Hydration status after exercise affect resting metabolic rate and heart rate variability

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Abstract

Introduction: Heart rate variability and resting metabolic rate are commonly to assess athlete’s physiological status and energy requirements. Exercise-induced dehydration can reach up to 5% of body mass per hour. Consequently, dehydration may have a profound physiological effect on human’s homeostasis.

Objectives: To compare the effects of dehydration and rehydration after exercise on heart rate variability and resting metabolic rate in college athletes.

Methods: 14 college athletes were divided into a dehydration group (n=7) and a rehydration group (n=7), both submitted to basal (T1) heart rate variability and resting metabolic rate measurements. After basal measurements both groups were actively dehydrated (-3.4 ± 0.4% of body mass for both groups). Afterwards, dehydration group rested, while rehydration group receive a fluid intake (during a 3 h period) equivalent to 150% of body mass loss achieved during active dehydration. Four hours after active dehydration heart rate variability and resting metabolic rate were re-assessed (T2).

Results: At T2 both rehydration group (+13%) and dehydration group (+30%) achieve a significant (p<0.05) increase in resting metabolic rate, however, only dehydration group showed a significant reduction in heart rate variability. More so, the change in resting metabolic rate was significantly higher in dehydration group compared to rehydration group.

Discussion: Hydric homeostasis after exercise affects resting metabolic rate and heart rate variability, highlighting the necessity to control hydration state before resting metabolic rate and heart rate variability assessment.

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Key words: Dehydration. Rehydration. Heart rate variability. Resting metabolic rate. Physiological test. Exercise.

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Discusión: la homeostasis hídrica después del ejercicio afecta la tasa metabólica basal y la variabilidad del ritmo cardíaco, destacando la necesidad de controlar el estado de hidratación antes de las evaluaciones de la tasa metabólica basal y variabilidad del ritmo cardíaco.

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Abbreviations

HRV: Heart rate variability.
RMR: Resting Metabolic Rate.
USG: Urine specific gravity.
DG: dehydration group.
RG: rehydration group.
RR: Average absolute time between each R-wave during 10-min supine test period.
pNN50: Percentage of successive RR that varied by 50 ms or more during 10-min supine test period.
pNN20: Percentage of successive RR that varied by 20 ms or more during 10-min supine test period.
LF/HF: Ratio low frequency (LF) [ms²]/high frequency [ms²].
rMSSD: Square root of the mean squared successive differences between adjacent RR intervals (ms) over the entire recording.
T1: baseline measurements.
T2: after rehydration measurements.

Introduction

Heart rate variability (HRV) is commonly used as a non-invasive easy to apply assessment tool during athlete’s physiological status determination. HRV indicate variations in beat-to-beat intervals and autonomic nervous system activity and can be analyzed by both linear methods within the domains of time and frequency analyses, as well as nonlinear methods. HRV has been studied extensively regarding athletes’s workloads determination, sympatho-vagal balance and vagal tone in relation with sports performance and overtraining syndrome prediction. Another non-invasive easy to apply assessment tool for athlete’s physiological status determination is resting metabolic rate (RMR). RMR is required to calculate athletes’ energy requirements so as to maintain their appropriate body mass and composition to achieve peak performance. The ability to accurately determine the energy requirements of athletes is integral to developing nutritional plans and providing recommendations to enhance sports performance. Under- or overestimation of athletes’ energy requirements can result in loss of body mass, increased fat mass, diminished sports performance, increased risk of sports injuries and potentially growth failures in young athletes.

Control of contaminants variables is a key feature during athlete’s physiological assessment. In this regard, HRV and RMR must be assessed under controlled conditions. However, hydration status among athletes can vary profoundly with exercise. Exercise-induced dehydration can lead to loss of up to 5% of body mass per hour. Moreover, athletes may have inadequate hydration and re-hydration strategies. Consequently, dehydration may have a profound physiological effect on human’s homeostasis, negatively affecting proteinuria, leukocyturia and erythrocyturia, baroreflex control, blood pressure and Angiotensin II levels. Therefore, it is possible that hydration status affect HRV and RMR. However the effect of active dehydration on HRV and RMR are poorly investigated. Thus, the purpose of this study is to investigate the effects of exercise-induced dehydration on HRV and RMR.

Methods

Participants

Initially 30 male college athletes were recruited to participate in this study, but because exclusion criteria (see below) only 14 were deemed suitable. Athletes fulfilled the following inclusion criteria: absence of renal or arterial diseases; no ingestion of coffee, alcohol, diuretics or any beta- or alpha blocker and no participation in vigorous physical activity of any type at least 3 days before measurements; and to have a basal urine specific gravity (Usg) between 1000 a 1009 gm/ml. All participants were carefully informed about the experimental procedures and about the possible risks and benefits associated with participation in the study and signed an informed consent document before any of the tests were performed. The study was conducted in accordance with the Declaration of Helsinki and was approved by the ethics committee of the responsible department.

