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Heart Rate Variability, Arterial Function and Reflected Waves in Healthy and Young Participants – Studying the Physiological Effect of Cold Water Ingestion

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ABSTRACT

Introduction: *The physiological consequences of drinking cold water have been debated, but are yet to be fully understood. Previous research has provided conflicting results regarding the modulation of the autonomic balance in response to cold water intake. Thus, we aimed at assessing the autonomic and hemodynamic changes in response to cold water intake in healthy young adults.*

Methods: *Twenty-four healthy and young volunteers (12 female), with a mean age of 20.46±1.06 years, were enrolled in a cross-sectional study. Two experimental sessions, one week apart, were randomly implemented to evaluate the effect of warm (control) and cold (intervention) water. In both sessions, heart rate variability (HRV) and hemodynamic analysis via carotid pulse wave analysis were performed, at baseline and immediately after the ingestion of 250 ml of either room temperature water (22ºC) or low temperature water (3°C).*

Results: *Frequency and time HRV parameters were considered for analysis, and significant changes were depicted in the cold water experimental condition, with an increase in the HF frequency component, on the RMSSD and pNN50 statistical parameters and on the triangular index, followed by a decrease in the nLF component. A shift in the sympathetic-vagal balance was thus identified through a decrease in the nLF/nHF ratio, highlighting a vagal activation following the cold water ingestion. A significant heart rate decrease was also observed as expected from the vagal activation in the intervention session, although the reduction was quite discrete in magnitude (heart rate mean difference: -3.04±3.94 bpm; p=.001). No significant hemodynamic changes were observed, although a trend for a reduction in the augmentation index (AiX) was observed following the cold water stimulation, suggestion an effect over the vascular tone, modifying the timing of the reflected waves, and explaining the significant decrease in aortic pulse pressure observed in males.*

Conclusions: *Cold water intake modulates the sympathetic-vagal balance, inducing an increase in the parasympathetic activity and producing mild and clinically irrelevant chronotropic and hemodynamic changes.*

Keywords: *Autonomic nervous system; cold water; heart rate variability; reflected waves*

INTRODUCTION

Drinking water is one of the essential daily activities and has been studied due to its hemodynamic and autonomic effects, which appear to have clinical relevance. The medical literature effectively suggests that somehow cold water intake affects heart function as evidenced by changes in the T wave on the electrocardiogram, heart rate (HR) and blood pressure (BP). [1-3] There are several factors that are cited as possible causes for these changes, such as autonomic response, vagus nerve stimulation, cooling of the esophagus and oral cavity, and direct effects of gastric distension, among others. [2,3]

Some effects of water ingestion have been described, for example, in a curious documented clinical case that references a sudden increase of the PR interval, from 0.16ms to 0.32ms, plus an increase in BP and HR, twenty seconds after the ingestion of low temperature water. The patient in the case had ischaemic heart disease in which the prolonged AV block induced by cold water was reversed with atropine, thus suggesting a mechanism mediated by the parasympathetic nervous system. [4]

In the past, guidelines did not recommend feeding hospitalized patients after acute myocardial infarction with liquids at extreme temperatures, whether hot or cold, due to its hypothetical effects on HR, BP, or even on the heart rhythm. However, a past study demonstrated no significant changes in any of the parameters mentioned above, aside a discrete chronotropic impact in some individuals. [5] Therefore, a physiological relationship between cold water intake and the autonomic modulation of the cardiovascular system is plausible in such conditions and in some people, although discrete in its magnitude. The existing confliting evidences thus provide an opportunity to experimentally test the hypothesis of the existence of an autonomic effect of cold water ingestion, approaching the effect on HR and arterial hemodynamics of healthy participants. The study of HR modulation through heart rate variability analysis (HRV) provides a consistent method to study eventual autonomic nervous system changes following such stimulation. There are several factors that have been found to modulate the HRV components, ranging from the renin-angiotensin-aldosterone system to the thermoregulatory system and the sympathetic and parasympathetic nervous system. The frequency domain analysis further provides the means to decompose the fundamental oscillatory components of HRV and understand how they change with the cold water stimuli. [6,7] Furthermore, the influence over vascular function and underlying hemodynamic profile, is also of major interest as it would allow an aditional insight into the autonomic modulation of vascular tone following cold water intake. Of particular interest, the modulation of the reflected waves, as expressed by the augmentation index (AiX), by the cold water is yet to be described. The AiX is a measure of the contribution of the reflected waves to the overall pulse wave contour, providing an integrated indicator of the dynamic properties of the arterial wall, dependent on the distance traveled by the reflected pulse wave, the duration of left ventricle ejection and the amplitude of the reflected wave – owing to the geometry, number and vascular tone of small arteries and arterioles. [8]

