Published in final edited form as:
PM R. 2013 July ; 5(7): 554–562. doi:10.1016/j.pmrj.2012.11.006.

Association of Daytime Somnolence With Executive Functioning in the First 6 Months After Adolescent Traumatic Brain Injury

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Abstract

Objective—To determine the relationship between severity of injury and self-reports and parent reports of daytime somnolence in adolescents after traumatic brain injury (TBI), and to determine the relationship between daytime somnolence and self-report and parent report of executive functioning in daily life.

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M.B.O. Disclosure: nothing to disclose
D.B. Disclosure: nothing to disclose

Presented in part, at the 2011 American Academy of Physical Medicine and Rehabilitation Meeting.

Design—Cross-sectional study conducted within the first 6 months (mean ± standard deviation 14.97 ± 7.51 weeks) after injury. Partial correlation controlling for injury severity was used to examine the associations of TBI severity with daytime somnolence and the association of daytime somnolence with executive functioning.

Setting—Outpatient visits at 3 children's hospitals and 2 general hospitals with pediatric trauma commitment.

Participants—A total of 102 adolescents, 12–18 years old, who sustained moderate-to-severe TBI (n = 60) or complicated mild TBI (n = 42).

Main Outcome Measurements—Parent-report Sleepiness Scale, Epworth Sleepiness Scale (youth report), Behavior Rating Inventory of Executive Function (BRIEF) (self-report and maternal report).

Results—Adolescents who sustained moderate-to-severe TBI had increased daytime somnolence compared with those with complicated mild injuries in the parent report but not in the youth report. Based on the parent report, 51% of adolescents with moderate-to-severe TBI showed significant daytime somnolence compared with 22% of those with complicated mild TBI. The parent report of daytime somnolence was associated with executive dysfunction on both the BRIEF self-report and parent report; however, the youth report of daytime somnolence was associated only with the BRIEF self-report.

Conclusions—The parent report of daytime somnolence correlated with TBI severity and predicted executive functioning difficulties of the teens in everyday circumstances. Although a correlation between daytime somnolence and executive dysfunction were also apparent on self-report, this did not differ based on injury severity. Teens tended to report fewer difficulties with executive function, which suggests that the teens have decreased awareness of their impairments.

INTRODUCTION

Traumatic brain injury (TBI) is a major cause of acquired disability in children [1], which results in physical impairments and difficulty with cognition, mood, behavior, social development, and school performance [2–9]. Many children also experience changes in their sleep patterns after TBI that could contribute to irritability, disrupted sleep, and daytime somnolence [10–12]. Parents report increased daytime somnolence and prolonged nocturnal sleep in grade-school children for months to years after severe TBI compared with mild TBI and controls [12]. In addition, Kaufman et al [13] documented insomnia and increased awakenings among children with mild TBI compared with age-matched healthy controls.

Adolescence constitutes a unique stage in the process of maturation into adult sleep patterns and cognitive and behavioral functioning. Sleep maturation is influenced by hormones of puberty, sleep hormones, circadian rhythms, and social and behavioral contexts [14–16]. Compared with both younger children and adults, adolescents have delayed sleep onset and tend toward later bedtimes, which results in shorter sleep duration during school nights [14–16]. The circadian system shifts, which affects the release of nocturnal hormones such as melatonin and alterations of body temperature regulation to promote sleep and wakefulness. In addition, adolescents experience a decrease in the duration of rapid eye movement and

non–rapid eye movement (particularly N3 or “slow wave”) sleep. Each of these changes may contribute to daytime somnolence in typically developing adolescents [15,16] and could plausibly either magnify or wash out the effects of TBI on daytime arousal in adolescents with brain injury. Consequently, although TBI appears to increase the risk for daytime somnolence in young children and adults, the unique developmental changes associated with adolescence warrant caution in applying previous findings to adolescents.

