Contemplations into respiration: effects of breathing and meditative movement on body and mind

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Chapter 4

Breath of life: the acute effects of respiratory locus and rate on autonomic activity and inhibitory control

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Abstract

Meditation practices are an increasingly popular way to decrease stress levels and enhance cognitive functioning. In order to explain the accumulating corroborative evidence of these claims, the respiratory vagal nerve stimulation model of contemplative practices (rVNS) was introduced (Gerritsen & Band, 2018). This model provides mechanisms by which positive effects on stress and cognition, can be explained by changes in respiratory patterns in its practitioners, both in the short and long term. This chapter contains the first attempt at testing the predictions of the rVNS model, namely: that changing the locus of breathing from the thorax to the abdomen and bringing respiration rate down, will acutely increase vagal tone heart rate variability (HRV), decrease sympathetic tone, and thus decrease stress. Furthermore, that this will also lead to increases in mental flexibility, by increasing executive functioning, specifically inhibition. Two experiments were run to test these predictions in a pre-test post-test within-subjects controlled design (n=29; n=34). The manipulation of respiratory parameters was done by guided breathing exercises in audio format. None of the predictions of the rVNS model came to pass. From the Bayesian analysis it was concluded that both experimental samples were underpowered. An extensive discussion on these findings, that identifies further factors that might have contributed to these null-results, is included.

1. Introduction

Respiration plays a central and critical function in complex organisms. It serves as a pillar of homeostasis, in other words: being able to live. By its essence, respiration — by transporting oxygen to organs and muscles — is absolutely necessary for any organism to function properly, dynamically and optimally. Respiratory processes themselves are highly complex and even today the neural mechanisms and substrates remain unresolved (Ashhad et al., 2022; Feldman & Del Negro, 2006). Despite the central position of respiration in homeostasis and the popularity of bottom-up explanations of complex behavior (e.g. embodied cognition, ideomotor theory), studies of its role in emotion and cognition have been lacking. A field of study that lends itself well to look for potential respiratory effects is that of contemplative practices. Contemplative practices, like mindfulness meditation, employ a variety of breathing techniques that serve as a manipulation of respiratory parameters and have shown to impose a wide range of beneficial effects on mental health (Schlechta Portella et al., 2021; Smart et al., 2022) and cognition, particularly on attentional control and executive functions (Casedas et al., 2020; Verhaeghen, 2021). Even short-term mindfulness training seems to benefit executive functions, notably inhibition and updating, although reported results are mixed (Zhou et al., 2020). However, the potential mediating or moderating effects of respiratory control on these dependents have been understudied. To fill this gap and the lack of focus on respiration in general, Gerritsen and Band (2018) provided a framework: the respiratory vagal nerve stimulation (rVNS) model of contemplative activities. According to this model respiratory patterns stimulate the autonomic nervous system, notably the vagal nerve complex, to a relaxed and flexible state. Through this state, cognition becomes more

adaptive to current demands, and this can lead to long-term improvements. This study was set up to test some of the rVNS model's predictions.

1.1. rVNS model

The primary claim of the rVNS model is that the slow breathing exercises inherent in many contemplative tradition can directly lead to many of the reported benefits, by phasically and tonically stimulating the parasympathetic branch of the autonomous nervous system. Hereby, the system shifts away from an arousal dominated stance towards a more relaxed one. This ameliorates stress-related pathology and enhances cognition in overly demanding contexts. Where normally the system would be taxed (stressed) beyond optimal settings, it can now remain inside these bounds. In our model, there are three respiratory parameters that influence parasympathetic tone, or rather autonomic state, in order of evidential support: 1) rate: slowing breathing down increases relaxation, with an optimal rate of 6 bpm on average (Laborde et al., 2017; You et al., 2021); 2) ratio: extending exhalation relative to inhalation relaxes (Cregg et al., 2017;; van Diest et al., 2014), for example: due to respiratory sinus arrythmia (Hirsch & Bishop, 1981); 3) locus: breathing by moving the abdomen relative to the thorax moves the autonomic state from fight/flight to rest/digest (Amann, 2012), as thoracic respiratory musculature is both inspiratory and expiratory, but abdominal musculature is only expiratory (Aliverti, 2016). Though note that automatic changes in rate and ratio might be confounding factors here.

The rVNS model proposes the vagal nerve complex as the neural mediator between respiratory patterns and emotional/cognitive effects and adopts heart rate variability (HRV) as a valid indicator of vagal tone. The

association between HRV and cognition was first proposed by Porges (1995, 2001) and further elaborated upon in the neurovisceral integration model by Thayer and Lane (2000). In the neurovisceral integration model, cardiac flexibility, reflected in HRV, stimulates mental flexibility, notably cognitive inhibition, by projections of the vagal nerve towards (ventromedial) prefrontal cortex. This connection is also the hypothesized pathway through which slow breathing exercises lead to cognitive enhancement of executive functions in the rVNS model. However, it's important to note that vagal mediation is not necessary in our model – contrary to what is suggested in its name. In our view, specific respiratory patterns may lead to an optimal state on the arousal/relaxation spectrum that will benefit cognition in any specific context. This means that controlled hyperventilation could also boost executive functioning, if the context demands it; for example due to an individual having a high state of relaxation in an arousal demanding setting. For a detailed discussion of the rVNS model and (cardiac) vagal tone measurements we refer to the original work (Gerritsen & Band, 2018) and to a subsequent work by unaffiliated authors that posits a more detailed neurophysiological foundation (Noble & Hochman, 2019). Since the publication of the rVNS model, several studies have indirectly or directly attempted to test some of its premises.

1.2. Studies linking respiration, HRV, and cognition

Zaccaro and colleagues (2018) conducted a systematic review of actively controlled trials that employed slow breathing exercises and included HRV measures, neurophysiological measurements, psychological/behavioral tests, or combinations of these. Following slow breathing interventions, vagal tone HRV increased, while relaxation went up and anxiety down. Zaccaro et

al.'s main conclusion is that the evidence for a link between parasympathetic tone and favorable behavioral outcomes exists, but it is scarce. They also note that there are conflicting findings and that the operationalizations of vagal tone HRV are highly divergent. Lastly, the reviewed studies did not directly test for correlations between vagal tone HRV and behavioral/psychological outcomes.

Spangler and colleagues (2018) tested predictions of the neurovisceral integration model in ecologically valid low and high stress conditions among soldiers using a virtual shooter simulator. They reported that higher HRV variability was associated with less successful response inhibition (false alarms), contrary to findings that show enhanced inhibition in high HRV conditions. However, they argued that this not contrary to their expectations, because in high risk environments (life threatening), the cost of a miss is relatively higher, making downgrading inhibition the adaptive setting, and thus flexible response. Note however, that reaction times were not reported and therefore any potential speed-accuracy tradeoff was hidden. Additionally, if one follows the reasoning that a liberal signal detection threshold was adaptive in the high risk setting and adaptivity is reflected in HRV variability, we would not only expect more hits in this condition, but also a positive correlation between HRV variability and hit rate, which was not found. Nonetheless, in this study a relationship between HRV and (response) inhibition was (re)established.

Two other studies linking slow-paced breathing and cardiac vagal tone (RMSSD) need mention. The first tried to disentangle slow-paced breathing with and without HRV biofeedback (Laborde et al., 2022). They found no difference between these conditions on RMSSD (went up) and self-reported emotional valence (more negative), arousal (went down) or control

(went up), from pre-test to post-test. Clearly, slow-paced breathing on its own is sufficient to produce effects on cardiac vagal tone. The second studied the dose-response relationship of slow-paced breathing exercises with four different durations (You et al., 2021). They found no differences on RMSSD by dose, either during intervention or the resting state post-measurement. RMSSD did go up as compared to control during the intervention phase. Evidently, even short bouts of breathing exercises are sufficient to produce effects on cardiac vagal tone. Although it seems that these effects are short-lived, as the return to baseline is apparent across conditions.

Another study looked into the complete chain of prediction: respiration, vagal tone HRV and inhibition (Laborde et al., 2019). This study used physical exertion as a psychological stress inducer, had a slow breathing exercise either before or after exercise (6 breaths-per-minute), and included both an inhibition task (Stroop) and valid cardiac vagal tone marker (RMSSD). The results showed that Stroop interference scores (accuracy) were lower after slow breathing and RMSSD went up accordingly. Interestingly enough, the cognitive effects were not mediated by RMSSD, which casts doubt on the cardiac vagal complex mechanism. Again note that this does not rule out relaxation as potential candidate, by other mediators.

Bonomini and colleagues (2020) looked at two other executive functioning subtypes: shifting and updating (Miyake et al., 2000). In two experiments, using a 2-back and task switching task, they tested whether a slow breathing exercise (<6 breaths-per-minute) – contrasted with two other different breathing exercises – changed autonomic state and through this influenced executive functioning. The authors report an increase of low-frequency HRV and increased general success rate in both tasks after slow breathing. No effects were found on reaction time in both tasks or on switch

costs in the task switching task. Clearly, this is not convincing evidence for increased executive functioning by slow breathing, nor can low-frequency HRV be interpreted as a single marker of cardiac vagal tone (Laborde et al., 2017), arguing against involvement of the vagal complex. Furthermore, it's important to note that this study was underpowered and of weak/unclear design. For example: the duration of the breathing exercise was not reported.

To our knowledge there are no (recent) studies looking at either the ratio of inhalation/exhalation or locus, and their associations with cognition and/or autonomous state. However, there is a growing body of research on divergent effects on cognition of respiration phase (inhalation vs exhalation), for example: on memory processes (Heck et al., 2019) and perception and production of quantities (Belli et al., 2021). Studies that report on locus, by referring to abdominal breathing, only include instructions towards these incorporated in a slow breathing condition, and are not contrasted with a thoracic locus condition. In our first attempt at testing the predictions of the rVNS model, we therefore set out to investigate the influence of the locus factor on cardiac vagal tone and executive functioning of the inhibition subtype, while mapping the indirect influence of the two factors – rate and ratio – as well.

1.4. Current study

The study consisted of two experiments, both randomized controlled trials, of two different samples and design.

Experiment 1 employed a within-subject pre-test post-test design with three conditions in three sessions: abdominal breathing, thoracic breathing and focused breathing. The acute effects of short breathing

exercises, which manipulated locus of breathing, were studied on phasic parasympathetic tone (RMSSD) and phasic sympathetic tone (pre-ejection period, PEP), using ECG/ICG measures; and on response inhibition (stop-signal reaction time, SSRT) by stop-signal task (SST). Locus was manipulated through audio recordings of 3 different guided breathing exercises, including a tutorial. In abdominal breathing condition the participants were instructed to expand and retract their abdomen during inspiration and expiration respectively, engaging the diaphragm more; during thoracic breathing intervention the participants had to expand and retract the sides of the ribcage instead; and in focused breathing condition they only had to pay attention to the locations of their breathing and were instructed to not change anything (semi-control condition). Respiration parameters, respiration rate and inhalation/exhalation ratio, were checked by pneumography. The research question in Experiment 1 is: do short locus of breathing exercises (abdominal breathing, thoracic breathing, focused breathing) lead to distinct autonomic states, as reflected in RMSSD and PEP changes, that in turn influence the efficiency of response inhibition, as measured by SSRT?

Our expectations were that autonomic state would be parasympathetically dominant in the abdominal breathing condition and sympathetically dominant in the thoracic breathing condition. This should be reflected in a lower respiration rate, higher RMSSD and higher PEP (PEP score is inversely related to sympathetic tone) for abdominal breathing intervention and higher or unchanged respiration rate, lower RMSSD and lower PEP for thoracic breathing intervention. Furthermore, we hypothesized that these autonomous states transfer to the post-test and lead to an increase in response inhibition, as indicated by a lower SSRT, in abdominal breathing condition, relative to thoracic breathing condition, from pre-test to post-test. In focused breathing condition we expected these physiological and cognitive

parameters to remain relatively unaffected. However, our expectations of Experiment 1 were not met. Importantly, respiration rate went down during all three interventions, while RMSSD and SSRT were seemingly unaffected. Interestingly, PEP went down during abdominal breathing (from a much higher baseline), as compared to thoracic breathing and focused breathing, indicating an increase in sympathetic tone. These results did not allow for clear conclusions, especially by the decrease of respiration rate across conditions. To provide more clarity, the second experiment was set up.

In Experiment 2 the semi-control condition focused breathing was dropped, as an orthogonal design was deemed sufficient. The guided audio was rewritten and instructions for pacing of breathing were added. In the abdominal breathing intervention participants were instructed to breathe slower than normal, while in thoracic breathing it was stressed to breathe slightly faster than normal. In both recordings, the pacing of the instruction itself, matched the direction of the explicit instructions (i.e. slow pace of instruction to breathe slower and vice versa). Note that, with this design the unique contribution of locus can't be studied, as respiration rate changes are a supposed and likely confound. The physiological measurement for sympathetic tone was changed to electrodermal activity (EDA) and skin-conductance level (SCL) used as its marker, by reason of feasibility and ease of experimentation. A third physiological resting state measurement time was added after intervention to determine carry-over of autonomic changes (recovery phase). The Simon task replaced the SST, as there is debate on the reliability of SSRT (Wöstmann et al., 2013) and thus whether changes therein actually reflect enhancement effects. The Simon task was chosen as it is theorized to be the most pure measure of cognitive inhibition, by lack of stimulus-response overlap (Hommel, 2011). Importantly, the Simon task does not suffer from data exclusion issues as frequently observed in the SST.

