Fluid Replacement During Exercise

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HISTORICAL INTRODUCTION

Included among the "Don'ts" in a 1909 monograph titled "Marathon Running" [152] was the admonition: "Don't get in the habit of drinking and eating in a Marathon race; some prominent runners do, but it is not beneficial." The advice of Arthur Newton, a leading ultramarathon runner of the first half of this century, who held world records for running distances from 50-130 miles [117] was similar: "Even in the warmest English weather, a twenty-six mile run ought to be manageable with no more than a single drink, or at most two" [115].

In 1957, the then holder of the world's four fastest times for the 42-km marathon footrace wrote: "There is no need to take any solid food at all (during a marathon race) and every effort should be made to do without liquid, as the moment food or drink is taken, the body has to start dealing with its digestion and in so doing some discomfort will almost invariably be felt" [135]. Another famous ultra-distance runner who set world records at 30, 40, and 50 miles in 1954 confirmed that this advice was widely accepted: "In those days it was quite fashionable not to drink, until one absolutely had to. After a race runners would recount with pride 'I only had a drink after 30 or 40 kilometres.' To run a complete marathon without any fluid replacement was regarded as the ultimate aim of most runners, and a test of their fitness." (J. Mekler, 1991, personal communication). This athlete, who once competed in a 100-mile race in which he first drank only after 75 miles, confirmed that this approach was still popular when he had run his last competitive ultramarathon race in 1969.

That these ideas could ever have held credence, let alone so recently, may seem absurd to the modern exercise physiologist. But the fluid requirements of athletes has attracted scientific interest only since the early 1970s. Virtually all of the textbooks of exercise physiology and sports medicine published before 1970 contain little or no reference to this topic [2, 12, 44, 52, 57, 89, 164]; one of the first to include a section on fluid replacement during exercise was the monograph by Costill [31]. Yet many studies showing the importance of adequate fluid replacement especially during industrial and military activities in the heat had already been published.
The building of the Hoover Dam near Boulder City, Nevada in the 1930s stimulated the first systematic studies of fluid (and electrolyte) requirements during work in temperatures ranging between 90–120°F. One early study described the clinical and biochemical features of so-called "heat cramps" in seven workers on this project [157]. Another study measured fluid intake and urine output during both work and rest [156] and concluded that construction work could be undertaken without major health risks even in these severe environmental conditions provided subjects were accommodated in air-conditioned lodgings [46]. Yet another early study showed that sweat sodium concentration fell within days of exposure to extreme heat becoming so low that "10 litres per day may be secreted without the necessity of an abnormal salt intake" [46].

More detailed studies by Adolph and Dill [7] showed that the daily fluid intakes of construction workers averaged about 4 liters/day but rose as a linear function of increasing maximum daily temperature. Sweat rates increased from a resting value of 400 ml/hr when sitting in shade, to maximum rates of 1500–1700 ml/hr during 1–2 hr of exercise in the heat. Subjects showed relatively little desire to drink during exercise; fluid ingestion was greatest immediately after exercise and with meals. In the absence of food ingestion, drinking stopped when approximately one-half of the fluid loss had been corrected. The authors suggested that: "...since some solute as well as water was lost, only enough fluid was required by the subject to render the concentration of the body fluids equal to their previous state." They also noted that all the water ingested an hour before exercise was lost in urine before the onset of exercise. But if the fluid contained sodium chloride "the water was retained for several hours before it was required for the formation of sweat." Subsequent studies suggested that 1 or more liters of fluid with a sodium chloride content of 0.5–0.9% (80–140 mmol/liter) ingested 2–3 hr before exercise would increase body fluid content [5].

Despite the clear reluctance of dehydrated subjects to replace their fluid losses fully during and immediately after exercise, their daily fluctuations in weight were no greater than those measured in winter climes, indicating that fluid imbalances were corrected diurnally: "All the deviations from water balance were on the negative side; water was expended and a debt built up, which was later paid off." In contrast, Dill [46] found that within minutes of being given water, the dog and the donkey (burro) replaced exactly all the fluid they had lost during exercise. In one case, a burro drank 12 liters in 5 minutes [46]. Among the mammals studied by then, therefore, dehydrated humans were unique in this inability to replace all the fluid lost during exercise, as soon as water was provided.

The demands of desert warfare during the Second World War stimulated the next and, possibly, the truly classic early studies of fluid replacement during exercise [5]. These magnificent studies established for the first time, many of the concepts that continue to intrigue modern physiologists. Interestingly, at the time the studies commenced, it was believed that men, like marathon runners, should be "trained to do without water." Among the classic findings were the following:

1. Maximal sweat rates during exercise under the most severe environmental desert conditions were of the order of 1.7 liters/hr; much higher rates were reported during exercise in conditions of high humidity (jungle heat). Sweat rates during exercise in desert heat rose as a linear function of body weight and, hence, metabolic rate and environmental temperature.
2. Sweat rates during exercise were not influenced by the level of dehydration. Thus, the capacity for evaporative heat loss during low-intensity exercise was not impaired by progressive dehydration. Despite this, the rectal temperature rose during exercise as a linear function of the level of dehydration.
3. Urine flow was reduced when levels of dehydration exceeded 3% but did not increase dehydration levels of 7%. Thus, progressive dehydration did not induce fluid conservation by decreasing urine output. Anuria was not detected in any subject under any conditions. The constant urine output was needed to excrete the solutes contained in urine.
4. Chloride was present in virtually all urine samples measured. Thus, "salt deficiency in the desert was not common."
5. Even when given free access to adequate fluids during exercise, subjects developed a progressive weight loss, termed "voluntary" dehydration. Food ingestion greatly increased fluid intake; at least one-half of the fluid ingested daily was taken with meals. Conversely, without an adequate fluid intake, food intake during meals was also reduced. But body weight was usually restored after the evening meal.
6. Unless corrected, the progressive dehydration that developed during exercise in the heat impaired physical performance. At low levels of dehydration, physical performance was impaired only during high-intensity exercise. Subjects became syncopal, however, and were unable to exercise even at low exercise intensities when their levels of dehydration exceeded 7%. Feelings of well-being and the ability to walk returned within minutes of the resumption of drinking. Maximal levels of dehydration compatible with life were of the order of 20%.
7. The fluid lost in sweat did not originate equally from the different fluid compartments but came predominantly from the extracellular compartment. This would impair cardiovascular function.
8. Heart rate and rectal temperature increased and stroke volume fell as
a linear function of increasing levels of dehydration. Rectal temperature rose 0.3°C for each 1% increase in the level of dehydration.

9. The level of dehydration correlated most accurately with the rise in rectal temperature, the standing heart rate, and the reduction in salivary flow. Salivary flow ceased at dehydration levels greater than 8%.

10. Sodium ingestion during exercise tended to increase the amount of fluid ingested and reduced the urine output.

These studies, which clearly established the value of fluid ingestion during exercise, made little immediate impact on the athletic community. The studies of Pugh et al. [138] and Wyndham and Strydom [169] finally drew attention to the need for fluid ingestion also by athletes during prolonged exercise.

