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**EFFECT OF TRAINING STRATEGIES AND
CREATINE SUPPLEMENTATION ON
PERFORMANCE AND METABOLISM
DURING SPRINT SWIMMING**

by

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ABSTRACT

Many scientific studies have considered physiological aspects of swimming, but largely in the areas of endurance or strength and power. This thesis includes six studies that attempt to provide more information about the metabolic responses to single and repeated sprint swimming and the physiological mechanisms behind the limitation to sprint swimming performance.

The first experimental chapter describes the metabolic responses to single and repeated sprinting in male and female swimmers. Peak blood lactate (male 18.7 and female 14.4 mmol l⁻¹; P<0.01) and ammonia (male 232.0 and female 154.3 μmol l⁻¹; P<0.05) values following repeated swimming (8 x 50 yards) were almost double those measured during a single 50 yards sprint and were significantly higher in males than females. It is likely that differences in body dimensions and composition between male and female swimmers account for the majority of the ~12% performance differences and higher metabolic response in males than females.

Energy contribution to single and repeated tethered swimming sprints was examined in chapter V. Determination of energy contribution by an accumulated oxygen deficit test found estimated anaerobic contribution of ~67% in 30 s sprinting and ~74%, ~53%, ~51% and ~47% during four 30 s sprint bouts. These were much lower than values estimated previously and recommended to coaches and swimmers in popular swimming texts. Energy contribution to 55 s maximal tethered swimming in chapter VI found anaerobic contributions of ~30-40%.

Metabolic responses to Controlled frequency breathing (CFB) have been studied previously in endurance swimming, but not in sprint swimming (chapter VI). There was increased hypercapnia, but no significant reduction in performance during 55 s maximal sprint tethered swimming between self-selected breathing and breathing every 10 strokes. Differences in metabolic responses (higher extraction of oxygen from inspired air and lower ventilation, oxygen consumption, carbon dioxide production and respiratory exchange ratio) suggest a greater efficiency during swimming with CFB. Swimmers who can train to overcome the urge to breath should not compromise

performance, but benefit from avoiding an increase in drag resistance while turning the head to breath.

Active recovery following intense swimming has been suggested to increase the speed of recovery and improve subsequent performance. Chapter VII illustrates that the timing and intensity of active recovery is crucial when prescribing repeated sets of repeated sprint training. Lower blood lactate was matched by a tendency for poorer performance in the trial using active recovery between repetitions. This demonstrates that the blood lactate concentration does not reflect the metabolic state of the muscle and therefore the ability to perform subsequent sprint swims.

Chapters VIII and IX consider the effects of creatine supplementation on sprint swimming. No differences in single sprint swimming performance were found, but creatine supplementation improve times in a typical training set of 8 x 50 yards by ~4 s. Faster times recorded in the creatine group support the hypothesis that increasing resting levels of creatine and phosphocreatine will enhance recovery during repeated sprints. Supplementing with 3g creatine day⁻¹ for 22-27 weeks had no additional benefit to race performance than just ‘loading’ before the training period and immediately prior to the major swimming race of the year. It is likely that any enhanced training adaptation would have to be from creatine supplementation allowing swimmers to perform more training rather than just supplementation *per se*.

The studies in this thesis describe the physiological and metabolic responses of elite male and female swimmers to single and repeated sprint swimming in detail for the first time. By manipulating breathing frequency during sprinting, metabolism altered but without compromising performance. Active recovery was successful in reducing blood lactate concentration, but performance was poorer. The blood metabolite and respiratory response to sprint training following interventions of this type allow us to determine the mechanisms behind the limitation to swimming performance. Creatine supplementation enhances repeated sprint swimming performance, but not training for success in competition. Results of this thesis suggest that phosphocreatine availability or energy supply are not limitations to sprint swimming training performance.

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To Mum and Dad –

I owe you everything

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CHAPTER I

INTRODUCTION

What advice can we give to help swimmers swim faster? This is the question that has intrigued and challenged coaches since at least the first modern day Olympiad in 1896. It has also been the source of considerable interest and drive for scientists, notably since the early work of Du Bois Raymond in 1905, followed by both Cureton and Karpovich in the early 1930's (Karpovich, 1933). The majority of Olympic swimming races (>80%) last for less than 2 min 30 s, and are characterised by maximal, fatiguing efforts that rely heavily on energy supply from anaerobic sources. Despite this, few studies have considered the physiological responses to sprint swimming. This thesis contributes to the sparse information available regarding sprint swimming, and uses maximal single and repeated sprints of ≤ 55 s in an attempt to understand the physiological mechanisms that determine the limitations to sprint swimming performance.

It is clear when researching the swimming physiology literature, that many studies have considered the endurance or the strength and power aspects of the sport (Costill, 1992; Reilly, 1990). In contrast, there is a paucity of information on the metabolic demands of sprint swimming. This reflects the perception of coaches that the endurance contribution to performance is of prime importance, as demonstrated by the high training volume and duration undertaken by elite competitive swimmers.

Recently however, the pressure to produce Olympic level swimmers and the limited information highlighting the anaerobic contribution to swimming (Capelli et al., 1998) have led many scientists to question the value of high volume, long duration programmes in favour of higher intensity sprint programmes (e.g. Costill, 1999). Despite this, it remains unclear exactly how much and what types of training will bring about optimal improvements in elite swimmers, and what physiological mechanisms underpin such changes. There is general disagreement as to the relative energy contributions to sprint swimming and the repeated sprint swimming commonly used in training (e.g. 50 m race: 98% anaerobic, Maglischo, 1993; or 70% anaerobic, Ring et al., 1996). The metabolic consequences of both single and repeated sprint swimming in both male and female swimmers have not been detailed extensively (Prado et al., 1999).

In training, it is generally accepted that swimming at high intensities (at least some of the time) is important (Costill et al., 1985c; Mujika et al., 1996), and that an ability to train at these intensities for longer and with shorter recoveries will result in greater adaptation. It is possible that programmes that use active recovery might increase the speed of recovery between exercise bouts, thus enhancing training performance and adaptation to training resulting in improved swimming performance. Responses to active recovery in swimming competition performance have been documented (Beckett and Steigbigel, 1993; Cazorla et al., 1983; Raeburn and Mackinnon, 1990), but no direct evidence exists regarding the best strategies for recovery during and between sprint training sets.

Much attention has focused on creatine (Cr) supplementation in sport since the early 1990's based on evidence that subjects may increase resting levels of Cr and PCr in the

muscle (Harris et al., 1992) and therefore improve sprint performance. Studies initially focused on effects of Cr on performance in swimming races (Burke et al., 1996; Mujika et al., 1996), which produced equivocal results. Several studies using other modes of exercise found that improvements in performance were found only in repeated, not single, sprint performance (Balsom et al., 1993a; Greenhaff et al., 1993b). If positive effects of Cr supplementation in swimming were found only in repeated swimming, the usefulness of using such a nutritional strategy would be in the training for competition, rather than the competition *per se*. There is a need to discover the exact physiological mechanisms behind improvements in repeated swimming, and whether supplementation with Cr would result in an improved ability to train and thereby enhance competition performance.

The aims of this thesis therefore are as follows:

1. To examine the physiological demands of sprint swimming performance.
2. To examine the effectiveness and underlying rationale of different training strategies and creatine supplementation as interventions to enhance sprint swimming performance.

The thesis has been organised into ten chapters. Chapter II is a detailed review of the literature specific to the physiology of swimming followed by general methods (chapter III). Each of the experimental chapters are then presented in order:

- The first experimental chapter (Chapter IV) describes the metabolic responses of males and females to single and repeated sprint swimming.

-
- The second experimental chapter (Chapter V) introduces a tethered swimming mechanism enabling the measurement of force output and the collection of expired air for the determination of oxygen uptake. A maximal accumulated oxygen deficit (MAOD) test was performed to estimate the energy contribution to single and repeated swimming sprints.
 - Chapter VI considers the physiological responses of controlled frequency breathing to maximal intensity swimming performance. The study again uses the tethered swimming equipment to measure performance and oxygen uptake.
 - The effects of active and passive recovery on performance and metabolism during repeated sprint swimming are investigated in Chapter VII. This study attempted to determine the effectiveness of active, in contrast to passive, recovery between repetitions and training sets commonly used by swimming coaches.
 - Chapter VIII describes the effects of oral creatine supplementation on single and repeated sprint swimming.
 - The purpose of the final experimental chapter (IX) was to examine the effects of creatine supplementation on training for competition in elite swimmers.
 - The final chapter (X) draws together the findings in the thesis and attempts to answer the question, what are the physiological limitations to swimming performance? Different aspects of preparing swimmers for competition are addressed and their likely impact on performance stated. Finally, the mechanisms behind the findings of the thesis are discussed and recommendations for practical applications made.

CHAPTER II

REVIEW OF LITERATURE

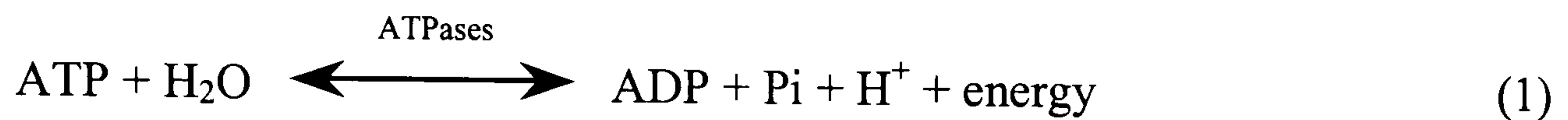
This review covers relevant physiological aspects of sprint swimming in relation to both training and competition. Sections are included on energy metabolism and fatigue relating to sprint swimming, although cycling and running studies have provided much of the direct information in these areas. The relationship of muscle strength and power and the cardio-respiratory responses to sprint swimming are considered. The competitive and training demands have been detailed and specific swimming testing research has been documented in section eleven. A significant section is also included on creatine supplementation and its relationship to sprint swimming.

2.1 Energy metabolism

2.1.1 ATP

Adenosine 5'-triphosphate (ATP) is the energy currency of life and responsible for all human movement and therefore exercise. It constantly needs to be replenished as the limited stores are broken down to provide energy. Resting concentrations have been measured at ~21 (Boobis et al., 1983; Cherry et al., 1998), ~25 (Soderland and Hultman 1991; Greenhaff et al., 1993b) and ~28 (Cheetham et al., 1986) mmol kg⁻¹ dry muscle mass (dm). Electrical stimulation at 50 Hz lasting 1.28 s to 2.56 s demonstrated that ATP can be hydrolysed at rates as high as 10.3 – 11.0 mmol ATP kg dm⁻¹ s⁻¹ (Hultman and Sjoholm, 1983). In 6 – 10 s of maximal sprint cycling, energy expenditures of 10.8 (Boobis et al., 1982) and 13.7 (Jones et al., 1985) mmol ATP kg⁻¹ dm s⁻¹ have been

recorded. If there were no processes to regenerate ATP, then after 2 – 4 s, the stores would be totally depleted and rigour would set in. The hydrolysis of ATP can be described as follows:



Where H₂O is water, ADP is adenosine 5'-diphosphate, Pi is inorganic phosphate and H⁺ is a hydrogen ion.

2.1.2 Phosphocreatine

Muscle biopsy studies have found that ATP concentration only decreases by ~20% following short-term (6-10 s) maximal exercise (Boobis, 1992; Bogdanis et al., 1995), demonstrating the rapid resyntheses of ATP by a number of different, but closely integrated, energy replacement systems that support ATP resynthesis. The muscle can resynthesise ATP in a number of ways (Spriet, 1995):



Equation (2) shows the reversible degradation of phosphocreatine (PCr) a high-energy compound that combines with ADP to regenerate ATP, a reaction that is catalysed by creatine phosphate kinase (CPK). The resultant Cr produced at the contractile site is transported back to the mitochondrial membrane where it combines with the inorganic phosphate given up by the degradation of ATP to ADP to resynthesise PCr. It is then transported back to the myofibril band to resynthesise contractile ATP (see Fig 2.1

below). This process also absorbs a hydrogen ion (H^+), which aids the buffering effect and counteracts the expected drop in pH.

Skeletal muscle phosphocreatine concentrations in humans have been reported in the range of ~ 68 (Greenhaff et al., 1988) for type I fibres and $\sim 90 \text{ mmol kg dm}^{-1}$ (Soderland et al., 1992) for type II muscle fibres. These concentrations on their own will only allow resynthesis of ATP for $\sim 8 \text{ s}$, as one mole of PCr will resynthesise one mole of ATP.

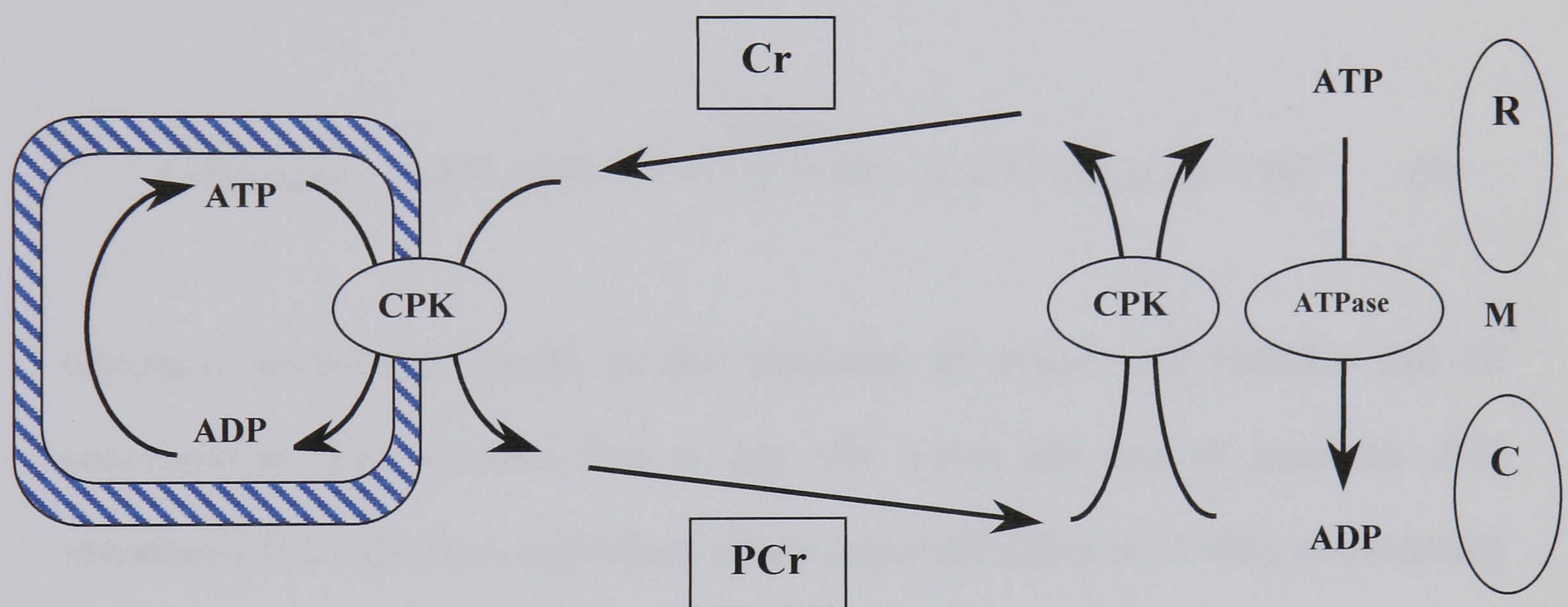
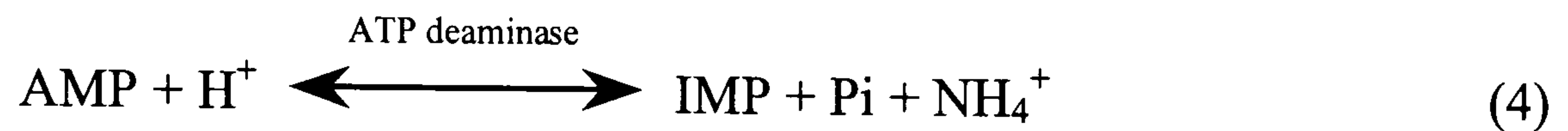


Figure 2.1 The phosphocreatine shuttle (adapted from Bessman and Savabi, 1990). C = muscle contracted; R = muscle relaxed; M = myofibril. Shaded area: mitochondrial membrane

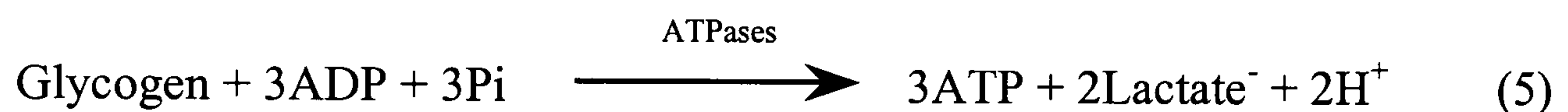
2.1.3 The adenylate kinase reaction

Muscle ATP can also be resynthesised in extreme exercise conditions (maximal intensity) through the adenylate kinase reaction (3), which combines two ADP molecules, usually following a pooling of ADP, to produce ATP and adenosine monophosphate (AMP). The adenylate kinase reaction is not reversible, and therefore AMP deamination takes place, forming inosine monophosphate (IMP), and excreted through the purine cycle; its eventual product being ammonia (NH_3 ; 4).



2.1.4 Anaerobic glycolysis

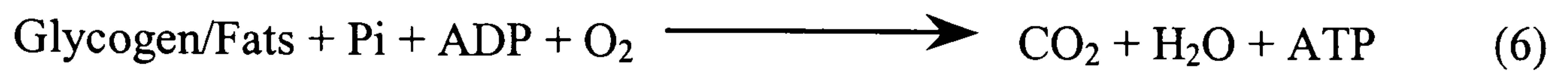
Equations 1 to 4 will only supply energy during maximal exercise for a relatively short time (~10 s). Fortunately the muscle can also utilise glycogen anaerobically as a fuel to resynthesise ATP (5).



Glycogen degradation results in the formation of lactate and increases the H^+ concentration. The resultant drop in pH will reduce the rate of anaerobic ATP resynthesis from glycogen and reduce power output (Nevill et al., 1996), or eventually cause a complete cessation of exercise (see sections 2.2.1 to 2.2.4).

2.1.5 Oxidative metabolism

Aerobic metabolism resynthesises ATP at a much slower rate than from anaerobic metabolism, but with greater efficiency and without fatigue inducing by-products. Glycogen and fats (and to a much lesser degree protein) are broken down, producing relatively large amounts of ATP (e.g. carbohydrate: 39 moles of ATP per mole of glycogen; fats: 129 moles of ATP per mole of palmitic acid), carbon dioxide and water. This breakdown is illustrated in equation 6 (Astrand and Rodahl, 1986).



This equation simplifies a complex sequence of chemical reactions that take place in the cell mitochondria and involve both the tricarboxylic acid (TCA) cycle and the electron transport chain (for details, see Maughan et al., 1997).

2.1.6 Energy supply during sprint swimming

There is no information regarding the direct measurement of muscle metabolites following high intensity swimming. This is perhaps a function of the swimming environment and the difficulty in obtaining biopsy data immediately (2-4 s) following the cessation of exercise. Information regarding the changes in muscle metabolites following sprint swimming must be taken from studies using other modes of exercise (e.g. running and cycling) over similar durations and intensities, or using blood metabolite measurements as a reflection of what has previously happened in the muscle. Table 2.1 shows the rate of ATP resynthesis from PCr and glycogen, and the total amount of ATP supplied in sprint running and cycling exercise. Maximal sprint exercise is characterised with a rapid breakdown of PCr from the onset of exercise. This rate of degradation appears to decline after only 1.3 to 2.5 s (Hultman and Sjoholm 1983b), although glycolysis reaches its maximal rate ~3 s (Greenhaff et al., 1996) to ~5 s (Maughan et al., 1997) after exercise begins. In a 6 s sprint approximately half the ATP regeneration is provided by PCr degradation (Gaitanos et al., 1993), the rest by anaerobic glycolysis and from aerobic sources.

Table 2.1 illustrates the decline in calculated ATP resynthesis rate, from 14.9 mmol kg⁻¹ dm s⁻¹ (Gaitanos et al., 1993) for 6 s cycling, 11.6 mmol kg⁻¹ dm s⁻¹ (Bogdanis et al.,

1996) for 10 s cycling, 7.5 mmol kg⁻¹ dm s⁻¹ (Bogdanis et al., 1995) for 30 s cycling and 6.0 mmol kg⁻¹ dm s⁻¹ (Nevill et al., 1989) for 30 s treadmill running and 3.4 mmol kg⁻¹ dm s⁻¹ (Withers et al., 1991) for 60 s cycling. Bangsbo et al. (1990) estimated that in 192 s, the value had dropped to 1.9 mmol kg⁻¹ dm s⁻¹, but that 1.6 mmol kg⁻¹ dm s⁻¹ (or ~84%) of the anaerobic ATP replacement over the total time was from glycolysis.

Table 2.1 Quantity and average rate of anaerobic ATP supply from phosphocreatine degradation and glycolysis (adapted from Spriet, 1995, Tables 1.2 and 1.3)

Study	Exercise mode & duration	Rate (mmol kg ⁻¹ dm s ⁻¹)		ATP provision (mmol kg ⁻¹ dm)			
		PCr	Glycol	ATP	PCr	Glycol	Total
Bangsbo et al. (1990)	IKE: 0-192 s	0.3	1.6	10	60	300 ¹	370
Boobis et al. (1982)	C: 0-6 s	6.0	4.8				
	C: 0-30 s	1.9	4.0				
Boobis et al. (1983)	C: 30 s	2.0	4.4				
Bogdanis et al. (1995)	C: 30 s			25.6	77.1	322	424.8
Bogdanis et al. (1996a)	C: 30 s			27.0	75.2	328	430.2
Cheetham et al. (1986)	NMT: 30 s	1.9	3.8				
Cherry et al. (1998)*	C: 30 s			22.6	79.0	–	
Costill et al (1983)	$\dot{V}O_2$ max R: 0-60 s	–	1.3				
	400 m R: 0-60 s	–	2.1				
Gaitanos et al. (1993)	C: 6 s			24.0	76.5	317	417.5
Hultman and Sjöholm 1983a)	ES: 1-1.28 s	~9.0	~2.0				
	ES: 1-2.56 s	~5.0	~5.3				
Hultman & Sjöholm (1983b)	ES: 0-10 s	~3.3	~2.3				
	ES: 10-20 s	~2.0	~3.0				
	ES: 20-30 s	~0.9	~3.8				
	ES: 0-30 s	~2.1	~3.0				
	ES: 0-50 s	~1.5	~3.4				
Hultman et al. (1967)	C: 0-77 s	0.8	–				
Jacobs et al. (1982)	C: 30 s			20.9	62.7	360	443.6
Jacobs et al. (1983)	C: 0-10 s, M/F	–	6.0/2.9				
	C: 0-30 s M/F	–	3.4/2.1				
Jones et al. (1985)*	IC: 30 s			10	45	190+	245+
Karlsson et al. (1975)	IM 50%: 0-90 s	0.8	1.1	3	70	153+	226
Kalsson & Saltin (1970)	C: 0-143 s	0.4	0.7	10	55	95+	160+
McCartney et al. (1986)	IC: 30 s	1.4	5.9	22.6	62.0	373	457.6
Nevill et al. (1989)	NMT: 30 s	1.9	4.1				
Spriet et al. (1987)	ES: 0-50 s	1.3	1.8	20	75	210	305
Withers et al. (1991)	C: 0-30 s	1.3	3.7				
	C: 0-60 s	0.9	2.5				

*: Combined values for all subjects, ¹: ATP contribution from accumulated muscle lactate, escaped lactate and accumulated glycolytic intermediates [range of durations as well as individual times]
ATP: Adenosine 5'- triphoshate, PCr: Phosphocreatine; Glycol: Glycolysis
C: Cycle ergometer, ES: Electrical stimulation, NMT: Non-motorised treadmill, IC: Isokinetic cycling, IKE: Isokinetic knee extensions, IM: Isometric, m: metres

2.2 Fatigue during sprint swimming

2.2.1 Definition

Fatigue can be defined as the inability to maintain the required or expected speed of activity or power output (Edwards, 1983). The cause of fatigue has been claimed to relate to both central and peripheral mechanisms, although any one of the stages in the 'chain of command' (Edwards 1983, p6) between the brain and the muscle contractile elements may contribute to reduced performance. The intensity and duration of the exercise will dictate to a large degree the cause of fatigue.

2.2.2 Sites of fatigue

Studies have reported that the decline in ATP resynthesis approximates the muscle's ability to generate force (Hultman et al., 1990). Some debate exists whether the drop in ATP resynthesis causes the decline in muscle force and therefore fatigue, or whether the decline in force development is affected by other factors such as the accumulation of muscle metabolites, reducing the energy demand and therefore the rate of ATP utilisation. The decline in skeletal muscle force that occurs during maximal sprinting is associated with many metabolic changes. There is a decrease in muscle ATP and PCr with an increase in ADP, inorganic phosphate (P_i) and H^+ . The accumulation of these products of ATP hydrolysis, in addition to a decrease in muscle pH, are likely to influence fatigue to a large extent during sprint swimming. Figure 2.2 illustrates the possible sites of fatigue within skeletal muscle that may be affected by products of ATP hydrolysis and will be discussed throughout the section.

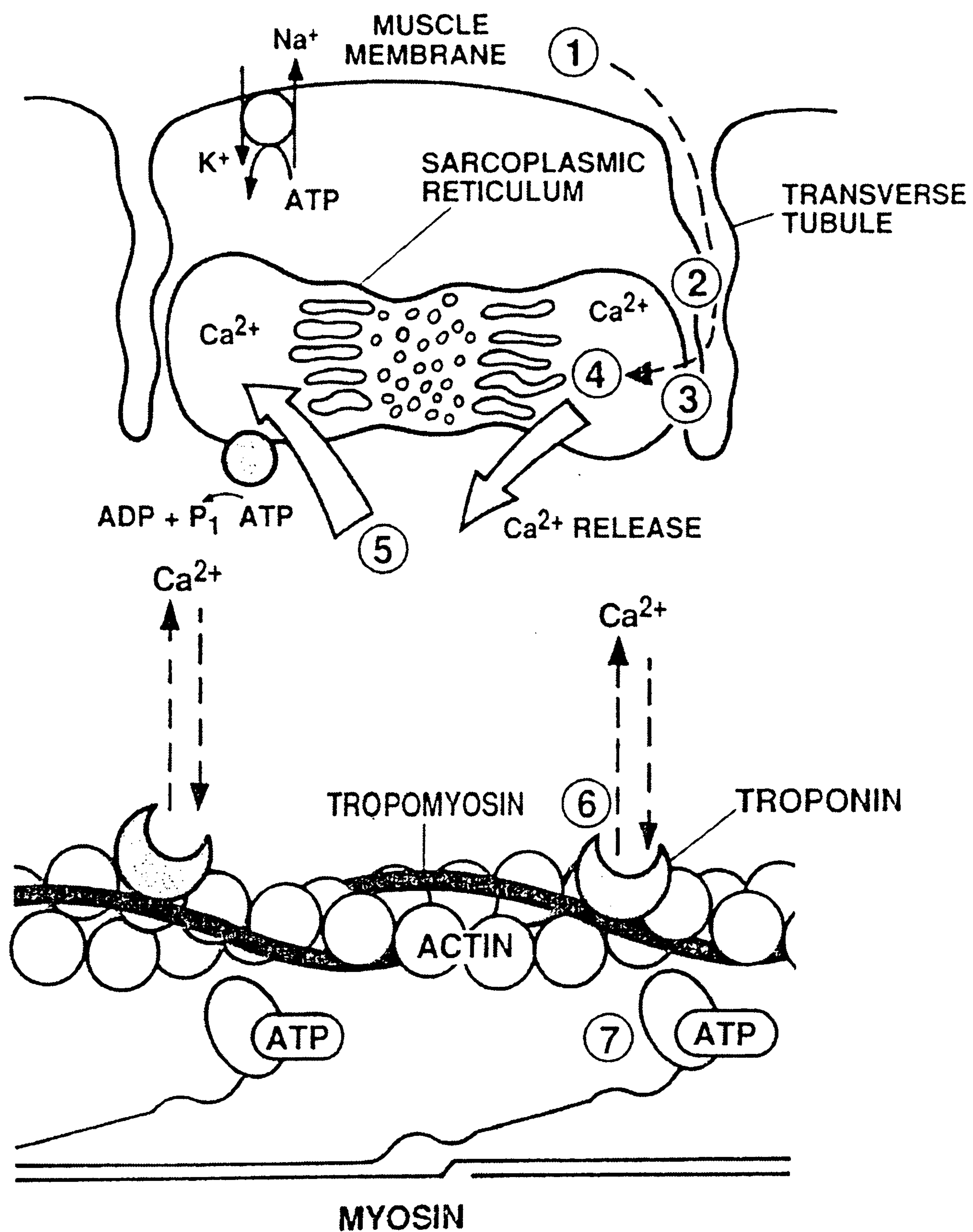


Figure 2.2 Diagrammatical representation of the major muscle cell components during excitation-contraction coupling and the possible sites of muscular fatigue during maximal exercise: 1. Cell membrane, 2. t-tubule charge movement, 3. mechanisms coupling t-tubular charge movement with SR Ca^{2+} release, 4. SR Ca^{2+} release, 5. SR Ca^{2+} reuptake, 6. Ca^{2+} binding to troponin, 7. Actomyosin hydrolysis of ATP, cross-bridge force development and cycling rate (from Fitts and Metzger, 1988).

2.2.3 Decline in phosphocreatine

The decline in the rate of resynthesis of adenosine triphosphate (ATP) as a result of depletion of phosphocreatine (PCr) is recognised as a possible cause of reduction in muscular power in maximal intensity exercise (Hultman et al., 1967). The glycolytic rate of ATP regeneration is slower than that of PCr and therefore as its contribution increases, potential contraction speed and therefore force generation will decline. Evidence for reduction in PCr as one of the mechanisms for reduced power output in sprinting is provided from both creatine supplementation (see section 2.12) and recovery studies. Recovery of PCr stores has been found to be significantly correlated with power output restoration in maximal sprinting (Bogdanis et al., 1995), although it would seem that this is not the only factor involved in fatigue as studies have also demonstrated a faster PCr recovery than the restoration of muscle contractile force (Hultman and Sjöholm, 1983a; McCartney et al., 1986).

2.2.4 The role of ADP, P_i and H^+

The hydrolysis of ATP produces significant increases in the concentrations of ADP, P_i and H^+ during maximal intensity exercise. It is possible that an accumulation of ADP will inhibit the release of ADP from the cross-bridges in the actomyosin complex. The result will be a reduction in cross-bridge dissociation, reducing the force generated and the maximal velocity of muscle fibre shortening (Jones and Round, 1990; Figure 2.3). Effects of ADP on skeletal muscle force generation are equivocal. Evidence for ADP causing force decreases have been shown in intact (Cady et al., 1989a) and skinned muscle (Godt and Nosek, 1985), although the impairment of cross-bridge detachment occurred outside the normal physiological range ($\sim 4 \text{ mmol l}^{-1}$; Jones and Round, 1990).

The impact of ADP on the reduction of force generation is likely to be from a slowing of the relaxation rate at the muscle fibril cross-bridge site. Step (iii) in Figure 2.3 shows that ADP release occurs before dissociation of the myosin head from its actin site and therefore an increase in ADP near the myosin head will slow down its release and therefore the cycling sequence (Jones and Round, 1990).

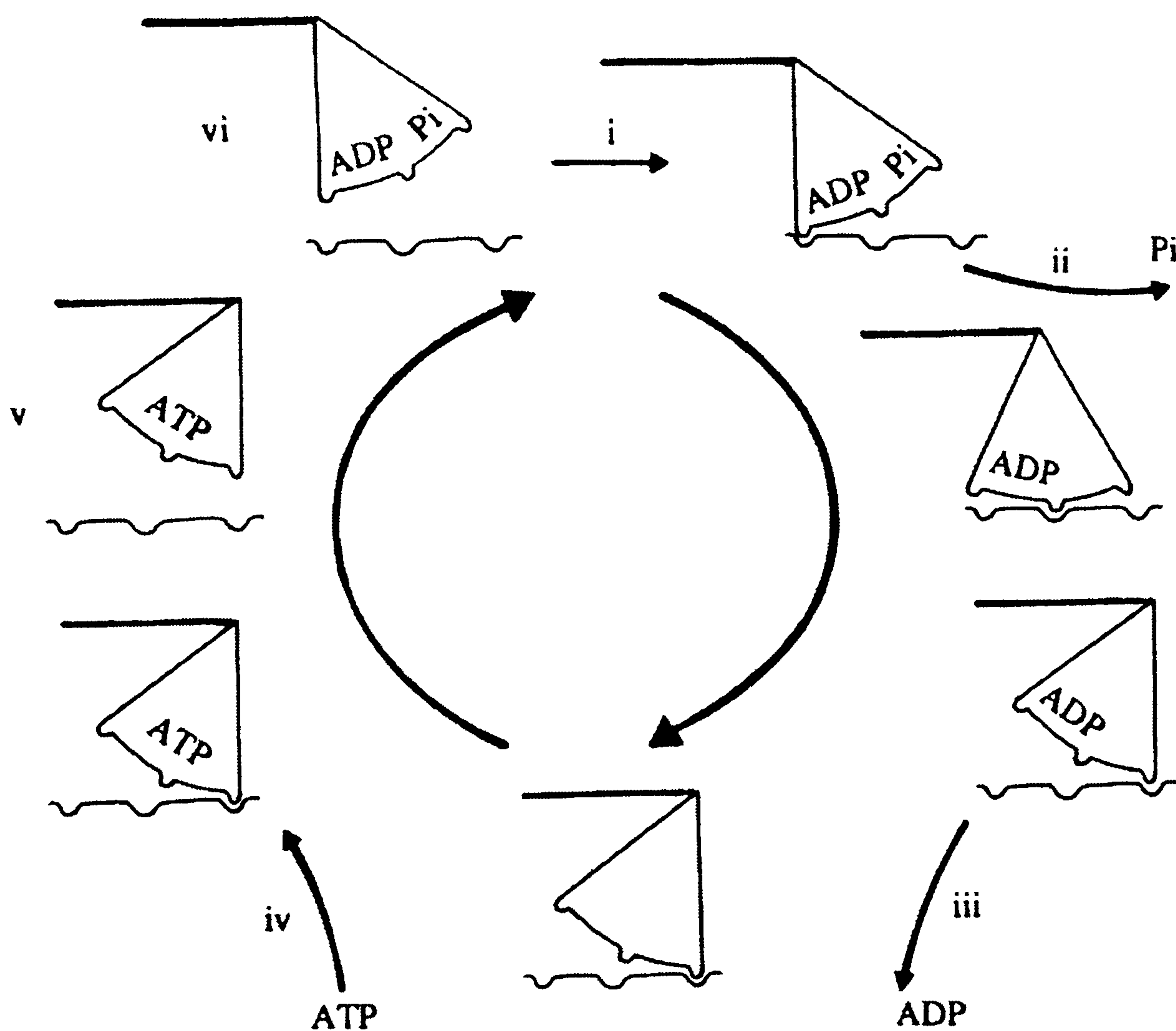


Figure 2.3 Stages in the cross-bridge cycle corresponding to the different biochemical steps. (i) Attachment of actin and myosin, giving stiffness to the muscle, but not generating force, (ii) P_i release from the actomyosin complex resulting in force generation from the rotating myosin head, (iii) ADP is released towards the end of rotation, (iv) The actomyosin complex can then bind ATP, (v) actin and myosin dissociate with myosin-bound ATP, (vi) ATP is hydrolysed, activating the myosin head ready to bind to actin again (from Jones and Round, 1990, p38).

Increase in P_i , from ATP-PCr splitting has been suggested as an important glycolytic regulator. A drop in muscle pH has been found to increase the proportion of P_i in its monoprotonated form (HPO_4^{2-}) over its diprotonated form ($H_2PO_4^-$) which will further decrease pH and limit glycogenolysis (Chasiotis et al., 1989). Reduction in force generation has been closely correlated with $H_2PO_4^-$ (Green, 1995), although maximal exercise, circulatory occlusion studies elevating P_i in combination with phosphorylase transformation are not enough to show high glycogenolytic rate (Ren and Hultman, 1989). It has also been suggested that P_i has a direct action on the muscle contractile mechanism, slowing the release of phosphate from actomyosin and leading to an accumulation of cross-bridge attachments where they are unable to develop force (step ii, Figure 2.3, Jones and Round, 1990).

A decrease in pH as a result of H^+ accumulation has been shown to inhibit the regulatory glycolytic enzymes phosphorylase and phosphofructokinase (PFK), which in turn will decrease the glycolytic rate (Hermansen, 1981; Hultman et al., 1990). Hydrogen ions also compete with Ca^{2+} for binding sites on troponin and therefore increase the amount of Ca^{2+} needed to achieve the same tension development (Green, 1990). An increase in H^+ has also been shown to limit PCr resynthesis, leading the creatine kinase reaction towards PCr breakdown and reducing the high-energy phosphate store (Hultman, 1990; Sahlin et al., 1983).

Accumulation of H^+ may also affect the activity of myosin ATPase, Ca^{2+} reuptake ATPase and Na^+-K^+ ATPase thereby slowing the cross-bridge relaxation rate and reducing force (Cady et al., 1989a; Cooke and Pate, 1990). Finally, H^+ might contribute

to fatigue by reducing Ca^{2+} SR release (Nakamura and Schwartz, 1972), or decrease the sensitivity of the force generating mechanisms (Fitts and Metzger, 1988).

Studies have demonstrated both a close relationship between a drop in pH and slowing of the muscle relaxation rate (Sahlin et al., 1981), and dissociation between the two (Cady et al., 1989b). Cady et al. (1989b) demonstrated muscle relaxation rate slowing in a subject with myophosphorylase deficiency (MFD; unable to produce H^+ from glycolysis) indicating that this mechanism of fatigue can occur independently of H^+ accumulation. Recovery of the relaxation rate was faster in the MFD patient than normal subjects suggesting that persistently low pH may be related to the rate of recovery of relaxation rate. Conflicting results from these studies suggest that perhaps other factors may contribute to fatigue during maximal exercise in humans, although it is important to consider the interaction between a number of causal factors including a drop in muscle pH.

2.2.5 The role of calcium and potassium

Calcium release from the SR is crucial in muscle function and three mechanisms linking calcium with fatigue have been identified (Allen et al., 1992). Firstly, a decline in intracellular Ca^{2+} release, perhaps related to a slow reuptake of Ca^{2+} during intense exercise. Secondly, a reduced sensitivity of the muscle myofilaments to Ca^{2+} (e.g. competition with H^+ for troponin binding sites). Finally, a reduction in maximal force development at saturating Ca^{2+} (Westerblad et al., 1991). The site of fatigue may occur at the muscle membrane (and t-tubules) rather than the contractile mechanism as the decline in isolated muscle fibre tension has been restored following the introduction of caffeine (Westerblad et al., 1991).

Muscle membrane excitability is dependant on the membrane potential and consequently the potassium (K^+) gradient across the membrane (Sjogaard, 1987). Extracellular K^+ concentration only needs to rise slightly to cause a reduction in muscle tension (Juel, 1988), and following maximal intensity exercise, would eventually result in a depolarisation block leading to fatigue (Vollestad and Sejersted, 1988). Intracellular K^+ loss can be attributed to the impairment of function in the sodium-potassium ($Na^+ - K^+$) pumps due to an energy deficiency, or the inhibition of the enzyme $Na^+ - K^+$ ATPase by ADP or H^+ accumulation (Sejersted and Hallen, 1987).

Two distinct recovery phases have been identified immediately post-sprinting, the first appears to be independent of metabolite resynthesis (Cherry et al., 1998; Westerblad et al., 1991). The initial (~2-3 s) recovery phase has been correlated well with the restoration of muscle membrane potential and intracellular sodium (Na^+) and K^+ , with a second, slower recovery phase that was linked with the resynthesis of PCr and a reduction in P_i (Balog and Fitts, 1996). The accumulation of K^+ in muscle interstitium has been proposed as a key fatigue factor, irrespective of muscle acidity, although the release of K^+ from the muscle cell may be caused by the decrease in pH (Bangsbo et al., 1996).

2.2.6 Glycolytic enzymes

The continuum of ATP resynthesis during maximal exercise relies heavily on glycolysis between ~10 and 120 s. Anaerobic ATP replenishment from glycolysis is slower (see Table 2.2) than that of PCr, but is further rate limited by inhibition of phosphofructokinase (PFK) a regulatory glycolytic enzyme and/or phosphorylase which catalyses glycogen breakdown. Evidence for these enzymes regulating the rate of

glycolysis are found from muscle biopsy studies that have measured a large accumulation of the glycolytic intermediates glucose- (G6P) and fructose-6-phosphate (F6P), but only modest increases in glycolytic intermediates below F6P (Boobis, 1983; Jones et al., 1985). It is unclear whether an increase in muscle H^+ or the build up of G6P providing feedback inhibition that affects the PFK reaction (Jones and Heigenhauser, 1992).

2.2.7 Lactic acid and ammonia

2.2.7.1 Introduction

Anaerobic glycolysis is maximally activated within a few seconds of the onset of sprint exercise (Greenhaff et al., 1996a), and results in a production of lactic acid and H^+ and a concomitant reduction in pH. The net ATP production is 3 moles ATP for every molecule of glycogen via anaerobic glycolysis, compared with 39 moles of ATP provided per molecule of glucose by aerobic oxidation (Astrand and Rodahl, 1986). Anaerobic glycolysis therefore can provide relatively high rates of ATP resynthesis, but at the expense of a reduced ATP output and therefore efficiency.

Lactic acid is a by-product of anaerobic glycolysis, although in the muscle it almost completely and immediately dissociates to lactate and H^+ (Sahlin, 1986). Lactate production is believed to be the main source of H^+ (~85% of total muscle accumulation; Sahlin, 1986 – the remainder mostly from PCr degradation and oxidative metabolism) and the resultant drop in muscle pH (Hultman and Sahlin, 1986). The conversion of pyruvate to lactate in the glycolytic pathway is catalysed by the enzyme lactate dehydrogenase. In low to moderate exercise intensities, pyruvate enters the citric acid (TCA) cycle and oxidation takes place. The produced H^+ combine with nicotinamide-

adenine dinucleotide (NAD) to produce NADH^+ and then pass to oxygen to produce water through the process of aerobic glycolysis. In high intensity exercise, the rate of oxygen demand for this process outstrips supply and both lactate and H^+ accumulate.

2.2.7.2 Muscle and blood, lactate and pH during sprinting

There is no information regarding blood and muscle pH or muscle lactate during sprint swimming, and therefore other modes of exercise will be reviewed. Sprint exercise relies heavily on anaerobic ATP resynthesis and results in both significant muscle, and subsequently (5-6 min; Cheetham et al., 1986) blood, lactate increases. Muscle lactate values are typically $<10 \text{ mmol kg}^{-1} \text{ dm}$ at rest, but rise to between $\sim 60 \text{ mmol kg}^{-1}$ (Jacobs et al., 1982) and $\sim 78 \text{ mmol kg}^{-1}$ (Cheetham et al., 1986) following maximal 30 s sprinting. Cheetham et al. (1986) found ~ 29 fold increases in muscle lactate following maximal 30 s sprint running on a nonmotorised treadmill, and values correlated well with blood lactate concentrations ($r=0.82$). High correlations between blood lactate and peak running speed ($r=0.93$) and peak power output corrected for body mass ($r=0.83$) were also observed.

Blood lactate concentration is dependent on muscle lactate production rate, the rate of diffusion from muscle cells and the rate of lactate removal (Gollnick and Hermansen, 1973). Bogdanis et al. (1995) found muscle lactate values of $\sim 120 \text{ mmol kg}^{-1} \text{ dm}$ after a 30 s cycle sprint and a corresponding blood lactate concentration of 13.6 mmol l^{-1} 6 min after the sprint. Blood pH dropped from 7.38 to 7.08, values similar to those in other single 30 s maximal sprints (Cherry et al., 1998; Nevill et al., 1989). The highest blood lactate ($\sim 16 - 18 \text{ mmol l}^{-1}$) and lowest blood pH values (~ 7.04) have been recorded following a 30 s maximal sprint by Allsop et al. (1990), Jones et al. (1985; blood lactate

only) and Nevill et al., 1996a, the latter using sprint-trained subjects. Single, maximal cycling or running bouts of between 45 and 120 s (Hermansen and Osnes, 1972; Sahlin et al., 1975; Spriet et al., 1987b) and repeated 30 s cycle sprints (Spriet et al., 1989) have shown to reduce muscle pH (from muscle biopsy) to their lowest values (~6.4-6.5).

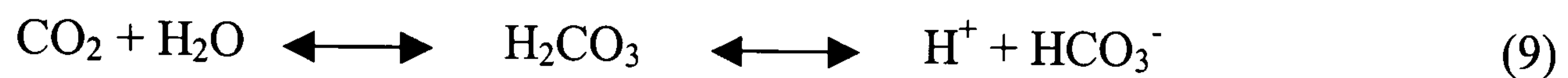
2.2.7.3 Blood lactate in swimming

Blood lactate values following 50 m sprint swimming in male and female national level swimmers reach ~10 mmol l⁻¹ (Avlonitou, 1996; Bonifazi et al., 1993), although values as high as ~12-15 mmol l⁻¹ have been reported in elite swimmers following maximal intensity racing (Holmer, 1972; Nadel et al., 1974; Telford et al., 1988). In one study, values of 25 mmol l⁻¹ were reached following 100 and 200 m race performances (Sawka 1979), values which exceed those recorded in repeated sprint cycling (~23 mmol l⁻¹ McCartney et al., 1986). In the most extensive study of post-competition blood lactate concentrations following 100, 200 and 400 m races, Chatard et al. (1988) reported values ranging from ~13 to ~17 mmol l⁻¹ with some subjects recording >20 mmol l⁻¹. Results compared favourably with an Olympic silver and bronze medallist who recorded values of ~17 mmol l⁻¹ in both 200 and 400 m race distances. A strong relationship was observed between the highest lactate values and individual best performance times (Chatard et al., 1988).

2.2.7.4 Buffering and acid/base balance

At the onset of maximal intensity exercise, glycolytic lactate accumulates with a similar increase in H⁺ and drop in pH. The drop in pH is dependent on the amount of H⁺ produced (related to lactate production) and the ability of the muscle to buffer H⁺ or release it into the circulation (Hultman and Sahlin, 1980). Muscle H⁺ buffering is

dependent on two main systems; physiochemical buffering including the bicarbonate- CO_2 system (Equation 9 below), and metabolic buffering, particularly from PCr degradation (see section 2.1.2).



The importance of adequate circulation to the muscle, and the ability of the lungs to remove CO_2 after buffering are crucial to metabolism during sprint exercise. In addition, improved circulation to the muscle will aid O_2 delivery and H^+ efflux from muscle, maintaining a more effective intracellular environment.

2.2.7.5 Plasma ammonia during swimming

In extreme exercise conditions, a pooling of ADP occurs and the adenylate kinase reaction (section 2.1.3) combines ADP molecules to resynthesis ATP with inosine monophosphate (IMP) and NH_4^+ produced in equal amounts (Sahlin and Katz, 1988). The NH_4^+ produced diffuses into the blood where it can be measured as an almost complete reflection of AMP deamination following maximal sprint exercise (Graham et al., 1993). Peak blood NH_4^+ concentrations have reached $>250 \mu\text{mol l}^{-1}$ in sprinters and $>150 \mu\text{mol l}^{-1}$ in endurance-trained subjects (Nevill et al., 1989), and $>150 \mu\text{mol l}^{-1}$ in senior trained swimmers following maximal sprint exercise (Prado, 1999). Strong, significant correlations ($>r = 0.80$) have been reported between post-sprint blood lactate, pH and plasma NH_4^+ following sprint cycling (Bogdanis et al., 1995) and swimming (Prado, 1998), although it is likely that this is a coincidental, rather than a causal link (Terjung and Tullson, 1992).

2.2.8 Summary

There are many metabolic changes that have been proposed as possible contributory factors in muscle fatigue following brief, maximal intensity exercise. The hydrolysis of ATP produces excess ADP, P_i and H^+ , all of which may affect the myofibril contractile mechanism. Increases in ADP may slow the ATP resynthesis rate. Accumulating P_i has been suggested as a glycolytic regulator and a fall in muscular pH (from an increase in H^+) is likely to be involved in a number of fatigue mechanisms such as increase in PCr breakdown, glycolytic inhibition, and the interference with the normal role of Ca^{2+} in the contractile mechanism.

2.3 Relative contribution of aerobic and anaerobic metabolism to sprint exercise

2.3.1 Introduction

Traditional attempts to quantify the contribution of each energy system to ATP resynthesis were based on two flawed assumptions. Firstly that each system worked sequentially (Margaria et al., 1969), and secondly that the oxygen debt (i.e. post-exercise oxygen consumption above the resting value) was an accurate predictor of the anaerobic energy contribution during exercise (Fox et al., 1969). We now know from direct measurements of muscle metabolites in muscle biopsies that there is a significant lactate accumulation from the onset of exercise, reflective of anaerobic glycolysis (Gaitanos et al., 1983; Boobis et al., 1982; Hultman and Sjodin, 1983) and a small, but significant (~5% in 10 s, Maughan et al., 1997) contribution from aerobic sources.

Studies that have measured muscle metabolites can provide a more direct estimate of anaerobic ATP resynthesis than previous estimates using O_2 debt. Equations (7) and (8)

from Spriet (1995) and Nevill et al. (1996a), respectively, allow good estimates of the anaerobic ATP provision, assuming that no lactate escapes from the muscle:

ATP provision from anaerobic metabolism =

$$\Delta \text{PCr} + 1.5 \Delta \text{lactate} + 2 (\Delta \text{ATP} - \Delta \text{ADP}) \quad (7)$$

ATP utilisation from anaerobic metabolism =

$$-2 (\Delta \text{ATP}) - \Delta \text{ADP} - \Delta \text{PCr} + 1.5 (\Delta \text{pyruvate}) + 1.5 (\Delta \text{lactate}) \quad (8)$$

Spriet (1995) provides an approximation for the calculation of relative energy contribution to maximal exercise from aerobic and anaerobic sources based on muscle metabolites and accumulated oxygen deficit data obtained from knee extensor exercise by Bangsbo et al. (1990):

- 0-30 s = 80% Anaerobic/20% Aerobic
- 60-90 s = 45% Anaerobic/55% Aerobic
- 120-192 s = 30% Anaerobic /70% Aerobic

2.3.2 Oxygen debt

‘Oxygen debt’ is the total amount of oxygen consumed during recovery above the resting value and first described by Hill and Lupton in 1923 (Green and Dawson, 1993).

The use of ‘oxygen debt’ as an accurate measurement of anaerobic metabolism has been discredited by several authors (Medbø, 1996; Saltin, 1990; Bangsbo et al., 1990). Bangsbo et al. (1990) showed that the technique overestimated anaerobic energy supply

by ~200% using 'oxygen debt' following maximal knee extension exercise of ~ 3min. The cause of this overestimation appears to come from restoration of temperature, hormonal balance and energy required to regain body homeostasis. This will result in elevation of the post-exercise oxygen consumption above that which represents 'repaying the debt' of anaerobic metabolism.

The over-estimation in anaerobic energy contribution to exercise from oxygen debt measurements has recently been confirmed following direct measurements of muscle metabolites before and immediately following exercise. Bogdanis et al., (1996) estimated the aerobic contribution to 30 s sprint cycling from muscle metabolites obtained by needle biopsy. The ATP utilised during the sprint was calculated from phosphocreatine, glycolytic intermediates and lactate production, and oxygen uptake measured during and post-sprint. The aerobic contribution to the single 30 s sprint was estimated at 29%, with a rise in contribution to 44% during a second 30 s sprint 4 min later. This higher value has been confirmed by Serresse et al., (1988; 28%) and Hill (1999; 37% for 49 s) using direct measurements, and appears to be the trend in studies using mathematical modelling and oxygen deficit techniques (for a full review, see Gastin, 2001).

2.3.3 Accumulated oxygen deficit

2.3.3.1 Introduction

The concept of oxygen deficit was first introduced by Krogh and Lindhard in 1920, and developed as a potential method to quantify anaerobic ATP provision by Karlsson and Saltin (1971). Further development by Medbø and co-workers (Medbø et al., 1988; Medbø and Tabata, 1989; Medbø and Burgers, 1990) have validated and popularised

the measurement as estimate of anaerobic capacity (see review by Gastin, 2001). Anaerobic capacity is the maximal amount of ATP resynthesised by anaerobic processes (Green and Dawson, 1993; Medbø et al., 1988), although not all physiologists believe it is possible to measure anaerobic capacity due to accuracy in methods and the close interaction and contribution of all energy supply processes during sprinting. If anaerobic capacity is to be estimated, then clear definitions of exactly what is being measured in addition to strict methodological controls must be made (Bangsbo, 1996; Green, 1994; Green and Dawson, 1993; Medbø, 1996).

Estimated anaerobic capacity (determined by maximal accumulated oxygen deficit, MAOD) varies significantly between individuals, is closely related to the muscle mass used and as a consequence is highly dependant on the mode of exercise used (Medbø and Burgers, 1990). Medbø and Burgers, 1990 found men had higher MAOD values than females and suggest a potential for greater trainability. However, the level of performer and training status was not clear, and it is likely that this will have more impact on training improvements than a difference between the sexes. Medbø et al. (1988) used treadmill running as the mode of exercise, and several others have studied MAOD in cycling. Since anaerobic capacity (estimated by MAOD) is specific to the exercise performed, similar research in swimming seems warranted.

2.3.3.2 Validity and criticisms of oxygen deficit

Recent dialogue between two of the world's leading scientists who have studied MAOD suggests that the use of the measurement is either unreliable and inaccurate (Bangsbo, 1996b), or relevant and valid as an indicator of anaerobic capacity (Medbø, 1996). Concern has been expressed about the measurement as 'whole-body' oxygen

consumption is considered as opposed to oxygen uptake for active muscles only. This difference should be minimal, although it has been suggested that during maximal sprint running, the upper body contribution to ‘whole-body’ oxygen consumption is much greater (Bangsbo, 1996b). Estimates are frequently made about the active muscle mass involved in the exercise and what proportions of muscles (and parts of muscles) are active during the exercise. The energy cost of the exercise is also taken as an estimate, usually from extrapolation of the submaximal oxygen consumption – intensity linear relationship. This assumes that mechanical efficiency at maximal intensities is the same as at submaximal levels, although Bangsbo et al. (1993) report a lower efficiency and therefore excessive oxygen uptake for a given speed. This will result in an underestimation of the maximal oxygen demand, and therefore estimated anaerobic capacity.

Despite this, Medbø et al. (1988) propose that the MAOD is an accurate or unbiased measure of anaerobic capacity if measurements during their reported protocols are adhered to and errors are minimised (Medbø, 1996). Medbø and Tabata (1993) found significant correlations between the MAOD and anaerobic ATP resynthesis as estimated from muscle metabolites obtained from biopsy needle. The MAOD also appears to be unaffected by hypoxia (Medbø et al., 1988) and is sensitive to changes following sprint-training (11% improvement in MAOD has been reported following 6 weeks of sprint-training; Medbø and Burgers, 1990). Oxygen deficit test validity is supported by Bangsbo (1996) but only in repeated tests that measure similar relative submaximal intensities, rather than speed *per se*. An improvement in the oxygen consumption – speed relationship (shift of the straight line to the right) will result in an underestimation of the O₂ demand and therefore the ‘real’ improvement in MAOD (and therefore

estimated anaerobic capacity) will be masked (Bangsbo, 1996b). The cause of the ‘real’ improvement observed is still debatable as it may be attributable to an improved mechanical efficiency (independent of changes in anaerobic capacity) following training (Bangsbo 1996).

The MAOD has been shown to be higher in sprint, compared with endurance-trained and untrained subjects (Medbø and Burgers, 1990; Scott et al., 1991), and in elite versus moderate level swimmers (Troup et al., 1990b). Gastin and Lawson (1994) found higher rates of ATP resynthesis from anaerobic metabolism in sprint cyclists compared with endurance trained triathletes, despite lower $\dot{V}O_{2\max}$ values. The sprint group showed faster O_2 kinetics (suggestive of a greater O_2 demand) and the final power output during the 90 s exercise bout was almost directly related to the O_2 consumption, suggesting that the difference between the groups must have come from a higher anaerobic capacity in the sprint group (Gastin and Lawson, 1994). The ability to discriminate between sprint and endurance performers and its sensitivity to changes following sprint training (Medbø and Burgers, 1990) makes the oxygen deficit a test a valuable method of estimating anaerobic capacity.

Although controversy still remains regarding the efficacy of using the MAOD as an estimation of anaerobic capacity, it appears to be one of the few realistic and viable methods of determining energy system contributions during swimming exercise in the absence of more invasive (muscle biopsy) data (Troup et al., 1994; Zoeller et al., 2000). Figure 2.2 details the estimated energy contribution to sprint exercise using direct measurements from muscle biopsy, the MAOD method and by mathematical modelling.

Table 2.2 Relative contributions of aerobic and anaerobic energy supply during short-term (< ~60 s) sprint exercise (expressed as a % of total energy supply, adapted from Gastin, 2001).

Author	Exercise Mode	Method	Duration (s)	Energy source (%)	
				PCr and Glycol	O ₂
Bogdanis et al. (1996a)	C	DM	30	66	34
Calbet et al. (1997)	C	MAODI	30	77	23
			45	69	31
Gastin and Lawson (1994)	C	MAODI	15	81	19
			30	71	29
			45	61	39
			60	54	46
Gastin (2001)*	Various	Various	0-20	82	18
			0-30	73	27
			0-45	63	37
			0-60	55	45
Gollnick and Hermansen (1973)	C	DM	10	83	17
			60	60	40
Hermansen and Medbø (1986)	R	MAODI	15	78	22
			30	71	29
			60	49-57	43-51
Hill (1999)	R	DM	49	63	37
			61	62	38
Kavanagh and Jacobs (1986)	C	MAODA	30	82	18
Medbø and Tabata (1989)	C	MAODI	30	66	34
Nummela and Rusko (1995)	R	MAODI	49	54	46
			50	63	37
O'Brien et al. (1997)	C	MAODI	30	70	30
			60	54	46
Serresse et al. (1988)	C	DM	30	72	28
Smith and Hill (1991)	C	DM	30	84	16
Spencer and Gastin (2001)	R	MAODI	22	71	29
			49	57	43
Spencer et al. (1996)	R	MAODI	52	54	46
Ward-Smith (1985)	R	MM	20	86	14
			45	72	28
Withers et al. (1991)	C	MAODI	30	72	28
			60	51	49

PCr: Phosphocreatine resynthesis; Glycol: Anaerobic glycolysis; O₂: Aerobic ATP resynthesis
Where figures are provided under both PCr and La, values represent total anaerobic ATP resynthesis
*Taken as a summary of data collected: Table II (Gastin, 2001)
R: Treadmill running, C: Cycling
MAODI: Maximal accumulated oxygen deficit using individual mechanical efficiency; MAODA: Maximal accumulated oxygen deficit using assumed mechanical efficiency; MM: Mathematical modelling; DM: Direct measurement of muscle metabolites

2.3.4 Relative energy contribution to sprint swimming

Several studies measuring accumulated oxygen deficit (AOD) have been carried out in a swimming flume by Troup and associates (Troup 1992a, 1992b, 1992c). An adaptation of the AOD test, first proposed by Hermansen and Medbø (1984), was used. Calculations were made from the following measurements and estimates:

- O₂ demand - product of O₂ cost and the duration of each swim
- O₂ deficit - difference between accumulated O₂ uptake and estimated O₂ demand for a given work intensity
- Maximum accumulated O₂ deficit (MAOD) - estimate of anaerobic capacity

Swimming economy (the oxygen uptake required for a given swimming velocity; Troup and Daniels, 1986) and maximal oxygen uptake were also established in a swimming flume. Using this method, Troup et al. (1992b) estimated anaerobic energy contribution over race distances of 100, 200 and 400 m (about 55 s, 120 s and 240 s respectively) at ~46, 28 and 14% for “high performers” and ~50, 35 and 22% for “low performers”, respectively. Results showed a large inter-individual variation in energy contributions to swimming, but the better performers had higher $\dot{V}O_{2\max}$ and MAOD values. Anaerobic capacity (estimated by MAOD) was a significant predictor of 100 m freestyle performance ($r=0.77$).

Troup et al. (1992c) considered the differences in energy contributions during different training interval sets. Sets contained the same total distance (6 x 200 m; 12 x 100 m; 24 x 50 m) with 1:2 work to rest ratios, and swum at the same intensity. The anaerobic component (as determined by MAOD) increased with shorter work durations and

therefore the aerobic energy contribution was found to be lowest in the 24 x 50 m set. It was not clear exactly how intensity was compared across training sets, although the similar pace used appears to be unrealistic based on a swimmer's ability to swim interval training sets at faster paces during shorter repetitions. Analysis of one interval training set, 12 x 100 m, with 3 work to rest ratios (1:2; 1:1.5; 1:1) found a faster speed of adjustment to $\dot{V}O_2$ demand with shorter work to rest ratios reflecting a greater aerobic contribution (Troup et al., 1992a). Longer rest between intervals produced higher oxygen deficit values (and therefore estimated anaerobic contribution).

Recommendations given to coaches about the relative contribution of each energy system to competitive events and training has followed the traditional estimates made from oxygen debt measurements. Table 2.3 compares the traditional textbook estimates for relative energy contribution to sprint swimming with more recent studies involving oxygen deficit measurements and mathematical modelling based on muscle metabolite data. Most of the textbook recommendations are adapted from a review by Houston (1978) and the text book by Eddington and Edgerton (1976), and are probably based on original oxygen debt measurements from the study by Fox et al., (1969).

It is obvious that a lack of direct measurements makes much of the values speculative, but recent attempts to quantify energy contribution by Ring et al., (1996) and Troup (1990) appear to follow the trends for a greater aerobic contribution to sprint swimming seen in other exercise modes (Table 2.3).

