THE RELATIONSHIP BETWEEN MAXIMAL AEROBIC POWER AND THE RECOVERY FROM MAXIMAL INTERMITTENT ANAEROBIC EXERCISE

by

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We accept this thesis as conforming to the required standard

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ABSTRACT

The purpose of this study was to examine the relationship between VO2 max, maximal cardiac output (Q) and arterial venous oxygen difference (a-vO2), and the recovery from maximal intensity intermittent anaerobic exercise. Twenty male university level rugby (n=16) and soccer (n=4) players volunteered for the study. Mean (standard deviation) age, mass and VO2 max was 21.9 (1.8) years, 84.7 (12.7) kg and 52.7 (6.9) $mL \bullet kg^{-1} \bullet min^{-1}$ respectively. The exercise protocols, separated by 36-48 hours, consisted of six 15s (90s active recovery) and ten 6s (30s active recovery) maximal intensity sprints on a Monarch friction braked cycle ergometer. In the 6 x 15s protocol, significant relationships were obtained between VO2 max ($mL \bullet kg^{-1} \bullet min^{-1}$) and percent drop-off in mean power in bouts 5 and 6 compared to bout 1 ($MPDO15_{5,6}$) and bout 6 compared to bout 1 ($MPDO15_6$) of r = -.49 (p = 0.03) and r = -.63 (p = 0.004) respectively. Correlations of r = -.62 (p = 0.002) and r = -.63 (p = 0.002) were obtained between VO2 max ($mL \bullet kg^{-1} \bullet min^{-1}$) and percent drop-off in peak power in bouts 5 and 6 compared to bout 1 ($PPDO15_{5,6}$) and percent drop-off in bout 6 compared to bout 1 ($PPDO15_6$). Percent drop-off in mean power during bouts 8, 9 and 10 compared to bout 1 of the 10 x 6s protocol ($MPDO6_{8,9,10}$) demonstrated a weaker relationship with VO2 max (r = -.2, p = 0.4).

Significant correlations were obtained between a-vO2 and $MPDO15_{5,6}$ ($r = -.54$, $p = 0.02$) and $MPDO15_6$ ($r = -.57$, $p = 0.01$). Percent oxygen consumption, when compared to the first, in the second ($VO2_{30-60}$), third ($VO2_{60-90}$), fourth ($VO2_{90-120}$) and fifth ($VO2_{120-150}$) 30s time period of recovery following the two protocols was also calculated. Correlations between VO2 max ($mL \bullet kg^{-1} \bullet min^{-1}$) and $VO2_{30-60}$ ($r = .51$, $p = 0.03$), $VO2_{60-90}$ ($r = .44$, $p = 0.06$), $VO2_{90-120}$ ($r = .42$, $p = 0.08$) and $VO2_{120-150}$ ($r = .47$, $p = 0.04$) as well as between a-vO2 and $VO2_{30-60}$ ($r = .38$, $p = 0.11$), $VO2_{60-90}$ ($r = .49$, $p = 0.03$), $VO2_{90-120}$ ($r = .64$, $p = 0.003$) and $VO2_{120-150}$ ($r = .6$, $p = 0.007$) were also obtained. The results suggest that maximal aerobic power, particularly the peripheral component, is an important determinate of the ability to recover from maximal anaerobic exercise and that an elevated creatine phosphate resynthesis rate is at least partly responsible.

Examiners:

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INTRODUCTION

Many sports, such as hockey and the various forms of football, demand intermittent bursts of maximal intensity exercise (Reilly, Secher, Snell & Williams, 1990). Although creatine phosphate (CP) is essentially depleted within 10 seconds following the onset of exercise (Jones, Heigenhauser, Kuksis, Matsos, Sutton & Toews, 1985), it is the most immediate reserve skeletal muscle has for the resynthesis of Adenosine Triphosphate (ATP), and so will be important in maintaining power output during exercise of this nature. The fact that muscle and blood lactate concentrations are significantly elevated following 6 (Boobis, Williams, & Wooten, 1982) and 10 (Jacobs, Tesch, Bar-Or, Karlsson & Dotan, 1983) seconds of maximal intensity work, implies that glycolysis also represents an important source of ATP resynthesis in short duration exercise. However, fatigue characteristics can be stimulated by lowering muscle pH (Curtin & Edman, 1989), performance is enhanced by the ingestion of sodium bicarbonate (Jones, Sutton, Taylor & Toews, 1977; Maclaren & Morgan, 1985) and the rate and amount of tension development decreased with an associated pH decrease in skinned muscle fibres (Fabiato & Fabiato, 1978). These findings indicate that the drop in muscle pH accompanying lactate formation may have a detrimental effect on subsequent performance. Although the exact mechanism for this effect remains obscure (Fitz & Metzger, 1993), a number of possibilities exist. As hydrogen ions (H^+) are produced when ATP undergoes hydrolysis during contraction, an increased muscle H^+

concentration resulting from the dissociation of lactic acid may inhibit Adenosine Triphosphatase (Donaldson, 1983), thus lowering power output. A decreased calcium release by the sarcoplasmic reticulum (Nakamura & Schwartz, 1970), as well as an inhibition of glycolytic enzymes such as phosphofructokinase (Danforth, 1965) and lactate dehydrogenase (Sjodin, 1976), represent other possible avenues through which an elevated hydrogen ion concentration may inhibit power development.

In order to maintain the intensity of subsequent performance, the return of exercising muscle to a state as closely nearing homeostatic pH as possible will be an important component of the recovery phase of intermittent exercise. An enhanced aerobic system has been suggested as facilitating this recovery (Rhodes & Twist, 1990). The oxidation to pyruvate and its subsequent passage through the Kreb cycle is a major fate of lactate (Mazzeo, Brooks, Schoeller & Bidinger, 1986; Donavan & Brooks, 1983), and as H^+ are consumed on an equimolar basis (Gladden & Yates, 1993), this process represents an important mechanism for the return of skeletal muscle towards homeostatic pH. The increased activity of type H lactate dehydrogenase (Apple & Rogers, 1986; Sjodin, 1976), increased mitochondrial content (Gollnick & King, 1969) and increased oxidative enzymatic activity (Gollnick, Armstrong, Saubert, Piehl & Saltin, 1972) shown to accompany endurance training should be advantageous in this process. In addition, an increased muscle to blood exchange surface resulting from a training induced increase in capillary density (Andersson, 1975; Andersson & Henriksson, 1977), may be beneficial in facilitating the movement of lactate from the muscle. A

significant correlation between blood lactate concentration and capillary density (Tesch & Wright, 1983) is consistent with this observation. Because creatine phosphate resynthesis is dependent upon the availability of oxygen (Sahlin, Harris & Hultman, 1979), the decreased blood to muscle diffusion distance resulting from the increased capillarization may also facilitate the resynthesis of CP by enhancing the delivery of oxygen. In addition, increased blood volume resulting from endurance training (Saltin, Blomquist, Mitchel, Johnson, Wildethal & Chapman, 1968) may also aid recovery by enhancing the athlete's buffering capacity.

Despite the implications for both competitive performance and the maintenance of training intensity, the literature contains no solid evidence demonstrating a relationship between aerobic power and anaerobic recovery. The few studies that have employed an intermittent exercise protocol in pursuit of an answer to this question (Gaiga & Docherty 1995; Schreiner, 1988) are inconclusive. Gaiga & Docherty (1995) reported performance improvements over a series of 30 second maximal effort Wingate tests on a cycle ergometer following aerobic interval training. Because of significant training effect upon the anaerobic system making these findings difficult to interpret, the results support future research in this area. For example, while the aerobically trained group demonstrated a significantly elevated total work output in all four bouts, the greatest improvement in this variable existed in the later stages of the intermittent protocol. This observation could be expected if the enhanced aerobic capabilities had enabled a faster

recovery from previous bouts, therefore facilitating the maintenance of performance.

Schreiner (1988) concluded that blood and muscle lactate accumulation and removal were not significantly different between high and low aerobic power groups. However, a mean difference in $\dot{V}O2$ max of 3.7 $mL \bullet kg^{-1} \bullet min^{-1}$ between groups may not have been sufficient to demonstrate a relationship. In addition, as the high aerobic power group had a mean $\dot{V}O2$ max of 54.7 $mL \bullet kg^{-1} \bullet min^{-1}$, well below that of elite players participating in intermittent sports such as soccer (Withers, Roberts & Davies, 1977), it is possible that even this group did not possess a suitable degree of adaptation to demonstrate a relationship. The high aerobic power group also had a significantly greater power output, making the results even more inconclusive. Although unable to document the effect on subsequent performance, a number of studies have examined the relationship between maximal aerobic power and metabolic recovery following a single bout of exercise (Evans & Cureton, 1983; Oyono-Enguelle et al. 1990; McCulley, Vandenbourne, De Meirleir, Posner & Leigh, 1992; Petersen & Cooke, 1994; Yoshida & Watari, 1993; Tesch, Thorsson & Fujitsuka, 1989). McCulley et al. (1992) reported a faster resynthesis of CP in middle distance runners than sprinters and controls and suggested that CP resynthesis represents a useful measure of oxidative capacity. This proposal is supported by Yoshida & Watari (1993) and Tesch et al. (1989). In contrast, Petersen & Cooke (1993) observed a similar time course of CP resynthesis in endurance athletes and a group of sedentary subjects. The endurance athletes did however demonstrate a significantly faster rate of return towards homeostatic

muscle pH. While Yoshida & Watari (1993) failed to report intracellular pH, the protocol employed by McCulley et al. (1992) was unsuccessful in shifting muscle pH from homeostatic values. These results would support the suggestion that this component may have an effect on CP resynthesis.

The importance of an efficient aerobic system in facilitating the maintenance of intermittent anaerobic performance is an important issue to resolve as it has implications for many sports in both training and competition. It would also appear beneficial to establish the relative importance of the central and peripheral components of aerobic power in this recovery process as different strategies have been suggested for the training of each (MacDougall & Sale, 1981).

