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Executive function and attention deficit hyperactivity disorder: stimulant medication and better executive function performance in children

S. KEMPTON, A. VANCE,[1] P. MARUFF, E. LUK, J. COSTIN AND C. PANTELIS

From the Department of Psychological Medicine, Monash University; Maroondah Hospital Child and Adolescent Mental Health Service; Mental Health Research Institute of Victoria; Department of Psychiatry, University of Melbourne; and Swinburne University, Victoria, Australia

ABSTRACT

Background. Executive function deficits have been reported repeatedly in children with Attention Deficit Hyperactivity Disorder (ADHD). Stimulant medication has been shown to be effective in improving cognitive performance on most executive function tasks, but neuropsychological tests of executive function in this population have yielded inconsistent results. Methodological limitations may explain these inconsistencies. This study aimed to measure executive function in medicated and non-medicated children with ADHD by using a computerized battery, the Cambridge Neuropsychological Test Automated Battery (CANTAB), which is sensitive to executive function deficits in older patients with frontostriatal neurological impairments.

Methods. Executive function was assessed in 30 children with ADHD: 15 were stimulant medication naïve and 15 were treated with stimulant medication. These two groups were compared to 15 age, sex and IQ matched controls.

Results. The unmedicated children with ADHD displayed specific cognitive impairments on executive function tasks of spatial short-term memory, spatial working memory, set-shifting ability and planning ability. Impairments were also seen on spatial recognition memory and delayed matching to sample, while pattern recognition memory remained intact. The medicated children with ADHD were not impaired on any of the above executive function tasks except for deficits in spatial recognition memory.

Conclusions. ADHD is associated with deficits in executive function. Stimulant medication is associated with better executive function performance. Prospective follow-up studies are required to examine these effects.

INTRODUCTION

Attention Deficit Hyperactivity Disorder (ADHD) describes a heterogeneous group of children believed to have developmentally inappropriate levels of inattention, impulsivity and overactivity (American Psychiatric Association, 1994). These symptoms are often observed early in life, are pervasive across many situations and are usually chronic. The frontal lobe and its subcortical connections are consistently implicated in neuropsychiatric models of the abnormal behaviour in ADHD (Luria, 1973; Becker *et al.* 1987; Welsh *et al.* 1991). This is because qualitative similarities exist between the behaviour of children with ADHD and that of animals and adult humans with lesions of the frontal lobe (Mattes, 1980). Furthermore, children with ADHD commonly show impairments on tests of executive function (Pennington & Ozonoff, 1996; Tannock, 1998). Executive function describes cognitive processes through which performance is optimized in situations requiring

[1] Address for correspondence: Dr Alasdair Vance, Maroondah Hospital Child and Adolescent Mental Health Service, 21 Ware Crescent, Ringwood East, Victoria 3135, Australia.

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the operation of a number of cognitive processes (Baddeley, 1986). It is required when effective new plans of action have been formulated and appropriate sequences of actions must be selected and scheduled. Components may include the enhancement of information 'on-line' (Goldman-Rakic, 1987), the organization of attentional resources (Shallice, 1982), the inhibition of inappropriate responses (Goldman-Rakic, 1987) and the monitoring of behaviour with respect to the affective or motivational state (Damasio, 1994). These different aspects of executive function have been linked to the functioning of the prefrontal cortex and its connections with the parietal and temporal lobe structures, the limbic structures such as the amygdala and the hippocampus, and the striatum.

Although neuropsychological studies of ADHD consistently find impairments on tests of executive function, there is still debate about the precise nature of such impairments (Pennington & Ozonoff, 1996). For example, some studies using the Wisconsin Card Sorting Test (WCST) have reported deficits in attentional set shifting in children with ADHD (Chelune *et al*. 1986; Gorenstein *et al*. 1989) while others have found no such impairments (Loge *et al*. 1990; Grodzinsky & Diamond, 1992). In addition, while studies of ADHD report poor performance on measures of impulsivity or response inhibition on the Continuous Performance Test (CPT) (Corkum & Siegel, 1993), on the Go-No-Go test (Trommer *et al*. 1988; Shue & Douglas, 1992) and on time slowness for Trails B (Boucugnani & Jones, 1989; Gorenstein *et al*. 1989; Shue & Douglas, 1992), others find no impairments on these tests (Grodzinsky & Diamond, 1992; McGee *et al*. 1989). Furthermore, some researchers have reported impairments of visuospatial processing (Robbins & Sahakian, 1983; Grodzinsky & Diamond, 1992) while others have found no impairments in these functions (McGee *et al*. 1989; Korkman & Pesonen, 1994). Finally, two studies have found impairments in planning ability, measured with the Tower of Hanoi task and the Wisconsin Card Sorting Test (WCST) (Pennington *et al*. 1993; Weyandt & Willis, 1994).