Studies employing the SST frequently have a large proportion of the sample that does not comply with the instruction to not wait for the stop-signal. Another change to the cognitive testing was the inclusion of a pre-test for each session, instead of only one baseline on the first session. Finally, as a self-report measure of (psychological) relaxation/stress the affect grid was added. The research question of Experiment 2 is: do short locus of breathing (abdominal breathing, thoracic breathing) exercises lead to distinct autonomic states, as reflected in RMSSD and SCL changes, that in turn influence the efficiency of cognitive inhibition, as measured by the Simon task?

The expectations for Experiment 2 closely mirror those of Experiment 1. We expect respiration rate to go down in abdominal breathing condition and up in thoracic breathing condition, which in this context is a manipulation check. Furthermore, we expect RMSSD to go up and SCL to go down during abdominal breathing, with the reverse observed direction of RMSSD and SCL during thoracic breathing. Furthermore we expect that these levels are mostly maintained during recovery, thus do not immediately return to baseline. We expect the affect grid to show a more positive and relaxed affect state from pre- to post-test in abdominal breathing condition as compared to thoracic breathing condition pre- to post-test. Lastly, we hypothesize that this autonomic state persists into the cognitive post-test, resulting in a more or less flexible and efficient performance. Concretely: we expect a larger decrease in Simon interference effect from pre- to post-test in abdominal breathing condition, than in thoracic breathing condition, in both reaction time and accuracy.

2. Experiment 1

2.1. Method

2.1.1. Participants

Participants were recruited at the Faculty of Social Sciences at the University of Leiden by flyer or by online registration through a research participation system. Participation was restricted by the following selection criteria: no experience in meditation, other contemplative practices and/or breathing exercises, no neurological or cardiopulmonary disease; and no medication that affects cardiac or respiratory systems. Power analysis (G*power, 1-β=.8) based on the measure with the least iterations (SST, 4 measurements), suggested that 24 participants needed to be recruited. Note that, at this time planned statistical analysis was to be classical, instead of Bayesian. In the end, 29 participants were recruited: 23 females (M = 22 years, 18-34) and 6 males (M = 31 years, 21-58). There were no drop-outs. Participants were asked to abstain from strenuous exercise (same day), alcohol (24 hours), caffeine (12 hours) and nutrition (1 hour) before testing. Screening took place at the start of each session; no participant was excluded (on these grounds). These experiments have been approved by the ethics committee of the Leiden University institute of psychology (V1-1215 and V1-1337). Participants received either course credit or financial compensation for participation, the majority of which were (psychology) students.

2.1.2. Procedure

2.1.2.1. Design

Experimental sessions were held between 8:00 and 20:00 at a lab in the Faculty of Social Sciences in Leiden. Each participant completed three sessions, one session every week, each session corresponding to one of the three breathing interventions (abdominal breathing; thoracic breathing; focused breathing). The order of sessions was counter-balanced. Testing time between sessions was held constant (max. deviation: 1 hour). At the start of the first session of the experiment participants were informed on the experiment and provided informed consent. Then a short survey was used to screen on the selection criteria.

2.1.2.2. Physiological measures

At the start of a session participants were seated in the testing booth and equipped with ECG/ICG electrode leads (Kendall foam electrodes) and two respiratory bands (transducer belts). The ECG/ICG set-up consisted of the standard 11 electrode configuration (3-way ECG and 8-way ICG lead). For ECG, one electrode was placed just below the right collar bone, 4 cm to the right of the sternum; one on the right and one on the left side of the abdomen just under the ribcage. For ICG, four electrodes were placed at both sides of the neck 3-4 cm apart from each other within the vertical plane and four electrodes were placed at both sides of the trunk. Two at each side, with one approximately at the 7-8th intercostal space and the other 4 cm above, at the 5-6th intercostal space. The respiratory bands (BN-RESP-XDCR) were placed around the thorax (at the 4-5th intercostal space) and the diaphragm (slightly above the navel). After complete configuration, ECG/ICG and

respiratory signals were monitored for noisy signals and equipment adjusted accordingly. ECG/ICG and respiration data were collected through Biopacs at 1000 HZ and recorded on computer (Acqknowledge 4.4.1).

2.1.2.3. Testing

The experimental session was scripted and conducted through the computer (E-prime 2.0), though verbal instructions and prompts were offered by the experimenter if needed. Sessions always started with the respiration, ECG and ICG baseline physiological measurements (resting state), where the participant was instructed to sit still for five minutes in an upright position (with hands resting on their legs) and a grey screen was shown on the monitor. Only during the first session was this followed by a baseline SST, as the design with pre-post measurements in every session was deemed too time invested. The respiratory intervention followed next and took ~10 minutes. During the intervention phase physiological measurements were taken, these are the active condition measurement (reactive). The experimental session finished with the post-intervention SST and a debriefing at the last session.

2.1.3. Interventions

The three different interventions (abdominal breathing; thoracic breathing; focused breathing) were performed in as much comparable circumstances as possible. Participants were instructed to sit upright with their feet flat on the floor and about hip distance apart, knees bent. Breathing exercise instructions were presented in audio through headphones. The audio scripts were written, spoken and recorded by the second author. The intro and outro were standardized across interventions. Each different protocol started

with a tutorial (~2:30 min), followed by the experimental phase (~5:30 min), during which the intervention physiological measurements were taken. See Appendix A for a complete transcript. Visual prompts on the exercise were provided at 0:45, 1:15 and then every 1:00 min on screen.

2.1.3.1. Abdominal breathing

The aim of the abdominal breathing protocol was to change the locus of expansion towards the diaphragm and away from the chest. Thereby, we expected an increase in RMSSD and decrease in PEP. We expected respiration rates to go down and inhalation/exhalation ratio to remain the same. In the abdominal breathing tutorial this was taught by instructing participants to put their hands on their abdomen, flanking the navel, and during exhalation pull their navel in (by flexing the abdominal muscles) and release muscle tension and feel the abdomen expand naturally during inhalation. During the experimental phase participants were instructed to place their hands on their knees, while continuing the abdominal style of breathing. Thus participants breathed without receiving feedback from the hands during the intervention.

2.1.3.2. Thoracic breathing

The aim of the thoracic breathing protocol to keep the locus of expansion in the chest area or to move it there if the diaphragm was naturally more involved. Through this exercise we expected an increase in sympathetic activity (PEP) and decrease in in parasympathetic activity (RMSSD). We expected respiration rates to remain the same or go up and inhalation/exhalation ratio to remain the same. In the thoracic breathing

tutorial participants were instructed to put their hands on the side of their chest, flanking the breastbone, and breathe into their ribs, under their hands; and feel their ribs expanding and contracting. Again: during the experimental phase they were instructed to retain this breathing style, but with their hands on their knees.

2.1.3.3. Focused breathing

The focused breathing protocol was designed as a semi-control condition, with the aim to control for attentional effects on autonomous nervous system activity and cognition. Participants were instructed to focus their attention on their breathing and be aware of the expansion and contraction of abdomen and chest, without changing anything in their breathing. We expected respiration rate to remain the same or go down, as we hypothesize that paying attention to breathing slows down breathing (see Gerritsen & Band, 2018) while the inhalation/exhalation ratio is expected to remain stable. During the focused breathing tutorial participants were instructed to place their left hand on the abdomen and the right on the side of the ribs and proceed to experience movement of their hands during breathing, while paying attention to their breath. During the experimental phase this protocol was repeated again, but without the hand placement, in other words to just experience their breathing.

2.1.4. Stop-signal task

To represent inhibitory control SSRT was taken to indicate response inhibition - the inhibitory process in a race between go and stop processes according to the horse-race model -as measured by SST (Band, van der

Molen, & Logan, 2003; Logan & Cowan, 1984). As the SST we used the same task as in a previous experiment (Gerritsen et al., 2020), constructed in E-prime 2.0. The task consisted of a practice block (9 trials) and 3 experimental blocks of 36 trials each. One third of trials were *stop trials* and two thirds *go trials*. Target stimuli were either 'X' or 'O' and had to be responded to by left or right hand (counter-balanced) as fast as possible. On *stop trials* a tone was sounded signaling participants to refrain from responding. The stop signal delay between target and stop signal was dynamically altered by staircase tracking procedure (30ms steps), which should result in a proportion of responding of ~50% (Ridderinkhof et al., 1999; Verbruggen & Logan, 2008). Participants were explicitly instructed to not wait for the tone, and feedback on screen prompted to not wait and speed up if responses were slow, after every trial. SSRT was obtained by the nth method. We follow the recommendations of Verbruggen and colleagues (2019). Because in 10 of the 29 participants at least 1 of the 4 measurements the p(respond) was lower than .25 or higher than .75, we had to exclude them from further analysis, resulting in n=19.

2.1.5. Physiological analysis

All physiological channels of every session were loaded into PhysioDataToolbox 0.50 (Sjak-Shie, 2021) from Acqknowledge 4.4.1 files (Biopac Systems, Inc, USA) and converted to .physioData file format. Two epochs were made per session: baseline and condition, of 5 min. each, as the recommended minimum for a reliable HRV measurement by the HRV Task Force (Malik et al., 1996). All pre-analyses were done by the dedicated PhysioDataToolbox analyzers.

2.1.5.1. Respiration

Respiration channels were analyzed for respiratory patterns by respiration analyzer (moving-mean of intercepts), with the following parameters: low-pass filter: 4 Hz; moving-mean multiplier: 2; min-max inhalation & exhalation duration: 1-20s; outlier filter threshold-span: 3.5s-100s; slope gradient threshold: 10%; smoothing window width: 5s. In about a quarter of epochs analyzer was unable to detect respiration peaks. For these, settings were changed to an inhalation minimum of 0.5s and outlier threshold of 5s. After this parameter tweak, peaks were detected normally by the toolbox. All signals of both respiration channels were checked for artefacts by eye. Peaks were removed on the basis of three criteria: 1) multiple peaks in one waveform, 2) large drift (lopsided) and 3) peaks detected in a flat curve (false positives from adjusted settings); as these resulted in under- or overestimation of respiration rate or duration of inhalation vs exhalation. Mean inhalations, exhalations, breath duration and amplitude were extracted for further analysis for each band. Breath duration was computed to breaths-per-minute (respiration rate). Inhalation/exhalation ratio was computed by dividing mean inhalation duration by mean exhalation duration. Note that, the duration is of either is relative and not absolute. The flat interval of the respiration curve is (arbitrarily) counted towards exhalation, as the analyzer times the start of inhalation by the rising curve. Changes in ratio are therefore the only analysis of interest. Tho/Abd ratio was determined by dividing the mean amplitude of the thoracic band by the mean amplitude of the abdominal band. Note that, the absolute value of respiration amplitude is in itself not an informative measure and it can vary widely even within-subjects, e.g. by the way it has been attached in a specific session. Therefore, it is only used to look for relative changes in the locus within a single session. But it has to be noted that even then other sources of change cannot be ruled out, such as

band movement. The PhysioDataToolbox 0.50 respiration analyzer follows the methods proposed by Lu et al. (2006).

2.1.5.2. ECG

Heart rate (HR) and IBIs were obtained from the PhysioDataToolbox ECG analyzer, that automatically detects R-peaks, with the following settings: high-lowpass filter: 1-50 Hz; min. R-peak: 0.35 mV; min. R-R: 0.3s; min-max IBI: 0.3-2s. In 3 epochs (of 174) the amplitude of the R-peak was frequently too low to be detected. Here the R-peak min. was set to 0.3mV. All ECG signals were checked by eye and artefacts removed manually. This was always done by removing IBIs from the signal and caused by: 1) missing R-peak or 2) wrong peak detection (noise or missing R). Artefacts were detected in a small subset of participants and within these only a few artefacts had to be removed for each epoch, per participant. HR and RMSSD were extracted for further analysis.

2.1.5.3. ICG

PEP (Newlin & Levenson, 1979) was obtained by ICG ensemble analyzer. The Q-point was manually selected in the ECG ensemble on the last downward peak before R (Q-peak) and B-point in the dZ/dt ensemble by picking the lowest point before the upwards slope towards C-peak. PEP is widely seen as a valid indicator of sympathetic tone (Berntson et al., 1994; Cacioppo et al., 1994). See Sherwood and colleagues (1990) for further details on ICG measures and analyses.

2.1.6. Statistical analyses

All analyses were done in JASP 0.15.0.0 with Bayesian counterparts of classic statistical tests. The models were always compared to null and the best-fitting model was deemed to have ample evidence for or against it if 3 < BF < 0.33. If the top model was within these bounds no definitive statement can be made between it and null (underpowered sample). Two Bayesian repeated measures ANOVA analyses were performed as manipulation and sanity check, on Tho/Abd ratio (Time) for each session and HR respectively (`session*time). Main tests of our hypothesis were Bayesian repeated measures ANOVA (*session*T\time*) on respiration rate, inhalation/exhalation ratio, RMSSD, PEP, SSRT, Go RT and Go Accuracy, with *session order* as a covariate. If there are three models or less in the analysis, all BF are reported, if there are more only the most dominant three are reported, when necessary followed by an analysis of relative effect. Analyses of respiration was performed on Tho channel as this had the strongest signal and least noise. Difference scores (D-scores) were obtained for each of the main psychophysiological dependents: respiration rate, inhalation/exhalation ratio, RMSSD and PEP, by subtracting baseline score from intervention score (Dt1t2). A Bayesian correlation matrix was constructed, testing for a relationship for any possible combination of these D-scores. If the session*time model was found to be dominant for the cognitive measure in the ANOVA, this matrix would be done a second time with SSRT for each time point, but only for the participants included in SST analysis.

