THE EFFECT OF DEHYDRATION AND REHYDRATION ON PHYSIOLOGICAL PARAMETERS ASSOCIATED WITH MAXIMAL ROWING PERFORMANCE

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ABSTRACT

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This study examined:

a) the effects of 24 hours of dehydration on body weight, plasma volume and various urinary variables and compared the efficacy of drinking water and solutions of varying sodium chloride concentration in restoring plasma volume after dehydration; and

b) the metabolic processes during performance in a maximal rowing trial following partial rehydration with water after rapid weight loss.

In study a), eighteen healthy active male subjects (non rowing) and eight highly trained elite oarsmen were voluntarily recruited to take part in the experiment. Body weight was reduced over 24 hours using a combination of exercise-induced dehydration, starvation and fluid restriction and was followed by a 90 minute period of fluid ingestion (250 ml cold fluid every 15 min.). The eighteen non rowing subjects were randomly assigned to one of three "saline groups" that were allocated, in a single blind manner, either a 0.1%, 0.2% or 0.3% sodium

chloride solution respectively to drink during the rehydration period. The eight rowers were allocated water. Venous blood samples were obtained without stasis before and after dehydration and after rehydration to determine changes in plasma volume. Urinary electrolytes were analysed on urine collected over the 24 hour dehydration period. This procedure, however, applied to the saline groups only. A control sample was collected during a similar 24 hour period without dehydration.

Body weight decreased 5.16 ± 0.14% for the rowers and 3.72 ± 0.18 % for the saline groups ($p<0.05$) after 24 hours of dehydration. Likewise, relative plasma volume decreased 12.5 ± 1.4 and 13.3 ± 0.6% for the rowers and saline groups respectively. There was no significant difference between the groups for this parameter. The calculation of the percent contribution of plasma volume to the change in total body water after dehydration demonstrated that the rowers lost significantly less plasma volume per unit loss of total body water than the saline groups ($p<0.05$).

The ingestion of 1.5 litres of saline over a 90 minute period was associated with significant plasma volume increases of 4.80 ± 0.54%, 6.42 ± 0.65% and 10.17 ± 0.65% in the 0.1%, 0.2% and 0.3% saline groups respectively ($p<0.05$). Each drink, however, left a significant plasma volume deficit after rehydration ($p<0.05$). The ingestion of water was associated with a plasma volume restoration of 6.02 ± 0.62% ($p<0.05$).

In study b), eight male rowers performed two maximal rowing trials, separated by one week, using a random cross-over design. One trial (N) was performed in the euhydrated state and the other (D) after the 24 hours of dehydration followed by rehydration. Both trials were performed on a Gjessing rowing ergometer and each trial required the completion of 4200 flywheel revolutions with 3 kg resistance. During each trial, heart rate was monitored and expired gases were collected for analysis. Muscle samples were obtained from the vastus lateralis by percutaneous needle biopsy before and after exercise and analysed for glycogen content. Venous blood samples were obtained without stasis before and five minutes after exercise and analysed for lactate.

The D trial was rowed with plasma volumes on average 7.8 ± 1.8% lower than the N trial ($p<0.05$). Times to complete the rowing trials increased from 7.02 ± 0.17 min. for the N trial to 7.38 ± 0.21 min. for the D trial ($p<0.05$). The average respiratory exchange ratio and net blood lactate accumulation decreased from 1.12 ± 0.01 and 8.77 ± 0.31 mmol l^{-1} for the N trial to 0.92 ± 0.02 and 6.77 ± 0.24 mmol l^{-1} for the D trial respectively ($p<0.05$). Muscle glycogen content decreased by 203.6 ± 18.6 mmol kg^{-1} D.W. for the N trial and 139.9 ± 13.4 kg^{-1} D.W. for the D trial indicating a lower rate of glycogen utilization during the D trial ($p<0.05$). No significant differences in oxygen uptake and heart rate were observed between trials. These results suggest that the dehydration/rehydration protocol used by these lightweight rowers reduces maximal rowing performance. This impaired exercise capacity may be attributed to, not only lowered plasma volume, but also decreased glycogen utilizing capacity.

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CHAPTER 1

GENERAL INTRODUCTION

The sport of rowing is conducted in two weight categories: lightweight (under 70 kg) and heavyweight (open). In sports that are conducted in weight divisions, many competitors believe that if they were to compete in a division below their natural weight, they would hold an advantage over their opponent. The absence of graduated weight classifications, as found in sports such as wrestling and Olympic weight lifting, has created a situation where many rowers, not successful in the heavy weight-category, attempt to compete in the lightweight category at body weights substantially below what may be considered their 'natural' level. The athletes frequently decrease their body fat stores to minimal levels and then resort to dehydration regimens to achieve the required weight. The most common dehydration regimen used by rowers involves relatively severe food and fluid restrictions in conjunction with training over the last few days prior to the event. If necessary, in the afternoon of the day prior to the race, the rower will exercise in heavy, and often water-proof, clothing. On the day of the

weigh-in, if still overweight, the rower will usually go without food or fluid until having weighed-in. If necessary, they will go on further "sweat runs" until the required weight is attained.

The Fédération Internationale des Sociétés d'Aviron (F.I.S.A.) rules of racing require that on the day of the race, the lightweight rower is required to weigh-in not more than two hours and not less than one hour before the advertised starting time of the event. If the rower successfully weighs-in two hours before the event starting time, it leaves approximately 90 minutes in which to attempt to rehydrate, assuming 30 minutes or so are usually spent warming up on the water. During this rehydration period, the rowers, having not eaten for quite some time, usually elect to consume food and drink only to satiety, rather than concentrating on drinking the quantity of fluid that is required physiologically. It is likely, therefore, that lightweight rowers are competing whilst in a significantly dehydrated state.

Anecdotal evidence exists that suggests that many coaches appear to be adopting the policy of selecting crews with larger statures than would normally have been selected for a lightweight crew. It is then often necessary for the members to dehydrate before each race. It is appropriate, therefore, to ask whether dehydration has a detrimental effect on rowing performance. An examination of the available literature demonstrated that the effect of dehydration on rowing performance has not been reported previously. The major aim of the present study, therefore, was to study the effect of rapid weight loss and attempted rehydration on competitive rowing performance. In these

studies, moderate weight losses over 24 hours were required. This was to be achieved by food and fluid restriction over the entire period and a light sweat run the afternoon prior to the day of an "all-out" rowing trial. Unlike the actual situation, however, subjects were rehydrated using a structured fluid ingestion regimen for a 90 minute period, which corresponds to the maximal time usually available to the athlete to rehydrate after the weigh-in. During the rehydration period, 1.5 litres of water were required to be consumed. The "dehydrated" rowing trial was compared to a normal control trial conducted seven days apart using a random crossover design. Physiological parameters associated with metabolic function were measured during each trial.

A possible limitation to the study is that performance may decrease in the dehydrated trial due to psychological reasons. It is believed, however, that this was adequately controlled for by selecting subjects who were training in a squad from which would be selected a crew to represent Australia at the next World Championships. Dehydration regimens, similar to the one used in the present study, also did not present as a novel situation to any of the rowers.

Dehydration has been previously associated with a substantial decrease in maximum physical work capacity (Saltin, 1964a, 1964b; Bosco *et al.*, 1974), decreases in plasma and blood volume (Costill and Fink, 1974; Costill and Sparks, 1974), stroke volume and cardiac output (Saltin, 1964a, 1964b; Sproles *et al.*, 1976). Anaerobic capacity appears to be unaffected by dehydration (Jacobs, 1980). The effects of

dehydration on strength and localized muscular endurance have been studied with equivocal results (Torranin *et al.*, 1979; Serfass *et al.*, 1984).

The loss of water after dehydration is replaced most conveniently by the oral consumption of fluids. The efficacy of a fluid for rehydration and the subsequent restoration of plasma volume is affected by the factors listed below:

a) Gastric emptying rate slows as the energy content of the drink increases (Brener, 1983; Hunt and Stubbs, 1975; Shafer *et al.*, 1985);

b) Hypotonic saline solutions (0.2-0.4% w/v) enhanced gastric emptying rate compared to distilled water (Hunt and Pothak, 1960);

c) Thirst is not a reliable indicator of hydration status (Costill and Sparks, 1973; Nielsen *et al.*, 1984a, Seckle *et al.*, 1986); and

d) The fluid must be sufficiently palatable to encourage a maximum volume of fluid to be consumed (White and Ford, 1983).

This study, therefore, also observed the effect of rehydrating with three saline solutions, up to a maximum concentration of 0.3% w/v, on the restoration of plasma volume.

The practice of dehydrating to make weight is continued despite severe warnings that it is potentially dangerous to one's health. Of particular note, position statements condemning the practice have been published by the American Medical Association (1956, 1967) and by the American College of Sports Medicine (1976). The following extracts have been obtained from the position statement issued by the latter organization in relation to wrestling practices:

> ...Under existing rules and practices, it is not uncommon for an individual to repeat this weight-losing process many times during the season because successful wrestlers compete in 15 to 30 matches per year...Even when one to five hours are allowed for purposes of rehydration after the weigh-in, this time is insufficient for fluid and electrolyte homoeostasis to be completely re-established...Officials should realize that the singular effects of these practices are associated with 1) a reduction in muscular strength; 2) a decrease in work performance times; 3) lower plasma and blood volumes; 4) a reduction in cardiac functioning...6) an impairment of thermoregulatory processes; 7) a decrease in renal blood flow.... Therefore, it is the position of the American College of Sports Medicine that the potential health hazards created by the procedure used to make weight...can be eliminated if state and national organizations will...3) Discourage the practise of fluid deprivation and dehydration. This can be accomplished by: a) Educating the coaches and wrestlers on the physiological and medical complications that can occur as a result of these practices; b) Prohibiting the single or combined use of rubber suits, steam rooms, hot boxes, saunas, laxatives, and diuretics to "make weight"; c) Scheduling weigh-ins just prior to competition; d) Scheduling more official weigh-ins between...matches.
>
> (American College of Sports Medicine, 1976)

F.I.S.A., although not condemning the practise of dehydration in lightweight rowing competition, has acknowledged that dehydration may decrease performance and place a great deal of physiological stress on, in particular, the renal and thermoregulatory systems. The Australian Rowing Council, however, have issued guidelines early in 1988 on drugs in sport and advised that athletes testing positive for diuretic use would face a life ban from participation in competitive rowing. The ban on diuretics, however, is related to the fact that it is known that such drugs are used in an attempt to mask the presence of steroids (by decreasing their concentration in the urine) and does not appear, in any way, to reflect an attitude towards the possible deleterious effects of dehydration on health and exercise performance.

CHAPTER 2

REVIEW OF LITERATURE

2.1. INTRODUCTION

The volume of literature published over the last eighty years, related in one way or another to dehydration and athletic performance, is very large. Unfortunately, most of the work involving dehydration has been done in conjunction with a thermal stress and heat acclimatization, which has largely complicated the issue. Further, a lack of consistency in experimental design has made comparison between relevant studies difficult.

This review focuses on the following areas:

a) The effect of dehydration and rehydration on haematological parameters;
b) The effect of dehydration on the urinary excretion of potassium and sodium
c) The effect of dehydration and rehydration on exercise performance;
d) The effect of dehydration on metabolic, cardiovascular and respiratory parameters during exercise;
e) The effects of dehydration on muscle glycogen utilization during exercise.

2.2. THE EFFECT OF DEHYDRATION AND REHYDRATION ON HAEMATOLOGICAL PARAMETERS

It is most common to use haematological parameters as indices of dehydration. Because the blood and its components are so dynamic, results based upon blood tests are frequently subject to errors due to the following factors:

a) postural effects on plasma volume;
b) the assumption that plasma protein remains in the vascular space; and
c) the assumption that mean corpuscular volume remains constant despite changes in plasma osmolality.

The effect of dehydration on plasma volume along with potential errors in the measurement of plasma volume will be discussed in detail subsequently.

2.2.1. Dehydration and change in plasma volume

The most obvious effect of dehydration is a consequent decrease in plasma volume (Costill and Fink, 1974; Costill and Sparks; 1973; Gaebelein and Senay, 1982). Because the decrease in plasma volume has

been implicated in the decrease in efficiency of the cardiovascular and thermoregulatory systems observed during exercise (Nielsen, 1986; Mnatzakanian and Vaccaro, 1986), its measurement has been a basic inclusion in many dehydration methodologies. The results have varied enormously from one study to another. For example, in studies reporting an approximate 5% loss of bodyweight, the decrease in plasma volume has been reported to vary from 25% (Saltin, 1964c) to 6% (Sawka *et al.*, 1984a). For a 4% loss of body weight, plasma volume has been reported to decrease 16% (Costill and Fink, 1974) to only 5% (Sawka *et al.*, 1984b; Candas *et al.*, 1986). Even within the studies themselves, large individual differences have been regularly reported (Costill and Fink, 1974; Saltin, 1964c; Sawka *et al.*, 1984a). Four reasons become apparent and can account for the range of results, viz:

a) the weight loss methodologies vary widely;
b) blood sampling techniques are inconsistent;
c) unjustified assumptions are being made about vascular dynamics; and
d) calculations have been incorrect.

2.2.1.1. Methods of rapid weight loss

Dehydration regimens to lose weight have varied widely. The amount of weight loss reported has varied from 1-2 percent loss of body weight (Armstrong *et al.*, 1985a) up to a loss of 7-11 percent of bodyweight (Tipton and Tcheng, 1970). The regimens have also varied widely in the time frames in which the weight was lost. In the study by Torranin *et al.* (1979), the subjects were exposed to heat for two hours and experienced a bodyweight loss of 4-5%. On the other hand, Caldwell *et al.* (1984) induced this level of weight loss in 48 hours using exercise induced dehydration. Finally, the methods used to induce the weight loss vary greatly. All weight loss regimens studied, included one, some or all of the following procedures: thermal exposure, exercise, fluid restriction, starvation and the administration of a diuretic. Even within a single mode of weight loss, variation in the methodologies has been considerable. Thermal exposures, for example, have varied in the length of exposure, whether the exposures were continuous or intermittent, whether the subject was heat acclimatized and/or trained, the temperature and relative humidity that was used and whether the exposure was passive or involved exercise. The exercise also varied in intensity, modality, posture and duration. All of the other techniques, e.g. diuretics, also involve a huge spectrum of variation. Some methodologies that have been used to induce weight loss are presented in Table 2.1.

TABLE 2.1 Methods of rapid weight loss

Author	Method
Biljani *et al.*, 1980 Buskirk *et al.*, 1958 Caldwell *et al.*, 1984 Candas *et al.*, 1986 Costill, 1977 Nielsen *et al.*, 1981 Saltin, 1964b Saltin, 1964c	Heat exposure
Caldwell *et al.*, 1984 Costill and Saltin, 1973 Kirsch *et al.*, 1981 Nadel, 1980 Nielsen *et al.*, 1981 Saltin, 1964a Saltin, 1964b	Exercise
Costill and Fink, 1974 Costill *et al.*, 1976a Craig *et al.*, 1966 Klinzing and Karpowicz, 1986	Exercise with heat exposure
Armstrong *et al.*, 1985a Caldwell *et al.*, 1984 Claremont *et al.*, 1976 Nielsen *et al.*, 1981	Diuretics
Bell *et al.*, 1982 Houston *et al.*, 1981 Klinzing and Karpowicz, 1986 Tuttle, 1943 Widerman *et al.*, 1982	Food and water restriction alone or in combination with other methods

Each methodology that is listed in Table 2.1 can cause a different haemodynamic response (Caldwell *et al.*, 1984; Claremont *et al.*, 1976; Lamb and Brodowicz, 1986). In addition, the blood sampling technique and/or timing can result in a variation greater than the effect of the experimental protocol. This problem will be discussed in the following subsection.

2.2.1.2. Blood sampling technique and timing

A loss of 11-15% of plasma volume has been demonstrated to occur when moving from a supine position to a motionless upright position (Thompson *et al.*, 1928; Waterfield, 1931). This compares to the changes in intravascular volume of a magnitude similar to that produced by thermal stress and exercise. Hagen *et al.* (1978, 1980) stated that some of the published data relating to plasma volume changes with heat and exercise may be incorrect through a failure to account for effects of posture. In spite of this observation, except the studies of Fawcett and Wynn (1960) and Eisenburg (1963), the possibility of measurements of intravascular volume responses to heat and exercise being confounded by changes in posture has not been addressed, as pointed out by Sarelius (1979) and Harrison (1985).

If blood is taken from the antecubital vein of a pendent arm and compared with blood taken previously from the same arm in a horizontal position, the haematocrit is greater in the pendent position, as a result of blood pooling, by an amount equivalent to a 4% decrease in blood volume (Eisenberg, 1963). Further, it has been observed that if blood is sampled with the assistance of a torniquet (average length of time with stasis before sampling was five seconds) the apparent plasma volume decreases also by approximately 4% compared to the other arm, which was sampled simultaneously without stasis (C.M.Burge, unpublished observation).

A number of studies did not take into account the effects of acute exercise on plasma volume (Dill *et al.*, 1930; Myhre *et al.*, 1982). Most commonly, exercise results in haemoconcentration (Bock *et al.*, 1927; Edwards *et al.*, 1983; Greenleaf *et al.*, 1979b; Harrison, 1985), or more rarely haemodilution (Cullumbine and Koch, 1949; Senay, 1972). The extent of these changes is dependent upon the exercise modality, intensity and postural position adopted during the exercise (Kaltreider and Meneely, 1940; Senay *et al.*, 1980; Hagen *et al.*, 1978; Harrison 1985). On cessation of the exercise, plasma volume rapidly returns towards pre-exercise levels and these changes are completed within 60 minutes post-exercise (Greenleaf *et al.*, 1979b; Harrison *et al.*, 1975). Acute thermal exposure also produces changes in plasma volume. On cessation of the exposure, plasma volume returns towards pre-exposure levels over approximately the same time span or less than that required for plasma volume recovery after exercise (Harrison *et al.*, 1983). If blood is sampled during these transitory phases, the true equilibrated plasma volume status would not be measured.

In a review by Harrison (1985), reference is made to eight studies in which the results should be interpreted with care on the following grounds:

a) the effects of exercise and posture could not be distinguished (Dill *et al.*, 1930; Myhre *et al.*, 1982);
b) control blood samples were taken with subjects reclining (Costill and Fink, 1974) or seated (Costill *et al.*, 1976b; Wilkerson *et*

al., 1977) and then compared with samples taken during upright exercise; and

c) the body position in which the control blood sample was taken was not stated (Greenleaf *et al.*, 1978; Maron *et al.*, 1975).

Subsequent to the Harrison review, three further studies have been published about which similar reservations can be held. On similar grounds, Candas *et al.* (1986, 1988) did not state the body position in which the control blood sample was taken. Nose *et al.* (1988a) compared a control sample taken in a thermoneutral environment to a sample taken ten minutes post-exercise in a hot environment after the subjects had been relocated (it is not stated how they were transported and it may be they walked) back to the thermoneutral chamber.

2.2.1.3. The measurement of plasma volume and assumptions about vascular dynamics

The measurement of plasma volume, in itself, has aroused much scientific scrutiny and criticism. Much early work measured changes in plasma volume directly by dye and isotope dilution methods (Kozlowski and Saltin, 1964; Fortney *et al.*, 1981). Many studies tagged plasma protein, assuming that the protein did not leave the vascular space. Saltin (1964c) determined plasma volume with Evan's blue dye (T-1824), according to the method described by Von Porat (1951) and Wiklander (1956). Because it has been demonstrated that plasma protein leaves the vascular space (Edwards *et al.*, 1983; Landis and Pappenheimer,

1963), the results of any research using tagged plasma protein to determine changes in plasma volume are doubtful and should be interpreted with care.

Of greater relevance to the accompanying research, is the measurement of relative changes in plasma volume. The principle of measuring relative plasma volume change assumes that the red cell is unable to leave the vascular space and that there is no significant addition of red cells from storage sites such as the spleen in humans (Costill and Saltin, 1974a; Von Fricke, 1965; Nylin, 1947; Uehlinger and Böhlman, 1961). Two schools of thought exist with respect to its measurement (Dill and Costill, 1974; Van Beaumont *et al.*, 1972). There is a controversy whether haematocrit ratios alone can be used to measure relative changes in plasma volume (Van Beaumont, 1972) or whether haemoglobin concentration also must be taken into account (Dill and Costill, 1974).

Van Beaumont (1972), indicated that change in plasma volume could be calculated, using the assumption that red blood cell volume remains constant, by change in haematocrit ratios (Hct) as follows:

$$\% \text{ change PV} = \frac{100}{100 - Hct_{pre}} \times 100 \left[\frac{(Hct_{pre} - Hct_{post})}{Hct_{post}} \right]$$

The assumption that red cell volume is stable even with changes in plasma osmolality has been criticized (Harrison, 1985), although Van Beaumont and colleagues have defended their stance in several studies (Van Beaumont *et al.*, 1972; Van Beaumont, 1973; Van Beaumont, 1974; Greenleaf *et al.*, 1979a; Greenleaf *et al.*, 1979b; Van Beaumont *et al.*, 1981).

Costill and Saltin (1974a) raised questions about Van Beaumont's three major assumptions used in the calculation of changes in plasma volume: i.e.;

a) that the total volume of circulating erythrocytes remained constant;
b) that the size of individual erythrocytes did not change; and
c) that the ratio between venous haematocrit and whole body haematocrit (F-cell ratio) remains unchanged with dehydration.

The research of Costill and Saltin (1974a) showed that both points (a) and (c) remained true after a 4% loss of body weight. A discrepancy between measured (^{125}I) and calculated changes in plasma volume, however, suggested that shrinkage of red blood cells may have occurred leading to an under-estimation of the venous haematocrit and subsequent calculation of the percent change in plasma volume (PV). Dill and Costill (1974) and Costill and Fink (1974) later confirmed that red cell shrinkage following dehydration did indeed occur.

To correct for changes in red blood cell volume, the ratio of haemoglobin (Hb) pre- to post-treatment should be included in the calculation in addition to the haematocrit ratios (Elkinton *et al.*, 1946; Dill and Costill, 1974) as follows:

$$\% \text{ change PV} = 100 \times \left[\frac{Hb_{pre}\ (100 - Hct_{post})}{Hb_{post}(100 - Hct_{pre})} \right] - 100$$

(Dill and Costill, 1974)

Many authors have opted for the Dill and Costill equation for calculation of relative changes in plasma volume in their studies (Collins *et al.*, 1986; Costill *et al.*, 1981; Fortney *et al.*, 1981; Nadel *et al.*, 1980; Pivarnic *et al.*, 1986; Pivarnic and Senay Jr., 1986; Pivarnik *et al.*, 1988).

2.2.1.4. Corrections and mathematical errors in the calculation of relative plasma volume

Haematocrit ratios are often multiplied by one and sometimes two correction factors. The correction factors most commonly used correct for trapped plasma and for the fact that the haematocrit averaged over the whole body is less than the haematocrit of venous blood (F-cell ratio). Harrison *et al.*, (1982) performed a mathematical appraisal of the effect of using or not using the correction factors in relation

to the calculation of relative plasma volume using the equation of Dill and Costill (1974).

The first correction factor considered was the effect of correcting for trapped plasma, the usual value being 0.96. The use of this correction factor had a negligible effect on percent change of plasma volume. Further, the error introduced by not correcting was much less than the random variability caused by measurement errors in haematocrit and haemoglobin (Harrison *et al.*, 1982). In addition, the correction factor using the microhaematocrit method also may be nearer to 0.98 (Garby and Vuille, 1961) than the 0.96 conventionally adopted. Therefore, because the error arising from the neglect of the effects of trapped plasma was so small compared to the random measurement error, there appears little to be gained by correcting the values of haematocrit for trapped plasma.

The second correction factor considered by Harrison *et al.* (1982) is the F-cell ratio (Gibson *et al.*, 1946). Over 80% of directly determined values for F are in the range 0.85-0.99, and a value of 0.91 (Chaplin *et al.*, 1953; Green *et al.*, 1987; Gregerson and Rawson, 1959) or 0.92 (Albert *et al.*, 1965) is generally adopted. It should be noted, however, that Harrison and co-workers consistently take an F-cell value of unity (Harrison, 1974; Harrison *et al.*, 1975, 1981). Harrison *et al.* (1982) determined that the errors introduced by failing to correct the measured haematocrit for an assumed value for the F-cell ratio are small, and are negligible compared with random measurement errors. In addition and also fortunately, Costill

and Saltin (1974a) found that the F-cell ratio also does not significantly change following exercise dehydration leading to a 4% loss of bodyweight. This observation lends validity to the Dill and Costill (1974) equation, which relies on the assumption that no change in the F-cell ratio occurs pre- and post-dehydration.

Inconsistencies in the reported measurement of changes in plasma volume can be attributed also to mathematical errors. For example, as Harrison in his review (1985) has indicated, some confusion has arisen because of multiplying changes in haematocrit ratio by 100 to change it to a percentage. For example, if the haematocrit changes from 40% to 50%, the magnitude of the change is 25%; not deceptively 10%. Harrison (1985) cited several reports where this type of interpretive error has occurred, e.g. Pugh (1969) and Saltin (1964c). Other studies (cited in Harrison, 1985) quoting relative changes in plasma volume did not convert the percent change in haematocrit:

$$100(Hct_1-Hct_2)/Hct_2)$$

to percent change in plasma volume by multiplying it by:-

$$100/(100-Hct_1)$$

e.g. Bazett *et al.* (1940), Bock *et al.* (1927), Gregerson and Rawson (1959), Joye and Poortmans (1970), Poortmans (1971), Senay Jr. (1970), Senay Jr. and Christensen (1965, 1968) and Stein *et al.* (1949).

