

# VibroAffect: The Underlying Affect Regulation Mechanism of a Vibrotactile Breathing Pacer

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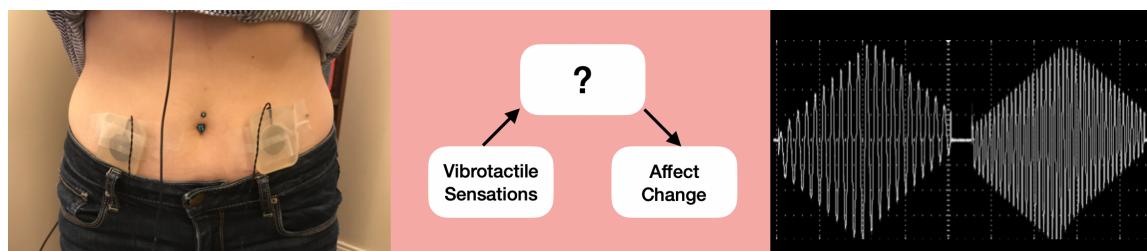


Fig. 1. Two tactors, attached to the abdomen (left), deliver personalized paced vibrations (right) with which participants can synchronize their breathing. One of the goals of this paper is to investigate the link between these patterns of vibrations and the change in two dependent variables of skin conductance and anxiety mediated by coarse measures of breathing (center).

Vibrotactile devices that assist in affect regulation are becoming more common, but the underlying mechanisms that link vibrotactile sensations to affect are not well understood. We investigate how a repetitive pattern of vibrations designed to guide users through slow-paced breathing impacted breathing rate and skin conductance. Even though participants could not fully shift their breathing to match the pacer rate while performing cognitive stressor tasks, their attempts led to a slower rate of breathing, greater breathing irregularity, and greater chest-to-abdomen displacement ratio compared to the control group. However, these changes were not related to changes in skin conductance or self-reported anxiety. This paper clarifies the link between vibrotactile sensation and psychophysiology and offers guidelines for evaluating other affect regulation technologies.

CCS Concepts: • Human-centered computing → Human computer interaction (HCI); Haptic devices; User studies; Empirical studies in HCI; • Computer systems organization → Sensors and actuators; • Hardware → PCB design and layout; Sensors and actuators.

Additional Key Words and Phrases: Haptic, Vibrotactile, Anxiety, Affect Regulation, Affect, Respiration, Slow-paced breathing, Pacer, Wearable, Linear Mixed Model, Skin conductance, Mediation, Empirical Mode Decomposition, Fast Fourier Transform

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## 1 INTRODUCTION

Affect regulation refers to things that we do (cognitively or behaviorally) to alter affective states that we judge to be unwanted or context-inappropriate [20]. Examples of affect regulation strategies include practicing slow-paced breathing, reappraisal<sup>1</sup>, distracting oneself, smoking, or taking a nap when one is experiencing anxiety, with some strategies being more adaptive than others for a variety of reasons (e.g., slow-paced breathing vs. smoking). Difficulties in affect regulation can negatively impact well-being and the ability to function in life.

There is considerable interest in using technology to help with affect regulation. In particular, vibrotactile technologies have been developed to increase our capacity to perform affect regulation in stressful situations [2, 4–8, 12, 14–17, 19, 28, 31, 32]. For example, to reduce anxiety, PIV [28] applies slow breathing-like vibrations to the abdomen, while Doppel [8], EmotionCheck [16], and ambienBeat [15] apply slow heartbeat-like vibrations to the wrist.

The underlying mechanisms that link vibrotactile sensations to affect are not well understood, partly because investigating the causal relationship of vibration pattern → affect regulation strategy → affect can be challenging when the link between patterns of vibrations and an affect regulation strategy is not well established. In this paper, we hypothesize that repetitive patterns of vibrations designed to guide users through slow-paced breathing will help in decreasing their breathing rate, which will then result in reduction in physiological (i.e., skin conductance) and subjective (i.e., self-reported) measures of anxiety. In this scenario, the link between the vibrotactile intervention and changes in breathing can be evaluated by measuring the breathing rate. However, in the case of repetitive heart-beat like patterns of vibrations, it is unclear from the literature what affect regulation strategy mediated a drop in self-reported measure of anxiety, skin conductance, or heart rate variability [8, 14–17, 31]. Is it reappraisal, placebo, a combination of multiple affect regulation strategies, or something else? Answering these questions is challenging because reliance on self-reported measures of usage and success rate of known affect regulation strategies may not explain the impact of vibrations on affect. Participants may not even have noticed what affect regulation strategy they were deploying during the stressful situation.

Miri et al. [28] have built a vibrotactile prototype that directly links vibrations to a known form of affect regulation (i.e., slow-paced breathing). This design facilitates studying the prototype's underlying regulatory mechanism. They showed a link between their vibration patterns and a drop in self-reported measure of anxiety (STAI-6 [26]) through a mixed-design experiment in which treatment and control group went through two repeated stressor blocks. The vibrotactile breathing pacer was activated only for the treatment group and only during the second stressor block. This design allowed us to test the effectiveness of the pacer while controlling for individual differences as well as habituation with the stressor. The stressor blocks consist of a stressor condition, called Stressor 1 or 2,<sup>2</sup> and two waiting periods that occur before and after each stressor. These waiting periods, called Pre-stressor and Post-stressor, allowed time to pass in-between stressors so that anxiety from the stressor would not affect measurements taken at other stages.

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<sup>1</sup>Cognitive reframing of an emotional event so as to have a different emotional meaning.

<sup>2</sup>Keep in mind the first stressor had no intervention while the treatment group received intervention during the second stressor condition.

We aimed to extend their prior work by identifying the effect of Miri et al.'s [28] pacer on psychophysiological measures collected in their study. We will also investigate the causal links between personalized patterns of vibrations and the two independent variables of skin conductance and self-reported anxiety, separately mediated through slow-paced breathing. We expected to observe that the repetitive patterns would guide users to inhale with one pattern and exhale with another so that they could shift their breathing, in a way that induces a calming effect in a stressful situation, as measured through skin conductance and self-reported measure of anxiety (STAI-6). We also expected to observe less dysfunctional breathing when the pacer was active as compared to when it was not. This is because slow, deep, and diaphragmatic breaths are associated with beneficial breathing chemistry while dysfunctional breathing (e.g., breath holding, deep but fast breathing or fast and shallow breaths) is linked to negative affective states such as anxiety [23].

In this paper, we will first discuss four confirmatory analyses testing the effect of the pacer in reducing breathing rate, breathing irregularity, the ratio of chest to abdominal displacement, and skin conductance. We then address two exploratory analyses to understand how breathing rate in the treatment group explains skin conductance and self-reported measure of anxiety (i.e., STAI-6). Miri et al. [28] reported an effect of the pacer in reducing STAI-6, and so we asked whether this effect was mediated by breathing rate. Additionally, if we observe an effect of the pacer in reducing skin conductance during our confirmatory analysis, we can then gain insight into this effect by testing whether it was mediated by breathing rate. These exploratory analyses could shed more light on the underlying affect regulation mechanism of the pacer.

The four confirmatory pre-registered hypotheses are as follows:

1. *Efficacy of pacer in reducing breathing rate.* We hypothesized an interaction effect between group (treatment and control) and condition (Stressor 1 and Stressor 2) for the dependent variable of estimated breathing rate. We expected to observe no change from Stressor 1 to Stressor 2 in the control group, but a lower breathing rate in the treatment group during stressor 2 when the pacer was active, which would indicate that participants were at least somewhat successful in following the pacer, despite being cognitively engaged performing stressful tasks.

2. *Efficacy of pacer in reducing breathing irregularity.* We hypothesized an interaction effect between group (treatment and control) and condition (Stressor 1 and Stressor 2) for the dependent variable of breathing irregularity. Specifically, we expected to observe less breathing irregularities from Stressor 1 to Stressor 2 in the treatment group, but no change in the control group. We thought that the pacer would contribute to active breathing (more regular breathing) while participants were performing stressor tasks.

3. *Efficacy of pacer in reducing chest-to-abdomen displacement ratio.* We hypothesized an interaction effect between group (treatment and control) and condition (Stressor 1 and Stressor 2) for the dependent variable of chest-to-abdomen displacement ratio. Specifically, we expected to observe no change in the chest-to-abdomen displacement ratio in the control group, but more abdominal breathing in the treatment group during stressor 2 when the pacer was active. We hypothesized more abdominal breathing when the pacer was active for two reasons: early in the protocol,<sup>3</sup> participants practiced abdominal breathing; and the factors deployed patterns of vibrations on the abdomen area which could engage the tendency of more abdominal slow-paced breathing [13, 29].

