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Fluid and Electrolyte Balance during Indoor Tennis Match Play

by

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Declaration

All rules and regulations have been followed in the course of the project, and it consists of all my own work. Acknowledgements have been made where help has been given.

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Abstract

Fluid intake, electrolyte balance, and effort intensity during one best of three set indoor singles tennis match (17 ± 2°C, 42 ± 9% humidity) was measured in 16 male University tennis players. Sweat samples were collected through application of an absorbent sweat patch to the forearm, calf, thigh and back of each player. Effort intensity was measured through comparisons of on-court heart rates to data obtained from a maximal treadmill test.

The mean sweat loss was 1219 ± 417 ml, mean fluid intake was 1087 ± 625 ml (players replaced on average 89% of fluid lost), mean whole body sweat rate was 0.72 ± 0.26 l/h and no significant body mass loss was observed from pre to post match. However, a large inter-individual variability existed (range 0.43 - 1.28 l/h). 15 out of 16 players chose to consume water during their match; and these fluid intake choices were sufficient to on the whole maintain plasma sodium levels. Two players provided pre-match urine samples above 900 mOsmol/kg while another four provided samples approaching this level, indicating some players were hypohydrated prior to match play. The mean sweat sodium concentration was 41 ± 15 mmol/l suggesting lower heat acclimation statuses than in players competing at warmer environments, and total sodium losses during match play were 1.12 ± 0.45 g (range 0.46 – 1.93 g). Again, large individual variations existed. On average, dietary and on-court electrolyte intake exceeded electrolyte loss during match play by a considerable margin, but in some player's there was not a great difference. Muscle cramping could occur if players fail to adequately replace both fluid and electrolyte losses that occur during match play, even in a comfortable indoor environment. Finally, indoor match play largely consisted of moderate intensity exercise, below ventilatory threshold, with a smaller high intensity contribution.

This study showed that in cool ambient conditions, sweat rates reached 1.28 l/h, and players ingested sufficient fluid to replace 89 ± 47% of sweat losses, suggesting that contrary to footballers, runners, and in some other sports, fluid replacement is easier to achieve in tennis due to the regular breaks in match play.

Chapter 1

Introduction

Professional tennis is played in a wide variety of climactic conditions across the world, and players may be faced with travelling to, and competing in, a hot climate and on to a cooler one in quick succession. One of the major challenges faced by elite players is being able to replace the fluid and electrolyte losses that they will invariably experience in a range of environments, in order to increase their chances of avoiding dehydration and muscle cramping, and perform at their highest level. Players must be able to dissipate heat effectively, even in the most challenging conditions if they are to avoid rapid dehydration, and must also ensure that they replace all fluid and electrolyte losses. This can often be very demanding since elite tennis match play has been previously shown to induce sweat rates of over 2.0 l/h (Bergeron 2003), and players must implement fluid intake strategies that attempt to fully replace these sweat losses. Because of this, it is important that players understand the effects of dehydration and electrolyte losses on performance, and are aware of their own potential fluid and electrolyte losses during match play.

The extent of sweat rates and electrolyte losses in many different sporting contexts has been comprehensively described; however research addressing these topic areas in tennis players is less widespread. Work has tended to focus on team sports such as football, basketball, and American football, and endurance sports such as long-distance and marathon running, and in a range of conditions. Bergeron and colleagues (1995a, 1995b, 1996, and 2003) have detailed the sweat rates, sweat electrolyte concentrations, and fluid intake strategies of tennis players outdoors in hot environments, including consecutive days of match play, and in players with a tendency to suffer heat cramps. However, information is lacking on sweat rates, electrolyte concentrations, and fluid balance of tennis players in cool and moderate temperatures, especially indoors where the heat loss mechanism of convective cooling is much lower. Bergeron et al. (2006) also investigated voluntary fluid intake and core temperature responses in adolescent players at around 26°C, but again this was held outdoors. Singles tennis matches can often last over two and a half hours, and although many tournaments are held in challenging environments, the majority of competitive tennis in Great Britain is played in an indoor environment, which is a valid reason for investigating these environmental conditions.

Sweat sampling techniques used by previous studies have been lacking in certain areas. Bergeron et al. (1995a) took sweat samples from the non-dominant forearm of players, but only left the collection patches on for 26.4 ± 4.1 minutes, and they were removed at a convenient changeover (mostly at the end of the first set), so concentration estimates could have been low. Indeed, Stofan et al. (2005) suggest that sweat sodium concentrations can increase by up to 20% as exercise training progresses from the first hour to the second hour. Therefore, in any study absorbent sweat patches that will stay in place for the duration of the match should be applied to remove this potential source of error. The use of enclosed patches to collect sweat samples has been the subject of much scrutiny, and it has been suggested that samples obtained by this method have overestimated actual sweat electrolyte concentrations by up to 30 to 40% (Shirreffs et al. 2006; Weschler 2008). However, it remains the only practical method of obtaining sweat samples in sporting situations without any major obstruction or hindrance to an individual's performance, and virtually all studies addressing electrolyte balance in sport have used this method.

The occurrence of dehydration during exercise has been shown to induce many physiological and thermoregulatory problems, and tennis players face a constant challenge to fully replace fluid losses during match play. With this known, players have still been observed to commence a match in a hypohydrated state (Hornery et al. 2007; Bergeron et al. 2006). Dehydration levels of 1.8 ± 0.9% body mass loss following a three set singles match have been reported by Bergeron et al. (1995) at 32°C, while others have shown slightly lower deficits, and a dehydration level of 2% has been shown to impair power production (Coyle et al. 2004), and cognitive function (Gopinanthan et al. 1998). Body fluid losses have been measured at a variety of temperatures in football (Shirreffs et al. 2006), along with American football (Fowkes Godek et al. 2005), basketball (Boatwright et al. 2003; Osterberg et al. 2005), and ice hockey (Palmer & Spriet 2008), however none have addressed tennis players in either indoor, or comfortable environmental conditions.

Sweat rates of between 1.7 l/h and 2.4 l/h have been recorded for single three set tennis matches in warm conditions (Bergeron et al. 1995a), while players with a history of heat cramps were shown to experience sweat rates of up to 3.4 l/h (Bergeron et al. 2003). These high sweat rates will bring about significant body fluid

deficits if players fail to rehydrate sufficiently, so it is a factor that players should be aware of. It is very difficult to rehydrate at a rate that matches these high sweat losses, because it is simply uncomfortable for some players to ingest the necessary amount of fluid at changeovers. As well as the considerable fluid losses that can occur during a match, sweating can lead to a large electrolyte deficit. Bergeron (2003) showed that sweat sodium concentrations of 17 male tennis players at temperatures ranging from 28.9°C to 35.6°C ranged between 23.0 mmol/l and 83 mmol/l, and these results are similar to a wide range of studies that have looked at sweat sodium concentrations in different sports and at different temperatures. The extent of sodium loss in sweat varies largely between individuals, and is linked closely to an individuals heat acclimation status; the more acclimated a player is, then the greater sweat rate they experience, and the less sodium they tend to lose in sweat (Sparling 2000). However, for all players, maintaining an electrolyte balance is vital to limit dehydration, fatigue and muscle cramping. Players must also be aware that their dietary intake of sodium may predispose them to suffering sodium depletion during matches, and should address this accordingly (Kovacs 2006).

Player's heat acclimation statuses could potentially affect the results from the present study, because athletes with a high level of heat acclimation have been shown to produce a greater sweat rate, but with a reduced sweat sodium concentration when compared to non-acclimated individuals in the same situations (Sparling 2000). The results from studies on fluid and electrolyte balance will give an indication as to how well acclimatised the players are to heat, which is obviously important when competing in warmer environments, and it would be interesting to compare these results to players who have been studied in warmer conditions.

Heat cramping or muscle cramping is believed to be the direct result of sodium depletion during exercise. Heat cramp sufferers tend to be salty sweaters (Eichner 2007), with research showing a greater magnitude of sodium loss in sweat in athletes prone to cramping versus those who were not (Stofan et al. 2005; Bergeron 2003). Bergeron (2003) suggests that the loss of fluid and sodium causes a contraction of the interstitial volume causing an increase in ionic and neurotransmitter concentrations; however the specific mechanisms behind the onset of heat cramping remain elusive,

and it is not thought that the protocol in the present study could bring about such problems, however it is always a possibility in extreme cases.

Effort intensity has been investigated using a couple of different methods. Some have simply measured heart rate responses across a match (Seliger et al. 1973; Kindermann et al. 1981; Mitchell et al. 1992), while some have calculated on-court heart rates as a percentage of maximal heart rates observed during an off-court test, such as a maximal run in a treadmill (Hornery et al. 2007; Christmass et al. 1998; Bergeron et al. 1991). These studies have observed that on-court heart rates seem to vary between 60% and 85% of a player's maximal heart rate. Seliger et al. (1973) managed to calculate the relative aerobic and anaerobic intensity contribution to match play. However, they used a complicated protocol containing only 10 minutes of actual match play: too short to allow players to reach maximal effort levels. Finally, Bernardi et al. (1998) used the direct measure of oxygen uptake using a portable metabolimeter to quantify effort intensity of three different playing styles, demonstrating that predominately baseline players work at a higher effort intensity (mean 59% $VO_{2\ MAX}$).

Study Aims

The main aims of the present study are to therefore to observe the fluid intake strategies, electrolyte losses, and match play intensities of elite tennis players in an indoor environment.

The key objectives are:

1. To assess the fluid intake strategy of players during indoor tennis match play in a moderate to cool environment;
2. To examine fluid balance and electrolyte balance over a period of indoor match play;
3. To quantify the intensity of match play in a moderate to cool indoor environment;
4. To make recommendations for future work, and to provide individuals with feedback about their fluid intake habits.

Hypothesis

It is hypothesised that the sweat rates and electrolyte balances recorded will be at the lower end of the range reported by many of the studies that have addressed this topic. This is because the study will be carried out indoors, and the environment should not be challenging enough to induce sweat rates above 1.5 l/h, and sweat sodium concentrations above 50 mmol/l, which is at the higher end of results reported by previous studies. Sodium losses throughout the best of three set match should not be high enough to threaten the daily dietary intake of sodium observed through analysis of player's dietary intake, unless a player's diet is particularly poor, or they are very poorly acclimated to heat, which would cause greater sodium loss for a given sweat rate. It is predicted that in accordance with previous studies looking at pre-exercise hydration in tennis players, some players may commence a tennis match in a poorly hydrated state, similar to observations from Hornery et al. (2007), and Bergeron et al. (2006). There should be a significant body mass loss from pre to post match, in accordance with other studies looking at fluid intake strategies in tennis, however the percentage of body mass loss should be less than those observed in warmer conditions, as the environment in this study is less challenging. Seliger et al. (1973) suggested that the energy contribution to match play was around 88% aerobic and 12% anaerobic; however Fox (1979) has suggested that the aerobic contribution is a lot lower; around 30%. These figures will vary because of the procedures used, so it is hypothesised that intensities observed in this study will be somewhere in the middle of these figures.

It is intended that the results from this study can be considered alongside those of studies addressing fluid intake and electrolyte balance in elite players competing in warmer environments, and it could help to improve player's knowledge of the electrolyte depletion that is possible during undemanding environmental conditions. Raising awareness of these factors is considered important for players to optimise their performance.

Chapter 2

Review of Literature

This review of literature aims to detail the wide range of research carried out previously that has investigated sweating responses, dehydration, fluid intake, electrolyte balance, and exercise intensity in a wide range of sporting contexts, and in doing so will focus on the following main topic areas to provide a comprehensive overview.

- Thermoregulation and the sweating response.
- Sweat rates in sporting environments.
- Dehydration, its physiological effects, and resulting effects upon performance.
- Thirst and fluid intake during sport.
- Electrolyte balance, and total electrolyte losses in sport.
- Effort intensity during match play.
- Metabolite responses during match play.

2.1 Thermoregulation and the Sweating Response

The hypothalamus is the body's temperature control centre, and keeps body temperature at 37°C, ± 2 or 3°C in all but the most extreme of conditions (Maughan 2003). During exercise, core body temperature increases, because roughly three quarters of the energy produced from metabolism during exercise is in the form of heat, with only a quarter being converted to movement (Hosey & Glazer 2004).

The main ways the body's heat loss mechanisms are activated are by either feedback from thermal receptors in the skin, or by changes in the blood temperature around the hypothalamus itself. The dissipation of heat is essential during exercise, and the body has four mechanisms of heat transfer; radiation, conduction, convection, and evaporation (Kovacs 2006; Bass & Inge 2001). Radiative heat exchange in tennis would tend to lean towards the gain of heat from solar energy, but also can occur through heat gain from the court surface. Heat loss through conduction occurs through direct contact with other particles, while heat loss through convection is dependant on the air temperature: if the air temperature is higher than body temperature there is no gradient for heat transfer. When the air is still, as is the case in indoor environments,

any heat transfer by convection is reduced to that which can be created by movement through the still air. When the air temperature is colder than skin temperature, then heat can be dissipated through radiation and convection, as well as by evaporation (Kovacs 2006), but when the air temperature exceeds body temperature these mechanisms are compromised, leaving evaporation as the most effective method of heat transfer during exercise (Dawson et al. 1985), particularly when the ambient temperature is above 20°C (Sparling 2000). According to Sparling (2000), the evaporation of sweat continues to dissipate heat from the body, even if the air temperature is higher than the core temperature. However, if the level of humidity is high, sweat vaporisation is reduced and body cooling is severely compromised. Because of this, under conditions of high-intensity exercise in hot humid conditions, evaporative cooling through sweating may become insufficient making these the most challenging conditions in which to play.

The main hormones involved in regulating the sweating response are aldosterone and vasopressin (Armstrong 2000). Exercise stimulates aldosterone, which helps to increase sodium conservation in the kidney. This helps to reduce the osmolality of sweat, thus the concentration of sodium in the sweat decreases during repeated heat exposure. This aids to conserve electrolytes. Also, exercise and/or hypohydration (reduced body fluid level) stimulates vasopressin (also called antidiuretic hormone), which increases the permeability of the kidneys to improve fluid retention (Armstrong 2000).

2.2 Sweating Rates, Dehydration & Fluid Intake

2.2.1 The Sweating Rate

The most efficient cooling mechanism is the evaporation of sweat from the skin (Hosey & Glazer 2004). The sweating rate has been shown to vary largely between individuals, and depends principally on factors such as exercise intensity, environment, heat acclimatisation, level of aerobic fitness and hydration status (Maughan et al. 2004, 2005; Bergeron et al. 1995a, 1995b, Maughan & Shirreffs 1997; Fowkes Godek et al. 2005; Kovacs 2006; Shirreffs et al. 2006; Bass & Inge

2001; Hosey & Glazer 2004). Bass & Inge (2001) stated that the sweat response is much less efficient in children than adults, not because of fewer sweat glands, but because each gland produces less sweat.

2.2.2 Sweat Rates in Sporting Environments

Studies have recorded the sweat rates of athletes in a wide range of sports, and have unsurprisingly recorded a wide range of results. Maughan et al. (2005) sampled 17 professional footballers during a 90-minute training session in a cool (5°C) environment, and recorded a mean sweat rate of 1.12 litres per hour (l/h), with a range of 0.71 to 1.77 l/h. In comparison, Shirreffs et al. (2005) sampled 26 footballers using the same methods with the same duration of training session, but in much warmer conditions (32°C) heat, and recorded a mean sweat rate of 1.46 l/h, with a range from 1.12 to 2.09 l/h. In another study by Maughan et al. (2004), they measured the same sweat collection sites during a training session of 24 premiership footballers at 24-29°C, and recorded a mean sweat rate of 1.35 ± 0.275 ml/h.

Passe et al. (2007) recorded sweat rates of 21.6 ml/kg/hr in experienced endurance runners during a 10-mile track race at 20.5 ± 0.7°C (they failed to state the mean body mass pre-exercise, thus making it impossible to calculate sweat rate in l/h). Fowkes Godek et al. (2005) studied 10 American Footballers and 5 cross-country runners in temperatures ranging from 26.1°C to 34.4°C, and calculated sweat rates of 2.15 l/h for the American Footballers and 1.56 l/h for the cross-country runners. However, no sweat collection actually took place in this study. Stofan et al. (2005) suggested that the clothing worn by American footballers creates an environment that increases fluid loss, as well as sodium loss through the skin, and Gavin (2003) has suggested that increasing the level of clothing imposes barriers to heat transfer and evaporation from the skin surface. This could also be applicable to ice hockey players, where Palmer & Spriet (2008) showed sweat rates of 1.8 ± 0.1 l/h during an intense practice session at 13.9°C.

Sweat rates in elite tennis players has been less thoroughly investigated, however Mitchell et al. (1992) recorded sweat rates of approximately 1.0 l/h in tennis players during a three-hour tennis match outdoors at a WBGT of 27°C, which appears quite

low for the warm conditions. Bergeron et al. (1995a) examined twenty Division 1 NCAA tennis players who played three matches in three days outdoors in 32°C heat and recorded sweat rates of between 1.71 l/h and 2.4 l/h. However, this study only collected sweat from the non-dominant forearm of each subject, and this may not have been representative of whole body sweat rates (Patterson et al. 2000). Also, Bergeron (1996) recorded sweat rates of 2.5 l/h for a 17-year old junior tennis player who had a history of suffering from heat cramps during matches of an extended duration, but again only the non-dominant forearm was used for sweat collection. Furthermore, all the studies looking at tennis players have been carried out outdoors in hot temperatures (between 27 and 32°C), and none have been carried out indoors, where the process of convective cooling as a method of heat loss is reduced.

Body mass losses in sporting environments obviously vary with environmental conditions, type of sport (opportunities for fluid ingestion), and the individual; however most sports observe body fluid deficits of between 1 and 3%.

Shirreffs et al. (2006) showed that during football training sessions at differing temperatures, players lost on average 1.5 ± 0.5% of their body mass throughout the 90-minute sessions. Furthermore, Bergeron et al. (1995a) showed that male college tennis players lost 1.3 ± 0.8% of their body mass during a best of three set tennis match outdoors in 32°C heat. Laursen et al. (2006) showed that 10 well-trained male athletes lost on average 2.3 ± 1.2 kg during the 2004 Ironman Western Australia event, but the body mass losses were well tolerated, because there was no evidence of thermoregulatory failure. In a different context, Schoffstall et al. (2001) stated that dehydration that results in a 1.5% body mass loss significantly decreased one-repetition maximum bench press performance, but that the effects of dehydration were overcome after a two-hour rest period and water consumption.

Boatwright et al. (2003) showed that each individual on an American college basketball team lost 2.2% of their body weight each practice, and this continued over 18 days of practice prior to their first game. However, as there was no mention of the intensity or duration of the practice sessions it is difficult to compare this to other studies. Clearly effective fluid replacement is essential across this long duration as repeated fluid loss could be detrimental to performance. Finally, Osterberg et al.

(2005) showed that 26% of players exceeded 1.5% of dehydration across the duration of a basketball game.

To conclude, Maughan & Shirreffs (1997) report that sweat rates of 1.0 to 2.0 l/h are characteristic of most forms of activity involving moderate to hard exercise, but rates of over 2.0 l/h are not uncommon during strenuous exercise or during high temperatures. Indeed, sweat rates over 2.0 l/h have been noted in American footballers, which could arise from the clothing worn. Finally, Sawka & Pandolf (1990) stated that fit, well-hydrated athletes could sweat up to 3.5 l/h during cycling or running in a hot environment. Given this range of sweat rates and the large inter individual variation it is of interest to players and coaches to examine the sweat rates during indoor tennis match play to add to the range of data available and to assess whether players can maintain a euhydrated state throughout play

Euhydration is the term used to indicate the normal daily body water content. Hyperhydration and hypohydration define conditions of increased and decreased body water content, respectively, while dehydration is defined as the process of losing body water that can occur when an individual progresses from a hyperhydrated state to euhydration, and then onto hypohydration (Greenleaf et al. 1992). However, according to Barr (1999), the term ‘dehydration’ can refer to both hypohydration, which the author classes as dehydration induced prior to exercise, and to exercise-induced dehydration, classed as dehydration that develops during exercise. It is considered important to distinguish between these two conditions (Noakes 1993; Barr 1999).

