THE EFFECTS OF SODIUM CHLORIDE INGESTION ON FLUID BALANCE AND BODY FLUID DISTRIBUTION DURING EXERCISE

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To Andrea

"A mole of a substance contains Avogadro's number (6.02×10^{23}) of molecules."

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DECLARATION

I, BARRY SANDERS, declare that the work on which this thesis is based is original (except where acknowledgements indicate otherwise) and that neither the whole work nor any part thereof has been, is being, or is to be submitted for any other degree at this or any other University.

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ABSTRACT

The aim of the first experiment of this thesis was to determine whether the ingestion of a concentrated sodium chloride solution (100mEq/l) during exercise would expand the plasma volume when fluid was ingested at approximately half the rate at which it was being lost as sweat. Six male cyclists exercised for 90 minutes in the heat (32 ± 1 °C, 55 ± 5% RH) at 66 ± 1 % of VO_{2max} while ingesting either no fluid (NF), water (W), or a saline (S) solution (100mEq/l). In the W and S trials, subjects drank 400ml of the fluid immediately prior to commencing exercise, and 100ml of fluid every 10 minutes during exercise until 80 minutes. In the S trial sodium chloride was ingested in capsules. One capsule containing 0.585g of sodium chloride was ingested with every 100ml of water. At the end of the 90 minute exercise bout they rested in a sitting position for one hour in cool conditions (22 ± 1 °C and 70 ± 5% RH).

After the initial drop in plasma volume due to the onset of exercise, plasma volume decreased progressively during the NF trial and was significantly less than the 10 minute value at 80 and 90 minutes ($p<0.0033$). At 40, 60, 80 and 90 minutes of exercise, the plasma volume in the NF trial was significantly less than in the W and the S trials ($p<0.05$). There was no significant difference between the W and the S trials at any time. Further, after the initial drop in plasma volume due to the onset of exercise, plasma volume did not decrease any further in either the W or the S trial.

Plasma sodium concentrations in the NF and the S trial were significantly elevated at 40, 60, 80 and 90 minutes ($p<0.0033$). Plasma sodium concentration in the NF and the S trials were also significantly higher than in the W trial at 80 and 90 minutes of exercise ($p<0.05$). Since the ingestion of a sodium chloride solution containing 100mEq/l did not have a beneficial effect on plasma volume and plasma sodium concentration, when fluid ingestion rates were approximately half of the rate of sweat loss, it is concluded the under these conditions, the ingestion of a concentrated sodium chloride beverage has no advantage over the ingestion of water.

The aim of the second experiment of this thesis was to determine the effect of varying concentrations of sodium chloride ingestion on fluid balance, when the rate of fluid ingestion matched the sweat rate. Six male cyclists cycled for 4 hours at 55% of VO_{2max} in mild conditions (20 ± 1°C and 70 ± 5% RH), while ingesting either a low salt (LS) (4.6 mEq/l), a medium salt (MS) (50 mEq/l) or a high salt (HS) (100 mEq/l) beverage. Each beverage also contained a glucose polymer in an 8% concentration (8g/100ml). The subjects ingested 400ml of beverage immediately prior to commencement of exercise, and 150ml of fluid every 10 minutes during exercise until 220 minutes. Sodium chloride in the MS and HS trials was given to the subjects as supplemental gel capsules so that the drink was palatable. At the end of exercise, subjects recovered in a sitting position for 30 minutes.

At the end of the 4 hours of exercise, fluid loss via the urine was significantly greater in the LS and the MS trials than in the HS trial ($p<0.05$). As a result, the fluid deficits in the LS and the MS trials were significantly greater than the fluid deficit in the HS trial. There was no significant difference between the MS and the LS trials for urinary fluid loss.

During the 4 hour exercise bout, plasma sodium concentrations in the LS, the MS and the HS trials were not significantly different from one another, nor were they significantly different from resting values.

There was no significant difference in the rectal temperature response to exercise in the three trials.

It can therefore be concluded that in conditions where fluid ingestion matches sweat rate, attenuation of urinary fluid loss to optimise fluid replacement, relies on the ingestion of sodium chloride in quantities greater than that lost in the sweat.

Therefore, for the ingestion of sodium chloride in excess of that which is currently available in sports drinks to beneficial, fluid must be ingested in volumes matching sweat loss.

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CHAPTER 1: RATIONALE AND AIMS

Montain and Coyle (1992b) showed that after two hours of cycling in the heat, the increase in core temperature, the magnitude of the cardiovascular drift, and the rating of perceived exertion were proportional to the level of dehydration that developed during exercise. Their work demonstrated that if fluid replacement with a 6% carbohydrate-electrolyte beverage closely matched fluid loss, the disturbance of cardiovascular function was less and the final core temperature and rating of perceived exertion were lower at the end of exercise. Further, they showed that a high rate of fluid intake attenuated hyperthermia and cardiovascular drift, by elevating skin blood flow and thus increasing the rate of heat dissipation.

Montain and Coyle (1992b) concluded that any level of dehydration is detrimental to cardiovascular function, thermoregulation and perceived exertion. This disproves the erroneous conclusion by Wyndham and Strydom (1969), that thermoregulation, cardiovascular function and performance are not significantly impaired until the level of dehydration is greater than 3%.

As a result of their findings, Montain and Coyle (1992b) suggest that one should replace at least 80% of fluid lost as sweat during exercise, in order to avoid the deleterious effects of progressive dehydration on cardiovascular function, thermoregulation and perception of effort. The researchers showed that fluid ingestion at rates close to exercise sweat rate are both possible and beneficial in cyclists. However, it has been observed that

athletes, especially endurance runners, voluntarily ingest considerably less fluid than required to replace fluid lost by sweating (Noakes et al, 1988). Most runners voluntarily ingest approximately 500-600 ml/h (Noakes et al, 1988; Lindeman, 1991), which, if they are sweating between 1000ml/h and 1500ml/h, constitutes only 33%-60% of the volume of fluid lost through sweating. This "voluntary dehydration" is progressive, and will adversely affect thermoregulation and cardiovascular function and therefore, possibly performance.

It is possible to explain why the rate of voluntary fluid ingestion during exercise, especially running, is less than the sweat rate. Firstly, human dipsogenic drive is an inaccurate measure of the state of hydration of the athlete (Dill, 1933; Pitts, 1944; Rothstein et al, 1947). Secondly, high rates of fluid ingestion result in an increased gastric volume which could cause gastric discomfort and hence a decrease in voluntary fluid ingestion, especially in runners unused to the ingestion of large fluid volumes during exercise (Noakes, 1993).

One of the aims of fluid replacement is to maintain plasma volume. Montain and Coyle (1992b) showed that incomplete fluid replacement can result in an increased heart rate due to a decreased plasma volume. Earlier, Nose et al (1988b) had demonstrated that the ingestion of a 77mEq/l sodium chloride solution, *during recovery* from exercise-induced dehydration, caused plasma volume expansion. Producers of sports drinks

have begun to include palatable amounts of sodium chloride in the beverages, in the hope that sodium chloride ingestion would help maintain plasma volume *during exercise*, by increasing osmotic pressure in the intravascular space. However, research has shown that these sports drinks, containing between 10mEq/l and 25 mEq/l of sodium chloride, have no greater effect on plasma volume during exercise than does the ingestion of plain water (Barr et al, 1991).

Therefore, the aim of Part 1 of this thesis was to determine whether the ingestion of a concentrated sodium chloride solution (100mEq/l) would expand plasma volume when fluid was ingested at approximately half the rate at which it was being lost as sweat.

The design was intended to simulate the performance condition in which athletes, in this case cyclists, only replaced 50% of their volume of sweat. The expected volume of sweat lost during a 90 minute cycle at 65% of VO_{2max}, the exercise model for this study, was determined in a preliminary trial.

Fluid ingestion usually only matches sweat rate when athletes are forced to ingest fluid beyond their perceived need to drink. In a study by Barr et al (1991), either water or a 25mEq/l saline solution was ingested to match sweat rate of 1 l/h. A marked diuresis of approximately 400 ml urine/h, which approximated 40% of the ingested fluid volume, was observed. The high

rate of urine loss would seem to defeat the purpose of fluid replacement. The diuresis may be caused by the tendency for plasma osmolality and plasma sodium concentration to decrease when water is consumed in large quantities (Barr et al, 1991). Pearcy et al (1956) found a similar diuresis in men drinking water in volumes matching sweat rate during a 25 hour period in the desert, including 4 hours of marching. However, they found that if sodium chloride was replaced in quantities approximating that lost in the sweat, then an inappropriate diuresis did not occur.

During recovery from dehydration, the ingestion of a concentrated sodium chloride solution (77 mEq/l) resulted in less urine production than did the ingestion of a similar volume of either water (Nose et al, 1988b) or a dilute saline solution (Nielsen et al, 1986).

The aim of Part 2 of this thesis was to determine the concentration of sodium chloride ingestion required to decrease diuresis during exercise in cool conditions (20°C), when the rate of fluid replacement matched the sweat rate.

The design in Part 2 was chosen specifically to achieve the aim of the study. Cool conditions (20°) and low intensity exercise of 55% of VO_{2max} for 4 hours, a low enough sweat rate, so that matching sweat rate with fluid replacement would not cause gastric discomfort in the subjects. This enabled the concentration of sodium chloride required to be determined

independent of volume.

Sodium ingestion during exercise has been shown to increase plasma sodium concentration and therefore, plasma osmolality (Nielsen, 1974; Snellen and Mitchell, 1972). At rest, the ingestion of sodium-containing solutions decreases sweat rate (Snellen and Mitchell, 1972). An increase in plasma sodium concentration prior to exercise will alter thermoregulation during subsequent exercise by increasing the threshold for the onset of sweating (Fortney et al, 1984; Greenleaf et al, 1974; Greenleaf et al, 1976; Kozlowski et al, 1980; Nielsen, 1973; Nielsen, 1974). During exercise in the heat, ingesting sodium containing solutions increases core temperature (Harrison et al, 1978). Montain and Coyle (1992b) have shown a direct relationship between the plasma osmolality and core temperature during 2 hours of cycling exercise. They suggest that fluid replacement during prolonged exercise should prevent an increase in plasma sodium concentration and osmolality, and therefore, an increase in core temperature. It is possible then, that the ingestion of excessive amounts of sodium chloride could have a detrimental effect on thermoregulation.

In summary, the ingestion of sodium chloride during prolonged exercise may be beneficial to the overall fluid balance by maintaining plasma volume (Nose et al, 1988b) and decreasing diuresis (Pearcy et al, 1956), but it may be detrimental to temperature regulation (Montain and Coyle, 1992b). This thesis will examine each of these beneficial effects of sodium ingestion in two separate studies.

CHAPTER 2: REVIEW

2.1 INTRODUCTION: THE HISTORICAL DEVELOPMENT OF SPORTS DRINKS

The ergogenic effect of adequate fluid ingestion during prolonged exercise was largely ignored by competitive long-distance athletes during the first part of this century (Noakes,1993). The International Amateur Athletics Federation (IAAF) ruling at that time was that during a marathon, runners could only take their first drink at 15km and then every 5 kilometres thereafter (Noakes,1993). It was thought, by coach and athlete alike, that to complete a marathon without ingesting any fluid was testimony to their fitness (Noakes,1993).

It was not until studies by Wyndham and Strydom (1969) and Pugh et al (1967) were interpreted as showing that exercise-induced dehydration leads to hyperthermia, that the IAAF changed its rules to allow unrestricted ingestion of fluids during long distance footraces. The possibility that performance would be diminished by progressive dehydration in the absence of adequate fluid ingestion, was not initially considered.

Recent studies have challenged the interpretation of the study by Wyndham and Strydom (1969) that hyperthermia is a common sequela of dehydration. Firstly, dehydration of greater than 7% is necessary for an athlete to develop heatstroke; this level of dehydration is rarely observed in marathon runners (Noakes, 1991b). Secondly, Gisolfi and Copping (1974)

demonstrated that, during treadmill running, a 5% dehydration accounted for only a 0.8°C increase in rectal temperature compared to rectal temperature when euhydrated. Thirdly, Noakes et al (1991a) have further shown that metabolic rate, rather than percent dehydration, is the major determinant of rectal temperature in marathon running. It follows therefore, that athletes competing in middle distance races, with a higher metabolic rate because of higher running speeds, have a greater chance of developing hyperthermia than athletes competing in marathons. Therefore, a 5% level of dehydration, which may occur during distance running, should not lead to heatstroke unless the athlete is running fast and therefore, already has a high metabolic rate (Gisolfi and Copping, 1974).
Although an athlete is unlikely to become hyperthermic with dehydration of less than 7%, a recent study by Montain and Coyle (1992b) has shown that while exercising at 70% of VO_{2max}, any level of dehydration is detrimental to thermoregulation. They showed that for every kilogram of weight loss during exercise due to sweating, oesophageal temperature increased by 0.3°C.

It is clear that fluid ingestion would be beneficial in limiting an increase in core temperature, but might not prevent hyperthermia when the intensity of the exercise is high.

Further, it has been shown that the ingestion of carbohydrate containing beverages during prolonged exercise, delays fatigue (Coyle et al.,1986). Other studies have confirmed the ergogenic effect of the ingestion of

carbohydrates during prolonged exercise (Coggan and Coyle, 1991; Coyle and Montain, 1992). More recently, the inclusion of sodium chloride in a carbohydrate beverage has been shown to improve fluid and carbohydrate absorption, improve palatability, expand plasma volume and decrease urinary fluid loss (Gisolfi et al, 1990b; Hubbard et al, 1990; Nadel et al, 1990; Pearcy et al, 1956).

These findings have resulted in the development of commercial beverages specifically for ingestion during and after exercise. The drinks have became known as "sports drinks".

2.2 FLUID AND ELECTROLYTE BALANCE

Prolonged exercise stresses the fluid and electrolyte balance of the body. This occurs due to the regulation of body temperature by sweating.

2.2.1 BODY WATER COMPARTMENTS

The total water content of an average man weighing 70kg is approximately 42 litres. This volume is divided into two major compartments by membranes. These are known as the intracellular and the extracellular compartments. About 26 litres of the 42 litres is found inside approximately 75 trillion cells (Guyton, 1981; Pivarnik, 1989). This is collectively known as the intracellular fluid. The fluid found outside the cells is called the extracellular fluid and makes up the remaining 16 litres. The extracellular fluid can be further subdivided into: plasma (3 l), interstitial fluid (10 l), and transcellular fluid (3 l).

The interstitial fluid is situated in the spaces between the cells. It is found mostly in a gel phase, with approximately 1% being in sol phase. Cerebrospinal fluid, ocular fluid, fluid in the joints and potential spaces, and digestive secretions comprise the transcellular fluid (Pivarnik, 1989; Guyton, 1981).

Values for extracellular volume, plasma volume, and total body water have been obtained using dye dilution techniques (Prentice et al, 1952; Ikkos et al, 1956; Von Porat, 1951). Intracellular volume is calculated by subtracting the extracellular volume from the total body water. Absolute volumes of the fluid compartments differ from person to person, but the relative proportions of the various compartments are fairly constant.

2.2.2 ELECTROLYTE COMPOSITION OF BODY FLUIDS

The major electrolyte constituents of the extracellular fluid are sodium (Na) and chloride (Cl) ions, and bicarbonate (HCO^-_3). Small quantities of potassium, calcium, magnesium, phosphate, organic acids and sulphate are also present (Guyton, 1981). The concentrations of the electrolytes in the plasma and the interstitial fluid are approximately the same. The concentrations of sodium and chloride are on average 142 mEq/l and 101 mEq/l, respectively.

Sodium is the major extracellular cation and the total exchangeable sodium in adult males, calculated from sodium isotope dilution studies, is ±41.9

mEq/kg body weight or ±17.5 mEq/cm height (Forbes and Perley, 1951). The coefficient of variation of these results is 13.4% and 8.3% respectively. A 70 kg man therefore has 2933 ± 381 mEq (approximately 68g) of exchangeable sodium in his body.

The intracellular ionic concentrations are vastly different from the extracellular concentrations. The major intracellular electrolytes are potassium and magnesium, with concentrations approximating 160 mEq/l and 35 mEq/l respectively. The concentrations of Na and Cl in this space are approximately 10 mEq/l and 2 mEq/l respectively.

Although the ionic compositions of the intracellular and extracellular compartments differ, the total number of molecules in each compartment, relative to the volume of that compartment, is similar. Therefore, each compartment exerts a similar osmotic pressure of approximately 290 mosm/kg H_2O. The intra- and extracellular compartments are separated by a semi-permeable membrane, which allows free movement of water only, across osmotic gradients that may exist as a result of a difference in solute concentration. Hence it is the solute content of each of the fluid compartments that determines the distribution of fluid between the compartments.

2.3 SWEAT

2.3.1 SWEAT GLAND MORPHOLOGY

Three types of sweat glands have been described in the literature (Sato et al, 1989). They are apocrine glands thought to serve a redundant sexual function in humans (Elizondo, 1977), apoeccrine sweat glands found only in the axilla involved in axillary sweating (Sato et al, 1989), and most importantly, eccrine glands which are the true thermal sweat glands.

Eccrine glands are found ubiquitously on the body surface, except for the lips, external auditory meatus and particular glabrous areas of the genitals (Sato et al, 1989). Between 2 and 4 million eccrine glands are found in the average human skin in various densities over the skin surface (Sato et al, 1989; Fortney, 1985). Each sweat glands consists of a simple tubular structure made up of a secretory coil and a duct. The duct forms a spiral in the epidermis and stratum corneum and opens directly onto the skin surface. The secretory coil is composed of three types of cells - clear (secretory) cells, dark (mucoid) cells and myoepithelial cells. Clear cells secrete precursor sweat. The function of the dark cells is still unknown. The myoepithelial cells are thought to provide mechanical support for the secretory coil wall, in order to withstand increases in luminal hydrostatic pressure when precursor sweat is secreted (Sato et al, 1989).

2.3.2 THE MECHANISM OF SWEAT PRODUCTION

Sweat glands are innervated mainly by postganglionic cholinergic fibres. Recent evidence suggests that adrenergic fibres may also innervate sweat glands. This is supported by the presence of β and α adrenergic receptors in eccrine sweat glands (Sato et al, 1989). Neural input and the presence of calcium ions are essential for sweat secretion (Sato et al, 1989).

Sweat secretion occurs in two stages (Sato et al, 1989; Fortney, 1985). Initially, cholinergic and calcium-stimulated active secretion of Na into the secretory coil of the sweat gland occurs. Water passively follows Na secretion. This luminal fluid is known as precursor sweat and is isotonic with plasma irrespective of the sweat rate. Then, Na cations are actively reabsorbed, without passive reabsorption of water. The luminal fluid, now called sweat, is hypotonic to plasma. Since the reabsorptive capacity of the sweat gland duct is limited and dependant on the duration that the luminal fluid stays in contact with its cells, the sweat sodium concentration is proportional to the sweat rate.

2.3.3 ELECTROLYTE COMPOSITION OF SWEAT

Sweat Na concentration has been reported to vary between 20 mEq/l at low sweat rates, and 100 mEq/l at nearly maximal sweat rates (Sato et al, 1989; Sato, 1977). Cl concentration in sweat also rises with increasing sweat rate, but is usually 20 to 25 mEq/l lower than that of the Na concentration in

both precursor sweat and sweat (Sato et al, 1989). The Na/Cl ratio in sweat has been reported to range between 1.0 and 1.4 (Candas and Botheral, 1990). Sweat potassium (K) concentration is around 5 to 7 mEq/l and is almost the same as its concentration in the plasma. The concentration of K in the sweat does not vary with sweat rate. Sweat also contains HCO_3 anions (15-20 mEq/l), lactate (10-15 mEq/l) and urea (15-25 mg/dl) (Sato et al, 1989). The osmolality of sweat is between 80-150 mOsm/kg H_2O (Sato et al, 1989).

2.3.4 FACTORS AFFECTING THE SWEAT RATE AND SWEAT IONIC COMPOSITION

Since sweat sodium and chloride concentrations are dependent on the sweat rate, factors that affect sweat rate will also affect their concentrations in the sweat.

i) Neural Control

Both the magnitude of heat produced by the body during exercise and the environmental thermal load influence sweat rate. The weighted sum of the core and skin temperature determines the sweat rate. Changes in core temperature have about 10 times the influence on sweat rate as do similar changes in the mean skin temperature (Nadel, 1979). These two temperatures are integrated by the anterior hypothalamus (Fortney, 1985). At a specific skin and core

temperature, which depends on the work rate and the environmental conditions, sweating is initiated and increases linearly with increases in core temperature. The temperature at which sweating starts is called the sweating threshold and the rate at which the sweat rate increases in relation to increasing core temperature is called the sweating sensitivity.

ii) Hormonal Control

a) Aldosterone increases ductal Na reabsorption and therefore decreases the concentration of Na in the sweat.

b) It remains to be established whether Vasopressin/ Antidiuretic Hormone (ADH) influences Na reabsorption in the sweat duct (Sato et al, 1989; Fasciolo et al, 1969). It has been suggested that ADH might have a direct effect on hypothalamic control of sweating (Senay, 1979; Fortney et al, 1981) or skin blood flow (Fortney, 1984).

iii) Plasma Osmolality

A 5% increase in plasma osmolality can delay the onset of cutaneous vasodilation and sweating independently of blood volume (Fortney, 1984; Nielsen, 1974). The mechanism is postulated to be due either to a change in sweat gland function, or to a direct effect of increased osmolality on the hypothalamus (Fortney, 1985). However, these

studies used a hypohydration model in which the subject's osmolality was altered prior to the commencement of exercise. During exercise-induced dehydration, the onset of sweating would have occurred well before plasma sodium concentrations reached the levels described by Fortney (1985). Therefore, during endurance exercise, elevated plasma sodium concentrations should have little effect on the sweat rate.

iv) Blood Volume

Fortney (1981,1984) has shown that a decrease in blood volume results in a decrease in sweating sensitivity. This means that the increase in sweat rate for a given increase in core temperature is attenuated. Fortney (1984) showed that maintenance of blood volume, using blood volume expansion, even when whole body dehydration exists, results in maintenance of forearm blood flow and sweating sensitivity. This suggests the importance of the maintenance of blood or plasma volume during exercise for optimum thermoregulation.

v) Skin Wetness

Skin wetness has been shown to decrease sweating. This is sometimes known as hidromeiosis or sweat gland fatigue (Fortney, 1985). It is thought that increasing skin wetness results in the swelling of epidermal cells resulting in the blockage of the sweat ducts (Frye et

al, 1983; Brown et al, 1965).

vi) Exercise training and heat acclimatisation

Exercise training and heat acclimatisation both result in a decrease in the threshold for sweating and an increase in sweating sensitivity. At a given temperature above the sweating threshold, there will be a larger sweat rate than before training and heat acclimatization (Nadel, 1979). Training appears to have a greater effect on sweating sensitivity than heat acclimatisation; while heat acclimatisation appears to have a greater effect on lowering the threshold for sweating than does exercise training (Nadel, 1974). It is postulated that these effects may be due to an increased sweat gland size (Sato, 1977), or altered central neural processing of thermal signals (Fortney, 1985).

Therefore, after training and acclimatisation, the resultant increased sweat rate leads to a concomitant decrease in the concentration of Na in the sweat. However, during exercise, an acclimatised individual will still lose a greater amount of Na than a non-acclimatised individual, because the increase in sweat rate is greater than the decrease in sweat Na concentration (Candas and Botheral, 1990).

2.4 THE EFFECT OF SWEATING ON FLUID AND ELECTROLYTE BALANCE

Sodium and chloride are the major electrolyte constituents of sweat. Therefore, fluid lost by sweating, will deplete the Na and Cl stores of the body, largely found in the extracellular space (Costill and Miller, 1980), to a far greater extent than any other electrolyte.

The total exchangeable sodium in an 70 kg, adult male is approximately 3000 mEq (68 g). A sweat rate of 1 l/h with an average Na sweat concentration of 50 mEq/l (Sato et al, 1989) would mean a loss of 50 mEq of Na per hour. An endurance event of between 2 and 4 hours, will result in a 4% to 7% deficit in body sodium stores if no sodium is replaced during the race.

Sweat is hypotonic in comparison to extracellular fluid, with relatively more water than electrolytes being lost from the body. Heavy sweating will cause the electrolyte concentration of the plasma and interstitial fluid to increase. Costill and Miller (1980) suggested that the need to replace water is of more importance than the need for electrolyte replacement while sweating. However, as will be discussed later, full replacement of body water loss during exercise is dependent on the replacement of the lost electrolytes.

2.5 INGESTION OF SODIUM CONTAINING SOLUTIONS AND FLUID/ELECTROLYTE BALANCE

2.5.1 INVOLUNTARY DEHYDRATION

Studies examining *ad libitum* drinking during exercise or during recovery from dehydration, show that voluntary ingestion of water does not match the total sweat loss or sweat previously lost (Rothstein et al, 1947; Dill, 1933; Dill et al., 1973; Nose, 1985,1986,1988b; Costill and Spark, 1973; Morimoto, 1981; Pitts, 1944; Sohar et al., 1962; Myhre et al, 1985). Despite the voluntary ingestion of water during exercise, progressive dehydration occurs. This has become known as involuntary dehydration.

While studying thermoregulatory differences between his dog and himself, Dill (1933) noted that during prolonged walking, the dog voluntarily replaced all the water it lost. Dill himself voluntarily ingested less water than he lost due to sweating. The dog was found only to have lost water, while Dill lost water and salt in his sweat. Dill concluded that thirst was primarily a function of the sodium concentration in the plasma rather than plasma volume. He proposed that the amount of water drunk was intrinsically determined by that required to maintain a constant osmolality. If sweat had a high NaCl content, less water would be needed to reach a normal osmolality.

The failure to retain ingested water without the replacement of sodium chloride was first suggested by Ladell (1965). Costill and Sparks (1973) showed a more rapid recovery of plasma volume deficit when subjects drank a glucose-electrolyte beverage during recovery from exercise than if they drank only water. Morimoto et al (1981) observed that the degree of involuntary dehydration was reduced when a glucose-electrolyte solution was ingested, rather than water, after thermally-induced dehydration. This suggests that more fluid will be ingested if the beverage contains carbohydrate and sodium chloride. It is difficult to determine whether the increased ingestion of fluid was due to sodium chloride replacement or improved palatability with the inclusion of glucose.

Nose et al. (1985,1986) demonstrated that the degree of involuntary dehydration in rats was less when they ingested water, as opposed to water containing sodium, during a bout of thermally-induced dehydration. It was proposed that involuntary dehydration is caused by the dilutional inhibition of drinking due to the loss of electrolytes. Further, it was suggested involuntary dehydration was a protective mechanism preventing the body from becoming hypotonic due to the intake of water.

Nose et al (1986) showed that a proportionately large amount of ingested water is retained in the intravascular space of rats when water only is ingested. They suggested that the volume-dependent component of the dipsogenic drive is compounded by the resultant decrease in osmolality of

the intravascular space.

Nose et al (1988b) subsequently studied the role of osmotic factors in involuntary dehydration in humans. Subjects were observed during a 3 hour period of rehydration with either water or a 0.45% sodium chloride beverage (77mEq/l sodium chloride was provided in gel capsules), following an exercise-induced dehydration of 2.3%. The cumulative volume of salt solution ingested *ad libitum* by the subjects was greater than that of water. Urine output measured during the rehydration period in which water was ingested tended to be greater than during rehydration with the salt solution. The urine output in the water trial may be due to the greater expansion of plasma volume and a decrease in renin activity (Nose et al, 1988c). Renin is involved in the renal retention of fluid and electrolytes. The decrease in plasma renin activity associated with the water trial, caused fluid loss even though the body was not fully rehydrated (Nose et al, 1988c). Net fluid gain was significantly greater during ingestion of the salt solution. This is explained by the relatively decreased fluid intake and increased urine output that occurred with the ingestion of water. The implication is that the degree of involuntary dehydration is less when sodium chloride is ingested during rehydration.

The study by Nose et al (1988b), demonstrated that the rate of *ad libitum* fluid ingestion decreased, and the volume of urine produced increased, as the overall fluid and cation balance of the body approaches isotonicity.

The corollary of this finding is that the degree of involuntary dehydration is determined by the cation deficit.

These findings confirm the importance of replacing sodium chloride, the major electrolyte component lost in sweat, in order to achieve cation balance and full body fluid rehydration.

The study by Nose et al (1988b) also showed that there is both an osmotic and volume-dependent drive to drinking. During rehydration with water, the osmotic drive to drink is reduced whereas during rehydration with a saline solution, the volume-dependent drive to drink is reduced. However, involuntary dehydration is less with saline than with water replacement (Nose et al, 1988b). This suggests that the osmotic drive to drink is stronger than the volume dependent drive to drink. Sodium chloride replacement therefore, becomes important in the attenuation of involuntary dehydration.

2.5.2 URINE OUTPUT

In a study as early as 1947, Adolph noted that while working in the desert, men drinking water and salt gained 110g while those drinking only water lost 600g. The salt group lost less urine than the group receiving only water. In a later study by Pearcy et al. (1956), subjects exercised intermittently over 25 hours in simulated desert heat. They followed a set drinking regime, of either water or a saline solution, in volumes sufficient to approximate that lost by

sweating. The saline solution contained sodium chloride in amounts approximating that which was lost in the sweat. At the end of the trial in which they consumed saline solution, the subjects had lost approximately 0.5 kg of body weight, and had a small positive sodium chloride balance. However, with the ingestion of water alone, there was a marked diuresis and weight loss of approximately 1.5 kg. The loss of large volumes of urine defeats the purpose of fluid replacement.

More recently, Nielsen et al (1986) found that subjects ingesting a sodium solution with a concentration of 128 mEq/l, lost less urine during two hours of rehydration than those that received drinks with a lower sodium concentration of 43 mEq/l.

These findings suggest that when drinking volumes equal to sweat production, optimum fluid balance might only be possible if sodium chloride balance is positive. As yet, no studies have established the optimum concentration of sodium chloride required to maintain fluid balance and minimise urine output during exercise.

2.5.3 THE HYPONATREMIA OF EXERCISE

Several early studies showed that laborers developed heat cramps when replacing sweat lost during work, with water alone (Ladell, 1949; Moss, 1923; Talbott, 1935; Talbott and Michelsen, 1933; Thrower, 1928). The incidence of these cramps was apparently reduced by the ingestion of salt containing solutions.

Although some of these studies (Ladell, 1949; Talbott, 1935; Talbott and Michelson, 1933) noted a decrease in serum osmolality in subjects with heat cramps, there is no firm evidence that a relationship between "heat cramps" and sodium deficiency exists (Noakes, 1992) since the scientific design of these studies was not sound. It is important however, that the consumption of large volumes of plain water can lead to decreases in both serum electrolyte concentrations and osmolality.

An increased number of cases of exercise related, symptomatic hyponatremia have been reported in the literature (Frizzell et al, 1986; Hiller et al, 1985; Irving et al, 1991; Noakes et al, 1985, 1990) since the IAAF altered its rules regulating drinking during prolonged exercise, and the number of entrants participating in ultra-distance events has increased. In these cases, the athletes have ingested large volumes of plain water. Despite a decrease in plasma sodium concentration and plasma osmolality, there has been inappropriate retention of ingested fluid by the body. Fluid overloads of between 4 and 12 litres have been reported in athletes admitted to

hospital with symptomatic hyponatremia (Noakes, 1992). It has been hypothesized that an inappropriate hormonal response occurs in these individuals in response to the fluid overload (Noakes, 1992), resulting in fluid retention.

In a study by Barr et al (1991), subjects cycled for 6 hours at a mild workload in the heat. The subjects received either water or a sodium chloride solution of 25 mEq/l in volumes matching sweat rate. Subjects sweated at rates of approximately 1100 ml/h. Serum sodium concentrations fell only slightly in the group ingesting water and remained at pre-exercise values in the group ingesting the sodium chloride solution during exercise. This suggests that serum sodium concentration is well regulated by the body and that exercise-induced hyponatremia is probably not due to the lack of salt replacement.

It is suggested that the development of symptomatic hyponatremia is not caused by the loss of large amounts of sodium chloride in sweat, but rather due to an inappropriate response to the ingestion of a large volume of plain water in predisposed persons (Barr et al 1991, Irving et al 1991). Whether the ingestion of sodium chloride in sports drinks during prolonged exercise will reduce the potential of susceptible individuals to develop symptomatic hyponatremia, is unclear. However, once hyponatremia has developed, the appropriate treatment is fluid restriction.

2.5.4 INTESTINAL FLUID ABSORPTION

The triple lumen tube technique is the preferred method for measuring net fluid and electrolyte absorption from the small intestine (Gisolfi et al, 1990). It is noted however, that one disadvantage of this technique is that only a 20cm - 40cm portion of the gut is perfused and therefore, what occurs in the rest of the small intestine has to be extrapolated from these results.