Pugh et al. [138] showed that competitors drank only 400 ml during a marathon race (0.12 liters/hr) and developed a mean weight loss of 2.9 kg (5.9% of initial body weight). As fluid loss, sweat rate, and postrectal temperature were highest in the race winner, Pugh et al. [138] concluded that "a high tolerance to fluid loss" seemed to be an important requirement for success in distance running. But they did not propose any role for fluid ingestion during exercise.

In their study, Wyndham and Strydom [169] found that athletes who became dehydrated by more than 3% during a series of 32-km foot races had elevated postrectal temperatures. As also found by Adolph [5], there was a linear relationship between the athletes' levels of dehydration and their postrectal temperatures, at least for levels of dehydration greater than 3%. The authors concluded that: (a) the level of dehydration was the most important factor determining the rectal temperature during prolonged exercise so (b) the avoidance of dehydration would be the critical factor preventing heat injury during prolonged exercise [168, 169]. Interestingly, the authors did not speculate that dehydration might influence running performance. Their finding that the race winners had the highest rectal temperatures and were the most dehydrated [119], may have dissuaded them.

Although the basis for some of their conclusions has been challenged [119, 121, 125], there is no doubt that it was especially the study of Wyndham and Strydom [169] that stimulated the modern interest in the role of fluid replacement during exercise.

MODERN STUDIES OF FLUID LOSS AND FLUID INGESTION DURING EXERCISE

There are two experimental models that have been used to study the effects of fluid loss and fluid intake on the physiological responses during exercise.

As emphasized by Coyle et al. [35, 36], fluid loss (hypohydration) induced either by the administration of diuretics, or exposure to sauna, or fluid restriction before exercise, produces physiological effects that are more marked than those that result from the form of dehydration that develops voluntarily during exercise when the rate of fluid loss exceeds the rate of fluid ingestion. In particular, the reduction in plasma volume and in physical performance for a given level of dehydration is greater with hypohydration [22, 35, 107].

For this reason, the result of the hypohydration studies that are reviewed in detail elsewhere [147] are perhaps most relevant for activities in which subjects deliberately dehydrate themselves before exercise, usually to make a specific competitive weight. These studies will not be considered further in this review; we will focus principally on studies of exercise-induced dehydration.

Effect of Exercise-induced Dehydration on Physical Performance

At least six studies [5, 15, 49, 91, 136, 151] have evaluated the effects of progressive exercise-induced dehydration on physical performance. Most have compared the performance of a group of subjects, usually military personnel, who either ingested fluid or restrained from fluid ingestion during prolonged exercise of relatively low intensity (walking) in dry heat. The measure of physical performance was the number of subjects in each group able to complete the prescribed exercise task.

Although few of the studies were subject to rigorous statistical analysis, the trend in all was for fewer subjects to complete the exercise task when they did not ingest fluid. Furthermore, performance was least impaired in subjects who maintained fluid balance during exercise. Fluid ingestion reduced rectal temperatures and heart rates during exercise; this effect was greatest when the rate of fluid ingestion equaled the sweat rate. In general, sweat rates were not influenced by fluid ingestion and fell only when the fluid deficit exceeded 2.5 liters [91]. Fluid ingestion also prevented the development of postural hypotension on cessation of exercise [49].

Most researchers observed that fluid ingestion had more obvious effects on the psyche than on the soma. The description of Bean and Eichna [15] is typical for subjects who did not ingest fluid during exercise: "An important change which the chart does not show was the actual condition of the men, their low morale and lack of vigor, their glassy eyes, their apathetic, torpid appearance, their 'don't-give-a-damn-for-anything' attitude, their uncoordinated stumbling, shuffling gait. Some were incapable of sustained purposeful action and were not fit for work. All they wanted to do was rest and drink" (p. 155). Eichna et al. [49] reported that dehydrated subjects were "reduced to apathetic, listless, plodding men straining to finish the same task" that they completed "energetically and cheerfully" when fully hydrated. Similarly,
Strydom et al. [151] reported that the fluid restriction caused their subjects to become morose, aggressive, and disobedient toward their superiors.

Since 1966, there have been relatively few studies that have looked specifically at the effects of fluid ingestion alone on athletic performance. Rather, the evaluation of carbohydrate ingestion has been emphasized [30, 36, 74, 75]. These studies generally show that carbohydrate ingestion enhances performance [30, 36] and the assumption is that this is due only to a metabolic effect. However, the addition of glucose to the ingested solution increases fluid absorption [66, 67]; hence, a part of the beneficial effect of carbohydrate ingestion during exercise could theoretically be due to an influence of the added carbohydrate on fluid balance.

Four modern studies have compared the exercise performance of subjects when they either ingested or did not ingest fluid during exercise. Maughan et al. [101] found that ingestion of either water or concentrated carbohydrate solutions did not increase endurance time at 70% \( V_{O_{2max}} \), which exhausted subjects in 70–75 minutes. Endurance was prolonged, however, in subjects who ingested a dilute carbohydrate/electrolyte solution.

In contrast, Barr et al. [14] showed that subjects who did not ingest fluid during prolonged exercise at 55% \( V_{O_{2max}} \) terminated exercise approximately 90 min earlier than when they ingested fluid. Levels of dehydration at exhaustion were >6%. Montain and Coyle [108] also found that, when they did not ingest fluid, subjects were less likely to complete exercise in the heat at 65% \( V_{O_{2max}} \).

Two studies have assessed the effects of fluid ingestion or infusion at higher exercise intensities. Deschamps et al. [43] reported that intravenous saline infusion did not enhance performance at 84% \( V_{O_{2max}} \), which exhausted subjects in approximately 21 min. Walsh et al. [163], however, found that subjects were able to exercise significantly longer during a subsequent exercise bout at 90% \( V_{O_{2max}} \) when they ingested fluid during a preceding 1-hr exercise bout at 70% \( V_{O_{2max}} \) in the heat. This effect was not because of differences in any measured physiological variable including rectal temperature, and occurred despite a difference in fluid balance of only 1.1 kg between dehydrated and fluid-repleted subjects. Ratings of perceived exertion were significantly lower when fluid was ingested.

In summary, the balance of evidence indicates that the ingestion of water enhances performances during both very prolonged exercise of low intensity and during exercise of somewhat higher intensity but shorter duration. However, this effect may be somewhat less than that achieved when the fluid contains carbohydrate either alone [166] or with electrolytes [30, 101]. Possibly the most consistent finding is that fluid ingestion markedly reduces the perception of effort during exercise of both low [5] and high-intensity exercise [163]. It is also probable that exercise performance during high-intensity exercise is impaired at levels of dehydration that do not influence performance at lower exercise intensities [5].

**Physiological Effects of Progressive Dehydration During Prolonged Exercise**

The physiological effects of exercise-induced dehydration have been studied by comparing the physiological responses of athletes when they replace either none, some, or all of their fluid lost during prolonged exercise. More recent studies have infused fluid intravenously during exercise to reverse any dehydration-induced fall in plasma volume. These latter studies have attempted to differentiate the physiological effects caused by reduction in plasma volume from those caused by dehydration-induced changes in serum osmolality.

**Plasma Volume.** Plasma volume falls at the initiation of exercise. This fall is influenced by the type and intensity of exercise, and by the posture adopted [35].

Thereafter, a progressive exercise-related fall in plasma volume is reduced in proportion to the amount of fluid ingested during exercise [14, 24, 100, 107, 108]. The change is least when most fluid is ingested [108] and can be prevented if the rate of fluid ingestion equals the rate of fluid loss [73].