2.2 Results

2.2.1. Respiration

2.2.1.1. Thoracic/abdominal ratio

Figure 1 shows Tho/Abd for all 3 interventions, from baseline to condition. Bayesian repeated-measures ANOVA [*time(2)*] of abdominal breathing session with *session order(6)* as a covariate indicated *time* as the best-fitting model with BF_{10}(*time*) = 58.9, error % = 1.9; followed by BF_{10}(*time + session order*) = 30.6, error % = 2.5; and BF_{10}(*session order*) = 0.53, error % = 1.4. So there is very strong evidence for Tho/Abd going down during the condition measurement, in other words: locus of respiration moved towards the abdomen in abdominal breathing condition. The Bayesian repeated-measures ANOVA [*time(2)+ session order(6)*] of thoracic breathing session indicated *session order* as the best-fitting model with BF_{10}(*session order*) = 1.4, error % = 52.1; followed by BF_{10}(*time*) = 0.26, error % = 1.2; and BF_{10}(*time+ session order*) = 0.18, error % = 2.4. So there is evidence against Tho/Abd changing from baseline to the condition measurement and it is unclear whether order had an effect. It seems that locus of respiration remained thoracic in thoracic breathing. Lastly, a Bayesian repeated-measures ANOVA [*time(2)+ session order(6)*] of focused breathing session indicated *null* as the best-fitting model, followed by *time* BF_{10}(*time*) = 0.71, error % = 1.4; BF_{10}(*session order*) = 0.47, error % = 0.6; and BF_{10}(*time + session order*) = 0.34, error % = 1.1. There seems to be no clear evidence either for or against changes in Tho/Abd from baseline to condition in focused breathing.

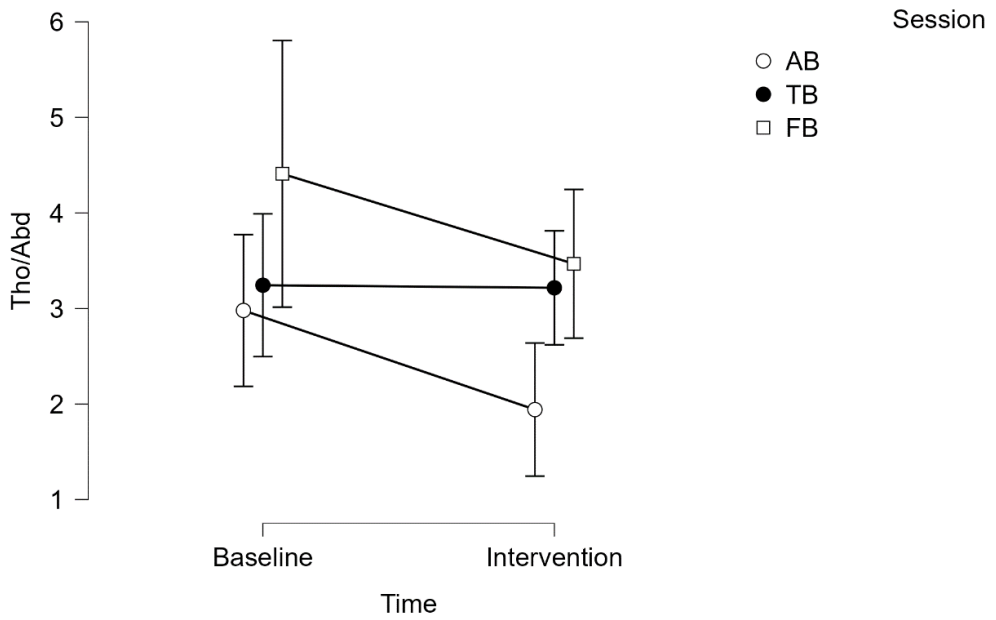

Figure 1: Ratio of *thoracic amplitude / abdominal amplitude for all three conditions, between baseline and intervention measurement. AB = Abdominal breathing; TB = Thoracic breathing; FB = Focused breathing; Tho= Thoracic amp (mV); Abd = Abdominal amp (mV).*

2.2.1.2. Respiration rate

Figure 2 shows respiration rate development for all interventions. The Bayesian repeated-measures ANOVA [*session(3)*time(2)+session order(6)*] showed *session+time* as the best-fitting model with BF_{10}(*session+time*) = $2.5*e^{21}$, error % = 7.2, but is very closely followed by *time:* BF_{10}(*time*) = $2.4*e^{21}$, error % = 1.7; and then BF_{10}(*time+session order*) = $1.6*e^{21}$, error % = 3.0. Analysis of effects produces a BF_{incl}(*time*) of infinity, a BF_{incl}(*session*) = 0.85 and BF_{incl}(*session order*) = 0.50. Clearly, respiration rate goes down in all three conditions in the intervention phase, as there is extreme evidence for this main effect.

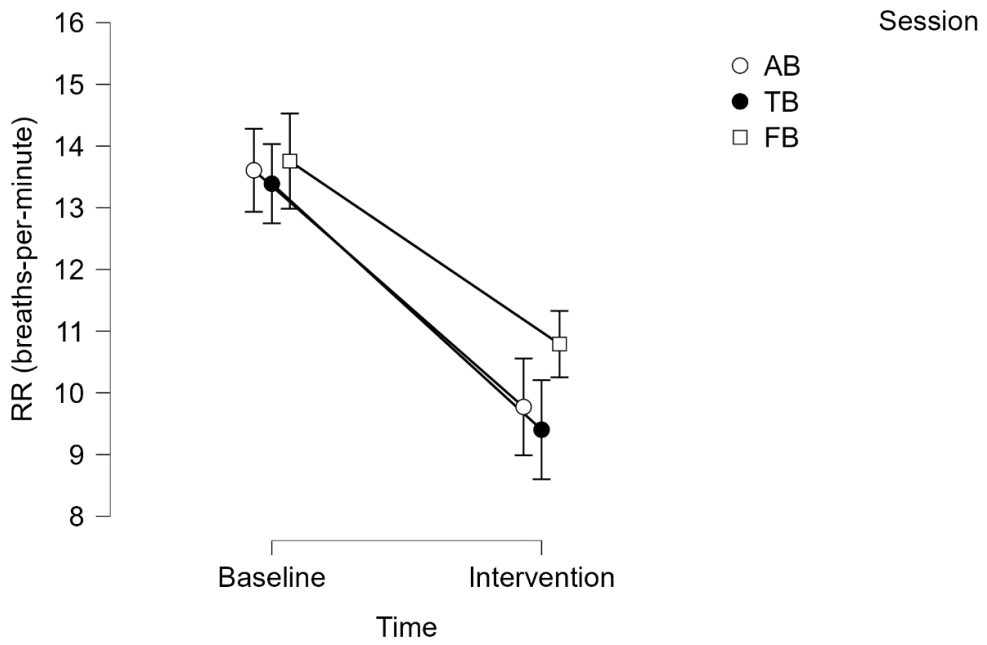

Figure 2: *RR for all three conditions, between baseline and intervention measurement (in breaths-per-minute). RR = respiration rate.*

2.2.1.3. Inhalation/exhalation ratio

Figure 3 shows the inhalation/exhalation ratio ratio across conditions and measurement points. Repeated measures Bayesian analysis [*session(3)*time(2)+session order(6)*] again shows dominance of BF(*time)* = 4.4*e^6, error % = 0.8; followed by BF(*time+session order)* = 1.8*e^6, error % = 1.3; and BF(*session+time+session*time)* = 1.3*e^6, error % = 2.7. Analysis of effects shows a contribution of BF_{incl}(*time*) = 4.4*e^6, a BF_{incl}(*session*) = 0.37 and BF_{incl}(*session*time*) = 0.93. In all three conditions participants increased inhalation as compared to exhalation duration, from baseline to intervention.

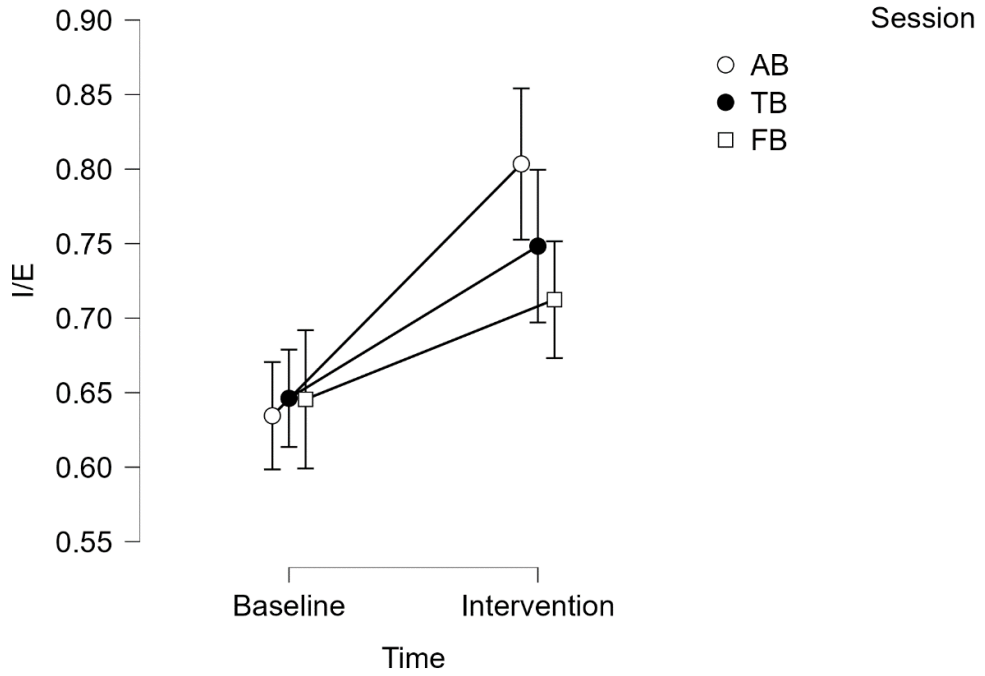

Figure 3: *I/E ratio for all three conditions, between baseline and intervention measurement. I/E = inhalation duration (ms) / exhalation duration (ms).*

2.2.2. ECG

2.2.2.1. Heart rate

As a sanity check HR was analyzed by Bayesian repeated measures ANOVA [*session(3)*time(2)+session order(6)*]: *null* was dominant, followed by BF_{10}(*session order*) = 0.54, error % = 2.3; BF_{10}(*time*) = 0.23, error % = 1.2; and BF_{10}(*time+session order*) = 0.13, error % = 2.9. HR did not change from baseline to condition measurements points. It is unclear whether session order had an effect on HR.

2.2.2.2. Heart rate variability

Figure 4 shows RMSSD per condition developing in time. A Bayesian repeated measures ANOVA [*session(3)*time(2)+session order(6)*] indicated that here also *null* was the strongest model. The next model was that of *time*: BF_{10}(*time*) = 0.95, error % = 2.9; then BF_{10}(*session order*) = 0.60, error % = 0.6; thirdly, BF_{10}(*time+session order*) = 0.54, error % = 1.2. Counter to expectation there was no evidence that HRV was affected by any intervention.

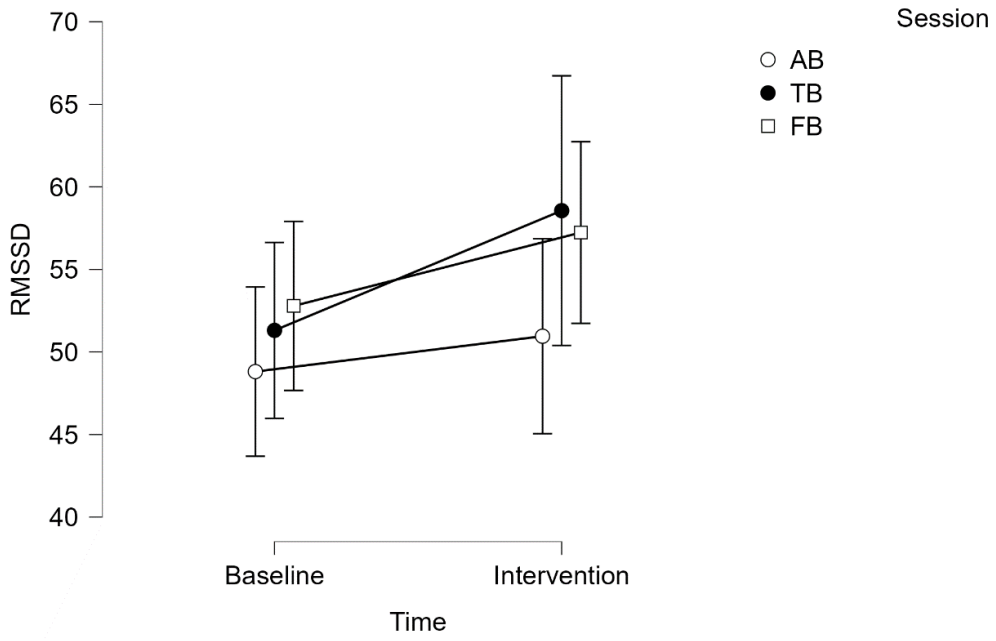

Figure 4: *RMSSD for all three conditions, between baseline and intervention measurement. RMSSD = root mean square of successive differences.*

2.2.3. ICG

2.2.3.1. PEP

Figure 5 shows changes in PEP from baseline to condition, for each condition (in ms). The Bayesian repeated measures ANOVA [*session(3)*time(2)+session order(6)*] did find evidence for effects. The fittest model being: BF_{10}(*session+time+session*time*) = $1.2*e^6$, error % = 31.7; then BF_{10}(*session+time+session*time+session order*) = $5.5*10^5$, error % = 3.3; thirdly, BF_{10}(*session+time*) = $2.1*10^3$, error % = 1.6. Analysis of effects showed the contribution of BF_{incl}(*session*) = $3.7*10^5$; BF_{incl}(*session*time*) = $1.1*10^3$; BF_{incl}(*time*) = 387.0; and BF_{incl}(*session order*) = 0.45. Though error is very high it seems there is extreme evidence for the complete model. Figure 4 shows that abdominal breathing condition starts out relatively low in sympathetic activity, but this goes markedly up during intervention (PEP going down). While sympathetic activity during thoracic breathing and focused breathing seems unaffected, if anything it goes up in thoracic breathing (PEP going down). This pattern of results is the opposite of our predictions.