4. *Efficacy of pacer in reducing skin conductance.* We hypothesized an interaction effect between group (treatment and control) and condition (Stressor 1 and Stressor 2) for the dependent variable of skin conductance. We expected to observe no change in the control group, but a lower skin conductance level in the treatment group, from Stressor 1 to

<sup>3</sup>See details of the experimental design in the protocol section.

Stressor 2. This is because we expected that slow-paced breathing deployed by the treatment group in the Stressor 2 condition when the pacer was active would be more effective in reducing skin conductance compared to other forms of affect regulation they may have deployed in Stressor 1.

The two exploratory hypotheses are as follows:

5. *Indirect effect of pacer in reducing anxiety through breathing rate.* After observing the effect of the pacer on dropping breathing rate (confirmation of hypothesis 1), we hypothesized that the low breathing rate in the treatment group could explain the change (in this case drop) in self-reported measure of anxiety (STAI-6) from Stressor 1 to Stressor 2.

6. *Indirect effect of pacer in reducing skin conductance through breathing rate.* After observing the effect of pacer on dropping skin conductance (confirmation of hypothesis 4), we hypothesized that the low breathing rate in the treatment group could explain the change (in this case drop) in skin conductance from Stressor 1 to Stressor 2.

The specific contributions of this paper are as follows:

- We report the results of a mixed-design pre-registered experiment to evaluate the effect of a breathing pacer on psychophysiology measures.
- We report the role of breathing rate in changing affect via a set of repetitive patterns of vibrations produced by a breathing pacer.
- We report a method to remove respiration artifacts and derive accurate estimates of breathing rate and irregularity as well as the ratio of chest-to-abdomen displacement ratio.

Our findings and methods of analysis may be of benefit to others in the HCI community who are designing devices for affect regulation and are investigating the link between vibrotactile patterns and affect as measured through both psychophysiology and self-report measures.

## 2 RELATED WORK

Compared to other studies evaluating vibrotactile technologies for affect regulation, this study measured a greater range of psychophysiology indicators, which allowed us to assess more comprehensively the mechanism of the breathing pacer on affect change. Besides the pacer, several other devices have been studied that use vibrotactile technology for affect regulation. These include: Doppel [8], BoostMeUp [17], and ambienBeat [15], all three of which provide heartbeat-like stimulation on the wrist; Spire Stone [32], which monitors users' respiration and delivers interventions through an app; and Just Breathe [31], Breath Booster [9], a series of in-car haptic and audio interventions for use while driving. Of the studies used to evaluate the other vibrotactile technologies mentioned, only two analyzed skin conductance as a measure of anxiety (Doppel, Just Breathe). In addition, only one analyzed respiratory data in any form (Just Breathe). Finally, none of these studies analyzed breathing regularity or chest-to-abdomen displacement ratio, both of which are known to have a link to change in affect [23, 34]. Our study used the measured skin conductance and breathing data, and also derived measures of breathing rate and regularity, as well as chest-to-abdomen displacement ratio. We were able to analyze the effect of using the breathing pacer on each measure. Based on our findings, we were also able to develop and analyze an explanatory hypothesis about the affect regulation mechanism of the breathing pacer.

## 3 METHODS

In this section, we briefly describe the study design carried on by Miri et al. An extensive explanation can be found in [28].

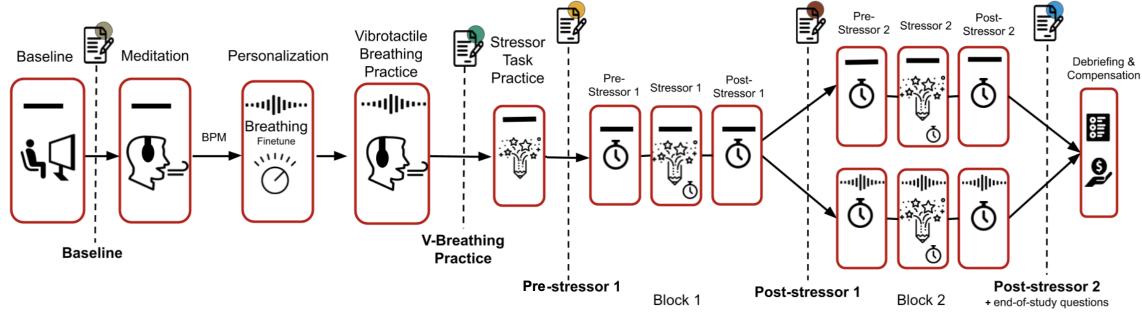


Fig. 2. Procedure flowchart of the study. All participants went through the same procedure until end of Post-stressor 1. At that point, participants who were randomly assigned to the control group went through Block 2 while receiving no vibrations (indicated by a solid black line), while participants who were randomly assigned to the treatment group go through Block 2 while receiving vibrations. Subjective measures of anxiety (i.e., STAI-6) were collected at five conditions of Baseline, V-Breathing Practice, Pre-stressor 1, Post-stressor 1, and Post-stressor 2 (indicated by a color-coded pen-and-paper icon). Physiological (i.e., skin conductance and respiration) measures were collected during the entire procedure. [28]

### 3.1 Participants

Participants were recruited via a university pool of students with the incentive of either course credit or hourly payment for their time. Out of the total of 100 volunteers, 3 had to be excluded due to procedural errors. 44 participants (29 female, 15 male) were randomly assigned to the treatment group and 53 (32 female, 21 male) to the control group.

### 3.2 Protocol

Before starting the study, participants placed a few devices on different locations of their body. Based on the recommendations from [27], each participant had two tactors taped to their abdomen. Other retrofittings included breathing gauges around the chest and abdomen and EDA sensor placed on their non-dominant hand. Participants were given noise-cancelling headphones to aid in blocking out any potential vibration sounds.<sup>4</sup>

The study consisted of the following stages: Baseline, Meditation, Personalization, Vibrotactile Breathing Practice, Stressor Task Practice, Block 1, and Block 2 (see Figure 2). All participants followed the same procedure until the end of Block 1, at which point the treatment group received the breathing pacer intervention while the control group did not. Below, we summarize the stages of the protocol and describe the stressor task used in Blocks 1 and 2.<sup>5</sup>

In the Baseline stage, participants watched a 5-minute video of a pipe changing colors and were asked to count how many times the pipe turned green; the objective of this stage was to obtain baseline physiology measurements. In the Meditation stage, participants listened to a 5-minute audio guiding them through slow-paced abdominal breathing techniques. The objective of this stage was twofold: to train participants to prepare them for synchronous breathing with the vibrations, and to estimate their slow-paced respiratory rate in breaths per minute.

In the Personalization stage, participants established a pace, frequency, and amplitude of vibrations that was personalized to the participant. Research assistants worked with participants to arrive at appropriate personalized frequencies

<sup>4</sup>C2 tactors used in this study can make audible noise in a quiet lab environment when vibrating at frequencies over 165Hz [1] without noise canceling headphones. In the personalization procedure C2 tactors were not driven beyond 180Hz which is non-audible when noise canceling headphones are in use.

<sup>5</sup>For more detailed information about the protocol and the stressor task, see the “Methods” section of Miri et al. [28]

and amplitudes. In the Vibrotactile Breathing (V-Breathing) Practice stage, participants practiced synchronizing their breathing with the personalized vibrations for 90 seconds.

In the Stressor Task Practice stage, participants were introduced to the compound remote associate task (CRA) to be used as a stressor. Compound remote associate questions consist of three English words whose answer is a fourth word that is associated with all of them. For example, the answer to the CRA question “man/glue/star” would be “super.” The specific CRA questions were taken from Bowden & Jung-Beeman [11]. Block 1 consisted of the first stressor and two waiting periods occurring before and after the stressor. Waiting periods, called Pre-stressor 1 and Post-stressor 1, allowed two minutes to pass between stressors so that any anxiety from the stressor would not affect measurements taken at other stages. The stressor task was 4 minutes long and consisted of 27 CRA questions with a time limit of 9 seconds each. Running out of time on a question was equivalent to an incorrect answer. If a question was answered correctly, participants would still have to wait the full 9 seconds before seeing the next question; this was done to ensure that all participants spent an equal amount of time in the stressor task. During Block 2, which was otherwise identical to Block 1, the treatment group received ongoing personalized vibrations and were instructed to conform their breathing with the sensation of the pacer.

