

Comparing attentional skills in children with acquired and developmental central nervous system disorders

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Abstract

Attentional impairments in children occur in the context of both developmental and acquired disorders involving the central nervous system (CNS) and may have implications for ongoing development, potentially impeding cognitive, educational, and behavioral functions. Using a continuous performance paradigm (CPT), this study compared attentional profiles of children with developmental and acquired conditions impacting on the CNS: (i) attention deficit–hyperactivity disorder (ADHD; $n = 27$); (ii) moderate traumatic brain injury (TBI; $n = 41$); (iii) acute lymphoblastic leukemia ($n = 31$); and (iv) insulin-dependent diabetes mellitus ($n = 39$). A healthy control group ($n = 46$) was also examined. Groups were compared on measures of sustained attention, selective attention, and response inhibition. In addition, measures of performance variability and deterioration and processing speed were examined. Results showed that children with ADHD exhibited global and severe attentional impairments in contrast to all other groups. Children with moderate TBI displayed mild attentional difficulties, restricted to selective and sustained attention domains. In conclusion, although CPT parameters differentiated the ADHD group from all others, a disorder-specific profile was not observed. (*JINS*, 2006, *12*, 519–531.)

Keywords: Sustained attention, Children, CNS, ADHD, TBI, Variability

INTRODUCTION

Abnormalities in attentional development are seen in a range of developmental disorders of childhood, for example, autism (Aronson et al., 1997; Casey et al., 1993; Wainwright-Sharp & Bryson, 1993), attention deficit–hyperactivity disorder (ADHD; Aman et al., 1998; August & Garfinkel, 1990; Levy & Hobbes, 2000), and Asperger's syndrome (Klin et al., 1995), as well as in children with central nervous system (CNS) dysfunction of multiple etiologies, including traumatic brain injury (TBI; Anderson et al., 2005; Anderson & Pentland, 1998; Catroppa & Anderson, 1999, 2003; Kaufmann et al., 1993), insulin-dependent diabetes mellitus (IDDM; Northam et al., 2001), Tourette's syndrome (Johannes et al., 2001; Yeates & Bornstein, 1994), and in children with acute lymphoblastic leukemia (ALL) treated with cranial irradiation (Anderson et al., 2004a; Brouwers

et al., 1985). As such impairments are described less commonly in adults (e.g., stroke, diabetes, cranial irradiation), it may be that attention is particularly vulnerable to disruption during childhood, when the brain is developing and when cognitive skills are emerging and consolidating. Furthermore, if a child is unable to attend efficiently, acquisition of new skills and knowledge may be limited. Support for the role of attention in development comes from reports of deterioration of intellectual abilities for children suffering from conditions for which attention deficits are common, for example, TBI (Anderson & Moore, 1995; Catroppa & Anderson, 1999, 2003; Ewing-Cobbs et al., 1998). An understanding of the nature of attention and its development, together with access to appropriate assessment methods, is vital for pediatric neuropsychology.

Models of Attention and Specific Attentional Components

The study of attention has been hindered by inconsistencies in terminology. In the 1800s, William James argued that

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“focalisation, concentration, and consciousness” were the key elements (James, 1890; pp. 403–404). A more contemporary description states that attention refers to “. . . all those aspects of human cognition that the subject can control . . . and to all aspects of cognition having to do with limited resources or capacity . . .” (Shiffrin, 1988, p. 739). Recently attempts have been made to compartmentalize attention into several separate, interacting components, forming a functional system (Mirsky et al., 1991; Posner, 1978; Stuss et al., 1995).

Sustained attention refers to the ability to maintain attention over time. Impairments in sustained attention are characterized as a gradual fall-off in performance over time. Fluctuating performance and individual variability are also described, with these skills mediated to a large extent by the reticular formation and brainstem (Mirsky et al., 1991), with some involvement of frontal regions (Stuss et al., 1995). *Selective, or focused, attention*, mediated by temporal, parietal, and striatal regions, is the ability to “filter” irrelevant stimuli or focus on a single stimuli (Cowan, 1997; Posner, 1984).

Several elements of attention are also conceptualized as overlapping with executive abilities, and represent the “executive” level of the attention system. *Response inhibition*, mediated primarily by means of frontal regions, refers to the capacity to inhibit an automatic or prepotent response. *Divided attention*, the capacity to attend to competing stimuli simultaneously and *shifting attention*, the ability to shift flexibly from one dimension to another, have also been linked to frontal lobes (Manly et al., 1999; Rebok et al., 1997). Finally, *processing speed*, linked to subcortical structures and anterior brain regions, reflects the rate at which tasks are completed and is commonly incorporated into attention models (Barkley, 1999; Ruff & Rothbart, 1996), reflecting the importance of these skills for effective attention, as well as their role in efficient performance on attentional measures. Disruption to this system may result in deficits in one or more aspects of attention (Mirsky, 1996; Stuss et al., 1995), which may then impact upon normal development.

Patterns of Attentional Function in Childhood Brain Disorders

The nature and degree of attention deficits have been shown to vary across disorders, due to underlying cerebral pathology or related to age at condition onset. The influence of such factors is well illustrated in TBI. In adult TBI, although many attentional skills are intact, processing speed is reduced (e.g., van Zomeren & Brouwer, 1994). For childhood injury, deficits are more global, incorporating processing speed and sustained and shifting attention (Catroppa & Anderson 1999, 2003; Ewing-Cobbs et al., 1998). Deficits vary according to injury severity and nature of underlying brain pathology, with minimal impairment after mild TBI (Willmott et al., 2000) but significant impairment after moderate/severe TBI (Anderson & Pentland, 1998; Ewing-Cobbs et al.,

1998). In contrast, developmental disorders (e.g., ADHD) are associated with reduced response inhibition, perhaps due to anterior cerebral dysfunction (Barkley, 1999; Shallice et al., 2002; Sowell et al., 2003), with other attentional components relatively intact.

Assessing Attention in Children

The young child has limited attentional capacity, reflecting the immaturity of the CNS, for example, unmyelinated axons and developing frontal lobes (Manly et al., 2001; Rebok et al., 1997). Development occurs in a set order and time frame, with anterior–posterior connections not fully developed until late childhood (Thatcher, 1991) and with different patterns of development identified for the separate elements of attention (Lane, 1978; Lane & Pearson, 1982; McKay et al., 1994; Rebok et al., 1997; Shepp et al., 1987). It is not surprising then that attention may be uniquely vulnerable to the impact of early brain insult, as has been demonstrated in several childhood conditions (Anderson et al., 2004b; Catroppa & Anderson, 2003; Kaufmann et al., 1993). Using Dennis’ (1989) heuristic, while these skills are developing they may be at risk for disruption, indicating that some knowledge of developmental processes in this domain is important to understanding the potential impact of brain insult through childhood. Thus, the thorough and accurate assessment and understanding of these skills in child practice is a high priority for clinicians.

The bulk of the literature on the development of attention has focused on the school-aged population. McKay and coworkers (1994) plotted development of sustained and selective attention and processing speed for children 6 to 13 years of age, and an adult sample. They reported early maturation of selective attention, stable sustained attention through middle childhood, and a developmental spurt around 11 years. Speed of processing showed gradual progress, with increments observed until 11 years. Similar findings are described by others (Manly et al., 2001; Rebok et al., 1997), with evidence of increases in all elements of attention from 8 to 10 years. These data emphasize the rapid development of attention through childhood and support the need for effective, age-sensitive clinical measures for its accurate assessment.

Currently, there are an increasing number of tests of childhood attention. However, the Continuous Performance Test (CPT; Rosvold et al., 1956) continues to be a commonly administered clinical paradigm, with several readily available commercial versions that generate a variety of indices of attention (Cooley & Morris, 1990; Levy & Hobbes, 2000). However, the traditional measures obtained and used clinically may be inadequately conceptualized, not necessarily tapping the attentional components described. For example, mean accuracy rates and reaction times, as measures of sustained attention (Mirsky et al., 1991), do not address fluctuation or deterioration in attentional performance. Furthermore, important information may be discarded when analysis is restricted to accuracy and reaction time vari-

ables. Van Zomeren and Brouwer (1994) advocate extending CPT indices to provide more detailed, component-specific data. Although less common in clinical practice, researchers previously have addressed this issue within specific groups of children with developmental and acquired disorders of the CNS, for example, ADHD (van der Meere & Sergeant, 1988), TBI (Catroppa & Anderson, 2003; Kaufmann et al., 1993), and cranial irradiation for the treatment of childhood leukemia (Anderson et al., 2004a). Results from these studies suggest that aspects of attention may be differentially affected in each condition, and comparison across findings raises the possibility that attentional profiles might well vary across groups. However, no study to date has compared CPT parameters across groups to determine whether this paradigm has the sensitivity to differentiate attentional patterns that might occur as a result of varying medical and neurological etiologies.