Table 2.3 Relative contributions of energy systems during sprint swimming (expressed as a % of total energy supply)

		50m			100m		
Author	Method	PCr	Glycol	Aerobic	PCr	Glycol	Aerobic
<i>Swimming texts</i>							
Allen (1981)	Not stated	90		10	80		20
Bartels (1982) ¹	Based on O ₂ debt				81.5		18.5
Bonen (1979)	Not stated	98	2	0	80	15	5
Costill (1992)	Not stated	80	15	5	70	20	10
Councilman (1977) ²	Not stated	85		15	60-70		30-40
Maglischo (1982)	Not stated	78	20	2	25	65	10
Maglischo (1993)	Not stated	50	48	2	25	65	10
Sharp (1991)	Not stated	60	30	10	20	55	25
Troup and Reese (1983)	Not stated	98	2	0	80	15	5
Houston (1978)	Estimation & O ₂ debt				80		20
<i>Original citations</i>							
Morton (1992) ³	MAODI: SB				50-56		44-50
Morton and Gustin (1997) ⁴	MAODI: SB				54		46
Ring et al (1996)	MM	35.2	43	21.8			
Average							
Ring et al (1996)	MM	35	35.9	29.1			
Sprinters							
Ring et al (1996)	MM	22.5	59.7	17.8			
Non-sprinter							
Troup (1990)	MAODI: F	69		31			

PCr: Phosphocreatine resynthesis; Glycol: Anaerobic glycolysis; O₂: Aerobic ATP regeneration
MAODI: Maximal accumulated oxygen deficit; MM: Mathematical modelling; SB: Swim bench; F: Flume
Some authors have included percentages based on duration, rather than distance. These have been placed under the nearest distance and noted below:
¹: Percentages given for 45 s; ²: Percentages given for 25 yards and 100 yards swimming; ³: Range of percentages given for 45 s and 60 s; ⁴: Percentages given for 60 s

2.4 Recovery from maximal sprinting

2.4.1 Introduction and mechanisms

The importance of short-term recovery from maximal sprint swimming is crucial in determining the best strategy for the construction of sprint training sets. Information

regarding the physiological mechanisms that limit sprint performance and the nature of their recovery following sprinting may suggest the best duration and type of recovery to perform during repeated sprints depending on the mechanism that is being stressed. For instance, if maximal sprint speed is prescribed for each sprint bout, recovery will need to be of sufficient duration to allow complete PCr resynthesis and restoration of muscle pH. The purpose of this section therefore is to detail the time-course of recovery mechanisms in different physiological systems in order that training strategies may maximise the effect of subsequent sprint swimming performance.

The use of repeated sprints as a model to examine the influence of metabolites on fatigue is well established (Bogdanis et al., 1996a; Gaitanos et al., 1993; McCartney et al., 1986). The type of exercise (mode, intensity and duration), time-course of recovery and the impact of recovery duration on subsequent performance all help to determine the contributory factors in fatigue as variables differ in their pattern of recovery (Nevill et al., 1996a).

Several studies have considered at least two phases of recovery of power output following maximal exercise; a rapid phase over the first 60-90 s accounting for ~80% of the peak power output (PPO) followed by a slower component lasting for several minutes (Bogdanis et al., 1995; Harris et al., 1976; Hitchcock et al., 1989; Holmyard et al., 1994). McCartney et al. (1986) found a ~20% decline in PPO in a second and third 30 s maximal sprint separated by 4 min rest between bouts. The fourth bout however showed no further decline in PPO. Significant relationships between PPO restoration and PCr resynthesis have been found following 90 s and 3 min recovery, but not when the recovery was extended to 6 min (Bogdanis et al., 1995). It is likely therefore that the

restoration of PPO immediately following maximal sprinting is largely dependent on PCr resynthesis, but that others factors are responsible for the longer-term (>3 min) restoration of PPO in recovery.

2.4.2 Ionic imbalance

Despite the dominant role that PCr resynthesis is likely to play in the first stages of recovery, recent research suggests ionic balance is also important in the immediate, rapid restoration of power output (Cherry et al., 1998). Rapid (<2-3 s) recovery of power output in sprint swimming is relevant within stroke cycles where contracting muscle alternates with brief recovery periods. In addition (and particularly during 25 m short course; SC swimming), periods of turning during the race provide brief periods of recovery in the propulsive muscles of the upper body. It is likely that this mechanism is, at least in part, responsible for the differences between success in SC and 50 m long course (LC) sprint swimming performance.

2.4.3 Phosphocreatine

Muscle analysis immediately post-30 s sprint exercise has regularly demonstrated PCr values between 65 and 70% lower than at rest (e.g. Jacobs et al., 1982; McCartney et al., 1986; Nevill et al., 1989; Withers et al, 1991) and has been found to be almost completely depleted following a second bout (Bogdanis et al., 1996a). The recovery of PCr and restoration of muscle force output is either closely linked (Bogdanis et al., 1995; Sahlin and Ren, 1989) or unrelated throughout recovery (Hultman et al., 1983; McCartney et al., 1986), which may be dependant on the duration and intensity of the exercise, muscle fibre composition, or the recovery sampling time used. Harris et al. (1976) found a PCr resynthesis half time (recovery time to reach 50% of the difference

between post-exercise and the resting value) of 22 s in 40-55 s isometric contractions or 21 s in ~9 min cycling. Bogdanis et al. (1995) found a slower mean PCr resynthesis rate (mean half time 56.6 s), although a large range in resynthesis rates were reported (half time ~25 s to ~85 s). Despite the differing results, both studies demonstrated similar initial average resynthesis rates. It is likely that the difference in exercise duration and mode, and the type of recovery (active or passive) results in a differing PCr resynthesis pattern (Bogdanis et al., 1995). Recovery of PCr was greater in ST (~68%) than FT (~50%) fibres 60 s after 30 maximal voluntary knee extensions (Tesch et al., 1989). Such information is crucial in constructing sprint training sets depending on the degree of PCr resynthesis required, and suggests that different recovery durations should be used between sprint and endurance-trained swimmers during sprint training.

Phosphocreatine resynthesis is dependent on adequate circulation and oxygen availability. Sahlin et al., (1979) incubated muscle samples in either O₂ or nitrogen following 6 min of exhaustive cycling. Fifteen minutes post-incubation, PCr in the O₂ environment had risen to 68% of the pre-exercise values, whereas there was almost no change in muscle PCr content of the sample kept in nitrogen. When blood flow is occluded to the muscle during and after exercise, PCr resynthesis is attenuated (Harris et al., 1976), supporting the importance of O₂ in PCr recovery. Training strategies that can improve muscle O₂ supply are likely to enhance PCr resynthesis and improve subsequent sprint swimming performance.

2.4.4 Lactate and pH

Lactate efflux from muscle is dependent on extracellular concentrations of both H^+ and HCO_3^- (Jones and Heigenhauser, 1992), and diffuses across the muscle membrane by either active or passive mechanisms (Hultman et al., 1980). Studies in which subjects have ingested sodium bicarbonate ($Na^+ HCO_3^-$) have been shown to increase blood pH and base excess (Costill et al., 1984) whilst improving interval swimming performance (Gao et al., 1988). It was suggested that the improved performance was related to an increased extracellular HCO_3^- that increased the H^+ concentration gradient, allowing a greater removal of H^+ from the muscle. An increased glycolytic activity was proposed as the main cause in performance improvement as blood lactate concentrations were significantly elevated following HCO_3^- ingestion.

Once diffused out of the muscle, lactate is distributed through the extracellular fluid and can be taken up by less active muscles and oxidised (Gollnick and Hermansen, 1973) or transported to the liver through the Cori cycle where gluconeogenesis converts lactate back to glucose and then glycogen by an energy dependent process (Brooks, 1986). The fate of lactate through either of these mechanisms appears to be dependent on the intensity and duration of the exercise, with local muscle oxidation being the major source during and immediately following maximal exercise (Brooks, 1991; Gollnick and Hermansen, 1973). Greater circulation and O_2 supply to muscles following sprint swimming will aid lactate and H^+ removal, and are therefore likely to be enhanced by active recovery following exercise (Gladden, 1989; see section 2.4.7).

2.4.5 Recovery of heart rate and oxygen consumption following sprint swimming

Heart rate (HR) and oxygen consumption ($\dot{V}O_2$), as in endurance exercise, begin to drop following maximal swimming exercise, although measurements of HR (Treffene et al., 1978) and $\dot{V}O_2$ (Lavoie et al., 1983; Costill et al., 1985c) taken immediately post-exercise are useful estimates of maximal values. The pattern of recovery is similar in both HR and $\dot{V}O_2$, and follows an exponential path back to resting levels as illustrated in Figure 2.4. Active (as opposed to passive) recovery will flatten the HR and O_2 recovery curves, but show a steeper (i.e. a faster rate of recovery) slope in the removal of both blood and muscle lactate (Carzorla et al., 1983).

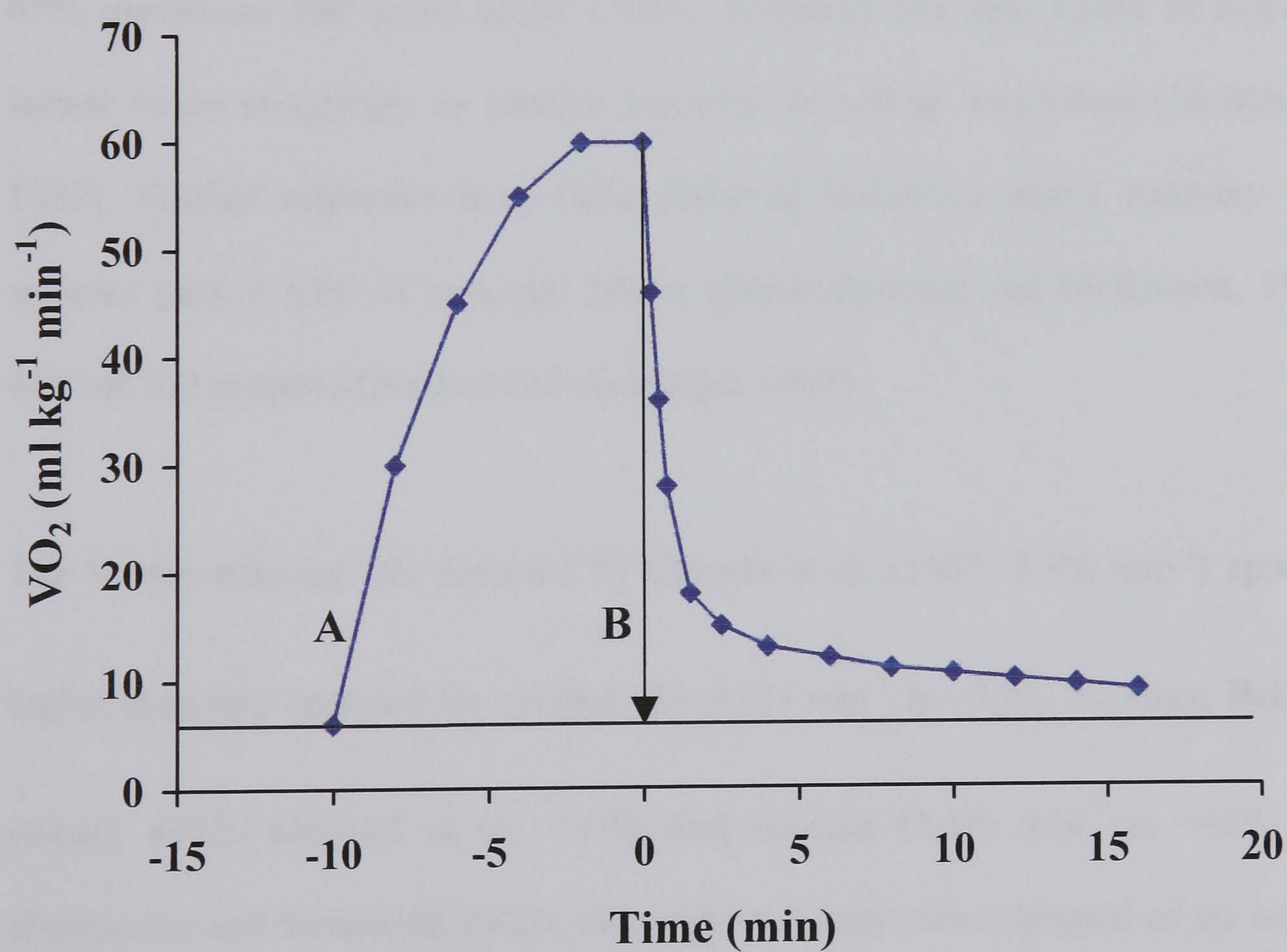


Figure 2.4 Oxygen consumption during exercise (A: -10 min to B: 0 min) and recovery (B onwards). The arrow represents the cessation of exercise.

2.4.6. Active recovery and post-exercise lactate removal

Lactate removal during recovery is consistently faster during active rather than passive exercise, but the rate is dependent on the intensity of recovery (Belcastro and Bonen, 1975), the final lactate concentration achieved (Issekutz et al., 1976), the fibre type of the subject (Bonen et al., 1978), the individual's training status (Taoutaou et al., 1996) and the muscle mass involved in exercise (reflective of exercise mode; Weltman et al., 1977). Following maximal swimming, Cazorla et al. (1983) found no significant differences between intensities of 60 and 75% of 100 m speed and a self-selected recovery speed. These three recovery intensities were equivalent to ~55%, 70% and 73% $\dot{V}O_{2\max}$ speed respectively and improved the blood lactate removal rate (reduction in blood lactate per s) by approximately three times. Recovery intensity of 65% maximum 100 yards speed (~65% $\dot{V}O_{2\max}$) has also found to remove blood lactate twice as quickly as passive recovery in college swimmers (McMaster et al., 1989). Similar responses have been observed following active recovery at a self-selected pace (~63% of maximal 100 m speed; Raeburn and McKinnon, 1990) or at 60% of 500 m speed (Beckett and Steigbigel, 1993).

The lactate removal rate reported by Cazorla et al. (1983; 5.3% min^{-1}) appears to be higher than that reported for cycling (2.9-3.2% min^{-1} at ~30% $\dot{V}O_{2\max}$; Belcastro and Bonen, 1975; McGrail et al., 1978) and running (4.5% min^{-1} at ~63% $\dot{V}O_{2\max}$; Hermansen and Stensvold, 1972), although the competitive standard of the subjects and the lower intensities selected may be responsible for the variations. The variation in intensities reported to enhance blood lactate removal is likely to be related to the

different standards and training status of the swimmer and assuming experienced and educated swimmers, a self-selected pace may provide the most practical and accurate method for individual prescription. Figure 2.4 compares the effect of active recovery on blood lactate removal following sprint swimming.

Table 2.4 The effect of active recovery after sprint swimming on Lactate removal.

Author	Subjects	Recovery Mode	Exercise Intensity	Post-La	La removal rate	Recovery intensity	Recovery duration
Beckett & Steigbigel, 1983	16 CS (8M, 8F)	Swim	200 yd race or 400 yd max	7.5 6.9	0.15-0.16 (2.0-2.3)	2 x 500 m 60% pace	15
Cazorla et al., 1983	7 MNS	Swim	3 x 100 m max, 5 min rest	11.3	HT 11.4-11.9	60-70% (100)	20
Felix et al., 1997	10 FCS	Swim	2 x 200 yd max, +14 m rest	~6.0	0.2 (~10) 0.15 (~15)	65% (200) 60% HRM	14
Krukau et al., 1987	30s	Swim or cycling	200 yd max	-	HT: 5.7 S 12.8 C	S-S	30
Lowden, 1992	6 MNS	Swim	200 yd max	9.5-11.4	0.3-0.5 (3.4-4.7)	65% (200)	10
McMaster et al., 1989	6 NS (3M, 3F)	Swim	200 yd max	7.2-8.0	0.3-0.4 (4.5-5.3)	65% (200)	15
Reaburn & Mackinnon, 1990	4 MMS	Swim	100m race	11.9-14.3	HT 12.55	63% (100)	20

S: Swimmers; N: National level swimmers; C: College; M: Males; F: Females; MMS: Males Master Swimmers. HT: Half-time (min); Post-: Post-exercise; La: Blood lactate (mmol l⁻¹); S-S: Self-selected pace. Rate of lactate removal: top numbers (mmol l⁻¹ min⁻¹), bottom numbers (%/min in brackets). Intensity of recovery: only the most efficient intensity is presented, numbers in brackets: distance from which the pace of recovery was calculated; HRM: Heart rate maximum; Duration of recovery (min)

2.4.7 Active recovery and subsequent performance

The enhancement of blood lactate removal following sprint swimming (Cazorla et al., 1983) has been shown, although the impact of active recovery on subsequent race performance has provided equivocal results (active vs. passive recovery, N.S.; McMurray, 1969). No information is available about the recovery between repeated sprints typical to swimming training or the impact of brief (<30 s) sprinting on subsequent sprint swimming performance, although information is available from other

exercise modes. Enhancement of PPO in a second cycle sprint following active recovery at 40% $\dot{V}O_{2\max}$ as opposed to passive recovery has been shown (Bogdanis et al., 1996c) suggesting that an enhanced blood flow to the muscles following maximal sprinting aided H^+ efflux from the muscle and improved the O_2 availability for PCr resynthesis (see section 2.4.3). Similar enhanced recovery of power output was found between four 15 s sprints with 4 min rest when cycling recovery was performed at 40% $\dot{V}O_{2\max}$ rather than passive rest (Connell and Maile, 1996). Other studies typically have used longer durations between sprints. Weltman (1979) and Weltman and Regan (1983) found no difference in maximal, constant-effort 5 min cycling performance despite a range of recovery exercise intensities, although blood lactate values were significantly lower following active, rather than passive, recovery.

Active as opposed to passive recovery has improved performance (higher pedal speed) in a second maximal sprint (Bogdanis et al., 1996c; Weltman et al., 1977). It is possible that the intensity and nature of the prior exercise will determine the most effective recovery protocol. Power output has been improved during repeated 6 s cycle sprints when either 30 s or 5 min active recovery has been conducted in comparison with passive recovery (Ahmaidi et al., 1996; Signorile et al., 1993). No differences between active swimming (self-selected pace) or passive recovery were observed in a 200 yards maximal swim following a standard swimming ergometer test, although the short rest interval (3 min) and lack of any blood lactate values reported make it difficult to draw any real conclusions from this report (McMurray, 1969). A more recent study found that both swimming and rowing active recovery attenuated performance, compared with passive recovery between two maximal 200 yards swims (Felix et al., 1997). Post- 200

yards blood lactate values of ~4-8 mmol l⁻¹ suggest that the performances were not truly maximal. Figure 2.5 describes the effects of active, compared with passive, recovery on subsequent performance(s).

Table 2.5 Effects of active recovery on subsequent exercise performance.

Author	Recovery Exercise		Blood lactate		Performance
	Mode	Type	Post-bout 1	Pre-bout 2	
Ahmaidi et al., 1996	C	5 min @ 32%	5.0-6.5 A 6.0-7.5 P	5.6-6.7 A 7.5-8.5 P*	PO Increased 7 x 6 s sprints*
Bangsbo et al., 1993	IE	10 min @ 10W	6.3	7.8 A vs. P (n.s.)	TE: 12% longer (n.s.)
Bogdanis et al., 1996	C	4 min @ 40%	9.0	12.6 A vs. P (n.s.)	A: PO *Increase 30s sprint
Bond et al., 1991	C, IE	20 min @ 30%	9.1 A 9.2 P	3.5 A 7.1 P	IE Performance (n.s.)
Felix et al., 1997	S	10 min @ 65% of 200 yards best	4.3	-	A S: Same time A R: + 1 s
	R	10 min @ 60% of HR Max	5.0 5.3 A	-	P: + 2.4 s*
McMurray, 1969	Swim	3 min, SS	-	-	200 yards swim (n.s.)
Siebers and McMurray, 1981	Walk	SS, mean values: W 33%	10.7	7.5 Walk	200 yards swim:
	Swim	S 43%		5.0 Swim	Swim vs. Walk (n.s.)
Signorile et al., 1993	C	30 s @ 360 kg m min ⁻¹	-	-	A: PO *Increase 8 x 6 s sprint
Thiriet et al., 1993	C	20 min @ 30%	16.0	8.0 A 14.0 P	4 x 2 min @ 130%: A: Preserve PO*
Weltman et al., 1977	C	10-20 min @ 360 kg m min ⁻¹	12.7 A 14.4 P	13.3 A 14.4 P	A: *Increase in pedal speed
Weltman et al., 1979	C	20 min @ 40%	16.0-18.0	5.0-8.0 A 12.0 P	No difference
	C	20 min @ 65%			
Weltman and Regan 1983	C	20 min @ 40%	10.0	4.0 A 5.6 P	No difference

Recovery exercise intensity % of $\dot{V}O_2$ max; SS: Self-selected
Mode of recovery exercise, S: Swimming; C: cycling, IE: Isokinetic knee extension; R: Rowing
Blood lactate (mmol l⁻¹); A: Active recovery; P: Passive recovery
Performance, PO: Power output, *: Significant difference A vs. P; TE: Time to exhaustion; n.s.: Not significant

2.4.8 Mechanisms behind active recovery

The increase in muscle blood flow during active recovery will help to remove lactate and H^+ from the muscle, constantly providing a better concentration gradient for efflux and buffering. Skeletal muscle is probably able to simultaneously oxidise lactate whilst producing it (Brooks et al., 1991; Gladden 1989), but an increased blood flow will also transport lactate to neighbouring inactive muscles for oxidation, or to the liver for gluconeogenesis. Oxygen uptake is related to arterial lactate production and removal (Brooks et al., 1985) and it would appear that the larger the muscle mass involved in active recovery, the greater the lactate removal (McGrail et al., 1978). An increase in muscle blood flow will also promote faster PCr resynthesis.

One final but important benefit of active over passive recovery involves the indirect contribution of active muscle to the maintenance of vascular pressure and venous return. Muscle blood flow during exercise is dependent on mean arterial and venous blood pressure and the resistance in the capillary beds (local vascular resistance). Blood flow increases if the mean arterial blood pressure increases or local vascular resistance decreases. Conversely if mean venous blood pressure or local vascular resistance increase, blood flow will decrease. Immediately following maximal exercise, the increased blood pressure drops rapidly if rest is passive. Active recovery however will help to maintain blood flow through the ‘muscle pump’ mechanism that repeatedly squeezes vessels and forces blood back to the heart. Figure 2.5 illustrates this process.

It is likely that a combination of the factors detailed that are responsible for enhanced performance following active, as opposed to passive recovery, although the mode,

intensity, duration and type of prior exercise will dictate the best strategy to optimise recovery and improve subsequent performance.

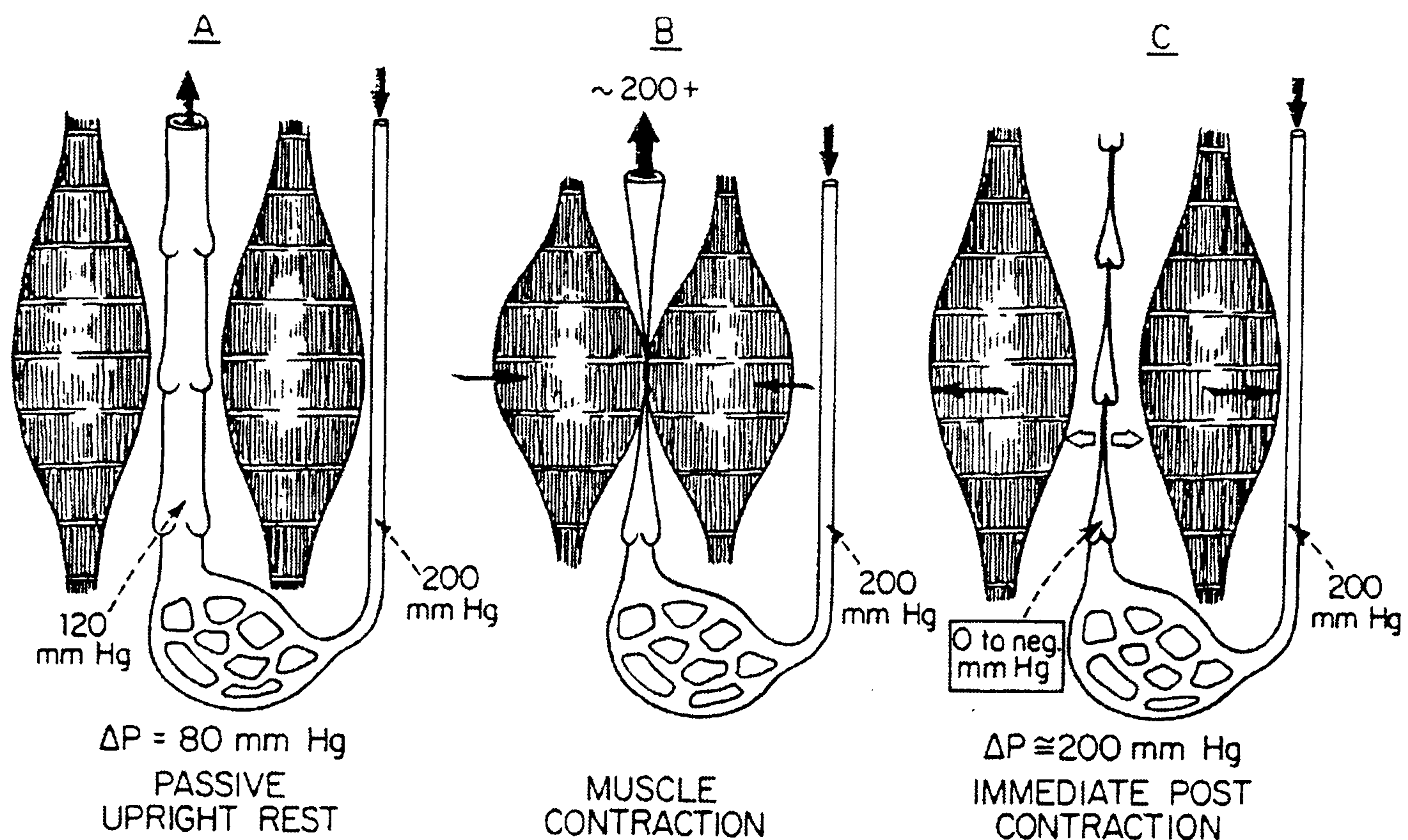


Figure 2.5 The muscle pump. A. Passive, upright rest; arterial and venous driving pressure are 200 and 120 mmHg respectively, with a net pressure into the veins of 80 mmHg. B. Muscle contraction empties the veins, momentarily blocks arterial inflow and greatly increases venous driving pressure back towards the heart (~200 mmHg). C. Immediately following contraction, pressure in emptied veins falls to zero and arterial to venous driving pressure is momentarily raised to 200 mmHg in upright posture (from Rowell, 1993).

2.5 Growth hormone responses to sprint swimming

Growth hormone (hGH) is the most abundant hormone secreted from the anterior pituitary gland and regulated by a neuroendocrine, 'negative' feedback mechanism. Following intense exercise, hGH stimulates the release of insulin-like growth factors

(IGF) which stimulate intracellular and extracellular growth actions involved in cellular repair and growth (Kraemer et al., 1992). Release of hGH into the blood is stimulated by many factors such as deep sleep, hypoglycemia (and fasting), amino acids (and therefore high protein meals) and stress, in addition to intense exercise. Conversely, its release is inhibited by hyperglycemia, cortisol, free fatty acids and high concentrations of hGH itself. The regulation of hGH appears to be under the control of two hormones produced by the hypothalamus; release is stimulated by growth hormone releasing hormone (GHRH), and inhibition mediated by somatostatin.

A substantial, acute hGH response has been shown following maximal sprint exercise, which is suggested to be “near maximal” (Nevill et al., 1996b). A single 30 s bout on a sprint treadmill elicited mean values of $\sim 40 \mu\text{g l}^{-1}$ in sprint males although some appeared to reach $\sim 60 \mu\text{g l}^{-1}$. These values were higher than for a mixed group of trained, male students in sprint cycling who recorded a mean value of $18 \mu\text{g l}^{-1}$ with a range of $\sim 4\text{-}40 \mu\text{g l}^{-1}$ (Stokes et al., 2000). The large range indicating a large inter-individual difference in the hGH response has led researchers to consider the total hGH release over time, and not just the peak value (Raynaud et al., 1981). The range in individual responses is seen in both the total hGH release and the time to peak, as well as the peak value itself, which is observed between ~ 15 and 40 min after a single 30 s sprint (Stokes et al., 2000).

Opinion differs between researchers on the effect of training on the growth hormone response to exercise. Studies have reported increases (Bonifazi et al., 1998; McCall et al., 1999), decreases (Weltman et al., 1997, Stokes et al., 2000) and no change (Kraemer et al., 1990) in post-exercise concentrations of hGH following training. Studies that

have demonstrated a blunted response to the exercise-induced hGH response following training (Stokes et al., 2000; Weltman et al., 1997) follow observations that a second sprint attenuates the hGH response (Cappon et al., 1994; Stokes et al., 2002). Whether this is due to a learning effect of the test, suppression of the autonegative feedback system or the training content undertaken, is not clear. In contrast, a longer-term enhancement of the hGH response has been observed by Bonifazi et al., (1998) following 18 weeks of mixed training in elite swimmers, although the exercise test used was a submaximal endurance training set. Further research is needed to determine the hGH response to sprint swimming.

2.6 Muscle strength and power

2.6.1 Strength, power and swimming performance

Strength and power have been identified as important factors in the contribution to swimming success and are regular elements of the training programmes of top swimmers (Strass 1988; Troup et al., 1994). Studies by Costill and co-workers in the early 1980's found strong correlations between sprint swimming speed and maximal swim bench arm power. Costill et al. (1980) found relationships of $r = 0.93$, 0.89 and 0.87 between maximal swim bench arm power and 25, 100 and 200 yards swimming time respectively. This relationship was confirmed in 40 male and female competitive swimmers by Sharp et al. (1982) using similar test protocols ($r = 0.90$).

When subjects are more homogeneous (USA National level), the relationship between swim bench power and swimming speed is weakened considerably ($r = 0.62$; Sharp et al., 1983). It would appear that swim bench peak power is not discriminatory between high-level swimmers, but that specific application of the power throughout the

swimming race is more important (Cappaert et al., 1992). Costill et al. (1992) reported better relationships between tethered swimming peak power and swimming time for 25 m ($r = 0.84$) than for swim bench peak power in a homogeneous group, as it was more specific to the mechanics and force application during swimming and could account for ~80% of the success in front crawl sprinting (Costill, 1999).

Following 6 weeks of strength training (in addition to swimming training), swimmers improved mean arm extension force by 12.5% and mean swimming speed by 7% and 7.3% in 25 m and 50 m respectively (Strass, 1988). Mean stroke length improvement (~8%) was suggested as the cause of this improvement, although rate of force development increased by nearly 25%. Miyashita and Kanehisa (1983) reported that isokinetic and isotonic training at speeds of 180° s^{-1} improved swimming performance. This transfer was not observed at low training speeds, suggesting a specific adaptation to fast twitch (FT) muscle fibres.

2.6.2 Effect of muscle fibre type on power output

Fast twitch fibres (FT) have greater PCr stores and glycolytic potential than slow twitch fibres (ST) and greater substrate utilisation rates following sprint exercise (Greenhaff et al., 1994c; Spriet, 1995; Nevill et al., 1996a). Muscle fibre PCr resynthesis rate is faster in ST than FT fibres (Soderland and Hultman, 1991) as the phosphocreatine shuttle (see Figure 2.1) appears to be dependent on transport through aerobic metabolism. Fast twitch fibres contract forcefully, but fatigue more quickly in contrast to ST fibres. Not surprisingly each fibre type is specialised with different physiological characteristics that have been adapted for specific contractile patterns (Satlin and Gollnick, 1983). Specific characteristics in FT fibres for high and rapid force development include higher

rates of ATP hydrolysis, actomyosin cycling rate, anaerobic ATP resynthesis and Ca^{2+} re-uptake by SR than in ST fibres (Green, 1990). Correspondingly, ST fibres rely more on aerobic oxidation, providing a more efficient energy supply that is sustainable for longer periods of time.

Resting ATP and PCr concentrations are approximately 50% higher in FT than ST muscle fibres (Fitts, 1992) and three to four-fold times higher ATP utilisation rates have been observed in FT fibres (Spriet, 1990). In addition, Ivy et al. (1987) demonstrated that lactate produced was 33% higher and PCr decline (~58% vs. ~31%) lower in FT compared to ST fibres following high intensity exhaustive exercise. A close relationship has also been shown between blood lactate, blood ammonia and percentage of FT fibres (Dudley et al., 1983).

Muscle fibre typing and their differing characteristics may indicate an individual's ability in sprint or endurance events or perhaps discriminate between sports. McCartney et al. (1983) discovered a 33% difference in power output during sprinting between their best and worst cyclists. The best performer recorded a peak power of ~2500 watts (W) compared to ~1700 W in the poorest cyclist. Fast twitch fibres percentages for the two subjects were 72% and 50% respectively. The higher percentage of FT fibres in addition to a larger muscle mass was attributed to the improved performance and could account for ~85% of the variance in maximal power output (Faulkner et al., 1992).

2.6.3 Muscle fibre types in swimmers

Large individual variations are reported in the literature for the fibre types of competitive swimmers. Table 2.6 illustrates this range and confirms the belief that fibre

type is a poor predictor of swimming ability in competitive swimming. Despite this, each fibre type will have different characteristics that may indicate the type of event and the likely metabolic responses. Studies have not related fibre type to specific events or distances, although the large variation in fibre types in competitive swimmers suggest that factors other than fibre typing such as technique, measured by economy swimming, may be better determinants of success (Troup et al., 1994).

Table 2.6 Mean percentage muscle fibre compositions in competitive swimmers

Study	Muscle	Subjects	FTa	FTb	ST
Costill et al. (1978)	PD	Sp		60-65	35-40
Costill et al. (1985a)	PD	WTC		32	68
Gerard et al. (1986)	VL	EM Sp	3.6	48.6	47.8
		EM End	2.9	36.5	60.6
		EF Sp	2.3	45.7	52.0
		EF End	0.3	37.7	62
Gollnick et al. (1972)	PD	CS		25	75
Houston et al. (1981)			29	9	62
Lavoie et al. (1981)	T	EM		50	50
		EF		60	40
Nygaard and Nielson (1978)			41	19	40
Prins (1981)	VL	Sp		76	24
		End		31	69
Trappe et al. (2001)	PD	WTC	35.5		64.5
Troup et al. (1994)	PD	WR		72	38

PD: Posterior deltoid; T: Ticeps brachii; VL: Vastus lateralis
Sp: Sprint swimmers; End: Endurance swimmers; WTC: Well-trained college swimmers; CS: College swimmers; EM: Elite male swimmers; EF: Elite female swimmers; WR: World record holder - 400 Front crawl

2.7 Cardio-respiratory function

2.7.1 Oxygen uptake in swimming

The introduction of a new testing ergometer (“A swimming flume”, Astrand and Englesson, 1972) allowed the first detailed measurements of respiratory function in swimming. Initial studies noted a linear relationship between O₂ uptake and speed

(Holmer, 1974a; 1974b), although subsequently, it was thought that an exponential model may fit the incremental response to swimming in high-level swimmers at faster speeds (Holmer, 1979). Higher level swimmers generally find it awkward to swim at slower speeds and consequently expend relatively more energy (as indicated by O_2) for a given speed. Similarly, at high velocities, a disproportional increase in drag demands relatively more energy expenditure per unit of speed increase (Holmer, 1979). Front crawl, in addition to being the fastest competitive stroke is also the most efficient with the contribution of the arms, the main source of propulsion in all strokes except breaststroke (see Figures 2.6 and 2.7 from Holmer, 1974a; 1974b).

Maximal oxygen uptake values of $3.5 - 4.2 \text{ l min}^{-1}$ have been reported for competitive swimmers (Bonen et al., 1980; Magel and Faulkner, 1967; Takahashi et al., 1992), although this figure rises to $\sim 5.0 \text{ l min}^{-1}$ in elite swimmers (Hermansen and Karlsson, 1967; Holmer, 1974a). Typical $\dot{\text{V}}\text{O}_2$ max ranges are $\sim 4.0 - 5.9 \text{ l min}^{-1}$ for male and $\sim 2.9 - 3.7 \text{ l min}^{-1}$ for female elite swimmers (Holmer et al., 1974a).

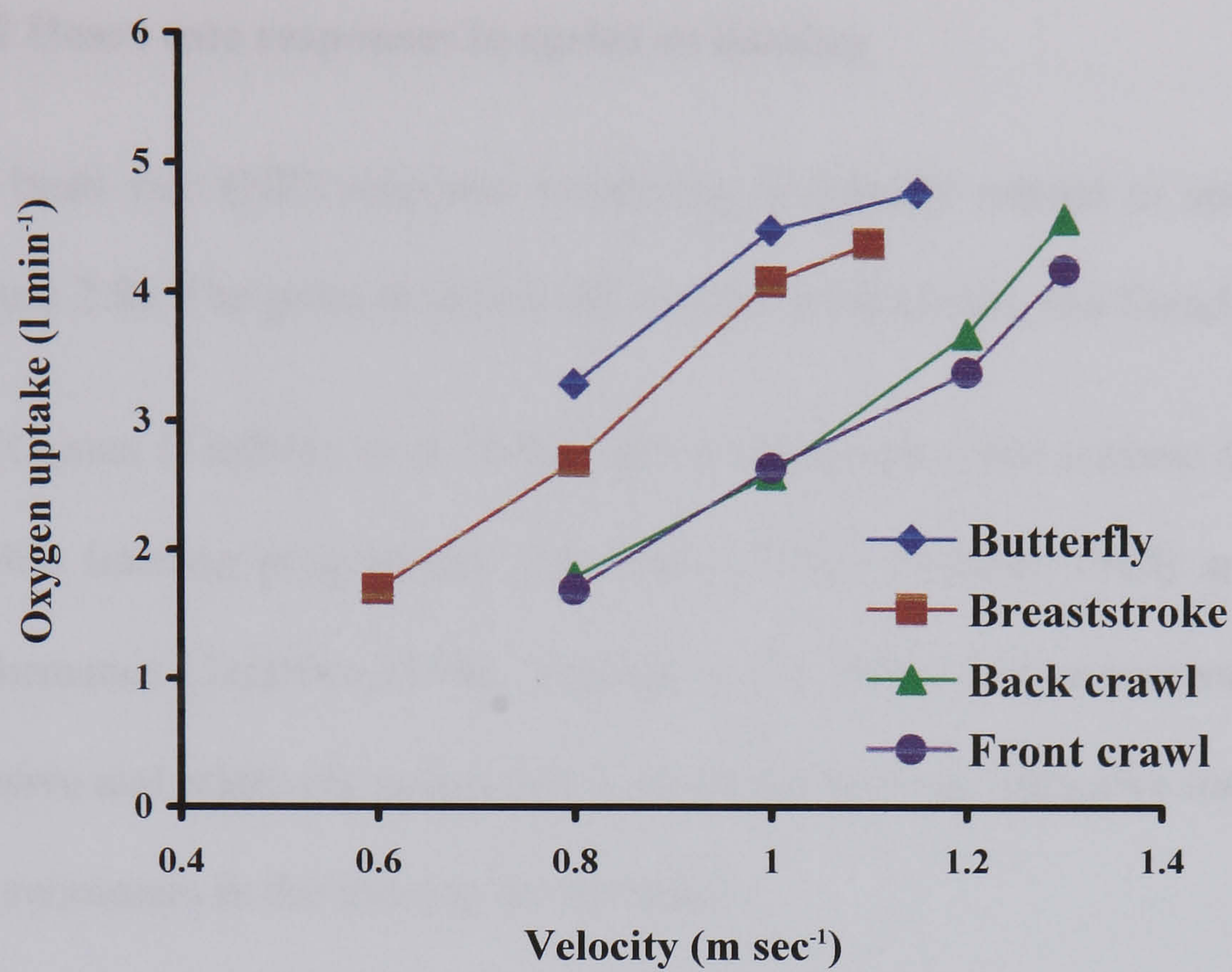
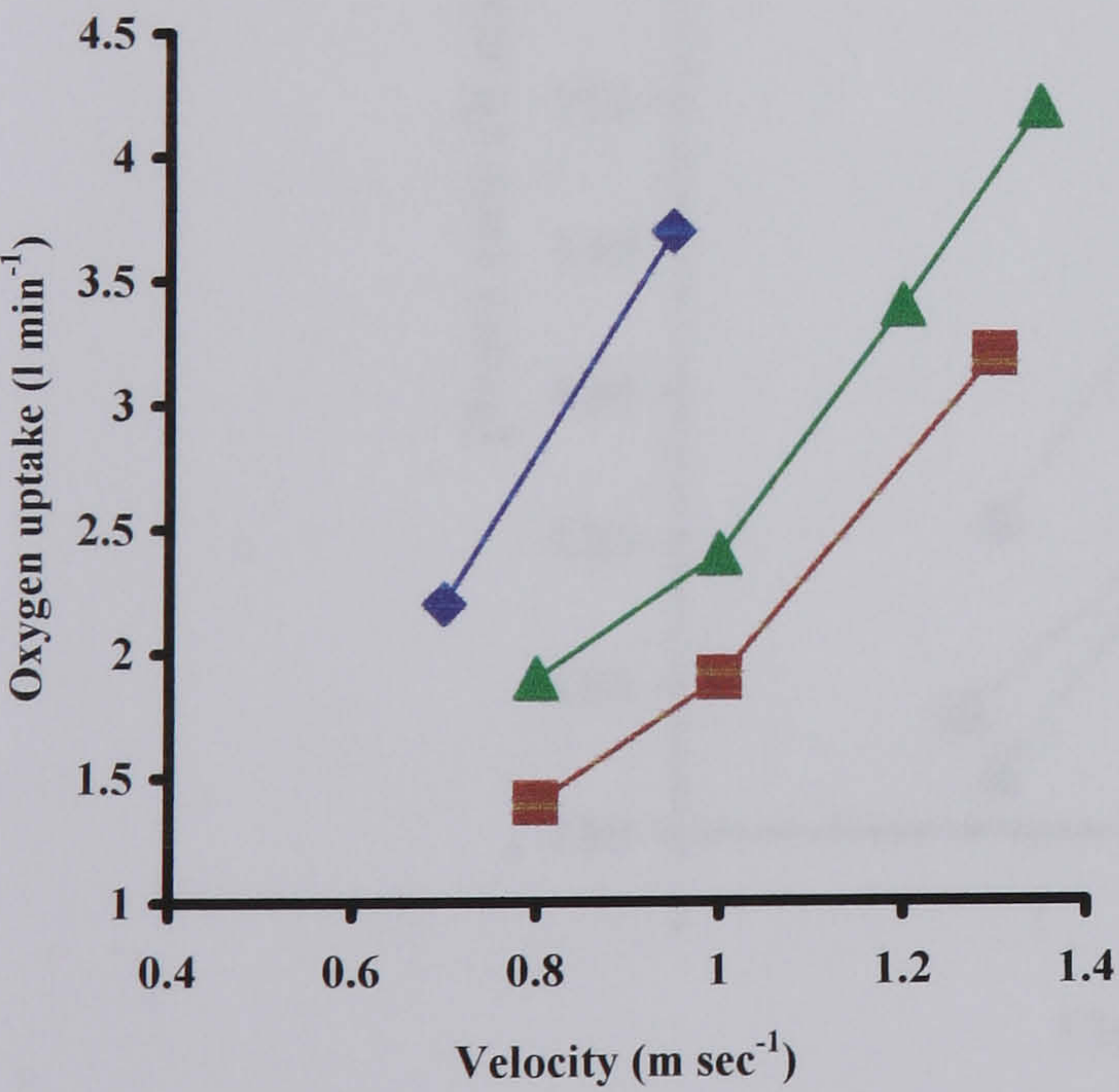


Figure 2.6 Mean oxygen uptake values for flume swimming at different velocities in the four competitive strokes (adapted from Holmer, 1974b).

Front crawl



Breaststroke

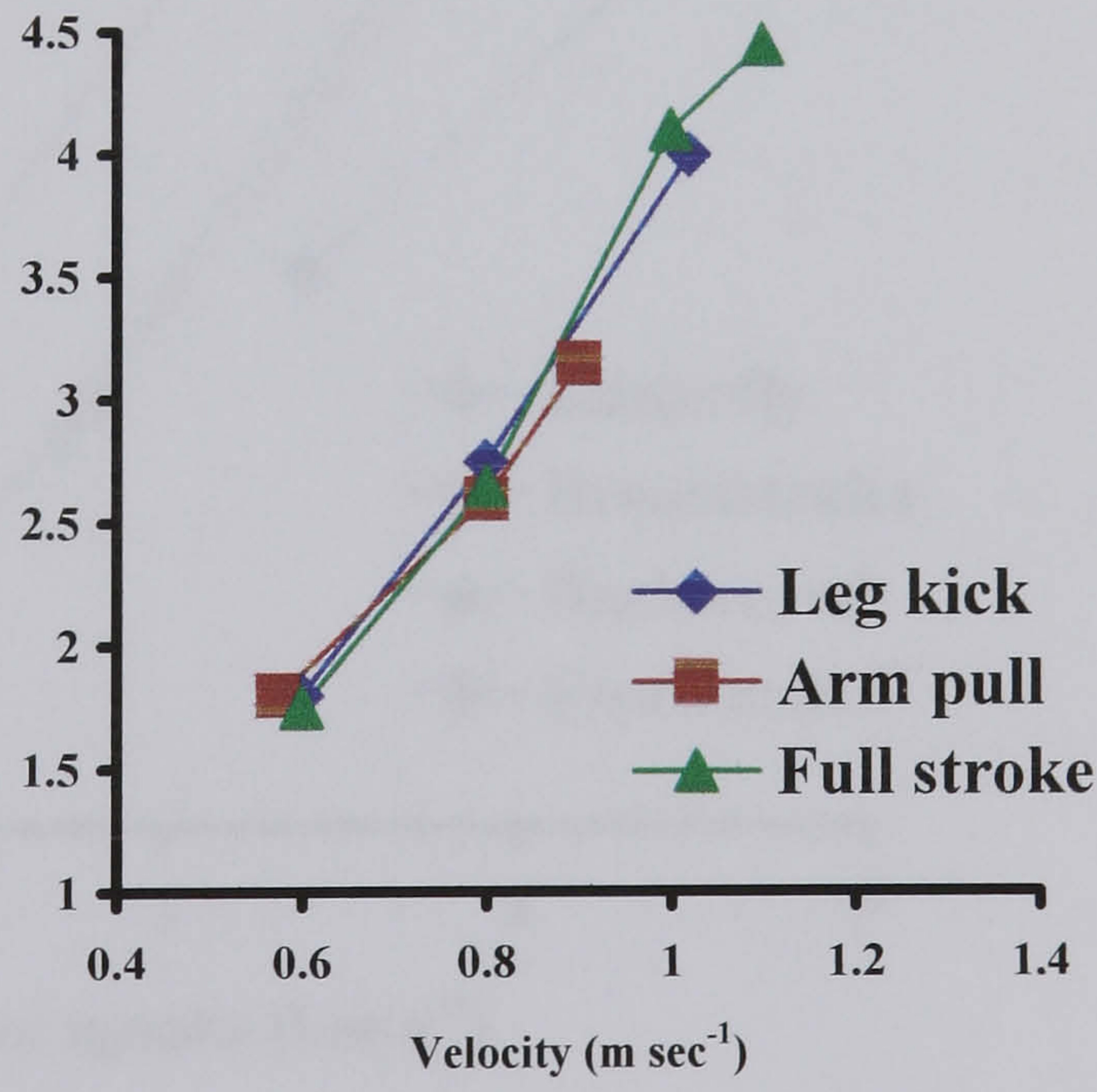


Figure 2.7 Mean oxygen uptake values for flume swimming at different velocities for arm pull, leg kick and full stoke during front crawl and breaststroke (adapted from Holmer, 1974b).

2.7.2 Heart rate responses to sprint swimming

The heart rate (HR) response swimming is linearly related to speed and to $\dot{V}O_2\text{max}$ (Figure 2.8). The point at which HR reaches a maximum has found to be closely related to $\dot{V}O_2\text{max}$ (Treffene et al 1978a) and is used with some success in the prescription of aerobic training programmes (Treffene 1978b; Treffene 1980) and the prediction of performance (Treffene 1978a; Treffene et al., 1983). HR measurement is practical non-invasive and relatively inexpensive, which makes it an attractive tool for use by coaches and swimmers in the training environment.

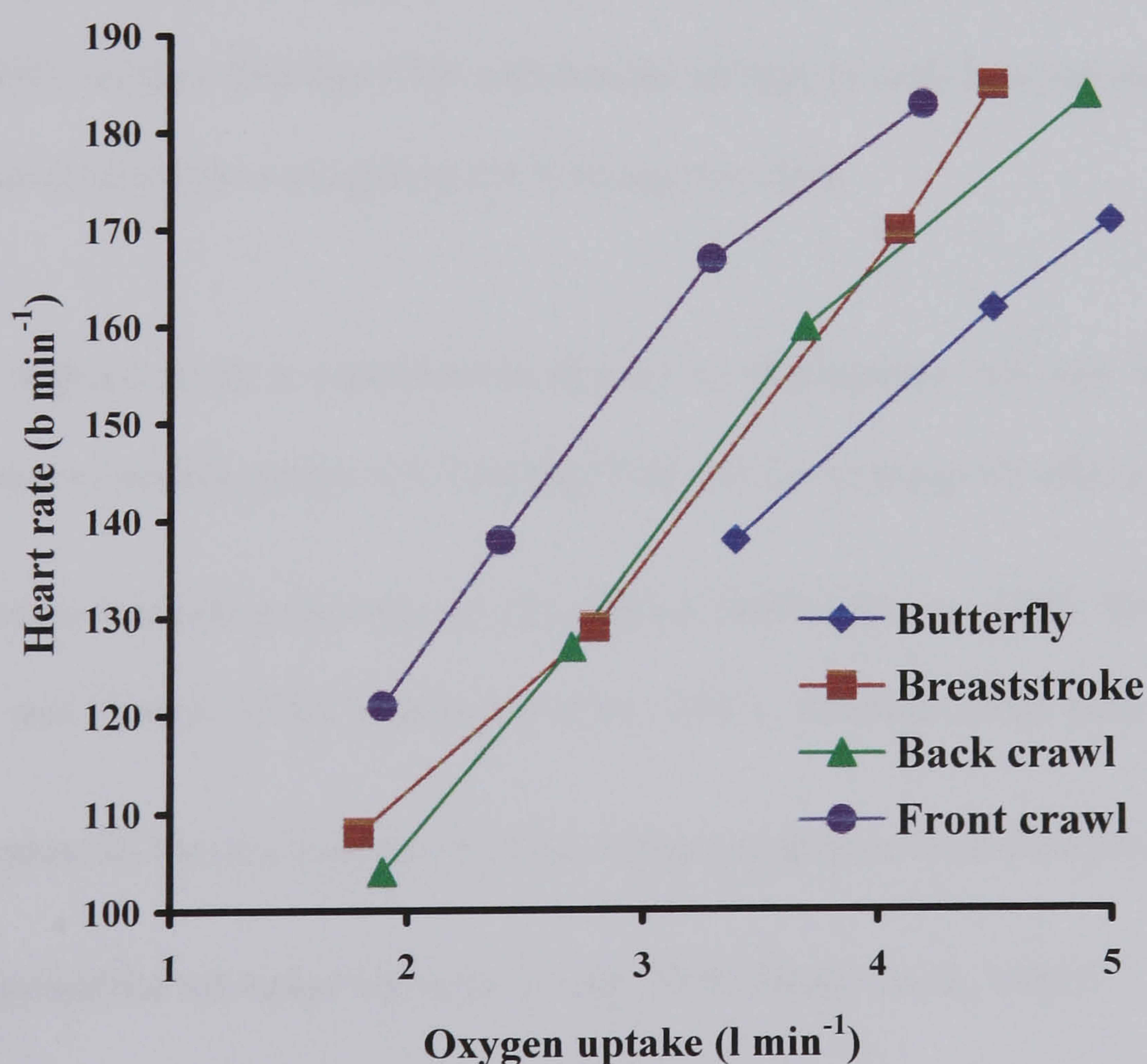


Figure 2.8 Mean values for heart rate in relation to oxygen uptake in the four competitive strokes during flume swimming (adapted from Holmer, 1974a).

2.7.3 Controlled frequency breathing

Hypoxic training or controlled frequency breathing (CFB) became popular in the 1970's following a number of authors who promoted its use in training with the intention of either simulating the effect of altitude training or stressing anaerobic energy systems through reducing the oxygen delivery to the working muscles (Councilman, 1978; Kedrowski, 1979). It is generally accepted that the effect of reducing breathing frequency will not necessarily create a hypoxic condition at the working muscles, but create hypercapnia (an increase in the arterial pressure of carbon dioxide – PaCO_2) and therefore the desire to breath (Coatsee and Terblanche, 1988; Craig, 1979; Matheson and McKenzie, 1988; Stager et al., 1986; Town and Vaness, 1990; Yamamoto et al., 1988). This response has lead to some authors suggesting a renaming of the practice to hypercapnia tolerance training as opposed to hypoxic training (Craig, 1979; Dicker et al., 1980), as they feel that CFB reduces the air supply and does not necessarily induce hypoxia (reduction in oxygen to the working muscles).

There appears to be a considerable amount of discrepancy between the physiological sequence of events observed following CFB and the consequent effects on metabolism.

Most authors report a decrease in $\dot{V}\text{O}_2$ (Hsieh and Hermiston, 1983; Stager et al., 1986; Town and Vaness, 1990; Yamamoto et al., 1987), although some have reported similar $\dot{V}\text{O}_2$ values citing an increase in tidal volume or greater O_2 extraction at the muscle as a compensation for lower $\dot{V}\text{E}$ rates (Craig, 1979; Dicker et al., 1980).

The alveolar partial pressure of oxygen (PaO_2) has also been shown to decline following CFB (see Table 2.7). Craig (1986), Hsieh and Hermiston (1983) and Stanford et al.

(1986) suggest that the magnitude of the drop in PaO_2 is not sufficient to reduce arterial O_2 saturation and therefore O_2 delivery to the working tissues. This may be a reflection of the short duration 4-6 s of CFB used by Craig (1986) simulating breath-holding duration following the turn in competitive swimming events. In the past 15 years, the increase in the underwater phase of the turn needed for success in international competition has grown, with breath-holding durations consequently longer. Perhaps longer breath-holding durations during exercise will demonstrate some reduction in oxygen delivery to the working muscles.

The 'knock-on' effect of hypercapnia has been further explored in attempt to determine the metabolic consequences of CFB. Conflicting blood lactate changes have been reported with CFB; authors reporting higher (Kedrowski, 1979; Matheson and McKenzie, 1988; Yamamoto et al., 1980), lower (Dicker et al., 1980; Graham et al., 1980; Holmer and Gullstrand, 1980; Town and Vaness, 1980) or the same (Ogita and Tabata, 1992, Stanford et al., 1986) values for CFB and normal breathing. This observation is further confused by conflict in the discrepancy between blood and muscle lactate. Graham et al. (1986) found similar muscle lactate values, but lower blood lactate, whereas Yamamoto et al. (1988) reported higher muscle lactate production, but this was not reflected in the blood.

Studies that have reported higher blood lactate values following CFB suggest that a reduction in O_2 delivery and a fall in PaO_2 results in an increased anaerobic energy delivery (Matheson and McKenzie, 1986; Yamamoto et al., 1988). Lower $\dot{\text{V}}\text{O}_2$ and PaO_2 values have also been found in conjunction with lower lactate values (Dicker et

al., 1980; Holmer and Gullstrand, 1980; Town and Vaness, 1980). Explanation for this is provided by Graham et al., (1980; 1986) who suggest that the increase in PaCO_2 has an inhibitory effect on glucose utilisation and glycolysis. The respiratory acidosis induced by hypercapnia may have an effect on both aerobic and anaerobic metabolism and therefore, an increase in energy supply may come from fat metabolism. This may explain the reduced RER values observed by several authors (Dicker et al., 1980; Graham et al., 1980; 1986; Yamamoto et al., 1988).

A fall in the ventilatory equivalent for oxygen ($\dot{V}\text{E}/\dot{V}\text{O}_2$) following CFB has been explained by an increased O_2 extraction in an attempt to maintain $\dot{V}\text{O}_2$ as $\dot{V}\text{E}$ falls. This has been shown by a decrease in $\text{FEO}_2\%$ and greater $\text{FECO}_2\%$ (Fraction of oxygen and carbon dioxide in expired air respectively; Dicker et al., 1980; Town and Vaness, 1980). Table 2.8 summarises the studies that have used CFB as an intervention, although none consider the impact of CFB on metabolism or performance during maximal sprint swimming.

Table 2.7 Summary of physiological and performance responses to controlled frequency breathing

Author(s)	Exercise Mode	Test	Findings: CFB effects
Coatsee and Terblanche 1988	C	36x[10 s +10 s rest]	↑ PO ₂ from ↑ post-exercise VE ↑ blood PCO ₂ and H ⁺ concentration
Craig 1979	C	Continuous Submax	↓ P _A O ₂ , ↑ P _A CO ₂ (hypercapnia) No difference: VE, VO ₂ , VCO ₂
Craig 1986	S	Competition turns	↑ PCO ₂ , ↓ PO ₂ don't affect performance if ≤ 5.5 s ↑ PCO ₂ (hypercapnia)
Dicker et al., 1980	TS	3x4 min	↓ Alveolar VE, ↑ P _A CO ₂ (hypercapnia) ↓ VE offset by ↑ TV and O ₂ extraction ↑ tolerance to hypercapnia, not hypoxia
Graham et al., 1980	C	Incremental (4 min) to exhaustion	↑ blood PCO ₂ , ↓ blood La CO ₂ inhibits glycolysis
Graham et al., 1986	ES	20 m @ 3Hz, dogs	Respiratory acidosis: ↓ blood La Muscle La unaffected ↓ blood La: impaired La from muscle, glycolytic inhibition (PFK) and ↑ lactate oxidation
Holmer & Gullstrand 1980	FS	Submaximal	↓ P _A O ₂ , VO ₂ , blood La ↑ P _A CO ₂ (hypercapnia)
Hsieh & Hermiston 1983	S	15x200y @ 80-88% of 200y PB	↓ VE, ↑ O ₂ extraction ↑ P _A CO ₂ and P _V CO ₂ (hypercapnia)
Matheson & McKenzie 1988	T	5x15 s @120% VO ₂ max	↑ respiratory acidosis Induced hypoxemia ↑ arterial lactate, ↑ reliance on anaerobic metabolism
Ogita & Tabata 1992	FS	Submax VO ₂ max	No difference: VE, VO ₂ , VCO ₂ , HR 12% ↓ VO ₂ max
Stager et al., 1986	AC	VO ₂ max 3x submaximal	≥ CFB6 Acute systemic hypercapnia & arterial hypoxia ≥ CFB6 ↓ VE, VO ₂ , VCO ₂ , ↑ recovery VCO ₂ , RER
Stanford et al., 1986	C	3x8 min LaT, -10%, +10%	↑ H ⁺ concentration, Same La ↓ blood O ₂ , ↑ PCO ₂ No ↓ tissue O ₂ : inhibition of respiratory compensation
Town & Vaness 1990	TS	4 min @ 80% VO ₂ max	↑ P _A CO ₂ , SR ↓ RER, VE, VO ₂ , VCO ₂ , P _A O ₂
Yamamoto et al., 1987	C	10x[30 s + 30 s rest]	↑ A-VO ₂ difference: arterial hypoxemia ↓ Alveolar VE, VO ₂ ↑ Alveolar VE, VO ₂ during rest ↑ P _A CO ₂ (hypercapnia), H ⁺ , HR
Yamamoto et al., 1988	C	16x4 min 30-90% VO ₂ max	↑ Δ La recovery ↑ Muscle La production, not reflected in blood ↓ Alveolar VE, ↑ P _A CO ₂ (hypercapnia)

S: Swimming, T: Treadmill running, ES: Electrical stimulation, AC: Arm cranking, C: Cycling, TS: Tethered swimming, FS: Flume swimming

Δ Change, ↑: Increase, ↓: Decrease CFB6: Controlled frequency breathing for 6 stroke arm strokes, La: Lactate, LaT: Lactate threshold, SR: Swimming stroke rate, TV: Tidal volume, RER: Respiratory exchange ratio, VE: Ventilation, VO₂: Oxygen uptake, VCO₂: Carbon dioxide production, P_AO₂/ CO₂: Alveolar partial pressure of oxygen/carbon dioxide, P_VO₂/ CO₂: Venous partial pressure of oxygen/carbon dioxide, H⁺: Hydrogen ion

2.8 Sex differences in swimming performance and metabolism

Despite prejudices and society opinion, many aspects of male and female physiology are the same. Muscle glycogen storage and the ability to increase supply following training are similar (Eddy et al, 1977). Endurance training responses show similar percentage improvements in cardiac output, oxygen consumption, and muscle enzymes activity (Eddy et al, 1977). However, there are important anthropometric and physiological differences between male and females. Typical population means for percentage body fat are ~15% for males and ~25% for females, although mean values for elite, International swimmers are ~9% and ~16% for male and females respectively (Ackland et al., 1991; Troup et al., 1994). If other body constituents are proportionately equal between male and female swimmers, males will also have a higher percentage muscle mass, perhaps in the region of 5-10% of total body muscle. In addition to muscle mass, it has been demonstrated that larger swimmers have the potential ability to generate more propulsion (Troup et al., 1994). Male swimmers have been reported to be ~7-10% taller and ~30% heavier than their elite female counterparts (Lavoie and Montpetit, 1986; Troup et al., 1994) which may account for the ~9-12% difference between male and female world records (see section 2.9, Table 2.8). Maximal oxygen uptake is closely related to body size, and in elite swimmers, values have been reported ~47% higher in males than females, which may account for some of the difference in performance between the sexes (Holmer, 1974a).

Very few studies have considered sex differences in 'anaerobic power' or 'anaerobic capacity' (Wells, 1986). Improvements of 16% in estimated anaerobic capacity, determined by MAOD following 6 weeks of training, were found in males, but only 5% in females (Medbø and Burgers, 1990). It is possible that 'anaerobic capacity' is more

trainable in males than females, although it is more likely that training status will impact the potential to improve 'anaerobic capacity' more than any sex differences.

Higher blood lactate concentration in males than females have been reported following maximal exercise (Jacobs and Tesch, 1981; Jacobs et al., 1983; Karlsson et al., 1981; Komi and Karlsson, 1978), perhaps due to the larger muscle mass involved during the sprint. Suggested mechanisms for lower blood lactate in females include an inborn 'glycogen sparing' metabolic profile (Karlsson and Jacobs, 1982), less pronounced glycolytic activity in skeletal muscle (Karlsson et al., 1981; Komi and Karlsson, 1978) and a higher capacity for lactate oxidation (Komi and Karlsson, 1978). Higher muscle lactate concentration have been found in male compared to female subjects (Jacobs et al., 1982; Jacobs et al., 1983). Conversely muscle lactate values of ~78 (Cheetham et al., 1986), ~86 (Nevill et al., 1989) and ~113 mmol kg⁻¹ dm (Cherry et al., 1998) fall within the ranges reported for males (~74 mmol kg⁻¹ dm - Jacobs et al., 1983 to ~126 mmol kg⁻¹ dm - Jones et al., 1985), suggesting little support for the contention that females may accumulate less muscle lactate than males. Telford et al. (1988) have shown that in highly-ranked male and female Australian swimmers, post-competition blood lactate values reach similar values.

Significant differences have been reported in performance tests that claim to estimate 'anaerobic power and capacity'; such as the Margaria-Kalamen test (Maud and Shultz, 1986), the Wingate 5s (Ben Ari et al., 1978) and the Wingate 30s and 90 s tests (Serresse et al., 1989). Several factors such as a lower percentage of type IIa fibers (Froese & Houston, 1987), a lower type IIa and IIb fibre area (Simoneau et al., 1985) and lower glycolytic enzyme activities (Komi & Karlsson 1978, Nygaard 1981,

Simoneau et al, 1985) have been suggested for the differences. Despite this, many sprint tests find that differences observed between male and female subjects diminish or are completely removed when values are reported relative to body mass or fat free mass (Bishop et al., 1985; Maud et al., 1986; Wells, 1986).