2.3 Dehydration, its physiological effects and resulting impact upon performance

Unless individuals commence exercise in a euhydrated state, and fluid which is lost during exercise is replaced, then physiologic function, thermoregulation and ultimately performance will be impaired. This has been shown extensively in different sporting contexts by Armstrong et al (1985); Barr (1999); Cheuvront et al. (2003); Walsh et al. (1994) amongst others.

2.3.1 Research on Dehydration: A Brief History

According to Noakes (1993), the building of the Hoover Dam in Nevada, USA, in the 1930's motivated some of the earliest studies of fluid losses and electrolyte balance. Indeed, Talbott et al. (1933 as cited in Noakes 1993) demonstrated the clinical features of heat cramps in workers on the building project. Other important studies in this field include Pitts et al. (1944), who analysed subjects who marched uphill on a treadmill for periods of 1 to 6 hours at 3.5mph. They measured heart rate and core temperature responses as well as oxygen utilisation under conditions of dehydration and fluid replacement, and found that heart rate and core temperature were maintained well under fluid replacement conditions, while under conditions of dehydration these variables increased steadily from the onset of exercise and exercise capacity was impaired.

The Second World War provided the basis for a series of studies that addressed dehydration (Adolph 1947 as cited in Noakes 1993). Among the important findings were that maximal sweat rates in a desert environment were about 1.7 litres per hour, subjects developed dehydration even when given unlimited access to fluids, and heart rates and rectal temperatures increased linearly with increasing dehydration.

Despite several studies indicating the effects of dehydration upon heart rate and temperature responses during exercise, it still appeared that many athletes and coaches believed that fluid restriction was beneficial to athletic performance many years ago. Noakes (1993) cited the 1957 marathon record holder, who stated, "There is no need to take any solid food at all, and every effort should be made to do without liquid, as the moment food or drink is taken, the body has to start dealing with its digestion and in so doing some discomfort will almost invariably be felt."

By the late 1960's it was discovered that athletes could lose significant amounts of body weight during exercise and experience an increase in core temperature (Pugh et al. 1967 as cited in Barr 1999), however Barr (1999) states that any detrimental effects on performance were not recognised since the winners of endurance races tended to be those experiencing the highest levels of dehydration. It was not until the

1970's that the negative effects of dehydration upon exercise performance were demonstrated (Barr 1999; Noakes 1993).

2.3.2 Effects of Dehydration on the Body

The effects of dehydration on an individual can be categorised into three main areas: thermoregulatory strain, cardiovascular strain, and effects upon mental function.

Exercise-induced dehydration has been shown to increase core temperature responses during exercise in both moderate and hot climates (Sawka et al. 2001; Noakes 1993; Sawka et al. 2007). It was suggested by Sawka et al. (2001) that this rise in core temperature occurs from a reduction in the effectiveness in the heat dissipation mechanisms during exercise, as there is no significant effect of dehydration on the metabolic rate, which could have also explained a rise in core temperature. A dehydration level of as little as 1% of body weight during exercise was associated with a significant increase in rectal temperature compared to the same exercise with normal hydration (Claremont et al. 1975).

The onset of dehydration forces the body to reduce the sweat rate and blood flow to the skin in an attempt to conserve body fluids, as well as maintaining the central blood volume and cardiac output. The maximal capacity to perfuse the skin with blood is also reduced with dehydration (Murray 1992). Furthermore, when athletes start to perform in a dehydrated state, the onset of sweating and the increase in skin blood flow begin at a higher body temperature, and both delays represent a thermoregulatory disadvantage. This supported by other studies that have looked at the effects of dehydration on the body (Noakes 1993).

Interestingly, Morgan et al. (2004) investigated the effects of dehydration upon sweat composition during prolonged exercise in the heat. They found that dehydration resulted in an increased concentration of sweat sodium compared to those in a euhydrated state, and this couldn't be put down to a difference in sweat rate since there was no significant difference in sweat rate between the dehydration and euhydration trials. The authors concluded that the elevated sweat sodium concentration caused by dehydration was "potentially related to a greater extracellular

fluid sodium concentration, plasma aldosterone, or sympathetic nervous system activity" (Morgan et al. 2004).

With hypohydration / dehydration there is a reduced rate of gastric emptying, which reduces the rate of rehydration of ingested beverages, and this can predispose athletes to feelings of bloatedness, nausea, and general gastric distress (Murray 1992; Noakes 1993). Plasma osmolality is increased (Murray 1992; Armstrong et al. 1994), because as fluid is lost, the concentration of particles left is increased, which may influence thirst mechanisms. Finally, Noakes (1993) also suggested that ratings of perceived exertion during exercise are increased in proportion to the fluid deficit.

As the level of dehydration increases, there is a reduction in venous return to the heart, which consequently reduces the stroke volume (Murray 1992; Cheuvront et al, 2003). The heart rate then increases to compensate for the drop in stroke volume, in an attempt to maintain the cardiac output (Dill & Costill 1974; Noakes 1993). This was demonstrated by Coyle & Montain (1992), who showed that for each litre of sweat loss, the heart rate increases by 8 beats per minute with a corresponding 1.0 litre per minute decrease in cardiac output. During sub-maximal exercise, it has been shown that 3 - 4% dehydration decreased the cardiac output, because the heart rate could not increase sufficiently enough to compensate for the decreased stroke volume (Sawka et al. 1979). Furthermore, Armstrong et al. (1997) suggested that mild dehydration (1% to 2% body mass loss) increased cardiovascular strain and the increase is directly related to the magnitude of dehydration accrued during prolonged exercise.

Mild dehydration (classed as 1% to 2% loss of body mass), has been shown not to affect mental function (Shirreffs et al. 2007). Sharma et al. (1986) showed no significant change in mental work under dehydration of 1%, 2% or 3% compared to normal conditions, while Edwards et al. (2007) showed that 1.5% to 2% dehydration did not impair mental concentration. However, Maughan (2003) suggested that the ability to concentrate and self-ratings of alertness declined progressively even when only a 1% to 2% body fluid deficit existed. It was suggested by Cian et al. (2000) that above 3% dehydration will affect mood and mental status, and also that the reduction in performance and mental readiness is proportionate to the degree of dehydration

beyond 3% of body mass losses (Shirreffs et al. 2007). Nielsen et al. (2001) hypothesised that fatigue due to hyperthermia during prolonged exercise in the heat was due in part to alterations in front cortical brain activity. They showed that alterations in the "electroencephalographic (EEG) activity in the frontal cortex reflected activity changes in the parts of the brain involved in the hyperthermia-associated reduced ability to exercise."

In conclusion, exercise-induced dehydration has been shown to affect the body's thermoregulatory and cardiovascular responses, and studies have also speculated that there may be an effect upon mental function. Sawka & Pandolf (1990) state that all of these responses serve to reduce an athlete's anaerobic capacity, muscular endurance, maximal aerobic power, and physical work capacity, and will clearly cause a decrement in performance.

2.3.3 Effects of Dehydration on Endurance Performance

Noakes (1993) observed that relatively few modern studies have looked at the effects of dehydration on performance, and this is true today as our understanding of elite athlete's hydration practices under competition situations remains limited. Instead of studying hydration, many researchers have focussed on the actions of carbohydrate supplementation on performance. However, dehydration levels of between 1 and 2% have insignificant effects on performance, while dehydration of greater than 2% appears to significantly impair endurance performance at 20 - 21°C (Coyle 2004).

Armstrong et al. (1985) induced dehydration on eight men using a diuretic, who then completed randomised races of 1,500 metres, 5,000 metres, and 10,000 metres while normally hydrated or with plasma volume reductions of around 10%. There were no conditions of thermal stress. They found that through the decrease in body weight, performance times increased by 0.16 minutes, 1.31 minutes, and 2.62 minutes respectively. The 5,000 metres and 10,000 metres times under a condition of dehydration were significantly different to times while euhydrated. Walsh et al. (1994) found similar results; they rode six trained male subjects for 60 minutes at 70% of their $VO_{2\ MAX}$ at 32°C, then to exhaustion at 90% of their $VO_{2\ MAX}$. They ingested a 400 ml bolus of 20 mmol/l sodium chloride or no fluid before, and 120 ml

of sodium chloride or no fluid every 10 minutes during the trial. This fluid ingestion significantly reduced the weight loss in the subjects, and that that cycling time to exhaustion was significantly increased under conditions of no-fluid intake (9.8 ± 3.9 min versus 6.8 ± 3.0 min).

Below et al. (1995) cycled eight males at 80% of their $VO_{2\ MAX}$ for 50 minutes in a warm environment, followed by a performance test on four occasions, and during the exercise they ingested either a carbohydrate solution or water. They found that both fluid replacement and carbohydrate ingestion independently improved high-intensity cycling performance compared to no fluid ingestion, and their effects were additive. However, Robinson et al. (1995) showed that water ingestion during a 1-hour performance cycle ride in a moderate environment of 20°C did not significantly improve the distance covered in the ride.

It appears that a dehydration level of 2% in warm environments has the capacity to decrease endurance exercise performance (Walsh et al. 1994; Below et al. 1995), while a dehydration of 2% body weight in cooler climates appears to have a less significant impact (McConell et al. 1997). This suggests that when athletes are exercising in warmer conditions, their main priority should be to fully offset sweat losses, while athletes in more temperate conditions could withstand this level of dehydration and prevent a reduction in performance. Coyle (2004) states that from a practical point of view, research should attempt to identify sports and environments where athletes can tolerate up to 2% dehydration without this causing a reduction in performance, and resulting heat illness etc.

To conclude, a dehydration level of between 2% and 7% consistently reduces exercise performance (Cheuvront et al. 2003), and this is in accordance with the vast majority of literature on this topic. However, Coyle (2004) suggests that athletes can tolerate a dehydration level of up to 2% in cooler conditions, and future work should attempt to identify these occurrences in different sporting environments.

2.3.4 Hypohydration and Possible Effects on Performance

Hypohydration is classed as the induction of a body water deficit prior to exercise, and is induced by fluid restriction, heat exposure, or through the use of diuretics (Barr 1999). One major difference of hypohydration compared to exercise-induced dehydration is that hypohydration leads to a large decrease in plasma volume, as opposed to the plasma volume remaining relatively constant during exercise-induced dehydration. Sawka et al. (1985) showed that hypohydration clearly impacts upon aerobic endurance, while the effects of hypohydration upon muscular strength and endurance are not clear (Barr 1999). Interestingly, Hornery et al. (2007) have suggested that technical elements of the service action were affected by adverse physiological conditions, including hypohydration.

2.3.5 Pre-exercise Hydration Status

Calculation of an individual's pre-exercise urine osmolality, and also their Urine Specific Gravity (USG) levels have been used as methods of determining pre-exercise hydration status (Maughan & Shirreffs 1997; Shirreffs et al. 2005). If an individual's pre-exercise urine osmolality is less than 900 mOsmol/kg, then they are considered to be in a euhydrated state, while a USG greater than 1.025 g/ml classes an individual as dehydrated. However, the ACSM's position stand concerning exercise and fluid replacement (Sawka et al. 2007) suggests that a pre-exercise urine osmolality of less than 700 mOsmol/kg is indicative of euhydration. Several incidences of hypohydration have been observed, especially in tennis players.

The majority of 38 football players tested were found to start a training session euhydrated (Shirreffs et al. 2005), while Maughan et al. (2004) found one professional footballer who had a reading of 1254 mOsmol/kg prior to a training session, which was classed as severely hypohydrated. 14 adolescent tennis players tended to turn up for training in a dehydrated state, shown by an average USG measurement of 1.025 ± 0.005 g/ml prior to the session being completed (Bergeron et al. 2006), while an average pre-match urine specific gravity reading of 1.022 ± 0.004 g/ml for 14 male professional tennis players during an international tennis tournament was observed (Hornery et al. 2007).

Bergeron et al. (1995a) showed that 12 male and 8 female US college tennis players did not, on average, start tennis matches in a euhydrated state: over half of the players had USG readings of greater than 1.025 g/ml. However, Peduzzi et al. (2005) recorded pre and post practice USG readings for eight rookie NFL players, and showed mean pre-exercise USG readings of 1.017 ± 0.001 g/ml compared to post-exercise readings of 1.022 ± 0.006 g/ml. This equated to osmolality readings of 672 ± 217 mOsmol/kg pre-exercise, and 740 ± 213 mOsmol/kg post exercise which showed that the rookie NFL players started practice well hydrated, and maintained their hydration levels well throughout the course of the practices.

2.3.6 Hyperhydration and Possible Effects on Performance

It has been clearly demonstrated that both hypohydration and dehydration can impair performance through several factors; therefore there may be sufficient cause to investigate whether commencing exercise in a hyperhydrated state can provide any advantage. Sawka et al. (2001) stated that many studies in this area have been poorly designed, and that some studies report lower core temperatures and higher sweat rates during exercise following hyperhydration, while some studies have not. Sawka et al. (2001) also suggested that studies have proved that glycerol can improve fluid retention. However, Murray (1992) concluded that hyperhydration does not provide a meaningful advantage compared to remaining well hydrated during exercise.

2.3.7 Possible Errors in the Estimation of Hydration Status from Changes in Body Mass

The method of estimating an individual's hydration status or alternatively calculating dehydration levels from changes in their body mass is extensively used; however, Maughan et al. (2007) suggested that there might be several sources of error, potentially leading to inaccurate results. The first source of error can arise through the amount of water that is lost through the respiratory passages, thus affecting calculations of sweat loss. Secondly, the water formed from the oxidation of metabolic fuels should be considered, and also osmotic disturbances may be sufficient enough to affect thirst perceptions and the rate and onset of sweating. Therefore, precise determination of the change in hydration status cannot be assessed by simply

measuring changes in body mass, but studying changes in body mass is useful to identify large discrepancies between fluid losses and fluid intake from ingested drinks.

2.4 Thirst and Fluid Intake

Thirst is defined by Greenleaf (1992) as the "sensation of dryness in the mouth and throat associated with a desire for liquids, and the bodily condition (as of dehydration) that induces this sensation or better described as the desire to drink resulting from a water deficit." Under resting conditions, the stimulus of thirst is adequate for fluid replacement because water balance is well maintained from day to day. However, under physiological conditions of stress thirst does not appear to be a sufficient stimulus for maintaining body fluid, and this is known as involuntary dehydration; where subjects drink to satiety but a water deficit still exists.

Up to 1.5 litres of body water could be lost before any feelings of thirst are felt by the athlete (Armstrong et al. 1985; Greenleaf 1992, Murray 1992), and by this time the negative effects of dehydration may already have commenced (Greenleaf 1992). This is supported by Bergeron et al. (1995a), who found no correlations between perceived thirst following a tennis match and sweat rates or body weight percentage change, which shores up the theory that the perception of thirst does not accurately indicate body water status. It also suggests that thirst is not a sufficient stimulus to prevent a substantial net body water loss during exercise in a hot environment (Greenleaf 1992).

The mechanisms behind the onset of the sensation of thirst are not well known. However, Bergeron et al. (1995b) suggested that hypothalamic osmoreceptors, extracellular fluid volume, angiotensin II, a dry mouth, and many other associated factors e.g. plasma sodium level, blood pressure, vasopressin and aldosterone levels all play roles in the mechanisms that controls thirst and fluid intake.

2.4.1 Voluntary Dehydration

The concept of voluntary dehydration is important when considering the effects of dehydration and an individual's fluid replacement strategy. The term 'voluntary

dehydration' appears to have no exact definition; instead it appears to arise from ad libitum drinking strategies (Sawka et al. 2001; Passe et al. 2007), or more precisely when a fluid deficit occurs even when an individual has sufficient time and fluid on hand to replace the body fluid they have lost, yet fail to do so (Armstrong et al. 1997). Murray (2007) stated that there are two main reasons for voluntary dehydration; firstly that fluid losses can be large, and secondly that the desire to ingest fluid rarely keeps pace with the rate of fluid loss.

The extent of voluntary fluid intake in adolescent tennis players was measured by Bergeron et al. (2006), and it was found that some players were seemingly dehydrated when they arrived for training even before any exercise induced fluid losses had occurred. This was shown by Urine Specific Gravity (USG) readings of 1.025 g/ml before both days of tennis play. In this study, fourteen adolescent tennis players completed two 120-minute training sessions drinking either unflavoured water in one session, or a carbohydrate electrolyte solution during the second session. On average, the players consumed 1693 ± 544 ml of water and 1832.7 ± 644.5 ml of a carbohydrate electrolyte solution in the respective trials. However, this fluid intake was not sufficient to match their fluid losses through sweating: the players lost 2290.8 ± 707.8 ml in the water trial and 2171 ± 576.5 ml in the carbohydrate trial. This situation of voluntary dehydration has been observed in other pieces of research: Dawson et al. (1985) who reported that during tennis matches of one-hour duration only 27% of the fluid lost by the players was replaced during the match, and players drank between only 130 ml and 600 ml. This resulted in a body mass deficit of 1.3% of body weight, which may not in itself be significant during a short match, but will most probably prove to be so during three set match lasting over 2 hours.

Passe et al. (2007) investigated the relationship between runner's perceptions of their fluid needs, and their drinking behaviour under heat stress. Eighteen marathon runners carried out a 10-mile track race, with limitless 6% carbohydrate-electrolyte solution available at miles 2, 4, 6 and 8. Also before and after the race they indicated how thirsty they felt, as well as a written survey after the test to find out their views on their sweat losses and hydration status. The results showed a prevalence of voluntary dehydration among the subjects, as on average their sweat loss was three times greater than their average fluid intake, even though limitless fluid was available. Furthermore,

the runners were able to estimate their level of fluid intake well, but they vastly underestimated their sweat losses: their perception of fluid loss was only 42.5% of actual fluid losses. This is interesting as the study used experienced runners who regularly competed in marathons and road races, and it could be assumed that they would have been well informed on their fluid intake and sweat losses during a race.

Armstrong et al. (1997) suggests voluntary dehydration is a complex behaviour, involving psychological and physiological components, and results in an increased core temperature and cardiovascular strain even if an individual commences exercise in a euhydrated state. Barr (1999) suggested that fluid deficits could arise through maximal gastric emptying rates being below maximal sweat rates. However, this is unlikely as many voluntary fluid intakes are low. Furthermore, Noakes (1993) stated that most athletes are "reluctant drinkers," and fail to ingest fluid at rates that match their sweat loss. However, the factors behind voluntary dehydration remain elusive (Noakes 1993). These issues make it of real interest to study the fluid intake habits of players and to inform them about their hydration practices.

2.4.2 Fluid Replacement, Gastric Emptying, and Carbohydrate-Electrolyte Drinks

The most effective defence against heat illness is provided by maintaining adequate hydration, and the ideal hydration protocol will balance water loss with water intake. Meeting this requirement is usually difficult because athletes tend to rehydrate poorly, often only replacing half the fluid that they have lost (Noakes 1993). This was previously addressed in the section on voluntary dehydration. It can also be difficult to rehydrate effectively if regular opportunities for rehydration are not available, as is the case in certain sports such as football, rugby etc. (Bergeron et al. 1995b). However, the issue of gastric emptying must be addressed while considering rehydration strategies.

Bergeron et al. (2006) reported that during a trial comparing the fluid intakes and core temperature responses of adolescent tennis players with water or a carbohydrate electrolyte drink, several players indicated feeling some gastrointestinal discomfort following carbohydrate ingestion. Gastrointestinal discomfort can occur when athletes consume a solution containing too great a level of carbohydrate; increasing the

carbohydrate level of a drink reduces the rate of gastric emptying of the fluid into the stomach, causing gastrointestinal distress (Bergeron et al. 1995b). Mitchell et al. (1992) investigated the effects of a carbohydrate drink on performance and fluid balance on 12 competitive players during two three-hour tennis matches. They showed that the ingestion of carbohydrate provided no particular improvement in performance, which was measured by a serve-velocity test and a shuttle run. Also, they found that the rate at which the researchers administered the drinks to the subjects: 11.5 ml/kg/hr was sufficient to prevent any significant dehydration: indeed on average only 1% of body mass was lost, and it was concluded that this was relatively acceptable for a three hour tennis match. Bartolozzi et al. (2005) showed that body weight and urinary hydration measures did not change during the first nine days of preseason training in sodium supplemented NFL players, and they concluded that by supplementing with two to four times more sodium than in commercial sports drinks, body weight could be maintained.

