

**THE EFFECTS OF WATER INGESTION ON HIGH INTENSITY CYCLING  
PERFORMANCE IN A MODERATE AMBIENT TEMPERATURE**

**Thesis submitted in partial fulfilment of the degree of Master of Philosophy  
(Sports Medicine)**

**by**

**Tracy Robinson, MBChB**

**The Medical Research Council and the University of Cape Town Bioenergetics of  
Exercise Research Unit**

**Department of Physiology**

**University of Cape Town Medical School**

**Observatory, 7925**

**South Africa**

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## DECLARATION

I, Tracy Anne Robinson, do hereby declare that this dissertation embodies only my original work except where acknowledgement indicates otherwise and that no part of it has been, or is being, submitted for a degree at this or any other university.

This thesis is presented in partial fulfilment of the requirements for the degree of MPhil (Sports Medicine).

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**ABSTRACT**

Eight endurance-trained cyclists rode as far as possible in 1 h on a stationary cycle-simulator in a moderate environment (20°C, 60% relative humidity, 3 m/s wind speed) while randomly receiving either no fluid (NF) or attempting to replace their ~1.7 l sweat loss measured in a previous 1 h familiarisation performance ride at ~85% of peak oxygen uptake ( $\text{VO}_2$  peak) with artificially sweetened, coloured water (F). During F the cyclists drank  $1.49 \pm 0.14$  l (values are mean  $\pm$  SEM), of which  $0.27 \pm 0.08$  l remained in the stomach at the end of exercise and  $0.20 \pm 0.05$  l was urinated after the trial. Thus, only  $1.02 \pm 0.12$  l of the ingested fluid was available to replace sweat losses during the 1 h performance ride. That fluid decreased the average heart rate from  $166 \pm 3$  to  $157 \pm 5$  beats/min ( $P < 0.0001$ ) and reduced the final serum  $[\text{Na}^+]$  and osmolalities from  $143 \pm 0.6$  to  $139 \pm 0.6$  mEq/l ( $P < 0.005$ ) and from  $294 \pm 1.7$  to  $290 \pm 1.9$  mOsm/l ( $P = 0.05$ ), respectively. Fluid ingestion did not attenuate rises in plasma anti diuretic hormone and angiotensin concentrations, or decrease the ~-15% falls in estimated plasma volume in the F and NF trials. Nor did fluid ingestion significantly effect the ~1.7 l/h sweat rates, the rises in rectal temperature (~36.6° to 38.3°C) or the ratings of perceived exertion in the two trials. Ingestion of ~1.5 l of fluid produced an uncomfortable stomach fullness and reduced the distance covered in 1 h from  $43.1 \pm 0.7$  to  $42.3 \pm 0.6$  km ( $P < 0.05$ ). Thus, trying to replace  $>1.0$  l/h sweat losses during high-intensity, short duration exercise in a moderate environment does not induce beneficial physiological effects, and may impair exercise performance.

## **Chapter One**

### **INTRODUCTION**

Since the early studies which demonstrated that exercise induced dehydration decreases endurance in subjects exercising at low work rates for many hours in the heat (Adolph 1938, Bean and Eichna 1943, Eichna et al. 1945, Ladell 1955, Pitts et al. 1946), surprisingly little attention has been paid to the influence of inadequate fluid ingestion on high intensity athletic performance. Recent studies of fluid replacement during exercise have largely focused on the reduction in endurance associated with the large ( $\geq 4\%$  of body mass) sweat losses incurred during prolonged ( $\geq 2$  hr), moderately low intensity [ $\leq 65\%$  of  $\text{VO}_2 \text{ max}$  (maximum oxygen uptake)] exercise at high ( $\geq 30^\circ\text{C}$ ) ambient temperatures (Barr et al. 1991, Montain and Coyle 1992).

To our knowledge, the only reports on the advantages of drinking during reasonably high intensity exercise in warm ( $\geq 30^\circ\text{C}$ ) environments are the studies of Walsh et al. (1994) and Below and Coyle (1993). Walsh et al (1994) found that when subjects drank sufficient fluid (a 20 mmol/l NaCl solution) to replace their  $\sim 1.1$  l sweat losses during a 1 hr ride at 70% of  $\text{VO}_2 \text{ max}$  in the heat, they were able to cycle for 34% longer during a subsequent exercise bout at 90% of  $\text{VO}_2 \text{ max}$ . Below and Coyle (1993) reported that with the ingestion of 1.3 l of water during a 50 min ride at 80% of  $\text{VO}_2 \text{ max}$  subjects were able to complete a subsequent workload 6.5% faster than without fluid ingestion. Hence, attempts to prevent even low ( $< 2\%$  of body mass) levels of dehydration probably improve high intensity exercise performance in the heat.

In contrast, the effects of drinking on high intensity exercise performance in cooler environments remain to be determined (Coyle and Hamilton 1990). Despite the fact that athletes can lose 1-2 l of sweat/hr during exercise at 75-85% of  $\text{VO}_2 \text{ max}$ , even in cool conditions, there is little information on how such fluid losses affect their ability to generate high work rates in moderate ambient temperatures. Accordingly, the aim of this study was to examine whether attempts to replace fluid losses during intense exercise might also improve athletic performance in moderate environmental conditions.



**Chapter Two**

**LITERATURE REVIEW**

**THE EFFECTS OF DEHYDRATION ON FLUID HOMEOSTASIS,  
CARDIOVASCULAR FUNCTION AND PERFORMANCE DURING  
EXERCISE**

## Background

The earliest studies to investigate the effects of fluid replacement on exercise capacity in humans were conducted in the 1930's, mostly in military or industrial settings. Talbott and colleagues (1933) studied 15 men working in an extremely hot environment [32-49°C, relative humidity (r.h.) 20%]. The subjects were accustomed to both the environmental conditions and the work being performed. Fluid intake and urine output were measured while the men were working and resting, and blood measurements for estimation of plasma volume, serum bicarbonate and serum chloride were undertaken before and after an eight hour shift. Both fluid intake and urine output were increased during the period of work. Although the workers sweated profusely, all physiological parameters measured remained within normal limits. Talbott et al. (1933) concluded that prolonged physical activity in the heat was not detrimental to an individual's health. Adolph and Dill (1938) later studied fluid replacement in construction workers working in the heat (31-39°C) and found that subjects only replaced approximately half of their fluid losses during or immediately after exercise. The results of this study initiated the concept of 'voluntary dehydration' in humans.

The next investigations were conducted during the 1940's on soldiers in the desert and resulted in some of the most important findings in the area of dehydration and exercise performance (Adolph 1947). During one investigation, men attempted to walk at ~6 km/hr for 50 min of each hour in temperatures of 31-34°C, until a distance of 33.6 km had been covered, or until exhausted. It was found that subjects became progressively dehydrated with exercise and that they became exhausted once body weights had declined by ~4-8%. Rectal temperature and pulse rates increased with exercise time. When water was ingested, less men failed to complete the march than when no fluid was ingested (one out of seven subjects vs. seven out of twelve). Those individuals who stopped walking for reasons other than exhaustion have been excluded.

These studies, however, were largely ignored by the athletic community. Indeed, it was not until 1953 that the International Amateur Athletics Federation (IAAF) made the first official reference to fluid replacement during long-distance running (IAAF Handbook, 1953). The rules at this time stated that "*refreshments shall (only) be provided by the organizers of the race after 15 km or 10 miles, and thereafter every 5 km or 3 miles*". It was only in the 1970's, after the studies by Pugh et al. (1967) and Wyndham and Strydom (1969) were completed, that marathon runners began to ingest fluids during endurance events. At that time, fluid ingestion was advocated to prevent heat injury rather than to enhance performance (Wyndham 1977), and for the next decade athletes were encouraged to drink water, in large volumes, during prolonged exercise.

### **The Effects of Prolonged Moderate Intensity Exercise on Physiological and Psychological function**

Heat generated during exercise leads to a rise in body temperature. The increase in body temperature of an athlete is proportional to his or her metabolic rate (which is determined by body mass) and the relative intensity (speed or work rate) of exercise (Noakes et al. 1991a). Since a large rise in body temperature is detrimental to the athletes' health and performance, heat must be dissipated by convection, conduction or radiation (Sawka and Pandolph 1990). However, as ambient temperature rises, the athlete becomes more dependant on evaporative heat loss (i.e. on sweating) for the dissipation of heat (Nielsen 1938).

#### *Sweat Rate*

Sweat rate is determined principally by metabolic rate (Costill 1977, Davies 1979, Davies et al. 1976, Greenhaff 1989, Greenhaff and Clough 1989, Noakes et al. 1991a, Wyndham et al.

