An Assessment of the Kinematic and Metabolic Characteristics of the Breaststroke

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Abstract

This thesis aimed to provide an examination of the kinematic and metabolic responses of breaststroke swimming, including the effects of changing pace. Study 1 provided a description of competition swimming including rarely reported temporal elements (start time (ST) to 15 m, turning times (TT s) for the 7.5 m ingress and 7.5 m egress, end time (ET) - final 5 m) as well as repeated measurements of turning times, mid-pool swimming velocity, stroke rate (SR) and stroke length (SL) as races progressed. Mid-pool swimming velocity, ST and TT were found to be significantly related with finishing time (FT) and each other suggesting that coaches should adopt an holistic approach to the training of breaststroke swimmers. Stroke rate and TT were found to increase as races evolved while mid-pool SV and SL decreased. A detailed comparison of the 100 m and 200 m events suggested that event specific preparation might be needed and that there was a potential for swimmers of the 200 m event to reduce their ST. In Study 2 a multiple regression analysis utilising kinematic and temporal variables demonstrated that SV was the primary determinant of FT. Turning time was the secondary determinant of FT in all events except the men's 100 m where ST had greater relative importance. The analysis produced precise predictive equations of FT which could be used by coaches to predict race performance and to prescribe race pace training. Studies 3, 4 and 5 established that breaststroke swimmers could be paced precisely and reliably using the Aquapacer™ during moderate to high intensity 200 m breaststroke trials, subject to the onset of fatigue. Stroke kinematic (SR, stroke count - SC) and metabolic responses (blood lactate, gas exchange and heart rate) elicited during the trials of Study 3 were found to be reproducible. During 200 m trials paced at 98 %, 100 % and 102 % of a subject's mean maximal 200 m speed (Study 4) SR was elevated to increase swimming velocity, and to compensate for a deterioration in SL caused by lactacidosis. Lactacidosis occurred because swimmers were operating beyond their maximal aerobic power even during the 98 % trial and hence the additional speed in the faster trials required an increased anaerobic contribution. In support of this, post exercise blood lactate concentrations were significantly negatively related to FT in the 98 % and 100 % trials. Turning times were initially shorter at the start of the faster trials but a marked deterioration followed demonstrating their sensitivity to lactacidosis. An anomaly across studies was that changes in kinematic variables were less predictable during the final 100 m of positively split men's 200 m races (Study 1) and trials (Study 4). A subsequent comparison of positively split, evenly split and negatively split 175 m trials (Study 5) demonstrated that stroke kinematics remained significantly related over the whole distance of the evenly split trial compared with the positively split trial. Subjects also demonstrated reduced blood lactate, RER and RPE values following the evenly split trial compared with the positively split trial. It was suggested that coaches should experiment with an evenly split race strategy to determine if it produces shorter FT s compared with the positively split patterns currently adopted in competition. A common finding was that breaststroke swimmers exhibited unique SR : SL ratios so that both SR and SL were poorly related to FT in all the studies. It was suggested that coaches, using the Aquapacer™ , could entrain a swimmer's ideal SR to elicit a more evenly paced and consistent competition performance. Pacing the ideal SR could also be used for a race specific test, because if a swimmer becomes better able to maintain TT s and SL over a given distance the FT would be improved which might indicate a potential for an improved competition performance. Finally, a model of a maximal even paced 200 m breaststroke swim was outlined and the effect of a change of pace discussed with respect to the model.

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Definition of Terms

Better - superior to another, in this thesis the term has been used in the context of finishing time eg. a better performer, would be a swimmer with a shorter finishing time

Elite - the choice part of a group, which in this thesis were swimmers who were competing in the A and B finals of major international competitions.

International - swimmers competing in the A and B finals of international championships (eg. European, World Cup).

Kinematics - a branch of dynamics that deals with aspects of motion without consideration of force or interaction. Kinematic variables measured in this thesis include swimming velocity, stroke rate and stroke count.

National - swimmers competing in the A and B finals of their national swimming championships (eg. Canadian, Welsh).

Pacing - a regulated rate of progress. In a number of studies in this thesis subjects attempted to coincide reaching a particular point in the pool with an audible signal given by a pacing device.

Temporal - denotes a distinction of time, which is not strictly speaking a kinematic parameter. Temporal variables measured in this thesis include start time, turning time and end time.

Variables

ET End time (s) - the time taken for a swimmer to complete the last 5 m of a race

HR Heart rate ($b.min^{-1}$)

La Blood lactate (mM)

O_2 Molecular oxygen

RER Repiratory exchange ratio (VCO_2/ VO_2)

RPE Rating of perceived exertion

SC Stroke count ($S.length^{-1}$) - the number of stroke cycles per length of the pool

SL	Stroke length (m) - the distance travelled per stroke cycle
SR	Stroke rate ($S.min^{-1}$) - the number of stroke cycles per minute
ST	Start time (s) - the time taken for a swimmer's head to reach 15 m following the starting signal
SV	Mid-pool swimming velocity ($m.s^{-1}$) - the velocity of the swimmer's head travelling from 15 m to 25 m (100 m event only) or from 25 m to 42.5 m (100 m and 200 m event) along the length of the pool
TT	Turning time (s) - the time taken for a swimmer's head to travel 7.5 m into the turn and then return to the same point
$\dot{V}E$	Expired minute ventilation ($l.min^{-1}$)
$\dot{V}E/\dot{V}O_2$	Ventilatory equivalent for oxygen
$\dot{V}CO_2$	Carbon dioxide production ($l.min^{-1}$)
$\dot{V}O_2$	Oxygen uptake ($l.min^{-1}$)

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Chapter 1

Introduction

1.0 Introduction

Scientific findings on swimming are traditionally based on the freestyle stroke. Such findings are not readily transferable to the other strokes, since a particular stroke has its own unique kinematic and metabolic characteristics. For example, the breaststroke arm pull is exceptional because unlike the arm pull of the other competitive strokes, it contributes less propulsion than the leg kick phase (Rennie *et al.*, 1973). The breaststroke is also characterised by a series of accelerations and decelerations during a stroke cycle, which are far more exaggerated than for other strokes. Indeed, D'Aquisto *et al.*(1988) observed that the leg kick element of the breaststroke yielded a peak velocity during the stroke cycle of 1.81 $m.s^{-1}$ while the leg recovery phase brought the swimmer to a virtual standstill (0.24 $m.s^{-1}$). Despite this, the breaststroke's leg propulsion phase is actually more energetically efficient than the freestyle's (Holmer, 1974a). However, due to the breaststroke arm pull being much less efficient, the whole stroke requires a greater oxygen uptake for a given swimming velocity than the freestyle (Holmer, 1974a; Lavoie and Montpetit, 1986).

The greater reliance on the leg kick in the breaststroke means that there are unique kinematic and metabolic characteristics for this stroke, particularly when a swimmer attempts to increase swimming velocity at racing velocities. However despite these distinctive differences, a review of the literature demonstrates that there has been a lack of scientific research investigating the

unique kinematic characteristics and metabolic responses of the breaststroke, at high swimming velocities close to racing pace.

A few researchers have focused on the kinematic aspects of breaststroke swimming (Craig and Pendergast, 1979 , Kennedy *et al.*, 1990, Chengalur and Brown, 1992; Wakayoshi *et al.*, 1992). Their studies have highlighted that during races, stroke rate (SR) increases in a compensatory manner as stroke length (SL) decreases. There has also been a tentative observation made that better swimmers might complete fewer stroke cycles during a race (Craig *et al.*, 1979; Wakayoshi *et al.*, 1992) because they are able to maintain a greater distance per stroke (Thompson and Haljand, 1997). Recently, researchers have investigated temporal elements such as the time taken to reach a set distance from the start of the race (start time), the time to travel a set distance into the turn and back out again (turning time - TT), and the time to complete the last few metres of the race (end time - ET) (Wakayoshi *et al.*, 1992; Thompson and Haljand, 1997).

The inter-relationships between these kinematic and temporal variables and their relationships with finishing time have not been comprehensively researched for the breaststroke. Furthermore, relationships within those variables which can be repeatedly measured, as a breaststroke race evolves (SR, SL, swimming velocity, TT) have not been established, and neither have differences in their absolute values. Finally, although there is some evidence that characteristic differences exist between the 100 m and 200 m events in

terms of SR and SL (Craig *et al.*, 1985), it has not been established if this is the case with respect to other kinematic and temporal variables. The lack of investigation in this area merits further research, particularly if improvements in swimming performance over time are more to do with advances in technical ability than in maximal energy expenditure (Pendergast *et al.*, 1977).

A fundamental requirement when manipulating pacing to investigate the associated kinematic and metabolic responses is to establish the precision and reproducibility of the pacing. A literature search of peer reviewed studies yielded only a small proportion that had attempted to establish a precise and reproducible method of pacing. This may be due to the fact that there has not been a gold standard method of pacing in swimming studies in the same way that a treadmill has become for running studies. Rather in swimming, the coach dictates the target time, while the actual precision of the pacing is left to the swimmer's own innate judgement, albeit aided by cursory glances toward the pool-side clock. As a consequence, a swimmer develops a fairly accurate sense of pacing as a result of being chronically involved in this practice. However, the degree of precision and reliability achieved has not been formally investigated. Consequently, the error inherent in unaided swimming pacing is not known, and although the coach may be convinced that a particular swimmer's pace judgement is precise enough for training purposes, this suggestion may in reality be incorrect.

Recently a novel pacing device, the Aquapacer™ (Challenge and Response, Inverurie, Scotland), has been developed which may overcome many of the problems associated with previous pacing systems. The Aquapacer™ comprises a programming unit which can be interfaced with a "coin size" programmable sounding unit. When programmed, the sounding unit is placed under the swimmer's cap close to the ear. Once activated the unit emits a distinctive "bleep" noise at a pre-determined frequency, accurate to 0.01 s. The unit can be programmed to pace the swimmer's velocity either in relation to lane markers or the swimmer's stroke rate. Martin and Thompson (1999, 2000) have recently reported that during a sub-maximal set of 10 x 100 m freestyle repetitions, the swimmer's pace judgement with the Aquapacer™ was both precise and reliable. These results should also be possible for the breaststroke, although such an investigation has not been undertaken.

The reproducibility of kinematic characteristics and metabolic responses during breaststroke swimming have not been quantified. If the Aquapacer™ was found to be both precise and reliable, in terms of pacing the breaststroke, then it would be entirely possible to investigate the reproducibility of selected kinematic and metabolic variables during high intensity breaststroke swimming.

It has been argued that breaststroke swimming performance is dependent on the total energy expenditure and the technical skill of the swimmer (Thompson and Haljand, 1997). Given that a breaststroke performance is multifactorial, it is not suprising that intra-individual competitive swimming performances often differ

by a small margin on a swim by swim basis. Thompson (1998) reported a 2.8 % difference in finishing time between heats and finals in national breaststroke swimmers. However, little, if any, quantitative data has been published for the breaststroke with regard to how kinematic or metabolic variables change during performances differing by only a few seconds. This means that the breaststroke "performance" is not as well understood as many coaches would perhaps admit to. In fact, how breaststroke swimmers manipulate their stroke kinematic characteristics in order to make fine adjustments to their performance at or near to racing velocity has not been researched. Neither have the effects of such kinematic adjustments on metabolism. For example, it remains unclear whether breaststroke swimmers favour an increase in SR rather than an increase in SL when attempting to increase swimming velocity at or near to racing pace. Fundamentally, the kinematic, temporal and metabolic responses associated with small changes within breaststroke pacing at or near to racing velocities have rarely been deliberately quantified, and certainly never in the same study.

The pacing of a race can be divided into three distinct strategies:

i) even pacing - where the pacing is the same throughout the race distance,

ii) positive pacing - where the pacing over the first half of the race is faster than over the second half of the race, and

iii) negative pacing - where the pacing over the first half of the race is slower than over the second half of the race.

The pacing strategy most suited to a given event is unknown, although a number of recent world records have been broken in predominantly aerobic events with either even paced or slightly negatively paced races (400 m freestyle swimming - Ian Thorpe, 1999; 5,000 m & 10,000 m running - Haile Gabraselassie, 1997; 5,000 m running - Daniel Komen, 1997; 10,000 m running - Paul Tergat, 1997). Maglischo (1993) has reported that in swimming races where energy from anaerobic sources predominates (i.e.100 m and 200 m), the second half of the race is often slower than the first half (positively split pacing). This is particularly so in breaststroke races, due to the onset of leg fatigue. Yet, there have been few investigations which have determined the pacing strategy most likely to maximise performance over a particular race distance.

Foster *et al.* (1994) suggested that pacing may be complicated by physiological mechanisms such that performance in shorter events might be limited by metabolite accumulation, while performance in longer events might be limited by substrate depletion. However this view may be too simplistic given that intra-muscular creatine phosphate concentration has been found to be depleted during high intensity exercise. What seems apparent is that if metabolite accumulation limits performance then the duration of the race is of consequence. For example, if a race is of sufficient duration and intensity that the accumulation of lactic acid and associated lowering of the pH within the

muscle leads to fatigue before the end of the race, then the pacing strategy is of great significance. Conversely a pacing strategy may not be required in races which are too short in duration either to, incur sufficient metabolite accumulation or creatine phosphate depletion, as fatigue may not be evident.

Both the 100 m and 200 m breaststroke events have been reported to elicit high levels of blood lactate (Thompson, 1998). These stroke-specific findings agree with a general view held by Holmer (1974) that successful 100 m and 200 m swimmers possess a pronounced anaerobic capacity. Pace judgement in such events would therefore seem to be of some importance given that there is a strong association between high lactic acid levels and fatigue. The need for an appropriate pacing strategy may also be more important in 200 m events, given that some researchers have indicated that greater blood lactate values might be associated with this event (Madsen and Lohberg, 1987; Thompson, 1998). At present there exist no studies which have compared the effect of even, positive and negative pacing on 200 m breaststroke performance, or the associated kinematic, temporal and metabolic responses.

This thesis aims to further provide an examination of the kinematic, temporal and metabolic characteristics of 100 m and 200 m breaststroke swimming, and to investigate the effect of changing pace. These aims will be addressed by the achievement of the following objectives:-

Objective 1 (Study1) - to evaluate kinematic and temporal variables during 100 m and 200 m breaststroke races in order to: establish interrelationships, assess changes within variables during the evolution of races, and identify if there are characteristic differences between 100 and 200 m events in national-elite male and female swimmers.

Objective 2 (Study 2) - to determine the relative importance of selected kinematic and temporal variables in relation to swimming performance in national-elite male and female 100 m and 200 m breaststroke swimmers.

Objective 3 (Study 3) - to establish the precision and reproducibility of sub-maximal breaststroke swimming using a novel pacing device (Aquapacer™) : and to assess the reproducibility of the associated kinematic and metabolic responses.

Objective 4 (Study 4) - to assess the effect on kinematic, temporal and metabolic responses when pacing is subtly manipulated during maximal 200 m breaststroke swimming using the Aquapacer™.

Objective 5 (Study 5) - to assess the effect of even, positive and negative split pacing strategies on kinematic, temporal and metabolic responses during 175 m breaststroke trials using the Aquapacer™.

Chapter 2

Review of Literature

2.0 Review of Literature

This review begins by identifying how recent developments in measurement techniques and the management of pacing control provide the oppurtunity for the kinematic and metabolic characteristics of the breaststroke to be assessed during high intensity swimming, with a greater degree of precision and reliability than has previously been possible. A synopsis of the current knowledge regarding the physical, physiological and kinematic characteristics of breaststroke swimmers from experimental and competitive situations follows. The limited understanding regarding how and which metabolic and kinematic variables alter with changes in swimming speeds, or between competitive performances is established. The research regarding how these variables explain performance differences between swimmers is also reviewed. Finally, the lack of research investigating the effect of changes within pace on kinematic and metabolic variables is highlighted, aswell as the need to establish the effect that different pacing strategies might have on these variables, in order to determine if one strategy is more appropriate than another for racing.

2.1 Introduction

Scientific investigation into swimming is thought to have begun with a series of studies by Liljestrand and Strenstrom (1919) and Liljestrand and Lindhard (1919). These studies reported oxygen uptake and cardiac output data, calculated from expired air collections, taken while swimmers were paced by a

rowing boat in a lake and during tethered swimming. Only limited research continued (Karpovich and Millman, 1944; Karpovich and Pestrecov, 1939) until the late 1950 s when applied physiological research into swimming began in earnest and led to a body of work being developed during the 1960 s. At this time research was particularly concerned with the energy consumption and maximal work capacity of swimmers in terms of the whole and partial stroke (arm and leg contributions) and the effect of water immersion on thermal balance. Comparisons with other exercise modalities were also of great interest to scientists. The reader is directed to a comprehensive review by Faulkner (1968) for further detail. In the 1970 s research began to emerge on the biomechanics of swimming notably through a number of international symposia (Clarys and Lewillie, 1971, 1975, Terauds and Bedingfield, 1979) and a review of work to date by Miller (1975). Congresses on swimming medicine also began in the 1970 s (Carney, Conroy and Hingerty, 1971; Eriksson and Furberg, 1978) through funding from the international swimming body, FINA. Further advances in physiological research were comprehensively reviewed by Holmer (1979) and Lavoie and Montpetit (1986). To the present day research into the biomechanical and physiological aspects of swimming has continued unabated, however over the years there has been a consistent bias toward research based on the front crawl stroke. This is perhaps natural as the majority of competitive and long distance events are swum in this manner, however it does mean that other swimming strokes such as the breaststroke are less well understood.

2.2 The development of physiological measurements and management of pacing control during free swimming

Largely due to the swimming environment, studies prior to 1970 developed a body of knowledge despite a lack of reliable measurement methods (Holmer, 1979). The constraints of the swimming pool, particular swimming strokes and turns at the end of each length made measurements during free swimming difficult. To overcome such difficulties subjects were asked to breath hold while swimming so that the oxygen cost could be estimated from post exercise respiratory gas exchange measurements (Karpovich and Millman, 1944), or post exercise respiratory measurements were backward extrapolated to estimate actual exercise responses; however these techniques were subsequently questioned as producing misleading results (Christensen, Hedman and Holmdahl, 1960; Astrand and Rodahl, 1977; Lavoie and Montpetit, 1986). For example early investigators concluded that an exponential relationship existed between oxygen uptake and swimming velocity (Karpovich and Millman, 1944), however similar studies incorporating improved methods of measurement have since reported the relationship to be linear, at least for the frontcrawl (Faulkner, 1968; McArdle *et al.*, 1971; Montpetit *et al.*, 1983).

A further shortcoming of the pre-1970 research was the lack of control that could be exerted over the swimming velocity of the swimmers during free swimming and this made studying the effects of different rates of work difficult. For this reason tethered swimming became a popular alternative to

free swimming in applied physiology research studies, particularly as it was demonstrated that physiological responses differed relatively little from those measured during free swimming (Le Pere and Porter, 1971; Heigenhauser and Faulkner, 1978). Tethered systems typically involved a pulley attached to weights (Costill, 1966) which allowed swimmers to exercise against a progressive load until unable to keep the load elevated (Magel and Faulkner, 1967). However, due to their restrictive nature tethered systems were often criticised for interfering with the technical performance of the swimmers. Jensen and Tihanyi (1978) reported increased drag forces at the upper arm compared with free swimming and Mosterd and Jonbloed (1964) argued that while the swimmer attempts to push forward during free swimming the reverse was true of tethered swimming. Later Cazorla *et al.* (1982) complained that swimmers were forced into oblique positions when heavy loads were used making it difficult for them to find support in turbulent flow. Subsequently it was acknowledged that biomechanical data from tethered swimming was not particularly worthwhile with regard to its application to free swimming, although this was not necessarily the case for applied physiological research (Holmer, 1974).

In an attempt to more closely mimic the conditions of free swimming scientists began to improve the level of sophistication of pacing systems. During the 1970 s systems became increasingly elaborate with controllable measurement - equipment platforms (di Prampero *et al.*, 1974; van Manen and Rijken, 1975) being used to either tow or pace swimmers. However, the emergence of the

swimming flume in Stockholm (1968) represents the greatest level of sophistication in swimming ergometry, even to the present day (Astrand and Englesson, 1972, Holmer and Hagland, 1978).

The original swimming flume consisted of a basin around which water was circulated and controlled by propeller pumps allowing a range of swimming velocities (0-2 $m.s^{-1}$) to be simulated with remarkable reproducibility (0.02 $m.s^{-1}$). Guide vanes created uniform, near-laminar flow in the central part of the water where the swimmer swam and a large side window allowed motion analysis to be undertaken both above and below the water line. Flumes have since been built in a number of countries and been acknowledged as very useful research tools particularly in the study of the body's haemodynamic and metabolic responses during swimming (Holmer and Haglund, 1978).

Maximum oxygen uptake findings in flume studies were found to be similar those of free swimming (Bonen *et al.*, 1980) which seemed to validate their use in terms of applying findings to free swimming, however they proved to be extremely expensive to build and so have not been accessible to most researchers. Also they are not universally popular amongst swimmers because as with tethered systems the stroke technique has to be adjusted. Consequently biomechanical findings generated in swimming flumes may, rather like tethered systems have limited application to free swimming.

One advantage of using tethered systems and swimming flumes was that ventilatory measurements could be made relatively easily where as during free swimming large gas collection bags (or even meteorlogical balloons !) had to be carried by a technician or placed on a portable cart (Anderson, 1960; McArdle *et al.*, 1971; Lavoie *et al.*, 1981b; Cazorla *et al.*, 1982). Despite these shortcomings similar values for $\dot{V}O_2$ peak were observed from portable gas collection bags compared with tethered and flume swimming (Bonnen *et al.*, 1980). However, the cumbersome nature of the mouthpieces, valves and tubing used in portable gas collection bag studies meant that it was debatable whether maximal free swimming speeds were actually being achieved (Montpetit *et al.*, 1981) as the economy of the swimmer was adversely affected (Leger *et al.*, 1980). These shortcomings cast doubt upon whether maximal ventilatory values were being obtained and likewise whether the similar values obtained in tethered and flume swimming studies had been maximal.

A further limitation of the portable gas collection bag method was that meaningful biomechanical research could not be undertaken at high swimming speeds, as the cumbersome apparatus was clearly interfering with the technical performance of the swimmer. In short the ecological validity of the biomechanical and ventilatory data obtained from supposedly maximal efforts during encumbered free swimming, tethered swimming and flume swimming was thought questionable.

However in 1973 another methodological advance was reported which allowed for the unemcumbered measurement of oxygen uptake. Di Prampero *et al.* (1973) observed that following supra-maximal exercise a time lag of 15 seconds existed before the onset of the oxygen recovery kinetics. Applying this knowledge Di Prampero *et al.* (1976) reported a backward extrapolation technique which precisely estimated the oxygen uptake of the subject from the collection of four, 20 second samples of expired air taken immediately following exercise. Leger *et al.* (1980) then applied this technique to swimming using a least-squares regression technique to allow the $\dot{V}O_2$ at time zero to be calculated from a single exponential regression curve of the four data points. Later Lavoie *et al.* (1983) further adapted the method by only collecting a single 20 second sample. In 1985, Costill *et al.* reported a series of experiments which used this shortened method following sub-maximal and maximal swimming efforts of 5-7 minutes duration to estimate oxygen uptake. Using linear regression they produced a predictive equation:

$y = 0.916\ x + 0.426$

where:

y = the predicted $\dot{V}O_{2STPD}$

x = the actual $\dot{V}O_{2STPD}$ measured from the 20 second post exercise expired air collection.

Sleivert and Mackinnon (1991) have since shown that this technique (linear extrapolation of one 20 second recovery sample) explains a similar amount of variation compared with a monoexponential technique using 5 recovery samples ($r^2 = 0.83$ vs $r^2 = 0.85$ respectively).

The validity of this technique was established when Montpetit *et al.* (1981) showed that peak oxygen uptake during free swimming was actually greater in good swimmers when they were swimming rather than running. This was important because it demonstrated that those studies which had previously shown $\dot{V}O_2$ max values to be lower during swimming than running (Dixon and Faulkner, 1971; Holmer 1974a, 1974b) or cycling (Holmer, 1972) may have been limited by the methodological techniques used to collect the swimming data. This finding clearly demonstrated the importance of evaluating swimmers during free unemcumbered swimming. The development of portable heart rate monitors and lactate analysers has meant that physiologists have since been increasingly able to undertake free swimming research in this way over the last 20 years.

The review so far has indicated how measurement methods have become increasingly sophisticated to the extent that establishing the metabolic and biomechanical characteristics of free swimming concurrently has become increasingly possible. However, this would assume that the management of pacing control had also been enhanced since the studies of the 1970 s. In fact a

review of the literature would suggest that even today there is not widespread acceptance of a particular pacing method during free swimming (Table 2.1).

Table 2.1 - Studies which have or have not reported the use of external pacing methods in swimming investigations

Author(s)	Date	Title	External pacing evident?
Colman *et al.*	1998	A comparison of the intracyclic velocity variation in breaststroke swimmers with flat and undulating styles	No
Peyrebryne *et al.*	1998	The effects of oral creatine supplementation on performance in single and repeated sprint swimming	No
Berger *et al.*	1997	Technique and energy losses in front crawl swimming	No
Stewart *et al.*	1997	Swimmers compliance with training prescription	No
Kolmogorov *et al.*	1997	Hydro-dynamic characteristics of competitive swimmers of different gender and performance levels	No
Mujika *et al.*	1996a	Creatine supplementation does not improve sprint performance in competitive swimmers	No
Mujika *et al.*	1996b	Hormonal responses to training and its tapering off in competitive swimmers: relationships with performance	No
Filaire *et al.*	1996	Saliva cortisol, physical exerciseand training: Influeb=nces of swimming and handball on cortisol concentrations in women.	No
Kapus *et al.*	1996	Relationship between blood lacate concentration and swimming success	No
Pelayo *et al.*	1996	Stroking characteristics in freestyle swimming and relationships with anthropometric characteristics	No
Tyndall *et al.*	1996	Cortisol, testosterone amnd insulin action during intense swimming training in humans	No
Zampora *et al.*	1996	Effects of underwater torque on the energy cost, drag and efficiency of front crawl swimming.	No
Mujika *et al.*	1995	Effects of training on performance in competitive swimming	Yes
Lowenstyn *et al.*	1994	Differences in peak blood lactate concentration in long course and short course swimming	No
Chatard *et al.*	1995	A comparison between swimmers and triathletes	No
Lin *et al.*	1993	Body roll and hand path in freestyle swimming: an experimental study	No
Duche *et al.*	1993	Analysis of performance of pre pubertal swimmers assessed from anthropometric bio-energetic characteristics	No
Ueda *et al.*	1993	Contribution of differential ratings of perceived exertion to overall exertion in women whilst swimming	No
Chatard *et al.*	1991	Energy cost of front crawl swimming in women	Yes
Ueda *et al.*	1991	Validity of heart rate and ratings of perceived exertion as indices of exercise intensity in a group of chidren while swimming	No
Chatard *et al.*	1990	Analysis of determinants of swimming economy in front crawl swimming	Yes
Deubern *et al.*	1990	Blood lacate responses in older swimmers during action and passive recovery following maximal sprint swimming	Yes
Haub *et al.*	1990	The effect of growth on drag in young swimmers	No
Town *et al.*	1990	Metabolic responses to contrrolled frequency breathing in competitive swimmers	No
Tharp *et al.*	1990	Reduction of saliva immunoglobulin levels by swimming training	Yes
Neuffer *et al.*	1987	Effect of reduced training on muscular strength of competitive swimmers	Yes
Gullestrand *et al.*	1987	Heart rate and blood lactate response to short intermittent work at race pace in highly trained swimmers	No
Grimston *et al.*	1986	Relationships among anthropometric stroking characteristics of college swimmers	No
Costill *et al.*	1985	Metabolic characteristics of skeletal muscle during detraining from competitive swimming	Yes
Hsieh *et al.*	1983	The acute effects of controlled breathing swimming on glycolytic parameters	No
McMurray *et al.*	1983	Plasma volume changes during submaximal swimming	No
Lavoie *et al.*	1982	Blood metabolites during prolonged exercise in swimming and leg cycling	Yes
Hichson *et al.*	1979	Effects of training on hormonal responses to exercise in competitive swimmers	No

This lack of a criterion pacing method means that the degree of precision and reliability of pacing during free swimming has rarely been investigated. Consequently many scientific investigations have either continued to rely upon the swimmer's own pace judgement or have avoided free swimming altogether and accepted the biomechanical limitations that exist with tethered sytems and swimming flumes.

A number of investigations have attempted to use pacing signals to control the swimming speed of their subjects. For example Lavoie *et al.* (1985) used audible pacing signals to pace swimmers during a multistage incremental swimming test designed to predict $\dot{V}O_2$. However, despite acceptable accuracy being reported by the authors the test has not been as widely accepted as the running counterpart (the Multistage Fitness test). A reason for this might be that it has been difficult for investigators to ensure that swimmers are able to hear an audible signal both above and below the water. Indeed the signal is usually given above the water (where the sound system resides) and so the swimmer is often unable to hear a signal when completing the underwater element of a stroke cycle. Montpetit *et al.* (1983) have also reported that audible signals are difficult to hear underwater if they are not situated in close proximity to the swimmer. They were also concerned that audible signals were distracting to swimmers nearby making it difficult to pace a number of swimmers at individualised paces at the same time.

Light sequencing has also been used to control the pace of swimmers although this method produces similar problems to auditory cues as the placement of the lights is generally either only above or only below the water line. Consequently it is difficult for the swimmer to observe them at all times during a stroke cycle and they can also be distracting to other swimmers. Placing the pacing lights on the poolside (Sano *et al.*, 1990) can also be problematic as this can result in unwanted lateral movements. Perhaps because of this the pool floor seems to be the more popular site for the placement of underwater lighting systems (Hallowell, 1983; Costill *et al.*, 1985; D'Aquisto *et al.*,1988; Keskinen, 1997a, 1997b).

A particular concern with using underwater lights is that swimmers have to be sure about where a particular body segment should be in relation to a lamp when it illuminates. Any misunderstanding can cause a relatively large error in pacing due to the slow velocities produced during swimming. In fact D'Aquisto *et al.* (1988) reported that their subjects were unable to maintain acceptable pacing precision using underwater pacing lights. Although, Keskinen (1997 a, 1997b) recently reported that both experienced and less experienced swimmers were able to reproduce pacing precisely using the Protom light sequencing system with a margin of error approximating to 1 second when swimming at 1.4 $m.s^{-1}$. These latter results may have been achieved because clear guidance was given to the subjects with regard to their body positioning and the lamps.

In the mid 1990 s another pacing system became available to scientists which had been developed by Patrick Miley, a part time swimming coach. The Aquapacer™ pacing system was designed to overcome the limitations of the other methods by placing an audible signal permanently in close proximity to the swimmer's ear. This was achieved by placing a sounding unit under the swimmer's cap (or more recently on the goggle strap) just behind or above the ear. The proximity of the sounding unit means the bleep can be heard whether the swimmer is above or below water and does not distract other swimmers. The device was originally conceived as a training aid to improve pacing awareness and as a way of teaching swimmers to learn to swim at an appropriate stroke rate when undertaking race pace training. Paul Palmer reputedly used the Aquapacer™ to develop a reproducible stroke rate in training prior to winning a silver medal in the 400 m freestyle at the 1996 Olympic Games.

The system is comprised of a hand held programming unit which is interfaced with a "coin size" programmable sounding unit. Once programmed the programming unit can transfer a regular pacing frequency to the sounding unit accurate to 0.01 s. By instructing the swimmer to ensure that a bleep noise from the sounding unit coincides with reaching a marker in the pool the scientist is able to control pacing. Using a prototype of the Aquapacer™ Martin and Thompson (1999) observed that extremely precise pacing was demonstrated by University team swimmers during a sub-maximal set of 10 x 100 m freestyle repetitions in morning and evening trials. Martin and

Thompson (2000) have since reported that pacing was also reproducible over 3 morning trials (95 % Limits of Agreement - 84.43 ± 1.7 s) and evening trials (95 % Limits of Agreement - 84.13 ± 1.02 s). In this last study, the pace of the trials was equivalent to that which would result in maximal lactate steady state, according to a calculation reported by Olbrecht *et al.* (1985) which utilised the distance covered by subjects over a timed 30 minute swim (T-30). Therefore the precision and reproducibility of pacing utilising the Aquapacer™ has still to be investigated empirically during high intensity swimming and for other strokes. Although it seems likely that a pacing method now exists which overcomes the concerns expressed by Holmer (1974) with regard to the lack of control of pacing in free swimming at different work rates.

To conclude this section there have been a number of important developments in methods and equipment in recent years. The backward extrapolation technique allows the swimmer's oxygen uptake to be determined following unemcumbered free swimming which means that the swimmer can proceed without being technically impeded and subsequently will be able to achieve maximal swimming speeds in experiments. This means simulated competition responses can be evaluated. Secondly the Aquapacer™ pacing system has the potential to elicit pecise pacing at high swimming speeds which will allow the effects of changes within pace to be evaluated effectively. To date these advances have not been used to investigate the kinematic and metabolic characteristics of the breaststroke.

2.3 The reliability of metabolic and kinematic variables during swimming

It has been suggested that the total variance in the metabolic response to exercise may be explained by:

i) technical variance (within subject or intraindividual variance due to the pacing method or measuring apparatus),

ii) biological variance (within subject or intraindividual variance), and

iii) true score variance (between subject or interindividual variance).

Adapted from Becque et al. (1993)

Holmer (1974) expressed concern as to whether measurement methods of the time were precise and reliable enough to estimate the total variance in metabolic response during swimming. Interestingly over the last 25 years there has been little research which has reported such data during free swimming probably because of the lack of a criterion pacing method. However, it has also been recently suggested that exercise physiology investigations in general do not habitually report the consistency of their measurements or arguably use inappropriate statistical techniques when doing so (Atkinson and Nevill, 1998). Subsequently it remains difficult for scientists to conclude whether the findings reported in free swimming studies are an artefact of measurement error or inter-daily biological variation. For example many studies have reported heart rate and blood lactate responses during swimming yet Lavoie and Montpetit (1986) have suggested that large variations may exist in these variables at a given

swimming speed which precludes their use for the monitoring of the training status of swimmers.

A few studies in the literature have attempted to establish within subject variability during sub-maximal and maximal exercise. Within subject variability for heart rate expressed as a percentage of mean exercise response has been shown to be < 5 % during sub-maximal exercise for a number of exercise modalities (running - Taylor, 1944; cycling - Becque *et al.*, 1993; swimming - Martin and Thompson, 2000) and may be reduced during maximal exercise (1.6 %, Taylor, 1944). Cellini *et al.* (1986) have also reported a high correlation between heart rate and swimming velocity cubed ($r = 0.99$) suggesting that relative stability exists in heart rate response (Baumgarter, 1989).

Within subject variability has been reported to be similar for ventilatory measurements during sub-maximal exercise (running - Taylor, 1944; Henry, 1951; Morgan *et al.*, 1991; Williams *et al.*, 1991; cycling - Coggan and Costill, 1983; Armstrong and Costill, 1985). However, Martin and Thompson (2000) have observed greater variability in ventilatory findings in swimmers using the backward extrapolation technique (Di Prampero, 1976), although relative reliability has been previously demonstrated for swimmers using repeated measurements with this technique (Montpetit *et al.*, 1981; Costill *et al.*, 1985). Finally, Martin and Thompson (2000) have reported evidence of large random

error in capillary blood lactate measurements at sub-maximal swimming speeds.

Since simple kinematic variables (stroke rate, stroke length) began to be researched in swimming (Curry, 1977; Craig *et al.*, 1979) there do not appear to have been any studies reporting the reliability of these variables during free swimming, until recently. Martin and Thompson (2000) reported that stroke rate measurements calculated from hand timing appear to be highly reproducible between trials. Stroke count was also concluded to demonstrate acceptable reproducibility although a greater degree of random error was apparent. This was thought to be due to the subjective nature of the determination of this measurement in their study.

It would appear that in recent years there has been continual development in the pacing control of free swimming and in the methods of measurement with regard to physiological variables. The recent innovation of the Aquapacer™ now provides the contemporary sports scientist with an oppurtunity to undertake research into the reliability of the metabolic and kinematic responses during breaststroke swimming. Additionally, the accuracy and reproducibility of pacing observed by Martin and Thompson (1999, 2000) would suggest that subtle manipulations in pacing might now be possible which would allow a much greater understanding of how breaststroke swimmers respond to changes in swimming velocity at racing pace, where the precision of pacing would be a precondition in order for meaningful scientific research to take place.

2.4 The physical characteristics of breaststroke swimmers

The age range demonstrated by competitive swimmers has tended to show less variability in the past than for other sports with the mean age of Olympic participants remaining relatively unchanged at approximately 19-20 years old for males and 17 years old for females (Khosla, 1984). Tanner (1964) identified that in certain events height and body mass may be related to performance and that definite body characteristics are identifiable in some successful Olympic swimmers. There has also been some evidence that finalists at the 1976 and 1980 Olympics were taller and heavier than non-finalists (Khosla, 1984). It has been suggested that tallness allows swimmers to:

i) swim at the same speed as a shorter individual with less power (Orvel *et al.*, 1981),

ii) cover less race distance by virue of the standing dive start, turn and finish with an outstretched hand, and to

iii) cover a greater distance per stroke.

However height has not always been found to correlate well with performance (Katch and Michael, 1973; Smith, 1978; Siders *et al.*, 1993; Pelayo *et al.*, 1996).

The tallest swimmers have been observed to be the backstroke and sprint specialists with sprint butterfly swimmers being the shortest (Spurgeon and Sargent, 1978; Spurgeon and Giesse, 1984). The physique of breaststroke and butterfly specialists has been shown to be less predictable than for crawl swimmers (Salvadori, 1983). It seems that height offers certain advantages to swimmers but is less highly related to performance in breaststroke events than in the crawl sprint and backstroke events.

Aswell as being taller than reference populations swimmers have been observed to possess a greater arm span to height ratio (Lavoie and Montpetit, 1986). A number of investigators have also identified that certain characteristics such as body size, body form, the surface area of propulsive body parts and floating capacity all have an important role in the determination of performance and stroke mechanics (Touissant *et al.*, 1983; Grimston and Hay, 1986). In slight contradiction to this Pelayo *et al.* (1996) failed to observe significant correlations between performance and a number of anthropometric variables (height, span, age, weight, foot size) in males, although some significant correlations were observed for females. However the authors felt this was due to the female group being more heterogeneous in terms of performance. On balance simple anthropometric variables may be non-discriminatory in the achievement of swimming performance.

Finally, the body composition of swimmers particularly elite ones tends toward leanness (Faulkner, 1968; Sprynarova and Parizkova, 1971; Novak *et al.*, 1978,). Males typically demonstrate values of between 5 - 10 % body fat whereas females range between 14 - 19 % (Lavoie and Montpetit, 1986).

2.5 The physiology of breaststroke swimming

A severe strain is imposed upon the breaststroke swimmer's ability to ventilate by limited oppurtunities to breath and the pressure of the surrounding water on the thoracic cavity. Airway resistance has been found to be much greater in breaststroke swimming than on land such that a 20-30 % decrease in lung compliance has been observed, although elastic resistance remains unchanged (Deroanne *et al.*, 1971). In swimming generally, vital capacity has been found to be reduced by approximately 10 %, but tidal volumes have been shown to increase to a greater percentage of total lung capacity than is found on land (Hong *et al.*, 1969; von Doblen and Holmer, 1974, Town and Vanness, 1990) probably to compensate for the restricted breathing frequency. Therefore it has been suggested that alveolar gas exchange may be affected by the synchronisation of the respiration rate with the stroke rate, the forced inspiratory phase against water pressure and the forced expiration into water (Holmer, 1979).

Despite these concerns it has been proposed that breaststroke swimmers display normal ventilation rather than hypoventilation during swimming, although in a

study of elite swimmers maximal pulmonary ventilation was shown to be lower than in maximal running (Holmer *et al.*, 1974a) undoubtably due to the restricted oppurtunities to breath (Town and Vanesse, 1990). However, PO_2, PCO_2 and pH in arterial and venous blood have been found to be remarkably similar during sub-maximal and maximal breaststroke swimming and running, despite the alveolar-arterial O_2 pressure gradient being lower in maximal breaststroke swimming (Holmer *et al.*, 1974b). Therefore surface swimming in comparison with running may limit ventilatory volumes but does not necessarily alter blood gas tensions and pH. The actual mechanics of respiration in terms of gas pressures remaining largely unaffected during surface swimming (Hong *et al.*, 1969). It appears that the lower total ventilation observed in breaststroke swimming still appears to provide an adequate alveolar ventilation to allow for the normal saturation of arterial blood, however the reduced ventilation would appear to have implications for aerobic capacity and the subsequent aerobic energy contribution possible during competitive breaststroke swimming.

Cardiovascular response has been found to be similar in breaststroke swimming and running (Holmer *et al.*, 1974b). Although immersion studies have indicated that the stroke volume is greater in swimming due to an improved diastolic filling in the supine position (Arborelius *et al.*, 1972; Lange, 1974) which results in a reduced heart rate response. Evidence to support this has come from the observation of a reduced heart rate response during swimming when sub-maximal swimming responses were compared with cycling

(Goodwin and Cumming, 1966; Magel, 1971; McArdle, Glaser and Magel, 1971), although contradictorily Holmer *et al.* 1974b have also observed a similar heart rate response for a given oxygen uptake when comparing breaststroke swimming and running.

Maximal cardiac output has been reported to be lower during maximal swimming compared with running due to a decreased heart rate response (Holmer *et al.*, 1974b). The cause of the reduction in maximal heart rate during swimming is at present not fully understood although it has been attributed to hydrostatic and gravitational effects or thermoregulatory demands (eg. water temperature), because the heart rate during maximal work appears to be dependent on internal body temperature and therefore may decrease following exposure to cold water due to convective heat loss (Nadel *et al.*, 1974; McArdle *et al.*, 1976). In such situations sub-maximal heart rates have also been shown to decrease for a given oxygen uptake (Holmer and Bergh, 1974; Galbo *et al.*, 1979). These observations may explain why optimal performances are observed in water temperatures of 28^0C to 30^0C, as at these water temperatures a swimmer's high work rate may maintain the body temperature over the relatively short duration of the race (Nadel *et al.*, 1974).

Interestingly, a decreased maximal cardiac output compared with running has not always been observed during swimming (Dixon and Faulkner, 1971). This is possibly because the increase in stroke volume observed during swimming

might counteract the small decrease in heart rate and so maintain the cardiac output.

An alternative explanation for a reduction in heart rate during surface swimming is that swimmers may utilise less musculature compared with runners and cyclists (Holmer, 1979), although the greater emphasis on leg propulsion in the breaststroke may partially contradict this argument. Finally, the increased stroke volume observed in swimmers suggests that a longer ventricular filling time would be required which might require a slight reduction in the maximal heart rate. This argument has also been put forward to explain the reduction in the maximum heart rate sometimes observed with endurance trained runners (Willmore and Costill, 1994) where a coincidental expansion of the blood volume has occurred.

2.6 The unique characteristics of the breaststroke

Drag varies as a function of the stroke cycle, body configurations and relative velocity of the body segments in the surrounding water (Holmer, 1979). Using physiological measurements scientists have attempted to quantify active drag during steady state swimming (Holmer, 1974a; Di Prampero *et al.*, 1974; Clarys, 1976) in order to determine mechanical work efficiency (Holmer, 1974a; Pendergast *et al.*, 1978a). Using this technique it has been estimated that the efficiency of the breaststroke is only 5-6 % (Holmer, 1974a), three times less than the frontcrawl (Pendergast *et al.*, 1978a). It has also been

calculated that elite breaststrokers expend 1.4 times as much energy as frontcrawl swimmers (Lavoie and Montpetit, 1986) probably because the leg kick has a much greater influence on swimming speed during breaststroke swimming compared with freestyle swimming (Holmer, 1974b). However the derivation of these estimations of efficiency have depended upon the assumption that intra-stroke cycle velocity fluctuations are small. Therefore it is possible that the breaststroke may be even less efficient than has been calculated, as it is has been shown that during a breaststroke stroke cycle large intracycle velocity fluctuations occur, which are greater than for other strokes (Miyashita, 1971; Craig and Pendergast, 1979; Holmer, 1979; Schleihauf, 1979; D'Aquisto *et al.*, 1988)

It is thought that the rate of energy expenditure during swimming increases exponentially with velocity irrespective of stroke primarily due to an increase in drag (Pendergast *et al.*, 1978b). This means that large velocity fluctuations within a stroke cycle, such as occur in the breaststroke are even more problematic at higher velocities. Consequently, the breaststroke swimmer needs to be aware of pacing a race correctly as the breaststroke is prone to larger increases in energy expenditure for a given increase in velocity.

The large accelerations that cause large velocity fluctuations within the breaststroke stroke cycle have been shown to occur during the propulsive phases of the arm pull and leg kick (Holmer, 1979b; D'Aquisto *et al*, 1988). The leg kick has been found to have a greater energy cost than that of the crawl

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strokes while the reverse is true of the arm pull (Holmer, 1974a, 1974b). However, taken as a whole the breaststroke has a much greater energy cost than the crawl strokes. Indeed these findings have been used to suggest that leg kicking is performance limiting in distance events.

Fig 2.1 A graphic illustration of the velocity curve for a world class female breaststroke swimmer (J. Hau, Costill *et al.*, 1987)

As a consequence of the leg kick, the breaststroke is always going to be less efficient than the crawl strokes, given that the extent of the acceleration (or velocity fluctuation) with in a stroke cycle is an indication of swimming efficiency (Kornecki and Bober, 1978; Holmer, 1979b). However it remains

unclear whether the breaststroke is less economical than the butterfly (Lavoie and Montpetit, 1986).

