



## Research article

# A comprehensive evaluation of linear and non-linear HRV parameters between paced breathing and stressful mental state

Kulbhushan Chand <sup>a,\*</sup>, Shilpa Chandra <sup>b</sup>, Varun Dutt <sup>b,\*\*</sup><sup>a</sup> IIT Mandi iHub and HCl Foundation, Indian Institute of Technology Mandi, Kamand, HP, India , 175005<sup>b</sup> Indian Institute of Technology Mandi, Kamand, HP, India , 175005

## ARTICLE INFO

## Keywords:

Heart rate variability  
Relaxation  
Stress  
Paced breathing  
Autonomic nervous system  
Linear and non-linear HRV

## ABSTRACT

**Background:** Heart rate variability (HRV) is a crucial metric that provides valuable insight into the balance between relaxation and stress. Previous research has shown that most HRV parameters improve during periods of mental relaxation, while decreasing during tasks involving cognitive workload. Although a comprehensive analysis of both linear and non-linear HRV parameters has been carried out in existing literature, there still exists a need for further research in this area. Additionally, limited knowledge exists regarding how specific interventions may influence the interpretation of these parameters and how the different parameters correlate under different interventions. This study aims to address these gaps by conducting a thorough comparison of different linear and non-linear HRV parameters under mentally relaxed versus stressful states.

**Methodology:** Participants were randomly and equally divided among two between-subjects groups: relaxed-stress (RS) (N = 22) and stress-relaxed (SR) (N = 22). In the RS group, a paced breathing task was given for 5 min to create relaxation, and was followed by a 5-min time-based mental calculation task to create stress. In the SR group, the order of the stress and relaxed tasks was reversed. There was a washout period of 15 min after the first task in both groups.

**Results:** Of the 37 HRV parameters, 33 differed significantly between the two interventions. The majority of the parameters exhibited an improving and degrading tendency of HRV parameters in the relaxed and stressed states, respectively. The correlation of the majority of HRV parameters decreases during stress, while prominent time domain and geometric domain parameters stand out in the correlation.

**Conclusion:** Overall, HRV parameters can be reliably used to assess a person's relaxed and stressed mental states during paced breathing and mental arithmetic task respectively. Furthermore, non-linear HRV parameters provide accurate estimators of the mental state, in addition to the commonly used linear parameters.

## 1. Introduction

HRV is the change in the time intervals between successive heartbeats, facilitating adaptability to environmental and psychological challenges [1]. This low-cost, non-invasive technique serves as a metric for cardiac autonomic modulation [2]. HRV has been

\* Corresponding author. IIT Mandi iHub and HCl Foundation, Indian Institute of Technology Mandi India, Kamand, HP, India - 175005

\*\* Corresponding author. Indian Institute of Technology Mandi, Kamand, HP, India - 175005

E-mail addresses: [kulbhushan@ihubiitmandi.in](mailto:kulbhushan@ihubiitmandi.in), [kulbhushan.chand@gmail.com](mailto:kulbhushan.chand@gmail.com) (K. Chand), [d22115@students.iitmandi.ac.in](mailto:d22115@students.iitmandi.ac.in) (S. Chandra), [varun@iitmandi.ac.in](mailto:varun@iitmandi.ac.in) (V. Dutt).

<https://doi.org/10.1016/j.heliyon.2024.e32195>

Received 9 January 2024; Received in revised form 22 May 2024; Accepted 29 May 2024

Available online 31 May 2024

2405-8440/© 2024 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

thoroughly investigated to gain insights into the impact of stress on cardiac autonomic functioning, thereby establishing itself as a reliable stress indicator [3–8]. It offers an indirect representation of intricate interactions for example as those between the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS), blood pressure (BP), gas exchange, and various organs including the heart and stomach [5].

Distinct pathological and non-pathological circumstances, ranging from cardiovascular diseases to psychological conditions, can alter HRV, positioning it as a potential health indicator [9] and also reported to differ between sex [10]. Moreover, HRV has implications as a biomarker for various conditions such as vascular tone, which is essential for blood pressure regulation and heart function [6,9]. Its association with self-rated health surpasses that of certain inflammatory and other biomarkers [11].

Respiratory sinus arrhythmia (RSA) or cardiac coherence refers to the fluctuation in heart rate during a breathing cycle, with an increase during inhalation and a subsequent decrease after exhalation. In the HRV field, most investigations used idle sitting or unconstrained deep breathing to induce relaxation, but only a few have used paced breathing. Paced breathing during the HRV biofeedback, involves intentional slow breathing to enhance RSA, usually at a pace of 4.5–7 breaths per minute (bpm). These techniques are recognized for their positive effects on well-being, stress reduction, and blood pressure regulation [12–14] and can also induce changes in the resting HRV and the brain circuits that help control HRV and regulate emotion [15]. Resonance breathing, for instance, augments vagal tone, subsequently enhancing HRV as a health and stress index [16]. The neuro-visceral integration model suggests that a heightened vagal tone can bolster cognitive functions [17]. Empirical evidence further underscores cognitive benefits, such as improved attention and brain functionality derived from paced breathing, mindfulness meditation, and aerobic exercise [18]. Moreover, paced breathing has been linked to reduced emotional distress, operating through the vagal pathway that influences various brain regions, including the amygdala and the hippocampus [19].

Therefore, HRV is postulated to reflect the efficiency of the body's response mechanism to diverse stimuli, emphasizing physiological stability [20]. However, recognizing the myriad factors influencing HRV is paramount before interpreting such responses [21]. Different mental states, such as relaxation or stress, can substantially alter HRV parameters [22]. For instance, relaxation typically amplifies HRV parameters owing to parasympathetic dominance, whereas stress reduces them because of heightened sympathetic activity [6]. The precise interpretation of these parameters often necessitates a thorough understanding of the interventions and analytical methodologies in place. A prominent example is how the decrease in LF power during relaxation might be misconstrued in specific contexts, such as when paced breathing impacts the LF band [23].

Various HRV parameters have shown a degree of correlation. SD1 correlates with the LF and HF band power [24], SD2 correlates with the RMSSD and HF band power [24,25], SD2/SD1 ratio correlates with LF/HF ratio [26], SDNN correlates with HF band power, pNN50, and RMSSD [27]. These correlations vary according to the intervention, and may show different results under relaxation and stress [26]. However, there is a lack of comprehensive studies evaluating most HRV parameters during relaxation and stress interventions.

In the area of HRV, it is typical to employ linear parameters for assessing alterations in heart rate and RSA induced by paced breathing routines [28]. Paced breathing at specific frequencies has been shown to influence HRV, with ascending and descending breathing rates producing consistent results [13]. Non-linear parameters, however, provide insight into the complex interactions between respiration, cardiovascular function, and autonomic modulation [29]. Research has indicated that increased RSA and elevated levels of HRV are linked to improved health results and extended lifespan [30]. Conversely, low RSA is linked to poorer health conditions, such as chronic stress, anxiety disorders, depression, PTSD, and aging [31]. Hence, focusing on both linear and non-linear aspects of HRV can offer a complete insight into cardiac autonomic regulation while performing paced breathing techniques.

Moreover, although numerous studies utilize LF power and LF/HF ratio to assess sympathetic functions, some question the reliability of employing these measures for such purposes [32]. This underscores the necessity for more robust investigations that compare HRV parameters across various interventions. Given these complex dynamics, employing a within-subjects design has emerged as an optimal approach to scrutinizing the influence of cardiorespiratory oscillations on behavior, minimizing interindividual differences.

This study is motivated by the detailed observations mentioned earlier, which seek to distinguish the patterns of both linear and non-linear HRV parameters during states of relaxation and stress. By comparing HRV parameters during relaxed states, such as paced breathing, and stressful states, such as mental calculation, this research provides a clearer differentiation between the two interventions. Specifically, this work stands apart in its exhaustive examination of both linear and non-linear HRV parameters under two contrasting interventions, relaxation and stress, leveraging short-term HRV recordings. The cornerstone of our study was the comprehensive assessment of HRV parameters, outlining the mean values and effect sizes extracted from these parameters. Additionally, the correlation analysis among the HRV parameters under the different interventions provided new insight into the use of interventions for relaxation and stress.

The subsequent sections will detail the preceding work that shaped the hypothesis concerning HRV parameter modulation during different states, elucidate the experimental design adopted, and outline the findings, culminating in a discussion on the broader implications of variations in HRV parameters in relaxed and stressed states.

## 2. Background

### 2.1. HRV parameters overview

HRV serves as a complex biomarker, providing crucial understandings into the operations of the autonomic nervous system. HRV parameters can be classified into linear and non-linear categories. Linear parameters are derived from the time domain (TD), frequency domain (FD), and geometric parameters (GD), while non-linear parameters utilize the irregularities in the time series derived from the

complexity of the mechanisms that regulates HRV [6]. The linear parameters are affected by non-stationarity of the data as opposed to non-linear parameters [33]. Therefore, during conventional recording length of 5 min, stationarity in the data is assumed [34].

**Time Domain Analysis:** Of the different analyses available, time-domain parameters are the most straightforward, providing a direct method for calculating HRV. The time-domain measures may use a range of recording lengths from <1 min to >24 h. Some of the time-domain metrics are SDNN, SDHR, RMSSD, NN50, pNN50, HTI, and TINN. Conventionally 5 min recording period is used for the measurement and should be consistent in length while comparison across different sessions or subjects [6,34]. For example SDNN recorded over 24 h period is considered as gold-standard for medical stratification of cardiac risk [34] and is not the same as SDNN computed over 5 min recording.

**Frequency Domain Analysis:** Complex FD parameters, including Very Low Frequency (VLF), Low Frequency (LF), High Frequency (HF), and the HF/LF ratio, are essential for assessing the influence of the autonomic nervous system on HRV, with data extracted either from a 24-h Holter ECG record or short-term recordings lasting 2–5 min [33]. The recording length is recommended to be at least 10 times the wavelength of the lower frequency bound, with an absolute minimum of 1 min recording for HF components, 2 min recording for LF components and 10 min recording for VLF component [34,35].

**Non-linear Analysis:** However, the heartbeat's underlying dynamics, steered by interactive cardiovascular control systems, are often too multifaceted to be entirely depicted using standard TD and FD analyses. This intricacy has prompted efforts to utilize the principles of non-linear dynamics, leading to the development of non-linear HRV parameters. Notably, while linear HRV parameters (barring geometric parameters) are susceptible to artifacts and missed beats, their non-linear counterparts are robust against these anomalies [36].

**Comparison Metrics:** Typically, HRV parameters are benchmarked against a baseline, either an idle sitting [35] or a controlled non-task situation that ensures an objective comparison, [37 p. 6. Situations juxtaposing mental stress against relaxation, for instance, enable a pronounced contrast between two diametric interventions.

As per the literature, the HRV parameters that shows a decrease in value during mental stressful intervention is PNS Index [38], SDNN [34,39], SDHR and MaxHR [35], DiffHR [6], RMSSD [27,39,40], NN50 and pNN50 [34,41,42], HTI [3,34], TINN [34,43], DC and DCmod [44,45], HFap and HFrp [6,46–49], SD1 [50,51], DFA $\alpha$ 1 [52,53], D2 [53,54], MeanLL [55,56], MaxLL [56], REC [55,56], DET [56], and ShEn [55–57].

The HRV parameters that increase in value during stress are SNS Index [38], SI [58], LFap and LFrp [6,34,59], LF/HF [34,59,60], SD2 [24], SD2/SD1 [61,62], ApEn [63–65], SampEn [63,66], DFA $\alpha$ 2 [52,53], and D2 [53,54]. The respiration rate RESP, although not an HRV parameter, may influence these parameters [39,67–69].

Specifically, the reported change in the direction of HRV parameters in the literature depends on the intervention. However, some parameters may deviate from the norms in the literature owing to other factors. For example, because of slow and paced breathing, RSA may become pronounced, potentially affecting the power in the LF and HF frequency bands [48,70]. The LF range (0.04–0.15 Hz) mainly reflects the baroreceptor activity during periods of rest [1]. Activities involving mental arithmetic tend to significantly enhance LF dominance in the RR spectrum [71]. Nevertheless, the vagal activity during respiration at a lower rate can effortlessly produce oscillations in HRV that extend into the LF band [72–74]. Therefore, LFap and LFrp may show increased values due to spillover of the RSA frequency at 0.1 Hz in the LF band [72–74]. Because of the issues described with the LF band, the LF/HF ratio is also influenced by RSA rhythms during slow and deep breathing or paced breathing [75].

Likewise, the HF range (0.15–0.4 Hz) signifies parasympathetic activity and is commonly referred to as the respiratory band due to its correlation with heart rate fluctuations during the respiratory cycle [6]. However, these changes in HR, which is referred to as RSA, are not necessarily an accurate indicator of cardiac vagal control [47]; however, a lower HF power has been linked to stress, panic, anxiety, or distress [46]. Fast paced and deep breathing (9–24 bpm), can change HF power without affecting vagal tone [47].

In addition to the FD parameters, SDHR, DiffHR, and MaxHR may be influenced by paced breathing because of the RSA effects at the resonant frequency. However, the MeanHR change across interventions reflects vagal tone, which may not be affected by paced breathing at the resonance frequency, unlike the large-scale shift in the RSA magnitude [35].

## 2.2. RSA and HRV

RSA (or cardiac coherence) is the term used to describe the fluctuations in the heart rate that occur as a result of changes during the breathing cycle. These changes are largely affected by the vagal tone and can be noticed within the frequency span of 0.15–0.4 Hz [76, 77]. A notable connection can be found between changes in the depth and timing of respiration and the amount, consistency, and timing of vagal cardiac output in individuals who are awake. Noteworthy differences are evident when comparing values during natural breathing and controlled, rhythmic breathing [70]. Under normal breathing conditions, the rhythmic alterations in vagal motoneuron activity associated with respiration commence during the exhalation phase, develop gradually, and do not completely manifest themselves during fast breathing rates [48]. Increased tidal volumes and reduced respiration rates directly impact and elevate RSA [39,67] by means of the central connection between the cardiac vagal motor neurons and the respiratory drive [69].

