Polysomnography-detected Bruxism in Children is Associated with Somatic Complaints but not Anxiety

Short Title: Sleep Bruxism in Children with and without Anxiety

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Abstract

Study Objectives: Sleep bruxism (SB) is common in children and is associated with somatic symptoms and sleep disturbance. Etiological theories posit the role of anxiety, suggesting youth with anxiety disorders may be at high-risk for SB, but empirical data are lacking. Furthermore, parent report rather than polysomnography (PSG) has been used to examine SB-anxiety relationships in children. We examined rates of PSG-detected compared to parent-reported SB in children with generalized anxiety disorder (GAD) and healthy controls. Associations among SB, somatic complaints, and sleep disturbance were also examined.

Methods: Thirty-one children, aged 7-11 years, completed one night of PSG monitoring and 7 daily reports of somatic symptoms. Bruxism events were scored during rapid eye movement (REM) sleep and non-REM sleep stages 1 and 2.

Results: Almost one-third of children showed evidence of SB based on PSG. No associations were identified between parent-reported and PSG-detected SB. Rates of SB did not differ between anxious and control groups, though children with GAD showed more tonic bruxisms during REM sleep. Presence of SB predicted more muscle aches and stomach aches, and children with SB had more awake time after sleep onset than non-bruxers.

Conclusions: Results indicate poor concordance between PSG-detected and parent-reported SB in children, suggesting that parent report alone is not a reliable method for detection. The lack of association between SB and anxiety status suggests that stress sensitivity rather than anxiety per se, may be predictive of SB. Associations between SB, somatic symptoms, and sleep disturbance are congruent with the broader literature.

Key words: Sleep, bruxism, children, anxiety, somatic, polysomnography

Brief Summary

Current Knowledge/Study Rationale: Research examining sleep bruxism (SB) in children has relied almost exclusively on parent reports which may be biased by certain parent characteristics and/or child behaviors (e.g., anxiety). We used both polysomnography (PSG) and parent reports to examine rates of SB in children with an anxiety disorder compared to healthy controls, including associations with daytime somatic symptoms and objective sleep disturbance.

Study Impact: Parent-reported and PSG-identified SB showed no association, raising concern about the reliability of subjective reports. Rates of SB were similar in the anxious and control groups suggesting that temperamental factors other than anxiety may be more closely associated with SB (e.g. stress sensitivity). SB was linked with somatic symptoms and sleep disturbance in all youth.

Sleep bruxism (SB) is a sleep-related movement disorder characterized by involuntary and non-functional grinding or clenching of the teeth. SB is more common in children than adults[1] though prevalence rates in youth range widely, from 13% to 49%.[2-5] Because a majority of nighttime bruxing episodes coincide with arousals from sleep,[6-8] sleep disruption is a common feature of SB. Further, since the force of bruxing during sleep often exceeds the amplitude of maximum bite force during waking hours, tooth and oral tissue damage are common.[9] Other clinical consequences of repeated and sustained masticatory muscle activity include temporomandibular joint pain, jaw movement limitation, muscle aches, and chronic headache.[10,11]

SB is also associated with a range of impairing somatic symptoms. Among adult sufferers, SB is most consistently associated with headaches.[12] Although more limited, findings from child studies mirror adult-based outcomes. For example, in one study, SB (detected via parent report) was identified in 29% of children who experienced migraines.[13] A linear relationship between migraine frequency and frequency of SB was also found. Another study based on polysomnography (PSG) found an increased prevalence of SB among children with tension headaches specifically.[14] Relationships between SB and other common types of somatic complaints in children, such as stomachaches and muscle aches, have been less commonly studied.