The complex nature of executive function impairments in children with ADHD is also evident when stimulant medication treatment is considered. All neurobiological theories of ADHD emphasize brain catecholamine function because of the established clinical response to stimulant medication of children with ADHD (Barkley, 1994; Heilman *et al*. 1991; Gualtieri & Hicks, 1985). However, psychopharmacological studies of both normal humans and animals find that stimulant medication itself can impair executive function (Dyme *et al*. 1982). For example, Robbins & Sahakian (1979) have proposed that stimulant medication may impair executive function by inducing 'over-focusing and perseveration'. However, in ADHD, stimulant medication has been found to improve performance on tests of executive function, especially when they are highly structured measures of attention or vigilance such as reaction time or continuous performance tests (Rappoport *et al*. 1980; Solanto & Conners, 1982). In addition, methylphenidate can increase the flexibility of performance and reduce perseverative responses on the WCST in ADHD (Douglas *et al*. 1986; Everitt *et al*. 1991). Furthermore, improvement in performance on tests of executive function can occur in a linear dose-response fashion (Douglas *et al*. 1988; Tannock *et al*. 1995). These paradoxical findings suggest that children with ADHD may require 'over-focusing and perseveration' to perform within normal limits on tests which require sustained and organized effort (Douglas & Parry, 1983).

Functional neuroimaging studies of children with ADHD also support the involvement of frontostriatal systems. For example studies of regional cerebral blood flow find that striatal hypoperfusion occurs centrally in the frontal lobes relative to normal controls and this hypoperfusion is corrected with stimulant medication (Lou *et al*. 1984, 1989). Decreased activity in frontal brain areas in ADHD children, compared with controls, during performance of several cognitive tasks has also been observed using brain electrical mapping method (BEAM) (Satterfield, 1986). Structural neuroimaging studies of ADHD using magnetic resonance imaging (MRI) have identified an increase in the size of the caudate nucleus and an abnormal asymmetry in these volumes in boys with ADHD compared with normal controls (Castellanos *et*

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al. 1994). The caudate nucleus has important afferent and efferent connections with the frontal lobes.

Taken together, these findings do implicate frontal and striatal brain areas in the pathophysiology of ADHD. In this context, the equivocal nature of the findings from neuropsychological studies is unusual. However, this may have occurred because a number of neuropsychological studies of ADHD have used tests of executive function that have been validated in adult humans with focal brain lesions (e.g. WCST) and may, therefore, have limited validity for use in children because of their complex and multifactorial nature. Secondly, the most common task used to investigate ADHD, the CPT, has limited neuropsychological validity as a measure of executive function (Maruff & Currie, 1996). Thirdly, the relationship between performance on many tests of executive function and the normal maturation of the central nervous system is not well understood (Diamond, 1990). Therefore, impaired performance on some tests of executive function in ADHD may merely reflect a normal but immature central nervous system rather than any ADHD-related pathology. To overcome such problems, the investigation of executive function in ADHD should be conducted both in medication naïve and medicated children. A carefully matched control group is also necessary. Of equal importance, the neuropsychological tests selected should: (1) have an established reliability and validity for measuring executive function; (2) have a well understood developmental trajectory in normal children; and (3) have a demonstrated sensitivity to catecholaminergic medications.

The aim of the current study was, therefore, to investigate executive function in stimulant naïve and stimulant medicated children with ADHD and matched controls. The Cambridge Neuropsychological Test Automated Battery was chosen to measure executive function because it has been used successfully to assess the development of executive function in normal children (Luciana & Nelson, 1998), the impairment of executive function in patients with focal brain lesions and neurodegenerative disease (Owen *et al*. 1990, 1995, 1996*a*, *b*, *c*; Pantelis *et al*. 1997; Purcell *et al*. 1998), and changes in executive function that occur as a consequence of manipulation of catecholaminergic systems (Elliot *et al*. 1997).

METHOD

Subjects

The total sample consisted of three groups of children: a control group of 15 normal children, an unmedicated ADHD group of 15 subjects and a medicated ADHD group of 15 subjects who had received individually tailored doses of stimulant medication (methylphenidate ($N = 2$) or D-amphetamine ($N = 13$); see Table 1). The subjects were aged from 6 to 12 years. To control for age related differences in performance, children in all three groups were carefully matched on age, gender and IQ. The children in the two ADHD groups met the criteria for: (1) a DSM-IV diagnosis of Attention Deficit Hyperactivity Disorder (American Psychiatric Association, 1994); and (2) a score greater than 1·5 standard deviations above the mean on the 10-item Abbreviated Conners Rating Scale (Conners, 1985).

The control group consisted of 15 community children matched to the ADHD groups on sex, age, intelligence (WISC-R), reading ability (NEALE), and spelling ability (WRAT) (Table 1). No control subject met the clinical criteria for ADHD or any other psychiatric condition.

Informed consent to participate in the investigation was obtained from each child's parent(s). All the children used in this study were living in a family home with at least one family member and were attending normal state schools. All subjects' Intelligence Quotients were above 70 and there was no history of neurological impairment, closed head injury, pre-natal or peri-natal birth trauma, chronic physical illness, sensory or motor impairment.

