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A Novel Perspective on Water Intake's Benefits on Cardiovascular Function: A Simple Strategy for Heart Health

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Abstract

Background

Hydration has been increasingly recognized as a modifiable factor with potential cardiovascular relevance. Whether acute water intake improves hemodynamic, vascular, and autonomic outcomes—and whether these responses differ according to home blood pressure phenotype—remains insufficiently characterized.

Aim

To evaluate the effects of acute water intake on cardiovascular and autonomic parameters in men, and to explore associated changes in circulating renin–angiotensin system peptides.

Methods

Thirty-seven healthy men underwent two laboratory assessments under standardized conditions: after fasting (post-fasting) and after acute water intake (post-water). Participants were categorized by home-based blood pressure as normal (N-BP; <130/≤80 mmHg) or abnormal (Ab-BP; ≥130 and/or > 80 mmHg). Hemodynamic and vascular parameters—including peripheral and central blood pressure indices, augmentation index, and pulse wave velocity—were assessed, alongside electrocardiography-derived heart rate variability spectral analysis. In a subset (N = 11), plasma RAS peptides were quantified by liquid chromatography–tandem mass spectrometry.

Results

Post-water intake, participants showed significant drops in renin-angiotensin peptides, heart rate, and augmentation index, with notable rises in very low frequency and heart rate variability, indicating robust autonomic modulation and potential cardiovascular benefits.

Conclusions

Acute water intake modulates hemodynamic, vascular, and autonomic parameters in men, with phenotype-dependent responses based on home blood pressure. Thus, hydration is a simple, impactful strategy to enhance cardiovascular health and guide personalized care.

Highlights

- Acute water intake improves autonomic balance.
- Acute water intake improves vascular parameters.
- Personalized hydration strategies show cardiovascular benefits

INTRODUCTION

Despite extensive research, determining the ideal individual water consumption for maintaining proper hydration remains a challenge due to the variability in water needs linked to cultural norms, metabolism, physical activity, and environmental conditions (McKinley & Johnson, 2004; Noda & Matsuda, 2022). Low water intake is associated with an increased risk of chronic diseases (Anon, 2005). For cardiovascular and renal health, maintaining sufficient plasma volume is crucial for renal perfusion and the balance of vasoactive substances (Zhang et al., 2019). Proper body hydration regulates blood pressure (BP) and facilitates nutrient transport while removing waste; however, insufficient plasma volume can impair kidney function and elevate cardiovascular stress, underscoring the need for individualized hydration strategies (Watso & Farquhar, 2019).

Understanding the role of the autonomic nervous system and vasoactive peptides, particularly angiotensin II (Ang II), is essential. The activity of Ang II correlates with its concentration, and increased levels of this peptide lead to negative pathophysiological effects (Watso & Farquhar, 2019). Adequate hydration supports renal perfusion, reduces renin release, and contributes to BP regulation (Zhang et al., 2019). In contrast, dehydration elevates Ang II levels, resulting in endothelial dysfunction and sympathetic activation, which promotes cardiovascular failure outcomes (Manz, 2007; Robinson et al., 2017).

In fact, water consumption significantly influences the autonomic nervous system and hemodynamic parameters (Oyewunmi et al., 2023). Signals from peripheral sensors that detect fluid volume initiate responses in the central nervous system, allowing for adjustments to maintain homeostasis (Forstenpointner et al., 2022). Notably, elderly individuals who are dehydrated often report lower levels of thirst while exhibiting higher renin activity and vasopressin concentrations, suggesting that the perception of thirst changes with age (Takamata et al., 1999). This highlights the importance of personalized hydration strategies that do not rely solely on thirst (McKinley & Johnson, 2004).

Surprisingly, despite water constituting a large proportion of body composition, few studies have emphasized its critical role in reducing cardiovascular risk (Watso & Farquhar, 2019). Recognizing the integral role of water in BP modulation and renal filtration (Malisova et al., 2016;Barroso et al., 2021a;Whelton et al., 2018a). Our hypothesis posits that personalized and optimal hydration levels are crucial for renal perfusion. Therefore, this investigation included subjects with both normal and abnormal BP regulation profiles to explore the impact of hydration on autonomic control and hemodynamic parameters.