METHODS

Population

Twenty-four clinically healthy and young participants were enrolled in a cross-sectional study aimed at identifying the modulation of the autonomic nervous system mediated by the ingestion of cold water, expressed in the changes in HRV and arterial function and reflected waves. All participants agreed to voluntarily participate in this study and gave their informed consent. The research protocol included two experimental sessions, in which the participants were evaluated before and after drinking a standard amount of water at two temperature conditions: room temperature and cold water. All the procedures were performed in an adequate and quiet laboratory, with controlled temperature and humidity conditions.

Heart Rate Variability

The participant was placed in supine position and standard ECG eletrodes with conductive gel were placed in the conventional 12 lead ECG positions. After a 10 minutes resting period, a standard resting 12 lead ECG was performed, followed by a 6 minute signal acquisition for the HRV study. The acquisition was performed with a PC-ECG 1200 equipment (Norav Medical, Germany), with a sampling rate of 500 Hz, a low pass filter of 20 Hz, and a notch filter of 50 Hz. The fiducial point of the R wave was identified by an algorithm of parabolic interpolation and a derivative plus threshold algorithm to locate a stable and noise-independent reference point. A quality check was performed for all the HRV acquisitions, and acquisitions with premature beats or artifacts were rejected. If the percentage of deletion was more than 5%, then the subject was excluded from the study. The HRV parameters were then extracted for further analysis, particularly: a) in the time domain: the NNcount (number of consecutive normal sinus (NN) beats in the active interval), max NN and min NN (respectively, longest and shortest NN intervals), Average NN (average of NN intervals), SDNN (standard deviation of all NN intervals), RMSSD (the square root of the mean of the sum of the squares of differences between adjacent NN intervals), HRV triangular Index (total number of all NN intervals divided by the height of the histogram of all NN intervals measured on a discrete scale), the NN50 (mean number of times in which the change in consecutive NN intervals exceeds 50 milliseconds) and the pNN50 (fraction of consecutive NN intervals that differ by more than 50 ms); b) in the frequency domain, after fast Fourier transformation: ULF (Power of the ultra low frequency range – 0.0001 and 0.003),

VLF (Power of the very low frequency range – 0.003 and 0.04), LF (Power of the low frequency range - 0.04 and 0.15 Hz), HF (Power of the high frequency range - 0.15 to 0.4 Hz) and the LF/HF ratio (reflects the absolute and relative changes between the sympathetic and parasympathetic components of the ANS, typifying the sympathetic-vagal balance on the heart). Spectral analysis normalization was also performed to minimize the effects of changes in the VLF. Thus, for the HF and LF normalization, the ratio between both the LF and HF components was made by the total power spectrum minus the VLF component and multiplied by 100. [6] All measured and reported parameters are according to the ESC/NASPE guidelines. [6]

Carotid Pressure Wave Analysis and Brachial Blood Pressure Measurement

The analysis of the carotid artery wave pressure was performed with the use of the Complior® system (ALAM, Paris), with the participant in supine position and after a 10 minutes resting period. Brachial blood pressure (bBP) was firstly obtained in the right arm using an automatic and clinically validated device (Riester ri-champion® N; Riester, India). The bBP was measured with an appropriate sized cuff positioned at the heart level about 2 to 3 cm above the antecubital fossa. Three consecutive measurements, with 2 minute intervals, were performed and the average of the three measurements was considered for analysis. Systolic blood pressure (bSBP) and diastolic blood pressure (bDBP) were obtained. Mean arterial blood pressure (bMAP) and pulse pressure (bPP) were calculated according to the formulas [bDBP+(bSBP-bDBP)/3] and [bSBP-bDBP], respectively.