In the initial continuous monitoring of RSA in a healthy individual, Angelone and Coulter [78] were the first to document this phenomenon. They showed that as the rate of respiration slowed down, the fluctuations in phase also decreased. At a respiratory pace of 4 bpm, there was a distinct alignment between HRV and the inhalation/exhalation cycle. However, the maximum amplitude of HRV was noted at 6 bpm (0.1 Hz), with a phase discrepancy of 90°. Numerous investigations [67,79,80] have validated the maximization of RSA at approximately 6 bpm. This is called the “resonant frequency effect” and shows cardiorespiratory resonance [81]. Further research suggests that when respiration is reduced to 6 bpm, both HRV and RSA are maximized; however, this resonant frequency varies across people [81–83]. Diaphragmatic breathing [84] and increased tidal volume [67,85,86] and have been demonstrated to

dramatically boost RSA, with the effect being stronger at lower respiratory rates. Numerous investigations, on the other hand, have also found that increasing respiration rate decreases RSA [67,78,87].

The effects of respiration rate on autonomic activity may be evaluated through LF/HF power and TD parameters using power spectral analysis adjusted for respiratory influence [79]. Using this technique, Chang et al. [88] found that healthy humans who breathed at 8 bpm instead of 12 and 16 bpm, resulted in a transition towards a more balanced parasympathetic state and an augmentation in vagal activity. Similarly, Zhang et al. [89] used TD parameters to describe the respiratory response patterns of vagal activity in healthy adults at respiration of at 8 to 18 bpm. By retraining vagally triggered cardiac resetting to periods of respiration, they discovered that delayed breathing increased vagal power [90]. Additionally, it has been demonstrated that when breathing is regulated, slow, and deep, sympathetic activity is more completely inhibited from the beginning of inspiration until the middle of expiration [91]. It has been proposed that persistent practice of slow breathing is essential to produce a lasting shift towards parasympathetic dominance, as evidenced by healthy adults who incorporated slow breathing into their daily routine over a period of 3 months [92].

In healthy individuals breathing at 15-3 bpm, Taylor et al. [86] investigated the impact of sympathetic blockage on RSA. They observed that across all breathing rates, the inhibition of cardiac sympathetic activity results in an increase in RSA. Their findings suggested that tonic vagal activity remains consistent across different breathing rates, leading them to propose that rapid breathing reduces the release of acetylcholine due to shortened expiration, thereby decreasing RSA. In contrast, acetylcholine release and hydrolysis are enhanced at the resonance frequency (6 bpm), resulting in the maximum RSA. In addition, Wang et al. [93] reported an enhanced propensity for HRV at 6 bpm when the inhalation/exhalation ratio was 1/1, and they attributed this observation to the optimal release and hydrolysis of acetylcholine.

### 2.3. Gap and expectations

While the existing literature offers fragmented insights into HRV dynamics under varying interventions, a holistic understanding remains elusive. We anticipate that HRV parameters will exhibit pronounced enhancement during relaxation interventions, specifically paced breathing, attributed to heightened PNS activity. Drawing from the aforementioned literature, our hypothesis suggests that most of the HRV parameters will improve during the relaxation intervention (paced breathing) compared to the stress intervention, primarily thought to result from the stimulation of the PNS. We also hypothesized that the prominent RSA (or cardiac coherence) during relaxation causes spill-over of the band power, which may lead to different results, as reported in the literature, during the comparison and correlation of HRV parameters between relaxation and stress interventions. We also expect to see most of the correlations between HRV parameters, as reported in the literature, such as between the TD and FD, and additionally some new insights that may help in establishing the suitability of using non-linear HRV parameters in HRV assessment studies. The primary objective of this study was a comprehensive evaluation of the variations in most available HRV parameters, both linear and non-linear, under relaxed and stressful interventions using short-term HRV recordings and to provide a comprehensive report of the mean values and effect sizes obtained with them.

## 3. Materials and methods

### 3.1. Participants

Forty-four individuals took part in this study, comprising 13 females and 31 males, with mean and standard deviation (SD) in age is  $24.43 \pm 4.18$  years. The minimum and maximum age is 19 and 34 years respectively. The sample size was determined based on a 90 % confidence level, 18 % margin of error, and 50 % population proportion at the Indian Institute of Technology Mandi, Himachal Pradesh, India.

Participants were required to meet the inclusion criterion of being healthy individuals between the ages of 18 and 35. Exclusion criteria were any history of diagnosed cardiac or psychiatric disorders, alcohol consumption, or smoking. None of the participants received any medical HRV or RSA biofeedback training.

This study followed the principles of the Declaration of Helsinki [94]. This study was reviewed and approved by Indian Institute of Technology Mandi, Ethical Approval Committee, with the approval number: IITM/IEC(H)/2023/VD/P2. All participants provided written informed consent prior to participate in the study. The research was conducted in 2023, with initial recruitment in February 2023 and final recruitment in May 2023.

### 3.2. Tools, statistical instruments, and measures

HRV data were captured using an emWave Pro Plus device (HeartMath Institute, California, USA) [95]. This Photoplethysmogram (PPG)<sup>1</sup> device records heartbeats through attachment to the earlobe, as illustrated in Fig. 1(a). The software that comes with it can

<sup>1</sup> The PPG modality has been effectively used in the literature for HRV studies [117–124]. In addition, we compared emWave Pro with AD8232 (ECG device) by recording 2 min of HRV data from  $N = 15$  participants under relaxation. Comparing the inter beat interval (IBI) data from PPG and ECG devices, we found a strong mean correlation with the raw IBI values ( $r = 0.95 \pm 0.01$ ). The root mean square error (RMSE) between ECG and PPG values was  $39.20 \pm 2.29$  ms and mean absolute percentage error (MAPE) was  $5.58 \pm 2.20$  %.

show and keep track of a person's heart rate and HRV in real time. After the acquisition, the raw RR peaks were exported as Comma Separated Values (CSV) files from the EM Wave Pro software (Graphical User Interface (GUI) of the software shown in Fig. 1(b)). The files exported from the Emwave Pro software were imported into Kubios [96] for pre-processing and computation of linear and non-linear HRV features. Kubios is a proficient and user-friendly program for analyzing HRV. The settings within the Kubios for the pre-processing of the RR peaks were set at the default values of "low" for "Automatic noise detection" and "automatic" for the "Beat Correction". HRV features were exported to CSV files, which were later extracted for statistical analysis.

### 3.3. Experiment design

As shown in Fig. 2, the experimental procedure comprised three primary phases: introduction, intervention, and feedback. In the introduction phase, participants received brief information regarding the experiment upon arrival, which lasted for less than 2 min. Subsequently, participants spent 2 min completing a straightforward questionnaire containing demographic information relevant to the study. Next, in the intervention stage, participants were randomly and equally divided between two between-subject groups: relaxed-stress (RS) (N = 22) and stress-relaxed (SR) (N = 22). The randomness was established using online random number generator [97]. The groups were matched for age [females: RS ( $22 \pm 3.10$ ) vs SR ( $23.86 \pm 2.61$ ),  $t(11) = -1.17$ ,  $p = 0.26$ ; males: RS ( $23.88 \pm 4.16$ ) vs SR ( $26.27 \pm 4.71$ ),  $t(29) = -1.50$ ,  $p = 0.14$ ] and gender ratio (females: males) [number of females: RS (6) vs SR (7),  $\chi^2(1, N = 13) = 0.08$ ,  $p = .78$ ; number of males: RS (16) vs SR (15),  $\chi^2(1, N = 31) = 0.03$ ,  $p = .86$ ]. In the RS group, a paced breathing task was given for 5 min to create relaxation followed by a 5-min time-based mental calculation task to create stress. In the SR group, the order of the stress and relaxed tasks was reversed. A 15 min wash-out period was incorporated after the initial group intervention (relaxation or stress) to minimize the carry-over effect from the first group to the second group (stress or relaxation). During washout period participants remained in idle state without engaging in any activity. Further elaboration on the relaxation and stress interventions is provided below:

**Relaxation group (paced breathing):** The participants followed an on-screen visual cue to maintain a breathing pace of 6 bpm for a duration of 5 min. The participants inhaled when the cue went up and exhaled when the cue went down, and were instructed to keep their focus on the visual cue at all times during the relaxation intervention.

**Stress group (using a mental arithmetic exam):** Participants undertook a 5-min arithmetic task using PEBL ver. 2.1, as illustrated in the software's GUI depicted in Fig. 1(c) [98,99]. The task was divided into three blocks, each lasting 100 s. The difficulty level of the questions increased with each consecutive block, and participants were required to answer time-constrained arithmetic questions. The results of each question were promptly displayed to the participant following their response.

In each relaxation or stress group, the real-time heart rate of the participants was measured and recorded for offline analysis. In the feedback stage, participants were asked for 2 min to provide feedback regarding the intervention and any other relevant information they wanted to share. The feedback was open-ended and the participants were free to describe anything about the intervention.

### 3.4. HRV parameters

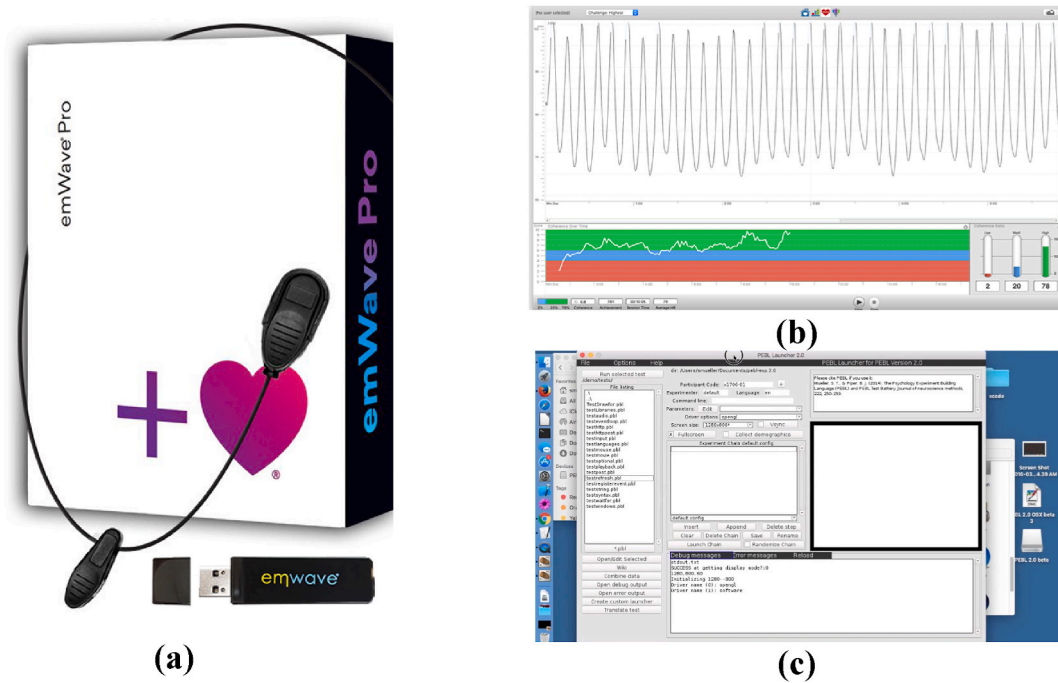
In total, 37 HRV parameters (including linear and non-linear) were computed using Kubios software. Table 1 briefly describes each HRV parameter that was separated into linear and non-linear parameters. For the linear parameters, we used PNS Index, SNS Index, SL, SDNN, MeanHR, SDHR, MinHR, MaxHR, DiffHR, RMSSD, NN50, pNN50, HTI, TINN, DC, DCmod, AC, ACmod, LFap, HFap, LFrp, HFrp, LF/HF, and RESP. The RESP was computed from the HRV data using the Kubios software [100]; therefore, we included it as an HRV parameter. The use of a respiration rate sensor may indicate a true value than that computed from the HRV. This limitation was partially overcome by asking participants to rigidly follow the respiration cue for inhalation and exhalation during relaxation tasks.

For the non-linear parameters, we evaluated SD1, SD2, SD2/SD1, ApEn, SampEn, DFA $\alpha$ 1, DFA $\alpha$ 2, D2, MeanLL, MaxLL, REC, DET, and ShEn.

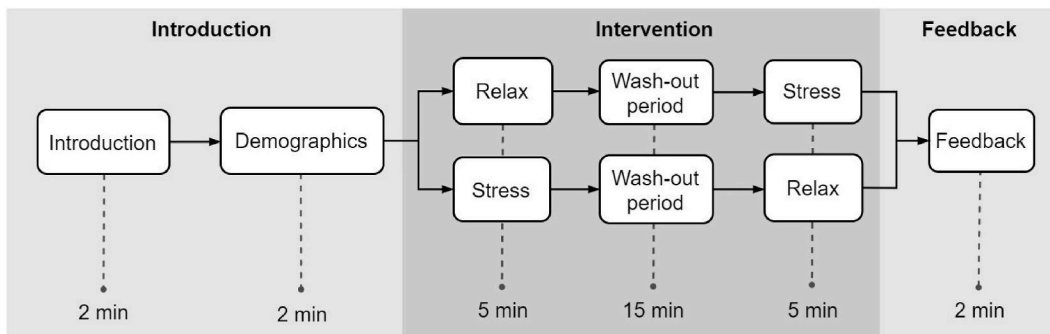
### 3.5. Statistical analysis

The statistical analysis was performed using JASP ver. 0.18 [106] and RStudio ver. 2023.06.1 [107]. Prior to the application of statistical tests, the Shapiro-Wilk test was utilized to assess the normality of differences in repeated HRV parameters observed during the two interventions, ensuring the accuracy of the analyses. Based on the outcomes of this test, either a one-sided paired *t*-test or Wilcoxon signed-rank test was employed to determine statistical significance. One-sided statistical tests were employed based on the findings from the literature, which describe the direction of the change in the HRV parameter under stress, as detailed in Section 2.1. A 95 % confidence interval was established for all comparisons. The power ( $1-\beta$ ) was set at 80 %, with a significance level ( $\alpha$ ) of 5 %. As part of the hypothesis testing process, mean values of the HRV parameters were compared between relaxed and stressed states. The stress groups were pooled across both RS and SR groups for the analyses. In addition, the relaxation groups were pooled across both the RS and SR groups for the analyses.