Procedure

The ADHD children were tested at the Maroondah Hospital Child and Adolescent Mental Health Service. Testing took approximately 2–3 hours and was conducted over two sessions: during the first session, a trained clinical psychologist administered the achievement tests to the child. The computerized neuropsychological tests were administered in a

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Table 1. *Subject characteristics*

	Controls ($N = 15$) Mean (S.D.)	ADHD medicated ($N = 15$) Mean (S.D.)	ADHD unmedicated ($N = 15$) Mean (S.D.)
N (male, female)	12, 3	13, 2	13, 2
Age	8·81 (1·48)	7·84 (1·64)	8·65 (1·53)
ACRS*		20·67 (6·09)	20·87 (5·40)
Verbal IQ	99·93 (12·23)	93·88 (13·63)	90·27 (10·89)
Performance IQ	103·13 (11·99)	98·31 (10·08)	103·67 (13·61)
Full Scale IQ	101·27 (12·27)	95·56 (10·97)	95·67 (11·65)
Spelling (SS)†	92·53 (13·90)	88·75 (17·69)	89·53 (14·19)
Arithmetic (SS)†	88·60 (15·72)	84·19 (15·91)	83·00 (10·92)
Reading (percentile)‡	43·53 (13·45)	31·06 (30·33)	39·73 (30·65)
Comprehension (percentile)‡	45·27 (16·00)	31·25 (32·96)	39·33 (30·01)
Medication dose	—	D-amphetamine methylphenidate	11·25 mg/day 35 mg/day

* ACRS – Abbreviated Conners Rating Scale.
† Standard Score from the WRAT.
‡ Reading and comprehension from the NEALE.
All comparisons between groups were non-significant.

second session. The control children were assessed in their own homes and usually received both forms of testing on the same day.

Clinical neuropsychological assessment

All subjects were tested on the Neale Analysis of Reading Ability Revised (Neale, 1988) to provide a measure of reading accuracy and comprehension. The Wechsler Intelligence Scale for Children (WISC-3) (Wechsler, 1991) was administered to provide verbal, performance and full-scale scores as a measure of intelligence. Additionally, the third edition of the Wide Range Achievement Test (WRAT-3) (Jastak & Wilkinson, 1984) was administered to provide a measure of spelling and arithmetic abilities.

Experimental computerized tests of executive function

The main testing procedures were taken from the Cambridge Neuropsychological Test Automated Battery (CANTAB) (Owen *et al*. 1990; Sahakian & Owen, 1992; Robbins *et al*. 1994), a series of cognitive tests presented on a high resolution IBM colour monitor with a touch sensitive screen. Subjects were seated approximately 0·5 metre from the monitor and it was explained that they would have to respond to stimuli by touching the screen. All children had reported previous experience with computers although none with a touch sensitive screen. Six subtests from the battery measuring attentional set-shifting, memory, and planning were administered to subjects in a pseudo-random order. The testing session lasted approximately 60–90 min. The tasks used in this study have been standardized in a large group of normal subjects (Robbins *et al*. 1994) and in normal children (Luciana & Nelson, 1998). Task administration in this study was according to standard protocols. The reader is referred to the extensive literature for detailed description of the CANTAB tasks. The CANTAB tests employed were as follows.

(*i*) *Spatial span*

This computerized version of the Corsi's block tapping task (Milner, 1971) assesses the subject's ability to remember a sequence of squares presented on the screen. The spatial short-term memory span is calculated as the highest level at which the subject has successfully recalled at least one sequence of boxes.

(*ii*) *Spatial working memory*

This self-ordered searching task (Petrides & Milner, 1982) measures working memory for spatial stimuli and requires the subject to use mnemonic information to work towards a goal. Returning to an 'empty' box already targeted on a particular search constituted a 'forgetting' or 'between search' error. A 'strategy' score was calculated to reflect how often a searching sequence was initiated from the same box during the trial.

(*iii*) *Tower of London planning task*

This test was originally derived from the 'Tower

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of London' (TOL) task and measures spatial planning and behavioural inhibition (Shallice, 1982). The accuracy of solution during the copying trials was defined as the number of test trials completed within the minimum number of moves and the total number of moves in excess of the minimum. For each test problem, a 'yoked control' condition was employed to provide baseline measures of motor initiation and execution times.

(*iv*) *Attentional set shifting task* (*ID/ED*)

This task assesses the subject's ability to maintain attention to different examples within a reinforced stimulus dimension (intradimensional shift: IDS) and then shift attention to the previously irrelevant stimulus dimension (extradimensional shift: EDS). The test automatically terminated if a subject had not reached criterion for a particular stage within 50 trials. Performance was defined as the 'stage reached' (out of nine) as well as the 'number of trials' to criterion at each stage.

