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# Effect of one-shot cognitive behavioral therapy on insomnia and heart rate variability of health care workers at the time of COVID-19 pandemic: a randomized controlled trial

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## Abstract

**Objectives** The emergence of sleep disturbances in response to major stressful events has been previously documented. Heart rate variability (HRV) is an objective marker that provides insight into autonomic nervous system dynamics. The aim of the present study was to examine the preliminary effectiveness of a one-shot session of cognitive behavioral therapy for insomnia (CBT-I) for frontline healthcare providers with acute insomnia.

**Methods** This study was conducted from 2020 to 2021 on healthcare workers with insomnia. The healthcare workers were randomly allocated to receive either one-shot cognitive behavioral therapy or routine care. Insomnia severity index (ISI) and heart rate variability were assessed before and 1 month after the interventions.

**Results** Among 57 patients ($n=31$ in the intervention group and $n=26$ in the control group), mean ($\pm$ SD) age of both groups were 34.6 ($\pm$9.5) and 36.6 ($\pm$6.9), respectively. Most participants in both groups were female (81% and 65% in the intervention and control groups, respectively; $p$-value = 0.10). Insomnia severity index score decreased in the intervention group from 13.3 to 6.7 ($p<0.001$). The change before and after the intervention was significant between the two groups for HF-normalized unit (high-frequency power band [0.15–0.40 Hz] in the normalized unit) and LF/HF (the ratio of low frequency to high frequency). HF-normalized unit increased in the intervention group (35.8 $\pm$ 21.5 vs. 45.6 $\pm$ 19.8 before and after the intervention, respectively), and decreased in the control group (43.9 $\pm$ 16.5 vs. 39.8 $\pm$ 18.5, before and after the intervention, respectively).

**Conclusion** The findings suggest that a single-shot session of cognitive behavioral therapy for insomnia is effective in managing acute insomnia symptoms in healthcare workers.

**Keywords** COVID-19 · Insomnia · Behavioral therapy

## Introduction

Epidemics, health disasters, and other incidents have been critical sources of stress and anxiety for the people who face them. Many studies have shown a rise in the prevalence of

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anxiety disorders, including sleep disorders, amid such disasters [1, 2]. The COVID-19 pandemic is a recent example of a stressful disaster. COVID-19 has caused anxiety disorders, depression, and insomnia among the general population and, in particular, healthcare workers (HCW) [3]. It has been indicated that moderately severe insomnia, anxiety, depression, and poor sleep quality are very common among healthcare workers who had experienced COVID-19-like symptoms [4]. Hyperarousal is a physiologic reaction and is the main feature of insomnia. Insomnia is associated with autonomous system pathologies and causes multiple findings in patients such as increased body temperature, increased metabolic rate, and decreased heart rate variability [5]. Heart rate variability shows a quantitative difference between two consecutive heartbeats and is highly affected by the parasympathetic system. A high heart rate variability indicates the healthy activity of the cardiovascular autonomous nervous system and a low high heart rate variability indicates possible associated diseases, including cardiovascular diseases [6]. Insomnia, especially if left untreated, could cause an increased risk of cardiovascular disorders and their risk factors [7]. Studies show autonomous system changes in insomnia are mainly a result of decreased heart rate variability and high sympathetic nervous system activity [8].

Based on some studies, patients with insomnia may benefit from hyperarousal treatment [9]. One of the non-pharmacological treatment methods for insomnia is one-shot cognitive behavioral therapy. Cognitive behavioral therapy sessions are a distinctive non-pharmacological therapeutic method [10, 11] that may eliminate the factors that cause long-term persistence of insomnia such as sleep-incompatible thoughts, beliefs, and behaviors [12]. The customary cognitive behavioral therapy sessions are often time-consuming and tedious [13, 14]. Therefore, efforts have been made to shorten and simplify this method [15–18].

Based on previous studies, cognitive behavioral therapy may be effective and associated with beneficial results in patients with insomnia who might suffer from heart rate variability. However, the existing data seem insufficient for providing a clear treatment strategy. Considering the prevalence and importance of the COVID-19 pandemic and its enormous psychological stress on healthcare workers, the aim of this study was to examine the effectiveness of one-shot cognitive behavioral therapy in the treatment of insomnia amongst healthcare workers in Iran during the COVID-19 pandemic.

