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Neuropsychological Performance of Youth with Secondary Attention-Deficit/Hyperactivity Disorder 6- and 12-Months after Traumatic Brain Injury

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Abstract

The present study compared executive dysfunction among children with attention-deficit/hyperactivity disorder (ADHD) after traumatic brain injury (TBI), also called secondary ADHD (S-ADHD), pre-injury ADHD and children with TBI only (i.e., no ADHD). Youth aged 6–16 years admitted for TBI to five trauma centers were enrolled ($n = 177$) and evaluated with a semi-structured psychiatric interview scheduled on three occasions (within 2 weeks of TBI, i.e., baseline assessment for pre-injury status; 6-months and 12-months post-TBI). This permitted the determination of 6- and 12-month post-injury classifications of membership in three mutually exclusive groups (S-ADHD; pre-injury ADHD; TBI-only). Several executive control measures were administered. Unremitted S-ADHD was present in 17/141 (12%) children at the 6-month assessment, and in 14/125 (11%) children at 12-months post-injury. The study found that children with S-ADHD exhibited deficient working memory, attention, and psychomotor speed as

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compared to children with pre-injury ADHD. Furthermore, the children with S-ADHD and the children with TBI-only were impaired compared to the children with pre-injury ADHD with regard to planning. No group differences related to response inhibition emerged. Age, but not injury severity, gender, or adaptive functioning was related to executive function outcome. Neuropsychological sequelae distinguish among children who develop S-ADHD following TBI and those with TBI only. Moreover, there appears to be a different pattern of executive control performance in those who develop S-ADHD than in children with pre-injury ADHD suggesting that differences exist in the underlying neural mechanisms that define each disorder, underscoring the need to identify targeted treatment interventions.

Keywords

Traumatic brain injury; Children; Adolescents; S-ADHD; P-ADHD; Neurocognitive function

INTRODUCTION

Traumatic brain injury (TBI) is a worldwide epidemic and major public health concern, and is the most frequent cause of death and acquired disability among children and adolescents (Hyder, Wunderlich, Puvanachandra, Gururaj, & Kobusingye, 2007). The confluence of the following three factors facilitate our goal of a neurocognitive-based investigation of different neural characteristics between children who have pre-injury attention deficit/hyperactivity disorder (P-ADHD), and those with injury induced ADHD: (1) Children with TBI frequently demonstrate notable deficits that involve executive control abilities (Fenwick, & Anderson, 1999; Horton, Soper, & Reynolds, 2010; Lajiness-O'Neill, Erdodi, & Bigler 2010; Levin et al., 1997); (2) The development of psychiatric disorders in children and adolescents after TBI has been reported (Bloom et al., 2001; Max et al., 1997). ADHD is among the most common psychiatric disorders in children post-injury (occurs in 15–20% and is referred to as secondary ADHD [S-ADHD]) (Gerring et al., 1998; Max et al., 1998, 2004); and (3) There are data suggesting that children with ADHD are overrepresented in pediatric TBI cohorts (Gerring et al., 1998).

Several factors have been found to be associated with the development of S-ADHD, including pre-injury behavioral difficulties, post-injury intellectual and adaptive dysfunction, and psychosocial adversity, socioeconomic disadvantage, and injury severity (Max et al., 2005; Schachar et al., 2004; Slomine et al., 2005). Many cases have also presented with lesions in frontal and subcortical brain areas, including the orbitofrontal cortex, basal ganglia, and thalamus (Gerring et al., 2000; Max et al., 2004, 2005). Of interest, abnormalities in these brain regions are similarly involved in P-ADHD (Dickstein, Bannon, Castellanos, & Milham 2006; Gopin & Healey, 2011). The pattern of executive control impairment (e.g., response inhibition) also suggests dysfunction of frontal-subcortical circuits (Barkley, 1997; Pennington and Ozonoff, 1996). In addition to its presence in children and adolescents with ADHD, poor inhibitory control has been a consequence of childhood TBI (Leblanc et al., 2005; Levin et al., 2002). For instance, Ornstein et al. (2013) found that children with P-ADHD and children with TBI but no history of ADHD before or after TBI (termed “TBI-only” from here on) presented with poorer inhibitory control than

did typically developing controls. Although there was no significant difference between children with P-ADHD and those with S-ADHD, the mean scores suggested that the pattern of inhibitory performance in children with S-ADHD was similar to that of children with P-ADHD, while performance among children with TBI only was similar to that of healthy controls. Nevertheless, the nature of such difficulty in S-ADHD is relatively unknown. Furthermore, there is a paucity of research evaluating the similarities and differences in performance on executive control tasks among children and adolescents with P-ADHD, TBI-only, and S-ADHD.

Few studies have examined aspects of executive functioning in children and adolescents with S-ADHD and of those published studies, the focus has been on the inhibition of a speeded motor response because this is one of the signature deficits of P-ADHD. Konrad, Gauggel, Manz, and Scholl (2000) found that children with ADHD and children with TBI both demonstrated impaired performance on the Stop Signal Task, a measure of inhibitory control, as compared to controls. However, there were no differences in performance between the TBI children with and without S-ADHD. In a later study conducted by Sinopoli, Schachar, and Dennis (2011), both children with P-ADHD and S-ADHD were found to have inhibitory deficits as compared to controls on the Stop Signal Task. However, mean scores were notably slower for the P-ADHD group, suggesting that abnormal inhibitory control in children with S-ADHD is not as severe as the impairment exhibited by children with P-ADHD. Schachar et al. (2004) found that deficits in motor response inhibition, using the same task as that used by Konrad et al. (2000) and Sinopoli et al. (2011) was present only when S-ADHD and severe TBI co-occurred. Also in children with severe TBI, Slomine et al. (2005) reported finding some selective attention and executive deficits (e.g., working memory) in children with TBI and ADHD (a combined group of children with both P-ADHD and S-ADHD) as compared to children with TBI-only.

There is some recognition that children with S-ADHD show a degree of executive control difficulty, albeit the nature and extent of such impairment remains to be determined. No study to date has compared these groups on aspects of executive function. Only one study (Slomine et al., 2005) has examined planning ability (i.e., ability to organize a sequence of moves). No differences were observed among the TBI-only, P-ADHD, and S-ADHD groups, for the Tower of Hanoi Test (Welsh, 1991).

Several longitudinal studies have found that recovery following TBI begins during the first few weeks after the injury, and through the first-year, but then asymptotes around the 2-year mark (Jaffe, Polissar, Fay, & Liao, 1995; Yeates et al., 2002). However, there is indication that inhibitory control deficits do persist following TBI (e.g., Levin et al., 2008; Sinopoli, Schachar, and Dennis, 2011). Furthermore, poor inhibitory control is not short-lived in children with ADHD (Barkley & Fischer, 2011; Fenwick & Anderson, 1999; Hinshaw, Carte, Fan, Jassy, & Owens, 2007; Thorell, 2007). To date, there have been no published studies that have evaluated the extent of executive control difficulties at 6-months and 12-months in children with S-ADHD.

