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Performance monitoring in children following traumatic brain injury

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Background: Executive control deficits are common sequelae of childhood traumatic brain injury (TBI). The goal of the current study was to assess a specific executive control function, performance monitoring, in children following TBI. **Methods:** Thirty-one children with mild–moderate TBI, 18 with severe TBI, and 37 control children without TBI, of comparable age and sex, performed the stop signal task, a speeded choice reaction time task. On occasion, they were presented with a signal to stop their responses. Performance monitoring was defined as the extent of slowing in go-task reaction time following failure to stop responses. **Results:** The TBI group as a whole demonstrated less post-error slowing than did controls. This finding suggested impaired error monitoring performance. In addition, time since injury and socioeconomic status predicted less slowing after stopped responses. **Conclusions:** We suggest that alterations in performance monitoring expressed as the inability to notice, regulate and adjust behavior to changing situations are an effect of TBI in children. **Keywords:** Performance monitoring, traumatic brain injury, children, head injury, neuropsychology, pediatrics.

Traumatic brain injury (TBI) affects nearly half a million children each year (Langlois, Rutland-Brown, & Thomas, 2005) and is the leading cause of death disability among children and adolescents. TBI is commonly caused by pedestrian or bicycle-associated collisions, or motor vehicle accidents (Middleton, 2001). About 85% of all injuries are considered mild (see for review Yeates, 2000). TBI in children frequently produces impairment of executive control processes (Kaufman, Fletcher, Levin, Miner, & Ewing-Cobbs, 1993; Levin et al., 1994, 1996; Kelly & Eyre, 1999; Dennis, Guger, Roncadin, Barnes, & Schachar, 2001; Christ, White, Brunstrom, & Abrams, 2003) that are mediated by frontal-subcortical pathways (Alexander, Delong, & Strick, 1986). The impact on executive control processes has been attributed to the vulnerability of prefrontal cortex to focal lesions and the relatively late maturation of this region. (Proficient executive control performance relies on the integrity of the frontal lobes, which serve to organize and regulate behavior through the mediation of the so-called 'executive functions,' a term that refers to a range of processes that 'enable a person to engage successfully in independent, purposive, self-serving behavior') (see Lezak, 1995, p. 42).

A core executive control system function is the ability to monitor and regulate behavior, which involves identification of and adjustment to errors in performance (Rabbitt & Rodgers, 1977; Logan, 1985). Deficient performance monitoring can result in post-error slowing that is often construed as a behavioral adaptation to a changing environment or to the detection and correction of an error. Hence, performance monitoring is considered an 'adaptive' process – once error detection has occurred, behavioral adjustments or remedial action can take place that result in fewer errors and enhanced task performance (Holroyd & Coles, 2002).

Impaired performance monitoring has been identified in various neurologic and psychiatric populations with presumed dysfunction of the frontostriatal circuits (see for review, Ullsperger, 2006). In addition, several studies have shown that adults with frontal lobe lesions exhibit a pattern suggestive of deficient performance monitoring; these subjects make errors and are unconcerned by the consequences of their behaviors, including the lack of goal attainment (Rylander, 1947; Tow & Whitty, 1953; Eslinger & Damasio, 1985).

However, there has been little systematic investigation of performance monitoring in children. Krusch et al. (1996) reported that normal children tend to slow following errors made in speeded choice reaction time tasks. Wiersema, van der Meere, and Roeyers (2007) used an event-related potentials paradigm to demonstrate that, like adults, children exhibit error awareness and adjustment of response strategies, despite showing signs of a less

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well-developed error detection system. Deficient performance monitoring has also been documented in children diagnosed with attention deficit hyperactivity disorder (Schachar et al., 2004), who differ from normally developing children in terms of error detection or post-error adjustment to errors as reflected in the extent of slowing after errors.

There are several reasons to predict deficient performance monitoring after TBI in children. TBI impairs self-awareness and meta-cognitive ability ('knowing about knowing') in a way that suggests insensitivity to performance errors (Dennis, Donnelly, Wilkinson, & Humphrey, 1996; Hanten, Bartha, & Levin, 2000; Sherer et al., 2003). And, regulatory control deficits can distract from a child's overall ability to learn and function independently, confound other cognitive deficits, and impede the effectiveness of intervention (see for review Tarazi, Mahone, & Zabel, 2007). In addition, TBI in children, especially when severe, commonly causes injury to the frontal cortex and cellular dysfunction in white matter regions (Levin et al., 1997) that mediate goal-directed, monitoring processes.

