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Dehydration does not influence cardiovascular reactivity to behavioural stress in young healthy humans

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Summary

Enhanced hydration increases the human cardiovascular reactivity to mental stress. If reduced water intake has the opposite effect, this would suggest controlling for water deprivation when studying such responses. Blood pressure, heart rate and parasympathetically dominated beat-to-beat heart rate fluctuations were assessed during resting baseline and mental stress. Two challenging cognitive-motor tasks, a 5-Choice Reaction Time Task (CRTT) and a Paced Auditory Serial Addition Task (PASAT), served as mental stress tests. Eight female and eight male volunteers were examined twice, after 24 h of water deprivation and after normal water intake (counterbalanced order, 7-day interval). Water deprivation resulted in moderate dehydration with a mean 2·6% decrease of total body weight. Dehydration did neither affect baseline blood pressure, heart rate, nor blood pressure reactivity to mental stress. However, dehydration slightly (−1·2 bpm) diminished heart rate reactivity to the PASAT ($P = 0{\cdot}03$) and increased beat-to-beat heart rate fluctuations in response to the CRTT ($P = 0{\cdot}05$). Dehydration intensified CRTT- and PASAT-induced reductions of beat-to-beat heart rate fluctuations in females (gender × dehydration interactions: $P = 0{\cdot}04$–$0{\cdot}05$). Moderate dehydration induced by water restriction has no effect on blood pressure reactivity to mental stress. The effects on heart rate reactivity are small. However, stress-induced parasympathetic withdrawal may be fortified during dehydration in females, which suggests controlling for water intake when studying such responses.

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*Although the program GPOWER uses the effect size f^2 we present the more common effect size ω^2. The two effect sizes can be transformed into one another by the formula: $\omega^2 = f^2/1 + f^2$.

Introduction

Increased cardiovascular reactivity to mental stress predicts future hypertension (Treiber *et al.*, 2001; Tuomisto *et al.*, 2005) and other cardiovascular events (Krantz *et al.*, 1999). Cardiovascular reactivity is relatively easy to assess. However, several important covariates should be considered. Individual characteristics which influence cardiovascular reactivity to mental stress are gender (Stone *et al.*, 1990; Stroud *et al.*, 2002), age (Rose *et al.*, 2004), and ethnic background (Shen *et al.*, 2004). Various stable psychological and behavioural traits like hostility (Fichera & Andreassi, 2000), type A behaviour (Melamed *et al.*, 1993) and locus of control (Peters *et al.*, 2003) may influence cardiovascular reactivity to stress. Furthermore, cardiovascular stress reactivity differs considerably within subjects, as is indicated by moderate test–retest reproducibility (Swain & Suls, 1996; Gerin *et al.*, 1998). Changing covariates may explain this. Of potential importance are physical exercise status (Blumenthal *et al.*, 1990; Hendrix & Hughes, 1997), social support (Kamarck *et al.*, 1990; Christenfeld & Gerin, 2000), smoking (Girdler *et al.*, 1997), eating (Uijtdehaage *et al.*, 1994), menstrual cycle phase (Girdler & Light, 1994; Sato & Miyake, 2004), time of day (Adan & Sanchez-Turet, 1996; Nebel *et al.*, 1996), sleep restriction (Meerlo *et al.*, 2002), oral contraceptive use (Emmons & Weidner, 1988; West *et al.*, 2001) and the use of other medications (Ruddel *et al.*, 1988).

Another behavioural factor which varies considerably between individuals and seems to have an impact on cardiovascular stress reactivity is the hydration status. Enhanced hydration may result from increased amount of water consumed. It has recently been shown that mildly enhanced hydration leads to changes in blood pressure during psychological stress tests (Rochette & Patterson, 2005). A possible explanation of this finding is given by a recent study which showed that enhanced hydration may result in higher circulatory blood and plasma volume (Veldhuijzen van Zanten *et al.*, 2005), which in turn increases cardiac preload, stroke volume and subsequently blood pressure reactivity.