The length of the small intestine, from the pylorus to the ileocecal valve, has been quoted on average to be 300cm long in the living human (Ganong, 1987). Although fairly short in length, the convolutions of the villi and microvilli provide a surface area for absorption of approximately 2 million cm^2 (Ganong, 1987). The rate of fluid and sodium absorption for a given perfusate has been shown to be greater in the jejunum than in the ileum (Schedl and Clifton, 1963).

The absorption of water requires the presence of sodium in a concentration close to 140 mEq/l. If sodium is not present at this concentration in the perfusate, then it will be provided by the intestinal juices (Gisolfi et al, 1990) or possibly by the extravascular fluid (Noakes, 1993). This suggests that the inclusion of sodium in the sports drink might enhance the rate of fluid absorption in the presence of carbohydrate.

A recent study by Gisolfi et al (1991) has shown that the addition of sodium chloride to water in the absence of carbohydrate, does not enhance water

or sodium absorption either at rest or during exercise. Isotonic sodium might even decrease the rate of fluid absorption as shown by Schedl and Clifton (1963) who compared the rate of absorption of water and Ringer's solution. Further, Lee et al (1955) found that a hypertonic sodium chloride solution decreases water absorption compared to water alone.

However, it has been shown that a carbohydrate-electrolyte perfusate, with an osmolality that is hypotonic or isotonic with respect to plasma, results in approximately a 6 fold increase in the rate of water absorption compared to an isotonic electrolyte beverage or water (Gisolfi et al, 1990, 1991; Leiper and Maughan, 1986). Further, the addition of carbohydrate to an electrolyte drink enhances sodium absorption primarily via "solvent drag" (Fortran, 1975). It also appears that in the presence of carbohydrate, a 120 mEq/l sodium concentration will result in a greater rate of sodium absorption than a 80 mEq/l sodium concentration (Fortran, 1975). Fortran (1975) also showed that chloride is the preferable anion to have present if sodium absorption is to be enhanced. Murray (1987) suggests that a maximum rate of water absorption occurs when intestinal sodium concentration is 90-120 mEq/l and glucose concentration is 60-160 mM. Lifshitz and Wapnir (1984), and Modigliani and Bernier (1971) showed that the optimum ratio of sodium to glucose needed to optimize water absorption is 2:1.

Therefore, although sodium is necessary for the absorption of water and carbohydrate, the addition of sodium alone to the perfusate will not

improve water absorption. However, carbohydrate will stimulate absorption of both sodium and water. Thus, if sodium is to be added to sports drinks in high concentrations, it should be added in the presence of carbohydrate.

It is suspected that exercise decreases splanchnic blood flow and affects water and sodium absorption from the small intestine (Rowell et al, 1986). However, Sjovall et al (1983) found that splanchnic nerve stimulation decreases blood flow to the muscularis and crypts of the intestinal wall without affecting blood flow to the villi, the absorptive sites. Thus if absorption is decreased during exercise, it may not be due to a decreased blood flow to the villi.

Barclay and Turnberg (1988) and Gisolfi et al (1991), using the triple-lumen technique, have both shown that, compared to rest, mild exercise decreased fluid absorption slightly when either plain water or an isotonic electrolyte beverage was perfused. However, when isotonic carbohydrate-electrolyte beverages were perfused, strenuous exercise (Fortran and Saltin, 1967) or exercise at 30%, 50% or 70% of VO_{2max} did not decrease water or sodium absorption from the small intestine.

Gisolfi et al (1991) proposed that the 6-10 fold increase in sodium and fluid reabsorption that occurs in the presence of carbohydrate, masks the small decrease in intestinal absorption that mild exercise might cause in the

absence of carbohydrate.

Therefore, the literature suggests that if carbohydrate is present in a sports drink, optimal absorption of water and sodium will occur in mild, moderate or severe exercise.

2.6 DISTRIBUTION OF BODY FLUID DURING EXERCISE

During prolonged exercise, several fluid shifts occur. Initially, fluid shifts in response to the onset of exercise. Subsequently, a response to the continuous loss of fluid and electrolytes, mainly sodium and chloride due to sweating, occurs. This latter response is dynamic, depending on the volume and type of fluid ingested during exercise, and determines, to a large extent, the fluid shifts at the end of exercise.

2.6.1 MEASUREMENT OF FLUID COMPARTMENT VOLUMES

Direct and indirect methods have been used to measure the volume and changes in volume of the fluid compartments in the body.

Dye dilution techniques have been used to measure directly the volume of the various compartments. Tritium is used to measure total body water (Prentice et al, 1952), inulin or thiosulphate the extracellular space (Ikkos et al, 1956; Cardozo and Edelman, 1952), and Evans Blue the plasma volume

(Von Porat, 1951). However, these methods require the individual to be stationary and often supine. This makes these techniques difficult to use when fluid shifts are to be measured in the exercising individual. Indirect techniques have been used to obtain approximations of the changes in volumes of the different compartments during exercise. The results from these indirect techniques should be regarded only as an approximation, because they have large coefficients of variation. However, they are useful for identifying relative fluid shifts during exercise.

Changes in the haematocrit and haemoglobin content of the blood are used to calculate the relative percentage change in plasma volume (Dill and Costill, 1974). By applying these relative percent changes to an estimated plasma volume (Retzlaff et al, 1969) the relative change in plasma volume can be calculated.

The chloride method has been used to calculate estimated changes in extracellular and intracellular volumes following dehydration (Costill et al, 1976, 1981; Nose et al, 1988a, 1988b). This method assumes that the resting membrane potential (RMP) remains unchanged and that 95% of the chloride is present in the extracellular space. According to Costill et al (1984), RMP after 2 hours of running at 50% VO_{2max}, increased by 6mV in working muscle whith no change in the inactive muscle RMP. Therefore, estimations of fluid shifts *during* prolonged exercise, using the chloride method, might only provide an idea of fluid shifts between compartments.

However, after 30 minutes recovery, the RMP of the active musculature returns to normal (Costill, 1984). Thus, interpretation of the results from the chloride method are more accurate *after* recovery.

2.6.2 FLUID SHIFTS OCCURRING AT THE ONSET OF EXERCISE

As exercise commences, there is a movement of fluid out of the intravascular space (Harrison, 1985; Senay and Pivarnik, 1985; Costill, 1977; Sjogaard and Saltin, 1982). This movement of fluid is caused by an alteration in the equilibrium of hydrostatic and oncotic forces, known as Starling forces, on the plasma and interstitial spaces of the capillary beds (Harrison, 1985; Ganong, 1987, pp 489). Factors that influence the equilibrium of Starling forces, and therefore, the amount of fluid shifting out of the intravascular space include: posture, exercise mode, exercise intensity, environmental conditions, state of training and pre-exercise hydration status (Harrison, 1985; Senay and Pivarnik, 1985). The increase in muscle perfusion pressure, which accompanies the onset of exercise, promotes increased filtration of fluid out of the intravascular space down the pressure gradient (Sjogaard and Saltin, 1982).

Muscle biopsies taken from active muscle 10 minutes after the initiation of exercise, show an increased water content (Costill, 1977). Fluid leaves the intravascular space in the region of the active muscle and enters the interstitial space surrounding the active muscle (Costill et al, 1981). Dumping

of hydrogen ions and other exercise metabolites, by the muscle cells, into the surrounding interstitial fluid is also thought to promote the movement of fluid out of the intravascular space (Lundvall, 1972). At inactive muscle sites, fluid content of the interstitium might stay the same (Costill, 1984) or fluid might be absorbed into the intravascular compartment (Harrison, 1985). Vasoconstriction of the capillaries in inactive muscle would favour the reabsorption of fluid into the intravascular space. However, since a larger proportion of the cardiac output goes to the active muscles, a net movement of fluid out of the intravascular space is seen (Harrison, 1985).

As a result of the responses described above, a decrease in plasma volume of on average 13% is usually observed at the onset of exercise at an intensity of 70% VO_{2max} (Costill, 1984).

2.6.3 FLUID DISTRIBUTION AFTER DEHYDRATION

The loss of hypotonic sweat from the body during progressive dehydration leaves the remaining fluid hypertonic. After exercise-induced dehydration, fluid in the various body compartments redistributes to equilibrate osmotic pressure.

Using the dye dilution technique, Kozlowski and Saltin (1964) showed that after exercise and thermal dehydration, more of the fluid lost in sweat came from the intracellular space than from the extracellular space. However,

these results are more suggestive than definitive because the base line body fluid measurements were not done on the same day as the experimental body fluid measurements.

Costill et al (1976) dehydrated subjects by 2.2, 4.1 or 5.8% of body weight. Using the chloride method, they showed that 30 minutes after the termination of exercise, at a dehydration level of 2.2%, proportionately more fluid was lost from the extracellular fluid. At 4.1% and 5.8% dehydration, the fluid deficit was distributed approximately equally between the extracellular and the intracellular spaces. They noted that regardless of the volume of the total fluid deficit, the plasma volume consistently provided ±10% of the fluid lost. In a further study, Costill et al (1981) use the chloride method after 30 minutes recovery, to show that at a 3% level of dehydration, 6% of the fluid deficit came from the plasma, and 37% and 57% from the interstitial and intracellular spaces respectively.

However, it has been proposed that since the major ions lost in sweat are from the extracellular compartment, the absolute volume of fluid lost from the extracellular compartment is greater than that from the intracellular space (Kozlowski and Saltin, 1964; Costill et al, 1976; Costill et al, 1981; Nose et al, 1988).

Nose et al (1988a) measured fluid shifts occurring after dehydration in humans, using the chloride method. Immediately following mild

dehydration, 19% of the fluid deficit was provided by the plasma and 42% and 39% by the interstitial and intracellular spaces, respectively. After 60 minutes recovery the distribution of the fluid loss had changed slightly, with 11% coming from the plasma volume, and 46% and 43% coming from the interstitial and intracellular spaces respectively. More importantly, they showed that sweat sodium concentration determines the contribution of fluid volume loss by the intracellular space, which in turn determines how effectively plasma volume is maintained during dehydration. If the concentration of sodium in sweat is low, plasma osmolality increases. As a result, there is an increase in the osmotic pressure exerted by plasma in the extracellular space on the intracellular space. Fluid therefore, shifts from the intracellular space into the intravascular space, and plasma volume is better maintained.

The above studies confirm that the distribution of fluid between the intracellular and extracellular space is dependent on the osmotic gradient between the two compartments.

Table 2.1 summarises the decreases in plasma volume that have been noted in dehydration studies. The table shows the change in plasma volume towards the end of exercise. Since the subjects were still exercising, hydrostatic pressures forcing fluid out of the plasma space were still present. The table shows that even at relatively high levels of dehydration, plasma volume is fairly well maintained. It also shows that the change in plasma

volume is influenced by the exercise intensity, the environmental conditions and the duration of the exercise bout.

Table 2.1. Decreases in plasma volume reported in exercise-induced dehydration experiments.

	CONDITIONS	EXERCISE	% DEHYD.	▲PV	REF.
1.	18°C, 50% RH	±3h @ 75% VO_{2max}	4.1%	↓ 3%	Kozlowski et al, 1964
	38°C, 35% RH	±2h @ 50% VO_{2max}	4.7%	↓ 11%	
2.	39.5°C, 25% RH	cycled until desired dehydration reached	2.2% 4.1% 5.8%	↓ 4% ↓ 8% ↓13.7%	Costill et al, 1976
3.	32°C, 65% RH	cycling, 2h, 50% VO_{2max}	1.44 kg	↓ 17%	Francis, 1979
4.	30°C, 46% RH	cycling, 2h, 50% VO_{2max}	2.9%	↓ 9%	Costill et al, 1981
5.	21°C, 65% RH	cycling, 2h	3.5%	↓ 7.9%	White & Ford, 1983
6.	49°C, 20% RH	walking, 70min, ±30% VO_{2max}	5%	↓ 4%	Sawka et al, 1984
7.	34°C	cycling, 4h, ± 85 watts	3%	↓ 6%	Candas et al, 1986
8.	30°C	cycling, 2h, 50% VO_{2max}	3%	↓ 16%	Nielson et al, 1986
9.	22°C	cycling, 2h, 70% VO_{2max}	2%	↓ 9%	Hamilton et al, 1991
10.	30°C, 50% RH	cycling, 4h, 55% VO_{2max}	6.3%	↓ 20%	Barr et al, 1991

PV = plasma volume; dehyd = dehydration; VO_{2max} = maximal oxygen consumption; RH = relative humidity.

2.6.4 FLUID DISTRIBUTION DURING REHYDRATION

The ingestion of sodium solutions will attenuate involuntary dehydration by decreasing urine output and increasing *ad libitum* drinking during rehydration. This section focuses on the effect of sodium ingestion on fluid distribution during rehydration, following a bout of exercise- or thermally-induced dehydration.

Nielsen et al (1986) noted that following a 3% dehydration, plasma volume returned to normal far quicker when subjects ingested beverages that

contained either carbohydrate, or sodium and carbohydrate, compared to a beverage containing potassium. The ionic composition of the different beverages may have affected the distribution of water between the body compartments. However, the slower recovery of plasma volume in the high potassium beverage may have been due to a marked diuresis noted with the ingestion of this beverage (Nielsen et al, 1986).

Nose et al (1988b) followed subjects during *ad libitum* drinking after a period of exercise induced dehydration. The group receiving water with salt capsules - so that the beverage had an effective concentration of 77 mEq/l - replaced 95% of the sodium lost due to sweating. They noted that in this group there was more rapid restoration of the extracellular volume (ECV). Extracellular volume was restored at 180 minutes of recovery in the saline group. In the group ingesting water only, ECV was still significantly depleted at 180 minutes of recovery. This was, in part, the result of a greater volume of fluid voluntarily ingested in the group receiving sodium, due to maintenance of an osmotic drive to drink. Nose et al concluded that the degree of rehydration achieved in each compartment, is determined by the ability to restore the ions lost from that compartment.

2.6.5 THE EFFECT OF SODIUM INGESTION ON PLASMA VOLUME DURING EXERCISE

A few studies have examined the influence of sodium and fluid ingestion on plasma volume during exercise. Beverages with a sodium concentration of between 6 and 25 mEq/l have been used (Barr et al, 1991; Candas et al, 1986; Francis, 1979; Millard-Stafford et al, 1992; Owen et al, 1986; Powers et al, 1990; White and Ford, 1983). None of these studies have examined the effect of drinking on fluid distribution in the other fluid compartments.

In most studies a low sodium concentration drink was found to have had a similar effect as the ingestion of only water, on both plasma sodium concentration and change in plasma volume (Barr et al, 1991; Millard-Stafford et al, 1992; Owen et al, 1986; Powers et al, 1990; White and Ford, 1983). In only one study (Candas et al, 1986) did an isotonic beverage with a sodium concentration of 23 mEq/l result in a relative increase in plasma volume. In this latter study by Candas, there was a relative decrease in plasma volume in the group receiving water. In the experimental protocol, Candas gave the subjects fluid to ingest during the last three hours of a 4 hour bout of intermittent cycling. However, blood was sampled after rest periods, and not while the subject was cycling, which may have affected the results.

There was also no difference in sweat rate or urine output (Millard-Stafford, 1992; Barr et al, 1991; Francis, 1979) if either water or a low sodium containing beverage was consumed. This suggests that a beverage with a sodium concentration of between 5 and 25 mmol/l will have the same effect on fluid distribution and fluid balance as the ingestion of pure water.

2.6.6 FLUID DISTRIBUTION DURING SALINE INFUSION

Fluid replacement studies are affected by variations in rates of intestinal fluid absorption. Some studies have tried to bypass this variable by infusing the fluid replacement solution directly into the plasma. None of these studies have infused water because this could lead to haemolysis and hyponatraemia. In these studies the control group has received a sham infusion.

Deschamps et al (1989) infused 0.9% saline while subjects cycled at 84% VO_{2max} until exhaustion. The mean volume of saline infused was 1.2 litres over an average time of 21 minutes. Infusion maintained plasma volume. The maintenance of plasma volume decreased heart rate and core temperature at exhaustion, but the time to exhaustion was not increased by the infusion. Therefore, plasma volume did not limit performance under these conditions. However, maintaining plasma volume during exercise has been shown to decrease the the rating of perceived exertion (Montain and Coyle, 1992b)

Nose et al (1990) infused 8.75 ml/kg body weight of 0.9% saline between minutes 20 and 50 of a 50 minute bout of moderate exercise in the heat. Plasma volume returned to normal in the infusion group at 50 minutes. They calculated that approximately 50% of the infusate remained in the intravascular space although the intravascular space comprised only 20% of the extracellular space. If the same preferential retention of fluid in the intravascular space occurred when drinking saline, then it would be very difficult to replace the fluid lost from the remainder of the extracellular space. The retention of fluid in the vascular space might switch off the volume dependent dipsogenic drive to drink, cause plasma renin activity to decrease, and increase urine output. This would make maintenance of euhydration during exercise difficult.

2.7 CONCLUSION

The literature shows that fluid balance during exercise can benefit from the inclusion of sodium chloride in the rehydration drink, both during and after exercise. Sodium chloride will have the following effects on fluid balance during exercise:-

- When water and salt capsules (with an effective concentration of ± 75 mEq/l) are ingested, there is improved voluntary fluid replacement during recovery, due to the maintenance of the drive to drink. If sodium chloride is present in the sports drink in a moderate concentration (<25mEq/l) there is an increased palatability of the drink, which may prompt even greater voluntary fluid ingestion.

- There is decreased diuresis during rehydration when sodium is ingested in quantities that maintain positive sodium balance.

- The ingestion of sodium chloride may help in the prevention of exercise-induced hyponatremia in susceptible individuals.

- In the presence of carbohydrates, optimum intestinal fluid absorption occurs when luminal sodium concentration is between 90 and 120 mEq/l.

The range of sodium chloride concentration found in sports drinks (10-25 mEq/l) is sufficient to improve the palatability of the drink and therefore improve fluid balance by increasing voluntary fluid ingestion by the athletes. However, to further improve fluid balance by the mechanisms noted above, sodium chloride needs to be replaced at rates equal to or greater than that lost in sweat. This is 5-10 times the amount of sodium that is currently present in sports drinks. Since a sodium concentration greater than approximately 30 mEq/l is unpalatable, sodium chloride intake from sports drinks would need to be supplemented by salt in tablet form.

Research is needed to evaluate the effect of sodium chloride supplementation on the maintenance of plasma volume during exercise in the heat, when athletes are less able to ingest the volumes of fluid required to match losses. Further, the effects of sodium chloride supplementation on the maintenance of overall fluid balance during prolonged, mild intensity exercise, also need to be examined.

CHAPTER 3: METHODS

Permission for use of human subjects in these experiments was granted by The Research and Ethics Committee of The University of Cape Town Medical School.

3.1 MAXIMAL EXERCISE TEST

3.1.1 TEST PROTOCOL

The protocol described by Hawley et al (1992) was used. After a 5 minute self-paced warm-up, the athlete was connected to the apparatus for measurement of oxygen consumption and commenced cycling at a workload of 3.33 watts/kg body weight. This initial workload was maintained for 150 seconds and thereafter increased by 50 watts for a further 150 seconds. After the second stage, the workload was increased by 25 watts every 150 seconds until volitional fatigue. Verbal encouragement was given to motivate maximum effort.

The number of seconds completed on the last workload was noted and used in the following equation to determine peak power output (PPO).

$$PPO = WL_{final} + (t/150 \text{ seconds} * 25 \text{ watts})$$

Where WL_{final} is the last workload the subject sustained for the full 150 seconds; t is the number of seconds sustained at the final, uncompleted workload of 25 watts.

3.1.2 DETERMINATION OF MAXIMAL OXYGEN CONSUMPTION

While cycling, subjects breathed through a one-way valve (Model 2700, Hans Rudolph Inc., Kansas City, Kansas, USA). A nose clip prevented nasal breathing. The inspiratory end of the Hans Rudolph one-way valve was connected via low resistance piping to a Mijnhart dry gas meter which measured inspired volume. A length of low resistance piping connected the expiratory end of the Hans Rudolph valve to a 15 L perspex mixing chamber containing baffles, in which expired air was collected and mixed. Air from this chamber was continuously pumped by an Ametek R-1 flow control pump, through a condenser, to the pick-up heads of an Ametek S-3A/1 oxygen analyzer and an Ametek CD-3A carbon dioxide analyzer (Thermox Instrument Divisions, Pittsburg, Pennsylvania, USA). Rate of oxygen consumption (VO_2), carbon dioxide production (VCO_2) and respiratory exchange ratio were calculated by an on-line computer (Sperry, Sperry Corporation, Salt Lake City, UT, USA) using software (Treadmill Monitor V1.2(c) 1988 A.R. Technology) based on conventional open-circuit spirometry as described by Scrimgeour et al (1986). The cumulative value for each minute of the test, for each of the above mentioned measurements, was printed out at the end of the test.

Equipment was calibrated before each test. The gas analyzers were calibrated using gases of known composition and the ventilometer was calibrated using a three litre Hans Rudolph syringe (Hans Rudolp Inc, Kansas, Kansas, USA).

3.2 ASSAYS

Blood was collected, without stasis, from an indwelling venous cannula (Jelco No. 20) into lithium heparin Vac-u-test tubes (Radem Laboratory Supplies, Sandton, South Africa). For those assays requiring plasma, the whole blood was spun at 2000 rpm for 15 minutes and the plasma was removed and stored at -20°C until assayed.

3.2.1 URINE, PLASMA AND SWEAT ELECTROLYTE CONCENTRATION

3.2.1.1 CHLORIDE

Chloride concentration was analysed using a Beckman Chloride Reagent Kit (Beckman Instruments, Inc. Galway, Ireland) on a Beckman SYNCHRON AS 80 system. Chloride concentration was determined by a colormetric titration method employing a Beckman chloride electrode and silver anode. The pair of silver detector electrodes detect the presence of silver ions in solution. The SYCHRON system determines the total silver ions required to titrate the chloride, which is directly proportional to the concentration of chloride present in the sample. The total silver ions required to titrate the chloride is directly proportional to the concentration of chloride in the sample.

3.2.1.2 SODIUM AND POTASSIUM

Sodium and potassium concentration was measured with a Beckman Sodium/Potassium Kit (Beckman Instruments, Inc. Galway, Ireland) on a Beckman SYCHRON AS 80 system, in conjunction with ion-selective sodium and potassium electrodes.

3.2.2 TOTAL PLASMA PROTEIN

A Lennon Diagnostics Total Protein Reagent Kit (Port Elizabeth, South Africa) was used on a Beckman SYCHRON AS 80 system. The protein in the sample combines with the copper in the reagent to form a deep blue chelate. The formation of this chelate is followed colorimetrically at 540nm. The rate of change of absorbance is used to determine the total protein content of the sample.

3.2.3 HAEMOGLOBIN

Haemoglobin concentration was measured using the cyanomethaemoglobin method (Hailine, 1958). 20µl of whole blood was mixed with 5ml of Drabkins solution (potassium ferricyanide-potassium cyanide) and left to stand for 15 minutes. When placed in the Drabkins solution, haemoglobin is oxidised and converted to cyanomethaemoglobin. The concentration of cyanomethaemoglobin is determined on the spectrophotometer at 540nm and is proportional to the concentration of haemoglobin originally present in the sample. Each sample was measured in duplicate.

3.2.4 HAEMATOCRIT

Haematocrit was determined by the micro-centrifuge technique. Measurements were made in triplicate and were not corrected for plasma trapped in the packed red blood cells.

3.2.5 PLASMA AND URINE CREATININE

A Beckman Creatinine Reagent Kit (Beckman Instruments, Inc. Galway, Ireland) was used to measure plasma and urine creatinine concentration on a Beckman SYCHRON AS 80 system. The sample was injected into the alkaline picrate reagent, which combines to form a red coloured complex. The resulting change in absorbance is monitored at 520nm. The rate of picrate-creatinine complex formation is directly proportional to the creatinine concentration of the sample.

3.2.6 PLASMA AND URINE OSMOLALITY

Osmolality of urine and plasma was measured by freezing point depression on an Osmomat 030 nanolitre osmometer.

3.3 SWEAT COLLECTION

Sweat was collected by placing a sodium free plastic bag over the subject's forearm (Nose et al, 1988a,b). This method of sweat collection was chosen firstly because we wanted to make serial collections of sweat and with the whole-body washdown method this would not have been possible (Sohar, 1965; Ladell, 1948; Van Heyningen and Weiner, 1952; Vellar, 1982). Secondly, the subject sweated quite profusely, especially in the study in the heat and if the whole-body washdown method had been used, the collection of all the sweat that dripped would have been difficult.

Before placing the bag on the subject's arm, the forearm and hand were washed well with distilled water and then dried thoroughly with gauze swabs. When placed on the arm, excess air was removed from the plastic bag by forcing air out of the open end. The open end was then taped closed around the subject's arm so that the bag was water tight.

After 20 minutes the bag was punctured with a needle and the sweat that had collected aspirated using a syringe. The bag was then removed and the next collection made from the other arm with a new bag. The swopping of arms was to prevent hidromeiosis; a decrease in sweat production as a result of wet skin (Nose et al, 1988a,b).

3.4 RECTAL TEMPERATURE

Rectal temperature was measured with a rectal probe (YSI 400 Probe, Simson Elcetric Co., Elgin, Ill, USA) inserted 10cm into the rectum. The probe was connected to a YSI Telethermometer (Simson Elcetric Co., Elgin, Ill, USA). Temperature was recorded to the nearest 0.1°C.

3.5 HEART RATE

A three lead ECG (Lohmeier M607, Lohmeier, Munich, Germany) was used to monitor heart rate.

3.6 SUBJECT MASS

The subjects recorded their nude weight on a Seca scale (Seca, Germany) to the nearest 100g. Before weighing themselves, the subjects voided their bladders and dried themselves well with a towel to remove any sweat that had not yet evaporated.

3.7 RATINGS OF PERCEIVED EXERTION

Subjects rated their feeling of perceived exertion on the 10 point Borg Scale (Borg, 1982).

3.8 CALCULATED PARAMETERS

3.8.1 PERCENT DEHYDRATION

Percent dehydration was calculated from the change in weight of the subject.

$$\% \text{ DEHYDRATION} = 100 * (W_B - W_A)/W_B \quad (1)$$

Where: W_B = weight before W_A = weight after

3.8.2 SWEAT RATE

Sweat rate was calculated as follows:

$$\text{SWEAT RATE} = (W_B - W_t - U_t + V_t)/t \quad (2)$$

Where: W_B = weight before W_t = weight at time t
U_t = volume of urine voided by time t
V_t = volume fluid ingested by time t
t = time in hours

Corrections for water production and respiratory water loss were not made as these are small and tend to cancel each other.

In our calculations, respiratory water loss was taken as being negligible compared to losses occurring in sweat and urine. For example during exercise at 55% of VO_{2max}, the following respiratory loss would occur if calculated using equations described by Mitchell (1972). These values were taken from one of the trials. At an oxygen consumption of 2,8 l/min and a RER of 0.93, the subject would be losing 1.1 gram of water per minute via the respiratory tract. This amount is small compared to the ± 17 g/min lost due to sweating in those experiments.

Likewise metabolic water production due to the oxidation of carbohydrate and fat would produce an insignificant amount of water compared to the volume being ingested and therefore was not included in the calculation of sweat rate. Pivarnik et al (1984) used the calculations of metabolic water production described by Consolazio et al (1963), to show that while exercising at 56% and 74% of VO_{2max}, metabolic water production provided only 1.7 and 2.4 grams of water per minute, respectively. By comparison subjects in this study ingested 10g or 15g of fluid every minute during exercise.

In addition the amount of respiratory water lost is approximately offset by the metabolic water production, and the difference between the two is too small to be of consequence. Hence they were not taken into account for the calculation of sweat rate.

3.8.3 SURFACE AREA AND ESTIMATED PLASMA VOLUME

Resting plasma volume in ml, was estimated from body surface area (Retz-laff et al, 1969). This method appears to be reliable (International Committee for Standardisation in Haematology, 1980). Surface area for each male subject was calculated from their height and weight (DuBois and DuBois 1916).

$$SA(m^2) = 0.00718 * Ht^{0.725}(cm) * W^{0.425}(kg) \quad (3)$$

$$PV(l) = 1{,}630 * SA \quad (4)$$

Where: SA = surface area PV = plasma volume
Ht = height W = weight

3.8.4 FLUID SHIFTS

Fluid shifts between the plasma volume (PV), interstitial volume (ISV) and intracellular volume (ICV) were calculated using equations developed and validated in previously published studies (Dill and Costill, 1974, Nose et al, 1988a,b, Costill et al, 1976).

Values for plasma volume and plasma electrolyte concentration at 10 or 15 minutes of exercise were taken as the zero value for the calculation of fluid shifts occurring <u>during</u> the exercise bout as the aim was to determine the magnitude of fluid shifts occurring due to fluid loss. Coyle and Hamilton (1990) suggested that "when changes in blood volume are made relative to the volume at rest, rather than relative to the euhydrated blood volume established early in exercise, the actual fluid shifts due to progressive

exercise per se tend to be obscured by the shifts occurring in the transition from rest to exercise." They also state that 10 to 15 minutes is "sufficient time for fluid shifts to occur before significant amounts of fluid (are) lost" due to sweating or urine production (Coyle and Hamilton (1990)).

Calculation of fluid distribution after recovery were made relative to pre-exercise plasma volume and plasma electrolyte concentrations.

Change in total body water was calculated as the sum of sweat loss, urine loss and fluid ingestion. In the first experiment it was assumed for the purpose of calculations, that after 90 minutes of exercise all the fluid ingested during the exercise bout had been absorbed. This is not completely acceptable, but there are no means of determining how much of the fluid had been absorbed and this study was interested in determining trends, rather than absolute values, from the calculations.

In Part Two, for the purpose of the calculation of fluid shifts it was assumed that absorption lagged 15 minutes behind fluid ingestion. The repetitive drinking pattern together with an initial large bolus that was used should have ensured that gastric emptying was not limiting. The calculations of Noakes et al (1991) predict that when following this drinking regime while drinking water, 75% of the gastric volume empties every 10 minutes and eventually matches the volume ingested every 10 minutes. Since carbohydrate-electrolyte solutions that are isotonic or hypotonic and have

a carbohydrate content less than 8% do not slow gastric emptying compared to water (Gisolfi and Duchman, 1992), the above prediction will probably hold true for the beverages ingested in this thesis.

3.8.4.1 SHIFTS IN PLASMA VOLUME

Shifts in plasma volume were calculated from the estimated plasma volume and from the percentage change in plasma volume occurring between time points.

3.8.4.1.1 PERCENT CHANGE IN PLASMA VOLUME

The percent change in plasma volume was calculated from the equations of Dill and Costill (1974) from values of haematocrit and haemoglobin. The calculation is as follows:-

$$BV_A = BV_B\ (Hb_B/Hb_A) = 100\ (Hb_B/Hb_A) \quad (5)$$

$$CV_A = BV_A\ (Hct_A) \quad (6)$$

$$PV_A = BV_A - CV_A \quad (7)$$

$$\Delta PV,\% = 100\ (PV_A - PV_B)/PV_B \quad (8)$$

Where:

A = after	B = before
PV = Plasma Volume	BV = Blood Volume
CV = Cell Volume	$BV_B = 100$
$PV_B = 100 - Hct_B$	

Percent changes in plasma volume occurring during exercise were compared to the value for plasma volume at 10 or 15 minutes of exercise. The percent change in plasma volume after exercise was calculated with respect to the resting plasma volume.

3.8.4.1.2 VOLUMETRIC CHANGE IN PLASMA VOLUME

The change in plasma volume, in ml, was calculated by multiplying the percent change in plasma volume and plasma volume (which was calculated using the equations 3 and 4).

3.8.4.2 SHIFTS IN INTERSTITIAL AND INTRACELLULAR VOLUME

The Chloride Method was used to calculate changes in interstitial fluid volume (ISV) and intracellular fluid volume (ICV) (Costill et al, 1976, Nose et al, 1988a,b).

The following assumptions are required for this method (Nose et al, 1988a):

1. Chloride in the sweat and urine comes only from the extracellular fluid space.
2. The Donnan factor for chloride between the plasma and the interstitial space is 1.05.
3. Chloride loss from the plasma and the interstitial space is proportional to the water loss from each space.