Consequently, it is the purpose of this study to investigate the following research questions:

1) Is maximal aerobic power related to the recovery from anaerobic exercise?

2)If so, are the important adaptations related to this recovery from anaerobic exercise, related to the central or peripheral component?

METHODOLOGY

SUBJECTS

Following approval of the study by the University Ethics Review Committee for research involving human subjects, twenty university level rugby (n=16) and soccer (n=4) players volunteered as subjects. Mean (standard deviation) age and body mass were 21.9 (1.8) years and 84.7 (12.7) kg respectively. Prior to testing, all subjects were given verbal and written information regarding the requirements and testing protocols, before written informed consent was obtained (Appendix 1).

Table 1 - Physiological characteristics of subjects (n=20)

	Mean (SD)	Range
Age (years)	21.9 (1.8)	18 - 24
Body Mass (kg)	84.7 (12.7)	64.6 - 111.1
Sum of 8 Skinfolds (mm)	85.3 (37.8)	41.0 - 160.7
$\dot{V}O_2$ max ($mL \cdot kg^{-1} \cdot min^{-1}$)	52.7 (6.9)	38.4 - 63.8
$\dot{V}O_2$ max ($L \cdot min^{-1}$)	4.4 (.47)	3.5 - 5.3

$\dot{V}O2$ max (standard deviation) for soccer players (n=4) and rugby players (n=16) was 61.1 (3.0) and 50.7 (5.9) $mL \bullet kg^{-1} \bullet min^{-1}$ respectively.

TESTING

Subjects visited the University of Victoria Sport and Fitness Centre on four different occasions. Measures for $\dot{V}O_2$ max, body composition and body mass were obtained on day 1. On day 3, all subjects completed intermittent power test 1 followed by intermittent power test 2 on day 5. On these two days a record of recovery oxygen kinetics was also obtained. On the final visit to the laboratory, which took place on day 8 of the data collection period, subjects completed a submaximal cardiac output test (Jones, 1988).

SUM OF SKINFOLDS

The same investigator recorded skinfold measurements for the biceps, triceps, subscapular, supra iliac, abdominals, supraspinale, thigh and calf for each subject.

MAXIMAL AEROBIC POWER

Maximal aerobic power was measured using a single continuous protocol on a Monarch cycle ergometer as described by Thoden (1991), in which subjects attempted to maintain a pedaling rate of 80 revs per minute. Initial resistance was 1.5 kiloponds (kp) and was increased by .5 kp every two minutes, until 3 kp, then resistance was increased every minute. $\dot{V}O_2$ max was considered to have been achieved when two or

more of the following criteria had been met: (a) a plateau or decline in oxygen consumption despite an increase in power output, (b) a respiratory exchange ratio which exceeded 1.15, (c) the achievement of predicted maximum heart rate, or (d) volitional fatigue. All subjects met this condition, with all but 4 satisfying at least 3 of these criteria.

During the test expired gases were collected by a two-way Rudolph valve and analysed every 30 seconds by a Horizon Metabolic Cart (MMC). The cart was calibrated before each test using primary standard gases.

CARDIAC OUTPUT

Cardiac output ($\dot{Q}$) was determined during steady state cycling at a workload corresponding to approximately 60% $\dot{V}O_2$ max. This load was applicable as stroke volume fails to demonstrate further increases with an associated rise in workload in excess of approximately 40% $\dot{V}O_2$ max (Åstrand & Rodahl, 1986; Coyle, Martin, Sinacone, Joyner, Hagberg & Holloszy, 1984).

Steady state was considered to have been reached when $\dot{V}E$ (±1.0 $L \cdot min^{-1}$) and $\dot{V}O_2$ (±0.1 $L \cdot min^{-1}$) remained constant and an end tidal CO_2 ($P_{ET}CO_2$) was achieved which was equal or less than the previous reading. At this point the re-breathing procedure was implemented which involved the subject hyperventilating for 20 seconds from either a 5L or 7L bag containing a mixture of 11 - 14 % CO_2 and O_2 balance. An extended description of this technique, as well as the table of $P_{ET}CO_2$ and $\dot{V}O_2$ ($L \cdot min^{-1}$) values used to determine the exact concentration of CO_2 for each individual subject, has been reported elsewhere (Jones,1988).

The advance exercise testing program for use with the Horizon MMC was used to calculate $\dot{Q}$ by applying the collected data to the indirect Fick equation (Jones, 1988), where

$$\dot{Q} = \frac{\dot{V}CO_2}{C_vCO_2 - C_aCO_2}$$

$\dot{V}CO_2$ is the expired CO_2 content ($L \cdot min^{-1}$ STPD), measured during steady state prior to the re-breathing process, and C_vCO_2 and C_aCO_2 represent the content of CO_2 in mixed venous and arterial blood respectively. End tidal fractional percentage CO_2 ($FECO_2$) was measured by a rapid gas analyser to give end tidal partial pressure of CO_2 ($P_{ET}CO_2$) and the assumption made that this value represented the partial pressure of carbon dioxide in arterial blood ($PaCO_2$)(Jones, 1988).

As gas from the re-breathing bag mixed with the alveolar gas, the PCO_2 plateaued indicating that the pressure of alveolar CO_2 (P_aCO_2) was equal to P_vCO_2.

P_aCO_2 and P_vCO_2 were then converted to C_aCO_2 and C_vCO_2 using the equation:

$$\log e\, CCO_2 = .396 \log e\, PCO_2 + 2.38 \qquad \text{(Jones, 1988)}$$

The determination of $\dot{Q}$ at a steady state submaximal workload and heart rate, using the procedure described above, allowed stroke volume to be calculated. In turn, stroke volume, maximum heart rate and maximal aerobic power were applied to the equation

$$\dot{V}O_2 = SV \times HR \times (a - vO_2)$$

to determine the relative contributions of the peripheral and central components to each subjects $\dot{V}O_2$ max.

Marks, Katch, Rochini, Beekman and Rosenthal (1985), in a review on the validity of cardiac output measured by CO2 rebreathing (Equilibration Method), cited a correlation range of .86 - .96 when compared to the direct Fick Method. Using the same method, Bhambhani, Norris & Bell (1994) reported a reliability coefficient of r=.89.

INTERMITTENT POWER TESTS

Each subject performed two maximal intensity intermittent tests. Both were conducted on the same Monarch friction braked cycle ergometer (model 868), with resistance set at 90 $g \bullet kg^{-1}$ of body mass. Both tests were proceeded by an identical warm-up consisting of 2 minutes continuous pedaling at a load corresponding to 35% $\dot{V}O_2$ max followed by two all out bursts at the testing load for approximately 2 seconds. These two periods of increased power output occurred within 30 seconds and were followed by another minute of continuous pedaling at 35% $\dot{V}O_2$ max before the test began.

To standardise the acceleration period at the beginning of each bout, the cue "pick it up ready and go" were always used with the call "go" corresponding to both the application of the resistance and the start of the clock. The first test involved 10 maximal intensity bouts of 6 seconds duration, separated by 30 seconds of active recovery at a load (60 RPM) which corresponded to 35% $\dot{V}O_2$ max. Flywheel rotations were recorded

each second by an electronic revolution counter in order to obtain the mean power output (MP6), defined as the average power output over the 6 second period. Values obtained for MP6 in bouts 8, 9 and 10 were each compared to bout 1 in order to gain a percent dropoff in MP6 from that achieved in bout 1. The average of the 3 percent drop off values were then calculated to gain an average percent dropoff in MP6 over bouts 8, 9 and 10 compared to bout 1 ($MPDO6_{8,9,10}$). This value constituted the dependant variable used in statistical analysis for this protocol.

In the second intermittent test, subjects performed maximally 6 bouts of 15 seconds cycling separated by 90 seconds of active recovery. Both the test and recovery phase involved identical workloads to that already stipulated for the 10 x 6 second test. Similarly flywheel rotations were recorded each second and used to obtain (a) mean power output (MP15), defined as the average power output over the 15 second period; and (b) the peak power output (PP15), defined as the average power output over the first three seconds of the 15 second period. These values were then used to obtain the percent drop off in MP15 and PP15 between bouts 6 compared to bout 1 ($MPDO15_6$ and $PPDO15_6$ respectively), as well as the average percent drop off in MP15 and PP15 of bouts 5 and 6 compared to bout 1, termed $MPDO15_{5,6}$ and $PPDO15_{5,6}$ respectively.

In addition, during the last recovery period of both protocols, and immediately before the final bout, a mouthpiece was inserted which was connected to a Beckman MMC. Using manual mode, oxygen consumption in 5 second time frames was recorded for a period of 5 minutes immediately following the final bout. During this period

subjects were completely stationary and sitting on the cycle ergometer. This data was used to calculate oxygen consumption (VO2) in the first 30 seconds of recovery and subsequent 30 second time frames through to the end of the 5 minute recovery period. Consequently, VO2 during the second (30-60), third (60-90), fourth (90-120) and fifth (120-150) thirty second time frames were compared to the first (0-30 seconds), resulting in a percent drop off for each, termed $VO2_{30\text{-}60}$, $VO2_{60\text{-}90}$, $VO2_{90\text{-}120}$, and $VO2_{120\text{-}150}$ respectively.

STATISTICAL PROCEDURES

To determine the relationship between variables, Pearson correlation co-efficients were calculated using Systat Faststat (Version 2.0). High and low aerobic power ($mL \bullet kg^{-1} \bullet min^{-1}$), arterial venous oxygen difference (a-vO2 ($mL \bullet L^{-1}$)) and $\dot{Q}$ ($L \bullet min^{-1}$) groups were formed containing the six highest and six lowest values for each variable respectively. This grouping method meant that the subject composition of each group was dependant on the variable concerned. Differences between group means were obtained using an independent t-test.

RESULTS

Figures 1 through 3 illustrate the drop-off in average mean power and average peak power between bouts for both the 10 x 6 second and 6 x 15 second protocols. Raw data is contained in Appendix 2.