There is some evidence that the ingestion of sodium-containing solutions during exercise may prevent the fall in plasma volume more effectively than the ingestion of pure water [23]; as discussed subsequently, water ingestion tends to reduce serum osmolality.

**Serum Osmolality and Serum Electrolyte Concentrations.** Serum osmolality rises if no fluid is ingested during prolonged exercise [24, 100, 101]. This rise is reduced by fluid ingestion [24, 100] and is least when the rate of fluid ingestion approximates the rate of fluid loss [108].

Changes in serum sodium concentrations parallel changes in serum osmolality because the serum sodium concentration is the major determinant of the serum osmolality. Hence, serum sodium concentrations rise with exercise-induced dehydration but are maintained when fluid is ingested [117, 120]. Interestingly, serum sodium concentrations and serum osmolality fall only when water is ingested during exercise [14, 23, 116]. This is a fundamental observation that invites explanation. No studies have yet reported that serum sodium concentrations fall in the absence of fluid ingestion during exercise; this would be unlikely as sweat is a hypotonic solution [32].

The rise in serum osmolality and serum sodium concentration correlates with the rise in esophageal temperature [108] and may be the stimulus for the reduction in sweating that develops at the higher levels of dehydration [91]. This suggests that an important goal of fluid
ingestion during exercise may be to prevent changes in serum osmolality or serum sodium concentrations, as originally proposed by David Dill [46].

**Sweat Rate During Exercise.** Some studies have shown that the sweat rate falls with increasing levels of dehydration [53, 71, 91, 149] whereas others have failed to show this effect [14, 24, 65, 73, 107, 108]. An early study found that sweat rate falls only above a certain level of dehydration [91].

Hypohydration studies indicate that sweat rate over the trunk but not the head may be selectively reduced by prior dehydration [25]. There may also be considerable individual variability in the effects of dehydration on sweat rates during exercise [149]; perhaps this explains the variable results reported in the literature. Hyperhydration prior to exercise increases sweat rate during subsequent exercise [110].

**Rectal or Esophageal Temperatures During Exercise.** The exercise-related rise in rectal temperature is attenuated by fluid ingestion during exercise [5, 14, 24, 33, 53, 65, 71, 73, 107, 108]. The rise is reduced in proportion to the amount of fluid ingested and is least when the rate of fluid ingestion approximates the sweat rate [71, 108]. Hence there is a linear relationship between the rise in esophageal temperature and the level of dehydration [107, 108] as also reported in some of the original field studies [5, 169].

Fluid ingestion reduces the rectal temperature response only after a minimum of 60–80 min of exercise [14, 73, 107, 108, 149, 150]. No effect of fluid ingestion on rectal temperature was found in a study of shorter duration but higher intensity [163], possibly because exercise terminated before 80 min.

The magnitude of this effect of fluid ingestion on the rise of rectal temperature is relatively small [122] so its real physiological relevance may be questioned. Most studies indicate that levels of dehydration of up to 5% (equivalent to a weight loss of 2–4 kg) usually increase rectal temperature by less than 1°C [14, 65, 73, 107, 108, 121]. Hyperhydration before exercise also decreases rectal temperature during subsequent exercise [111].

**Heart Rate and Stroke Volume.** Heart rate is increased [5, 24, 71, 100, 149] and stroke volume reduced in proportion to the fluid deficit that develops during exercise [108]. Cardiac output and stroke volume do not fall when the rate of fluid ingestion is sufficient to prevent dehydration [73]. But heart rate is elevated even when dehydration is prevented by adequate fluid ingestion during exercise [73]; hence, dehydration is not the sole cause of the progressive increase in heart rate during prolonged exercise. Heart rate is also reduced by hyperhydration before exercise [110].

**Skin Blood Flow.** Fluid ingestion maintains higher rates of forearm blood flow during exercise [107, 108] and of forearm and calf blood flow at rest during prolonged heat exposure [82]. The reduction of forearm blood flow is proportional to the level of dehydration [108]. Hence, fluid ingestion during exercise may attenuate the development of hyperthermia by maintaining skin blood flow [107].

**Perception of Effort.** The perception of effort during exercise is increased in proportion to the fluid deficit [108]. Even partial fluid replacement has a significant effect on the perception of effort during exercise of high intensity [163]. The major psychosomatic effects of fluid restriction during prolonged exercise of low intensity and their rapid reversal with fluid ingestion, have been described.

**Hormonal Changes.** Plasma concentrations or activities of the fluid and electrolyte-regulating hormones, specifically, atrial natriuretic peptide (ANP), antidiuretic hormone (ADH—also arginine vasopressin—AVP), aldosterone, and renin, increase during prolonged exercise [9, 17, 64, 162]. With the exception of ANP, these concentrations may remain elevated for up to 31 hr after exercise [9]. In general, ADH activities rise in response to increasing serum osmolality [159, 162], whereas plasma renin activity may follow changes in either plasma or extracellular fluid volumes.

There is also a paradoxical increase in the plasma activity of the diuretic and natriuretic hormone ANP during prolonged exercise when plasma volume is reduced [9, 162]. The diuretic effects of ANP are probably inhibited by the increased activity of the renin-angiotensin-aldosterone axis and the increased serum ADH concentrations [9, 161].

Fluid ingestion during exercise reduces the hormonal activities and concentrations during exercise; concentrations are further reduced when subjects hyperhydrate before exercise [17]. Of these, the rise in plasma ADH activity is most affected by either hyperhydration before or fluid ingestion during exercise; this effect may be relatively independent of the nature of the fluid ingested. In contrast, the increased activity of the renin-angiotensin-aldosterone axis is reduced more by the ingestion of sodium-containing solutions compared with pure water [17]. Thrasher et al. [159] reported essentially the same findings in dehydrated dogs; water ingestion reduced plasma ADH activity, whereas ingestion of an electrolyte solution with the same composition as the extracellular fluid volume reduced plasma renin activity.

As described, water ingestion, especially during exercise, causes serum osmolality to remain the same or to fall; this would reduce ADH secretion. Why the ingestion of sodium-containing solutions specifically reduces plasma renin activity and aldosterone concentrations is not known. Possibly, the effect is because of the maintenance of higher plasma volumes when electrolyte-containing solutions are ingested [17, 23].

**Fluid Ingestion and Renal Function.** Renal function during prolonged exercise like marathon running is unaffected by levels of
dehydration less than 4% [86–88] and is enhanced during recovery in those who retain fluid during exercise [85]. The classic studies also found that renal function was not influenced by levels of dehydration less than 7% [4]. Anuria has been reported in one runner who drank inadequately and lost 11% of her body weight during an 88-km ultramarathon [88].

Renal fluid and electrolyte excretion may be influenced by the nature of the fluid ingested during exercise, but this has been infrequently studied during exercise [14] or even at rest [20]. The renal response to the ingestion, during exercise, of fluids with different electrolyte contents will be influenced by a number of factors including the relative amount of sodium and water lost in sweat, the extent of the sodium movement from the interstitial space into the intestinal lumen [66, 67] and the relative rates of sodium and fluid absorption from the ingested solution. As yet, there are no studies that have addressed these issues, either singly or collectively.

