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original article

Effect of water intake on postexercise cardiovascular recovery

Efeito da ingestão hídrica sobre a recuperação cardiovascular pós-exercício

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Abstract – Physical exercise elicits an increase in heart rate (HR), blood pressure (BP) and, consequently, in the rate-pressure product (RPP). Recovery of HR immediately after exercise indicates cardiovascular health. Blood pressure also decreases after exercise, occasionally reaching values lower than pre-exercise levels (postexercise hypotension). Studies have shown a positive effect of water intake on HR recovery after exercise. However, little is known about the effect of water intake on postexercise BP and RPP responses. The objective of this study was to evaluate the effects of water intake on postexercise cardiac work assessed by HR, BP and RPP. Fourteen healthy volunteers (22 ± 1.4 years) participated in the study. The experimental session consisted of HR, systolic (SBP) and diastolic BP (DBP) recording at rest, followed by submaximal exercise on a cycle ergometer. Next, the subjects consumed water and the cardiovascular variables were recorded during recovery. In addition, a control session without postexercise water intake was performed. The RPP was calculated from the product of HR and SBP. Water intake prevented a postexercise hypotensive effect on DBP, but accelerated postexercise HR and RPP reduction during recovery when compared to the control session. It was concluded that water intake is an effective strategy to reduce postexercise cardiac work.

Key words: Exercise; Heart rate; Postexercise hypotension; Water intake

Resumo – *O exercício físico promove a elevação da frequência cardíaca (FC), pressão arterial (PA) e, por consequência, do duplo produto (DP). Imediatamente após o término do exercício, há a recuperação da FC; resposta que indica boa saúde cardiovascular. A PA também apresenta queda pós-exercício, atingindo, eventualmente, valores abaixo do repouso (hipotensão pós-exercício; HPE). Estudos têm demonstrado efeito positivo da ingestão hídrica (IH) sobre a recuperação da FC pós-exercício. Pouco se sabe a respeito do efeito dessa estratégia sobre o comportamento da PA e do DP nesse período. O objetivo do estudo foi investigar o efeito da IH sobre o trabalho cardiovascular pós-exercício, por meio da avaliação da FC, PA e DP. Quatorze voluntários saudáveis (22 ± 1,4 anos) participaram desse estudo. A sessão experimental constou do registro da FC e PA sistólica (PAS) e diastólica (PAD) de repouso, seguido de exercício físico submáximo em cicloergômetro. Posteriormente, realizou-se a IH e registro das variáveis cardiovasculares na recuperação. Adicionalmente, realizou-se uma sessão controle, excluindo-se a IH pós-exercício. O DP foi calculado a partir do produto da FC pela PAS. A IH impediu a ocorrência de HPE na PAD, porém acelerou a redução da FC e do DP, no período da recuperação pós-exercício, quando comparada à sessão controle. Pode-se concluir que a IH é uma estratégia eficiente na redução do trabalho cardiovascular pós-exercício.*

Palavras-chave: *Exercício; Frequência cardíaca; Hipotensão pós-exercício; Ingestão de água.*

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INTRODUCTION

Intense physical activity elicits an increase in heart rate (HR) and blood pressure (BP) and, consequently, in myocardial oxygen consumption. A decrease in HR is seen immediately after exercise, reaching baseline values within the first minutes of recovery[1]. With respect to BP recovery, a progressive reduction in this variable occurs until baseline values, occasionally reaching values lower than pre-exercise levels, a phenomenon called postexercise hypotension (PEH)[2].

HR recovery is the rate at which the HR returns to the resting condition after exercise and has been shown to be a predictor of mortality[1]. With respect to postexercise BP, the occurrence of PEH is a desirable response since it indicates a reduction in cardiac work[2]. Studies have shown that PEH can persist for 24 hours, a fact demonstrating its clinical relevance[3,4].

Strategies that improve postexercise cardiovascular recovery should be investigated. Water intake has been proposed by our group as an effective strategy to improve HR recovery after moderate intensity exercise[5,6]. However, studies have shown that water intake promotes an increase in BP through sympathetic activation[7], a response that may inhibit PEH. In this respect, Endo et al.[8] investigated the effects of water intake on postexercise BP responses and observed the absence of PEH. However, the subjects were submitted to exercise of moderate intensity. In view of the influence of exercise intensity on the magnitude of PEH[9], it is possible that the suppressive effect of water intake on PEH does not occur after intense exercise.