Ideal fluid replacement beverages should taste good and not induce gastrointestinal distress (Bergeron et al. 1995b). They should contain some carbohydrate (6 to 8g per 100 ml) to help maintain blood glucose (Sawka et al. 2007). Carbohydrate ingestion also helps to increase fluid osmolality. It should also contain sodium, which helps maintain the extra-cellular fluid volume without affecting thirst. Most sports drinks contain 10-20 mmol/l, which is adequate for electrolyte restoration. The topic of rehydration, incorporating the content of sports drinks and amounts that should be ingested is addressed further on. Finally, Boatwright et al. (2003) suggest a standard post-exercise rehydration strategy of drinking a solution containing 5-8% carbohydrates and approximately 60 mmol of sodium should be implemented after basketball games

The majority of studies that look at the ideal concentration of a carbohydrate drink seem to differ in their recommendations; however, the ideal carbohydrate concentration appears to be between 3% and 8%.

Studies have looked at the both the effects of volume and carbohydrate content on the rate of gastric emptying, while the osmolality, pH, and temperature are considered of lesser importance (Maughan & Leiper 1999). Maughan & Leiper (1999) state that

gastric emptying is largely affected by the volume of fluid in the stomach, and increasing the fluid volume will speed the emptying rate. However, gastric emptying is slowed by increasing the carbohydrate content of the solution (Vist & Maughan 1994, 1995), however the concentration at which this occurs varies on the literature, probably because of the range of protocols used.

Vist & Maughan (1994) compared the rate of gastric emptying of 20 g/l, 40 g/l and 60 g/l glucose solutions against water over a one-hour period. 20 g/l solution emptied at same rate as water. After the first 10 minutes, the 40 g/l and 60 g/l solutions were emptied slower, while unexpectedly the greater glucose concentrations delivered more glucose to the small intestine. This study demonstrated that glucose solutions of 20 g/l emptied at same rate as water, while increased glucose concentrations emptied slower. Vist & Maughan (1995) similarly reported that more dilute carbohydrate solutions empty faster, but deliver less carbohydrate to the small intestine than more concentrate solutions. Alternatively, Gant et al. (2007) studied gastric emptying during prolonged (60-minute) intermittent shuttle running at 30°C using a carbohydrate solution and flavoured water, and found no differences between total fluid volume emptied from stomach during each 15 minute exercise period. However, core temperature was increased following carbohydrate ingestion, and sprint performance was enhanced

Studies have compared the effects on performance of ingesting solutions with different carbohydrate concentrations (Tsintzas et al. 1995). Murray et al. (1989) found that when compared with a 6% solution, there was no benefit of consuming an 8% or 10% carbohydrate solution, while Tsintzas et al. (1995) concluded that a 5.5% carbohydrate solution produced a better performance than a 6.9% carbohydrate solution in a marathon run. It can be concluded that no optimal concentration has been found: according to Murray et al. (1989), this is because the physiologic, sensory, and performance responses of ingesting different carbohydrate solutions have not been well researched.

2.4.3 Hydration Issues in Tennis

There are certain factors unique to tennis which could influence a player's body water status. As a player walks to the changeover, they may not be motivated to drink even though a body fluid deficit may exist, as has been demonstrated in other sports. However, during changeovers, players can be seated for up to 90 seconds, and during this period there is a positive postural influence on the plasma volume, and no large muscle group activity, which causes the potential for a hypovolemic stimulus to drink to be reduced (Bergeron, 1995b). Even if there is enough fluid at courtside and a player is motivated to drink, it is often difficult to consume enough fluid to offset sweat loss. However, opportunities to drink are regularly available, and therefore large fluid deficits should be preventable.

Again, the effect of gastric emptying must also be considered. The rate of gastric emptying rarely exceeds 1.2 l/h, and to keep pace with 2.0 l/h sweat rates is difficult, verging on impossible, because it is uncomfortable to ingest large amounts of fluid in one sitting (Bergeron et al. 1995b). Bergeron et al. (2006) suggested that as previous research has shown that players should take in 0.12 to 0.24 l of fluid at every changeover, sweat rates of over 2.0 l/h could leave a body fluid deficit of up to 50%, which could severely impair cardiologic function, increase core temperature, and decrease performance. Therefore, the challenge for tennis players is clear.

2.5 Electrolyte Balance

Sodium (Na^+) is the predominant extracellular cation, and is found primarily in the extracellular fluids of the body (Bergeron et al. 1995b). It is absorbed in the upper small intestine, and although sodium can be reabsorbed by the tubules of the kidneys, some sodium is lost daily in sweat and faeces. Aldosterone helps in the maintenance of sodium homeostasis by increasing sodium reabsorption in the kidney, while Angiotensin II aids in sodium conservation at low serum sodium concentrations. Sodium plays a role in the maintenance of fluid balance and osmotic pressure in the body, and functions in nerve impulse transmission, muscle contraction, and in formation of the bone mineral apatite (Bergeron et al. 1995b). The major source of

dietary sodium is common table salt (NaCl). Research has examined the extent of sodium losses during all types of exercise, and has also investigated the role of sodium losses in the onset of heat-related muscle cramps. The other main electrolytes lost in sweat are chloride (Cl^-), and potassium (K^+). Magnesium and calcium are also lost, but in insignificant amounts (Bergeron et al. 2005b).

2.5.1 Sweat Electrolyte Content

Electrolyte losses can impair cardiovascular and thermoregulatory function, and if electrolyte losses are of a sufficient magnitude, then performance can be adversely affected (Maughan et al. 2004; Sawka et al. 2001; Murray 1992; Bergeron 2003; Kovacs 2006 etc.). Potassium, magnesium, and chloride are also lost in sweat, but they are not lost in sufficient volume to have any effect on performance. Table 1 shows a summary of studies that have examined the sweat sodium concentrations during exercise.

Maughan et al. (2005) recorded a mean sweat sodium concentration of 49 mmol/l from 17 male footballers following a 90 minute training session in a cool environment and this is comparable other studies that have assessed footballers at a range of conditions (Shirreffs et al. 2005; Maughan et al. 2004). Bergeron recorded sweat sodium concentrations of 22.4 ± 9.4 mmol/l for US college tennis players outdoors in 32°C (Bergeron et al. 1995a), and also recorded a concentration of 35.9 mmol/l for one male junior tennis player who had a history of suffering heat cramps (Bergeron et al. 1996).

Bergeron et al. (1995a) recorded a mean sweat potassium concentration of 4.5 mmol/l during midday singles matches across a four-day period. In addition, Bergeron (1996) recorded a sweat potassium concentration of 5.4 mmol/l in a study of one tennis player who suffered from heat cramping.

To summarise, the sweat sodium concentrations of athletes recently reported in a wide range of sports fall between 20 and 60 mmol/l, and this supports the work of Shirreffs et al. (1997), who stated that sweat electrolyte concentrations tend to fall inside the range of between 20 and 80 mmol/l for sodium, 4 to 8 mmol/l for potassium, and 20

Table 1: A summary of studies examining sweat sodium concentrations during exercise in a range of temperatures.

Authors	**Sport**	**Subjects**	**Protocol/Conditions**	**Sweat collection sites**	**Sweat Sodium (Mean ± SD where applicable)**
Bergeron et al. (1995a)	Tennis	20 US College Division 1 players (12 male 8 female)	3 days: 2 singles 1 doubles matches per day 32.2°C 53.9% humidity	Non-dominant forearm	Males: 22.4 ± 9.4 mmol /l Females: 21.4 ± 12.1mmol/l
Bergeron (1996)	Tennis	One 17 year old elite male player with a history of heat cramping during matches	1 best of three set match 31.6°C 62% humidity	Non-dominant forearm	35.9 mmol/l
Shirreffs et al. (2005)	Football	26 male professionals, results collected from a subgroup of 7	90 min practice session 32.3°C 20% humidity	Chest, arm, back and thigh	30.2 ± 18.8 mmol/l
Maughan et al. (2005)	Football	17 male professional footballers	90 min practice session 5°C 81% humidity	Chest, forearm, back, thigh	43 mmol/l
Greene et al. (2007)	American Football	9 male heat cramp sufferers and 9 male control team-mates	2 hour practice session on 3 consecutive days WBGT: 79.5	Upper forearm	Crampers: 55.7 ± 20 mmol/l Non-crampers: 44 ± 18 mmol/l

Table 1 (continued):					
Maughan et al. (2004)	Football	24 male premiership footballers	90 min practice session between 24 29°C	Chest, forearm, back, thigh	49 mmol/l
Stofan et al. (2005)	American Football	5 division 1 male college players with a history of heat cramps, and 5 male team-mates who had never cramped	Tested during the first 30 minutes of a practice session 22.7 – 26.0°C	Forearm	54.6 ± 16.2 mmol/l in players with cramping history 25.3 ± 10.0 mmol/l in players with no cramping history

to 60 mmol/l for chloride. The vast majority of studies have reported sweat electrolyte concentrations within these ranges (Maughan et al. 2004, 2005; Shirreffs et al. 2005; Bergeron et al. 1995a; Fowkes Godek et al. 2005). These studies also demonstrate the considerable variation that exists between individuals in terms of electrolyte losses, shown by large ranges in results (Sawka et al. 2007). According to Maughan & Shirreffs (1997), training status and acclimatisation can account for part of the variation, but it has been shown that in some cases, one individual can lose up to five times as much sodium than another during the same training session when equally acclimated (Shirreffs et al. 2005).

2.5.2 Total Electrolyte Losses

The total electrolyte losses that occur during exercise can be important to know if there is any possibility of experiencing a substantial sodium loss, which could lead to a variety of problems if the magnitude is great enough (Bergeron 1995b). Observations range depending on the individuals effort intensity, their predisposition for electrolyte loss during exercise, and the environmental conditions.

Maughan et al. (2004) studied footballers during a 90-minute training session in a cool environment, and measured an average total sodium loss of 73 mmol, which is similar to the results of Shirreffs et al. (2005), who recorded an average total sodium loss of 67 mmol over the same duration of training session in the warm conditions. However, the results ranged from 26 mmol of sodium lost to 129 mmol of sodium lost, showing how dependant upon the individual electrolyte losses, as well as sweat rates, can be. Bergeron (1996) found that a 17-year-old male tennis player with a history of suffering heat cramps lost 88.9 mmol of sodium per hour of play in a warm environment, which came close to exceeding his average daily sodium intake. It was recommended that the player increased his dietary sodium intake to reduce the chances of heat cramps occurring.

2.5.3 Effect of Heat Acclimation on the Sweating Response

Heat acclimation occurs when a person moves from a cool to a hot climate or from a hot-dry to a hot-wet climate (Sparling 2000). It typically requires 10 to 14 days in the warmer climate, but 75% of the adaptation occurs in the first 5 days (Armstrong

2000). Acclimation can increase the sweating capacity from about 1.5 l/h to 4.0 l/h, and this is accompanied by a more complete and even distribution of sweating, which can be advantageous in humid heat. Sweat sodium losses decrease because of an increased secretion of aldosterone. Furthermore, training in the heat encourages physiological changes that promote exercise capacity (Murray 1992; Sawka et al. 2001). Cutaneous blood flow increases, and this shunting of blood to the skin improves heat loss from the body (Sparling 2000). The plasma volume is increased, less sodium is lost in sweat, the onset of sweating and the rise in skin blood flow occurs at a lower core temperature. The fall in the sweating threshold is important for keeping core temperatures from increasing rapidly during the early stages of exercise. In addition, the author states that heat acclimatisation enhances voluntary fluid consumption, reducing the extent of voluntary dehydration.

To conclude, being acclimatised to a hot environment has several physiological and thermoregulatory advantages. Essentially, acclimatised individuals can exercise with lower core and skin temperatures, as well as at reduced heart rates, compared to a non-acclimatised person.

2.5.4 Relationship between Sweat Sodium Concentration and Sweat Rate

Many studies have reported the heat acclimation causes a significant decrease in sweat sodium ion concentration (Kirby & Convertino 1986; Nielsen et al. 1997). However, according to Buono et al. (2007), these studies have failed to take into account the effect that changing the sweat rate can have on the sweat sodium concentration. It was also stated in this paper that changes in sweat sodium concentration following heat acclimation could be misleading if they are not presented in relation to sweat rate, because the amount of sodium ions that escape reabsorption is known to increase linearly with increasing sweat rate (Buono et al. 2007).

In the study by Buono et al. (2007), eight healthy male subjects completed a 10-day heat acclimation protocol. They found that sweat osmolality was reduced for a given sweat rate as a result of heat acclimation. During low levels of sweat production, significant amounts of sodium and chloride ions can be reabsorbed from the sweat as it moves along the duct. However, the rate of sodium and chloride reabsorption has a

finite capacity, and at high sweat rates there is insufficient time for reabsorption to occur. As mentioned previously, this is shown by the fact that the sweat sodium concentration increases linearly with increasing sweat rate. Buono et al. (2007) showed that heat acclimation shifted this line to the right, suggesting that heat acclimation improves the reabsorption of the sweat duct. This suggests that as an individual becomes more acclimatised, they are able to produce higher sweat rates but with a lower electrolyte concentration as compared to an individual who is not acclimatised.

2.5.5 Evidence of Sodium Depletion in Heat Cramps

Water and sodium losses from profuse/repeated sweating have been cited as primary contributing factors to onset of heat cramps, a condition which begins with the subtle twitching of muscles and can progress to the cramping of whole muscle groups (Stofan et al. 2005; Bergeron et al. 1995a, b, 1996, 2003; Maughan et al. 2004; Shirreffs et al. 2005, 2006; Hosey & Glazer 2004; Eichner 2007, Sawka et al. 2007).

Stofan et al. (2005) carried out an observational study where Division 1 collegiate American footballers who had a history of suffering heat cramps were matched with team-mates who had never cramped. By taking sweat samples from a patch on the player's forearms, they found that the sweat sodium concentrations in those with a history of cramping were almost double that of those who did not cramp: 54.6 ± 16.2 mmol/l compared to 25.3 ± 10.0 mmol/l.

Bergeron (1996) studied a 17-year-old nationally ranked male tennis player who had often suffered heat cramps in his quadriceps, hamstring, and calf muscles for two years. During a singles match against a competitively matched opponent, sweat was collected form the subject's non-dominant forearm, and this took place at a changeover around 20 minutes into the match. Bergeron found that the player had a sweat rate of 2.5 l/h, and that his sweat sodium loss rates were extensive (89.8 mmol per hour of play). It was recommended that the player increase his dietary sodium intake to 261-348 mmol, which is higher than the average daily sodium intake, and in the 9 months post study, the player had not experienced any heat cramps. Also, Bergeron (2003) stated that heat cramps tend to occur in the later rounds of

tournament competition, where a period of extensive training or match-play has closely succeeded the onset of the cramps. This idea is supported by Bergeron et al. (1995a), as it was shown that playing tennis matches on repeated days caused a cumulative deficit in plasma sodium levels. If a player feels the onset of muscle cramping, the first step in treatment should be the consumption of an appropriate salt solution: Bergeron (2003) recommended 3g of salt dissolved in 16-20 ounces of Gatorade.

Conversely, Jung et al. (2005) suggested that dehydration and electrolyte losses were not the sole causes of exercise-associated muscle cramps, because in a study using a protocol of several different cramp-inducing exercises 69% of subjects experienced cramping when they were supplemented with electrolytes and fully hydrated. However, this study would not have taken into account any previous exercise, which is relevant due to the nature of repeated sodium losses on the incidence of muscle cramping.

To conclude, research is undivided on the primary causes of heat cramps. Some studies have suggested a increased sodium loss in cramp-affected athletes, while Sulzer et al. (2005) concluded that muscle cramps were nor associated with higher dehydration levels, or serum electrolyte differences, while instead they concluded that cramping may be caused by an increased neuromuscular activity (shown by increased EMG amplitudes).

2.5.6 Hyponatremia

The ingestion of large volumes of plain water following exercise-induced dehydration causes a fall in plasma osmolality and in the plasma sodium concentration (Maughan & Shirreffs 1997), and this could, in extreme cases, lead to hyponatremia. Also known as low sodium concentration or water intoxication, it can occur as a result of “whole body fluid overload,” (Noakes 2002) which results from continued high water or fluid intake. It is diagnosed when serum sodium concentrations falls below 135 mEq/l (Swain 2004).

Hyponatremia exists in either symptomatic or asymptomatic forms (O'Connor 2006). Athletes suffering from symptomatic hyponatremia, the more serious of the two, are those that lose the least body weight during exercise, and are the ones who have excessively ingested water, thus having the lowest serum sodium levels. Noakes (1992) states that hyponatremia becomes symptomatic when the volume of fluid ingested exceeds 2 to 3 litres. Asymptomatic hyponatremia tends to occur when athletes fail to replace fluid losses sufficiently, and present milder symptoms of the condition.

The overwhelming consensus in the literature is that the most common cause of hyponatremia is an excessive intake of fluid, mainly water, during exercise. Noakes (2002) states that the belief held by athletes and coaches that individuals should drink as much fluid as possible during exercise has increased the occurrence of this condition, and its prevalence could be "eliminated from sport immediately" if all athletes were made aware of how the condition arises, and the dangers of suffering from it.

Hyponatremia is a relatively common condition among ultra-endurance athletes, demonstrated by Hew-Butler et al. (2003)'s observation that 21 out of 55 finishers of the 2000 Houston marathon that required intravenous care were found to be hyponatremic.

2.5.7 Fluid and Electrolyte Replacement

Optimal rates of fluid replacement during exercise relate to the athlete's ability to tolerate fluid ingestion, and the body's ability to absorb it. As demonstrated previously, Robinson et al. (1995) showed that water replacement does not improve 1 hour cycling performance in ambient temperatures, while Below et al. (1995) suggested that fluid and carbohydrate ingestion independently improved performance during 1 hour of exercise. These differences may be down to the differences in exercise intensity, and differences in protocols used. However, McConell et al. (1997) showed an improved performance with fluid replacement following 2 hours of exercise. As a matter of habit, athletes should aim to fully replace sweat losses, at a rate that as closely matches sweat rates as possible (Coyle 2004).

The need for sodium supplementation stems for the need to replace electrolyte losses that could be sustained through exercise. Also, a carbohydrate-electrolyte solution promotes fluid absorption more effectively than plain water (Bergeron et al. 2006). This is because in the presence of glucose, water transport is enhanced. Bergeron et al. (2006) also state that sodium present enhances palatability, and replaces the portion of the sodium pool lost in sweat.

Maughan & Lieper (1995) showed that urine output following ingestion of a sodium-containing fluid in a dehydrated state was inversely proportional to the sodium content of the ingested fluid. Also, between trials with 2 mmol and 100 mmol of sodium, there was a fluid balance difference of 787 ml, which is clearly a substantial difference in hydration status. Furthermore, Bartolozzi et al. (2005) concluded that by supplementing with two to four times for sodium than that present in commercial sports drinks, body weight can be maintained; suggesting that there are not sufficient electrolyte levels in some commercial sports drinks.

The American College of Sports Medicine guidelines suggest that rehydration strategies should focus on re-establishing body weight to pre-exercise levels, and this could involve ingesting up to 150-200% of fluid losses because of urine production. Optimal rehydration is only achieved if the electrolytes lost in sweat are replaced along with water (Maughan & Shirreffs 1997). Maughan & Shirreffs (1997) stated the typical sports drink contains between 10 and 25 mmol/l of sodium. Increasing the sodium concentration in drinks could leave them unpalatable, meaning salt tablets may be a preferred method of supplementation. However, the sodium concentration of many rehydration solutions to treat diarrhoea induced dehydration is 80 mmol/l, which is at the upper limit of sweat sodium concentrations observed in many different sporting environments, and these drinks are not unpleasant to taste. The 2007 ACSM position stand for fluid and electrolyte replacement suggests a sodium concentration of 20-50 mmol/l to aid sodium replenishment (Sawka et al. 2007).