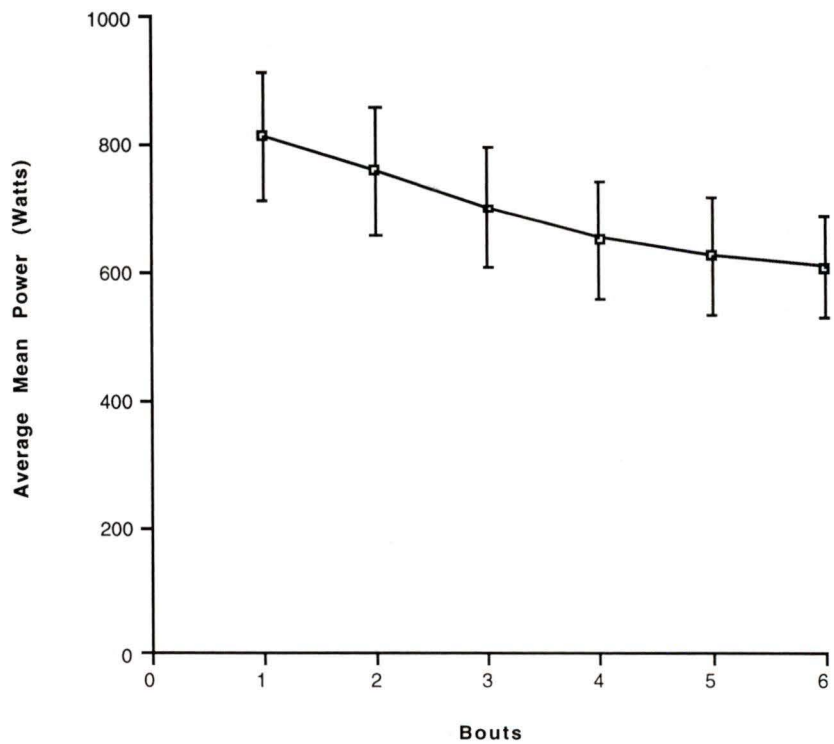

Figure 1 - Average Mean Power (Watts) for 6 x 15 second protocol.

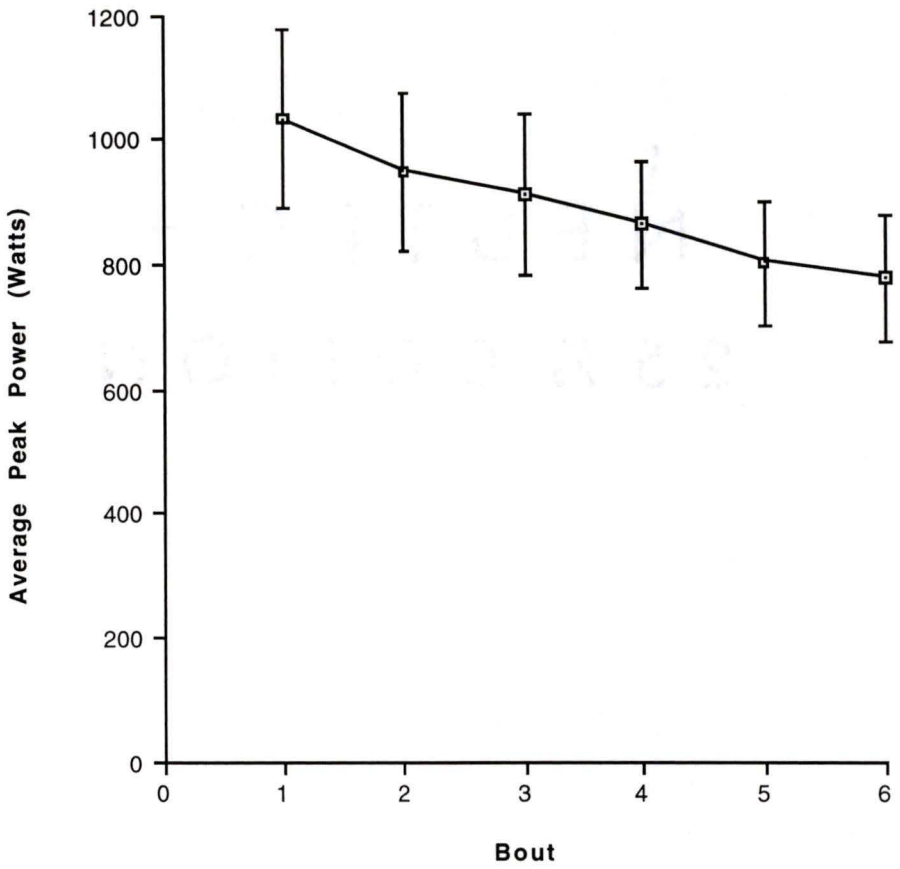

Figure 2 - Average Peak Power (Watts) for 6 x 15 second protocol

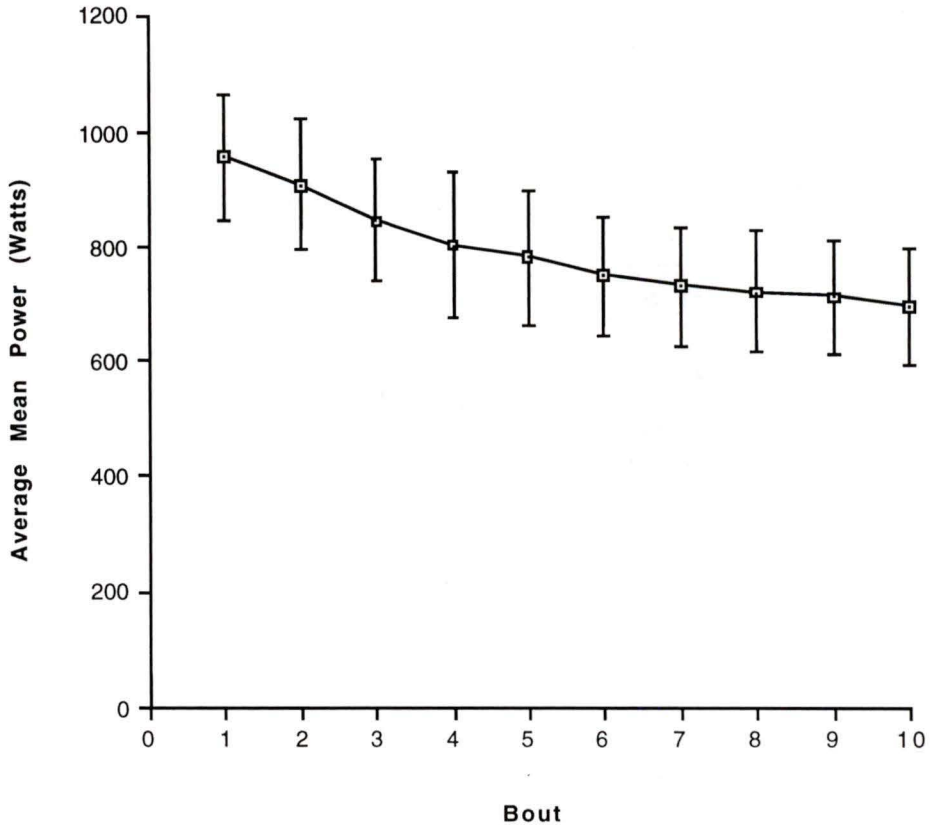

Figure 3 - Average Mean Power (Watts) for 10 x 6 second protocol.

1. What is the relationship between maximal aerobic power and recovery from anaerobic exercise?

Table 2 - Relationship between $\dot{V}O2$ max ($mL \cdot kg^{-1} \cdot min^{-1}$) and $MPDO15_6$, $MP15DO_{5,6}$, $MPDO6_{8,9,10}$, $PPDO15_6$ and $PPDO15_{5,6}$ (% DO).

	r	p
$MPDO15_6$	- .63	.004
$MPDO15_{5,6}$	- .49	.03
$MPDO6_{8,9,10}$	- .2	.4
$PPDO15_6$	- .63	.004
$PPDO15_{5,6}$	- .62	.002

All dependent variables from the 6 x 15 second protocol correlated with $\dot{V}O2$ max ($mL \cdot kg^{-1} \cdot min^{-1}$) to the $p < 0.05$ level (table 2). The relationship between $\dot{V}O2$ max ($mL \cdot kg^{-1} \cdot min^{-1}$) and $MPDO6_{8,9,10}$ was not as strong (table 2).

The correlation between these five variables ($MPDO15_6$, $MPDO15_{5,6}$, $MPDO6_{8,9,10}$, $PPDO15_6$ and $PPDO15_{56}$) and the absolute measure of $\dot{V}O2$ max ($L \cdot min^{-1}$) was r = -.38 (p = 0.1), r = -.4 (p = 0.09), r = -.41 (p = 0.07), r = -.16 (p = 0.38) and r = -.16 (p = 0.51) respectively.

The high aerobic power (HAP) group had a mean $\dot{V}O2$ of 60.0 (2.8) $mL \cdot kg^{-1} \cdot min^{-1}$ compared to 44.7 (4.5) $mL \cdot kg^{-1} \cdot min^{-1}$ for the low aerobic power (LAP) group ($p < 0.001$).

Table 3 - Group means (SD) for $MPDO15_6$, $MPDO15_{5,6}$, $MPDO6_{8,9,10}$, $PPDO15_6$ and $PPDO15_{5,6}$ (%DO) for HAP and LAP groups.

	GROUP MEANS (SD)		p
	HAP (%DO)	LAP (%DO)	
$MPDO15_6$	20.4 (3.1)	28.6 (5.0)	.01
$MPDO15_{5,6}$	20.9 (4.1)	26.5 (4.9)	.06
$MPDO6_{8,9,10}$	22.9 (8.4)	27.9 (3.6)	.22
$PPDO15_6$	18.3 (2.5)	32.1 (7.1)	.004
$PP15DO_{5,6}$	18.8 (4.3)	29.6 (4.5)	.002

The HAP group had lower percent drop-offs than the LAP group in both mean and peak power scores during the 15 second intervals (table 3). This difference was less substantial for the 6 second interval test (table 3).

