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Attentional functions in children and adolescents with attention-deficit/hyperactivity disorder with and without comorbid tic disorder

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Summary. Although the coexistence of attention-deficit/hyperactivity disorder (ADHD) and tic disorder (TD) is common, the nature of association is yet not fully understood. Thus, the aim of the present study was to explore attentional dysfunction in children with pure ADHD compared to children with comorbid ADHD + TD. Three groups of 20 children each, aged 8–15 years with either ADHD, ADHD + chronic tic disorder or Tourette syndrome (ADHD + TD) and a healthy control group were compared in their performance on three computerized attention tasks. Tasks of sustained attention, selective attention and interference control were employed. In addition, parental ratings of ADHD symptom severity and behaviour problems were obtained. Both clinical groups were rated as equally inattentive, however, externalising symptoms were more severe in the ADHD group. Objective measures of attentional performance revealed differences between the groups: whereas the ADHD group was markedly impaired in sustaining attention and selective attention/inhibitory control, the ADHD + TD group only showed marginal deficits in selective attention/inhibitory control. Possible explanations for the superior performance of the comorbid group are discussed: In particular, the results may indicate that in some patients, the tic disorder produces behavioural symptoms of ADHD, but not the broad neurocognitive deficits that usually are associated with ADHD. Alternatively, compensatory neural mechanisms of TD patients may result in a better neuropsychological performance of comorbid patients relative to patients suffering from pure ADHD.

Keywords: Tic disorder; attention-deficit/hyperactivity disorder; comorbidity; attention; children

Introduction

Tourette syndrome (TS) is a neuropsychiatric disorder characterized by motor and vocal tics that usually have their onset around the age of 5–6 years. At onset, patients often exhibit simple motor tics, such as eye blinking, followed by complex motor tics, which involve movements like thrusting arms. Vocal tics typically appear some time later after the onset of first motor tics (Leckman et al. 2001). Chronic tic disorders, characterized by either chronic (>1 year) motor or vocal tics, are presumably on the same spectrum as TS (Cohen et al. 1985). Several lines of evidence, including neuroimaging, neurophysiological and biochemical studies, suggest that the basal ganglias and in particular, cortico-striato-thalamo-cortical circuits are involved in the pathology of tic disorders (TD) (Olson 2004; Voelker et al. 2004; Vloet et al. 2006). These circuits are thought to mediate behavioural inhibition, initiation and monitoring (Alexander et al. 1990).

Psychiatric disorders are co-diagnosed in 90% of TD patients (Freeman et al. 2000), with ADHD being the most common comorbid condition. ADHD is present in approximately 50% of all tic patients (Rothenberger and Banaschewski 2005), whereas about one third of ADHD patients suffer from TD (Spencer et al. 1998). ADHD symptoms are typically manifest 2–3 years before the onset of tics. In a minority of patients, tics precede ADHD (Spencer et al. 2001). The reasons for the high co-occurrence of TD and ADHD have been intensively discussed in the last decades but exact etiological mechanisms still remain unclear. There is evidence of common neurobiological substrates, in particular overlapping abnormalities in cortico-striato-thalamo-cortical circuits (Vloet et al. 2006).

In both ADHD and TD patients, attention problems have been described (Comings 1990; Barkley 2003). Inattention

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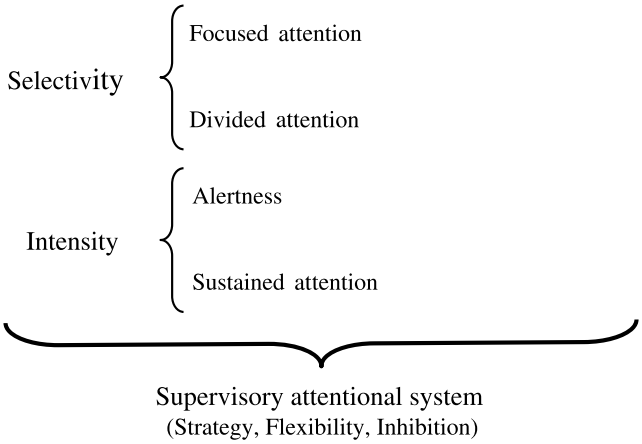

Fig. 1. A schedule of the theoretical framework of attentional functions according to Van Zomeren and Brouwer (1994)

is one of the core symptoms in ADHD and has been intensively investigated, with systematic attempts to explore specific attention deficits based on well-defined concepts of attention. Concepts of attention generally distinguish between selectivity and intensity of attention. Selectivity refers to the process that modulates responsiveness to specific stimuli constellations by giving priority to certain stimuli, whereas intensity describes the ability to activate and sustain attention over time. In addition, a supervisory attentional system is assumed to act as an executive control mechanism, modulating the two domains of selectivity and intensity (Van Zomeren and Brouwer 1994). Figure 1 illustrates this theoretical framework with appropriate paradigms for the assessment of the respective attentional functions. This concept of attention is supported by clinical studies (Spikman et al. 2001) as well as by functional imaging data (Cabeza and Nyberg 2000).