Thus, we set out to determine whether: (1) children with S-ADHD show executive control deficits; (2) the pattern of impairment differs between P-ADHD and S-ADHD; and (3) the

pattern of performance differs at 6- and 12-months post-injury. We were also interested in exploring whether S-ADHD would be related to certain pre-injury child functioning, such as adaptive functioning, SES, and injury severity. Based on the extant research, we hypothesized that children with: (1) S-ADHD would demonstrate executive function deficits; (2) P-ADHD would show greater dysfunction than that seen in S-ADHD (given their existent vulnerability and subsequent trauma), who in turn would present with greater impairment than those with TBI-only; and hence, (3) P-ADHD would maintain the worst performance overall, while the children with S-ADHD would present like those with TBI-only at 12-months post-injury. Defining a neurocognitive profile of S-ADHD may provide important clues about the mechanisms involved in the expression of the syndrome, and possibly indicate different neural underpinnings between P-ADHD and S-ADHD.

METHODS

Psychiatric Assessment

The sample consisted of 177 children with mild to severe TBI recruited during their initial hospitalization following a TBI at five academic medical centers. Pre-injury DSM-IV-based psychiatric diagnoses (Diagnostic and Statistical Manual of Mental Disorders, 4th Edition; American Psychiatric Association, 1994) were derived at the baseline assessment using a semi-structured interview (Schedule for Affective Disorders and Schizophrenia for School-Aged Children, Present and Lifetime Version; Kaufman et al., 1997). Best-estimate Psychiatric diagnoses (Leckman et al., 1982) were determined based on the parent and child reports from the K-SADS interviews and the Survey Diagnostic Instrument (Boyle et al., 1996), where available. The same procedures were followed at the 6-month follow-up and the 12-month followup to document the presence of psychiatric disorders, including onset of S-ADHD at the respective points post-injury. The exclusion criteria included pre-existing schizophrenia, pervasive developmental disorder or autistic disorder, mental deficiency, and injury due to child abuse. Signed consent and assent forms were obtained, consistent with research requirements of the Institutional Review Boards at each participating site.

Injury severity was based on the lowest post-resuscitation score on the Glasgow Coma Scale (GCS) (Teasdale & Jennett, 1974) and derived from each child's medical record, upon admission to the hospital. The scale is based on verbal response, eye-opening behavior, and motor responses, and ranges from 3 to 15 with higher GCS scores representing better responsiveness. Head injury severity was classified as mild (13–15), moderate (9–12), or severe (3–8).

Socioeconomic status (SES) was assessed through the Four Factor Index (Hollingshead, 1975). Scores were derived from a formula involving both maternal and paternal educational and occupational levels. Scores range from 8–66, with higher scores indicating higher status.

Adaptive functioning at baseline as well as at 6- and 12-months was measured using the Vineland Adaptive Behavior Scale (Sparrow, Balla, & Cicchetti, 1984). This involved a semi-structured interview with the parent (usually the mother), and surveyed activities that the child usually demonstrates in the environment. An overall composite score and separate

standard scores for Socialization, Daily Living and Communication domains were generated.

Measures

In addition to the baseline assessment, the children were assessed at 6-months and 12-months post-injury with a series of tests selected for their sensitivity to TBI-related neurocognitive dysfunction. Although participants were seen at three time points (i.e., baseline, 6-months, and 12-months), cognitive testing was conducted at the 6-months and 12-month time points only. The measures used were as follows:

The *Wechsler Abbreviated Scale of Intelligence* (WASI) (Wechsler, 1997) was administered to all children at 6-months post-injury. The WASI is a test of intelligence that evaluates verbal and non-verbal knowledge and reasoning. The test provides a full-scale, performance- and verbal-based intelligence score (VIQ). The VIQ is typically least sensitive to TBI and reported here to establish an estimate of baseline premorbid function.

The *stop-signal paradigm* (Logan, 1994) provides a direct measure of the speed for executing and voluntarily inhibiting a motor response and involves two concurrent tasks, a “go” task and a “stop” task. The go task involves a simple choice reaction time task. The stop task involves a tone emitted from the computer, which follows the presentation of the go task stimulus and instructs participants to withhold their response on that particular trial. The tone occurs randomly on 25% of trials and the later the tone is presented, the more difficult it is to stop the response to the go stimulus. Inhibitory control depends on the latency of two independent processes – the response to the go signal (go reaction time, goRT) and the ability to inhibit response to the stop signal. The outcome of the race between go and stop processes depends on the interval between onset of the go signal and the onset of the stop signal, referred to as stop signal delay. This “tracking” algorithm converges on the stop signal delay at which individuals are able to inhibit 50% of the time. The mean latency of the goRT is observable from the 75% of trials in which no stop signal is presented. The latency of the stop process is unobservable. If the individual stops, no response is evident. If the go process finishes before the stop process, the individual responds as if no stop had been presented. However, the latency of the unobserved stop process can be computed by subtracting the mean delay (at which the individual inhibits 50% of the time) from the mean goRT. Slower speed of this stopping process (i.e., a larger latency) reflects deficient inhibition. For each participant, the stop signal reaction time (SSRT), the probability of inhibiting, goRT, and the probability of correct go trials was recorded.

The *Tower of London* (TOL) (Shallice, 1982) assesses planning skill that involves the ability to look ahead, follow rules, conceive of alternative solutions to the problem, and to weigh and make choices. The test requires that the participant arrive at the most direct, fewest move solution by determining the order of moves necessary to rearrange three coloured beads on pegs of three disks of different heights, while complying with a set of specific rules. Data were collected for total planning time (the time elapsed before the first move made) and solution time (the time taken to come to the final solution for that trial). The number of broken rules was also calculated.

Semantic and phonological working memory were evaluated using the computerized Letter Identity and Letter Rhyme N-Back tasks, respectively (Levin et al., 2002). Each has three levels of memory load: 1-back, 2-back, and 3-back. There is also a 0-back condition that imposes minimal memory load. The Letter Identity condition involved matching the same alphabetic letters printed in different cases. A similar letter matching task was used for the Letter Rhyme condition; however, in this case, matching is based on letters that rhyme. For each level, a string of 40 letters appeared one at a time for 2 s on the screen. The participant responded by pressing a button with the preferred hand when a match occurred or, in the 0-load condition, when a designated target appeared. The percentage of hits (i.e., detection of targets) and false alarms was recorded.

Attentional processes in single and dual task performance were evaluated using the *Divided Attention Task* (DAT). The DAT assesses the ability to allocate attentional resources when simultaneously engaged in performing two independent tasks (Hiscock, Kinsbourne, Samuels, & Krause, 1987). Timed comparisons were evaluated for performing the single task of finger tapping (i.e., a measure of psychomotor speed) versus simultaneously performing the dual tasks of finger tapping and reciting a nursery rhyme.

