Objective  Determine the effect of moderate and severe traumatic brain injuries (TBI) on the sleep of school-aged children.  Methods  A concurrent cohort-prospective design compared children aged 6–12 years who sustained moderate TBI (baseline \( n = 56 \)), severe TBI (\( n = 53 \)), or only orthopedic injuries (\( n = 80 \)). Retrospective parental report of pre-injury sleep was collected about 3 weeks post-injury. Post-injury assessments occurred prospectively a mean of 6, 12, and 48 months later.  Results  Growth curve analyses compared the groups over time. The moderate TBI group had worse pre-injury sleep than the other groups. The moderate TBI and orthopedic injury groups displayed a small decline in sleep problems from pre- to post-injury. Children with severe TBI displayed increased post-injury sleep problems.  Conclusions  Children who sustain severe TBI are at elevated risk for post-injury sleep problems. Because sleep problems may result in daytime impairments and family distress, additional clinical and research attention is warranted.

Key words  brain injury; children; longitudinal; orthopedic injury; pediatrics; sleep.

Moderate and severe traumatic brain injuries (TBI) have been known for some time to significantly impact daytime behavioral and adaptive functioning. In recent years, there also has been increasing appreciation of the nocturnal effects of TBI. Sleep problems in adults after a TBI can persist several years post-injury, and include nightmares and dyssomnias that can present as inadequate sleep or as excessive sleep propensity due to poor sleep quality (Bryant, Marosszeky, Crooks, & Gurka, 2000; Masel, Scheibel, Kimbark, & Kuna, 2001; Thaxton & Myers, 2002). As these authors have noted, the etiology of such sleep problems may include pain or discomfort, damage to neural circuits important in the sleep process, and emotional trauma. All of these potential contributors to sleep pathology can occur in childhood TBI, yet very little relevant research has been published. A few studies have identified vaguely defined sleep disturbances after childhood head trauma (Farmer, Singer, Melleits, Hall, & Charney, 1987; Hooper et al., 2004; Nakayama, Gardner, & Rogers, 1990; Pillar et al., 2003). However, sleep problems may follow a wide range of traumatic injuries, including orthopedic injuries (OI) (Kaufman et al., 2001). Moreover, no study has systematically examined sleep across more severe grades of pediatric TBI, despite neuropsychological and behavioral evidence of worse outcomes in the most severely injured children (Yeates et al., 2002, 2004). Finally, the time course of post-injury sleep problems is unknown, as group studies have rarely followed children for more than a few months post-injury.
The lack of research on pediatric sleep following TBI is noteworthy in light of evidence of an interrelationship between sleep pathology and the daytime functioning of children and their families. Children who have undergone experimental sleep restriction or have naturally occurring dysomnia have elevated rates of daytime behavioral disturbances (Blunden & Beebe, 2006). Sleep pathology in children also is a major source of parent and family stress (Mindell & Owens, 2003). Insofar as sleep pathology following pediatric TBI contributes to diminished motivation, caregiver distress, and cognitive and behavioral disturbances, it may pose a significant threat to rehabilitation efforts and functional recovery.

In this brief report, we analyze parent-reported sleep data that were collected in the course of a larger prospective study of the neuropsychological and family impact of moderate or severe pediatric TBI. Based upon the adult literature, we hypothesized that children who sustained a TBI, especially a severe TBI, would be reported to display more post-injury sleep problems than children who sustained only OIs. This study also examined the time course of post-injury sleep concerns up to several years post-injury, although we did not have specific hypotheses with regard to change over time.

Method

Prior publications detail the procedures for the larger project (Taylor et al., 2001; Yeates et al., 2004). Briefly, the project employed a concurrent cohort-prospective design in which school-age children with moderate TBI (mTBI), severe TBI (sTBI), or only OIs were recruited while hospitalized for their injuries and followed for several years. During the course of data collection, parents were asked to rate pre-injury child sleep behaviors as part of a baseline assessment completed an average of 3 weeks post-injury. Parent-report of sleep behaviors was then acquired prospectively 6 ± 1 (M ± SD) months, 12 ± 1 months, and 48 ± 11 months later to assess functioning over time.

The initial sample comprised 189 children recruited from four hospitals in Ohio, including 56 with mTBI, 53 with sTBI, and 80 with OI. Criteria for inclusion were: (a) at least one night’s hospitalization for OIs or for moderate or severe TBI due to closed head injury; (b) age at injury between 6 and 12 years; (c) no evidence of child abuse or previous neurological disorder; and (d) residence in an English-speaking household. Severe TBI was defined based on a lowest post-resuscitation Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974) of 8 or less. Moderate TBI was defined by a GCS score of 9–12, or a GCS score of 13–15 if accompanied by radiographic abnormality or persistent neurologic abnormality. Some children in the TBI groups had OIs, but the OI group was limited to children without symptoms suggestive of possible CNS insult (e.g., signs of concussion).