Experimental protocol

All participants were asked to consume 1.5 liters of water per day during 48 h prior to measurements. Participants were cited to the laboratory at 10:00 am and randomly divided in two groups, a dehydration group (DG, n = 7) and a rehydration group (RG, n = 7) (descriptive characteristics in table I). The sample size was computed according to the changes observed in several HRV measures in a group of physically active males submitted to a dehydration-rehydration protocol similar as that used in the present study. A statistical power analysis revealed that a total of 5 participants per group would yield a power of 80% and α = 0.05. Both groups were submitted to the following baseline measurements (T1):

Table I

<table>
<thead>
<tr>
<th>Subjects’ basal characteristics</th>
<th>Rehydration group (n = 7)</th>
<th>Dehydration group (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>21.6 ± 1.2</td>
<td>20.7 ± 2.0</td>
</tr>
<tr>
<td>Height, cm</td>
<td>172 ± 5.1</td>
<td>175 ± 1.6</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>24.7 ± 0.6</td>
<td>24.6 ± 1.2</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation.
Body mass and height

A high precision (i.e. 0.1 kg) mechanical scale with an incorporated stadiometer (SECA model M20812, Germany) was used for anthropometric measurements.

Urine specific gravity (U\text{SG}).

A portable refractometer (Robinar model Spx, USA) previously validated\(^6\) was used to assess U\text{SG} and was classified according to previously described values\(^7\).

Heart rate variability (HRV)

A Polar S810 monitoring system (Polar Electro, Finland) previously validated\(^8\) was used for HRV assessment, including: a) average absolute time between each R-wave during 10-min supine test period (RR), b) percentage of successive RR that varied by 50 ms or more during 10-min supine test period (pNN50), c) percentage of successive RR that varied by 20 ms or more during 10-min supine test period (pNN20), d) ratio low frequency (LF) [ms\(^2\)]/high frequency [ms\(^2\)] (LF/HF) and e) square root of the mean squared successive differences between adjacent RR intervals (ms) over the entire recording (rMSSD). HRV was measured at all times in a quiet room during a 10 min period. Data was analyzed in Kubios Software HRV 2.0 (Biosignal Analysis and Medical Imaging Group, Kuopio, Finland).

Resting metabolic rate (RMR)

RMR was assessed after lying in a resting position during 16 min in a thermo-neutral environment\(^9\) using indirect calorimetry with a validated\(^2\) and calibrated standard metabolic cart (Cosmed FitMate Pro, Rome, Italy).

After T1 both groups were submitted to an active dehydration protocol (carried out at 32°C room temperature), consisting in 1 min of exercise and 1 min of rest, interspersing 5 different exercises (i.e. squats, push-ups, box jumps, 20-m sprints and slalom 20-m runs) for a total period of 45 min. According to a pilot study, the active dehydration protocol was aimed at inducing a dehydration equivalent to 3.5% of body mass, which was checked by body mass measurements every 15 min. After active dehydration protocol, DG rested quietly during 4 h, while during the same time period participants from the RG receive a fluid intake (in a period of 3 h) equivalent to 150% of their body mass loss, as suggested previously\(^10\). Afterwards both groups were re-assessed (T2) for body mass, U\text{SG}, HRV and RMR, as described above. Both T1 and T2 measurements were overseen by the same investigator, who was blinded to the hydration condition of participants.

Statistical analysis

All values are reported as mean ± standard deviation. (SD). Normality and homoscedasticity assumptions for all data before and after intervention were checked, respectively with the Shapiro–Wilk and Levene’s tests. As data was non-parametrical, the effect of dehydration was analyzed using Mann Whitney U Test and Wilcoxon paired t-test. Alpha level was set at p<0.05. All statistical calculations were performed using the GraphPad Prism statistical package version 6.00 (GraphPad Software, La Jolla California, USA).

Results

At T1 no basal differences were observed between groups for any of the dependent variables (table II).

