WHAT ARE THE PHYSIOLOGICAL EFFECTS OF DEHYDRATION ON WORK AND SPORT PERFORMANCE?

Introduction

During prolonged exercise in neutral or slightly warm environments the maintenance of high sweat rates may lead to progressive dehydration which will be accompanied by impaired performance. Mild dehydration will impair exercise capacity and prevent the athlete/worker from making the best use of their skills. In locomotor sports, such as running or cycling, it has been demonstrated that race times at major championship events are generally poor when ambient temperature and humidity are high, and sweat losses are large. Severe dehydration is potentially fatal: exercise in the dehydrated state leads to a rapid elevation in core temperature and heart rate, and the onset of heat illness. An adequate fluid intake before, during and after exercise can help to avoid the negative effects of dehydration. Fluid requirements will depend on work rate, the ambient climatic conditions, and also on individual physiological and biochemical characteristics of the athlete/worker. Of all the physiological perturbations that can cause early fatigue during exercise, dehydration is arguably the most important, if only because the consequences are potentially life threatening.

In an early study it was shown that the amount of fluid lost as sweat was closely correlated with body weight. It is more meaningful, therefore, to express the effect of water deficit upon body temperature as a percentage deficit from the initial body weight, rather than absolute volumes of water deficit. Consequently, one of the most important factors influencing the level to which rectal temperature rises during exercise is the percent decrease in body weight due to the water deficit.

Dehydration limits a wide range of cardiovascular and thermoregulatory responses, culminating in an increase in core temperature of 0.10 - 0.40°C for every 1% decrease in body weight as water. One of the physiological explanations for the impairment in cardiovascular and thermoregulatory responses due to dehydration is the reduction in skin blood flow and sweating rate which inevitably leads to a rise in core temperature, if the exercise intensity is maintained.

Loss of fluid of 1% body weight (600 - 800ml) by sweating may lead to reduced performance. It is has been reported that exercise performance is impaired when an individual is dehydrated by as little as 2% of body weight, and that losses in excess of 5% body weight can decrease the capacity for work by as much as 30%. If the body water deficit exceeds 3% of initial body weight, then, even in cool conditions rectal temperature will rise. If the deficit exceeds 4%, rectal temperature may rise to dangerous levels. It is also generally accepted that cognitive function is also impaired when dehydration and hyperthermia are present. Although there is limited information available, performance in a variety of tests of cognitive function was adversely effected when the level of dehydration, which was induced by exercise in the heat, reached 2% of initial body weight. In addition, indices of sustained attention, error rate, response time, and task accuracy are all negatively affected during heat exposure, even when sedentary. Studies of running and cycling have shown that both sprint and endurance exercise performance may be adversely affected by minimal levels of dehydration.

The danger to health, and even of death, from not drinking adequate amounts of water during exercise, and particularly in the heat, is well documented. Heat exhaustion due to water deficit in men working in hot atmospheres is well recognised by climate physiologists, and the main symptom is that the subject becomes very fatigued and cannot continue to work at the required rate.

In the non exercising condition, a loss of 2% body weight as fluid produces some thirst and oliguria. A loss of 4% body weight (20% of ECF in acute dehydration) causes oliguria, tachycardia, and often postural hypotension. An acute extracellular fluid loss of 6% of body weight is a life threatening event, reducing interstitial fluid and plasma volumes by 30%, and compromising both blood pressure and renal function.

Studies have shown the marked deterioration in the performance of soldiers (n= 30) after a march (25 miles) in desert-like areas, in which water was restricted. The climatic conditions during this study were far from severe, with morning temperatures of 68°F (20°C) dry bulb, and not exceeding 85°F (29.4°C), for the entire study. The soldiers with water restriction had significantly greater heart rates, rectal temperatures and progressively reduced sweating rates as the dehydration progressed, for the same workload.

Insufficient fluid replacement may result in dehydration during exercise. This may impair body heat dissipation and endurance performance as a result of decreased plasma volume, increased plasma osmolality, decreased sweat rate and skin blood flow, and raised core temperature.