As well as the chosen stroke, drag, technique and training background are also thought to influence the energy cost of swimming (Holmer, 1972, 1974c; di Prampero *et al.*, 1974; Pendergast *et al.*, 1977). In fact it has been suggested that the technical ability of the swimmer at each stroke is of greater importance than any systematic stroke differences (Pendergast *et al.*, 1978c) in terms of oxygen uptake per unit of distance travelled. Therefore caution must be exercised when comparing data from different strokes where swimmers are of different standards.

A further concern particular to breaststroke research is that since 1987 a rule change has allowed the hands of breaststroke swimmers to break the surface of the water and for the head to be submerged during a stroke cycle. As a consequence two distinct swimming styles have developed in international swimming: flat and undulating. The development of the undulating style has been an attempt by swimmers to reduce the large intra-cyclic variation in the horizontal velocity of the body's centre of mass, as it is thought that this requires the swimmer to overcome greater inertia and higher hydrodynamic resistances than if less variation were present. Indeed the ability of a swimmer to maintain a high swimming speed is thought by some researchers to be more highly related to avoiding decreases in the velocities of some of the phases of the stroke cycle rather than to the strength of the swimmer and the production

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of greater momentary maximal velocities (Costill *et al.*, 1987; Bober and Czabanski., 1995).

Figure 2.2 A comparison of the swimming velocity curves for two female breaststroke swimmers (Costill *et al.*, 1978)

Colman *et al.* (1998) have observed that less variation is apparent in the horizontal velocity of the body's centre of mass during the undulating style and attributed this to the movements above the water surface creating a momentum

of energy transfer. The fast backward trunk rotation indicative of this style generates resistance which provides a platform for when the shoulder girdle and upper arm move quickly forward, followed by a fast forward movement of the upper trunk and head rotation. This forward trunk rotation provides a forward acceleration of the body parts, as at this point they are out of the water and subsequently an additional propulsive influence is provided within the stroke cycle. However at present there is no definitive evidence that the undulating style is the preferred choice of style in competitive swimming.

Sections 2.5 and 2.6 have highlighted that the breaststroke is increasingly mechanically inefficient at higher swimming speeds. Yet fundamentally there have been few studies which have examined the breaststroke in terms of the additional energy cost commensurate with increases in swimming speed.

2.7 Aerobic and anaerobic energy contribution during competitive breaststroke swimming

The aerobic and anaerobic contribution to the continual resynthesis of ATP during maximal swimming has been well documented (Hermansen and Karlsson, 1967; Hermanssen, 1969; Houston, 1978; Troup, 1984; Maglischo, 1993). In maximal efforts exceeding 3 minutes it is estimated that a 60 : 40 aerobic anaerobic contribution occurs, with the anaerobic contribution increasing the shorter the event duration (Houston, 1978; Maglischo, 1993). Troup (1984) has estimated that the anaerobic contribution for particular

competitive events may in fact be increasing because swimming speeds are increasing. Consequently a high anaerobic tolerance is thought to be an important capacity for competitive swimmers (Town and Vanness., 1990).

Oxygen debt and peak post exercise blood lactate values have traditionally been used to estimate that a high anaerobic ATP contribution is required by swimmers in 100 m and 200 m events (Gollnick and Hermanssen, 1973; di Prampero *et al.*, 1978; Mader, *et al.*, 1978). Maximal oxygen debt has been estimated to be as high as 15-18 l O_2 in better swimmers (Hermanssen and Karlssen, 1967; Hermanssen, 1969) while post exercise blood plasma lactate values as high as 15-18 mM have also been reported (Madsen and Lohberg, 1987; Sawka *et al.*, 1979). More recently Thompson (1998) observed whole blood lactate values of 9.5 ± 1.3 mM and 10.7 ± 1.4 mM in national standard 100 m and 200 m breaststroke swimmers and Lowenstyn *et al.* (1994) has reported similar values for 200 m freestyle swimmers. Further evidence that it is the anaerobic capacity that predominates in competitive swimming has been inferred by Hermanssen and Karlssen (1967). They found that maximal oxygen debt and peak blood lactate values increased in swimmers who trained over a competitive season, while maximal oxygen uptake did not. Maglischo (1982) has also presented evidence that the lactate tolerance of swimmers increased during training for 100 yd and 200 yd events. An increased emphasis on anaerobic training closer to important competitions perhaps explains these findings as the recruitment and hence adaptation of certain metabolic pathways and fibre types is dependent on the duration, type and intensity of muscular

work being undertaken (Holmer, 1979). Olbrecht (2000) has also observed that improvements in the anaerobic capacity of swimmers over the course of a season coincided with a decrease in their aerobic swimming economy which supports the idea that swimmers employ a change of emphasis toward anaerobic development closer to important competitions.

It has been shown that glycogen depletion begins in Type 11b fibres before progressing to Type IIa then Type I fibres during supra-maximal dynamic exercise (Secher and Nygaard, 1976; Thomson *et al.*, 1978) with the reverse order being observed during sub-maximal dynamic work (Thomsen *et al.*, 1978). However the degree of involvement of the different muscle fibre types in 100 m and 200 m swimming events is complicated by the fact that there is a mixed contribution of fibre types being recruited particularly in 200 m events. It has been suggested that in 50 m to 200 m swimming events Type 11b fibres are utilised along with Type 11a fibres to a greater degree than Type 1 fibres (Costill, 1992). The depletion of the Type 11 fibres of glycogen before Type 1 fibres during 25-100 m high intensity training efforts supports this view (Costill, 1978; Houston, 1978) and the view presented earlier that a greater proportion of energy may be being contributed from anaerobic metabolism in the shorter swimming events (Maglischo, 1993).

It has also been suggested that glycogen depletion may have been observed in swimmers who completed a number of competitive swims over a few days. Sawka *et al.* (1979) found reduced blood lactate concentrations when freestyle

swimmers participated in more than 3 freestyle races during a single championship while Thompson (1998) observed that blood lactate concentrations were not significantly elevated following significantly faster final swims compared with heat swims. In both studies the authors attributed their findings to glycogen depletion being present in fast twitch fibres.

However the contribution of Type 1 fibres in competitive swimming events should not be understated because it has been reported that Type 1 fibres predominate in both the arm and leg musculature of swimmers (Gollnick *et al.*, 1972; Nygaard and Nielson, 1978; Houston *et al.*, 1981) although this is not always the case (Costill *et al.*, 1978; Lavoie *et al.*, 1981a). Type 1 deltoidius muscle fibres in elite swimmers have also been shown to be greater in size than Type 11 fibres (Costill *et al.*, 1992) presumably due to the large amount of aerobic training traditionally undertaken in swimming. This would also suggest that a well developed aerobic energy transfer system is utilised during competitive swimming races. The finding that swimmers are also able to attain anaerobic thresholds close to 90 % $\dot{V}O_2$ max clearly shows an extreme level of aerobic conditioning (Smith *et al.*, 1984), although sprinters may often demonstrate lower anaerobic thresholds (Treffene, 1979a).

At present it is unclear if the aerobic energy system is maximally stressed during the course of breaststroke races. Therefore it is not known whether it is additional anaerobic energy contribution alone or a mixture of additional

anaerobic and aerobic energy contribution which allows a swimmer to achieve faster swimming speeds between heat and final performances in competition.

2.8 Pre-requisites of competitive breaststroke performance

A small number of early swimming investigations have attempted to improve the understanding of the pre-requisites for swimming performance by predicting performance capacity from physical and physiological measurements (Charbonnier *et al.*, 1975; Sprague, 1976; Klissouras and Sinning, 1978) and performance tests (Jackson *et al.*, 1975). However the relationship between sub-maximal blood lactate values and velocity has not been investigated in swimming to the extent it has been in running. This may seem surprising as sub-maximal blood lactate values have been shown to explain more of the variance in running performance than maximum oxygen uptake in homogeneous and heterogeneous groups (Yoshida *et al.*, 1990, 1993; Sjodin *et al.*, 1982). Also the velocity at which certain blood lactate measurements (2 mM, 4 mM, lactate threshold, lactate turn point) are elicited during incremental laboratory treadmill tests have been reported to correlate significantly with a range of endurance running distances (Morgan *et al.*, 1989; Ramsbottom *et al.*, 1988, 1992 (5 km); Noakes *et al.*, 1990; Spurway *et al.*,1992; Jones., 1994 (8 km); Grant *et al.*, 1997 (3 km)). However, despite these findings the relationship between blood lactate measures and performance in swimming is not so well documented. In fact only a few studies have attempted to investigate this relationship.

Some 20 years ago Mader *et al.* (1978) began using post-exercise blood lactate samples elicited from a sub-maximal swim (400 m at 85 % of maximal speed) and a maximal 100 m swim, 30 minutes apart, to predict the potential swimming velocity of a swimmer and to monitor physiological training status. This method was latterly adopted by Elliot and Haber (1983) to predict 100 m breaststroke performance using linear regression to extrapolate to a maximal blood lactate concentration. The reason why sub-maximal lactate values have not been investigated as thoroughly in swimming as in running is possibly because 80 % of competitive swimming events involve racing over distances of 200 m or less (Troup and Trappe, 1994) which at national level take under 3 minutes to complete. Consequently it would be expected that the significant anaerobic energy contribution in such events would put into question the meaningfulness of sub-maximal endurance markers.

A few investigations have however studied the relationship between the heart rate and blood lactate response with swimming speed during incremental tests in order to monitor and evaluate training (Treffene, 1979b; Maglischo *et al.*, 1984; Sharp *et al.*, 1984). These studies have found that heart rate values increased for a blood lactate concentration of 4 mM over a season of training (Maglischo *et al.*, 1982). Costill (1992) subsequently proposed that decreases in sub-maximal blood lactate concentrations following a standard swim of 200 m - 400 m at 80 - 100 % $\dot{V}O_2$ max indicated that certain training adaptations will have occurred relating to the swimmer:

i) becoming more efficient,

ii) possessing an improved aerobic power,

iii) producing less lactate and/or being able to remove lactate faster.

At present no studies to the author's knowledge have reported a reduction in post exercise sub-maximal blood lactate concentrations during a constant load or incremental protocol specifically for the breaststroke.

Costill (1992) has questioned whether swimmers are able to demonstrate a continual decline in post-exercise blood lactate concentrations. He has suggested that improvements during the first 6 - 10 weeks of predominantly aerobic training are possible, but additional decrements in lactate concentration for a given swimming velocity may not occur despite many more weeks of training.

However, Touretski (1993) has reported longitudinal improvements in swimming velocities corresponding to blood lactate concentrations of 4 mM (v-4 mM) and 8 mM (v-8 mM) for Alexandre Popov, during preparations for his gold medal winning performances in the 1992 Olympic Games. Touretski deduced from this that the development of aerobic capacity was important even for shorter distance events. Madsen and Lohberg (1987) have also suggested that an improvement in competition velocity can be more efficiently achieved by improving the v-4 mM than by improving the lactic acid production

capacity. For this reason v-4 mM has also been used as a guide for classifying the training of the competitive swimmer (Olbrecht *et al.*, 1985; Madsen and Lohberg, 1987; Pyne, 1989) because it estimates the upper limit of aerobic capacity beyond which there is a rapid increase in lactic acid appearance (Mader *et al.*, 1976) and a subsequent accumulation of hydrogen ions within the muscle.

To date there remains no direct evidence that an adjustment in lactate kinetics during sub-maximal exercise is meaningful in relation to the prediction of a competitive breaststroke performance. Although Kapus *et al.* (1998) have reported significant correlations between v-4 mM elicited during an incremental freestyle test and the 100 m and 200 m breaststroke race performances of 6 international swimmers. However, because they did not use a breaststroke incremental protocol their findings may only indicate that the better breaststroke swimmers in their study coincidentally happened to be more proficient freestyle swimmers.

Treffene (1978b) has argued that any increase in the swimming velocity that corresponds with attaining a maximum heart rate (v-MHR) during an incremental swimming protocol (3-5 x 200 m) indictates that there is the potential for an improved race performance. An increase in the v-MHR over a period of training would be preceded by a lowered heart rate at a given sub-maximal speed. Improvements in v-MHR therefore suggest an improved aerobic work capacity at a range of speeds which Treffene felt would lead to an

improvement in race performance. In 1982, Treffene constructed a number of predictive equations which required only the knowledge of a swimmer's v-MHR and 100 m performance time to predict freestyle times for 200 m - 1,500 m swimming events. The rationale for measuring changes in v-MHR during 100 m and 200 m breaststroke swimming would presumably be the same but to date no data has been reported.

The use of sub-maximal indirect tests to predict $\dot{V}O_2$ max from the $\dot{V}O_2$ - heart rate relationship have tended to be avoided in swimming possibly due to the large inter-individual variations reported in the energy cost / oxygen uptake values in some studies (Lavoie and Montpetit, 1986). Although, Lavoie *et al.* (1985) did develop an incremental swimming test to elicit both a $\dot{V}O_2$ value and a maximal swimming velocity in order to provide an indication of functional maximal aerobic power (FMAP). This combined measure of maximal aerobic power and mechanical efficiency has however not been reported with regard to the breaststroke .

2.9 The use of kinematic variables to predict swimming performance

Interestingly, Holmer (1979) stated that there had been little improvement in the physiological capacity (maximal oxygen uptake and lactate values) of elite swimmers over a 10 to 20 year period. Therefore improvements in performances over the same period might have more to do with advances in technical factors (technical ability, stroke mechanics etc) than physiological

ones. Indeed, Pendergast *et al.* (1977) had already hypothesised that technical advancements provided a greater potential for improvement in a swimmer's performance than increasing maximal rates of energy expenditure.

Subsequently, Pendergast *et al.* (1977, 1978a) attempted to combine both a physiological measure (rate of energy expenditure) and a biomechanical measure (ratio of efficiency to drag) in order to calculate a swimmer's maximal velocity, on the basis that the biomechanical aspects might have the greater potential to improve. Soon after this work was published stroke rate and stroke length data began to be reported because their product was thought to determine the swimming velocity of the swimmer (Craig and Pendergast, 1979). There was also a notion that this type of data would provide valuable information regarding the optimal stroke rate and stroke mechanics of swimmers (Craig *et al.*, 1979; Craig and Pendergast, 1979).

McMurray *et al.* (1990) reported a study monitoring the Arm Stroke Index (Lavoie *et al.*, 1985) over a competitive season. The ASI was determined by counting the number of strokes taken over the distance swum and dividing by the mean velocity for the distance covered. From data collected during a maximal 200 yd swim they suggested that faster collegiate breaststroke swimmers (n=11) produced lower ASI's ($r = 0.71$) than slower ones and that over a competitive season the ASI at a given speed would decrease by 5-10%. Unfortunately, the calculation of ASI was prone to innaccuracy because the number of strokes were actually counted by the exercising swimmer. Also no

mention was made as to whether partial strokes were being counted prior to turns.

2.10 The kinematic characteristics of competitive breaststroke swimming

To date there has been little evidence in the research to suggest how improvements in performances are achieved in terms of changes within stroke kinematics, although a number of studies have been reported which have analysed stroke kinematics during competitions in an attempt to determine what differentiates performers.

Mid-pool swimming velocity has been found to be highly predictive of finishing time in breaststroke swimming (Wakayoshi *et al.*, 1992) possibly because better swimmers are better able to maintain their stroke length (Thompson, 1997) and hence swimming velocity (Curry, 1975; Craig and Pendergast, 1979). Yet individually both stroke rate and stroke length have been found to be poor predictors of finishing time (D'Aquisto *et al.*, 1988; Kennedy *et al.*, 1990; Chengalur and Brown., 1992) because individual breaststroke swimmers exhibit unique combinations (Satori, 1975, 1976). However, Maglischo (1993) has expressed the opinion that despite competitive swimmer's having their own optimum stroke rate-stroke length combination, the range they demonstrate may be small enough for generalisations to be made concerning an optimal stroke rate for a particular event. This may or may not be true because to date there is little evidence to argue this point either way.

From data collected during the 1987 NCAA Men's 50 m Swimming Championship and 1988 Olympic Games Maglischo (1993) suggests that a range for stroke rate might be 50-55 and 40-45 strokes.min^{-1} in the 100 m and 200 m breaststroke events respectively, while the range for stroke length might be 1.64-1.74 and 1.88-2.18 m.

Atha and Manley (1992) have provided a possible explanation for the interindividual variation in stroke rate and stroke length relationships. They concluded that mean stroke velocity was independent of stroke rate but dependent on the intra-stroke peak velocity during constant velocity swimming. However they also stated that there was an optimum stroke period above or below which peak velocity would decrease. Additionally it has been suggested that swimmers with excellent kicks or very large hands might adopt a lower stroke rate, while shorter swimmers with less effective kicks might adopt a higher stroke rate (Grimston and Hay, 1984; Weiss *et al.*, 1988).

There is currently a lack of detailed information available in the scientific literature with regard to how stroke kinematics are manipulated by breaststroke swimmers during high intensity swimming. Although it is known that the stroke rate of breaststroke swimmers will tend to increase during a race in a compensatory manner as fatigue develops and the stroke length decreases (Pai *et al.*, 1984; Craig *et al.*, 1985; Chengalur and Brown, 1992; Kennedy *et al.*, 1990; Thompson and Haljand, 1997). It would appear that a sensitive inverted U shape relationship exists between swimming velocity and stroke rate so that

any miscalculation in stroke rate prescription may have significant implications for performance (East, 1970; Craig and Pendergast, 1979; Swaine and Reilly, 1983; Maglischo, 1993). At present it is unclear whether breaststroke swimmers favour an increase in stroke rate or an increase in stroke length when attempting to increase swimming velocity at or near to racing pace, although it is known that at sub-maximal swimming speeds any increase in both stroke rate and stroke length can effect a change of pace (Manley and Atha, 1992). Also, Keskinen and Komi (1993) have shown that an increase in swimming speed occurred in freestyle swimming as result of a disproportional increase in stroke rate and a decrease in stroke length during a series of sub-maximal to maximal swims. Interestingly they noted that above the lactate threshold a strong association existed between the increase in blood lactate accumulation and the decrease observed in stroke length. They suggested that the stroke length was reduced as a result of the lactate accumulation while stroke rate was less affected as it was primarily determined by the ability to maintain neural activation.

Maglischo (1993) has suggested that the optimal stroke rate will not change particularly when swimmers have shaved and tapered for a race distance, rather an increase in stroke length occurs which leads to a subtle increase in swimming velocity. Maglischo (1993) attributed an increase in stroke length to a decrease in drag rather than a concious effort by the swimmers to achieve a greater distance per stroke which means that an increase in stroke length may

not necessarily be observed when swimmers attempt to change pace during high intensity swimming.

It has been reported that skillful freestyle swimmers are able to maintain stroke length while increasing their stroke rate at sub-maximal to maximal speeds (Touretzski, 1993). However, this suggestion was based on the ability of an Olympic Champion freestyle swimmer and so might not be applicable to other swimmers, or indeed for a less economical stroke such as the breaststroke. For example Craig *et al.* (1979) have reported that 100 m freestyle US Olympic trialists when compared to swimmers in more slowly swum events achieved shorter finishing times by increasing their stroke rate while at the same time suffering a small decrease in stroke length. Swaine and Reilly (1983) ventured this to mean that the optimum stroke rate - stroke length relationship is influenced by the competitive distance, which adds support to the view that stroke rate may be increased, in preference to stroke length as swimming velocity increases.

It is also important to consider that there are certain distinctive traits which have been observed in the breaststroke technique which have repercussions for stroke kinematics. Craig and Pendergast (1979) have reported that the stroke length and stroke rate of breaststroke swimmers respectively decrease and increase to a lesser extent than those of freestyle swimmers during a race. This occurs because the breaststroke stroke cycle is epitomised by larger accelerations and decelerations than the other strokes which means that

increasing stroke rate beyond a fairly narrow band of tolerance, may prove counterproductive at high speeds due to the extra energy cost associated with an increased number of accelerations. In support of this Chollet *et al.* (1996) have suggested that 200 m breaststroke swimmers might favour a longer glide phase when compared with 100 m swimmers because fewer energy consuming acceleration and decelerations would be required (Manley and Atha,1992). D'Aquisto *et al.* (1988) have also reported that better breaststroke swimmers spend a greater amount of time in the glide and recovery phases during a stroke cycle which enables them to cover a greater distance per stroke than less talented swimmers at a given swimming speed. However this observation was determined from sub-maximal rather than high intensity swimming.

2.11 The effect of a change of pace on metabolic variables during high intensity breaststroke swimming

At present there remain many unanswered questions regarding the effect of a change of pace on stroke kinematics during high intensity breaststroke swimming. Unfortunately the effect of a change of pace on metabolic variables during high intensity breaststroke swimming is arguably even less clear. Maglischo (1993) has suggested that the energy requirement for a given swimming speed may be reduced by increasing the distance covered with each stroke and reducing stroke rate or conversely by adopting the opposite course of action ! This lack of definitive guidance is perhaps of greatest concern to coaches of breaststroke swimmers because the breaststroke, being less

economical than the crawl strokes for a given swimming velocity (Holmer, 1974a), is perhaps the most likely of the competitive strokes to be affected by changes in metabolism when stroke kinematics are manipulated to effect a change of pace. Yet to date there is little definitive research suggesting that a small change in performance involves a greater energy expenditure during competitive breaststroke swimming.

Sawka *et al.* (1979) reported that post exercise capillary blood lactate samples increased by 18 % when finishing time decreased by 3.6 % in a comparison of non-competitive and competitive breaststroke performances (n=3). However, Thompson (1998) found no significant differences in peak capillary blood lactate concentrations between the heat and final swims of national 100 m and 200 m breaststroke swimmers, when finishing time differed by 2.8 % (n=6). The disparity between these studies may have been due to the sampling technique. Sawka *et al.* (1979) took all their samples on 5 minutes while Thompson (1998) repeatedly sampled to obtain a peak value, on the basis that lactate efflux from muscle would be sensitive to subtle changes within work rate and that there exists considerable inter-individual variation in the time course of peak lactate determination. However, the small subject numbers in both studys are a limitation. There also remains a question as to whether the error inherent within blood lactate sampling means that this variable is sensitive to subtle changes within breaststroke swimming pace.

There has been little research in swimming or other exercise modalities, specifically reporting changes within ventilation with respect to subtle manipulations within pace. However, there has been limited research reporting the effect on ventilation of changes in cadence. For example it has been suggested that the fact that distance swimmers adopt a lower stroke rate and a longer stroke length than sprinters (Pelayo *et al.*, 1996) may mean that increasing stroke rate is energetically expensive (Weiss *et al.*, 1988). Swaine and Reilly (1983) have also reported small (but significant) changes within $\dot{V}O_2$ and $\dot{V}E$, when small changes were observed in stroke rate during high intensity freestyle swimming simulations on a swimbench. In contrast, small changes about an individual's optimum stride rate, have been shown to have little effect on $\dot{V}O_2$ in running (Cavanagh and Williams, 1982). Hence, although it is not appropriate to compare data from different exercise modalities, it might be fair to say that, at present, the ability of ventilatory responses to be sensitive to changes within kinematics is equivocal. Also, as these studies have manipulated stroke rate and stride rate directly, rather than pacing, their findings are of limited application.

To conclude, it is clear from the research that breaststroke swimmers will attempt to adopt a stroke rate - stroke length combination to maximise their swimming velocity in order to achieve a particular swimming pace. However, at near maximal or racing velocities it is unclear exactly how breaststroke swimmers manipulate their stroke kinematics to achieve an increase in

swimming velocity. It is also not known how subtle manipulations within kinematics affect metabolism.

2.12 Temporal variables (start time, turning time, end time)

The advent of sophisticated video playback, digitising and computer analysis techniques have allowed the kinematic analysis of competitive swimming to become more complex. Temporal elements such as the time taken to reach a set distance after the start and the time taken for the ingress and egress of each turn have been identified as key components of races (Guimares and Hay, 1985; Hay, 1988; Wakayoshi *et al.*, 1992). It has been shown that 20 - 40 % of a breaststroke race is spent turning depending on whether it is taking place in a 50 m or 25 m pool (Guimares and Hay, 1985; Blanksby *et al.*, 1998). Breaststroke turns have also been found to account for a greater proportion of the total event time than the turns for the other four strokes (Newble, 1982; Thayer and Hay, 1984). This is because breaststroke swimmers take the longest time to surface after a turn as they are allowed one arm stroke and one leg kick after the turn while fully submerged and because they have a smaller mean velocity off the wall than the swimmers of the other strokes (Chow *et al.*, 1984).

Newble (1982) found the breaststroke turn to be the most varied of the competitive strokes due to the greater technical expertise required. Blanksby *et al.* (1998) have since shown significant commonality of variance between the

time taken to turn (5m in to 5m out from the turn) and the finishing time for a 50 m breaststroke trial which may suggest that a better performance requires that the swimmer is able to turn more quickly as well as being able to swim faster. It must be stated however that this data was gathered with age group swimmers and may or may not be applicable to senior competitive swimmers. Finally, Thompson and Haljand (1997) have reported that the time taken to complete the last 5 m of a race may be important in elite breaststroke swimmers as European finalists were able to perform this component in a shorter time than British and Welsh Championship finalists.

Currently it is not known if these temporal kinematic variables would account for all or some of the changes observed in the performance of breaststroke swimmers from race to race, or for a change in the speed of a high intensity trial. Rather research to date has been concerned with how these elements impact on performance on single occasions. For example Arrelano *et al.* (1994) have reported that start time, turning time and end time had a direct bearing on the outcome of freestyle events in the 1992 Olympic Games but did not report data for the breaststroke events. However Thompson and Haljand (1997) have observed that these elements accounted for 31-38 % of the difference in the overall performance of European and British finalists in 100 m and 200 m breaststroke events which suggests that faster swimmers perform these elements more effectively. This would also suggest that in order for breaststroke swimmers to effect a change of pace they would have to perform these elements more effectively.

2.13 The need for race pacing strategies in breaststroke swimming

If swimmers fail to pace properly they lose stroking power, coordination and speed due to a reduction in the rate of energy production resulting from metabolic acidosis (Maglischo, 1993). Conversely by swimming more slowly in the early part of a race lactic acid accumulation will occur more slowly allowing the maintenance of swimming speed throughout (even pacing) or possibly an increase in speed in the latter part of the race (negative pacing). However, beginning a race too slowly may result in a failure to exploit the maximal rate of energy production possible and lead to underperformance. Maglischo (1993) suggests that most world and national championship races are swum in an even fashion, although some swimmers in recent competitions have swum the second half of a race faster than the first half (negative pacing). The reverse of this strategy (positive pacing) has in the main been an unsuccessful race strategy for competitive swimmers even in 100 m races (Maglischo, 1993).

It has been shown that 100 m and 200 m breaststroke swimmers swim the first half of a race in less time than the second half (Schweer, 1985; Maglischo, 1993) although these authors have speculated that the pacing was actually even, when the dive start and turns were accounted for. These elements result in the split time at the halfway point of a competitive breaststroke race being 2-3

seconds shorter than the split time for the second half of the race (Maglischo, 1993).

Anita Nall — 2:25.35 1992 world record 200 m Time for 100 m = 1:09.29				**Michael Barrowman — 2:10.16** 1992 world record 200 m Time for 100 m = 1:02.12			
Distance	Cumulative Time	Split Time	Drop-off	Distance	Cumulative Time	Split Time	Drop-off
50	33.19	33.19		50	30.43	30.43	
100	1:10.19	37.00	+3.81	100	1:03.91	33.48	+3.05
150	1:47.53	37.34	+0.34	150	1:37.12	33.21	−0.27
200	2:25.35 (1:15.16)	37.82	+0.48	200	2:10.16 (1:06.25)	33.04	+0.17
Drop-off from first to second 100 = +4.97				Drop-off from first to second 100 = +3.22			
Mary Ellen Blanchard — 2:09.06 1989 American record 200 yd Time for 100 yd = 1:00.66				**Michael Barrowman — 1:53.77** 1990 American record 200 yd Time for 100 yd = 53.77			
50	30.07	30.07		50	26.27	26.27	
100	1:03.08	33.01	+2.94	100	55.26	28.99	+2.72
150	1:35.61	32.53	−0.48	150	1:24.31	29.05	+0.06
200	2:09.06 (1:05.98)	33.45	+0.92	200	1:53.77 (58.51)	29.46	+0.41
Drop-off from first to second 100 = +2.90				Drop-off from first to second 100 = +3.25			

Table 2.2 - Representative splits for World and American records for 200 m and 200 yd breaststroke swims (Maglischo, 1993)

However, Thompson and Haljand (1997) have recently highlighted that national and international breaststroke swimmers adopt a faster swimming velocity over the first half of their races in both the 100 m and 200 m events (n=96). This is interesting as Maglischo (1993) has stated that he feels that breaststrokers could improve their overall finishing time if they did not swim as fast over the first 50

m of a 200 m breaststroke race, although to date there has been no empirical evidence to suggest that this may be true. However there have been studies comparing pacing strategies with similar timecourses in different activities.

Positive pacing has often been evident during the final stages of air resisted events (1 km - 4 km cycling, speed skating and 800 m running) and may be the pacing method of choice in activities taking approximately 60 s to complete in quantitative performance prediction models (Foster *et al.*, 1994). Ariyoshi *et al.* (1979a) have also reported that a fast/slow pacing strategy during a 4 minute, 1,400 m run resulted in better endurance being observed in a subsequent all out effort than either slow/fast or even paced strategies. A later study replicating the protocol (excepting the all out effort) found that lower blood lactate and RPE values were demonstrated during the fast/slow effort (Ariyoshi *et al.* 1979b). This was attributed to the strategy eliciting greater central drive at the beginning of the exercise which enouraged a more rapid oxygen consumption and the fact that the slowing of the pace over the trial ensured that oxidative muscle fibres were more able to remove blood lactate toward the end of the trial. Conversely other researchers have suggested that positive pacing might be detrimental to performance in non-swimming activities taking slightly longer than a 200 m breaststroke event (> 3 mins, Robinson *et al*, 1958; Foster *et al.*, 1993; Foster *et al.*, 1994) or even in activities lasting 30 seconds (Cherry *et al.*, 1997).

Few investigations have attempted to determine the pacing strategy most likely to maximise performance over a particular race distance which is suprising given that almost any race would have the potential to induce fatigue. For example a number of studies have shown 50 % reductions in power output over 30 -50 s all out efforts across different protocols and ergometric technology (McCartney *et al.*, 1983; Cheetham *et al.*, 1986; Withers *et al.*, 1991) possibly due to the rate of anaerobic glycolysis beginning to decrease after only 15 s of exercise (Jacobs *et al.*, 1983; Song *et al.*, 1988). Foster *et al.* (1994) have suggested that in different events pacing may be complicated by different physiological mechanisms.

Performance in shorter events might be limited by metabolite accumulation while in longer events substrate depletion might be the limiting factor. However this view might be too simplistic as it remains equivocal whether substrate depletion is the main cause of fatigue in heavy exercise of short duration, because ATP concentrations have been shown to be only 20-40 % depleted after heavy exercise (Foster *et al.*, 1994) despite creatine phosphate depletion (50-85 %). However, Harris *et al.* (1974) reported an improvement in performance during exercise of a similar intensity and duration to a 200 m breaststroke race following creatine supplementation which might indicate that substrate depletion is of importance in middle distance events. Conversely Terrillion *et al.*(1996) did not find an improvement from creatine supplementation in 700 m running, although their subjects may have been non-responders to creatine loading (Demant and Rhodes, 1999).

Crucially if metabolite accumulation is performance limiting then the duration of the race is of consequence because fatigue would be evident in any race that is of suffucient duration and intensity to cause an accumulation of lactic acid and an associated lowering of pH within the muscle before the end of the race. Conversely, some events are too short in duration to incur fatigue through an accumulation of metabolites or even the depletion of creatine phosphate. These activities may not require a pacing strategy because a deterioration in performance may not occur throughout the duration of the event. Consequently, van Ingen Schenau *et al.* (1994) suggest that in short lasting sprint events an all out effort is what is required.

Critically, both the 100 m and 200 m breaststroke events have been reported to elicit high levels of blood lactate accumulation (Thompson, 1998). These findings support Holmer (1974) who stated that 100 m and 200 m swimmers require a pronounced anaerobic capacity in order to be successful. Therefore the judgement of pace in these events is likely to be of some importance given the strong association between high lactic acid levels and fatigue. For example Letzelter and Freitag (1983) found that 100 m freestyle swimmers (n = 68) demonstrated a marked drop off in race speed irrespective of gender, as their races evolved in the 1980 German National Team Championships. Thompson and Haljand (1997) have also reported a decrease in mid-pool swimming velocity as 100 m and 200 m breaststroke races evolve. The implication from these data is that pacing studies are needed to determine if one pacing strategy

is more appropriate than another for 100 m and 200 m swimming events. Of these two events it would appear that pacing is particularly crucial in the longer event as Madsen and Lohberg (1987) and Thompson (1998) have observed 200 m races eliciting greater blood lactate values than 100 m events. However few investigations have attempted to determine the effect of different pacing strategies on the outcome of middle distance (2-4 minute duration) events (Foster *et al.*, 1993). As the 200 m breaststroke event generally takes 2-3 minutes to complete it sits well within the context of this statement and to the author's knowledge no study's have been published to date with regard to pacing interventions in breaststroke swimming.

2.14 Summary

Research on breaststroke swimming has tended to be concerned with it's unique mechanical characteristics and associated energy costs. There has only been limited research assessing the kinematic, temporal and metabolic characteristics of breaststroke swimming particularly at racing speeds. Kinematic research has been undertaken during competitions but little has occurred in controlled race simulations. Also relationships between and within kinematic and temporal variables, how they evolve over the course of races and their relationships with race performance have not been fully addressed. Consequently a comprehensive competition analysis is required for the breaststroke including measurements of the start, turns and mid-pool swimming velocity.

Controlled race simulations have not been widely attempted in swimming because there has not been an accepted method of pacing swimmers during free swimming, and because a method of measuring ventilation without impeding the swimmer has not been available. However, the recent development of the Aquapacer™ and the backward extrapolation technique for the estimation of oxygen uptake provide the potential for such studies to be carried out, although it needs to be established if the pacing elicited by the Aquapacer™ and the associated metabolic and kinematic responses are precise and reliable. Providing this was found to be the case it would be possible to undertake race simulations to determine how stroke kinematics are manipulated by breaststroke swimmers to effect a change of pace and the effect such changes would have on metabolism. Lastly, the inefficient nature of the breaststroke means that pace judgement is very important at racing speeds. Yet to date no studies have been conducted to determine the pacing method most appropriate for this stroke. Research is needed to examine the effect that different pacing strategies have on kinematic, temporal and metabolic variables, particularly for the 200 m as there is evidence of greater lactacidosis in this event compared with the 100 m.

Chapter 3

General Methods

3.0 General Methods

This chapter outlines the general methods, including calibrations of equipment, used in the various studies whilst more specific methods are incorporated into individual studies. All experimental work was completed in the 25 m swimming pool and Physiology and Kinanthropometry laboratory of the University of Wales Institute, Cardiff (UWIC). The laboratory has been accredited by the British Association of Sports and Exercise Sciences (BASES).

3.1 Determination of pacing

Pacing was determined using the Aquapacer™ (Challenge and Response, Inverurie, Scotland) in studies 3-5. The Aquapacer™ consists of a hand held programming unit which can be interfaced with a programmable "coin size" sounding unit (see Plate 3.1). Using the keyboard and display on the programming unit the operater sets a time period to hundredths of a second to separate when audible bleeps will occur from the sounding unit. The time duration over which the bleeping signal continues to repeat is also programmed by the operater. The program is then down loaded to the sounding unit which is subseqently stowed by the ear under the swimmer's cap (see Plate 3.2). When activated by the swimmer (by pressing a start button) the sounding unit emits six bleeps signalling a 5 second count down, with the swimmer pushing off on the sixth bleep. The sounding unit then bleeps regularly at the set time interval. In this way the device can be used to pace the stroke rate of a swimmer. For

example the swimmer can coincide reaching a particular point in the stroke cycle with the bleep signal. Alternatively the swimming speed of the swimmer can be paced by instructing the swimmer to coincide the bleep signal with reaching a particular point in the pool. In this thesis the Aquapacer™ sounding unit was programmed to emit a bleep which coincided with the swimmer progressing every 12.5 m, either to a pool-side marker placed 12.5 m along the length of the pool or with the swimmer's feet touching the wall at the turn.

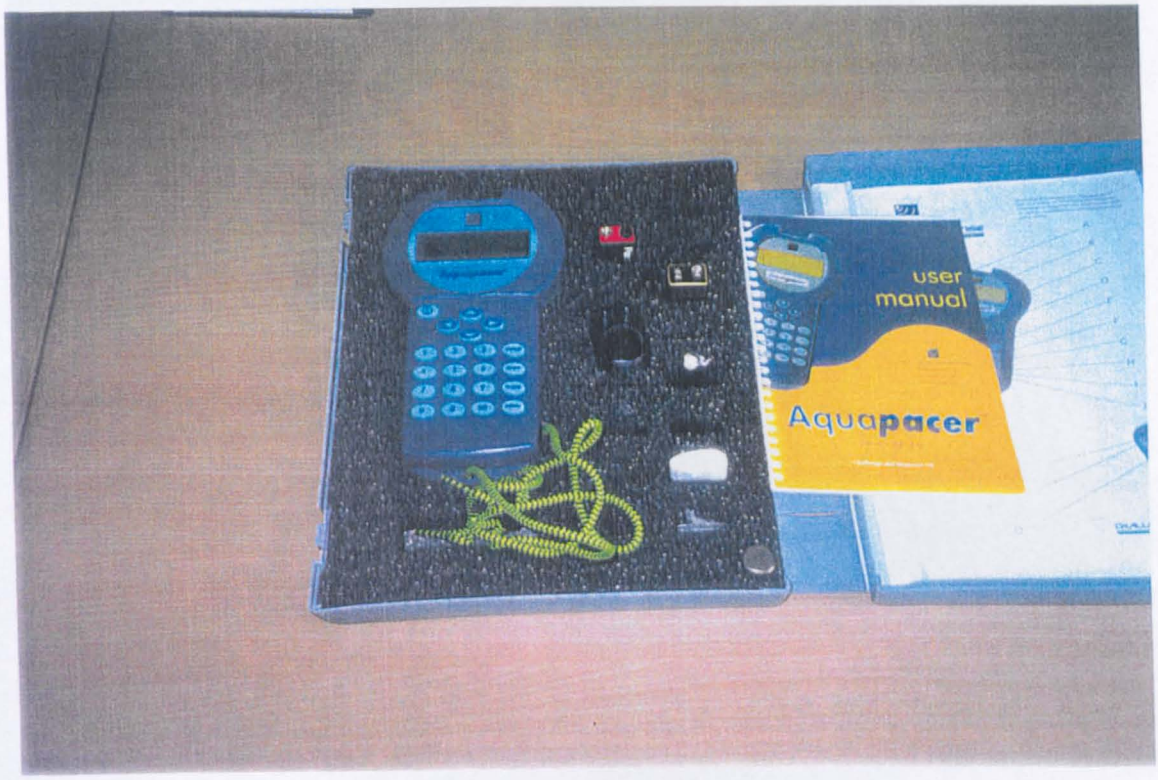

Plate 3.1 The Aquapacer™ system

An additional function of the Aquapacer™ system allows a change in the bleeping frequency after a pre-determined time period. Using this function it was possible to alter the swimming speed of the subjects halfway through 175 m trials in Chapter 8, by programming a change to occur in the bleeping frequency when the subjects had covered 87.5 m. This was possible because the desired pace of the swimmer was known and hence the time to travel 87.5 m could be calculated.

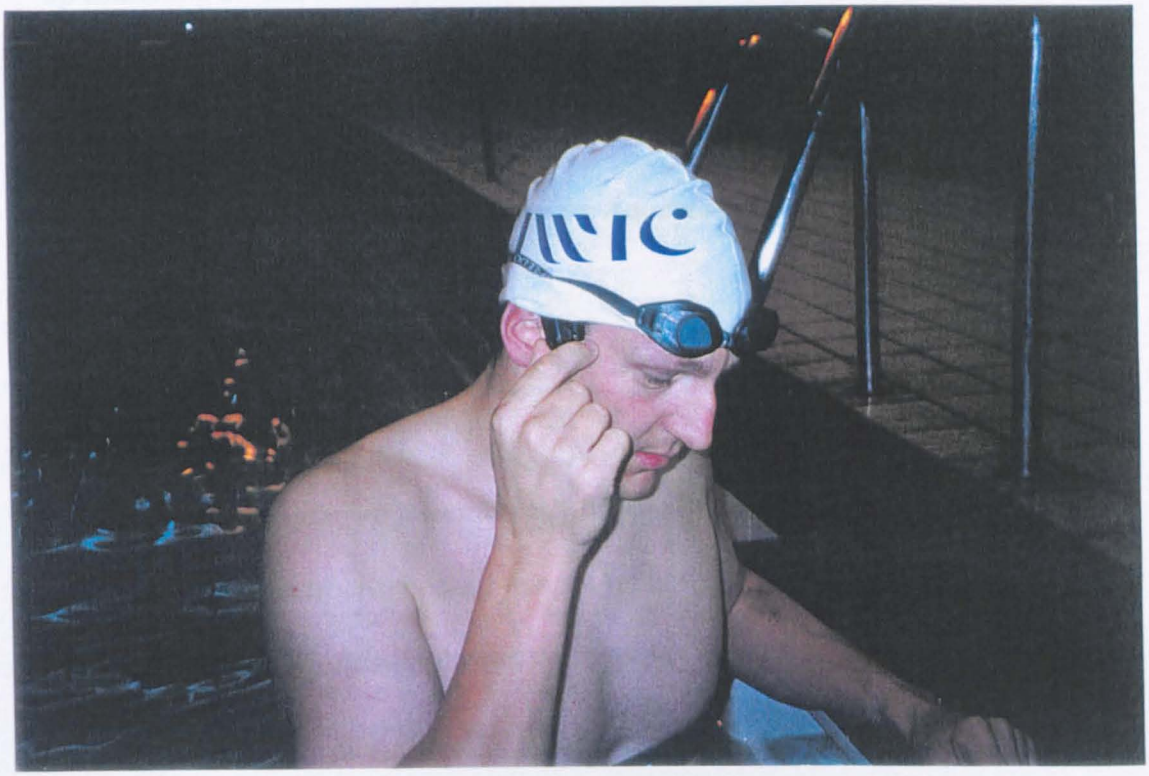

Plate 3.2 A swimmer with the Aquapacer™ sounding unit placed under the cap

A pilot study with five breaststroke swimmers (age 24 ± 4 yrs; height 1.77 ± 0.04 m; body mass 80.3 ± 4.6 kg) recruited from swimming clubs in South Wales demonstrated that they were able to habituate rapidly to the device over four sub-maximal, 50 m breaststroke swims with 30 s recoveries. The swimmers produced actual finishing times within ± 0.8 s of the predicted finishing time (~ 42 s), after two of the repetitions, in all cases (95 % Limits of Agreement -0.2 ± 0.6 s; Bland and Altman, 1986). Therefore a pre-test warm up consisting of six, 50 m repititions was considered acceptable to habituate subjects prior to each of the studies 3-5. Martin and Thompson (1999, 2000) have also reported that swimmer's paced both accurately and reliably during sub-maximal freestyle swimming using the Aquapacer™.

3.2 Determination of heart rate

Heart rate was determined telemetrically using a Polar Sports tester heart rate monitor (Polar Electro, Kempele, Finland) set to record at 5 second intervals. The memory recall function allowed the determination of the heart rate at the mid-point of trials as well as at exercise cessation. Calibration was undertaken by the manufacturer, prior to delivery, using a 12 lead ECG as the criterion measurement apparatus. Jones (1994) also reported that heart rates determined by this system and concomitantly by a 3-lead ECG were highly correlated ($r = 0.99$) during incremental running so that the line of regression ($y = 1.02 x - 1.74$) was not significantly different from the line of identity.

3.3 Determination of gas exchange variables

On cessation of exercise a Hans Rudolph, full face mask, was placed firmly over the swimmer's nose and mouth to ensure a good seal (Plate 3.3). The subject then expired air into a 100 litre Douglas bag through wide bore low resistance Falconia tubing which was approximately 1 metre in length. Expired air was collected over a 20 s period or to the nearest second thereafter to allow for completion of the last whole breath. Expired air collections were analysed within two hours of each trial for % O_2 and % CO_2 composition using a Servomex 1440c paramagnetic transducer and infrared bench top analyser. Prior to use a 3 point calibration was undertaken using BOC certified gases and atmospheric air. A sample of pure nitrogen gas (100 %) allowed the analyser's zero point to be set for oxygen and carbon dioxide by turning the zero screw. Thereafter a sample of atmospheric air (20.9 % oxygen, 0.03% carbon dioxide) was drawn through the analyser to establish the upper limits of the analyser's range and to ensure that gain had not occurred, using the span screw to make the necessary fine adjustments. A mid-point calibration was then undertaken using a calibration gas (12 % oxygen, 5% carbon dioxide) to check the linearity of the analyser. Calibration gases were transported from the gas cylinder to the analyser in purpose made impermeable pouches which had one inlet / outlet valve. Repeated samples were collected and drawn through the analyser until no further adjustments were required at any point of the calibration procedure.