An extensive body of literature supports the notion that a deficit in executive functions of attention seems to be central in ADHD (Durston 2003). Children diagnosed with ADHD perform worse than controls in go/nogo, stop-signal and stroop tasks (Seidman et al. 1995, 1997; Oosterlaan and Sergeant 1998; Tannock 1998; Schachar et al. 2000; Konrad et al. 2001) and in other tasks of cognitive control (Pennington et al. 1993; Aman et al. 1998; Grodzinsky et al. 1999). A number of studies reported deficits in sustained attention or vigilance (Corkum and Siegel 1993; Taylor et al. 1998; Sergeant et al. 1999; Hanisch et al. 2004), while other studies did not find deficits in these tasks (Van der Meere and Sergeant 1988; Sergeant and Van der Meere 1990). A meta-analytic study (Huang-Pollock and Nigg 2003) reports only small, non-significant effects for deficits in the intensity domain of attention. Another meta-analytic review (Losier et al. 1996) and recent studies (Hervey et al. 2006; Johnson et al. 2007; Klein et al. 2007) stress hat reaction times in alerting tasks tend to be within the normal range, whereas deficits in sustaining attention and increased variability of reaction time are most prevalent.

Despite apparently frequent reports of attention problems in TD, few studies have comprehensively addressed this field of research. A number of early studies compared TD patients with healthy controls on task related to executive attention and reported deficits in TD patients (e.g. Bornstein 1991; Channon et al. 1992). However, some of these studies have not systematically accounted or screened for comorbid conditions, such as ADHD or obsessive-compulsive disorder (OCD). Since both ADHD and OCD are associated with deficient executive control (Sergeant et al. 2002), the question as to whether the TD itself accounts for the deficits or whether they can be attributed to comorbid conditions remains unclear. Most recent studies that carefully took account of comorbidity support a circumscribed and mild, if not even absent impairment in behavioural measures of executive attention in both children and adults with TD (Yeates and Bornstein 1994; de Groot et al. 1997; Ozonoff and Strayer 1998; Channon et al. 2006; Denckla 2006). Most of the studies that investigated performance of TD patients in sustaining attention have reported slowed reaction times (Channon et al. 1992; Harris et al. 1995; Shucard et al. 1997) although one study could not replicate this finding (Sherman et al. 1998). Slowed reaction time in TD has also been reported for other cognitive tasks (Schuerholz et al. 1996; Channon et al. 1998).

Despite recent attempts to tackle the question as to the nature of the frequent coexistence of TD + ADHD (e.g. Moll et al. 2001; Yordanova et al. 2006), to date there have been only few studies that compared ADHD + TD with ADHD in the attention domain. Como (1993) compared ADHD + TD patients with ADHD patients on tasks of attentional ability. Unexpectedly, the comorbid group performed better than the ADHD group in these tasks. Similarly, Sherman and colleagues (1998) could show superior performance of the comorbid group in sustaining attention. Based on the consideration that tics may be perceived as hyperactivity/restlessness, the authors raised the assumption that TD may produce behavioural ADHD symptoms without the neurocognitive deficits that are usually associated with ADHD. Both groups in the study of Sherman et al. were comparable with respect to behavioral ratings of hyperactive symptoms, however, no data were provided for measures of inattention or impulsivity.

The main objective of the current study was thus to further explore attentional dysfunction in TD and ADHD. Based on a theoretical framework of attention, children

with ADHD were compared with ADHD + TD children using a selection of three computerized attention paradigms that covered the attention domains. In order to facilitate interpretation of possible group differences, an additional aim was to further characterize both groups not only by means of objective symptom measures but also by assessing subjective ratings of ADHD related symptoms.

We predicted that both clinical groups would demonstrate deficits in attentional performance when compared to a healthy control group. However, based on previous research, we assumed that attentional dysfunction in the combined group would be less severe compared to the ADHD group. It was hypothesized that the ADHD + TD group would show slower reaction times across tasks but not the typical deficits observed in ADHD children such as impaired executive attentional performance and increased variability of reaction time.

