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RESEARCH NOTE

Postural control anomalies in children with Tourette syndrome

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Abstract The goal of the present study was to determine whether postural control is affected in Gilles-de-la-Tourette syndrome (TS). Center of pressure (COP) displacements were recorded in children with TS and unaffected siblings in three conditions using a force platform: (1) Eyes-Open, (2) Eyes-Closed, (3) One-Leg standing with eyes open. The COP range and velocity were higher in children with TS than in unaffected siblings in all conditions. These differences could not be attributed to age, present tic severity, comorbidities (hyperactivity and compulsions) or medication. The data suggest that sub-clinical postural control anomalies are present in TS.

Keywords Gilles-de-la-Tourette syndrome · Postural control · Basal ganglia

Introduction

Gilles-de-la-Tourette syndrome (TS) is a neurodevelopmental disorder characterized by involuntary motor and vocal tics. TS is often associated with various comorbidities such as obsessive-compulsive behavior and hyperactivity (Leckman 2002; Robertson 2000). Several sources of evidence suggest a dysfunction in fronto-striatal systems in TS. Increased white matter and asymmetry in the frontal lobe was shown in TS (Hong et al. 2002). Also, reduced volume, abnormal asymmetries (Hyde et al. 1995; Peterson et al. 1993; Singer et al. 1993) as well as increased dopamine binding within the caudate nucleus (Malison et al. 1995; Wolf et al. 1996) have been observed in TS. In addition, abnormal blood flow was found in both basal ganglia and the frontal lobe in patients with TS (Buckingham et al. 1993; Diler et al. 2002).

Postural control deficits are typical symptoms in basal ganglia disorders (Visser and Bloem 2005). Clinical balance problems have not been investigated in TS. However, sub-clinical postural control anomalies may be present in TS and could interact with development.

Fundamental changes take place in postural control during normal development (Kirshenbaum et al. 2001). The stability of postural sway improves significantly over the first years (Hayes et al. 1985; Riach and Starkes 1994; Taguchi and Tada 1988). Young children show large and fast corrections of the center of pressure (COP), the point location of the vertical ground reaction force vector, in order to keep the center of mass (COM) within the base of support. As children get older (around 7 to 8-years old), the COP shows smaller and slower deviations during standing (Kirshenbaum et al. 2001). This postural improvement has been linked to a better use of sensory (proprioceptive, visual, vestibular) information and to better sensorimotor integration (Kirshenbaum et al.

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2001; Riach and Starkes 1994; Taguchi and Tada 1988). If present, postural control deficits in children with TS could result in larger and faster deviations of the COP.

The goal of the present study was to examine static balance in children and adolescents with TS. Postural control was measured during quiet stance with eyes open or closed to determine if children with TS rely more on vision for postural control. Participants were also asked to maintain balance on one leg with eyes open in order to examine postural control anomalies in a more challenging non-static condition, i.e. when frequent postural adjustments are necessary because of large deviations between COP and COM.

Method

Participants

Two groups of children ranging from 7 to 16-years old were tested. Thirty-three children with TS were compared to 16 age-matched controls with no history of motor, cognitive or psychiatric disorders. Table 1 presents the demographic and clinical data of the participants. All participants had normal or corrected-to-normal vision. For all participants, written informed consent to participate in the study was obtained according to the rules of the institution. The study was approved by the institutional ethics committee and was carried out along the guidelines of the Helsinki declaration. Exclusion criteria were: (a) inability to provide consent, (b) a history of head injury or other neurological disorders which may mimic TS, (c) induction of tics by drugs or other causes, (d) a psychotic disorder or a pervasive developmental disorder.

Clinical evaluation

Tics and comorbidities were evaluated by trained professionals (neurologists, psychiatrists and neuropsychologists) in both patients and siblings. TS was diagnosed using criteria from the Tourette Syndrome Classification Study Group (1993) and classified as: (1) Definite if they met all criteria, (2) probable if tics are not observed by a specialist, (3) possible if the tics began after 21 years, lasted less than 1 year, or if the person was not conscious of her tics. Only individuals diagnosed with definite or probable TS were considered as positive for TS in this study as all probable TS patients showed multiple tics during at least one year. Tic severity was evaluated using the Yale Global Tic Severity (YGTSS, Leckman et al. 1989). Obsessive-compulsive (OCD) symptoms were evaluated using the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS, Goodman et al. 1989; score from 0 to 40). Attention deficit-hyperactivity disorder (ADHD) symptoms were evaluated through an interview using DSM-IV criteria and hyperactivity–impulsivity criteria were compiled (from 0 to 9).