1970), and is influenced by environmental conditions and the athletes' clothing (Adolph 1947, Molnar et al. 1946, Shapiro et al. 1982, Strydom et al. 1966). In the majority of endurance athletes sweat rates do not exceed 1.2 l/hr (Noakes et al. 1991a, 1993). Studies on the effects of dehydration on sweat rate have produced variable results, with some studies showing a decrease in sweat rate with dehydration (Ekblom et al. 1970, Greenleaf and Castle 1971, Ladell 1955, Strydom et al. 1975) while others have shown no effect (Barr et al. 1991, Candas et al. 1988, Gisolfi and Copping 1974, Hamilton et al. 1991, Montain and Coyle 1992a,b). It may be that only high levels of dehydration (losses of  $>2.5$  l body water after 60-110 min exercise) lead to a decrease in sweat rate (Ladell 1955). Rises in serum sodium concentration and osmolality with dehydration have been shown to correlate with increased oesophageal temperature (Montain and Coyle 1992b), and may therefore be a stimulus for the reduction in sweat rate at high levels of dehydration.

### *Serum Osmolality*

Associated with sweat loss is a concomitant loss of sodium and chloride ions (Costill 1977) from the extracellular compartment (Sawka 1988). Since sweat is hypotonic when compared to extracellular fluid, sweating leads to a rise in serum osmolality (Candas et al. 1988, Coyle and Hamilton 1990, Maughan et al. 1987, Nose et al. 1988a, Senay 1979). The precise concentration of electrolytes contained in sweat depends on the athletes' level of fitness and state of heat acclimation (Allan and Wilson 1971). Heat acclimation leads to an increase in sweat rate and a reduction of the ion (mainly sodium) concentration in sweat (Kirby and Convertino 1986). Reductions in the changes in sweat and serum sodium concentrations during exercise help maintain serum osmolality and plasma volume.

### *Plasma Volume*

During the first 5-10 min of exercise, prior to any fluid loss due to sweating, there is a reduction in plasma volume. This is due to the increase in blood pressure with the onset of

exercise which shifts fluid out of the intravascular compartment and into the interstitium. The magnitude of the reduction in plasma volume is dependant on the type and intensity of exercise as well as the posture adopted (Coyle and Hamilton, 1990). Following this initial decrease, sweating leads to a progressive decline in plasma volume and total body water, especially when exercising in the heat (Harrison 1985, Sawka et al. 1984, Senay and Pivarnik 1985). When dehydration is less than approximately 3% of body mass, the decline in body water is predominantly from the extracellular compartment. However, as levels of dehydration increase, more fluid is lost from the intracellular compartment (Sawka and Pandolph 1990). When exercising in neutral or cool environments ( $<25^{\circ}\text{C}$ ) at 65 - 75%  $\text{VO}_2$  max, plasma volume remains relatively constant (Costill et al. 1970, Sawka et al. 1979, 1980), even with large (4-6%) declines in body mass. Coyle and Hamilton (1990) concluded that 'since plasma volume remains constant but mass decreases, fluid must be lost from the interstitial and intracellular compartments'.

### *Body Temperature*

Accelerated fluid losses with an increase in exercise intensity are associated with rises in oesophageal temperatures (Montain and Coyle 1992a,b). Hyperthermia (defined as a rectal temperature of  $>40^{\circ}\text{C}$ ) can occur even when exercising in a cool environment if the exercise is at sufficiently high intensity, since the increase in body temperature is proportional to metabolic rate (Hughson et al. 1983, Noakes et al. 1991a, Robinson 1963).

### *Cardiovascular drift*

During prolonged exercise there is also a progressive increase in heart rate while stroke volume and cardiac output decreases (Adolph 1947, Candas et al. 1988, Greenleaf and Castle 1971, Maughan et al. 1987, Strydom et al. 1975). This cardiovascular drift is seemingly related to the fluid deficit incurred before (Heaps et al. 1994) or during exercise (Montain and Coyle 1992a,b) and increases with increasing environmental temperature and relative exercise

intensity (Coyle and Hamilton 1990). The major cause of cardiovascular drift is thought to be the shift of the circulating blood volume to the skin for the dissipation of heat (Ekeland 1967, Ekeland et al. 1967, Johnson and Rowell 1975, Raven and Stevens 1988, Rowell 1986).

### *Hormonal Changes*

The circulating concentrations of hormones such as ADH, atrial natriuretic peptide (ANP) and those of the renin-angiotensin-aldosterone system, which regulate fluid and electrolyte concentrations, increase during prolonged exercise (Altenkirch et al. 1990, Brandenberger et al. 1989, Wade and Freund 1990). The extent of the increase in hormonal concentrations is influenced by both the exercise intensity and its duration (Wade and Claybaugh 1980), as well as the type of activity and posture adopted (Coyle and Hamilton 1990, Guezennec et al. 1986). Plasma ADH concentration tends to increase with an increase in serum osmolality (Thrasher et al. 1981, Wade and Freund 1990), while both ADH and angiotensin concentrations increase with increasing exercise intensity (Convertino et al. 1981, 1983) and with an increasing water deficit (Brandenberger et al. 1986, Francesconi et al. 1983, 1985). An increase in renin activity may follow a fall in either plasma or extracellular fluid volumes. Angiotensin II concentrations correlate with plasma renin activity (Reid and Ganong 1977, Staessen et al. 1987), while aldosterone concentrations mimic those of renin and angiotensin II (Reid and Ganong 1977).

While numerous studies have investigated the response of the above hormones to exercise, few have focused on their actions, such as their role in the regulation of blood volume during exercise. The increase in ADH, aldosterone and renin concentrations does not appear to influence either sweat rate or the solute content of sweat, nor does the elevated concentration of ADH appear to increase renal reabsorption of water (Wade and Freund 1990). However, the increased aldosterone concentration does promote renal tubular reabsorption of sodium, resulting in renal conservation of fluid and electrolytes. The elevated aldosterone levels may

also be responsible for the reduced excretion of solutes which occurs following exercise since aldosterone concentrations remain elevated for a prolonged time following cessation of exercise. It has been postulated that the renin-angiotensin system may play an important role in the regulation of thirst (Wade and Freund 1990).

Another hormonal system which shows increased activity with exercise is the sympatho-adrenal system. Plasma adrenaline and noradrenaline levels increase with increased exercise intensity, and are also influenced by exercise duration (Galbo et al. 1975). Catecholamines are important in reducing renal blood flow (Baer and McGiff 1980), which leads to a reduction in urine volume and solute excretion (Wade and Claybaugh 1980), resulting in net renal conservation of fluid and electrolytes.

### *Psychological changes*

As early as the 1940's investigators noted the effects of dehydration on mental function. Dehydrated subjects were observed to have low morale and to be apathetic, but were energetic and cheerful when performing the same task in a euhydrated state (Bean and Eichna 1943, Eichna et al. 1945). Recently, studies have shown that ratings of perceived exertion (RPE) increase in proportion to levels of dehydration when exercising in the heat (Montain and Coyle 1992, Walsh et al. 1994).

### **Fluid Replacement during Exercise**

The progressive decrease in plasma volume caused by sweat losses can be reduced or prevented by ingesting water during prolonged, moderate-intensity exercise (Hamilton et al. 1991). In practice, however, it is extremely difficult to drink sufficient fluid to fully replace

(>1 l/hr) sweat losses. Even if fluids are readily available and made more palatable by cooling (Armstrong et al. 1985, Sandick et al. 1984), flavouring (Epstein and Sohar 1985, Hubbard et al. 1990, Morimoto et al. 1981) or the addition of sodium (Costill et al. 1975, Hubbard et al. 1981), humans seldom drink more than 0.5-0.6 l/hr (Noakes 1993) and therefore most endurance athletes undergo a degree of voluntarily dehydration.

Voluntary dehydration may be the result of a number of factors. One possible explanation is that drinking a small amount wets the mouth and reduces one of the stimuli for drinking (Armstrong et al. 1984, Hubbard et al. 1990). Another possibility is that the ingestion of large volumes of fluid during exercise results in abdominal discomfort. Abdominal discomfort occurs to some extent while cycling (Mitchell and Voss 1991), but is a particular problem during running (Brouns et al. 1987, Costill et al. 1970). A third explanation for incomplete fluid replacement may be that dipsogenic drive ceases prior to complete fluid replacement. Nose et al. (1988b) have shown that changes in both serum osmolality and plasma volume may affect dipsogenic drive. Ingestion of water returns serum osmolality to isotonicity, while ingestion of sodium chloride solutions restores plasma and extracellular volumes. Both situations result in the cessation of dipsogenic drive before intracellular fluid losses are replaced (Noakes 1993).