Although craniofacial structure and dental irregularities (e.g., dental malocclusion) can produce SB, etiological theories consistently highlight the role of psychological factors, and anxiety in particular.[15-17] However, findings among adult samples are largely inconsistent. Elevated levels of trait anxiety[18,19] and anxiety disorders[16] among bruxers have been found in some studies, while others have failed to find such differences.[20] Another study linked SB with somatic anxiety and muscle tension specifically,[17] and still other studies provide evidence of a relationship between anxiety and bruxism during wake but not sleep.[21,22]

Research examining SB-anxiety relationships in children is more limited but has produced more consistent findings. Several researchers have found significantly higher levels of self and parent-reported anxiety among children with SB compared to control groups.[23-25] Among an adolescent sample, Türkoğlu et al. found significantly higher levels of state anxiety and anxiety sensitivity as well as rates of anxiety disorders among children with SB compared to a matched control group. Generalized anxiety disorder and social phobia were the two most common diagnoses identified.[26]

An important limitation of previous studies is their reliance on parent report for diagnosing SB. In addition to the common use of non-validated questionnaires, parents tend to underestimate SB in their children,[27] except perhaps when sleeping in close proximity.[2] Other studies have relied on physical evidence of tooth damage among children recruited from dental clinics. Tooth damage is not exclusive to SB however, and signs of wear may not become apparent until the disorder has been present for several years. Further, investigation of youth recruited directly from dental clinics elevates the possibility of referral/selection bias.[28] For these reasons, measurement of muscle activity via submental electromyogram (EMG) as part of overnight polysomnography (PSG) monitoring is considered the most reliable indicator of SB.

The current study examined rates of SB in children with generalized anxiety disorder (GAD) compared to matched, typically-developing children. None of the participants were recruited specifically for bruxism, dental concerns, or sleep problems. Presence of SB was determined based on standard overnight PSG and concordance with parent reports of SB was examined. We also compared rates of SB based on diagnostic group status, including type of bruxism events and the sleep stage in which SB occurred. Finally, we investigated whether PSG-detected SB was related to daytime somatic complaints and sleep disturbance in all children. Based on previous research we expected to find higher rates of SB in the clinically-

anxious group and that SB would correspond with greater somatic complaints and wake minutes during the sleep period (i.e., sleep disturbance).

Methods

Participants

The sample included 31 children (14 male) aged 7-11 years (*M* = 8.87, *SD* = 1.45). A total of 14 children met criteria for primary generalized anxiety disorder (GAD) and 17 had no psychiatric diagnoses. Children were recruited via flyers and print advertisements for a study about emotion and behavior at a pediatric hospital in Washington DC. To be eligible, children had to be fluent in English, live with a parent or caregiver for a minimum of one year, and be enrolled in regular education classes. Exclusion criteria included the use of any medications or any illnesses known to impact sleep, current use of treatment services for anxiety or sleep problems, full scale IQ < 85, a current or lifetime history of psychotic disorder, pervasive developmental disorder, bipolar disorder, eating disorder, substance use, or suicidal ideation/self-harm. Also, because a primary aim of the parent study was to characterize the sleep architecture of children with GAD compared to healthy controls, any child with previously diagnosed or suspected breathing-related sleep disorder (e.g., based on parent report of snoring, gasps, or pauses in breathing during sleep) was excluded from participating due to potential confounding effects on sleep architecture. PSG confirmed that no child had a breathing-related sleep disorder.

Demographic characteristics for the full sample and both groups are included in Table 1. No significant differences were detected between groups for any demographic variable. Among the clinically-anxious sample, comorbid diagnoses included social anxiety disorder (*n* = 3), Attention-deficit/hyperactivity disorder (*n* = 3), separation anxiety disorder (*n* = 1), specific phobia (*n* = 1), depression (*n* = 1), and oppositional defiant disorder (*n* = 1).

Study Procedures

All procedures were approved by an Institutional Review Board and parents and children provided consent/assent. Study participation involved an initial diagnostic assessment, one week of actigraphy and nightly phone calls, and an overnight PSG. During the diagnostic assessment, children and their parents completed separate semi-structured interviews and filled out questionnaires. Afterward, eligible children were given actigraphs to wear for one week. Each evening during the actigraphy week, study staff telephoned families and asked parents and children about anxiety and somatic symptoms experienced that day. Immediately following the one-week assessment, children completed an overnight PSG in a sleep laboratory, accompanied by their parent or guardian. Actigraphy data were examined to ensure adequate sleep prior to the PSG night. All families were compensated for their time.