Apparatus and postural parameters

Center of foot pressure displacements were recorded using a force platform (AMTI, ACG model, Watertown, MA, USA) and data were collected with an acquisition software (NetForce®, AMTI). The data were recorded with a sampling frequency of 50 Hz. The raw data were low-pass filtered at 6 Hz using a dual-pass second-order Butterworth digital filter. Data were processed on a Pentium computer using MATLAB® software, version 6.0.

The sway parameters investigated were the COP range (mm) and the mean velocity (mm/s) of COP

Table 1 Demographic and clinical data of participants

Characteristic	TS ($n = 33$)	Siblings ($n = 16$)	Test statistic
Age in years (SD)	10.18 (2.5)	11.25 (2.6)	$t(47) = 1.38$, $P = 0.17$
Gender (M:F)	23:10	5:11	$\chi^2 = 6.50$, $P = 0.01$
Weight in kg (SD)	38 (12)	44 (14)	$t(47) = 1.46$, $P = 0.15$
Height in cm (SD)	145 (17)	153 (16)	$t(47) = 1.62$, $P = 0.11$
Dominant leg (Right:Left)	32:1	15:1	$\chi^2 = 2.85$, $P = 0.55$
# of children with ADHD (%)	12 (36)	0	
# of hyperactivity-impulsivity criteria (SD)	3.4 (3.3)	0.3 (1.0)	$t(47) = -3.56$, $P = 0.0009$
# of children with OCD (%)	8 (24)	1 (6)	$t(44) = -1.60$, $P = 0.11$
YBOCS score mean (SD)	10.2 (10.4)	5.2 (6.4)	
Medication # (%)	20 (65)	0	
Neuroleptics[a]	7 (23)		
Clonidine	7 (23)		
Stimulants[b]	13 (42)		
Antidepressants[c]	1 (3)		

[a] Pimozide, risperidone,
[b] Methylphenidate, amphetamine, pemoline, concerta,
[c] Venlafaxine

displacements along the anterior–posterior (A/P) and medial–lateral (M/L) axes. According to Kirshenbaum et al. (2001) the range of COP is an indicator of the effectiveness of the postural performance. The mean velocity of the COP was recently recognized as the most reliable COP postural parameter since only two trials are necessary to obtain a consistent measure of postural steadiness (Lafond et al. 2004; Raymakers et al. 2005). More importantly, the velocity of the COP reflects the control strategy used by the children. Slower COP displacement is associated with feedback-based postural control.

Procedure

First, the height and weight of each participant was measured. The dominant foot was determined by asking participants which foot they would use to kick a ball. The task was conducted without shoes. Participants were asked to maintain a quiet upright standing posture and remain as stable as possible for the duration of each trial. Their feet were placed at hip width in a natural position and their arms were at their sides. To obtain identical postural configurations between trials, marks were placed on the force plate to mark the positions of the feet. Participants were also asked to fixate at a red dot (10 mm in diameter) placed 1.5 m in front of them at eye level.

Data collection started when the feet were adequately positioned and when the participant was stable on the platform. Three trials of 30 s were recorded in each of the three conditions (Eyes-Open, Eyes-Closed, One-Leg), which was demonstrated to be sufficient for reliable postural control measures (LeClair and Riach 1996). The order of presentation of the experimental conditions was randomized across participants. In the Eyes-Closed condition, participants were verbally asked to close their eyes and recording was initiated about 2 s later. In the One-Leg condition, participants were instructed to stand on their dominant leg for 30 s or as long as possible. Recording for the One-Leg condition was initiated when a stable position was reached, i.e. about 2–3 s after the dominant leg was lifted.

Data reduction

Because of a high level of variability observed in the COP at the beginning and end of the movement, the first and the last 4 s were removed from the analyses. In the One-Leg condition, the trials in which the dominant leg touched the ground before the end of the trial were also removed from the analyses. Four participants (two controls and two children with TS) were removed from the analyses because they failed to maintain their balance on any of the three trials. For that reason, the One-Leg condition was analyzed separately. Also, trials in the One-Leg condition that differed by more than two standard deviations from the group mean were discarded. The goal of this procedure was to target and remove extreme values derived from aberrant COP displacements. A total of 6.9% of the trials in the One-Leg condition were removed for that reason.

Results

Two-leg conditions

Each dependent variable was first submitted individually to a 2 Groups × 2 Conditions (Eyes-Open, Eyes-Closed) ANCOVA with repeated measures on the condition variable. Age may contribute significantly to COP measures and was used as covariate in the analysis.