GASTRIC EMPTYING AND INTESTINAL ABSORPTION. Dehydration impairs gastric emptying [113] and could theoretically limit fluid replacement during prolonged exercise. In contrast, intestinal absorption may increase when blood volume falls [148].

But gastric emptying should not limit fluid replacement during exercise as high rates of gastric emptying can always be achieved with the appropriate drinking patterns [127]. It is more probable that fluid absorption by the small bowel could limit fluid replacement especially when only plain water is ingested at high rates during prolonged exercise (see later).

INFLUENCE OF EXERCISE-RELATED FALL IN PLASMA VOLUME ON PHYSIOLOGICAL RESPONSES DURING EXERCISE. An important question is whether changes in serum osmolality or in plasma volume regulate the physiological responses to exercise-induced dehydration. The infusion of isotonic fluids that maintain plasma volume without altering serum osmolality can differentiate between these possibilities [62].

BODY TEMPERATURE, SKIN BLOOD FLOW AND SWEAT RATE. Fortney et al. [62] showed that the infusion of an isotonic saline solution reduced both the core temperature and total sweat output during exercise. The authors concluded that the maintenance of plasma volume increased skin blood flow thereby increasing convective heat losses. Hence, evaporative heat losses were reduced. In contrast, increased serum osmolality raises the temperature threshold at which skin blood flow increased and delayed the onset of sweating, thereby favoring heat retention [62, 63].

Montain and Coyle [107] infused a 6% dextran solution to increase plasma volume above the level maintained by fluid ingestion during exercise. Serum sodium concentrations and serum osmolality increased when subjects did not ingest fluid during exercise but were maintained at pre-exercise concentrations by saline infusion. Unlike the findings of Fortney et al. [63], plasma volume expansion alone did not influence either the esophageal or rectal temperature response to exercise, or forearm skin blood flow. Nor did fluid infusion prevent the rise in the rating of perceived exertion as effectively as did fluid ingestion.

Thus, maintaining or increasing plasma volume by fluid ingestion does not explain the temperature-lowering effect of fluid ingestion during exercise. Rather, fluid ingestion may influence thermoregulation by preventing the rise in serum osmolality or serum sodium concentrations [107] with maintenance of forearm blood flow [128]. These findings support Dill’s [46] proposal that the goal of fluid ingestion during exercise should be the maintenance of serum osmolality and serum sodium concentrations.

Cardiovascular Drift

Compared with the findings when no fluid was ingested, cardiac output and stroke volume were higher with intravenous infusion; but this effect was less than that achieved by full fluid replacement [107].

The authors concluded that the cardiovascular drift shown by an increase in heart rate and a fall in stroke volume and cardiac output during prolonged exercise is not solely caused by the fall in plasma volume. Thus, the beneficial physiological effects of fluid ingestion during exercise cannot be due solely to the maintenance of plasma volume and central cardiovascular function.

Hamilton et al. [73] have also shown that ingesting fluid at rates that prevent dehydration maintains cardiac output and stroke volume during prolonged exercise. Other components of cardiovascular drift, in particular the rise in heart rate and in oxygen consumption, were prevented only by the addition of a glucose infusion. They have proposed that especially the rise in oxygen consumption during prolonged exercise results from a catecholamine-mediated stimulation of metabolism that is prevented by glucose infusion.

OPTIMUM RATES OF FLUID INTAKE DURING EXERCISE

The evidence so far presented suggests that the principal aim of fluid ingestion during exercise is to prevent any rise in serum osmolality or serum sodium concentrations. A secondary goal is also to prevent any change in plasma volume. Few studies have considered the interacting influences of the rates of fluid loss and fluid ingestion, and the composition of the ingested solution on changes in these variables during exercise. Rather, it has been assumed that the optimum rate of fluid ingestion is always that which equals the rate of fluid loss. It is clear, however, that most subjects do not voluntarily replace all the fluid lost during exercise [5]. Therefore, it is appropriate first to discuss the
factors that influence the rates of fluid loss and fluid ingestion during exercise as this may identify those exercising conditions when the development of voluntary dehydration is more likely. The possible effects of the nature of the ingested fluid on changes in serum osmolality and plasma volume under these conditions will also be briefly considered.

**Rates of Fluid Loss During Exercise**

The rate of sweat loss, which is the principal determinant of fluid loss from the body during exercise, is determined mainly by the metabolic rate [32, 39, 40, 69, 70, 125, 171]. At least in running, the metabolic rate is determined by the body mass and the running speed; in nonweight-bearing activities like cycling, the velocity of movement becomes the principal determinant of the metabolic rate. Barr and Costill [13] have predicted sweat rates for subjects of different masses running at different speeds. Figure 10.1, drawn from their data, shows the interaction of body mass and running speed on sweat rate and predicts, for example, that heavier athletes running quite slowly can have sweat rates equal to those of smaller runners running much faster.

![Figure 10.1](image)

**Figure 10.1**


The prediction of this figure is that sweat rates will seldom be greater than 1.2 liters/hr in runners weighing less than 70 kg. Such high sweat rates are probable only in runners weighing 80 kg or more running faster than 12 km/hr. Heavier runners probably achieve these racing speeds only infrequently, at least for prolonged periods, for example, in marathon races.

The finding that sweat rates measured in runners during longer distance races are seldom greater than 1.2 liters/hr (for review, see [125]) confirms the general accuracy of these predictions. Higher sweat rates are usually recorded only when the environmental conditions are more severe (dry bulb temperature > 25°C) or when the activity is held indoors without the benefit of adequate convective cooling. For example, compared with sweat rates measured at the same metabolic rate in wind-still conditions indoors, sweat rates of cyclists are reduced by up to 38% during outdoor exercise [20].

Therefore, it seems probable that the reports of much higher sweat rates measured in the laboratory [10, 105] could possibly be explained, in part, by the absence of adequate convective cooling during exercise indoors.

**Rates of Fluid Ingestion During Exercise**

Table 10.1 lists the reported rates of fluid intake in both competitive and experimental studies both outdoors and in the laboratory, in runners, cyclists, and triathletes competing over a wide range of distances. Table 10.2 lists the absolute and relative exercise-induced weight changes in subjects competing in the same or similar events.

Table 10.1 shows that the rates of fluid intake during exercise vary considerably but are seldom more than about 500 ml/hr except in subjects cycling in the laboratory when forced to ingest fluid at higher rates. One conclusion might be that subjects voluntarily choose to drink about 500 ml/hr during exercise with little likelihood that rates > 1 liter/hr will be achieved except under laboratory conditions.

Because these rates of fluid ingestion are less than sweat rates voluntary dehydration develops (Table 10.2), at least during those activities lasting less than about 6 hr. In longer events, especially those lasting many days, there is a tendency for body weight to increase during exercise.

A striking feature of Table 10.2, not previously noted, is the remarkable constancy of the weight loss (2–3 kg) experienced by athletes during prolonged exercise. This appears to be relatively independent of either the type or duration of the activity. It is as if total weight loss during exercise is a regulated variable.