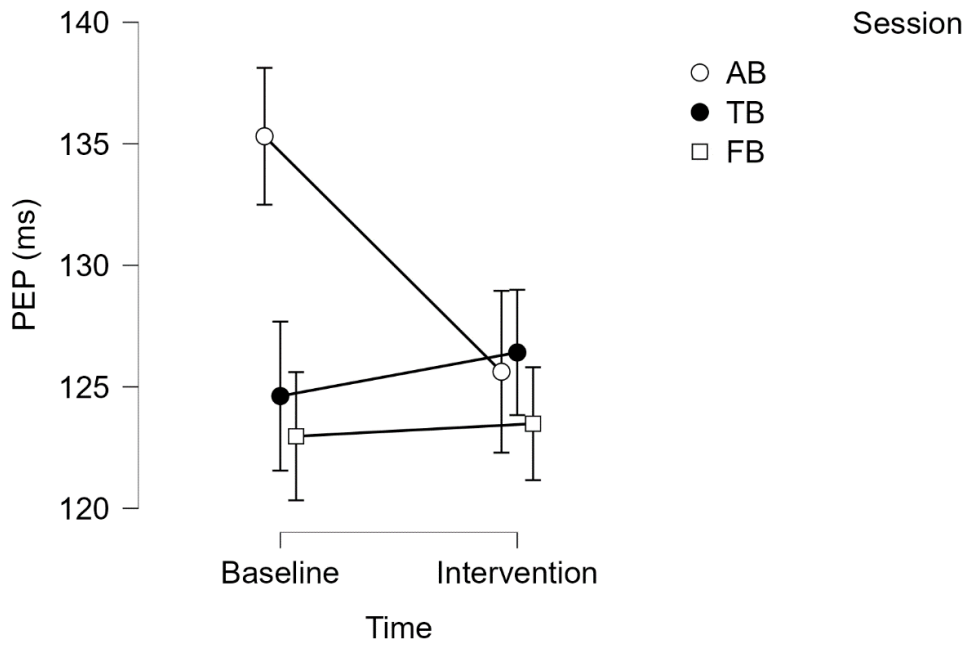

Figure 5: *PEP for all three conditions, between baseline and intervention measurement. PEP = pre-ejection period.*

2.2.4. Stop-signal task

Bayesian repeated-measures ANOVA [*time(4)+ session order(6)*] of Go trial accuracy indicated *null* as fittest model. There is strong evidence that both *time* and *time+session order* have no effect on Go accuracy. The same analysis on Go RT, produces similar results: BF_{10}(*session order*) = 1.05, error % = 1.9; BF_{10}(*time+session order*) = 0.32, error % = 22.1; and BF_{10}(*time*) = 0.22, error % = 0.6. There is evidence that neither *time* nor the complete model explains Go RT results.

Figure 6 shows SSRT across the four conditions. Bayesian repeated-measures ANOVA [*time(4)+ session order(6)*] indicated the complete model as fittest BF_{10}(*time+session order*) = 1.9, error % = 1.3; followed by BF_{10}(*time*) = 1.9, error % = 0.4; and lastly BF_{10}(*session order*) = 0.93,

error % = 0.8. There is insufficient evidence that *time* had an effect on SSRT in either direction, which is not surprising as 10 participants were excluded because of waiting: p(respond) < .25, and thus the sample was underpowered. However, looking at Figure 6 a pattern emerges where SSRT goes down across conditions from baseline, but markedly after abdominal breathing.

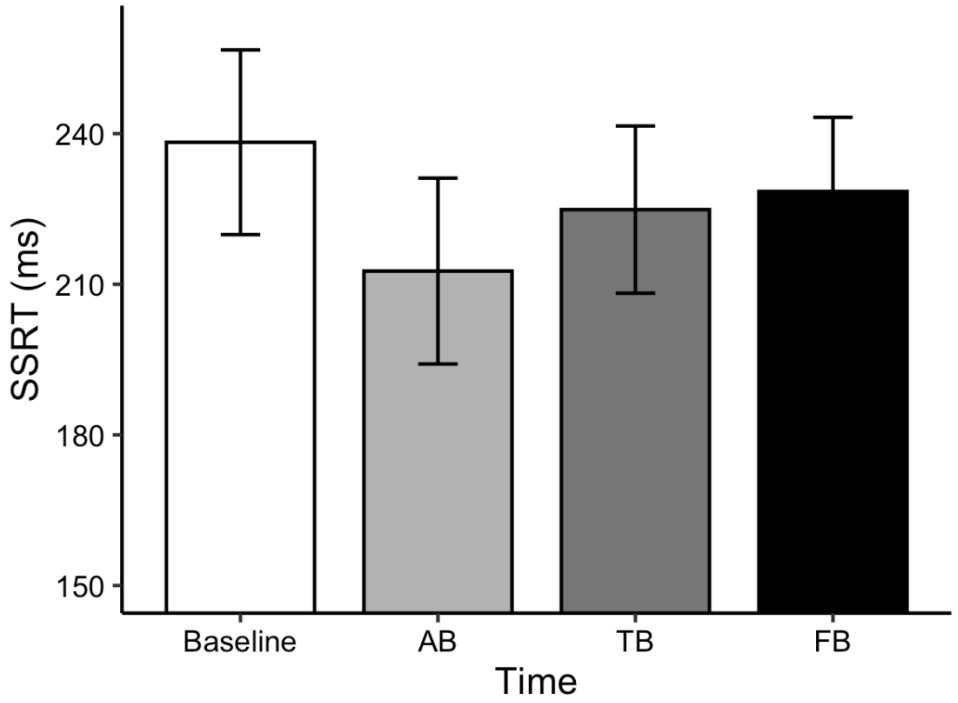

Figure 6: *SSRT across the four measurements: baseline (first session) and after AB, TB, FB. SSRT = stop-signal reaction time in ms.*

2.2.5. D-score correlations

See Appendix B Table 1 for the complete matrix. Only four of the possible correlations had a BF_{10}>10. All of which were uninformative, for example: correlations of respiration rate D-scores between sessions. An

important note: there was no relation found between respiration and RMSSD D-scores, which was unexpected.

3. Experiment 2

3.1. Method

If not reported, the methodology is the same as in Experiment 1. Only deviations from the design and procedure are mentioned in this section.

3.1.1. Participants

In total 41 participants were recruited. The first 3 participants were dropped because the ECG was not set up correctly (parameter settings). Another 4 had to be dropped because the ECG signal was lost during the session (at a measurement point), making it unusable for analysis and interpretation. This left n=34 for analysis, including 3 males (M=25.3, 23-28) and 31 females (M=20.7, 18-28). Next to the prescriptions described above, participants were given 180 ml of water before each session and not allowed to drink anything during the session, with the aim to level effects of hydration on HRV (Heathers et al., 2018).

3.1.2. Procedure

3.1.2.1. Design

Participants completed only two sessions: abdominal breathing and thoracic breathing. Each session took ~75 minutes.

3.1.2.2. Physiological measures

Participants were equipped with 3 ECG and 2 EDA electrodes (on the left hand), after skin preparation. Two respiration bands were used in the same way as described above.

3.1.2.3. Testing

The experiment started with the baseline resting state physiological measurement of respiration, ECG and EDA (~5 min); followed by the pre-intervention affect grid and Simon task. Then the intervention commenced, during which condition physiological measurements (reactive) were taken. Another resting state respiration, ECG and EDA followed (recovery), after which the post-intervention affect grid and Simon task finished the testing phase.

3.1.3. Interventions

Abdominal breathing condition and thoracic breathing scripts were rewritten and recorded by the first author, using another voice-actor as audio guide. The main change was that abdominal breathing protocol now explicitly emphasized slower breathing than normal and thoracic breathing protocol emphasized slightly speeded breathing. This was also reflected in the pace and tone of the voice recording. See Appendix A for the complete transcript.

3.1.4. Behavioral measures

3.1.4.1. Affect grid

The affect grid (Russell et al., 1989) was taken immediately pre- and post-intervention to assess self-reported changes in arousal and pleasure (positive-negative affect). It consists of two dimensions on the axes of a 9x9 grid, where a participant places a marker on the grid to signal current state. The middle of the grid, center square, reflects a neutral stance, while moving down or left moves to unaroused or negative and moving up or right adjusts to aroused or positive, respectively.

3.1.4.2. Simon task

The Simon effect – interference scores of conflicting stimulus spatial dimensions on stimulus and response in accuracy and RT – was taken as indicator of cognitive inhibition. Simon task (Simon, 1969) consisted of a practice block of 40 trials and 3 testing blocks of 60 trials each. Trials started with a centered black fixation square presented for 1000ms (against the light grey background). Then either a blue or green circle target appeared to the left or right of fixation for 1500ms or until a response was made. Participants had to respond to the color of the stimulus, ignoring the location, with either Q or P keys (counter-balanced). Accuracy and reaction time data is compared for congruent and incongruent trials, where congruent trials are defined as corresponding location of stimulus and response (e.g. left on screen; Q response) and incongruent trials as juxtaposed positioning (e.g. left on screen; P response). Note that stimulus location is irrelevant to the task goals. The interference here (Simon effect) is an operationalization of cognitive inhibition. Each session had a pre-test and post-test Simon task. Pre-test

followed baseline physiology and preceded affect grid pre-test; post-test followed post-test affect grid.

3.1.5. Physiological analyses

PhysioDataToolbox 0.6.1 was used instead. Three epochs of 5 min. were analyzed for each channel: baseline resting state, during the intervention (reactive) and resting state post-intervention (recovery).

3.1.5.1. Respiration

Inhalation min. and max. were set to 0.5 and outlier threshold of 5s for all epochs.

3.1.5.2. ECG

Identical to Experiment 1.

3.1.5.3. EDA

EDA channel was checked for quality of signal. No changes were made. Skin conductance level (SCL) was obtained per epoch.

3.1.6. Statistical analyses

Almost identical to Experiment 1, apart from SCL substituting for PEP and Simon effects replacing SSRT. Furthermore, a second D-score

(Dt2t3) was calculated between measurement point 2 and 3, by subtracting the intervention values from the recovery values.

3.2. Results

3.2.1. Respiration

3.2.1.1. Thoracic/abdominal ratio

Figure 7 shows Tho/Abd for the 2 interventions, from baseline to recovery. Bayesian repeated-measures ANOVA [*time(3)*] of abdominal breathing session with *session order(2)* as a covariate indicated *time* as the best-fitting model with BF_{10}(*time*) = 12.5, error % = 0.7; followed by BF_{10}(*time + session order*) = 4.6, error % = 2.3; and finally *session order*: BF_{10}(*session order*) = 0.34, error % = 1.8. There is strong evidence that the relative amplitude of Tho and Abd changes across abdominal breathing condition measurements. As seen in Figure 7, Abd locus increases during intervention and recovers during recovery to higher Tho locus.

The Bayesian repeated-measures ANOVA [*time(3)+session order(2)*] analysis of thoracic breathing session had *null* as dominant, followed by *session order*: BF_{10}(*session order*) = 0.72, error % = 1.7; followed by BF_{10}(*time*) = 0.46, error % = 0.8; and finally BF_{10}(*time + session order*) = 0.34, error % = 1.5. For thoracic breathing, there is no evidence for any effect of any of the factors.

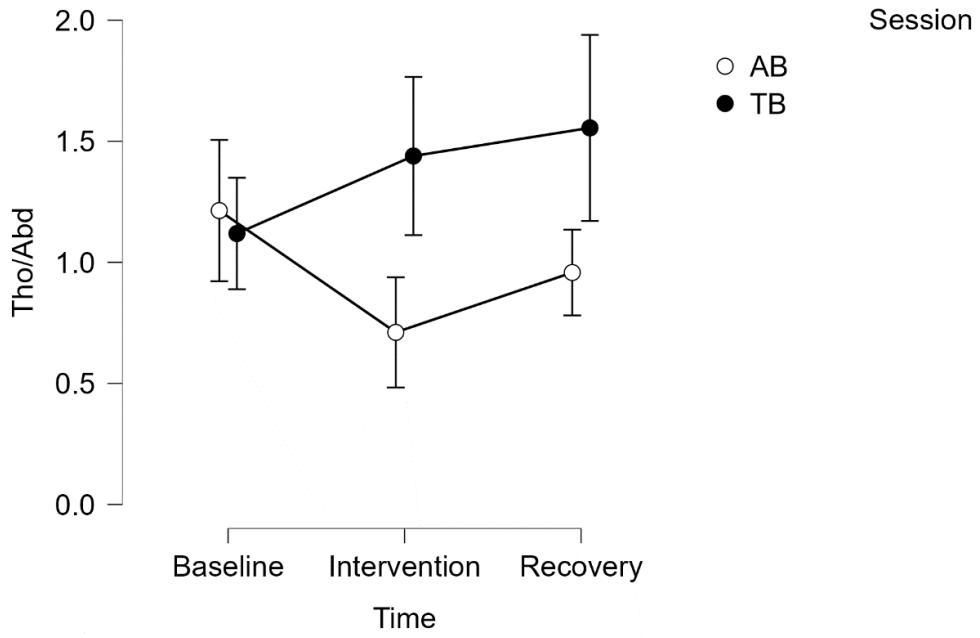

Figure 7: Ratio of *thoracic amplitude (mV) / abdominal amplitude (mV) for session AB and TB, across the 3 measurement. Tho=Thoracic amp; Abd=Abdominal amp.*

3.2.1.2. Respiration rate

Figure 8 shows respiration rate development for both sessions. As a manipulation check a Bayesian repeated-measures ANOVA [*session(2)*time(2)+session order(2)*] analysis was performed, It showed the complete model as best-fitting: BF_{10}(*session+time+session order+session*time*) = $5.1*e^{22}$, error % = 2.7; followed by BF_{10}(*session+time+session*time*) = $3.5*e^{22}$, error % = 2.1; and thirdly by all main effects: BF_{10}(*time+session+session order*) = $1.3*e^{6}$, error % = 2.3. In the analysis of effects all BF_{incl} are infinity, except for BF_{incl}(*session order*) = 1.5. Looking at Figure 8 and the extreme evidence for the complete model it can be concluded that respiration rate goes down during the intervention phase in abdominal breathing condition, in contrast to thoracic breathing

where it goes up. In both sessions it returns towards baseline during the recovery phase. Clearly, participants followed the instructions and performed according to expectation in Experiment 2, as compared to Experiment 1. However, note that, in abdominal breathing condition even during intervention the "golden" respiration rate of 6 breaths-per-minute (van Diest et al., 2014) is not reached on average, by far.