Materials and Methods

This cross-sectional study was approved by the Ethics Committee of the Universidade Federal de Ciências da Saúde de Porto Alegre (UFCSPA; CAAE 9751819.4.0000.5345), and all procedures adhered to the principles of the Declaration of Helsinki. Data were collected in a quiet room (23 ± 1 °C) between March and May 2023.

This is a cross-sectional study, utilizing a convenience sample of 37 male individuals. The inclusion criteria were adults aged 18 years or older, without any diagnosed preexisting conditions, and not undergoing medication treatment. The exclusion criterion was female individuals, due to potential variations in their current estrogen cycle.

During the initial contact, we provided a detailed explanation of the procedures and informed participants about the requirement for two appointments scheduled one week apart. In the first appointment, after signing informed consent forms and completing a questionnaire, participants' medical histories were reviewed. Blood pressure was measured in both arms to determine which arm exhibited the higher average blood pressure; this arm was subsequently selected for home measurements. Participants received training in home measurement techniques (Stergiou et al., 1998, 2010) to mitigate the effects of white coat hypertension and masked hypertension. Each participant was provided with a standardized blood pressure monitor (OMRON®, Omron Dalian Co., Ltd., model HEM-7113).

At home, while fasting and with an empty bladder, volunteers conducted three measurements of heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) at one-minute intervals over four consecutive days. This procedure was repeated before the last meal of the evening, resulting in a total of 24 home readings (Williams et al., 2018; Villar et al., 2020).

Seven days after the start of measurements, volunteers returned to the UFCSPA Translational Laboratory in the morning, having fasted for 8–10 hours while avoiding caffeine, other stimulants, strenuous exercise, and alcohol for 24 hours prior to the visit. They underwent an electrocardiogram (ECG), vascular function tests, and venous blood collection from the cubital fossa. After hydrating until their bladder felt full, they emptied it, and the measurements were repeated. All blood pressure measurements were analyzed for categorization into two groups: normal BP (SBP; SBP<130 and DBP≤80 mmHg) and abnormal BP (ABP; SBP ≥ 130 and/or DBP>80 mmHg) (Whelton et al., 2018b; Barroso et al., 2021b).

Electrocardiogram acquisition

ECG recordings lasting 10 minutes were obtained from fasting subjects in the morning, both pre- and post-water consumption. The subjects lay supine in a tranquil environment, without jewelry or metal, and remained still during the recording. ECGs were captured at 600 Hz using a Wincardio system (Micromed Biotecnologia, batch 53, series no. 1444107, ANVISA registration: 10,307,270,007) and later subjected to spectral analysis using an autoregressive model.

Autonomic evaluation

After detecting the pulse intervals, the heart period was automatically calculated on a beat-to-beat basis as the time interval between two consecutive systolic peaks, also known as pulse interval (PI). All

detections were carefully checked to avoid erroneous or missed beats. Sequences of 200 beats were randomly chosen; if they presented nonstationary episodes, they were discarded, and a new random selection was performed. The stationarity of the series was tested as previously reported (Magagnin et al., 2011). Frequency domain analysis of heart rate variability (HRV) was conducted using an autoregressive algorithm on PI sequences (tachograms). The power spectral density was calculated for each time series.

In this study, three spectral components were considered: very low frequency (VLF) from 0 to 0.04 Hz, low frequency (LF) from 0.04 to 0.15 Hz, and high frequency (HF) from 0.15 to 0.5 Hz. The spectral components were expressed in absolute units (LFa or HFa; s^2) and normalized units (LFnu or HFnu; %). Normalization involves dividing the power of a given spectral component by the total power and then multiplying the ratio by 100 (Montano et al., 1994). Additionally, the absolute LF/HF ratio, which represents cardiac sympathetic and parasympathetic balance, was calculated for each segment (Montano et al., 2009).