The Present Study

Mindful of the frequent inclusion of the CPT in clinical assessment batteries, the limitations identified by van Zomeren and Brouwer (1994), and debate regarding the nature and cause of attentional impairments in children, the present study had two aims: (1) to comprehensively investigate the efficacy of the CPT within the framework of contemporary models of attention (Stuss et al., 1995; van Zomeren & Brouwer, 1994) by identifying and measuring variables relating to components of the attention system, specifically sustained and selective attention, response inhibition, and processing speed; and (2) to test our expectation that the CPT could differentiate attentional profiles across clinical groups and, thus, have relevance for accurate diagnosis and intervention.

To meet these goals, children with developmental and acquired brain disorders were investigated. CNS conditions were chosen for comparison based on (1) documentation of impaired attention; (2) reported deficits in one or more attention components under study, enabling formulation of predictions regarding the pattern of impairment expected for each group; and (3) evidence of cerebral pathology or dysfunction from neuroimaging studies. Such cross-disorder comparisons, where groups can be compared with respect to their responses on the same test procedures, provide a means of determining both similar and distinct features, which might be associated with specific conditions, as well as potentially illuminating underlying brain-behavior relationships. This strategy has been illustrated in studies comparing the attentional profiles of children with attention deficit hyperactivity disorder to those with traumatic brain injury (Konrad et al., 2000) and learning disabilities (Micallef et al., 2001; Wu et al., 2002).

Attention deficit-hyperactivity disorder (ADHD)

Impairments in attention are considered the key feature of ADHD (Heaton et al., 2001), with response inhibition the

primary deficit (Barkley, 1999; Lajoie et al., 2005; Manly et al., 2001). Impairments in sustained and selective attention and processing speed are reported less consistently (Hooks et al., 1994; Micallef et al., 2001; Seidel & Joschko, 1990; van der Meere & Sergeant, 1988). Recent structural and functional imaging studies, have identified dysfunction within frontal brain regions (Baron-Cohen & Moriarty, 1995; Reeve & Schandler, 2001; Rubia et al., 1999; Semrud-Clikeman et al., 2000; Sowell et al., 2003).

Traumatic brain injury (TBI)

Although results vary, deficits in sustained attention and processing speed are commonly identified post-TBI, with impairments in selective attention and response inhibition less consistently reported (Anderson & Pentland, 1998; Dennis et al., 1995; Ewing-Cobbs et al., 1998; Schachar et al., 2004). Residual impairments are greatest after severe TBI, although moderate insults have also been associated with deficits. In contrast, attention skills after mild TBI appear intact (Anderson et al., 2004b). Imaging studies describe a characteristic pattern of cerebral pathology, primarily involving diffuse white matter injury and brainstem and anterior brain insult.

Insulin-dependent diabetes mellitus (IDDM)

Although not typically associated with cognitive sequelae in adults, studies of childhood IDDM report attention deficits, including reduced selective attention and speed of response (Hagen et al., 1990; Holmes, 1990; Northam et al., 1998), linked to lowered glucose levels, and associated abnormalities in the metabolism of neurotransmitters, most commonly in the prefrontal cortex and to a lesser extent the posterior cortex (McCall & Figlewicz, 1997; Tallroth et al., 1992), as well as history of hypoglycemic seizures.

Acute lymphoblastic leukemia (ALL)

Studies reporting on children treated with a combination of cranial irradiation and chemotherapy, document slowed processing speed and deficits in selective attention (Anderson et al., 2000; Cousens et al., 1988). In contrast, sustained and shifting attention skills appear to be spared (Anderson et al., 2004a; Goff et al., 1980). Acute pathology is uncommon; however studies conducted postradiation demonstrate delayed subcortical pathology in these children (Kingma et al., 1993; Paakko et al., 1992).

Based on cognitive and imaging literature, we predicted that the CPT would identify specific patterns of attentional impairments across the groups: (1) sustained attention deficits would be evident for the TBI group only, in keeping with brainstem and anterior pathology; (2) reduced response inhibition would be identified for children with ADHD and TBI, linked to frontal lobe involvement; (3) slowed processing speed, reflecting more diffuse pathology, would occur in TBI, IDDM, and ALL groups; and (4) deficits in selective attention would be specific to IDDM. To examine the

validity of the CPT in assessing more specific attentional weaknesses, as opposed to global cognitive impairments or motor or sensory deficits, children who had sustained severe brain insult (e.g., severe TBI, high-dose cranial irradiation) or who had residual neurological deficits (e.g., epilepsy, hydrocephalus, hemiplegia) were excluded from participation.

METHOD

Participants

The sample comprised 184 children (108 males). Justification for the choice of clinical groups is described above. Clinical group selection was on a consecutive basis by means of outpatient clinics (ADHD, IDDM) or record review (ALL, TBI) at the Royal Children's Hospital (RCH), Melbourne, Australia. In each of these groups, attentional impairments have been reported consistently, as has evidence of suspected or documented CNS dysfunction in regions argued to mediate attention. A control group was included for comparison. Inclusion criteria for all groups were (1) 8 to 15 years of age at assessment, (2) no premorbid or current history of neurological or sensory disorder, (3) no premorbid history of learning disability; and (4) attending mainstream school.

ADHD (n = 27)

Children recruited to this group represented consecutive referrals to a specialist clinic for evaluation of ADHD. Children were evaluated by a pediatrician and met Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV; American Psychiatric Association, 1994) criteria for a diagnosis of ADHD, based on clinical observations and parent report. In addition, parents and teachers completed the Rowe Behavioural Rating Inventory (RBRI; Rowe & Rowe, 1995), a 20-item scale, which provides three subscales: inattention, restlessness, irritability. Children were only included in the ADHD group if RBRI ratings were within the clinical range (>98th centile), for inattention and restlessness subscales, on either parent or teacher measures. Children with comorbid learning disability or psychiatric disorder were excluded from participation. The Wide Range Achievement Test—3 (Wilkinson, 1993), DSM-IV classification, and Conners' Parent Rating Scale (Goyette et al., 1978) were used to assess these domains. Although some children were receiving medication for the disorder, all had been off medication for 48 hours before testing.

TBI (n = 41)

Participants had sustained TBI of moderate degree at least 2 years before assessment. Children were ascertained by means of medical records review, and represented consecutive identifications meeting selection criteria. Moderate injury was chosen as these children had evidence of brain injury on brain imaging but no residual neurological abnor-

malities. Children with severe TBI were excluded, due to the well-documented and global nature of neurobehavioral sequelae, including intellectual (Anderson & Moore, 1995) and physical disability and neurological deficits (Catroppa & Anderson, 1999). Mean age at injury was 5.5 years ($SD = 2.9$), and mean time since injury was 6.1 years ($SD = 3.3$). All children had experienced a period of altered consciousness, with injuries of sufficient severity to warrant admission to a tertiary medical center. Specific inclusion criteria for this group were: (1) lowest Glasgow Coma Scale score (GCS) in the 24 hours after injury, 9–12; (2) duration of posttraumatic amnesia (PTA), 1–24 hours; (3) no evidence of neurological deficits; and (4) no premorbid or current diagnosis of ADHD or other neurological, developmental, or psychiatric disorder, based on pediatric evaluation. Mean GCS score was 10.5 ($SD = 2.8$).

IDDM (n = 39)

This group included consecutive admissions with a diagnosis of IDDM (type 1 diabetes) 2 years before participation in this study, who met study criteria. Mean age at diagnosis was 9.3 years ($SD = 2.4$). In the 2 years since diagnosis, 46% of the group had suffered at least one hypoglycemic episode resulting in altered consciousness, with 18% having episodes associated with convulsions and 13% having episodes associated with coma. No child had a residual neurological condition. Criteria for inclusion were (1) 5 years of age or older at diagnosis; (2) no premorbid or current diagnosis of ADHD or other neurological, developmental, or psychiatric disorder, based on pediatric evaluation; and (3) blood glucose levels within the normal range at assessment.

Childhood ALL (n = 31)

Participants were recruited by means of record review and were considered for inclusion if treated for ALL according to the ANZCCSG Study (V) protocol (Waters, 1992). Cranial irradiation was administered between 2 and 5 years of age, after children had achieved remission after induction chemotherapy. Children received a course of cranial irradiation (18 Gy) in combination with four doses of intrathecal methotrexate given at weekly intervals. Children also received two doses of intrathecal methotrexate, before irradiation, given on day 1 and day 21 of the chemotherapy regimen. All children met the following criteria: (1) dose of cranial irradiation therapy administered = 18 Gy; (2) single course of treatment and relapse-free before assessment; and (3) no premorbid or current diagnosis of ADHD or other neurological, developmental, or psychiatric disorder, based on pediatric evaluation.