For the correlation analysis, we used RStudio ver. 2023.06.1 [107]. To reduce the number of correlated pairs and isolate the highly correlated variable counterparts, a criterion of  $|r| \geq 0.9$  and  $p < 0.05$  was chosen. Based on these criteria, the variables were either strongly positively correlated ( $r \geq 0.9$  and  $p < 0.05$ ), strongly negatively correlated ( $r \leq -0.9$  and  $p < 0.05$ ), or uncorrelated ( $|r| \leq 0.9$  or  $p > 0.05$ )



**Fig. 1.** (a) Hardware of the emWave Pro Plus data acquisition system. The ear clip was connected to the earlobe of the participant and the hardware was connected to the computer’s USB port. (b) The emWave Pro Plus data acquisition system software shows the GUI of the visual cue and real-time HRV recording. (c) The GUI of the PEBL software was used to present the mathematical task to the participant.



**Fig. 2.** Experimental design of the study.

### 3.6. Artifact correction

During the data acquisition process, it is common for the RR intervals to be affected by HF artifacts caused by participant movement. These artifacts can significantly impact HRV features, leading to inaccurate values. Short-term TD parameters, absolute power FD parameters, and SD1 are particularly sensitive to these artifacts. To address this issue, we employed artifact correction during pre-processing of RR peaks using Kubios software prior to calculating HRV features. The percentage mean RR beat correction applied in the pre-processing stage varied between interventions: Relax -  $0.79 \pm 1.87$ ; Stress -  $1.73 \pm 1.65$ . The minimum and maximum percentage corrections of the RR beats for the relax intervention were 0 % and 2.96 %, respectively, and those for the stress intervention were 0 % and 5.31 %, respectively. Notably, a larger correction ( $t = 2.49, p = 0.008, d_z = 0.38$ ) was required for the stress group because participants made head movements triggered by their emotional responses to incorrect arithmetic test answers. However, the mean beat correction applied was well below the tolerance limits of 80 % from the literature [36,108].

**Table 1**  
HRV parameters evaluated in the study.

SN	HRV Parameter	Units	Description
<b>Linear parameters</b>			
1	PNS Index	Unitless (UL)	Indicates an increase in PNS activity or resting HRV [38]
2	SNS Index	UL	Indicates a decrease in SNS activity or resting HRV [38]
3	SI	UL	Stress Index (SI) is the square root of Baevsky's stress index [58]
4	SDNN	ms	SD of all NN intervals
5	MeanHR	bpm	Mean heart rate
6	SDHR	bpm	SD of heart rate
7	MinHR	bpm	Minimum HR calculated using moving average of 5 beats
8	MaxHR	bpm	Maximum HR calculated using moving average of 5 beats
9	DiffHR	bpm	Difference between the highest and lowest heart rate.
10	RMSSD	ms	Root mean square of successive RR interval differences
11	NN50	UL	Count of consecutive RR interval pairs with a difference exceeding 50 ms
12	pNN50	%	Proportion of consecutive RR intervals with a difference exceeding 50 ms
13	HTI	UL	HRV triangular index (HTI) is derived by dividing the integral of the density of the RR interval histogram by its height
14	TINN	ms	Triangular interpolation of NN (TINN) measures interval histogram's baseline width [34]
15	DC	ms	Deceleration capacity (DC) is an indicator of cardiac parasympathetic regulation which detects the elongation of the RR interval over 4 consecutive beats [44,45]
16	DCmod	ms	Modified Deceleration capacity is same as DC but takes into account the prolongation of RR interval between 2 consecutive beats [44,45]
17	AC	ms	Acceleration capacity (AC) is an indicator of cardiac sympathetic modulation which elongation of the RR interval over 4 consecutive beats [44,45].
18	ACmod	ms	Modified Deceleration capacity is same as AC but takes into account the prolongation of RR interval between 2 consecutive beats [44,45]
19	LFap	ms <sup>2</sup>	LF band (0.04–0.15 Hz) absolute power
20	HFap	ms <sup>2</sup>	HF band (0.15–0.4 Hz) absolute power
21	LFrp	%	LF band relative power
22	HFRp	%	HF band relative power
23	LF/HF	%	LF and HF band power ratio in terms of their relative powers.
24	RESP	Hz	Respiration rate
<b>Non-linear parameters</b>			
1	SD1	ms	SD of Poincaré plot perpendicular (transverse) to the line-of-identity [61,101]
2	SD2	ms	SD of the Poincaré plot along (longitudinal) the line-of-identity [61,101]
3	SD2/SD1	%	Ratio of SD2 and SD1 [61,101]
4	ApEn	UL	Approximate entropy (ApEn), measuring the regularity and complexity [63]
5	SampEn	UL	Sample entropy (SampEn), measuring the regularity and complexity [63]
6	DFA $\alpha$ 1	UL	Short-term fluctuations in Detrended fluctuation analysis (DFA) [52,102]
7	DFA $\alpha$ 2	UL	Long-term fluctuations in DFA [52,102]
8	D2	UL	Correlation dimension (D2), evaluating minimum quantity of variables needed to establish a model of system dynamics [103]
9	MeanLL	beats	Mean line length from the Recurrence plot analysis (RPA) [104,105]
10	MaxLL	beats	Maximum line length from the RPA [104,105]
11	REC	%	Recurrence (REC) rate from the RPA [104,105]
12	DET	%	Determinism (DET) from the RPA [104,105]
13	ShEn	–	Shannon entropy (ShEn) from the RPA [104,105]

## 4. Results

### 4.1. Comparison of HRV parameters under relax and stress intervention

The data in Table 2 displays the linear and non-linear HRV parameters assessed during the relaxation and stress interventions. Statistical tests revealed that significantly higher mean values were obtained during stress for the SNS Index, Stress Index, MinHR, AC, HFRp, RESP, ApEn, SampEn, and DFA $\alpha$ 2.

Similarly, significantly lower mean values were obtained during the stress intervention for SDNN, SDHR, MaxHR, DiffHR, RMSSD, HTI, TINN, DC, LFap, HFap, LFRp, LF/HF ratio, SD1, SD2, SD2/SD1 ratio, DFA $\alpha$ 1, D2, MeanLL, MaxLL, REC, DET, and ShEn.

However, no significant differences were observed for the PNS Index, MeanHR, NN50, pNN50, DCmod, and ACmod.

### 4.2. Correlation analysis of HRV parameters under relax and stress intervention

Fig. 3(a) and (b) show the heatmap of the correlation between the 37 HRV parameters evaluated in this study during the relaxation and stress interventions, respectively. All levels of correlation from  $-1$  to  $1$  are shown as color-coded squares.

When comparing these 3 cases over the relaxation and stress intervention, we have 9 different comparison sets; for example, two of these conditions are variables strongly positively correlated in relaxation and strongly positively correlated in stress, and variables strongly positively correlated in relaxation and uncorrelated in stress. The above criteria yielded 31 highly correlated pairs, as follows:

**Table 2**  
Mean and SD values of HRV parameters in the relaxation and stress groups.

SN	HRV Parameter	Relax (Mean ± SD)	Stress (Mean ± SD)	t or W statistic	p value	effect size ( $d_e$ )
<b>Linear parameters</b>						
1	PNS Index ↓	-1.18 ± 0.68	-1.18 ± 0.58	-0.10	0.46	-0.01 ○
2	SNS Index ↑ <sup>a</sup>	1.70 ± 1.18	2.27 ± 1.39	3.90	<0.001	0.67 ●●
3	SI ↑	9.35 ± 2.95	13.09 ± 3.25	8.22	<0.001	1.24 ●●
4	SDNN ↓ <sup>a</sup>	62.52 ± 22.16	37.03 ± 10.05	-5.50	<0.001	-0.95 ●●
5	MeanHR ↑	90.85 ± 11.08	92.21 ± 13.10	0.75	0.23	0.13 ∞
6	SDHR ↓ <sup>a</sup>	8.65 ± 2.53	5.33 ± 1.81	-5.52	<0.001	-0.96 ●●
7	MinHR ↑	74.69 ± 9.05	79.91 ± 9.48	5.44	<0.001	0.82 ●●
8	MaxHR ↓ <sup>a</sup>	110.83 ± 12.50	108.53 ± 15.51	-2.18	0.01	-0.38 ●
9	DiffHR ↓	36.14 ± 8.09	28.62 ± 10.30	-5.43	<0.001	-0.82 ●●
10	RMSSD ↓	43.91 ± 14.24	40.23 ± 10.92	-1.70	0.049	-0.26 ∞
11	NN50 ↓	83.14 ± 42.97	74.20 ± 33.56	-1.41	0.08	-0.21 ∞
12	pNN50 ↓	21.15 ± 12.32	18.81 ± 9.78	-1.33	0.09	-0.20 ∞
13	HTI ↓ <sup>a</sup>	15.21 ± 3.70	9.68 ± 2.40	-5.32	<0.001	-0.92 ●●
14	TINN ↓	294.25 ± 93.86	191.98 ± 47.13	-7.87	<0.001	-1.19 ●●
15	DC ↓ <sup>a</sup>	35.25 ± 30.66	21.54 ± 10.01	-2.31	0.01	-0.40 ●
16	DCmod ↓ <sup>a</sup>	47.43 ± 21.89	41.21 ± 11.65	-0.57	0.29	-0.10 ∞
17	AC ↑ <sup>a</sup>	-33.40 ± 22.81	-22.33 ± 9.72	3.17	<0.001	0.55 ●
18	ACmod ↑	-44.77 ± 15.32	-42.05 ± 11.22	1.24	0.11	0.19 ∞
19	LFap ↓ <sup>a</sup>	3322.61 ± 2889.84	629.77 ± 508.38	-5.51	<0.001	-0.95 ●●
20	HFap ↓ <sup>a</sup>	753.75 ± 1105.20	455.26 ± 368.10	-2.26	0.01	-0.34 ●
21	LFrp ↓	81.05 ± 14.89	53.59 ± 12.97	-10.34	<0.001	-1.56 ●●
22	HFrp ↑	16.39 ± 15.24	39.90 ± 14.36	8.58	<0.001	1.29 ●●
23	LF/HF ↓ <sup>a</sup>	8.85 ± 6.46	1.83 ± 1.84	-5.45	<0.001	-0.94 ●●
24	RESP ↑	0.26 ± 0.09	0.35 ± 0.07	5.63	<0.001	0.85 ●●
<b>Non-linear parameters</b>						
1	SD1 ↓	31.09 ± 10.09	28.49 ± 7.76	-1.69	0.049	-0.25 ∞
2	SD2 ↓ <sup>a</sup>	82.65 ± 30.21	43.52 ± 13.42	-5.52	<0.001	-0.96 ●●
3	SD2/SD1 ↓ <sup>a</sup>	2.66 ± 0.48	1.56 ± 0.43	-5.40	<0.001	-0.94 ●●
4	ApEn ↑ <sup>a</sup>	1.17 ± 0.18	1.24 ± 0.16	2.81	0.002	0.49 ●
5	SampEn ↑	1.51 ± 0.27	1.99 ± 0.15	10.44	<0.001	1.57 ●●
6	DFAα1 ↓	1.47 ± 0.16	0.95 ± 0.22	-12.99	<0.001	-1.96 ●●
7	DFAα2 ↑	0.25 ± 0.11	0.45 ± 0.13	9.69	<0.001	1.46 ●●
8	D2 ↓	3.02 ± 0.84	2.29 ± 1.36	-3.64	<0.001	-0.55 ●●
9	MeanLL ↓	11.97 ± 2.72	7.23 ± 1.44	-9.64	<0.001	-1.45 ●●
10	MaxLL ↓ <sup>a</sup>	253.82 ± 121.97	69.39 ± 63.81	-5.37	<0.001	-0.93 ●●
11	REC ↓	28.23 ± 3.79	20.54 ± 4.80	-8.45	<0.001	-1.27 ●●
12	DET ↓	98.17 ± 0.82	94.98 ± 1.66	-10.73	<0.001	-1.62 ●●
13	ShEn ↓	3.15 ± 0.22	2.68 ± 0.22	-9.15	<0.001	-1.38 ●●

Note.

↓ Alternative hypothesis specifies that the stress measure is less than the relax measure.

↑ Alternative hypothesis specifies that the stress measure is greater than the relax measure.

●● Large Effect; ● Moderate Effect; ∞ Small Effect; ○ Trivial Effect.

<sup>a</sup> Shapiro-Wilk test shows deviation from normality in the difference in HRV parameters taken during the relaxation and stress intervention. A one-sided paired t-test (t) or the Wilcoxon test (W) was applied for normal and non-normal data respectively.

since there are two Pearson's r values, we have reported the lowest |r| value from comparing variables under relaxation and stress. The p values were <0.05, for all the reported results.

