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Sleep Disturbance and Baroreceptor Sensitivity in Women with Posttraumatic Stress Disorder

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Abstract

In a previous study, women with posttraumatic stress disorder (PTSD) had greater objective sleep disturbance than those without. In a separate previous study, women with PTSD were also found to have lower baroreceptor sensitivity (BRS), an index of blood pressure regulation. In the present study, we concurrently assessed BRS and objective sleep by diagnostic status. Comparison of 32 women with PTSD to 21 women without PTSD revealed an interaction between BRS and sleep efficiency, Wake after Sleep Onset percentage, and sleep fragmentation. Lower BRS was associated with poorer sleep in women with PTSD, but not in those without. Future research should investigate causal relationships between sleep and blood pressure regulation in those with PTSD.

Research on posttraumatic stress disorder (PTSD)-associated health conditions is often focused on the physiological sequelae of prolonged stress. Although mechanisms are unclear, associations between PTSD and degraded health have been reported in numerous studies (Schnurr & Green, 2004), including some suggesting cardiovascular manifestations of PTSD (Bedi & Arora, 2007). Baroreceptor sensitivity (BRS), a measure of parasympathetic cardiac functioning, is an index of the body's ability to regulate blood pressure. In a sample of women with a range of trauma histories, those with PTSD had lower resting BRS than women without PTSD (Hughes, Feldman, & Beckham, 2006). Recently, evidence was presented for a relationship between PTSD and cardiac outcomes: Vietnam Veterans with PTSD were twice as likely to have died of early-onset heart disease at a 15-year follow-up relative to their non-PTSD counterparts (Boscarino, 2008).

Most individuals with PTSD complain of sleep disturbance (Ohayon & Shapiro, 2000). A recently published meta-analysis focused on polysomnographic studies of individuals with PTSD proposed an explanation for inconsistent findings across earlier studies and bolstered the argument in favor of objective evidence for these complaints (Kobayashi, Boarts, & Delahanty, 2007). Trauma-related sleep disturbance is associated with self-reported poorer health (Clum, Nishith, & Resick, 2001), and disrupted sleep is proposed as one possible mechanism explaining the consistent relationship between PTSD and the development of medical conditions (Germain, Shear, Hall, & Buysse, 2006).

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Given the empirical evidence for bivariate relationships between PTSD, cardiac function, and sleep disturbance, as well as our previous findings of greater objective sleep disturbance (Calhoun, et al., 2007) and lower BRS (Hughes, Dennis, & Beckham, 2007) in women with PTSD relative to those without, we examined the possibility that the relationship between objective sleep disturbance and cardiac function may differ by diagnostic status. In the current study, we examined associations between baroreceptor sensitivity, objective sleep disturbance, and diagnostic status in a sample of women.

Method

Participants

Women with PTSD and controls completed actigraphy (N = 124) as part of their participation in a larger study examining the relationships between hostility, health, and PTSD in women. Only women with complete actigraphy and BRS data (n = 53) were included in the analyses conducted for this study. Excluded participants (n = 71) were younger (M age = 36 years) than included participants (M age = 42 years), F (1, 122) = 5.76, $p < .05$, but did not differ in terms of race, marital status, BMI, cardiac medication use, PTSD severity, or self-reported sleep quality. Women with PTSD (n = 32) were compared to women without PTSD or depression (n = 21) on sleep disturbance and BRS. Recruitment flyers, consent procedures, and diagnostic evaluations based on the Clinician Administered PTSD Scale (Blake, et al., 1995) are presented in detail elsewhere (Beckham, Flood, Dennis, & Calhoun, 2009).

Participants with a diagnosis of PTSD were significantly older (PTSD M age = 45, SD = 13, control M age = 37, SD = 13), had higher BMI (PTSD M BMI = 32.07, SD = 7.41, control M BMI = 26.94, SD = 6.32), and were more likely to be unemployed (PTSD N = 13, 40.63%, control N = 2, 9.52%, χ^2 (1, N = 52) =6.04 relative to control subjects. The two cohorts did not differ significantly on race, African American, Caucasian, and other, χ^2 (1, N = 52) = 2.63, *ns*, marital status, married, and unmarried, $\chi^2 < 1$, or years of education, ≤ 12, 13–16, ≥ 17, χ^2 (2, N = 53) = 2.40, *ns*. Veteran representation was greater in the PTSD group (31%) than the control group (0%), χ^2 (1, N = 53) = 8.09, $p < .05$. All participants endorsed exposure to at least one lifetime traumatic experience. Participants with PTSD endorsed a median of seven events (range = 1–19) meeting criterion A for PTSD diagnosis as compared to a median of three events (range = 0–9) in the control group. BRS was negatively correlated with age in the sample, r (53) = −.51, $p < .001$, and subgroups. Participants with PTSD were more likely to be taking cardiac-affecting medications (37.5%) than the control group (4.8%), χ^2 (1, N = 53) = 7.34, $p < .01$. The following medications were considered cardiac-affecting medications: beta blockers, other antihypertensives, anticholinergics, and alpha-adrenergics. Only one participant in the entire sample (in the PTSD group) endorsed current use of sedative/hypnotic medications.

