Postexercise rehydration: effect of Na\textsuperscript{+} and volume on restoration of fluid spaces and cardiovascular function

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Received 5 October 1999; accepted in final form 9 May 2000

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Mitchell, J. B., M. D. Phillips, S. P. Mercer, H. L. Baylies, and F. X. Pizza. Postexercise rehydration: effect of Na\textsuperscript{+} and volume on restoration of fluid spaces and cardiovascular function. J Appl Physiol 89: 1302–1309, 2000.—Our purpose was to study the interaction between Na\textsuperscript{+} content and fluid volume on rehydration (RH) and restoration of fluid spaces and cardiovascular (CV) function. Ten men completed four trials in which they exercised in a 35°C environment until dehydrated by 2.9% body mass, were rehydrated for 180 min, and exercised for an additional 20 min. Four RH regimens were tested: low volume (100% fluid replacement)-low Na\textsuperscript{+} (LL), low volume-high (50 mM) Na\textsuperscript{+} (LH), high volume (150% fluid replacement)-low Na\textsuperscript{+} (HL), and high volume-high Na\textsuperscript{+} (HH). Blood and urine samples were collected and body mass was measured before and after exercise and every hour during RH. Before and after the dehydration exercise and during the 20 min of exercise after RH, cardiac output was measured. Fluid compartment (intracellular and extracellular) restoration and percent change in plasma volume were calculated using the Cl\textsuperscript{−} and hematocrit/Hb methods, respectively. RH was greater \( (P < 0.05) \) in HL and HH \( (102.0 \pm 15.2 \) and \( 103.7 \pm 14.7\% \), respectively) than in LL and LH \( (70.7 \pm 10.5 \) and \( 75.9 \pm 6.3\% \), respectively). Intracellular RH was greater in HL \( (1.12 \pm 0.4 \) liters) than in all other conditions \( (0.83 \pm 0.3, 0.69 \pm 0.2, \) and \( 0.73 \pm 0.3 \) liter for LL, LH, and HH, respectively), whereas extracellular RH (including plasma volume) was greater in HL and HH \( (1.35 \pm 0.8 \) and \( 1.63 \pm 0.4 \) liters, respectively) than in LL and LH \( (0.83 \pm 0.3 \) and \( 1.05 \pm 0.4 \) liters, respectively). CV function (based on stroke volume, heart rate, and cardiac output) was restored equally in all conditions. These data indicate that greater RH can be achieved through larger volumes of fluid and is not affected by Na\textsuperscript{+} content within the range tested. Higher Na\textsuperscript{+} content favors extracellular fluid filling, whereas intracellular fluid benefits from higher volumes of fluid with lower Na\textsuperscript{+}. Alterations in Na\textsuperscript{+} and/or volume within the range tested do not affect the degree of restoration of CV function.

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was administered in capsule form (equivalent to ~77 mM) during an ad libitum rehydration procedure, it produced an accelerated extracellular recovery compared with plain water. This finding is similar to that reported by Nielsen et al. (15) when a high-Na⁺ (127 mM) solution was used. Both of these levels of Na⁺ are higher than the recommended concentration for a post-exercise oral rehydration solution (4) and may pose a problem with beverage palatability. The use of different volumes of fluid combined with Na⁺ content within recommended concentrations provides an opportunity to investigate the influence of these variables on the movement of fluid into the various body fluid spaces.

The purpose of this investigation was to examine the interaction between Na⁺ content and volume ingested on the postexercise whole body rehydration and the restoration of cardiovascular function and fluid compartments. Specifically, using a two-by-two design, we investigated the ingestion of fluid at 100 and 150% of body mass lost with Na⁺ at 25 and 50 mM. It was hypothesized that the most effective rehydration would occur with the 150%-50 mM treatment and that this would selectively restore the extracellular space to a greater extent than the other treatments. Furthermore, it was hypothesized that the recovery of cardiovascular function would be influenced by the level of rehydration.

**METHODS**

**Subjects.** Ten moderately trained men capable of performing ≥90 min of cycling at 60% of their maximal O₂ power (V̇O₂ max) were chosen as subjects. Their age, weight, and V̇O₂ max were 27.5 ± 5.8 yr, 79.60 ± 11.9 kg, and 3.88 ± 0.47 l/min, respectively. The subjects first completed a medical history questionnaire and then read and signed an institutionally approved informed consent form.