Measures

Diagnostic interview. All children underwent structured diagnostic interviews using the Anxiety Disorders Interview Schedule for DSM-IV - Child and Parent versions (ADIS-C/P).[29] The ADIS-C/P assesses a range of clinical symptoms, including anxiety, mood, and externalizing disorders and is considered the gold standard for diagnosing anxiety disorders in children, with high inter-rater and test-retest reliability.[30,31]

Daily Symptom Reports. Phone calls were completed daily on 7 consecutive evenings during which the parent and child indicated whether the child experienced any of the following symptoms that day: 1) muscle aches/tension, 2) headaches, and 3) stomach aches. All symptoms were rated on a scale from 0 (none) to 3 (a lot).

Bruxism Measures.

Child Sleep Habits Questionnaire (CSHQ).[32] Parents completed the CSHQ, a well-validated scale used to screen for a variety of sleep problems in children.[32] The CSHQ includes the item, “Child grinds teeth during sleep” to which parents responded using 3-point scale: 1

(rarely), 2 (sometimes), 3 (usually). SB was considered to be present if parents reported that their child *sometimes* or *usually* grinds their teeth at night.

Polysomnography (PSG). Standard, multichannel PSG was conducted using Medcare amplifiers and Rembrandt 9.0 Sleep Acquisition Software. Registered polysomnographic sleep technicians (RPSGT) experienced in working with children and scoring pediatric sleep records conducted and scored sleep studies in 30-second epochs based on American Academy of Sleep Medicine (AASM) criteria.[33] All studies were conducted and scored under the supervision of a board-certified sleep physician. All technicians were blinded to child diagnostic status. Electroencephalogram (EEG; frontal, central, and occipital regions), electrooculogram (EOG), electromyogram (EMG; submental, right/left tibial), electrocardiogram (ECG), nasal pressure, thoracic and abdominal respiratory effort, and oximetry data were collected.

PSG-Detected Bruxism. Bruxism events were scored by trained study staff with previous PSG scoring experience and blind to child group status. Scoring was conducted in 30 second epochs in accordance with AASM criteria.[33] Bruxism events were scored during rapid eye movement (REM), non-REM sleep stage 1 (N1), and non-REM sleep stage 2 (N2). Powerline filters were set at 60Hz, with additional high and low pass EMG filters set at 100Hz and 10Hz, respectively. Bruxism events were classified as either tonic (sustained EMG burst lasting more than 2 seconds), phasic (3 or more rhythmic EMG bursts of 0.25 to 2 seconds in duration), or mixed (both sustained and rhythmic). Children showing more than 2 bruxism episodes per hour of sleep were diagnosed with SB.[34,35]

Statistical Analyses

Normality analyses were conducted on all variables associated with PSG-identified SB (e.g., total episodes per hour, episodes per hour during N2, episodes per hour during REM) and daytime symptoms. The Shapiro-Wilk test indicated that all variables deviated significantly from normality ($p < .001$), thus non-parametric analyses were conducted. Since overall endorsement

of child and parent-reported symptoms during the 7-days of daily phone calls was low (i.e., the distribution was positively skewed), daily symptoms were examined as dichotomous variables whereby the symptom was considered to be present if either the child or parent endorsed its presence. Group differences in categorical variables (e.g., parent-reported and PSG-detected SB) were examined using chi square tests, and continuous variables (e.g., bruxism episodes per hour) were examined using Mann Whitney U tests. Regression models were conducted to examine whether SB was associated with daytime somatic symptoms.