Even if athletes are forced to ingest sufficient fluids to replace fluid losses, the fluid must still be emptied from the stomach and absorbed by the small intestine before it is available to replace extracellular or intracellular fluid losses. Originally the rate of gastric emptying was thought to be the most important determinant limiting fluid availability and has been investigated extensively. Factors affecting the rate of gastric emptying include fluid osmolality (Carnot and Chassevant 1905, Hunt and Pathack 1960, Rehrer et al. 1993), temperature (McArthur and Feldman 1989, Sun et al. 1988), caloric content (Costill and Saltin 1991, Coyle et al. 1978, Fordtran and Saltin 1967) and gastric volume (Costill and Saltin 1974,



Marbaix 1898, Mitchell and Voss 1991, Noakes et al. 1991b, Rehrer et al. 1990). Of these factors, gastric volume may be the most important (Noakes et al. 1991b, Rehrer et al. 1990). Rates of gastric emptying fall rapidly as the volume in the stomach decreases (Hunt and Knox 1969, Gisolfi and Duchman 1992, Rehrer et al. 1990). Irrespective of the osmolality of the ingested fluid, rates of gastric emptying are determined by the volume of fluid ingested (Hawley et al. 1992, Moodley et al. 1992).

Rates of maximal intestinal water absorption are more difficult to study (Schedl et al. 1994) and are therefore less well documented. Diamond (1991) has suggested that the amount of fluid that can be absorbed by the gut may be limited. Barr et al. (1991) and Costill and Sparks (1973) conducted studies where fluid replacement equalled fluid losses and found that not all of the ingested fluid could be accounted for in the extracellular or intracellular fluid pools, suggesting that it had not all been absorbed. A limited rate of intestinal absorption may explain the sensation of abdominal fullness and is also supported by the results of Mitchell et al. (1991) where a forced ingestion of 1.6 l of fluid per hr lead to the development of diarrhoea in 25% of the subjects. At present the maximum rate of fluid absorption in the small bowel is not known, but it appears that it may be less than the rate of sweat loss during intensive exercise (Abbott 1936, Noakes 1993).

The effects of various types and intensities of exercise on rates of absorption are also not clear. Fordtran and Saltin (1967) exercised subjects at 70%  $\text{VO}_2 \text{ max}$  for 1 hr and found that intestinal absorption and gastric emptying rates were no different to those at rest. However, Costill and Saltin (1974) found that rates of gastric emptying decreased when exercise intensity exceeded 70% of  $\text{VO}_2 \text{ max}$ .

## **The Effects of Fluid Replacement on Physiological and Psychological Function during Prolonged Exercise**

### *Plasma Volume*

Senay and Pivarnik (1985) reviewed those studies which had examined the effects of fluid ingestion on plasma volume during exercise in the heat. They found that the results were very variable in seemingly similar experiments, possibly due to differences in exercise intensity, duration, posture, mode of exercise, skin temperature, state of training and heat acclimation. However, in a cool or neutral environment fluid ingested during exercise does not appear to accumulate in the intravascular compartment since plasma volume remains stable (Coyle and Hamilton 1990). This suggests that the ingested fluid attenuates the decline in intracellular or interstitial fluid.

### *Serum Osmolality and Sweat Rate*

As discussed previously, the effect of dehydration on sweat rate is variable. However, the rise in serum osmolality and sodium concentration with prolonged exercise is reduced with fluid ingestion, and is least when the rate of fluid ingestion approximates the rate of fluid loss (Barr et al. 1991, Candas et al. 1986, Candas et al. 1988, Maughan et al. 1987). Fluid replacement may therefore be more important in attenuating the rise in serum osmolality than in maintaining plasma volume. Hyperosmolality can elevate core temperature by decreasing sweat rates at high levels of dehydration (Fortney et al. 1985, Harrison et al. 1978, Sawka et al. 1985).

### *Body Temperature*

The increase in body temperature after 60-80 min of exercise is attenuated by fluid ingestion (Adolph 1947, Barr et al. 1991, Candas et al. 1988, Costill et al. 1970, Ekblom et al. 1970,

Strydom et al. 1975, Strydom and Holdsworth 1968), particularly when rates of fluid ingestion approximate sweat rates (Greenleaf and Castle 1971, Montain and Coyle 1992a,b). However, the attenuation of the rise in core temperature is relatively small (Noakes et al. 1991c). Most studies show that prevention of dehydration of up to 5% of body mass only decreases rectal temperature by  $<1^{\circ}\text{C}$  (Barr et al. 1991, Gisolfi and Copping 1974, Hamilton et al. 1991, Montain and Coyle 1992a,b, Noakes et al. 1988).

#### *Forearm Blood Flow*

During prolonged exercise, forearm blood flow is reduced in proportion to the level of dehydration (Montain and Coyle 1992b). Fluid ingestion may assist in thermoregulation by preventing a rise in serum osmolality and a decrease in skin blood flow, rather than by maintaining plasma volume. Montain and Coyle (1992a,b) have shown that where fluid ingestion attenuates the rise in serum osmolality and prevents a decline in forearm blood flow, the increase in core temperature during prolonged exercise in the heat is also attenuated. In contrast, when plasma volume was maintained by intravenous infusion without altering serum osmolality, there was no attenuation of the rise in core temperature (Montain and Coyle 1992a).

#### *Cardiovascular Drift*

If the rate of fluid ingestion is sufficient to replace fluid losses, then the decrement in stroke volume (Hamilton et al. 1991) and cardiac output (Hamilton et al. 1991, Montain and Coyle 1992a,b) which occur during prolonged exercise in the heat can be minimised. This is, however, not the case for heart rate (Hamilton et al. 1991, Montain and Coyle 1992a,b), which suggests that dehydration is not the sole cause of the increase in the heart rate. It is postulated that rising catecholamine levels may also be partially responsible for the increased heart rate during prolonged exercise. This hypothesis has been supported by studies showing

that either hyperglycaemia (induced by glucose infusion) (Hamilton et al. 1991) or  $\beta$ -blockade (Kallis et al. 1988) attenuated cardiac drift.

### *Hormonal Changes*

Ingestion of fluid either prior to or during exercise reduces the rise in ADH, aldosterone, renin and ANP (Brandenberger et al. 1989). Plasma ADH is most affected and appears to be independent of the type of fluid ingested. However, the increase in the renin-angiotensin-aldosterone system appears to be reduced more by sodium-containing solutions than by plain water (Brandenberger et al. 1989). This may be due to the maintenance of higher plasma volumes with the ingestion of electrolyte solutions than with water (Brandenberger et al. 1989, Noakes 1992).

### *Psychological response*

Fluid ingestion lowers the RPE during prolonged exercise in the heat and even partial fluid replacement significantly decreases the perception of exertion during high intensity exercise (Montain and Coyle 1992b). Montain and Coyle (1992a) also noted that RPE values were lower with fluid ingestion than with fluid infusion when both were given in sufficient quantities to maintain plasma volume.

## **The Effects of Dehydration on Exercise Performance**

In order to study the effects of dehydration on the physiological responses to exercise, early researchers dehydrated subjects by means of saunas, diuretics or fluid restriction prior to exercise testing. However, hypohydration has more profound effects on physiological

function than does exercise-induced dehydration at the same level of fluid loss (Caldwell et al. 1984, Coyle and Hamilton 1990, Montain and Coyle 1992a) and these studies are more applicable to sports where athletes deliberately dehydrate themselves in order to make a specific weight category. For the purposes of this review only those studies where dehydration occurred during exercise have been discussed.

### *Exercise performance in the heat*

#### *Low and moderate intensity exercise*

The majority of studies on exercise performance and fluid intake have been conducted at low to moderate exercise intensities (50-65% of  $\text{VO}_2 \text{ max}$ ) in the heat. Pitts et al. (1944) conducted studies where subjects walked at 5.6 km/hr up an incline of 2.5% for periods of up to 6 hr in the heat ( $\sim 38^\circ\text{C}$ ). Ladell (1955) conducted several experiments where subjects performed stair stepping intermittently for 2-4 hr in temperatures of  $\sim 32\text{-}35^\circ\text{C}$ . Both investigators found that subjects were better able to complete the task if fluids were ingested. It also appeared that hyperthermia and cardiovascular drift were attenuated by fluid ingestion and that it was best to replace all the fluid lost through sweating.

Two more recent studies confirmed these findings. Barr et al. (1991) had subjects exercise at 55% of  $\text{VO}_2 \text{ max}$  for 6 hr ( $30^\circ\text{C}$ , 50% r. h.) and found that subjects terminated the trial after approximately 4.5 hr when they did not ingest water compared to 6 hr if water was ingested. Montain and Coyle (1992) conducted a trial under similar conditions (62-67% of  $\text{VO}_2 \text{ max}$ ,  $33^\circ\text{C}$ , 50% r.h., wind speed 2.5 m/sec) for 2 hr. These workers also found that subjects were more likely to complete the trial if fluids (a carbohydrate-electrolyte solution) were ingested during the exercise bout.

### *High intensity exercise*

Although fluid replacement during prolonged low-intensity exercise has received a great deal of attention, the importance of hydration during high-intensity exercise of shorter durations has not been extensively investigated. Changes in physiological parameters may be less over a short time-period and may therefore be more difficult to measure, while the capacity for fluid replacement is also limited. Athletes have found that drinking during intense exercise is more difficult to co-ordinate, may lead to stomach fullness or nausea, and interferes with their ability to concentrate (Coyle and Hamilton 1990).