Plate 3.3 A swimmer expiring through a Hans Rudolph face mask and Falconia tubing into a Douglas bag

Following the determination of % O_2 and % CO_2, the volume of expired air (ATPS) collected was determined by a Harvard Dry Gas Meter (Harvard Instruments, Edenbridge, UK) and vacuum pump. A value of 0.5 litres was added to the measured gas volume to compensate for the volume of gas extracted from the Douglas bag by the Servomex 1440c analyser during the determination of % O_2 and % CO_2 composition. A temperature guage situated at the inlet port measured the air temperature as it was drawn through the Harvard Dry Gas meter. Gas volumes (VO_{2ATPS}, VCO_{2ATPS}, VE_{ATPS}) were calculated in

STPD using the Expair Software package knowing the % O_2, % CO_2 values, period of gas collection, volume of expired gas, it's temperature and room barometric pressure (mercury barometer). Prior to use the Harvard Dry Gas meter was calibrated by inflating a previously evacuated Douglas Bag with precisely 36 litres of air using a 3 litre syringe (Cardiokinetics, Manchester, UK). The bag was then evacuated by the meter and the reading accepted if it fell within the range of 35-37 litres.

3.4 Determination of lysed blood lactate concentration.

Prior to and following exercise, capillary blood samples were taken from a puncture wound made on a subject's earlobe using a sterile lancet (Analox Instruments, London, UK). Prior to blood sampling the earlobes were cleaned using a Mediswab alcohol wipe and dried using a tissue. Blood was collected into a lysing tube (Analox Instruments, London, UK) and mixed thoroughly before being capped and transported to a fridge for storage. Within 24 hours the tubes were taken from the fridge and allowed to stand for 30 minutes at room temperature (manufacturer's recommendations) before duplicate samples were taken from each tube and analysed for lactate concentration (Analox GM7, Analox Instruments, London, UK).

Prior to use, the Analox GM7 was calibrated in the following manner. Having checked that the baseline was stable at zero and that the electrode potential was within acceptable limits (Analox recommendations), a two point calibration

was undertaken using 3 mM and 8 mM-lactate standard solutions (Analox Instruments, London, UK). The lactate standard solutions and lactate reagent (Analox Instruments, London, UK) were removed from a fridge 60 minutes prior to calibration in order to attain room temperature. The analyser's tubes were primed with lactate reagent prior to calibration by cycling the reagent through the system 6 times. A 7 µl sample of calibration standard was then drawn by micropipette and injected into the analyser. Seven microlitre samples were adopted because the Analox GM7 retains linearity within the range of 0 - 10 mM lactate concentration with this volume. However a lactate concentration beyond 10 mM would require that 3.5 µl samples were used to retain linearity (Analox Instruments recommendations). A result within ± 0.2 mM of the lactate standard's concentration was considered acceptable only when this was consistently achieved over a number of trials.

To assess the reliability of the blood assay measurement 2 lysing capillary tubes (Analox Instruments, London, UK) containing a fluoride / heparin / nitrite mixture were filled with capillary blood taken from the earlobe of a subject whilst at rest and following each stage of a three stage incremental cycling protocol designed to elicit a low - hard exercise intensity. On three days, 24 hours apart, a number of repeated measurements were made from each of the eight tubes collected (2 per stage). The coefficient of variation for each tube over the three days was calculated to be between 5 - 7 % depending on the absolute magnitude of the lactate concentration.

3.5 Determination of the Rating of Perceived Exertion (RPE)

Standardised instructions, based on guidelines recommended in the BASES Physiological Testing Guidelines 3rd Edition (1997), were given to subjects in order to familiarise them with the RPE scale prior to testing. These instructions informed the subjects how to anchor their rating of perceived exertion according to the Borg scale (1986). Subjects were instructed not to attempt to conciously match the score given from a previous trial during the study.

3.6 Determination of stroke rate (SR) measurement

In studies 1 and 2 stroke rates were calculated using digitising technology and video playback (see Chapter *4.2.2 - 4.2.4*), whereas in studies 3, 4 and 5 they were computed from hand timed measurements taken as the event happened. In studies 3, 4 and 5 the investigator measured the stroke rate by recording the time taken for the swimmer to complete three consecutive stroke cycles using a Base 3 function stopwatch (Base 3 function, Timestar, Germany), which immediately converted the timing into strokes per minute ($S.min^{-1}$).

The variation in this type of stroke rate measurement was determined in a pilot study. A video camera (Panasonic) positioned on a tripod at the pool side videoed a paced (Aquapacer™) breaststroke swimmer over six, 50 m sub-maximal breaststroke repetitions (mean finishing time 42.6 ± 0.8 s) with 30

second recoveries. From video playback the investigator took ten repeated stroke rate measurements of the same three stroke cycles, between 10 m to 20 m of each length, during repetitions 1, 3 and 6. The measurement began when the swimmer's head entered the water, prior to the leg propulsion phase, and ended three complete stroke cycles later at the same point. The watch computed the stroke rate per minute to the nearest tenth of a stroke. The 95 % limits of agreement (Bland and Altman, 1986) were calculated to be ± 0.8 $S.min^{-1}$ between repetitions 1, 3 and 6, while the coefficient of variation for each individual repetition was 2.7 %.

3.7 Determination of stroke count (SC) measurement

In studies 1 and 2 stroke length was calculated from the mid-pool swimming velocity divided by the stroke rate. In these studies both mid-pool swimming velocity and the stroke rate were determined using digitising technology (see sections Chapter *4.2.2 - 4.2.4*). In studies 3, 4 and 5 stroke count was measured in preference to stroke length. This was primarily because of the difficulty in obtaining a video camera for these latter studies.

It is possible to calculate stroke length from a stroke count using the following equation:

Estimated stroke length	=	Distance covered	/	Stroke count
(m)		(m)		(no of strokes)

however this would overestimate the actual stroke length by 13 - 16 % because the distance covered during the turns would not be accounted for (Pelayo *et al.*, 1999). Consequently, it was decided not to estimate the stroke length in studies 3, 4 and 5 in this way but rather to only record the stroke count. The assumption was also made that changes in stroke count reflect changes in stroke length (Pyne, 1997).

In studies 3, 4 and 5 stroke counts were determined by an experienced investigator by counting the number of strokes (to the nearest half stroke) taken per length. This method was thought to be acceptable as from the pilot study described earlier in section *3.6* a coefficient of variation of <5 % was found for 20 repeated measurements of the stroke count by the investigator from the video playback.

Chapter 4

An analysis of selected kinematic and temporal variables in national to elite male and female 100 m and 200 m breaststroke swimmers

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4.1 Introduction

Since the original work of East (1970), only a few studies have investigated the kinematic variables which influence the race performance of 100 m and 200 m breaststroke swimmers. These studies have concentrated on the relationship between swimming velocity (SV), stroke rate (SR) and stroke length (SL). Early investigations calculated these variables from hand timings as the event happened (Craig and Pendergast, 1979; Craig *et al.*, 1985). Unfortunately, the measurement of SL was overestimated because it was calculated on the assumption that SL = SV/SR, where the initial calculation of SV was based on event distance / finishing time (FT). The calculation did not account for the dive start or any variation in mid-pool swimming velocity and turning times at the end of each length. Early studies have also been criticised for using small numbers of swimmers of differing abilities who were assessed in training rather than in competition (Kennedy *et al.*, 1990).

More recent studies have used sophisticated video playback, digitising, and computer analysis techniques to measure SR, SL and mid-pool SV (Kennedy *et al.*, 1990; Chengular and Brown, 1992). However these studies have not considered some temporal elements of the race. These elements comprise the time taken to reach a set distance from the start of the race (start time, ST) and the time taken to travel a set distance into the turn at the end of each length of the pool and out to the same point, i.e. - turn time (TT) (Hay, 1988; Wakayoshi

et al., 1992). The time taken to complete the final 5 m of the race, or end time (ET), has also been reported (Thompson and Haljand, 1997).

Data generated in the freestyle events of the 1992 Olympic Games suggested that ST, TT and ET had a direct bearing on race outcome (Arrelano *et al.*, 1994), although this type of analysis has not been undertaken for the breaststroke event. Thompson and Haljand (1997) recently reported that temporal elements accounted for 31-38% of the overall time difference in FT in 100 m and 200 m events when comparing European and British finalists. There is currently little evidence, in the breaststroke, to establish if the temporal elements are related to performance, the swimming elements (SV, SR and SL) or to one another.

There is also a dearth of research investigating the evolution of the kinematic and temporal elements over the race distance. Subsequently, it is unclear if significant changes occur within kinematic and temporal variables as breaststroke races progress. Finally, a comparison of the 100 m and 200 m events has not previously been undertaken. Therefore, what differentiates one event from another in terms of the kinematic and temporal variables outlined is not known. Consquently, coaches of these events lack event specific information which might better inform their coaching practice.

Therefore the aims of this study were to:

i) compare changes in kinematic and temporal variables as 100 m and 200 m races progress;

ii) to evaluate the inter-relationships between kinematic and temporal variables and performance;

iii) to make comparisons between kinematic and temporal variables from 100 m and 200 m races to identify event specific differences.

4.2 Method

4.2.1 Subjects

Subjects were A and B finalists in men's and women's 100 m and 200 m breaststroke events in 12 World, International and National Championships between 1992 and 1997. Data were collected with the permission of the organisers, FINA and LEN and with the full knowledge of the competing teams.

4.2.2 Equipment and measurements

Five panning cameras (Sony G100) were used to film each race (Figure 4.1). In each case they were placed at high vantage points 5 m, 7.5 m, 15 m, 25 m and 42.5 m along the length of the pool-side to allow each portion of a race to be filmed in sequence. During each race, an experienced technician followed the event and switched from one camera to the next at each change over point, so that a recording was made onto a single video tape. Camera 1 recorded the final 5 m of the race - the end time (ET), camera 2 recorded the turning time (TT) from 7.5 m into the turn and back out to 7.5 m from the turn, camera 3 recorded the dive start to 15 m - the start time (ST), camera 4 recorded the mid-portion of the length to determine SV and SR. Camera 5 recorded TT at the opposite end of the pool. Each camera recorded at 50 Hz, allowing frame by frame measurements to be accurate to 0.02 s. Digital analysis was performed from video playback (Panasonic AG - MD830E) using a system designed by

Professor Haljand of Tallin University, Estonia. A computer was interfaced, with a controller circuit board and video playback system. Each video frame was sequentially encoded so that when played back, the computer was able to detect which frame was being played. Consequently, the time taken for a swimmer to move a known distance in the pool could be measured by the computer counting the requisite number of frames.

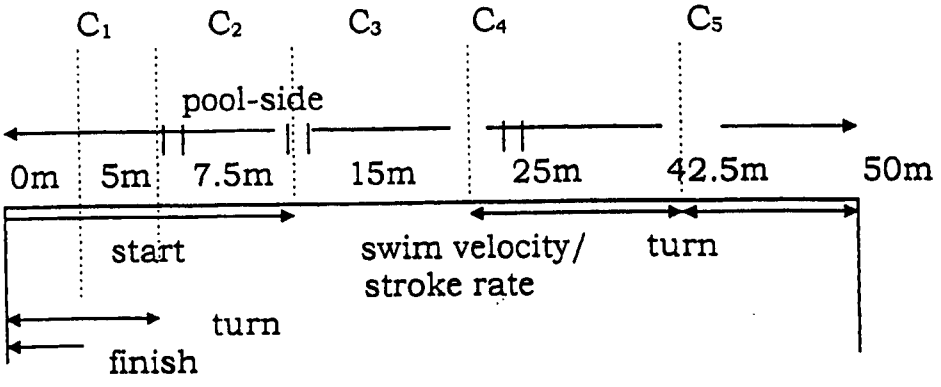


Figure 4.1 - Positioning of the five pool-side cameras (C1 ....C5) and distance markers for the calculation of kinematic and temporal elements in each event. Distances are not to scale.

4.2.3 Measurement of start time (ST), turning time (TT) and end time (ET) - (temporal variables).

Digital lines were superimposed onto the video playback at 5 m, 7.5 m, 15 m, 25 m and 42.5 m using pool-side calibration markings. The measurement of ST began with the start signal, which activated the timing system, and ended when the swimmer's head touched the digital line superimposed onto the video tape at 15 m from the start. The measurement of ET began with the swimmer's head touching the digital line 5 m from the end wall. The time taken, from this point, for the swimmer's hand to the touch the wall was then recorded. It was assumed that the swimmer's head was 0.5 m behind the wall at this point, which meant that the head had travelled 4.5 m in the time recorded. Knowing the head's velocity over 4.5 m allowed the time it would take to travel 5 m to be determined. Turn time (TT) was calculated by measuring the time taken for the swimmer's head to touch a digital line 7.5 m from the end wall and to return to the same point. For the 200 m events, a mean TT was calculated from turns made at 50 m, 100 m and 150 m.

4.2.4 Measurement of mid-pool swimming velocity (SV) and stroke rate (SR), and calculation of stroke length (SL)- (kinematic variables).

Stroke rate (SR) was calculated from the number of frames required to complete a stroke cycle, once the swimmer's head reached the 25 m digital line. Mid-pool swimming velocity (SV) was calculated from the time taken for the swimmer's head to travel from the 15m line to the 25 m line and 25 m line to the 42.5 m line for the 100 m event, and 25 m line to the 42.5 m line for the 200 m event. Stroke length (SL) was then calculated from the formula: SL (m) = SV ($m.s^{-1}$)/SR ($cycles.s^{-1}$). Mean values for SV, SR and SL were calculated for each race.

4.2.5 Descriptive and statistical analyses

Mean and standard deviations were calculated for the data. Detail regarding the sample groups can be found in Table 4.1. Dependent t-tests (for 100 m data) and one way repeated measures ANOVAs (for 200 m data) were used to compare mean differences within SV, TT, SR and SL as the 100 m and 200 m races progressed. One way ANOVA was used to compare starting, mean turning, mean swimming and end velocities within events. A post hoc test (Tukey's HSD test) was incorporated to identify where significant differences occurred ($P<0.05$). Independent t-tests were used to compare selected variables at comparable distances during 100 m and 200 m races (SV, SR, SL, TT and ET). The size of the effect of mean differences were assessed using omega

squared calculations in an attempt to account for unexplained variance (Vincent, 1995). Correlation coefficients were determined with a significance level set at $P<0.05$. Correlations were undertaken to examine if relationships changed between certain variables (those which could be determined for individual lengths) and finishing time as races evolved, and if the relationships within these selected variables changed as races evolved. Finally, correlations between certain variables occurring in the same length were calculated to determine if relationships existed (eg. ST was correlated with mid-pool SV in the first length).

4.3 Results

Comparisons between kinematic and temporal variables and how they change as a race progresses are presented in Tables 4.2 - 4.3. Relationships between kinematic and temporal variables are presented in Tables 4.4 - 4.8. Finally, kinematic and temporal variables from 100 m and 200 m races are compared in Table 4.8.

Table 4.1 - Mean (± s) finishing times (FT) for 100 m and 200 m breaststroke finalists in 12 World, International and National Championships between 1992-1997

Event	(n)	FT Mean (s)	FT ± s (s)	Min (s)	Max (s)
100 m men	159	65.05	2.62	60.65	72.35
100 m women	158	74.04	3.66	67.69	82.64
200 m men	159	141.47	6.15	132.57	154.69
200 m women	158	158.66	7.87	144.90	172.52

Table 4.2 - Comparisons of velocities for kinematic and temporal variables (mean ± s) within events

Event	Mean Race velocity ($m.s^{-1}$)	Start velocity to 15 m ($m.s^{-1}$)	Mean mid-pool swimming velocity ($m.s^{-1}$)	Mean turning velocity (7.5 m in & 7.5 m out) ($m.s^{-1}$)	End velocity (Final 5 m) ($m.s^{-1}$)	Statistic	$Omega^2$
100 m men	1.54** ±0.05	2.00** ±0.11	1.45** ±0.05	1.60** ±0.07	1.37** ±0.09	F 4267.2	0.96
100 m women	1.35** ±0.00	1.70** ±0.10	1.29** ±0.06	1.39** ±0.07	1.19** ±0.08	F 5088.2	0.95
200 m men	1.42** ±0.06	1.94** ±0.12	1.35** ±0.06	1.48** ±0.09	1.27** ±0.10	F 5010.2	0.95
200 m women	1.26** ±0.06	1.65** ±0.10	1.23** ±0.06	1.33** ±0.07	1.13** ±0.08	F 5201.4	0.97

** denotes $P<0.01$ between all conditions within each event

The velocities achieved in the start and turning phases were significantly greater than mean race velocity ($P<0.01$) which was significantly greater than mean mid-pool swimming velocity (as a result of the inclusion of ST and TT) (Table 4.2). Swimming velocity decreased significantly over each consecutive 50 m (Table 4.3) demonstrating a positive split pacing strategy during races. In the 200 m event the mean mid-pool SV values for lengths 2, 3 and 4, although significantly decreasing, demonstrate a much smaller decrease in SV (typically 0.1 $m.s^{-1}.length^{-1}$) than is observed following the first length. It is important to note that the standard deviation for mid-pool SV for lengths 3 and 4, in the male 200 m is almost twice that observed for lengths 1 and 2, or for any of the lengths in the female event. The increase in the

dispersion of the SV in lengths 3 and 4 also coincided with worsening relationships being observed between these variables and FT (Table 4.5).

Table 4.3 - Comparisons of kinematic and temporal variables (mean ± s) for male and female 100 m and 200 m breaststroke swimmers

	Swimming velocity ($m.s^{-1}$)					
	SV length 1	**SV length 2**	**SV length 3**	**SV length 4**	**Statistic**	**Omega²**
100 m						
male	1.49±0.05[a]	1.40±0.06	NA	NA	t 27.99	71 %
female	1.33±0.07[a]	1.24±0.07	NA	NA	t 26.43	69 %
200 m						
male	1.41±0.07[a]	1.33±0.07[b]	1.32±0.12[c]	1.31±0.12[a]	F 124.15	41 %
female	1.27±0.07[a]	1.20±0.07[a]	1.19±0.07[a]	1.18±0.06[a]	F 573.40	77 %
	Stroke rate ($cycles.min^{-1}$)					
	SR length 1	**SR length 2**	**SR length 3**	**SR length 4**	**Statistic**	**Omega²**
100 m						
male	49.2±5.4[a]	51.0±5.2	NA	NA	t -4.62	6 %
female	49.5±5.8[a]	49.7±5.7	NA	NA	t -0.62	NA
200 m						
male	38.6±4.2[a]	37.1±4.5[a]	38.8±5.4[a]	43.0±5.9[a]	F 91.80	35 %
female	40.8±5.2[a]	38.8±5.3[b]	39.6±5.0[c]	43.4±5.7[a]	F 73.42	30 %
	Stroke length (m)					
	SL length 1	**SL length 2**	**SL length 3**	**SL length 4**	**Statistic**	**Omega²**
100 m						
male	1.85±0.30[a]	1.67±0.17	NA	NA	t 9.42	22 %
female	1.63±0.19[a]	1.52±0.18	NA	NA	t 11.91	31 %
200 m						
male	2.22±0.25[a]	2.18±0.27[a]	2.04±0.29[a]	1.84±0.25[a]	F 199.66	54 %
female	1.89±0.25[a]	1.89±0.25[a]	1.82±0.24[a]	1.66±0.21[a]	F 112.71	39 %
	Turning times (s)					
	TT at 50 m	**TT at 100 m**	**TT at 150 m**	**Statistic**	**Omega²**	
200 m						
male	9.83±0.59[a]	10.17±10.7[b]	10.35±1.00[c]	F 36.01	17 %	
female	11.28±0.65[a]	11.69±0.78[a]	11.82±0.74[a]	F 306.02	66 %	

[a] significant at P<0.01 from all conditions

[b] significant at P<0.01 from all conditions except at 150 m where significant at P<0.05

[c] significant at P<0.01 from all conditions except at 100 m where significant at P<0.05

In the men's 100 m (Table 4.3), SR increased significantly (by 3.6%, $P<0.01$) on the second length while remaining relatively constant in the female race. Despite this, mid-pool SV fell by 6-7% in both male and female events due to a disproportionate decrease in stroke length (9.7% & 6.7%, $P<0.01$)(Table 4.3). The act of swimming the first length significantly faster than the final length coincided with the swimmers being unable to maintain their SL during the second length.

This pattern was also found when comparing the first 100m with the second 100m of the 200m events (Table 4.3), although in the first two lengths of the 200 m events, the SR pattern observed was actually opposite to that seen in the 100 m event. For example in the women's event a 5% decrease in SR resulted in a similar decrease in mid-pool SV while a slightly greater loss in mid-pool SV was observed in the male subjects as a result of 3.9 % fall in SR coupled with a 1.8% decrease in SL. In both the male and female 200 m events, the SR s adopted were generally at their lowest during the second length but increased thereafter with the greatest SR in the final length ($P<0.01$).

Table 4.4 - Interrelationships between finishing time (FT), swimming velocity (SV), turning time (TT) and end time (ET) for male and female 100 m breaststroke swimmers

	ST (s)	SV mean ($m.s^{-1}$)	SV length 1a ($m.s^{-1}$)	SV length 1b ($m.s^{-1}$)	SV length 2a ($m.s^{-1}$)	SV length 2b ($m.s^{-1}$)	TT 50m (s)	ET (s)
FT (s)	0.87 ** (0.89**)	-0.97** (-0.92**)	-0.85** (-0.90**)	-0.91** (-0.94**)	-0.90** (-0.95**)	-0.91** (-0.92**)	0.84** (0.92**)	0.76** (0.82**)
ST (s)		-0.80** (-0.85**)	-0.71** (-0.82**)				0.78** (0.87**)	0.60** (0.70**)
SV mean ($m.s^{-1}$)			0.90** (0.93**)	0.92** (0.95**)	0.92** (0.95**)	0.90** (0.92**)	-0.74** (-0.87**)	-0.67** (-0.78**)
TT 50m (s)				-0.76** (-0.86**)	-0.60** (-0.82**)			0.62** (0.71**)
ET (s)		-0.67** (-0.78**)				-0.75** (-0.76**)		

Key to abbreviations in the tables:

length 1a = 15 to 25 m in length 1, length 1b = 25 to 42.5 m in length 1

Female results in brackets ( ), * denotes $P<0.05$, ** denotes $P<0.01$

Mean mid-pool SV explains most of the variation in FT ($r^2 = >0.85$), in both 100 m and 200 m events (Tables 4.4 and 4.5). Measurements of mid-pool SV taken at 4 equidistant stages during the 100m and 200m races also demonstrated significant correlations which were highly predictive of FT (r>-0.85 to -0.99), except in the men's 200 m where correlations were only moderately predictive of FT over the last two lengths (r=-0.70 & -0.67 respectively).

Table 4.5 - Interrelationships between finishing time (FT), swimming velocity (SV), turning time (TT) and end time (ET) for male and female 200 m breaststroke swimmers

	ST (s)	SV mean ($m.s^{-1}$)	SV length 1 ($m.s^{-1}$)	SV length 2 ($m.s^{-1}$)	SV length 3 ($m.s^{-1}$)	SV length 4 ($m.s^{-1}$)	TT mean (s)	TT 50m (s)	TT 100m (s)	TT 150m (s)	ET (s)
FT (s)	0.85** (0.88**)	-0.99** (-0.99**)	-0.91** (-0.96**)	-0.94** (-0.96**)	-0.70** (-0.94**)	-0.67** (-0.99**)	0.94** (0.96**)	0.90** (0.92**)	0.46** (0.95**)	0.38** (0.92**)	0.65** (0.77**)
ST (s)		-0.81** (-0.86**)	-0.80** (-0.85**)				0.85** (0.85**)	0.85** (0.85**)			0.48** (0.64**)
SV mean ($m.s^{-1}$)			0.99** (0.94**)	0.95** (0.97**)	0.66** (0.98**)	0.63** (0.94**)	-0.89** (-0.91**)				-0.61** (-0.74**)
TT at 50m (s)			-0.85** (-0.86**)	-0.85** (-0.85**)			0.96** (0.96**)		0.46** (0.92**)	0.36** (0.90**)	
TT at 100m (s)				-0.46** (-0.88**)	-0.29** (-0.91**)		0.54** (0.98**)	0.46** (0.92**)		0.95** (0.94**)	
TT at 150m (s)					0.36** (-0.90**)	0.38** (-0.86**)	0.45** (0.97**)	0.36** (0.90**)	0.95** (0.94**)		0.22* (0.72**)
ET (s)		-0.61** (-0.74**)				-0.54** (-0.82**)	-0.54** (0.72**)			0.22* (0.72**)	

Female results in brackets ( )
* denotes P<0.05
** denotes P<0.01

Relationships between mean TT and FT in the 100 m and 200 m were significant with the greater relationships being observed in the 200 m, reflecting that an increased proportion of the race is spent turning (Tables 4.4 and 4.5). Logically, as mid-pool SV was highly correlated with FT, the relationship between mid-pool SV and TT was also significant (r = >-0.86) throughout the women's 200 m event. However in the men's 200 m, although the relationship between mean TT and FT was high, only the first turn was highly correlated with FT (r = 0.90). Indeed the relationship between TT and FT over the last 2 turns of the men's 200 m, although significant, was poor enough to preclude a prediction of one from the other without incurring significant error. The relationship between mean TT and turning times for the last 2 turns were

relatively poor in the men's 200 m event and this was due to a poor, but still significant, relationship between TT at 50 m and the last two turns. The final 2 turns were highly correlated suggesting that the changes in the pattern observed within turning times were consistent over the last two turns.

Start times were significantly related to FT and mean mid-pool SV for all events (Tables 4.4 and 4.5). ST was also moderately correlated with mid-pool SV, TT and ET, as was mean mid-pool SV with mean TT. This suggests that swimmers are relatively proficient at each kinematic component.

End time was moderately correlated with FT in all events but was more significant in the 100 m reflecting its relative importance in that event (Tables 4.4 and 4.5). The relationship between mid-pool SV and ET, in the 100 m and women's 200 m was moderate demonstrating how the momentum of the final part of the length reflects partially on the finish over the last 5m of the race. This relationship was however poor in the men's 200 m and may be a reflection of the greater variation in SV observed in this event.

Table 4.6 - Interrelationships between finishing time (FT), mid-pool swimming velocity (SV), stroke rate (SR) and stroke length (SL) for male and female 100 m breaststroke swimmers

	SR mean (cycles. min^{-1})	SR length 1 (cycles. min^{-1})	SR length 2 (cycles. min^{-1})	SL mean (m)	SL length 1 (m)	SL length 2 (m)	SV mean ($m.s^{-1}$)	SV length 1 ($m.s^{-1}$)	SV length 2 ($m.s^{-1}$)
FT (s)	-0.19 (-0.13)	-0.12 (-0.05)	-0.22* (-0.19)	-0.39** (-0.32**)	-0.18 (-0.28**)	-0.10 (-0.27**)	-0.97** (-0.99**)	-0.91** (-0.93**)	-0.91** (-0.92**)
SR mean (cycles. min^{-1})		0.89** (0.94**)	0.89** (0.94**)	-0.87** (-0.90**)			0.23* (0.15)		
SR length 1 (cycles. min^{-1})			0.57** (0.76**)		-0.85** (-0.91**)			0.29* (0.15)	
SR length 2 (cycles. min^{-1})					-0.88** (-0.88**)				0.21* (0.14)
SL mean (m)					0.94** (0.95**)	0.80** (0.95**)	0.07 (0.25*)		
SL length 1 (m)						0.53** (0.80**)		-0.07 (0.25*)	
SL length 2 (m)									0.24* (0.28**)

Female results in brackets ( )

* denotes $P<0.05$

** denotes $P<0.01$

Table 4.7 - Interrelationships between finishing time (FT), swimming velocity (SV), stroke rate (SR) and stroke length (SL) for male and female 200 m breaststroke swimmers

	SR mean (cycles. min^{-1})	SR length 1 (cycles. min^{-1})	SR length 2 (cycles. min^{-1}))	SR length 3 (cycles. min^{-1})	SR length 4 (cycles. min^{-1})	SL mean (cycles. min^{-1})	SL length 1 (m)	SL length 2 (m)	SL length 3 (m)	SL length 4 (m)	SV mean (m.s^{-1})	SV length 1 (m.s^{-1})	SV length 2 (m.s^{-1})	SV length 3 (m.s^{-1})	SV length 4 (m.s^{-1})
FT	-0.08 (-0.20*)	-0.02 (-0.05)	-0.02 (-0.08)	-0.14 (-0.12)	-0.26** (-0.20*)	-0.38** (-0.36**)	-0.36** (-0.32**)	-0.35** (-0.34**)	-0.42** (-0.34**)	-0.30** (-0.23*)	-0.99** (-0.99**)	-0.91** (-0.92**)	-0.94** (-0.96**)	-0.70** (-0.98**)	-0.68** (-0.94**)
SR mean (cycles.min^{-1})		0.76** (0.83**)	0.89** (0.92**)	0.76** (0.91**)	0.63** (0.82**)	-0.87** (-0.86**)					0.11 (0.15)				
SR length 1 (cycles.min^{-1})			0.72** (0.75**)	0.46** (0.64**)	0.22* (0.51**)		-0.68** (-0.89**)					0.20* (0.15)			
SR length 2 (cycles.min^{-1})				0.68** (0.89**)	0.41** (0.63**)			-0.91** (-0.89**)					0.14 (0.18)		
SR length 3 (cycles.min^{-1})					0.74** (0.70**)				-0.32** (-0.86**)					0.51** (0.12)	
SR length 4 (cycles.min^{-1})										-0.30** (-0.88**)					0.57** (0.25*)
SL mean (m)							0.84** (0.84**)	0.93** (0.92**)	0.79** (0.89**)	0.63** (0.83**)	0.36** (0.34**)				
SL length 1 (m)								0.78** (0.73**)	0.62** (0.62**)	0.38** (0.54**)		0.21* (0.26**)			
SL length 2 (m)									0.72** (0.79**)	0.47** (0.68**)			0.23* (0.25*)		
SL length 3 (m)										0.77** (0.70**)				0.61** (0.37**)	
SL length 4 (m)															0.57** (0.25*)

Female results in brackets ( ), * denotes P<0.05, ** denotes P<0.01

Mean SR and mean SL were highly inversely related during both breaststroke events (Tables 4.6 and 4.7) and for both sexes ($r = >-0.86$) demonstrating that a high SR often coincides with a low SL. However, the relationship between SR and SL became weaker as the race progressed in the men's 200 m race (Table 4.7).

Mean SR was only significantly correlated with FT in the female 200 m event, however little of the variance in FT was explained by the variable ($r^2 = <5\%$)(Table 4.7). A possible explanation was provided by the relationships between SR measurements made on consecutive and non-consecutive lengths, which demonstrated only poor-moderate relationships. Relationships between SL and FT were also poor ($r<-0.4$), although significant. Therefore a shorter FT was related to a longer SL, although the majority of the variation in FT was not explained by SL.

Finally, a comparison between 100 m and 200 m events was made for kinematic variables over corresponding distances (Table 4.8). Mean values for race velocity, mid-pool SV and SR were significantly higher ($P<0.01$) for the 100 m compared to the 200 m while ST, SL, TT and ET were significantly lower ($P<0.01$). The greater velocity in the 100 m (3-5%) was achieved with the adoption of a higher SR coupled with a shorter SL.

Table 4.8 - Comparisons between 100 m and 200 m breaststroke swimmers for selected kinematic and temporal variables (mean ± s)

	Male 100m	Male 200m	t	Omega^2	Female 100m	Female 200m	t	Omega^2
Mean race velocity ($m.s^{-1}$)	1.54** ±0.05	1.42 ±0.06	193.0	99%	1.35** ±0.07	1.26 ±0.06	130.1	98%
ST (s)	7.52** ±0.43	7.75 ±0.45	8.6	19%	8.84** ±0.50	9.05 ±0.53	7.8	6.5%
SV length 1 ($m.s^{-1}$)	1.49** ±0.05	1.41 ±0.07	24.0	64%	1.33** ±0.07	1.27 ±0.07	16.6	46%
SV length 2 ($m.s^{-1}$)	1.40** ±0.06	1.33 ±0.07	20.5	57%	1.24** ±0.07	1.20 ±0.06	9.7	23%
SR length 1 (cycles. min^{-1})	49.2** ±5.4	38.6 ±4.2	19.5	54%	49.5** ±5.8	40.8 ±5.2	15.8	44%
SR length 2 (cycles. min^{-1})	51.0** ±5.2	37.1 ±4.5	24.9	66%	49.7** ±5.7	38.8 ±5.3	19.2	54%
SL length 1 (m)	1.85** ±0.30	2.22 ±0.25	12.4	32%	1.63** ±0.19	1.89 ±0.25	12.7	34%
SL length 2 (m)	1.67** ±0.17	2.18 ±0.27	21.4	59%	1.52** ±0.18	1.89 ±0.25	17.4	50%
$TT_{50\,m}$ (s)	9.40** ±0.40	9.83 ±0.59	12.4	32%	10.84** ±0.57	11.28 ±0.65	12.3	32%
ET (s)	3.31** ±0.22	3.54 ±0.33	9.4	22%	3.79** ±0.27	4.00 ±0.02	9.2	21%

** denotes $P<0.01$

4.4 Discussion

Wakayoshi *et al.* (1992) suggested that SVs were significantly greater in swimmers who produced faster race times when comparing national and elite swimmers. The findings in this study are largely in agreement, with mean mid-pool SV and individual mid-pool SV measurements being strongly negatively related to FT in both 100m and 200m breaststroke events. However in the men's 200 m, poorer relationships were observed during the last two lengths which suggests that, unlike the other events, the relationship between mid-pool SV and FT diminishes in the final 100 m of the men's event.

Maglischo (1993) suggested that successful breaststroke swimmers adopt an even paced race when the dive start is accounted for (by subtracting 2-3 s from the split time at half distance). However, the observations from our study are that mid-pool SV significantly decreases over each consecutive 50 m of a race, with the first length being swum 6-7% faster than the final length irrespective of race distance or sex. This demonstrates that national - elite standard breaststroke swimmers tend to adopt a positively split pacing strategy for their races.

Whether this practice is a normal race strategy or the product of the swimmer's anxiety is not known but the end result is a decreasing SV. For example, in the 200 m the swimmers typically demonstrate a slight decrement in SV over the last 3 lengths, which is likely to be related to the onset of leg fatigue; due to the

heavy reliance on leg propulsion in the breaststroke (Maglischo, 1993) resulting in metabolic acidosis (Thompson, 1998). Our findings would suggest that the adoption of an even paced race strategy needs to be evaluated in order to investigate whether fatigue can be attenuated, resulting in a higher mean SV during breaststroke races.

Mean SR and SL were poorly correlated with FT, in agreement with previous studies (Wakayoshi *et al.*, 1982; Kennedy *et al.*, 1990; Chengalur and Brown, 1992), and were only poorly to moderately correlated with mid-pool SV. A possible explanation in the 200 m was that the relationships between SR and SL tended to worsen over the race distance, suggesting that swimmers do not maintain a given SR : SL combination. Neither do more successful swimmers adopt a lower SR and a greater SL when compared to less successful swimmers (Wakayoshi *et al.*, 1982).

In 1993, Maglischo advised that breaststroke swimmers maintain a constant SR and hence a constant energy expenditure throughout a race. However it appears that competitive swimmers do not generally conform to this practice. Rather individual swimmers generally adopt a unique SR : SL combination, which changes over the race distance, presumably as a consequence of fatigue resulting from the positively split race pattern.

The effect of adopting a disproportionately high SV during the first length commonly resulted in a fall in SL on each subsequent length. This could be

indicative of fatigue leading to a poor body alignment and increased drag. If this was the case then the subsequent increase in energy expenditure would reduce swimming economy. Indeed in the 100 m events both sexes suffered a loss in SV during the second length because they were unable to increase SR sufficiently to compensate for the loss in SL. Interestingly in the 200 m events SR was actually reduced during the second length, perhaps in an effort to conserve energy. Even so, SL was reduced in the men's event (although it was maintained in the women's event). However, thereafter, SR increased on each subsequent length in an unsuccessful attempt to compensate for a perpetually decreasing SL.

The tactic of increasing SR to compensate for a decreasing SL is in agreement with previous work (Kennedy *et al.*, 1990; Chengalur and Brown, 1992). It must be noted that during the last 2 lengths of the men's race the relationship between SR and SL fell dramatically ($r = -0.3$ to -0.32), although remaining significant ($P<0.01$). This increase in stroke variation coincided with a marked increase in the standard deviation of mid-pool SV, which may partly explain the deterioration in the relationship with FT.

The temporal elements constitute a large proportion of both 100 m (35%) and 200 m races (32.5%), with the starting and turning velocities determining that mean race velocity is greater than mean mid-pool SV (Table 4.7). Thompson and Haljand (1997) have calculated that male and female British Championship finalists, when compared with European Championship finalists, took 0.62 s

and 0.39 s longer respectively in the 100 m event, and 1.15 s and 1.79 s longer respectively in the 200 m event to complete the start, turn(s) and final 5m of the race. Wakayoshi *et al.* (1992) have reported that male Pan Pacific finalists turned significantly faster than Japanese Olympic trial swimmers and coincidentally achieved faster FT s.

In the present study strong correlations were observed between mean TT and FT, and between individual TT s and mid-pool SV s, in the 100 m and female 200 m events. This demonstrates that elite achieve faster turning times possibly as a result of better technical ability and / or as a result of faster approach velocities (resulting in faster exit velocities). However in the men's 200 m, only the first of the three turns was highly correlated with FT ($r = 0.90$). The poor relationships observed between FT and TT over the last 2 turns are interesting as they coincide with the deterioration in the relationship between FT and mid-pool SV described earlier. The relationship between TT and mid-pool SV also deteriorates after the first turn ($r = <0.5$) suggesting that both variables became increasingly unlikely to explain the variance in one another. Therefore it appears that the ability to predict FT, from mid-pool SV and TT, becomes increasingly difficult in the final stages of the men's 200 m event.

Some researchers have suggested that turning times in the 200 m may increase as a race progresses but have not reported this finding to be statistically significant (Maglishco, 1993; Thompson and Haljand, 1997). The present study's data confirms that turning times do increase significantly (by

approximately 0.5 s for both sexes) as breaststroke races progress, a finding which may be linked to changes within mid-pool SV.

Wakayoshi *et al.* (1982) compared 2 groups of elite / national swimmers with significantly different mean FT s and found no differences between their mean ST s. However, in the present study, starting times were related to both FT and mean mid-pool SV, for all events, suggesting that elite swimmers might be faster starters. A reason for the lack of agreement between the two studies might be that the present study contained a much larger sample group which was more heterogeneous in its composition. Of further interest was that ST and mid-pool SV in the first length were moderately correlated suggesting that the momentum from a fast start was maintained during the first length.

Inter-relationships between ST, mean mid-pool SV, mean TT and ET were also investigated to determine if elite swimmers possessed an holistic proficiency for each facet of a race. Moderate relationships were found between ST and mid-pool SV, TT and ET, and mean mid-pool SV with mean TT suggesting that swimmers are generally relatively proficient for each kinematic component.

Chollet *et al.* (1996) reported that race velocity and SR were greater in 100m events than in 200 m events with SL being significantly lower. However SL was overestimated having been calculated from the race velocity divided by SR. Also SR was measured from hand timings and hence possibly prone to significant error. In the present study mean values for race velocity, mid-pool

SV and SR were significantly higher for the 100 m compared to the 200 m while ST, SL, TT and ET were significantly lower; which does support the findings of Chollet *et al.* (1996). Notably, the greater velocity in the 100 m (3-5%) was achieved with the adoption of a greater SR coupled with a shorter SL. Chollet *et al.* (1996) attributed this to 100 m swimmers favouring the propulsive phase of the stroke which led them to increase SR with a resultant decrease in SL, while the 200 m swimmers tried to optimise swimming economy by lengthening their glide phase and hence reduced their SR. It is not possible to discuss if anthropometry influenced the stroke kinematics adopted in the present study as the swimmers were not matched for age, height, body mass and arm length, although poor relationships have been previously observed between SR, SL and height (Kennedy *et al.*, 1990; Chengalur and Brown, 1992).

As approach velocities were slower, turning times at 50 m were greater in the 200 m event compared to the 100 m event. However, the differential observed in ST between the 100 m and 200 m events was surprising. Fatigue or pacing strategies would not apply at this point and so the extra time taken for the 200 m swimmers to reach 15 m from the start can only be explained by differences in physical characteristics between specialist 100 m and 200 m swimmers or their competence with the starting technique. It must be stated that the effect size of the ST comparisons although large for the men's event were moderately small for the women's events.

Finally, the very large competition sample reported in this study contained swimmers ranging in ability from national standard to the world's best and so overcomes many of concerns expressed by Kennedy *et al.*, 1990 for studies of this type (small sample size, non-competitive, heterogeneous sample groups). The number of variables reported in this study are also more conclusive than any similar study previously published.

4.5 Conclusions

A comparison of changes in kinematic and temporal variables during 100 m and 200 m races found that mid-pool SV, TT and SL generally deteriorated as the races progressed, which was attributed to swimmers adopting a positively split race strategy. An evaluation of the inter-relationships between kinematic and temporal variables and performance demonstrated that the primary correllate with FT was mid-pool SV, with TT and ST being moderately related, suggesting that elite breaststroke swimmers perform better across a number of kinematic and temporal elements. Stroke rate and SL were either not related to FT or were poorly related to FT, with less than 18 % of the variance in FT being explained by these variables. Stroke rate and SL were negatively related to each other. Male 200 m swimmers were anomalous in that they demonstrated a greater variation in mid-pool SV and TT, and poor correlations with FT in the latter stages of the race. Finally, a comparison of variables between 100 m and 200 m events found that 100 m swimmers exhibit greater mid-pool SV s and SR s coupled with shorter ST s, TT s and
SL s than 200 m swimmers suggesting that event specific preparation might be appropriate.

Chapter 5

The relative importance of selected kinematic and temporal variables in relation to swimming performance in national to elite male and female 100 m and 200 m breaststroke swimmers

5.1 Introduction

The analysis in Chapter 4 identified a number of key variables which were significantly related to finishing time (FT). However, the relative importance of these elements with regard to FT was not investigated. Although, the primary correlate of FT was mean mid-pool SV and logically, given the large proportion of the race it was representing, it can be assumed to be the variable of primary importance in predicting FT. Based on this premise coaches currently direct the vast majority of a swimmer's training towards improving swimming velocity over the race distance.

However, the work of Arellano *et al.* (1994) and Thompson and Haljand (1997) has highlighted that the temporal elements (start time (ST), turning time (TT) and end time (ET)) also have a direct bearing on race outcome during freestyle and breaststroke events respectively. Haljand (1997) has argued that the importance of the turns has often been underestimated. Thus it seems apparent that when attempting to detect the main determinants of performance, a wide-ranging approach to race analysis should be adopted involving both kinematic and temporal elements. Indeed, recent evidence would suggest that it would be inappropriate to ignore the temporal elements when analysing the performance of a swimmer. Similarly swimming coaches might want to consider placing greater emphasis in training on the swimmer's starts, turns and judgement of the finish. The analysis in this next chapter will provide the sports scientist, coach and swimmer with a better understanding of the relative importance of the

kinematic and temporal elements with regard to FT and so further inform their practise. The coach may then be better able to prioritise training time accordingly.

The aim of the present study was to determine the relative importance of SV, stroke rate (SR), stroke length (SL), ST, TT and ET with regard to FT using data from 15 World, International and National Championships over a five year period. To achieve this aim it was necessary to produce multiple regression equations for each of the 100 m and 200 m breaststroke events because the primary strength of multiple regression analysis lies in its use as a technique for establishing the relative importance of independent variables (IV s) with regard to the dependent variable (DV) (Vincent, 1995). The equations produced are intended to accurately predict FT, so the application of such predictive equations for swimming coaches will also be discussed.

5.2 Method

5.2.1 Subjects

Subjects were A and B finalists in men's and women's 100 m and 200 m breaststroke events in 15 World, International and National Championships between 1992 and 1998 (Table 5.1). Data were collected with the permission of the organisers, FINA and LEN and with the full knowledge of the competing teams.

Table 5.1 - Mean (± s) finishing times (FT) for breaststroke finalists in 15 World, International and National championships between 1992 - 1998.

Event	(n)	FT Mean (s)	FT ± s (s)
100 m men	159	65.05	2.62
100 m women	157	74.06	3.66
200 m men	172	142.71	7.41
200 m women	172	159.92	8.73

5.2.2 Equipment and measurements

The same procedures were used as described in Chapter 4, (sections *4.2.1 - 4.2.4)*. The kinematic and temporal elements measured (ST, SV, TT and ET) accounted for 70 % and 65 % of the race distance for the 100 m and 200 m

events respectively. This was assumed to elicit a precise prediction of FT from multiple regression analysis.

5.2.3 Identification of appropriate independent variables

As a result of pilot data analysis, it was shown that both SR and SL were inversely related. The relationship was so great (r^2 = >0.90) that multicollinearity was considered to exist (Lewis-Beck, 1993) and led to SL being eliminated from the analysis. This decision was made on the basis that SL was not actually directly measured and that if the predictive equations generated in this study were to be practically applied by swimming coaches using limited equipment then SL would prove the more difficult variable to measure (hand held stopwatches now allow the coach to measure SR from timing 3-5 consecutive stroke cycles).

5.2.4 Statistical analyses

All statistical procedures in the present study were performed using Minitab version 8.2. An alpha level of $P<0.05$ was applied to all data analysed.

5.2.4.1 Experimental and cross-validation groups

Each sample group was randomly split into an *experimental group* and a *cross validation group.* The data from the experimental group was used to develop the multiple regression equations to predict swimming performance (men's 100 m: $n = 130$; women's 100 m: $n = 129$; men's 200 m: $n = 116$; women's 200 m: $n = 116$), whereas the data from the *cross-validation group* was used to test the validity of the predictive equation (men's 100 m: $n = 29$; women's 100 m: $n = 28$; men's 200 m: $n = 56$; women's 200 m: $n = 56$). Through the production of precise and valid predictive equations the relative importance of the kinematic and temporal variables with regard to finishing time would be determined. The size of the experimental groups were calculated on the basis that 20 - 30 data points would be required per independent variable used in the regression analysis (Vincent, 1995).