Characteristics of the initial samples and those seen at each follow-up point were detailed by Taylor, Yeates and colleagues (Taylor et al., 2001; Yeates et al., 2004) and will be only briefly reviewed here. At baseline, the groups did not significantly differ in age, gender, or ethnicity; the pooled sample was 67% boys and 68% Caucasian, with an average age at injury of 9.5 ± 2.0 years. The OI group was on average of lower socioeconomic status (SES) than the two TBI groups, which were equivalent. Duration of hospitalization and severity of injuries not involving the head were equivalent across the sTBI and OI groups, but both groups had more severe injuries than the mTBI group. Subject attrition ranged from 14–28% across follow-up evaluations. Attrition was highest in the OI group and among those of lower SES. Attrition was not significantly related to age at injury, sex, or ethnicity.

Following Gregory and O’Connor (2002), our assessment of sleep was comprised of seven items derived from the Child Behavior Checklist (CBCL; (Achenbach, 1991): overtired, trouble sleeping, sleeps more than most children (day or night), sleeps less than most children, has nightmares, talks or walks in sleep, and wets the bed, each rated as “not true” (scored 0), “somewhat or sometimes true” (1), or “very true or often true” (2). The summed composite of these items was our outcome variable; its stability across post-injury time points (r = .47–.56, median = .56) was similar to that of the traditional eight CBCL subscales across the same assessments (r = .50–.79, median = .59).

Results and Discussion

Table 1 provides descriptive information on the timing of assessments and sleep symptoms across groups. Rather than reflecting three tightly timed assessment points, the follow-ups occurred at variable times post-injury (e.g., the long-term follow-up occurred 2.4–5.8 years post-injury). This argues for treating time since injury in a continuous fashion. Growth curve analysis (GCA) estimates parameters based upon all available data, allowing for occasional missing data and for flexible modeling of time (Singer & Willett, 2003). A GCA was conducted...
using SAS version 9.1 (Proc GENMOD) via a mixed model based upon the Poisson distribution to control for biased estimates due to significant skew. The occurrence of a discrete injury event warranted consideration of both discontinuous (injury-related) and continuous (time since injury) effects. Following the suggestion of Singer and Willett (2003), both were entered concurrently into the statistical model. The effect of the injury event was modeled by contrasting the baseline data with all post-injury points, while time since injury was modeled in 3-month increments. Analyses modeled the following predictors: the main effects of group (OI, mTBI, sTBI), the injury event (pre- vs. post-injury; PPI), and time since injury, as well as the interactions of group-by-PPI (i.e., differential effects of the injury event across groups) and group-by-time (i.e., differential recovery pattern across groups). Only the linear effect of time since injury was modeled as quadratic effects were found not to be significant in a random effects omnibus test. Preliminary analyses indicated that gender, age at injury, and ethnicity were not significant covariates of sleep over time. Although SES was a significant covariate of sleep over time, exploratory entry of SES into the models did not substantially alter the findings reported below. Similarly, excluding subjects who were taking antiepileptic medication at one or more follow-up point (sTBI n = 7, mTBI n = 3, OI n = 1) did not alter these findings.

The overall model was significant, \( \chi^2 (8) = 31.75, p = .0001 \). As shown in Fig. 1a, a significant cross-group effect was noted in baseline (intercept) scores, using SAS version 9.1 (Proc GENMOD) via a mixed model based upon the Poisson distribution to control for biased estimates due to significant skew. The occurrence of a discrete injury event warranted consideration of both discontinuous (injury-related) and continuous (time since injury) effects. Following the suggestion of Singer and Willett (2003), both were entered concurrently into the statistical model. The effect of the injury event was modeled by contrasting the baseline data with all post-injury points, while time since injury was modeled in 3-month increments. Analyses modeled the following predictors: the main effects of group (OI, mTBI, sTBI), the injury event (pre- vs. post-injury; PPI), and time since injury, as well as the interactions of group-by-PPI (i.e., differential effects of the injury event across groups) and group-by-time (i.e., differential recovery pattern across groups). Only the linear effect of time since injury was modeled as quadratic effects were found not to be significant in a random effects omnibus test. Preliminary analyses indicated that gender, age at injury, and ethnicity were not significant covariates of sleep over time. Although SES was a significant covariate of sleep over time, exploratory entry of SES into the models did not substantially alter the findings reported below. Similarly, excluding subjects who were taking antiepileptic medication at one or more follow-up point (sTBI n = 7, mTBI n = 3, OI n = 1) did not alter these findings.