## 4 MEASURES

### 4.1 Respiration Measures

Respiratory measures include breathing rate (number of breaths per minute), breathing irregularity (coefficient of variance), and ratio of abdomen to chest breathing (slope of a regression line fitted over a Konno-Mead [24, 33] plot) in a response window. Before extracting these measurements of interest, we used an empirical mode decomposition (EMD) based signal processing technique, inspired by Liu et al. [25], to remove tissue artifacts (e.g., any muscle movement), posture change, and background noise, from respiratory signals. In addition, we applied a smoothing technique using a rolling window of  $\frac{2}{3} \times$  sampling frequency (in our case 256 Hz). Below, we describe our approach to artifact removal in more detail.

**4.1.1 Artifact Removal of Respiration Signal.** Empirical Mode Decomposition (EMD) [21] decomposes a time-varying signal into a sum of finite components, referred to as intrinsic mode functions (IMFs), each of which represents an oscillatory mode (see Figure 3). A critical step in achieving an artifact-free signal is selecting the appropriate IMFs that contribute to the signal and excluding those IMFs that mainly contribute to the contamination of the signal. In the context of a respiration signal derived from ribcage or abdomen displacement, tissue artifacts and background noise are usually present in higher frequency bands (i.e., several first IMFs) while lower frequency bands (i.e., several last IMFs) often correspond to posture changes. By removing these high and low frequency bands corresponding to such artifacts, an artifact-free respiratory signal can be reconstructed by summing up the remaining IMFs.

Several researchers have explored applying the EMD algorithm directly to the respiration signal. Karagiannis et al. [22] proposed an EMD-based technique on experimental respiration signals derived from an accelerometer X and Y axis. Their technique conducted denoising by removing several IMFs in higher bands for a partial respiration signal reconstruction. Gan et al. [18] evaluated a contactless respiration rate measurement using an optical displacement sensor which utilized an EMD based approach by removing several very high and very low frequencies. Liu et al. [25] developed an EMD based algorithm to remove tissue artifacts from respiration signals. They identified an appropriate cutoff threshold to remove several first IMFs using IMFs detected power and the mutual information between each IMF and the input signal.

Among past applications of EMD to breathing artifact removal, Liu et al.'s work was most closely applicable to our data, but when we applied their technique, it resulted in some data that could not be used to extract breathing measurements (see Figure 4). Rather than using any of the three EMD-based techniques, we found that a more generic approach suitable for our dataset was to statically remove the first two IMFs (which always correspond to tissue artifacts) and the last IMF (which are always associated with the participant's posture change). This resulted in less artifact removal, but still good enough to estimate breathing rate using Fast Fourier Transform (FFT). We found that the removal of the lowest detected IMF was more important than the first two highest detected IMFs in measuring breathing rates, as described next.

**4.1.2 Breathing Rate.** A simple approach to measure breathing rate is to identify and count all peaks in the respiration signal and divide the count by the duration of the signal. This approach can be flawed when the respiration signal is not free from artifacts, but is often used because other methods present difficulties. For example, while using a FFT is promising for determining the dominant frequency, outliers in a respiration signal, commonly formed due to posture change or change from active to passive breathing, result in the FFT to return a value close to zero as the dominant frequency (i.e., respiration rate), which is invalid.

To more accurately identify dominant frequencies of a respiration signal, we attempted to remove artifacts as much as possible first, using the generic EMD approach described above, and then identified outlier peaks and troughs as those which were more than three standard deviations away from the mean value of the peaks or valleys.<sup>6</sup> We then split the data into the segments between the outliers and ran FFT on each segment to retrieve the dominant frequency

<sup>6</sup>To extract the peaks related information, we used Python `find_peaks` with the following settings: `find_peaks(signal, distance = 2 * sampling_frequency, width = .8 * sampling_frequency, prominence = .3)`.

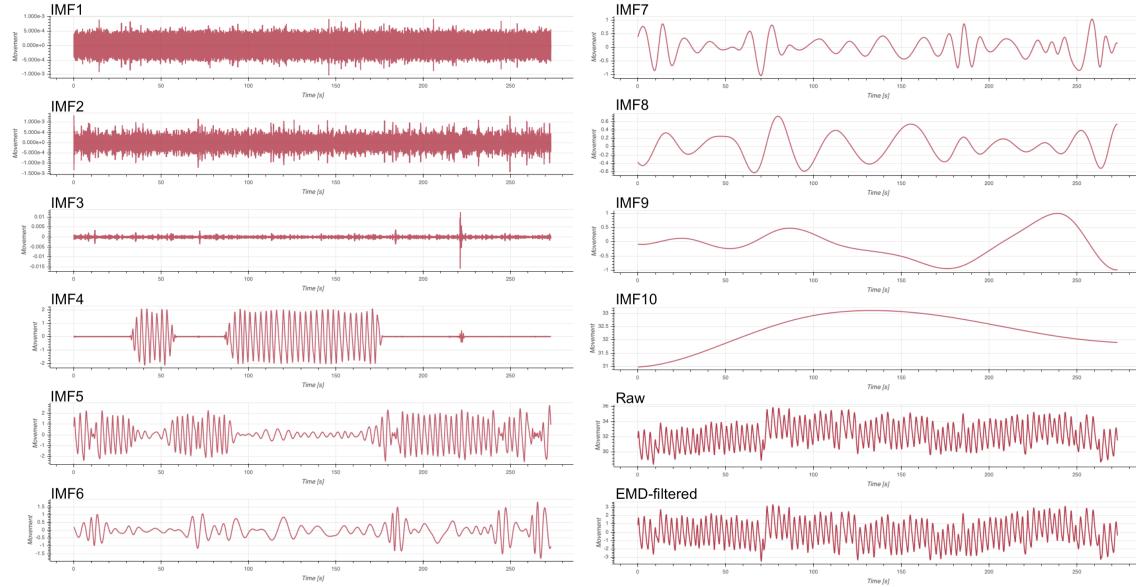


Fig. 3. An example of empirical mode decomposition (EMD) approach to decompose a respiratory signal into nine intrinsic mode functions (IMFs) components. We removed the first two and the last IMF to filter tissue artifacts (e.g., any muscle movement), posture change, and background noise from the respiratory signal.

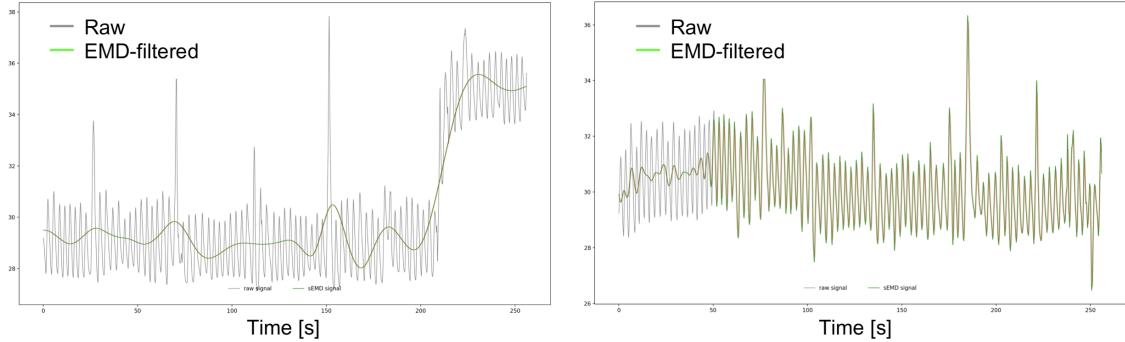


Fig. 4. Examples of Liu et al.’s method [25] on two respiratory signals (green) which resulted in unusable data, overlaid by the raw signal (gray). Note, we cannot extract correct breathing measurements such as breathing rate from the filtered data.

for that segment. If no outlier was detected, we ran FFT on the entire outlier-free segment. We then computed the average of the frequencies to measure the breathing rate. However, this approach was still not fully robust, as there were several segments in which the FFT returned invalid values as the dominant frequency, which indicated that the cutoff of 3 standard deviations for outlier identification needed to be further restricted.

We ultimately chose a simple and robust approach for measuring breathing rates. We first removed artifacts as much as possible, using the generic EMD approach described above, and then computed a set of breathing rate averages using a rolling window of 5 breaths, and returned the median of this set as the dominant breathing frequency. The rolling window size of 5 was chosen because we failed to find a significant difference between what this algorithm computes versus what the FFT-based approach computes for segments where it returned a valid result.