Afterwards, a piezoelectric sensor of the Complior device was positioned over the right common carotid artery, at the point of maximum signal amplitude, stability and detail. After the stabilization of the signal, the recording of pressure curves during a time frame of 15 seconds ensued. Central BP (cBP) and AiX were than extracted for analysis. The AiX expresses the percentage of the central PP (cPP) corresponding to the augmentation pressure (AP), this being the difference between the second and first systolic peak of the pulse wave. Carotid pulse waves were calibrated with each participant's reference brachial MAP and DBP. Measurements were blindly performed by the same operator, and the quality of the recordings was evaluated by two independent and blind observers, with considerable experience regarding the method.

Procedure

All the included participants were informed of the procedure, and gave their written informed consent to participate. The experimental design comprised two independent sessions (one week apart): a control session - ingestion of 250 ml of room-temperature water (22ºC); an intervention session - ingestion of 250 ml low temperature water (3ºC). In order to eliminate the water volume as a potential confounder, or source of bias, a small amount of water was ingested by the participants (250ml) in order to have the least possible influence of the intragastric pressure on the sympathetic-vagal balance.[10] The order of the sessions was randomized, keeping the same overall structure of the experimental sessions. Relevant demographic and clinical data were collected at the first session. Height and weight were measured in a calibrated scale, and the BMI was calculated as weight (Kg)/ height^2(m^2). Waist circumference and neck's perimeter were measured with an appropriate measuring tape. All evaluations were performed in the morning, in a laboratory with appropriate conditions and controlled humidity, temperature, light and sound. The participants were instructed to refrain from smoking, eating and drinking at least 5 hours prior to the experimental sessions. Baseline HRV and hemodynamic analysis were performed after a resting period in supine position and immediately (about 2 minutes) after the water intake. Changes from baseline to post-water intake were compared in both experimental sessions.

Statistical Analysis

Statistical analysis was performed with the SPSS for Windows, version 22.0 (SPSS package, IBM, USA). The distribution of the variables was tested for normality using the Shapiro-Wilks test and for homogeneity of variance by the Levene's test. A simple descriptive statistic was used to characterize the study population and to assess the distribution of the variables.The comparisons between groups were made through the $\chi 2$ test, for categorical variables, or the Student's *t* test for independent

samples, for quantitative variables. For the within-subject comparisons, we used the pairwise Student's *t* test, or the repeated measures ANOVA, as best suited. For the repeated measures ANOVA, the Greenhouse-Geisser correction for the degrees of freedom was adopted whenever sphericity violation was verified. All multiple comparisons meant to localize the significant effects of a factor were based on the Bonferroni correction. The criterion of statistical significance used was $p \leq 0.05$ for a confidence interval of 95%.

RESULTS

Twenty-four subjects were recruited for this study, 12 males and 12 females, with a mean age of 20.46±1.06 years. All of them were clinically healthy. Mean body mass index (BMI) was 23.01±3.77 kg/m^2. Smoking habits were identified in 9 participants and 16 participants referred a family history of cardiovascular diseases. No differences were observed in terms of age, BMI, and overall clinical profile in regards to gender, with the exception of bBP, which was higher in males (cf.Table 1).