Only two studies have been undertaken to examine the effects of fluid ingestion on high-intensity exercise capacity in the heat. Below and Coyle (1993) exercised cyclists at 80% of  $\text{VO}_2 \text{ max}$  for 50 min ( $31^\circ\text{C}$ , 54% r.h., 3.5m/sec wind speed), while ingesting either 1.33 l or 0.2 l of fluid (either water or a 6% carbohydrate solution). Performance times in a subsequent high-intensity time trial were 6.5% faster when subjects ingested the larger fluid volume, regardless of the carbohydrate content. Fluid ingestion was also found to attenuate hyperthermia (by  $0.33 \pm 0.04^\circ\text{C}$ ) and reduce heart rate (by  $4 \pm 1$  b/min).

Walsh et al. (1994) also showed that when subjects ingested 1 l of a 20 mmol/l Na Cl solution during a 1 hr cycling bout at 70% of  $\text{VO}_2 \text{ max}$  in the heat ( $30^\circ\text{C}$ , 50% r.h., 3 km/hr wind speed), they were able to exercise for significantly longer ( $9.8 \pm 3.9$  vs.  $6.5 \pm 3$  min;  $P < 0.005$ ) during a subsequent exercise bout at 90% of  $\text{VO}_2 \text{ max}$  than when no fluid was ingested. The improvement in endurance occurred despite the fact that the subjects were only 1.8% dehydrated when no fluid was ingested.

*Exercise performance in moderate or cool environments*

Despite the fact that most exercise is conducted in moderate or cool environments (temperature < 25°C) there are very little data on the effects of fluid ingestion on exercise performance at those temperatures. Powers et al. (1990) showed that the ingestion of a glucose or glucose/electrolyte solution improved endurance capacity in cyclists exercising at ~85% of  $\text{VO}_2 \text{ max}$  for 35-40 min in cool conditions (20-22°C) compared to the ingestion of water, but did not compare this to no fluid ingestion. The only other study that has been performed under similar conditions was carried out using a fluid infusion (Deschamps et al. 1989). In that study subjects cycled at 84% of  $\text{VO}_2 \text{ max}$  for ~21 min in a moderate environment (24°C, 26% humidity). Subjects received an intravenous infusion of 0.9% saline sufficient to maintain plasma volume (69 ml/min) on one occasion and a negligible amount on the other. It was expected that maintenance of plasma volume would lower core temperature and heart rate and result in an increase in performance. Although temperature and heart rate were in fact found to be lower with saline infusion, no improvement in endurance capacity occurred.

Thus several questions remain unresolved. Firstly, few studies have addressed the question of whether performance per se rather than endurance capacity (as measured by exercise time to exhaustion) is impaired by dehydration. Secondly, most researchers have suggested that it is optimal to replace as much of fluid losses as is possible, yet no studies have been conducted where full fluid replacement has occurred. Thirdly, there is little information on the effects of fluid ingestion on high intensity exercise and, finally, few studies have been conducted in moderate ambient conditions.

**Chapter Three**

**METHODS**



Athletic performance was measured in eight endurance trained male cyclists who rode regularly for more than 90 min/day, 4-6 days/week. The investigation was approved by the Research and Ethics Committee of the Faculty of Medicine of the University of Cape Town and each subject signed an informed consent, in accordance with the guidelines of the American College of Sports Medicine (1994).

The subjects' ages, heights and body masses are shown in Table 1, together with their peak oxygen uptake ( $VO_{2\text{ peak}}$ ), peak sustained power outputs (PPO) and heart rate peaks ( $HR_{\text{peak}}$ ).

Table 1. Subject characteristics

Age (yr)	Height (m)	Body mass (kg)	$VO_{2\text{ peak}}$ (l/min)	PPO (W)	$HR_{\text{peak}}$ (bpm)
25.3	1.82	78.2	5.17	433	182
$\pm 1.4$	$\pm 0.02$	$\pm 2.4$	$\pm 0.31$	$\pm 27$	$\pm 3$

Values are means  $\pm$ SEM (n = 8).

$VO_{2\text{ peak}}$  and PPO values were measured during an incremental exercise test to exhaustion on an electronically braked cycle-ergometer (Lode, Groningen, Holland). In this test, the subjects started exercise at a work rate of 3.33 W/kg body mass and, thereafter, the work rate was increased by first 50 W and then 25 W every 150 sec, as described by Hawley and Noakes (1992).

On another day, the subjects undertook a performance ride against the clock, in which they cycled as far as possible in 1 hr on a cycle-simulator at an ambient temperature of 20°C, a relative humidity of 60% and a wind speed of 3 m/s, in a locally-designed environmental chamber (Ventex Environmental Chambers, JT Thermal Consulting Company, Somerset West, South Africa). This familiarisation ride, which was performed under the same conditions as in the subsequently described experimental trials, was primarily designed to minimise any learning effect in successive experimental trials.

An additional purpose of the 1 hr familiarisation ride was to determine how much fluid each subject should attempt to drink in order to replace his total sweat loss. Individual sweat rates were estimated from the differences between the subjects' pre- and post-exercise nude body masses, and ranged from 1.0 to 1.9 l/hr, irrespective of their body mass. Body masses were measured on a Seca precision balance (Model 770, Bonn, Germany), after the subject had urinated and towelled dry. No corrections were made for the small, (~100 g) predicted weight losses resulting from the expiration of hydrogen and carbon atoms as H<sub>2</sub>O and CO<sub>2</sub> during the ride (Pivarnik et al. 1984, Pugh et al, 1957).

In the performance trials, the subjects rode their own racing bicycles mounted on a "Kingcycle" ergometer (EDS Portapront Ltd, High Wycombe, Bucks, United Kingdom). Bicycles were attached to the ergometer by the front fork and an adjustable bottom bracket. The bottom bracket was used to adjust the rolling resistance of the rear tyre on an air-braked flywheel to match that of a 70 kg cyclist on a level road. From that rolling resistance and the output of a photo-optic sensor monitoring the velocity of the flywheel, in revolutions per second (RPS), an IBM compatible computer calculated the power output, in Watts (W), that would be generated by a 70 kg cyclist riding at that speed on level terrain, using the following equation:

$$W = 0.000136 \text{ RPS}^3 + 1.09 \text{ RPS}$$

Before each test, the tension on the bottom bracket was adjusted to produce a rolling resistance of a 70 kg cyclist, by a series of 'run down' calibrations, in which the subject accelerated to a work rate of ~ 300 W and then immediately stopped pedalling, while remaining seated on their bike in a crouched time-trial position. These calibrations were repeated until the computer display indicated that the slowing of the flywheel matched the reference power decay curve of a 70 kg cyclist.

Preliminary data on the test to test reliability of the "Kingcycle" ergometer have been obtained from six cyclists who each performed three 40 km time-trials. The mean and standard deviation of the coefficient of variation of the times taken to complete the rides were  $0.97 \pm 0.50\%$  (G.S. Palmer, personal communication).

Following the familiarisation ride, the subjects were asked to return to the laboratory at the same time of day, one and two weeks later, to repeat a random order of two experimental performance rides. Over that period, the subjects continued their normal training, but rested on the day before each trial. Subjects were asked to consume the same food and beverages and refrain from alcohol for 24 hr prior to a trial. Training and dietary records were kept to check compliance. On the day of a trial, subjects reported to the laboratory at least 3 hr post prandial and ingested 5 ml/kg body mass of water 2 hr before the onset of exercise. A 24 hr abstinence from alcohol and the consumption of ~250 ml of water 2 hr before the trials was designed to ensure euhydration at the start of exercise. Euhydration was subsequently confirmed by similar pre-trial body masses, haematocrits, haemoglobin concentrations and serum osmolalities (Table 2), as described later.

On arrival at the laboratory, the subjects first performed the previously outlined 'run down' calibrations on the "Kingcycle" ergometer and then urinated before their pre-exercise nude body mass was determined. After the subjects had been weighed, an 18 gauge cannula was

inserted into a forearm vein for the collection of blood samples, and thermistor probes were positioned ~15 cm into the rectum and on the calf, thigh, chest and upper right arm, to monitor rectal and weighted skin temperatures (Ramanathan 1964).

Once the cannula and thermistors were in place, the subjects entered the environmental chamber and sat still for 10 min while the thermistors were connected to a YSI telethermometer (described below) and a venous blood sample (20 ml) was withdrawn. Following the collection of the pre-exercise blood sample, the subjects "warmed-up" on their bicycles for 5 min and then rode as far as possible in 1 hr, with no feedback other than a stop-watch attached to their handlebars.