## Methods and materials

This is a randomized controlled trial that was performed in 2021 in the Alzahra and Khorshid hospitals affiliated with the Isfahan University of Medical Sciences. The current study was conducted on healthcare workers who suffer from acute insomnia. The study protocol was approved by the Research Committee of the Isfahan University of Medical Sciences and the Ethics committee has confirmed it (Ethics code: IR.MUI.MED.REC.1399.1153, Iranian Registry for Clinical Trials (IRCT) code: IRCT20171219037964N4). The declaration of Helsinki was followed at every step. We report the results following the Consolidated Standards for Reporting Trials (CONSORT).

The inclusion criteria were age over 18 years, healthcare workers in Alzahra and Khorshid hospitals, having an insomnia severity index score of 8 or higher, and signing the written informed consent to participate in this study. The exclusion criteria were having any previous history of heart disease, seizure, bipolar disorders, narcolepsy, and other sleep disturbance disorders, or a desire to start physical activity in the following months, or undergoing treatment with antiarrhythmic or beta-adrenergic blockade medications.

To calculate the sample size, we considered a significance level of 0.05 and a power of 80%. Using the formula for calculating sample size in clinical trials, $n=\frac{\left(z_{1-\frac{\alpha}{2}}+z_{\beta}\right)^{2}\left(S_{1}^{2}+S_{2}^{2}\right)}{\left(\mu_{1}-\mu_{2}\right)^{2}}$, an ISI score of $9.6 \pm 5.0$ for the intervention group and $12.6 \pm 3.5$ for the control group [12], the sample size was calculated as 31 individuals per group.

An advertisement was published in Alzahra and Khorshid hospitals' online channels, inviting healthcare workers who suffer from acute insomnia to participate in this study. After describing the study for those who enrolled, participants were recruited based on the mentioned criteria.

Participants were randomly allocated into two groups of intervention and control using a simple randomization method (ratio, 1:1). A random sequence was produced using "randomization.com" and made available to the research assistant. Each person was given a unique code. The research assistant placed each person in the relevant group by matching the person's code with the random sequence. The person in charge of follow-up and the person in charge of data analysis were unaware of the study groups.

Data on demographic variables including age, sex (male/female), job (nurse/physician, medical student, other healthcare workers roles), education (diploma and education level), having any comorbidities (yes/no), and using any medication (yes/no) were gathered at the beginning of the study.

Participants in the intervention group were asked to complete a sleep diary sheet documenting their sleep patterns in the previous 2 weeks. Then, they were divided into groups of 2–3 to receive the one-shot cognitive behavioral therapy for insomnia session. In the cognitive behavioral therapy for insomnia session, the therapist evaluated the diaries, took a brief history, then explained the stimulus control and some cognitions about insomnia and maladaptive behaviors that

may convert acute insomnia into chronic insomnia. The sleep restriction strategy is applied and some pamphlets that included information about insomnia and its treatment were given to them. No interventions were performed for the participants in the control group. Primary outcomes, including insomnia state and heart rate variability, were evaluated before and 1 month after the intervention.

## Insomnia state

The Iranian version of the insomnia severity index was used to measure insomnia [19]. The insomnia severity index is a self-assessment scale designed by Morin et al. [20]. This scale consists of seven questions on a Likert scale (0: not at all, 5: all the time). The total score varies from zero to 28 and is divided into four categories; 0–7: no insomnia, 8–14: mild insomnia, 15–21: moderate insomnia, and 22–28: severe insomnia.

## Heart rate variability

After 30 min of sitting in a relaxed situation, continuous raw ECG data were derived from a modified Lead-II configuration by using ECG data from the PSG device. Since we needed information in the form of European Data Format (EDF), we used the PSG device for recording ECG (Fig. 1). During the recordings, the participants were lying down in a silent room. We recorded for 5 min and then the data were exported in EDF and analyzed with MATLAB software capable of analyzing heart rate variability (Fig. 2) [21].

As recommended by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology [20], 5-min heart rate variability epochs were analyzed to obtain power spectral estimates including low-frequency (LF) heart rate variability (0.04–0.15 Hz) in ms2 and normalized unit, high-frequency (HF) power band (0.15–0.40 Hz) in ms2 and NU, and the ratio of low frequency to high frequency (LF/HF ratio).