Material and methods

Subjects

A total of 20 children with ADHD, 20 children with ADHD and comorbid tic disorder (9 with Tourette syndrome, 11 with chronic motor tics) and 20 healthy controls participated in the study. Their age ranged from 8 to 15 years (M = 11.3, SD = 1.9). Only patients with an IQ above 84 (as measured with the CFT-20 or WISC-III) were included. There were no significant differences between the groups with regard to age, IQ or proportion of girls ($p > 0.05$ for all comparisons between groups). Table 1 summarizes the major demographic data.

Patients were recruited from our inpatient or outpatient Department of Child and Adolescent Psychiatry. Psychiatric classification according to DSM-IV (American Psychiatric Association, 1994) was based on the diagnosis of an experienced clinician, including the developmental history of the child, playroom observation, pediatric examination and parents' evaluations with the child behaviour checklist (CBCL, Achenbach 1991). Exclusion criteria were any potentially confounding diagnoses such as obsessive-compulsive disorder, psychosis, mania, major depression, or substance abuse, and also pervasive developmental disorders. Comorbid diagnoses and CBCL ratings of the two clinical groups are summarized in Table 2. As shown in Table 2, the sample represents a typical clinical sample with high comorbidity rates. In both clinical groups, conduct disorder (CD)/oppositional defiant disorder (ODD) was the most frequent comorbidity, followed by specific developmental disorders. No significant differences between groups regarding comorbidity rates were found ($p > 0.05$). Inattention and externalizing scores in both groups were >60, ensuring that patients showed behavioural ADHD symptoms at the time of testing. There were no significant group differences for CBCL internalizing or inattention scores. However, externalizing symptoms were rated as more severe in the ADHD group ($p < 0.05$).

In order to exclude a psychiatric history or acute psychiatric symptoms of children in the control group, a German semi structured interview was conducted with the parents (K-DIPS; Unnewehr et al. 1995). Besides, parents confirmed the absence of present tics or a lifetime history of tics as assessed via the respective section from Kiddie-SADS-Present and Lifetime Version (K-SADS-PL; Kaufman et al. 1997; German adaptation by Delmo et al. 2000).

Procedure

All subjects received a standardized computerized neuropsychological assessment, which lasted about 30 min. All children were tested in the morning. Upon arrival, subjects were seated on a comfortable chair in front of the monitor. All children received identical spoken instructions. To make sure that all children were able and willing to comply with the task requirements, all tasks were preceded by practice trials.

If children with ADHD received stimulants ($n = 13$ in the ADHD, $n = 6$ in the ADHD + TD group), these were deposed 48 h before testing. None of the children were medicated with atomoxetine. Three children with tic disorder received tiapride (Tiapridex®) during testing. All other children were free of medication.

Dependent measures

According to the concept of attention (Fig. 1), three tasks were selected. The intensity domain was assessed by a sustained attention task and the supervisory attentional system was tested with a set shifting task. A go/nogo paradigm was selected to account for both the selectivity domain of attention and the supervisory attentional system.

Sustained attention

The sustained attention task involved the continuous and consecutive presentation of 50 series of twelve different dot patterns (600 signals; DeSonneville 2001). In each series an equal number of 3-dot, 4-dot, or 5-dot patterns were presented in a pseudo-random manner. The child was instructed to push the 'yes' button with the dominant hand whenever a 4-dot pattern (target) was presented, and to press the 'no' button with the non-dominant hand if the presented pattern contained 3 or 5 dots (non-targets).

Go/nogo

In the go/nogo paradigm (Fimm and Zimmermann 2001) a motor response with the dominant hand was either initiated (go) or inhibited (nogo) depend-

Table 1. *Demographic data of the total sample, of clinical groups and of healthy controls*

	Total group ($n = 60$)	Controls ($n = 20$)	ADHD ($n = 20$)	ADHD + TD ($n = 20$)	Group difference
Age (M, SD)	11.3 (1.9)	11.5 (1.7)	11.2 (2.3)	11.3 (1.7)	n.s.[a]
IQ (M, SD)	100.0 (9.5)	101.6 (10.4)	99.3 (10.2)	99.2 (8.2)	n.s.[b]
Sex (n; % girls)	15; 25%	5; 25%	4; 20%	6; 30%	n.s.[c]

ADHD attention-deficit/hyperactivity disorder, *TD* tic disorder, *ADHD + TD* comorbid group.
[a] ANOVA: $F(2,57) = 0.10$.
[b] ANOVA: $F(2,57) = 0.40$.
[c] χ^2-Pearson = 0.77.

