An auditory heartbeat stimulus can influence heart rate and psychological experience

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Abstract

Stress and anxiety are prevalent in modern society and can cause negative impact on both physical and mental wellbeing. Due to these damaging effects, many people look for non-invasive, convenient methods to reduce chronic exposure to stress, and to reduce their daily levels of anxiety. Cardiac physiological coupling (CPC), also termed cardiac physiological synchrony (CPS), has been explored as a way to influence autonomic activity by aligning a person's heart rate with an external rhythm. Although CPS has been shown effective through delivery of a tactile heartbeat-like stimulus, the benefits of an auditory-only stimulus are not well characterised. We therefore used auditory-only HR to provide an external stimulus and examined whether audio alone could modulate heart rate over the short term in adult volunteers, or whether a tactile component might be necessary. This small-scale study found that the HR of participants decreased following a 'slow' auditory HR stimulus, with a 'fast' stimulus having mixed effects across individuals. Correspondingly, people were better at predicting the change in HR for the 'slow' versus 'fast' auditory stimulus. The results suggest that larger scale volunteer studies, with additional physiological and psychological measures, would be valuable to assess the relevance of auditory only stimuli for inducing alignment between external rhythm and autonomic responses. This work is exploratory and non-clinical, and it does not evaluate treatment effects or make medical claims.

Introduction

Chronic stress and anxiety are linked to numerous physical and mental health conditions including cancer, coronary heart disease, accidental injuries, respiratory disorders, cirrhosis of the liver and suicide [1]. The relationship between chronic stress and health is thought to arise from a combination of environmental stress, psychological (emotional) stress and biological stress [2], with almost 180,000 people/year in the UK dying from some form of stress-related illness [1]. Many people are therefore seeking non-invasive and convenient methods to reduce the stress and anxiety

imposed by modern day living such as through lifestyle changes and the use of technology. One novel approach to ameliorating physiological stress has been shown through Cardiac physiological synchrony (CPS) type strategies [3]. CPS is when the heart rate (HR) and heart rate variability (HRV) of individuals unconsciously couples to people with whom they are sharing experiences. It is unclear what drives this synchrony and whether it occurs directly as a cardiac function or indirectly through cognitive or respiratory processes. As previous studies have shown the benefits of a tactile heartbeat stimulus to reduce HR [3, 4] , we wanted to test the efficacy of audio-only heartbeat to determine whether this would be used as an effective alternative when a tactile device was unavailable, or inconvenient to use during daily activities. Resting heart rate (RHR) serves as a vital indicator of cardiovascular health and autonomic nervous system activity. For adults, a typical RHR ranges between 60-100 beats per minute (bpm), and varies during the day with a night-time decrease [5] with well-trained athletes often exhibiting lower rates, sometimes as low as 30 - 40 bpm [6]. We therefore aimed to compare the effects of a 'slow' versus 'fast' auditory heartbeat on the HR of a participant to understand the ability of auditory-only stimulus on CPS. Participants were given no instructions other than to listen passively (implicit learning), and therefore the study does not investigate a biofeedback component (explicit learning) of the task. In addition, the psychological effect of the auditory HR may differ from the physiological effect on CPS, we therefore asked participants to give unbiased and open-ended feedback without asking any specific questions or explaining the purpose of the testing in relation to stress and anxiety.

Materials and Methods

A total of 13 (7 Male, 6 Female; mean age = 39, s.d. = 15.3) volunteers took part in the study. Following reading of the participant information sheet, all participants gave their written informed consent. This was a non-clinical volunteer study and did not involve patients. Participants were given a heart rate monitor to wear around their chest (Polar H9, Polar Electro Oy, Finland). The device uses an ECG to transmit heart rate and RR interval data via Bluetooth Low Energy (BLE). The signal was recorded using a Bluetooth-compatible mobile application (PolarFlow, Polar Electro Oy, Finland) for data logging and analysis. Participants were asked to wear headphones (Sony WH-1000XM3, Sony, Japan) and listen to an audio track consisting of 10 min no sound baseline (B1). Following B1, participants received 10 min of auditory stimulus of 'slow' heartbeat (S1; 54 bpm). Participants then listened to 10 min of no sound inter-stimulus baseline (B2) to determine that HR returned to resting HR (B1) before Stimulus 2. Stimulus 2 (S2) consisted of 10 min of 'fast' heartbeat (S2; 125 bpm). At the end of the audio track, participants were asked to give feedback of their experience, with no further prompts in an attempt to gain unbiased and open-ended understanding of each participant's experiences.