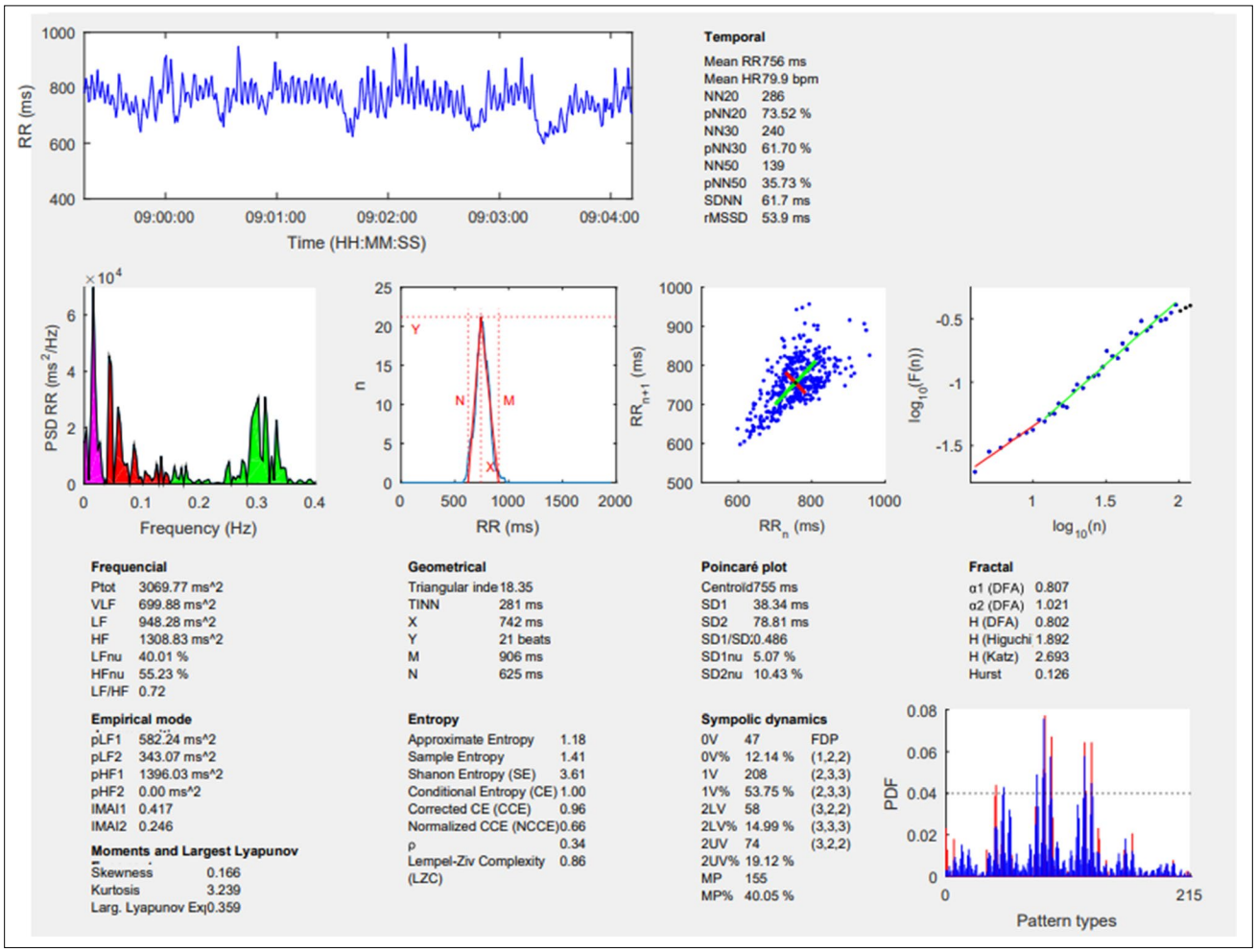


**Fig. 1** An example of an electrocardiogram which is analyzed for heart rate variability

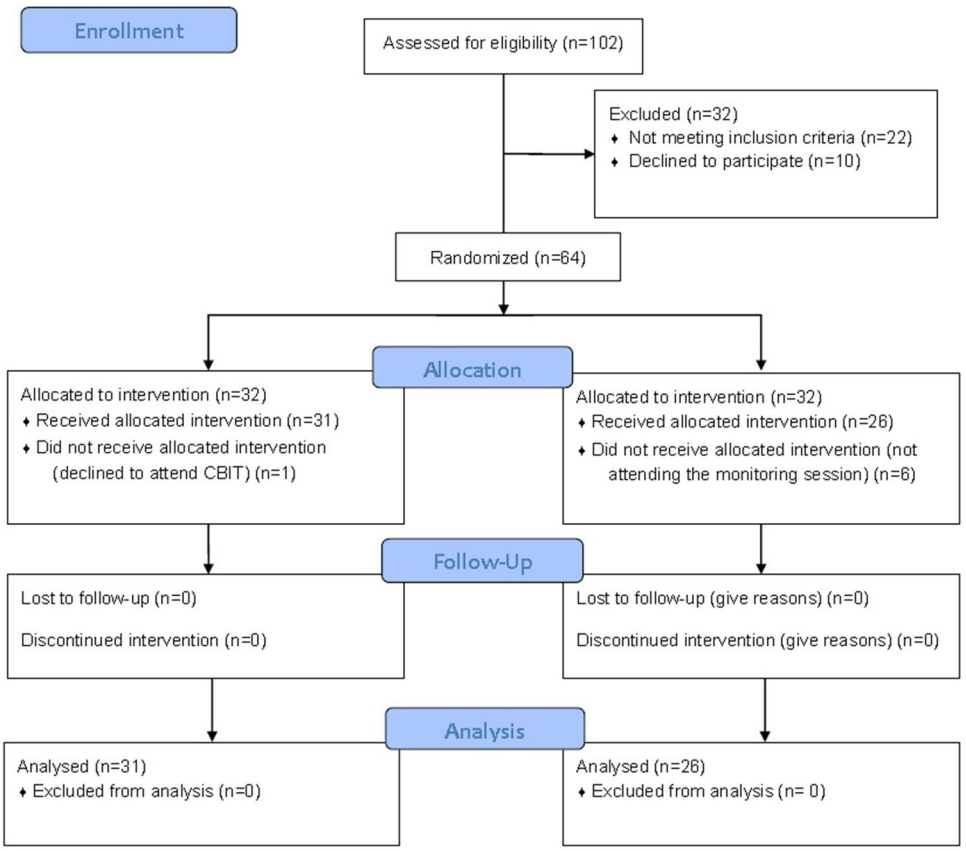


**Fig. 2** The CONSORT flow diagram of the study

## Data analysis

Data were analyzed using SPSS version 22 (SPSS Inc., Chicago, IL, USA). Mean and standard deviation (SD) were used to describe continuous variables. Frequency was used to describe categorical variables. The chi-square test was used to compare qualitative variables between two groups. The Mann–Whitney test was used to compare quantitative variables between two groups before and after the intervention. The level of significance was set at 0.05.

## Results

From the 102 healthcare workers who voluntarily enrolled in this study, 64 participants with the inclusion and exclusion criteria were randomly assigned into two groups. One participant in the intervention group declined to attend the cognitive behavioral therapy for insomnia session. Six participants in the control group refused to participate in the monitoring session. The data from the 31 participants in the intervention group and from the 26 participants in the control group were analyzed (Fig. 2).

The mean (± SD) age of participants in the intervention and control groups were 34.6 (± 9.5) and 36.6 (± 6.9), respectively. Most of the participants in both groups were female (81% and 65% in the intervention and control groups, respectively; *p*-value: 0.1). The distribution of job positions was not statistically different between the two groups, and most of the participants in both groups were nurses (55% in the intervention group, 54% in the control group; *p*-value: 0.8). Considering the education level, most of the participants had higher education, and the education level was the same between the two groups (*p*-value: 0.3). Other demographic variables are presented in Table 1.