Furthermore, the effect of water intake on the postexercise rate-pressure product (RPP) remains unknown. This variable – systolic BP (SBP) x HR – estimates myocardial work[10] and has an important prognostic value, with higher RPP values being associated with an increased risk of cardiovascular events[11]. Considering the positive effect of water intake on HR recovery and its possible suppressive effect on PEH, RPP evaluate the final cardiovascular effects promoted by the exercise with water intake. The objective of the present study was to evaluate the effect of water intake on HR, BP and RPP responses during recovery after a session of intense exercise.

METHODOLOGICAL PROCEDURES

Subjects

Fourteen healthy, non-athlete male university students (22 ± 1.4 years; 24.1 ± 1.6 kg/m^2) participated in the study. Exclusion criteria were the presence of cardiovascular diseases and the use of any medication. The participants were asked not to consume alcoholic or caffeinated beverages and not to perform any exercise 24 h before the beginning of the tests. In addition, the volunteers were instructed not drink water or to eat 2 h prior to the tests. All volunteers agreed to participate in the study by signing the free informed consent form. The study was approved by the Ethics Committee of Universidade Federal de Juiz de Fora (Permit No. 198/2011) and was con-

ducted according to guidelines for research involving humans (Resolution 196 of October 10, 1996 of the National Health Council).

Data collection protocol

The experimental sessions were conducted in the Laboratory of Motor Evaluation at an ambient temperature of 18 to 24ºC. The sessions were held randomly on 3 non-consecutive days in the afternoon (14:00 to 16:00 h). On the first visit, the subjects were submitted to anthropometric assessment and evaluation of maximal aerobic fitness. Body weight was measured to the nearest 0.1 kg with a digital scale (ID-1500, Filizola®, Brazil). Height was measured to the nearest 0.1 cm using a stadiometer (110, Welmy®, Brazil). The body mass index (BMI) was calculated by dividing body weight by the square of the height. For assessment of maximal aerobic fitness, the subjects underwent an incremental test on an electromagnetically braked cycle ergometer (Ergo 167 cycle, Ergofit®, Germany) at an initial power of 50 W and increments of 25 W/min. Expired gases were analyzed with a metabolic analyzer (VO2000, MedGraphics®, USA), which was calibrated before each test. The criteria used for the determination of maximum effort were a respiratory quotient > 1.10 and HR higher than 90% of the maximum predicted for age[12]. Maximal oxygen uptake (VO_2max) was determined as the highest mean obtained over a period of 20 s during the test. The respiratory compensation point (RCP) was defined based on the loss of linearity in the VE/VCO_2 ratio[13].

Visits 2 and 3 were randomized and consisted of water intake and control sessions. First, the volunteers collected urine samples and self-assessed urine color[14], followed by the measurement of pre-exercise body weight. Next, the subjects rested in the supine position for 10 min. During the resting period, BP and HR were measured in the 5th and 10th minute. The subjects then exercised on a cycle ergometer for 30 min at a load corresponding to 80% of RCP. During exercise, HR and perceived exertion (Borg CR10 Scale) were recorded at intervals of 5 min. Immediately after the end of exercise, the subjects consumed 7.5 ml water per kg body weight (mean ingested volume of ~500 ml) over a period of 30 s at room temperature[15,16]. Next, the recovery period was started during which the subjects remained in the supine position for 60 min and BP and HR were recorded at intervals of 15 min (15, 30, 45, and 60 min). After the recovery period, postexercise body weight was measured. The control session consisted of the same steps as the water intake session, except for postexercise water intake.

Cardiovascular variables

BP was measured by the auscultatory method, with the subject in the supine position, using a mercury column sphygmomanometer (Takaoka®, Brazil). Korotkoff phase I and IV sounds were defined for the identification of SBP and diastolic BP (DBP), respectively. HR was measured simultaneously with BP throughout the experimental period using a HR monitor (RS800cx, Polar Electro Oy®, Finland). The RPP at rest and during recovery was calculated

using the following formula: RPP = SBP x HR.