Results

Baseline Heart Rate Observations (B1)

Participants' (n=13) baseline heart rates were generally aligned with expected norms (60-100 bpm) and ranged from 41.70 to 80.08 bpm (mean = 66.24 bpm, s.d. = 10.31 bpm), with 1 highly athletic adult showing the lowest HR of 41.70 bpm. A repeated-measures ANOVA revealed a significant effect of the condition on heart rate (F(3, 36) = 7.20, p-value = 0.0007).

Heart Rate Response to Stimulus 1 (S1; 54 bpm)

At the group level, post-hoc paired *t*-tests revealed a statistically significant reduction in HR from Baseline to Stimulus 1 (S1; 54 bpm) (Figure 1; t(12) = 4.76, p-value = 0.002, Bonferroni-corrected).

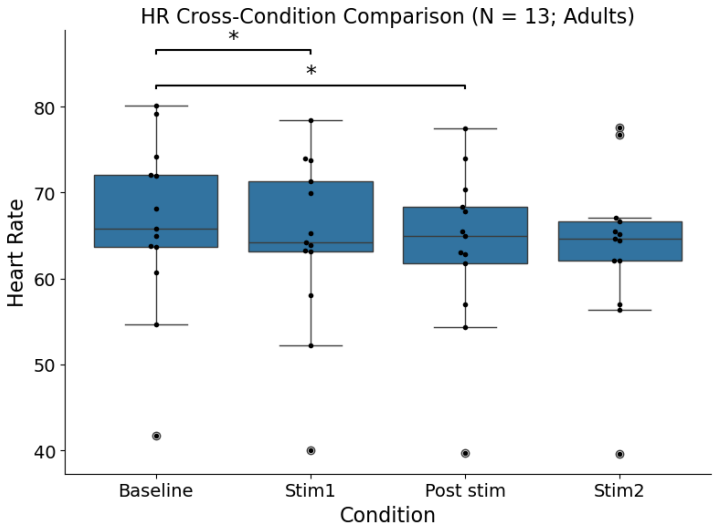

Figure 1. Group-level heart rate (HR) responses across experimental conditions (N = 13).

Individual-level changes in HR were also assessed between all condition pairs (Figure 2). 100% of participants experienced a decrease in heart rate from baseline (B1) to Stimulus 1 (S1), with changes ranging from −0.31% to −6.81% and an average reduction of −2.77% (Figure 2a), corresponding to a mean absolute reduction of approximately 1.84 bpm from baseline. This consistent pattern suggests a calming, parasympathetic effect from the slow auditory rhythm.

Post-Stimulus (B2)

After the Stimulus 1 (S1; 54 bpm) session, participants entered a 10 min silent baseline phase (Post-Stimulus, B2), during which we continued monitoring heart rate to assess whether any physiological changes were sustained, and whether resting heart rate was restored before S2. At the group level, there was a statistically significant decrease in HR from baseline in this phase, (Repeated Measures ANOVA; p-value = 0.008; F = 4.14), with all participants maintaining a lower HR than their

baseline during this silent phase (B2 < B1; Figure 2b), with reductions during the Post-Stimulus session ranging from −0.31% to −11.18%, with an average decrease of −3.84% from baseline.

When comparing HR during Stimulus 1 to values in the Post-Stimulus session (S1 versus B2), no statistically-significant results were obtained (Repeated Measures ANOVA; p-value = 0.08; Table 1). At the individual level, we found that 69.2% of participants maintained a lower heart rate than during Stimulus 1 (B2 < S1), showing continued downward regulation or stability (Figure 2c). The remaining 30.8% of adults showed a modest rebound, with increases ranging from 0.00% to 3.92% (Figure 2c).