Less is known about the influence of mild dehydration. Mild dehydration may result from exercise, heat, profound sweating, diuretic drugs or reduced drinking and is therefore very common in everyday life. The amount of water intake varies within- and between-subjects. Importantly, thirst and water drinking are associated with the experience of stress (Greenleaf, 1992). Therefore, dehydration induced by reduced water drinking may be an important factor influencing the cardiovascular stress response, and thus should be taken into consideration when conducting stress reactivity studies. Based on the finding that blood pressure reactivity increases with enhanced water intake (Rochette & Patterson, 2005), it could be speculated that reduced water intake will decrease the cardiovascular stress reactivity. Indeed, dehydration may decrease blood volume and blood pressure. Subsequent baroreflex unloading, and reflex activation of the sympathetic nervous system (Haberthur *et al.*, 2003), as well as dehydration-induced vasopressin released from the pituitary gland, may lead to peripheral vasoconstriction and successive blood pressure elevation. These mechanisms may be sufficient to compensate for the blood volume reduction during unchallenging baseline conditions, but insufficient to guarantee full blood pressure responsiveness during mental stress.

Up to now, it is unknown whether mild dehydration induced by reduced drinking has any impact on the cardiovascular reactions to psychological stress. Based on earlier studies (Rochette & Patterson, 2005), we hypothesize a diminishing effect of dehydration on the cardiovascular stress reactivity. If this is the case, future reactivity studies should consider water deprivation as a potential covariate which may affect cardiovascular responses to mental stress.

This study focuses on cardiovascular reactivity data, such as heart rate and blood pressure reactivity. In addition, a time domain-based index heart rate variability (HRV), the root mean square of successive differences (RMSSD) of interbeat intervals (IBI) (Routledge *et al.*, 2002) was used because it represents a simple and readily understood time domain measures of parasympathetic cardiac control (Hayano *et al.*, 1991) that is rather resistant to the effects of changing breathing patterns (Penttila *et al.*, 2001).

Materials and methods

Participants

Eight female students with regular menstruation and without contraceptive intake for the last 3 months [age mean: 25 years; age range: 21–34; body mass index (BMI) mean: 19·2 ± 1·3 kg m^{-2}, BMI range: 18–21] and eight men (age mean: 28 years; age range: 20–34; BMI mean: 22·6 ± 1·7 kg m^{-2}; BMI range: 20–25) agreed to participate in the investigation. Participants were recruited by announcements at the medical school of the University of Basel. All subjects were healthy non-smokers. They were on no medications and have shown no evidence for drug abuse. The study was approved by the local Ethic Committee, and informed consent was collected prior to the beginning of the investigation from each participant.

Procedure

All participants were tested twice, under the dehydration and the normal water intake condition. The order of the conditions was counterbalanced. Between both conditions was an interval of 6–8 days. Women were tested during the follicular phase to ensure that their osmotic pressure was same as the osmotic pressure of men (Stachenfeld *et al.*, 2001). Each testing involved 2 days. The study began at 8:00 AM on day 1 and took all in all 28 h (including the test session period from 8:00 to 12:00 on day 2). To ensure compliance with the water deprivation protocol subjects stayed in hospital for the whole time of the investigation. During dehydration subjects had no access to fluids and were supposed to do their ordinary activities (e.g. school readings) in the hospital. They were only allowed to ingest food containing <75% water by weight. Participants in the control condition were allowed to freely consume beverages, with the exception of coffee and alcoholic drinks.