Hew-Butler et al. (2005) studied 413 triathletes that completed the 2001 Cape Town Ironman Triathlon to assess whether sodium supplementation is necessary to maintain serum sodium concentrations during prolonged endurance exercise and prevent the development of hyponatremia. They found that athletes who ingested either placebo

or salt tablets ad libitum all maintained their serum sodium concentrations within the normal range while completing the Ironman Triathlon, thus concluding that sodium supplementation was unnecessary during prolonged endurance exercise. They also found in this study that sodium supplementation did not result in any improvement in performance; there were no significant differences in finishing times between the placebo and sodium groups.

2.5.8 Dietary Sodium Intake for Athletes and Non-Athletes

In the Institute of Medicine's (IOM) 2004 release of Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate, it recommended that the daily sodium intake in healthy males between 19 and 50 years of age was 1.5 g of sodium (corresponding to 3.8 grams of salt). This paper also stated that this recommended level is way below the estimated 3.2 grams per day of sodium consumed daily on average by individuals in the United States. Older adults and the elderly require somewhat less sodium based on lower energy intakes. For athletes, these guidelines do not apply, as sodium is lost through sweat, but it is useful to be aware of recommendations for normal individuals.

Work that has addressed athlete's sodium intake with respect to training and competition is less readily available, as many studies instead focus on the deleterious effects of dehydration upon performance. Stofan et al. (2005) assessed five US college football players with a history of heat cramps, and recorded that across a two-day period, with two practices on each day, on average the players took in 5.7 ± 3.2 g of sodium in their fluids, along with 10.3 ± 4.1 g of sodium in food. This was compared to 1.5 ± 1.8 g and 7.4 ± 3.3 g in their non-cramping team-mates. Furthermore, they found that the players with a history of cramping lost twice as much sodium in their sweat when compared to their team-mates.

Shirreffs et al. (2006) stated that 43 out 48 adult footballers were observed to lose less than 3 to 4 grams of sodium during a 90-minute training session, or a match and the authors stated that these losses would not be sufficient to warrant specific attention, as dietary intake should be large enough. The maximum sodium loss observed was 5.1 g, and in this case, some attention should be paid to sodium replacement during exercise.

It appears that in tennis for example, if matches are being played on consecutive days, then there is a case to increase the sodium intake in the player's diet to counterbalance the effect of a lowered sodium plasma level, as found by Bergeron et al. (1995a). However, if matches are just being played on a one-off basis, there seems little evidence to increase the intake of sodium beforehand, as a carbohydrate-electrolyte drink ingested during the match should provide enough benefit.

When exercise sessions are separated by several days then no special attention to sodium replacement should be required, and the normal dietary sodium intake should be sufficient (Maughan et al. 2005). However, if sodium losses of up to 120 mmol occurs, like those recorded in some footballers by Maughan et al. (2005) and Shirreffs et al. (2004), then the normal diet is unlikely to adequately replace these losses.

According to Bergeron, (2000), there are also individual differences in sodium balance, and those who suffer from dehydration and heat cramps are those who lose an extensive amount of sodium in their sweat, as well as having a typically low salt intake in their diet. Adding salt to the diet can help prevent a sodium deficit, however for serious athletes it is recommended to follow a specific plan so they are accustomed to it for competition. Furthermore, if as in tennis there is a need to compete more than once in day, an electrolyte containing sports drink should be consumed so it can be rapidly digested (Bergeron 2003).

2.5.9 Methods of Sweat Collection: Whole Body Washdown versus Local Sampling Methods

Although a wide variation in sweat electrolyte levels have been observed in many studies, there may be significant errors in the process used for data collection (Shirreffs & Maughan 1997). The two main methods of sweat sampling used are the collection of sweat from a specific region of the body using a patch or enclosing bag, or some variation on the whole-body wash down technique.

Using a patch or enclosing bag to collect sweat has been shown to over-estimate electrolyte concentrations, and this has been attributed to a difference in composition

caused by the restriction placed by the patch or the bag on the evaporation of sweat (Weschler, 2008, Shirreffs & Maughan 1997). This procedure may overestimate whole-body sweat sodium losses by approximately 30-40% (Shirreffs et al. 2005).

Patterson et al. (2000) aimed to assess whether sweat collected from different sites could accurately estimate whole-body concentrations. By collecting sweat from 11 different sites, as well as the whole-body, they discovered that the whole-body sweat rate derived from the 11 sites overestimated the whole body mass loss. In addition, they showed that the regional sweat sodium and chloride concentrations were much larger than the whole body values. This study also showed that several sampling sites possessed strong relationships with whole-body concentration of constituents. Patterson et al. (2000) demonstrated that taking the mean of eight skin sites was no more accurate than taking the mean of four skin sites, concluding that the sweat sodium concentration and electrolyte loss can be accurately predicted from regional sweat collections. The thigh, forearm, calf, foot, and lower back sites displayed the greatest correlation coefficients to whole-body sweat rates and electrolyte concentrations, and this influenced the decision to use the thigh, calf, forearm, and lower back sites in the present study.

The vast majority of studies that investigate sweat rates and electrolytes in different athletes tend use patches to absorb sweat during competition, as this method is the least obstructive to athletic performance. This is indeed the case in tennis, where studies by Bergeron (1995a), Bergeron (1996), Bergeron (2003), and Mitchell (1992) have used adhesive patches to collect samples, as this causes the least amount of hindrance. However, the study by Bergeron et al. (1995a) only placed a sweat patch on the subjects for an average of 26 ± 4.1 minutes during the match and only from the non-dominant forearm of the player.

Shirreffs & Maughan (1997) devised a new method of sweat collection, which involved a variation on the whole-body wash down technique, but this could only be effective when the exercise is performed stationary, such as on a cycle ergometer.

2.6 Effort Intensity during Match play

Tennis is characterised by intermittent exercise, of alternating short bouts of high intensity (4-10 seconds) exercise, and short recovery bouts (10-20 seconds) interrupted by breaks of longer duration (60-90 seconds) (Fernandez et al. 2006). Effective playing time is between 20% and 30% of match duration on clay courts, and 10-15% on faster courts (indoor hard, grass). Many factors can affect the intensity of tennis match play, including a player's tactical behaviour, playing surface, and environmental factors. Indeed, men and women participate in significantly longer rallies at the French Open (slow clay court surface), than other Grand Slams, and similarly rallies at Wimbledon (fast grass court surface) are significantly shorter. A summary of the main studies relating to on-court heart rates and match intensity is provided in Table 2.

Several studies have investigated heart rate responses during singles match play, with a calculation of exercise intensity through comparison with heart rates and VO_2 scores recorded during sub-maximal exercise. This has been done based on the linear relationship between heart rate and oxygen consumption (VO_2) which is observed during sub-maximal continuous exercise (Dill 1942). There exists an important limitation of this method however: in that assessing effort intensity in this way presents problems linked to dehydration and core body temperature regulation. This issue, termed "cardiovascular drift" is associated with sweating and a redistribution of blood so that peripheral circulation is increased. Since fluid is lost through sweating, venous return is reduced, decreasing the stroke volume. The heart rate then increases to maintain a constant cardiac output. Heart rate has been shown to increase by 10 to 15% (Achten & Jeukendrup 2003), and is accentuated by increasing dehydration and heat stress.

Hornery et al. (2007) studied 14 male tennis players during tournament play on two court surfaces (hard and clay), and recorded average heart rates of 152 beats per minute (hard) and 146 bpm (clay). These values were 93% (hard) and 94% (clay) of the maximal heart rates previously recorded when the subjects completed a 20 metre shuttle max test. Christmass et al. (1998) recorded a HR Max of 86.1% during rallies from the second end-change to the end of a match and that showed that a significant

Table 2: A summary of the main studies relating to on-court heart rates and match intensity.

Authors	**Subjects**	**Protocol/Conditions**	**Average/Maximum Heart Rates**	**Percentage of Maximum Heart rate (% MaxHR) (Mean ± SD)**
Elliot et al. (1985)	8 male college tennis players	Four one hour singles matches at 21.5°C WGBT	Average heart rate while serving = 157 bpm Average heart rate while receiving = 148 bpm	82.5% of maximum heart rate while serving 77.4% of maximum heart rate while receiving
Bergeron et al. (1991)	10 male Division 1 college tennis players	10 matches lasting 85 minutes, indoors on hard courts at approximately 17°C	Average heart rate = 144.6 ± 13.2 bpm	61.4% of maximum heart rates
Christmass et al. (1998)	8 state level tennis players	8 singles matches, lasting 90 minutes after a 10-minute warm-up on outdoor hard courts at 20 ± 1°C	Maximum heart rate = 189 ± 3 bpm	86.1 ± 1% during rallies 82.8 ± 1.1% during recovery (excluding change of ends)
Seliger et al. (1973)	16 Nationally ranked Czech male tennis players	16 matches, indoors on hard courts 5 minute warm-up, then 10 minute match followed by 26 minute recovery (temperature not stated)	Average heart rate ranged from 132 to 151 bpm	No calculation of percentage of maximum heart rate carried out in the study.

Table 2 (continued):

Docherty (1982)	42 men split into three ability groups	30 minute singles match after a 10 minute warm-up, played outdoors on hard courts between 20 and 24°C	Not stated in bpm	Percentage of maximum heart rates ranged between 65% and 71% for the 3 ability groups.
Dawson et al. (1985)	8 male college tennis players	Four one hour singles matches at 21.5°C WGBT on an outdoor hard court	Average heart rate = 152 ± 4.0 bpm	Percentage of maximum heart rate = 79 ± 2.0%
Bernardi et al. (1998)	Seven regionally ranked (non-professional) players, grouped into three playing styles (baseline/attacking/whole court)	15 matches (each subject played 2 matches) on a clay court	Baseline: 165 ± 7 bpm Attacking: 121 ± 15 bpm Whole court: 153 ± 6 bpm	Baseline: 82.5% Attacking: 63.6% Whole court: 79.6%

standard 85 minutes of singles tennis was 144.6 ± 13.2 bpm. This corresponded to 61.4% of the maximal heart rate score obtained during a treadmill test.

Some studies have simply stated the average and maximal heart rates observed during tennis matches, which is a less useful indication of match intensity. Seliger et al. (1973) studied 16 tennis players indoors, who undertook a 5-minute warm-up, followed by 10 minutes of match play, and then a 26-minute recovery period. Average heart rate readings of between 132 to 151 bpm were recorded. The protocol employed in this study is unusual, and seems unlikely to be able to give true representations of average and maximal heart rates. Kindermann et al. (1981) recorded an average heart rate of 145.9 ± 19.8 bpm in a singles tennis match of one hour duration, while in a squash match of the same duration the average heart rate was higher: 163.3 ± 14.8 bpm.

Mitchell et al. (1992) studied heart rate variations, among other factors, in 12 tennis players who played a three-hour tennis match having consumed either a 7.5 g per 100 ml carbohydrate drink, or a water placebo. They found that after 10 minutes of match play the heart rates in both trials were around 155 bpm, and in the water placebo trial heart rates reduced steadily over time to about 141 bpm. Comparatively, in the carbohydrate trial each drop in heart rate was then followed by a rise, ultimately reaching around 140 bpm after 3 hours. Smekal et al. (2001) recorded average heart rates for 20 male tennis players of 151 ± 19 bpm across 10 matches of 50-minute duration.

Smekal et al. (2001) also recorded respiratory gas exchange measures every 10 seconds for both tennis players during 50-minute matches. In total, 135 games were analysed in this study, and the average $VO_{2\ MAX}$ value was 29.1 ± 5.6 ml/kg/min and for a single game, the value ranged from 10.4 to 47.8 ml/kg/min They reported that these score were similar to $VO_{2\ MAX}$ values of U.S collegiate players, but lower than those obtained for professional players, and also concluded that the energy demands of tennis were rather low. They proceeded by assuming that average values do not effectively represent the physical activity patterns during a tennis match. The highest average VO_2 obtained for the entire game recorded was 47.8 ml/kg/min and they stated that the scores for high intensity games may serve as a guide for energy demands required to sustain high-intensity periods predominantly by aerobic mechanisms of energy supply. The mean $VO_{2\ MAX}$ results from Smekal et al. (2001) support the review by Fernandez et al. (1996), who

stated that players range between 46% and 54% of their $VO_{2\ MAX}$ (between 23 ml/kg/min and 29 ml/kg/min).

Bernardi et al. (1998) assessed three different styles of tennis players (baseline, attacking, and whole court players), on a clay court, and demonstrated that the longer the duration of a rally, then the higher the intensity of exercise. By calculating the ventilatory thresholds of these players using a maximal treadmill test, they were able to quantify the intensity of effort. Baseline players achieved a mean heart rate of 165 ± 7 bpm, equating to 82.5% of maximal heart rates: 40 bpm greater than those classified as attacking players. The mean percentage of $VO_{2\ MAX}$ recorded for baseline players was 59%, which the authors classified as being sufficiently high as to induce longer-term cardiovascular adaptations. The more attacking, and whole court playing styles were not of sufficient intensity to maintain or develop cardiovascular fitness.

Tennis is characterised by both anaerobic and aerobic metabolic responses, although conclusions differ as to the contributions of these pathways (Christmass et al. 1998), attributed to limitations in the study design. Seliger et al. (1973) suggested that that tennis involves 88% aerobic activity and 12% anaerobic activity from their study analysing energy metabolism in tennis.

Interestingly, Elliot et al. (1985) showed that the percentage of maximal heart rates during points and in recovery were higher during serving games as opposed to when they were receiving: 82.5% of maximum heart rate while serving compared to 77.4% while receiving. The authors attributed this to both the physical effort involved in serving, and potential emotional effects of serving on heart rates. Finally, Docherty (1982) showed that heart rate responses in tennis were significantly lower than those recorded in squash and badminton, and this was attributed to the extent of the margin of error; it was considered higher in squash because of the nature of the sport, because it is walled, compared to tennis where it is more open.

To conclude, several studies have investigated the heart rate responses to playing singles tennis. The average heart rates observed appear to be between 140 to 150 bpm, while maximal heart rates reach up to 179 bpm (Bergeron et al. 1991). Bernardi et al. (1998) recorded average heart rates of 165 bpm on a clay court. Some studies have expressed the

heart rates as a percentage of maximal heart rates calculated through off-court methods (Dawson et al. 1985; Bergeron et al. 1991; Christmass et al. 1998, Bernardi et al. 1998), and this is a more informative way of representing the intensity at which players work at during a match. Players in the studies reviewed have been shown to work at between 61.4% and 86.4% of their maximal heart rates. Since the average physiological responses to tennis match play have been shown to be modest, Fernandez et al. (2006) suggest that the stop-start natures of tennis cause mean values to lose their importance. Instead, they state that the high intensity periods should be more closely analysed.

2.6.1 Metabolite responses during Match play

Several studies have suggested that lactate concentrations remain low during tennis match play (Konig et al. 2000; Bergeron et al. 1991). In the study by Smekal et al. (2000), during 10 tennis matches lasting 50 minutes, blood lactate concentrations were measured at the end of each game and the average blood lactate level was 2.07 ± 0.88 mmol/l, with a range from 0.7 to 5.2 mmol/l. In addition, Christmass et al. (1998) recorded a pre-exercise plasma lactate concentration of 2.13 ± 0.32 mmol/l, and also showed that the lactate concentration increased significantly to reach a peak of 5.86 ± 1.33 mmol/l at the sixth changeover during a singles match.

Fernandez et al. (2006) found that blood lactate concentrations were significantly higher during service games while compared to returning games; however Smekal et al. (2001) suggested that there was no difference between serving and returning games. Interestingly, Ferrauti et al. (2001) suggested that high lactate concentrations during long rallies may influence certain point situations. They showed that when the recovery between rallies is too short, the running speed for stroke preparation, and the stroke speed is decreased, meaning that if lactate is not metabolized effectively performance may be adversely affected.

Blood glucose levels did not significantly change from pre to post exercise following 85 minutes of singles tennis match play indoors at 17°C (Bergeron et al. 1991). They also showed no changes in plasma lactate during play, and this was attributed to the subjects being well endurance trained, as endurance trained individuals have been shown to have lower lactate concentrations than untrained individuals. Kindermann et al. (1981)

suggested that during a one-hour singles tennis match, glucose levels remained unaltered; whereas a slight depletion of glucose levels throughout squash match play was observed.

To conclude, blood lactate appear to stay fairly low during match play, and there exists a possibility that lactate concentrations increase during serving games (Fernandez et al. 2006). There also appears to be little change in blood glucose levels pre to post match.

Chapter 3

Methodology

16 male subjects between the ages of 18-37 from the University of Stirling Scholarship Tennis Squad and the Club Performance Programme were recruited for the study through a process of individual communication with each subject. 14 subjects were currently or had been in the previous year nationally ranked between 15 and 750 in Great Britain, and competed regularly in tournament play, while two regularly competed in tournaments but were not nationally ranked. Subject characteristics were age; 21.5 ± 4.4 years, weight; 75.6 ± 8.6 kg, height; 179.5 ± 6.9 cm, peak VO_2; 4.46 ± 0.62 l/min.

The research project was approved by the University of Stirling Sports Studies Ethics Committee. The experimental procedures and possible risks were explained to each subject and they gave their consent to participate. There were two main parts to the study: a graded running test to exhaustion on a treadmill, and a best of three set tennis match indoors at the University Tennis Centre.

3.1 Graded Running Test to Exhaustion:

The participants arrived at the Gannochy Sports Centre and underwent a graded maximal running test to exhaustion to determine their VO_2 peak and their heart rate responses to exercise. Upon arrival, the subject completed a pre-participation screening questionnaire to confirm they were fit to participate in the study. Subject's age, height and weight were recorded, their resting blood pressure and heart rate was recorded using a portable machine, and a heart rate monitor (Polar S625X) was attached to their chest.

The subject's VO_2, and V_E, and heart rate were measured continuously using on-line gas analysis system (Sensormedics VMax 29, Holland) and a continuous incremental protocol performed on a Marquette 2000 treadmill. A mouthpiece and nose clip was attached to the subject, and the heart rate monitor positioned. Once baseline expired gas analysis measures were obtained then the treadmill was increased to the starting speed of 10km/h for each subject. The gradient was set at 1% to start. For the first 6 minutes the speed increased by 1 km/h every 2 minutes. After 6 minutes, the gradient was increased by 2% every minute until the participant reached volitional exhaustion. Once the subject reached exhaustion, they were instructed to straddle the running belt, and the speed and gradient were reduced to 3.5 km/h and 1% respectively, so the subject could walk for recovery. Finally, the mouthpiece and nose clip were removed. The data from the test

was then averaged over 1 minute intervals and transferred into Microsoft Excel for analysis.

3.1.1 Calculation of heart rate and VO_2 for translation of on-court heart rate data

The calculation of heart rate zones for the on-court test was carried out as follows: VO_2 results were plotted against V_E results for each player and the end of zone 1 (first ventilatory threshold) was taken as the first point which corresponded with a break in linearity. Zone 2 started from this point, and then finished with the next break in linearity, and this was repeated to give the third zone (zone 3). The VO_2 value corresponding to the beginning and end of each zone was then entered in the equation which was provided, from plotting the heart rates with VO_2 results obtained at one minute intervals. This gave the equivalent heart rate value of each boundary. This method provided three zones of intensity (Seiler & Kjerland 2006), from which it was possible to calculate how long each player spent in a specific zone during their match. The breakpoints and zones identified were independently verified by having two people assess the figures.