Healthy controls (n = 46)

These children were selected from local schools and were chosen from class rolls. Matching was conducted at the group level, with the control sample constructed to match

clinical groups as closely as possible with respect to age and gender. Only children with no history of ADHD or other neurological, developmental, or psychiatric disorder (based on information collected from demographic questionnaire) were recruited.

Procedure

Children and families were contacted to participate in specific research studies at RCH and were required to provide written informed consent before their inclusion in the research, in keeping with hospital ethics requirements. Participation rates for all groups exceeded 80%. Parents provided details of each child's medical, educational, and developmental history and socioeconomic status (SES). SES was derived from parent occupation, using the Daniel Scale of Occupational Prestige (Daniel, 1983), which generates a rating between 1 and 6.9 with higher scores reflecting lower SES.

Neuropsychological Measures

Neuropsychological measures included the Wechsler Intelligence Scale for Children (WISC-III: Wechsler, 1991) and CPT (Rosvold et al., 1956). For the ADHD and TBI groups, Full Scale IQ (FSIQ) was derived from a short-form of the WISC-III (Vocabulary, Object Assembly, Similarities, Block Design), as described by Sattler (1988). All other participants completed the total WISC-III. The CPT measures sustained and selective attention, response inhibition and processing speed. The simultaneous discrimination version was used in this study, with monochrome visual display of stimuli. The CPT incorporated 600 trials in which two letters were flashed onto a computer screen for a duration of 500 milliseconds (ms), with an interstimulus interval of 1500 ms. The child was seated before the computer screen, and provided with a response box where the yellow button represented a "yes" and the blue button a "no". Children were instructed to respond to all stimuli by pressing the "yes" button if a "C" was flashed on the screen and the "no" button if neither of the letters was a "C". A practice session of 30 trials was administered to ensure understanding of task requirements. Total task duration was 20 min, with 20 percent ($n = 120$) of presentations including the target letter, which was pseudorandomly distributed throughout the 600 stimuli. For the duration of the task, the examiner sat by the child to monitor performance and compliance.

CPT Variables

Several variables were assessed by the CPT:

1. *Selective attention* was defined in terms of both the total number of correct responses (CORR: Yes-Yes: "Yes" response when target is presented; No-No: "No" response when target is not presented) and the total number of omission errors (OMM: No-Yes: "No" response when target is presented).
2. *Response inhibition/impulsivity* was defined as the total number of commission errors (COM: Yes-No: "Yes" response when no target is presented).
3. *Processing speed* was defined as the mean reaction time (RT) for No-No responses, in keeping with previous studies.
4. *Sustained attention* was assessed by several indices. The standard deviation of reaction time (SDRT) provided a measure of intraindividual variability, an aspect of sustained attention. Lapses in attention (LAP) were defined as two or more consecutive responses from any of the following categories—incorrect response (omission, commission), delayed or "no" response (>1500 ms), or impulsive response (<200 ms). Impulsive responses were only recorded where responses had been provided for the preceding stimulus, to discriminate these data from very late responses. Both the number and length of lapses were considered in analysis. The length of lapses assessed tendencies for children to make multiple sequential "lapse" responses. Time on task (TT) effects were operationalized by dividing the 20-min CPT task into four 5-min blocks and analyzing differences in performance across blocks. TT measured changes in attention with time on task. Sustained attention was assumed to deteriorate with time on task where deficits were present. Distinctions between these attentional constructs were based on previous research that has used the CPT in various groups of children with both normal development and developmental and acquired CNS disorders (Anderson et al., 2000; Anderson & Pentland, 1998; Catroppa & Anderson, 2003; Mirsky, 1996; Rebok et al., 1997).

Statistical Analysis

Group differences for intellectual and demographic variables were examined using analysis of variance (ANOVA). Distributions for CORR, OMM, and LAP were significantly skewed and logarithmic transformations were undertaken. As CORR was negatively skewed, this variable was "reflected" during the logarithmic transformation, resulting in a log scale in which high values represent fewer correct responses. For attention measures divided into 5-min blocks, repeated measures multivariate analysis of covariance (MANCOVA), covarying for age, gender, and IQ, were conducted across groups. Where statistical differences were identified, *post hoc* analyses (Bonferroni's *t* test) were used to determine group differences. Further analyses were conducted to investigate the rates of impairment across groups on attention variables. Due to significant age effects for these variables, analyses were conducted using the following age categories: 8–9 years, 10–11 years, 12–13 years, 14–15 years. The χ^2 analyses were conducted within these age categories and frequencies of impaired (scores $> 90^{\text{th}}$

centile) and unimpaired (scores \leq 90th centile) performances were compared across groups. The relationship of age and IQ with CPT parameters, as well as associations between the CPT parameters, were assessed using Pearson's Correlation Coefficient.

RESULTS

Demographic and IQ Characteristics

There were no group differences for age at testing or SES, although FSIQ differed across groups, $F(4,176) = 9.38$, $p < .001$, with the ALL group demonstrating a lower mean FSIQ than other groups, as illustrated in Table 1.

Group Differences in CPT Scores

Group differences were detected for CORR, $F(4,176) = 8.06$, $p < .001$ (data for CPT variable across groups are provided in Table 2). The ADHD group recorded the poorest results, being significantly less accurate overall than the IDDM ($p < .001$), ALL ($p = .015$), and control ($p < .001$) groups (see Figure 1). The TBI group also scored less well than controls on this measure, although this difference was only marginally significant ($p = .064$).

Similar group differences were found for OMM, $F(4,176) = 9.58$, $p < .001$, see Figure 2. *Post hoc* analyses revealed that the ADHD group recorded more omission errors than the IDDM ($p < .001$) and control ($p < .001$) groups, whereas the TBI group made more omission errors than controls ($p = .003$). The Time \times Group interaction was also significant: Wilks' Lambda = .875, $p = .024$, partial $\eta^2 = .043$. As shown in Figure 2, the likely basis of the interaction was the difference in patterns of performance across blocks for the ADHD group compared with the other groups. Specifically, the ADHD group recorded more OMMs during Block 4 than during Block 1, whereas the other groups showed the reverse pattern.

As shown in Figure 3, analysis of SDRT also revealed a significant group difference: $F(4,176) = 7.61$, $p < .001$. *Post hoc* analyses again demonstrated poorest performance in the ADHD group, which exhibited significantly greater

variations in response speed than the TBI ($p < .001$), IDDM ($p < .001$), ALL ($p < .001$), and control ($p = .001$) groups. The TT effect was significant as well, Wilks' Lambda = .936, $p = .009$, partial $\eta^2 = .064$, indicating increases in SDRT with time on task.

Group differences were also found for LAP number: $F(4,176) = 9.51$, $p < .001$. Consistent with the previous results, the ADHD group recorded significantly more lapses than the TBI ($p = .021$), IDDM ($p < .001$), ALL ($p < .001$), and control ($p < .001$) groups. In addition, as illustrated in Figure 4, children in the ADHD group exhibited longer sequences of lapses in comparison to the other groups, $\chi^2(4) = 32.23$, $p < .001$. Analyses failed to reveal any significant effects for COM or RT.

Impairment Rates Across Groups

The results of χ^2 analyses were consistent with the findings reported above. First, no group differences were identified in rates of impairment for mean RT; however, analysis of impairments in error rates did show some group patterns. The rate of impairment was significantly elevated in the ADHD group in contrast to controls for COM (51.9%), $\chi^2(1) = 4.93$, $p = .026$; CORR (70.4%), $\chi^2(1) = 24.83$, $p < .001$; LAP number (70.4%), $\chi^2(1) = 22.57$, $p < .001$; and OMM (48.1%), $\chi^2(1) = 17.23$, $p < .001$. The rate of impairment in the TBI was significantly elevated relative to controls for CORR (36.6%), $\chi^2(1) = 6.56$, $p = .01$; LAP number (36.6%), $\chi^2(1) = 5.24$, $p = .022$; and OMM (22.0%), $\chi^2(1) = 4.34$, $p < .04$.