Concurrent with these changes in sleep patterns and day-time somnolence, adolescents experience continued maturation of higher-level neuropsychological processes, including “executive functions,” such as planning, problem solving, and regulation of emotions and behaviors [17]. The protracted period of development of these skills is believed to contribute to the developmental spike in risky behaviors during adolescence [5,18]. Executive functions also appear to be particularly sensitive to inadequate sleep, which leads some researchers to speculate that the chronic sleep restriction experienced by many adolescents contributes to significant functional deficits, with long-term consequences [5,19]. Indeed, daytime somnolence induced by sleep deprivation has been causally linked to changes in cognition, behavior, and mood in adolescents [14,19–23].

Given that TBI is known to cause significant problems with executive function, working memory, attention, and mood, it is important to better understand the association of daytime somnolence with these functions as well. A few studies have shown worse performance on tasks of sustained attention and memory in adults with TBI and sleep disorders [24–26]. However, to our knowledge, no studies to date have examined the associations between sleep dysfunction and executive function in adolescents who are recovering from TBI. The first aim of this study was to determine the relationship between TBI severity and daytime somnolence in adolescents. The second aim was to determine the relationship of daytime somnolence to executive dysfunction in adolescents after TBI. We hypothesized that (a) individuals with more severe TBI would have more daytime somnolence, and (b) daytime somnolence would be associated with greater executive dysfunction regardless of the severity of the injury.

METHODS

Current analyses were based on data collected in the context of a larger study of a Web-based, family problem-solving treatment program for adolescents with TBI [27]. From 2007 to 2011, trauma registries from 5 participating centers across the United States (3 pediatric hospitals and 2 general hospitals with pediatric trauma commitment) were screened for patients between the ages of 12 and 18 years who were admitted to the hospital, at least overnight, with either a primary or secondary diagnosis of TBI. TBI was defined as an alteration in consciousness secondary to an external mechanical force. Patients were excluded if the etiology of their TBI was a penetrating injury such as a gunshot wound, anoxia, or inflicted trauma by parents or caregivers. Patients were also excluded if their TBI occurred more than 6 months before enrollment in the study. TBI severity was defined based on the Glasgow Coma Scale (GCS), with complicated mild TBI defined as a GCS score of 13–15 with abnormal imaging on computed tomography or magnetic resonance imaging, moderate TBI as GCS 9–12, and severe TBI as GCS ≤8.

As part of the baseline assessment conducted within the initial 6 months after injury, adolescents and parents completed questionnaires regarding the youths' sleep habits. Ninety-three percent of the parent respondents were mothers, and we chose to focus on maternal report in the analyses to eliminate any potential bias between caregivers. The youth questionnaire included the Epworth Sleepiness Scale (ESS) [28], an 8-item measure with questions that ask the participant to rate how likely he or she is to fall asleep in certain situations. A score of 10 or more on the ESS is indicative of excessive daytime somnolence [28,29]. Parents completed the 5-item Parent-Report Sleepiness Scale (PSS) [23], which also examines severity of drowsiness. Beebe et al [23] found that PSS scores of 5 or more were present in 11% of healthy adolescents when well rested, but in 74% of the same teens after 5 nights of sleep restriction [23]. Reflecting the general paucity of research on somnolence after pediatric TBI, neither the ESS nor the PSS has been previously used with this population. However, the ESS has established psychometric support in prior studies of both healthy adolescents and those with sleep-disordered breathing [30–32], and has been shown to correlate with both parent report and objective measures of executive functioning [30]. In healthy adolescents, the PSS has also shown good reliability, sensitivity to experimental sleep restriction, and convergent validity with an established sleep questionnaire [23]. In the current sample, the internal consistency of both was modest but acceptable (ESS, $\alpha = 0.68$; PSS, $\alpha = 0.61$).