A correlation of r = .51 (p = 0.03) was obtained between $\dot{V}O2$ max ($mL \bullet kg^{-1} \bullet min^{-1}$) and the percent drop in oxygen consumption during the second 30 seconds of recovery compared to the first thirty seconds of recovery ($VO2_{30\text{-}60}$). Correlation values of r = .44 (p = 0.06), r = .42 (p = 0.08) and r = .47 (p = 0.04) were obtained for the relationship between $\dot{V}O2$ max ($mL \bullet kg^{-1} \bullet min^{-}$) and $VO2_{60\text{-}90}$, $VO2_{90\text{-}120}$ and $VO2_{120\text{-}150}$ respectively.

2. What is the relationship between the peripheral component (a-vO2) of $\dot{V}O2$ max and the ability to recover from anaerobic exercise.

The peripheral component of $\dot{V}O2$ max correlated highest with $MPDO15_6$ and $MPDO15_{5,6}$ (table 4). The relationship between a-vO2 max and both $PPDO15_6$ and $PPDO15_{5,6}$ was not as strong while $MPDO6_{8,9,10}$ displayed the weakest relationship of all 5 variables (table 4).

The high a-vO2 (Ha-vO2) group had a mean a-vO2 of 187.6 (13.8) $mL \bullet L^{-1}$compared to 146.6 (12.9) $mL \bullet L^{-1}$ for the low a-vO2 (La-vO2) group ($p < 0.001$).

Correlations obtained between a-vO2 and $VO2_{30\text{-}60}$, $VO2_{60\text{-}90}$, $VO2_{90\text{-}120}$ and $VO2_{120\text{-}150}$ were r = .38 (p = 0.11), r = .49 (p = 0.03), r = .64 (p = 0.003) and r = .6 (p = 0.007) respectively.

Table 4 - Relationship between a-vO2 ($mL \cdot L^{-1}$) and $MPDO15_6$, $MPDO15_{5,6}$, $MPDO6_{8,9,10}$, $PPDO15_6$ and $PPDO15_{5,6}$. (%DO)

	r	p
$MPDO15_6$	- .57	.01
$MPDO15_{5,6}$	- .54	.02
$MPDO6_{8,9,10}$	- .06	.81
$PPDO15_6$	- .17	.49
$PPDO15_{5,6}$	- .22	.36

Table 5 - Group Means of $MPDO15_6$, $MPDO15_{5,6}$, $MPDO6_{8,9,10}$, $PPDO15_6$ and $PPDO15_{5,6}$ (%DO) for Ha-vO2 and La-vO2 groups.

	Group Means (SD)		p
	Ha-vO2 (%DO)	La-vO2 (%DO)	
MPDO1556	19.89 (2.01)	29.15 (5.52)	.008
$MPDO15_{5,6}$	19.52 (2.96)	27.8 (4.72)	.006
$MPDO6_{8,9,10}$	21.65 (7.4)	25.83 (3.47)	.25
$PPDO15_6$	17.28 (2.96)	27.43 (7.34)	.02
$PPDO15_{5,6}$	17.08 (3.78)	25.94 (6.26)	.02

3. What is the relationship between $\dot{Q}$, representing the central component of $\dot{V}O2$ max, and the ability to recover from anaerobic exercise?

$\dot{Q}$ failed to correlate with any variable from either protocol to the $p < 0.05$ level (table 6).

The high $\dot{Q}$ (HQ) group had a mean $\dot{Q}$ of 31.3 (1.6) $L \cdot min^{-1}$ compared to 22.7 (2.6) $L \cdot min^{-1}$ for the low $\dot{Q}$ (LQ) group ($p < 0.001$).

Table 6 - Relationship between $\dot{Q}$ ($L \cdot min^{-1}$) and $MPDO15_6$, $MPDO15_{5,6}$, $MPDO6_{8,9,10}$, $PPDO15_6$ and $PPDO15_{5,6}$. (%DO)

	r	p
$MPDO15_6$	- .2	.42
$MPDO15_{5,6}$	- .16	.51
$MPDO6_{8,9,10}$	- .34	.14
$PPDO15_6$	- .09	.71
$PPDO15_{5,6}$	- .02	.94

The relationships obtained between $\dot{Q}$ and $VO2_{30-60}$, $VO2_{60-90}$, $VO2_{90-120}$ and $VO2_{120-150}$ were $r = -.09$ ($p = 0.7$), $r = -.31$ ($p = 0.2$), $r = -.41$ ($p = 0.08$) and $r = -.42$ ($p = 0.07$) respectively.

Table 7 - Group means (SD) of $MPDO15_6$, $MPDO15_{5,6}$, $MPDO6_{8,9,10}$, $PPDO15_6$ and $PPDO15_{5,6}$ (%DO) for both HQ and LQ groups.

	Group Means (SD)		p
	HQ (%DO)	LQ (%DO)	
$MPDO15_6$	27.9 (5.9)	26.1 (4.7)	.57
$MP15DO_{5,6}$	26.1 (4.9)	25.2 (4.9)	.77
$MPDO6_{8,9,10}$	25.6 (3.6)	30.5 (4.9)	.06
$PPDO15_6$	25.5 (5.3)	26.8 (10.7)	.81
$PPDO15_{5,6}$	24.7 (4.7)	24.5 (7.0)	.95

DISCUSSION

The mean value for maximal aerobic power (MAP) for the rugby players in the present study (50.7 (5.9) $mL \cdot kg^{-1} \cdot min^{-1}$) was comparable with those reported elsewhere in the literature. Williams, Reid and Couts (1973) reported a mean $\dot{V}O2$ max of 50.3 (5.1) $mL \cdot kg^{-1} \cdot min^{-1}$ for a group of eleven university rugby players using a cycle ergometer, while Bell (1980) using the same technique, obtained a mean $\dot{V}O2$ max of 46.3 (8.9) $mL \cdot kg^{-1} \cdot min^{-1}$ in a group of 20 college level players. The slight discrepancy between the results reported by Bell (1980) and both Williams et al. (1973) and the present study can be explained by the fact that the subjects utilized by Bell (1980) were all forwards who tend to have a lower relative $\dot{V}O2$ max than other players at this level (Reilly, 1990), a trend supported by this study.

Mean $\dot{V}O2$ max for the soccer players (61.1 (3.0) $mL \cdot kg^{-1} \cdot min^{-1}$) was similar to that obtained for a group of top level Australian players of 62.0 $mL \cdot kg^{-1} \cdot min^{-1}$ (Withers, Roberts & Davies,1977), yet slightly lower than the 64.0 $mL \cdot kg^{-1} \cdot min^{-1}$ reported for top level Italian amateur players (Faina, Gallozzi, Lupo, Colli, Sassi & Marini, 1988).

As demonstrated by figures 1 through 3, both protocols were successful in fatiguing the subjects to the point where power output could not be maintained to the level exhibited in the previous bout. While no study was found that had used a 6 x 15 second protocol, Gaitanos, Williams, Boobis and Brooks (1993) also utilized a 10 x 6 second

protocol with 30 seconds recovery between bouts. As with subjects in the present study, the 8 male physical education students showed a consistent drop off in mean power, to the point where bout 10 (639 W) was 73% of that achieved in bout 1 (870 W). This was comparable to the present study where average mean power output in the final bout (693 W) was 71% of that obtained in bout 1 (956 W). In a similar protocol, Jenkins, Brooks & Williamson (1994) reported an 80% drop off in mean power between bouts 1 (711 W) and 10 (539 W) in a group of 6 physically active males. However a lower resistance of 0.075 $g \cdot kg^{-1}$ of body weight was used and may have been responsible for both the failure of the protocol to fatigue subjects to the same extent, as well as the lower mean power outputs.

The primary purpose of the study was to examine the relationship between MAP and the ability to recover from maximal intensity intermittent exercise. As discussed above, the literature suggests that the adaptations that accompany endurance training, such as an increased capillary density (Anderson, 1975; Anderson & Henriksson, 1977) and an increased mitochondrial content (Gollnick et al. 1972), may be beneficial in returning the contracting muscle cells towards homeostasis following exercise. Evidence exists to suggest that a faster resynthesis of creatine phosphate (Yoshida & Watari, 1993; Tesch et al. 1989; McCulley et al. 1992) and an increased ability to return intracellular pH towards homeostatic values (Oyono - Enguelle et al. 1990, Petersen & Cooke, 1994) may be the principal mechanisms by which this is possible. It was thus hypothesized that subjects with an elevated MAP would be better

able to maintain power output in subsequent bouts of short term maximal exercise.

Correlation coefficients of r = -.63 (p = 0.004) and r= - .49 (p = 0.03) for the drop off in mean power ($MPDO15_6$ and $MPDO15_{5,6}$) and r = -.63 (p = 0.004) and r = -.62 (p = 0.002) for the drop off in peak power ($PPDO15_6$ and $PPDO15_{5,6}$) during the 6 x 15 second protocol seem consistent with this hypothesis. While to the authors knowledge no other studies have examined this question using a 6 x 15 second protocol, these results lend support to the suggestions made by Gaiga and Docherty (1993). Following interval training that was successful in significantly elevating MAP, subjects demonstrated an increased power output over four 30 second bouts of maximal exercise on a cycle ergometer. While these results were inconclusive due to an obvious training effect on the anaerobic system, and thus a higher initial power output, the fact remained that in two of the performance variables measured the greatest differences were evident in the final two bouts. This led to the conclusion that the elevated MAP may have been at least partially responsible for the improved performance due to the role of aerobic adaptations in facilitating recovery. Unfortunately both Gaiga and Docherty (1993) and the present study lack the ability to determine the exact mechanism by which the adaptations associated with an elevated MAP are acting. The work of Petersen and Cooke (1994) suggests that a faster return to homeostatic pH of the muscle cell may be at least partially responsible. It was found that following 2 minutes of unilateral plantar flexion, the increase in pH during recovery was faster in a group of 7 endurance trained subjects when compared to their sedentary

counterparts. This suggestion is further supported by studies that have demonstrated a greater lactate clearance rate following endurance training (Donavan & Brooks, 1983; Stanley, Wiseneski, Gertz, Neese & Brooks, 1988) and a significant relationship between $\dot{V}O2$ max and the time course of lactate removal (Oyono-Enguelle et al. 1990).