Heart Rate Response to Stimulus 2 (S2; 125 bpm)

In the final phase, participants were exposed to a 'fast' auditory heartbeat stimulus at 125 bpm for 10 minutes (S2; 125 bpm). While we hypothesised a potential arousing effect, results were mixed. This phase was designed to assess whether an elevated rhythm would produce alerting effects, in contrast to the calming impact of slower rhythms.

During this phase, no statistically significant decreases were obtained from baseline (B1) after multiple comparisons corrections (S2 versus B1, Table 1). This appeared to be due to the mixed effects of S2 with some people experiencing trends of decreased or increased HR on the individual level. At the individual level, 84.6% of participants (11 out of 13) showed a decrease in HR during S2 compared to their baseline (B1), with reductions ranging from −0.60% to −15.31% (S2 versus B1; Figure 2d). However, 15.4% (2 out of 13) exhibited a modest increase in HR (+3.16% and +3.48%; Figure 2d).

When comparing HR values between B2 and S2, no statistically significant differences were obtained at the group level. At the individual level, 53.8% of participants (7 out of 13) experienced a decrease in heart rate during S2 relative to B2. Thus, even with a faster stimulus tempo, the majority of participants still exhibited downward HR modulation — possibly due to cumulative calming effects. 46.2% (6 out of 13) participants experienced a modest increase in heart rate from Post-Stimulus, with changes ranging from +0.08% to +3.80% (Figure 2e).

Condition A	Condition B	T	p-value (uncorrected)	p-value (Bonferroni-corrected)
B1	S1 (54 bpm)	4.76	0.0004	**0.002**
B1	B2	4.14	0.001	**0.008**
B1	S2 (125 bpm)	2.66	0.02	0.12
S1	B2	1.89	0.08	0.4
S1	S2	1.18	0.2	1
B2	S2	0.34	0.7	1

Table 1. Results of Repeated Measures ANOVA with Post-Hoc Pairwise Comparisons (N = 13). The T-statistic (T in this table) indicates the standardised difference between the means of each two conditions compared, relative to the variability in the data. The Bonferroni correction accounts for multiple comparisons by adjusting the significance threshold, thus reducing the risk of false positives. All values are provided for transparency.

We also compared HR during Stimulus 1 with that during Stimulus 2 (S1 versus S2) and no statistically significant differences were observed at the group level (Table 1). 30.7% (4 out 13) of subjects showed an increase in HR during Stimulus 2 compared to Stimulus 1 ranging from 0.23% to +7.82% (Figure 2f). 69.2% (9 out of 13) of the participants showed a decrease in HR between these 2 conditions, ranging from -9.11% to -0.07%.