The first goal of the current study was to assess performance monitoring in children with TBI. Performance monitoring in children who had suffered severe or mild–moderate TBI was compared to the performance of typically developing children. Children with traumatic brain injury were expected to manifest a deficit in performance monitoring. We also directly compared the two head-injured groups in order to evaluate the effect of severity on performance monitoring.

The present study considered whether performance monitoring varied with three variables known to affect neurocognitive outcome after childhood TBI: age at injury, time since injury, and socioeconomic status. Consequences of childhood brain injury and subsequent recovery depend on developmental factors and environmental influences, including socioeconomic status. For several neuropsychological outcomes, younger children who sustain TBI have poorer outcomes than those whose injuries occur in later childhood or adolescence (see for review Dennis, Wilkinson, Koski, & Humphreys, 1995; Gronwall, Wrightson, & McGinn, 1997; Taylor & Alden, 1997). The second aim was to evaluate the effect of age at injury and time since injury on performance monitoring. In regard to social environment, socioeconomic status can predict neurocognitive outcome after brain injury (Yeates et al., 1997), independent of injury severity (Max et al., 1999).

In order to detect errors and subsequent adjustment of performance, we used the stop-signal task. The stop-signal paradigm is a task that involves execution of a speeded motor response and occasional stopping of the response (Logan, 1985, 1994; Logan, Schachar, & Tannock, 1997). The go task is a speeded choice reaction time task. The stop task involves the random presentation of a signal following the go stimulus that instructs the participant to stop their ongoing response. The approach enables the examination of performance monitoring by evaluating the latency of go responses following failed inhibition trials. We identified non-stopped responses or trials in which a stop signal was presented, but the subject failed to stop, and the mean reaction time for the first correct go task response (go RT) that immediately followed each inhibition failure (Error + 1 RT; E + 1). We thus calculated post-error slowing as the difference between mean E + 1 RT and mean go RT to index performance monitoring.

Methods

Participants

Participants were 49 children (18 girls, 31 boys) who had sustained mild–moderate or severe TBI resulting from closed head trauma. Participation in the study was contingent on the acquisition of consent and assent forms completed by the parents and child, and with approval from the research ethics board at each participating institution. Participants were recruited from consecutive admissions of children at Memorial-Hermann Hospital, Texas Children's Hospital and Ben Taub General Hospital (Houston), Children's Hospital (Dallas), and The Hospital for Sick Children (Toronto) as part of an ongoing project on the outcome of TBI. These children were recruited for the study at the time of their initial hospital assessment and were followed prospectively at several time points over two years. The children from these clinics underwent a comprehensive evaluation that included screening for physical, motor or language limitations, and semi-structured clinical interviews and questionnaires with the parents and child. All were seen by a board-certified child psychiatrist. In addition, all children underwent standardized IQ testing with an abbreviated version of the Wechsler Intelligence Scale for Children – 3rd Edition (Wechsler, 1991). Performance monitoring was evaluated using the stop-signal paradigm and was assessed 6 months after the injury, on average.

The majority of children who suffered mild–moderate head injury (21) were involved in a motor vehicle crash, or experienced a fall. In comparison, the children with severe head injury (12) predominately suffered a motor vehicle crash, which included being struck by the vehicle. Mild–moderate TBI (n = 31) was defined by a brief loss of consciousness limited to no more than 15 minutes, a Glasgow Coma Scale (GCS) score of 13–15 (Teasdale & Jennett, 1974) after the child reached the emergency center, and no subsequent neurological deterioration (GCS score below 13 within 24 hours after injury). In addition, nearly half the children had positive CT findings (i.e., GCS = 13–15 with brain lesion). Severe TBI (n = 18) corresponded to a lowest post-resuscitation GCS score of 8 or less, regardless of CT findings. Although not all children had CT scans, CT findings revealed that there were 30 (61%) children with identifiable lesions, and 13 (26.5%) without a lesion. Of the 30 children with lesions, 9 had bilateral frontal lesions (3 mild cases, 6 severe cases),

9 had right frontal lesion (3 mild, 6 severe), 6 had left frontal lesion (5 mild, 1 severe) and 6 had non-frontal lesions (4 mild, 2 severe).