2.2.2. The effect of rehydration on the restoration of plasma volume

Lightweight rowers who have dehydrated to qualify for the weight category usually have a period of approximately 90 minutes in which to rehydrate. Wrestlers may have up to 5 hours to do so. The popularity of wrestling in North American and Canadian universities, has led to a significant amount of research on the problems associated with the rehydration of wrestlers who have dehydrated to make weight categories. Attempts to study the rehydration of rowers in conditions related to their specific environment have not been previously published.

A variety of techniques has been used to achieve rehydration making comparison between studies difficult. For example, rehydration regimens have been structured, with fixed amounts of fluid being consumed at regular time intervals (Costill and Sparks, 1973; Costill and Saltin, 1974b; Torranin *et al.*, 1979) or *ad libitum*, where the subject simply drinks to satiety (Klinzing and Karpowicz, 1986). On occasions the subject has been permitted to eat during the rehydration period (Houston *et al.*, 1981). There has been also considerable variation in the osmolality and the energy and electrolyte content of the rehydration fluids used. Lamb and Brodowicz (1986) and Murray (1987) have reviewed in depth the various drink formulations used to achieve rapid rehydration after dehydration.

Previous discussion has indicated that plasma volume significantly decreases following dehydration. The decreased plasma volume has been

implicated in the reduced physical working capacity observed following dehydration (Candas *et al.*, 1986; Saltin, 1964b). Rehydration research, therefore, has usually concentrated on methods of returning plasma volume back to euhydration levels (Lamb and Brodowicz, 1986). In the main, studies have shown that a few hours of rehydration are sufficient to return most physiological parameters to near normal values at rest, except plasma volume (Lamb and Brodowicz, 1986).

2.2.2.1. Rehydrating with water versus dilute solutions of sodium chloride.

Nielsen (1984a) has pointed out that the rate of restoration of the fluid lost is limited by the rate of gastric emptying, the rate of absorption of fluid from the intestinal lumen and the equilibrium processes between the body water compartments. In the case of *ad libitum* rehydration regimens, the loss of the dipsogenic drive occurs long before euhydration status is achieved (Nielsen *et al.*, 1984a; Nose *et al.*, 1988b; Stricker and Jalowiec, 1970) partly because stomach distension has been associated with the early termination of drinking (Geelen *et al.*, 1984; Rolls *et al.*, 1980; Seckle *et al.*, 1986). Further, when the osmolality and/or chloride levels of the vascular compartment become normalized, even if the volume has not returned to normal, the thirst sensation usually ceases (Dill *et al.*, 1933). If serum osmolality and serum sodium levels decrease below euhydrated levels, there is also a rapid and large increase in

urine production mediated via osmoreceptor suppression of vasopressin release (Brandenburger *et al.*, 1986; Robertson, 1974). This results in excretion of water via the kidneys before the water loss is fully replaced. In relation to the above discussion, the rapid consumption of large volumes of water usually results in a decrease in serum osmolality and a resultant diuresis before plasma volume is normalized.

The ideal rehydration fluid would contain sufficient quantities of electrolyte such that when all the fluid that has been lost is replaced, serum electrolytes and osmolality would be normal. In the exercise context, however, other factors have to be considered, viz:

a) The relative importance of rehydrating the extracellular fluid compartment compared to the intracellular compartment on maximal performance has not been studied. On the one hand, fluids containing significant levels of sodium have been demonstrated to rehydrate predominantly the extracellular compartment (Nielsen *et al.*, 1986). Ingestion of a high potassium fluid, on the other hand, in addition to preferentially rehydrating the intracellular space, appeared to enhance urine production and natriuresis (Nielsen *et al.*, 1986). There is no evidence, however, that the inclusion of potassium in the high potassium drink specifically resulted in intracellular rehydration as a drink high in carbohydrate but low in potassium was equally effective in this regard.

b) To replace the lost plasma volume completely and prevent the hypo-osmotic induced diuresis from occurring, a solution of 0.45% sodium chloride, or greater, is required to be drunk (Nose *et al.*, 1986). The large volumes required to be drunk could be expected to induce feelings of fullness and nausea mainly because of retarded gastric emptying (Hunt, 1959). Competitors, for this reason, would not voluntarily choose to drink such concentrated solutions before competing in a race or similar event. A 0.3% solution appears to represent the limits of voluntary toleration and allaesthesia for large volumes in such a situation (personal observation).

c) Gastric emptying is significantly slowed in the presence of solutions with high osmotic pressures (Costill and Saltin, 1974; Fordtran and Saltin, 1967; Hunt and Pothak, 1960). However, gastric emptying (Hunt and Pothak, 1960) and intestinal absorption (Schultz, 1981) is facilitated if a small amount of sodium chloride is present compared to that of distilled water. This possibly stimulates the active uptake of sodium that would carry water with it.

Davis *et al.* (1987), using deuterium (D_2O) labelled beverages, demonstrated that the accumulation of D_2O in the plasma was significantly greater after ingestion of a hypotonic saline drink compared to water after 90 minutes. In many studies, carbohydrate in various concentrations has also been added to the rehydration medium, making comparisons difficult (Allen *et al.*, 1977; Corrigan *et al.*, 1984; Costill and Sparks, 1973; Torranin *et al.*, 1979). In

their study, Costill and Sparks (1973) used a rehydration fluid that consisted either of a glucose-electrolyte drink or distilled water. The subjects were required to drink an amount calculated to be the equivalent of 7.7% of the lost body weight every fifteen minutes such that after three hours of rehydration, their lost body weight would be regained. Bodyweight, however, is not a reliable indicator of fluid status after rehydration because a significant proportion of the fluid will have remained in the gut or been passed into the bladder (Costill and Sparks, 1973). Therefore, it is not a surprising result that although neither solution completely restored plasma volume after three hours, the glucose-electrolyte drink was significantly more effective. Mack *et al.* (1986) obtained similar results using a hypotonic NaCl solution. Davis *et al.* (1987) found that there was very little difference in the accumulation of D_2O in the plasma of a hypotonic electrolyte solution that also contained 6% glucose compared to the hypotonic saline solution alone. Solutions containing greater than 6% glucose, however, significantly retarded gastric emptying. Costill and Saltin (1974b), also reported that gastric emptying rate decreased from 17 ml min^{-1} for a 0.2% (34 mmol l^{-1}) saline solution to 10 ml min^{-1} following the addition of 5.0% glucose.

2.3. THE EFFECT OF DEHYDRATION ON THE URINARY EXCRETION OF SODIUM AND POTASSIUM

In dehydration, the rate of sodium reabsorption is increased, despite raised plasma osmolality, in order to minimize the urinary loss of water (Gauer *et al.*, 1970; Nielsen *et al.*, 1984a). Aldosterone acts on the distal convoluted tubule to promote the active reabsorption of sodium in exchange for potassium (Hierholzer and Weiderholt, 1976). An increased potassium excretion after dehydration, therefore, should be evident. Observations to this effect have been made by Costill *et al.* (1976b), Mnatzakanian and Vaccaro (1984) and Zambraski *et al.* (1975).

2.4. THE EFFECT OF DEHYDRATION AND REHYDRATION ON PERFORMANCE

A substantial volume of literature exists reporting the effects of dehydration and rehydration on performance. Unfortunately, as previously discussed, meaningful comparisons between studies have been made very difficult because of different degrees of dehydration, methods and time periods in which the dehydration was achieved and the method of rehydration attempted. Finally, to add further to the plethora of methods, the tasks chosen in which to quantify changes in performance after dehydration have also varied.

2.4.1. Physical work capacity after dehydration

A representative but not inclusive list of studies demonstrating the deleterious effect of dehydration on physical working capacity (PWC) is shown in Table 2.2. Rowing involves a combination of power and endurance. Elite rowers are characterized by their very high maximal oxygen uptakes and strength development and their capacity to tolerate very high levels of acidosis (Hagerman *et al.*, 1979). To formulate a hypothesis on the effect of dehydration on rowing performance, therefore, it is necessary to consider the effects of dehydration individually on:

a) endurance capacity;

b) anaerobic capacity; and

c) strength and muscular endurance.

TABLE 2.2 List of studies demonstrating the deleterious effect of dehydration on physical working capacity

Author	Method of Dehyd.	Period of Dehyd.	Degree of Dehyd.	Performance Modality	Result
Saltin 1964a	Exerc 70% VO_2max Thermal Thermal/Exerc 56% VO_2max	2.4-4 hrs	2.3-5.6% 2.7-5.6% 2.7-6.6%	Cycle ergo	Submax ex heart rate increased Max PWC decreased
Craig et al. 1966	46 C Reclining/ Walking	5-6 hr	4.3%	Brisk walking to exhaustion	48% dec in time to exhaustion
Herbert et al. 1972	Ad.Lib. Loss	4 days	4.8%	PWC-170 test cycle ergo	PWC decreased
Costill et al. 1973	a) Heavy Exercise b) Thermal	a) 2-2.5 hr b) 3-3.5 hr	a) 4.0% b) 4.0%	Cycle ergo 80% VO_2max for 5 min	a) PWC decreased b) PWC decreased
Palmer 1969	Thermal	Rapid as possible	4.8%	PWC-160 test	Submax ex heart rate increased
Ribisl et al. 1970	Ad. Lib.	48 hr	5.0%	PWC-170 test	PWC decreased
Torranin et al. 1979	Thermal	2 hr	4.0%	Muscular Endur a) Isometric b) Isotonic	 31% decrease 29% decrease
Caldwell et al. 1984	a) Thermal b) Diuretic c) Exercise	a) 24 hr b) 24 hr c) 48 hr	a) 4.1% b) 4.1% c) 4.1%	Incremental VO_2max test	max PWC decreased all trials
Nielsen et al. 1981	a) Exercise-50% VO_2 cycle ergo b) Thermal c) Diuretic	a) 90-160 mins b) - c) -	a) 2.5% b) 2.5% c) 2.5%	Bike ergo incremental exercise test of 50% (of VO_2max)-8 mins 70%-5 mins 105% to exhaustion	 a) 44% dec PWC b) 35% dec PWC c) 18% dec PWC
Armstrong et al. 1985	Diuretic	5 hr	1.9-2.1%	Track running	5,000m and 10,000m running times greater 1,500m not affected
Klinzing and Karpowicz, 1986	Ad lib.	50 hr	5.0%	Wrestling skills test	Wrestling performance decrease

2.4.1.1. The effect of dehydration on endurance performance

The consensus of previous investigations indicates that dehydration causes a substantial decrease in maximal physical work capacity in endurance type exercise. Armstrong *et al.* (1985a) induced a weight loss of between 1.9% and 2.1% in five hours following the administration of 40 mg of the diuretic furosemide. In the dehydrated state, subjects achieved less work during an incremental $\dot{V}O_2max$ test on a treadmill. Using similar methods, Caldwell *et al.* (1984) also observed comparable results. Of greater practical significance, Armstrong *et al.* (1985a) demonstrated that competitive track running performance was detrimentally affected over distances of 5,000 metres and 10,000 metres. Their linear regression analysis indicated that each 1% decrease in body weight, following diuretic use, corresponded to running time increases of 0.39 (r= -0.65) and 1.57 (r= -0.40) minutes respectively for the 5,000 metres and 10,000 metres track running trials (Armstrong *et al.*, 1985a).

Some studies specifically report that exercise induced dehydration causes the greatest deleterious effect on physical work capacity compared to other forms of dehydration (Costill and Saltin 1973, 1975; Nielsen *et al.*, 1981). The subjects in these studies had induced the dehydration by exercising submaximally for a number of hours and then, within 30-90 minutes after the cessation of this exercise, performed a strenuous exercise test. Physical work capacity in these tests, as expected, decreased significantly. However, how much of

performance decrement in these experiments can be attributed to the effects of dehydration? Asmussen *et al.* (1974) observed that in euhydrated subjects, maximum physical work capacity was detrimentally affected when a strenuous exercise test was preceded by several hours of submaximal exercise. This kind of prior work could have depleted the muscle glycogen stores (Klausen *et al.*, 1975). This point will be discussed in greater detail later (section 2.6).

As Caldwell *et al.* (1984) identified, only a few dehydration studies used methods that are actually used by athletes to compare differences in dehydration protocols. For example, the methods used by Costill and Saltin (1975), of exercise induced dehydration, would be very unlikely to be adopted as a technique for weight loss by athletes immediately prior to competition. The majority of weight is usually lost a night or so before the day of the event using light exercise and fluid restriction. Caldwell *et al.* (1984), using such a regimen over a 48 hour period, demonstrated that this form of exercise-dehydration was significantly less detrimental to performance than the more rapid thermal or diuretic induced forms of dehydration. It is of interest that Caldwell *et al.* (1984) reported that there were no significant changes in muscle glycogen content within any of the test groups before and after dehydration.

A common criticism of dehydration research is that the decreased physical work capacity may be due to psychological factors. Saltin, performing as a subject in his study (1964b), worked as hard as he could and is convinced that the other subjects also did their best.

Similarly, Armstrong *et al.* (1985a), reported that the track trials were performed under competitive conditions with very highly motivated individuals. As proposed by Saltin (1964b), the increased performance times seem to imply a decreased physiological ability to perform hard muscular work.

2.4.1.2. The effect of dehydration on anaerobic performance

Jacobs (1981) observed that after thermal dehydration of 5% of body weight, no deleterious effects on the ability to perform the Wingate anaerobic test nor change in peak lactate accumulation were evident (Jacobs, 1980). Bell *et al.* (1982) similarly found that the ability to perform maximal anaerobic work was not affected by dehydration.

2.4.1.3. The effect of dehydration on strength and muscular endurance

The assessment of the effect of dehydration upon muscular strength and endurance has not been made in specific sporting environments. Most studies have assessed the function of isolated muscle groups using isometric and isotonic tasks with equivocal results (Serfass *et al.*, 1984; Torranin *et al.*, 1979; Bosco *et al.*, 1974).

Some studies have examined the effects of dehydration on the hand flexor (hand gripping) muscles. Serfass *et al.* (1984) induced a 5%

loss of body weight in trained wrestlers over a three day period. It was not stated how the weight loss was achieved. The standard exercise task required maximal contractions of the hand flexor muscles to be performed at a rate of thirty contractions per minute for six minutes using a hand dynamonometer. They did not identify any deleterious effects from dehydration on initial strength, final strength or force/time integrals. Similarly, Saltin (1964a), Singer and Weiss (1968) and Tuttle (1943) also did not report any decrements in hand grip strength of wrestlers following losses of up to 5% of bodyweight. In contrast to the Serfass *et al.* (1984) study, Torranin *et al.* (1979) reported a significant decrease in muscular endurance of the hand flexor muscles following dehydration. In this study, a 4% loss of body weight was achieved by inactive thermal exposure for three hours. The standard exercise task required regular contractions of 75% of a previously determined maximal voluntary contraction (75% 1-MVC) to be performed at a rate of thirty contractions per minute, again on a hand dynamonometer. The test ceased when 75% 1-MVC failed to be achieved on two consecutive contractions.

Torranin *et al.* (1979) also reported the effects of dehydration on isometric and isotonic muscular endurance in various other muscle groups. For the isotonic measurements, arm curls (one arm), bench press and leg press were performed. The standard exercise test, as previously mentioned for the hand grip test, required contractions of 75% 1-MVC at a rate of thirty contractions per minute. The standard isometric exercise test required a sustained contraction of 75% 1-MVC to be maintained for as long as possible. When the results of all four muscle

groups were averaged, dehydration was associated with a 31% decrease in isotonic endurance time and a 29% decrease in isometric endurance time. Their results also suggested that different muscle groups may be affected differently in dehydration. During isometric work, performance during the leg press and hand grip exercises appeared to be affected significantly whereas the arm curl and bench press exercises were relatively unaffected. During isotonic work, performance similarly decreased for the leg press and hand grip exercises. The isotonic arm curl and bench press exercises were left relatively unaffected. The major muscle groups involved in hip and leg extension are also required in rowing. It is of interest that the isotonic leg press exercise, as used in the Torranin *et al.* (1979) study, was affected by dehydration to a greater degree than the musculature associated with the arm and pectoral girdle. Bosco *et al.* (1974) also reported that isotonic muscular endurance decreased following dehydration. After a 5.7% loss of bodyweight induced over three days, the number of sit-ups able to be performed in two minutes decreased by 9% (Bosco *et al.*, 1974).

2.4.2. Physical work capacity after rehydration

Many physiological variables, important for endurance exercise and that are adversely affected by dehydration, will return towards normal after rehydration, albeit not completely in the limited time before competition (Allen *et al.*, 1977; Costill and Sparks, 1973; Herbert and Ribsl; 1972; Sproles *et al.*, 1976). Hence physical work

capacity, although improved compared to a dehydrated trial, usually remains substantially below that obtained when euhydrated (Corrigan *et al.*, 1984; Houston *et al.*, 1981). After four hours of rehydration, Torranin *et al.* (1979) found that muscular endurance was still depressed after an initial 4% loss of bodyweight. A study by Houston *et al.* (1981) showed that three hours of refeeding and rehydration with self-selected foods and beverages after 8% loss of bodyweight induced by dehydration and fasting did not return isokinetic knee extension strength to normal. Finally, another study (Corrigan *et al.*, 1984) compared different beverages for rehydration by using various combinations of electrolytes and either glucose or glucose polymer. The authors found that performance in a 2-3 minute simultaneous arm and leg cycling task of maximal intensity was not restored to normal after five hours of rehydration with any of the beverages.

2.5. THE EFFECT OF DEHYDRATION ON RESPIRATORY, METABOLIC AND CARDIOVASCULAR VARIABLES

A reasonable consensus between studies has been obtained with respect to the effect of dehydration on various metabolic and respiratory variables. Maximum oxygen uptake is either unchanged (Armstrong *et al.*, 1985a; Bock *et al.*, 1967; Houston *et al.*, 1981; Saltin 1964a,c) or decreases slightly (Caldwell *et al.*, 1984; Sawka *et al.*, 1984b). Studies that report the effect of dehydration on the respiratory exchange ratio and peak ventilation rate during high

intensity exercise of six to seven minutes duration have not been able to be located. During submaximal exercise ranging from 50 to 85 percent of maximal oxygen uptake, a decrease in RER has been reported (Costill *et al.*, 1976a; Saltin, 1964a, 1964b).

Dehydrated subjects commonly experience a decrease in blood lactate accumulation after maximal exercise of several minutes duration when compared to a similar euhydrated trial (Nielsen *et al.*, 1981; Caldwell *et al.*, 1984; Klinzing and Karpowicz, 1986; Armstrong *et al.*, 1985a). In a study by Saltin (1964c), thermally dehydrated subjects complained regularly that the legs were the "limiting factors" at the maximal work load suggesting a high lactate accumulation. This observation, however, was not reflected in the measured blood lactate accumulation.

At maximal work intensity, there is no change in maximum heartrate after dehydration (Armstrong *et al.*, 1985a; Caldwell *et al.*, 1984; Klinzing and Karpowicz, 1986). If the dehydration induced loss of plasma volume is less than 6%, vasoconstriction in peripheral beds can usually compensate for the reduced blood volume and assist in maintaining stroke volume and cardiac output (Nielsen, 1984b; Claremont *et al.*, 1976; Klinzing and Karpowicz, 1986; Sproles *et al.*, 1976). End diastolic volume is usually reduced (Sawka *et al.*, 1984a) because of decreased filling pressure (Hales, 1986). Presumably stroke volume is maintained as a result of increased sympathetic tone.

Following substantial losses of sweat, the relative increase in the circulating volume of red blood cells is associated with higher blood viscosities (Vandewalle *et al.*, 1988; Mnatzakanian and Vaccaro, 1984). Increases in blood viscosity has been associated with a decrease in cardiac efficiency due to an increased resistance to blood flow (Fowler and Holmes 1975; Gorden *et al.*, 1974). It is of interest that subjects who maximally exercise with artificially increased blood viscosities appear to experience fatigue earlier compared to a similar trial with unadjusted blood viscosities (Dintenfass and Lake, 1977). Letcher *et al.*, (1981) and Ernst (1985), also found that the resting blood viscosity of trained subjects was lower than that of untrained subjects. This difference in blood viscosity was due to a lower fibrinogen concentration in the trained subjects. This may help explain the observation that trained subjects appear to tolerate dehydration better than untrained subjects (Saltin, 1964a; Buskirk *et al.*, 1958). Whether changes in blood viscosity are significantly associated with performance decrements in dehydration remains to be elucidated.

2.6. THE EFFECT OF DEHYDRATION ON MUSCLE GLYCOGEN LEVELS AND UTILIZATION RATE DURING EXERCISE

Muscle glycogen content following dehydration has been measured only on a relatively small number of occasions (Caldwell *et al.*, 1984; Costill and Saltin, 1975; Costill *et al.*, 1976a; Costill *et al.*, 1981; Houston *et al.*, 1981; Nielsen *et al.*, 1986). In these studies, except the studies of Caldwell *et al.* (1984) and Houston *et al.* (1981), dehydration was induced using submaximal exercise for a number of hours prior to an exercise performance test. As mentioned previously (section 2.4.1.1), Asmussan *et al.* (1974) observed that work capacity in euhydrated subjects was reduced when preceded by several hours of submaximal exercise.

In the study by Costill and Saltin (1975), a 4% loss of bodyweight was achieved by continuous running for 1.5 to 2.5 hours. Unfortunately the intensity of the submaximal exercise was not reported. Prolonged exercise of between 60% and 74% of $\dot{V}O_2max$ for two hours has been demonstrated to deplete completely the glycogen content of the slow twitch (ST) muscle fibres and substantially deplete the fast twitch (FT_a) fibres but having a much lesser effect on the FT_b fibres (Gollnick *et al.*, 1973a, 1974b). Thirty minutes after completion of the prolonged submaximal exercise, the dehydrated exercise performance test, involving 5 minutes of bicycle ergometer activity at 80 - 85% $\dot{V}O_2max$ was performed. The exercise test probably would have predominantly recruited the FT_a and ST fibres also (Gollnick *et*

al., 1973b, 1974b). Hence, the decrease in performance reported in the study of Costill and Saltin (1975) cannot be attributed fully to the effect of dehydration alone but also to a prior depletion of glycogen in the muscle fibres predominantly recruited during the exercise test (i.e., ST and FT_a fibres).

Houston *et al.* (1981) studied a group of wrestlers who were required initially to restrict food intake for 48 hours. Muscle glycogen levels decreased significantly from an initial level of 62.3 ± 3.3 mmol glucosyl units kg^{-1} wet weight of tissue (mmol kg^{-1} W.W.) to 44.0 ± 3.0 mmol kg^{-1} W.W. For a further 24 hour period, food intake continued to be restricted in addition to fluid intake also being decreased by two thirds. A final additional 24 hour period involved an almost complete restriction of food intake and no fluid intake at all. During the final 48 hour period, muscle glycogen decreased significantly from 44.0 ± 3.0 mmol kg^{-1} W.W. to 33.9 ± 1.8 mmol kg^{-1} W.W. Light exercise training had been continued during the 96 hour period and most probably contributed significantly to the decrease in muscle glycogen levels. After a further three hour period of rehydration and consumption of food containing mostly simple carbohydrate, muscle glycogen levels did not increase significantly and remained at 38.6 mmol kg^{-1} W.W. In this study, the decreases in muscle glycogen probably could be attributed to the effects of 96 hours of food restriction. The failure to synthesize muscle glycogen in the three hour period of rehydration, however, may be attributed possibly to muscle trauma (Costill *et al.*, 1988).

Costill *et al.* (1988) demonstrated that muscle glycogen storage was affected by repeated biopsies from the same muscle. They found that samples taken from regions distal and proximal to the initial biopsy contained, on average, less glycogen than the contralateral control leg. These differences, however, were only significant in the distal muscle sample. Alterations in muscle glycogen storage were seen to persist for ten days after the first biopsy. They concluded that in studies requiring repeated muscle biopsies over several days, consideration should be given to the effects on impaired glycogen storage from biopsy trauma. The problem can be minimized by sampling muscle distal (approximately 3 cm) to the site of the previous biopsy, although some reduction in glycogen storage could be expected in these specimens (Costill *et al.*, 1988). This information may explain why Houston *et al.* (1981) observed no glycogen synthesis during rehydration because all biopsy samples had been obtained from the same muscle.

Caldwell *et al.* (1984) compared three methods of rapid weight loss on the muscle glycogen levels of wrestlers and weight lifters. Following diuretic or thermally induced dehydration over a 24 hour period, muscle glycogen levels were not significantly affected. It should be noted, however, that performance was significantly affected after these treatments. The third treatment required an increase in training volume over a 48 hour period which Caldwell and colleagues

termed exercise-dehydration. It is not made clear, however, whether food and/or fluid was restricted during this period. Muscle glycogen following the exercise-dehydration treatment did not decrease significantly. It was of interest that lactate accumulation during the incremental $\dot{V}O_2$max tests was lower following all dehydration treatment conditions despite the unchanged initial muscle glycogen levels.

One study provided evidence that dehydration may decrease muscle glycogen utilization rate during exercise (Costill and Saltin, 1975). These authors compared the effects of dehydration induced by prolonged submaximal exercise or inactive thermal exposure on the distribution of water and electrolytes between the internal and external phases of skeletal muscle. Muscle glycogen levels were also measured. Their research design involved completing a five minute control cycling task at 80-85% $\dot{V}O_2$max, which was then shortly followed by dehydration. Dehydration (either thermal or exercise) proceeded until a 4% loss of body weight had been achieved. After weight loss and thirty minutes rest, the five minute exercise performance test was again performed. Unfortunately, Costill and Saltin did not report absolute muscle glycogen concentration, but reported relative changes. The changes in glycogen concentration following each trial have been estimated from a figure provided in their original report and are presented in Table 2.3.