Cardiovascular beat-to-beat data were assessed at prestress baseline (5 min) and during mental stress. A standard lead II electrocardiogram and continuous blood pressure (Finapres system, Ohmeda, Englewood, CO, USA), were recorded. Analogue to digital conversion was performed at 1000 Hz for offline analysis of IBI and beat-to-beat blood pressure data. Blood pressure and heart rate were averaged per person and period. A customized computer program was used to calculate the RMSSD of IBI as an index of parasympathetically dominated beat-to-beat heart rate fluctuations (Buchholz *et al.*, 2003; Nava *et al.*, 2004). Prior to calculating RMSSD the beat-to-beat values of IBI were edited for outliers due to artefacts or ectopic myocardial activity. Finapres is known to overestimate blood pressure. Thus, intermittent oscillometric cuff blood pressure (by Dinamap, Criticon, FL, USA) was assessed during prestress baseline, and compared with Finapres blood pressure data. Subjects were seated in a semi-recumbent position. During the 5 min assessment of cardiovascular baseline activity subjects were instructed to close their eyes, relax, not to move and not to speak. Furthermore, participants were asked to keep their left hand still during PASAT and CRTT; the left hand was placed at heart level.

Subjects participated in preliminary test sessions 1 week before the investigation to become familiar with the stress tests and the test environment. During the control and the dehydration condition both stress tests were presented twice. To minimize unspecific adaptation effects, only data of the second stress testing session were considered in the analysis (see also Szinnai *et al.*, 2005).

PASAT

The Paced Auditory Serial Addition Task (PASAT) can be used either as a test of divided attention and working memory, or as

an instrument to induce stress (Mathias *et al.*, 2004; Philippsen *et al.*, 2007). Participants were presented one-digit numbers via headphones. They were asked to add each number to the previous one and to tell the sum aloud. Numbers were administered in intervals of 2·5 s. The total task took 180 s. Detailed descriptions of the test procedure are published elsewhere (Schächinger *et al.*, 2003; Szinnai *et al.*, 2005).

CRTT

The 5-Choice Reaction Time Task (CRTT) can be used to test visual attention and cognitive-motor speed, as well as to induce stress (Schächinger *et al.*, 2000; Philippsen *et al.*, 2007). Subjects were requested to respond to coloured lights (red, blue, green, yellow and white), which were presented in random order, as accurately and fast as possible by pressing a button of the same colour. The inter-stimulus interval was adapted to the subject's performance, leading to a constant false response rate of 50%. The total test time was 5 min. Detailed descriptions of the test are published elsewhere (Schächinger *et al.*, 1999, 2003; Szinnai *et al.*, 2005).

Thirst ratings

Participants were asked to indicate how thirsty they feel by placing a mark on a 100 mm line with the extremes 'Not at all thirsty' and 'Very thirsty'. Changes compared with the predeprivation state were calculated for each individual on day 2 before and at the end of the cognitive function tests.

Ratings of tiredness, effort and concentration

A 5-point Likert scale ranging from 'true' to 'not true' (items: whacked, tired, exhausted, weary, worn out, lazy) was used to quantify the tiredness of each individual at the end of the cognitive testing. Effort and concentration were measured with the help of a visual analogue scale. The questions were: 'How strong was the effort?' and 'How much did you have to concentrate to accomplish the tasks successfully?' [extreme answers: 'very strong(ly)' versus 'not at all strong(ly)'].

Statistical analysis

Cardiovascular stress reactivity was calculated as simple difference score: Δ = Stress value − Baseline.

The effects of hydration status (within-subject factor: normal versus dehydration) and gender (between-subject factor) on baseline measures and cardiovascular stress-induced changes of heart rate, IBI, RMSSDibi and Finapres blood pressure were tested by mixed-design ANOVAs. Testing was carried out for each of the two stress tests, separately.

An ANOVA procedure was also used to assess baseline differences between Finapres versus cuff blood pressure (within-subject factor), and their potential interactions with hydration status (within-subject factor) and gender (between-subject factor).

All statistical calculations were performed with SAS software (release 8.0, WinNT, SAS Institute, Cary, NC, USA).