**4.1.3 Breathing Irregularity.** To measure the irregularity in breathing depth, we computed the coefficient of variance (CV) of the respiration signals, which is the standard deviation expressed as a percentage of a signal mean (i.e.,  $\frac{SD}{Mean} \times 100$ ). A higher CV corresponds to more irregularity in the depth of breathing.

**4.1.4 Chest to Abdomen Breathing Ratio.** To measure the ratio of chest-to-abdomen displacement, we examined ribcage-abdominal motion using the method of Konno and Mead [24, 33], where the extensions of the abdomen and rib cage are displayed on an XY plot. There is no axis corresponding to time on a Konno-Mead plot; instead, the breathing waves together generate a trace on the XY plot. When the chest and abdomen move relatively in synchronization, the trace is a figure that slopes upwards and to the right. We then fit in an ordinary least squares (OLS) regression line which is a measure of the ratio of abdomen to chest motion: slopes less than 1 result from more abdominal compared to chest breathing. Figure 5 shows Konno-Mead plots for participant subject s021 during Stressor 1 and Stressor 2. This participant was in the treatment group and so received the pacer intervention during Stressor 2. The figure shows more abdominal breathing during Stressor 2 as compared to Stressor 1.

## 4.2 Skin Conductance

The mapping between observed skin conductance (SC) and sympathetic arousal (SA) is known as skin conductance analysis. There are two general approaches for such analysis: a model-based approach which uses mathematical models to map between observed SC and SA, and a model-free approach, such as the average of an observed SC within a certain

time frame. Ledalab [10] is a widely used package that infers SA from observed SC. We used two model-based measures produced by Ledalab: CDA.nSCR, which is the total number of significant peaks in the phasic (fast-changing) drive, and CDA.SCR, which is the average phasic driver value in a response window.<sup>7</sup> We also used SC.mean, which is the averaged observed skin conductance model-free approach over a response window. This value is computed using a model-free approach. See the Appendix section for details on how the LedaLab package computes CDA.nSCR and CDA.SCR.

#### 4.3 Anxiety and Affect Regulation

To measure anxiety, Miri et al. collected responses to the State-Trait Anxiety Inventory (STAI-6) at the five conditions of Baseline, V-Breathing Practice, Pre-stressor 1, Post-stressor 1, and Post-stressor 2 (indicated by a color-coded pen-and-paper icon in Figure 2). STAI-6 is a brief six-item version of the State-Trait Anxiety Inventory (STAI). Using STAI-6 instead of STAI-20 enables collecting anxiety measures more frequently, while minimizing participant burden. STAI-6 scores correlate highly with scores on the full-length STAI-20 ( $r = 0.95$ ). [26]

### 5 RESULTS AND DISCUSSION

For all confirmatory analyses, we ran linear mixed models to test the interaction effect between group (treatment and control) and condition (Stressor 1 and Stressor 2) on the dependent measures while controlling for stable individual differences. Models were implemented in R with the lme4 package according to the following specifications: `lmer(DV ~ condition * group + (1 | id))`. The effect confidence intervals were bootstrapped.

#### 5.1 Confirmatory Analysis Results and Discussion

We ran linear mixed models on three measures of breathing rate, breathing irregularity (coefficient of variance), and ratio of abdomen to chest displacement (slope of a regression line fitted over Konno-Mead plot) and found interaction effects

<sup>7</sup>For replicability purposes, here is a sample invocation of the Ledalab script with a skin conductance sampling rate of 256 Hz: `Ledalab('path_to_data', 'open', 'text', 'downsample', +2, 'analyze', 'CDA', 'optimize', 2, 'export_era', [6 240 .05 1])`. The response window is 6 to 240 seconds, and the threshold for SCR peaks is .05 microsiemens.

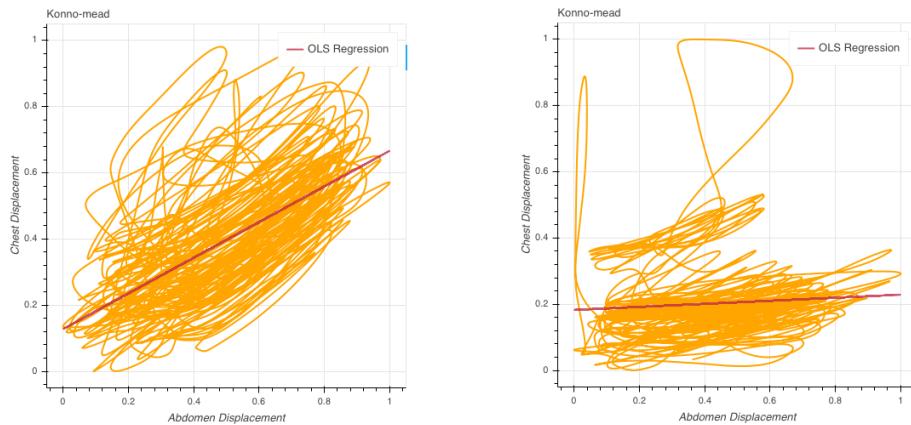
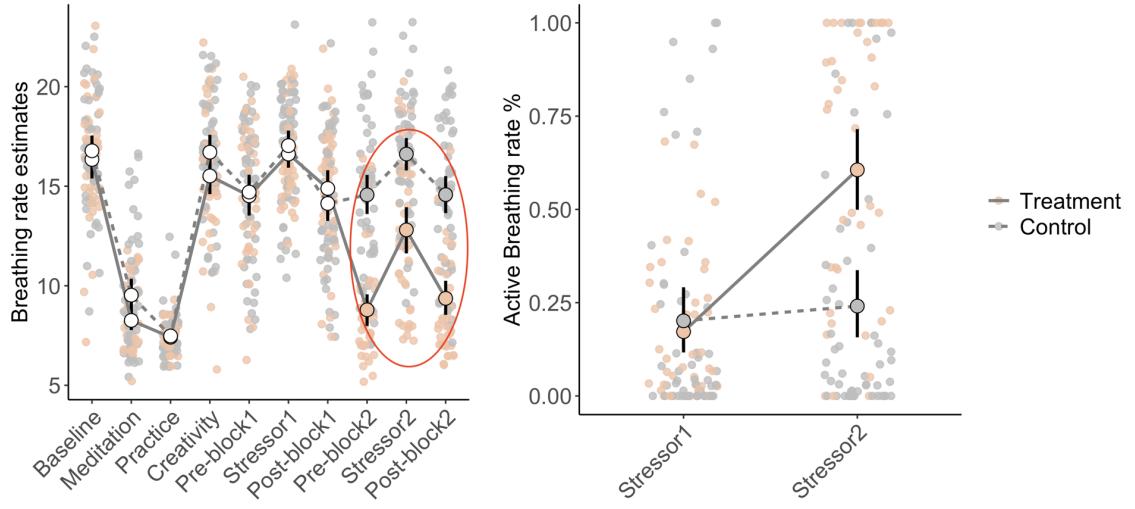


Fig. 5. Examples of Konno-Mead plots for two participants in the treatment group while the pacer was active during the Stressor 2 condition. The participant on the left engaged in more chest breathing as compared to the participant on the right, indicated with the red regression line.



**Fig. 6. Left:** Average breathing rate estimate during conditions of the study for both treatment and control groups. An interaction effect was observed between group (treatment and control) and condition (Stressor 1 and Stressor 2): the treatment group receiving vibrotactile patterns during Stressor 2 experienced a drop in the breathing rate compared to the control group. The solid line indicates the treatment group; the dotted line indicates the control group. Note that the treatment group breathing was significantly lower compared to the control group when the pacer was active (highlighted by the red circle). **Right:** Average active breathing rate during Stressor 1 and 2 conditions as reported on a scale of 1 to 100 by both treatment and control groups. An interaction effect was observed between group (treatment and control) and condition (Stressor 1 and Stressor 2): we did find that receiving vibrotactile patterns had an influence on portion of active breathing.

between the condition (Stressor 1 and Stressor 2) and group (treatment and control) for all three of them. While findings confirmed our expectation regarding slowed breathing, they did not support our expectation that most participants would be able to follow the pacer for the whole duration of Stressor 2. Instead, they were more likely to switch back and forth between passive breathing when solving a problem and active breathing when they were able to attend to the pacer. This pattern of breathing resulted in more irregularity in breathing data, and every time a switch between passive to active breathing occurred, more chest breathing became involved. The subsections below describe our findings in more detail.