Athletes develop symptoms of discomfort when they attempt to drink at rates equal to the higher sweat rates. Thus, both runners [33] and cyclists [106] develop symptoms of “fullness” when ingesting fluid a
### TABLE 10.1
Reported Levels of Dehydration Developing in Athletes Competing in Running, Triathlon, and Cycling Races of Different Distances

<table>
<thead>
<tr>
<th>Race Distance (km)</th>
<th>% Wt Loss</th>
<th>Kg Loss/Gain</th>
<th>Reference</th>
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<tr>
<td>Running</td>
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<td>32</td>
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<td>37</td>
</tr>
<tr>
<td>90</td>
<td>3.5</td>
<td>2.7</td>
<td>90</td>
</tr>
<tr>
<td>100–160</td>
<td>0.6</td>
<td>0.5</td>
<td>114</td>
</tr>
<tr>
<td>24-hr race (132–188 km)</td>
<td>0.6</td>
<td>0.5</td>
<td>61</td>
</tr>
<tr>
<td>24-hr race (121–249 km)</td>
<td>3.2</td>
<td>2.1</td>
<td>60</td>
</tr>
<tr>
<td>24-hr race (160–199 km)</td>
<td>3.0</td>
<td>2.7</td>
<td>123</td>
</tr>
<tr>
<td>960 km (5 days)</td>
<td>0.8</td>
<td>0.5</td>
<td>143</td>
</tr>
</tbody>
</table>

**Triathlon**

<table>
<thead>
<tr>
<th>km</th>
<th>% Wt Loss</th>
<th>Kg Loss/Gain</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>52</td>
<td>4.0</td>
<td>2.9</td>
<td>105</td>
</tr>
<tr>
<td>52</td>
<td>1.7</td>
<td>1.2</td>
<td>134</td>
</tr>
<tr>
<td>110</td>
<td>3.3</td>
<td>2.3</td>
<td>158</td>
</tr>
<tr>
<td>115</td>
<td>2.8</td>
<td>2.1</td>
<td>134</td>
</tr>
<tr>
<td>160</td>
<td>4.5</td>
<td>3.2</td>
<td>160</td>
</tr>
<tr>
<td>174</td>
<td>3.2</td>
<td>2.2</td>
<td>61</td>
</tr>
<tr>
<td>226</td>
<td>4.1 (male)†</td>
<td>3.2 (male)</td>
<td>58</td>
</tr>
<tr>
<td>226</td>
<td>3.6‡</td>
<td>2.8</td>
<td>50</td>
</tr>
<tr>
<td>226</td>
<td>3.7</td>
<td>2.8</td>
<td>134</td>
</tr>
<tr>
<td>Cycling race</td>
<td>+3.5</td>
<td>+2.8</td>
<td>96</td>
</tr>
</tbody>
</table>

§Kg Loss/Gain during exercise.

Note that weight loss is greater among males than females.

Most of weight loss in the triathlon occurred in the run section (1.6 kg vs. 0.7 kg in cycling) despite the shorter duration of the run (04:34 vs. 06:08).

### TABLE 10.2
Reported Rates of Fluid Intake of Athletes Competing in Running, Triathlon, and Cycling Races of Different Distances

<table>
<thead>
<tr>
<th>Distance/Intensity/Duration</th>
<th>Mean Rate of Fluid Intake (liters/hr)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>(km) (% VO₂max)⁴ (hr.min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Running</td>
<td></td>
<td></td>
</tr>
<tr>
<td>32</td>
<td>0.05</td>
<td>140</td>
</tr>
<tr>
<td>42</td>
<td>0.15</td>
<td>160</td>
</tr>
<tr>
<td>42</td>
<td>0.40</td>
<td>29</td>
</tr>
<tr>
<td>42</td>
<td>0.41</td>
<td>97</td>
</tr>
<tr>
<td>42</td>
<td>0.19</td>
<td>98</td>
</tr>
<tr>
<td>42</td>
<td>0.38</td>
<td>99</td>
</tr>
<tr>
<td>42</td>
<td>0.45–0.49</td>
<td>121</td>
</tr>
<tr>
<td>42</td>
<td>0.48–0.59</td>
<td>121</td>
</tr>
<tr>
<td>42</td>
<td>0.60</td>
<td>125</td>
</tr>
<tr>
<td>42</td>
<td>0.12</td>
<td>138</td>
</tr>
<tr>
<td>42</td>
<td>0.15</td>
<td>140</td>
</tr>
<tr>
<td>56</td>
<td>0.49–0.62</td>
<td>121</td>
</tr>
<tr>
<td>56</td>
<td>0.40–0.46</td>
<td>121</td>
</tr>
<tr>
<td>67</td>
<td>0.40 (male)</td>
<td>139</td>
</tr>
<tr>
<td>67</td>
<td>0.31 (female)</td>
<td>139</td>
</tr>
<tr>
<td>80</td>
<td>0.50</td>
<td>145</td>
</tr>
<tr>
<td>90</td>
<td>0.48</td>
<td>37</td>
</tr>
<tr>
<td>960</td>
<td>0.53</td>
<td>143</td>
</tr>
<tr>
<td>Running (Laboratory)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>70% VO₂max × 02:00</td>
<td>0.08–0.12</td>
<td>16</td>
</tr>
<tr>
<td>70% VO₂max × 04:00</td>
<td>0.21</td>
<td>41</td>
</tr>
<tr>
<td>71% VO₂max × 02:00</td>
<td>0.88</td>
<td>33</td>
</tr>
<tr>
<td>75% VO₂max × 02:00</td>
<td>0.60</td>
<td>65</td>
</tr>
<tr>
<td>76% VO₂max × 03 km</td>
<td>0.48</td>
<td>106</td>
</tr>
<tr>
<td>Triathlon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>52</td>
<td>0.6</td>
<td>105</td>
</tr>
<tr>
<td>Cycling (Competition)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 800 km</td>
<td>0.62</td>
<td>96</td>
</tr>
<tr>
<td>55% VO₂max × 06:00</td>
<td>1.2</td>
<td>14</td>
</tr>
<tr>
<td>60% VO₂max × 03:00</td>
<td>0.9</td>
<td>26</td>
</tr>
<tr>
<td>60% VO₂max × 03:00</td>
<td>1.1</td>
<td>144</td>
</tr>
<tr>
<td>70% VO₂max × 02:00</td>
<td>0.8–1.6</td>
<td>106</td>
</tr>
<tr>
<td>Rest</td>
<td>00:40</td>
<td>1.5</td>
</tr>
</tbody>
</table>

⁴VO₂max = maximum oxygen consumption.

rates equal to or greater than 800 ml/hr. Runners were unable to sustain these high rates even for 2 hr [33] whereas cyclists reported increasing levels of discomfort when ingestion rates were greater than 800 ml/h [106]. One-half of the cyclists ingesting 1200 ml/hr and all subject ingesting 1600 ml/hr were “visibly uncomfortable.” Interestingly, 25% of subjects developed diarrhea when ingesting 1.6 liters/hr [106] indicating...
that this rate of fluid ingestion exceeded the combined maximum rate of fluid absorption of both the small and large bowels.

Brouns et al. [18] showed that the rate of fluid ingestion of subjects encouraged to drink as much as possible during a simulated triathlon, was two to three times higher in the cycling leg (600–800 ml/hr) than in the running leg (100–300 ml/hr). This suggests that running reduces the desire to drink more than does cycling [19].