### Insomnia

The mean insomnia severity index score before the intervention was not statistically different between groups (13.3 ± 3.7 vs. 13.3 ± 4.4 in the intervention and control groups, respectively; *p*-value: 0.9). The mean score of all seven questions in the insomnia severity index were also the same between the two groups before the intervention ($p$-value > 0.05). The mean insomnia severity index score after the intervention was lower in the intervention group in comparison to the control group (6.7 ± 4.5 vs. 13.6 ± 6.3, respectively; $p$-value < 0.001(; the difference in insomnia severity index score before and after the intervention was also statistically significant comparing the two groups ($p$-value < 0.001). The results for all seven questions are presented in Table 2.

**Table 1** Baseline demographics of intervention and control group

| | Intervention ($N=31$) | Control ($N=26$) | $p$-value |
|---|---|---|---|
| Age | 34.6 ± 9.5 | 36.6 ± 6.9 | 0.2* |
| Sex | | | 0.1** |
| Male | 6 (19.4) | 9 (34.6) | |
| Female | 25 (81) | 17 (65) | |
| Job | | | 0.8** |
| Nurse | 17 (55) | 14 (54) | |
| Medical student and physician | 8 (26) | 8 (31) | |
| Other healthcare workers | 6 (19) | 4 (15) | |
| Education | | | 0.3** |
| Diploma and less | 11 (36) | 6 (23) | |
| Higher education | 20 (65) | 20 (77) | |
| Any past medical history | | | 0.2** |
| Yes | 9 (29) | 4 (15) | |
| No | 22 (71) | 22 (85) | |
| Any medication | | | 0.3** |
| Yes | 13 (42) | 8 (31) | |
| No | 18(58) | 18(69) | |