Table 2 shows the mean and standard deviations of the COP range and velocity parameters in both the M/L and the A/P axes. The COP range was larger in children with TS than in controls in both axes, $F(1, 46) = 4.3$, $P = 0.046$ for the A/P axis and, $F(1,46) = 4.6$, $P = 0.036$ for the M/L axis. Children with TS also displayed faster COP displacements than controls in both axes, $F(1, 46) = 13.0$, $P = 0.0008$ for the A/P axis and $F(1, 46) = 12.5$, $P = 0.0009$ for the M/L axis.

In addition, the COP range was higher in the Eyes-Closed condition than in the Eyes-Open condition in the A/P axis, $F(1, 46) = 7.9$, $P = 0.01$ but not in the M/L axis ($P = 0.12$). The visual feedback condition did

Table 2 Mean (standard deviation) of the COP parameters obtained for children with TS and controls for all conditions

	Participants	
	TS	Siblings
Range A/P		
Eyes-Open	28.9 (11.1)	21.8 (5.2)
Eyes-Closed	33.6 (14.5)	27.5 (8.7)
One-Leg	50.0 (12.8)	38.8 (8.6)
Range M/L		
Eyes-Open	29.0 (10.5)	22.7 (6.5)
Eyes-Closed	31.2 (11.8)	26.1 (6.2)
One-Leg	56.7 (21.3)	40.1 (14.5)
Velocity A/P		
Eyes-open	15.8 (6.4)	10.9 (2.0)
Eyes-Closed	16.1 (3.7)	12.4 (2.1)
One-Leg	29.9 (8.9)	22.0 (5.0)
Velocity M/L		
Eyes-Open	13.3 (3.8)	10.1 (2.1)
Eyes-Closed	13.8 (2.8)	11.3 (2.3)
One-Leg	39.8 (14.3)	28.0 (11.1)

not affect the velocity of the COP. No significant Group × Condition interaction was observed ($P > 0.05$).

One-Leg condition

For the One-Leg condition, each measure was submitted to an ANCOVA contrasting the two groups with age as a covariate. The number of trials in which participants kept the dominant leg from touching the platform during 30 s. was similar in the two groups (2.56 vs. 2.45 for controls and children with TS respectively, $P > 0.05$) and so was the average trial duration (27.5 s in controls and 27.8 s in children with TS, $P > 0.05$). In the following analyses, only successful trials (trial duration of 30 s) were considered.

As shown in Table 2, the range of the COP was larger and the COP velocity was faster in children with TS as compared to controls in the A/P axis, $F(1,42) = 8.4$, $P = 0.006$; $F(1,42) = 7.51$, $P = 0.009$ for the range and velocity respectively, as well as in the M/L axis, $F(1,42) = 5.8$, $P = 0.02$; $F(1,42) = 5.6$, $P = 0.022$ for the range and velocity, respectively.

In a complementary analysis, we compared the One-Leg condition to the Two-leg Eyes-Open condition to determine whether the complexity of the task had a differential effect on postural control in children with TS. An ANCOVA contrasted the two groups and two conditions (One-Leg and Eyes-Open) with age as covariate. The only significant Group × Condition interaction obtained concerned the COP velocity, $F(1, 42) = 4.6$, $P = 0.038$ and a non-significant trend for range $F(1,42) = 2.7$, $P = 0.10$ in the M/L axis. Children with TS were more affected than controls in the One-Leg condition as compared to the Two-leg condition.

Effects of demographic and clinical variables

Age

Correlations were performed between postural measures and age. There was a strong negative correlation between age and COP velocity for all conditions and variables (r values between -0.42 and -0.84, $P < 0.05$) with the exception of COP velocity in controls for the One-Leg condition ($r = -0.27$, $P > 0.05$). The COP range did not correlate with age in any of the groups or conditions (r values between 0.006 and 0.11, $P > 0.05$).

Gender

In order to determine whether the differences between groups might be caused by the different gender distribution in the two groups, boys and girls were compared within each group with 2 (Gender) × 2 (Eyes-Open, Eyes-Closed) ANCOVAs on each measure with age as covariate. Similar tests on gender were also conducted for the One-Leg condition. These analyses revealed that postural control was similar between boys and girls in each group for all measures ($P > 0.10$).

Tics and comorbidities

Correlations were examined between postural measures and clinical measures of tic severity. None of the postural measures were correlated with present tic severity in the three conditions (r values between -0.02 and 0.30, $P > 0.05$). Also, no significant correlations were found between postural measures and the YBOCS score (r values between -0.19 and 0.18, $P > 0.05$).