**5.1.1 Efficacy of the Pacer in Reducing Breathing Rate.** Consistent with our hypothesis, the treatment group's breathing rate dropped from Stressor 1 to Stressor 2 as a result of the pacer intervention (see Figure 6, left, and Figure 7). The observable large effect (intercept = 17.02,  $\beta = 3.27$ ,  $p < .0001$ , CI = [2.18, 4.36]) suggests that most participants followed the instructions and did try to pace their breathing with the pacer whenever they could. However, some participants succeeded more than others in implementing slow-paced breathing throughout the 4 minutes of Stressor 2.

To quantify how successful participants were in following the pacer, we derived a measure of active breathing percentage. We split the set of rolling windows of breaths per minute, from which we derived the median breathing rate, into two subsets with a cutoff value of 14 breaths per minute. We chose these cutoffs because passive breathing occurs at a rate higher than 14 breaths per minute and has short amplitude. Active breathing, on the other hand, is slower than 14 breaths per minute and has a large amplitude [23].

Figure 6, right, shows the ratio of active breathing in two conditions of Stressor 1 and 2. Treatment and control group were performing an average of 19% of active breathing.<sup>8</sup> During Stressor 2, the proportion of active breathing increased to approximately 57% for the treatment group when the pacer was active, but did not significantly change for the control group. Out of the 44 participants in the treatment group, 11 participants were able to bring their average breathing rate down to a value close to the vibrations pace (a number between 5 to 9 which was determined during the personalization procedure), 27 demonstrated a combination of passive and active breathing and 6 engaged in no active breathing to follow the pacer. Figure 8 shows examples of participants belonging to each of these categories.

While the average breathing rate in the treatment group dropped significantly, the majority of the participants were not able to breathe at a rate consistently lower than nine breaths per minute<sup>9</sup> during Stressor 2. Instead, they engaged in some combination of passive breathing and active breathing. Given that most of the time participants were not active breathing, we might expect that irregularity in breathing would not, in fact, be decreased as we originally hypothesized.

**5.1.2 Efficacy of the Pacer in Reducing Breathing Irregularity.** We observed an interaction effect between group (treatment and control) and condition (Stressor 1 and Stressor 2) for breathing irregularity (intercept = 46.43,  $\beta = -7.25$ ,  $p = .002$ , CI = [-11.64, -2.74]). However, in contrast to our hypothesis, this was characterized by an increase rather than a decrease in breathing irregularity from Stressor 1 to Stressor 2 in the treatment group (shown in Figure 9, left). Although this is inconsistent with our original hypothesis, it supports our earlier observation that participants switched between passive and active breathing rather than consistently performing active slow-paced breathing while the pacer was active.

<sup>8</sup>That is, of the breaths taken in this condition, 19% were at a rate slow enough to be considered to be active breathing. Because of the rolling average used to compute the breathing rates, the first 4 breaths are not included in this percentage.

<sup>9</sup>The pacer's breathing pace ranged from 5 to 9 for all individuals in the study.

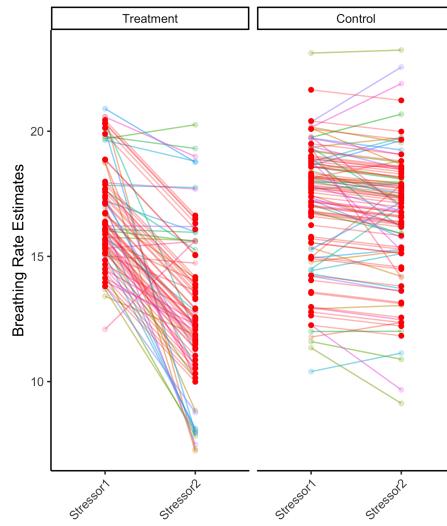
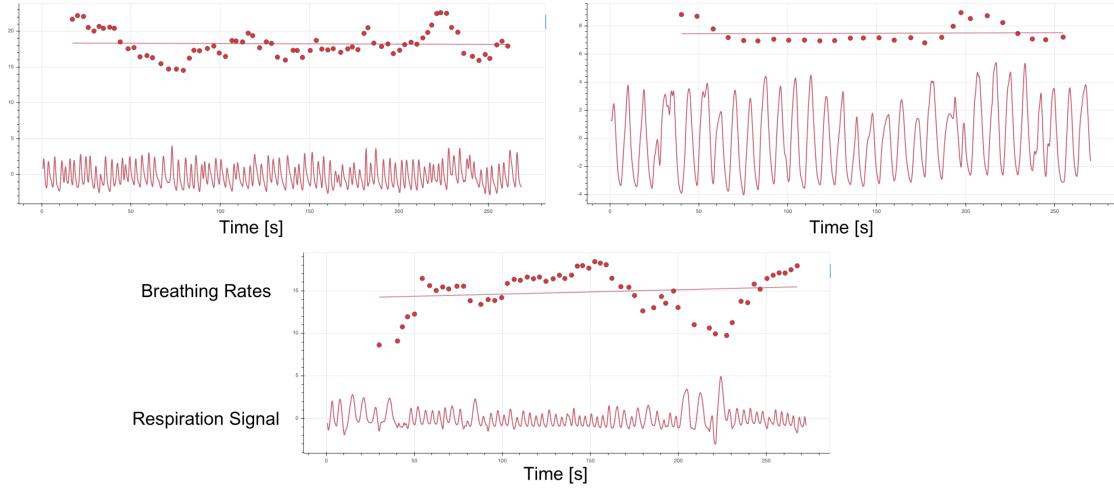


Fig. 7. Linear mixed model fit to breathing rate estimates. Note that the model fit lines with low negative slopes for the control group and lines with large negative slopes for the treatment group (shown in red), in both cases considering individualized intercepts. The model predicts a significant drop in breathing rate for the treatment group going from the Stressor 1 to Stressor 2 condition.



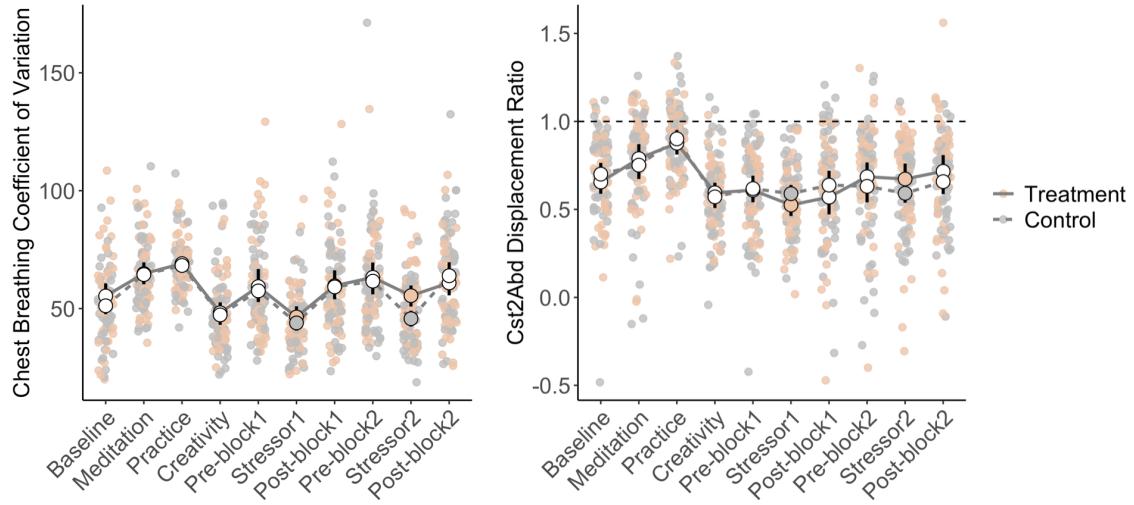
**Fig. 8.** Examples of breathing signals. The red dots are rolling average breathing rates with window of five breaths, and the red line shows the linear regression for the breathing rates. **Top left:** Passive breathing. **Top right:** Active breathing. **Bottom:** Combination of both passive and active breathing. All three examples were from participants in the treatment group who received pacer intervention during the Stressor 2 condition. The cutoff to categorize a breathing rate as active was 14 breaths per minute or slower.