TABLE 2.3 Estimated changes in muscle glycogen content (mmol kg^{-1} D.W.) from graph presented in Costill and Saltin (1975).

	Thermal Group	Exercise Group
Control trial	-118	-91
During dehydration	+82	-55
Dehydrated trial	-82	-46

It is unexplained why the subjects in the exercise dehydration group utilized less glycogen in the control trial than the thermal group. The finding that muscle glycogen concentration increased during the thermal exposure is also unexplained as the glycogen content is reported as dry weight and therefore should not be influenced by changes in muscle water content. As expected, muscle glycogen substantially decreased during the prolonged submaximal exercise. The precipitous decrease in glycogen utilization during the dehydrated exercise trial compared to the control trial in the exercise dehydration group could have been as a result of complete depletion of the ST fibres and partial depletion of the FT_a fibres, as discussed previously. In addition, if at the beginning of the dehydrated trial, muscle glycogen levels were less than 160 mmol kg^{-1} D.W., then exercise performance and lactate production would be considerably impaired, as demonstrated by Jacobs (1981). In the preceding control trial and during dehydration, the exercise group suffered a decrement in glycogen of 106 mmol kg^{-1} D.W.. Hence,

unless they had a starting level of 306 mmol kg^{-1} D.W., they would have fallen below this threshold level at the commencement of the dehydrated trial. This could account for the decrease in glycogen utilization rate in the exercise-dehydration group. Nevertheless, glycogen utilization by the thermal group also decreased for the dehydrated exercise trial. Unfortunately, simultaneous euhydrated controls were not run to observe whether any residual effects from the first exercise bout may have influenced glycogen utilization pattens in the second exercise bout or whether the decreased glycogen utilization could be attributed to dehydration effects.

CHAPTER 3

METHODS AND PROCEDURES

3.1. INTRODUCTION

This study has two major objectives:

a) To observe the effects of 24 hours of dehydration on body weight, plasma volume and various urinary variables and to observe the efficacy of drinking water and solutions of varying sodium chloride concentration in restoring plasma volume after dehydration;

b) To examine the metabolic processes during performance in a maximal rowing trial following partial rehydration with water after rapid weight loss.

The rationale for the 24 hours of dehydration and the 90 minute rehydration period was to create an experimental design that closely resembled the actual regimen of dehydration used by the lightweight rower. It has been shown previously that the inclusion of sodium chloride in the rehydration fluid may help in a more rapid restoration of plasma volume compared to that of water (Hunt and Pathak, 1960). Four solutions, plain water, 0.1%, 0.2% and 0.3% w/v saline solution, were selected to study the effect of varying sodium chloride

concentration in restoring plasma volume. This protocol leaves scope to compare the effect of rehydration with each various test solution on maximal rowing performance. In this thesis rowing performance following rehydration with water only is presented.

This study was approved by the Footscray Institute of Technology Human Experimentation Ethics Committee. All subjects were fully informed of any risks associated with the procedures. All subjects were required to sign an informed consent statement and complete a health risk questionnaire before commencing the trials (see Appendix A).

3.2. THE EFFECT OF 24 HOURS OF DEHYDRATION ON BODY WEIGHT, PLASMA VOLUME AND VARIOUS URINARY VARIABLES

3.2.1. The Subjects

Individual subject data for each drink group are presented in Table 3.1.

TABLE 3.1 Individual data for each drink group

GROUP subject #	BODYWEIGHT Pre-Dehy (Kg)	Post-Dehy (Kg)	% diff (%)	Sum Skinfolds (mm)	Height (cm)	Age (yr)
0.1%						
1	61.02	59.64	2.27	44.1	173	22
2	83.30	80.01	3.95	60.3	185	20
3	60.20	57.50	4.49	66.5	165	25
4	65.75	63.25	3.80	106.6	166	18
5	74.62	70.92	4.96	64.3	182	24
6	66.00	63.98	3.06	55.7	168	18
0.2%						
7	58.93	57.10	3.10	100.9	168	20
8	85.92	83.36	2.97	68.8	185	20
9	70.09	67.95	3.05	114.9	172	32
10	70.74	68.27	3.50	55.7	174	22
11	72.81	69.91	3.98	71.1	179	21
12	84.25	80.78	4.12	67.0	184	23
0.3%						
13	66.41	63.42	4.43	65.2	172	26
14	60.39	57.60	4.63	66.5	167	25
15	67.87	66.12	2.58	65.8	178	19
16	80.47	77.45	3.75	101.2	185	20
17	75.14	71.92	4.28	69.4	176	22
18	72.56	69.58	4.12	68.0	179	22
ROWERS						
1	70.99	67.41	5.04	45.0	185	21
2	71.38	67.22	5.86	59.0	186	21
3	72.31	68.58	5.16	56.7	185	24
4	75.21	71.43	5.02	75.2	181	20
5	76.43	72.45	5.21	55.7	189	19
6	67.49	64.55	4.36	64.1	172	21
7	73.02	69.04	5.45	107.3	175	20
8	80.68	76.52	5.16	86.0	182	25

3.2.2. Pre-dehydration

Eighteen healthy active male subjects and eight highly trained elite oarsmen voluntarily consented to take part in this experiment. Anthropometric data from each subject is shown in Table 3.1. The subjects were weighed nude in the morning preceding dehydration (euhydration weight). Body weight was determined using a Sauter electronic scale accurate to 0.005 kg. This initial weigh-in was conducted five hours post-prandially. No strenuous exercise was permitted within 24 hours before to this weight measurement. All subjects were asked to void their bladders and empty the bowels (if possible) before this and subsequent weight measurements. Following the weight measurement, a 2 ml blood sample without stasis was drawn from a vein in the antecubital space, with the arm extended, after the subject had been supine for fifteen minutes (this procedure was followed for all subsequent blood samples that were to be used for the analysis of changes in plasma volume). Posture-induced changes in blood volume occur rapidly and are usually complete within 15-20 minutes (Hagen et al., 1978). Finally, adiposity levels were estimated by measuring and summing together the skinfold thickness at eight standard sites (triceps, biceps, suprailiac, abdominal, axillary, subscapular, thigh and calf) (Telford et al., 1984).

3.2.3. Dehydration

In the 24 hours following the euhydrated weight measurement, the rowers attempted to lose 5% of their bodyweight. Within the rowing group all subjects but one achieved the 5% target. Because the non-rowing subjects had never been required to lose weight rapidly by dehydrating before this experiment, it was accepted that the 5% target may be very difficult for them to achieve. For this reason, the non-rowing subjects attempted to lose 3% of their bodyweight or greater, if possible. Within the non-rowing group, all subjects but two achieved the 3% target. All subjects were asked to lose the prescribed weight through a combination of restricted fluid and food intake and exercise in sweat gear. All exercise was to have been completed the evening before the weigh-in. In one case only, however, insufficient weight had been lost 1.5 hours prior to the weigh-in. He exercised lightly for half an hour in waterproof clothing on a treadmill in the laboratory. The use of saunas and/or diuretics were not permitted for any subject. All urine passed by the non-rowers during the 24 hour dehydration period was collected. Each subject was requested to keep an accurate record of his fluid and food consumption and activities during the weight loss period. The individual diaries are presented in appendix D.

Following the 24 hour dehydration period, body weight was measured and a blood sample taken. Immediately following the collection of this blood sample, rehydration commenced.

3.3. THE EFFICACY OF WATER AND SOLUTIONS OF VARYING SODIUM CHLORIDE CONCENTRATION IN RESTORING PLASMA VOLUME AFTER DEHYDRATION

The rehydration protocol involved the ingestion of 250 ml of cold fluid (4°C) every 15 minutes for 90 minutes. The total amount of fluid ingested, therefore, was 1.5 litres. The eighteen non-rowers were randomly assigned in a single blind manner to one of three "saline groups": Group "0.1%" were given a 0.1% NaCl solution to drink during the rehydration period and groups "0.2%" and "0.3%" drank the 0.2% and 0.3% saline solutions respectively. The eight rowers were given only water to drink. Half an hour after the final drink, another blood sample was taken from the saline subjects. The post-rehydration procedure for the rowers is described in the following section.

3.4. METABOLIC PROCESSES DURING PERFORMANCE IN A MAXIMAL ROWING TRIAL FOLLOWING PARTIAL REHYDRATION AFTER RAPID WEIGHT LOSS

The eight rowers were lightweight oarsmen of national representative standard. All subjects were familiar with dehydration regimens similar to the one used in the present study. A random cross-over design was used in which each subject performed two maximal rowing trials; a normal euhydrated trial and a dehydrated trial. The trials were separated by one week.

Any reference to "dehydrated" trial and other cognate phrases refer to the situation where, before the trial, the subjects were partially rehydrated after having achieved a loss of five percent bodyweight by dehydration. The "normal" trial and other cognate phrases refer to the situation where no dehydration took place before the rowing trial.

3.4.1. Post-rehydration

Thirty minutes after the last drink, rehydrated body weight was determined, a rehydrated/pre-trial blood sample and a pre-trial biopsy of the vastus lateralis muscle was obtained. The rowers were then permitted to warm up before the trial.

3.4.2. The rowing trial

The rowing trials were performed on a Gjessing rowing ergometer. The ergometer was used in preference to on-water rowing in order to increase control and avoid varying environmental factors such as wind, tide and ambient temperature. All athletes were very familiar with the Gjessing ergometer as this apparatus had been used by Australian selectors to assist with the selection of National team members. The subjects often obtained test-retest scores of within 30 flywheel revolutions of each other (less then 0.8% difference) in a six minute all-out trial (M.Owen, National Coach; Personal Communication).

The trial was designed to simulate the amount of work completed by lightweight coxless pairs during a 2000 metre race. Performance was quantified as the minimum time it took to achieve a work target of 4200 revolutions of the flywheel with 3.0 kg resistance added. Throughout the trials, progressive flywheel scores, stroke ratings, heartrates and metabolic data were recorded every thirty seconds.

Warm ups for both the normal and dehydrated trials were of ten minutes duration. Light rowing was interspersed on three occasions by a burst of high intensity rowing of less than 10 seconds duration. Within thirty seconds of the completion of the warm up, the trial commenced. Athletes were requested to assume that the task was a competitive event and to pace themselves accordingly. Within two minutes from the time of completion of the trial, a post-trial muscle biopsy was taken through

the same cutaneous incision used to obtain the pre-trial biopsy. Five minutes post-exercise completion, a post-trial blood sample was taken for blood lactate determination.

3.5. ANALYSES

3.5.1. Blood analyses

Euhydrated, dehydrated and rehydrated blood samples were analysed for haematocrit. Data presented are averages of triplicate measurements for the rowers and quadruplicate measurements for the saline groups. Haemoglobin concentration for both groups was measured in triplicate using the Cyanmethemoglobin method (Sigma, 1984). For details of the method see appendix B. Relative plasma volume changes were determined using the following equation:

$$\% \text{ change PV} = 100 \times \left[\frac{Hb_{pre}\ (100 - Hct_{post})}{Hb_{post}(100 - Hct_{pre})} \right] - 100$$

(Dill and Costill, 1974)

Pre- and post-trial blood samples were analysed for blood lactate concentration using a standard enzymatic assay (Sigma, 1968). Details of the analysis are found in appendix B.

3.5.2. Urine analyses

The 24 hour urine samples taken from the saline groups (non rowing group) were analysed for volume and potassium and sodium ion concentration using emission flame spectrophotometry (GBC 903 Atomic Absorption Spectrophotometer in the emission mode). Urine samples were diluted by a factor of either 400 or 500 to bring the electrolyte concentration into the region of peak sensitivity of the spectrophotometer. Details of the analysis can be found in appendix B.

3.5.3. Muscle glycogen

Pre- and Post-Trial muscle biopsy samples were taken through the same incision approximately one third of the length from the proximal edge of the patella to spina iliaca anterior superior. The samples were removed from the deep portion of the vastus lateralis muscle and immediately frozen in liquid nitrogen and stored at -80°C. Muscle samples taken in association with the second rowing trial, one week later, were obtained from the contralateral leg. For analysis, the samples were freeze dried for 48 hours at -60°C (Edwards Modulyo freeze dryer) to account for possible changes the water content of the muscle in dehydration as demonstrated by (Costill et al., 1976a). Following freeze drying, the muscle fibres were dissected free from blood and connective tissue and weighed to the nearest 0.01 mg. The muscle fibres

were analysed for glycogen content using the enzymatic procedure of Passonneau and Lauderdale (1974). Details of the analysis can be found in Appendix B.

3.5.4. Metabolic measurements

Metabolic measurements during the trials were made with open circuit spirometry. During both exercise tests, air was inspired and expired through a Hans-Rudolf valve (dead space = 80 cm^3) and large diameter (Internal diameter = 3.5 cm) plastic tubing. Gas Temperature of the expired gas was measured by a Yellow Springs temperature probe (Model 43TA) placed in the expired air tubing. Expired air was directed through a spirometer (Pneumoscan S30) and into a mixing chamber. Samples of gas were continually pumped from the mixing chamber at a flow rate of 300 ml min^{-1} into gas analysers that measured the oxygen (Applied Electrochemistry O_2 S-3A) and carbon dioxide (Applied Electrochemistry CO_2 CD-3A) concentration of the expired gases. Before each test the gas analysers were calibrated with commercially prepared gas mixtures whose compositions had been verified by the Lloyd-Haldane apparatus. Data collected by various metabolic instruments were analysed online by an IBM XT personal computer programmed to calculate $\dot{V}O_2$ using standard techniques for open circuit indirect colorimetry (Consolazio et al., 1963). The highest oxygen consumption recorded during an averaged thirty second period during the continuous maximal exercise test was determined to be $\dot{V}O_2$ peak. This

was expressed in units of l min^{-1}. Heart rates were determined using telemetry (Sports Tester, Polar Electro, Fitness Technology, Finland. Model PE-3000) every 30 seconds.

3.6. STATISTICAL ANALYSES

Data analyses were done using BMDP Statistical Software. Where appropriate, data were analysed using one-way and two-way analyses of variance with repeated measures or regression analysis. Significant interaction effects for two-way ANOVA were examined post hoc using Simple Main Effects. Where significant differences between means were still unable to be identified, Newman-Keuls post hoc analyses were employed. Significant differences for one-way ANOVA were analysed using Newman-Keuls multiple comparison procedure. Where appropriate, data were analysed using two-tailed paired t-tests. The level of probability considered to reject the null hypothesis was set at $p<0.05$. Subsequent reference to statistical significance assumes $p<0.05$ unless otherwise specified.

CHAPTER 4

THE EFFECT OF 24 HOURS OF DEHYDRATION ON BODYWEIGHT, PLASMA VOLUME AND URINE ELECTROLYTES ($[K^+]$ AND $[Na^+]$) AND THE EFFECT OF REHYDRATION ON PLASMA VOLUME

4.1. INTRODUCTION

The effect of dehydration on plasma volume has been previously investigated by many authors (cf review by Harrison, 1985). It is well known that acute weight loss, achieved by dehydrating, results in a loss of plasma volume (Harrison, 1985). Whether there are changes in plasma electrolyte concentration and osmolality depend on the method used to induce the weight loss. The use of diuretics results in hypovolaemia accompanied with an almost proportionate loss of electrolytes in the urine. As a result, diuretics cause minimal disturbances to the concentrations of circulating electrolytes or resting muscle water (Armstrong *et al.*, 1985a; Bergstrom and Hultman, 1966). Sweat is ordinarily hypotonic to intracellular and extracellular fluid (Costill, 1977; Sato, 1977). Therefore plasma will become hyper-osmotic when dehydration is mediated primarily by sweat loss (Sawka *et al.*, 1983, Senay and Christensen, 1965). The principal ions lost in sweat are those from the extracellular fluid, sodium and chloride, whilst little potassium is lost (Costill 1977). Since the extracellular fluid has a

far smaller volume than the intracellular fluid, there is a disproportionately larger loss of water from the plasma and interstitial spaces than from the larger intracellular compartment (Costill *et al.*, 1976a; Costill 1977). However, the absolute loss of water from the compartments is found to be quite evenly distributed between the extracellular and intracellular spaces, except at low levels of dehydration (<2.0%) (Costill *et al.*, 1976a; Costill, 1977). At all stages of dehydration, Costill (1976a) and Nose *et al.* (1988a) found that plasma volume changes account for 10-11% of the total body water loss.

Various attempts to rehydrate subjects after dehydration have been made (cf reviews by Murray, 1987; Lamb and Brodowicz, 1986). Fluids have been used with varying concentrations of electrolyte, glucose or a glucose polymer, combinations of these or water alone. As previously mentioned (Section 2.2.2.1), the rate of restoration of the fluid loss is limited by a deficiency of the thirst sensation, the rate of absorption of fluid from the intestine and the equilibrium processes between the intracellular and extracellular compartments (Nielsen, 1984a). After a 4-5% weight loss, subjects were unable to restore bodyweight (Klinzing and Karpowicz, 1986) or plasma volume (Costill and Sparks, 1973) within a three hour limit when allowed to rehydrate taking fluid *ad libitum*. Methodologies involving a structured rehydration regimen (Costill and Sparks, 1973; Nose *et al.*, 1988b; Torranin *et al.*, 1979) achieved better success towards restoring bodyweight back to euhydration levels after a 4-5% initial weight loss in the same three hour limit; however, plasma volume was still not fully restored.

Unfortunately, because a portion of fluid may remain in the gastro-intestinal tract (Costill and Sparks, 1973) or in the bladder after having been excreted through the kidneys (Costill and Sparks, 1973), achieving a physiological euhydration in less then three hours appears to be a very difficult task.

This chapter reports on the effects of 24 hours of dehydration subsequently followed by a 90 minute structured rehydration regimen on plasma volume. Urine volume, K^+ and Na^+ concentration were analysed pre- and post-dehydration. One and a half litres of 0.1%, 0.2% and 0.3% saline solutions were consumed by three separate groups (n=6) respectively (See section 3.3). One subject participated in both the 0.1% and 0.3% saline groups. The rowing group (n=8), making up a fourth group, drank water.

In this and succeeding chapters, the following phrases should be interpreted as follows:

(a) "euhydrated status" or "PV0" is the euhydrated plasma volume status and serves as a point of reference. For the purpose of statistical analyses, PV0 has been defined as: PV0 = 0.0 %

(b) "decrease in plasma volume" or "ΔPV1" means the change in plasma volume from the euhydrated state (PV0) to the state immediately after the 24 hour period of dehydration (PV1);

(c) "restoration of plasma volume" or "ΔPV2" means the change in plasma volume from the state immediately after the 24 hour period of dehydration (PV1) to the state immediately after the 90 minute period of rehydration;

(d) "deficit in plasma volume" or " ΔPV3" refers to the plasma volume status immediately before the commencement of the rowing trial. The relationship of ΔPV3 to ΔPV1 and ΔPV2 is illustrated in Figure 4.1.

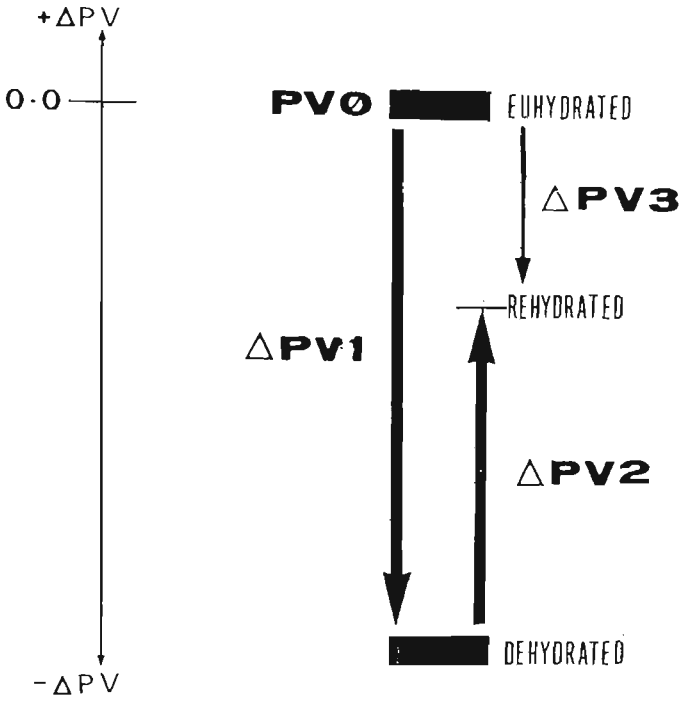


Figure 4.1

Vector diagram showing the relationship of ΔPV3 to ΔPVI and ΔPV2

4.2. RESULTS

4.2.1. The subjects

The mean subject data for each group are presented in Table 4.1.

TABLE 4.1 Subject data.

GROUP	BODYWEIGHT Pre-Dehy (Kg)	BODYWEIGHT Post-Dehy (Kg)	% diff (%)	Sum Skinfolds (mm)	Height (cm)	Age (yr)	N
SALINE GP 0.1%	68.48 ± 3.63	65.88 ± 3.39	3.75 ± 0.40	60.1 ± 8.3	174.0 ± 8.0	21.2 ± 1.2	6
SALINE GP 0.2%	73.79 ± 4.09	71.23 ± 3.91	3.45 ± 0.20	73.4 ± 9.1	177.0 ± 2.8	23.0 ± 1.9	6
SALINE GP 0.3%	70.47 ± 2.89	67.68 ± 2.82	3.96 ± 0.30	65.7 ± 7.8	175.8 ± 3.1	22.3 ± 1.1	6
SALINE MEAN	70.92 ± 2.01	68.26 ± 1.92	3.72 ± 0.18	66.4 ± 4.5	176.1 ± 1.9	22.2 ± 0.8	18
ROWERS MEAN	73.47 ± 1.47	69.65 ± 1.31	5.15 * ± 0.15	62.3 ± 6.5	181.9 ± 2.0	21.4 ± 0.7	8

* Significantly different from Saline Groups.

Values are mean ± S.E.

There were no significant differences between the saline groups with respect to bodyweight pre- and post-dehydration, % loss of bodyweight,

sum of skinfold thicknesses, height and age (One-way ANOVA). Therefore the data from each saline group for each variable was pooled to give the "pooled saline group mean". There was no difference in any variable between the pooled saline group and the rowers except percent loss of bodyweight (One-way ANOVA). The pooled saline group lost 3.72 ± 0.18% of bodyweight compared to the rowing group who lost 5.15 ± 0.14% of bodyweight (One-way ANOVA). The bodyweight post-dehydration was significantly different from the bodyweight pre-dehydration for all groups (t-test).

4.2.2. The relationship between weight loss and plasma volume decrease

ΔPV1 was -14.6 ± 1.3%, -12.2 ± 0.9% and -13.1 ± 1.0% for the 0.1%, 0.2% and 0.3% saline drink groups respectively (pooled saline group mean = -13.3 ± 0.6%) [Figure 4.4] and -12.5 ± 1.4% for the rowers [Figure 4.3] after the 24 hour period of dehydration. There were no significant differences between the groups (One-way ANOVA).

No relationship was found to exist between percent loss of bodyweight and ΔPV1 [Figure 4.2]. Compared to the pooled saline group, however, the ratio of the loss of plasma volume to loss of bodyweight was less for the rowers. Plasma volume decreased on average 3.6% for every 1% decrease in body weight for the pooled saline group but only 2.5% per 1% decrease in bodyweight for the rowers.

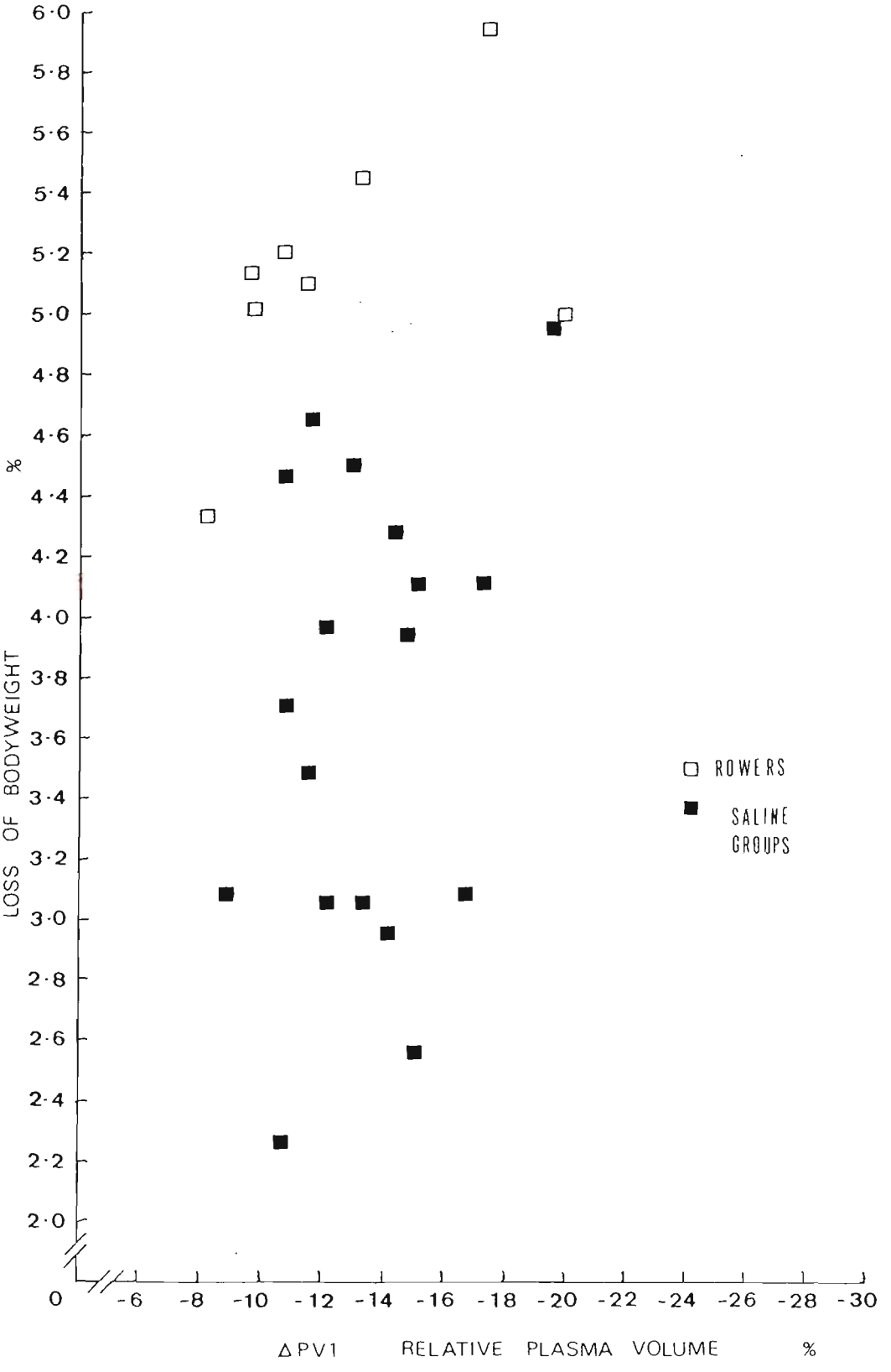


Figure 4.2

The relationship between % loss of bodyweight and ΔPVI

4.2.3. Changes in Urinary Volume, K+ and Na+ following dehydration in the pooled saline group.

The effect of dehydration on various urinary variables in the non-rowing group is presented in Table 4.2.