**Experimental design.** The subjects were first tested for V̇O₂ max by use of a standard graded cycle ergometer test, the results of which were used to determine their target sub-maximal workload during the experimental trials. Each subject was then subjected to four rehydration conditions conducted in a randomized counterbalanced design, with each trial separated by ≥1 wk. The following conditions were imposed: 1) low volume (100% of volume lost)-low (25 mM) Na⁺ (LL), 2) low volume-high (50 mM) Na⁺ (LH), 3) high volume-low Na⁺ (HL), and 4) high volume-high Na⁺ (HH). The four conditions consisted of 90 min of exercise-induced dehydration, a 30-min transition period, and 180 min of rehydration. A 20-min steady-state ride was conducted after rehydration to assess the recovery of cardiovascular function. During the 24 h before each condition, diet, fluid intake, and exercise were standardized.

**Experimental testing.** Before exercise, a venous blood sample was taken after 30 min of seated rest. The subjects emptied their bladders, and nude body mass was measured using a digital scale accurate to 20 g. Subjects were fitted with a rectal thermistor inserted to a depth of 12 cm, and a telemetry heart rate monitor (Polar) was placed around the chest. The 90-min exercise bout was conducted in an environmentally controlled chamber adjusted to 35°C and 55% relative humidity. Each subject rode on a Monark cycle ergometer at ~60% of V̇O₂ max until 2.5% of initial body weight was lost or until a core temperature of 39°C was reached. Core temperature was monitored continuously during exercise and recorded every 15 min. During the first and last 10 min of exercise, a series of three CO₂ rebreathing maneuvers were conducted to determine cardiac output. During this same period, O₂ uptake was measured by analysis of expired gases collected in Douglas bags, and heart rates were recorded. Sweat samples were obtained from an arm bag at 20, 50, and 80 min of the ride. The arm was rinsed thoroughly with distilled water and dried with a clean towel before each collection.

Immediately after exercise, the subjects were removed to a normal room temperature environment (23°C, 50% relative humidity). They rested in a seated position for 30 min before rehydration to allow the reversal of the exercise-induced plasma volume shift. At the end of this transition period, a blood sample was taken from an antecubital vein, a urine sample was collected and measured, and nude body mass was obtained. Total dehydration was calculated at this point; thus dehydrations were based on the difference between the initial nude body mass and the body mass at the end of the transition period.

After the transition period, subjects ingested the first drink of the rehydration period, which was administered as a “priming dose” and was 30% of the total to be ingested for the particular condition. The subsequent volumes were administered at 30-min intervals and were one-fifth of the remaining volume to be ingested for each condition (14% of the total volume). In the LL and LH conditions, the total volume consumed equaled the total loss in body mass. In the HL and HH conditions, subjects consumed 1.5 times the body mass loss. The priming dose in the LL and LH conditions averaged 683 ml, and the subsequent volume was 319 ml for a total volume ingested of 2.28 liters. The priming dose in the HL and HH conditions averaged 1,017 ml, and the subsequent volume was 475 ml for a total volume of 3.39 liters. The rehydration solution was a noncommercial beverage containing 50 g/l maltodextrin and 25 or 50 mM Na⁺ for the low- and high-Na⁺ conditions, respectively. A solution containing carbohydrate was used to facilitate intestinal absorption. At 60, 120, and 180 min of rehydration, a blood sample was drawn and urine volume and body weight were measured.

After the 180-min rehydration protocol, the subjects returned to the heat chamber and completed an additional 20 min of exercise at the same intensity used for the dehydration exercise. After a steady state was reached, the CO₂ rebreathing maneuver was again conducted. Heart rates were recorded, and respiratory gases were collected in Douglas bags.

**Blood and fluid analyses.** Whole blood samples were analyzed for hematocrit and Hb by use of the microcapillary tube and cyanmethemoglobin methods, respectively. Plasma samples were obtained and frozen for later analysis of osmolality (freeze-point depression; Advanced Instruments, Norwood, MA), electrolytes (Na⁺, K⁺, and Cl⁻; Nova V Analyzer), and antidiuretic hormone (ADH) by RIA (Diagnostic Products, Los Angeles, CA). Urine and sweat samples were collected, and a sample was frozen for determination of urine electrolytes (Na⁺, K⁺, and Cl⁻; Nova V Analyzer).

