



Meditation makes me sick: Meditation and sensitivity to parasympathetic nervous system stimulation

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INTRODUCTION

- ☐ Meditations are in medical research literature approached as relaxation techniques with beneficial health effects (stress reduction, improvement of cardiovascular system) [1]. However, meditation and health research suffers from many theoretical and methodological shortcomings [2, 3] supporting only positive effects of meditations. Negative effects thus have been fairly omitted in a scientific literature.
- ☐ Meditation, in general, stimulates parasympathetic nervous system and should lead to relaxed states. Yet in our previous study aimed every 6th participant reported during meditation head spinning, nausea, extreme sweating, over warming and faintness. In this project we focused on these effects of meditation practice.

HYPOTHESES

- ☐ H1: There will be a significant increase in parasympathetic autonomous nervous activity and decrease or no change in sympathetic autonomous nervous activity during meditation (Phase 2) compared to the time before meditation (Phase 1).
 - ☐ Activity of parasympathetic nervous system is detectable in increase of normalized power in the high frequency range (HF normalized) and decrease or no change of normalized power in low frequency range (LF normalized) of a heart rate [4, 5].
- ☐ H2: Parasympathetic activity (HF normalized) during meditation (Phase 2) will be significantly increased in participants scoring high on the Nausea Profile compared to other participants.

PARTICIPANTS & METHODS

- ☐ Participants: 57 university students (42 females and 15 males, mean age = 23). Participants were divided according to their Nausea Profile score into the three groups (19 in each).
- ☐ Within-subject experimental design consisted of three phases: rest phase before meditation (Phase 1), meditation (Phase 2) and after meditation (Phase 3).
- ☐ Treatment: Meditation practice called Anápána sati aimed at breath was operationalized as counting breaths while having eyes closed.
- ☐ Questionnaire: The Nausea Profile [7].

PROCEDURE

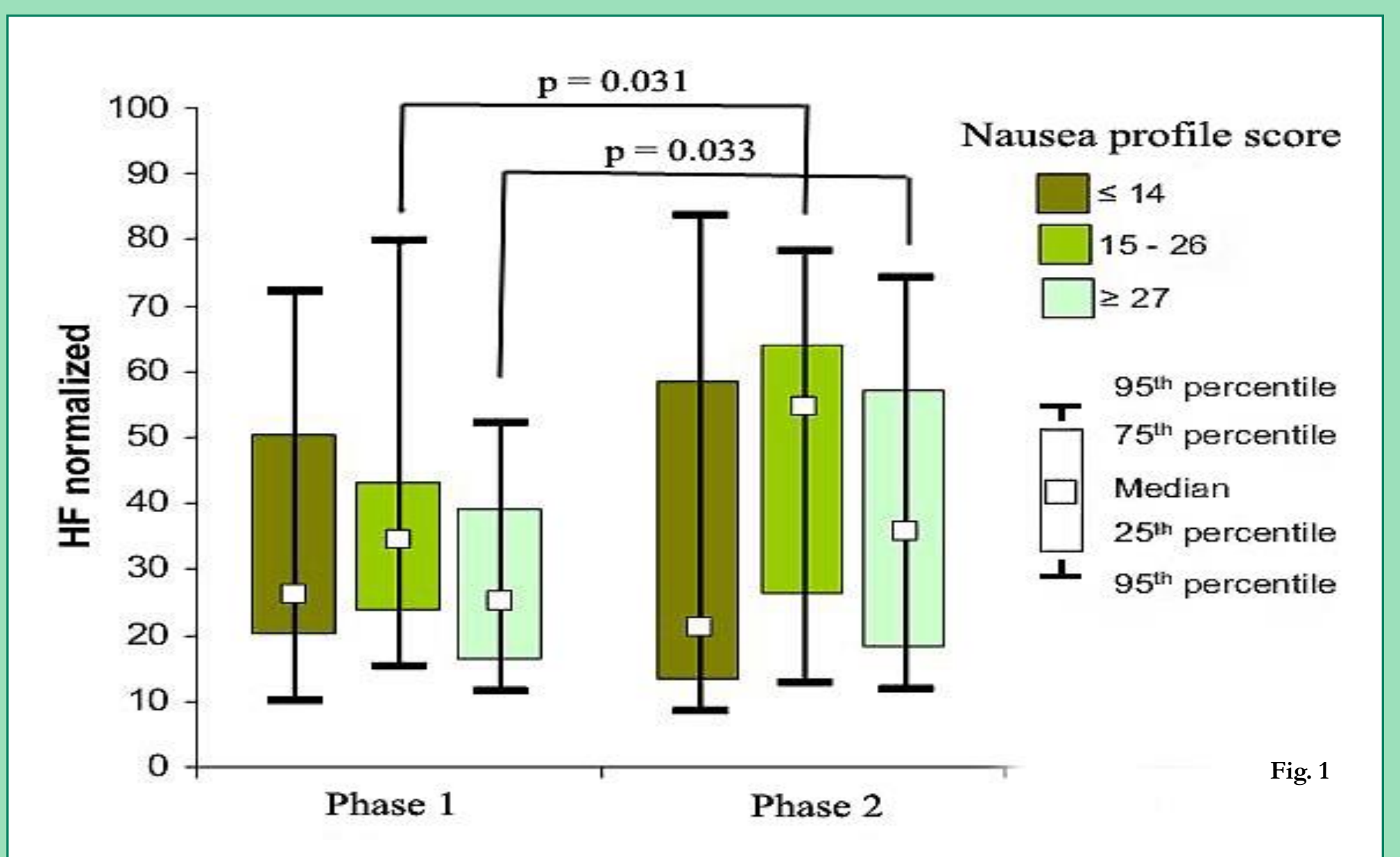
- ☐ Participant received three-lead Holter EKG (MARS GE Medical) and noninvasive hemodynamic continual monitoring of blood pressure CNAP 500 HD and went through three phases. In post meditation phase participant filled in the Nausea Profile and an open questionnaire for subjective report.
- ☐ Deceleration capacity (DC) [6] of heart rate (HR) was determined as the indicator of a parasympathetic nervous activity. For comparison of parasympathetic and sympathetic nervous activity were stated following parameters [5]: heart rate variability (HRV), normalized power in the low frequency range (LF normalized), normalized power in the high frequency range (HF normalized) with efferent vagal activity as a major contribution and LF/HF ratio as a marker of sympathetic vagal balance.