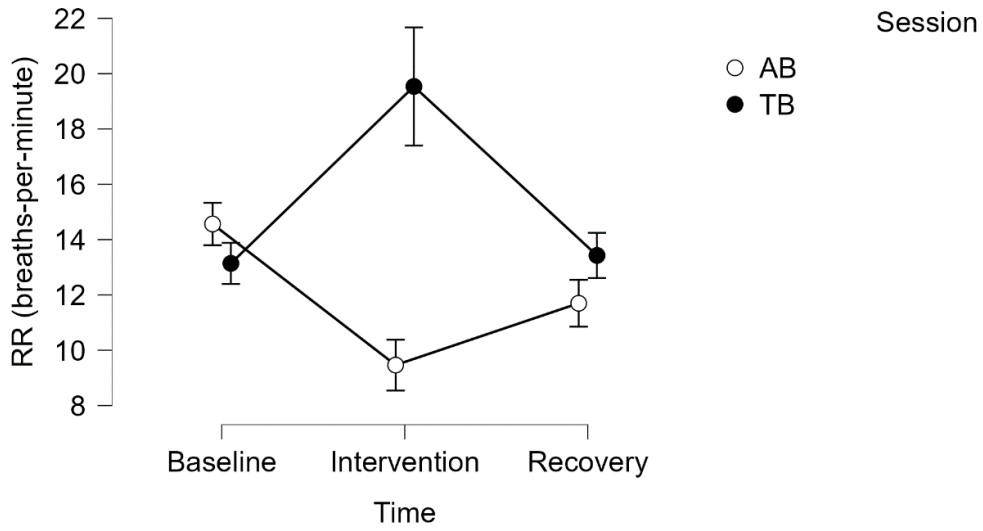

Figure 8: *RR for both sessions, across measurement points (in breaths-per-minute). RR=respiration rate.*

3.2.1.3. Inhalation/exhalation ratio

Figure 9 shows the inhalation/exhalation ratio ratio, for the three measurement points baseline, condition and recovery for each session. A Bayesian repeated-measures ANOVA [*session(2)*time(2)+session order(2)*] analysis indicated as best-fitting: BF_{10}(*session+time+session*time*) = $1.7*e^6$, error % = 1.8; followed by the complete model: BF_{10}(*session+time+session order+session*time*) = $4.9*10^5$, error % = 4.2; and then *time*: BF_{10}(*time*) = $4.7*10^6$, error % = 6.3. The analysis of effects gives the strongest

contribution of *time:* BF_{incl}(*time*) = 1.1*e^6; then *session*time*: BF_{incl}(*session*time*) = 9.6; thirdly *session* BF_{incl}(*session*) = 2.7; and finally: BF(*session order*): 0.29. Evidently, ratio changes across measurement where inhalation length goes up during both interventions, though more strongly for thoracic breathing, and goes down again in recovery.

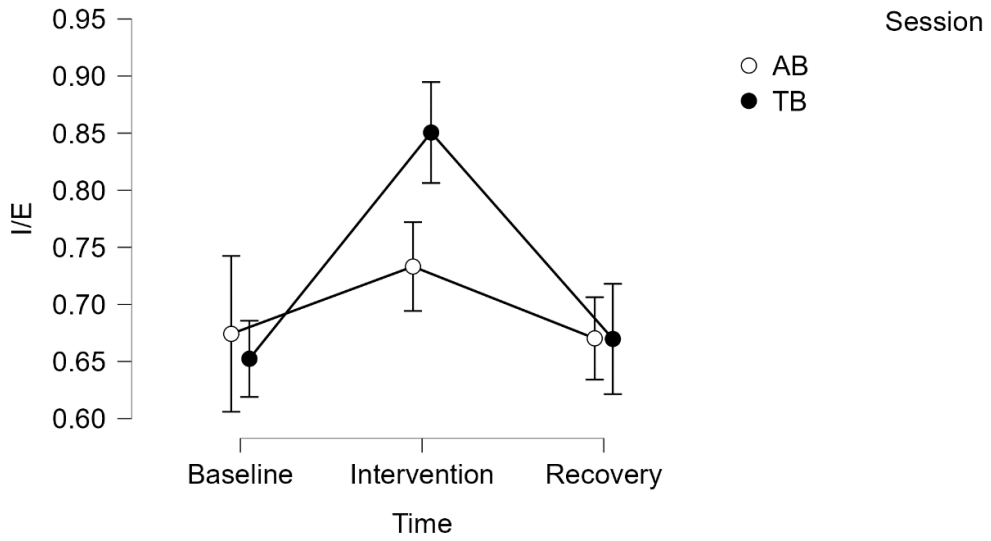

Figure 9: *I/E ratio for AB and TB, from baseline to recovery measurement. I/E ratio = inhalation duration (ms) / exhalation duration (ms).*

3.2.2. ECG

3.2.2.1. Heart rate

Heart rate was analyzed by Bayesian repeated measures ANOVA [*session(2)*time(2)+session order(2)*]. The model with main effects *session* and *time* was dominant: BF_{10}(*session+time)* = 747, error % = 1.6; followed by all main effect model: BF_{10}(*session+time+session order*) = 379, error % = 3.7; and thirdly: BF_{10}(*session+time+session*time*) = 116, error % = 2.3.

Analysis of effects showed a relative higher contribution of *session*: BF_{incl}(*session*) = 55.1; than *time* BF_{incl}(*time*) = 11.8. Clearly, heart rate is higher during the thoracic breathing than abdominal breathing session and it goes up during both interventions, going down again during recovery. Apparently, both interventions are taxing. See Figure 10 for a visualization.

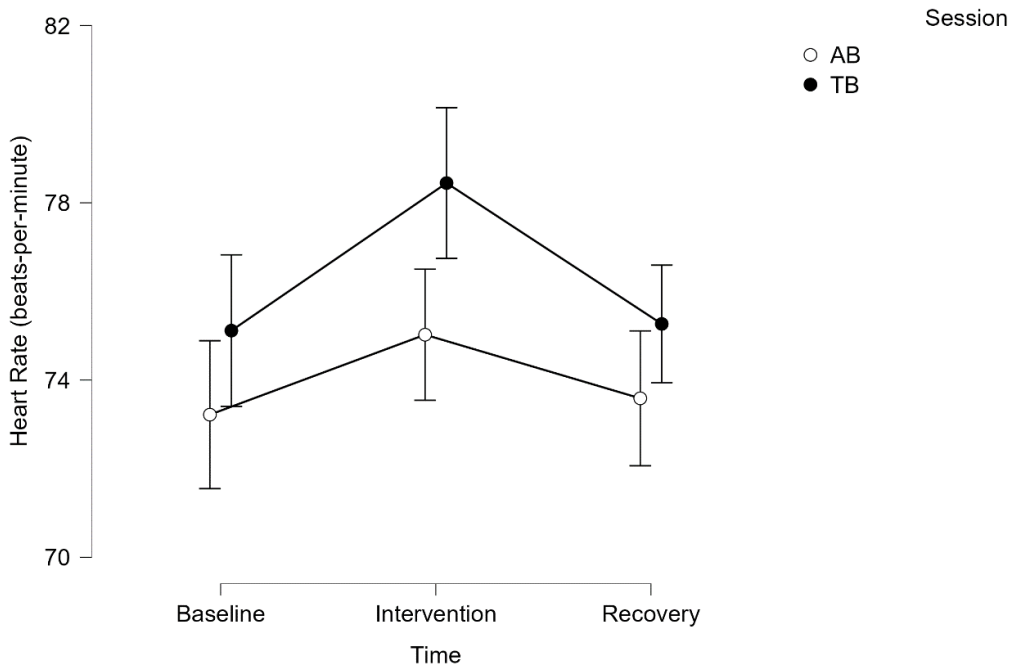

Figure 10: *Heart rate (in beats-per-minute) for both session, between baseline and recovery measurement.*

3.2.2.2. Heart rate variability

RMSSD was analyzed by Bayesian repeated measures ANOVA [*session(2)*time(2)+session order(2)*], see Figure 11. The model with only the main effect *session* was dominant: BF_{10}(*session)* = 55.5, error % = 7.1; followed by: BF_{10}(*session+session order*) = 24.6, error % = 2.6; and thirdly: BF_{10}(*session+time*) = 13.2, error % = 1.7. An analysis of effects only showed a significant contribution of *session*: BF_{incl}(*session*) = 38.1. Our expectations

regarding HRV changes were not met. It seems that overall RMSSD was higher in the abdominal breathing than thoracic breathing session, even before intervention. In Figure 11 a dip can be seen in RMSSD during intervention, but this is not reflected in the analysis by an interaction effect.

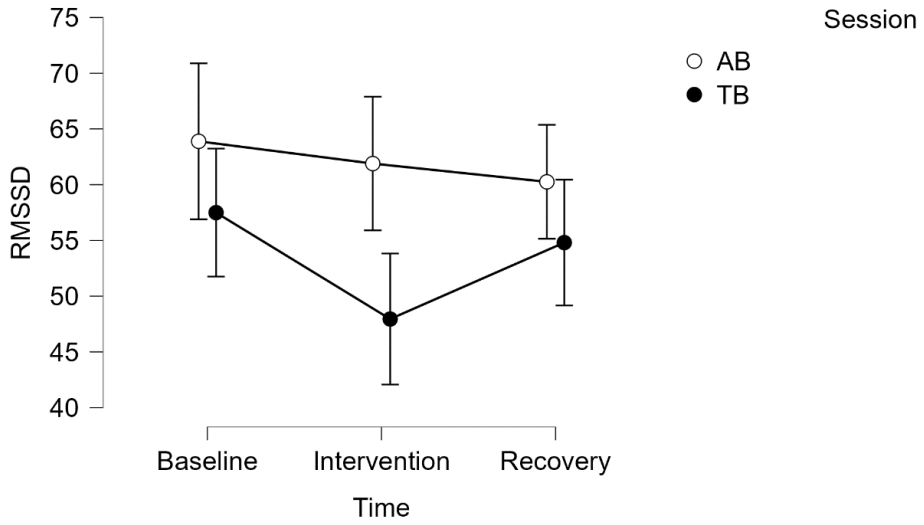

Figure 11: *RMSSD for the two sessions, between baseline and recovery measurement. RMSSD = root mean square of successive differences.*

3.2.3. EDA

3.2.3.1 SCL

See Figure 12 for a visualization of SCL mean development. A Bayesian repeated-measures ANOVA [*session(2)*time(2)+session order(2)*] analysis indicated as best-fitting the *null*, followed by: BF_{10}(*session order)* = 0.77, error % = 2.2; and thirdly: BF_{10}(*time*) = 0.17, error % = 1.0. Expectations on stress development were also not met. Although Figure 12 suggests an increase in SCL during the thoracic breathing session and after intervention for abdominal breathing session, there is actually clear evidence

against all effects apart from session order and extreme evidence against this interaction: BF_{incl}(*session*time*) = 0.008.