Example

- VO_2 was plotted against V_E in Microsoft Excel (Figure 1). The three zones were then calculated from the breaks in linearity.
- In this example, Zone 1 = < 2.25
  Zone 2 = 2.25 to 3.75
  Zone 3 = > 3.75

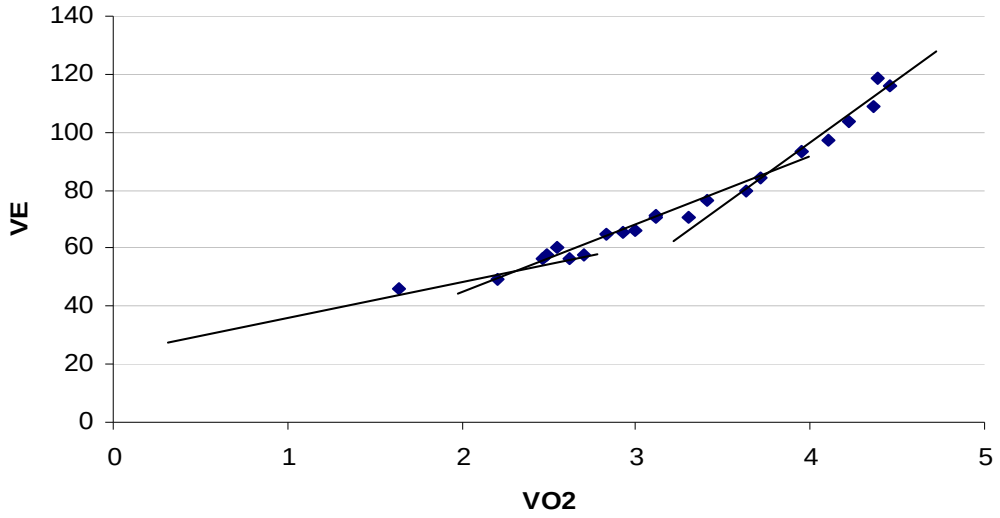

Figure 1: Graph showing VO_2 plotted against VE gained from the graded running test, and zones calculated from breaks in linearity.

- Heart rate was then plotted against VO_2 (Figure 2). The boundary of each zone was then substituted into the equation provided from the best fit line, to give the heart rate boundaries (bpm)
- This corresponds to Zone 1 = <149 bpm

  Zone 2 = 149 -185 bpm

  Zone 3 = >185 bpm

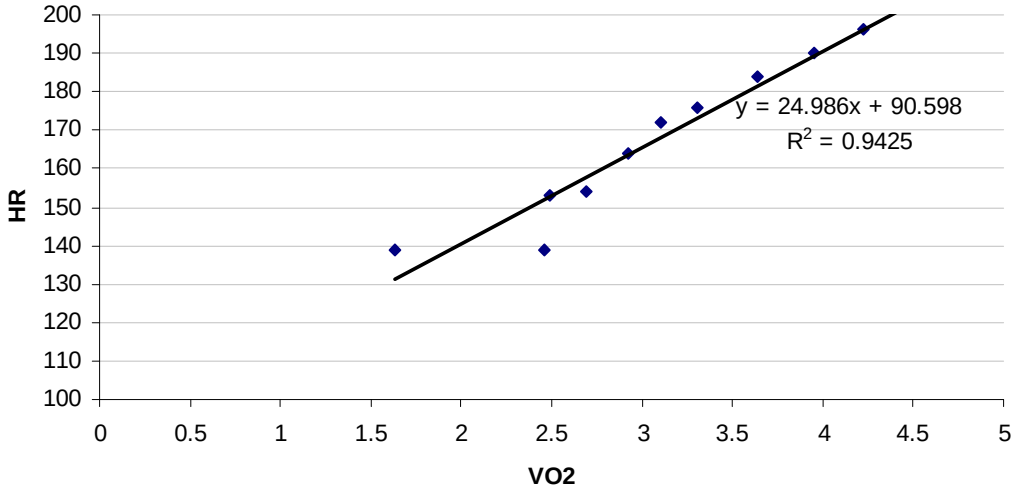

Figure 2: VO_2 plotted against heart rate responses from the graded running test, with VO_2 zones substituted into equation produced from best-fit line.

3.2 Best of 3 Set Tennis Match

3.2.1 Data Collection

Subjects arrived at the tennis centre having completed a food and fluid intake diary for the previous 48 hours. No dietary or lifestyle restrictions were placed on them. Upon arrival, they were asked to provide a urine sample, and then their nude body mass was measured using a set of precision balance scales. Subjects were also required to provide a small capillary blood sample. Blood samples were taken from either their 3rd or 4th finger on the non dominant hand. The finger was wiped clean with an alcohol wipe, wiped dry with a tissue, and the blood was drawn using an 'Accu-Chek Soft Clix-pro' sampling pen. The first drop of blood drawn was wiped clean with a tissue, and a 175µl capillary tube was then filled.

Subjects then attached a heart rate monitor (Polar S625X) to their chest, placed the heart monitor watch around their wrist, and these remained there for the duration of the match. Sweat patches (3M Tegaderm absorbent patches, area 10cm^2) were then applied to four sites on the subjects body; the midpoint of the right thigh between the knee and hip, the middle of the widest point of the right calf, the inside of the right forearm midway between the elbow and wrist, and the middle of the lower back, just above the short line. All sites were shaved with a hand-held plastic razor before applying the patch, ensuring the site was free of any hair that could prevent patch from staying on throughout the entire duration of the match. The patches were placed on the subject's body after each skin site was prepared by wiping with an alcohol wipe, washing with deionised water, and drying with a clean electrolyte-free swab gauze. Before the patches were applied, each patch (and all its accompanying wrapping) was weighed using a set of electronic scales (Amlab ACB 300, weighed to two decimal places). After the patches were applied, all the left over wrapping and packaging associated with the patch was weighed, giving the exact weight of the patch that had been applied.

The participants then completed a standard 5-minute match warm-up, which included players warming up groundstrokes, volleys and serves. They then played a best of three set match against a competitively matched opponent (Players were required to wear only shirts and shorts: no tracksuits were permitted, and opponents were matched as closely as

possible by Lawn Tennis Association rating and national ranking). Players were required to retrieve their own balls during the match. The heart rate monitor was started when subjects started their match and this began storing the heart rate data at 5-second intervals.

The temperature and humidity of the tennis centre was recorded on a digital meter (Radiometer Copenhagen). The subjects were allowed to consume any fluid that they wished during the match, however there were not permitted to consume immediately before or after the match. At the end of every change over their drinks bottle was removed and weighed using a set of electronic scales (Ohaus CS-2000 Compact) to find out exactly how much they had consumed. The time of each changeover was also noted. Also, a small sample of the beverage consumed on court was stored and analysed to discover its electrolyte content. Subjects were not permitted to spit out any of the fluid in their drinks bottles, or use it to wet their hair or face.

Once the match had been completed, subjects were then immediately taken back to the laboratory, where a three-minute post-match capillary blood sample was taken using the protocol outlined previously. The sweat patches were then removed using a pair of sterile forceps (a new pair for each patch), and were placed inside a plastic tube which was in turn placed inside a sterile urine collection tube (the empty tube had been weighed previously). This tube with the patch inside was then weighed, to obtain the volume of sweat that was absorbed by the patch. Care was taken to ensure that the researcher's hands did not touch any of the patches at any time to ensure that no contamination occurred. The patches were placed inside the tube so that the inside of the patch (that had been in contact with the skin surface) was facing towards the outside of the tube, to improve the chance of all of the sweat being removed during centrifugation.

The subjects then provided another urine sample prior to their final nude body mass being recorded. Subjects were instructed to towel themselves down before weighing to remove any excess sweat that would influence their weight.

The four sweat patches in their containers were then placed in a centrifuge (Jouan BR4), and spun at 5000 rpm for 10 minutes to separate the sweat from the patch. Once this had been completed, the sweat that was obtained from each patch was pipetted out and into

separate eppendorf tubes. A new pipette tip was used for each sweat sample to avoid contamination. The weight of the eppendorf tube and the sweat recovered was measured on the electronic scales so that the amount of sweat that was actually removed from the patch was measured. This information meant it was possible to see how much of the sweat absorbed in the patch was recovered after centrifugation. The sweat samples were labelled, and stored in the fridge until further analysis was carried out.

3.2.2 Urine Analysis

The pre and post match urine samples were separately poured into a measuring cylinder to accurately record the volume obtained. Then, a sample of the pre-match urine sample was pipetted into an eppendorf tube, and duplicate aliquots were tested for osmolality using the freezing point depression technique (Roebling Osmometer). Prior to the samples being analysed, the osmometer was calibrated using standards of known concentration and distilled deionised water blanks to ensure accurate results. The measurement process was then repeated with duplicate aliquots of the post-match urine sample.

To obtain the concentrations of the electrolytes in urine, 150µl of the pre-match and post-match urine samples were mixed in an eppendorf tube with 300µl of urine diluent (Radiometer Copenhagen). Different pipette tips were used for measuring each urine sample and the urine diluent. An aspirator tube was then placed in the eppendorf tube, and the sample analysed using an electrolyte metabolite laboratory (Radiometer EML 105, ion selective electrode method). These samples were measured for sodium, potassium and chloride concentrations.

3.2.3 Sweat Analysis

Sweat samples were also analysed using the EML105 analyser, previously validated as shown in Appendix A. For each sampling site, 150µl of sweat was pipetted into an eppendorf tube, and 300µl of diluent (Radiometer Copenhagen) was added. The samples were then aspirated through the Radiometer EML 105 analyser in the same way as the urine samples, and also measured for sodium, potassium and chloride levels. The drinks consumed on-court were also entered into the EML 105 analyser in the same way, and measured for sodium, potassium and chloride levels.

Sweat samples were also tested for osmolality using the freezing point depression technique (Roebling Osmometer), using the same procedure as for urine osmolality analysis.

3.2.4 Estimation of Fluid Losses

No account of respiratory water losses, or water losses due to substrate exchange was made when calculating sweat rates and whole body sweat losses from change in nude body mass. This is in accordance with previous research by Maughan et al. (2004) and Shirreffs et al. (2005). All the calculations are based solely on fluid intake and body mass change because the respiratory water loss and water loss through substrate exchange is likely to be small.

3.2.5 Blood Analysis

The pre and post match capillary blood samples were also analysed using the EML 105 analyser immediately after they had been collected, which analysed the sample for glucose, lactate, sodium, potassium and chloride. All safety measures were taken to prevent blood transmission between the subject, the researchers, and any work surfaces used.

3.2.6 Heart Rate Data

All heart rate data was uploaded from the heart rate monitors and was stored in the Polar ProTrainer application. It was then converted in spreadsheet form and transferred into Microsoft Excel for analysis.

3.2.7 Dietary Analysis

Subjects completed a two-day record of their food and fluid intake prior to the on-court test. This diet was then analysed for macronutrient content and electrolyte content to determine habitual intake. All dietary analysis was carried out using the Microdiet application (University of Salford, UK reference database).

3.2.8 Statistical Analysis

Pre and post match blood glucose, lactate, sodium, potassium and chloride values, as well as sweat electrolytes and osmolality concentrations, and pre and post match urine electrolytes and osmolality were analysed using paired T-tests. Values from different skin sites were compared directly by comparison of mean values. Correlation analysis was performed to investigate relationships between pre-match urine osmolality and fluid intake, sweat rate and drinking rate, and whole body sweat loss and total fluid intake and were completed using Pearson's correlation analysis. All statistical tests were undertaken using SPSS version 15.0 (Statistical package for the Social Sciences). All data are reported as Mean ± SD.

Chapter 4

Results

16 players completed the study. Each match was played indoors on a hard court surface. The mean temperature was 17 ± 2 °C, and the mean humidity was 42 ± 9 %. The mean playing time was 75 ± 14 minutes.

4.1 Blood Electrolytes / Metabolites

Table 3 shows blood glucose, lactate, sodium, potassium and chloride concentrations both prior to, and 3 minutes following a best of three set match.

Table 3: Blood glucose, lactate, sodium, potassium and chloride concentration pre and post match. No significance was observed in any measures between pre and post exercise. Values are mean ± SD for 16 subjects.

	Pre Match	Post Match
Glucose (mmol/l)	6.16 ± 1.04	5.69 ± 0.91
Lactate (mmol/l)	1.24 ± 0.32	2.03 ± 1.08
Na^+ (mmol/l)	139 ± 1.27	139 ± 2.44
K^+ (mmol/l)	4.2 ± 0.3	4.1 ± 0.7
Cl^- (mmol/l)	105 ± 1.61	105 ± 2.56

4.2 Fluid Intake

The mean fluid intake during a best of three set tennis match among 16 subjects was 1087 ± 625 ml. This ranged from 303 ml in a match lasting 58 minutes 45 seconds minutes to 2509 ml in a match lasting 1 hour and 17 minutes. The fluid intake observed for each player is shown in Figure 3. The mean fluid intake per hour was 947 ± 604 ml/h (range 311 to 2535 ml/h).

Fifteen out of sixteen players consumed tap water during their match. Analysis of the water revealed that it contained 4 mmol/l of sodium, 0 mmol/l of potassium, and 6 mmol/l of chloride. One player consumed a commercial diluted orange flavoured drink, and consumed 1226 ml throughout the duration of his match. This drink contained 8 mmol/l of sodium, 1.4 mmol/l of potassium, and 14 mmol/l of chloride.

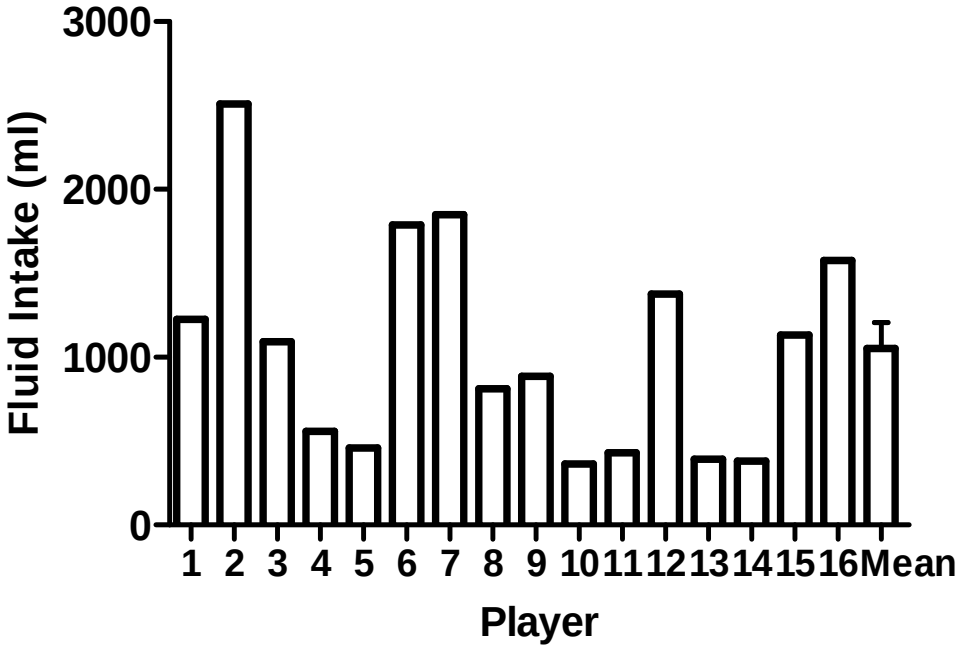

Figure 3: Fluid intake of 16 subjects during a best of three set tennis match, and mean ± SD fluid intake.

The mean fluid intake and mean percentage of total fluid intake that was consumed at each change of ends during the matches, as well as the mean playing time of each change of ends are shown in Table 4.

From this table, it can be seen that the studied players drinking habits were relatively varied. Also, players gain an opportunity to replace fluid between every 6 to 10 minutes. By looking at Figure 4, which demonstrates the mean fluid intake pattern across a match, players consumed the most fluid in the first change of ends. The level of fluid intake declined progressively until changeover 5, and remains relatively constant throughout the remainder of the match.

Table 4: Fluid intake, percentage of total fluid intake and mean playing time at each changeover. Values are mean ± SD for 16 subjects, apart from at changeover 9 (13 players), 10 (10 players), 11 (7 players), 12 (4 players), and 13 (1 player).

Changeover	Fluid Intake (ml) (Mean ± SD)	% of Total Intake (Mean ± SD)	Mean Playing Time (min) (Mean ± SD)
1	171 ± 127	16 ± 11	2.57 ± 1.08
2	114 ± 78	12 ± 5	8.38 ± 3.02
3	111 ± 77	11 ± 9	15.47 ± 4.35
4	101 ± 82	9 ± 4	23.03 ± 5.43
5	88 ± 86	7 ± 5	30.35 ± 6.21
6	130 ± 120	12 ± 6	36.09 ± 6.36
7	81 ± 83	6 ± 6	42.04 ± 6.53
8	87 ± 47	10 ± 7	50.43 ± 7.13
9 (13 players)	103 ± 74	8 ± 8	56.55 ± 7.51
10 (10 players)	85 ± 62	4 ± 4	62.36 ± 7.01
11 (7 players)	79 ± 79	3 ± 4	70.34 ± 8.43
12 (4 players)	109 ± 104	3 ± 5	80.14 ± 11.19
13 (1 player)	0	0	86.20

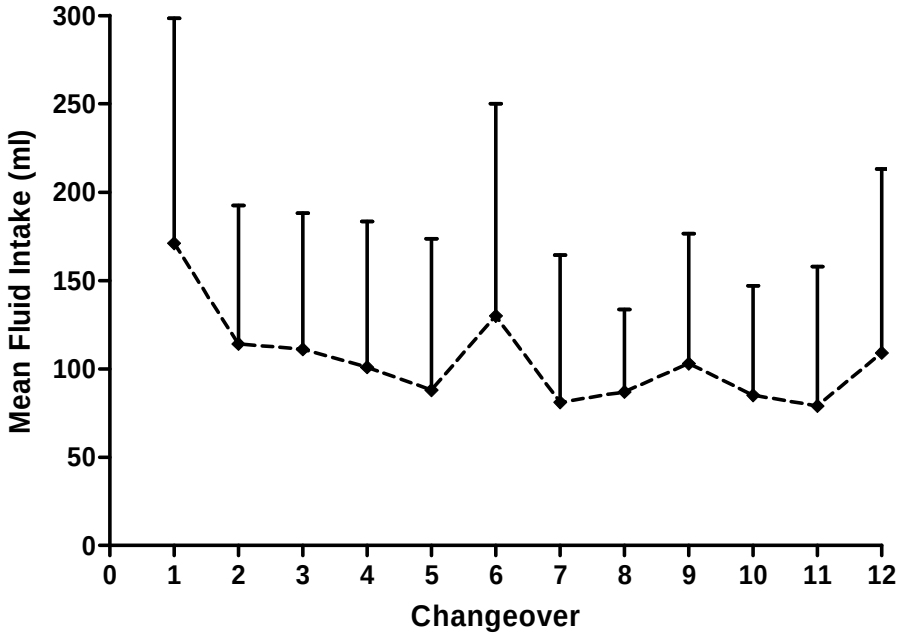

Figure 4: Graph showing mean fluid intake (ml) at each changeover across the duration of the matches. Values are mean ± SD for 16 players for the first eight changeovers, then 13 players at changeover 9, 10 players at changeover 10, 7 at changeover 11, 4 at changeover 12, and 1 player at changeover 13.

4.3 Body Mass Changes

Figure 5 shows the range of percentage body mass changes observed from pre match to post match for the 16 players. Body mass changes ranged from 1.6% dehydration, to 1.3% gain in mass. The average body mass change was a loss of 0.11 ± 0.55 kg. There was no significant difference in body mass between pre and post match ($p>0.05$), and it can be seen clearly from Figure 5 that a wide range of body mass changes occurred.

Figure 5: Body mass changes across a match. Values shown are the percentage of body mass lost from pre to post match.

4.4 Whole Body Sweat Loss and Sweat Rates

The mean whole body sweat loss during the best of three set tennis match was 1219 ± 417 ml. This ranged from 463 ml to 2298 ml. The mean whole body sweat rate was 0.72 ± 0.26 l/h. The range observed was between 0.43 and 1.28 l/h.

4.5 Electrolyte Concentration, Osmolality and Local Sweat Rates at Sample Sites

Table 5 displays the sweat electrolyte concentrations, sweat osmolality, and sweat rate at each of the four sampling sites. Sweat sodium and chloride concentrations were significantly higher at the back site than any other sampling sites ($p<0.05$). No significance was observed in sweat osmolality between sampling sites. The sweat rate from the back site was significantly greater than from the other three sites, while the sweat rate from the forearm was also significantly greater than that obtained from the calf site ($p<0.05$).

Table 5: Sweat electrolyte concentrations, osmolality and local sweat rate at each of the four sweat sampling sites. Values are Mean ± SD for 16 subjects. * indicates significant differences between back and all other sites, $p<0.05$. ♦ indicates significant difference to calf site ($p<0.05$).