In direct contrast, Schreiner (1988) found no difference between blood and muscle lactate accumulation or removal between a high aerobic power group ($\dot{V}02$ max > 54.6 $mL \cdot kg^{-1} \cdot min^{-1}$) and a low aerobic power group ($\dot{V}O2$ < 50.9 $mL \cdot kg^{-1} \cdot min^{-1}$) during four 30 second bouts of maximal intensity cycling. However these results appear less than conclusive as there was a significant difference in the power output between the two groups. In addition, it is questionable whether a difference of 3.7 $mL \cdot kg^{-1} \cdot min^{-1}$ between groups would constitute oxidative adaptations of a significant magnitude. It would appear from the evidence cited above that the subjects with an elevated MAP may be exhibiting a faster recovery rate of intracellular pH within exercising muscle. In the present study (6 x 15 second protocol) a 90 second active recovery separated each bout. Despite no significant difference in intracellular pH between groups upon the completion of exercise, after 90 seconds of recovery Petersen & Cooke (1994) reported a pH of 6.4 vs. 6.65 ($p < 0.05$) for sedentary vs. endurance subjects respectively. As a passive recovery was utilized, and an active recovery has been shown to more effectively eradicate exercise induced acidosis (Hermanssen & Stensvold, 1972), it is possible the effects due to oxidative adaptation may be even more pronounced in the present study. Consequently the correlations achieved between $\dot{V}O2$ max and the 6 x 15 second protocol

variables are possibly the result of a reduction in the adverse effects of an elevated H^+ concentration. As discussed above these could include an end product inhibition of the hydrolysis of ATP (Donaldson, 1983), the inhibitory effects upon glycolytic enzymes such as PFK (Maclaren, Gibson, Parry-Billings & Edwards, 1989) or a detrimental effect upon the release of calcium from the sarcoplasmic reticulum during contraction (Nakamura & Schwartz, 1970).

Irrespective of whether an elevated H^+ concentration has an adverse effect on contraction, both the literature and the results of the present study suggest that the rate of resynthesis of CP during recovery may be another avenue through which an elevated $\dot{V}O_2$ max may have contributed towards the results obtained in the 6 x 15 second protocol (Yoshida & Watari, 1993; McCulley et al. 1992; Tesch et al. 1989). The recovery of $\dot{V}O_2$ in the first few minutes after exercise has been demonstrated to closely follow the resynthesis of CP (Hultman et al. 1967; Piiper & Spiller, 1970). Obtained correlations with MAP of $r = .51$ ($p = 0.03$), $r = .44$ ($p = 0.06$), $r = .42$ ($p = 0.08$) and $r=.47$ ($p = 0.04$) for $VO_{2_{30\text{-}60}}$, $VO_{2_{60\text{-}90}}$, $VO_{2_{90\text{-}120}}$ and $VO_{2_{120\text{-}150}}$ respectively, showed subjects with an elevated MAP tended to recover towards resting $\dot{V}O_2$ faster. The results of Hultman et al. (1967) and Piiper & Spiller (1970) cited above infer that these subjects recovered towards resting CP levels faster. In agreement with this, Yoshida & Watari (1993) reported a CP half time of 25.3 seconds for a group of 5 male long distance runners compared to 37.2 seconds for 6 healthy males ($p < 0.05$). These results are supported by McCulley et al. (1992) who used 16 male track athletes and 7 controls in a successful effort to demonstrate a significant relationship ($p < 0.001$)

between preferred running event and the initial rate of CP resynthesis following 5 minutes repeated plantar flexion against resistance. Despite the fact that CP was depleted to a similar amount in all subjects (approximately 65% of resting levels), the long distance runners were reported to have a maximum rate of CP resynthesis of 64.8 $m \bullet mol \bullet min^{-1} \bullet kg^{-1}$ of muscle compared to 38.6 $m \bullet mol \bullet min^{-1} \bullet kg^{-1}$ of muscle for the control subjects. If, as the evidence discussed suggests, CP is being resynthesised faster in more aerobically adapt individuals, it would appear to have the ability to be a significant factor in producing the correlation values obtained within the present study. However as the protocol used by McCulley et al. (1992) failed to shift pH from homeostatic values, and intracellular pH upon the completion of exercise, although not reported by Yoshida & Watari (1993), is unlikely to have risen to near the same levels as those experienced by subjects involved in the present study, caution is warranted when making this transition. As the resynthesis of CP involves the release of H^+, under circumstances where the muscle cell is experiencing exercise induced acidosis, it is possible a direct product inhibition of the reaction governed by creatine kinase may result, and there are a number of studies consistent with this suggestion. In a technique similar to Yoshida & Watari (1993) and McCulley et al. (1992), Petersen & Cooke (1994) failed to show any difference in the rate of CP resynthesis between endurance and sedentary subjects. The fact that intracellular pH fell to below 6.5 for both groups may have been a significant factor. Also consistent with this proposal is the demonstration of both a slower rate of recovery of CP for intense than light or moderate exercise (McCann, Mole & Caton,

1995; Arnold, Mathews & Radda, 1984; Taylor et al. 1986) and a relationship between muscle pH and the action of creatine kinase (Sahlin, Harris & Hultman, 1979).

In relation to the present study, it is therefore possible that both an elevated lactate clearance ability and CP resynthesis rate were inter-related. Those subjects with an elevated MAP may have been able to return intracellular pH towards homeostatic values faster, thereby reducing the product inhibition on the creatine kinase reaction, thus enabling a faster CP time constant to more effectively resynthesise this vital high energy phosphate. Consequently, and in association with the reduction in other adverse effects of H^+ concentration discussed above, mean and peak power could be maintained at a higher level in the subsequent 15 second bout. It must be noted however that while a number of the correlation's achieved significance, a coefficient of r = -.6 for example still leaves 64% of the variance unaccounted for. This demonstrates that factors other than those related to an elevated aerobic power are also contributing. The ability to counter fatigue will depend not only on the characteristics of the muscle but also factors that effect muscle activation and sequencing. Part of this unexplained drop off therefore may be a result of factors such as a fatigue effect on the higher levels of the nervous system through a suppression of motor neuron excitability or a decreased central drive (Bigland - Ritchie, 1984).

If this is the case the relatively low correlation between $MPDO6_{8,9,10}$ and $\dot{V}O2$ max ($mL \bullet kg^{-1} \bullet min^{-1}$) is at first surprising. It was hypothesized that the same aerobic adaptations that would enable faster recovery from a bout of 15 second maximal exercise would also be beneficial

following a bout of 6 seconds duration. This suggestion seems reasonable given that the protocol could be expected to significantly elevate lactate and reduce CP concentrations in working muscle (Gaitanos et al. 1993). Although subjects with a higher MAP still tended to maintain mean power more effectively ($r = -.2$), the results do not appear to be significant ($p = .42$). This conclusion is supported by the similarity between group means ($MPDO6_{8,9,10}$) for the HAP group (22%) and the LAP group (27%) ($p = .22$).

Given the discussion to this point, it appears likely that if MAP is an important determinant of the recovery from maximal anaerobic exercise the ability to restore pH to resting values will be an important advantage. It is possible that a 30 second time frame for recovery was not long enough to allow the adaptations that accompany an elevated MAP to have a significant effect. As previously discussed, Petersen & Cooke (1994) demonstrated a significantly faster rate of intracellular pH recovery in endurance athletes following exercise. This did not become apparent however until 40 seconds into the recovery period. Before this time pH continued to drop despite a state of inactivity, attributed to both the H^+ release from glycolysis, and the further release of H^+ that accompanies the resynthesis of CP. As an active recovery was utilized in the present study, the time parameters for pH recovery reported by Petersen & Cooke (1994) may not be relevant. However, for the reasons cited above, it may have been unreasonable to expect a significant difference in pH restoration in the recovery time available. Again the nature of the recovery period rather than the length or number of bouts suggests another reason why MAP may have demonstrated a weaker

relationship with the maintenance of mean power in the 10 x 6 second protocol. As already indicated, the half-time for resynthesis of CP is around 30 seconds (Petersen & Cooke, 1994; Harris, Edwards, Hultman, Nordesjo, Nylind & Sahlin 1976). As this corresponds to the length of the recovery period, it would be expected that around one half of the CP used during the bout could be restored before the next bout. The difference in resynthesised CP therefore between those with hypothesised high and low restoration rates (high and low $\dot{V}O2$ max respectively) would be a lot less than in the protocol leaving 90 seconds for this task.

The significance of this difference is likely to be further challenged by the fact that as an active recovery was used, some of the ATP that would otherwise be available for CP restoration will still be required for power output. With these considerations in mind it is not surprising that a weaker relationship was obtained for the 10 x 6 second protocol.

Given that the results of the 6 x 15 second protocol suggested that MAP may be an important determinate in the ability to recover from anaerobic exercise, the question arises as to whether the adaptations responsible were centrally or peripherally located. As different training methods have been suggested for each (MacDougal & Sale, 1981), this would appear to be an important question to answer to ensure the efficiency of training for athletes involved in intermittent type sports. The relationship between the ability to maintain mean power output in the 6 x 15 second protocol ($MPDO15_6$ and $MPDO15_{5,6}$) and both a-vO2 and $\dot{V}O2$ max were similar ($r = -.57$, $p = 0.01$ and $r = -.54$, $p = 0.02$ vs. $r = -.63$, $p = 0.004$ and $r = -.49$, $p = 0.03$ respectively). This result, along with

much weaker correlations between $\dot{Q}$ and both $MPDO15_6$ (r=-.2, p=0.42) and $MPDO15_{5,6}$ (r=-.16, p=.51) suggest that the dominant adaptations influencing the ability to maintain mean power are peripherally located. The fact that the Ha-vO2 group had lower average values for $MPDO15_6$ (p = 0.008) and $MPDO15_{5,6}$ (p = 0.006) than the La-vO2 group is consistent with this conclusion.