Hydration status

The pre-exercise hydration status was determined by urine collection and self-assessment of urine color using an 8-point urine color chart[14]. Comparing the urine color with the points of the chart, pre-exercise hydration status was classified as euhydration (1 to 3 points), moderate dehydration (4 to 6 points), and severe dehydration (> 6 points).

The absolute (kg) and relative (%) variation in body weight during the session was used to evaluate postexercise hydration status[17]. On the basis of percent weight loss, postexercise hydration status was classified as euhydration (+1 to -1%), mild dehydration (-1 to -3%), moderate dehydration (-3 to -5%), and severe dehydration (> -5%)[18].

Statistical analysis

The results are reported as the mean ± standard deviation. Normality, homogeneity and sphericity of the data were evaluated by the Shapiro-Wilk test, Levene's test and Mauchly's test, respectively. The Student *t*-test for dependent measures was used to compare cardiovascular variables obtained at rest and during exercise between the water intake and control sessions, as well as pre- and postexercise hydration status. Two-way analysis of variance (ANOVA) for repeated measures was used for analysis of the recovery variables. The principal factors were the intervention (water intake and control) and time points (15, 30, 45 and 60 min pre- and postexercise). A p value < 0.05 was accepted as significant and Duncan's test was used to identify existing differences. High statistical power (0.85-0.95) was observed for all tests (parameters used: $n = 14$, $\alpha = 0.05$, and effect size = 0.4-0.5). All analyses were performed using the Statistica software (v. 8.0, StatSoft, Inc., USA).

RESULTS

Table 1 shows the anthropometric and cardiorespiratory variables of the subjects studied.

Table 1. Characterization of the sample.

Variable	Mean ± standard deviation
Age (years)	22 ± 1.4
Height (m)	1.74 ± 0.1
Body weight (kg) BMI (kg/m^2)	74.0 ± 6.8 24.1 ± 1.6
VO_{2max} (ml.kg.min^{-1})	46.7 ± 7.8
W_{max} (watts) W_{RCP} (watts)	253.5 ± 53.8 144.6 ± 18.7
W_{EXE} (watts)	115.7 ± 14.6

BMI: body mass index; VO2max: maximal oxygen uptake; Wmax: maximum load reached in the exercise test; WPCR: load obtained at the respiratory compensation point; WEXE: exercise load corresponding to 80% of WRCP.

Pre-exercise cardiovascular variables

Pre-exercise SBP, DBP, HR and RPP obtained during the water intake and control sessions are shown in Table 2. There was no significant difference in any of the variables between the two sessions.

Table 2. Pre-exercise cardiovascular variables.

	Control session	Water intake session	p
SBP (mmHg)	112 ± 2.5	116 ± 2.6	0.403
DBP (mmHg)	71 ± 1.7	69 ± 2.1	0.481
HR (bpm)	65 ± 8.4	69 ± 17.2	0.433
RPP (mmHg . bpm)	7372 ± 574	8037 ± 349	0.433

SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; RPP: rate-pressure product.

Exercise session

The mean HR values were 160.8 ± 9.4 and 161.7 ± 9.8 bpm in the water intake and control sessions, respectively. The mean ratings of perceived exertion on the Borg CR10 scale were 5 ± 1 and 5 ± 1.3 (heavy) in the water intake and control sessions, respectively. Both HR and rating of perceived exertion were similar in the two sessions ($p > 0.05$), conferring the same relative intensity in the two conditions studied.

Hydration status

Table 3 shows the variables related to hydration status. Pre-exercise hydration status was similar in the two sessions. With respect to postexercise hydration status, both sessions resulted in a significant reduction of body weight. However, weight loss was higher after the control session compared to the water intake session.

Table 3. Indicators of pre- and postexercise hydration status

	Control session	Water intake session	p
UCS	3 ± 0.8	3 ± 0.7	0.643
Initial weight (kg)	74.7 ± 7.1	74.6 ± 7.3	0.986
Final weight (kg)	74.1 ± 7.4†	74.5 ± 7.3†	0.910
Weight loss (kg)	0.4 ± 0.1	0.09 ± 0.09*	< 0.001
%WL	0.6 ± 0.2	0.1 ± 0.1*	< 0.001

UCS: urinary color scale; %WL: percent weight loss during the session. * Significant difference compared to the control session; † significant difference compared to initial weight ($p < 0.05$).