5.2.4.2 Development of the predictive equations

The nature of the relationship between the dependent variable (predicted swimming finishing time (FT_P) in this instance) and the independent variables (ST, SV, SR, TT, ET) was expressed in the form: $Y = a + b_1X_1 + b_2X_2 + b_3X_3 .... + b_kX_k$, where $X_1, X_2, X_3, ... , X_k$ are the independent variables, a is the intercept and $b_1, b_2, b_3, ... , b_k$ are the regression coefficients for the independent variables. Each regression coefficient expresses the amount of change in the dependent variable with the effect of all other independent

variables in the equation being controlled. The regression coefficient also expresses the unique contribution of the relevant independent variable to the dependent variable (Bryman and Cramer, 1996).

Where a computed P value, from the t-test performed on each of the independent variables used in the multiple regression equation, was greater than 0.01, that variable was removed from the list of independent variables, as it was considered to be statistically not significant to the prediction. Individual subject data points that were highlighted by the analysis as being highly influential to the regression equation were also removed. As a result of both of these conditions, the regression equation was subsequently re-cast (Vincent, 1995).

In the development of the final predictive equations, due regard was also taken of the combination of independent variables that provided the lowest standard error of prediction (s_{YX}) corresponding to the highest adjusted multiple coefficient of determination (R^2_{adj}).

5.2.4.3 Standardised regression coefficients (beta (ß) *weights)*

Because independent variables were inevitably derived from measures made using different units of measurement, all variables were converted into Z-scores, and the regression equation re-cast using these standardised variables. In this way it was possible to compare independent variables directly and so address

the aim of the study which was to establish which variable was the relatively more important factor relevant to the dependent variable.

5.2.4.4 Cross-validation

In the present study, data for the cross-validation groups were entered into the final predictive equation developed for each of the four swimming events, and for each individual in the relevant cross-validation group, a finishing time was predicted (FT_P). Error (residual) scores were computed by subtracting FT_P from the measured FT (FT_M) [FT_M - FT_P]. The degree of agreement between FT_M and FT_P was analysed using methods recommended by Bland and Altman (1986) and by Nevill and Atkinson (1997).

In the present study, the validity of each of the swimming predictive equations developed was evaluated by expressing the limits of agreement between FT_M and FT_P both in their 95 % limits of agreement raw score format (i.e. expressed in seconds (s)), as well as the antilogarithm ratio (i.e. dimensionless) limits of agreement.

5.3 Results

5.3.1 Normality of the original data

Despite the relatively large sample sizes, for both 100 m and 200 m events the male swimmer's data was slightly skewed positively ($Z_{skew} > +2.0$). Conversely, for both 100 m and 200 m events, the female's results meet the criteria for normality ($Z_{skew} < +2.0$). However, in all events analysed, the original data sets exhibit curves that are significantly platykurtic ($Z_{kurt} < 0.0$). The subjects that comprised the samples, were relatively homogeneous with the majority of performances recorded being from nationally and internationally ranked breaststroke swimmers. Consequently, it might be argued that performances recorded may cluster at the lower end of a limited continuum, with few swimmers performing at the upper end (i.e. world-ranked), thereby providing the positive skew. Similarly, it seems reasonable to speculate that the peak of any subsequent data curves would be likely to be more flat than normal. For these reasons parametric techniques were used for the analyses of these data.

5.3.2 Predictive equations

The regression equations predicting finishing time (FT_P) are given in Tables 5.2 - 5.5. It can be seen that they appear to be very precise (s_{YX} values range from ±0.25 s to 0.64 s) for both 100 m and 200 m breaststroke events; with little variance remaining unexplained (R^2adj values range from 98.1% to 99.6%).

Table 5.2 - Final prediction equation and data summary for the prediction of finishing time (FTp (s)) in the men's 100 m breaststroke

FT_P (s) = 95.34 + (1.05 ST (s)) - (33.4 SV (m s^{-1})) + (1.14 TT (s))

Predictor	Coefficient	*t*-ratio	*P*	ß-weight
Constant	95.34	39.12	0.00	
ST (s)	1.05	6.90	0.00	0.173
SV (m s^{-1})	-33.37	-34.65	0.00	-0.713
TT (s)	1.14	7.67	0.00	0.170

s_{YX} = ±0.36 s R^2adj = 98.1% $F(3,124) = 2244.87$ ($P < 0.00$)

Table 5.3 - Final prediction equation and data summary for the prediction of finishing time (FTp (s)) in the women's 100 m breaststroke

FT_P (s) = 94.72 + (0.76 ST (s)) - (36.5 SV (m s^{-1})) + (1.25 TT (s)) +(1.72 ET (s))

Predictor	Coefficient	*t*-ratio	*P*	ß-weight
Constant	94.72	37.31	0.00	
ST (s)	0.76	7.57	0.00	0.106
SV (m s^{-1})	-36.52	-35.00	0.00	-0.627
TT (s)	1.25	12.61	0.00	0.193
ET (s)	1.72	10.23	0.00	0.129

s_{YX} = ±0.25 s R^2adj = 99.5% $F(4,121) = 6247.24$ ($P < 0.00$)

Table 5.4 - Final prediction equation and data summary for the prediction of finishing time (FTp (s)) in the men's 200 m breaststroke

FT_P (s) = 197.5 + (0.87 ST (s)) - (74.7 SV (m s^{-1})) + (3.20 TT (s)) +(1.89 ETs))

Predictor	Coefficient	*t*-ratio	*P*	ß-weight
Constant	197.48	33.95	0.00	
ST (s)	0.87	3.29	0.00	0.055
SV (m s^{-1})	-74.74	-30.59	0.00	-0.642
TT (s)	3.20	12.77	0.00	0.426
ET (s)	1.89	5.12	0.00	0.113

s_{YX} = 0.64 s R^2adj = 99.3% $F(4,106) = 4078.51$ ($P < 0.00$)

Table 5.5 - Final prediction equation and data summary for the prediction of finishing time (FTp (s)) in the women's 200 m breaststroke

FT_P (s) = 231.4 - (95.9 SV (m s^{-1})) + (3.88 TT (s))

Predictor	Coefficient	*t*-ratio	*P*	ß-weight
Constant	231.42	53.81	0.00	
SV (m s^{-1})	-95.90	-50.21	0.00	-0.732
TT (s)	3.88	21.58	0.00	0.310

s_{YX} = 0.52 s R^2adj = 99.6% $F(2,109) = 15689.66$ ($P < 0.00$)

5.3.3 Validation of the predictive equations

In order to validate the predictive equations developed using the experimental group they were tested on an equivalent sample, the cross validation group. A comparison between FT_M with FT_P using cross-validation groups was undertaken. The analyses identified homoscedastic data in only one event - the women's 100 m (Table 5.6).

Table 5.6 - Cross validation summary

Event	r	Z_{skew}	Z_{kurt}	t-ratio	P
100 m men	0.072	-0.001	-3.2	0.36	0.72
100 m women	0.517*	0.057	-3.20	0.37	0.72
200 m men	0.034	0.048	-2.63	3.85	0.00
200 m women	0.070	1.350	-3.59	4.11	0.00

* signifies a statistically significant correlation $r_{26} = 0.479$ ($P < 0.01$)

Limits of agreement (95%) for men's and women's 100 m events were calculated as -0.51 s to +0.55 s and -0.63 s to +0.67 s respectively (Figures 5.2 and 5.3) indicating a very small error in terms of the FT_M of the cross validation group compared with the FT_P.

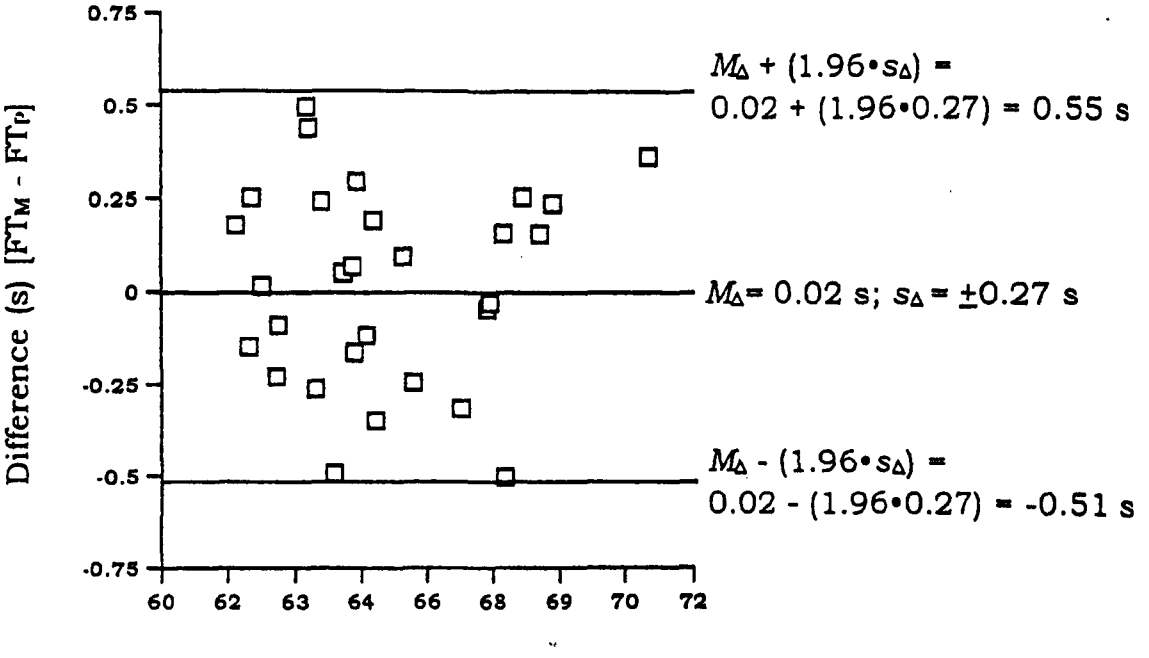


Figure 5.1 Bland and Altman plot for men's 100 m breaststroke. 95 % limits of agreement results are superimposed

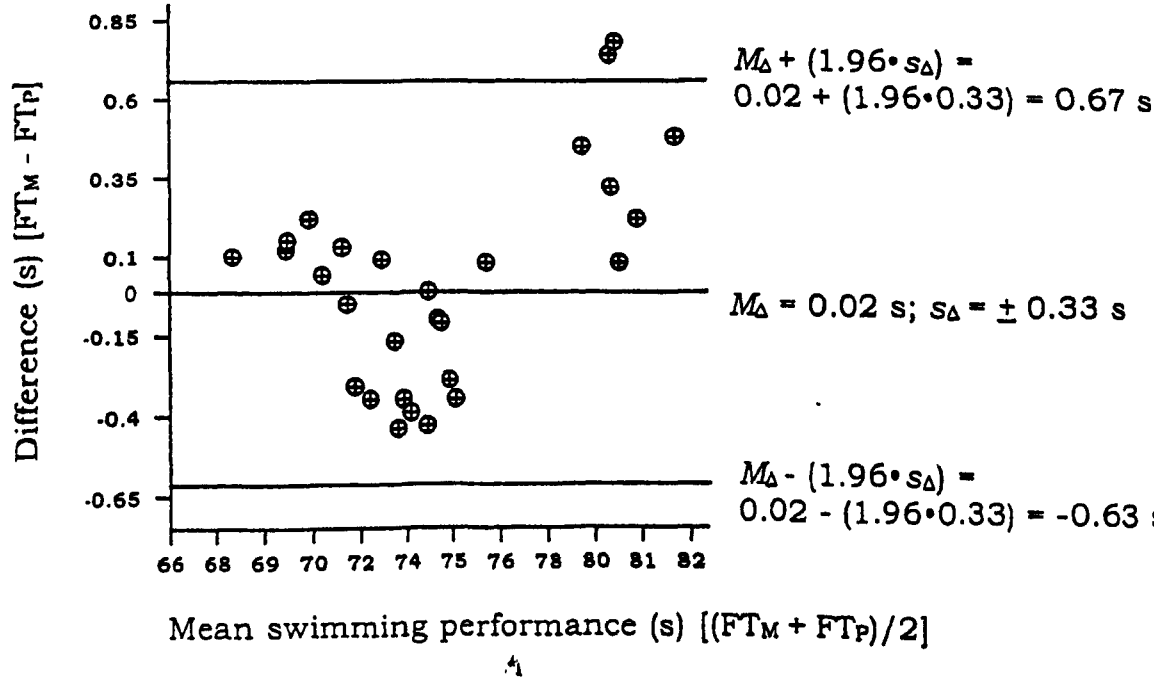


Figure 5.2 Bland and Altman plot for women's 100 m breaststroke. 95 % limits of agreement results are superimposed

These correspond to $\log_e$-transformed values of 0.992 to 1.008 (men's 100 m) and 0.992 to 1.009 (women's 100 m) (Table 5.7). Interestingly, Bland (1995) suggested that the percent difference (%D) and coefficient of variation (CV%) values generated between FT_M and FT_P should compare favourably. Indeed, in the present study these values are identical (Table 5.7).

Table 5.7 - Cross validation summary ($\log_e$-transformed data)

		95%LoA			
Event	*r*	lower	upper	% change	CV(%)
100m men	0.016	0.992	1.008	0.80	0.80
100m women	0.473**	0.992	1.009	0.88	0.88
200m men	-0.034	0.993	1.012	0.92	0.92
200m women	-0.036	0.995	1.010	0.75	0.75

** signifies a statistically significant correlation $r_{26} = 0.374$ ($P < 0.05$)

These are acceptable predictive errors for coaches of competitive swimmers, particularly when the sample groups encompass county standard to world-class swimmers (men: mean FT = 65.07 ± 2.62 s, range 60.65 to 72.35 s; women: mean FT = 74.06 ± 3.66 s, range 67.69 to 82.64 s).

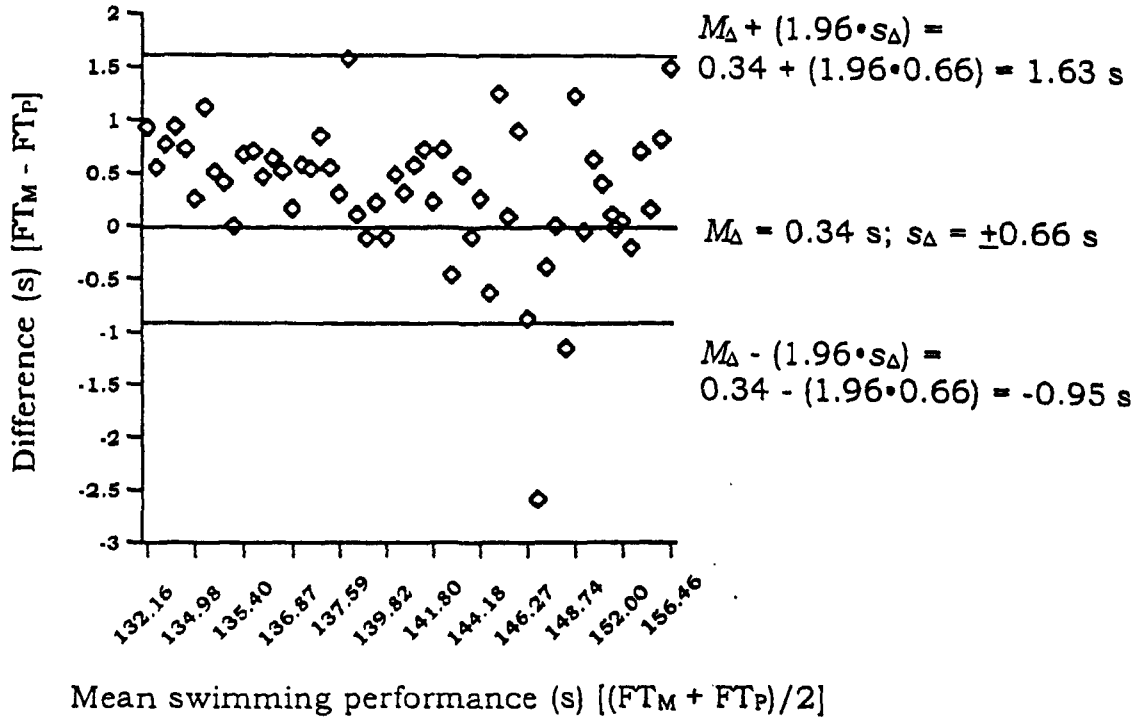


Figure 5.3 Bland and Altman plot for men's 200 m breaststroke. 95 % limits of agreement results are superimposed

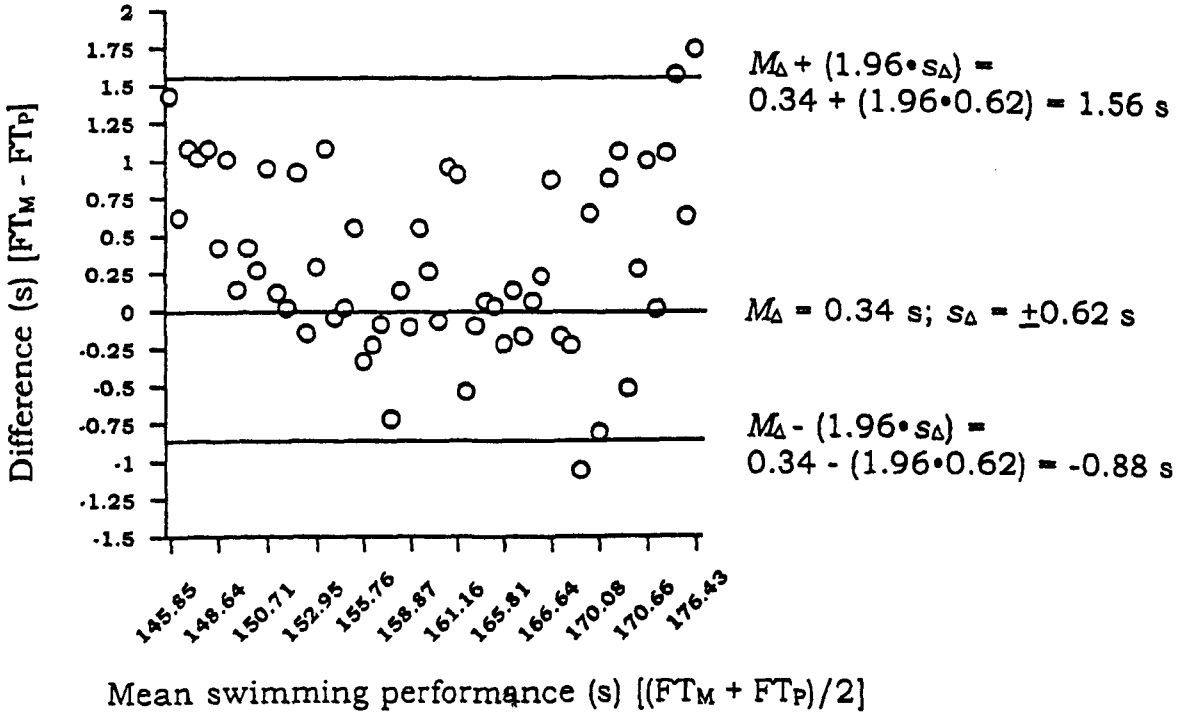


Figure 5.4 Bland and Altman plot for women's 200 m breaststroke. 95 % limits of agreement results are superimposed

Limits of Agreement for men's and women's 200 m events were -0.95 s to +1.63 s and -0.88 s to +1.56 s respectively (Figures 5.4 and 5.5). From Table 5.7 the corresponding $\log_e$-transformed values are given as 0.993 to 1.012 (men's 200 m) and 0.995 to 1.010 (women's 200 m). Once again, Bland's (1995) suggestion that the %D and CV% values generated between FT_M and FT_P should compare favourably is substantiated by the present data - these values are identically matched and confirms that the equations are valid.

From Table 5.6 and Figures 5.4 and 5.5, it can be seen that with respect to the 200 m event for both males and females, significant systematic bias resulted in a FT_P which slightly underestimated that of FT_M (men's 200m: $M_D = 0.34 \pm 0.66$ s, $t = 3.85$ ($P < 0.01$); women's 200 m: $M_D = 0.34 \pm 0.62$ s, $t = 4.11$ ($P < 0.01$)). The greater predictive error in 200 m FT is acceptable considering that a 200 m race lasts more than twice that of a 100 m race, and the analyses include two extra turning time (TT) and swimming velocity (SV) measurements, which tend to increase and decrease respectively as a race progresses (Thompson and Haljand, 1997). Finally, more heterogeneous sample groups are apparent in the 200 m events compared to those in the 100 m events (men; mean FT = 142.71 ± 7.41 s, range 132.57 - 163.27 s; women; mean FT = 159.92 ± 8.73 s, range 144.9 - 178.25 s).

5.3.4 Relative importance of kinematic and temporal variables with regard to finishing time

The aim of the study was to identify the relative importance of the independent variables (ST, SV, TT, ET, SR) with regard to FT. This was possible because the sample size provided the basis for precise predictive equations to be developed which have subsequently been shown to be valid using a cross validation group. The relative importance of the independent variables in each event can be observed in Table 5.8.

Table 5.8 - ß-weight summary for male and female 100 m and 200 m breaststroke

	Start time	Swimming velocity	Turn time	End time
100 m male	0.173	-0.713	0.170	NS
100 m female	0.106	-0.627	0.193	0.129
200 m male	0.055	-0.642	0.426	0.113
200 m female	NS	-0.732	0.310	NS

Swimming velocity demonstrated the largest ß-weight in both male and female 100 m and 200 m events justifying the premise of coaches that it is the most important element to develop in terms of race performance. Turning time possessed the second largest ß-weight in the 200 m events and the women's 100 m which suggests that apart from SV it has greater relative importance than the other temporal variables in the majority of breaststroke events. Start time demonstrated a slightly larger ß-weight

and hence greater relative importance than TT in the men's 100 m, but was relatively less important than SV, TT and ET in the male's 200 m and statistically insignificant to the prediction of FT in the female's 200 m. Finally, ET was a significant independent variable in the male's 200 m and female's 100 m, while SR was removed from all the equations as it proved to be statistically insignificant to the prediction of FT.

5.4 Discussion

Swimming velocity was the primary determinant of FT in all of the predictive equations. This finding was not unexpected due to the large portion of the race for which this variable accounts. However this finding also supports the view that more successful swimmers possess a greater SV during a race and are better able to maintain SV than less successful swimmers (Wakayoshi *et al*, 1992; Thompson and Haljand, 1997, Chapter 4). Subsequently, many elite coaches now acknowledge that when training at race pace, SV is a key programme requirement. Unfortunately, the ability of a coach to estimate a swimmer's race pace SV in training has been limited by the lack of SV measurements being taken during actual competition. However, by rearranging the regression equations offered in the present study, the coach would be able to estimate SV, providing FT_P is realistically estimated and TT and ST measurements are made in training sessions. The coach and sports scientist, using a pacing device such as the Aquapacer ™ (Challenge and Response, Inverurie, Scotland), could accurately pace a swimmer's predicted SV and monitor the ability of the swimmer to achieve an FT_P.

Turning time was the second most important determinant of FT_P except in the men's 100 m event where it was displaced by ST. The importance of TT is supported by data reported by Thompson and Haljand (1997) who compared British and European finalists in the women's 100 m, 200 m and men's 200 m events and observed that differences in TT accounted for 17.6%, 20.1% and

29.3% of the time difference in FT_M respectively. They also found that differences in TT in the men's 100 m event were less obvious, as they accounted for only 10.4% of the difference in FT_M. Therefore it appears that in the men's 100 m the relationship between TT and FT is poorer than in the other events.

The greater significance of the relationship between TT and FT over the 200 m event compared with the 100 m event is logical for a number of reasons. Firstly, two extra turns are completed in the longer race. Secondly, TT s have been observed to increase as a race progresses, suggesting a close inverse relationship with SV, because SV tends to decrease as a race progresses. For example, Thompson and Haljand (1997) noted that, European male and female 200 m finalists took 0.59 s and 0.50 s longer respectively to complete their turns on the final length compared to the first length of a race, whereas the TT s of British and Welsh finalists slowed to a greater extent (0.64 - 0.76 s). This demonstrates that the faster turning European finalists were also better able to maintain their TT s during the race which adds to the relative importance of turning in a 200 m race.

The turns are clearly important kinematic components during breaststroke races and so their measurement and subsequent monitoring should provide meaningful information. For example the coach may wish to simulate a race pace TT using a pacing device to ensure a correct SV into the turn, in order to determine if the swimmer is technically proficient. The coach may also wish to

substitute a simulated race pace TT into the regression equations generated from this study in order to predict a FT or SV.

Turning time can easily be measured by the coach provided that reliable and precise hand timed measurements are made, and that pool-side markings (7.5 m out from the turn) are accurate. In this instance the coach should also consider that TT s will take 0.5 - 0.8 s longer to complete as a 200 m race progresses, depending on the standard of the swimmer. A realistic estimate of a mean TT would be (e.g. simulated race pace: *international swimmers* = TT + 0.25; *county standard swimmers* = TT + 0.40 s), to account for the variation in the turning times over the 200 m race distance, before using the equations.

A swimmer's SV over the last 5 m of a race (the end time (ET)) is thought to be an important technical element, because a prolonged glide to the finish will result in a marked drop off in SV. However in our analysis, ET was a significant independent variable in two of the final predictive equations, (women's 100 m (Table 5.8) and men's 200 m (Table 5.8) where it held only the third largest - ß-weight of the independent variables present in those equations. This might be because ET is influenced by the SV in the latter part of the final length of any race, and as a result is adding relatively little to the predictive power of the equation. Thompson and Haljand (1997) supported this view when they observed that European breaststroke finalists possessed a greater SV in the final length and a lower ET when compared to Welsh and British finalists. Finally, the effect of ET in the present regression equations may be limited by

the fact that values for ET ranged from 3.18 - 4.64 s which constitutes a relatively small proportion of races taking between 60.85 - 178.25 s to complete.

Following further analysis, it was decided that ET could be excluded, from the two regression equations in which it remained, because the predictive power of the re-cast regression equations were found to be largely unaffected by its absence. This finding is important, as the exclusion of ET aids the practical application of these equations to the swimming coach. A realistic measurement of ET would require a maximal effort by the swimmer over the full race distance, which would preclude the necessity for a predictive equation. Also, the coach would require sophisticated equipment to accurately measure ET as the errors possible in hand-timing would be too large over the time duration being measured (i.e. < 4.0 s). Re-cast regression equations excuding ET were found to be:

Women's 100 m (equation based on $n = 125$):

$FT_P(s) = [110.8 + (0.79 \bullet ST(s)) - (43.2 \bullet SV(m\ s^{-1})) + (1.15 \bullet TT(s)]$

$s_{YX} = 0.34$ s; R^2adj = 99.1%

Men's 200 m (equation based on $n = 111$):

$FT_P(s) = [208.5 + (0.73 \bullet ST(s)) - (79.3 \bullet SV(m\ s^{-1})) + (3.50 \bullet TT(s)]$

$s_{YX} = 0.71$ s; R_2adj = 99.2%

The relative importance of ST was most obvious in the men's 100 m event. Logically, given that the FT for the 100 m event is less than half that of the 200 m event, the relative importance of ST might be expected to increase for the 100 m event. However, the mean ST, in the men's 100 m, was 0.29 s less than the mean ST for the 200 m event; a finding which has been observed previously (Thompson and Haljand, 1997; Chapter 4). This anomaly is intriguing as it might be expected that there should be no obvious difference in terms of technical proficiency between the events. Although, this finding might reflect that a greater emphasis is placed on starting practise in the training of specialist 100 m swimmers. Also, the swimmer's race strategy might be an issue, in that the 100 m swimmer would always be trying to achieve a faster split time, and hence may be focusing more strongly at the start and be more likely to achieve a faster ST.

Finally, SR was poorly correlated with FT, and so was eliminated from the analysis. This finding is in agreement with a number of other studies (Kennedy *et al.*, 1990; Chegalur and Brown, 1992; Wakayoshi, *et al.*, 1992; Chapter 4) and adds further support to the view that a swimmer will tend to adopt a unique SR : SL combination (Kennedy *et al.*, 1990; Chengalur and Brown, 1992; Wakayoshi *et al.*, 1992).

5.6 Conclusions

The independent variables (SV, TTand ST) in the regression equations were strong predictors of FT in both 100 m and 200 m events. Beta-weights suggest SV to be the most powerful determinant of FT in all equations. Turn time was found to be of particular importance in the 200 m events whilst ST was significant in the men's 100 m. End time featured in only two of the final predictive equations from which it's removal was suggested on practical grounds. Finally, given a working knowledge of these equations, and the ability to reliably and precisely measure the independent variables required, it is possible for coaches to predict, within acceptable limits, a FT for breaststroke swimmers ranging in ability from county to world-class standard. Alternatively, the coach can predict SV, having estimated a realistic FT_P, for the accurate prescription of race pace training.

Chapter 6

6a Assessment of the precision of self-paced sub-maximal 200 m breaststroke swimming

6b Establishing the precision and reproducibility of sub-maximal 200 m breaststroke swimming using a novel pacing device (Aquapacer™); and assessment of the reproducibility of the associated metabolic and kinematic responses

6.1 Introduction

Reliability has been defined as the "consistency of measurements of an individual's performance on a test, or the absence of measurement error" (Atkinson and Nevill, 1998a). In reality error will always be present but providing the measurement instrument or individual performance is precise enough then the error may be deemed acceptable. Therefore when test-retest experiments are conducted with exercising human subjects at a given exercise intensity over a short period of time (eg. 3-5 days) it is a important that any repeated measurements taken during or after each exercise bout are both precise and reproducible. This is known as stability reliability where there is consistent day to day variability (Baumgarter, 1989), and without this findings may be interpreted as being due to measurement error or inter-daily biological variation. Large errors in this aspect would preclude the scientist from attempting any interventions.

Two studies in this thesis manipulate the pacing of swimmers (the independent variable) in order to determine the associated metabolic and kinematic responses (dependent variables). Therefore a fundamental requirement was to establish the precision and reproducibility of pacing during breaststroke swimming before experimental manipulations were introduced.

Historically there has not been a gold standard method of pacing in swimming. Rather, in swimming squads, the coach dictates the target time, while the actual

precision of the pacing is left to the swimmer's judgement, albeit aided by cursory glances toward the pool-side clock. As a consequence a swimmer may develop a fairly accurate sense of pacing as a result of being chronically involved in this practise. However, the degree of precision and reliability achieved has not been formally investigated.

In recent years a small number of investigations have attempted to externally control pacing. Swimming Flumes have been used since the 1970 s (Astrand and Englesson, 1972; Holmer and Hagland, 1978); however they have proven to be expensive and do not allow swimmers to swim with the same technique as they would during "free swimming" nor can turns be incorporated into experiments. Hence cheaper and more practical alternatives have been investigated. These methods tend to fall into two categories - audible pacing signals and light sequencing (Hallowell, 1983; Montpetit *et al.*, 1983; Costill *et al.*, 1985; Sano *et al.*, 1990). Unfortunately both of these systems have been reported to be problematic due to the signal being inaudible or out of sight during a large proportion of each stroke cycle (Montpetit *et al.*, 1983; D'Aquisto *et al.*, 1988).

Recently a novel audible pacing system (Aquapacer™, Challenge and Response, Inverurie, Scotland) has been developed which was designed to overcome the problems associated with the other pacing systems. Martin and Thompson (1999, 2000) have reported the system to elicit precise and reliable pacing during sub-maximal freestyle swimming. Following such encouraging

results it was decided to investigate the Aquapacer™ as the method of choice for pacing in this thesis.

Few studies in the literature have attempted to establish within subject variability of metabolic and kinematic responses during sub-maximal and maximal exercise. Taylor (1944) reported within subject variability for heart rate (HR), minute ventilation ($\dot{V}E$) and oxygen uptake ($\dot{V}O_2$) of 4.1 %, 8.0 % and 6.5 % during sub-maximal exercise and 1.6 %, 7.2 % and 7.6 % during maximal exercise respectively. Henry (1951) observed a 7.5 % variation in $\dot{V}O_2$ during sub-maximal exercise whereas inter-daily variation in $\dot{V}E$ and $\dot{V}O_2$ have been reported to be < 4 % in sub-maximal cycling (Armstrong and Costill, 1985), while a coefficient of variation of < 3 % has been reported for $\dot{V}O_2$ data during treadmill exercise (Morgan *et al.*, 1991; Williams *et al.*, 1991). However in the first swimming study to date to have used the Aquapacer™, Martin and Thompson (2000) reported greater variability in ventilatory and blood lactate measurements to those previously reported. They also reported acceptable reproducibility for both stroke rate and stroke count. However their study was based on sub-maximal freestyle swimming and so their findings may not be applicable to high intensity breaststroke swimming.

Therefore, the aims of the 2 linked studies in this chapter were to ascertain :

i) if breaststroke swimmers could pace precisely using a poolside clock (Study 6a);

ii) if breaststroke swimmers could pace precisely and reliably with the Aquapacer™ (Study 6b); and

iii) if measurements of kinematic variables (stroke rate and stroke count), metabolic responses (heart rate, capillary blood lactate and ventilatory measurements) and Rating of Perceived Exertion (RPE) would be reliable, if pacing was proven to be precise and reliable (Study 6b).

6.2 Method

Study 6a - Assessment of the precision of self-paced sub-maximal 200 m breaststroke swimming

6.2.1 Subjects

Fifteen male swimmers were recruited from the Welsh national swimming squads and three swimming clubs in South and West Wales (Table 6.1). Prior to completing the study subjects were fully informed about the demands and procedures of the study and gave their written consent to participate. A health screening questionnaire was administered (UWIC Physiology laboratory procedure) and scrutinised prior to subjects being permitted to begin the studies (Appendix 1). Subjects then read a Volunteer Consent form, outlining the purpose and procedure of the study and gave informed consent prior to beginning the study. Ethical approval for each study was granted by the Ethics Committee of Liverpool John Moores University.

Subjects were informed that they should not attempt a test unless they were in good health and that they could terminate an exercise test at any time. They were asked to report to the laboratory in a rested state having completed no exercise or only very light exercise the previous day. Self report diaries were issued to confirm this when studies took a number of days to complete. They were also asked to abstain from alcohol, caffeine and fatty foods on the day of

testing, and not to consume food in the three hours before a test. Information was also given about how to ensure euhydration prior to testing.

Table 6.1 Physical and anthropometric characteristics of the subjects.

Subject	Age (yrs)	Height (m)	Body mass (kg)	200 m Time trial time (s)
AA	21	1.74	72.3	150
AC	22	1.76	72.3	142
AN	31	1.83	80.9	146
BG	19	1.80	76.5	142
JO	18	1.83	78.9	152
BH	20	1.84	78.3	142
TH	22	1.82	81.0	150
KJ	23	1.72	76.2	146
KT	24	1.78	79.6	158
MO	22	1.76	81.2	148
ST	20	1.75	69.6	148
PA	19	1.79	74.2	152
SR	25	1.80	80.3	142
JI	24	1.84	83.2	150
JK	23	1.81	78.6	142
Mean	22	1.79	77.5	147.3
± s	± 3	± 0.03	± 3.9	± 4.8

6.2.2 Experimental design

Subjects completed two, 200 m sub-maximal breastroke swims 5 minutes apart, approximating to 92 % and 95 % of a maximal 200 m effort. Subjects used a poolside clock to judge their pace. Hand timings of 100 m split times and 200 m finishing times were taken and compared with predicted finishing times.

6.2.3 Protocol and conduct of the study

From an estimated finishing time for a maximal 200 m breaststroke time trial (200_{TT}), target times were calculated for two, 200 m sub-maximal breaststroke repetitions as follows:

Repetition 1 Target time (s) = estimated 200_{TT} time (s) + 12 s

Repetition 2 Target time (s) = estimated 200_{TT} time (s) + 8 s

Following a self selected warm-up each swimmer completed the two, 200 m sub-maximal breaststroke repetitions with 5 minutes recovery. A verbal instruction of the target time was given to the swimmers immediately prior to each repetition. Otherwise the swimmers swam both repetitions unaided, except for the presence of a pool side clock, which they were able to observe during the repetitions. The times to complete each 100 m and 200 m were recorded for each repetition by hand timing.

Study 6b - Establishing the precision and reproducibility of sub-maximal 200 m breaststroke swimming using a novel pacing device (Aquapacer™); and assessment of the reproducibility of the associated metabolic and kinematic responses.

6.2.4 Subjects

Ten male swimmers were recruited from either the Welsh national swimming squad or the UWIC swimming team (Table 6.2). Subject preparation was as outlined in *6.2.1* except that a phlebotomy questionnaire was also administered (UWIC Physiology laboratory procedure) and scrutinised prior to the subjects beginning the study.

The stature and body mass of subjects were measured using a portable stadiometer (Holtain) and electronic weighing scales (Seca 770). Skinfold measurements were taken with skinfold calipers (John Bull, British Instruments Ltd, England) according to BASES Physiological Testing Guidelines (Bird and Davison., 1987).

Table 6.2 Physical and anthropometric characteristics of the subjects.

Subject	Age (yrs)	Height (m)	Body mass (kg)	Sum of 4 skinfolds (mm)	200 m Time Trial time (s)
JI	21	1.82	72.6	21.5	193.0
KE	20	1.81	76.4	29.8	170.4
SA	20	1.62	64.2	38.0	189.6
SI	19	1.68	65.8	35.5	186.1
HA	21	1.74	69.8	23.6	160.9
IA	22	1.76	84.2	54.2	180.0
IAI	21	1.80	82.2	30.5	185.2
AN	31	1.83	80.9	29.7	145.2
JO	18	1.83	78.9	27.5	151.3
PA	19	1.79	74.2	28.9	153.6
Mean	21	1.77	74.9	31.9	171.5
± s	± 4	± 0.07	± 6.9	± 9.2	± 17.6

6.2.5 Experimental design

Subjects completed a self paced maximal 200 m breaststroke time trial (200_{TT}) from a push start. From the finishing time, the time to complete 200 m breaststroke repetitions at 85 % and 95 % of the 200_{TT} pace were calculated. Seventy two hours later subjects swam two, 200 m breaststroke repititions at these calculated paces. A pacing device (Aquapacer™, Challenge and Response, Inverurie, Scotland) was used to pace the swimmers accurately at the calculated paces (Chapter 3.1). The actual times taken for the subjects to complete 100 m and 200 m were measured to establish the precision of pacing from a comparison with the predicted time. Kinematic (stroke rate, stroke count), metabolic (heart rate, blood lactate and gas exchange variables) and Rating of Perceived Exertion (RPE) reponses were also measured. Subjects

then repeated the two, 200 m efforts on two further occasions 48 hours apart in order to establish the reproducibility of pacing and the associated kinematic, metabolic and RPE responses.

6.2.6 Protocol and conduct of the study

6.2.6.1 Calculation of the pace of the trials

Following a self selected warm up comprising of at least 800 m of swimming each subject completed a timed maximal 200_{TT} from a push start. Times representing 85 % and 95 % of the 200_{TT} pace were then calculated as:

85 % of 200_{TT} pace = 1.15 (200_{TT} time (s))

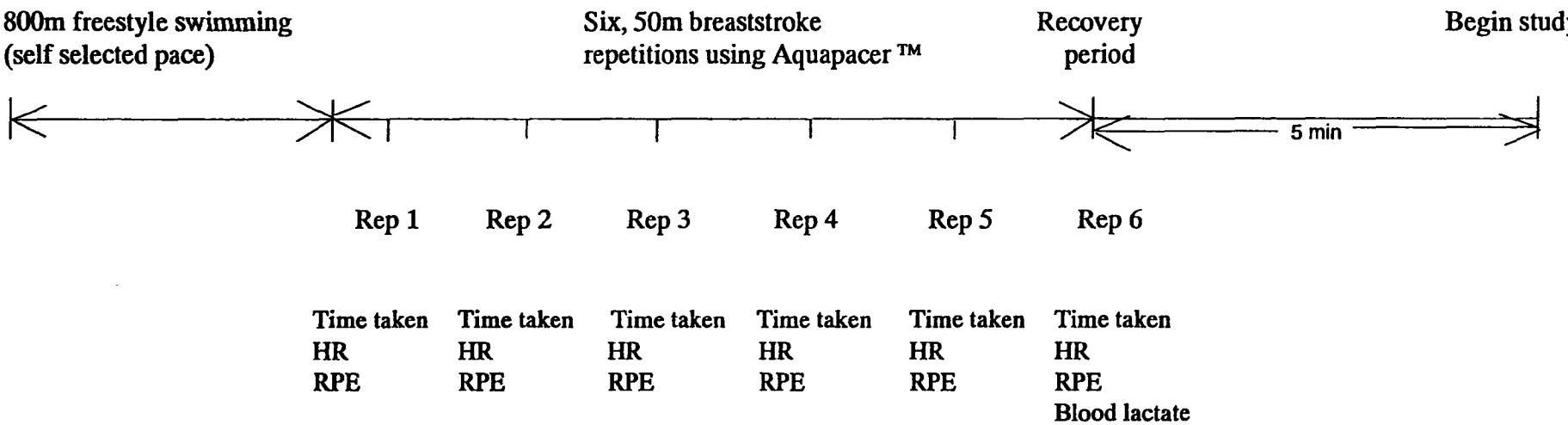
95 % 200_{TT} pace = 1.05 (200_{TT} time (s))

6.2.6.2 Standardised warm-up

At least 72 h after the 200_{TT}, the subjects completed a warm up consisting of 800 m of self-paced freestyle swimming followed by six, 50 m repetitions of breaststroke swimming with 30 s recovery in between. The Aquapacer™ was used to pace the breaststroke repititions. The target time for each 50 m repetition was calculated as : 200_{TT} time + 20 s / 4. The Aquapacer™ sounding unit was then programmed to emit a bleep (to the nearest 0.01 s) to coincide with the swimmer progressing every 12.5 m, either to a pool-side marker placed

at 12.5 m along the length or to coincide with the swimmer's feet touching the wall at the turn. To facilitate this the swimmers were instructed to always "turn on the bleep by ensuring that their feet contacted the wall as the bleep sounded". The swimmer was also instructed to push off gently from the wall at the start of each repetition so as to not get ahead of the pacing signal. The practise of pacing every 12.5 m and instructing subjects to avoid a powerful push off at the start of each trial were adopted for each study thereafter.

After programming the Aquapacer™, it was placed in the swimmer's cap just behind the ear so as to be easily heard and not to interfere with the placement of the swimmer's goggles. Hand timed finishing times (Actual $_{FT}$) were taken at the end of each repetition to judge the habituation of the swimmer to the Aquapacer™. Heart rate (Polar Sports tester) and RPE scale (Borg, 1986) measurements were taken within 5 s of the completion of each repetition to ensure that the effort was light-moderate and constant throughout (Chapter 3.2 and 3.5). A capillary blood lactate sample was taken from the subject's earlobe into a lysing tube within 45 s of the final repetition of the warm-up finishing. An attempt was made to ensure that samples were taken as close to 30 s post exercise as possible because blood lactate concentrations below and approximating to the lactate threshold have been previously shown to remain steady during this time period (Gullestrand *et al.*, 1994). The blood sample was thoroughly mixed before being capped and placed on an ice pack and transported to a fridge for storage. Prior to any further swimming the subject was given a 5 minute recovery period.

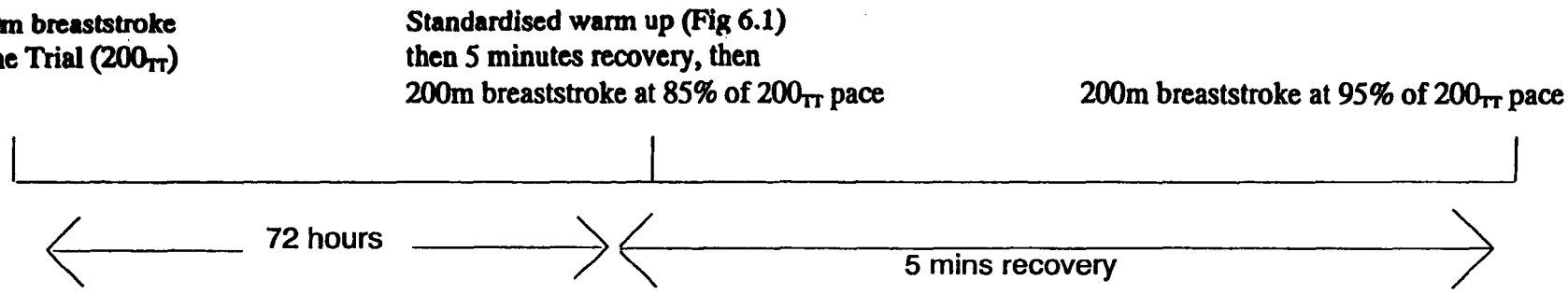


* 50 metre times were measured to check habituation to pacing.

* HR and RPE were taken <5 s after each repetition and a capillary blood sample was taken <45s after repetition six to determine blood lactate concentration. Data was used to confirm that the effort was light-moderate.