**Calculations.** Percent dehydration was calculated as the difference between the prehydration and preexercise body mass divided by the preexercise body mass. Percent rehydration was calculated as the body mass gain during rehydration divided by the total mass lost. Percent change in plasma volume was calculated according to the method of Dill and Costill (3). Sweat loss was calculated as the body mass loss with adjustments made for respiratory water loss and metabolic carbon loss. Na⁺ balance was determined by subtracting absolute Na⁺ loss (sweat and urine volume multiplied by
their respective Na\textsuperscript{+} concentrations) from total Na\textsuperscript{+} intake in the rehydration solutions. Changes in fluid compartments during rehydration were calculated using the Cl\textsuperscript{–} method described by Costill et al. (1) and Nose et al. (17). A Donnan factor of 0.95 was used in this calculation. Absolute plasma volume was assumed to be 7% of preexercise body mass for the purposes of these calculations.

**Statistical analyses.** A three-factor ANOVA for repeated measures was conducted for the majority of the dependent variables. The first factor was “volume” and had two levels (100 and 150%), the second factor was “Na\textsuperscript{+}” and also had two levels (25 and 50 mM), and the third factor was “time” and had various levels depending on the number of samples taken. Variables that were analyzed at only one time point (percent dehydration, fluid compartment data, and Na\textsuperscript{+} balance) were analyzed using a two-factor ANOVA. Differences detected by the ANOVA were analyzed further using a Newman-Keuls post hoc test. Relationships between selected dependent measures were conducted using Pearson product correlations. Significance was accepted at $P < 0.05$.

**RESULTS**

**Rehydration and urine volume results.** The level of rehydration was significantly greater in both high-volume trials than in both low-volume trials (Fig. 1). This difference was present at all three hourly time points. The restoration of plasma volume, expressed as percent change, showed a main effect for Na\textsuperscript{+} with no interaction with time or volume; however, the differences between the LL and HL conditions compared with the LH and HH conditions appeared to be present throughout the rehydration period (Fig. 2). The restoration of extracellular fluid and the interstitial fluid volumes showed a main effect for volume; thus the HL and HH conditions were greater than the LL and LH conditions (Fig. 3). On the other hand, the restoration of intracellular fluid volume showed a significant Na\textsuperscript{+}-by-volume interaction, such that the HL condition was significantly greater than all others. Urine production demonstrated a volume-by-time interaction, with the level of urine production significantly greater in the HL and HH conditions than in the LL and LH conditions at 2 and 3 h of rehydration (Fig. 4).

**Na\textsuperscript{+} results.** The amount of Na\textsuperscript{+} actually consumed varied because of the manipulation of concentration and volume. These values were $65.6 \pm 9.8, 117 \pm 19.4,$
95.4 ± 17.9, and 179 ± 30 mM for the LL, LH, HL, and HH conditions, respectively. Plasma Na⁺ demonstrated only a main effect for time, with an elevation occurring at the postexercise time point for all conditions (Fig. 5). Plasma osmolality showed the same pattern of significance as plasma Na⁺. Urine Na⁺, however, showed an Na⁺-by-time interaction such that the levels in the LH and HH conditions were greater than in the LL and HL conditions but only at 2 and 3 h of rehydration (Fig. 5). The calculated values of Na⁺ balance, expressed as an Na⁺ deficit based on urine and sweat loss vs. intake, showed a main effect for Na⁺ (Fig. 5). The deficit in the LH and HH conditions was significantly less than in the LL and HL conditions, with the deficit in the HH condition approaching zero.

**Cardiovascular, temperature, and hormonal results.**

All three cardiovascular variables showed a significant time effect, with cardiac output and stroke volume decreasing significantly after the dehydration exercise and then returning to preexercise levels after rehydration (Fig. 6). Heart rate demonstrated the opposite response. Core temperature increased in all conditions at the end of the dehydration exercise and again at the end of the 20 min of exercise that followed rehydration (Table 1). None of the conditions varied from each other. ADH increased after exercise; however, none of the conditions were significantly different from each other (Fig. 7).

**DISCUSSION**

The primary findings of this investigation were that when 25 mM Na⁺ was compared with 50 mM Na⁺, the Na⁺ content of the rehydration beverage did not influence whole body rehydration; however, the ingestion of 150 vs. 100% of fluid lost significantly improved the level of rehydration. On the basis of these findings, no interaction between Na⁺ and volume was observed for whole body rehydration. On the other hand, the differ-

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**Fig. 4.** Urine volume. Post Ex, after exercise. *Significant volume × time interaction. HL and HH are different from LL and LH at hours 2 and 3 (P < 0.05).