(*v*) *Simultaneous and delayed matching to sample* (*DMTS*)

This task measures the subject's ability to remember a complex target stimulus (in each case, a rectangular shape consisting of different arrangements of shape and colour components), which was presented on the screen for 4 seconds and then re-presented with a 0-, 4-, or 12-second delay. Performance was defined as the percentage of correct responses at the simultaneous and each of the three delay levels.

(*vi*) *Pattern/spatial recognition*

For pattern recognition, subjects' ability to recognize a previously presented abstract coloured pattern from two stimuli is assessed. Performance was defined as the percentage of correct responses across two trials (24 stimuli). For spatial recognition, the task is to recognize the spatial locations of target stimuli. Performance was defined as the percentage of correct responses for the 20 trials.

Statistical analysis

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS/PC) (Nie *et al.* 1970). Subject characteristics were compared across the three groups using one-way analysis of variance (ANOVA) (Table 1). Latencies for the 'Tower of London' task were measured to the nearest 10 ms, and transformed using logarithmic (base 10) transformations to reduce skewness in the distribution (Kullback, 1968). Performance on the spatial working memory task, DMTS task and the Tower of London measures of cognitive and motor speed were compared with a repeated measures analysis of variance (ANOVA) using a two-factor design that included a between-subjects factor (group) and a within-subjects factor (e.g. difficulty level). To protect against violation of the homogeneity of covariance assumption, analysis of within-group effects or interactions with degrees of freedom greater than one were examined using a repeated measures design within MANOVA (Wilk's Multivariate test of significance). Performance on the spatial span task, the spatial working memory strategy measure, the spatial and pattern recognition tasks, simultaneous matching to sample, and the TOL accuracy measures were compared between groups using one-way ANOVA. Where the omnibus F was significant, the *post-hoc* Studentized Newman–Keuls (SNK) procedure ($P < 0{\cdot}05$) was conducted to determine the source of this significance. Performance on the ID/ED set shift was examined by comparing the percentage of subjects who succeeded in reaching criterion at each stage of the task. The resulting contingency tables were analysed using the likelihood ratio method, which allows for use where cell frequencies were small, with the resulting statistic 2i being distributed as χ^2 (Kullback, 1968). To investigate the relationship between short-term memory (span) and spatial working memory, Pearson's product moment correlation coefficients were employed. This same procedure was used to calculate the relationship between subjects' clinical variables and the measures of cognitive function.

RESULTS

The group performance means for each cognitive task are summarized and compared in Table 2. There were no differences between groups for strategy score on the spatial working memory task, error rate on the simultaneous matching to sample, thinking times and initial movement time for the TOL, and number of correct

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Table 2. *Group comparison*

Measure	Controls ($N = 15$) Mean (S.D.)	ADHD medicated ($N = 15$) Mean (S.D.)	ADHD unmedicated ($N = 15$) Mean (S.D.)	Group comparison
Spatial span	4·93 (1·03)	4·40 (0·91)	3·69 (1·0)	F*
Spatial working memory				
Total between-search errors	40·73 (10·30)	50·93 (17·51)	58·44 (16·39)	F*
Strategy score – unmodified	36·33 (3·13)	37·40 (3·50)	37·38 (2·92)	F
Tower of London				
Number of minimum move solutions	7·33 (1·59)	6·73 (1·94)	5·06 (2·32)	F*
Total moves in excess of the minimum	12·19 (9·02)	14·13 (2·67)	15·20 (1·66)	F*
Initial thinking time	3·72 (1·33)	3·93 (1·22)	4·41 (2·27)	F
Subsequent thinking time	2·70 (0·55)	3·07 (0·39)	3·04 (0·48)	F
Initial movement time	3·74 (0·20)	3·80 (0·28)	3·71 (0·23)	F
Subsequent movement time	3·89 (0·22)	4·06 (0·34)	4·10 (0·34)	F*
Attentional set shifting				
Proportion reaching IDS	93%	100%	87%	χ^2
Proportion reaching IDR	93%	100%	75%	χ^2*
Proportion reaching EDS	93%	93%	63%	χ^2*
Proportion reaching EDR	93%	93%	63%	χ^2*
IDR-trials to criterion	14·47 (10·68)	12·13 (5·42)	26·00 (18·40)	χ^2*
EDS-trials to criterion	12·60 (10·68)	20·33 (16·45)	27·75 (19·25)	χ^2*
Delayed matching to sample				
Simultaneous (% correct)	94·0 (8·28)	86·67 (16·76)	80·0 (24·22)	F
Total responses (% correct)	78·87 (9·75)	68·83 (17·25)	60·31 (18·39)	F*
Average (% correct)	73·78 (12·21)	61·56 (16·52)	53·75 (20·4)	F*
Spatial recognition				
Total responses (% correct)	76·67 (14·10)	63·0 (14·86)	65·16 (12·47)	F*
Pattern recognition				
Total responses (% correct)	79·77 (12·29)	77·47 (17·17)	73·66 (20·05)	F

* Denotes significance at $P < 0·05$.

responses on the pattern recognition task. Furthermore, there were no interactions between task difficulty and group for TOL and Delayed Matching to Sample Tasks.