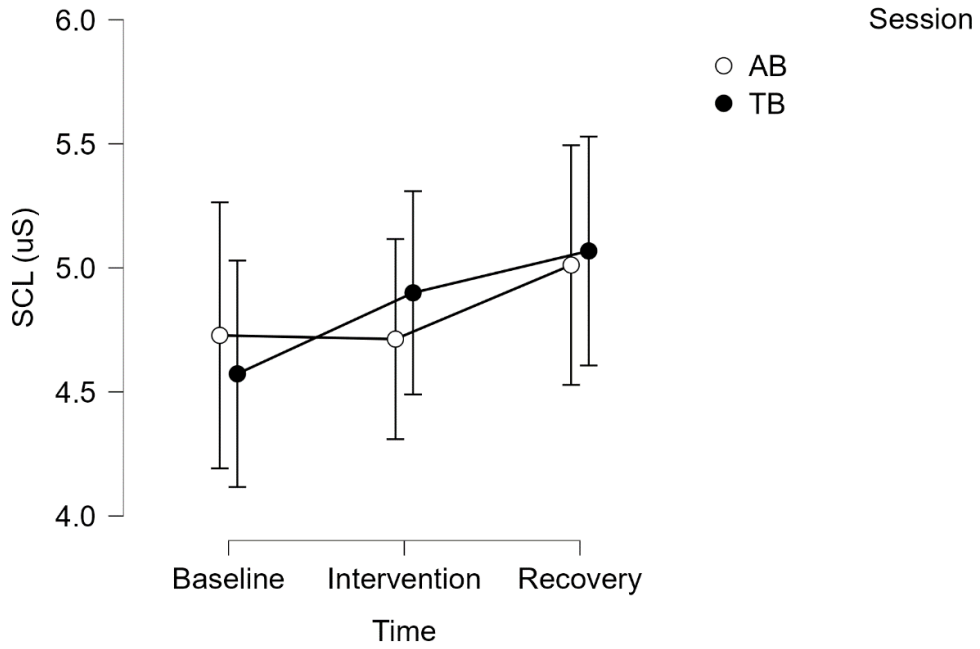

Figure 12: *SCL for the two sessions, between baseline and recovery measurement. SCL = Skin conductance level in microSiemens.*

3.2.4. Behavioral measures

3.2.4.1. Affect grid

Figure 13 shows plots for the self-reported arousal and pleasure components of the affect grid, during both sessions. Bayesian repeated-measures ANOVAs [*session(2)*time(2)+session order(2)*] were performed for both dimensions.

In arousal the analysis indicated BF_{10}(*session+time*) = $8.3*10^3$, error % = 4.2 as best-fitting; followed by: BF_{10}(*session+time+session*time)*

= 3.8*10^3, error % = 2.0; then thirdly: BF_{10}(*session+time+session order*) = 2.5*10^3, error % = 2.6. The major contribution according to analysis of effects here is *time*: BF_{incl}(*time*) = 2.7*10^3; with a moderate effect of *session*: BF_{incl}(*session*) = 3.7. Clearly, arousal goes down after the intervention and there might be an overall lower arousal level in abdominal breathing condition, as compared to thoracic breathing condition, that according to the second model might be because of the interaction effect: after abdominal breathing arousal goes down more than after thoracic breathing.

For pleasure the most dominant model was BF_{10}(*session+time+session*time)* = 2.4, error % = 3.1; followed by the complete model BF_{10}(*session+time+session order+session*time)* = 0.9, error % = 3.3. This does not merit any conclusion regarding these effect, as the sample is underpowered. However, when looking at the analysis of effects and Figure 13 an interaction effect can be seen: BF_{incl}(*session*time*) = 5.3. There is slight indication that abdominal breathing produces pleasure, while thoracic breathing diminishes it.

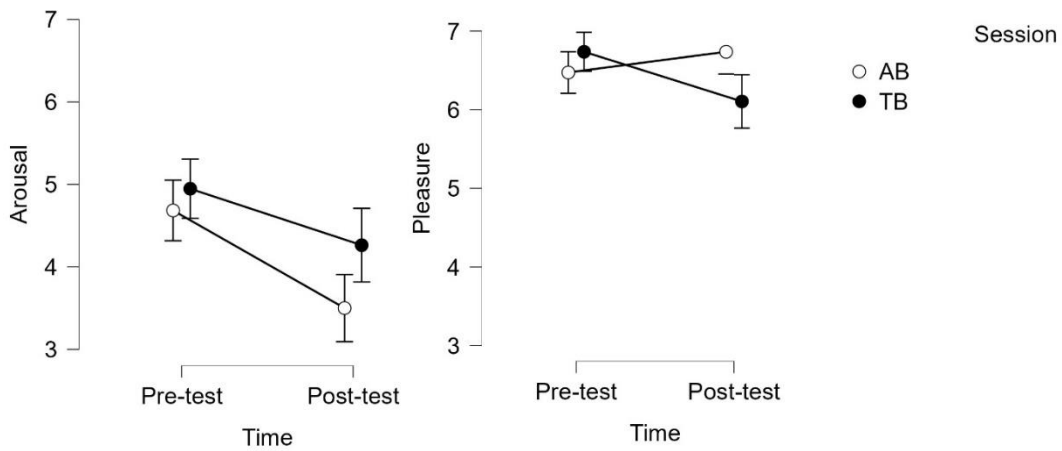

Figure 13: *Affect grid dimensions for arousal (left) and pleasure (right) for both sessions, pre-test to post-test.*

3.2.4.2. Simon task

3.2.4.2.1. Accuracy

A Bayesian repeated-measures ANOVA [*session(2)*time(2)*congruency(2)+session order(2)*] was performed on the accuracy data of the Simon task. The dominant model was: BF_{10}(*session+time+congruency+session*time)* = $1.3*10^4$, error % = 3.0; then by: BF_{10}(*time+ congruency)* = $7.1*10^3$, error % = 2.0; and closely followed by: BF_{10}(*congruency)* = $6.8*10^3$, error % = 1.0. Analysis of effect shows the dominant factor to be *congruency:* BF_{incl}(*congruency)* = $4.1*10^3$. Figure 14 plots these accuracy means. Clearly, congruency had an effect where overall congruent trials were more accurate. The dominant model and Figure 14 also suggest an interaction effect, where after thoracic breathing accuracy goes down, while after abdominal breathing accuracy seems unaffected or even goes slightly up for congruent trials.

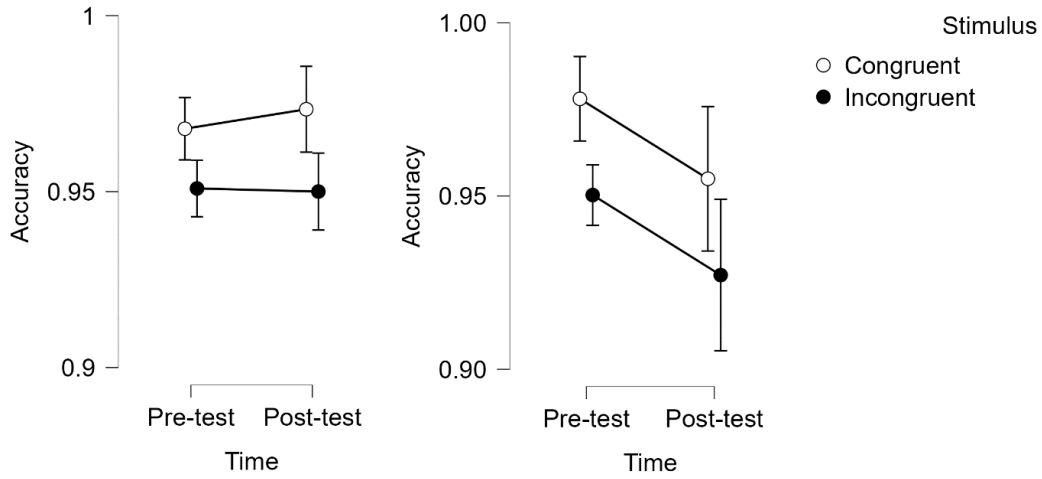

Figure 14: *Simon task accuracy data. Left graph Abdominal session and right graph Thoracic session.*

3.2.4.2.2. Reaction time

A Bayesian repeated-measures ANOVA [*session(2)*time(2)*congruency(2)+session order(2)*] was performed on the reaction time data. The dominant model was: BF_{10}(*congruency)* = 2.3*e^6, error % = 0.9; followed by: BF_{10}(*congruency+session order)* = 1.8*e^6, error % = 2.7; and then by: BF_{10}(*session+congruency)* = 4.7*10^5, error % = 1.3. Also here, analysis of effect showed the single dominant factor to be *congruency:* BF_{incl}(*congruency)* = 3.8*e^7. Figure 15 visualizes this data. Evidently, only congruency had an effect on reaction times, where congruent trials were faster than incongruent trials across all conditions.

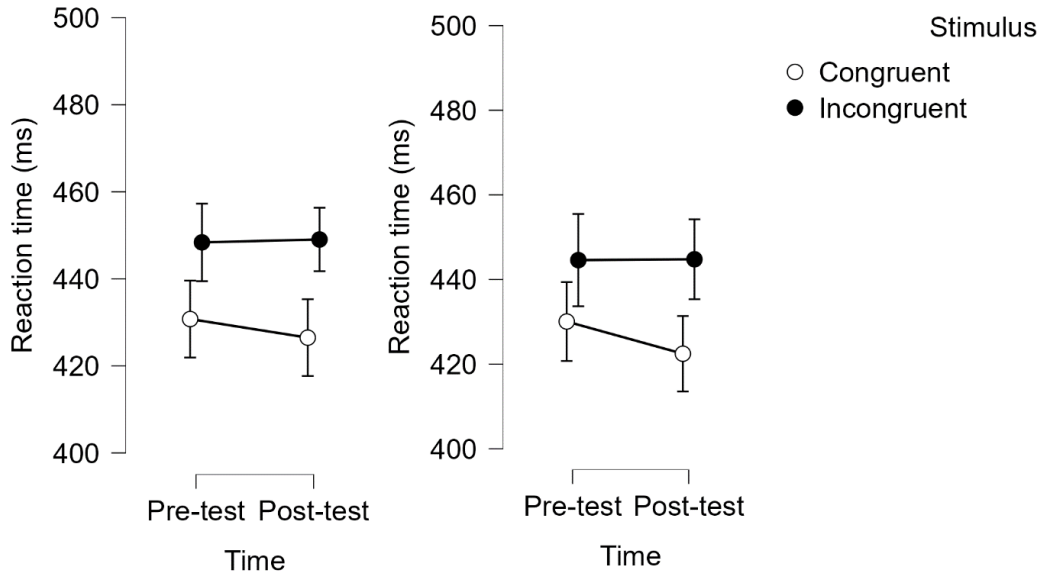

Figure 15: *Simon task reaction time data. Left graph Abdominal session and right graph Thoracic session.*

3.2.5. D-score correlations

See Appendix B Table 2 for the full matrix of both Dt1t2 and Dt2t3 scores. Seven of the possible correlations had a $BF_{10}>10$, all of which were equally uninformative as in Experiment 1, for example: correlations of RMSSD D-scores within a session. Also here, not a single correlation was found between respiration and RMSSD D-scores, violating expectation.

4. Discussion

For this study we set out to test predictions of the rVNS model (Gerritsen & Band, 2018) onto cognition; more specifically the indirect influence of the locus of breathing. It was predicted i) that abdominal as compared to thoracic breathing would lead to relatively more activity of the parasympathetic and less of the sympathetic nervous system, as reflected in HRV and PEP/SCL respectively; ii) that increased parasympathetic activity would be associated with more efficient inhibitory control, as reflected in the speed of response inhibition and the ability to resist Simon interference. Overviewing the results of both experiments, we can safely conclude that these predictions have not been met.

In Experiment 1, instructions emphasizing thoracic or abdominal breathing were not accompanied by a relative and divergent change in respiration rate. In all three conditions, including semi-control focused breathing, respiration rate went down: participants slowed down their breathing during any intervention. This violated the rationale behind the expectation that these manipulations could induce a parasympathetic dominated abdominal condition versus a sympathetically dominated thoracic condition. In our view, shifting the locus towards the abdomen would signal a more relaxed state, making respiration rate go down and this would lead to a higher RMSSD and thus cardiac vagal tone.

Then, with slow-paced breathing present in each condition, our expectation would be that RMSSD would go up during all interventions. Surprisingly, there was no main effect of condition on RMSSD. So, not only was the expected interaction effect on RMSSD

of *session*time* absent, cardiac vagal tone did not go up at all during any intervention, which would be expected from previous studies due to a slowing of breathing. Do note that, though respiration rate went down, it did not reach the golden rate of 6 breaths-per-minute (van Diest et al., 2014): both abdominal breathing and thoracic breathing went down to 9-10, while focused breathing went down to 11. Simply stated, breathing pace might have not gone down low enough to produce effects on cardiac vagal tone.

Similarly, sympathetic tone also showed a markedly different pattern than hypothesized. During abdominal breathing condition sympathetic tone went up (PEP decreased) instead of down from baseline levels, as compared to thoracic breathing and focused breathing. Though do note that, this was from a higher baseline level and PEP seemed to normalize during abdominal breathing (see Figure 5). Apparently, participants' stress levels were not comparable at the start of the three sessions, though the design was within-subjects and sessions counter-balanced in order. Perhaps, the participants expected abdominal breathing to be relaxing and showed anticipatory relaxation effects. But this was followed by a manipulation that is somewhat stressful itself, at least for beginners, that drove down PEP. Another possibility, is that manual picking of Q-point and R-point was systematically different and thus temporarily skewed for one session over the others, making these results an artefact. Either way, it is safest to not connect definitive conclusions to the PEP findings in Experiment 1.

The predicted differential effects on cognition were also not observed in the SST. This might be expected from the respiration and

cardiac vagal tone results. However, we might still expect differences in response inhibition or overall reaction time, as there are divergent effects on sympathetic tone. It has to be noted that a numerical trend in the predicted direction was observed for SSRT. The baseline measurement had the slowest response, followed by focused breathing, thoracic breathing; and lastly and most markedly faster: abdominal breathing. In other words, there is a slight suggestion that participants were a bit faster at stopping after the abdominal breathing intervention. A reason why the interaction model did not meet a sufficient evidence level (Bayesian factor above 3) might be that the sample was severely underpowered. Of the original sample, 10 of the 29 participants had to be excluded. Because they waited for the stop signal, regardless of repeated verbal instructions and visual prompts to not do so. Also, note that the results are difficult to interpret as there was only a baseline measurement at the first session.