Power analysis

A pilot study of 16 healthy non-smokers (Philippsen *et al.*, 2007) was performed to assess reproducibility of CRTT systolic and diastolic blood pressure reactivity scores, as well as standard deviation of test–retest changes. Test–retest interval was 1 week. Testing was performed in the morning. All procedures were same as described above. Blood pressure reactivity scores on day 1 were (systolic, mean ± SD) 10 ± 9 mmHg, and (diastolic) 8 ± 6 mmHg. Reactivity scores on day 2 were 11 ± 8 mmHg, and 8 ± 4 mmHg, respectively. Pearson test–retest correlations of CRTT reactivity scores were: $r = 0{\cdot}55$, $P = 0{\cdot}03$ (systolic blood pressure), and $r = 0{\cdot}67$, $P = 0{\cdot}005$ (diastolic blood pressure). Differences (and standard deviation of differences) between day 1 and day 2 CRTT reactivity scores were: 1 ± 7·8 mmHg (systolic blood pressure), and 0·2 ± 4·6 mmHg (diastolic blood pressure).

The Power of the employed F-test model was calculated with GPOWER software (Faul & Erdfelder, 1992). Given a α-level of 0·05 and a sample size of 16 subjects the power $1 - \beta$ to detect a medium-sized effect of $\omega^2 = 0{\cdot}06$ is 0·49 for the F-test of the between-subjects factor (gender) and 0·81 for the F-tests of the within-subjects factor (dehydration) and the gender × dehydration interaction. As the power of the employed statistical tests is lower than the recommended power of 0·90, effect sizes are presented as a help to assess the relevance of the results*. According to Cohen (1992) an effect of $\omega^2 = 0{\cdot}01$ is considered as small, an effect of $\omega^2 = 0{\cdot}06$ as medium-sized and an effect of $\omega^2 = 0{\cdot}16$ as large. A small effect size indicates that even a test with a higher power would most likely not have detected a significant result, while a large effect size suggests that an insignificant finding is due to the insufficient power of the test (i.e. the too small sample size).

Results

Dehydration protocol

Plasma osmolality, urine sodium concentrations and urine osmolality increased significantly during dehydration (all P-values: $<0{\cdot}001$). A significant weight loss during the first 24 h occurred in the dehydration (mean decrease: 2·6%; $P<0{\cdot}001$) but not in the control condition. Moreover, subjective ratings of thirst were significantly higher in the dehydration than in the control phase ($P<0{\cdot}01$). Further evidence for the success of the dehydration protocol is presented by Szinnai *et al.* (2005).

Dehydration, gender and cardiovascular reactivity

Table 1 summarizes the effects of hydration status (normal versus dehydration) and gender on cardiovascular measures

Table 1 Influence of gender and hydration status on cardiovascular parameters at baseline and during stress tests.