The calculation is as follows:-

$$\Delta Cl^-_{ecv} = Cl^-_{in} - Cl^-_{u} - Cl^-_{s} \quad (9)$$

$$\Delta Cl^-_{ecv} = \Delta Cl^-_{isv} + \Delta Cl^-_{pl} \quad (10)$$

$$\Delta ISV = 1/1.05 * \Delta Cl^-_{isv}/\Delta Cl^-_{pl} * \Delta PV \quad (11)$$

$$\Delta ECV = \Delta PV + \Delta ISV \quad (12)$$

$$\Delta ICV = \Delta TW - \Delta ECV \quad (13)$$

where ICV indicates the intracellular fluid space, ISV denotes interstitial fluid space, TW indicates total body water and subscripts pl, isv, ecv, in, u and s indicated plasma, interstitial and extracellular fluid spaces, intake, urine and sweat, respectively.

Nose et al (1985) demonstrated that the decreases in ^{51}Cr-EDTA in various tissues after thermal dehydration were strongly correlated with their loss of sodium ($r=0.97$, $p<0.001$), which itself was highly correlated to chloride losses. Changes in the ECV space determined by the chloride method, were almost identical to those calculated from the ^{51}Cr-EDTA dilution method.

3.8.4.3 EXAMPLE OF FLUID SHIFT CALCULATIONS

As an example, consider the following data taken from one subject during a trial lasting 4 hours with 30 minutes recovery after the four hours. The subject was 178cm tall and weighed 67.8kg, with an estimated plasma volume of 3007ml. The following data were taken after four hours of exercise while intermittently ingesting 3,850 litres of a 100mEq/L sodium chloride beverage followed by 30 minutes recovery.

At 30 minutes recovery, assuming all the ingested fluid and electrolytes had been absorbed, the following data were obtained:

urine Cl^- loss = 103 mEq
sweat Cl^- loss = 156 mEq
plasma volume before = 3007 ml
plasma volume after = 3239 ml
total plasma Cl^- before = 325 mEq
total plasma Cl^- after = 369 mEq
ingested Cl^- = 385 mEq
Δ total body water (ΔTW) = +100ml

then using equations 9 to 13, ΔPV = +232ml, ΔISV = +403 ml, and ΔICV = -534 ml.

3.9 STATISTICAL ANALYSIS

A Latin Square Randomisation was used to randomise the order in which the treatments were presented to the subjects. In both projects one and two, there were three treatments (A,B and C) and six subjects (1 through 6). The Latin Square randomisation for both projects was as follows:

SUBJECT	TREATMENT			SUBJECT	TREATMENT		
1	A	B	C	4	A	C	B
2	B	C	A	5	B	A	C
3	C	A	B	6	C	B	A

A Latin Square analysis of variance was used to test for treatment differences. Where needed, covariance adjustments were made to a variable. This test incorporated a test for residual effects in the change over design. No significant residual effects were seen with any of the variables. A significant difference between means was taken as $p<0.05$.

To analyse differences over time within a treatment an approximated t-ratio was obtained as (difference of means between two time points)/(combined standard error for that treatment). The appropriate significance level to use for this comparison was calculated using the Bonferroni technique for multiple comparisons. The significance level was calculated by taking the standard significance level, ie 5%, and dividing by the number of comparisons. There were 6 means, thus the number of

possible comparisons was 15. Therefore the appropriate significance level, using the Bonferroni Technique, is 5 divided by 15, which equals a significance level of $p<0.0033$.

To compare fluid-cation balance between different treatments, the Wilks' Lambda statistic was used.

CHAPTER 4: PART 1. THE EFFECT OF SODIUM INGESTION WHEN THE RATE OF FLUID INGESTION DOES NOT MATCH SWEAT RATE

4.1 EXPERIMENT DESIGN AND PROTOCOL

4.1.1 SUBJECTS

Six healthy, trained, non-heat-acclimated male cyclists were recruited by advertising on the University of Cape Town campus. Trained was defined as routinely cycling at least 100 km/week. Subjects gave their written consent before participating in the experiment. The characteristics of the 6 subjects are given in Table 4.1.

Table 4.1. Subject characteristics (means ± SEM)

Age (yr)	24 ± 1.6
Height (cm)	179.8 ± 1.2
Weight (kg)	78.0 ± 2.3
* Surface Area (m^2)	1.97 ± 0.03
† Plasma Volume (ml)	3215 ± 44
VO_{2max} (ml O_2 STP/kg/min)	58.5 ± 4.0
Peak Power Output (watts)	381 ± 17
% VO_{2max} (%)	66 ± 0.5

VO_{2max} was taken as the highest average 1-min value for oxygen consumption during an incremental bicycle ergometry protocol.
% VO_{2max} is the percentage of maximum oxygen consumption used during the trials.
* Surface area calculated from height and weight (DuBois and DuBois, 1916)
† Plasma volume calculated from surface area (Retzlaff et al, 1969).

4.1.2 PROTOCOL

Subjects performed an incremental cycle ergometer test to exhaustion to determine their VO_{2max} and peak power output (PPO).

Subsequently subjects were required to report to the laboratory on three consecutive occasions a week apart. Testing took place at the same time on each occasion. Throughout the testing period, the subjects were instructed to maintain the same training program. On the day prior to each test, the subjects rested and ingested a high carbohydrate diet (>8g/kg body weight) with generous amounts of water. Subjects were asked to refrain from drinking caffeine containing beverages (coffee, tea and carbonated cola drinks). On the morning of the trial, subjects were asked to eat 2-4 slices of toast with jam or honey and to drink 250 ml of water 2 hours before reporting to the laboratory. For 24 hours prior to testing, the subjects were asked to refrain from drinking caffeine containing beverages (coffee, tea and carbonated cola drinks).

On one of each of the three occasions, subjects exercised in a heat chamber (32°C and 55% RH), while ingesting one of the following:

a) No beverage (**NF trial**)

b) Tap water (**W trial**)

c) Capsules containing 0.585g (10 mEq) sodium chloride (NaCl) with every 100ml of tap water (**S trial**). This gives a NaCl concentration of 100 mEq/l.

The order in which they received the above treatments was randomised according to a Latin Square Randomisation.

In the trials in which fluid was ingested, the following drinking regime was adhered to: on commencing exercise, subjects consumed 400ml of the appropriate treatment drink. Every 10 minutes thereafter, they ingested 100ml of the treatment drink, with the last drink being ingested at 80 minutes of exercise. This drinking regime has been reported to optimise gastric emptying and provide approximately 1200 ml of fluid to the subject during the 90 minutes of cycling (Noakes et al, 1991).

On arriving in the laboratory an indwelling cannula (Jelco No. 20) was placed in a forearm vein of the subject, and a stopcock attached. During the trial, heparinised saline was used to keep the cannula patent. Before each sample was taken, the dead space in the cannula was cleared of the heparinised saline. All blood samples were assayed for haematocrit, haemoglobin content, and sodium, chloride and total protein concentrations.

The subject then voided his bladder and weighed himself nude. The subject redressed in cycling shorts, socks and cycling shoes and entered the environmental chamber, which had been set to 32 ± 1 °C and 55 ± 5 % relative humidity. The windspeed was 4 ± 1 km/h. A resting blood sample was taken from the subject after he had been seated for 15 minutes on the

stationary ergometer.

After a 5 minute warmup, the workload was set to elicit 65% VO_{2max}. In the W and S trials subjects commenced the drinking regime. The set workload was maintained for the next 90 minutes. Between minutes 6 and 10, on-line oxygen consumption was measured. and checked to approximate 65% of VO_{2max}.

Ten minutes after commencing cycling, a blood sample was drawn, and thereafter blood was sampled at 20, 40, 60, 80 and 90 minutes.

Sweat was collected every 20 minutes from alternating arms by the "forearm bag method". Sweat was aspirated from the sweat bag, placed in an 1,5 ml reaction tube and frozen at -20°C until it was assayed for electrolytes.

On completing the 90 minutes of exercise, the subject voided his bladder into a collection bottle and then weighed himself nude after being thoroughly dried with a towel. Urine volume was measured to the nearest millilitre using a measuring cylinder. A sample of urine was stored frozen at -20°C until it was assayed for electrolyte concentrations and osmolality.

The subject then remained seated in a chair in an upright position for one hour, at which time a final blood sample was taken. He then voided his bladder again and weighed himself nude.

4.2 RESULTS

4.2.1 HEART RATE

Mean heart rate, shown in Figure 4.1, was significantly increased at 30 minutes with respect to 10 minutes ($p<0.0033$) in the three trials. Heart rate in the No Fluid trial (NF) continued to increase, and was significantly higher at 70 and 90 minutes than the value at 30 minutes. Heart rates in the Saline (S) and Water (W) trials were not different from values at 30 minutes during the rest of the exercise bout. No significant difference was found between the W and S trials, whereas heart rate during the NF trial was significantly higher than both the W and S trials at minutes 70 and 90 ($p<0.05$ and $p<0.005$, respectively).

4.2.2 PLASMA SODIUM, CHLORIDE AND PROTEIN CONCENTRATIONS

Plasma sodium, chloride and protein concentrations are shown in Figures 4.2, 4.3 and 4.4. They are expressed as the adjusted means and standard deviations with time 0 as the co-variate.

Plasma sodium concentration (Fig. 4.2) in the NF trial was significantly elevated, compared to zero minutes, at minutes 20, 40, 60, 80 and 90 ($p<0.0033$). In the S trial, plasma sodium concentration was significantly higher than the resting value at minutes 40, 60, 80 and 90 ($p<0.0033$). The average increase in plasma sodium concentration at 90 minutes of exercise was 4.6 ± 0.7 mEq/l in the NF trial and 4.2 ± 0.7 mEq/l in the S trial. After 60 minutes recovery, plasma sodium concentration in both the NF and S trials

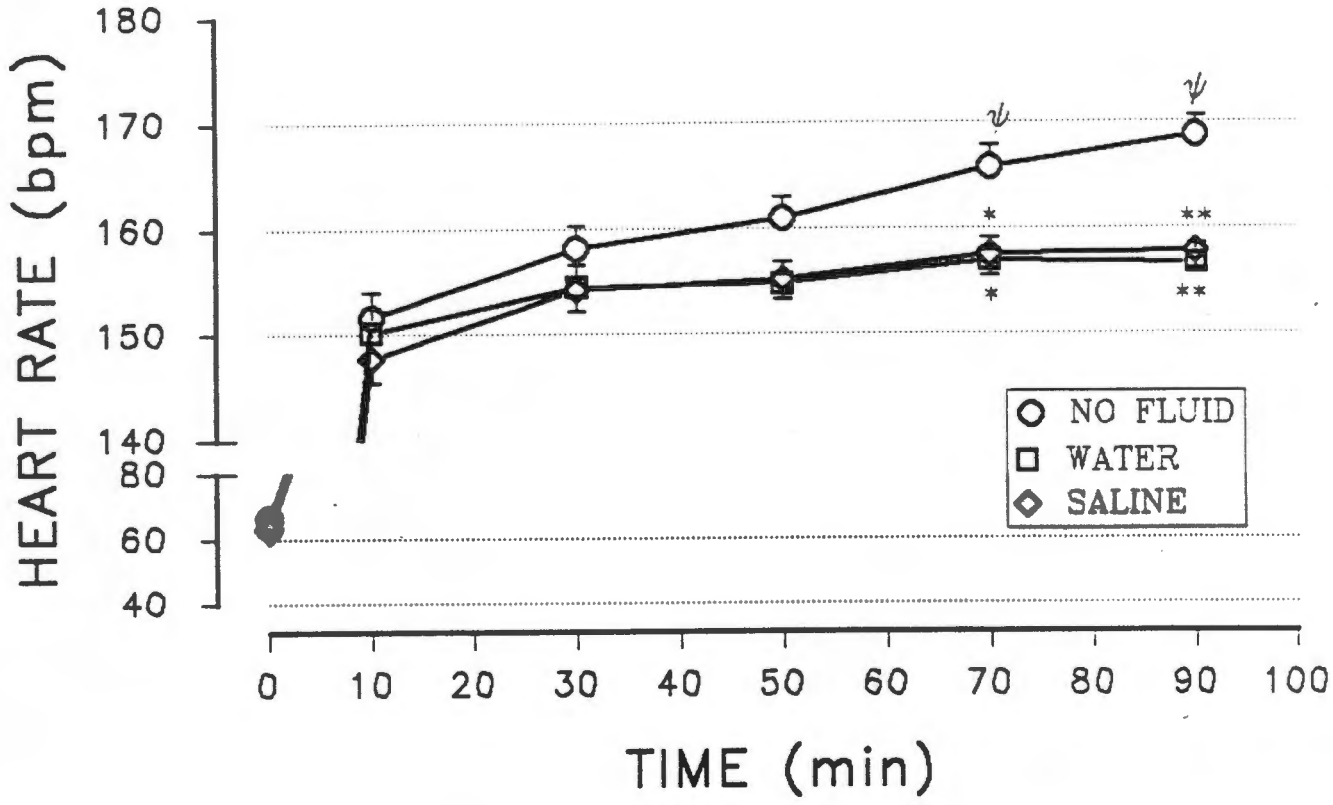

Figure 4.1 – Heart rate (beats per minute, BPM) during the No Fluid, the Water and the Saline trials (means ±SEM, n=6). *,** denotes significantly different from No Fluid (* $p<0.05$ ** $p<0.005$); ψ denotes significantly different from T_{30} ($p<0.0033$).

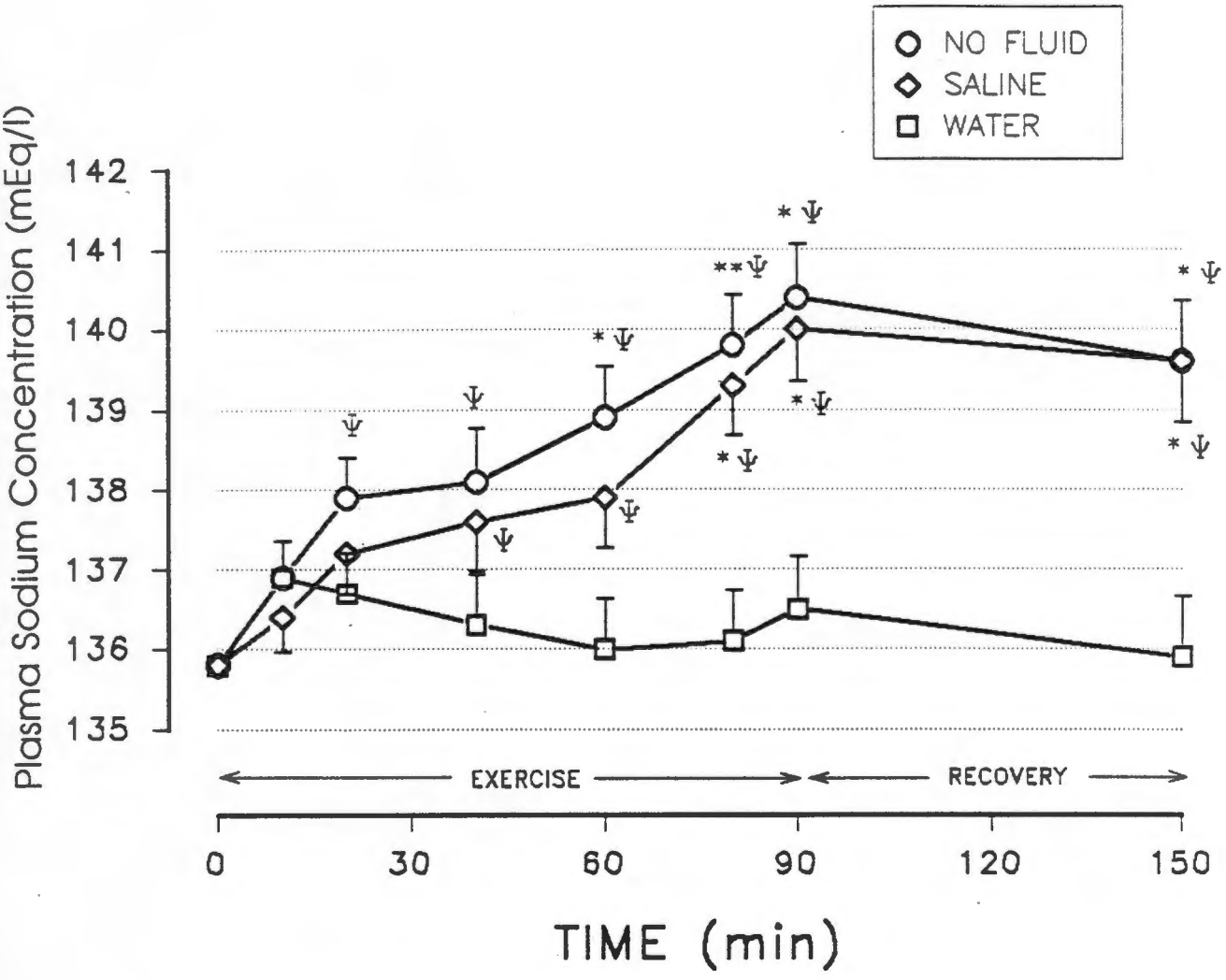

Figure 4.2 -- Plasma sodium concentration (mEq/l) during the No Fluid, the Water and the Saline trials (mean ±SEM, n=6). *,** denotes significantly different from Water (* $p<0.05$ ** $p<0.005$); ψ denotes significantly different from T_0 ($p<0.0033$).

were still significantly elevated compared with 0 minutes ($p<0.0033$). Plasma sodium concentration in the W trial was not significantly different from the resting value for the duration of the trial, including the value during recovery.

No significant difference in the plasma sodium concentration between the NF and S trial was detected during the exercise bout or at the end of recovery. Plasma sodium concentration in the NF trial was significantly higher than that in the W trial at 60 minutes ($p<0.05$) and at 80 and 90 minutes ($p<0.01$). Plasma sodium concentrations in the S trial were significantly higher than in the W trial at minutes 80 and 90 ($p<0.05$). Plasma sodium concentration in both the NF and S trials was significantly higher at 60 minutes of recovery than in the W trial.

Plasma chloride concentration (Fig. 4.3) in the NF trial increased gradually during the exercise period and was significantly higher than the resting value at 40, 60, 80 and 90 minutes ($p<0.0033$). Chloride concentration also increased over time in the S trial and was significantly higher than the resting value at minutes 10, 20, 40, 60, 80 and 90 during exercise ($p<0.0033$). Plasma chloride concentration in the W trial was not significantly higher than the control value at any time during exercise or at the end of recovery.

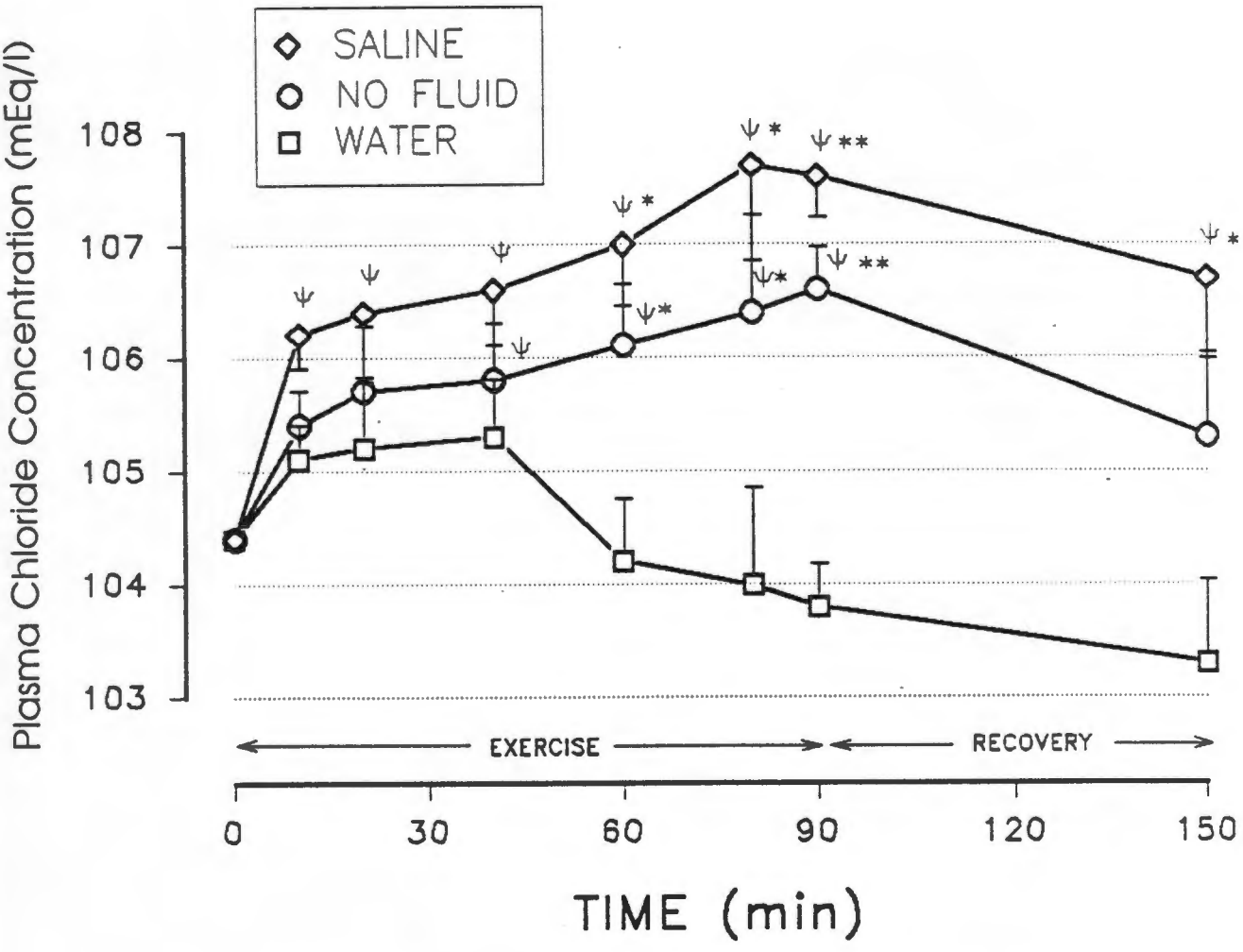

Figure 4.3 – Plasma chloride concentration (mEq/l) during the No Fluid, the Water and the Saline trials (mean ±SEM, n=6). *,** denotes significantly different from Water (* $p<0.05$ ** $p<0.005$); ψ denotes significantly different from T_0 ($p<0.0033$).

At the end of recovery, plasma chloride concentration in the S trial was still significantly elevated compared to zero minutes, whereas the recovery value in the NF trial was not significantly different from the value at time zero.

Plasma chloride concentrations in the NF and S trial were significantly higher than values in the W trial at 60, 80 ($p<0.05$) and 90 minutes ($p<0.005$). There was no significant difference between the end of recovery values for chloride concentration in the NF and S trial, and the NF and W trial, but the value in the S trial was still significantly higher than the value for the W trial ($p<0.05$) at 60 minutes recovery.

In all three trials, total plasma protein concentration (TPP) increased significantly ($p<0.0033$) from zero to 10 minutes (Figure 4.4). TPP during exercise in the NF trial was significantly higher than the 10 minute value at 60, 80 and 90 minutes ($p<0.0033$), while in the S and W trials TPP during the rest of exercise was not different from the 10 minute value. TPP was higher in the NF trial than in the W trial at 40, 80 and 90 minutes ($p<0.05$). NF total plasma protein content was also higher than that in the S trial at 40 minutes. At the end of recovery, both W and S plasma protein concentrations were significantly lower than in the NF trial ($p<0.05$). The total plasma protein concentration in the S trial at the end of 60 minutes of recovery was also lower than the value in the W trial value ($p<0.05$).

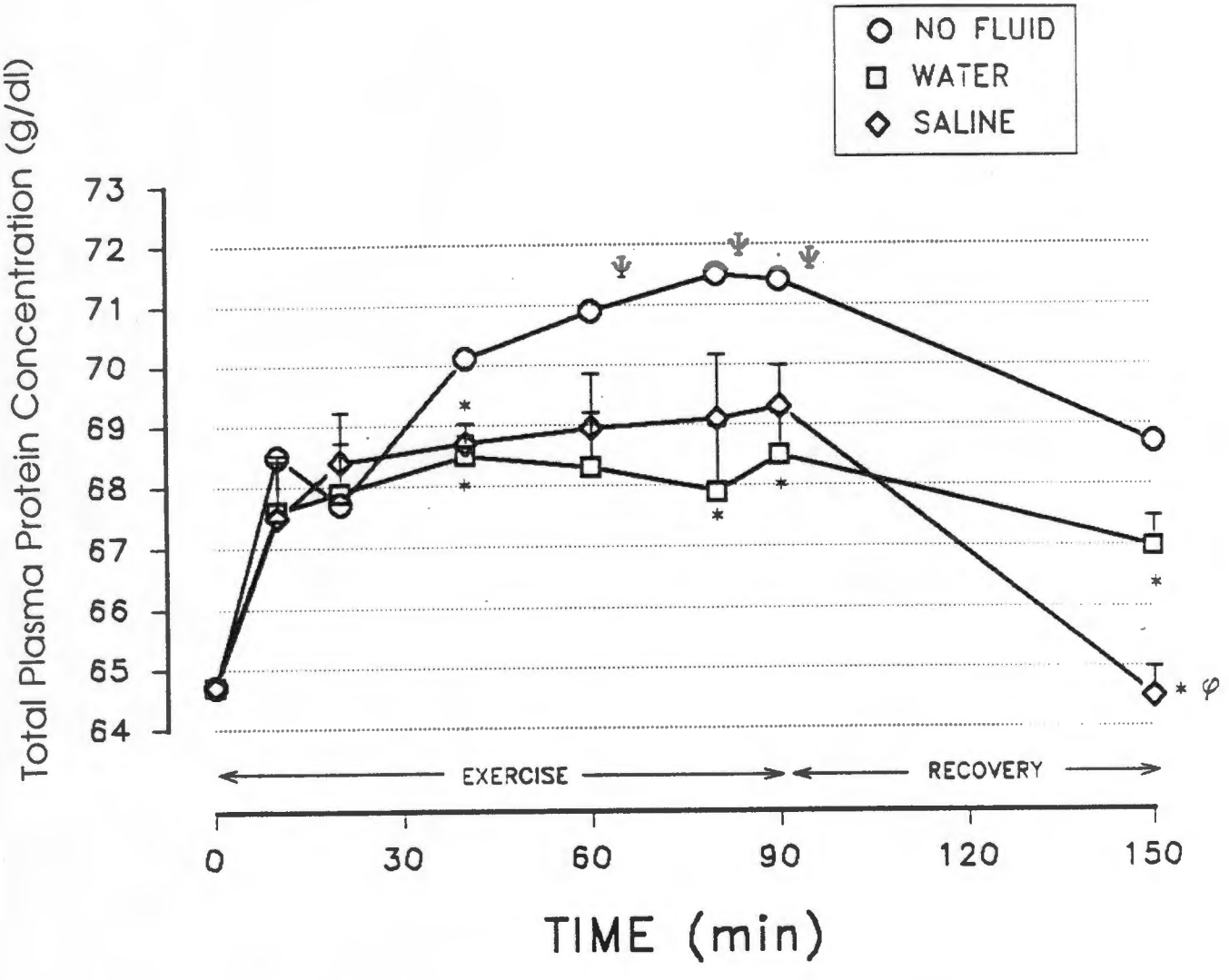

Figure 4.4 – Total plasma protein concentration (g/dl) during the No Fluid, the Water and the Saline trials (mean ±SEM, n=6). * denotes significantly different from No Fluid ($p<0.05$); ψ denotes significantly different from T_0 ($p<0.0033$); φ denotes significantly different from Water ($p<0.05$).

4.2.3 FLUID, SODIUM AND CATION BALANCE

Table 4.2 shows that the fluid balance was negative in all three trials. Due to fluid ingestion of 1.2 litres over the 90 minutes of cycling in the W and S trials, there was significantly more dehydration in subjects during the NF trial than in either the W or the S trials. At the end of 60 minutes recovery there was an average weight loss of 2.4 ± 0.06 kg and a percent dehydration of 3.09 ± 0.06%, in the NF trials. The levels of dehydration in the W and S trials were 1.58 ± 0.09% and 1.77 ± 0.10%, respectively.

Table 4.2. Fluid balance in the No Fluid, the Water and the Saline trials at the end of 90 minutes of exercise and at the end of the 60 minutes recovery period following the exercise.

	No Fluid	Water	Saline
Fluid intake (l)	0^a	1.2^b	1.2^b
Losses during exercise			
Sweat (l)	2.31 ± 0.06	2.19 ± 0.09	2.36 ± 0.08
Urine (l)	0.069 ± 0.024	0.107 ± 0.022	0.103 ± 0.022
Total fluid loss at the end of exercise (l)	2.38 ± 0.06	1.09 ± 0.08	1.26 ± 0.09
% Fluid loss replaced by the end of exercise	0%	52%	53%
Losses during recovery			
Sweat (l)	0.02 ± 0.02^a	0.13 ± 0.04^b	0.12 ± 0.07^b
Urine (l)	0.011 ± 0.005^a	0.025 ± 0.006^b	0.028 ± 0.006^b
Total fluid loss at the end of recovery (l)	2.4 ± 0.06	1.24 ± 0.08	1.40 ± 0.09
% Fluid loss replaced at the end of recovery	0%	49%	46%
% Dehydration at the end of recovery	3.09 ± 0.06^a	1.58 ± 0.09^b	1.77 ± 0.10^b

All values are expressed as mean ± SEM. Sweat loss was calculated from weight loss corrected for urine loss.
^{a,b,c}; means within a row with different superscripts were significantly different ($p < 0.05$).

Fluid losses in all three trials were very similar with the exception of the urine volume during recovery. During the NF trial, subjects passed significantly less urine during the hour of recovery than during either the W or S trials ($p<0.05$). However, there were no significant differences between the overall urine volumes in each of the three trials. Sweat volume and therefore sweat rate during exercise, was similar in all three trials. The sweat volumes measured during recovery are small compared to those recorded during exercise in all three trials. In all three groups there was continued sweating during recovery with subjects in the NF trial sweating less during recovery than during either the W or S trials. The higher sweat volumes measured during recovery in the S and W trials compared to the NF trials, may have been in part due to incomplete drying of the subjects after the exercise bout. This would have resulted in an apparent increase in sweating during recovery and a slight decrease in sweating for the preceding bout of exercise.

The percent of the fluid loss that was replaced during exercise in the W trial was 52%, and during the S trial was 53%. By the end of recovery, the percent of fluid loss replaced overall in the W and S trials, was 49% and 46% respectively. Although not significant, this apparent difference may be accounted for by urine passed during the recovery period.

Sodium balance in all three trials was negative (Table 4.3). The net sodium balance in the S group was significantly less negative than in the NF and W

trials. In the S trial, 83% of the sodium lost during exercise, and 81% of the sodium loss during exercise and recovery, was replaced.

The overall sodium deficits in the NF, W and S trials were 119.5 ± 2.8 mEq, 124.1 ± 8.7 mEq, and 28.1 ± 10.2 mEq respectively. There was no significant difference in sodium losses between the three trials, except for the urine sodium loss during recovery. Significantly more sodium was lost in the urine during recovery in the S trial than in the NF trial ($p<0.05$). There was no difference in urine sodium loss between W and NF and between W and S. The difference in urine sodium loss between NF and S may be ascribed to the greater amount of urine passed in the S trial recovery period than in the NF trial during recovery (Table 4.3).

Table 4.3. Sodium balance in the No Fluid, the Water and the Saline trials at the end of 90 minutes exercise and at the end of the 60 minute recovery period following exercise.

	No Fluid	Water	Saline
Sodium intake (mEq)	0	0	120
Losses during exercise			
Sweat (mEq)	111.8 ± 4.0	113.3 ± 9.6	135.5 ± 8.9
Urine (mEq)	6.6 ± 2.2	8.5 ± 1.4	9.6 ± 1.4
% Sodium loss replaced at the end of exercise	0%	0%	83%
Losses during recovery			
Sweat (mEq)	Sweat could not be collected due to negligible sweat rate.		
Urine (mEq)	1.1 ± 0.6^{a}	2.3 ± 0.6^{a,b}	3.5 ± 0.8^{b}
% Sodium loss replaced at the end of recovery	0%	0%	81%
Total sodium balance at the end of recovery (mEq)	-119.5 ± 2.8^{a}	-124.1 ± 8.7^{a}	-28.1 ± 10.2^{b}

All values are expressed as mean ± SEM.
^{a,b,c}; means within a row with different superscripts were significantly different ($p<0.05$).

Table 4.4. Total cation balance in the No Fluid, the Water and the Saline trials at the end of the 60 minute recovery (including the exercise period).

	No Fluid	Water	Saline
Urine & Sweat K Losses (mEq)	-42.85 ± 5.4	-50.78 ± 5.4	-52.19 ± 4.2
Na Balance (mEq)	-119.5^{a} ± 2.8	-124.1^{a} ± 8.7	-28.1^{b} ± 10.2
Cation Balance (mEq)	-162.4^{a} ± 5.4	-174.9^{a} ± 8.7	-80.29^{b} ± 10.2

Values are expressed as mean ± SEM.
Means within a row with different superscripts differ significantly ($p<0.05$).
Na balance obtained from Table 4.3.
^{a,b,c}; means within a row with different superscripts were significantly different ($p<0.05$).