RESULTS

Demographic Characteristics

Six-month assessment—One hundred seventy-seven children underwent a baseline assessment during their initial hospitalization. One hundred forty-one children of the original sample of 177 (80%) returned for the 6-month psychiatric assessment. The returning group was not significantly different from those who did not return for age, gender, GCS, SES, psychosocial adversity, or for pre-injury adaptive behavior. Unremitted S-ADHD was present in 17/141 (12%) study participants (see Table 1). The children with P-ADHD included 26/141 (18%) study participants, while the TBI-only group included 98/141 (70%) study participants. Demographic characteristics were compared using chi-square tests or univariate analysis of variance (ANOVA). There was a significant difference among all groups for gender [$\chi^2(2) = 8.41; p < .05$]. The P-ADHD group included a significantly higher percentage of males (92%) than did the children with S-ADHD (58%) ($p < .01$) and TBI-only groups (64%) ($p < .05$). The groups did not differ for age of injury, age of assessment, GCS, and VIQ. SES differed among the groups [$F(2,138) = 4.52; p = .01$], wherein the children with S-ADHD had significantly lower mean SES than the TBI-only group ($p < .05$). No other group difference emerged for SES status. The groups also differed for adaptive functioning (Composite Index: $F(2,139) = 10.82; p < .001$), revealing that the children with S-ADHD and P-ADHD demonstrated poorer adaptive functioning compared to those without ADHD. Furthermore, the group with P-ADHD and those with S-ADHD demonstrated poorer communication skills as compared to the children without ADHD (p 's $< .05$). There was also a significant difference between the children with S-ADHD and the children with TBI-only for socialization skills ($p < .05$). No other significant comparisons emerged.

Twelve-month assessment—One child from the original sample of 177 children had a second TBI between the 6- and 12-month assessments that made her ineligible for these analyses. One hundred twenty-five of the remaining 176 children (71%) returned for the 12-month psychiatric assessment. Termination of the funding cycle accounted for nine of the children who did not return; therefore, effective participation was 125/167 (75%). The returning group was not significantly different from those who did not return for age, gender, GCS, SES, psychosocial adversity, or for pre-injury adaptive behavior.

At the 12-month assessment, unremitted S-ADHD was present in 14/125 (11%) study participants. The P-ADHD group comprised 22/125 (18%) of study participants, while the TBI-only group comprised 89/125 (71%) of study participants. Gender remained significant among the three groups [$\chi^2(2) = 9.52; p < .05$], with a similar distribution as that seen at 6-months post-injury. There were no differences among the groups for age at injury, age at assessment, and the GCS. In this instance, the groups did not differ for SES. Significant findings for adaptive functioning remained [$F(2,110) = 12.51; p < .001$], whereby children with S-ADHD showed communication difficulties as compared to the TBI-only group ($p < .05$). The children with S-ADHD and P-ADHD also had poorer socialization skills than the TBI-only group (p 's $< .05$). There were no other significant group comparisons.

Neuropsychological Test Results

All statistical details are presented in Tables 2 and 3. Those children evaluated at 6-months and 12-months post-injury were not identical. Some children did not complete cognitive testing at the 12-month mark. For this reason, the data were examined using separate ANOVAs for each time point. The Games-Howell *post hoc* test was used to further explore significant effects, where necessary, due to small sample size and unequal variances. Regression analyses were done to consider group differences for gender and SES. Where significant, it is noted in the text.

Response Inhibition

Contrary to Hypotheses 2 and 3, which stated that children with S-ADHD would show executive function deficits, and that children with P-ADHD would demonstrate worse performance than those with S-ADHD who would in turn would show impairment as compared to the TBI-only group, there were no significant differences among the groups at 6-months and 12-months post-injury for response inhibition as measured by the SSP.

Planning Ability

Repeated-measures ANOVA was used to account for the multiple levels of difficulty of the TOL test. Group differences for gender and SES were observed. Linear regression analyses were conducted at each time point to evaluate whether neurocognitive performance was related to the ADHD diagnosis among TBI children after controlling for age at injury, gender, severity, SES, and adaptive functioning. The goodness of fit of the resulting model was evaluated with the R squared coefficient.

For planning ability as measured by the TOL, group differences emerged for total solution time at 6-months following TBI [$F(2,142) = 3.51; p < .05$]. *Post hoc* analyses revealed that

both the TBI-only and S-ADHD groups took a longer time to reach the solution as compared to the P-ADHD group. As predicted (see Hypothesis 1), children with S-ADHD demonstrated planning difficulty; however, contrary to Hypotheses 2 and 3, the children with P-ADHD maintained the best performance overall. No other group differences emerged. There were no differences among the groups for total planning time and total rules broken. There were no group differences for any of the measures at 12-months post-injury. Regression revealed that age at injury (i.e., older age at injury was associated with better performance) related to performance for the TOL total solution time (raw scores, not age-standardized; 6-months: $B = -15$; $p < .05$; 12-months: $B = -14.43$; $p < .01$). The TOL dependent measures were not found to vary as a function of SES, gender, adaptive functioning, or injury severity (i.e., GCS).

In summary, the TBI-only and S-ADHD groups showed planning difficulty, while the P-ADHD children reached the solutions fastest and maintained the best performance overall.

Working Memory

The N-Back tasks were analyzed using repeated-measures ANOVA due to multiple levels of difficulty. No group differences emerged at the 6-month time point for the Letter Identity N-Back task. Consistent with Hypothesis 1 at 12-months post-injury, the S-ADHD group made fewer target hits on the task for the 2-back condition as compared to the TBI-only group [$F(2,100) = 4.73$]; $p < .05$]. Partly consistent with Hypothesis 2, the P-ADHD group made more false alarms for the 1-back condition as compared to the TBI-only group [$F(2,100) = 5.58$]; $p < .01$]. Although the mean false alarms for the children with S-ADHD appear greater than that for both the TBI-only and P-ADHD groups, *post hoc* analysis revealed no significant difference. On the Letter Rhyme N-Back task, there was a group by level interaction for false alarms at 6-months post-injury [$F(6,339) = 4.39$]; $p < .001$]; however, *post hoc* analysis revealed no pair wise differences. At the 12-month time point, the S-ADHD group made fewer target hits for the 1-back condition as compared to both the P-ADHD and TBI-only groups [$F(2,95) = 4.27$]; $p < .05$]. In contrast to the findings at 6-months post-injury, no group differences emerged for false alarms on the Letter Rhyme N-Back task at the 12-month time point. No other group differences emerged.

Overall, the children with S-ADHD made fewer target hits at both time-points, the P-ADHD group demonstrated variable performance, while the TBI-only group maintained the best performance.

Attention

For dual task attentional processes as assessed by the DAT, there was reduced psychomotor speed bilaterally at 6-months post injury [Right hand: $F(2,116) = 4.40$; $p < .05$; Left hand: $F(2,117) = 4.65$; $p < .01$], with the children with S-ADHD showing slowed performance as compared to the P-ADHD group only ($p < .05$). Group differences for psychomotor speed also emerged bilaterally [right hand: $F(2,107) = 6.63$; $p < .01$; left hand: $F(2,108) = 5.03$; $p < .01$] at 12-months post-injury, with the S-ADHD and TBI-only groups showing slowed performance as compared to the P-ADHD group. Of interest, and in contrast to Hypotheses 2 and 3, group differences in the dual task emerged bilaterally [right hand: $F(2,107) = 5.30$;

$p < .01$; left hand: $F(2,108) = 3.77$; $p < .05$] at 12-months post-injury only, with the children with S-ADHD exhibiting more difficulty with dual task attention processes (i.e., fewer words correct) as compared to the TBI-only and P-ADHD groups (p 's $< .05$). Performance did not differ between the TBI-only and P-ADHD groups. Regression revealed that age at injury (i.e., older age at injury was associated with better performance) related to right- and left-hand tapping performance (6-months: right-hand: $B = 3.59$; $p < .001$; left-hand: $B = 3.28$; $p < .001$; 12-months: right-hand: $B = 3.31$, $p < .001$; left-hand: $B = 3.22$; $p < .001$) and dual task performance (6-months: right-hand: $B = 5.53$; $p < .001$; left-hand: $B = 5.73$, $p < .001$; 12-months: right-hand: $B = 5.36$, $p < .001$; left-hand: $B = 5.42$, $p < .001$).