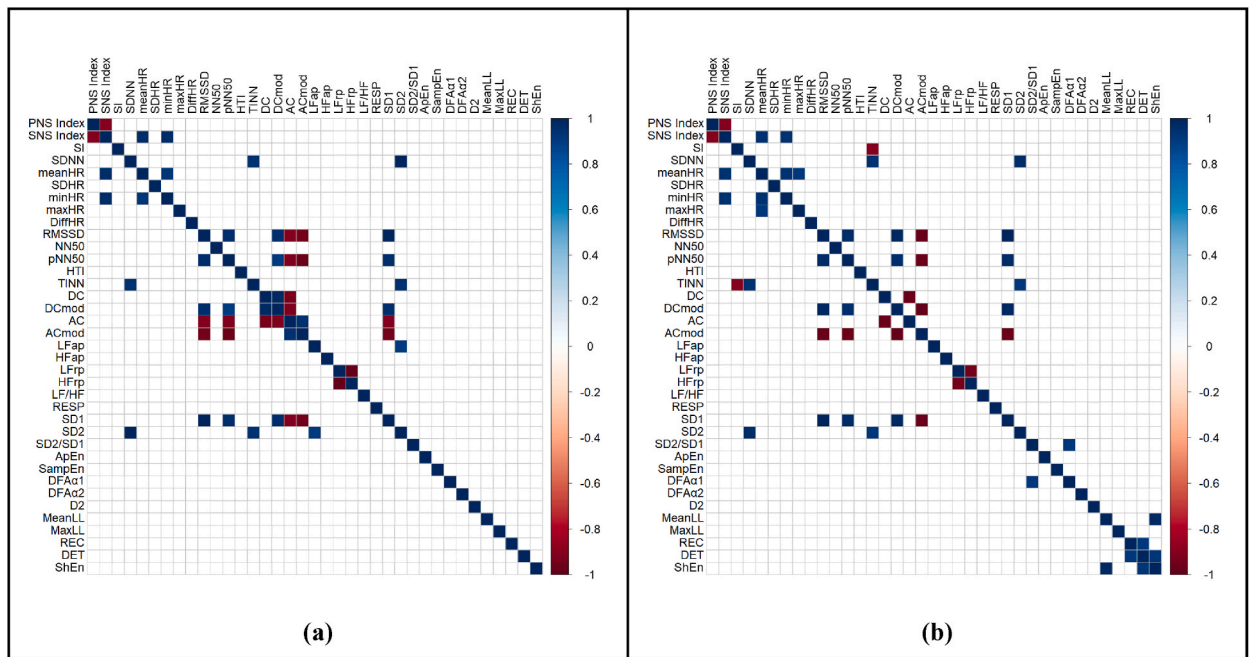
The variable pairs that were strongly positively correlated during both relaxation and stress are SNS Index and meanHR,  $r(35) = 0.95, p < 0.001$ ; SNS Index and minHR,  $r(35) = 0.95, p < 0.001$ ; SDNN and TINN,  $r(35) = 0.94, p < 0.001$ ; SDNN and SD2,  $r(35) = 0.97, p < 0.001$ ; meanHR and minHR,  $r(35) = 0.93, p < 0.001$ ; RMSSD and pNN50,  $r(35) = 0.96, p < 0.001$ ; RMSSD and DCmod,  $r(35) = 0.95, p < 0.001$ ; RMSSD and SD1,  $r(35) = 1, p < 0.001$ ; pNN50 and DCmod,  $r(35) = 0.90, p < 0.001$ ; pNN50 and SD1,  $r(35) = 0.96, p < 0.001$ ; TINN and SD2,  $r(35) = 0.92, p < 0.001$ ; DCmod and SD1,  $r(35) = 0.96, p < 0.001$ ;

The variable pairs that were strongly negatively correlated during both relaxation and stress were the PNS Index and SNS Index ( $r(35) = -0.90$ ), RMSSD and ACmod ( $r(35) = -0.95$ ), pNN50 and ACmod,  $r(35) = -0.96, p < 0.001$ ; DC and AC,  $r(35) = -0.94, p < 0.001$ ; ACmod and SD1,  $r(35) = -0.95, p < 0.001$ ; and LFRp and HFrp,  $r(35) = -0.94, p < 0.001$ .

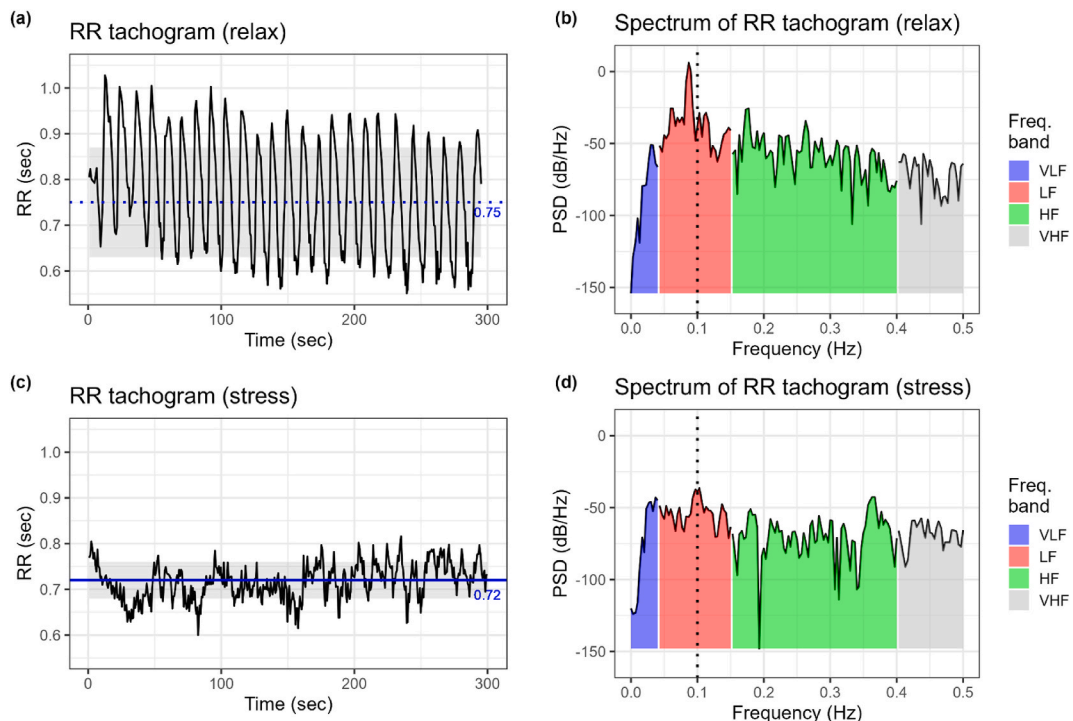
The variable pairs that were strongly positively correlated during relaxation and uncorrelated during stress were DC and DCmod (stress:  $r(35) = 0.75$ ), AC and ACmod (stress:  $r(35) = 0.71$ ), and LFap and SD2 (stress:  $r(35) = 0.84$ ). The variable pairs that were strongly negatively correlated during relaxation and uncorrelated during stress were RMSSD and AC(stress:  $r(35) = -0.57$ ), pNN50 and AC (stress:  $r(35) = -0.65$ ), DCmod and AC(stress:  $r(35) = -0.67$ ), and AC and SD1(stress:  $r(35) = -0.57$ ).

The variable pairs that were uncorrelated during relaxation and strongly positively correlated during stress were MeanHR and MaxHR (relax:  $r(35) = 0.88$ ), SD2/SD1 and DFAα1 (relax:  $r(35) = 0.86$ ), MeanLL and ShEn (relax:  $r(35) = 0.77$ ), REC and DET (relax:  $r(35) = 0.77$ ), and DET and ShEn (relax:  $r(35) = 0.77$ ). The variable pairs that were uncorrelated during relaxation and strongly negatively correlated during stress were SI and TINN (relaxation:  $r(35) = -0.90$ ) and DCmod and ACmod (relaxation:  $r(35) = -0.89$ ).





**Fig. 3.** Correlation plot of HRV parameters during (a) relaxation and (b) stress. Only significant correlations ( $p < 0.05$ ) are shown in the figure. The variables are arranged as shown in Table 1. Each square’s color signifies the correlation between each pair of variables, ranging from red (−1) to blue (+1). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)



**Fig. 4.** Data from one of the participants showing the TD (left) and FD (right) representation of the RR tachogram taken during 5 min of relaxation (top) and stress (bottom). In TD plots (left), RR intervals are shown with a mean (dotted blue line) and SD (gray band). In the FD plots (right), four frequency bands are shown in the spectrum of the RR tachogram: Very Low Frequency (VLF), Low Frequency (LF), High Frequency (HF), and Very High Frequency (VHF). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

### 4.3. RSA under relaxation and stress

The RSA or cardiac coherence is characterized by a shortening of the R–R interval during inspiration and a prolongation during expiration. The dominance of the RSA during the relaxation interventions can be visualized as shown in Fig. 4(a–d), which displays the TD and FD of the RR tachogram of one of the participants under relaxation and stress. In the TD, the variations in the beat-to-beat RR during relaxation (Fig. 4(a)) were more pronounced compared to the stress (Fig. 4(c)). For the participant, the mean and SD values of the RR interval was  $0.75 \pm 0.12$  ms in relaxation and  $0.72 \pm 0.04$  ms in stress. The SD of the RR intervals is larger during relaxation which is an indicator of the heightened amplitude of RSA at paced breathing of 0.1 Hz. This SD diminishes during stress due to uncontrolled breathing in several frequency bands. This effect can also be noted from the frequency plot. The absolute power in the LF band was  $13066 \text{ ms}^2$  in relaxation and  $349 \text{ ms}^2$  in stress. The absolute power in the HF band was  $917 \text{ ms}^2$  in relaxation and  $211 \text{ ms}^2$  in stress. The larger peak near 0.1 Hz in the LF band during relaxation (Fig. 4(b)) as compared to that during stress (Fig. 4(d)) is due to 0.1 Hz rhythm in the RR tachogram, which is caused by the synchrony of heartbeats with a respiration rate of 0.1 Hz. Similarly, the LF band relative power was 93.27 % for relaxation and 54.16 % for stress. The HF band relative power was 6.54 % for relaxation and 32.75 % for stress.

## 5. Discussion

The primary objective of this research was to assess the hypothesis that, by examining a range of linear and non-linear HRV parameters, there would be more notable enhancements in HRV during mentally relaxed tasks compared to stressful ones. Various HRV parameters were found to significantly improve during relaxation intervention. Improvement was assessed based on whether an HRV parameter was expected to increase or decrease during paced breathing compared with the mental arithmetic task. Here, HRV parameter changes due to paced breathing may be influenced by either the vagal or sympathetic system.

### 5.1. Linear HRV parameters

Linear HRV parameters, including SNS Index, SI, MinHR, AC, HFrp, and RESP, exhibited significantly higher mean values in the stress group.

The SNS Index was determined using the mean RR interval, SI, and SD2 in normalized units [38]. The significant decrease in the SD2 parameter with a large effect size may have contributed to the significant difference obtained with the computed SNS Index. SI represents sympathetic or central regulatory activity [58]. The higher mean SI values in the stress group indicated sympathetic activation. It should be noted that similar geometric parameters (HTI) also exhibited a significant change among the interventions.

The mean of MinHR values were significantly higher in the stress group. This may be due to the RSA (or cardiac coherence), which is pronounced during paced breathing and exhibits larger fluctuations in HR than during stressful tasks [35]. Therefore, MinHR during stress was higher because the fluctuation around the mean HR value was likely minimal during stress [35]. Moreover, serving as a comprehensive gauge of all periodic acceleration-related oscillations, the elevated average value of the AC indicates cardiac sympathetic modulation, as it encompasses the reduction of the RR interval over 4 consecutive beats [44,45]. The significant increase in HFrp may be related to the LF band power (see further discussion below).

The mean values of the respiration rate during stress were also higher than those during relaxation, with a large obtained effect size. This phenomenon might be attributed to the participants engaging in paced breathing, resulting in mean respiration rates closely aligned with the resonating frequency of 0.1 Hz. It is worth noting that the choice of 0.1 Hz as the respiration rate is motivated by prior literature, where similar assumptions have been made for paced breathing [4,109–113]. Furthermore, literature demonstrates that cardiac coherence can be maximized via vagal predominance to reduce stress [109]. However, it may also be true that at 0.1 Hz respiration rate, it may be difficult to discern vagal and sympathetic regulations as reasons for increased cardiac coherence. Thus, it is likely that the cardiac coherence could be a result of either vagal or sympathetic regulations. Furthermore, the average RESP values are not closely approaching 0.1 Hz for the paced breathing group. Although the visual cue was set to oscillate at a rate of 0.1 Hz during the paced breathing experiment, the deviation of the RESP values from this rate may be attributed to the fact that the respiration rate is derived from the HRV signal and may not always be exact [100]. However, this can be addressed in the future studies by using a respiration rate sensor. It is noteworthy that the participants in the paced breathing group were able to maintain lower average RESP values than those in the stress group, indicating their adherence to the paced breathing protocol. The use of HRV to derive respiration rate may be seen as a limitation in this work, and in the future, we will use an external breathing rate sensor.

The linear HRV parameters found to have significantly lower mean values in the stress group were SDNN, SDHR, MaxHR, DiffHR, RMSSD, HTI, TINN, DC, LFap, HFap, LFrp, and LF/HF ratio.

The lower mean value of SDNN appears to be an indicator of sympathetic dominance and reduced vagal tone. Moreover, the obtained range (20.02 ms–113.86 ms) of the computed SDNN is comparable to the nominal values of 32 ms–93 ms, as reported in the literature [70]. Furthermore, SDNN is derived from a brief 5-min recording, where the predominant source of variation arises from parasympathetically mediated RSA, especially in the context of slow-paced breathing protocols [39].