**Fig. 5.** A: plasma Na⁺. B: absolute urine Na⁺ loss. C: Na⁺ balance. *Significant time effect. Postexercise value is different from all others in all conditions. Na⁺ × time interaction. LH and HH are different from LL and HL at hours 2 and 3 (P < 0.05).
ence in Na\(^+\) content and volume did influence the levels of extracellular vs. intracellular rehydration. Finally, the degree of restoration of cardiovascular function was not affected by the Na\(^+\) content or the volume of fluid ingested.

Rehydration. A variety of rehydration protocols have been reported in the literature over the past several years, many of which have been designed to investigate the influence of electrolyte content and fluid volume on rapid rehydration. Conflicting findings regarding the level of Na\(^+\) and volume necessary to produce optimal rehydration have been reported; however, variations in the rehydration protocols employed may partially explain the discrepancies (2, 5, 7, 15, 17). Although many studies have employed serial feedings, the protocols typically employed by Maughan et al. (8–10) and Shirreffs et al. (20) involve the ingestion of large volumes of fluid (2–3 liters) in a relatively short (30–60 min) period of time. In the present study, the volume of fluid was consumed over a 2.5-h period in serial feedings every 30 min. This protocol more closely approximates ingestion patterns that might be used in actual practice, since extreme stomach fullness would be avoided. On the other hand, a significant strength of the studies by Maughan et al. is that their subjects were followed for 5.5–6.0 h, thus providing a more complete picture of the effect of the fluid consumed on kidney function.

Previous work from our laboratory (11) and by Maughan et al. (9) has shown that, to maximize rehydration, a volume of fluid of ≥150% of that lost must be ingested. In addition, the solution must contain an amount of Na\(^+\) or other cation sufficient to prevent significant urine production. When Na\(^+\) levels are relatively low, even large volumes of fluid do not appear adequate to produce rapid rehydration. With the ingestion of 14 mM Na\(^+\) (volume = 150% of fluid lost), Mitchell et al. (11) achieved only 73% rehydration after 3 h of rehydration, despite ingestion of 150% of fluid lost. Maughan and Leiper (8) showed that, 5.5 h after ingestion of 150% of fluid lost at 26 mM Na\(^+\), subjects had retained only 53% of that consumed, thus achieving ~80% rehydration. Even with volumes of 150 and 200% of that lost, after 6 h, Shirreffs et al. (20) reported that fluid balance was not achieved with a 23 mM Na\(^+\) solution. These findings would suggest that the optimal Na\(^+\) concentration is somewhere above 25 mM. Conversely, in the present investigation, increasing Na\(^+\) concentration from 25 to 50 mM did not improve rehydration, suggesting that no further benefits are gained by doubling the concentration. It may be that the discrepancy can be explained by the use of a 3-h protocol compared with a 5- or 6-h protocol; however, direct comparisons are not possible, because the difference in the ingestion pattern may influence urine production and the associated percent rehydration. The final 2–4 h of the longer protocols by Maughan and Leiper (8) produced relatively small changes in net fluid balance; thus the duration of the protocol may not be an issue. Furthermore, in general agreement with the present findings, Wemple et al. (21) found that the

![Fig. 6. A: cardiac output. B: heart rate. C: stroke volume. Rehydr, rehydration. *Significant time effect (P < 0.05). Postexercise value is different from all others in all conditions.](image-url)
increase in Na\(^+\) from 25 to 50 mM did not produce an enhanced whole body rehydration during a 3-h protocol.

The present findings are not in agreement with the concept that Na\(^+\) replacement must occur to accomplish complete rehydration (8). The relatively large Na\(^+\) deficit in the HL condition did not interfere with fluid restoration, since 100% rehydration was achieved. Because of the manipulation of volume and Na\(^+\) concentration, the LH condition actually produced a smaller Na\(^+\) deficit than the HL condition. This combination did not produce a significantly greater fluid replacement than the other low-volume condition (LL), which resulted in the largest deficit. Calculations of Na\(^+\) balance are, of course, influenced by sweat Na\(^+\) concentrations, which vary between subjects and according to the sampling site. The use of arm bag sweat collection may have led to relatively high sweat Na\(^+\) concentrations, which may have exaggerated the calculated deficit in all conditions. Regardless of the magnitude of the Na\(^+\) deficit, however, the present data did not produce a graded response that would indicate a relationship between Na\(^+\) replacement and fluid gain.

