14}C-glucose injection than following ^{14}C-lactate injection. Restoration of muscle glycogen paralleled restoration of blood glucose levels. However, their results do not rule out some direct conversion of lactate to glycogen within the muscle, especially in the FT muscle fibres (Bass et al., 1969), but indicate that the direct conversion of lactate to glycogen in muscle is not of great importance.

The rate of lactate uptake by the muscle is related to the blood lactate concentration, the existing lactate concentration gradient between the muscle and blood compartments, and the level of muscle glycogen stores. When arterial lactate concentrations are increased during exercise, blood flow and oxygen uptake are increased in the non-working muscles, which may result in increased lactate metabolism in these tissues (Ahlborg et al., 1975). This is supported by results from Poortmans et al. (1978) and Harris et al. (1962). Significant removal of lactate from the blood by non-exercising muscles stops soon (approximately 10 minutes) after the cessation of exercise (Poortmans et al., 1978). The probable increased uptake of lactate in ST fibre as it becomes recruited within the working muscle group may also come into play. Considering the mass of muscle tissue, and in light of the results of many studies (Hermansen and Stensvold, 1972; Hermansen et al., 1975; Issekutz et al., 1976; Minaire and Forichon, 1975), it seems that skeletal muscle, rather than liver, serves as the main site of lactate removal during exercise. Donovan and Brooks

(1983) obtained results on female Wistar rats showing that the effect of training is not on production of lactate but on its clearance from the blood. Isotopically labeled [2-^{3}H]- and [U-^{14}C]lactate was used to measure rates of lactate turnover. No significant difference was observed between trained and untrained subjects in relation to the lactate turnover rates. It appears that lactate turnover is directly correlated with metabolic rate (VO_2) during exercise. Trained animals had less lactate at a given submaximal exercise due to higher lactate clearance rates compared to untrained animals and not due to lower lactate production. Higher blood glucose levels in trained animals during heavy exercise are due to a greater glucose flux that is supported by a lesser loss of lactate carbon to oxidation and a greater conversion of lactate to glucose.

It has been shown that the net rate of lactate removal rises, with increasing exercise intensity up to some critical workload (Belcastro and Bonen, 1975; Hermansen and Stensvold, 1972; Davies et al., 1970). The optimal intensity of recovery exercise for lactate removal would result when the lactate production rate is minimal and the removal rate is maximal. The identification of this optimal intensity in humans has understandably resulted in somewhat discrepant results since experimental designs and work tasks have varied among studies.

The findings of Gisolfi et al. (1966) concurred with previous studies which have shown that lactate removal during active recovery exercise occurs more rapidly than at rest. They also found that removal rates varied with exercise intensity, being greater at 52% and 53% than at 39% and 43% $VO_{2\ max}$. Subsequently, the possibility that an optimal specific exercise intensity may exist for the most rapid removal of lactate from the blood was studied by Hermansen and Stensvold (1972). In this study, lactate removal was monitored during treadmill recovery exercise at 30, 60, 70, and 80% VO_2 max. The critical level for both increased production and decreased removal of

lactate was found to lie between 60% and 70% $VO_{2\ max}$. In a more recent study by Bonen and Belcastro (1976), lactate removal rates at 61% $VO_{2\ max}$ were similar to those of Hermansen and Stensvold (1972).

In studies employing bicycle ergometer exercise, Davies et al. (1970) found that following 6 minutes of exercise at 80% $VO_{2\ max}$, lactate removal rates increased with exercise intensity up to a workload corresponding to 40% $VO_{2\ max}$ beyond which the removal rate began to decrease. Subsequently, Belcastro and Bonen (1975) reported that the average optimal recovery exercise intensity for bicycle ergometer exercise was 32% $VO_{2\ max}$. In addition, these investigators found that lactate removal could be regulated quite well by the subjects, without any feedback, as long as they maintained a constant workload. Bonen et al. (1978a) found that there was a moderate but significant correlation between %ST fibres of the vastus lateralis and lactate removal rates during moderately active exercise recovery ($r= 0.544$, $P=0.05$). Tesch and Wright (1983) also found that following 50 consecutive maximal voluntary knee extensions (with a Cybex II isokinetic device), fatigue and recovery respectively were correlated with capillary density ($r= -0.71$ and 0.69) but not fibre type distribution. Their data suggest that lactate elimination from the exercising muscle is partly dependent upon the capillary supply and subsequently influences the rate of muscle force recovery (Tesch and Wright, 1983).

The apparent differences between the optimal recovery exercise intensities of bicycle ergometer and treadmill exercise have been interpreted as possibly resulting from differences in the physical condition of the subjects, the muscle mass involved in the exercise performed, or the muscle fibre type distribution of the individuals (McGrail et al., 1977).