TABLE 4.2 The effect of dehydration on urinary variables in the pooled saline group.

URINE	NORMAL	DEHYDRATED
24 hour volume (ml)	1404.7 ± 65.8	664.4 ± 77.7*
Ave. Flow rate (ml min^{-1})	0.98 ± 0.05	0.46 ± 0.05*
$[K^+]$ (mmol l^{-1})	52.29 ± 1.67	141.42 ± 8.40*
Total excretion K^+ over 24 hours (mmol)	73.98 ± 4.60	84.59 ± 4.35
$[Na^+]$ (mmol l^{-1})	83.66 ± 5.81	127.15 ± 7.63*
Total excretion Na^+ over 24 hours (mmol)	114.49 ± 7.52	81.33 ± 8.11*

* Significantly different from normal N=18
Mean ± S.E.

The volume of urine collected over 24 hours, in the euhydrated state, were similar to the volumes observed by Armstrong *et al.* (1985b) and Lijnen *et al.* (1985). During the dehydration period, urine production decreased significantly. Costill *et al.* (1975)

observed similar decreases in urine volume in dehydration. In that study, urine production decreased from a control value of approximately 1300 ml per 24 hours to 600 ml per 24 hours during the loss of 4.2% of body weight.

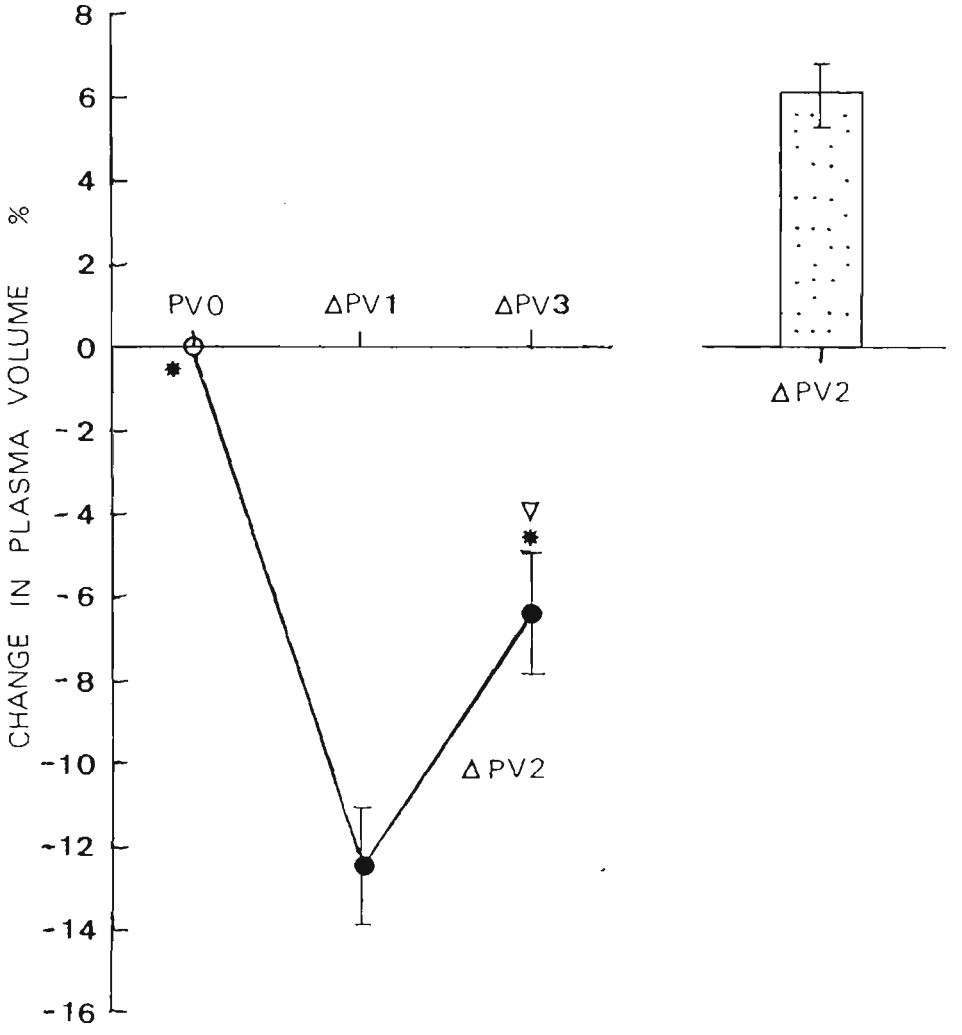
The total urinary excretion of potassium increased but failed to reach significance during the dehydration period relative to the normal collection period. On the other hand, the total urinary excretion of sodium decreased significantly during the dehydration period relative to the normal collection period. Similar observed changes in urinary electrolytes following dehydration have been published previously (Costill *et al.*, 1976b; Mnatzakanian and Vaccaro, 1984; Zambraski *et al.*, 1975).

4.2.4. The efficacy of drinking water and solutions of varying sodium chloride concentration in restoring plasma volume after dehydration

The rowing group was rehydrated with water alone. The efficacy of drinking water in restoring plasma volume (Δ PV2) is presented in Figure 4.3. After the 90 minute rehydration period, plasma volume (Δ PV2) significantly increased (Δ PV1 significantly different from Δ PV3) by 6.0 $\pm$ 0.6%. Despite the increase in plasma volume, a significant deficit (Δ PV3 significantly different from PV0) of 6.5 $\pm$ 1.3% remained after rehydration with water (One-way ANOVA, Newman-Keuls multiple comparison).

The efficacy of increasing sodium chloride concentration in restoring plasma volume (Δ PV2) is presented in Figure 4.4. The 0.1%, 0.2% and 0.3% solutions were associated with a plasma volume restoration (ΔPV2) of +4.8 ± 0.4%, +6.4 ± 0.4% and +10.2 ± 0.6% ($p<0.05$) to leave a plasma volume deficit (ΔPV3) of 9.8% ± 1.2%, 5.8 ± 0.8% and 3.0 ± 0.9% respectively ($p<0.05$).

Consumption of each saline solution caused a significant restoration of plasma volume (Δ PV1 significantly different from Δ PV3) although each group remained with a significant plasma volume deficit after rehydration (Δ PV3 significantly different from ΔPV0) (ANOVA, Newman-Keuls Post Hoc Analysis). There was no significant difference between the 0.1% and the 0.2% saline solutions with respect to plasma volume restoration during rehydration (ΔPV2). The restoration of plasma volume in the 0.3% group, however, was significantly greater than in either the 0.1% and 0.2% groups.

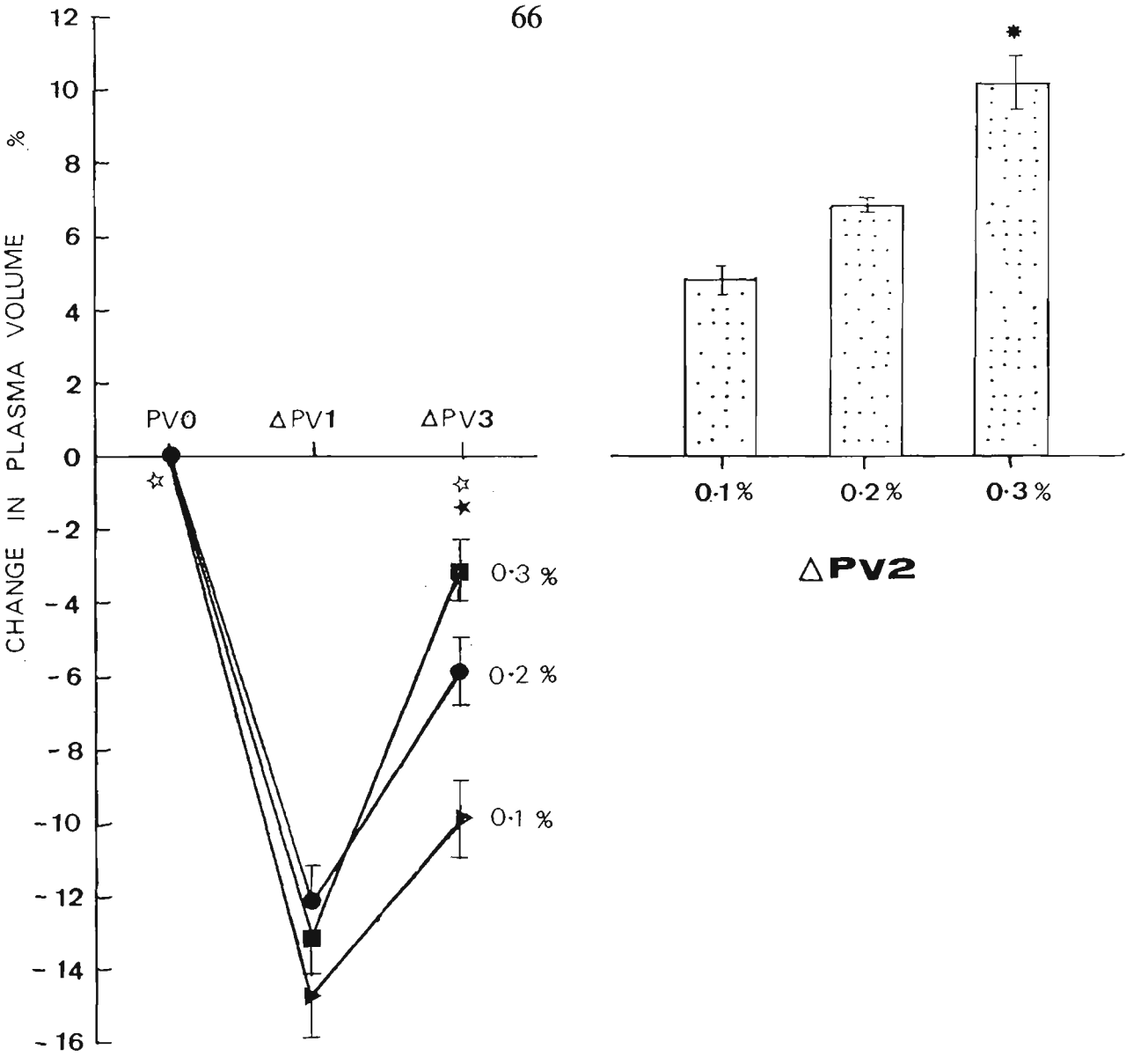


✱ Significantly different from ΔPV1

▽ Significantly different from ΔPV0

Figure 4.3

The efficacy of drinking water in restoring plasma volume after dehydration



☆ Significantly different from ΔPVI

★ Significantly different from ΔPV0

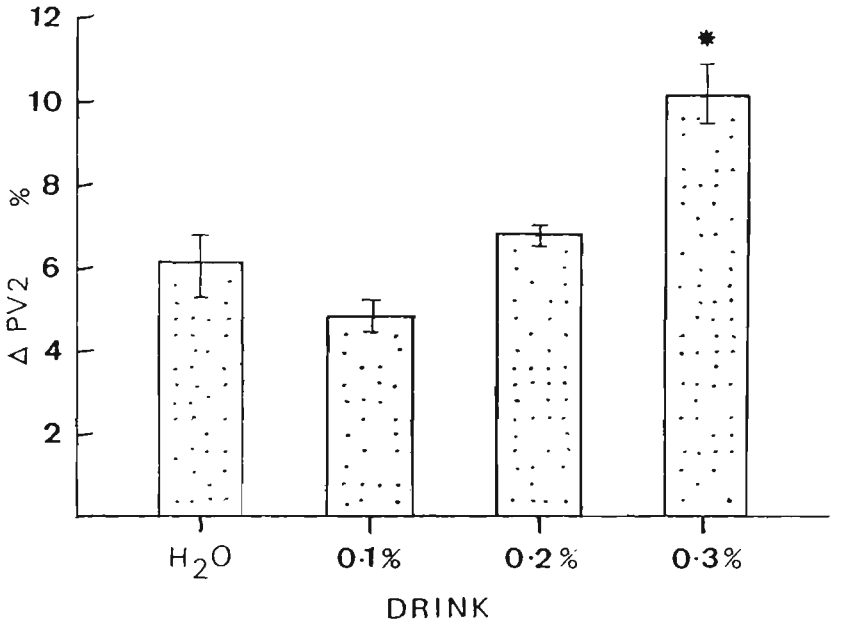
✸ Significantly different from 0.1% and 0.2%

Figure 4.4

The efficacy of increasing sodium chloride concentration in restoring plasma volume

4.2.5. Comparison of the efficacy of water versus three saline solutions in restoring plasma volume after dehydration

The efficacy of drinking water versus the three saline solutions in restoring plasma volume after dehydration (Δ PV2) is presented in Figure 4.5. The efficacy of water in restoring plasma volume was not significantly different from that of the 0.1% and the 0.2% saline solutions (One-way ANOVA). The 0.3% saline solution, on the other hand, had a significantly greater effect on plasma volume restoration (Δ PV2) compared to water (One-way ANOVA, Newman-Keuls multiple comparison).



* Significantly different from H_2O, 0.1% and 0.2%

Figure 4.5

The efficacy of drinking water versus three saline solutions in restoring plasma volume after dehydration

4.3. DISCUSSION

4.3.1. Rapid weight loss and changes in plasma volume

The 24 hour dehydration protocol caused a significant decrease in plasma volume. A mean decrease in bodyweight of 3.7 ± 0.2% in the pooled saline group was associated with a plasma volume change of -13.3 ± 0.6%. Several authors have reported similar changes. Horstman and Horvath (1972) observed that after seven hours of heat exposure (48°C), a 3.6% decrease in bodyweight of their subjects was accompanied by a 13.6% loss of plasma volume. In the present study, the rowers, despite a significant greater loss of body weight (5.15 ± 0.15%), lost an amount of plasma volume (12.5 ± 1.4%) similar to that experienced by the pooled saline group. Evidence gained from the questionnaire, that each subject was required to complete, showed that the members of the pooled saline group were not participating in a strenuous training program as were the rowers. In addition, the rowers' oxygen consumption data (see chapter 6) would place the rowers in an elite category in terms of their aerobic fitness (McCardle *et al.*, 1981), which could be attained only through regular and sustained high intensity exercise. On this basis it seems reasonable to assume a difference in training status between groups although no tests were performed to verify this. Similar decreases in plasma volume to that observed for the rowers were obtained by Sawka *et al.* (1984a). They induced a 5% loss of body weight over a 24 hour period using voluntary dehydration and light exercise in moderately hot conditions. Most of the weight loss occurred during the

light exercise session, which had been conducted fifteen hours before the end of the 24 hour period. Plasma volume decreased on average only 5% after the 24 hour period.

Estimates of the percent contribution of plasma volume to the change in total body water for the rowers and pooled saline group were made from the following data and equations and is presented in Table 4.3. It is assumed that total body water (TBWater) is 60% of euhydrated body weight (BWeight) and plasma volume (PV) is 5% of bodyweight (Costill *et al.*, 1976a; Nielsen *et al.*, 1986; Sawka *et al.*, 1984). Although there is evidence to suggest that the intravascular fluid compartment in trained and/or heat acclimatized subjects is slightly larger than that of untrained subjects (Harrison, 1985), such a difference is not likely to be so large as to invalidate the assumptions used in the following equations.

The variables required for the calculation of percent contribution of plasma volume to the change in total body water after dehydration are:

decrease on plasma volume (%) - $\Delta PV1$

loss of body weight (%) - $\Delta BWeight$

initial body weight prior to dehydration (kg) - ($BWeight_{init}$)

Using the above, the following variables can be calculated:

$$\%\Delta TBWater = \%\Delta BWeight \times 100/60$$

$$TBWater_{pre} = BWeight_{init} \times 0.60$$

$$PV_{pre} = BWeight_{init} \times 0.05$$

$$TBWater_{post} = TBWater_{pre} - (TBWater_{pre} \times \%\Delta BWeight/100)$$

$$PV_{post} = PV_{pre} - (PV_{pre} \times \%\Delta PV/100)$$

If A = % contribution of plasma volume to the change in total body water after dehydration, then:

$$A = \left[\frac{\frac{PV_{pre}}{TBWater_{pre}} - \frac{PV_{post}}{TBWater_{post}}}{1 - \frac{TBWater_{post}}{TBWater_{pre}}} \right] \times 100$$

Using the above equation, the relative contribution of plasma volume decline to total body water loss (A) for the rowers and pooled saline group is shown in Table 4.3.

TABLE 4.3 Relative contribution of plasma volume decline to total body water loss (A) for the rowers and pooled saline group

	BW (kg)	ΔBW (%)	ΔPV1 (%)	A (%)
Rowers	73.47 ± 1.47	5.15 ± 0.15	12.5 ± 1.4	4.1 ± 1.4
Saline	70.92 ± 2.01	3.72 ± 0.18	13.3 ± 0.6	10.8 * ± 1.2

* Different from rowers
Values are mean ± S.E.

As Table 4.3 shows, the change in plasma volume contributes about 10.8% to the change in total body water in the pooled saline group. This is similar to that found by Costill *et al.* (1976a) and Nose *et al.* (1988a). However, in the rowers, plasma volume contributed significantly less, in a relative sense, to the decrease in total body water. These observations suggest that a number of other variables, in addition to relative weight loss, may have played a role in influencing the intra/extra vascular fluid dynamics after rapid weight loss. Training status, as displayed by the rowers versus the pooled saline group, may be one of these factors. The following subsection will discuss training status in relation to loss of plasma volume after dehydration.

4.3.2 Training status and loss of plasma volume after dehydration.

In the literature, the terms haemoconcentration and hypovolaemia have often been used, incorrectly, as if they were interchangable (Harrison, 1985). In the following discussion, these terms are defined as having the following meanings:

a) Haemoconcentration - refers to the loss of plasma water from the vascular space such that there is a relative increase in haematocrit and haemoglobin concentration; and

b) Hypovolaemia - a loss of central blood volume due to thermoregulatory vasodilation in the cutaneous vascular bed (or haemorrhage) such that haematocrit and haemoglobin concentration remain unchanged.

It has been established previously that trained individuals lose less plasma volume when compared to untrained individuals during exercise of the same absolute intensity (Convertino *et al.*, 1983) and during heat stress (Senay, 1972, 1975 and 1978). The phenomenon that trained individuals lose relatively less plasma volume than untrained individuals in dehydration, without the acute effects of exercise or heat stress being present immediately before measurement, has not been reported previously.

The haemodynamic response of a trained and/or heat acclimatized subject is quite different to that of an unacclimatized subject when exposed to a thermal stress. Many authors have attempted to attribute this phenomenon simply to a net loss or gain of plasma protein from within the vascular system (Freund *et al.*, 1987; Harrison, 1981; Senay, 1972, 1975, 1978, 1979). For example, Senay (1972, 1975, 1978, 1979) noted that on entering a hot environment, haemoconcentration was observed in untrained and/or unacclimatized subjects. As an explanation, Senay proposed that due to thermoregulatory induced vasodilation within the cutaneous circulatory bed, circulating plasma protein was lost through the walls of the cutaneous capillaries into the interstitium with an attendant loss of water. The increased perfusion pressure within the cutaneous vascular bed would have increased lymph flow leading to a return of some plasma protein via the thoracic lymph duct, however, the rate of return was insufficient to compensate the amount of protein lost from the capillaries. On the other hand, Senay also observed that for heat acclimatized and/or trained subjects, haemoconcentration did not occur on exposure to heat. In fact, on occasions the subjects experienced haemodilution. Senay proposed, here, that the cutaneous capillaries must have become less permeable to plasma proteins allowing the rate of return of plasma protein to the circulation via the lymph to equal or exceed the rate of loss from the capillaries. There is, however, no experimental evidence to date that confirms Senay's hypothesis that cutaneous capillaries become less permeable to plasma protein following training and/or heat acclimatization. Further, it is unlikely that Senay's mechanism

explains the observation made in the present experiment as cutaneous vasodilation would be required to initiate the plasma protein augmentation process. The subjects in the present study had not been exposed to a thermal stress for quite some hours prior to the dehydrated blood sample except for one individual who exercised lightly 90 minutes prior to the weigh-in. When the blood sample was taken in the dehydrated state, the subjects were all demonstrating marked cutaneous vasoconstriction (as evidenced by peripheral venous shutdown), a phenomenon that has been reported previously to occur in dehydrated subjects (Horstman and Horvath, 1972; Horowitz and Meiri, 1985; Nadel, 1980).

Loss of protein from the intravascular space and the subsequent movement of water has also been reported to occur during intense exercise (Harrison, 1981). Following exercise Harrison *et al.* (1981) also observed that more protein was returned to the intravascular space during recovery than that which was lost during the exercise itself. The explanation advanced was that lymph flow may have remained elevated above pre-exercise levels after exercise, thereby augmenting the total plasma protein mass. Senay (1986) confirmed that there was little doubt that a plasma volume expansion (haemodilution) occurred after cessation of exercise but added that the same phenomenon also occurred immediately after unacclimatized subjects were removed from a thermal stress. Similarly, Green *et al.* (1987) observed that after three consecutive days of cycling (2 hours per day at 65% peak $\dot{V}O_2$), plasma volume in untrained males increased by approximately 20%. In another related study (Green *et al.*, 1989), it was noticed also,

however, that serum protein and albumin concentrations were 8-10% lower ($p<0.05$) than the pre-experimental levels and that this effect was persistent during both rest and exercise. Melin *et al.* (1980), on the other hand, reported that there was no difference in plasma protein concentration between highly trained and untrained groups at rest. These apparently conflicting observations do, however, correspond with the observation that heat acclimatized subjects expanded their plasma volume during daily heat exposures and lost that volume between exposure sessions whereas unacclimatized subjects maintained their expanded plasma volumes between sessions (Senay, 1986). It is also probable, on the one hand, that highly trained athletes experience haemodilution on cessation of exercise but then rapidly lose that extra plasma volume. On the other hand, untrained individuals appear to maintain the post-exercise haemodilution between daily training sessions.

With respect to the present study, however, exercise induced plasma protein and water shifts should have returned to pre-exercise levels, particularly with respect to the rowers, at the time of the dehydrated blood sample. Because there is no difference in resting plasma protein concentration between trained and untrained individuals (Melin *et al.*, 1980) (although this remains to be verified in resting dehydrated subjects), it is unlikely that differing plasma protein dynamics in the trained and untrained subjects contributed to the differences observed in the present study.

A difference in sweat tonicity secreted by the rowers and the pooled saline group during exercise may, however, explain the differences

observed. During exercise, trained subjects are able to secrete a sweat of far lower tonicity than their untrained counterparts (Harrison *et al.*, 1981). It is possible that the rowers were able to maintain a relatively higher extracellular osmolar mass compared to the pooled saline group after their exercise during the dehydration period. The resultant higher extracellular osmolality in the rowers may have resulted in a greater net movement of fluid from the intracellular compartment into the extracellular compartment and therefore indirectly contributed to the maintenance of plasma volume. It is of interest, therefore, that Koszlowski and Saltin (1964) observed also that plasma volume levels were retained to the very last whilst total body water declined during work induced dehydration. They concluded that the water losses, in absolute terms, were primarily taken from the intracellular fluid compartment. Other observations, however, have indicated that the extracellular fluid contributes at least as much (Costill *et al.*, 1976a) or more (Nielsen *et al.*, 1986) to total body water loss than the intracellular fluid. The difference may well reflect training status of the individuals. It appears that at rest, therefore, trained individuals may have a greater capacity to draw upon intracellular water reserves in dehydration for the maintenance of plasma volume than untrained individuals. Considering this, training status may need to be more carefully controlled in future dehydration research.