Lower correlations between a-vO2 and both $PPDO15_6$ (r = -.17, p = .49) and $PPDO15_{5,6}$ (r = -.22, p = .36) were primarily caused by the presence of two outliers which defied explanation and prevented these variables from displaying much stronger relationships. A difference between Ha-vO2 and La-vO2 groups of 9% and 10 % in average $PPDO15_6$ (p = 0.02) and $PPDO15_{5,6}$ (p = 0.02) respectively was consistent with this hidden trend.

These results would suggest that the correlations between $\dot{V}O2$ max and 6 x 15 second variables achieved in the present study are primarily the result of oxidative adaptations at the tissue level rather that those that effect oxygen delivery. As the majority of adaptations that occur during endurance training, and were hypothesized to be advantageous in anaerobic recovery are peripherally related, this is not a surprising result. As the principal avenue of lactate removal is through oxidative metabolism (Mazzeo et al. 1986; Donavan & Brooks, 1983), a process which consumes H^+ on an equimolar basis (Gladden & Yates, 1993), the increased levels of mitochondria (Gollnick & King, 1969; Holloszy, 1967) and associated oxidative enzymatic activity (Gollnick et al. 1972) known to accompany endurance training would appear advantageous. In addition, as CP is primarily resynthesised by oxidative phosphorylation

(Pipper & Spiller, 1970; Sahlin et al. 1979), the more mitochondria and hence Krebs cycle enzymes available to the exercising muscle cell in which to resynthesise levels of this high energy phosphate the better. The fact that slow twitch fibers, known to have a greater oxidative potential (Essen, Jansson, Henriksson, Taylor & Saltin, 1975), possess a faster rate of CP resynthesis than their fast twitch counterparts (Tesch et al. 1989) is consistent with this proposal. A greater local blood flow due to increased cappillarization, known to both perfuse ST fibers (Fox, Bowers & Foss, 1988) and accompany endurance training (Anderson, 1975; Anderson & Henriksson, 1977), may also be a significant factor by allowing greater O2 extraction from the blood. While this study lacks the ability to determine which of these peripheral adaptations are primarily responsible for the more successful maintenance of power, it does allow a conclusion to be drawn. As some of the correlations achieved between a-vO2 and the drop in VO2 during the first few minutes of recovery ($VO2_{30\text{-}60}$ ($r = .38$, $p = 0.11$), $VO2_{60\text{-}90}$ ($r=.49$, $p=0.03$), $VO2_{90\text{-}120}$ ($r = .64$, $p = 0.003$) and $VO2_{120\text{-}150}$ ($r = .6$, $p = 0.007$) were amongst the highest acheived in the study, it seems plausible to suggest that peripheral adaptations were manifesting themselves in an increased CP restoration rate. It must be stressed however that recovery VO2 was only recorded following the final bout during a passive recovery and not during the active recovery utilized throughout the protocol.

In summary, results obtained for the 6 x 15 second protocol would appear to support to the importance of local adaptation in recovery from maximal intermittent exercise of this nature.

The relationship between a-vO2 and $MPDO6_{8,9,10}$ (r=-.06, p=.81 is weaker than that achieved with $\dot{V}O2$ max, and along with the difference in average values between high and low groups for this variable (4%, p=.25) suggests little relationship between the peripheral component of $\dot{V}O2$ max and performance in the 10 x 6 second protocol. This result can be explained in two ways. Firstly there exists no relationship between the maintenance of performance in a protocol of this nature and MAP, and hence there will be no relationship of any significance with either the central or peripheral component of MAP. This is probably not the case as a 10 x 6 second protocol shown to elevate blood lactate and deplete CP (Gaitanos et al. 1993) could therefore be expected to place similar demands on recovery as those required in a 6 x 15 second protocol. What seems more likely is that, as discussed earlier, the time (30 seconds) and nature (active) of the recovery was such that a significant relationship with $\dot{V}O2$ max was not achieved and thus any relationship between $\dot{Q}$ and a-vO2 as components of $\dot{V}O2$ max will be similarly weak. Correlations of r = .06 (p = 0.81) and r = .34 (p = 0.14) for $MPDO6_{8,9,10}$ with a-vO2 and $\dot{Q}$ respectively are not inconsistent with this notion.

It was also hypothesized that central adaptations possessed the potential to assist the body in returning to homeostasis and thus facilitate subsequent performance. Endurance training has been demonstrated to increase blood volume (Saltin et al. 1968) which would appear to improve the buffering capacity of sodium bicarbonate within the blood, as well as the concentration mediated diffusion of lactate from the muscle. In addition, as endurance training increases maximal $\dot{Q}$ (Ekblom & Hermansen, 1968) there would appear to exist an increased potential

for lactate transport to the liver or non exercising muscle in order to under go oxidation or glyconeogenesis. The results of the present study fail to demonstrate these possibilities as significant factors and there appears to be a number of possible explanations. Firstly that these central adaptations are assisting in the return of the exercising muscle cell towards homeostatic pH but that H^+ concentration has little or no effect on muscle contraction and hence no effect on the maintenance of power output. Considering the evidence discussed to this point this appears unlikely. The second possibility is that due to an active recovery, and hence the need to maintain a certain level of power output during recovery, conditions facilitated the oxidation of lactate as a fuel in order to maintain power output, thus relegating those mechanisms involved in lactate removal out of the cell to an insignificant status. If this is the case then these central adaptations may be proven more beneficial in a protocol involving a passive recovery where the exercising cell would rely more heavily on dissipation into the blood and subsequent buffering or eradication at other sites. The third possibility is that the removal of lactate into the blood is not entirely dependent upon simple diffusion and that the ability of the blood to absorb lactate and move it to other sites for oxidation is therefore not the limiting factor on restoring muscle pH. As the majority of lactate appears to cross the sarcolemmal membrane by facilitated transport, which is saturable at high lactate concentrations (Roth & Brooks, 1990a; Roth & Brooks, 1990b), this appears a distinct possibility.

SUMMARY

While the results of the 10 x 6 second protocol do not support this notion, the hypothesis that $\dot{V}O_2$ max is important in anaerobic recovery is well supported by the outcome of the 6 x 15 second protocol. The study also suggests that of the adaptations that could be expected to accompany aerobic training, it is those peripherally located that are most beneficial, at least during a protocol similar to the one employed here. If this is the case, the implication is that training for activities of this nature should be sport specific. As peripheral adaptations occur at the muscle level, it would seem important that any endurance training create a hypoxic environment in those muscles utilized within the sport. Based on the obtained results the following conclusions are made:

1. Aerobic power is important in the recovery from repeat bouts of anaerobic exercise of 15 seconds duration.

2. Adaptations which enhance a-vO2 are the most beneficial during a protocol of this nature.

3. More experimentation is required in an effort to determine the importance of both aerobic power and the central and peripheral components of this attribute in intermittent work of a shorter duration (eg. 6 seconds)

4. Aerobic power, and in particular the a-vO2 component, promote a faster resynthesis of CP during passive recovery following maximal intermittent exercise.

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APPENDIX ONE

LETTER OF CONSENT

THE EFFECTS OF AEROBIC POWER ON INTERMITTENT ANAEROBIC PERFORMANCE

LETTER OF CONSENT

I understand that this research project is studying the effects of aerobic fitness on short duration high intensity exercise. I understand that the experimental procedure requires my attendance at the human fitness testing laboratory on four separate occasions so that by the completion of the study I will have completed each of the four tests outlined below.

TEST 1 - A graded aerobic power ($\dot{V}O_2$ max) test that will require me to pedal a cycle ergometer at gradually increasing workloads until exhaustion. This test will be approximately 8-11 minutes in duration.

TEST 2 - A non - invasive cardiac output test that will require me to pedal a cycle ergometer for approximately 4 minutes at 60% effort.

TEST 3 - An intermittent exercise test that will require me to pedal at maximal intensity for 6 repetitions of 15 seconds duration with 1 1/2 minutes recovery between each repetition.

TEST 4 - An intermittent exercise test that will require me to pedal at maximal intensity for 10 repetitions of 6 seconds duration with 30 seconds recovery between each repetition.

I understand that any data collected in the study will remain confidential and will be kept in a locked filing cabinet. Furthermore I understand that my name will not be attached to any published results and that code numbers will be used to identify the results obtained from individual subjects.

I understand that whether I participate or choose not to participate will have no bearing on team selection or future scholarship decisions. I understand that by signing this form I am agreeing to participate in this study however I still have the right to withdraw at any time without explanation.

SIGNATURE: _____

EXPERIMENTER: _____

DATE: _____

APPENDIX TWO

POWER OUTPUT RAW DATA

Table 8 - Average mean power (MP) (Standard Deviation) and average peak power (pp) (Standard Deviation) for the 6 x 15 second protocol and 10 x 6 second protocol (Watts).

MP (6 x15s)	BOUT	PP (6 x 15s)	MP (10 x 6s)
813 (107)	1	1035 (142)	956 (106)
761 (100)	2	949 (127)	908 (113)
701 (95)	3	913 (129)	846 (106)
652 (91)	4	863 (100)	803 (127)
626 (92)	5	802 (100)	780 (118)
608 (78)	6	779 (101)	749 (104)
	7		730 (101)
	8		721 (107)
	9		710 (101)
	10		693 (100)

APPENDIX THREE

REVIEW OF LITERATURE

Many sports, such as hockey soccer and rugby, demand intermittent bursts of maximal intensity exercise. Creatine phosphate (CP) represents the most immediate reserve skeletal muscle possesses for adenosine triphosphate (ATP) resynthesis during intense exercise. However this store of high energy phosphate is essentially depleted within 10 seconds following the onset of maximal exercise (Jacobs, Tesch, Bar-Or, Karlsson & Dotan, 1983; Jones, Heigenhauser, Kuksis, Matsos, Sutton & Toews, 1985). Glycolysis represents the secondary pathway for ATP replenishment and the fact that lactate concentrations are significantly elevated following 10 seconds of maximal intensity work (Jacobs et al. 1983), suggests that not only is this pathway activated almost immediately upon the onset of muscular contraction, but also that it represents an important source of ATP replenishment in exercise of even a very short duration. In support of this observation, Hultman and Sjoholm (1983) demonstrated that as much as 50% of the ATP resynthesised between 1.26 seconds and 2.5 seconds following the onset of electrical stimulation is the result of the formation of lactate.