Results

Missing Data

Three participants were missing data for two of the seven daily phone calls and parent-report of SB was missing for one child. Three children had some missing data during PSG recordings due to lost EEG or EMG electrodes during the night, though all PSG recordings contained N1, N2 and REM sleep. Among the three children with missing PSG data, one showed evidence of SB while two were considered non-bruxers. Because the presence of SB was detected based on bruxism events per hour of sleep, and given the small sample size, we included partial PSG records in our analysis.

Rates of SB Based on PSG and Parent Reports

According to PSG data, 32.3% (*n* = 10) of all children showed evidence of SB. By comparison, 26.7% (*n* = 8) of children were identified to have SB according to parent-reports. However, a chi square test showed no significant association between parent-reported and PSG-detected SB in the full sample. We also examined within-group associations between PSG and parented-detected SB for anxious and healthy children separately. Fisher's exact tests showed no association between measurements in either group.

SB in Children with and without Anxiety Disorders

As shown in Table 2, overall rates of SB did not differ between the clinically-anxious and control groups based on either PSG or parent-reports. Given the exploratory nature of the study, we also examined whether the groups differed in terms of type of bruxism events (tonic or phasic) or the sleep stage (N1, N2 or REM sleep) in which they occurred. No group differences were detected for tonic or phasic bruxism episodes or for SB episodes detected during N1, N2, or REM sleep. However, children with GAD showed a significantly greater proportion of tonic bruxism episodes during REM sleep ($U = 63.50$, $p = .03$). No other group differences were detected.

SB and Somatic Symptoms

Logistic regression models were used to examine whether SB predicted different types of somatic complaints. Three models were run with number of PSG-detected bruxism episodes per hour as the independent variable and each of the three somatic symptoms (headache, muscle ache, and stomach ache) as dependent variables. Number of bruxism episodes per hour significantly predicted the presence of muscle aches ($p = .01$) and stomach aches ($p = .02$) but not headaches in all children (see Table 3).

Impact of SB on Sleep

Comparison of PSG sleep variables and architecture between the clinically-anxious and control children included in the current study have been reported elsewhere, with no differences found for total sleep time (TST) or wake minutes after sleep onset (WASO).[36] We therefore provide descriptive statistics for sleep variables based on SB status (only) in Table 4. As shown, sleep onset latency, TST, and sleep efficiency did not differ between children with and without PSG-identified SB. Similarly, no differences in sleep stage distribution (N1, N2 or REM percentage) or arousal index were found based on SB status. However, children in the SB group showed a significantly greater proportion of WASO than the control group (Mann Whitney $U = 44.0$, $p < .01$).

Discussion

To our knowledge, this is the first study to use overnight PSG to detect SB in a sample of school-aged children with anxiety disorders and matched typically-developing controls. Several interesting findings emerged. First, overall rates of SB found using PSG (32.3%) and parent reports (26.7%) are generally consistent with rates found in several previous studies. At the same time, we found no relationship between the children identified using each of these methods. Because PSG is the most reliable method available for detecting SB, these results suggest that parents incorrectly identify some children as having SB while missing others. While night-to-night variability in SB might account for reduction in agreement rates, we found no meaningful relationship overall between parent reports and PSG. The clinical and research implications of this finding are significant in suggesting that parent reports alone are inadequate for reliable detection of SB in children.

A number of studies have documented elevated rates of anxiety and anxiety disorders (including GAD) among children with SB,[23-26] leading us to expect to find higher rates of SB in our clinical group. However, no differences in SB were detected between clinically-anxious and typically-developing children based on either PSG or parent reports. Also, since only PSG can provide information about the type and timing bruxing events, we compared the groups based on type of bruxism episodes (tonic and phasic) and the sleep stage (N2 and REM) in which they occurred. Non-significant differences were found for all comparisons with the exception of a significantly greater proportion of tonic bruxism episodes during REM sleep among children with GAD. Although interesting, the reliability of this finding is unclear given the number of comparisons in our study and it awaits replication.

