Gross efficiency, maximal muscle function and cycling endurance exercise

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Abstract

Prolonged moderate intensity cycle exercise is associated with a gradual and progressive reduction in gross efficiency (GE). It was speculated that this loss of GE might reflect a reduction in maximal muscle function and result in a parallel decline in aerobic performance. The effect of prolonged moderate intensity cycle exercise on maximal muscle function and anaerobic performance has not been clearly established. This thesis examined the impact of 1-2 h of cycling exercise at 60-65% maximal aerobic power in well-trained cyclists on subsequent changes in aerobic and anaerobic performance, GE and maximal muscle function.

A reduction in GE (-8.4%, $P < 0.005$) and 5 minute performance trial (-3.7%, $P < 0.005$) was found following 75 min of exercise in 10 cyclists. Furthermore, the change in GE was significantly correlated with the change in 5 minute performance ($r = 0.91$, $P = 0.01$). A subsequent study found that both peak and mean 30 s sprint performance were significantly reduced (-2.7%, $P < 0.05$, and –5.3%, $P = 0.01$, respectively) in nine cyclists following a 70 min exercise bout. Neither the reduction in peak or mean sprint power output were significantly correlated with the change in GE. The final study examined the effects of 2 h at 60% maximal aerobic power and 2 h of recovery on GE and maximal isokinetic cycling power output (MICPO) in 11 cyclists. Further, the effects of exercise induced dehydration and carbohydrate (CHO) intake during recovery on GE and MICPO were studied on nine of the subjects. A significant exercise induced reduction in both GE (-5.3%, $P < 0.05$) and MICPO (-3.8%, $P < 0.05$) was observed after 2 h of exercise. Both variables recovered slowly following exercise with GE still significantly reduced after 2 h and MICPO after 1 h of recovery. The loss of GE was increased by exercise induced dehydration but was not significantly influenced by CHO ingestion in recovery. Neither dehydration nor CHO ingestion affected the changes in MICPO.

It is concluded that prolonged moderate intensity cycle exercise results in a significant decrease in aerobic and anaerobic performance, GE and maximal muscle function. The reduction in aerobic performance was associated with the decline in GE, which did not appear to be primarily mediated by changes in maximal muscle function. Following prolonged exercise maximal muscle function and GE are restored slowly. Mechanisms

with a similar temporal pattern in recovery are implicated as causes of fatigue during prolonged exercise, such as muscle glycogen depletion and non-metabolic fatigue.

Table of Contents

List Of Tables

List of Figures

Publications and Conference Communications

The following works from this thesis have been presented and published as detailed below. See Appendix A for full text.

Publications

Passfield, L., and J. H. Doust, (1998) Influence of changes in gross efficiency on endurance cycling performance. Journal of Sports Sciences, Vol. 16(5): 477-478.

Jones S. M. and L. Passfield, (1998) The dynamic calibration of bicycle power measuring cranks. In: The Engineering of Sport, S.J. Haake (ed.), Blackwell Science, Oxford. Pages: 265-274.

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Conference Communications

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Jones, S. M. and L. Passfield (1998) The dynamic calibration of bicycle power measuring cranks. 2nd International Conference on Engineering of Sport, Sheffield University, UK.

Passfield, L. and J. H. Doust, (1997) Effect of endurance exercise on 30s Wingate sprint in cyclists. Physiological Society, Cambridge University, UK.

Passfield, L. and J. H. Doust, (1997) Influence of changes in gross efficiency on endurance cycling performance. 2nd European Congress of Sports Sciences, Copenhagen.

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Chapter 1: Introduction

1.1 Determinants of endurance performance

Competitive endurance cyclists regularly train and race at moderate to high exercise intensities for prolonged periods. Despite competing in races of 90 miles or more which may last for several hours, competitors average over 50% of peak aerobic power output (PAPO[1]) or higher. Figure 1.1 depicts the power output profile measured directly from the cranks (SRM, Julich, Germany) of a cyclist competing in a British elite category race. Both the absolute power output (W) averaged over 60 s and the corresponding relative intensity (% PAPO) are shown.

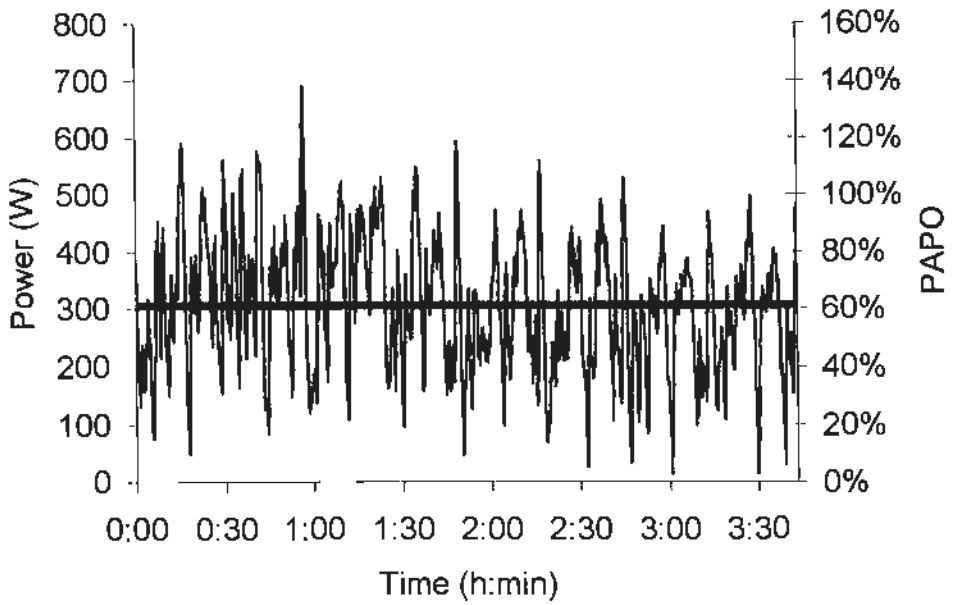


Figure 1.1 Power output profile of a cyclist in a British elite category race. Data are averaged over 60 s. The thick horizontal line indicates the average power output for the race. Source: unpublished observations, L. Passfield.

The highly correlated indices of peak pulmonary oxygen uptake ($\dot{V}O_{2peak}$) and PAPO (Keen et al. 1991; Hawley and Noakes, 1992) are widely acknowledged to be important

[1] PAPO is the highest 60 s power output achieved during a test of peak aerobic power, see section 4.8.2

determinants of cycling endurance performance (Burke et al. 1977; Burke, 1980; Sjøgaard et al. 1986; Coyle et al. 1988; Saris et al. 1989; Hawley and Noakes, 1992; Neumann, 1992; Coyle, 1995; Olds et al. 1995; Passfield and Hale, 1995, 1997; Passfield et al. 1997). This is emphasised in Figure 1.1 by the high power output (300 W) averaged for almost 4 h. In general, individuals possessing a larger $\dot{V}O_{2peak}$ are advantaged in competitive cycling situations because they are able to achieve higher sustainable rates of energy expenditure, i.e. supported predominantly by aerobic metabolism. Furthermore, any given power output represents a lower relative exercise intensity and therefore a reduced metabolic stress. Consequently, significant correlations between indices of peak aerobic power and endurance cycling performance have been suggested (Coyle et al. 1991a; Hawley & Noakes, 1992).

1.2 Defining fatigue

Vollestad and colleagues have repeatedly argued that an accurate definition of fatigue is important for an appropriate interpretation of the consequences of prolonged exercise (Vollestad & Sejersted, 1988; Sejersted & Vollestad, 1992; Vollestad, 1997). Fatigue has often been defined after Edwards (1983) as an inability to maintain the required or expected force or power output. Vollestad and colleagues point out that Edwards's definition tends to confuse fatigue and exhaustion by implicitly ignoring any changes preceding the point where an individual is unable to maintain the required force or power output. Fatigue that occurs during prolonged exercise is likely to be the manifestation of ongoing processes and characterised by a reduction in maximal muscle function over time. Vollestad argues that fatigue should, therefore, be interpreted as: "any exercise-induced reduction in the capacity to generate force or power output" (Vollestad, 1997, pp. 220). Fatigue has occurred where a loss of maximum force or power output can be demonstrated. Exhaustion, by contrast, corresponds to the time point where a fixed work rate can no longer be sustained. Typically, at exhaustion the work rate has to be reduced considerably over a short period of time if exercise is to be continued. This terminology will be adopted throughout the thesis.

1.3 Factors limiting endurance performance

Variations in endurance performance between individuals are largely determined by aerobic power. Within an individual however, performance is thought to be limited by

several different factors either separately or in combination. These include substrate availability (carbohydrate - CHO), dehydration and hyperthermia, (for reviews see Conlee, 1987; Coyle & Hamilton, 1990; Green, 1991; Terrados & Maughan, 1995). Curiously, no one has attempted to incorporate performance determining and limiting factors into an integrated model of endurance performance. This may be due to the definition of fatigue frequently adopted (Vollestad, 1997), and the perpetuation of weak experimental designs used to examine endurance physiology (McLellan et al. 1995; Jeukendrup et al. 1996). Whatever the cause, it is clear that very little research has been conducted on the impact of dynamic endurance exercise on subsequent muscle function (Nielsen et al. 1993; Rademaker et al. 1994b; Sahlin & Seger, 1995). The paucity of data on maximal muscle function and endurance exercise is in contrast to the extensive literature on the fatiguing consequences of isometric and high intensity exercise, (for reviews see Vollestad and Sejersted, 1988; Lännergren, 1992; Sahlin, 1992a; Fitts, 1994; Allen et al. 1995).

1.4 Experimental design and endurance exercise

It is possible that the mechanisms of fatigue and exhaustion are to some degree distinct and separate (Vollestad and Sejersted, 1988). Previous studies employing endurance exercise trials have tended to use time to exhaustion as their reference criterion (Coyle et al. 1986; Brouns et al. 1989), as opposed to the extent of fatigue. The latter approach is superior not only by quantifying fatigue prior to exhaustion, but in providing substantially greater repeatability (Krebs and Powers, 1989; Hickey et al. 1992; McLellan et al. 1995; Jeukendrup et al. 1996). Furthermore, an experimental protocol designed to quantify the extent of fatigue possesses a greater ecological validity in comparison with exercise time to exhaustion, and may also yield insights into the mechanisms of fatigue (Keizer et al. 1987; Rademaker et al. 1994b; Sahlin and Seger, 1995). Finally, it is noted that endurance performance is much more likely to be limited by the development of fatigue rather than exhaustion.

1.5 Fatigue and endurance exercise

The difference between individuals in PAPO due to varying levels of fitness is thought to be of greater magnitude than that caused by fatigue resulting from moderate intensity exercise. Passfield and Hale (1997) found PAPO to be reduced by approximately 30 W

following 3 h at 60% $\dot{V}O_{2peak}$, clearly less than the difference in PAPO that would be expected between riders (Burke et al. 1977; Burke, 1980; Coyle et al. 1991a; Hawley & Noakes, 1992; Craig et al. 1993). It is expected therefore, that peak aerobic power plays a primary role in dictating endurance performance by setting the relative intensity for any given velocity. Peak aerobic power may change little despite extensive training, particularly in elite cyclists (Sjøgaard, 1984; Kuipers et al. 1985; Jeukendrup et al. 1992; Barbeau et al. 1993; Weston et al. 1997). Accordingly, amongst athletes of comparable peak aerobic power, differences in fatigue resistance may assume greater significance.

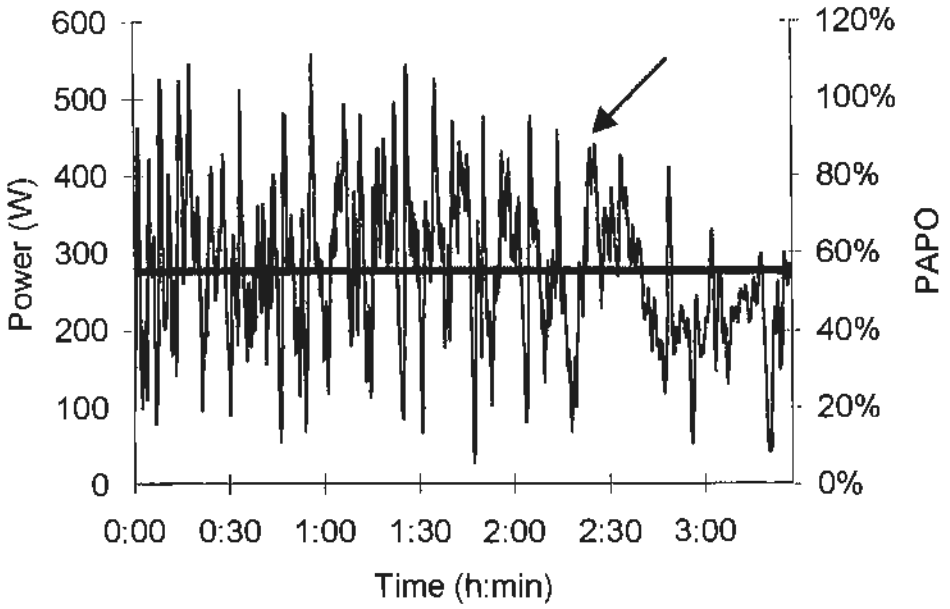


Figure 1.2 Power output profile of a cyclist failing to maintain contact with the leading group (arrow) in a British elite category race. Data are averaged over 60 s. The thick horizontal line indicates the average power output for the race. Source: unpublished observations, L. Passfield.

The practical implications of these theoretical considerations are illustrated in Figure 1.2, which shows further power data for a competitive cyclist. In this race the cyclist was unable to stay with the leading group (indicated by arrow) and forced to chase, eventually having to ride to the finish alone. It is evident that the power output the rider was able to generate had declined appreciably from the earlier stages of the race. These data demonstrate that the rider had fatigued considerably at the point he was unable to

remain with the leading group. In situations such as this, where athletes race each other as opposed to the clock, the capacity to resist fatigue may be decisive in determining the outcome of the race.

Current models of endurance performance do not appear to promote a comprehensive understanding of relevant physiology, since the effects of fatigue are generally ignored (e.g. Coyle 1995). Coyle's model focuses upon determinants of the highest sustainable rate of oxygen consumption ($\dot{V}O_2$) and power output. Whilst this model provides a useful insight into the factors which dictate initial performance velocity, it overlooks the impact of any exercise induced changes on the determining variables. Of particular interest are the effects of prolonged cycle exercise on gross efficiency (GE), assumed in the model of Coyle to remain stable. A progressive increase in $\dot{V}O_2$ ($\dot{V}O_2$ drift) is commonly observed however, even during prolonged cycle exercise at a constant power output (Hagberg et al. 1978; Coyle et al. 1991b; Hamilton et al. 1991; Hagan et al. 1992; Passfield & Hale, 1997). This $\dot{V}O_2$ drift is likely to be greater than that required for changes in substrate utilisation, implying GE has been reduced. Moreover, Passfield & Hale (1997) have found that GE following a 3 h endurance trial was strongly correlated with the observed drop in PAPO. This finding suggests that changes in GE may provide an index of fatigue during prolonged cycle exercise. The cause for this change in GE is unknown.

1.6 Thesis delimitations

The present thesis was delimited to considering the effects of prolonged moderate intensity cycle exercise on subsequent performance, GE and maximal muscle function. A cycling based exercise model is particularly relevant in providing an understanding of endurance performance. Interpretation of cycle exercise induced responses are unlikely to be confounded by resultant muscle damage (Kuipers and Keizer, 1988), probably due to the lack of eccentric muscle actions in cycling. Furthermore, cycle exercise permits strict control of power output and cadence, and particularly in trained subjects is likely to result in the recruitment of a relatively stable lower limb muscle mass, (i.e. restrict the contribution of the upper body). Throughout this thesis exercise in the range of 40-65% $\dot{V}O_{2peak}$ will be referred to as of moderate intensity. Whilst this nomenclature is consistent with the majority of literature on endurance physiology, some discrepancies

are evident. Typically, researchers interested in the pulmonary oxygen kinetics define the exercise domains not by $\%\dot{V}O_{2peak}$, but rather in relation to a "lactate threshold".

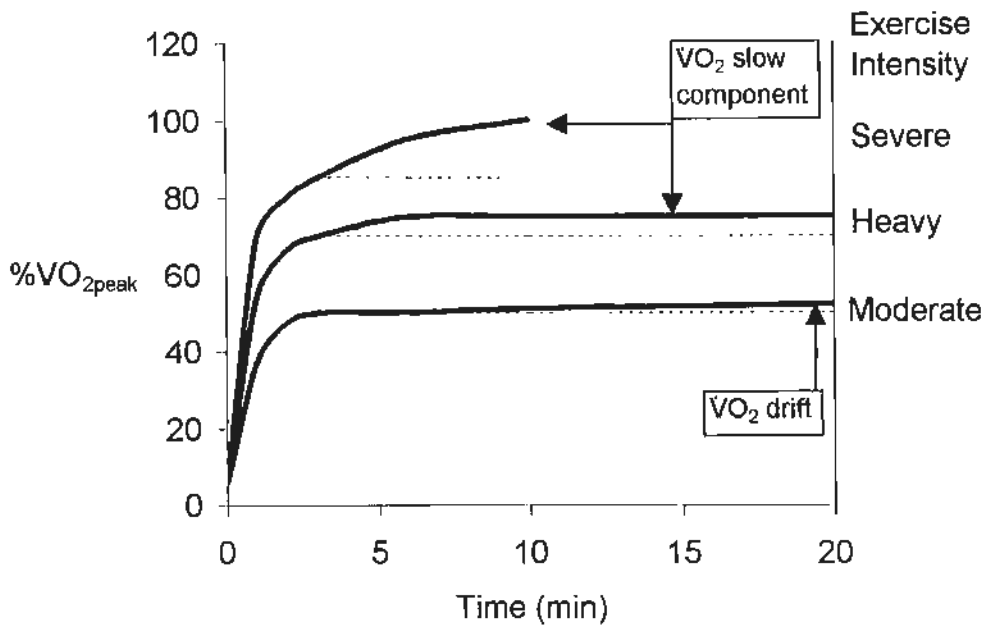


Figure 1.3 A schematic representation of the relationship between pulmonary oxygen uptake kinetics and exercise intensity. The $\dot{V}O_2$ drift at moderate intensity and $\dot{V}O_{2SC}$ at heavy and severe intensities are indicated by the projection of $\dot{V}O_2$ above the dotted lines. Modified from Poole and Richardson (1997).

Figure 1.3 above, schematically presents the pulmonary oxygen uptake kinetic responses for moderate to severe exercise intensities. As discussed previously and indicated in Figure 1.3, prolonged moderate intensity exercise is associated with a gradual drift in $\dot{V}O_2$ (Hagberg et al. 1978, Hagan et al. 1992, Passfield and Hale, 1997). Exercise of this nature is associated with a low and stable blood lactate concentration. In contrast, heavy and severe exercise intensities do not result in the achievement of a rapid $\dot{V}O_2$ steady state. Instead, an increasing $\dot{V}O_2$ slow component ($\dot{V}O_{2SC}$) is evident, arising some 80 to 110 s after the onset of exercise (indicated as projecting beyond the dotted lines in Figure 1.3). Heavy exercise results in a delayed and elevated $\dot{V}O_2$ steady state, whilst during severe exercise $\dot{V}O_2$ increases continuously to $\dot{V}O_{2peak}$ and ensuing exhaustion (Sloniger et al. 1996). An elevated blood lactate response is noted during heavy and

severe exercise which is often temporally similar to the $\dot{V}O_2$ response (for recent review see Poole and Richardson, 1997). Gaesser & Poole (1996) contend that the $\dot{V}O_2$ drift observed during prolonged moderate intensity exercise should be viewed as separate and distinct from the $\dot{V}O_{2SC}$ that characterises higher intensity exercise. Accordingly, it is suggested that minor differences in the method of denoting exercise domains is of little significance within this thesis, which is concerned with the effects of exercise of 'moderate' intensity, i.e. 40-65% $\dot{V}O_{2peak}$.

1.7 Summary

In summary, the effects of prolonged, moderate intensity cycle exercise, not resulting in exhaustion, do not appear to have been extensively examined. In particular, the extant literature features little research on the effects of such exercise on GE, maximal muscle function, endurance performance and the possible inter-relations among these parameters. The findings of the present thesis are of relevance not only to athletes competing in endurance events, but may provide an insight into the mechanisms of fatigue associated with this type of exercise.

Chapter 2: Cycling gross efficiency

2.1 Introduction

The pioneering work of Lavoiser and his contemporaries of the 18th and early 19th centuries has been suggested to form the foundations of our modern scientific processes and understanding in human physiology (Benedict and Cathcart, 1913). Further advancement in the understanding of human physiology of that time, appears to have been retarded by the perpetuation of the phlogiston theory, a focus on the measurement of CO_2 production rather than O_2 consumption ($\dot{V}O_2$), and the lack of sophistication in ergometry systems. Benedict and Cathcart (1913) suggest that the proposal by J.R. Mayer in the middle of the 19th century of the law of conservation of energy markedly influenced thinking at that time. Consequently, physiologists began to turn their attention to the issue of the balance of energy in human metabolism at rest and in exercise. As calorimetry and ergometry systems were progressively refined through the latter part of the 19th century, the efficiency of human work attracted increasing attention. This culminated in the publication of several authoritative works on human muscular efficiency and influencing factors in the early part of this century (Benedict and Cathcart, 1913; Hill, 1922; Dickinson, 1929; Garry and Wishart, 1931).

Since the early part of the 20th century much of the research into human muscular efficiency has employed cycle ergometry, as this was thought to allow the accurate quantification of energy output. Consequently, more recent research has tended to compare indices of cycling efficiency with that of isolated muscle, and to examine factors which may influence this relationship (Whipp and Wasserman, 1969; Gaesser and Brooks, 1975; Nordeen-Snyder, 1977; Suzuki, 1979; Hagberg et al. 1981; Coyle et al. 1992). Much of this work using cycle ergometry has focused upon the effect of work rate and cadence on calculated efficiency (Pugh, 1974; Gaesser and Brooks, 1975; Hagberg et al. 1981; Seabury et al. 1977). Surprisingly, the performance implications of economy of movement appear to have been acknowledged much earlier for running than cycling (c.f. Costill and Fox, 1969; Horowitz et al, 1994).

2.2 Defining efficiency

During steady state cycle ergometry efficiency and economy have been extensively used to provide a convenient index of how efficiently an individual can convert chemical energy into mechanical power (Gaesser and Brooks, 1975; Suzuki, 1979; Hagberg et al. 1981; Künstlinger et al. 1985; Barbeau et al. 1993; Capelli et al. 1993b; Kenny et al. 1995; Nickleberry and Brooks, 1996). The most commonly used measures of efficiency are gross efficiency (GE) and economy of movement. Gross efficiency may be defined as the ratio of power output to power input (Frederick, 1992). In calculating GE, external work accomplished is commonly used instead of power output, with power input correspondingly replaced with energy expenditure. The energy equivalent of steady state $\dot{V}O_2$ and respiratory exchange ratio (RER) is used to calculate energy expenditure. The term 'gross efficiency' is normally reported as a percentage, whilst 'economy' is used when an efficiency rating is expressed in watts·L^{-1} $\dot{V}O_2$·min^{-1} (Coyle et al. 1991a) or as an absolute $\dot{V}O_2$ for a given work rate (Swain and Wilcox, 1992). Economy measures do not therefore, take account of the changes in O_2 requirement resulting from differences in substrate utilisation. This convention will be followed throughout this thesis.

Several measures of efficiency have been suggested to provide more valid indices of muscular efficiency than GE (Whipp and Wasserman, 1969; Gaesser and Brooks, 1975; Suzuki, 1979; Coyle et al. 1992; Sidossis et al. 1992). These include, net, work and delta efficiency. All are defined below.

Equations 2.1

$$Gross\ Efficiency = \frac{external\ work\ accomplished}{energy\ expenditure} x100\%$$

$$Net\ Efficiency = \frac{external\ work\ accomplished}{(energy\ expenditure - resting\ energy\ expenditure)} x100\%$$

$$Work\ Efficiency = \frac{external\ work\ accomplished}{(energy\ expenditure - energy\ expenditure\ at\ 0\ work\ rate)} x100\%$$

$$Delta\ Efficiency = \frac{increment\ in\ external\ work\ accomplished}{increment\ in\ energy\ expenditure} x100\%$$

After Stainsby et al. (1980)

Early in this century the difficulties inherent in estimating muscular efficiency from cycle ergometry were fully appreciated (Garry and Wishart, 1931). These problems remain today and it is important to acknowledge that GE should not be interpreted as a measure of true muscular efficiency (Stainsby et al. 1980). Stainsby et al. point out that energy expenditure during exercise includes unmeasured work. The cost of moving limbs, stabilising body parts, cardiac and ventilatory work, ion pumping, substrate synthesis and delivery will all add to the total energy consumption. Also, not all the muscular force generated will be applied effectively to the ergometer and translated into external work accomplished. Some energy expenditure is 'wasted' in stretching, compressing or bending components (Lafortune and Cavanagh, 1983) and frictional losses within the ergometer (Whitt and Wilson, 1974; Kyle and Caiozzo, 1986). The extent of the total 'wasted' energy expenditure is likely to vary in proportion to the external power output and remains to be clearly established (Whitt and Wilson, 1974; Kyle and Caiozzo, 1986; Hibi et al. 1996). Coyle and colleagues (Coyle et al. 1992; Sidossis et al. 1992) have suggested the most valid way of estimating muscular efficiency is to measure delta efficiency (ΔE). By using the ratio of change in work accomplished to change in energy expended, Coyle et al. argued they were partitioning out the influence of unmeasured work. Stainsby et al. (1980) point out that any type of baseline subtraction is valid only in situations where the unmeasured work remains constant across exercise conditions. These authors point out that, since ΔE is necessarily calculated from data collected over two or more different work rates, this assumption becomes untenable. Exercise induced changes in splanchnic metabolism, body temperature, ventilation, catecholamine release and lactate turnover are all cited by Stainsby et al. as factors which may serve to change the contribution of the unmeasured work at different power outputs.

2.3 Measuring efficiency

In order to measure efficiency reliably all gas collections must take place under steady state exercising conditions, otherwise measured pulmonary $\dot{V}O_2$ will not reflect muscle O_2 consumption. At the onset of exercise or in the transition from one work rate to another, during light to moderate intensities, pulmonary $\dot{V}O_2$ increases in a mono-exponential manner to reach a steady state within 2-3 minutes (Whipp and Wasserman, 1972). Heavy exercise intensities, characterised by a sustained metabolic acidosis, have

been found to result in a delayed steady state, or in severe exercise for $\dot{V}O_2$ to continue rising to $\dot{V}O_{2peak}$ and ensuing exhaustion (Poole et al. 1988; Whipp, 1994; Sloniger et al. 1996). Consequently, when examining carefully the relationship between $\dot{V}O_2$ and power output a marked deviation from linearity at higher intensities is observed (Stuart et al. 1981; Green and Dawson, 1995; Zoladz et al. 1995). The exercise intensities eliciting this additional O_2 consumption, or O_2 slow component (O_{2SC}), should not be used to determine efficiency values. This is because the calculated energy expenditure will be heavily dependent upon the timing of the expired gas collection and thus the extent of O_{2SC}. All efficiency measures should be collected during light to moderate exercise, where the effect of an O_{2SC} is negligible.

Indirect calorimetry can be used to provide a valid means of estimating oxidised substrate under steady-state exercising conditions (Jansson, 1982; Romijn et al.1992). The validity of calculating energy expenditure from $\dot{V}O_2$ depends upon the equivalence of RER and muscle RQ. Jansson (1982) found RER and leg RQ to respond in a similar manner during exercise at 65% $\dot{V}O_{2peak}$ after dietary manipulation to vary the proportions of CHO and fat oxidised. Romijn et al, (1992) have found indirect calorimetry to be valid also at relatively high exercise intensities ($\approx$ 85% $\dot{V}O_{2peak}$). During non-steady state exercise, either plasma bicarbonate buffering or transient CO_2 storage may invalidate indirect calorimetry by varying the fraction of expired CO_2 (Whipp, 1987). It should be noted that even where pulmonary gas exchange is measured under exercising steady state conditions, RER may not necessarily reflect muscle RQ, as the RER is an integrated response of all metabolically active tissues in the body (Riley et al. 1996). Any errors in RER measurement are likely to have only minor impact on the estimated energy expenditure. It can easily be demonstrated from the formulae of Lusk (1924), that an extreme miscalculation resulting in a change in RER from 0.707 to 1.0 will increase calculated energy expenditure by 7.7%. Decreasing RER by 0.05 reduces calculated energy expenditure by 1.3%, typically increasing GE by 0.4%.

2.4 Factors influencing gross efficiency

There are a large number of published studies examining factors thought likely to influence GE and economy in cycle ergometry. Where repeated measures of GE are made

the influence of these variables may need to be carefully controlled. Many of the factors known or suggested to influence GE are summarised in the table below.

Table 2.1 Factors influencing cycling gross efficiency and economy

Parameter	Effect	Study
Power output	Y	Garry and Wishart, 1931; Jackson and Banister, 1967; Gaesser and Brooks, 1975; Seabury et al. 1977; Stuart et al. 1981; Faria et al. 1982; Patterson et al. 1983; Croisant and Boileau, 1984; Coast and Welch, 1985; Künstlinger et al. 1985; Ericson and Nisell, 1988; Berry et al. 1993; Keuny et al. 1995; Nickleberry and Brooks, 1996; Londeree et al. 1997; Marsh and Martin, 1997.
Cadence	Y	Benedict and Cathcart, 1913; Hill, 1922; Dickinson, 1929; Garry and Wishart, 1931; Jackson and Banister, 1967; Gaesser and Brooks, 1975; Seabury et al. 1977; Suzuki, 1979; Miyashita and Kanehisa, 1980; Hagberg et al. 1981; Stuart et al. 1981; Faria et al. 1982; Patterson et al. 1983; Croisant and Boileau, 1984; Merrill and White, 1984; Coast and Welch, 1985; Coast et al. 1986; Ericson and Nisell, 1988; Ahlquist et al. 1992; Swain and Wilcox, 1992; Rademaker et al. 1994; Nickleberry and Brooks, 1996; Takaishi et al. 1996; Londeree et al. 1997; Marsh and Martin, 1997.
Fibre type	Y?	Suzuki, 1979; Jansson and Kaijser, 1987; Coyle et al. 1992; Horowitz et al. 1994.
Muscle action	Y	Abbot et al. 1952; Ryschon et al. 1997.
Body position and size	Y	Sheunum and deVries, 1976; Nordeen-Snyder, 1977; Ryschon and Stray-Gundersen, 1991; Berry et al. 1993; Origenes et al. 1993; Francescato et al.1995; Heil et al. 1995, 1997; Gnehm et al. 1997; Londeree et al. 1997; Price and Donne, 1997.
Pedalling dynamics	N?	Henderson et al. 1977; Carmichael et al. 1982; Conrad and Thomas, 1983; Lafortune and Cavanagh, 1983; Patterson et al. 1983; Coyle et al. 1991a; Cullen et al. 1992; Rademaker et al. 1994a.
Fitness	N?	Jansson aud Kaijser, 1987; Nickleberry and Brooks, 1996; Marsh and Martin, 1997.
Ergometer	Y?	Patterson et al. 1983; Kenny et al. 1995; Passfield et al. 1998.
Age	N	Rowland et al. 1990.

2.4.1 Power output

Gross efficiency increases with power output (Jackson and Banister, 1967; Gaesser and Brooks, 1975; Seabury et al. 1977; Stuart et al. 1981; Croisant and Boileau, 1984; Coast and Welch, 1985). This is largely due to unmeasured work forming a smaller percentage of total energy expenditure at higher work rates. It is also possible that the pattern of force application on the pedal contributes to this apparent change in efficiency. Several authors have found pedal forces to be directed more efficiently at higher power outputs (Ericson and Nisell, 1988; Patterson and Moreno, 1990; Sanderson, 1991). Theoretically,

it is likely that muscular efficiency is reduced at higher work rates (Gaesser and Brooks, 1975). This is due to the progressive recruitment of less efficient Type II fibres at higher work rates (Gollnick et al. 1974; Vollestad et al. 1984). This change in recruitment is also likely to result in the optimal velocity for peak efficiency occurring at increasingly faster cadences (Goldspink, 1978). These points are discussed in more detail in a subsequent section 2.4.3 below.

2.4.2 Cadence[1]

Of all the variables considered, it is the effect of cadence on efficiency that has attracted the majority of research attention (see Table 2.1). Most authors agree that cadence influences efficiency markedly. In general, higher cadences are associated with a decreased efficiency. The major stimulus for research is the frequent finding that experienced cyclists in particular, adopt a cadence considerably faster than their most efficient (Gaesser and Brooks, 1975; Seabury et al. 1977; Faria et al. 1982; Patterson et al. 1983; Merrill and White, 1984; Nickleberry and Brooks, 1996; Marsh and Martin, 1997). One notable exception is the study of (Hagberg et al. 1981), which found experienced cyclists' preferred cadence also to be their most economical.

Several studies have demonstrated the relationship between cadence and energy expenditure is approximately quadratic in form (Dickinson, 1929; Garry and Wishart, 1931; Jackson and Banister, 1967; Seabury et al. 1977; Hagberg et al. 1981; Takaishi et al. 1996). Those studies that did not find a minimum tend not to have examined the particularly slow cadences (< 40 rev·min^{-1}), (e.g. Benedict and Cathcart, 1913; Marsh and Martin, 1997). Several factors contribute to the reduced efficiency observed at higher cadences. These include the increased cost of internal work; the reduced effectiveness of pedal force application and probable changes in muscle fibre recruitment. The oxygen cost of moving the legs, measured as unloaded pedalling, is increased with faster pedalling (Whipp and Wasserman, 1969; Gaesser and Brooks, 1975; Hagberg et al. 1981). This is thought to be due largely to the increased energy cost of moving the legs and stabilising other body parts. The O_2 cost of unloaded cycling forms the subtracted

[1] Strictly cadence should be reported in SI units (i.e. rad·s^{-1}). For clarity rev·min^{-1} will be adopted throughout this thesis.

baseline for the calculation of work efficiency, but has been criticised by Gaesser and Brooks (1975) for resulting in unrealistic values. This may not be surprising given cycling without resistance represents a novel task for most cyclists.

It has been clearly established that the pattern of force application on the pedal is compromised at higher pedalling rates (Lafortune and Cavanagh, 1980; Patterson et al. 1983; Cavanagh and Sanderson, 1986; Gregor and Rugg, 1986; Ericson and Nisell, 1988; Patterson and Moreno, 1990; Sanderson, 1991; Faria, 1992, Rademaker et al. 1994a). Thus, at faster cadences cyclists are unable to direct their pedal forces as effectively, i.e. tangentially to the crank (Cavanagh and Sanderson, 1986; Ericson and Nisell, 1988; Sanderson, 1991; Rademaker et al. 1994a). Patterson and Moreno (1990) have suggested that the quadriceps muscle is unable to contract and relax with sufficient speed to enable optimally directed pedal forces when the cadence approaches 120 rev·min^{-1}. These authors suggest that the twitch time of the quadriceps muscle is similar to that of the propulsive phase for each pedal at this cadence.

The factors discussed above may explain why faster pedalling rates are less efficient, but cannot account for the minimum apparent in the studies cited earlier. It is speculated that the quadratic curve for $\dot{V}O_2$ at increasing cadences may be due to changes in muscle fibre recruitment. At slow and fast cadences an increased recruitment of less efficient Type II muscle fibres may occur (Barstow et al. 1996). For a given power output low cadences require higher peak forces to be generated at the pedal (Sjøgaard, 1978; Künstlinger et al. 1985; Sargeant and Greig, 1988; Patterson and Moreno, 1990), whilst fast pedalling increases the velocity of muscle contraction. Both of these factors are likely to favour the recruitment of Type II fibres (Sargeant, 1994).

2.4.2.1 Efficiency, cadence and power output

Croisant and Boileau (1984) point out that the effects of cadence, braking load and power output on efficiency (and other metabolic responses) should not be considered separately. Indeed, there is a wealth of data demonstrating the interaction of power output and cadence with efficiency (Jackson and Banister, 1967; Gaesser and Brooks, 1975; Seabury et al. 1977; Faria et al. 1982; Patterson et al. 1983; Böning et al. 1984;

Croisant and Boileau, 1984; Coast and Welch, 1985; Faria, 1992; Sidossis et al. 1992; Nickleberry and Brooks, 1996; Londeree et al. 1997; Marsh and Martin, 1997). Coast and Welch, (1985) found optimal cadence to increase linearly with power output. This conclusion is broadly supported by many of the studies, although several demonstrate no effect of cadence rather than a linear relationship (Seabury et al. 1977; Faria et al. 1982). The reason for the increase in optimal cadence at higher power outputs has not been readily explained. Coast and Welch (1985, pp. 342) describe the adoption of a strategy of increasing cadence with power as a "logical phenomenon". This observation does not provide an explanation of their results. It is speculated that peak efficiency shifts to higher contraction velocities as a result of changes in muscle fibre recruitment. Experimental evidence for this speculation is provided by the observation that no effect of cadence was found on GE at high exercise intensities (80-90% $\dot{V}O_{2pcak}$), despite a significant increase in the cost of unloaded pedalling (Sidossis et al. 1992). Further research is required in this area.

2.4.2.2 Optimal cadence

The frequently cited study of Hagberg et al. (1981) is notable in finding experienced racing cyclists are most efficient at their preferred cadence. These researchers examined the responses of 7 experienced racing cyclists riding their own bicycles on a treadmill at approximately 80% $\dot{V}O_{2pcak}$. Hagberg et al. found cadences both below and above the preferred rate to be less efficient, with a more pronounced increase in $\dot{V}O_2$ above. Several unsatisfactory explanations have been offered for these apparently divergent results. Seabury et al. (1977) have erroneously assumed that the inertial difference of the lighter crankset and wheels on a racing cycle in comparison with a heavy Monark flywheel may partly account for the apparent discrepancy. This misunderstanding stems from a failure to appreciate the inertia of a cyclist on a treadmill is much greater than that of a Monark ergometer when one remembers to account (correctly) for the mass of the cyclist too. Coast and Welch (1985) suggest that the champion status of Hagberg et al.'s, subjects may have influenced their results. Hagberg et al. do not refer to their subjects as champions, nor is their reported $\dot{V}O_{2peak}$ of 67 $ml \cdot kg^{-1} \cdot min^{-1}$ commensurate with that of World and Olympic champion cyclists tested by this author previously, or reported in the

literature (Sjøgaard et al. 1986; Saris et al. 1989). Merrill and White (1984) simply suggest that coach and peer pressure influence the choice of cadence.

A close examination of the study of Hagberg et al. suggests their work should be interpreted with caution. The efficiency values reported are work efficiency values rather than the more common economy or GE values. The chosen work rate of 80% $\dot{V}O_{2peak}$ makes the achievement of a metabolic steady state quite unlikely which is confirmed by the reported blood lactate concentrations of approximately 6.0 $mmol \cdot L^{-1}$. Further, the selection of a high exercise intensity is known to result in a faster optimal cadences (see 2.4.2.1 above). Surprisingly, cadence was not tightly controlled with some subjects changing their preference during the course of the study. Even the form of ergometer employed in the study (treadmill riding) may have influenced the conclusions drawn (Passfield et al. 1998).

Patterson et al. (1983) suggested that preferred cadence may be selected not to minimise energy expenditure, but to reduce pedal forces. They speculated that this may lessen the extent of peripheral fatigue developed during exercise. The combined findings of more recent studies support the veracity of this hypothesis. The adoption of a faster cadence, particularly at higher work rates may be beneficial in improving muscle blood flow. Sejersted et al. (1984) have suggested that isometric forces in excess of 15% of MVC are sufficient to impair muscle blood flow. Higher cadences are associated with reduced peak pedal forces which may minimise venous or even arterial occlusion (Sjøgaard, 1978; Künstlinger et al. 1985; Sargeant and Greig, 1988; Patterson and Moreno, 1990). These lower peak forces may also alter fibre recruitment patterns (Sargeant, 1994). In 1992 Ahlquist et al. found that Type II muscle fibres were more heavily glycogen depleted following 30 minutes at 85% $\dot{V}O_{2peak}$ when pedalling at 50 $rev \cdot min^{-1}$ as opposed to 100 $rev \cdot min^{-1}$. These results may in part explain the findings of a later study of Nickleberry and Brooks (1996) who examined the time to exhaustion at 75% $\dot{V}O_{2peak}$ in recreational and competitive cyclists riding at 50 and 80 $rev \cdot min^{-1}$. Unsurprisingly, Nickleberry and Brooks found the competitive cyclists were able to last almost twice as long as the recreational riders at both cadences. Both groups however, increased their time to exhaustion at the higher cadence.

Takaishi et al. (1994, 1996) examined the effect of cadence on the rate of fatigue development. They used the slope of iEMG over time, whilst riding at 85% $\dot{V}O_{2peak}$ for 15 minutes, as an index of fatigue. In the later study these authors found the minimum in the relationship between iEMG slope and cadence to occur at a higher cadence than that for oxygen uptake. Takaishi et al. concluded that cyclists prefer higher cadences in order to minimise neuromuscular fatigue. Marsh and Martin (1997) compared preferred and optimal cadence in two trained subject groups (cyclists and runners) and a untrained group. Marsh and Martin found no differences in preferred cadence between the cyclists and runners, suggesting that training specificity was not a factor in pedal rate selection. The non-trained group however, preferred a cadence much closer to their optimal and significantly slower than that of the trained groups. Marsh and Martin inferred from these results that the greater aerobic capacity of their trained groups enabled the selection of a less efficient cadence in order to minimise muscle fatigue. The non-trained group cannot afford to increase energy expenditure unnecessarily as this will result in a significant increase in their (limited) oxygen uptake and consequently greater metabolic strain.