Brooks (1986) also proposed the idea of a 'lactate shuttle' within active muscle. The author points out that as suggested from A-V difference measurements of lactate

and tracer across muscle, approximately only half of the lactate formed in a working muscle bed is released into the venous circulation. Approximately half the lactate formed in such a muscle, together with a significant quantity of lactate removed from the arterial circulation, is combusted within the muscle and appears as CO_2 in the venous blood (Brooks, 1986). Based on results from Baldwin et al. (1978, 1977) who studied the metabolic characteristics of the various mammalian fibre types, Brooks (1986) suggests that lactate produced as the result of recruitment of type IIb fibres (FT), or because of the mechanics of the contractile process, diffuses toward and is transported into type I (ST) or type IIa fibres (FOG) where the lactate is oxidized. In this way, the more glycolytic fibres within a working muscle bed shuttle oxidizable substrate (i.e. lactate) to the neighboring cells with higher respiratory rates.

LACTIC ACID AS AN INDICATOR OF EXERCISE INTENSITY AND RECOVERY

Lactic acid has often been used as an indicator of the intensity of exercise and has also been investigated as an indicator of recovery from exercise (Astrand and Rodahl, 1986; Evans and Cureton, 1983; Weltman et al., 1979; Klausen et al., 1972; Hirvonen et al., 1987; Jorfeldt et al., 1978; Bonen et al., 1978b; Arabas et al., 1987; Thimm and Meir zu Verl, 1984). The relation between blood lactate concentration and work intensity is curvilinear. Astrand and Rodahl (1986) mention that one of the criteria for reaching $VO_{2\ max}$ is a blood lactate concentration of at least 8mmol/l. OBLA (onset of blood lactate accumulation) has also been used as an indicator of the intensity of exercise (Sjodin et al., 1982). Lactate recovery, as detailed previously, has also been the subject of much investigation. The findings conclude that the rate of blood lactate dissapearance increases if light aerobic exercise is performed during the recovery period (Evans and Cureton, 1983; Koutedakis and Sharp, 1985; Bonen et al., 1978b; Weltman et al., 1979; Jorfeldt et al., 1978). The lactate recovery was measured

on average for 20 minutes. As mentioned previously, the removal of lactate during exercise may be influenced by the state of training of an individual (McGrail et al., 1977). Endurance athletes have a higher activity of LDH-1 and LDH-2 isoenzyme compared to normal subjects (Sjodin et al., 1976). Long distance running (at 70-80% of max HR) increased the H-LDH/M-LDH ratio. Sjodin et al. (1976) theorized that because of its point of attachment (inner membrane of the mitochondrion), LDH enzymes may be involved in a transport mechanism. Also, an increase in V_{OBLA} was partly explained by an increased potential to oxidize lactate due to an increased LDH-H activity that occurred following training (Sjodin et al., 1982). As mentionned previously, Bonen et al. (1978a) found a moderate but significant correlation (r=0.544, P=0.05) between %ST fibres of the vastus lateralis and lactate removal rates during moderately active exercise recovery. This would support the theory of Sjodin et al. (1978) since LDH-H is predominantly found in ST muscle fibres (Sjodin et al., 1976; Tesch et al., 1978). However, Tesch and Wright (1983) found that recovery from fatigue following 50 consecutive maximal voluntary knee extensions was correlated with capillary density (r=0.69) but not with fibre type distribution. The authors suggested that lactate elimination from the exercising muscle is partly dependent upon the capillary supply and subsequently influences the rate of muscle force recovery. This would partly explain why Evans and Cureton (1983) found that training improves lactate recovery during exercise but not during rest. Since blood flow to the working muscle is greatly diminished during the 30 minutes of rest when lactate measurements were taken, lactate could not then be as effectively transported to other organs capable of removing lactate, such as the inactive skeletal muscles, heart and liver (Evans and Cureton, 1983), not to mention that the usage of lactate as a fuel may be increased. However, no study that looked at improved lactate recovery speed has been done. Theoretically, based on the results from Brooks (1986) showing that approximately

only half of the lactate formed in a working muscle bed is released into the venous circulation and also based on his proposal of a lactate shuttle where lactate would diffuse from a type IIb fibre to a type I or IIa fibre, lactate could more quickly be oxidized in those muscles if those muscles where trained and had a higher activity of LDH-1 and LDH-2 isozymes (which has been shown to be true for trained muscles (Sjodin et al., 1976). In this scenario, the lactate equilibrium between muscle and blood may occur more rapidly, especially if this would be joined with an improved buffer capacity which can be obtained through anaerobic training. Lactate measurements taken early following exercise may then give interesting information to the researcher and the athlete. However, this is only theory and as mentioned by Evans and Cureton (1983), a study of the effects of conditioning on the peak rate of blood lactate removal is clearly needed.