					Main effect						Interaction		
	Women		Men		DH			Gender			DH* gender		
	Control	Dehydration	Control	Dehydration	F	P-value	ω^2	F	P-value	ω^2	F	P-value	ω^2
Baseline													
Heart rate (bpm)	65·1 (8·3)	63·4 (8·5)	61·0 (11·0)	60·8 (8·9)	0·33	0·29	0·00	0·61	0·45	0·00	0·20	0·66	0·00
Ibi (ms)	939 (112·0)	967 (121·0)	1018 (194·0)	1013 (151·0)	0·18	0·34	0·00	0·83	0·38	0·00	0·38	0·55	0·00
RMSSDibi (ms)	56·6 (19·7)	62·5 (29·0)	51·7 (32·0)	51·4 (19·5)	0·40	0·27	0·00	0·44	0·52	0·00	0·49	0·49	0·00
Finapres (mmHg)													
Syst bp	111·2 (15·7)	108·5 (5·6)	137·0 (14·8)	131·9 (11·1)	1·37	0·13	0·01	21·97	0·0003	0·40	0·14	0·72	0·00
Diast bp	60·2 (8·3)	57·8 (9·8)	72·8 (12·8)	69·0 (7·3)	1·13	0·16	0·01	9·37	0·01	0·21	0·05	0·82	0·00
Cuff (mmHg)													
Syst bp	104·1 (7·1)	103·6 (4·5)	122·4 (9·3)	119·0 (9·9)	1·55	0·11	0·02	20·63	0·0005	0·38	0·86	0·37	0·00
Diast bp	62·3 (16·4)	60·1 ± 6·1	68·0 ± 8·7	70·0 (9·7)	0·00	0·48	0·00	2·93	0·11	0·06	0·50	0·49	0·00
Δ CRTT													
Heart rate (bpm)	10·0 (6·1)	8·9 (3·3)	13·4 (3·5)	12·9 (5·2)	0·54	0·24	0·00	3·18	0·10	0·06	0·08	0·78	0·00
Ibi (ms)	−119·2 (61·6)	−122·6 (50·5)	−194·7 (90·1)	−184 (85·3)	0·04	0·43	0·00	4·52	0·05	0·10	0·15	0·70	0·00
RMSSDibi (ms)	−4·9 (27·2)	−22·6 (28·7)	−26·8 (21·9)	−23·9 (13·7)	3·21	0·05	0·06	1·33	0·27	0·01	4·73	0·04	0·10
Finapres (mmHg)													
Syst bp	8·0 (10·6)	5·2 (4·9)	12·8 (7·4)	15·3 (5·3)	0·01	0·47	0·00	5·50	0·03	0·12	1·95	0·18	0·03
Diast bp	5·5 (3·3)	4·8 (3·4)	10·1 (5·8)	11·3 (4·3)	0·05	0·42	0·00	9·10	0·01	0·24	0·63	0·44	0·00
Δ PASAT													
Heart rate (bpm)	7·4 (4·6)	6·7 (4·0)	14·7 (5·7)	12·0 (6·2)	4·73	0·03	0·10	6·48	0·02	0·15	1·50	0·24	0·02
Ibi (ms)	−92·6 (61·8)	−96·5 (61·9)	−202·7 (103·1)	−168·9 (92·2)	1·07	0·17	0·00	5·67	0·03	0·13	1·72	0·21	0·02
RMSSDibi (ms)	−7·2 (17·0)	−21·4 (18·8)	−24·4 (21·4)	−19·0 (12·2)	1·00	0·16	0·00	0·90	0·36	0·00	4·50	0·05	0·10
Finapres (mmHg)													
Syst bp	10·4 (14·4)	6·4 (7·0)	19·4 (9·0)	21·3 (5·0)	0·18	0·34	0·00	9·09	0·01	0·20	1·26	0·28	0·01
Diast bp	7·2 (6·0)	4·1 (5·0)	11·0 (4·9)	12·5 (3·7)	0·34	0·28	0·00	8·74	0·01	0·20	2·75	0·12	0·05

Data represent mean values, standard deviations are given in brackets.
CRTT, Choice Reaction Time Task; PASAT, Paced Auditory Serial Addition Task; CO, control; DH, dehydration; ibi, interbeat interval; syst bp, systolic blood pressure; diast bp, diastolic blood pressure; RMSSDibi, root mean square successive difference of interbeat intervals.
Conventions for the judgement of the effect size ω^2: $\omega^2 = 0{\cdot}01$ – small effect, $\omega^2 = 0{\cdot}06$ – medium-sized effect, $\omega^2 = 0{\cdot}16$ – large effect.

during baseline, CRTT and PASAT. Cuff blood pressure data were available during baseline, only.

In the PASAT a significant, medium-sized effect of dehydration on heart rate occurred ($P = 0{\cdot}03$). PASAT-induced heart rate reactivity decreased during dehydration. This effect was not observed in the CRTT ($P = 0{\cdot}24$). In the CRTT a significant, medium-sized effect of dehydration on the RMSSDibi was obtained ($P = 0{\cdot}05$) indicating a higher RMSSDibi in the dehydration than in the normal hydration condition. This effect, however, was carried solely by the increased RMSSDibi of females in response to the CRTT (see below). The effect of dehydration on the RMSSDibi did not reach significance in the PASAT ($P = 0{\cdot}16$). No main effects of dehydration on IBI, systolic and diastolic blood pressure could be found; neither at baseline nor for cardiovascular reactivity during CRTT, and PASAT (all P-values: >0·10).