2.4.3 Fibre type

Goldspink (1978) reviewed evidence that Type I isolated muscle has been demonstrated to be more efficient than Type II. During isometric muscle actions Goldspink reports Type I fibres to be over three times more economical than Type II. Type I muscle fibres are also thought to be more efficient for concentric contractions although, the shortening velocity is acknowledged as an important determinant of contraction efficiency. At high velocities of shortening the Type II muscle fibres are most efficient. As the velocity of shortening is reduced below 2 muscle lengths·s^{-1}, Type I muscle fibres become the most efficient. The velocity of peak efficiency is found at approximately 1 and 5 muscle lengths·s^{-1} for Type I and II muscle fibres respectively, with peak efficiency some 50% higher in Type I fibres. These conclusions are also supported by Wendt and Gibbs (1973) and Crow and Kushmerick (1982), who both conclude that isometric contractions are approximately 3 times more economical in Type I mammalian muscle. For cycle exercise the optimal shortening velocity for Type I muscle is thought to correspond to a cadence of 60-80 rev·min^{-1} (Coyle et al. 1992; Sargeant 1996). Further research is warranted to

confirm these conclusions, as little data is available on the efficiency of human muscle during dynamic contractions.

Coyle et al. (1992) have suggested that most of the variability in efficiency observed in well-trained cyclists is due to differences in fibre type composition. Muscle fibre type distribution has been demonstrated to correlate with both GE and ΔE (Suzuki, 1979; Coyle et al. 1992; Horowitz et al. 1994). Suzuki examined efficiency at two cadences, in 6 subjects with widely differing muscle fibre compositions. Three subjects possessed a mean Type I composition of the vastus lateralis of 78%, whilst the other subjects were found to have a similar percentage of Type II fibres. No difference was observed in ΔE between the two groups when pedalling at 60 rev·min^{-1}, whilst the high Type II% group were more efficient at 100 rev·min^{-1}. In contrast, the more recent studies of Coyle and colleagues found the percentage of Type I fibres in the vastus lateralis muscle to be positively correlated with GE and ΔE and also efficiency during a novel two-legged extension task (Coyle et al. 1992; Horowitz et al. 1994). Based upon their data Coyle et al. (1992, pp. 785) provide an equation for estimating GE from percentage of Type I fibres.

Equation 2.2

$$GE = 0.0671\ (\%\text{Type I}) + 16.991$$

Moreover, in the later study (Horowitz et al. 1994), the authors provided evidence for the robust nature of this relationship by dividing their subjects into two groups based upon GE (i.e. high and low) finding a corresponding dichotomy in the fibre type composition of the two groups.

Further evidence in support of Coyle et al's findings may be found in the study of Jansson and Kaijser (1987). These authors compared metabolic responses in trained cyclists and untrained individuals exercising at 65% $\dot{V}O_{2peak}$. They observed their trained subjects had a significantly higher percentage of Type I fibres (70% vs. 40%) and a greater GE (22% vs. 19%). The close correspondence of the studies of Coyle et al. (1992) and Jansson and Kaijser (1987) is demonstrated by substituting the data for the trained and untrained groups of Jansson and Kaijser into Equation 2.2 of Coyle et al. The calculated GE values

of 22% and 20%, based upon the percentage of Type I fibres measured by Jansson and Kaijser, are very similar to those actually reported earlier for this study.

Not all researchers are in agreement on the issue of fibre type and efficiency (Suzuki, 1979; Medbø, 1990). Medbø (1990) suggested studies documenting a progressive drift in $\dot{V}O_2$ during prolonged exercise can be interpreted as evidence that fibre type does not affect efficiency. This postulate is clearly incorrect, as close examination of the studies cited by Medbø and others of similar nature demonstrates (see section 2.7.1). Medbø's study found O_2 uptake to increase linearly with exercise intensity in 16 subjects with no correlation between %Type I fibres and efficiency. Medbø assumed a linear relationship between work rate and $\dot{V}O_2$, an assumption which is increasingly regarded as incorrect (Stuart et al. 1981; Green and Dawson, 1995; Zoladz et al. 1995). This assumption may mask a difference in efficiency of Type I and II fibres (Suzuki, 1979; Medbø, 1990), as Barstow et al. (1996) have demonstrated an increased oxygen cost of high intensity exercise to be associated with a greater percentage of Type II fibres. Experimental differences in subject numbers, training background and cadence may have contributed to the divergent results of the studies of Suzuki (1979) and Coyle et al. (1992). Suzuki collected muscle samples from the quadriceps femoris of only 6 subjects (PE students and laboratory staff). The Coyle group's data were gathered from a large number of fibres from the vastus lateralis muscle of 2-3 times as many highly trained cyclists. Furthermore, Suzuki employed cadences of 60 and 100 rev·min^{-1} in contrast to the earlier Coyle study where cadence was standardised at 80 rev·min^{-1}. Coyle et al. estimate that this cadence corresponds to the optimal rate of muscle shortening for peak contraction efficiency of the Type I fibres. Furthermore, the results and conclusions reported by Suzuki have been criticised (Stuart et al. 1981). Stuart et al. observe that Suzuki failed to point out that GE tended to be higher at both cadences and low work rates for the high percent Type I group.

2.4.4 Muscle action

There are large reported differences in the efficiency of different muscle actions (Ryschon et al. 1997). This was elegantly demonstrated by Abbot et al. (1952) who created a cycle ergometer which enabled two subjects to exercise back to back, with one pedalling

forward and the other trying to resist the pedalling motion while pedalling backwards. Abbot et al. found both a much higher economy and increased time to exhaustion with negative as opposed to positive work. Although some co-contraction of leg muscles is thought to occur during cycling (Hull et al. 1988) the extent of this is very small. The majority of effective torque generated in cycling is thus thought to be achieved primarily by concentric muscle action.

2.4.5 Body position and size

Body position during cycling and body size have been demonstrated to affect GE. Nordeen-Snyder (1977) and Shennum and deVries (1976) examined the effect of large changes in saddle height on economy. Both studies found the relationship between saddle height and economy to produce a distinct minimum, with high saddle positions causing a greater decrease in economy. Ericson and Nisell (1988) and Sanderson (1991) have found that the effective force application on the pedal remains unchanged with changes in saddle height. These combined findings suggest the change in economy with saddle position may be related to alterations in the force-length relationship of the active muscle groups.

The effect of other changes in cycling position (seat tube angle and indirectly hip and trunk angle) have been found to affect economy in several investigations (Heil et al. 1995, 1997; Gnehm et al. 1997; Price and Donne, 1997), but not all (Origenes et al. 1993). Heil et al. (1995) found that a more vertical seat tube angle improved economy in a mixed group of triathletes and cyclists; a finding that was confirmed in experienced cyclists by Price and Donne (1997). A later study by Heil et al. (1997) extended these observations by demonstrating that seat tube, hip and trunk angle interact to confound the determination of an optimal position. The riders' preferred position tended to correspond to the most economical one. Origenes et al. (1993) did not detect an effect of upper body posture on $\dot{V}O_2$ or related ventilatory measures. It is notable that in this study inexperienced cyclists were recruited as subjects. It is possible that training adaptations resulting in a greater efficiency are specific to the particular training position and may be influenced by the muscle force-length relationship. The determination of an optimal cycling position is problematic as any change may affect the range of motion in several joints.

The oxygen cost of pedalling is found to be influenced by lower limb mass (Cotes, 1969; Anton-Kuchly et al. 1984; Berry et al. 1993; Francescato et al. 1995). Comparative studies of normal and obese individuals (Anton-Kuchly et al. 1984) and studies using leg weights to increase lower limb mass (Francescato et al. 1995) conclude that a greater lower limb mass significantly increases the oxygen cost of pedalling.

2.4.6 Pedalling dynamics

Somewhat surprisingly the pattern of force application on the pedal does not appear to influence cycling economy (Lafortune and Cavanagh, 1983; Coyle et al. 1991a). To be effective all forces applied to the pedal should act tangentially to the crank. Any force applied in other directions will result in twisting or shearing moments being created about the pedal or crank. Coyle et al. found that better cyclists are not characterised by a higher index in the effectiveness of force application on the pedal. Indeed, the elite cyclists were found to apply pedal forces less effectively, but were able to generate a higher torque through the propulsive phase of the pedal cycle. Consequently, it appears that research aimed at optimising pedalling efficiency appears to be ill-founded (Lafortune and Cavanagh, 1980; Lafortune et al. 1983). In contrast, Lafortune and Cavanagh (1983) found that the use of toe-clips to secure the foot to the pedal resulted in a significant change in the pattern of force application and a decrease in exercising $\dot{V}O_2$ (2.08 $L \cdot min^{-1}$ vs. 2.03 $L \cdot min^{-1}$). However, net efficiency showed only a low correlation with pedalling effectiveness over all subjects and trials. These findings may well be due to the inexperienced cyclists studied. This conclusion is supported by the study of Cavanagh and Sanderson (1986), who found that foot position on the pedal did not influence pedalling mechanics in elite cyclists.

Cullen et al. (1992) examined the effect of non-circular chainrings on $\dot{V}O_2$ in trained cyclists at 3 different pedalling rates and 2 work rates. In contrast to the earlier the study of Henderson et al. (1977), who did not use experienced cyclists, no difference was found. In summary, little evidence exists to support the notion that experienced cyclists can further increase their efficiency by modifying aspects of their pedalling technique.

Inexperienced cyclists may benefit from the use of toe-clips, although further research is warranted to verify these conclusions.

2.4.7 Fitness

Studies on the relationship between fitness and efficiency have yielded contradictory findings. Recently Nickleberry and Brooks (1996) found no difference in either GE or ΔE when comparing recreational and competitive cyclists. This interpretation may have been confounded by the relatively small difference in fitness between the 12 riders in the two groups. Only the slower cadence of 50 rev·min^{-1} resulted in a significant difference in $\dot{V}O_{2peak}$ (3.74 vs. 3.22 L·min^{-1}), whilst testing conducted at 80 rev·min^{-1} failed to reveal a difference. Stuart et al. (1981) examined efficiency values in 5 sprint and 5 endurance trained runners with differing $\dot{V}O_{2peak}$. They found no difference in ΔE although GE tended to be higher in the endurance trained athletes. These authors concluded that ΔE is not affected by differences in $\dot{V}O_{2peak}$ or type of training. A low statistical power and cycling experience in the subjects may have masked an apparent trend for higher efficiencies in the fitter subjects.

A significant difference in GE (22 vs. 19%) between 5 trained and 5 untrained subjects was observed in the study of Jansson and Kaijser (1987). In this study large differences were observed in $\dot{V}O_{2peak}$ (3.29 vs. 4.81 L·min^{-1}), oxidative enzyme activities (100% greater in trained) and muscle fibre composition (70% vs. 40% Type I fibres) of the 2 groups. This raises the possibility that it is not fitness per se that influences GE but the cumulative effects of training on muscle morphology. It is also possible that the higher GE of the trained subjects reflects the higher absolute work rate at which they were tested.

2.4.8 Ergometry

The importance of using cycling specific ergometry in exercise testing has been variously acknowledged (Strømme et al. 1977; Hagberg et al. 1981; Merrill and White, 1984; Keen et al. 1991; Kenny et al. 1995; Gnehm et al. 1997). Few researchers however, have examined the effects of different ergometer characteristics on the physiological responses

to exercise. Patterson et al. (1983) compared physiological and perceptual responses in 8 inexperienced cyclists whilst exercising on an ergometry system with 2 different flywheel masses at several cadences (40-90 rev·min^{-1}). Varying the flywheel mass (1.5 kg to 35.9 kg) with the resulting change in the moment of inertia (from 0.1 kg m^2 to 1.65 kg m^2) did not result in differing $\dot{V}O_2$ except at a cadence of 50 rev·min^{-1} and 30% $\dot{V}O_{2peak}$. No difference was found in the pattern of force application with either flywheel. By contrast, Kenny et al. (1995) found the $\dot{V}O_2$ - heart rate relationship to be significantly altered under field cycling conditions compared with laboratory riding on an ergometer or treadmill. The authors concluded that care should be exercised when attempting to equate cardio-respiratory measures from laboratory and field testing conditions.

A recent study by this author and colleagues (Passfield et al. 1998) revealed significant differences in economy, peak aerobic power output (PAPO) and $\dot{V}O_{2peak}$ between two forms of laboratory based, cycling specific ergometry. Experienced cyclists rode a standard cycle on both a cycling specific Kingcycle ergometer (Kingcycle, High Wycombe, U.K.) and on a treadmill. Treadmill riding resulted in higher cycling economy, PAPO and $\dot{V}O_{2peak}$ despite subjects using the same cycle and power measuring system. Thus, it appears that differing forms of ergometry may influence efficiency in experienced cyclists.

2.5 Summary

The preceding sections have examined GE and economy and reviewed a number of factors that have been suggested to alter cycling efficiency or economy. It is apparent that any measurement of efficiency based upon pulmonary gas collection during cycle ergometry cannot be accepted simply as muscular efficiency. Despite this there is strong evidence that muscular efficiency contributes significantly to the determination of both GE and ΔE. Type I muscle fibres have been reported to contract with greater efficiency than fast twitch muscle during the normal range of speeds employed in endurance cycling, and a strong relationship between efficiency and muscle fibre composition has been demonstrated. Many factors have been found to modify efficiency, particularly cadence, power output and changes in cycling position. Studies on the pattern of force application suggests that the interpretation of efficiency is not significantly influenced by this factor.

Comparison of efficiency measures are therefore, only valid under conditions where cadence, power output and cycling position are strictly controlled. It is a working assumption of this thesis that changes in GE determined from pulmonary gas exchange reflect a change in muscular efficiency, where the above factors have been controlled. The effect of endurance exercise on GE, and the veracity of the assumption that it reflects a change in muscular efficiency are examined in the following sections.

2.6 Cycling economy and endurance performance

Endurance performance has been clearly demonstrated to be influenced by cycling economy (Horowitz et al. 1994). As discussed previously (section 2.4.3), Coyle et al. (1992) found a significant correlation between %Type I vastus lateralis fibres and cycling GE and ΔE. This finding was also repeated in a later study (Horowitz et al. 1994) investigating the effect of fibre type and cycling economy on endurance performance. Horowitz et al. divided their highly trained subjects, to create two groups with high and normal percentages of Type I fibres. The high Type I group were able to produce an average 9% greater power output for the same average $\dot{V}O_2$ during a 1 h performance trial. The difference in performance economy was independent of $\dot{V}O_{2peak}$ and indices of metabolic strain.

Recently, this author has demonstrated that acute reductions in GE (and therefore economy), occur during prolonged endurance exercise in elite cyclists (Passfield and Hale, 1997). Furthermore, those cyclists finishing the endurance trial with a higher GE were better able to maintain PAPO. This was evidenced by a strong correlation ($r = 0.94$) between GE at the end of exercise and the change in PAPO induced by the endurance trial. The combined observations of Horowitz et al. (1994) and Passfield and Hale (1997) suggest that acute modifications in GE may be reflected in aerobic performance. Further research is required to test this hypothesis.

2.7 Endurance exercise, $\dot{V}O_2$ drift and GE

During prolonged moderate to heavy intensity cycle exercise[2] a progressive rise in $\dot{V}O_2$ (i.e. reduction in cycling economy) is commonplace (c.f. Vollestad et al. 1984; Sahlin et al. 1990; Coyle et al. 1991b; Hamilton et al. 1991; Hagan et al. 1992). The gradual increase in $\dot{V}O_2$ over time (hereafter referred to as $\dot{V}O_2$ drift) is frequently unacknowledged or given only cursory attention (e.g. Hamilton et al. 1991; Sahlin et al. 1990). The temporal similarity between $\dot{V}O_2$ and heart rate drift may have led to an unquestioning acceptance of both these only partially related phenomena. The $\dot{V}O_2$ drift is commonly assumed to reflect a shift in substrate utilisation from carbohydrate to fat metabolism and its consequent increase in exercising $\dot{V}O_2$. As discussed previously (section 2.2), the calculation of GE is derived from the energy equivalent of the O_2 consumed. Consequently, GE is unaffected by a change in $\dot{V}O_2$ caused by a shift in substrate utilisation. An increased $\dot{V}O_2$ (i.e. small $\dot{V}O_2$ drift) combined with a reduced RER, results in a lower calculated energy equivalent for the O_2 consumed and thus a constant GE. Any decrease in GE observed during exercise therefore, implicates a substrate independent mechanism. Unfortunately, changes in GE during endurance exercise have not been generally reported.

2.7.1 Changing GE and $\dot{V}O_2$ drift

Despite a $\dot{V}O_2$ drift being generally observed during endurance exercise, few studies have attempted to provide an insight into its aetiology. Where changes in GE have been considered, an increased cardio-respiratory cost has been suggested to be the cause (Sahlin et al. 1990). This assumption is not thought to be correct as illustrated by Sahlin et al.'s own data. These researchers found $\dot{V}O_2$ to increase significantly from 20 min (2.49 $L \cdot min^{-1}$) to exhaustion (2.68 $L \cdot min^{-1}$). This change in $\dot{V}O_2$ was accompanied by a reduction in RER from 0.98 to 0.96. To compensate for the shift in substrate utilisation inferred from the change in RER, a 0.5% increase in $\dot{V}O_2$ to 2.50 $L \cdot min^{-1}$ is required to maintain GE (Lusk, 1924). The additional 0.18 $L \cdot min^{-1}$ increase in $\dot{V}O_2$ ($\approx$ 14%) represents a corresponding decrease in GE and must be accounted for by other factors.

[2] See Figure 1.3 for definition of exercise intensities

Aaron et al. (1992) have calculated ventilation in the range reported by Sahlin et al. (increasing from 68 to 87 $L \cdot min^{-1}$) to cost 1.3 ml O_2 per additional L. Clearly, even after the increased cost of ventilation is deducted a substantial part of the $\dot{V}O_2$ drift remains to be attributed to additional mechanisms. Few data exist quantifying the oxygen cost of cardiac work during moderate intensity exercise. There is however, no reason to assume that cardiac work, or its O_2 cost, increases markedly during prolonged exercise independently of other known mediators of exercising $\dot{V}O_2$.

Hagberg et al. (1978) endeavoured to determine the primary causes of the $\dot{V}O_2$ drift observed during a 20 minute exercise trial. In this study 18 subjects attempted to complete 2 trials at 65% and 80% $\dot{V}O_{2peak}$. A significant increase in $\dot{V}O_2$ from the 5th to 20th minute was found in around 80% of trials for both conditions. Hagberg et al. calculated that changes in substrate utilisation, blood lactate, ventilation, and rectal temperature accounted for only 60% of the rise in $\dot{V}O_2$ found whilst exercising at 65% $\dot{V}O_{2peak}$. The authors speculated that changes in catecholamines and (muscular) efficiency were responsible for the remaining change in $\dot{V}O_2$ found at the lower intensity. Although not reported directly, a decreased GE is implicit in these results. The veracity of Hagberg and colleagues' calculations is questionable, however, as the calculations for the higher work rate considerably overestimated the $\dot{V}O_2$ drift.

More recently Hagan et al. (1992) in a similar style of study, examined the effect of cadence (60 and 90 $rev \cdot min^{-1}$) on responses to 45 minutes of exercise. Five subjects exercised at 50% and 65% $\dot{V}O_{2peak}$ in both cadence conditions. A significant $\dot{V}O_2$ drift was evident from 10 to 45 minutes in all 4 trials, but was attenuated at the cadence of 60 $rev \cdot min^{-1}$. The exercise intensity did not influence the rate of $\dot{V}O_2$ drift. In agreement with Hagberg et al, (1978), the authors could not explain the magnitude of the change in $\dot{V}O_2$, with around 65% of the increase being unaccounted for. Noting a significant reduction in GE over time, Hagan et al. proposed neuromuscular factors were responsible for the $\dot{V}O_2$ drift.

Only one study has examined changes in GE during endurance exercise in trained cyclists (Passfield and Hale, 1997). These researchers examined the change in GE for 8 elite

cyclists riding for 3 hours at 50% of PAPO (≈ 60% $\dot{V}O_{2peak}$). Over the course of the endurance trial GE declined significantly from 20.1% at 0.5 h to 19.3% at 3 h. Peak aerobic power output measured immediately after the endurance trial was also significantly reduced by an average of 30 watts.

In summary, endurance exercise is associated with a gradual increase in oxygen consumption. Despite its common occurrence very few investigators have examined the cause of $\dot{V}O_2$ drift during prolonged exercise. A reduction in GE seems to drive the $\dot{V}O_2$ drift, as changes in substrate utilisation cannot account for the additional O_2 required. The $\dot{V}O_2$ drift cannot readily be explained by an increase in cardio-respiratory work. The next section considers alternative explanations.

2.7.2 Source of change in GE

There are several possible explanations for the change in GE found during endurance exercise. These can be broadly divided into the following categories.

Arising from the exercising muscle:

I. A reduction in the effective application of force on the pedals.

II. An increase in metabolism, for example induced by changes in muscle temperature, hydration, circulating hormones and catecholamines, lactate turnover.

III. An increased energy cost of muscular force production.

IV. A reduction in the force-generating capacity of the active fibres and a concomitant increase in muscle fibre recruitment to compensate.

Arising outwith the exercising muscle:

I. An increase in unmeasured work, e.g. increase in muscular work not contributing directly to force production.

II. Increased metabolism in previously less active tissues and organs.

Strong evidence exists to support the contention that the $\dot{V}O_2$ drift arises primarily from the active muscle. González-Alonso et al. (1997) demonstrated a progressive increase in muscle $\dot{V}O_2$ during prolonged exercise in the heat. A similar conclusion can be drawn from the data of Sahlin et al. (1990, pp: 835, Table 1) in which subjects rode to exhaustion at 74% $\dot{V}O_{2peak}$. From the a-v O_2 differences and leg blood flow data it can be

calculated that leg O_2 utilisation increased by 7%, similar to the reported increase in pulmonary $\dot{V}O_2$. This congruence in leg and pulmonary $\dot{V}O_2$ is supported by the findings of several other studies (Sullivan et al. 1987; Poole et al. 1991, 1992; Poole, 1994; Grassi et al. 1996). These authors have documented that up to 90% of pulmonary $\dot{V}O_2$ arises directly from muscle metabolism, even when cycling at high exercise intensities. The studies of both Coyle et al. (1992) and Horowitz et al. (1994) reviewed earlier, provide further indication that muscle metabolism is a major determinant of exercising $\dot{V}O_2$ and hence GE. Finally, repeated isometric actions are also linked with a progressive increase in $\dot{V}O_2$ (Vollestad et al. 1990; Sejersted and Vollestad, 1992; Saugen and Vollestad, 1996). This increase in $\dot{V}O_2$ has been directly attributed to changes in muscle contraction efficiency.

2.7.3 Mechanisms for change in GE

I. *A reduction in the effective application of force on the pedals.*

The contribution of changes in the effective application of forces upon the pedal are minimal, as peak force generated on the pedal within one revolution does not appear to be altered. Sjøgaard (1978) did not find peak tension per pedal thrust altered in subjects cycling to exhaustion at 85% $\dot{V}O_{2peak}$. Similar results are reported by Rademaker et al. (1994a) who found no change in peak isokinetic cycling power output following 45 min at 80% $\dot{V}O_{2peak}$. Moreover, the lack of correlation between the pattern of force application and GE (Lafortune and Cavanagh, 1983; Coyle et al. 1991a) implies changes in pedalling technique have limited scope to affect GE. It is acknowledged however, that the results of the studies cited above do not preclude the possibility that changes in force application pattern contribute significantly to $\dot{V}O_2$ drift. Further research is required to verify this conclusion.

II. *Up-regulation of muscle metabolism induced by e.g. changes in muscle temperature, hydration, circulating hormones and catecholamines, lactate turnover.*

Additional support for the contribution of an intrinsic modification of muscle function to the reduction in GE arises from the observation that many other putative mediators do not influence $\dot{V}O_2$ drift. Hagberg et al. (1978) and Hagan et al. (1992) accounted for

changes in substrate utilisation, ventilation, blood lactate removal and body temperature, but still could not explain a significant portion of the $\dot{V}O_2$ drift detected. Poole and Richardson (1997) reviewed likely mechanisms of the $\dot{V}O_2$ slow component. These authors discount the significance of cardiac, ventilatory and auxiliary muscle work in increasing $\dot{V}O_2$ at exercise intensities higher than those of interest here. Increases in lactate metabolism, catecholamine concentration, muscle temperature, extracellular potassium and muscle pH are also discounted by Poole and Richardson. Despite the obvious corollaries Gaesser and Poole (1996) have stated that $\dot{V}O_2$ drift of moderate intensity exercise should be viewed as separate and distinct from the O_2 slow component that characterises higher intensity exercise (see section 1.6). Nonetheless, all the factors discussed above are known to be related to exercise intensity. This observation suggests any changes observed at moderate, as opposed to heavy or severe exercise intensities, are likely to be of even less significance.

Circulating catecholamines have been variously implicated in the aetiology of the $\dot{V}O_2$ drift (Hagberg et al. 1978; Kalis et al. 1988; Coyle et al. 1991b; Hamilton et al. 1991; Hagan et al. 1992). The combined results of Coyle et al. (1991b) and Hamilton et al. (1991) demonstrated that the $\dot{V}O_2$ drift was abolished by a hyperglycaemic clamp (10 $mmol \cdot L^{-1}$). The eradication of the $\dot{V}O_2$ drift was thought to stem from the intravenous glucose infusion lowering circulating catecholamine concentrations (Hamilton et al. 1991). A decrease in GE may be calculated, albeit under unconventional metabolic circumstances. The hyperglycaemic clamp maintained CHO oxidation throughout the 2 h trial, so tbat the minimal increase in $\dot{V}O_2$ (3.20 vs. 3.24 $L \cdot min^{-1}$) was, unusually, combined with an increase in RER (0.88 to 0.90) late in exercise. Assuming power output remained constant under both conditions, the change in GE was reduced by half for hyperglycaemia compared with control (21.0% to 20.7% and 20.8% to 20.1% respectively). Selective β-blockade (β_2) has also been demonstrated to prevent a $\dot{V}O_2$ drift (Kalis et al. 1988). On three occasions 14 untrained males cycled in the heat (32 °C) at 40% $\dot{V}O_{2pcak}$ for 90 min, having been given a β–blocker (propranolol [β_{1+2}], atenolol [β_1]) or a placebo. Exercise following the propranolol administration was not associated with a $\dot{V}O_2$ drift in contrast to the other conditions. Interpretation of this study is potentially confounded by the low relative intensity and $\dot{V}O_2$ ($\approx$ 1.63 $L \cdot min^{-1}$) which

probably reduced the extent of the $\dot{V}O_2$ drift (Epstein et al. 1984). Also, the rate of energy expenditure in the propranolol trial was lower than for the other conditions ($\dot{V}O_2$ 1.58 vs. 1.68 $L \cdot min^{-1}$ for placebo). Interestingly a common observation of both Coyle et al. (1991b) and Kalis et al. (1988) was that the reduced $\dot{V}O_2$ drift was accompanied by maintenance of CHO oxidation late in exercise.

The notion that catecholamines are linked to $\dot{V}O_2$ drift is directly contradicted by the results of several studies (Jansson et al. 1986; McConell et al. 1994; Mora-Rodríguez et al. 1996; Febbraio et al. 1998). The studies of Febbraio, Jansson, Mora-Rodríguez and colleagues, have demonstrated that elevating circulating catecholamines by infusion does not result in an increased $\dot{V}O_2$ drift during prolonged exercise. Furthermore, Mora-Rodríguez et al. and McConell et al. demonstrated that reducing plasma catecholamine concentration during endurance exercise by ingestion or infusion of CHO also does not influence the magnitude of the $\dot{V}O_2$ drift. Particularly convincing are the results of Mora-Rodríguez et al. (1996). These researchers compared the effects of a hyperglycaemic clamp (> 11 $mmol \cdot L^{-1}$), adrenaline infusion and saline infusion on responses to exercise (120-150 min, 65% $\dot{V}O_{2peak}$) in the heat (33 °C). Significant differences between infusion conditions were found for several parameters including plasma catecholamines, insulin, oesophageal temperature and heart rate, but not for $\dot{V}O_2$. The results of Mora-Rodríguez et al. challenge the findings of Coyle et al. (1991b) and Hamilton et al. (1991) whose combined data argue that a hyperglycaemic clamp attenuates the $\dot{V}O_2$ drift. It is possible that these equivocal results arise from the different environmental conditions of the respective studies.

Endurance exercise in the heat and dehydration have been variously demonstrated to result in a higher core temperature, muscle temperature and plasma catecholamines as well as greater glycogen utilisation (Coyle et al. 1991b; Hamilton et al. 1991; Montain and Coyle, 1992b; Febbraio et al. 1994; Hargreaves et al. 1996; González-Alonso et al. 1997). Yet none of the above studies found these factors to be associated with an effect on $\dot{V}O_2$ drift. Coyle and co-researchers (Coyle et al. 1991b; Hamilton et al. 1991) studied 8-10 well-trained cyclists riding for 2 hours (70% of $\dot{V}O_{2peak}$ and 22 °C). A 6% increase in $\dot{V}O_2$ was evident in both dehydration and euhydration conditions. Changes in substrate

utilisation alone cannot account for this $\dot{V}O_2$ drift. The studies of Montain and Coyle (1992b), Febbraio et al. (1994) and Hargreaves et al. (1996) all examined effects of heat stress and/or dehydration, but none of these authors report a consequent increase in exercise $\dot{V}O_2$.

III. & IV. *An increased energy cost of muscular force production. A reduction in the force-generating capacity of the active fibres and a concomitant increase in muscle fibre recruitment to compensate.*

The study of González-Alonso et al. (1997) referred to in the previous section, examined muscle $\dot{V}O_2$ in seven trained subjects exercising at 60% $\dot{V}O_{2peak}$ in the heat to exhaustion without fluid, and for the same duration with fluid. No difference in muscle $\dot{V}O_2$ was evident between hydration conditions, although a significant $\dot{V}O_2$ drift was found. Significant differences between hydration conditions were found however, for core and muscle temperature, muscle lactate, leg blood flow, arterial adrenaline concentration, and muscle glycogen utilisation. In a personal communication, González-Alonso (1998) concludes, "the progressive increase in oxygen consumption during exercise is confined to the exercising skeletal muscle".

A likely mechanism for the $\dot{V}O_2$ drift is a progressive recruitment of muscle fibres. Vollestad et al. (1984) found that prolonged exercise at 75% $\dot{V}O_{2peak}$ resulted in glycogen depletion from both Type I and IIA fibres from the onset of exercise. Later in exercise a glycogen decrease was observed in Type IIAB and then in Type IIB fibres. The results of Vollestad et al. suggest that a progressive recruitment of Type II muscle fibres occurs during prolonged exercise to exhaustion. The $\dot{V}O_2$ drift may arise from the increased energy cost of recruiting more muscle fibres to produce the same work rate. This effect is likely to be magnified by the lower aerobic efficiency of these additional Type II fibres, as discussed previously (2.4.3). A possible confounding influence on the study of Vollestad et al. is mild glycogenolysis in inactive fibres. Hargreaves, (1995) notes that preliminary evidence for glycogen depletion in inactive muscle groups has been recorded in rats. Bonen et al. (1985) found muscle glycogen was lowered not just in the active leg, but also the inactive leg during one legged cycling. The inactive leg showed no sign of EMG

activity and Bonen et al. urge caution on the interpretation of fibre recruitment based on glycogen depletion studies.

The studies of Vollestad and colleagues provide an alternative suggestion for the cause of $\dot{V}O_2$ drift. During repeated isometric muscle actions to exhaustion a drift in exercise $\dot{V}O_2$ similar to that seen in dynamic exercise has been found (Vollestad et al. 1990; Sejersted and Vollestad, 1992; Saugen and Vollestad, 1996). The additional $\dot{V}O_2$ is seen to arise directly from the active muscle mass and is associated with a gradual decline in MVC. Saugen and Vollestad, (1996) examined metabolic heat production during a bout of repeated isometric actions at 30% MVC. These researchers found that a progressive increase in the rate of temperature rise during each muscle action was paralleled by a 76% increase in $\dot{V}O_2$. Furthermore, comparison with the rate of temperature rise at 50% MVC suggested that progressive fibre recruitment did not fully account for the $\dot{V}O_2$ drift. Saugen and Vollestad postulate that the $\dot{V}O_2$ drift is due to a change in the energy cost of contraction. It is speculated that a similar phenomenon may be found during dynamic exercise. An exercise induced change in force production and energy turnover within the active fibres, coupled to or in conjunction with, a progressive fibre recruitment (Vollestad et al. 1984) could explain the ΔGE observed.

In conclusion, the $\dot{V}O_2$ drift observed during prolonged exercise arises largely from the active muscle and is associated with a reduction in GE. Heat stress, dehydration and associated circulatory and metabolic changes can only explain a minor portion of the $\dot{V}O_2$ drift observed. Two causes acting singly or in combination have been suggested to explain the major proportions $\dot{V}O_2$ drift: an exercise-induced increase in the energy cost of muscle contraction and/or a reduction in the force-generating capacity of the active fibres.

2.8 Summary of cycling gross efficiency

An interest in the efficiency of human movement can be traced to the antecedents of modern human physiological research. Since this time considerable data have been published on human efficiency. Of the many factors know to influence GE, cadence,

power output and cycling position have been demonstrated to exert a significant effect. These parameters must be strictly controlled throughout comparative work on efficiency.

Type I muscle fibres are thought to contract with a greater efficiency than Type II fibres at cadences typical of endurance cycling. This morphological characteristic of the active skeletal muscle mass has been established to be largely responsible for determining GE and the success in endurance performance of individuals with similar aerobic fitness. Furthermore, a progressive $\dot{V}O_2$ drift has been demonstrated to occur during prolonged moderate intensity exercise in elite cyclists and is associated with a reduced GE. It is speculated that following prolonged exercise any acute modification of GE may be mirrored in endurance performance. The causes of the $\dot{V}O_2$ drift remain to be established, but are thought to relate directly to a change in muscle function.

[redacted]

Chapter 3: Maximal muscle function and endurance exercise

3.1 Maximal muscle function

The fundamental properties of skeletal muscle structure and fibre composition are thought to dictate its force-velocity and length-tension relationships (Edgerton et al. 1986; Jones, 1992; Sargeant, 1994). The relation between muscle force-velocity and heat production is a rectangular hyperbola and was first described by A.V. Hill (1938) from isolated frog muscle. Wilkie (1950) was subsequently able to demonstrate that the force-velocity curve can be used to characterise simple dynamic movements in humans. The velocity of movement and the muscle length influence the maximum muscle force or power output that can be generated. During dynamic muscle action such as cycling, the force-velocity curve is best represented as a power-velocity relationship. The power-velocity relationship approximates an inverted 'U' in form, with peak power output occurring at the optimal cadence (V_{opt}) of approximately 120 rev·min^{-1} (Sargeant et al. 1981; Sargeant et al. 1984; McCartney et al. 1985).

3.1.1 Isometric exercise

Isometric exercise testing allows a clear insight into muscle function as the effect of changes in muscle action velocity is prevented and muscle length may be controlled. Further, direct electrical stimulation may provide greater insight into the mechanisms of fatigue whilst precluding any possible effects of central fatigue. Extrapolation of findings from studies employing isometric protocols are of questionable value in furthering understanding of the demands of dynamic muscle function, since muscle fatigue has been demonstrated to alter the power-velocity curve (de Haan et al. 1989). The impact of such changes cannot be detected from static exercise testing. Moreover, isometric muscle action requiring forces above approximately 15% MVC is thought to generate sufficient intra-muscular pressure to occlude blood flow (Sejersted et al. 1984). Consequently, fatigue resulting from sustained or repeated isometric muscle actions may be critically influenced by the effects of restricted blood flow.

3.1.2 Dynamic exercise

Strict experimental control is complicated for dynamic muscle function assessment. The analysis of even simple, single joint movements is problematic (Wilkie, 1950). During complex movements such as cycling, muscle action velocity and length change constantly. In cycle ergometer testing situations where cadence is not tightly controlled, the assessment of muscle function may be confounded by changes in action velocity with respect to V_{opt}. Furthermore, the effects of fatigue may serve to amplify these problems. Isokinetic cycling implies only that muscle function is tested at a constant cadence, as the force-velocity relationship is appreciably modified by the limb lever system (McCartney et al. 1985). Also, Kannus (1994) notes that isokinetic dynamometry represents an activity rarely experienced outside of the laboratory situation. Isokinetic dynamometry does not therefore, provide a perfect solution, but may offer the opportunity for further insight into the factors limiting maximal dynamic exercise.

3.1.2.1 Fibre type

Muscle fibre composition not only influences efficiency as discussed previously (section 2.4.3), but also has a profound effect on the power-velocity relationship. The maximum shortening velocity of Type II fibres is approximately 3 to 4 times greater than that of Type I fibres (Sargeant, 1994). Type II fibres achieve higher power outputs at faster contraction velocities but are inherently more fatiguable. Type I fibres appear to be specialised for efficient protracted use at lower power outputs and contraction velocities. Thus, in situations requiring a high maximum power output or very rapid rates of muscle contraction, a high percentage of Type II fibres may present a considerable advantage. This has been demonstrated by McCartney et al. (1983), who found up to a 33% difference in maximum power output between subjects. McCartney et al. found that the highest power output was achieved at a cadence of 162 rev·min^{-1} by a subject with 72% of Type II fibres in the vastus lateralis. In comparison, the lowest peak power output was reached at a cadence of 119 rev·min^{-1} by a subject with 53% of Type II fibres.

3.2 Short-term prior exercise and recovery

Much of the work examining the effect of prior exercise on maximal cycling muscle function has been conducted by Sargeant and colleagues (Sargeant and Dolan, 1987; Beelen and Sargeant, 1991; Beelen and Sargeant, 1993). These studies examined the effect of short-term (< 10 min) prior exercise on maximal isokinetic cycling power output (MICPO). All investigations were in agreement, finding prior exercise of 6 min at approximately 90% $\dot{V}O_{2peak}$ resulted in a significant reduction in MICPO. Sargeant and Dolan (1987) examined the time course for the recovery of peak power output following the prior exercise. They found MICPO was initially restored rapidly with a half-time of approximately 32 s. These data are similar to those of Hitchcock (1989) who examined the recovery time course in isokinetic leg extension power following various intensities of exercise. Both studies noted the recovery time course for peak isokinetic power was similar to that found for phosphocreatine (PCr) resynthesis (half-time = 21 s, Harris et al. 1976).

3.2.1.1 Phosphocreatine and fatigue

The importance of PCr availability for maximum sprint performance is well established, especially during repeated sprint exercise (McCartney et al. 1986; Sargeant and Dolan, 1987; Gaitanos et al. 1993; Bogdanis et al. 1996; Casey et al. 1996a; Trump et al. 1996). The studies of Gaitanos et al. (1993) and Bogdanis et al. (1996) have demonstrated that the extent of PCr resynthesis occurring after sprint exercise dictates the peak power output developed in the subsequent sprint. Furthermore, Casey et al. (1996a) found a 30 s isokinetic sprint at 80 rev·min^{-1} resulted in greater depletion in Type II fibres of ATP and PCr in comparison with Type I fibres. The Type II fibres also restored these phosphogens more slowly, such that in a subsequent sprint ATP and PCr utilisation were significantly reduced in the Type II fibres. Casey et al. hypothesised that this lower phosphogen contribution from the Type II fibres was responsible for the reduced work done in the second sprint. Thus, any fatiguing effect of short-term prior exercise on maximum muscle function seems likely to be mediated by changes in PCr content.

3.2.1.2 pH and fatigue

It is possible that fatigue may arise from exercise induced changes in intracellular pH. Compelling evidence for the role of H^+ ion accumulation in the aetiology of fatigue comes from studies on skinned animal muscle fibres (for reviews see Allen et al. 1995; Fitts, 1994). This proposition is not supported by the results of the previously cited repeated sprint studies (Gaitanos et al. 1993; Bogdanis et al. 1996). During repeated high intensity exercise bouts the relationship between pH and fatigue has been dissociated. In recovery from high intensity exercise, intracellular pH remains depressed for several minutes as H^+ ions are released due to PCr resynthesis. Despite this low pH, the recovery of both maximum isometric force (Miller et al. 1987; Sahlin and Ren, 1989) and peak power output (Gaitanos et al. 1993; Bogdanis et al. 1996) have been demonstrated in humans. Using NMR to monitor changes in pH, Wilson et al. (1988) were also able to demonstrate that the relationship between fatigue and pH could be manipulated during maximal exercise. Thus, the fatiguing effect of pH observed on skinned fibres appears to be directly contradicted by studies in humans. A recent study by Westerblad et al. (1997) may allow the reconciliation of these equivocal findings. Westerblad et al. found the fatiguing effect of low intracellular pH on muscle function to be temperature dependent; a point overlooked by previous researchers. Westerblad et al. opine that the role of low pH in muscle fatigue is minor, when their results are extrapolated to body temperature. Thus, changes in muscle pH are considered unlikely to limit measures of maximal muscle function. It is possible that a pH mediated inhibition of glycogenolysis may limit exercise which relies heavily on this metabolic pathway (Spriet et al. 1989), e.g. time of maximal isometric force maintained to exhaustion (Sahlin and Ren, 1989).

3.2.2 Short-term prior exercise intensity

3.2.2.1 Dynamic measures

Several investigators have examined the effect of different prior exercise intensities on subsequent maximal muscle function with unequivocal results (Hoffman et al. 1985; Ferretti et al. 1987; Sargeant and Dolan, 1987; Hitchcock, 1989; Fulco et al. 1995). All studies found the intensity of prior exercise exerts a significant effect on subsequent maximal muscle function. Sargeant and Dolan (1987) found that prior exercise up to

approximately 60% $\dot{V}O_{2peak}$ tended to enhance MICPO. Beyond 60% $\dot{V}O_{2peak}$ MICPO was found to be progressively diminished. The improvement in muscle function following lower intensity exercise is probably due to an increase in muscle temperature (T_m) (Sargeant, 1987) as no warm up was allowed before the first sprint. Ferretti et al. (1987) measured the effect of prior exercise intensity and duration on instantaneous power output. Instantaneous power output was calculated from subjects jumping on a force platform within a few seconds of the preceding exercise. Congruent with the results of Sargeant and Dolan (1987), peak power output did not change following exercise between 30 and 50% $\dot{V}O_{2peak}$. A small decline in peak power output occurred following exercise between 50 and 70% $\dot{V}O_{2peak}$, and higher intensities resulted in a more rapid decline. In contrast to Sargeant and Dolan, Ferretti et al. suggested that the decline in peak instantaneous power output reflects a reduction in hydrolysis of immediately available ATP. These authors' reasoning is questionable as the short time between exercise and the jump test, in addition to the time taken to develop peak power output during the jump, are sufficient for a significant contribution from PCr to occur.