2.9 Swimming competition

2.9.1 Demands of competitive events

Swimming races range between ~22 s and ~24 s for 50 m events and ~900 s (15 min) and ~500 s (8 min 20 s) for 1500 m/800 m in male and female world class swimmers (Table 2.8). The majority of the events (>80% including relays) last <150 s and are characterised by a large variations in stroke length (SL; 1.78 – 3.23 m in males and 1.7 – 2.24 m in females) and stroke rate (SR; 36.5 – 59.7 strokes min⁻¹ in males and 36.5 – 63.0 strokes min⁻¹ in females). Analysis of swimmers in the finals of the 2000 Olympic games suggest that no pattern exists between the fastest swimmers and either stroke length or stroke rate characteristics, consistent with recent reports (Chengalur and Brown, 1992; Kennedy et al., 1990; Thompson et al., 2000). Clearly, the swimmer who can maintain the longest stroke, highest stroke rate combination throughout the race will be the fastest, but this interaction is individual (Craig and Pendergast, 1979).

Table 2.8 World record swimming times and selected kinematic variables for Olympic Champions

Event	Male			Female		
	WR	SR (st min ⁻¹)	SL (m)	WR	SR (st min ⁻¹)	SL (m)
50 F/C	21.64	59.7	2.17	24.13	63.0	1.85
100 F/C	47.84	51.8	2.31	53.77	54.8	1.95
200 F/C	104.06	46.0	2.39	116.78	47.9	2.04
400 F/C	220.17	36.5	3.23	243.85	55.2	1.95
800 F/C	—	—	—	496.22	53.7	1.74
1500 F/C	874.56	38.2	2.55	—	—	—
100 B/C	53.60	51.8	2.05	60.16	49.6	1.95
200 B/C	115.87	44.1	2.23	126.62	43.7	2.10
100 Breast	59.94	53.5	1.78	66.52	50.5	1.7
200 Breast	130.16	36.4	2.49	142.99	36.5	2.24
100 Fly	51.81	57.2	1.91	56.61	56.6	1.81
200 Fly	114.58	48.8	2.07	125.81	51.9	1.75

Distances in metres (m); F/C: Front crawl; B/C: Back crawl; Breast: Breast stroke; Fly: Butterfly
WR: World record as at 1.11.01; SR: Stroke rate (strokes per minute); SL: Stroke length (metres)
SR and SL values are taken as the mean for the Olympic Champion 2000 (Mason et al., 2000)

An analysis of swimmers over an eight-year period found that performance improvements were due to increases in stroke length and not stroke rate (Craig et al., 1985). This change will allow swimmers to swim at the same speed for a lower stroke rate and therefore improve propelling efficiency (Wakayoshi et al., 1995). An increase in stroke rate and decrease in stroke length towards the end of a race has been consistently reported (Chengalur and Brown, 1992; Craig and Pendergrast, 1979; Kennedy et al., 1990; Thompson et al., 2000). This is a clear indication of fatigue, similar to the decline (albeit not as great) observed in pedal revolutions on an ergometer following maximal sprint cycling (Nevill et al., 1996a).

In 50 m sprint swimming, Wilke, 1992 demonstrated that swimming speed declined over each 10 m segment, but that success was determined by a slower decline in speed. Swimmer’s stroke rate declined over 50 m by 13%, but was not different between

standards or gender. Male stroke length was 0.17 m longer than females which accounted for the performance differences, and may indicate that the ability to maintain a high SR at an individual's optimal stroke length is a limiting factor in determining sprint swimming performance (Wilke, 1992).

2.9.2 Sex differences in performance

World record performance times differed between male and female swimmers by an average of $10.83 \pm 1.24\%$ (range: 9.26% for the 100 m Butterfly to 12.4% for the 100 m front crawl). This did not include the 800 m front crawl for females and the 1500 m front crawl for males (comparative speed difference = 6.39%). Males also have markedly longer stroke length, although stroke rate values appear to be remarkably similar in most events (except 400 m and 800/1500 m front crawl). This supports the findings reported in the detailed kinematic analysis carried out by Thompson et al. (2000) in elite breaststroke swimmers.

2.10 Swimming training

2.10.1 Training volume

Training has varied significantly over the years, but an exponential increase in volume has been apparent between 1910 and 1990. Improvements in world record times and general standards has been attributed to the substantial increase in training volume, although these standards have continued to rise in the 1990's despite a noticeable reduction in training volume (Costill, 1999). A long term (25 weeks) training study showed that swimmers who increased their volume (by increasing frequency – twice per day as opposed to once) of training for part of the season did not improve their end of season performances more than the control group (Costill et al., 1991). The high volume

swimming group decreased muscle power and sprinting speed during the high volume training phase, although this decline recovered after a taper period before the major competition. Both groups improved swimming power and performance in the competition by similar amounts, suggesting that the additional volume of training had no additional benefit to competitive performance (Costill et al., 1991; Costill, 1999). It remains to be seen whether or not the same effects would be apparent over longer periods of time (e.g. a number of years), with higher level of swimmer than the collegiate swimmers used in this study, or with a differing content of training between the two conditions. It is likely that the intensity of training has a greater impact on training for performance (Mujika et al., 1995), and that the high volumes of training are beneficial in preparation for high intensity training (Troup et al., 1994) and for improving swimming economy (Van Handel et al., 1988).

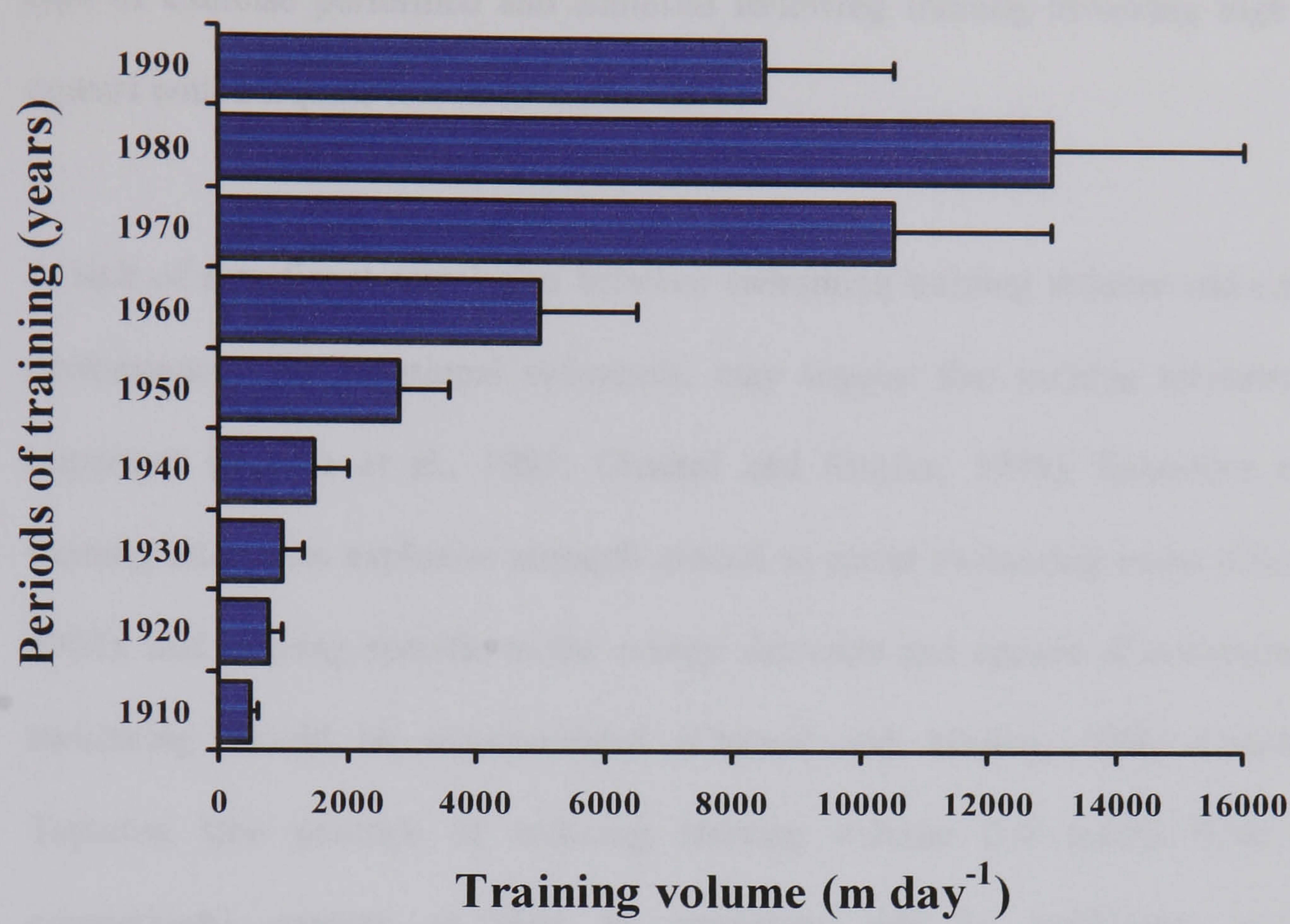


Figure 2.9 Training volume estimated from textbook and swimming journal reports between 1910 and 2000. Values are approximate means for the decade, with error bars indicating range of distances (adapted from Costill, 1999).

2.10.2 Training intensity

Research suggests that buffering capacity is a trainable mechanism. Sharp et al. (1986) improved buffering capacity by an average of 37% in a group of subjects who undertook substantial sprint training. This resulted in a 22% increase of work output in a test of high speed cycling to exhaustion. Mean muscle (vastus lateralis) lactate was 19% greater and muscle pH was higher after training, suggesting an increase in buffering capacity. Other physiological adaptations responsible for the increase in sprint performance after training are an increase in lactate production during sprinting (Saltin et al., 1976), and an increase in glycolytic enzyme activity (Gollnick et al., 1972; Saubert et al., 1973). It is likely that adaptations such as these will be specific to the

type of exercise performed and achieved following training involving high intensity repeats with adequate rest intervals.

A lack of significant correlation between swimming training volume and competitive performance in international swimmers, may suggest that training intensity is more important (Mujika et al., 1995; Chatard and Mujika, 1998). Excessive endurance training attenuates explosive strength crucial to sprint swimming races (Costill et al., 1988), and training specific to the energy demands and speeds of competitive sprint swimming should be recommended (Chatard and Mujika, 1998; Costill, 1999). Tapering (the practice of reducing training volume 2-4 weeks from a major competition) appears to play an important role in facilitating performance improvements (3-4% - Neuffer et al., 1987) through adequate recovery (Mujika, 1998), improving power output (Costill et al., 1991), the procedure of 'shaving down' (Sharp et al., 1989), enhanced neural function (Costill, 1998) and improved swimming economy at race speeds (Van Handel et al., 1988). Recently Trappe et al. (2001) observed a 14-17% gain in arm strength following a taper, accompanied by an increase in muscle fibre shortening velocity and power. Muscle fibre strength gains were specific to type II fibres demonstrating a 12% increase in size. It is suggested that improvements in performance following taper are part due to positive changes in the contractile elements of skeletal muscle fibres (Trappe et al., 2001).

A considerable amount of debate remains regarding training volume, intensity and frequency. On one hand general coaching practice dictates that (at least some) high volume training is critical for competitive success, perhaps through improved swimming economy, endurance performance or preparation for higher intensity training

(Troup et al., 1994). On the other, the duration of the majority of competitive events and the research by Costill and colleagues would suggest that lower volume higher intensity training should be undertaken. It is clear that no longitudinal data (2-5 years) with training content related to performance exists and that to control such a study would be nearly impossible.

Perhaps the most important training principle is that of individualism, adjusting each swimmers programme based on stage of development, demands of the targeted competitive event, training and competitive experience, physical capacity, physiological training response, sex and emotional maturity (Troup et al., 1994). A study that examined the responses of strength and endurance of swimmers in the same training programme found enormous differences in both and quite varied amongst the eight subjects (Strength gains: 12 – 42%; $\dot{V}O_2\text{max}$ gains: 7 – 23%; Costill, 1999).

2.11 Laboratory and pool tests to monitor swimming performance

2.11.1 Sprint swimming

Several methods to measure sprint swimming ability have been developed, although they are time consuming and generally provide little more information than we might obtain from a stopwatch during an all-out swim test or a set of maximal sprint intervals (Costill et al., 1992). Despite this claim, there are many studies using a wide range of equipment and protocols to evaluate sprint-swimming performance.

Sprint swimming research equipment includes an upper body arm-ergometer that has been used in conjunction with a Wingate 30 s test (Pizza et al., 1989; McGuire et al.,

1989; Rohrs et al., 1989; Rotstein et al., 1988; Williams and Hawley, 1989). In such tests, the measurement of peak power output (PPO), mean power output (MPO) and total work performed has been significantly correlated with swimming performance over distances of 25, 50, 100 and 200 yards/meters (Pizza et al., 1989; McGuire et al., 1989; Ribeiro et al., 1988; Rohrs et al., 1989; Rotstein et al., 1988; Williams and Hawley, 1989). Significant correlations (0.58-0.78) were also found by Rohrs et al. (1989) between tethered swimming and upper body Wingate test MPO and PPO and race performances. Studies suggest that sprint laboratory or tethered sprint swimming are valid (Rohrs et al., 1989; Rotstein et al., 1988; Williams and Hawley, 1989), can predict sprint swimming speed (correlations between 0.73 and 0.91 - Pizza et al., 1989; Rotstein et al., 1988; Williams and Hawley, 1989), and discriminates between competitive swimming standard (Rohrs et al., 1989).

Takahashi et al (1992a) developed a dry land swim bench test based on the protocol suggested by Medbø et al. (1988) and calculations originally devised by Hermansen and Medbø (1984). They compared the results obtained on the swim bench with a similar test performed in a swimming flume. Significant and relatively high ($r = 0.79 - 0.83$) correlations were obtained between oxygen deficit in the swimming flume test and selected dry-land power variables. The authors conclude that the 45 s maximal swim bench test can be used as an accurate predictor of a swimmer's "anaerobic profile".

The study carried out by Takahashi et al (1992b) involved identical techniques and protocols employed on the swim bench and in the swimming flume to estimate anaerobic capacity using the oxygen deficit method (see above). The Tests were carried out on age-group swimmers (range = 9.5-18 years), and found that younger swimmers

(< 13.5 average) perform (even) short duration exercise with a large energy contribution from aerobic sources. Improvement in MAOD reflected increases in muscle area during growth, and assisted in predicting swimming performance ability.

2.11.2 Endurance swimming

The majority of tests of endurance ability have adapted tests and techniques developed in the laboratory for other exercise modalities. These include monitoring the blood lactate responses to submaximal swimming (usually using the speed corresponding to 4 mmol l⁻¹ as a reference point; Mader et al., 1978; Pyne et al., 2001; Sharp et al., 1984), the lactate minimum, critical speed (MacLaren and Coulson, 1999; Toussaint et al., 1998; Wakayoshi et al., 1992), mathematical modelling (Holroyd and Swanwick, 1993), backward extrapolation of the post-exercise O₂ uptake curve (Costill et al., 1985c; Lavoie et al., 1983; Montpetit et al., 1981) $\dot{V}O_2$ max (Bonen et al., 1980; Magel and Faulkner, 1967; Troup et al., 1992a), swimming economy (Troup, 1984), swimming speed at a fixed or percentage of maximum heart rate (Sharp et al., 1984; Treffene et al., 1977) and the calculation of swimming energetics (di Prampero, 1986; Capelli et al., 1998; Toussaint and Hollander, 1994; Toussaint et al., 2000).

2.11.3 Testing equipment

Ideally, swimming testing should take place in the water and during 'free swimming'. This is often not possible due to the medium, the horizontal displacement (25 – 50 m), movement of the limbs, position of the head in water etc. A number of alternative solutions have been suggested, all with some degree of inaccuracy or impracticality. Test methodologies can be split into free swimming, flume swimming, tethered

swimming and swim bench, the order in which they appear to be most specific to actual swimming movements (Bollens et al., 1988; Clarys, 1986).

2.11.3.1 Free swimming

This usually requires one of two approaches. Firstly, that a technician (Costill et al., 1992; Lavoie, 1982) or overhead gantry (see German Sport University, Cologne) follows the swimmer throughout the length of the swim, or that measurements are taken immediately post-exercise. The main problem with the first approach relates to how true the swimming action is with attachments to parts of the body including the breathing apparatus. In addition, the turning action in front crawl swimming is not possible. The second approach allows for a true action, but relies on the accuracy of being able to predict the physiological responses of swimmers from metabolic measurements post-exercise. Despite this, many studies report satisfactory post-exercise values for heart rate (Treffene et al., 1977), lactate (Mader et al., 1978; Maglischo et al., 1984; Sharp et al., 1984) and oxygen consumption (Costill et al., 1992; Lavoie et al., 1983).

2.11.3.2 Flume swimming

In an attempt to simulate free swimming as closely as possible, while retaining control over the subject in a static position, Astrand and Englesson (1972) introduced the swimming flume or water treadmill, a device that allows swimming against circulating water at a controlled speed. Swimmers have to swim faster to stay on the spot, and the experimenter can control water speed and physiological testing easily.

The high cost and accessibility of swimming flumes preclude their use by all but those specialist institutes that have invested in such equipment. Despite this regular research

projects using flume testing are reported from Hamburg, Germany; Rome, Italy; Colorado Springs, USA; Tokyo, Japan and Otago, New Zealand. One criticism of this technique, which may alter fluid mechanics, is that the swimmer remains static against moving water as opposed to moving through static water. Whilst this may not be a significant issue at slower speeds, swimmers attempting to sprint at maximal speeds are limited to $\sim 2 \text{ m s}^{-1}$ (Mark Foster – World Record holder, personal correspondence) somewhat slower than that recorded in competition ($\sim 2.4\text{--}2.1 \text{ m s}^{-1}$ throughout a 50 m race, Haljand 2001, Mason 2000).

Much of the early swimming research carried out in flumes involved the description of cardio-respiratory and metabolic responses to swimming (Holmer 1974a; 1974b). Troup and co-workers presented several papers at the 1990 Biomechanics and Medicine in Swimming Congress in Liverpool using swimming flume testing. They considered the physiological responses of swimmers to training intensity and rest (Barzdukas et al., 1992a; Troup et al., 1992a), training duration and volume (D'Acquisto et al., 1992a; 1992b), energy contribution to performance (Troup et al., 1992c) and training (Troup et al., 1992c) and dry land (Takahashi et al., 1992a) and swimming (Takahashi et al., 1992b) estimates of 'anaerobic power'.

2.11.3.3 Tethered swimming

The development of tethered swimming devices in the 1960's (Lavoie and Montpetit, 1986), allowed swimming specific testing at a fraction of the cost of building a flume.

Values for tethered swimming $\dot{V}O_{2\text{max}}$ of 4.14 and 4.20 l min^{-1} (Magel and Faulkner, 1967), and 4.10 l min^{-1} (Dixon and Faulkner, 1971) compare very favourably with

recent measurements in swimming flumes ($4.25 \pm 0.53 \text{ l min}^{-1}$; Takahashi et al., 1992b). When the same swimmers were measured during flume and tethered swimming, no differences in $\dot{V}O_{2\text{max}}$ were found (3.55 vs. 3.53 l min^{-1} respectively; Bonen et al., 1980).

Taylor et al. (2001) found tethered swimming reliability in mean force output (MFO) measurements, but not in peak force output (PFO) in age group swimmers, with ratio limits of agreement of 12-15%. A tethered swimming device was introduced by Costill et al. (1986) and found to be a more specific measurement of swimming power as it incorporated the elements of mechanics and application of force, important in swimming success. Perhaps EMG provides the strongest support for the use of tethered swimming as a specific testing device. Clarys and co-workers have compared many swimming movements on dry land and in the water (Clarys, 1985; Clarys et al., 1988) and found a close pattern of muscular contraction between tethered and free swimming (Bollens et al., 1988). The EMG data were assessed using quality ratios and found similar amplitude and timing of muscle activity with a majority of either analogue or identical patterns between the two types of exercise. Tethered swimming was described as similar to free swimming, although the arm muscle activity was similar whilst leg muscle activity was not (Bollens et al., 1988). Perhaps the restriction in body movement and lack of familiarisation with tethered swimming may be attributed to some of the observed differences.

Some criticisms have been levelled at the use of tethered swimming based on the fact that there is no forward movement involved and consequently that the mechanics of the stroke technique may alter (Di Prampero, 1974; Nelson and Goldfuss, 1971). The

position of the tether rope may also alter body position tending to lower the legs and increasing the body inclination (Maglischo et al., 1984). In addition, the lack of any head turning movement for breathing may alter mechanics. Karpovich (1933) found that turning the head to breathe during swimming increased drag by about 0.5 lb and 1.5 lb at speeds of 3 and 5 feet per second, although Payton et al. (1999) observed no difference in underwater pulling patterns with or without breathing. Nelson and Goldfuss (1971) suggested the arm pull during freestyle swimming is capable of applying greater force to the water when the breathing action is not performed. Alteration in stroke mechanics during the breathing action may become exaggerated under fatigue conditions, although the resistance of the mouthpiece and valve used for expired air collection may counteract the advantage gained in not breathing. Despite the potential shortcomings of tethered swimming, it appears to be a relatively convenient, inexpensive and specific testing mechanism for the assessment of power in competitive swimmers (Costill et al., 1986).

2.11.3.4 Swim bench

Dry-land testing with the use of a swim bench has been used as a mode of exercise which simulates the swimming movement. The swim bench is attractive in terms of low cost and practicality, but validity with respect to free swimming is questionable. Peak oxygen uptake values during swim bench testing for example, have been found to be only ~75% of that found on land by the same swimmers (Swaine and Reilly 1983). Possibly the most conclusive flaw in the specificity of swim bench testing was observed by Olbrecht and Clarys (1984) and Clarys (1985) during electromyographic (EMG) measurement (records the sequence, pattern and intensity of the muscle's electrical activity). The study found that electrical activity in 25 muscles differed significantly

between free swimming and dry-land swimming-simulation devices (including the isokinetic swim bench; Clarys, 1985, Fig 2). The EMG activity differed in time phase, intensity and force (size and frequency of peaks), and demonstrated different movement patterns.

Despite this, recent attempts have been made to correct some of the problems associated with the use of swim benches as a piece of swimming-specific test equipment by incorporating a leg (as well as arm) resistant pulley to the bench (Swaine, 1997). This method of testing has recently been used to assess cardiopulmonary and metabolic responses (Swaine, 1997; Konstantaki and Swaine, 1999), differences between leg and arm power (Swaine, 2000), compared to semi-tethered swimming (Swaine and Doyle, 1998), discriminate between swimming standard (Konstantaki and Swaine, 1999) in competitive swimmers with some success. The authors accept that although the movements are not similar to that of free swimming, the model may provide data that assesses capabilities of swimmers discreet from technical ability such as swimming-specific muscle power output.

2.12 Creatine supplementation

2.12.1 Introduction

Creatine (Cr) is an organic compound found naturally in food, which plays an important role in muscle metabolism (see section 2.3). It was first discovered in 1832 by Chevrall who observed that wild foxes had higher concentrations than those kept in captivity, suggesting a link with exercise (Balsom et al., 1994). It can be synthesised endogenously in the liver, kidneys and pancreas from glycine, methionine and arginine, although most humans will obtain the majority of their daily requirement from a normal

diet containing meat and fish. Daily creatine turnover has been estimated at ~1.6% of the total Cr pool (Balsom et al., 1995), or ~2g day⁻¹ for a 70 kg male (Walker, 1979).

2.12.2 Creatine supplementation and physiological responses

Short-term increases in body mass following 5-6 day Cr supplementation regimens have been reported in some studies (Balsom et al., 1993a; Balsom et al., 1995; Greenhaff et al., 1994; Kreider et al., 1998; Redondo et al., 1996; Vandenberghe et al., 1996), but not others (Earnest et al., 1996; Grindstaff et al., 1997; Redondo et al., 1996; Terrillon et al., 1997). It is believed that Cr enters muscle cells by water influx and therefore an increase in muscle Cr uptake will necessitate water uptake into the muscle (Volek et al., 1997). It is thought that the additional Cr absorbed into muscles and the associated water holding it in solution are responsible for increases in body and lean body mass (Balsom et al., 1994). Longer term increases in lean body mass have also been reported following resistance training (Kreider et al., 1996; Stout et al., 1997; Vandenberghe et al., 1997) which may be related to an enhanced protein synthesis as a result of both training and supplementing with Cr (Bessman and Sabavi, 1988).

Recent studies have shown that oral creatine supplementation can increase the total concentration of creatine and PCr in skeletal muscle and the rate of PCr resynthesis (Greenhaff et al., 1993a; 1994a; 1994b; Harris et al., 1992); increase muscle peak and mean power output during cycling (Birch et al., 1994); increase total work and reduce fatigue during repeated maximal exercise (Balsom et al., 1993; Greenhaff et al., 1993b, 1994a; Bogdanis et al., 1996b); improve running performance times (Harris et al., 1993); and improve recovery in repeated bouts of high intensity exercise (Balsom et al., 1993a; Greenhaff et al., 1993a, 1993b, 1994a; Bogdanis et al., 1996b). These results

suggest that oral creatine supplementation can improve performance in high intensity short term exercise by increasing resting concentrations of creatine and PCr and improving the rate of PCr and ATP resynthesis in skeletal muscle. Table 2.9 details some of the studies that support physiological changes and may lead to the ergogenic benefits of Cr supplementation.

Table 2.9 Mechanisms for creatine enhancing performance from muscle biopsy studies.

Author	Subjects	Dose		Finding
<i>Increased intramuscular Cr and PCr</i>				
Balsom et al. (1995)	7 M	6d x 20g	5 x 6 s sprints	Resting TCr increase: 129 – 152 mM (18%)
Casey et al. (1996)	9 M	5d x 20g	2 x 30 s IC	Resting TCr increase: 23 mM PCr increase in both type I & II fibres
Febbraio et al. (1995)	6 M	5d x 20g	5 x 60 s 120%	Increase in TCr and PCr
Green et al. (1996)	21 M	5d x 20g	OCS	Resting TCr increase: 122 – 143 mM (18%)
Greenhaff et al. (1993a)	10 M 2 F	5d x 20g	+CHO	Resting TCr increase: 124 – 158 mM (27%)
Greenhaff et al. (1994)	8 M	5d x 20g	-	Resting TCr increase: 15%
Harris, et al. (1992)	12 M 5 F	4,7,10,21d x 20-30g	-	Resting PCr increase: 55 – 67 mM (20%)
Hultman et al. (1996)	31 M	6d x 20g +22d x 2g 28d x 3g	-	Resting TCr increase: 127 – 149 mM (20%) Resting PCr increase: 67 – 91 mM (36%) Resting TCr increase: 20% Maintenance of +20% level
Myburgh et al. (1996)	13 T C	7d x 20g	1 hour Cycle	Resting TCr increase: 121 – 147 mM (21%) TCr increase correlated with % type II fibres
Ruden et al. (1996)	4 M 5 F	4d x 20g	30 s	Resting TCr increase: 21 mM
Volek et al. (1999)	19 M	7d x 25g 77d x 5g	1 RM BP&S	Resting TCr increase: 123 – 150 mM (22%) Drop to 136 mM after 12 weeks; >placebo
<i>Greater phosphocreatine resynthesis or metabolic efficiency</i>				
Balsom et al. (1993a)	16 M	6d x 25g	10 x 6 s	Fatigue attenuated by ~8%
Balsom et al. (1995)	7 M	6d x 20g	5 x 6 s sprints	PCr increase post-sprints: 40 – 70 mM (52%) 5% increase in final sprint
Birch et al. (1994)	14 M	5d x 20g	3 x 30 s	10.5% increase in PPO & MPO for bouts 1 & 2
Casey et al. (1996)	9 M	5d x 20g	2 x 30 s IC	More work after OCS, ATP loss 31% lower
Greenhaff et al. (1993b)	10 M 2 F	5d x 20g	30 x 5 sets IC	Improvement in PCr resynthesis: 42% @ 120 s 6.8% improvement in repeated sprints

M: Males; F: Females; T: Trained; C: Cyclists; d: Day; g: grams; mM: mmol kg⁻¹ dm
OCS: Oral creatine supplementation; @: Post-exercise; + CHO: Creatine supplemented with carbohydrate
IC: Isokinetic cycling; BP: Bench press; S: Squat; %: $\dot{V}O_2$ max; PPO, MPO: Peak and mean power output

2.12.3 Creatine supplementation and performance

In a recent study, Cooke et al. (1995) reported no significant differences in performance during the first or second sprint of 2 cycle ergometer sprints separated by 20 min rest after oral creatine supplementation (20 g creatine day⁻¹ for 5 days). Thus, the efficacy of creatine supplementation for performance enhancement during a single sprint may be

questioned. Conflicting results have been reported for nearly all forms of exercise following Cr supplementation. A maximal single contraction or peak power during sprint cycling or maximal isokinetic contractions has been shown to improve (Birch et al., 1994; Greenhaff et al., 1993b; Stout et al., 1997; Vandenberghe et al., 1996; Volek et al., 1997) or remain unchanged (Balsom et al., 1993; Greenhaff et al., 1994, Hamilton-Ward et al., 1997) following creatine supplementation. Many of the improvements following ingestion have been seen in repetitive sprints thus emphasising the potential role of creatine supplementation in enhancing recovery following sprinting (Balsom et al., 1995; Birch et al., 1994; Casey et al., 1996; Dawson et al., 1995; Earnest et al., 1995; Greenhaff et al., 1993b; Prevost et al., 1997; Stout et al., 1997).

Fewer studies have examined the effects of oral creatine supplementation on sports performance (in comparison with performance during laboratory tests such as isokinetic cycling), although many studies examining repeated running (Aaserud et al., 1998; Lefavi et al., 1998), maximal cycling (Jacobs et al., 1997; Prevost et al., 1997), maximal kayaking (McNaughton et al., 1998) and maximal rowing (Rossiter et al., 1996) have reported improvements in performance.

2.12.4 Creatine supplementation and sprint swimming

Oral creatine supplementation studies using elite competitive swimmers show similar findings with no improvements in performance in 25 m, 50 m and 100 m race distances (Burke et al., 1996; Mujika et al., 1996). Since the benefit from creatine supplementation involves faster recovery and improved performance in the latter repetitions of a multiple sprint set (Balsom et al., 1993a; Bogdanis et al., 1996b), researchers have considered the effects of Cr supplementation on repeated sprint

swimming. Swimming performance in repeated sprints has been found to improve following creatine supplementation (Grindstaff et al., 1998; Leenders et al., 1999) although the metabolic responses to repeated sprint swimming in elite, senior swimmers have not yet been studied. The benefit of this ergogenic effect in swimming is likely to be in enhancing training where repeated bouts of high intensity swimming are regularly performed as opposed to competition where single sprint performance prevails. Differences in the number of repeated sprints, repeat duration, rest interval between sprints and the intensity that the sprints are performed are all likely to influence any possible mechanism attributed to performance improvements following Cr supplementation. Table 2.10 describes the various swimming experiments conducted following Cr supplementation.

Table 2.10 Results of creatine supplementation studies involving maximal swimming exercise.

Author	Subjects	Dose	Swim mode	Results
Burke et al. (1996)	32 E M&F	5d x 20g	100 m time	No differences in time
Grindstaff et al. (1997)	18 J C M&F	9d x 21g	3x100 m 3x50 m	No differences in cumulative time, faster repeats in reps 2 & 3 post Cr
Leenders et al. (1996)	6 E F	6d x 20g 8d x 10g	6x50: 3 min 12x100: 2.5 min	3.9% improvement in mean 50 m speed No differences in 100 m times
Mujika et al. (1996)	20 HT M	5d x 20g	25, 50, 100 m time	No differences in time
Thompson et al. (1996)	10 M 2 F	42d x 2g	100 m time	No differences in time
Theodorou et al. (1999)	8 E M&F	5d x 20g	12x50 m 8x100 m	Improvement in repeated sprint performance, but no additional benefit after training

M: Males; F: Females; E: Elite; HT: Highly trained; J: Junior; C: Competitive
Cr: Creatine supplementation; d: Day; g: grams; 6x50: Repeated sprints with rest interval

2.12.5 Creatine supplementation and training

An enhanced gain in high intensity intermittent exercise capacity in isokinetic arm flexions after a 10 week period of strength training with creatine supplementation has been shown in sedentary subjects (Vandenberghe et al., 1996; 1997). A recent study has found no additional differences in the performance of elite swimmers who have followed a creatine 'loading' period with either a low dosage (5 g day^{-1}) 'maintenance' period or no supplementation (Theodorou et al., 1999). This study did not investigate the physiological responses of creatine supplementation to training and was conducted over a relatively short, 8-week period. To date, there are no longer term (>10 weeks) training studies that have investigated the physiological effects of oral creatine supplementation on elite performers training in a sports-specific environment. It would appear that very little information exists about the long term effects of creatine on training, perhaps because of the time-onerous nature of such experiments, the difficulty in adequately controlling conditions or the difficulty in separating changes in performance due to Cr supplementation or training.

2.12.6 The individual response to Creatine supplementation

The different responses to performance following creatine supplementation can be attributed to exercise mode, intensity, duration and type of contraction (Williams et al., 2000), but also the resting concentration skeletal muscle TCr. Maximal total intracellular creatine concentration of $\sim 150\text{-}160 \text{ mmol kg dm}^{-1}$ (Greenhaff, 1995; Harris et al., 1992) appears to be the upper limit in humans and the ability to improve performance following creatine supplementation is strongly related to those with lower initial levels who increase skeletal muscle TCr significantly. These individuals have been termed 'responders' (as opposed to 'non-responders'; Greenhaff et al., 1994a).

CHAPTER III

GENERAL METHODS

3.1 Introduction

This chapter includes elements of methodology that were common to two or more of the experimental chapters. Two main testing themes included the use of a typical swimming sprint training set (8 x 50 yards) and the development and use of a tethered swimming mechanism. All studies gained approval from the Loughborough University Ethical Advisory Committee following submission of detailed proposal, an example of which is included in Appendix A. This chapter describes in general the subjects used, the test equipment, and instruments used, test rationale and procedures, blood and gas collection, storage and analysis and the performance data collected (Appendices B and C provide details of the blood metabolite and human growth hormone assays). A description of the calculations and statistical techniques used in the thesis are provided, in addition to a short section dealing with reliability of tethered swimming measurement conducted in chapter VII.

3.2 Subjects

Throughout the six experimental chapters, successive generations of Loughborough University team swimmers participated in the studies. Despite this, all subjects were termed senior (over 19 years old) and had represented their district or won medals at National senior or junior events within the previous 24 months. Subjects were generally

ranked in the top 20 in Great Britain for their particular event and had 8-12 years of regular training and competitive experience. Many of the participants were full international swimmers and included one World Championship gold and one silver medallist. This level of swimmer was described as elite for the purpose of this thesis and compares favourably with such descriptions in the literature.

All subjects were involved in a regular training and competitive programme (7-10 x 2 hours swimming sessions per week; 35,000 – 55,000 yards + 2-6 x 1 h land training per week) and had regular experience of both competition swimming, as well as the repeated sprint training sets such as the ones used in these studies. Subjects were informed about the purpose of the study, any known risks involved and gave their written consent to participate (see Appendix A).

3.3 Test preparation

Subjects were required to refrain from consuming alcohol for the day prior to all tests, and performed light exercise (4,000-5,000 m swimming training per day controlled by the experimenter) throughout the studies. During the three days prior to test number 1, subjects were required to record all food and drink intake in a food record diary and requested to follow the same (pre-test 1) diet and eating schedule before test 2 and any subsequent test. Subjects re-tested during any study performed their test at the same time of the day (± 1 h). All test sessions took place in the Sports Science Research Laboratories and Sports Hall swimming pool (25 yards) at Loughborough University. Average pool temperature throughout the six studies was 29°C ($\pm 1^{\circ}\text{C}$). Throughout this thesis, yards will be stated when used, although for comparison, literature

references often use metric values (e.g. 22.86 m, 45.72 m and 91.44 m for 25 yards, 50 yards and 100 yards respectively).

A standardised warm up (approximately 25 min) was performed in preparation for each test during all studies:

- 10 min easy Swim
- 5 min easy leg kick and arm pull
- 4 x 50 yards Front crawl, reduce swimming time from repetition 1 to 4
 - Rest Interval = 20 s
 - Last 50 = ~ maximum HR –30 beats min⁻¹
- 100 yards easy swim
- Pick up towel and rest for 5 min
- Resting Blood Sample
- Test Begins

3.4 Test protocols

A variety of different test protocols were used throughout the six chapters, involving single sprints, repeated sprints and an incremental endurance test in free swimming. In addition, single and repeated sprints, submaximal and maximal endurance tests were performed during tethered swimming. Two chapters included Cr supplementation protocols of either loading or loading and maintenance dosages. Each of these protocols will be described in the following sections and are summarised in table 3.1.

Table 3.1 Protocols used during each of the six experimental chapters

<i>Protocols</i>	<i>Mode</i>	Chapter					
		IV	V	VI	VII	VIII	IX
50 yards	FS	●				●	●
8 x 50 yards	FS	●				●	●
5 x 200 yards incremental	FS						●
4 x 50 yards	FS				●		
30 s	T		●	●	●		
4 x 30 s @ 95%	T				●		
55 s	T			●			
4 x 5 min submaximal	T		●	●			
4 x 3 min $\dot{V}O_2$ max	T		●	●			
Cr loading						● ¹	● ²
Cr maintenance							●

All protocols maximal unless stated; 95% of 30 s MPO
●: Protocol used or product measured; Cr: Creatine; ¹: 5 days x 9 g day⁻¹; ²: 5 days x 20 g day⁻¹
FS: Free swimming; T: Tethered swimming

3.4.1 Free swimming

3.4.1.1 Repeated sprints

In chapters IV, VII, VIII and IX, single or repeated sprints of 50 yards were used as the test protocol. A race distance of 50 yards was selected as the closest to a true 'sprint' in competitive swimming. Conveniently, the swimming duration (~22-23 s and 25-26 s for males and females, respectively) allows for some comparison with sprint studies carried out using other modes of exercise (e.g. 30 s cycle ergometer and treadmill sprinting).

The distance and rest period for the sprint interval training set used in chapters IV, VIII and IX, were devised after two pilot studies in which the swimmers completed a set of 5 x 100 yards on an interval of 4 min, and a set of 12 x 50 yards on 2 min. Subjects were asked to exert maximum effort on the first repetition of the set and also on subsequent

repetitions. Results from both tests suggested that capability of the swimmers warranted a shorter rest interval in order to induce a noticeable fatigue profile, and that 12 sprints or 5 x 100 yards were too daunting for the swimmers to give maximum effort on the first sprint. Therefore, 8 x 50 yards starting every 1 min 30 s was chosen and thus the first sprint of this set could be compared directly with single 50 yards sprint performance to establish that effort was maximal at the start of the set.

In order to assess reliability of a 50 yards maximal swimming sprint, 95% limits of agreement was applied to the single 50 yards and first repetition of the 8 x 50 yards set (Bland and Altman, 1986). Figures 3.1 and 3.2 show the limits of agreement between the two sets of data for male and female subjects respectively. Coefficients of variation were 2.6% and 3.1% for male and female tests respectively.

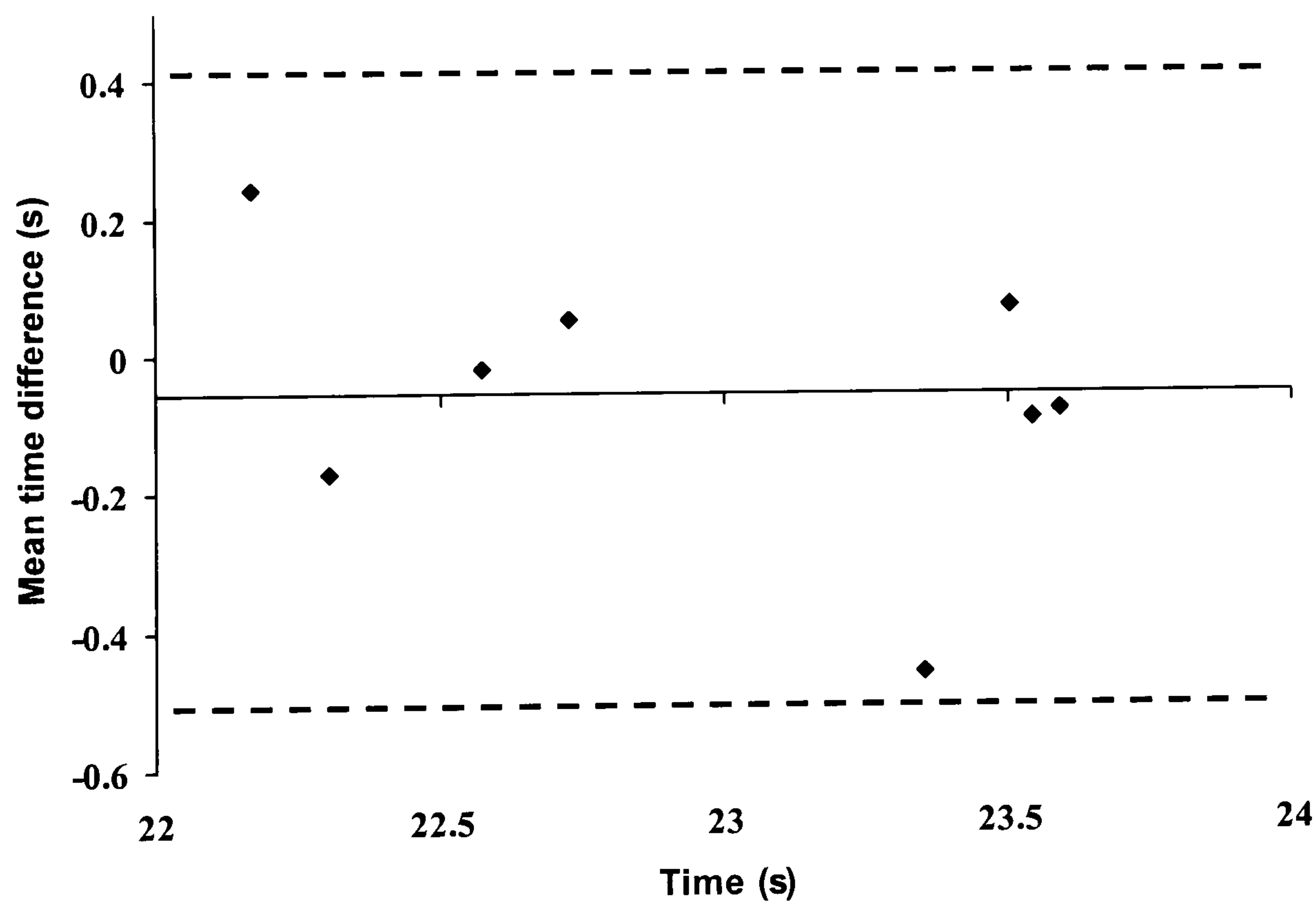


Figure 3.1 Limits of agreement for male subjects between 50 yards and the first repetition of 8 x 50 yards maximal sprint swims

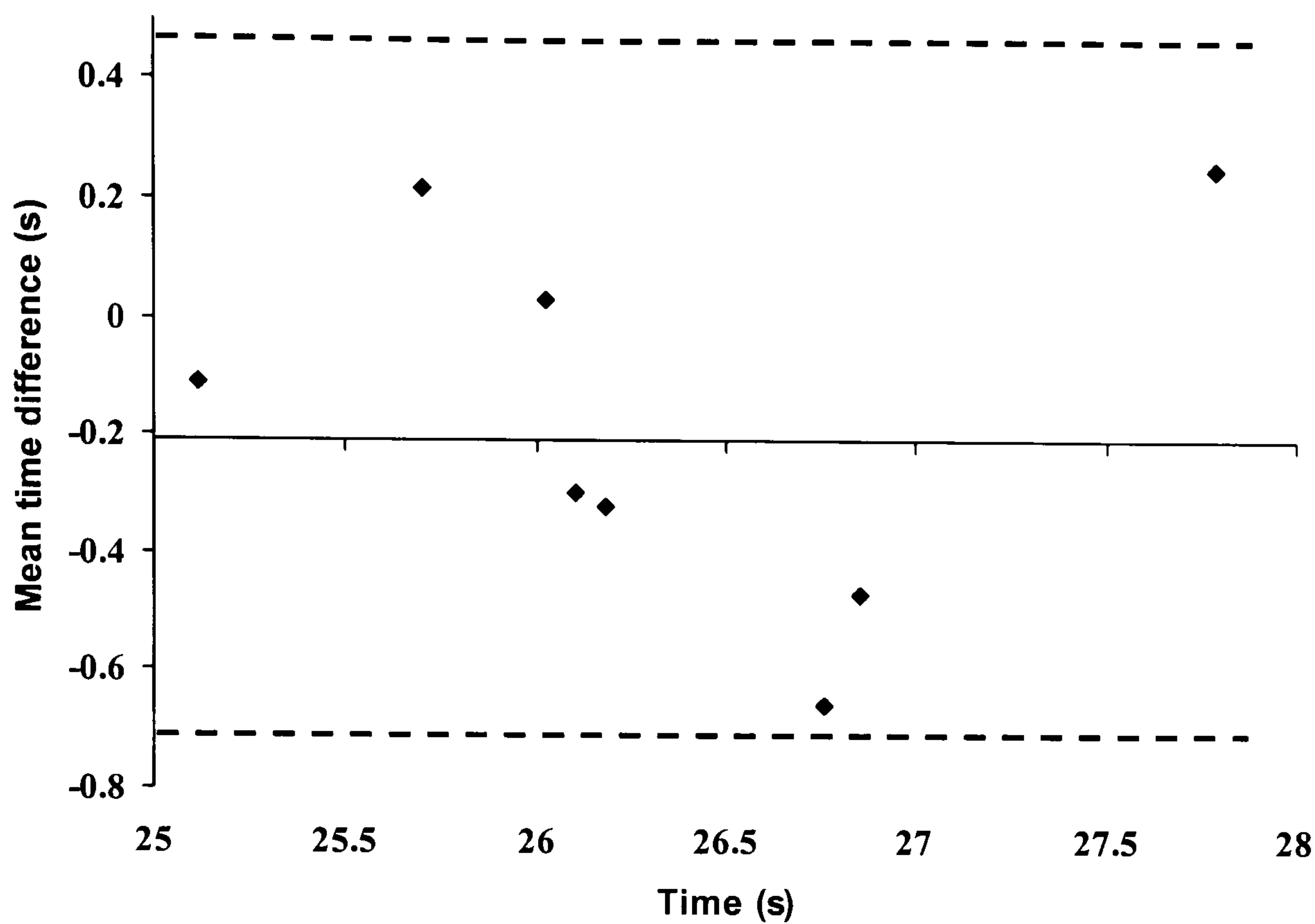


Figure 3.2 Limits of agreement for female subjects between 50 yards and the first repetition of 8 x 50 yards maximal sprint swims

The 8 x 50 yards set is similar to a 6 x 50 m test set used to predict race performance (Troup et al., 1994). The mean 50 m time multiplied by two correlates well with 100 m race time ($r = 0.98$; Troup et al., 1994), and has been used as a sprint training set in other studies (Leenders et al., 1998). A test set of 10 x 50 m with either ~30 s (Theodorou et al., 1999) or 1 min 30 s rest (Costill et al., 1992) have also been reported.

All repetitions took place with a dive from racing blocks and were timed from an official start. 'Anti-wave' lane ropes were used to divide the lanes as in competition to reduce unnatural water resistance. Subjects were asked to exert maximum effort on each repetition, whilst maintaining a constant stroking technique. Experienced timekeepers

using chronograph stopwatches recorded swimming times in duplicate. In chapters IV, VIII and IX, a reference time for one 50 yards sprint was obtained. The objective in the second, 8 x 50 yards test was to repeat that time as closely as possible on the first 50 yards swim, and then on successive repetitions.

In chapter VII, the main trials involved a combination of tethered and free swimming. The free swimming section involved 4 x 50 yards sprints preceded by 4 x 30 s tethered swimming repeats and a different recovery protocol on three separate occasions. The total duration of sprinting (i.e. tethered and free swimming) would have been similar to that performed in 8 x 50 yards sprints. The 4 x 50 yards sprints were recorded from a 'push' start from in the water due to the close proximity of the tether mechanism and to simulate training conditions.

3.4.1.2 Endurance test

In order to determine changes in endurance over the training period in Chapter IX, a typical speed-lactate swimming test (adapted from Harrison et al., 1992; Pyne et al., 2001) comprising 5 x 200 yards incremental repeats was used. The repeats used in the speed-lactate test were performed from a 'push' start from in the water and were incremental beginning at ~75% of personal best pace (self-paced) and increasing in even stages to maximum effort on the final 200 yards. Capillary blood samples were taken following each section, and with the recorded times a swimming speed-lactate relationship was established.

3.4.1.3 Stroke rate and stroke length

In chapter VII, stroke rate and length was recorded during 4 x 50 yards sprints in order to determine whether fatigue affected stroke mechanics. Each arm stroke was recorded manually and calculations made later on the following basis:

- $SR \text{ (strokes min}^{-1}\text{)} = (\text{strokes to complete the distance} / \text{time in s for the distance}) \times 60$
- $SL \text{ (yards stroke}^{-1}\text{)} = \text{distance to swim} / \text{strokes to complete the distance}$

3.4.2 Tethered swimming

3.4.2.1 General

Chapters V, VI and VII used a tethered swimming apparatus (Figure 3.3) to enable the collection of expired air for later analysis. A system of three pulleys allowed either incremental loading or a fixed restraint against the swimmer's forward motion. A basket with known loads allowed a swimmer to apply force to support the load whilst remaining in a stationary position in the pool. This known load (and therefore applied force) could be increased throughout an incremental test, and plotted against respiratory indices such as oxygen consumption.

A maximal sprint test was also possible by fixing the basket and the subject attempting to swim away from the mechanism. A force transducer interfaced to a computer measured the force applied by the swimmer at zero velocity to a non-elastic rope attached around the waist. The signal was displayed on the computer screen (BBC model B microcomputer) and stored on disk for further analysis. Before each test the

force transducer was calibrated using known loads. The force transducer has a manufacturers accuracy of $\pm 0.1\%$, and if the relationship between the known loads is less than $r^2 = 0.97$, the calibration is rejected and has to be repeated. It was also possible to measure force produced during semi-tethered swimming whilst the basket was supported in order to accurately determine the applied force and inter-and intra-cycle variations. Figure 3.4 shows the mechanism with fixings and computer interface.

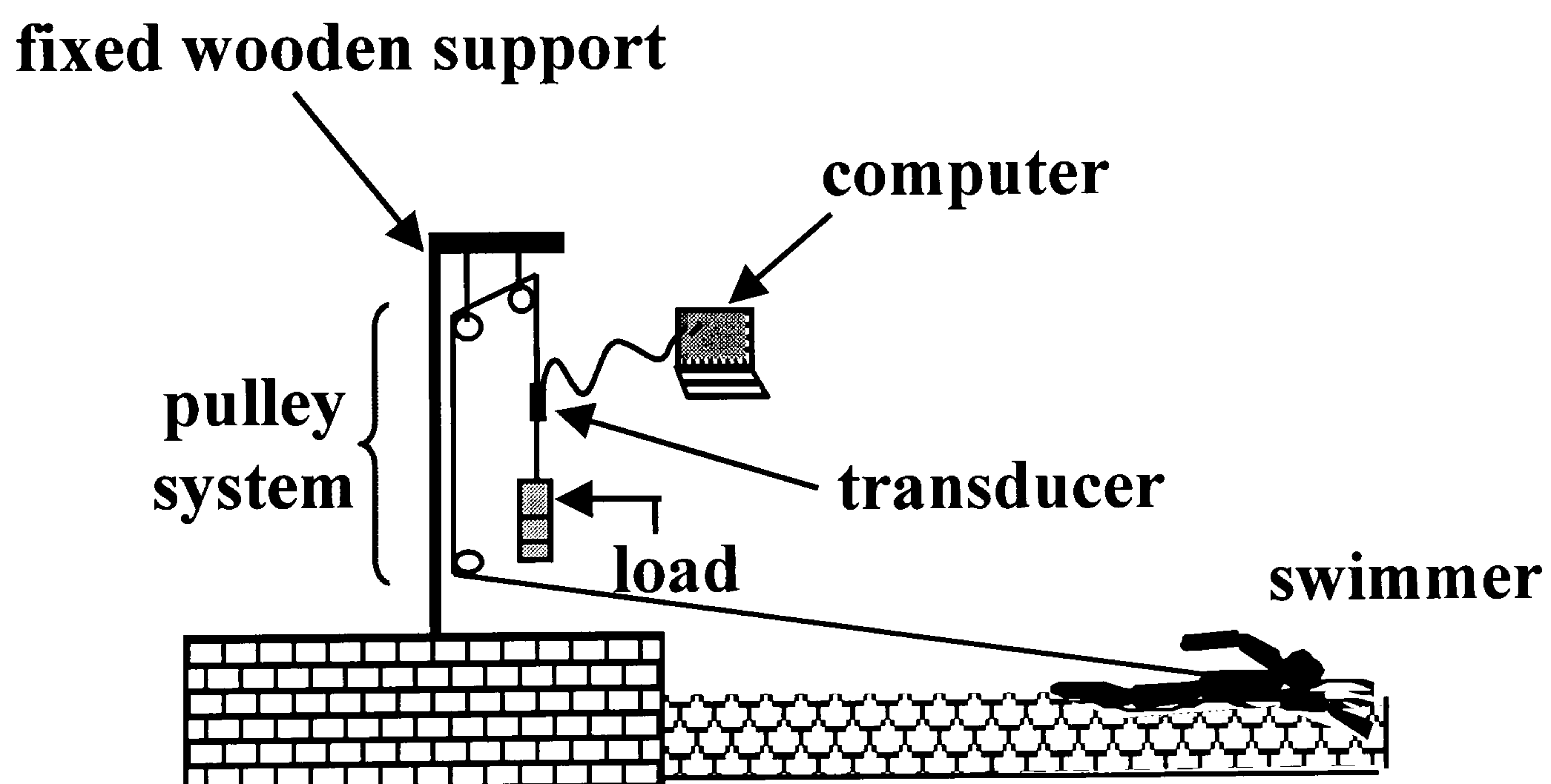


Figure 3.3 Diagrammatical representation of the semi-tethered swimming mechanism



Figure 3.4 The tethered swimming mechanism with force transducer and computer interface

3.4.2.2 Familiarisation

Swimmers all had experience of tethered swimming in training, but completed one comprehensive familiarisation session where they performed submaximal stages of the endurance tests (section 3.4.2.4) and short maximal sprints with a nose clip and the mouthpiece in position. This was conducted before any of the main study tests began.

3.4.2.3 Reliability of tethered swimming

During chapter VII, subjects performed three main trials comprising 4 x 30 s high intensity tethered swimming sprints (section 3.4.2.7), followed by differing recovery protocols and 4 x 50 yards maximal swimming. Trials were randomised, although the aim of the first set of 4 x 30 s tethered swimming was to standardise the work load. No differences in work load were observed between trials, and in order to determine the reliability of mean force output the 95% limits of agreement were applied to repetition one of trials two and three for each subject (Bland and Altman, 1986). The differences in force output and actual mean force output between the two tests is shown in figure 3.5, and the coefficient of variation for internal consistency was 6.5%. Analysis was carried out on the other 30 s repetitions between trials two and three, and all were within limits, similar to that shown in Figure 3.5. Coefficients of variation for the other repetitions ranged from 6.7% – 10.7%.

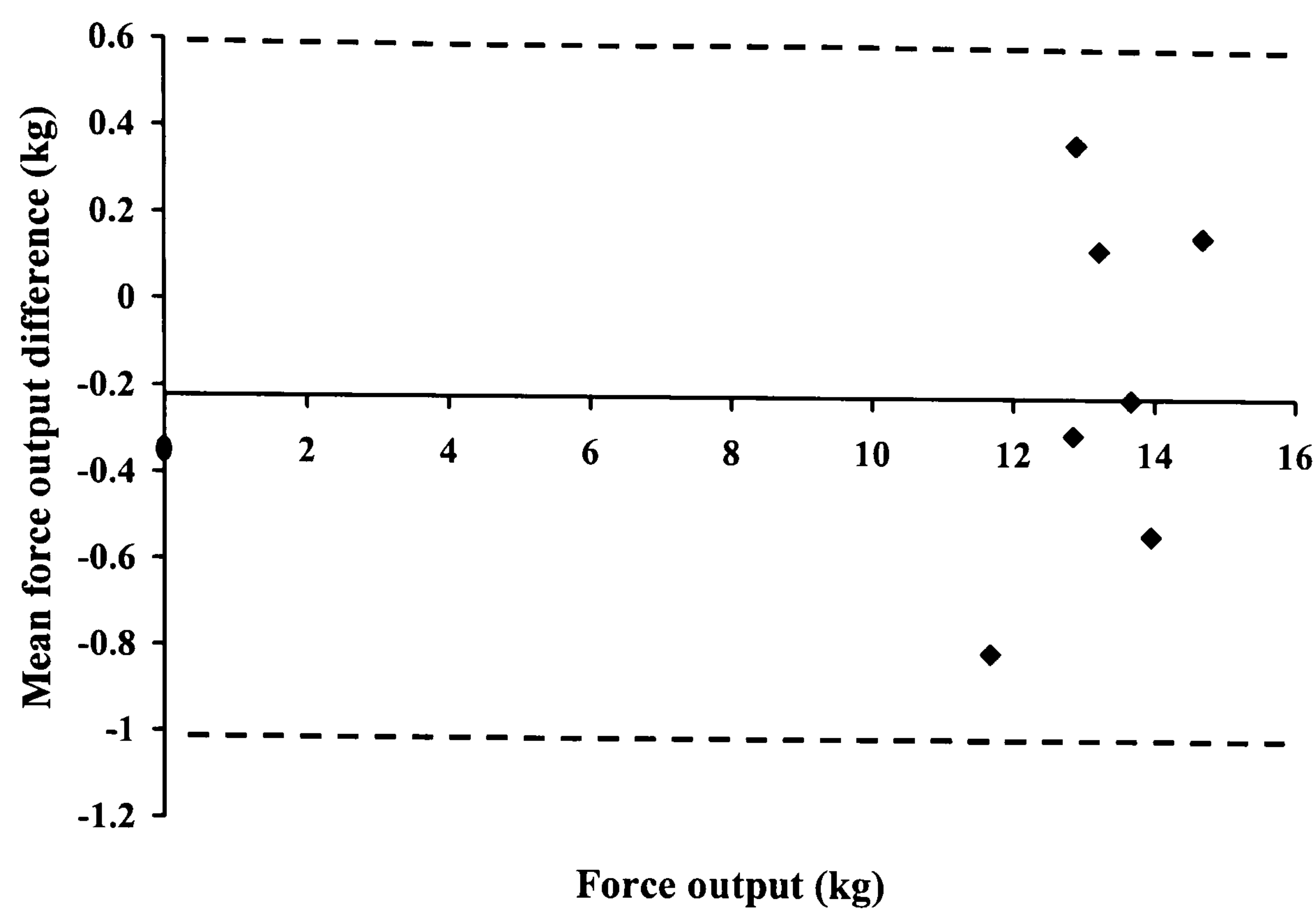


Figure 3.5 Mean force output differences during repetition 1 and limits of agreement between the second and third 4 x 30 s trial (n = 8; 13.95, -0.54: 2 values)

3.4.2.4 $\dot{V}O_2$ max test

Based on observations gained from the familiarisation sessions, subjects performed 3-5 stages of 3 min to determine $\dot{V}O_2$ max, similar to previous protocols reported in the literature (Bonen et al, 1980; Magel and Faulkner, 1967). The load was increased every stage by 1.0 or 0.5 kg whilst subjects maintained a stationary position adjacent to a mark on the pool bottom. Expired air was collected in the third minute of each 3 min stage, with a 15 s break between stages to increase the weight and allow subjects to check the correct positioning of mouthpiece and nose clip. When subjects approached their maximal capability, a clear sign by waving their hand was given, and a final one minute expired air sample was collected. A test-retest correlation of $r = 0.93$ for tethered

swimming $\dot{V}O_{2\max}$ was reported by Magel and Faulkner (1967) and close relationships have been reported between tethered and flume swimming $\dot{V}O_{2\max}$ ($r = 0.99$; Bonen et al., 1980), and tethered and free swimming $\dot{V}O_{2\max}$ ($r = 0.90$; Magel and Faulkner, 1967).

3.4.2.5 Submaximal endurance test

In order to establish a measure of swimming economy, a submaximal test was conducted, expired air collected and an oxygen consumption ($\dot{V}O_2$) vs. load relationship calculated. Subjects performed 4 x 5 min stages with 30 s rest between each. Load was increased by 1.0 or 0.5 kg and expired air collected in the final minute of each stage. Swimmers stopped for 10 s after 3 min in each 5 min stage to attach the nose clip and insert the mouthpiece, as in a pilot study, it was found that swimmers were not able to keep the mouthpiece in position for the 20 min test without considerable distress through jaw fatigue. Five minute stages were chosen to ensure that steady state swimming had been achieved. If steady state was not achieved and $\dot{V}O_2$ values were underestimated, economy would be overestimated and the error passed on to calculations using this measurement. Again a pilot study showed no significant difference in $\dot{V}O_2$ values obtained in the fourth or fifth minute of submaximal tethered swimming (values $\leq 0.2 \text{ l min}^{-1}$ higher).

3.4.2.6 Maximal 30 s sprint

A maximal 30 s sprint was used to obtain information such as peak and mean power output and fatigue index, previously used as estimates or indicators of anaerobic power, capacity and sprint capability; the time frame being similar to a 50 m sprint allowing comparison with sprint, race swimming. Swimmers were highly motivated for this test and received strong verbal encouragement. A starting position with arms extended was used and a sculling action and kicking established tension in the cord before a countdown and maximal effort was given on the command “Go!”. Expired air was collected with mouthpiece and nose clip in place throughout the maximal 30 s period.

3.4.2.7 Calculation of MAOD

The regression line between $\dot{V}O_2$ and force output obtained during the 4 stage submaximal test was extended to the force output obtained at $\dot{V}O_{2max}$ and then to that measured during the 30 s maximal sprint (as described by Medbø et al., 1988). The $\dot{V}O_2$ at the force output obtained during the 30 s sprint was described as the O_2 demand and the MAOD calculated by subtracting the $\dot{V}O_{2max}$ from O_2 demand. Estimates of aerobic and anaerobic energy contributions to exercise were made on the following basis:

- $\dot{V}O_2$ \equiv aerobic energy contribution
- MAOD \equiv anaerobic energy contribution

3.4.2.8 Repeated sprints

The first section (Set A) of the main trials in chapter VII consisted of 4 x 30 s bouts of semi-tethered swimming separated with 30 s of passive rest. Although the load was supported in the suspended cradle, the actual force on the rope was measured by the force transducer and was used for the analysis of the results. During all three conditions, the swimming intensity of Set A was kept at a similar level by setting the applied load the same across trials. The load applied on the basket during Set A was equal to 95% of the individual mean force attained during 30 s of the tethered swimming sprint.