RELATIONSHIP BETWEEN HEART RATE AND LACTIC ACID

The relationship between heart rate (HR) and lactic acid at work and during recovery will be examined in this section.

Lactate concentration was one of the metabolic factors previously identified as being able to affect the HR drive. In 1976, Tibes et al. measured femoral venous $[K^+]$, $[H^+]$,[Pi], osmolality, and PO_2 on a bicycle ergometer at graded increasing work rates. They found that at a given VO_2, the increase femoral venous $[K^+]$, $[H^+]$, [Pi], osmolality (OSM), and the decrease of PO_2 were significantly lower in trained than in untrained subjects. In the same proportion the increases of HR, ventilation (V_E), and respiratory quotient (Q) were diminished. Thus in trained and untrained subjects, identical and highly significantly correlated regression lines of femoral venous $[K^+]$, $[H^+]$, [Pi], osmolality, and PO_2 with HR, V_E, and Q were obtained. These constituents changed in the same proportion as the relative VO_2 in the trained and

untrained. No relationship with [Na+], [Ca^{++}], and [Mg^{++}] was found. A regression equation in relation to HR was found:

$$HR = 61.7\,[K^+] + 19.0\,[H^+] + 1.14\,OSM + 0.6\,[Pi] - 0.5\,PO_2 - 602$$

$$r = 0.912{,}2\ P < 0.001$$

Since [H^+] and OSM are greatly affected by [lactate], higher femoral venous [lactate] may be linked according to this study to elevation of HR, V_E, and blood flow (Tibes et al., 1976). In 1977, Tibes et al. showed that [lactate] (10-30 mmol), alone or in combination with concentration of K^+, Pi, and OSM that is found in the interstitium of working muscles, could strongly activate the C-fibre units that are responsible for the cardiovascular respiratory reflex from the dynamically working muscles. CO_2 (10%) and lack of O_2 were almost ineffective. A-fibres did not respond to the chemical changes (McCloskey and Mitchell, 1972; Tibes and Groth, 1977; Tibes, 1977). In 1978, Tibes et al. found that most group III and IV nerve fibres showed a polymodal sensitivity against increasing [K^+] (to 10 mmol), OSM (to 330mOSM/kg H_2O), [lactate] (to 15mmol), and [Pi] (to 5 mmol) (in post. rat ext. dig. long. muscle). Thimm et al. (1984) and Thimm and Meier zu Verl (1984) later studied the response of HR in relation to the perfusion of the rat's hind leg with concentrations of K^+, OSM, lactic acid, and Pi that reflected the changes occurring during heavy exercise. Only perfusion with a solution enriched with lactic acid elicited a significant increase in HR. Lactic acid concentration of 5 mmol/l up to 25 mmol/l evoked significant increases in HR (Thimm et al., 1984). After separating the effects of pH and lactate, it was found that the receptors were sensitive to lactate but especially at low pH (6.8). In 1984, Thimm and Meier zu Verl found that HR increases when the limits of normal ranges of pH and lactate in the resting muscle are exceeded. Thus it was concluded that [lactate] and HR increases are correlated not only under perfusion conditions but also

when lactic acid is produced by the muscle itself. Moreover, their experiment tends to show that there is a HR drive even when the physiological ranges of the resting muscle are only slightly exceeded. The authors suggest that lactic acid possibly causes a HR drive in light exercise also.

These studies indicate a relationship between HR and [Lac] during exercise but the mechanism which controls its return to baseline is still unknown. Lactic acid has been mentioned because of its relation with HR during exercise but, following exercise, the concentration of lactic acid in the interstitial space remains elevated for several minutes and yet during this time Saltin et al. (1981) found that HR was already decreasing. Contrarily, Thimm et al. (1984) found that the increases in HR lasted for several minutes although the lactate concentration in perfusion outflow decreased during that time. The authors hypothesized that this prolonged effect on HR may occur because the lactate concentration in interstitial space remains relatively high due to slow back-diffusion from interstitial space to the capillaries. However, it is also possible, as mentioned by Thimm et al. (1984), that the mechanisms which control the increase in HR are different from those which control the return to base-line. The possibility of lactate concentration affecting HR recovery from exercise is not yet clear.

THE INFLUENCE OF EXERCISE SPECIFICITY ON HEART RATE AND LACTIC ACID RESPONSES

The specificity of different muscle fibres in relation to their aerobic or anaerobic energy production has been described. The specificity of exercise also seems to influence the responses of heart rate and lactic acid. Saltin et al. (1976) studied subjects who performed endurance training with one leg and sprint training with the other. In other groups, one leg was trained and the other one served as a control.