All data are presented in number (percent), except for age which is presented in mean ± SD

*Mann–Whitney test, **Chi-square

**Table 2** Insomnia severity index in the intervention and control group before and after the intervention

| | Intervention ($n=31$) | Control ($n=26$) | $p$-value comparing before and after the intervention | $p$-value comparing the mean difference between two groups |
|---|---|---|---|---|
| ISI score | | | | 0.000 |
| Before | 13.3 (3.7) | 13.3 (4.4) | 0.9 | |
| After | 6.7 (4.5) | 13.6 (6.3) | 0.000 | |
| Difficulty falling asleep | | | | 0.000 |
| Before | 2.1 (0.9) | 2.0 (0.9) | 0.8 | |
| After | 1.0 (0.8) | 2.0 (1.1) | 0.001 | |
| Difficulty staying asleep | | | | 0.001 |
| Before | 1.6 (0.9) | 1.3 (1.1) | 0.3 | |
| After | 0.8 (1.0) | 1.3 (1.1) | 0.05 | |
| Problem waking up too early | | | | 0.017 |
| Before | 0.9 (0.8) | 1.1 (1.1) | 0.3 | |
| After | 0.4 (0.6) | 1.3 (1.0) | 0.000 | |
| Satisfaction with the current sleep pattern | | | | 0.000 |
| Before | 2.7 (0.8) | 2.6 (1.0) | 0.7 | |
| After | 1.3 (0.7) | 2.3 (1.1) | 0.000 | |
| Interfering with daily function | | | | 0.000 |
| Before | 2.4 (0.9) | 2.2 (1.0) | 0.6 | |
| After | 1.4 (1.2) | 2.6 (1.2) | 0.001 | |
| Noticeable for others in terms of impairing the quality of life | | | | 0.025 |
| Before | 1.4 (1.1) | 1.9 (1.1) | 0.07 | |
| After | 0.7 (0.8) | 2.0 (1.3) | 0.000 | |
| Worried/distressed about a current sleep problem | | | | 0.000 |
| Before | 2.0 (1.0) | 1.8 (1.0) | 0.4 | |
| After | 0.9 (1.0) | 2.0 (1.4) | 0.002 | |

Data are presented in mean ± SD. $p$-values are estimated using a Mann–Whitney test. *ISI*, insomnia severity index

### Heart rate variability

Considering frequential indexes, all indexes were the same between the two groups at baseline ($p$-value > 0.05). The mean (± SD) of LF after the intervention in the intervention group was 613.9 (± 433.2) and 364.2 (± 344.6) in the control group, which was statistically significant ($p$-value: 0.01). Also, the mean of HF after the study in the intervention group was higher than the control group (1166.4 ± 1505.7 vs. 294.4 ± 257.6, respectively; $p$-value: 0.01).

The change of frequential indexes before and after the intervention was not statistically significant between the two groups for all indexes except for HFnu and LF/HF. HFnu increases in the intervention group (35.8 ± 21.5 vs. 45.6 ± 19.8 before and after the intervention, respectively), and decreases in the control group (43.9 ± 16.5 vs. 39.8 ± 18.5, before and after the intervention, respectively). The mean of the difference before and after the intervention was statistically significant between groups ($p$-value: 0.01). The mean of LF/HF decreases in the intervention group (2.3 ± 2.2 vs. 1.1 ± 0.8 before and after the intervention, respectively) and increases in the control group (1.1 ± 0.9 vs. 1.2 ± 0.8 before and after the intervention, respectively). This change was statistically significant ($p$-value: 0.001). Table 3 demonstrates heart rate variability indexes for the intervention and control groups, before and after the intervention.

## Discussion

The primary aim of this study was to determine the effectiveness of a "single-shot" cognitive behavioral therapy for insomnia, with an accompanying information pamphlet, in the treatment of acute insomnia of healthcare workers. The results of our study suggest that this variant of cognitive behavioral therapy for insomnia is suitable for most healthcare workers with acute insomnia and can improve all insomnia symptoms based on insomnia severity index score.

