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The impact of posttraumatic stress disorder versus resilience on nocturnal autonomic nervous system activity as functions of sleep stage and time of sleep

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HIGHLIGHTS

- Autonomic activity as a function of sleep stage was associated with PTSD status.
- Heart rate was elevated in PTSD compared to resilience at the beginning of sleep.
- Heart rate declined over time of sleep at a faster rate in PTSD than resilience.

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ABSTRACT

Posttraumatic stress disorder (PTSD) has been associated with sleep disturbances including alterations in sleep stages and recently, elevated nocturnal autonomic nervous system (ANS) arousal (i.e., dominance of the sympathetic nervous system over the parasympathetic nervous system). Data suggest that sleep contributes to the regulation of ANS activity. In our previous ambulatory heart rate variability (HRV) monitoring study, strong relationships between sleep and nocturnal ANS activity in resilient participants (i.e., individuals who had never had PTSD despite exposure to high-impact trauma) were not seen with PTSD. In this study, we examined the impact of PTSD vs. resilience on ANS activity as a function of sleep stage and time of sleep. Participants (age 18–35) with current PTSD ($n = 38$) and resilience ($n = 33$) completed two overnight polysomnography recordings in a lab setting. The second night electrocardiogram was analyzed for frequency domain HRV parameters and heart rate within rapid-eye-movement (REM) and non-REM (NREM) sleep periods. Results indicated that ANS arousal indexed by HRV was greater during REM compared with NREM sleep and that the REM–NREM difference was greater in the PTSD than in the resilient participants. This effect of PTSD was reduced to non-significance when analyses controlled for REM sleep percentage, which was lower with PTSD. Exploratory analyses revealed that the REM–NREM difference in HRV was correlated with REM sleep percentage in resilient participants, but not with PTSD. In contrast with our data from home settings, the present study did not find increased overall nocturnal ANS arousal with PTSD. Analyses did reveal higher heart rate during initial NREM sleep with more rapid decline over the course of NREM sleep with PTSD compared with resilience. Findings suggest that elevated ANS arousal indexed by heart rate with PTSD is specific to the early part of sleep and possible impairment in regulating ANS activity with PTSD related to REM sleep.

1. Introduction

Insomnia and recurrent nightmares related to trauma are common symptoms of posttraumatic stress disorder (PTSD) [1,2] and are included in the hyperarousal and re-experience symptom clusters of its diagnostic criteria [3]. Despite the prominence of those symptoms, objective indices of nocturnal hyperarousal have been elusive. Polysomnographic (PSG) studies have not consistently documented impaired sleep initiation and maintenance in PTSD [4]. Studies examining nocturnal arousal in PTSD have utilized indices of autonomic nervous system (ANS) activity including heart rate (HR) and HR variability (HRV). Some, but not all, studies have found evidence for heightened ANS arousal [i.e., dominance of the sympathetic nervous system over the parasympathetic nervous system indexed by increased HR and a decreased high-frequency (HF) component of HRV] during sleep with PTSD [5–8].

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Studies have also suggested associations of PTSD with sleep-stage specific alterations, including increased shallow sleep (Stage 1) and reduced slow-wave sleep [4], increased rapid-eye-movement (REM) density, and fragmented patterns of REM sleep [9–11]. Trauma-related nightmares are prominent symptoms of PTSD [12], and dreams are associated with REM sleep [13]. ANS activity changes as a function of sleep stage in normal sleepers [14,15]. We could only identify two studies that examined sleep-stage specific ANS activity in trauma-exposed individuals, one with developing PTSD [16] and the other with established PTSD [7]. The study by Mellman et al. [16] examined nocturnal ANS activity within a month of a traumatic injury using ratios of low frequency to high frequency components (LF/HF) of HRV as an index of sympathetic tone [17]. They found elevated LF/HF during REM sleep in injured patients who subsequently developed PTSD compared with those who did not develop the disorder. Consistent with studies of normal sleepers [14,15], LF/HF and HR were greater during REM sleep than during non-REM (NREM) sleep in this study (LF/HF) and the study by Woodward et al. [7] (HR). The study by Woodward et al. further suggested that PTSD and the percentage of sleep comprised of REM sleep (REM%) moderate this REM–NREM difference in ANS activity since the veterans with PTSD had smaller REM–NREM differences in HR than controls and that REM% was negatively correlated with REM–NREM HR differences in those with PTSD. HRV was not reported in the study. Therefore, no study has reported HRV during sleep as a function of sleep stage in established PTSD.