Age and IQ Effects

As expected, significant linear relationships were observed between age and CPT parameters, with the correlation coefficients ranging from .310 ($p < .001$) for mean reaction time to .506 for number of lapses. In all cases, performance improved with increasing age. In contrast, IQ correlated weakly with CPT parameters, with the correlation coefficients ranging from .058 ($p > .05$) for COM to .208 ($p = .005$) for SDRT. Given the lack of association between IQ and CPT parameters, all MANCOVAs were repeated,

Table 1. Demographic and IQ characteristics of sample

	ADHD (<i>n</i> = 27)	TBI (<i>n</i> = 41)	IDDM (<i>n</i> = 39)	ALL (<i>n</i> = 31)	Controls (<i>n</i> = 46)
Gender (no. males)	22	29	16	17	24
SES, mean (SD)	4.9 (1.0)	4.80 (.97)	4.75 (1.1)	4.9 (1.0)	4.8 (1.2)
Age at testing, mean (SD)	11.8 (1.8)	11.6 (2.0)	11.6 (2.3)	12.4 (1.7)	11.5 (2.4)
IQ, mean (SD)	102.3 (13.6)	101.3 (14.4)	102.6 (14.2)	90.4 (12.8)*	108.5 (9.0)

Note. SES was determined using the Daniel Scale of Occupational Prestige (1983). IQ is the estimated IQ derived from short form of WISC-III, including Vocabulary, Object Assembly, Similarities, Block Design. The ALL group is significantly different to all other groups for IQ, $p < .001$. IQ, intelligence quotient; ADHD, attention deficit-hyperactivity disorder; TBI, traumatic brain injury; IDDM, insulin-dependent diabetes mellitus; ALL, acute lymphoblastic leukemia; SES, socioeconomic status; WISC-III, Wechsler Intelligence Scale for Children.

Table 2. Adjusted group means on CPT variables

	ADHD (n = 27)	TBI (n = 41)	IDDM (n = 39)	ALL (n = 31)	Controls (n = 46)
	Adjusted mean (95% CI)	Adjusted mean (95% CI)	Adjusted mean (95% CI)	Adjusted mean (95% CI)	Adjusted mean (95% CI)
Total correct ^a # b** c ⁺ d**	1.90 (1.77, 2.03)	1.69 (1.58, 1.80)	1.49 (1.37, 1.61)	1.58 (1.45, 1.72)	1.49 (1.39, 1.59)
Total omissions ^a b** d** e*	1.24 (1.12, 1.37)	1.07 (0.97, 1.17)	0.85 (0.73, 0.96)	0.99 (0.86, 1.12)	0.80 (0.70, 0.90)
Total commissions	31.3 (25.0, 37.5)	30.8 (25.8, 35.8)	24.5 (18.7, 30.2)	27.1 (20.8, 33.4)	24.7 (19.8, 29.5)
Mean RT	650.8 (607.0, 694.5)	584.8 (549.6, 620.0)	589.8 (549.2, 630.5)	602.8 (558.6, 647.1)	612.5 (578.6, 646.4)
SD of RT ^a b** c** d*	238.5 (217.0, 260.0)	174.3 (157.0, 191.6)	172.8 (152.8, 192.7)	166.3 (144.6, 188.1)	182.4 (165.8, 199.0)
Total lapses ^a c ⁺ b** c** d**	1.14 (0.98, 1.30)	0.80 (0.67, 0.93)	0.64 (0.49, 0.79)	0.55 (0.39, 0.72)	0.59 (0.46, 0.71)
					p value
					<.001
					<.001
					.224
					.165
					<.001
					<.001
					Partial η^2
					.155
					.179
					.032
					.036
					.147
					.178

Note. Means adjusted for age, gender, and IQ; ^aLog scale; # High values represent fewer correct responses. Significant *post hoc* results: ^aADHD vs. TBI; ^bADHD vs. IDDM; ^cADHD vs. ALL; ^dADHD vs. Controls; ^eTBI vs. Controls. **p < .001, *p < .01, +p < .05. CPT, Continuous Performance Test; ADHD, attention deficit-hyperactivity disorder; TBI, traumatic brain injury; IDDM, insulin-dependent diabetes mellitus; ALL, acute lymphoblastic leukemia; CI, confidence interval; RT, reaction time.

excluding IQ as a covariate, but this failed to alter any of the statistical conclusions.

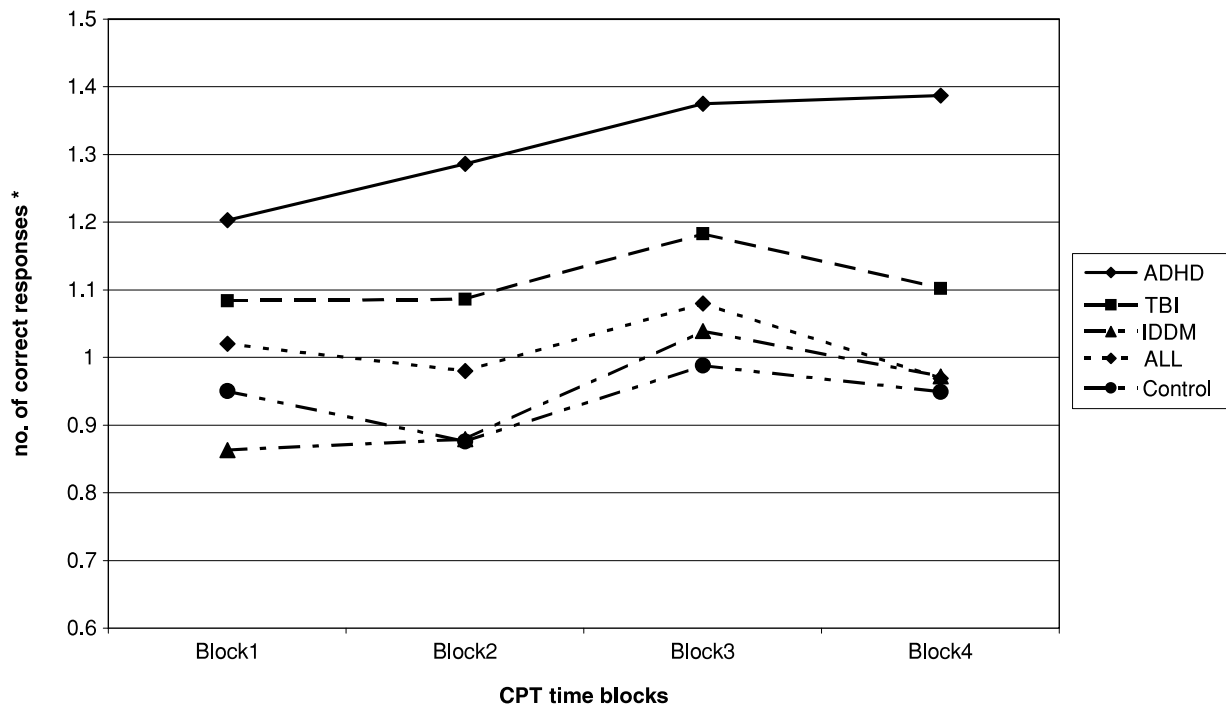
Association between CPT Parameters

For the total sample, lapses of attention correlated with CORR ($r = .894, p < .001$), OMM ($r = .792, p < .001$), COM ($r = .752, p < .001$), and SDRT ($r = .692, p < .001$). As expected, CORR correlated strongly with OMM ($r = .871, p < .001$) and COM ($r = .815, p < .001$). RT was only associated with SDRT ($r = .720, p < .001$).