For spatial span, ANOVA was significant ($F(2, 43) = 6·22$, $P = 0·004$) and *post-hoc* SNK tests revealed that the unmedicated ADHD group had significantly shorter spatial span than either the medicated ADHD group or the controls.

Fig. 1 shows the mean between-search errors for each group according to the difficulty level of the task. There was a significant effect of group ($F(2, 43) = 5·34$, $P = 0·01$) and task difficulty (Wilk's $\lambda = 0·06$, $F(2, 43) = 155·04$, $P = 0·001$), but no interaction between the group and difficulty factors (Wilk's $\lambda = 0·71$, $F(2, 43) = 1·82$, $P = 0·08$). Investigation of the group effect showed that, when arranged across level of difficulty, the unmedicated ADHD group made significantly more between-search errors than controls.

Subjects in the unmedicated ADHD group were significantly impaired on the ID/ED set-shifting task. Fig. 2 shows that 93% of control and medicated ADHD subjects were able to complete all stages successfully. However, only

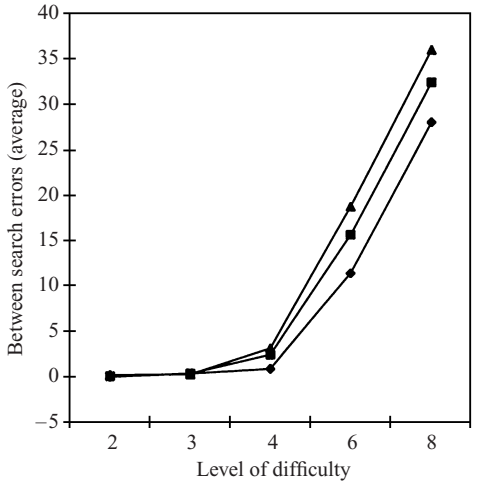


Fig. 1. Mean number of 'between-search' errors at each level of difficulty on the spatial working memory task. (◆, Control; ■, medicated; ▲, unmedicated.)

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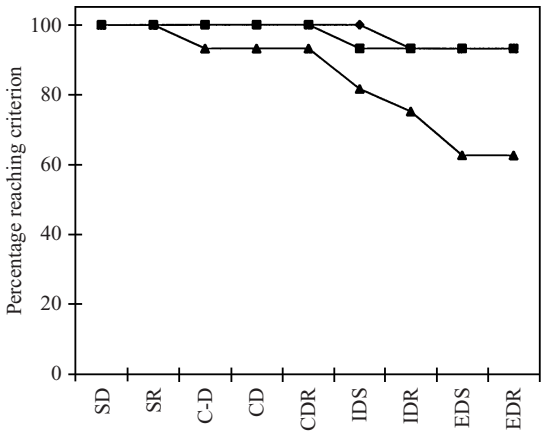


FIG. 2. Cumulative failure at each stage of the visual discrimination learning/attentional set shifting task. Stages: SD, simple discrimination; SR, simple reversal; C-D, compound discrimination; CD, superimposed compound discrimination; CDR, superimposed compound discrimination reversal; IDS, intradimensional shift; IDR, intradimensional reversal; EDS, extradimensional shift; EDR, extradimensional reversal. (◆, Control; ■, medicated; ▲, unmedicated.)

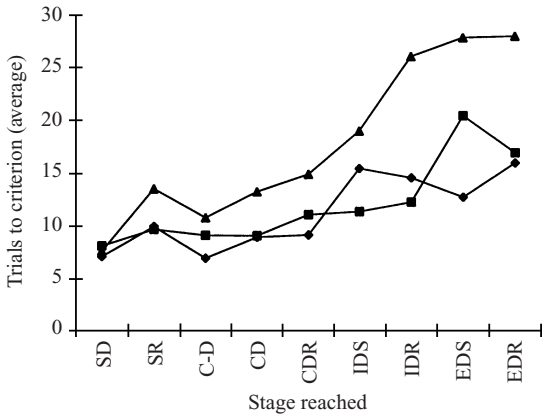


FIG. 3. Mean trials to criterion at each stage of the visual discrimination learning/attentional set shifting task. Stages: SD, simple discrimination; SR, simple reversal; C-D compound discrimination; CD, superimposed compound discrimination; CDR, superimposed compound discrimination reversal; IDS, intradimensional shift; IDR, intradimensional reversal; EDS, extradimensional shift; EDR, extradimensional reversal. (◆, Control; ■, medicated ADHD; ▲, unmedicated ADHD.)