In explaining the unpredicted pattern of results, there is no reason to question the compliance. The instructions aimed at manipulating the locus of breathing seem to have been followed, as Tho/Abd went down during abdominal breathing, while it remained the same during thoracic breathing. In Experiment 1, explicit instructions to change respiration rate were absent. However, the pace and rhythm of the (sparse) audio instruction were similar for all three conditions: a slower pace than in a conversation and in a relaxed tone of voice. Unconscious imitation of these non-verbal cues may have resulted in respiration rate going down across all breathing conditions in Experiment 1. To address abovementioned issues and to retest the predictions, Experiment 2 was set up.

The main change of design in Experiment 2 was in the interventions. The scripts for audio guidance were rewritten and added explicit instructions to contrast abdominal breathing condition and thoracic breathing condition in breathing pace. The focused breathing session was dropped. As the cognitive task, SST was replaced by the Simon task for multiple reasons. SSRT is not very sensitive to enhanced inhibition (Wöstmann et al., 2013). Furthermore, participants have to be excluded from the analysis if they deviate from the instructions aimed at approximately 50% stop success, as often happened in Experiment 1. Lastly, interference costs in Simon task are also viewed as a more valid measure of (cognitive) inhibition (Hommel, 2011). Another addition was the affect grid: a self-report measure of arousal and pleasure. As the final change, another physiological resting state measurement was introduced after the intervention (recovery), to test for wipe-out of possible RMSSD increases and thus transfer of a state of mental flexibility to the cognitive task.

However, Experiment 2 also did not show the predicted interaction effect on any of the main measures: RMSSD, SCL and Simon effect. Experiment 2 did pass the manipulation checks, so compliance was also not the issue here. As expected, Tho/Abd decreased during abdominal breathing intervention, meaning participants shifted their breathing towards their abdomen. Furthermore, respiration rate went up during thoracic breathing and down during abdominal breathing, then returning to baseline in recovery measurement. In other words, participants breathed at a slower pace during abdominal breathing and at a faster pace during thoracic breathing, than they did at baseline. However, similar to

Experiment 1, it has to be noted that mean respiration rate was not anywhere near the optimal rate of 6 breaths-per-minute (van Diest et al., 2014) during abdominal breathing intervention, but rather around 10 breaths-per-minute. Perhaps for this reason, cardiac vagal tone did not show any effect of respiration rate going down during abdominal breathing.

There seemed to be no arousal differences between sessions in Experiment 2, at least as picked up by SCL. Then again, inhalation duration increased relative to exhalation duration during both interventions, most markedly for thoracic breathing, and returned to baseline levels in recovery. The same pattern can be observed in heart rate. This is relevant, as respiration characterized by prolonged expiration triggers a relaxation response, while the reverse suppresses parasympathetic activity (Benson, 1975; Komori, 2018). Clearly, both interventions are cardiopulmonary taxing. This is not necessarily an indication of the scale of vagal involvement, but could hide enhancement effects that rely on relaxation. Interestingly, self-reported arousal on the affect grid goes down after both interventions. Taken together, it is hard to make definitive statements on the extent of sympathetic nervous system involvement, due to our manipulations. As a side note, one could debate on whether SCL is a valid indicator of sympathetic tone, or is only suited to monitor acute psychological stress and anxiety (Fowles, 1981, 1986). Arousal and stress are many-facetted phenomena, with different mechanisms, timing and dynamics. For example, a recent study that used ambulatory tools to measure physiological stress levels found a negative relationship between SCL and self-reported stress (Tutunji et

al., 2021). Here, high self-reported stress was associated with decreased SCL instead of the predicted reverse relationship.

The Simon task results did not provide stronger statistical support for our hypothesis than the SST, though the statistical power was higher. On the contrary, the numerically lower SSRT found in Experiment 1 after abdominal breathing, was not reflected in Experiment 2 by a smaller Simon effect. What does stand out is the accuracy trend in the thoracic breathing session (Figure 14). Accuracy on both congruent and incongruent trial types goes steeply down for thoracic breathing, from pre- to post-test. We suggest that this is due to the high variability in this condition. Though the increased cardiopulmonary stress during thoracic breathing intervention – as seen in inhalation/exhalation ratio ratio and heart rate – might also play a role. Overviewing the lack of cognitive effects we conclude that there is no evidence for cognitive enhancement of inhibitory control. Furthermore, on the basis of these null-results, we can also not conclude that Simon effect is a preferable inhibition enhancement measure over SSRT.

To further investigate the lack of finding a relationship between a respiration condition and cardiac vagal tone and look at the associations of the main physiological independents and mediators of the rVNS model, the D-score correlation matrixes were made for both experiments. This lead to the most striking of unexpected results: in neither experiment a correlation between respiration rate and RMSSD was found. A number of directions offer an explanation for this pattern of results.

Firstly, the interventions were short (~10 min). This duration may have been too short to induce an rVNS effect, reflected in cardiac vagal tone measures during intervention. Let alone a carry-over effect to recovery phase and the proposed transfer to the cognitive post-test phase. This issue of dose might be exacerbated by the inexperience of participants. The active duration of vagal stimulation might even have been briefer by sub-optimal adoption of respiratory patterns. As an example of a study with a longer duration: Laborde and colleagues (2019) employed a slow deep breathing exercise of 15 min., with 2 min. breaks every 5 min. They did find an increase in RMSSD and cognitive inhibition (Stroop interference). Though note that, the rVNS mechanism was absent in their analysis as well: RMSSD did not mediate the cognitive effects. On the other hand, the aforementioned study by You and colleagues (2021) showed no differences in RMSSD increase by the duration of breathing exercise, opposing this dose-response interpretation of null-findings. Their shortest intervention was just 5 min. and the longest 20 min. So if 5 min. of slow-paced breathing is enough to produce an increase in RMSSD, we would still expect an increase in cardiac vagal tone as respiration rate goes down, which we did not find.

Secondly, another explanation for the absence of association between respiration rate and RMSSD, might be that abdominal breathing did not bring breathing pace down sufficiently. Even though there was a response to the manipulation in abdominal breathing condition in Experiment 2 (respiration rate went down), the pace did not come close to reaching the optimal rate of 6 breaths-per-minute. Only six participants showed an respiration rate lower than 7, of which four have an respiration rate under 6. The optimal rate might be

a necessary condition for observing changes in RMSSD, on any timescale. Supporting this is that all the studies that were reported in the aforementioned systematic review that showed an increase in cardiac vagal tone, used a breathing exercise that went down to at least 6 breaths-per-minute (Zaccaro et al., 2018). This also applied to the trial with the different and ultra-short duration (You et al., 2021). Then again, if we assume this threshold exists: that the optimal rate has to be reached to observe a vagal response; then we would still expect some correlation between Dt1t2 scores of respiration rate and those of RMSSD. At the very least, for the individuals that do lower their pace towards the golden rate, we would expect the trends of respiration rate and RMSSD to covary. To observe this, we would need a larger sample.

Thirdly, we suggest that the association between respiration rate and RMSSD might still be present in our study, but remains hidden. This could be due to a parallel increase in sympathetic activity, which down-regulates parasympathetic tone. In other words, the respiration interventions, and especially AB, produce stress. An indication for this stressfulness is the increase in heart rate and relative inhalation duration during both interventions in Experiment 2. Do note that, there was no clear increase in sympathetic tone as picked up by either PEP or SCL in Experiment 1 and 2, respectively. Still, our findings do suggest that consciously controlling ones respiratory patterns produces some cardiopulmonary stress for the inexperienced. We propose that when participants are asked to manipulate their locus of breathing – and thus intentionally change muscle coordination to patterns that are underused in laymen, that have little experience in breathing exercises or meditation – this

produces sympathetic activity. This might especially apply to abdominal localized breathing, as intentionally using diaphragmatic and abdominal musculature is an unusual way to breathe. That thoracic breathing is the default setting of breathing can be seen in our data. In thoracic breathing sessions Abd/Tho does not change from baseline to intervention. In other words: the locus did not shift further towards the chest. The normal way of breathing is already dominated by thoracic (and diaphragmatic) musculature. The finding of a numerically faster SSRT after AB, might also fit this interpretation. An increase in arousal is accompanied by faster responses and shorter stopping reaction times. Summarizing our inferences: consciously manipulating respiratory musculature and especially changing its locus taxes cognitive resources and might produce psychological and physiological stress.

Fourthly, to explain the null-results on inhibitory control, we suggest that acute state enhancement effects on cognition might not be feasible. This short time scale might be insufficient to produce acute but linearly static effects on cognitive control. This is regardless of whether these potential effects are mediated by autonomous nervous system activity. Of course, if a phasic cardiac vagal tone increase is necessary to produce cognitive effects we would not observe them in these studies. So from our findings we can't conclude the reason for this null-result. There are studies finding no effects on cognition. For example, a recent high powered study on all three executive functioning components – shifting, updating and inhibition - found no enhancement on any, by a brief mindfulness meditation intervention (Baranski, 2021). We are certainly not suggesting that cognitive functioning does not dynamically fluctuate. Respiratory phase effects

on cognition are well-documented (Belli et al., 2021; Heck et al., 2019). Increase and decrease in performance that matches the sinus waveform of inspiration and expiration would likely not produce a bottom-line increase in cognition. Though testing whether relative increases of inhalation/exhalation phases could contribute to a linear increase in performance would be an interesting line of enquiry.

Fifthly, these null-results might be a symptom of the replication crisis. Failure to replicate (cognitive) effects might be because the conclusions of previous studies were based on type 1 errors. The failure to replicate previous findings might actually be increasing significantly. This will be due to more studies being conducted that aim to replicate and that more studies reporting null-findings are published. But also simply by the statistics that are widely used. An increasing amount of researchers are currently using Bayesian statistics, instead of the classical p-testing. A new wave of null-findings might be therefore be caused by stronger more robust and evidence-based statistics (Fucci et al., 2022). Clearly, this study is also using the Bayesian counterparts of classical statistical tests. However, many of the recent studies we have reported without null-findings are still using classical statistics (e.g. Laborde et al., 2021; You et al., 2021). This might be a reason for conflicting findings.

Lastly, both our experiments' samples are underpowered. There might be simply too little variance to show relationships between respiration, vagal tone and cognition.

Through all these limitations, it is too soon to conclude that the predictions of the rVNS model under study here, have been falsified. However, we do conclude that this study provides no evidence at all

that confirms rVNS predictions. Furthermore, we conclude that the selective study of the locus of breathing factor is not very promising. At least, when studied acutely with a short intervention.

Future studies should take into account the lessons learned from these two experiments. Firstly, if researchers want to study the influence of the locus component in breathing patterns or even just include a deep abdominal respiration style, we suggest including a biofeedback set-up. Either using a respiratory band (placed at the diaphragm) or an ECG for HRV feedback might make it easier to teach these complicated motor patterns to inexperienced participants. The tutorial should then also be lengthened, as biofeedback needs many training rounds. Indeed, a study using such a respiratory feedback set-up in a virtual reality environment has shown promise (Blum et al., 2020). Another recent case study on police officers in a zombie shooter also shows great potential (Michela et al., 2022). It studied the effects of slow-paced breathing biofeedback on decision making under stress. Participants being able to control their breathing patterns were superior in performance, due to decreased physiological stress.

Secondly, if the aim is to study phasic changes in autonomic functioning and their connection to cognition, we suggest to use longer interventions than 10 min. We suggest 15 min. and over, with breaks every 5 min, following Laborde et al. (2019). We believe a longer duration, or a higher dose, will produce changes in autonomic tone with higher fidelity, longer carry-over to subsequent phases and thereby will increase the chances of transfer to cognitive functioning.

Thirdly, we deem audio guidance unsuited as a manipulation of respiratory patterns. Even when the pace of instruction matches the intended respiration rate, there will be non-compliance and too much variation. This makes aiming at a given respiration rate, like the optimal rate of 6 breaths-per-minute, next to impossible. Stimuli that have the potential to more directly manipulate respiration rate and optionally inhalation/exhalation ratio are warranted. Simple two-dimensional visual cues will suffice. For example: a colored ball that expands and contracts, reflecting inhalation and exhalation respectively, can be used to visualize the respiratory goal parameters. When a biofeedback virtual reality design is used, stimuli can go far beyond that in visuals and dynamics. Added benefits of these stimuli is that even breath holding can be easily incorporated. Furthermore, potential confounds and noise, such as emotional reactivity to the audio guide's voice will be eliminated.

Fourthly, when testing for more enduring changes in the autonomic nervous system and cognitive enhancement, a longitudinal design with many intervention sessions is needed. We believe bringing about tonal changes has the most promise in producing the proposed beneficial effects of the rVNS model.

Fifthly, another option for progress in the respiratory cognition field is to eliminate the proposed mediator of rVNS entirely. We predict that without involvement of the vagal nerve complex, respiratory patterns should still produce phasic and tonic changes in autonomic functioning and thereby influence cognition. For this end we might simply substitute HRV with breath rate variability (Ashhad et al., 2022). The temporal dynamics of the respiratory system are like

the cardiac parameters a valid indicator of the flexibility and responsiveness of the organism at large, including behavior and neural state (Glass, 2001). Soni and Muniyandi (2019) made an attempt to produce a breath rate variability measure and were able to differentiate meditators from non-meditators by it. It was even superior to HRV on the short time scale.

Finally, another promising line of enquiry might be mapping individual respiratory patterns in different natural and ambulatory contexts. Through this an individualized respiratory parameter, like breath rate variability, can be extracted. Then it would be possible to offer personalized breathing exercises. Interventions can be adapted to a specific individual in a specific setting. For example: one person might systematically be on the low end of the arousal spectrum, which negatively affects their performance on a cognitively demanding task. This individual might actually benefit from an intervention that increases respiration rate, instead of lowering it to the one-size-fits-all optimal rate. Which in this case might actually have adverse effects instead; lowering the arousal state further. Clearly, we are still at the start of the study on the potential benefits of breathing.