However the metabolic consequences of high intensity short duration work appear to have an adverse effect on subsequent performance (Spreit, Lindinger, McKelvie, Heigenhauser & Jones, 1989; Jenkins, Brooks & Williamson, 1994). While factors such as substrate depletion and thermal stress may act as fatiguing agents in prolonged exercise, the exact mechanisms responsible for a fatigue

induced loss of short term power output remain debatable (Fitz & Metzger, 1993). Evidence exists that suggests an increase in hydrogen ions (H^+), resulting from both the hydrolysis of ATP and the dissociation of the anaerobic end product lactic acid, has an adverse effect on muscular contraction and hence the maintenance of performance. Studies that have utilized skinned muscle fibers have demonstrated both a decreased ability to develop tension and a slowing of the rate of tension development as a result of a decrease in muscular pH from 7.4 to 6.2 (Fabiato & Fabiato, 1978). Hirche, Hombach, Langhor Wacker and Busse (1975) observed that by making perfusing blood either more acidic or alkalitic, using ammonium chloride and sodium bicarbonate respectively, the endurance capacity of the canine gastrocnemius in response to electrical stimulation could be altered. It was postulated that an increase in blood alkalinity favored an improved H^+ removal rate, thereby delaying the exercise induced drop in muscle pH and in turn delaying muscle fatigue. The observations that fatigue characteristics could be stimulated by lowering pH (Curtin & Edman, 1989) and that performance wass enhanced by the ingestion of sodium bicarbonate (Jones, Sutton, Taylor & Toews, 1977; MaClaren & Morgan, 1985) provides further evidence of a relationship between muscle pH and fatigue.

If a decrease in pH represents a potential fatigue agent as the evidence cited above suggests, there exists a number of mechanisms by which an increase in the concentration of H^+ may interfere with the contractile process and hence interfere with power development.

Donaldson (1983), used pealed mammalian skeletal muscle fibers to examine the effects of a decrease in pH on force production and noted slow twitch oxidative , fast twitch oxidative and fast twitch glycolytic lost 12%, 25% and 44% of maximum force respectively as a result of intracellular acidosis. Due to the methodology employed, some possible effects of H^+ on the contraction process were eliminated and it was suggested that the increase in H^+ concentration caused an end-product inhibition of the reaction catalyzed by actomyosin ATPase (Equation 1), thus lowering force generation.

$$ATP \rightleftharpoons ADP + P + H^+$$

(Equation1)

Studies which employed the skinned fiber technique have also suggested that an increased H^+ concentration may have a direct effect on the contractile process by influencing calcium release from the sarcoplasmic reticulum (SR). Nakamura and Schwartz (1970) demonstrated that a drop in pH from 7.56 to 6.46 resulted in a decreased calcium release from the SR and concluded that the affinity of the SR for calcium depends specifically on pH. Although caution must be exercised when analyzing these results as they were obtained in vitro, fatigued muscle shows a reduced rate of peak tension development (Vergara, Rapoport, & Nassar-Gentina, 1977) which appears consistent with an increased binding capacity of calcium to the sarcoplasmic reticulum at low pH.

In addition, Donaldson, Hermansen and Bolles (1978) observed that under acidic conditions (pH = 6.5) an increased calcium

concentration was required to develop the same tension as that which was obtained at pH = 7.0. The possibility also exists that an increased hydrogen ion concentration hinders cross bridge formation and hence muscle contraction by competing with calcium for troponin C binding sites.

An exercise induced rise in muscle pH may also pose a threat to performance by hindering the rate of ATP production. Hill (1928) observed that the production of lactate ceased when the pH of electrically stimulated muscle reached 6.3. Further evidence for the possibility of an adverse effect of H^+ production on the rate of ATP resynthesis can be found in the fact that phosphofructokinase (PFK), considered to be a rate limiting enzyme of glycolysis (Fox, Bowers & Foss, 1989), is almost completely inhibited at a pH of 6.5 (cited in MaClaren, Gibson, Parry-Billings & Edwards, 1989). It has also been argued (McLaren et al. 1989) that this inhibition of PFK will mean an increased concentration of the substrate fructose -6- phosphate, and in turn of glucose -6- phosphate which will inhibit both hexokinase and phosphorylase. This appears consistent with the fact that an elevated H^+ concentration slows the transformation of phosphorylase b to the more active phosphorylase a, as well as lowering the maximum level of active phosphorylase within the muscle. (Sahlin, 1975; Chasiotis, Sahlin & Hultman, 1982). This effect may explain the results of Spreit et al. (1989) who used muscle biopsies between three 30 second bouts of maximum cycling to examine the relationship of work output to glycolysis. Both glycogenolysis and pathway intermediates such as glucose 6 phosphate, glycerol 3 phosphate and pyruvate decreased

following the third bout of exercise indicating a reduced glycolytic flux. It was suggested that as muscle pH was as its lowest during this time period, phosphorylase activity had been inhibited thereby resulting in an inhibition of glycolysis.

Apart from the evidence discussed above regarding the possible adverse effects of lowered pH on the activity of glycolytic enzymes such as phosphorylase and pyruvate kinase, ATP production from the glycolytic pathway may be inhibited by the reduction of pyruvate (Sjodin, 1976). In this study it was found that the activity of lactate dehydrogenase (type M) was inhibited by the presence of its product in skeletal muscle. As concentrations of lactate were similar to those found in muscle during heavy exercise, this observation suggests that the presence of lactic acid in muscle has a product inhibitory effect on glycolysis.

To summarize the argument to this point, the drop in intra muscular pH that accompanies the breakdown of ATP and accelerated glycolysis at the onset of high intensity exercise has the potential to adversely effect performance. Consequently, an important component of the recovery phase during intermittent high intensity exercise, will be the bodies ability to return to a state as closely nearing homeostasis as possible in order to maximize subsequent performance. As the replenishment of CP involves the release of H^+, a return of skeletal muscle towards normal pH may also facilitate this component of recovery by removing any product inhibition of the forward reaction (Equation 2).

$$\text{Creatine} + \text{ATP} \rightleftharpoons \text{CP} + \text{ADP} + H^{+} \quad \text{(Eqn.2)}$$

The close relationship between muscle pH and the action of creatine kinase (Sahlin, Harris & Hultman, 1979) is consistent with an inhibition of this nature, as may be the findings of Spreit et al. (1989) and Jansson, Dudley, Norman and Tesch (1989) who after one and four minutes respectively, reported a limited restitution of creatine phosphate following successive bouts of high intensity exercise. As the half time for creatine phosphate replenishment has been reported to be approximately 25-35 seconds (McCulley, Vandenborne, DeMeirleir, Posner & Leigh, 1992; Harris, Edwards, Hultman, Nordesjo, Nyland & Sahlin, 1976) the protocol utilized in these two studies must have caused circumstances which inhibited CP resynthesis. As both protocols involved repeated bouts of high intensity exercise that were successful in significantly elevating lactate concentrations, the associated rise in H^{+} may have been responsible for this inhibition. The demonstration that the rate of CP recovery is slower for intense than light or moderate exercise (McCann, Mole & Caton, 1995; Arnold, Mathews, & Radda, 1984; Taylor et al. 1986) is consistent with this proposal.

Apart from the buffering of lactic acid by sodium carbonate within the blood, a number of other pathways such as glycogen synthesis and oxidation within the mitochondria are available for the body to rid itself of the dissociated components of lactic acid during the recovery phase (Gladden, 1989). However of the pathways available, the oxidation to pyruvate and the subsequent passage through the Krebs

Cycle as part of oxidative metabolism appears to be the major fate of lactate (Mazzeo, Brooks, Schoeller & Budinger, 1986: Donavan & Brooks, 1983). As hydrogen ions are consumed on an equimolar basis in this process (Gladden & Yates, 1993), it represents a vital mechanism in restoring pH towards homeostatic values and thus maximizing the potential for subsequent performance.

Given the importance of oxidative metabolism in restoring lactate concentrations to normal values, it is possible that individuals with a more effective aerobic system will be better equipped to restore homeostatic muscle pH. This implies that performance should be maintained at a higher standard during intermittent work where rest periods are not of a long enough duration to enable full recovery. The finding that endurance training results in an increased activity of type H lactate dehydrogenase (Apple & Rogers, 1986; Sjodin , 1976) which facilitates the first step of lactate oxidation; its conversion to pyruvate, appears consistent with this proposal.

Endurance training resulting in an increased $\dot{V}O2$ max has also been demonstrated to increase blood volume (Saltin, Blomqvuist, Mitchel, Johnson, Wildenthal & Chapman 1968) and cardiac output (Ekblom & Hermansen, 1968). This increased potential for muscular blood flow would appear to be beneficial in transporting lactate more rapidly to either non exercising muscle or the liver where it can be eradicated by either glycogenosis or oxidative metabolism via the Krebs Cycle. In addition, a larger blood volume may facilitate the concentration mediated diffusion of lactate from the muscle to blood . There exists little evidence to either support or contradict this

proposal. However Sahlin et al. (1979) found that while the lactate/pyruvate ratio of muscle was greatly increased following exercise, this value showed no trend towards resting values during a 2 minute recovery period in which local circulation was prevented. A similar protocol (Sahlin, Harris & Hultman, 1975) resulted in no change in intra muscular pH for a 2 minute occluded circulation recovery period and in addition reported that a subsequent 20 - 25 seconds of returned perfusion was sufficient to significantly return pH towards resting values. Although these results do not prove a relationship between an increased aerobic potential and lactate dissipation, they do demonstrate the importance of blood flow in the recovery process. It is also possible that during exercise that is succesful in severely elevating lactate concentrations, the rate of lactate and thus H^+ removal from the cell may be the limiting factor and not the ability of the blood to accept these metabolites. This appears to be a distinct possibility given that the majority of lactate appears to cross the sarcolemal membrane by facilitated transport which is saturable at high lactate concentrations (Roth & Brooks, 1990a; Roth & Brooks, 1990b).