Table 4.4 shows the cation balance in the three trials at the end of 60 minutes of recovery. There was no significant difference in the urinary and sweat losses of potassium between the three trials. Cation balance was negative in all three trials. The net cation balance in the S trial was significantly less negative than in the NF and W trials.

Figure 4.5 shows the relationship between fluid and cation balance in relation to a theoretical isotonic line ($y = 0.15x$). The means with SE bars are plotted at 60 minutes recovery. The intersection of the *x*- and *y*-axes (the origin) represents the pre-exercise condition. The area above the isotonic line reflects hypertonic body fluid and the area below the isotonic line represents hypotonic body fluid. At the end of 60 minutes of recovery, the fluid-cation balance in all three trials had resulted in hypertonicity in the body fluids. The fluid-cation balance (Fig. 4.5) in all three trials was significantly different from one another at the end of recovery ($p<0.0001$). The ingestion of water alone (W), resulted in a cation-fluid balance closest to the theoretical isotonic line. However, the S trial resulted in a cation-fluid balance closest to the origin.

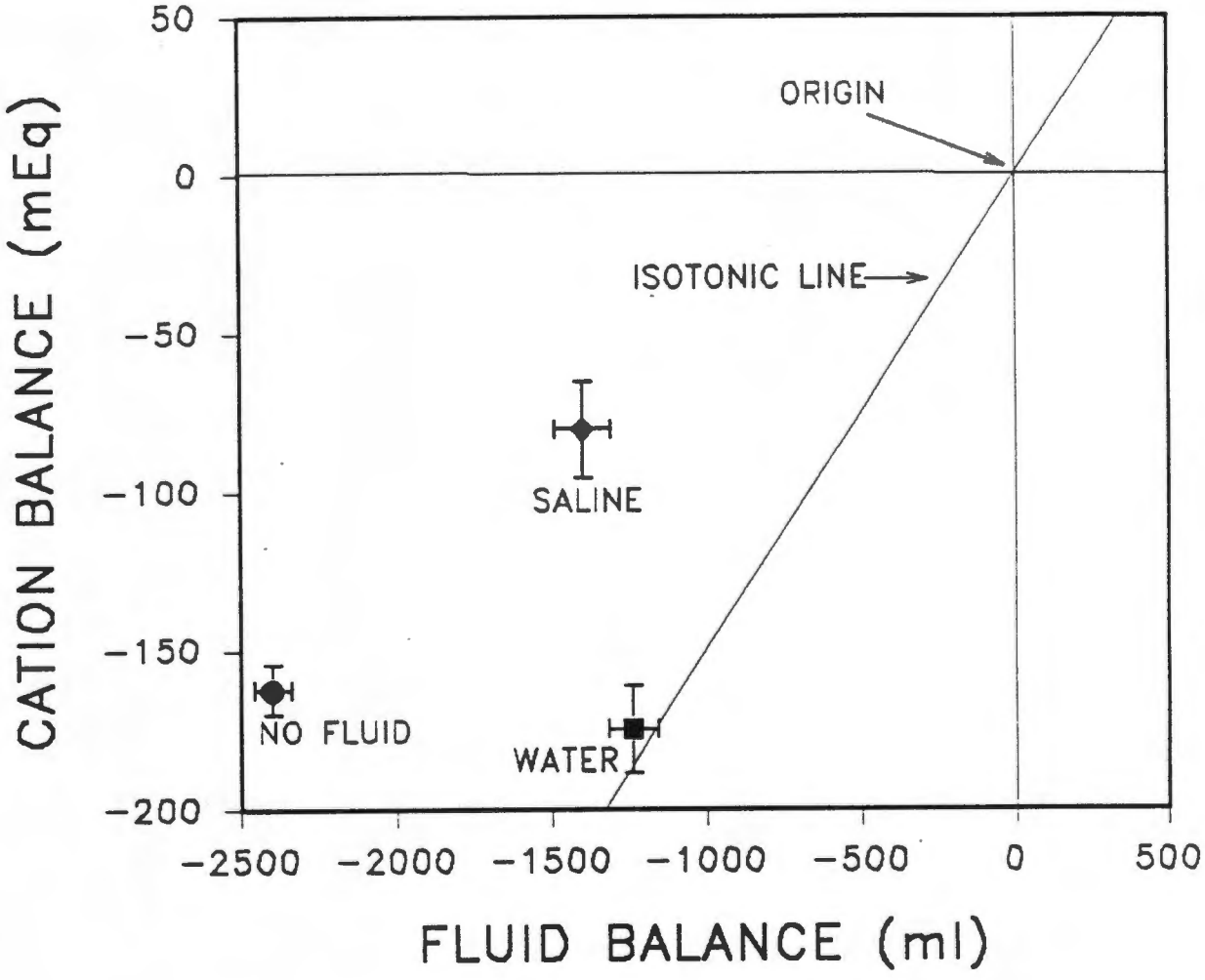

Figure 4.5 -- Fluid vs Cation Balance in the No Fluid, the Water and the Saline trials, at the end of the 60 minute recovery period following the 90 minute exercise bout (means ±SEM, n=6). The isotonic line is a theoretical line ($y = 0.15x$). All points are significantly different from one another ($p<0.0001$).

4.2.4 PERCENT CHANGE IN PLASMA VOLUME

Figure 4.6 shows the percentage change in plasma volume during exercise and recovery with respect to zero minutes.

The initial reduction in plasma volume due to the onset of exercise, was not different in the three trials. The average decrease in plasma volume for the three trials at 10 minutes was 10 ± 0.6%.

With respect to 10 minutes, plasma volume in the NF group continued to decrease during the subsequent 80 minutes of cycling and was significantly lower than minute 10 at minutes 20, 40, 60 ($p<0.05$) and 80 and 90 minutes ($p<0.0005$). In the W and S trials plasma volume did not change after minute 10 for the next 80 minutes of cycling.

The change in plasma volume in the NF trial was significantly more than the change in plasma volume of the W and S trials at minutes 40, 60, 80 and 90 ($p<0.05$). There was no significant difference in the change in plasma volume between the W and S trials.

At 60 minutes of recovery, plasma volume in both the W and NF trials was still significantly decreased ($p<0.0005$) compared to pre-exercise volume. In contrast at 60 minutes of recovery, plasma volume in the S trial had returned to pre-exercise levels and was significantly higher than the plasma volume at the end of recovery in both the NF and S trials ($p<0.05$).

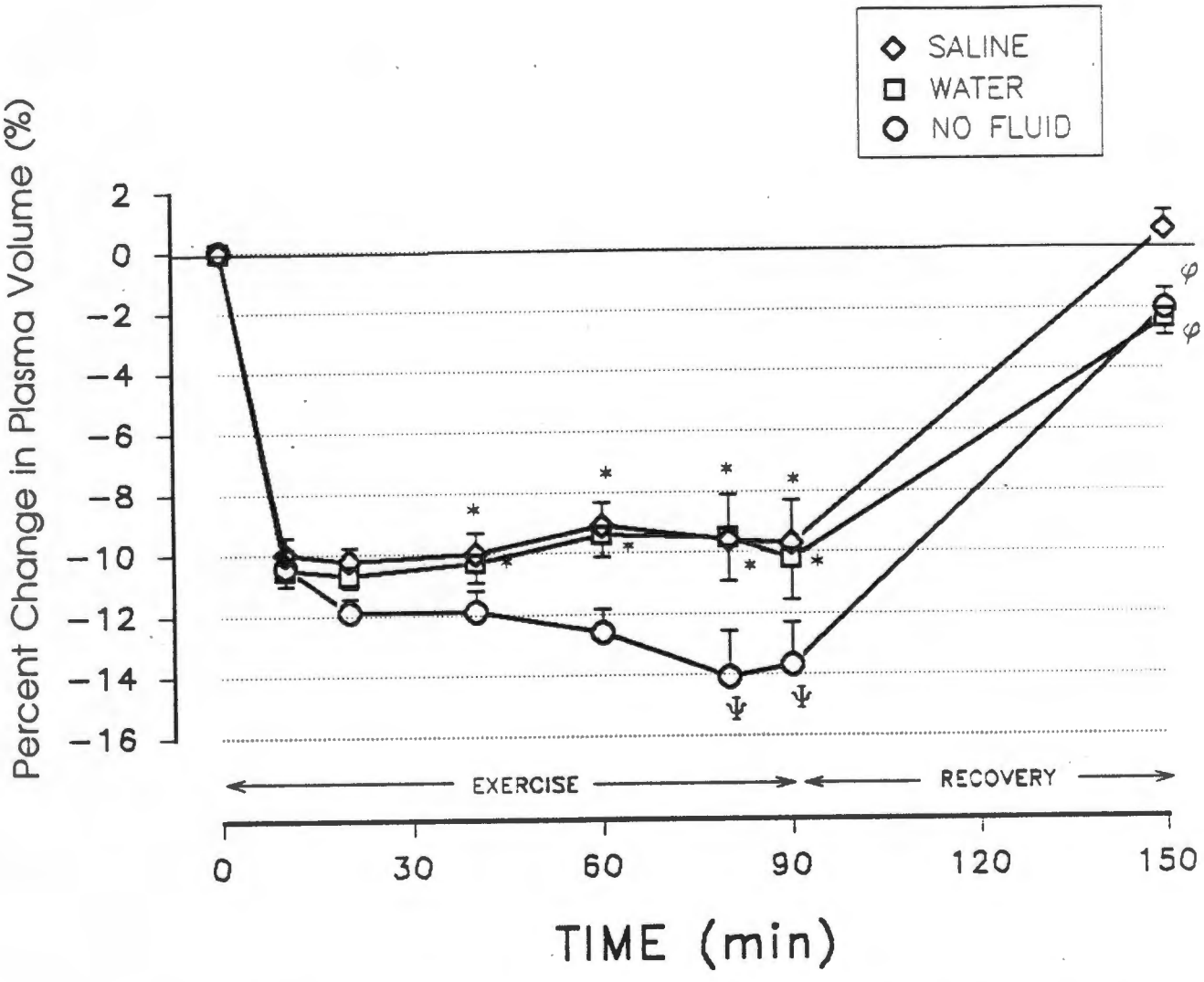

Figure 4.6 -- Percent change in plasma volume during the No Fluid, the Water and the Saline trials (mean ±SEM, n=6). * denotes significantly different from No Fluid ($p<0.05$); ψ denotes significantly different from T_{10} ($p<0.0033$); φ denotes significantly different from T_0 ($p<0.0033$).

4.2.5 VOLUME CHANGES OF FLUID COMPARTMENTS DURING EXERCISE

The drop in plasma volume that occurred during the first 10 minutes is due to the initiation of exercise and was similar in all of the three trials. In order to examine changes in volume of the intravascular compartment due to the loss of sweat and ingestion of fluid, changes in volume were calculated relative to the 10 minute value. Changes in the volume of the interstitial and intracellular compartments during exercise were also calculated with respect to their values at 10 minutes.

Figure 4.7 shows the calculated changes in plasma volume (PV), interstitial volume (ISV) and intracellular volume (ICV) in the three trials after 90 minutes of exercise. In the NF trial PV decreased by 106 ± 30 ml, ISV decreased by 1145 ± 385 ml and ICV decreased by 1016 ± 367 ml. In the W trial, PV increased by 5 ± 30 ml, ISV decreased by 1403 ± 385 ml and ICV increased by 314 ± 367 ml. In the S trial, PV increased by 6 ± 30 ml, ISV decreased by 338 ± 385 ml and ICV decreased by 919 ± 367 ml. Since total fluid loss in the NF trial was approximately double that of the W and S trials, it is not realistic to compare the fluid shifts between the NF and W trials, and the NF and S trials. However, since the fluid balance was the same in the S and W trials the differences in fluid shifts between these two conditions is comparable.

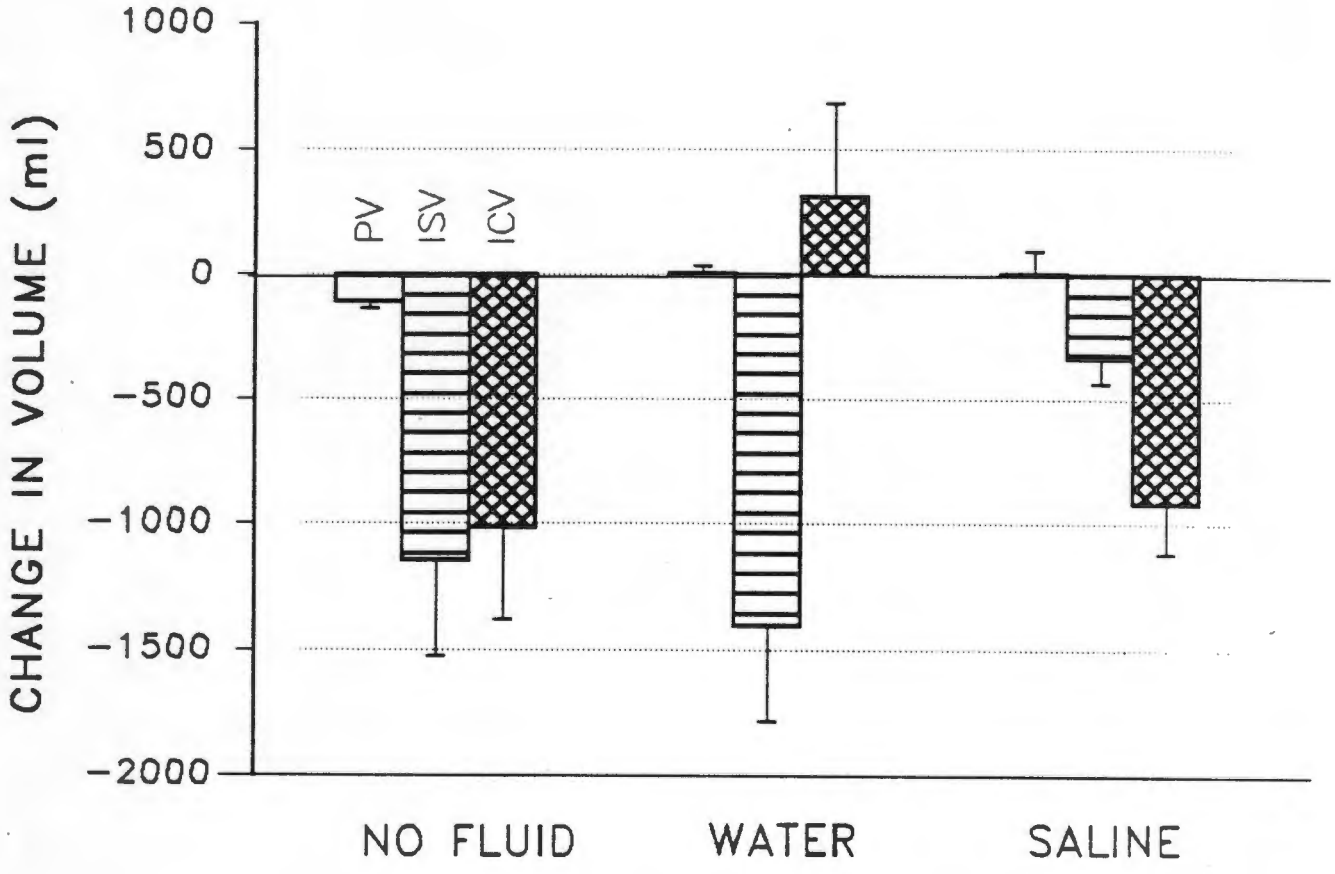

Figure 4.7 – Change in volume of fluid compartments at the end of the 90 minute exercise bout (mean ±SEM, n=6). The change in volume is calculated with respect to the volume of the the compartment at 10 minutes. PV, Plasma Volume; ISV, Interstitial Volume; ICV, Intracellular Volume,

There was a trend for more of the fluid lost during exercise (Fig. 4.7) in the W trial to come from the ISV, whereas in the S trial more of the fluid loss tended to come from the ICV. When the fluid shifts were expressed in terms of the relative % contribution that each compartment made to the fluid deficit (Fig. 4.8), the difference in the trend between W and S becomes more apparent.

In the NF trial, 44.7% of the fluid loss came from the ICV and 55.3 from the extracellular volume (PV + ISV). In the W trial, all the fluid loss arose from the ISV. There was also a shift of fluid from the interstitial to intracellular volume so that intracellular volume increased. After 90 minutes of exercise in the S trial, 73.5% of the fluid loss had been provided by the intracellular space and the rest by the interstitial volume.

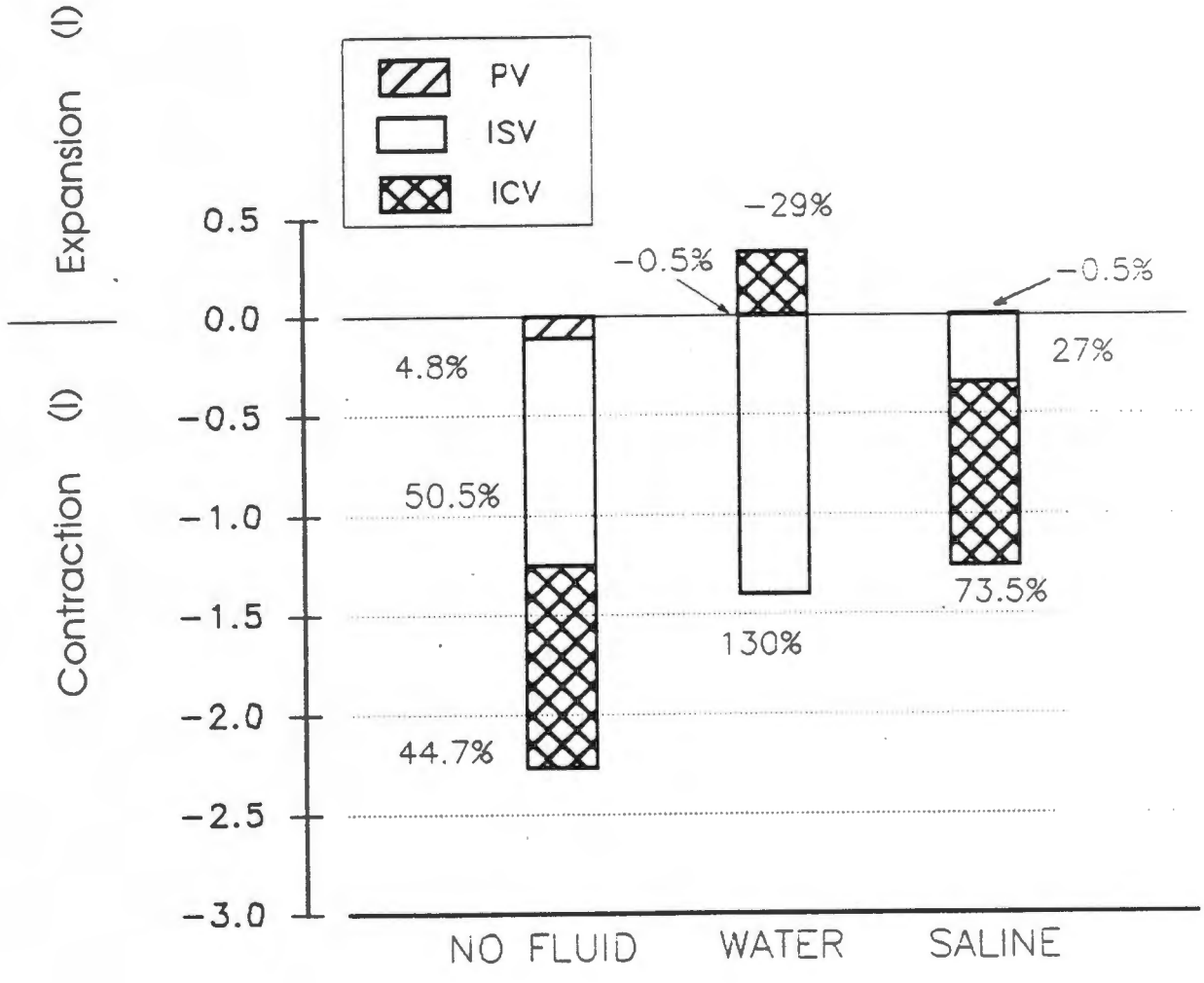

Figure 4.8 – Distribution of fluid deficit and the percent contribution of intracellular, interstitial and intravascular compartment to the fluid deficit at the end of the 90 minute exercise bout in the No Fluid, the Water and the Saline trials (mean, n=6). A "-" sign for % contribution indicates an expansion of that compartment and no contribution to the fluid deficit. The changes in volume were calculated relative to the volume of the compartments at 10 minutes. PV, Plasma Volume; ISV, Interstitial Volume; ICV, Intracellular Volume

4.2.6 VOLUME CHANGES OF FLUID COMPARTMENTS AFTER RECOVERY

Fluid shifts at the end of recovery were calculated with respect to zero minutes.

Figure 4.9 shows the calculated changes in plasma volume, interstitial volume and intracellular volume for the three trials. Since fluid balance was similar in the W and S trials it is possible to compare the fluid shifts occurring in the two conditions. Fluid shifts in the NF trials cannot be directly compared to the S and W trials since fluid deficit in the NF condition was almost double that of W and S.

In the NF trial, PV was decreased 66 ± 20 ml, ISV was decreased by 1536 ± 152 ml and ICV was decreased by 798 ± 185 ml after 60 minutes recovery. After 60 minutes recovery in the W trial, PV decreased by 77 ± 20 ml, ISV by 930 ± 152 ml and ICV by 210 ± 185 ml. After 60 minutes recovery in the S trial, PV had increased by 17 ± 20 ml, ISV decreased by 20 ± 152 ml and ICV by 1381 ± 185 ml.

In all three compartments viz. PV, ISV and ICV, the ingestion of saline (S) during exercise in the heat resulted in change in volume that was significantly different to that which occurred when water (W) was ingested. In the S trial, the decrease in the plasma volume ($p<0.05$) and interstitial

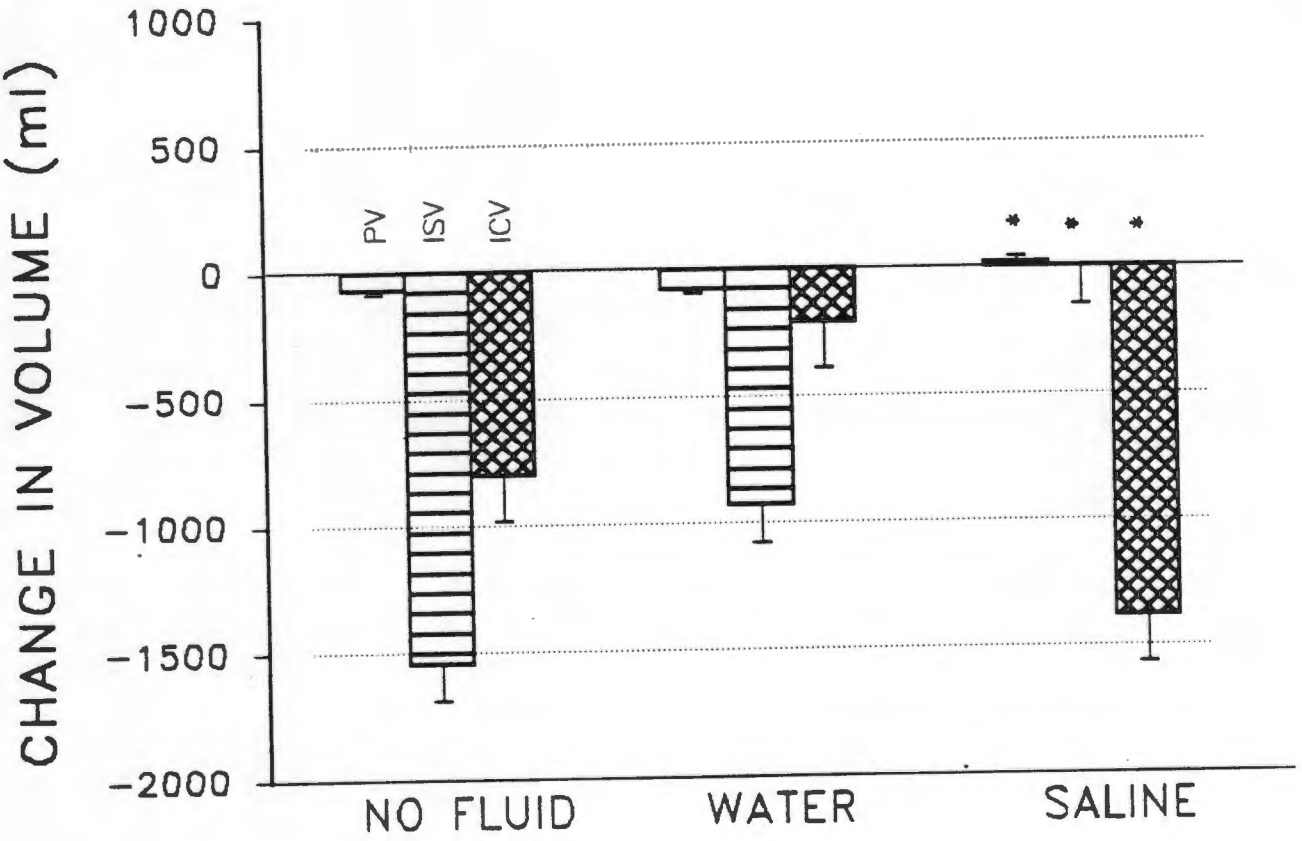

Figure 4.9 – Change in volume of fluid compartments in the No Fluid, the Water and the Saline trials, at the end of the 60 minute recovery period following the exercise bout (mean ±SEM, n=6). * denotes significantly different from Water ($p<0.05$). PV, Plasma Volume; ISV, Interstitial Volume; ICV, Intracellular Volume

volume ($p<0.01$) were significantly less than the respective decreases found in the W trial. At 60 minutes of recovery, the decrease in the intracellular volume was significantly greater ($p<0.005$) in the S group than in the W trial.

This can also be seen in Figure 4.10 in which the shifts in fluid from each compartment is expressed as a percentage contribution to the fluid deficit. After recovery in the NF condition, 67% of the fluid loss that occurred due to sweating in the preceding exercise bout, had come from the extracellular volume (PV + ISV). When ingesting only water (W), 83% of the fluid deficit came from the extracellular compartment and 17% from the intracellular compartment. In contrast, when drinking 100 mEq/l sodium chloride solution, 100% of the fluid loss came from the intracellular compartment.

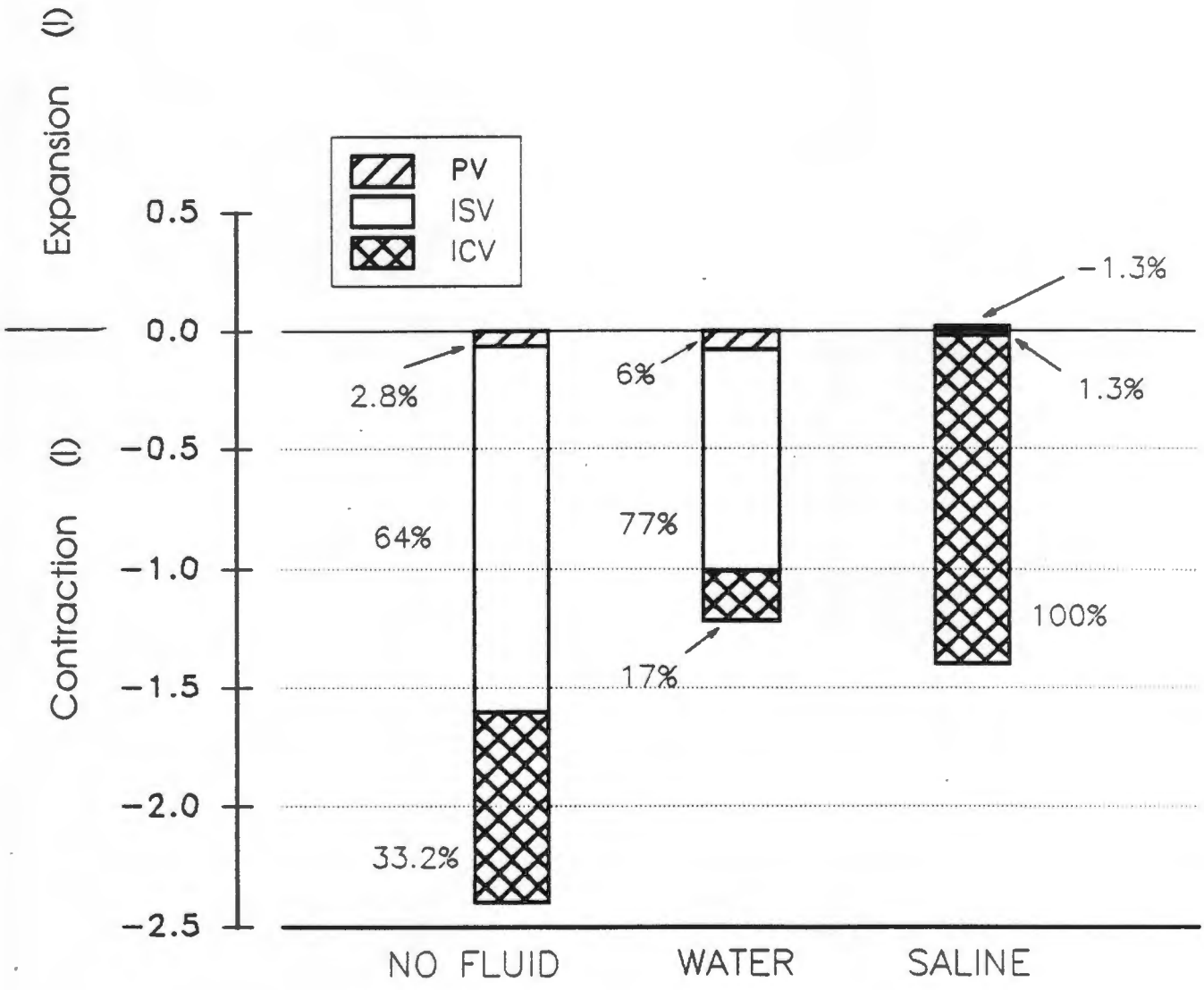

Figure 4.10 -- Distribution of fluid deficit and the percent contribution of the intravascular, interstitial and intracellular compartments to the fluid deficit at the end of the 60 minute recovery period following the exercise bout, in the No Fluid, the Water and the Saline trials (mean, n=6). A "-" sign for % contribution indicates an expansion of that compartment and no contribution to the fluid deficit. The change in volume was calculated relative to the volume of each compartment at 0 minutes. PV, Plasma Volume; ISV, Interstitial Volume; ICV, Intracellular Volume.

4.3 DISCUSSION

This project was designed to simulate exercise conditions in which fluid ingestion can only replace approximately 50% of the fluid lost via sweating. The aim was to determine whether the ingestion of a sodium chloride solution would expand plasma volume compared to the ingestion of only water. It was also the intention of this project to determine whether, under these conditions, sodium chloride ingestion has beneficial effects on fluid, sodium and cation balance.

4.3.1 PLASMA VOLUME

In the no-fluid trial (NF), plasma volume gradually decreased during the 90 minute bout of cycling in the heat. A progressive decline in plasma volume has previously been reported during exercise in the heat when no fluid is ingested (Koslowski et al, 1984; Francis, 1979; Costill et al, 1981; Candas et al, 1986; Nielsen et al, 1986; Barr et al, 1991) (see Table 2.1).

Replacement of 50% of the sweat loss with either water (W) or sodium chloride solution with a concentration of 100mEq/l (S), maintained plasma volume, with respect to 10 minutes, during the 90 minutes of exercise in the heat. Montain and Coyle (1992b, in press) similarly found that replacement of 48% of the fluid loss with a 6% glucose-electrolyte sports drink maintained plasma volume, with respect to 10 minutes, during 2 hours of cycling at 62-67% of VO_{2max} in the heat (33°C, 50% RH).

The ingestion of a sodium chloride solution (100mEq/l) in the saline trial (S) did not expand plasma volume compared to the ingestion of water alone (W). The rationale behind the inclusion of sodium chloride in the beverage under these conditions, was that an increased plasma sodium content would increase the osmotic pressure of the intravascular space thereby drawing fluid into it and expanding the plasma volume. A possible explanation why this did not occur might be that the increased intravascular hydrostatic pressure caused by exercise in the heat overshadowed the increased intravascular osmotic pressure tending to draw fluid into the intravascular space.