63% of unmedicated patients were able to complete all stages of the task ($2i = 6{\cdot}64$, $P = 0{\cdot}04$). The proportion of subjects passing each stage of the task was also analysed non-cumulatively (Purcell *et al.* 1998), by analysing the data for those subjects actually attempting each stage. Although the groups did not differ at the IDS stage ($2i = 2{\cdot}77$, NS), more unmedicated ADHD subjects failed the task at the IDR stage ($2i = 6{\cdot}28$, $P = 0{\cdot}04$), the EDS stage ($2i = 6{\cdot}64$, $P = 0{\cdot}03$) and the EDR stage ($2i = 6{\cdot}64$, $P = 0{\cdot}03$). The numbers of trials required to attain criterion at each stage were also analysed non-cumulatively. Fig. 3 shows that unmedicated ADHD subjects required more trials to reach criterion than the controls and the medicated ADHD subjects, at the IDR ($F(2, 37) = 5{\cdot}227$, $P = 0{\cdot}01$) stage, and more trials to reach criterion than the control group at the EDS ($F(2, 35) = 3{\cdot}49$, $P = 0{\cdot}04$) and EDR stages ($F(2, 35) = 3{\cdot}79$, $P = 0{\cdot}03$).

For the TOL task, two measures relating to the number of moves required to reach solution were calculated. There was a significant difference between the three groups for the total minimum move solutions across test trials ($F(2, 43) = 5{\cdot}49$, $P = 0{\cdot}01$) and for the total minimum number of moves to solution across all trials ($F(2, 43) = 5{\cdot}38$, $P = 0{\cdot}01$). Comparison

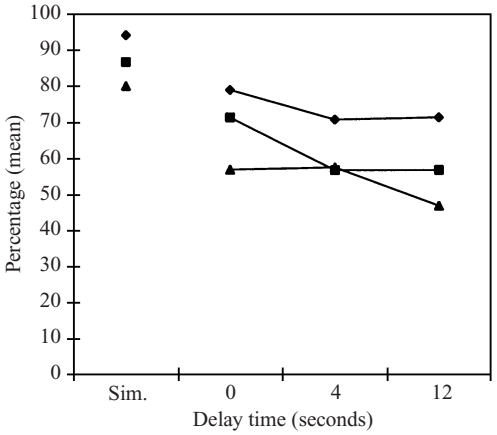


FIG. 4. Mean percentage correct across simultaneous and three delay conditions on the delayed matching to sample task. (◆, Control; ■, medicated; ▲, unmedicated.)

between the groups indicated that the unmedicated ADHD group was significantly impaired compared to controls and the ADHD medicated group.

Using a 'yoked' control condition, both movement and thinking latencies were assessed. The subsequent movement time measured in the TOL task was nominated as the time taken for the subject to compare two sets of stimuli, execute the necessary 'following' move and

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continue to repeat these following movements until the task was completed. In contrast, the initial movement time reflected the execution of only the first 'following' move in the sequence. These provided a measure of motor speed to complete each task within a specified time period. The results for the subsequent movement latencies for the groups at each difficulty level of the 'following' task, revealed significant main effects for group ($F(2,40) = 3{\cdot}54$, $P = 0{\cdot}04$) and task difficulty (Wilk's $\lambda = 0{\cdot}15$, $F(2,40) = 69{\cdot}62$, $P < 0{\cdot}005$). Response latencies in the unmedicated ADHD group performance were significantly slower than the controls and medicated ADHD group.

Fig. 4 shows the percentage correct scores for the simultaneous and the three delay conditions of the DMTS task. No significant main effects were evident for the simultaneous condition ($F(2,43) = 2{\cdot}382$, $P = 0{\cdot}104$). MANOVA indicated a significant effect for group ($F(2,43) = 5{\cdot}56$, $P = 0{\cdot}007$) and difficulty (Wilk's $\lambda = 0{\cdot}75$, $F(2,43) = 5{\cdot}88$, $P = 0{\cdot}002$), but no significant group by difficulty interaction. *Post hoc* (SNK) analysis indicated that, when averaged across the 0, 4 and 12 second delays, the average percentage error rates in the control group and the medicated ADHD group were significantly less than in the unmedicated ADHD group.

For the spatial recognition task, both the unmedicated ADHD and medicated ADHD groups recognized fewer spatial locations correctly compared to the control groups ($F(2,43) = 4{\cdot}27$, $P = 0{\cdot}02$). There were no differences for the pattern recognition memory task ($F(2,43) = 0{\cdot}506$, $P = 0{\cdot}606$).

Correlations between CANTAB tasks

To examine the role of short-term memory capacity on working memory performance, correlations were performed between spatial span and the spatial working memory measures (see Pantelis *et al.* 1997). For the patients with ADHD there were significant negative correlations between spatial span and between-search errors (unmedicated, $r = -0{\cdot}55$, $P = 0{\cdot}027$ and medicated, $r = -0{\cdot}63$, $P = 0{\cdot}013$), although this association was not observed in the control group.

Span was correlated with both of the TOL accuracy measures (minimum move solutions for all trials and minimum move solutions for test trials) for the unmedicated ADHD group ($r = 0{\cdot}622$, $P = 0{\cdot}01$) and control group ($r = 0{\cdot}58$, $P = 0{\cdot}018$), respectively. The medicated ADHD group's performance on minimum move solutions for all trials also correlated significantly with span ($r = 0{\cdot}535$, $P = 0{\cdot}01$).