STATISTICAL ANALYSES

- ☐ For comparison of dependent within-subject data was used Wilcoxon paired test.
- ☐ For comparison of independent between-subjects data was used Kruskal-Wallis test.

RESULTS

- ☐ DC significantly increased ($p < .001$) in both males ($p = .013$) and females ($p = .001$) during meditation (Phase 2). Normalized HF significantly increased and normalized LF significantly decreased during meditation (Phase 2) in all participants ($p = .008$) (see Tab. 1).
- ☐ HF normalized significantly increased in Phase 2 in comparison to the Phase 1, but only in participants who scored medium (15–26) (from mdn = 34.6 (15.3; 79.7) to mdn = 55 (13; 78), difference mdn = -12.1 (-44.3; 26.8), $p = .031$) and high (≥ 27) (from mdn = 25.2 (11.5; 52.1) to mdn = 35.6 (11.8; 74.2), difference mdn = -5.41 (-52.58; 23.21), $p = .033$) in the Nausea Profile (see Fig. 1).
- ☐ Meditation increased parasympathetic nervous activity in all participants. In some individuals rapid activation of a parasympathetic nervous system during meditation led to nauseous feelings.



DISCUSSION

- ☐ Both hypotheses were tentatively supported in analyses. Remaining issues:
 - ☐ Unexpectedly some measured heart activity parameters didn't correspond with each other appropriately.
 - ☐ Continuous blood pressure change measures are expected to reveal more and are being analyzed in these days.

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	All participants N = 57 Median (5 th ; 95 th percentile)	Sex		p1
		Females N = 42 Median (5 th ; 95 th percentile)	Males N = 15 Median (5 th ; 95 th percentile)	
DC				
Phase 1	10.3(6.7;18.2)	9.9(6.69;18.15)	11.1(7.5;17.7)	0,578
Phase 2	12.3(7.3;30.1)	12.0(7.7;28.7)	15.7(10.0;34.9)	0,365
Phase 3	9.58(5.91;15.03)	9.08(5.91;13.65)	11.5(6.8;17.8)	0,013
Phase 1 - Phase 2 diff.	-1.48(-17.19;2.03)	-1.38(-16.54;1.92)	-4.20(-17.19;5.15)	0,056
Phase 2 - Phase 3 diff.	3.15(-3.42;21.02)	3.15(-1.52;18.60)	4.08(-6.32;26.60)	0,835
p2	<0.001	0,001	0,013	
p3	<0.001	<0.001	0,035	
LF normalized				
Phase 1	69(32;89)	69(34;89)	71(20;80)	0,905
Phase 2	61(25;90)	63(27;89)	59(22;90)	0,587
Phase 3	74(38;89)	72(38;88)	78(56;91)	0,354
Phase 1 - Phase 2 diff.	4.94(-23.21;43.26)	4.32(-23.21;37.40)	12.0(-18.6;52.6)	0,566
Phase 2 - Phase 3 diff.	-8.82(-44.97;20.07)	-6.37(-39.03;20.07)	-23.8(-65.4;15.3)	0,130
p2	0,008	0,024	0,116	
p3	<0.001	0,007	0,011	
HF normalized				
Phase 1	30.6(11.5;67.5)	30.9(11.5;65.5)	28.6(19.6;79.7)	0,905
Phase 2	39.0(10.4;74.9)	37.3(11.0;72.7)	41.2(10.4;78.4)	0,587
Phase 3	26.2(11.2;62.0)	27.5(12.2;62.0)	22.3(8.4;43.9)	0,354
Phase 1 - Phase 2 diff.	-4.94(-43.26;23.21)	-4.32(-37.40;23.21)	-12.0(-52.6;18.6)	0,566
Phase 2 - Phase 3 diff.	8.82(-20.07;44.97)	6.37(-20.07;39.03)	23.8(-15.3;65.4)	0,130
p2	0,008	0,024	0,116	
p3	<0.001	0,007	0,011	

Tab.1

p1 is statistical significance of difference between groups evaluated by the Kruskal-Wallis test.

p2 is statistical significance of difference within the same individuals in Phase 1 and Phase 2 evaluated by Wilcoxon paired test.

p3 is statistical significance of difference within the same individuals in Phase 2 and Phase 3 evaluated by Wilcoxon paired test.