In the group that had trained one leg by sprint and the other by endurance, the increase in maximal oxygen uptake was, on the average, 0.32 and 0.54 liters/min respectively. In the group that had trained one leg, a marked reduction in heart rate during one-legged submaximal exercise was noted. When exercising the untrained leg only, a much smaller reduction in the heart rate was observed compared to the pretraining level. A decrease in lactate response and enhancement of SDH activity were observed only in exercise with the untrained leg. Henriksson (1977) noted, after two months of training, a 27% higher SDH activity in the trained leg than in the untrained one. The maximal oxygen uptake increased 11% and 4% respectively in the testing of the trained and the untrained leg. At a submaximal two-leg exercise at an average of about 70% of $VO_{2\ max}$ (for one hour) the subjects apparently worked harder with the trained leg. However, the degree of utilization of free fatty acids was higher in the trained than in the untrained leg indicating a difference in the oxidative capacity. As mentioned by Astrand and Rodahl (1986), these studies seem to indicate that irrespective of what factors trigger the adaptations at the cellular level, causing effects mainly in those muscles which are being trained, there are no, or only modest, effects elicited in nontrained muscles. For this reason it is important that the testing of the athletes be specific to their sport. The more specific the test is to the sport movement, the more valid is the test (Sale and Norman, 1982).

Again in relation to lactic acid response to exercise, Sjodin et al. (1976) measured the effect of physical training on LDH activity and LDH isozyme pattern in human skeletal muscle. The effects of three different training protocols were examined: 1) aerobic training performed as interval training, 2) aerobic training performed as extreme distance running, and 3) anaerobic training for two months.

After anaerobic training no change in LDH properties could be detected, although the running performance improved. This improvement was attributed to a

possibly faster lactate release from the muscle which may have directly or indirectly slowed down the development of muscle fatigue. A possible explanation may be that, as found by Sahlin and Henrikssonn (1984), the buffer capacity of the trained muscles was increased which may result in a faster lactate release from the muscle. These findings of unchanged LDH activity after anaerobic training are identical with the results obtained after endurance training using the interval training principle as well as after strength training performed as weight lifting or sprint running (Thorstensson, unpublished data, in Sjodin, 1976).

However, the extreme distance running (at 70-80% of maximal HR) resulted in a decrease in total LDH activity and an increase in relative activity of LDH-1 and LDH-2 heart specific isozymes. A relationship was also shown between the relative activity of these isozymes and the training distance covered. From these results, it seems that the lactic acid response may change in relation to the intensity specificity of training.

PHYSIOLOGY OF BADMINTON PERFORMANCE

As mentioned by Astrand and Rodahl (1986), badminton is an intermittent sport where bursts of work are separated by rest periods. It can be a very demanding physical sport, even for those who only practise it for the exercise. Compared to volleyball, tennis, swimming, gymnastics and cross-country running, badminton is either as physically demanding or more when the results are expressed in kcal/min (Coad et al., 1979). However, as mentioned by Docherty (1978), the higher the level of skill (competition), the greater the stress placed upon the individual's cardiovascular system (higher average heart rate).

The amount of research in relation to the sport of badminton is not very extensive; however, some interesting scientific studies have been performed. Two

time-motion analyses were conducted on the 1979 and the 1981 Badminton World Championships (men's single final) (Reed, 1981). It was then determined that the average work period (rally) lasted 10 seconds and the average rest period lasted also 10 seconds. Agnevik (1970, in Astrand and Rodahl, 1986) studied a group of top Swedish badminton players and showed, from recordings of heart rates during important international matches, that the players attained near maximal values (90%-100% of max HR) in singles matches. He also found that the mean maximal oxygen intake was respectively 55.6 ml/kg/min and 46.9 ml/kg/min for men and women. After this, Mikkelsen (1979) published the most extensive physiological study to date performed on elite badminton players. For his study, Mikkelsen had selected subjects from the best male Danish badminton players (the Thomas Cup Team) as well as the best female players (the Uber Cup Team). This study found that, in relation to mean fibre composition, male badminton players had 63% type I (ST) fibres in medial vastus lateralis (m.V.L.). The mean fibre composition for women was 56% ST. For both groups, one third of the fibres were IIa (FTa) fibres. Few IIB (FTb) fibres were found, 4% among males and 9% among females. Badminton players are also characterised by a preferred leg which is the leg of the racquet hand. For example, for a right handed player the right leg is the preferred one. All players in this study were right handed so the right leg was their preferred leg. Both sexes had more ST fibres in the right m.V.L. than in the left m.V.L.. Fiber area measurements showed that, for all subjects, the ST fibres were bigger than the FT (8%) and FOG (18%). The ST fibres were also 13% bigger in the right V.L. compared to the ones in the left V.L.. Thus they also occupied a bigger cross-sectional part of the muscle (65% for males and 60% for females). FT fibres were also found to be 12% larger in the right V.L. than in the left V.L..