Behavioral manifestations of executive dysfunction were assessed by using the Behavior Rating Inventory of Executive Function (BRIEF) [33,34], completed independently by each adolescent and mother at the time of the baseline assessment. For each reporter, we analyzed the Behavioral Regulation Index (BRI), which is a composite measure of the adolescent's ability to regulate behavior and emotions, and to flexibly adapt to changing demands; and (b) the Metacognition Index (MI), a composite measure of the ability to self-initiate tasks, see tasks through to completion, plan, organize, and self-monitor one's work and behaviors. The BRIEF indexes have strong evidence for reliability and validity in both parent- and self-report formats across a range of adolescent populations, including those with neurologic conditions [33,34]. The BRIEF has demonstrated good internal consistency, inter-rater reliability, and test–retest reliability, and has been validated in pediatric TBI [33,35–37], although some researchers have questioned the insight of adolescents into their own functioning after TBI [18]. Age- and gender-normed Global Executive Composite scores (combining the BRI and the MI) ≥65 are considered suggestive of a clinical concern, with 5%–11% of healthy adolescents exceeding this threshold [33,34]. Descriptive information about the injury and participants was gleaned from a chart review (lowest postinjury GCS, age at injury) and parent report (demographics). Adolescents were also administered the 2-subtest version of the Wechsler Abbreviated Scales of Intelligence [38] during the baseline assessment to obtain an estimate of overall intelligence quotient (IQ).

The participants with moderate and severe TBI were pooled into a single moderate-severe TBI group (GCS < 13) both to maximize statistical power (there were only 14 eligible participants with moderate TBI) and because preliminary analyses indicated that daytime somnolence levels were very similar in participants with moderate or severe TBI. Specifically, on self-reported daytime somnolence, the mean ± standard deviation (SD) score for adolescents with moderate TBI was 5.79 ± 3.49, whereas adolescents with severe

TBI had a mean ± SD score of 7.07 ± 3.47 (P = .23). On parent report of somnolence, adolescents with moderate TBI had a mean ± SD score of 5.29 ± 2.05, and adolescents with severe TBI had a mean ± SD score of 4.70 ± 2.42 (P = .42).

Preliminary analyses indicated minimal skew and no clear outliers on the daytime somnolence and BRIEF indexes. Preliminary analyses also investigated the potential utility of the following covariates: age at injury, time since injury, and overall IQ. None of these significantly differed across groups (Table 1). In addition, no covariate was significantly associated with either parent report or self-report of sleepiness, so none was considered an appropriate covariate for group comparisons on daytime somnolence and executive functioning. Independent-sample t tests addressed the directional (1-tailed) hypothesis that the moderate-severe TBI group would show greater somnolence and higher (worse) scores on the BRIEF BRI and MI. To provide a complementary, clinically relevant view of the same data, the percentage of adolescents falling in clinical ranges on each measure was calculated and compared across groups by using the Fisher exact test. Partial correlation was used to examine the association between reported daytime somnolence (ESS and PSS) and executive function (BRIEF). Unadjusted analysis was initially performed, followed by adjusted analysis when controlling for injury severity.

We also performed exploratory analyses to determine whether the relationships among TBI severity, daytime somnolence, and cognitive functioning are affected by mood, substance use, and medications, because these factors have been shown in previous studies to affect sleep and executive function in healthy adolescents [39–45]. To assess mood and substance use, we used the Child and Adolescent Functional Assessment Scale [46,47], a structured clinical interview of caregivers and/or parents that focuses on adolescent behavioral functioning across multiple settings. It has been used widely to assess clinical outcomes in children with serious emotional disturbances [46,47] and has excellent inter-rater reliability, with correlation coefficients that range from 0.74 to 0.99 [47]. The Child and Adolescent Functional Assessment Scale generates a total score as well as clinical ratings in 8 subscales: school, home, community, behavior toward others, moods and/or emotions, self-harmful behaviors, substance abuse, and thinking [46,47]. The moods and/or emotions and substance abuse subscales were entered into the regression models as covariates to determine the effect of their inclusion on the hypothesized relationships. Medications that the adolescents were taking were provided by the parents. Medications were considered to have the potential to affect sleep based on the mechanism of action, adverse effect profiles, and our clinical experience. Because there was no way to quantify the impact of individual medications on sleep or executive function, the above analyses were repeated after removing all participants who were taking any medications with the potential to affect sleep to preclude the possibility of medication use as a confounding factor.