Participants were excluded if they fulfilled any of the following criteria: pre-injury neurological disorder, developmental or psychiatric disorder, learning problems, history of child abuse or a penetrating missile, concurrent use of medication other than a psychostimulant at the time of the evaluation, and children who did not speak English. All had normal vision and a full-scale IQ above 80. A comparison group of 37 typically developing children, similar in distributions of age and IQ, were recruited through advertisements placed in local newspapers and on staff bulletin boards of hospitals. Comparison children were assessed in the same way using parent interview and questionnaires and had to be free of lifetime psychiatric disorder and TBI.

Parents were asked about their child's pre-injury development and behavior during a baseline assessment which was typically performed within one month after injury. The calculation of socioeconomic status (SES) was derived by means of the Four Factor Index (Hollingshead, 1975). Classification depended on scores obtained from a formula involving parental education and occupation. Values on this scale ranged from 8 to 66, with higher values representing higher SES.

Table 1 summarizes the demographic and clinical features of study participants. Results indicated that the three groups did not differ in terms of age at testing (overall mean for the three groups: 10.5 years; range 5.3–15.3 years), or gender ratios (61.6% males), but they did differ in SES [F (2, 61) = 7.03, $p < .01$], reflecting that control subjects came from higher socioeconomic backgrounds than the severely injured children. The mild and severe TBI groups did not differ for age at injury (overall mean: 9.9 years; range: 5.0–15.1 years), or interval since their injury (overall mean: 6 months; range: 2–12 months).

Procedure

Measure of performance monitoring. This computerized paradigm entails pressing one of two keys as quickly as possible, the go response, and withholding a key press when a tone is sounded, the stop response. The tone occurs randomly on 25% of trials; the timing of the tone varies, thus making it easier or harder to stop. Whether or not an individual is able to stop a response is modeled as the outcome of a 'race' between the go and stop processes. If the go task process finishes before the stop task response, the response to the go task will occur. However, if the stop task response finishes before the go task response, the response to the go task will be inhibited. Stop-signal delay was adjusted by a tracking procedure, increasing by 50 ms if subjects inhibited successfully and decreasing by 50 ms if subjects failed to inhibit. With this tracking procedure, the individual is able to inhibit 50% of the time (for review see Logan et al., 1997).

The go-task stimuli were uppercase letters X and O presented in the center of the screen for 1000 msec. Each trial was preceded by a 500 msec fixation point that was presented in the middle of the screen, and then followed by a 2000 msec blank screen. Each trial was 3.5 seconds. The stop signal was a tone of 1000 Hz. The task was presented in 8 blocks of 32 trials. Children held a push-button box and were instructed to use the left index finger on the X button and their right index finger on the O button. The X or O stimuli appeared equally often in each block. Each participant had practice trials prior to the experiment.

Statistical analyses

To measure performance monitoring, we identified trials in which a stop signal was presented, but the participant failed to stop (non-stopped responses). We determined mean reaction time (RT) for the first correct go-task response that immediately followed each inhibition failure (Error + 1 RT; E + 1). We then calculated post-error slowing as the difference between mean E + 1 RT and mean go RT.

We also computed the mean latency of the go RT which was observable from the 75% of trials in which no stop signal was presented, and the latency of the inhibition process (stop signal reaction time, SSRT, which represents the time required to withhold the response after the tone is signaled) by subtracting mean delay (at which the subject inhibits 50% of the time) from mean go RT.

We examined performance monitoring and group differences by comparing the extent of slowing after failed inhibition in the mild–moderate, severe and comparison groups using univariate ANOVAs and, where necessary, applied post-hoc Tukey comparisons. Next, linear regression analysis was conducted to assess the effect of age at injury, GCS, SES, and gender on post-error slowing. The regression analyses began with a saturated model and proceeded with backward

Table 1 Demographic characteristics of participants

Mean	Severe TBI (N = 18)	Mild–moderate TBI (N = 31)	Controls (N = 37)	F/X^2 value significance
Age at test (years)	10.8 (2.7)	10.3 (2.9)	10.5 (2.3)	.23 ns
Range:	6.1–15.2	5.3–15.3	6.8–14.7	
Sex (% male)	61.1	64.5	59.5	.18 ns
SES	26.7 (12.54)	38.1 (14.93)	44.5 (14.39)	7.07**
Range:	10.5–50.5	12.5–61.0	15.5–63.5	Controls > Severe TBI
Age at injury (years)	10.3 (2.8)	10.0 (2.9)		.48 ns
Range:	5.6–14.6	5.0–15.1		
Years post injury	.5 (.1)	.5 (.2)		.29 ns

Note: ns = non-significant; ** = $p < .01$.

elimination of non-significant terms. The goodness of fit of the resulting model was evaluated with the R-squared coefficient (R^2).