Generally, the S-ADHD group showed slowed performance at both 6-months and 12-months post-injury. Of interest, the S-ADHD group exhibited more difficulty with dual task attention processes at 12-months post-injury only.

DISCUSSION

The purpose of the present study was to examine neurocognitive dysfunction in S-ADHD at 6-months and 12-months post-TBI as compared to children with TBI who had P-ADHD and to those with TBI only. This study expands upon the few previously published reports of neurocognitive performance in S-ADHD. Based on the small published literature, we anticipated executive function impairment among the children with S-ADHD. Partly consistent with Hypothesis 1, children with S-ADHD demonstrated some neurocognitive compromise in the domains of attention and working memory as compared to the P-ADHD and TBI-only children. We also expected that children with P-ADHD would show greater dysfunction than that seen in S-ADHD, due to their existent vulnerability and subsequent trauma. However, contrary to Hypothesis 2, the S-ADHD group as compared to the other two groups demonstrated the most difficulty on the above-noted cognitive processes. The children with S-ADHD were found to have working memory difficulty at both 6-months and 12-months post-injury. Slowed psychomotor speed was also noted at both time points, while attentional issues emerged at 12-months post-injury, suggesting that an attention deficit is not just a function of slowed cognition, and that cognitive compromise emerges beyond the acute phase of injury. Children with S-ADHD and TBI-only were found to respond more slowly than the children with P-ADHD for planning ability. Finally, Hypothesis 3 stated that children with P-ADHD would maintain the worst performance overall, while children with S-ADHD would present like those with TBI-only at 12-months post-injury. The hypothesis was also not supported. Attentional difficulties emerged for the S-ADHD group at 12-months post-injury. The finding is consistent with the published literature in adults which indicates problems with attention following TBI (Azouvi, Couillet, & Leclercq, 2004; Park, Moscovich, & Robertson, 1999). Furthermore, we noted that children with S-ADHD in particular presented with reduced adaptive functioning, but such disadvantage does not appear to have influenced S-ADHD neurocognitive performance.

Among those studies that have examined neurocognitive dysfunction in S-ADHD, the focus has been on inhibitory control processes. In the present study, however, evaluation of mean inhibitory control performance on the SSP revealed no significant group differences at either time point. Similarly, when compared to healthy controls (see Ornstein et al., 2013), no

differences among groups emerged when compared at 6-months post-injury, even though all groups showed slower SSRT (P-ADHD: 306.43 ms; S-ADHD: 313.53 ms; TBI-only: 306.72 ms) compared to the controls (229 ms). Previous research and current study findings question the relation of S-ADHD and the putative cognitive marker found in primary ADHD. Konrad et al. (2000) found that both the TBI and ADHD groups differed from controls for SSRT in the SSP, while a comparison between children with and without S-ADHD revealed no group differences. Clearly, the neurobiological substrate of S-ADHD and poor response inhibition may only partially overlap.

There are several other factors that distinguish between the present study and the study done by Konrad et al. (2000). Konrad and colleagues sampled children with much younger age at injury (range, 4–11 years) in the chronic stage of recovery (up to 6-years post-injury), who had greater severity of injury as denoted by at least a 3-month hospital stay post-injury. Each of these factors has been shown to affect the nature and extent of neurocognitive dysfunction in TBI (in the direction of poorer outcome; Chapman & McKinnon, 2000; Taylor, 2004; Taylor & Alden, 1997). Although not explicitly stated by Konrad et al., these variables could have contributed to the significantly greater task difficulty evidenced among their sample (SSRT: TBI = 455 ms; S-ADHD = 473 ms as compared to TBI = 306 ms and S-ADHD = 313 ms among our sample at 6-months post-injury). In fact, based on observation, our numbers improved over time among all groups, indicating rather intact inhibitory control performance at the 1-year mark. Hence, it is unclear at this point whether response inhibition is a function of ADHD per se, at least in children hospitalized for TBI. Moreover, injury severity may confer vulnerability to impairment; for example, response inhibition deficits were found in children with S-ADHD and severe TBI during the chronic phase of injury (Slomine et al., 2005). However, more studies are needed to address the influence of injury severity among children with S-ADHD.

With regard to planning ability, study findings revealed that the children with S-ADHD were found to perform no differently from children with and without pre-injury ADHD for planning time and rule violations. However, both children with S-ADHD and children with TBI-only had difficulty with task completion as compared to the children with P-ADHD at 6-months post-injury. Similar results were found when compared to control data (see Anderson, Anderson, & Lajoie, 1996). That is, the children with P-ADHD had the least difficulty with task completion. In a study of children with wide-ranging age (6–16 years) and severity of injury limited to severe TBI, Slomine et al. (2005) found no differences between children with TBI-only and those with S-ADHD using a similar planning-type task. The authors concluded that injury severity and age at injury seem to be more important predictors of performance than a diagnosis of ADHD. In the current study, regression analyses revealed that GCS did not affect planning performance and no differences among the groups emerged with regard to GCS. Nevertheless, inspection of raw GCS scores shows that the P-ADHD group had the highest scores, indicating a TBI of lower severity. S-ADHD has previously been found to be related to TBI of greater severity (Max et al., 2004). It is possible that a relationship between injury severity and planning ability is most prominent with more severe injury. Thus, our findings indicate that this specific executive function may be sensitive to the effects of brain injury (Shum et al., 2009), while not particularly sensitive to ADHD.

With respect to age at injury, Levin et al. (1997) reported that younger children with TBI show planning deficits. From an evaluation of the current data, it appears that older children likewise do better on this measure. Moreover, it seems that the children with P-ADHD appear on average older than the other participants. Regression revealed that older age at injury was associated with better performance for the TOL total solution time, which may explain why children with P-ADHD maintained the best performance overall. This is supported by the developmental trajectory associated with executive functions; that is, executive functioning processes develop sequentially throughout childhood and adolescence (Best, Miller, & Jones, 2009), and are related to the maturation of the frontal lobes (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001; Travis, 1998). More specifically, planning ability tends to develop rapidly in typically developing children between ages 7 and 10 years, and then more gradually throughout adolescence (Anderson, 2002).

Given the few published studies evaluating planning ability in TBI generally, and for S-ADHD more specifically, further research is necessary to substantiate the current results.