Hitchcock (1989) also examined dynamic maximal muscle function following prior exercise. Subjects cycled at either 60, 80, 100 or 120% $\dot{V}O_{2peak}$ before measurement of isokinetic single leg extension power. A significant reduction in peak power output was observed for all intensities, with a decrement in proportion to the prior exercise intensity. The pattern of recovery for all prior exercise intensities was similar to that for PCr resynthesis, following a two component exponential curve. Consequently, power output was fully restored following 1 min of recovery after exercise at 60 and 80% $\dot{V}O_{2peak}$, but delayed beyond 4 and 8 min following prior exercise at 100 and 120% $\dot{V}O_{2peak}$ respectively.

3.2.2.2 Static measures

Similar responses have also been found for static muscle function following dynamic exercise (Hoffman et al. 1985; Fulco et al. 1995). Hoffman et al. measured both maximum isometric force and time to exhaustion at 40% MVC in the leg extensors, following up to 20 min of prior exercise at 20, 60 and 80% $\dot{V}O_{2peak}$. No effect of prior exercise on MVC was found following exercise at either 20 or 60% $\dot{V}O_{2peak}$. Exercise at

80%$\dot{V}O_{2peak}$ reduced MVC to 85% after 1 min and it had fallen further to 70% by 20 min of prior exercise. Time to exhaustion at 40% MVC was more markedly affected by both prior exercise intensity and duration (up to 10 min). This is in agreement with the findings of others (e.g. Sahlin and Ren, 1989), and suggests that different factors regulate maximal muscle force and the capacity to maintain a sustained contraction. Using a novel ergometer Fulco et al. (1995) also monitored changes in MVC resulting from dynamic leg exercise in 8 male subjects. These researchers examined MVC after 2 and 4 min of dynamic single leg extensions at 66, 78, 100 and 131% of $\dot{V}O_{2peak}$. The prior exercise tended to reduce MVC with significant reductions found following the 2 higher work rates at both 2 and 4 min. The trial at 100% $\dot{V}O_{2peak}$ resulted in MVC being significantly reduced to 72% and 65% after 2 and 4 minutes of exercise respectively.

3.2.3 Short-term prior exercise and cadence

The cadence adopted both for the prior exercise and the subsequent sprint have been shown to influence the degree of fatigue (Beelen and Sargeant, 1991; Beelen and Sargeant, 1993). Both studies of Beelen and Sargeant found subsequent sprint power was more markedly reduced when sprinting at faster cadences (120 rev·min^{-1}) compared with slow (60 rev·min^{-1}). This was assumed to be due to a greater reliance during the sprint on fatiguable Type II fibres at the faster cadence. Beelen and Sargeant (1993) also found that prior exercise (236 W) performed at 120 but not 60 rev·min^{-1} significantly reduced peak power output. The authors speculate that the results are due to the faster cadence requiring a greater recruitment of more fatiguable Type II fibres. Cherry et al. (1997) dispute this speculation of Beelen and Sargeant, claiming there is little evidence of selective fatigue of Type II fibres at faster cadences. Instead they propose that the number and/or speed of muscle actions may be the critical factor. By manipulating the braking loads for 3 different 30 s sprints, Cherry et al. were able to examine the effects of altering sprint cadence, peak and average power output attained on the subsequent peak power output in a 6 s sprint. The 30 s sprint resulting in the development of the highest peak power (722 vs. 702 W) and greatest work done (14.5 vs. 13.3 kJ) did not induce the most fatigue in the subsequent 6 s sprint peak power output (534 vs. 420 W respectively). Instead the trial conducted at the fastest cadence (96, vs. 69 and 75 rev·min^{-1}) resulted in a significantly lower 6 s sprint peak power output. It is

acknowledged that in the context of this discussion, the maximal intensity of prior exercise may confound the interpretation of these results.

In all the above studies the duration of the prior exercise was relatively short-term, i.e. duration generally less than 10 min. The experiments discussed in the subsequent sections are concerned with the effects of prolonged prior exercise, i.e. duration generally in excess of 30 min.

3.3 Prolonged exercise

3.3.1 Isometric exercise

Prolonged exercise of repeated isometric muscle actions has been demonstrated to result in a significant reduction in maximum voluntary and electrically stimulated isometric actions (Vollestad et al. 1988; Vollestad et al. 1990; Saugen and Vollestad, 1996). Repeated 6 s isometric muscle actions at 30% MVC followed by 4 s rest did not change muscle PCr, ATP, lactate or glycogen when measured after 30 min of exercise (Vollestad et al. 1988). At exhaustion Vollestad et al. found a large decrease in PCr accompanied by only modest changes in ATP, lactate and glycogen. The temporal pattern of fatigue in both MVC and electrically stimulated force was different, declining progressively throughout exercise. The authors concluded that central fatigue, substrate depletion and changes in muscle pH or lactic acid concentration cannot be implicated in the rate of muscle fatigue observed. Instead they speculate that an impairment of excitation-contraction coupling may be involved. The cause for exhaustion could not be determined, although Vollestad et al. suggest it may be due to an energy crisis within the muscle as evidenced by the rapid decline in PCr and drop in ATP. In subsequent studies Vollestad and colleagues have demonstrated that the loss of force generating capacity is mirrored by a progressive increase in $\dot{V}O_2$ (Vollestad et al. 1990). This $\dot{V}O_2$ drift is similar to that seen in endurance exercise and reviewed earlier (section 2.7). A significant factor in the increased rate of energy expenditure appears to be a reduced contraction efficiency (Sejersted and Vollestad, 1992; Saugen and Vollestad, 1996). An increase in muscle fibre recruitment and T_m were not found to contribute significantly (Saugen and Vollestad, 1996). The relevance of these characteristics and mechanisms of fatigue in isometric

exercise to dynamic exercise situations remains unclear, although obvious corollaries exist.

3.3.2 Dynamic exercise

3.3.2.1 Effects of non-specific dynamic exercise on static muscle function

Fulco et al. (1995) report that MVC fell progressively throughout dynamic single leg extension exercise during a trial to exhaustion at 83% $\dot{V}O_{2peak}$. The iEMG of the quadriceps muscles changed in a similar manner during the repeated MVC's. The dynamic exercise was associated with a progressively rising iEMG. At exhaustion iEMG for MVC and dynamic exercise had almost converged. Although comparable to the findings of Vollestad et al. for isometric exercise, Fulco et al. claim their results to be the first data demonstrating a serial decline in MVC after dynamic leg exercise. In a subsequent investigation Fulco et al. (1996) used the same method to compare muscle fatigue and exhaustion in normoxia and hypobaric hypoxia (464 mm Hg). The hypoxic condition resulted in a twofold increase in the rate of fatigue and a significantly shorter time to exhaustion (19 vs. 43 min). A progressive $\dot{V}O_2$ drift was evident for both conditions, but was more marked in hypoxia resulting in a significantly greater $\dot{V}O_2$ at 10 min and exhaustion. The MVC at exhaustion was similar under both conditions and was mirrored by a decline in iEMG activity. Fulco et al. report that at exhaustion MVC remained nearly three times greater than the required peak dynamic force ($\approx$ 274 N vs. 98 N respectively). They suggest that exhaustion was unlikely to be due to an inability to generate sufficient torque with the knee extensors. Instead they postulate that a reduced velocity of muscle shortening was responsible, this is consistent with the findings of others (de Haan et al. 1989; Beelen et al. 1995). In support of this postulation Fulco et al. observed that at exhaustion each subject was able to continue exercise, albeit at a slower rate of movement. Further research is required to ascertain the extent to which these results are applicable to exercise involving a larger active muscle mass.

3.3.2.2 Running

Maximal muscle function has been variously examined following prolonged walking and running exercise (Davies and White, 1982; Sherman et al. 1984; Davies and Thompson, 1986). All three studies found a significant drop in maximal muscle function for both MVC (Davies and White, 1982; Davies and Thompson, 1986) and isokinetic leg extension (Sherman et al. 1984). Sherman et al. tracked the recovery time course of 10 runners after competing in a marathon. A greater fatigue was apparent at low angular velocities compared with high (1.1 vs. 5.3 $rad.s^{-1}$) in contrast to the effects of short-term cycling (Beelen and Sargeant, 1991). Peak torque was significantly reduced immediately after the race and fell further over the following 24 h. Seven days after the race peak torque was still significantly lower than pre-marathon values, irrespective of whether subjects had resumed training or not. The pattern of recovery implicates muscle damage, commonly observed following running (Rogers et al. 1985; Dressendorfer et al. 1991; Fry et al. 1991; Westerland et al. 1992), as a cause of the reduced muscle function measures. This is in contrast to cycling exercise, where severe fatigue may be induced without any evidence of muscle damage (Kuipers and Keizer, 1988).

3.3.2.3 Effects of prolonged cycling on non-specific muscle function

The effects of prolonged cycling on subsequent isometric muscle function are not widely documented (Hoffman et al. 1985; Nielsen et al. 1993; Sahlin and Seger, 1995). As reported earlier, Hoffman et al, found no effect of 20 min cycling at either 20 or 60% $\dot{V}O_{2peak}$ on MVC, whilst at 80% $\dot{V}O_{2peak}$ a significant reduction after 1 min of approximately 15% was doubled to 30% by 20 min. Although Hoffman et al. did not find a significant effect of cycling at 60% $\dot{V}O_{2peak}$ for 20 min, a progressive decline in MVC over time was apparent. It is possible therefore, that further cycling at this intensity may have resulted in significant fatigue. This postulate is not borne out by the data of Nielsen et al. (1993). Nielsen et al. found no change in MVC after 90 min cycling at 60% $\dot{V}O_{2peak}$ in a cool environment or to exhaustion in the heat (40 °C).

Recently, Sahlin and Seger (1995) investigated the effects of cycling to exhaustion at 75% $\dot{V}O_{2peak}$ on maximal isometric and isokinetic (concentric and eccentric at 1.05

rad·s^{-1}) leg extension torque. Maximal isometric force and isokinetic concentric torque were measured in seven moderately trained men at 5 and 40 min of exercise, exhaustion (≈ 85 min) and 5, 15 and 30 min of recovery. All measures of muscle force generating capacity were significantly decreased to approximately 70% at exhaustion and recovered slowly, remaining significantly depressed after 30 min of recovery (except for eccentric force). During exercise MVC fell rapidly initially, to be 9% lower after 5 min whilst concentric torque remained unchanged. Thereafter, both parameters appeared to have declined in a progressive manner until exhaustion. The recovery of MVC appeared to occur in two phases, the first of which was rapid occurring over the first few minutes, but MVC was still reduced by 20% after 30 min of recovery. Electrical stimulation did not change the MVC recorded either pre or post exercise, rejecting an influence of central fatigue. Isokinetic concentric force restoration was reported to vary widely and had not changed significantly from exhaustion after 30 min.

3.3.2.4 Effects of prolonged cycling on peak power output

Like isometric muscle function, the effects of prolonged cycling on peak power output do not appear to have been extensively investigated (Capelli et al. 1993a; Rademaker et al. 1994a; Rademaker et al. 1994b). Capelli et al. (1993a) measured maximum work done in 10 pedal revolutions every 5 or 6 min throughout 20 or 35 min of continuous exercise at 50, 65, 75 and 80% $\dot{V}O_{2peak}$. In agreement with studies on the effects of prior exercise, discussed previously (section 3.2.2), the work done in the first sprint decreased linearly with the intensity of exercise. Thereafter, at 50% $\dot{V}O_{2peak}$ sprinting power remained unchanged, whilst at intensities above 60%, sprinting power declined and $\dot{V}O_2$, and blood lactate concentration increased continuously. Significant correlations between the change in sprinting power and change in $\dot{V}O_2$ ($r = 0.27$) and hetween change in $\dot{V}O_2$ and blood lactate ($r = 0.55$) were found for the pooled data for all subjects at every time point. These observations may suggest a link between $\dot{V}O_2$ drift and muscle function.

Rademaker et al. (1994a, 1994b) examined the effects of 45 min at 80% $\dot{V}O_{2peak}$ on MICPO measured at a cadence of 120 rev·min^{-1}. In the first study (Rademaker et al. 1994a) no significant change in MICPO was found following prior exercise at a cadence of either 60 or 120 rev·min^{-1} or when compared to control (1394, 1418 and 1406 W

respectively). Rademaker et al. speculated that an increase in T_m (3.2 °C) counteracted the exercise induced fatigue. In a subsequent study (Rademaker et al. 1994b), 45 min cycling at 80% $\dot{V}O_{2peak}$ and 120 rev·min^{-1} was again found not to alter significantly MICPO from control values after (1466 vs. 1471 W respectively). The authors report that a parallel experiment found a 5 min warm up significantly increased T_m (2.5 °C) and MICPO (1767 W). Rademaker et al. argued that if this sprint is used as the appropriate control a significant reduction following the prolonged exercise was evident. This was reinforced by the finding that a 6 minute recovery saw an increase in MICPO to 1632 W which was still 8% lower than after the warm up.

It is notable that a significant reduction in maximum muscle function has been found only following exercise at intensities greater than 60% $\dot{V}O_{2peak}$. Although, the problems with the studies of Rademaker et al. (1994a, 1994b) emphasise the potential for changes in T_m to obscure fatigue effects. The higher exercise intensity (75-80% vs. 60% $\dot{V}O_{2peak}$) could explain the significant drop in MVC found by Sahlin and Seger (1995) and Hoffman et al. (1985), but not Nielsen et al. (1993). Changes in cycling power output also appear to be intensity dependent, as Rademaker et al. and Capelli et al. both found a decline in sprinting power with exercise performed at intensities above 60% $\dot{V}O_{2peak}$. It is tempting to speculate that, like short-term prior exercise, changes in PCr may explain the fatigue induced by sustained exercise. During prolonged cycling exercise (≈70% $\dot{V}O_{2peak}$) PCr is gradually depleted (Norman et al. 1987) with PCr found to be significantly reduced from rest at exhaustion (Broberg and Sahlin, 1989). It is not clear whether this occurs at 60% $\dot{V}O_{2peak}$, however, as Ball-Burnett et al. (1991) did not find changes in PCr concentration. The suggested link between PCr and fatigue in prolonged exercise is clearly incompatible with slow recovery found by Sahlin and Seger (1995) and possibly Rademaker et al. (1994b).

In summary, the fundamental properties of skeletal muscle are thought to determine its force-velocity and length-tension relationships. These parameters should be strictly controlled during assessment of maximal muscle function. Both isometric and dynamic muscle function are reduced by short-term prior exercise. The magnitude of the fatigue appears to be related to the exercise intensity of the prior exercise and is thought to be

related to PCr availability. The effects of prolonged moderate intensity exercise on subsequent muscle function have been largely unstudied. Prolonged isometric exercise results in a progressive reduction in MVC, which is inversely mirrored by a $\dot{V}O_2$ drift. A similar effect on MVC has been reported for dynamic exercise involving single leg extensions. Endurance cycling at > 75% $\dot{V}O_{2peak}$ results in a reduction in both static and dynamic indices of quadriceps muscle function and cycling peak power output. The effects of prolonged moderate intensity exercise (60% $\dot{V}O_{2peak}$) are unknown, although MVC is not thought to be reduced.

3.4 Limiting factors in endurance exercise and maximal muscle function

The primary factors thought to limit endurance exercise and performance are reduced CHO availability, dehydration and hyperthermia (Costill and Hargreaves, 1992; Coyle and Montain, 1992; Maughan, 1992; Terrados and Maughan, 1995). The roles that each of these factors may play in reducing maximal muscle function and high intensity exercise following prolonged moderate exercise are reviewed below.

3.4.1 Glycogen depletion[1]

The relationship between muscle glycogen availability and the capacity to perform prolonged exercise has been recognised for a considerable time (Bergström et al. 1967; Hermansen et al. 1967). Many authors are of the opinion that single bouts of high intensity exercise will result in exhaustion considerably before muscle glycogen becomes depleted (Costill, 1988; Abernethy et al. 1990; Sahlin, 1992a; Fitts, 1994; Hawley et al. 1997b). It is surprising then, that research findings are equivocal on whether dietary CHO intake and muscle glycogen availability limits high intensity exercise (see table below). Table 3.1 provides a summary of studies examining the effects of glycogen manipulation and CHO intake on high intensity exercise. The details provided include the duration of duration of the high intensity exercise examined (time), the reported or estimated muscle glycogen concentration in the low CHO condition (concentration), an indication of whether this value is measured or estimated from comparable studies (measured),

[1] All glycogen concentrations have been converted to mmol·kg^{-1} dry mass (x 4.3) where appropriate.

whether prior exercise was controlled for each condition (exercise) and whether a reduction in maximal muscle function or performance was associated with reduced CHO availability (effect).

Table 3.1 Summary of effect of glycogen availability on high intensity exercise.

Authors	Time (s)	Concentration mmol·kg^{-1} dm	Measured	Exercise Control	Effect
Houston et al. (1981)	PPO	145	Y	D	Y
Greenhaff et al. (1988c)	200	300*	N	D	Y
Jenkins et al. (1993)	5 x 60	--	--	D	Y
Langfort et al. (1997)	PPO + 30	--	--	D	N+Y
Asmussen et al. (1974)	> 120	--	--	N	Y
Jacobs (1981a)	PPO	--	--	N	Y
Jacobs (1981b)	PPO	--	--	N	Y
Maughan and Poole (1981)	300	150	N	P	Y
Young and Davies (1984)	MVC	--	--	P	Y
Greenhaff et al. (1987)	300	150†	N	P	Y
Manghan (1988)	50	--	--	N	Y
Bangsbo et al. (1992)	2 x < 175		Y	N	Y+N
" " " "	*1st 175*	*375*	"	"	*N*
" " " "	*2nd 175*	*310*	"	"	*Y*
Casey et al. (1996b)	4 x 30	25	N	P	Y
Wootton and Williams (1984)	2 x 30	--	--	P	N
Symons and Jacobs (1989)	PPO + MVC	153	Y	Y	N
Grisdale et al. (1990)	MVC	126	Y	Y	N
Ren et al. (1990)	--	155	Y	N	N
Spriet et al. (1990)	MVC	82	Y	Y	N
Spencer and Katz (1991)	--	201	Y	P	N
Lambert et al. (1994)	PPO	292	Y	Y	N
Vandenberghe et al. (1995)	175	364	Y	Y	N
Hargreaves et al. (1997)	PPO + 75	364	Y	Y	N

PPO: Peak power output, Y: Yes, N: No, P: Partial and D: Dict only. See text for more details. Where muscle glycogen concentrations are indicated but not measnred, the concentration has been estimated from comparable studies, (see relevant text for further details). * Muscle glycogen concentration estimated from Greenhaff et al. (1988b). † See Maughan and Poole (1981).

3.4.1.1 Extent of glycogen depletion

The ambiguity in the literature on the influence of glycogen availability may be a consequence of the extent of prior depletion. Several authors have proposed that depleting glycogen below a critical concentration will compromise sprint performance (Jacobs, 1981b; Jacobs, 1987; Costill, 1988; Bangsbo et al. 1992). Maughan et al. (1997) point out glycogen availability is unlikely to be limiting in situations where the end

exercise concentration is above the K_m for phosphorylase. Bangsbo et al. (1992) observe the *in vitro* K_m for phosphorylase is reported to be very low (2 mmol) and suggest that the enzyme is fully saturated at muscle glycogen concentrations above 170 mmol·kg^{-1} dry mass. This suggestion is in agreement with Jacobs (1981b, 1987) who found muscle lactate concentrations are reduced once muscle glycogen falls below 170 to 220 mmol·kg^{-1} dry mass. Contradictory evidence is provided by several studies that have not found low muscle glycogen ($\leq$ 200 mmol·kg^{-1} dry mass) to alter glycogenolytic rate during dynamic exercise or electrical stimulation (Ren et al. 1990; Spriet et al. 1990; Spencer and Katz, 1991). Ren et al. (1990) reported that electrically stimulated muscle glycogenolysis in humans was not affected by a drop in muscle glycogen concentration to 155 mmol·kg^{-1} dry mass. Similarly, Spencer and Katz, (1991) found glycolysis during cycle exercise at 95% $\dot{V}O_{2peak}$ was not affected by reducing muscle glycogen from 583 to 201 mmol·kg^{-1} dry mass. It was speculated that glycolytic flux was maintained in the low CHO condition by an increase in free ADP and AMP and consequent allosteric regulation of PFK. Although changes in ADP and AMP were not observed, they were inferred from the observation of a greater accumulation of IMP in the low CHO condition. Thus, the muscle glycogen concentration at which glycolysis is compromised remains unclear, but appears likely to be below 200 mmol·kg^{-1} dry mass. It is notable that reduced CHO availability appears to be critical for longer or repeated bouts of high intensity exercise lasting several minutes (Maughan and Poole, 1981; Jenkins et al. 1993; Casey et al. 1996b) in contrast to single shorter efforts of less than 80 s (Symons and Jacobs, 1989; Vandenberghe et al. 1995; Hargreaves et al. 1997).

In Table 3.1 an effect of reduced CHO availability and the duration of high intensity exercise is not immediately evident. This may be due to confounding methodological influences of prior exercise and dietary composition which are discussed subsequently. Interpretation of glycogen data from mixed muscle is also problematic, as Jacobs et al. (1981) have found the nature of the prior exercise results in fibre type specific glycogen depletion. Moreover, the different prior exercise protocols also result in selective effects on subsequent muscular strength measures. Greenhaff et al. (1994) have also demonstrated a more extensive Type II fibre glycogen depletion after sprint running. Furthermore, Fridén et al. (1989) report intracellular muscle glycogen depots to be clearly

differentiated, with a marked depletion between the I-bands and from sub-sarcolemmal spaces after repeated sprint cycling. These studies provide evidence that the nature of the exercise to deplete muscle glycogen will influence not only its extent, but also the fibres and sub-cellular compartments from which glycogen is drawn. Consequently, a critical selective glycogen depletion might not be evident from mixed muscle biopsy data or conversely, extensive glycogen depletion may occur without depletion of crucial intracellular glycogen depots.

3.4.1.2 Acid-base balance

From the table above, dietary manipulation alone is seen to reduce high intensity exercise capacity (Houston et al. 1981; Greenhaff et al. 1988c; Jenkins et al. 1993; Langfort et al. 1997). Greenhaff et al. have published a series of studies on the metabolic consequences of altering dietary composition (Greenhaff et al. 1987, 1988a, 1988b, 1988c). High CHO diets are associated with a greater consumption of alkaline foodstuffs, whilst high fat and protein diets increase dietary acid intake. Consequently, Greenhaff et al. found dietary manipulations typical of those employed during glycogen depletion regimens, to alter significantly acid-base balance. The low CHO diets are also associated with a diminished capacity for high intensity exercise, which Greenhaff et al, speculated may be due to the diet induced changes in acid-base balance. The results of Jenkins et al. (1993) and Langfort et al. (1997) provide evidence that changes in dietary composition can also influence both short (30 s) and repeated sprint exercise. Consistent with the earlier suggestion that pH does not influence maximal muscle function (section 3.2.1.2), the study of Langfort et al. did not find a low CHO diet to reduce peak power output. It should also be noted that the results of Houston et al. are due to the effects of energy and fluid intake restriction, not a variation in dietary composition.

3.4.1.3 Prior exercise

An effect of glycogen depleting exercise on subsequent high intensity performance has been previously demonstrated (Young and Davies, 1984; Grisdale et al. 1990). Young and Davies found the fatigue induced by prior exercise was augmented under conditions of low muscle glycogen. Grisdale et al. found a pronounced fatiguing effect of prior exercise, but not of reducing muscle glycogen to 126 $mmol \cdot kg^{-1}$ dry mass on time to

exhaustion with isometric exercise. These findings may explain the decline in performance found in those studies that did not strictly control for an order effect of prior exercise (Jacobs, 1981a, 1981b; Maughan, 1988). In Table 3.1 the degree of experimental control appears to be closely associated with whether a significant reduction in high intensity exercise was observed. It is striking that no effect was observed in those studies employing strict experimental control (crossover trials and random allocation) of exercise and dietary regimens. In contrast, a lack of, or partial experimental control with respect to diet and prior exercise, (by adopting a sequential administration of conditions, e.g. normal diet, depleting exercise, low CHO diet, high CHO diet), characterises those studies which find high intensity exercise to be compromised by low CHO intake. Unfortunately, none of the studies adopting partial (sequential) control have provided biopsy data to enable further insight into the reason for this apparent effect.

3.4.1.4 Severe glycogen depletion, sustained and repeated high intensity exercise

Experimental evidence of the effects of extensive glycogen depletion on high intensity exercise is provided by Maughan and Poole (1981) and Casey et al. (1996b). Maughan and Poole (1981) found time to exhaustion at 104% $\dot{V}O_{2peak}$ was significantly affected by a combination of exercise and diet. Subjects were tested on days 1, 4 and 7 following normal, low CHO and high CHO diets respectively. Time to exhaustion was significantly reduced following severe exercise to lower muscle glycogen and 2 days on a low CHO diet (4.87 vs. 3.32 min). The subsequent high CHO diet significantly extended time to exhaustion beyond that of the other two dietary conditions (6.65 min). Casey et al. (1996b) found repeated high intensity performance to be compromised under conditions of low CHO intake. They examined MICPO in 4 repeated bouts of 30 s sprints in 11 subjects who were randomly allocated to either high or low CHO diets. After performing the control sprints, subjects completed an exercise regimen which had previously been demonstrated to result in severe muscle glycogen depletion under low CHO conditions (25 $mmol \cdot kg^{-1}$ dry muscle). A significant difference in total work done for each of the first three sprints was found with low CHO group but not the high CHO group. The final sprint was not different for either low or high CHO. Unfortunately, a pre-existing difference between high and low CHO groups in mean sprint power was evident; it is not known if or how this may have influenced these findings. The significant reduction in the

3rd sprint of the low CHO condition is somewhat unexpected. An earlier study by McCartney et al. (1986) who used the same repeated sprint protocol found muscle glycogen did not change beyond the 2nd sprint. This may indicate that factors other than glycogen manipulation influenced the work done during the 3rd sprint e.g. dietary induced metabolic acidosis. Neither the study of Maughan and Poole, nor Casey et al. measured muscle glycogen concentrations. Both studies however, are likely to have achieved extensive glycogen depletion in the low CHO intake condition and also during the high intensity exercise.

3.4.1.5 Moderate glycogen depletion and short bouts of high intensity exercise

Moderate reductions in muscle glycogen availability do not appear to reduce peak power output or performance. The recent study of Hargreaves et al. (1997) examined the influence of moderate glycogen depletion on performance in a 75 s maximal cycling test in 9 experienced racing cyclists. Glycogen depletion was induced by riding for 1 h at 70% $\dot{V}O_{2peak}$ followed by 6 x 30 s sprints. Following this exercise subjects were assigned to a high or low CHO diet for 24 h in a random counter-balanced design. Muscle glycogen was significantly altered by the combination of exercise and diet to 578 mmol·kg^{-1} dry mass and 364 mmol·kg^{-1} dry mass after high and low CHO intake respectively. Despite the difference in glycogen content, peak power (1185 vs. 1179 W), and mean power (547 vs. 554 W) were similar for the high and low glycogen conditions respectively, as were maximal accumulated O_2 deficit, and concentrations of muscle and blood lactate. Hargreaves et al. concluded that increased muscle glycogen availability does not influence performance or anaerobic energy production. Coincidentally, the study of Vandenberghe et al. (1995) induced almost identical muscle glycogen concentrations following low and high CHO intake. These authors did not detect an influence of CHO availability on cycle time to exhaustion at 125% $\dot{V}O_{2peak}$ (174 vs. 176 s).

3.4.1.6 Moderate glycogen depletion and repeated high intensity exercise

The effect of moderate glycogen depletion on repeated bouts of high intensity exercise is more difficult to ascertain. Bangsbo et al. (1992) examined time to exhaustion during one-legged knee extension exercise under normal and elevated muscle glycogen levels. Bangsbo et al. report no difference between normal and high leg glycogen content in time

to exhaustion (2.82 vs. 2.92 min) during high intensity exercise. This finding is in agreement with those studies that find no effect of moderate glycogen depletion on a single bout of high intensity exercise. Following 1 h of recovery the subjects of Bangsbo et al. performed a subsequent exercise bout where a significant reduction in glycogenolysis and time to exhaustion for the normal glycogen leg were observed. Neither glycogenolysis nor time to exhaustion was altered in the high glycogen leg for the successive bout. No correlation between muscle glycogen concentration and its utilisation during high intensity exercise was found. Bangsbo et al. concluded that factors other than muscle glycogen concentration reduced high intensity performance in the second exercise bout.

Post exercise muscle glycogen concentration in the depleted leg averaged approximately 220 $mmol \cdot kg^{-1}$ dry mass. Thus, it is possible that at the end of the second exercise bout in the depleted leg, glycogen concentrations dropped below the value critical for the maintenance of glycolytic flux. Further support for this suggestion is provided by the study of Wootton and Williams (1984). These authors did not find an effect of low CHO intake on 2 repeated 30 s sprints, separated by 15 min. The apparent discrepancy of the studies of Bangsbo et al. and Wootton and Williams could be due to the extent of glycogen utilisation during the high intensity exercise. This conjecture cannot be verified as Wootton and Williams did not report any muscle biopsy data.

3.4.1.7 Glycogen depletion following prolonged moderate intensity exercise

Finally, it is pertinent to consider whether the extent of glycogen depletion occurring during prolonged moderate intensity exercise is likely to result in limiting levels of muscle glycogen for subsequent high intensity exercise. Muscle glycogen concentrations following endurance cycling at 60% $\dot{V}O_{2peak}$, have been reported to be 172 and 86 $mmol \cdot kg^{-1}$ dry mass after 1 and 2 h respectively (Gollnick et al. 1974). Ball-Burnett et al. (1991) examined glycogen depletion in separate fibre types with single leg cycling at 60% $\dot{V}O_{2peak}$. These researchers report glycogen concentrations of 110 and 210 $mmol \cdot kg^{-1}$ dry mass after 1 h and 82 and 175 $mmol \cdot kg^{-1}$ dry mass after 2 h or at exhaustion, in Type I and II fibres respectively. Neither of these studies recruited well-trained subjects who are likely to exhibit reduced glycogenolysis during exercise of this nature. Studies by Coyle

and colleagues who examined responses at 70% $\dot{V}O_{2peak}$ in trained cyclists confirm this suggestion, with higher levels of muscle glycogen after 2 h of 260-300 mmol·kg^{-1} dry mass being reported (Coyle et al. 1986; Coyle et al. 1991b). Recently, González-Alonso et al. (1997) reported that trained cyclists exercising in the heat at 60% $\dot{V}O_{2peak}$, were still found to have muscle glycogen concentrations of 306 mm.kg^{-1} dry mass at exhaustion (140 min).

In conclusion, studies of the effects of glycogen availability on maximal muscle function and high intensity performance are equivocal in their findings. It has been proposed that muscle glycogen availability does not limit high intensity exercise until the reduction reaches a critical value. The muscle glycogen concentration at this critical value is currently unclear, but is likely to be below 200 mmol·kg^{-1} dry mass and influenced by the amount of glycogen required for the high intensity exercise. This suggestion is supported by the observations that severe muscle glycogen depletion is associated with a diminished performance of high intensity exercise whilst moderate depletion has no effect. Various methodological factors may also have contributed to the seemingly variable influence of muscle glycogen availability. In particular, the selective glycogen depletion and subsequent fatiguing effects of glycogen depleting exercise represent a potentially confounding variable. Also, the dietary manipulation associated with glycogen depletion regimens has been found to induce changes in acid-base status. It seems likely that glycogen concentrations following prolonged moderate intensity exercise ($\leq$ 2 h) in trained subjects would be sufficient to maintain maximal sprint performance. In those studies where an effect of prior exercise is implicated independently of muscle glycogen, additional factors must be responsible.

3.4.2 Muscle temperature

Changes in T_m are known to modify dynamic (Binkhorst et al. 1977; Bergh and Ekblom, 1979; Davies and Young, 1983; Sargeant, 1987; Rademaker et al. 1994b; Sargeant and Rademaker, 1996), but not necessarily static maximal muscle function (Binkhorst et al. 1977; Bergh and Ekblom, 1979; Davies and Young, 1983). A reduction in isometric endurance with increased T_m has been reported (Edwards et al. 1972; Segal et al. 1986), and as mentioned previously (section 3.2.2), may implicate different factors in regulating

maximal muscle force and the capacity to maintain a sustained contraction. Davies and Young (1983) compared the effects of heating and cooling the leg with temperature at rest on maximum muscle function. These researchers found a 3 °C increase in leg temperature to 39.5 °C had minimal effect on either MVC or maximum cycling power output. Cooling to 28 °C, however, significantly reduced MVC and markedly decreased cycling power output. Increasing leg temperature significantly reduced the time to peak tension and half-relaxation time. Davies and Young also suggested that time to peak tension was important in determining maximum power output.

Binkhorst et al. measured the effects of temperature in the range of 22-38 °C on the handgrip muscles of 10 subjects. Maximum power and calculated maximum velocity as well as the shape of the force-velocity curve were all found to change with increasing temperature. No effect of changing muscle temperature was found on maximum isometric force. Bergh and Ekblom (1979) drew similar conclusions to Binkhorst et al. (1977) about the effects of temperature on dynamic and static muscle function. Bergh and Ekblom (1979) found that dynamic measures of maximum muscle function were positively related to T_m over the range of 30 to 40 °C. A movement velocity specific effect of temperature was noted, with leg extension peak torque increasing by 2.1%·°C^{-1} at 0 rad.s^{-1} and 4.9%·°C^{-1} at 3.14 rad.s^{-1}. Bergh and Ekblom concluded that changes in T_m muscle temperature are of minimal importance for the generation of isometric force, but are of much greater significance during dynamic exercise.

The findings of Bergh and Ekblom and Binkhorst et al. were also supported by Sargeant (1987), who examined the influence of muscle temperature on MICPO. Sargeant compared MICPO following 30 min of rest at room temperature (control) with measures after 45 min of leg immersion in a water bath at temperatures of 44, 18 and 12 °C. The MICPO measured at 95 rev·min^{-1} following leg warming increased approximately 11% over the control value. Leg cooling significantly reduced peak power output by 12 and 21% after immersion at 18 and 12 °C respectively. Two of the four subjects also sprinted at three different cadences (54, 95, and 140 rev·min^{-1}) to examine the effect of temperature on muscle power-velocity characteristics. Raising leg temperature increased peak power output more markedly at the fast rather than the slow cadence. Representing

these data as power-velocity curves, raising leg temperature could be seen to increase MICPO, V_{opt} and also (by extrapolation) maximum cadence.

Sargeant and Rademaker (1996) investigated the interaction between T_m, cadence and muscle fibre type. The effects on MICPO of changing T_m and cadence were calculated for eight subjects with reference to their vastus lateralis composition of Type I muscle fibres. The Q_{10} was calculated for the increase in power output at each cadence and in relation to the percentage of Type I fibres. A small effect of temperature was found at the slow cadence and for a low Type I fibre proportion. The temperature mediated effect increased linearly with pedal rate and with greater Type I fibre proportions. Accordingly, Sargeant and Rademaker were able to infer a Type I fibre specific effect of T_m within the range of cycling cadences studied (60, 110 and 140 rev·min^{-1}). This acts to modify acutely these Type I fibres, transforming their power-velocity characteristics towards those of Type II.

In conclusion, raising muscle temperature appears to alter the power-velocity curve of the active muscle mass. This acts to increase the peak power output, V_{opt} and possibly the maximum velocity of contraction. Across the range of generally adopted cadences, this shift in the power-velocity curve is likely to result in an increase in power production only of Type I fibres. Despite this, relatively small changes in muscle temperature can result in marked alterations in peak power output particularly at faster cadences. During static exercise, however, increasing muscle temperature would not be predicted to increase MVC markedly although cooling may reduce force production.

3.4.3 Dehydration

The deleterious effects of dehydration on endurance performance have been well established (for reviews see Coyle and Hamilton 1990; Coyle and Montain, 1992; Sawka, 1992; Terrados and Maughan, 1995). In contrast, it is likely that dehydration of up to 5 to 7% body mass does not alter either dynamic or static maximal muscle function (see Table 3.2). Singer and Weiss (1968) also review several additional unpublished studies that show no effect of dehydration on maximal muscular function. Indeed, Coyle and Hamilton (1990) suggest there is little rationale for an effect of dehydration on maximal muscle function. Furthermore, these authors point out that those studies which have

found an effect of dehydration tend to have adopted a chronic dehydration and energy restriction regimen to achieve the body mass loss (e.g. Bosco et al. 1968; Bosco et al. 1974; Houston et al. 1981; Webster et al. 1988). This suggests that factors other than dehydration may have been significant in the reduced muscle function. In addition, the appropriateness of the statistical analysis employed by Bosco et al. (1968, 1974) has been questioned (Serfass et al. 1984), whilst the study of Houston et al. found a significant reduction in peak torque only at the slowest angular velocity (0.5 $rad \cdot s^{-1}$) tested.

Table 3.2 Summary of effects of dehydration on maximal muscle function.

Authors	Procedure	Δ Mass	Measure	Effect
Tuttle (1943)	C, CD, E	-4.9%	IM	N
Ahlman and Karvonen (1961)	H, E	-2.2 kg	IM	N
Saltin (1964a)	H, E	-4.0%	IM	N
Greenleaf et al. (1966)	C, CD	-6.9%	IM	N
Bosco et al. (1968)	C, CD	-3.1%	IM	Y?
Singer and Weiss (1968)	C, CD, E?	-7.1%	IM	N
Bosco et al. (1974)	C, CD	-5.8%	IM	Y?
Torranin et al. (1979)	H	-4.0%	IM(E)	Y
Bijlani and Sharma (1980)	H, E	-3.0%	IM	N
Jacobs (1980)	H	-5.0%	PPO	N
Houston et al. (1981)	C, CD	-8.0%	IK	Y?
Mnatzakanian and Vaccaro (1982)	?	-4.0%	IK(E)	N
Serfass et al. (1984)	CD?	-5.0%	IM	N
Webster et al. (1988)	CD, E?	-4.9%	IK + PPO	Y
Walsh et al. (1994)	H, E	-1.8%	PPO	N
Greiwe et al. (1998)	H	-3.8%	IM	N

Procedure - dehydration induced by C: Caloric restriction, CD: Chronic dehydration, H: Heat E: Exercise, ? unclear. Measure - IK: Isokinetic torque, IM: Isometric force, PPO: Peak power output, (E): Endurance.

The study of Torranin (1979) is striking in finding a significant reduction with acute dehydration only. These investigators found time to exhaustion at 75% MVC and 75% of 1 repetition maximum (1RM) to be significantly reduced following an acute heat induced dehydration of 4% body mass. Following dehydration, the differential responses of time to exhaustion and maximum isometric force have been noted by Bosco et al. (1974) and have been discussed previously in other contexts (section 3.2.2 and 3.4.2). Serfass et al. (1984) were unsure of the significance of the findings of Torranin et al. commenting on the inconsistent response of different muscle groups to dehydration. It is notable that the

time to exhaustion protocol has been criticised by several authors for its unacceptable variability (Krebs and Powers, 1989; Hickey et al. 1992; McLellan et al. 1995; Jeukendrup et al. 1996).

The results of Serfass et al. (1984) seem unequivocal in contrast to those of Torranin et al. They found no effect of a 5% body mass loss over a 3 day period, nor of attempted rapid rehydration, on MVC or the rate of fatigue development. Dynamic maximal exercise (30 s Wingate sprint) has also been reported to be unaffected by (thermally induced) dehydration of 2, 4 and 5% body mass (Jacobs, 1980). In fact, when average power to body mass is compared across conditions, an increase approaching statistical significance (P=0.06) is observed for the 5% dehydration condition. Walsh et al. (1994) compared performance in a series of 5 s sprints after cyclists had ridden for 1 h at 70% $\dot{V}O_{2peak}$, in 32 °C with and without fluid intake. Dehydration of 1.8% resulted in a significant decrease in time to exhaustion at 90% $\dot{V}O_{2peak}$, but not in 5 s sprint performance.

In conclusion, dehydration does not appear to be associated with significant reductions in either static or dynamic maximal muscle function. Those studies that have reported compromised muscle function following dehydration have been suggested to be methodologically weak.

3.5 Summary

Both dynamic and static muscle function have been found to be reduced by short-term high intensity prior exercise. It is likely that this reduction in maximal muscle function is caused by a reduction of intra-muscular PCr. The effects of prolonged exercise are less clear, with exercise $\geq$ 70 $\dot{V}O_{2peak}$ being found to result in significant reductions in isometric and dynamic leg extension torque, as well as MICPO. The effects of prolonged exercise at intensities below 70% $\dot{V}O_{2peak}$ appear to have been largely ignored by researchers. Glycogen depletion, dehydration and hyperthermia are generally held to be primary limiting factors during endurance exercise. Although the evidence is sometimes contradictory, these factors are not thought to limit maximal muscle function. Indeed,

increases in muscle temperature have been demonstrated to result in a marked increase in MICPO.

3.6 Thesis aims

The aim of this thesis was to examine changes in performance, GE and maximal muscle function following prolonged, moderate but non-exhaustive cycling exercise in well-trained cyclists. Specifically, the following experimental hypotheses were addressed.

3.6.1 Hypotheses

1. Prolonged moderate intensity exercise results in a decrease in GE, and performance in well-trained cyclists and that these changes are related.
2. Prolonged moderate intensity exercise results in a decrease in maximal muscle function in well-trained cyclists and this is related to the change in GE.
3. Exercise induced changes in GE and maximal muscle function during prolonged submaximal exercise are influenced by hydration and carbohydrate status.

Chapter 4: Methods

4.1 Cycle ergometry

All exercise testing was conducted using a modified Monark 814e cycle ergometer unless otherwise stated. The ergometer had been fitted with a racing saddle which was adjusted to the same height as the subject's own training cycle. The ergometer was further modified to accept a power measuring crankset (PMC – SRM, Julich, Germany) and is described in detail in the following chapter. Power output was measured and recorded with the PMC.