Fluid Losses

Fluid losses are distributed in varying proportions among the plasma, extracellular and intracellular water. The decrease in plasma volume which accompanies dehydration may be of particular importance in influencing work capacity; blood flow to muscles must be maintained to provide oxygen and substrates, but a high blood flow to the skin is also necessary to convect heat to the body surface where it can be dissipated. When there is a high heat stress, and blood volume has been reduced by

sweat loss during prolonged exercise, there may be difficulty in meeting the requirement for a high blood flow to both tissues. In this situation, skin blood flow is likely to be compromised, allowing central venous pressure and blood flow to be maintained, but reducing heat loss and causing body temperature to rise.

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Voluntary/Involuntary Dehydration

Numerous investigators report that ad libitum water intake results in incomplete water replacement or voluntary dehydration during exercise and heat exposure.

In one study the stress factors (heat, exercise and dehydration) were separated statistically, and it was concluded that voluntary drinking in a hot environment was 146% greater than that in a cool environment; when a person was hypohydrated, drinking was 109% greater than when hydrated; and drinking during exercise was only 41% greater than that when at rest.

In a normally active subject, drinking begins when body weight (water) is reduced by about 0.8% (600g). The more stressful the environment the greater the water deficit before drinking begins. This delay in drinking has been called voluntary dehydration, but a more appropriate term could be involuntary dehydration or hypohydration, because the delay in drinking is not cognitive. The degree of involuntary dehydration is significantly reduced by four days of heat exposure (8 hours total). Accompanying this reduction is a significant decrease in time to the first drink, a significant increase in the number of drinks, and a significantly greater volume per drink. The body prefers a slightly hyperosmotic hypovolemia which occurs after involuntary dehydration, to a hypoosmotic hypervolemia which occurs after replacing 100% of sweat losses with water. To correct the latter would require increased sweating and urinary losses, with accompanying electrolyte (osmoles) losses, while the former requires only the consumption of pure water. In general, dissolved solutes increase fluid consumption if they are present in hyperosmolar concentrations, and decrease drinking if they are present in hyposmolar concentrations. Hyperosmotic concentrations require a greater volume of water to excrete the additional solute load.

Humans have a delayed period of rehydration after thermal dehydration which has been termed involuntary dehydration. It has been suggested that thirst is primarily a function of the sodium concentration in plasma rather than the plasma volume. Total water loss secondary to dehydration is estimated from body weight loss.

Weight losses of 1.0 - 2.5kg are usual during games played in temperate climates, with the loss being greater at international level games, and less in players performing at a lower standard. A body weight loss of 1kg (1.4% of body weight) was reported in a study where players consumed 1L of fluid during the game, indicating a total sweat loss of close to 2 litres (no ambient temperature or relative humidity data were

provided with these results) In games played in the heat (33°C; 40% RH) losses of almost 4 litres were observed, although the mean loss was 2.0 - 2.5 litres; when players performed in the cool (13°C; 7% RH) the sweat rates were much less at 0.85 litres. Large sweat losses of up to 3.5 litres in some individuals resulted in seriously impairment of both physical and cognitive performance.

Similar results have been obtained from other sports involving similar levels of activity. In rugby players (two 40 minute halves; 80minutes total), a mean sweat loss of 2.1 and 2.05 litres respectively. Measurements on Australian Rules footballers (100mins of play): on a warm day (27°C) the estimated sweat loss was 3.19 litres, and fluid intake was 0.74 litres; on a hot day (38°C) sweat loss increased to 3.63 litres, but fluid intake was proportionately increased to 1.5 litres, so that net body deficit was greater on the cool day (2.45kg) than on the hot day (2.13kg). In the same sport played at 12 - 15°C, a mean weight loss of 1.4kg, and a fluid intake of 0.19 litres was recorded.