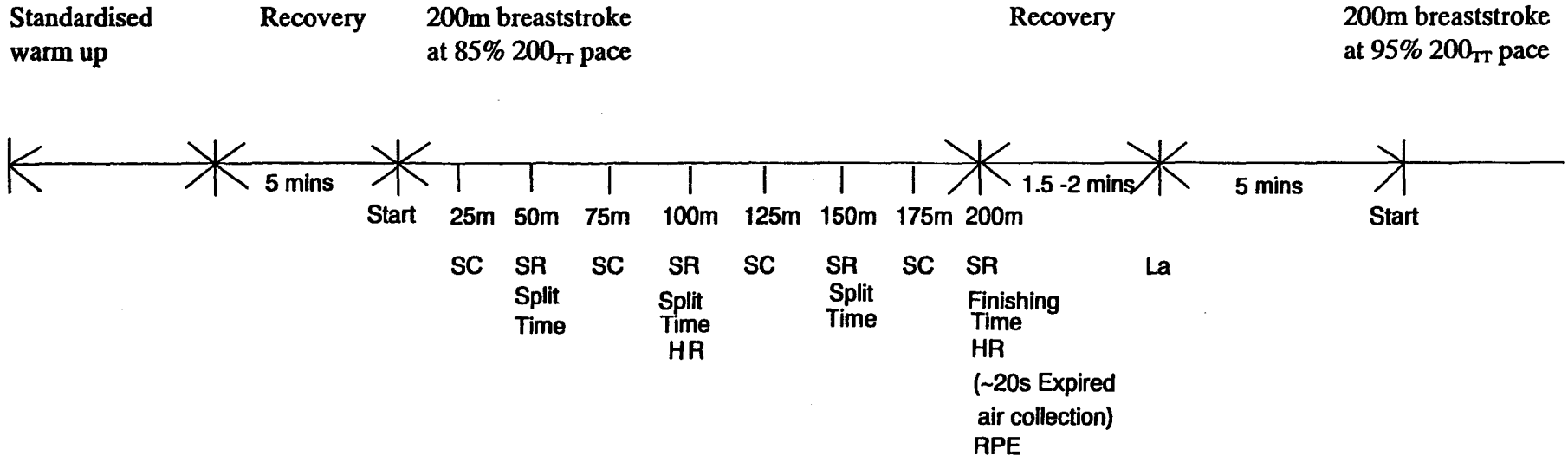
* Key : HR - heart rate, RPE - Rating of Perceived Exertion, La-blood sample taken for lactate determination.

Fig 6.1 Standardised warm-up



The two submaximal 200m breaststroke swims were then repeated 48 hours and 96 hours later.

Fig 6.2 Experimental design



*

All measurements taken during the 200m breaststroke swim at 85% 200_{TT} pace were repeated during the 200m breaststroke swim at 95% 200_{TT} pace.

Key : SC-stroke count, SR-stroke rate, HR-heart rate, 20s Expired air collection for determination of $\dot{V}O_2$, $\dot{V}CO_2$ RER and $\dot{V}E$, La-blood sample taken for lactate determination, RPE - Rating of Perceived Exertion.

Fig 6.3 Timecourse of measurements

6.2.6.3 Trial procedures

The Aquapacer™ was then re-programmed to pace the swimmer's impending 200 m effort at a pace which would elicit the 85 % of the 200_{TT} time, with audible bleeping signals set to coincide with every 12.5 m throughout the trial.

Five seconds prior to the swimmer beginning the 200 m effort the heart rate monitor's memory function was activated to record at 5 s intervals, so as to allow the precise estimation of the heart rate at the 100 m point. During the trial stroke rate was recorded during the 2nd, 4th, 6th and 8th lengths (Chapter 3.6), while stroke count was recorded during the 1st, 3rd, 5th and 7th lengths. Stroke count was recorded by an experienced investigator who counted only full and half strokes (Chapter 3.7). Hand timing measurements were taken every 50 m to ensure pacing was accurate, although only the 100 m and 200 m times were recorded in the results. Subjects were instructed to hold their breath during the final stroke (Costill *et al.*, 1985). A Hans Rudolph, full face mask, was then placed firmly over the swimmer's nose and mouth to ensure a good seal, whereupon on the instruction 'GO' the valve to an evacuated Douglas bag was opened and the subject expired air into it for 20 s through low resistance Falconia tubing. During this procedure the subject's heart rate was recorded (~ 5 s post swim) and checked against the recorded value from the memory function, at a later date. RPE was then recorded, before a single capillary blood lactate sample was then taken from the same earlobe, as previously described,

between 1.5 - 2 minutes after swim cessation. The same procedure as described after the warm -up was then repeated, except that this time the subject was paced to achieve a target time of 95 % 200_{TT}. Each swimmer repeated the protocol on two further occasions, 48 hours apart, at the same time of day (± 60mins) to minimise diurnal biological variation (Reilly *et al.*, 1984).

The Douglas bags were analysed, within 2 hours of the event, for percentage O_2 and CO_2 composition (Servomex 1440C) and volume (Harvard Dry Gas Meter) (Chapter 3.3). Volumes of $O_{2\ ATPS}$ and $CO_{2\ ATPS}$ were calculated and converted to values in STPD, using the "Expair" software package. In order to complete this calculation the subject's age, height and body mass had to be entered along with the temperature (^{0}C) of the expired air as it passed through the Harvard Dry Gas Meter and the laboratorie's atmospheric temperature (mmHg). Absolute values for $\dot{V}O_{2STPD}$ were then re-calculated in order to estimate the oxygen uptake occurring at cessation of exercise bearing in mind that a 20 s post exercise expired air collection would underestimate $\dot{V}O_{2STPD}$ at exercise cessation due to the rapid onset of oxygen recovery kinetics. A predictive equation reported by Costill *et al.* (1985) was used to predict the $\dot{V}O_{2STPD}$ at exercise cessation using the actual $\dot{V}O_{2STPD}$ value from the 20 s post exercise expired air collection. The predictive equation was:

$y = 0.916\ x + 0.426$

where:

y = the predicted $\dot{V}O_{2STPD}$

x = the actual $\dot{V}O_{2STPD}$ measured from a 20 s post exercise expired air collection.

Finally, Blood lactate samples were analysed within 24 hours of the event (Analox GM7) having been removed from the fridge for 30 minutes and remixed once the caps were removed from the ends of the lysing tubes (Chapter 3.4). Duplicate samples were measured in all cases.

6.2.7 Statistical analyses

All procedures were undertaken using the Minitab v11 and SPSS v7.5 software packages. A level of significance of $P<0.05$ was adopted throughout. The Anderson-Darling Test was used to test for normality to ensure that parametric analyses could be used. Guidelines for adopting parametric repeated measures analysis, such as considering sphericity as the main assumption to be upheld, were reviewed prior to analysis. Dependent t-tests and One-way ANOVA were utilised to establish if a large systematic bias (relative to random error) was present while the terms implicit in both tests were used in the calculation of random error (Atkinson and Nevill, 1998). If a significant bias was observed

then Tukey's HSD post-hoc test was adopted (for ANOVA) and the omega-squared statistic was used to estimate the meaningfulness of the finding.

In order to estimate random error, 95 % Limits of Agreement (LoA, Bland and Altman, 1986) were adopted to establish precision of the pacing (actual finishing times (FT_{Act}) compared with predicted finishing times (FT_P) and to calculate the reliability of pacing, kinematic and metabolic reponses across the three trials (Nevill and Atkinson, 1998).

Before calculating the 95 % LoA, to assess the precision of the pacing (Study 6a and Study 6b), a Bland and Altman plot (Bland and Altman, 1986) was produced to provide an indication of systematic bias and random error (Atkinson and Nevill, 1998). The presence of heteroscedascity (where the residual errors are proportional to the magnitude of the measured value) was then investigated from a plot of absolute residual errors against individual means and the calculation of a correlation coefficent. A significant positive correlation confirmed the presence of heteroscedascity. The absolute differences were then tested for normality (Anderson-Darling test). Providing that heteroscedascity and an abnormal distribution for residual errors were not detected then absolute 95 % LoA were calculated in the actual units of measurement. The 95th percentile was convened as recommended by the British Standards Institute for presenting reliability data (Atkinson and Nevill, 1998). Even if no significant systematic bias was detected (dependent t-test)

any observable bias was reported so as to express "total error" (bias and random error) as recommended by Atkinson and Nevill (1998).

If heteroscedascity or an abnormal distribution were observed then logarithmic (natural) transformation of the data was to be undertaken prior to the calculation of 95 % LoA (Bland and Altman, 1986). Anti-logging the data would then provide ratio LoA expressed on a ratio scale (*/- 95 % LoA). Previous tests for bias would also be required to be performed on log transformed data in this eventuality.

In order to calculate agreement across 3 trials (Study 6b), ANOVA was used (Bland, 1996). ANOVA identifies systematic bias and also estimates within-subject measurement error (S_w^2, Nevill and Atkinson, 1998). If no evidence of heteroscedascity was detected from:

i) redidual errors (between trials) against fitted values, and

ii) there was no significant correlation between absolute residuals against the fitted values, and

it had been determined that the absolute residual errors were normally distributed (Anderson Darling plot); then the random error components of 95 % LoA were calculated as:

95 % LoA = ± 1.96 (SQR (2* S_w^2))

Once again ratio LoA were to be calculated if the residual errors were either not normally distributed or demonstrating heteroscedascity.

6.3 Results

Table 6.3 - Agreement between actual finishing times (Actual $_{FT}$) and predicted finishing times (Predicted $_{FT}$) (mean ± s) for the 200 m self paced sub-maximal trials (Study 6a)

Comparison	$200_{TT\ FT}$ + 12 s (~92 % $200_{TT\ FT}$)				$200_{TT\ FT}$ + 8 s (~95 % $200_{TT\ FT}$)			
	FT (s)	t	Sig	95% LoA	FT (s)	t	Sig	95% LoA
Actual $_{FT}$	159.5 ± 5.9	-0.12	NS	- 0.2 ± 2.7	155.0 ± 4.9	-0.06	NS	- 0.4 ± 2.2
vs								
Predicted $_{FT}$	159.7 ± 5.1				155.3 ± 4.8			

Table 6.4 - Agreement between actual finishing times (Actual $_{FT}$) and predicted finishing times (Predicted $_{FT}$) (mean ± s) for the 200 m sub-maximal trials (Study 6b)

Comparison	85 % 200_{TTFT} trial				95 % 200_{TTFT} trial			
	FT (s)	t	Sig	95% LoA	FT (s)	t	Sig	95% LoA
Trial 1								
Actual $_{FT}$	197.4 ± 20.3	1.04	NS	0.2 ± 1.0	179.5 ± 18.5	8.09	P<0.01	-0.6 ± 0.4
Predicted $_{FT}$	197.3 ± 20.3				180.1 ± 18.5			
Trial 2								
Actual $_{FT}$	197.4 ± 20.2	0.73	NS	-0.1 ±1.2	179.8 ± 18.6	0.90	NS	-0.3 ±1.8
Predicted $_{FT}$	197.3 ± 20.3				180.1 ± 18.5			
Trial 3								
Actual $_{FT}$	196.9 ± 20.1	-2.22	NS	-0.4 ± 0.8	179.7 ± 18.5	1.19	NS	-0.3 ± 1.7
Predicted $_{FT}$	197.3 ± 20.3				180.1 ± 18.5			

85 % 200_{TT} trial means a submaximal 200 m trial at 85 % of the pace of a maximal 200 m time trial

Table 6.5 - Agreement between actual 100 m split times (Actual $_{SpT}$) and predicted 100 m split times (Predicted $_{SpT}$) (mean ± s) during the sub-maximal 200 m trials (Study 6b)

Comparison	85 % $200_{TT\ FT}$ trial				95 % $200_{TT\ FT}$ trial			
	ST (s)	t	Sig	95% (LoA)	ST (s)	t	Sig	95 % (LoA)
Trial 1								
Actual $_{SpT}$	99.2 ± 10.4	2.69	NS	0.5 ± 1.2	89.8 ± 9.3	-1.15	NS	0.0 ± 1.4
Predicted $_{SpT}$	98.6 ± 10.1				90.1 ± 9.3			
Trial 2								
Actual $_{SpT}$	99.0 ± 10.5	1.46	NS	0.3 ± 1.8	89.5 ± 9.6	-1.78	NS	-0.5 ± 1.9
Predicted $_{SpT}$	98.6 ± 10.1				90.1 ± 9.3			
Trial 3								
Actual $_{SpT}$	98.5 ± 10.2	-0.86	NS	0.0 ± 0.9	89.8 ± 9.6	-1.01	NS	-0.3 ± 1.4
Predicted $_{SpT}$	98.6 ± 10.1				90.1 ± 9.3			

Table 6.6 - Reliability of actual finishing times (FT_{Act}) (mean ± s) over the three trials (Study 6b)

Comparisons	F statistic	Sig	95% LoA (s)
85 % $200_{IT\ FT}$ Trials Trial 1(FT_{Act}) vs Trial 2 (FT_{Act}) vs Trial 3 (FT_{Act})	2.42	NS	± 1.66
95 % $200_{IT\ FT}$ Trials Trial 1(FT_{Act}) vs Trial 2 (FT_{Act}) vs Trial 3 (FT_{Act})	0.46	NS	± 1.84

Table 6.7 - Reliability of actual 100 m split times (SpT_{Act}) (mean ± s) over the three trials (Study 6b)

Comparisons	F statistic	Sig	95% LoA (s)
85 % $200_{IT\ FT}$ Trials Trial 1(SpT_{Act}) vs Trial 2 (SpT_{Act}) vs Trial 3 (SpT_{Act})	3.31	NS	± 1.66
95 % $200_{IT\ FT}$ Trials Trial 1(SpT_{Act}) vs Trial 2 (SpT_{Act}) vs Trial 3 (SpT_{Act})	0.772	NS	± 1.67

Table 6.8 - Reliability of metabolic responses (mean ± s) over the three, 85 % 200_{PT} trials (Study 6b)

Variable	Mean ± s	F statistic	Significance	95 % LoA
Heart rate ($b.min^{-1}$)				
HR at 100 m				
Trial 1	151 ± 11	2.58	N.S	± 7
Trial 2	152 ± 10			
Trial 3	149 ± 10			
HR at 200 m				
Trial 1	158 ± 13	0.31	N.S	± 8
Trial 2	157 ± 9			
Trial 3	157 ± 13			
Lactate (mM)				
Trial 1	4.0 ± 1.5	0.89	N.S	± 1.3
Trial 2	3.7 ± 1.1			
Trial 3	3.8 ± 1.4			
$\dot{V}O_{2\,STPD}$ ($l.min^{-1}$)				
Trial 1	2.80 ± 0.74	0.68	N.S	± 0.33
Trial 2	2.84 ± 0.73			
Trial 3	2.77 ± 0.70			
Pred $\dot{V}O_{2\,STPD}$ ($l.min^{-1}$)				
Trial 1	2.99 ± 0.68	0.65	N.S	± 0.33
Trial 2	3.02 ± 0.67			
Trial 3	2.96 ± 0.65			
$\dot{V}CO_{2\,STPD}$ ($l.min^{-1}$)				
Trial 1	2.67 ± 0.97	0.12	N.S	± 0.87
Trial 2	2.73 ± 0.87			
Trial 3	2.69 ± 0.71			
$\dot{V}E_{STPD}$ ($l.min^{-1}$)				
Trial 1	70.27 ± 25.75	2.38	N.S	± 9.28
Trial 2	72.13 ± 25.50			
Trial 3	73.53 ± 27.91			
RER				
Trial 1	0.98 ± 0.12	0.36	N.S	± 0.17
Trial 2	0.96 ± 0.12			
Trial 3	0.95 ± 0.11			

Table 6.9 - Reliability of metabolic responses (mean ± s) over the three, 95 % 200_{TT} trials (Study 6b)

Variable	Mean ± s	F statistic	Significance	95 % LoA
Heart rate ($b.min^{-1}$)				
HR at 100 m				
Trial 1	161 ± 10	2.33	N.S	± 5
Trial 2	163 ± 10			
Trial 3	162 ± 10			
HR at 200 m				
Trial 1	170 ± 15	2.03	N.S	± 6
Trial 2	172 ± 13			
Trial 3	171 ± 14			
Lactate (mM)				
Trial 1	7.4 ± 2.4	1.97	N.S	± 1.2
Trial 2	7.4 ± 2.4			
Trial 3	7.7 ± 2.2			
$\dot{V}O_{2\ STPD}$ ($l.min^{-1}$)				
Trial 1	3.56 ± 0.75	3.43	N.S	± 0.39
Trial 2	3.48 ± 0.80			
Trial 3	3.65 ± 0.68			
Pred $\dot{V}O_{2\ STPD}$ ($l.min^{-1}$)				
Trial 1	3.67 ± 0.69	3.32	N.S	± 0.38
Trial 2	3.60 ± 0.73			
Trial 3	3.77 ± 0.62			
$\dot{V}CO_{2\ STPD}$ ($l.min^{-1}$)				
Trial 1	4.03 ± 1.07	2.02	N.S	± 0.43
Trial 2	3.96 ± 1.14			
Trial 3	4.11 ± 1.04			
$\dot{V}E_{STPD}$ ($l.min^{-1}$)				
Trial 1	91.26 ± 31.14	1.16	N.S	± 9.32
Trial 2	93.13 ± 31.53			
Trial 3	93.34 ± 31.96			
RER				
Trial 1	1.11 ± 0.12	1.93	N.S	± 0.07
Trial 2	1.12 ±0.11			
Trial 3	1.10 ±0.13			

Table 6.10 - Reliability of Rating of Perceived Exertion responses (mean ± s) over the three, 85 % 200_{TT} trials and 95 % 200_{TT} trials (Study 6b)

Variable	Mean ± s	F statistic	Significance	95 % LoA
85 % 200_{TT} RPE				
Trial 1	11 ± 2	0.57	NS	± 2
Trial 2	11 ± 3			
Trial 3	11 ± 3			
95 % 200_{TT} RPE				
Trial 1	15 ± 2	0.04	NS	± 2
Trial 2	15 ± 3			
Trial 3	15 ± 2			

Table 6.11 - Reliability of stroke rate and stroke count responses (mean ± s) over the three, 85 % 200_{TT} trials and 95 % 200_{TT} trials (Study 6b)

Variable	Mean ± s	F statistic	Significance	95 % LoA
Stroke rate ($S.min^{-1}$)				
85 % 200_{TT}				
Trial 1	27.7 ± 5.2	0.18	NS	± 0.6
Trial 2	28.0 ± 4.9			
Trial 3	28.0 ± 5.1			
95 % 200_{TT}				
Trial 1	30.6 ± 5.8	1.45	NS	± 0.5
Trial 2	31.0 ± 5.9			
Trial 3	31.1 ± 5.9			
Stroke count ($S.length^{-1}$)				
85 % 200_{TT}				
Trial 1	8.6 ± 1.7	1.21	NS	± 1.3
Trial 2	8.7 ± 1.7			
Trial 3	8.5 ± 1.6			
95 % 200_{TT}				
Trial 1	9.4 ± 1.7	3.43	NS	± 1.3
Trial 2	9.5 ± 1.6			
Trial 3	9.1 ± 1.5			

6.4 Discussion

6.4.1 Precision of pacing

The findings from Study 6a would suggest that unaided (self-paced) pacing was precise because no significant bias was detected. This result was not unexpected given that the sample was relatively homogeneous (200 m time range: min 142 s, max 158 s) of a high standard of performance (estimated mean 200 m time 147.3 ± 4.8 s), and chronically training. They might therefore be expected to be good at pacing. However, the 95 % LoA suggests that these swimmers were not pacing as precisely as a coach might expect. The limits estimated propose that accomplished swimmers pace within ± 2.2 to ± 2.7 s of their target time over a swimming duration of approximately 155 - 160 s. This may seem a relatively small error (1.4 - 1.7 %) in pace judgement, and one which may be acceptable to a coach in training situations, but it appears from the findings of Study 6b that the Aquapacer™ provides the scientist with a more precise method of pacing swimmers for experimental and fitness testing purposes.

A comparison of the 95 % LoA for unaided (Study 6a) and paced trials (Study 6b) against respective target times suggests that the Aquapacer™ is the more precise method for the pacing of breaststroke swimmers. This is highlighted particularly well in both studies when the swimmers completed a trial at 95 %

of the pace of a maximal 200 m swim. During the paced study the mean 95 % LoA, for the 3 trials, including observable bias, was -1.8 to + 1.3 s which is markedly more precise than the self paced (unaided) condition (-2.6 to +1.8 s). Indeed the comparison is more impressive when the 95 % LoA (excluding bias) are expressed as a percentage of the target time (0.9 % Aquapacer™ vs 1.7 % self-paced). This comparison proved to be just as disparate for the more slowly swum trials (0.5 % Aquapacer™ vs 1.4 % self-paced). What must also be considered here is that Study 6b utilised a more heterogeneous sample (200_{TT} times range: min 145.2 s, max 193.0 s) than Study 6a, and that subjects in Study 6b experienced a greater differential (and hence adjustment) in pacing between their two, 200 m sub-maximal repetitions.

The 95 % LoA for 100 m split times demonstrated similar results to the actual finishing times for each paced trial, although if expressed as a percentage of the predicted time there was evidence of slightly greater error. This finding suggests that over the final 100 m of the paced trials the swimmers became more precise in their judgement, perhaps because the effect of the push-off (which proved difficult for the swimmer's to judge at the start of the trial) became less influential as the trial progressed. Also the swimmer's experience of turning at the correct pace would have been increasing throughout the trial.

Surprisingly, significant bias was detected for the first paced trial at 95 % 200_{TT} pace, but this was because every swimmer in the trial completed it in a slightly faster time than was programmed (0.28 s - 0.96 s). This anomaly led to 76 %

of the total variation being explained (omega^2 = 0.76) by the paced trial. In contrast the 95 % LoA were only -0.6 ± 0.4 s (total error -0.2 s to -1 s) which indicates that this trial was the most precise of the 3 conducted. The bias detected was possibly a chance result stemming from the unusual occurrence of every swimmer slightly underestimating their target time. Indeed, the 100 m split times for this trial did not demonstrate significant bias and neither was significant bias observed in any of the other trials, which suggests that this was a type 1 error.

A further aim of Study 6b was to determine if the swimmer's pacing would be consistent over 3 trials. No significant bias was detected and the LoA were small for both swimming speeds, suggesting that the swimmer's were habituated prior to each trial. This means that the six, 50 m breaststroke efforts during the warm-up were sufficient to habituate the swimmers. This is impressive considering that the majority of swimmers in this study had no prior experience of the Aquapacer™. Therefore it appears that the Aquapacer™ provides a precise and reliable method of pacing swimmers, who rapidly habituate to the device.

6.4.2 Reliability of metabolic, kinematic and RPE measurements

Fundamentally there were no results indicating significant bias for any variables during any of the trials, which suggests that the 48-72 h period between trials was sufficiently long to allow complete recovery. The measurement error in

heart rate (HR) responses ranged between 5 and 8 b.min^{-1} for the 85 % 200_{TT} and 95 % 200_{TT} trials representing an error of only 5 % and 3.5 % respectively. This seems acceptable given that a slight daily variation in pool temperature or a small degree of dehydration could result in similar variation during sub-maximal exercise. Also, Martin and Thompson (2000) have reported similar internal consistent reliability in HR response for freestyle swimming during a sub-maximal training set, based on a T-30 swimming speed (Olbrecht *et al.*, 1985). While Taylor (1944) and Becque *et al.*, (1993) have reported similar within subject variability (S^2_w) expressed as a percentage of mean exercise response, for sub-maximal cycling and treadmill running (3.2 % and 4.1 %, r = 0.78 respectively). These findings would suggest that there is acceptable stability reliability in HR response (Baumgarter, 1989) and hence, there appears to be good agreement between HR responses during repeated bouts of sub-maximal and near maximal exercise. Cellini *et al.* (1986) have also previously reported a high correlation (r=0.99) between the HR - (swimming velocity)^3 relationship determined from an incremental protocol, which supports the notion that HR response demonstrates relative reliability (Baumgarter, 1989) during swimming.

Interestingly, it was noticeable during the trials that mean HR increased by ~ 10 b.min^{-1} between 100 m and 200 m, for both swimming speeds, suggesting that the majority of the adjustment in HR kinetics occurred within 90 s of the repetitions beginning, and only a 5 % change thereafter. However, as the subject's heart rate responses were not recorded for the 200_{TT} it is not possible

to suggest the percentage of maximum HR that was attained by the swimmers during the sub-maximal reproducibility trials.

According to the 95 % LoA, the error inherent within capillary blood lactate determination appears large for the 85 % 200_{TT}, but not so large for the 95 % 200_{TT} trial. Martin and Thompson (2000) have also reported evidence of large random error in mid - range blood lactate concentrations over consecutive trials, although neither in their study nor in the present study was significant bias observed. However, a shortcoming of repeated measures ANOVA is that it is possible for a small bias to be calculated when there is relatively high random error (Atkinson and Nevill, 1998). It is important to note that the measurement error, excluding bias for the 95 % 200_{TT} trial, was much lower (16.2 %, when expressed as a percentage of the mean lactate concentration of the 3 trials), and so is more acceptable.

The collection of a 20 s expired air sample allowed for the determination of a number of respiratory variables ($\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$ and RER), although it must be stated that this method was specifically developed only for the determination of $\dot{V}O_2$ at exercise cessation. The $\dot{V}O_2$ and predicted $\dot{V}O_2$ values from the 95 % 200_{TT} trials demonstrated acceptable agreement (11.9 % of mean $\dot{V}O_2$ of the 3 trials) and within subject variability expressed as a percentage of mean $\dot{V}O_2$ was similar to that observed for sub-maximal (Taylor, 1944; Becque *et al.*, 1993) and maximal exercise (Taylor 1944; Katch *et al.*, 1982) from direct measurement. However, Martin and Thompson (2000) have reported greater

within-subject variability between sub-maximal trials in freestyle swimming (95 % LoA, >30 % of mean $\dot{V}O_2$ of the 3 trials). While in support of the BE method Montpetit *et al.* (1981) have previously observed good relative reliability using the BE method (r=0.92, t statistic - NS) from 7 swimmers following a test and retest, while Costill *et al.* (1985) reported a daily test retest variation of ± 5.9 % and a high correlation (r=0.97) when 52 swimmers completed maximal 400 yd freestyle swims on 2 separate days. The variance they observed was also similar to data reported for repeated gas collections in cycle ergometry (Coggan and Costill, 1983; Armstrong and Costill, 1985).

Interestingly, there was proportionately larger random error in $\dot{V}O_2$ for the 85 % 200_{TT} trial compared to the 95 % 200_{TT} trial. Unfortunately, the number of breaths taken during the 20 s sampling time were not counted in all cases making it impossible to comment as to whether some of the subjects attempted to voluntarily control their breathing frequency and tidal volume following one or more of the 85 % TT trials. Although, it was noted that breathing frequency changed by one breath in 2 subjects following one trial compared to a previous trial. However, 95 % LoA for VE demonstrated good reliability for all trials which suggests that breathing patterns were not altered markedly (no obvious signs of hypoventilation or hyperventilation). The results for $\dot{V}E$ also seem appropriate given that the within-subject variability for $\dot{V}E$ was similar to other studies (Taylor, 1944; Armstrong and Costill, 1985; Becque *et al.*, 1993) although lower than those reported by Martin and Thompson (2000). The disparity in terms of the $\dot{V}O_2$ and $\dot{V}E$ results with the Martin and Thompson

(2000) study may have been due to their subjects demonstrating altered breathing patterns. Although, this disagreement could also be explained by the different strokes used.

In fact, in the present study what is likely to have caused the proportionately larger random error in $\dot{V}O_2$ following the 85 % 200_{TT} trial, compared with the 95 % 200_{TT} trial, is that a consistent measurement error in $\dot{V}O_2$ determination was detected in both trials (95 % LoA $<\pm$ 0.4 $l.min^{-1}$ in both cases). This consistent error would result in a greater random error relative to actual $\dot{V}O_2$ following the slower 85 % 200_{TT} trial, because of the reduced $\dot{V}O_2$ associated with the lesser effort employed at that pace.

The $\dot{V}CO_2$ results demonstrated a similar but more exaggerated pattern to the $\dot{V}O_2$ data, in terms of measurement error, because the 95 % LoA were markedly higher for the 85 % 200_{TT} trial. This was not unexpected as it is known that $\dot{V}CO_2$ measurements generally demonstrate greater variability than $\dot{V}O_2$ measurements; because CO_2 exchange is less constant, due to the presence of large CO_2 pools in the body which can be altered by deep breathing or intense exercise. The recovery of $\dot{V}CO_2$ post exercise is also known to be very rapid (Robergs and Roberts, 1997) particularly at lower exercise intensities. Hence, measurements may not reflect CO_2 tissue production unless they are confined to resting and steady state situations (Willmore and Costill, 1994). The fact that 95 % LoA for $\dot{V}CO_2$ were less for the 95 % 200_{TT} trial would suggest that the greater disparity observed following the 85 % 200_{TT} trial was due to variability

in depth of breathing, although this cannot be proven for reasons stated earlier. Interestingly, in the present study there was proportionately less random error demonstrated in both $\dot{V}O_2$, $\dot{V}CO_2$ (and consequently RER measurements) during the more intense exercise (95 % 200_{TT} trials), which is again reassuring, given that many of the studies in this thesis are to be carried out at this and higher exercise intensities. This greater consistency in respiratory response post exercise at higher exercise intensities, may be due to the recovery in respiratory kinetics being delayed and /or subjects being less able to conciously change their breathing patterns.

Ninety five percent LoA values for RPE were 18 - 20 % of mean trial values which may be acceptable given the subjective nature of the scale and are in agreement with Martin and Thompson (2000). It was however noticeable that 3 of the 4 slowest swimmers reported RPE values as low as 13 despite swimming at 95 % 200_{TT} pace ! This may suggest that either they were not anchored precisely to the scale or that they were less able to perceive exertion. It is possible that their lack of sensitivity might be due to the fact that they were not training as often as the other subjects.

Stroke rate (SR) measurements proved to be highly reproducible both in terms of the intra-individual variability of the investigator's measurement error and the subject's technical performance, with little evidence of random error evident. The latter finding is in agreement Martin and Thompson (2000), although they reported less agreement between trials. It is therefore likely that

differences in SR might be detected when a particular intervention is being investigated. Finally, mean SR was found to increase while distance per stroke decreased (as indicated in gross terms by the increase in mean SC) when comparing the 95 % 200_{TT} trial to the 85 % 200_{TT} trial. This is consistent with the findings in Studies 1 and 2 when breaststroke swimmers were under significant physical stress.

Stroke count (SC) demonstrated greater random error than SR, possibly due to greater measurement error, resulting from the subjective determination of whether the swimmer had completed a half or a full stroke. Nonetheless this shortcoming did not result in unacceptable agreement between measurements, probably due to the fact that each subject's measurement was in effect a mean value, resulting from measurements made during individual lengths by an experienced investigator.

Prior to making any conclusions it must be mentioned that the use of the 95 % LoA is still a relatively new concept in the field of sports science. Hence, Lamb and Thurlow (1999) have suggested that there is a need for " tolerable limits to be established a priori by experts in the field of interest". To date this has not happened, although Atkinson *et al.* (1999) have attempted to help the situation by reporting a nomogram based on ratio LoA to estimate the sample size necessary to detect percentage changes in performance following a repeated measures experiment. The nomogram would suggest that the sample size in the present study was appropriate to estimate the reliability of the Aquapacer™.

However, Atkinson *et al.* (1999) commented that making a decision as to how much measurement error indicates reliability remains difficult.

6.5 Conclusions

The first aim was to establish if breaststroke swimmers could pace precisely using a poolside clock. In Study 6a no significant bias was observed between actual and predicted finishing times and a relatively small error in pace judgement was detected suggesting that acceptable precision was demonstrated. However, the Aquapacer™ (Study 6b) was also observed to provide a precise method of pacing which was reproducible (aim ii) and had less error associated with it than self paced swimming. A significant systematic bias in pacing was observed in the split time of one of the paced trials, however a type 1 error was thought to have occurred. The final aim was to determine if kinematic, metabolic and RPE measurements were reliable across repeated trials. Agreement was found to be good between trials for HR, $\dot{V}O_2$, VE and SR for the 85 % 200_{IT} trials and 95 % 200_{IT} trials, and $\dot{V}CO_2$ and RER for the 95 % 200_{IT} trial. Stroke count and RPE demonstrated acceptable measurement error (95 % LoA were 14 - 20 % of the mean of the trials) during the 85 % 200_{IT} trials and 95 % 200_{IT} trials, while blood lactate concentration was found to demonstrate acceptable agreement following the 95 % 200_{IT} trials, but not the 85 % 200_{IT} trials.

Chapter 7

The effect of manipulations in pacing on kinematic, temporal and metabolic variables during maximal 200 m breaststroke swimming.

7.1 Introduction

The multifactorial nature of a sporting performance means that intra-individual competitive performances will often differ by a small margin. For the breaststroke Thompson (1998) reported a 2.8 % difference in finishing time (FT) between heats and finals in national swimmers. However little, if any, quantitative data has been published for the breaststroke with regard to how an individual's kinematic or metabolic responses change between performances, where FT s differ by a few percent.

It seems likely that a shorter FT would require an increase in mid-pool swimming velocity (SV), as SV has been found to be highly predictive of FT in breaststroke swimming (Wakayoshi *et al.*,1992; Chapter 4). However it is unclear whether an increase in SV would arise from an increase in stroke rate or stroke length, or a combination of the two, as both are poor predictors of FT (D'Aquisto *et al.*, 1988; Kennedy *et al.*, 1990; Chengalur and Brown., 1992; Chapters 4 and 5), although Chollet *et al.* (1996) have suggested that stroke rate (SR) may increase while stroke length (SL) may decrease.

For a given SV the breaststroke is known to be less economical than the crawl strokes (Holmer, 1974), so it is perhaps the most likely to demonstrate significant changes in metabolism when SV is increased slightly. However there is little evidence to show that a small improvement in race performance requires an additional energy contribution. Sawka *et al.* (1979) have reported

that post exercise capillary blood lactate samples were elevated by 18 % when FT decreased by 3.6 %, from a comparison of non-competitive and competitive breaststroke performances (n=3). In contrast Thompson (1998) found no significant differences in peak capillary blood lactate concentrations between the heat and final swims of national 100 m and 200 m breaststroke swimmers when FT differed by 2.8 % (n=6). There has also been little research specifically reporting changes within ventilation with respect to subtle manipulations of pace. Swaine and Reilly (1983) have reported small (but significant) changes within $\dot{V}O_2$ and $\dot{V}E$ when small changes were observed in SR during high intensity freestyle swimming simulations on a swimbench. However as this study manipulated SR rather than pacing it's findings are of limited application.

What is clear from Chapter 4 is that breaststroke swimmers will try to adopt a unique SR : SL combination when trying to achieve a particular swimming speed. However, it is not clear how stroke kinematics are manipulated to achieve a subtle change of pace at maximal racing velocities in breaststroke swimming. It is also not known how subtle changes in kinematics affect metabolism

Therefore the aim of this study was to investigate the effect of manipulations of pacing on selected kinematic, temporal and metabolic variables during maximal breaststroke swimming.

7.2 METHOD

7.2.1 Subjects

Nine male swimmers (Table 7.1) were recruited from the Welsh national swimming squads and three swimming clubs in South and West Wales. Prior to completing the study subjects were fully informed about the demands and procedures of the study and gave their written consent to participate. Health screening and phlebotomy questionnaires were administered (UWIC Physiology laboratory procedure) and scrutinised prior to subjects being permitted to begin the studies (Appendix 1). Subjects then read a Volunteer Consent form, outlining the purpose and procedure of the study and gave informed consent prior to beginning the study. Ethical approval for each study was granted by the Ethics Committee of Liverpool John Moores University.

Subjects were informed that they should not attempt a test unless they were in good health and that they could terminate an exercise test at any time. They were asked to report to the laboratory in a rested state having completed no exercise or only very light exercise the previous day. Self report diaries were issued to confirm this when studies took a number of days to complete. They were also asked to abstain from alcohol, caffeine and fatty foods on the day of testing, and not to consume food in the three hours before a test. Information was also given about how to ensure euhydration prior to testing.

Stature and body mass of subjects were measured using a portable stadiometer (Holtain) and electronic weighing scales (Seca 770). Arm length was measured by the distance from the acronium process to the end of the middle finger using a standard measuring tape (Chatard *et al.*, 1990). Skinfold measurements were taken with skinfold calipers (John Bull, British Instruments Ltd, England) according to BASES Physiological Testing Guidelines (Bird and Davison., 1987).

Finally, hydrostatic mass was measured by applying a known mass to the middle of the swimmer's back until it became just submerged, while the swimmer adopted a tuck position, face down in the water. The subject was allowed to take a breath prior to adopting the position (Chatard *et al.*, 1990).

Table 7.1 - Physical and anthropometric characteristics of the subjects.

Subject	Age	Height	Arm length	Body mass	Hydro-static mass	Sum of 4 skin-folds	200 m Time Trial time
	(yrs)	(m)	(m)	(kg)	(kg)	(mm)	(s)
AT	33	1.85	0.84	85.2	2.4	38.0	170.1
AA	21	1.82	0.83	73.7	3.2	32.2	143.1
JL	18	1.83	0.81	74.1	2.2	27.7	149.9
RL	19	1.76	0.82	67.3	2.1	17.6	170.3
JB	20	1.78	0.83	79.5	2.2	39.3	174.2
I	22	1.70	0.78	84.6	1.5	44.2	180.2
PM	24	1.83	0.83	88.2	2.2	32.4	146.1
IM	21	1.79	0.82	75.5	2.3	30.5	154.8
AH	25	1.80	0.82	72.6	2.3	29.8	152.4
Mean ± s	23 ± 5	1.80 ± 0.04	0.82 ± 0.02	77.5 ± 6.9	2.3 ± 0.4	37.4 ± 11.6	163.3 ± 14.0

7.2.2 Experimental design

Subjects completed a self paced maximal 200 m breaststroke time trial (200_{TT}) from a push start. From the finishing time, the times to complete 200 m repetitions at 98 %, 100 % and 102 % of the 200_{TT} pace were calculated. Seventytwo hours later subjects swam a 200 m trial at one of these paces. The other trials were undertaken on separate days at least 48 hours apart. Subjects completed the trials following a standardised warm-up and in randomised order. The Aquapacer™ system was used to pace the swimmers. Actual times to complete 50 m, 100 m, 150 m and 200 m were measured in each trial to establish the precision of pacing from a comparison with the predicted time. Kinematic (SR, stroke count - SC), metabolic (heart rate, blood lactate, gas exchange variables) and Rating of Perceived Exertion (RPE) responses were also measured.

7.2.3 Protocol and conduct of the study

7.2.3.1 Calculation of the pace of the trials

Prior to completing the main study each subject completed a maximal 200_{TT} from a push start following a self selected warm up comprising of at least 800 m of swimming. Following the 200_{TT} target times representing 98 % and 102 % of the 200_{TT} pace were calculated as follows:

98 % 200_{TT} pace = 1.02 (200_{TT} time (s))

102 % 200_{TT} pace = 0.98 (200_{TT} time (s))

At least 72 h after the 200_{TT}, the subjects completed a series of three, 200 m breaststroke swims 48 h apart, at either 98 %, 100 % or 102 % 200_{TT} pace. The swims were completed in randomised order and at the same time of day (± 60mins) to minimise diurnal variation (Reilly *et al.*, 1984).

7.2.3.2 Standardised warm-up

Prior to each effort the subjects completed the standardised warm-up described in Chapter 6.2.6.2 and the same measurements were taken. Prior to any further swimming the subject was given a 5 minute recovery period.

7.2.3.3 Trial procedures

The Aquapacer™ was re-programmed following the warm-up to pace the swimmer's impending 200 m effort evenly, with audible bleeping signals set to coincide with every 12.5 m travelled. The swimmer's pace was set to either elicit the 98 %, 100 % or 102 % 200_{TT} time depending on which trial the swimmer was due to complete. Hand timing measurements were taken every 50 m to ensure pacing was accurate. Measurements of heart rate (at 100 m and post exercise), SR (in lengths 2, 4, 6 and 8), SC (in lengths 1, 3, 5 and 7), gas

exchange variables and RPE (post exercise) were as described previously in Chapter 6.2.6.3. A single capillary blood lactate sample was taken from the earlobe 3, 7, 11 and 13 minutes after swim cessation. Thompson (1998) having observed that peak blood lactate concentrations occurred approximately 7 minutes post exercise following national 200 m breaststroke races. Blood lactate samples were analysed within 24 hours of the event using an Analox GM7 analyser, having been removed from the fridge for 30 minutes and remixed, once the caps were removed from the ends of the lysing tubes. When lactate concentrations greater than 10 mM were observed, 3.5 µl samples were drawn from the lysing capillary tubes for analysis rather than 7 µl samples normally drawn for lower lactate values. Duplicate samples were measured in all cases.

7.2.4 Statistical analyses

All procedures were undertaken using the Minitab v11 and SPSS v7.5 software packages. A level of significance of $P<0.05$ was adopted throughout. The Anderson-Darling Test was used to test for normality to ensure that parametric analyses could be used. Guidelines for adopting parametric repeated measures analysis, such as considering sphericity as the main assumption to be upheld, were reviewed prior to analysis. Dependent t-tests and One-way ANOVA were utilised to establish if a large systematic bias (relative to random error) was present between trials in terms of pacing and metabolic parameters. Factorial ANOVA s were calculated to determine the effect of pacing (Factor A) and time

course of the trials (Factor B) on HR, SR, SC and TT. Post-hoc comparisons were completed for any significant interactions and for main effects, where there were more than two levels. For post-hoc comparisons of interaction effects, a correction developed by Cicchetti (1972) was applied to reduce the chance of Type II errors, by adjusting k (number of groups) to reflect the number of unconfounded comparisons being performed (Heiman, 1999). For all ANOVA calculations the Huynh Feldt adjustment was used if an epsilon value <0.75 was observed in the analyses (Vincent, 1995), otherwise violation of the assumption of sphericity was considered to be minimal. If a significant bias was observed then Tukey's HSD post hoc test was adopted (for ANOVA) and the omega-squared statistic was used to estimate the meaningfulness of the finding. Finally, Pearson's Product Moment correlation coefficient was calculated to determine relationships between variables.

In order to estimate the random error within pacing precision, 95 % Limits of Agreement (95 % LoA, Bland and Altman, 1986) were utilised to compare actual finishing times with predicted finishing (target) times. A detailed description of this type of statistical analysis was outlined in Chapter 6.

7.3 Results

Table 7.2 establishes the error in the pacing of the 3 trials while Table 7.3 compares actual split and finishing times between the trials. Tables 7.4 - 7.10 compare metabolic, kinematic and temporal responses between trials. Tables 7.11 - 7.14 report the relationships of anthropometric, metabolic, kinematic and temporal variables with finishing time, between trials and within trials.

Table 7.2 - Comparison and 95 % Limits of Agreement between predicted and actual times (mean ± s) for split times (SpT) and finishing times (FT)

Trial	Predicted times (s)	Actual times (s)	t	omega^2	95 % LoA	r
98 % 200_{FT} trial						
50 m SpT	40.7 ± 3.5	40.1* ± 3.3	2.85	26 %	- 0.6 ± 1.3	0.99
100 m SpT	81.5 ± 7.1	81.3 ± 7.1	0.46	-	-0.2 ± 2.0	0.99
150 m SpT	122.3 ± 10.7	122.2 ± 10.6	0.11	-	-0.0 ± 1.2	0.99
200 m FT	163.3 ±14.0	163.6 ± 14.1	-1.28	-	0.2 ± 1.1	0.98
100 % 200_{FT} trial						
50 m SpT	40.0 ± 3.4	39.6 ± 3.2	1.83	-	-0.4 ± 1.32	0.99
100 m SpT	80.1 ± 6.8	79.9 ± 6.9	0.45	-	-0.1 ± 1.8	0.99
150 m SpT	120.1 ± 10.2	120.5 ± 10.3	-1.93	-	0.4 ± 1.3	0.99
200 m FT	160.1 ± 13.6	161.3* ± 13.9	-3.25	32 %	1.2 ± 2.2	0.98
102 % 200_{FT} trial						
50 m SpT	39.1 ± 3.1	38.8 ± 3.3	1.77	-	-0.4 ± 1.2	0.99
100 m SpT	78.2 ± 6.7	78.3 ± 6.7	0.80	-	-0.2 ± 1.5	0.99
150 m SpT	117.7 ± 10.1	118.7* ± 9.6	-3.18	31 %	1.0 ± 1.8	0.99
200 m FT	156.5 ± 12.7	160.0** ± 12.6	-6.52	68 %	3.5 ± 3.1	0.98

* denotes $P<0.05$, ** denotes $P<0.01$

Significant differences were observed between actual and predicted times at 50 m (98 % 200_{FT} trial), 100 m (102 % 200_{FT} trial) and 200 m (100 % 200_{FT} and 102 % 200_{FT} trials) (Table 7.2). The initial finding may have been due to an over exuberent push off, while the other findings are probably an expression of developing fatigue. The 95 % LoA demonstrate acceptable pacing precision, until the last 50 m and 100 m of the 100 % 200_{FT} and 102 % 200_{FT} trials. These findings, and those comparing actual times between trials (Table 7.3), offer support to the validity of the protocol. Finally, the 102 % 200_{FT} trial demonstrated positive pacing in that the first 100 m was swum significantly faster than the second 100 m ($P<0.01$).