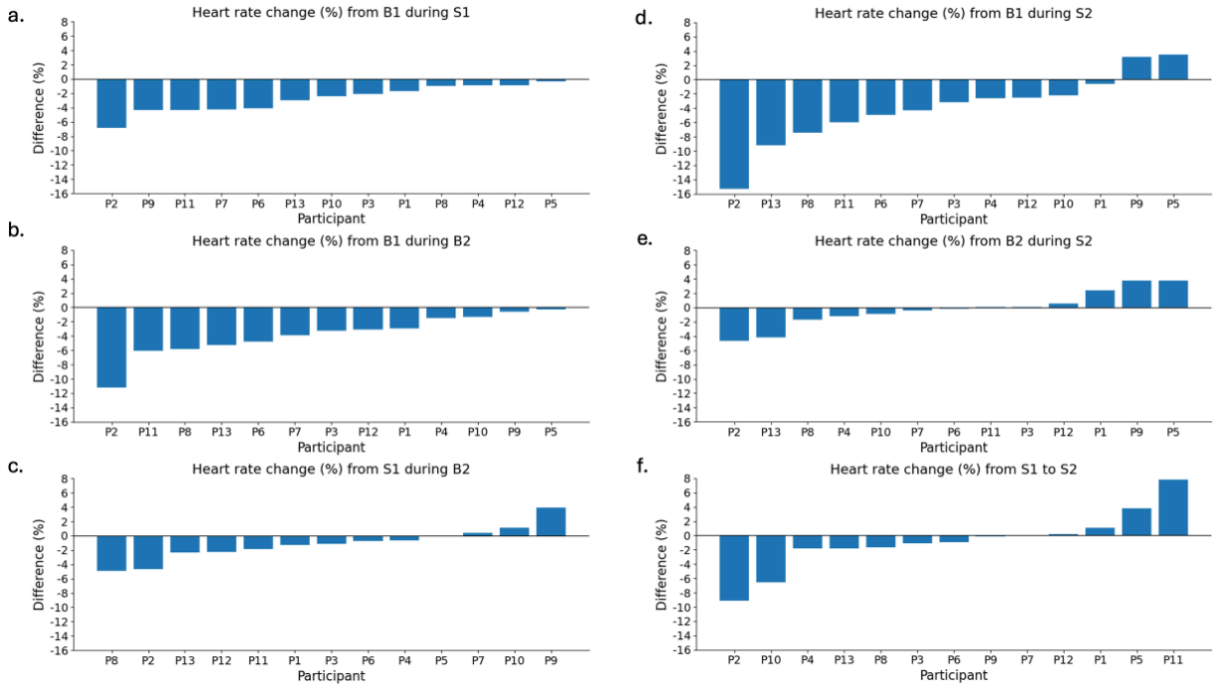

Figure 2. Individual-level changes between condition pairs (expressed as%) in all participants.

Participant feedback on 'slow' (S12) versus 'fast' (S2) heart rate audio

Participants were asked for their post-testing feedback without any specific questions asked to each participant to gain an unbiased and open-ended understanding of their experience during the S1, B2 and S2 auditory stimuli. General feedback included volume sensitivity (*"sound was slightly loud", "audio started and was very loud", "stimulation was quite loud");* and looping and naturalness of audio (*"no variability… became more of a beat rather than a heartbeat", "not perfectly looping… vaguely irritating"*). Feedback of S1 was that the slower heart sound was preferred compared to S2 (*"slower sound is more relaxing"; "stim1 felt more paced as his own heartbeat"*). Participants also related S1 or B2 to sleepiness and relaxation (*"4 on sleepiness scale - nearly fell asleep", "started feeling sleepy when it stopped", "dozed off… was awake when sound started", "didn't properly fall asleep but was dozing"*).People related S2 to anxiety or irritation (*"fast HR was horrible and made me feel really anxious", "stim2 felt too fast, almost like it was causing him to speed up", "faster one was more irritating, harder to relax to"*). Although this feedback is helpful to understand people's initial reactions to S1 and S2, more detailed feedback would be helpful to generate further thematic

analysis and understand the similarities but also the differences in the experiences between participants.

Discussion

The results showed a statistically significant reduction in heart rate of −2.77% (≈ −1.84 bpm), during exposure to a slow auditory rhythm (S1) compared to baseline rest (B1). Although modest in magnitude, resting heart rate is a stable physiological measure, and small changes therefore reflect meaningful autonomic modulation. In line with this scale of effect, a meta-analysis of cognitive-behavioral therapy (CBT) studies in post-traumatic stress disorder reported a weighted mean heart-rate difference of −1.81 bpm [7] between CBT-treated patients and control groups, with a larger difference of −2.80 bpm [7] reported for traditional exposure-based therapies (Table 2). Notably, our present effect (−1.84 bpm) was observed in a non-clinical population during a single 10-minute resting session, suggesting that measurable autonomic modulation can be elicited under minimal exposure conditions. Future studies should assess whether larger or cumulative effects emerge with repeated use or in populations with elevated baseline stress or anxiety.

Study	Design	HR change
Current study	Within-subject (B1 → S1)	≈ −1.84 bpm
CBT meta-analysis (PTSD) [7]	Between-group (CBT vs control)	−1.81 bpm (WMD)
Exposure therapy meta-analysis [7]	Between-group	−2.80 bpm (WMD)

Table 2. Comparison of the magnitude of heart-rate change (bpm) in the present within-subject study and the weighted mean differences (bpm) reported in the meta-analysis of CBT and exposure-based interventions by Gonçalves et al. (2015) [7].