Effect of water intake on postexercise cardiovascular variables

Blood pressure. Postexercise SBP and DBP were similar in the two sessions (Figure 1A and 1B, respectively). When compared to resting BP values, no differences in postexercise SBP or DBP were observed at any time of recovery after the water intake session. On the other hand, DBP was significantly reduced in the 45th minute of recovery after the control session when compared to resting values (i.e., PEH). No difference was observed for SBP.

Heart rate. There was no significant difference in HR values between sessions (Figure 1C). When compared to resting values, an increase in HR

was only observed 15 min after the water intake session, with no differences thereafter. In contrast, an elevated HR was observed throughout the recovery period after the control session.

Rate-pressure product. No differences in the RPP were observed between the water intake and control sessions throughout the recovery period (Figure 1D). The RPP was significantly higher only in the 15th minute of recovery after the water intake session when compared to resting RPP. In contrast, after the control session, significantly higher values were observed throughout the recovery period.

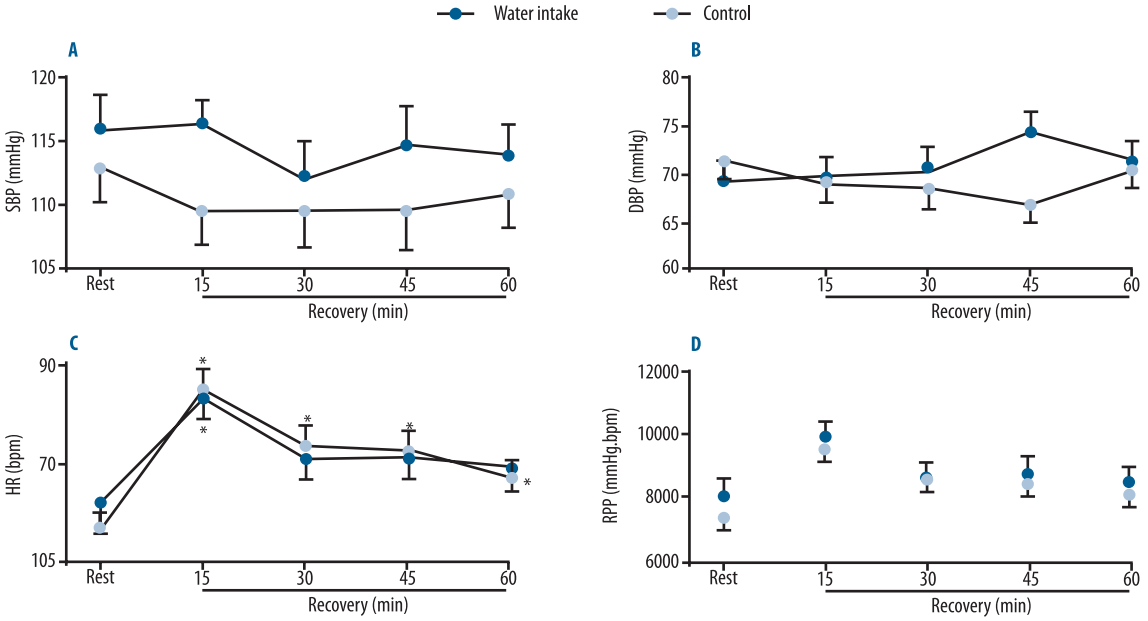

Figure 1. Systolic (SBP) (A) and diastolic blood pressure (DBP) (B), heart rate (HR) (C) and rate-pressure product (RPP) (D) at rest and at 15, 30, 45 and 60 min of recovery after exercise. Dark blue circle: water intake session; Light Blue circle: control session. * Significant difference compared to pre-exercise values.

DISCUSSION

The present study demonstrated an effect of water intake on postexercise cardiovascular recovery. The main findings were: i) water intake prevented the occurrence of a postexercise hypotensive effect on DBP, but ii) accelerated the reduction in HR during the postexercise recovery period, and iii) water intake accelerated the reduction in BP during the recovery period when compared to the control session. The present results therefore confirm the positive effect of water intake on the response of postexercise cardiovascular variables.