During the no fluid trial (NF), the subjects received no fluid, other than 10 ml of water every 15 min to wet their mouths. In the fluid trial (F), they were provided with sufficient cool ( $5^{\circ}\text{C}$ ), artificially sweetened, orange-flavoured water to replace their (1.0 - 1.9 l/h) fluid losses in the previous familiarisation ride. Flavoured water was delivered to the cyclist's mouth via a static plastic tube connected to a drinking bottle suspended from the ceiling of the chamber. The cyclists were instructed to drink  $629 \pm 22$  ml of water (8 ml/kg body mass) at the start of the "warm-up" and the rest of the water in five equal aliquots at 0, 10, 20, 30 and 40 min in the 60 min performance ride. Such a drinking pattern has previously been reported to result in high rates of gastric emptying *throughout* exercise (Rehrer et al. 1990). No fluid was ingested during the last 10 - 20 min of exercise since it was considered unlikely that it would be absorbed prior to completion of the trial. Although cyclists were encouraged to empty the drinking bottle at the end of each 10 min time period, after 50 min only  $859 \pm 123$  ml had been consumed. By ingesting  $1.49 \pm 0.14$  l of flavoured water during exercise, the cyclists managed to replace ~ 85% of their fluid losses during the trial.

During the performance rides cycling speeds, distances covered and power outputs were calculated by the "Kingcycle" computer. Heart rates were recorded every 60 sec with a

"Polar" heart rate monitor (Polar Electro OY, Kempele, Finland). Changes in rectal and skin temperatures from resting values were measured with the YSI telethermometer (Model 4002, Yellow Springs, Ohio, USA) at 10 min intervals. As the temperatures were being recorded, the subjects were asked also to indicate their ratings of perceived exertion (RPE), using the 20 point Borg scale (Borg 1975), and to rank their stomach fullness on a scale of 1 (empty) to 5 (bloated) as described by Hawley et al. (1991).

In addition, venous blood samples (20 ml) were collected after 5, 20, 40, and 60 min of the performance ride. These samples, plus the blood sample that had been withdrawn at rest, were placed into the appropriate tubes (described subsequently) and stored on ice until the end of the trial. Following the collection of each blood sample, the venous cannula was flushed with 1-2 ml of sterile saline containing heparin (5 U/ml) to prevent coagulation. For this reason the first 2 ml of the following sample was discarded.

Within 5 min of completing the F trial, a No. 14 French Levine nasogastric tube was inserted to aspirate the residual gastric contents. This procedure was not followed after the NF trial since gastric contents were likely to be negligible following the ingestion of only 30 ml during the trial. The cannula and thermistors were removed, and urine outputs were recorded. The volume of fluid remaining in the stomach and the volume of urine excreted were subtracted from the volume of fluid ingested to calculate the volume of fluid available to replace sweat losses during exercise. Knowing the volume of fluid 'retained' allowed calculation of the sweat rates from the decreases in post-exercise, nude, dry body masses in both the trials.

Venous blood samples (20 ml each) collected prior to and during the experimental trials were divided into three aliquots. One aliquot (2 ml) was placed into a tube containing lithium heparin for determination of the haematocrit and haemoglobin concentrations immediately after the trial. Another aliquot (10 ml) was allowed to clot (in a tube containing SST gel and clot activator), spun at 2500 x g for 12 min in a Sigma 302K refrigerated (4°C) centrifuge

(Laborzentrifugen, Germany) and the supernatant was stored at  $-20^{\circ}\text{C}$  for determinations of serum sodium and potassium concentrations and osmolality. The remaining blood (8 ml) was placed in a cold ( $4^{\circ}\text{C}$ ) lithium heparin tube, centrifuged at  $2500 \times g$  and the supernatant was stored at  $-20^{\circ}\text{C}$  for measurements of plasma ADH and angiotensin II concentrations.

Haematocrits and haemoglobin concentrations were measured in triplicate by micro-centrifugation and the cyanomethaemoglobin spectrophotometric method respectively (Hainline 1958). The estimated decreases in plasma volume during exercise were calculated from the haematocrit and haemoglobin concentration changes, as described by Dill and Costill (1977).

Serum osmolalities were determined from freezing point depressions in an Osmette A automatic osmometer (Precision Systems Inc., Newton, Massachusetts, U.S.A.) and plasma sodium and potassium concentrations were assayed with ion selective electrodes (KNA 1 Radiometer, Copenhagen, Denmark). Plasma antidiuretic hormone (ADH) and angiotensin II concentrations were determined in acetone extracted plasma samples by specific radioimmunoassays, previously described in detail (Gray and Simon 1983, Simon-Opperman et al. 1986).

All results are presented as means  $\pm$ SEM from the number ( $n = 8$ ) of subjects who performed the trials. Differences over time between trials were analysed for statistical significance by a two way analysis of variance (ANOVA) for repeated measures. Significant treatment  $\times$  time differences were located using a Scheffe post-hoc test. Single differences between trials were assessed with a paired Student's  $t$  test using two tailed values of  $p$ . A value of  $p < 0.05$  was regarded as statistically significant.

## **Chapter Four**

### **RESULTS**

At the start of the NF and F performance rides, subjects were euhydrated as indicated by a similar body mass, haemoglobin concentration, haematocrit and serum osmolality (Table 2).

Table 2. Evidence of euhydration before exercise

Trial	Mass (kg)	Haemoglobin (g/100 ml)	Haematocrit (%)	Osmolality (mOsm/l)
No fluid	78.1 $\pm$ 2.5	14.9 $\pm$ 0.4	40.2 $\pm$ 1.0	280.7 $\pm$ 0.7
Fluid	78.2 $\pm$ 2.4	15.2 $\pm$ 0.3	40.3 $\pm$ 1.0	281.8 $\pm$ 0.3

Results are means  $\pm$  SEM (n = 8). Differences between trials were not significant.

### Fluid ingestion

With the provision of enough flavoured water to replace their (1-1.9 l) sweat losses in a previous familiarisation ride, the subjects managed to drink 1.49  $\pm$  0.14 l of fluid during exercise (Table 3). However, 0.27  $\pm$  0.08 l of the ingested fluid remained in the stomach at the end of the trial and 0.20  $\pm$  0.05 l was excreted in the urine. Only 1.02  $\pm$  0.12 l of the ingested fluid was 'retained'.

### Sweat rates

While the retention of  $\sim$ 1.0 l of ingested fluid reduced the subjects' body mass losses during the ride from 1.78  $\pm$  0.15 to 0.72  $\pm$  0.12 kg (p < 0.0001), it had no effect on their estimated sweat rates (Table 3). When the weight of the retained fluid was added to the loss of body



mass during the fluid trial, the calculated sweat rates were the same as those in the no fluid trial ( $1.74 \pm 0.10$  vs.  $1.78 \pm 0.15$  l/hr).

Table 3. Effects of fluid ingestion on weight loss, urine output and sweat rates during exercise with and without fluid

	No fluid	Fluid
Volume ingested (l)	-	$1.49 \pm 0.1$
Residual gastric volume (l)	-	$0.27 \pm 0.08$
Volume urinated (l)	$0.11 \pm 0.03$	$0.20 \pm 0.05^*$
Volume retained (l)	-	$1.02 \pm 0.12$
Loss of body mass (kg/hr)	$1.78 \pm 0.15$	$0.73 \pm 0.12^{**}$
Sweat rate (l/hr)	$1.78 \pm 0.15$	$1.74 \pm 0.10$

Values are means  $\pm$  SEM (n = 8). Fluid ingestion increased urine output (\* p < 0.001) and helped maintain body mass (\*\* p < 0.0001), but had no significant effect on sweat rates.

### Urine output

Fluid ingestion also had little effect on urine accumulation during the trials (Table 3).

Although drinking increased the volume of urine produced during exercise from  $0.11 \pm 0.03$  to  $0.20 \pm 0.05$  l/hr (p < 0.001), the  $\sim 0.09$  l increase in urine formation was negligible compared to the  $1.21 \pm 0.11$  l of fluid emptied from stomach into the intestine.

### Skin and rectal temperatures

The large (~1.2 l) volume of fluid delivered to the intestine also had no effect on the changes in mean skin and rectal temperatures during the trials (Table 4). With or without fluid ingestion, mean skin temperatures decreased from  $31.1 \pm 0.5$  to  $\sim 26.4 \pm 0.6^\circ\text{C}$  and rectal temperatures rose from  $36.7 \pm 0.2$  to  $38.3 \pm 0.3^\circ\text{C}$  by the end of exercise. Although rectal temperatures appeared lower during the second 30 min of the F trial than the NF trial, these differences were not statistically significant.

Table 4. Changes in mean skin and rectal temperatures during exercise, with and without fluid.