4.3.2 HEART RATE

Cardiovascular drift is the gradual rise is heart rate observed during prolonged upright exercise. This gradual increase in heart rate compensates for the gradual decrease in venous return and stroke volume, so that cardiac output remains constant. The decreased venous return is thought to be caused by a progressive shift of blood volume to the peripheral skin circulation for thermoregulation (Brooks and Fahey, 1985). Cardiovascular drift is exacerbated when exercise occurs in the heat, and when the individual is dehydrated and has a decreased plasma volume (Montain and Coyle, 1992b in press; Barr et al, 1990; Hamilton et al, 1991).

Thus the gradual increase in heart rate observed during the NF trial was probably due to the gradual decrease in plasma volume. The same gradual

increase in heart rate during the W and the S trials was not observed since plasma volume was well maintained.

4.3.3 FLUID AND CATION BALANCE

Figure 4.5 summarises the relationship between whole-body fluid and cation balance at the end of the 60 minute recovery period, including the 90 minutes of exercise. In this figure there are two components to the measure of disturbance of fluid-cation balance. 1) The proximity of the resultant points to the isotonic line, represents the degree of hypertonicity (if it lies above the isotonic line) or hypotonicity (if it lies below the isotonic line). 2) The distance of a point from the origin represents the overall disturbance of fluid and cation balance from the pre-exercise state.

Under conditions where fluid ingestion matches fluid loss, the ideal drink would be the one that resulted in a fluid-cation balance close to the origin along the isotonic line. However, under these conditions, the fluid balance portion (x-axis in Figure 4.5) of the fluid-cation balance in the W and the S trials is limited by the low fluid ingestion. Therefore, it is probable that under these conditions, the drink that causes the smallest divergence from the isotonic line would the better drink. Thus water is probably the drink that results in the least disturbance of homeostasis under these conditions. The deviation of the fluid-cation balance in the S trial from the isotonic line is mirrored in the elevated plasma sodium concentration that was observed.

Upon speculation, there may however, be a beneficial effect of the hypertonic deviation observed in the S trial. It has been noted that in order for fluid lost via sweating to be completely replaced, the solutes lost in the sweat first have to be replaced (Dill, 1933; Ladell, 1965). More recently Nose et al (1988b) have shown that following dehydration, replacement of the sodium chloride deficit is necessary for rapid rehydration and retention of the ingested fluid in the extracellular space. In this project, subjects were not given fluid during the recovery period. However if they had been, according to the work by Nose et al (1988b), the improved cation balance in the S trial would have facilitated faster rehydration during the recovery period.

4.3.4 PLASMA SODIUM CONCENTRATION

In the NF group in this study, the plasma sodium concentration increased gradually during the 90 minutes of exercise. Plasma sodium concentration has been shown to increase in proportion to the level of dehydration (Montain and Coyle, 1992b in press; Nielsen et al, 1986; Costill and Sparks, 1973; Barr et al, 1991; Noakes et al, 1990). This gradual increase in plasma sodium concentration is due to the loss of hypotonic sweat.

Insufficient replacement of fluid loss with water may also lead to an increase in plasma sodium concentration while exercising in the heat (Owen et al, 1986). Replacement of only 40% of the fluid loss with a glucose-electrolyte drink ((Na) = 17 mEq/l) resulted in an increase in plasma sodium

concentration of 4% (Whiting et al, 1984). In this experiment when approximately 50% of fluid loss was replaced with water, there was no increase in plasma sodium concentration. However, 50% replacement of fluid loss with a 100mEq/l saline solution caused the plasma sodium concentration increase similar to that found in the NF condition. Therefore, the volume of fluid replaced and the sodium content of the beverage are critical factors determining whether there is a significant increase in plasma sodium concentration.

The increase in plasma sodium concentration in the S trial may also have been partly due to the lack of a shift of fluid into the intravascular space, and the lack of removal of excess sodium by the kidneys, despite the increase in plasma sodium concentration.

An increased plasma sodium concentration could adversely affect thermoregulation by decreasing skin blood flow and therefore decrease heat dissipation (Harrison et al, 1978; Fortney et al, 1988; Sawka, 1992; Montain and Coyle, 1992b in press). Rectal temperature was not reported during this study although the intention was to measure it. In 2 of the 6 subjects, the data was incomplete due to the rectal probe becoming mispositioned in 3 of their trials. A further one subject refused to insert the rectal probe for the last 2 of 3 trials because he found it too uncomfortable during the first trial. Hence the relationship between plasma sodium concentration and thermoregulation could not accurately be discussed in

this project.

4.3.5 URINE AND SWEAT PRODUCTION

Sweat rate and urine output were similar whether water or a saline solution was ingested during exercise (Table 4.2).

The urine output was within the expected range of urine output of 0.8 - 1.5 ml/min (Zambraski, 1990). Dehydration levels of 4% and over have been reported to affect kidney function (Irving et al, 1990a; Irving et al, 1986; Irving et al, 1990b) and anuria was reported with a level of dehydration of 11% after a 90km foot race (Irving et al, 1990b). Therefore 3% dehydration, as seen in this study, may not have been sufficiently severe to have any effect on kidney function during the exercise bout.

During recovery, urine loss in the NF group was lower than either the S or W trials. Although statistically significant, a ± 13 ml difference between means is probably not physiologically significant.

4.3.6 CHANGES IN INTERSTITIAL AND INTRACELLULAR VOLUMES

4.3.6.1 DURING EXERCISE

In calculating the changes in volume of the fluid compartments, one must appreciate that the chloride method used to calculated fluid shifts between the intracellular and extracellular compartments depends on the assumption that membrane potential between these two spaces does not alter during exercise (Nose et al, 1988a,b; Costill et al, 1976; Costill et al, 1981); and that changes in membrane potential will obscure these calculations (Costill, 1977; Pivarnik, 1989). One should therefore be cautious in interpreting these results. They should be seen as a representation of relative change in fluid volumes, but not as the absolute volume of fluid that may have shifted.

The results show that in the NF trial, fluid loss via sweat was derived equally from both the extracellular (interstitial + intravascular) and the intracellular volumes (Fig 4.8).

However, relative to the compartment size, a greater percentage of the fluid deficit came from the extracelluar compartment than from the intracellular compartment. With a fluid loss of 2.4 l, in the proportions seen in Figure 4.8 (and assuming the volume of the ECV is 13 l and that of the ICV is 26 l), there would be approximately a 10% decrease in the extracellular volume and only a 4% decrease in the volume of the intracellular space. The reason why a greater fluid loss came from the ECV than from the ICV

could be because the fluid lost in sweat follows the sodium losses from the body. Since sodium is the major cation of the ECV (Sawka and Pandolf, 1990), most of the sweat sodium loss would have come from this space.

In the W and the S trials, there was no significant difference in the calculated changes in interstitial and intracellular volume (Fig 4.7).

4.3.6.2 DURING RECOVERY

At 60 minutes recovery, it is possible to calculate with greater certainty the distribution of fluid using the chloride method, since membrane potential would have returned to normal by this time (Pivarnik, 1989).

During recovery, the distribution of fluid between the intracellular, interstitial and intravascular volume is also dependent on the Starling forces (osmotic, oncotic and hydrostatic). However the hydrostatic force squeezing fluid out of the intravascular space will be lower at rest than during exercise.

The results in this study for the NF trial (Fig. 4.9 and 4.10) are similar to those of Costill et al (1976) and Nose et al (1988a). Costill et al (1976) found that after a dehydration of 3%, 70% of the fluid deficit came from the ECV and 30% from the ICV. Similarly Nose et al (1988a) found that after 60 minutes recovery after dehydration of 2.3% of body weight, 57% of the fluid deficit came from the ECV and 43% came from the ICV.

At the end of the recovery period in the water trial, proportionately more fluid had been lost from the extracellular space than from the intracellular space (Fig 4.10). However in the S trial, almost all the fluid lost during the preceding bout of exercise had come from the intracellular space (Fig 4.10).

Nose et al (1988a) found that during recovery from dehydration, the sweat sodium concentration determines the volume of fluid mobilised from the intracellular space. The data from this study would suggest that the extent of sodium chloride replacement during exercise, is proportional to the mobilisation of fluid from the intracellular compartment observed at the end of the 60 minutes recovery period.

The ingested sodium chloride, in the S trial, resides in the extracellular volume and causes an osmotic shift of fluid from the intracellular volume to the extracellular space during recovery. This results in the fluid deficit in the S trial coming only from the intracellular space (Fig 4.10). While in the W, the relative changes in the compartmental volumes are similar to that which is seen in the NF trial (Fig 4.10).

It is unclear why a significant difference between the distribution of body fluid in subjects drinking water alone or saline solution was not present at the end of the exercise period, but was present at the end of 60 minutes recovery. One possibility is that the chloride method does not provide

reliable results during prolonged exercise, due to a change in the resting membrane potential. Another possibility is that the osmotic force of an increased sodium content in the extracellular space is, for an unknown reason, only capable of inducing fluid shifts when the body is not exercising.

4.4 CONCLUSIONS

Since the ingestion of a sodium chloride solution containing 100mEq/l did not have a beneficial effect on plasma volume and plasma sodium concentration, when fluid ingestion rates were approximately half of the rate of sweat loss, it is concluded that there would be no advantage over water in the ingestion of a concentrated sodium chloride beverage under these conditions.

CHAPTER 5: PART 2. THE EFFECT OF SODIUM REPLACEMENT WHEN FLUID INGESTION MATCHES SWEAT RATE

5.1 PROTOCOL AND PROCEDURE

5.1.1 SUBJECTS

Six healthy, trained cyclists were recruited for this study from advertisements placed on the University of Cape Town campus. To be considered trained the volunteers for this study had to be cycling at least 100 km per week. All subjects gave their written consent before participating in the study. Table 5.1 gives the characteristics of the 6 volunteers.

TABLE 5.1 SUBJECT CHARACTERISTICS

AGE (yrs)	23 ± 1
WEIGHT (kg)	80.4 ± 3.0
HEIGHT (cm)	182.2 ± 2.2
* **SURFACE AREA (m^2)**	2.02 ± 2.2
† **PLASMA VOLUME (ml)**	3293 ± 80
VO_{2MAX} (ml/kg/min)	60.3 ± 2
PEAK POWER OUTPUT (watts)	395 ± 10
%VO_{2MAX} (%)	55.3 ± 0.95

Values are expressed as means ± SEM.
VO_{2max} was taken as the highest average 1-min value for oxygen consumption during an incremental bicycle ergometry protocol.
% VO_{2max} is the percentage of maximum oxygen consumption used during the trials.
* Surface area calculated from height and weight (DuBois and DuBois, 1916)
† Plasma volume calculated from surface area (Retzlaff et al, 1969).

5.1.2 PROTOCOL

One week prior to the commencement of the experiment, subjects underwent an incremental maximal cycle ergometer test to exhaustion to determine their VO_{2max} and peak power output and a submaximal test to determine the workload necessary to elicit 55% of VO_{2max}.

Experimental trials were separated by 7 days, with the day before each trial being a rest day. The trials for each subject were held at the same time of the day each week. The subjects were asked to keep their training consistent over the four weeks of testing.

Subjects completed three trials each in a randomised order determined by a Latin Square Randomisation. Each trial comprised four hours of intermittent cycling at 55% VO_{2max}. The subjects were required to complete 8 bouts of 25 minutes cycling with 5 minutes rest between each bout.
Subjects rested for half an hour in a upright seated position after the last 25 minute exercise bout. Trials were conducted in a temperature of 20 ± 1°C and relative humidity of 70 ± 5%. The windspeed was 0 km/hour.

The exercise model was the same in each trial, but the bevergae ingested differed on each occassion. The control drink was a carbohydrate electrolyte (LS) beverage containing 8% carbohydrate (8g/100ml) and a sodium chloride concentration of 4.6 mEq/l. In each of the other 2 trials, the subjects consumed the same drink as in the LS trial, but supplemented

sodium chloride with the simultaneous ingestion of gel capsules containing sodium chloride. For the medium salt trial (MS), the effective NaCl concentration of the drink was 50mEq/L. In the high salt trial, the effective sodium chloride concentration of the beverage was 100mEq/l. Table 5.2 shows the composition of each drink.

TABLE 5.2 Composition of the beverages ingested during the Low Salt, the Medium Salt, and the High Salt trials.

DRINK	(NaCl) (mEq/l)	Osmolality (mOsm/kg H_2O)	(CHO) (g/100 ml)	Capsule Content (1 capsule/150 ml)
LS	4.6	140	8	----------
MS	50	192	8	398 mg NaCl
HS	100	285	8	836 mg NaCl

(NaCl) = sodium chloride concentration, (CHO) = carbohydrate concentration.
LS, Low Salt Drink; MS, Medium Salt Drink; HS, High Salt Drink

After an initial bolus of 400ml at the beginning of exercise, subjects ingested 150ml, every 10 minutes until 220 minutes of exercise. This pattern of fluid replacement is reported to ensure that gastric emptying does not limit fluid delivery. After the first hour, 150ml is described as being emptied from the stomach every 10-15 minutes (Noakes et al, 1991).

On the day before each trial, the subjects "carbo-loaded" with a high carbohydrate diet (>8g/kg body mass). For 24 hours before each trial they also refrained from drinking any caffeine containing beverages. On the morning of each trial, the subjects consumed breakfast one and half hours before presenting themselves at the laboratory. The meal consisted of 2-4 slices of toast with jam or honey and 250 ml of water.

On arriving in the laboratory, an indwelling cannula with 3-way stopcock was inserted into a forearm vein of the subject. Between blood sampling, the cannula was kept patent with heparinised saline. Before sampling, the fluid in the dead space of the cannula was discarded. The subject then inserted a rectal probe 10cm beyond the rectal sphincter and a three lead ECG was connected to the subject's chest.

After sitting still in an upright position for 20 minutes, a resting blood sample was taken and resting heart rate and rectal temperature were measured. The subject then voided his bladder and weighed himself nude before donning cycling shorts, cycling shoes and socks.

The cyclist then mounted the ergometer and after a 5 minute self-paced warm-up, started the trial and the drinking regime. Blood samples were taken at 15, 55, 115, 175 and 235 minutes during exercise and at the end of 30 minutes of recovery. Whole blood was immediately assayed for haematocrit and haemoglobin content. The remaining blood was centrifuged at 2000 rpm for 15 minutes and plasma removed and frozen for later analysis. Plasma was assayed for osmolality, sodium, chloride, total plasma protein and creatinine concentrations. Heart rate and rectal temperature were measured at 15 and 25 minutes and thereafter half hourly for the remainder of the trial. During the 5 minute rest period at the end of each hour, each subject voided his bladder and weighed himself

nude. Urine was collected and nude weight was obtained at the end of 30 minutes of recovery. The volume of urine voided at each time point was measured and a sample of each was frozen and later assayed for osmolality, electrolytes, and creatinine concentrations.

5.2 RESULTS

5.2.1 HEART RATE

There was no difference at any time point between the heart rate means of the different trials (Fig. 5.1). Heart rates at 180 minutes and 240 minutes in the LS and MS trials were significantly higher than the 15 minute values ($p<0.0033$). Heart rate at 240 minutes in the HS trial was significantly higher than at minute 15 ($p<0.0033$).

5.2.2 RECTAL TEMPERATURE

Figure 5.2 shows the average rectal temperatures during the three trials. There was no significant difference in the rectal temperatures at any time during the different trials. Rectal temperature increased significantly compared to resting rectal temperature in all three trials. However, rectal temperature did not increase significantly during the last three hours of exercise in any of the treatments. Rectal temperature after 30 minutes recovery in all three trials was not different from the resting value.

5.2.3 FLUID BALANCE

Subjects ingested 3850 ml of fluid during the 4 hours of exercise. Since it was not possible for rate of intestinal absorption to be measured directly, we have assumed that fluid and sodium absorption lagged 15 minutes behind fluid ingestion. Taking into account this lag in absorption, after 240 minutes of exercise approximately 89% of the sweat loss was replaced in the LS trial, 95% in the MS and 100% in the HS trial.

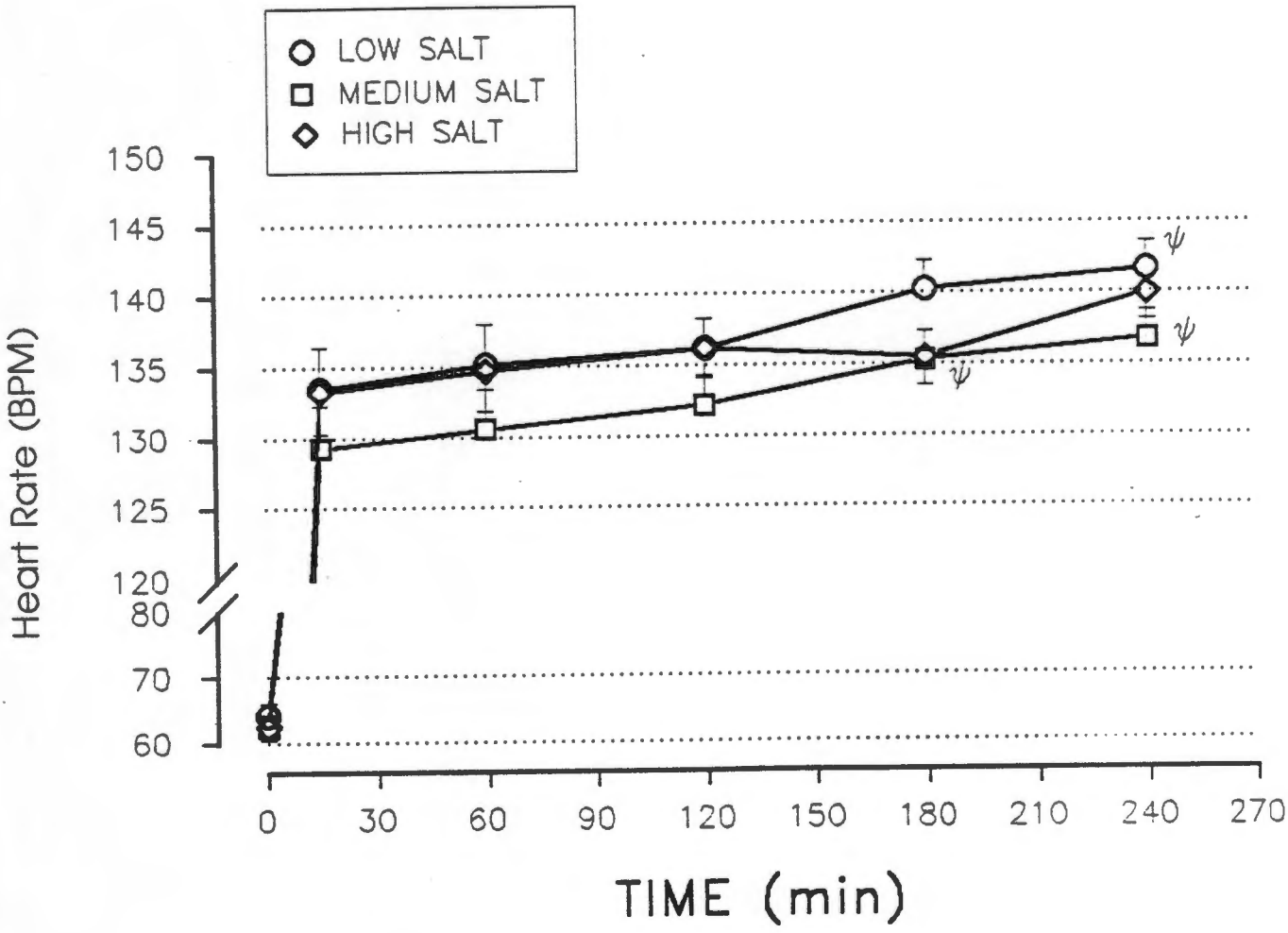

Figure 5.1 -- Heart rate (beats per minute, BPM)during exercise in the LS, the MS, and the HS trials (mean ±SEM, n=6). ψ denotes significantly different from T_{15} ($p<0.0033$).

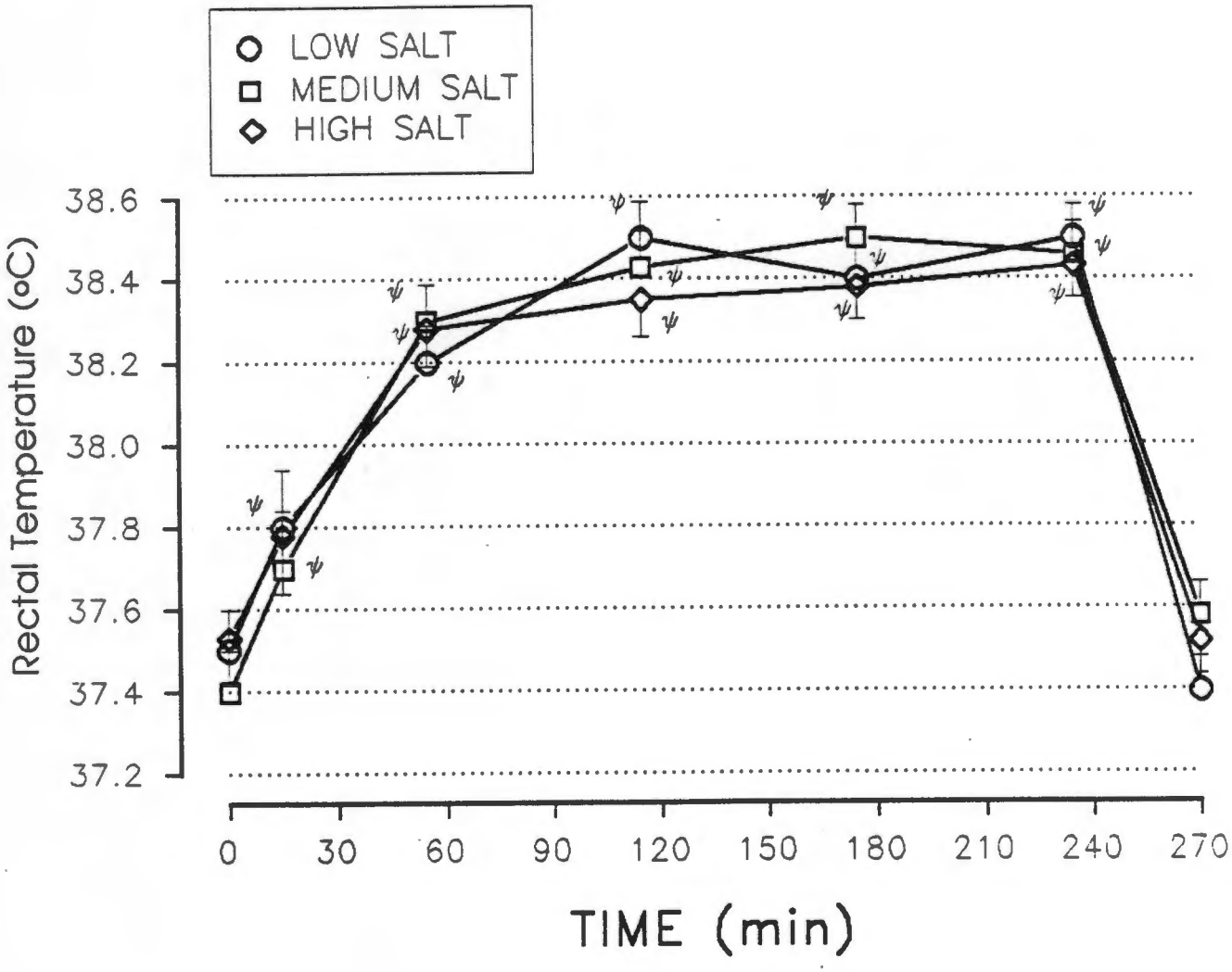

Figure 5.2 – Rectal temperature (°C) during exercise in the LS, the MS and the HS trials (mean ±SEM, n=6). ψ denotes significantly different from T_0 ($p<0.0033$).

By adding the urine volumes lost during the trial, then at the end of exercise, an average of 72% of the total fluid loss had been replaced in the LS trial, 73% in the MS trial and a significantly greater 87% in the HS trial ($p<0.05$). The resulting percent dehydration at the end of exercise in the LS, MS, and HS trials was $1.23 \pm 0.27\%$, $1.18 \pm 0.27\%$ and $0.19 \pm 0.27\%$, respectively. The percent dehydration in the HS trial after recovery was significantly less than in either the LS or MS trials ($p<0.05$).

At the end of 30 minutes of recovery, 96% of the sweat lost in the LS trial had been replaced by fluid ingestion, 101% in the MS trials and 109% in the HS trial. Accounting for the cumulative urinary fluid loss by the end of recovery, the percent replacement of total fluid loss in the LS, MS and HS trials were on average 76%, 77% and 93% respectively. The resulting percent dehydration in the LS, MS and HS trials were $1.49 \pm 0.24\%$, $1.42 \pm 0.24\%$ and $0.29 \pm 0.24\%$, respectively. Percent dehydration in the HS trials after recovery was significantly less than in the other two trials ($p<0.05$).

TABLE 5.3. FLUID BALANCE IN THE LOW SALT, THE MEDIUM SALT, AND THE HIGH SALT TRIALS DURING EXERCISE AND AT THE END OF THE 30 MINUTE RECOVERY PERIOD

TRIAL	60 MINUTES			
	Sweat Loss (ml)	Urine Loss (ml)	Estimated Fluid Absorption (ml)	Fluid Balance (ml)
LS	988 ± 53	179[a,b] ± 42	800	-367[a] ± 41
MS	868 ± 53	298[a] ± 42	800	-367[a] ± 41
HS	832 ± 53	149[b] ± 42	800	-170[b] ± 41

TRIAL	120 MINUTES			
	Sweat Loss (ml)	Urine Loss (ml)	Estimated Fluid Absorption (ml)	Fluid Balance (ml)
LS	1937 ± 150	494[a,b] ± 98	1700	-730 ± 162
MS	1847 ± 150	720[a] ± 98	1700	-868 ± 162
HS	1690 ± 150	347[b] ± 98	1700	-320 ± 162

TRIAL	180 MINUTES			
	Sweat Loss (ml)	Urine Loss (ml)	Estimated Fluid Absorption (ml)	Fluid Balance (ml)
LS	2945 ± 164	705[a] ± 121	2600	-1050[a] ± 163
MS	2816 ± 164	926[a] ± 121	2600	-1142[a] ± 163
HS	2547 ± 164	434[b] ± 121	2600	-387[b] ± 163

TRIAL	240 MINUTES			
	Sweat Loss (ml)	Urine Loss (ml)	Estimated Fluid Absorption (ml)	Fluid Balance (ml)
LS	3931 ± 232	919[a] ± 148	3500	-1350[a] ± 234
MS	3690 ± 232	1134[a] ± 148	3500	-1325[a] ± 234
HS	3498 ± 232	537[b] ± 148	3500	-505[b] ± 234

TRIAL	270 MINUTES (AFTER RECOVERY)			
	Sweat Loss (ml)	Urine Loss (ml)	Estimated Fluid Absorption (ml)	Fluid Balance (ml)
LS	3996 ± 232	1055[a] ± 142	3850	-1201[a] ± 234
MS	3820 ± 232	1196[a] ± 142	3850	-1166[a] ± 234
HS	3531 ± 232	608[b] ± 142	3850	-289[b] ± 234

All values are expressed as mean ± SEM. Sweat loss was calculated from weight loss corrected for urine loss.
LS, Low Salt Trial; MS, Medium Salt Trial; HS, High Salt Trial
[a,b,c] means in a column with different superscript differ significantly, $p<0.05$

Cumulative urinary fluid loss (Table 5.3) in the MS trial was significantly more than that in the HS trial at all time points ($p<0.05$). Cumulative urinary fluid loss at 180, 240 and 270 min was significantly higher in the LS trial than in the HS trial ($p<0.01$). Figure 5.3 shows the renal free water clearance at each hour during and after exercise. The renal free water clearance in the MS trial was significantly higher than during the HS trial at 120, 180 and 240 minutes ($p<0.05$). Free water clearance in the LS trial had a tendency to be different from the HS trial at 180 minutes ($p=0.0585$), but was significantly higher than the HS trial at 240 minutes and after recovery ($p<0.05$). Free water clearance in the LS trial was significantly higher than the MS trial at 240 minutes and after 30 minutes recovery ($p<0.05$). Free water clearance during the HS trial was always negative.

There was no significant difference at any time point between the cumulative sweat losses (Table 5.3) in the three trials. Average sweat rate over the 4 hours was 0.98 ± 0.06 l/h in the LS trial, 0.91 l/h ± 0.06 in the MS trial, and 0.89 ± 0.06 l/h in the HS trial. There was no significant difference in the average sweat rate for the 4 hours between the three trials. Also there was no correlation between individual sweat rate over the 4 hours when regressed against sodium concentration of the beverage, using linear regression ($r=0.046$).

Fluid deficit (Table 5.3) in the HS trial was significantly less than LS and MS at all time points except 120 minutes during the trial ($p<0.05$).

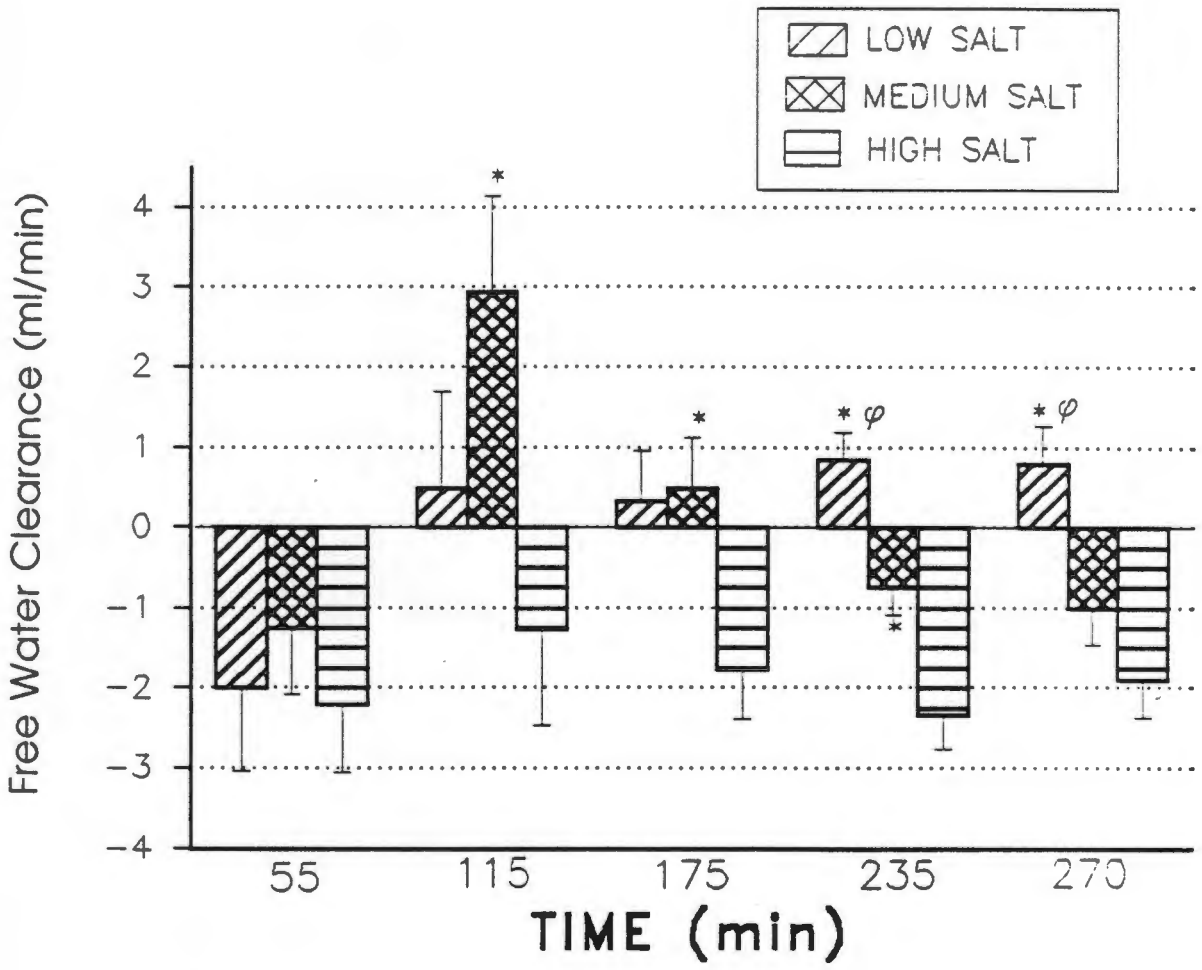

Figure 5.3 – Kidney free water clearance (ml/min) during exercise and at the end of the 30 minute recovery period in the LS, the MS and the HS trials (mean ±SEM, n=6). * denotes significantly different from High Salt ($p<0.05$); φ denotes significantly different from Medium Salt ($p<0.05$).