Previous studies linking SB with elevated rates of child anxiety have relied solely on parent reports and/or dental examination. While our study is unique in its inclusion of children with clinical levels of anxiety, rates of SB among children with GAD and healthy controls did not

differ based on either PSG or parents reports. Thus, in line with findings from several adult-based studies,[20,37,38] anxiety may be an insensitive predictor of SB in children. Although expensive and unnecessary in most cases, PSG is the only method by which SB can be detected unequivocally.[39] A further possible explanation for divergent findings includes the fact that our sample was not recruited from a dental clinic, nor were assessment measures specific to teeth grinding or clenching, both of which might significantly increase the subjective detection of SB.

Another novel finding among our child sample includes a greater incidence of muscle aches and stomachaches among children with SB. Ferreira-Bacci and colleagues[24] similarly found elevated levels of somatic symptoms, including stomachaches, among children with SB. However, contrary to a wealth of data in both children and adults, we found no association with headaches in our sample. It bears mentioning that previous findings suggest a link between SB and tension headaches specifically[14] which are typically triggered by stress and characterized by bilateral, tightening pain of the scalp or neck.[40] Since tension headaches also tend to be milder in pain intensity and shorter in duration than other types of headaches,[40] they may have been more easily overlooked during evening phone calls. It is also possible that sensitivity to stress[20,37,38] moderates the occurrence of tension headaches in children, which was not examined in the present study.

In line with previous research, children with SB evidenced significantly greater sleep disruption in the form of wake minutes during the sleep period than non-SB children. The specific consequences of sleep disruption in this population are poorly understood at present, and the extent to which sleep fragmentation, either alone or in conjunction with sustained masticatory muscle activity, might produce greater physical and mental health problems among children who brux is also not known. Previous research has nonetheless found number of

arousals during sleep to correspond with both higher somatic and behavior problems in children with SB,[6] suggesting this to be a vital area for future investigations.

Our study has several limitations. First, our sample size was relatively small and our results require replication among larger samples of clinically-anxious youth. Children in the anxious group were not receiving any form of treatment which could be indicative of less severe anxiety than found in other clinically-anxious samples. One night of PSG monitoring might have been inadequate to capture SB among children who grind/clench their teeth only occasionally. PSG was also conducted in a novel, sleep laboratory environment which could have increased stress and/or anxiety in all children and artificially reduced some of the variability observed between groups. Although we did not utilize video monitoring in the current study, this can be helpful in distinguishing SB from other orofacial and masticatory movements. We also assessed somatic symptoms during the week leading up to the PSG night which is less ideal than assessing SB and somatic complaints over the same period.

It also seems important to note that we identified SB based on the amplitude of submental EMG activity relative to background EMG. Although this method is consistent with AASM criteria and previous studies, it may be less sensitive in detecting SB when concern regarding the overall variability of muscle activity exists. For example, individuals with GAD show increased levels of muscle activity and tension during wake[6] which may also be present during the sleep period and complicate detection of SB. Future studies using PSG to investigate SB in this population might therefore consider the use of alternative methods.

In conclusion, reported rates of SB in children vary greatly, with the highest rates identified in studies relying solely on parent reports.[1] In addition to the range of factors that can bias subjective assessments, the current lack of validated diagnostic criteria for SB in children undoubtedly contributes to divergent findings in the literature. We found roughly one-third of our sample to have evidence of SB based on PSG, but overall agreement with parent-detected SB

was low. Our study also failed to provide evidence of higher rates of SB among a group of clinically-anxious compared to typically-developing controls (based on either PSG or parent reports). We did identify a greater proportion of tonic bruxism episodes during REM sleep in the anxious group, but this novel finding awaits replication. Lastly, consistent with previous research, the presence of SB was associated with greater somatic complaints and sleep disruption in all children. Investigation into whether and how these symptoms might serve as mechanisms for the development of other health problems among children with SB is critical.