Hypohydration/Dehydration

As a consequence of free fluid exchange, hypohydration effects each fluid compartment. When body water loss is minimal, the water deficit comes primarily from the ECF. As more body water is lost, a proportionately greater percentage is lost from the ICF space.

It is known that exercise or heat-induced hypohydration increases the osmotic pressure of the plasma. Eccrine sweat is normally hypotonic relative to plasma, therefore the plasma becomes hyperosmotic when the hypohydration is induced by exercise and sweat secretion. Hypohydration combined with hyperthermia in a moderate environment reduces maximal aerobic power by 6% and exercise time by 12% from euhydration levels.

Hypohydration linearly increased the core temperature response during exercise in the heat (0.15°C) for each 1% decrease in body weight.

The change in ICF volume is a function of the change in plasma osmolality. In addition, the loss of ECF water was correlated with sodium lost in the sweat, and the changes in ICF to be inversely correlated with sodium in the sweat.

In a study plasma volume decreased by 9.4% immediately following dehydration exposure, compared to the preexposure value. This deficit recovered to -5% at 30 minutes, and -5.6% at 60 minutes of recovery when the subjects had no access to fluids. The increase in plasma osmolality during dehydration was a function of the loss of free water from the body. The free water loss caused an increase in plasma osmolality, resulting in fluid mobilisation from the ICF space to maintain ECF volume. At a given level of dehydration, the sodium concentration in the sweat determines the volume of fluid mobilised from the ICF space, thereby determining the effective maintenance of circulating blood volume. This emphasises the importance of producing a more dilute sweat in the heat adaptation process.

Age and Hypohydration/Dehydration

It has been reported that the renal concentrating capacity in response to dehydration decreases with age, becoming evident between 45 - 50 years of age. The regulation of plasma sodium also seems to be affected by the aging process, and there may be impaired fluid and electrolyte balance due to the reduced ability to detect changes in hydration status. There is also reportedly a difference in thirst perception in the aged. In addition, there is an increased secretion of ADH in response to osmotic stimuli, but a reduced secretion in response to hypovolemic signals concludes that there is a difference in the ability of younger and older healthy men to maintain plasma volume during passive heat exposure.

Effect of Fluid Replacement on Core Temperature and CV Drift

Accompanying the progressive hyperthermia (significant increase in core temperature) associated with exercise-induced dehydration, is a downward drift in central venous pressure (decrease of systemic arterial, and pulmonary arterial) and stroke volume (reduction in right ventricular end-diastolic volume pressure), and a rise in heart rate, that have been termed 'cardiovascular drift. The cardiac output also declines during prolonged exercise when the reduction in stroke volume is relatively greater than the concomitant increase in heart rate. Two factors which are likely to contribute to the cardiovascular drift during prolonged exercise are the blood volume reduction that occurs when sweat losses are not replaced, and a redistribution of blood volume from the central to the peripheral circulation. Increases in body temperature may also contribute to part of the increase in heart rate documented during prolonged exercise.

Possible Mechanisms for the Effects of Fluid Replacement

Fluid ingestion, however, can attenuate cardiovascular drift and hyperthermia. The mechanisms by which fluid replacement attenuates heat storage during prolonged exercise remain unresolved. Fluid replacement during exercise may serve to maintain sweat rates and/or skin blood flow and therefore preserve the ability to dissipate heat. In addition, several experiments have demonstrated that raising extracellular osmolality and/or sodium concentrations can impair heat dissipation. It has been hypothesised, therefore, that fluid replacement attenuates heat storage by reducing the increase in tonicity that accompanies the loss of hypotonic sweat during exercise in the heat. However, there is also evidence that reductions in blood volume and reduced cardiac filling can impair heat dissipation during exercise heat stress.

Effects on Thermoregulation of Beginning Exercise Dehydrated or Diuretic Administration

When subjects begin exercise in the dehydrated state, the onset of sweating is delayed, and sweating rate at any given core temperature is reduced. Relatively large reductions in blood volume (490 - 680ml), induced by diuretic administration, showed elevated core temperatures during exercise heat stress, probably due to slope of the core temperature/sweat rate relationship being reduced.