Correlations of demographic/clinical measures and CANTAB tasks

For the unmedicated ADHD subjects full scale IQ was positively correlated with the Delayed Matching to Sample Task, total percentage correct ($r = 0{\cdot}303$, $P = 0{\cdot}04$) and the average percentage correct ($r = 0{\cdot}345$, $P = 0{\cdot}02$). Scores on the full scale IQ and Neale reading percentile were negatively correlated with subsequent movement time at the two-move ($r = -0{\cdot}32$, $P = 0{\cdot}03$) ($r = -0{\cdot}47$, $P = 0{\cdot}001$) and the three-move ($r = -0{\cdot}379$, $P = 0{\cdot}009$) ($r = -0{\cdot}42$, $P = 0{\cdot}003$) problems. A negative correlation was also found between the Neale and spatial span ($r = -0{\cdot}313$, $P = 0{\cdot}03$), indicating poor spatial span was related to poor reading ability. Age did not correlate significantly with the ADHD children's impaired performance on the other measures of cognitive function.

DISCUSSION

The results from the study indicate that unmedicated ADHD children were impaired on tasks of executive function, including planning ability, movement time, attentional set shifting and spatial working memory. No impairments were evident, however, for strategy use on the spatial working memory task. Unmedicated children with ADHD also showed abnormal performance on some tests of visuospatial memory and learning indicating abnormalities in spatial recognition and delayed matching to sample. The group of medicated ADHD children in this study showed no impairment on any of the executive function tasks, although this group did perform poorly on the spatial recognition memory task. These results suggest that cognitive abnormalities do occur in children with ADHD and these are most apparent on tests of executive function. These cognitive impairments do not occur in medicated children with ADHD, suggesting the stimulant medication normalizes executive function in ADHD.

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The results show that although the unmedicated ADHD group performed worse than controls on tests of executive function, this was not a general abnormality across all tests or all performance indices. Abnormal performance occurred only on specific aspects of test performance. For example, on the TOL test, abnormal performance was evident in the increased number of moves necessary to complete only the more difficult levels of the test. However, the latency to respond to this test was normal. Yet, when required merely to copy the moves that they had made while solving the TOL problems, unmedicated children with ADHD showed much longer latencies for movements than controls. Unmedicated children with ADHD also showed smaller spatial spans than controls suggesting a reduction in their ability to hold information in their spatial memory. The increased number of between-search errors on the spatial working memory task indicated that this impairment also occurred when children were required to manipulate spatial information within working memory. Interestingly, unmedicated children with ADHD did not differ from normals for strategy scores on the spatial working memory task. Therefore, their poorer performance on this test was related to their decreased spatial span. This hypothesis is supported by the finding that spatial span, but not strategy, was correlated with between search errors. These results suggest that children in this age group are not yet able to develop systematic strategies to assist performance. The unmedicated children with ADHD were also impaired on the set-shifting task at the intradimensional shift stage. At this stage, the children were required to use a rule for the selection of specific stimuli, that had been learned previously, and apply it to new exemplars of the same class of stimuli. The unmedicated ADHD group were unable to perform this action. On the tests of spatial memory, unmedicated ADHD subjects were also impaired in their ability to recognize spatial locations and to hold spatial information in memory for any period (i.e. > 0 second delay) on the delayed matching to sample task. Importantly, the unmedicated ADHD group showed no general slowing of responses and no general increase in errors performed.

When taken together this pattern of abnormal performance is qualitatively similar to that found, on the same tasks, in adults with focal lesions of the frontal lobes, Parkinson's disease and Huntington's disease and in non-human primates with focal lesions of the dorsolateral prefrontal cortex (Mattes, 1980; Owen *et al.* 1990; Kimberg & Farah, 1993; Pantelis *et al.* 1997). In each of these reports the deficits in executive function were attributed to a dysfunction of frontostriatal systems, although some differences in executive abnormalities were associated with disruption to different levels of these systems. Importantly, this pattern of abnormal executive function is not observed in adults with lesions of the lateral or medial temporal lobes or in children with cerebellar signs associated with petrol-related encephalopathy (Maruff *et al.* 1998). Therefore, although we must be cautious in comparing abnormal cognitive performance in children to that of adults with focal brain lesions, the results are consistent with the hypothesis that abnormalities of executive function in ADHD reflect a dysfunction of frontostriatal pathways.

This interpretation of the results is also consistent with a number of other studies which have identified abnormal performance on other tests of executive function such as the WCST (Chelune *et al.* 1986; Boucugnani & Jones, 1989; Gorenstein *et al.* 1989; Shue & Douglas, 1992). Boucugnani & Jones (1989) and Chelune *et al.* (1986) pointed to the high perseverative error score of their ADHD children as an indicator of dysfunction of the inhibitory forebrain system. In the present study, the high failure rate at the intradimensional reversal stage suggests that unmedicated ADHD subjects may have made more perseverative responses because of an inability to inhibit the incorrect response.