Abbreviations

AASM	American Academy of Sleep Medicine
ADIS-C/P	Anxiety Disorders Interview Schedule for Children and Parents
CSHQ	Child Sleep Habits Questionnaire
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders 4th Edition
ECG	Electrocardiogram
EEG	Electroencephalogram
EMG	Electromyogram
EOG	Electrooculogram
GAD	Generalized Anxiety Disorder
N1	Non-REM stage 1
N2	Non-REM stage 2
PSG	Polysomnography
REM	Rapid Eye Movement
SB	Sleep Bruxism
TST	Total Sleep Time

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Table 1. Group Demographics for the Full sample, Anxious, and Control groups

	Anxious (n = 14)	Control (n = 17)	Total Sample (N = 31)
	M (SD) / % (n)	M (SD) / % (n)	M (SD) / % (n)
Age	8.58 (1.60)	9.11 (1.32)	8.87 (1.46)
Gender (Female)	57.1% (8)	52.9% (9)	54.8 (17)
Race/Ethnicity			
Caucasian	64.3% (9)	76.5% (13)	71% (22)
African-American	7.1% (1)	17.6% (3)	12.9% (4)
Asian	7.1% (1)	0% (0)	3.2% (1)
Hispanic	7.1% (1)	0% (0)	3.2% (1)
Other/Biracial	14.3% (2)	5.9% (1)	9.7 (3)
Income			
< $10K	0% (0)	11.8% (2)	6.5% (2)
$10-40K	14.3% (2)	0% (0)	6.5% (2)
$40-60K	7.1% (1)	0% (0)	3.2% (1)
$60-80K	0% (1)	5.9% (1)	3.2% (1)
$80-100K	7.1% (1)	17.6% (3)	12.9% (4)
>$100K	71.4% (10)	64.7% (11)	67.7% (21)

Maternal Education			
Some grade school	7.1% (1)	0% (0)	3.2% (1)
Completed high school	0% (0)	11.8% (2)	6.5 (2)
Some College	28.6% (4)	0% (0)	12.9 (4)
Completed College	14.3% (2)	41.2% (7)	29% (9)
Advanced Degree	50% (7)	47.1% (8)	48.4% (15)
Paternal Education			
Some grade school	0% (0)	0% (0)	0 % (0)
Completed high school	7.1% (1)	5.9% (1)	6.5% (2)
Some College	14.3% (2)	5.9% (1)	9.7% (3)
Completed College	7.1% (1)	52.9% (9)	32.3% (10)
Advanced Degree	64.3% (9)	29.4% (5)	45.2% (14)
Parent Marital Status			
Married	71.4% (10)	88.2% (15)	81% (25)
Other (e.g., Single, Divorced)	28.6% (4)	11.8/5 (2)	19% (6)

Table 2. Differences in SB Variables based on Diagnostic Group Status

	Total Sample (N = 31)	Anxious (n = 14)	Control (n = 17)	*U* or *X*	*p* value	Effect Size *R*
Overall Bruxism						
Parent-reported SB (#/%)	8(26.7%)	4(28.6%)	4(25%)	.14	.71	.07
PSG-detected SB (#/%)	10(32.3%)	5(35.7%)	5(29.4%)	.05	.83	.04
Total Bruxism Index (M/SD)[a]	1.83(1.55)	1.97(1.40)	1.71(1.69)	99.0	.44	.08
Bruxism Events N1 (M/SD)[b]	1.26(2.14)	1.29(2.27)	1.24(2.11)	118.5	.99	.01
Bruxism Index N2 (M/SD)[c]	2.33(2.69)	2.58(3.00)	2.12(2.47)	111.5	.77	.05
Bruxism Index REM (M/SD)[c]	2.47(2.23)	3.04(2.54)	1.99(1.88)	87.0	.20	.23
Phasic Bruxism						
Total Phasic Bruxism Index (M/SD)[a]	1.23(1.25)	1.26(1.02)	1.21(1.45)	102.5	.53	.02
Phasic Bruxism Events N1 (M/SD)[b]	.94(1.77)	.93(1.82)	.94(1.78)	113.0	.83	.00
Phasic Bruxism Index N2 (M/SD)[c]	1.38(1.87)	1.69(2.58)	1.61(2.32)	111.0	.76	.02
Phasic Bruxism Index REM (M/SD)[c]	2.21(4.89)	1.94(2.03)	1.09(1.23)	88.0	.23	.25