Measures

Objective measures included estimation of sleep parameters based upon actigraphy, and estimation of BRS derived from blood pressure (BP) and inter-beat interval. Actigraphy data was collected by participants at home over a one-week period using a wrist-watch style actigraph (Mini-Mitter Inc., Sun River, OR) to derive objective estimates of time in bed, total sleep time, sleep onset latency, wake after sleep onset, wake after sleep onset wake after sleep onset percentage (percentage of time awake after falling asleep and before final awakening), and sleep efficiency (percentage of time sleeping while in bed), in addition to fragmentation index (a measure of sleep fragmentation). Actigraphy data was collected within one month of the laboratory collected BP and inter-beat interval readings.

A Finapres BP monitor (Ohmeda, Madison, WI) was used to assess BP, and inter-beat interval was estimated from blood pressure pulse wave. This data was collected in the laboratory on one occasion during a five-minute resting period. Average values were computed from the beat-to-beat BP record for BP and inter-beat interval. Mean heart rate was calculated as 60,000 divided by the mean of the inter-beat interval. An estimate of BRS was derived from systolic blood pressure and inter-beat interval values (Di Rienzo, Parati, Mancia, Pedotti, & Castiglioni, 1997). Lower values of BRS reflect less parasympathetic nervous system activity. Additional information regarding calculation of BRS can be found in Hughes et al, 2007.

Data Analysis

Analyses were conducted on BRS and mean sleep parameter scores for participants providing at least three nights of actigraphy data. Group differences were assessed using standard Chi-square procedures for categorical measures, t-tests for normally distributed data, and nonparametric Wilcoxon rank tests for non-normal distributions. Within each cohort, Fisher's *z* transformations were applied to compute partial correlation coefficients between BRS and sleep parameters, which were subsequently used to test for group differences. A generalized linear regression procedure was used to regress BRS on the following variables, controlling for covariates: sleep parameters (modeled singularly); a proxy for group status (PTSD = 1; Control= 0); and an interaction term (group status by sleep parameter). A main effects and an interaction model were estimated for each sleep parameter. The interaction term was used to assess for differential associations between sleep parameters and BRS, based upon group status. Because the BRS distribution was non-normal, models were estimated using an assumed gamma distribution with a log link (SAS 9.1; PROC GENMOD). Tests of model fit were based on Chi-square statistics associated with scaled deviance scores.

Results

Group differences on sleep parameters and BRS are presented in Table 1. Actigraphy readings suggest that, relative to controls, the PTSD group had more time in bed, more wake time after sleep onset, more wake percentage, longer latency to sleep onset, and lower sleep efficiency, in addition to more fragmented sleep. Groups did not differ on total sleep time. The PTSD group had lower BRS.

Table 2 outlines the *z* transformed Fisher partial correlations between BRS and the sleep parameters by group, and z tests for significance of differences between coefficients across groups. Correlations coefficients for relationships between BRS and the sleep parameters were significantly different between groups for wake after sleep onset percent and fragmentation index. No other comparisons attained significance.

Results of the multivariable regression analyses for interaction models are presented in Table 3. Main effects were not observed for sleep parameters. Among interaction models, the estimated coefficients associated with wake after sleep onset percentage, sleep efficiency, and fragmentation index attained significance. The coefficient estimated for the interaction of group and sleep efficiency was positive, indicating that as sleep efficiency increased in magnitude the negative association between a diagnosis of PTSD and BRS was mitigated. Negative coefficients were estimated for the interaction between group and fragmentation index and group and wake after sleep onset percent, indicating that the negative association between a diagnosis of PTSD and BRS was exacerbated as the magnitude of these sleep indices increased. Changes in the magnitude of sleep indices had no effect on BRS among control subjects for any of the sleep parameters.

Discussion

We evaluated whether the relationships between daytime BRS and objective sleep disturbance in individuals with PTSD differed from controls. Correlational analyses revealed differences in the relationship between actigraphy-assessed sleep disturbance and BRS in women with PTSD relative to controls. Multivariate interaction analyses also revealed differential associations between sleep and BRS by group. Greater sleep efficiency, lower wake percentage, and more consolidated sleep (lesser sleep fragmentation) were associated with higher BRS in women with PTSD. These relationships were not observed in the control group. The findings of our secondary analyses support one of several explanations that may be elucidated through future research. Specifically, improved sleep may promote increased blood pressure regulation in women with PTSD. Alternatively, it could be that reduced parasympathetic nervous system regulation, manifest in this sample as reduced blood pressure regulation, may increase the risk of developing PTSD and its' associated sleep disturbance. Studies designed to examine causal relationships will be needed to distinguish between these and other explanations.