For working memory performance, there was no evidence of a disproportionate decline from the 0-back to 3-back conditions among any of the groups on the Letter Identity or Letter Rhythm N-Back tasks at the 6-month time point, indicating that all groups coped with the additional working memory load presented. Even though all children made some “false alarms,” there was no significant difference for this dependent measure between the groups. At 12-months post-injury, however, the children with P-ADHD were found to make more false alarms than did the TBI-only group. No other group differences emerged with respect to this index. For “target hits” on both the Letter Identity and Letter Rhythm N-Back tasks, the children with S-ADHD exhibited difficulty as compared to the other groups at 12-months post-injury, especially for the more demanding conditions.

We propose that the children with S-ADHD are presenting with a sustained attention deficit within the context of working memory. In fact, the pattern of performance among the S-ADHD children may reflect a vigilance decrement, consistent with that reported by Robin, Max, Stierwalt, Guenger, and Lindgren (1999), who speak to a particular attention “fatigue” (p. 706) among children with TBI who tend to show decreasing performance over a short duration of time. To our knowledge, no previous study has examined this phenomenon in children with S-ADHD. Furthermore, one study noted that increasing working memory load impacts vigilance performance, leading to a decrease in perceptual sensitivity to a given target (Helton & Russell, 2011). This could be reflected by slower and fewer hits, but not more false alarms, as demonstrated by the children with S-ADHD in the present study, and consistent with the results reported by Wassenberg, Max, Lindgren, and Schatz (2004), who noted that omission errors on a continuous performance test predicted a diagnosis of S-ADHD. Based on the small literature and the results of the current study, children with S-ADHD appear to exhibit greater attention/vigilance difficulty at 12-months post-injury as compared to the children with P-ADHD or TBI-only, indicating a sustained attention deficit specific to S-ADHD.

A specific attention deficit among the children with S-ADHD is also supported by this group’s difficulty with dual attentional processes. Performance did not differ between the

children with and without pre-injury ADHD at the 6-month assessment; however, difficulty with dual attentional processes emerged only at 12-month for the S-ADHD children and, appears independent of psychomotor speed. Our findings are consistent with previously published studies that have revealed persistent attention deficits 1- and 2-years after TBI (e.g., Ginstfeldt & Emanuelson, 2010).

We must acknowledge several limitations with the current work. It is possible that executive dysfunction is associated with only certain subtypes of S-ADHD. Neither S-ADHD nor P-ADHD were uniform in terms of clinical subtype. However, studies of children with developmental ADHD suggest that ADHD inattentive and combined subtypes have similar findings on executive function (Klorman et al., 1999; Schachar, Mota, Logan, Tannock, & Klim, 2000). Another limitation includes the attrition of sample over time, which precluded a longitudinal versus cross-sectional study design. Such a design would facilitate the evaluation of recovery over time. There was also no inclusion of healthy control children as the basis for comparison.

Several strengths of the study should also be noted. This is the largest pediatric TBI sample examining S-ADHD. Documentation of the categories according to the presence of ADHD and S-ADHD was done using the clinical gold standard of using a semi-structured psychiatric interview with a best-estimate rating that included consideration of behavioral ratings from teachers, when available. Some earlier studies used behavior checklist cut-offs to make the diagnosis of ADHD, which is a less sensitive method than the interview approach (Brown, Chadwick, Shaffer, Rutter, & Traub, 1981; Max et al., 1997; Schwartz et al., 2003; Wassenberg, Max, Koele, & Firme, 2004).

In summary, this study examined executive functioning performance in children with S-ADHD, along with P-ADHD and TBI-only. To date, neurocognitive findings and in particular, those related to executive control processes, have been rather sparse when it comes to the evaluation of children with S-ADHD. It has been found that children with S-ADHD in particular show deficits in working memory, attention, and psychomotor speed, especially at 12-months post-injury. This pattern of impairment and in the absence of deficient response inhibition indicates possibly different neural underpinnings between P-ADHD and S-ADHD. Those specific mechanisms whereby predisposing factors lead to S-ADHD neurocognitive phenomenology remain to be determined. Thus, further work to better define the neurocognitive profile of S-ADHD is warranted.

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References

- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th. Washington, DC: American Psychiatric Press; 1994.
- Anderson P. Assessment and development of executive function (EF) during childhood. *Child Neuropsychology*. 2002; 8(2):71–82. [PubMed: 12638061]
- Anderson P, Anderson V, Lajoie G. The tower of London test: Validation and standardization for pediatric populations. *The Clinical Neuropsychologist*. 1996; 10(1):54–65.
- Anderson VA, Anderson P, Northam E, Jacobs R, Catroppa C. Development of executive functions through late childhood and adolescence in an Australian sample. *Developmental Neuropsychology*. 2001; 20(1):385–406. [PubMed: 11827095]
- Azouvi P, Couillet J, Leclercq M. Divided attention and mental effort after severe traumatic brain injury. *Neuropsychologia*. 2004; 42:1260–1268. [PubMed: 15178177]
- Barkley RA. Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychology Bulletin*. 1997; 121:65–94.
- Barkley RA, Fischer M. Predicting impairment in major life activities and occupational functioning in hyperactive children as adults: Self-reported executive function (EF) deficits vs. EF tests. *Developmental Neuropsychology*. 2011; 36:137–161. [PubMed: 21347918]
- Best JR, Miller PH, Jones LL. Executive functions after age 5: Changes and correlates. *Developmental Review*. 2009; 29(3):180–200. [PubMed: 20161467]
- Bloom DR, Levin HS, Ewing-Cobbs L, Saunders AE, Song J, Fletcher JM, Kowatch RA. Lifetime and novel psychiatric disorders after pediatric traumatic brain injury. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2001; 40:572–579. [PubMed: 11349702]
- Boyle MH, Offord DR, Racine, et al. Identifying thresholds for classifying childhood psychiatric disorders. *Issues and Prospects*. *Journal of the American Academy of Child and Adolescent Psychiatry*. 1996; 35:1440–1448. [PubMed: 8936910]
- Brown G, Chadwick O, Shaffer D, Rutter M, Traub M. A prospective study of children with head injuries: III. Psychiatric sequelae. *Psychological Medicine*. 1981; 11(1):63–78. [PubMed: 7208747]
- Chapman SB, McKinnon L. Discussion of developmental plasticity: Factors affecting cognitive outcome after pediatric traumatic brain injury. *Journal of Communication Disorders*. 2000; 33:333–344. [PubMed: 11001160]
- Dickstein SG, Bannon K, Castellanos FX, Milham MP. The neural correlates of attention deficit hyperactivity disorder: An ALE meta-analysis. *Journal of Child Psychology & Psychiatry*. 2006; 47:1051–1062. [PubMed: 17073984]
- Fenwick T, Anderson V. Impairments of attention following childhood traumatic brain injury. *Child Neuropsychology*. 1999; 5:213–223. [PubMed: 10925705]
- Helton WS, Russell PN. Working memory load and the vigilance decrement. *Experimental Brain Research*. 2011; 212:429–437. [PubMed: 21643711]
- Hinshaw SP, Carte ET, Fan C, Jassy JS, Owens EB. Neuropsychological functioning of girls with attention-deficit/hyperactivity disorder followed prospectively into adolescence: Evidence for continuing deficits? *Neuropsychology*. 2007; 21:263–273. [PubMed: 17402826]
- Hiscock, Kinsbourne, Samuels, Krause. Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*. 1987; 18:643–662.
- Hollingshead, A. Four factor index of social status. New Haven, CT: Yale University, Department of Sociology; 1975.
- Horton AM, Soper HV, Reynolds CR. Executive functions in children with traumatic brain injury. *Applied Neuropsychology*. 2010; 17:99–103. [PubMed: 20467949]
- Hyder AA, Wunderlich CA, Puvanachandra P, Gururaj G, Kobusingye OC. The impact of traumatic brain injuries: A global perspective. *Neurorehabilitation*. 2007; 22:341–353. [PubMed: 18162698]
- Gerring J, Brandy K, Chen A, Quinn C, Herskovits E, Bandeen-Roche K, Bryan RN. Neuroimaging variables related to development of secondary attention deficit hyperactivity disorder after closed head injury in children and adolescents. *Brain Injury*. 2000; 14:205–218. [PubMed: 10759038]