Table1. *Overall characteristics of the study population.*

	Total (n=24)	Female (n=12)	Male (n=12)	p
Age, years	20.46±1.06	20.58±0.90	20.33±1.23	.576
BMI, Kg/m^2	23.01±3.77	22.56±3.93	23.46±3.72	.572
Tobacco Yes, n(%) No, n(%)	9 (37.5) 15 (62.5)	3 (25) 9 (75)	6 (50) 6 (50)	.400
Family history of CVD Yes, n(%) No, n(%)	16 (66.7) 8 (33.3)	10 (83.3) 2 (16.7)	6 (50.0) 6 (50.0)	.193
bSBP, mmHg	118.08±13.56	113.75±15.69	122.42±9.87	.120
bDBP, mmHg	68.46±8.37	69.17±11.12	67.75±4.65	.688
bMAP, mmHg	73.33±5.10	60.92±2.97	85.75±4.69	.012
bPP, mmHg	49.63±11.15	44.58±9.84	54.67±10.38	.023
HR, bpm	69.00±8.51	67.75±8.63	70.25±8.58	.484

BMI: body mass index; CVD: cardiovascular diseases; bSBP: brachial systolic blood pressure; bDBP: brachial diastolic blood pressure; bMAP: brachial mean blood pressure; bPP: brachial pulse pressure; HR: heart rate

The impact of water ingestion on HRV is represented in table 2, in both experimental conditions – warm water and cold water. No significant changes were seen regarding the warm water ingestion experimental condition. However, statistical significant variations were depicted in the cold water experimental condition, both in the time and the frequency domain. In the frequency domain, a significant increase was depicted in the HF component (mean difference: 30.54±53.60; p=.013) and the nHF (mean difference: 4.72±9.23; p=.020), and a contrariwise decrease was observed in the nLF (mean difference: -4.72±9.23; p=.023), thus explaining a significant decrease in the nLF/nHF from 1.26±1.01 at baseline to 1.17±0.98 post cold water ingestion (mean difference: -0.31±0.60; p=.023). In the time domain HRV analysis, following cold water ingestion a significant increase was observed in the RMSSD (p=.038), the triangular index (p<.001) and the pNN50 (p=.003), accompanied by a significant decrease in HR (baseline: 73.92±11.05 bpm; post-cold water: 70.17±10.37 bpm; p=.001). Taking all together, a reinforcement was identified in the vagus-related HRV parameters following the stimulation with cold water, and an opposite reduction in the nLF parameter further strengthens the indication of o modulation of the sympathetic-vagal balance in favour of a vagal predominance.

The hemodynamic changes are represented in table 3. No significant differences were observed regarding the variation in all the considered hemodynamic parameters in both experimental conditions, even though a curious trend was observed regarding the AiX parameter with an opposite direction of change in the two conditions, becoming more negative in the cold water condition (mean difference: -2.81±12.54) and invariant in the warm water condition (mean difference: -0.01±1.32). For brachial and central BP, no significant changes were observed.

Table 2. *Variation of the HRV parameters before and after the ingestion of water in the control (left panel) and intervention (right panel) experimental conditions.*