DISCUSSION

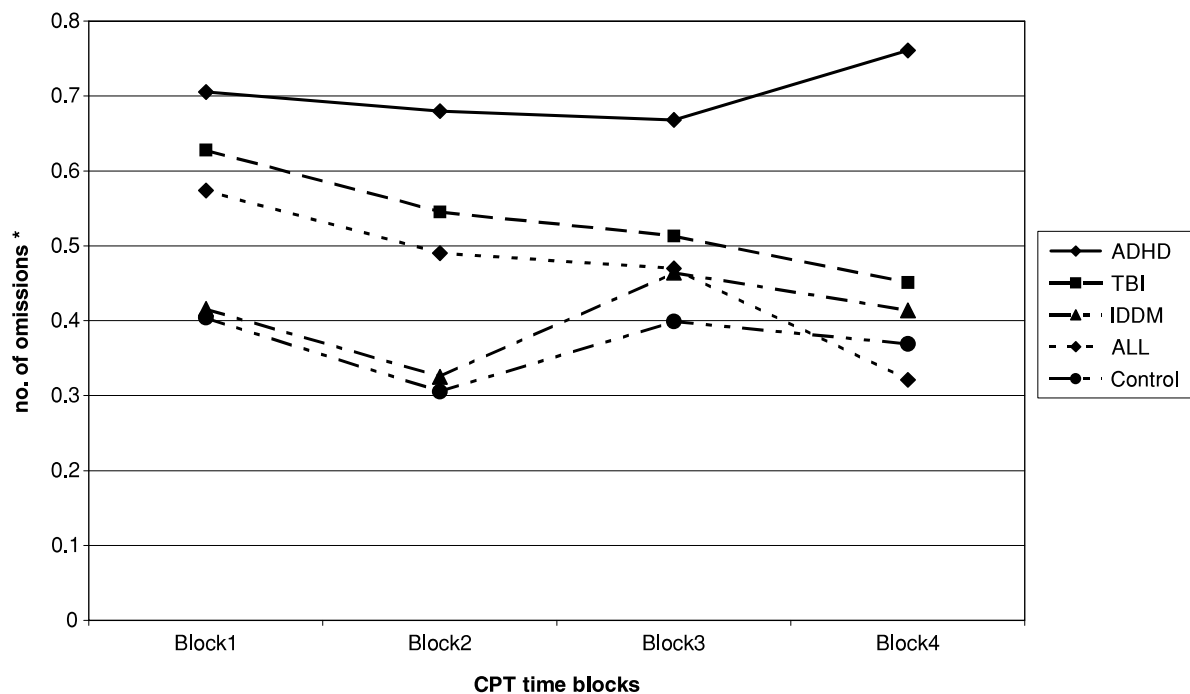
The present study aimed to (1) investigate the clinical efficacy of the CPT by identifying and measuring variables relating to specific components of the attention system (sustained and selective attention, response inhibition, processing speed); and (2) compare performances across developmental and acquired CNS disorders previously found to be associated with attentional impairments. The study used a simultaneous discrimination version of the CPT previously identified as sensitive to the presence to attentional impairment, associated with childhood brain insult (e.g., severe TBI: Anderson et al., 2004b; 2005; Anderson & Pentland, 1998; Catroppa et al., 1999). CPT indices were extended to include variables tapping response variability that were predicted to be sensitive to sustained and selective attention, response inhibition, and processing speed.

The pattern of attentional impairment predicted on the basis of previous cognitive and neuroanatomical research was generally not supported. Children with ADHD performed most poorly on all CPT measures, suggesting that the presence of a developmental attention deficit may impact most significantly on the integrity of the attentional system. When interpreting this finding, it is important to note that the ADHD group, due to its diagnostic characteristics, was recruited based on evidence of impaired attention (albeit based on behavioral rather than cognitive indices); therefore, our findings are not unexpected. Children with a history of moderate TBI demonstrated mild attention problems. Compared with controls, the latter group had a higher rate of impairment in selective and sustained attention and showed a nonsignificant trend for poorer response inhibition. Children with ALL and IDDM were undifferentiated from controls, despite previous reports of clinically significant attentional problems (e.g., Anderson et al., 2004a, Cousens et al., 1988; Northam et al., 1998), suggesting that the CPT may not be sensitive to all aspects of the attentional system. For example, higher-order attentional domains, such as attentional shifting and divided attention are not measured by the CPT paradigm, and may be more vulnerable in these samples. Alternatively, the lack of significant impairment detected in these groups may be due to small sample sizes of the clinical groups or to the exclusion of children with more severe brain insult. In support of this latter possibility, researchers examining the impact of childhood brain



* Reflected log scale - High values represent fewer correct responses.

Fig. 1. Mean number of correct responses for each time block across groups. Asterisk indicates reflected log scale, for which high values represent fewer correct responses. ADHD, attention deficit-hyperactivity disorder; TBI, traumatic brain injury; IDDM, insulin-dependent diabetes mellitus; ALL, acute lymphoblastic leukemia.



* Log scale.

Fig. 2. Mean number of omission errors for each time block across groups. ADHD, attention deficit-hyperactivity disorder; TBI, traumatic brain injury; IDDM, insulin-dependent diabetes mellitus; ALL, acute lymphoblastic leukemia.

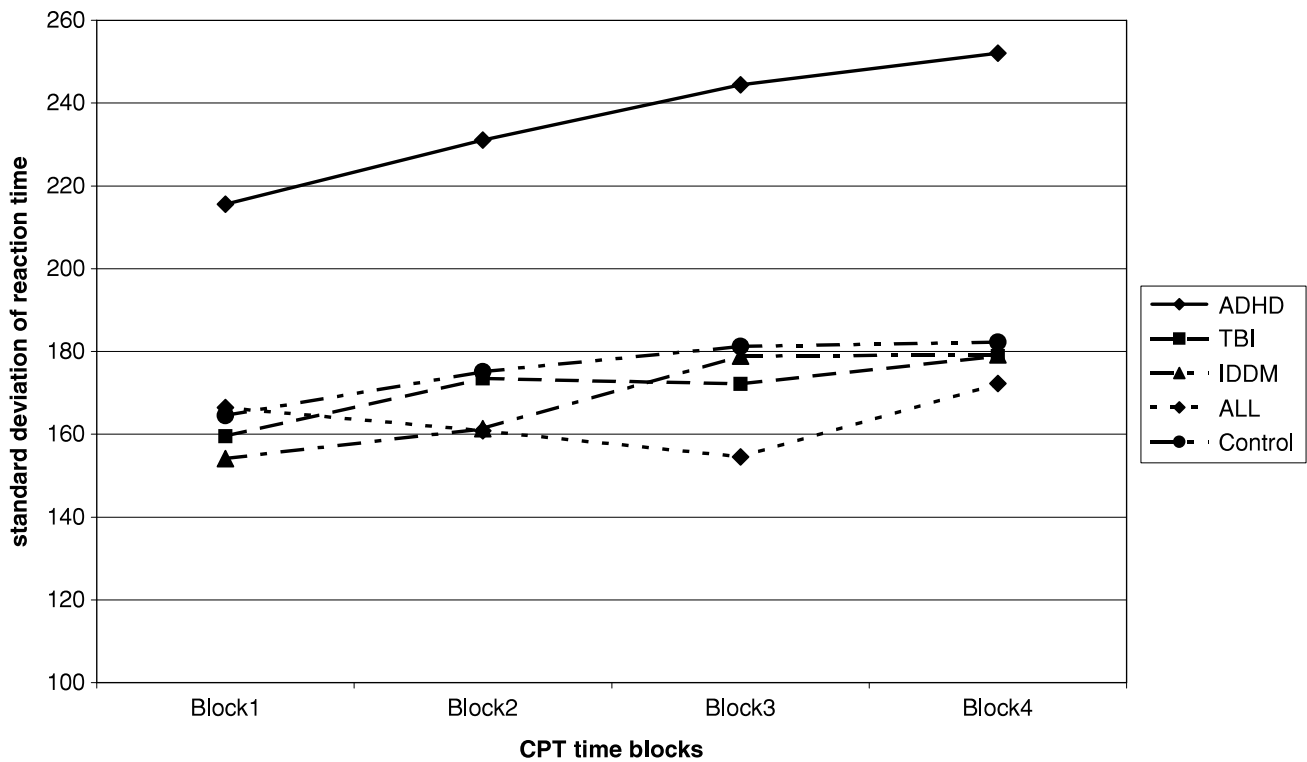


Fig. 3. Standard deviation of reaction times for each time block across groups. ADHD, attention deficit–hyperactivity disorder; TBI, traumatic brain injury; IDDM, insulin-dependent diabetes mellitus; ALL, acute lymphoblastic leukemia.