The capillary supply showed that there were more capillaries in the ST and FT fibres of the right V.L. than in the left. The SDH activity also appears to be greater in the right V.L. than in the left V.L. by 17%. The circumference measurements of the calves and the thighs also showed a significantly larger right leg. The strength and the holding time at 50% MVC were both greater for the right leg.

Maximal oxygen uptake was higher than those reported by Agnevik (1970). Results of 59.4 ml/kg/min and 52.9 ml/kg/min were respectively found for men and women. Heart rates recorded telemetrically during open and national competitions showed that the heart rates in singles were less than 10 beats from maximal heart rate. Blood lactates however reached a highest concentration of only 5.73 mmol/l and the mean value was 2.98 (+/- 0.80 mmol/l).

From these results, Mikkelsen (1979) theorized that nearly all of the energy required by the elite badminton player comes from a high central as well as a high peripheral aerobic capacity. He also concludes that the myoglobin stores in the muscles liberated enough oxygen to enable periods of work up to a length of about 10 seconds to be performed almost totally aerobically. For this reason, the anaerobic system does not come into play and very little lactate is formed through anaerobic glycogenolysis.

However, some questions arise in relation to this conclusion. If the sport of badminton is almost completely aerobic, it's not clear why the $VO_{2\ max}$ of the elite players are not higher. Also, studies done in 1979 from Jansson et al. (1983) and Nemeth et al. (1983) have shown that myoglobin concentration in muscle does not increase with training. Jansson et al. (1983) found an increased capillary density and an increased CS activity following training but a similar concentration of myoglobin was found in trained and untrained subjects. Nemeth et al. (1983) found that FT fibres had 2/3 as much myoglobin as the ST muscle fibres. Myoglobin concentration was

actually found to be higher in the control group than in the trained group. They also noted that the myoglobin content was not affected in either fibre group by detraining despite changes in glycogenolytic and oxidative enzymes. These findings do not tend to support the conclusion brought forward by Mikkelsen (1979).

Other studies (Gollnick and Hermansen, 1973; McCartney et al., 1986; Jacobs, 1983) show that the energy yielded at the beginning of exercise is in most part produced from the splitting of high energy phosphates (ATP and CP). Glycolysis has also been shown to be very low at the beginning of exercise. The combination of these factors (anaerobic metabolism) would support a low formation of lactate and this added to a good lactate recovery (aerobic metabolism) could explain the low level of blood lactate concentration found in elite badminton players. If this hypothesis holds true however, the longer the rally, the higher the lactate concentration in blood. A pilot study confirmed this hypothesis. As mentioned by Thoden et al. (1982), racquet sports which require a series of brief (5-20 sec.) bursts of highly intense energy separated by periods of lower intensity recovery derive most of the energy directly from non-oxidative sources. Also, in badminton, reaction speed is most important, but basic speed and speed endurance also play a role. Moving as fast as possible and being able to maintain this speed during the whole match is what matters. Reed (1987) proposed a training strategy for badminton players known as the 'Double-A' training strategy. It is based on the theory that badminton players need to train both aerobically and anaerobically (alactic energy system). The aerobic training would improve lactate removal and the alactic training would improve high energy phosphate capacity and also possibly improve the buffer capacity of the trained muscle. The specific goal of this training strategy is to keep lactate levels at a minimum since, as has been presented previously, too large an increase in muscle lactate concentration may result in muscular fatigue. Also, the results of several studies have suggested that

high intensity activity of a few seconds or a few minutes in duration is impaired if blood lactate levels are elevated past 4-6 mmol/L prior to exercise (Jacobs, 1986).

SUMMARY BY SECTIONS

Intensity of Exercise Response:.

Blood lactate concentration will increase in a curvilinear fashion with exercise intensity (VO_2). Heart rate will increase linearly with VO_2. Blood lactate concentration will increase due to the formation of energy (lactate being the end-product of anaerobic glycolysis). The increase in heart rate reflects the adaptation of the circulatory system which strives to bring oxygen to the muscle while also, through an increased blood flow to the working muscles, washing out unwanted end-products. This is to maintain the internal equilibrium of the muscle cell.

Regulation of Heart Rate During Exercise and Recovery.

The heart rate is regulated very precisely by the actions of the parasympathetic and sympathetic systems which act on the SA node and the conducting system of the heart. Epinephrine which is liberated by the adrenal medulla also increases the heart rate.

The cardiovascular and respiratory responses are mediated through central command, humoral drives and reflexes originating in the muscle. Factors being able to initiate the reflexes are: 1) mechanical, 2) $[K^+]$, 3) $[H^+]$, 4) osmolality, 5) [lactate], 6) [Pi], and 7) thermal stimuli. However, some controversy remains about the major candidates. The reflex responses are mediated by group III and IV afferents. The mechanism that controls the return of heart rate to its baseline value is still unknown.