	Rest	10 min	20 min	30 min	40 min	50 min	60 min
$T_S$ ( $^\circ\text{C}$ )							
NF	31.3 $\pm 0.6$	28.4 $\pm 0.3$	27.1 $\pm 0.8$	26.9 $\pm 0.8$	26.6 $\pm 0.9$	26.4 $\pm 0.9$	26.6 $\pm 0.6$
F	30.9 $\pm 0.4$	27.8 $\pm 0.3$	27.8 $\pm 0.4$	27.4 $\pm 0.4$	27.0 $\pm 0.6$	26.7 $\pm 0.6$	26.2 $\pm 0.6$
$T_R$ ( $^\circ\text{C}$ )							
NF	36.8 $\pm 0.1$	37.4 $\pm 0.1$	37.9 $\pm 0.2$	38.0 $\pm 0.2$	38.3 $\pm 0.2$	38.5 $\pm 0.2$	38.6 $\pm 0.2$
F	36.5 $\pm 0.2$	37.2 $\pm 0.2$	37.9 $\pm 0.1$	38.0 $\pm 0.2$	38.1 $\pm 0.2$	38.2 $\pm 0.1$	38.1 $\pm 0.2$

$T_S$ , skin temperature;  $T_R$ , rectal temperature. Values are means  $\pm$ SEM ( $n = 8$ ). Differences were not significant.

### Ratings of perceived exertion and stomach fullness

Ratings of perceived exertion were also similar between trials (Table 5). The only difference was that when the subjects ingested fluid, they felt bloated (Table 6). During the F trial, ratings of abdominal fullness rose from  $3.1 \pm 0.4$  units after 10 min to  $4.1 \pm 0.23$  units at 50 min and then declined slightly to  $3.8 \pm 0.3$  during the last 10 min of the ride, when no further fluid was ingested (Table 6).

Table 5. Ratings of perceived exertion during the two exercise trials

	10 min	20 min	30 min	40 min	50 min	60 min
No fluid	14.1 $\pm 0.7$	14.1 $\pm 0.5$	15.1 $\pm 0.5$	16.6 $\pm 0.3$	17.4 $\pm 0.3$	19.8 $\pm 0.2$
Fluid	13.9 $\pm 0.6$	14.6 $\pm 0.4$	14.9 $\pm 0.8$	16.3 $\pm 0.5$	17.1 $\pm 0.5$	18.9 $\pm 0.4$

Values are means  $\pm$ SEM ( $n = 8$ ) from a 20 point Borg scale (Borg 1975). Differences were not significant.

Table 6. Ratings of abdominal fullness during the fluid exercise trial

	10 min	20 min	30 min	40 min	50 min	60 min
Fluid	3.1 $\pm 0.4$	3.5 $\pm 0.3$	4.0 $\pm 0.3$	4.1 $\pm 0.4$	4.1 $\pm 0.2$	3.8 $\pm 0.3$

Values are means  $\pm$ SEMs ( $n = 8$ ). Abdominal fullness was ranked from 1 (empty) to 5 (extreme discomfort).

## Cycling performance

Fluid ingestion reduced the 'distance covered' in the 1 hr performance rides from  $43.05 \pm 0.68$  km to  $42.32 \pm 0.58$  km ( $P < 0.05$ , Figure 1).

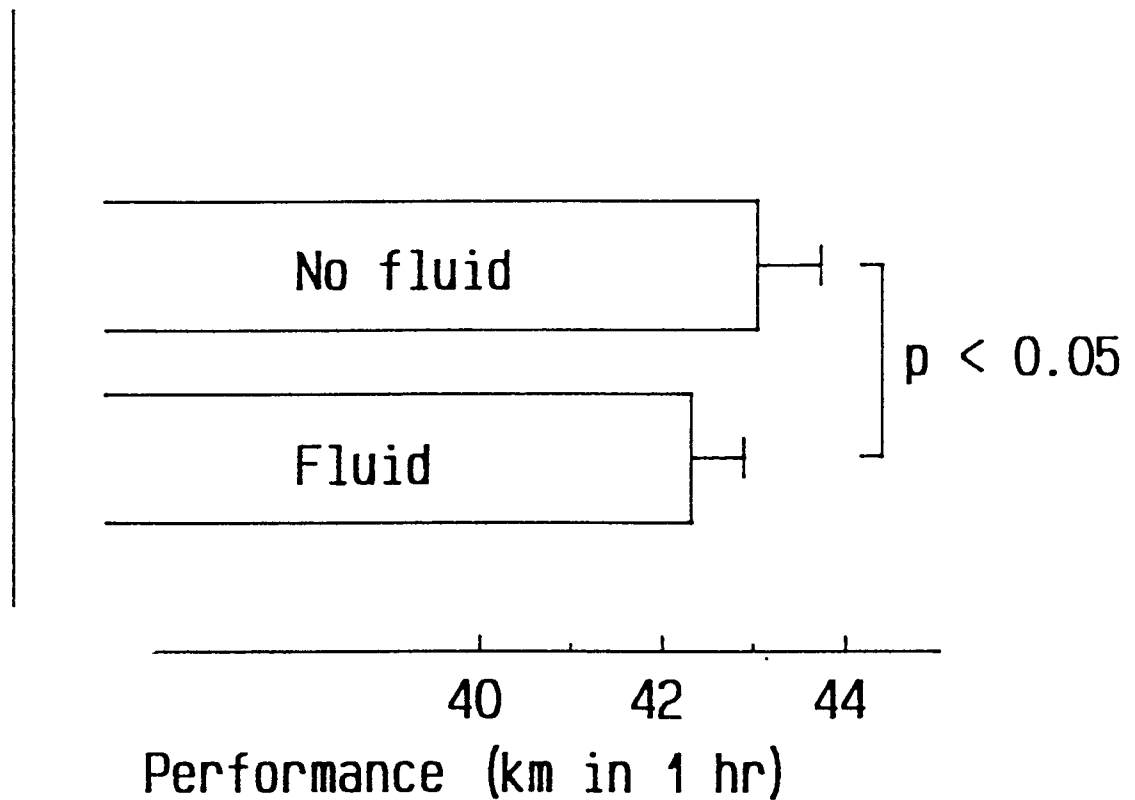


Figure 1. Distances covered during the F and NF trials.

Values are means  $\pm$  SEM ( $n=8$ ). Distances covered in the NF trial and the habituation ride were not significantly different.

Decreases in work rate with fluid ingestion were most apparent between the 25th and 45th min of the 1 hr rides (Figure 2A). Over this period, the lower work rates in the F trial than in the NF trial reduced the mean work rate from  $303 \pm 2.7$  to  $293 \pm 2.6$  W ( $P < 0.05$ ).

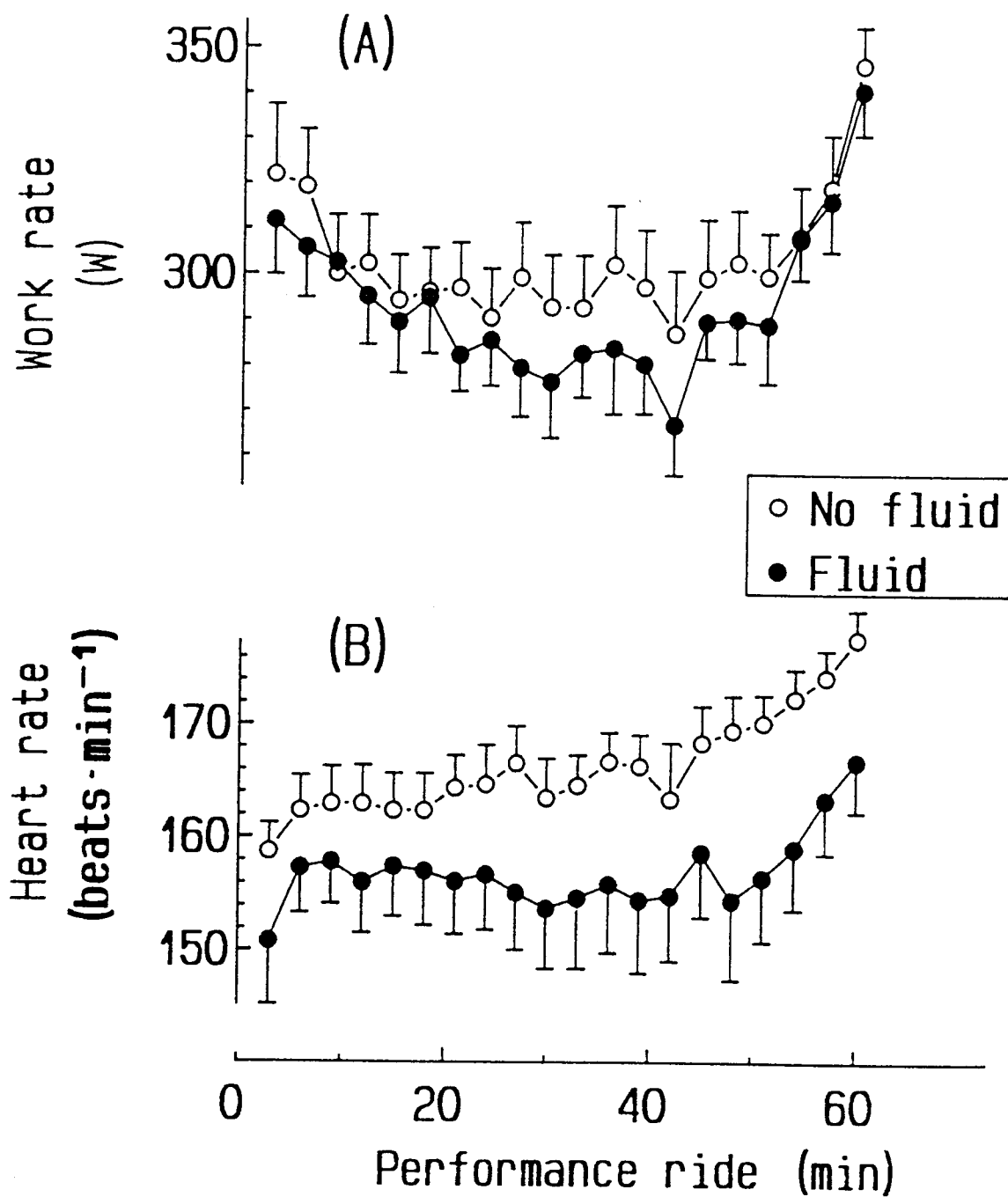


Figure 2. Power outputs and heart rates during exercise with and without fluid.