The lower values obtained with the SDHR also indicate the diminishing effects of the RSA in the stress group than in the paced breathing group, with relatively minimal fluctuations and thus a lower SD in the HR than in the MeanHR [35]. Similar to MinHR, MaxHR was found to be greater during paced breathing because of the larger RSA amplitude and hence larger fluctuations around the mean HR value [35], which is evident from the lower mean value obtained for maxHR in the stress group than in the paced breathing group.

The lower mean values of the DiffHR obtained in the stress group may be due to the higher respiration rates exhibited in the stress group, which produces lower RSA amplitudes [6]. The average variation between the maximum and minimum HR within each respiratory cycle is especially sensitive to changes in breathing rate, independent of vagus nerve activity, and reflects the RSA rather than directly indicating vagal tone [6].

The RMSSD was also lower in the stress group. The obtained range (25.10 ms–85.41 ms) of RMSSD computed is comparable to the nominal values of 19 ms–75 ms, as reported in the literature [70]. RMSSD is known to correlate with HF in the FD parameters of HRV (see also Fig. 3(a) and (b)) [40]. This is also evident from the statistical significance of HF. However, the effect size obtained with the RMSSD was small compared with the moderate effect size obtained with the HF parameter. This finding may be because RMSSD is comparatively less influenced by respiration when compared to HF band parameters [114]. In the literature, a decrease in RMSSD may indicate a decrease in vagally mediated changes reflected in HRV [39,40]. However, in our results, the enhancement in the RMSSD was primarily due to the heightened amplitude of the cardiac coherence (RSA) due to 0.1 Hz respiration rate, and it may not directly indicate the vagal dominance.

HTI also decreased under stress. Moreover, the reported HTI values corroborate with the normal values reported in the literature, where  $HTI \leq 20.42$  and  $RMSSD \leq 0.068$  predicted normal heart rhythm [43,115], and the inclusion criteria for the study were that participants did not have any reported health condition. Together with RMSSD, HTI can distinguish between normal heart rhythms and arrhythmias [115] and access relaxed mental states after intervention, such as listening to classical music (ragas) [3]. Likewise, lower mean values of TINN obtained in the stress group indicated sympathetic activation during stress [43].

The DC also exhibited lower mean values than the stress group. This result signifies a reduced vagal modulation under stress [44, 45]. Both LFap and LFrp exhibited lower mean values in the stress group in comparison to the paced breathing group. The LF range primarily mirrors baroreceptor activity on the time of resting phases [1] and is expected to increase under stress [6]. However, during instances of reduced respiration rates, vagal activity can easily generate oscillations in heart rhythms that extend into the LF band [72–74]. This reasoning explains the increase in LFap and LFrp during paced breathing. The HF power on the other hand is relatively less affected by the intervention. HFap also decreased in the stress group, but with a moderate effect compared to the large effect of LFap. Slow and deep breathing, within the 9–24 bpm, can change HF power without affecting vagal tone [6,47]. In our study, participants breathed at exactly 0.1 Hz with the help of a visual cue. This likely increased the LF power but the HF band starting from 0.15 Hz saw a reduced share of the power.

The LF and HF power distributions can be explained by the percentage shares (LFrp and HFrp) of the power in the respective bands. The obtained LFrp was approximately 5 times higher than HFrp during paced breathing. This supports the premise that paced breathing at 0.1 Hz caused the dominance of the RSA to fall in the LF spectrum and eventually enhances the LF power. However, during the stressful task, the obtained LFrp was almost the same (1.5 times) as the HFrp. This was perhaps due to breathing at wider rates during stress tasks and, thus, distributed RSA frequency power occurring over the wider spectrum that encompasses both the LF and HF bands.

Although there was a significant but moderate increase in HFrp during stressful tasks compared to paced breathing, it should be contextually evaluated along with LFrp. HFrp decreased during paced breathing, likely due to the added share of LFrp, which occurs due to paced breathing. This apparently made HFrp significantly higher during the stressful tasks. The interplay between the relative proportions of LFrp and HFrp skewed the LF/HF ratio. The mean value of the LF/HF ratio was significantly lower during stress, which is contrary to the increase in the LF/HF ratio during sympathetic dominance in stressful tasks [6,34,59]. Most likely, the larger share of band power in the LF range during paced breathing decreased the LF/HF ratio during stressful tasks compared to paced breathing. It should be noted that the absolute and relative power comparison of the spectral bands largely indicates the contrast between paced breathing and stress tasks, and is heavily influenced by the elevated cardiac coherence. It is less likely due to the sole influence of vagal modulation.

Furthermore, no significant differences were observed for the PNS Index, MeanHR, NN50, pNN50, DCmod, and ACmod.

The equivalent mean values obtained with the PNS Index during the interventions may be the result of an insignificant change in the HR and the small effect size obtained with the RMSSD and SD1, which are some of the parameters used to compute the PNS Index. Similarly, the mean HR showed no difference among the groups, which corroborates with the findings in the literature that with slow, deep breathing (6 bpm in this study), increased fluctuations in the HR could be observed without a significant change in the mean HR value [6].

Similarly, the potential contributors to the equivalence of the mean values of NN50 across groups can be slow and deep breathing, which can change the HRV parameters without affecting vagal tone [6,47]. The explanation for the similar average pNN50 values across the groups is that slow and deep breathing at the resonating frequency may contribute to the small effect size obtained with the pNN50. This is because changes in respiratory rate and volume can significantly impact HRV parameters without necessarily affecting vagal tone [6,47].

The equivalence of the mean values of DCmod and ACmod obtained for the groups may be attributed to the reduced window length used to compute DCmod as well as ACmod, which may not effectively capture the deceleration over a wider time range [44,45].

## 5.2. Non-linear HRV parameters

Non-linear parameters, including entropy, are utilized to quantify the degree of regularity and unpredictability of fluctuations over a time series. Although non-linear measures may differentiate between paced breathing and stress tasks, they may not directly point towards vagal dominance. As a result, the increase in non-linear measures may not be the result of sole vagal dominance. Rather, it could be due to either the vagal or the sympathetic system.

The non-linear HRV parameters that were found to have significantly higher mean values in the stress group were ApEn, SampEn, and DFA $\alpha$ 2.

We observed that ApEn during stress had significantly higher mean values than during relaxation. Lower ApEn values indicate a more regular and predictable signal, whereas higher ApEn values suggest increased unpredictability in successive RR interval fluctuations [63–65]. The regularity and predictability in the HRV during relaxation tasks were likely due to the enhanced RSA, which can be seen as sinusoidal variations with increased peak-to-peak values compared to the jittery HRV under stress.

SampEn also increased significantly in the stress group. Its interpretation can be similar to that of ApEn, where it is expected to increase in low predictable and chaotic signals such as HRV during stress [63,66]. DFA $\alpha$ 2 also exhibited a higher mean value in the stress group than that in the relaxation group. According to literature, DFA $\alpha$ 2 shows higher values during the mentally active state [102], in high-risk cardiac patients [52], and during exercise [53].

The non-linear HRV parameters that were found to have significantly lower mean values in the stress group were SD1, SD2, SD2/SD1 ratio, DFA $\alpha$ 1, D2, MeanLL, MaxLL, REC, DET, and ShEn, respectively.

All the Poincaré plot indices showed lower mean values in the stress group, but with a small effect size with SD1 and a large effect size with SD2 and SD2/SD1 ratio, and also correlated with the TD and FD parameters (See Fig. 3(a) and (b)). The SD1 is similar to baroreflex sensitivity (BRS), RMSSD, DiffHR, pNN50, SDNN, and power in the LF and HF bands [6,25]. SD2 is similar to LF power and BRS [24,61]. Finally, the SD2/SD1 ratio is similar to the LF/HF ratio [6,116]. SD1 had a lower mean value in the stress group, which corroborates the findings in the literature that SD1 decreases during stress [50] and is considered a pure measure of parasympathetic activity [51].

Similar to its TD and FD counterparts, RMSSD and HF power, SD1 achieved statistically small effect sizes. Similarly, SD2 showed a significantly lower mean value in the stress group. Although SD2 length reflects the sympathetic modulation [24], the decrease in the SD2 during stress could be attributed to the paced breathing at 0.1 Hz that covered the LF spectrum. Due to paced breathing, the relative length of SD2 during the relaxation task was more (1.8 times) than during stress. With the SD2/SD1 ratio, we observed a significant decrease in the stress group. This finding is contrary to the literature, where SD2/SD1 decrease represents a decrease in sympathetic activity [61,62], which primarily occurs during relaxation. The skewed SD2/SD1 ratio is likely the result of the relative balance between the SD2 and SD1 values, which is also seen with the LF/HF ratio in the linear parameters. Although geometric parameters are more robust to artifacts, we have seen the correlation of the geometric parameters with the FD parameters, which corroborates with earlier literature [50].

DFA $\alpha$ 1 showed lower mean values in the stress group, which was likely due to parasympathetic withdrawal during stress [53].

The D2 shows significantly lower mean values in the stress group. It exhibits a positive correlation with the RMSSD and power in the HF band, and its values are observed to decline during stress, as depicted in Fig. 3(b) [53,54].

All HRV parameters derived from RPA (MeanLL, MaxLL, REC, DET, and ShEn) also showed a significant decrease under stress. High RPA values imply low complexity in the dynamics of the system [105]. The lower values of the RPA measures during stress corroborate with the literature [55,56].

### 5.3. Correlation of HRV parameters

It is evident from the correlation results that a significant number of HRV parameters were correlated during both relaxation and stress interventions. A general visual trend of a decrease in correlation values was observed during stress compared to the relaxation intervention. The correlation results can be summarized by grouping HRV parameters according to their increase or decrease during stress intervention. These two groups were previously discussed in the background section, where they were derived as per earlier studies. Most of the grouped parameters showed a high intragroup positive correlation and a high intergroup negative correlation. For example, the RMSSD was positively correlated with the PNS Index, SDNN, SDHR, NN50, HTI, TINN, DC, DCmod, HFap, HFrp, SD1, SD2, and D2. All these parameters form intra-group parameter comparisons, which are expected to decrease under stress. Similarly, RMSSD was negatively correlated with the SNS Index, SI, AC, ACmod, LFRp, RESP, SD1, and APEn. These all form intergroup parameter comparisons.

The correlation analysis results indicated a strong correlation among the TD, GD, and non-linear parameters, which remained consistent during both relaxation and stress. These correlating pairs can be used in multivariate analysis to classify relaxation and stress interventions. From the results, the strongly correlating parameters that decrease under stress were the PNS Index, SDNN, RMSSD, pNN50, TINN, SD2, DCmod, SD1, SD2, LFRp, and DC. Likewise, the strongly correlated parameters that increased under stress were the SNS Index, Mean HR, Min. HR, AC, ACmod, and HFrp. Interestingly, the DC and AC parameters become less correlated with others compared to their derived parameters DCmod and ACmod. However, DCmod and ACmod were insignificant when compared separately in the relaxing and stress interventions. In addition, DC and AC become uncorrelated with their derived parameters DCmod and ACmod, respectively, under stress. The results showed that DC and AC were better suited for comparing relaxation and stress interventions than their modified parameters DCmod and ACmod.

Another interesting finding was that the parameters become strongly correlated under relaxation and become uncorrelated under stress and vice versa. These correlations suggest the effects of interventions on the relationship between the parameters. For example, DC and DCmod, AC and ACmod, LFap, and SD2 are strongly positively correlated under relaxation and become uncorrelated under stress. Paced breathing may be the likely reason for this strong relationship during relaxation. LFap shows heightened power owing to RSA effects, which is also reflected in its non-linear counterpart. However, during stress, the LFap power decreased significantly more than SD2, as evident from their t statistic and effect size from Table 2; their relationship likely became less strong than our set threshold ( $|r| \geq 0.9$ ) and was shown to be uncorrelated. Similarly, the enhanced RSA, which causes smoothness during relaxation, and

diminished RSA, which causes increased jitteriness during stress, likely affected the computations of DC and AC with their modified parameters because the modified parameters use 2 beats instead of 4 beats in the original parameter. Similarly, AC was strongly negatively correlated and became uncorrelated under stress with RMSSD, pNN50, DCmod, and SD1. In contrast, the non-linear parameters show a strong positive correlation under stress and become uncorrelated in relaxation.

Another intriguing finding was the dominance of respiratory sinus arrhythmia (RSA), as indicated by the SD of RR peaks, during relaxation as opposed to stress. This observation potentially elucidates some of the correlations observed among the HRV parameters. As discussed previously, during the discussion of the linear HRV parameters, the FD HRV parameter values show different results, as expected from the literature. This was because of the larger value of RSA during relaxation (paced breathing at 0.1 Hz), which was absent during the stress intervention. Therefore, the respiration frequency spills over to the LF range and exhibits an elevated LF band power. Therefore, during relaxation, we observed a moderate to strong positive correlation between LFap and the PNS Index, SDNN, RMSSD, NN50, TINN, DC, DCmod, RESP, SD1, and DFA $\alpha$ 1. This positive correlation between LFap and the other variables decreases during stress. The same RSA effect can be seen with the increase in the correlation of RESP with HFap during stress because of the increase in respiration rate and subsequent increases in overlapping respiration frequency band with HF band power compared to the relaxation group. This effect was also observed in the non-linear counterpart of the frequency bands. From the literature, LF and HF correlate with SD2 and SD1, respectively [24,25], and the correlation results also corroborate these findings.