	Na^+ (mmol/l)	K^+ (mmol/l)	Cl^- (mmol/l)	Osmolality (mOsmol/kg)	Sweat Rate $mg \cdot cm^{2} \cdot min^{-1}$
Forearm	37 ± 14	4.9 ± 2.0	31 ± 13	111 ± 21	0.54 ± 0.19♦
Calf	38 ± 14	5.0 ± 2.0	34 ± 14	118 ± 26	0.36 ± 0.18
Thigh	38 ± 12	4.5 ± 2.0	34 ± 12	112 ± 13	0.43 ± 0.23
Back	49 ± 18*	4.5 ± 1.0	46 ± 19*	125 ± 13	0.94 ± 0.37*
Mean†	41 ± 15	4.7 ± 1.7	36 ± 15	117 ± 35	0.57 ± 0.19
Range	23 to 79	2.8 to 7.0	19 to 73.5	96 to 160	0.28 to 0.94

† - indicates an equally weighted mean value from the four sampling sites.

4.6 Urine Measures

Table 6 displays the mean urine volumes, electrolyte concentrations, and osmolality obtained in samples collected before and after the matches.

Table 6: Urine volume, electrolyte concentration, and osmolality pre and post match. * indicates significant difference between pre and post match urine volume ($p<0.05$). Figures are mean ± SD.

	Pre Match	Post Match
Volume (ml)	44 ± 32	94 ± 63*
Na^+ (mmol/l)	115 ± 54	52 ± 38
K^+ (mmol/l)	49 ± 26	54 ± 32
Cl^- (mmol/l)	139 ± 74	107 ± 54
Osmolality (mOsmol/kg)	788 ± 158	695 ± 207

Two players had a pre-exercise urine osmolality of above the previously stated threshold for dehydration of 900 mOsmol/kg (Shirreffs 2003). Four more players had pre-exercise urine osmolality readings of close to this threshold. The range of readings was 366 to 1099 mOsmol/kg.

4.7 Net Fluid Balance

There was a mean net body fluid deficit of 132 ± 83 ml from pre to post match (Fig.6). Thus, players ingested sufficient fluid to replace 89 ± 47% of fluid losses due to sweating.

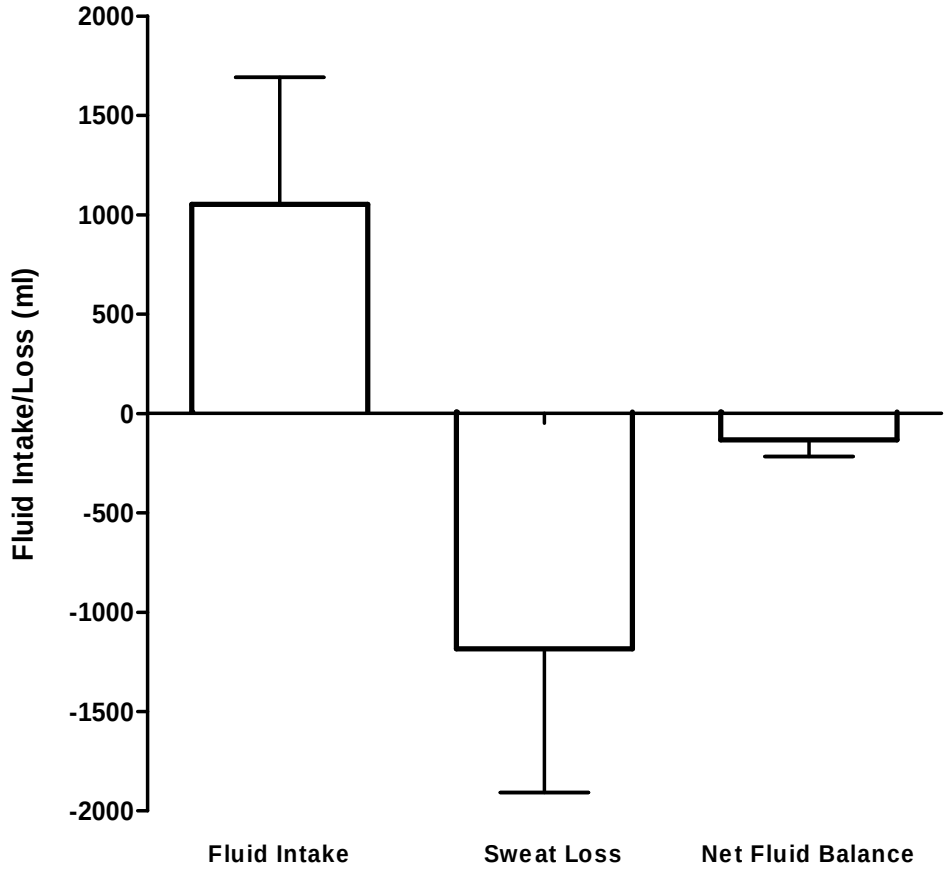

Figure 6: Mean fluid intake, mean whole body sweat loss, and resultant mean net body fluid balance of players following a best of three set match indoors. Values are mean ± SD.

There was a significant relationship ($p < 0.05$, $r = 0.51$) between whole body sweat loss and total fluid intake during the matches (Fig.7), however this is not clearly visible. However, no relationship existed between sweat rate and drinking rate ($p > 0.05$, $r = 0.21$, Fig.8), or total fluid intake and pre match urine osmolality ($p > 0.05$, $r = 0.06$, Fig.9).

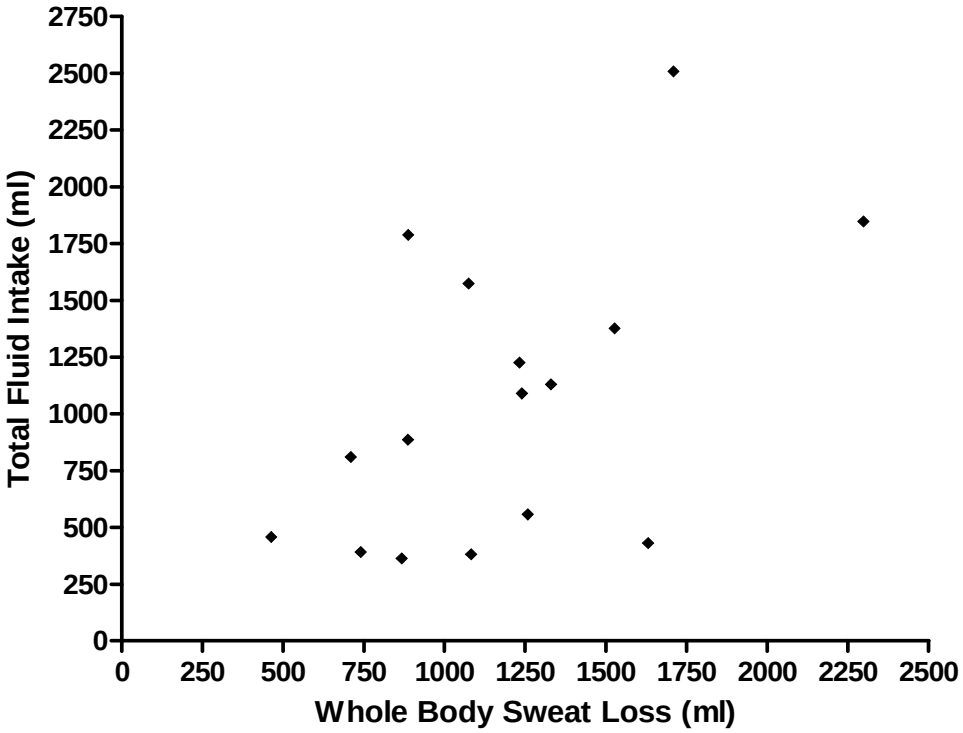

Figure 7: Relationship between whole body sweat loss (ml) and total fluid intake (ml). A relationship was observed between these two variables ($p<0.05$).

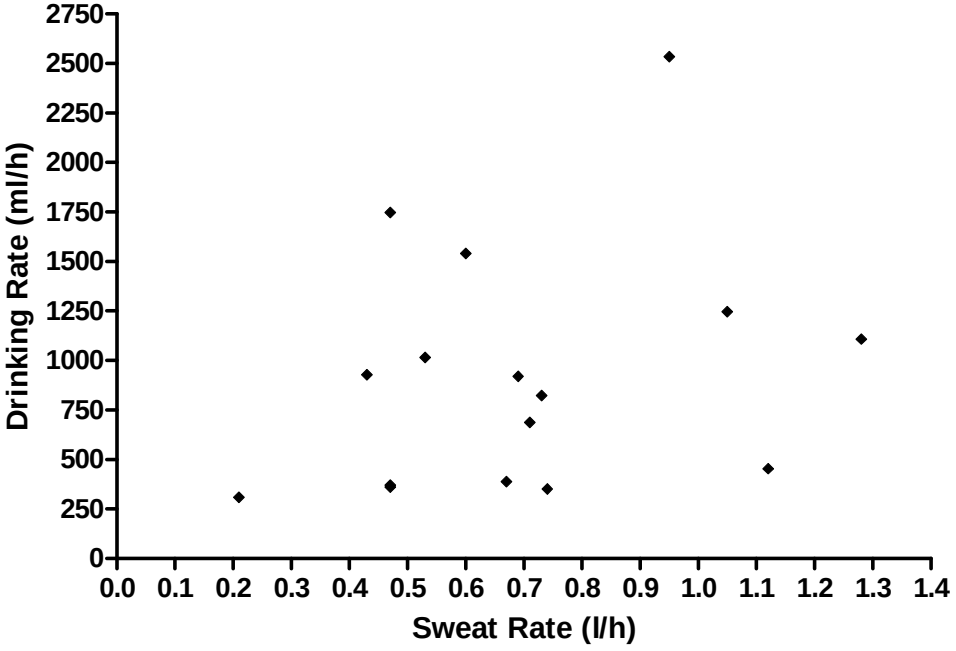

Figure 8: The relationship between sweat rate (l/h) and on-court drinking rate (l/h). No significant correlation was observed ($p>0.05$)

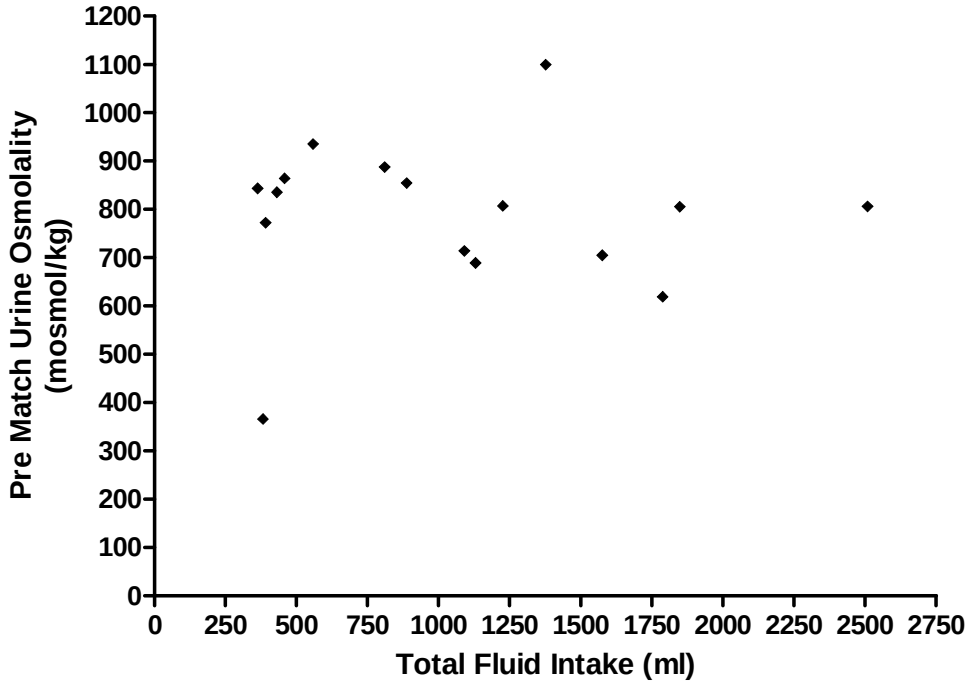

Figure 9: The relationship between total fluid intake (ml) and the pre-exercise urine osmolality. No correlation was observed ($p>0.05$).

4.8 Electrolyte Losses and Electrolyte Balance

The mean ± SD electrolyte losses (from sweat and urine losses) observed during the matches, as well as the mean dietary electrolyte intakes are displayed in Table 7. Electrolyte ingestion was calculated from each player's habitual dietary intake on two days, and averaged to give mean values.

Table 7: Sweat and urine electrolyte losses, and dietary electrolyte intake, shown as Mean ± SD.

	Sodium (g)	Potassium (g)	Chloride (g)	Energy Intake (kcal)
Sweat + Urine Loss	1.12 ± 0.46	0.23 ± 0.15	1.55 ± 0.70	N/A
Dietary Intake	2.75 ± 1.06	2.22 ± 0.67	4.32 ± 1.74	1752 ± 347

The net electrolyte balance is shown in figures 10 to 12 with a net positive sodium balance, a net positive potassium balance and a net positive chloride balance based on habitual dietary intake versus electrolyte losses during the match play.

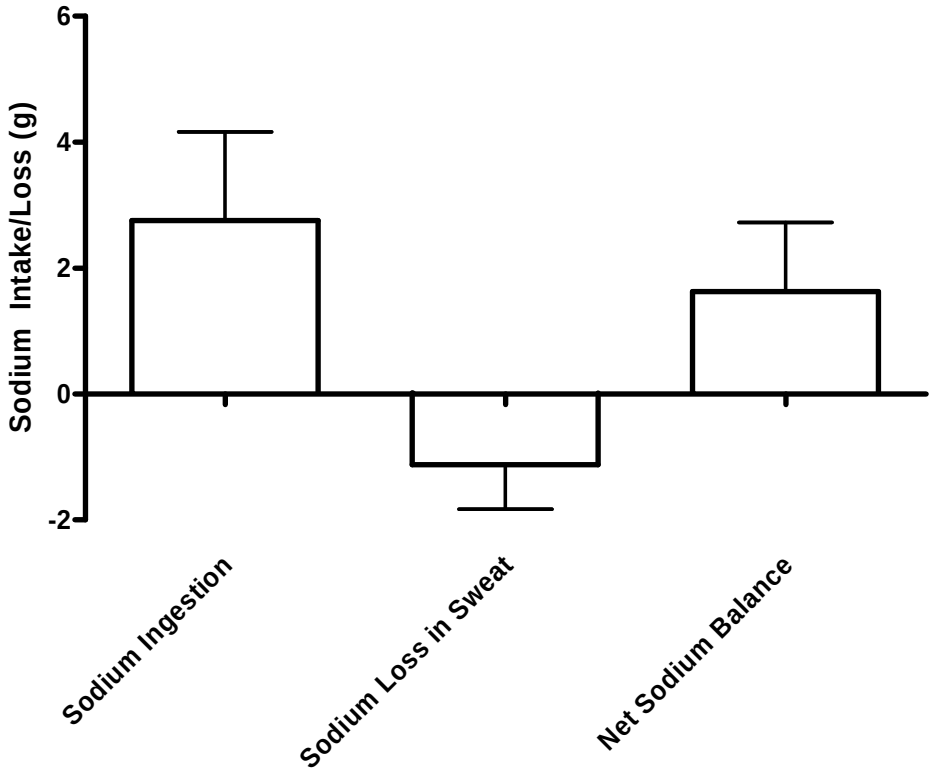

Figure 10: Sodium ingestion (daily diet plus ingestion during match), sodium loss in sweat, and resultant net sodium balance of 16 players. Values are mean ± SD.

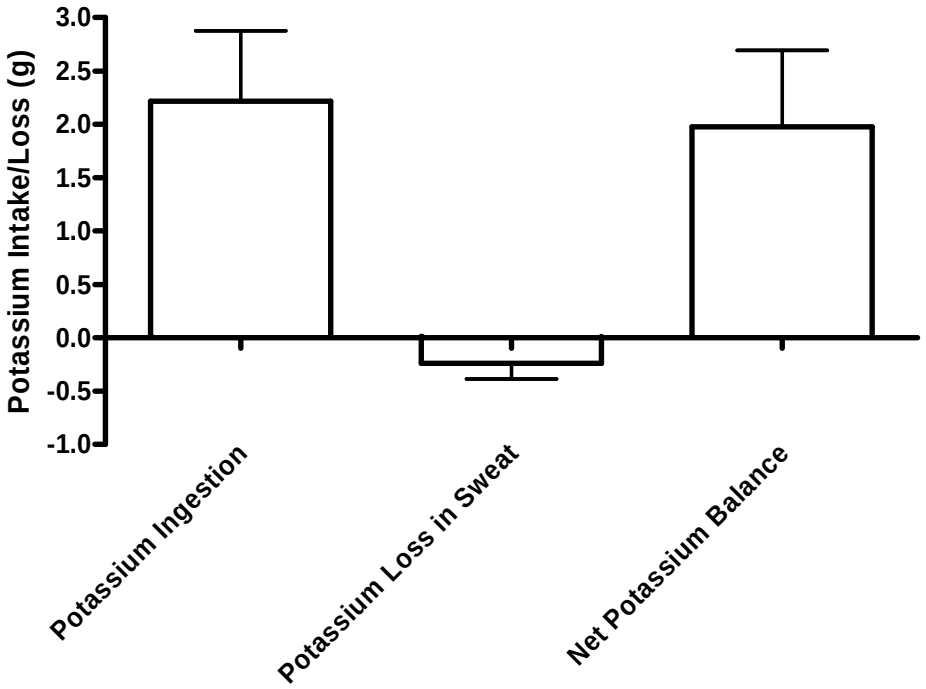

Figure 11: Potassium ingestion (daily diet plus ingestion during match), potassium loss in sweat, and resultant net potassium balance of players. Values are mean ± SD.

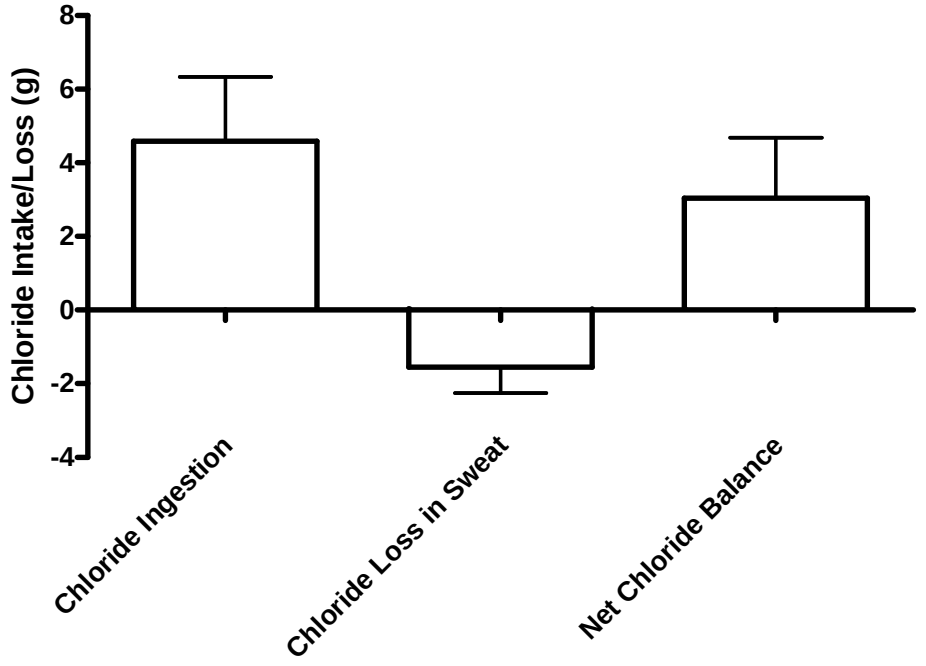

Figure 12: Chloride ingestion (daily diet plus ingestion during match), chloride loss in sweat, and resultant net chloride balance of players. Values are mean ± SD.

Given that 15 out of 16 players ingested water only (containing 4 mmol/l sodium), then for the tennis match period alone there would have been moderately large electrolyte deficits.