5.2.4 SODIUM BALANCE

Table 5.4 shows the cumulative sodium losses, gains and net balance during the trials. Sodium absorption was assumed to lag 15 minutes behind ingestion.

Due to the design of the study sodium ingestion and therefore cumulative sodium absorption, was significantly different between all trials at all time points. After exercise, and again taking into account a 15 minute lag in sodium absorption, 8.5% of the sweat sodium loss was replaced in the LS trial, 109% in the MS trial and 244% in the HS trial. If urine sodium loss is included, then 7.5% of the total sodium loss was replaced in the LS trial, 82% in the MS trial and 184% in the HS trial. By the end of recovery, 8% of the total sodium loss was replaced in the LS trial, 88% in the MS trial and 191% in the HS trial.

There was no significant difference in cumulative sweat sodium loss between trials at any time point.

Total urinary sodium loss (mEq) was significantly higher in the MS trial than in the LS trial at 120, 180, and 240 minutes ($p<0.05$) and at 30 minutes recovery ($p<0.005$). Total urinary sodium loss was significantly higher during the HS trial than in the LS trial at 180 and 240 minutes ($p<0.05$) and after 30 minutes recovery ($p<0.005$). There was no significant difference in total urinary sodium loss between the HS and MS trials.

Table 5.4 shows that sodium balance at 60, 120, 180, 240 and 270 minutes was significantly different between all three trials ($p<0.0005$); with sodium balance in the HS trial > sodium balance in the MS trials > sodium balance in the LS trial at all time points. The net sodium balance in the LS and MS trials was negative throughout exercise and at the end of 30 minutes of recovery. Sodium balance in the HS trial was positive throughout the trial.

TABLE 5.4 SODIUM BALANCE IN THE LOW SALT, THE MEDIUM SALT, AND THE HIGH SALT TRIALS DURING 240 MINUTES OF EXERCISE AND AT THE END OF THE 30 MINUTE RECOVERY PERIOD.

TRIAL	60 MINUTES				120 MINUTES			
	URINE LOSS (mEq)	SWEAT LOSS (mEq)	ESTIMATED SODIUM ABSORPTION (mEq)	BALANCE (mEq)	URINE LOSS (mEq)	SWEAT LOSS (mEq)	ESTIMATED SODIUM ABSORPTION (mEq)	BALANCE (mEq)
LS	8.3 ± 3.0	40.4 ± 3.8	3.7	-45.0^{e} ± 2.3	17.2^{e} ± 3.2	87.6 ± 8.9	7.8	-97.0^{e} ± 6.7
MS	17.6 ± 3.0	32.7 ± 3.8	40	-10.2^{f} ± 2.3	28.6^{b} ± 3.2	71.2 ± 8.9	85	-14.8^{f} ± 6.7
HS	10.6 ± 3.0	32.2 ± 3.8	80	37.2^{g} ± 2.3	23.3^{a,b} ± 3.2	66.8 ± 8.9	170	79.9^{g} ± 6.7

TRIAL	180 MINUTES				240 MINUTES			
	URINE LOSS (mEq)	SWEAT LOSS (mEq)	ESTIMATED SODIUM ABSORPTION (mEq)	BALANCE (mEq)	URINE LOSS (mEq)	SWEAT LOSS (mEq)	ESTIMATED SODIUM ABSORPTION (mEq)	BALANCE (mEq)
LS	22.3^{a} ± 3.7	136.6 ± 13.4	12	-146.9^{e} ± 11.1	26.1^{a} ± 5.0	188.3 ± 18.4	16.1	-198.2^{e} ± 15.9
MS	38.7^{b} ± 3.7	116.8 ± 13.4	130	-25.5^{f} ± 11.1	50.1^{b} ± 5.0	161.3 ± 18.4	175	-36.4^{f} ± 15.9
HS	36.1^{b} ± 3.7	104.9 ± 13.4	260	119.0^{g} ± 11.1	47.0^{b} ± 5.0	143.6 ± 18.4	350	159.4^{g} ± 15.9

TRIAL	270 MINUTES (AFTER RECOVERY)			
	URINE LOSS (mEq)	SWEAT LOSS (mEq)	ESTIMATED SODIUM ABSORPTION (mEq)	BALANCE (mEq)
LS	27.7^{h} ± 4.5	188.3 ± 18.4	17.7	-198.2^{e} ± 17.2
MS	56.3^{i} ± 4.5	161.3 ± 18.4	192	-25.6^{f} ± 17.2
HS	58.5^{i} ± 4.5	143.6 ± 18.4	385	183.0^{g} ± 17.2

All values are expressed as mean ± SEM. Sweat loss was calculated from weight loss corrected for urine loss.
LS, Low Salt Trial
MS, Medium Salt Trial
HS, High Salt Trial
^{a,b,c} means in a column with different superscripts differ significantly, $p<0.05$
^{h,i,j} means in a column with different superscripts differ significantly, $p<0.005$
^{e,f,g} means in a column with different superscripts differ significantly, $p<0.0005$

Figure 5.4 shows the renal sodium clearance in the three trials at 60, 120, 180, 240 minutes and after 30 minutes recovery. Sodium clearance in the HS trial was greater than in the MS trial at 120 minutes ($p<0.05$), 240 minutes ($p<0.005$) and at 30 minutes recovery. Sodium clearance in the HS trial was also significantly larger than during the LS trial at 180 minutes ($p<0.05$), 240 minutes ($p<0.005$) and at 30 minutes of recovery ($p<0.005$). There was no significant difference at any time in the sodium clearance between the MS and LS trials.

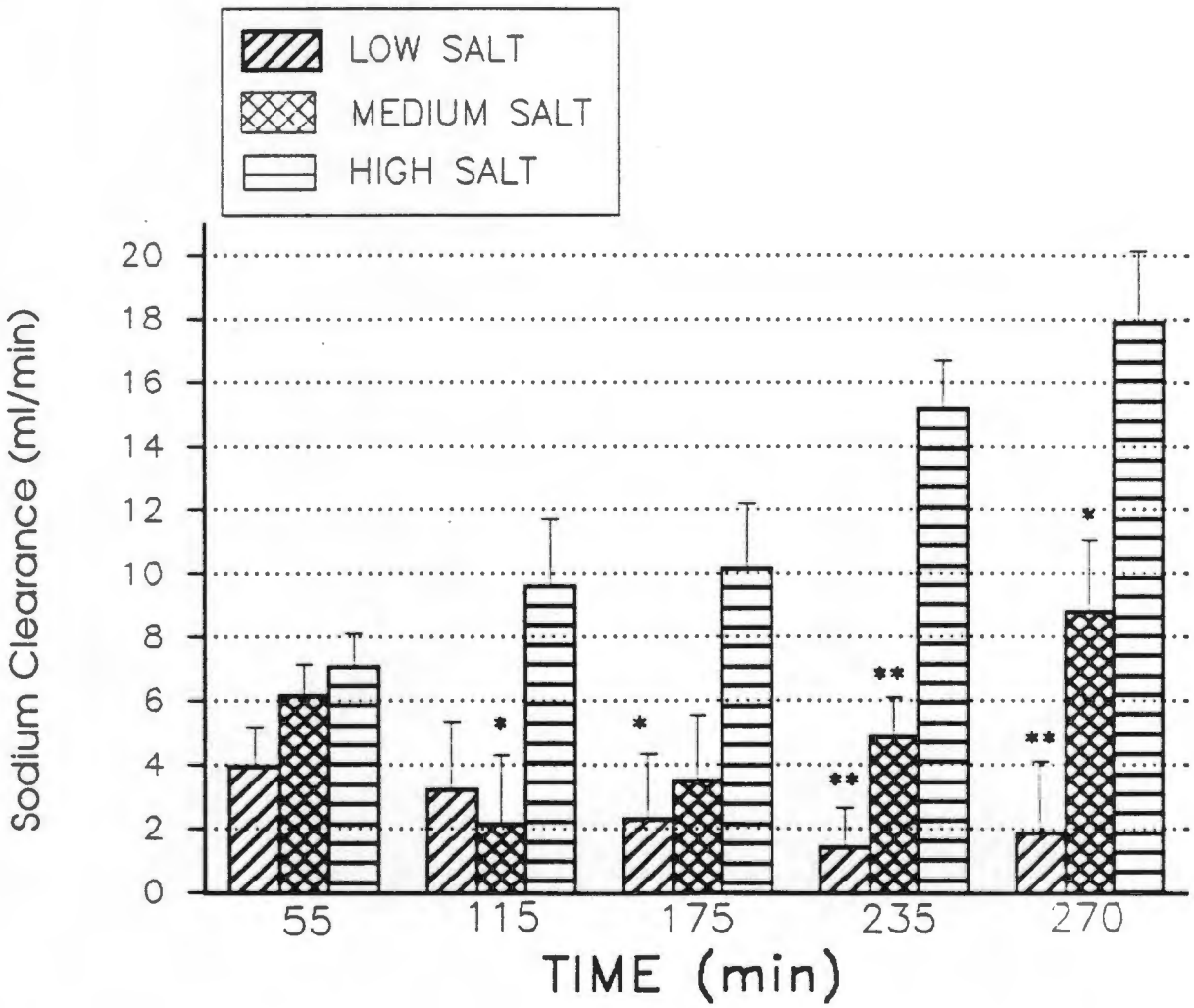

Figure 5.4 -- Kidney sodium clearance (ml/min) during exercise and at the end of the 30 minute recovery period in the LS, the MS and the HS trials (mean ±SEM, n=6). *,** denotes significantly different from High Salt (* $p<0.05$, ** $p<0.005$)

Table 5.5 shows the hourly cation balance for the 4 hours of exercise, as well as the cation balance at 30 minutes recovery. The cation balance comprises the sodium balance (derived from Table 5.4) and sweat and urinary potassium losses. The cation balance in all three trials was significantly different from one another at all time points ($p<0.0005$).

TABLE 5.5 CATION BALANCE IN THE LOW SALT, THE MEDIUM SALT AND THE HIGH SALT TRIALS DURING EXERCISE AND AT THE END OF THE 30 MINUTE RECOVERY PERIOD.

	60 MIN			120 MIN			180 MIN		
	LS	MS	HS	LS	MS	HS	LS	MS	HS
Sodium Balance (mEq)	-45.0^a ± 2.6	-10.2^b ± 2.6	37.2^c ± 2.6	-97.0^a ± 6.7	-14.8^b ± 6.7	79.9^c ± 6.7	-146.9^a ± 11.1	-25.5^b ± 11.1	119.0^c ± 11.1
Potassium Balance (mEq)	-20.7 ± 2.5	-21.2 ± 2.5	-19.9 ± 2.5	-42.9 ± 2.5	-40.7 ± 2.5	-38.8 ± 2.5	-60.7 ± 2.6	-58.0 ± 2.6	-57.9 ± 2.6
Cation Balance (mEq)	-65.7^a ± 2.6	-31.4^b ± 2.6	17.3^c ± 2.6	-139.9^a ± 5.7	-55.5^b ± 5.7	41.1^c ± 5.7	-207.6^a ± 11.1	-83.5^b ± 11.1	61.1^c ± 11.1

	240 MIN			270 MIN (RECOVERY)		
	LS	MS	HS	LS	MS	HS
Sodium Balance (mEq)	-198.2^a ± 15.9	-36.4^b ± 15.9	159.4^c ± 15.9	-198.2^a ± 17.2	-25.6^b ± 17.2	183.0^c ± 17.2
Potassium Balance (mEq)	-77.6 ± 4.2	-76.6 ± 4.2	-77.1 ± 4.2	-81.3 ± 2.7	-81.0 ± 2.7	-85.4 ± 2.7
Cation Balance (mEq)	-275.8^a ± 15.9	-112.6^b ± 15.9	-82.3^c ± 15.9	-279.5^a ± 17.2	-106.6^b ± 17.2	97.6^c ± 17.2

All values are expressed as mean ± SE.
Means in the same row with a different superscript are significantly different from one another ($p<0.0005$).
Sodium balance was obtained from Table 5.4.
LS = Low Salt trial, MS = Medium Salt trial, HS = High Salt trial.

Figure 5.5 shows the relationship between fluid and cation balance in the three trials at 60, 120, 180, 240 minutes and after recovery, as well as a theoretical isotonic line ($y = 0.15x$). The means are plotted with SE bars at 60 minute intervals from the pre-exercise condition (0 min) to the end of 4 hours of exercise (60, 120, 180, 240 mins) and at 30 minutes of recovery. The intersection of the *x*- and *y*-axes represent the pre-exercise condition (control). The area above the isotonic line reflects hypertonic body fluid and the area below the isotonic line represents hypotonic body fluids. At all time points the fluid-cation balance between all groups was significantly different from one another ($p<0.0001$).

At all time points, the fluid-cation balance in the LS trial was hypotonic and became progressively more hypotonic during the trial. The fluid-cation balance in the MS and HS trials was hypertonic at all time points. At 30 135 origin than either the LS or MS trials.

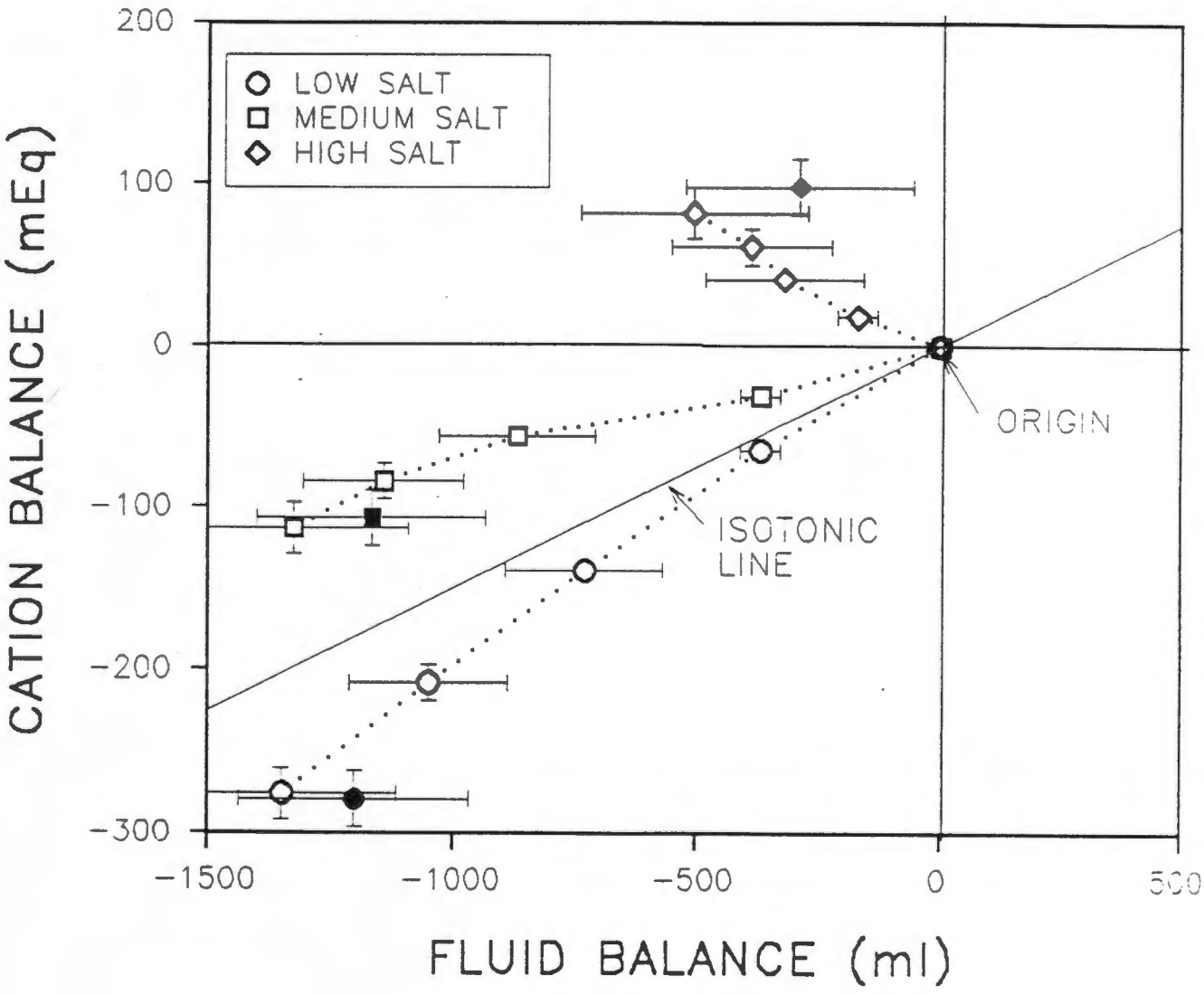

Figure 5.5 – Fluid vs Cation balance during exercise and at the end of the 30 minute recovery period in the LS, the MS and the HS trials (mean ±SEM, n=6). Means are plotted with SEM bars at 60 minute intervals over the period of 4 hours (open symbols), as well as at the end of the 30 minute recovery (closed symbols). The isotonic line is a theoretical line ($y = 0.15x$). At all time points, all three trials are significantly different from one another ($p<0.0001$).

5.2.5 PLASMA ELECTROLYTE AND PROTEIN CONCENTRATIONS

Figure 5.6 shows the change in plasma sodium concentrations compared to the pre-exercise values. There were no significant differences between trials at any time point.

The changes in plasma osmolality during the trials are shown in Figure 5.7. The change in plasma osmolality in the HS trial was significantly different from that during the LS trial at 120, 180 and 240 minutes ($p<0.05$) and from the MS trial at 15 and 120 minutes ($p<0.05$). The change in osmolality at 15 minutes during the LS trial was significantly different to the change in plasma osmolality in the MS trial at 15 minutes ($p<0.05$). The change in osmolality during the MS trial was significantly greater than during the LS trial at 180 and 240 minutes ($p<0.05$). But in all treatments, the plasma osmolality was not significantly different from the resting pre-exercise value.

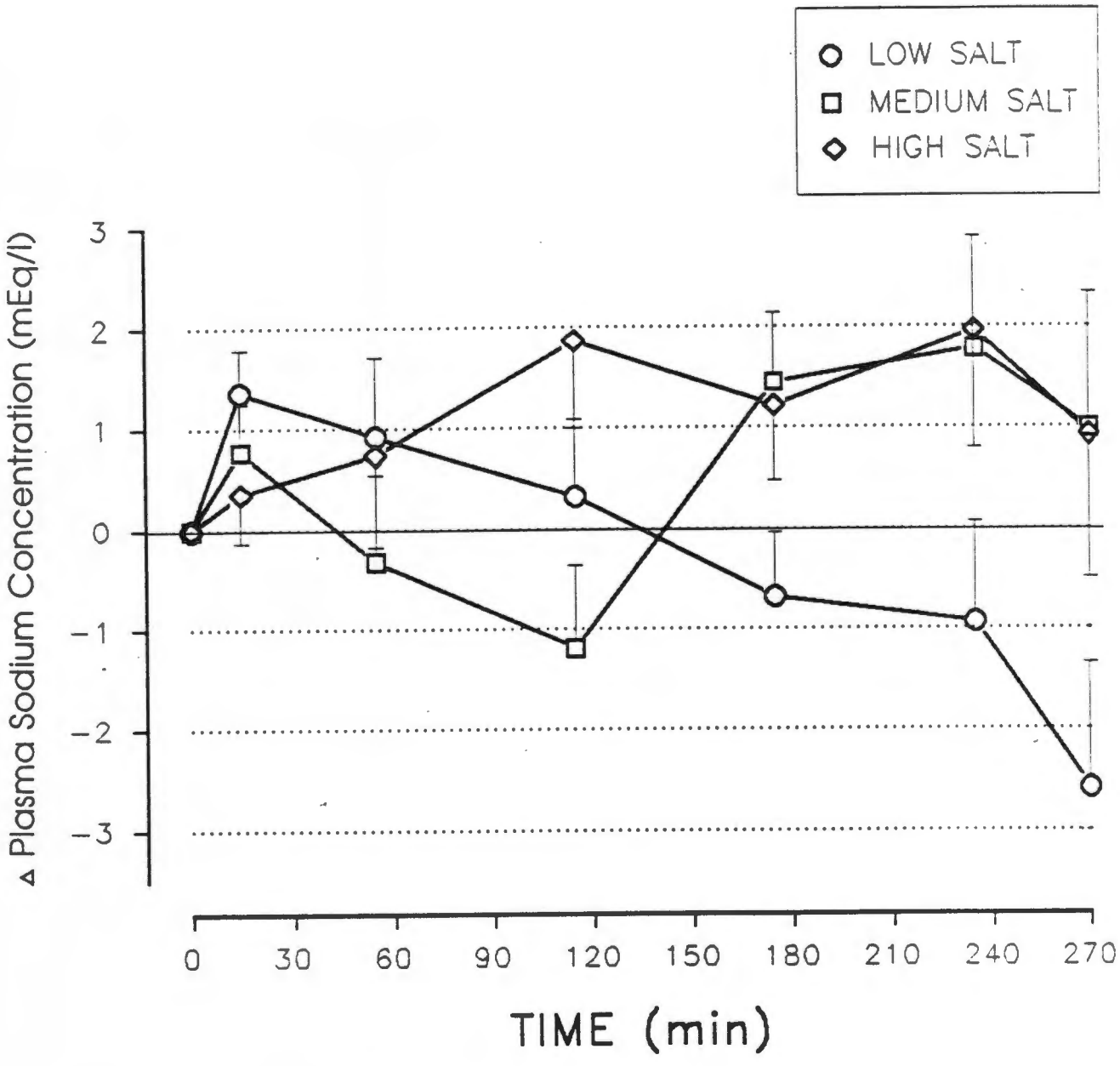

Figure 5.6 -- Change in plasma sodium concentration (mEq) during exercise and at the end of the 30 minute recovery period in the LS, the MS and the HS trials (mean ±SEM, n=6).

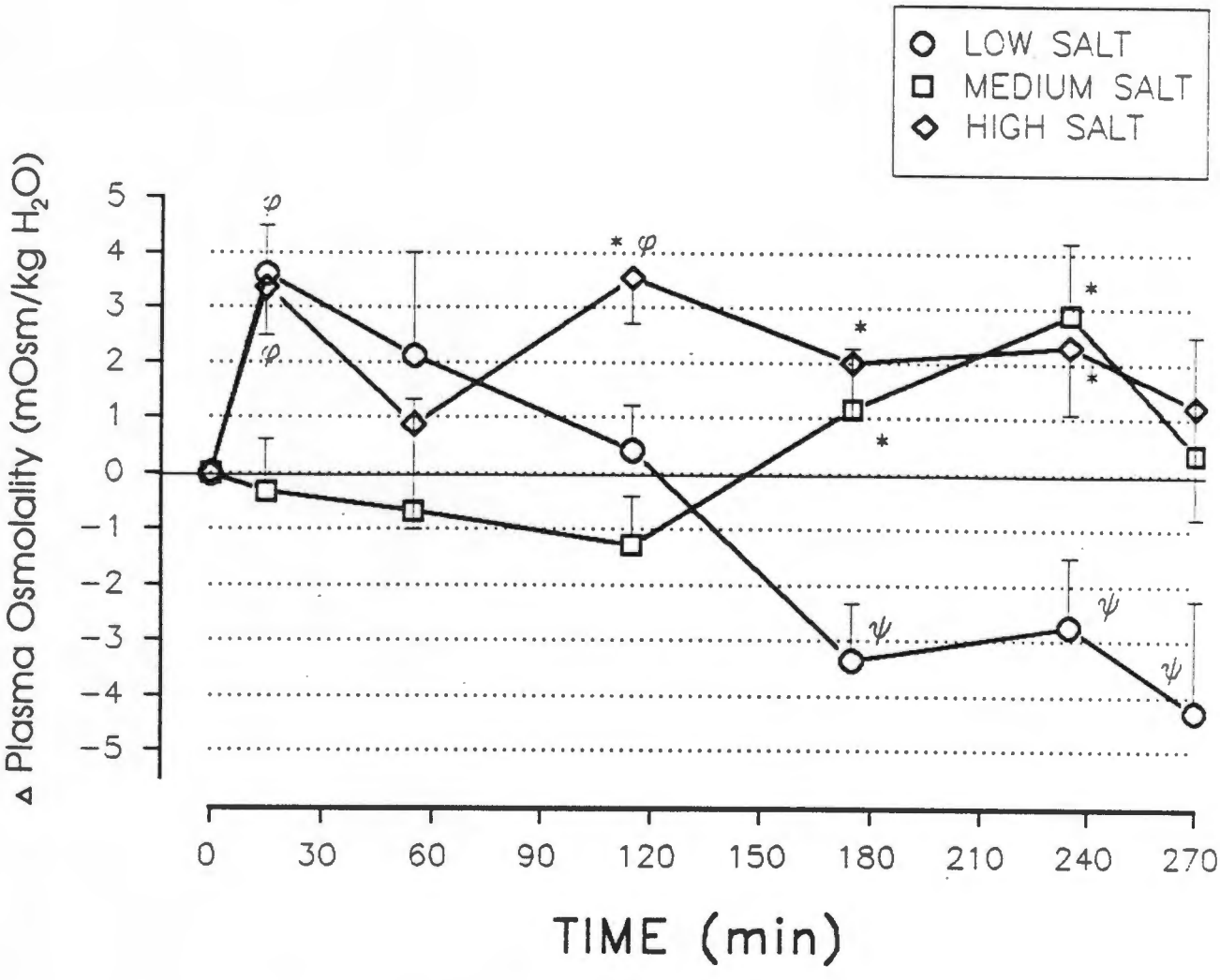

Figure 5.7 – Change in plasma osmolality (mOsm/kg H_2O) during exercise and at the end of the 30 minute recovery period in the LS, the MS and the HS trials (mean ±SEM, n=6). * denotes significantly different from Low Salt ($p<0.05$); φ denotes significantly different from Medium Salt ($p<0.05$); ψ denotes significantly different from T_{15} ($p<0,0033$).

Figure 5.8 shows the change in plasma protein concentrations. At 120 minutes, the change in plasma protein concentration in the MS and HS trial was significantly less than the change in the LS trial ($p<0.05$). At 180 minutes, the change in plasma protein concentration in the HS trial was significantly lower than in both the LS ($p<0.005$) and MS ($p<0.05$) trials. The change in plasma protein concentration in the MS was also significantly less than in the LS trial ($p<0.05$). The change in plasma protein concentration in the HS trial at the end of 30 minutes of recovery was significantly different from the change in both the MS and LS trials ($p<0.05$).

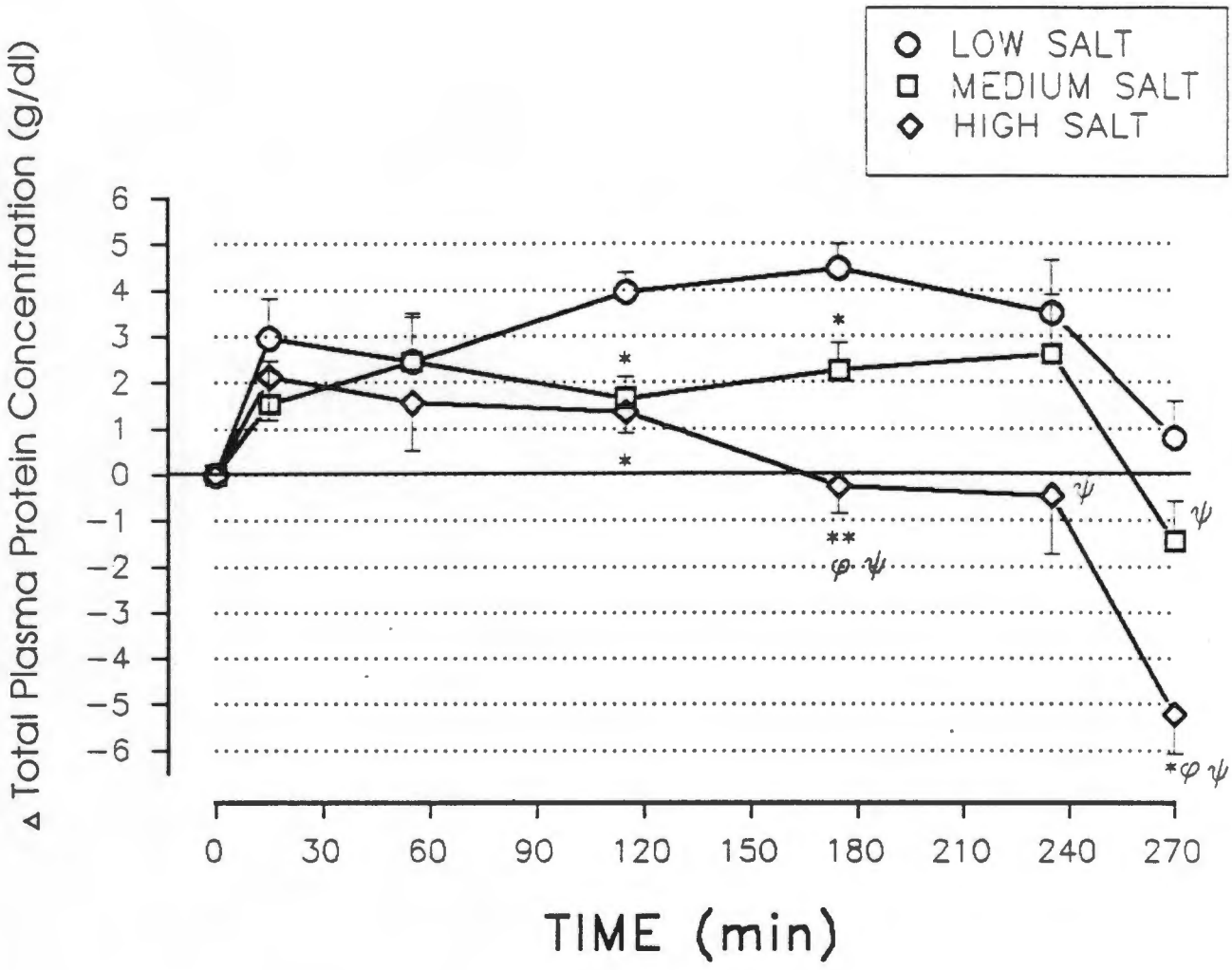

Figure 5.8 – Change in plasma protein concentrations (g/dl) during exercise and at the end of the 30 minute recovery period in the LS, the MS and the HS trials (mean ±SEM, n=6). *,** denotes significantly different from Low Salt (* $p<0.05$; ** $p<0.005$); φ denotes significantly different from Medium Salt ($p<0.05$); ψ denotes significantly different from T_{15} ($p<0.0033$).

5.2.6 CHANGE IN PLASMA VOLUME

The change in plasma volume during exercise was calculated relative to the plasma volume at 15 minutes because the aim was to determine the effect of fluid ingestion during exercise. If it was calculated relative to the plasma volume before exercise commenced then plasma volume would be affected by the onset of exercise (Coyle and Hamilton, 1990).

Plasma volume in the LS trial continued to decrease after 15 minutes and was significantly different from the 15 minute value at 180 and 240 minutes ($p<0.0033$) respectively (Figure 5.9). In contrast, plasma volume in the HS trial at 180 and 240 minutes was significantly higher than the 15 minutes value.

The change in plasma volume in the LS trial was significantly different from HS at 180 minutes ($p<0.05$) and at 240 minutes ($p<0.0005$) (Fig 5.9). The change in plasma volume in the MS trial was also significantly less than in the HS trial at 240 minutes ($p<0.05$) (Fig 5.9). There was a significant difference in the change in plasma volume between LS and MS at 240 minutes ($p<0.05$) (Fig 5.9).