Table 7.3 - Comparison of actual split times (SpT) and finishing times (FT) between trials (mean ± s)

	98 % 200_{FT} trial	100 % 200_{FT} trial	102 % 200_{FT} trial	F	omega^2
50 m SpT	40.1 ± 3.3	39.6 ± 3.2	38.8^{a*} ± 3.3	14.51	71 %
100 m SpT	81.3 ± 7.1	79.9^{a*} ± 6.9	78.3^{a,b**} ± 6.7	22.14	69 %
150 m SpT	122.2 ± 10.6	120.5^{a*} ± 10.3	118.7^{a**} ± 9.6	35.02	76 %
200 m FT	163.6 ± 14.1	161.3^{a**} ± 13.9	160.0^{a**,b*} ± 12.6	28.37	74 %

Notes for Table 7.3 -

* denotes $P<0.05$, ** denotes $P<0.01$
a denotes significantly different from 98 % 200_{FT} trial
b denotes significantly different from 100 % 200_{FT} trial

Table 7.4 - Comparison of RPE and selected metabolic variables between trials (mean ± s)

Trial	98 % 200_{TT} trial	100 % 200_{TT} trial	102 % 200_{TT} trial	F	omega^2
Blood lactate (mM)					
Post warm-up	2.7 ± 0.7	2.6 ± 0.4	2.7 ± 0.8	0.22	-
Post trial (peak value)	9.1 ± 1.9	9.6 ±1.8	$11.3^{a**,b*}$ ± 1.2	9.97	48 %
Change in lactate (peak - post warm-up value)	6.4 ± 2.0	7.2 ± 2.0	$8.7^{a,b**}$ ± 1.6	16.67	62 %
Time to reach peak lactate (mins)	4.7 ± 3.0	6.1 ± 2.7	6.0 ± 3.2	0.43	-
Respiratory variables					
$\dot{V}O_2$ ($l.min^{-1}$)	4.00 ± 0.71	4.13 ± 0.67	4.11 ± 0.67	1.26	-
$\dot{V}O_2$ / hydrostatic-mass ($l.min^{-1}.kg^{-1}$)	1.86 ± 0.66	1.92 ± 0.63	1.90 ± 0.61	1.95	-
$\dot{V}CO_2$ ($l.min^{-1}$)	4.82 ± 0.72	4.96 ± 0.76	5.08 ± 0.54	1.05	-
RER	1.19 ± 0.10	1.21 ± 0.07	$1.25^{a,b*}$ ± 0.12	3.84	24 %
$\dot{V}E$ ($l.min^{-1}$)	102.30 ± 23.78	111.08 ± 20.22	111.6 ± 17.75	2.22	-
$\dot{V}E/\dot{V}O_2$	26.57 ± 3.36	27.04 ± 3.46	27.45 ± 3.80	0.92	-
Rating of Perceived Exertion	16 ± 2	17 ± 1	18^{a*} ± 1	14.87	49 %

Notes for Table 7.4 -

* P<0.05, ** P<0.01
a denotes significantly different from 98 % 200_{TT} trial
b denotes significantly different from 100 % 200_{TT} trial

In Table 7.4 the 102 % 200_{IT} trial RER, peak lactate and "change in lactate" values were significantly greater than for the other trials, while RPE was significantly greater than the 98 % 200_{IT} trial. Heart rate response demonstrated a significant interaction between the pace of the trials and the timecourse of the trials (Table 7.5). The significant difference in the mean heart rate (HR) measurement at 100 m (across the trials), compared to that at 200 m, was due to slower HR kinetics being demonstrated in the 98 % 200_{IT} trial (Table 7.9). By 200 m there were no significant differences in HR response between trials suggesting that responses were near maximal at this point. The similarity of the HR response at 100 m for the 100 % 200_{IT} and 102 % 200_{IT} trials suggests that HR kinetics were operating near maximally during the 100 % 200_{IT} trial, and hence no further increase was observed in the 102 % 200_{IT} trial despite a faster pace being observed early on.

Stroke rate was significantly increased in the 102 % 200_{IT} trial compared with the 98 % 200_{IT} trial (Table 7.6), suggesting that breaststroke swimmers favour an increase in SR when attempting to increase swimming velocity. Stroke rate was also found to increase consistently in each trial as they progressed, which appears to be an attempt by the swimmers to maintain SV as SC increased (Table 7.7).

Stroke count demonstrated a significant interaction between the pace of the trials and the timecourse of the trials (Table 7.7). It was observed that SC

increased with the pace of the trials and also with the duration of the trials (Tables 7.9 and 7.10). Hence at a SV approximating to 98 % 200_{FT} pace, any further increase in SV resulted in a decrease in SL and an increase in SC. Stroke length deteriorated further as the swimmer progressed along the 200 m trial. The initial reduction in stroke length, in order to achieve a faster SV (by increasing SR disproportionately), was not unexpected given that, in Chapter 4, 100 m swimmers were found to exhibit a greater SV than 200 m swimmers, as a result of adopting a greater SR and a lower SL. The deterioration in SL as the trials progressed is certainly linked to the onset of fatigue, which increasingly interfered with the technical competence of the swimmer's stroke as the trials progressed.

Turning times also demonstrated a significant interaction between the pace of the trials and the timecourse of the trials (Table 7.7). Over the first two measured turns (at 25 m & 75 m), the swimmers pacing at the higher velocities took less time to complete the manoeuvre. However as the trials progressed, the differences in turning times became less discernible (eg. at 125 m), until at the final turn, the 98 % 200_{FT} trial demonstrated a significantly lower turning time than the other trials (Table 7.9). It was also noticeable that within the trials there was evidence of deterioration in turning performance in all trials, but this was potentiated by the pace of the trials (Table 7.10). Turning time performance is therefore sensitive to the rate of fatigue being experienced in breaststroke swimming.

Table 7.5 - Comparison of means within subjects (Two factorial ANOVA) for heart rate (mean ± s)

Heart rate ($b.min^{-1}$)

Main Effects

Pace of trials

98 % 200_{TT} trial	100 % 200_{TT} trial	102 % 200_{TT} trial	F	Sig	$Omega^2$
$179^{b,c**}$ ± 9	183 ± 8	184 ± 9	7.11	P<0.01	0.40

Timecourse of heart rate measurement

HR at 100 m	HR at 200 m	F	Sig	$Omega^2$
179^{b**} ± 10	184 ± 7	11.34	P<0.01	0.51

Interaction	F	Sig	$Omega^2$
	4.00	P<0.05	0.29

Notes for Table 7.5 -

* denotes P<0.05, ** denotes P<0.01

a denotes significantly different from 98 % 200_{TT} trial, or HR at 100 m

b denotes significantly different from 100 % 200_{TT} trial, or HR at 200 m

c denotes significantly different from 102 % 200_{TT} trial

Table 7.6 - Comparison of means within subjects (Two factorial ANOVA) for stroke rate (mean ± s)

Stroke Rate ($S.min^{-1}$)

Main Effects

Pace of trials

98 % 200_{TT} trial	100 % 200_{TT} trial	102 % 200_{TT} trial	F	Sig	$Omega^2$
31.3 ± 2.8	34.5 ± 4.3	36.7^{a*} ± 4.0	24.44	P<0.01	0.69

Timecourse of stroke rate measurement

SR in length 2	SR in length 4	SR in length 6	SR in length 8	F	Sig	$Omega^2$
31.8 ± 3.5	33.2^{a**} ± 3.7	$34.8^{a,b**}$ ± 3.8	$36.8^{a,b,c**}$ ± 3.9	77.30	P<0.01	0.90

Interaction	F	Sig	$Omega^2$
	2.09	NS	-

Notes for Table 7.6 -

* denotes P<0.05, ** denotes P<0.01

a denotes significantly different from 98 % 200_{TT} trial, or length 2

b denotes significantly different from 100 % 200_{TT} trial, or length 4

c denotes significantly different from 102 % 200_{TT} trial, or length 6

d denotes significantly different from length 8

Table 7.7 - Comparison of means within subjects (Two factorial ANOVA) for stroke count (mean ± s)

Stroke count (S.25 m^{-1})

Main Effects

Pace of trials

98 % 200_{TT} trial	100 % 200_{TT} trial	102 % 200_{TT} trial	F	Sig	Omega^2
8.2 ±1.0	8.8^{a**} ±1.5	9.5^{a,b**} ±1.6	21.60	P<0.01	0.83

Timecourse of stroke count measurement

SC in length 1	SC in length 3	SC in length 5	SC in length 7	F	Sig	Omega^2
7.9 ±1.0	8.5^{a**} ±1.3	9.1^{a,b**} ±1.5	9.9^{a,b,c**} ±1.6	47.69	P<0.01	0.67

Interaction

F	Sig	Omega^2
3.11	P<0.05	0.28

Notes for Table 7.7 -

* denotes P<0.05, ** denotes P<0.01

a denotes significantly different from 98 % 200_{TT} trial, or length 1

b denotes significantly different from 100 % 200_{TT} trial, or length 3

c denotes significantly different from 102 % 200_{TT} trial, or length 5

Table 7.8 - Comparison of means within subjects (Two factorial ANOVA) for turning time (mean ± s)

Turning time (s)

Main Effects

Pace of trials

98 % 200_{TT} trial	100 % 200_{TT} trial	102 % 200_{TT} trial	F	Sig	Omega^2
12.0 ± 1.0	11.9^{a**} ± 1.0	11.7^{a,b**} ± 0.8	4.09	P<0.05	0.29

Timecourse of turning time measurement

TT at 25 m	TT at 75 m	TT at 125 m	TT at 175 m	F	Sig	Omega^2
11.5 ± 0.8	11.6 ± 0.9	12.0^{a,b**} ± 1.0	12.3^{a,b,c**} ± 0.8	9.56	P<0.01	0.54

Interaction

F	Sig	Omega^2
5.19	P<0.01	0.39

Notes for Table 7.8 -

* denotes P<0.05, ** denotes P<0.01

a denotes significantly different from 98 % TT trial, or turning time at 25 m

b denotes significantly different from 100 % TT trial, or turning time at 75 m

c denotes significantly different from 102 % TT trial, or turning time at 125 m

Table 7.9 - Post hoc comparison for interaction effects between trials for heart rate, stroke count and turning time (mean ± s)

	98 % 200_{TT} trial	100 % 200_{TT} trial	102 % 200_{TT} trial
Heart rate ($b.min^{-1}$)			
HR at 100 m	175 ± 11	181[a**] ± 10	182[a**] ± 11
HR at 200 m	183 ± 8	184 ± 7	186 ± 7
Stroke count ($S.25\ m^{-1}$)			
Length 1	7.4 ± 0.7	7.8[a**] ± 1.3	8.4[a,b**] ± 1.1
Length 3	7.9 ± 1.1	8.6[a**] ± 1.4	9.0[a,b**] ± 1.5
Length 5	8.7 ± 1.1	8.9[a**] ± 1.8	9.8[a,b**] ± 1.6
Length 7	8.9 ± 1.3	9.9[a*] ± 1.7	10.8[a,b**] ± 2.1
Turning time (s)			
at 25 m	11.9 ± 1.1	11.5[a**] ±1.1	11.3[a,b**] ± 1.0
at 75 m	12.0 ± 1.1	11.7[a**] ± 1.1	11.3[a,b**] ± 0.9
at 125 m	12.1 ±1.0	12.0 ± 0.9	11.9[a,b**] ± 0.6
at 175 m	12.1 ± 0.8	12.3[a**] ±0.9	12.3[a**] ± 0.8

Notes for Table 7.9 -

* denotes P<0.05, ** denotes P<0.01
a denotes significantly different from 98 % 200_{TT} trial
b denotes significantly different from 100 % 200_{TT} trial
c denotes significantly different from 102 % 200_{TT} trial

Table 7.10 - Post hoc comparison for interaction effects within trials for heart rate, stroke count and turning time (mean ± s)

Columns	A	B	C	
Heart rate ($b.min^{-1}$)	HR at 100 m	HR at 200 m		
98 % 200_{TT} trial	175 ± 11	183 ^{a**} ± 8		
100 % 200_{TT} trial	181 ± 10	184 ± 7		
102 % 200_{TT} trial	182 ± 11	186 ±7		
Stroke count ($S.25\ m^{-1}$)	SC Length 1	SC Length 3	SC Length 5	SC Length 7
98 % 200_{TT} trial	7.4 ± 0.7	7.9^{a**} ± 1.1	$8.7^{a,b**}$ ± 1.1	$8.9^{a,b,c**}$ ± 1.3
100 % 200_{TT} trial	7.8 ± 1.3	8.6^{a**} ± 1.4	$8.9^{a,b**}$ ± 1.8	$9.9^{a,b,c**}$ ± 1.7
102 % 200_{TT} trial	8.4 ± 1.1	9.0 ± 1.5	$9.8^{a,b**}$ ± 1.6	$10.8^{a,b,c**}$ ± 1.1
Turning time (s)	TT at 25 m	TT at 75 m	TT at 125 m	TT at 175 m
98 % 200_{TT} trial	11.9 ± 1.1	12.0^{a**} ± 1.1	12.0^{a**} ± 1.0	$12.1^{a,b,c**}$ ± 0.8
100 % 200_{TT} trial	11.5 ± 1.1	11.7^{a**} ± 1.10	$12.0^{a,b*}$ ± 0.9	$12.3^{a,b,c**}$ ± 0.9
102 % 200_{TT} trial	11.3 ± 1.0	11.3 ± 0.9	$11.9^{a,b*}$ ± 0.6	$12.3^{a,b,c**}$ ± 0.8

Notes for Table 7.10 -

* denotes P<0.05, ** denotes P<0.01
a denotes significantly different from column A,
b denotes significantly different from column B,
c denotes significantly different from column C

Finishing time was found to be significantly related to the "sum of 4 skinfolds", in all the trials (r = 0.67 to 0.69), and to peak lactate and "change in lactate" concentration in the 98 % 200_{TT} and 100 % 200_{TT} trials (r = -0.69 to -0.83 (Table 7.11). While, between the trials, significant relationships were observed for HR at 200 m, peak lactate, "change in lactate" and ventilatory parameters (Table 7.12). Finishing times were found to be significantly related between trials and to mean TT (Table 7.13). Stroke rate, SC and TT were found to be significantly related between trials and generally within trials (Table 7.14); although in the 102 % 200_{TT} trial, there was some evidence of the SR and TT relationships within trials deteriorating.

Table 7.11 - Relationship between finishing time (FT) and rating of perceived exertion and selected anthropometric and metabolic variables

	FT 98 % 200$_{TT}$ trial	FT 100 % 200$_{TT}$ trial	FT 102 % 200$_{TT}$ trial
Anthropometric variables			
Age (yrs)	0.12	0.15	0.16
Body mass (kg)	0.18	0.18	0.19
Hydrostatic mass (kg)	-0.66*	-0.62	-0.62
Sum of 4 skinfolds (mm)	0.67*	0.67*	0.69*
Height (m)	-0.65	-0.61	-0.63
Arm length (m)	-0.32	-0.36	-0.34
Metabolic variables			
lactate post warm-up (mM)	0.33	0.35	0.45
lactate peak (mM)	-0.77*	-0.74*	-0.21
Change in lactate (peak - post warm-up) (mM)	-0.83**	-0.69*	-0.63
Time to lactate peak (mins)	-0.54	-0.20	0.44
HR at 100 m ($b.min^{-1}$)	-0.46	-0.54	-0.57
HR at 200 m ($b.min^{-1}$)	-0.61	-0.58	-0.52
$\dot{V}O_2$ ($l.min^{-1}$)	0.69*	0.49	0.31
$\dot{V}O_2$/hydrostatic mass ($l.min^{-1}.kg^{-1}$)	0.74*	0.64	0.58
RER	0.12	0.10	-0.40
$\dot{V}E$ ($l.min^{-1}$)	0.39	0.28	-0.23
$\dot{V}E/\dot{V}O_2$	-0.38	-0.16	-0.63
Rating of perceived exertion			
RPE	-0.47	-0.62	-0.46

Notes for Table 7.11

* denotes P<0.05 ** denotes P<0.01
Relationships are between variables within the same trial only,
eg. HR at 100 m vs FT 98 % 200$_{TT}$ trial would utilise the HR at 100 m from the 98 % 200$_{TT}$ trial.

Table 7.12 - Relationships between selected metabolic variables (and rating of perceived exertion) between the paced trials

	100 % 200_{PT} trial	102 % 200_{PT} trial
HR at 100 m ($b.min^{-1}$)		
98 % 200_{PT} trial	0.83**	0.92**
100 % 200_{PT} trial		0.90**
HR at 200 m ($b.min^{-1}$)		
98 % 200_{PT} trial	0.86**	0.89**
100 % 200_{PT} trial		0.91**
lactate post warm-up (mM)		
98 % 200_{PT} trial	0.61	0.62
100 % 200_{PT} trial		0.57
lactate peak (mM)		
98 % 200_{PT} trial	0.74*	0.31
100 % 200_{PT} trial		0.67*
Change in lactate (mM) (peak-post warm-up)		
98 % 200_{PT} trial	0.86**	0.66*
100 % 200_{PT} trial		0.90**
Time to lactate peak (mins)		
98 % 200_{PT} trial	-0.29	-0.44
100 % 200_{PT} trial		-0.30
$\dot{V}O_2$ ($l.min^{-1}$)		
98 % 200_{PT} trial	0.94**	0.88**
100 % 200_{PT} trial		0.94**
$\dot{V}O_2$/hydrostatic mass ($l.min^{-1}.kg^{-1}$)		
98 % 200_{PT} trial	0.99**	0.99**
100 % 200_{PT} trial		0.97**
$\dot{V}E$ ($l.min^{-1}$)		
98 % 200_{PT} trial	0.90**	0.66*
100 % 200_{PT} trial		0.69*
RER		
98 % 200_{PT} trial	0.87**	0.74*
100 % 200_{PT} trial		0.69*
$\dot{V}E/\dot{V}O_2$		
98 % 200_{PT} trial	0.92**	0.85**
100 % 200_{PT} trial		0.80**
RPE		
98 % 200_{PT} trial	0.32	0.52
100 % 200_{PT} trial		0.37

* denotes $P<0.05$ ** denotes $P<0.01$

Table 7.13 - Relationships between and within selected kinematic and temporal variables

	FT 98 % 200_{FT} (s)	FT 100 % 200_{FT} (s)	FT 102 % 200_{FT} (s)	Mean Stroke rate 98 % 200_{FT} ($S.min^{-1}$)	Mean Stroke rate 100 % 200_{FT} ($S.min^{-1}$)	Mean Stroke rate 102 % 200_{FT} ($S.min^{-1}$)	Mean Stroke count FT 98 % 200_{FT} ($S.25\ m^{-1}$)	Mean Stroke count FT 100 % 200_{FT} ($S.25\ m^{-1}$)	Mean Stroke count FT 102 % 200_{FT} ($S.25\ m^{-1}$)	Mean Turning times 98 % 200_{FT} (s)	Mean Turning times 100 % 200_{FT} (s)	Mean Turning times 102 % 200_{FT} (s)
FT												
98 % 200_{FT}		0.99**	0.99**	-0.18			0.68*			0.96**		
100 % 200_{FT}			0.99**		-0.12			0.60			0.97**	
102 % 200_{FT}						-0.19			0.63			0.96**
Mean stroke rate ($S.min^{-1}$)												
98 % 200_{FT}					0.71*	0.81**	0.44					
100 % 200_{FT}						0.95**		0.68*				
102 % 200_{FT}									0.57			
Mean stroke count ($S.25\ m^{-1}$)												
98 % 200_{FT}								0.95**	0.97**			
100 % 200_{FT}									0.96**			
Mean turning times (s)												
98 % 200_{FT}											0.94**	0.95**
100 % 200_{FT}												0.96**

* denotes P<0.05 ** denotes P<0.01

Table 7.14 - Relationships between selected kinematic and temporal variables within trials

Trials	Length	Length	Length
Stroke rate ($S.min^{-1}$)	SR Length 2	SR Length 4	SR Length 6
98 % 200_{FT} trial			
Length 4	0.98**		
Length 6	0.94**	0.97**	
Length 8	0.90**	0.91**	0.95**
100 % 200_{FT} trial			
Length 4	0.90**		
Length 6	0.87**	0.99**	
Length 8	0.83**	0.89**	0.99**
102 % 200_{FT} trial			
Length 4	0.54		
Length 6	0.72*	0.88**	
Length 8	0.72*	0.89**	0.92**
Stroke Count ($S.25\ m^{-1}$)	SC Length 1	SC Length 3	SC Length 5
98 % 200_{FT} trial			
Length 3	0.89**		
Length 5	0.98**	0.92**	
Length 7	0.74*	0.83**	0.51
100 % 200_{FT} trial			
Length 3	0.95**		
Length 5	0.97**	0.97**	
Length 7	0.84**	0.91**	0.88**
102 % 200_{FT} trial			
Length 3	0.89**		
Length 5	0.91**	0.97**	
Length 7	0.93**	0.98**	0.95**
Turning times (s)	at 25 m	at 75 m	at 125 m
98 % 200_{FT} trial			
at 75 m	0.97**		
at 125 m	0.91**	0.96**	
at 175 m	0.95**	0.96**	0.73*
100 % 200_{FT} trial			
at 75 m	0.94**		
at 125 m	0.87**	0.89**	
at 175 m	0.75*	0.81**	0.97**
102 % 200_{FT} trial			
at 75 m	0.98**		
at 125 m	0.72*	0.74*	
at 175 m	0.57	0.59	0.96**

* denotes $P<0.05$ ** denotes $P<0.01$

7.4 Discussion

7.4.1 Precision of pacing

This study required that subjects swam paced trials at proportionally higher speeds than for the previous study. Hence, it was of interest to note if the precise pacing observed in that study would be reproduced during the near maximal and maximal efforts of the trials in the present study. Reassuringly, 95 % LoA between predicted and actual times for the 98 % 200_{IT} trial demonstrated similar agreement to that found in the previous study. Significant bias was detected for the 50 m split times, probably as a result of subjects pushing off too hard at the start of the trial and getting ahead of the pace. Attempts were made to avoid this by pre-empting each trial with a verbal instruction not to do so. However, as the trials were randomised some subjects might have pushed off slightly too hard in the slower trial because they had done so in a previously faster trial. Also, as in Chapter 6, the swimmer's pacing became more precise as the trial progressed due to habituation.

During the 100 % 200_{IT} and 102 % 200_{IT} trials pacing was less precise. Initially, this was perhaps because the swimmer's technique was put under pressure by swimming at maximal 200 m speeds, and later as fatigue began to compromise technique. However, correlations of FT's between trials were highly significant demonstrating that subjects largely maintained their rank order across trials despite progressive fatigue. A significant bias was only detected over the final

50 m of the 100 % 200_{IT} trial and 100 m of the 102 % 200_{IT} trial suggesting that the swimmers were unable to hold pace. This demonstrated that the study's protocol was valid because it was progressive fatigue which precluded precise pacing rather than the Aquapacer™ device. In fact, only one subject was able to finish inside the 100 % 200_{IT} predicted time which suggests that the effort for this trial was near maximal. Despite the onset of fatigue, swimmers were still able to pace within 2 % of the FT (using 95 % LoA) during the 100 % 200_{IT} trial, and within 2.4 % of the 150 m split time during the 102 % 200_{IT} trial, rising to 4 % of the FT at the finish. This represents evidence that swimmers are able to pace precisely at high swimming speeds using the Aquapacer™ until fatigue becomes an overriding factor. Not surprisingly, this was most evident in the 102 % 200_{IT} trial where the swimmers actually demonstrated a "positively" paced trial, by swimming precisely 2 % faster than the 100 % 200_{IT} trial for 100 m, but thereafter they slowed down to finish only 0.8 % faster. The positive split pacing observed in the 102 % 200_{IT} trial was similar to the pacing strategy highlighted in Chapter 4 during racing and in the present study this strategy yielded a significantly faster FT than the more evenly paced 100 % 200_{IT} trial ($P<0.05$). This may be important as it provides some support for favouring the adoption of a positively split race pacing strategy, rather than an evenly split one.

7.4.2 Metabolic variables

Peak lactate, change in lactate (peak - post warm-up value) and RER values were significantly higher following the 102 % 200_{TT} trial. This finding is in agreement with Sawka *et al.* (1979) who observed an increase in blood lactate when FT decreased in freestyle swimming. The coincidental increase in both lactate and RER in the present study was not unexpected given that $\dot{V}CO_2$ has previously been observed to increase relative to $\dot{V}O_2$, as lactate concentrations increased and plasma bicarbonate levels decreased during high intensity exercise (Stringer *et al.*, 1995). The slightly faster FT achieved in the 102 % 200_{TT} trial compared to the 100 % 200_{TT} trial appears to have required a significantly greater energy contribution from anaerobic glycolysis and subseqently an increased buffering response. Therefore, positively split swimming promotes greater lactic acid production than even paced swimming and leads to a significantly greater lactic acid accumulation. The fatigue inducing effects of metabolic acidosis probably led to the swimmer's slowing significantly over the final 100 m of the 102 % 200_{TT} trial. Also the significantly greater mean RPE scores reported for the 102 % 200_{TT} trial reflected the greater discomfort being experienced by the swimmers from such acute metabolic conditions.

There are a number of possible reasons for the greater lactate appearance in the 102 % 200_{TT} trial. Firstly, the initial increase in swimming velocity requires an

increased recruitment of fast glycolytic muscle fibres or a higher activation of them (Cherry *et al.*, 1997), due to a need for greater force production to overcome an increase in drag. Secondly, the increased energy demand has to be increasingly met by anaerobic energy contribution, because at such exercise intensities aerobic energy production is less able to provide energy at the required rate. For example it has been well documented that there is an increase in anaerobic contribution as swimming velocity increases (Hermansen and Karlsson, 1967; Hermansen, 1969; Houston, 1978; Maglishco, 1982; Troup, 1984) and that anaerobic energy sources become the primary energy contributor in swimming events lasting less than 3 minutes (Maglishco, 1993). In support of this, the relationship between $\dot{V}O_2$ and FT deteriorated in the present study as the intensity of the trials increased.

An increase in SC was observed in the 102 % 200_{FT} trial indicating that the swimmers completed a greater total number of stroke cycles than in the other trials. This would have increased the energy demand, because a greater number of energy consuming accelarations would have had to have been completed (Manley and Atha, 1992), following the large decelerations in swimming velocity which occur, with each leg recovery phase in breaststroke swimming (D' Aquisto *et al.*, 1988). Paradoxically, despite the swimmer having more oppurtunities to breath, the ventilatory and heart rate measurements in the present study would suggest that no significant increase in aerobic energy production occurred to meet the increased energy demand. Hence, the anaerobic energy production would have needed to increase.

This may be why it has been suggested that 200 m swimmers tend to favour a longer glide phase in order to limit energy expenditure (Chollet *et al.*, 1996) and indeed this was found to be the case in Chapter 4, when a comparison of 100 m and 200 m events was made. A number of studies have reported that better swimmers might be better able to maintain a greater SL (Craig *et al.*, 1985; Thompson and Haljand, 1997) which would also serve to limit increases in SC. The increases in SC observed in the 102 % 200_{FF} trial would have resulted in more frequent decelerations per length at a time when the swimmer was actually attempting to swim faster, and hence more powerful propulsive phases would have had to have been produced and more often. This would have placed a greater reliance on the fast glycolytic muscle fibres in this trial, than in the other trials.

Lactate values were not significantly different between the two slower trials despite a 2 % difference in mean trial velocity. This may have been due to the swimmers in the 98 % 200_{FF} trial experiencing restricted respiration, as a result of having fewer oppurtunities to breath. In support of this notion significantly fewer strokes were taken per length in this trial compared to the others, and a reduced VE was coincidentally observed; which could have led subsequently to muscle hypoxia. If hypoxia did occur then additional anaerobic glycolysis could have resulted, due to a slowed time constant for $\dot{V}O_2$ at the early stages of the exercise (Engelen *et al.*, 1996). This would have led to a similar lactate

response to that observed for the 100 % 200_{TT} trial. For example, during hypoxic swimming (where less frequent breathing is allowed) researchers have reported that end tidal O_2 pressures are slightly reduced while CO_2 pressures are increased (Holmer and Gullestrand., 1979) and in the present study while RER measurements were coincidentally similar between the trials, mean $\dot{V}E$ and $\dot{V}O_2$ values were actually slightly lower for the 98 % 200_{TT} trial. This might suggest that $\dot{V}CO_2$ production was slightly greater than expected in the 98 % 200_{TT} trial.

There is also evidence in the literature that restricted breathing does not occur during breaststroke swimming, even at high exercise intensities. Holmer *et al.* (1974) compared O_2 and CO_2 saturations in arterial blood during breaststroke swimming and running at both sub-maximal and maximal intensities and found them to be similar. They reported that despite a lower total ventilation being observed in breaststroke swimming, alveolar ventilation was sufficient and arterial saturation adequate. Therefore, although the swimmers in the present study did not demonstrate hyperventilation during maximal exercise (evident in the $\dot{V}E/\dot{V}O_2$ results), this does not necessarily mean that pulmonary ventilation during the 98 % 200_{TT} trial was characteristic of hypoventilation either.

It must also be stated that the lack of significant differences between $\dot{V}E$, $\dot{V}O_2$, lactate concentrations, and RER between these trials could merely be due to measurement error. For example 95 % LoA s from the repeated 95 % 200_{TT} trials (Chapter 6) would encompass the differences observed for these

parameters between the 98 % 200_{FT} and 100 % 200_{FT} trials. Also, Thompson (1998) previously reported a non-significant increase in lactate concentration when national standard 200 m breaststroke swimmers completed a final swim 2.8 % faster than a heat swim, which suggests that lactate measurements can be insensitive to subtle changes within FT. Thus, it does appear that there may be some difficulty in predicting improvements in swimming performance based on changes in post event ventilatory and lactate concentrations.

However, there was some evidence in the present study that post exercise peak lactate concentrations may predict FT during even paced near maximal 200 m efforts. Both peak lactate concentration and change in lactate concentration values demonstrated significant negative relationships with FT for both the 98 % 200_{FT} and 100 % 200_{FT} trials. Also, significant relationships were observed for post-exercise lactate concentrations between trials suggesting relative reliability. It is possible that faster swimmers might exhibit greater lactate concentrations for a given percentage of a maximum effort during even paced swimming. This seems likely given that $\dot{V}O_2$ / distance ratio increases as a function of velocity in swimming (Holmer, 1979) and so in order for a swimmer to further increase SV, when already operating at high levels of aerobic power, the anaerobic contribution would also be expected to be increased.

However, it seems unlikely that it is merely differences in absolute anaerobic capacity that distinguishes the better performers, because the relationships between peak lactate and FT for the 102 % 200_{FT} trial, (which elicited the

greatest peak lactate responses), were not significant. Therefore, what distinguishes the more successful swimmers physiology from the others may be their ability to buffer and remove lactic acid, having recruited additional fast glycolytic muscle fibres.

Post exercise ventilatory responses were not significantly different between trials and so might be considered to be insensitive to subtle manipulations within pacing. In support of this the 95 % LoA reported in Chapter 6 would encompass the differences between the means of the trials. However, another argument as to why ventilation did not change markedly between trials might be that the swimmers were actually exercising at very high percentages of their aerobic power in all of the trials, and so were unable to increase their oxygen uptake significantly across trials. This seems likely given that at sub-maximal swimming velocities it is accepted that energy consumption increases exponentially with SV (Holmer, 1979), and so at high velocities even a small increase in SV would be expected to produce a noticeable change in $\dot{V}O_2$. Yet, this did not occurr in the present study. Also, Holmer (1974) reported similar peak $\dot{V}O_2$ values for elite breaststroke swimmers to those in the present study, for high swimming speeds. This supports the view that the swimmers were operating at near maximal percentages of their aerobic power during the trials. Finally, the magnitude of the subject's HR responses at exercise cessation would also support this view.

Differences in HR were detected at 100 m between the 98 % 200_{TT} trial and the others, which presumably reflected the greater requirement for aerobic energy contribution at the start of the 100 % 200_{TT} and 102 % 200_{TT} trials. This difference in HR response probably reflected the greater recruitment of fast glycolytic muscle fibres in the 100 % 200_{TT} and 102 % 200_{TT} trials which would have subsequently increased the oxygen cost of the exercise, and driven the fast $\dot{V}O_2$ component (and HR kinetics) more rapidly toward their maximum capacity (Xu and Rhodes, 1999). It was also notable that 96-99 % of the peak HR was achieved in the first 100 m of the 3 trials, indicating that a significant anaerobic contribution must have occurred throughout the trials as the metabolic stress detected would have been above that which would have elicited the anaerobic threshold. To conclude, it appears that a near maximal ventilatory and cardiovascular response occurred at the 98 % 200_{TT} pace, which became further elevated in the other trials, albeit non-significantly, possibly as a result of additional fast glycolytic muscle fibre recruitment when SV was increased slightly.

These findings may explain why the relationship between FT and swimming economy (derived from $\dot{V}O_2$ measurements) has not been found to be significant at maximal SV s (D' Aquisto *et al.*, 1988; Van Handel *et al.*, 1988), despite elite swimmers demonstrating better economy at sub-maximal SV s in some studies (Pendergast, 1978), although not all (D' Aquisto *et al.*, 1988). Maglishco (1993) has suggested that the usefulness of swimming economy measurements may be limited to evaluating changes in stroke mechanics.

During maximal efforts over 2-3 minutes it is more likely that the recruitment of fast glycolytic muscle fibres and the ability to remove and buffer lactic acid may be of more concern in performance prediction than the $\dot{V}O_2$ / distance ratio. Indeed, it appears that breaststroke swimmers at or near to 200 m race pace operate beyond the intensity at which the aerobic pathway provides the majority of ATP for muscular work. Hence the $\dot{V}O_2$ / distance relationship is less relevant in 200 m events, than those with a longer time course (eg 800 m or 1,500 m swimming).

In summary, peak lactate concentrations, RER and RPE values differentiated between the even paced (100 % 200_{FT}) and positively paced (102 % 200_{FT}) trials, where a significant difference in FT (0.8 %, $P<0.05$) occurred. Yet physiological variables, at exercise cessation, were not found to be significantly different between the two more even paced trials (98 % 200_{FT} and 100 % 200_{FT} trials), where a 2 % change in mean FT occurred.

Relationships between ventilatory measures and FT were generally either not significant or significant but rather weak and so these measures may not be of value in the prediction of FT. The same conclusions would also be true for the cardiovascular and anthropometric variables except that the sum of 4 skinfolds demonstrated a significant but weak relationship with FT in all of the trials. The commonality of this finding was due to the same skinfold total being used on each occasion and the fact that swimmers maintained their relative position, in each trial, due to the precision of the pacing.

Peak lactate concentrations were generally achieved between 3 and 9 minutes for the 2 faster trials which is largely in agreement with previous data (Thompson, 1998; Howat and Robson, 1999) for breaststroke swimmers. This suggests that post competition lactate testing should occur until at least 9 minutes post exercise, due to the large inter-individual variation that is possible. There was however no evidence to support Howatt and Robson's (1999) statement that a ".. well paced race will show reasonably high lactate levels eg. 6-8 mM at the 5 minute mark, rising by 2-3 mM at 7 minutes, perhaps by another 1-2 mM at 9 minutes before flattening off or falling at 11 minutes". The findings of the present study may explain why the performances of freestyles swimmers were not significantly correlated with lactate concentrations taken 3 and 12 minutes post exercise (Pelayo *et al.*, 1996), as these blood samples may have straddled the sample which would have elicited a peak lactate concentration. Finally, it was observed that the time to achieve a peak lactate following slightly slower or faster efforts was unpredictable, because in the present study there was no relationship between the times taken to attain a lactate peak across trials. This finding further supports the view that it is necessary for multiple lactate measurements to be taken post exercise when near or maximal efforts have taken place.

7.4.3 Kinematic and temporal measurements

A comparison of mean SR across trials demonstrated that SR became increasingly elevated in order to increase swimming velocity (SV), although a significant increase was only detected in the 102 % 200_{FT} trial. Therefore it appears that breaststroke swimmers increase SR, when intially attempting to increase SV at or near to racing velocities. When SR measurements from individual lengths were compared it was observed that SR consistently increased throughout each trial. This perpetual increase in SR appears to be linked to the development of fatigue, because despite the increase in SR, SV was still observed to decrease in the latter stages of both the 100 % 200_{FT} and 102 % 200_{FT} trials.

Mean SC was also observed to increase significantly between trials, signifying that a deterioration in SL occurred as the pace of the trials increased. This finding explains why SR was found to increase along with the pace of the trials, because as SV is the product of SR and SL, then SR would have to increase in order to compensate for a decrease in SL and to an increase in SV. These findings confirm that breaststroke swimmers preferentially increase SR rather than SL when attempting to increase SV at or near to 200 m racing pace.

Stroke count was also observed to increase as the trials progressed which suggests fatigue was affecting the technical performance of the swimmers, resulting in a loss of propulsion; or what the coaches describe as "an inability to

hold water". Consequently, the swimmer increased SR in a compensatory manner, to maintain SV which is a finding common in racing (Thompson and Haljand, 1997). In fact, SC appears to be particularly sensitive to increases in physiological stress, because with each slight increase in pace a significant increase in SC was observed. It also appears that compensatory increases in SR are possible, until the reduction in SL is too great, at which point SV begins to decrease. These findings suggest that a fine line exists between success and failure when a coach asks a swimmer to adopt an aggressive positive pacing strategy in a race because of the associated rapid onset of fatigue and reduction in technical efficiency.

Interestingly, SR measurements were significantly related between trials suggesting that swimmers who adopt a relatively high SR at one pace, maintain this distinctive trait at another. The same findings were also apparent for SC relationships. Thus a consistent interindividual difference may exist between swimmers for both SR and SC. Relationships within trials for both variables were also highly correlated which suggests that the swimmers tended to increase both SR and SC in a similar manner when fatigue developed. There is compelling evidence that both SR and SC increase when breaststroke swimmers attempt to increase SV within ± 2 % of their 200 m time trial mean velocity, or when fatigue progressively occurs at such SVs.

Poorer relationships than in this study were reported within SR and SC measurements for the men's 200 m races analysed in Chapter 4. This was

probably due to the nature of the pacing in theses races as a 6 % decrease in mean SV was observed between the first and second 50 m (with 1 % decrements thereafter), whereas in the 102 % 200_{m} trial only a 4 % decrement in SV was observed over the final 100 m of the trial. The large reduction in SV between the first 50 m and second 50 m in 200 m racing may have contributed to the more erratic SR production that was observed. This suggests that stroke kinematics change more markedly and less predictably during races, because pacing is less controlled and even paced. Therefore it would be instructive to investigate the benefits of pacing a race more evenly in order to determine whether such a strategy would produce a more efficient and ultimately faster race performance, given that changes in stroke kinematics would be more progressive and predictable.

Stroke kinematics have been previously found to be generally poor predictors of FT (Wakayoshi *et al.*, 1982; D' Aquisto *et al.*, 1988; Kennedy *et al.*, 1990; Chengalur and Brown., 1992) reflecting the uniqueness of the SR-SL ratio that breaststroke swimmers exhibit. However, as discussed previously the races analysed in these chapters were positively split with pacing tending to be less even paced than the trials in the present study. Nevertheless, relationships between stroke kinematics and FT were also found to be poor in this study and hence such variables appear to be of limited use in predicting performance whether pacing is even or positive. However, the fact that stroke kinematics are more predictable when trials are evenly paced may be of use in terms of fitness testing when coaches wish to determine the causes of changes in performances.

A significant interaction was found to exist in the turning time (TT) data between the pace and timecourse of the trials. Initially, the faster trials produced faster turns, presumably due to faster approach velocities, as there was some evidence in Chapter 4, that TT s were significantly related to mid-pool SV s. However, at the fifth turn the TT s for the 98 % 200_{IT} and 100 % 200_{IT} trials were no longer significantly different, probably due to the onset of fatigue reducing the differential between them. Subsequently as fatigue increased TT s were observed to increase markedly over the final turn of the 100 % 200_{IT} and 102 % 200_{IT} trials to the extent that they were significantly slower than the 98 % 200_{IT} trial. Therefore, although swimmers are able to turn faster when not suffering significant fatigue, their ability to do so may become increasingly undermined as fatigue develops. These findings are in agreement with those in Chapter 4 and support the suggestions made by other investigators (Maglishco, 1993; Thompson and Haljand, 1997) that TT s are sensitive to fatigue.

Mean TT s in men's 200 m races have previously been found to be highly correlated with FT (Chapter 4) and useful in terms of predicting FT (Chapter 5); although individual TT s tended to demonstrate greater intra-individual variability as the races progressed, which reduced their predictive power (Chapter 4). In the present study, TT s were also found to be strongly correlated with FT. There were also strong relationships observed between mean TT s across trials suggesting that better breaststrokers will turn consistently faster

than lesser performers at SV s at, or near to maximal 200 m efforts. Within trial relationships were also moderately - highly correlated for the even paced 98 % 200_{TT} and 100 % 200_{TT} trials, however, it was noticeable that relationships between non-consecutive turns were not significant in the positively paced trial (102 % 200_{TT}) which is similar to the findings observed in Chapter 4. Thus it appears that TT s may, like stroke kinematics, be less predictable in a positively paced trial (or race), as changes within TT s are less consistent. In summary, TT s are sensitive to fatigue and prone to greater intra-individual variability during positively paced efforts, when the rate of fatigue is more pronounced.

7.5 Conclusions

The aim of this study was to investigate the effect of manipulating pace on selected kinematic, temporal and metabolic variables during maximal swimming. Subjects were found to pace precisely throughout the whole of the 98 % 200_{TT} trial, 75 % of the 100 % 200_{TT} trial and 50 % of the 102 % 200_{TT} trial suggesting that pacing only became imprecise as fatigue developed. Oxygen uptake and $\dot{V}E$ did not significantly increase as the pace of the trials increased because subjects were exercising at a high percentage of their aerobic power even during the slowest trial. There were no significant differences in any physiological measurements between the 98 % 200_{TT} and 100 % 200_{TT} trials. However, the 102 % 200_{TT} (positively paced) trial produced significant greater post exercise peak lactate, RER and RPE responses compared with the other trials demonstrating that there was an increased anaerobic energy cost and perceived exertion associated with this trial.

Stroke rate and SC were found to increase significantly with the pace of the trials. The increase in SR served to increase SV between trials, and to maintain pace within trials, albeit unsuccessfully in the latter stages of the two faster trials when a disproportionate increase in SC occurred due to fatigue. Both SR and SC were found to be poor predictors of FT. Turning times were significantly shorter in the early stages of the faster paced trials, however the situation was reversed as the trials progressed. Therefore, the turning ability of swimmers appears to be acutely sensitive to fatigue. As in Chapter 4, TT's

were highly related to FT such that better swimmers tended to turn faster. Finally, there was evidence that SR and TT relationships within trials deteriorated in the 102 % 200_{TT} trial possibly due to the greater metabolic stress associated with the trial.

Chapter 8

The effect of even, positive and negative split pacing strategies on kinematic, temporal and metabolic parameters during 175 m breaststroke trials

8.1 Introduction

The pacing of swimming races can be divided into three distinct strategies:

i) evenly split pacing (even pacing) - where the split times are the same throughout the race distance,

ii) positively split pacing (positive pacing) - where the split time for the first half of the race is less than the split time over the second half of the race, and

iii) negatively split pacing (negative pacing) - where the split time for the first half of the race is greater than over the second half of the race.

The dive start and turns have been suggested to be why split times at the halfway point of breaststroke races are often 2-3 seconds shorter than the split time for the final half of the race (Maglischo, 1993). However, Thompson and Haljand (1997) have recently highlighted that national and international 100 m and 200 m breaststroke swimmers also tend to adopt a faster swimming velocity over the first half of their races. This was confirmed from the results of a previous study. Findings from research suggest that positively split pacing might actually be detrimental to performance, however such findings have been

limited to non-swimming activities which took longer to complete than a 200 m breaststroke event and were affected differently by the starting element (> 3 mins, Robinson *et al.*, 1958; Foster *et al.*, 1993; Foster *et al.*, 1994).

Madsen and Lohberg (1987) suggested that 200 m swimming races elicit greater blood lactate values than 100 m races in all strokes and Thompson (1998) has reported supporting data specifically for the breaststroke. Therefore given the strong relationship between elevated levels of blood lactate concentration and fatigue it would appear that pacing is particularly crucial in 200 m events. However to the author's knowledge no studies have been published with regard to pacing interventions in swimming. Indeed few investigations have attempted to determine the effect of different pacing strategies on the outcome of middle distance (2-4 minute duration) events for any exercise modality (Foster *et al.*, 1993). Subsequently it is difficult to make assumptions regarding the 200 m breaststroke event which takes 2.2-3 minutes to complete in competitive swimming.

In the previous study swimmers were observed to adopt a positively paced strategy when attempting to swim at 102 % of their mean 200 m time trial speed (the 102 % 200_{TT} trial), because they were unable to maintain their swimming speed as the trial progressed. This "positively paced" trial was completed in a faster finishing time than the more evenly paced 100 % 200_{TT} trial despite a significantly greater blood lactate concentration and RER value being observed. Unfortunately because of the significant difference in the finishing time of the trials it was not possible to determine if the greater

metabolic stress evident in the positively paced 102 % 200_{TT} trial was because of the pacing strategy or the shorter finishing time.

If swimmers were able to complete breaststroke trials in the same time while undertaking different pacing strategies, it would be possible to identify the metabolic responses associated with the pacing strategies. The aim of this study was to investigate the effect of even, positive and negative split pacing strategies on selected kinematic and metabolic variables during 175 m breaststroke trials at the average speed of a maximal 200 m trial. The effect of pacing strategies on the relationship between kinematic and metabolic variables with finishing time were also investigated.

8.2 METHOD

8.2.1 Subjects

Nine male swimmers (Table 8.1) were recruited from Welsh national swimming squads and four swimming clubs in South and West Wales. Prior to completing the study subjects were fully informed about the demands and procedures of the study and gave their written consent to participate. Health screening and phlebotomy questionaires were administered (UWIC Physiology laboratory procedure) and scrutinised prior to subjects being permitted to begin the studies (Appendix 1). Subjects then read a Volunteer Consent form, outlining the purpose and procedure of the study and gave informed consent prior to beginning the study. Ethical approval for each study was granted by the Ethics Committee of Liverpool John Moores University.