4.2 Indirect calorimetry

All indirect calorimetry was conducted with the same gas analysis system. During gas collection subjects breathed through a rubber mouthpiece (Collins, MA. USA.) fitted to a low resistance T-shaped breathing valve assembly (University of Brighton, UK.). A similar arrangement has been demonstrated to have a resistance to flow of < 3 cm H_2O (inspiration) and < 1 cm H_2O (expiration - Jakeman and Davies, 1979). The valve-box was connected to a 200 L Douglas bag by 1.5 m of 3.75 cm bore Falconia tubing. All gas collections were for a whole number of respiratory cycles, starting and finishing during inspiration. Whenever possible collections were of sufficient length to result in an expired gas volume > 100 L. Any expired gas volumes of < 30 L were discarded.

Gas fractions of O_2% and CO_2% were determined directly from the Douglas bag with an 1100 series paramagnetic oxygen analyser and a 1490 series infra-red carbon dioxide analyser (Servomex, Crowborough, UK.) respectively. Each Douglas bag was sampled at a flow rate of 0.5 $L \cdot min^{-1}$ for 90 s to flush out the dead space between the Douglas bag and the analysers and to allow the gas analysers to stabilise. Water vapour was removed prior to analysis as recommended by Beaver (1973) for partial pressure gas analysis systems. The gases were dried by passage through a condenser (Bühler PKE 3, Ratingen, Germany) maintained at 4 °C.

The expirate volume was measured by evacuating the Douglas bag at ≈ 60 $L \cdot min^{-1}$ through a dry gas volume meter (Harvard Apparatus Ltd, Edenbridge, UK.) with a

vacuum pump. The Douglas bag was gently squeezed to help expel the residual volume. All volumes were corrected for the aliquot removed for gas analysis. Gas temperature was monitored by a thermistor at the inlet of the dry gas volume meter. Barometric pressure was recorded from a mercury column barometer (FD and Co. Ltd., Watford, UK.). Oxygen consumption ($\dot{V}O_2$ $l.min^{-1}$ STPD) and respiratory exchange ratio, (RER) were calculated by standard Haldane transformation assuming minimal nitrogen retention or production (Wilmore & Costill, 1973).

Immediately prior to use and periodically during extended use, the gas analysers were calibrated with BOC certified gases. A zero was established for both analysers with pure nitrogen gas. The O_2 analyser's span was calibrated with external ambient air to 20.93%. The CO_2 analyser was calibrated with a BOC gas (14.93% O_2 and 5.82% CO_2) which also provided a linearity check for the O_2 concentration. If O_2 gas concentration varied from the expected value by $> \pm 0.02\%$ the calibration procedure was repeated.

Energy expenditure, CHO and fat oxidation rates were estimated from steady-state RER and $\dot{V}O_2$ using the formulae of Lusk (1924). Gross efficiency was calculated as the percent ratio of power output to power input (Frederick, 1992). Power output was recorded using the PMC and power input was assumed to be equivalent to the rate of energy expenditure. A 15 s delay was used in the measurement of power input with respect to power output to reflect the leg-lung transit time (Barstow & Molé, 1991).

4.3 Heart rate

Heart rate was measured using a short range radio telemetry system (Polar Sports Tester, Kemple, Finland) and recorded along with data from the PMC on the PMC's data logger.

4.4 Blood sampling

The thumb-prick capillary blood sample method was used for all blood collection. The puncture site was swabbed and dried before puncture (Autoclix, (Boehringer Mannheim GmbH, Neuss, Germany).

4.4.1 Blood lactate and blood glucose

Whole blood lactate and glucose concentrations were determined from samples collected in 300µL microvettes (Microvette CB 300, Starstedt, Nümbrecht, Germany) containing 1 mg fluoride and 15 I.U. heparin per ml blood. These samples were analysed with a bench-top semi-automated analyser (YSI 2300, Yellow Springs Instruments, Yellow Springs, OH.) which requires 25 µL aliquots. Blood samples were analysed within 10 minutes of collection. The analyser was set to auto-calibrate every 60 minutes and the initial calibration was verified prior to use with a 10 mmol·L glucose and 5 mmol·L lactate standard (Yellow Springs Instruments, Yellow Springs, OH.). The typical laboratory CV for this analysis is $\leq 2.9\%$.

4.4.2 Haematocrit

A 25 µL blood sample was collected for the determination of haematocrit in a heparinised glass capillary tube (Hawksley and Sons Ltd., Lancing, UK.), immediately centrifuged (Jouan A13, Hawksley and Sons Ltd.,) and measured to the nearest 0.5% with a micro-haematocrit reader (Hawksley and Sons Ltd.).

4.4.3 Haemoglobin

Haemoglobin was determined by the cyanmethaemoglobin method, (Boehringer Mannheim GmbH, Neuss, Germany). A 20 µL capillary blood sample was immediately mixed with Drabkins reagent and stored away from light. Within 24 h the absorbance (A) of the blood sample at a wavelength of 546 nm was determined with a dual beam spectrophotometer (Shimadzu UV 160). The haemoglobin concentration was determined according to the formula below.

Equation 4.1

$$\text{Haemoglobin } (g \cdot dL^{-1}) = 36.77 \times A$$

4.5 Rectal temperature

Rectal temperature was monitored with a flexible disposable thermistor (YSI 4491E, Yellow Springs Instruments, Yellow Springs, OH.) placed 10 cm beyond the anal sphincter.

4.6 Statistical analysis

All statistical analysis was conducted using SPSS for Windows (SPSS Inc., ver. 6). Power analysis was conducted using nQuery (ver. 2).

4.7 Maximal isokinetic cycling power output

An isokinetic cycling dynamometer was constructed by mounting a modified racing cycle equipped with a PMC on a treadmill (Woodway, GMBH, Weil-am-Rhein, Germany). Subjects were able to freewheel whilst the treadmill belt speed was set to a velocity predetermined to elicit the desired cadence. Once instructed to start sprinting, subjects were unable to increase their cadence beyond that dictated by the belt speed of the treadmill. The PMC was used to measure torque and interfaced directly to an IBM compatible computer to enable rapid data sampling. The PMC signal, proportional to torque, was recorded continuously through the parallel port of the computer at 10 ms intervals throughout each sprint. The PMC cranks were calibrated prior to each testing session according to the manufacturer's instructions.

4.8 Pre-testing protocols

Prior to each experiment subjects reported to the laboratory for the determination of submaximal and maximal exercise responses (see below). The main purpose of this testing was to assess each subject's peak aerobic power and determine the appropriate power output for the endurance trials.

On reporting to the laboratory the normal cycling position of the subject was replicated as closely as possible on the Monark ergometer. This position was maintained throughout all subsequent testing.

4.8.1 Submaximal testing

A continuous testing protocol lasting approximately 25 min was used to determine the power output - $\dot{V}O_2$ relationship for each subject. Five stages estimated to elicit 40, 50, 60, 70 and 80% of $\dot{V}O_{2peak}$ were undertaken for 5 min in a manner similar to Coyle et al. (1983). Expired gas and blood lactate measures were taken in the last minute of each

stage. The regression of power output - $\dot{V}O_2$ was calculated and used to determine the power output for each of the endurance trials.

4.8.2 Maximum aerobic power

The peak rate of pulmonary oxygen consumption ($\dot{V}O_{2peak}$) and highest associated 60 s average power output (peak aerobic power output - PAPO) were determined according to the protocol of Keen et al. (1991). Subjects started at a power output of 150-250 W and power output was increased by 20 $W \cdot min^{-1}$ until exhaustion. Expired gas was collected serially throughout over 45-60 s periods. Peak oxygen consumption was defined as the highest measured $\dot{V}O_2$. In agreement with Noakes (1988, 1997), and in contrast to this author's previous experience (Keen et al. 1991), a plateau in oxygen uptake was not evident in most subjects. This was not found despite the attainment of additional criteria commonly associated with maximal exercise i.e. RER > 1.1, and MHR with 10% of age related maximum. Accordingly, the term $\dot{V}O_{2peak}$ is preferred to $\dot{V}O_{2max}$ as a descriptor of maximum aerobic power. This position is supported by an extensive range of evidence that O_2 conductances at each step in the transport chain integrate to define maximal aerobic power (for a recent review see Wagner (1996). Many factors have been demonstrated to modify these O_2 conductances at several steps in the O_2 transport chain. Accordingly, the highest oxygen consumption observed in any exercise test, reflects the integrated response to the various O_2 limited conductances presented by the specific testing conditions.

4.9 Familiarisation

Subjects were fully familiarised with all testing protocols prior to any testing. If the subject had no previous laboratory experience an endurance trial and a test of maximal aerobic power were conducted to promote familiarity with laboratory procedure and measures. Other familiarisation procedures are detailed in the experimental chapters as appropriate.

4.10 Subject control

The importance of strict compliance with pre-experimental procedures was stressed to subjects. Prior to each laboratory visit subjects were requested to ensure that they were

well rested, fully hydrated and had consumed a meal high in CHO at least 2 h beforehand. In particular, subjects were asked to avoid training the day before any laboratory visit and to prepare for each test as if it were a race. No formal dietary control was exercised.

4.11 Subject consent

Prior to all testing subjects gave informed consent to take part in the study and were screened for factors likely to increase the risk of exhaustive exercise. All the procedures employed in this thesis have been approved by the University of Brighton ethical committee.

4.12 Experimental design

The frequent adoption of time to exhaustion as a performance measure, particularly for endurance exercise, has been challenged by several authors (Krebs and Powers, 1989; Hickey et al. 1992; McLellan et al. 1995; Passfield and Hale, 1995; Jeukendrup et al. 1996). McLellan et al. demonstrated that the coefficient of variation (CV) of time to exhaustion varied widely from 2.8 to 31.4%. The authors calculated that to achieve an experimental power of 80% at least 40 subjects should be tested. These findings were reinforced and extended by Jeukendrup et al. who found an average CV of 26.6% for time to exhaustion. In contrast, performance based measures were found to have CV's of around 3.5%. Day to day variability in aerobic power has been found to range from 3.0 to 6.8% over the course of 1 year (Kuipers et al. 1985). Eliminating this variability could reduce the CV's found by Jeukendrup et al. even further. Accordingly, novel experimental designs were adopted in this thesis that sought to maximise experimental power and increase the repeatability of performance measures.

Studies of fatigue during prolonged dynamic exercise have tended to focus upon the point of exhaustion. As pointed out by Vollestad (1997) this approach implicitly ignores the events preceding exhaustion, which may provide additional insight into the aetiology of fatigue during endurance exercise. This thesis examined the effects of non-exhaustive, prolonged, moderate intensity exercise in order to gain further insight into the mechanisms of fatigue associated with exercise of this nature.

Chapter 5: Calibration of power measuring cranks

5.1 Introduction

The recent development of an accurate power measuring crankset (PMC - SRM, Julich, Germany) which can be fitted directly to a bicycle provides a useful sport specific cycle ergometry system in both laboratory and field situations. The manufacturer claims an accuracy for these PMC of ± 2.5%. The facility to measure power output directly from the crank offers distinct advantages to the researcher employing cycle ergometry. The use of PMC enables direct power measurement in field cycling situations, (e.g. Figures 1.1 and 1.2). Modifying a conventional cycle ergometer by fitting a PMC allows the accurate measurement of power output in laboratory situations. The measurement of power output by PMC, rather than calculating directly from the ergometer is advantageous in test situations where marked changes in flywheel angular velocity occur. Lakomy (1986) has found that conventional methods for calculating power output underestimated peak power output by 36% by failing to account for the energy required for flywheel acceleration. Due to its site of power measurement, the use of PMC circumvents this problem. Furthermore, (Sargeant and Davies, 1977) report that cadence typically varies by ± 10% during steady state constant load cycling. The use of PMC may, therefore, improve the accuracy of work rate measurement even during constant load cycling situations.

The veracity of the manufacturer's claims for the accuracy of the PMC has not been independently established. The aim of this study therefore, was to examine the validity of the PMC in the laboratory setting through comparison with a standard laboratory cycle ergometer.

5.2 Method

5.2.1 Power measuring crankset

The PMC may be fitted to a cycle ergometer in place of its normal crankset. The torque generated at the crank axle is measured by 4 strain gauges ("professional" model)

situated between the crank arms and the chain-rings. The strain gauges are oriented in such a manner that their deformation is proportional to the effective pedalling torque (i.e. the resultant force acting tangentially on the crank). Cadence and torque signals are inductively transmitted at 500 kHz from the crank to a sensor connected to the data recorder which stores averaged data at user defined intervals from 0.05 s to 120 s. Power is calculated from torque and angular velocity. The relationship between the frequency output of the strain gauges and torque is determined during manufacture and considered constant. A zero value is established dynamically with the PMC unloaded prior to each use. This zero position is known to be influenced by changes in temperature, crank bolt and chain-ring bolt tension.

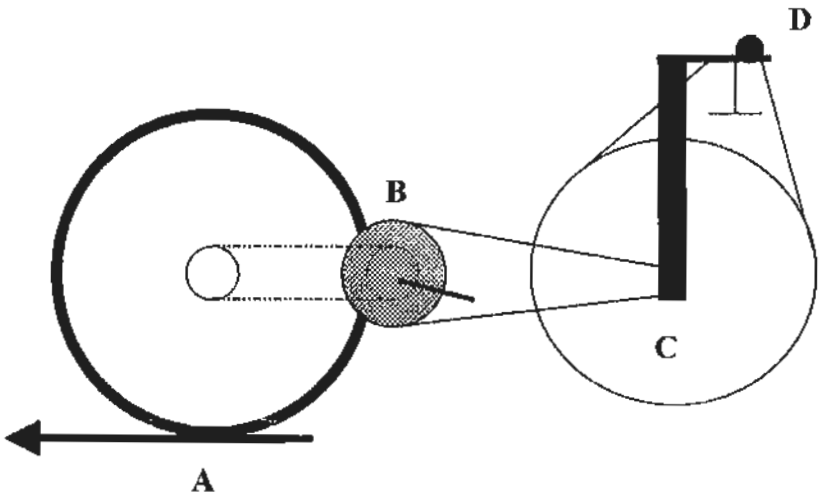


Figure 5.1 Schematic diagram of method used for comparing Monark and PMC ergometers.
A: Treadmill belt driving bicycle wheel. B: PMC driven by chain from bicycle wheel and connected to Monark flywheel. C: Monark ergometer flywheel. D: Braking device - friction belt, pulley and weight pan.

5.2.2 Comparison of ergometers

A weight pan loaded, friction braked Monark 814 ergometer (Monark, Varberg, Sweden), modified to accept a PMC, was used to compare known braking powers between the respective ergometry systems. To minimise the variations in power output, seen within and between pedal revolutions during normal cycling use (Sargeant and Davies, 1977), the Monark ergometer was further adapted to be driven by a motorised

treadmill (Woodway, GMBH, Weil-am-Rhein, Germany). The treadmill was used to drive a bicycle rear wheel which was connected in turn to the Monark ergometer via the PMC and a second chain drive (see Figure 5.1). A new drive chain and friction belt were fitted to the Monark ergometer at the start of the study.

5.2.3 Calculated braking power

The Monark ergometer braking power is produced by applying a frictional load to the rotating flywheel. Braking power at the Monark ergometer flywheel was calculated from Equation 5.1 below.

Equation 5.1

$$\text{Power (W)} = \text{torque (Nm)} \times \text{angular velocity (rad.s}^{-1}\text{)}$$

The mass of each braking weight was confirmed with a digital balance to the nearest 0.5 g. Acceleration due to gravity was assumed to be 9.81 $m.s^{-2}$. The radius of the flywheel was calculated by dividing its circumference, (determined with a flexible steel measure to within 1mm), by 2π. A cycle computer was used to record the flywheel revolutions.

5.2.4 Experimental protocol

A total of four comparative trials were conducted under varying conditions. During trials 1 and 2 the Monark ergometer was treadmill driven to elicit a cadence of approximately 90 $rev{\cdot}min^{-1}$. This rate was chosen as it is similar to the preferred cadence of well-trained cyclists pedalling a Monark ergometer (see Chapter 9). These trials were conducted on two separate occasions to provide a measure of test-retest reliability. For trial 3 the treadmill speed was increased to give a cadence of approximately 150 $rev{\cdot}min^{-1}$. Trial 4 was conducted with a volunteer pedalling the Monark ergometer rather than driving it with the treadmill. This trial was undertaken to examine whether pedalling the Monark ergometer altered the calculated agreement between the two ergometers. Braking weights were applied to generate 13 distinct braking powers ranging from approximately 50 W to 550 W for trials 1, 2 and 4. Due to the greater flywheel angular velocity in trial 3, a greater range of braking powers (80 W to 935 W) were examined. Prior to each trial the Monark ergometer was run for several minutes to warm the flywheel and minimise any possible change in the coefficient of friction between the Monark ergometer friction belt and flywheel (Woods et al, 1994). Each braking load was maintained for at least

75 s with power recorded simultaneously for Monark ergometer and PMC. All data were collected for a minimum of 60 s, averaged over 1 s intervals. The initial 15 s data collection at each new braking load were disregarded to allow sufficient time for the ergometers to stabilise.

5.2.5 Statistical analysis

A scatter diagram and correlation coefficient (r^2) were used to examine initially the linearity of the relationship between Monark ergometer and PMC. Limits of agreement (Bland and Altman, 1986) were then determined to quantify the differences between Monark ergometer and all PMC. The limits of agreement were set at 95% and are presented as both absolute values and in a ratio form (i.e. percentage) similar to that recommended by Nevill and Atkinson (1997).

5.3 Results

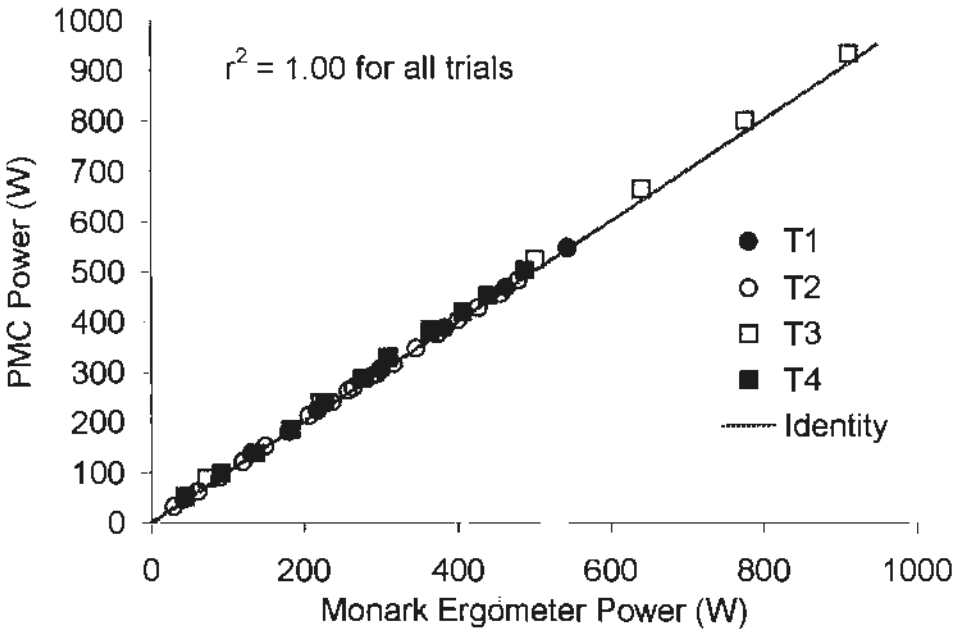


Figure 5.2 Correlation between Monark and PMC ergometers for all four trials.

An almost perfect linear relationship and correlation (r^2 = 1.00) was observed between the Monark ergometer and PMC for all trials (Figure 5.2). The limits of agreement for the two forms of ergometry are based upon the variability in PMC values compared with

those, calculated from first principles, for the ME. A Bland-Altman plot revealed a small bias and ratio effect in the differences between Monark ergometer and PMC, an example of which is provided in Figure 5.3. These factors act to decrease the apparent agreement between methods. Nevill and Atkinson (1997) suggest these problems may be minimised by log transformation and subsequent reporting of the data as ratio rather than absolute limits of agreement. The data in this study appeared not to exhibit heteroscedasticity and to be over-corrected by log transformation. Consequently, a linear regression equation was derived for the PMC from Monark ergometer and the residuals used to calculate limits of agreement (Figure 5.4).

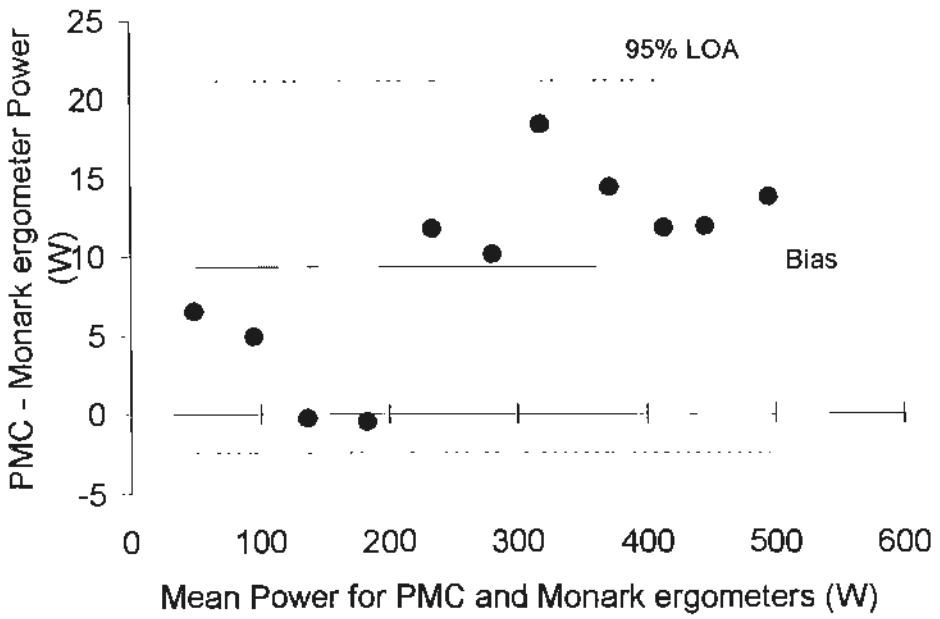


Figure 5.3 Mean difference and 95% limits of agreement for Monark and PMC ergometers for Trial 4.

The regression equation, absolute and estimated percentage limits of agreement for each PMC trial are presented in Table 5.1. A small degree of variability was found between the Monark ergometer and PMC with 95% of the differences being within ± 2.1 W; expressed in a ratio form this corresponds to approximately ± 1.8%. Subtle variations in treadmill speed between trials altered the Monark ergometer braking power and prevented a direct test-retest comparison of the PMC. Reliability may be assessed

indirectly however, by comparing ME-PMC regression and agreement for Trials 1 and 2 (Table 5.1).

Table 5.1 Regression and limits of agreement (LOA) for Monark ergometer and PMC.

Trial	Range (W)	Regression Slope	Regression Y-Intercept	95% LOA (W)	95% LOA (%)
1) 90 rev·min^{-1}	50-550 (W)	0.996	4.8	± 2.1 (W)	± 1.78 %
2) 2nd trial	30-485 (W)	0.997	1.9	± 3.6 (W)	± 1.87 %
3) 150 rev·min^{-1}	80-935 (W)	1.006	15.6	± 3.3 (W)	± 1.05 %
4) Pedalling	50-500 (W)	1.028	1.8	± 8.6 (W)	± 5.95 %

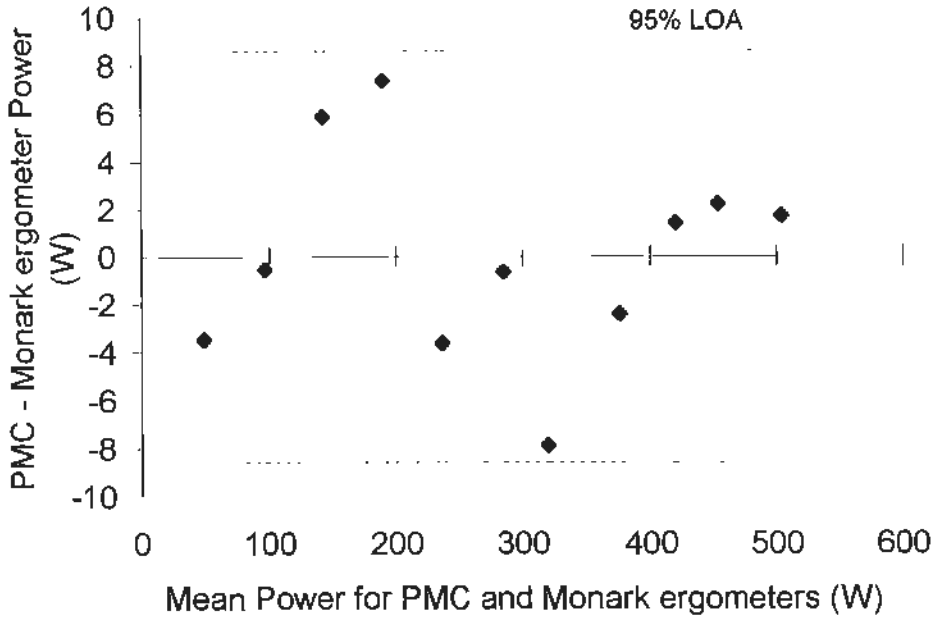


Figure 5.4 Mean difference and 95% limits of agreement for residuals of regression between Monark and PMC ergometers of Trial 4.

5.4 Discussion

This study has demonstrated a close agreement between the Monark ergometer and PMC at a cadence of 90 rev·min^{-1}. The almost perfect linear relationship and the low variability in the differences between the two forms of ergometry provide strong evidence for the validity of the PMC. The manufacturer of the PMC suggests an error range for this "Professional" model of ± 2.5%. The data from this study corroborate these figures. Furthermore, a much closer greater degree of agreement was found between the two ergometers in the present study than for those reported by Wilmore et al. (1982).

The repeatability of the PMC could not be examined directly with limits of agreement due to subtle differences in treadmill speed between trials. This resulted in different braking loads being generated by the Monark ergometer for each trial. A comparison of regression coefficients for the repeated trials does however, provide an indirect assessment of repeatability. From the regression equations in Table 5.1 it can be seen that the PMC demonstrate changes in gradient of 0.1% and the Y-intercept varies by less than 3 watts. It seems likely therefore, that the reliability of both ergometry systems is high. Further indication for the high repeatability of the PMC may be found by comparing predicted values from the regression equations. Across the range of braking powers examined, the between regression differences are greatest at the Y-intercepts. This highlights the importance of determining an accurate zero position for the PMC prior to use.

Small but discernible bias and ratio effects are apparent in a Bland-Altman plot of the data for Trial 4 (Figure 5.3). Not all trials were affected to the degree depicted, but the 95% limits of agreement were reduced in every case by correcting for bias and ratio effects with linear regression. With uncorrected data the Bland and Altman (1986) method still finds good agreement with a bias of 3.6 and 1.3 W and 95% limits of agreement of ± 2.4 and ± 3.6 W for Trials 1 and 2 respectively. Nevill and Atkinson (1997) suggest that most measurements recorded on a ratio scale are subject to heteroscedasticity and encourage researchers to adopt ratio limits of agreement. These

authors suggest that heteroscedasticity occurs because data recorded on a ratio scale have little potential for absolute error for measures of low value. As the value of the measure of interest increases so too does the potential for larger absolute errors. Nevill and Atkinson also point out that it is common to envisage errors, i.e. disagreement between methods, in ratio (i.e. percentage) form, as opposed to absolute differences. Consequently, Nevill and Atkinson propose the use of log-log transformation prior to the calculation of limits of agreement.

The data from the current study do not exhibit clear signs of heteroscedasticity, but a small bias and ratio difference is observed. In this situation a log-log transformation was not thought to be appropriate, as this transformation forces the relationship between the two methods to the origin (i.e. Monark power = PMC power = 0 W). Since the calibration of the PMC is achieved by adjusting the zero position, it is clear a priori that the two ergometry systems may be reasonably expected not to agree precisely at 0 W. The strong linear relationship between Monark ergometer and PMC observed in all cases, made removal of the bias and ratio effect by linear regression reasonable. This procedure cannot remove heteroscedasticity, but does allow an unbiased and ratio corrected assessment of the limits of agreement. The use of linear regression prior to comparison also enables both constant and ratio differences to be quantified separately. As the Monark ergometer braking power is not thought to reflect true power input, (see below), it appears more useful to examine the underlying agreement rather than the absolute differences. A regression correction was considered most appropriate method of achieving this.

A further comparison between Monark ergometer and PMC was conducted at a cadence of 150 rev.min^{-1}. This different cadence did not alter the linearity of the relationship between ergometers. Indeed, the increased angular velocity of the flywheel permitted a greater range of braking power outputs to be examined. This is probably the cause of the increased percentage agreement for Monark ergometer and PMC. The other noticeable change with the faster pedal frequency is a greater bias reflected as an increased Y-intercept in the linear regression equation for this trial (Table 5.1). This is entirely consistent with increased frictional losses in the power transmission due to the greater angular velocity of this system.

The manufacturer of Monark ergometer acknowledges that a difference exists between power calculated from braking force and the true power input (Monark Instruction Manual). This is caused by losses in the transmission of force from the pedal to the braking point at the flywheel. Monark suggest 9% of power input is dissipated in this process. Lakomy (1986) points out that power input is further underestimated in certain situations when the inertia of the flywheel is ignored. Changes in energy input are associated with changes in flywheel angular velocity and not accounted for with the conventional equations of Monark ergometer braking power. In sprint tests requiring large accelerations Lakomy calculates that uncorrected peak power may underestimate corrected values by over 35%. The effect of the small changes in flywheel speed during steady state pedalling is not known. As the PMC is thought to measure power input, any changes in Monark ergometer flywheel angular velocity may create a disparity between the two ergometry systems. Accordingly, a motorised treadmill was used to drive the Monark ergometer in an attempt to minimise variations in power input.

Trial 4 was conducted to examine the effect of pedalling the Monark ergometer on the agreement between methods. The 95% limits of agreement were found to be almost three times wider for this trial than the other conditions (± 8.6 W - Table 5.1). This observation justifies the use of the treadmill to drive the ergometer in an attempt to minimise variations in power output. Furthermore, this finding raises the possibility that the small variations seen within and over pedal cycles may significantly affect the calculated power output due to the unaccounted energy for each flywheel acceleration or deceleration (Lakomy, 1986). The increase in the regression equation slope for Trial 4 suggests that this unaccounted energy may result in an underestimation of the true work rate.

An unexpected finding of this study is the close agreement in absolute power between the two ergometry systems. Due to the different sites of power measurement the PMC were expected to record significantly higher braking powers than the Monark ergometer. This is because power measured by the PMC includes bottom bracket and wheel bearing friction, and losses in the chain drive. None of these factors is included in the calculation of Monark power. Whitt and Wilson, (1974, pp. 134) suggest that in conditions similar to the present study a new clean chain may reduce mechanical efficiency by only 1.5%. These authors also suggest that the total frictional losses of an ergometer are likely to be

approximately 5%. Kyle and Caiozzo (1986) examined frictional losses directly by using a motor to drive a Monark ergometer at a constant pedal rate and comparing power input with output. At a cadence of 50 rev·min^{-1} Kyle and Caiozzo found the percentage of energy lost increased with power output from 1.9% at 100 W to 3.9% at 300 W. Extrapolation of Kyle and Caiozzo's data suggests frictional losses could be greater than 5% at the highest braking powers in this study, particularly as their adopted cadence was approximately half that of the present study. Larger losses in mechanical efficiency have been reported by Woods et al. (1994) and Hibi et al. (1996) of 2-14% and 17-49% respectively. Both studies compared power input with output. Woods et al. used a pendulum braked Monark for their comparisons, but report experiencing problems with zero stability and load creep. Hibi et al. made their comparisons whilst subjects performed a 3 s maximal effort, therefore conditions were not steady state and power outputs were quite high (around 1 kW). Both studies are in agreement with Kyle and Caiozzo (1986) finding that the percentage error is variable over the range of braking powers examined. Hibi et al. observed that the energy losses in their study appear to be dictated by the magnitude of the forces applied and/or the angular velocity of the transmission system. Given the close agreement in the present study between Monark ergometer and PMC, it seems likely that both ergometers underestimate true power input.

The surprisingly close agreement between Monark ergometer and PMC could be explained by the dynamic calibration procedure employed by the manufacturer during fabrication. Each PMC is fitted to a lathe and connected by a bicycle chain to the dynamometer which measures the torque generated (U. Schoberer, SRM, personal communication). In principle therefore, the calibration slope obtained should include much of the frictional losses in the power transmission. As these are likely to vary with pedal rate and power output this assumption remains to be verified. Certainly, the small difference from unity in the gradient of the PMC regression equations (0.4%) does not appear consistent with the different points of power measurement on the Monark ergometer (flywheel vs. crank).

In conclusion, the comparison of new devices and techniques against criterion measures enables the extent of agreement to be assessed. A new device found to agree closely with the criterion measure may then be used in its place. In the present study the relationship

between PMC and Monark ergometer has been demonstrated to be highly linear, with only small differences between methods. An absolute difference between ergometry systems of 2 to 3.6 W was found between ergometer systems, corresponding to relative differences of 1 to 2%. Van Praagh et al. (1992) suggest an acceptable margin of mechanical error in ergometry should be considered to be less than 5%. The PMC examined was found to be within this standard. It is concluded that the PMC provides a valid method of assessing power output in the laboratory environment. The PMC ergometer provides a superior power output measurement system in situations where marked changes in flywheel angular velocity occur. Furthermore, the appreciable difference between ergometers found when the Monark was pedalled, may recommend the use of PMC even during constant power conditions. Further research is required to establish the long term reliability of the PMC in the laboratory, and its agreement with true power input.

Chapter 6: Calibration of open circuit spirometry

6.1 Introduction

During all experiments energy expenditure was determined by open circuit spirometry. These experiments were conducted to assess the validity of the open circuit spirometry procedure to be used in this thesis. In particular, the aim was to quantify the effect of factors that influence the measurement accuracy of expired gas concentration and volume.

6.2 Part I: Measurement of gas concentrations

This study examined the reliability of the gas sampling procedure. In addition, the influence of the residual volume of a Douglas bag after its evacuation on subsequently collected gas concentrations was determined. Also the rate of leakage or diffusion of collected gases from the Douglas bag was measured.

6.3 Method

6.3.1 Reliability of determining gas concentrations

Approximately 100 L of expirate was collected from a subject undertaking moderate intensity exercise. The concentration of O_2 and CO_2 were repeatedly determined from this bag on 20 separate occasions to determine the variability in sampling. During repeated sampling the gas analysers were running continuously and were re-calibrated after analysis of 10 samples.

6.3.2 Residual volume

The residual volume remaining after evacuation was determined in 13 different Douglas bags and one Douglas bag on 6 separate occasions. The residual volume was measured by gas dilution. This method was preferred to volumetric measurement as the variability in residual volume was thought to affect subsequently measured gas concentrations more

profoundly than gas volumes. During moderate intensity exercise about 50 L of expirate was collected in each Douglas bag. These bags were subsequently analysed for O2 and CO_2 concentrations and evacuated with a vacuum pump following normal laboratory procedures. Immediately following evacuation a Hans Rudolph syringe (Hans Rudolph Inc., Kansas City, MO. USA.) was used to introduce exactly 7 litres of outside air into each Douglas bag. The 7 litre air sample was gathered from outside the laboratory building well away from any possible contaminating ventilation exhaust systems and assumed to consist of 20.93% O_2 and 0.03% CO_2. The Douglas bags were then re-analysed for the new gas concentrations. The residual volume was calculated by simultaneous equation by both the changes in O_2 and CO_2 concentrations. The resolution of this method was approximately 30 ml.

6.3.3 Gas exchange between Douglas Bag and ambient air

The rate of exchange of O_2 and CO_2 between a Douglas bag and the laboratory environment was measured by periodically determining the gas concentration of the bag over a period of 192 h. Approximately 70 L of expirate was collected in a Douglas bag from a volunteer engaged in moderate intensity exercise. The subject was requested to adopt a slow, deep breathing pattern to maximise the respective changes in F_EO_2 and F_ECO_2. It was assumed that all gas exchange between the Douglas bag and environment would be reflected by a concomitant change in the concentration of gases within the bag.

6.4 Results

6.4.1 Reliability of determining gas concentrations

Table 6.1 provides details of the mean, standard deviation (SD) and coefficient of variation (CV) for the 20 repeated gas samples. The O_2 analyser exhibited slightly less variability than the CO_2 analyser as evidenced by the respective CV's of 0.05% and 0.45%.

Table 6.1 Variability in 20 samples from a Douglas bag.

	Concentration O_2%	Concentration CO_2%
Mean	16.18	4.44
SD	0.01	0.02
CV	0.05%	0.45%

6.4.2 Residual volume

Table 6.2 Example residual volume calculation. See text for details.

Gas	Prior % concentration	Post % concentration	Calculated residual volume (L)
O_2%	16.58	20.14	1.553
CO_2%	4.25	0.84	1.622

Example data for a residual volume calculation are provided in Table 6.2. The composition of gases in the residual volume were assumed to be those of the prior gas concentration. The post concentration is that measured after adding 7 L of outside air. If no residual volume was present concentrations of 20.93% and 0.03% for O_2 and CO_2 were expected. Any change from these concentrations was assumed to reflect the extent of dilution by the residual volume. The mean residual volume for 13 separate Douglas bags was between 1.487 and 1.582 L as determined from changes in O_2% and CO_2% respectively. The mean, SD and CV for all 13 Douglas bags and for the repeated measures on 1 bag are provided in Table 6.3.

Table 6.3 Residual volume for 13 Douglas bags and 6 repeated measures on 1 bag.

	Residual volume from O_2% (L)		Residual volume from CO_2% (L)	
	13 bags	1 bag	13 bags	1 bag
Mean	1.487	2.051	1.552	2.045
SD	0.228	0.164	0.226	0.204
CV	15.4%	8.0%	14.6%	9.9%

Table 6.4 provides example data of the effect of various reasonable changes in residual volume and its composition. The changes in residual volume reflect ± 2 SD from the mean on subsequent gas sample composition (Table 6.3). The example changes in gas

composition are based on the author's experience of likely extremes and a "typical" F_EO_2 of 16.25%. Also calculated is the effect of a residual volume comprised of room air. This would cause the greatest error in measurement of a subsequent gas concentration, for a sample of expirate with an O_2 concentration of 16.25% actually being recorded as 16.39%.

Table 6.4 Effect of changes in residual volume and its gas concentration on measured concentration of $\%O_2$ for a 40 L Douglas bag sample.

Example #	Actual O_2%	Residual volume	Residual $\%O_2$	Measured $\%O_2$	Absolute O_2% difference
1	14.50	1.487	18.00	14.63	0.13
2	16.25	1.030	14.50	16.29	0.04
3	16.25	1.944	14.50	16.33	0.08
4	16.25	1.487	20.93	16.42	0.17

6.4.3 Gas leakage or diffusion from Douglas Bag

An essentially linear relationship between time and change in gas concentrations for both O_2 and CO_2 was observed (Figure 6.1). The rate of O_2 loss from the Douglas bag was slower than CO_2 with concentration changes of 0.005% $O_2 \cdot h^{-1}$ and –0.015% $CO_2 \cdot h^{-1}$.

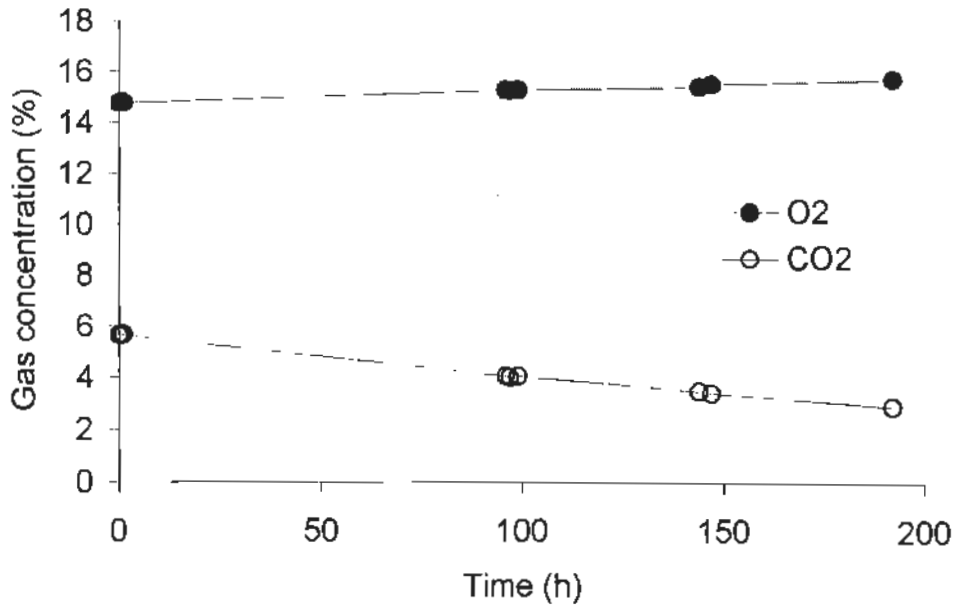


Figure 6.1 Rate of loss of O_2 and CO_2 over time.

6.5 Discussion

6.5.1 Reliability of determining gas concentrations

This study has demonstrated that the repeatability of the gas sampling procedure is extremely high. A slightly lower variability was experienced for O_2 than CO_2 but in both situations the CV was less than 0.5%. Factors that may influence the CV are the stability of the gas analysers, the reliability of the re-calibration procedure, and possible variation in flow rate through the gas analysers during sampling. The very low CV's suggest that these variables are unlikely to provide cause for concern.

6.5.2 Residual volume

The residual volumes of all the Douglas bags in the laboratory were determined by gas dilution in this study., After evacuation with a vacuum pump, typically 1.5 L of air remained in the Douglas bags. The consistent difference in residual volume as determined by O_2% and CO_2% is probably due to the different resolution of the respective analysers and the precision offered by the magnitude of the change in gas concentrations. The variability of the residual volume was quite high (CV ≈ 15%), but in absolute terms this amounts to 95% of bags varying by ± 0.4 L. This magnitude of change is not considered significant in the calculation of either $\dot{V}O_2$ or $\dot{V}_E$ provided the volume of expirate collected is reasonable (i.e. > 30 L).