**5.1.3 Efficacy of the Pacer in Reducing Chest to Abdomen Displacement Ratio.** Inconsistent with our hypothesis, when participants switched between passive and active breathing, they demonstrated more chest than abdominal displacement (intercept =  $-.75$ ,  $\beta = -.34$ ,  $p = .03$ , CI =  $[-.64, -.03]$ ). The observed effect was in the opposite direction of what we had anticipated, which was to observe more abdominal than chest movement when the pacer was active (see Figure 9, right).

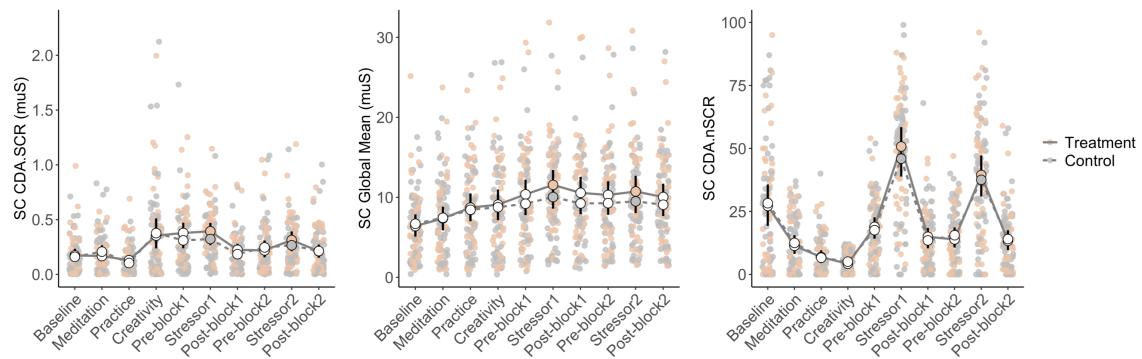
**5.1.4 Efficacy of the Pacer in Reducing Skin Conductance.** We ran linear mixed models on three measures of skin conductance: CDA.SCR, CDA.nSCR, and CS.mean. Although we only pre-registered our hypothesis for the measure CDA.SCR, when we did not observe the interaction effect, we used the other two measures of CDA.nSCR and CS.mean to ensure that none of the three measures showed the hypothesized effect. We found no observable interaction effect between group (treatment and control) and condition (Stressor 1 and Stressor 2) for either CDA.SCR (intercept =  $.39$ ,  $\beta = .02$ ,  $p = .36$ , CI =  $[.02, -.001]$ ), CDA.nSCR (intercept =  $54.86$ ,  $\beta = 4.45$ ,  $p = .14$ , CI =  $[4.45, .11]$ ), or CS.mean (intercept =  $11.55$ ,  $\beta = .28$ ,  $p = .24$ , CI =  $[-.21, .70]$ ) (see Figure 10, center and right). That is, we did not find a significant drop in skin conductance (i.e., a drop in sympathetic arousal) as a result of the breathing pacer intervention using either model-based or model-free approaches.

Inconsistent with our hypotheses, the results suggest that using slow-paced breathing as an affect regulation strategy during the Stressor 2 condition was not found to be any less or more effective than any other affect regulation strategies that the participants may have engaged in during the Stressor 1 condition. In other words, we know that participants attempted to use slow-paced breathing to lower their anxiety during Stressor 2, but it was not any more or less effective than whatever affect regulation method they may have deployed during the Stressor 1 condition during which there were no pacer vibrations.

In an attempt to unravel why we failed to observe the expected effect, we investigated the following alternative explanations and ruled out the ones that were not plausible.



**Fig. 9. Left:** Average breathing irregularity (coefficient of variance) during the study for both treatment and control groups. An interaction effect was observed between group (treatment and control) and condition (Stressor 1 and Stressor 2): the treatment group receiving vibrotactile patterns during Stressor 2 experienced an increase in chest breathing irregularity compared to the control group (see colored dots). Solid line indicates treatment group; dotted line indicates control group. **Right:** Chest to abdomen displacement ratio during the study for both treatment and control groups. An interaction effect was observed between group (treatment and control) and condition (Stressor 1 and Stressor 2): the treatment group receiving vibrotactile patterns during Stressor 2 experienced an increase in chest breathing compared to the control group (see colored dots). Solid line indicates treatment group; dotted line indicates control group.



**Fig. 10.** Three measures of skin conductance all indicating no difference between the treatment and control group. **Right:** The large inferred number of peaks in the phasic drive (CDA.nSCR) during Stressor 1 and Stressor 2 demonstrated that the participants experienced significantly higher levels of sympathetic arousal during the stressor tasks

First, we investigated the validity of the SC data. Figure 10 shows that the SC values were within the expected ranges and they dropped during the V-breathing Practice condition. This suggests that the SC data obtained in this study were valid and reliable as they revealed a well-known link between slow paced breathing and drop in skin conductance.

Second, we investigated whether participants experienced sympathetic arousal as an indication of stress during Stressor 1 and 2. Consistent with our expectation, the large inferred number of peaks in the phasic drive (CDA.nSCR) during Stressor 1 and Stressor 2 demonstrated that the participants experienced significantly higher levels of sympathetic arousal during the stressor tasks segments as compared to the rest of the study conditions. This suggests that the CRA tasks were indeed stressful for the participants (Figure 10, right).

Third, we investigated whether there was a habituation effect to the stressor when participants began Stressor 2. Consistent with our expectation, we observed a habituation effect from Stressor 1 to Stressor 2 in both the treatment and control groups. A main effect of condition on all three measures of CDA.SCR (intercept = .39,  $\beta = -.08$ ,  $p < .001$ , CI = [-.08, .0007]), CDA.nSCR (intercept = 54.86,  $\beta = -13.90$ ,  $p < .001$ , CI = [-13.90, -.04]), and CS.mean (intercept = 11.55,  $\beta = -.82$ ,  $p < .001$ , CI = [-1.15, -.48]) were observed, which indicates that the average observed SC and the inferred sympathetic arousal both decreased from the Stressor 2 to Stressor 1 condition, regardless of the group. This suggests a habituation effect with the CRA tasks in the Stressor 2 condition.

Taken together, these findings show that the measurements and experimental design were meaningful. The investigations below are more speculative in terms of what we observed.

It is conceivable that the effects of anxiety on skin conductance were overshadowed by effects of cognitive load induced by the CRA task. The creativity tasks used in this study were cognitively intensive and required a significant amount of attention.<sup>10</sup> High cognitive load has been associated with an increase in skin conductance response [30]. Therefore, it is plausible that the cognitive demands of the CRA tasks induced a strong upward force on skin conductance that overshadowed the stress reduction effect we were hoping to observe. Indeed, we found that the performance in the CRA did not differ between group (treatment and control) and condition (Stressor 1 and Stressor 2) suggesting that the cognitive contribution to the skin conductance signal remained constant. It is possible that this portion of the cognitive load of skin conductance was strong enough that any change in the stress-related portion of the skin conductance signal was not easily detectable.

Finally, we postulated that the 4-minute window size for the stressors may have contributed to the disappearance of the interaction effect. Perhaps instead, we should have focused on the first minute or the last minute window of the stressors. During Pre-stressor 2, the participant practiced slow-paced breathing, which could give the treatment group a better head start in keeping their breathing rate low, which in turn would lead to lower skin conductance during perhaps the first minute of the stressor. And, during the last minute of the stressor, the treatment group participant has had some time to acquaint themselves with following the pacer while performing CRA tasks. Perhaps the first three minutes can be thought of as a practice period before the final minute's performance, and the effect of active breathing on reducing skin conductance became more visible during the final minute.

We decided to investigate this further by examining the existence of the interaction effect at the beginning and at the end of the skin conductance window. With a window of the first minute of the stressors, we failed to observe an interaction effect. Therefore, the early start of slow-paced breathing did not enhance the skin conductance drop. Note that during the two minutes of the Pre-stressor 2 condition, the slow-paced breathing deployed by the treatment group was not more or less effective in reducing skin conductance than other forms of affect regulation that control group

<sup>10</sup>In addition, the nine second timer, the loud announcement of “correct” and “incorrect” as feedback at the end of each task, and the nature of the compound association of the tasks had all contributed to stress increase.