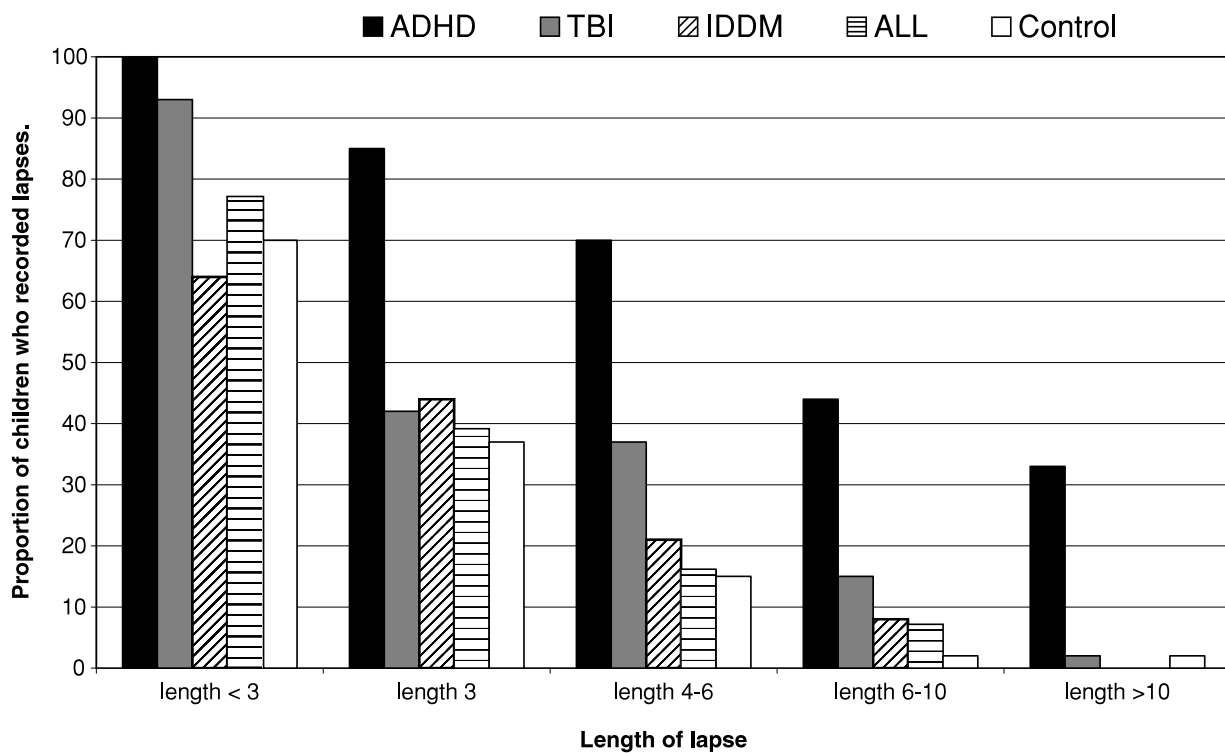


Fig. 4. Length of attentional lapses across groups. ADHD, attention deficit–hyperactivity disorder; TBI, traumatic brain injury; IDDM, insulin-dependent diabetes mellitus; ALL, acute lymphoblastic leukemia.

insult have noted that group differences in cognitive function are not universal but may be attributed to the subset of children usually with more severe injury or the presence of medical/neurological complications (e.g., Anderson et al., 2000; Ponsford et al., 1997; Taylor et al., 1993). Furthermore, studies examining the impact of more mild/moderate insults often fail to identify significant sequelae (Anderson et al., 2000, 2001; Taylor et al., 1993; Yeates et al., 1995).

Attention Deficit–Hyperactivity Disorder

The results for the ADHD group are particularly striking and demonstrated a profile consistent with global and substantive attentional deficits. Of note, this group performed worse than all other groups, even the ALL group, which recorded a significantly lower FSIQ. They were clearly impaired in selective attention (CORR, COM) and the capacity to sustain efficient performance over time (frequency and length LAP, SDRT). They also demonstrated gradually deteriorating response accuracy over time. These results are in keeping with clinical observations and behavioral symptoms frequently described for children with ADHD. Of interest, specific impairments in response inhibition were not evident. Also of note is this group's average intellectual ability. In the context of significant and chronic attention deficits, intact intellectual abilities may be somewhat surprising and support the observation of a weak association between the components of attention addressed in this study and the development of cognitive abilities, as has been observed in previous research (e.g., Wu et al., 2002).

Traumatic Brain Injury

Children with moderate TBI were not consistently different on CPT measures from the control, IDDM, or ALL groups, despite the likelihood of dysfunction within cerebral regions involved in the attentional system. It was only for OMM that a significant group difference was detected in analysis of continuous measures from the CPT, with the TBI group performing similarly to the ADHD group but different from controls on this variable. The TBI group also had higher rates of impairment in this measure than controls, with 22% of the TBI group recording impaired performances. Additional differences included higher rates of lapses in attention (36% of children within the impaired range) and longer duration lapses in the TBI group relative to controls. Such attentional fluctuations occurred relatively consistently across TT, with mean performances across sequential time blocks providing no evidence for the predicted gradual deterioration in performance associated with impaired sustained attention.

A final observation for this group relates to processing speed. Whereas previous research with adults has documented slowed response after TBI (Ponsford & Kinsella, 1992), findings for children have been inconsistent. In the current study, the TBI and control groups did not differ

significantly in RT, suggesting that, for the relatively simple responses required on the CPT task, children with a history of moderate TBI do not demonstrate slower responses.

Acute Lymphoblastic Leukemia and Insulin-Dependent Diabetes Type 1

These groups performed similarly to controls for measures of selective attention, response inhibition, processing speed, and sustained attention, with no evidence of fluctuating or deteriorating performances. It may be that attentional deficits in these groups are associated with the more “executive” aspects of attention not tapped by the CPT, for example shifting and dividing attention, or with response modalities other than those required by the CPT (e.g., language, visuo-motor domains). Further research is needed to investigate these possibilities. Of note, such deficiencies have been reported for children with ALL (Anderson et al., 2000, 2004a) treated with cranial irradiation and may explain the unexpectedly low intellectual scores recorded by this group. It is also likely that the exclusion of children with frank neurological sequelae (e.g., seizures, neurological abnormalities) impacted on group results. Previous studies documenting attentional impairments have incorporated all levels of illness severity in their samples (Brouwers et al., 1985; Hagen et al., 1990; Holmes, 1990; Kingma et al., 1993; Northam et al., 2001; Paakko et al., 1992). The current results do suggest that, where children have experienced episodes of moderate brain trauma (low levels of cranial radiation, hyperglycemia), aspects of the attentional system may be more intact.

Several study limitations must be considered when interpreting findings. First, although sensitive to attentional impairment after more severe pathology (Anderson et al., 2004a, 2005; Anderson & Pentland, 1998; Catroppa & Anderson, 2003), the simultaneous discrimination, forced-choice CPT version used in this study may have minimized sensitivity to detect impulsive responses. Furthermore, CPT paradigms with an emphasis on lower-order skills such as selective and sustained attention have limited capacity to comprehensively evaluate attentional skills in a manner that allows translation to day-to-day function. Newly developed attentional measures (e.g. Manly et al., 1999), which attempt to assess the attentional system more exhaustively, may be considered in future research addressing these issues. To gain a more complete assessment of attentional profiles, and thus contribute to theoretical models, additional measures need to be incorporated into test protocols. Inclusion of tasks tapping auditory modalities and higher level attentional components, such as shifting and divided attention, should be considered. Future studies documenting both structural and functional brain activity, as well as timing of onset of brain dysfunction, may provide critical information with respect to the bases of attentional impairments in children.

Recruiting samples sustaining insults of similar severity across different childhood brain disorders is problematic.

We are confident, based on previous knowledge of these samples, that we have assembled groups characterized by moderate brain insults. However, severity determinations are not precise, and future research that includes brain imaging data may be better able to characterize the degree of brain insult. Furthermore, within the ADHD group, there are potential subgroup differences in attentional function that have not been examined in this study, due to small sample size. Classification into subtypes such as inattentive and hyperactive/impulsive may provide further evidence relevant for clinical practice. Finally, our decision to exclude children with a diagnosis of ADHD from the TBI, IDDM, and ALL groups may have impacted on study findings, and future research dividing these groups according to the presence or absence of ADHD symptoms may shed additional light on current findings.

The results of this study have several clinical implications. First, findings demonstrate that the CPT can effectively identify significant attentional difficulties, particularly in the domains of sustained and selective attention. Second, attentional problems, at least as measured by the CPT task, do not appear to characterize all children with brain-related conditions. Furthermore, findings suggest that the CPT paradigm (at least that used in the current study) does not provide an exhaustive measure of the integrity of the attentional system in children. For example, our failure to observe deficits in response inhibition, even in children with ADHD, suggests that this ability construct may be inadequately assessed. Furthermore, lack of evidence of group differences in response speed was surprising and may indicate that the nature of processing speed deficits in these groups is not captured by the CPT's simple RT measures, and needs to be evaluated by means of tasks with a higher cognitive loading (e.g., Coding: WISC-III; Symbol Search: TEA-Ch). Importantly, the CPT does not purport to measure higher-order attention domains such as shifting and divided attention. Thus, our findings suggest that, where attention deficits are suspected, although the CPT may be useful, other measures need to be incorporated into the clinician's test protocol to gain a full profile of attentional strengths and weaknesses.

In summary, the present study used a traditional clinical measure of attention, the CPT, to examine the attention profiles of four groups of children with a history of disorders involving the CNS, as well as a healthy control sample. The CPT was found to be effective in differentiating children with ADHD from other groups under investigation, with severe and global attentional difficulties associated with this diagnosis. Children with moderate TBI were found to exhibit specific selective attention deficits, with a trend to poorer performance in other attentional domains. Other conditions with known CNS involvement (ALL, IDDM) showed no such problems, suggesting that the attentional systems examined in the present study may not be consistently compromised, at least in more moderate forms of these conditions, or that the CPT may be insensitive to such effects.