Hence, peak rates of fluid ingestion under ideal conditions are seldom greater than 800 ml/hr. Feelings of abdominal fullness seem to prevent higher rates of ingestion, especially during running.

Whereas this upper limit of voluntary fluid ingestion during exercise may be set by progressive gastric distension due to impaired gastric emptying [106], another possibility is that fluid ingested at such high rates may also accumulate at other sites, including the colon. The assumption that the small bowel has an unlimited capacity for both fluid and energy assimilation is currently under review [45]. Indeed, Budington and Diamond [21] have suggested: "... the intestine never possesses an enormous excess absorptive capacity. Instead, just enough intestine and transporters are synthesized to absorb the expected nutrient loads so that biosynthetic energy is not wasted on unneeded issue and molecules but ingested nutrients are not wasted either. The intestine exemplifies the motto, 'enough but not too much.'"

Their suggestion is that the maximum absorptive capacity of the small bowel at least for glucose is approximately twice the usual carbohydrate intake. If this holds also for fluid absorption, and if the usual fluid intake of humans is 180–200 ml/hr [56], then the maximum rates of small bowel fluid absorption might be approximately only 360–400 ml/hr. Interestingly, this value approximates the voluntary rates of fluid ingestion of most subjects during exercise (Table 10.2), but is well below both their average sweat rates [121] and the maximum rates of fluid ingestion of some athletes who develop fluid retention during prolonged exercise [85].

At present, the maximum rates of small bowel fluid and glucose absorption either at rest or during exercise are not known. There is sufficient information, however, to suggest that these rates may be sufficiently low to limit optimum fluid and carbohydrate replacement during exercise.

Thus, a number of studies [74, 75, 109, 139, 141] have shown that less than about 40% of the carbohydrate ingested during exercise is oxidized by the muscles, possibly because intestinal glucose absorption is limiting. Peak rates of exogenous glucose oxidation by muscle are approximately 1 g/min regardless of the type of carbohydrate ingested [75]. This rate is equal to that given historically as the maximum rate of glucose absorption from the small bowel [1, 72]. As glucose absorption stimulates water absorption [65, 66], low rates of glucose absorption might indicate that water absorption also occurs relatively slowly. Indeed, Gisolfi et al. [66] have also calculated that only 37% of fluid infused into the duodenum and jejunum at rates of 900 ml/hr was absorbed; others have reported that the maximum rate of water absorption from an isotonic solution with a sodium concentration of 110 mmol/liter was only 800 ml/hr [42].

Similarly, studies in which fluid replacement either during [14] or after exercise [34] equaled fluid losses found that not all the ingested fluid could be accounted for by its appearance in the extracellular or intracellular fluid pools, suggesting that not all was rapidly absorbed [116]. Indeed, Costill and Sparks [34] suggested that: "a large fraction of the ingested water remained in the gastrointestinal tract or was shifted to the extravascular compartment."

In summary, the maximum rates of fluid absorption by the small bowel during exercise are not known but could be less than either the highest rates of fluid loss incurred by some athletes during more intensive exercise, or the highest rates of fluid ingestion of some less competitive runners especially during prolonged exercise [85]. Any unabsorbed fluid would accumulate in the large bowel where its presence might: (a) explain the symptoms of fullness experienced by some athletes ingesting large fluid volumes during prolonged exercise, and (b) lead to a potential "third space" effect that could be a factor in the etiology of the hyponatremia of exercise (see later).

Clearly, unless the maximum rates of fluid absorption by the small bowel are known, it is difficult to prescribe how much fluid should be ingested during exercise [116]. But, if intestinal absorptive capacity is regulated by demand and can increase when demand increases chronically [45], it follows that the chronic ingestion of large volumes of fluid either during or after exercise might increase intestinal absorptive capacity.

Influence of Changes in Serum Osmolality, Plasma Volume, and Other Factors on Drinking Behavior in Humans

In contrast to dogs [4, 159], the donkey (burro) [46], the monkey [11] and the Bedouin goat [27], only humans develop voluntary dehydration when given free access to fluid either during or after exercise in the heat. Other mammals immediately replace all their fluid losses as soon as water is provided. The volume ingested by the dog appears to be regulated by receptors in the oropharynx [6, 159]. Drinking rapidly inhibits ADH release, probably in response to an oropharyngeal reflex [11].

Dill [46] proposed that humans develop voluntary dehydration during exercise because only they lose sodium chloride in sweat. As a result, serum osmolality rises less during exercise-induced dehydration in humans. But, as all mammals seemed to drink sufficiently to maintain constant serum osmolality, Dill [46] postulated that the fluid intake of humans would always be inadequate to replace their sweat losses.
Nose et al. [130] have shown that the drinking behavior of dehydrated humans is regulated by changes in both serum osmolality and plasma volume. Hence, dipogenic drive in dehydrated humans ceases when either osmolality or plasma volume is corrected by the ingestion of either plain water or a sodium-containing solution, respectively. Thus, the ingestion of plain water prematurely inhibits drinking by causing serum osmolality to return to isosmotic before either fluid or sodium losses are replaced; sodium chloride ingestion also terminates drinking prematurely by restoring plasma volume prematurely. But changes in osmolality and plasma volume during exercise-induced dehydration in humans are not independent of each other; the rising serum osmolality with dehydration acts to maintain plasma volume and to reduce the volume-dependent drive for fluid replacement [130]. The result is that whether or not they ingest plain water or sodium chloride solutions, dehydrated humans stop drinking before they are fully rehydrated.

In addition, water ingestion increases free water loss [130] whereas elective restoration of plasma volume with sodium-containing solutions inhibits secretion of sodium-retaining hormones [131], increasing osmotic diuresis. Perhaps it is these complex interactions that also explain why humans are unable to prevent the development of voluntary dehydration during exercise. Others suggest that it is the rapid alleviation of the symptoms that initiate drinking including, among others, dryness of the mouth, which causes the premature cessation of drinking before full rehydration has occurred [84, 146]. Indeed, subjects can be classified as either reluctant” or “avid” drinkers [153, 154] on the basis of the volume of fluid they ingest during exercise. Fluid ingestion during exercise is enhanced by drinking cold, sweet fluids [56, 84, 146]. Simultaneous food consumption also increases fluid consumption [5, 84].

There is a need to establish factors that will enhance fluid ingestion during exercise, thereby limiting the extent to which voluntary dehydration develops.

Role of Sodium Chloride Ingestion

The predictions of Adolph and Dill [7] that the fluid deficit that develops during exercise is corrected only when the sodium chloride deficit has been corrected so that “water cannot be held until the missing osmoles re made good” [92] has been confirmed by the detailed studies of Nose and colleagues [112, 129–131, 133]. The essential conclusion from these studies is that the sodium content of the extracellular space must regulate the extracellular fluid volume [112, 129]. As a result, the extracellular fluid volume must contract whenever a sodium deficiency develops. This explains why serum sodium concentrations remain constant (or increase slightly) during prolonged exercise when both sodium and water are lost, even if plain water in limited amounts is replaced [120].

There are two exceptions to this general rule that the extracellular fluid volume is regulated by its sodium chloride content so that serum sodium concentration will remain within a narrow range regardless of the degree of fluid or sodium deficit. The first is the condition of true sodium chloride deficiency [102–104]; the other is the hyponatremia of exercise [120]. These are discussed in subsequent sections. In both conditions, the volume of the extracellular space is increased out of proportion to its sodium chloride content.