RESULTS

Study Sample Characteristics

Of the 211 adolescents identified for recruitment who met inclusion criteria, 70 declined to participate, 9 could not be reached, and 30 did not complete either the teen or the maternal report of sleepiness (because it was added to the study battery after recruitment was

underway), which left 102 adolescents for analyses. Forty-two sustained complicated mild TBI, and 60 met criteria for moderate-severe TBI. Age, gender, race/ethnicity, time since injury, IQ, and cause of injury did not differ as a function of injury severity (Tables 1 and 2).

Executive Function and Sleep Rating Characteristics

As shown in Table 3, parents reported that adolescents who had sustained moderate-severe TBI averaged worse in behavior regulation ($P = .035$) and had higher rates of impaired behavior regulation ($P = .019$) than those with complicated mild TBI, with similar but less robust statistical trends reported with respect to metacognitive impairment ($P = .082$ and $P = .060$, respectively). According to self-report, however, there were no significant differences based on TBI severity in average scores or rates of impairment on either behavior regulation or metacognition scales. In addition, adolescents reported less difficulty with executive function than did their parents in the sample as a whole. In all, 35% of parents reported that their teens with brain injuries had clinically impaired behavior regulation, but only 17% of the teens themselves reported such impairment. Similarly, the rate of impaired metacognitive functioning reported by parents was 32%, whereas that reported by the adolescents was 18%.

Parent reports indicated that adolescents who sustained moderate-severe TBI were, on average, more somnolent than those with complicated mild TBI ($P = .006$) (Table 3). Parents of adolescents with moderate-severe TBI were more than twice as likely to report clinically significant daytime somnolence than parents of those with complicated mild TBI ($P = .005$) based on an established cutoff on the PSS. The overall rate of parent-reported excessive daytime somnolence across TBI groups was 40%, considerably higher than the 12% seen in well-rested adolescents [23]. In contrast, roughly 20% of the total sample exceeded thresholds on the self-report ESS, and the TBI severity groups did not significantly differ in mean scores or in the portion who reported clinically significant somnolence ($P > .10$).

Relationship Between Daytime Somnolence and Executive Function

As shown in Table 4, parent report of daytime somnolence was significantly associated with executive dysfunction in both behavior regulation and metacognition, as reported by both the parent and the teen. Adolescent self-report of daytime somnolence was similarly associated with executive dysfunction as measured via self-report but not as measured via parent report. Correlations were not markedly changed by controlling for injury severity, as indexed either by the TBI-severity group or the GCS as a continuous variable.

Exploratory Analyses, Including Mood, Substance Use, and Medications

Of note, 6% of the total sample reported substance use and 59% reported problems with mood. The associations between daytime somnolence and executive function were repeated after controlling for severity of injury, substance use, and mood. The adjusted partial correlations were virtually unchanged, and *P* values remained significant (data not shown). Finally, we considered whether the associations between daytime somnolence and executive function ratings were due to a subset of participants who were taking medications that might affect sleep. Eight adolescents were taking a medication that promotes sleep, which included

melatonin, trazodone, quetiapine, and tramadol. Conversely, 7 adolescents were taking amphetamines at the time of data collection, 4 of whom were taking the medication before injury, presumably for preinjury attention disorders. Three adolescents were taking amantadine. Antiepileptics, such as levetiracetam, phenytoin, oxcarbazepine, and topiramate, were being used after injury in 6 adolescents. Five adolescents were taking antidepressants, and 8 adolescents were taking narcotics for pain. Although such medication use could affect (or be a reaction to) postinjury daytime somnolence, all of the above findings were unchanged after removing from all of the analyses the 24 participants on these medications (data not shown), aside from the predictable effects of reducing the sample size on statistical power.