Our findings are consistent with a study of anxious individuals that revealed higher heart rate, lower heart rate variability, and more movement during sleep in anxious individuals relative to non-anxious controls (Aikins & Craske, 2008). Our findings also converge with those for depressed insomniacs in which middle and late night insomnia were associated with lower cardiovascular control and which also found related improvements in both insomnia and cardiac vagal control from pre to post-assessment (Rottenberg, Chambers, Aleen, & Manber, 2007).

Limitations of the study include small sample size, and the need for replication with nighttime BRS assessment. Since BRS is calculated using HR and inter-beat interval, we would expect variation in BRS across sleep stage, as is the case with HR. Thus, our daytime BRS findings may translate only to certain sleep stages. Although we controlled for the effect of certain co-variates in our analyses, statistical control has its own limitations. The PTSD group was older and used more cardiac-affecting medications than the control group. BRS typically decreases with age, and cardiac medication usage can affect BRS differentially depending on drug class. Thus, the effects of these co-varying factors could be reflected in our findings, as well as respiration rate and tidal volume which were not assessed.

Nonetheless, our preliminary results are intriguing, particularly when considered in light of the evidence from Boscarino (2008) for early-age heart disease in PTSD-positive veterans, and associations between sleep disturbance and cardiovascular disease (Bonnet & Arand, 2007). Future studies could examine the relationship between sleep disturbance and cardiovascular disease in PTSD and trauma-exposed populations using longitudinal design, and compare these findings to those for healthy individuals.

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References

Aikins DE, Craske MG. Sleep-based heart period variability in panic disorder with and without nocturnal panic attacks. Journal of Anxiety Disorders. 2008; 22:453–463. [PubMed: 17449220]

Beckham J, Flood A, Dennis M, Calhoun P. Ambulatory cardiovascular activity and hostility ratings in women with chronic posttraumatic stress disorder. Biological Psychiatry. 2009; 65:268–272. [PubMed: 18692171]

Bedi US, Arora R. Cardiovascular manifestations of posttraumatic stress disorder. Journal of the National Medical Association. 2007; 99:642–649. [PubMed: 17595933]

Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Gusman FD, Charney DS, et al. The development of a Clinician-Administered PTSD Scale. Journal of Traumatic Stress. 1995; 8:75–90. [PubMed: 7712061]

Bonnet MH, Arand DL. Cardiovascular implications of poor sleep. Sleep Medicine Clinics. 2007; 2:529–538.

Boscarino JA. A prospective study of PTSD and early-age heart disease mortality among Vietnam veterans: Implications for surveillance and prevention. Psychsomatic Medicine. 2008; 70:668–676.

Calhoun PS, Wiley M, Dennis MF, Means MK, Edinger JD, Beckham JC. Objective evidence of sleep disturbance in women with posttraumatic stress disorder. Journal of Traumatic Stress. 2007; 20:1009–1018. [PubMed: 18157880]

Clum GA, Nishith P, Resick PA. Trauma-related sleep disturbance and self-reported physical health symptoms in treatment-seeking female rape victims. Journal of Nervous & Mental Disease. 2001; 189:618–622. [PubMed: 11580006]

Di Rienzo M, Parati G, Mancia G, Pedotti A, Castiglioni P. Investigating baroreflex control of circulation using signal processing techniques. IEEE engineering in medicine and biology magazine: The quarterly magazine of the Engineering in Medicine and Biology Society. 1997; 16:86–95.

Germain A, Shear MK, Hall M, Buysse DJ. Effects of a brief behavioral treatment for PTSD-related sleep disturbances: A pilot study. Behavior Research and Therapy. 2006; 45:627–632.

Hughes JW, Dennis MF, Beckham JC. Baroreceptor sensitivity at rest and during stress in women with posttraumatic stress disorder or major depressive disorder. Journal of Traumatic Stress. 2007; 20:667–676. [PubMed: 17955541]

Hughes JW, Feldman ME, Beckham JC. Posttraumatic stress disorder is associated with attenuated baroreceptor sensitivity among female, but not male, smokers. Biological Psychology. 2006; 71:296–302. [PubMed: 16011871]

Kobayashi I, Boarts JM, Delahanty DL. Polysomnographically measured sleep abnormalities in PTSD: A meta-analytic review. Psychophysiology. 2007; 44:660–669. [PubMed: 17521374]

Ohayon MM, Shapiro CM. Sleep disturbances and psychiatric disorders associated with posttraumatic stress disorder in the general population. Comprehensive Psychiatry. 2000; 41:469–478. [PubMed: 11086154]

Rottenberg J, Chambers AS, Allen JJ, Manber R. Cardiac vagal control in the severity and course of depression: The importance of symptomatic heterogeneity. Journal of Affective Disorders. 2007; 103:173–179. [PubMed: 17320191]

Schnurr, PP.; Green, BL. Trauma and health: Physical health consequences of exposure to extreme stress. Washington, DC: American Psychological Association; 2004.