Vascular parameters

Subsequently to the ECG data collection, the peripheral (p) SBP (pSBP), DBP (pDBP), and pulse pressure (pPP), as well as central (c) cSBP, cDBP, and cPP data were recorded. Furthermore, HR, cardiac output (CO), cardiac index (CI), total vascular resistance (TVR), pulse wave velocity (PWV), and augmentation index (Aix) were measured before and after drinking water using a validated oscillometric BP measurement device (Mobil-O-Graph 24 h PWA Monitor, IEM GmbH, Stolberg, Germany) (Trachet et al., 2010; Hametner et al., 2013). The cuff was placed around the participant's arm, and three automated readings were taken at three-minute intervals to calculate all parameters. Subsequently, the average of the three measurements was determined. PWV estimates were derived from built-in ARCSolver algorithms.

Determination of plasma concentrations of vasoactive substances by liquid chromatography-tandem mass spectrometry (LC-MS/MS)

Samples containing 4 mL of venous blood were collected in EDTA tubes with the P8340 protease inhibitor cocktail from Sigma-Aldrich (Merck). After centrifugation at refrigerated temperatures, the plasma was aliquoted and stored for analysis of renin-angiotensin system peptides as follows: An aliquot of 950 µL acetone was added to 50 µL of plasma samples and shaken for 60 seconds. Following this, the samples were centrifuged at 9,000 × g for 6 minutes, and the supernatant was collected, dried under nitrogen, and resuspended in 50 µL of acetonitrile immediately before analysis.

The analytical system included a Nexera-i LC-2040C coupled with an LCMS-8045 triple quadrupole mass spectrometer (Shimadzu, Kyoto, Japan). Electrospray ionization parameters in positive mode were set as follows: capillary voltage at 4,500 V; desolvation line temperature up to 250°C; heating block temperature

up to 400°C; drying gas flow up to 10 L/min; and nebulizing gas flow at 3 L/min. Multiple reaction monitoring (MRM) was conducted using the following fragmentation patterns. Alamandine: m/z 428.45 → 110.10; 428.45 → 211.15; 428.45 → 11.10. Ang I: m/z 649.10 → 110.10; 649.10 → 269.10; 649.10 → 426.25. Ang A: m/z 501.80 → 70.10; 501.80 → 263.10; 501.80 → 110.05. Ang-(1–7): m/z 450.30 → 110.10; 450.30 → 70.10; 450.30 → 392.65. Ang II: m/z 523.80 → 263.20; 523.80 → 70.20; 523.80 → 110.15.

Chromatographic separation was performed using an Acquity UPLC® C18 column (2.1 x 50 mm, 1.7 µm particle size) (Waters Corporation, Ireland) in gradient elution mode at a flow rate of 0.3 mL/min. The mobile phase consisted of water (solvent A) and acetonitrile (solvent B), both fortified with 0.1% formic acid, programmed as follows: 0–0.5 min, 2% B; 0.5–3.0 min, 2–100% B; 3.0–3.5 min, 100% B; 3.5–3.8 min, 100–2% B; 3.8–8 min, 2% B. The column oven was maintained at 50°C. Data were processed using LabSolutions software (Shimadzu, Kyoto, Japan).

Statistical analysis

The sample size was determined to check the data for normal distribution, applying the Shapiro-Wilk test. Depending on whether the data were parametric or non-parametric, either a paired t-test or a Wilcoxon matched-pairs signed-rank test was used, as specified in the table legends. A simple linear regression analysis was conducted to explore the association between the volume of water intake per body weight and various independent variables, including VLF, LFa, HFa, LFnu, HFnu, HRV, HR, LF/HF ratio, 0V, 1V, 2LV, 2UV, SBP, DBP, PP, PWV, AIx, cSBP, cDBP, cPP, CO, TVR, and CI. The linear association of each variable with the normalized water intake was scrutinized.