DISCUSSION

Findings in this study provide partial support for our first hypothesis that greater severity of TBI would be associated with increased daytime somnolence. Parent, but not adolescent, report of daytime somnolence was higher in the moderate-severe TBI group compared with the complicated mild TBI group. At first glance, this might be interpreted as inconsistent evidence of clinically significant daytime somnolence after adolescent TBI. However, an alternative explanation is that adolescents display less insight into their difficulties after TBI than do their parents [18]. We acknowledge that the length of time since injury can potentially bias our results when presuming that the complicated mild TBI group would recover faster and, therefore, display fewer symptoms compared with the adolescents with moderate or severe TBI. However, it is worth noting that the time since injury was unrelated to either parent- or teen-reported sleepiness in preliminary analyses. Regardless of injury severity, adolescents with TBI failed to report the executive functioning difficulties on formal questionnaires that might be expected in light of their recent history of TBI. In contrast, parents reported worse executive functioning than published norms in both groups, with the moderate-severe TBI group faring worse. Others have found that adolescents who are the most severely injured tend to show the greatest discrepancy from their parents in their report of at least some aspects of executive functioning, which suggests that the more severe the TBI, the poorer the adolescent's ability to recognize executive functioning impairments [18]. Analysis of the present findings suggest that this may extend to teens' self-report of daytime somnolence as well.

The presence of parent-reported daytime somnolence after moderate-to-severe TBI is consistent with prior work that shows that both younger children [12] and adults [48] who have sustained moderate or severe TBI have greater daytime somnolence compared with those who have mild TBI or healthy controls. However, it is acknowledged that the pediatric literature remains limited and that the adult findings have at times been mixed, perhaps related in part to the same concerns about self-report after TBI that were evident here [49–52]. Excessive sleepiness occurred in a sizable proportion of our sample, with 40% of adolescents per parent report and 20% per self-report reaching levels of clinically significant daytime somnolence seen in only 12% of healthy, well-rested adolescents [23].

Our finding that parents reported worse executive functioning in adolescents who sustained more severe TBI is not surprising given the well-established sensitivity of executive

functioning to TBI [5,18,19]. New in the current findings is the demonstration of an association between daytime somnolence and executive dysfunction. This was particularly evident for parent-reported daytime somnolence, which significantly predicted executive dysfunction as reported independently by the parent and adolescent. This cross-informant correlation (parent-reported daytime somnolence with adolescent-reported executive functioning) is noteworthy, because it argues against the presence of a simple "reporter effect," in which artificial associations might be generated by gathering data from a single informant. Also, the associations between parent-reported daytime somnolence and executive functioning were present even after controlling for TBI severity, which lends credence to the idea that this was not a confounding influence on results.

The overall findings are in agreement with theoretical and research accounts of the relationship between sleep disturbances and executive dysfunction in both adults and children [34,53–55]. Pediatric daytime somnolence related to sleep deprivation or disruption has been causally linked to a number of prominent functional deficits, including poor school performance and motor vehicle crashes in adolescents [54] as well as mood disruption [39,56]. Although the current study is correlational and cannot establish causation, it seems very likely that the high rate of excessive daytime somnolence observed in our adolescents after TBI is clinically significant and worthy of routine assessment and intervention. Procedural overviews of pediatric sleep assessments have been provided elsewhere [57–59]. A key goal of such an assessment is to determine the mechanism by which a given adolescent with TBI might be experiencing excessive daytime somnolence, which might include nocturnal behavioral disturbance or anxiety that results in insomnia, the disruptive effects of mechanical or neuropathic pain on sleep, obstructive sleep apnea, medication effects, inflammatory processes, or a direct injury to the broadly distributed circuits that regulate sleep and arousal. Determining the underlying mechanism can lead to disorder-specific treatment, such as the use of continuous positive airway pressure for obstructive sleep apnea, medications such as modafinil for posttraumatic hypersomnia, and medications such as trazodone and melatonin for insomnia [49,60–62]. Improving sleep would, it is hoped, lead to better overall outcomes. Makley et al [63] suggested that individuals with TBI who do not experience sleep disturbances are more likely to have shorter hospital stays and less functional impairment.