Table 2. *Clinical data of children with ADHD and of children with ADHD + TD*

Comorbid disorders	ADHD ($n = 20$)	ADHD + TD ($n = 20$)	χ^2-Pearson
Oppositional defiant/conduct	35%	40%	n.s.
Specific developmental disorders	35%	20%	n.s.
Anxiety	15%	10%	n.s.
Affective disorder	15%	5%	n.s.
	M (SD)	M (SD)	*t*-tests
CBCL (parents rating)[a]			
Internalizing	66.6 (11.1)	66.5 (8.1)	n.s.
Externalizing	70.6 (7.8)	65.2 (7.4)	$p < 0.05$
Inattention	70.7 (11.0)	70.2 (9.2)	n.s.

ADHD attention-deficit/hyperactivity disorder, *TD* tic disorder, *ADHD + TD* comorbid group.
CBCL child behaviour checklist.
[a] refers to the unmedicated status.

ing on whether an "x" (go) or a "+" (nogo) stimulus appeared. The task comprised 50 trials.

The dependent measures of the sustained attention and the go/nogo task were the reaction time (median for the go/nogo and mean for the sustained attention task) and its standard deviation, the number of misses and false alarms.

Visual set shifting

In the visual set shifting task (DeSonneville 2001), which consisted of 70 trials, the signal was a bar with a coloured square, which jumped from left to right and vice versa. The square might change its colour from red to green or from green to red. Depending on the colour of the square after the jump, the child had to copy the movement or was required to mirror the movement of the square (i.e. press the left key in response to a rightward movement or the right key for a leftward one). The dependent measures of this task were the number of errors and the reaction time.

Statistical analysis

To examine the relationship between attention parameters, Pearson product-moment correlations were computed. A multivariate analysis of variance followed by univariate analyses of variance with group as between-subject factor and age as covariate were conducted for the attention parameters of the three paradigms. A multivariate approach was chosen since attention parameters correlated substantially (see results section). Age was included as a covariate since this variable showed significant negative correlations with go/nogo and sustained attention reaction time, sustained attention standard deviation and visual set shifting errors ($p < 0.05$). Attention variables did not significantly correlate with IQ ($p > 0.05$).

In the case of significant group effects in the analyses of variance, further single comparisons were made. To control for Type I error, a Tukey adjustment (if variances were homogenous) or a Tamhane adjustment (if variances were inhomogenous) was applied for these single comparisons. Effect sizes were calculated using eta square (η^2).

Results

Correlations between attention parameters

Within task, significant negative correlations were found between reaction time and errors in the go/nogo task indicating a speed-accuracy trade-off across all groups in the go/nogo task ($r = -0.32$, $p < 0.05$ for misses, $r = -0.45$, $p < 0.0001$ for false alarms). Further, significant correlations were revealed between reaction time and standard deviation ($r = 0.81$, $p < 0.0001$) as well as between misses and false alarms ($r = 0.40$, $p < 0.01$) in the sustained attention task. Regarding the relationship between parameters across different tasks, significant correlations were found between all reaction time parameters (between 0.30 and 0.37, $p < 0.05$). Similarly, error parameters of the three tasks correlated substantially: significant correlations were found between go/nogo false alarms and sustained at-

Table 3. *Neuropsychological test performance of clinical groups and control subjects*

	Controls ($n = 20$) M (SD)	ADHD ($n = 20$) M (SD)	ADHD + TD ($n = 20$) M (SD)	*F*	*p*	Partial η^2
Sustained attention						
RT	1246.8 (253.0)	1197.5 (277.3)	1172.0 (309.9)	0.76	n.s.	0.03
SD	603.1 (236.3)	601.3 (262.9)	537.0 (206.9)	0.76	n.s.	0.03
FA	15.8 (7.9)	29.7 (20.7)*	18.2 (15.0)	4.46	0.016	0.14
MIS	22.9 (12.0)	28.3 (22.6)	27.7 (19.0)	0.52	n.s.	0.02
Go/nogo						
RT	441.6 (124.6)	430.0 (119.6)	467.5 (117.1)	0.56	n.s.	0.02
SD	108.6 (37.2)	145.6 (99.0)	122.6 (31.1)	1.64	n.s.	0.06
FA	5.6 (4.9)	15.5 (12.7)*	10.0 (6.2)(*)	6.39	0.003	0.19
MIS	3.3 (3.7)	4.1 (5.1)	3.3 (5.0)	0.18	n.s.	0.01
Set shifting						
RT	1238.7 (340.9)	1091.0 (280.1)	1131.3 (357.2)	1.31	n.s.	0.05
Errors	11.7 (7.4)	18.0 (14.1)	14.4 (10.8)	1.56	n.s.	0.05