An increase in the pressure response to water intake at rest has been reported in the literature[19-21]. Studies suggest that gastric distension resulting from water consumption stimulates mechanoreceptors present in the stomach, causing sympathetic activation, an increase in total peripheral resistance, and consequent elevation of BP[19,22,23]. In addition, factors related to the hypo-osmotic properties of water seem to influence the BP response to water intake[24], promoting an acute activation of osmoreceptors (Trvp4)

that are sensitive to alterations in osmolarity in the portal circulation and liver and increasing sympathetic nerve activity[24,25].

On the other hand, there is a lack of studies investigating the influence of water intake on the postexercise pressure response. In a literature review, we found only one study conducted by Endo et al.[8] that evaluated the effect of water intake on BP during the recovery period. The authors observed that *ad libitum* water intake during exercise prevented the reduction in postexercise SBP and DBP. However, in that study the subjects performed aerobic exercise of moderate intensity. Considering the influence of exercise intensity on the magnitude of PEH[9,26], one of the hypotheses of the present study was that the suppressive effect of water intake on PEH may not occur after intense exercise. This hypothesis was not confirmed, since water intake was found to prevent the occurrence of a postexercise hypotensive effect on DBP only at 45 min of recovery. Although SBP did not differ significantly between sessions, the mean SBP values were consistently elevated in the water intake session.

Although water intake prevented a postexercise hypotensive effect on DBP, this strategy accelerated the reduction in postexercise HR. The same has been demonstrated by our group after moderate exercise[6]. The recovery of HR is promoted by vagal reactivation and removal of cardiac sympathetic activity after exercise[27]. In fact, other studies have demonstrated a positive effect of water intake on postexercise vagal reactivation. Vianna et al.[16] observed higher postexercise vagal cardiac tonus after ingestion of 500 ml water when compared to the control condition. Also within this context, De Oliveira et al.[5] found that water intake resulted in higher HR variability values during the recovery period.

The mechanisms whereby water intake promotes an increase in postexercise vagal activity remain unclear. However, it can be suggested that this response is a consequence of the BP increase through activation of the baroreflex system[15] or of the increase in systolic volume through activation of cardiopulmonary receptors[28]. However, regardless of the mechanisms underlying the finding of the present study, the acceleration of HR recovery is a clinically important response in view of its proven cardioprotective effect[2].

Although the observation of the individual effect of water intake on postexercise HR and BP permits to study the cardiovascular responses elicited by this strategy, the combined investigation of these variables leads to a better understanding of the final cardiovascular impact of water intake. The product of HR and SBP, the RPP, has shown to be highly correlated with myocardial oxygen consumption[10] and is therefore considered an important measure of myocardial work. The present results indicate that water intake accelerates the reduction in postexercise RPP, suggesting lower cardiac work during this period. Therefore, despite the contrasting results regarding the effect of water intake on HR and BP, it seems reasonable to conclude that the final cardiovascular impact of exercise is reduced after water intake. This fact encourages the use of this approach not only as a hydration strategy, but also for cardiovascular protection.

Limitations

Some limitations of the study should be mentioned. Most investigations evaluating the influence of water intake on cardiovascular variables have administered a volume of 500 ml. In the present study designed to establish individual values, an arbitrary volume (7.5 ml/kg body weight) was used, which corresponds to 500 ml for an average 70-kg adult.

The evaluation of hydration status based on a urine color scale is certainly not as precise as other methods (e.g., urine dilution, urine volume, and plasma osmolality)[29]. However, studies have reported high agreement between this measure and other reliable methods used for the evaluation of hydration status (urine gravity and osmolarity and weight loss)[14,30]. Furthermore, although the method used did not permit to identify the degree of hydration with precision, the urine color values were similar in the sessions, thus guaranteeing that the subjects were at the same hydration level at the beginning of the experiment.

Finally, the results cannot be extrapolated to risk groups. For this purpose, it would be necessary to repeat the present protocol in patients with cardiovascular risk factors or cardiovascular diseases.

CONCLUSION

Water intake prevents PEH, but interferes positively with HR recovery and accelerates the reduction in postexercise RPP. These findings suggest that postexercise water intake is a useful resource to accelerate the return of HR to baseline values and to reduce myocardial work, serving as a cardioprotective strategy during postexercise recovery. Further studies are needed to elucidate the mechanisms underlying the responses observed here, as well as similar studies involving risk groups.

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