3.4.2.9 Race simulation

In chapter VI, a race simulated swim of 55 s maximal tethered swimming was used to compare performance during different breathing frequencies. The mean personal best for 100 m Front crawl was 53.35 ± 1.18 s, and therefore 55 s was chosen as the nearest ‘round’ number to this time. The conditions and instructions for the test were identical to the 30 s sprint.

3.4.2.10 Stroke rate measurement

During tethered swimming, swimmers arm-strokes were recorded by depressing the space bar of a microcomputer every second stroke (once per stroke cycle). The time to complete each stroke cycle in addition to the total number of strokes was recorded and stored on a disk. The stroke rates produced in the first main trial during the 4 x 30 s tethered swimming bouts were retrieved as bleeps in the next two trials. The swimmer was asked to follow the same stroke rate according to the bleeps.

3.5 Equipment and measurements

During each chapter, different items of equipment were used in data collection, and physiological products obtained for further analysis. Table 3.2 details the products measured over the six experimental chapters.

Table 3.2 Metabolic, hormonal, urinary or cardio-respiratory products measured during each of the six experimental chapters

	Chapter					
	IV	V	VI	VII	VIII	IX
<i>Products measured</i>						
Capillary blood lactate	●	●	●	●	●	●
Venous blood lactate	●				●	●
Capillary pH		●		●		
Venous pH	●				●	●
Ammonia	●				●	●
Haemoglobin	●				●	●
Haematocrit	●				●	●
Growth hormone						●
Urinary Cr, Creatinine					●	●
$\dot{V}E$, $\dot{V}O_2$, $\dot{V}O_{2max}$		●	●			
MAOD		●	●			

$\dot{V}E$: Ventilation; $\dot{V}O_2$: Oxygen uptake; $\dot{V}CO_2$: Carbon dioxide production; MAOD: Maximal accumulated oxygen deficit;
●: Protocol used or product measured; Cr: Creatine

3.5.1 Heart rate, height and body mass

Height and body mass (and where possible in chapter IX, skinfold measurements – Durnin and Wormsley, 1974) were measured on each visit to the laboratory in swimming attire. Heart rate was recorded at rest and throughout all the exercise phases of the investigation by short-range telemetry (Polar Electro PE3000 Kempele, Finland).

A transmitter was strapped to the chest, level with the sternum, and secured under a lycra 'triathlon top' (Speedo Europe Ltd.) to ensure a constant contact between electrodes and skin. The receiver was vacuum-sealed in a water-proof plastic bag, and placed under the costume.

3.5.2 Expired air collection and analysis

In chapters V, VI and VII, expired air samples were collected in Douglas bags during the tethered swimming tests using an adapted two-way respiratory valve (Dal Monte et al., 1994; Toussaint et al., 1987). Figure 3.6 shows the mouthpiece that was attached to a heavy metal block to ensure that the air in the valve system didn't force the mechanism to the surface of the water.

All expired samples collected were analysed within 10 min for O₂ and CO₂ concentration using O₂ paramagnetic analyser (570A Servomex) and CO₂ infrared analyser (LIRA Gas monitor 3250). Analysers were previously calibrated with gases of a known mixture. Gas volumes were measured using a Harvard dry gas meter. Values obtained were corrected for STPD and the Haldane transformation used to correct for differences in inspired and expired air volumes.

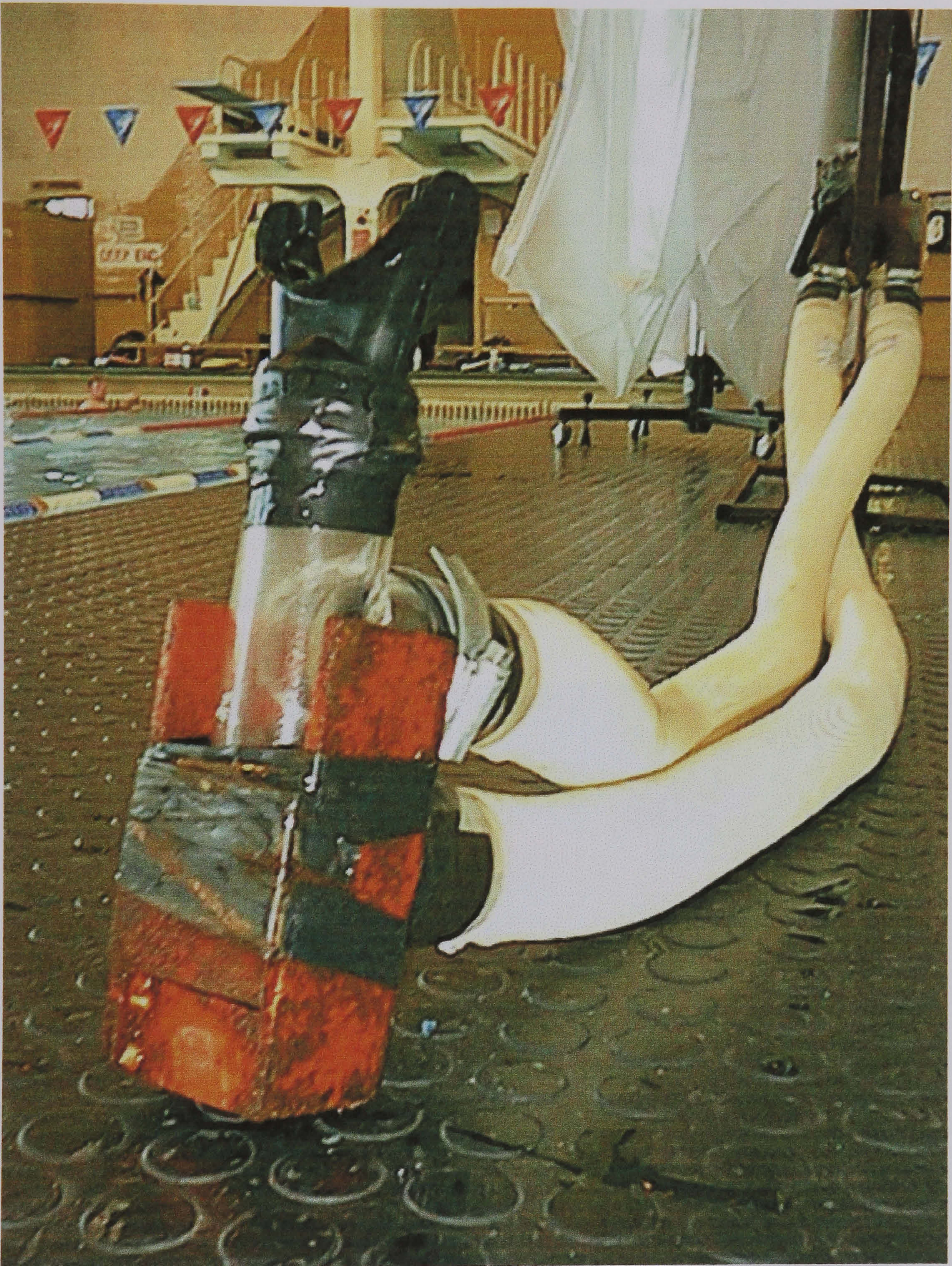


Figure 3.6 Respiratory valve and falconia tubing used during tethered swimming (adapted from Dal Monte, 1994; Toussaint et al., 1987).

In order to obtain expired air samples, a metal frame was constructed which allowed an arm to extend out over the edge of the pool. Attached to this arm was a pipe system that could be suspended above the swimmer and falconia tubing attached to the lower end of the pipes and the respiratory valve. Two pipes allowed both inspired and expired air transfer in conjunction with the respiratory valve system. The top end of the expired air pipe was attached to a Douglas bag to allow gas collection. Figures 3.7 and 3.8 illustrate the metal frame and respiratory system during tethered swimming. Previous reports suggested that body position and leg kick were lower than normal during tethered swimming (Di Prampero, 1974; Maglischo et al., 1984) and that the cord tended to pull the swimmers down. Pilot studies' using a floatation belt appeared to counteract this effect very well and was used throughout the tethered swimming tests.

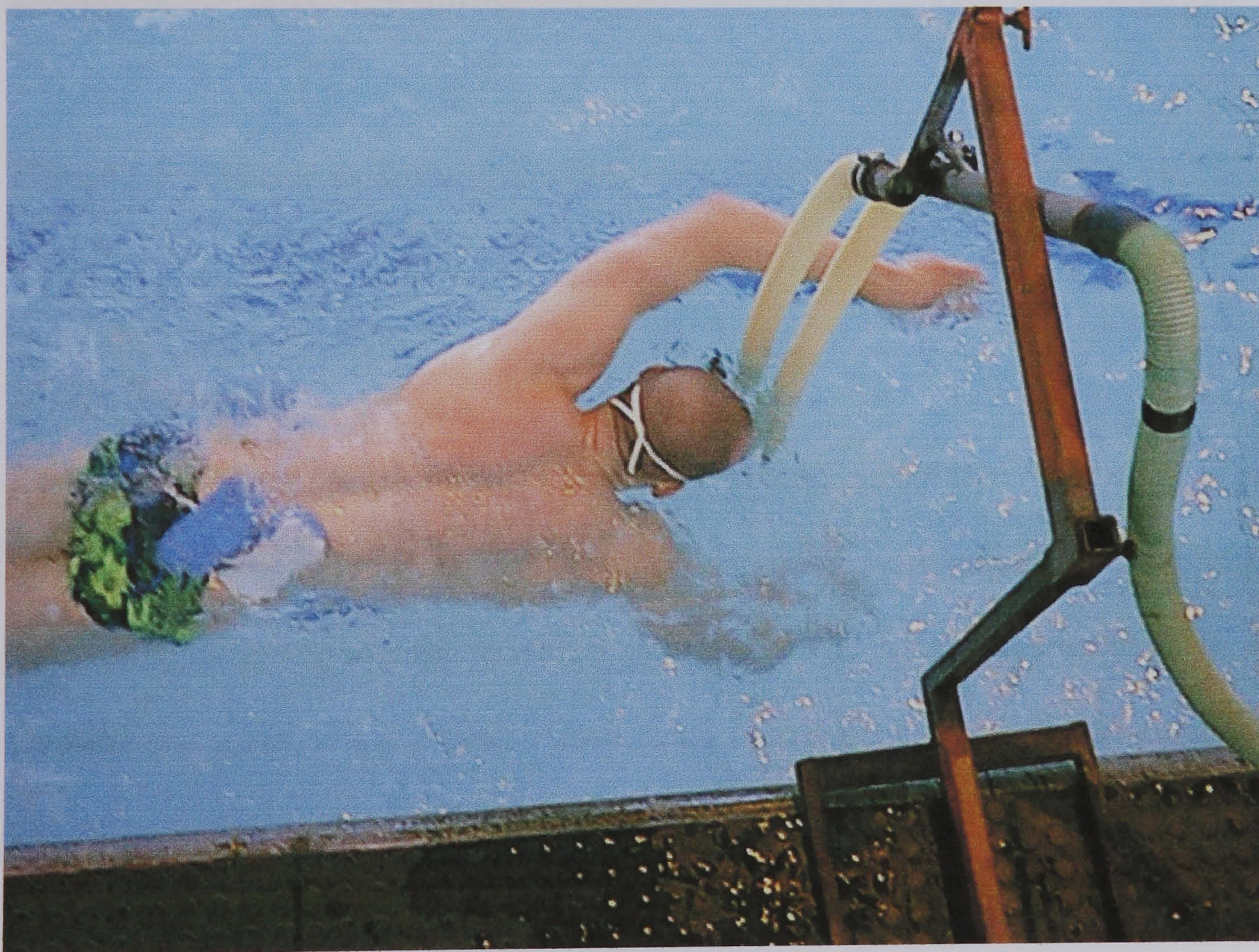


Figure 3.7 Aerial view of swimming during tethered swimming. The metal frame (right) allows for the breathing apparatus to be suspended in an unrestricted position above the swimmer, and the floatation belt can be seen around the swimmer's waist.

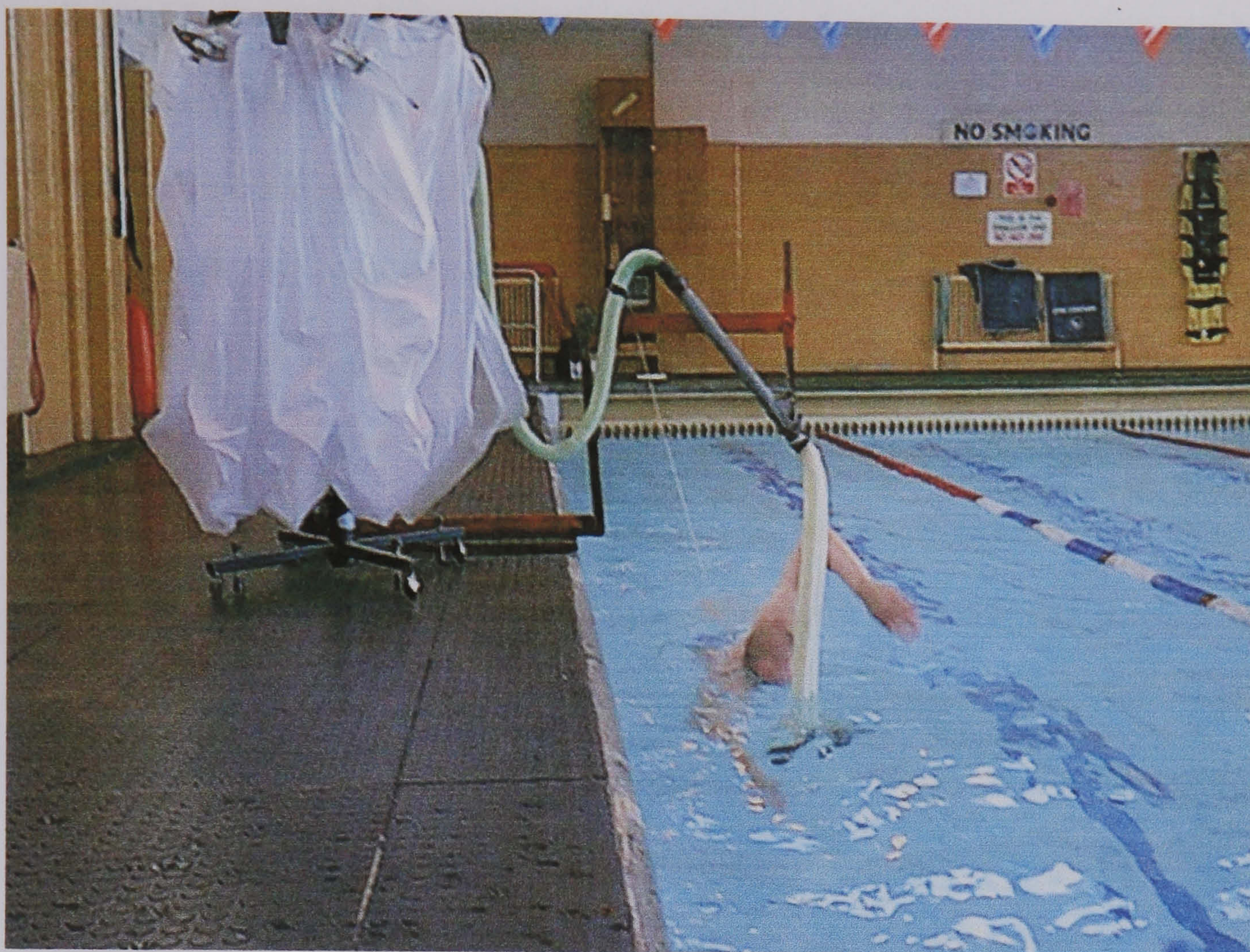


Figure 3.8 Tethered swimming allowing simultaneous expired air collection

3.5.2.1 Breathing

During the 55 s simulated swim, three different breathing patterns were used. In order to record actual breathing (as opposed to instructed), a pressure gauge was attached inside the expired air tubing and the sensitivity adjusted. Each expiration created an increase in pressure that set off a light clearly visible to the experimenter. Each breath was recorded according to the number of swimming stroke cycle it occurred, and an indication of the breathing rate throughout the 55 s sprint obtained.

3.5.3 Blood collection and analysis

Table 3.2 shows the different blood metabolite products measured during each chapter. Antecubital venous blood samples (~11 ml) were obtained at rest, before the standardized warm-up, and following exercise in the 8 x 50 yards tests in chapters IV, VIII and IX. All samples were taken with the subject supine, but the swimmers did have to climb out of the pool and walk approximately 15 m to the laboratory. Samples were dispensed into lithium heparin, calcium heparin and serum tubes (see Figure 3.9) and metabolites assayed at a later date (Appendix B). Blood pH was measured immediately using a pH blood gas monitor (either Radiometer PHM 73 and BMS 3 Mk2 or Radiometer ABL 5, both Copenhagen, Denmark). Intra- assay coefficient of variation for repeated analysis were <0.2% for blood pH. Haematocrit concentration was determined in triplicate using 30 µl microhaematocrit tubes which were centrifuged and read (Hawksley Micro-haematocrit instruments England). Haemoglobin was measured using the Cyanmethaemoglobin method (Boehringer Mannheim, GmbH test-combination Germany). Changes in plasma volume were estimated from the haemoglobin and haematocrit values using the method described by Dill and Costill (1974).

One ml of whole blood was dispensed into a lithium heparin tube, centrifuged at 12,000 revs min⁻¹ for 3 min, the plasma supernatant drawn off and stored at -70°C. Within 48 h, samples were thawed and assayed enzymatically for ammonia (Boehringer Mannheim, MPR 1 kit). Intra- and inter-assay coefficients of variation for repeated analysis of 1.7-4.1% and 2.4-4.5%, respectively. In chapter IX only, an aliquot of the venous blood sample (4-5 ml) was allowed to clot for 30 min, centrifuged, the serum drawn off and stored at -70°C for later analysis of hGH. The serum was analysed for hGH by routine

ELISA (Biosource CytoscreenTM, Nivelles, Belgium) that had a sensitivity of 0.11 mU.l⁻¹ and intra- (duplicate samples) and inter-assay (between three separate analyses) coefficients of variation for repeated analysis of 2.1-3.6% and 6.8-7.1%, respectively.

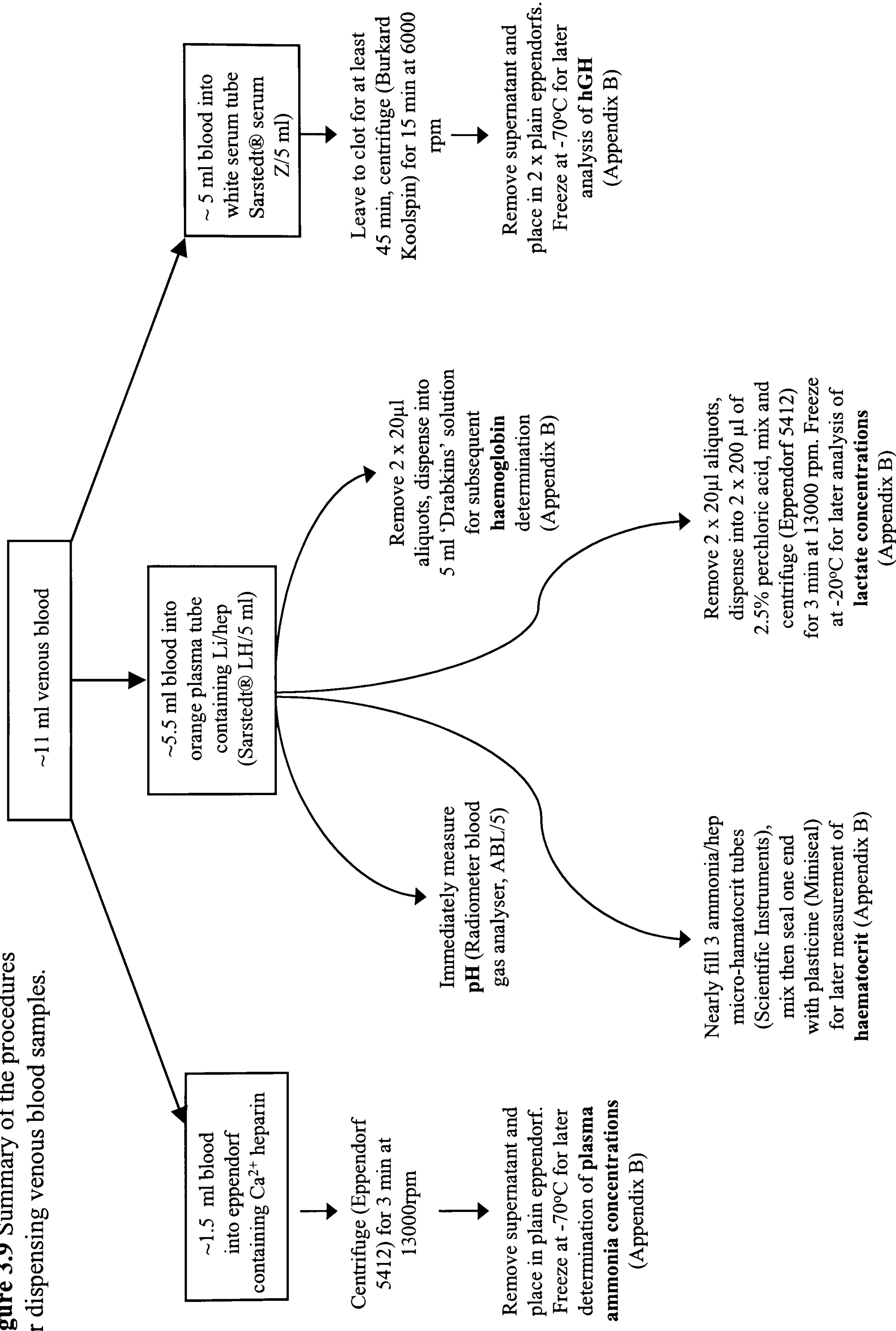
In all main tests throughout the thesis, duplicate 20 µl samples of blood were obtained pre- and post-exercise (see each chapter for time points) from a small thumb-prick in order to determine lactate concentration. Blood samples were dispensed into tubes containing 200 µl of 2.5% perchloric acid, mixed and centrifuged for 3 min. Tubes were then stored at -20°C and assayed enzymatically using the method described by Maughan (1982) at a later date. Intra- and inter-assay coefficients of variation for repeated analysis with blood lactate were 1.8-2.7% and 2.3-4.3%, respectively.

3.5.4 Urine Analysis

In order to determine the retention of creatine after supplementation in chapters VIII and IX (9 g or 20 g day⁻¹ for five days) a selection of subjects provided all urine samples over a 9-day period, the daily volumes recorded and aliquots taken from each and frozen for later analysis.

Urine was assayed enzymatically for creatine and creatinine using a commercial kit for creatinine determination (Boehringer Mannheim, MPR 2 Creatinine PAP). Creatine measurements were made by omitting the first creatininase step (Delanghe et al., 1988) and read spectrophotometrically against standards of known concentrations. Total creatine excretion was corrected for the mean increase in creatinine in accordance with Rossiter et al. (1996). Intra-assay coefficients of variation for repeated analysis on duplicate urine samples were 4.9% for creatine and 4.3% for creatinine.

Figure 3.9 Summary of the procedures for dispensing venous blood samples.



3.6 Analysis of results

A two-way analysis of variance (trials x time) was used to examine differences between means in chapters V, VI and VII. Significant differences between means were located using the Tukey post-hoc test. Relationships between variables in all studies were examined using the Pearson product moment correlation coefficient. Values are presented as means \pm standard deviation (S.D.) and significance set at the $P < 0.05$ level.

A three-way analysis of variance (ANOVA) or two-way ANOVA with repeated measures (SPSS) was used where appropriate to examine differences between the control and creatine group (main effect group), between all subjects pre- and post-supplementation (main effect trial) and to examine the response of all subjects over time (main effect time) in chapters VIII and IX. For significant F ratios, a paired *Students t*-test was used to determine the cause of the variance using the *Bonferroni* correction. Pearson product moment correlation was used to identify any relationship between variables. Differing responses between the groups as a result of supplementation were identified by group-trial and group-trial-time interactions.

CHAPTER IV

PHYSIOLOGICAL RESPONSES OF MALES AND FEMALES TO SINGLE AND REPEATED SPRINT SWIMMING

4.1 Introduction

The decline in rate of ATP resynthesis as a result of phosphocreatine (PCr) degradation is recognised as major cause of the reduction in muscle power during maximal sprint exercise (Bogdanis et al., 1995; Newsholme et al., 1992; Spriet, 1995). Evidence for these statements are based on muscle biopsy studies using cycling, running and arm-cranking (Costill et al, 1992; Saltin, 1990). Unfortunately it is not possible to describe the muscle metabolite changes during sprint swimming using the biopsy method due to practical difficulties. The information regarding the blood metabolite responses following sprint swimming is also limited and has generally focused on the measurement of peak blood lactate concentration following exercise (Bishop and Martino, 1993; Bonifazi et al., 1993; Madsen and Lohberg 1987; Sawka et al., 1979; Telford et al., 1986).

The majority of Olympic swimming events (>80%) last less than 2 min 30 s and therefore rely heavily on anaerobic energy supply (Houston, 1978; Troup and Trappe, 1994). Despite this, the majority of research that appears in the literature involves physiological responses to endurance swimming. Information regarding the physiological responses to sprint swimming has been less forthcoming, and the blood

pH and plasma ammonia responses following sprint swimming have not been described in any detail. One study reported plasma ammonia concentrations for senior and junior swimmers, although subject's training status was low and only metabolic (with no performance) data following sprint swimming was reported (Prado, 1999). Children (~11 years old) recorded similar blood lactate and plasma ammonia concentrations before and after training ($\sim 5 \text{ mmol l}^{-1}$ and $\sim 50 \text{ } \mu\text{mol l}^{-1}$, respectively), and whilst adults recorded similar lactate concentrations ($\sim 12 \text{ mmol l}^{-1}$), plasma ammonia concentrations following 100 m sprinting were reduced following 6 weeks of training (~ 149 vs. $\sim 126 \text{ } \mu\text{mol l}^{-1}$; Prado, 1999). There is a paucity of information regarding the metabolic responses following repeated sprint swimming typically used in training, nor is there any detailed information regarding the physiological and performance responses to sprint swimming.

A limited number of experiments have recruited female subjects, particularly in sprint swimming. General reviews detailing the physiological differences to exercise between males and females have generally focused on body size, body composition, muscular strength, and cardiorespiratory endurance (Drinkwater, 1984; Wells, 1986; NSCA Journal roundtable, 1985). Comparisons of changes in muscle metabolism following sprint running or cycling suggest either a greater metabolic response to sprinting in males (Jacobs et al., 1983; Karlsson and Jacobs, 1979; Komi and Karlsson 1978) or metabolic changes of similar magnitude (Cherry et al. 1998; Nevill et al., 1989) between male and female subjects. The purpose of this study therefore was to describe the physiological responses of elite male and female swimmers to maximal single (50 yards) and repeated (8 x 50 yards) sprint swimming.

4.2 Methods

Eight male and eight female elite swimmers from the Loughborough University swimming team performed two sprint tests; a single 50 yards sprint under race conditions, and a sprint training set of 8 x 50 yards, beginning every 1 min 30 s. Male and female swimmers were similar in competitive standard between groups, and each group contained 4 sprint and 4 mid-distance specialists of which 5 subject’s best stroke in each group was front crawl. Subject characteristics are shown in Table 4.1 and the study design, measurements and timing of the test protocol is shown in Figure 4.1. Details of the subject preparation, procedures, blood collection, handling and analysis can be found in chapter III. Venous and capillary blood samples were collected at rest, 1 and 5 min post-sprints, and capillary blood samples pre-sprint and 10 and 15 min post-sprint (Figure 4.2). A two-way analysis of variance (sex x time) was used to examine differences between means and located using the Tukey post-hoc test. Relationships between variables were examined using the Pearson product moment correlation coefficient. Values are presented as means \pm standard deviation (S.D.).

Table 4.1 Subject physical characteristics and personal best (P.B.) times (n = 8; data are the mean \pm S.D.).

Subjects	Age (years)	Height (cm)	Weight (kg)	50 m F/C P.B. (s)	50 Yards F/C P.B. (s)
Male					
Mean	22	183.0**	76.3**	24.56**	22.10**
S.D.	± 3	± 6.5	± 6.8	± 0.37	± 0.35
Female					
Mean	20	170.1	63.5	28.14	25.38
S.D.	± 2	± 4.5	± 4.4	± 0.57	± 0.40

P.B.: Personal best time; F/C: Front crawl
** P<0.01, male vs. female

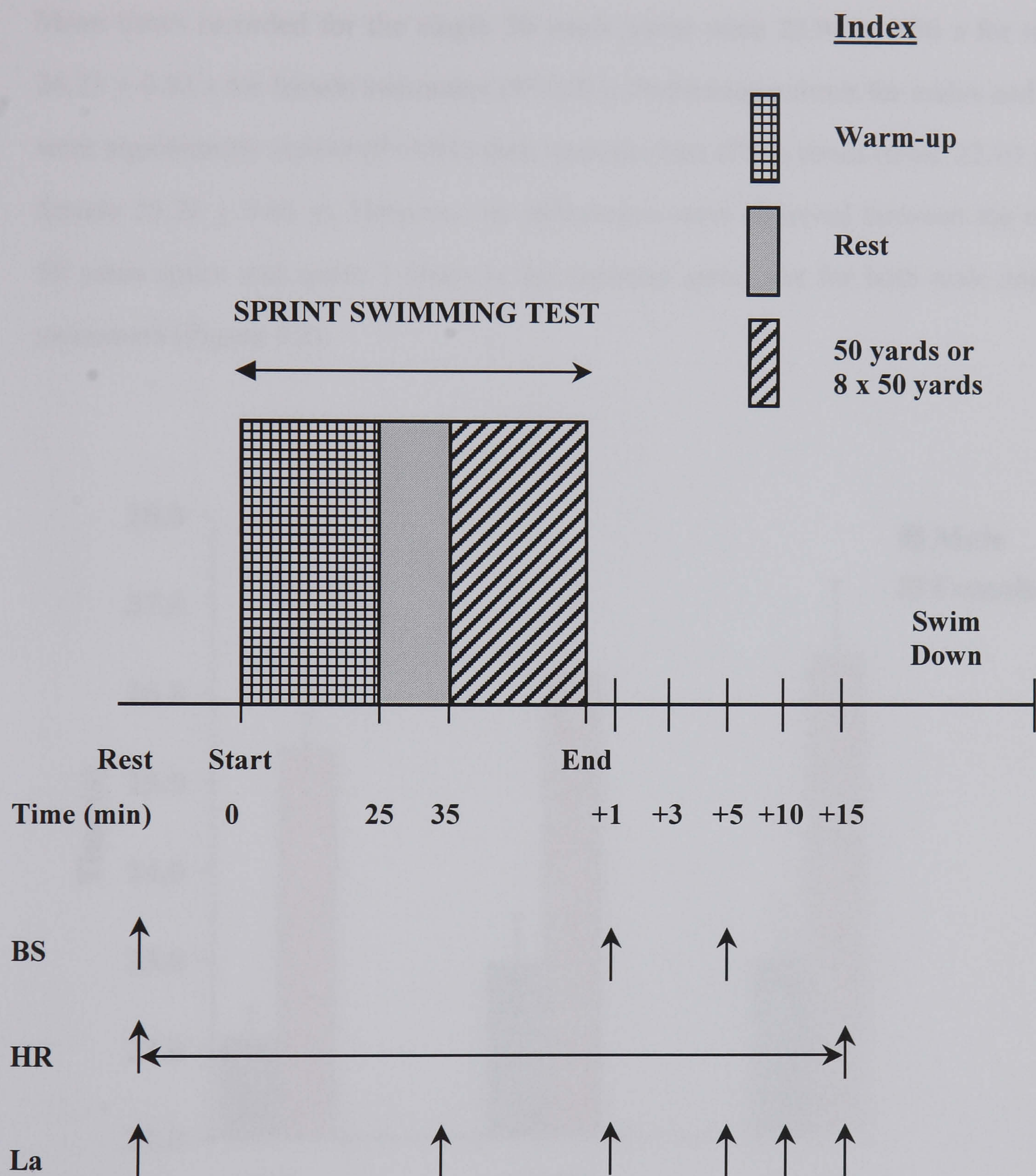


Figure 4.1 Schematic illustration of the 1 x 50 yards or 8 x 50 yards swimming protocol (BS = venous blood sample, HR = heart rate, La = fingertip capillary blood sample).

4.3 Results

4.3.1 Performance times

Mean times recorded for the single 50 yards sprint were 22.94 ± 0.56 s for male and 26.23 ± 0.82 s for female swimmers ($P<0.01$). Performance times for males and females were significantly slower ($P<0.01$) than personal best (P.B.) times (male 22.10 ± 0.35 s, female 25.38 ± 0.40 s). However, no differences were observed between the reference 50 yards sprint and sprint 1 times in the repeated sprint test for both male and female swimmers (Figure 4.2).

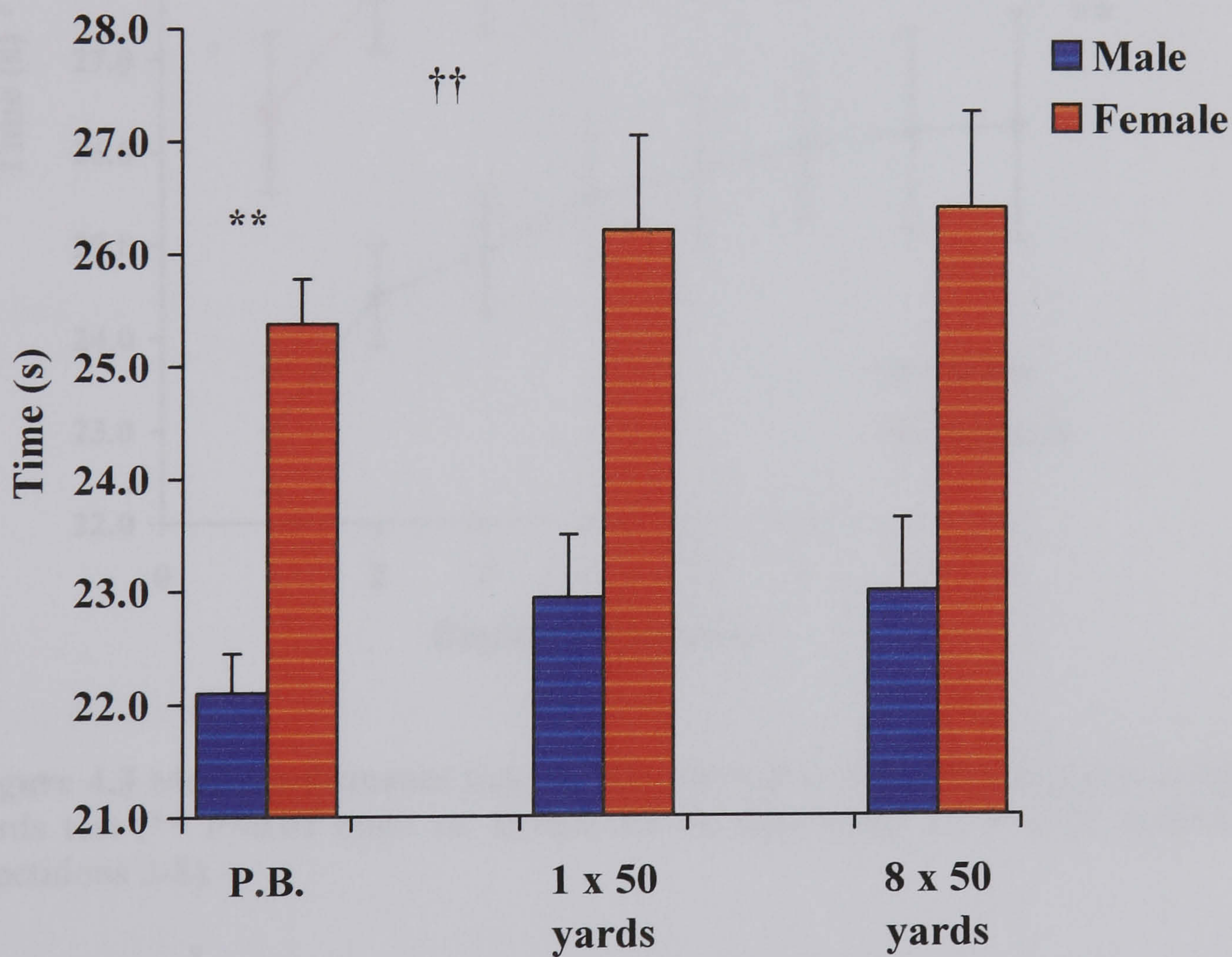


Figure 4.2 Personal best (P.B.) times and mean performance times \pm S.D. for male and female subjects in the 1 x 50 yards test and the first repetition of the 8 x 50 yards test (male and female: †† P.B. vs. 1 x 50 yards and first repetition of the 8 x 50 yards test $P<0.01$; 1 x 50 yards vs. 8 x 50 yards, N.S.; male vs. female: ** $P<0.01$).

During the repeated sprint test, mean times declined (repetition number 1 to 8) from 23.00 ± 0.64 s to 26.29 ± 1.25 s for males and 26.40 ± 0.84 s to 29.29 ± 1.26 s for females. Male performance times were faster than females for all repetitions ($P<0.01$) and both group's times were significantly faster for the first 50 yards sprint compared with repetitions 2 to 8 ($P<0.01$; Figure 4.3).

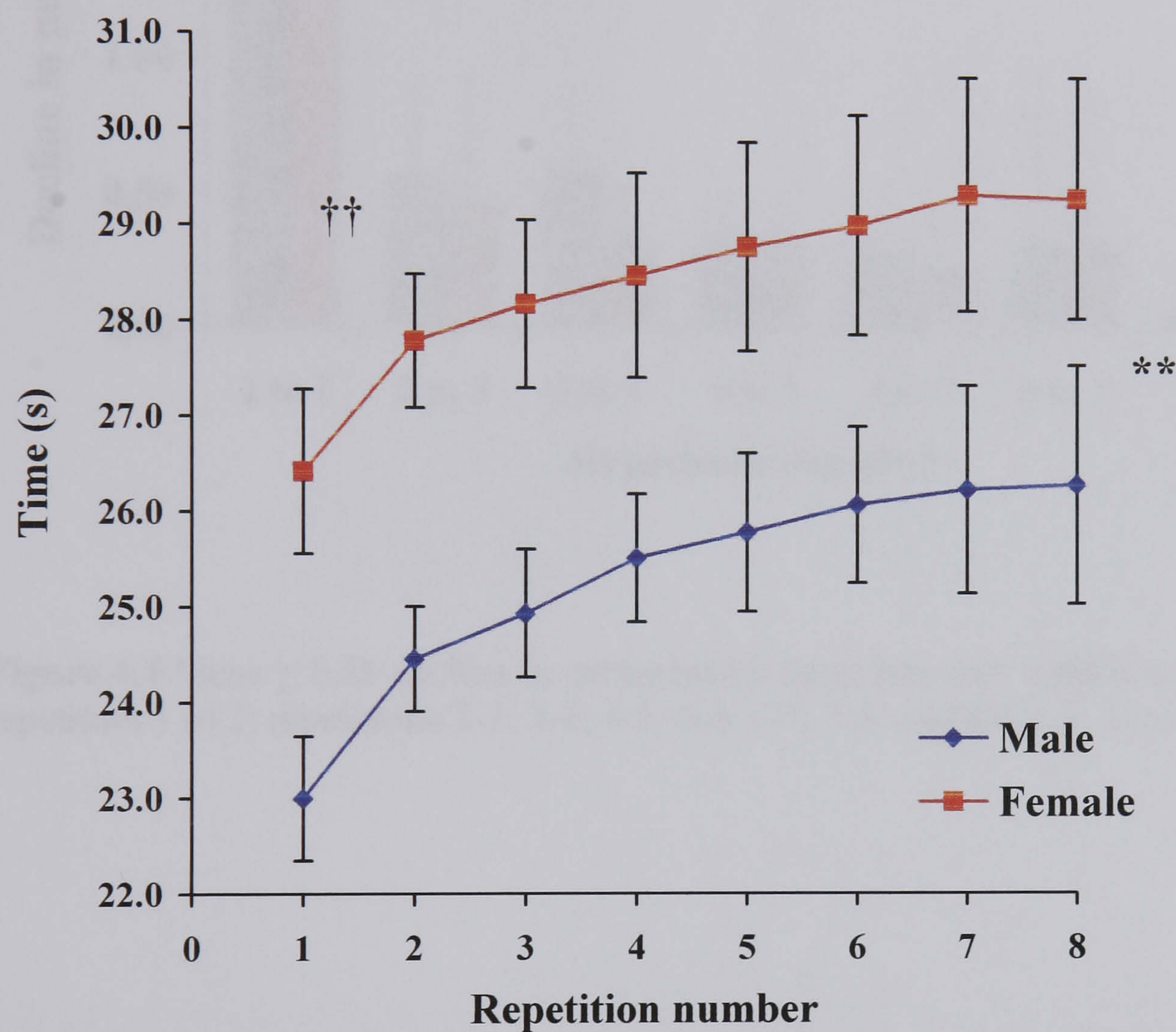


Figure 4.3 Mean performance times \pm S.D. for male and female subjects in the 8 x 50 yards test (** $P<0.01$ male vs. female for all repetitions; †† $P<0.01$ repetition 1 vs. repetitions 2-8).

Decline in performance times during the 8 x 50 yards test was greatest from the first to the second sprint (1.46 ± 0.58 s for males and 1.37 ± 0.56 s for females). The decline between repetitions reduced as the 8 x 50 yards test progressed and was least between repetitions 7 and 8 (males 0.05 ± 0.23 ; females 0.09 ± 0.36 ; Figure 4.4).

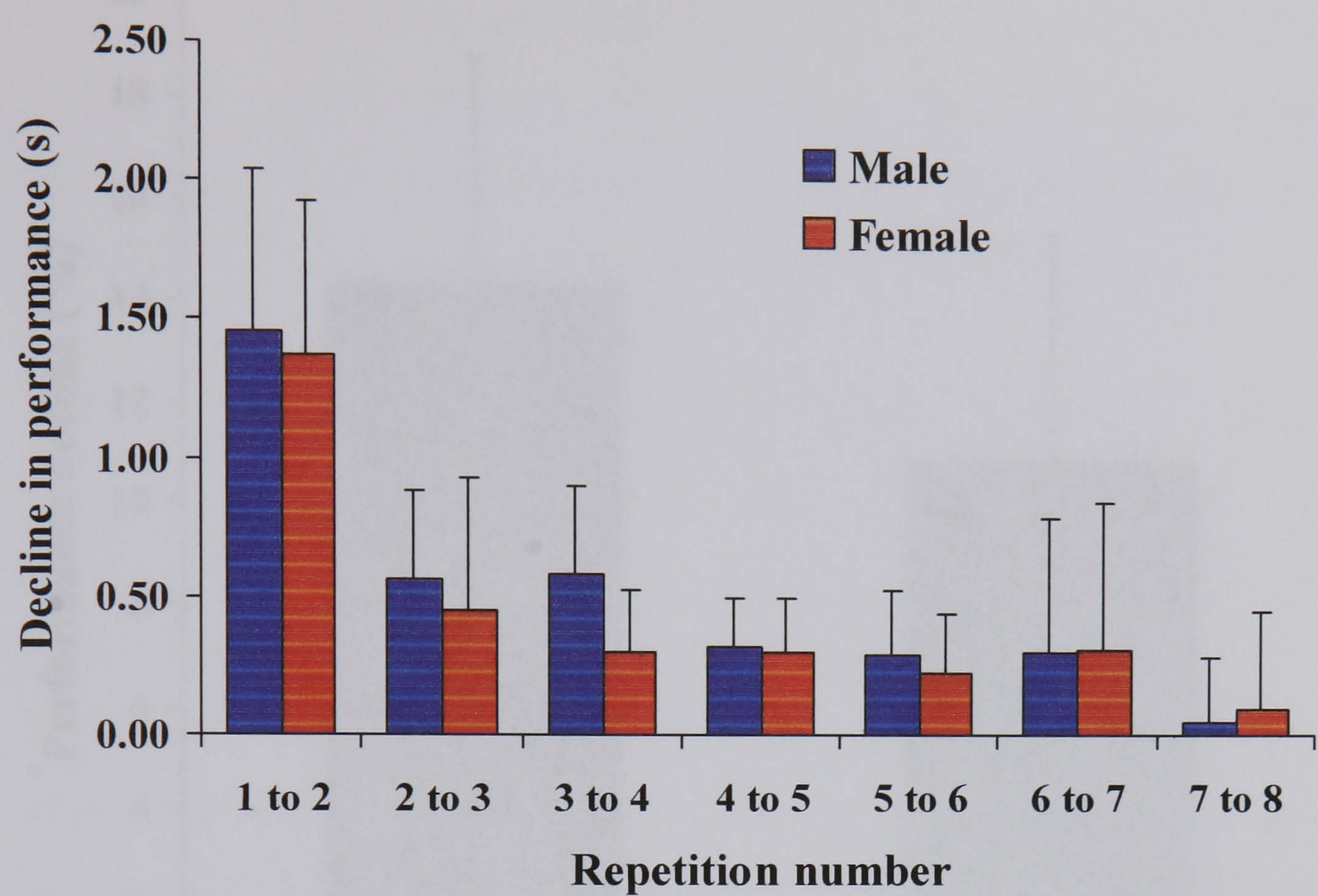


Figure 4.4 Mean \pm S.D. decline in performance times between repetitions ($P<0.01$ repetition 1 to 2; repetitions 2-3, 3-4, 4-5, 5-6, 6-7, 7-8, and male vs. female, N.S.).

Mean performance times during 8 x 50 yards sprinting declined from the first to last sprint by $14.3 \pm 4.5\%$ and $10.9 \pm 4.5\%$ for male and female swimmers respectively (male vs. female, NS; Figure 4.5).

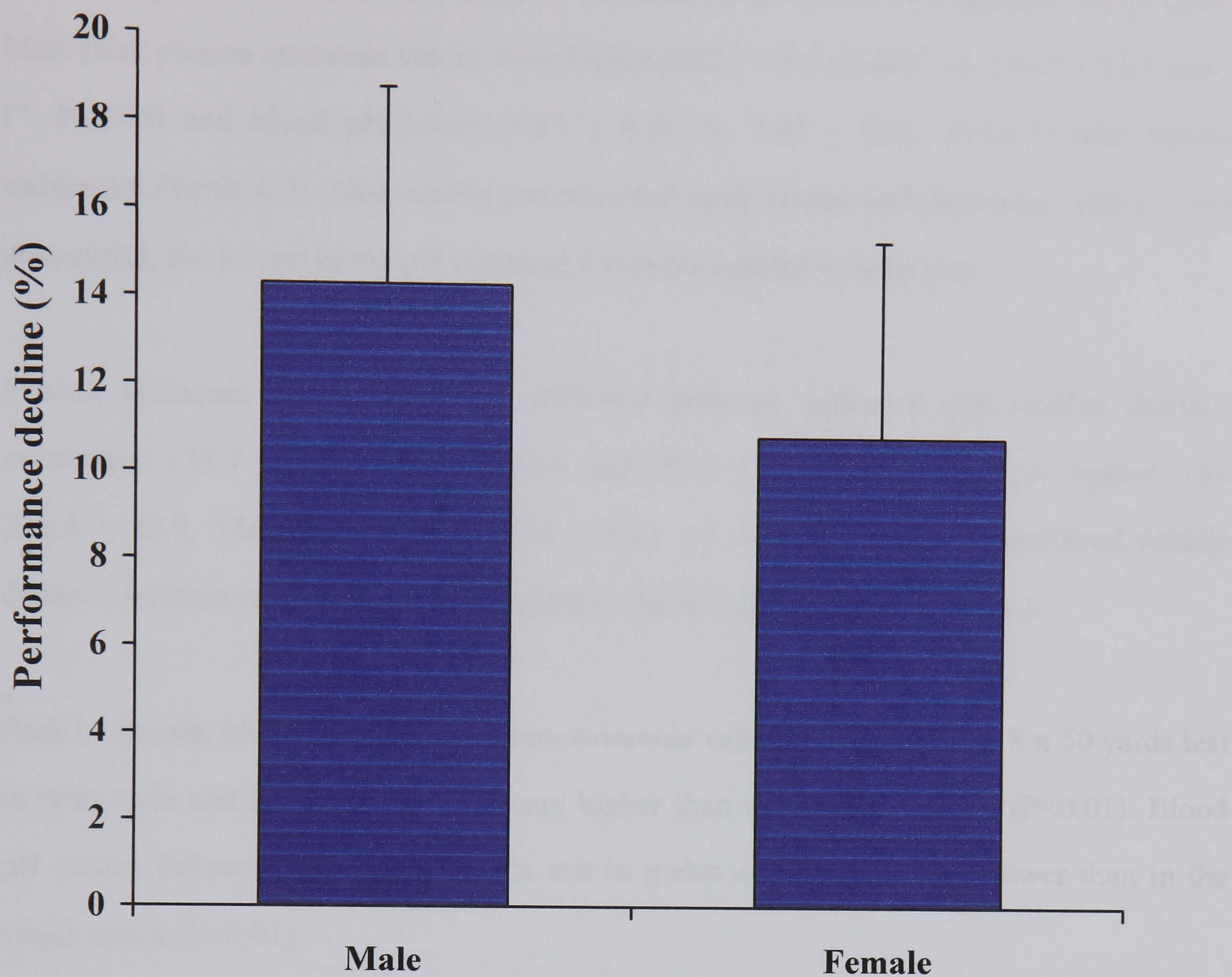


Figure 4.5 Percent decline in performance \pm S.D. from repetition number 1 to 8 (male vs. female, NS).

4.3.2 Metabolic responses

No differences were observed between males in females in resting values, or in heart rate, pH and ammonia following the 50 yards sprint. Peak capillary blood lactate concentrations rose to 9.9 ± 1.3 mmol l⁻¹ for males and 8.6 ± 2.4 mmol l⁻¹ for females following the 1 x 50 yards sprint test (P<0.05). Following the 8 x 50 yards test, peak blood lactates were 18.7 ± 2.7 mmol l⁻¹ and 14.4 ± 2.5 mmol l⁻¹ for male and female swimmers respectively (P<0.01).

Values ranged from 15.1 to 23.9 mmol l⁻¹ for males and 11.2 to 19.4 mmol l⁻¹ for females. Male peak plasma ammonia values were higher ($232 \pm 73.0 \mu\text{mol l}^{-1}$ vs. $154.3 \pm 62.6 \mu\text{mol l}^{-1}$, $P<0.05$) and blood pH lower (6.97 ± 0.06 vs. 7.05 ± 0.05 , $P<0.01$) than female swimmers (Table 4.2). Most swimmers recorded peak lactate and ammonia values 5 min post-sprint, but lowest blood pH occurred 1 min post-sprint in both tests.

Plasma ammonia values were not different between sprinters and middle distance swimmers: 233.7 ± 109.2 , 172.3 ± 90.1 and 203.0 ± 98.3 for sprinters, compared with 230.4 ± 22.9 , 136.4 ± 13.0 and 183.4 ± 53.1 for male, female and combined middle distance swimmers, respectively (all sprint vs. middle distance groups, N.S.).

Peak heart rate, blood lactate and plasma ammonia values recorded in the 8 x 50 yards test in both male and female swimmers were higher than in the single sprint ($P<0.01$). Blood pH values following the 8 x 50 yards test in males and females were lower than in the single sprint ($P<0.01$).

Table 4.2 Metabolic responses of male and female swimmers to single and repeated sprint swimming (n = 8; data are the mean \pm S.D.; * $P<0.05$ male vs. female, ^a $P<0.05$ 1 x 50 yards vs. 8 x 50 yards, ** $P<0.01$ male vs. female, ^{aa} $P<0.01$ 1 x 50 yards vs. 8 x 50 yards).

Test	50 yards sprint				8 x 50 yards			
	Female		Male		Female		Male	
Sample	Pre-exercise	Peak	Pre-exercise	Peak	Pre-exercise	Peak	Pre-exercise	Peak
Heart rate	114	175	109	179	112 \pm 16	190	110	190**
(beats min ⁻¹) ^{aa}	\pm 18	\pm 7	\pm 21	\pm 16		\pm 4	\pm 20	\pm 14
Blood lactate	1.3	8.6	1.6	9.9 *	1.1 \pm 0.6	14.4	1.1	18.6**
(mmol l ⁻¹) ^{aa}	\pm 1.1	\pm 2.4	\pm 0.6	\pm 1.3		\pm 2.5	\pm 0.6	\pm 2.7
Ammonia	31.6	48.0	34.9	89.9	31.1 \pm 8.7	154.3	34.1	232.0*
($\mu\text{mol l}^{-1}$) ^{aa}	\pm 10.5	\pm 44.7	\pm 9.0	\pm 29.0		\pm 62.6	\pm 20.3	\pm 73.0
Blood pH ^a	7.37	7.14	7.38	7.11	7.39	7.05	7.37	6.97**
	\pm 0.01	\pm 0.06	\pm 0.04	\pm 0.04	\pm 0.02	\pm 0.05	\pm 0.03	\pm 0.06
Plasma volume		-7.3		-7.4		-8.9		-11.0
(% change)		\pm 4.1		\pm 6.3		\pm 6.8		\pm 4.1

4.4 Discussion

The purpose of this study was to describe and compare the physiological responses to single and repeated sprint swimming in elite male and female swimmers. Metabolic responses were far greater following a typical repeated sprint training set (8 x 50 yards) compared with a single 50 yards sprint bout. Male and female metabolic responses to single sprints were similar, but males had a significantly greater response following 8 x 50 yards sprints than females.

Times recorded in the 50 yards sprint were used as a reference of the current sprint performance status of the swimmers. No significant differences were observed between the single sprint and the first sprint of the 8 x 50 yards test, an indication that a maximal sprint was recorded in both tests and that the swimmers were highly motivated. However, personal best times were significantly faster than those recorded in the 50 yards test and the first repetition of the 8 x 50 yards training set. This was probably due to lack of specific competition preparation i.e. lack of 'taper' and 'shaving down' (Costill et al., 1992; Sharp and Costill, 1989). Three to four percent improvements in performance time after a taper have been reported (Costill et al., 1985c; Neufer et al., 1987). Mean 50 yards times were 3.8% and 3.3% slower than personal bests for male and female swimmers, respectively. The first repetition of the 8 x 50 yards test was 4.1% slower than personal best times for both males and females, within the range suggested for non-tapered, non-competitive performance (Costill, 1999; Troup et al., 1994).

The only significant decline in performance during the 8 x 50 yards test occurred between the first and second sprints (Figure 4.3). Repeated, maximal exercise over ~22-

26 s will greatly reduce muscle PCr and therefore slow ATP resynthesis and speed or force of muscular contraction (Gaitanos et al., 1993; Hasson and Barnes, 1986; Serresse et al., 1988). In addition, the rest interval of ~65 s is inadequate to allow total regeneration of PCr (PCr resynthesis half time ~57 s; Bogdanis et al., 1995). Furthermore, an increase in lactate and concomitant H^+ production would lower pH, contribute to gradually greater fatigue and reduce performance. The significant drop in performance time between repetitions one and two may reflect glycolytic inhibition from repetition two onwards. A drop in pH has been shown previously to inhibit the regulatory enzymes phosphorylase and PFK, thereby reducing the glycolytic rate and the rate of energy delivery for muscular contraction (Hermansen, 1981; see section 2.2.4, p15). Perhaps the main source of energy supply from repetitions two to eight was provided through aerobic sources (slower energy delivery) with some contribution from the recovery of PCr.

Mean performance times between the first and eighth repetition declined by 14.3% and 10.9% for male and female swimmers respectively. Individual values ranged from 11.7% to 23.4% for males and 6.9% to 17.7% for females, perhaps reflecting the mixture of sprint and endurance swimmers used in this sample group. When sprint and middle distance specialists were compared, mean decline in performance between repetition one and eight was significantly higher in sprint swimmers. Sprint male, female and overall decline was 17.0 ± 5.5 , 14.2 ± 3.3 , $15.6 \pm 4.5\%$ respectively, compared to 11.7 ± 0.9 , 7.9 ± 3.2 , $9.8 \pm 3.0\%$ for the middle distance swimmers in this study. This suggests that the performance differences to repeated sprint swimming will be determined more by training specialism (sprint or endurance), than by sex.

Male performances were faster than females in all sprints ($P < 0.01$), and were 14.9% and 11.4% faster on the first and eighth sprint, respectively. Personal best times in this subject group were 14.8% slower and test 50 yards times 14.3% slower in females than males, a slightly greater difference than that between the male and female 50 m world records (11.5%). Higher percentage body fat for females (and therefore lower active muscle mass) in similar level swimmers (~10% for males and ~18% females: Heusner, 1985; ~9% for male and ~16% for females: Troup et al., 1994) suggest that performance differences between the sexes in this study are largely due to a greater muscle mass and consequently a greater upper body muscle power which has been shown to be highly correlated with sprint swimming speed (Costill et al., 1983; Sharp et al., 1982). It has been suggested that even in competitive swimmers, females are able to generate relatively less propulsion from the arm stroke (than legs) than males, recording on average only 50% of the upper body strength available to their male counterparts (Costill et al., 1992).

Measurement of strength represented per kilogram of muscle mass (NSCA Roundtable, 1985) is a more accurate method to compare the strength of muscle fibers, although strength per kilogram of total body mass reflects the power available for swimming performance (Maglischo, 1993; Lavoie and Montpetit, 1986). On this basis female swimming times reflect a lower muscle power than that of the male swimmers. Blood lactate was ~29% and plasma ammonia ~50% higher in males than females. When this was represented per kg body mass, the values dropped to ~7% ($P < 0.05$) and ~25% ($P < 0.05$) for blood lactate and plasma ammonia respectively.

Peak capillary blood lactate values were observed in most subjects at 3 min after the 50 yards sprint and 5 min after the 8 x 50 yards sprints. Single 50 yards lactate values are similar to those reported by Bonifazi et al. (1993) and Chatard et al. (1988), although only approximately half of the concentration reached following the repeated sprints test (Table 4.2). While it is possible that the increase in H^+ , reflected in the decline in blood pH and associated with increases in blood lactate, limited performance in the 50 yards sprint, pH was much lower following the 8 x 50 yards set. Therefore, a major limitation to single sprint performance, in addition to the degree of muscle power generation, may be the decline in PCr and the slower rate of energy supply from other sources, such as anaerobic and aerobic glycolysis.

Blood lactate values following repeated sprints were higher than the 9-15 mmol l⁻¹ reported by Holmer (1972) and Nadel et al. (1974) for maximal, single-effort 100 and 200 m non-competitive swimming. If a training effect is to be achieved from this type of repeated sprint set, metabolic responses appear to be more specifically suited to competitive events of longer duration than 50 m (e.g. 200 m; Sawka et al., 1979). Small, but significant differences in blood lactate between males and females following 50 yards sprinting were greatly extended during 8 x 50 yards (mean values 1.3 and 4.3 mmol l⁻¹ higher in males than females for 50 yards and 8 x 50 yards, respectively). Similar sex differences have been found in other forms of exercise with higher peak power and rate of decline, greater total work and higher blood lactate reported (Froese and Houston, 1987; Jacobs et al., 1983). Higher blood lactate values in males in the present study perhaps reflect the greater differential in muscle mass involved in sprint swimming (largely arm dominated which shows greater difference between males and

females: NSCA Roundtable, 1985; Wells, 1986) and a greater total work carried out by male swimmers in the ~12% faster performance times throughout the 8 repetitions.

Higher blood lactate levels in males than females have been reported following maximal cycling (Jacobs and Tesch, 1981; Jacobs et al., 1983; Karlsson et al., 1981; Komi and Karlsson, 1978), perhaps due to the larger muscle mass involved during the sprint. However, Telford et al, (1988) have shown that in highly-ranked male and female Australian swimmers, post-competition blood lactate values reach similar values, suggesting that the competitive standard and motivation of the subject group may discriminate better between subjects with high blood lactate concentrations. Significant differences between males and females have been reported in 'anaerobic power and capacity' tests, although many sprint tests find that differences observed between male and female subjects diminish or are completely removed when values are reported relative to body weight or fat free mass (Bishop et al., 1985; Maud et al., 1986; Wells, 1984).

The decline in blood pH was ~0.2 following single and ~0.3-0.4 following repeated sprint swimming performance, similar to those reported using other exercise modes (Allsop et al., 1990; Cheetham et al., 1986). The pattern of decline matched the extent of the increase in blood lactate with values being significantly lower for males than females, and lower for all subjects following repeated as opposed to single 50 yards sprint swimming (first demonstrated in cycling and running by Hermansen and Osnes, 1972). The post-sprint pH sex difference supports the suggestion that a larger muscle mass is being recruited (Mayhew and Salm, 1990). Lactate production is responsible for

>85% of H^+ released into the blood (Sahlin, 1986), and suggests a causal relationship between the lactate and pH differences between male and female swimmers.

Resting ammonia values were typical of those previously reported (Prellwitz et al., 1973), but lower than those for elite swimmers following ~22-26 s sprint swimming (Strass et al., 1994) in a swimming flume. Relatively high values ($\sim 140 - 160 \mu\text{mol l}^{-1}$) were reported despite relatively low ($\sim 6 - 7 \text{ mmol l}^{-1}$) blood lactate values, although lack of information contained in this abstract makes interpretation of this information difficult. Peak 50 yards plasma ammonia values are lower than those recorded in low-level adult swimmers for 100 m, but higher than those for 20 m sprinting (Prado, 1999).

Higher peak plasma ammonia values in the 8 x 50 yards set support the extent of greater metabolic responses following repeated as opposed to single sprints, and illustrate a greater response in males than females (Itoh and Ohkuwa, 1993). Values were similar to those reported in other exercise modes for high intensity exercise (Buono et al., 1984; Graham et al., 1990; Nevill et al., 1989; Cherry et al., 1998). The enzyme AMP deaminase which catalyses the breakdown of AMP to inosine monophosphate (IMP) and accelerates purine metabolism (ammonia is the end product) has been shown to be prominent in type II, but not type I fibres (Dudley et al., 1983; Meyer et al., 1980). This may imply that in this study males have a higher percentage of type II fibres than females, but blood as opposed to muscle values may only allow speculation in this study. It is more likely that a larger active muscle mass (from an ~30% larger body mass) and mean muscle fibre size (30% larger in males: Nygaard, 1982) are more likely causes of higher plasma ammonia concentrations in males.

The results of this study show that the known competitive performance differences between male and female swimmers can be replicated in single and repeated sprint swimming. The significantly greater metabolic responses in males as indicated by higher blood lactate and plasma ammonia and lower blood pH values suggest a larger active muscle mass during sprint swimming and suggest that female swimmers who are able to increase muscle mass, may also be able to improve their sprint swimming performance to a greater extent. Metabolic differences following repeated sprints suggest that the typical training set of 8 x 50 yards used in this study may be more specific training for longer distances such as 100 and 200 m swimming races.

CHAPTER V

ESTIMATING THE ENERGY CONTRIBUTION DURING SINGLE AND REPEATED HIGH INTENSITY TETHERED SWIMMING

5.1 Introduction

The importance of identifying the energy contribution to competitive swimming events has long been the subject of interest for scientists and coaches in prescribing training. Estimates for a 50 m swimming race range from 98% and 2% (Maglischo, 1993) to ~70% and ~30% (Ring et al., 1996; Troup, 1990) for anaerobic and aerobic energy contributions respectively. Thus, no clear consensus exists between scientists or coaches regarding the energy contribution to short, single sprints.