Heart Rate as an Indicator of Exercise Intensity and Recovery.

As mentioned previously, heart rate increases linearly with VO_2, thus making it a good physiological parameter to monitor intensity of work. Also, the higher the intensity of work, the higher the average recovery. For a given workload, the more fit the individual, the lower the average submaximal heart rate.

Heart rate recovery curves give some information in relation to the fitness level of the athletes; however, the slow phase of the recovery may not be linked to lactate recovery as previously theorized.

Regulation of Blood Lactate During Exercise and Recovery.

ATP production

ATP is the energy source for muscle contraction. ATP can be replenished from several sources: CP (the fastest source), glycogen , glucose, and triglycerides (intramuscular and in the adipocytes). Protein catabolism is of minimal significance when compared to the energy supply of the other fuels.

Lactate Production

The concentration of NAD^+ may be the single most important factor involved in regulating the production of lactate. With oxygen, through the operation of the Krebs Cycle and the electron transport chain NAD^+ is provided via the malate shuttle (mostly in ST) and the glycerol phosphate shuttle (in FT). The activity of some of the enzymes involved in theses shuttles can be increased with exercise.

If the exercise is too intense, NAD^+ is provided through a reaction in which pyruvate is reduced to lactic acid. The rate of glycolysis is under dual control: 1)intracellular metabolic control and 2)hormonal control.

Lactate in blood

Lactic acid is ionized in the body and permeates across the cell membrane in the form of lactate and H^+. Lactate accumulates in the muscle and diffuses into the blood. The muscle membrane seems to be the main barrier to lactate diffusion out of the muscle. An increase in the muscle and blood lactate concentration leads to a decrease in pH which may result in mechanical failure by changing the intracellular processes associated with tension development and subsequent relaxation (impaired neuromuscular transmission, failure of muscle action potentials, and impaired E-C coupling).

Muscle fibre types and lactate

Three muscle fibre types have been identified: FT which is glycolytic in nature, ST which has a high oxidative capacity, and FOG which is an intermediate between theses two (see table 2).The enzyme LDH exists under five isoenzyme forms: LDH-1 to LDH-5. LDH-1 and LDH-2 (H-LDH) resist the formation of lactate and facilitate the reformation of pyruvate from lactate. LDH-4 and LDH-5 (M-LDH) strongly favour the formation of lactate. M-LDH is relatively more abundant in FT while ST has a predominance of H-LDH. The FT:ST ratio, for most muscle, varies genetically among individuals. More lactate accumulates in FT than in ST. Enzyme capacity increases with endurance training and is more highly correlated with long submaximal work than with maximal aerobic power.

Lactate removal

Lactate may be removed from blood by skeletal muscle, the liver, the heart, the kidneys, and the brain (minimal in the brain and the kidneys). Oxidation in the skeletal muscle is the main site of lactate removal during and following exercise. The rate of lactate removal by muscle is increased up to about 60-70% $VO_{2\ max}$. Only half of the lactate formed in a working muscle bed is liberated in the venous

circulation. Approximately half is combusted in the muscle bed and appears as CO_2 in the venous blood. A muscle lactate shuttle system has been suggested which consists of the transport of lactate from the FT fibres to the more oxidative ST and FOG fibres.

Lactic Acid as an Indicator of Exercise Intensity and Recovery.

Lactic acid which increases curvilinearly with exercise intensity (VO_2) is used to monitor exercise intensity. Training has been found to improve lactate recovery during exercise but not at rest. Studies are needed that look at lactate recovery speed.

Relationship Between Heart Rate and Lactic Acid.

Lactate accumulation and a drop in pH increase the heart rate. However, the recovery of lactate does not follow the heart rate recovery. The possibility of [lac] affecting heart rate recovery from exercise has not been ruled out.

The Influence of Exercise Specificity on Heart Rate and Lactic Acid Responses.

The responses of heart rate and lactate will vary in relation to the specificity of the exercise movement to training and in relation to the specificity of exercise intensity to training intensity.

Physiology of Badminton Performance.

Badminton is an intermittent sport in which, at an international caliber, bursts of work of 10 seconds on average are separated by rest periods lasting also 10 seconds on average. International caliber badminton players attain near maximal values for HR (90%-100% of max. HR). The mean $VO_{2\ max}$ of international players was respectively 59.4 ml/kg/min and 52.9 ml/kg/min for men and women. The preferred leg of badminton players had larger ST and FOG fibres and a better capillary supply to these fibres.

Since myoglobin concentration in muscle does not increase with training and since the longer the rally, the higher is the lactate concentration in blood, badminton is both an anaerobic and aerobic sport.