To assess the association between water intake and all parameters, considering the patients' mean arterial pressure (MAP), we conducted a repeated measures analysis of covariance (ANCOVA) with adjustment for MAP (supplementary tables). Prior to conducting the ANCOVA, we tested the assumptions of independence between the dependent variable and the covariate, as well as the homogeneity of variance of the regression predictors. Both assumptions were met, as indicated by appropriate statistical tests.

Given the sample size of the study and the adjustment for MAP, we opted to consider the entire sample for the analyses instead of stratifying by normal or altered pressure. We compared the values of the parameters of interest pre- and post-water administration. The ANCOVA was performed using SPSS Version 20 and Microsoft Excel 2010 (SPSS Version 20, IBM Corp., Armonk, NY, USA; Microsoft Excel 2010, Microsoft Corporation, Redmond, WA, USA). Results were considered significant at a significance level of $P < 0.05$.

RESULTS

Table 1 presents the cohort characteristics, along with mean SBP, DBP, PP, and HR measured at home, which were used to classify participants into the BP-N or BP-Ab groups. These BP measurements may

differ from those obtained in the laboratory. The groups were separated based on the criteria for normal and abnormal BP, which stratifies our analysis by SBP and DBP values. Furthermore, to ensure pressure adjustment and reliability of the results, we also conducted an analysis of covariance (ANCOVA) adjusted for BP values. The volume of water consumed did not differ significantly between the groups, whether measured in milliliters (N-BP = 1698 ± 408; Ab-DP = 1732 ± 727, p = 0.86) or in mL/kg (N-BP = 19.65 ± 10.62; Ab-DP = 20.16 ± 4.78, p = 0.84).

Table 1
Cohort Characteristics According to Home-Based Blood Pressure Groupings

Characteristic	N-BP (n = 26)	Ab-BP (n = 11)	P
Age (years)	33.12 ± 9.54	33.09 ± 8.49	0.990
Body mass index (kg / m^2)	27.28 ± 5.03	30.23 ± 6.42	0.140
Water consumption (ml)	1698 ± 408	1732 ± 727	0.860
Water consumption (ml/kg)	19.65 ± 10.62	20.16 ± 4.78	0.840
SBP, mmHg	117.76 ± 7.55	135.64 ± 7.98	$2x10^{-8}$
DBP, mmHg	66.92 ± 7.20	80.45 ± 13.27	0.003
PP, mmHg	51.00 ± 7.54	54.91 ± 13.00	0.262
HR	70.41 ± 8.20	77.36 ± 12.26	0.052

N-BP= normal and Ab-BP= abnormal blood pressure (BP) groups, according to Home-Based BP; SBP= Systolic BP; DBP= Diastolic BP; PP= pulse pressure; HR= heart rate. The Student–Newman–Keuls method was employed for the comparison of unpaired samples. The data is presented as means ± standard deviation, with a P-value of 0.05 or less denoting statistical significance.

Table 2 shows that after water consumption, there was a notable improvement in cardiovascular parameters in both groups, with significant increases in cSBP and pDBP, accompanied by a reduction in HR. However, in the N-BP group, there was a significant reduction in HR, pPP, CO, IC, AIx, and an increase in TVR, cSBP, cDBP, and pDBP. The pSBP before and after water intake did not change in the N-BP group but increased in the Ab-BP group. In the Ab-BP group, no significant changes were observed in cPAD, cPP, pPP, CO, IC, TVR, and AIx; however, pPAS, pPAD, and PWV increased remarkably.

Table 2
Comparison of laboratory-measured hemodynamic values according to home-based blood pressure groupings.