Appendix A

Intervention instructions experiment 1:

Color coding: Visual (PC Monitor) Audio

General introduction

We gaan zo beginnen met een ademhalingsoefening die zal bestaan uit 2 onderdelen: een tutorial en de ademhalingsoefening zelf. Probeer de gehele ademhalingsoefening zo natuurlijk mogelijk te blijven ademhalen terwijl je de instructies volgt.

Je mag nu eerst de koptelefoon opzetten. De instructies zullen zo voortgezet worden via een audiobandje middels de koptelefoon. Volg de instructies zo zorgvuldig en nauwkeurig mogelijk.

Mocht er iets niet duidelijk zijn tijdens de tutorial laat dit dan aan het eind van de tutorial, dus voordat je begint aan de ademhalingsoefening weten aan de begeleider. Mocht je voor nu nog vragen hebben kun je deze aan de begeleider stellen.

Succes!

Introduction audio (1.5 min.)

Ga ontspannen, maar rechtop zitten, plaats beide voeten op de grond en je knieën op heup breedte of iets breder.

Probeer niet tegen de achterkant van de stoel te leunen.

Zit recht op, zo recht op mogelijk, en rest de palmen van je handen op je bovenbenen.

Breng nu je schouders langzaam omhoog richting je oren en laat ze daarna weer zakken.

Vergeet niet zo ontspannen en rechtop mogelijk te blijven zitten.

Plaats nu je linker hand zacht tegen je buik en je rechter hand tegen je borst.

Voel voor een moment hoe de buik en ribben naar binnen bewegen op een uitademing.

(8sec)

Voel nu ook voor een moment hoe de buik en ribben naar buiten bewegen op een inademing.

(8sec)

Plaats dan nu je handen terug op je bovenbenen.

Start of the tutorial round (2.5 min)

- *thoracic breathing*:

Welnu, plaats beide handen tegen de zijkant van je ribben ter hoogte van je borstbeen.

Met de volgende inademing, adem in via de zijkant van je ribben, zodat je voelt dat je handen en ribben naar de zijkant bewegen.

Wanneer je uitademt bewegen de ribben naar binnen, je voelt dat je handen en ribben naar binnen bewegen..

Als je inademt, adem volledig in, dus zorg dat je met elke ademhaling volledig in en volledig uit ademt met je borstkas.

Breng dan nu je handen van je ribben naar je bovenbenen, zit ontspannen en probeer zonder de feedback van je handen via de zijkant van je ribben te blijven ademen voor de volgende dertig seconden.

(30sec)

Uitstekend, dit was het einde van de oefening, probeer ontspannen te blijven zitten met beide voeten op de grond.

Wanneer je er klaar voor bent mag je op de spatiebalk klikken om de ademhalingsoefening voort te zetten. Dit keer zal je op dezelfde manier ademhalen maar voor een langere periode. Probeer de ademhaling niet te forceren, probeer zo natuurlijk mogelijk te blijven ademen terwijl je de instructies opvolgt.

- *AB*:

Welnu, plaats beide handen op je buik met je middelvingers ongeveer ter hoogte van je navel.

Met de volgende inademing, adem in via je buik, zodat je voelt dat je handen en buik naar voren bewegen.

Wanneer je uitademt trek je je buik en navel in, zodat je voelt dat je handen en buik naar binnen bewegen.

Als je inademt, adem volledig in, dus zorg dat je met elke ademhaling volledig in en volledig uit ademt met de buik.

Breng dan nu je handen van je buik naar je bovenbenen, zit ontspannen en probeer zonder de feedback van je handen via je buik te blijven ademen voor de volgende dertig seconden.

Uitstekend, dit was het einde van de oefening, probeer ontspannen te blijven zitten met beide voeten op de grond.

Wanneer je er klaar voor bent mag je op de spatiebalk klikken om de ademhalings-oefening voort te zetten. Dit keer zal je op dezelfde manier ademhalen maar voor een langere periode. Probeer de ademhaling niet te forceren, probeer zo natuurlijk mogelijk te blijven ademen terwijl je de instructies opvolgt.

- *focused breathing*:

Welnu, plaats je linker hand terug op je buik en je rechter hand op je borst.

Probeer bewust te worden van je inademing zonder er iets aan te willen veranderen, zodat je voelt dat de buik en/of ribben uitzetten.

Probeer daarnaast ook bewust te worden van de uitademing, zonder er iets aan te willen veranderen, zodat je voelt dat de buik en/of ribben naar binnen bewegen.

Zorg ervoor dat je elke in- en uitademing opmerkt, wanneer je merkt dat je afgeleid bent, probeer dan weer je gedachten naar je ademhaling te brengen.

Breng dan nu je handen van je buik en borst naar je bovenbenen, zit ontspannen en probeer zonder de feedback van je handen bewust te blijven van je ademhaling voor de volgende dertig seconden.

Uitstekend, dit was het einde van de oefening, probeer ontspannen te blijven zitten met beide voeten op de grond.

Wanneer je er klaar voor bent mag je op de spatiebalk klikken om de ademhalings-oefening voort te zetten. Dit keer zal je op dezelfde manier ademhalen maar voor een langere periode. Probeer de ademhaling niet te forceren, probeer zo natuurlijk mogelijk te blijven ademen terwijl je de instructies opvolgt.

Breathing exercise

- *TB*:

Welnu, ga opnieuw ontspannen zitten, met een rechte rug en je handen rustend op je bovenbenen.

Probeer nu zo goed mogelijk adem te halen via de zijkant van je ribben, in je eigen tempo voor de volgende 5 minuten.

30 (seconden)

Blijf ademhalen via de zijkant van je ribben.

30 (seconden)

Probeer recht op te blijven zitten terwijl je in- en uitademt via de zijkant van je borstkas.

(1 minute...)

Ga zo door, blijf zo goed mogelijk via de zijkant van je borstkas in- en uitademen, blijf te allen tijde ontspannen.

(1 minute...)

Vergeet niet volledig in- en uit- te blijven ademen met de zijkant van je ribben.

(1 minute...)

Nog 1 minuut te gaan, blijf ademen via de zijkant van je ribben.

(1 minute...)

Uitstekend, dit is het einde van de ademhalingsoefening, blijf rustig zitten met beide voeten op de grond.

Wanneer je er klaar voor bent, klik op de spatiebalk om door te gaan naar de volgende opdracht.

- *AB*:

Welnu, ga opnieuw ontspannen zitten, met een rechte rug en je handen rustend op je bovenbenen.

Probeer nu zo goed mogelijk adem te halen via je buik, in je eigen tempo voor de volgende 5 minuten.

(30 seconds...)

Blijf ademhalen via je buik.

(30 seconds...)

Probeer recht op te blijven zitten terwijl je in- en uitademt via de buik.

(1 minute...)

Ga zo door, blijf zo goed mogelijk via de buik in- en uitademen, blijf te allen tijde ontspannen.

(1 minute...)

Vergeet niet volledig in- en uit- te blijven ademen via de buik.

(1 minute...)

Nog 1 minuut te gaan, blijf ademen via de buik.

(1 minute...)

Uitstekend, dit is het einde van de ademhalingsoefening, blijf rustig zitten met beide voeten op de grond.

Wanneer je er klaar voor bent, klik op de spatiebalk om door te gaan naar de volgende opdracht.

- *focused breathing*:

Welnu, ga opnieuw ontspannen zitten, met een rechte rug en je handen rustend op je bovenbenen.

Probeer nu zo goed mogelijk je ademhaling te volgen, zorg er voor dat je bewust bent van elke in- en uitademing zonder er iets aan te willen veranderen, voor de volgende 5 minuten.

(30 seconds...)

Blijf met je gedachten bij je ademhaling.

(30 seconds...)

Probeer recht op te blijven zitten terwijl je je bewust bent van je ademhaling.

(1 minute...)

Welnu, als je gedachte afgedwaald waren, breng je aandacht opnieuw naar de ademhaling.

(1 minute...)

Vergeet niet bewust te zijn van je ademhaling, zonder hier iets aan te willen veranderen.

(1 minute...)

Nog 1 minuut te gaan, blijf met je gedachten bij de ademhaling.

(1 minute...)

Uitstekend, dit is het einde van de ademhalingsoefening, blijf rustig zitten met beide voeten op de grond.

Wanneer je er klaar voor bent, klik op de spatiebalk om door te gaan naar de volgende opdracht.

Appendix B

Table 2: *Correlation matrix for main physiological variables of experiment 2. AB=abdominal breathing session; TB=thoracic breathing session; Dt1t2=difference score variable between: measurement time 2 (intervention) - measurement time 1 (baseline); Dt2t3=difference score variable between: measurement time 3 (recovery) - measurement time 2 (intervention); BMP=breaths-per-minute; I/E ratio = inhalation duration (ms) / exhalation duration (ms); RMSSD= root mean square of successive differences; SCL=skin conductance level.*

Variable		AB Dt1t2 BMP	AB Dt1t2 I/E	AB Dt1t2 RMSSD	AB Dt1t2 SCL	AB Dt2t3 BMP	AB Dt2t3 I/E	AB Dt2t3 RMSSD	AB Dt2t3 SCL	TB Dt1t2 BMP	TB Dt1t2 I/E	TB Dt1t2 RMSSD	TB Dt1t2 SCL	TB Dt2t3 BMP	TB Dt2t3 I/E	TB Dt2t3 RMSSD	TB Dt2t3 SCL
AB Dt1t2 BMP	Pearson's r	—															
	BF_{10}	—															
AB Dt1t2 I/E	Pearson's r	0.12	—														
	BF_{10}	0.26	—														
AB Dt1t2 RMSSD	Pearson's r	-0.09	0.01	—													
	BF_{10}	0.23	0.20	—													
AB Dt1t2 SCL	Pearson's r	0.01	0.18	-0.18	—												
	BF_{10}	0.20	0.36	0.35	—												
AB Dt2t3 BMP	Pearson's r	-0.49 *	0.00	0.09	0.04	—											
	BF_{10}	23.52	0.20	0.23	0.21	—											
AB Dt2t3 I/E	Pearson's r	-0.09	-0.30	-0.03	-0.22	0.01	—										
	BF_{10}	0.23	0.96	0.21	0.46	0.20	—										
AB Dt2t3 RMSSD	Pearson's r	0.28	-0.25	-0.60 ***	-0.03	-0.28	0.21	—									
	BF_{10}	0.81	0.61	455.16	0.21	0.82	0.44	—									
AB Dt2t3 SCL	Pearson's r	0.10	0.28	-0.46 *	0.39	0.05	-0.23	0.15	—								
	BF_{10}	0.24	0.79	12.80	3.55	0.21	0.50	0.29	—								
TB Dt1t2 BMP	Pearson's r	-0.01	-0.09	0.05	-0.13	0.10	0.20	-0.22	0.01	—							
	BF_{10}	0.20	0.23	0.21	0.27	0.24	0.40	0.49	0.20	—							
TB Dt1t2 I/E	Pearson's r	0.17	0.05	-0.18	-0.03	-0.12	-0.03	-0.12	0.18	0.47 *	—						
	BF_{10}	0.33	0.21	0.36	0.21	0.26	0.21	0.26	0.34	15.27	—						
TB Dt1t2 RMSSD	Pearson's r	-0.15	0.00	0.36	-0.24	0.08	-0.23	-0.20	-0.26	-0.34	-0.16	—					
	BF_{10}	0.30	0.20	2.24	0.57	0.23	0.50	0.39	0.69	1.75	0.32	—					
TB Dt1t2 SCL	Pearson's r	-0.07	0.01	-0.17	0.34	0.03	-0.07	0.02	0.18	0.18	-0.09	-0.29	—				
	BF_{10}	0.22	0.20	0.33	1.62	0.21	0.22	0.20	0.36	0.34	0.23	0.95	—				
TB Dt2t3 BMP	Pearson's r	0.15	0.18	0.02	0.01	-0.14	-0.19	0.24	-0.08	-0.93 ***	-0.53 **	0.28	-0.17	—			
	BF_{10}	0.30	0.37	0.20	0.20	0.28	0.37	0.56	0.23	1.24*e14	51.18	0.78	0.32	—			
TB Dt2t3 I/E	Pearson's r	0.01	0.41	0.11	0.03	-0.01	0.11	-0.09	0.00	-0.13	-0.43	-0.06	0.02	0.29	—		
	BF_{10}	0.20	4.97	0.25	0.21	0.20	0.25	0.23	0.20	0.27	6.83	0.22	0.20	0.93	—		
TB Dt2t3 RMSSD	Pearson's r	0.14	-0.29	-0.41	0.30	0.00	-0.01	0.25	0.41	0.36	0.19	-0.57 ***	0.25	-0.43	-0.26	—	
	BF_{10}	0.28	0.91	4.99	0.96	0.20	0.20	0.63	5.02	2.05	0.38	148.19	0.58	7.13	0.69	—	
TB Dt2t3 SCL	Pearson's r	-0.01	0.21	0.10	0.28	0.03	-0.17	-0.08	0.23	-0.36	-0.26	-0.05	-0.20	0.39	0.24	-0.03	—
	BF_{10}	0.20	0.43	0.24	0.85	0.21	0.33	0.23	0.51	2.13	0.67	0.21	0.40	3.54	0.57	0.20	—

* $BF_{10} > 10$, ** $BF_{10} > 30$, *** $BF_{10} > 100$