The major implication of a variable residual volume is its potential for mixing with a subsequent gas sample. It was for this reason that the residual volumes were determined by gas dilution rather than syringe. For example, a residual volume comprised entirely of room air (O_2 = 20.93%) when mixed with expirate will increase the apparent F_EO_2% of a subject. The theoretical consequences of this dilution effect are explored in Table 6.4. Example 1 demonstrates that assumed extremes for F_EO_2 of 14.5% and 18.0% are mixed from a 40 L sample and 1.487 L residual volume respectively, the measured O_2% concentration for the sample would be 14.63% a difference of 0.13% from its actual value. This same example translated into differences in $\dot{V}O_2$ amounts to approximately 0.1 L·min^{-1}. This is more than twice the effect of the straightforward volume change without regard for its effect on gas composition. It is important to acknowledge that

smaller collection volumes will be affected to a greater extent. Examples 2 and 3 illustrate the effect of changes of ± 2 SD in residual volume on a common F_EO_2 value (16.25%). Example 4 demonstrates the greatest effect is created when the residual volume consists of ambient air.

James and Doust (1997) report that the CV of $\dot{V}O_2$ determination during moderate intensity treadmill running is 1.4%. The size and variability of the residual volumes found in this experiment could account for a large proportion of this CV. The residual volume mixing effect should be carefully controlled. This could be achieved by "flushing out" all Douglas bags prior to use and such a strategy is recommended. It may be possible to reduce the residual volume markedly by applying greater suction. The compliance of the Douglas bag makes minimisation of the residual volume difficult both to achieve and verify. Furthermore, the negative pressure induced by the removal of the residual volume will result in air being sucked into the bag as the subsequent collection is commenced. As the collection is timed to start on the inspiratory phase of the respiratory cycle this would result in variable error.

6.5.3 Gas leakage or diffusion from Douglas Bag

The rates of exchange of O_2 and CO_2 were both found to be fairly slow, although CO_2 was the more rapid. This is probably due to CO_2 being a more diffusive molecule. The rate of change in both O_2 and CO_2 per hour was below the resolution of their respective analysers, therefore the study was conducted over an extended period. The essentially linear loss of both gases supports the validity of this procedure. Clearly, reasonably short time periods (i.e. < 1 h) between gas sample collection and analysis will not result in meaningful changes in the measured gas concentrations.

In conclusion this study has demonstrated that the procedures adopted within this thesis for determining the concentrations of expired air samples are subject to small sources of error and variability. It is suggested that the following factors are likely to dictate the accuracy with which expired gas concentrations may be measured:

- The calibration of the gas analysers.
- The stability of the gas analysers.

- The control of partial pressure of water vapour.
- The diffusion or leakage of gases from the Douglas bag.
- The dilution of the gas sample with the residual volume.

Of these factors, the residual volume is thought to exert the most marked effect by mixing with the subsequent gas sample. This error may be minimised by "flushing" the Douglas bags immediately prior to use.

6.6 Part II: Gas Volume measurement

This study was conducted to determine the agreement between two different methods of determining gas volume and if appropriate to derive a calibration curve. The two methods being compared were measuring gas volume with a 7 L gas syringe and with a dry gas meter.

6.7 Method

A 7 L gas syringe (Hans Rudolph Inc., Kansas City, MO. USA.) was used to compare and calibrate known gas volumes with a dry gas volume meter (Harvard Apparatus Ltd, Edenbridge, UK.). This gas meter is used for normal laboratory gas volume measurement. The syringe method was found to be highly reproducible by using it to both fill and empty a Douglas bag. The repeated measurements obtained with the 7 L syringe were found to agree to within 50 ml irrespective of the volume syringed over a range of 10 to 150 L.

The 7 L syringe was connected to a Douglas bag via a gas tap and a short length of Falconia tubing. A range of volumes (10 to 160 L) of ambient air were syringed into a Douglas bag. The Douglas bag was immediately evacuated through the dry gas volume meter with a vacuum pump at a flow rate of 60 $L \cdot min^{-1}$. The system was bunged while the vacuum pump was started to check no leaks existed and to help maintain a constant rate of flow. Once empty the Douglas bag was gently squeezed to help expel as much of the residual volume as possible. In order to replicate normal laboratory practice all agreement trials reported were undertaken with any possible changes in the residual volume ignored.

6.7.1 Statistical analysis

The linearity of the relationship between both methods was determined by scatter plot and correlation coefficient (r^2). A calibration equation was obtained by linear regression with the syringed volume being derived from the metered volume. Finally, the agreement between the two methods was examined by determining the 95% limits of agreement (Bland & Altman, 1986) using the residuals from the calibration equation as described in the previous chapter.

6.8 Results

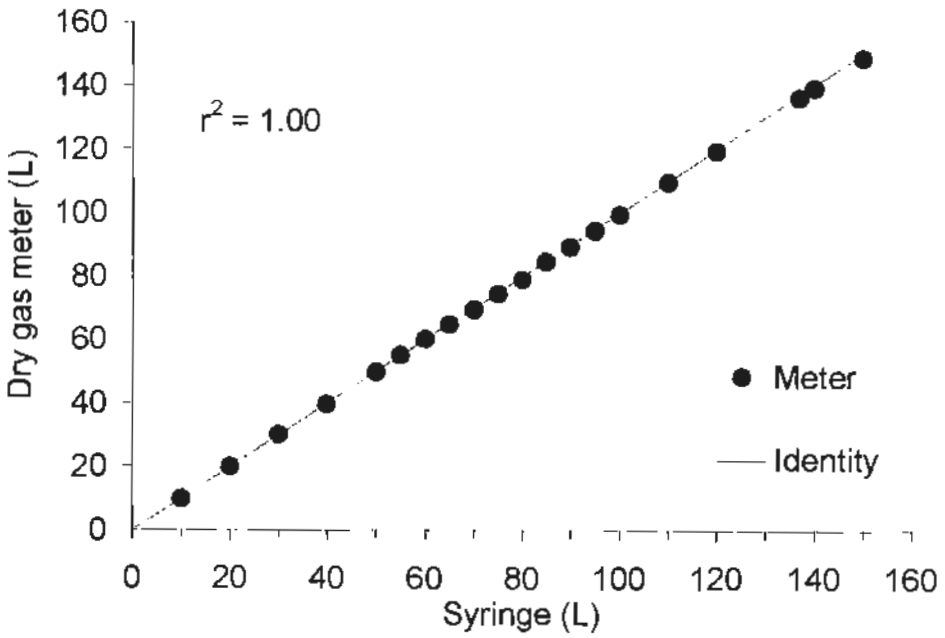


Figure 6.2 The relationship between syringe and metered volumes. Line of identity also shown.

Figure 6.2 shows the high linear correlation between syringe and gas volume meter values (r^2 = 1.00). A very small difference from unity was observed in the relationship between the two meters.

Equation 6.1

Syringe volume (L) = meter volume (L) x 1.008 – 0.069

The 95% limits of agreement were ± 0.82 L for the raw volume data and ± 0.49 L for the residuals from calibration Equation 6.1. The raw score differences had a bias of 0.59 L which was removed by linear regression. A Bland-Altman plot is shown for the residuals from the calibration equation (Figure 6.3).

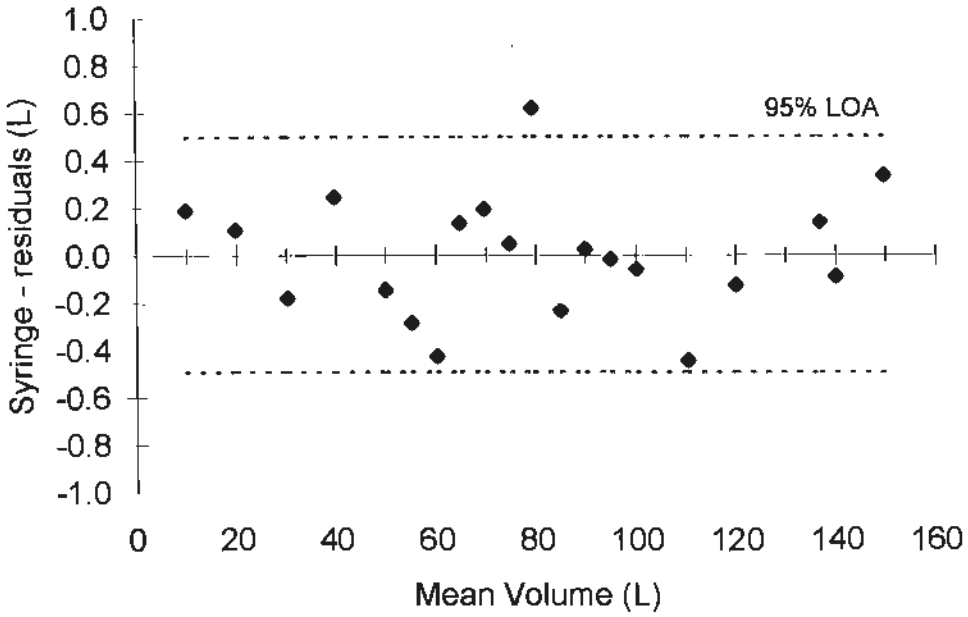


Figure 6.3 Bland-Altman plot of difference between syringe and corrected meter volumes, with 95% limits of agreement shown.

6.9 Discussion

This study has demonstrated that excellent agreement exists between a 7 L gas syringe and a dry gas volume meter with 95% of the differences falling within ± 0.49 L of the corrected volume. Even uncorrected volumes provide an acceptable level of agreement of less than ± 1 L. These values were found despite ignoring the possible changes between measurement methods caused by variation in the residual volume of the Douglas bag after evacuation. The degree of variation between methods found in the present study is considered insignificant provided a reasonable expirate sample volume is collected (i.e. > 30 L). It is concluded that the dry gas volume meter readings

corrected by Equation 6.1 may be satisfactorily used in place of the a 7 L syringe during normal laboratory work.

6.10 General discussion

The current and previous chapters have examined a number of factors which may affect the accuracy of $\dot{V}O_2$ and GE determination. High levels of agreement have been found between criterion and laboratory measures. The impact of the reported variability in these measures on calculated $\dot{V}O_2$ and GE are presented in Table 6.5 for comparison. These figures are based upon changes of 1.96 SD on an assumed $\dot{V}O_2$ of 3.12 $L \cdot min^{-1}$ and a power output of 230 watts. The figures highlight the importance of minimising the dilution effect of a Douglas bag residual volume which can potentially introduce three times the error of any other parameter.

Table 6.5 Effects of variation in gas and power measurements on calculated $\dot{V}O_2$ and GE.

Parameter	Δ1.96 SD	$\Delta\dot{V}O_2$ ($mL \cdot min^{-1}$)	ΔGE
PMC	± 2.1 W	NA	0.20%
Gas sampling variability	± 0.02%	17	0.09%
Residual volume dilution	± 0.08%	66	0.35%
Residual volume	± 0.45 L	10	0.14%
O_2 diffusion in 1 h	± 0.005%	04	0.02%
Gas volume	± 0.49 L	22	0.15%

Chapter 7: Effects of endurance exercise on gross efficiency and performance

7.1 Introduction

Racing cyclists often train and compete for several hours at intensities of 60% $\dot{V}O_{2peak}$ and above. Although glycogen depletion and dehydration are widely held to be limiting in exercise of this nature, the precise mechanisms by which they cause fatigue remain unclear (Conlee, 1987; Coggan and Coyle, 1991; Green, 1991; Sahlin and Seger, 1995). Previous work by this author has demonstrated that a progressive reduction in GE is apparent, even in elite cyclists, when exercising for 3 h at 60% $\dot{V}O_{2peak}$ (Passfield and Hale, 1997). A strong correlation ($r = -0.94$) was found between GE at the end of the 3 h trial and the reduction in PAPO. It is possible that a gradual decrease in GE may contribute significantly to the reduction in performance associated with prolonged exercise. The prolonged duration of the endurance exercise in the study of Passfield and Hale may have caused marked glycogen depletion and dehydration. Consequently, it is not known whether these factors were independent of, or associated with the observed reduction in GE and PAPO. Additionally, several studies have demonstrated the determining role of peak aerobic power in endurance cycling performance (Burke et al. 1977; Burke, 1980; Sjøgaard et al. 1986; Miller and Manfredi, 1987; Coyle et al. 1988; Coyle et al. 1991a; Hawley and Noakes, 1992; Wright et al. 1994), but the implications of an acute exercise induced reduction in PAPO for performance are not known.

This study was conducted to determine whether GE and cycling performance are compromised following sustained moderate intensity cycle exercise under conditions where both glycogen depletion and dehydration are not likely limiting factors. Furthermore, it was speculated that any reduction in GE would be associated with a concomitant change in aerobic performance. Accordingly, this study examined the effects of a 75 min exercise bout at 60% $\dot{V}O_{2peak}$ on changes in GE and performance in experienced endurance trained cyclists. The mean power output in a maximal 5 min trial was employed as the performance measure. Maximal exercise of this duration was

thought to be short enough to be repeatable without excessive fatigue, whilst still principally dependent on aerobic metabolism.

7.2 Method

7.2.1 Subjects

Ten male cyclists gave informed consent to take part in this study. Their physical characteristics are presented in Table 7.1. All subjects were engaged in regular endurance cycle training of at least 3-4 sessions per week at the time of the study. Most subjects were competitive cyclists in pre-season training routinely exercising for more than 75 min daily.

Table 7.1 Subject physical characteristics.

	Mass (kg)	Height (m)	$\dot{V}O_{2peak}$ ($L \cdot min^{-1}$)	$\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	$\%\dot{V}O_{2peak}$ at LT
Mean	69.0	1.79	4.11	60.4	59%
SD	13.4	0.07	0.50	7.4	2.4%

7.2.2 Pre-testing and familiarisation

The relationship between $\dot{V}O_2$ and power output and the measurement of $\dot{V}O_{2peak}$ and PAPO were determined from two separate tests as described previously (see section 4.8). Subjects were allowed to select their preferred pedal rate, which was then maintained throughout all testing periods, except for the performance trials. Additionally, three indices of lactate threshold were calculated from the submaximal test to examine their correlation with endurance performance. The first sustained rise in blood lactate above baseline (LT%), a 1 $mmol \cdot L^{-1}$ increase in blood lactate concentration above baseline (Coyle LT% - Coyle et al. 1983), and the power output eliciting a blood lactate concentration of 4 $mmol \cdot L^{-1}$ (4 $mmol \cdot L^{-1}$%) were all calculated and expressed as a percentage of $\dot{V}O_{2peak}$.

In order to ensure that the subjects were familiar with the strenuous nature of the performance trials and consistent in their pacing strategy (Foster et al. 1993), subjects

completed a minimum of two familiarisation performance trials before commencing the study. During these familiarisation trials subjects were encouraged to produce an evenly paced maximal effort.

7.2.3 Experimental procedures

A randomly ordered crossover design consisting of two conditions, a 75 min endurance trial and a control trial, was employed in this study (Figure 7.1). At the start of both conditions subjects performed a controlled 6 min warm-up at 60% $\dot{V}O_{2peak}$ (WU_1). Subjects then attempted to complete as much work as possible in a 5 min performance trial (PT_1). For the endurance trial subjects were then allowed a brief rest (3 min) before continuing to exercise at 60% $\dot{V}O_{2peak}$ for 75 min and repeating the 5 min performance trial (PT_2). In the control trial subjects rested for 72 min before repeating the 6 min warm-up (WU_2) and 5 min performance trial (PT_2). The braking load on the Monark ergometer was identical for all four performance trials and was calculated to elicit 90% of each subject's PAPO at a pedal rate of 95 rev.min^{-1}. During endurance exercise subjects ingested 10 ml·kg^{-1} body mass·h^{-1} of an 8% glucose polymer solution. A standard drink volume of 500 ml with a similar CHO content to the endurance trial was consumed in recovery between PT_1 and PT_2 during the control trial.

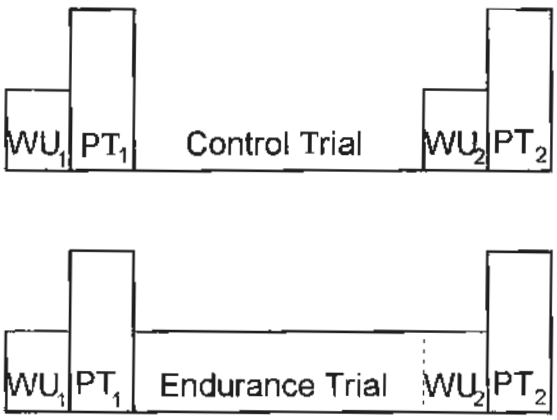


Figure 7.1 Schematic diagram of testing protocol.

Expired gas was collected from 5-6 min of WU_{1+2} for calculation of GE and also at 25 and 50 min during endurance exercise. For nine of the subjects expired gas was also

collected serially from 45 s during the performance trials. Thumb-prick capillary blood samples were taken at the end of both WU_{1+2} and 1 min post all performance trials for the determination of blood lactate concentrations. Seven of the subjects consented to the measurement of rectal temperature (T_R) which was recorded at 10 min intervals during endurance exercise. Throughout all exercise periods heart rate was recorded continuously, along with power output and pedal rate using the PMC and stored at 1 s intervals.

7.2.4 Data analysis

A Shapiro-Wilks test was used to test for normality of distribution for all data prior to further statistical analysis. In order to maximise statistical power, a paired t-test with a 0.05 two-sided significance level was used to compare pre to post condition differences (Δ) between control and endurance trials. This statistical procedure was planned a priori. Power analysis (nQuery Advisor, ver. 2.0) was performed with the data from initial subjects to ensure that sufficient subjects were recruited to have 80% power to detect a difference in PT_2-PT_1 (ΔPT) and in GE_2-GE_1 (ΔGE) measures between control and endurance conditions. Where appropriate the relation between variables was examined by Pearson's correlation coefficient. Statistical significance was accepted if $P < 0.05$ was found. All data are presented as mean and SD.

7.3 Results

The mean data for the warm up (WU) exercise and the change (Δ) for both control (C) and endurance (E) trial conditions are presented in Table 7.2. At the end of WU_1 before the endurance trial, the mean $\dot{V}O_2$ was 2.37 $L \cdot min^{-1}$ which represented 58 (4)% $\dot{V}O_{2peak}$. Following the endurance trial during WU_2, $\dot{V}O_2$ had increased (0.24 $L \cdot min^{-1}$) and RER was lower, these changes were significantly different from those in the control condition ($P < 0.005$). Power output was similar at all times and a significant difference in ΔGE was found for the endurance compared with the control condition ($P < 0.005$). The change in blood lactate from WU_1 to WU_2 was not significantly different between control and endurance conditions.

Table 7.2 Warm up (WU) 1 & 2 in control (C) and endurance (E) exercise - mean (SD)

	C-WU_1	C-WU_2	E-WU_1	E-WU_2	ΔC-WU	ΔE-WU
Power output (W)	186 (28)	185 (28)	182 (30.1)	183 (29.0)	-1 (3)	2 (5)
$\dot{V}O_2$ ($L.min^{-1}$)	2.45 (0.27)	2.56 (0.22)	2.37 (0.32)	2.62 (0.33)	0.11 (0.17)	0.24 (0.13)***
RER	0.97 (0.02)	0.98 (0.03)	0.97 (0.04)	0.94 (0.05)	0.01 (0.02)	-0.03 (0.03)***
GE (%)	22.2 (1.64)	21.6 (1.61)	22.6 (0.91)	20.7 (1.07)	-0.6 (0.5)	-1.8 (0.8)***
Lactate ($mmol.L^{-1}$)	1.6 (0.5)	2.1 (0.5)	1.3 (0.6)	1.4 (1.0)	0.5 (0.6)	0.0 (0.7)

*** The difference (Δ) is significant between endurance and control exercise ($P < 0.005$).

Table 7.3 Performance Trials 1 and 2 in control and endurance - Mean (SD)

	C-PT_1	C-PT_2	E-PT_1	E-PT_2	ΔCPT	ΔEPT
Mean power ($\bar{W}$)	323 (42)	323 (43)	321 (45)	309 (46)	0 (5)	-12 (7)***
Peak HR ($beats.min^{-1}$)	192 (8)	193 (8)	188 (7)	191 (6)	-2 (2)	3 (3)
Mean $\dot{V}O_2$ ($L.min^{-1}$)*	3.85 (0.42)	3.93 (0.45)	3.88 (0.54)	3.96 (0.56)	0.07 (0.05)	-0.07 (0.13)
Post ex. lactate ($mmol.L^{-1}$)	9.2 (1.5)	8.5 (1.3)	8.5 (2.3)	7.4 (2.1)	0.6 (1.2)	-1.1 (0.9)

***The change is significantly greater in endurance compared with control trial ($P < 0.005$).*N=9

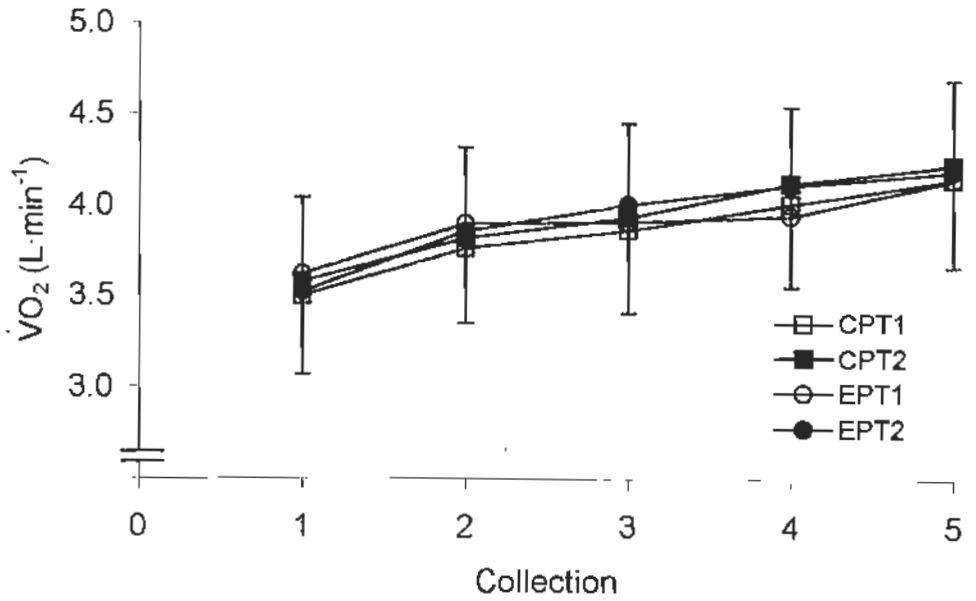


Figure 7.2 Performance trial $\dot{V}O_2$ before and after control (C) and endurance (E) conditions. Data are mean and SD, n = 9.

The performance trial data are shown in Table 7.3 and the $\dot{V}O_2$ data are also presented graphically in Figure 7.2. Average power output was identical for PT_1 and PT_2 in the control condition, but was reduced by 12 W following the endurance trial. This resulted in a significant difference in ΔPT between conditions ($P < 0.005$). The PT_1 - PT_2 differences for peak heart rate, mean $\dot{V}O_2$ and post exercise blood lactate were not significantly different between control and endurance trials.

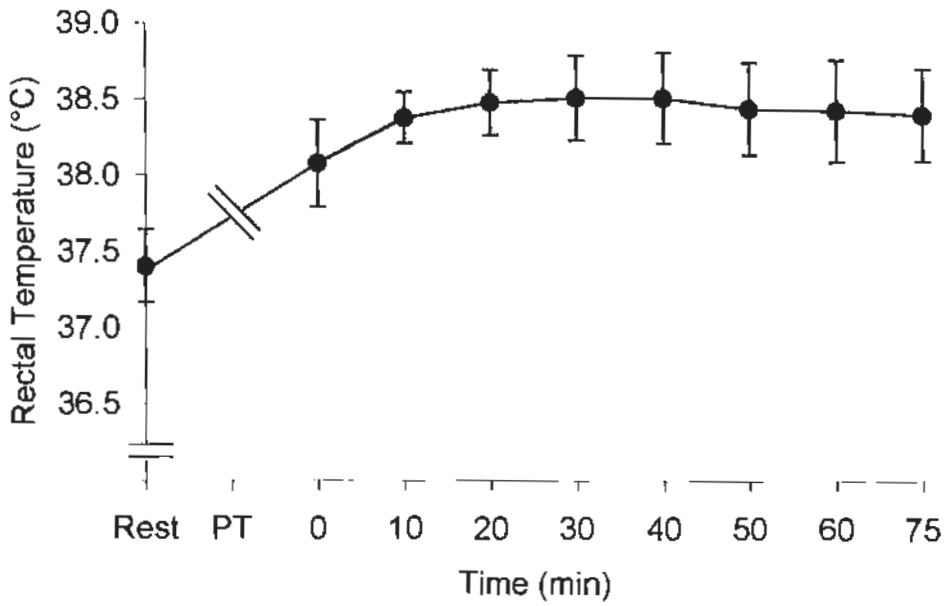


Figure 7.3 Rectal temperature during endurance trial. N = 7, mean (SD).

Table 7.4 Expired gas data at 25 and 50 min during endurance trial.

	25 min	50 min
$\dot{V}O_2$ ($L \cdot min^{-1}$)	2.49 (0.31)	2.57 (0.34)
RER	0.93 (0.04)	0.94 (0.04)
GE (%)	21.5 (1.10)	21.1 (1.09)
CHO oxidation ($g \cdot min^{-1}$)	2.29 (0.54)	2.44 (0.55)

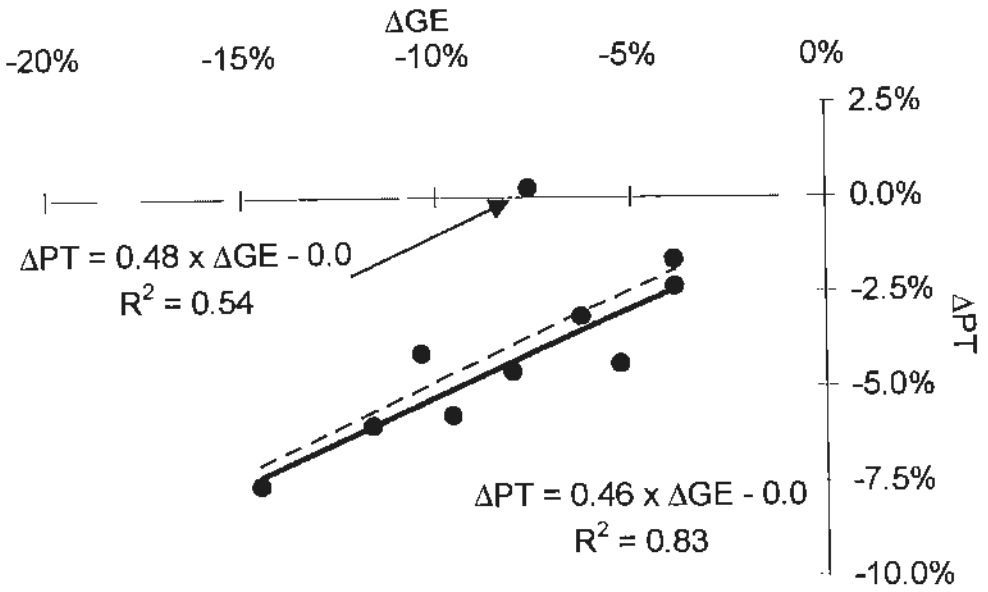


Figure 7.4 Correlation between ΔGE and ΔPT with and without outlier (indicated). Dotted line indicates regression with outlier included.

At the onset of endurance exercise T_R rose appreciably and then tended to plateau or even drop slightly over the last 25 min (Figure 7.3). Exercising $\dot{V}O_2$, RER, GE and estimated rates of CHO oxidation for gases collected during endurance at 25 and 50 min are presented in Table 7.4. A significant correlation between ΔGE and ΔPT was observed for all subjects of $r = 0.73$, $P < 0.05$. This correlation was strengthened by the removal of a significant outlier, (Snedecor and Cochran, 1967, pp. 157) to $r = 0.91$, $P = 0.001$. Both correlations are depicted in Figure 7.4. No significant correlation ($r < 0.57$) was found between $\dot{V}O_{2peak}$, PAPO or indices of lactate threshold (LT%, Coyle LT%, 4 mmol·L^{-1}%) and the change in PT for the endurance trial condition. A strong correlation ($r = 0.95$) was found between mean PT power output and PAPO and between mean PT power output and $\dot{V}O_{2peak}$. Weaker correlations observed mean PT power output and the various indices of lactate threshold (Table 7.5).

Table 7.5 Correlation between various indices of aerobic power and 5 min performance.

	Mean PT	PAPO	$\dot{V}O_{2peak}$	LT%	Coyle LT%
PAPO	0.95 $P < 0.001$	--			
$\dot{V}O_{2peak}$	0.93 $P < 0.001$	0.87 $P = 0.001$	--		
LT%	0.83 $P = 0.004$	0.94 $P < 0.001$	0.73 $P = 0.017$	--	
Coyle LT%	0.81 $P = 0.005$	0.87 $P = 0.001$	0.74 $P = 0.015$	0.90 $P < 0.001$	--
4 mmol·L^{-1}%	0.39 $P > 0.05$	0.46 $P > 0.05$	0.36 $P > 0.05$	0.59 $P > 0.05$	0.79 $P = 0.006$

7.4 Discussion

This study has found that both GE and 5 min performance are significantly reduced following 75 min at 60% $\dot{V}O_{2peak}$ in trained cyclists. The effect of moderate intensity endurance exercise on performance has not previously been examined in this manner. The significant decrements in GE (-8.4%) and PT (-3.7%) occurred despite the trained status of the subjects, the moderate intensity and non-exhaustive nature of the exercise and the ingestion of water and CHO. These findings were facilitated by the adoption of a novel experimental protocol, which yielded a very low performance trial CV of 1.6% (calculated from the two control PT and the first endurance PT). This is one of the lowest CV's reported for endurance performance data and is in marked contrast to the CV of around 26% for time to exhaustion (McLellan et al. 1995; Jeukendrup et al. 1996). Only the study of Hickey et al. (1992) has reported a lower CV for endurance performance (1.01%), but their protocol like most, does not enable pre - post comparisons. The ability of the present method to quantify exercise induced changes in performance and related parameters and its high reproducibility, recommend it as a useful model for future studies involving endurance exercise.

7.4.1 Factors limiting performance

The primary factors thought to limit endurance performance include hyperthermia, dehydration and substrate availability (particularly muscle glycogen) (Costill and

Hargreaves, 1992; Coyle and Montain, 1992; Terrados and Maughan, 1995). The precise role of each of these factors during moderate intensity exercise remains to be established clearly. The pattern of change in T_R suggests changes in body temperature were unlikely to have been a cause of fatigue. Following PT_1 T_R rose initially then stabilised or declined slightly over the final 25 min (Figure 7.3). Rectal temperatures were not particularly high (≈ 38.5 °C) and consistently less than 39.8 °C, a value Nielsen et al. (1993) have suggested to be critical as it coincides with exhaustion in the heat. Moreover, the small changes in temperature suggested subjects were able to maintain a comfortable thermal equilibrium.

The question of whether fluid ingestion during endurance exercise can or need fully compensate for the effects of dehydration remains the source of debate (Noakes et al. 1988; Coyle and Hamilton 1990). Recent studies have documented the deleterious effects of even moderate dehydration on performance (Walsh et al. 1994; Below et al. 1995). Both the studies of Walsh et al. and Below et al. found performance to be improved by fluid ingestion during approximately 1 h of exercise in the heat (32°C). By contrast, a recent study (Robinson et al. 1995) found that at normal laboratory temperatures prevention of mild dehydration did not enhance performance. All the above investigations required subjects to ride at 70-85% $\dot{V}O_{2peak}$, higher than the present study. As sweat rate is thought to be linked to metabolic rate (Saltin et al. 1968; Noakes et al. 1991), the effects of dehydration were more marked than in the present study. Finally, the effects of dehydration were minimised as all subjects consumed > 600 ml fluid during endurance exercise. Consequently, dehydration is not thought to have contributed significantly to the fatigue observed.

Whilst CHO availability appears to be related to the development of exhaustion during prolonged endurance exercise at 70-80% $\dot{V}O_{2peak}$ (Bergström et al. 1967; Hermansen et al. 1967; Coyle et al. 1986), its role in exercise of shorter duration at lower intensities is unclear. Recently, the ingestion of a CHO solution immediately prior to and during high intensity exercise of approximately 1 h has been demonstrated to enhance performance (Anantaraman et al. 1995; Ball et al. 1995; Below et al. 1995; El-Sayed et al. 1995; El-Sayed et al. 1997; Jeukendrup et al. 1997). These studies have demonstrated that the administration of 25-70 g CHO provides an ergogenic effect on sprint and endurance

performance following exercise of only 50-60 min duration. The ingestion of approximately 60 g CHO in the present study is likely to have reduced the rate of fatigue and benefited performance. The mechanism for this ergogenic effect remains obscure as Hawley et al. (1992) have found that less than 20 g of exogenous CHO are oxidised in the first hour of exercise.

The effects of glycogen availability during moderate duration exercise and on high intensity exercise following endurance exercise have not been extensively examined. A recent study by Hawley et al. (1997a) provides evidence against muscle glycogen availability being critical for moderate duration endurance performance. Hawley et al. found no benefit from a glycogen loading regimen on 1 h performance trial. In contrast, Pizza et al. have found that a 3-day high CHO diet enhanced time to exhaustion at $\dot{V}O_{2peak}$ following a 15 submaximal run at 75% $\dot{V}O_{2peak}$. It is possible however, that this improvement was mediated by changes in acid-base balance rather than muscle glycogen availability (see section 3.4.1.2). The effects of glycogen availability on high intensity exercise (reviewed previously - section 3.4) also appear equivocal. A review of available literature finds most studies are consistent with the hypothesis that only severe glycogen depletion reduces high intensity performance. Total CHO oxidation during the endurance exercise was calculated at 180 g glucose (Table 7.4) and is estimated to correspond to approximately 240 mmol·kg^{-1} dry muscle mass of glycogen (Bergström et al. 1967; Jansson and Kaijser, 1982; Coyle, 1995). The amount of oxidised CHO was considerably less than for the subjects of Bergström et al. (1967) exercising to exhaustion following a mixed diet (327 g CHO). Furthermore, the absence of a significant pre - post endurance trial effect on peak $\dot{V}O_2$, heart rate and blood lactate suggests that maximal rates of energy expenditure were not compromised. The extent to which muscle glycogen depletion affected performance in the present study cannot be precisely determined as glycogen concentrations were not measured. Reasonable evidence exists however, to implicate factors additional to muscle glycogen depletion.

7.4.2 Changes in performance and GE

A new finding of this study was the significant correlation between ΔGE and ΔPT ($r = 0.73$). This significant correlation was found despite the influence of an apparent

outlier. This outlying data point was recorded from a subject who suggested that his PT_1 was not maximal prior to the endurance trial. Peak heart rate, $\dot{V}O_2$ and post exercise lactate for this PT where all markedly lower than for this subject's other performance trials; a response in contrast to that of the other subjects (Table 7.3). An outlier test (Snedecor and Cochran, 1967, pp. 157) yielded a significant result ($P < 0.05$) for ΔPT of this subject. Accordingly, the data were re-plotted with this point omitted whereupon the correlation increased appreciably to $r = 0.91$ ($P < 0.001$ - Figure 7.4).

Horowitz et al. (1994) have demonstrated that GE[1] influences the power output sustainable in a 1 h performance trial. Two groups of cyclists were found to be significantly different in GE (20.4% vs. 21.9%) and power output (315 vs. 342 W) sustained during a 1 h performance trial despite achieving a matching $\dot{V}O_2$ (4.46 vs. 4.48 $L \cdot min^{-1}$). Neither maximal aerobic power nor the aerobic contribution to PT_2 appear to have been reduced following the endurance trial in the present study (see Table 7.3 and Figure 7.2). Any reduction in GE would be expected to cause a similar decline in performance. This is the first study to demonstrate that an acute reduction of cycling GE is associated with a parallel decrease in performance. This finding is consistent with the correlation between GE at 3 h and change in PAPO ($r = -0.94$) found by Passfield and Hale (1997). Indeed, a similar relationship was apparent in the present study between GE at the end of endurance exercise (WU_2) and ΔPT ($r = 0.72$, $P < 0.05$). These observations suggest that the change in GE provides an indication of the extent of fatigue. Those factors that dictate the change in GE during endurance exercise may, therefore, be implicated in limiting endurance performance. Maintaining endurance performance during prolonged exercise requires the ability to resist these changes in GE.

The gradient of the relationship between ΔGE and ΔPT (Figure 7.4) may indicate that these two parameters are not causally related. It is suggested, however, that the gradient of 0.46 rather than 1.0 is due to the first measurement of GE being made before rather than after the PT. Accordingly, it is likely that part of the reduction in GE was induced by PT_1 and consequently altered the slope of the relationship between these two

[1] Strictly, Horowitz et al. measured cycling economy not GE, but their suggested terminology is adopted here for clarity.

parameters. This proposition is supported by examination of the changes in GE occurring during the endurance ride (Table 7.4), which reveals that over 50% of the reduction in GE has occurred within the first 25 min of exercise. Gross efficiency is also lower during WU_2 compared with WU_1 in the control condition, reinforcing the suggestion that the first PT reduces subsequent GE measures. The GE measurement was taken before the PT because $\dot{V}O_2$ was likely to remain elevated for several minutes post exercise which would have invalidated any GE measurement recorded during this time.

7.4.3 Mechanisms for change in GE

A significant and progressive increase in $\dot{V}O_2$ during endurance exercise was found in this study (cf. Table 7.2 and Table 7.4). A similar $\dot{V}O_2$ drift has been documented in previous studies during endurance cycling exercise (Hagberg et al. 1978; Kalis et al. 1988; Sahlin et al. 1990; Hamilton et al. 1991; Hagan et al. 1992; González-Alonso et al. 1997), the cause of which is not known. Postulated causative mechanisms for a $\dot{V}O_2$ drift include changes in substrate utilisation, body temperature, lactate removal, ventilation and circulating catecholamines (Hagberg et al. 1978; Hagan et al. 1992; Poole et al. 1992). These factors are considered unlikely to have contributed significantly to the $\dot{V}O_2$ drift found in this study. The influence of changes in substrate utilisation as well as any minor variations in power output was removed by expressing the $\dot{V}O_2$ drift as a change in GE. The temporal dissimilarity in the pattern of change for T_R and GE, suggests a change in body temperature unlikely as a cause of $\dot{V}O_2$ drift. Furthermore, T_R has been demonstrated to respond in a similar manner to quadriceps muscle temperature during prolonged moderate intensity exercise with a lag of approximately 10 min (Saltin et al. 1968). Thus temperature mediated changes in whole body or active muscle $\dot{V}O_2$ are unlikely to have meaningfully influenced the ΔGE.

There is no obvious reason why a change in blood lactate concentration should result in an elevated exercising $\dot{V}O_2$. This is supported by the observation in the present study that blood lactate concentration was stable between WU_1 and WU_2 (Table 7.2), despite the ΔGE. Furthermore, muscle biopsy data obtained during exercise at 60% $\dot{V}O_{2peak}$, indicates that glycogenolysis and muscle lactate concentration are gradually reduced over time (Ball-Burnett et al. 1991). From the results of Aaron et al. (1992) the oxygen

cost of increased ventilation (mean 8 $L \cdot min^{-1}$) was estimated to contribute a trivial 14 $ml \cdot min^{-1}$ O_2 to the $\dot{V}O_2$ drift. Catecholamines were not measured during this study and therefore their effect on the ΔGE cannot be assessed directly. The possible role of catecholamines has been discussed previously in this thesis, however, (section 2.7.3), and is not thought to influence $\dot{V}O_2$ drift greatly.

It has been previously speculated that a progressive increase in muscle fibre recruitment may he responsible for the $\dot{V}O_2$ drift (section 2.7.3). This progressive recruitment may be necessitated by a reduction in the contraction efficiency or force generating capacity of the active fibres. Whilst the data from this study cannot confirm or refute this suggestion, all observations are consistent with these hypotheses and several other putative mediators of ΔGE have been dismissed. In conclusion, it seems likely that the pulmonary $\dot{V}O_2$ drift arises largely from the active (leg) muscle mass. This conclusion is supported by the finding that leg $\dot{V}O_2$ drifts in a similar manner to pulmonary $\dot{V}O_2$ (Sahlin et al. 1990; Poole et al. 1991; González-Alonso et al. 1997).

7.4.4 Aerobic power and endurance capacity

The importance of a high aerobic power as a key determinant of endurance performance was evident in this study. The high predictive ability of indices of aerobic power for cycling performance reported by many researchers (see Chapter 1) is reinforced by the strong correlation found between these variables and PT power in this study (Table 7.5). In contrast, the non-significant correlation between indices of aerobic power and the change in performance induced by the endurance trial suggests that a high aerobic power is not associated with the ability to reduce exercise induced fatigue. This finding is in agreement with previous observations by this author who was unable to find a significant correlation between indices of aerobic power and changes in PAPO following a 3 h endurance trial (Passfield and Hale, 1995).

7.4.5 Conclusion

In conclusion, this study has found that a non-exhaustive bout of moderate intensity exercise resulted in a significant reduction in 5 min performance and GE. The change in these two parameters was strongly correlated. The cause of these changes is not known.