Limited work has been conducted to investigate fluid compartment restoration during postexercise rehydration; however, Nose et al. (17) and Nielsen et al. (15) showed that high Na\(^+\) concentrations favor extracellular refilling. In both cases, the Na\(^+\) levels were quite high, with ~70 and 128 mM used by Nose et al. (18) and Nielsen et al., respectively. In the present investigation, the main effect for volume, with no Na\(^+\)-by-volume interaction, suggests that the difference between 25 and 50 mM was not great enough to influence the degree of extracellular restoration. Furthermore, it may be that 25 mM is an adequate level of Na\(^+\), and once this level is reached, the introduction of a greater volume of available fluid then becomes a more powerful factor in extracellular rehydration. From a practical point of view, extremely high Na\(^+\) concentrations may present a palatability problem; thus the lower end of the functional range would be preferable. Relative to intracellular rehydration, the findings differ from those of the extracellular compartment. The greater intracellular restoration in the HL condition suggests that when volume is high and Na\(^+\) is low, the cells are able to take advantage of the greater fluid availability and the absence of the Na\(^+\)-induced osmotic forces that favor the extracellular space. Of the combinations of Na\(^+\) and volume tested in this study, none of which were specifically designed to promote intracellular rehydration, the HL condition appears to be the most effective for intracellular rehydration. Other combinations of cations ingested with different volumes should be examined to determine an optimal combination for maximizing intracellular restoration.

The Na\(^+\) main effect found for plasma volume restoration suggests that, for the vascular space, Na\(^+\) was a more significant factor than volume; however, complete restoration occurred in all trials by 2 h of recovery. In fact, in the high-Na\(^+\) conditions, there was actually an expansion of plasma volume above the preexercise level, an effect that can probably be attributed to the osmotic force created by the presence of Na\(^+\). The selective restoration of plasma volume, particularly in the presence of high Na\(^+\), has been reported by others (2, 18, 21). Although it might be assumed that Na\(^+\) intake would have an effect on plasma Na\(^+\) and/or osmolality, which in turn would affect plasma volume, neither variable exhibited condition differences as a function of Na\(^+\) concentration. This is likely due to the fact that the restoration of plasma volume comes about in an attempt to normalize plasma Na\(^+\) and osmolality; thus what we observed is

### Table 1. Core temperature responses

<table>
<thead>
<tr>
<th>Condition</th>
<th>Preexercise</th>
<th>Postexercise</th>
<th>Postrehydration</th>
<th>Postexercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>LL</td>
<td>36.88 ± 0.09</td>
<td>38.87 ± 0.08*</td>
<td>37.13 ± 0.08</td>
<td>37.43 ± 0.08†</td>
</tr>
<tr>
<td>LH</td>
<td>36.97 ± 0.09</td>
<td>38.68 ± 0.08*</td>
<td>36.91 ± 0.05</td>
<td>37.32 ± 0.07†</td>
</tr>
<tr>
<td>HL</td>
<td>36.86 ± 0.07</td>
<td>38.74 ± 0.12*</td>
<td>36.97 ± 0.05</td>
<td>37.32 ± 0.07†</td>
</tr>
<tr>
<td>HH</td>
<td>36.82 ± 0.09</td>
<td>38.82 ± 0.13*</td>
<td>37.02 ± 0.07</td>
<td>37.32 ± 0.07†</td>
</tr>
</tbody>
</table>

Values are means ± SE in °C. LL, low volume (100% fluid replacement)-low (25 mM) Na\(^+\); LH, low volume-high (50 mM) Na\(^+\); HL, high volume (150% fluid replacement)-low Na\(^+\); HH, high volume-high Na\(^+\). *Significantly different from all others; †significantly different from both preexercise values, P < 0.05.

*Fig. 7. Antidiuretic hormone. *Significant time effect (P < 0.05). Postexercise value is different from all others in all conditions.
the outcome of the normalization process. It appears that the vascular space is more sensitive to the influence of Na\(^+\) than the extracellular fluid as a whole, since, in the latter, significant increases occurred only in combination with higher volumes of fluid ingested.

*Urine and hormonal results.** Numerous studies have shown that the primary obstacle to rapid rehydration is the loss of large amounts of fluid in the form of urine. As mentioned previously, if Na\(^+\) or some other cation is added, kidney function and the concomitant hormonal responses may be altered so that fluid is retained (8, 20). The poor rehydration reported by Mitchell et al. (11) with a 14 mM solution was due to high urine volumes. Shirreffs et al. (20) also showed that urine production tended to be greater with rehydration with 23 than with 61 mM Na\(^+\); thus it was advantageous to increase Na\(^+\) above 25 mM. In the present study, the urine results showed only a main effect for volume; thus there was no advantage to increasing Na\(^+\) above 25 mM.