4.3.3. The relationship between dehydration and urine volume, K^+ and Na^+ excretion

During the dehydration period, urine production and the total urinary excretion of sodium decreased significantly. Glomerular filtration rate and urine flow levels have been found to decrease linearly with the severity of dehydration up to a loss of eight percent of body weight. At this point, maximal levels of renal water reabsorption are elicited (Gauer *et al.*, 1970). In the presence of hypovolaemia, the secretion of arginine vasopressin is stimulated and water is subsequently reabsorbed from the renal tubule. The glossopharyngeal afferent pathways from the aortic arch and carotid sinus are the main pathways whereby changes in arterial pressure are associated with alterations in vasopressin (Berl *et al.*, 1974; Schrier *et al.*, 1979; Share and Levy, 1962). Reduced mean renal arterial pressure which accompanies dehydration (Laragh and Sealey, 1973; Gauer *et al.*, 1970) also stimulates renin mediated aldosterone release despite elevated plasma sodium levels, as demonstrated by Nose (1988c), causing the reabsorption of sodium and water. Finally, decreased levels of circulating atrial natriuretic peptide have been demonstrated to occur in dehydrated rats (Schwartz *et al.*, 1986; Takayanagi *et al.*, 1985) and this also may have contributed to the decrease in natriuresis observed.

Total urinary potassium excretion increased during the dehydration period. An increase in plasma angiotensin II levels or plasma potassium concentration will cause an increase in the rate of aldosterone synthesis. Aldosterone acts on the distal convoluted tubule to promote the active reabsorption of sodium in exchange for potassium (Hierholzer and Wiederholt, 1976). The amount of potassium lost in the urine during the dehydration period, however, appears to be physiologically insignificant when compared to the total body stores of intracellular potassium. During the 24 hour dehydration period, 84.6 mmol of potassium was excreted compared to 74.0 mmol lost whilst euhydrated, leaving a net increase of 10.6 mmol of potassium lost during dehydration beyond that which would have been lost in any case. If body water is lost in equal proportions from the extracellular and intracellular compartments (for untrained subjects) and plasma potassium levels do not significantly change in dehydration (Costill, 1977), the loss of potassium along with the extracellular fluid would account for 7.9 mmol of the excreted potassium. Therefore, only 3 mmol of the lost potassium could have originated from the intracellular space.

4.3.4. The restoration of plasma volume following rehydration

The direct comparison between the rowers and the pooled saline group, with respect to the restoration of plasma volume during rehydration, should be interpreted with care considering the conclusion made in relation to the effects of training status on plasma volume changes during dehydration (section 4.3.1.1.). Differences in training

status between the groups also may result in differing distributions of the ingested fluid between the body fluid compartments.

The 0.3% sodium chloride solution used for rehydration, was associated with a significantly greater restoration of plasma volume compared to water and the 0.1% and 0.2% saline solutions respectfully. Davis *et al.* (1987), using Deuterium labelled beverages observed that accumulation of D_2O in plasma was faster using hypotonic saline as a rehydration fluid than water.

Gastric emptying rate appears to be the primary limiting factor in the delivery of water and minerals to the circulation (Wheeler and Banwell, 1986). Hunt and Pathak (1960) observed a three fold increase in gastric emptying rate when a dilute saline solution in the range of 0.2% - 0.4% was drunk compared to that of distilled water. Another variable affecting gastric emptying rate is the residual volume. If the fluid volume in the stomach is less than 200 ml, gastric emptying rate slows (Costill and Saltin, 1974b; Fordtran and Saltin, 1967). On the other hand, when gastric volumes exceed 600 to 800 ml, gastric emptying is retarded and often accompanied by feelings of fullness and nausea (Costill and Saltin, 1974b; Greenleaf and Sargent (1965). As a practical suggestion therefore, fluids should be ingested as frequently as practicable and in as large a volume as is tolerable to hasten gastric emptying for most athletes. The volume of intake has been found to be 150-250 ml per 15-20 minutes (Lamb and Brodowicz, 1986).

Costill and Sparks (1973) observed that whilst their subjects were rapidly rehydrating using water, the subjects' serum sodium and chloride levels fell rapidly and were 2-4% below normal at the end of the three hour rehydration period. Despite incomplete plasma volume replacement, rapid decreases in serum osmolality are known to result in large increases in urine production that is probably mediated by osmoreceptor regulation of vasopressin secretion (Robertson, 1974). On the other hand, Costill and Sparks (1973) observed that when a fluid containing electrolytes was consumed, plasma osmolality did not decrease below normal by the end of the rehydration period. In the present study, the rowers, who drank water, found they needed to urinate during the rehydration period despite their still significant plasma volume deficit. On the other hand, the subjects who rehydrated with the 0.3% saline solution did not experience a diuresis.

Many authors have added various quantities of carbohydrate to their rehydration medium, not only to improve palatability of the fluid, but also to attempt to maintain blood glucose levels and/or to replenish depleted muscle glycogen stores (Murray, 1987). Where carbohydrate supplementation is particularly desired, the addition of small amounts of sodium will greatly enhance the rate of intestinal absorption of a glucose solution (Fisher and Gardner, 1974). The rate of absorption of a sodium solution alone, however, is not greatly enhanced by the presence of glucose (Fisher and Gardner, 1974). Further, gastric emptying rate is increasingly inhibited in proportion to the energy content of the drink (Murray, 1987). Consequently, if rehydration in a limited period of time is the major criterion, the addition of glucose

may slow the rehydration process. Finally, maximum performance ordinarily does not return to control values after rapid rehydration, regardless of the composition of the drink (Lamb and Brodowicz, 1986).

These findings, however, must be weighed against the replacement of relatively large volumes in terms of the purely subjective difficulty in the provision of an "acceptable" form of fluid. Thus consideration of the palatability and in addition to the gastric acceptance qualities of the respective fluid treatments must be considered in the selection of an appropriate fluid medium (White and Ford, 1983).

CHAPTER 5

THE EFFECT OF 24 HOURS OF DEHYDRATION FOLLOWED BY PARTIAL REHYDRATION ON ROWING PERFORMANCE

5.1. INTRODUCTION

It has been established that dehydration has a deleterious effect on endurance type exercise (Armstrong *et al.*, 1985a; Caldwell *et al.*, 1984). On the other hand, anaerobic type activity is not affected by dehydration (Jacobs, 1980). The effect of dehydration on strength is still not firmly established (Serfass *et al.*, 1984; Torranin *et al.*, 1979).

A maximal rowing trial over 2000 metres requires a mixture of endurance and power (Klavora, 1976, 1982). The ability to produce powerful concentric contractions involving all major muscle groups (Larsson and Forsberg, 1980; Pyke *et al.*, 1979; Secher, 1975; Williams, 1976), 34 to 38 times per minute for six and eight minutes, is of paramount importance to achieve success in the sport.

Achieving physiological euhydration after a 5% or greater weight loss in less then five hours appears to be a very difficult task, with a significant proportion of the fluid remaining in the gut or excreted via the kidneys (Costill and Sparks, 1973). The rules of racing in the lightweight rowing category may allow as little as thirty minutes in which to attempt to rehydrate. At best, only ninety minutes are available to achieve euhydration. It would appear that most lightweight rowers who have dehydrated to make the weight requirements are likely to be competing whilst in a significantly dehydrated state.

This chapter reports the effects of a 5% loss of bodyweight over twenty four hours subsequently followed by a ninety minute structured rehydration regimen on maximal rowing performance. During the rehydration period, the rowers drank water only.

5.2. RESULTS

5.2.1. Subjects

Individual data on plasma volume changes and trial times for the rowers is presented in Table 5.1.

TABLE 5.1 Individual data on plasma volume changes and trial times for the rowers

subject #	PLASMA VOLUME CHANGES PV1 (%)	PV2 (%)	PV3 (%)	TRIAL TIMES Normal (N) (mins)	Dehyd (D) (mins)	Difference (D-N) (mins)
1	-19.9	+6.8	-13.1	6.35	7.25	0.90
2	-17.4	+7.7	- 9.8	7.72	8.42	0.70
3	-11.3	+7.4	- 3.9	7.30	7.68	0.38
4	-10.3	+6.2	- 4.1	7.18	7.18	0.00
5	-10.8	+7.9	- 2.9	7.30	7.22	-0.08
6	- 8.0	+3.5	- 4.5	6.50	6.60	0.10
7	-13.2	+4.6	- 8.6	7.28	7.87	0.59
8	- 9.4	+4.1	- 5.3	6.57	6.85	0.28
Mean ± S.E.	-12.5 ± 1.4	+6.0 ± 0.6	- 6.3 ± 1.3	7.02 ± 0.17	7.38 ± 0.21	0.36 ± 0.12

5.2.2. The trial times

The effect of dehydration on trial times is illustrated in Figure 5.1. The dehydrated subjects took significantly longer times in achieving the target of 4200 flywheel revolutions (7.38 ± 0.21 and 7.02 ± 0.17 minutes for the dehydrated and normal trials respectively). There were no significant differences in average stroke rating between the normal and dehydrated trials.

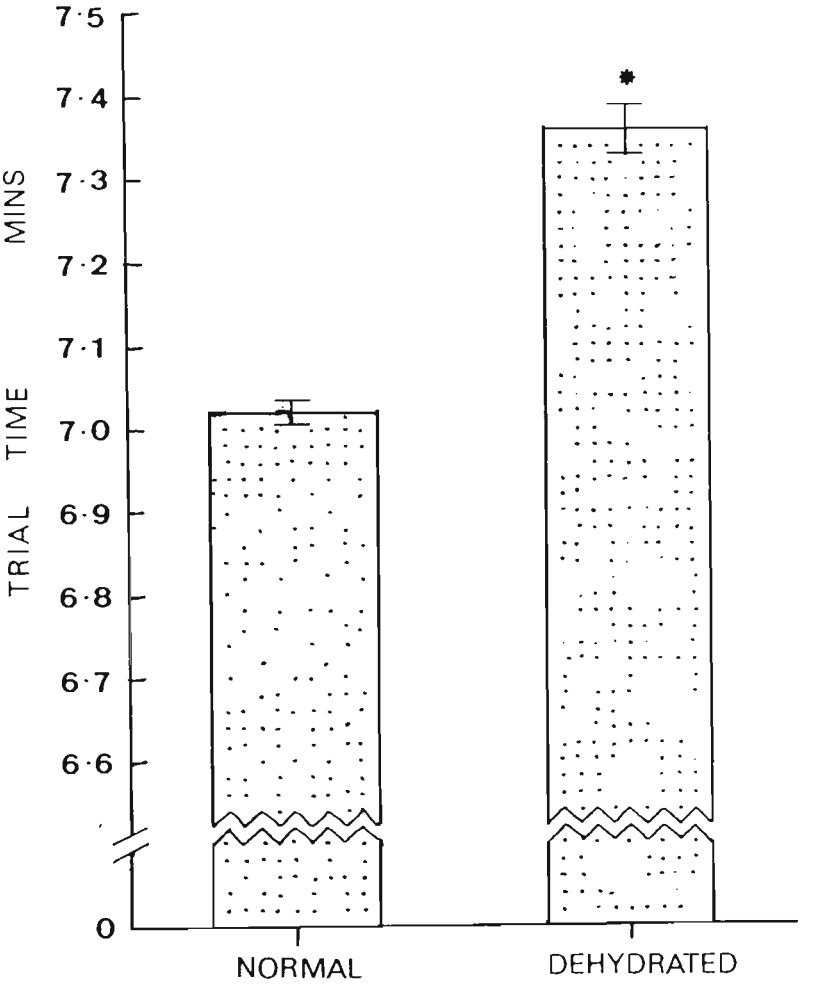


Figure 5.1

The effect of dehydration on trial times

5.2.3. The effect of dehydration on performance during the trial.

The first of the matched flywheel revolution data points became missing at time 6.50 minutes due to subject #1 completing his normal trial in 6.35 minutes. Therefore, performance was analysed only during the first six minutes of each trial in order to have paired data (dehydrated trial-normal trial) over all time intervals in the analysis. The effect of dehydration on cumulative work output during the rowing trials is illustrated in Figure 5.2. During the first six minutes of the trial, a significantly lower total work output was achieved during the dehydrated trial compared to the normal trial (ANOVA). The difference in work output was significant at 2.50 minutes and for all time intervals after 3.00 minutes. Non cumulative work output during the rowing the trials is illustrated in Figure 5.3. During each 0.50 minute time interval, non-cumulative work was consistently lower for the dehydrated trial except during the second 30 second period. The differences in non-cumulative work output were significant at minutes 2.0, 2.5, 3.5 and 4.0.

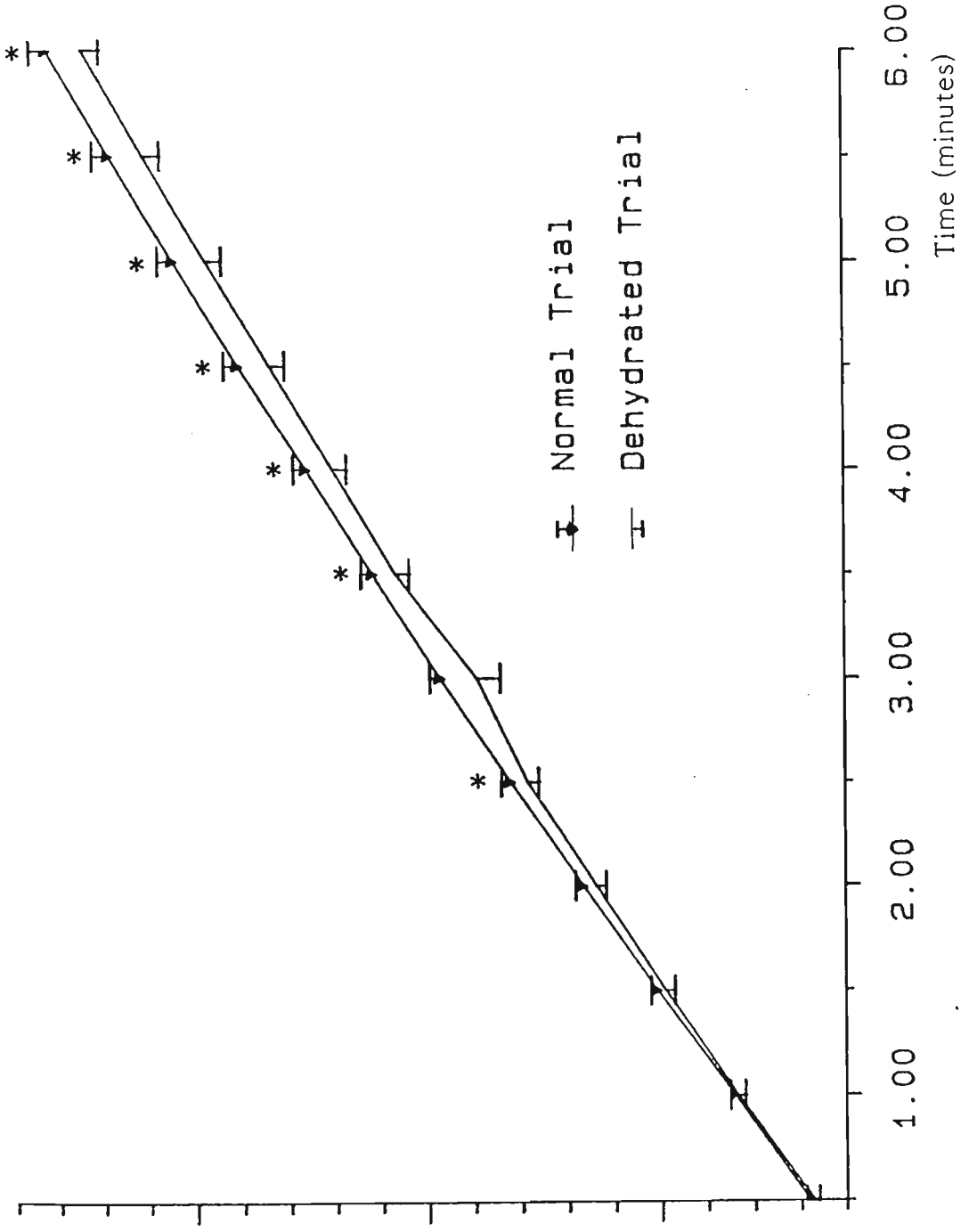


Figure 5.2 **Mean number of flywheel revolutions accumulated over the first six minutes of each trial.**

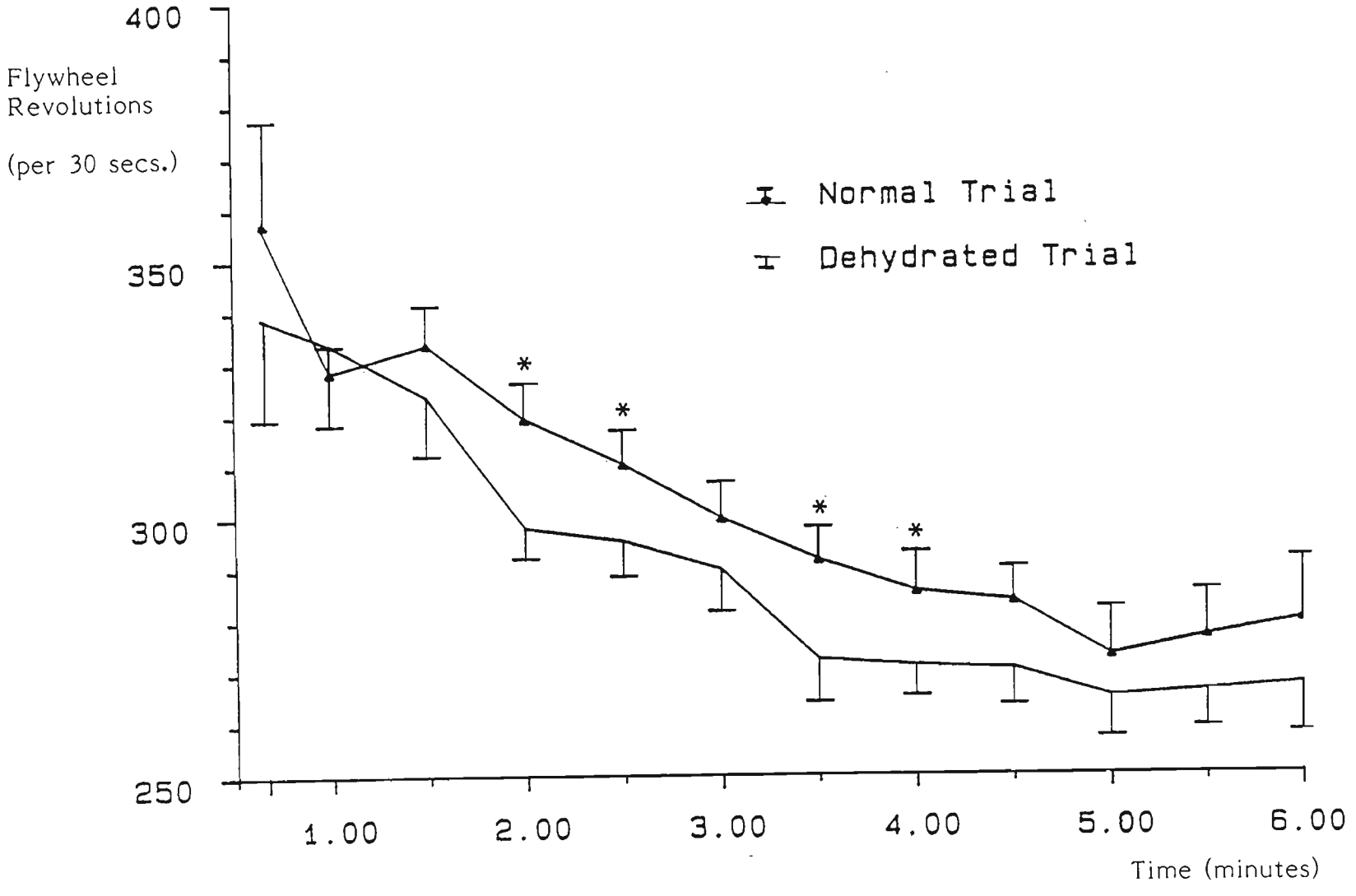


Figure 5.3 **Mean number of flywheel revolutions accumulated during each 30 second period over the first six minutes of each trial.**

5.2.4. The effect of plasma volume changes on performance

The difference in time to complete the dehydrated and normal trials was related to plasma volume status. The increase in time to complete the dehydrated trial was linearly related to the decrease in plasma volume (Δ PV1) after dehydration ($r=0.87$, $p<0.01$; Figure 5.4). However, the increase in time to complete the dehydrated trial was more strongly related to the deficit in plasma volume (Δ PV3) after rehydration ($r=0.93$, $p<0.01$); Figure 5.5). It is apparent that a plasma volume deficit (Δ PV3) greater than 4% has a pronounced effect on rowing performance whereas a deficit of less than 4% appears to have little or no effect.

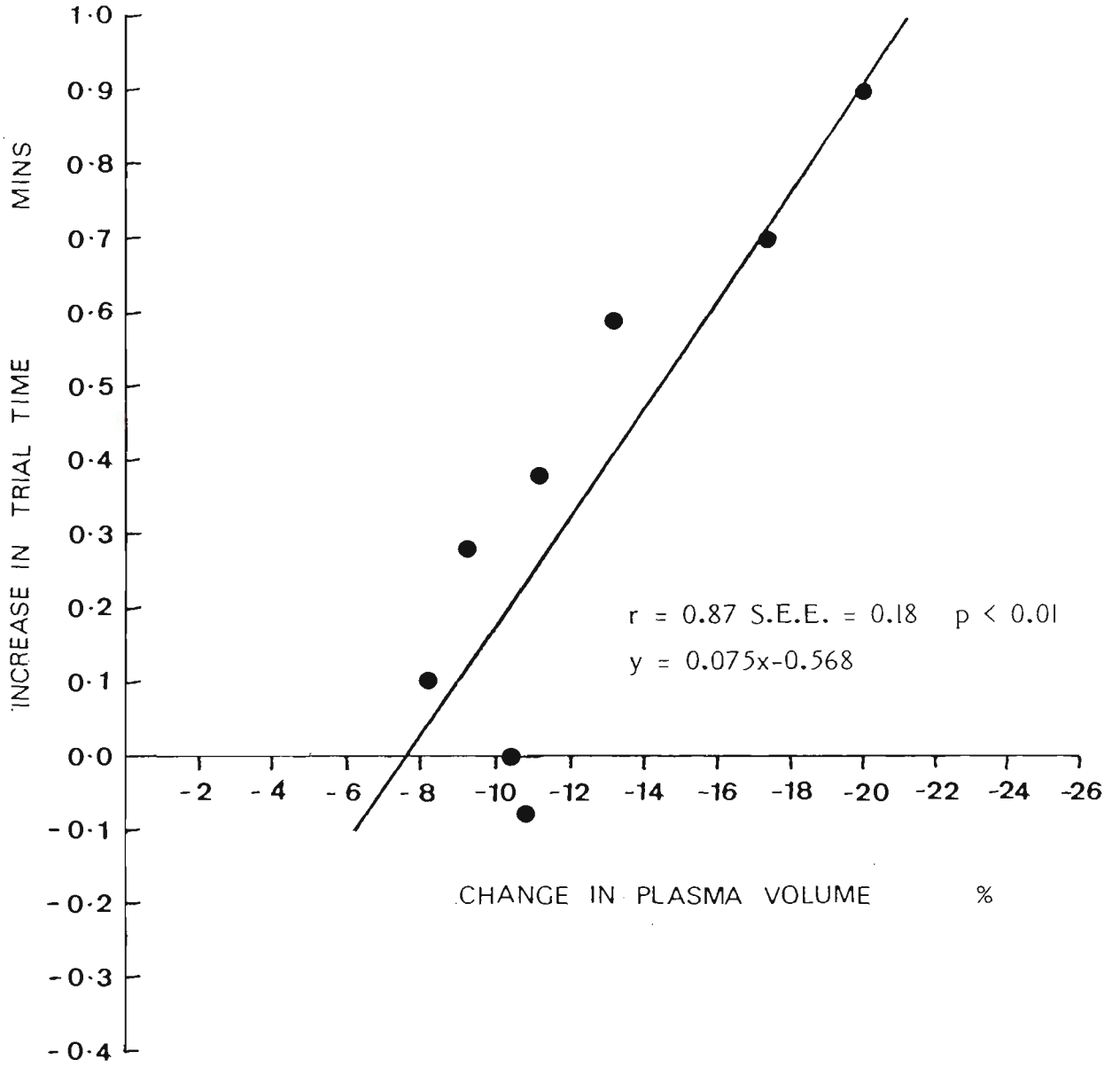


Figure 5.4

ΔPVI versus increase in time to complete the dehydrated trial

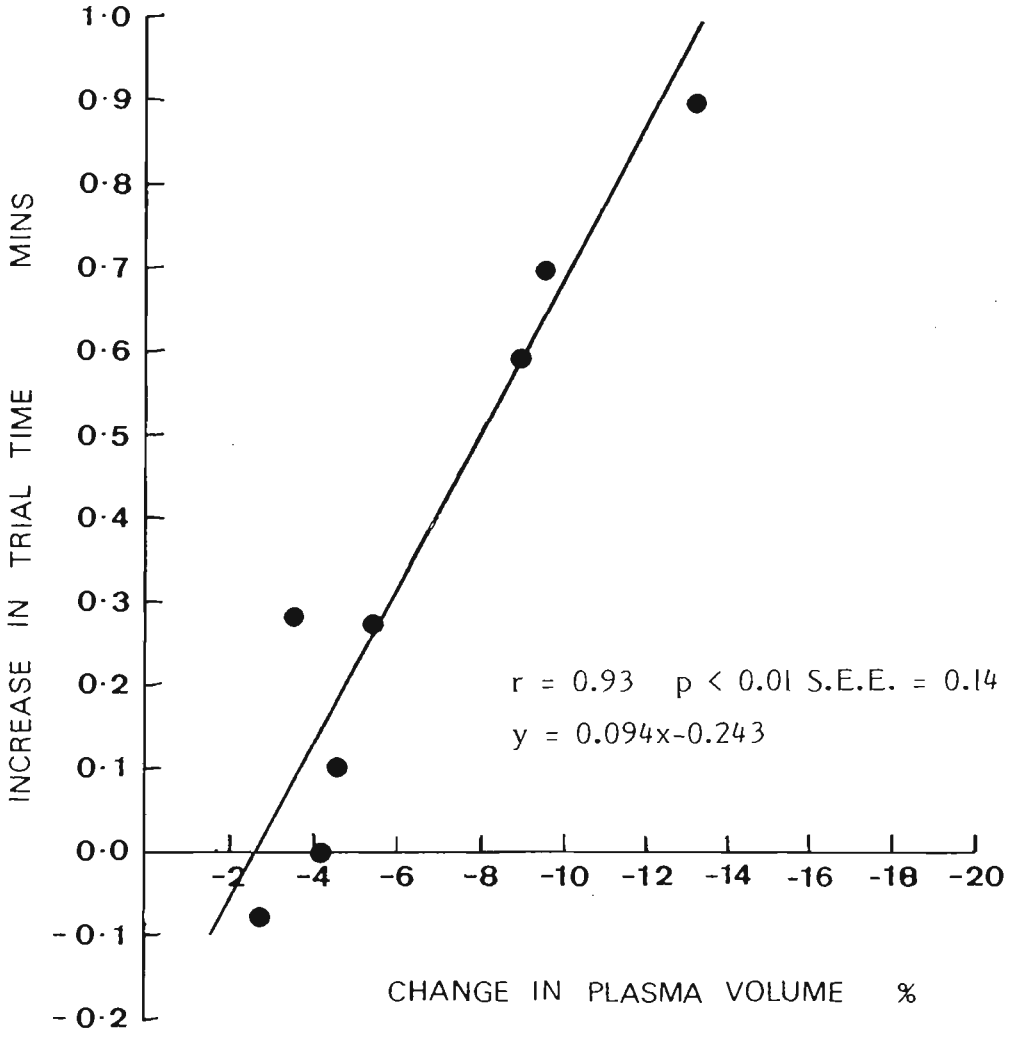


Figure 5.5

ΔPV3 versus increase in time to complete the dehydrated trial

5.3 DISCUSSION

5.3.1. Rowing performance and dehydration

The results show that dehydration caused a significant decrease in the ability to sustain work at a high intensity. On average, it took approximately 22 seconds longer to complete the dehydrated time trial compared to the normal trial. This increase in time to "cover the same distance" could be equated to losing a boat race by approximately 117 metres in an eight-oared race or approximately 95 metres in a paired-oared event over 2000 metres Increased times were likewise observed in the data of Armstrong *et al.* (1985a) for running trials of 5,000 metres and 10,000 metres

Elite level performance in rowing requires not only being able to produce powerful concentric contractions 32-38 times per minute for up to eight minutes, but also to maintain a high level of technique whilst suffering, at the same time, from almost intolerable pain and fatigue. By optimizing technique, the rower minimizes the oscillations in boat speed that occur during each stroke cycle. A decrease in the level of technique will usually manifest itself as a decrease in mean boat speed. On the rowing ergometer, boat speed is equated to the number of flywheel revolutions produced in a selected period of time. A similar

technique is also required to minimize oscillations in the rotational velocity of the flywheel during each stroke cycle on the rowing ergometer.