ADHD attention deficit/hyperactivity disorder, *TD* tic disorder, *ADHD + TD* comorbid group.
RT reaction time, *FA* false alarms, *MIS* misses.
* ADHD > Controls, $p < 0.05$; (*) ADHD + TD > Controls, $p < 0.10$.

tention errors ($r = 0.28$, $p < 0.05$ for false alarms and $r = 0.35$, $p < 0.01$ for misses), and between sustained attention misses and set shifting errors ($r = 0.26$, $p < 0.05$). Besides, sustained attention standard deviation correlated significantly with both go/nogo reaction time ($r = 0.29$, $p < 0.05$) and go/nogo false alarms ($r = 0.26$, $p < 0.05$).

Analysis of attention performance

The mean scores for all attention parameters and the statistical results are summarized in Table 3. The MANCOVA revealed a significant main effect for group ($F(20,96) = 1.77$, $p < 0.05$, $\eta^2 = 0.27$). Follow-up analyses of variance revealed significant group differences for the number of false alarms in the sustained attention ($F(2,56) = 4.46$; $p < 0.05$) and in the go/nogo task ($F(2,56) = 6.39$; $p < 0.01$). Further single comparisons showed that the ADHD group made more false alarm errors than the control group in the go/nogo task ($p < 0.05$, $\eta^2 = 0.22$; Fig. 2). Besides, a tendency for a group difference between the controls and the comorbid group was revealed, showing that the number of false alarms in the ADHD + TD group was higher than in controls ($p < 0.10$, $\eta^2 = 0.14$). The number of false alarms in the go/nogo task did not differ between the two clinical groups ($p > 0.05$, $\eta^2 = 0.08$). Single comparisons for the number of false alarms in the sustained attention task showed that ADHD children performed worse than the control group ($p < 0.05$, $\eta^2 = 0.17$; Fig. 3). No significant group differences were revealed for the comparison of the two clinical groups ($p > 0.05$, $\eta^2 = 0.10$) or for the comparison between controls and the ADHD + TD group ($p > 0.05$, $\eta^2 = 0.01$). No main effect of group was found for the visual set shifting task.

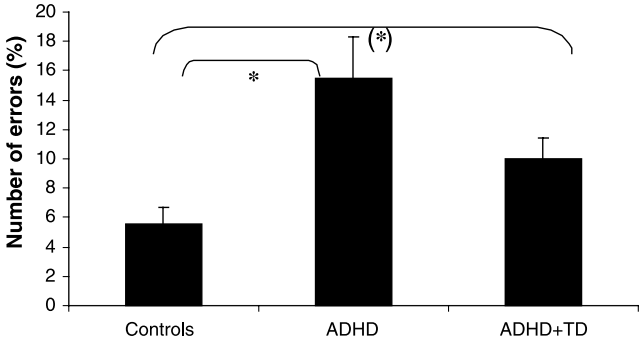

Fig. 2. Percentage of false alarm errors (M, SE) in the go/nogo task. *ADHD* attention-deficit/hyperactivity disorder, *TD* tic disorder, *ADHD + TD* comorbid group. * $p < 0.05$; (*) $p < 0.10$

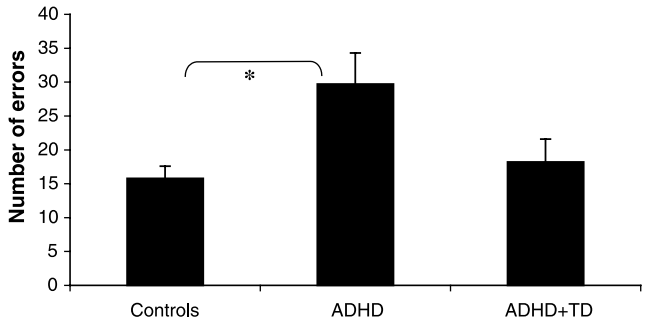

Fig. 3. Number of false alarm errors (M, SE) in the sustained attention task. *ADHD* attention-deficit/hyperactivity disorder, *TD* tic disorder, *ADHD + TD* comorbid group. * $p < 0.05$

Although differences between the ADHD and the ADHD + TD group did not reach significance, it is worth noting that on a descriptive level, for all three tasks the same result pattern regarding the number of errors (including misses and false alarms) and the standard deviation of reaction time emerged: the comorbid group made less errors and had lower standard deviations than the ADHD group.