Recent studies that have considered the relative energy contribution to sprint cycling and running have suggested a far greater aerobic contribution to sprinting than was previously thought (see Gastin, 2001). This has been supported recently in swimming by mathematical modelling (Ring et al., 1996) and oxygen deficit studies conducted in a swimming flume (Troup et al., 1992b; Troup and Trappe, 1994). No information exists about the energy contribution to repeated sprints commonly used in swimming training, although it is known that for a second 30 s sprint cycling bout, 4 min after a first, there is an ~44% aerobic contribution to energy supply (Bogdanis et al., 1995).

Thus, there are conflicting reports regarding the relative energy contribution to 50 m sprint swimming and a lack of information in relation to repeated sprint bouts often prescribed in training for this event. Therefore, the purpose of the present study was to estimate the energy contribution to a single 30 s maximal intensity fully tethered swim (FTS), and 4 x 30 s high intensity semi-tethered swimming (STS) bouts.

5.2 Methods

Eight elite, male competitive swimmers (subject characteristics – Table 5.1) performed a series of tests using a tethered swimming mechanism (see Figures 3.1 and 3.2). Three initial tests were performed: a 4-stage submaximal progressive STS test (section 3.4.2.4), a VO₂ max STS test (section 3.4.2.3) and a maximal 30 s FTS sprint (section 3.4.2.5). Subjects then performed 4 x 30 s STS bouts with an applied load equivalent to 95% of the mean load (ML) achieved in the 30 s FTS, each separated by 30 s of passive rest. Front Crawl was used in all tests; during which expired air samples and stroke rate data were collected. Capillary blood samples were collected pre-test and 1 min post-Sprint 4 for the determination of pH and lactate.

Table 5.1 Subject physical characteristics and personal best (P.B.) times (n = 8; data are the mean ± S.D.).

Subjects	Age (years)	Height (cm)	Weight (kg)	VO ₂ max (l min ⁻¹)	100 m F/C P.B. (s)
Mean	20	182.0	75.0	4.2	53.92
S.D.	± 2	± 5.4	± 5.5	± 0.3	± 1.39

P.B.: Personal best time; F/C: Front crawl

Accumulated oxygen deficit was determined according to Medbø et al., (1988) and assumed to be the difference between estimated oxygen demand and oxygen uptake. A regression equation was calculated between swimming load and oxygen consumption measured during the submaximal test (mean: $r = 0.990$). The mean load achieved during the 30 s maximal sprint and during the repeated 30 s sprints was entered into the regression equation and the estimated oxygen demand for each sprint was calculated (see dotted arrow, Figure 5.1). The oxygen consumption measured during each sprint (maximal 30 s and 4 x 30 s) was subtracted from the oxygen demand and the oxygen deficit calculated. The oxygen deficit was assumed to be the anaerobic energy contribution to sprinting and calculated as a percentage of the total energy demand.

A one-way analysis of variance was used to examine differences between means, and significant differences located using the Tukey post-hoc test. Relationships between variables were examined using the Pearson product moment correlation coefficient. Values are presented as means \pm standard deviation (S.D.).

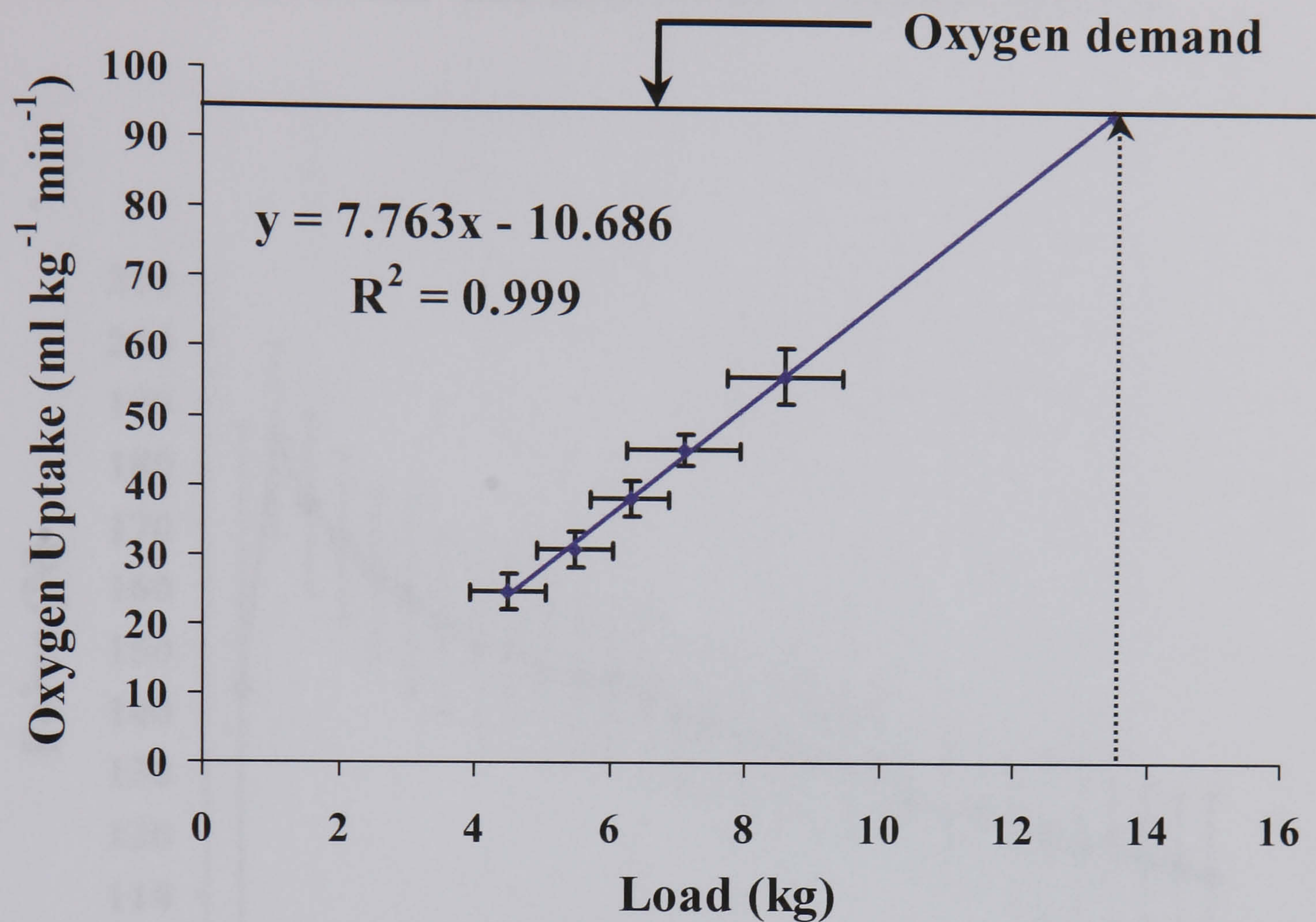


Figure 5.1 Calculating oxygen demand from submaximal swimming (extrapolated regression line) and the average load measured during each 30 s sprint (dotted arrow).

5.3 Results

5.3.1 Maximal 30 s sprint

The computer averaged force output over each 1 s period during the 30 s sprints (Figure 5.2). Peak force was 186.2 ± 16.4 N and achieved by two subjects by the 1st and six subjects by the 2nd second of this sprint. Power output declined in all subjects following 2 s of sprinting to a minimum value of 113.4 ± 13.3 N with a mean fatigue index of $38.7 \pm 8.5\%$. Mean force production during the maximal 30 s FTS sprint was 138.7 ± 18.4 N, which corresponds to a mean load of 14.1 ± 1.5 kg (Figure 5.2). The mean load for swimmers during the 4 x 30 s sprints was therefore set at 13.4 ± 1.1 kg (95% ML).

Stroke rate declined ($P<0.05$) during the last 15 s of the 30 s sprint dropping from 114 ± 17.0 to 96 ± 22.6 strokes min^{-1} from the first to last 5 s segment (Figure 5.3).

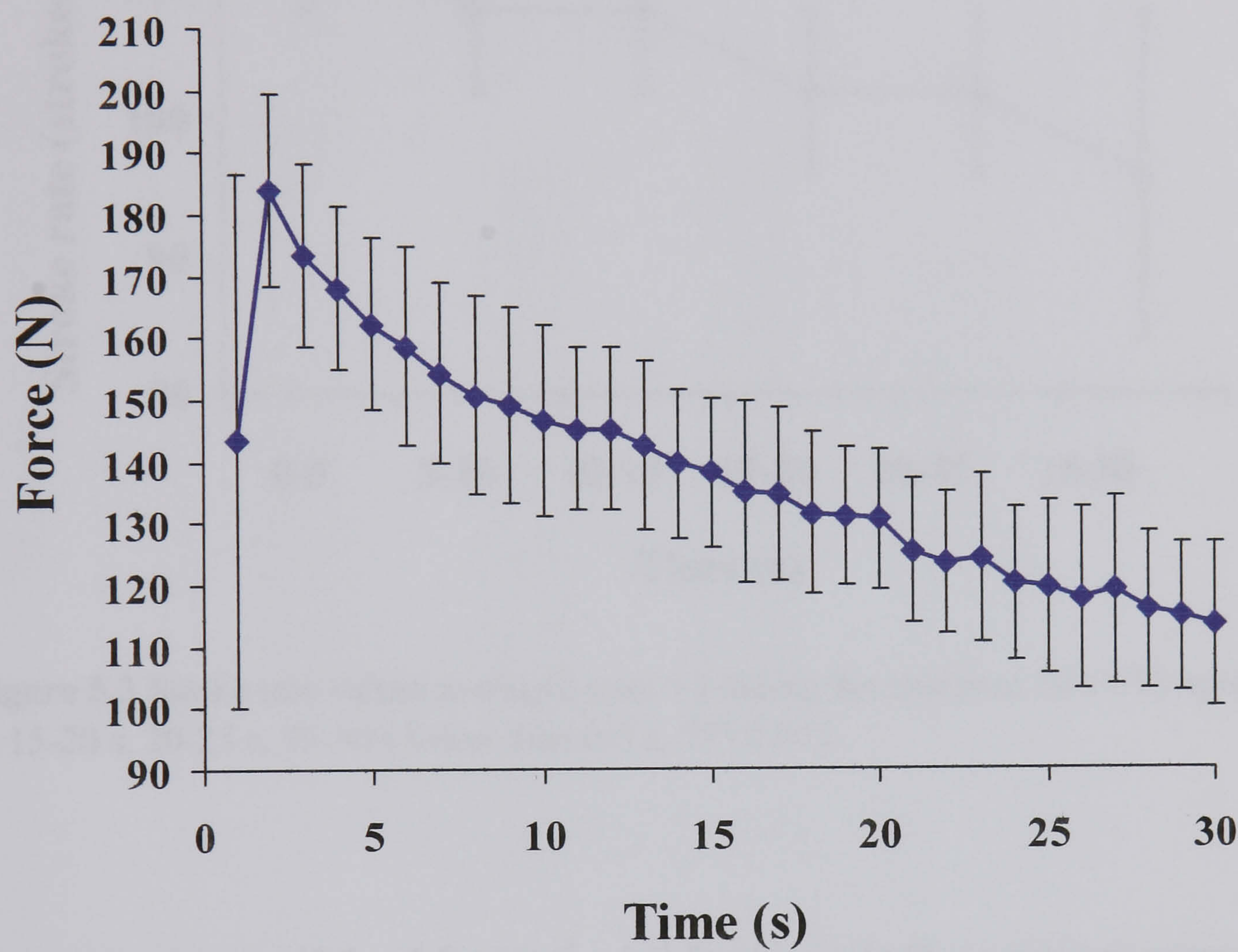


Figure 5.2 Mean force output for 30 s maximal tethered swimming ($n = 8$).

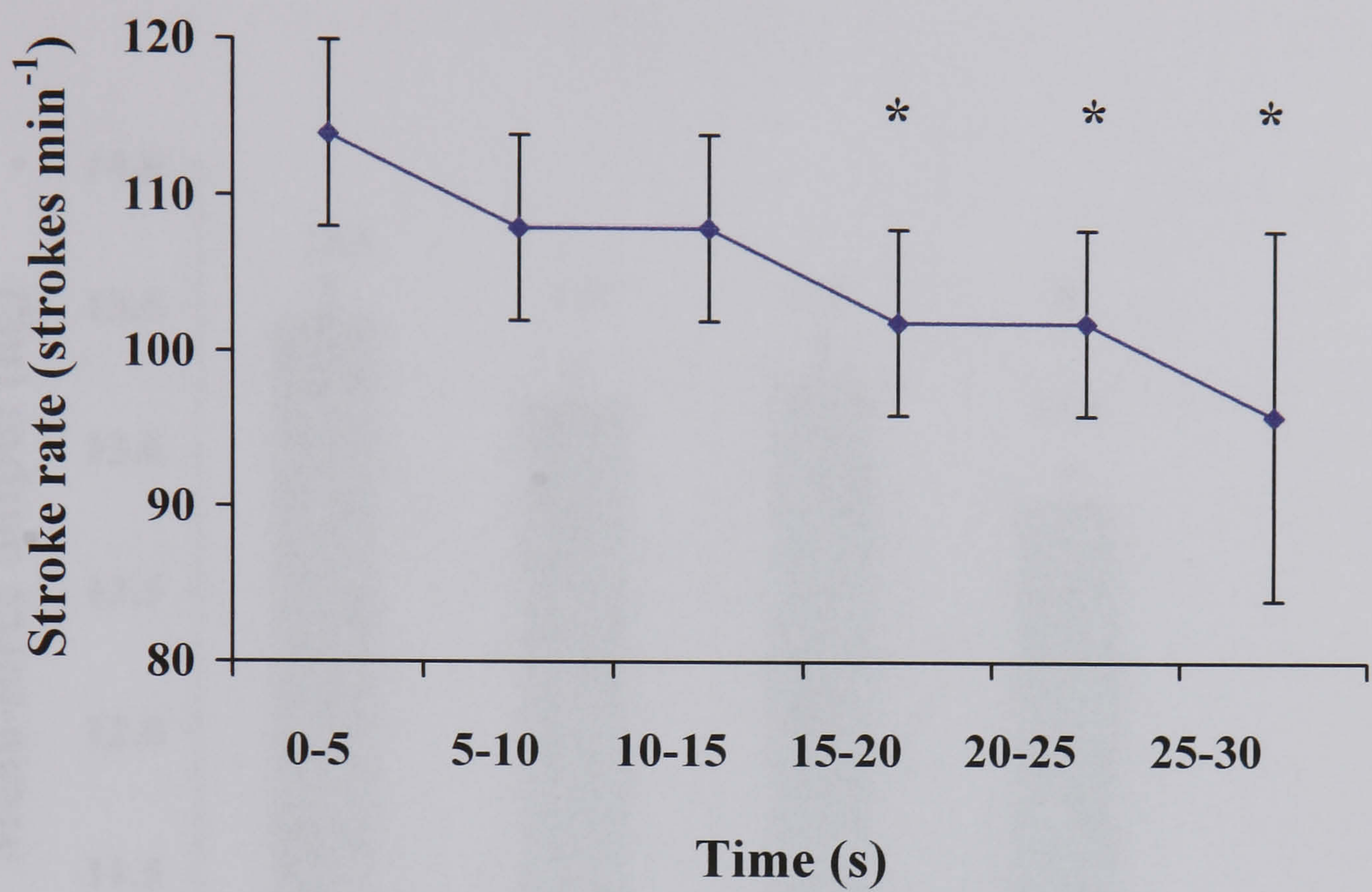


Figure 5.3 Stroke rate values averaged over 5 s during the maximal 30 s FTS sprint (n = 8; 15-20 s, 20-25 s, 25-30 s lower than 0-5 s, *P<0.05).

Oxygen uptake was $33.2 \pm 2.5 \text{ ml kg}^{-1} \text{ min}^{-1}$ for the single 30 s sprint and extrapolation of the regression line up to the mean force produced during the single FTS 30 s sprint and revealed an oxygen demand of $98.9 \pm 13.2 \text{ ml kg}^{-1} \text{ min}^{-1}$. Accumulated oxygen deficit was $65.6 \pm 12.8 \text{ ml kg}^{-1} \text{ min}^{-1}$, for the 30 s sprint, and therefore estimated anaerobic energy contribution was $66.1 \pm 7.3\%$ for the 30 s FTS sprint.

5.3.2 Repeated sprints

The mean load for swimmers during the 4 x 30 s STS sprints was set at $13.4 \pm 1.1 \text{ kg}$ (95% ML), although mean actual measured loads for each repetition were 13.5 ± 0.9 ,

13.2 ± 0.9, 13.2 ± 0.9, 12.8 ± 1.0 kg for bouts 1 to 4, respectively (bout 1 vs. bout 4, P<0.05; Figure 5.4).

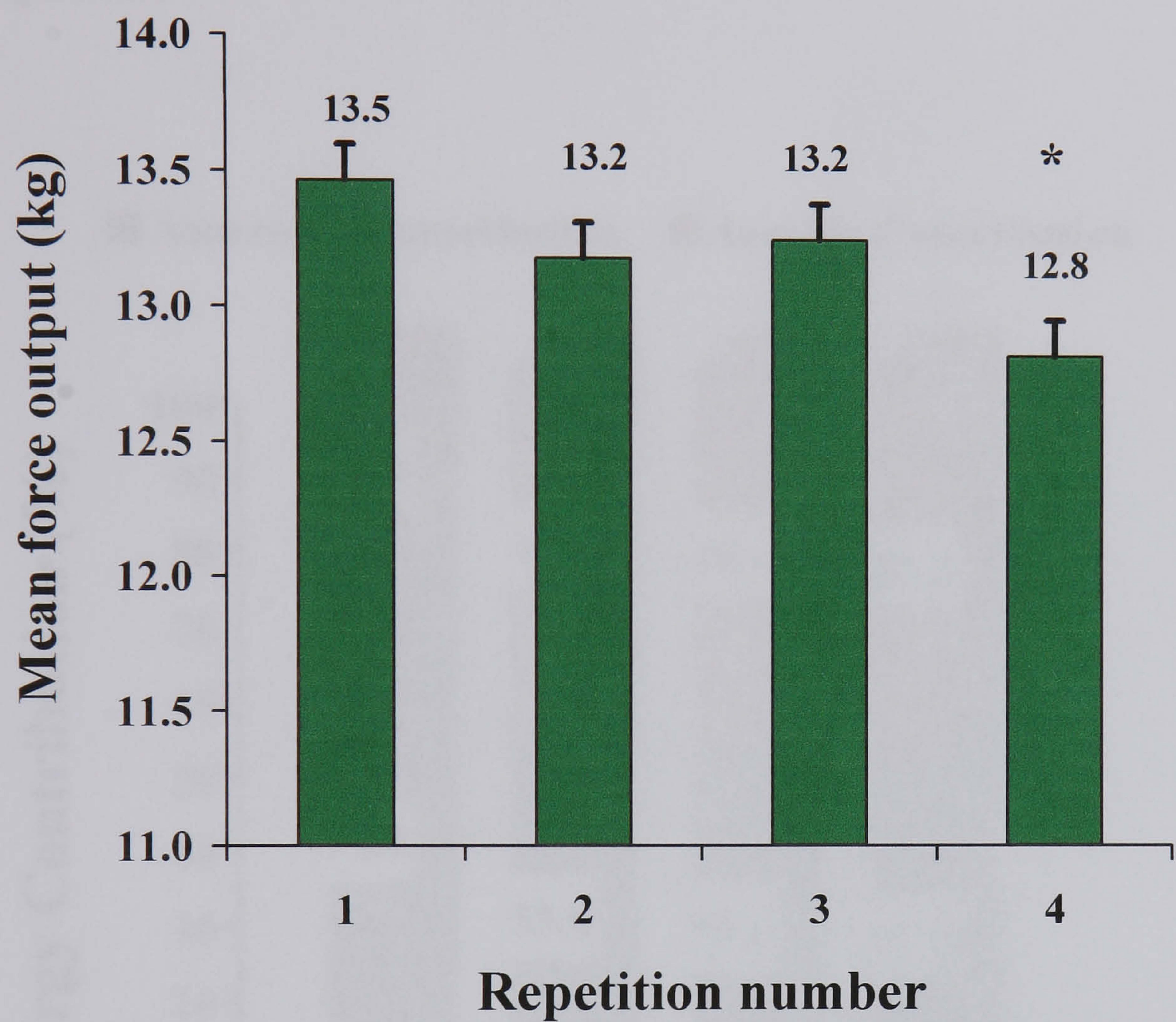


Figure 5.4 Mean load measured during each repetitions during 4 x 30 s STS (n = 8; bout 1 vs. bout 4, *P<0.05).

Oxygen uptake was 24.4 ± 4.1, 43.3 ± 5.1, 45.6 ± 3.6, 46.9 ± 4.0 ml kg⁻¹ min⁻¹ for bouts 1 to 4 respectively (bout 1 vs. bouts 2-4, P<0.01) and extrapolation of the regression line to the mean force produced during each of the four STS bouts, revealed an oxygen demand of 94.0 ± 11.8, 92.8 ± 12.1, 93.3 ± 11.8, 90.9 ± 11.6 ml kg⁻¹ min⁻¹ respectively. Accumulated oxygen deficit was 69.6 ± 9.5, 49.5 ± 10.8, 47.7 ± 10.5 and 43.9 ± 10.8 ml

$\text{kg}^{-1} \text{ min}^{-1}$ (bout 1 vs. bouts 2-4, $P<0.01$) for the 4 x 30 s bouts respectively. Estimated anaerobic energy contribution therefore was $74 \pm 3.3\%$, $53.0 \pm 5.9\%$, $50.6 \pm 5.3\%$, and $47.8 \pm 6.3\%$ for bouts 1 to 4 during the 4 x 30 s STS test (bout 1 vs. bouts 2-4, $P<0.01$; Figure 5.5).

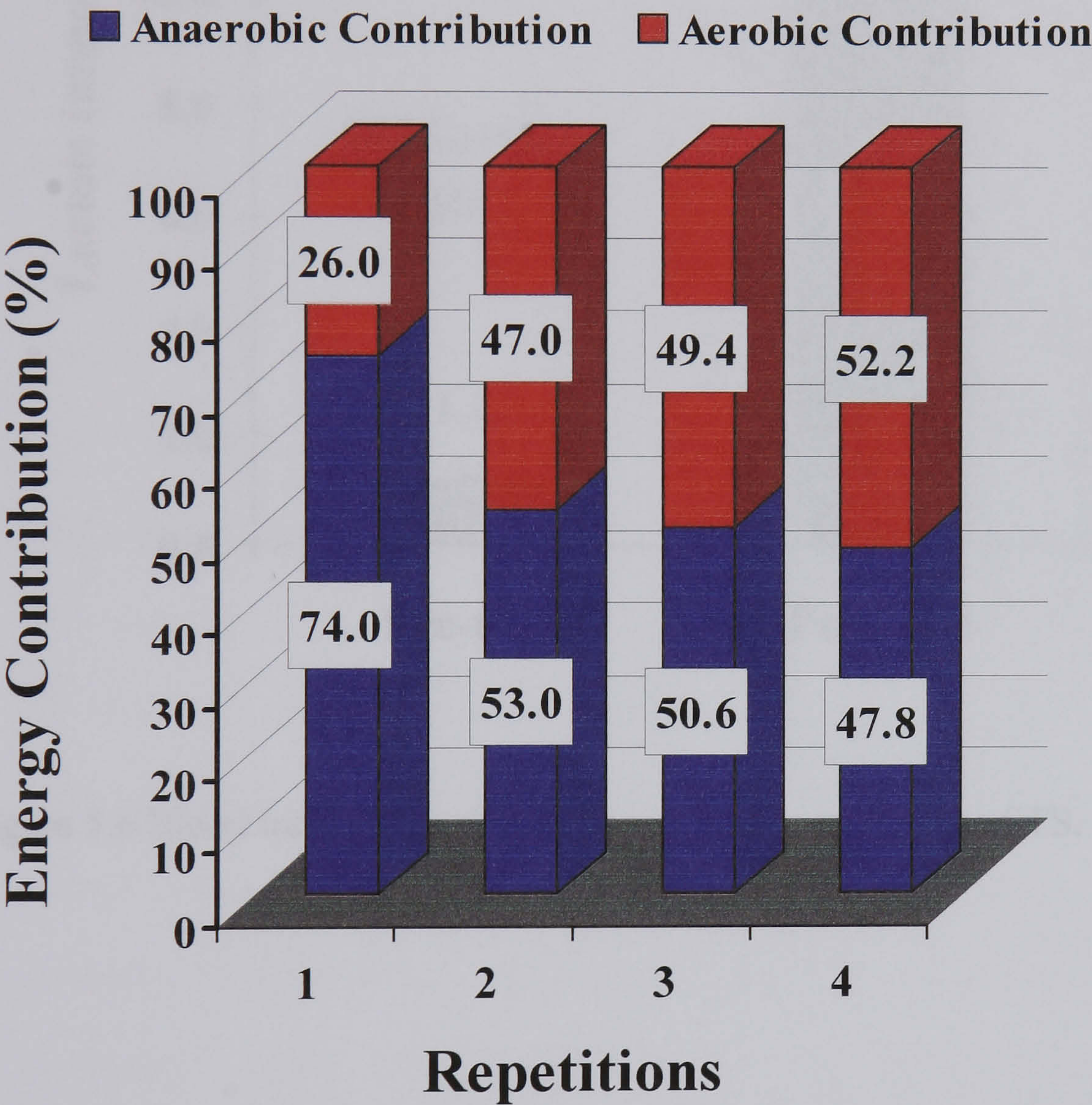


Figure 5.5 Estimated energy contribution during 4 x 30 s STS ($n = 8$; bout 1 vs. bouts 2-4, $P<0.01$).

Blood lactate and pH one min after the fourth bout in the 4 x 30 s STS test were $12.1 \pm 3.6 \text{ mmol l}^{-1}$ and 7.23 ± 0.10 respectively (Figure 5.6).

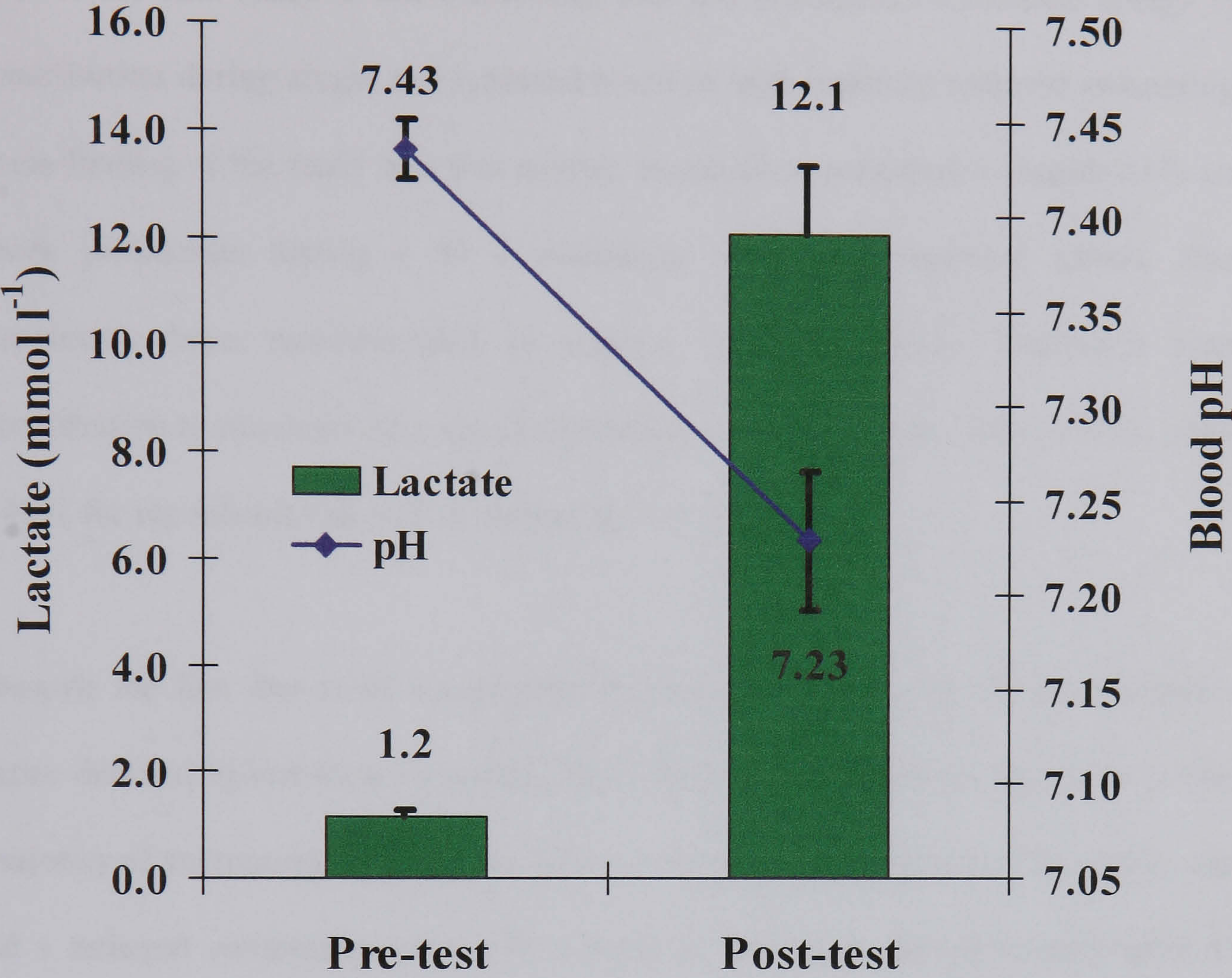


Figure 5.6 Blood lactate and pH values pre- and post- 4 x 30 s STS.

5.4 Discussion

This is the first study to our knowledge that has attempted to estimate energy system contribution during single and repeated bouts of high intensity tethered swimming. The main finding of the study was that aerobic metabolism contributes considerably more to work production during a 30 s swimming sprint and repeated sprints, than has previously been recommended in popular swimming texts. Estimated anaerobic contribution to maximal 30 s sprint swimming was ~66%, and ~74%, ~53%, ~51% and ~48% for repetitions one to four during the 4 x 30 s test.

Despite the fact that most competitive swimming distances are of short duration and more detailed information regarding high velocity swimming energetics is needed, the majority of swimming research has focused on submaximal efforts. The single, maximal 30 s tethered swimming sprint (30 s max) in this study found an estimated aerobic contribution to the energy demand of ~34%. This value is similar to that found in elite swimmers during 50 m flume swimming (31%; Troup, 1990), although higher than that recorded during 50 m free swimming (22%; Ring et al., 1996) using mathematical modeling. Trappe (1996) reported ~19% aerobic contribution for 50 m swimming competitive events, while recently Capelli et al. (1998) estimated ~15% aerobic contribution during 25 s of high intensity crawl swimming using a theoretical model to calculate energetics. The range of aerobic contribution for 50 m sprinting for the four competitive strokes was between 15.4% (front crawl) and 27.1% (breaststroke). The mean race time for breaststroke was ~33 s, close to the duration used during maximal tethered swimming in the present study.

Although the muscle biopsy technique is the most accurate and direct method for quantification of energy demands during sprinting, no direct measurements of the metabolic demands during sprint swimming have been published, perhaps due to the difficulties of obtaining samples in the swimming environment immediately following exercise. Following 30 s of sprint cycling, Bogdanis et al. (1996a) reported a 34% aerobic contribution to energy supply, whilst values of 40% Medbø and Tabata (1989) and 28% Withers et al. (1991) have also been reported.

The range in differences of aerobic contribution in cycling studies of 28-40% can be partly explained by the training status of the subjects (Granier et al., 1995; Scott et al., 1991; Gastin and Lawson, 1994). It has been suggested that training and level of swimming ability may alter the energy system contribution during swimming bouts (Troup et al., 1992a). Nevertheless, the findings from the present study and from cycling studies using muscle biopsy, showed higher aerobic contribution compared with the swimming studies using mathematical modeling (Ring et al., 1996; Capelli et al., 1998). Running studies that employed mathematical modeling techniques also found lower aerobic contribution to sprinting, estimated from 20 and 45 s bouts (~20% for 30 s sprint running: Peronnet and Thibault, 1989; Ward-Smith, 1985). Troup et al. (1992b) found similar results to the present study using the MAOD method during flume swimming, suggesting that the methodology used to estimate energy contribution is important when comparing values from different studies.

The duration of 50 m swimming for elite competitive swimmers is 22-24 s, some 6-8 s shorter than the 30s duration used in this study and in most of the cycling studies. The 6-8 s difference in duration between the 50 m swimming and cycling studies may alter

the metabolic demands. During the last 10-20 s of a sprint, aerobic metabolism is progressively increased in comparison with the initial seconds of the sprint when PCr degradation and glycolysis predominate ATP resynthesis (Bogdanis et al., 1996a). The similarity in results between the present study, those of Troup (1990) and the cycling studies suggest that in estimating energy contribution during sprinting, the duration, rather than the mode of exercise is the crucial factor.

An early increase in the aerobic energy contribution during a 30 s bout may be attributed to a number of factors, such as the rapid activation of glycolysis from the first seconds of the all-out sprint (Boobis et al., 1982; Gaitanos et al., 1993; Maughan et al., 1997), increasing muscle lactate and H^+ with a concomitant drop in muscle pH. A reduction in muscle force in this study after the first 10 s of the 30 s sprint may reflect the dramatic reduction in PCr (Bogdanis et al., 1995; Hirvonen et al., 1987) and the rate of anaerobic glycolysis compared to the first seconds of the sprint (Bogdanis et al., 1996a). It is likely that an increased reliance on aerobic metabolism occurred between 10-20 s, and to an even greater extent during the last 10 s (20-30 s) of the 30 s tethered swimming sprint. The greater contribution of aerobic metabolism will provide ATP at a slower rate and may partly cause the reduction in force. All muscle fibers are activated during the initial seconds of the sprint, when the stroke rate was very high, but type II fibers may become energy depleted earlier than type I during a 30 s sprint (Greenhaff et al., 1994c). Given the early fatigue of type II fibers, reliance on type I slow contracting fibers cannot be excluded. Type I fibers work aerobically further increasing the relative contribution of this mechanism to the total force produced.

Force produced by the swimmers during the 30 s max was rapidly reduced after reaching the peak value (1-2 s) in a similar manner to that observed during sprint cycling (Nevill et al., 1996a) and may be attributed to metabolic, mechanical factors, or a combination of both. The ability to sustain a high stroke rate during the last stages of a 50 m sprint swimming race is a crucial factor for success (Wirtz et al., 1992; Wilke, 1992). In the present study, the stroke rate during the last 15 s of the sprint was decreased compared with the initial 5 s. It is likely that the speed of movement (stroke rate) affects force production during tethered swimming at least up to a rate of 120 strokes min^{-1} (Adams et al., 1984). During 30 s sprint cycling, a reduction in force of ~28% occurs independently from the speed of movement (Beelen et al., 1995). In the present study, force output decreased by ~40% over 30 s while stroke rate decreased only 16%, suggesting that mechanical factors on their own, cannot explain this rapid decrease in the produced force. It is possible then that metabolic factors affected the force production during the last seconds of the sprint and that this may have occurred in concert with the decline in stroke rate.

Estimated energy contribution during maximal intensity repeated sprints in swimming, have not been examined, although studies in cycling have shown that aerobic metabolism increases during repeated high intensity bouts (McCartney et al., 1986; Spriet et al., 1989). Gaitanos et. al. (1993) showed that during the last 6 s sprint in a set of ten sprints, aerobic contribution and PCr were the main sources for energy production, since anaerobic glycolysis was reduced. Bogdanis et. al. (1996) found an increased aerobic contribution in the second 30 s sprint after four minutes of recovery, accounting for ~45% of the energy supply during the second sprint.

Reduction in the rate of anaerobic glycolysis has been frequently reported in studies that have employed repeated exercise bouts, although the reduction in the glycolytic flux was higher than the reduction in the external power (Bogdanis et al., 1996a). Power reductions of ~18-20%, during a second, compared with the first sprint have been reported in cycling studies (Spriet et al., 1989; McCartney et al., 1986; Gaitanos et al., 1993; Bogdanis et al., 1996a). The energy needed to maintain a higher than expected power output following the dramatic reduction in anaerobic glycolysis, was provided from aerobic metabolism (~45% in a second sprint: Bogdanis et al., 1996).

In the present study aerobic contribution contributed ~50% of the total energy demand, even during the last of four sprint bouts (constant applied load). The intensity chosen was not maximal (95% of MFO during 30 s max), but the inability of swimmers to maintain the required force output suggests an element of fatigue (Edwards, 1983) towards the end of the set. The intensity of the 4 x 30 s tethered swimming bouts (~154% of VO_2max) approximates that of 100 m swimming intensity (140% of the VO_2max) during competition (Troup, 1990). Blood lactate values recorded after a 100 m race for swimmers of the same age and performance level were 13.1 mmol l^{-1} (Avlonitou, 1996), similar to the value measured in the present study (12.1 mmol l^{-1}) and confirming the high intensity of the repeated sprint set.

Intensity during the 4 x 30 s bouts was >150% of $\text{VO}_2 \text{ max}$, higher than most studies reporting repeated swimming sprints. During 25 m swimming repetitions at a velocity equivalent to 100 m pace and an exercise to rest ratio of 1:1, Gullstrand and Lawrence (1987) found that blood lactate did not rise above 4 mmol l^{-1} . During 100 m swimming bouts at 120% of VO_2max , the aerobic contribution to energy supply is dominant

(Troup et al., 1992a), whilst maintaining swimming velocity and reducing the resting interval will increase the aerobic contribution further (Barzdukas et al., 1992a). In the present study the exercise to rest ratio was also 1:1, perhaps too short for adequate recovery given the duration of the sprints and the high exercise intensity. It is clear that aerobic metabolism cannot supply energy at the rate required for the exercise intensity used in this study, and may have been a contributory factor in the reduction to ~91% of the 30 s max mean applied force during the last bout. The decrease in force produced during the last bout cannot be attributed solely to mechanical factors or technique as stroke rate was similar during the 4 x 30 s bouts. It is likely that metabolic changes accumulating over the course of the four bouts eventually resulted in fatigue (Edwards, 1983). An H^+ induced inhibition of PFK and phosphorylase were perhaps responsible for the reduction of anaerobic glycolysis, whilst it is possible that H^+ in competing with Ca^{2+} for binding sites in troponin C, reduced the SR sensitivity to Ca^{2+} (Allen et al., 1992) and reduced muscle contractile efficiency. In conditions of low muscle pH and reduced anaerobic glycolysis, PCr and aerobic metabolism are the main contributors to energy production. Phosphocreatine decreases dramatically during a 30 s sprint and its recovery process is very slow (half-time ~57 s: Bogdanis et al., 1995). Following the first two 30 s bouts it is likely that PCr stores were reduced, given the very short recovery period, and therefore aerobic metabolism was required to provided the main source of energy supply.

The higher anaerobic contribution during the first 30 s bout in the 4 x 30 trial (intensity 95%) compared to the single 30 s sprint (intensity 100%), was surprising. It is possible that the different mode of tethered swimming exercise (FTS *vs.* STS) contributed to swimmers using a different technique to lift the load. Differences in tethered swimming

techniques have not been reported and perhaps require further investigation. It is likely that the degree of pacing during the first repetition of the 4 x 30 s set may have contributed to the differences in energy contribution (Cherry et al., 1998) as this mode of exercise has been found to be more economical.

Estimated aerobic contribution to energy supply may have been overestimated in the repeated sprints. The linear extrapolation used to estimate oxygen demand is assumed to be the same during the steady state sub-maximal test and during the 4 x 30 s sprints. It is likely that due to the effects of fatigue as the repeated sprints progressed, efficiency would have decreased and therefore the extrapolated line would have underestimated the oxygen demand. This in turn would have led to an overestimate in the estimated aerobic contribution.

In summary, the estimate of ~66% anaerobic energy contribution for a single 30 s sprint is similar to that reported previously using a swimming flume (Troup, 1990). However, the energy contribution to repeated sprints in swimming has not been previously reported, and by only sprint 4 the anaerobic contribution is reduced to ~48%. This is an important factor when considering the design of swimming training sets, as textbook recommendations for sprint training use durations, intensities and rest intervals similar to those used in the present study. Therefore, the relatively low anaerobic and high aerobic energy contribution during repeated high intensity swimming should be considered when prescribing swimming training sets of this nature.

CHAPTER VI

PHYSIOLOGICAL RESPONSES OF CONTROLLED FREQUENCY BREATHING TO MAXIMAL TETHERED SWIMMING

6.1 Introduction

The use of restricted breathing, or controlled frequency breathing (CFB), in swimming has been employed as a training strategy for many years and is often referred to as ‘hypoxic training’ (Counsilman, 1978; Dicker et al., 1980). It was thought that by limiting inspired air, a reduction in the amount of oxygen available for muscular work would result and therefore cause muscle hypoxia, similar to that experienced at altitude (Kedrowski, 1979). However, the majority of studies reporting the effects of CFB have found that the reduction in $\dot{V}E$ caused by restricting inspired air does not decrease O_2 to the muscles as O_2 extraction is improved. Rather, CFB causes an increase in CO_2 production resulting in hypercapnea (increase in venous partial pressure of CO_2) and a therefore a desire to breath (Coatsee and Terblanche, 1988; Craig, 1979; Dicker et al., 1980; Stager et al., 1986; Town and Vaness, 1990).

It has been suggested that the practice of CFB may cause a relative increase in the ‘oxygen debt’ incurred by the exercise in addition to increasing the amount of anaerobic metabolism and consequently lactate production. The increase in CO_2 production and its acidic state may further add to a fall in pH (Reilly, 1990), and by forcing the athlete to endure a higher level of acidosis it is suggested that the athletes resistance to such

conditions is increased (Kedrowski, 1979). Several studies in swimming and cycling support this idea reporting higher blood lactate values for CFB compared with normal breathing (Kedrowski, 1979; Matheson and McKenzie, 1988; Yamamoto et al., 1980). In contrast, lower blood lactate values have also been observed following CFB in submaximal, endurance swimming (Dicker et al., 1980; Holmer and Gullstrand 1980; Town and Vaness, 1980). Despite the proliferation of swimming studies using CFB, all have been performed using submaximal exercise protocols and consequently blood lactate concentrations are low, below that expected to limit performance. Thus, the effect of CFB on the blood lactate response to sprint swimming has not been examined.

Following a reduced breathing frequency, it is possible that less disturbance to stroke mechanics will result in a reduction in body resistance (Karpovich, 1933) and allow the development of greater forces and consequently higher swimming speeds (Nelson and Goldfuss, 1971). Payton et al. (1999) recently found no difference in underwater pulling patterns when subjects performed either head down or breathing front crawl swimming. Subjects swam at a 'race' speed, but were not fatigued and were observed over a single stroke cycle. If the breathing action does compromise stroke mechanics, it is likely to do so to a greater extent under fatigue conditions, but the effect of CFB on maximal sprinting of durations between 30 and 60 s has not been studied in swimming.

Thus, the purpose of the present study was to examine the effect of CFB on sprint swimming performance and metabolism, using tethered swimming, and to estimate the relative energy contribution to sprinting during CFB. It was hypothesised that restricting the breathing frequency would compromise swimming performance by restricting the aerobic contribution to energy supply.

6.2 Methods

Eight elite, male competitive swimmers (subject characteristics – Table 6.1) performed a series of tests using a tethered swimming mechanism (see Figures 3.1 and 3.2). Three initial tests were performed: a 4-stage submaximal progressive STS test (section 3.4.2.4), a $\dot{V}O_2$ max STS test (section 3.4.2.3) and a maximal 30 s FTS sprint (section 3.4.2.5). Subjects then performed three randomised trials, each comprising 55 s of maximal tethered swimming, and performed with a self-selected (BSS), every 4th arm-stroke (B4) and every 10th arm-stroke (B10) breathing frequency. The test duration was chosen as the closest ‘round’ time to the subject’s best 100 m front crawl time (Figure 6.1), and swimmers were instructed to swim the 55 s maximally as they would in a 100 m race. Breathing frequencies were chosen following a pilot study that showed no differences in performance (and therefore no limitation) following a breathing frequency of every 8 strokes.

Table 6.1 Subject physical characteristics and personal best (P.B.) times (n = 8; data are the mean \pm S.D.).

Subjects	Age (years)	Height (cm)	Weight (kg)	$\dot{V}O_2$ max (l min ⁻¹)	100 m F/C P.B. (s)
Mean	22	183.5	76.5	4.3	53.35
S.D.	\pm 3	\pm 5.5	\pm 6.8	\pm 0.3	\pm 1.18

P.B.: Personal best time; F/C: Front crawl

Expired air samples were collected throughout the main trials and during both the $\dot{V}O_2$ max and submaximal tests. Capillary blood lactate was measured in the three main trials 1, 3, 5, 10 and 15 min after the 55 s sprint. Accumulated oxygen deficit was determined according to Medbø et al., (1988) and detailed in both the methods (chapter III) and

section 5.2. Mean regression lines between swimming load and oxygen consumption measured during the submaximal test were $r = 0.990 \pm 0.011$; range: $r = 0.968 - 0.9996$. Expired air collection and analysis is detailed in chapter III, section 3.5.2, and the measurement of breathing frequency in section 3.5.2.1. Details of subject preparation, procedures, blood collection, handling and analysis can be found in chapter III.

Linear regression equations were applied to the submaximal data for calculation of accumulated oxygen deficit. One-way analysis of variance with repeated measures using the Scheffe f test for post hoc analysis was used to determine any differences between the CFB trial data. The accepted level of significance was set at 95% ($P < 0.05$). Results are presented as mean \pm S.D.

6.3 Results

6.3.1 Performance

No differences in performances (determined by peak force, mean force or fatigue index) were observed between trials during 55 s maximal tethered swimming. Mean \pm S.D. forces recorded are shown in Table 6.2 and the pattern of force output during the three trials are illustrated in Figure 6.1.

Table 6.2 Peak and mean force output and fatigue index recorded during the 3 CFB trials (mean \pm S.D., n = 8)

	BSS	B4	B10
Peak force output (N)	135.7 \pm 23.2	128.3 \pm 26.8	130.5 \pm 18.4
Mean force output (N)	101.3 \pm 13.1	92.3 \pm 11.6	93.6 \pm 11.1
Fatigue index (%)	45.7 \pm 11.4	47.0 \pm 10.1	46.8 \pm 9.8

N: Newtons; BSS: Breathing self-selected; B4: Breathing every 4 arm strokes; B10: Breathing every 10 arm strokes

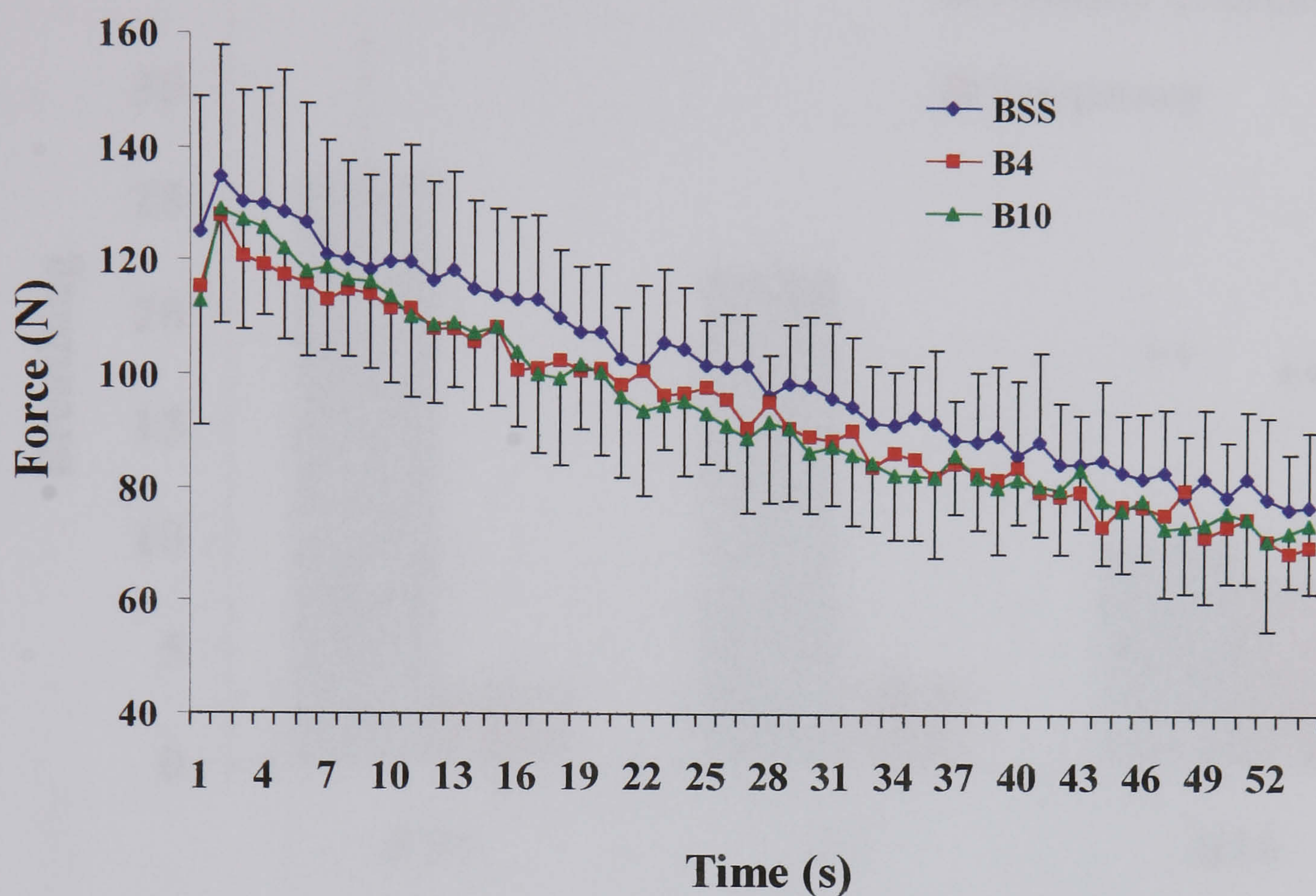


Figure 6.1 Force output during 55 s maximal tethered swimming using self-selected breathing (BSS), breathing every 4th (B4) and every 10th (B10) arm stroke.

6.3.2 Respiratory responses

There was no statistically significant difference between trials in stroke rate (BSS, 93 ± 7.3 ; B4, 89.2 ± 8.4 ; B10, 90 ± 8.3 strokes min^{-1}) and between breathing frequency in BSS and B4. Breathing frequency in B10 was significantly lower than both BSS and B4 ($P < 0.01$; Figure 6.1). Actual breathing frequencies recorded were 3.44 ± 0.82 , 3.67 ± 0.28 and 8.26 ± 1.07 strokes per breath for BSS, B4 and B10, respectively. Only one subject completed B10 as requested.

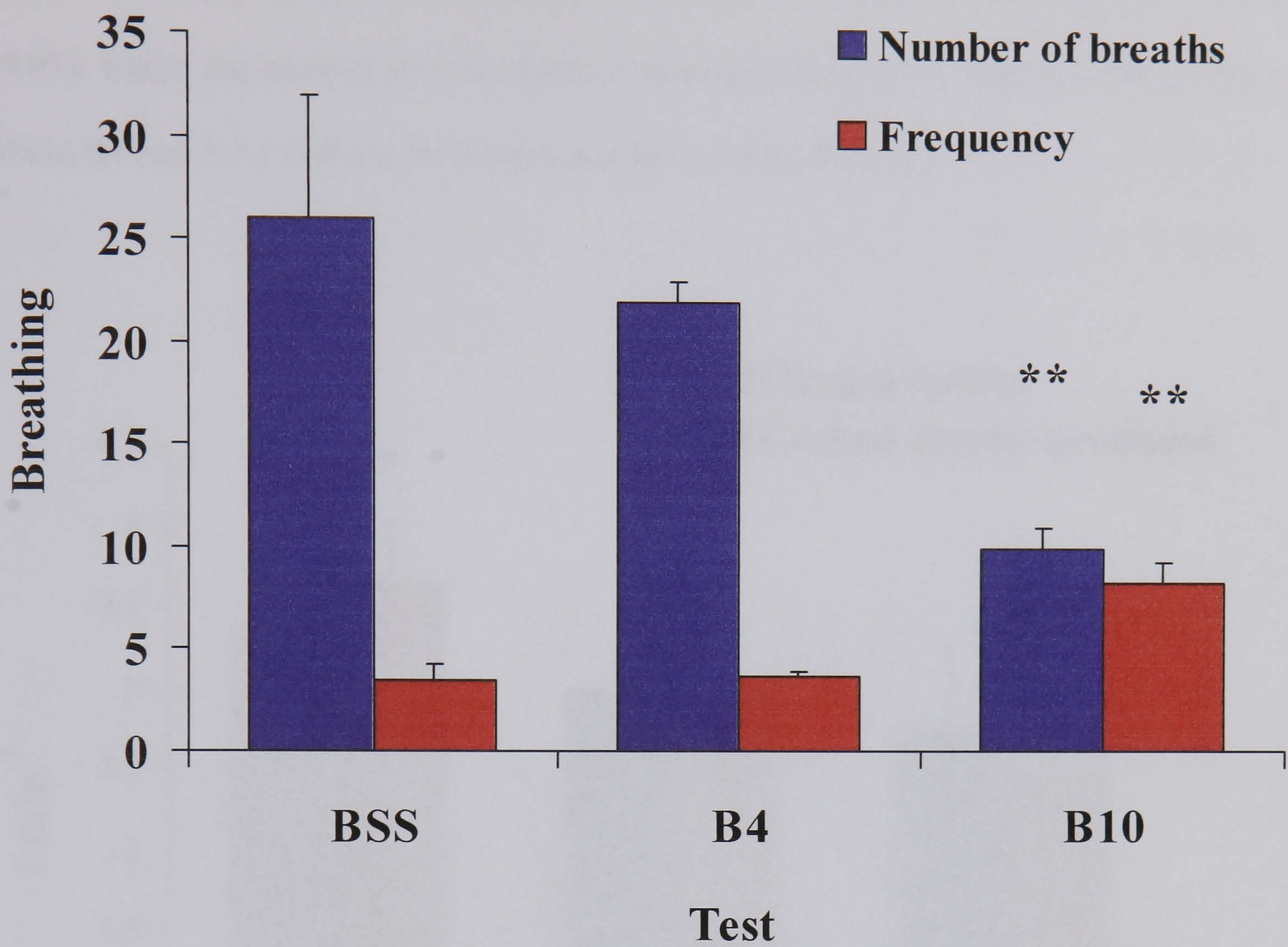


Figure 6.2 Number of breaths taken and frequency of breathing (strokes per breath) for the three trials (mean \pm S.D., n = 8)
**P<0.01, B10 vs. BSS and B4

Expired air \dot{V}_E was 85.6 ± 15.9 , 63.4 ± 13.9 and 44.3 ± 13.8 l min⁻¹ for BSS, B4 and B10 trials, respectively (BSS vs. B4, P<0.05; BSS vs. B10, P<0.01), representing a drop of ~26% between BSS and B4, ~30% between B4 and B10, and ~48% between BSS and B10. Oxygen uptake and $\dot{V}CO_2$ produced was 3.36 ± 0.28 and 3.65 ± 0.38 l min⁻¹ for BSS, 2.99 ± 0.18 and 2.96 ± 0.35 l min⁻¹ for B4, and 2.75 ± 0.55 and 2.48 ± 0.64 l min⁻¹ for B10 (Figure 6.3). Oxygen uptake was higher in BSS than B10 (P<0.05) and $\dot{V}CO_2$

higher in BSS and B4 than B10 ($P<0.05$). The fraction of O_2 in expired air dropped from $16.8 \pm 0.9\%$ in BSS to $16.0 \pm 1.0\%$ in B4 and $14.6 \pm 1.3\%$ in B10 (BSS and B4 vs. B10, $P<0.05$), whilst the expired air CO_2 fraction increased from $4.4 \pm 0.6\%$ in BSS to $4.9 \pm 0.6\%$ in B4 and $5.7 \pm 0.6\%$ in B10 (BSS and B4 vs. B10, $P<0.05$).

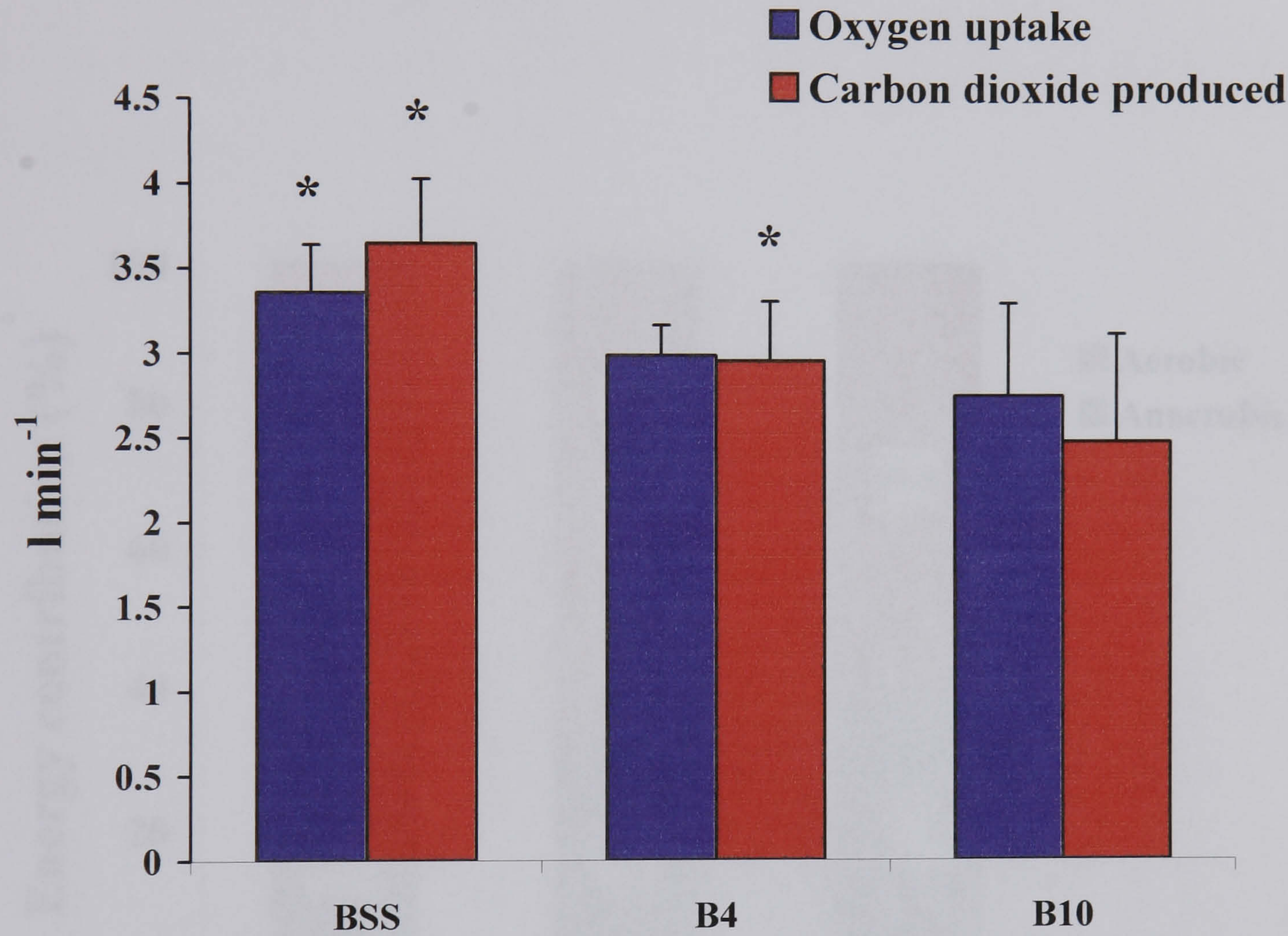


Figure 6.3 Oxygen uptake and carbon dioxide produced during the three trials (mean \pm S.D., $n = 8$). * $P<0.05$ vs. B10

Respiratory exchange ratios were 1.09 ± 0.13 , 0.99 ± 0.12 and 0.90 ± 0.13 (BSS vs. B10, $P<0.05$), whilst ventilatory equivalents ($\dot{V}E/\dot{V}O_2$) were 25.7 ± 5.4 , 21.1 ± 4.2 and 16.0 ± 3.4 for BSS, B4 and B10 respectively (BSS vs. B10, $P<0.05$).

6.2.3 Accumulated oxygen deficit

No differences were found in accumulated oxygen deficit values between trials (BSS, 20.4 ± 8.4 ; B4, 20.2 ± 8.8 ; B10, 23.8 ± 8.1 ml kg⁻¹ min⁻¹), and therefore estimated anaerobic energy contribution to the 55 s sprints was 30.9%, 32.8% and 39.6% for BSS, B4 and B10 respectively (Figure 6.4).

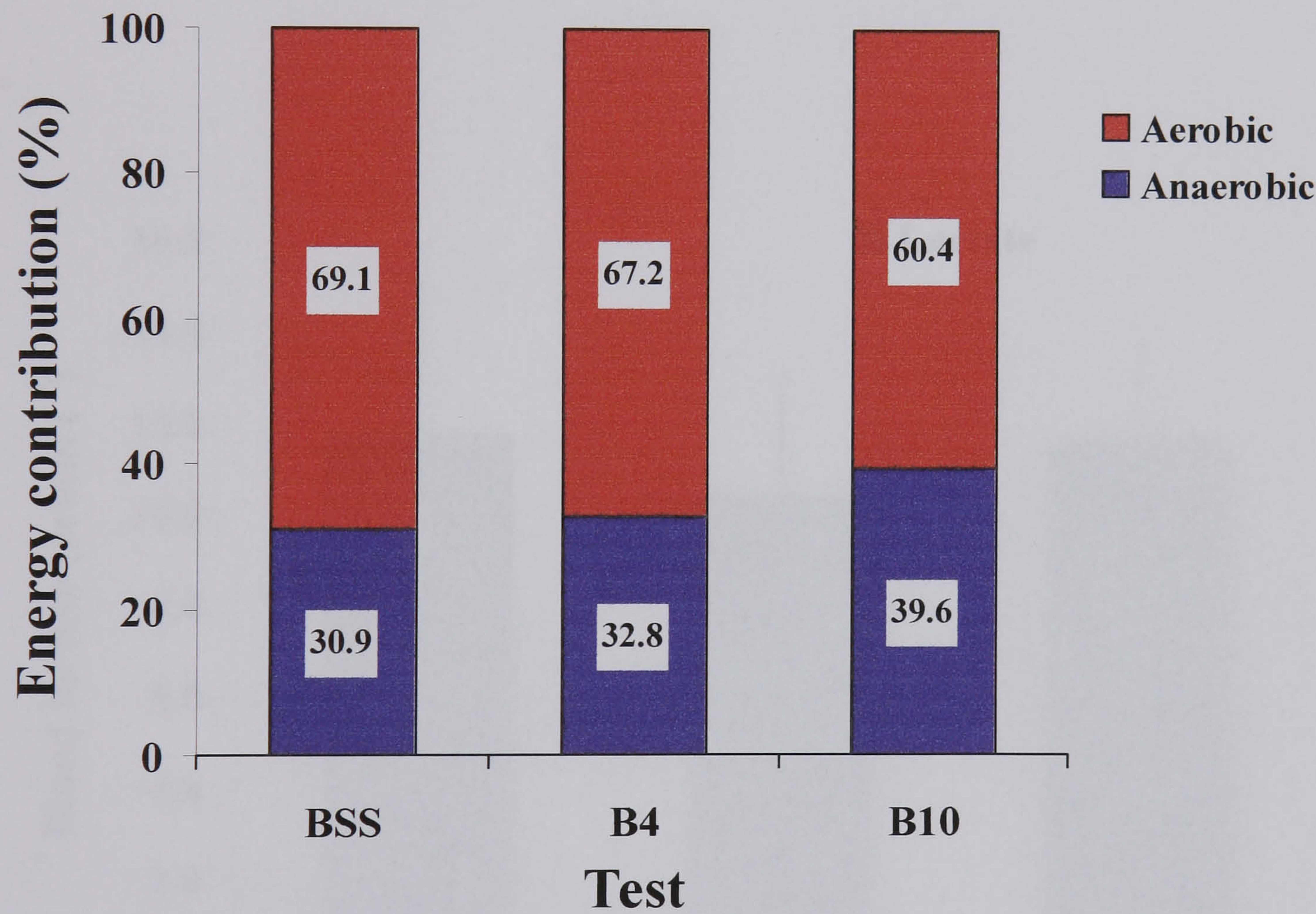


Figure 6.4 Estimated relative energy contribution from aerobic and anaerobic metabolism during the three trials (mean \pm S.D., n = 8; N.S.)