## 6. Conclusion and future work

HRV parameters are valuable indicators of an individual's physiological response to stress and relaxation. During a mentally stressful state, HRV parameters differ significantly from those during a mentally relaxed state. Under stress, a reduced vagal tone indicates an elevated sympathetic response. Conversely, enhanced vagal tone during relaxation points toward a healthier autonomic balance and increased parasympathetic response. However, the change in mean values of some HRV parameters during an intervention is not straightforward and may depend on the type of interventions. For example, the slow and deep breathing techniques commonly used for mental relaxation during baseline HRV measurements may also increase the RSA component and affect FD HRV parameters. In this study, we compared the effects of relaxation and stress interventions on the HRV parameters. Paced breathing at a resonating 0.1 Hz frequency was used to induce relaxation, while time-based mental arithmetic tasks were used to induce stress. All the TD, geometric, and non-linear parameters showed an increase or decrease in their mean values across the two different interventions, as reported in the literature and prior studies. However, the FD parameters were affected by breathing at the resonant frequency during relaxation. The same is also observed in the correlation analysis of the HRV parameters during the relaxation and stress states, where the GD parameters are correlated with the TD parameters and are affected by paced breathing. The results indicated that the observed difference in the mean values of the LF and HF absolute powers and the LF/HF ratio were influenced by breathing at a resonant frequency, and the results should be interpreted accordingly. This study has a few limitations. One is the use of PPG instead of ECG for data acquisition. For this purpose, we presented our additional testing of the PPG device used for data acquisition with an ECG device, where we found a strong correlation between the metrics obtained by both devices. Another limitation is the extraction of the respiration rate from the HRV data, which can be mitigated in future studies using an additional respiration rate sensor and other intervention groups at specific breathing rates. Also, we did not use any subjective psychological measures to assess the mental state of the participants. In future studies, subjective measures may be correlated with HRV parameters. Further, a control group with an idle sitting position may be used in the future to compare it with the upper and lower bounds of HRV parameters. Since our aim was to contrast relaxation and stress, the comparison with idle sitting can be considered for future research. As most of the HRV parameters significantly differed with large effect sizes, the interventions used in the study may be employed as baseline measurement interventions for the upper and lower limits of HRV parameters. Thus, monitoring HRV can provide insights into an individual's overall stress levels and well-being, enabling the adoption of appropriate strategies for managing stress and promoting relaxation. Future HRV research may also delve into other relaxation protocols such as pranayama and deep breathing and stress interventions such as n-back and memory recall tasks and social stress that may similarly influence HRV parameters. Additionally, investigating the effects of social interactions, environmental noise, and virtual reality interventions on HRV could lead to novel insights into stress management and relaxation strategies.

## Ethics statement

This study was reviewed and approved by Indian Institute of Technology Mandi, Ethical Approval Committee, with the approval number: IITM/IEC(H)/2023/VD/P2. All participants/patients (or their proxies/legal guardians) provided written informed consent prior to participate in the study.

## Data availability statement

The authors will provide the raw data upon request, without any undue reservation.

## Funding

IIT Mandi iHub and HCl Foundation, and Indian Institute of Technology Mandi (IIT Mandi) provided the essential financial and computational resources for this study.

## CRediT authorship contribution statement

**Kulbhushan Chand:** Writing – review & editing, Writing – original draft, Validation, Methodology, Formal analysis, Conceptualization. **Shilpa Chandra:** Writing – original draft, Data curation, Conceptualization. **Varun Dutt:** Writing – review & editing, Supervision, Resources, Funding acquisition.

## Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

Kulbhushan Chand reports the infrastructure, tools, and other support were provided by IIT Mandi iHub and HCl Foundation. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Acknowledgment

The research is supported by IIT Mandi iHub and HCl Foundation, and Indian Institute of Technology Mandi (IIT Mandi). We are grateful for the infrastructure, tools, and resources provided by IIT Mandi iHub and HCl Foundation.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.heliyon.2024.e32195>.

## References

- [1] R. Mccraty, F. Shaffer, Heart rate variability: new Perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk, *Glob. Adv. Health Med.* 4 (1) (Jan. 2015) 46–61, <https://doi.org/10.7453/gahmj.2014.073>.
- [2] H. Tsuji, et al., Impact of reduced heart rate variability on risk for cardiac Events, *Circulation* 94 (11) (Dec. 1996) 2850–2855, <https://doi.org/10.1161/01.CIR.94.11.2850>.
- [3] S. Chandra, K. Chand, V. Dutt, Impact of Indian classical Raga in Immersive Environments on human psycho-physiological parameters, in: *Proceedings Of the 16th International Conference On Pervasive Technologies Related To Assistive Environments*, in PETRA '23, Association for Computing Machinery, New York, NY, USA, Aug. 2023, pp. 746–753, <https://doi.org/10.1145/3594806.3596555>. Available: <https://dl.acm.org/doi/10.1145/3594806.3596555>. (Accessed 11 August 2023).
- [4] K. Chand, A. Khosla, Efficacy of using retro games in multimodal biofeedback systems for mental relaxation, *Int. J. Gaming Comput.-Mediat. Simul. IJGCMS* 14 (1) (Jan. 2022) 1–23, <https://doi.org/10.4018/IJGCMS.295874>.
- [5] H.A. Paul, Biofeedback: a practitioner's guide, fourth edition, edited by M. Schwartz & F. Andrasik, *Child Fam. Behav. Ther.* 39 (2) (Apr. 2017) 161–170, <https://doi.org/10.1080/07317107.2017.1307683>.
- [6] F. Shaffer, J.P. Ginsberg, An overview of heart rate variability metrics and norms, *Front. Public Health* 5 (2017), <https://doi.org/10.3389/fpubh.2017.00258>. Available: <https://www.frontiersin.org/articles/10.3389/fpubh.2017.00258/full>. (Accessed 11 January 2021).
- [7] S. Battaglia, J.F. Thayer, Functional interplay between central and autonomic nervous systems in human fear conditioning, *Trends Neurosci.* 45 (7) (Jul. 2022) 504–506, <https://doi.org/10.1016/j.tins.2022.04.003>.
- [8] S. Battaglia, C. Nazzi, J.F. Thayer, Heart's tale of trauma: fear-conditioned heart rate changes in post-traumatic stress disorder, *Acta Psychiatr. Scand.* 148 (5) (2023) 463–466, <https://doi.org/10.1111/acps.13602>.
- [9] H.A. Young, D. Benton, Heart-rate variability: a biomarker to study the influence of nutrition on physiological and psychological health? *Behav. Pharmacol.* 29 (2 and 3) (Apr. 2018) 140, <https://doi.org/10.1097/FBP.0000000000000383>.
- [10] J. Koenig, J.F. Thayer, Sex differences in healthy human heart rate variability: a meta-analysis, *Neurosci. Biobehav. Rev.* 64 (May 2016) 288–310, <https://doi.org/10.1016/j.neubiorev.2016.03.007>.
- [11] M.N. Jarczok, et al., Investigating the associations of self-rated health: heart rate variability is more strongly associated than inflammatory and other frequently used biomarkers in a cross sectional occupational sample, *PLoS One* 10 (2) (Feb. 2015) e0117196, <https://doi.org/10.1371/journal.pone.0117196>.
- [12] K. Chand, A. Khosla, BioNES: a plug-and-play MATLAB-based tool to use NES games for multimodal biofeedback, *SoftwareX* 19 (Jul. 2022) 101184, <https://doi.org/10.1016/j.softx.2022.101184>.
- [13] P.R. Steffen, T. Austin, A. DeBarros, T. Brown, The impact of resonance frequency breathing on measures of heart rate variability, blood pressure, and mood, *Front. Public Health* 5 (2017). Available: <https://www.frontiersin.org/articles/10.3389/fpubh.2017.00222>. (Accessed 1 April 2023).
- [14] P.M. Lehrer, R. Gevirtz, Heart rate variability biofeedback: how and why does it work? *Front. Psychol.* 5 (2014) <https://doi.org/10.3389/fpsyg.2014.00756>. Available: <https://www.frontiersin.org/articles/10.3389/fpsyg.2014.00756>. (Accessed 18 August 2023).
- [15] H.J. Yoo, et al., Heart rate variability (HRV) changes and cortical volume changes in a randomized trial of five weeks of daily HRV biofeedback in younger and older adults, *Int. J. Psychophysiol.* 181 (Nov. 2022) 50–63, <https://doi.org/10.1016/j.ijpsycho.2022.08.006>.
- [16] J.F. Thayer, F. Åhs, M. Fredrikson, J.J. Sollers, T.D. Wager, A meta-analysis of heart rate variability and neuroimaging studies: implications for heart rate variability as a marker of stress and health, *Neurosci. Biobehav. Rev.* 36 (2) (Feb. 2012) 747–756, <https://doi.org/10.1016/j.neubiorev.2011.11.009>.
- [17] P.M. Lehrer, E. Vaschillo, B. Vaschillo, Resonant frequency biofeedback training to increase cardiac variability: rationale and manual for training, *Appl. Psychophysiol. Biofeedback* 25 (3) (Sep. 2000) 177–191, <https://doi.org/10.1023/A:1009554825745>.
- [18] E.I. de Bruin, J.E. van der Zwan, S.M. Bögels, A RCT comparing daily mindfulness meditations, biofeedback exercises, and daily physical exercise on attention control, executive functioning, mindful awareness, self-compassion, and worrying in stressed young adults, *Mindfulness* 7 (5) (Oct. 2016) 1182–1192, <https://doi.org/10.1007/s12671-016-0561-5>.
- [19] R.P. Brown, P.L. Gerbarg, F. Muench, Breathing practices for treatment of psychiatric and stress-related medical conditions, *Psychiatr. Clin.* 36 (1) (Mar. 2013) 121–140, <https://doi.org/10.1016/j.psc.2013.01.001>.
- [20] H. ChuDuc, K. NguyenPhan, D. NguyenViet, A review of heart rate variability and its applications, *APCBEE Procedia* 7 (Jan. 2013) 80–85, <https://doi.org/10.1016/j.apcbec.2013.08.016>.