N-BP (n = 26)				Ab-BP (n = 11)		
	Pre-water	Post-water	P	Pre-water	Post-water	P
cSBP, mmHg	108 ± 14.74	110 ± 9.45	0.041	120 ± 24	126 ± 25	0.016
cDBP, mmHg	76 ± 10.25	80 ± 9.33	0.001	88 ± 23	92 ± 24	0.062
cPP, mmHg	31 ± 7.74	29 ± 5.50	0.126	32 ± 5.79	34 ± 8.50	0.212
pSBP, mmHg	119 ± 14	120 ± 8.27	0.424	132 ± 22	137 ± 23	0.056
pDBP, mmHg	75 ± 10.27	80 ± 10.07	0.001	86 ± 23	91 ± 24	0.033
pPP, mmHg	44 ± 9.44	40 ± 7.05	0.015	45 ± 10.18	45 ± 9.82	0.471
HR, bpm	71 ± 13.99	59 ± 9.88	0.001	72 ± 9.63	63 ± 11.42	0.005
CO, L/min	5.04 ± 0.85	4.76 ± 0.70	0.017	4.70 ± 0.72	4.72 ± 1.14	0.563
TVR, dyns/cm ^{5}	1.11 ± 0.10	1.20 ± 0.12	0.005	1.32 ± 0.23	1.39 ± 0.28	0.236
CI, L/min/m²	2.49 ± 0.51	2.338 ± 0.41	0.014	2.15 ± 0.49	2.23 ± 0.56	0.918
PWV, m/s	5.65 ± 0.79	5.62 ± 0.68	0.627	6.00 ± 1.04	6.12 ± 1.06	0.036
AIx (%)	8.93 ± 7.56	2.67 ± 5.91	0.001	11.09 ± 7.11	8.69 ± 8.92	0.182

BP = blood pressure; N-BP= normal and Ab-BP= abnormal BP groups, according to Home-Based BP; Pre= moment before drinking water; Post= moment after drinking water; p=peripheral; c= central; cSBP and pSBP= central and peripheral systolic BP, respectively; cDBP and pDBP= central and peripheral diastolic BP, respectively; cPP and pPP= central and peripheral pulse pressure, respectively; HR= heart rate; CO= cardiac output; TVR= total vascular resistance; CI= cardiac index; PWV= pulse wave velocity; AIx= augmentation index. The Wilcoxon was employed for the comparison of unpaired samples. The data is presented as means ± standard deviation, with a P-value of 0.05 or less denoting statistical significance.

After water consumption, both groups showed a significant increase in the LFa, HFa, HRV, and VLF components. Importantly, in the N-BP group, there was a significant reduction in the LF/HF ratio, suggesting improved sympathovagal balance in these individuals (Table 3).

Table 3
Comparison of laboratory measured Spectral and symbolic analysis values according to home-based blood pressure groupings.

	Spectral Analysis					
	N-BP (n = 26)			Ab- BP (n = 11)		
	Pre-water	Post-water	P	Pre-water	Post-water	P
VLF (ms^2)	877 ± 667	1585 ± 1646	0.032	872 ± 1153	2747 ± 4173	0.008
HRV (ms^2)	3079 ± 2786	6006 ± 7262	0.002	3006 ± 4043	7295 ± 9993	0.003
LFa (ms^2)	1116 ± 1523	2366 ± 3850	0.001	1010 ± 1254	2111 ± 2114	0.003
HFa (ms^2)	1069 ± 1434	2041 ± 2551	0.001	1107 ± 1743	2402 ± 3372	0.006
LFnu	56 ± 22	52 ± 17	0.155	60 ± 16	61 ± 16	0.304
HFnu	41 ± 21	44 ± 16	0.309	40 ± 16	38 ± 16	0.304
LF/HF ratio	2.24 ± 2.13	1.42 ± 0.83	0.045	2.11 ± 1.68	2.26 ± 1.95	0.423
	Symbolic Analysis					
	Pre-water	Post-water	P	Pre-water	Post-water	P
0V pattern	0.21 ± 0.12	0.17 ± 0.09	0.158	0.22 ± 0.12	0.23 ± 0.12	0.441
1V pattern	0.45 ± 0.07	0.43 ± 0.10	0.757	0.46 ± 0.05	0.45 ± 0.04	0.332
2LV pattern	0.11 ± 0.04	0.10 ± 0.04	0.302	0.12 ± 0.06	0.11 ± 0.05	0.629
2UV pattern	0.20 ± 0.15	0.24 ± 0.15	0.023	0.18 ± 0.18	0.19 ± 0.09	0.332

N-BP= normal and Ab-BP= abnormal blood pressure (BP) group, according to Home-Based BP; Pre= moment before drinking water; Post= moment after drinking water; VLF= Very low frequency component; HRV= Heart rate variability; LF = Low frequency component; HF= High frequency component; a=absolute values; nu= normalized units; 0V = no variation; 1V = one variation; 2LV = two low variations and 2UV = two upper variations. Although some data are parametric, the Wilcoxon matched pairs signed rank was employed. The data is presented as means ± standard deviation, with a P-value of 0.05 or less denoting statistical significance.