6.3.4 Lactate and heart rate responses

Peak blood lactate concentrations following 30 s maximal tethered swimming were 11.6 ± 3.5 , 10.3 ± 2.6 and 11.4 ± 2.4 mmol l⁻¹ for BSS, B4 and B10 trials respectively (N.S.; Figure 6.4). Peak blood lactate concentrations occurred at either 3 or 5 min after each trial, and were still raised to $\sim 7 - 9$ mmol l⁻¹ 15 min after the 55 s sprint. Mean peak heart rates were 168 ± 18 , 162 ± 11 and 164 ± 15 b min⁻¹ for BSS, B4 and B10 trials respectively (N.S.).

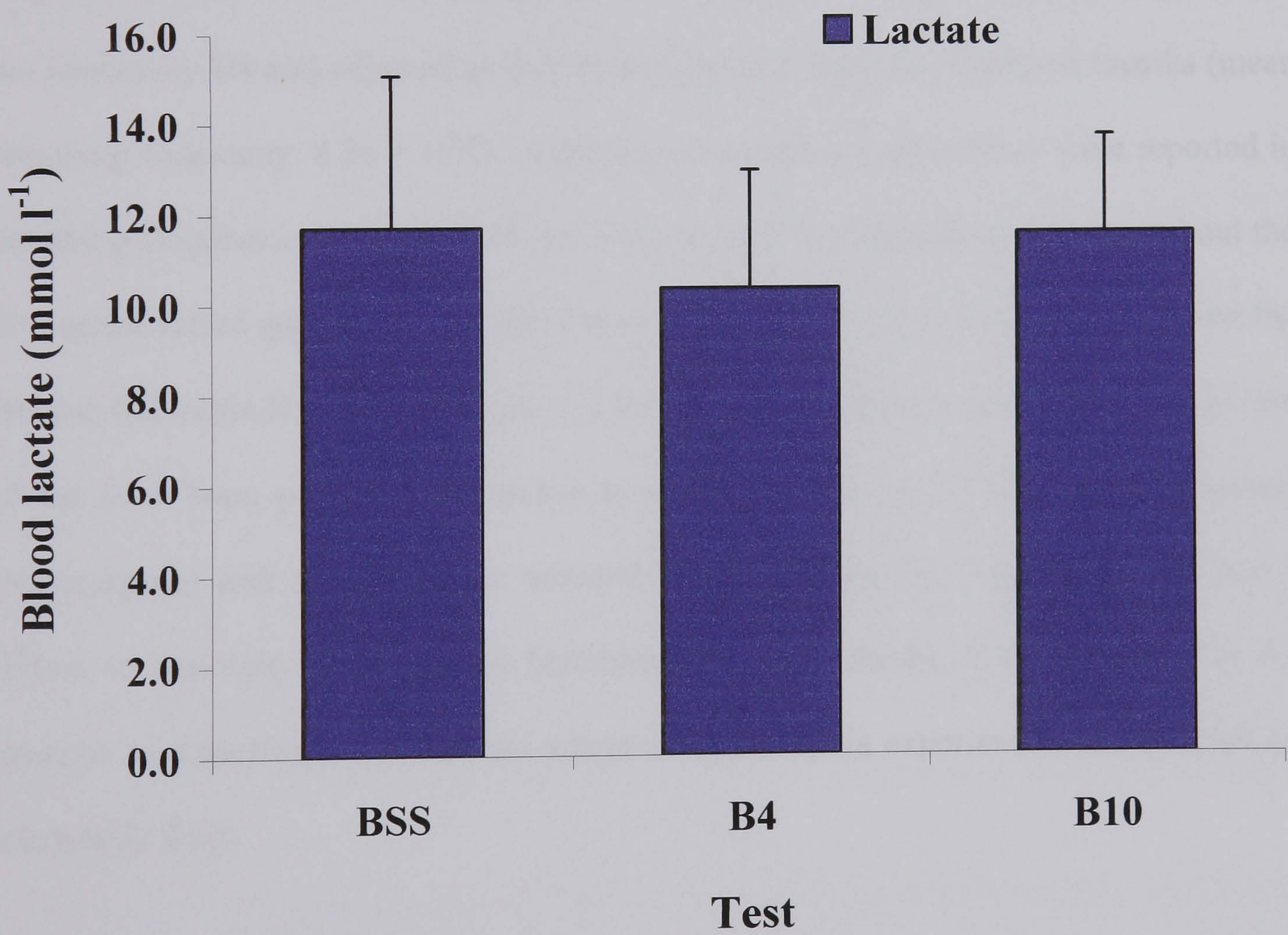


Figure 6.5 Peak blood lactate values during the three trials (mean ± S.D., n = 8; N.S.)

6.4 Discussion

This study is the first to our knowledge that has examined the physiological effects of controlled frequency breathing on maximal sprint swimming. The main finding was that performance was not affected by restricting breathing during sprinting, despite lower \dot{V}_E , $\dot{V}O_2$, $\dot{V}CO_2$ and RER, in the B10 trial. The peak blood lactate concentration and heart rate response to sprint swimming was unaffected by CFB.

A reduction in breathing frequency has been thought to increase stroke rate due to the increased desire to breath and therefore attempting to shorten the time between breaths by increasing the rate of stroking (Craig, 1979; Dicker et al., 1980). This was not shown in the present study even in maximal sprint swimming, although subjects perhaps were not limited by B4 and adjusted to B10 by taking more than the requested breaths (mean breathing frequency: 8.26 ± 1.07). Although no significant differences were reported in breathing frequencies between BSS and B4, the pattern of breathing rate throughout the 55 s sprint varied quite markedly. Breathing frequency was very constant throughout the B4 test, but in the BSS trial, it began at a low rate and increased rapidly towards the end of the 55 s bout, perhaps in response to a build up in venous CO_2 partial pressure (hypercapnia) and a progressive demand placed on aerobic metabolism. Subject's failure to maintain the requested breathing frequency during B10 suggests that the exercise was maximal, and that the extent of hypercapnia experienced had reached an intolerable level.

Evidence for the state of hypercapnia experienced by the swimmers is further provided by lower $\dot{V}CO_2$ and higher $FECO_2$ during B10, responses that have been shown to occur

in conjunction with increases in arterial CO₂ pressure (Dicker et al., 1980; Hsieh and Hermiston, 1983; Matheson and McKenzie, 1988; Stager et al., 1986; Yamamoto et al., 1988). The increase in CO₂ production (and consequently its concentration in the blood) is the main respiratory drive and signals the body to inhale. The extent of this condition was extreme as subjects were no longer able to maintain the required breathing frequency and acknowledged verbally on completion of each test. Despite the obvious discomfort suffered by the swimmers under these conditions, this distraction did not appear to affect performance. If swimmers are able to improve their tolerance to hypercapnia through training (Craig, 1979; Dicker et al., 1980), then it is possible that decreased breathing during sprint swimming may actually benefit performance by reducing the need to turn the head and therefore disrupt stroke mechanics (Nelson and Goldfuss, 1971).

The decrease in $\dot{V}E$ (B10 = ~50% of the BSS value) was not matched to the same degree by the reduction in $\dot{V}O_2$ (B10 ~18% lower than the BSS value). Thus, an increase in O₂ extraction occurred in B10 compared with that in BSS and B4, supported by the increased fraction of O₂ measured and the reduction in $\dot{V}E/\dot{V}O_2$ (Dicker et al., 1980; Town and Vaness, 1990). A greater tidal volume of air in addition to longer contact with the alveolar walls (to enhance O₂ uptake by blood) may compensate for the reduction in ventilation. Tidal volume increased by ~75% during B10 compared with BSS in attempt to maintain $\dot{V}O_2$ during hypoventilation. Lower oxygen uptake during B10 compared to BSS is expected, but does not match the drop in $\dot{V}E$. This has been previously observed in some submaximal swimming studies (Stager et al., 1986; Town and Vaness, 1990), but

not in others (similar O_2 values: Craig, 1979; Dicker et al., 1980). The submaximal exercise intensities used in these studies is likely to effect the reduction in $\dot{\text{V}}\text{O}_2$, as lower intensities will allow the body to adjust and meet similar O_2 demands by increasing extraction from limited $\dot{\text{V}}\text{E}$. The reduction in $\dot{\text{V}}\text{O}_2$ and lower $\dot{\text{V}}\text{E}/\dot{\text{V}}\text{O}_2$ (BSS, ~26 vs. B10, 16) in the present study with no difference in performance suggests that the swimmers were more economical during the B10 trial.

Lower RER values during B10 compared with BSS (0.90 vs. 1.09) suggests a decrease in glycolysis and has been shown to be related to the onset of hypercapnia, a condition associated with decreased glucose utilisation and glycolytic inhibition in red blood cells (Graham et al., 1980). A reduction in RER in conjunction with an increase in CO_2 expired air fraction and increases in inspired air O_2 fraction have been consistently reported in submaximal swimming studies (Dicker et al., 1980; Holmer and Gullstrand, 1980; Town and Vaness, 1990).

No differences were found in accumulated oxygen deficit and estimated energy contribution between trials, although the estimated anaerobic contribution during B10 rose to 39.6% (from 30.9% in the BSS trial), perhaps reflective of the restricted oxygen supply. Large inter-individual differences, perhaps reflecting the mixed range of sprint and middle-distance swimmers used in the subject group, masked any possible effect between trials (sprinters have been found to exhibit 30% higher MAOD than endurance performers; Medbø and Burgers, 1990). The values for AOD and therefore estimated anaerobic energy contribution are lower than those for sprint cycling and running determined by this method. Ranges in aerobic contribution to ~60 s sprinting are

between 37% for running (Olesen et al., 1994) and 54% for cycling (Craig et al., 1995), although most studies report values between 45 and 50% (Gastin and Lawson, 1994; Hermansen and Medbø, 1984; O'Brien et al., 1997; Withers et al., 1991; Withers et al., 1993).

The few studies that have considered swimming also suggest a lower aerobic contribution to that found in the present study. Troup and Trappe (1994) suggest a 47% aerobic contribution for a 100 m race from experiments carried out in a swimming flume, similar to that of both Morton (1992) and Morton and Gastin (1997) who report values of 44-50% and 46% respectively during 60 s swim bench sprinting. It is possible that in the present study, excess breathing occurred in response to the unusual head position (face constantly in the water). Some swimmers reported a feeling of anxiety with restriction of breathing, which may have resulted in hyperventilation during BSS and B4. Both the tether mechanism and the breathing apparatus may have altered stroke mechanics by restricting maximal arm stroking. If an element of pacing was included in the sprints, then it is likely that a smaller anaerobic energy contribution, and larger aerobic contribution was made to the sprint. This mechanism has been reported in cycling studies using more constant rate pedal speeds with the same mean power performed as that in an all-out sprint strategy (Beleen and Sargeant, 1994; Cherry et al. 1998). Support for this is provided by the lower peak force and more gradual decline in force output compared with the 30 s sprint used in chapter V (see Figures 6.1 and 5.3).

The relative intensity of 55 s performance was not as high as that suggested by Troup and Daniels (1986) and Troup et al. (1992b) for 100 m race performance. They suggest

that the intensity during 100 m race swimming is equivalent to $\sim 140\%$ $\dot{V}O_{2\max}$ (Troup and Daniels, 1986; Troup et al., 1992b) which is higher than the relative intensity estimated in the present study (mean force equivalent to $\sim 113\%$, $\sim 105\%$ and $\sim 104\%$ of $\dot{V}O_{2\max}$ for BSS, B4 and B10 trials respectively). The lower than expected intensity supports the higher aerobic contribution observed in this study during 55 s tethered swimming. It is possible that in competition, motivation will be higher than that experienced during the study, and certainly the awareness of the race duration is possible (from the position in the pool), whereas during stationary tethered swimming, subjects are unaware of the time completed at points throughout the 55 s. It is also likely that the tethered swimming mechanism places an unnatural restriction on swimmers, and they do not get the 'boost' from a dive start experienced in competitive races, or the localised muscle resting during the turning action, and may account for some of the differences observed in the present study.

Previous studies have reported either an increase in blood lactate due to increased reliance on anaerobic glycolysis (Matheson and McKenzie, 1986; Yamamoto et al., 1988) or decrease due to an inhibition of glycolysis or a delayed lactate release from the muscle (Graham et al., 1980; Holmer and Gullstrand, 1980) following CFB. Blood lactate concentrations were not different between trials in the present study, perhaps because muscle hypoxia did not occur and that values reflect the similar 55 s performances and energy supply (Town and Vaness, et al., 1990).

In front crawl swimming, unlike most other modes of exercise, the rate (and to a certain extent depth) of breathing is restricted to the position of the face in the water. Breathing

rate will be dictated by the duration of each stroke cycle and the regularity of the body and head in turning laterally to obtain an inhalation. It is these factors that require swimmers to hold their breath, even for short periods within the stroke cycle, although this is unlikely to influence metabolism or performance (Craig, 1986). Restriction of breathing may impede stroke mechanics if swimmers are not familiar with such practices (Craig, 1979; Town and Vaness, 1990), and the impact of head turning on performance or stroke mechanics under fatigue conditions has not been studied (Payton et al., 1999).

The main finding of the present study was that despite a significant reduction in breathing frequency and differences in respiratory metabolism, changes had no effect on 55 s maximal tethered swimming performance. Thus, the hypothesis that CFB would compromise tethered swimming performance was rejected. The impact of the breathing action under fatigue conditions during sprint swimming may compromise stroke mechanics and therefore performance. A reduction in breathing frequency may help to avoid this to some extent, although swimmers should employ restricted breathing during sprint training in order to familiarize themselves with this practice and perhaps learn to cope better with the effects of hypercapnia.

CHAPTER VII

THE EFFECTS OF ACTIVE AND PASSIVE RECOVERY ON PERFORMANCE DURING REPEATED SPRINT SWIMMING

7.1 Introduction

One of the most common practices during the training of swimmers is to use sets of repeated sprints. The aim of the swimmer in such training sets is to maintain the highest possible speed in each repetition (Maglischo, 1993), but this is not usually attainable in repeated sprints due to the accumulating effects of fatigue (Balsom et al., 1993a; Hermansen and Osnes, 1972). The inability of the swimmer to maintain maximal speed in each repetition is associated with fatigue and is commonly related to high muscle lactate and hydrogen ion (H^+) concentrations and low phosphocreatine (PCr; Cheetham et al., 1986; Bogdanis et al., 1995). One method used to accelerate recovery between repeated bouts of sprint exercise is active recovery. The aim is to increase muscle blood flow, which will in turn increase the rate of PCr resynthesis and lactate/ H^+ removal.

A faster decrease in blood lactate concentration after maximal swimming exercise has been shown in active compared with passive recovery (Cazorla et al., 1983; McMaster et al., 1989; Reaburn and Mackinnon, 1990). Specifically, Cazorla et al. (1983) have demonstrated this response using active recovery at 60 - 65% of 100 m pace (the

speed equivalent to individual best time) corresponding to a swimming intensity of 55 - 73% of $\dot{V}O_2\text{max}$ in comparison with passive recovery.

Cycling studies have also shown that performance during repeated sprints was enhanced after active compared with passive recovery (Signorile et al., 1993; Bogdanis et al., 1996c; Ahmaidi et al., 1996). The majority of these studies have however concentrated on performance of short duration sprints (6 s: Ahmaidi et al., 1996; Signorile et al., 1993), only two maximal bouts (Ainsworth et al., 1993; Bogdanis et al., 1996b), long recovery duration (10-20 min: Bangsbo et al., 1993; Weltman et al., 1977; Weltman et al., 1979) or longer duration, lower intensity repeats (Thiriet et al., 1993).

Several swimming studies have focused on the post-exercise decrease in blood lactate concentration as a measure of recovery (Beckett and Steigbigel 1983; Cazorla et al., 1983; McMaster et al., 1989; Reaburn and Mackinnon, 1990), although subsequent performance was not measured. The limited number of studies which have considered recovery modes and subsequent performance in swimming have concentrated on 200 yards race performance and once more, longer recovery durations (14/15 min: Felix et al., 1997; McMurray, 1969; Siebers and McMurray, 1981).

There are no studies that have examined the effect of active recovery during repeated sprint swimming by employing protocols that simulate swimming training practices, particularly sprint distances (i.e. 50 yards/metres), with repeated bouts and sets of bouts with relatively short duration recovery times (\sim 1 min 30 s to 5 min). Short

recovery (3 – 5 min) between sprints allows the role of H^+ and PCr to be examined as the recovery of PCr and pH in the blood and the muscle occur at different rates. The purpose of this study therefore was to examine the effects of active and passive recovery during and between sets of repeated sprint swimming bouts, similar to those commonly used during swimming training. It was hypothesised that performance times would be faster when active recovery was performed between repetitions and between sets of repetitions in sprint training due to a faster PCr resynthesis and restoration of pH.

7.2 Methods

Eight elite, male competitive swimmers (subject characteristics – see Chapter 5, Table 5.1) performed a series of tests using both a tethered swimming mechanism (see Figures 3.1 and 3.2) and repeated, free swimming bouts (see section 3.4.1.1, final paragraph). Two initial tests were performed: a $\dot{V}O_{2\max}$ STS test (section 3.4.2.3) and a maximal 30 s FTS sprint (section 3.4.2.5). Swimmers then conducted three randomly assigned main trials, separated by 3-7 days and performed at the same time of the day (± 1 h). Front Crawl was used in all tests; during which expired air samples and stroke rate data were collected.

On completion of the warm-up, a swimmer was attached to the belt, dried, kept warm and rested on a chair at the side of the pool. Tests began 15 min after the end of the warm-up. The first section (Set A) consisted of 4 x 30 s bouts of semi-tethered swimming separated with 30 s of passive rest. The actual force on the rope was measured by the force transducer (Figure 3.1 and 3.2) and was used for the analysis of

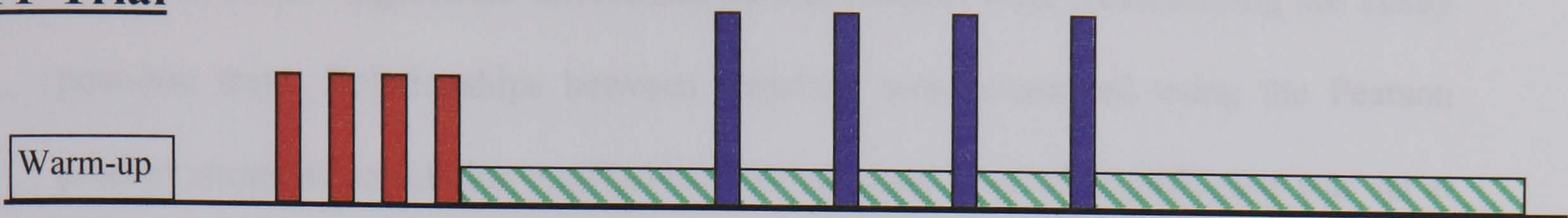
the results. During all three conditions, the swimming intensity of Set A was kept at a similar level by setting the applied load the same across trials. The load applied on the basket during Set A was equal to 95% of the individual mean force attained during 30 s of the tethered swimming sprint. This provided a standardised volume and intensity of fatiguing work performed prior to each of the three trials, similar to that experienced during swimming training.

The second section of the test (Set B) consisted of 4 x 50 yards of maximal Front Crawl swimming at intervals of 2 min (exercise time plus recovery time = 2 min) and each beginning from a push start from within the pool. The two sections (Set A and B) were separated by 5 min of recovery. On one occasion, recovery between Sets A and B and during the repetitions of Set B was passive, with swimmers standing in the water at the shallow end of the pool (PP Trial). On a second occasion, recovery between Sets A and B was 1 min passive, 3 min active and 1 min passive (Figure 7.1), but during Set B was passive (AP Trial). On a third occasion, recovery between sets was active as in the AP trial, and recovery between the repetitions of Set B was also active (AA Trial). During the AP and AA trials, 1 min passive, 3 min active and 1 min passive enabled capillary blood collection post-set A and pre-set B, and allowed swimmers to prepare for the next section.

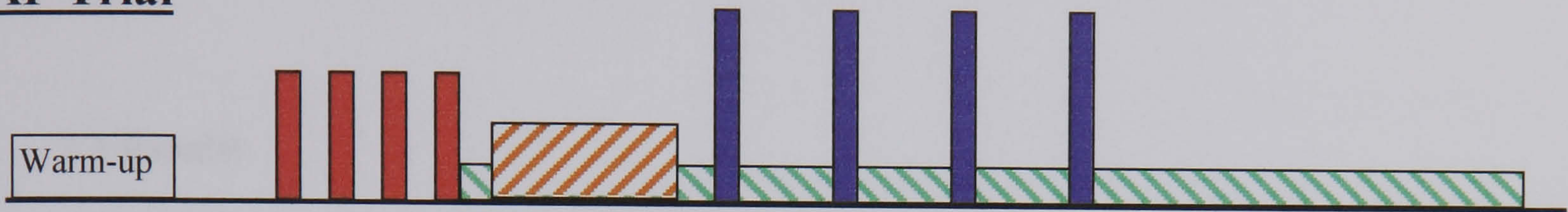
Swimmers in the AA trial began active recovery within 5 s of each 50 yards repetition and stopped swimming 15 s before the start of the next repetition. Recovery on completion of Set B was always passive. Active recovery pace on all occasions was 60% of the average speed calculated from the individual's best time for 100 m front crawl. Swimmers were comfortably able to adhere to the prescribed pace.

The study design, measurements and timing of the test protocol are shown in Figure 7.1. Capillary blood samples were taken 3 min before the start of each main trial, 30 s after Set A, 30 s before the start of Set B and 1, 3, 5 and 8 min after the end of Set B. Blood samples were taken simultaneously from thumbs of both hands for analysis of both capillary blood lactate and pH. On completion of Set A, swimmers approached the shallow end of the pool where they could stand and be detached from the belt within 15 s. Further details of the subject preparation, procedures, blood and expired air collection, handling and analysis can be found in chapter III.

PP Trial



AP Trial



AA Trial

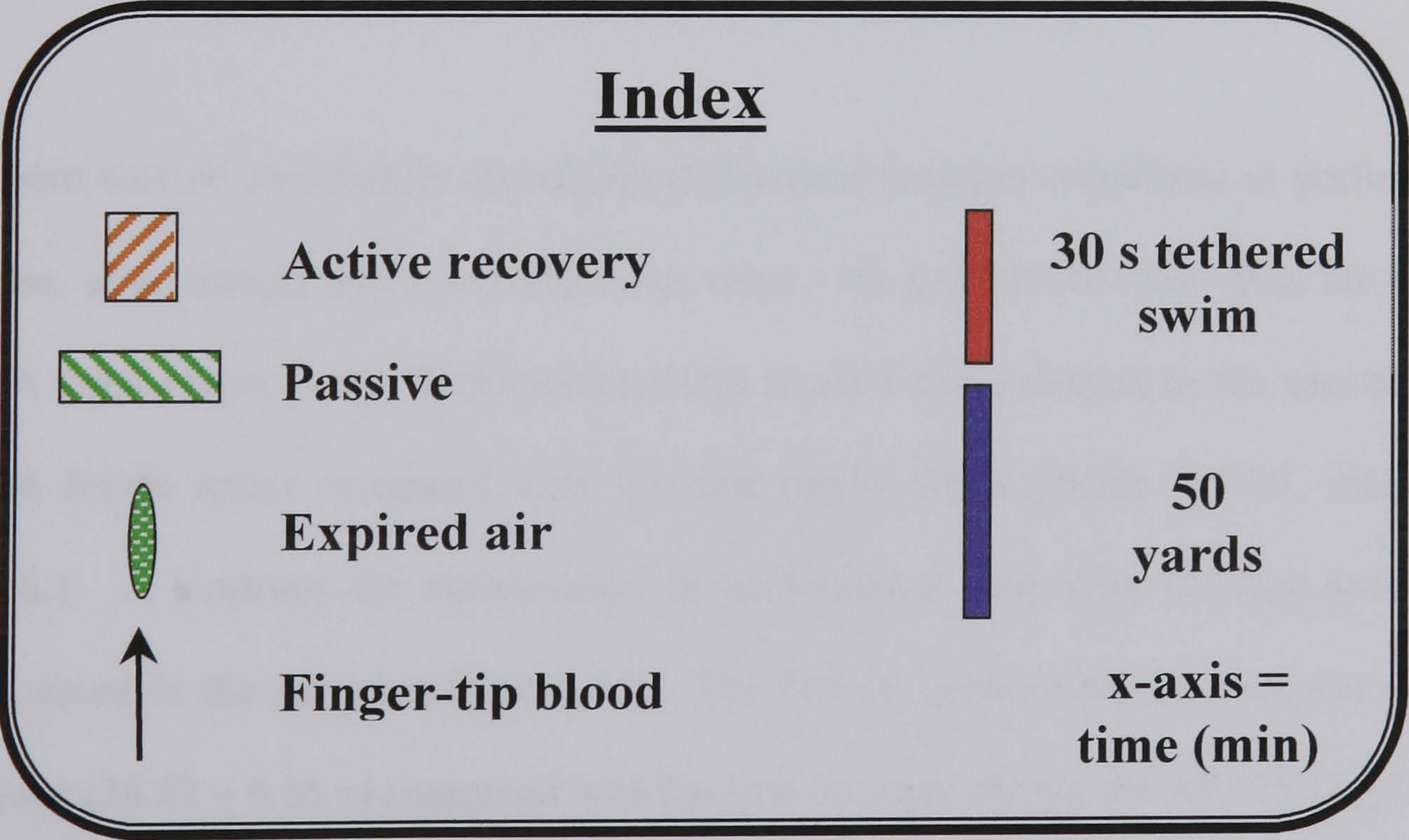
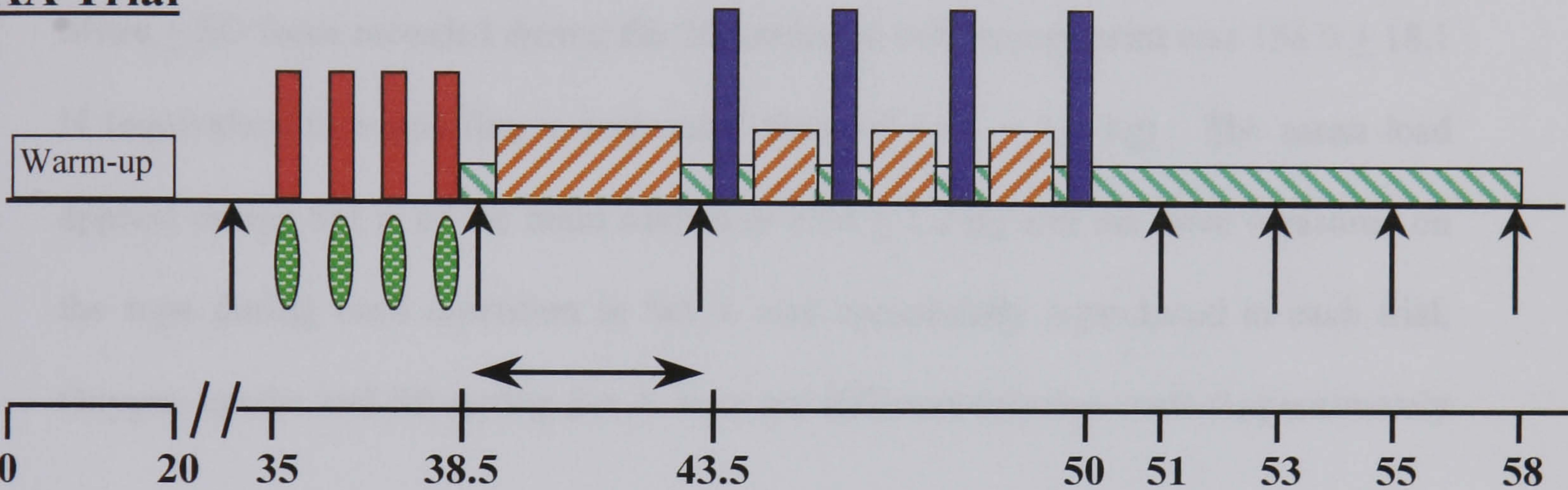


Figure 7.1 Schematic illustration of the 3 experimental protocols used in the study.

A two-way analysis of variance (trials x time) was used to examine differences between means. Significant differences between means were located using the Tukey post-hoc test. Relationships between variables were examined using the Pearson product moment correlation coefficient. The accepted level of significance was set at 95% ($P < 0.05$). Results are presented as mean \pm S.D.

7.3 Results

7.3.1 Performance

Mean \pm SD force recorded during the 30 s tethered swimming sprint was 138.6 ± 18.1 N (equivalent to supporting a suspended mass of 14.1 ± 1.2 kg). The mean load applied during Set A of the main trials was 13.4 ± 1.2 kg and the force measured on the rope during each repetition in Set A was successfully reproduced in each trial. Oxygen uptake and SR during Set A were not different between trials (approximately 70 - 75% of $\dot{V}O_2$ max and 86 strokes min^{-1} respectively). The load corresponded to ~156% of the load attained on the $\dot{V}O_2$ max test.

There was no statistically significant differences between conditions in performance time, even though mean AP trial times were ~1% and ~2.5% faster than the PP and AA trials respectively. Performance times in all 3 trials slowed in the second, third and fourth sprint compared with the first (main effect sprints $P < 0.01$, interaction N.S.). A tendency for maintenance in performance time after the first sprint was observed in the AP trials (Figure 7.2). The first 50 yards in the PP trial was slightly faster (24.82 ± 0.35 s) compared with the first 50 yards during the AP (25.11 ± 0.46 s) and AA (25.27 ± 0.40 s), (N.S.). During the remaining three sprints, performance in

the AP trial (25.22 ± 0.36 , 25.47 ± 0.42 , 25.50 ± 0.30 s) was consistently better than the PP (25.65 ± 0.36 , 25.96 ± 0.33 , 25.89 ± 0.29 s) and AA (26.23 ± 0.43 , 26.18 ± 0.37 , 26.12 ± 0.44 s) trials (interaction N.S.).

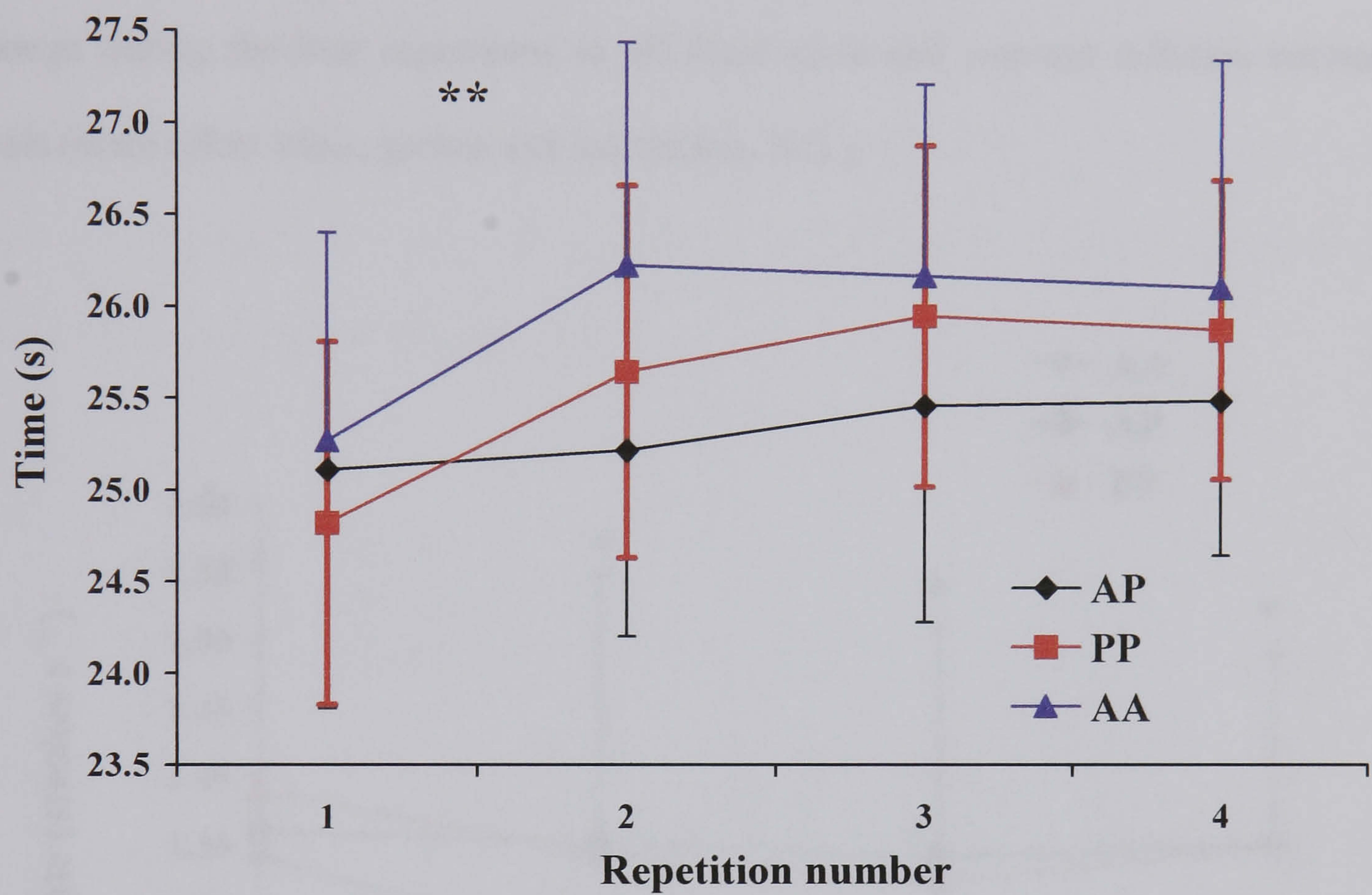


Figure 7.2 Performance times during the 4 x 50 yards sprints (n = 8, mean \pm S.D.). ** P<0.01 denotes difference from the first sprint.

Similar patterns were seen when performance time was analysed for the first or second 25 yards split (main effect trials $P>0.05$, main effect repetitions $P<0.01$, interaction N.S.). Fatigue index (FI) during Set B, expressed as the percentage time difference between first 25 yards of first sprint and second 25 yards of the fourth sprint was lower in the AP trial although not statistically significant (PP $9.04 \pm 1.36\%$, AP $6.31 \pm 1.22\%$, AA $8.02 \pm 1.87\%$; N.S.).

7.3.2 Stroke rate and Stroke length

Mean SR was lower in the AA trial compared with the AP and PP trials (main effect trials $P<0.05$). Stroke rate also declined during the second, third and fourth sprint compared with the first (main effect sprints $P<0.05$, interaction N.S.). A tendency for maintenance of SR was observed in the AP trials (Figure 7.3). Mean SL did not change during the four repetitions in all three trials and was not different between trials (main effect trials, sprints and interaction, N.S.).

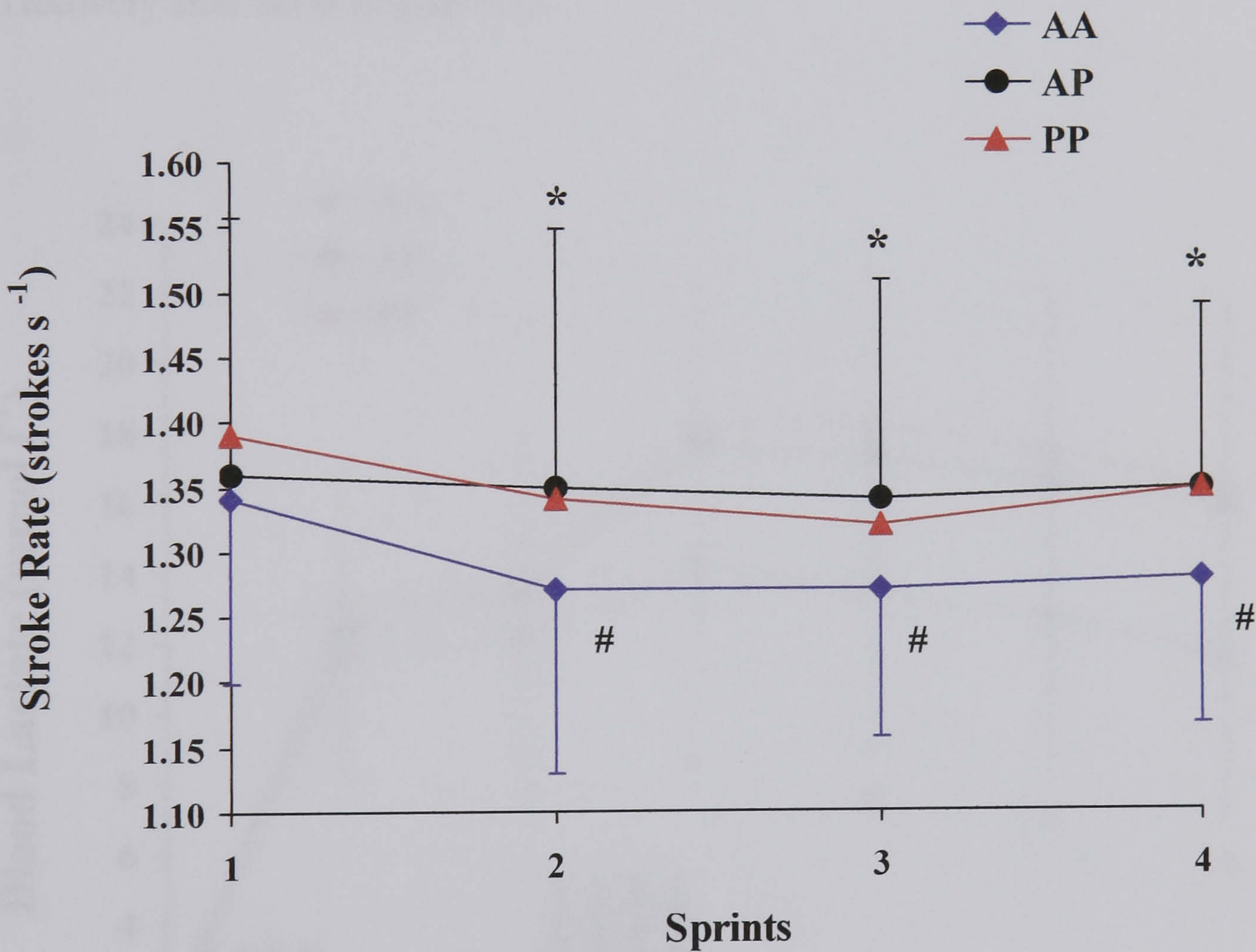


Figure 7.3 Stroke Rates during the 4 x 50 yards sprints (n = 8, mean \pm S.D.).
* $P<0.05$ denotes difference from first sprint. # $P<0.05$ AA vs. AP and PP trials.

7.3.3 Metabolic responses

Blood lactate concentrations 30 s after completion of Set A were 13.0 ± 1.1 , 12.4 ± 1.2 , 11.7 ± 1.2 mmol l⁻¹ for PP, AP, AA trials respectively (N.S.). Values remained at similar levels until the end of the 5 min recovery period (Figure 7.4). Peak blood lactate values measured occurred one min after Set B in all three trials. The highest peak blood lactate concentration was observed in the AP and PP trials (17.6 ± 1.2 and 18.3 ± 1.3 mmol l⁻¹ respectively) while peak blood lactate was lower in the AA trial (14.1 ± 1.6 mmol l⁻¹, $P < 0.01$). These blood lactate differences were still evident 8 min into recovery after Set B (Figure 7.4).

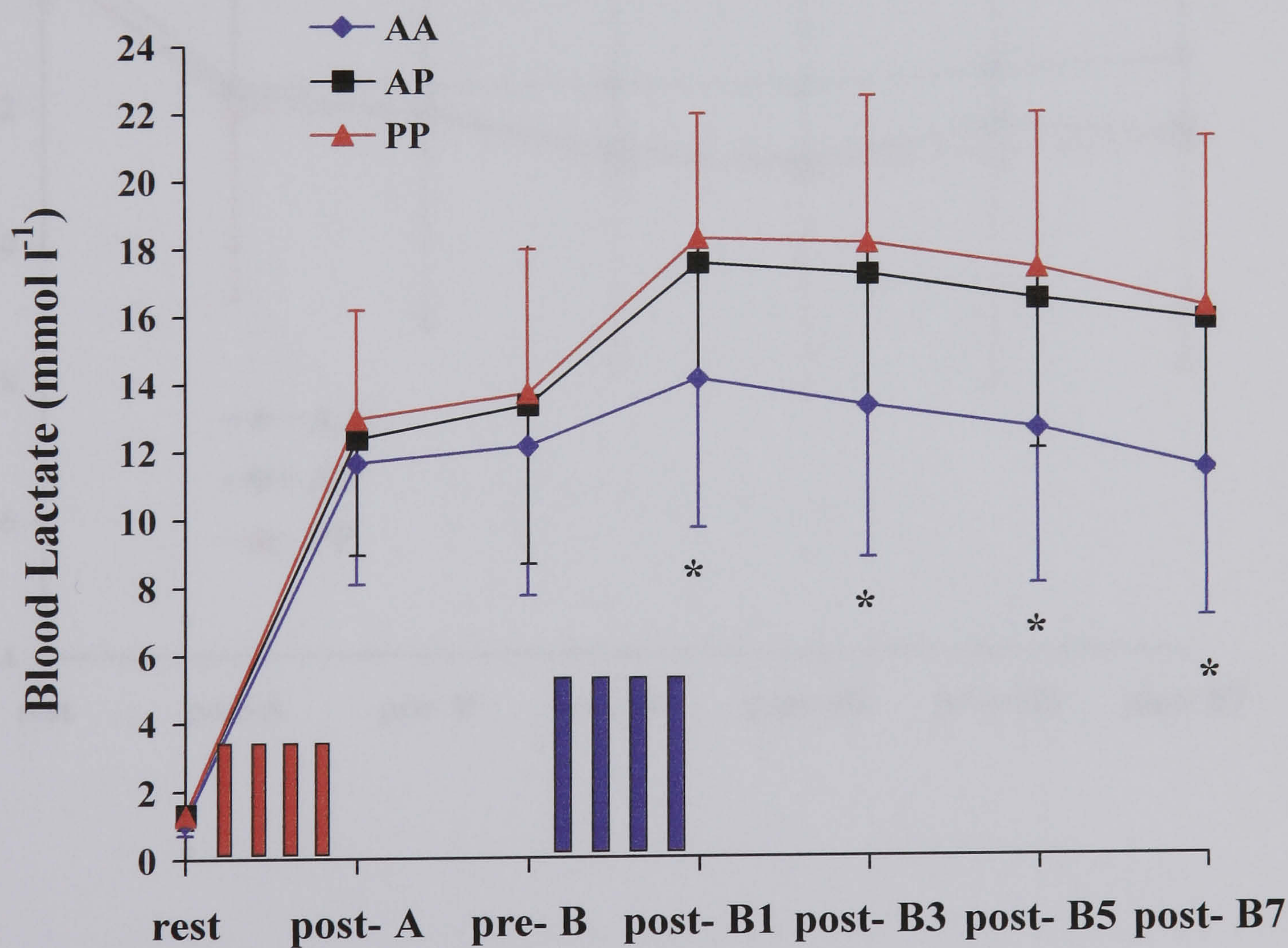


Figure 7.4 Blood lactate values during trials (n = 8, mean \pm S.D.).
* $P < 0.05$ AA vs. AP and PP trials.

Decline in blood pH values closely followed the increases observed in blood lactate (Figure 7.5). Lowest blood pH was observed 3 min after Set B both in AP and AA trials (7.11 ± 0.11). Strong negative correlations were found between blood lactate concentration and blood pH, during all trials ($r = -0.91$ to -0.94 , $P < 0.01$).

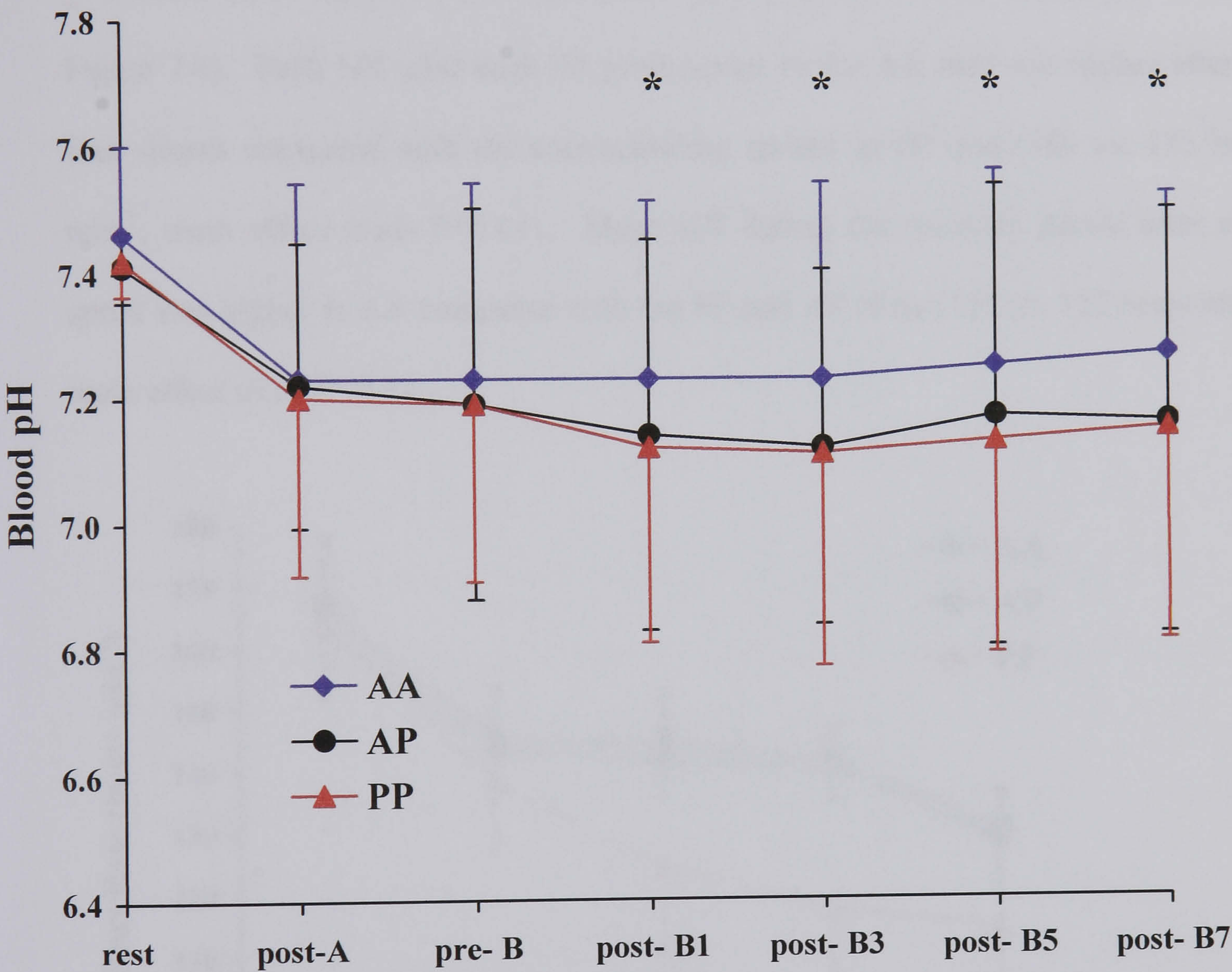


Figure 7.5 Blood pH values during the trials ($n = 8$, mean \pm S.D.).
* $P < 0.05$ AA vs. AP and PP trials.

7.3.4 Heart rate responses

The heart rate (HR) response was similar during Set A and during the first and second min of the recovery period between Set A and B between trials (main effect trials, N.S.). In the third, fourth and fifth min of recovery (between Set A and B), HR in the AP (144 ± 3 , 141 ± 2 , 130 ± 2 beats min^{-1} respectively) and AA (141 ± 2 , 140 ± 2 , 128 ± 3 beats min^{-1} respectively) trials was higher than the PP (124 ± 5 , 117 ± 4 , 114 ± 4 beats min^{-1} respectively) trial (main effect trials $P < 0.01$, interaction $P < 0.01$; Figure 7.6). Peak HR after each 50 yards sprint in the AA trial was higher after all four sprints compared with the corresponding sprints in PP trial (180 vs. 175 beats min^{-1} , main effect trials $P < 0.01$). Mean HR during the recovery period after each sprint was higher in AA compared with the PP and AP trials (165 vs. 152 beats min^{-1} , main effect trials $P < 0.01$).

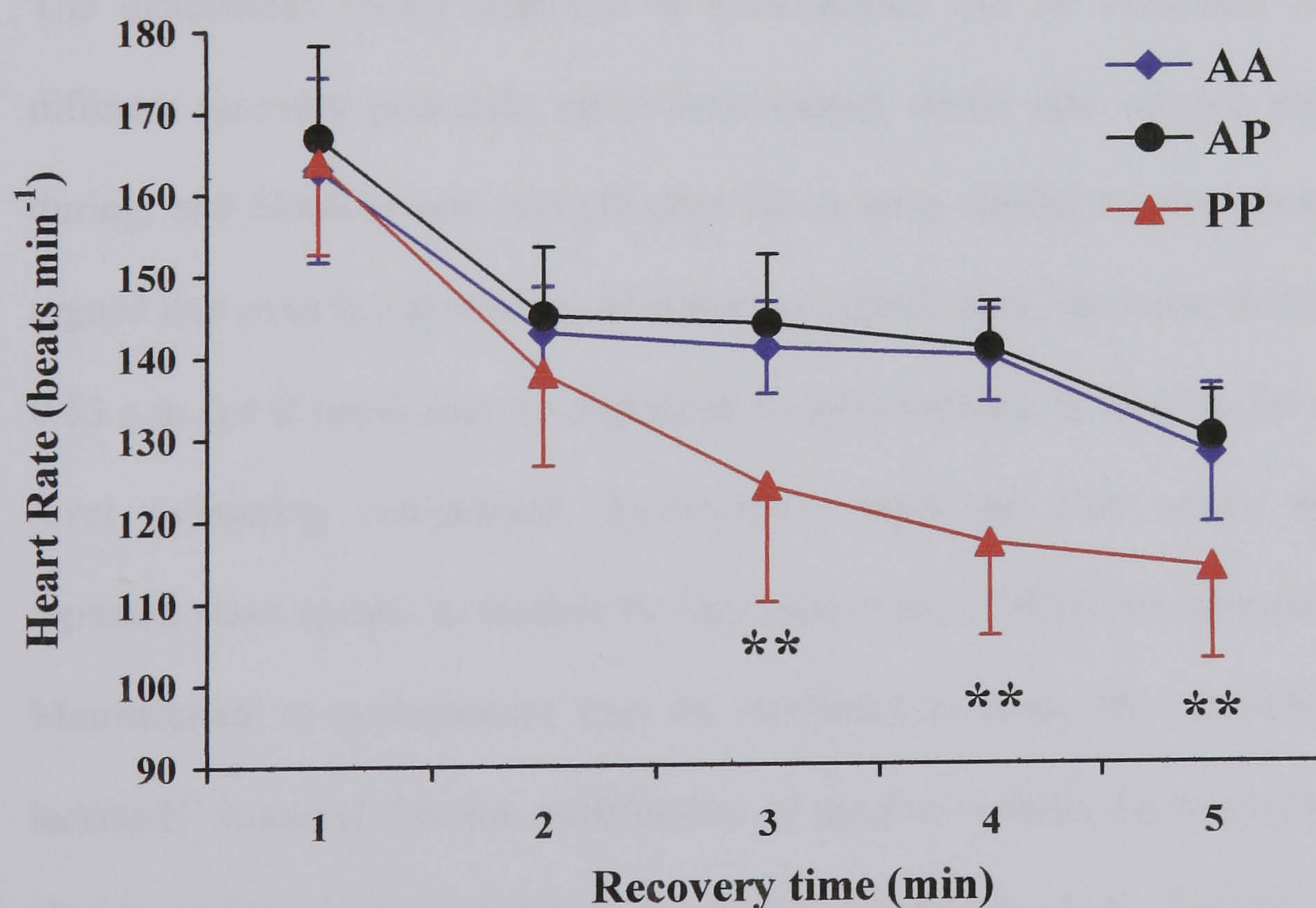


Figure 7.6 Heart rate between Set A and B during the trials ($n = 8$, mean \pm S.D.).

** $P < 0.01$ PP vs. AP and AA trials. Differences over time are not shown for clarity.

7.4 Discussion

The main finding of this study was that performance during Set B was similar in all three conditions, with if anything a tendency for poorer performance in the AA trial shown by a lower stroke rate. It was expected that active recovery between sets and the repetitions of Set B would enhance muscle blood flow, resulting in a faster PCr resynthesis, lactate removal and restoration of acid-base balance. This in turn would result in improved sprint times during Set B. In fact, performances in the AP trial (active recovery performed between Sets A and B, but passive between the repetitions of Set B) showed the fastest times over the 4 x 50 yards sprints. Mean performance times in the AP trial were ~1% and ~2.5% faster (N.S.) than the PP and AA trials respectively, and declined less over the four repetitions (0.39 s) in the AP trial, compared with performance in the PP (1.07 s) and AA (0.85 s) trials.

The differences (N.S.) observed in performance can be attributed to the effect of different recovery protocols, since force output, stroke rate, oxygen uptake, heart rate during, and blood lactate and pH after Set A were similar between trials. It could be argued that even in the absence of statistical significance, the mean differences of 0.26 - 0.63 s in Set B times may be important for performance in training for success in high level swimming competition. Performance improved after active recovery during repeated short sprints in studies by Signorile et al., (1993) and Ahmaidi et al., (1996). Maintenance in performance may be attributed to faster PCr resynthesis or muscle lactate/H⁺ removal, but the contribution of aerobic metabolism to energy supply could also have been increased during the sprints (Bogdanis et al., 1996a). Therefore any change in anaerobic (PCr, glycolysis) or aerobic contribution to energy supply because of the active recovery could have affected performance.

It is likely that following Set A (4 x 30 s at $\sim 156\%$ $\dot{V}O_2$ max), PCr stores would have been significantly depleted. Bogdanis et al., (1995) found an average PCr resynthesis half-time of ~ 57 s following a maximal 30s cycle sprint. Indeed, PCr concentration had increased to 65% of the rest value by 1 min 30 s and $\sim 86\%$ by 6 min following passive rest. Ainsworth et al., (1993) observed that at least 9 min of recovery was necessary to restore power production after maximal sprint cycling. Incomplete PCr resynthesis prior to Set B would have placed additional demand on energy supply from anaerobic glycolysis and aerobic metabolism. As the rest intervals became shorter in Set B, this effect would have become more apparent. Bogdanis et al., (1996a) estimated an aerobic energy contribution of $\sim 44\%$ in a second 30 s sprint, whilst Trump et al., (1996) found that 70% of the total energy contribution was provided by aerobic metabolism by the fourth 30 s cycle sprint. In chapter V, aerobic energy contribution to 4 x 30 s tethered swimming sprints was $\sim 26\%$, $\sim 47\%$, $\sim 49\%$ and $\sim 53\%$ for repetitions one to four respectively.

A significant reduction in stroke rate (SR) may explain the decline in performance in the AA trial. An inability to maintain a high SR during swimming races may be a consequence of fatigue (Craig and Pendergast, 1979; Wilke, 1992). In studies that used cycling as a mode of exercise, increased pedal speed was one of the factors associated with better performance after active recovery (Bogdanis et al., 1996c; Weltman et al., 1977). A decreased SR in swimming may be attributed to a slowing of the muscle relaxation rate. This may be related to an increased H^+ and its effects on the Ca^{2+} and myosin ATPase (Cady et al., 1989a) or to impaired function of sarcoplasmic reticulum (Allen et al., 1992). The slowing of the muscle relaxation rate may be greater in fast

twitch fibres which are used predominantly in fast muscle actions and are more rapidly depleted of PCr and glycogen (Greenhaff et al., 1994c). The ability to perform at high contraction and relaxation rates is probably affected by the energy status of the cell (Allen et al., 1992).

At the end of active recovery between sets the heart rate was higher in the AP and AA trials compared with the PP trial. This indicates an increased metabolic rate and a higher aerobic metabolism due to the previously performed active recovery (aerobic exercise). Recently, Bogdanis et al., (1996b) observed an increase in oxygen uptake in a second 30 s sprint after active compared with passive recovery. Aerobic contribution may have increased in the AA trial, because of the active recovery performed between repetitions, but this was not reflected in the performance times, which tended to be slower than in the AP trial. Despite lower blood lactate concentrations in the AA trial, Set B performances were not improved.

An increase in the contribution of energy from aerobic sources may have a negative impact on performance if it is provided at the cost of energy supply from anaerobic glycolysis. It is possible that in the AA trial, aerobic metabolism was activated to a greater extent between the repetitions of Set B. An increase in citrate production (an important aerobic metabolic intermediate) in the tri-carboxylic acid (TCA) may have inhibited phosphofructokinase (PFK) (Spriet, 1995), crucial in regulating glycolysis. It is possible that this may have increased aerobic metabolism (through increased fatty acid oxidation) whilst limiting glycolysis, reducing lactate production (and H^+ associated with this process) and providing a slower production of ATP for muscle

contraction. This may be one reason for the lower blood lactate values measured and the reduction in performance times during Set B of the AA trial.

Blood lactate after the completion of Set A was the same in all three trials and it was not changed by 5 min active recovery, consistent with other studies (Bonen and Belcastro, 1976; Cazorla et al., 1983; McGrail et al., 1978). However, similar blood lactate and pH values before the start of Set B does not mean that muscle lactate and intracellular acidity were the same. Blood lactate concentration is the result of its rate of production and removal (Hubbard, 1973). An increased blood flow between sets in the AA and AP trials may have removed H^+ faster, restoring the acid balance in the muscle. This increased efflux of H^+ from muscle would normally result in a greater decrease in blood pH following active recovery. In the present study blood pH was not different after passive or active recovery. However, active recovery will also stimulate H^+ uptake and utilisation by other muscles (Bangsbo et al., 1993) thus attenuating or even reversing the possible H^+ increase. It is possible therefore that muscle H^+ concentration was lower before Set B after active recovery allowing a better cell function.

The novel design of the present study found that the AP trial demonstrated better performance results than the AA trial and may highlight the relative successes of active and passive recovery following maximal (Set B) and near-maximal (Set A) repeated sprinting and also the timing and intensity of the active recovery. Active recovery at the intensity chosen in this study (60% of 100 m P.B. pace) was not as effective as passive recovery in restoring performance during the relatively short (~1 min 30 s) time between repetitions in Set B. Active recovery performed between repetitions may have been too intense immediately following maximal intensity sprint swimming. The

intensity of active recovery was selected to be the most efficient reported in the literature for lactate removal (Carzorla et al., 1983; McMaster et al., 1989). However, since blood lactate and pH at the beginning of a sprint do not correlate with subsequent performance (Bogdanis et al., 1996a; Felix et al., 1997; Siebers and McMurray, 1981; Weltman et al., 1979), it is likely that a lower intensity recovery may be more effective for subsequent performance in short recovery, high intensity repeated sprint swims.

The duration of active recovery in the present study was shorter than others used in previous swimming experiments (Carzorla et al., 1983; McMaster et al., 1989). This was in order to keep the rest intervals as close as possible to real training conditions and may have negated the effect of active recovery. Indeed, a longer recovery may have enhanced subsequent performance.

The results would suggest that either passive or extremely low intensity recovery should be performed immediately following maximal intensity repeated sprint swimming before higher intensity (e.g. 60% of 100 m best time) swimming during the slower phase of recovery. The difficulty is in determining the exact point at which this change should take place. This would probably involve invasive measurements and since the point is likely to vary amongst individuals, experienced competitive swimmers are probably best placed to determine their ideal pace (self selected). It is not surprising therefore that Carzorla et al., (1983) found enhanced lactate removal with active recovery using both self-selected pace and a pace equivalent to 60% of race velocity.

In conclusion, the hypothesis that active recovery would be the most effective for swimming performance between sprint repetitions and sets of repetitions was rejected.

The tendency for maintenance of performance in the AP trial may be attributed to the beneficial effects of active recovery between Sets A and B, such as a faster restoration of acid balance within the muscle cell, a faster resynthesis of PCr, or an increased aerobic metabolism. It would appear that when active recovery is performed both between sets and between sprints, it might have detrimental effect on swimming performance. This paradoxical effect may indicate that the intensity of the active recovery was too high, or that a period of passive rest initially followed by active recovery is needed for optimal recovery of performance. Further research is needed to establish the most efficient duration, intensity and timing of active recovery in repeated sprint swimming not only for the faster removal of blood lactate, but more importantly for better performance in subsequent sprint swimming.

CHAPTER VIII

EFFECTS OF ORAL CREATINE SUPPLEMENTATION ON SINGLE AND REPEATED SPRINT SWIMMING

8.1 Introduction

The decline in the rate of resynthesis of adenosine triphosphate (ATP) as a result of depletion of phosphocreatine (PCr) is recognised as a possible cause of reduction in muscular power in maximal intensity exercise (Hultman et al., 1967). Recent studies have shown that oral creatine supplementation can increase the total concentration of creatine and PCr in skeletal muscle and the rate of PCr resynthesis (Greenhaff et al., 1993a, 1994a, 1994b; Harris et al., 1992); increase muscle peak and mean power output during cycling (Birch et al., 1994); increase total work and reduce fatigue during repeated maximal exercise (Balsom et al., 1993; Greenhaff et al., 1993b, 1994a; Bogdanis et al., 1996b); improve running performance times (Harris et al., 1993); and improve recovery in repeated bouts of high intensity exercise (Balsom et al., 1993a; Greenhaff et al., 1993a, 1993b, 1994a; Bogdanis et al., 1996b).