Badminton is a sport requiring high heart rates due to repeated high speed requirement. Low lactate accumulation is also required to prevent fatigue and a reduction in speed. Thus, it seems important to know how heart rate and lactate recovery are related, how the relation varies between men and women, and how the relation varies in time.

METHODOLOGY

INTRODUCTION

This chapter will outline the subject population, exercise protocol, analysis and assay of blood lactate and the statistical design used in this study.

SUBJECTS

The subjects in this study were all members of the Canadian National badminton team. These included male and female players, all between the ages of 18 and 30 years of age. The data for this study were accumulated over a period of two years. The players train year round and were tested three times a year. The results of the first testing session for every player were used for this study. All players had been previously required to give their informed consent upon accepting national team status through the respective sport-governing association.

GENERAL EXPERIMENTAL PROCEDURES

Exercise Protocol.

The procedure followed was the 4-corner badminton fitness test (Reed, 1981). Following explanation of the testing procedures the subject was thoroughly warmed-up through general exercises and half-speed practice of the test pattern (see Figure 1, p. 63). The subject then waited in the center of the court for the 'go' signal. At this signal the subject started and went through the pattern as quickly as possible. The time to completion of one pattern (one trial) was timed by stopwatch and was approximatively 10 seconds.

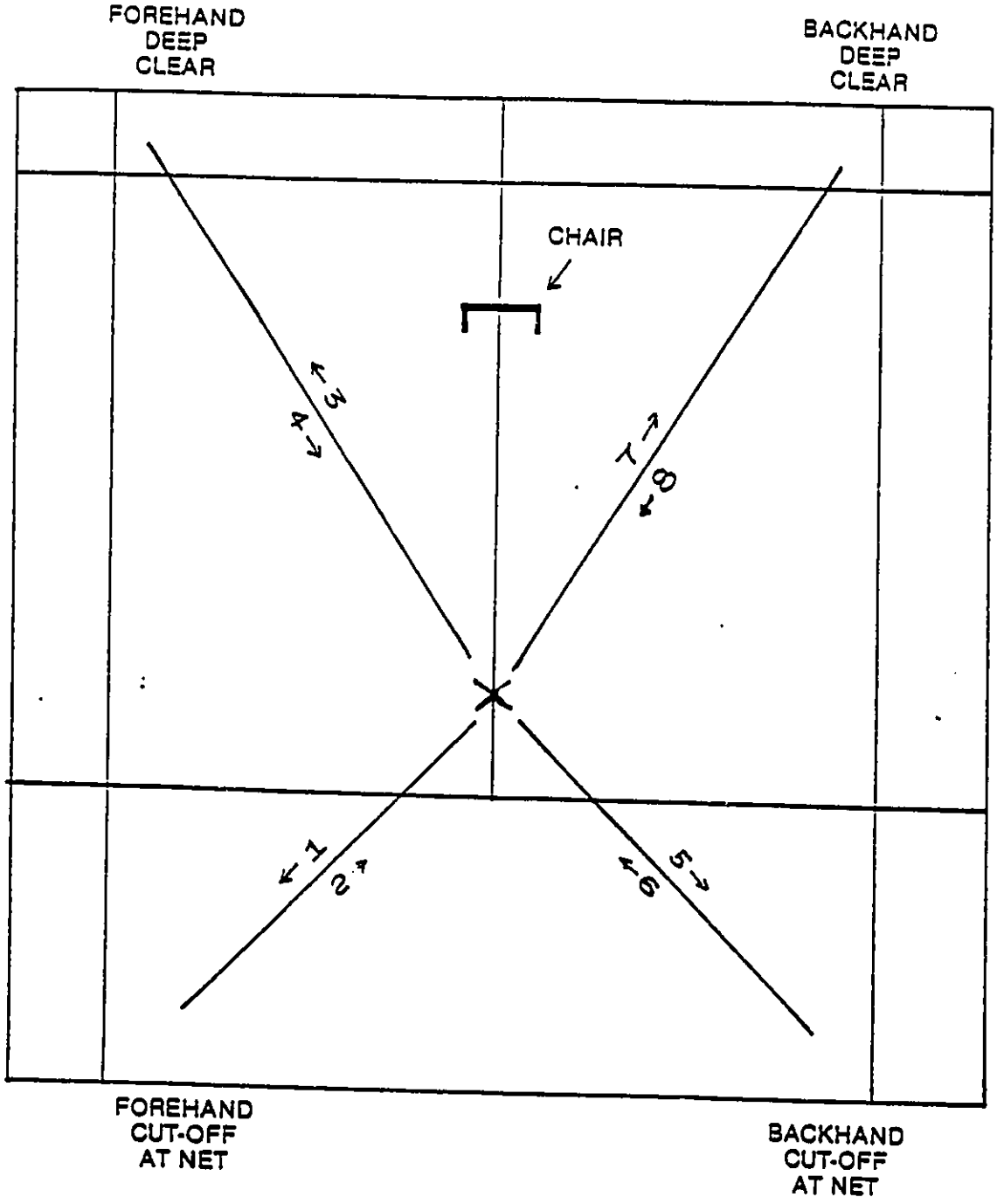


Figure 1. 4 CORNER TEST PATTERN. From *A manual on the training of badminton players: Level III Provincial Coach.* (p. 30) by A. Reed, 1981, Ottawa: Canadian Badminton Association.