Dehydration possibly may affect rowing technique in two ways. First, is the effect of dehydration on cognitive responses and motor co-ordination. Strydom *et al.* (1968), using rifle marksmen, demonstrated that following dehydration accuracy dropped 15-20% below control scores. It is recognized, however, that the validity of relating this evidence to rowing may be questioned, because a fine motor skill has been compared to a gross motor skill. Bell *et al.* (1964) monitored visual and auditory alertness during exposure to hot and humid conditions. Results indicated that a greater proportion of signals was missed as dehydration began to occur. If co-ordination is impaired, even though peak power and stroke rating may remain the same, mean boat speed will decrease because of the increased variation in boat speed occurring during each stroke cycle. Secondly, dehydration may induce fatigue faster in some muscle groups compared to others and again impair overall muscle coordination. The data presented by Torranin *et al.* (1979) suggested that the isotonic endurance of the muscles involved in leg extension were affected adversely to a greater degree in thermally dehydrated subjects than the musculature associated with the arm and pectoral girdle.

5.3.2. Rowing performance and changes in plasma volume

The decrease in plasma volume after the dehydration regimen (△PV1) was reported in the previous chapter. Following the 90 minute period of rehydration, plasma volume increased but euhydration levels were not achieved. In contrast to Nielsen *et al.* (1981), a relationship between plasma volume status after dehydration and performance was observed. A better relationship, however, was observed with respect to the difference in time to complete the dehydrated and normal trials versus plasma volume deficit (△ PV3) (Figure 5.5) compared to difference in times and △PV1 (Figure 5.4). This suggests, as might be expected, that performance is likely to be more related to the plasma volume status immediately prior to the commencement of the trial (△PV3) than to the dehydrated plasma volume status (△ PV1) prior to rehydration. It also lends support to the view that rehydration does have a beneficial effect towards restoring performance after acute weight loss (Nielsen *et al.* 1986).

The performance of two subjects (#4 and #5) was unaffected by a plasma volume deficit (△PV3) of approximately 4% during the dehydrated trial. On the other hand, two other subjects (#3 and #6), who also carried a plasma deficit of approximately 4%, were affected to the same degree as the others in proportion to the extent of their body weight loss (Figure 5.5). Several authors have shown that for plasma volume losses of less than six percent, vasoconstriction in peripheral vascular beds can usually compensate for the reduced blood volume and assist in

maintaining stroke volume and cardiac output (Nielsen, 1984b; Claremont *et al.*, 1976; Klinzing and Karpowicz, 1986; Sproles *et al.*, 1976). In the present study, however, the rowers appeared to be unable to sustain performance levels with plasma volume deficits greater than four percent of euhydrated levels.

CHAPTER 6

THE EFFECT OF 24 HOURS OF DEHYDRATION FOLLOWED BY PARTIAL REHYDRATION ON METABOLIC AND CARDIORESPIRATORY VARIABLES DURING A MAXIMAL ROWING TRIAL.

6.1. INTRODUCTION

Decreased plasma volumes have been associated with higher heart rates during submaximal workloads (Nielsen, 1984a; Sawka *et al.*, 1984b; Candas *et al.*, 1988); or at maximum heart rate a decreased tolerance for work (Caldwell *et al.*, 1984; Saltin 1964b). Symptoms appear to be very similar to those elicited during work in the heat; temperature regulation efficiency is decreased (Nielsen, 1984b) and the threshold for the onset of sweating is increased (Nielsen, 1974; Fortney *et al.*, 1984). Dehydration, like heat stress, causes a marked decrease in physical working capacity (Nielsen, 1984a). It is not clear, however, whether the decreases in physical work capacity either can be attributed primarily to changes in cardiovascular factors or to biochemical changes in metabolism.

6.2. RESULTS

The effects of dehydration and subsequent rehydration on cardiorespiratory and metabolic variables during a maximal rowing trial are presented in Table 6.1.

TABLE 6.1 The effects of dehydration and subsequent rehydration on cardiorespiratory and metabolic variables during a maximal rowing trial.

	NORMAL	DEHYDRATED
Trial Time (mins)	7.02 ± 0.17	7.38 ± 0.21#
Peak Oxygen Consump (l min^{-1})	4.65 ± 0.20	4.68 ± 0.14
Peak Ventilation Rate (l min^{-1})	147.2 ± 1.6	146.5 ± 1.6
Ave $\dot{V}e/\dot{V}CO_2$ ¶	32.95 ± 0.07	34.01 ± 0.06
Ave Respiratory Exchange Ratio	1.12 ± 0.01	0.92 ± 0.02#
Peak Heart Rate (beats min^{-1})	188 ± 0.7	188 ± 0.9
Net Blood Lactate accumulation (mmol l^{-1})	8.77 ± 0.31	6.77 ± 0.24#

Significantly different from Normal Trial
Values are mean ± S.E.

¶ Where averages are quoted, data from minute 1 has been excluded from the calculation.

6.2.1. Peak Oxygen Consumption

In the dehydrated trial, there was no significant difference in peak oxygen consumption compared to the normal trial (Table 6.1). Although $\dot{V}O_2max$ was not measured, the peak oxygen consumption under the conditions has been shown to be an equivalent measure of maximum oxygen uptake (Mahler *et al.*, 1984). These authors compared the results of an all out six minute rowing trial (similar to the one in the present study) to an incremental rowing exercise test. They obtained no significant difference between the trials for maximum heart rate, maximum oxygen consumption and maximum ventilation rate. On the basis of their results, it is reasonable to assume that the peak oxygen consumption obtained in this study is representative of each subject's maximum oxygen uptake.

6.2.2. Peak ventilation rate, maximum heart rate and average respiratory exchange ratio during the trials

The peak ventilation rate during the dehydrated trial was not significantly different from the normal trial (Table 6.1). There was also no significant difference in peak heart rate between the trials. As discussed previously with respect to maximal oxygen uptake, it is reasonable to assume that the peak heart rate obtained in this study is representative also of maximum heart rate (Mahler *et al.*, 1984).

The RER can be used to estimate the relative proportions of fat and carbohydrate being oxidized (McArdle *et al.*, 1981). The RER in this study, however, may give only a tentative indication of substrate oxidation. This is because during exercise of high intensity, the expired CO_2 may not be representative of its production from metabolism due to hyperventilation and metabolic acidosis. During the normal trial, the subjects consistently showed an RER greater than unity, which is indicative of the above phenomenon (Table 6.1). On the other hand, the average RER during the dehydrated trial was significantly lower and suggests (within its limitations) that there was an increase in fat oxidation for that trial. This conclusion is strongly supported by the blood lactate data.

6.2.3. Blood Lactate Concentration

Pre- and Post- trial net blood lactate accumulation for both hydration states is shown in figure 6.1. During the normal trial, blood lactate concentration increased significantly from 2.11 ± 0.25 mmol l^{-1} pre-trial to 11.01 ± 1.11 mmol l^{-1} post-trial. During the dehydrated trial, blood lactate concentration increased significantly from 1.89 ± 0.19 mmol l^{-1} pre trial to 8.78 ± 0.72 mmol l^{-1}. There was no significant difference between pre-trial blood lactate concentration for the dehydrated and normal trials. Post-trial blood lactate accumulation, however, was significantly lower for the dehydrated trial than for the normal trial (ANOVA).

6.3. DISCUSSION

6.3.1. The effect of dehydration on the cardiovascular system during exercise

No significance difference in maximum heart rate after dehydration was observed in the present study. Similar observations have also been reported previously (Armstrong *et al.*, 1985a; Klinzing and Karpowicz, 1986; Nielsen *et al.*, 1984a). A decrease in plasma volume, which causes a decrease in cardiac filling pressure (Hales, 1986), activates baroreceptor reflexes that cause vasoconstriction in the peripheral vascular beds and an increase in heart rate (Nielsen, 1984b). At submaximal exercise intensities, therefore, the athlete has a cardiovascular reserve, i.e. the capacity to increase heart rate to compensate for the decrease in plasma volume. As heart rate approaches maximum, the lack of cardiovascular reserve correlates well with a decrease in maximal work capacity (Nadel *et al.*, 1987).

The skin vasculature plays an important part in thermoregulation and regulation of central blood volume. Where there is competition for a limited blood volume, such as during maximal exercise whilst dehydrated, the cardiovascular system eventually dominates at the expense of thermoregulation (Hales, 1986). It is believed that impaired thermoregulation did not contribute to the decrease in physical work capacity of the dehydrated rowers due to the relatively short duration

of the rowing trials. As mentioned previously (chapter 5), a four percent plasma volume deficit did, however, appear to represent the limit with which peripheral vasoconstriction was able to compensate for the loss of central blood volume during the dehydrated trial.

6.3.2. Blood lactate accumulation, the respiratory exchange ratio and peak oxygen uptake during the rowing trials

The average RER in the dehydrated trial was lower compared to the normal trial. During submaximal exercise ranging from 50 to 85 percent of maximal oxygen uptake a decrease in RER has been observed (Costill *et al.*, 1976a; Saltin, 1964a, 1964b). There are no reports, however, of the effect of dehydration on RER during prolonged high intensity exercise of six to seven minutes duration.

There was no significant change in peak oxygen uptake despite the lower work rate during the dehydrated trial compared to the normal trial. Similar observations in dehydrated subjects have been reported previously (Armstrong *et al.*, 1985a; Baum *et al.*, 1986; Nielsen, 1984a). Caldwell *et al.* (1984), on the other hand, did report a slight decrease in $\dot{V}O_2max$. These authors observed concomitantly a moderate decrease in ventilation rate ($\dot{V}e$). They attributed the decrease in Ve to diminished strength and endurance of the respiratory musculature after dehydration based on results reported by Torranin *et al.* (1979). That there was no change in peak oxygen uptake, as reported in the present study, can be accounted for by assuming that

there was a greater contribution of fat to energy metabolism, as suggested by the decreased average RER (within its limitations) and the decreased net blood lactate accumulation. In the combustion of carbohydrate, one litre of oxygen will yield 21.1 kJ of energy, but for fat only 19.6 kJ (Passmore and Eastwood, 1986). The greater the contribution of fat to energy metabolism, the lower the work output per unit volume of oxygen consumed.

Decreases in lactate accumulation have been reported previously for dehydrated subjects during maximal exercise lasting four to seventeen minutes (Armstrong *et al.*, 1985a; Caldwell *et al.*, 1984; Klinzing and Karpowicz, 1986; Nielsen *et al.*, 1981; Saltin, 1964a, 1964c). The decrease in blood lactate accumulation can be accounted for by a number of mechanisms, viz:

a) There is a decrease in lactate production rate related to the reduced work rate;
b) There is a decrease in lactate efflux from the muscle;
c) The clearance of lactate may have increased;
d) The muscles of the subjects may be glycogen depleted; and
e) Dehydration may itself inhibit carbohydrate metabolism, which will manifest itself in a lower accumulation of lactate.

The decreased accumulation of blood lactate during the dehydrated trial may have been simply a function of the decreased work rate. The average workrate to blood lactate accumulation for both trials, therefore, has been compared, viz:

The average work rate for the Gjessing rowing ergometer was estimated as follows:

$$\text{Average speed of flywheel} = \frac{\text{Total flywheel revolutions}}{\text{Trial time}}$$

$$= \text{Revolutions sec}^{-1}$$

Flywheel circumference: 1 m

Resistance: 3 kg

$$\text{Work rate} = \frac{\text{Resistance x Distance}}{\text{Time}} = \text{kgm min}^{-1}$$

$6.1162 \text{ kgm min}^{-1} = 1 \text{ W}$

	NORMAL TRIAL	DEHYDRATED TRIAL
Ave. flywheel speed	9.97 revs sec^{-1}	9.48 revs sec^{-1}
Ave. work rate	293.53 W	279.11 W
Net lactate accumulation	8.77 ± 0.31 mmol l^{-1}	6.77 ± 0.24 mmol l^{-1}

The ratios:

	$\frac{\text{Dehydrated}}{\text{Normal}}$
Ave. work rate	0.95
Net lactate accumulation	0.77

The ratios of average work rate to net lactate accumulation are substantially different. A 5% decrease in work rate corresponded to approximately a 23% decrease in blood lactate accumulation for the dehydrated trial. It is unlikely that, at such high workloads, which the present study required, the relatively small changes in work rate could completely account for the large decrease in lactate accumulation observed for the dehydrated trial.

The efflux of lactate is linked to H^+ transport out of the cell (Heisler, 1973). The efficiency of the transport process can be reduced by extracellular acidosis and a low bicarbonate concentration outside the cell (Sutton *et al.* 1981). It is probable that the dehydrated subjects were slightly keto-acidotic compared to the normal subjects, just before the rowing trial, because of the low dietary intake of carbohydrate during the 24 hour dehydration period. It is believed, however, that this keto-acidosis was insignificant when compared to the exercise-induced acidosis that would have been incurred during the rowing trials. In addition, the $\dot{V}e/\dot{V}CO_2$ ratio indicates, according to Chiesa *et al.* (1969) and Jones *et al.* (1977), that the subjects experienced a greater metabolic acidosis during the normal trial than during the dehydrated trial. For these reasons, it appears unlikely that mechanism (b) described above can account for the altered blood lactate accumulation levels observed for the dehydrated trial.

Lactate uptake by human muscle can occur even during relatively strenuous exercise (Gollnick *et al.*, 1974; Essen *et al.*, 1975).

The lactate uptake is more evident, however, when the slow twitch fibres are glycogen depleted (Gollnick *et al.*, 1974). Jacobs (1981) also observed that blood lactate concentration and glycogen utilization rate only decreased when pre-exercise muscle glycogen levels had been experimentally reduced to less than 40 mmol kg^{-1} wet weight (approximately 160 mmol kg^{-1} dry weight). Although the subjects in the dehydrated trial had lower starting and finishing muscle glycogen levels compared to the normal trial (see chapter 7), they had not reached the level of glycogen depletion where these changes have been observed to occur. Further, there is evidence that during strenuous exercise (>80% VO_2max) glycogen depleted subjects have a significantly greater ventilation rate (Heigenhauser *et al.*, 1983). No significant difference in ventilation rate between the trials was observed in the present study. If anything, the ventilation rate was slightly lower during the dehydrated trial. Hence muscle lactate uptake and muscle glycogen depletion are also unlikely to have accounted for the lower blood lactate accumulation after the dehydrated trial.

Finally, Armstrong *et al.* (1985a) and Costill *et al.* (1976a) have both suggested previously, that in dehydration, a change in energy metabolism may occur resulting in an increased utilization of lipid as a fuel for exercise. In the present study, the significantly lower average RER observed during the dehydrated trial is an indication, at least, of a shift in substrate utilization. A decrease in the rate of glycogen utilization during the dehydrated trial would provide evidence also that dehydration itself may inhibit carbohydrate metabolism and manifest itself in a lower accumulation of lactate.

CHAPTER 7

MUSCLE GLYCOGEN UTILIZATION IN A MAXIMAL ROWING TRIAL FOLLOWING DEHYDRATION

7.1. INTRODUCTION

The utilization of muscle glycogen in dehydrated subjects during a maximal rowing trial has not been reported previously in the published literature. A maximal rowing trial lasting approximately six to seven minutes relies heavily on carbohydrate as the major substrate for metabolism (Hagerman *et al.*, 1979; Jackson and Secher, 1976).

Several studies in which pre-exercise glycogen levels in dehydrated subjects have been reported, conclude that dehydration, particularly exercise induced dehydration, depletes the muscle glycogen stores and is a major explanation for the decrease in maximal work capacity in dehydrated subjects (Costill and Saltin, 1973, 1975; Nielsen *et al.*, 1981). As discussed previously (section 2.6), it is impossible to separate the effects of glycogen depletion and the effects of dehydration on glycogen utilization rate where dehydration had been induced by prolonged submaximal exercise.

7.2. RESULTS

7.2.1. Muscle glycogen concentration

The mean muscle glycogen concentrations before and after the exercise trials are presented in Table 7.1.

TABLE 7.1 Mean muscle glycogen concentration (mmol glucosyl units kg^{-1} dry weight muscle tissue).

	NORMAL	DEHYDRATED
Pre-Trial	606.8 ± 37.9	394.7 ± 26.0 #
Post-Trial	403.2 ± 41.3 *	254.8 ± 15.3 #*
Difference	203.6 ± 18.6	139.9 ± 13.4 #

\# Significantly different from Normal
* Significantly different from Pre-trial
Values are Mean ± S.E., n=7

The resting muscle glycogen content was significantly lower before the dehydrated trial compared to the normal trial. During the trial, the dehydrated subjects also used significantly less glycogen (ANOVA).

7.3. DISCUSSION

7.3.1. Glycogen utilization rate during maximal exercise in dehydrated subjects

Table 7.1 shows that the rowers, prior to the dehydrated trial, had lower muscle glycogen levels compared to the normal trial. These levels, however, assuming a muscle water content of approximately 80%, were still within the normal range for the vastus lateralis muscle (50-90 mmol kg^{-1} wet weight) observed in subjects who were not glycogen depleted (Saltin and Gollnick, 1983).

The design of this research did not allow for a direct determination of the reasons for the decrease in the rate of glycogen utilization during the dehydrated trial. Three possible mechanisms may explain these results.

7.3.2. Possible Mechanisms

(a) **Work rate versus glycogen utilization rate.** Less work per unit time was performed during the dehydrated trial, reducing demand on the need for rapid glycogenolysis. To test the hypothesis that the decreased utilization of glycogen for the dehydrated trial may have been simply a

function of the decreased work rate, the average work rate to glycogen utilization rate for both trials were compared as follows:

The average work rate for the Gjessing rowing ergometer was calculated previously in Section 6.3.2.

The average rate of glycogen utilization was estimated viz:

$$\text{Ave. glycogen utilization rate} = \frac{\text{glycogen pre-post trial}}{\text{Trial Time}}$$

$$= \text{mmol glycosyl units kg}^{-1}\text{ dry weight sec}^{-1}$$

	NORMAL TRIAL	DEHYDRATED TRIAL
Ave. speed Flywheel	9.97 revs sec^{-1}	9.48 revs sec^{-1}
Ave. work rate	293.53 W	279.11 W
Ave. glycogen utilizn rate	4.8 mmol kg^{-1} sec^{-1}	3.2 mmol kg^{-1} sec^{-1}

The ratios: $\frac{\text{Dehydrated}}{\text{Normal}}$

Ave. work rate	0.95
Ave. Glycogen utilizn rate	0.65

The ratios of work rate to average glycogen utilization rate are substantially different. A 5% decrease in work rate corresponded to approximately a 35% decrease in glycogen utilization rate. It is unlikely, that at such high workloads as required in the present study, the comparatively small changes in work rate would completely account for the large decrease in glycogen utilization rate observed for the dehydrated trial.

(b) **Initial muscle glycogen content versus glycogen utilization rate.** The initial muscle glycogen content was higher for the normal trial compared to the dehydrated trial, possibly resulting in a greater utilization rate. It is well established that subjects performing exercise whilst glycogen depleted, use less glycogen, accumulate less lactate and have lower exercise endurance than when exercising with normal glycogen levels (Bergström *et al.*, 1967; Gollnick *et al.*, 1981). The subjects in the present study had very high glycogen levels prior to the normal trial (606.8 ± 37.9 mmol kg^{-1} dry weight) and normal to moderately high glycogen levels prior to the dehydrated trial (394.7 ± 26.0 mmol kg^{-1} dry weight). In studies where the rate of glycogen utilization during exercise with supercompensated muscle glycogen levels have been compared to exercise with normal glycogen levels, results have been equivocal (Richter and Galbo, 1986; Katz and Spencer, 1990). Richter and Galbo (1986) electrically stimulated perfused isolated hindquarters of rats (repeated maximal twitches) that were either glycogen supercompensated or normal (controls). They found

that increased initial muscle glycogen content was associated with increased glycogen breakdown and lactate release. Studies on humans have shown similar trends except that, as Richter and Galbo (1986) identified, changing the fat content of the diet causes marked hormonal, metabolic and circulatory changes that may influence glycogenolysis.

In contrast to the results obtained by Richter and Galbo (1986), Katz and Spencer (1990) found that glycogen availability did not measurably alter muscle glycolysis nor lactate lactate accumulation in the muscle during intense exercise. Eight subjects cycled at 95% of maximal oxygen uptake on two occasions: the first to fatigue having previously lowered muscle glycogen levels with a combination of diet and exercise to 201 ± 31 mmol kg^{-1} dry weight (LG); and the second at the same workload and duration as the first, having previously raised muscle glycogen stores to 583 ± 40 mmol kg^{-1} dry weight (HG). In the LG state, inosine monophosphate (IMP) levels were significantly elevated but the accumulation of fructose-6-phosphate (F,6-P) was markedly attenuated. Because F,6-P is an activator of PFK (Newsholme and Start, 1973), the attenuated accumulation of F,6-P should have theoretically decreased PFK activity and subsequently inhibited glycolysis. To explain their observations, Katz and Spencer (1990) proposed that in the LG state elevated levels of adenosine monophosphate (AMP), which also stimulates PFK (Newsholme and Start, 1973), may have been activating PFK and offsetting the reduction in F,6-P. Although they had not reported AMP levels, they inferred a rise in intramuscular levels of this metabolite from the observed increases in IMP levels.

(c) **Inhibition of glycolysis.** Glycogenolysis and/or glycolysis may have been directly inhibited by dehydration and this may explain the decreased maximum physical work capacity observed in dehydrated subjects. As reported previously (Section 6.3.2), the dehydrated trial was accompanied by a significantly lower blood lactate level and respiratory exchange ratio. It is apparent that even though glycogen was available, it was not able to be used as rapidly during the dehydrated trial compared to the normal trial, and subsequently, the capacity to perform work decreased.

As mentioned previously, a change in energy metabolism, resulting in an increased utilization of lipid as a fuel, appears to have occurred. A mechanism that might be proposed is that dehydration may decrease the activity of one or some rate-limiting enzymes in the glycolytic pathway leading to an increased reliance on β-oxidation. There appears to be no published information reporting the effects of dehydration on major glycolytic rate-limiting enzymes such as PFK. An indication that some carbohydrate catabolizing enzymes may be inhibited in dehydration was presented in a report by Levchenko and Schetinina (1978). They observed that glucose 6-phosphate dehydrogenase activity in liver tissue of dehydrated rats had been inhibited. Glucose 6-phosphate dehydrogenase catalyses the oxidation of glucose 6-phosphate to form 6-phosphogluconic acid, the first intermediate on the pentose monophosphate shunt. This pathway is relatively insignificant in energy metabolism contributing only about 10% of the catabolism of glucose in any circumstances (Coleman, 1980).