- Gerring JP, Brady KD, Chen A, Vasa R, Grados M, Bandeen-Roche KJ, Denckla MB. Premorbid prevalence of ADHD and development of secondary ADHD after closed head injury. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1998; 37:647–654. [PubMed: 9628085]
- Ginstfeldt T, Emanuelson I. An overview of attention deficits after paediatric traumatic brain injury. *Brain Injury*. 2010; 24:1123–1134. [PubMed: 20715886]
- Gopin CB, Healey DM. The neural and neurocognitive determinants of ADHD. *Journal of Infant, Child, and Adolescent Psychotherapy*. 2011; 10:13–31.
- Jaffe KM, Polissar NL, Fay GC, Liao S. Recovery trends over three years following pediatric traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*. 1995; 76:17–26. [PubMed: 7811169]
- Kaufman J, Birmaher B, Brent D, Rao U, Flynn C, Moreci P, Ryan N. Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1997; 36:980–988. [PubMed: 9204677]
- Konrad K, Gauggel S, Manz A, Scholl M. Inhibitory control in children with traumatic brain injury (TBI) and children with attention deficit/hyperactivity disorder (ADHD). *Brain Injury*. 2000; 14:859–875. [PubMed: 11076133]
- klorman R, Hazel-Fernandez LA, Shaywitz SE, Fletcher JM, Marchione KE, Holahan JM, Shaywitz BA. Executive functioning deficits in attention-deficit/hyperactivity disorder are independent of oppositional defiant or reading disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1999; 38:1148–1155. [PubMed: 10504814]
- Lajiness-O'Neill R, Erdodi E, Bigler ED. Memory and learning in pediatric traumatic brain injury: A review and examination of moderators of outcome. *Applied Neuropsychology*. 2010; 17:83–92. [PubMed: 20467947]
- Leblanc N, Chen S, Swank PR, Ewing-Cobbs L, Barnes M, Dennis M, Schachar R. Response inhibition after traumatic brain injury (TBI) in children: Impairment and recovery. *Developmental Neuropsychology*. 2005; 28:829–848. [PubMed: 16266251]
- Leckman JF, Sholomskas D, Thompson D, Belanger A, Weissman MM. Best estimate of lifetime psychiatric diagnosis: A methodological study. *Archives of General Psychiatry*. 1982; 39(8):879–883. [PubMed: 7103676]
- Levin HS, Hanten G, Chang CC, Zhang L, Schachar R, Ewing-Cobbs L, Max JE. Working memory after traumatic brain injury in children. *Annals of Neurology*. 2002; 52:82–88.
- Levin H, Hanten G, Max J, Li X, Swank P, Ewing-Cobbs L, Schachar R. Symptoms of attention-deficit/hyperactivity disorder following traumatic brain injury in children. *Journal of Developmental & Behavioral Pediatrics*. 2007; 28(2):108–118. [PubMed: 17435461]
- Levin HS, Hanten G, Zhang L, Swank PR, Hunter J. Selective impairment of inhibition after TBI in children. *Journal of Clinical & Experimental Neuropsychology*. 2004; 26:589–597. [PubMed: 15370381]
- Levin HS, Song J, Scheibel RS, Fletcher JM, Harward H, Lilly MM, Goldstein F. Concept formation and problem-solving following closed head injury in children. *Journal of the International Neuropsychological Society*. 1997; 3:598–607. [PubMed: 9448373]
- Levin HS, Wilde EA, et al. Diffusion tensor imaging in relation to cognitive and functional outcome of traumatic brain injury in children. *Journal of Head Trauma Rehabilitation*. 2008; 23:197–208. [PubMed: 18650764]
- Logan, GD. On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In: Dagenbach, DD.; Carr, TH., editors. *Inhibitory processes in attention, memory, and language*. San Diego: Academic Press; 1994. p. 189-239.
- Max JE, Arndt S, Castillo CS, Bokura H, Robin DA, Lindgren SD, Mattheis PJ. Attention-deficit hyperactivity symptomatology after traumatic brain injury: A prospective study. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1998; 37:841–847. [PubMed: 9695446]
- Max JE, Lansing AE, Koele SL, Castillo CC, Bokura H, Schachar R, Williams KE. Attention deficit hyperactivity disorder in children and adolescents following traumatic brain injury. *Developmental Neuropsychology*. 2004; 25:159–177. [PubMed: 14984333]