Values are means  $\pm$  SEM ( $n=8$ ). Power and heart rates have been averaged over each successive 3 min periods.

### **Heart-rates**

Heart rates were also lower when the subjects drank during exercise (Figure 2B). In the NF trial, the mean heart rate was  $166 \pm 0.7$  beats/min and, in the F trial, it was  $157 \pm 1.2$  beats/min ( $P < 0.0001$ ).

### **Plasma volume**

In contrast, fluid ingestion did not attenuate the estimated decreases in plasma volume during exercise (Figure 3A). In both trials, plasma volumes fell by 12-13 % in the first 5 min of the performance ride and, by the end of exercise, they had declined by 15-17 %.

### **Serum potassium and sodium concentrations**

There were also no significant differences in the rises in serum potassium concentrations in the no fluid and fluid trials. With or without fluid ingestion, as the plasma volumes fell by 12 - 13% in the first 5 min of exercise, potassium concentrations rose from 4.7-4.8 mmol/l to 5.6-5.7 mmol/l and then remained constant for the rest of the trial (Table 7).

Table 7. Potassium concentrations during exercise with and without fluid

Potassium (mmol/l)	Rest	5 min	20 min	40 min	60 min
NF	4.77 $\pm$ 0.08	5.77 $\pm$ 0.1	5.65 $\pm$ 0.14	5.65 $\pm$ 0.23	5.63 $\pm$ 0.31
F	4.70 $\pm$ 0.13	5.58 $\pm$ 0.16	5.58 $\pm$ 0.16	5.68 $\pm$ 0.16	6.15 $\pm$ 0.11

Values are means  $\pm$ SEM (n=8). Differences between the NF and F trials were not significant.

Fluid ingestion also did not attenuate the increases in serum sodium concentrations. Serum sodium concentrations increased from  $\sim$ 138 to  $\sim$ 140 mEq/l during the first five min of both trials but, thereafter, differences emerged (Figure 3B). Whereas the serum sodium concentrations continued to rise from 140.6  $\pm$ 0.9 to 143.0  $\pm$ 0.6 mEq/l in the no fluid trial, they fell from 140.3  $\pm$ 0.7 to 138.6  $\pm$ 0.5 mEq/l after 20 min of exercise in the fluid trial and, then, remained 2-3 mEq/l lower than in the no fluid trial until the end of the ride ( $p < 0.005$ ).

### Serum osmolality

A similar pattern was also seen with the increases in serum osmolality during the performance rides (Figure 3C). Again the initial rise in serum osmolality from  $\sim$ 281 to  $\sim$ 288 mOsm/l was similar in the two trials but, after 20 min of exercise, serum osmolalities tended to be lower when fluid was ingested. Drinking reduced final serum osmolalities in the performance rides from 294  $\pm$ 1.7 to 290  $\pm$ 1.9 mOsm/l ( $p = 0.054$ ).

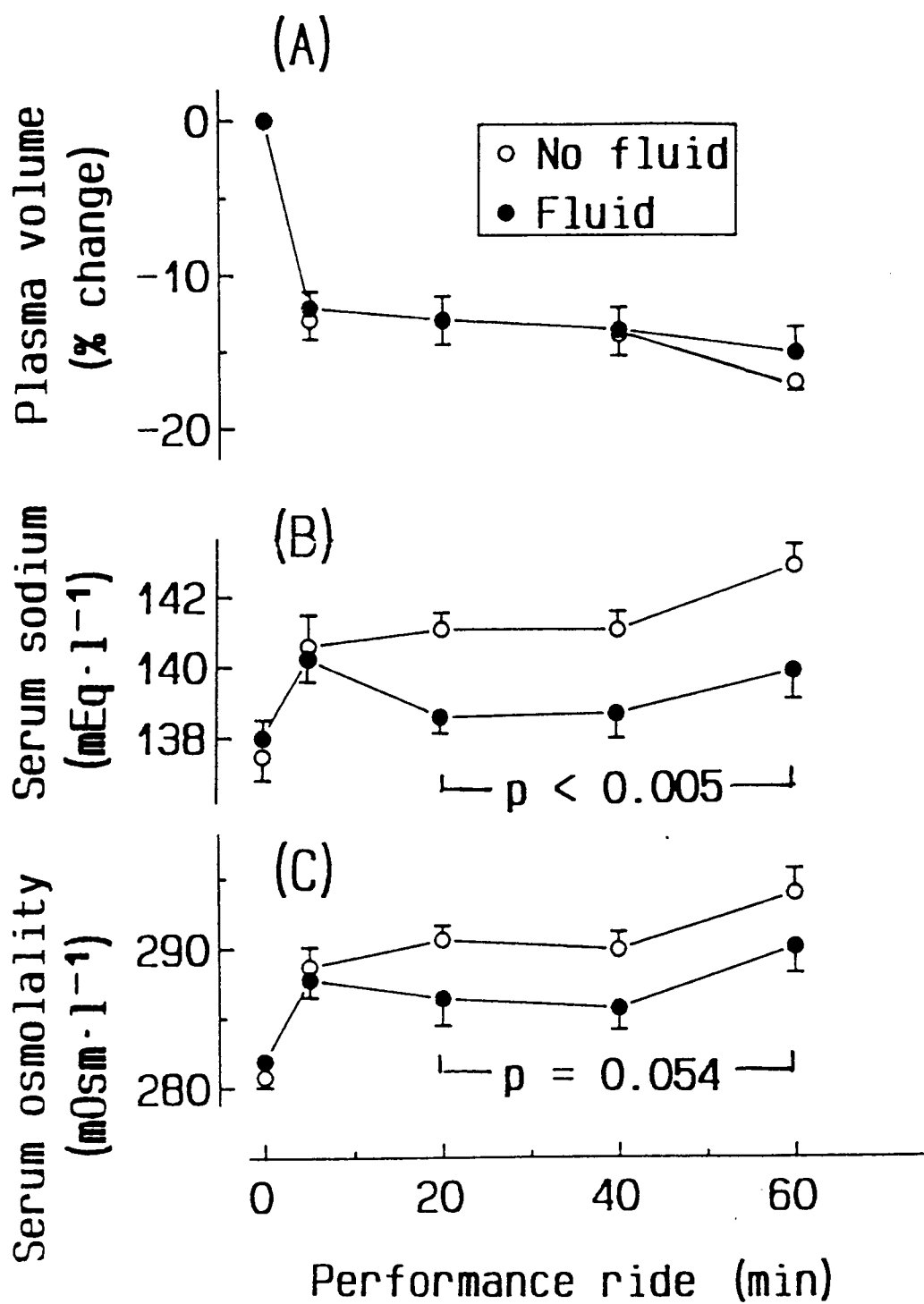


Figure 3. Changes in plasma volume (A), serum sodium concentration (B) and osmolality (C) during the two trials. Values are means  $\pm$ SEM (n=8).



### Plasma antidiuretic hormone (ADH) and angiotensin II concentrations

Lower serum sodium concentrations and osmolalities during the performance rides with fluid ingestion, however, did not measurably affect the rises in plasma ADH and angiotensin II concentrations (Table 8). Although ADH and angiotensin II concentrations tended to be slightly lower in the fluid trial than in the no fluid trial, the differences were not significant.