The concept that dehydration causes a large reduction in blood volume during exercise was derived from experiments on individuals who had become dehydrated before exercise, not during exercise. Evidence that reductions in blood volume can hinder temperature regulation during exercise has been derived from experiments in which a higher oesophageal temperature, reduced sweating and reduced forearm blood flow, were observed when subjects began to exercise after an acute 490 - 680ml reduction in blood volume. A 9 - 12% diuretic induced reduction in blood volume can negatively affect running performance. These experiments clearly demonstrate that relatively large reductions in blood volume, prior to exercise, can alter temperature regulation and performance during subsequent exercise. However, such large reductions in blood volume do not usually occur with exercise dehydration.

Blood volume during a no fluid trial declined only ~250mL during the 5 - 120min period of exercise (assuming 80mL of blood per kg). This, and the findings of other authors, demonstrate that the small decline in blood volume during exercise dehydration in well trained subjects is not responsible for the rise in core temperature observed during exercise when no fluid is ingested.

It is well documented that fluid replacement of sweat losses during exercise can attenuate the rise in core temperature by 0.5 - 0.8°C. It is postulated that fluid ingestion reduces hyperthermia by the maintenance of blood volume. This premise is based on reports that blood volume expansion can reduce core temperature during exercise by 0.1 - 0.2°C and that relatively large reductions in blood volume (490 - 680ml) induced by diuretic administration resulted in elevated core temperatures during exercise heat stress.

In a study it was postulated that volume infusion during strenuous exercise (84% VO_2max), by expanding blood volume, would allow better skin blood flow and better temperature homeostasis, and thereby improve endurance time. The results showed that plasma volume decreased less during the infusion trial, and this reportedly improved fluid homeostasis which resulted in lower core temperature (39°C±0.2 vs. 38.5°C±0.2) and heart rate (194 vs. 186bpm) in the infusion compared to the no infusion trial. The authors, however, found no difference in endurance time, H^+, peak O_2 uptake, CO_2 production, end-tidal partial pressure of CO_2, blood lactate, or blood pressure between the trial conditions. It was concluded that saline infusion improved heat dissipation as evidenced by a lower core temperature, but did not improve endurance. The effects of saline and bicarbonate infusions on thermoregulation were investigated in exercising rats. The rats exercised in environmental temperatures of 35°C, which is high, but they found no difference in core temperature between the control and the infused rats.

There are discrepancies in data relating to plasma volume changes during dehydration and rehydration, which are difficult to explain. Because sweat is hypotonic, the loss of water from prolonged sweating causes an increase in plasma osmolality. As the lost water has to be replaced by rehydration, fluid replacement could be more or less efficient depending on its osmolality. In fact, recovery of the plasma constituents results from the interaction of many factors which effect transit and absorption in the gastrointestinal tract. Examples of findings which illustrate the complexity of these interactions are that gastric emptying is modified by exercise intensity and body hydration status; the presence of glucose inhibits water absorption and, isotonic saline

solutions can be absorbed even more rapidly than water. In a study five subjects exercised for prolonged periods (4 hours at 34°C) while ingesting either no fluid, water, a hypotonic, hypertonic or isotonic solution. Rectal temperatures were significantly higher for the no fluid trial and hypertonic fluid ingestion trial, whereas skin temperatures were not influenced by the hydration procedure. There were no differences in body weight loss among experimental trials, mean final rehydration after fluid intake was 82%. Heart rate was higher in the no fluid trial, while no difference was found among conditions in body weight loss or mean hourly sweat rates. Sweating sensitivity was lowest in the dehydrated condition, and highest in the water one. Modifications in plasma volume and osmolality demonstrated that no fluid induced hyperosmotic hypovolemia; ingestion of an isotonic solution led to rapid isoosmotic hypervolemia; and, water led to slightly hypoosmotic (less 5mosmol/L) euvolemia.