These findings could be of high importance and are in line with the previous literature on the impact of a single-shot cognitive behavioral therapy for insomnia intervention for acute insomnia [11, 12]. The identification and management of insomnia is fundamental for healthcare workers. An important point is that previous studies have also reported higher rates of insomnia (34%–36%), anxiety (45%), and depressive symptoms (50%) among individuals at high risk of COVID-19 infection compared to the general population [3, 22]. Additionally, many studies have established that insomnia itself is a risk factor for developing depression, anxiety, and suicide [23]. The findings from the present study suggest that a single shot of cognitive behavioral therapy for insomnia, with an accompanying self-help pamphlet, has promise in this setting for individuals with acute insomnia.

**Table 3** Comparison of outcome variables before and after the intervention and between two groups

| | | Intervention ($n$ = 31) | Control ($n$ = 26) | $p$-value comparing before and after the intervention | $p$-value comparing the mean difference between two groups |
|---|---|---|---|---|---|
| Frequencial | Vlf | | | | 0.30 |
| | Before | 743.3 (639.5) | 551.9 (401.3) | 0.50 | |
| | After | 898.2 (630.9) | 667.9 (701.7) | 0.07 | |
| | LF | | | | 0.80 |
| | Before | 663.6 (559.5) | 408.8 (436.1) | 0.07 | |
| | After | 613.9 (433.2) | 364.2 (344.6) | 0.01 | |
| | HF | | | | 0.07 |
| | Before | 760.1 (1053.9) | 733.1 (1146.5) | 0.70 | |
| | After | 1166.4 (1505.7) | 294.4 (257.6) | 0.01 | |
| | LF-normalized unit | | | | 0.60 |
| | Before | 47.6 (19.01) | 39.6 (11.7) | 0.05 | |
| | After | 40.2 (17.5) | 41.4 (14.6) | 0.90 | |
| | HF-normalized unit | | | | 0.01 |
| | Before | 35.8 (21.5) | 43.9 (16.5) | 0.07 | |
| | After | 45.6 (19.8) | 39.8 (18.5) | 0.40 | |
| | LF/HF | | | | 0.001 |
| | Before | 2.3 (2.2) | 1.1 (0.9) | 0.05 | |
| | After | 1.1 (0.8) | 1.2 (0.8) | 0.70 | |

Data are presented in mean ± SD. $p$-values are estimated using a Mann–Whitney test. *HF*, high frequency; *HF-normalized unit*, high frequency–normalized unit; *LF*, low frequency; *LF-normalized unit*, low frequency–normalized unit; *Vlf*, very low frequency

In the spectral analysis of heart rate variability, the LF band region was previously called the baroreceptor range because it mainly reflects baroreceptor activity during resting conditions [24]. LF power may be produced by both the parasympathic nervous system (PNS), sympathic nervous system (SNS), and blood pressure (BP) regulation via baroreceptors [24–26]. In resting conditions, the LF band reflects baroreflex activity and not cardiac sympathetic innervation [12]. By evaluating the neurophysiology, it should be noted that efferent vagal activity contributes significantly to the HF component, as seen in clinical and experimental observations of autonomic maneuvers such as electrical vagal stimulation, muscarinic receptor blockade, and vagotomy [25, 27, 28].

Regarding LF/HF ratio, there are some studies [29] that challenge the belief that the LF/HF ratio measures "sympatho-vagal balance" [30, 31]. Nonbelievers say that LF power is not a pure index of SNS drive. Half of the variability in this frequency band is due to the PNS, and unknown factors produce a smaller proportion. PNS and SNS interactions are complex, non-linear, and frequently non-reciprocal. Additionally, confounding by respiratory mechanics and resting heart rate creates uncertainty regarding PNS and SNS contributions to the LF/HF ratio during the measurement period [32].

Shaffer et al. [33] warned that the LF/HF ratio is controversial because of different processes, for example, the SNS contribution to LF power varies profoundly with testing conditions.

In our evaluation, this ratio decreased after the intervention compared to the control group. By frequency domain analysis of heart rate variability, a comparison of the two groups after 1 month shows a significant decrease in LF value in the control group. If we assume the LF is a sympathetic activity, then the results are in favor of disability of intervention for SNS suppression. This would be against our hypothesis but consistent with recent studies [33]. This may be due to our method of heart rate variability recording (people were lying down); or since the LF-normalized unit values had not changed, it is not important.

Another finding of this study is that we observed significantly increased PNS activity (according to HF-normalized unit and HF value), compared with baseline and control groups, after cognitive behavioral therapy for insomnia in the intervention group. There was evidence that cognitive behavioral therapy positively affects changes in inflammation in patients with insomnia [34] and significant heart rate variability changes in the responders group suggesting that successful insomnia treatment reduces sympathetic hyperactivity and stimulates parasympathetic activity. These are consistent with our results.