Chapter 8: Effect of endurance exercise on 30s Wingate sprint performance in cyclists

8.1 Introduction

Whilst previous research has considered the effect of prior exercise on subsequent muscle function, no research has examined the combined effects of sustained exercise (i.e. duration of > 30 min.) at a moderate exercise intensity (i.e. approximately 60% $\dot{V}O_{2peak}$) on subsequent dynamic muscle function. Some insight can be gained from those studies which have examined some of these factors separately. Sargeant and Dolan (1987) have demonstrated that 6 min of prior exercise at intensities greater than approximately 70% $\dot{V}O_{2peak}$ resulted in a decline in subsequent maximal isokinetic cycling power output (MICPO). Rademaker et al. (1994a, 1994b) have found MICPO to be unaffected by 45 min of prior exercise at 80% $\dot{V}O_{2peak}$. The authors also present data from a parallel experiment however, which suggests that the effects of a rise in muscle temperature during exercise balanced those of fatigue. Sahlin and Seger (1995) found maximal dynamic and static leg extension torque to be compromised following exercise to exhaustion at 75% $\dot{V}O_{2peak}$. Other studies examining static muscle function after moderate intensity exercise have found no effect. Hoffman et al. (1985) found that 20 min of exercise at 60% $\dot{V}O_{2peak}$ did not reduce MVC, in contrast to exercise at 80% which resulted in a 30% reduction. The data of Hoffman et al. at 60% $\dot{V}O_{2peak}$ indicated a progressive pattern of fatigue however, which may become more marked following exercise of a longer duration. Prolonged cycle exercise at 60% $\dot{V}O_{2peak}$ either in the cool or the heat was not found to affect MVC (Nielsen et al. 1993). Statistical power was likely to be very weak due to the limited number of subjects recruited (n = 3 and n = 4). It is also unclear whether the results of studies on isometric muscle function apply to dynamic exercise, due to the different activation patterns involved (Saugen and Vollestad, 1996), and possibly different muscle group recruitment (Green and Patla, 1992).

The previous study in this thesis found that GE and 5 min performance are reduced hy a 75 min bout of exercise at 60% $\dot{V}O_{2peak}$ and the reduction in both variables is closely

associated. It was speculated that a cause for the decrease in GE may be a reduction in the force generating capacity of the active fibres. Further, it was also of interest to determine whether endurance exercise results in a reduction in short-term (i.e. largely anaerobic), power production which could also have contributed to the decline in performance observed.

The aim of the present study was to examine the effects of extended moderate intensity exercise (70 min, 65% $\dot{V}O_{2peak}$) on 30s maximal unpaced (Wingate) sprint performance in trained cyclists. Trained cyclists were recruited as subjects as they were very familiar with both extended and maximal exercise. A similar experimental design to the previous study, including a control trial was adopted.

8.2 Method

8.2.1 Subjects

Nine males, all experienced racing cyclists gave informed consent to take part in this study. Their physical characteristics are presented in Table 8.1. All subjects were engaged in regular endurance cycle training of at least 3-4 sessions per week at the time of the study. Most subjects were competitive cyclists in pre-season training generally exercising for 75 min or more on a regular basis.

Table 8.1 Subject physical characteristics

	Mass (kg)	Ht (m)	$\dot{V}O_{2peak}$ ($L \cdot min^{-1}$)	$\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	$\%\dot{V}O_{2peak}$ at LT
Mean	69.0	1.78	4.42	64.3	61%
SD	6.5	0.04	0.36	4.7	5.4%

8.2.2 Pre-testing and familiarisation

The relationship between $\dot{V}O_2$ and power output and the measurement of $\dot{V}O_{2peak}$ and PAPO were determined from two separate tests as described previously (see section 4.8).

Subjects were allowed to select their preferred cadence (90, SD 7.1 rev·min^{-1}), which was used throughout all pre-testing and the endurance trial.

Each subject performed at least two 30 s sprints prior to taking part in the study to ensure they were familiar with the maximal effort required.

8.2.3 Experimental procedures

A randomly ordered crossover design consisting of two conditions, a 60 min endurance trial and a control trial was employed in this study (Figure 8.1). In order to control the effects of changes in T_m and V_{opt} each sprint was preceded by 10 min warm up exercise (WU) at a standard cadence (65% $\dot{V}O_{2peak}$, 100 rev·min^{-1}). These conditions were maintained until the start of a 30 s Wingate sprint against a braking load of 0.09 kg·kg^{-1} body mass. During the endurance condition after the first warm up (WU_1) and sprint (S_1), subjects were allowed a brief rest (3 min) before continuing to exercise at 65% $\dot{V}O_{2peak}$ for 60 min. In the control trial subjects rested for the equivalent time (63 min). On completion of the exercise or rest period all subjects repeated the 10 min warm-up exercise (WU_2) before performing a second 30 s sprint (S_2).

Peak power output (PPO) measured as the highest 1 s value, mean power (MPO) and fatigue index (FI) were calculated for each sprint test. Expired gas was collected from 8 to 9 min of $WU_{1 + 2}$ for calculation of GE and also at 20 and 40 min during endurance exercise. Thumb-prick capillary blood samples were taken during the last min of both $WU_{1 + 2}$ and 3 min post all sprint performances for the determination of blood lactate concentration. Throughout all exercise periods heart rate was recorded continuously with power output and pedal rate using the PMC and stored at 1 s intervals. Between sprints subjects ingested approximately equal amounts of CHO (≈ 70 g) as 600 ml of a 12%, or 10 ml·kg^{-1}·h^{-1} of an 8% glucose polymer solution during control and endurance trial conditions respectively.

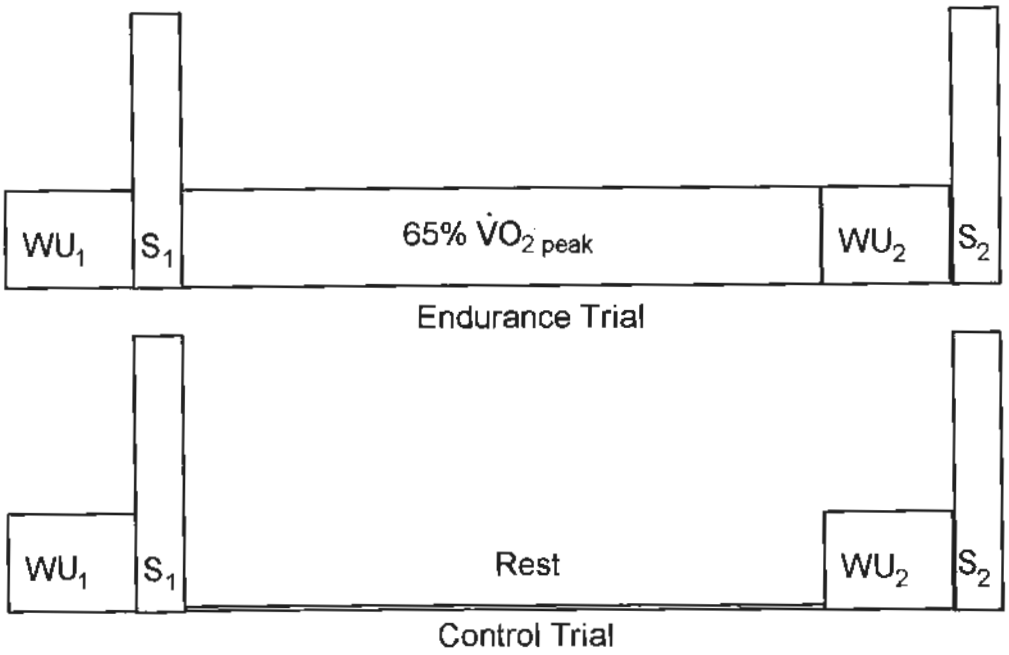


Figure 8.1 Schematic diagram of testing protocol

8.2.4 Data analysis

A Shapiro-Wilks test was used to test the normality of distribution of data prior to further statistical analysis. In order to maximise statistical power, a paired t-test with a 0.05 two-sided significance level was used to compare pre - post trial differences (Δ) between control and endurance trial conditions. This statistical procedure was planned a priori and power analysis (nQuery Advisor, ver. 2.0) was employed to ensure that sufficient subjects were recruited to have 80% power to detect a difference in control and endurance trial condition means for ΔMPO and ΔGE measures. A two-way ANOVA with repeated measures (exercise condition x time) and a Friedman test were conducted to test for differences in sprint cadence and time to PPO between sprints respectively. Where appropriate the relation between variables was examined by Pearson's correlation coefficient. Statistical significance was accepted if $P < 0.05$ was found. Data are presented as mean (SD) unless otherwise specified.

8.3 Results

The warm up (WU) data are presented in Table 8.2 below. The $\dot{V}O_2$ during WU_1 of both control and endurance trial conditions was equivalent to 67 (4.5)% $\dot{V}O_{2peak}$. The changes

between WU_1 and WU_2 (Δ) in the control condition were minor for $\dot{V}O_2$, RER, GE and blood lactate. In the endurance trial condition exercise $\dot{V}O_2$ increased whilst RER, GE and blood lactate all decreased. Consequently the changes in $\dot{V}O_2$ ($P < 0.05$), RER ($P < 0.05$), GE ($P = 0.01$) and blood lactate ($P = 0.01$) were all greater for the endurance in comparison with the control condition.

Table 8.2 Respiratory and blood variables during WU_1+$_2$ in control (C) and endurance (E) conditions and the calculated change (Δ).

	C-WU_1	C-WU_2	E-WU_1	E-WU_2	Δ CWU_{2-1}	Δ EWU_{2-1}
$\dot{V}O_2$ (L.min^{-1})	2.93 (0.33)	2.94 (0.32)	2.97 (0.35)	3.11 (0.31)	0.01 (0.09)	0.13 (0.12)*
RER	0.99 (0.04)	0.99 (0.04)	0.97 (0.03)	0.94 (0.04)	0.0 (0.02)	-0.04 (0.03)*
GE %	21.8 (0.7)	21.7 (0.9)	21.8 (0.8)	20.8 (1.4)	-0.1 (0.5)	-1.0 (0.8)**
Lactate (mmol.L^{-1})	2.2 (1.0)	2.1 (0.7)	2.1 (0.7)	1.5 (0.5)	0.2 (0.7)	-0.6 (0.5)**

*P < 0.05, **P = 0.01, for ΔC compared with ΔE.

The ΔPPO ($P < 0.05$) and ΔMPO ($P = 0.01$), but not ΔFI ($P > 0.4$) were significantly greater following exercise at 67% $\dot{V}O_{2peak}$ than the control condition (Table 8.3). The change in blood lactate concentration 3 min post sprint was greater also for the endurance trial condition in comparison with the control ($P < 0.005$). No significant correlation was found between the change in either peak or mean sprint power output and the change in GE ($P > 0.05$) for the endurance trial condition (Figure 8.2 - data shown only for ΔPPO). A 2-way ANOVA revealed no difference in peak cadence, cadence at PPO, nor in time to PPO between sprints (Table 8.3). The CV of sprint performance, calculated from the sprints in the control condition and the first sprint in the endurance trial, was 2.2% for MPO and 3.2% for PPO.

Table 8.3 Sprint power output, fatigue index, cadence and post sprint blood lactate concentration.

	$C\text{-}S_1$	$C\text{-}S_2$	$E\text{-}S_1$	$E\text{-}S_2$	$\Delta CS_{2\text{-}1}$	$\Delta ES_{2\text{-}1}$
Peak power output (W)	991 (177)	998 (178)	985 (150)	959 (169)	7 (29)	-27 (38)*
Mean power output (W)	667 (85)	668 (80)	663 (94)	628 (93	1 (11)	-35 (24)**
Fatigue index (%)	58 (17)	58 (17)	55 (14)	56 (13)	0 (4)	1 (3)
Lactate ($mmol.L^{-1}$)	8.5 (1.57)	8.2 (1.28)	8.6 (1.17)	6.6 (1.55)	0.3 (1.1)	-2.0 (1.3)***
Peak $rev{\cdot}min^{-1}$	139 (15)	139 (14)	142 (16)	137 (17)	--	--
Time to PPO (s)	3 (3-6)	4 (3-4)	4 (2-6)	3 (3-4)	--	--
PPO ($rev{\cdot}min^{-1}$)	123 (8.7)	122 (7.8)	125 (13.9)	124 (9.1)	--	--

*$P < 0.05$, **$P = 0.01$, ***$P < 0.005$, for ΔC compared with ΔE. Time to PPO = median (range).

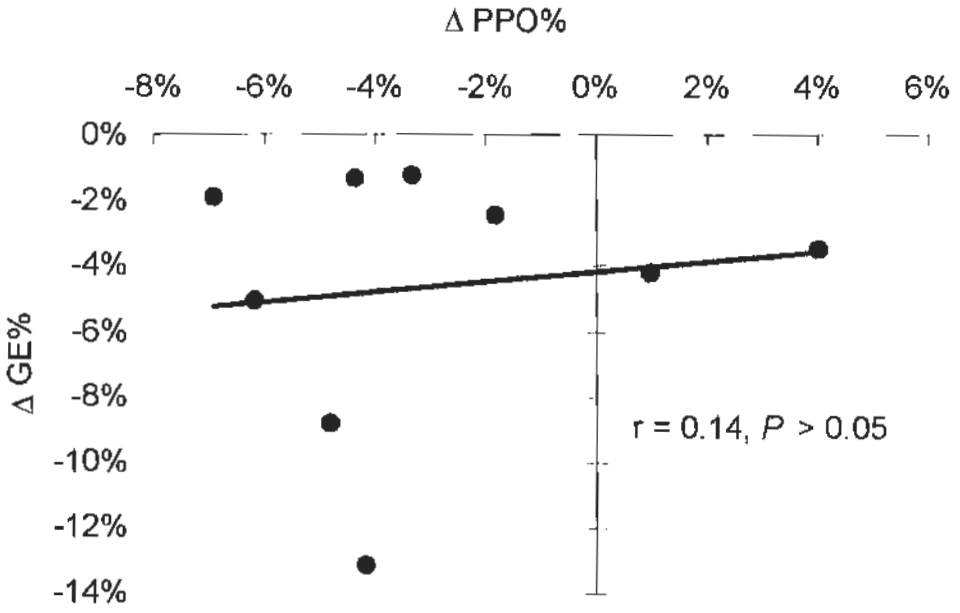


Figure 8.2 Correlation between ΔPPO% and ΔGE% for the endurance trial.

8.4 Discussion

This study has found that 70 min of exercise at approximately 65% $\dot{V}O_{2peak}$ results in a significant reduction in both PPO and MPO during a subsequent 30 s sprint. Peak power outputs in the present study were comparable with those found for similar calibre cyclists (approximately 990 W) by Tanaka et al. (1993). Several procedural differences make true comparison problematic and the 10 min of prior exercise in the present study may have influenced subsequent sprint performance (Sargeant 1987; Sargeant and Dolan, 1987).

8.4.1 Methodology

The greater relative reduction in MPO of 5.3% (-35 W) in comparison with the reduction in PPO of 2.6% (-27 W) could be indicative of a selective fatigue of factors more critical to the development of MPO. Alternatively, a greater measurement variability may have influenced the ΔPPO. This is supported by the lower CV for MPO vs. PPO (2.2% vs. 3.2%) and the observation that all subjects were consistent in experiencing a drop in MPO whilst two recorded an increased PPO following the endurance trial. Performance over a shorter duration is associated with a higher degree of variability. This is evident from a comparison of the CV's for the present and previous studies' (3.2 and 2.2 vs. 1.6% respectively), and is supported by the comparable findings of Hickey et al. (1992). These authors found that time-trial performance averaging 33 s had a CV of 2.4%, whilst longer time-trials of 12 and 105 min had lower CV's of around 1%. The reason for the greater variability in PPO may be due to an inherently greater sensitivity to variables such as subject motivation, skill and cadence.

8.4.2 Warm up exercise

In order to control for the influence of T_{in} and V_{opt} on PPO, each sprint was preceded by a 10 min warm up at 100 rev·min^{-1}. It is possible that part of the fatigue induced reduction in PPO was still masked by an increase in muscle temperature, but the extent of this effect is likely to have been minor. Hoffman et al. (1985) have found that T_m rises exponentially from rest at 60% $\dot{V}O_{2peak}$, tending to have reached a plateau by 10 min. Cadence at PPO ranged from 122-125 rev·min^{-1} (Table 8.3) which is typical of the

optimal cadence (V_{opt} - 110-140 rev·min^{-1}) reported previously for isokinetic cycling (Sargeant et al. 1981; McCartney et al. 1983; Sargeant et al. 1984; McCartney et al. 1985). No significant difference between sprints was found for the cadence at PPO, indicating that the starting cadence of 100 rev·min^{-1} was successful in constraining the attainment of PPO to around V_{opt}. This finding implies that the reduction in PPO following endurance exercise cannot be ascribed to PPO being elicited at a different point on the power-velocity curve. Differences in the energy required to accelerate the flywheel are also an unlikely cause for the difference in PPO. The measurement of power output by PMC ensures that all energy input in this manner is correctly accounted for as recommended by Lakomy (1986). Also the starting cadence of 100 rev·min^{-1} will have reduced the degree of acceleration required.

8.4.3 Force-velocity effects

As the sprint testing was not conducted at a constant cadence, the power output produced was expected to be influenced by the changes in cadence with respect to V_{opt}. As discussed above, the PPO is not thought to have been affected hy changes in cadence. Unsurprisingly, the lower MPO following the endurance trial required a slower mean cadence. The end cadence averaged across all sprints was 88 (SD 11) rev·min^{-1}. It is likely that the decline in power output during each sprint was augmented as cadence fell progressively below V_{opt}. Furthermore, it is tempting to suggest the ΔMPO following the endurance trial could also have been exaggerated in this manner. This suggestion is refuted however, by the observation that ΔFI was not different between conditions.

The functional significance of power-velocity related changes is challenged by the results of de Haan et al. (1989). These researchers found fatigue induced a 66% decrease in maximum shortening velocity and a concomitant leftward shift of the power-velocity curve of rat gastrocnemius muscle in situ. Comparable effects have been reported in humans with prior high intensity exercise resulting in a significant reduction in the rate of force development (Beelen et al. 1995). Beelen and Sargeant (1991) have demonstrated a selective reduction in PPO at fast but not slow cadences. These results suggest that muscle fatigue alters the force-velocity characteristics of muscle, reducing V_{opt}. Accordingly, neither conventional nor isokinetic ergometry assessments of muscular power output is likely to be optimal in situations where marked fatigue occurs.

A similar FI and rate of decline in power output was found in the present study to those employing isokinetic testing (55-58% vs. 50-60%, 24.4 vs. 24.5 $W \cdot s^{-1}$ respectively - McCartney et al. 1983, 1986). This finding is consistent with fatigue induced changes in V_{opt}, as a lower FI would otherwise be expected under isokinetic conditions. Finally, it is noted that extrapolation of laboratory sprint testing to competitive cycling situations is further complicated by the fact that power output increases in proportion to cycling velocity (and thus cadence) cubed.

8.4.4 Sprint energetics

The energetic contribution from ATP and PCr, glycogenolysis and aerobic metabolism to a 30 s sprint have been variously estimated to contribute 17-28%, 48-56% and 16-33% respectively (Serresse et al. 1988; Smith and Hill, 1991; Medbø and Tabata, 1993; Nevill et al. 1996). The PPO and power output generated in the first 10 s of an all-out test has often been used as an indicator of alactic work capacity (Serresse et al. 1988; Smith and Hill, 1991; Green, 1995). Several recent studies emphasise the significant contribution of PCr during the first part of the sprint. Phosphocreatine has been found to be decreased by 57% after only 6s (Gaitanos et al. 1993) and extensively depleted after 10 s (Hultman et al. 1991; Bogdanis et al. 1996). Preventing PCr resynthesis in one leg during recovery from sprinting reduces subsequent power output by 15%, largely in the initial 15s of the sprint (Trump et al. 1996).

In the present study, the post exercise reduction in PPO may therefore, implicate a glycogen-independent fatigue mechanism. Contradicting this contention, however, are the findings that glycogenolysis is activated rapidly at the start of exercise, reaching near maximal rates within 3 s and contributing significantly to ATP resynthesis even in the first few seconds (Hultman and Sjöholm, 1983; Jacobs et al. 1983; Hultman et al. 1991; Gaitanos et al. 1993; Nevill et al. 1996). This analysis implies there was overall a greater potential for glycogenolysis-mediated influence on 30 s sprint performance.

8.4.5 Effects of endurance exercise

A reduction in muscle ATP, PCr and glycogen levels is generally evident following endurance exercise and these changes are summarised in Table 8.4. Significant

reductions have been reported for the change in ATP despite being small in extent (Broberg and Sahlin, 1989; Green et al. 1989; Sahlin et al. 1990; Green et al. 1991), but they are not thought to be of practical significance. The change in PCr associated with endurance exercise is highly variable. Norman et al. (1987) found insignificant changes in PCr following exercise to exhaustion (mean time 80 min) at 68% $\dot{V}O_{2peak}$. By contrast, PCr was reduced by almost 60% at exhaustion after cycling at 67% $\dot{V}O_{2peak}$ in the study of Broberg and Sahlin (1989). The reason for this disparity is not clear. Several studies provide evidence that a substantial reduction in PCr occurs early in endurance exercise with only minor changes thereafter (Norman et al. 1987; Sahlin et al. 1990; Green et al. 1991; Ball-Burnett et al. 1991). Indeed, Sahlin et al. (1990) found PCr reduced to almost 50% of resting levels after only 5 min of exercise at 75% $\dot{V}O_{2peak}$. In the present study it seems probable therefore, that the majority of PCr depletion would have occurred during the warm-up prior to the first sprint.

Table 8.4 Changes in ATP, PCr and glycogen following endurance exercise

Authors	N	$\dot{V}O_{2peak}$ $ml \cdot kg^{-1} \cdot min^{-1}$	$\dot{V}O_{2peak}$ %	Duration	Δ ATP	Δ PCr	Δ glycogen
Green et al. (1991)	8	53	59	99 min	↓	↓ 15 min[1]	-43%
Ball-Burnett et al. (1991)	6	One leg	61	120 min	-5% [NS]	-13% [NS]	-68%
Green et al. (1989)	6	45	65	120 min	-22%	-63%	-80%
Hargreaves et al. (1996)	5	61	67	120 min	0% [NS]	-22% [NS]	-52%
Broberg & Sahlin, (1989)	8	47	67	65 min	-7%	-59%	-81%
Norman et al. (1987)	7	47	68	80 min	↓ [NS]	↓ [NS]	-66%
Sahlin et al. (1990)	7	45	74	75 min	-7%	-67%	-89%

↔ = no further change, ↓ unspecified reduction, [NS] $P > 0.05$, [1] no further reduction after 15 min

The role of glycogen depletion in the reduced sprint performance remains to be determined. It is evident from Table 8.4 that glycogen utilisation rates are variable during prolonged exercise. The study of Hargreaves et al. (1996) is notable in recruiting the fittest subjects and also reporting one of the lowest rates of glycogen depletion. The 52% glycogen depletion found by Hargreaves et al. (1996) followed endurance exercise of a similar exercise intensity, but over 40% longer than the present study. Several studies have demonstrated mixed muscle glycogen usage to be approximately 75-85 $mmol \cdot kg^{-1}$ dry mass during a maximal 30 s sprint (McCartney et al. 1986; Greenhaff et al. 1994; Bogdanis et al. 1996). Accordingly, these combined observations imply an extensive glycogen depletion prior to the second sprint seems unlikely.

The lower post sprint blood lactate concentration may indicate that muscle glycogen availability is associated with or causative in the reduced sprint performance (Jacobs, 1981b; Jacobs, 1987). Of note however, is the discrepancy between the exercise induced change in post sprint blood lactate of approximately -30% and the ΔMPO which was only -5%. The lack of stoichiometry in changes of lactate and MPO is suggestive of an associative rather than causative link between these variables. Green and Dawson, (1993) urge caution in the interpretation of post sprint blood lactate concentrations, which they point out may be confounded by changes in factors known to influence lactate kinetics and diffusion space (e.g. plasma volume and muscle perfusion). A similar discrepancy was noted between muscle lactate concentrations and isometric fatigue following glycogen manipulation (Jacobs, 1981b). It seems likely therefore, that lactate production rather than dispersal is the cause of the reduced blood lactate. Additional factors that may have acted to reduce blood lactate production are an exercise induced inhibition of phosphorylase activation (Constable et al. 1986) and increased pyruvate dehydrogenase complex activity (Spriet et al. 1989; Timmons et al. 1996).

During repeated sprints it has been suggested that the contribution of aerobic metabolism gains increasing importance (Spriet et al. 1989; Gaitanos et al. 1993; Bogdanis et al. 1996; Casey et al. 1996a; Trump et al. 1996). Bogdanis et al. (1996) found $\dot{V}O_2$ increased from 2.68 to 3.17 $L \cdot min^{-1}$ during successive 30 s sprints separated by a 4 min recovery. These findings may indicate an increased contribution from aerobic metabolism to the second sprint. An increased aerobic contribution could also account for the reduced post sprint blood lactate concentration. Pulmonary O_2 kinetics are known to be affected by prior exercise but research on the possible mechanisms remains equivocal (Gerbino et al. 1996; Grassi et al. 1996; MacDonald et al. 1997). As a 10 min warm up preceded the initial sprint in the present study, it is not known whether the endurance trial could have exerted an additional effect on O_2 kinetics during the second sprint. Activation of the pyruvate dehydrogenase complex (PDC) has been found to enhance O_2 kinetics (Timmons et al. 1996) and was suggested to explain the reduced lactate production found by Spriet et al. (1989).

In addition to glycogen depletion, prolonged exercise is widely held to be limited by hypoglycaemia, dehydration and hyperthermia, either separately or in combination. The minimal reliance on blood glucose during sprint exercise and the CHO supplementation during the endurance trial, preclude hypoglycaemia as a strong candidate in the aetiology of fatigue. A review of literature concluded that there is little convincing evidence that exercise induced dehydration limits maximal muscle function (see section 3.4.3). Moreover, dehydration was unlikely to have been marked in the present study due to the moderate exercise intensity and fluid intake regime adopted. Finally, and as discussed previously (section 8.4.2), any exercise induced hyperthermia was likely to augment not diminish maximal muscular power following the endurance trial.

8.4.6 Gross efficiency and peak power output

Following endurance exercise GE was significantly reduced, but no relationship was evident between ΔPPO or ΔMPO and ΔGE. The speculation that changes in GE observed during prolonged endurance exercise are driven by a reduction in force generating capacity appears unfounded. Several factors may have served to obscure a possible correlation however. The different cadences adopted during endurance exercise, GE and PPO measurement, combined with the relatively minor range of ΔPPO and ΔGE, and the inherent variability of PPO measurement could all have been significant in this respect. The similarity in the magnitude of change in ΔMPO (-5.3%) and ΔGE (-4.6%) may both be indicative of compromised muscular function and suggests further research is warranted.

8.4.7 Additional mechanisms

The main findings from this study are that PPO and MPO are significantly reduced following sustained moderate intensity exercise. These findings are in contrast to the results of Hoffman et al. (1985), Nielsen et al. (1993) and Rademaker et al. (1994a). The results of Hoffman and Nielsen were for isometric exercise which may explain the discrepancy. A later study by Rademaker et al. (1994b) did demonstrate a significant reduction in MICPO, albeit following exercise at a higher intensity (80% $\dot{V}O_{2peak}$) than the present study. Rademaker et al. (1994b) also found that MICPO was only partially restored following a 6 min recovery and suggest that different mechanisms of fatigue are

implicated following prolonged exercise in contrast to that of a shorter duration. Sahlin and Seger (1995) also found a delayed recovery in both dynamic and static leg extension torque following exercise to exhaustion at 75% $\dot{V}O_{2peak}$. Under these conditions however, it is difficult to distinguish between the effects of glycogen depletion or other sources of long term fatigue, e.g. (Edwards et al. 1977). A delayed recovery is not thought to be due to muscle damage following cycle exercise (Kuipers and Keizer, 1988), although research in this area is clearly lacking.

8.4.8 Conclusion

A sustained bout of moderate exercise appears to compromise both PPO and MPO during a 30 s sprint. Contradicting an earlier speculation, the ΔGE was not associated with the reduction in either MPO or PPO. The mechanisms for the changes in sprint performance and GE remain to be established.

Chapter 9: Changes in gross efficiency, peak power and strength: the effects of exercise, recovery, hydration and carbohydrate ingestion

9.1 Introduction

The mechanisms for the ΔGE and ΔPPO observed in the previous experiments remain to be determined. In the previous studies CHO solutions were ingested during exercise in an attempt to minimise the effects of fatigue. In the present study, fluid and CHO ingestion were manipulated in order to examine the influence of their availability on changes in muscle function and GE. Two possible common mechanisms causing the ΔGE and ΔPPO are muscle glycogen depletion and dehydration. During endurance exercise a progressive glycogen depletion occurs (Bergström and Hultman, 1967; Hermansen et al. 1967; Vollestad et al. 1984), which may necessitate an increased recruitment of additional (less efficient) muscle fibres, thereby elevating exercise $\dot{V}O_2$. At the end of exercise the resulting lower muscle glycogen availability may reduce subsequent sprint power output (Maughan and Poole, 1981; Casey et al. 1996a).

Several studies have reported no change in exercising $\dot{V}O_2$ with dehydration, (Saltin, 1964a; Hamilton et al. 1991; Dengel et al. 1992; Montain and Coyle, 1992b), but data for GE have not been previously reported. Dehydration is associated with increases in core and muscle temperature (Hargreaves et al. 1996) which have been further suggested to increase exercising $\dot{V}O_2$ (Hagberg et al. 1978; Hagan et al. 1992). The reported effects of dehydration on muscle strength and short-term performance are equivocal (see section 3.4.3). Only Walsh et al. (1994) have examined the impact of exercise induced dehydration on cycling PPO, finding no effect. Their results may have been confounded by a prior performance trial and not correcting for flywheel inertia (Lakomy, 1986). No study has examined the effect of dehydration on maximal isokinetic cycling power output (MICPO). Recently, Hargreaves et al. (1996) demonstrated that exercise induced dehydration increases the rate of glycogenolysis by 16% in a 2 h endurance trial at 67% $\dot{V}O_{2peak}$. Thus exercise induced dehydration provides a means of examining the effect of manipulating hydration, temperature and glycogen availability on muscular power and strength and GE.

Rademaker et al. (1994b) observed a delayed recovery of MICPO following sustained exercise at 80% $\dot{V}O_{2peak}$. Sahlin and Seger (1995) also report that static and dynamic leg extension torque remain depressed 30 minutes after exercising to exhaustion at 75% $\dot{V}O_{2peak}$. Rademaker et al. have suggested that fatigue of a longer lasting nature succeeds prolonged exercise, in contrast to prior exercise of 6 min or less. The time course of recovery following prolonged moderate intensity has not been examined. Furthermore, monitoring GE and maximal muscle function during recovery may provide an opportunity to dissociate the effects of temperature, dehydration and glycogen depletion, as each of these variables is thought to recover with a different time course (Saltin et al. 1968; Piehl, 1974; Maughan and Leiper, 1995; Shirreffs et al. 1996). Additionally, the rate of glycogen resynthesis is known to be markedly accelerated by CHO ingestion (Ivy et al. 1988) which could be used to augment muscle glycogen differences between conditions.

In the previous experiment it was suggested that several factors may have served to obscure a possible correlation between ΔGE and ΔPPO. These included the different cadences adopted during endurance exercise, GE and PPO measurement, and the relatively minor range of ΔPPO and ΔGE. These factors can be minimised by the use of an isokinetic cycle ergometer for testing MICPO and prolonging the period of endurance exercise to create greater changes in GE and MICPO.

In summary, this final study aimed to develop upon previous studies by examining the effects of manipulating fluid and CHO intake. This study sought to characterise the changes in GE and MICPO during and in recovery from, a prolonged bout of moderate intensity cycle ergometry (120 min, 60% $\dot{V}O_{2peak}$). Furthermore, the influence of exercise induced dehydration and CHO ingestion in recovery on GE, MICPO and maximal isometric force were investigated.

9.2 Method

9.2.1 Subjects

Eleven experienced male road-racing cyclists gave informed consent to take part in this study which had University of Brighton ethical committee approval. All subjects were competing regularly in races of 2 h or more up until, but not during the course of the study. The subjects characteristics (mean (SD)) were, mass 70.8 (7.3) kg; height 1.81 (0.07) m; $\dot{V}O_{2peak}$ 4.44 (0.55) L·min^{-1}.

9.2.2 Pre-testing and familiarisation

The relationship between $\dot{V}O_2$ and power output and the measurement of $\dot{V}O_{2peak}$ and PAPO were determined from two separate tests as described previously (see section 4.8).

Each subject performed a minimum of five familiarisation 6 s isokinetic sprints or until an asymptote in MICPO was reached, prior to taking part in the study. Subjects were also required to complete several tests of maximal isometric force.

9.2.3 Experimental procedures

9.2.3.1 Subject control

Before the study subjects were briefed on the importance of reporting to the laboratory in a similar nutritional and physical condition for all testing sessions. A dietary record and training diary were kept by each subject for two days prior to visiting the laboratory for experimental testing. Subjects were asked to repeat the same dietary and training regimens prior to their successive visit. In particular, subjects were requested to eat a meal high in CHO 3 - 5 h prior to testing and to consume 5 ml·kg body mass^{-1} of water 2 hours before reporting to the laboratory. Testing was conducted at varying times of day, but always at the same time for each subject and at intervals of 4 to 7 days apart.

9.2.3.2 Endurance exercise

Subjects undertook two endurance trials, (euhydration and dehydration) in an ordered crossover design, being alternately assigned to each condition. The endurance exercise consisted of 2 h at a power output calculated to elicit 60% $\dot{V}O_{2peak}$ initially. After 5 min and then after 30, 60, 90 and 120 min of the endurance trial, exercise was interrupted for approximately 4 min in order for the subject to be weighed and perform an isokinetic sprint. Subjects were required to pedal at 88 - 90 rev·min^{-1} throughout all testing sessions, as this was found to be the average preferred cadence from the previous two experiments. Following the endurance trial subjects recovered sitting quietly in the laboratory for a further 2 h. During the recovery period after 25 min and then at 30 min intervals subjects remounted the ergometer and resumed pedalling at the same cadence and power output for an additional 5 min.

9.2.3.3 Isokinetic testing

Maximal isokinetic cycling power output was determined as described previously (section 4.7), from 6 s sprints during the endurance exercise (at 5, 30, 60, 90 and 120 min) and in recovery after exercise (at 150, 180, 210 and 240 min). Prior to each sprint the subjects towelled dry and were weighed before positioning themselves on the isokinetic cycle ergometer. Following exactly 3 min rest subjects performed a maximal isokinetic sprint at 90 rev·min^{-1} from a stationary position, i.e. no prior pedalling. This cadence was adopted so that sprint and endurance exercise were performed at the same pedalling rate. The subjects then returned to endurance exercise or recovery. The experimental protocol is detailed in diagrammatic form below (Figure *9.1*).

9.2.3.4 Isometric testing

Maximal isometric force (MVC) was recorded using two separate procedures with the right leg except for one subject who had sustained a joint injury in this leg a year previously. The leg MVC of each subject was measured firstly by pressing on the pedal of the cycle ergometer with the crank fixed in a forward horizontal position in a similar manner to that described by Künstlinger et al. (1985) and McCartney et al. (1985). Body position was standardised on the ergometer and torque was recorded using the PMC at a sampling rate of 50 ms. This test was followed by a measurement of seated leg extension force recorded with a strain-gauge interfaced to a computer (MIE, Leeds). Testing was conducted with the knee joint positioned at a 90° angle. The MVC

measurements were made on three occasions following the isokinetic sprint at 5 and 120 min and also at the end of the recovery period (240 min). The ergometer test was always conducted first 1.5 min after the 6 s isokinetic sprint, with the seated leg extension force measured a further 1.5 min later. The MVC was recorded as the highest 1 s average force and the highest instantaneous force recorded during a maximal 5 s bout for the ergometer and seated leg extension exercise respectively.

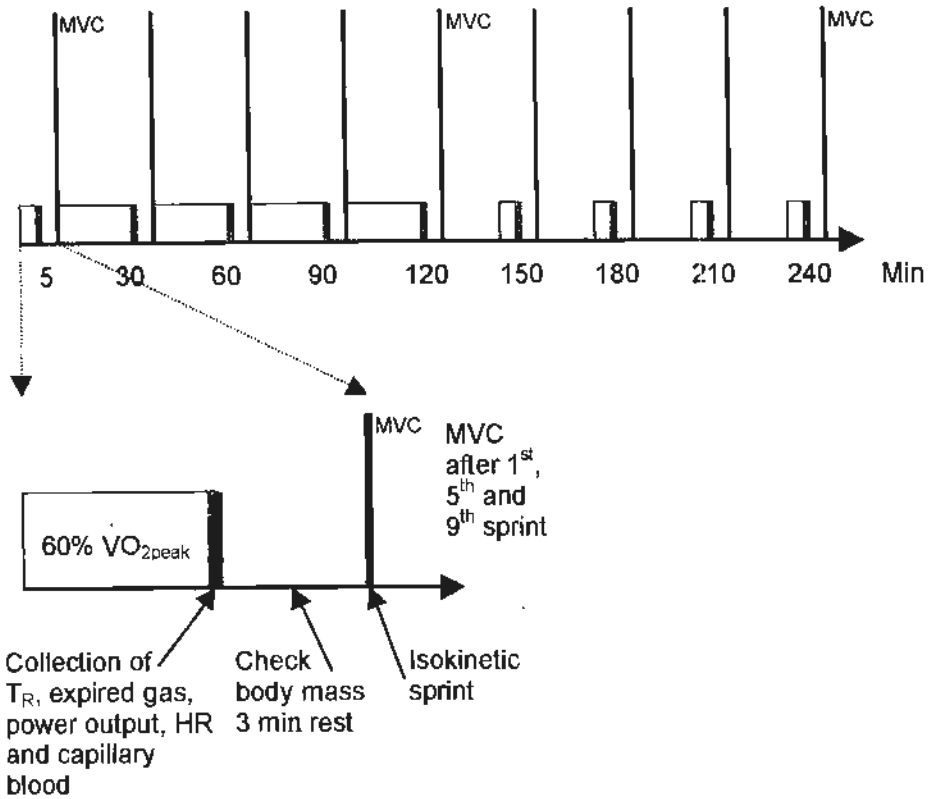


Figure 9.1 Schematic diagram of experimental protocol

9.2.3.5 Fluid and carbohydrate ingestion

During the endurance trial subjects either ingested sufficient sodium solution (50 mmol·L^{-1} Na^+) to maintain body mass throughout the exercise period (euhydration) or voluntarily minimised fluid intake (dehydration). In the euhydration trial the rate of fluid intake was adjusted if necessary following each weighing. Nine of the subjects agreed to take part in both the euhydration and dehydration endurance trials, whilst the remaining two subjects completed only the euhydration trial. Following the euhydration trial subjects were given 1.5 g CHO·kg^{-1} body mass·h^{-1} in order to maximise glycogen

resynthesis (Ivy et al. 1988). The CHO was administered in both liquid and solid form (1 g CHO·kg^{-1} body mass as a 15% glucose polymer solution and 35 g CHO in an energy bar respectively). These CHO feedings occurred immediately post euhydration endurance trial and at 60 min of recovery. Following the dehydration trial, subjects were required to rehydrate as rapidly as possible to greater than 100% prior exercise body weight with a 50 mmol·L^{-1} sodium solution. The same sodium solution was used during exercise in the euhydration trial and in recovery from the dehydration trial. The solution was formulated using sodium chloride and filtered tap water, chilled to 5 °C, and flavoured to each subject's preferred taste with a low calorie fruit cordial.

9.2.3.6 Metabolic and blood measurements

Expired gas was collected over the last 60 s of exercise prior to each isokinetic sprint during both the endurance trial and recovery periods. Power output, heart rate and rectal temperature (T_R) were also recorded at these times and a thumb-prick capillary blood sample was taken for the determination of blood glucose and lactate concentrations. Blood haemoglobin (Hb) and haematocrit (Hct) levels were measured only from the nine subjects who undertook both hydration conditions. The Hb and Hct values were determined from capillary blood samples taken at 5, 60 and 120 min of exercise and 180 and 240 min during recovery. Changes in plasma volume were calculated from Hb and Hct according to Dill and Costill (1974).

9.2.4 Data analysis

A two-way ANOVA with repeated measures (hydration condition x time) was used to compare measures in the euhydration with dehydration trials and subsequent recovery periods with data from nine subjects. Where a significant F ratio was found, differences between means were located with a Tukey post-hoc test. In order to maximise statistical power for the time based effects, analysis of GE, isokinetic power and MVC were further examined with a one-way ANOVA with repeated measures on the data from all 11 subjects for the euhydration condition. Post hoc analysis of time dependent changes was conducted with Dunnett's test as the difference between the 5 min value and subsequent time points. Statistical significance was accepted if $P < 0.05$ was found. All data are presented as mean and SD for n = 9, unless otherwise indicated.

9.3 Results

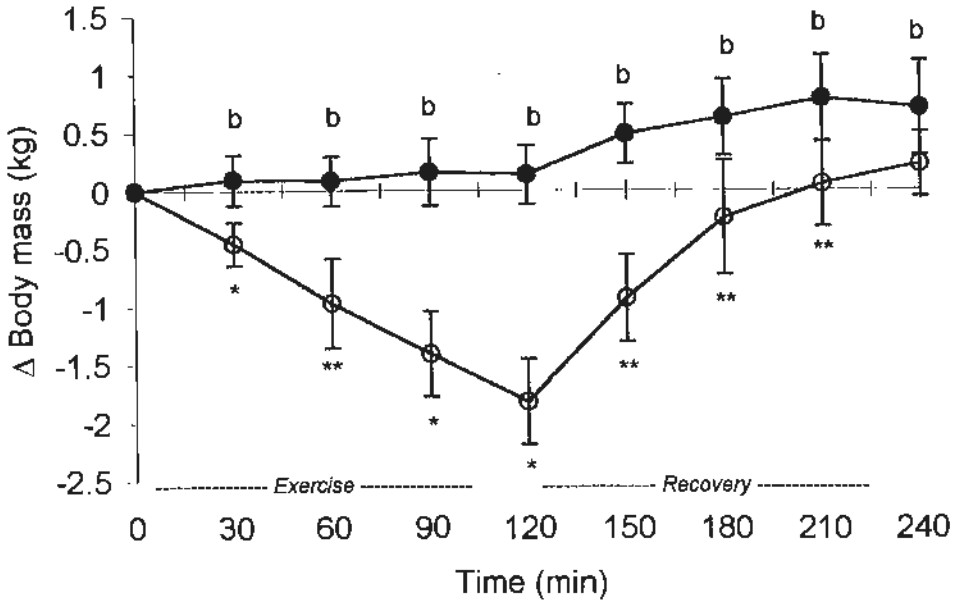


Figure 9.2 Changes in body mass during exercise and recovery periods in euhydration (filled symbols) and dehydration (open symbols) trials. Significant difference between conditions b ($P < 0.01$) and from preceding value * ($P < 0.05$) and ** ($P < 0.01$) are indicated.