- [21] J. Fatissou, V. Oswald, F. Lalonde, Influence diagram of physiological and environmental factors affecting heart rate variability: an extended literature overview, *Heart Int.* 11 (1) (Jan. 2016), <https://doi.org/10.5301/heartint.5000232> heartint.5000232.
- [22] S. Delliaux, A. Delaforte, J.-C. Deharo, G. Chaumet, Mental workload alters heart rate variability, lowering non-linear dynamics, *Front. Physiol.* 10 (2019). Available: <https://www.frontiersin.org/articles/10.3389/fphys.2019.00565>. (Accessed 30 July 2023).
- [23] J. Hayano, E. Yuda, Pitfalls of assessment of autonomic function by heart rate variability, *J. Physiol. Anthropol.* 38 (1) (Mar. 2019) 3, <https://doi.org/10.1186/s40101-019-0193-2>.
- [24] M. Brennan, M. Palaniswami, P. Kamen, Poincaré plot interpretation using a physiological model of HRV based on a network of oscillators, *Am. J. Physiol. Heart Circ. Physiol.* (Nov. 2002), <https://doi.org/10.1152/ajpheart.00405.2000>. Available: <https://journals.physiology.org/doi/10.1152/ajpheart.00405.2000>. (Accessed 20 September 2023).
- [25] A.B. Ciccone, J.A. Siedlik, J.M. Wecht, J.A. Deckert, N.D. Nguyen, J.P. Weir, Reminder: RMSSD and SD1 are identical heart rate variability metrics, *Muscle Nerve* 56 (4) (2017) 674–678, <https://doi.org/10.1002/mus.25573>.
- [26] M.P. Tulppo, T.H. Makikallio, T.E. Takala, T. Seppanen, H.V. Huikuri, Quantitative beat-to-beat analysis of heart rate dynamics during exercise, *Am. J. Physiol. Heart Circ. Physiol.* 271 (1) (Jul. 1996) H244–H252, <https://doi.org/10.1152/ajpheart.1996.271.1.H244>.
- [27] J.T. Bigger, P. Albrecht, R.C. Steinman, L.M. Rolnitzky, J.L. Fleiss, R.J. Cohen, Comparison of time- and frequency domain-based measures of cardiac parasympathetic activity in Holter recordings after myocardial infarction, *Am. J. Cardiol.* 64 (8) (Sep. 1989) 536–538, [https://doi.org/10.1016/0002-9149\(89\)90436-0](https://doi.org/10.1016/0002-9149(89)90436-0).
- [28] G.E. Billman, H.V. Huikuri, J. Sacha, K. Trimmel, An introduction to heart rate variability: methodological considerations and clinical applications, *Front. Physiol.* 6 (2015), <https://doi.org/10.3389/fphys.2015.00055>. Available: <https://www.frontiersin.org/articles/10.3389/fphys.2015.00055>. (Accessed 24 August 2023).
- [29] J.A. Jo, A. Blasi, E.M. Valladares, R. Juarez, A. Baydur, M.C.K. Khoo, A nonlinear model of cardiac autonomic control in obstructive sleep apnea syndrome, *Ann. Biomed. Eng.* 35 (8) (Aug. 2007) 1425–1443, <https://doi.org/10.1007/s10439-007-9299-5>.
- [30] A. Hernández-Vicente, et al., Heart rate variability and exceptional longevity, *Front. Physiol.* 11 (2020). Available, <https://www.frontiersin.org/articles/10.3389/fphys.2020.566399>. (Accessed 24 September 2023).
- [31] J.A. Chalmers, D.S. Quintana, M.J.-A. Abbott, A.H. Kemp, Anxiety disorders are associated with reduced heart rate variability: a meta-analysis, *Front. Psychiatr.* 5 (2014). Available: <https://www.frontiersin.org/articles/10.3389/fpsy.2014.00080>. (Accessed 24 September 2023).
- [32] G. Billman, The LF/HF ratio does not accurately measure cardiac sympatho-vagal balance, *Front. Physiol.* 4 (2013). Available: <https://www.frontiersin.org/articles/10.3389/fphys.2013.00026>. (Accessed 24 August 2023).
- [33] B. Francesco, et al., Linear and nonlinear heart rate variability indexes in clinical practice, *Comput. Math. Methods Med.* 2012 (Feb. 2012) e219080, <https://doi.org/10.1155/2012/219080>.
- [34] M. Malik, et al., Heart rate variability : standards of measurement, physiological interpretation, and clinical use, *Eur. Heart J.* 17 (3) (Mar. 1996) 354–381, <https://doi.org/10.1093/oxfordjournals.eurheartj.a014868>.
- [35] S. Laborde, E. Mosley, J.F. Thayer, Heart rate variability and cardiac vagal tone in psychophysiological research – recommendations for experiment planning, data analysis, and data reporting, *Front. Psychol.* 8 (2017), <https://doi.org/10.3389/fpsyg.2017.00213>. Available: <https://www.frontiersin.org/articles/10.3389/fpsyg.2017.00213>. (Accessed 28 July 2023).
- [36] N.J.C. Stapelberg, D.L. Neumann, D.H.K. Shum, H. McConnell, I. Hamilton-Craig, The sensitivity of 38 heart rate variability measures to the addition of artifact in human and artificial 24-hr cardiac recordings, *Ann. Noninvasive Electrocardiol.* 23 (1) (Jul. 2017) e12483, <https://doi.org/10.1111/anec.12483>.
- [37] D.S. Quintana, J.A.J. Heathers, Considerations in the assessment of heart rate variability in biobehavioral research, *Front. Psychol.* 5 (2014), <https://doi.org/10.3389/fpsyg.2014.00805>. Available: <https://www.frontiersin.org/articles/10.3389/fpsyg.2014.00805>. (Accessed 15 September 2023).
- [38] PNS and SNS indexes in evaluating autonomic function - Kubios, Available: <https://www.kubios.com/hrv-ans-function/>. (Accessed 29 August 2023).
- [39] F. Shaffer, R. McCraty, C.L. Zerr, A healthy heart is not a metronome: an integrative review of the heart's anatomy and heart rate variability, *Front. Psychol.* 5 (2014), <https://doi.org/10.3389/fpsyg.2014.01040>. Available: <https://www.frontiersin.org/articles/10.3389/fpsyg.2014.01040>. (Accessed 18 August 2023).
- [40] R.E. Kleiger, P.K. Stein, J.T. Bigger Jr., Heart rate variability: measurement and clinical utility, *Ann. Noninvasive Electrocardiol.* 10 (1) (2005) 88–101, <https://doi.org/10.1111/j.1542-474X.2005.10101.x>.
- [41] K. Umetani, D.H. Singer, R. McCraty, M. Atkinson, Twenty-four hour time domain heart rate variability and heart rate: relations to age and gender over nine decades, *J. Am. Coll. Cardiol.* 31 (3) (Mar. 1998) 593–601, [https://doi.org/10.1016/S0735-1097\(97\)00554-8](https://doi.org/10.1016/S0735-1097(97)00554-8).
- [42] H. Otzenberger, et al., Dynamic heart rate variability: a tool for exploring sympathovagal balance continuously during sleep in men, *Am. J. Physiol. Heart Circ. Physiol.* 275 (3) (Sep. 1998) H946–H950, <https://doi.org/10.1152/ajpheart.1998.275.3.H946>.
- [43] H.-K. Yang, et al., Application for the wearable heart activity monitoring system : analysis of the autonomic function of HRV, in: 2008 30th Annual International Conference of the IEEE Engineering in Medicine and Biology Society, Aug. 2008, pp. 1258–1261, <https://doi.org/10.1109/IEMBS.2008.4649392>.
- [44] O. Nasario-Junior, P.R. Benchimol-Barbosa, J. Nadal, Refining the deceleration capacity index in phase-rectified signal averaging to assess physical conditioning level, *J. Electrocardiol.* 47 (3) (May 2014) 306–310, <https://doi.org/10.1016/j.jelectrocard.2013.12.006>.
- [45] A. Bauer, et al., Deceleration capacity of heart rate as a predictor of mortality after myocardial infarction: cohort study, *Lancet* 367 (9523) (May 2006) 1674–1681, [https://doi.org/10.1016/S0140-6736\(06\)68735-7](https://doi.org/10.1016/S0140-6736(06)68735-7).
- [46] J.F. Thayer, S.S. Yamamoto, J.F. Brosschot, The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors, *Int. J. Cardiol.* 141 (2) (May 2010) 122–131, <https://doi.org/10.1016/j.ijcard.2009.09.543>.
- [47] P. Grossman, E.W. Taylor, Toward understanding respiratory sinus arrhythmia: relations to cardiac vagal tone, evolution and biobehavioral functions, *Biol. Psychol.* 74 (2) (Feb. 2007) 263–285, <https://doi.org/10.1016/j.biopsycho.2005.11.014>.
- [48] D.L. Eckberg, Human sinus arrhythmia as an index of vagal cardiac outflow, *J. Appl. Physiol.* 54 (4) (Apr. 1983) 961–966, <https://doi.org/10.1152/jappl.1983.54.4.961>.
- [49] D.L. Eckberg, M.J. Eckberg, Human sinus node responses to repetitive, ramped carotid baroreceptor stimuli, *Am. J. Physiol. Heart Circ. Physiol.* 242 (4) (Apr. 1982) H638–H644, <https://doi.org/10.1152/ajpheart.1982.242.4.H638>.
- [50] C.-H. Hsu, et al., Poincaré plot indexes of heart rate variability detect dynamic autonomic modulation during general anesthesia induction, *Acta Anaesthesiol. Taiwanica* 50 (1) (Mar. 2012) 12–18, <https://doi.org/10.1016/j.aat.2012.03.002>.
- [51] P.W. Kamen, H. Krum, A.M. Tonkin, Poincaré plot of heart rate variability allows quantitative display of parasympathetic nervous activity in humans, *Clin. Sci.* 91 (2) (Aug. 1996) 201–208, <https://doi.org/10.1042/cs0910201>.
- [52] C.-K. Peng, S. Havlin, H.E. Stanley, A.L. Goldberger, Quantification of scaling exponents and crossover phenomena in nonstationary heartbeat time series, *Chaos Interdiscip. J. Nonlinear Sci.* 5 (1) (Mar. 1995) 82–87, <https://doi.org/10.1063/1.166141>.
- [53] M. Weippert, K. Behrens, A. Rieger, M. Kumar, M. Behrens, Effects of breathing patterns and light exercise on linear and nonlinear heart rate variability, *Appl. Physiol. Nutr. Metabol.* 40 (8) (Aug. 2015) 762–768, <https://doi.org/10.1139/apnm-2014-0493>.
- [54] C. Schubert, M. Lambert, R.A. Nelesen, W. Bardwell, J.-B. Choi, J.E. Dimsdale, Effects of stress on heart rate complexity—a comparison between short-term and chronic stress, *Biol. Psychol.* 80 (3) (Mar. 2009) 325–332, <https://doi.org/10.1016/j.biopsycho.2008.11.005>.
- [55] R. Castaldo, P. Melillo, L. Pecchia, Acute mental stress detection via ultra-short term HRV analysis, in: D.A. Jaffray (Ed.), *World Congress on Medical Physics and Biomedical Engineering*, June 7–12, 2015, Toronto, Canada, Springer International Publishing, Cham, 2015, pp. 1068–1071, [https://doi.org/10.1007/978-3-319-19387-8\\_260](https://doi.org/10.1007/978-3-319-19387-8_260). IFMBE Proceedings.
- [56] M.K. Moridani, Z. Mahabadi, N. Javadi, Heart rate variability features for different stress classification, *Bratislava Med. J.* 121 (9) (2020) 619–627, <https://doi.org/10.4149/BLL.2020.107>.
- [57] H. Young, D. Benton, We should be using nonlinear indices when relating heart-rate dynamics to cognition and mood, *Sci. Rep.* 5 (1) (Nov. 2015) 1, <https://doi.org/10.1038/srep16619>.

- [58] R. Baevsky, A. Berseneva, *Methodical Recommendations Use Kardivar System for Determination of the Stress Level and Estimation of the Body Adaptability Standards of Measurements and Physiological Interpretation*, 2008. Moscow.
- [59] G.G. Berntson, et al., Heart rate variability: origins, methods, and interpretive caveats, *Psychophysiology* 34 (6) (1997) 623–648, <https://doi.org/10.1111/j.1469-8986.1997.tb02140.x>.
- [60] S.E. Taylor, Tend and befriend: biobehavioral bases of affiliation under stress, *Curr. Dir. Psychol. Sci.* 15 (6) (Dec. 2006) 273–277, <https://doi.org/10.1111/j.1467-8721.2006.00451.x>.
- [61] M. Brennan, M. Palaniswami, P. Kamen, Do existing measures of Poincare plot geometry reflect nonlinear features of heart rate variability? *IEEE Trans. Biomed. Eng.* 48 (11) (Nov. 2001) 1342–1347, <https://doi.org/10.1109/10.959330>.
- [62] M.A. Woo, W.G. Stevenson, D.K. Moser, R.B. Trelease, R.M. Harper, Patterns of beat-to-beat heart rate variability in advanced heart failure, *Am. Heart J.* 123 (3) (Mar. 1992) 704–710, [https://doi.org/10.1016/0002-8703\(92\)90510-3](https://doi.org/10.1016/0002-8703(92)90510-3).
- [63] J.S. Richman, J.R. Moorman, Physiological time-series analysis using approximate entropy and sample entropy, *Am. J. Physiol. Heart Circ. Physiol.* 278 (6) (Jun. 2000) H2039–H2049, <https://doi.org/10.1152/ajpheart.2000.278.6.H2039>.
- [64] Y. Fusheng, H. Bo, T. Qingyu, Approximate entropy and its application in biosignal analysis, in: M. Akay (Ed.), *Nonlinear Biomedical Signal Processing, Dynamic Analysis and Modeling*, Wiley-IEEE Press, 2009, <https://doi.org/10.1109/9780470545379.ch3>. Available: <https://ieeexplore.ieee.org/document/5263187>. (Accessed 29 August 2023).
- [65] F. Beckers, D. Ramaekers, A.E. Aubert, Approximate entropy of heart rate variability: validation of methods and application in heart failure, *Cardiovasc. Eng. Int. J.* 1 (4) (Dec. 2001) 177–182, <https://doi.org/10.1023/A:1015212328405>.
- [66] N. Lippman, K.M. Stein, B.B. Lerman, Comparison of methods for removal of ectopy in measurement of heart rate variability, *Am. J. Physiol. Heart Circ. Physiol.* 267 (1) (Jul. 1994) H411–H418, <https://doi.org/10.1152/ajpheart.1994.267.1.H411>.
- [67] J.A. Hirsch, B. Bishop, Respiratory sinus arrhythmia in humans: how breathing pattern modulates heart rate, *Am. J. Physiol. Heart Circ. Physiol.* 241 (4) (Oct. 1981) H620–H629, <https://doi.org/10.1152/ajpheart.1981.241.4.H620>.
- [68] P. Pilowsky, Good vibrations? Respiratory rhythms in the central control of blood pressure, *Clin. Exp. Pharmacol. Physiol.* 22 (9) (1995) 594–604, <https://doi.org/10.1111/j.1440-1681.1995.tb02072.x>.
- [69] J. Hayano, F. Yasuma, A. Okada, S. Mukai, T. Fujinami, Respiratory sinus arrhythmia, *Circulation* 94 (4) (Aug. 1996) 842–847, <https://doi.org/10.1161/01.CIR.94.4.842>.
- [70] D. Nunan, G.R.H. Sandercock, D.A. Brodie, A quantitative systematic review of normal values for short-term heart rate variability in healthy adults, *Pacing Clin. Electrophysiol.* 33 (11) (2010) 1407–1417, <https://doi.org/10.1111/j.1540-8159.2010.02841.x>.
- [71] L. Bernardi, et al., Effects of controlled breathing, mental activity and mental stress with or without verbalization on heart rate variability, *J. Am. Coll. Cardiol.* 35 (6) (May 2000) 1462–1469, [https://doi.org/10.1016/S0735-1097\(00\)00595-7](https://doi.org/10.1016/S0735-1097(00)00595-7).
- [72] A.K. Ahmed, J.B. Harness, A.J. Mearns, Respiratory control of heart rate, *Eur. J. Appl. Physiol.* 50 (1) (Feb. 1982) 95–104, <https://doi.org/10.1007/BF00952248>.
- [73] P.M. Lehrer, et al., Heart rate variability biofeedback increases baroreflex gain and peak expiratory flow, *Psychosom. Med.* 65 (5) (Oct. 2003) 796, <https://doi.org/10.1097/01.PSY.0000089200.81962.19>.
- [74] W.A. Tiller, R. McCraty, M. Atkinson, *Cardiac coherence: a new, noninvasive measure of autonomic nervous system order*, *Alternative Ther. Health Med.* 2 (1) (Jan. 1996) 52–65.
- [75] G. Billman, Heart rate variability – a historical perspective, *Front. Physiol.* 2 (2011). Available, <https://www.frontiersin.org/articles/10.3389/fphys.2011.00086>. (Accessed 24 August 2023).
- [76] G.G. Berntson, J.T. Cacioppo, K.S. Quigley, Respiratory sinus arrhythmia: autonomic origins, physiological mechanisms, and psychophysiological implications, *Psychophysiology* 30 (2) (1993) 183–196, <https://doi.org/10.1111/j.1469-8986.1993.tb01731.x>.
- [77] F. Yasuma, J. Hayano, Respiratory sinus arrhythmia: why does the heartbeat synchronize with respiratory rhythm? *Chest* 125 (2) (Feb. 2004) 683–690, <https://doi.org/10.1378/chest.125.2.683>.
- [78] A. Angelone, N.A. Coulter, Respiratory sinus arrhythmia: a frequency dependent phenomenon, *J. Appl. Physiol.* 19 (3) (May 1964) 479–482, <https://doi.org/10.1152/jappl.1964.19.3.479>.
- [79] T.E. Brown, L.A. Beightol, J. Koh, D.L. Eckberg, Important influence of respiration on human R-R interval power spectra is largely ignored, *J. Appl. Physiol.* 75 (5) (Nov. 1993) 2310–2317, <https://doi.org/10.1152/jappl.1993.75.5.2310>.
- [80] A. Ben-Tal, S.S. Shamailov, J.F.R. Paton, Central regulation of heart rate and the appearance of respiratory sinus arrhythmia: new insights from mathematical modeling, *Math. Biosci.* 255 (Sep. 2014) 71–82, <https://doi.org/10.1016/j.mbs.2014.06.015>.
- [81] E.G. Vaschillo, B. Vaschillo, P.M. Lehrer, Characteristics of resonance in heart rate variability stimulated by biofeedback, *Appl. Psychophysiol. Biofeedback* 31 (2) (Jun. 2006) 129–142, <https://doi.org/10.1007/s10484-006-9009-3>.
- [82] L. Bernardi, C. Porta, A. Gabutti, L. Spicuzza, P. Sleight, Modulatory effects of respiration, *Auton. Neurosci.* 90 (1) (Jul. 2001) 47–56, [https://doi.org/10.1016/S1566-0702\(01\)00267-3](https://doi.org/10.1016/S1566-0702(01)00267-3).
- [83] P.Y.W. Sin, D.C. Galletly, Y.C. Tzeng, Influence of breathing frequency on the pattern of respiratory sinus arrhythmia and blood pressure: old questions revisited, *Am. J. Physiol. Heart Circ. Physiol.* 298 (5) (May 2010) H1588–H1599, <https://doi.org/10.1152/ajpheart.00036.2010>.
- [84] S.E. Stromberg, M.E. Russell, C.R. Carlson, Diaphragmatic breathing and its effectiveness for the management of motion sickness, *Aerosp. Med. Hum. Perform.* 86 (5) (May 2015) 452–457, <https://doi.org/10.3357/AMHP.4152.2015>.
- [85] D. Laude, M. Goldman, P. Escourrou, J.-L. Elghozi, Effect of breathing pattern on blood pressure and heart rate oscillations in humans, *Clin. Exp. Pharmacol. Physiol.* 20 (10) (1993) 619–626, <https://doi.org/10.1111/j.1440-1681.1993.tb01643.x>.
- [86] J.A. Taylor, C.W. Myers, J.R. Halliwill, H. Seidel, D.L. Eckberg, Sympathetic restraint of respiratory sinus arrhythmia: implications for vagal-cardiac tone assessment in humans, *Am. J. Physiol. Heart Circ. Physiol.* 280 (6) (Jun. 2001) H2804–H2814, <https://doi.org/10.1152/ajpheart.2001.280.6.H2804>.
- [87] D.L. Eckberg, Y.T. Kifle, V.L. Roberts, Phase relationship between normal human respiration and baroreflex responsiveness, *J. Physiol.* 304 (1) (1980) 489–502, <https://doi.org/10.1113/jphysiol.1980.sp013338>.
- [88] Q. Chang, R. Liu, Z. Shen, Effects of slow breathing rate on blood pressure and heart rate variabilities, *Int. J. Cardiol.* 169 (1) (Oct. 2013) e6–e8, <https://doi.org/10.1016/j.ijcard.2013.08.121>.
- [89] P.Z. Zhang, W.N. Tapp, S.S. Reisman, B.H. Natelson, Respiration response curve analysis of heart rate variability, *IEEE Trans. Biomed. Eng.* 44 (4) (Apr. 1997) 321–325, <https://doi.org/10.1109/10.563302>.
- [90] J. Jalife, V.A. Slenker, J.J. Salata, D.C. Michaels, Dynamic vagal control of pacemaker activity in the mammalian sinoatrial node, *Circ. Res.* 52 (6) (Jun. 1983) 642–656, <https://doi.org/10.1161/01.RES.52.6.642>.
- [91] D.R. Seals, N.O. Suwarno, J.A. Dempsey, Influence of lung volume on sympathetic nerve discharge in normal humans, *Circ. Res.* 67 (1) (Jul. 1990) 130–141, <https://doi.org/10.1161/01.RES.67.1.130>.
- [92] G.K. Pal, S. Velkumary, null Madanmohan, *Effect of short-term practice of breathing exercises on autonomic functions in normal human volunteers*, *Indian J. Med. Res.* 120 (2) (Aug. 2004) 115–121.
- [93] Y.-P. Wang, T.B.J. Kuo, C.-T. Lai, J.-W. Chu, C.C.H. Yang, Effects of respiratory time ratio on heart rate variability and spontaneous baroreflex sensitivity, *J. Appl. Physiol.* 115 (11) (Dec. 2013) 1648–1655, <https://doi.org/10.1152/jappphysiol.00163.2013>.
- [94] WMA declaration of Helsinki – ethical principles for medical research involving human subjects – WMA – the world medical association, Available: <https://www.wma.net/policies-post/wma-declaration-of-helsinki-ethical-principles-for-medical-research-involving-human-subjects/>. (Accessed 1 July 2023).
- [95] Meta quest VR headsets, accessories & equipment | meta quest | meta store, Available: <https://www.meta.com/quest/>. (Accessed 11 March 2023).
- [96] M.P. Tarvainen, J.-P. Niskanen, J.A. Lipponen, P.O. Ranta-aho, P.A. Karjalainen, Kubios HRV – heart rate variability analysis software, *Comput. Methods Progr. Biomed.* 113 (1) (Jan. 2014) 210–220, <https://doi.org/10.1016/j.cmpb.2013.07.024>.
- [97] M. Haahr, RANDOM.ORG: true random number service, Available: <https://www.random.org>, 1998. (Accessed 1 June 2018).