Table 8. ADH and Angiotensin II concentrations during exercise with and without fluid

	Rest	5 min	20 min	40 min	60 min
ADH (pg/ml)					
(NF)	5.3 ±0.4	7.0 ±0.9	8.3 ±0.9	8.7 ±0.7	12.8 ±2.1
(F)	4.9 ±0.3	6.2 ±0.6	6.0 ±0.5	7.9 ±1.7	9.8 ±2.4
Angiotensin II (pg/ml)					
(NF)	9.8 ±1.3	21.7 ±6.1	61.6 ±24.7	99.0 ±30.7	98.7 ±27.1
(F)	12.8 ±1.3	17.9 ±1.6	35.1 ±19.1	68.8 ±17.1	115.5 ±32.1

Values are means ±SEM (n=8). Differences between the NF and F trials were not significant.

**Chapter Five**

**DISCUSSION**

In this investigation, eight endurance-trained (Table 1), euhydrated (Table 2) cyclists rode as far as possible in 1 hr in a moderate (20°C) environment, while receiving either no fluid or attempting to ingest the measured ~1.7 l volume of fluid they had lost in a previous 1 hr familiarisation performance ride. Although previous studies have evaluated the effects of fluid ingestion on high intensity (>80% of  $\text{VO}_2 \text{ max}$ ) exercise performance in the heat (Below and Coyle 1993, Walsh et al. 1994), to our knowledge, this is the first study conducted under moderate ambient conditions.

The first finding of the current investigation was that the cyclists were unable to replace their total sweat losses during the 1 hr high intensity (85% of  $\text{VO}_2 \text{ peak}$ ) performance ride. When the subjects attempted to replace their ~1.7 l fluid losses, they managed to consume only ~1.5 l of which ~0.3 l remained in the stomach at the end of exercise and ~0.2 l was urinated at the end of the trial (Table 3). Thus, only ~1.0 l of the ingested flavoured water was available to replace sweat losses during the 1 hr performance trial (Table 3).

Similar rates of ingested fluid retention have also been found in other studies where cyclists' sweat rates exceeded 1.0 l/hr. In those studies, cyclists rode at 62-70% of  $\text{VO}_2 \text{ max}$  in ambient temperatures of either 22°C (Hamilton et al. 1991) or 32-33°C (Montain and Coyle 1992a,b, Walsh et al. 1994). Under both warm and hot conditions, the cyclists' fluid losses were 1.2-1.4 l/h and the volumes of 'retained' fluid were ~0.9, 1.0 l/h (minus urine volume) and ~1.2 l/h (plus urine volume), respectively (Hamilton et al. 1991, Montain and Coyle 1992a,b, Walsh et al. 1994). It may therefore not be practical for athletes to drink sufficient volumes of fluid to fully replace their high (>1.5 l/h) sweat rates during moderate to high intensity exercise.

The second finding was that the ~1.0 l of 'retained' fluid had a negligible effect on urine production during exercise (Table 3) and no effect on the sweat rates (Table 3), the changes in skin and rectal temperatures (Table 4), or RPE values (Table 5). At the end of both performance rides, the ~38°C rectal temperatures were comparable to those found after 1 h of

exercise in the heat (Barr et al. 1991, Hamilton et al. 1991, Montain and Coyle 1992a,b, Walsh et al. 1994).

Others have also found that fluid ingestion does not significantly increase sweat rates or attenuate the rises in core temperature in the first 60 min of exercise even in the heat (Barr et al. 1991, Hamilton et al. 1991, Montain and Coyle 1992a,b, Walsh et al. 1994). However, fluid ingestion did reduce RPE during exercise at high ( $> 30^{\circ}\text{C}$ ) ambient temperatures (Barr et al. 1991, Hamilton et al. 1991, Montain and Coyle 1992a,b, Walsh et al. 1994) and increased exercise time to exhaustion in hot conditions (Barr et al. 1991, Below and Coyle 1993, Walsh et al. 1994).

In contrast to the studies in the heat, the results of the current study showed that drinking at rates of  $\sim 1.5$  l/hr failed to improve performance during high intensity exercise at moderate ( $20^{\circ}\text{C}$ ) ambient temperatures (Figure 1). Fluid ingested at the rates used in this study reduced the 'distance covered' in the 1 hr performance ride from  $43.05 \pm 0.68$  km to  $42.32 \pm 0.58$  km ( $P < 0.05$ ).

Decreases in work rate with fluid ingestion were most evident between the 25th and 45th min of the 1 hr rides (Figure 2A). In contrast, heart rates were consistently lower throughout the fluid trial, irrespective of the changes in work rates (Figure 2B).

Several investigations have demonstrated that fluid ingestion attenuates the rise in heart rate without maintaining measurably higher blood volumes and stroke volumes during exercise (Costill et al 1970, Montain and Coyle 1992b, Heaps et al. 1994). However, the mechanism(s) for this observation are not well understood ( Montain and Coyle 1992b).

Nor is the impairment in cycling performance with fluid ingestion easily explained. One possibility is that drinking interfered with the subjects ability to concentrate on cycling at a

high (85% of  $\text{VO}_2 \text{ max}$ ) exercise intensity, as has previously been proposed (Coyle and Hamilton 1990). Ratings of abdominal fullness during the 1 hr performance ride in the fluid trial indicated that most of the subjects felt uncomfortably bloated (Table 6) and suggest that some of the ingested fluid may have remained in the intestine. Although maximum rates of fluid absorption are  $\sim 0.8$  l/hr at rest (Davis et al. 1980), several groups have suggested that these may be limited during exercise (Barr et al. 1991, Williams et al. 1976) and may be considerably lower at high exercise intensities (Fordtran and Saltin 1967).

Certainly, the ingested fluid was not retained in the vascular compartment. Fluid ingestion had no measurable effect on the estimated declines in plasma volume during exercise (Figure 3A). Others have also observed that fluid ingestion does not usually significantly attenuate the early falls in plasma volume (Barr et al. 1991, Montain and Coyle 1992a, Walsh et al. 1994). Early declines in plasma volume appear to be more dependent on the exercise intensity than on whether or not fluid is ingested (Gisolfi and Copping 1974). In the first hour of exercise, the plasma volumes were reduced by 8-12% at 55-70% of  $\text{VO}_2 \text{ max}$  (Barr et al. 1991, Montain and Coyle 1992a), by  $\sim 15\%$  at  $\sim 85\%$  of  $\text{VO}_2 \text{ max}$  (Powers et al. 1990, Figure 3A) and by 17-18% at 90% of  $\text{VO}_2 \text{ max}$ , with or without fluid ingestion (Walsh et al. 1994). It is possible that conservation of plasma volume was not superior with water ingestion because water provides no osmotic impetus to retain fluid in the vascular space. In this regard, Costill (1970) and Montain and Coyle (1992b) have reported that plasma volumes were better maintained with the ingestion of carbohydrate-electrolyte solutions.

A failure of water consumption to significantly decrease the decline in plasma volume during the 1 hr performance rides was also reflected in the comparable rises in serum potassium concentrations in the two trials and in the similar initial increases in serum sodium concentration and osmolality during the first 5 min of exercise. Only after 5 min of exercise did fluid ingestion prevent further rises in serum sodium concentration and osmolality (Figures 3B and 3C).

The 2-3 mEq/l decreases in serum sodium concentration with fluid ingestion, however, were (a) less than the 6-7 mEq/l reductions in serum sodium concentrations at the end of more prolonged (2-6 hr) exercise in persons ingesting fluid at high rates (Barr et al. 1991, Montain and Coyle 1992b) and (b) not sufficient to attenuate the rises in plasma ADH and angiotensin II concentrations during exercise (Table 7). Although the lower serum sodium concentrations and osmolalities in the fluid trial tended to decrease the plasma ADH concentrations, the differences were not significant.

These data therefore suggest that it may be neither advisable nor practical for athletes to drink sufficient fluid to fully replace their total sweat losses during high ( $\sim 85\%$  of  $VO_{2\max}$ ) intensity, short ( $< 1$  hr) duration exercise in moderate ( $\sim 20^{\circ}\text{C}$ ) ambient temperatures. Attempts to attenuate falls in plasma volume and rises in core temperature by replacing  $\sim 80\%$  of sweat rates are probably more important in prolonged ( $> 2$  hr) moderate intensity exercise in the heat (Montain and Coyle 1992b) where fluid losses are incurred for long enough to make dehydration and hyperthermia a primary concern (Coyle and Hamilton 1990). During the 1 hr performance rides in a moderate environment, the maximum retention of  $\sim 1.0$  l of ingested fluid had no measurable effects on the changes in plasma volume or rectal temperature. The only effects of the cyclists' attempts to fully replace their sweat rates during exercise at  $\sim 85\%$  of  $VO_{2\max}$  in  $20^{\circ}\text{C}$  ambient temperatures was an uncomfortable stomach fullness and a decrease in performance.

Of course it should be realised that these results are only applicable to exercise of high intensity ( $> 80\%$   $VO_{2\max}$ ) and short duration ( $\sim 60$  min) occurring in a moderate environment ( $\sim 20^{\circ}\text{C}$ ). Whether ad libitum rates of fluid intake or the addition of carbohydrates and/or electrolytes to the fluid would result in improved performance remains to be determined.

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