4.9 Dietary Analysis

The mean carbohydrate fat and protein contributions to the player's daily dietary intake are shown in Figure 13. This amounted to a mean intake of 209 g of CHO, 73 g of fat, and 69 g of protein per day. The mean total energy intake was 1752 ± 347 kcal.

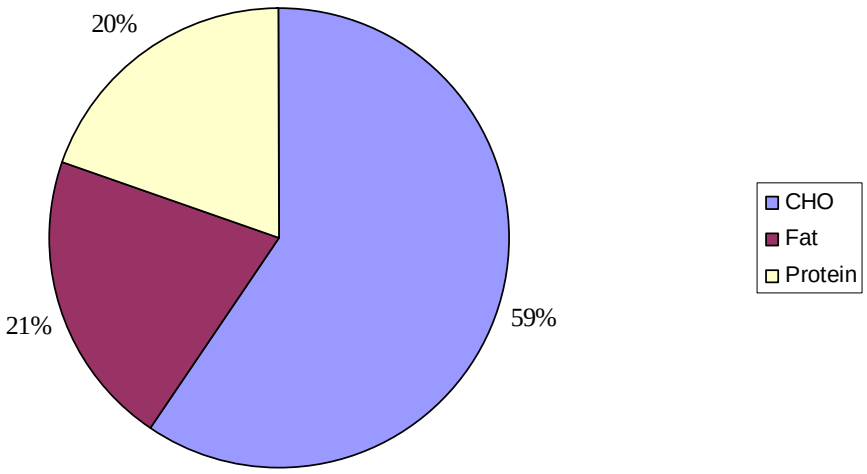

Figure 13: Dietary analysis of 16 tennis players. Values are the mean of a two-day recording period, and show percentage contribution of carbohydrate, fat and protein to total Kcal intake using the Atwater factors.

4.10 On-Court Match Intensity

Heart rates on court ranged from 75 bpm to 192 bpm indicating intensities in the range from rest to almost maximal intensity.

Players spent on average 74% of playing time in a low intensity zone. They spent 25% at a high intensity, in a zone above the ventilatory threshold, and they spent 1% in a zone of near maximal effort However, only two of sixteen players spent any playing time in the maximal intensity zone: one player spent 12% in this zone, while the other spent 2%. This is shown in Figure 14.

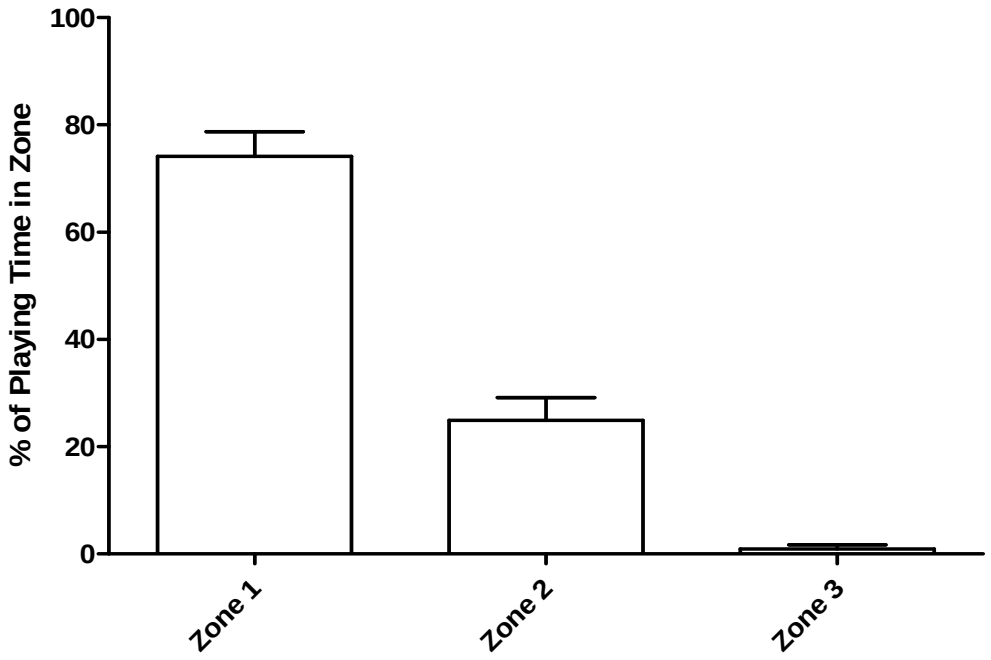

Figure 14: Graph showing the percentage of playing time spent in Zone 1 (low intensity), Zone 2 (high intensity), and Zone 3 (maximal intensity) effort zones. Values are mean ± SD for 16 subjects. The heart rate zones ranged between <123 bpm to <174 bpm for Zone 1, 123-180 bpm to 174-196 bpm for Zone 2 and >167 bpm to >197 bpm for Zone 3.

The mean heart rate over the course of a match is shown in Figure 15. The mean percentage of maximum heart rate was 71.4 ± 6.11%, ranging from 62.5% to 85.1%.

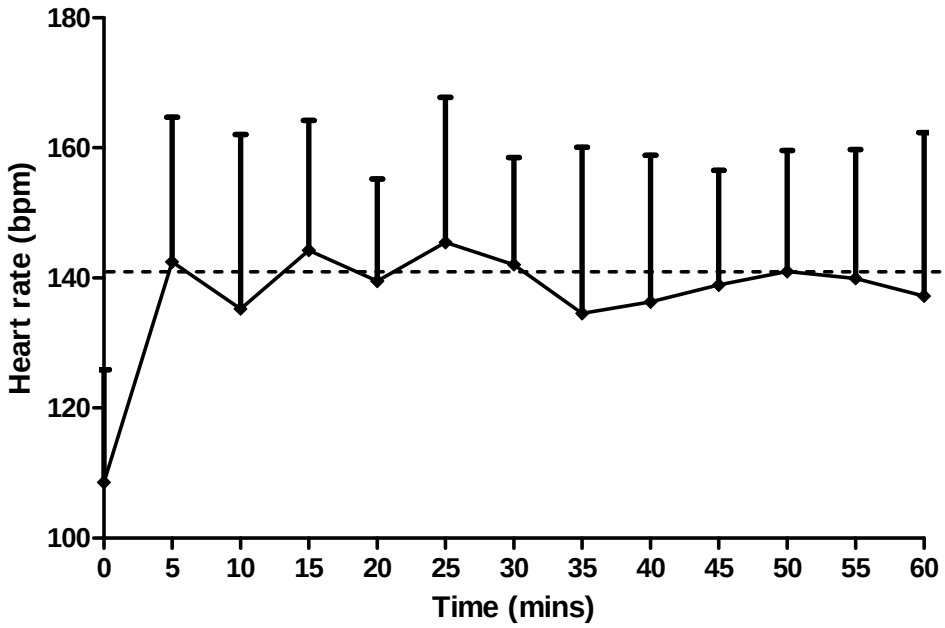

Figure 15: Graph showing the mean heart rate over the course of a match. Values are Mean ± SD. The mean heart rate corresponding to the first ventilatory threshold is also displayed.

Chapter 5

Discussion

This study characterised the fluid and electrolyte balance of nationally ranked male tennis players during indoor match play. The key observations in the present work were that the players were largely involved in moderate intensity exercise and sweat rates similar to those expected in a warm environment in tennis at this intensity (Bergeron et al. 1995a) were observed in the majority of players. Also, the sweat rates observed in the present study (0.72 ± 0.26 l/h) fit in with those observed by Galloway & Maughan (1997), who showed that cycling at 70% brought about sweat rates of 0.65 l/h and 0.78 l/h at 11°C and 21°C respectively. A wide range of sweat electrolyte concentrations were observed and consumption of water was the norm during match play. Urine osmolality measurements indicated that some players were moderately dehydrated prior to, and upon completion of match play. Varied fluid volume intakes were observed, but on average the players replaced 946 ± 602 ml/h, which approximately equated to sweat losses (89 ± 47% replacement) without any change in plasma sodium concentration.

Only a handful of studies have examined electrolyte balance and fluid intake strategies in tennis players, and this is the first to examine these factors in an indoor environment, where the temperature did not exceed 20°C on any occasion. It is also the first to closely analyse players' on-court fluid ingestion patterns.

5.1 Pre-Match Hydration Status

Several players appeared to be hypohydrated, or were close to being so, prior to their match. The mean pre-exercise urine osmolality was 788 ± 158 mOsmol/kg; however two players had results of 935 mOsmol/kg and 1099 mOsmol/kg respectively, with both being greater than the 900 mOsmol/kg level for dehydration stated by Shirreffs et al. (2003). As well as two players commencing their match dehydrated, seven other players had results greater than 800mOsmol/kg, approaching the threshold for dehydration, which is concerning but not entirely surprising, as it has been reported before by Bergeron et al. (2006) and Hornery et al. (2007) that tennis players had a tendency of commencing match play hypohydrated. It is not clear whether this is a pattern evident in other sports as well, but Maughan et al. (2004) noticed that some footballers were mildly hypohydrated, and one severely so (1254 mOsmol/kg), prior to a 90 minute training session, while Palmer & Spriet (2008) showed that a group of 44 junior ice hockey players were largely on the verge of hypohydration prior to an intense one hour practice

session. An athlete who commences exercise in a hypohydrated state is at an increased risk of suffering heat illness, and will more than likely not perform at optimum levels (Sawka & Pandolf 1990). Indeed, Hornery et al. (2007) suggested that even technical elements of the service action were affected by adverse physiological conditions, including hypohydration. However, the environmental conditions during which players performed in the present study are unlikely to pose any threat to thermoregulation. Studies have recommended that individuals drink approximately 500 ml, or 6-8 ml per kilogram body mass, of water or a carbohydrate-electrolyte drink two hours before they go on court (Maughan & Shirreffs 1997; Shirreffs et al. 2006), or in the hour before (Casa et al. 2000). Indeed, the ACSM's 2007 position stand (Sawka et al. 2007) suggests 5-7 ml per kilogram body mass slowly consumed within four hours of the beginning of a match. This should improve hydration status pre-training session or match, but this may not always be possible if players train or compete early on in the day. However, players should aim to adhere to these guidelines wherever possible to avoid commencing a match in a hypohydrated state.

Despite some players commencing exercise in a hypohydrated state, they did not consume more fluid during the match than the other players, nor did they have a different sweat rate. Also, somewhat surprisingly, no relationship was observed between pre-exercise urine osmolality and fluid intake, as it might be suggested that if a player started a match dehydrated, he should consume more fluid to try and correct this. However, it has been demonstrated that thirst is not a good indicator of body water status (Greenleaf 1992), so a small fluid deficit could exist without the player's knowledge.

5.2 Match play Intensity and Environmental Conditions

In an entirely unrestricted indoor environment, match play consisted of largely moderate intensity exercise, below the ventilatory threshold, with a quarter of the time spent above the ventilatory threshold. This can be compared to the work of Seliger et al (1973), who suggested tennis included 88% aerobic and 12% anaerobic contributions, and Fox et al. (1979), who suggested that the aerobic contribution was as low as 30%. However, the data from the present study could be considered more reliable as it does not place any restrictions on the length of matches; if the length is controlled, there is a risk of the players not having the opportunity of reaching their highest effort levels. In the study by

Seliger et al. (1973), the analysed match-play duration was ten minutes, which is not long enough to get a full profile of exercise intensity. The range in time spent above the ventilatory threshold in the present study could be attributed to differing effort levels on the player's part; however, an important factor is a player's or opponents playing style. If a player has an aggressive style and regularly serves and volleys or approaches the net, the points will be shorter, and may prevent the player's heart rate from increasing as high as if they were regularly competing in long rallies. However, often rushing to the net could cause heart rate to increase, whereas good baseline play could result in less strenuous work being performed, and a lower heart rate. Bernardi et al. (1998) showed that mean and maximal heart rates and VO_2 scores of baseline players were higher than whole-court players and more aggressive players on a clay court surface, and showed that the baseline players reached on average 59% of their $VO_{2\ MAX}$, suggesting that long-term cardiovascular adaptations could be achieved, since to maintain or develop cardiorespiratory fitness, exercise should be between 50 and 85% $VO_{2\ MAX.}$ This was confirmed by Fernandez et al. (2006), who suggested that players who were considered attacking or more aggressive were found to have lower VO_2 values than baseline players. This suggests a relationship between rally length and exercise intensity. Another important factor is the court surface; the present study used a fast indoor hard court surface, which tends to induce shorter rallies, and it is possible that a slower outdoor hard court or clay court would provide results with a higher intensity. However, this will obviously depend on the individual, as some players will naturally play with a higher intensity than others will.

The conditions in the present study were similar to the conditions found in many national indoor tournaments; and probably fairly similar to those tournaments held outdoors in Great Britain, except with the absence of any wind. The conditions were probably slightly lower than indoor conditions in summer months, since the data collection took place between October and January. However the temperatures and humidities were most probably lower, and less challenging than the majority of tournaments held overseas, both indoors and outdoors, so the conditions will have been more comfortable for the players involved.

5.3 Sweat Sodium Concentrations and Total Sodium Losses

The vast majority of sweat electrolyte concentrations observed in the study fell into the normal physiologic ranges for sweat electrolyte concentrations of 20 to 80 mmol/l for sodium, 4 to 8 mmol/l for potassium, and 20 to 60 mmol/l for chloride (ranges stated by Shirreffs et al. 1997), apart from one reading of 93 mmol/l at the back site for one player. Sweat sodium concentrations observed in this study (41 ± 15 mmol/l at 17 ± 2°C) are fairly similar to those results observed in a range of sports, including professional footballers (30 ± 19 mmol/l at 32 ± 3°C (Shirreffs et al. 2005), and 49 ± 12 mmol/l at 24 to 29°C (Maughan et al. 2004)). However, the observed results were slightly higher than observed in cooler (5°C, Maughan et al. 2005) conditions in professional footballers, and in elite University tennis players outdoors at mean 32.2 ± 1.5°C (Bergeron et al. 1995). That the sweat sodium level was slightly higher than in other tennis players who train and compete regularly in warmer environments suggests that several players in the present study were not as well heat acclimated as the American College players studied by Bergeron et al (1995a). The more exposure a player has at warmer environmental conditions, then both sweat sodium concentration and sweat osmolality are reduced for a given sweat rate, through increased reabsorptive ability of the eccrine sweat duct (Buono et al. 2007). Indeed, one player experienced a mean sweat sodium concentration of 78.5 mmol/l, which is at the top end of the normal physiologic range. This was achieved indoors at a temperature of 20°C, humidity of 56%, and during a match lasting 57 minutes, which certainly are not challenging environmental conditions. This player experienced the second largest total sodium loss (1.6 g), in the shortest match duration, however he managed to maintain his body weight from pre to post match. This player's sweat rate was only 0.53 l/h and this low sweat rate, coupled with a high sweat sodium concentration, suggests that he was not heat acclimated. This player would struggle with both sodium depletion and an inability to lose heat through evaporation if he played in warm humid conditions, whereas a more heat acclimated player would have a lower sweat sodium concentration, helping to delay sodium depletion, as well as have the ability to dissipate heat more effectively through a higher sweat rate.

Players in the present study understandably lost much less sodium during their match compared to tennis players who suffer heat cramps in warm conditions (Bergeron 2003), and American footballers with same heat cramping conditions (Stofan et al. 2005).

However, the sodium losses were comparable to non-cramp afflicted American footballers in between 22.7°C and 30.8°C (Stofan et al. 2005). Three players experienced sodium losses that were dangerously close to their daily dietary sodium intake, in fact one player recorded a daily dietary sodium intake of 1.91 g, yet lost 1.75 g of sodium during his match. If this player was to compete in two matches in the same day or on consecutive days throughout a week or two-week period, with the same dietary sodium intake, then there is a considerable risk of him experiencing problems from a chronic sodium deficit. Players should be made aware of their dietary sodium intakes and potential sodium losses during match play, and be able to make the necessary increases in dietary sodium intake in days leading up to the start of a tournament, during a tournament, and also on-court through food and fluid ingestion. The ingestion of a carbohydrate-electrolyte drink both off and on-court should help to maintain sodium levels if a player is due to compete in multiple matches on the same day. Bergeron et al. (1995a) demonstrated that when three matches were played on consecutive days, there was progressive depletion in plasma sodium levels from the previous day, and this trend continued onto a fourth day.

If the players involved in the present study went into a tournament situation, where they are required to compete more than once, they should increase their dietary sodium intake to combat the sodium depletion that would occur. As mentioned before, sodium depletion has been shown to be the primary cause of heat cramps, which can be disastrous during a match, and increasing sodium intake becomes even more important when the tournament is played at higher temperatures and humidities. If consuming a full meal in between matches in not comfortable, then players should try to consume salted snacks, to help maintain sodium levels (Sawka et al. 2007), and consuming a carbohydrate-electrolyte drink at changeovers will help further.

The wide range of standard deviations observed in this study demonstrates the large variability that exists between individuals with respect to sweat electrolyte concentrations, and means that looking at the range of results reveals more than simply looking at standard deviations. This allows one to identify specific individuals who may warrant further investigation. Consequently, it is difficult to make a general conclusion on the sweat sodium concentrations of teams or groups recorded, or make direct comparisons to other groups or teams. However, it does provide support to the theory

that individual advice is vital (Maughan et al. 2005, Palmer & Spriet 2008), and should not be aimed at groups or teams of players because recommendations that might help one player could have no affect on another.

Overall, the sweat electrolyte concentrations of these elite British University tennis players observed fell into the normal reported ranges; however they were slightly higher than recordings in American College tennis players at warmer temperatures. This suggests a poorer level of heat acclimation in some of the players in the present study, not surprising considering the amount of training and competitive tennis played indoors, and the climate in the UK when playing outdoors. Since data was collected between October and January, the average daily temperature would be lower than that in summer, so electrolyte losses could have been greater in these players if data collection was carried out in summer months. Total sodium losses for a small number of players came close to exceeding dietary sodium intake, and players must be informed on how to increase sodium intake both in the diet, before and between matches, and on-court during a match.

5.4 Sweat Rates & Fluid Intake

The average whole body sweat rate of 0.71 ± 0.25 l/h is lower than recorded in professional footballers in cool conditions of 5°C (Maughan et al. 2005), and at hot conditions of between 24 and 29°C and 32 ± 3°C (Maughan et al. 2004; Shirreffs et al. 2005), American footballers at between 22.7°C and 30.2°C (Stofan et al. 2005), endurance athletes in comfortable (20.5 ± 0.7°C) conditions (Passe et al. 2007), and tennis players in hot (32.3 ± 1.5°C) environments (Bergeron et al. 1995). The sweat rate in the present study is also over 1 l/h lower than observed in American footballers and runners across two days of training at 31.3 ± 0.9°C (Fowkes Godek et al. 2005). The uniform worn by American footballers could increase sweat rates: Gavin (2003) has suggested that increasing the level of clothing imposes barriers to heat transfer and evaporation from the skin surface. The low whole body sweat rate observed in the present study could be a direct result of the undemanding environmental conditions, but could again be an indicator of a low level of heat acclimation in the players involved, as increased heat acclimation causes an increased sweat rate, which improves heat loss via evaporative cooling. Results are also lower than the 1.8 ± 0.1 l/h observed in elite junior ice hockey players during an intense practice session at 13.9°C. However, the low sweat

rate in the present study is more likely to be linked to players only being allowed to wear t-shirt, shorts, socks and shoes in a mean temperature of 17°C, and with relatively lower exercise intensity, so the player's state of acclimation would be less of a factor.

The mean fluid intake of 1087 ± 625 ml, at an average match length of 75 ± 14 minutes, is similar to observations in professional footballers during 90 minute training sessions in both cool (Maughan et al. 2005) and warm temperatures (Maughan et al. 2004; Shirreffs et al. 2005), but distinctly lower than tennis players in warmer outdoor environments (1700 ± 500 ml at 32.2 ± 1.5°C). On average, players replaced 89 ± 47% of their whole body sweat loss in the present study, which is larger than reported by Elliot et al. (1985), where the mean fluid intake across one 60 minute singles match was 250 ml, representing only 27% of the total fluid loss. However, results were similar to Hornery et al. (2007), where players replaced 77% and 89% of total fluid loss during hard and clay court singles matches. This suggests that the players in the present study had good drinking habits to replace such a large proportion of sweat losses.