<table>
<thead>
<tr>
<th>Table II</th>
<th>Effect of dehydration and rehydration on body weight, resting metabolic rate and heart rate variability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>Rehydration group (n = 7)</strong></td>
</tr>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>72.7 ± 2.5</td>
</tr>
<tr>
<td>Urine specific gravity, gm/ml (^1)</td>
<td>1.005 ± 0.0008</td>
</tr>
<tr>
<td>Resting metabolic rate, kcal/d (^1)</td>
<td>2211 ± 64.0</td>
</tr>
<tr>
<td>RR, ms</td>
<td>910 ± 126</td>
</tr>
<tr>
<td>pNN50, %</td>
<td>26.0 ± 15.5</td>
</tr>
<tr>
<td>pNN20, %</td>
<td>61.8 ± 11.1</td>
</tr>
<tr>
<td>rMSSD, ms</td>
<td>47.6 ± 14.9</td>
</tr>
<tr>
<td>LF/HF ratio, ms(^2)</td>
<td>3.0 ± 1.7</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation. * ** denotes significant difference with the corresponding baseline value (p<0.05 and p<0.01, respectively). † denote that baseline to T2 change is significantly higher compared with that of the rehydration group (p<0.05). RR: average absolute time between each R-wave during 10-min supine test period; pNN50 y pNN20: percentage of successive RR that varied by 50 ms or more and by 20 ms or more, during 10-min supine test period; LF/HF ratio: low frequency (LF) / high frequency (HF) ratio; rMSSD: square root of the mean squared successive differences between adjacent RR intervals (ms) over an entire recording; T2: after active dehydration.

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At T2 the only significant (p<0.05) change observed in RG was an increase in RMR (+13%). On the other side, DG showed a significant (p<0.05) change in body mass (-4.1%), \( U_{ag} \) (+1.8%), RMR (+30%), RR (-21%), pNN50 (+44%), pNN20 (-27%), rMSSD (-35%) and LF/HF ratio (+41%). More so, the baseline to T2 change in body mass, \( U_{ag} \), RR, pNN50, pNN20, rMSSD, LF/HF ratio (table II) and RMR (Fig. 1) in DG is significantly higher compared with that of the RG.

Discussion

One of the main outcomes of this study is that hydration status affects HRV, specifically our data shows that dehydration (i.e. DG) reduce HRV (i.e. reduced parasympathetic activity, increased sympathetic activity or both). Our results are similar to those previously reported in participants submitted to exercise under heat stress\(^\text{36}\), where dehydration, although induced a lower LF/HF ratio, caused an overall reduced vagal tone. Also in line with our results, it had been shown that HRV assessed trough RR and pNN50 were positively correlated with hydration status assessed by total body water analysis and negatively correlated with \( U_{ag} \), independent from fitness level or sex. In the same line, in other species (e.g. rats) dehydration also induced a reduction in parasympathetic activity\(^\text{25}\), suggesting that the effect of hydration status on HRV may be species-independent. It is possible that the effect of dehydration on HRV had been potentiated by hyperthermia, as the combination of these two physiological conditions may induce both an increased sympathetic and a decreased parasympathetic activity.\(^\text{25}\) In fact, dehydration increases heat storage rate and reduces the ability to dissipate body heat, which can be related to both hyperosmolality and hypovolemia, resulting in an increased cardiovascular strain\(^\text{36}\). Although all participants in our study were apparently healthy and free of renal or arterial diseases, in other populations it has been stated that a reduced parasympathetic activity may be linked to an increased risk of arrhythmias and development of coronary heart disease\(^\text{27, 28}\), reinforcing the importance of adequate hydration strategies. Our results indicate that both RG and DG increase RMR after active dehydration. Previously, it has been observed an increased RMR after exercise during 24 h or more\(^\text{30, 31}\), which can be related to several factors, like intensity, duration and mode of exercise\(^\text{31}\), training state\(^\text{32}\) and maybe the gender of the participant\(^\text{33}\). From a physiological point of view the increased RMR after exercise can be related to replenishment of oxygen depot (i.e. hemoglobin, myoglobin) and high-energy phosphates (i.e. ATP, PC) resynthesis, lactate removal-oxidation, increased Cori cycle activity, increased ventilation, higher heart rate, increased blood circulation, thermogenic effect of increased core temperature and catecholamines, increased triacylglycerol/free fatty acid re-esterification, changes from carbohydrate to lipids preference for energy metabolism and exercise-induced muscle damage\(^\text{34, 35}\). Although it is not surprising to observe an increased RMR after 4 h of exercise in RG and DG, we found a significantly higher RMR change in DG compared to RG (Figure 1). Therefore, another main and novel outcome of this study was that hydration status affects RMR, specifically our data shows that dehydration (i.e. DG) increase RMR. Previously, dehydration during submaximal\(^\text{35}\), \(^\text{36}\) high-intensity\(^\text{36}\) even maximal\(^\text{36}\) exercises did not affect oxygen consumption. More so, previous reports show that passively-induced dehydration did not affect post intervention oxygen consumption\(^\text{40}\). The difference between our results and previous studies may be related with the degree of dehydration achieved (i.e. 3.5% in our study vs. 2% in Coles et al., 2006).

In conclusion, hidric homeostasis affects RMR and HRV, highlighting the necessity to control hydration state before RMR and HRV assessment.

The authors have no conflict of interest.

Reference