Fluid ingestion during the euhydration trial successfully maintained body mass with no significant change found at any point during exercise (Figure 9.2). Dehydration caused a progressive reduction in body mass, resulting in a significant difference ($P < 0.01$) between hydration conditions at all exercise time points after the first. After 2 h of exercise, body mass was reduced by 1.8 kg (2.5%). In the recovery period following the euhydration trial body mass increased 0.6 kg ($P < 0.01$) from 120 min to 240 min due to the CHO solution ingestion. Following dehydration body mass was restored by 210 min, whereafter a non-significant increase occurred. Throughout the recovery period body mass was significantly lower with rehydration than CHO intake. This difference between conditions (0.7 kg, $P < 0.01$) persisted even at the end of the recovery periods despite body mass being fully restored after dehydration. The difference between

recovery conditions at the end of the recovery periods largely reflected the significant post-exercise increase in body mass with CHO intake.

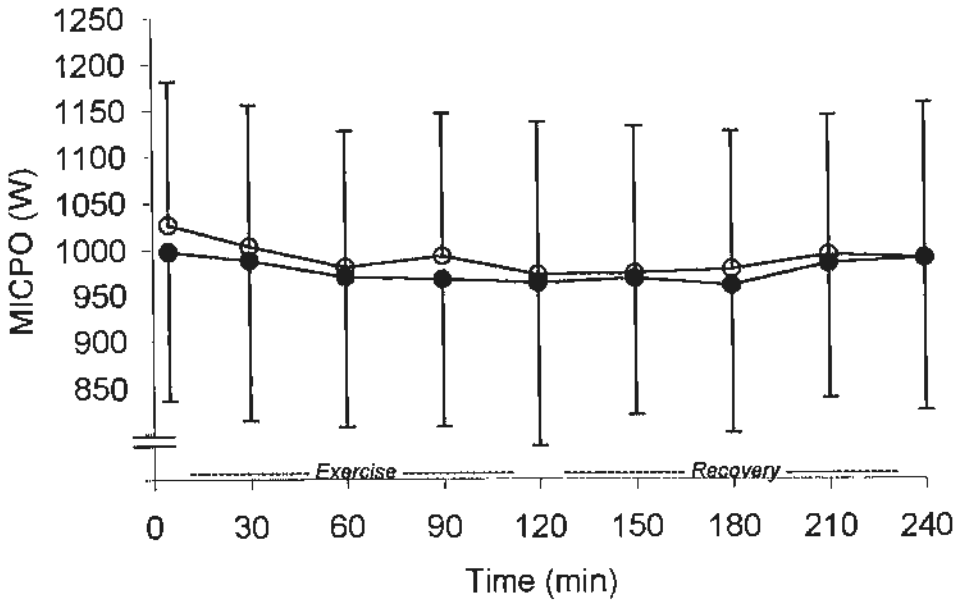


Figure 9.3 Changes in MICPO during exercise in euhydration (filled symbols) and dehydration (open symbols) and subsequent recovery. Data are presented as mean and SD, n = 9.

A significant effect of time, but not hydration nor an interaction, was found for MICPO (Figure 9.3). The significant changes over time were further examined with a one way ANOVA on data from 11 subjects during the euhydration trial (Figure 9.4). Maximal isokinetic cycling power output had declined significantly at 90 and 120 min of endurance exercise from 994 W to 957 and 956 W respectively ($P < 0.05$). After exercise MICPO recovered slowly, with no significant effect of time during the recovery period (MICPO = 974 W at 240 min). Maximal isokinetic cycling power output was found to be still significantly depressed from the initial value at 180 min (950 W, $P < 0.01$, Figure 9.4). The correlation between ΔMICPO and ΔGE was highly variable for individual data, with both significant positive and negative correlations observed (data not shown).

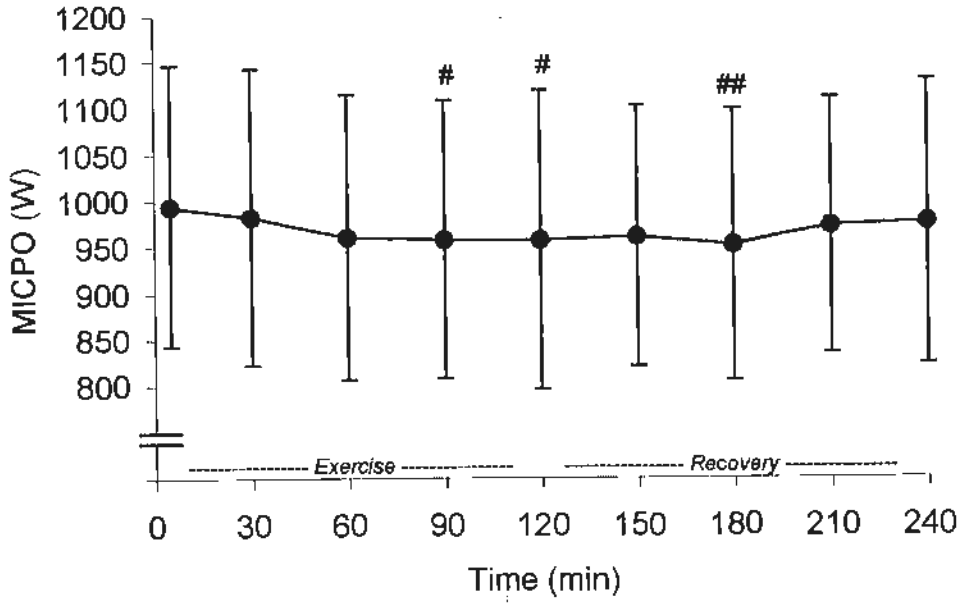


Figure 9.4 Changes over time in MICPO during euhydration exercise and subsequent recovery. Significant difference from 5 min indicated, # ($P < 0.05$), ## ($P < 0.01$). Data are presented as mean and SD, n = 11.

A significant effect of time and an interaction (hydration x time) for the exercise period was found for GE (Figure 9.5). A Tukey post hoc test revealed a significant difference between euhydration and dehydration at 90 min, GE then fell significantly from 90 to 120 min in euhydration. In the recovery period a significant main effect of time was found and there was a tendency for GE to be higher with CHO intake ($P = 0.09$). Post hoc testing found GE to have increased significantly from 120 min at 180, 210 and 240 min following euhydration ($P < 0.05$). No significant recovery of GE was detected following the dehydration trial, nor was a significant interaction between conditions found ($P = 0.77$). The time dependent effect was further examined for the euhydration exercise and recovery with the data from all 11 subjects and a one-way ANOVA with repeated measures (Figure 9.6). A Dunnett's post hoc test did not find GE to be significantly reduced from the control (5 min = 23.4%) value until 120 min of exercise (22.0%, $P < 0.01$). Gross efficiency appeared to recover in an exponential manner, but still remained significantly ($P < 0.01$) below the initial value throughout the recovery period (240 min = 22.6%).

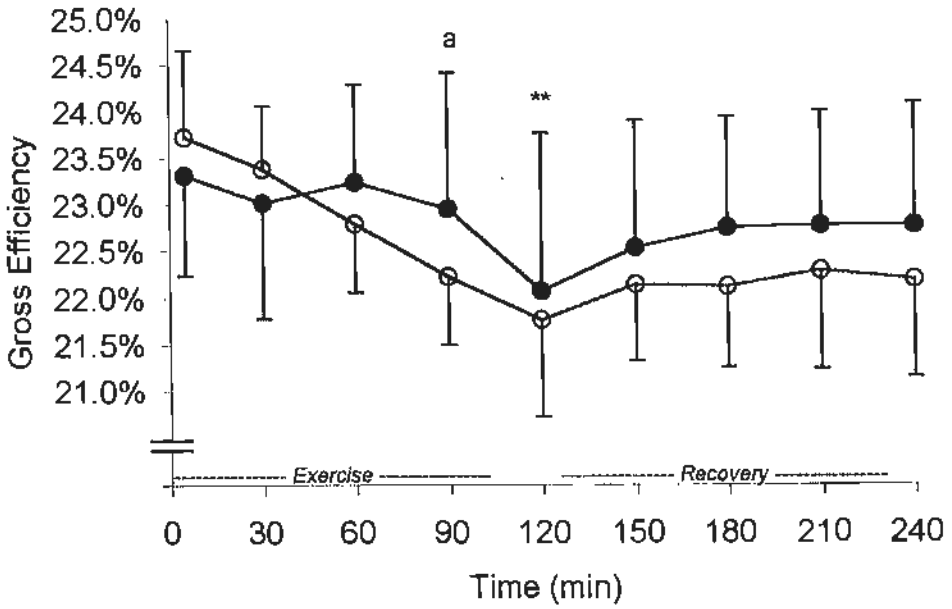


Figure 9.5 GE during exercise and recovery periods in euhydration (filled symbols) and dehydration (open symbols) trials. A significant difference between conditions is indicated by a ($P < 0.05$) and from the preceding value ** ($P < 0.01$). Data are mea.. and SD, n = 9.

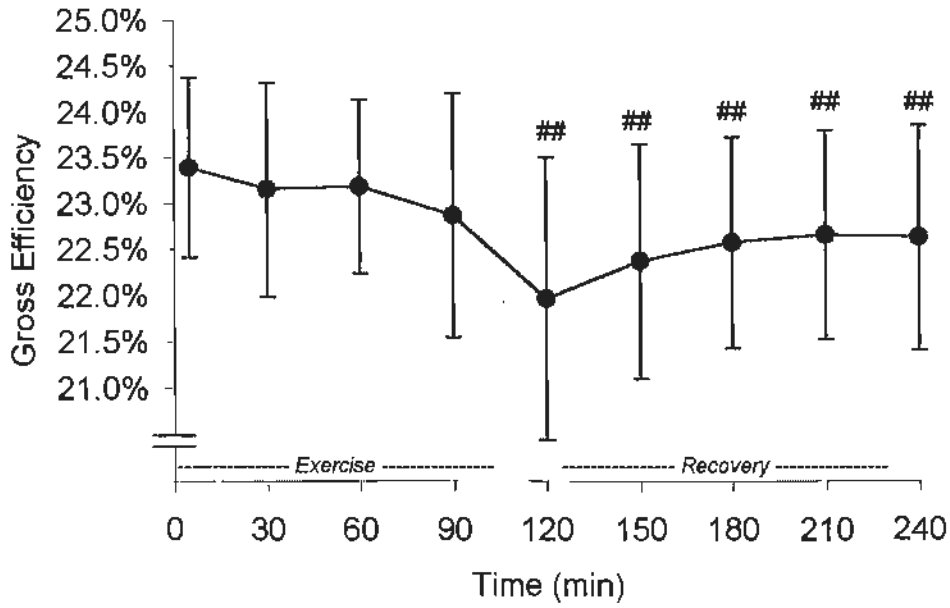


Figure 9.6 Changes in GE over time during exercise in euhydration and subsequent recovery. ## indicates significantly lower than 5 min ($P < 0.01$), n = 11.

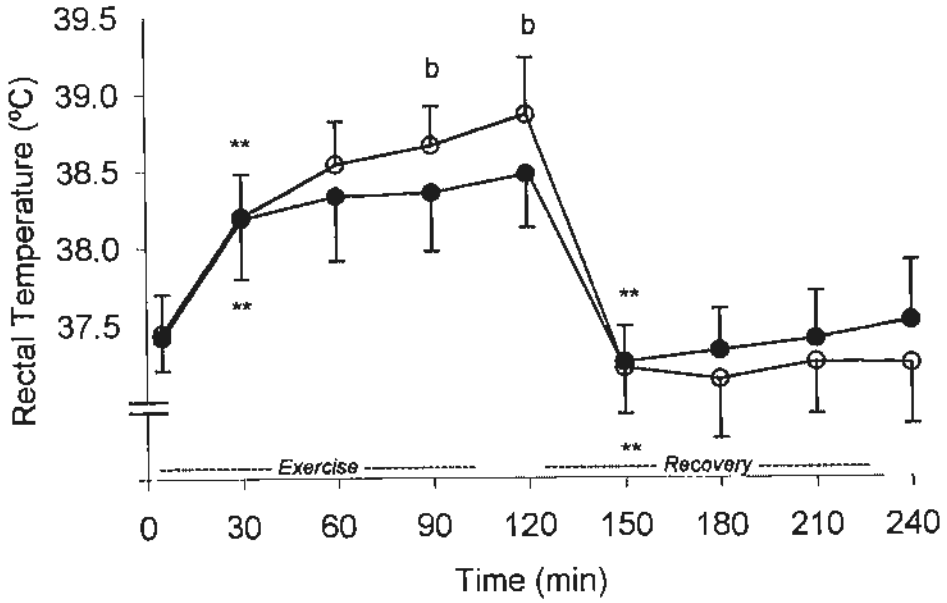


Figure 9.7 T_R during exercise and recovery periods in euhydration (filled symbols) and dehydration (open symbols) trials. Significant differences between conditions b ($P < 0.01$) and from preceding value ** ($P < 0.01$) are indicated.

Rectal temperature increased significantly ($P < 0.01$, Figure 9.7) in both hydration conditions from 37.4 °C at 5 min to 38.2 °C at 30 min. Thereafter, T_R remained similar throughout exercise with euhydration. By contrast, T_R rose continually during exercise in the dehydration trial, such that a significant difference ($P < 0.01$) was observed between hydration conditions at 90 min. By 120 min the difference had increased further to 38.5 vs. 38.9 °C for euhydration and dehydration respectively ($P < 0.01$). In the recovery period T_R was measured following each 5 min warm up at 60% $\dot{V}O_{2peak}$. Following the endurance trial, T_R fell significantly from 120 min to approximately 37.3 °C at 150 min in both conditions with no further significant differences found during the recovery period either within or between conditions.

Heart rate increased from the 5 min value of 143 beats·min^{-1} to 151 beats·min^{-1} at 90 min ($P < 0.05$) and 158 beats·min^{-1} at 120 min ($P < 0.01$) during exercise in the euhydration condition (Figure 9.8). In the first 30 min of the dehydration trial HR had increased by 11 beats·min^{-1} ($P < 0.01$) from 140 to 151 beats·min^{-1}. A continued HR drift was evident with a further significant increase occurring during the final 30 min of exercise from 161 to 170 beats·min^{-1} ($P < 0.05$). By the end of the dehydration trial, the increase in HR was double that of the euhydration trial (31 beats·min^{-1}, $P < 0.01$). In the

recovery period heart rate was measured during the 5 min warm up exercise at 60% $\dot{V}O_{2peak}$. Following the euhydration trial HR fell 5 beats·min^{-1} from 120 to 150 min ($P > 0.05$). Thereafter HR increased progressively and was 9 beats·min^{-1} higher than 150 min at the end of the recovery period ($P < 0.01$). Rehydration resulted in HR falling by 12 beats·min^{-1} ($P < 0.01$) from 120 min to 150 min after which no significant change was found.

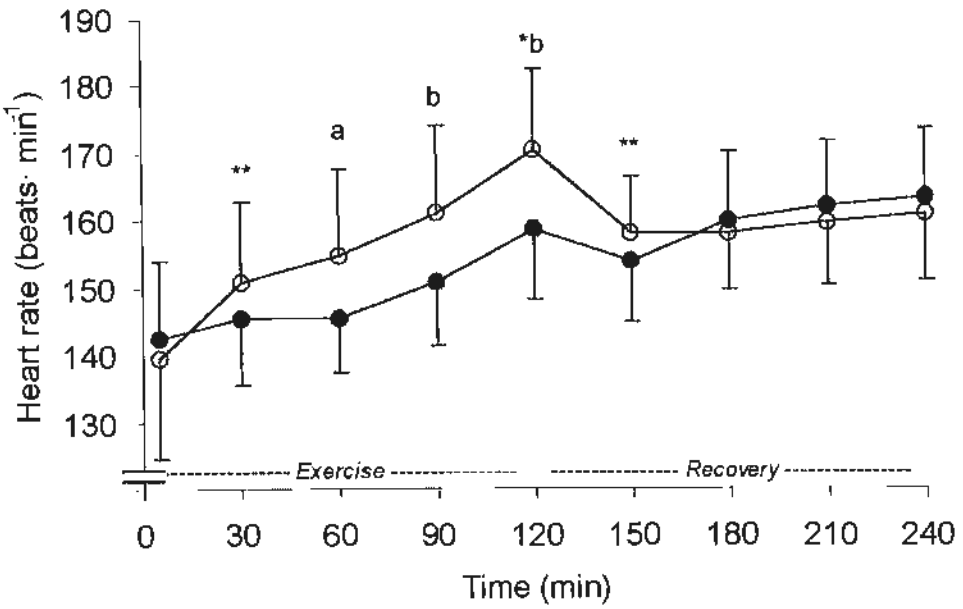


Figure 9.8 Heart rate during exercise and recovery periods in euhydration (filled symbols) and dehydration (open symbols) trials. Significant differences between conditions a ($P < 0.05$), b ($P < 0.01$) and from preceding value * ($P < 0.05$) and ** ($P < 0.01$) are indicated.

The RER fell progressively during exercise with a main effect of time, but not hydration nor interaction being found (Figure 9.9). The RER at 120 min of exercise (0.91) was significantly lower than the RER at 5 min (0.98) for the euhydration trial only ($P < 0.05$). During the recovery period and following CHO ingestion, RER measured at the end of warm up exercise, rose progressively from 180 min and was significantly higher than 150 min (0.88) at 210 (0.92, $P < 0.05$) and 240 min (0.93, $P < 0.01$). After dehydration RER continued to fall and was significantly lower than the end of exercise at 180 (0.87), 210 (0.85) and 240 min (0.84, $P < 0.01$). Consequently, significant main effects of time and condition and an interaction were found, with differences in RER between conditions being evident at 180 ($P < 0.05$), 210 and 240 min ($P < 0.01$).

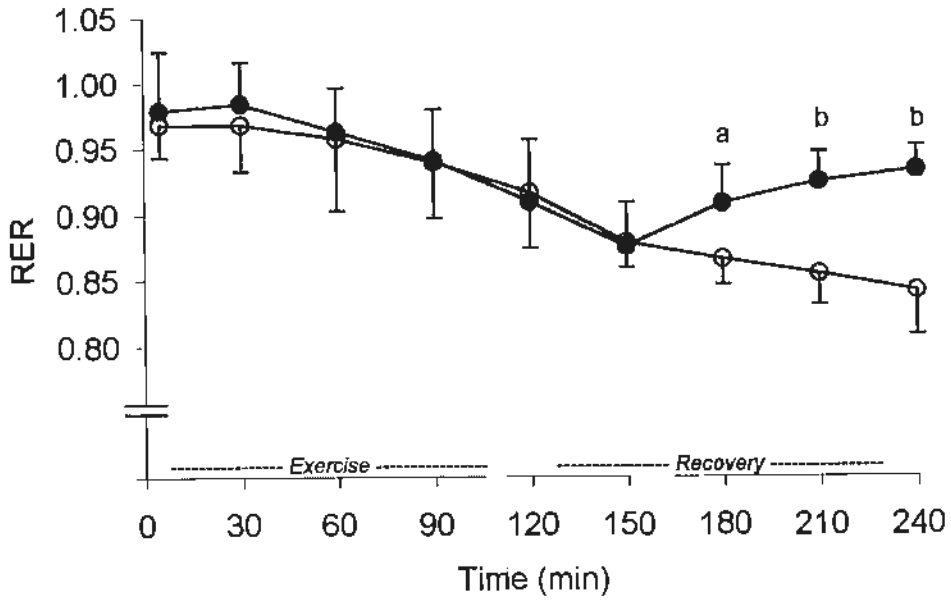


Figure 9.9 RER during exercise and recovery periods in euhydration (filled symbols) and dehydration (open symbols) trials. Significant differences between conditions during recovery a ($P < 0.05$), b ($P < 0.01$) are indicated.

A similar pattern to RER was found for estimated rates of CHO and fat oxidation (Figure 9.10 and Figure 9.11). The endurance exercise resulted in a main effect of time for both CHO and fat oxidation, but there was no effect of hydration, nor a hydration x time interaction. Fat oxidation rose progressively throughout exercise (5 and 30 min < 120 min, $P < 0.01$), whilst CHO oxidation declined inversely (5, 30 and 60 min > 12[illegible] min, $P < 0.01$). Significant main effects of hydration, time and interaction (hydration x time) were found for both CHO and fat oxidation during the recovery period. Fat oxidation continued to increase ($P < 0.01$) in both conditions from 120 to 150 min, whilst CHO oxidation fell concomitantly ($P < 0.01$). Beyond 150 min fat utilisation continued to increase throughout the rehydration recovery period (150 and 180 min < 240 min, $P < 0.01$). After 150 min following the euhydration trial a significant reductio[illegible] in fat utilisation occurred (150 and 180 > 240 min, $P < 0.01$, and $P < 0.05$ respectively). Estimated CHO oxidation responded in a similar, but inverse manner, continuing to fall in the rehydration trial (150 min > 240 min, $P < 0.01$) whilst rising sharply after 150 min with CHO ingestion (150 min < 210 and 240 min, $P < 0.01$).

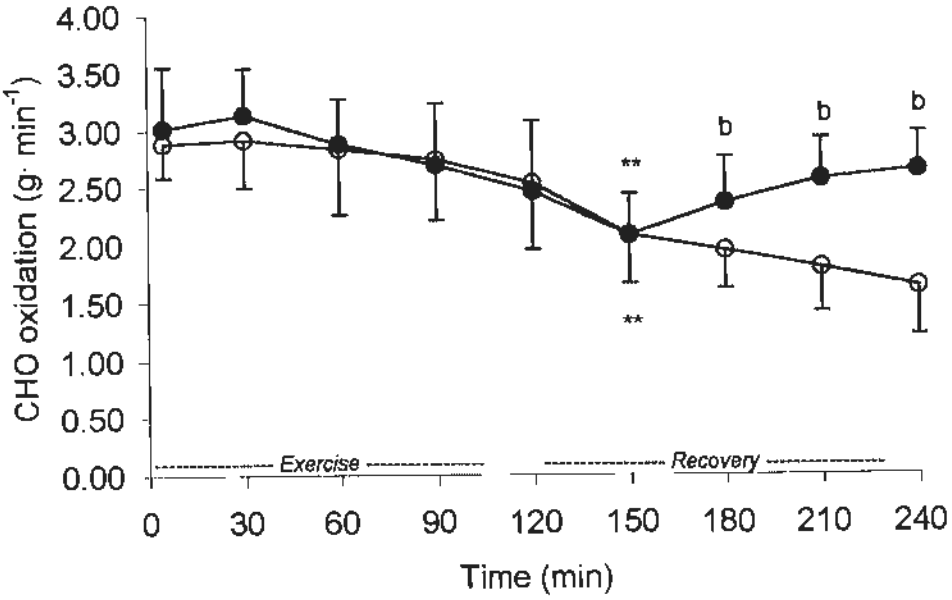


Figure 9.10 Changes in estimated CHO oxidation during exercise and recovery in euhydration (filled symbols) and dehydration (open symbols). Significant differences between conditions b ($P < 0.01$) and preceding value ** ($P < 0.01$) are indicated.

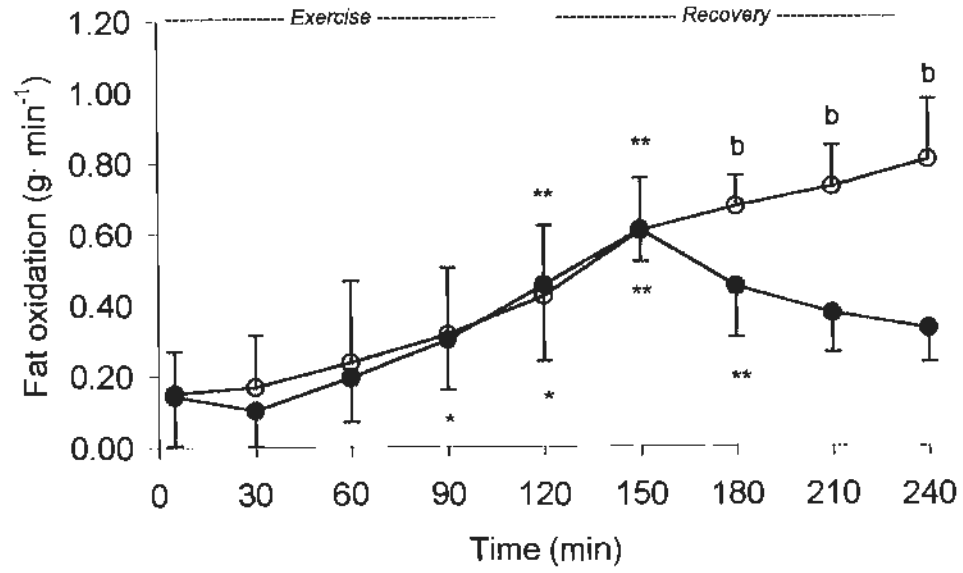


Figure 9.11 Changes in estimated fat oxidation during exercise and recovery in euhydration (filled symbols) and dehydration (open symbols). Significant differences between conditions b ($P < 0.01$) and preceding value * ($P < 0.05$) and ** ($P < 0.01$) are indicated.

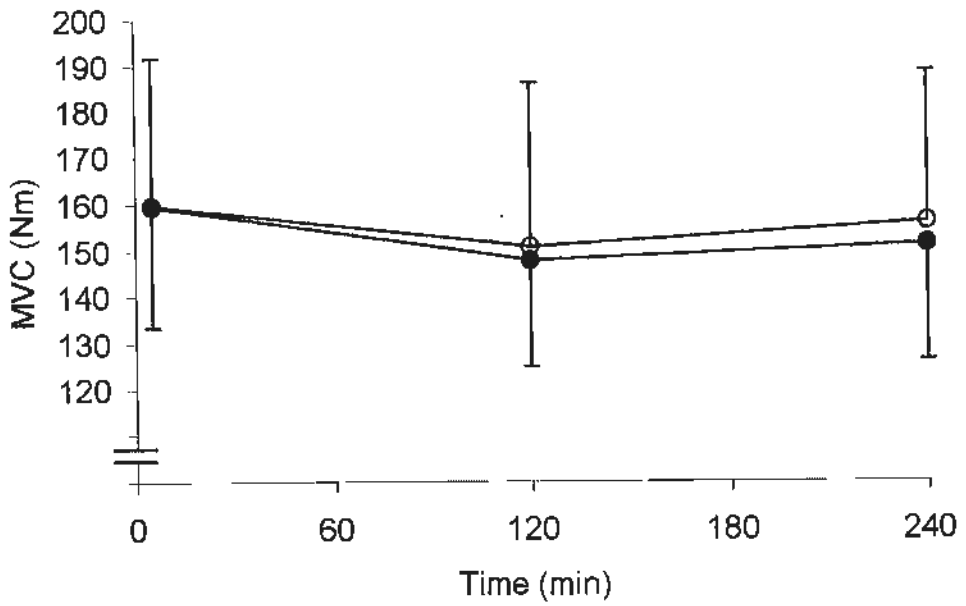


Figure 9.12 Changes in ergometer MVC following exercise and recovery in euhydration (filled symbols) and dehydration (open symbols) trials. Data are mean and SD, n = 9.

A two-way ANOVA did not reveal a significant effect of hydration nor an interaction for maximal isometric force measured with either the ergometer or seated leg extension testing protocol (data for the ergometer test are presented in Figure 9.12). A one-way ANOVA on data for all 11 subjects in the euhydration trial found the effect of time on maximal isometric force measured on the cycle ergometer approached significance however, ($P = 0.06$). A Dunnett's post-hoc test revealed a significant reduction in post exercise MVC from 154.4 (26.3) Nm to 144.4 (22.6) Nm ($P < 0.05$), which was partially restored to 148.2 (24.8) Nm ($P > 0.05$) after 2 h of recovery (Figure 9.13). No effect of time was found for the seated leg extension MVC ($P > 0.1$)

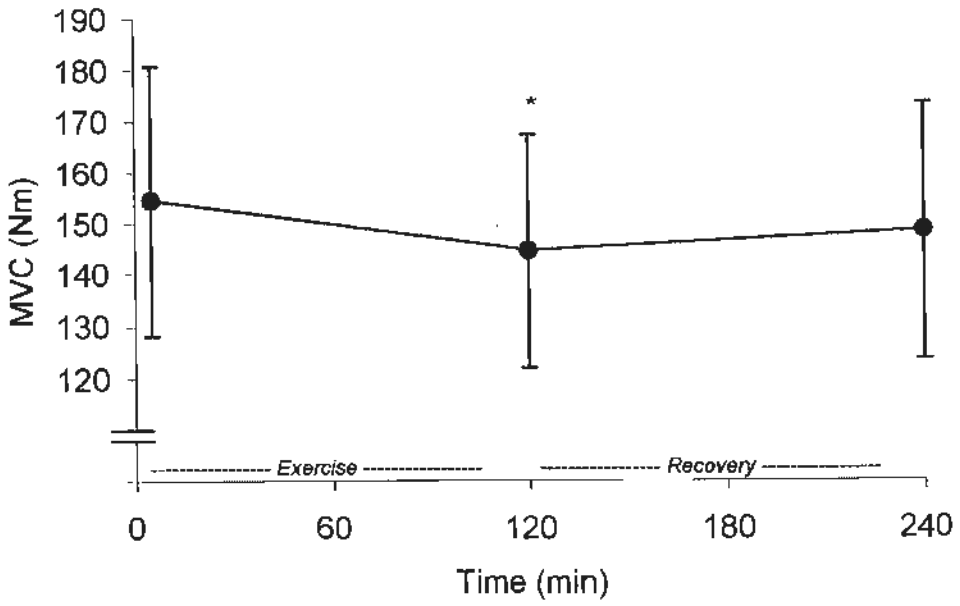


Figure 9.13 MVC measured on the ergometer before and after 2 h of exercise and recovery for euhydration. * $P < 0.05$ from 5 min. Data are mean and SD, n = 11.

A significant main effect of time only was found for blood glucose during exercise, falling from 4 mmol·L^{-1} to approximately 3.0 mmol·L^{-1}. Following CHO ingestion a significant sustained elevation in blood glucose (≈ 5.0 mmol·L^{-1}) was apparent from 15 min until the end of the recovery period. During the rehydration period blood glucose remained low, but stable at approximately 3.2 mmol·L^{-1}. Technical difficulties resulted in complete data being available for only 8 subjects during exercise and 6 subjects during recovery.

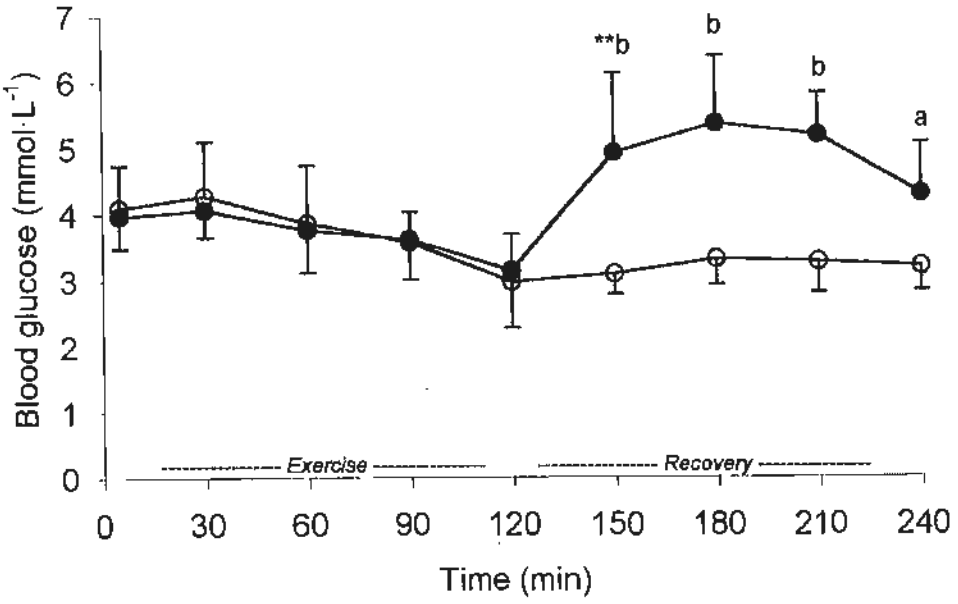


Figure 9.14 Blood glucose during exercise and recovery in euhydration (filled symbols) and dehydration (open symbols). Significant differences between conditions are indicated a ($P < 0.05$), b $P < 0.01$). Data are mean and SD, n = 8 for exercise and n = 6 for recovery.

The data for blood lactate response were not found to be normally distributed at several time points during the dehydration trial (Shapiro-Wilks test, $P < 0.05$). A two-way ANOVA does not require a normal distribution of raw scores however, but normally distributed residuals for the ANOVA model (Altman, 1991, pp. 330). Furthermore Howell, (1997, pp. 321) notes that the ANOVA is a very robust procedure especially against violations of the assumption of normality. As the residuals for the ANOVA model demonstrated a normal distribution, this method of analysis was preferred to the non-parametric Friedman test. No main effects were found for the blood lactate response during exercise (Figure 9.15). In recovery a significant main effect of time and interaction (hydration x time) was found. The rehydration recovery trial resulted in significant drop in blood lactate from 120 min to 240 min. During CHO ingestion recovery blood lactate rose significantly from 120 min to 150 min, dropped significantly from 150 min to 180 min and dropped again from 210 to 240 min. No difference between conditions was found. Technical difficulties resulted in complete data being available for 8 subjects for the recovery period.

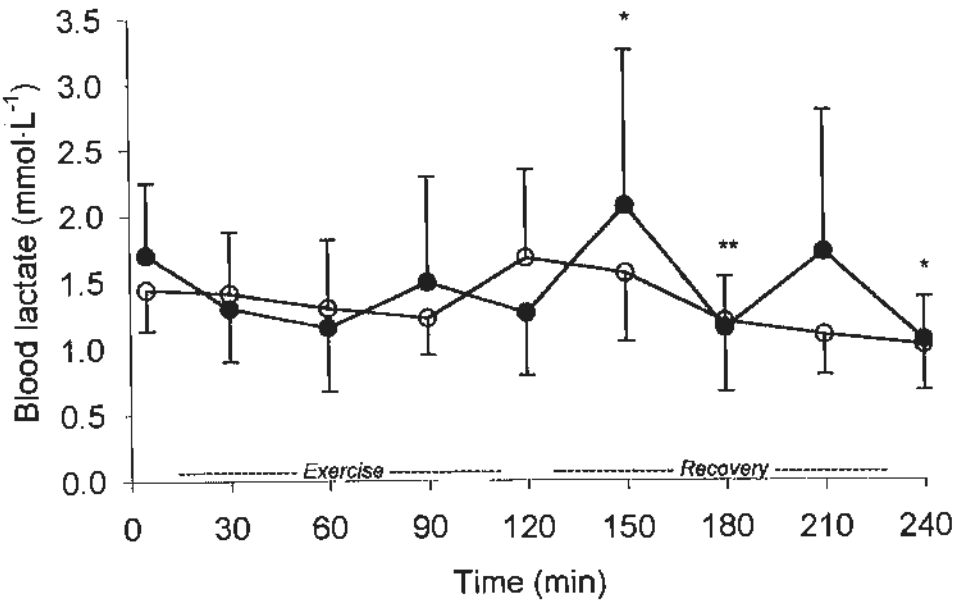


Figure 9.15 Blood lactate during exercise and recovery in euhydration (filled symbols) and dehydration (open symbols). Significant differences from preceding values are indicated * ($P < 0.05$) ** ($P < 0.01$). Data are mean and SD, n = 9 for exercise and n = 8 for recovery.

A paired t-test revealed no significant difference in initial values of Hb or Hct between hydration conditions ($P > 0.05$). No significant changes in calculated plasma volume were found (Figure 9.16), although there was a tendency for a main effect of time ($P = 0.07$). Technical problems resulted in data for 60 and 180 min time points being complete for only 5 subjects. Accordingly, statistical analysis was conducted on data for 0, 120 and 240 min. Data at 60 and 180 min are presented for comparison only (n = 5).

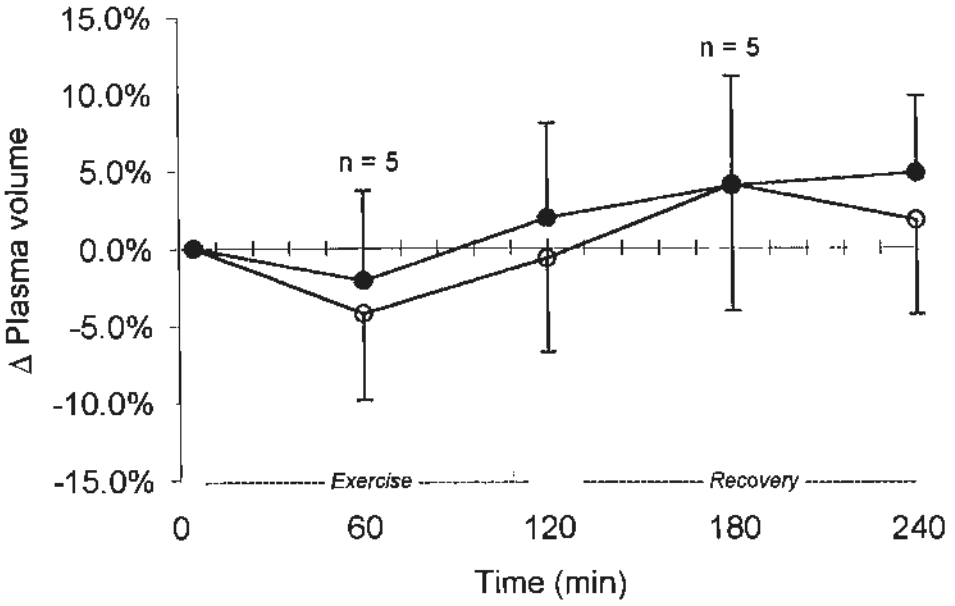


Figure 9.16 Calculated changes in plasma volume in exercise and recovery for euhydration (filled symbols) and dehydration (open symbols) conditions. Data are mean and SD, n = 9 except where indicated.

9.4 Discussion

The present study was designed to examine the effects on GE and MICPO of manipulating hydration during exercise and CHO status during recovery. The main findings of this study were that 120 min of endurance exercise at 61 (SD 3.6)% and 59 (SD 4.4)% $\dot{V}O_{2peak}$ for euhydration and dehydration respectively results in a significant reduction in both GE (Δ = -6.1%) and MICPO (Δ = -3.8%) and that both GE and MICPO recover slowly. The post exercise changes in GE and MICPO were also accompanied by a significant reduction in maximal isometric force (-6.5%), which had been partly restored following the 2 h recovery period ($P > 0.05$). The progressive reduction in MICPO during exercise is similar for dynamic muscle function to the findings of Fulco et al. (1995, 1996), who claim to have been the first to demonstrate a serial decline in MVC resulting from sustained dynamic leg extension exercise.

9.4.1 Dehydration

The significant and progressive increases in T_R, and HR and the reduction in body mass during exercise in the dehydration trial are all in agreement with previous studies on the effects of exercise induced dehydration (Saltin, 1964a; Greenleaf and Castle, 1971; Hamilton et al. 1991; Montain and Coyle, 1992b; Hargreaves et al. 1996). The change in plasma volume was not significantly different between conditions and is similar to the recent findings of Robinson et al. (1995) and Fallowfield et al. (1996), but not Hargreaves et al. (1996). Coyle and Hamilton (1990) suggest from a review literature that plasma volume is well defended during prolonged exercise in neutral environments after the initial adjustment to exercise. Also, Fallowfield et al. (1996) have remarked that the high variance in the measurement of plasma volume can obscure a difference between conditions despite careful experimental control.

9.4.2 Rehydration

Following the dehydration trial subjects were required to restore body mass to greater than 100% of initial value as rapidly as possible by consuming a 50 $mmol \cdot L^{-1}$ sodium solution. Restoration of fluid balance generally required 90 min of recovery (210 min - Figure 9.2). The composition of the rehydration solution has been demonstrated to be important in restoring and maintaining fluid and sodium balance (Nose et al. 1988; González-Alonso et al. 1992; Maughan and Leiper, 1995; Shirreffs et al. 1996). The studies of Nose et al. (1988) and González-Alonso et al. (1992) found rehydration with a sodium electrolyte beverage to be more effective in restoring total body water and blood volume than water alone. Plain water intake was associated with an increased urine formation and lower fluid retention. Maughan and Leiper (1995) have suggested that a 50 $mmol \cdot L^{-1}$ sodium solution, similar to that adopted in the present study, is effective in restoring both fluid and sodium balance. Furthermore, Shirreffs et al. (1996) have reported that the sodium content and the ingested fluid volume interact to determine the efficacy of the rehydration solution. These authors found that a fluid intake representing 150% body mass loss and a high sodium concentration (61 $mmol \cdot L^{-1}$) was required to maintain fluid balance after 2 h recovery. In the present study, whilst all subjects consumed at least 100% of the volume required to restore body mass. None of the subjects could achieve the fluid intake of 150% recommended by

Shirreffs et al. particularly with the periodic exercise in recovery. The consistent plasma volume (Figure 9.16) and heart rate (Figure 9.8) responses in recovery for the euhydration and dehydration trials suggests that fluid balance was not meaningfully different over the last hour of the recovery period.

9.4.3 CHO ingestion during recovery

The effects of CHO intake in recovery on the restoration of GE and MICPO were examined by selective CHO feeding. Following the euhydration exercise subjects consumed 1.5 g CHO·kg body mass^{-1}·h^{-1}, whilst after the dehydration trial subjects consumed saline only. The CHO ingestion was associated with a sustained elevation of blood glucose (Figure 9.14) followed by an increase in RER (Figure 9.9) and estimated CHO oxidation (Figure 9.10).