There is abundant evidence of the regulatory influence of increased plasma free fatty acids on muscle glycogen utilization rate (Costill *et al.*, 1977b; Rennie *et al.*, 1976). Costill *et al.* (1977b) demonstrated this effect in subjects performing treadmill exercise at 75% maximum oxygen uptake for 30 min. The rate of muscle glycogen depletion was decreased significantly when plasma free fatty acid levels had been experimentally elevated prior to exercise. Increased free fatty acid oxidation produces an accumulation of citrate in skeletal muscle (Garland *et al.*, 1963; Rennie *et al.*, 1976) which subsequently has an inhibitory effect on PFK activity (Garland *et al.*, 1963; Randle *et al.*, 1964).

Subsequent to the investigations reported here, the author participated in a further study that also involved a 24 hour period of dehydration and 90 minutes of rehydration prior to an all-out rowing trial. Preliminary data from this study indicated that plasma free fatty acids were elevated following the 24 hour period of dehydration and remained elevated following rehydration and the rowing trial (unpublished observation). It is likely that such a rise in plasma free fatty acids could be attributed to cortisol as it is well known that stress leads to an elevation of plasma cortisol levels (McMahon *et al.*, 1988; Tepperman, 1980). Cortisol enhances lipolysis (Feldman and Loose, 1977; Leboeuf *et al.*, 1962) predominantly via its permissive effect on adrenaline (Exton *et al.*, 1969; Schaeffer *et al.*, 1969; Shafrir and Steinberg, 1960). As dehydration is a physiological stress, it is reasonable to expect that plasma cortisol levels would be elevated in this state. Indeed, it has been observed in thermally

dehydrated subjects that plasma cortisol levels did increase significantly and that this phenomenon was reversed following the administration of fluids (Brandenberger *et al.*, 1986; Francis, 1979).

Thus, in the present study, it is likely that plasma free fatty acids were increased following dehydration as a result of elevated cortisol levels. The resultant increase in fatty acid acid oxidation would have produced an accumulation of citrate in the muscle that inhibited PFK and subsequently inhibited glycolysis.

CHAPTER 8

SUMMARY AND CONCLUSIONS

The protocol of dehydration over 24 hours resulted in a 3.72 ± 0.18 weight loss of body in the saline groups and a 5.16 ± 0.14% loss of bodyweight in the rowers. Despite the different weight losses experienced by the two groups, the decrease in relative plasma volume was not significantly different. It is possible that differences in training status may have contributed to this effect. During the dehydration period, urinary excretion of sodium and water decreased; the reabsorption of sodium being a fundamental mechanism in the retention of fluid by the kidneys. Concurrently, urinary excretion of potassium was observed to increase, although not significantly. Sodium reabsorption is activated by aldosterone mediated sodium/potassium pumps in the distal convoluted tubule. The reabsorption of sodium, therefore, requires a certain loss of potassium.

The ingestion of a 0.3% saline solutions had a significantly greater effect on plasma volume restoration compared to water, 0.1% and 0.2% saline solutions. No drink, however, fully restored plasma volume to euhydration levels in the 90 minute rehydration period, leaving all subjects with a significant plasma volume deficit.

The dehydration - rehydration protocol was associated with a significant decrease in maximum physical working capacity in competitive rowers. The dehydrated subjects took significantly longer to complete a preset work target of 4200 flywheel revolutions with 3 kg resistance on a Gjessing rowing ergometer. A significant relationship was established between the deficit in plasma volume after rehydration and rowing performance. The effects of dehydration on performance appeared to be minimized in the subjects whose plasma volume deficits were restored to within 4% of euhydrated levels after rehydration.

There were no differences in maximum oxygen consumption, peak ventilation rate nor maximum heart rate between the dehydrated and normal rowing trials despite the average lower work rate during the dehydrated trial.

Significant in the present study, was the observation that the muscle glycogen utilization rate during exhaustive exercise in dehydration was significantly lower. The dehydrated trial was also accompanied by a significantly lower blood lactate level and respiratory exchange ratio. It was apparent that even though muscle glycogen was available, during the dehydrated trial, it was not able to be used as rapidly and subsequently the capacity to perform work decreased. Several mechanisms were proposed to explain this phenomenon with the most likely being the effect of elevated cortisol levels on plasma free

fatty acid levels and the resultant inhibition of PFK by citrate. Further research is currently underway to examine in more detail the effect of dehydration on carbohydrate metabolism.

The decrease in physical work capacity during exhaustive exercise in dehydration can be attributed to two major variables: a) a decrease in plasma volume, and an inability to rapidly regain the lost plasma volume during rehydration; and b) a decrease in the capacity to utilize glycogen.

APPENDIX A

INFORMED CONSENT STATEMENTS

FOOTSCRAY INSTITUTE OF TECHNOLOGY

STANDARD CONSENT FORM FOR SUBJECTS INVOLVED IN EXPERIMENTS

1. CERTIFICATION BY SUBJECT

I, (please print)

of

certify that I have the legal ability to give valid consent and that I am voluntarily giving my consent to participate in the experiment entitled:

THE EFFECT OF 24 HOURS OF DEHYDRATION ON PLASMA VOLUME AND URINARY VARIABLES AND THE EFFECT OF REHYDRATION ON THE RESTORATION OF PLASMA VOLUME

being conducted at Footscray Institute of Technology by:

CAROLINE M. BURGE and supervised by Dr MICHAEL F. CAREY and Dr WARREN R. PAYNE.

I certify that the objectives of the experiment, together with any risks to me associated with the procedures listed hereunder to be carried out in the experiment, have been fully explained to me by CAROLINE BURGE and that I freely consent to participation involving the use on me of these procedures.

PROCEDURES: See attached sheet.

I certify that I have had the opportunity to have my questions answered and that I can withdraw from this experiment at any time and that this withdrawal will not jeopardise me in any way.

Signed:)Date:

Witness:)Date:

2. CERTIFICATION BY PRINCIPAL INVESTIGATOR

I, CAROLINE M. BURGE have explained fully the objectives, risks and procedures of the abovenamed experiment to the subject named herein.

Signed:) Date:)

NOTES:

1. Those signing this form are reminded that while research workers have a duty to advance knowledge by research, the rights of the individual subject take precedence over expected benefits to knowledge or to the community.

2. The experimenter is reminded of the need to observe confidentiality, when appropriate, to protect the interests of subjects.

3. Subjects who are employees of the Institute should be advised that participation in the experiment does not affect in any way their entitlement or right to receive workers' compensation.

CARDIOVASCULAR AND RENAL RISK FACTOR QUESTIONNAIRE

In order to be eligible to participate in the abovenamed experiment you are required to complete the following questionnaire which is designed to assess the risk of you having a cardiovascular event or renal problems occuring during the experimental period.

Name:_____ Date:_____

Age: _____ years Height:_____ cms

Weight: _____ kgs

Give a brief description of your average activity pattern in the past 12 months

_____

_____

Please circle appropriate response:

1. Are you overweight? Yes No Don't know

2. Do you smoke? Yes No

3. Are you a diabetic? Yes No Don't know

4. Have you ever had any kidney problems? Yes No

   If yes, please elaborate _____

5. Do you have high blood pressure? Yes No Don't know

6. Are you on any medication? Yes No

   If yes, what is the medication _____

7. Has you family a history of premature cardiovascular problems (eg heart attack, stroke)? Yes No Don't know

8. Do you have any medical complaint or any other reason which you know of which you think may prevent you from participating in this experiment? Yes No

   If so please elaborate _____

9. Have you had rheumatic fever? Yes No

I, _____ believe that the answers to these questions are true and correct.

Signed:_____ Date:_____

PROCEDURES - NON ROWING GROUP

This experiment will require you to lose as much weight as possible (up to a limit of 5% of bodyweight) in 24 hours. Following the weight loss period, you will be rehydrated for 90 minutes with a drink whose composition is known only to the experimenter.

The experimental period will commence on Thursday morning on _____/_____/19_____ at 9 am. For the next 24 hours please do not perform any heavy work, training or labour. Please ensure also that you are adequately hydrated (particularly if it is a hot day). Please do not eat anything after 4 a.m. but drink 250 ml of fruit juice at 7 a.m. on Friday morning.

Friday 9 a.m.: You will be required to come to Footscray Institute of Technology Human Performance Laboratory, Level 4, Building L, where you will be accurately weighed in private with no clothes on. Prior to the weighing, you will be asked to completely empty your bladder and to void your bowels if possible. Following weighing, you will lie down for 15 minutes after which a small blood sample will be taken from a vein in your arm. After that, your body composition will be determined using the pinchfold test (which is a completely painless procedure). Before you leave, you will be given a 2 litre bottle into which you will pass all of your urine for the next 24 hours. Please try to keep this bottle refrigerated as much as possible while it is in your possession. You will be required to keep an accurate diary of your food intake and activities during this time.

For the next 24 hours, please try not to drink or to eat any succulent fruit or vegetables. Small amounts of bread or dry biscuits are permissible. Stay warmly dressed all day. In the afternoon, wrap yourself in warm clothing that is also preferably windproof and exercise lightly for a couple of hours. This will encourage sweating. Try to have lost at least 3% of your bodyweight or more by this time. Do not exercise after 7 p.m. Remain warmly dressed

Saturday 7:00 a.m.: Please report back to the Human Performance Laboratory. Please also bring the 2 litre urine bottle with you. If less than 3% of body weight has been lost, you may be required to exercise for a further half hour.

Saturday 9 a.m.: Completely empty your bladder into urine bottle for the final time and then give the bottle to the experimenter. Your weight will then be accurately determined again. Following weighing, a blood sample will be taken after you have been supine for 15 minutes. Following the blood sample, you will be given to drink 250 ml of cold fluid every 15 minutes for 90 minutes. Immediately after having consumed the sixth and last drink, please lie down for another fifteen minutes after which a final blood sample will be taken.

During the 24 hour period of fluid restriction, you may experience a slight headache, feelings of fatigue and shortened concentration span. For that reason it is advisable not to plan anything of importance on Friday night. You will also experience a period of intense thirst, however, this should decrease substantially after a while.

The use of venepuncture (puncturing of a vein) can lead to the possibility of bruising and/or infection. The use of sterilized disposable syringes and needles, swabs etc., however, will markedly reduce the possibility of infection. The use of experienced and qualified staff will reduce the likelihood of bruising as bruising is primarily caused by poor venepuncture technique. Although the probability of infection and significant bruising is quite small, if by chance it does eventuate, we suggest you consult with your doctor immediately.

FOOTSCRAY INSTITUTE OF TECHNOLOGY

STANDARD CONSENT FORM FOR SUBJECTS INVOLVED IN EXPERIMENTS

1. CERTIFICATION BY SUBJECT

I, (please print)

of

certify that I have the legal ability to give valid consent and that I am voluntarily giving my consent to participate in the experiment entitled:

THE INFLUENCE OF DEHYDRATION AND SUBSEQUENT REHYDRATION ON ROWING ERGOMETER PERFORMANCE

being conducted at Footscray Institute of Technology by:

CAROLINE M. BURGE and supervised by Dr MICHAEL F. CAREY and
Dr WARREN R. PAYNE.

I certify that the objectives of the experiment, together with any risks to me associated with the procedures listed hereunder to be carried out in the experiment, have been fully explained to me by CAROLINE BURGE and that I freely consent to participation involving the use on me of these procedures.

PROCEDURES: See attached sheet.

I certify that I have had the opportunity to have my questions answered and that I can withdraw from this experiment at any time and that this withdrawal will not jeopardise me in any way.

Signed:)Date:

Witness:)Date:

2. CERTIFICATION BY PRINCIPAL INVESTIGATOR

I, CAROLINE M. BURGE have explained fully the objectives, risks and procedures of the abovenamed experiment to the subject named herein.

Signed:) Date:)

NOTES:

1. Those signing this form are reminded that while research workers have a duty to advance knowledge by research, the rights of the individual subject take precedence over expected benefits to knowledge or to the community.

2. The experimenter is reminded of the need to observe confidentiality, when appropriate, to protect the interests of subjects.

3. Subjects who are employees of the Institute should be advised that participation in the experiment does not affect in any way their entitlement or right to receive workers' compensation.

CARDIOVASCULAR AND RENAL RISK FACTOR QUESTIONNAIRE

In order to be eligible to participate in the abovenamed experiment you are required to complete the following questionnaire which is designed to assess the risk of you having a cardiovascular event or renal problems occuring during the experimental period.

Name:_____ Date:_____

Age: _____ years Height:_____ cms

Weight: _____ kgs

Give a brief description of your average activity pattern in the past 12 months

_____

_____

Please circle appropriate response:

1. Are you overweight? Yes No Don't know

2. Do you smoke? Yes No

3. Are you a diabetic? Yes No Don't know

4. Have you ever had any kidney problems? Yes No
   If yes, please elaborate _____

5. Do you have high blood pressure? Yes No Don't know

6. Are you on any medication? Yes No
   If yes, what is the medication _____

7. Has you family a history of premature cardiovascular problems (eg heart attack, stroke)? Yes No Don't know

8. Do you have any medical complaint or any other reason which you know of which you think may prevent you from participating in this experiment? Yes No
   If so please elaborate _____

9. Have you had rheumatic fever? Yes No

I, _____ believe that the answers to these questions are true and correct.

Signed:_____ Date:_____

MUSCLE BIOPSY QUESTIONNAIRE

Name:_____

Address:_____

1. Have you or your family suffered from any tendency to excessively bleed (eg haemophilia)?

Yes No Don't know

If yes, please elaborate_____

2. Are you allergic to local anaesthetic?

Yes No Don't know

If yes, please elaborate_____

3. Do you have any skin allergies?

Yes No Don't know

If yes, please elaborate_____

4. Have you any allergies that should be made known?

Yes No Don't know

If yes, please elaborate_____

5. Are you currently on any medication?

Yes No

If yes, what is the medication?_____

6. Do you have any other medical problems that should be made known?

Yes No Don't know

If yes, please elaborate_____

I, _____ believe that the answers to these questions are true and correct.

Signed:_____ Date:_____

PROCEDURES - ROWING GROUP DEHYDRATION PROTOCOL

This experiment will require you to lose 5% of your bodyweight in 24 hours. Following the weight loss period, you will be rehydrated for 90 minutes with a drink whose composition is known only to the experimenter.

The experimental period will commence on Friday morning on _____/_____/19___ at 9 am. For the next 24 hours please do not perform any heavy work, training or labour. Please ensure also that you are adequately hydrated (particularly if it is a hot day). Please do not eat anything after 4 a.m. but drink 250 ml of fruit juice at 7 a.m. on Saturday morning.

Saturday 9 a.m.: You will be required to come to Footscray Institute of Technology Human Performance Laboratory, Level 4, Building L, where you will be accurately weighed in private with no clothes on. Prior to the weighing, you will be asked to completely empty your bladder and to void your bowels if possible. Following weighing, you will lie down for 15 minutes after which a small blood sample will be taken from a vein in your arm. After that, your body composition will be determined using the pinchfold test (which is a completely painless procedure). You will be required to keep an accurate diary of your food intake and activities during this time.

For the next 24 hours, please try not to drink or to eat any succulent fruit or vegetables. Small amounts of bread or dry biscuits are permissible. Stay warmly dressed all day. In the afternoon, wrap yourself in warm clothing that is also preferably windproof and exercise lightly for a couple of hours. This will encourage sweating. Try to have lost at least 4% of your bodyweight or more by this time. Do not exercise after 7 p.m. Remain warmly dressed

Sunday 7:00 a.m.: Please report back to the Human Performance Laboratory. If less than 4.5% of body weight has been lost, you may be required to exercise for a further half hour in sweat gear.

Sunday 9 a.m.: Your weight will be accurately determined again. Following weighing, a blood sample will be taken after you have been supine for 15 minutes. Following the blood sample, you will be given to drink 250 ml of cold fluid every 15 minutes for 90 minutes. Immediately after having consumed the sixth and last drink, please lie down for another fifteen minutes after which a final blood sample will be taken. After the blood sample has been taken, a muscle biopsy sample will be obtained.

You will then be permitted to warm up lightly on the rowing ergometer for ten minutes. During the warm up you may have three hard bursts lasting no more than 10 seconds. You will also be fitted with a headpiece, mouthpiece and noseclip to enable all exhaled air to be analysed. A heart rate monitor will be fitted around your chest also.

After completion of the warmup, the trial will commence using a formal start command. Pace yourself accordingly as for a 2000m race. Your aim is to reach 4200 revolutions as fast as possible. Immediately after the trial, another muscle biopsy will be taken. Five minutes later, a final blood sample will be obtained.

The use of venepuncture (puncturing of a vein) can lead to the possibility of bruising and/or infection. The use of sterilized disposable syringes and needles, swabs etc., however, will markedly reduce the possibility of infection. The use of experienced and qualified staff will reduce the likelihood of bruising as bruising is primarily caused by poor venepuncture technique. Although the probability of infection and significant bruising is quite small, if by chance it does eventuate, we suggest you consult with your doctor immediately.

The muscle biopsy is a relatively painless procedure that is used to obtain small samples of skeletal muscle tissue for metabolic analysis. A small incision is made in the skin overlying the muscle, under local anaesthetic. The biopsy needle is then inserted into the muscle and a small piece of tissue removed from the muscle. During this part of the procedure you may feel some pressure and a tendency for the muscle to cramp, however, this only persists for a few seconds. Following the biopsy the incision will be closed and a pressure bandage applied for 24 hours. It is common for subjects to experience some mild soreness in the muscle over the next 2-3 days, however this does pass and does not restrict movement. In some rare cases mild haematomas have been reported, but these symptoms disappear within a week. The whole procedure will be performed under sterile conditions by a qualified medical practitioner.

APPENDIX B

DETAILS OF ANALYTICAL CHEMICAL METHODS

B.1 BLOOD LACTATE

Principle:- The enzyme lactate dehydrogenase (LDH) catalyses the the transfer of hydrogen from the cofactor NADH to pyruvic acid to produce lactic acid. However, the equilibrium highly favours the reduction of pyruvic acid:

$$\text{Lactic acid} + NAD^{+} \xrightleftharpoons{LDH} \text{Pyruvic acid} + NADH$$

$$K_{eq} = 3.6 \times 10^{11}\ M^{-1}$$

In the presence of an excess of the cofactor NAD+ and lactate dehydrogenase activity, and at a pH of 9.6, nearly all of the lactate, in deproteinized whole blood, is converted to pyruvate. To ensure the reaction goes to completion, pyruvic acid, in the presence of hydrazine, is removed by converting it to pyruvatehydrazone. The appearance of NADH yields an observable method to quantify the concentration of lactate originally present. The concentration of NADH was measured spectrophotometrically at wavelength of 340 nm using a spectrophotometer (Shimadzu UV-120-02) in conjunction with a flow cell apparatus (Sigma Technical Bulletin, No 826 UV, Oct 1986).

A set of standards ranging in concentration from 1 mM to 20 mM was run with each analysis in conjunction with a reagent blank. A typical standard curve is shown in Figure B.1. Each point is based on the average of four measurements at each concentration level.

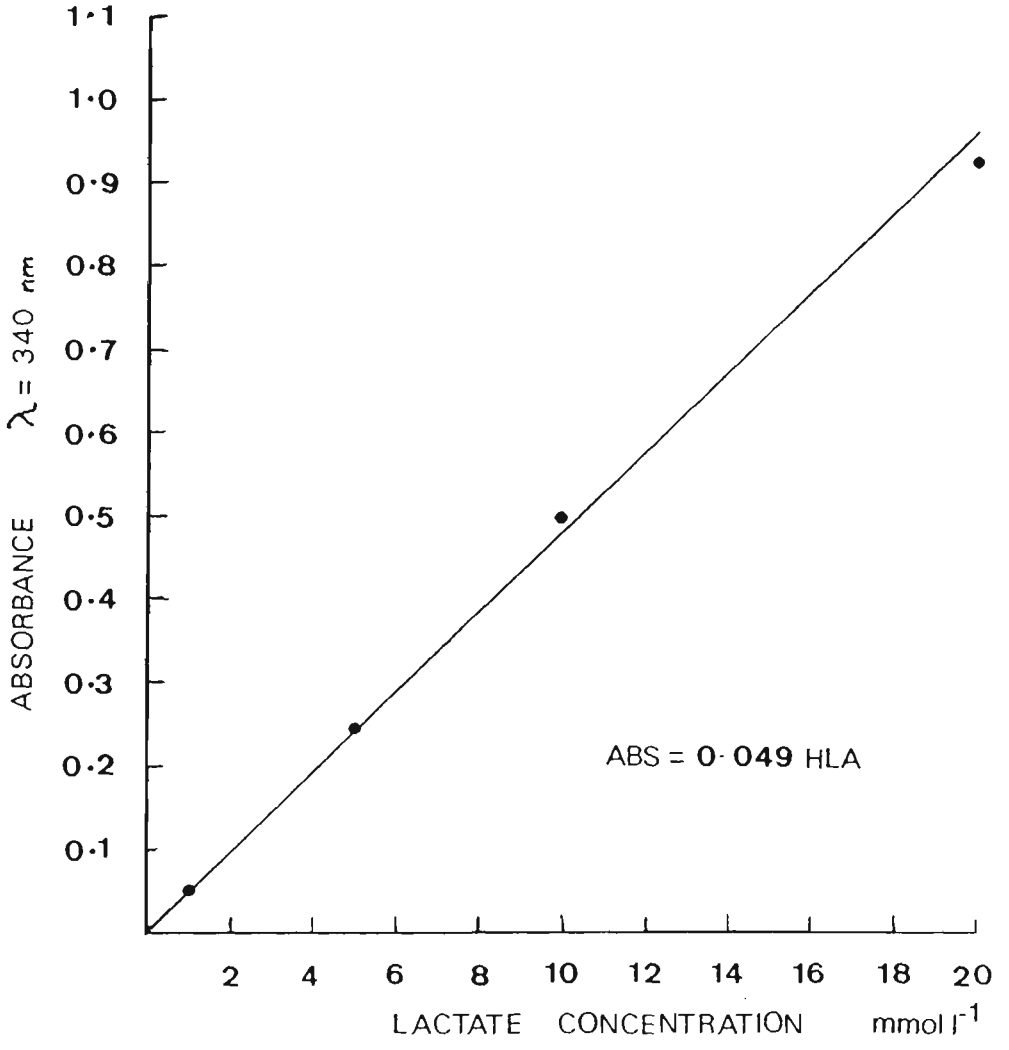


Figure B.1

Standard curve for the determination of blood lactate concentration

B.2 HAEMOGLOBIN

Principle:- When placed in a solution of potassium ferricyanide-potassium cyanide, haemoglobin is oxidized to methaemoglobin and converted to cyanmethaemoglobin. The concentration of cyanmethaemoglobin can be determined spectrophotometrically at a wavelength of 540 nm and is proportional to the concentration of haemoglobin originally present (Sigma: Procedure No 525, 1984). A set of standards ranging in concentration from 6.0 g% to 18.0 g% was run with each analysis. The haemoglobin standard was supplied by Sigma Diagnostics (Catalogue No. 525-18). A typical standard curve is shown in Figure B.2.

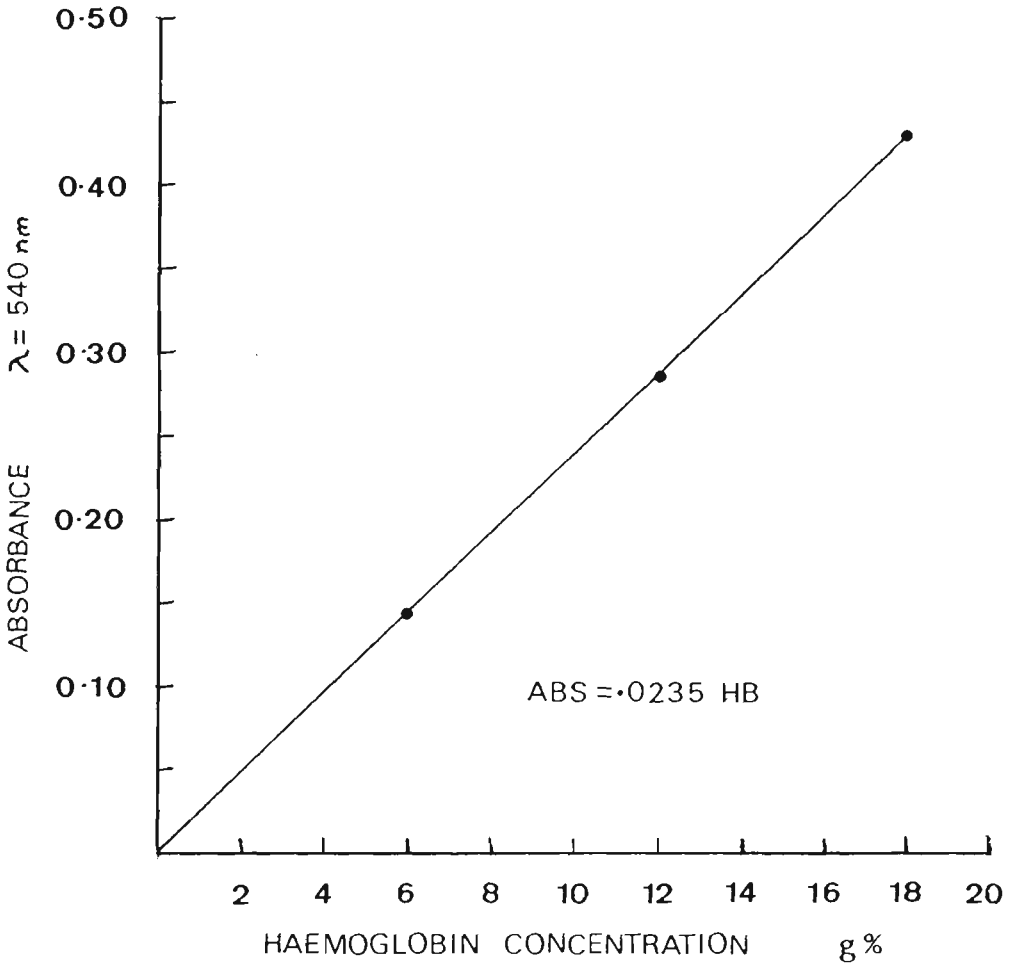


Figure B.2

Standard curve for the determination of haemoglobin concentration

B.3 MUSCLE GLYCOGEN

Principle:- Glycogen is a macromolecule consisting of alpha 1-4 and alpha 1-6 linkages of glucose residues. Hydrolysis of the glycogen macromolecule yields free glucose residues. The free glucose residues can be assayed through the following series of reactions:

$$\text{glucose} + \text{ATP} \xrightarrow{\text{hexokinase}} \text{glucose 6-phosphate} + \text{ADP}$$

$$\text{glucose 6-phosphate} + \text{NADP}^{+} \xrightarrow[\text{dehydrogenase}]{\text{glucose 6-phosphate}} \text{6-phospho-gluconolactone} + \text{NADPH}$$

The glucose is phosphorylated to glucose 6-phosphate and in the presence of excess NADP+ and glucose 6-P dehydrogenase yields 6-phosphogluconolactone. The appearance of NADPH is observed fluorimetrically at a wavelength of 365 nm absorption and 455 nm emission. The fluorimeter was calibrated with a solution of NADH and the response to a selection of standard dilutions was linear. Hence, a reagent blank and a single standard could be run for each analysis provided the same conditions were used as those during the calibration.