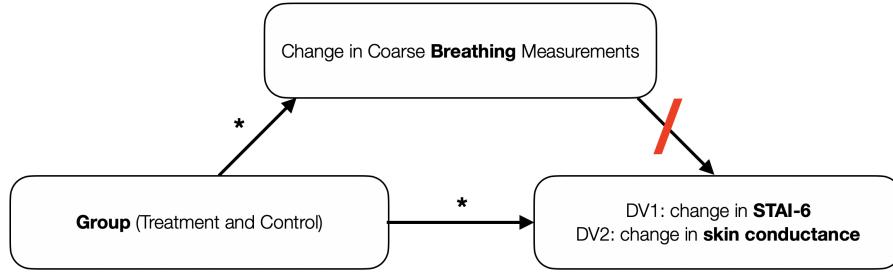


Fig. 11. Mediation models corresponding to Algorithm 1. We did not find the causal links between personalized patterns of vibrations and the two independent variables of skin conductance and self-reported anxiety separately mediated through breathing measures.

participants deployed (see Figure 10). We think that is why we did not observe the interaction effect between group and condition (Stressor 1 and Stressor 2) for skin conductance during the first minute of stressors.

With a window size of the final minute of the stressor, we observed an interaction effect (intercept = 11.12,  $\beta = .60$ ,  $p = .01$ , CI = [.16, 1.04]) between group (treatment and control) and condition (Stressor 1 and Stressor 2). This suggests that participants benefited from becoming acquainted with using the pacer while faced with CRA tasks. Perhaps the first three minutes can be thought of as a practice period before the final minute's performance, and the effect of active breathing on reducing skin conductance became more visible during the final minute. With such practice, the CRA task-induced component that increases skin conductance was weakened compared to the stress reduction component of active breathing that reduces skin conductance.

## 5.2 Exploratory Analysis Results and Discussion

In this section, we investigate the links between the pacer's patterns of vibrations and the two independent variables of skin conductance and self-reported anxiety, separately mediated through breathing rate. To test the mediation effect, we used the `mediate` function of the `mediation` package in R (See Algorithm 1 for sample code). We failed to find the mediation effect of the breathing rate to explain the link between both the pacer and self-reported anxiety as well as the pacer and the skin conductance. The details of the findings and the possible explanations are detailed below.

**5.2.1 Indirect effect of pacer in reducing anxiety through breathing rate.** Miri et al. found an interaction effect between group (treatment and control) and condition (Stressor 1 and Stressor 2) for STAI-6 which suggested a calming effect of the pacer in reducing stress (See [28], Results and Discussion section). We tested whether this effect can be explained by the breathing rate, and did not find the mediation effect (ACME<sup>11</sup> = .03, CI = [-1.69, 1.71],  $p = .97$ ; ADE = -2.45, CI = [-5.56, .5],  $p = .09$ ; total effect = -2.41, CI = [-4.83, -.10],  $p = .04$ ) (Figure 12, left).

One plausible explanation for not observing this causal link is because the self-reported anxiety measures were not collected immediately after the stressors sections. Instead, they were collected at the end of the 2-minute recovery time (Post-stressor 1 and Post-stressor 2). Observe in Figure 10, right, that during the recovery time, level of sympathetic arousal had significantly dropped for both the treatment and control groups, but there was no significant difference between the two in the level of this drop. Therefore, it is possible that the retrospective measure of STAI didn't capture the nature of anxiety that could have been captured had the questionnaires been administered immediately after the

<sup>11</sup>Average Causal Mediation Effects

stressor conditions. Perhaps that is why Miri et al. didn't observe a larger interaction effect and we failed to observe the mediation effect through breathing rate.

Another plausible explanation is that the reduction in self-reported anxiety was mediated by mechanisms other than breathing rate, including a placebo effect. We observed that the participants attempted to pace their breathing with the vibrations. Therefore they may have concluded that their explicit efforts may have changed their affective state into a more calm state. Unfortunately, we cannot tease apart which explanation is more plausible in this study.

**5.2.2 Indirect effect of pacer in reducing skin conductance through breathing parameters.** Because we did not find an effect in skin conductance reduction for the whole window of four minutes, we ran the mediation analysis for the window of the last minute, and found the pacer effect in reducing skin conductance calculated through global mean (see Section 5.1.4). We failed to find the mediation effect that could explain a causal path between vibrotactile patterns of the pacer and skin conductance via breathing rate. However, we observed a direct effect and a total effect that links the two together (ACME = .03, CI = [−.21, −.29],  $p = .76$ ; ADE = −.59, CI = [−1.16, −.02],  $p = .04$ ; total effect = −.55, CI = [−1.02, −.11],  $p = .01$ ). This means that the pacer had a direct impact on skin conductance reduction, but it was not explainable through breathing rate (see Figure 12, right). This then raises the question of what can explain the drop in skin conductance. This is an important question that remains unresolved, which we discuss in the Design Implication section.

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**Algorithm 1:** Testing the mediation effect of breathing rate change to explain drop in skin conductance
 