Several medium-sized to large effects of gender on cardiovascular parameters appeared. At baseline significant effects of gender were obtained for systolic and diastolic blood pressure data. Men had higher values than women. In the CRTT significant effects of gender were found for stress-induced changes of IBI, systolic and diastolic blood pressure. Reactivity scores were higher in men than in women. Concerning PASAT-induced reactivity scores significant effects of gender occurred for heart rate, IBI, systolic and diastolic blood pressure. Again, all reactivity scores were higher in men than in women.

No significant interaction effects of hydration status × gender were observed for baseline and stress-induced changes of heart rate, IBI, systolic and diastolic blood pressure. However, for RMSSDibi an interaction between hydration status × gender was found for both, CRTT- and PASAT-induced reactivity scores (CRTT: $P = 0{\cdot}04$; PASAT: $P = 0{\cdot}05$). Paired t-tests revealed that the effect of the hydration status on stress-induced changes of RMSSDibi is only significant in females, this is true for the CRTT [$t(7) = 2{\cdot}74$, $P = 0{\cdot}02$], as well as the PASAT [$t(7) = 2{\cdot}47$, $P = 0{\cdot}03$]. Hydration status did not effect stress-induced changes of RMSSDibi in males [CRTT: $t(7) = 0{\cdot}06$, $P = 0{\cdot}95$; PASAT: $t(7) = 0{\cdot}11$, $P = 0{\cdot}92$].

Effort, concentration and tiredness

Subjects reported significantly higher effort, concentration and tiredness in the dehydration compared with the control condition (all P-values: <0·05; see Szinnai *et al.*, 2005).

Finapres versus cuff blood pressure

The comparison of the two blood pressure measurement methods revealed higher Finapres systolic [$F(1;14) = 7{\cdot}48$, $P<0{\cdot}001$] blood pressure data, with a significant interaction of method × gender on systolic blood pressure [$F(1;14) = 5{\cdot}31$, $P<0{\cdot}03$] indicating that Finapres overestimation was more pronounced in men. This interaction approached significance for diastolic blood pressure data [$F(1;14) = 3{\cdot}1$, $P = 0{\cdot}09$], too. However, there was no main effect of the blood pressure measurement method on diastolic blood pressure data. In no case these patterns were further obscured by the hydration status.

Discussion

Only few authors addressed the influence of hydration status on cardiovascular reactivity to mental stress (Rochette & Patterson, 2005; Veldhuijzen van Zanten *et al.*, 2005). As far as we know, there have been no studies that investigated the influence of mild dehydration on the cardiovascular reactivity to psychological stress, which is astonishing since dehydration caused by exercise or reduced water consumption is very common in many people. The present study aimed to investigate the impact of water deprivation on cardiovascular reactions to stress by testing the same participants both under dehydration and normal hydration conditions. Given the absence of any main effect of the hydration status on stress-induced blood pressure reactivity we conclude that dehydration does not substantially influence blood pressure reactivity to stress.

There are two possible explanations for the absence of an effect of dehydration on blood pressure reactivity. Mechanisms responsible for the compensatory adjustment of blood pressure during dehydration (i.e. vasopressin, and increased sympathetic nervous system activity) may interact with stress reactivity (Rivier & Vale, 1983) in a complex fashion, and compensate for any potential dehydration effects. Secondly, the participants of this study reported a significantly higher level of tiredness during the behavioural tasks and a higher level of concentration necessary to achieve the task requirements during the dehydration condition (Szinnai *et al.*, 2005). Both, the higher tiredness and the higher concentration might have had a decreasing effect on the blood pressure reactivity to stress, thus compensating for a dehydration-induced increase in the blood pressure reactivity.