Cardiovascular drift is primarily due to the displacement of blood to the cutaneous circulation. However, a similar rise in heart rate was observed during two stages of both prolonged (90 mins) cycle and swimming exercises. In spite of differences in peripheral pooling and sweating, due to the varying hydrostatic forces in the two forms of exercise, the secondary rise in heart rate was similar. It was unlikely that the continuous increase in heart rate was due to peripheral displacement of the central blood volume.

Fluid ingestion, compared with no fluid ingestion, during 120mins of cycling at 70% VO_2max, resulted in a higher stroke volume, cardiac output and blood volume, and a lower heart rate during the last hour of exercise. In addition, serum osmolality and electrolyte concentrations remained no different between the two treatments for the first hour of exercise, however serum osmolality (290mOsm/kg vs. 302mOsm/kg) and sodium concentration (145mmol/L v 149mmol/L) increased progressively during the second hour of exercise in the no fluid trial. It was suggested that the addition of carbohydrate to the replacement fluid was partially responsible for the maintenance of serum osmolality seen during their fluid replacement trials. Sweat electrolyte concentrations were not considered.

Reduction in blood volume observed during exercise dehydration does contribute to reductions in cardiac output and stroke volume during prolonged exercise. This and the findings of others, however, demonstrate that cardiovascular drift can occur independently of reductions in blood volume. These findings suggest that fluid replacement attenuates cardiovascular drift, in part, by altering blood flow or blood volume distribution.

Another study showed an important finding in that fluid replacement (80% of sweat losses) increased forearm blood flow after 100mins of exercise, compared with the no fluid trial and a blood volume expansion trial, suggesting that increased skin blood flow was responsible for the attenuation of hyperthermia. A higher skin blood flow could reduce the heat storage by increasing heat transfer from the body core to the periphery and increase the evaporation of sweat from the skin surface. Furthermore, fluid replacement increases skin blood flow by some mechanism other than by increasing blood volume. The results of the fluid replacement trial resulted in significantly lower serum osmolality values; therefore fluid replacement may increase

skin blood flow by preventing an increase in osmolality. Sweat electrolyte concentrations were not reported.

Other data support the hypothesis that fluid replacement reduces hyperthermia during the second hour of exercise by maintaining a high skin blood flow and possibly by preventing significant increases in serum osmolality and/or sodium concentrations during exercise.

Effect of Fluid Ingestion on Hyperthermia

The mechanisms by which fluid ingestion attenuates the rise in core temperature during exercise remain unclear. Several investigators have reported increased sweat rates with fluid ingestion than with no fluid ingestion. Others have reported no difference in sweat rate. Depending on the experimental protocol, elevated body temperatures may or may not be associated with reduction in sweat rates. It has also been demonstrated on other occasions that sweating rates could be reduced without dramatic elevation in core temperature through the hydromeiotic phenomenon, as long as no change occurs in the evaporation rates, therefore questions whether or not the sweating decline is the factor responsible for the elevation of temperature and heart rate during dehydration. One researcher determined that fluid ingestion resulted in increases in skin blood flow, independent of increases in blood volume, and concluded that the attenuated rises in core temperature observed with fluid ingestion may be due to increased skin blood flow. It was also reported that sweat rates were no different between fluid ingestion and no fluid ingestion trials. This study concluded that the attenuation of hyperthermia evidenced when fluid is ingested during exercise heat stress compared when fluid is not ingested is not due to the small increase in blood volume. This was determined by the use of a blood volume expander which maintained the blood volume similar to that expected when fluid was ingested during exercise. Given that the blood volume expansion resulted in similar rectal and oesophageal temperatures, forearm blood flow and serum osmolality and serum sodium concentrations as the no fluid ingestion trial, fluid ingestion attenuates the hyperthermia by some other mechanism than the maintenance of blood volume.