As reported above, a significant negative correlation was found between reaction time and errors in the go/nogo task, thus indicating a speed-accuracy trade-off. To rule out the possibility that the reported group differences regarding the number of false alarms in the go/nogo and in the sustained attention task could be attributed to a distinct speed-accuracy trade-off pattern in one group (e.g. in the way that especially patients in the ADHD group may have reduced their accuracy in order to fasten their speed), correlations between reaction time and false alarms in these two tasks were calculated separately for the three groups and transformed in Fisher's Z scores. The three Z scores were compared using χ^2. Correlations between speed and false alarms in the sustained attention task did not differ significantly between the groups and were all nonsignificant ($r = 0.08$ for controls, 0.03 for ADHD and -0.23 for ADHD + TD, $p > 0.05$ for comparison between groups). In the go/nogo task, a speed-accuracy trade-off could be shown for all groups. Again, no significant differences for correlations between false alarms and reaction time were revealed ($r = -0.41$ for controls, -0.55 for ADHD and $r = -0.58$ for ADHD + TD, $p > 0.05$ for comparison between groups). Thus, the reported group differences cannot be explained by differential speed-accuracy patterns.

To examine more precisely the origin of patients' deficits in attentional performance, multiple regression analyses were performed including the subjects of the two clinical groups. The dependent variables were the number of false alarms in the go/nogo and in the sustained attention task respectively. Independent variables – which all were entered simultaneously into the model – were subjects' age,

Table 4. *Multiple regression analyses*

	R	R^2	R^2 adj.	F	p	Predict	B	Beta	t	p
Go/nogo false alarms	0.53	0.28	0.19	3.11	0.029	Constant	4.37		0.22	0.828
						Age	−0.73	−0.14	−0.89	0.378
						ODD/Conduct	8.34	0.39	2.09	0.044
						CBCL-Inatt.	−0.07	−0.06	−0.37	0.713
						CBCL-Extern.	0.26	0.20	1.10	0.280
Sustained attention false alarms	0.33	0.11	0.00	1.00	0.424	Constant	−4.20		−0.11	0.916
						Age	−0.69	−0.08	−0.44	0.666
						ODD/Conduct	2.78	0.07	0.35	0.728
						CBCL-Inatt.	−0.20	−0.11	−0.57	0.572
						CBCL-Extern.	0.74	0.31	1.54	0.133

ODD oppositional defiant disorder, *Conduct* conduct disorder, *CBCL-Inatt.* child behaviour checklist inattention score, *CBCL-Extern.* child behaviour checklist externalizing score.
20 subjects with ADHD and 20 subjects with ADHD + TD were included in the analyses.

the diagnosis of ODD/CD (coded as either present or absent), CBCL externalizing and inattention score. The independent variables were selected such as to include age and severity of ADHD psychopathology, both generally known to be related to attentional performance. Including CBCL externalizing score seemed particularly important, since the two clinical groups differed in this rating (see Material and methods), thus raising the question whether the more pronounced impairment of the ADHD group in attentional functions could be accounted for by the relatively higher externalizing symptoms score. ODD/CD, being the most frequent comorbidity in our sample, was included since it has been shown to be associated with deficient inhibitory control (Sergeant et al. 2002).

The results of the multiple regression analyses presented in Table 4 demonstrate that the variables included could significantly predict the number of false alarms in the go/nogo task but not in the sustained attention task. The diagnosis of ODD/CD was strongly associated with an increased number of false alarms in the go/nogo task. Moreover, although this correlation was not proved to be significant, the number of false alarms in the sustained attention task tended to be associated with elevated externalizing symptoms scores.

Discussion

Based on a theoretical framework of attention, the aim of the present study was to compare ADHD children with ADHD + TD children on different attentional tasks. Consistent with previous findings (Como et al. 1993; Sherman et al. 1998; Rothenberger et al. 2000) we found ADHD + TD patients to be less impaired than ADHD patients. When compared to healthy controls, ADHD children performed worse in the sustained attention and go/nogo task (reflected by an increased number of false alarms), whereas ADHD + TD children only were marginally impaired in their performance in the go/nogo task.