All participants showed a reduction in heart rate from baseline (B1) to the slow auditory stimulus (S1), further supporting the robustness of this response across individuals. Although the absolute magnitude of change varied, the reduction of HR consistency of direction suggests that slow heartbeat-like auditory cues may elicit a parasympathetic-like response in healthy volunteers.

During the Post-Stimulus session (B2), when no auditory stimulus was presented, the heart rate remained lower compared to the initial baseline (B1), indicating a lingering calming physiological effect of the stimulus. These findings may suggest that for most participants, the calming physiological effects of auditory stimulus can persist even after the stimulus is removed, which is an important insight for practical applications. An additional contributing factor could be that the study environment itself may have contributed to a gradual calming effect. Sitting quietly, focusing on bodily sensations, and anticipating structured phases may induce a mild mindful or placebo-like settling of autonomic activity, independent of the auditory stimulus.

A limitation of this study is the lack of a control condition or counterbalanced order of S1 and S2, which restricts our ability to determine whether the observed effects reflect the auditory stimulus itself or cumulative relaxation over time. In addition, the small sample size (n = 13) limits the

generalisability of the findings and means that statistical results should be interpreted cautiously. Nonetheless, the immediate reduction observed during S1 suggests that at least part of the effect is stimulus-driven, even though cumulative relaxation or environmental settling effects cannot be fully ruled out.

In addition, future studies may compare the effectiveness of different stimulus rates as a percentage of each person's resting heart rate. Tuning the stimulus to the individual participant may make the change in HR more significant. Previous studies have shown ±3 % of a person's HR is optimal to cause cardiac physiological synchrony [4], whereas we used a constant S1 'slow' and S2 'fast' HR irrespective of the resting heart rate baseline (B1) or poststimulus (B2) that was initially measured before the auditory stimulus.

The question of whether a standard auditory 'slow' heartbeat can reduce an individual's heart rate appears to be answered affirmatively by emerging evidence. This phenomenon may be understood through the lens of heart rate synchronisation – a process in which an individual's physiological rhythms align with external cues – potentially involving mechanisms of implicit learning [8, 9]. In contrast, heart rate modulation through biofeedback typically engages explicit learning, where individuals consciously attempt to control their heart rate using direct feedback [10]. While explicit and implicit learning are distinct, they may be related in this context: listening passively to a slow heartbeat may lead to unconscious entrainment, particularly when no instruction or feedback is given. This suggests that heart rate synchronisation could offer a subtle, socially and physiologically grounded route to autonomic regulation, distinct from but complementary to explicit feedback-based methods [3].

Conclusion

A slow auditory heartbeat (54 bpm) was associated with short term reductions in heart rate, relative to baseline in this small volunteer sample, while responses to a fast heartbeat varied across individuals. These findings support further controlled research with larger samples, randomised stimulus order, randomised pulses, individualised tempo conditions, and additional physiological and psychological measures. This study is exploratory and non-clinical and does not evaluate treatment effects.

Author contributions

RI - data analysis, writing. LSE - data collection, editing. MR - writing, editing, supervision. S-MJ - data collection, project conception, experimental design, supervision.

Ethics approval and consent to participate

This activity involved healthy adult volunteers and did not involve patients, diagnosis or treatment. It was reviewed as minimal risk volunteer testing of auditory stimuli. According to local institutional guidance, formal clinical research ethics committee review was not required for non-clinical

volunteer studies of this nature. All participants provided written informed consent to take part, and provided consent for publication of aggregate, anonymised findings.

Consent to participate

Informed consent to take part in this evaluation was obtained from the participant.

Consent to publication

Informed consent for publication was obtained from the participant.

Funding

Author Soo Min (Lucy) Jung is supported by Cambridge Neuroworks Fellowship.

Conflicts of interest

All authors are employees, inventors or collaborators of LYEONS, the company that developed the prototype device described herein. This affiliation represents a potential conflict of interest. We declare that we have taken care to present findings objectively and transparently. The prototype used is not a medical device and is not marketed for therapeutic use.

Acknowledgement

We thank all participants for their time and involvement in this study.

Disclaimer

This exploratory study does not evaluate or imply therapeutic effects, diagnostic capabilities, or clinical performance of any product. The prototype device used in testing is not a medical device and is not intended for medical use.

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