Tonic Bruxism

Total Tonic Bruxism Index (M/SD)[a]	.56(.51)	.68(.51)	.45(.50)	80.5	.13	.22
Tonic Bruxism Events N1 (M/SD)[b]	.32(.65)	.36(.63)	.29(.69)	108.5	.66	.05
Tonic Bruxism Index N2 (M/SD)[c]	.60(.63)	.76(.71)	.46(.54)	82.0	.14	.27
Tonic Bruxism Index REM (M/SD)[c]	.98(1.10)	1.23(.78)	.78(1.30)	63.5	.03	.40

[a]Total Bruxism Index calculated as total number of bruxism events divided by TST in hours

[b]Bruxism Index was not calculated in N1 as less than one hour of N1 occurred during the night.

[c]N2, REM Bruxism Index calculated as number of bruxism events per stage divided by TST for each sleep stage

SB: Sleep Bruxism; PSG: Polysomnography; REM: Rapid Eye Movement; N1: Non REM Stage 1; N2: Non REM stage 2; TST: Total Sleep Time

Table 3 - Sleep Bruxism as a Predictor of Child Somatic Complaints

	Muscle Ache		Stomach Ache		Headache	
Predictor	B (SE)	Odds ratio (CI)	B (SE)	Odds ratio (CI)	B (SE)	Odds ratio (CI)
Bruxism Per Hour						
Constant	-1.75(0.72)	0.17	-1.54(0.69)	0.21	-.39(0.57)	0.68
Bruxism per hour	0.78*(0.34)	2.19(1.12, 4.27)	0.67*(0.32)	1.95(1.04, 3.62)	0.10(0.24)	1.11(0.70, 1.78)
Model Statistics[1]	R^2 =.29; $X^2(1) = 6.64$, $p = .01$		$R^2 = .23$; $X^2(1) = 5.37$, $p = .02$		$R^2 = .01$; $X^2(1) = .21$, $p = .65$	

Note: * $p < .05$. [1]R^2 calculated using Nagelkerke criteria

Table 4 - Descriptive Statistics for Sleep Variables based on SB Status

	No SB (n = 21)	SB (n = 10)	*U*	*p* value	Effect size *R*
Total Sleep Minutes (M/SD)	496.4(61.0)	499.4(63.5)	98.0	.78	.05
Sleep Onset Latency Minutes (M/SD)	44.2(31.6)	38.1(27.9)	95.0	.69	.10
Sleep Efficiency (%; M/SD)	87.5(6.4)	84.1(6.5)	72.5	.17	.25
N1 minutes (M/SD)	7.4(6.3)	8.4(3.5)	74.0	.20	.10
N2 Minutes (M/SD)	242.0(32.0)	244.9(23.6)	91.0	.55	.11
N3 Minutes (M/SD)	120.4(25.4)	113.8(14.5)	89.5	.53	.16
REM Sleep Minutes (M/SD)	107.7(24.4)	106.3(23.6)	103.5	.95	.01
%N1 (M/SD)	1.6(1.4)	1.8(7.6)	75.0	.21	.02
%N2 (M/SD)	50.8(5.9)	51.7(3.0)	91.0	.57	.10
%N3 (M/SD)	25.3(5.1)	24.1(3.5)	89.5	.53	.14
%REM (M/SD)	22.4(3.8)	22.4(2.6)	100.5	.86	.00
WASO (M/SD)	26.3(22.4)	52.5(29.2)	44.0	.01	.46
Arousal Index (M/SD)	8.2(2.9)	9.6(1.8)	65.5	.10	.28

Note: N1= Non-REM stage 1 sleep; N2 = Non-REM stage 2 sleep; N3 = NREM stage 3 sleep; REM = Rapid Eye Movement sleep; WASO = Wake After Sleep Onset