As expected, Table 4 illustrates the drastic reduction in the plasma concentration of renin-angiotensin system peptides after water ingestion. The results were consistent across both groups, even when combined. Considering that the significance of an independent variable does not inherently imply causality, it's crucial to interpret these findings with caution. (Schneider et al. 2010).

Table 4
– Renin-Angiotensin System peptides plasma concentration (N = 11)

Variables (ng/mL)	Pre-water	Post-water	P
Angiotensin I	0.1188 ± 0.0165	0.0876 ± 0.0117 (-26%)	0.000018
Angiotensin II	0.0539 ± 0.0098	0.0377 ± 0.0110 (-30%)	0.000135
Angiotensin A	0.1011 ± 0.0171	0.0720 ± 0.0171 (-29%)	0.000062
Angiotensin 1–7	0.0551 ± 0.0096	0.0388 ± 0.0089 (-29%)	0.000141
Alamandine	0.1532 ± 0.0252	0.1047 ± 0.0234 (-32%)	0.000154
Values expressed as mean ± standard deviation.			

The results in Table 5 clearly demonstrate that the relationship between volume ingested (in milliliters) and body weight (in kilograms) is not associated with the specific cardiovascular variables under study, except for the variables HR, VLF, HRV, and HFa, which showed a significant association with water volume ingested.

Table 5
– Simple Linear Regression Analysis of the Association Between Water Intake in milliliters and Dependent Variables.

			Confidence interval			
	R^2	P_1	Coefficient	Lower 95%	Superior 95%	P_2
pSBP	0.000	0.934	0.006	-0.142	0.154	0.934
pDBP	0.011	0.548	-0.044	-0.193	0.104	0.548
pPP	0.027	0.339	0.137	-0.150	0.425	0.339
HR	0.195	0.007	-0.293	-0.500	-0.850	0.007
cSBP	0.003	0.759	-0.021	-0.161	0.118	0.759
cDBP	0.004	0.701	-0.029	-0.179	1,220	0.701
cPP	0.000	0.942	0.013	-0.342	3,680	0.942
CO	0.095	0.068	-2.58	-5,359	0.200	0.068
TVR	0.104	0.055	11.09	-0.258	22,438	0.055
CI	0.001	0.849	-0.501	-5.809	4,807	0.849
PWV	0.052	0.181	-1,864	-4.639	0.910	0.181
AIx	0.000	0.980	-0.004	-0.330	0.322	0.980
VLF	0.243	0.002	0.001	0.000	0.002	0.002
HRV	0.194	0.007	0.000	0.000	0.001	0.007
LFa	0.074	0.110	0.001	0.000	0.001	0.110
HFa	0.171	0.012	0.001	0.000	0.002	0.012
LFnu	0.069	0.123	-10	-23	2,931	0.123
HFnu	0.087	0.081	12	-1,588	26	0.081
LF/HF	0.037	0.037	-1.013	-2.812	0.785	0.260
0V	0.087	0.081	-19	-40	2,420	0.081

P= peripheral; c= central; BP = blood pressure; SBP= Systolic BP; DBP= Diastolic BP; PP= pulse pressure; HR= heart rate; CO= cardiac output; TVR= total vascular resistance; CI= cardiac index; PWV= pulse wave velocity; AI= augmentation index; VLF= Very low frequency component; HRV= Heart rate variability; LF = Low frequency component; HF= High frequency component; a=absolute values; nu= normalized units. 0V = no variation; 1V = one variation; 2LV = two low variations and 2UV = two upper variations. P_1 represents the significance level obtained from the ANOVA test, whereas P_2 denotes the significance level derived from the Simple Linear Regression analysis. In both cases, a P-value of 0.05 or lower indicates statistical significance .