These results suggest that oral creatine supplementation can improve performance in high intensity short term exercise by increasing resting concentrations of creatine and PCr and improving the rate of PCr and ATP resynthesis in skeletal muscle. However, all of these studies have used high dosages (i.e. 20-30 g day⁻¹) and none have examined sprint swimming performance using elite competitors.

Despite claims by the manufacturer of a creatine supplement (Ergomax™ C150) that swimming performance can be enhanced by 5% as a result of creatine supplementation, there are at present no reports of any swimming studies in the literature to confirm or refute these statements. In addition, the recommended dosage of their product (9 g creatine day⁻¹ for 5 days) is lower than those used in the above mentioned studies (20-25 g day⁻¹ for 5-6 days). Furthermore in a recent study, Cooke et al. (1995) reported no significant differences in performance during the first or second sprint of 2 cycle ergometer sprints separated by 20 min rest after oral creatine supplementation (20 g creatine day⁻¹ for 5 days). Thus, the efficacy of creatine supplementation for performance enhancement during a single sprint may be questioned.

In summary, there have been very few studies that have examined the effects of oral creatine supplementation on sports performance (in comparison with performance during laboratory tests such as isokinetic cycling), or that have used any group of subjects other than sedentary or active recreational sports people. In addition the uptake of creatine at a dosage of 9 g day⁻¹ is unknown, but has been shown to occur at dosages as low as 3 g day⁻¹ (Hultman et al., 1996). Thus the purpose of the present study was to examine the effects of oral creatine supplementation on the performance and metabolic responses of elite competitive swimmers during a single sprint swim of 50 yards and during a training set of 8 x 50 yards, and to estimate creatine uptake at 9 g day⁻¹ in this group of swimmers. It was hypothesised that creatine supplementation at 9 g day⁻¹ would enhance repeated, but not single sprint swimming performance.

8.2 Methods

Fourteen male swimmers from Loughborough University swimming club performed two sprint tests; a single 50 yards sprint under race conditions, and a sprint training set of 8 x 50 yards, beginning every 1 min 30 s. Subject characteristics are shown in Figure 8.1 and the study design, measurements and timing of the test protocol is shown in chapter IV, Figure 4.2. Subjects were randomly assigned to groups using a double blind design, and performed the 50 yards and 8 x 50 yards set before and the 8 x 50 yards set after the 5-day period of supplementation (see 8.2.1 below). The first repetition of the 8 x 50 yards post-supplementation was used in comparison with the single 50 yards test pre-supplementation. Tests were conducted one week apart to allow healing from needle punctures, full recovery after each test and standardisation (repeated tests were carried out at the same time and same day of the week). Details of the subject preparation, procedures, blood collection, handling and analysis can be found in chapter III.

Table 8.1 Subject physical characteristics and personal best (P.B.) times for Cr (n = 8) and Control (n = 8) groups; data are the mean ± S.D.

Subjects	Age (years)	Height (cm)	Weight (kg)	50 m F/C P.B. (s)	50 Yards F/C P.B. (s)
Cr					
Mean	20	181.2	75.6	24.73	22.26
S.D.	± 2	± 4.8	± 5.2	± 0.41	± 0.37
Control					
Mean	21	183.4	75.9	24.73	22.26
S.D.	± 2	± 6.2	± 9.3	± 0.36	± 0.33

P.B.: Personal best time; F/C: Front crawl; Cr: Creatine supplementation

A subsequent study was carried out more than 6 months after the original supplementation period to determine the retention of creatine after supplementation with 9 g day⁻¹ for five days. It was felt that the urinary study was too onerous to conduct for subjects during the supplementation period in addition to the testing protocol. Six male subjects (3 from the original Cr and 3 from the original control groups) followed an identical creatine supplementation regimen (see supplementation period). All urine samples from the 6 subjects were collected over a 9-day period, the daily volumes recorded and aliquots taken from each and frozen for later analysis (see chapter III, section 3.5.4). Samples were collected for 3 days prior to the 5-day supplementation period (to establish a baseline excretion for creatinine – see Rossiter et al., 1996), during the 5 days and day following the supplementation period.

8.2.1 Creatine supplementation (5 days)

Subjects were provided with 15 pre-measured packets of powder (either 3 g creatine + 1.5 g maltodextrin [i.e. 3 Powdered ErgomaxTM C150 tablets] + 1.5 g glucose: Cr, or 6 g glucose: control) and instructed to mix the powder in hot water or orange cordial for immediate consumption at 9:00, 13:00 and 17:00 h each day during the supplementation period. This loading procedure was recommended by the manufacturers and claimed to improve sprint race performance by 5%. Subjects were randomly assigned to Cr or control groups using a double blind design.

A three way analysis of variance or two-way where appropriate (Statistica/Mac) was used to examine differences between the control and creatine group (main effect group), between all subjects before and after supplementation (main effect trial) and to examine the response of all subjects over time (main effect time). As the main effect time was

always statistically significant at the $P < 0.01$ level, this main effect is not referred to in the tables or figures. Differing responses between the groups as a result of supplementation were identified by group-trial and group-trial-time interactions. Values are presented as means \pm standard deviation (S.D.).

8.3 Results

8.3.1 Performance times

Mean times recorded for the single 50 yards sprint were 22.95 ± 0.51 s pre-treatment and 23.24 ± 0.70 s post-treatment for the Cr group and 23.36 ± 0.50 s and 23.45 ± 0.58 s for the control group (Cr vs. control, N.S.). While performance times were not significantly different between groups or between treatments, the 50 yards times in the present study were significantly slower ($P < 0.01$) than personal best (P.B.) times for both groups (Cr PB 22.26 ± 0.33 s, control P.B. 22.26 ± 0.37 s). However, sprint 1 times in the repeated sprint test were the same as those achieved in the reference single sprint (single sprint time vs. sprint 1 of 8 x 50 yards, N.S.).

During the repeated sprint test, mean times declined (repetition number 1 to 8) from 23.35 ± 0.68 s to 26.32 ± 1.34 s in the control group and 23.20 ± 0.67 s to 26.85 ± 0.42 s in the Cr group before supplementation, and from 23.59 ± 0.66 s to 26.19 ± 1.48 s in the control group and 23.39 ± 0.54 s to 25.73 ± 0.26 s in the Cr group after supplementation ($P < 0.03$, group-trial interaction: Figure 8.1). The overall improvement is confirmed by the reduction in total sprint time in the Cr group after supplementation: 204.28 ± 5.02 s vs. 200.19 ± 3.86 s, whilst the control group showed no improvement: 204.65 ± 8.04 s vs. 204.31 ± 8.69 s (group-trial interaction, $P < 0.05$).

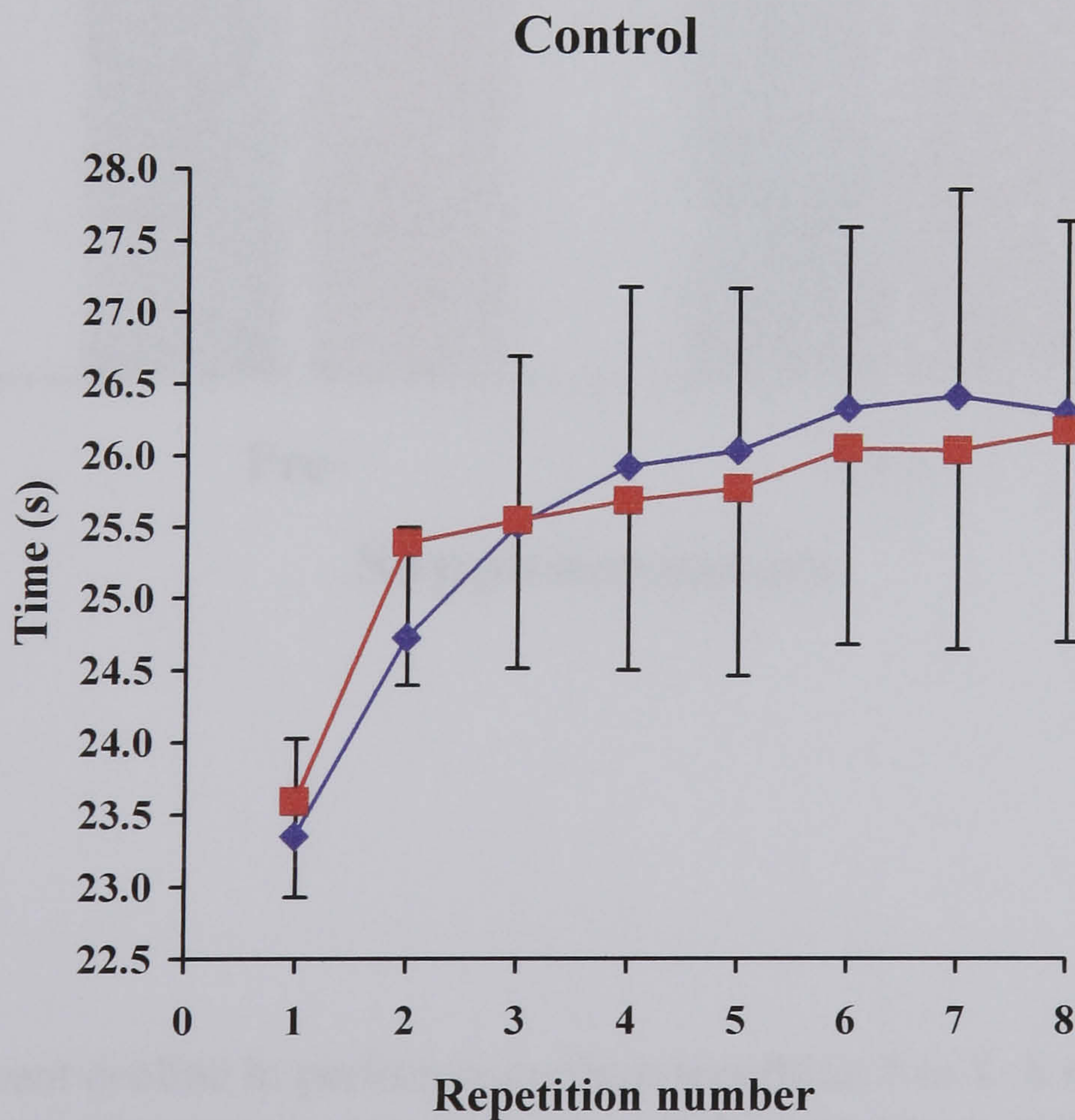
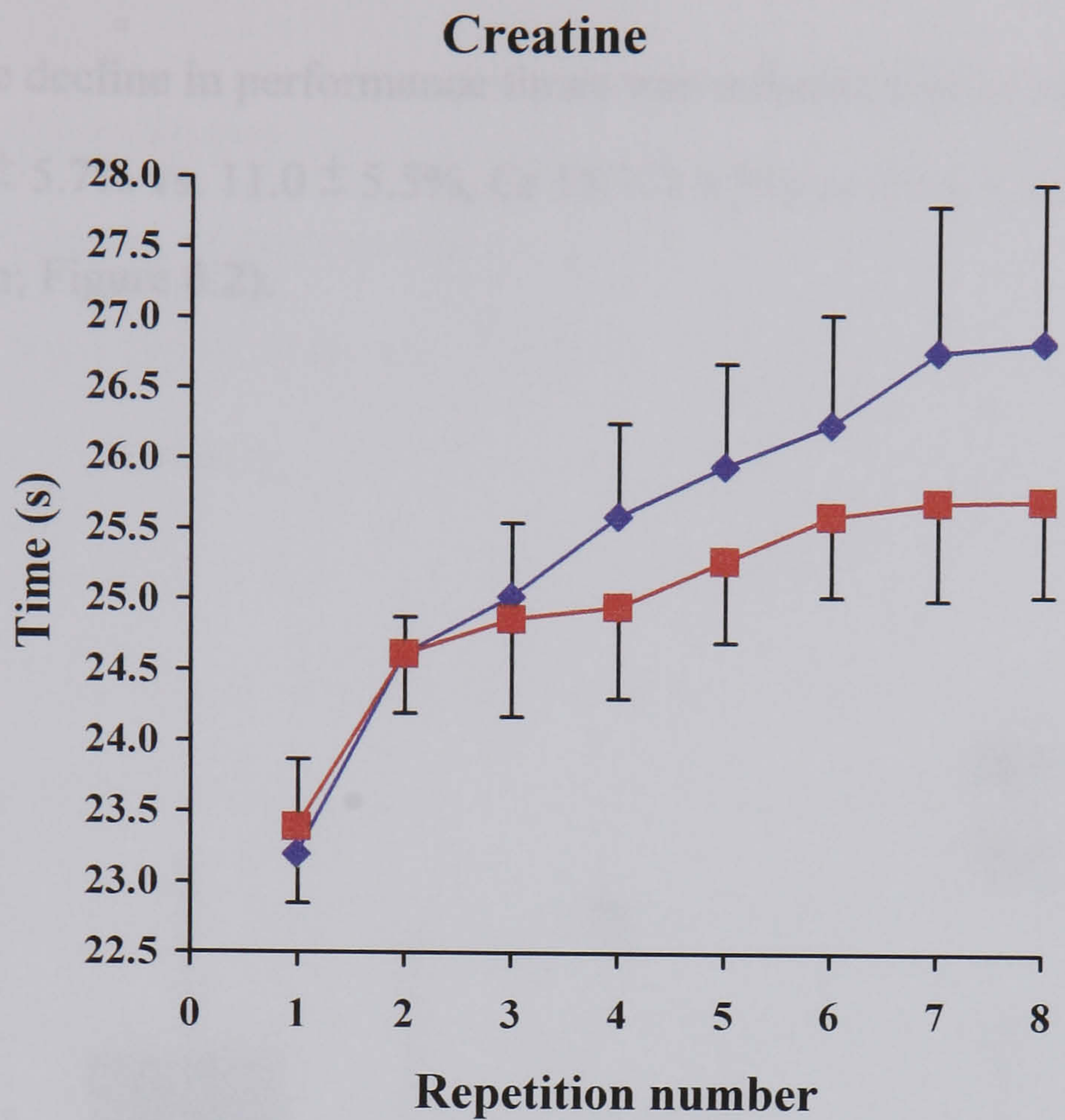


Figure 8.1 Performance times for Cr and control groups during 8 x 50 yards sprints pre- and post-supplementation (Cr n = 7, Control n = 7). Pre-supplementation data are presented in blue, Post-supplementation data in red

a group x time x trial interaction $P < 0.05$

The percentage decline in performance times was reduced after creatine supplementation (Control $12.7 \pm 5.7\%$ vs. $11.0 \pm 5.5\%$, Cr $15.7 \pm 4.3\%$ vs. $10.0 \pm 2.5\%$, $P<0.05$; group-trial interaction; Figure 8.2).

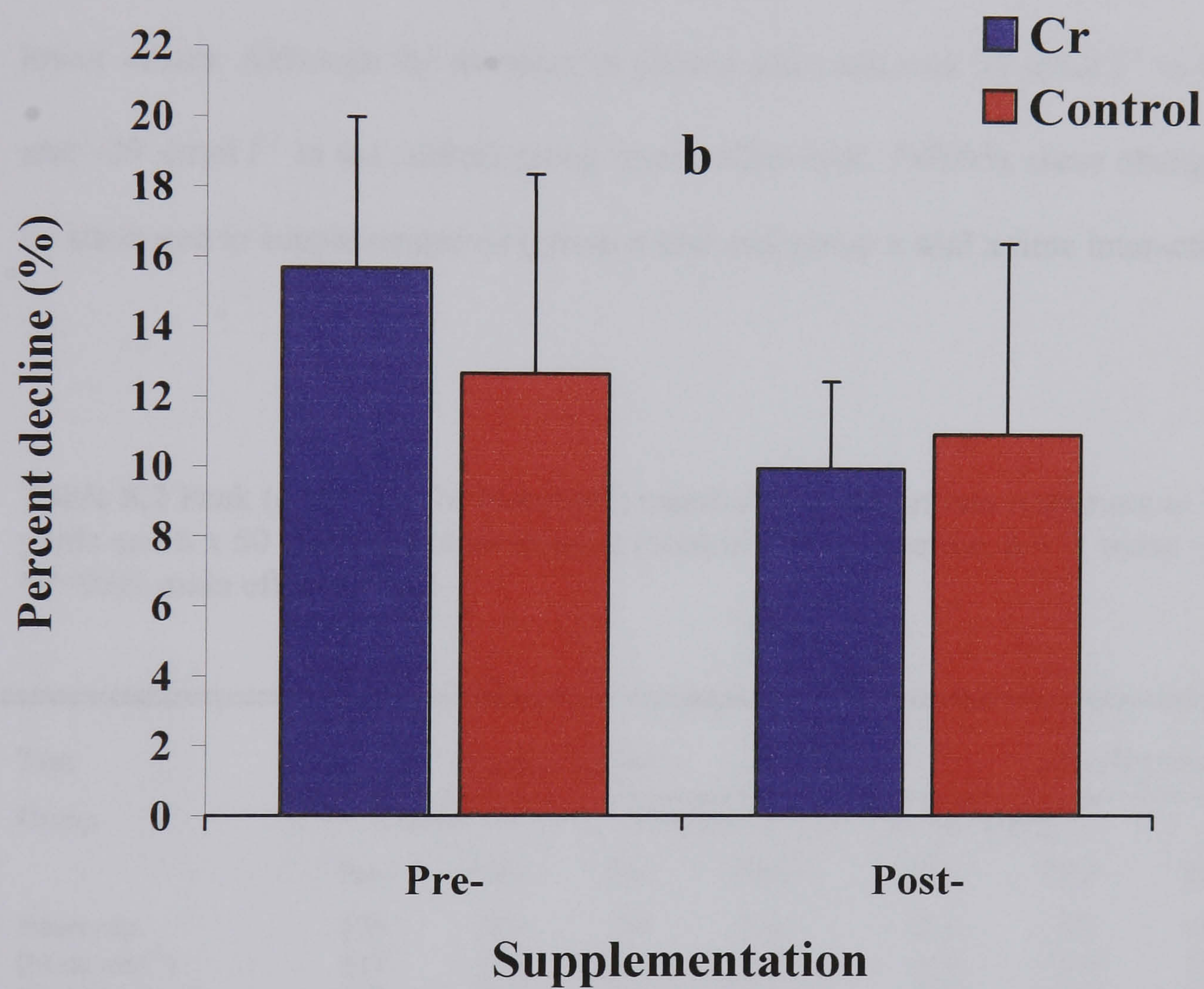


Figure 8.2 Percent decline in performance from repetition 1 to 8 (8 x 50 yards test) for the control and creatine groups, pre- and post-supplementation (control, n = 7; creatine, n = 7; mean + S.D.).
b $P<0.05$ (group x trial interaction)

8.3.2 Metabolic responses

Metabolic responses of both groups in all tests are shown in Table 8.2. No significant differences were found with any metabolic variable between groups or treatments, although peak blood lactate concentrations were significantly higher ($P<0.01$) in the 8 x 50 yards test than the 1 x 50 yards test. In the Cr group, one subject recorded the same peak ammonia value pre- and post-creatine supplementation and the other six recorded lower values. Although the decrease in plasma ammonia was $53\text{ }\mu\text{mol l}^{-1}$ in the Cr group and $\sim 39\text{ }\mu\text{mol l}^{-1}$ in the control group (main effect trial, $P<0.05$), these changes could not be attributed to supplementation (group x trial and group x trial x time interactions, N.S.).

Table 8.2 Peak (or lowest for blood pH) metabolic and heart rate responses to 1 x 50 yards and 8 x 50 yards sprint swimming (control, n = 7; creatine, n = 7; mean \pm S.D.)
* $P<0.05$ main effect of trial

Test	50 yards sprint				8 x 50 yards			
	Control		Creatine		Control		Creatine	
Group	Pre-	Post-	Pre-	Post-	Pre-	Post-	Pre-	Post-
Heart rate	176	175	180	171	186	177	188	181
(beats min ⁻¹)	± 17	± 15	± 18	± 9	± 13	± 10	± 15	± 17
Blood lactate	9.7	9.6	9.7	9.3	18.0	16.8	17.8	17.3
(mmol l ⁻¹)	± 2.4	± 2.3	± 0.6	± 1.0	± 1.9	± 2.7	± 3.0	± 1.8
Ammonia					217.9	178.8	227.5	174.5
($\mu\text{mol l}^{-1}$) *					± 49.8	± 51.1	± 75.8	± 20.0
Blood pH					6.98	7.00	6.98	7.01
					± 0.05	± 0.03	± 0.07	± 0.04

Plasma ammonia and blood pH were determined from venous blood. Blood lactate concentrations were determined from capillary samples for both trials as venous samples were not taken during the single sprint.

Urinary analysis

Mean daily creatinine excretion was 1.63 ± 0.35 g over the first three days of collection and 2.21 ± 0.26 g during the five-day period of supplementation ($P < 0.001$). No creatine was recovered in the urine during the first three days, but rose from 1.32 ± 0.52 g on the first day of supplementation to 4.45 ± 1.03 g by the fifth day (Figure 8.3).

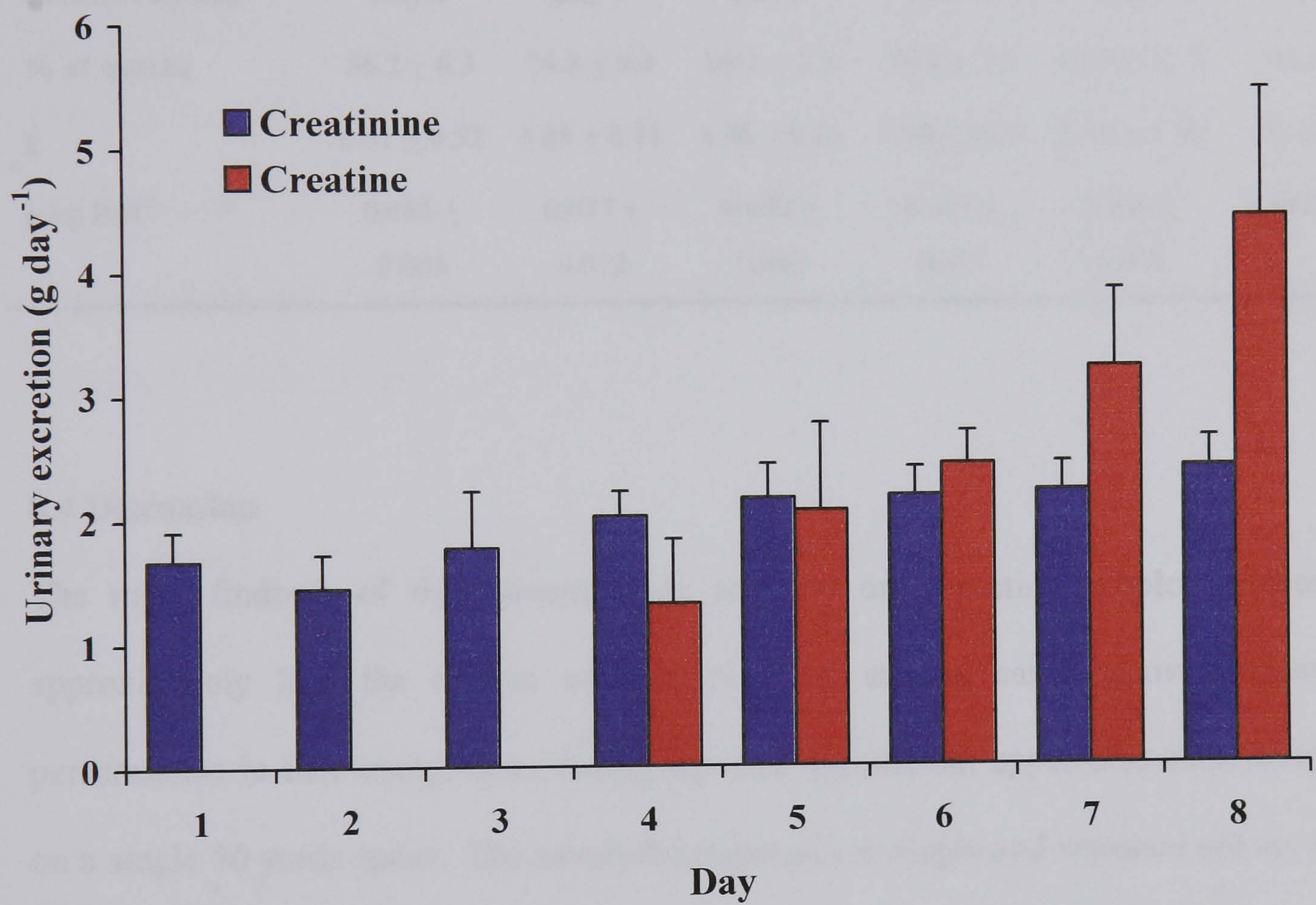


Figure 8.3 Urinary creatine and creatinine excretion over an 8-day period ($n = 6$). Creatine supplementation (9 g day^{-1}) took place on days 4-8.

Creatine uptake was highest on day one of supplementation (6.61 ± 0.52 g) and declined each day to 3.49 ± 1.04 g by day 5. Creatine retained was $86.2 \pm 6.3\%$ for day one and $45.9 \pm 12.7\%$ for day 5 of the intake value. The total amount of creatine retained was 26.1 ± 2.0 g or $66.9 \pm 5.1\%$ of the total administered over five days (Table 8.3). The range of creatine retention was 23.4 - 27.8 g or 59.2 - 71.5%.

Table 8.3 Creatine uptake during creatine supplementation of 9 g creatine monohydrate (7.92 g creatine) per day for 5 days (n = 6, mean \pm S.D.)

Creatine uptake	Day 4	Day 5	Day 6	Day 7	Day 8	Total
% of uptake	86.2 ± 6.3	74.8 ± 9.4	69.3 ± 3.3	58.8 ± 7.5	45.9 ± 12.7	66.9 ± 5.1
g	6.61 ± 0.52	5.85 ± 0.71	5.48 ± 0.26	4.69 ± 0.63	3.49 ± 1.04	26.12 ± 2.01
g kg BM ⁻¹	$0.086 \pm$ 0.008	$0.077 \pm$ 0.012	$0.072 \pm$ 0.006	$0.061 \pm$ 0.011	$0.046 \pm$ 0.016	0.342 ± 0.046

8.4 Discussion

The main findings of the present study are that oral creatine supplementation at approximately half the dosage used in previous studies can improve swimming performance in elite competitors during repeated sprints, but appears to have no effect on a single 50 yards sprint. The metabolic responses to single and repeated sprints were similar even though performance times in the repeated sprints test were faster after creatine supplementation.

The time recorded in the 1 x 50 yards sprint was used as a reference of the current sprint performance status of the swimmer. No significant differences were observed between the 1 x 50 yards time and the first repetition in the 8 x 50 yards set; an indication that a maximal sprint was recorded in both tests and that the swimmers were highly

motivated. However, personal best times were significantly faster than the 1 x 50 yards and the first repetition of the 8 x 50 yards. This was probably due to lack of specific competition preparation i.e. lack of 'taper' and 'shaving down' (Costill et al., 1992; Sharp and Costill, 1989), as 3 – 4% improvements in performance time after a taper have been reported (Costill, 1999).

The results of the present study support the findings of other authors who observed no differences in peak power output or performance in single exercise bouts following creatine supplementation - Balsom et al. (1993) in the first 6 s bout of maximal cycling exercise; Bogdanis et al. (1996) in the first 10 s treadmill sprint of a series; Cooke et al. (1995) in the first of two 15 s cycle ergometer sprints; Greenhaff et al. (1993b) in maximal isometric leg extensor torque; Odland et al. (1994) in short-term maximal sprint cycle performance.

Although performance in a single bout of exercise seems to be unaffected by creatine supplementation in this and earlier studies, it has been shown that the total creatine content of skeletal muscle is increased by 20-50% of which approximately 20% could be accounted for as PCr (Harris et al., 1992). Increasing the PCr concentration of the muscle would, therefore, appear to have no effect on peak power production or performance in a single bout of short duration exercise, despite the view that availability of PCr may be a limitation in the recovery of power output (Bogdanis et al., 1995). An explanation for this observation may involve the velocity (V) of the creatine kinase reaction that is, in part, determined by substrate concentration. At the start of exercise this value will be close to maximum (V_{\max}), and therefore to increase the substrate

(PCr) concentration further will not affect the velocity of this reaction and consequently peak power output and short term swimming performance.

However, creatine supplementation did improve performance towards the end of the 8 x 50 yd test in this group of elite swimmers. As performance was improved, it seems possible that even 9 g creatine day⁻¹ for 5 days may raise the muscle creatine content, thus providing a mechanism for the improvement. Evidence for this suggestion is provided by the urinary analysis data which showed a ~67% or ~26 g retention of creatine over the 5 day period. The percentage retention of creatine was higher than in previous studies (15-32%: Greenhaff et al., 1994b; 25-55% - Rossiter et al., 1996), but the total amount of creatine retained and the estimated muscle creatine uptake (28.0 ± 3.8 mmol kg dry weight⁻¹), assuming a muscle mass estimate of 40% of body mass and muscle water content of 3.3 litres per 4.3 kg wet muscle weight (Harris et al., 1992), were similar to the values reported by Rossiter and co-workers (~35 g and 38.1 ± 10 mmol kg dry weight⁻¹). In addition, a recent study has shown that creatine uptake into the muscle occurs even at lower dosages (3 g day⁻¹) after 14 days of supplementation (Hultman et al., 1996). In the present study, uptake of creatine may have been facilitated by increased blood flow as a result of the supervised training (4,000-5,000 m per day) undertaken by the swimmers during the supplementation period. For example, Harris et al. (1992) found that subjects who performed single-legged exercise showed muscle creatine increases of ~44 mmol kg dry weight⁻¹ in the exercised leg compared with only ~30 mmol kg dry weight⁻¹ in the non-exercised leg. Also the administration of creatine together with glucose and maltodextrin close to meals with a high carbohydrate content may have aided muscle creatine uptake by raising serum insulin levels (Green et al., 1995). Furthermore, these University students on low finance may have had a dietary deficiency of creatine. Thus it

seems that 9 g day^{-1} did increase muscle creatine content. Therefore, the mechanism of improvement in repeated sprints in the present study may have been that creatine concentration was maintained above the K_m (*Michaelis constant*: enzyme reaction velocity half-time; Maughan et al., 1997) value ($\sim 19 \text{ mmol l}^{-1}$ assuming full activation) for the creatine kinase reaction thereby increasing the rate of PCr resynthesis between sprints (Greenhaff et al., 1994b).

Improved buffering through the increase in muscle creatine may additionally have caused improvements observed in the 8 x 50 yards test. Adenosine 5'-triphosphate resynthesis from ADP and PCr consumes a hydrogen ion (H^+) in the process. An increase in the PCr turnover rate through greater creatine content in the muscle will therefore consume more H^+ and improve muscle buffering capacity. In the present study the changes in blood pH were similar before and after supplementation, despite repeated sprint performance improvements.

Blood lactate values recorded in the single sprint were similar to those reported by Balsom et al. (1993a), Bonifazi et al. (1993), and Greenhaff et al. (1993a), and slightly lower than those reported by Birch et al. (1994). In accordance with Birch et al. (1994) and Greenhaff et al. (1993a), blood lactate values in the present study were not significantly different after creatine supplementation. These findings contradict the significant decline in blood lactate after creatine supplementation found by Balsom et al. (1993a), although reductions observed in that study were small ($10.8 - 9.0 \text{ mmol l}^{-1}$). In the 8 x 50 yards test peak blood lactate values were higher than those of 9 to 15 mmol l^{-1} reported by Holmer (1972) and Nadel et al. (1974) for maximal swimming

over 30 s to 5 min, but similar in range to those reported by Sawka et al. (1979) and Pelayo et al. (1996) in repeated swimming sprints. Creatine supplementation did not alter the blood lactate response even though sprint swimming performance was improved suggesting that the higher rate of ATP resynthesis required for the faster swims was achieved without any change in the contribution from glycolysis to energy supply.

Plasma ammonia values, reflecting AMP deamination, measured in the 8 x 50 yards test may give further information regarding the mechanisms responsible for the performance improvements observed by the Cr group during the later repetitions (Banister et al. 1985; Greenhaff et al., 1993b). This reaction is accelerated in extreme, high intensity exercise bouts (such as in this study) to support high ATP turnover rates. Although plasma ammonia values were lower post-supplementation, there was no difference between the groups. Large inter-individual differences may have caused this effect. In addition, the smaller creatine dosage used in the present experiment may not have exaggerated the change in purine metabolism to the same extent as in other studies (Balsom et al., 1993a; Greenhaff et al., 1993b; Bogdanis et al., 1996b). However, six out of seven subjects in the Cr group showed a decline in plasma ammonia after supplementation. This coupled with an improvement in performance times in repetitions towards the end of the 8 x 50 yards test indicate support of high ATP turnover rates from sources other than AMP deamination; most likely from an increased contribution from PCr degradation as a result of increased PCr resynthesis.

In summary, the findings of the present study suggest that oral creatine supplementation (9 g day^{-1} for 5 days) does not affect performance of elite swimmers over 50 yards but

does improve performance during a repeated sprint swimming set (8 x 50 yards). Evidence for creatine supplementation as the cause of performance improvements is provided from the urinary analysis results which showed ~67% (~26 g) retention of the administered creatine. The probable mechanism accounting for the performance improvement is an increase in muscle creatine content leading to increased PCr resynthesis and thus higher pre-exercise muscle PCr content in the latter sprints of the 8 x 50 yards set.

CHAPTER IX

EFFECTS OF CREATINE SUPPLEMENTATION ON TRAINING FOR COMPETITION IN ELITE COMPETITIVE SWIMMERS

9.1 Introduction

Oral creatine (Cr) supplementation has been demonstrated to increase resting concentrations of creatine and phosphocreatine in skeletal muscle (Harris et al., 1992), and improve performance during intermittent high intensity exercise through an increased recovery rate (Balsom et al., 1993a; Greenhaff et al., 1993a). In contrast, improvements in performance have not been identified during single sprint bouts of cycling, running or knee extensions (Balsom et al., 1993a; Cooke et al., 1995; Greenhaff et al., 1993a) or in elite competitive swimmers during 25 m, 50 m/yards or 100 m race distances (chapter VIII; Burke et al., 1996; Mujika et al., 1996). Swimming performance in repeated sprints has been found to improve following creatine supplementation (chapter VIII; Grindstaff et al., 1998; Leenders et al., 1999). The benefit of this ergogenic effect in swimming is likely to be in enhancing training, where repeated bouts of high intensity swimming are regularly performed, rather than in competition, where single sprint performance prevails.

Ten weeks of strength training combined with creatine supplementation has been shown to result in an enhanced gain in high intensity intermittent exercise capacity during isokinetic arm flexions in previously sedentary subjects compared to a control group (Vandenberghe et al., 1996, 1997). A recent study did not identify any differences in the

repeated sprint performance of elite swimmers between a low dosage ‘maintenance’ period (5 g day^{-1}) and no supplementation (Theodorou et al., 1999). However, this study did not investigate the physiological responses of creatine supplementation to training and was conducted over a relatively short, 8-week period. To date, there are no longer term (>10 weeks) training studies that have investigated the physiological effects of oral creatine supplementation on elite performers training in a sports-specific environment.

Sprint exercise has been shown to be a potent stimulant for human growth hormone (hGH) secretion (Nevill et al., 1996b; Stokes et al., 2002). In addition, a recent study found acute creatine supplementation (a single 20 g dose) to enhance hGH secretion in a similar manner to that observed following sprint exercise, perhaps causing a Cr induced heavy chain protein synthesis (Schedel et al., 2000). Eighteen weeks of swimming training has also been found to enhance the hGH response following an endurance training set (Bonifazi et al., 1998). No study has yet considered the hGH response to sprint swimming over a longer period of training (>8 weeks) combined with a commonly used creatine supplementation regimen (e.g. 20 g day^{-1} for 5 days).

The purpose of this study therefore, is to determine the performance, metabolic and hormonal effects of oral creatine supplementation on 22-27 weeks of training for competition in elite swimmers and to test the hypothesis that supplementing with creatine (3 g day^{-1}) over a 7-month training period will enhance training and consequently improve performance.

9.2 Methods

Twenty-three swimmers (14 male, 9 female) from Loughborough University swimming club began the training study. Three subjects dropped out during the course of the 28-week training period due to injury (2 males) and illness (1 female), leaving 12 male and 8 female swimmers. The protocol was approved by the Ethical Committee of Loughborough University who were satisfied that there are no known or reported side effects from creatine supplementation at the doses (20 g of creatine per day for 5 days, or 3 g per day for ≤ 196 days) prescribed for this study (Poortmans and Francaux, 1999; Schilling et al., 2001).

Subject characteristics are shown in Table 9.1 and the study design, measurements and timing of the test protocol is shown in Figure 9.1. Subjects performed the 5 x 200 yards (3.4.1.2) and 8 x 50 yards set (chapter III, 3.4.1.1) before and the 8 x 50 yards set after the 5-day period of supplementation were then assigned (pairs matched for time, event and standard) to an experimental (creatine supplementation, Cr) and control (glucose) group (see below). Details of the subject preparation, procedures, blood collection, handling and analysis can be found in chapter III.

Table 9.1 Subject physical characteristics and personal best (P.B.) times (N.S. between the groups) for Cr (n = 9; 5 male, 4 female) and Control (n = 11; 7 male, 4 female) groups; data are the mean \pm S.D.

Subjects	Age (years)	Height (cm)	Weight (kg)	100 m F/C P.B. (s) M	100 m F/C P.B. (s) F
Cr					
Mean	20	180.2	74.7	52.6	57.9
S.D.	± 1	± 11.1	± 15.2	± 1.3	± 1.8
Control					
Mean	20	179.1	73.7	54.2	58.5
S.D.	± 2	± 9.4	± 11.6	± 1.8	± 1.8

P.B.: Personal best time; F/C: Front crawl; Cr: Creatine supplementation; M: Male; F: Female

Subjects performed five testing sessions during the course of the swimming season: On two occasions a typical speed-lactate swimming test comprising 5 x 200 yards incremental repeats (ET – chapter III, 3.4.1.2; adapted from Harrison et al., 1992; Pyne et al., 2001), and on three occasions a maximal sprint training set of 8 x 50 yards (T; chapter III, 3.4.1.1). Test sessions took place before (both tests: T1 and ET1) and after a 5-day supplementation period (8 x 50 yards test only: T2), and both tests (T3 and ET2) after a 22-27 week training period that culminated in the GB World Championship Trials or other appropriate event within a two-week period. The supplementation period involved two high dosage 'loading' phases (chapter III, 3.4.3.1) and a low dosage (chapter III, 3.4.3.2) 'maintenance' phase (Hultman et al., 1996; Vandenberghe et al., 1997). One week before the GB World Championship Trials (or relevant alternative event), all subjects were again provided with 20 pre-measured packets (as above) and conducted a similar 'loading' regimen. The only difference between the groups, and therefore the intervention, was the Cr or placebo condition during the training period.

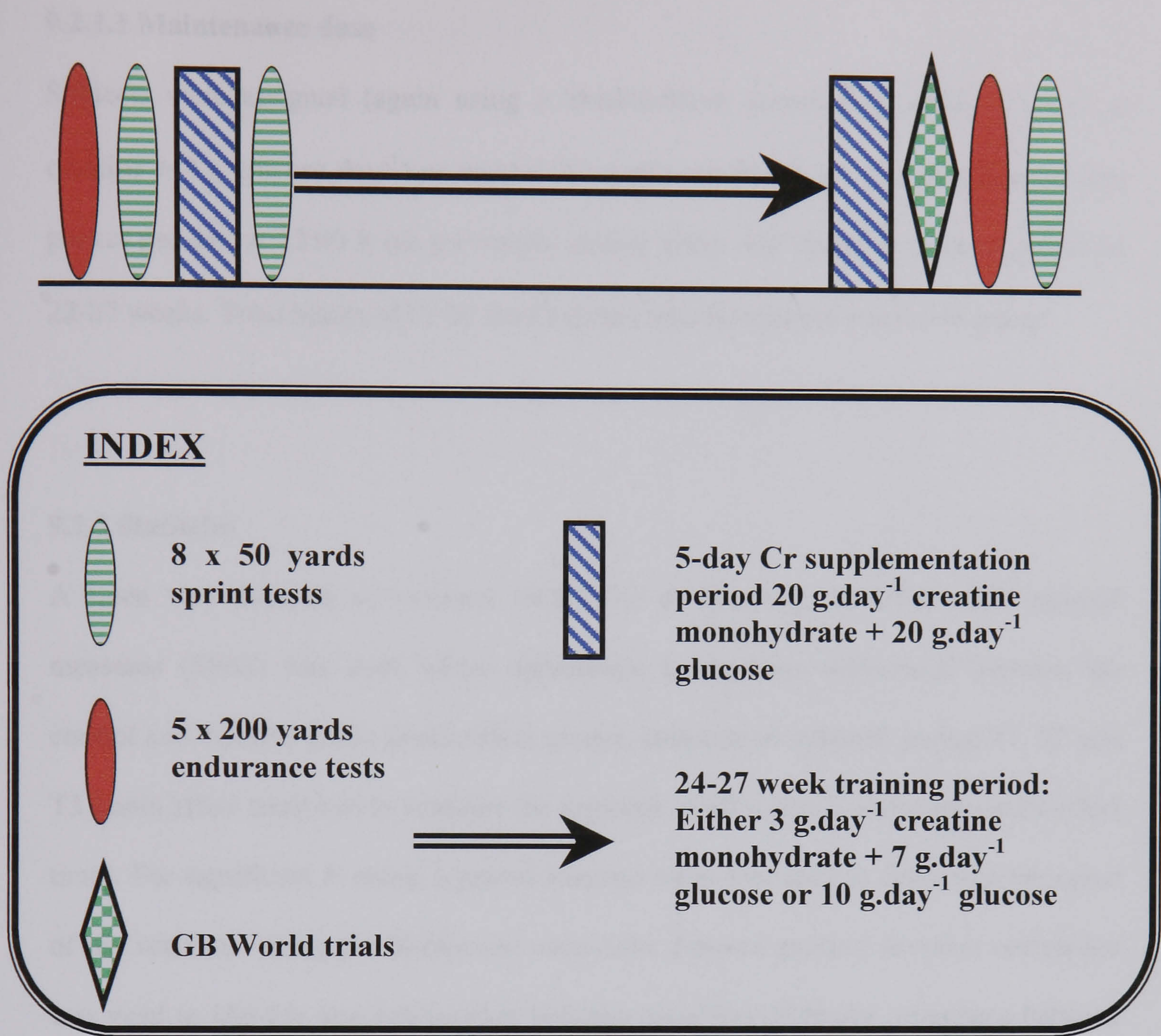


Figure 9.1 Schematic illustration of the training, competition and test protocols.

9.2.1 Creatine supplementation

9.2.1.1 Loading dose (5 days)

In chapter IX, all subjects were provided with 20 pre-measured supplement packets (5 g creatine [i.e. ‘CreatineTM’, Optima Health Care, Cardiff, U.K.] + 5 g glucose) and instructed to mix the powder in hot water or cordial for immediate consumption at 9:00, 12:00, 15:00 and 18:00 h each day during the ‘loading’ supplementation period.

9.2.1.1 Maintenance dose

Subjects were assigned (again using a 'double-blind' protocol) to either a Cr (3 g creatine + 7 g glucose day⁻¹) or control (10 g glucose day⁻¹) group and consumed one packet per day at 12:00 h (as previously stated) every day during a training period of 22-27 weeks. Total intake of Cr by the Cr group over this period was ≤ 546 grams.

9.2.2 Statistics

A three way analysis of variance (ANOVA) or two-way ANOVA with repeated measures (SPSS) was used where appropriate to examine differences between the control and creatine group (main effect group), between all subjects during T1, T2 and T3 (main effect trial) and to examine the response of all subjects over time (main effect time). For significant *F* ratios, a paired *Students* t-test was used to determine the cause of the variance using the *Bonferroni* correction. Pearson product moment correlation was used to identify any relationship between variables. Differing responses between the groups as a result of supplementation were identified by group-trial and group-trial-time interactions. Values are presented as means \pm standard deviation (S.D.) and significance set at the $P < 0.05$ level.

Performance data for all subjects was collected, although due to subject drop out, one subject's fear of needles and spoilt samples, metabolic and hormonal data is for a smaller number. Numbers for each analysis for both groups are detailed clearly in tables and figures legends. Since the main effect time was always statistically significant at the $P < 0.01$ level (except for 2 repetitions during the 8 x 50 yards test) this main effect is not referred to in the tables or figures. Times have been combined for male and female

subjects to avoid small group sizes ($n = 4$), although statistically, no gender-group differences were observed.

9.3 Results

9.3.1 Body composition

Body mass for T1, T2 and T3 was 75.0 ± 15.5 kg, 74.8 ± 15.5 kg and 75.6 ± 15.6 kg, and 73.7 ± 11.7 kg, 73.7 ± 11.4 kg and 72.9 ± 9.8 kg for Cr and control groups, respectively (group and trial main effects, N.S.). The 4-site sum of skinfolds was 36.3 ± 13.1 mm, 36.7 ± 12.8 mm and 37.9 ± 11.5 mm for the Cr group, and 50.1 ± 17.5 mm, 50.6 ± 17.1 mm and 47.1 ± 9.4 mm for the control group in the 3 trials respectively (Cr vs. control $P < 0.05$; group x trial interaction, N.S.).

9.3.2 Training and competition

Mean performance times for the subject's best event during the training period changed by 1.90 ± 1.91 % and 0.14 ± 1.14 % in the Cr group compared with 0.86 ± 1.60 % and -0.49 ± 0.95 % in the control group for short course (S.C. - 25 m pool) and long course (L.C. - 50 m pool) times respectively (S.C. pre- vs. post-training, $P < 0.05$; group x trial interaction and L.C. times, N.S.: Figure 9.2). Training therefore was effective for SC competition, but not different between Cr and control groups.

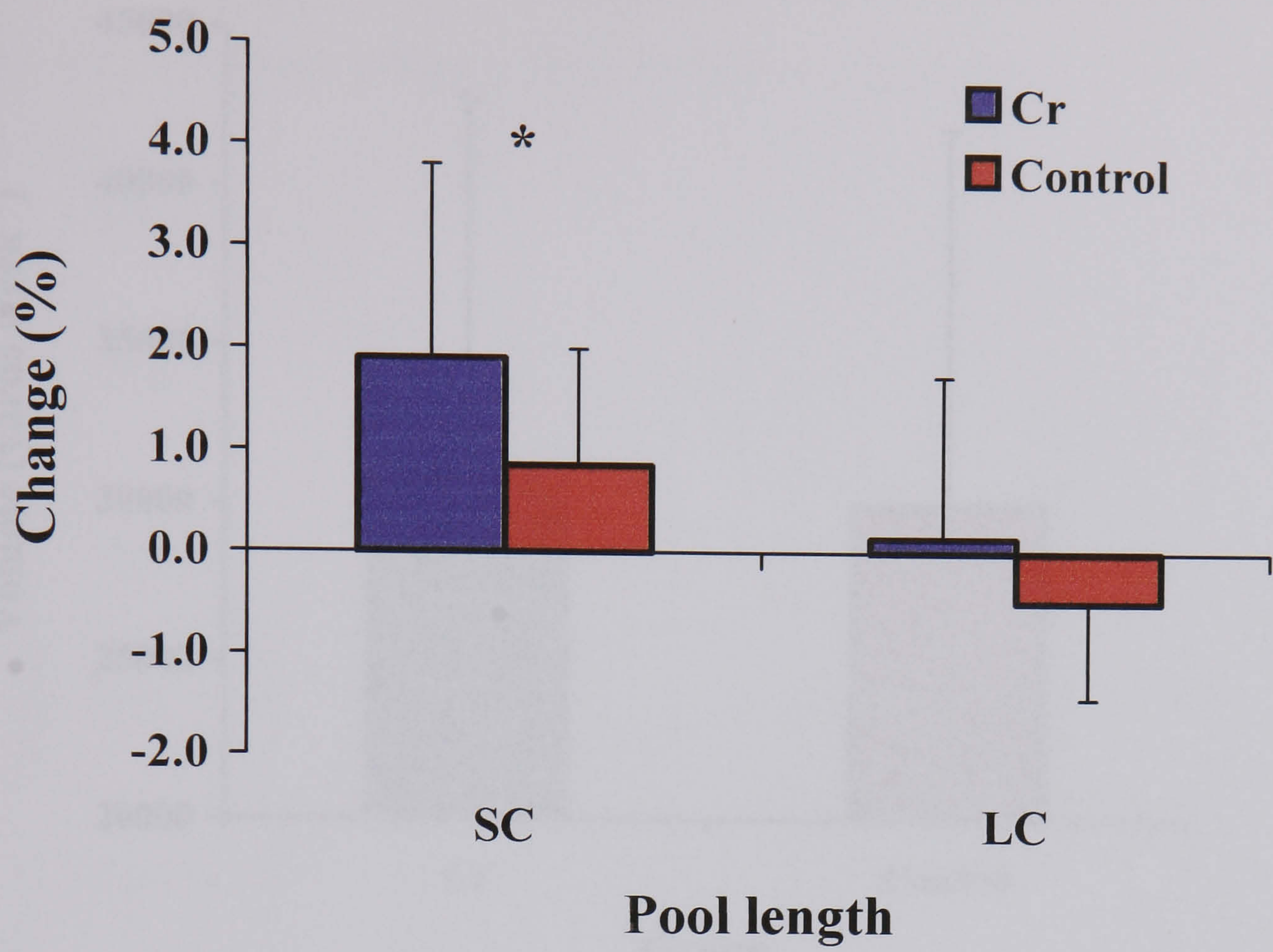


Figure 9.2 Competition best-event performance changes conducted throughout the study (Cr n = 9; control n = 11; mean \pm S.D.).
*P<0.05 Short Course pre- vs. post-training

No differences in training volume were observed between groups throughout the 22-27 week period (Cr 31,150 \pm 11,750 vs. Control 30,050 \pm 11,950 yards week⁻¹; N.S.). These values include periods of taper and the Christmas holiday where training volumes were significantly reduced (Figure 9.3).

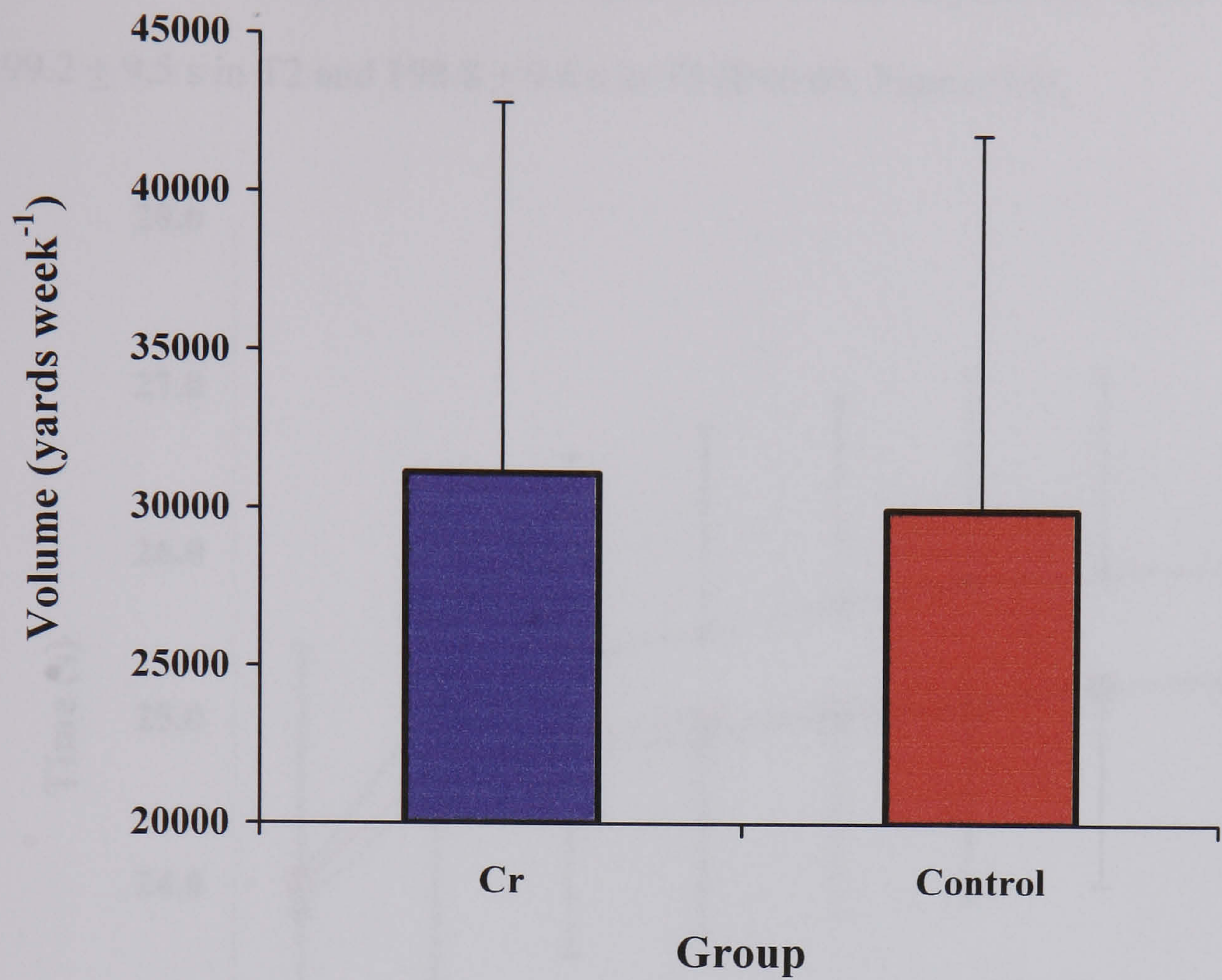


Figure 9.3 Swimmer training volumes conducted throughout the study (Cr n = 9; control n = 11; mean \pm S.D.).

9.3.3 Performance times

Performance times recorded for the first repetition in the 8 x 50 yards sprint test were 23.88 ± 1.68 s in T1, 23.84 ± 1.50 s in T2 and 23.70 ± 1.58 s in T3 for the Cr group and 24.20 ± 1.09 s, 24.22 ± 1.34 s and 24.00 ± 1.20 s for the control group (group and trial main effects, N.S.: Figure 9.4 and 9.5).

During the repeated sprint test, 50 yards times slowed in all subjects (repetition number 1 to 8) by $8.1 \pm 3.1\%$, $5.3 \pm 2.5\%$ and $6.0 \pm 2.8\%$ in T1, T2 and T3, respectively (T1 vs. T2

and T3, $P<0.05$: Figure 9.4). Total sprint time for all subjects was 203.5 ± 9.9 s in T1 vs. 199.2 ± 9.5 s in T2 and 198.8 ± 9.6 s in T3 ($P<0.05$: Figure 9.4).

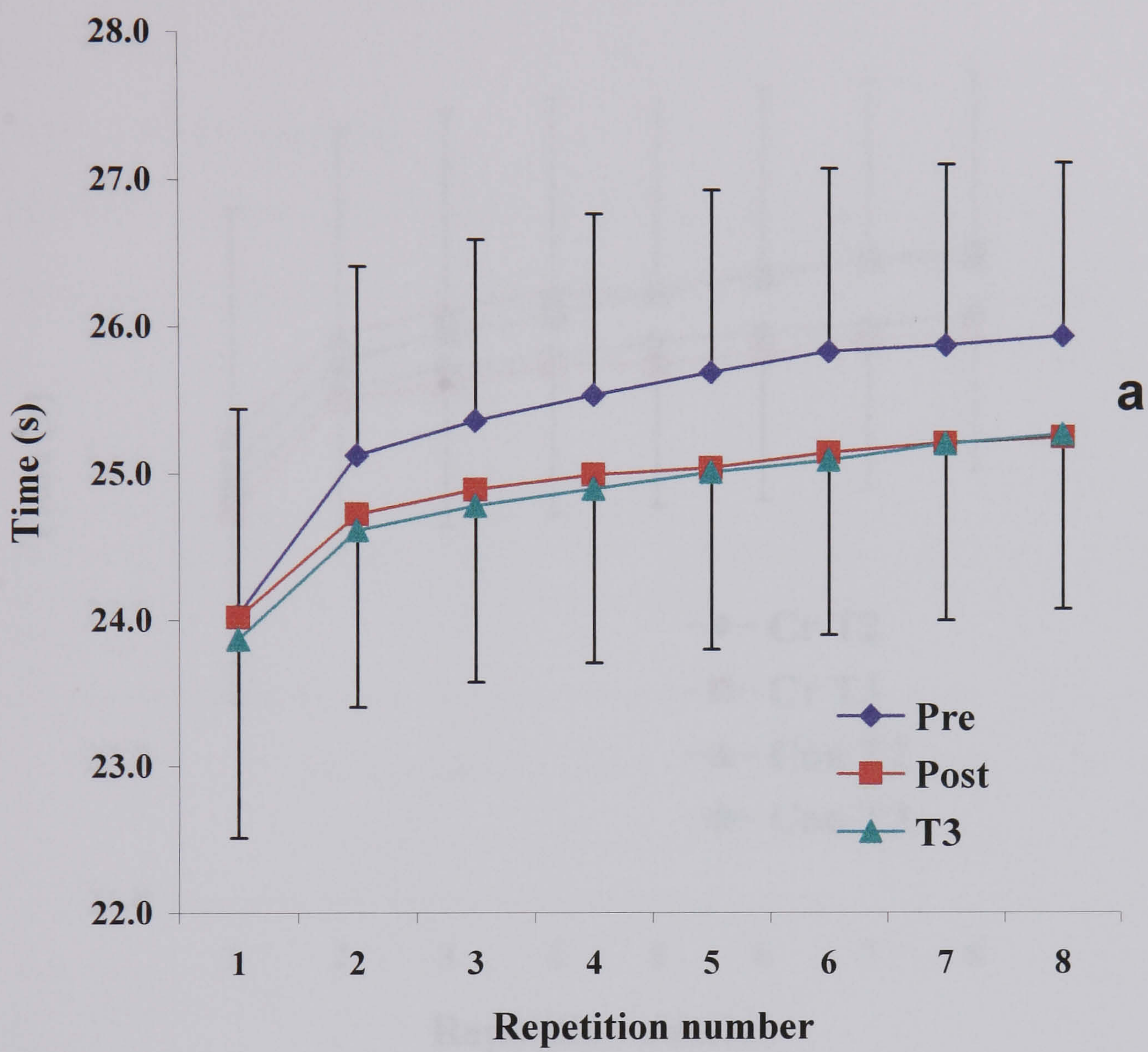


Figure 9.4 Mean performance times for all subjects in the three 8 x 50 yards tests (n = 20; mean \pm S.D.).
a $P<0.05$ test 1 vs. test 2 and 3.

No differences in the decline in performance time (rep 1 to rep 8 during the 8 x 50 yards test) between Cr and control groups were observed (Cr $8.2 \pm 2.8\%$, $5.3 \pm 2.2\%$ and $5.5 \pm 3.0\%$, control $8.1 \pm 3.45\%$, $5.3 \pm 2.8\%$ and $6.5 \pm 2.6\%$ for T1, T2 and T3 respectively,

N.S.: Figure 9.5). Values for T2 and T3 only for Cr and control groups have been represented for clarity.

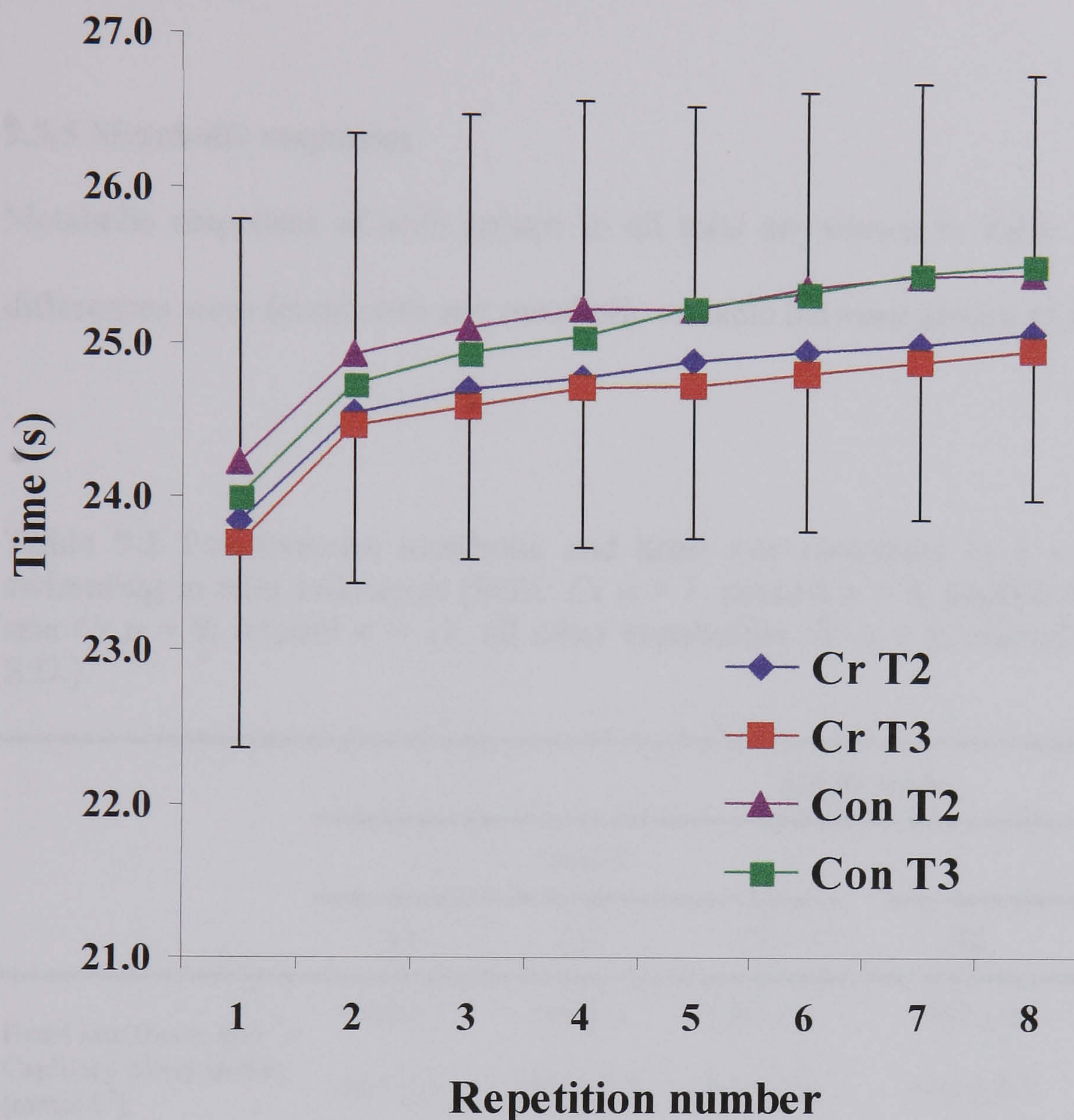


Figure 9.5 Mean performance times for Cr and control groups pre- and post-training in the 8 x 50 yards tests (Cr $n = 9$; control $n = 11$; mean \pm S.D.).