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REFERENCES

- Aman, C., Roberts, R., & Pennington, B. (1998). A neuropsychological examination of the underlying deficit in attention deficit hyperactivity disorder: Frontal lobe versus right parietal lobe theories. *Developmental Neuropsychology*, *34*, 956–969.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders*. (4th ed.). Washington, DC: American Psychiatric Association Press.
- Anderson, V., Catroppa, C., Morse, S., Haritou, F., & Rosenfeld, J. (2005). Attentional and processing skills following traumatic brain injury in early childhood. *Brain Injury*, *19*, 699–710.
- Anderson, V., Catroppa, C., Morse, S., Haritou, F., & Rosenfeld, J. (2001). Outcome from mild head injury in young children: A prospective study. *Journal of Clinical and Experimental Neuropsychology*, *23*, 705–717.
- Anderson, V., Godber, T., Smibert, E., Weiskop, S., & Ekert, H. (2004a). Impairments of attention following treatment with cranial radiation and chemotherapy with children. *Journal of Clinical and Experimental Neuropsychology*, *26*, 684–697.
- Anderson, V., Godber, T., Smibert, E., Weiskop, S., & Ekert, H. (2000). Cognitive and academic outcome following cranial irradiation and chemotherapy in children: A longitudinal study. *British Journal of Cancer*, *82*, 255–262.
- Anderson, V. & Moore, C. (1995). Age at injury as a predictor of outcome following pediatric head injury. *Child Neuropsychology*, *1*, 187–202.
- Anderson, V., Morse, S., Catroppa, C., Haritou, F., & Rosenfeld, J. (2004b). Thirty-month outcome from early childhood head injury: A prospective analysis of neurobehavioral recovery. *Brain*, *127*, 2608–2620.
- Anderson, V. & Pentland, L. (1998). Residual attention deficits following childhood head injury: Implications for ongoing development. *Neuropsychological Rehabilitation*, *8*, 283–300.
- Aronson, M., Hagberg, B., & Gillberg, C. (1997). Attention deficits and autistic spectrum problems in children exposed to alcohol during gestation: A follow-up study. *Developmental Medicine and Child Neurology*, *39*, 583–587.
- August, G.J. & Garfinkel, B.D. (1990). Comorbidity of AD/HD and reading disability among clinic referred children. *Journal of Abnormal Child Psychology*, *18*, 29–45.
- Barkley, R.A. (1999). Theories of attention-deficit hyperactivity disorder. In H. Quay & A. Hogan (Eds.), *Handbook of disruptive behavior disorders* (pp. 295–313). New York: Kluwer Academic/Plenum.
- Baron-Cohen, S. & Moriarty, J. (1995). Developmental dysexecutive syndrome: Does it exist? A neuropsychological perspective. In M. Robertson & E. Valsamma (Eds.), *Movement and allied disorder in childhood* (pp. 306–316). Chichester, UK: Wiley.
- Brouwers, P., Riccardi, R., Fedio, R., & Poplack, D. (1985). Long-term neuropsychological sequelae of childhood leukemia: Cor-

- relation with CT brain scan abnormalities. *Journal of Pediatrics*, *106*, 723–728.
- Casey, B., Gordon, C., Mannheim, G., & Rumsey, J. (1993). Dysfunctional attention in autistic savants. *Journal of Clinical and Experimental Neuropsychology*, *15*, 933–946.
- Catroppa, C. & Anderson, V. (2003). Children's attentional skills two years post-TBI. *Developmental Neuropsychology*, *23*, 359–373.
- Catroppa, C. & Anderson, V. (1999). Attention in the acute phase following pediatric head injury. *Child Neuropsychology*, *5*, 251–265.
- Catroppa, C., Anderson, V., & Stargatt, R. (1999). A prospective analysis of the recovery of attention following pediatric head injury. *Journal of the International Neuropsychological Society*, *5*, 48–57.
- Cooley, E.L. & Morris, R.D. (1990). Attention in children: A neuropsychologically based model for assessment. *Developmental Neuropsychology*, *6*, 239–274.
- Cousens, P., Ungerer, J.A., Crawford, J.A., & Stevens, M. (1988). Cognitive effects of childhood leukemia therapy: A case for four specific deficits. *Journal of Pediatric Psychology*, *16*, 475–488.
- Cowan, N. (1997). *Attention and memory*. New York: Oxford University Press.
- Daniel, A. (1983). *Power, privilege and prestige: Occupations in Australia*. Melbourne: Longman Cheshire.
- Dennis, M. (1989). Language and the young damaged brain. In T. Boll & B.K. Bryant (Eds.), *Clinical neuropsychology and brain function: Research, measurement and practice* (pp. 85–124). Washington, DC: American Psychological Association.
- Dennis, M., Wilkinson, M., Koski, L., & Humphreys, R.P. (1995). Attention deficits in the long term after childhood head injury. In S.H. Broman, & M.E. Michel (Eds.), *Traumatic head injury in children*. New York: Oxford University Press.
- Ewing-Cobbs, L., Prasad, M., Fletcher, J.M., Levin, H.S., Miner, E., & Eisenberg, H. (1998). Attention after pediatric traumatic brain injury: A multidimensional assessment. *Child Neuropsychology*, *4*, 35–48.
- Goff, J.R., Anderson, H.R., & Cooper, P.F. (1980). Distractibility and memory deficits in long-term survivors of acute lymphoblastic leukemia. *Developmental and Behavioral Pediatrics*, *1*, 153–163.
- Goyette, C., Conners, C., & Ulrich, R. (1978). Normative data on revised Conners Parent and Teacher rating Scales. *Journal of Abnormal Child Psychology*, *6*, 221–236.
- Hagen, J.W., Barclay, C.R., Anderson, B.J., Feeman, D.J., Segal, S., Bacon, G., & Goldstein, G.W. (1990). Intellectual functioning and strategy use in children with insulin-dependent diabetes mellitus. *Child Development*, *61*, 1714–1727.
- Heaton, S., Reader, S., Preston, A., Fennell, E., Puyana, O., Gill, N., & Johnson, J. (2001). The Test of Everyday Attention for Children (TEA-Ch): Patterns of performance in children with ADHD and clinical controls. *Child Neuropsychology*, *7*, 251–264.
- Holmes, C.S. (1990). Neuropsychological sequelae of acute and chronic blood glucose disruption in adults with insulin-dependent diabetes. In C. Holmes (Ed.), *Neuropsychological and behavioral aspects of diabetes* (pp. 122–154). New York: Springer-Verlag.
- Hooks, K., Milich, R., & Lorch, E. (1994). Sustained and selective attention in boys with attention deficit hyperactivity disorder. *Journal of Child Clinical Psychology*, *23*, 69–77.
- James, W. (1890). *The principles of psychology*. New York: Henry Holt.
- Johannes, S., Wieringa, B., Mantey, M., Nagey, W., Rada, D., Muller-Vahl, K., Wmrich, H., Dengler, R., Munte, T., & Dietrich, D. (2001). Altered inhibition of motor responses in Tourette syndrome and obsessive compulsive disorder. *Acta Neurologica Scandinavica*, *104*, 36–43.
- Kaufmann, P., Fletcher, J., Levin, H., Miner, M., & Ewing-Cobbs, L. (1993). Attention disturbance after pediatric closed head injury. *Journal of Child Neurology*, *8*, 348–353.
- Kingma, A., Mooyart, E.L., Kamps, W.A., Nieuwenhuizen, P., & Wilminck, J.T. (1993). Magnetic resonance imaging of the brain and neuropsychological evaluation in children treated for acute lymphoblastic leukemia at a young age. *American Journal of Pediatric Hematology and Oncology*, *15*, 231–238.
- Klin, A., Sparrow, S., Volkman, F., Cicetti, D., & Rourke, B. (1995). Asperger syndrome. In B. Rourke (Ed.), *Syndrome of non-verbal learning disabilities* (pp. 93–119). New York: Guilford.
- Konrad, K., Gauggel, S., Manz, A., & Scholl, M. (2000). Inhibitory control in children with traumatic brain injury (TBI) and children with attention deficit/hyperactivity disorder (ADHD). *Brain Injury*, *14*, 859–875.
- Lajoie, G., Anderson, V., Tucker, A., Robertson, I., & Manly, T. (2005). Effects of methylphenidate on attention skills in children with attention deficit/hyperactivity disorder. *Brain Impairment*, *6*, 21–32.
- Lane, D.M. (1978). Developmental changes in attention deployment skills. *Journal of Experimental Child Psychology*, *28*, 16–29.
- Lane, D.M. & Pearson, D.A. (1982). The development of selective attention. *Merrill-Palmer Quarterly*, *28*, 317–337.
- Levy, F. & Hobbes, G. (2000). Discrimination of attention deficit hyperactivity disorder by the Continuous Performance Test. *Journal of Paediatrics and Child Health*, *33*, 384–387.
- McCall, A.L. & Foglewicz, D.P. (1997). How does diabetes mellitus produce brain dysfunction? *Diabetes Spectrum*, *10*, 25–32.
- McKay, K., Halperin, J., Schwartz, S., & Sharma, V. (1994). Developmental analysis of three aspects of information processing: Sustained attention, selective attention, and response organization. *Developmental Neuropsychology*, *10*, 121–132.
- Manly, T., Anderson, V., Nimmo-Smith, I., Turner, A., Watson, P., & Robertson, I. (2001). The differential assessment of children's attention: The Test of Everyday Attention for Children (TEA-Ch): Normative sample and ADHD performance. *Journal of Child Psychology and Psychiatry*, *42*, 1065–1087.
- Manly, T., Robertson, I.H., Anderson, C., & Nimmo-Smith, I. (1999). *TEA-CH: The Test of Everyday Attention for Children*. Bury St Edmunds, UK: Thames Valley Test Co.
- Micallef, S., Anderson, J., Anderson, V., Robertson, I., & Manly, T. (2001). Sustained and selective attention in children with Attention Deficit/Hyperactivity Disorder and specific learning disabilities. *Clinical Neuropsychological Assessment*, *2*, 1–23.
- Mirsky, A.F. (1996). Disorders of attention: A neuropsychological perspective. In G. Lyon & N. Krasnegor (Eds.), *Attention, memory and executive function* (pp. 71–95). Baltimore: Paul H. Brookes Publishing.
- Mirsky, A.F., Anthony, B.J., Duncan, C.C., Ahearn, M.B., & Kellam, D.G. (1991). Analysis of the elements of attention: A neuropsychological approach. *Neuropsychology Review*, *2*, 109–145.
- Northam, E., Anderson, P., Jacobs, R., Hughes, M., Warne, G., & Werther, G. (2001). Neuropsychological profiles of children