Previous evaluation of cognitive behavioral therapy for insomnia's effect on heart rate variability done by Jarrin et al. [32] shows no significant differences in mean heart rate variability changes between responders and no responders or between remitters and non-remitters in either S2 or REM. However, there were some fundamental differences between the methodologies of that study and this study. Such differences may be justified in the way that our samples were taken from acute sleepless healthcare workers with probably high levels of anxiety, especially during the COVID-19 pandemic, instead of patients with chronic insomnia. Furthermore, we assessed the ECGs of our samples while awake in a lying position, not during sleep. We should note that one-shot cognitive behavioral therapy for insomnia was done for the intervention group instead of full cognitive behavioral therapy for insomnia, which is more time-consuming. Therefore, it is believed that long-term treatment in the other study may be responsible for different results.

## Conclusion

We find that one-shot cognitive behavioral therapy for insomnia is an effective treatment for acute insomnia of COVID-19 in frontline healthcare workers, can cause an increment of parasympathetic activity, and changes the autonomous system balance towards lower SNS and higher PNS activity 1 month after therapy. Accordingly, future research is needed to examine whether or not treatment is associated with improvements in other cardiovascular biochemistry and physiology (e.g., lipids, endothelial function) because these data would support the notion that cognitive behavioral therapy for insomnia reduces the risk of future cardiovascular disease or its precursors. Furthermore, it would be valuable to include longitudinal, randomized clinical trials to establish whether or not treatment produces a cardiovascular safeguard.

**Funding** The current research was granted by the Isfahan University of Medical Sciences.

**Data availability** The data are available in a ZIP file and could be uploaded when requested.

## Declarations

**Ethics approval** All procedures performed in the study were in accordance with the ethical standards of the Isfahan University of Medical Science Research Committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards (ethics code: IR.MUI.MED.REC.1399.1153, Iranian Registry for Clinical Trials (IRCT) code: IRCT20171219037964N4).

**Studies involving humans and/or animals** Not applicable.

**Consent to participate** Freely given, informed consent to participate in the study was obtained from participants in this study. The informed consents were confirmed by the Ethics Committee of Isfahan University of Medical Sciences.