9.3.4 Endurance tests

Speeds corresponding to a fixed blood lactate concentration of 4 mmol l^{-1} (V4) changed between tests from 1.57 ± 0.05 and $1.58 \pm 0.05 \text{ yards s}^{-1}$ to 1.60 ± 0.11 and $1.54 \pm 0.09 \text{ yards s}^{-1}$ for Cr and control groups respectively (Cr vs. control, N.S.). The change in values represent changes of $1.54 \pm 5.56\%$ in the Cr group and $-2.33 \pm 4.78\%$ for control

subjects. A significant ($P<0.05$) correlation was found between the changes in V4 values and S.C. times ($r = 0.50$), but not between V4 and L.C. changes ($r = 0.28$).

9.3.5 Metabolic responses

Metabolic responses of both groups in all tests are shown in Table 9.2. No significant differences were found with any metabolic variable between groups or treatments.

Table 9.2 Post-exercise metabolic and heart rate responses to 8 x 50 yards sprint swimming in elite swimmers (hGH: Cr n = 7, control n = 8; capillary blood and heart rate Cr n = 9; control n = 11; all other metabolites Cr n = 8; control n = 11; mean \pm S.D.).

	8 X 50 yards					
	Control			Creatine		
	T1	T2	T3	T1	T2	T3
Heart rate (beats min ⁻¹)	184 \pm 7	185 \pm 6	183 \pm 8	185 \pm 9	185 \pm 7	182 \pm 8
Capillary blood lactate (mmol l ⁻¹)	14.5 \pm 2.1	15.0 \pm 2.3	15.1 \pm 4.2	14.6 \pm 2.2	15.0 \pm 2.1	14.4 \pm 2.2
Venous blood lactate (mmol l ⁻¹)	14.3 \pm 2.4	14.9 \pm 2.7	15.4 \pm 4.6	14.8 \pm 2.3	15.1 \pm 2.3	15.5 \pm 2.0
Ammonia (μ mol l ⁻¹)	200.7 \pm 56.0	193.7 \pm 67.8	213.7 \pm 94.9	226.7 \pm 48.7	213.5 \pm 63.9	192.9 \pm 85.3
Blood pH	7.10 \pm 0.09	7.07 \pm 0.10	7.07 \pm 0.11	7.07 \pm 0.06	7.07 \pm 0.09	7.06 \pm 0.04
Haemoglobin (g dl ⁻¹)	15.1 \pm 1.0	15.3 \pm 0.9	15.7 \pm 2.2	15.2 \pm 1.3	15.3 \pm 1.2	15.8 \pm 1.4
Haematocrit (%)	46.1 \pm 2.6	47.2 \pm 2.1	47.4 \pm 2.1	46.8 \pm 3.6	47.5 \pm 3.1	47.8 \pm 4.1
Plasma Volume (% change)	-2.3 \pm 2.0	-4.3 \pm 2.4	-5.9 \pm 5.0	-3.2 \pm 1.9	-4.6 \pm 2.0	-6.9 \pm 4.6

9.3.6 Growth Hormone response

Serum growth hormone concentrations (Figure 9.6) increased from rest to post-exercise in all tests ($P<0.01$). In T1, T2 and T3, post-exercise values reached 28.95 ± 18.27 and

$30.42 \pm 14.60 \mu\text{g l}^{-1}$; $14.24 \pm 7.32 \mu\text{g l}^{-1}$ and 21.48 ± 13.96 ; 22.63 ± 17.75 and $23.76 \pm 16.22 \mu\text{g l}^{-1}$ for Cr and control groups, respectively (test x time interaction effect $P < 0.05$; Figure 9.6).

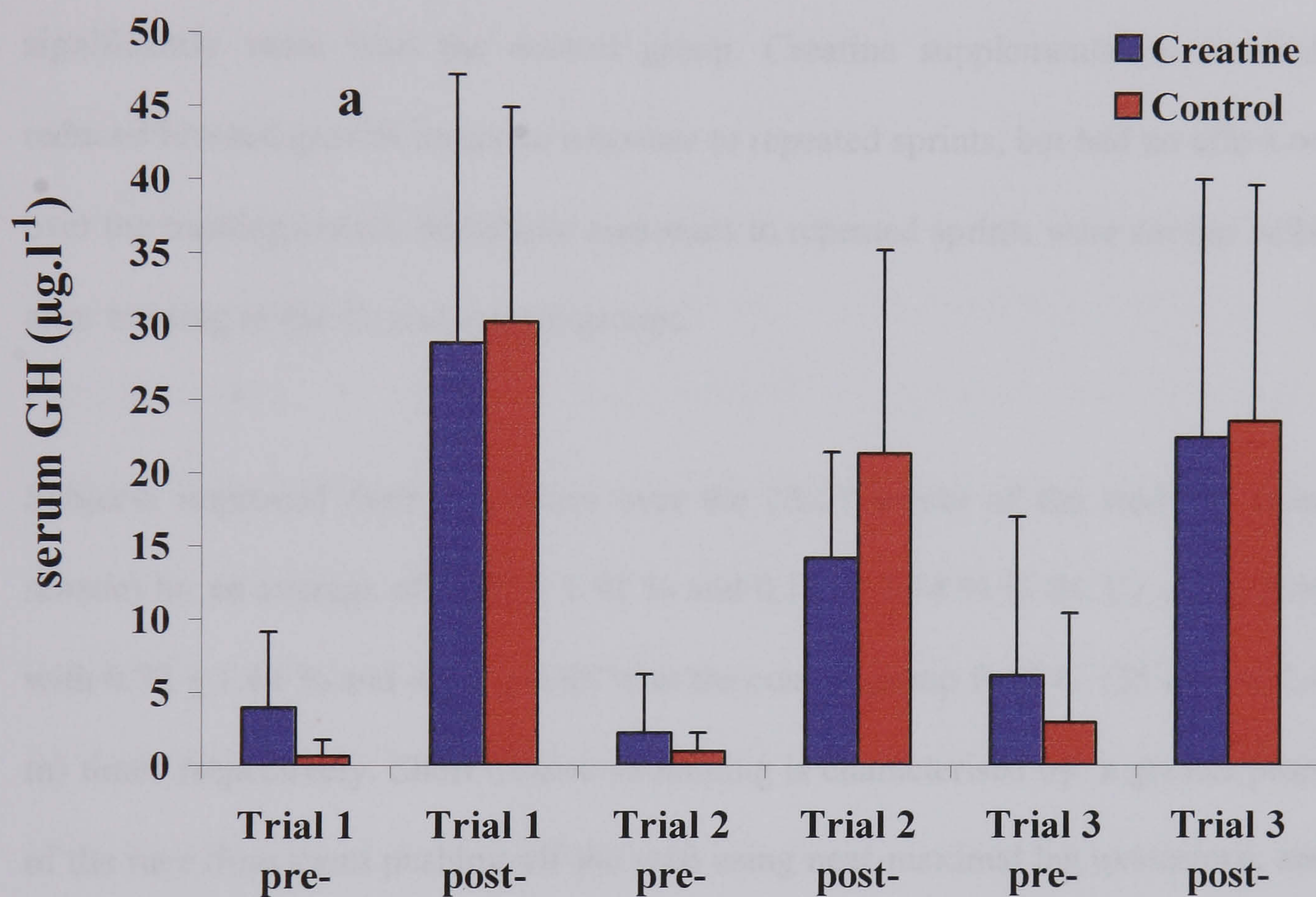


Figure 9.6 Human growth hormone responses to 8 x 50 yards sprints pre- and post-creatine supplementation and post-training for Cr and control groups (Cr $n = 7$; control $n = 8$; mean \pm S.D.).

a $P < 0.05$ trial 1 vs. trial 2 and 3.

9.4 Discussion

The main finding of the present study was that 22-27 weeks oral creatine supplementation during training does not appear to improve competitive swimming performance in elite competitors. Subjects improved their repeated sprint performance after an initial loading phase (similar to that reported in chapter VIII), but those who continued to maintain Cr levels failed to improve their competitive performances significantly more than the control group. Creatine supplementation resulted in a reduced/blunted growth hormone response to repeated sprints, but had no effect on hGH over the training period. Metabolic responses to repeated sprints were similar before and after training in the Cr and control groups.

Subjects improved their best times over the 22-27 weeks of the study (a swimming season) by an average of $1.90 \pm 1.91 \%$ and $0.14 \pm 1.14 \%$ in the Cr group compared with $0.72 \pm 1.64 \%$ and $-0.49 \pm 0.95 \%$ in the control group for S.C. (25 m) and L.C. (50 m) times respectively. Short Course swimming is characterised by a greater proportion of the race time spent pushing off the wall using near-maximal leg extensions, and may have been more familiar to this group of swimmers who train in this length of pool. These improvements appear small, but reflect the difficulty in elite swimmers to continually improve as they reach their own maximal performance levels (Pyne et al 2001).

The size of changes in competitive performance indicates that any differences between groups that might have been expected in this study could have been too small to detect in the statistical analysis. It is likely that the training *per se* will lead to greater

improvements than simply supplementing with creatine, and that these effects will disguise more subtle changes between groups in this study. There were large inter-individual differences (as indicated by the standard deviations) that were larger than the mean values recorded.

Studies involving creatine supplementation continue to demonstrate differing responses to performance under different exercise modalities, intensities, durations and contraction types (see the monograph by Williams et al., 2000). One of the reasons attributed to this inconsistency of findings relates to the individual variation in increasing skeletal muscle total creatine levels (TCr) following creatine supplementation. These have been termed 'responders' and 'non-responders' (Greenhaff et al., 1994; Greenhaff et al., 1996; Snow, 1997) and appear to be dependant on the initial levels of TCr in the muscle. Maximal total intracellular creatine concentration of $\sim 150\text{-}160\text{ mmol kg dm}^{-1}$ (Clark, 1997; Greenhaff, 1995; Harris et al., 1992) appears to be the upper limit in humans and the ability to improve performance following creatine supplementation is strongly related to those with lower initial levels who increase skeletal muscle TCr significantly.

By the same token, those who have naturally high levels of TCr in the muscle are unlikely to demonstrate further increases (i.e. there is a ceiling to or saturation point beyond which TCr cannot rise). In this group of elite swimmers, subjects appeared to 'respond' to supplementation (T1 to T2), but not to additional creatine supplementation during 22-27 weeks training. It is possible that subjects in the control group retained the high level of TCr obtained during the initial 'loading' phase for some time during the training period. There was a trend towards larger improvements in the Cr group,

although caution should be applied to these results as a whole as there was no significant interaction effect ($P = 0.12$).

A further confounding variable in this study relates to the wash-out time for the creatine. All subjects initially performed a normal Cr loading regimen (20g day^{-1} for 5 days) before the training period. Hultman et al., (1996) suggest that following supplementation, subjects' muscle Cr levels decline gradually to pre-supplementation levels in 30 days. Lemon (1995) however suggests that 35 days following Cr supplementation some subjects may still have elevated muscle TCr levels. This may certainly be the case if a high meat/fish diet is consumed following the supplementation period (although this was not monitored in the present study). If some of the subjects in the control group in this study had elevated muscle TCr levels for 4-8 weeks following the supplementation, then they will have received the same benefits as the Cr group.

The hypothesis set at the beginning of the study suggested that Cr supplementation during training would lead to improved performances by allowing subjects to train at higher intensities, for longer and with shorter recovery (Theodorou et al., 1999). In essence Cr supplementation would improve subject's ability to undertake a greater volume and intensity of training, allowing better adaptation and therefore improve competitive performance. In this study, it is likely that swimmers in both groups trained at a similar level throughout the season. A positive effect of supplementing with creatine may have been seen if the Cr group had also trained at a higher intensity than the control group, suggesting that taking creatine on its own has little effect. The present study was conducted in a double-blind fashion with neither experimenter nor swimmers aware of the group they were in. To suggest to the coach that some swimmers (Cr

group) should train harder and for longer than other (control group), would not have been practical, realistic or allow the study to retain the tight control using a double-blind protocol. Perhaps a study in the future may answer the question of whether Cr and more training (as opposed to just supplementing with Cr) will demonstrate greater performance improvements than normal.

Analysis of the speed-lactate test (5 x 200 yards endurance test) showed a mixed response in performance to the exercise period. The V4 was used as a reference point to indicate endurance fitness during the training phase. This metabolic indict has previously been used to describe seasonal changes during competitive swimming periods (Sharp et al., 1984) and is strongly correlated to performance in endurance swimming events (Bonifazi et al., 1993; Ribiero et al., 1990). It was expected that most subjects would increased their V4 during the course of the season and whilst some subjects improved, many others did not (Cr $+1.54 \pm 5.56\%$, control $-2.33 \pm 4.78\%$). Although every effort was made to test swimmers as soon as possible after the main competition, some swimmers may have spent 3-4 weeks in a state of reduced training prior to their main event followed by little or no training for 7-10 days. This may have resulted in a reduction in endurance capacity (Costill et al., 1985a; Costill et al., 1985c; Neufer et al., 1987).

Growth hormone values measured were within the range of other studies with mean values slightly higher than those reported by Pullinen et al., (2001) and Stokes (2002) ($\sim 15\text{-}25\mu\text{g l}^{-1}$), but lower than male sprint trained athletes reported by Nevill et al., (1996b; $\sim 35\text{-}50\mu\text{g l}^{-1}$). The hGH response showed large inter-individual differences (as

illustrated by the large standard deviations) with ranges of $\sim 5\text{--}52\mu\text{g l}^{-1}$, $\sim 6\text{--}42\mu\text{g l}^{-1}$ and $\sim 2\text{--}55\mu\text{g l}^{-1}$ in the pre- and post-supplementation, and post-training tests. This range in values was very similar in both male and female swimmers and did not appear to be related to sprint or endurance specialisation. This is at odds with previous reports (Nevill et al., 1996b) although many of the swimmers would fall into a mid-category in between those proposed by Nevill and associates, and also sprint swimmers tend to undertake relatively large amounts of endurance training.

Previous studies have demonstrated an extremely varied hGH release into the blood with a large range of peak and time to peak values in addition to total release (Nevill et al., 1996b; Raynaud et al., 1983; Stokes 2001). It is unfortunate that only one blood sample was taken ~ 1 min post-exercise as it is likely that this would have slightly under-estimated the hGH response to repeated sprint swimming with peak values usually observed between 15 and 40 min post-exercise (Nevill et al., 1996; Raynaud et al., 1981; Stokes 2002). Although the post-exercise hGH value was recorded at +1 min, the value also represents a time point ~ 13 min following the first repetition. The hGH values therefore probably were close to, but slightly below the expected peaks.

Opinion differs between researchers on the effect of training on the growth hormone response to exercise. Studies have reported increases (Bonifazi et al., 1998; McCall et al., 1999), decreases (Bloom et al., 1976; Weltman et al., 1997, Stokes et al., 2000) and no change (Kraemer et al., 1990) in post-exercise concentrations of hGH following training. The present study demonstrated a blunted hGH response to repeated sprints following one week of Cr supplementation. Whether this was due to a learning effect of

the test, the training undertaken during the week, the Cr supplementation or a combination of these factors is not clear.

Schedel et al., (2000) observed a hGH response following a single 20 g ingestion of Cr, without exercise, which they suggest is due to an increased intracellular ADP concentration (Wallimann et al., 1992). A hGH response is observed following exercise and linked to the adenylate kinase (AK) reaction and the activation of creatine kinase (CK; Schedel et al., 2000), although similar ammonia values (despite different hGH concentrations) before and after supplementation in the present study do not support this. However, the hGH response following Cr supplementation (Schedel et al., 2000) was considerably smaller than those recorded following sprint exercise and that observed in the present study. What is not clear is whether a second (or repeated) 20 g dose (s) demonstrates a similar release of hGH or if the response is attenuated as observed in sprint exercise (Stokes, 2002).

Previous studies have demonstrated a blunted response to the exercise-induced hGH response following training (Stokes, 1999; Weltman et al., 1997). The time course suggested by Weltman et al., 1997 for this change to occur was within the first 3 weeks. The results of this study suggest that that time-course may be shorter (i.e. 1 week), although following a longer period of training (22-27 weeks), the hGH response reverted back to a similar level to that recorded in the pre-supplementation trial. This longer-term enhancement of the hGH response is consistent with results observed by Bonifazi et al., (1998) following 18 weeks of mixed training in elite swimmers. In fact if T2 is taken as the base line, the results of this study are almost identical to those of Bonifazi et al. (1998) following training.

The hGH values showed a pattern of response within individuals over the 3-tests, and subjects could have been categorised as 'low-responders' (6), 'moderate-responders' (4) and 'high-responders' (5; Schedel et al., 2000). Four of the 'high-responders' were female and taking oral contraceptives (O.C.), although one further female, also taking O.C., was in the 'low-responders' group. Higher resting hGH values have been found previously in females who were taking OC (Bonnen et al., 1991). Further research is needed to determine the impact of O.C. on the hGH response to exercise and training.

In summary, the findings of the present study confirms that oral creatine supplementation (20 g day^{-1} for 5 days) improves repeated sprint swimming performance (as observed in chapter VIII), but that an additional supplementation protocol over 22-27 weeks of training does not appear to enhance the competitive performance of elite swimmers more than training on its own. The hGH response to repeated sprint swimming is blunted over the first week during creatine supplementation, but reverts back to pre-supplementation levels by the end of the competitive (22-27 weeks) season.

CHAPTER X

GENERAL DISCUSSION

10.1 Introduction

The purpose of this chapter is to collate the findings of the six experimental studies included in the thesis, to explain the physiological basis for the results and to suggest how this information assists in an understanding of the training and supplementation strategies for success in competitive swimming. The chapter summarises the findings of each experimental study and examines the physiological mechanisms behind the performance and metabolic results.

10.2 Main findings

- The metabolic responses of males and females to 50 yards and 8 x 50 yards sprint swimming were described in chapter IV. Peak blood lactate (male: 18.7 mmol l^{-1} and female: 14.4 mmol l^{-1}) and ammonia (male: $232.0 \text{ } \mu\text{mol l}^{-1}$ and female: $154.3 \text{ } \mu\text{mol l}^{-1}$) values following repeated swimming (8 x 50 yards) were almost double those measured during a single 50 yards sprint and were significantly higher in males than females.
- Chapter V introduces a tethered swimming mechanism for measurement of force output and collection of expired air for the determination of oxygen uptake during sprint swimming. A maximal accumulated oxygen deficit (MAOD) test enabled estimation of the anaerobic energy contribution to single and repeated swimming

sprints. Peak and mean force production during 30 s maximal tethered swimming was ~186 N, and 139 N respectively, although force set at 95% mean force output resulted in fatigue (indicated by an inability to maintain the requested force output) during 4 x 30 s semi-tethered bouts. Estimated anaerobic energy contribution was ~66% for the 30 s FTS sprint and ~74%, ~53%, ~51% and ~48% for bouts 1 to 4 during the 4 x 30 s STS test (bout 1 vs. bouts 2-4, $P < 0.01$), much higher than previously thought for 50 yards races and training sets commonly prescribed to improve sprint performance.

- The physiological responses of controlled frequency breathing to maximal intensity swimming performance were examined in Chapter VI. Three maximal 55 s tethered swimming trials were compared: breathing with a self-selected pattern, every 4th and every 10th arm stroke. There were no differences between trials for performance (peak or mean force output, fatigue index or stroke rate), blood lactate and heart rate (N.S.). Minute ventilation, expired air oxygen concentration, oxygen uptake and carbon dioxide production, the respiratory exchange ratio and the ventilatory equivalent for oxygen were all higher ($P < 0.05$), whereas expired air carbon dioxide concentration was lower in BSS than B10 ($P < 0.05$). Estimated aerobic contribution to 55 s tethered swimming was ~30 – 40%, although a decrease in $\dot{V}O_2$ observed during B10 tends to suggest a decrease in oxidative metabolic processes.
- Chapter VII describes the effect of active and passive recovery on performance and metabolism during repeated sprint training sets commonly used by swimming coaches. A first set of 4 x 30 s fatiguing tethered swimming was succeeded by one of three 4 x 50 yards sets with active or passive recovery between sets and/or

repetitions. The decline in performance during Set B was less marked when swimmers recovered actively between sets, but not between repetitions. Combined times under these conditions were ~2.5% faster than those in the trial using active recovery between sets and repetitions (N.S.). Blood lactate after Set B was higher and blood pH was lower ($P < 0.05$) when recovery was passive throughout ($18.3 \pm 1.3 \text{ mmol l}^{-1}$, 7.12 ± 0.11) or active between sets, but passive between repetitions ($17.6 \pm 1.2 \text{ mmol l}^{-1}$, 7.14 ± 0.11) compared with active recovery throughout ($14.1 \pm 1.6 \text{ mmol l}^{-1}$, 7.23 ± 0.10).

- The effects of 9 g day^{-1} oral creatine supplementation on 50 yards and 8 x 50 yards sprint swimming performance were studied in chapter VIII. Mean single sprint times were unchanged as a result of supplementation, but performance was improved as a result of creatine supplementation; percentage decline in performance was reduced (Cr $15.7 \pm 4.3\%$ vs. $10.0 \pm 2.5\%$; Control $12.7 \pm 5.7\%$ vs. $11.0 \pm 5.5\%$) and total swimming time was ~4 s faster ($P < 0.05$, group x trial interaction). Blood lactate, pH and plasma ammonia were similar before and after creatine supplementation (N.S.). A ~67% (~26 g) retention of the administered creatine estimated by urinary analysis provided evidence to support the supplementation regimen as the factor responsible for the performance improvements
- The effect of creatine supplementation on training for competition in elite swimmers was examined in chapter IX. Mean competition times changed by $+1.90 \pm 1.91\%$ for S.C. and $+0.14 \pm 1.14\%$ for L.C. and $+0.72 \pm 1.64\%$ for S.C. and $-0.59 \pm 0.82\%$ for L.C. in the Cr and control groups respectively ($P < 0.05$, S.C. improvements; L.C. changes and group x trial interactions, N.S.). Despite a

similar improvement in repeated sprint performance to that observed in chapter VIII following creatine supplementation, no further differences were found in race performance when supplementing with 3g creatine day⁻¹ for 22-27 weeks, although large inter-individual differences in race performance changes and training were observed. No differences between Cr or control maintenance groups were found in blood metabolites, although the hGH response to repeated sprints was blunted after creatine supplementation and training.

10.3 Sex differences between single and repeated sprint swimming

The physiological and metabolic responses (heart rate, blood lactate and pH, plasma ammonia, and changes in plasma volume) to maximal single and repeated swimming reported in chapter IV represent the most comprehensive description so far of male and female swimmers. Performance differences between the sexes observed in chapter IV during 50 yards sprinting were ~14%, similar to the difference in blood lactate (~13% higher in males than females), but lower than the difference in plasma ammonia (~47% higher in males than females). These differences were matched in performance during 8 x 50 yards (~14% to ~11% higher in males between repetitions one to eight), although blood metabolites changed to a different degree. Blood lactate concentrations were ~29% higher in males than females, whereas plasma ammonia differences were similar to the single sprint (~51% higher in males). Blood pH values were similar in males and females during a single 50 yards sprint, but mean values were lower ($P < 0.01$) in males than females following 8 x 50 yards sprints.

Differences in body size and composition will impact both on swimming performance and muscle metabolism because of resultant differences in the size of the available

muscle mass during sprint swimming. Sprint swimming speed is highly correlated to muscle power ($r = 0.86$ to $r = 0.94$ for 100 to 25 yards performance; Troup et al., 1980; Sharp et al., 1982). Mean body mass and height in 1992 U.S.A. Olympic level swimmers were 87.1 and 64.4 kg and 190.5 and 172.7 cm for male and female swimmers respectively, compared with 76.3 and 63.5 kg and 183.0 and 170.1 cm in the present study. Differences between the two studies are much greater in male than female swimmers. Mean percentage body fat reported by Troup et al. (1994) were ~70% higher in females than male swimmers (9.4% vs. 15.9%). Differences have previously been reported in $\dot{V}O_2\text{max}$ (male: 5.0 l min^{-1} , female: 3.4 l min^{-1} ; Holmer et al., 1974) which is closely related to body size, and based on percentage body fat; females will have proportionately less as well as lower absolute muscle mass. In chapter IV, female body mass was ~20% lower than male body mass which is likely to account for a large proportion of the performance differences (~14% to ~11%) between male and female swimmers during single and repeated sprints. When blood metabolites were reported relative to body mass the difference between the sexes reduced markedly from ~29% to ~7% for blood lactate and ~50% to ~24% for plasma ammonia.

In chapters IV, VIII and IX, 8 x 50 yards was used as the repeated sprint swimming test. Combined metabolite data from the 3 chapters with the number of test occasions for both male and female subjects are shown in table 10.1. Care must be taken in drawing conclusions from this grouped data as male and females were not always tested during the same study (as indicated by group numbers).

Table 10.1 Combined metabolic data for repeated sprints used throughout the thesis

	Sex	Number	Mean \pm S.D.	Range
Blood pH	M	68	7.04 \pm 0.08	6.89 – 7.19
	F	34	7.06 \pm 0.07	6.90 – 7.22
Ammonia ($\mu\text{mol l}^{-1}$)	M	65	212.5 \pm 67.3	389.4 – 79.6
	F	34	173.8 \pm 57.5	331.0 – 94.0
Blood lactate (mmol l^{-1})	M	65	16.1 \pm 2.7	22.90 – 10.7
	F	34	14.4 \pm 2.2	19.4 – 11.1

M: Male; F: Female

The large inter-individual differences (reflected in the range of values) perhaps demonstrate that factors other than sex determines the extent of the metabolic responses to sprint swimming. It is likely for instance that competitive standard and sprint- or endurance-training background will impact on both performance and metabolic responses to repeated sprint swimming. Body size and composition variations within each sex may have a bearing on sprint performance during competitive swimming. Thus, training strategies that enable a female to decrease body fat and increase muscle mass are likely to have a positive effect on sprint performance if all other factors are equal. The magnitude of the metabolic responses (table 10.1) following repeated sprints suggest that the typical training set of 8 x 50 yards used in this study may be more specific training for longer distances such as 100 and 200 m swimming races.

10.4 Energy contribution to single and repeated sprint swimming

Knowledge of the aerobic/anaerobic energy contribution to single and repeated sprint swimming is of importance to scientists and coaches in constructing training programmes for success in sprint races. Previous estimates based on traditional ‘oxygen

debt' measurements have been discredited and over-estimate the anaerobic contribution to sprint swimming (see table 2.6). Recent estimates of anaerobic energy contribution to 50 m and 100 m sprint swimming performance have been used using mathematical/theoretical models and oxygen deficit testing during flume swimming. The information presented in chapters V and VI is the first estimate (using oxygen deficit) of the relative energy contribution to repeated sprint swimming similar to that used during training, and the first estimates of the aerobic/anaerobic contribution during 30 s and 55 s single-sprint, maximal tethered swimming.

Estimated aerobic energy contribution was ~34% for the 30 s FTS sprint and 26%, 47%, 49%, 52% for bouts 1 to 4 during the 4 x 30 s STS test, much higher than previously thought for 50 m/yards races and training sets commonly prescribed to improve sprint performance (2%: Maglischo, 1993). Aerobic energy contribution to 55 s maximal tethered swimming, estimated in chapter VI at ~60 – 69% was also higher than previously suggested (~43 – 54%: Capelli et al., 1998; Morton and Gastin, 1997; Troup and Trappe, 1994). The most likely explanation for higher aerobic contribution during the 55 s test was the restriction in breathing and therefore a more paced than 'all-out' effort allowing greater contribution from aerobic metabolism. More research is needed to confirm the relative energy contribution to sprint swimming, and in particular repeated sprints such as those used during training. It is likely that aerobic energy supply plays a greater role than previously thought and that this role is progressively extended during repeated sprint swimming.

10.5 Training strategies in preparation for sprint swimming

10.5.1 Breathing frequency

In chapter VI it was reported that a significant reduction in breathing frequency and subsequent changes in respiratory metabolism, had no effect on 55 s maximal tethered swimming performance. This study is the first to our knowledge that has examined the physiological effects of controlled frequency breathing on maximal sprint swimming.

Breathing frequency was reduced in the B10 trial, resulting in lower \dot{V}_E , $\dot{V}O_2$, $\dot{V}CO_2$ and RER, although no differences in blood lactate or heart rate were observed between trials. Therefore, the impact of the breathing action under fatigue conditions during sprint swimming may compromise stroke mechanics and consequently performance, although there is no evidence from this study that reducing the number of breaths will enhance performance. This may be because the possible benefits, in terms of improved swimming economy, are offset by changes in metabolism that have the potential to inhibit performance. Thus, any possible performance benefit of CFB swimming may be individually specific, dependent on technique and it is not likely to be of widespread benefit.

10.5.2 Recovery methods

Active recovery and its effect on both blood lactate removal (Cazorla et al., 1983; McMaster et al., 1989; Raeburn and Mackinnon, 1990) and subsequent performance (Felix et al., 1997; Siebers and McMurray, 1981) have been studied in swimming following maximal race performance. The information in chapter VII is the first consideration of both the metabolic and performance responses to active and passive recovery between sprint bouts and sets commonly used in training for sprint swimming.

It is likely that the recovery between sets (5 min) was enhanced by active swimming (as opposed to being passive), perhaps due to a faster restoration of acid balance within the muscle cell, a faster resynthesis of PCr, or an increased aerobic metabolism. When active recovery was performed between sets and between sprints, it had a detrimental effect on swimming performance. This paradoxical effect may indicate that the intensity of the active recovery was too high (%60 of 100 m P.B. pace), or the duration (2 min) too low. Perhaps a period of passive rest initially followed by active recovery is needed for optimal recovery of performance.

Lactate was lower during the AA trial, which suggests that blood lactate removal was faster. However the present study demonstrated that blood lactate is a poor predictor of subsequent sprint swimming performance. It is likely that other factors (such as lower muscle H^+ and higher pre-sprint PCr) will determine success in swimming repeats. Active recovery may still be an important means of enhancing the speed of recovery and delaying fatigue during training (as results of the AP trial showed), but further research is needed to establish the most efficient duration, intensity and timing of active recovery in order to improve repeated sprint performance in subsequent exercise.

10.6 Creatine supplementation and sprint swimming

The effects of creatine supplementation in swimming are investigated in chapters VIII and IX. The study in chapter VIII is the first to report the effects of creatine supplementation on repeated sprint swimming, whilst the study reported in chapter IX is the only long term (longer than 10 weeks) training study that has reported both performance, metabolic and hormonal effects of supplementing with creatine during training for competition in elite swimmers. In both studies, improvements were shown

during repeated, but not single, sprint swimming performance (see Figures 8.1 and 9.3). Evidence for creatine supplementation as the cause of performance improvements is provided from the urinary analysis results which showed ~67% (~26 g) retention of the administered creatine, a lower quantity, but higher proportion than that reported by Rossiter et al. (1996) using higher supplementation quantities (~70 – 100 g over 5 days). It is likely that performance improvements are a result of increases in muscle creatine content leading to improved PCr resynthesis and thus higher pre-exercise muscle PCr content towards the end of the 8 x 50 yards set.

Following the results of chapter VIII, it was hypothesised that the benefit in repeated sprint performance might lead to an enhanced training-induced performance improvement. However, following a loading phase, an additional supplementation protocol over 22-27 weeks of training did not appear to enhance the competitive performance of elite swimmers more than training on its own. Large inter-individual differences in training and competition results masked any statistical differences between Cr and control training groups, which may suggest that any added benefit of creatine maintenance may be dependant on an individual's natural muscle Cr concentration. It is possible that creatine supplementation in addition to higher intensity training may result in improved performances, although a study that attempt to determine this would be difficult to control due to a lack of a 'double-blind' approach. Results from these studies suggest that creatine availability and energy supply are not a limitation of sprint performance in swimming, but limit performance in repeated sprint swimming.

10.7 Recommendations

In summary, the results of this thesis may direct scientists and coaches in future by suggesting that the following advice is adopted:

- Swimmers (particularly females) will benefit from larger muscle mass and reduced body fat, assuming that all other factors remain unchanged.
- Repeated sprint training sets such as 8 x 50 yards may be more specific physiological conditioning for 200 m events than 50 m.
- Aerobic energy contribution to single (~33%) and repeated (~26%, ~47%, ~49% and ~52%) sprint swimming is much higher than previously thought.
- Restricting breathing frequency appears not to effect sprint performance, but changes in respiratory function suggest that training to cope with hypercapnia may allow the mechanical benefits of not having to turn the head to breath.
- Active recovery is a useful tool in improving subsequent performance, but only if the timing and intensity of active recovery is correctly prescribed.
- Creatine supplementation is likely to improve repeated, but not single sprint swimming performance, although it is unlikely that longer term creatine maintenance will enhance training for performance, any more than a five day loading dose every 4 months.
- The only possible way for creatine supplementation to enhance race performance is by enhancing the adaptation to training, and therefore, creatine and extra training should be undertaken.

10.8 Future research

It is surprising that despite the wealth of scientific studies (685 in peer reviewed journals by 1994; Clarys, 1996), none have considered direct measurement of muscle metabolites following sprint swimming. It is likely that results would be very similar to those observed following sprint cycling or running (see chapter II), but differences may occur due to the specific musculature involved and the stroke technique. The maximal mean stroke rate in elite swimmers during sprinting is ~ 70 stroke cycles min^{-1} (see figure 2.8), although this may rise to ~ 90 stroke cycles min^{-1} at different stages during a race. Even so, this value is less than half that experienced during the first few seconds in sprint cycling (Nevill et al., 1996a) and much less than the $140 \text{ revs min}^{-1}$ used in constant paced sprints (Jones et al., 1985; McCartney et al., 1983). It is possible that this reduction in limb movement speed may affect the metabolic responses to sprint swimming, demonstrating a pacing effect and perhaps lower fatigue (Cherry et al., 1997a). In addition, we may speculate that the short intra-cyclical periods of recovery during the recovery phase during swimming may assist in the restoration of ionic imbalances and therefore rapid recovery of power output (Cherry et al., 1998).

Differences in the metabolic responses to sprint swimming compared to cycling or running may involve the total muscle mass and its recruitment pattern. Clarys (1986; 1988) has conducted comprehensive electromyography (EMG) research into muscle action during the swimming action and other sports. The largest electrical activity from muscles occurs in the muscles of the trunk (latissimus dorsi and abdominals) and upper leg (gluteus maximus, quadriceps and hamstrings). The arm-stroke is the dominant action in creating propulsion and clearly reflected in the powerful extension and adduction of the shoulder joint by the latissimus dorsi. However, despite the significance

of the upper leg and abdominal muscle activation, it is unlikely that these muscles will limit performance and may act more in assisting technique and balance as opposed to contributing to propulsion. Is there a metabolic ‘sparing effect’ with the majority of maximal muscle contraction initiated by the (relatively small) upper body muscles with lower activity from the (large) muscle mass of the trunk and lower body? Conversely, does the whole-body activation of muscle during sprint swimming result in a greater metabolic effect? Future research combining EMG and metabolic measurements may help to clarify the extent of the physiological demands on swimmers and the mechanisms behind the limitations to sprint swimming performance.

There remains some doubt as to the specific reasons for performance differences between males and females. Body size and composition are key contributors to the differences, but further studies using both male and female subjects of similar standard and sprint/endurance background would be welcome in identifying all sex differences in physiology. Several studies have considered single performance swimming, but a paucity of information exists regarding the physiological responses of swimmers to repeated sprinting, commonly practiced in training. The impact of such training on competition has been restricted by the short length of interventions or standard of swimmer used (Costill et al., 1985c; Neufer et al., 1987), or a lack of detailed physiological measurements made over longer term training periods (Chatard and Mujika, 1998; Mujika et al., 1998; Pelayo et al., 1996). Future studies may include more detailed metabolic and hormonal responses to training, over longer periods (e.g. 2 – 3 years) under controlled conditions, although in most cases, the level of swimmer and the degree of study control are inversely related.

Different training strategies have been used in attempts to stress particular physiological mechanisms in an attempt to push back the boundaries of competitive swimming performance. Chapters VI and VII describe two such strategies (breath holding and the types of recovery), but perhaps future research may determine the true benefits and mechanisms behind different types of land conditioning for swimming. In addition, perhaps land training to a greater degree (e.g. 6-8 sessions per week as opposed to 3; Tanaka et al., 1993) may demonstrate greater performance improvements. A recent rise in training programmes that use combinations of hand paddles and fins (flippers) suggests that the study of training equipment use seems warranted.

Many aspects of creatine supplementation and swimming have been studied, although studies have yet to determine what the long-term impact of supplementation regimens have on health. Continuation of two medium-term studies (5 – 8 years; Francaux and Poortmans, 1999; Schilling et al., 2001) may help to answer this question. Perhaps the key question to be answered for the swimmer and coach would involve the individual response to supplementation, but at present this is only possible through muscle biopsy.

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APPENDICIES

APPENDIX A

Example Ethical committee proposal

APPENDIX B

Blood metabolite assays

APPENDIX C

Serum human growth hormone assay

RESEARCH PROPOSAL FOR HUMAN BIOLOGICAL INVESTIGATIONS

It has been demonstrated that oral creatine supplementation can increase resting concentrations of creatine and phosphocreatine in skeletal muscle (Harris et al., 1992) and improve performance through an increased recovery rate during intermittent high intensity exercise (Balsom et al., 1993; Greenhaff et al., 1993). It would appear that these improvements are not matched during single sprint exercise (Balsom et al., 1993; Cooke et al., 1995; Greenhaff et al., 1993). Recent studies with elite competitive swimmers show similar findings with no improvements in performance in 25m, 50m and 100m race distances (Burke et al., 1996, Mujika et al., 1996). As the benefit from creatine supplementation involves faster recovery and improved performance in the latter repetitions of a multiple sprint set (Peyrebrune et al., 1998), the relevance to swimming would appear to be in enhancing sprint training for competition. An enhanced gain in high intensity intermittent exercise capacity in isokinetic arm flexions after a 10 week period of strength training with creatine supplementation has been shown (Vandenberghe et al., 1996). A recent study has found no additional differences in swimmers who have followed a creatine 'loading' period with either a low dosage 'maintenance' period or no supplementation (Theodorou et al., 1999). This study did not investigate the physiological responses of creatine supplementation to training and was conducted over a relatively short period (8 weeks). To date, there are no longer term (>10 weeks) training studies that have investigated the physiological effects of oral creatine supplementation on elite performers training in a sports-specific environment. The purpose of this study therefore, is to determine the effects of oral creatine supplementation on 28 weeks of training for competition in elite swimmers. Test sessions will take place before and after a 5 day supplementation period, and after a 27 week training period which will culminate at the GB World Championship Trials. The supplementation period will involve a high dosage 'loading' phase and a low dosage 'maintenance' phase (Hultman et al., 1996; Vandenberghe et al., 1996). Testing sessions will include a speed-lactate swim test and sprint training set comprising 8 X 50 yards. Subjects will be randomly assigned to either a creatine supplementation or placebo (glucose) group and have finger-tip and venous (8 x 50 test only) blood samples taken.

(iv) Names and status of investigators:

Dr. Mary E. Nevill (Lecturer, Dept. of P.E., Sports Science and Recreation Management, Loughborough University)

Mr. Michael C. Peyrebrune (PhD Student, Dept. of P.E., Sports Science and Recreation Management, Loughborough University)

(v) Subjects (see Section 6.3e):

Thirty-two healthy male subjects will be recruited by notice from the student swimming team at Loughborough University and if necessary, from local swimming clubs. All subjects will be currently involved in a regular training (minimum 7 sessions/week) and competitive programme and have swum at senior county level or above. They will have had previous experience of the maximal and sub-maximal sprint swimming training sets, as well as the training content used in this study.

Prior to receiving their informed consent, subjects will be fully informed of the demands of the experiment (in "layman's terms") and the possible risks and discomforts that would be associated with their participation.

Subjects will also have the opportunity to ask for further information and for clarification of the procedures involved. In addition, they will be required to complete a questionnaire on their medical history (see attached) in the presence of an experimenter (to provide clarification and assistance).

(vi) Location (any special facilities to be used):

The experimental work will be carried out in the Sports Science Research Laboratories and Sports Hall swimming pool in the Department of Physical Education, Sports Science and Recreation Management, at Loughborough University. Competition times will be recorded throughout their normal competitive season (October to March) and culminating in the World Championship Trials in Sheffield.

(vii) Duration (including demand on subject's time):

Subjects will be involved in five testing sessions: two speed-lactate and three 8 X 50 yards maximal sprint training sets. The duration of each test (including changing, blood sampling, warming up, the test itself and warming down) will be 70 min. Training and competition carried out throughout the study period will be approximately 16 hours per week and 6 X 3/4 days respectively. This involvement will match the swimmer's normal training and competition regime and is **not** additional time commitment demanded by the study. Time spent filling in the medical questionnaire and informed consent forms is included within the test timings.

(viii) Reasons for undertaking the study (e.g. contract, student research):

Ph.D. research project.

(ix) Methodology (a brief outline of research design):

A standardised warm up will be conducted in preparation for each test. Swimmers normally spend 20-30 minutes warming up during every training session. One of the following tests will then be conducted by each subject:

1. A speed-lactate swimming test before and after a 28 week training period.
2. A maximal sprint training set of 8 X 50 yards before and after a period of dietary supplementation (5 days) and after a 27 week period of training.

All repetitions during the 8 X 50 yards test will take place from a racing dive in the pool and will be timed. Anti-wave lane ropes will divide the lines as in competition to reduce unnatural water resistance. Subjects will be asked to exert maximum effort on each repetition, whilst maintaining a constant stroking technique.

During the five days prior to the second 8 X 50 yards test, all subjects will be required to consume the 'loading' supplement (4 X [5g creatine + 5g glucose day⁻¹] for 5 days), dissolving the powder in a warm, caffeine-free drink. Total intake of creatine by all subjects over this period will be 100 grammes.

Immediately following the second 8 X 50 yards test, subjects will be assigned (using a 'double-blind' protocol) to either a supplementary (3g creatine + 7g glucose day⁻¹) or placebo (10g glucose day⁻¹) group. Subjects will consume the powder as previously stated every day for a period of 27 weeks. Total intake of creatine by any subject over this period will be 567 grammes.

Competition data will be recorded electronically at all competitions between October 2000 and April 2001. All creatine used in the study will be purchased by the Department of Physical Education, Sports Science and Recreation Management, at Loughborough University.

(x) Procedures and measurements (for experimental and control subjects):

i) During the 'loading' phase, subjects will be provided with 20 pre-measured packets of powder (5g creatine + 5g glucose) and instructions to mix the powder in a warm, caffeine-free drink for immediate consumption with a meal at 9:00, 12:00, 15:00 and 18:00 hours each day during the 5 day supplementation period.

ii) During the 'maintenance' phase, subjects will be provided with 189 pre-measured packets of powder (either 3g creatine + 7g glucose or 10g glucose) and instructions to mix the powder in a warm, caffeine-free drink for immediate consumption with a meal at the same time each day during the training period.

(x) Procedures and measurements (continued):

- iii) On each visit to the laboratory, the volunteer's height and weight will be determined. Strict observance of an individual's privacy will be observed at all times during each measurement.
- iv) Heart rate will be monitored at rest and throughout all the exercise phases of the testing sessions by means of short-range telemetry (Sport Tester, PE 3000). This involves placing two self-adhesive electrodes on the subject's chest. A receiver will be vacuum-sealed in a waterproof plastic bag, and placed under the costume. Subjects will wear lycra vests specifically designed for use with the heart rate monitor which fit comfortably over the shoulder and ensure a constant contact between electrodes and skin.
- v) During the speed-lactate test, two 20 µl samples of blood will be taken pre- and 1 minute after each of the 5 repetitions from a small thumb-prick.
- vi) In the 8 X 50 yards protocol, two 20 µl samples of blood will be taken pre- and 1 and 3 minutes post-test from a small thumb-prick.
- vii) 10 ml venous blood samples will be taken from a needle at rest and one minute after cessation of exercise in the 8 X 50 test only. The needle will be inserted into a forearm vein by Dr. Mary E. Nevill while the subject is seated. No more than 20 ml of blood will be collected from each subject during a testing session.

(xi) Possible risks, discomforts and/or distress (see Section 6.3k):

There are no known or reported side effects from creatine supplementation at the doses (20 g of creatine per day for 5 days, or 3 g per day for 189 days) prescribed for this study (Poortmans and Francaux, 1999).

All procedures will be carried out in accordance with the Code of Practice for Workers having Contact with Body Fluids. This study requires volunteers to exercise maximally. Subjects will be encouraged to stop exercising at any time that the demands of the test become intolerable. The risks of injury are minimal as test sessions are well controlled with two investigators present at all times.

The investigators are, at all times, vigilant in their observations of volunteers performing under the prescribed experimental conditions, and are ready to end the test should the volunteer report, or even appear, unduly stressed.

In accordance with Departmental safety guidelines, there will be a qualified lifeguard present on the poolside at all testing sessions.

There is a risk that local bruising from intravenous blood sampling may occur.

(xii) Names of investigators and personal experience of proposed procedures and/or methodologies:

Finger-tip blood samples will be conducted by Mr. Michael Peyrebrune who is practiced in using this technique. Research students will also carry out other routine measurements.

(xiii) Details of any payments to be made to the subjects:

None

(xiv) Whether the investigators have any financial or other "interest" in the project:

None

(xv) Whether the University's insurers have indicated that they are content for the University's Public Liability Policy to apply to the proposed investigation (Committee use only):

(xvi) Whether insurance cover additional to (xv) has been arranged by the investigator (see Section 6.30):

No

(xvii) In the case of studies involving new drugs or radioisotopes, written approval for the study must be obtained from the appropriate national body and submitted with the protocol. State if applicable:

Not applicable

(xviii) Declaration

I have read the University's Code of Practice on Investigations on Human Subjects and completed this application.

Signature of applicant: _____

Signature of Head of Department: _____

Date: _____

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HEALTH SCREEN FOR STUDY VOLUNTEERS

Name

It is important that volunteers participating in research studies are currently in good health and have had no significant medical problems in the past. This is to ensure (i) their own continuing well-being and (ii) to avoid the possibility of individual health issues confounding study outcomes.

Please complete this brief questionnaire to confirm fitness to participate:

1. **At present**, do you have any health problem for which you are:
- (a) on medication, prescribed by a doctor or otherwise

Yes ☐ No ☐

(b) attending your doctor

Yes ☐ No ☐

(c) on a hospital waiting list

Yes ☐ No ☐
2. **In the past two years**, have you had any illness which require you to:
- (a) consult your doctor

Yes ☐ No ☐

(b) attend a hospital for an appointment

Yes ☐ No ☐

(c) be admitted to hospital

Yes ☐ No ☐
3. **In the past two years**, have you had any illness which require you to:
- (a) Convulsions/epilepsy

Yes ☐ No ☐

(b) Asthma

Yes ☐ No ☐

(c) Eczema

Yes ☐ No ☐

(d) Diabetes

Yes ☐ No ☐

(e) A blood disorder, e.g. Clotting problems

Yes ☐ No ☐

(f) Head injury

Yes ☐ No ☐

(g) Digestive problems

Yes ☐ No ☐

(h) Heart problems

Yes ☐ No ☐

(i) Problems with bones or joints

Yes ☐ No ☐

(j) Disturbance of balance/coordination

Yes ☐ No ☐

(k) Numbness in hands or feet

Yes ☐ No ☐

(l) Disturbance of vision

Yes ☐ No ☐

(m) Ear/hearing problems

Yes ☐ No ☐

(n) Thyroid problems, e.g. rapid loss or gain of weight

Yes ☐ No ☐

(o) Kidney or liver problems

Yes ☐ No ☐

(p) An allergic reaction, e.g. swelling, breathing difficulties

Yes ☐ No ☐

4. **Has any**, otherwise healthy, member of your family under
the age of 35 died suddenly, soon after exercise Yes ☐ No ☐

**If yes to any question, please describe briefly if you wish (e.g. to confirm if problem was/is short-lived, insignificant or well controlled
.....**

5. **Additional questions for female participants:**

(a) Have your periods started yet? Yes ☐ No ☐
If Yes, please answer (b) to (e)

(b) At what age did your periods start (as accurately as you can remember)?
Age: Years and Months

(c) Are your periods normal/regular? Yes ☐ No ☐

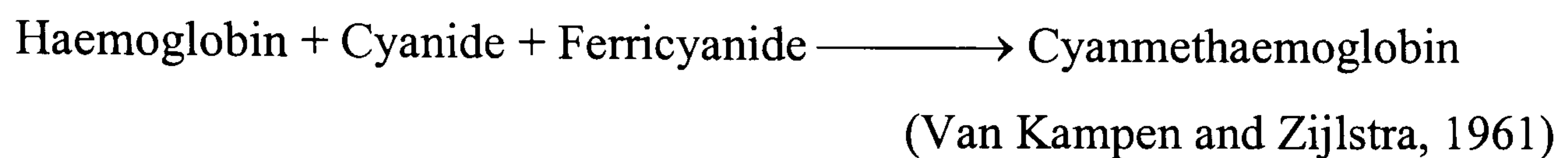
(d) Are you on “the pill”? Yes ☐ No ☐

(e) Could you be pregnant? Yes ☐ No ☐

Thank you for your cooperation!

Haemoglobin (Cyanmethaemoglobin Method)

Principle



Reaction mixture

The reaction mixture ('Drabkins') was made from a kit (Boehringer Mannheim) by diluting with distilled water (1000ml) and contained phosphate buffer, potassium cyanide, potassium ferricyanide and detergent. It was then stored in a brown bottle at between +15°C and +20°C.

Procedure

1. In duplicate, 20µl of whole blood was added to 5.0 ml of the reaction mixture in a plastic tube and mixed.
2. The tubes were left to stand for at least 5 min, but not longer than 24 hr. The exact time varied between runs but in most cases samples were analysed at the end of a trial and the time samples were left to stand did not exceed 6 hr.
3. The absorbance of the sample was measured using a spectrophotometer (Cecil Instruments) at a wavelength of 546 nm in a cuvette with a 1.0cm light path. The reaction mixture on its own was used as a blank in order to zero the spectrophotometer. Relative absorbance (A) (absorbance of sample – absorbance of blank) was therefore taken as the reading on the spectrophotometer.
4. The haemoglobin concentration of the sample was then calculated from the following equation:

$$\text{Haemoglobin concentration (g.100 ml}^{-1}\text{)} = (37.2 \times A) + 0.06$$

(Wintrobe, 1956)

Heamatocrit (% Cell Volume)

Procedure

1. In triplicate, haematocrit tubes (Scientific Instruments) were approximately three-quarters filled with whole blood. The blood was then run up and down the tube to mix it.
2. The tubes were sealed at one end with plasticine by placing them in a 'miniseal' tray. They were then left for analysis at the end of the trial. Samples were not left for more than 7 hr before they were analysed.
3. Tubes were placed in a microcentrifuge (Hawksley Ltd.) with the sealed end towards the outside and centrifuged for 14 min.
4. Percentage cell volume was calculated by placing each tube in a reader (Hawksley Ltd.) where the base (bottom) line of the reader was aligned with the base of the red cells and the top line intersected the top of the plasma. The middle line was then adjusted so that it intersected the top of the red cells and the percentage cell volume read from the scale.

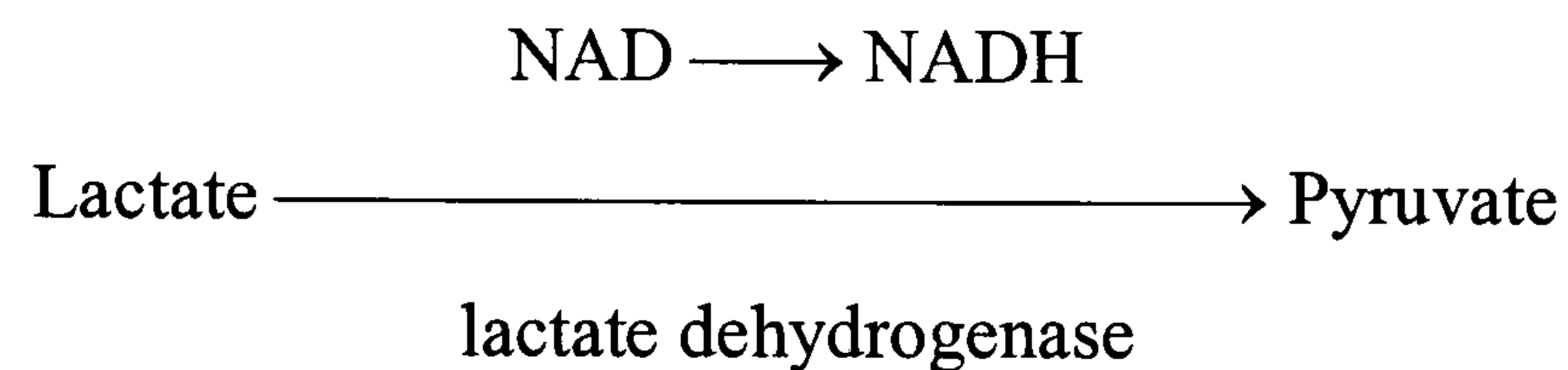
Plasma Volume

Once haemoglobin and haematocrit values were known, plasma volume could be calculated using the method of Dill and Costill (1974).

Lactate

Principle

The fluorimetric assay for lactate is based on that described by Maughan (1982).



The lactate concentrations reported in Chapters IV, VI, VIII and IX were obtained by dispensing the samples and reagents by hand and analysing using a fluorimeter (Locarte) (non-automated analyser protocol) whilst those in Chapters V and VII were obtained using an automated analyser (Cobas Bio, Roche Diagnostics) protocol. In essence, these two methods are the same but both procedures will be explained in full.

Non-automated analyser protocol

Reaction mixture

2.0 mg NAD

10.0 µl LDH

per 1.0 ml of hydrazine buffer (pH ~9.4)

Standards

In addition to a blank of 2.5% perchloric acid, working standards of 1, 2.5, 5, 10, 20 and where appropriate, 30 mM were prepared from 1.0 mM Sodium L-Lactate stock solution.

Protocol (each sample was analysed in duplicate)

1. After being allowed to thaw at room temperature, samples were mixed (Whirlimix) and centrifuged for 3 min at $13000 \text{ rev.min}^{-1}$ (Eppendorf Centrifuge 5415C).
2. 20 μl of either standard or supernatant was pipetted into a glass fluorimeter tube and 200 μl of reaction mixture was added.
3. The tubes were mixed thoroughly (Whirlimix), covered, and left to incubate at room temperature for 30 min.
4. 1.0 ml of lactate diluent (0.07M HCL) was added to each tube and they were mixed thoroughly (Whirlimix) in order to stop the reaction.
5. The fluorescence of the blanks, standards and samples were measured (Locarte).
6. A linear regression plot of the standards was made and the lactate concentration of the samples was calculated.

Automated analyser protocol**Reaction mixture**

1.7 mg NAD

7.0 μl LDH

per 1.0 ml of hydrazine buffer (pH ~9.4)

Standards

The Cobas Bio automated analyser automatically blanks itself. The reagent tray can only accommodate three standards and so standards with concentrations over the appropriate range were used. For the studies described in this thesis, standards of 2, 10 20 mM and where appropriate, 30mM were chosen. In addition a commercially available quality control (Sigma) at a concentration of 2.1 mM was run with each batch

of samples. Both standards and quality control were diluted in 2.5% perchloric acid in the same ratio as the samples.

Procedure

1. The automated analyser (Cobas Bio, Roche Diagnostics) was switched on and the self-check was completed.
2. The test code for the lactate assay was selected on the keyboard.
3. The standards, quality control and samples were mixed thoroughly (Whirlimix) and centrifuged for 3 min at 13000 rev.min⁻¹ (Eppendorf Centrifuge 5415C).
4. The reagent tray was filled with the three standards and the reaction mixture. Pressing the 'START' button initiated the programme on the analyser. When the assay was complete, the concentrations of the standards were printed both in units and as a percentage of their expected values (i.e. 2, 10 and 20 mM). This step was repeated until all standards were 99-101% of their expected concentrations.
5. Approximately 100 µl of sample or quality control was placed into each of the 25 cups comprising the sample disc. During each run two of the cups (the first and last) contained the quality control. The sample cups were pressed firmly into position on the disc and it was placed on the turntable.
6. The reagent tray was re-filled with reaction mixture, if necessary, and the assay was run. The concentration of each sample and quality control was printed in mM.

Ammonia

Blood Collection

When the blood was drawn ~1 ml whole blood was dispensed into a calcium heparinised eppendorf tube (prepared using the method described below). Samples were centrifuged immediately for 3 min at $13000 \text{ rev.min}^{-1}$ (Eppendorf Centrifuge 5415C). The plasma was then removed and pipetted into a plain eppendorf tube and immediately snap frozen until the end of the trial and then stored at -70°C until it was analysed, not more than 48 hr later. The time from blood being drawn to freezing was kept to a minimum in order to prevent contamination from external nitrogen sources and was always less than 15 min.

Preparation of Ca^{2+} /Hep tubes

1. The required amount of Ca^{2+} /Hep was calculated:

1 mg (or ml) of Ca^{2+} /Hep contains 183 units; 15 units are needed per ml of whole blood and each eppendorf tube would contain ~1.5 ml of whole blood. Therefore, each eppendorf tube required ~30 units of Ca^{2+} /Hep (rounded up to allow for wastage).

E.g. for a batch of 300 eppendorf tubes, 9000 units (300×30) would be required. This is equivalent to ~50 mg ($9000 \div 183$) of Ca^{2+} /Hep which would be added to 1.2 ml (4×300) of distilled water.

2. $4\mu\text{l}$ of this solution was then be pipetted into each eppendorf tube.

Principle

The spectrophotometric assay is based on the methods described by Neeley and Phillipson (1968) and van Anken and Schiphorst (1974):



The decrease in absorbance at 340 nm, due to the oxidation of NADPH, is proportional to the plasma ammonia concentration.

Reaction mixture

The reagents are found in bottles found in a commercially available kit (Sigma Diagnostics).

Reagent solution

The reagent solution was reconstituted with the volume of distilled water indicated on each vial label. Each reagent then contained:

2-Oxoglutarate 3.4 mmol.L⁻¹

NADPH 0.23 mmol.L⁻¹

In addition the reagent mixture contained buffer, stabilisers and nonreactive fillers.

Enzyme solution

L-Glutamate dehydrogenase 1200 U/ml

Glycerol 50 % (v/v)

Phosphate buffer pH 7.4

Standards

Each kit included an ammonia control solution at a concentration of $294 \mu\text{mol.L}^{-1}$. In addition control solutions at ammonia concentrations of 29.5 and $118 \mu\text{mol.L}^{-1}$ were used (Sigma Diagnostics).

Procedure

1. A series of 1.5 ml disposable plastic cuvettes with a lightpath of 1cm were set up for BLANK, STANDARD 1 ($29.5 \mu\text{mol.L}^{-1}$, in duplicate), STANDARD 2 ($118 \mu\text{mol.L}^{-1}$, in duplicate), STANDARD 3 ($294 \mu\text{mol.L}^{-1}$, in duplicate) and SAMPLES.
2. 0.5 ml of the reagent solution was added to each cuvette.
3. 150 μl of distilled water was added to the BLANK cuvette, 100 μl of distilled water and 50 μl of ammonia control solution to each of the STANDARD cuvettes and 150 μl of plasma to each of the SAMPLE cuvettes.
4. Cuvettes were covered with sealing film (Nescofilm, Bando Chemical Ind. Ltd., Kobe, Japan) and mixed by gentle inversion.
5. Cuvettes were allowed to equilibrate for approximately 3 min at room temperature.
6. INITIAL absorbance of cuvettes were read (Cecil Instruments) & recorded: 340 nm.
7. 5 μl of enzyme solution was added to each cuvette, cuvettes were mixed by gentle inversion and left to stand for approximately 5 min at room temperature for the reaction to complete.
8. FINAL absorbance of each cuvette was read and recorded at 340 nm.
9. The ammonia concentration was calculated using the following equations:

$$\Delta A = \text{INITIAL } A - \text{FINAL } A$$

$$\text{STANDARD Ammonia } (\mu\text{mol.l}^{-1}) = ((\Delta A_{\text{STANDARD}} - \Delta A_{\text{BLANK}}) \times 35.8) \times 58.8$$

$$\text{SAMPLE Ammonia } (\mu\text{mol.l}^{-1}) = ((\Delta A_{\text{SAMPLE}} - \Delta A_{\text{BLANK}}) \times 11.93) \times 58.8$$

Where the factor 58.8 converts $\mu\text{g.ml}^{-1}$ to $\mu\text{mol.l}^{-1}$.

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Serum human growth hormone analysis

Principle

A commercially available kit (Medgenix-hGH-EASIA, Biosource) was used for the determination of serum human growth hormone (hGH) concentrations by routine ELISA. Microtitration plates pre-coated with specific monoclonal antibodies are supplied into which serum samples and standards are pipetted. Standards and samples containing hGH react with capture antibodies coated on the wells of the microtitration plates (Mabs 1) and with monoclonal antibodies (Mabs 2) labelled with horse radish peroxidase (HRP). During incubation a sandwich forms (coated Mabs 1 – hormone – Mabs 2 – HRP). The microtitration plate is washed to remove any unbound enzyme labelled antibodies. A revelation solution is added and incubated before a stopping agent is added and the microtitration plate is read at the appropriate wavelength. The amount of substrate turnover is determined colorimetrically by measuring the absorbance which is proportional to the hormone concentration.

Reagents

- Microtitration plate
- Standard 0 mU.l⁻¹ in sheep serum, sodium merthiolate
- Standards 1, 5, 15, 100 mU.l⁻¹ in sheep serum, sodium merthiolate
- Controls 1 and 2 in human serum, sodium merthiolate
- Anti-hGH-HRP conjugate in Tris-HCl buffer with bovine serum albumin and preservatives

-
- Tween 20, 20% (washing solution)
 - Chromogen TMB (Tetramethylbenzidine)
 - Substrate buffer, H₂O₂ in acetate/citrate buffer
 - H₂SO₄ 1.8 N (stopping reagent)

Procedure

1. Reagents were prepared according to the instructions in each kit.
2. 50µl of each standard, control or sample was pipetted into the appropriate wells.
3. 50µl of Anti-hGH-HRP conjugate was pipetted into each well.
4. The standards, controls and samples were incubated for 1 hr at room temperature on a horizontal shaker (Automix III, Heidolph) set at 700 ± 100 rpm.
5. The plate was washed using an automated washer (Denley Wellwash 4 mk 2) by aspirating the liquid from each well, dispensing 0.4 ml of washing solution into each well, aspirating the content of each well, dispensing 0.4 ml of washing solution into each well for a second time and aspirating the content of each well.
6. 200µl of freshly prepared revelation solution (chromogen TMB and substrate buffer) was pipetted into each well within 15 min following washing.
7. The standards, controls and samples were incubated for 15 min at room temperature on a horizontal shaker (Automix III, Heidolph) set at 700 ± 100 rpm, avoiding direct sunlight.
8. 50µl of stopping reagent was pipetted into each well.
9. Absorbance was measured within 1 hr using an ELISA plate reader (Anthos htII microplate reader, Anthos Labtec Instruments) and the results printed.