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```

# Breathing Rate Change model
bpm.model = lm(BPM2 ~ BPM1, data = df)
# Exclude the NAs
bpm.model.no.NA = update(bpm.model, na.action = na.exclude)
# Add column of residuals to the data
df["BPM_change"] = resid(bpm.model.no.NA)
#-----
# Skin Conductance Change model
sc.model = lm(SC2 ~ SC1, data = df)
# Exclude the NAs
sc.model.no.NA = update(sc.model, na.action = na.exclude)
# Add column of residuals to the data
df["SC_change"] = resid(sc.model.no.NA)
#-----
# Mediator model
M.model = lm(BPM_change ~ group, data = df)
#-----
# Predictor model
Y.model = lm(SC_change ~ group + BPM_change, data = df)
#-----
# Bootstrap the mediation effect
fitMedBoot = mediation::mediate(M.model, Y.model, boot=TRUE, sims=1000, treat="group",
  mediator="BPM_change", control.value = "Control", treat.value = "Treatment")
summary(fitMedBoot)
plot(fitMedBoot)
  
```

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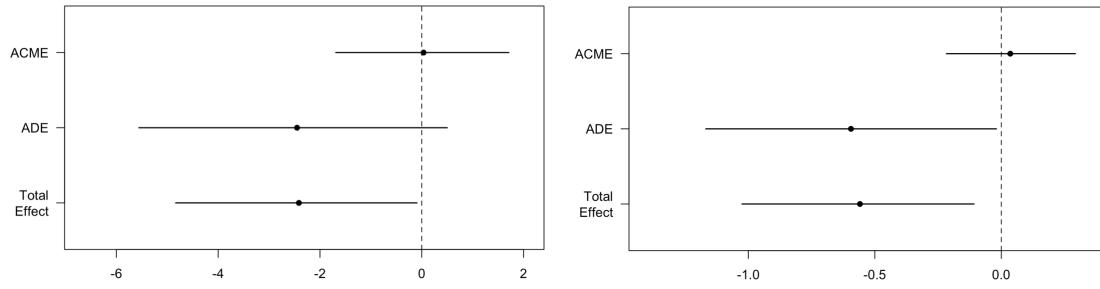


Fig. 12. Testing the mediation effect of breathing rate in explaining the link between vibrotactile patterns and drop in anxiety (left) and skin conductance for the window of the last minute (right). Note that in both figures, no indirect effect (ACME) was found but there exists a total effect indicating the calming effect of the pacer in dropping both anxiety and skin conductance, though not explainable by the breathing rate estimate.

## 6 DESIGN IMPLICATIONS

In this section, we address the following questions: Given that we didn't find a mediation effect of breathing on reducing anxiety and skin conductance, what else can explain its effect? Would we expect to observe a mediation effect of breathing for other forms of vibrotactile patterns? Could it be that Miri et al. didn't choose the appropriate stressor for their study? Lastly, how important is participants' skill in using the pacer under stressful conditions to seeing an effect?

### 6.1 What explains the drop in skin conductance and anxiety if not a mediation effect of breathing rate?

We further tested whether the other measures of breathing we collected (i.e., breathing regularity as well as ratio of active breathing and chest to abdomen breathing) could explain the pacer's effect as a mediator. Unfortunately, none of them showed a mediation effect to explain either the drop in the skin conductance in the last minute window, or anxiety (STAI-6). We postulate that this is because the breathing measures in this study are coarse-grained measurements of what occurred during a four-minute stressor interval. Investigation of other breathing-related data, such as expressing the breathing measures as a time series, could be illuminating. If we were to fail to find a mediation effect of time-series related measures, then alternative explanations linking drop in skin conductance to anxiety (such as placebo or mindset effects) would become more plausible, yet much harder to test for and requiring new experimental design.

### 6.2 Do other forms of vibrotactile pattern impact breathing? For example, would heart-beat like vibrations change the breathing rate? If so, would it convert passive breathing into active, or would we expect passive breathing, but at a slower rate?

Costa et al. [17] reported an increase in heart rate variability (HRV) when the participant was presented with slow heart-beat like vibrotactile patterns as compared with fast patterns. They did not speculate on a mechanism that led to this increase. Since HRV and breathing rate are closely linked, it would be worth examining how breathing patterns changed under the same conditions. We would be surprised if participants were to switch to active breathing (BPM < 13) while the slow heart-beat like patterns of vibrations were active, but it would be illuminating to see if people's passive breathing rates were reduced, and if so by how much.

### 6.3 What is an appropriate choice for a stressor?

The CRA task was selected by Miri et al. [28] as a reproducible model of the stress that arises from solving hard conceptual problems. The Miri et al. [28] study used a mixed design which enables greater statistical power than a between-subjects design. It also helps to address threats to internal validity, meaning that it better enables experimenters to rule out alternative explanations for observed results. The CRA task was particularly suitable for this type of design.

Stressors and stressful situations come in various types, ranging from low to high cognitive demand, as well as anticipatory, in-the-moment, or ruminating stress. The CRA stressor is highly cognitively demanding and is an in-the-moment stressor: the stress starts when the CRA tasks are presented, and ceases when the tasks cease. Our findings suggested that participants often switched their attention between the CRA task and the pacer. Importantly, the extent of attending to the pacer was unrelated to CRA performance. For instance, the CRA scores of the 8 participants who were able to follow the pacer throughout the entire Stressor 2 segment, did not differ significantly from the average score. Likewise, those who did fully ignore the pacer (as determined by them not using active breathing during the Stressor 2 condition) didn't have a score any higher or lower than the average score. Instead, the participants' self-reported strategy to calm themselves down during Stressor 2 suggested that some participants switched their attention to the pacer when they missed some tasks, and some others deliberately switched their attention to the pacer to calm down at a cost of missing a question or two. Among these participant, s016 wrote "*Focused on breathing, especially when I missed a question*"; s085 wrote "*stop when I was getting flustered and focus on matching my breathing to the vibrations to calm myself even if that meant I would miss 1 or 2 questions*"; s109 wrote "*I tried to breathe with the vibrations but it was a little more difficult to do so consistently because I was focused on trying to get more words correct*"; and s067 wrote "*I was really anxious and wanted to finish the task but one thing that helped is the vibrations at times. I would close my eyes or while I was thinking I would notice the vibration and try to sync my breathing to it which helped me calm down and do better.*" These observations suggest that the breathing pacer is usable in the types of stressful situations represented by the CRA task.

Ideally, the efficacy of an affect regulation technology should be tested with respect to different types of stressors. Understanding how breathing is impacted by different types of stressful situations would be valuable to examine the causal link between an affect regulation technology → breathing pattern → affect change. If successful, then we could suggest particular technologies to users based on the type of stressors and the situation for which they are expecting to use the technology.

### 6.4 What is the relationship between the type of stressor and the skill of the user in breathing with the pacer?

Independent of the issue of the type of stressor, is practice using the pacer with the stressor important? We observed the effect of the pacer in dropping skin conductance during the last minute of the stressor, which suggests that the effect may be due to participants' increasing skill in using the pacer under stressful conditions. This raises questions about the kind of practice that is useful. How much is needed? How persistent is the effect of practice—for example, would a third stressor block have shown a reduction in skin conductance by the treatment group? More practice may allow participants to master the act of matching their breathing with the pacer without using up cognitive resources required by the stressor task.

These questions suggest a  $2 \times 3 \times 2$  table with stressor type (low versus high cognitive load), situation (in the moment versus, anticipatory, or after) as one axis and the experience level (novice versus expert) as the other axis. This study

addresses the combination of novice users of the pacer experiencing an in-the moment, cognitively demanding stressor. In the future, it would be useful to sample from other combinations as well.

## 7 CONCLUSION AND FUTURE WORK

The study by Miri et al. [28] found evidence that using the breathing pacer reduced subjective experiences of anxiety, as measured by STAI-6. In this study, we confirmed that the pacer lowered the rate of breathing while also increasing its variability and shifting movements from abdomen to the chest region. Participants engaged on average in 57% of active breathing when the pacer was active and increased their chest movement. We also found evidence that the pacer reduced anxiety, as measured by reduced skin conductance in the last minute of the four-minute stressor condition. We expected to find that the reductions in self-reported anxiety and skin conductance were mediated through breathing rate, especially since the link between slow-paced breathing and reduced skin conductance is well established; but we did not. This was surprising: since the device works by pacing one's breathing, we expected measures of breathing to explain the observed drop in anxiety. Indeed, we did not find evidence that any of the coarse breathing-related measures used in our study were a mediator. Yet, we did see that using the pacer had an effect in reducing participants' breathing rate and changing their breathing patterns. Hence, the mechanism of affect regulation for this particular technology remains a mystery.

We found several hints to explain the lack of meditation. First, we found that using the pacer reduced skin conductance during the last minute of the 4 minute stressor. While this, too, was not mediated by breathing rate (or any other coarse breathing metric), it does suggest that practice in using the pacer was important. Second, the fact that we didn't see a mediation effect of breathing rate may imply that the large cognitive demands placed on the participant by CRA tasks overshadowed the reduction of skin conductance during stressors within the window of the entire 4 minutes. Third, it is possible that breathing did play a role, but the breathing metrics we used were too coarse-grained to detect it. Investigating breathing-related measurement as a time series may lead to insights that will help solve this mystery.

There is considerable interest in technology that uses vibrotactile effects to assist in affect regulation. The mystery of its mechanism is significant to more than just our single study. Understanding the causal links between vibrotactile effects and affect is important in the context of the interaction among stressor types (e.g., low versus high cognitive load), situation (in the moment versus, anticipatory, or after), and user expertise (novice versus expert). Understanding how other technologies perform in this interaction space is important as well.

## 8 ACKNOWLEDGMENTS

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## 9 APPENDICES

### A MODEL-BASED APPROACHES TO SKIN CONDUCTANCE ANALYSIS

The mapping between observed skin conductance (SC) and sympathetic arousal (SA) is known as skin conductance analysis. This analysis uses two general approaches: a model-based approach which uses mathematical models to map between observed SC and SA, and a model-free approach, such as the average mean of an observed SC within a certain time frame. The model-free approach is simple but it is limited in measuring SA because it does not account for overlapping skin conductance responses (SCRs). A model-based approach addresses this limitation by first mapping SC data to sudomotor nerve (SN) activity, which contains discrete peaks that do not overlap, and then, inferring SA from

SN activity. Despite the complexity of model-based approaches, they are preferred to model-free approaches because they provide more accurate estimations of stress responses.

Among model-based approaches, Ledalab [10] is one of the widely used packages to infer SA from observed SC. It infers SA from observed SC in two steps: inference SN from SC, followed by inference of SA levels from SN activity. This relation can be expressed as  $SA \rightarrow SN \rightarrow SC$ . Ledalab uses a causal operation to estimate sudomotor nerve activity from skin conductance and then uses a peak detection method to infer sympathetic arousal from the estimated sudomotor nerve activity. The causal operation, to infer sudomotor nerve activity involves two consecutive steps. The first is a deconvolution of the observed skin conductance with an impulse response function, which is an estimate of a single skin conductance response<sup>12</sup>. The deconvolution results in a *driver* signal that contains both tonic (slow-changing) and phasic (fast-changing) components. The second is an estimation of the tonic activity from the driver in four consecutive sub-steps: (1) smoothing the driver signal using a convolution with a Gaussian function; (2) identifying peaks and local minimums before and after each pick in the smoothed driver (any points that are not within a peak section are considered as part of the tonic driver); (3) interpolating missing data values to create a continuous tonic signal; and (4) subtracting the tonic signal from the original driver to calculate the phasic component of the driver. The derived phasic component (“phasic driver”) is used as an estimation of sudomotor nerve activity [10].

To infer sympathetic arousal, Ledalab detects peaks in the phasic driver above the 0.01 microsiemens threshold. Depending on the desired level of sensitivity the threshold may be set higher than 0.01 microsiemens. Ledalab then presents three estimations for sympathetic arousal: CDA.AmpSum which reports the sum of the amplitudes of peaks in the phasic driver during a response window, CDA.nSCR which represents the total number of significant (above the threshold) peaks in a response window, and CDA.SCR which represents the average phasic driver value in a response window. [3]

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<sup>12</sup>The impulse response function formula is  $e^{(-t/2)} - e^{(-t/.75)}$   $t = 0 : .01 : 20$

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