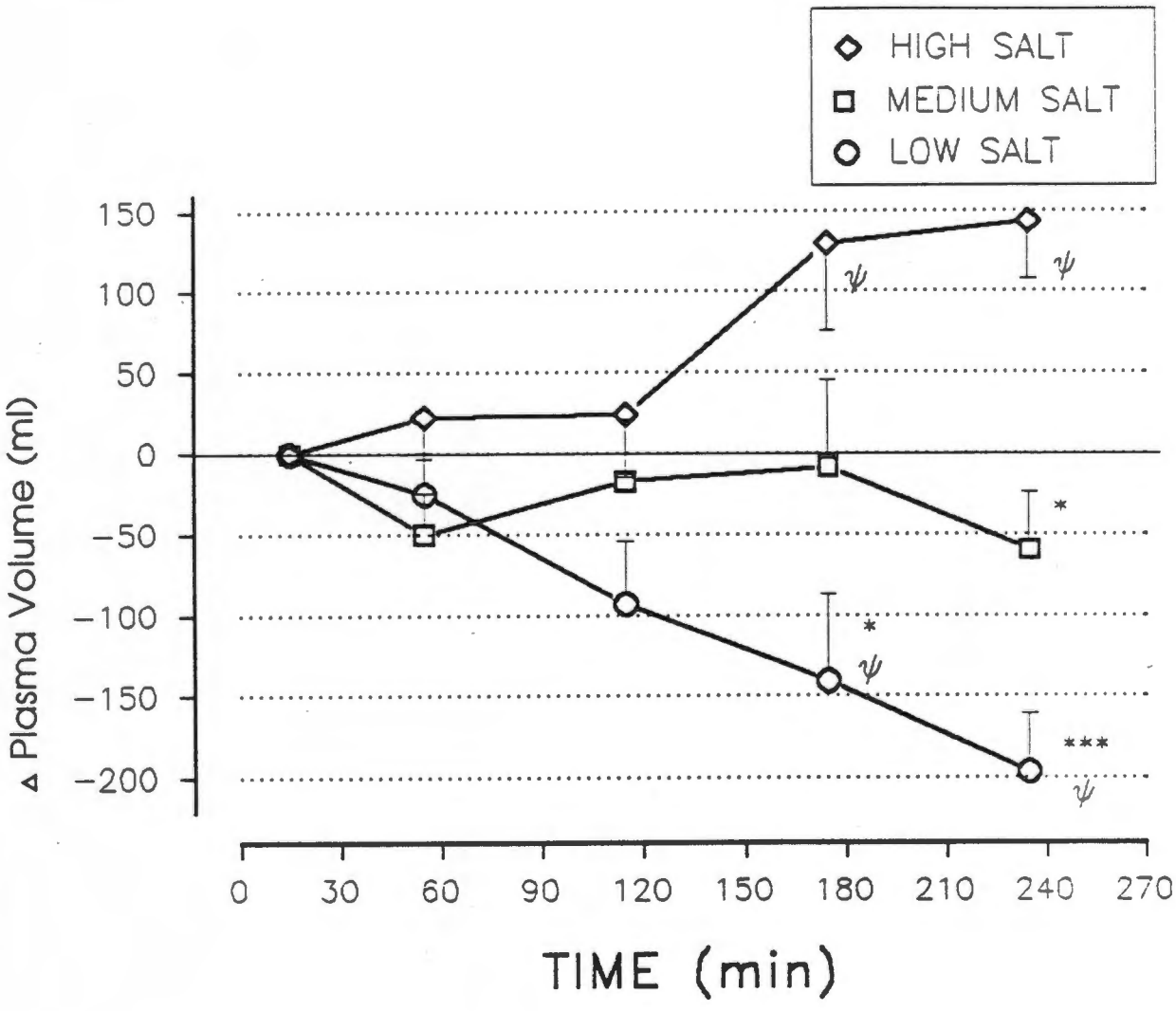

Figure 5.9 -- Change in plasma volume (ml), with respect to 15 minutes, during exercise in the LS, the MS and the HS trials (mean ±SEM, n=6). *,*** denotes significantly different from High Salt (* $p<0.05$; *** $p<0.0005$); ψ denotes significantly different from T_{15} ($p<0.0033$).

At 30 minutes recovery, plasma volume in the LS trials was significantly less than at time zero ($p<0.0033$). It was also significantly less than in both the HS ($p<0.005$) and MS ($p<0.05$) trials. Plasma volume in both the HS and MS trials was not different from pre-exercise values at 30 minutes recovery, and there was no significant difference between the two trials.

5.2.7 VOLUME CHANGES OF FLUID COMPARTMENTS DURING EXERCISE

The changes in volume of the fluid compartments were calculated with respect to 15 minutes. These calculations use the chloride shift method and assume that fluid absorption lags 15 minutes behind ingestion. Figures 5.9, 5.10, 5.11, 5.12 and 5.13 show the changes in the plasma volume, interstitial volume and the intracellular volume at 60, 120, 180 and 240 minutes with respect to 15 minutes.

There was a relative increase in plasma volume (Figure 5.9) in the HS trial over time. Plasma volume in the MS trial remained unchanged and gradually decreased in the LS trial over the 240 minutes. Plasma volume in the LS trial was significantly less than in the HS trial at 180 minutes ($p<0.05$) and 240 minutes ($p<0.0005$). Plasma volume in the MS trial was significantly less than in the HS trial at 240 minutes ($p<0.005$). Plasma volume at 240 minutes in the LS trial was also significantly lower than in the MS trial ($p<0.05$).

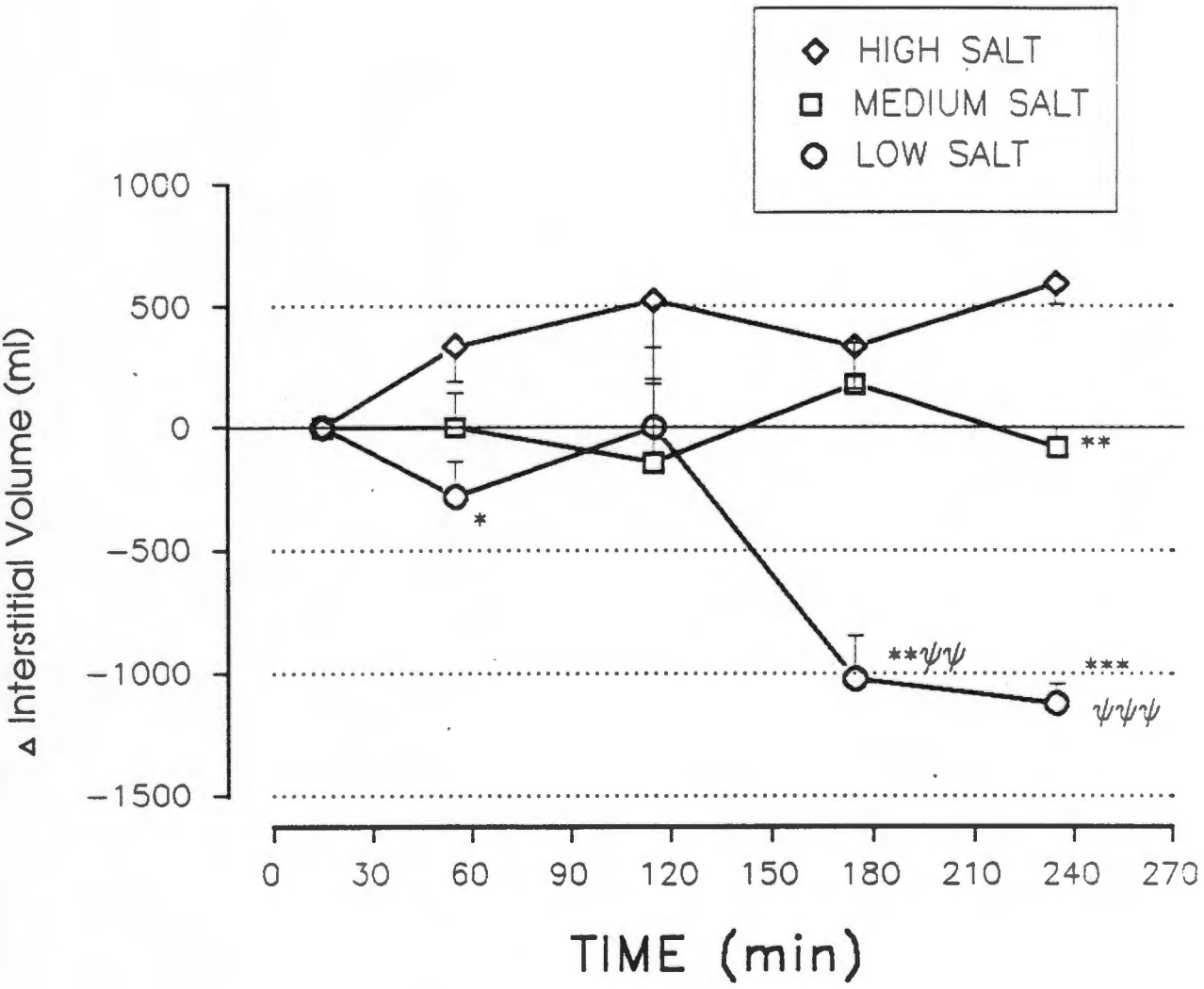

Figure 5.10 – Change in interstitial volume (ml), relative to 15 minutes, during exercise in the LS, the MS and the HS trials (mean ±SEM, n=6). The plot includes the outlier in the Low Salt Trial at 120 minutes. *,**,*** denotes significantly different from High Salt (* $p<0.05$; ** $p<0.005$; *** $p<0.0005$); ψ, ψψ, ψψψ denotes significantly different from Medium Salt (ψ $p<0.05$; ψψ $p<0.005$; ψψψ $p<0.0005$).

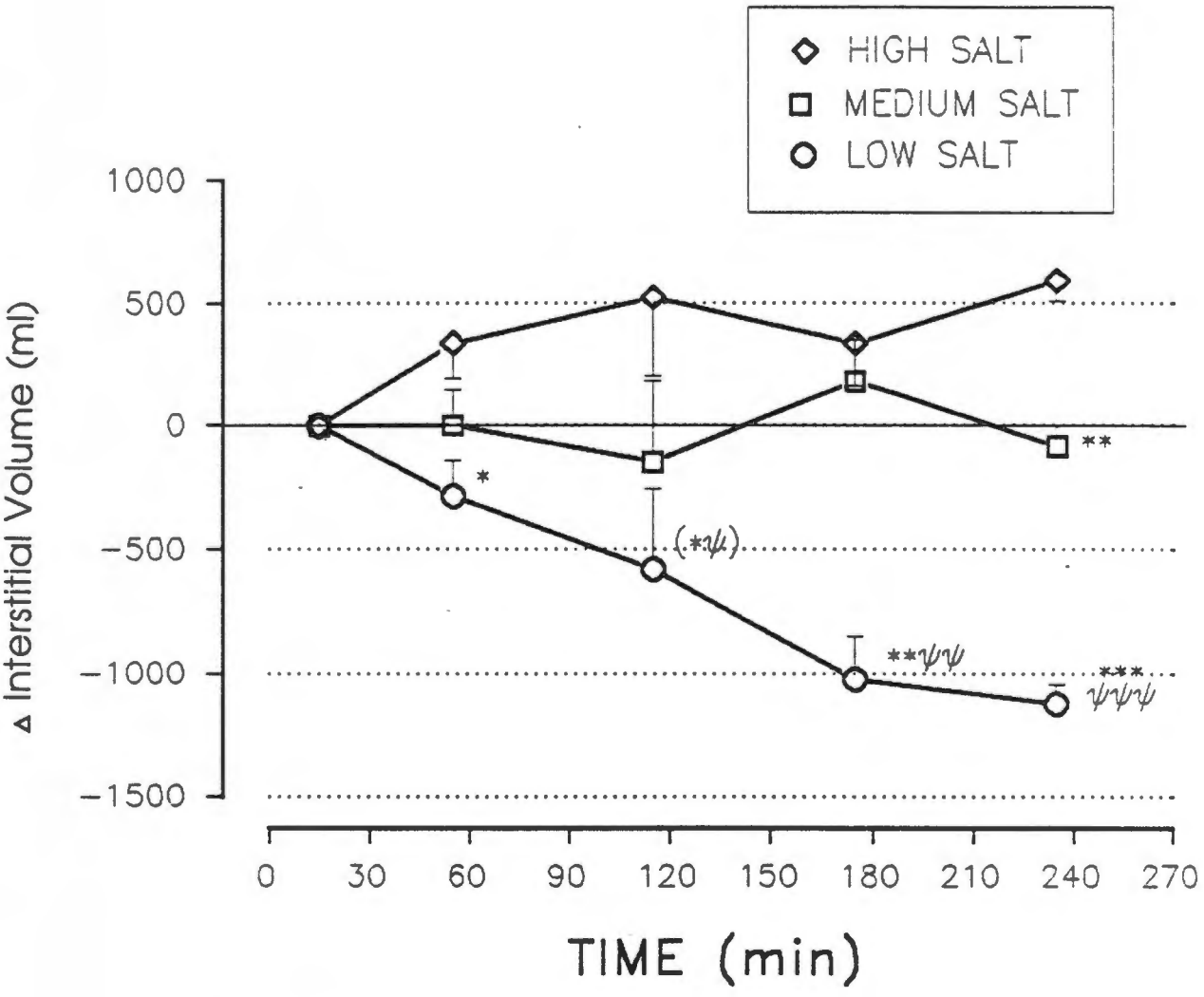

Figure 5.11 -- Change in interstitial volume (ml), relative to 15 minutes, during exercise in the LS, the MS and the HS trials (mean ±SEM, n=6). The plot excludes the outlier in the Low Salt Trial at 120 minutes. *,**,*** denotes significantly different from High Salt (* $p<0.05$; ** $p<0.005$; *** $p<0.0005$); ψ, ψψ, ψψψ denotes significantly different from Medium Salt (ψ $p<0.05$; ψψ $p<0.005$; ψψψ $p<0.0005$).

Interstitial volume (Figure 5.10 and 5.11) in the MS trial remained unchanged during the exercise bout, but there was a relative decrease in ISV in the LS trial. In contrast, there was a relative increase in interstitial volume in the HS trial. Interstitial volume in the LS trial was significantly lower than in the HS trial at 60 minutes ($p<0.05$), 180 minutes ($p<0.005$) and 240 minutes ($p<0.0005$). It was also significantly different from the interstitial volume of the MS trial at 180 minutes ($p<0.005$) and 240 minutes ($p<0.0005$). At 240 minutes, the interstitial volume in the MS trial was significantly less than in the HS trial ($p<0.005$).

Intracellular volume (Figures 5.12 and 5.13) in the LS trial remained unchanged during the exercise bout, but there was a relative decrease in ICV in the HS and MS trial. The change in intracellular volume in the LS trial was significantly less than in the HS trial at 180 and 240 minutes ($p<0.05$), and was also significantly less than in the MS trial at 180 minutes ($p<0.005$) and 240 minutes ($p<0.05$). There was no significant difference in the change in intracellular volume between the HS and MS trials.

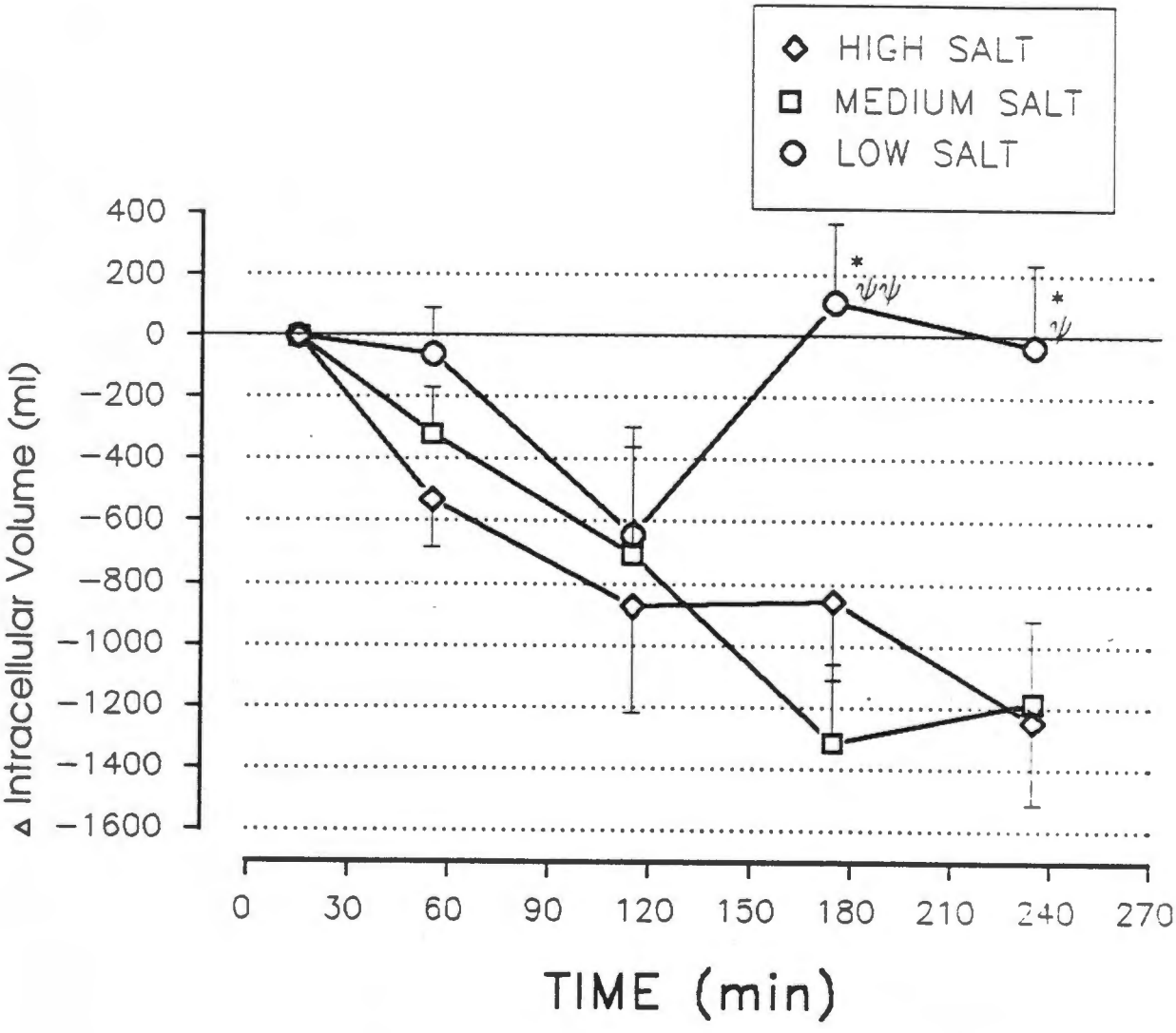

Figure 5.12 -- Change in intracellular volume (ml), relative to 15 minutes, during exercise in the LS, the MS and the HS trials (mean ±SEM, n=6). The plot includes the outlier in the Low Salt Trial at 120 minutes. * denotes significantly different from High Salt (* $p<0.05$); ψ, ψψ denotes significantly different from Medium Salt (ψ $p<0.05$; ψψ $p<0.005$).

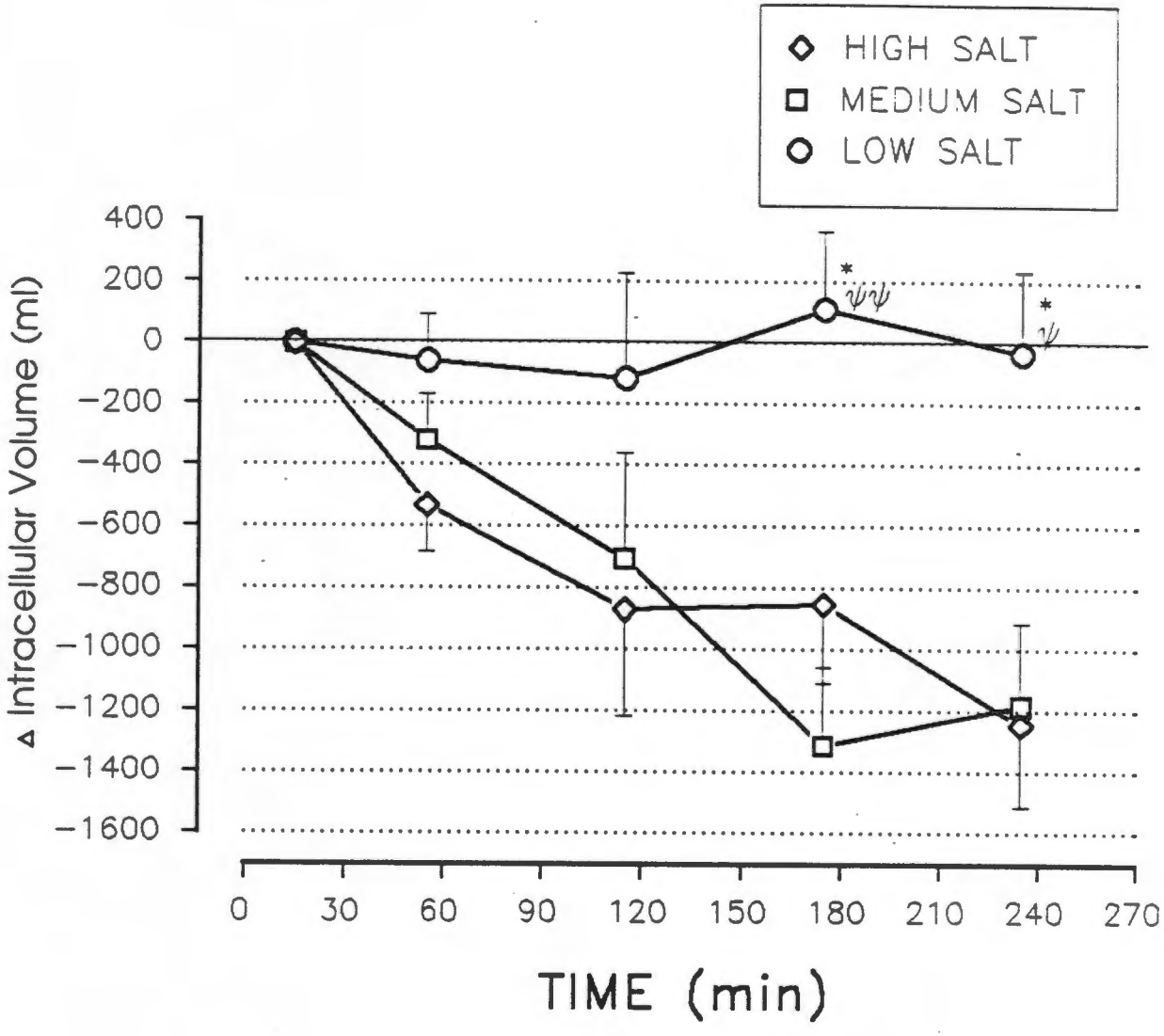

Figure 5.13 – Change in intracellular volume (ml), relative to 15 minutes, during exercise in the LS, the MS and the HS trials (mean ±SEM, n=6). The plot excludes the outlier in the Low Salt Trial at 120 minutes. * denotes significantly different from High Salt (* $p<0.05$); ψ, ψψ denotes significantly different from Medium Salt (ψ $p<0.05$; ψψ $p<0.005$)

An outlying value, that was greater than 3 standard deviations away from the means for interstitial and intracellular volume, was found in the LS trial at 120 minutes. It is statistically acceptable to call a value greater than 3 standard deviations away from the mean, an outlier and exclude it. The outlier was the result of the calculation of change in interstitial volume (Equation 11 in methods). The change in plasma chloride in the outlying subject was < 1, which resulted in an extremely large value for change in interstitial volume.

The results for the change in interstitial and intracellular volume, without the outlier, are presented in Figures 5.11 and 5.13 respectively. Removal of the outlier resulted in a gradual decrease in the interstitial volume and the maintenance of intracellular volume over the four hours in the LS trial.

Figures 5.14, 5.15 and 5.16 show the change in volume of the plasma, interstitial, intracellular compartments and cumulative fluid deficit over the 4 hours of cycling in the LS, MS and HS trials respectively. It can be seen that during the LS trial (Fig. 5.14), the majority of the fluid deficit originates from the interstitial volume. In the MS trial (Fig. 5.15) most of the fluid deficit came from the intracellular volume. In the HS trial (Fig. 5.16), it can be seen that all the fluid deficit is provided by the intracellular space, and that there is a shift of fluid, in excess of the fluid deficit, out of the intracellular space. This fluid results in the expansion of the extracellular fluid space (interstitial + intravascular compartments).

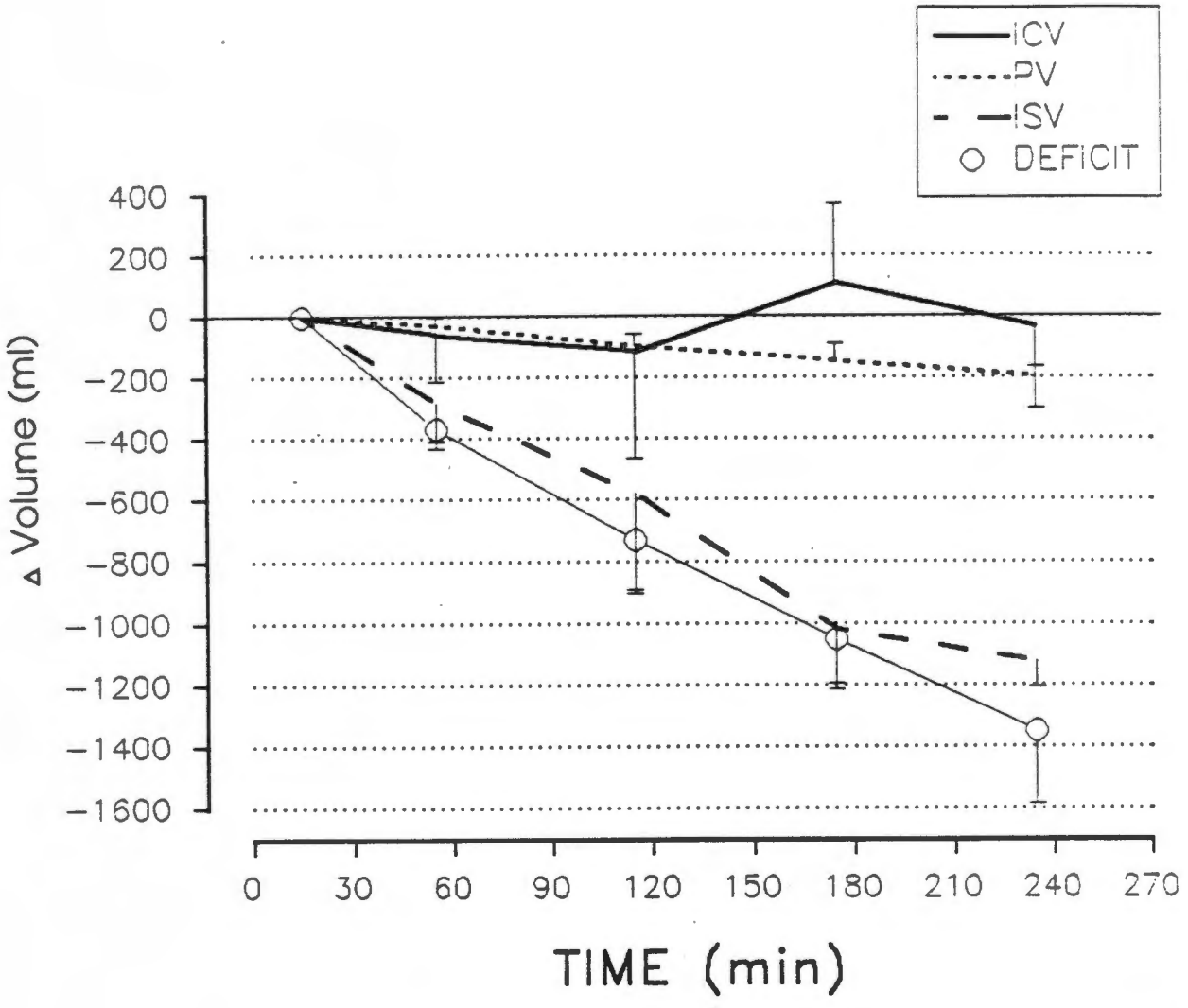

Figure 5.14 – Change in volume of the intravascular, interstitial and intracellular space in the Low Salt Trial during exercise (means ±SEM, n=6). The changes in volume were calculated relative to the compartment volumes at 15 minutes. The figure also shows the net fluid balance. It can be seen that in the Low Salt trial the ISV provides most of the fluid for the fluid deficit.

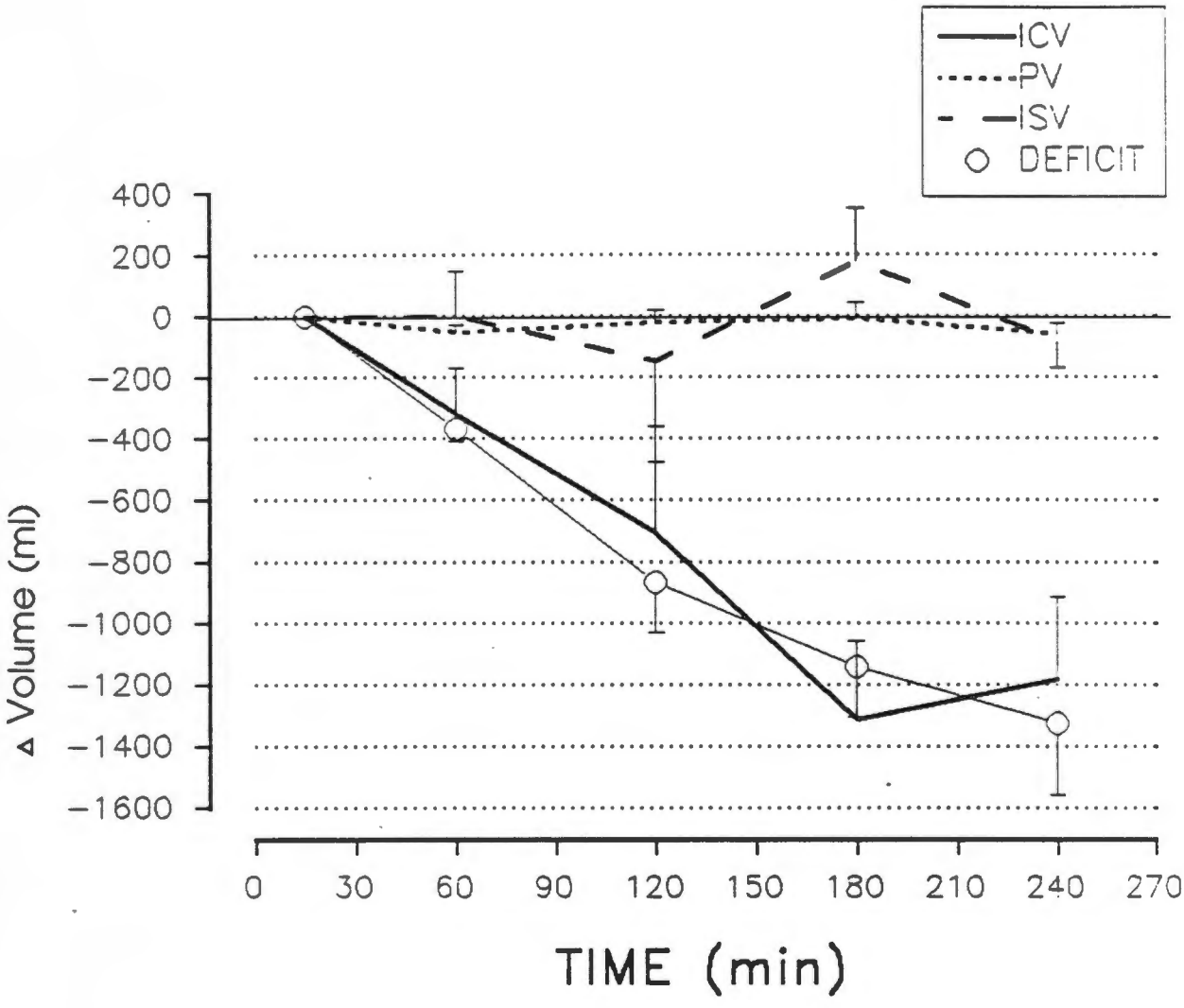

Figure 5.15 -- Change in volume of the intravascular, interstitial and intracellular space in the Medium Salt Trial during exercise (mean ±SEM, n=6). The changes in volume were calculated relative to the compartment volumes at 15 minutes. The figure also shows the net fluid balance. Notice that most of the fluid deficit was provided by the ICV, whereas PV and ISV remain unchanged.