Results

Table 2 shows stop signal task performance in the three study groups. One-way ANOVA with post-hoc comparisons showed the children with TBI slowed significantly less than the controls [F (2, 83) = 5.6, p = .05]. The mild–moderate injury group did not significantly differ from the severely injured group on post-error slowing. In addition, no significant differences emerged for slowing after non-stopped response among the TBI groups classified according to lesion location (data not shown). There was also a 61ms difference in SSRT between the children with TBI and controls [F (1, 84) = 8.37, p < .01]. There were no differences among the three groups in terms of mean percentage of correct go trials, mean go RT, or mean percentage of successful inhibition.

Regression analysis with all participants revealed that slowing after non-stopped responses did not vary with SES, age at test and gender, but did vary among the three groups (β = –.29, p < .01). Again, this latter finding revealed that controls slowed the most. There was no interaction between age at test and group.

Regression analysis with TBI participants showed that post-error slowing was significantly correlated with time since injury (β = –.30, p < .05) and SES (β = –.31, p < .05), but not correlated with severity, age at injury, or SSRT.

Discussion

The current study investigated performance monitoring in children with mild–moderate and severe TBI and normal controls. The results of the present study indicate that childhood TBI has an adverse impact on performance monitoring. Children with TBI slowed significantly less than typically developing children after failing to stop. The performance monitoring difference shown by children with TBI was not attributable to age or gender, nor was the difference explained by global slowing because there were no group differences in mean RT or accuracy on the go task. Moreover, difficulty in post-error slowing and SSRT were not linked in the TBI groups. To date, this is the first study to highlight disturbed performance monitoring following childhood TBI.

Several studies have shown that severity of TBI in children is directly related to the extent of executive control deficits, post-injury (Bakker & Anderson, 1999; Catroppa, Anderson, & Stargatt, 1999; Max et al., 1999; Ewing-Cobbs, Brookshire, Scott, & Fletcher, 1998 c.f.). Moreover, many studies have linked deficient performance monitoring to dysfunction of frontostriatal circuitry (Coles, Scheffers, & Holroyd, 2001; Dehaene, Posner, & Tucker, 1994; Gehring, Himle, & Nisenson, 2000; Gehring & Knight, 2000; Nieuwenhuis et al., 2002; Paus, 2001; Scheffers & Coles, 2000; van Veen & Carter, 2002a & b). We found that the presence of explicit frontal lesions was not required for performance monitoring deficits in children with TBI, and that the performance among the children with severe TBI was similar to that of the mild–moderately injured group. This unexpected finding may reflect variation in the presence of focal frontal lobe lesions among children with TBI of a range of injury severity (Mendelsohn et al., 1992). In addition, frontal lobe injury in those children may not have impaired consciousness or function in a way that affects GCS; GCS may be a significant predictor of cognitive outcome (e.g., Campbell, Kuehn, Richards, Ventureyra, & Hutchison, 2004).

Impaired speed of response as measured by mean goRT was not observed in the TBI sample in general, and specifically, in cases with severe TBI. Previous research has demonstrated slower go responses in moderate–severe children with TBI on measures of inhibitory control and in particular, with the use of the stop-signal paradigm (Konrad, Gauggel, Manz, & Scholl 2000; c.f. Catroppa & Anderson, 2003; Brookshire, Levin, Song, & Zhang, 2004). It is of interest to note that while the difference in reaction time was not statistically significant, results suggest

Table 2 Performance in the stop-signal paradigm

Mean	Severe TBI (N = 18)	Mild–moderate TBI (N = 31)	Controls (N = 37)	Effect size	F-value significance
Go accuracy (%)	92.8 (8.7)	91.5 (6.9)	94.5 (5.6)		1.7 ns
% inhibition	49.8 (5.3)	49.1 (7.0)	51.5 (2.8)		2.2 ns
SSRT (ms)	298.3 (179.9)	291.5 (117.6)	233.8 (90.2)		8.37 **
					Controls < Mild & Severe TBI
Go RT (ms)	598.7 (130.1)	573.5 (146.5)	541.0 (138.3)		1.2 ns
E + 1 RT (ms)	625.3 (131.1)	595.1 (149.2)	603.9 (151.6)		.25 ns
Slowing after non-stopped responses (ms)	26.6 (43.8)	21.6 (47.9)	62.9 (49.8)	.133	5.6 **
					Controls > Mild & Severe TBI