- Max JE, Robin DA, Lindgren SD, Smith WL Jr, Sato Y, Mattheis PJ, Castillo CS. Traumatic brain injury in children and adolescents: Psychiatric disorders at two years. *Journal of the American Academy of Child and Adolescent Psychiatry*. 1997; 36:1278–1285. [PubMed: 9291730]
- Max JE, Schachar R, Levin HS, Ewing-Cobbs L, Chapman SB, Dennis M, Landis J. Predictors of attention-deficit/hyperactivity disorder within six months after traumatic brain injury. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2005; 44:1032–1040. [PubMed: 16175108]
- Ornstein TJ, Max JE, Schachar R, Dennis M, Barnes M, Ewing-Cobbs L, Levin HS. Response inhibition in children with and without ADHD after traumatic brain injury. *Journal of Neuropsychology*. 2013; 7:1–11. [PubMed: 23464806]
- Park NW, Moscovitch M, Robertson IH. Divided attention impairments after traumatic brain injury. *Neuropsychologia*. 1999; 37(10):1119–1133. [PubMed: 10509834]
- Pennington BF, Ozonoff S. Executive functions and developmental psychopathology. *Journal of Child Psychology & Psychiatry*. 1996; 37:51–87. [PubMed: 8655658]
- Robin DA, Max JE, Stierwalt JA, Guenzer LC, Lindgren SD. Sustained attention in children and adolescents with traumatic brain injury. *Aphasiology*. 1999; 13(9–11):701–708.
- Schachar R, Levin HS, Max JE, Purvis K, Chen S. Attention deficit hyperactivity disorder symptoms and response inhibition after closed head injury in children: Do preinjury behavior and injury severity predict outcome? *Developmental Neuropsychology*. 2004; 25:179–198. [PubMed: 14984334]
- Schachar R, Mota V, Logan G, Tannock R, Klim P. Confirmation of an inhibitory control deficit in attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology*. 2000; 28:227–235. [PubMed: 10885681]
- Schwartz L, Taylor HG, Drotar D, Yeates KO, Wade SL, Stancin T. Long-term behavior problems following pediatric traumatic brain injury: Prevalence, predictors, and correlates. *Journal of Pediatric Psychology*. 2003; 28(4):251–263. [PubMed: 12730282]
- Shallice T. Specific impairments of planning. *Philosophical Transactions of the Royal Society of London Series B-Biological Sciences*. 1982; 298:199–209.
- Shum D, Gill H, Banks M, Maujean A, Griffin J, Ward H. Planning ability following moderate to severe traumatic brain injury: Performance on a 4-disk version of the Tower of London. *Brain Impairment*. 2009; 10(03):320–324.
- Sinopoli KJ, Schachar R, Dennis M. Traumatic brain injury and secondary attention deficit/hyperactivity disorder in children and adolescents: The effect of reward on inhibitory control. *Journal of Clinical and Experimental Neuropsychology*. 2011; 33:805–819. [PubMed: 21598155]
- Slomine BS, Salorio CF, Grados MA, Vasa RA, Christensen JR, Gering JP. Differences in attention, executive functioning, and memory in children with and without ADHD after severe traumatic brain injury. *Journal of the International Neuropsychological Society*. 2005; 11:645–653. [PubMed: 16212692]
- Sparrow, S.; Balla, D.; Cicchetti, D. *The Vineland Adaptive Behavior Scales*. Circle Pines, MN: American Guidance Services; 1984.
- Taylor HG. Research on outcomes of pediatric traumatic brain injury: Current advances and future directions. *Developmental Neuropsychology*. 2004; 25:199–225. [PubMed: 14984335]
- Taylor GH, Alden J. Age-related differences in outcomes following childhood brain insults: An introduction and overview. *Journal of the International Neuropsychological Society*. 1997; 3:555–567. [PubMed: 9448369]
- Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet*. 1974; 2:81–84. [PubMed: 4136544]
- Thorell LB. Do delay aversion and executive function deficits make distinct contributions to the functional impact of ADHD symptoms? A study of early academic skill deficits. *Journal of Child Psychology and Psychiatry*. 2007; 48(11):1061–1070. [PubMed: 17995481]
- Travis F. Cortical and cognitive development in 4th, 8th, and 12th grade students: The contribution of speed of processing and executive functioning to cognitive development. *Biological Psychology*. 1998; 48(1):37–56. [PubMed: 9676358]

- Wassenberg R, Max JE, Koele SL, Firme K. Classifying psychiatric disorders after traumatic brain injury and orthopaedic injury in children: Adequacy of K-SADS versus CBCL. *Brain Injury*. 2004; 18(4):377–390. [PubMed: 14742151]
- Wassenberg R, Max JE, Lindgren SD, Schatz A. Sustained attention in children and adolescents after traumatic brain injury: Relation to severity of injury, adaptive functioning, ADHD and social background. *Brain Injury*. 2004; 18(8):751–764. [PubMed: 15204316]
- Wechsler, D. WAIS-III administration and scoring manual. San Antonio, TX: The Psychological Corporation; 1997.
- Welsh MC. Rule-guided behavior and self-monitoring on the Tower of Hanoi disk-transfer task. *Cognitive Development*. 1991; 6(1):59–76.
- Yeates OK, Taylor HG, Wade SL, Drotar D, Stancin T, Minich N. A prospective study of short- and long-term neuropsychological outcomes after traumatic brain injury in children. *Neuropsychology*. 2002; 16:514–523. [PubMed: 12382990]

Table 1

Study participants' demographic and injury characteristics

	TBI-only	P-ADHD	S-ADHD	F-values/ χ^2
6 Months post-injury	n = 98	n = 26	n = 17	
Age of injury (years)	10.20 (2.80)	10.51 (2.85)	9.69 (3.08)	0.44
Age of assessment (years)	10.74 (2.80)	11.07 (2.85)	10.24 (3.09)	0.44
Gender (% male)	64%	92%	58%	8.41 **
WASI Verbal IQ	99.38 (16.68)	95.24 (14.48)	93.13 (15.66)	1.35
GCS	10.66 (4.13)	12.23 (3.68)	9.53 (4.90)	2.39
SES	39.73 (12.04)	35.21 (13.00)	30.76 (12.94)	4.52 **
Adaptive Behaviour Composite	96.69 (14.21)	87.65 (12.74)	80.59 (19.68)	10.82 ***
Communication	98.01 (12.70)	90.81 (13.31)	86.12 (18.26)	7.27 ***
Daily Living Skills	97.38 (13.35)	91.46 (11.40)	84.82 (21.86)	6.45 ***◆
Socialization	97.07 (13.32)	89.58 (14.96)	81.06 (18.64)	10.35 ***
12 Months Post-injury	n = 89	n = 22	n = 14	
Age of injury (years)	9.98 (2.85)	10.53 (2.71)	9.29 (2.22)	0.86
Age of assessment (years)	10.98 (2.81)	11.62 (2.71)	10.37 (2.17)	0.94
Gender (% male)	58%	91%	64%	9.52 *
GCS	10.67 (4.14)	12.23 (3.84)	9.93 (4.75)	1.62
SES	38.73 (12.08)	35.45 (14.00)	31.68 (12.66)	2.17
Adaptive Behaviour Composite	97.69 (14.10)	86.41 (17.74)	77.64 (18.47)	12.51 ***
Communication	97.86 (13.15)	88.68 (16.06)	80.79 (19.65)	9.88 ***
Daily Living Skills	98.47 (13.60)	90.41 (16.61)	86.93 (20.15)	4.98 ***◆
Socialization	98.95 (12.15)	88.14 (19.05)	76.57 (20.08)	15.85 ***

Note. Values are expressed as means (standard deviation) except where indicated. S-ADHD subtypes at 6 months: inattentive ($n = 7$), not otherwise specified ($n = 6$), combined ($n = 3$), and hyperactive/impulsive ($n = 1$); S-ADHD subtypes at 12 months: inattentive ($n = 6$), not otherwise specified ($n = 5$), combined ($n = 1$), and hyperactive/impulsive ($n = 2$). P-ADHD subtypes at 6 months: inattentive ($n = 8$), not otherwise specified ($n = 6$), combined ($n = 8$), and hyperactive/impulsive ($n = 4$); P-ADHD subtypes at 12 months: inattentive ($n = 6$), not otherwise specified ($n = 6$), combined ($n = 7$), and hyperactive/impulsive ($n = 3$).

* < 0.05;

** 0.01;

*** 0.001;

◆ Significant omnibus result, but no significant pairwise comparisons.