			Confidence interval			
	R^2	P_1	Coefficient	Lower 95%	Superior 95%	P_2
1V	0.010	0.567	-7,274	-33	18	0.567
2LV	0.006	0.648	10	-37	58	0.648
2UV	0.092	0.072	15	-1,445	32	0.072

P= peripheral; c= central; BP = blood pressure; SBP= Systolic BP; DBP= Diastolic BP; PP= pulse pressure; HR= heart rate; CO= cardiac output; TVR= total vascular resistance; CI= cardiac index; PWV= pulse wave velocity; AI= augmentation index; VLF= Very low frequency component; HRV= Heart rate variability; LF = Low frequency component; HF= High frequency component; a=absolute values; nu= normalized units. 0V = no variation; 1V = one variation; 2LV = two low variations and 2UV = two upper variations. P_1 represents the significance level obtained from the ANOVA test, whereas P_2 denotes the significance level derived from the Simple Linear Regression analysis. In both cases, a P-value of 0.05 or lower indicates statistical significance .

DISCUSSION

Our study provides novel evidence that water intake, intended to stimulate renal filtration, significantly improves both sympathetic and parasympathetic contributions to cardiac function and peripheral hemodynamic, independent of the total water volume consumed. This effect was more pronounced in N-BP individuals, with notable enhancements observed in VLF, LF/HF ratio, HRV, and AIx. In fact, these parameters are predictors of CV risk (Algra et al., 1993; Tsuji et al., 1994; Janner et al., 2012a), highlighting the potential protective benefits of proper hydration on CV health.

These results are consistent with the literature demonstrating that the acute consumption of water, particularly in volumes ranging from 350 to 500 mL, can elevate SBP and DBP and reduce heart rate in patients with orthostatic hypotension. These effects are notably less pronounced in individuals without comorbidities (Oyewunmi et al., 2023). Conversely, increasing hydration dilutes the blood and reduces the concentration of vasoactive peptides such as angiotensin II, which can have a positive impact on cardiovascular health, particularly when maintained over the long term.

Furthermore, in the N-BP participants, the ingested water volume was likely evenly distributed across the circulatory system. Owing to the superior elasticity of the aortic artery, there was a modest elevation in cSBP by 2 mmHg and in cDBP by 4 mmHg. This rise in cDBP suggests that the aorta's elastic properties efficiently support blood flow during diastole, contributing to an increase in TVR. Additionally, in the N-BP group, water intake resulted in a decrease in pPP, likely because of the notable increase in pDBP without a corresponding increase in pSBP. This aligns with the literature that links lower ppP to a reduced risk of coronary heart disease (Franklin et al., 1999a).

However, the Ab-BP group had a less favorable vascular response, with equivalent increases in pSBP and pDBP maintaining pPP. The absence of hemodynamic improvement, as indicated by the significant increase in cSBP without an adequate decrease in cPP, likely suggests lower aortic elasticity compared

to the N-BP group. Additionally, in the Ab-BP group, the reduction in HR was not sufficient to decrease CO, since the LF/HF ratio remained unchanged after water ingestion. This indicates that there was no decrease in sympathetic involvement necessary for HR reduction.

Our findings partially agree with existing research showing a significant elevation in pSBP and pDBP following 480 mL of consumption (Jordan et al., 2000). The authors attributed this to heightened sympathetic participation without a corresponding decrease in HR, possibly because of autonomic failure. Similarly, Cariga and Mathias documented high pSBP and pDBP in patients with sympathetic denervation and autonomic failure, accompanied by a reduction in HR yet a stable CO (Cariga & Mathias, 2001). Such outcomes are anticipated in patients with autonomic dysfunction, which adversely affects cardiovascular control.