	Control session – warm water				Intervention session – cold water			
	Before	**After**	**Dif (CI)**	**p**	**Before**	**After**	**Dif (CI)**	**p**
LF, ms^2	173±82	170±73	-3.15±66.15 (--31.09;24.78)	.817	172±72	165±66	-6.36±45.10 (-25.86;13.15)	.506
HF, ms^2	190±99	184±95	-5.54±61.47 (-31.50;20.42)	.663	163±69	193±83	30.54±53.60 (-39.54;-1.98)	**.013**
nLF/nHF	1.40±1.30	1.40±1.19	-0.01±1.10 (-0.47;0.46)	.995	1.26±1.01	1.17±0.98	-0.31±0.60 (-0.57;-0.05)	**.023**
nLF, n.u.	0.49±0.20	0.49±0.20	-0.01±0.12 (-0.04;0.06)	.757	0.47±0.17	0.49±0.17	-4.72±9.23 (-8.72-0.73)	**.023**
nHF, n.u.	0.51±0.20	0.51±0.20	-0.01±0.12 (-0.06;0.04)	.757	0.53±0.17	0.51±0.17	4.72±9.23 (0.73;8.72)	**.020**
RMSSD, ms	77.35±39.39	81.97±47.31	4.61±17.10 (-2.61;11.83)	.200	77.86±34.70	89.86±10.67	12.24±26.63 (0.72;23.75)	**.038**
SDNN, ms	80±26	82±30	2.10±17.00 (-5.08;9.28)	.552	80±27.74	84±29.16	3.71±15.46 (-2.98;10.39)	.262
Triangular Index	21.00±4.84	22.50±6.41	1.50±4.06 (-0.21;3.22)	.120	21.19±6.21	24.48±7.25	3.30±3.48 (1.79;4.80)	**<.001**
NN50, count	73.29±35.87	74.12±34.77	0.83±14.39 (-5.24;6.91)	.779	73.52±34.18	75.57±31.93	2.04±15.17 (-4.52;8.60)	.525
pNN50, count	19.47±10.50	20.18±10.30	0.71±3.39 (-5.24;6.91)	.313	19.22±9.22	21.58±9.36	2.36±3.33 (0.92;3.80)	**.003**
Heart Rate, bpm	69.00±8.51	68.58±9.55	-0.42±7.28 (-3.49;2.66)	.782	73.92±11.05	70.17±10.37	-3.04±3.94 (-4.71;-1.38)	**.001**

SDNN: Standard deviation of the mean of the NN intervals; LF: low frequency; HF: high frequency; LF/HF: ratio of the low frequency power to the high frequency power; nLF: normalized low frequency; nHF: normalized high frequency;RMSSD: square root of the mean of the sum of the squares of differences between adjacent NN intervals;NN50: mean number of times in which the change in consecutive NN intervals exceeds 50; pNN50: fraction of consecutive NN intervals that differ by more than 50 ms; n.u.: normalized units

Table3. *Variation of the hemodinamic parameters before and after the ingestion of water in the control (left panel) and intervention (right panel) experimental conditions.*

	Control session – warm water				Intervention session – warm water			
	Before	**After**	**Difference (95 CI)**	**p**	**Before**	**After**	**Difference (95 CI)**	**p**
bSBP, mmHg	118.08±13.56	117.58±10.59	-0.50±10.07 (-4.75;3.75)	.810	110.29±12.95	110.29±11.45	0.00±10.10 (-4.26;4.26)	1.000
bDBP, mmHg	68.46±8.37	68.04±7.30	-0.42±4.17 (-2.18;1.34)	.629	65.08±7.89	65.17±7.00	0.08±6.95 (-2.85;3.02)	.954
bMBP, mmHg	73.33±25.10	80.67±7.30	7.33±24.66 (-3.08;17.75)	.159	76.04±15.57	80.13±7.79	4.08±18.22 (-3.61;11.78)	.284
bPP, mmHg	49.63±11.15	49.54±10.88	-0.08±9.05 (-3.90;3.74)	.964	45.21±9.02	45.13±8.07	-0.08±7.77 (-3.36;3.20)	.959
cSBP, mmHg	112.71±14.49	111.75±10.40	-0.96±10.64 (-5.45;3.54)	.663	106.29±14.01	105.08±11.91	-1.21±10.82 (-5.68;3.35)	.589
cPP, mmHg	44.25±12.03	43.71±9.35	-0.54±9.84 (-4.70;3.61)	.790	41.21±10.90	39.92±8.69	-1.29±9.02 (-5.10;2.52)	.490
AiX, %	-18.14±5.55	-18.15±5.92	-0.01±1.32 (-1.86;1.70)	.973	-24.79±17.53	-27.59±18.94	-2.81±12.54 (-8.10;2.49)	.284

bSBP: brachial systolic blood pressure; bDBP: brachial diastolic blood pressure; bMAP: brachial mean arterial pressure; bPP: brachial pulse pressure; cSBP: central systolic blood pressure; cPP: central pulse pressure; AiX: augmentation index.