Subjects were informed that they should not attempt a test unless they were in good health and that they could terminate an exercise test at any time. They were asked to report to the laboratory in a rested state having completed no exercise or only very light exercise the previous day. Self report diaries were issued to confirm this when studies took a number of days to complete. They were also asked to abstain from alcohol, caffeine and fatty foods on the day of testing, and not to consume food in the three hours before a test. Information was also given about how to ensure euhydration prior to testing.

Subjects' stature and body mass of the subjects were measured using a portable stadiometer (Holtain) and electronic weighing scales (Seca 770). Arm length was measured by the distance from the acronium process to the end of the middle finger using a standard measuring tape (Chatard *et al.*, 1996). Skinfold measurements were taken with skinfold calipers (John Bull, British Instruments Ltd, England) according to BASES Physiological Testing Guidelines (Bird and Davison., 1987). Finally, hydrostatic mass was measured by applying a known mass to the middle of the swimmer's back until it became just submerged, while the swimmer adopted a tuck position, face down in the water (Chatard *et al.*, 1990). The subject was allowed to take a breath prior to adopting this position.

Table 8.1 - Physical and anthropometric characteristics of the subjects.

Subject	Age	Height	Arm length	Body mass	Hydro-static mass	Sum of 4 Skin-folds	200 m Time Trial time
	(yrs)	(m)	(m)	(kg)	(kg)	(mm)	(s)
AA	22	1.82	0.82	74.2	3.1	31.8	142.9
JL	19	1.83	0.81	75.1	2.2	26.5	148.6
RL	19	1.77	0.82	67.5	2.1	18.5	171.3
JB	21	1.79	0.84	81.2	2.3	35.2	173.2
II	22	1.71	0.78	84.2	1.6	42.1	181.2
PM	24	1.83	0.83	87.8	2.2	31.6	145.3
IF	20	1.78	0.81	74.3	2.2	33.2	151.3
SD	26	1.82	0.82	80.3	2.3	35.2	155.3
GM	18	1.70	0.78	69.8	1.8	25.1	158.3
Mean	21	1.78	0.81	77.2	2.2	31.0	158.6
± s	±3	±0.05	±0.02	±6.7	±0.4	±6.9	±13.6

8.2.2 Experimental design

Subjects completed a maximal 200 m breaststroke time trial (200_{TT}), from which the time taken to complete 175 m at mean time trial pace was calculated. Later subjects completed a series of three, 175 m breaststroke swims, 48 hours apart at either an even, positively split or a negatively split pace. Kinematic (stroke rate, stroke count, turning times), metabolic (heart rate, blood lactate and gas exchange variables) and Rating of Perceived Exertion (RPE) responses were measured.

8.2.3 *Protocol and conduct of the study*

8.2.3.1 Calculation of the pace of the trials

Prior to commencing the main study, each subject completed a maximal 200_{TT} from a push start following a self selected warm up comprising of at least 800 m of swimming. A finishing time for all of the three pacing trials was then calculated as:

175 m times (s) = (200 m 200_{TT} (s) / 8) * 7.

A distance of 175 m was chosen because the previous study demonstrated that during evenly split or positively split paced maximal 200 m trials there was a likelihood that swimmers would not be able to pace precisely over the final

25 m. In this circumstance a comparison of physiological and kinematic responses elicited from differently paced trials would be less meaningful.

At least 72 h after the 200_{TT}, the subjects completed a series of three, 175 m breaststroke swims 48 h apart at either an even, positive or negative pace. The evenly paced trial was swum at the mean 200_{TT} speed throughout. The positively paced trial was swum at 102 % of the mean 200_{TT} speed upto 87.5 m and then at 98 % of the mean 200_{TT} speed over the final 87.5 m. The negatively paced trial was swum in the reverse manner to the positively paced trial. The swims were completed in randomised order and at the same time of day (± 60mins) to minimise diurnal biological variation (Reilly *et al.*, 1984).

8.2.3.2 Standardised warm-up

Prior to each effort the subjects completed a warm-up as previously described in Chapter 6.2.6.2 with the same measurements being taken. Prior to any further swimming, the subject was given a 5 minute recovery period.

8.2.3.3 Trial procedures

The Aquapacer™ was then re-programmed following the warm-up to pace the swimmer's impending 175 m effort, with audible bleeping signals set to coincide with every 12.5 m travelled. The swimmer's pace was set to either elicit an even split, positive split or negative split depending on which trial subject was due to complete. Hand timings were taken at 50 m and at 87.5 m

during each trial to ensure the pacing was precise. Measurements of heart rate (at 87.5 m and post exercise), gas exchange variables (post exercise) and RPE (post exercise) were as described previously in Chapter 6.2.6.3. Measurements of stroke rate (SR) and stroke count (SC) (see Chapter 3.6 and 3.7) were recorded during the 2nd, 3rd, 6th and 7th lengths. Stroke rate and SC values from the second and third lengths were averaged as were the values for the sixth and seventh lengths. Single capillary blood lactate samples were taken from an earlobe at 3, 7, 11 and 13 minutes after the swim. Blood lactate samples were analysed within 24 hours of the trial (Analox GM7) having been removed from the fridge for 30 minutes and remixed. When lactate concentrations > 10 mM were observed 3.5 µl samples were drawn from the lysing capillary tubes for analysis instead of the 7 µl samples drawn for lower lactate values. Duplicate samples were measured in all cases.

8.2.4 Statistical analyses

All procedures were undertaken using the Minitab v11 and SPSS v7.5 software packages. A level of significance of $P<0.05$ was adopted throughout. The Anderson Darling Test was used to test for normality to ensure that parametric analyses could be used. Guidelines for adopting parametric repeated measures analysis, such as considering sphericity as the main assumption to be upheld, were reviewed prior to analysis. In order to estimate the random error within pacing precision, 95 % Limits of Agreement (95 % LoA, Bland and Altman, 1986) were utilised to compare actual times with predicted (target) times. Dependent t-tests and One-way ANOVA were utilised to establish if a large

systematic bias (relative to random error) was present between trials in terms of pacing and metabolic parameters. Factorial ANOVA s were calculated to determine the effect of pacing (Factor A) and time course of the trials (Factor B) on HR, SR, SC and TT. Post hoc comparisons were completed for any significant interactions and for main effects, where there were more than two levels. For post hoc comparisons of interaction effects, a correction developed by Cicchetti (1972) was applied to reduce the chance of Type II errors, by adjusting k (number of groups) to reflect the number of unconfounded comparisons being performed (Heiman, 1999, p 459). For all ANOVA calculations the Huynh Feldt adjustment was used if an epsilon value <0.75 was observed in the analyses (Vincent, 1995), otherwise violation of the assumption of spericity was considered to be minimal. If a significant difference was observed then Tukey's HSD post hoc test was adopted (for ANOVA) and the omega-squared statistic was used to estimate the meaningfulness of the finding. Finally, Pearson's Product Moment correlation coefficient was calculated to determine relationships between variables.

The precision of the pacing was acceptable in all trials, with the evenly paced trial demonstrating the least random error (Table 8.2). The pacing precision ensured that there were significant differences in split times between trials ($P<0.01$) but not in finishing times (FT s).

Table 8.2 - Comparison between predicted and actual times for split times (SpT) and finishing times (FT) (mean ± s) and 95 % Limits of Agreement

Trial	Predicted times (s)	Actual times (s)	t	95 % LoA	Ratio 95% LoA	r
Evenly paced trial						
87.5 m SpT	74.0 ± 6.6	74.1 ± 6.5	0.73	0.1 ± 0.8	-	0.99
175 m FT	148.0 ± 13.2	148.6 ± 13.6	0.10	0.6 ± 1.4	1.01 */÷1.02	0.99
Positively paced trial						
87.5 m SpT	72.5 ± 6.5	72.6 ± 6.4	1.45	0.1 ± 0.4	-	0.99
175 m FT	148.0 ± 13.2	149.0 ± 13.5	0.17	1.0 ± 2.0	1.01 */÷1.04	0.99
Negatively paced trial						
87.5 m SpT	75.5 ± 6.7	75.7 ± 6.9	1.80	0.2 ± 0.7	-	0.99
175 m FT	148.0 ± 13.2	149.2 ± 12.5	0.22	1.2 ± 1.8	-	0.99

* denotes $P<0.05$, ** denotes $P<0.01$

These findings validate the experimental protocol which intended that subjects paced precisely despite experiencing progressive fatigue. The protocol design was based on findings from Chapters 4, 5 and 7 which suggested that limiting the trial distance to 175 m in combination with a 4 % change in pace would allow subjects to pace precisely throughout while allowing the trial to be

reasonably representative of the changes in pace observed in races, albeit without the influence of a dive start and a competitive environment.

Table 8.3 - Comparison of actual split times (SpT) and finishing times (FT) (mean ± s) between trials

	Evenly paced trial	Positively paced trial	Negatively paced trial	F	omega^2
87.5 m SpT	74.1^{a,b}**	72.6^{b}**	75.7	144.4	0.91
	± 6.5	± 6.4	± 6.9		
175 m FT	148.6	149.0	149.2	1.41	-
	± 13.6	± 13.5	± 12.5		

Notes for Table 8.3

** denotes (P<0.01)
a denotes significantly different from Positively paced trial
b denotes significantly different from Negatively paced trial

The 200_{TT} elicited greater blood lactate concentrations and RER values than the other trials. The evenly paced trial demonstrated significantly lower post exercise blood lactate and RER values compared with the positively paced trial and the 200_{TT} (Table 8.4). The 200_{TT} was positively paced (FT 148.0 ± 13.2 s, SpT 72.2 ± 8.6 s) and hence both "positively split trials" demonstrated greater anaerobic energy contributions than the evenly paced trial. RPE was significantly greater in the positively split trials than the evenly paced trial. All other metabolic variables were not significantly different between trials, except for mean trial heart rate which was significantly lower for the negatively paced trial (Table 8.5).

Table 8.4 - Comparison of RPE and selected metabolic variables (mean ± s) between trials

Trial	Evenly paced trial	Positively paced trial	Negatively paced trial	200_{TT}	F	omega^2
Blood lactate (mM)						
Post trial (peak value)	$8.9^{b*,d**}$ ± 2.1	10.9 ± 2.8	9.5 ± 2.9	$11.1^{a**,b,c*}$ ± 3.2	5.86	0.47
Respiratory variables						
$\dot{V}O_2$ ($l.min^{-1}$)	3.59 ± 0.67	3.72 ± 0.80	3.50 ± 0.56	3.78 ± 0.39	0.79	-
$\dot{V}O_2$ / hydrostatic-mass ($l.min^{-1}.kg^{-1}$)						
Number of breaths in 20 s expired air collection	13 ± 3	13 ± 3	13 ± 3	13 ± 2	0.39	-
RER	$1.17^{b,d*}$ ± 0.08	1.24 ± 0.08	1.19 ± 0.13	$1.27^{a**,b,c*}$ ± 0.10	3.82	0.35
$\dot{V}E$ ($l.min^{-1}$)	92.0 ± 26.8	89.2 ± 26.3	97.0 ± 27.6	91.8 ± 20.2	2.08	-
$\dot{V}E/\dot{V}O_2$	25.0 ± 5.7	27.8 ± 5.1	26.4 ± 5.1	29.3 ± 7.4	1.74	-
Rating of Perceived Exertion	$17^{b,d*}$ ± 2	18 ± 1	17 ± 2	18 ± 2	3.82	0.34

Notes for Table 8.4

* P<0.05, ** P<0.01
a denotes significantly different from Evenly paced trial
b denotes significantly different from Positively paced trial
c denotes significantly different from Negatively paced trial
d denotes significantly different from 200_{TT}

Table 8.5 - Comparison of means within subjects (Two factorial ANOVA) for heart rate (mean ± s)

Heart Rate ($b.min^{-1}$)

Main Effects

Pace of trials

Evenly paced trial	Positively paced trial	Negatively paced trial	200_{TT}	F	Sig	$Omega^2$
175 ± 10	176 ± 12	172** ± 11	175 ± 9	5.70	P<0.01	0.43

Timecourse of heart rate measurement

Mean heart rate upto 50 % of trial distance	Mean heart rate during final 50 % of trial distance	F	Sig	$Omega^2$
174 ± 10	176 ± 11	2.50	ns	-

Interaction	F	Sig	$Omega^2$
	2.1	ns	-

Notes for Table 8.5 -
* denotes P<0.05, ** denotes P<0.01

Table 8.6 - Comparison of means within subjects (Two factorial ANOVA) for stroke rate (mean ± s)

Stroke Rate ($S.min^{-1}$)

Main Effects

Pace of trials

Evenly paced trial	Positively paced trial	Negatively paced trial	200_{TT}	F	Sig	$Omega^2$
32.9 ± 6.7	34.5 ± 5.6	31.9^{a*} ± 5.7	35.7 ± 5.9	7.65	P<0.01	0.53

Timecourse of stroke rate measurement

Mean SR upto 50 % of trial distance	Mean SR during final 50 % of trial distance	F	Sig	$Omega^2$
32.6** ± 5.5	34.9 ± 6.5	14.51	P<0.01	0.67

Interaction	F	Sig	$Omega^2$
	5.38	P<0.01	0.47

Notes for Table 8.6 -
* denotes P<0.05, ** denotes P<0.01
a denotes significantly different from 200_{TT}

There was a significant interaction between the pacing pattern of the trials and the timecourse of the trial (Table 8.6). Post hoc analyses detected that between trials the evenly split and negatively split trials demonstrated significantly lower SR measurements than either the positively split trial or the 200_{TT} over the first 50 % of the distance of the trial (Table 8.9). During the final 50 % of the negatively paced trial SR was found to be significantly lower than for the 200_{TT} (Table 8.9). Within trials the stroke rate was significantly elevated during the final 50 % of the trial distance in the evenly split paced ($P<0.05$) and negatively split paced ($P<0.01$) trials (Table 8.10).

Table 8.7 - Comparison of means within subjects (Two factorial ANOVA) for stroke count (mean ± s)

Stroke count (S.25 m^{-1})						
Main Effects						
Pace of trials						
Evenly paced trial	Positively paced trial	Negatively paced trial	200_{TT}	F	Sig	$Omega^2$
9.6 ± 2.2	10.0 ± 1.8	9.3** ± 1.8	10.4 ± 1.9	5.812	P<0.01	0.46
Timecourse of stroke count measurement						
Mean SC upto 50 % of trial distance		Mean SC during final 50 % of trial distance		F	Sig	$Omega^2$
9.3** ± 1.8		10.3 ± 2.0		38.39	P<0.01	0.82
Interaction				F	Sig	$Omega^2$
				2.65	ns	-

Notes for Table 8.7 -

* denotes $P<0.05$, ** denotes $P<0.01$
a denotes significantly different from 200_{TT}

Stroke count was significantly lower in the negatively paced trial than 200_{TT}. Stroke count during the final 50 % of the trials was significantly greater than in the first half of all the trials which suggests that stroke length shortened as trials evolved.

Table 8.8 - Comparison of means within subjects (Two factorial ANOVA) for turning times (mean ± s)

Turning time (s)						
Main Effects						
Pace of trials						
Evenly paced trial	Positively paced trial	Negatively paced trial	200_{TT}	F	Sig	Omega^2
12.6 ± 1.0	$12.4^{a,c,d*}$ ± 1.0	$13.2^{a,b,d**}$ ± 1.1	12.6 ± 1.2	8.81	P<0.01	0.46
Timecourse of turning time measurement						
Turning times upto 50 % of trial distance		Turning times during final 50 % of trial distance		F	Sig	Omega^2
12.6** ± 1.1		12.9 ± 1.3		6.39	P<0.01	0.36
Interaction				F	Sig	Omega^2
				4.65	P<0.01	0.32

Notes for Table 8.8

* denotes P<0.05, ** denotes P<0.01
a denotes significantly different from Evenly paced trial
b denotes significantly different from Positively paced trial
c denotes significantly different from Negatively paced trial
d denotes significantly different from 200_{TT}

There was a significant interaction between the pacing of the trials and the timecourse of the trials (Table 8.8). Upto halfway through the trials turning times (TT s) for the 200_{TT} and the positively paced trial were significantly shorter than for the evenly paced and negatively paced trials. The TT s of the negatively paced trials were significantly longer than the evenly paced trial. During the final half of the trials TT s were not significantly different between trials. Within trials (Table 8.10) the TT s of the 200_{TT} and positively paced

trials lengthened significantly as the trials progressed, while the reverse occurred in the negatively paced trial.

Table 8.9 - Post hoc comparison for interaction effects between trials for stroke rate and turning times (mean ± s)

	Evenly paced trial	Positively paced trial	Negatively paced trial	200_{TT}
Stroke rate ($S.min^{-1}$)				
Stroke rate upto 50 % of trial distance	$31.6^{b*,d**}$ ± 6.3	34.1 ± 5.0	$29.6^{b**,d**}$ ± 5.1	34.9 ± 5.7
Stroke rate during final 50 % of trial distance	34.2 ± 7.2	34.8 ± 6.6	34.1^{d*} ± 5.7	35.4 ± 6.4
Turning Times (s)				
Turning times upto 50 % of trial distance	$12.5^{b,c**}$ ± 1.1	12.1 ± 1.1	$13.5^{a,b,d**}$ ± 1.1	12.2 ± 1.2
Turning times during final 50 % of trial distance	12.6 ± 1.0	12.8 ± 1.0	13.0 ± 1.1	13.0 ± 1.2

Notes for Table 8.9

* denotes P<0.05, ** denotes P<0.01
a denotes significantly different from Evenly paced trial
b denotes significantly different from Positively paced trial
c denotes significantly different from Negatively paced trial
d denotes significantly different from 200_{TT}

Table 8.10 - Post hoc comparison for interaction effects within trials for stroke rate and turning times (mean ± s)

Trials	Stroke rate upto 50 % of trial distance ($S.min^{-1}$)	Stroke rate during final 50 % of trial distance ($S.min^{-1}$)
Evenly paced trial	31.6* ± 6.3	34.2 ± 7.2
Positively paced trial	34.1 ± 5.0	34.8 ± 6.6
Negatively paced trial	29.6** ± 5.1	34.1 ± 5.7
200_{TT}	34.9 ± 5.7	36.5 ± 6.4
	Turning time upto 50 % of trial distance (s)	Turning time during final 50 % of trial distance (s)
Evenly paced trial	12.5 ± 1.1	12.6 ± 1.0
Positively paced trial	12.1** ± 1.1	12.8 ± 1.0
Negatively paced trial	13.5* ± 1.1	13.0 ± 1.1
200_{TT}	12.2** ± 1.2	12.9 ± 1.2

* denotes $P<0.05$, ** denotes $P<0.01$

Of the anthropometric variables only the Sum of 4 skinfolds was significantly related to trial finishing time (FT). Peak lactate and RPE demonstrated a significant relationship with FT in the evenly paced trial ($P<0.05$, Table 8.11). However a large amount of the variance in FT remained unexplained with these variables (r^2, max 61 %, min 49.6 %). Of the kinematic variables only TT s were significantly related to FT.

Table 8.11 - Relationship between finishing time (FT) and selected variables

	FT Even paced trial (s)	FT Positively paced trial (s)	FT Negatively paced trial (s)	FT 200_{TT} (s)
Anthropometric variables				
Age (yrs)	0.19	0.18	0.19	0.20
Body mass (kg)	0.23	0.20	0.22	0.25
Hydrostatic mass (kg)	-0.61	-0.59	-0.60	0.61
Sum of 4 skinfolds (mm)	0.69*	0.68*	0.68*	0.70*
Height (m)	-0.64	-0.61	-0.63	-0.62
Arm length (m)	-0.40	-0.36	-0.37	-0.38
Metabolic variables				
lactate post warm-up (mM)	0.25	0.29	0.40	0.30
lactate peak (mM)	-0.76*	-0.73*	-0.70*	-0.69*
Heart rate at 87.5 m ($b.min^{-1}$)	-0.60	-0.45	-0.60	-0.51
Heart rate at 175 m ($b.min^{-1}$)	-0.56	-0.59	-0.53	-0.45
$\dot{V}O_2$ ($l.min^{-1}$)	0.64	0.65	0.56	0.64
$\dot{V}O_2$/hydrostatic mass ($l.min^{-1}.kg^{-1}$)	0.66	0.67	0.52	0.63
RER	0.23	0.12	0.43	0.13
$\dot{V}E$ ($l.min^{-1}$)	0.45	0.35	0.46	0.23
$\dot{V}E/\dot{V}O_2$	-0.32	-0.41	-0.12	-0.55
Rating of perceived exertion				
RPE	-0.71*	-0.61	-0.32	-0.59
Kinematic variables				
Stroke rate ($S.min^{-1}$)	-0.20	-0.15	-0.10	-0.15
Stroke count ($S.25\ m^{-1}$)	-0.31	-0.41	-0.25	-0.35
Turning times (s)	0.90**	0.85**	0.93**	0.87**

* denotes $P<0.05$ ** denotes $P<0.01$

Table 8.12 - Relationships between selected metabolic variables (and rating of perceived exertion) between the paced trials

	Positively paced trial	Negatively paced trial	200_{PT}
Heart rate at 87.5 m ($b.min^{-1}$)			
Even paced trial	0.83**	0.75*	0.95**
Positively paced trial		0.72*	0.90**
Negatively paced trial			0.91**
Heart rate at 175 m ($b.min^{-1}$)			
Even paced trial	0.89**	0.95**	0.96**
Positively paced trial		0.95**	0.89**
Negatively paced trial			0.92**
lactate post warm up (mM)			
Even paced trial	0.56	0.45	0.48
Positively paced trial		0.59	0.46
Negatively paced trial			0.36
Peak lactate (mM)			
Even paced trial	0.85*	0.94**	0.85*
Positively paced trial		0.83*	0.80*
Negatively paced trial			0.86*
$\dot{V}O_2$ ($l.min^{-1}$)			
Even paced trial	0.77*	0.71	0.88**
Positively paced trial		0.67	0.89**
Negatively paced trial			0.44
$\dot{V}O_2$/hydrostatic mass ($l.min^{-1}$)			
Even paced trial	0.82*	0.72	0.91**
Positively paced trial		0.63	0.93**
Negatively paced trial			0.50
RER			
Even paced trial	0.70	0.96**	0.50
Positively paced trial		0.67	0.86**
Negatively paced trial			0.44
$\dot{V}E$ ($l.min^{-1}$)			
Even paced trial	0.96**	0.97**	0.59
Positively paced trial		0.95**	0.62
Negatively paced trial			0.59
$\dot{V}E/\dot{V}O_2$			
Even paced trial	0.95**	0.81*	0.52
Positively paced trial		0.83*	0.67
Negatively paced trial			0.83*
RPE			
Even paced trial	0.65	0.31	0.57
Positively paced trial		0.46	0.67
Negatively paced trial			0.46

* denotes $P<0.05$, ** denotes $P<0.01$

Peak lactate and HR responses were significantly related between all trials ($P<0.01$, Table 8.12). Heart rate, peak lactate, $\dot{V}0_2$, $\dot{V}0_2$ / hydrostatic mass and RER

demonstrated a significant relationship between the 200_{TT} and the positively paced trial. A significant relationship was also observed between the evenly paced trial and the negatively paced trial for heart rate, peak lactate, $\dot{V}E$ and $\dot{V}E / \dot{V}0_2$.

Table 8.13 - Relationships between selected kinematic and temporal variables between trials

	Positively paced trial	Negatively paced trial	200_{TT}
Stroke rate upto 50 % of trial distance			
Even paced trial	0.87*	0.96**	0.90**
Positively paced trial		0.91**	0.81*
Negatively paced trial			0.91**
Stroke rate during final 50 % of trial distance			
Even paced trial	0.91**	0.92**	0.91**
Positively paced trial		0.93**	0.88**
Negatively paced trial			0.88**
Stroke count upto 50 % of trial distance			
Even paced trial	0.92**	0.95**	0.96**
Positively paced trial		0.91**	0.89**
Negatively paced trial			0.90**
Stroke count during final 50 % of trial distance			
Even paced trial	0.86*	0.98**	0.99**
Positively paced trial		0.90**	0.84*
Negatively paced trial			0.96**
Turning time upto 50 % of trial distance			
Even paced trial	0.90**	0.83*	0.93**
Positively paced trial		0.81*	0.95**
Negatively paced trial			
Turning time during final 50 % of trial distance			
Even paced trial	0.65	0.81*	0.60
Positively paced trial		0.50	0.84*
Negatively paced trial			0.48

* denotes $P<0.05$, ** denotes $P<0.01$

Stroke rate, SC and TT were all significantly related between trials up to 50 % of the trial distance (Table 8.13). This indicates interindividual relative stability despite changes in pacing. During the final half of the trials TT s were significantly related between the two positively split trials (positively paced trial and the 200_{TT}) and also between the evenly paced trial and the negatively

paced trial. Within the trials significant relationships were observed for SR and SC in all trials (Table 8.14). Turning times were significantly related within the evenly paced and negatively paced trials.

Table 8.14 - Relationships between selected kinematic and temporal variables within trials

Variable upto 50 % trial distance vs Variable during final 50 % of trial distance	r
Stroke rate	
Even paced trial	0.98**
Positively paced trial	0.95**
Negatively paced trial	0.94**
200_{TT}	0.97**
Stroke count	
Even paced trial	0.99**
Positively paced trial	0.88**
Negatively paced trial	0.88**
200_{TT}	0.99**
Turning times	
Even paced trial	0.89**
Positively paced trial	0.65
Negatively paced trial	0.81*
200_{TT}	0.56

* denotes $P<0.05$ ** denotes $P<0.01$

8. 4 Discussion

Acceptable agreement was observed between predicted and actual times and subsequently split times were significantly different between trials while the finishing times (FT s) were not. This suggests the swimmers habituated well to the Aquapacer ™ and validates the design of the protocol, which intended that the swimmers attain the same FT despite swimming each of the 3 trials in a different manner. Random error was less evident in the FT of the evenly paced trial but was similar for split times across trials. This difference may be attributed to the absence of a change of pace in the evenly paced trial and suggests that changing pace within an intense swim may increase the chance of a pacing error.

The positively paced trial was found to exhibit significantly greater blood lactate and RER values than the evenly paced trial despite FT s not being significantly different. This is a similar finding to that observed in Chapter 7 where a significantly greater lactate concentration was elicited from the "positively paced" 102 % 200_{TT} trial compared with the more "evenly paced" 100 % 200_{TT} trial, except that in Chapter 7 the "positively paced" 102 % 200_{TT} trial was found to produce a faster FT. Therefore it appears that the raised blood lactate in Chapter 7 was at least partly due to the type of pacing observed (positive) and not merely because a slightly faster trial speed was achieved.

High blood lactates were observed in all trials as glycolysis contributes significantly to ATP resynthesis from anaerobic metabolism in maximal

exercise taking between 120 -180 s (Karlsson and Saltin, 1970; Bangsbo *et al.*, 1990; Medbo and Tabata, 1993). However the measured increase in blood lactate in the positively paced trial would indicate that a small increase in swimming velocity at the beginning of a maximal breaststroke effort requires an increased energy contribution from anaerobic glycolysis, because blood lactate release has been shown to be almost linearly related to the muscle lactate gradient (Bangsbo *et al.*, 1993).

Muscle lactate accumulation is more rapid during intense exercise (Karlsson and Saltin, 1970; Jacobs *et al.*, 1983; McCartney *et al.*, 1983; Cheetham *et al.*, 1986) so it is likely that a faster starting positively paced trial would cause a more rapid accumulation of lactic acid in the muscle during the first half the trial (Cherry *et al.*, 1997). For instance Foster *et al.* (1993b) suggested that small changes in exercise intensity, within a range of already high intensities, might impact significantly on muscle lactate concentration. In the present study the early lactate accumulation may have been due to a greater recruitment or activation of recruited fast glycolytic fibres as the swimmers coincidentally exhibited a greater stroke rate in the first half of the trial. Alternatively the rate of oxygen uptake and hence aerobic energy contribution may have been less well matched to the energy requirements at this work rate.

These assumptions may explain why a greater initial lactate accumulation occurred at the beginning of the positively paced trial, however it must be remembered that in the present study blood lactate was measured post exercise, which means that a greater absolute lactate accumulation was observed at the

end of the positively paced trial despite a slower swimming speed over the final 87.5 m. This finding might indicate that the greater emphasis on fast glycolytic fibre recruitment during the faster start to the trial did not ease during the slower part of the trial. This may have been due to the maintenance of a high SR throughout the trial, or an earlier onset of metabolic acidosis which compromised aerobic metabolism and force production due to the subsequent inhibition of aerobic enzyme activity and contractile mechanism in the slow oxidative muscle fibres.

The 200_{TT} yielded significantly greater blood lactate and RER values than the other trials. This was probably due to a combination of the increased swimming duration and the positively split pacing observed in this trial. The choice of pacing in this trial would also indicate that subjects were typical of competitive breaststroke swimmers, as the strategy of positive pacing was also freely chosen by the majority of swimmers reported in Chapter 4. However this tendency towards a positive pace is hard to explain because the swimmers in the present study reported significantly higher RPE values in positively split trials compared to the evenly paced trial, probably due to increased metabolic acidosis (Kostka and Cafarelli, 1982). Also there is little if any evidence to suggest that positively split pacing can elicit shorter FT s in breaststroke swimming compared with other pacing methods. Indeed the only experimental evidence to support this in swimming was observed in Chapter 7, although Ariyoshi *et al.* (1979a) have reported that a fast/slow pattern of effort over a 1,400 m run allowed for better endurance on a subsequent all out sprint.

However they also observed a smaller oxygen debt which is contradictory to the findings of the present study.

It is perhaps surprising that many coaches encourage swimmers to pace positively as it is well known that an increase in muscle lactate accumulation and resultantly low pH are thought to be primary factors in the development of fatigue in intense exercise of this duration (Foster *et al.*, 1994). The findings of this study might suggest that an even or negative pacing strategy might be more suitable for 200 m breaststroke events. In support of this Van Schenau *et al.* (1994) have suggested that in exercise lasting longer than 80-100 s even pacing is preferable while Foster *et al.* (1994) have suggested that even pacing is preferable, in middle distance and longer events with dominant aerobic energy contribution.

In contrast to the present study, Foster *et al.* (1993a) and Leger and Ferguson (1974) observed no significant differences in blood lactate concentrations in cycling and running when positively paced trials were compared with evenly and negatively paced trials. Ariyoshi *et al.* (1979b) however reported a reduced lactate concentration for a fast/slow paced 1,400 m run. None of these studies are directly comparable to the present one. Foster *et al.* (1993a) analysed only a single blood lactate sample 3 minutes after exercise while in this study a number of post exercise blood lactate samples were taken which allowed for a more accurate determination of a peak sample. Also Foster *et al.* (1993a) only paced their subjects to the mid-point of the trial so that pacing was left uncontrolled during the second half. Therefore although an evenly paced cycle

trial elicited a faster 2000 m FT this may have been due to the subjects pacing the second half of the trial better in this trial than in the other ones, rather than there being performance differences resulting from physiological responses. Indeed Foster *et al.* (1993a) found no differences in the physiological parameters they measured between trials. Leger and Ferguson (1974) adopted a complex experimental design comparing two pacing strategies incorporating two changes in pace. However an identical middle section occurred in both trials which may have marginalised the physiological differences between the trials immediately following the initial paces of the trials. Ariyoshi *et al.* (1979b) also used a complex protocol (4 changes in speed) for their positively and negatively paced trials, but also required that all their subjects ran at the same speeds irrespective of ability. This would have made the trials much more physically demanding for some of their subjects than for others. Finally the mean RPE values they reported (13-15) would suggest that their subjects were exercising at a lower intensity than the subjects in the present study.

The findings of the present study are supported by Robinson *et al.* (1958) whose subjects also attempted to complete a distance (~ 1,245 m running) in a certain time using the three pacing strategies. They observed that positive pacing led to a greater blood lactate accumulation compared with evenly or negatively split trials. The starting pace of their positively paced trial (107 % of the even pace) was more exagerated than in the present study and consequently may have produced a critical muscle lactate value and associated fatigue well before the conclusion of the trial (Foster *et al.* 1993b).

The negatively paced trial of the study by Robinson *et al.* (1974) elicited less lactic acid and total oxygen consumption than the other trials, which was attributed to oxygen uptake kinetics being more closely matched to the energy demand at the beginning of the exercise. However in the present study there were no differences observed in post exercise $\dot{V}O_2$, $\dot{V}E$ or $\dot{V}E$ / $\dot{V}O_2$ responses between trials. These findings were also common to a number of other pacing studies which have measured ventilatory measurements during and after exercise (Adams and Bernaur, 1968; Leger and Ferguson, 1974; Ariyoshi *et al.*, 1979; Foster *et al.*,1993; Chapter 7). Leger and Ferguson (1974) concluded this to mean that there was no redistribution of the (aerobic) energy source despite subtle manipulations in pacing. These findings may have been due to the swimmers operating at very near to their maximal aerobic power at trial cessation, because the fast component of the EPOC (where ventilation was measured in the present study) increases linearly with work rate (Barstow, 1994) and hence near maximal aerobic measurements would produce similar EPOC responses. This suggestion is reasonable considering that the swimmers in the present study were exercising for more than 2 min and 40 s on average and it has been shown that 85 % of $\dot{V}O_2$ max can be attained during a 30 s Wingate test (Kavanagh and Jacobs, 1987) or over 2 minutes of exercise during a 4 minute run (Ariyoshi *et al.*, 1979b).

Differences in findings between the present study and the study by Robinson *et al.* (1958) can be explained in a number of ways. Firstly their findings were based on only three subjects, and only one of these was reported to be maximally exerting himself by the end of the trial. Secondly, the subjects in the

present study were more highly trained which allowed them to sustain exercise at much higher levels of physical exertion. Buffering capacity can increase as a result of short term endurance training (Robergs and Roberts, 1997) and this might have enabled the swimmers to achieve their predicted FT s in the positively paced trial despite higher lactate concentrations than those reported by Robinson *et al.* (1958). The post exercise RER values of the present study confirm that an exhalation of extra CO_2 took place which suggests CO_2 tissue accumulation had occurred due to buffering. Thirdly, aerobic training can impart greater mitochondrial and capillary densities and lead to faster $\dot{V}O_2$ kinetics (Barstow, 1994). This adaptation might explain why the swimmers demonstrated no significant differences in post exercise ventilatory values between trials, as they were more able to approach their maximal aerobic power at the cessation of the trial irrespective of the pacing strategy involved.

Finally, there might have been sufficient random error present in the ventilatory measurements of the present study that it was not possible to detect subtle differences between trials. However, this is unlikely as Chapter 6 demonstrated acceptable agreement between ventilatory measures following identical bouts of breaststroke swimming and Ariyoshi *et al.* (1979b) have also observed non-significant differences in $\dot{V}O_2$ between differently paced trials where a 3 % change in pace occurred.

Heart rate response was significantly lower in the negatively paced trial compared to the other trials, which may have been due to the slower initial pace reducing the HR kinetics of this trial.For example in Chapter 7 the HR response

halfway through the 98 % 200_{TT} trial was significantly lower than for the faster starting 100 % 200_{TT} and 102 % 200_{TT} trials, although there were no differences in HR response between these trials by the end. A similar finding was observed by Ariyoshi *et al.* (1979a) who reported a significantly lower HR response in a slower starting trial compared to a faster starting trial after 60 s of exercise. A lower heart rate response is of some importance as cardiac output is thought to be one mechanism driving $\dot{V}O_2$ kinetics at the start of exercise (0 s to 15-25 s, Wasserman *et al.*, 1974). Consequently a lowered HR response may imply a compromise of the aerobic energy contribution and may explain why the negatively paced trial failed to demonstrate a lower mean lactate concentration compared to the evenly paced trial. Although the attempt to pick up the pace in the final 87.5 m may also explain this finding.

However, a further explanation is that in the negatively paced trial two subjects produced a much lower HR response than for the others which subsequently reduced the relationship between the HR response from this trial and the others. Therefore the bias observed may actually be due to measurement error within these specific measurements, rather than a change in physiological status resulting from the pacing strategy.

Peak lactate demonstrated a significant negative relationship with FT in all trials suggesting that faster times are achieved by those who exhibit greater blood lactate values. These findings support Holmer (1974) who stated that faster 200 m swimmers require a pronounced anaerobic capacity. It appears that faster swimming speeds in breaststroke swimming seem to be

achieved by an increased anaerobic energy contribution possibly reflecting the additional recruitment of the less efficient fast twitch muscle fibres. The ability to buffer lactic acid in order to maintain force production might also differentiate faster swimmers from slower swimmers particularly in unevenly paced trials.

Peak $\dot{V}O_2$ does not appear to be a useful predictor of performance in 175 m and 200 m breaststroke swimming because ventilatory measures were not significantly related to FT in this study or in Chapter 7 (during the 100 % 200_{TT} and 102 % 200_{TT} trials). Given the intensity of the trials and the homogeneous measurements for peak $\dot{V}O_2$ it would appear that there was little difference in terms of the peak aerobic power of the swimmers despite the subject group being relatively heterogeneous in terms of performance (200_{TT} min 142.9 s, max 181.2 s). D'Aquisto *et al.* (1988) have also reported non-significant differences in $\dot{V}0_2$ between better and poorer male breaststrokers during a maximal 400 yd swim.

It seems suprising that the faster swimmers did not demonstrate a greater peak $\dot{V}O_2$ as a result of the extra aerobic training they were routinely undertaking. However, at the time of this study the faster swimmers were also undertaking a greater volume of higher intensity swimming to improve their anaerobic capabilities prior to competition. This type of training may have concomitantly decreased their aerobic capacity as improvements in anaerobic capacity have been suggested to coincide with a reduction in aerobic swimming economy in swimmers (Olbrecht, 2000). Thus it might be that changes in anaerobic energy

utilisation which allow one breaststroker to swim faster than another over 200 m, may also diminish interindividual differences in aerobic capacity. Alternatively the faster swimmers did not have any more, or longer, oppurtunities to breath than the slower swimmers during trials (as evidenced by SR and SC not being related to FT), which may have caused ventilatory measurements to have been "capped". Indeed it appears that SR does not alter much beyond a narrow range of frequency during high intensity breaststroke swimming, which means it may be difficult to increase ventilation beyond a certain point. For example SR was only elevated by 5 $S.min^{-1}$ despite a 4 % difference in pace between the Negatively paced and Positively paced trials. If limited ventilatory oppurtunities do "cap" the aerobic power during breaststroke swimming whatever the standard of swimmer, then this may explain why the anaerobic capacity becomes the defining energy producing mechanism between swimmers of different standards.

Of the anthropometric parameters the sum of 4 skinfolds indicated a positive significant relationship with FT suggesting that a lower body fat is related to higher performance. However the coefficient of determination was only 0.49 and so the majority of variation in FT was not explained by this factor. A similar finding was apparent in Chapter 7 so it would seem that faster breaststroke swimmers also tend to possess a lower body fat, although the lowest body fat measured in this study, 10.5 % is not particularly low. A previous study has reported body fat scores ranging from 5 % - 10 % for elite male swimmers (Lavoie and Montpetit, 1986).

Stroke rates were significantly lower in the evenly and negatively paced trials compared to the 200_{IT} and positively paced trials, during the first half of the trials but not the second half. This was due to the SR increasing in the evenly and negatively paced trials as they progressed. In the evenly paced trial SR probably increased as fatigue developed because a significant increase in SC was evident, while in the Negatively paced trial a combination of fatigue and an attempt by the swimmers to increase swimming velocity (following the slower start) may have accounted for the rise in SR. The greater initial SR s observed in the positively split trials undoubtably increased the swimmer's initial speed, however in the latter stages of these trials the SR reached a plateau. This was probably to maintain stroke efficiency, as increasing SR further at this point might have resulted in a loss in distance per stroke, while decreasing SR and increasing distance per stroke would probably have been even less efficient.

It appears that in high intensity breaststroke swimming increases in SR are confined to a small range. It is also apparent that a swimmer cannot operate at the upper end of this SR range throughout a 175 m trial without a significant deterioration in distance per stroke. Unfortunately at this point the swimmer is then unable to increase the SR further to compensate for this and so slows down. However, when a swimmer begins with a more evenly paced effort, then the SR is initially lower and fatigue develops less rapidly. The SR can then be significantly increased to maintain SV as distance per stroke deteriorates. However at the present time this practice is not widely used amongst competitive breaststroke swimmers.

The mean SC in the 200_{TT} was significantly greater than in the negatively paced trial. This was probably due to greater fatigue being evident in the 200_{TT} trial due to the extra 25 m being completed. Findings in the previous study would support this as there were marked increases in SC over the final 50 m of both the 100 % 200_{TT} and 102 % 200_{TT} trials. Finally, SR and SC were found to correlate poorly with FT. This has been attributed to breaststroke swimmers possessing a unique SR : SC combination in Chapters 4, 5 and 7. The significant correlations between and within trials in this study and in Chapter 7 also suggest that these characteristics are generally maintained irrespective of pacing strategy and progressive fatigue. Therefore those swimmers demonstrating comparitively high stroke rates at one speed will do so at another during high intensity breaststroke swimming.

Turning times (TT s) were found to demonstrate a significant interaction between the pace of the trials and the timecourse of the trials. In the first half of the trials the TT s in the positively split trials (positively paced trial & 200_{TT}) were significantly shorter than those completed in the Evenly and Negatively paced trials. The TT s of the negatively paced trials were also significantly slower than the evenly paced trial. Therefore the initial pace of these trials seems to have affected the speed of the turns. This is consistent with swimmers demonstrating shorter TT s when being paced at greater velocities (Chapter 7) and when exhibiting faster mid-pool swimming velocities (Chapter 4). These observations may explain why a strong relationship has been found to exist between mean TT and FT in all of these studies.

TT s increased significantly as positively split trials evolved. In contrast TT s did not alter significantly as the evenly paced trial progressed and significantly improved in the final half of the negatively paced trials. Consequently there were found to be no differences in TT s between trials during their final half. Since TT s appear to be acutely sensitive to fatigue (Chapter 7) the evenly and negatively paced trials seem to be demonstrating less evidence of this which is consistent with the level of lactacidosis observed in these trials.

The improvements seen in TT s in the negatively paced trial were probably due to:

i) the swimmers increasing their swimming speed over the second half of the trial, and

ii) because the initial TT s in this trial took disproportionately longer than the other trials which meant there was room for significant improvement when the swimmers increased their swimming speed later in the trial.

The disproportionately slow turns in the negatively split trial may have been due to swimmers making greater errors when approaching early turns at a slow pace. Alternatively the swimmers may have waited on the turns to get back on pace if they had begun the trial too quickly, because in the 98 % 200_{TT} trial of Chapter 7 some swimmers did report that they had "waited on the wall" when they had got ahead of the pace.

During the first half of the trials TT s were significantly correlated between trials demonstrating that swimmers tended to turn in a consistent relative order when not fatigued. However, correlations between the positively split trials and the other trials deteriorated over the latter part of the trials. Poor correlations observed for TT s within the positively paced trials may account for the deterioration in these relationships. Finally, TT s have been shown to demonstrate greater intra-individual variability toward the end of 200 m efforts in positively split men's races (Chapter 4) and trials (Chapter 7). It now seems that a likely cause might be the debilitating effect of metabolic acidosis as increases in TT error have coincided with a greater appearance of blood lactate in the positively split trials of this study and Chapter 7.

8.5 Conclusion

The primary aim of this study was to investigate the effect of even, positive and negative pacing on metabolic, kinematic and temporal responses during near maximal 175 m breaststroke trials. In all of the trials subjects were found to pace precisely, although less error was evident in the FT of the evenly paced trial which may have been due to less fatigue or because the constant pace helped the swimmer's to pace more easily. The evenly paced trial required less anaerobic energy contribution compared with the positively paced trial and the perception of effort was significantly lower. Post exercise cardiovascular and ventilatory measurements were similar across all pacing strategies because swimmers were operating at near peak aerobic power irrespective of the pacing strategy.

Stroke rate was manipulated by the swimmers to achieve the desired pace. In the evenly paced trial SR progressively increased to maintain speed as distance per stroke deteriorated, while in the negatively paced trial SR was increased in the latter part of the trial to elevate swimming speed. However in the positively paced trial the SR remained unchanged throughout despite SC increasing and subsequently swimming speed fell. Turning times were sensitive to swimming velocity and fatigue as they deteriorated, remained unchanged and improved during the positively paced, evenly paced and negatively paced trials respectively. As a result no differences in TT s were observed between trials in their latter halves.

The reduced blood lactate, RPE and unchanged TT s observed in the evenly paced trial suggests that less physical strain was evident in this trial compared with the positively paced trial, which means that even pacing is an alternative pacing strategy for competitive breaststroke swimmers.

Finally, the effect of pacing strategy on the relationship between kinematic, temporal and metabolic variables and FT was investigated. Irrespective of the pacing strategy, peak blood lactate demonstrated a negative relationship with FT while sum of 4 skinfolds and mean TT s demonstrated a positive relationship with FT indicating that a greater anaerobic energy contribution, lower body fat and shorter mean turning time are associated with faster breaststroke swimming over 175 m.

Chapter 9

General Discussion and Conclusions

9.0 General Discussion

9.1 Summary of key findings and application of these findings to coaching

This thesis aimed to provide a better understanding of breaststroke swimming from an examination of it's kinematic, temporal and metabolic characteristics, including the effect of changing pace. Five studies were undertaken to achieve these aims. The first 2 studies evaluated kinematic and temporal variables during 100 m and 200 m breaststroke races in order to establish inter-relationships, assess changes within variables during the evolution of races, identify characteristic differences between 100 and 200 m events and determine the relative importance of these variables to swimming performance.