Endurance training has also been demonstrated to increase capillary density and thus surface area (Anderson, 1975; Anderson & Henriksson, 1977). As the capillary surface area represents the muscle to blood exchange surface for metabolic substances, a training induced increase in this component should also facilitate the removal of lactate from muscle to blood. Tesch & Wright (1983) obtained a significant correlation between blood lactate concentration and

capillary density following 50 maximal voluntary knee extensions. Recovery, as indicated by a fatigue index (subsequent torque as a percentage of initial torque), also was found to correlate with capillary density. In addition, Graham, Sjogaard, Lollgreen and Saltin (1978) demonstrated that after submaximal cycling, the muscle to blood lactate concentration gradient was lower in those subjects with predominately slow twitch muscle fibers compared to those with predominantly fast twitch fibers. As slow twitch fibers have a more developed capillary network (Andersson, 1975; Anderson & Henrikssson, 1977) it is possible that the resultant larger capillary surface area, enabled a greater lactate output by exercising muscle.

The increased mitochondrial content (Gollnick & King, 1969; Holloszy, 1967), and associated increase in oxidative enzymatic activity (Holloszy, 1967; Gollnick, Armstrong, Saubert, Piehl & Saltin, 1972) that results from endurance training, may also be of benefit in the oxidation of lactate. Jansson, Dudley, Norman & Tesch (1989), reported a significant negative correlation between citrate synthase and muscle lactate concentration. Whether this apparent faster lactate dissipation was a result of an increased lactate oxidation rate is unknown. It is possible that other factors are significant such as a slower rate of production or a faster removal of lactate into the blood due to a greater capillary surface area that may have existed in those fibers with a high citrate synthase activity level.

Apart from possibly reducing the derogatory effects of a reduced muscle pH, the literature also suggests that an elevated maximal aerobic power (MAP) may be beneficial in increasing the CP

resynthesis rate during recovery. Sahlin et al. (1975) found that after 25 seconds of maximum voluntary contraction of the knee extensors creatine phosphate failed to show any recovery during the subsequent 2 minute period in which circulation was prevented. In fact a decrease of creatine phosphate was actually observed. When local blood flow was restored for a period of 20 - 25 seconds there was a significant return towards resting values. Saltin et al. (1979), using incubation of post exercise samples in either oxygen rich or oxygen deprived environments, demonstrated the importance of oxygen availability to the resynthesis of CP. While 68% of resting creatine phosphate values were restored in the oxygen rich environment, complete inhibition of this recovery process was observed in the oxygen deprived sample. Further demonstration of the importance of oxidative phosphorylation in the resynthesis of CP comes from Tesch, Thorsson and Fujitsuka (1989) who upon the completion of 30 maximum voluntary knee extensions, found no difference in CP levels between fast and slow twitch fibers. However 60 seconds into recovery the ST fibers had a significantly elevated CP level, leading the authors to conclude that the ST fibres, well adapted to oxidative phosphorylation (Gollnick & King, 1969; Gollnick et al. 1972), exhibited a faster CP resynthsesis rate. These results, along with the possibility of a relationship between muscle pH and CP recovery (Harris et al, 1976; Sahlin, et al. 1979), suggests that an increased aerobic potential would also be beneficial during recovery by promoting a faster resynthesis of this high energy phosphate. There exists evidence to support this notion. Yoshida & Watari (1993) reported a significantly faster ($p < 0.05$) CP

resynthesis rate in a group of long distance runners when compared to controls following 2 minutes of femoral flexion. This is supported by McCulley et al. (1992) who examined the CP resynthesis rate in a group of long distance runners, track athletes and controls with a significant relationship ($p < 0.001$) between the length of running event and CP resynthesis rate being reported. In addition, it was reported that this high energy phosphate could be restored at a maximum rate of 64.8 $m \cdot mol \cdot min^{-1} \cdot kg^{-1}$ of muscle by long distance runners compared to 38.6 $m \cdot mol \cdot min^{-1} \cdot kg^{-1}$ of muscle for their control counterparts. Despite this evidence there exists no study to the authors knowledge that has demonstrated an improved restoration rate in the form of an increased subsequent performance.

It is also possible that evidence for an improved ability to resynthesise CP could be seen in a faster recovery towards resting $\dot{V}O2$ levels following exercise. Margaria, Edwards & Dill (1933) and Hill, Long & Lupton (1924) were responsible for the pioneering work in the establishment of a fast and slow component of O2 debt repayment following exercise. While there appears to be a number of factors that effect the slow component, such as temperature (Brooks & Gaesser, 1980) and catacholimine release (Harris, 1980), the close relationship between the fast component of recovery and CP resynthesis has been demonstrated in both human (Hultman, Bergstrom & McLennon-Anderson, 1967) and canine (Piiper & Spiller, 1970) muscle.

Although the indirect evidence cited above appears to suggest that an enhanced aerobic system will facilitate the maintenance of performance in repeated bursts of high intensity anaerobic exercise,

the current literature fails to demonstrate a direct relationship. This appears primarily due to a lack of published research attempting to examine this question. Gaiga and Docherty (1993) used a nine week interval training program to increase aerobic power and this work represents one of the few studies that have attempted to demonstrate a relationship. Performance over four repeats of a 30 second maximal effort Wingate test on a cycle ergometer was compared to pre-training results and those of a control group. The training group showed performance improvements that were particularly noticeable over the last two bouts of exercise, suggesting a possible enhanced recovery due to an increased aerobic power. However significant increases in performance in bouts one and two indicated that the aerobic training program had also been effective in improving anaerobic performance making the results difficult to interpret.

Schreiner (1988) compared muscle lactate removal during four 30 second bursts of maximal cycling between a high aerobic power group ($\dot{V}O2$ max > 54.6 $mL \cdot kg^{-1} \cdot min^{-1}$) and a low aerobic power group ($\dot{V}O2$ max < 50.9 $mL \cdot kg^{-1} \cdot min^{-1}$). The results of the study led to the conclusions that aerobic power has no effect on muscle lactate accumulation, muscle lactate removal, blood lactate concentration or blood lactate dissipation following four minutes of recovery. However these results also appear inconclusive as there was a significant difference in the power output between the two groups. In addition, it appears possible that the gapin aerobic power between groups (3.7 $mL \cdot kg^{-1} \cdot min^{-1}$) was insufficient to demonstrate any significant difference.

Jansson et al. (1989) utilized three 30 second bouts of unilateral knee extensions with a 60 second rest period between each one in an effort to examine if there was a relationship between aerobic oxidative potential of skeletal muscle and recovery after exercise. It was reported that peak torque and CP concentration following recovery positively correlated with citrate synthase activity, however any relationship with MAP was not reported.

Although unable to examine subsequent performance, there exists a number of studies in the literature that have documented metabolic recovery following a single bout of exercise (Evans & Cureton, 1983; Oyono-Enguelle et al. , 1990; McCulley et al. 1992; Petersen & Cooke, 1994; Yoshida & Watari, 1993). Of those studies making reference to aerobic potential, results again appear contradicting. Evans and Cureton (1983) concluded that an increased $\dot{V}O2$ max did not significantly alter blood lactate concentration or dissipation, while Oyono-Enguelle et al. (1990) reported a significant correlation between $\dot{V}O2$ max and the ability to remove lactate from muscle. It is important to note however that these studies utilized very different protocols and as such caution must be exercised when making comparisons. Oyono - Enguelle et al. (1990) for example, employed a graded exercise routine that resulted in a bout of exercise lasting between 20 and 32 minutes depending on the aerobic potential of the individual. Consequently the benefit of this study is questionable in regards to recovery after short duration maximum intensity exercise. Also of interest, and possibly as an explanation for the contradicting results, is that Evans and Cureton (1983) based their conclusions on

the rate of lactate dissipation from the blood, a value that was not significantly altered by endurance training. However, as blood lactate in this group was significantly elevated 3 minutes into the post training recovery period, the time of the first sample, a possible training effect may have been acting to facilitate the more effective movement of lactate from the working muscle. It is therefore possible that a measure of either muscle pH or subsequent performance would have demonstrated an aerobic training induced adaptation that was facilitating the return of muscle pH towards homeostatic values, and which was not evident in a protocol utilizing blood lactate concentration alone.

Petersen and Cooke (1994) compared endurance trained and sedentary individuals using magnetic resonance spectroscopy. While no difference in the time course of creatine phosphate resynthesis was observed, the endurance trained group showed a faster recovery towards resting pH values. No attempt was made however to evaluate this effect on subsequent performance. In contrast, McCulley et al. (1992) and Yoshida & Watari (1993) report the existence of a relationship between creatine phosphate resynthesis and MAP. It is important to note that McCulley et al. (1992), unlike that of Peterson & Cooke (1993), reported resting pH values at the completion of the protocol further reinforcing the possibility of a previously discussed effect of pH on creatine phosphate resynthesis. Yoshida & Watari (1993) failed to report pH values.

In conclusion, while the argument for the importance of an efficient aerobic system to recover from high intensity intermittent work seems

sound, its value in maintaining subsequent performance has not been demonstrated in the literature. However it appears an important issue to resolve so as to maximize the efficiency of training for sports involving activity of this nature. If it becomes apparent that the benefits towards recovery are minimal, coaches and athletes may be better advised to invest their time in other forms of training, such as short duration speed work. If on the other hand an efficient aerobic system does facilitate recovery, and hence subsequent performance, a characteristic which would be advantageous in maintaining intensity during training as well as competition, it would appear to be beneficial in establishing the relative importance of the central and peripheral components of aerobic power, as different strategies have been suggested for the training of each (McDougall & Sale, 1981).

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