Stratifying the effects of cold water by gender, a similar effect was depicted for males and females, with an increase in the HRV measures related to the vagus activity, mainly, an increase

in the nHF, RMSSD, pNN50 and the HRV triangular index, and a decrease in the measures of autonomic sympathetic activity, particularly the nLF parameter. Consequently, the nLF/HF and the HR decreased in both genders. Curiously, a trend for a decrease in the AiX was also observed in the cold water condition in both genders, adding to a particular significant effect over the cPP observed in the males (cPP mean reduction: -5.42±8.79 mmHg; CI: -10.99;0.01; p=.05).

Discussion and Conclusions

The effect of water ingestion at extreme temperatures (either cold or hot) has been a matter of debate, and to date, conflicting evidence exists, either demonstrating important cardiovascular changes mediated by autonomic stimulation, or showing no significant effects. [4,5] Considering the main findings in our study, a clear and sound modulatory effect of cold water over the autonomic sympathetic-vagal balance was observed, even though no significant hemodynamic changes were concorrently observed rather than a trend for a decrease in the AiX component, and a significant reduction in cPP in males. Changes in both spectral and time domain HRV analysis were identified, particularly over the HF (increase) and the LF (decrease) frequency components, and the RMSD, triangular index and the pNN50 statistical components (all increasing). These combinations of findings observed only after cold water ingestion, imply a modulation of the vagal tonus, possibly mediated by the thermosensitive activation of afferent pathways of the esophagus, stomach, duodenum and oral cavity. [1] A further reinforcement to this vagal modulation comes from the observation of a significant reduction in the nLF/nHF ratio, explained mainly by the increase in the HF component, which is the frequency domain component associated with the parasympathetic *at tempus* action, [6] previously found to increase following cold water ingestion. [8,9] The observed nLF/nHF change clearly moves the sympathetic-vagal balance in favour of an increase in the vagal influence, which also explains the significant, but rather modest (about 3 bpm) reduction in HR observed after the cold water ingestion. Thus, even though a significant shift in the autonomic balance occurred with the cold water ingestion, its physiologic manifestations were quite discrete in magnitude, and therefore failed to produce sharp variations in terms of heart rhythm and hemodynamic profile. In fact, from the hemodynamic point of view, there were no changes regardless of water intake temperature, concurring with all thermal impact evaluation studies. Although not significant, but curious nevertheless, the AiX demonstrated opposite trends, with a trend for a decrease after the ingestion of cold water, and an invariance after the ingestion of water at room temperature. The trend towards a reduction in the AiX following the cold water intake could translate into a later arrival of the reflected wave into the proxima part of the aorta as a possible consequence of a concurrent discrete decrease in peripheral resistance due to arterial vasodilation, explaining the significant reductnion in cPP observed in males (and a similar trend in females, although non-significant), supporting to some extent a thermodependent hemodynamic modulation of the sympathetic-vagal balance.

Regardless the magnitude of the effect, our results favor the view that cold water intake involves some degree of modulation of the sympathetic-vagal balance, to a more parasympathetic predominance, as a consequence of several possible mechanisms, of which the thermosensitive activation of the nerve fibers predisposed all the way through the gastrointestinal is particularly favoured by our findings and previous research. [1,8-14]

Some limitations advise caution when interpreting the results, mainly the small population and the particular demographic characteristics, being a quite young and healthy population. Furthermore, the influence of several chronobiological features were also not controlled, such as the stage of the menstrual cycle in females, which has an important role over the dynamic properties of the peripheral vasculature and endothelial function. [11]

In conclusion, cold water intake modulates the sympathetic-vagal balance, inducing an increase in the parasympathetic activity, although this modulation is unlikely to produce major or clinically relevant chronotropic or hemodynamic changes. Further studies are needed to better understand the influence of cold water intake on other populations and to provide a clear physiological explanation for the mechanisms underlying this thermodependent autonomic arousal.

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