Limitations

Unfortunately, preinjury assessment of daytime somnolence was not collected and is a limitation of this study. Assessing somnolence before injury would have permitted us to determine the extent to which group differences reflected postinjury increases in sleepiness in the moderate- to-severe TBI group. An additional limitation is that daytime somnolence measures were subjective, relying on the adolescent and the parent to accurately report sleepiness rather than fatigue. Although the ESS has been shown to be significantly correlated with the Multiple Sleep Latency Test [64], having an objective measure of somnolence such as the Multiple Sleep Latency Test would lend more credence to the findings in this study. Also, we did not assess the potential mechanisms, such as pain and sleep-disordered breathing, which might underlie daytime somnolence after adolescent TBI. In the absence of a matched control group of adolescents without TBI (eg, healthy

adolescents, those who experienced orthopedic injuries but not TBI), we were unable to determine the extent to which daytime somnolence and the relationship with executive functioning are specifically associated with the TBI. Future studies should include such a control group and investigators would be well advised to examine more nuanced measures of TBI severity, such as the duration of posttraumatic amnesia or changes in Coma Recovery Scale Revised scores, because GCS is an important but incomplete severity index. Because our study found increased levels of clinically significant daytime somnolence across GCS groups compared with population norms, additional studies could include those individuals with computed tomography–negative mild TBI to determine whether those with abnormal neuroimaging are at increased risk for excessive daytime somnolence and executive dysfunction, regardless of injury severity. Finally, because our functional outcome assessments were limited to questionnaire-based assessment of executive functioning, it would be helpful to examine the relationships between daytime somnolence and other outcome measures after TBI, including scholastic performance, psychiatric functioning, and performance on cognitive tests.

Despite these limitations, to our knowledge this is the first study to demonstrate relationships among TBI severity, daytime somnolence, and reports of executive functioning in adolescents with subacute TBI. Due to the sparse literature in this area, there is a significant need for future work to build upon the current findings to clarify the prevalence of, mechanisms behind, consequences of, and interventions for, sleep problems in adolescents after TBI.

CONCLUSION

Adolescents with TBI have higher rates of excessive daytime somnolence compared with population norms, with higher rates noted in those with moderate-to-severe TBI. Parent report of daytime somnolence correlated with TBI severity and predicted executive functioning difficulties of the teens in everyday circumstances. Consistent with others' findings that adolescents may lack insight into their functioning after TBI, teen report of both executive functioning and daytime somnolence did not vary by injury severity. Given that excessive somnolence appears to be common among adolescents after TBI and has been causally linked to a number of functional deficits, this symptom merits further clinical attention in this population, as well as further characterization of the association between daytime somnolence and both cognitive and behavioral recovery after pediatric TBI.

Acknowledgments

We thank Dr Linda Michaud, MD, and Dr Narong Simakajornboon, MD, for their dedication and valuable input regarding the content of this manuscript.

B.G.K. Disclosure related to this publication: grant, NIH (direct funds received and money to institution)

H.G.T. Disclosure related to this publication: grant, NIH (money to institution)

T.M.B. Disclosure related to this publication: grant, NIH (money to institution)

M.W.K. Disclosure related to this publication: grant, Colorado TBI Trust Fund, NIH grant R01-MH073764 from the National Institute of Mental Health

S.L.W. Disclosure related to this publication: grant, NIMH

Disclosures outside this publication: consultancy, Western Oregon University; grants/grants pending, direct funds received, NICHD and NIDRR; payment for lectures including service on speakers bureaus, University of San Diego, ACRM, AAPM&R

Research support: Supported in part by National Institutes of Health grant R01-MH073764 from the National Institute of Mental Health and by a grant from the Colorado Traumatic Brain Injury Trust Fund Research Program, Colorado Department of Human Services, Division of Vocational Rehabilitation, Traumatic Brain Injury Program.

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Table 1

Sample characteristics

	Complicated Mild TBI (n = 42)	Moderate-Severe TBI (n = 60)	*P* Value
Age at injury, mean ± SD, y	14.67 ± 1.74	14.76 ± 1.87	.80
Boys, %	66.7	63.3	.73
Race/ethnicity, %			.41
White	73.8	83.3	
Black	19.0	10.0	
Other	7.1	6.7	
Lowest GCS, mean ± SD	14.43 ± 0.67	6.62 ± 3.10	<.001
Weeks since injury, mean ± SD	14.05 ± 8.02	15.62 ± 7.12	.30
WASI IQ, mean ± SD	98.46 ± 14.39	98.33 ± 12.64	.96

TBI = traumatic brain injury; SD = standard deviation; GCS = Glasgow Coma Scale; WASI IQ = Wechsler Abbreviated Scale of Intelligence overall intelligence quotient.