Executive function in children treated with stimulant medication

Brain catecholamine function has been emphasized in neurobiological theories of ADHD, largely due to the established clinical response to stimulant medication of children with ADHD (Heilman *et al.* 1991). Methylphenidate and D-amphetamine, which act primarily on dopaminergic brain systems, are the most commonly used stimulant medications for ADHD. In the current study, the majority of the medicated group were taking methylphenidate.

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Unfortunately, the low numbers of subjects taking D-amphetamine ruled out any comparison between the effects of the different treatments on cognitive performance. Nevertheless, it was remarkable that children who were taking stimulant medication generally showed little or no abnormality of executive function. This result is also consistent with some previous findings (Douglas *et al.* 1986; Everitt *et al.* 1991). For example, a previous study of executive function in ADHD, which used performance on the WCST to define executive function, found improved performance following stimulant medication (Douglas *et al.* 1995). Taken together these studies suggest that deficits in executive function arise because of a dysfunction of catecholaminergic function rather than any disruption to brain structure. Alternatively, an ADHD-related disruption to brain structure may be compensated for by stimulant medication. In either case, the observation that there were no cognitive deficits in children receiving stimulant medication is important clinically especially as such medication has been shown to reliably impair executive function in normal healthy subjects (Elliot *et al.* 1997). In addition, it suggests that ADHD symptoms and ADHD-related cognitive deficits arise from a common source. However, repeated measure, placebo controlled, experimental designs are needed to investigate these issues further.

Developmental aspects of executive function deficits in ADHD

Diminished motivation, lower education and IQ level were not thought to influence subject performance in this study as global impairments across the CANTAB tasks were not evident in either group of ADHD subjects. Rather, performance on these tasks reflects the cognitive ability in children with ADHD. Several authors have suggested that ADHD children have developmental 'lag' and perform at a level approximately 2 years behind their age-matched cohorts (Chelune & Baer, 1986; Boucugnani & Jones, 1989; Shue & Douglas, 1992). By comparing the qualitative performance of ADHD children in this study to the results of normal 6- and 8-year-old children in a recent study by Luciana & Nelson (1998), it is possible to explore this notion of developmental 'lag'. Control subjects in this study (mean age 8 years) performed similarly to the 8-year-old subjects on the CANTAB tasks in the comparison study, indicating consistency on these measures for children of this age. Comparison of the performance of ADHD subjects to that of the 6-year-old subjects in the Luciana & Nelson study, indicates that the two groups were performing at a qualitatively similar level, with both groups scoring lower than the control and 8-year-old subjects. This supports the literature concerning a developmental 'lag' in children with ADHD. However, the fact that stimulant medication was associated with a better executive function performance in this study, suggests any maturational delay may reflect a delay in some aspect of catecholaminergic innervation of frontostriatal systems. Further research should investigate this issue specifically.

Methodological issues

It could be argued that the difference between the ADHD groups on tests of executive function occurred because stimulant medication improved behaviour in general and this allowed the children with ADHD to concentrate on performing the tests. We believe that if this had been the case, then a general improvement in both the speed and accuracy of performance on the CANTAB tests would have been evident. Clearly, this was not the case as improved cognitive function was evident only for those variables where significant differences in performance had been observed between the unmedicated ADHD group and controls. On a related issue, differences between the three subject groups were unlikely to be due to age, IQ, or education level as subjects were selected and matched carefully on these variables. Finally, in this study, the medicated ADHD children were receiving an individually tailored dose of stimulant medication. Previous research has found that higher medication doses do not constrict cognitive functioning, but rather improve cognitive functioning in a dose-dependent linear curve (Douglas *et al.* 1988). It could be argued that medication doses might have influenced some task performances in this study (i.e. if they were considered too low). However, this was not thought to be the case, as an appropriate medication dose for each child was considered relevant to the degree of their symptomatology. It would be of interest, how-

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ever, for future studies to explore differing dose effects on cognitive performance using the CANTAB.

Conclusion

The present study has demonstrated a broad pattern of cognitive impairments in a group of unmedicated ADHD children. By using a variety of executive function measures, the study demonstrated that, relative to controls, specific cognitive impairments can be seen on tasks of spatial short-term memory, spatial working memory and ability to shift cognitive set, and marked deficits are seen on planning ability including response slowing. Although the unmedicated ADHD group was performing adequately on pattern recognition memory, impairments were recognized on measures of spatial recognition memory and delayed matching to sample.

The medicated ADHD group was not impaired on any of the executive function tasks although remained impaired on the spatial recognition memory task. That is, stimulant medication was associated with a better executive function performance. This finding supports frontostriatal system involvement in ADHD and implicates the frontostriatal system as responsible for catecholamine-dependent effects on cognition.

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