Performance of ADHD children

The impaired performance of the ADHD group in the go/nogo task is consistent with an extensive body of literature (Nigg 2006) suggesting a deficit in executive attention in ADHD. Unlike assumed, we found no impairment in the set shifting task, the second test aiming at executive attention, while the majority of studies reported set shifting deficits in ADHD (Sergeant et al. 2002). However, evidence for executive deficits in ADHD is particularly robust when the task involves suppressing a prepotent response (as required in a go/nogo task), whereas evidence is less clear when inhibition comprises suppressing a conflicting response (as required in a set shifting task). Moreover, unimpaired performance in set shifting could also be attributed to the rather small sample size of the present study, especially since there is substantial variability in neuropsychological functioning across and within ADHD samples (Doyle 2006). This may also explain why we did not find evidence for an increased variability of reaction time. The deficit of the ADHD group in sustaining attention is in line with the literature, although effects for this attentional domain have been reported to be smaller than for executive attentional function (Huang-Pollock and Nigg 2003).

Performance of ADHD + TD children

In line with our hypothesis, we did not find marked deficits in executive attentional performance in the comorbid group as they typically are observed in ADHD children. In contrary to our prediction, we found no evidence for slowed

reaction times in the comorbid group across task. Slowed reaction times in children and adults with Tourette syndrome have repeatedly been reported (e.g. Channon et al. 1992; Harris et al. 1995; Georgiou et al. 1996; Shucard et al. 1997) and were often interpreted in terms of cognitive slowing which is not associated with a general cognitive impairment (''bradyphrenia'') (Schuerholz et al. 1996). This interpretation has also been put into question claiming that slowed reaction times do not reflect an attentional deficit per se but rather a conservative response bias resulting in a speed-accuracy trade-off (Shucard et al. 1997). One reason why we did neither find evidence for slowed reaction times in terms of bradyphrenia nor for a conservative response pattern might be the less severe symptom severity in our patients with TD. Previous investigations focused on patients with Tourette syndrome, whereas more than half of our subjects suffered from chronic tic disorder. This difference may plausibly explain our result since Tourette syndrome has been characterized as the more severe condition (Cohen et al. 1985) and it has been shown that tic severity is negatively correlated with neuropsychological performance (Sherman et al. 1998). On a descriptive level, our data indeed indicated that patients with Tourette syndrome showed higher reaction times than patients with chronic tic disorder across all three tasks. However, this association could not be proved to be significant.

Models of ADHD + TD comorbidity and integration of results

The finding that the ADHD + TD group was found to be less impaired than the pure ADHD group raises questions about the nature of ADHD in the comorbid group. Based on psychophysiological, psychopathological and neuropsychological findings, there has been a vivid debate about the coexistence of TD and ADHD in the last years and mainly three attempts trying to explain ADHD and TD co-occurence have been discussed intensively.

(1) The additive hypothesis suggests that TD and ADHD are independent pathological sources. When both conditions co-occur, deficits of both disorders sum up. Evidence for the additive model comes from a TMS study (Moll et al. 2001) showing that ADHD and TD independently contribute to aberrant excitability of the motor system. Studies assessing psychopathological features also corroborate additive effects, stressing that patients suffering from TD and ADHD are affected most seriously in terms of psychosocial functioning, comorbid disorders or psychiatric symptoms and behaviour disturbances (e.g. Pierre et al. 1999; Spencer et al. 1998; Gadow et al. 2002; Roessner et al. 2007).

(2) Another hypothesis posits that ADHD + TD reflects a separate nosologic entity. This hypothesis is supported by ERP studies form the Rothenberger group (Yordanova et al. 1997, 2006) detecting unique activity patterns in the comorbid group.

(3) The phenocopy hypothesis suggests that one disorder causes *behavioural* symptoms of the second ''comorbid'' disorder, which may be reflected by a similar neuropsychological profile of the comorbid group and of one pure group. As outlined above, this explanation is bolstered by previous findings on attentional functioning in patients diagnosed with TD and ADHD (Como 1993; Sherman et al. 1998; Rothenberger et al. 2000) and our results can also be brought in line with this hypothesis. Thus, one might speculate that TD pathology may have confounded ADHD diagnosis and that at least in some patients of the comorbid group, TD may have produced behavioural symptoms of ADHD without its broad neurocognitive deficits. This interpretation is supported by our results on behavioural problems. Parents' CBCL ratings of externalizing behaviour and inattentiveness in the ADHD + TD group were well above the normative range. Moreover, symptoms of inattentiveness in children of the ADHD and in children of the comorbid group were rated as equally severe, which is of particular interest since the attention problem subscale has been shown to have high predictive and discriminative value for ADHD (Steingard et al. 1992; Chen et al. 1994). To sum up, on a behavioural level, the comorbid patients showed typical ADHD related symptoms that were not present on the neuropsychological level.