Mean mid-pool swimming velocity (SV), start time (ST) and mean turning time (TT) were found to be significantly related to finishing time (FT) (Table 9.1) and to each other, suggesting that the coach should adopt an holistic approach to the training of breaststroke swimmers. Indeed coaches and sports scientists should regularly measure ST s and TT s in training and competition so that the swimmer receives precise and objective feedback about them, as they account for approximately 30 m of a 100 m race and 60 m of a 200 m race. Currently the feedback given to swimmers by coaches in British competitions rarely contains objective measurements of the ST and TT s, rather a great deal of discussion takes

place about the general measure of split times, which is insufficient information on which to base an evaluation of race performance. Predictive equations were developed from the second study to help the coach to monitor and evaluate improvements in these kinematic variables.

Turning times were found to be sensitive to fatigue, particularly during positive paced swimming. For this reason and the fact that TT s were significantly related to FT it was suggested that TT s may be a useful performance indicator for coaches. Start time was found to be relatively more important than TT in the male 100 m event while the reverse was true for the 200 m event (Table 9.1). Furthermore values for mid-pool SV s and SR s were significantly greater in the 100 m while ST s, TT s and SL s were significantly lower. These findings indicated that 100 m and 200 m races should be prepared for differently, and arguably that swimmers should specialise in one of these events. It was also felt that there is a potential for improvement in the ST of 200 m swimmers.

Table 9.1 Factors determining performance for breaststroke swimming

Event	Variable	Relative importance	Relationship with Finishing time (FT)	Pacing (positive, even, negative)	Chapters
100 m men	Mid-pool SV ($m.s^{-1}$)	1°	-ve	positive	4, 5
	ST (s)	2°	+ve	positive	4, 5
	TT (s)	3°	+ve	positive	4, 5
100 m women	Mid-pool SV ($m.s^{-1}$)	1°	-ve	positive	4, 5
	TT (s)	2°	+ve	positive	4, 5
	ET (s)	3°	+ve	positive	4, 5
	ST (s)	4°	+ve	positive	4, 5
200 m men	Mid-pool SV ($m.s^{-1}$)	1°	-ve	positive	4, 5
	TT (s)	2°	+ve	positive, even, negative	4, 5, 7, 8
	ET (s)	3°	+ve	positive	4, 5
	ST (s)	4°	+ve	positive	4, 5
	Peak La post-exercise (mM)	?	-ve	positive, even, negative	7, 8
	Change in La (peak - rest) (mM)	?	-ve	even	7
	Peak $\dot{V}O_2$ ($l.min^{-1}$)	?	-ve	even	7
	Peak $\dot{V}O_2$/ hydrostatic mass ($l.min^{-1}.kg^{-1}$)	?	-ve	even	7
	Sum of 4 skinfolds score (mm)	?	+ve	positive, even, negative	7, 8
	Hydrostatic mass (kg)	?	-ve	even	7
	RPE	?	-ve	even	8
200 m women	Mid-pool SV ($m.s^{-1}$)	1°	-ve	positive	4, 5
	TT (s)	2°	+ve	positive	4, 5

KEY: Finishing time - FT, mid-pool swimming velocity - SV, start time - ST, turning time - TT, end time (final 5 m) - ET, La - blood lactate, $\dot{V}O_2$ - oxygen uptake

Data from male 200 m and 175 m trials in Chapters 7 and 8 demonstrated that a number of metabolic and anthropometric variables were also related to FT (Table 9.1). Post-exercise peak blood lactate was found to be negatively related to FT whether the pacing was positively split (as in races), evenly split or negatively split, indicating that 200 m breaststroke swimmers require a high anaerobic capacity. It

should be noted that a number of other variables were found to be related to FT only when the pacing of the trials was evenly split. These findings may have limited practical application to coaches given that the majority of breaststroke races are positively split as their relative importance to race performance is difficult to predict (Table 9.1). However as a high anaerobic capacity appears to be a pre-requisite for performance, the use of buffering agents (sodium bicarbonate and sodium citrate) in training and competition may be useful in attenuating fatigue during 200 m breaststroke swimming (Simmons and Hardt, 1973; Inbar *et al.*, 1981; Wilkes, *et al.*, 1983; Parry-Billings and MacLaren, 1986; McNaughton and Cedaro, 1992).

Stroke rate (SR) and stroke length (SL) were found to be poorly correlated with FT because individual swimmers adopt unique SR-SL ratios. Additionally in Chapters 7 and 8 it was observed that swimmers with high SR s and stroke counts (SC s) maintained this trend during trials, which indicated that better swimmers do not necessarily complete fewer stroke cycles during races as has been previously suggested (Craig *et al.*, 1979; Wakayoshi *et al.*, 1992).

Swimmers increased SR in preference to SL when they increased their swimming speed during high intensity swimming trials. It was also found that SR s became elevated to compensate (unsuccessfully) for a disproportionately decreasing SL over the course of 100 m and 200 m races. There was evidence during 175 m

maximal trials (Chapter 8) that this increase in the SR-SL ratio could be attenuated when subjects swam at an even pace rather than a positively split pace (as found in races). It is suggested that coaches could identify the most efficient SR-SL ratio for an individual swimmer during even paced swimming, and then entrain it in order to ascertain if it produces a more consistent kinematic response and even pace in competition.

The Aquapacer^{TM} can be used to pace a SR and so may be of use for training a swimmer to maintain the SR that is most productive in terms of SV. The stage of a training set at which the swimmer's mid-pool SV deteriorates due to a fall in SL could then be monitored over the course of a season to determine progress. However the SR might need to be adjusted slightly if the swimmer becomes more or less able to maintain SL at the identified SR (McMurray *et al.*, 1990). The coach would need to undertake regular monitoring to determine this. For example, race simulation training could be undertaken by swimmers so that fine adjustments are made when required eg. in a swimmer's SR. As the ability of male 200 m swimmers to maintain technique is less predictable under conditions of metabolic acidosis (Chapter 4) then race simulation training appears to be crucial in this event to try to ensure that swimmers consistently produce a level of performance with regard to their SV and turns in competition.

The Aquapacer™ was observed to elicit precise and reliable pacing across a range of speeds (Chapters 6 to 8) and so may provide the coach with a method for entraining race specific pacing. A race-specific test could be developed by programming the Aquapacer™ to exercise the swimmer at the race pace SR over a set distance, as any improvements in mean SL and mean TT (and hence FT) over repeated tests would provide a strong indication of the potential for a faster race performance. This type of test would be more suited to 50 m pools as they are more physically demanding (Lowenstyn *et al.*, 1994) and would allow the swimmer sufficient time to adopt the SR between the turns. The coach would then have a tool for the non-invasive determination of improvements in event-specific performance, although taking physiological and psychological measurements at the same time should provide further explanation as to why a performance may have improved. Metabolic, kinematic and RPE responses were found to be reproducible during even paced swimming and so these variables could also be used to monitor the training adaptations of swimmers. Finally, the Aquapacer™ accurately paced swimmers through a change in pace (Chapter 8) suggesting that it might be a useful tool for training a swimmer to learn a complex race strategy.

Study 4 assessed the effect on kinematic, temporal and metabolic variables when pacing was subtly manipulated during high intensity trials. The lack of significant differences in ventilatory and cardiovascular responses at the end of trials paced at, just below and just above the mean speed for a 200 m time trial meant that

cardiorespiratory variables were found to be poor predictors of FT. These findings also suggest that breaststroke swimmers exercise at an intensity close to their peak aerobic power during near maximal or maximal 200 m efforts. Subsequently the additional energy production needed to increase SV during high intensity breaststroke swimming comes from anaerobic glycolysis. In support of this, peak blood lactate values were found to be significantly negatively correlated with FT (Table 9.1). Consequently it is suggested that coaches need to program training sets to elicit a high percentage of their swimmer's aerobic power and at intensities beyond this in order to stimulate race specific metabolic adaptations.

It was observed during high intensity breaststroke swimming that the SR could only be manipulated within a small range without causing premature metabolic acidosis. Consequently the suggestion made by Maglischo (1993) that generalisations can be made regarding the best stroke rates for breaststroke events may be inappropriate because a small error in this estimation might be catastrophic for an individual's performance (Fig 9.1). Coaches need to spend time with individual swimmers identifying precisely a racing SR in order to ensure consistency in performance. However swimmers may not be able to avoid a decrease in their SL over the course of a maximal 200 m effort even if they are swimming at their ideal SR (Fig 9.1).

Stroke kinematics were observed to be more significantly related throughout a maximal 200 m swim when pacing was evenly split rather than positively split.

This was thought to be the result of less muscular fatigue leading to fewer technical problems such as a loss of timing within the stroke cycle. For this reason a fifth study was undertaken to compare positive, even and negative pacing strategies.

Study 5 assessed the effect of even, positive and negative split pacing strategies on kinematic, temporal and metabolic responses during 175 m trials. This was a novel study as to date no studies have undertaken a comparison of pacing strategies in swimming, and of those which have for other exercise modalities, few have been conducted over a 2-4 minute period. It was observed during the evenly split trial that subjects demonstrated reduced blood lactate, RER and RPE values compared with the positively split trial. These findings indicated that following the evenly split trial the subjects might have had a greater potential to swim faster over a further 25 m, had they been required to do so. Less random error in pacing was also evident in the evenly split trial suggesting that the FT was more easily achieved and that pacing strategies requiring a change in pace might be difficult to adjust to during high intensity efforts. For these reasons it is recommended that coaches experiment with an evenly paced race strategy to determine if it offers performance improvements compared with the positively split pacing strategy currently being employed during competitive breaststroke swimming.

9.2 A model of the kinematic and metabolic characteristics of high intensity breaststroke swimming

9.2.1 Introduction to the model

The five studies have reported data about the kinematic and metabolic characteristics of breaststroke swimming across a range of speeds and changes in pace. From this data a model of a maximal, even paced 200 m breaststroke swim has been outlined in which the kinematic and metabolic variables are integrated (Fig 9.1). The model describes how kinematic variables initially dictate the swimming pace and metabolic response, however this situation changes in the latter stages of the swim as the metabolic response begins to increasingly dictate the kinematic response and subsequently the swimming pace.

Step 1 of the model is critical with regard to how the final 50 m of a 200 m race is swum. For example if a swimmer slightly over exaggerates Step 1 (ie. if the initial SR is too high) this may lead to an earlier onset of muscular fatigue resulting in an increase in TT along with an additional 1-2 % increase in SC and a reduction in the SV (Fig 9.1 Step 11b). What follows is a brief discussion of the effect of manipulating Step 1 of the model as could occur in competitive situations.

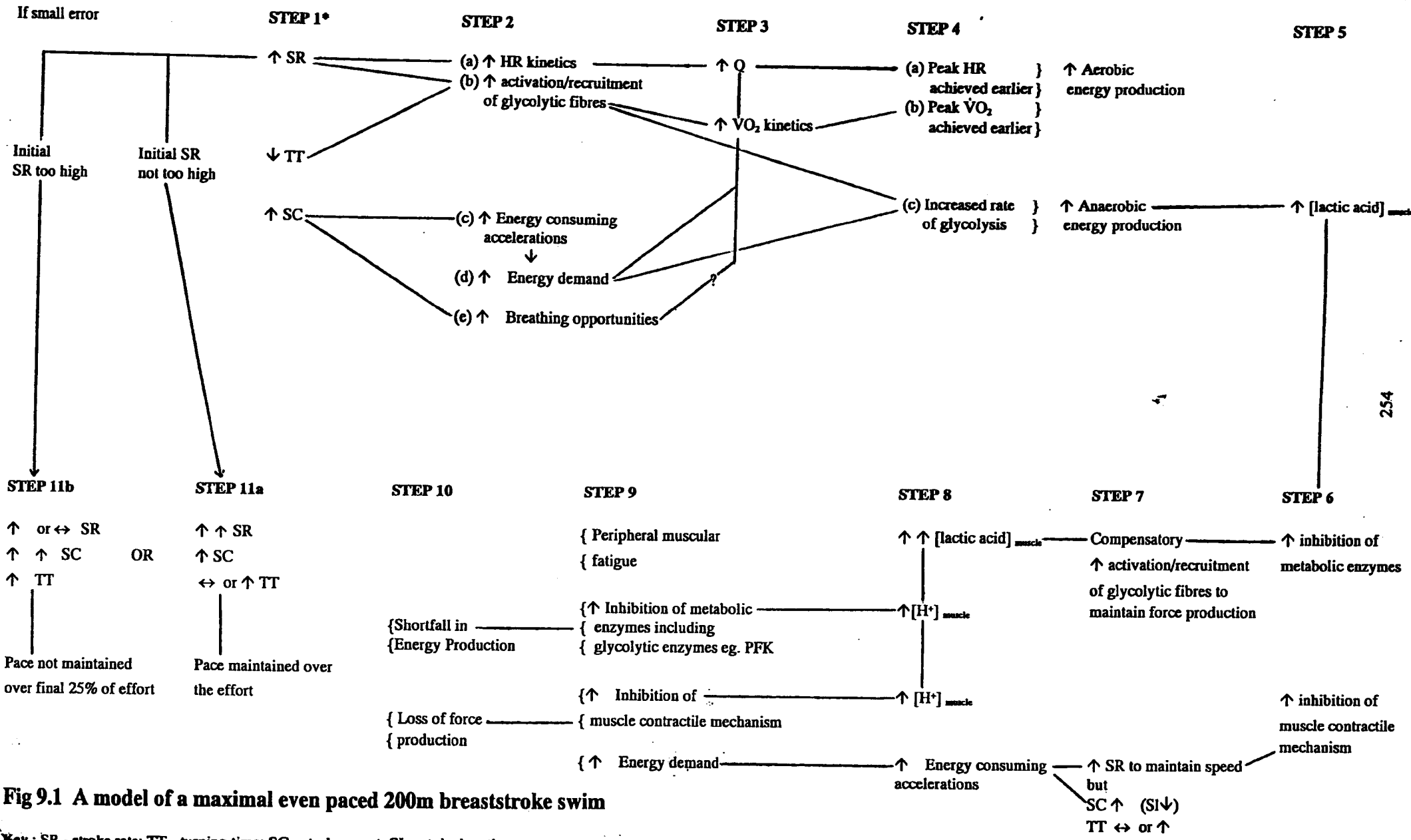


Fig 9.1 A model of a maximal even paced 200m breaststroke swim

Key : SR - stroke rate; TT - turning time; SC - stroke count; SL - stoke length,
Step 1 shows how SR, SC and TT are manipulated compared with a near maximal effort

9.2.2 The effect of positive pacing on the model

If a swimmer were to attempt to swim at a speed two percent greater than the model (ie. a maximal, even paced 200 m swim) it would result in Step 1 of the model becoming exaggerated, although data from Chapters 7 and 8 would indicate that Steps 2a, 3, 4a and 4b might not be. Consequently Step 4c would have to be increased, to meet the elevated energy demand, which would exaggerate Steps 6-10 causing a fall in speed, despite increased buffering, over the final 50 m. It is unclear whether slightly positively pacing a 200 m effort in this way would elicit a better performance than an even paced effort as the studies in this thesis did not investigate this. However some authors have suggested that in middle distance events or in events lasting longer than 80-100 s performance may be optimised through even pacing (Foster *et al.*, 1993a; Van Schenau *et al.*, 1994).

A concern of positively paced swims, at least for the scientist, is that the relationship within kinematic and temporal variables can deteriorate, making the model less predictable. For example relationships within TT s were observed to deteriorate in the positively split trial of Chapter 8, possibly due to technical problems caused by the additional lactacidosis. A further factor to consider is that positive pacing increases the perception of effort which may affect the commitment of the swimmers toward their final turns.

During races a 6-7 % decrease in mid-pool SV occurs, with the majority of this change happening in the first 50 m. Such a strategy would further exaggerate Step 1 and Steps 5 to 10 of the model. As a consequence, the initial mid-pool SV would be greater than that of the model but would then decline and eventually reach a lower value, due to the more rapid onset of metabolic acidosis. The decline in mid-pool SV is also likely to be proportionally greater in male 200 m races compared to female 200 m races as male breaststroke swimmers experience a greater lactacidosis (WASA Sports Science Support Programme 1994-2000). This greater acidosis may explain why relationships between FT, mid-pool SV and TT deteriorate markedly in the final lengths of men's 200 m races but not in women's 200 m races (Chapter 4).

9.2.3 The effect of a reduction in pace or of negative pacing

If a swimmer were to attempt to swim 200 m at a speed two percent less than the model, as might occur during a heat swim, the SR and SC values in Step 1 would decrease while the TT value would increase, leading to a suppression of Steps 2, 3 and 4a. However findings from Study 4 would suggest that Steps 4b and 5 may be no different in magnitude to those of the model, since the post-exercise peak blood lactate values from the 98 % 200_{TT} and 100 % 200_{TT} trials were not found to be significantly different. This result has implications as to whether holding back during heat swims has any physiological benefit.

This unexpectedly high blood lactate production might have been due to the aerobic energy contribution being compromised by a reduced number of breathing oppurtunties or by the slowing of the heart rate reducing the cardiac output. Alternatively technical changes made in the stroke cycle might have caused a greater activation of fast glycolytic fibres. For example the timing of the stroke or the swimmer's body position might have been adversely affected and created a requirement for a greater force production. However, despite the unexpectedly high lactacidosis, the lower overall energy demand of swimming at this speed, due to fewer stroke cycles having to be completed, will enable the swimming speed to be maintained.

Based on the findings in Study 5 adopting a negative pacing strategy for a 200 m swim would initially result in the same alterations to the model as just described. However at the halfway stage the swimmer would have to change pace and a greater pacing error becomes evident which might indicate that breaststroke swimmers experience technical difficulties when increasing pace. A further concern with a negative pacing strategy is that the heart rate response may be reduced although it is not known whether this would have any effect on the $\dot{V}O_2$ fast component, as no significant difference was found between the post-exercise peak $\dot{V}O_2$ measurements of the negatively, evenly and positively split trials of Study 5. However there was a tendency for a slightly greater lactate production following

the negatively split trial compared with the evenly split trial which might indicate that an additional anerobic energy contribution had been required. Alternatively, a greater lactacidosis might have resulted from the significant increase in the SR and decrease in the TT s in the final stages of the negatively split trial, at a time when there would have been fatigue developing (~Step 7 of the model).

9.3 Conclusions

The aim of this thesis was to provide an examination of the kinematic, temporal and metabolic characteristics of breaststroke swimming. Firstly, an evaluation of kinematic and temporal variables during 100 m and 200 m races was undertaken (Objective 1). The findings showed that the ability of swimmers to achieve and maintain the highest possible SV should be the primary concern of coaches, although considerable emphasis should also be placed on improving the starts, turns and end times of swimmers as elite swimmers were observed to perform better in each of these elements. The relative importance of these variables with respect to FT (Objective 2) was found to be dependent upon the swimming event and gender of the swimmer. Consequently the emphasis of the training needs to take this into account. Swimmers were also found to exhibit unique SR-SL ratios indicating that coaches need to identify the most effective combination on a purely individual basis.

In order to examine the metabolic characteristics of breaststroke swimming experimental trials were required. However, it was necessary to establish whether precise and reliable pacing could be achieved and whether the associated kinematic and metabolic responses were reproducible (Objective 3). Across a range of speeds the Aquapacer™ pacing system was observed to produce precise pacing until fatigue prevented swimmers maintaining pace. Reliable pacing was also

established at near maximal swimming speeds across repeated trials and the associated kinematic and metabolic responses were found to demonstrate acceptable agreement, except for the post-exercise blood lactate response following the moderate intensity trial.

Previously there had been little, if any, quantitative data reported about how kinematic and metabolic responses change during breaststroke performances differing by only a few percent, such as often occurrs between the heat and final of a competition. Subsequently subtle manipulations in pace during high intensity 200 m trials were investigated with regard to their effect on kinematic, temporal and metabolic responses (Objective 4). It was found that breaststroke swimmers typically increase SR and decrease TT s in order to increase pace. Stroke rate was also observed to be increased in the later stages of trials to maintain swimming speed when SL was decreasing due to fatigue. At high swimming speeds a coincidental increase in SC occurrs which increases the energy cost of the swim because of the resulting increase in the number of energy consuming accelerations. At near maximal speeds this additional energy cost was met by the anaerobic energy contribution, as the aerobic energy contribution was already maximised. Therefore it was suggested that breaststroke swimmers require well developed anaerobic and buffering capacities. However a high aerobic power is also important to maximise the aerobic energy contribution. This study also confirmed that the deterioration in SV and turning speed observed in races was likely to be

due to metabolic fatigue, as when a greater deterioration in mid-pool SV and turning speed was observed their was a coincidental increase in the post-exercise peak blood lactate response. Also a comparison of evenly split trials paced at, and just below the maximal 200 m speed of subjects suggested that there does not seem to be any metabolic benefit in swimming heats slower than finals.

The reliance on anaerobic metabolism during maximal breaststroke swimming means that the initial pace of a race is critical. The current trend in racing is to aggressively positively split the effort which results in fatigue being evident in the early stages of races. In the men's 200 m event this results in changes within mid-pool SV and TT s becoming unpredictable. Therefore a comparison of the effect of even, positive and negative pacing on kinematic, temporal and metabolic responses was undertaken (Objective 5). Even pacing was found to reduce the anaerobic energy contribution and attenuate increases in TT s and the SR-SL ratio compared with positive pacing over 175 m trials, however it was not investigated if even pacing would elicit a shorter FT.

The achievement of the objectives in this thesis has provided an examination of the kinematic and metabolic characteristics of the breaststroke, and led to the development of an integrative model of 200 m breaststroke swimming. The model provides an overview about how breaststroke swimmers achieve a change of pace and the implications for kinematics and metabolism.

9.4 Recommendations for further research

This thesis examined pre-requisites for performance during races and maximal trials. In these studies data was gathered from single performances. It would be instructive for a kinematic analysis to be undertaken of repeated competition performances in order to determine which variables change and by how much. This would further inform the coach about the emphasis to place in training on the different elements of a swimmer's performance.

The ability to predict performance is of great concern to coaches and swimmers. It was suggested in this thesis that a race specific test might be developed based on the swimmer completing a fixed distance at a fixed SR, as changes in the SL, TT s and FT with each re-test would indicate the potential or not for an improved competition performance. It was recommended that such a test would be best undertaken in a 50 m pool so that the swimmers had time to adopt the necessary SR following turns. The validity and reliablility of such a test needs to be researched. Many national swimmers will also regularly undertake a discontinuous incremental exercise test eliciting graphs of swimming speed plotted against blood lactate, heart rate and $\dot{V}O_2$ measurements (and sometimes kinematic data) in order to monitor changes in fitness. To date no studies have reported the relationship between sub-maximal measurements and swimming performance for the breaststroke, neither has it been established if changes within them coincide with changes in swimming

performance. These questions need to be investigated along with a comparison of the different protocols being used to clarify their purpose to sports scientists.

The SR adopted by a competitive breaststroke swimmer has been shown to be crucial particularly in the early stages of maximal 200 m swims. Research needs to conducted to determine if swimmers are able to self-select the most effective SR for them. If they cannot then a protocol could be developed for determining the ideal SR for a swimmer which could also be used to monitor changes in stroke kinematics over the course of a season. Work also needs to be undertaken to determine if the ideal SR can be entrained and reproduced in competition.

There was evidence in Studies 4 and 5 that even pacing attenuates metabolic acidosis and the RPE of 200 m breaststroke swimmers, however it was not established if even pacing would provide a performance benefit. Further studies are required to determine this. The lactacidosis evident from maximal 200 m breaststroke swimming would also suggest that research into the effect of buffering agents on performance is needed.

Finally, this thesis did not distinguish between flat and undulating breaststroke swimming styles and hence research needs to be undertaken to determine if one style is actually more effective and if so why. Such work would benefit from measurements of $\dot{V}O_2$ so that comparisons of swimming economy can be made.

Ideally gas exchange measurements would need to be taken during unimpeded free swimming, which unfortunately was not possible for this thesis.

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Appendix I

University of Wales Institute, Cardiff

Exercise Physiology and Kinanthropometry Laboratory

Pre -test Questionnaire

STRICTLY CONFIDENTIAL
Please answer questions truthfully and completely. The sole purpose of this questionnaire is to ensure that you are in a fit and healthy state to complete the exercise test.

Name:..... D.O.B.:.....

1. Have you had to consult your doctor within the last 6 months? yes/no*
   If yes, please give relevant details to test supervisor.

2. Are you presently taking any form of medication? yes/no*
   If yes, please give details to test supervisor.

3. Do you suffer, or have you ever suffered, from

Asthma?	yes/no*
Diabetes?	yes/no*
Bronchitis?	yes/no*
Epilepsy?	yes/no*
High blood pressure?	yes/no*

4. Do you suffer, or have you ever suffered from, any form of heart complaint?
   yes/no*

5. Is there a history of heart disease in your family? yes/no*

6. Do you currently have any form of muscle or joint injury? yes/no*

7. Have you had any cause to suspend your normal training in the last two weeks? yes/no*

8. Is there anything to your knowledge that may prevent you from successfully completing the tests that have been outlined to you? yes/no*

I certify that I have answered these questions truthfully to the best of my knowledge.

Participant's signature:.....

Date:.....

* please delete as appropriate.

University of Wales Institute, Cardiff

Exercise Physiology and Kinanthropometry Laboratory

Informed Consent Form

Please complete all the details below. This information is required solely for laboratory records.

Name:..... D.O.B.:.....

Address:.....

.....

Telephone:.....

Please read the following statements carefully. Please sign only when you agree with the statements and when you have had any relevant questions answered.

The full details of the tests have been explained to me. I am clear about what will be involved and I am aware of the purpose of the tests, the potential benefits and the potential risks.

I know that I am not obliged to complete the tests. I am free to stop the test at any point and for any reason.

The test results are confidential and will only be communicated to others such as my coach if agreed in advance.

I have no injury or illness that will affect my ability to successfully complete the tests.

Signature of Participant:.....

Date:.....

Signature of Tester:.....

DONATION

Information given will be treated with strictest confidentiality

All questions to be answered by 'ticking' appropriate box

		Yes	No	Not Known
1.	Are you receiving any medicines, dental treatment, have had recent illness or attending hospital outpatients?			
2.	Have you had ears pierced, acupuncture or have been tattooed in the last six months?			
3.	Have you ever been advised by a doctor not to give blood?			
4.	Are you or have you ever suffered from any of the following?			
	Allergy (hay fever, asthma etc)			
	Anaemia or other blood disorders			
	Brucellosis			
	Cancer			
	Diabetes			
	Epilepsy (fits)			
	Glandular fever (in last 2 years)			
	Heart disease			
	Hepatitis (jaundice) or been in contact with a case in last 6 months			
	High blood pressure (except during pregnancy)			
	Kidney disease			
	Peptic ulcers			
	Stroke			
	Thyroid disease (goitre etc)			
	Tropical disease especially malaria			
	Venereal disease			
5.	Is your lifestyle likely to place you at increase risk of HIV infection (AIDS)?			

DECLARATIONS

I have had explained to me, and fully understand, the reasons for donating my blood.

Signed: _____

I have not answered 'yes' to any of the questions listed and to the best of my knowledge am fully eligible to donate blood and do so of my own free will.

Signed: _____ Dated: _____

Signature of Phlebotomist: _____ Dated: _____

Appendix II

Published scientific papers

Journal of Sports Sciences, 2000, **18**, 421–431

An analysis of selected kinematic variables in national and elite male and female 100-m and 200-m breaststroke swimmers

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The kinematic analysis of competition breaststroke swimming has tended to focus on the mean values of swimming speed, stroke rate and stroke length; values in individual lengths, as well as the start, turns and finish, have largely been ignored. This study includes all such variables and aims to improve the coach's holistic understanding of breaststroke racing by determining the relationships and differences between and within these selected kinematic variables. We also compare 100-m events with 200-m events to determine if there are characteristic differences between them. Competitive breaststroke swimming performances in 100-m events (males: $n = 159$, finishing time = 65.05 ± 2.62 s; females: $n = 158$, finishing time = 74.04 ± 3.66 s) and 200-m events (males: $n = 159$, finishing time = 141.47 ± 6.15 s; females: $n = 158$, finishing time = 158.66 ± 7.87 s) were collected and analysed from 12 world, international and national championships. The better 100-m and 200-m breaststroke swimmers were found to demonstrate greater competency in the kinematic variables measured, except stroke kinematics, which were unique to each individual. These findings suggest that coaches should place emphasis on all of the kinematic components in training and that they should attempt to identify the stroke rate to stroke length ratio most appropriate for the individual. Finally, characteristic differences do exist between the 100-m and 200-m events, which has implications for how swimmers might train for each event.

Keywords: mid-pool speed, pacing, performance, starts, turns.

Introduction

Since the original work of East (1970), few studies have investigated the kinematic variables that influence the race performance of 100-m and 200-m breaststroke swimmers. Such studies have concentrated on the relationship between swimming speed, stroke rate and stroke length. Early investigations calculated these variables from hand timings made as the event proceeded (Craig and Pendergast, 1979; Craig *et al.*, 1985). Unfortunately, the measurement of stroke length was overestimated because it was calculated from the assumption that stroke length = swimming speed/stroke rate, where the initial calculation of swimming speed was based on event distance divided by finishing time. This meant that the calculation did not account for the dive start, or any variation in mid-pool swimming speed and turning times at the end of each length. Early studies have also been criticized for using small numbers of swimmers of differing abilities, who were analysed in training rather than in competition (Kennedy *et al.*, 1990).

More recent studies have used sophisticated video playback, digitizing and computer analysis techniques to measure stroke rate, stroke length and mid-pool swimming speed (Kennedy *et al.*, 1990; Chengular and Brown, 1992); however, these studies did not consider the *non-swimming* elements of the race. These elements comprise the time taken to reach a set distance from the start of the race and the time taken to travel a set distance into the turn at the end of each length of the pool and out to the same point-turning time (Hay, 1988; Wakayoshi *et al.*, 1992). More recently, the time taken to complete the final 5 m of the race, or end time, has also been reported (Thompson and Haljand, 1997).

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Table 2. Interrelationships between finishing time, start time, swimming speed, turning time and end time for male and female 100-m breaststroke swimmers

		Swimming speed ($m \cdot s^{-1}$)[a]						
	Start time (s)	Mean	Length 1a	Length 1b	Length 2a	Length 2b	Turning time, 50 m (s)	End time (s)
Finishing time (s)								
males	0.87**	−0.97**	−0.85**	−0.91**	−0.90**	−0.91**	0.84**	0.76**
females	0.89**	−0.92**	−0.90**	−0.94**	−0.95**	−0.92**	0.92**	0.82**
Start time (s)								
males		−0.80**	−0.71**				0.78**	0.60**
females		−0.85**	−0.82**				0.87**	0.70**
Mean swimming speed ($m \cdot s^{-1}$)								
males			0.90**	0.92**	0.92**	0.90**	−0.74**	−0.67**
females			0.93**	0.95**	0.95**	0.92**	−0.87**	−0.78**
Turning time, 50 m (s)								
males				−0.76**	−0.60**			0.62**
females				−0.86**	−0.82**			0.71**
End time (s)								
males		−0.67**				−0.75**		
females		−0.78**				−0.76**		

[a] Length 1a = 15 m to 25 m in length 1, length 1b = 25 m to 42.5 m in length 1.
* $P < 0.05$, ** $P < 0.01$.

had travelled 4.5 m in the time recorded. Knowing the head's speed over 4.5 m allowed the time it would take to travel 5 m to be determined. Turning time was calculated by measuring the time taken for the swimmer's head to touch a digital line 7.5 m from the end wall and to return to the same point. A mean turning time was calculated in the 200-m event from turns made at 50, 100 and 150 m.

Measurement of mid-pool swimming speed, stroke rate and stroke length – 'swimming' variables

Stroke rate was calculated from the number of frames required to complete a stroke cycle, once the swimmer's head reached the 25-m digital line. Mid-pool swimming speed was calculated from the time taken for the swimmer's head to travel from the 15-m line to the 25-m line (100-m event only) and from the 25-m line to the 42.5-m line. Stroke length was then calculated using the formula: stroke length (m) = swimming speed ($m \cdot s^{-1}$) divided by stroke rate (cycles $\cdot s^{-1}$). Mean values for swimming speed, stroke rate and stroke length were calculated from measurements made during each length of the race.

Statistical analyses

Means, standard deviations and values for skewness and kurtosis were calculated for the data; details can be found in Table 1. Correlation coefficients were determined among selected variables with significance set at 0.05. Dependent *t*-tests (for the 100-m data) and one-way analyses of variance (for the 200-m data) were used to compare mean differences within mid-pool swimming speed, turning time, stroke rate and stroke length as the 100-m and 200-m races progressed. A *post-hoc* test (Tukey's HSD test) was incorporated to identify where differences occurred following a significant analysis of variance ($P < 0.05$). Independent *t*-tests were used to compare selected variables at comparable distances during the 100-m and 200-m races (mid-pool swimming speed, stroke rate, stroke length, turning time and end time). The effect size of mean differences was assessed using omega-squared calculations in an attempt to account for unexplained variance (Vincent, 1995).

Results

Mid-pool swimming speed explained most of the variation in finishing time in both the 100-m and 200-m events (Tables 2 and 3). Measurements of mid-pool swimming speed taken at four equidistant stages during the 100-m and 200-m races also resulted in very high correlations with finishing time ($r = -0.85$ to -0.99), except in the men's 200-m, where only moderate correlations were observed over the last two lengths ($r = -0.70$ and -0.67 respectively).

PAGE NUMBERING AS ORIGINAL

Table 4. Comparisons of repeated kinematic measurements for male and female 100-m and 200-m breaststroke swimmers

	100 m		200 m	
	Males	Females	Males	Females
Swimming speed ($m \cdot s^{-1}$)				
Length 1	1.49 ± 0.05^a	1.33 ± 0.07^a	1.41 ± 0.07^a	1.27 ± 0.07^a
Length 2	1.40 ± 0.06	1.24 ± 0.07	1.33 ± 0.07^b	1.20 ± 0.07^a
Length 3	N.A.	N.A.	1.32 ± 0.12^c	1.19 ± 0.07^a
Length 4	N.A.	N.A.	1.31 ± 0.12^a	1.18 ± 0.06^a
Statistic	$t = 27.99$	$t = 26.43$	$F = 124.1$	$F = 573.4$
Omega^2	71%	69%	41%	77%
Stroke rate ($cycles \cdot min^{-1}$)				
Length 1	49.2 ± 5.4^a	49.5 ± 5.8^a	38.6 ± 4.2^a	40.8 ± 5.2^a
Length 2	51.0 ± 5.2	49.7 ± 5.7	37.1 ± 4.5^b	38.8 ± 5.3^b
Length 3	N.A.	N.A.	38.8 ± 5.4^a	39.6 ± 5.0^c
Length 4	N.A.	N.A.	43.0 ± 5.9^a	43.4 ± 5.7^a
Statistic	$t = -4.62$	$t = -0.61$	$F = 91.80$	$F = 73.42$
Omega^2	6%	N.A.	35%	30%
Stroke length (m)				
Length 1	1.85 ± 0.30^a	1.63 ± 0.19^a	2.22 ± 0.25^a	1.89 ± 0.25^a
Length 2	1.67 ± 0.17	1.52 ± 0.18	2.18 ± 0.27^a	1.89 ± 0.25^a
Length 3	N.A.	N.A.	2.04 ± 0.29^a	1.82 ± 0.24^a
Length 4	N.A.	N.A.	1.84 ± 0.25^a	1.66 ± 0.21^a
Statistic	$t = 9.42$	$t = 11.91$	$F = 199.7$	$F = 112.7$
Omega^2	22%	31%	54%	39%
Turning times (s)				
50 m			9.83 ± 0.59^a	11.28 ± 0.65^a
100 m			10.17 ± 1.07^b	11.69 ± 0.78^a
150 m			10.35 ± 1.00^c	11.82 ± 0.74^a
Statistic			$F = 306.0$	$F = 306.0$
Omega^2			17%	66%

[a] Significant at $P < 0.01$ from all conditions.
[b] Significant at $P < 0.01$ from all conditions except at 150 m, where significant at $P < 0.05$.
[c] Significant at $P < 0.01$ from all conditions except at 100 m, where significant at $P < 0.05$.
N.A. = not applicable.

Mid-pool swimming speed decreased significantly over each consecutive 50 m (Table 4), demonstrating a positive split-pacing strategy during races. The effect size for these data is borne out by the large omega-squared values computed (Vincent, 1995).

In the 200-m event, the mean mid-pool swimming speeds for lengths 2, 3 and 4, although decreasing significantly, demonstrated a much smaller decrease (typically 0.1 $m \cdot s^{-1}$ per length) than was observed after the first length. It is important to note that the standard deviation for mid-pool swimming speed for lengths 3 and 4 in the men's 200 m is almost twice that observed for lengths 1 and 2, or for any of the lengths in the women's event. The increase in the dispersion of the mid-pool swimming speed during lengths 3 and 4 also coincided with worsening relationships between these variables and finishing time.

Mean stroke rate was only significantly correlated with finishing time in the women's 200-m event; however, little of the variance in finishing time was explained by the variable ($r^2 < 5\%$) (Tables 5 and 6). A possible explanation was provided by the relationships between stroke rate measurements made on consecutive and non-consecutive lengths, which demonstrated only poor to moderate relationships.

Relationships between stroke length and finishing time were also poor ($r < -0.4$), although significant. Therefore, a faster finishing time was related to a longer stroke length, although most of the variation in finishing time was not explained by stroke length.

Table 6. Interrelationships between finishing time, swimming speed, stroke rate and stroke length for male and female 200-m breaststroke swimmers

	Stroke rate (cycles · min^{-1})					Stroke length (m)					Swimming speed (m · s^{-1})				
	Mean	Length 1	Length 2	Length 3	Length 4	Mean	Length 1	Length 2	Length 3	Length 4	Mean	Length 1	Length 2	Length 3	Length 4
Finishing time (s)															
males	−0.08	−0.02	−0.02	−0.14	−0.26**	−0.38**	−0.36**	−0.35**	−0.42**	−0.30**	−0.99**	−0.91**	−0.94**	−0.70**	−0.68**
females	−0.20*	−0.05	−0.08	−0.12	−0.20*	−0.36**	−0.32**	−0.34**	−0.34**	−0.23*	−0.99**	−0.92**	−0.96**	−0.98**	−0.94**
Stroke rate, mean (cycles · min^{-1})															
males		0.76**	0.89**	0.76**	0.63**	−0.87**					0.11				
females		0.83**	0.92**	0.91**	0.82**	−0.86**					0.15				
Stroke rate, length 1 (cycles · min^{-1})															
males			0.72**	0.46**	0.22*		−0.68**					0.20*			
females			0.75**	0.64**	0.51**		−0.89**					0.15			
Stroke rate, length 2 (cycles · min^{-1})															
males				0.68**	0.41**			−0.91**					0.14		
females				0.89**	0.63**			−0.89**					0.18		
Stroke rate, length 3 (cycles · min^{-1})															
males					0.74**				−0.32**					0.51**	
females					0.70**				−0.86**					0.12	
Stroke rate, length 4 (cycles · min^{-1})															
males										−0.30**					0.57**
females										−0.88**					0.25*
Stroke length, mean (m)															
males							0.84**	0.93**	0.79**	0.63**	0.36**				
females							0.84**	0.92**	0.89**	0.83**	0.34**				
Stroke length, length 1 (m)															
males								0.78**	0.62**	0.38**		0.21*			
females								0.73**	0.62**	0.54**		0.26**			
Stroke length, length 2 (m)															
males									0.72**	0.47**			0.23*		
females									0.79**	0.68**			0.25*		
Stroke length, length 3 (m)															
males										0.77**				0.61**	
females										0.70**				0.37**	
Stroke length, length 4 (m)															
males															0.57**
females															0.25*

*$P < 0.05$, **$P < 0.01$.

may be a reflection of the greater variation in swimming speed observed in this event.

Finally, a comparison between the 100-m and 200-m events was made for kinematic variables over corresponding distances (Table 8). Mean values for race speed, mid-pool swimming speed and stroke rate were significantly higher ($P < 0.01$) for the 100 m than the 200 m, while start time, stroke length, turning time and end time were significantly lower ($P < 0.01$). The greater speed in the 100 m (3–5%) was achieved by adopting a higher stroke rate coupled with a shorter stroke length.

Discussion

In 1992, Wakayoshi *et al.* suggested that swimming speed might be significantly higher in better performing national and elite swimmers. Our findings are largely in agreement with those of Wakayoshi *et al.* Mean mid-pool swimming speed and individual mid-pool swimming speed were generally strongly negatively related to finishing time in both the 100-m and 200-m breaststroke events. However, in the men's 200 m, only moderate correlations were observed during the last two lengths, suggesting that, unlike the other events, the ability to predict finishing time from mid-pool swimming speed diminishes in the last 200 m of the men's event.

Maglischo (1993) suggested that successful breaststroke swimmers adopt an even paced race, when the dive start is accounted for (by subtracting 2–3 s from the split-time at half distance). However, we observed that mid-pool swimming speed decreases significantly over each consecutive 50 m of a race, with the first length being swum 6–7% faster than the final length irrespective of race distance or sex. This demonstrates that national and elite breaststroke swimmers tend to adopt a positively split pacing strategy for their races.

Whether this practice is a deliberate race strategy or the product of the swimmer's excitement is unclear, but it needs to be clarified, as the end result is an ever diminishing swimming speed. For example, in the 200 m, swimmers typically demonstrated a slight drop in swimming speed over the last three lengths, which is probably related to the onset of leg fatigue, owing to the heavy reliance on leg propulsion in the breaststroke (Maglischo, 1993) resulting in metabolic acidosis (Thompson, 1998). Our findings suggest that the adoption of an even paced race strategy needs to be evaluated to establish whether fatigue can be attenuated slightly, resulting in a higher mean swimming speed during breaststroke races.

Mean stroke rate and stroke length were poorly correlated with finishing time, in line with the results of previous studies (Kennedy *et al.*, 1990; Chengalur and Brown, 1992; Wakayoshi *et al.*, 1992), and were only poor to moderately correlated with mid-pool swimming speed. A possible explanation in the 200 m was that the relationships within stroke rate and stroke length tended to worsen over the race distance, suggesting that swimmers do not maintain a given stroke rate to stroke length ratio during a race; neither do better swimmers adopt a lower stroke rate and a greater stroke length than less able swimmers (Wakayoshi *et al.*, 1992).

Maglischo (1993) advised that breaststroke swimmers maintain a constant stroke rate and hence a constant energy expenditure throughout a race; however, it would appear that competitive swimmers do not generally conform to this practice. Indeed, the positive pacing we have highlighted might not allow the swimmer to follow Maglischo's advice. Rather, individual swimmers generally adopt a unique stroke rate to stroke length ratio that changes over the race distance, presumably as a consequence of fatigue resulting in the positively split race pattern.

The effect of adopting a disproportionately high swimming speed during the first length commonly resulted in a fall in stroke length on each subsequent length. This could be indicative of fatigue leading to a poor body alignment and increased drag. If this were the case, then the subsequent increase in energy expenditure would reduce swimming economy still further. Indeed, in the 100-m events, both men and women suffered a reduction in swimming speed during the second length because they were unable to increase stroke rate sufficiently to compensate for the loss in stroke length. Interestingly, in the 200-m events, stroke rate was reduced during the second length, perhaps in an effort to conserve energy; even so, stroke length fell in the men's event (although it was maintained in the women's event). Thereafter, however, stroke rate increased on each subsequent length in an unsuccessful attempt to compensate for a perpetually decreasing stroke length.

The tactic of increasing stroke rate to compensate for a decreasing stroke length is in line with previous work (Kennedy *et al.*, 1990; Chengalur and Brown, 1992). However, it must be noted that, during the last two lengths of the men's race, the relationship between stroke rate and stroke length fell dramatically ($r = -0.3$ to -0.32), although it remained significant ($P < 0.01$). This increase in stroke variation coincided with a marked increase in the standard deviation of mid-pool swimming speed, which may in part explain the deterioration in its relationship with finishing time.

The 'non-swimming' kinematic elements constitute a large proportion of both 100-m (35%) and 200-m (32.5%) races, with the starting and turning speeds determining that mean race speed is greater than mean mid-pool swimming speed (Table 7); however, little information is available on these variables. Thompson

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type (small sample size, non-competitive, heterogeneous sample). To our knowledge, we have reported on more variables in this study than any previously published.

In summary, the main correlate of finishing time was mid-pool swimming speed, with turning time and start time being moderately related to finishing time, suggesting that competitive breaststroke swimmers demonstrate an holistic approach to their race performance. Therefore, coaches should perhaps adopt a similar philosophy when designing training programmes. Stroke rate and stroke length were not related to finishing time but were negatively related to each other. The male 200-m swimmers were anomalous in that they demonstrated a much greater variation in mid-pool swimming speed and turning time and poor correlations with finishing time in the latter stages of the race. Mid-pool swimming speed, turning time and stroke length were generally found to deteriorate as the races progressed, which was attributed to the swimmers adopting a positively split race strategy. Therefore, it might be instructive for coaches and sport scientists to consider whether a more even paced race strategy might result in less deterioration in the kinematic variables measured here and, subsequently, an improvement in performance. Finally, 100-m swimmers exhibited greater mid-pool swimming speeds and stroke rates coupled with shorter start times, turning times and stroke lengths than 200-m swimmers. This suggests that event-specific preparation is appropriate.

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