Despite players on the whole managing to replace their sweat loss, these cases must be addressed individually. The range in fluid intake observed in the present study was massive, and importantly three players consumed significantly less fluid than they lost through whole body sweating, indeed one only consumed 431 ml, but lost 1631 ml through sweating (so only replaced 26% of fluid losses) and incurred a 1.6% loss of body mass. If the three players mentioned were competing at higher environmental temperature conditions, or during longer matches in the same conditions, then by replacing only a fraction of the fluid they lost they will experience great body mass deficits. Dehydration of over 2% body mass can reduce performance by impairing power production (Coyle et al. 2004), and cognitive function (Gopinanthan et al. 1998) and these are likely to occur in tennis if players fail to adequately rehydrate. Hornery et al. (2007) also suggested a negative impact of dehydration upon service technique, whereby they showed that the consistency in the height of the throwing arm at ball release was inversely correlated with both progressive match time and body mass deficit. If these players competed at much warmer and humid conditions with the same level of rehydration, then they are at serious risk of developing heat illness.

Due to the nature of tennis, match play always provides opportunities for regular rehydration and the changeovers appear to have been used effectively in the players who prevented a body fluid deficit. However, the observation that some players failed to replace their sweat losses with unrestricted fluid intake supports the presence of "voluntary hypohydration" observed in many different sporting environments (Passe et al. 2007; Palmer & Spriet 2008). With the lack of any severe environmental conditions, there appears to have been no great strain upon most of the players involved in the present study, but the players who failed to replace anywhere near their fluid losses should be made aware of the implications. There did not appear to be a relationship between poor fluid replacement and success in the match. All other studies looking at dehydration in tennis players have reported a significant body mass loss from pre to post match, however this was not the case in the present study (only a mean body mass loss of 0.15%) Elliot et al. (1985) showed an average fluid loss of 0.91 kg (1.3% body weight) after one singles match outdoors at WGBT 21.5°C, while Bergeron et al. (1995a) reported a mean body mass loss of 1.8% following 2 singles matches and 1 doubles match on the same day, but this was to be expected because of the greater amount of match play in the study, and the more environmentally challenging conditions (32.2 ± 1.5°C). Hornery et al. (2007) demonstrated an average 1.05% body mass loss following one match on hard courts, and 0.32% following one match on a clay court. It could be possible that the lack of any severe environmental conditions, and the short match duration may have prevented any significant body mass loss over time in the present study, but most players tended to replace, or nearly fully replace their sweat losses. However, the issue that some players failed to replace even half of their whole body sweat loss, and experienced a fairly large body mass deficit during a match lasting little over an hour suggests that education of these players is required.

5.5 Drink Choice

The drink choice employed by players also raises an important point regarding players' attitudes towards hydration during match play. Of the 16 players observed, 15 players chose to drink water during their match, and the one player who did not only consumed a commercial orange flavour concentrate drink, which was diluted down with water. This could suggest that this group of players made poor beverage choices when it comes to rehydration strategies during matches, and they fail to take into account any electrolyte

depletion that they could suffer when they compete. Alternatively, it could be that they realised they were playing one match in a non-extreme environment, they were unlikely to experience any problems, and were satisfied that consuming water would be sufficient. If players went into repeated matches lasting two and half hours plus and consumed only water, especially in more challenging environmental conditions, they could put themselves at risk of developing severe dehydration, as well as experiencing muscle cramping and possibly induce hyponatremia in extreme cases if excess fluid was consumed.

Players should as a matter of habit consume a carbohydrate-electrolyte drink during match play, because the benefits of replacing the sodium lost in sweat, as well as water are clear. With electrolyte ingestion, voluntary fluid intake is greater, while fluid loss in urine is significantly lower for a given fluid intake volume (Maughan & Leiper 1995; Nose et al. 1988), and the ingested sodium helps to maintain plasma sodium levels, preventing heat illness. Bergeron et al. (2006) also showed that when tennis players consumed a carbohydrate electrolyte drink during a match, they experienced a lower core body temperature than when the consumed water. These factors represent a clear benefit to the hydration status of a player. The ingestion of carbohydrate in fluids ingested during performance may provide an improvement in skill performance. Results are not conclusive, but Bottoms et al. (2006) showed that fewer squash shots missed a scoring zone during a skill test when fatigued if carbohydrate had been ingested during the fatiguing exercise. Studies in other sports such as soccer have shown that carbohydrate ingestion can improve skill in tests such as soccer passing tests and shooting tests (Ali et al. 2007). However, Mitchell et al. (1992) observed no skill benefit when testing serve velocities, shuttle run tests, and counts of serve percentages and unforced errors during three hour tennis matches. Therefore, ingestion of carbohydrate is at worst likely to have no impact upon skill performance but at best could improve skill retention when fatigued.

In support of the players drinking strategies, they maintained their serum sodium levels well. The mean serum sodium level was maintained pre to post match even though fifteen out of sixteen players drank water; 139 mmol/l pre match to 139 mmol/l post match. Therefore, consuming water was sufficient in maintaining serum sodium levels from pre to post match overall, suggesting that on the whole water intake matching sweat losses does not result in a reduction in plasma sodium concentration under the environmental

conditions in the present study. This could be due to the short length of matches in the present study, and in more extreme environmental conditions, or if more than one match was being played and larger were fluid volumes were ingested, then some level of sodium depletion may have been observed. This would need to be addressed accordingly, through increases in dietary sodium and/or electrolyte ingestion.

5.6 Fluid Intake Patterns during Match-Play

No specific pattern existed in terms of whether players "front loaded" or "back loaded" with fluid during a match. Each player seemed to have their own individual pattern of fluid consumption, with some consuming the majority of their fluid totals in the first half of the match, some consuming more fluid towards the end of the match, and some maintaining a consistent fluid intake at every changeover. Several players failed to consume any fluids at certain changes of ends, demonstrating poor habits, and players should be made aware of the effects of fluid loss and dehydration to prevent these.

Players have very regular opportunities to consume fluid during matches; in this study changeovers occurred on average every 6 to 10 minutes, therefore a player not consuming any fluid and suffering dehydration is self-inflicted, more so than in sports such as football, rugby and athletics where breaks are not regular. No other study has attempted to monitor fluid intake at each changeover, so it is difficult to suggest optimal intakes at certain points in matches, but it could be advised that players maintain an even fluid intake throughout the course of a match, or front load with fluid in the first half of a match. Maintaining an even fluid balance throughout a match can be recommended from consideration of gastric emptying rates, as consuming too much of a carbohydrate-electrolyte drink at one changeover could cause gastrointestinal discomfort (Febbraio et al. 1996). Studies have adopted a "front-loading" method of sorts in investigations of performance effects: for example Anantaraman et al. (1995) studied the difference in performance between a sweetened placebo and a 10% glucose solution, and gave 300ml immediately prior to exercise, and then a smaller amount every 15 minutes during 60 minutes of cycling at 90% $VO_{2\ MAX}$. Gastric emptying studies would suggest that if fluid ingested during exercise is to benefit the player before the end of the match then the player would need to ingest this fluid early on particularly in shorter matches.

Front-loading with a carbohydrate-electrolyte drink during a match would allow a greater carbohydrate store at a player's disposal for latter stages of matches, but may cause gastrointestinal discomfort during the earlier parts of matches if the carbohydrate content was too great. Coyle (2004) suggests that athletes should aim to consume enough fluid to finish exercise with up to a 2% reduction in body weight, provided that the drinking pattern minimises the volume of fluid in the gut towards the end of exercise, and they could achieve this by "front-loading" with fluid early on during exercise, and then maintaining an even fluid volume throughout. This is because it is often difficult to offset dehydration when the sweat rate exceeds 1.1 l/h, since this requires an athlete to perform with a gastric fluid volume of between 0.6 litres to 1.0 litre of fluid, which could be uncomfortable. Alternatively, maintaining an even fluid intake across a match would provide a constant fluid and carbohydrate delivery to the intestine, and should keep pace with sweat rate up to a certain point. For example, marathon runners should attempt to ingest fluid at a constant pace that will match the rate of dehydration that occurs (Coyle and Montain 1992).

5.7 On-Court Fluid Intake

In a one-off match lasting up to an hour and half in non-challenging conditions, then consuming water should be sufficient during changeovers. However, players should consider a carbohydrate-electrolyte drink regardless, because it is impossible to accurately predict the length of a match. If a match progresses past one to one and half hours in duration, then consuming a carbohydrate-electrolyte drink during changeovers is vital. This will help to replace a proportion of the sodium lost in sweat, help to maintain serum sodium levels, and minimise urine output in order to maintain hydration levels. Also, a lower core body temperature may result (Bergeron et al. 2006). The ideal carbohydrate content should be between 4% and 6%, because any greater than this has been shown to reduce the rate of absorption into the body, as well as cause gastrointestinal discomfort (Bergeron 1995b). The ideal electrolyte content varies largely between individuals; however typical sports drinks contain 10 to 25 mmol/l of sodium (Maughan & Shirreffs 1997). Players should be made aware of their typical sodium losses during match play at a variety of temperatures, however being able to fully match electrolyte intake with sweat electrolyte loss hour by hour is nearly impossible and may be of limited practical value (Maughan & Shirreffs 1997). As well as the stated benefits

from electrolyte ingestion, the carbohydrate content will help to delay fatigue if a match progresses for longer periods of time. Skill improvement is inconclusive, but any effect can only be beneficial to performance. Recommendations for fluid volumes during match play vary, but players should aim to replace 100% of fluid loss during exercise, particularly if intervals between matches are short.

5.8 Sodium Supplementation in the Diet

When a match lasts for little more than an hour, and at comfortable temperatures and humidities, there is little evidence to warrant sodium supplementation, and normal dietary intakes should suffice (Bergeron et al. 1995a, Maughan et al. 2004). However, when the player competes in tournament situations, especially in warmer and more humid climates, then additional sodium supplementation could be vital. Firstly, the player should be aware of his/her heat acclimation status, so they will be aware of whether they lose a large amount of sodium in sweat, and what volume of sweat they produce per hour under different environmental conditions.

With research suggesting that there is progressive plasma sodium depletion across three days of tennis match play (Bergeron et al. 1995a) players should aim to increase their dietary sodium intake on days during the tournament to combat the sodium losses in later rounds of tournaments. If players are competing twice in one day, they should aim to consume a large amount of sodium (1 - 2 g based on range of sodium losses observed) in their meal between matches to help replace the sodium loss caused by the previous match. Sometimes players may not be able to stomach a large meal in between matches, so carbohydrate-electrolyte drinks can be used to replenish electrolyte stores. Aside from meals, players should consider consuming small salted snacks, to help maintain sodium levels. These can also be consumed if comfortable at changeovers during match play, since regular opportunities exist.

Muscle cramping stems from the triad of fluid loss, salt loss, and muscle fatigue (Eichner 2007). Stofan et al. (2005) demonstrated slightly higher gross fluid losses, and much higher sweat sodium concentrations in American footballers who suffered heat cramping compared to non-cramping team-mates, while Bergeron et al. (2003) showed greater sweat rates and sweat sodium concentrations in healthy male tennis players who had a

history of heat cramping. The players most susceptible to suffering this condition in the present study would be those "salty sweaters" i.e. players with the highest sweat sodium concentrations who suffered the greatest fluid losses throughout their match. Bergeron (2003) has suggested that sodium and fluid losses "contract interstitial volume to mechanically deform nerve endings and increase ionic and neurotransmitter concentrations" (Bergeron 2003, as cited in Eichner 2007). However, the mechanisms behind the onset of these cramps remain largely unexplained.

In not so challenging conditions, the above recommendations do not become so vital. However, if poorly acclimated players are competing, then the recommendations still apply, and they should aim to increase their dietary sodium intake, as well as consume electrolytes on court at changeovers to offset their higher sweat losses and less effective heat loss mechanisms.

5.9 Recovery from Match Play

If a player will return to court up to an hour following the completion of a previous match, then they should aim to fully replace their fluid and electrolyte losses (Sawka et al. 2007), as well as consume either liquid or solid carbohydrate to optimise glycogen resynthesis (Bergeron et al. 1995b). A carbohydrate-electrolyte drink should be sufficient, as some players may not feel comfortable trying to consume a meal in short spaces between matches. According to Gisolfi & Duchman (1992), the carbohydrate-electrolyte drink should contain sodium concentrations in the region of 30 to 40 mmol/l, while Maughan & Shirreffs (1997) suggest that 10 to 25 mmol/l should suffice. Sawka et al. 2007 suggest that if rapid recovery from dehydration is required, then players should drink up to 1.5 litres per kilogram of body weight lost. The ACSM's position stand on fluid and electrolyte replacement suggests that if time permits, regular meals, as well as plentiful water and carbohydrate intake should provide sufficient fluid and electrolytes to restore euhydration, so this should be sufficient if matches are on successive or alternative days.

5.10 Limitations of the study

5.10.1 Sweat Collection: Problems with Local Sweat Sampling

This study collected samples from the forearm, calf, thigh and back sites, and the sweat sodium and chloride concentrations were significantly higher from the back site than the other three sites. These four sampling sites were selected from results presented by Patterson et al. (2000), who demonstrated that whole body concentrations of sweat sodium and chloride could be accurately predicted using an area weighted mean of four skin sampling sites. The four sites chosen for this study were the ones that showed the greatest correlation coefficients for analysis between regional and whole body sweat sodium and chloride concentrations.

However, there is much debate as to the reliability of sweat sampling from local skin sites using enclosed coverings like in the present study. Weschler (2008) stated that electrolyte concentrations obtained from local sampling using patches or coverings are artificially high, as they restrict the process of evaporation. Therefore, the sampling methods used in this study may have overestimated sweat electrolyte concentrations, and this is in accordance other studies including Palacios et al. (2003), and Shirreffs & Maughan (1997) who have also noted the same problems. The whole body wash-down technique is known to provide more accurate measurements of sweat electrolyte concentrations, since it does not restrict the evaporation of the sweat; however difficulties lie in being able to take these measurements while players compete in unrestricted conditions, and their performance is not hindered by measurement. Shirreffs & Maughan (1997) reported an improved method of sweat collection, but implementation into studies on tennis players would be impossible because of the obstructive nature of the sampling method, which involved placing a large plastic bag over a cycle ergometer upon which subjects exercised.

5.10.2 Measurement of Body Core Temperature

Another possible limitation to the study is that no measurements of body core temperature took place, which would have been a good marker of thermal load during the matches studied. Gant et al. (2006) investigated the validity of results from ingestible

temperature sensor capsules, as measuring core temperature using traditional methods is invasive and not possible during exercise. They showed small test-retest variability between results using these capsules during intermittent shuttle running, and this method could be applied to future studies in this area as an accurate way of measuring core body temperature during competitive environments.

5.10.3 Effort Intensity Calculation

There are a couple of potential limitations which related to the effort intensity calculations adopted in the present study that must be mentioned. Firstly, using an effort intensity measurement based on heart rate observations in itself brings about certain problems linked to an occurrence termed "cardiovascular drift," which can arise from increasing dehydration and thermal stress. Since extended exercise will bring about sweat losses, there is resultant fluid shift from the plasma to the tissues. The progressive fall in plasma volume decreases the cardiac filling pressure, causing a reduced stroke volume. This will cause a compensatory heart rate increase to attempt to maintain a relatively constant cardiac output: cardiovascular drift describes this gradual increase in heart rate which occurs as exercise progresses. Achten & Jeukendrup (2003) stated that increases in both core temperature and dehydration are closely linked to increases in heart rate. This is noted by reviews looking at matchplay intensity such as Fernandez et al. (2006), who state that care should be taken when interpreting heart rate responses during match play.

Secondly, the use of the pulmonary ventilation (VE) method to calculate the ventilatory thresholds (used in the present study) could be less reliable than using the ventilatory equivalents method of assessing exercise intensity. This equivalent method is calculated by plotting VE/VO_2 against VE/VCO_2, and taking the ventilatory threshold as the first rise in VE/VO_2 without an associated rise in VE/VCO_2. This method is considered more reliable as it uses two assessment criteria (a dual method of intensity assessment). The VE method used in this study also runs the risk of interpreting the first ventilatory threshold as the respiratory compensation point (RCP). The increment of VE against VO_2 has, in fact, two inflection points (Nakano 2000). The first is termed the first ventilatory threshold, which occurs at a lower VO_2, while the second inflection point, which occurs as exercise becomes heavier, is where a steeper increase in VE and VO_2 occurs. This point is termed the RCP. However, care was made in the present study to distinguish

between these two points, and ensure an accurate assessment of the first ventilatory threshold.

5.11 Conclusions

The results of this study provide an overview of the electrolyte balance and fluid intake strategies in British nationally ranked tennis players. In a comfortable indoor environment (17 ± 2°C, 42 ± 9% humidity), the sweat electrolyte concentrations recorded all fell at the lower end of the normal physiologic ranges stated by Maughan & Shirreffs (1997), yet were slightly above results observed by other studies carried out in elite sportspeople in hotter more humid conditions. This may reflect the lower sweat rates but also suggests that the players studied were not as well heat acclimated when compared to other sportspeople, who would presumably be used to training and competing at warmer temperatures. The total electrolyte losses from match play did not come close to exceeding the average daily electrolyte intake across the whole group, yet in individual cases sodium losses from match play did come close to matching the daily sodium intake. This is an issue that players should be made aware of and could be important in multi-day tournament situations. The whole body sweat rate for this group of players was 0.72 ± 0.26 l/h, which was lower than previous recordings of sweat rates in elite tennis players but previous reports were from higher ambient temperature conditions with players who were presumably more heat acclimated and would thus have a higher sweat rate (Bergeron et al. 1995a; Bergeron 1996, 2003). On average, players managed to maintain their body weight fairly well from pre to post exercise (89 ± 47% of fluid lost was replaced), with there being no significant body mass loss over time, however the range observed was large, with some players consuming far more fluid than they lost in sweat (one player replacing 148%), but some only replacing around 25% of their sweat loss. On the whole, fluid intake habits were good, but it was clear that certain players required education.

There is a strong basis for recommending that if these players are due to compete at high temperatures and humidities, they should be aware that they could suffer a high sodium loss, which could predispose them to suffering heat cramps. Players should supplement with a carbohydrate/electrolyte drink, to maintain their electrolyte levels and reduce the risk of suffering dehydration, fatigue, and heat cramps, as well as be able to increase their

dietary sodium intake to combat sodium depletion. In the scope of this study, however, this was not necessary: it was shown that players maintained their serum sodium levels from pre to post match, by only consuming water, and managed to prevent a significant body fluid loss across the whole group.

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Appendix A

Additional work was undertaken to validate the reliability of the Radiometer EML 105 ion electrode analyser. Sodium Chloride and Potassium Chloride standards of known concentration were prepared, and following calibration 10 samples of each were run through both the ion electrode analyser, and the flame photometer.

The coefficients of variation for both standards ran through each machine are shown in Table 8, and the mean ± SD results for the standards using the ion electrode analyser are shown in Table 9.

Table 8: Coefficients of variation of 10 samples of NaCl and KCl standards analysed using the Radiometer EML 105 ion electrode analyser and flame photometer.

Standard	CV% Radiometer EML 105	CV% Flame Photometer
37.5 mmol sodium	1.34	2.09
75 mmol sodium	1.33	2.32
150 mmol sodium	1.37	1.19
300 mmol sodium	1.49	0.58
21.875 mmol potassium	1.24	1.54
43.75 mmol potassium	0.93	0.75
87.5 mmol potassium	1.45	1.28
175 mmol potassium	1.53	0.78

Table 9: Mean ± SD of 10 samples of NaCl and KCl standards analysed using the Radiometer EML 105 ion electrode analyser.

Standard	Mean ± SD Radiometer EML 105 (mmol)
37.5 mmol sodium	38.4 ± 0.52
75 mmol sodium	72.6 ± 0.97
150 mmol sodium	145.7 ± 2.00
300 mmol sodium	299.1 ± 4.46
21.875 mmol potassium	21.3 ± 0.26
43.75 mmol potassium	42.4 ± 0.39
87.5 mmol potassium	86.3 ± 1.25
175 mmol potassium	174.9 ± 2.68