Table 1

Means, Standard Deviations, and Difference Statistics for Actigraphy and BRS scores

Sleep Parameters	**PTSD (*n* = 32)**		**Control (*n* = 21)**		
	M	***(SD)***	***M***	***(SD)***	***F* (1, 51)**
Time in bed (minutes)	487.62	(72.40)	449.10	(60.50)	4.07*
Total sleep time (minutes)	376.56	(76.38)	375.56	(51.62)	< 1.00
Wake after sleep onset (minutes)	64.71	(24.71)	48.57	(22.17)	5.86*
Wake after sleep onset percent	14.87	(6.45)	11.65	(4.05)	4.16*
Sleep efficiency (%)	77.37	(11.10)	83.95	(5.67)	6.27*
Sleep onset latency (minutes)	26.72	(28.72)	10.32	(10.67)	6.25*
Fragmentation index	35.79	(13.65)	27.66	(8.22)	6.01*
Baroreceptor sensitivity (BRS)	11.83	(6.15)	21.74	(15.73)	10.38**

* $p < .05$.

** $p < .01$.

Table 2

Fisher Partial Correlations between BRS and Actigraphy Sleep Parameters (controlling age, BMI, and Cardiac-affecting Medications)

	BRS		Difference Statistic
	PTSD (*n* = 28)	**Controls (*n*= 17)**	***Z***
Time in bed	−.15	−.21	< 1.00
Total sleep time	.02	−.36	−1.26
WASO[a]	−.45	−.10	1.16
WASO %	−.42	.23	−2.16*
Sleep efficiency (%)	.24	−.30	−1.80
Sleep onset latency	.10	.27	< 1.00
Fragmentation index	−.38	.35	2.44*

* p <.05,

[a] Wake after sleep onset

Table 3

Summary of Generalized Linear Regression Models predicting BRS (N = 53)

Variable	Coefficient	*SE*	X^2
Model 1 (Time in Bed)			
Age	−0.02	0.01	11.81***
BMI	−0.00	0.01	<1.00
Cardiac-affecting medications	−0.04	0.20	<1.00
TIB[a]	−0.00	0.00	1.98
Group	−1.18	1.06	1.24
TIB X Group	0.00	0.00	<1.00
Model 2 (Total Sleep Time)			
Age	−0.02	0.01	9.80**
BMI	−0.00	0.01	<1.00
Cardiac-affecting medications	−0.07	0.20	<1.00
TST[b]	−0.00	0.00	3.36
Group	1.96	0.96	4.19*
TST X Group	0.00	0.00	2.88
Model 3 (Wake After Sleep Onset)			
Age	−0.02	0.01	13.05***
BMI	0.00	0.01	<1.00
Cardiac-affecting medications	−0.03	0.19	<1.00
WASO[c]	−0.00	0.00	<1.00
Group	0.08	0.34	<1.00
WASO X Group	−0.01	0.01	1.65
Model 4 (Wake After Sleep Onset Percentage)			
Age	−0.02	0.01	10.42**
BMI	0.00	0.01	<1.00
Cardiac-affecting medications	−0.05	0.18	<1.00
WASO%	0.03	0.03	1.18
Group	0.48	0.38	1.59
WASO % X Group	−0.07	0.03	5.81*
Model 5 (Sleep Efficiency)			
Age	−0.02	0.01	10.11**
BMI	0.00	0.01	<1.00
Cardiac-affecting medications	−0.05	0.20	<1.00
Sleep efficiency	−0.02	0.02	1.34
Group	−3.62	1.73	4.70*
Sleep Efficiency X Group	0.04	0.02	3.86*
Model 6 (Sleep Onset Latency)			
Age	−0.02	0.01	10.88**

Variable	Coefficient	*SE*	X^2
BMI	−0.00	0.01	<1.00
Cardiac-affecting medications	−0.08	0.20	<1.00
SOL[d]	0.01	0.01	1.60
Group	−0.24	0.21	1.30
SOL X Group	−0.01	0.01	1.36
Model 7 (Fragmentation Index)			
Age	−0.02	0.01	12.24***
BMI	0.00	0.01	<1.00
Cardiac-affecting medications	−0.01	0.18	<1.00
Fragmentation index	0.02	0.01	2.04
Group	0.71	0.44	2.71
Fragmentation Index X Group	−0.04	0.01	6.94**

*$p < .05$,

**$p < .01$,

***$p < .001$.

[a]Time in Bed,

[b]Total Sleep Time,

[c]Wake after Sleep Onset,

[d]Sleep Onset Latency