Our results did not indicate a strong relationship between externalizing symptoms, which were rated as more severe in the ADHD group, and false alarms in the sustained attention and go/nogo task. However, in future studies it would be favourable to match groups for behavioural ADHD symptoms to entirely rule out the possibility that differences in ADHD pathology cause group differences in neuropsychological performance.

Another possible explanation for the results of the present study might be the frontal lobe compensation hypothesis put forward by Leckman et al. (2006). The authors suggest that in patients with TD, the prefrontal cortex compensates aberrant thalamocortical and striatal activity. Evidence comes from a recent EEG study in adults with Tourette syndrome (Serrien et al. 2005). The authors showed elevated frontomesial alpha coherence in these patients during inhibition of a prepotent response in a go/nogo task compared to healthy controls. Coherence in the same network was increased when patients were asked to actively suppress tics. Since behavioural performance

did not differ between groups, Serrien and co-workers (2005) concluded that the identified coherence patterns might be adaptive and compensate for deficient inhibitory control. Relating to our findings, one might also suggest that in the comorbid group similar TD associated adaptive mechanisms might have ameliorated inhibitory control deficits associated with both TD and ADHD. Since only adults were included in the study of Serrien et al. (2005) it is an important future issue to find out whether adaptive mechanisms already occur in children with TD. Studies including children and adults with TD are certainly needed to further examine the frontal lobe compensation hypothesis and to assess developmental changes.

In light of the heterogeneous findings as to the nature of TD + ADHD comorbidity, Yordanova and co-workers (2006) recently proposed a model suggesting that ADHD and TD pathology independently influence basic cerebral functions, like e.g. cortical excitability or sleep-wave regulation. In contrary, cognitive demands may result in complex interaction of both ADHD and TD pathology. In line with other researchers (Rizzo et al. 2007) we agree that the different models of TD and ADHD comorbidity presented above might not always be mutually exclusive. More research combining both behavioural and brain related measures (e.g. imaging studies) is needed to further explore the nature of co-occuring ADHD and TD.

Disentangling the overlap of ADHD and TD in the attention domain but also in several other domains has important clinical implications. Therapeutic strategies, including pharmacological treatments, known to be beneficial for ADHD or TD respectively might further need to be adapted in comorbid conditions. In particular, information about specific or additional problems in comorbid patients on the one hand and possible advantageous adaptive mechanisms on the other hand is crucial for our understanding of difficulties such patients experience in daily life. On an individual level, neuropsychological investigations of attention might be taken into account as an additional information in order to decide whether psychopharmacological treatment should be administered to children comorbid for ADHD and tics.

Limitations

There are some limitations to our study. First and most importantly, we did not include a pure TD group in our study. However, a 2×2 factorial design including healthy controls and patients with ADHD, ADHD + TD and TD is needed to draw more stringent conclusions about the overlap between the two disorders. Although recruitment of a pure TD group is difficult, more studies using a 2×2 factorial design are important for our understanding of ADHD + TD comorbidity.

Second, the majority of our subjects suffered from comorbid conditions and especially ODD/CD was frequently co-diagnosed. Our analysis revealed a strong relationship between ODD/CD diagnoses and increased errors in the go/nogo task. Although the frequency of comorbid conditions did not differ between the clinical groups, future investigations should try to exclude at least those comorbid diagnoses that might substantially influence the measures assessed.

Third, we included patients medicated with tiapride and thus, influences of neuroleptic medication on attentional performance cannot be ruled out. Taking into account that only three patients received tiapride and that we were able to exclude the effect of stimulants, this limitation seems of minor importance.

Conclusions

In summary, children with ADHD + TD were found to be less impaired in attentional performance than children with ADHD only. The results may indicate that the comorbid group has comprised at least some patients that can be characterized as a behavioural phenocopy of ADHD patients. Alternatively, TD related compensatory neural mechanisms may have led to increased inhibitory control in the comorbid group compared to the pure ADHD group. Future studies including also a pure TD group are certainly needed in order to better explain neuropsychological findings in the comorbid group. Besides, further research including patients of different ages and combining different methods is warranted to further characterize ADHD and TD comorbidity in different domains and in order to adopt and further develop therapeutic treatments for this patient group.

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