B.4 URINARY SODIUM AND POTASSIUM CONCENTRATION

The analysis for Na+ and K+ were performed separately using flame emission spectrophotometry. A set of standards ranging in concentration from 0 ppm to 10 ppm of Na+ or K+ respectively was run with each analysis. The standard curves used for each of the analyses are provided in Figures B.3 and B.4. The relative standard deviation (RSD) for multiple measurements of each aqueous standard for the potassium concentration determination ranged between 0.591% (6 ppm, n=10) and 0.97% (2 ppm, n=10). The RSD of each aqueous standard for the sodium concentration ranged between 1.23 (4ppm, n=10) and 1.97 (6ppm, n=11).

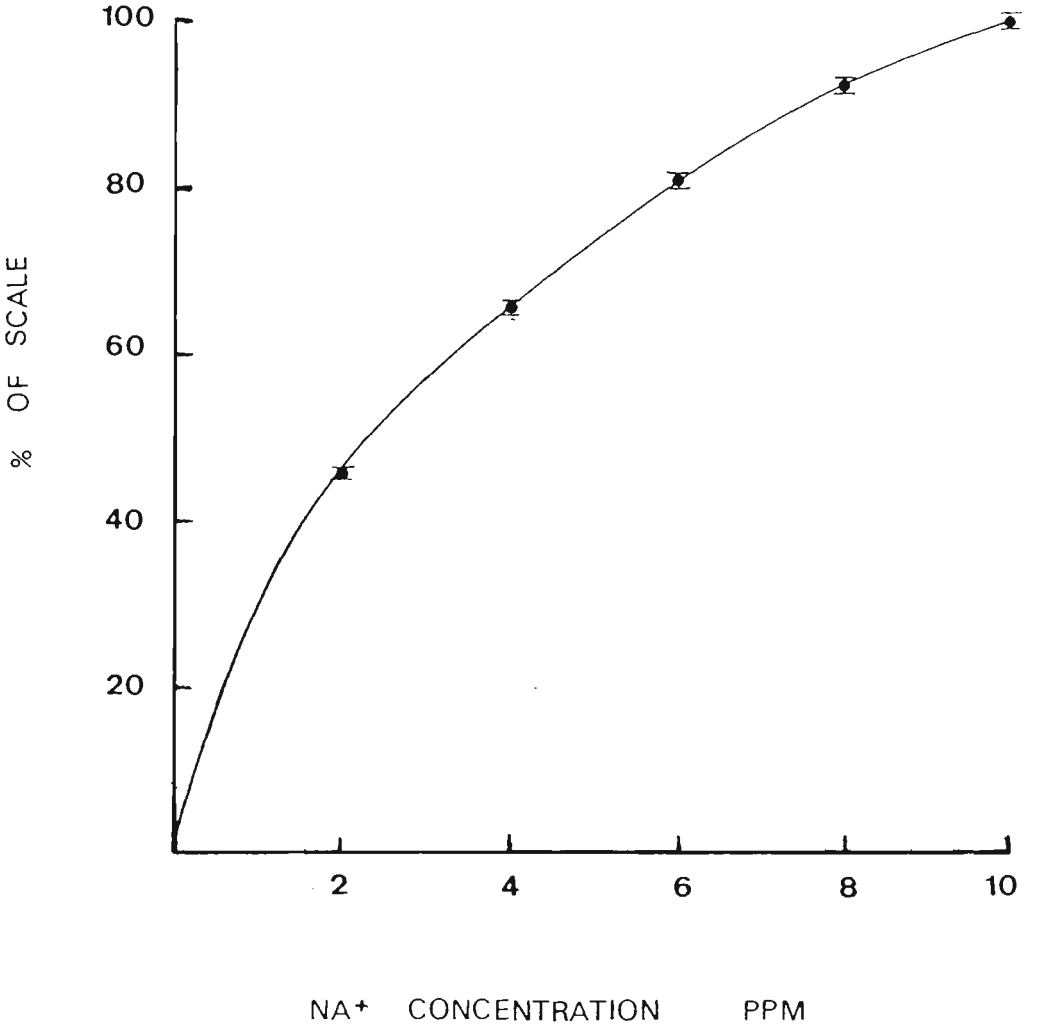


Figure B.3

Standard curve for the determination of Na^+ concentration

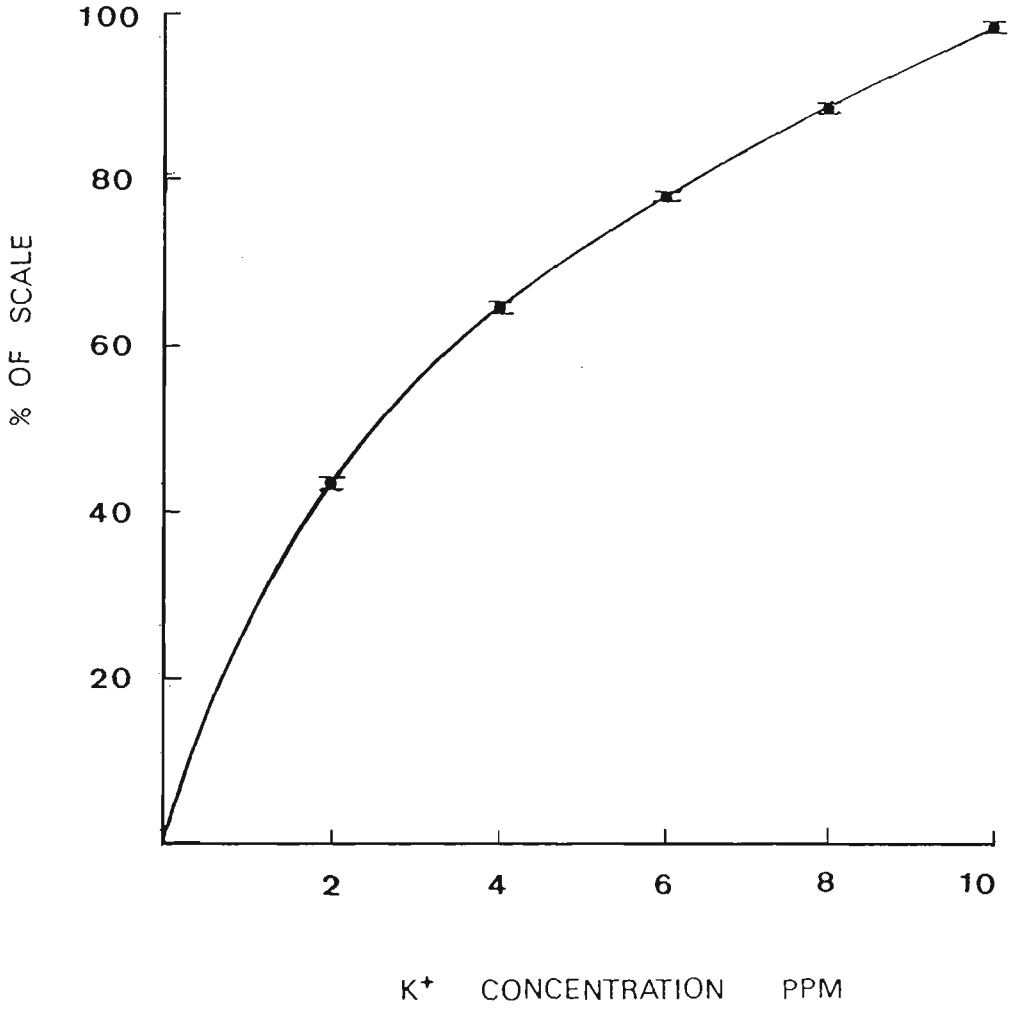


Figure B.4

Standard curve for the determination of K^+ concentration

APPENDIX C

STATISTICAL TABLES

C.1 NUMBER OF FLYWHEEL REVOLUTIONS VERSUS TIME AND HYDRATION

i) ANOVA

	df	MS	F	P
Time (A)	11	1.736E7	1391	0.000
Error	77	9.603E5		
Hydratn (B)	1	4.952E5	9.70	0.017
Error	7	3.572E5		
A x B	11	1.505E4	3.53	0.000
Error	77	4267		

ii) SIMPLE MAIN EFFECTS

TIME	df	MS	MSerror	F	P
0:30	1	3630.1	580.8	6.25	0.014*
1:00	1	689.1	2128.0	0.32	0.587
1:30	1	5112.3	2782.2	1.84	0.217
2:00	1	12656.3	3195.5	3.96	0.087
2:30	1	21025.0	3603.6	5.83	0.046*
3:00	1	97656.2	20742.0	4.71	0.067
3:30	1	40501.6	4626.6	8.75	0.021*
4:00	1	52900.0	5000.0	10.58	0.014*
4:30	1	71155.6	6309.1	11.28	0.012*
5:00	1	77006.2	7892.0	9.76	0.017*
5:30	1	90300.3	9814.5	9.20	0.019*
6:00	1	188139.1	31303.3	6.01	0.044*
Error	7				

C.2 NUMBER OF NONCUMULATIVE FLYWHEEL REVOLUTIONS VERSUS TIME AND HYDRATION

i) ANOVA

	df	MS	F	P
Time (A)	11	12424.3	13.6	0.000
Error	11	913.6		
Hydratn (B)	1	4485.3	2.96	0.129
Error	7	1512.9		
A x B	11	695.7	1.22	0.285
Error	77	568.1		

ii) SIMPLE MAIN EFFECTS

TIME	df	MS	MSerror	F	P
0:30	1	552.3	783.2	0.71	0.429
1:00	1	3690.6	4269.1	0.86	0.383
1:30	1	410.1	619.3	0.66	0.443
2:00	1	1827.6	129.0	14.17	0.007*
2:30	1	900.0	39.3	22.91	0.002*
3:00	1	400.0	171.4	2.33	0.171
3:30	1	1501.6	119.4	12.57	0.009*
4:00	1	52900.0	5000.0	10.58	0.014*
4:30	1	826.6	230.1	3.59	0.099
5:00	1	689.1	242.6	2.84	0.136
5:30	1	264.1	306.9	0.86	0.384
6:00	1	451.6	162.3	2.78	0.139
Error	7				

C.3 CHANGES IN PLASMA VOLUME - SALINE GROUPS

i) ANOVA

	df	MS	F	P
Drink (A)	2	8.830	1.24	0.3160
Error	15	7.097		
PV (B)	3	534.254	217.68	0.000
A x B	6	27.647	6.36	0.000
A x B	45	2.454		

ii) SIMPLE MAIN EFFECTS

	df	MS	MSerror	F	P
0.1%	3	238.584	2.454	97.21	0.000*
0.2%	3	149.185	2.454	60.79	0.000*
0.3%	3	222.597	2.454	90.70	0.000*
Error	45				
PV1	2	8.895	7.070	1.26	0.312
PV2	2	45.570	1.610	28.30	0.000*
PV3	2	68.533	5.779	11.86	0.000*
PV0	2	0.000	0.000	1.00	1.000
Error	15				

iii) NEWMAN-KEULS POST HOC ANALYSIS

LEVEL	DRINK		0.1%	0.2%	0.3%	r	q(r,s)	cv
PV2		Mean	4.802	6.425	10.175			
	0.1%	4.802		1.623	5.373*	3	3.67	1.90
	0.2%	6.425			3.750*	2	3.01	1.56
	0.3%	10.175						

LEVEL	DRINK		0.1%	0.2%	0.3%	r	q(r,s)	cv
PV3		Mean	3.097	5.772	9.810			
	0.1%	3.097		2.675	6.713*	3	3.67	3.60
	0.2%	5.772			4.038*	2	3.01	2.95
	0.3%	9.810						

LEVEL	PV		PV0	PV2	PV3	PV1	r	q(r,s)	cv
0.1%		Mean	0.000	4.802	9.817	14.612			
	PV0	0.000		4.802*	9.817*	14.612*	4	3.96	2.53
	PV2	4.802			5.015*	9.810*	3	3.58	2.29
	PV3	9.810				4.802*	2	2.95	1.89
	PV1	14.612							

Table C.3 continued.

LEVEL	PV		PV0	PV3	PV2	PV1	r	q(r,s)	cv
0.2%		Mean	0.000	5.772	6.425	12.197			
	PV0	0.000		5.772*	6.425*	12.197*	4	3.96	2.53
	PV3	5.772			0.650	6.425*	3	3.58	2.29
	PV2	6.425				5.772*	2	2.95	1.89
	PV1	12.197							

LEVEL	PV		PV0	PV3	PV2	PV1	r	q(r,s)	cv
0.3%		Mean	0.000	3.097	10.175	13.133			
	PV0	0.000		3.097*	10.175*	13.133*	4	3.96	2.53
	PV3	3.097			7.078*	10.036*	3	3.58	2.29
	PV2	10.175				2.958*	2	2.95	1.89
	PV1	13.133							

C.4 CHANGES IN PLASMA VOLUME - ROWERS

i) ONEWAY ANOVA

	df	MS	F	P
Time	3	209.87	39.54	0.000*
Error	28	5.30		

ii) NEWMAN-KEULS MULTIPLE COMPARISON

LEVEL			PV0	PV2	PV3	PV1	r	q(r,s)	cv
PV		Mean	0.000	6.020	6.516	12.536			
	PV0	0.000		6.020*	6.516*	12.536*	4	3.86	3.14
	PV2	6.020			0.496	6.516*	3	3.49	2.84
	PV3	6.516				6.020*	2	2.90	2.36
	PV1	12.536							

C.5 RESTORATION OF PLASMA VOLUME - 0.1%, 0.2%, 0.3% SALINE AND WATER

i) ONEWAY ANOVA

	df	MS	F	P
Time	3	32.648	15.81	0.000*
Error	22	2.06		

ii) NEWMAN-KEULS MULTIPLE COMPARISON

LEVEL			0.1%	Water	0.2%	0.3%	r	q(r,s)	cv
DRINK		Mean	4.802	6.020	6.425	10.173			
	0.1%	4.802		1.218	1.623	5.371*	4	3.92	2.30
	Water	6.020			0.405	4.153*	3	3.54	1.94
	0.2%	6.425				3.751*	2	2.93	1.71
	0.3%	10.173							

C.6 SUM OF SKINFOLDS - ROWERS AND SALINE GROUPS

ONEWAY ANOVA

	df	MS	F	P
Group	3	210.28	0.589	0.633
Error	22	361.38		

C.7 HEIGHT - ROWERS AND SALINE GROUPS

ONEWAY ANOVA

	df	MS	F	P
Group	3	57.83	1.260	0.320
Error	17	45.90		

C.8 AGE - ROWERS AND SALINE GROUPS

ONEWAY ANOVA

	df	MS	F	P
Group	3	4.60	0.468	0.707
Error	22	9.82		

C.9 PRE-DEHYDRATION BODYWEIGHT - ROWERS AND SALINE GROUPS

ONEWAY ANOVA

	df	MS	F	P
Group	3	40.85	0.713	0.555
Error	22	57.30		

C.10 POST-DEHYDRATION BODYWEIGHT - ROWERS AND SALINE GROUPS

ONEWAY ANOVA

	df	MS	F	P
Group	3	33.12	0.641	0.596
Error	22	51.64		

C.11 % LOSS OF BODYWEIGHT - ROWERS AND SALINE GROUPS

i) ONEWAY ANOVA

	df	MS	F	P
Group	3	4.056	9.015	0.000*
Error	22	0.450		

ii) NEWMAN-KEULS MULTIPLE COMPARISON

		0.2%	0.1%	0.3%	ROWERS	r	q(r,s)	cv
	Mean	3.453	3.755	3.965	5.157			
0.2%	3.453		0.302	0.512	1.704*	4	3.93	1.08
0.1%	3.755			0.210	1.402*	3	3.54	0.70
0.3%	3.965				1.192*	2	3.92	1.07
ROWERS	5.157							

C.12 PRE AND POST TRIAL LACTATE CONCENTRATION VERSES HYDRATION

i) ANOVA

	df	MS	F	P
Time (A)	1	433.40	89.67	0.000
Error	6	4.83		
Hydratn (B)	1	10.93	5.66	0.055
Error	6	1.93		
A x B	1	7.48	8.10	0.0298
Error	6	0.92		

ii) SIMPLE MAIN EFFECTS

	df	MS	MSerror	F	P
Pre	1	0.165	0.499	0.33	0.586
Post	1	18.240	2.354	7.75	0.038*
Error	6				
Normal	1	227.324	3.627	76.45	0.000*
Dehyd	1	163.544	2.127	76.87	0.000*
Error	6				

C.13 PRE AND POST TRIAL GLYCOGEN CONCENTRATION VERSES HYDRATION

i) ANOVA

	df	MS	F	P
Time (A)	1	206417.7	139.1	0.000*
Error	6	1484.1		
Hydratn (B)	1	227358.3	35.15	0.001*
Error	6	6469.1		
A x B	1	7113.7	19.81	0.004*
Error	6	359.1		

ii) SIMPLE MAIN EFFECTS

	df	MS	MSerror	F	P
Pre	1	157452.4	2867.6	54.91	0.000*
Post	1	77019.6	3960.6	19.45	0.004*
Error	6				
Normal	1	145085.4	7270.0	119.74	0.000*
Dehyd	1	68446.1	631.6	108.36	0.000*
Error	6				

C.14 REGRESSION ANALYSIS FOR PV1 VERSUS INCREASE IN TRIAL TIME

ANOVA

	df	MS	F	P
Regression	1	0.6498	18.75	0.005*
Residual	6	0.0346		

C.15 REGRESSION ANALYSIS FOR PV3 VERSUS INCREASE IN TRIAL TIME

ANOVA

	df	MS	F	P
Regression	1	0.7996	37.88	0.000*
Residual	6	0.0211		

APPENDIX D

DIARIES OF THE INDIVIDUAL SUBJECTS DURING THE 24 HOUR DEHYDRATION PERIOD

DIET AND ACTIVITY DIARIES OF THE ROWERS

#1

Saturday am weigh in:

FOOD	ACTIVITY
1 piece toast	80 min light rowing
Half bowl bran flakes/cornflake mix	60 min light rowing
150 ml water and powdered skim milk	40 min cycle with sweat gear
2 pieces toast	
dry branflakes/cornflakes mix	
2 spoon skim milk powder	
Sunday morning am:	
	25 min cycle/run on exercise bike/treadmill
Sunday morning weigh in:	
90 min rehydration with water	
rowing trial.	

****

#2

Saturday am weigh in:

FOOD	ACTIVITY
1 apple	5 km jog with sweat gear
2 muesli bars	5 km jog with sweat gear
1 slice bread	
sultanas on bread	
1 orange	
1 muesli bar	
Sunday morning am weigh in:	
90 min rehydration with water	
rowing trial.	

****

#3

Saturday am weigh in:

FOOD	ACTIVITY
2 pieces of toast	1.25 hours light row
2 oranges	30 min run in sweat gear
1 serve plain pasta (200g)	
200 ml water	

Sunday morning am weigh in:
90 min rehydration with water
rowing trial.

****

#4

Saturday am weigh in:

FOOD	ACTIVITY
Half glass water	30 mins run with sweat gear
small piece fish	30 mins run with sweat gear
2 slices bread	
scrape of butter	
tuna sandwich	
1 mandarine	

Sunday morning am weigh in:
90 min rehydration with water
rowing trial.

****

#5

Saturday am weigh in:

FOOD	ACTIVITY
2 teaspoons apricot jam	40 min row
1 Violet Crumble	40 min row
1 Mars Bar	30 min cycle with sweat gear
1 Snickers	
1 Bowl rice with Soy Sauce	
1 glass water	

Sunday morning am weigh in:
90 min rehydration with water
rowing trial.

****

#6

Saturday am weigh in:

FOOD	ACTIVITY
250 ml skim milk	35 min run with sweat gear
2 pieces bread	40 min light row
300 g noodles	40 min light row
1 piece bread	
1 apple	

Sunday morning am weigh in:
90 min rehydration with water
rowing trial.

****

#7

Saturday am weigh in:

FOOD	ACTIVITY
1 Slice wholegrain toast	long walk shopping
1 tablespoon honey	75 min cycle with sweat gear
2 desert spoons of stewed apple	35 min run/walk/jog with sweat gear.
100 gm non-fat plain yogurt	30 min run/jog/walk with sweat gear.
200 ml water	
2 spoons of apple	
1 spoon sultanas	

Sunday morning am weigh in:
90 min rehydration with water
rowing trial.

****

#8

Saturday am weigh in:

FOOD	ACTIVITY
2 slices bread	30 min cycle with sweat gear
50 ml water	40 min run with sweat gear
small bowl rice	30 min light row
1 apple	
50 ml water	
small bowl rice	

Sunday morning am weigh in:
90 min rehydration with water
rowing trial.

****

DIET AND ACTIVITY DIARIES OF THE POOLED SALINE GROUP

#1

Friday am weigh in:

FOOD	ACTIVITY
50 ml water	40 min run with sweat gear
half biscuit	20 min run with sweat gear
piece bread	

****

#2

Friday am weigh in:

FOOD	ACTIVITY
1 slice bread	90 min racquetball
small sips water	with sweat gear
biscuit	30 min light run
50 ml water	

****

#3

Friday am weigh in:

FOOD	ACTIVITY
1 slice bread	40 min cycle with sweat gear
50 ml water	25 min run with sweat gear
1 apple	

****

#4

Friday am weigh in:

FOOD	ACTIVITY
1 slices bread	20 min cycle with sweat gear
50 ml water	45 min run with sweat gear
small piece c'lope	

#5

Friday am weigh in:

FOOD	ACTIVITY
1 slice bread	75 min squash
50 ml water	45 min run with sweat gear
3 cracker biscuits	
4 grapes	

****

#6

Friday am weigh in:

FOOD	ACTIVITY
1 slice bread	lacross 40 min with jumper
50 ml water	40 min run with sweat gear
2 cracker b'scuit	

****

#7

Friday am weigh in:

FOOD	ACTIVITY
1 slice bread	30 min run with sweat gear
50 ml water	2 hour brisk walk with with sweat gear

****

#8

Friday am weigh in:

FOOD	ACTIVITY
1 slice bread	30 min cycle with sweat gear
50 ml water	40 min run with sweat gear
chewing gum	

#9

Friday am weigh in:

FOOD	ACTIVITY
2 slices bread	2 hours football training
50 ml water	

****

#10

Friday am weigh in:

FOOD	ACTIVITY
2 slices bread	30 min cycle with sweat gear
50 ml water	40 min run with sweat gear

****

#11

Friday am weigh in:

FOOD	ACTIVITY
1 pk fruit tingles	40 min run with sweat gear
50 ml water	30 min run with sweat gear

****

#12

Friday am weigh in:

FOOD	ACTIVITY
1 slice bread	30 min cycle with sweat gear
50 ml water	40 min squash

#13

Friday am weigh in:

FOOD	ACTIVITY
1 hotdog	Took stairs all day at work
1 apple	25 min run with sweat gear

****

#14

Friday am weigh in:

FOOD	ACTIVITY
1 slice bread	2 hour brisk walk in warm gear

****

#15

Friday am weigh in:

FOOD	ACTIVITY
1 slice bread	15 min cycle with sweat gear
50 ml water	40 min run with sweat gear
7 jaffas	

****

#16

Friday am weigh in:

FOOD	ACTIVITY
2 slices bread	70 min cycle with sweat gear
50 ml water	

#17

Friday am weigh in:

FOOD	ACTIVITY
2 slices bread 50 ml water	70 min cycle with sweat gear

****

#18

Friday am weigh in:

FOOD	ACTIVITY
2 slices bread 50 ml water	70 min cycle with sweat gear

****

(Note: Subjects 16, 17 and 18 worked together for the entire experimental period.

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