WASI = Wechsler Abbreviated Scale of Intelligence. GCS = Glasgow Coma Scale; SES = socioeconomic status,

Table 2

Neuropsychological performance among the groups at 6-months post-injury

Variable	TBI-only	P-ADHD	S-ADHD	F-values	η_p^2
Stop-Signal Paradigm					
SSRT	306.72 (184.85)	306.43 (197.25)	313.53 (150.36)	0.01	.004
Go RT	658.83 (196.09)	701.33 (195.04)	658.00 (165.40)	0.43	.005
Go Accuracy (% Correct)	93.87 (6.25)	93.60 (5.16)	91.91 (6.28)	0.46	.009
Percentage of Inhibition	51.12 (7.81)	51.25 (9.98)	51.03 (2.55)	0.00	.002
Tower of London					
Total Planning Time (s)	121.23 (108.71)	84.36 (40.30)	117.47 (51.56)	1.58	.022
Total Solution Time (s)	340.38 (162.50)	260.45 (77.21)	350.01 (70.18)	3.51*	.041
Total Rule Violations	0.25 (0.76)	0.40 (1.04)	0.35 (0.70)	0.41	.005
Working Memory					
Identity Target Hits					
0-back	11.92 (0.31)	11.85 (0.37)	11.86 (0.36)	0.68	.009
1-back	11.28 (1.16)	11.08 (1.26)	10.21 (2.20)	3.93	.058
2-back	8.71 (3.11)	7.92 (3.39)	6.29 (3.17)	3.76	.057
3-back	6.78 (3.18)	6.38 (3.14)	5.77 (2.62)	0.67	.009
identity False Alarms					
0-back	0.48 (1.02)	0.54 (1.48)	0.43 (0.51)	0.05	.099
1-back	0.70 (1.83)	1.65 (3.82)	0.93 (1.64)	1.68	.021
2-back	1.17 (2.49)	1.31 (1.35)	1.21 (1.63)	0.04	.002
3-back	2.44 (2.99)	2.88 (3.99)	2.00 (1.78)	0.38	.004
Rhyme Target Hits					
0-back	10.48 (2.03)	9.61 (3.14)	8.62 (3.18)	3.92	.064
1-back	9.64 (3.05)	8.61 (3.88)	7.62 (3.48)	2.64	.043
2-back	6.46 (3.12)	5.91 (3.06)	5.33 (3.03)	0.84	.018
3-back	5.95 (2.84)	5.57 (3.17)	4.33 (2.27)	1.71	.032
Rhyme False Alarms					
0-back	1.35 (2.44)	2.43 (3.00)	4.62 (5.56)	6.94***◆	.108

Variable	TBI-only	P-ADHD	S-ADHD	F-values	η_p^2
1-back	2.40 (3.28)	2.57 (3.54)	4.15 (4.10)	1.48	.022
2-back	3.90 (3.75)	4.35 (4.46)	3.58 (3.60)	0.18	.002
3-back	5.27 (4.44)	3.91 (3.06)	3.67 (2.64)	1.56	.032
Divided Attention					
Right Hand Tapping	55.46 (16.02)	63.82 (13.57)	49.00 (14.28)	4.40*	.060
Rhyme/Right Tapping	83.19 (24.70)	88.91 (20.49)	72.00 (24.97)	2.23	.030
Left Hand Tapping	51.10 (13.25)	56.23 (11.99)	43.00 (12.46)	4.65**	.063
Rhyme/Left Tapping	81.13 (23.54)	85.86 (21.76)	69.00 (25.48)	2.41	.032

Note. Values are expressed as means (standard deviation) except where indicated.

* 0.05;

** 0.01;

*** 0.001;

◆ Significant omnibus result, but no significant pairwise comparisons.

Go RT = go reaction time; SSRT = stop-signal reaction time; s = seconds.

Table 3

Neuropsychological performance among the groups at 12-months post-injury

Variable	TBI-only	P-ADHD	S-ADHD	F-values	η_p^2
Stop-Signal Paradigm					
SSRT	269.30 (163.26)	295.98 (138.12)	362.44 (186.45)	1.43	.003
Go RT	631.89 (203.30)	696.96 (189.97)	776.28 (188.62)	2.60	.010
Go Accuracy (% Correct)	94.24 (6.77)	93.69 (6.02)	89.31 (11.65)	1.90	.004
Percentage of Inhibition	51.80 (7.02)	52.72 (4.25)	52.58 (4.57)	0.21	.006
Tower of London					
Total Planning Time (s)	104.38 (86.31)	100.66 (82.85)	95.62 (23.73)	0.08	.000
Total Solution Time (s)	323.52 (148.29)	301.08 (155.68)	341.84 (104.55)	0.36	.005
Total Rule Violations	0.11 (0.42)	0.36 (0.95)	0.29 (0.61)	1.93	.025
Working Memory					
Identity Target Hits					
0-back	11.88 (0.36)	11.87 (0.47)	11.91 (0.29)	0.75	.003
1-back	11.14 (1.32)	11.27 (1.58)	10.58 (1.38)	1.04	.001
2-back	9.25 (2.89)	8.64 (2.49)	6.50 (3.34)	4.73*	.043
3-back	6.73 (3.23)	7.36 (2.95)	5.08 (3.03)	2.06	.022
Identity False Alarms					
0-back	0.29 (0.77)	0.59 (1.18)	0.50 (1.73)	0.84	.026
1-back	0.46 (0.87)	1.23 (1.15)	1.50 (2.65)	5.58**	.059
2-back	0.78 (1.37)	1.32 (2.32)	1.17 (2.08)	0.95	.010
3-back	2.57 (3.06)	2.86 (2.95)	3.00 (3.59)	0.15	.002
Rhyme Targets Hits					
0-back	10.04 (2.64)	11.00 (2.70)	9.73 (2.41)	1.39	.052
1-back	9.33 (3.49)	10.40 (2.19)	6.91 (2.70)	4.27*	.067
2-back	6.78 (3.34)	7.30 (2.54)	5.40 (3.67)	1.16	.074
3-back	6.43 (2.96)	6.85 (2.60)	5.60 (3.72)	0.59	.044
Rhyme False Alarms					
0-back	1.72 (2.45)	2.75 (4.53)	3.54 (3.30)	2.18	.027

Variable	TBI-only	P-ADHD	S-ADHD	F-values	η_p^2
1-back	2.69 (3.03)	3.30 (2.98)	3.55 (4.03)	0.55	.006
2-back	4.00 (3.53)	3.70 (3.50)	6.40 (6.67)	1.81	.008
3-back	5.40 (4.31)	4.75 (3.13)	5.40 (0.70)	0.17	.031
Divided Attention					
Right Hand Tapping	59.05 (14.88)	66.95 (11.75)	48.62 (14.64)	6.63**	.085
Rhyme/Right Tapping	88.66 (24.62)	89.95 (12.23)	67.38 (19.08)	5.30**	.069
Left Hand Tapping	52.95 (12.77)	60.29 (8.57)	47.54 (11.77)	5.03**	.100
Rhyme/Left Tapping	85.26 (25.45)	89.57 (9.95)	68.69 (17.25)	3.77*	.061

Note. Legend: Values are expressed as means (standard deviation) except where indicated. Go RT = go reaction time; SSRT = stop-signal reaction time; s = seconds;

* 0.05;

** 0.01;

*** 0.001;

◆ Significant omnibus result, but no significant pairwise comparisons