Nose et al. [129] also found that, at any level of dehydration, the less sodium chloride lost in sweat, the greater the loss of fluid from the intracellular compartment. Thus, a reduction of sweat sodium losses with protection of the circulating volume is an important benefit of heat acclimatization and training.

It follows that an important benefit of ingesting sodium chloride during or after exercise might be better maintenance and more rapid restoration of the extracellular volume and possibly also the plasma volume [17, 23, 34, 116] although this is not always found [100, 137]. This effect would be enhanced by the addition of carbohydrate, which further increases the rate of fluid absorption from the solution ingested during exercise [67].

Figure 10.2 shows the rates of water, sodium, and carbohydrate absorption from different solutions ingested at rest. It shows that optimum fluid absorption occurs from isotonic carbohydrate/electrolyte solutions, whereas rates of absorption from water and electrolyte-containing solutions are not greatly different. Sodium absorption, however, is enhanced from electrolyte-containing solutions. This latter finding would explain why sodium-containing solutions are more likely to maintain plasma volume whereas the ingestion of water is more likely to influence plasma osmolality.

On the other hand, carbohydrate absorption is most rapid from concentrated (10%) carbohydrate solutions. But these solutions induce net water movement into the small bowel and could, therefore, be a factor contributing to the hyponatremia of exercise (see later).

APPLIED MEDICAL ASPECTS

“Salt-Deficiency” vs. “Water-Deficiency” Heat Exhaustion

An original classification of the heat illnesses distinguishes “salt-deficiency heat exhaustion” from “water-deficiency heat exhaustion” [28]. This distinction has persisted in the literature [83, 94]. Salt deficiency heat exhaustion is considered to develop when fluid replace-
FIGURE 10.2

Fluid Replacement During Exercise | 317

ment is adequate but replacement of salt is inadequate; water-deficiency heat exhaustion is believed to result from the converse. But if body sodium content regulates the extracellular fluid volume [112, 129–131], it is difficult to understand how there could be these distinct conditions. Nor do the accepted descriptions of the two conditions provide robust guidelines for their differentiation on either clinical or biochemical ground [83, 94].

The original distinction was made on the basis of an experimental model developed to induce salt deficiency in animals. In this model [38, 54, 81, 111], true sodium chloride deficiency was induced by injecting a 5% glucose solution into the peritoneal cavity of experimental animals; the solution that was withdrawn 4–5 hr later had a high sodium chloride content but a low protein content. The characteristics of this salt-deficiency syndrome were the following: hypotonic hyponatremia; a large fluid shift from the extracellular to the intracellular fluid compartments causing intracellular overhydration; and extracellular dehydration with a disproportionate reduction in plasma volume. The fall in plasma volume was greater than that developing in pure water loss and was associated with a large reduction in the total circulating protein content. Circulatory changes were more severe in salt-deficiency than in water-deficiency dehydration; animals with salt depletion were incapacitated whereas animals with pure water deficiency remained “vigorously healthy” with no evidence of peripheral vascular collapse.

Elkinton et al. [54] concluded that the greater loss of circulating plasma protein content probably explained the larger fall in plasma volume and the more severe circulatory collapse with salt depletion than with water depletion. They concluded that “whenever there is any salt loss to the external environment or temporary segregation of salt by pooling in the gut (author’s emphasis), peritoneum, or in the traumatized region, shock will be produced with greater ease than would otherwise be the case.” It is understandable why these original researchers considered that subjects with more severe heat injury must be suffering from salt-deficiency heat exhaustion whereas those with less severe circulatory abnormalities had water deficiency alone.

It is extremely difficult, however, perhaps impossible, for healthy humans to develop salt deficiency regardless of the environment in which they live and the amount of exercise they perform [47, 56, 170]. Indeed, their inability to discover a single case of salt deficiency either in the literature or in their personal experience of 20 yr work in environmental physiology led Epstein and Sohar [56] to suggest that the mythical condition of “salt deficiency heat exhaustion” is another example of “christening by conjecture.” “Such a syndrome” they conclude “has never been proven to exist.”

Rather, sodium chloride deficiency has only ever been induced in healthy humans by combining a salt-free diet with daily exercise or sauna
exposure to increase sodium chloride losses [102–104]. A cardinal feature of the syndrome was severe physical incapacitation that prevented any desire or attempt to exercise.

Figure 10.3 from McCance [102] depicts mean water and sodium balance in subjects from one of their studies. It shows that for the first 2 and the last 4 days of the experiment, water balance was maintained, but from days 3–12, relative water conservation occurred despite a continuing sodium deficit. Hyponatremia developed during this period and became progressively more severe despite a contraction of the extracellular fluid volume by up to 38% [104].

These findings can be explained if the initial response to human salt deficiency is a contraction of the extracellular fluid volume with maintenance of a normal or elevated serum sodium concentration (days 0–2 on Fig. 10.2); thereafter there is relative conservation of the extracellular fluid volume despite increasing sodium deficit. During this period, hyponatremia develops. McCance [102] concluded: "... the human body comprised between (a) maintenance of its total osmotic pressure at the expense of anhydremia, a reduction of blood volume, rise of haemoglobin, proteins and colloidal osmotic pressure in the serum, and (b) maintenance of its plasma and extracellular fluid volumes at the expense of a reduction in the concentrations of sodium and chloride in the serum, with a fall in its total osmotic pressure." The important point is that because the extracellular sodium content determines the extracellular fluid volume in health [112, 129, 130], any fall in serum sodium concentration can occur only if this interdependent relationship is lost so that the normal regulation of the extracellular fluid volume is disturbed.

The particular relevance of these studies is that they add important insights into the condition of the hyponatremia of exercise. This condition, which is reviewed in detail elsewhere [120], occurs typically in persons involved in very prolonged exercise during which they ingest large volumes of fluid, frequently containing 5–10% carbohydrate. Although it has been argued that large sodium chloride losses in sweat contribute to the pathogenesis of this condition [76–79], studies of runners with this condition have established that abnormal fluid retention, and not sodium deficiency, is the sine qua non for the development of the serious symptomatic form of this condition [85].

One proposal is that a third space effect similar to that produced in the experimental model of salt deficiency, may be operative. A large (±2 liters) volume of unabsorbed fluid in the gut could, particularly if it has a high carbohydrate content, induce a sufficiently large sodium movement from the extracellular space into the unabsorbed fluid [66, 67, 139] to produce the changes found in experimental sodium chloride deficiency with hypotonic hyponatremia, intracellular overhydration, and a fall in plasma volume and in circulating protein content [120]. It is possible that sustained high rates of fluid intake (> 1 liter/hr), achieved uniquely only by some athletes during prolonged exercise [85] could exceed the fluid-absorbing capacity of the small bowel leading to this third space effect in the large bowel.

In summary, the hyponatremia of exercise appears to be due to the combination of at least three abnormalities: abnormal fluid retention, possibly on the basis of inappropriate ADH secretion; disturbed regulation of the extracellular fluid volume so that the normal relationship between its volume and sodium chloride content is lost, and possibly a third space effect with movement of sodium into unabsorbed fluid in the large bowel.