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Follow-up contrasts indicated that, although the sTBI and OI groups did not differ at the pre-injury baseline, $\chi^2 (1) = .01, p = .94$, the mTBI group scored higher at baseline than the OI group, $\chi^2 (1) = 5.23, p = .02$, and the sTBI group, $\chi^2 (1) = 4.33, p = .04$. This finding of elevated baseline sleep concerns in the mTBI group was unexpected, but may reflect the well-documented tendency for children who sustain TBI to have displayed a variety of problem behaviors prior to their injuries (Yeates, 2000).

Importantly, this baseline difference in sleep symptoms does not address whether the groups differed in how the injury affected sleep. Such a difference was reflected in a significant PPI-by-group interaction, $\chi^2 (2) = 8.34, p = .016$. To see this effect more clearly, Fig. 1b artificially holds the baseline scores constant. Contrast analyses indicated that the occurrence of moderate TBI and OI had roughly comparable effects on sleep, $\chi^2 (1) = 0.0, p = .99$, but both injury types differed from severe TBI, $\chi^2 (1) = 6.74 - 7.92, p < .01$. Whereas children in both the mTBI and OI groups were reported to show slightly fewer sleep problems immediately after their injuries than pre-injury, those who had sustained severe TBI were reported to display a worsening of their sleep following their injuries. The sTBI group then had some improvement in their sleep over time, but remained discrepant from the OI group. There was no significant group-by-time interaction, $\chi^2 (2) = 1.51, p = .47$, so some of the gains may reflect normative declines in sleep complaints during middle childhood (Mindell & Owens, 2003).

The small mean raw scores evidenced in Fig. 1 raise questions about whether only a few outliers were driving these results. To explore this, we conducted a logistic mixed model, classifying subjects as having a “sleep problem” at a time point if they received a sleep composite of three or more. Although this cutoff was somewhat arbitrary, it yielded follow-up prevalence estimates of 6–9% for sleep problems in the OI group, similar to the rate reported in the general population (Mindell & Owens, 2003). Results mirrored those from our primary analysis: (a) the mTBI group had higher rates of baseline sleep problems than the OI and sTBI groups, (b) both the mTBI and OI groups displayed a slight decline in the prevalence of sleep problems from pre- to post-injury, (c) the sTBI group displayed an injury-associated increase in sleep problems, with the prevalence of sleep problems nearly doubling from 16% at baseline to 31% post-injury, and (d) there was no significant group-by-time interaction.

These findings imply that severe pediatric TBI markedly increases the risk for sleep problems in a manner that is not evident after moderate TBI or OIs. The use of an orthopedically injured comparison group allowed us to exclude a number of potential confounds, including maturation, pain, and discomfort. However, we can not definitively rule out the impact of emotional trauma (Levi, Drotar, Yeates, & Taylor, 1999). Direct neurological injury may also be at play. TBI impacts the broad sleep-arousal system that extends from the brainstem through the diencephalon (Pace-Schott & Hobson, 2002). That system may continue to be disrupted well beyond resolution of coma in some children after severe TBI. Daytime sleepiness and nocturnal sleep duration were particularly increased post-TBI (Table I), consistent with clinical observations of children in the weeks to months post-injury. Importantly, many children who sustained TBIs were reported to continue to show both symptoms several years later.

Present findings are tempered by several design limitations. Subjects who dropped out tended to be of lower SES. Although covarying for SES did not substantially alter our results, it is not certain how well findings generalize to children from lower SES backgrounds. Also, the research design was quasi-experimental and relied on retrospective ratings of pre-injury sleep, which places limits on the confidence with which causal inferences can be drawn. However, short of random assignment to injury type or a huge cohort study with initial data collection prior to injury, the current design was perhaps the strongest possible. Additionally, the sleep measure used here covered only limited aspects of sleep functioning and has received scant psychometric attention, with validation work particularly needed. This reflects a general limitation of the field; as of the time of this study no broad band parent-report sleep questionnaire had been validated across a wide range of pediatric sleep problems. Finally, it will be important for future research to move beyond parent-report measures (as used here) to integrate objective sleep assessments.

Although limitations in our outcome measure suggest that these findings should be considered preliminary, they nevertheless represent an important addition to the research literature. This study had design strengths (e.g., large sample, multi year longitudinal design with intermediate data collection points, inclusion of an orthopedic-injury comparison group) that allowed for unprecedented examination of the short- and long-term effect of moderate and severe TBI on sleep functioning.
in children. The current findings direct clinicians to watch for emerging sleep problems after severe pediatric TBI, and argue for the development and implementation of sleep interventions for brain-injured children. Sleep-related clinical guidelines have been developed for adults following brain injuries (e.g., Thaxton & Myers, 2002), but we are not aware of any such guides for children. Similarly, although behavioral sleep interventions have been developed for children with other medical and developmental conditions (e.g., Wiggs & France, 2000), the efficacy of such interventions for brain-injured children is unknown. We hope that present findings spur increased clinical and research attention to pediatric sleep, a domain of behavioral functioning that is clearly at risk following brain injury but has received little attention to date.

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