**Consent for publication** Not applicable.

**Conflict of interest** The authors declare no competing interests.

## References


1. Askenasy J, Lewin I (1996) The impact of missile warfare on self-reported sleep quality. Part 1. Sleep 19:47–51
2. Kalayjian A, Kanazi RL, Aberson CL, Feygin L (2002) A cross-cultural study of the psychosocial and spiritual impact of natural disaster. Int J Group Tensions 31:175–186
3. Zhang C, Yang L, Liu S, Ma S, Wang Y, Cai Z, Du H, Li R, Kang L, Su M (2020) Survey of insomnia and related social psychological factors among medical staff involved in the 2019 novel coronavirus disease outbreak. Front Psych 11:306
4. Amra B, Salmasi M, Soltaninejad F, Sami R, Nickpour M, Mansourian M, Ghasemi K and Morin CM (2021) Healthcare workers' sleep and mood disturbances during COVID-19 outbreak in an Iranian referral center. Sleep Breath 25(4):2197–2204
5. Bonnet MH, Arand DL (2010) Hyperarousal and insomnia: state of the science. Sleep Med Rev 14:9–15
6. Task Force of the European Society of Cardiology the North A Electrophysiology (1996) Heart rate variability: standards of measurement, physiological interpretation, and clinical use. Circulation 93:1043–1065
7. Laugsand LE, Vatten LJ, Platou C, Janszky I (2011) Insomnia and the risk of acute myocardial infarction: a population study. Circulation 124:2073–2081
8. Farina B, Dittoni S, Colicchio S, Testani E, Losurdo A, Gnoni V, Di Blasi C, Brunetti R, Contardi A, Mazza S (2014) Heart rate and heart rate variability modification in chronic insomnia patients. Behav Sleep Med 12:290–306
9. Bonnet M, Arand D (1997) Hyperarousal and insomnia. Sleep Med Rev 1:97–108
10. Ellis JG (2019) Cognitive behavioral therapy for insomnia and acute insomnia: considerations and controversies. Sleep Med Clin 14:267–274
11. Randall C, Nowakowski S and Ellis JG (2019) Managing acute insomnia in prison: evaluation of a "one-shot" cognitive behavioral therapy for insomnia (CBT-I) intervention. Behav Sleep Med 17(6):827–836
12. Ellis JG, Cushing T, Germain A (2015) Treating acute insomnia: a randomized controlled trial of a "single-shot" of cognitive behavioral therapy for insomnia. Sleep 38:971–978
13 Gotts Z, Deary V, Newton JL, Ellis J (2016) Treatment of insomnia reduces fatigue in chronic fatigue syndrome in those able to comply with the intervention. Fatigue: Biomed Health Behav 4:208–216
14. Lamberg L (2008) Despite effectiveness, behavioral therapy for chronic insomnia still underused. JAMA 300:2474–2475
15. Edinger JD, Sampson WS (2003) A primary care "friendly" cognitive behavioral insomnia therapy. Sleep 26:177–182
16. Germain A, Moul DE, Franzen PL, Miewald JM, Reynolds CF, Monk TH, Buysse DJ (2006) Effects of a brief behavioral treatment for late-life insomnia: preliminary findings. J Clin Sleep Med 2:407–408
17. Lovato N, Lack L, Wright H, Kennaway DJ (2014) Evaluation of a brief treatment program of cognitive behavior therapy for insomnia in older adults. Sleep 37:117–126
18. Troxel WM, Conrad TS, Germain A, Buysse DJ (2013) Predictors of treatment response to brief behavioral treatment of insomnia (BBTI) in older adults. J Clin Sleep Med 9:1281–1289
19. Yazdi Z, Sadeghniiat-Haghighi K, Zohal MA, Elmizadeh K (2012) Validity and reliability of the Iranian version of the insomnia severity index. Malaysian J Med Sci: MJMS 19:31
20. Morin CM (1993) Insomnia: psychological assessment and management. Guilford press
21. Pichot V, Roche F, Celle S, Barthelemy J-C, Chouchou F (2016) HRVanalysis: a free software for analyzing cardiac autonomic activity. Front Physiol 7:557
22. Lai J, Ma S, Wang Y, Cai Z, Hu J, Wei N, Wu J, Du H, Chen T, Li R (2020) Factors associated with mental health outcomes among health care workers exposed to coronavirus disease 2019. JAMA Netw Open 3:e203976–e203976
23. Baglioni C, Battagliese G, Feige B, Spiegelhalder K, Nissen C, Voderholzer U, Lombardo C, Riemann D (2011) Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies. J Affect Disord 135:10–19
24. McCraty R, Shaffer F (2015) Heart rate variability: new perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk. Global Adv Health Med 4:46–61
25 Akselrod S, Gordon D, Ubel FA, Shannon DC, Berger AC, Cohen RJ (1981) Power spectrum analysis of heart rate fluctuation: a quantitative probe of beat-to-beat cardiovascular control. Science 213:220–222
26. Van den Bergh O (2021) Principles and practice of stress management. Guilford Publications
27. Pomeranz B, Macaulay R, Caudill MA, Kutz I, Adam D, Gordon D, Kilborn KM, Barger AC, Shannon DC, Cohen RJ (1985) Assessment of autonomic function in humans by heart rate spectral analysis. Am J Phys Heart Circ Phys 248:H151–H153
28. Malliani A, Pagani M, Lombardi F, Cerutti S (1991) Cardiovascular neural regulation explored in the frequency domain. Circulation 84:482–492
29. Billman GE (2013) The LF/HF ratio does not accurately measure cardiac sympatho-vagal balance. Front Physiol 4:26
30. Pagani M, Lombardi F, Guzzetti S, Sandrone G, Rimoldi O, Malfatto G, Cerutti S, Malliani A (1984) Power spectral density of heart rate variability as an index of sympatho-vagal interaction in normal and hypertensive subjects. J. Hypertens. Supplement: official journal of the International Society of Hypertension 2:S383–S385
31. Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R, Pizzinelli P, Sandrone G, Malfatto G, Dell'Orto S, Piccaluga E (1986) Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. Circ Res 59:178–193
32. Jarrin DC, Chen IY, Ivers H, Lamy M, Vallières A, Morin CM (2016) Nocturnal heart rate variability in patients treated with cognitive–behavioral therapy for insomnia. Health Psychol 35:638
33. Shaffer F, McCraty R, Zerr CL (2014) A healthy heart is not a metronome: an integrative review of the heart's anatomy and heart rate variability. Front Psychol 5:1040
34. Irwin MR, Olmstead R, Carrillo C, Sadeghi N, Breen EC, Witarama T, Yokomizo M, Lavretsky H, Carroll JE, Motivala SJ (2014) Cognitive behavioral therapy vs. Tai Chi for late life insomnia and inflammatory risk: a randomized controlled comparative efficacy trial. Sleep 37:1543–1552