The activity of fluid-regulating hormones such as ADH is important in providing a mechanism for the control of urine production. It is known that ADH release is stimulated by increased osmolality, which is detected by receptors in the hypothalamus (13, 16, 19). Extracellular Na\(^+\) concentration plays a major role in determining the osmolality; thus any dilution of the extracellular space will signal the inhibition of ADH release, which, in turn, promotes water loss via the increased permeability of the distal tubule. This scenario explains the large urine production and poor rehydration observed with the consumption of dilute oral rehydration solutions. As expected, exercise-induced dehydration elicited an increase in ADH levels in the present study; however, at 1 h of rehydration, ADH levels were not significantly different from pre-exercise levels. Physiologically significant elevations in some conditions may have been detectable if blood samples had been taken earlier in recovery. Plasma Na\(^+\) and plasma volume and ADH were restored at approximately the same time. Although there was an Na\(^+\)-by-time interaction for absolute urine Na\(^+\) loss, the total amount of Na\(^+\) unloaded in the urine over the 3-h period was relatively small (2–10 mM). Combined with the Na\(^+\) balance calculation, which shows a fairly large deficit in all but the HH condition, these data indicate that the majority of the Na\(^+\) ingested was retained, especially in the vascular space, and probably in the extracellular space in general. The result was removal of the stimulus to release ADH and possibly other hormones such as aldosterone and angiotensin. In the low-volume trials, therefore, there was not an excess of fluid that could be used to overcome the increase in urine production that took place during the 2nd and 3rd h of rehydration. Because there was not an Na\(^+\) effect on urine volume or ADH levels, the increase from 25 to 50 mM did not alter urine or hormonal dynamics.

*Cardiovascular responses.** Despite the differences in whole body rehydration produced by the manipulation of volume ingested, the recovery of cardiovascular function was not affected by volume or by Na\(^+\) levels. Cardiovascular drift, as indicated by increased heart rate and decreased stroke volume, has been attributed to the degree of dehydration induced during exercise (12). It is not known whether this same relationship holds true after multiple hours of recovery in the presence of varying degrees of rehydration. An additional consideration is the influence of hyperthermia on cardiovascular drift, especially when, as is usually the case, high core temperatures are present in combination with dehydration. When a rehydration model is used, the extended recovery period allows core temperature to normalize. It is possible, therefore, to study the effect of various levels of hydration on cardiovascular responses independent of elevations in temperature and the concomitant increases in subcutaneous blood responses associated with decreases in stroke volume and elevations in heart rate (12, 14).

Although limited data are available on postrehydration responses, the findings of Costill and Sparks (2), Heaps et al. (6), and Nielsen et al. (15) represent conflicting findings relative to the recovery of cardiovascular function after rehydration. The present findings are in general agreement with those of Costill and Sparks, since all aspects of cardiovascular function had returned to predehydration levels whether rehydration was complete, as in the LL and LH conditions, or whether it was at 100%, as in the HL and HH conditions. Our findings do not agree with those of Heaps et al., since they reported continued cardiovascular drift after 65% rehydration, even in the presence of complete blood volume restoration. The present findings are, however, in general agreement with those of Montain and Coyle (12), since cardiovascular function was restored with ~71–76% rehydration. These values fall between those of Heaps et al. and the 81% replacement reported to prevent extreme cardiovascular drift when fluid is consumed during exercise (12). On the basis of these percentages, a critical level of postexercise rehydration necessary to restore cardiovascular function cannot be definitively identified. The role of selective restoration of the vascular space in the recovery of cardiovascular function may also be an important factor; however, the conflicting findings from the few researchers who have addressed this issue also preclude definitive conclusions.

*Conclusion.* For whole body rehydration there was no benefit in adding Na\(^+\) beyond the 25 mM level; however, as shown previously (9, 11), greater rehydration can be achieved with large volumes. On the other hand, for fluid compartment restoration, there does appear to be an interaction between Na\(^+\) and volume, particularly relative to the intracellular space. It is apparent that plasma volume and, possibly, the entire extracellular space benefit from the presence of Na\(^+\); however, it would be of interest to examine the effects of different levels of the intracellular cation K\(^+\) on fluid compartment restoration. Finally, cardiovascular restoration was complete after 3 h of rehydration, regardless of whether fluid or Na\(^+\) replacement was complete;
thus a minimum level of fluid replacement may be
necessary to restore cardiovascular function, particu-
larly if it is accompanied by a complete plasma volume
restoration.

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