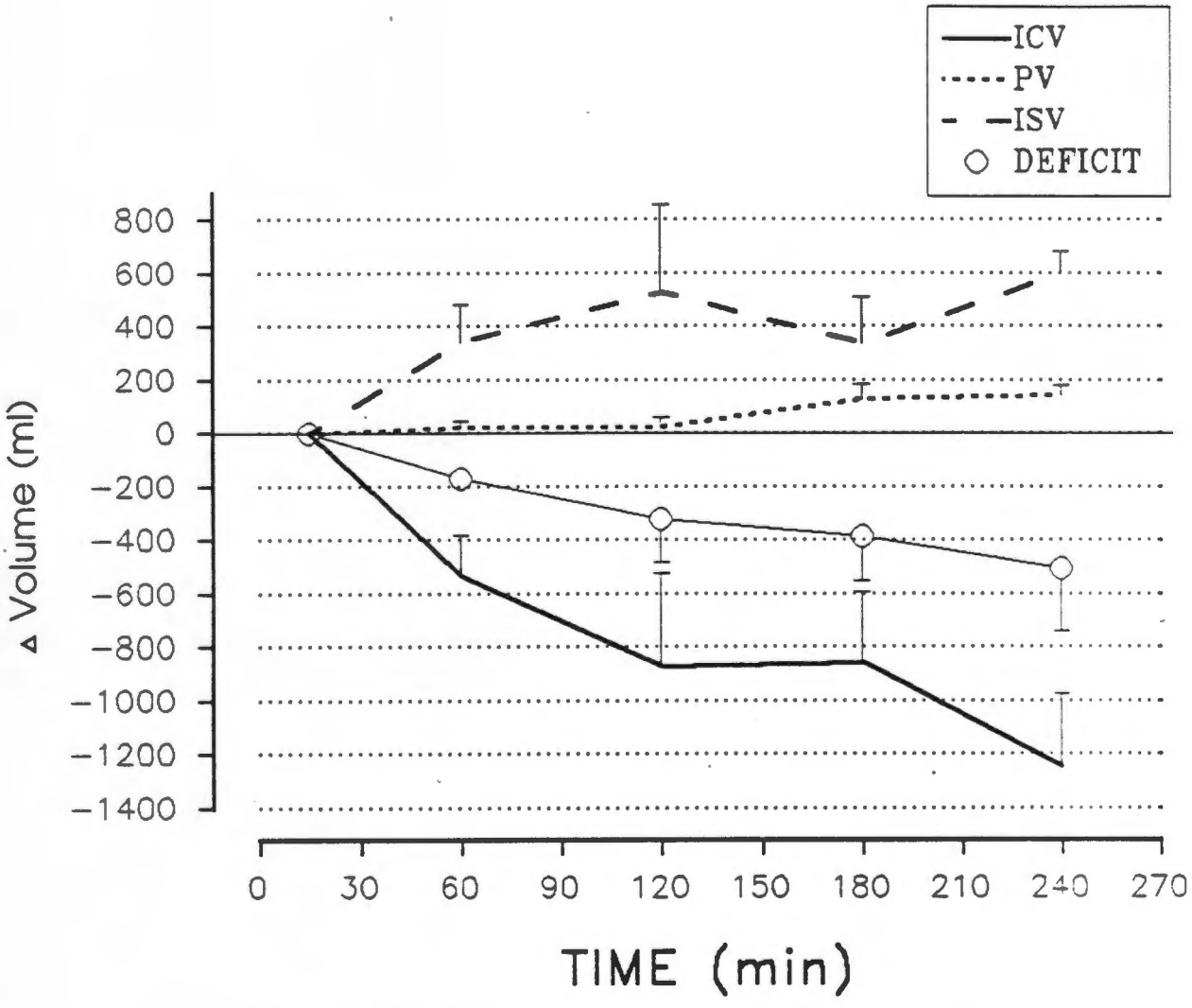

Figure 5.16 -- Change in volume of the intravascular, interstitial and intracellular space in the High Salt Trial during exercise (mean ±SEM, n=6). The changes in volume were calculated relative to the compartment volumes at 15 minutes. The figure also shows the net fluid balance. Loss of fluid from the ICV was greater than the fluid deficit. This fluid loss from the ICV, expanded the PV and ISV.

Figure 5.17 shows the change in volume of the three fluid compartments. It also expresses the contribution that each compartment makes to the fluid deficit as a percentage of the net fluid balance at 240 minutes of exercise. A negative percent contribution means that the volume of that compartment was expanded at 240 minutes, due to the shift of fluid into it from other compartments. A positive percent contribution, that is greater than 100%, means that all the fluid deficit came from that compartment and that there was an additional shift of fluid out of that compartment.

In the LS trial, 14.5% of the fluid deficit came from the plasma volume, 83.2% from the interstitial volume and 2.3% from the intracellular volume. In the MS trial, the plasma contributed 4.5% of the fluid deficit, the interstitial space contributed 5.3% and the intracellular volume contributed 89.2% of the deficit. All the fluid deficit in the HS trial came from the intracellular space, and fluid, in excess of the fluid deficit, moved out of the intracellular volume into the extracellular space. As a result, intracellular volume decreased by 245.5% of the fluid deficit, plasma volume expanded by 28.3% of the fluid deficit and interstitial volume expanded by 118.2% of the fluid deficit.

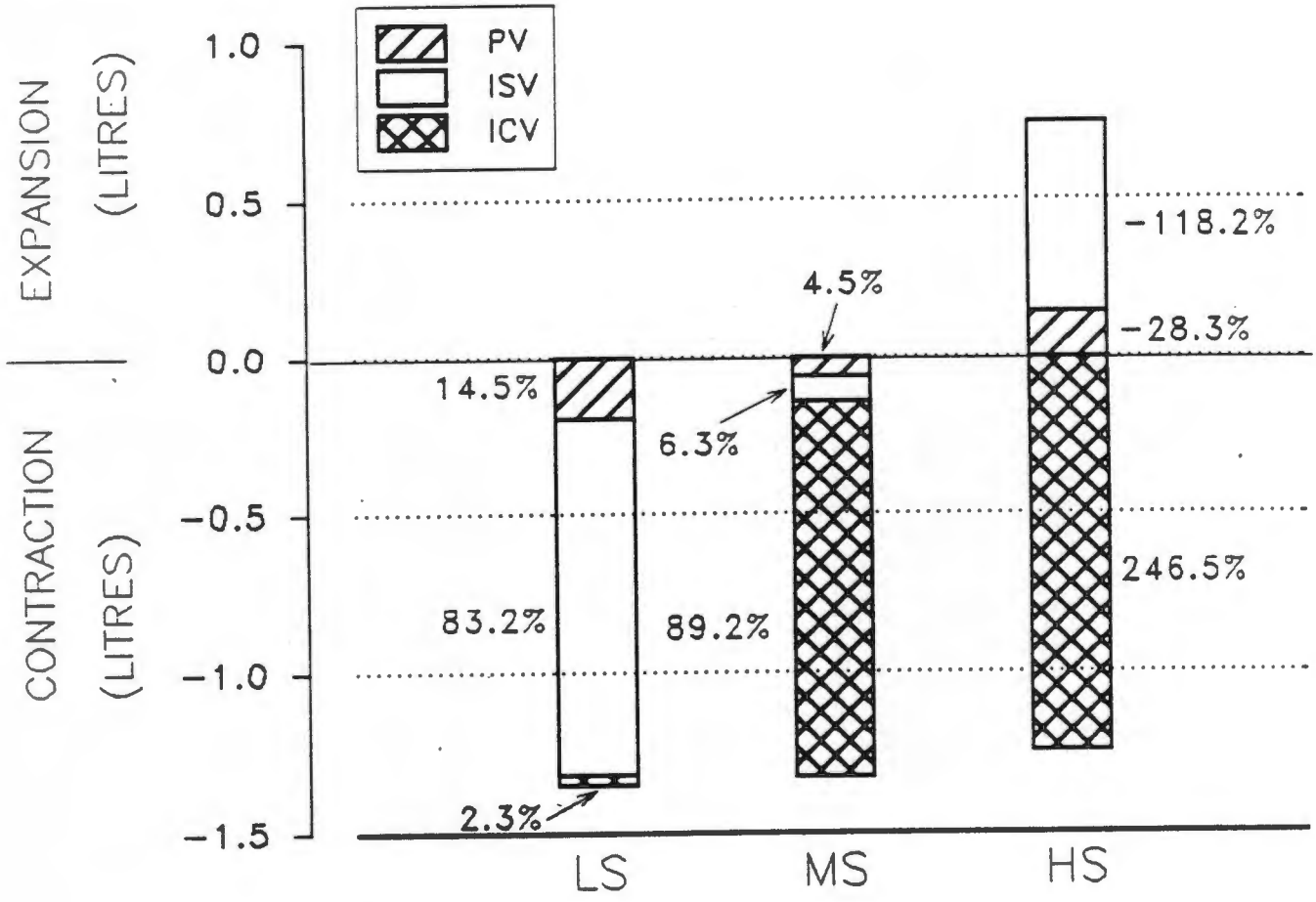

Figure 5.17 – Percent contribution that each compartment had made to the fluid deficit at the end of 4 hours of exercise (mean, n=6). A % greater than 100% means that the contraction or expansion of the compartment was greater than the net fluid deficit. A positive % means contraction of the compartment, whereas a negative % means an expansion of the compartment.

5.2.8 CHANGES IN VOLUME OF FLUID COMPARTMENTS AT THE END OF RECOVERY

Figure 5.18 shows the change in volume of the three compartments at the end of 30 minutes recovery. Changes in volume at the end of recovery were calculated with respect to pre-exercise values at zero minutes.

At the end of 30 minutes recovery, the change in plasma volume in the LS trial was significantly different from that in both the MS ($p<0.05$) and HS trials ($p<0.005$). There was no significant difference in the change in plasma volume between the HS and MS trials.

The change in interstitial volume in the LS trial was significantly less than in the HS trial ($p<0.005$). The change in volume of the interstitial fluid in the MS trial was significantly less than in the HS trial ($p<0.05$). There was no significant difference in the change in interstitial volume in the MS and LS trials.

The change in volume of the intracellular space in the LS trial was significantly different from the change in volume in the HS trial. Changes in intracellular volume were not significantly different between the HS and MS trials, or between the MS and LS trials.

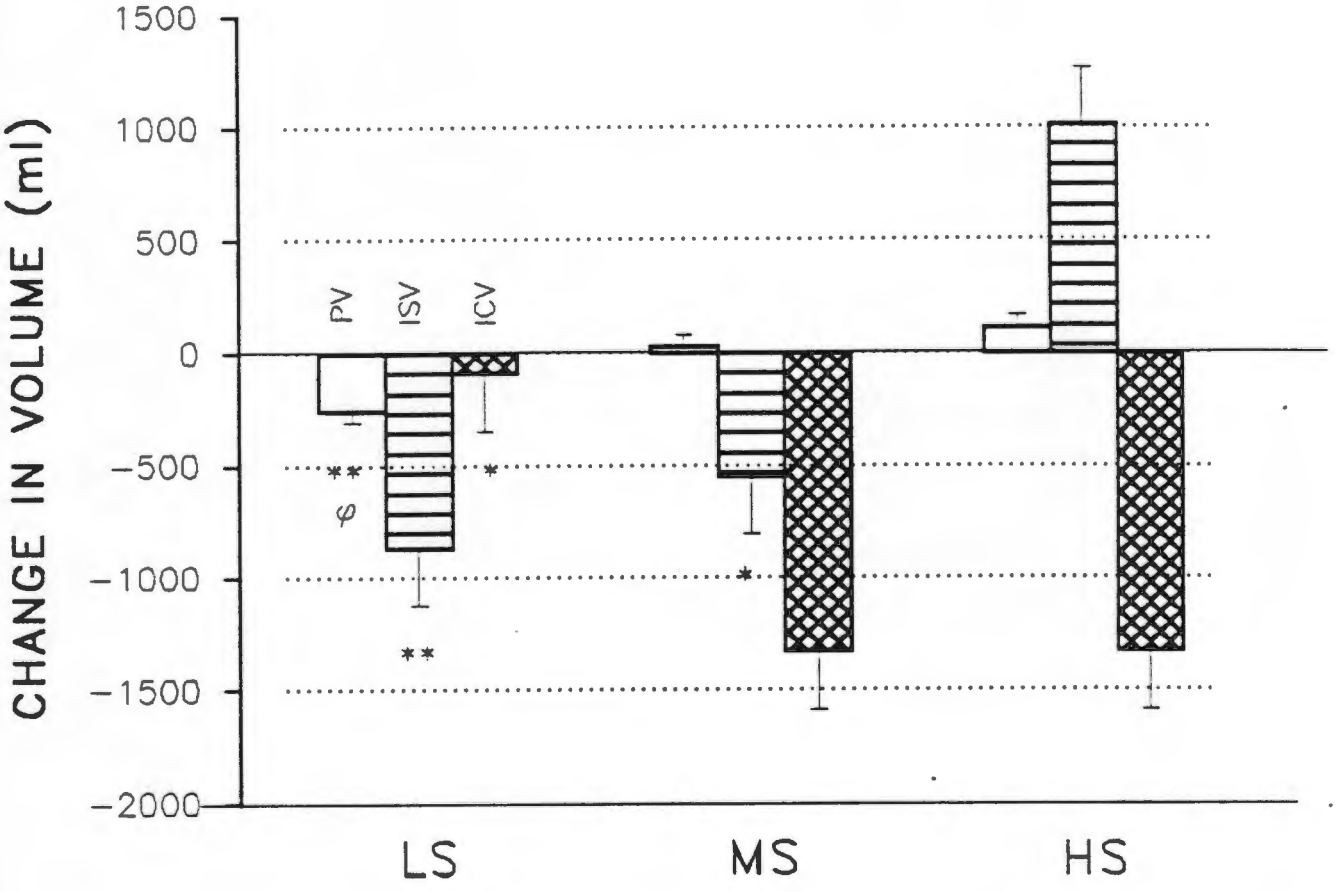

Figure 5.18 -- Change in volume (ml) of the intravascular, the interstitial and the intracellular space after the 30 minute recovery period in the LS, the MS and the HS trials (mean ±SEM). The change in volume was calculated relative to the compartment volume at 0 minutes. *,** denotes significantly different from the High Salt Trial (* $p<0.05$; ** $p<0.005$); φ denotes significantly different from the Medium Salt Trial (φ $p<0.05$).

Figure 5.19 shows the relative contribution that each compartment made to the fluid deficit in the three trials as a percentage of the net fluid balance at the end of the 30 minute recovery period. In the LS trial, 21% of the fluid deficit came from the plasma volume, 71.8% from the interstitial space and only 7.2% from the intracellular space. In the MS trial the fluid deficit was shared between the interstitial and the intracellular space. The plasma volume did not provide any fluid to the fluid deficit. In the HS trial, all the fluid deficit came from the intracellular space. As well as providing all the fluid deficit, an additional volume of fluid from the intracellular space, moved out of the intracellular into the extracellular space. At the end of the 30 minute recovery period following the exercise bout, there was a decrease in intracellular volume equal to 661% of the fluid deficit. Plasma volume was expanded by 55.5% of the fluid deficit and the interstitial volume was expanded by 505.5% of the fluid deficit.

Compartment volumes exhibited the same relative changes after 30 minutes recovery as were present at the end of the 4 hours of exercise. A direct statistical comparison between the recovery and the exercise can not be made because they were calculated with respect to different reference points. However, it still appears that during recovery there is a further shift of fluid out of the intracellular space and into the extracellular space in the MS and the HS trials.

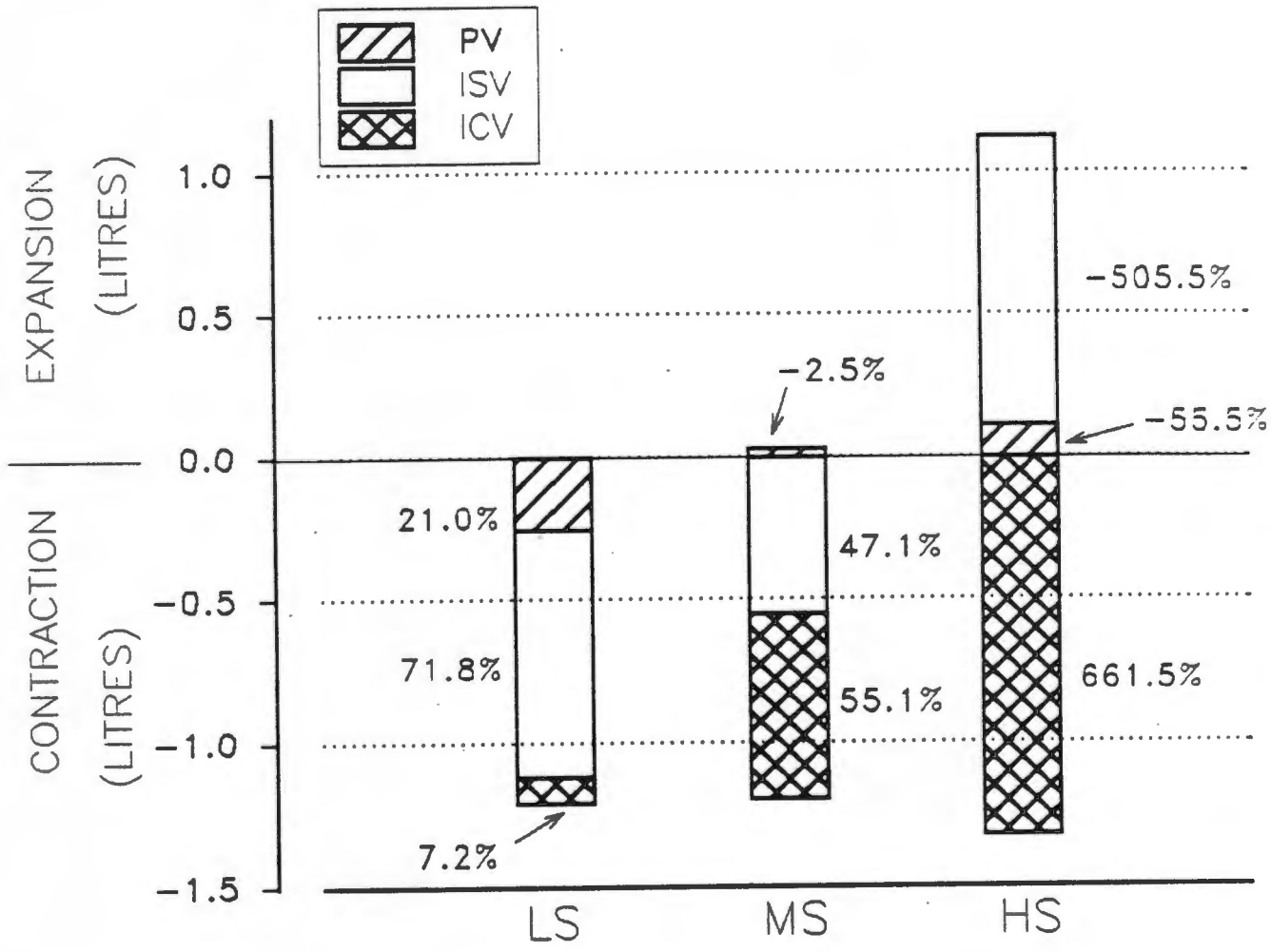

Figure 5.19 – Percent contribution that each compartment had made to net fluid deficit at the end of the 30 minute recovery period. A % greater than 100% means that the contraction or expansion of the compartment was greater than the net fluid deficit. A positive % means contraction of the compartment, whereas a negative % means an expansion of the compartment.

5.3 DISCUSSION

This project was designed to simulate conditions of prolonged, mild intensity exercise, where fluid replacement rates can match sweating rates. The aim was to determine whether sodium chloride ingestion in excess of that which is lost in sweat is necessary to optimise fluid balance under these conditions.

5.3.1 URINE PRODUCTION

The purpose of fluid ingestion during exercise, besides for delivering carbohydrate, is to replace the fluid lost due to sweating. If the fluid that has been ingested is lost via the urine, the effectiveness of the fluid replacement would be compromised.

Under the conditions in this study, it was found that only the High Salt drink (HS) resulted in an attenuation of fluid loss via the urine (Table 5.3). However, with the Low Salt (LS) and the Medium Salt (MS) trials there was a marked diuresis.

A possible explanation for the decreased urinary fluid loss in the HS trial is that the fluid was not being absorbed from the small intestine. This, however, is unlikely since it has been shown (Gisolfi, 1990b; Gisolfi et al, 1991) that fluid with a similar composition to the HS trial drink is rapidly absorbed from the small intestine.

During prolonged mild intensity exercise, a conspicuous diuresis has previously been noted when either water (Pearcy et al, 1956; Barr et al, 1990) or a carbohydrate-electrolyte beverage with a sodium concentration of 25mEq/l (Barr et al, 1990) is ingested in volumes matching sweat rate.

Pearcy et al (1956) noted that when subjects were given a sodium chloride solution of approximately the same concentration as sweat, there was a decrease in urinary fluid loss.

In contrast to this experiment, it was found that urinary fluid loss was not reduced when a beverage containing approximately the same sodium concentration as sweat was ingested during the MS trial. This may have been due to better absorption of the MS beverage in this trial than the sodium chloride solution in the experiment by Pearcy et al (1956), since their drink did not contain carbohydrate. It has been shown that when carbohydrate is added to a sodium chloride solution, there is an increase in the rate of fluid absorption from the small intestine of between 6 to 10 fold (Gisolfi et al, 1991). The increased rate of fluid absorption in the MS trial may have stimulated a diuretic response to the drink, despite the fact that the sodium chloride content of the drink matched that lost via sweating.

The possible mechanism involved in the attenuation of urine production in the HS trial will be discussed in the next section.

5.3.2 FLUID AND CATION BALANCE

The net fluid and cation balance, at any time point during this experiment, was the sum of the quantity of fluid and cations lost in the sweat, the volume and composition of the beverage ingested, and the volume and composition of the urine produced (Figure 5.5).

Sweating rate and the composition of the sweat is dependent on the exercise intensity (Fortney, 1985), the ambient conditions (Fortney, 1985), and the state of training and acclimatisation of the subjects (Nadel, 1974; Nadel, 1979). In this experiment, sweating rate and cation loss in the sweat were no different between the three trials. Therefore, the loss of fluid and cations in the sweat did not contribute to any differences between treatments seen in the net fluid and cation balance.

The volume of fluid ingested was the same for the three treatments. However, as the aim was to examine the effects of different sodium chloride concentrations on the fluid balance, the sodium chloride composition of the three drinks were different from one another.

The volume and composition of the urinary fluid loss was the result of the stress that the beverages placed on homeostatic control of isotonicity. Homeostatic control of the body's isotonicity is effected primarily by the kidneys. Receptors for changes in plasma sodium concentration, plasma osmolality and plasma volume act on the kidneys via hormonal messengers

in order to restore deviations from the set-point. The primary hormones involved in this homeostatic mechanism are the renin-angiotensin-aldosterone axis and vasopressin. Aldosterone responds primarily to changes in the plasma volume, while vasopressin responds primarily to changes in plasma sodium concentration and plasma osmolality.

In the LS trial, it was found that the kidneys had a positive free water clearance and a low sodium clearance. The ingestion of the LS beverage would have had the effect of decreasing the plasma osmolality. The major hormonal effect of this would have been a decrease in vasopressin, which would have lead to an increased fluid loss via the kidneys. Further, plasma volume continued to decrease during the 4 hours of cycling. This would have resulted in an increase in aldosterone and an increased sodium reabsorption by the kidneys. These two responses would have had the effect of opposing the decrease in plasma osmolality and plasma volume.

In the MS trial, there was a positive free water clearance until the end of the third hour of cycling, after which it became negative. Sodium clearance, however, started low and gradually increased over the duration of the experiment. This suggests that the initial response to the ingestion of the MS beverage was a decrease in vasopressin which would have resulted in less fluid reabsorption by the kidneys. During the latter part of the cycle, there may have been a decrease in aldosterone which would have resulted in a decreased reabsorption of sodium by the kidneys. Since water follows

sodium loss in the kidneys, an increased loss of sodium, due to the action of aldosterone, would have also increased the loss of fluid via the kidneys. These two hormonal responses resulted in a plasma sodium concentration and plasma osmolality that did not differ significantly from the control value for the duration of the experiment. It is noted that the observation that fluid loss in the MS trial was similar to that seen during LS trial, despite the ingestion of additional sodium. This may be because the sodium chloride content of the MS drink was still hypotonic relative to the plasma.

When the HS drink was ingested in volumes matching sweat rate, it would have had the effect of increasing the plasma sodium concentration and increasing plasma volume. Given the findings, that free water clearance was negative and sodium clearance was larger than in either the LS and MS trials, the predominant hormonal response would have been an increased vasopressin in response to the increased osmolality. This would have resulted in an increased fluid reabsorption by the kidneys and the observation that free water clearance was negative. Concomitantly, the increased plasma volume would have caused a decrease in aldosterone, which in turn would have decreased sodium reabsorption by the kidneys. The increased sodium loss via the urine in this trial did not however increase the urinary fluid loss as it seemed to do in the MS trial. This might have been due to the overriding effect of vasopressin in the HS trial. The above described hormonal response may explain the observation that plasma sodium concentration and plasma osmolality were not significantly different

from the control values for the duration of the experiment.

Figure 5.5 summarises the relationship between cation and fluid balance in the LS, the MS and the HS trials during the four hours of cycling and at the end of the 30 minute recovery period. A theoretical isotonic line ($y = 0.15x$) is also shown. The origin is the pre-exercise point at which stage no sodium or fluid had been lost. The area above the isotonic line represents a situation in which the body fluids are hypertonic. The area under the line represents the condition in which the body fluids are hypotonic. It can be seen that the ingestion of the low salt beverage (LS) resulted in a cation-fluid balance slightly below the isotonic line. Ingestion of the 50 mEq/l beverage (MS) resulted in a slight hypertonicity of the body fluids despite a negative cation and fluid balance. Ingestion of the 100 mEq/l beverage (HS) also resulted in a relative hypertonicity of the body fluids, but this was due to a positive cation balance and only a slight fluid deficit. The HS drink was the only condition in which a positive cation balance was attained under the experimental conditions.

In this figure there are two components to the measurement of disturbance of fluid-cation balance. 1) The perpendicular distance of the treatment point to the isotonic lines represents the degree of hypertonicity or hypotonicity, and 2) the distance of the point from the origin represents the overall disturbance of fluid and cation balance from the pre-exercise state.

If isotonicity is the preferred set-point during exercise, then the resultant fluid-cation balance during the experiment is the consequence of the homeostatic mechanism to maintain isotonicity with the different beverages. There are slight deviations from the isotonic line in all three trials, with the greatest being in the HS trial.

However, the aim of fluid ingestion for the athlete during exercise would be to try replace all the fluid loss via sweating, so as to maintain fluid balance. Although all trials received fluid in volumes approximating sweat rate, fluid balance was not well maintained in the LS and the MS trials due to diuresis. However, in the HS trial fluid loss via urine was attenuated due to the large quantity of sodium ingested.

5.3.3 FLUID SHIFTS

It should be pointed out that since calculations of fluid shifts using the chloride shift method involve a number of assumptions, the volume shifts yielded by these equations should not be taken as absolute numbers, but rather should be interpreted as relative changes in volume of the compartment. However, Nose et al (1985), have reported that the chloride method correlated well ($r=0.97$, $p<0.001$) with the ^{51}Cr-EDTA method of measuring changes in volume of the ECV.

5.3.3.1 DURING EXERCISE

The ingestion of the different beverages in this experiment resulted in significantly different fluid shifts occurring between the intravascular, interstitial and intracellular compartments.

As can be seen from the results, during exercise, fluid shifts were calculated relative to 15 minutes of exercise. It was found that when a low salt beverage was ingested, most of the fluid deficit due to the loss of fluid in the urine and sweat, came from the interstitial compartment, and therefore the extracellular compartment (Fig. 5.14). During ingestion of the moderate salt beverage, most of the fluid loss came from the intracellular compartment (Fig. 5.15). During exercise in the HS trial, all of the fluid loss came from the intracellular compartment. Further, in the HS trial, in addition to the decrease in the volume of the intracellular compartment due to urine and sweat losses, there was a shift of fluid from the intracellular compartment to the extracellular compartment causing an expansion of the interstitial and intravascular compartments (Fig. 5.16).

These findings suggest that the amount of sodium absorbed determined the extent of fluid loss from each compartment.

Movement of fluid between the intracellular and the interstitial space is a result of an osmotic pressure difference between the two compartments. The membranes separating the intracellular and interstitial space are

selectively permeable to electrolytes, but permeable to water. Therefore if a solute is added to the one side, fluid will move down the osmotic gradient towards the side with more solute. This will equalise the osmotic pressure difference between the two sides.

Since sodium is the primary cation of the extracellular space, a large proportion of the absorbed sodium will remain in the extracellular space. The net sodium content of the extracellular space is a balance between sodium absorbed from the gut and sodium lost in the urine and sweat. If the net sodium content of the extracellular space increases, there will be an increased osmotic pressure in this space. Due to the resulting osmotic pressure difference between the extracellular and intracellular spaces, fluid will then move by osmosis from the intracellular compartment into the extracellular compartments. If on the other hand, the ingested fluid dilutes the solute contents of the extracellular space, fluid will move by osmosis from the extracellular space into the intracellular space.

Hence, in this study it was observed that the more sodium that was ingested, and therefore the greater the solute content of the extracellular space, the greater the shift of fluid out of the intracellular space.

5.3.3.2 AT THE END OF THE RECOVERY PERIOD

At the end of the 30 minute recovery period, the fluid shifts profile of the three trials, with respect to pre-exercise values, were similar to those seen at

the end of 4 hours of exercise. Figure 5.17 shows the change in volume of the fluid compartments in the different trials. The trend is still the same, namely that the more sodium chloride ingested during the exercise bout, and therefore the greater the sodium content of the extracellular space, the greater the shifts of fluid from the intracellular to the extracellular space.

5.3.4 PLASMA VOLUME

From the results in this study, it also appears that the change in plasma volume is affected by the quantity of sodium chloride ingested during the exercise bout (Figure 5.9). The more positive the sodium balance, the greater the increase in plasma volume. Nose et al (1988a) showed that during dehydration, the less sodium lost via sweat, the less the decrease in plasma volume.

The change in volume of the intravascular space mirrored changes in volume of the interstitial space. However, the magnitude of PV changes were smaller than in the interstitial space. This suggests that plasma volume is preserved during exercise.

Changes in total plasma protein concentration (Figure 5.8) mirrored changes in plasma volume.

5.3.5 HEART RATE

Although the change in plasma volume was significantly different between all three trials after 240 minutes of cycling in this experiment, no significant differences in heart rate between the treatments was observed (Figure 5.1). It has previously been observed that during exercise a decrease in plasma volume is associated with an increased heart rate, while an increase in plasma volume may cause a decrease in heart rate compared to control values (Montain and Coyle, 1992b in press).

In this trial the lack of correlation between change in plasma volume and heart rate could be due to the method of ascertaining heart rate. Heart rate was measured hourly just before the rest period. This discrete measurement may not provide information about heart rate during the rest of the hour and could be the reason why differences were not observed. A more informative method of measuring differences in heart rate between the trials may have been to record heart rate continuously and to compare the sum of hourly heart beats or calculate the average heart rate during each hour.

5.3.6 RECTAL TEMPERATURE

A further finding from this study was that during exercise at 55% of VO_{2max}, while ingesting fluid at rates approximating sweat loss, the ingestion of large amounts of sodium did not alter the rectal temperature response, and sweat rate was not decreased compared to the ingestion of a low salt

beverage (Figure 5.2).

The study by Montain and Coyle (1992b) has shown a relationship between core temperature and plasma sodium concentration. Since plasma sodium concentration (Figure 5.6) was unaffected by the ingestion of the high salt beverage, it was not surprising to find that rectal temperature was not correlated to the quantity of sodium chloride ingested. Had plasma sodium concentration increased with the ingestion of the high salt beverage during the HS trial, an increase in rectal temperature compared to the LS trial would have been expected. Plasma sodium concentration did not increase in the HS trial because of a large sodium excretion by the kidneys and the retention of ingested fluid in the extracellular space.

5.4 CONCLUSIONS

During exercise at 55% VO_{2max} in 20°C at 70% relative humidity, the ingestion of a 8% carbohydrate solution containing sodium chloride in amounts approximately double that which was lost via sweat, and in volumes matching the sweat rate, 1) improved fluid balance by decreasing the diuresis associated with high rates of fluid ingestion; 2) did not result in an increase in the plasma sodium concentration; and 3) did not adversely affect thermoregulation by decreasing sweat rate or by increasing rectal temperature.

It can therefore be concluded that in conditions where fluid ingestion matches sweat rate, optimum fluid replacement relies on sodium chloride being ingested in quantities greater than which it is lost from the body via sweat.

THESIS CONCLUSIONS

The first study suggests that during exercise simulating competitive conditions, plasma volume is not increased when ingested fluid replaces only half that lost through sweating, despite the addition of sodium chloride in amounts double that which is being lost via sweating. The second study uses an exercise model in which all fluid lost via sweating is replaced, and shows an increased plasma volume and attenuation of urine output when ingesting sodium chloride in amounts double that which is being lost via the sweat. When comparing the two conditions examined in this thesis, it can be concluded that ingestion of sodium chloride in quantities greater than the amounts being lost in the sweat is only beneficial when the rate of fluid ingestion matches the sweat. Since most athletes do not replace all the fluid that they are losing due to sweat during a race, there is no benefit in advising athletes to ingest additional salt, particularly if they are going to be exercising in the heat. Sodium chloride tablet ingestion may be of benefit in conditions where exercising individuals are able to ingest fluid in volumes matching sweat rate, such as troops in desert manoeuvres.

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