In order to determine whether the group differences observed might be caused by a subgroup of children presenting ADHD, analyses were performed without children with ADHD. In these analyses, COP range and velocity were still significantly different between children with TS and controls in both axes.

Medication

Analyses also verified whether the effects observed on the entire sample were due to a sub-group with a specific medication. When participants taking neuroleptics, clonidine, or stimulants were removed from the sample, group differences were still present with only one exception; removal of participants taking neuroleptics affected the group difference in COP range in M/L axis, $F(1, 39) = 2.68$, $P = 0.1$ and A/P axis, $F(1, 39) = 1.64$, $P = 0.21$ in the two-leg conditions. COP velocity was significantly different in the two groups in all comparisons.

Discussion

The results of the present study reveal sub-clinical postural control anomalies in children with TS. Children with TS displayed larger and faster center of pressure displacements than controls. These differences, especially the ones concerning COP velocity, are independent of medication, gender, tic severity and behavioral comorbidities suggesting that sub-clinical postural anomalies are characteristic of TS.

One hypothesis is that the postural control anomalies shown by children with TS are linked to impaired access to sensory information. COP displacements are affected

by the loss of sensory information (Fitzpatrick et al. 1994; Horak et al. 1990; Paulus et al. 1984). Impaired proprioception has often been proposed to explain various movement deficits in basal ganglia disorders (Fellows et al. 1997; Klockgether and Dichgans 1994; Meyer et al. 1992; Poizner et al. 1998). However, several studies suggest that there are no sensory deficits in TS (Abbruzzese and Berardelli 2003). Also, in the present study, children with TS were no more affected by the removal of visual feedback than controls suggesting that a sensory deficit does not explain our results.

Another hypothesis is that TS children may show difficulties in sensorimotor integration, i.e. in correcting center of mass displacements online using sensory information. Children with TS were more affected in the One-Leg condition showing that the TS deficit is exacerbated when postural control is heavily dependent on online corrections. Also, TS children can show sensorimotor integration problems in other contexts. For example, there is evidence that children with TS exhibit excessive grip force when grasping an object, suggesting an impaired ability to transform sensory information into adequate motor responses (Nowak et al. 2005). Excessive grip force is also present in Parkinson's disease (Fellows et al. 1998) and Huntington's disease (Gordon et al. 2000). Faster postural corrections in TS could be viewed as excessive responses to sensory feedback on COM deviations. There is growing evidence that fronto-striatal dysfunctions are associated with deficits in feedback-based corrections of voluntary movements (Desmurget et al. 2004; Lemay et al. 2005; Smith et al. 2000). Future work will be needed to fully characterize the sensorimotor integration problem in children with TS.

Postural anomalies have been found in other neurodevelopmental disorders such as autism (Minshew et al. 2004), dyslexia (Pozzo et al. 2006), and developmental coordination disorder (Geuze 2005). This may suggest that the postural control system is especially vulnerable to neurodevelopmental damage. Postural anomalies can be linked to a number of underlying substrates including cerebellar or a striatal dysfunctions. The cerebellum is involved in rapid motor adjustments in postural control and cerebellar dysfunction could explain the larger COP range and faster COP velocity in children with TS. However, there is little evidence of cerebellar anomalies in children with TS (Diler et al. 2002; Hong et al. 2002; Moriarty et al. 1995). Alternatively, the postural control deficit observed in TS could be related to basal ganglia dysfunction. The basal ganglia are strongly connected to the pedunculopontine tegmental nucleus (PTN) which projects to reticulospinal systems involved in posture (Hyde et al. 1995; Shink et al. 1997). There is evidence of mesencephalic anomalies in TS (Garraux et al. 2006). Also, PTN dysfunction has been associated with postural instability and gait akinesia in Parkinson's disease (Pahapill and Lozano 2000) and stimulation of the PTN in patients with Parkinson's disease can result in a decrease in postural instability (Plaha and Gill 2005). The basal ganglia contribute to many aspects of feedback-based postural control including sensory signal processing, sensorimotor integration as well as attentional modulation (Abbruzzese and Berardelli 2003; Visser and Bloem 2005).

The generability of our findings is of course limited by the fact that TS is a heterogenous syndrome with a variable expression and changes in symptoms over time. This variability may have reduced the effects observed. In conclusion, the present data indicate that children with TS show clear sub-clinical postural anomalies. This observation adds a new dimension to the TS phenotype, which shed some light on the underlying pathophysiology.

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