Table 2

Injury characteristics

Cause of Injury	Complicated Mild TBI (n = 40) No. (%)*	Moderate-Severe TBI (n = 56) No. (%)*
MVC	8 (20.0)	17 (30.4)
Pedestrian collision with vehicle	3 (7.5)	9 (16.1)
Bicycle collision with vehicle	2 (5.0)	3 (5.4)
Other bicycle accident	8 (20.0)	2 (3.6)
Recreational transport collision with vehicle†	1 (2.5)	1 (1.8)
Other recreational transport accident†	2 (5.0)	11 (19.6)
Fall	3 (7.5)	3 (5.4)
Sport or recreation	8 (20.0)	7 (12.5)
Assault	3 (7.5)	2 (3.6)
Injury on playground equipment	1 (2.5)	0 (0.0)
Rough housing	1 (2.5)	1 (1.8)

TBI = traumatic brain injury; MVC = motor vehicle collision.

*Cause of injury was not available for 6 patients.

†Recreational transport refers to transport with all-terrain vehicles, go-carts, motorbikes, and skateboards, and sledding, skiing, and horseback riding.

Table 3

Executive functioning and daytime somnolence across TBI groups*

	Complicated Mild TBI, mean (SD)	Moderate-Severe TBI, mean (SD)	*P* Value†	Complicated Mild TBI, % Impaired	Moderate-Severe TBI, % Impaired	*P* Value†
Parent report						
BRIEF BRI	56.4 ± 10.7	60.9 ± 12.1	.035	18.9	41.4	.019
BRIEF MI	59.1 ± 10.0	61.9 ± 9.1	.082	21.6	39.0	.060
Daytime somnolence	3.67 ± 1.88	4.85 ± 2.29	.006	22.2	50.8	.005
Adolescent report						
BRIEF BRI	53.1 ± 12.6	51.9 ± 11.4	.253	19.0	13.6	.317
BRIEF MI	53.3 ± 13.3	50.9 ± 10.7	.154	19.0	11.9	.294
Daytime somnolence	7.63 ± 4.21	6.76 ± 3.49	.131	24.4	17.2	.267

TBI = traumatic brain injury; SD = standard deviation; BRIEF = Behavior Rating Inventory of Executive Functioning; BRI = Behavioral Regulation Index; MI = Metacognition Index.

*Higher scores reflect greater impairment.

†*P* value refers to the directional (1-tailed) significance of cross-group comparisons based on independent-samples *t* test or Fisher exact test.

Table 4

Unadjusted and adjusted correlations between daytime somnolence and executive functioning

	Associations With Parent-Reported Somnolence			Associations With Self-Reported Somnolence		
		Adjusted Partial Correlation†			Adjusted Partial Correlation†	
BRIEF	Unadjusted Correlation (uncorrected)*	Covarying TBI Group	Covarying Lowest GCS	Unadjusted Correlation (uncorrected)*	Covarying TBI Group	Covarying Lowest GCS
Parent-report BRI	.38¶	.35¶	.38¶	−0.17	−0.14	−0.17
Parent-report MI	.28§	.25‡	.28§	−0.16	−0.14	−0.16
Self-report BRI	.32§	.35¶	.35¶	.20‡	.20‡	.20‡
Self-report MI	.21‡	.26‡	.25‡	.27§	.26§	.27§

BRIEF = Behavior Rating Inventory of Executive Functioning; TBI = traumatic brain injury; GCS = Glasgow Coma Scale; BRI = Behavioral Regulation Index; MI = Metacognitive Index.

*Unadjusted correlations represent simple Pearson *r* values.

Partial correlations are adjusted for TBI severity, as alternatively indexed by TBI group (complicated mild vs moderate-severe) or by lowest documented GCS score.

P values are based on exploratory (2-tailed) significance tests:

‡$P < .05$,

§$P \leq .01$,

¶$P \leq .001$.