- with type 1 diabetes 6 years after disease onset. *Diabetes Care*, 24, 1541–1546.
- Northam, E., Anderson, P., Werther, G., Warne, G., Adler, R., & Andrewes, D. (1998). Neuropsychological complications of insulin dependent diabetes in children two years after disease onset. *Diabetes Care*, 21, 379–384.
- Paakko, E., Vainionpaa, L., Lanning, M., Laitinen, J., & Pyhtinen, J. (1992). White matter changes in children treated for acute lymphoblastic leukemia. *Cancer*, 70, 2728–2733.
- Ponsford, J. & Kinsella, G. (1992). Attentional deficits following closed head injury. *Journal of Clinical and Experimental Neuropsychology*, 14, 822–838.
- Ponsford, J., Willmott, C., Rothwell, A., Cameron, P., Kelly, A., Ayton, G., Curran, C., & Nelms, R. (1997). Cognitive and behavioral outcome following mild traumatic brain injury in children. *Journal of the International Neuropsychological Society*, 3, 225.
- Posner, M. (1984). Selective attention and the storage of information. In G. Lynch, J. McGaugh, & N. Weinberger (Eds.), *Neurobiology of learning and memory*. New York: Guilford Press.
- Posner, M.I. (1978). *Chronometric exploration of mind*. Hillsdale, NJ: Erlbaum.
- Rebok, G., Smith, C., Pascualvaca, D., Mirsky, A., Anthony, B., & Kellam, S. (1997). Developmental changes in attentional performance in urban children from eight to thirteen years. *Child Neuropsychology*, 3, 47–60.
- Reeve, W. & Schandler, S. (2001). Frontal lobe functioning in adolescents with attention deficit hyperactivity disorder. *Adolescence*, 36, 749–765.
- Rosvold, H.E., Mirsky, A.F., Sarason, I., Bransome, E.D., & Beck, L.H. (1956). A continuous performance test of brain damage. *Journal of Consulting Psychology*, 20, 343–350.
- Rowe, J.K. & Rowe, K.S. (1995). *Rowe behavioural rating inventory profiles user's guide*. Centre for Applied Educational Research and Department of Paediatrics. Victoria, Australia: The University of Melbourne.
- Rubia, K., Overmeyer, S., Taylor, R., Brammer, M., Williams, S., & Simmons, A. (1999). Hypofrontality in attention deficit hyperactivity disorder during higher-order motor control: A study with functional MRI. *American Journal of Psychiatry*, 156, 891–896.
- Ruff, H. & Rothbart, M. (1996). *Attention in early development: Themes and variations*. New York: Oxford University Press.
- Sattler, J. (1988). *Assessment of children's intelligence and special abilities* (3rd ed.). Boston: Allen & Bacon.
- Schachar, R., Levin, H.S., Max, J., Purvis, K., & Chen, S. (2004). Attention deficit hyperactivity disorder symptoms and response inhibition after closed head injury in children: Do preinjury behavior and injury severity predict outcome? *Developmental Neuropsychology*, 25, 179–198.
- Seidel, W. & Joschko, M. (1990). Evidence of difficulties in sustained attention in children with ADHD. *Journal of Abnormal Child Psychology*, 18, 217–229.
- Semrud-Clikeman, M., Steingard, R., Filipek, P., Biederman, J., Bekken, K., & Renshaw, P. (2000). Using MRI to examine brain-behavior relationships in males with attention deficit hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 477–484.
- Shallice, T., Marzocchi, G.M., Coser, S., Del Savio, M., Meuter, R.F., & Rumiati, R.I. (2002). Executive function profile of children with ADHD. *Developmental Neuropsychology*, 21, 43–71.
- Shepp, B.E., Barrett, S.E., & Kolbet, L.I. (1987). The development of selective attention: Holistic perception versus resource allocation. *Journal of Experimental Child Psychology*, 43, 159–180.
- Shiffrin, R.M. (1988). Attention. In Atkinson, R.C., Herrnstein, R.J., Lindzey, G., & Luce, R.D. (Eds.), *Stevens' Handbook of Experimental Psychology, 2nd Edition*, pp. 739–811. New York: Wiley.
- Sowell, E., Thompson, P., Welcome, S., Henkenius, A., Toga, A., & Petresen, B. (2003). Cortical abnormalities in children and adolescents with attention deficit/hyperactivity disorder. *Lancet*, 362, 1699–1707.
- Stuss, D.T., Shallice, T., Alexander, M., & Picton, T. (1995). A multidisciplinary approach to anterior attentional functions. *Annals of the New York Academy of Science*, 769, 191–211.
- Tallroth, G., Ryding, E., & Agardh, C. (1992). Regional cerebral blood flow in normal man during insulin-induced hypoglycemia and in the recovery period following glucose infusion. *Metabolism*, 41, 717–721.
- Taylor, H., Barry, C., & Schatschneider, C. (1993). School-age consequences of *Haemophilus influenzae Type B* meningitis. *Journal of Clinical Child Psychology*, 22, 196–206.
- Thatcher, R.W. (1991). Maturation of the human frontal lobes. Physiological evidence for staging. *Developmental Neuropsychology*, 7, 397–419.
- van der Meere, J.J. & Sergeant, J.A. (1988). Focused attention in pervasively hyperactive children. *Journal of Abnormal Child Psychology*, 16, 627–639.
- van Zomer, A.H. & Brouwer, W. (1994). *Clinical neuropsychology of attention*. New York: Oxford University Press.
- Wainwright-Sharp, J. & Bryson, S. (1993). Visual orienting deficits in high functioning people with autism. *Journal of Autism and Developmental Disorders*, 23, 1–13.
- Waters, K.D. (1992). A randomized clinical trial of modified BFM therapy versus modified high dose asparaginase therapy in childhood acute lymphoblastic leukemia. *Medical Paediatric Oncology*, 20, abstract 83.
- Wechsler, D. (1991). *Manual for the Wechsler Intelligence Test for Children—Third edition (WISC-III)*. San Antonio, TX: Psychological Corporation.
- Wilkinson, G.S. (1993). *Wide Range Achievement Test Administration Manual* (1993 ed.). Delaware, MD: Wide Range Inc.
- Willmott, C., Anderson, V., & Anderson, P. (2000). Attention following pediatric head injury: A developmental perspective. *Developmental Neuropsychology*, 17, 361–379.
- Wu, K., Anderson, V., & Castiello, U. (2002). Neuropsychological evaluation of deficits in executive functioning for ADHD children with or without LD. *Developmental Neuropsychology*, 22, 501–531.
- Yeates, K., Blumstein, E., Patterson, C., & Delis, D. (1995). Verbal learning and memory following pediatric closed head injury. *Journal of the International Neuropsychological Society*, 1, 78–87.
- Yeates, K. & Bornstein, R. (1994). Attention deficit disorder and neuropsychological functioning in children with Tourette's syndrome. *Neuropsychology*, 8, 65–74.