The subjects performed one repeat every 20 seconds and had to perform a total of six repeats. Following this, two physiological recovery variables were measured: a 5 minute heart rate recovery period (HRR), from post 0 min. to post 5 min. every 30 sec. for the first 2 minutes and then every minute, and a 5 min. blood lactate recovery period (LaR), from post 1 min. to post 5 min. every minute.

Monitoring of Heart Rate.

Before the beginning of the test, a Sport Tester™ PE-3000 is fitted to the player in order to monitor the heart rate recovery following the test. The Sport Tester™ PE-3000 (Polar Electro Co.) heart rate monitor is based on the detection of the ECG-signal of the heart that is transmitted from an electrode belt to a microelectronic receiver which gives a digital readout in bpm. The receiver was worn on the wrist of the non-racquet hand.

Immediately following the last trial, the test administrator started the recovery clock and simultaneously recorded the displayed heart rate of the player. The heart rate was then noted from the receiver display and recorded every 30 seconds for the first two minutes and then every minute for three more minutes.

Analysis of Blood Lactate.

At the end of the 6th trial, the person taking the blood samples started a recovery clock. The player walked over from the badminton court and stood beside the person taking the blood samples. The fingertip was cleaned with an alcohol swab and allowed to dry before lancing for each sample to avoid contamination of the sample. A sample of blood was then drawn by capillary action into a heparinized tube and exactly 20 μl of blood were then transferred from the capillary tube and immediately haemolysed with 380 μl of diluting solution which contains 1g/l sodium azide (Kontron Medical.

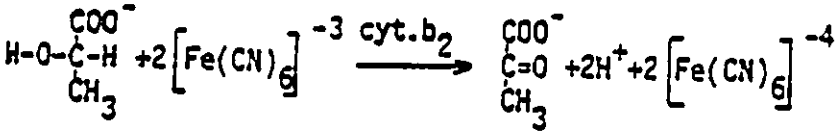
1981). Thus the total volume per tube was 400 µl. The tubes were then closed with a cap and parafilm to ensure an airtight seal. All samples were analyzed within a 48-hour time period which had been previously shown (Kontron Medical, 1981) to be stable for lactic acid determination. The samples were taken every minute for a period of five minutes. This is the maximum amount of rest time that the players are allowed between the 2nd and 3rd games of a match.

Lactate Assay.

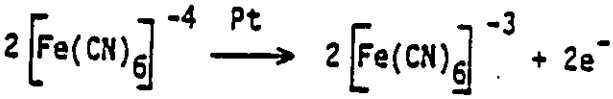
Blood lactate concentration was determined within 48 hours of sampling time using a Kontron Medical 640 Lactate Analyzer™ (Roche Bio-Electronics).

This method involves:

a) the enzymatic oxidizatiou of lactate to pyruvate in the presence of cytochrome b2:



and b) the electrochemical oxidation of the hexocyanoferrate II formed:



The two electrons released for each lactate molecule oxidized cause an electrical current to develop. The linear relationship between the current developed and the lactate concentration is achieved by first order kinetics of the enzymatic reaction. Thus, a current proportional to the lactate concentration is measured and the lactate concentration displayed in mmol/l on the readout.

The lactate analyzer is calibrated before each subject with a 5.00 mmol/l standard solution and the precision of the measurement (SD) is +/- 0.2 mmol/l or +/- 5% of the reading (whichever is greater) (Kontron Medical, 1981).

STATISTICAL ANALYSES

Descriptive.

Initially, descriptive analyses were performed to provide means and standard deviations for all the heart rate and lactate values obtained during recovery, including the peak values. The mean recovery curves for heart rate and lactate were also plotted over time. An index was generated which looked at the ratio HR:[lactate] for peak and recovery values. In order to determine the relationship between heart rate recovery and lactate recovery, a correlational matrix of heart rate and lactate values over time was used. This was performed on each of the male, female and pooled data.

ANOVA.

A two way analysis of variance (ANOVA) was used to analyze the collected data. The gender and time of recovery were the two independent variables in the analysis while heart rate, blood [lactate] and the HR/[lactate] ratio served as the dependent variables.

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