Dehydration had little effects on heart rate. There was a medium-sized effect of dehydration on heart rate in the PASAT. A similar finding was not obtained for CRTT-induced heart rate reactivity. This discrepancy might be related to the characteristics of the employed tests, as the PASAT requires a verbal report, whereas the CRTT is strictly non-verbal. Speaking is in complex interaction with cardiovascular reactions and the functioning of the autonomic nervous system (Freed *et al.*, 1989). Therefore, we suggest considering the hydration status as a potentially important covariate especially when studying heart rate reactivity to stressors which require verbal activity (such as the PASAT).

Reduction of parasympathetic activity during stress may contribute to cardiac vulnerability (Binkley *et al.*, 1991; Gulli *et al.*, 2001). Indeed, parasympathetic withdrawal was found during CRTT (Langewitz *et al.*, 1994) and other stress tests (Buchholz *et al.*, 2003). This, however, is the first study to suggests parasympathetic withdrawal induced by the PASAT. Furthermore, we could show that this effect is in complex interaction with gender, and the hydration status. During the control condition women had smaller stress-induced parasympathetic withdrawal, a protective pattern in accordance with the reduced female cardiovascular event rate (Vögele *et al.*, 1997). However, dehydration enhanced stress-induced parasympathetic withdrawal in females, so that stress-induced parasympathetic withdrawal was similar in men and women in the dehydration condition. This suggests that dehydration may induce a harmful parasympathetic response pattern in females.

Gender differences in cardiovascular reactivity have been frequently documented (Allen *et al.*, 1993; Lawler *et al.*, 1995; Traustadottir *et al.*, 2003). We found greater stress-induced changes in heart rate, IBI, systolic and diastolic blood pressure in men. However, other authors have reported different results (Stone *et al.*, 1990). This discrepancy may be related to the set of stressors used in the current study, as it was suggested that women and men are sensitive for different kinds of stressors (Stroud *et al.*, 2002).

Some limitations of this study have to be considered. We studied healthy young subjects with rather homogenous education level only. Therefore, our study does not exclude that other participants, elderly, or patient groups might have shown other results. It remains also unknown whether dehydration may impact stress reactivity in response to other stress eliciting conditions, such as psychosocial or physical stressors. However, PASAT and CRTT are truly challenging tasks that are frequently used to measure cognitive-motor functioning of individuals (Diamond *et al.*, 1997; Schächinger *et al.*, 2003; Dirette, 2004; Shucard *et al.*, 2004; Au Duong *et al.*, 2005; Szinnai *et al.*, 2005). Different authors have used them successfully to induce stress (Langewitz *et al.*, 1994; Schächinger *et al.*, 2000; Mathias *et al.*, 2004; Philippsen *et al.*, 2007), and the range of cardiovascular responses elicited by these tests is comparable with that of other mental stress tests (Becker *et al.*, 1996; Hoshikawa & Yamamoto, 1997).

Several blood pressure measurement techniques have been employed in psychophysiological stress research, such as invasive direct intra-arterial measurement of blood pressure, intermittent non-invasive auscultatory and oscillometric methods, as well as continuous Finapres methodology. From all these methods, the latter is most likely affected by blood volume changes since peripheral reflex vasoconstriction may result from blood volume loss and Finapres accuracy is limited during states of peripheral vasoconstriction (Imholz *et al.*, 1992). Thus, we aimed to determine Finapres accuracy during the dehydration condition. Dinamap reference blood pressure

readings were taken during prestress baseline while Finapres blood pressure was assessed at the opposite arm. Such comparisons could not be performed during stress conditions as it may have had interfered with the task requirements. We found that Finapres overestimates intermittent cuff blood pressure, and that this overestimation is more pronounced in males than females. However, we did not find any interaction with the hydration status. Thus, Finapres accuracy was not affected by our experimental treatment.

We conclude that moderate dehydration induced by water restriction has no – or little – effect on blood pressure reactivity to mental stress. The effects on heart rate reactivity are small to moderate. However, stress-induced parasympathetic withdrawal may be fortified during dehydration in females, which suggests controlling for water intake when studying such responses.

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