- [98] S.T. Mueller, Psychology experiment building language download, Available: <https://sourceforge.net/projects/pebl/>. (Accessed 10 March 2023).
- [99] S.T. Mueller, B.J. Piper, The psychology experiment building language (PEBL) and PEBL test battery, *J. Neurosci. Methods* 222 (Jan. 2014) 250–259, <https://doi.org/10.1016/j.jneumeth.2013.10.024>.
- [100] J.A. Lipponen, M.P. Tarvainen, Accuracy of Kubios HRV Software Respiratory Rate Estimation Algorithms, Kubios Oy, Finland, 2021. Available: [https://www.kubios.com/downloads/RESP\\_white\\_paper.pdf](https://www.kubios.com/downloads/RESP_white_paper.pdf).
- [101] M.J. G. S. Carrasco, R. González, O. Yáñez, Correlation among Poincaré plot indexes and time and frequency domain measures of heart rate variability, *J. Med. Eng. Technol.* 25 (6) (Jan. 2001) 240–248, <https://doi.org/10.1080/03091900110086651>.
- [102] T. Penzel, J.W. Kantelhardt, L. Grote, J.H. Peter, A. Bunde, Comparison of detrended fluctuation analysis and spectral analysis for heart rate variability in sleep and sleep apnea, *IEEE Trans. Biomed. Eng.* 50 (10) (Oct. 2003) 1143–1151, <https://doi.org/10.1109/TBME.2003.817636>.
- [103] B. Henry, N. Lovell, F. Camacho, Nonlinear dynamics time series analysis, in: M. Akay (Ed.), *Nonlinear Biomedical Signal Processing, Dynamic Analysis and Modeling*, Wiley-IEEE Press, 2009, <https://doi.org/10.1109/9780470545379.ch1>. Available: <https://ieeexplore.ieee.org/document/5263181>. (Accessed 29 August 2023).
- [104] J.P. Zbilut, N. Thomasson, C.L. Webber, Recurrence quantification analysis as a tool for nonlinear exploration of nonstationary cardiac signals, *Med. Eng. Phys.* 24 (1) (Jan. 2002) 53–60, [https://doi.org/10.1016/S1350-4533\(01\)00112-6](https://doi.org/10.1016/S1350-4533(01)00112-6).
- [105] C.L. Webber, J.P. Zbilut, Dynamical assessment of physiological systems and states using recurrence plot strategies, *J. Appl. Physiol.* 76 (2) (Feb. 1994) 965–973, <https://doi.org/10.1152/jappl.1994.76.2.965>.
- [106] JASP Team, “JASP.” the JASP Statistics Project, Dec. 22, 2021. Available: <https://jasp-stats.org/>. (Accessed 24 December 2021).
- [107] RStudio Team, RStudio: integrated development environment for R. RStudio, PBC, Boston, MA, Dec. 24, 2021. Available: <http://www.rstudio.com/>. (Accessed 25 December 2021).
- [108] M. Peltola, Role of editing of R-R intervals in the analysis of heart rate variability, *Front. Physiol.* 3 (2012), <https://doi.org/10.3389/fphys.2012.00148>. Available: <https://www.frontiersin.org/articles/10.3389/fphys.2012.00148>. (Accessed 23 August 2023).
- [109] R. Reiner, Integrating a portable biofeedback device into clinical practice for patients with anxiety disorders: results of a pilot study, *Appl. Psychophysiol. Biofeedback* 33 (1) (Mar. 2008) 55–61, <https://doi.org/10.1007/s10484-007-9046-6>.
- [110] L. Mulaffer, M.A. Zafar, B. Ahmed, Analyzing player engagement for biofeedback games, in: 2019 IEEE 7th International Conference on Serious Games and Applications for Health (SeGAH), Aug. 2019, pp. 1–5, <https://doi.org/10.1109/SeGAH.2019.8882481>.
- [111] A. Parmandi, R. Gutierrez-Osuna, Partial reinforcement in game biofeedback for relaxation training, *IEEE Trans. Affect. Comput.* 12 (1) (Jan. 2021) 141–153, <https://doi.org/10.1109/TAFFC.2018.2842727>.
- [112] A. Parmandi, R. Gutierrez-Osuna, Physiological modalities for relaxation skill transfer in biofeedback games, *IEEE J. Biomed. Health Inform.* 21 (2) (Mar. 2017) 361–371, <https://doi.org/10.1109/JBHI.2015.2511665>.
- [113] Z. Wang, A. Parmandi, R. Gutierrez-Osuna, BioPad: leveraging off-the-shelf video games for stress self-regulation, *IEEE J. Biomed. Health Inform.* 22 (1) (Jan. 2018) 47–55, <https://doi.org/10.1109/JBHI.2017.2671788>.
- [114] L.K. Hill, A. Siebenbrock, Are all measures created equal? Heart rate variability and respiration - biomed 2009, *Biomed. Sci. Instrum.* 45 (2009) 71–76.
- [115] A. Jovic, N. Bogunovic, Electrocardiogram analysis using a combination of statistical, geometric, and nonlinear heart rate variability features, *Artif. Intell. Med.* 51 (3) (Mar. 2011) 175–186, <https://doi.org/10.1016/j.artmed.2010.09.005>.
- [116] P. Guzik, et al., Correlations between the Poincaré plot and conventional heart rate variability parameters assessed during paced breathing, *J. Physiol. Sci.* 57 (1) (2007) 63–71, <https://doi.org/10.2170/physiolsci.RP005506>.
- [117] M. Umair, N. Chalabianloo, C. Sas, C. Ersoy, HRV and stress: a mixed-methods approach for comparison of wearable heart rate sensors for biofeedback, *IEEE Access* 9 (2021) 14005–14024, <https://doi.org/10.1109/ACCESS.2021.3052131>.
- [118] B. Vescio, M. Salsone, A. Gambardella, A. Quattrone, Comparison between electrocardiographic and earlobe pulse photoplethysmographic detection for evaluating heart rate variability in healthy subjects in short- and long-term recordings, *Sensors* 18 (3) (Mar. 2018) 3, <https://doi.org/10.3390/s18030844>.
- [119] G. Lu, F. Yang, Limitations of oximetry to measure heart rate variability measures, *Cardiovasc. Eng.* 9 (3) (Sep. 2009) 119–125, <https://doi.org/10.1007/s10558-009-9082-3>.
- [120] W. Jiang, et al., A wearable tele-health system towards monitoring COVID-19 and chronic diseases, *IEEE Rev. Biomed. Eng.* 15 (2022) 61–84, <https://doi.org/10.1109/RBME.2021.3069815>.
- [121] R. Mukherjee, S.K. Ghorai, B. Gupta, T. Chakravarty, Development of a wearable remote cardiac health monitoring with alerting system, *Instrum. Exp. Tech.* 63 (2) (Apr. 2020) 273–283, <https://doi.org/10.1134/S002044122002013X>.
- [122] N. Aimie-Salleh, N.A.A. Ghani, N. Hasanudin, S.N.S. Shafie, Heart rate variability recording system using photoplethysmography sensor, *Auton. Nerv. Syst. Monit. - Heart Rate Var.* (Nov. 2019), <https://doi.org/10.5772/intechopen.89901>. Available: <https://www.intechopen.com/books/autonomic-nervous-system-monitoring-heart-rate-variability/heart-rate-variability-recording-system-using-photoplethysmography-sensor>. (Accessed 9 October 2020).
- [123] G. Lu, F. Yang, J.A. Taylor, J.F. Stein, A comparison of photoplethysmography and ECG recording to analyse heart rate variability in healthy subjects, *J. Med. Eng. Technol.* 33 (8) (Nov. 2009) 634–641, <https://doi.org/10.3109/03091900903150998>.
- [124] E. Gil, M. Orini, R. Bailón, J.M. Vergara, L. Mainardi, P. Laguna, Photoplethysmography pulse rate variability as a surrogate measurement of heart rate variability during non-stationary conditions, *Physiol. Meas.* 31 (9) (Aug. 2010) 1271, <https://doi.org/10.1088/0967-3334/31/9/015>.