Conversely, studies involving young, healthy individuals have demonstrated an improvement in the gain of arterial baroreflex control following water consumption (Brown et al., 2005a; Vianna et al., 2018), which contributes to BP maintenance in this situation (Vianna et al., 2018). This phenomenon, known as the osmopressor response, appears to be mediated by the activation of the sympathetic nervous system (Jordan et al., 2000; May & Jordan, 2011). According to Routledge et al., water intake induces sympathetic vasoconstriction without changing BP of healthy young adults, potentially due to a simultaneous increase in cardiac vagal control. Their findings confirm an elevation in parasympathetic activity and HRV, effectively assessed using ambulatory ECG. (Tsuji et al., 1994; Routledge et al., 2002).

We observed an increase in sympathetic and parasympathetic participation in both groups. However, it is noteworthy that only the N-BP group exhibited a pronounced decrease in the LF/HF ratio, indicating a relative reduction in sympathetic in relation to parasympathetic participation. This result aligns with literature suggesting that hydration promotes a reduction in sympathetic activity as a side effect of enhanced vagal participation due to gastric distention (Charkoudian et al., 2005; Yun et al., 2005). Furthermore, an increase in HRV and a significant increase in the VLF component were observed in both groups after water consumption. This outcome is beneficial, as there is an established inverse correlation between VLF and the incidence of sudden cardiac death (Bigger et al., 1993; Tsuji et al., 1994; Hadase et al., 2004; Schmidt et al., 2005).

Furthermore, data from the literature demonstrated that water consumption improves autonomic control, HRV, arterial function, and wave reflection in healthy young individuals (Brown et al., 2005b; May & Jordan, 2011). These findings highlight the pivotal role of the autonomic nervous system in maintaining cardiovascular health through its precise modulation of heart rate.

In the N-BP group, a significant decrease in the HR was observed, which was associated with a corresponding reduction in CO. This reduction likely represents a compensatory mechanism in response to a slight elevation of TVR, probably due to an increase in circulating volume. Furthermore, considering that the PWV is inversely proportional to the elasticity of the arterial wall and to the blood density, it is not surprising that there was this slight increase in PWV only in Ab-BP group after water load. The

decrease in blood viscosity facilitates the pulse wave propagation due to the reduced friction of the blood cells with the vessel wall.

Notably, according to the literature (Janner et al., 2012b), the substantial reduction in AIx, which was confirmed by ANCOVA analysis, indicates a decrease in SBP, potentially due to the timing of the reflected wave shifting to align more with the diastolic phase following water intake, —a change that favors coronary perfusion. This concept is further corroborated by the increase in cDBP and pDBP, paired with the significant decrease in pPP, reinforcing the notion of a beneficial cardiovascular response in normotensive subjects (Franklin et al., 1999b) .

In contrast, there was no notable change in the AIx in the Ab-BP group, which presented a less effective adaptive response; CO remained the same, and PWV increased significantly. The significant HR decrease failed to lower CO, indicating poorer circulation adaptation. This is evidenced by the unchanged TVR and a significant rise in PWV after water consumption. The stable AIx post-water intake suggests a lower level of hemodynamic adjustment to the timing of the reflected wave.

Our study revealed a range of important insights into cardiovascular function in response to water overload, though it has some limitations that warrant consideration. Firstly, we did not include female participants, and the cohort size is modest. Additionally, while our findings suggest a shift in autonomic balance, interpreting the LF component can be complex, as it does not solely reflect sympathetic activity. The LF component is influenced by both sympathetic and parasympathetic stimuli, so caution should be exercised when using LF/HF as a definitive measure of autonomic balance. Furthermore, we did not measure baseline intracellular and extracellular fluid volumes, which could have influenced the results.

Conversely, this study offers a pioneering comparative analysis of water intake and its physiological effects. By revealing significant effects, our study highlights the potential benefits of water intake on BP control and vascular hemodynamics. These findings address a crucial gap in research on hydration's role in CV health and advance our understanding of the body's complex responses to hydration. We emphasize the importance of a personalized approach to water intake, recognizing that the ideal volume varies among individuals based on renal function and hydration status, which influences urine production.

Declarations

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