The sequence in which these abnormalities develops is not known. But the similarities between the hyponatremia of exercise and the experimental model of salt deficiency cannot be ignored [120]. This suggests that a third space effect may be crucial for the development of this condition.
Exercise-associated Collapse

After it had been found that rectal temperature rose as an apparently linear function of dehydration in marathon runners [169], it took only a small leap of faith to conclude that athletes who collapse after prolonged exercise must be suffering from a heat disorder caused by dehydration. As a result, it became fashionable to treat all athletes who collapsed during or after prolonged exercise with intravenous fluids [3, 51, 76–79, 93, 95, 132, 167].

But recent studies have shown that runners with this condition of exercise-associated collapse (EAC) [142] are not more likely to be hyperthermic than are control runners who do not collapse; indeed, a significant proportion may be hypothermic (Holzhausen L.M., and Noakes, T.D. Unpublished observations). Nor is there any evidence that subjects with EAC are more dehydrated than are noncollapsing controls [118, 122]. In addition, intravenous fluid therapy retards rather than expedites the recovery of these subjects [55]; this form of therapy may also induce iatrogenic hypotension and hyperglycemia [122] and can be life-threatening in those with symptomatic hypotension due to fluid overload [85, 126].

On the basis of the evidence that: (a) the majority (> 85%) of subjects with EAC collapse after the cessation of exercise (Holzhausen, L.M., and Noakes, T.D. Unpublished observations), and (b) that postural hypotension, often severe, is present in virtually all runners who complete ultramarathon races [80], we have proposed that postural hypotension probably caused by fluid displacement to the compliant veins of the lower limb, is the most likely cause of EAC [80, 118, 119]. This is not a novel proposal. For nearly 70 years it has been known that postural hypotension occurs frequently during exercise in the heat and is the principal abnormality in the condition incorrectly termed “heat exhaustion” [8, 49, 50, 155].

As the degree of postexercise postural hypotension is unrelated to the level of dehydration developed during exercise [80], it follows that intravenous fluid therapy is not the most rational form of therapy for EAC. Rather, therapy should aim to expedite return of blood from the peripheral pools in the lower limbs [19, 118]. This can be achieved by elevating the pelvis and legs; a form of therapy that has proven effective [68]. Failure to respond to this form of therapy may indicate the presence of dehydration.

Two additional points require emphasis. First, the most severe levels of exercise-induced dehydration yet reported [5] did not cause sudden collapse with circulatory failure, at least in the recumbent position. Rather, severely dehydrated subjects chose to lie down, possibly because they developed postural hypotension on standing. Hence, the development of postural hypotension could perhaps be the safety mechanism that prevents dehydrated subjects from continuing to exercise to levels of dehydration that would induce irreversible circulatory failure.

Second, Adolph [5] observed that blood biochemical markers were poor indicators of the level of dehydration, as also recently confirmed by us [80]. Adolph therefore proposed that the level of dehydration should be determined by conventional clinical parameters. In particular, intense thirst, a change in the timbre of the voice, a dry mouth, and an inability to produce sputum indicated more severe dehydration.

In our experience, these are extremely uncommon findings in subjects whether or not they collapse after prolonged exercise. This observation is compatible with the finding that more severe levels of dehydration occur uncommonly in endurance athletes (Table 10.1).

**SUMMARY**

Current evidence indicates that adequate fluid ingestion during exercise enhances athletic performance, prevents a fall in plasma volume, stroke volume, cardiac output and skin blood flow, maintains serum sodium concentrations and serum osmolality, lowers rectal temperature and the perception of effort, and prevents a progressive rise in heart rate. Rates of sweating and urine flow are not influenced by fluid ingestion. The evidence suggests that the maintenance of serum osmolality and serum sodium concentrations at pre-exercise levels is the important determinant of these beneficial effects of fluid ingestion on cardiovascular function and thermoregulation. The provision of glucose in the ingested solution may be necessary to optimize performance; glucose ingestion that enhances fluid and sodium absorption in the small bowel may also prevent a progressive rise in oxygen consumption during exercise. Sweetened carbohydrate-containing drinks may also increase fluid intake during exercise, thereby minimizing voluntary dehydration.

Hence, the optimum solution for ingestion during exercise should provide carbohydrate, probably at rates of about 1 g/min and electrolytes in concentrations that, when drunk at the optimum rate, maintain serum osmolality and plasma volume at pre-exercise levels by replacing exactly the water and electrolyte losses from the extracellular space. At present, the composition of the fluid that will optimize electrolyte and fluid replacement of the extracellular space is not established. Neither are the optimum rates of fluid ingestion during exercise known. At low sweat rates (< 1 liter/hr), it is probable that all of the lost fluid can and should be replaced; rates of fluid ingestion needed to offset higher sweat rates may exceed the maximum intestinal absorptive capacity for water. Furthermore, high rates of fluid intake (> 1 liter/hr) are achieved with difficulty during exercise, especially when running, and are likely
to lead to feelings of abdominal discomfort, possibly due to the accumulation of unabsorbed fluid in the small bowel or colon. Practicing to drink regularly during training might reduce the severity and frequency of these symptoms, possibly by increasing intestinal absorptive capacity.

Most athletes are "reluctant" drinkers during exercise and do not ingest fluid at rates equal to their rates of fluid loss; hence, they develop progressive (voluntary) dehydration during prolonged exercise. Surprisingly, the level of voluntary dehydration that develops during exercise is relatively independent of the duration or intensity of the activity. The factors that explain these phenomena remain elusive. Fluid consumption during exercise is enhanced by the ingestion of cold, sweet fluids. Simultaneous food consumption also stimulates fluid ingestion.

Despite the modest sodium losses in sweat, the serum sodium concentration remains relatively unchanged during exercise. This results from a regulated contraction of the extracellular volume, which develops in proportion to the sodium deficit. As its sodium content regulates the volume of the extracellular space, correction of any fluid loss incurred during exercise requires that electrolyte losses, especially sodium, also be corrected.

Failure of adequate contraction of the extracellular volume in response to sweat sodium losses is characteristic of the hyponatremia of exercise in which there is also abnormal fluid retention in subjects who sustain very high rates of fluid intake (> 1.3 liters/hr) for prolonged periods (> 6 hr). These subjects exhibit the extreme of "avid" drinking behavior during exercise.

It is frequently assumed that severe dehydration is the critical factor explaining the development of both exercise-induced heatstroke and the apparently related condition of EAC. Yet there is no evidence that dehydration plays an essential role in either condition. EAC, in particular, is not a heat disorder and appears to be caused by the rapid onset of postural hypotension with the cessation of exercise. Elevation of the lower limbs, rather than intravenous fluid therapy, is the initial treatment of choice.

Future research needs to establish (a) techniques that will increase the fluid intakes of "reluctant" drinkers during exercise so that the extent of the voluntary dehydration can be reduced or prevented; and (b) the nature of the ingested solution that will optimize maintenance of serum osmolality and plasma volume during exercise under different exercise conditions and in different individuals whose sweat sodium and water losses, and rates of intestinal absorption of electrolytes and water may differ substantially as a result of individual variability and levels of training. Indeed, the complex interrelationship between all of these diverse variables remains to be clarified.

ACKNOWLEDGMENTS

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