An estimate of cutaneous blood flow during moderate exercise in a thermoneutral environment is 3L/min. Redistribution of flow from visceral organs and inactive muscle cannot account for the increased skin flow. Hence, it is assumed at a constant cardiac output, active muscle flow would have to be compromised, or cardiac output would have to increase. At a critical core temperature, the need for temperature homeostasis would take precedence over muscle blood flow. A decrease in muscle blood flow can be postulated due to a reduction in the peak cardiac output, resulting from increased skin blood flow and blood pooling in the compliant skin vasculature. Exercise would therefore decrease, as muscle blood flow would become inadequate for energy demands
Reductions in cardiac output during exercise in the heat would only be evidenced when the demand for skin blood flow is high enough to exhaust the reserves in cardiac output. This reserve can be estimated using the formula cardiac output = (0.06wt) + 5.5 VO2 (L/min).

Reduction in sweating rate was clearly observed during very pronounced dehydration (reduction in plasma volume of 12-16%), and there is no doubt that large

hypovolemia is associated with sweating depression. However, some other researchers failed to show any alteration in sweat output with dehydration in conditions similar to those of other experiments. Temperature regulation and thus sweating response during dehydration is a complex mechanism strongly dependent on both plasma volume and osmolarity changes. In opposition to this conclusion, others showed that the sweating sensitivity after the ingestion of a hypertonic (846mOsm/L) solution was also somewhat depressed, although blood volume and osmolality were in fact not greatly modified. Even in the isotonic fluid ingestion trial, the sweat rate was not greater than in the other hydration conditions (water and a hypotonic solution), and this confirms that hyperhydration via plasma volume expansion has no direct consequence on temperature and sweating regulation. A 3% dehydration induced a hyperosmotic hypovolemia associated with increased heart rate and depressed sweating sensitivity. Hydration will tend to compensate for all physiological disturbances, but fluid composition is of some importance for recovery: there was a tendency for greater heart rates and a smaller sweating sensitivity during rehydration with a hypertonic glucose solution. Water and hypotonic solutions allowed all physiological parameters to return to their pre - test levels. Ingestion of isotonic sucrose solution, even in quantities smaller than the fluid losses, induced, in addition to physiological recovery, a plasma volume expansion necessary for the maintenance of blood pressure at levels compatible with prolonged exercise in the heat.

Recommendations for Fluid Intake

To prevent dehydration people should drink an extra cupful of water every hour (extra 1.5 - 2L per day) and be weighed three times per day because rapid weight loss in the heat means water loss not fat loss. People losing weight should therefore drink more - possibly even 10L- to maintain body weight. Urine should be plentiful and light coloured. Urine that is dark, strong smelling, or of reduced volume means the person probably has a large fluid deficit. Salt depletion from sweating causes tiredness, irritability, giddiness, fainting, cramps and loss of performance. During the first ten days of heat exposure, additional salt should be taken as salt tablets or dilute solution such as Aqualyte. A normal diet should provide sufficient of the other electrolytes. If need be 10 - 20mEq of sodium could be added to the hydration fluid for exercise (60 - 90% VO2max) of 1 - 3hours in duration; 20 - 30mEqu of sodium for exercise (30 - 70% VO2max) exceeding 3 hrs; and, 30 - 40mEqu in the recovery fluid.

Insufficient fluid replacement may result in dehydration during exercise. This may impair body heat dissipation and endurance performance as a result of decreased plasma volume, increased plasma osmolality, decreased sweat rate and skin blood flow, and raised core temperature.

A status of severe dehydration may limit sweat production and skin blood flow and therefore evaporative capacity. Because sweat evaporation is a major route of heat dissipation, it is clear that a status of severe dehydration, which will impair blood flow as well as sweat production, may lead to hyperthermia by a reduction in heat elimination. Because dehydration depends on quantitative sweat loss, and the latter is determined by exercise intensity, duration, environmental temperature and clothing, the interactive effects of these factors should be considered.

In high environmental temperatures, 60% of cardiac output may pass through the skin for cooling and sweat production, and unacclimatised people will perform less well in the heat.