Sleep appears to have a critical role in regulating ANS activity. Normalized LF (also an index of sympathetic tone) and HR have been found to be reduced during both nighttime and daytime sleep compared with wake [18]. Reductions from sleep onset to the morning in HR and increase in pre-ejection period (consistent with decreased sympathetic tone) have been documented in normal sleepers [19–21], and these effects were also observed after controlling for sleep stage [15]. However, effects of time of sleep on ANS activity indexed primarily by HF measures have not been as consistent with findings of both increased and decreased parasympathetic tone as well as no change across sleep time [14,15,19,22,23]. In the aforementioned study by Mellman et al. of recently injured patients [16], LF/HF was higher during the first than the last REM period in both injured patients who subsequently did and did not develop PTSD. Bertram et al. [24] examined HR change over the course of sleep measured by actigraphy in individual with and without PTSD and found a reduction of HR across sleep time in both groups; however, this study did not control for the effects of sleep stages on HR, nor did it report HRV as a function of time of sleep. Therefore, influences of PTSD on ANS activity during REM and NREM sleep over the course of sleep have not been examined in established PTSD.

Arousal during sleep could also be affected by the sleep environment. Most PSG studies in PTSD have been conducted in laboratory settings, and it has been suggested that these environments contributed to the lack of consistent evidence for impaired sleep initiation and maintenance [25,26]. Researchers have anecdotally reported that participants with PTSD reported having slept better in the lab than their homes with a technician being “on guard” outside their sleeping room [25]. In fact, the two home PSG studies found longer sleep latency, reduced total sleep time and sleep maintenance in individuals with PTSD compared with those without PTSD [26,27]. We recently reported that individuals with PTSD had lower parasympathetic tone indexed by normalized high frequency (nHF) during sleep at home compared with resilient individuals (i.e., individuals who had never had PTSD despite exposure to high-impact trauma) [28].

In summary, PTSD has been associated with elevated ANS arousal during sleep and alterations in sleep stages; however, investigation into ANS activity as a function of sleep stage in PTSD and resilience has been limited despite connections between ANS activity and sleep stages. In addition, ANS arousal decreases over the course of sleep in normal sleepers. The reduction of HR across time asleep was also observed in individuals with PTSD in an actigraphic study; however, effects of sleep stage were not controlled in this study. Further, HRV as a function of time of sleep was not reported in this study despite that there have been discrepancies in findings between studies examining HR and HRV across sleep time in normal sleepers. The purpose of this report is to add the literature on nocturnal ANS activity in PTSD by describing the impact of PTSD vs. resilience on ANS activity as functions of sleep stage and time of sleep using HRV parameters and HR. Due to the suggestion of influences of the sleep environment on nocturnal arousal, we also examined whether the lower parasympathetic tone with PTSD compared with resilience found in our previous ambulatory study manifests in a lab setting.

2. Materials and methods

2.1. Participants and procedure

Participants of the present report were the subsample of physically healthy young adult African Americans (age 18–35 years) who met criteria for current full or subthreshold PTSD or resilience (the criteria for these groups are described below) and completed laboratory PSG as a part of a larger study on PTSD, sleep, neighborhood stress, and nocturnal blood pressure and ANS activity. Participants were recruited from the Washington, DC metropolitan area through flyers and referral from prior participants. During the initial phone or in-person screening, potential participants were excluded if they were found to have a body mass index ≥40, ongoing medical disorder that can affect blood pressure, use of medications that can affect blood pressure or sleep, severe mental disorders (psychotic disorders, bipolar disorder, severe recurrent depression), consuming >5 cups of coffee per day or its equivalent, smoking >20 cigarettes per day, drinking >14 alcoholic drinks/week in men or >7 drinks/week in women, and habitual bedtime and rise time after 2 AM and 10 AM, respectively, or habitual napping >1 h/day. Following the screening, participants were invited to the Howard University Clinical Research Unit where they completed informed consent and self-report surveys that included a demographic questionnaire, a checklist of traumatic experiences, and a measure of PTSD symptom severity ($N = 543$).

A subset of participants selected from those who filled out the self-report surveys were invited to the laboratory phase and completed a clinical interview, urine toxicology screening, height and weight measurement, and two consecutive-night PSG recordings in the research unit ($n = 185$). Participants for the laboratory phase were selected based on their answers to the self-report surveys to balance the sample by trauma exposure and PTSD status and gender and to increase representation of community residents as oppose to students living on campus. Additional exclusion criteria evaluated during the laboratory phase were sleep apnea defined as an apnea/hypopnea index of ≥10 on a screening sleep recording, positive urine toxicology for illicit drugs, current alcohol or drug dependence, and current psychiatric disorder other than PTSD, phobic disorder, generalized anxiety disorder, or depression that was secondary to PTSD evaluated through the structured clinical interview. Participants received $25 for completing the self-report survey, another $25 for the clinical interview, and $125 for each PSG recording. The study procedure was approved by the Howard University Institutional Review Board.

For the present analyses, we selected only participants who met the criteria for current PTSD ($n = 53$) or resilience ($n = 43$) and completed the second PSG recording. The PTSD participants met the Diagnostic and Statistical Manual of Mental Disorders — Fourth Edition [29] criteria for current PTSD or subthreshold criteria (meeting the criteria for at least two of the three symptom clusters). The resilient participants had never met PTSD criteria despite exposure to a high-impact traumatic event. High-impact trauma was designated for traumatic events that have been associated with high risk for engendering PTSD in prior