Note: RT = reaction time; SSRT = stop signal reaction time; C + 1 RT = reaction time on go trials following stopped responses; E + 1 RT = reaction time on go trials following non-stopped responses; ms = milliseconds; ns = non significant; ** = p < .01.

that the children with TBI and those who suffered more severe injury did reveal slowed responding compared to controls (please refer to Table 2 means(SD)). Nevertheless, the present results indicate that the TBI children were not simply slow in responding to all stimuli, and importantly, emphasize deficient performance monitoring following childhood TBI.

Frontal lobe areas of the brain, particularly the posterior medial frontal cortex and lateral prefrontal cortex, have been implicated in performance monitoring. In fact, convergent evidence from neuroimaging studies indicates neuroanatomical demarcation of frontal lobe brain structures involved in regulating task performance in an adaptive manner (e.g., Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004a; Ridderinkhof, van den Wildenberg, & Segalowitz, & Carter, 2004b; Chevrier, Noseworthy, Schachar, 2007). Moreover, new emerging research hints at altered performance monitoring and differences in prefrontal brain function associated with dopaminergic genes (Kramer et al., 2007). Therefore, further investigation of the size and location of specific lesions and white matter disruption using advanced brain imaging might help to identify and characterize lesion-deficit associations. And, neurophysiological correlates of impaired performance monitoring as related to dopaminergic functioning suggest a possible underlying neural mechanism of executive control deficits, including performance monitoring impairment as expressed in children following brain injury.

We also addressed the influence of injury-related factors and environment on performance monitoring. Studies have clearly demonstrated that environmental and injury-related factors are linked to cognitive outcomes following TBI, and as well, act in concert to determine post-injury recovery of function (for review see Yeates, 2000). In the present study, deficient performance monitoring was related to SES, it was vulnerable to brain injury, and persisted, on average, at least six months post-injury. Consistent with this finding, recovery from TBI is exacerbated by family environments characterized by poorer family resources and overall functioning (e.g., Taylor et al., 1999; Yeates et al., 2004). In addition, children with TBI exhibit problems with social adjustment, peer interaction and information processing (see for review Yeates et al., 2007) that may be associated with deficits in action/error monitoring. Perhaps this combination of deficits arises from injury to overlapping brain structures critical for both social cognition and executive control processes. Nevertheless, 'the neural and social cognitive substrates of the persisting deficits are unclear' (Yeates et al., 2007, p. 550). Deficiencies in performance may become increasingly apparent over time, a possibility that underscores the importance of longitudinal investigation with longer follow-up intervals.

Limitations of the present study include its cross-sectional design and brain imaging confined to identification of focal lesions without measurement of lesion volume. Perhaps, certain factors such as volume of frontal brain lesions significantly influence performance on this measure of executive control, and may be a more salient predictor of performance than GCS. More advanced brain imaging such as diffusion tensor imaging could evaluate the integrity of circuitry implicated in response inhibition and performance monitoring. The apparent deficit in performance monitoring may be related to the fact that TBI alters the ability to make adjustments in response criteria following unsuccessful attempts to stop a response. TBI may also impair the ability to manage response conflict (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Botvinick, Braver, Barch, Carter, & Cohen, 2001; Gehring & Fencsik, 2001). In the case of the stop task, response conflict arises from the similarity of the motor response associated with stopped and non-stopped responses (Van Boxtel, Van der Molen, Jennings, & Brunia, 2001). More research is necessary, as knowledge about the precise mechanism involved in the performance monitoring system in children is inconclusive.

In summary, the present study demonstrates that children who have suffered a TBI exhibit a performance monitoring deficit. They slow less than typically developing children following failed efforts to stop a response. The relative failure to slow after erroneous inhibition responses may reflect faulty performance monitoring; an important executive control process that is independent of an inhibitory control deficit and generalized cognitive impairment, such as might be evident in slow or inaccurate responding. Alteration in performance monitoring could result in the inconsistent, inaccurate and poorly regulated behavior, deficient self-regulated learning, and lack of awareness of performance which often persist after TBI.

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