Glycogen synthase activity and corresponding rates of glycogen synthesis are known to be markedly increased by exercise and the extent of glycogen depletion (Kochan et al. 1979; Zachwieja et al. 1991). Glycogen resynthesis may be complete within 24 h, depending upon the amount and timing of CHO intake post exercise (Piehl, 1974; Blom et al. 1987; Keizer et al. 1987; Ivy et al. 1988; Brouns et al. 1989). Ivy et al. (1988) found the rate of glycogen resynthesis to be three times greater when CHO was consumed immediately as opposed to 2 h post exercise. Carbohydrate intake in both conditions consisted of 2 g CHO·kg body mass^{-1}·2 h^{-1} of a 25% glucose polymer solution. Muscle glycogen resynthesis rates were 33 vs. 11 mmol·kg^{-1}·h^{-1} for the immediate and delayed CHO intake respectively and probably occurred in both Type I and II fibres (Piehl, 1974; Kuipers et al. 1987). In the present study, CHO was administered in both liquid and solid form to improve palatability. The form of CHO ingested has not been found to influence the rate of glycogen resynthesis (Keizer et al. 1987). Consequently, the ingestion of CHO in recovery following euhydration, but not dehydration, is expected to have resulted in considerable differences in muscle glycogen content during the recovery period.

9.4.4 Substrate utilisation

Dehydration did not appear to influence substrate utilisation as reflected by the similarity between exercising hydration conditions in RER, blood lactate and calculated CHO oxidation. This finding is in contrast to some previous studies (Fallowfield et al. 1996; Hargreaves et al. 1996; González-Alonso et al. 1997), but not all (Dengel et al. 1992; Walsh et al. 1994). Accordingly, there was little evidence that fluid restriction during exercise increased the rate of muscle glycogen utilisation as found by Hargreaves et al. (1996). A dehydration induced alteration in substrate utilisation may have been less pronounced due to the lower exercise intensity and associated heat stress of the present study in comparison with those studies that have reported an effect. A small effect size and the inherent variability of RER response may also have made detection difficult. These points are reinforced by closer examination of the data of Hargreaves et al. (1996). These researchers found RER was significantly higher only at 60 and 120 min of exercise (0.93 vs. 0.91 and 0.89 vs. 0.86), despite all five subjects increasing glycogen utilisation under dehydration as measured by muscle biopsy. Blood lactate was significantly elevated surprisingly at 30 min, but not again until 120 min. Furthermore, it can be calculated that the 16% increase in estimated total CHO oxidation found by Hargreaves et al. requires only a 3.5% change in RER (i.e. average RER 0.01 lower).

An increased rate of glycogen utilisation has been demonstrated to result from a higher core temperature (Febbraio et al. 1996) and muscle temperature (Febbraio et al. 1996b) and increased circulating catecholamines (Febbraio et al. 1998). These effects are generally associated with exercise induced dehydration (Greenleaf and Castle, 1971; MacDougall et al. 1974; Hamilton et al. 1991; Montain and Coyle, 1992b; Walsh et al. 1994; González-Alonso et al. 1995; Robinson et al. 1995; Fallowfield et al. 1996; González-Alonso et al. 1997). The higher T_R of the present study is in agreement with these observations and in combination with the elevated HR is consistent with increased T_m and circulating levels of catecholamines. It is possible that dehydration in the present study increased the rate of muscle glycogen utilisation, but changes in corresponding whole body indices of CHO utilisation have been obscured.

An unexpected observation in recovery following CHO ingestion was that RER and estimated CHO oxidation did not increase rapidly. A significant elevation in blood glucose ($P < 0.01$) at 30 min of recovery (150 min) failed to prevent a significant decline in calculated CHO oxidation ($P < 0.01$) until the expired gas collection at 1 h of recovery (180 min). It appears that substrate utilisation was not immediately altered by a marked elevation in blood glucose, even after prolonged exercise when blood glucose was low. These results are similar to those of Coggan and Coyle (1987, 1989) who surprisingly do not comment on this aspect of their data. Both studies of Coggan and Coyle examined the effect of consuming 200 g of CHO late in exercise on subsequent time to exhaustion. In comparison with a placebo beverage, CHO ingestion significantly increased plasma glucose, RER and subsequent time to exhaustion. Close examination of their data reveals that RER was not elevated until approximately 30 min post CHO ingestion. These findings contradict the suggestion of Hawley et al. (1994) that exogenous glucose oxidation is regulated by blood glucose concentration. Other possible explanations for the delay in increasing CHO oxidation are that the rate of muscle glucose uptake is limited; blood glucose cannot be oxidised directly on entry into muscle cell; or the factors that regulate substrate utilisation do not respond rapidly to changes in CHO availability. It is speculated that in the present study, exogenous CHO may have influenced substrate utilisation only after being incorporated into muscle glycogen due to the intermittent nature of the exercise in recovery.

9.4.5 Gross Efficiency

Endurance exercise in euhydration resulted in a significant decline in GE, but surprisingly this change occurred late in exercise rather than progressively as in previous studies and the dehydration trial. The reason for this unusual response is not known. By the end of exercise the mean ΔGE was -6.1%. Recovery of GE appeared to be two phased in response. There was an initial significant and seemingly exponential restoration of GE which reached a plateau by 1.5 h and remained unchanged over the last 30 min. Throughout the recovery period GE remained significantly below the control value ($P < 0.01$). The time for complete restoration of GE is consequently difficult to estimate, but may have a half time of several hours or more. The plateau in GE recovery was evident despite the intake of 1.5 g CHO·kg body mass^{-1}·h^{-1}.

A 55% greater decline in GE was associated with dehydration of 2.5% body mass, as indicated by the significant interaction (hydration x time). This finding is inconsistent with the majority of previous studies, which have observed no effect of dehydration on $\dot{V}O_2$ (Saltin, 1964a, 1964b; Hamilton et al. 1991; Dengel et al. 1992; Montain and Coyle, 1992a, 1992b; Hargreaves et al. 1996; González-Alonso et al. 1995; González-Alonso et al. 1997). It is possible that the unusual pattern of decline in GE in the euhydration trial may have been responsible. Following dehydration and in contrast to CHO ingestion, GE did not recover significantly ($P > 0.05$). As no interaction was found during recovery following dehydration ($P = 0.77$), it is suggested that the tendency for a higher GE with CHO intake ($P = 0.09$) may have been an effect of the preceding exercise rather than the recovery manipulation. It appears that the mechanisms responsible for the ΔGE are characterised as changing progressively during endurance exercise and recovering slowly afterwards. Furthermore, proposed mechanisms for the ΔGE may be influenced by dehydration, but are not altered markedly by increased CHO availability in recovery.

Muscle glycogen is an obvious candidate for the ΔGE, as it is depleted progressively during exercise (Hermansen et al. 1967; Vollestad et al. 1984; Coyle et al. 1986; Jansson and Kaijser, 1987), and resynthesised over a period of many hours Piehl, 1974; Keizer et al. 1987; Kuipers et al. 1987; Ivy et al. 1988; Brouns et al. 1989b). The finding of a significant interaction during exercise (hydration x time) for ΔGE is in agreement with a glycogen depletion mediated hypothesis. As discussed previously, Hargreaves et al. (1996) have found glycogen utilisation to be 16% greater during exercise induced dehydration in a 2 h trial at 67% $\dot{V}O_{2peak}$. The recovery of GE is not consistent with the anticipated changes in muscle glycogen concentration. The different CHO ingestion regimens following dehydration and rehydration were likely to create large differences in muscle glycogen content (Ivy et al. 1988), yet no significant difference between conditions for GE was observed. Furthermore, the recovery of GE appeared to plateau 1.5 h into recovery, in contrast to expected rates of glycogen resynthesis (Piehl, 1974; Blom et al. 1987; Ivy et al. 1988). Comparison of the ΔGE between hydration conditions implicates both glycogen dependent and independent processes as mechanisms.

The cause of the ΔGE and particularly the greater change observed with dehydration cannot easily be identified. The reduction in total body water during the dehydration trial is not thought to be directly responsible for the greater ΔGE, as rehydration during recovery did not restore GE. The dissociation in the pattern of recovery between ΔGE and rectal temperature is in agreement with the findings reported in Chapter 7 and reinforces the suggestion that changes in body temperature do not directly influence GE. Despite increasing glycogen utilisation, dehydration has not been found to affect muscle ATP, and PCr concentration (Hargreaves et al. 1996) or change circulating electrolyte concentrations (Walsh et al. 1994; Robinson et al. 1995; Fallowfield et al. 1996; González-Alonso et al. 1997) compared with lower levels of dehydration or maintaining fluid balance. Although two studies report elevations in serum sodium, this is may be caused by an increase in serum osmolality (Montain and Coyle, 1992b; Walsh et al. 1994) and may not be meaningful in this context. Catecholamine response has been found to be higher with dehydration (González-Alonso et al. 1995; Hargreaves et al. 1996; González-Alonso et al. 1997) and are thought to be mediated by increased core and muscle temperatures (Febbraio et al. 1994; González-Alonso et al. 1995; Hargreaves et al. 1996; Fallowfield et al. 1996; Mora-Rodríguez et al. 1996; González-Alonso et al. 1997. The equivocal results on the influence of catecholamines on exercising $\dot{V}O_2$ have been reviewed previously (section 2.7.3) and at normal laboratory temperatures are not thought to be significant.

9.4.6 Maximal Isokinetic Cycling Power Output

Moderate intensity exercise resulted in a progressive reduction in MICPO, which led to a drop of 38 W after 2 h (-3.8%, $P < 0.05$). The relatively small reduction in MICPO is in contrast to the 301 W (17%) decrement calculated by Rademaker et al. (1994b), and also the 25% decrement in isokinetic leg extension torque reported by Sahlin and Seger (1995). The higher exercise intensity and absence of a recovery period following exercise probably account for these divergent results. It is interesting to note that the studies of Rademaker et al. and Sahlin and Seger, report greater levels of fatigue, but more rapid rates of recovery in comparison with the present study. This observation is probably due to the greater metabolic stress of the higher exercise intensities and is emphasised by the end exercise blood lactate concentrations of 4.9 and 2.8 $mmol \cdot L^{-1}$ found by Rademaker et al. and Sahlin and Seger respectively, in comparison with

1.3 $mmol \cdot L^{-1}$ in the present study. In the present study, a 3 min recovery period prior to the determination of MICPO was allowed. In contrast, Rademaker et al. tested MICPO immediately on completion of exercise, whilst Sahlin and Seger cuffed their subjects' legs to occlude blood flow between endurance exercise and muscle function testing. Any acute reduction in PCr associated with endurance exercise (Norman et al. 1987; Broberg and Sahlin, 1989; Green et al. 1989; Ball-Burnett et al. 1991; Green et al. 1991) and its influence on MICPO was likely to have been largely reversed following the rest period in the present study (Hultman et al. 1967; Harris et al. 1976).

In parallel with the changes in GE, and despite the minor degree of fatigue, MICPO was found to recover only slowly. Following 60 min of recovery with CHO intake MICPO was still significantly below initial values ($P < 0.01$). Consistent with the findings of Jacobs, (1980) and Walsh et al. (1994), dehydration did not further reduce peak power output. Moreover, in agreement with those studies that find CHO availability does not influence peak power output (Symons and Jacobs, 1989; Vandenberghe et al. 1995; Hargreaves et al. 1997), no difference in the restoration of MICPO was found during recovery with CHO ingestion compared to that for rehydration (saline) only.

The mechanisms resulting in the ΔMICPO remain to be clearly established. Neither exercise induced dehydration, nor CHO ingestion during recovery appear to have altered the fatigue observed. It is possible that a greater level of fatigue resulting from exercise induced dehydration was balanced by a concomitant increase in muscle temperature and the resulting enhanced power output (Sargeant, 1983; Sargeant, 1987; Rademaker et al. 1994b). Any fatigue masked in this manner was reversed within 30 min however, as recovery data show both T_R and MICPO were comparable between conditions. It is possible that the change in MIPCO and GE share a common aetiology. A similar temporal pattern in response to prolonged exercise and also in recovery, is evident between ΔGE and ΔMICPO. Support for a link between GE and maximal muscle function is provided by the observations of Vollestad et al. (1990) and Sejersted and Vollestad (1992) who have found that a $\dot{V}O_2$ drift inversely mirrors the decline in MVC during prolonged repetitive isometric exercise. Given that the reductions in GE and MICPO are different in the present study (ΔGE = 6.1% and ΔMICPO = 3.8%), it is unlikely that this is caused by a common mechanism. In addition, these two parameters

were dissociated by dehydration which appeared to increase the ΔGE but not ΔMICPO. The correlation between ΔGE and ΔMICPO for individual data was also found to be variable.

9.4.7 Maximal isometric force

Maximal isometric force measured on the ergometer was significantly decreased from 154 to 144 Nm post exercise. The 2 h recovery period was sufficient to restore force partially to 148 Nm, which was not significantly different from initial force, ($P > 0.05$). Seated leg extension force did not change significantly from 179 Nm at 5 min to 175 Nm at 120 min ($P > 0.1$) which is in agreement with Nielsen et al. (1993). No effect of dehydration during exercise or CHO intake during recovery was observed for either isometric test.

The difference in maximum static torque generated in the two procedures is thought to be due to a difference in joint angles and muscle recruitment. The body position and muscle lengths adopted during a seated leg extension are not thought to correspond to those occurring during cycling. The reason for the significant change in peak torque on the ergometer but not during the seated leg extension, is likely to be related to the associated changes in muscle length and recruitment. This finding questions the interpretation of previous studies where the effects of prior cycling exercise on isometric force have been assessed in this manner (Hoffman et al. 1985; Nielsen et al. 1993). It is possible that the post exercise delay in testing seated isometric force is responsible for a significant effect not being found. The ergometer isometric test was conducted approximately 4.5 min after each exercise bout and the seated leg extension test a further 1.5 min after this, i.e. 6 min post exercise. This is considered unlikely to have affected the results of the isometric tests. The ergometer MVC was not fully restored with 2 h recovery following the endurance exercise, (albeit a non significant difference $P > 0.05$). The short delay in testing seated MVC considered in this context does not appear important. A rapid recovery of MVC is probably due to PCr resynthesis. Recovery of PCr occurs exponentially after exercise with a half-time of approximately 20 s (Harris et al. 1976). An extensive PCr resynthesis between isometric tests (4.5 - 6 min of recovery) is therefore improbable as PCr recovery would have been largely complete prior to the first test. Large changes in acid-base balance are also

unlikely, due to the moderate intensity of the endurance exercise and pH is not thought to impair force production (Sahlin and Ren, 1989).

9.4.8 Non-metabolic fatigue

The new findings of this study are the progressive exercise induced reduction and slow recovery of GE and dynamic force in particular. Edwards et al. (1977) have described a muscle fatigue observed at low frequencies of stimulation that persists for many hours (e.g. up to 24 h) after the fatiguing exercise. The low frequency fatigue was induced by repeated electrical stimulation, isometric and dynamic exercise (including 60 min of cycle ergometry at 60% $\dot{V}O_{2peak}$). Edwards et al. suggested that the fatigue was the result of an impairment in the excitation-contraction coupling of the active muscle, as changes in ATP, PCr and surface EMG were not found to be associated with the reduced low frequency force. Subsequent studies have tended to confirm the "non-metabolic" origin of low frequency fatigue, its prolonged recovery and the probable role of excitation-contraction coupling in its aetiology (Miller et al. 1987; Moussavi et al. 1989; Baker et al. 1993). Recently Chin and Allen (1997) have found evidence for both glycogen dependent and independent mechanisms of fatigue. Moreover, they postulated that excitation-contraction coupling could be compromised by both mechanisms. An impairment of excitation-contraction coupling may reduce muscular efficiency and account for the ΔGE, in addition to the lower values of maximal muscle function. Studies on the role of a non-metabolic fatigue in the aetiology of fatigue during prolonged exercise, and its influence on dynamic muscular efficiency have not been published to this author's knowledge.

9.4.9 Conclusion

The main findings of this study are that GE, MICPO and MVC are all reduced following a prolonged bout of moderate intensity exercise. In recovery all three parameters appear to be restored slowly. Dehydration appears to have increased the reduction in GE, but did not affect the measures of maximal muscle function. The ingestion of CHO during recovery, thought to influence markedly the rate of muscle glycogen synthesis, did not alter the recovery of GE, MICPO or MVC. The cause for these exercise induced changes is not known, but a fatigue of non-metabolic origin is

speculated. Finally, the differential response of ΔGE and ΔMICPO to exercise and dehydration suggests these two parameters are not tightly linked.

Chapter 10: General Discussion

10.1 Power measuring cranks

The first experiment described in this thesis (Chapter 5) was a calibration study of a power measuring crank (SRM, Julich, Germany). Used subsequently for all power output measurements, the PMC was demonstrated to agree closely with the power output of a Monark ergometer (95% limits of agreement = ± 2.1 W). A difference between the true power input and that measured by either the PMC or the Monark was noted. The difference between power input and power measured arises from frictional losses in the chain and bearings and the ineffective forces applied to the ergometer. For this reason, it is probable that previous studies based upon cycle ergometry have underestimated muscular efficiency (e.g. Whipp and Wasserman, 1969; Gaesser and Brooks, 1975; Suzuki, 1979; Coyle et al. 1992).

The 95% limits of agreement between Monark and PMC increased fourfold when the ergometer was pedalled by a subject instead of being treadmill driven. The different limits of agreement were attributed to the increased fluctuations in pedal cadence which have been reported by Sargeant and Davies (1977) to amount to ± 10 rev·min^{-1}. Conventional calculations of power output from friction loaded ergometers do not account for these changes in energy input associated with variations in flywheel angular velocity. Lakomy (1986) found that peak power output was underestimated by over 30% when the inertia of the flywheel was not considered. The PMC is able to account for these variations in work rate within and between pedal revolutions due to its site of power measurement. It is possible that in this thesis, the accurate quantification of work rate afforded by the use of the PMC has provided greater precision in the calculation of changes in GE.

10.2 Endurance performance

The findings of Experiment 1 (in Chapter 7[1]) highlight the distinction between aerobic power and endurance capacity. Although these terms are often used synonymously (e.g. Coyle et al. 1988), their contributions in determining endurance performance were found to be distinct and separate. The importance of peak aerobic power in determining endurance performance is widely accepted (Burke et al. 1977; Burke, 1980; Sjøgaard et al. 1986; Miller and Manfredi, 1987; Coyle et al. 1988, 1991a; Hawley and Noakes, 1992; Wright et al. 1994). Consistent with these studies, and in particular the findings of Hawley and Noakes (1992), was the strong correlation in Experiment 1 found between PAPO and performance power output. Endurance capacity reflects an individual's ability to resist fatigue at a constant work rate during prolonged exercise (e.g. Williams et al. 1990). Performance power in Experiment 1 was reduced following a sustained bout of moderate intensity exercise. This fatigue was evident in trained subjects, despite the exercise being of moderate intensity and duration and the ingestion of CHO and fluid during exercise. Thus, it can be seen that endurance performance is not governed solely by peak aerobic power, but also by the ability to resist fatigue during endurance exercise. Neither indices of peak aerobic power, nor blood lactate accumulation correlated significantly with the change in performance observed following endurance exercise. This finding provides clear evidence that peak aerobic power and endurance capacity are not related.

10.3 Gross efficiency

To date, the effects and nature of fatigue during prolonged cycle exercise have received little attention. This thesis demonstrates that a ΔGE is consistently observed during sustained moderate intensity exercise, and is implicated in the fatigue observed. Factors influencing the ΔGE have not been extensively examined previously. The exercise induced ΔGE from all experimental conditions of this thesis are summarised in Table 10.1. For comparative purposes the data are normalised to the change in GE per h. Gross efficiency was reduced progressively during exercise -0.65% to -0.85% per h under

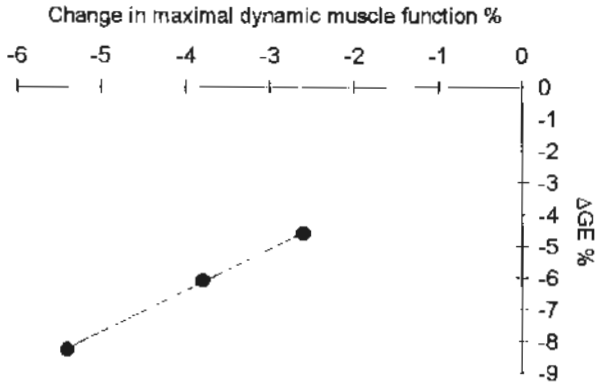
[1] For clarity the experiment detailed in Chapter 7 will be referred to as Experiment 1, Chapter 8 as Experiment 2 and Chapter 9 as Experiment 3.

normal exercising conditions (Experiments 2 & 3). The combined data of Table 10.1 imply that a 5 min bout of high intensity exercise results in a rapid and sustained reduction in GE, whilst in contrast, a 30 s maximal sprint has little effect. The ΔGE for the endurance trial in Experiment 1 is approximately twice as large as for the other exercising conditions. Furthermore, a ΔGE is apparent in the control trial of Experiment 1, where a 70 min rest was allowed between GE measures. These observations support the suggestion in Chapter 7 that the reduction in GE was substantially greater than the change in performance due to the timing of data collection (i.e. before instead of after PT_1). The persistent reduction in GE following PT_1 in the control trial of Experiment 1, is in agreement with the recovery data of Experiment 3. The analysis of Table 10.1 leads to the speculation that an interaction between exercise intensity and duration is apparent in the ΔGE.

Table 10.1 Changes in GE per h

Experiment	ΔGE per h
1C	-0.45
1	-1.43
2C	-0.01
2	-0.85
3E	-0.65
3D	-1.02

C = Control trial, E = Euhydration, D = Dehydration



muscle function and GE for Experiments 2 & 3.

10.4 Maximal muscle function

As the changes in GE are thought to arise directly from the active muscle mass (see section 2.7.3), it was of interest to determine whether maximal muscle function was also compromised in situations where GE was reduced. Experiment 2 revealed that both 30 s peak and mean power output were compromised by prior moderate intensity endurance exercise. No association was found between the reductions in sprint performance and GE (ΔPPO -2.6% vs. ΔGE -4.6%). A similar result was obtained in Experiment 3, where

MICPO was found to be significantly reduced (-3.8, and -5.4%, for euhydration and dehydration n = 11 and 9 respectively) but the corresponding values for ΔGE (-6.1 and -8.29%) were not consistently correlated for individual data. The variable nature of the measures of GE and in particular MICPO may be responsible for the inconsistent individual responses. Notably, the mean exercise induced changes in maximal dynamic muscle function and GE are correspondent for Experiment 2 and both conditions of Experiment 3 (Figure 10.1 above) and may indicate these parameters are related. In this graph maximal muscle function is represented by peak power output for Experiment 2 and MICPO for Experiment 3.

In contrast to the effect of GE on performance, the reduction in maximal muscle function is not thought to limit aerobic performance directly. Fulco et al. (1996) observed that MVC at exhaustion following sustained dynamic leg extension exercise, was still three times higher than the required peak dynamic force. In the present thesis prolonged moderate intensity cycle exercise reduced peak power output by 2.5 - 5.5%. Sargeant and Greig, (1988) have reported that peak pedal forces at $\dot{V}O_{2peak}$ require 48% and 44% of maximum, at cadences of 86 and 100 rev·min^{-1} respectively. It is concluded therefore, that the small exercise induced reductions in peak power output associated with endurance exercise are unlikely to prove limiting, except during maximal sprint exercise. Significantly, the majority of cycle road races tend to finish with a group sprint. Accordingly, these small exercise induced changes in peak power output may be important in determining the outcome of such races.

10.5 Mechanisms for change in GE and PPO

Fulco et al. (1996) suggest that the maximal velocity of shortening was compromised following dynamic leg extension exercise at ≥ 79% $\dot{V}O_{2peak}$. In support of this postulate, significant fatigue induced reductions in maximum power output, V_{opt} and maximum velocity have been demonstrated with rat muscle *in situ* (de Haan et al. 1989). Also, Beelen et al. (1995) have reported a marked decrease in the maximum rate of force development following a 25 s isokinetic sprint in humans. The studies of Fulco et al. (1996) de Haan et al. (1989) and Beelen et al. (1995) have all induced fatigue by heavy or severe exercise. It is not known if their findings are applicable to prolonged moderate

intensity cycle exercise, since the effects of exercise of this nature on the power-velocity characteristics of human muscle are not thought to have been researched.

The present thesis has demonstrated that a progressive change during exercise, and a slow recovery following exercise, characterised both the change in MICPO and GE. Consequently, it is possible that both the reduction in peak power output and GE share a common aetiology. Two separate observations demonstrate that changes in GE and peak power output are not, however, tightly coupled. Clear differences in the magnitude of the changes in GE compared with and PPO and MICPO were evident, with GE reduced to a larger extent than peak power output in all situations. Secondly, a differential response to exercise induced dehydration was found. Maximal isokinetic cycling power output was not affected by fluid ingestion, whilst the decline in GE appeared to be augmented by dehydration. A difference in exercising $\dot{V}O_2$ with dehydration has not generally been reported previously and the reason for this observation remains obscure.

Postulated mechanisms for the cause of the changes in GE resulting from prolonged moderate intensity cycle exercise need to be consistent with the following observations:

1) Change progressively during moderate intensity exercise
2) Recover slowly following exercise
3) Depend upon duration of exercise
4) Exhibit an exercise intensity-duration interaction
5) Possess glycogen dependent and independent processes

It is considered that the main source of the reduced GE is the active muscle mass. This is supported by the findings of previous studies indicating a $\dot{V}O_2$ drift arises directly from the active muscle mass (Sahlin et al. 1990; Poole et al. 1992; González-Alonso et al. 1997). The observation from the present thesis that maximal muscle function is compromised by prolonged moderate intensity exercise is also consistent with this hypothesis. The specific mechanisms of muscle fatigue cannot be determined. A non-metabolic origin may implicated by the extended recovery time common to both. An impairment of excitation-contraction coupling is consistent with the observations of previous researchers investigating this phenomenon. In the absence of a more detailed characterisation of the effects of fatigue on maximal muscle function, or direct muscle biopsy data, a more detailed analysis is beyond the scope of this thesis. Of note however,

are the findings of a recent study by Chin and Allen (1997). These authors found evidence for both glycogen dependent and independent processes in fatigue induced reductions in muscle force and Ca^{2+} release.

10.6 Recommendations for future research

This thesis and associated literature review has identified several areas warranting further investigation. The causes of the changes in GE and maximal dynamic and static muscle function resulting from sustained moderate intensity exercise remain to be established. There are several avenues for further research which may contribute to the understanding of the mechanisms involved.

- A progressive fibre recruitment may contribute to the ΔGE, but it is not clear whether this is a primary cause or effect. Selectively fatiguing different fibre pools with variable intensity exercise may provide a greater understanding of the role of fibre recruitment in $\dot{V}O_2$ drift.
- A significant role for fatigue of a non-metabolic origin has been discussed in this thesis. Little is known about the possible effects of non-metabolic fatigue arising during sustained dynamic moderate intensity exercise. Muscle biopsy samples taken at the end of prolonged moderate intensity exercise could confirm whether reductions in GE and peak power output occur in the absence of any meaningful metabolic disturbances.
- Further insight into the mechanisms of fatigue may also be gained by characterising the effects of endurance exercise on the power-velocity properties of the active muscle mass.
- Throughout this thesis it has been assumed that the pattern of force application on the pedal remains relatively constant. Indeed it is noted that both the change in GE and peak power output could be accounted for by a less efficient pattern of force application on the pedal. Whilst previous research has failed to demonstrate a correlation between GE and efficient force application, the effects of endurance exercise on this parameter are thought to be unknown. The ability of subjects to maintain an effective application of muscular force on the pedals under varying conditions of fatigue needs to be confirmed.

- Manipulating muscle temperature may provide a means of identifying whether the fatigue is specific to Type I or II fibre populations.
- An examination of the relationship between ΔGE and exercise intensity. As the exercise intensity increases any distinction between $\dot{V}O_{2SC}$ and $\dot{V}O_2$ drift is expected to disappear. Furthermore, investigation into the interaction between exercise intensity, duration and ΔGE may be useful.
- Determination of the time course required for full restoration of GE and peak power output following endurance exercise.
- This thesis is thought to provide the first study to demonstrate the importance ΔGE in dictating performance. The relevance of ΔGE to other performance situations or as a mediator of fatigue during endurance exercise remains to be determined.

10.7 General conclusions

This thesis has argued that the adoption of an appropriate definition of fatigue is integral to the development of a comprehensive understanding of the demands of sustained moderate intensity exercise. In defining fatigue as a reduction in the capacity to generate maximum power/force, the utilisation of a more ecologically valid and reliable experimental model to examine the effects of endurance exercise was possible.

This thesis has demonstrated that 30 s and 5 min performance, GE and maximal muscle function are all compromised by sustained moderate intensity cycle exercise. The causes of these reductions remain to be determined. The strong correlation observed between the change in performance and GE implicates a reduction in efficiency as an important limiting factor during endurance exercise. During prolonged moderate intensity exercise GE and MICPO were found to be progressively reduced. The rate of recovery of both GE and MICPO following prolonged exercise was slow and appeared to be independent of CHO availability. Consistent with previous findings exercise induced dehydration did not influence ΔMICPO, but it was found to augment ΔGE. The causes of the exercise induced reduction in GE remain to be established, but were speculated to relate to a compromised muscle function. A temporal similarity in exercise and subsequent recovery responses of GE and MICPO supports this speculation. Research investigating the contribution of alterations in muscle fibre recruitment and fatigue of a non-metabolic

origin is proposed as the most promising lines of further enquiry in extending our understanding of the factors limiting endurance exercise.

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INFLUENCE OF CHANGES IN CYCLING GROSS EFFICIENCY ON ENDURANCE PERFORMANCE

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INTRODUCTION

Racing cyclists may often train and compete for several hours at intensities greater than 60% $\dot{V}O_{2max}$. It is widely accepted that both glycogen depletion and dehydration may play important roles in the aetiology of fatigue during endurance exercise of this nature. Additionally, it is commonly observed that at intensities above 60% $VO_{2\,max}$ oxygen consumption continues to increase gradually beyond the initial 3 min of exercise, Hagberg et al (1978). The precise mechanisms by which these factors may influence the development of muscular fatigue remains to be elucidated. Recently Passfield and Hale, (1997) have demonstrated that this "O_2 drift" is apparent even in elite cyclists exercising at 60% $\dot{V}O_{2max}$ and is associated with a gradual decline in gross efficiency. Furthermore, they found a strong negative correlation between gross efficiency and a decrease in peak aerobic power output observed immediately following the 3 hours trial. Consequently, we were interested to determine whether endurance performance is compromised in a situation where it is likely that neither glycogen availability nor dehydration are limiting factors. This study examined the effects of a 75 min endurance ride at 60% $\dot{V}O_{2max}$ on changes in gross efficiency (GE) and average power output during an all-out 5 min performance trial (PT) in cyclists.

METHODS

Nine active males, all training regularly gave informed consent to take part in this study. These men weighed (mean ± SD) 69.6 ± 14 kg, with $\dot{V}O_{2max}$ 4.13 ± 0.5 $L{\cdot}min^{-1}$, and blood lactate threshold (LT - first rise above baseline) 61% ± 9% $\dot{V}O_{2max}$. Subjects were familiarised with all laboratory equipment and exercise protocols before commencing the study. The study was conducted in a well ventilated laboratory maintained at a temperature of 18.9 ± 0.9 °C. A modified Monark ergometer (model 819) with SRM powermeter (Julich, Germany), infinitely adjustable saddle height, racing saddle and the subjects' own pedals was used for all exercise trials. All power outputs and cadences were verified by use of the SRM powermeter. The subjects undertook two trials, a 75 min endurance trial (ET) and a control trial (CT) in a randomly ordered crossover design. In both conditions after a 6 min warm-up at 60% $\dot{V}O_{2max}$ (WU_1) the subjects attempted to complete as much work as possible in a 5 min performance trial (PT_1). During ET subjects were then allowed a brief rest (3 min) before continuing to exercise at 60% $\dot{V}O_{2max}$ for 75 min and repeating the 5 min performance trial (PT_2). In the control trial subjects simply rested for 72 min before repeating the 6 min warm-up (WU_2) and 5 min performance trial (PT_2). Between PT_1 and PT_2 subjects ingested 10 $ml.kg^{-1}.h^{-1}$ of an 8%, or 500 ml of a 12%, carbohydrate solution during ET and CT respectively. Expired gas was collected between min 5 and 6 of all warm up exercise and between min 74 and 75 (WU_2) of the endurance trial for calculation of GE. Expired gas was also collected serially from 45 s in all performance trials. Thumb-prick capillary blood samples were collected at the end of both $WU_1 + _2$ and 1 min post all performance trials for the determination of blood lactate concentrations. A paired t-test on the difference was used to compare ET and CT results.

RESULTS

The changes in gross efficiency and blood lactate response during WU_1 and WU_2 are shown in table 1. Table 2 lists the average power outputs, mean $\dot{V}O_2$ and peak blood lactate values for the performance trials in both conditions.

Table 1: Warm Up 1 and 2 in CT and ET (Mean ± SD)

	CT-WU_1	CT-WU_2	ET-WU_1	ET-WU_2
Power Output W	185 ± 29	184 ± 29	180 ± 31	182 ± 30
$\dot{V}O_2$ L·min^{-1}	2.42 ± 0.27	2.48 ± 0.29	2.37 ± 0.34	2.61 ± 0.35
Gross Efficiency %	22.4 ± 1.7	21.8 ± 1.6	22.4 ± 0.8	20.7 ± 1.1
Blood Lactate mmol·L^{-1}	1.4 ± 0.3	1.8 ± 0.5	1.3 ± 0.5	1.3 ± 0.5

The change in gross efficiency (ΔGE) was significantly greater at the end of ET compared with CT ($p < 0.005$).

Table 2: Performance Trials 1 and 2 in CT and ET (Mean ± SD)

	CT-PT_1	CT-PT_2	ET-PT_1	ET-PT_2
Mean Power W	325 ± 44	325 ± 45	322 ± 47	310 ± 48
Mean $\dot{V}O_2$ L·min^{-1}	3.85 ± 0.43	3.93 ± 0.46	3.90 ± 0.54	3.93 ± 0.56
Peak Lactate mmol·L^{-1}	9.0 ± 1.5	8.3 ± 1.2	8.3 ± 2.3	7.4 ± 2.2

Performance was reduced significantly (ΔPT) in ET (-3.7%, $p = 0.005$) in comparison with CT where average power remained unchanged. A significant correlation between ΔGE and ΔPT was observed $r = 0.70$ ($r = 0.90$ with the removal of a significant outlier). A significant correlation was also observed between relative $\dot{V}O_{2max}$ and ΔPT ($r = 0.74$) but not with LT and ΔPT.

DISCUSSION

This study demonstrates that GE and performance are reduced following an endurance trial at 60% $\dot{V}O_{2max}$ for 75 min and that these two variables are strongly correlated. The cause of the reduced efficiency during endurance exercise is not currently fully understood. It is speculated that an increase in the energy cost of muscle contraction or ATP re-synthesis and a progressive increase in fibre recruitment are responsible. In the present experiment it is not thought likely that dehydration or glycogen depletion would have been of sufficient extent to influence ΔGE and ΔPT due to the moderate volume of work performed and the preventative strategies implemented. Relative $\dot{V}O_{2max}$ correlated with ΔPT, implying a higher aerobic power is associated with a greater resistance to fatigue. The performance trial protocol and careful subject control yielded exceptionally low CV's (1.7% ± 1.3%) which recommends it as a useful model for investigating endurance performance.

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Effect of endurance exercise on 30 s Wingate sprint in cyclists

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The effects of exercise on subsequent muscle function have been variously documented (e.g. Sargeant & Dolan, 1987; Rademaker *et al.* 1994). To our knowledge, changes in sprint performance following prolonged, non-exhaustive, moderate intensity exercise in trained, endurance racing cyclists has not been examined. We studied the effect of extended moderate intensity exercise (70 min, 65 % $V_{O2,peak}$) on 30 s maximal unpaced (Wingate) performance in nine male cyclists.

A modified Monark ergometer (model 814) with SRM powermeter (Julich, Germany), infinitely adjustable saddle height, racing saddle and the subjects' own pedals was used for all exercise trials. Power output and cadence were measured by use of the SRM powermeter, which measures torque directly from the crank with strain-gauges. Subjects performed two conditions: control (C) and exercise (E), both consisting of two 30 s sprints separated by 60 min of either rest or pedalling at 65 % $V_{O2,peak}$, respectively. Immediately prior to each sprint, subjects worked at a controlled cadence of 100 rev min^{-1} for an additional 10 min. Between sprints subjects ingested 500 ml of a 12 %, or 10 ml kg^{-1} h^{-1} of an 8 % carbohydrate solution during C and E, respectively. Peak power output (PPO) measured as the highest 1 s value, mean power (MPO) and fatigue index (FI) were calculated for each test. A thumb-prick capillary blood sample was taken 3 min post-sprint to determine blood lactate concentration $[Lac]_{post}$. A paired *t* test on the difference between first and second sprint was used to compare the effect of exercise with rest.

Exercise at 65 % $V_{O2,peak}$ caused a significant drop in PPO and MPO when compared with the control trial, whilst FI did not change significantly from 55 ± 14 to 56 ± 13 % ($P > 0{\cdot}4$) (see Table 1). A significant reduction in $[Lac]_{post}$ ($P < 0{\cdot}005$) following the exercise trial was also observed, from 3·6 (1·2) to 6·6 (1·5) mM.

Table 1. Sprint results

	Control trial		Endurance trial	
	Pre	Post	Pre	Post
Peak power (W)	991 (177)	908 (178)	985 (150)	859 (169)*
Mean power (W)	687 (85)	618 (80)	683 (94)	628 (93)†
Fatigue index (%)	58 (17)	58 (17)	55 (14)	56 (13)

All values are given as mean (s.d.). Control vs. endurance: * $P < 0{\cdot}05$; † $P < 0{\cdot}01$.

An extended bout of prior moderate exercise appears to compromise both peak and average sprinting power. The mechanism for these changes in Wingate performance is unknown.

All procedures employed in this study were approved by the University of Brighton Ethics Committee.

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CHANGES IN MAXIMAL ISOKINETIC CYCLING POWER DURING PROLONGED EXERCISE AND IN RECOVERY

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We have recently demonstrated significant reductions in peak and average 30s sprint power are found following 70 min of non-exhaustive, moderate intensity, cycling exercise (Passfield and Doust, 1998). This study sought to extend these observations by examining changes in isokinetic cycling sprint power output during 2 hours of exercise and subsequent recovery.

Eleven experienced male racing cyclists (mean ± SD); mass 70.8 ± 7.3 kg; height 1.81 ± 0.07 m; peak VO_2 4.44 ± 0.55 l.min^{-1} gave informed consent to take part in this study, which had University of Brighton ethical committee approval. Subjects exercised for 2 hours at 60% VO_{2max} whilst pedalling at 90 rev.min^{-1} and for a further 5 min at 25, 55, 85 and 115 min of recovery. During exercise body mass was maintained by the ingestion of a 50 mmol.l^{-1} Na^+ solution. On completing the endurance trial subjects ingested 1.5 g.kg^{-1} BM.h^{-1} carbohydrate in a combination of liquid and solid form. Maximal isokinetic cycling power output during a 6s sprint at 90 rev.min^{-1} was determined at 5, 30, 60, 90 and 120 min of exercise and also at 30, 60, 90 and 120 min of recovery. A 3 min rest period immediately preceded every sprint. Rectal temperature and thumb-prick capillary blood lactate and glucose were measured during the last 60s of exercise before each sprint. A one-way ANOVA with repeated measures was used to examine changes over time (F value considered significant at $P<0.05$). A difference between 5 min and subsequent time points was located with Dunnett's post hoc test.

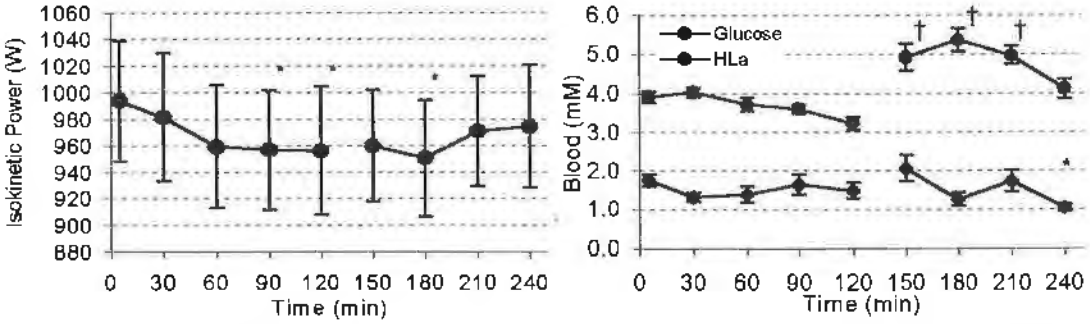

Figure 1. A: Isokinetic Power Output. Figure 1.B: Blood Glucose and Lactate Concentrations. All values are mean ± SE. * $P<0.05$ from 5 min value. † $P<0.01$ from 5 min value ($P<0.01$).

Maximal isokinetic cycling power output declined progressively during exercise and was restored slowly during recovery (Fig. 1.A). A significant reduction in power output was found at 90 and 120 min of exercise and 60 min of recovery. Rectal temperature was significantly elevated (≈38.5°C, $P<0.01$) from 30 min until the end of the exercise period, thereafter it dropped rapidly to ≈37.2°C and remained similar to initial values throughout the recovery period. Blood lactate did not change significantly until the last time point in recovery, where a significant decrease in concentration was observed (Fig. 1.B). A gradual non-significant, decline in blood glucose was seen in the later stages of the endurance trial. This was immediately reversed in the recovery period where carbohydrate ingestion resulted in a significant elevation in blood glucose from 30 to 90 min (Fig. 1.B).

This study demonstrates that maximal isokinetic cycling power output in well-trained cyclists is significantly reduced during 2 h of non-exhaustive exercise. The decline in power output occurred despite an increase in body temperature and the moderate exercise intensity; i.e. characterised by an unchanging blood lactate concentration. Furthermore, the recovery of maximal isokinetic power output was gradual, with a significant reduction still found 1 h after exercise despite carbohydrate ingestion in recovery. The cause for the decrement in power output cannot be determined from this study, but the slow rate of recovery implicates processes known to have a similar temporal pattern; e.g. muscle glycogen depletion and non-metabolic fatigue (Sahlin and Seger, 1995).

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