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Reactive balance performance and neuromuscular and cognitive responses to unpredictable balance perturbations in children with developmental coordination disorder

Abstract

Developmental coordination disorder (DCD) is a common motor disorder affecting balance performance. However, few studies have investigated reactive balance performance and the underlying mechanisms in children with DCD. This study aimed to compare the reactive balance performance, lower limb muscle reflex contraction latency and attention level in response to unpredictable balance perturbations between 100 typically developing children and 120 children with DCD (with and without comorbid autism spectrum disorder) aged 6–9 years. Reactive balance performance was evaluated using a motor control test (MCT) conducted on a computerized dynamic posturography machine. The lower limb postural muscle responses and attention level before, during and after a MCT were measured using surface electromyography and electroencephalography, respectively. The results revealed that relative to typically developing children, those with DCD had a significantly longer MCT latency score in the backward platform translation condition ($p = 0.048$) but a significantly shorter latency score in the forward platform translation condition ($p = 0.024$). The MCT composite latency scores and the corresponding lower limb muscle onset latencies were similar between the groups. Children with DCD also demonstrated a lower attention level during and after sudden backward ($p = 0.042$) and forward ($p = 0.031$) platform translations, compared to typically developing children. Children with DCD were less attentive in response to postural threats, and their balance responses were direction-specific. Balance training for children with DCD might require an additional emphasis on sudden posterior-to-anterior balance perturbations, as well as on problems with inattention.

Keywords:
Dyspraxia; Postural control; Neuromuscular reaction time; Mental concentration

1. Introduction

Developmental coordination disorder (DCD) is a neurodevelopmental disorder affecting approximately 5–6% of primary school-aged children [1]. This disorder, which is more common in boys than in girls, affects motor planning and coordination and severely interferes with a child's daily activities and academic performance [1]. Impaired balance control is the most significant of the many motor deficits presenting in children with DCD, affecting 73–87% of the DCD population [2]. Specifically, reactive balance control is the most concerning issue for parents and children, as it is the first line of defense against unexpected balance perturbations and is essential for many daily activities, such as standing in a moving bus [3,4].

To date, few studies have investigated reactive balance performance and the underlying mechanisms in children with DCD. To the best of our knowledge, only 3 research teams have assessed reactive balance performance and the associated neuromuscular responses in this population [4–6]. Williams and Castro [5] first reported that children with and without DCD exhibited similar latency in postural muscle activation onset in response to an unexpected platform translation. This finding was concurred by Geuze [6], who perturbed participants at the trunk level to elicit postural responses. However, when using a setup similar to that used in the study by Geuze [6], we recently found that children with DCD had delayed lower limb muscle activation onset times, which were related to poor motor (ball) skills [4]. We postulated that this discrepancy in findings between studies could be attributed to differences in experimental setups and methodologies. Therefore, standardized laboratory measures were needed to verify the results.

Balance reactions are not fully automatic reflex actions. Emerging evidence has shown that these reactions require attention, especially in children with disabilities [7,8]. For example, children with dyslexia had significantly impaired balance reactions when their attention was split between a balance task and a secondary counting/reaction time task [7]. Additionally, we found that children with DCD exhibited inferior motor and functional balance performances and were less attentive to movements than were their typically developing peers. Inattention explained 14.1–17.5% of the variances in motor performance (including balance performance) in the DCD population [9]. However, no previous study has specifically examined attention during reactive balance tasks in children with DCD.

Therefore, the present study aimed to compare the reactive balance performances, lower limb muscle reflex contraction latencies and attention levels in response to unpredictable balance perturbations between children with DCD and typically developing children. This study hypothesized that children with DCD would exhibit inferior reactive balance control, a longer leg muscle reflex contraction latency and a lower attention level in response to unpredictable balance perturbations, compared to their typically developing peers.

2. Methods

2.1. Participants

Children with DCD and typically developing children were recruited from local primary schools, non-government organizations that provide rehabilitation services for children with special needs, child assessment centers where DCD was diagnosed,

parent groups and our database of DCD participants via poster-based advertising, invitation letters, WhatsApp and online social media. All children were screened by two experienced physiotherapists via telephone and face-to-face assessments, using the following criteria. The inclusion criteria for the DCD group were: an age of 6–9 years, a formal diagnosis of DCD based on the Diagnostic and Statistical Manual of Mental Disorders 5 [1], a total impairment score corresponding to ≤15th percentile on the Movement Assessment Battery for Children (MABC) [10], a total score of ≤46 (5–7 years 11 months old) or ≤55 (8–9 years 11 months old) on the DCD questionnaire 2007 [11], attendance at a mainstream school, an intelligence level within the normal range and no experience with the Brain Computer Interface system or a similar apparatus. The inclusion criteria for the control group (i.e., typically developing children) were similar to those of the DCD group, except that children in the control group did not have a diagnosis of DCD nor meet the criteria of DCD on MABC.

The exclusion criteria for both groups were: comorbid attention deficit hyperactivity disorder (ADHD) or a T score of ≥70 on the Child Behavior Checklist (CBC) [12]; any significant cognitive, psychiatric (comorbid autism spectrum disorder [ASD] was included), congenital, musculoskeletal, movement, neurological or cardiopulmonary disorder that could affect cognitive or motor performance; receipt of active treatments; demonstration of excessive disruptive behavior or an inability to follow instructions.

Ethical approval was provided by the Human Research Ethics Committee of the University of Hong Kong. A detailed explanation was given to each participant and parent and written informed consent was obtained. Data collection was performed by two experienced physiotherapists and trained research assistants in the Balance and Neural Control Laboratory of the Hong Kong Polytechnic University. All procedures were performed in accordance with the principles of the Declaration of Helsinki [13].

2.2. Outcome measurements

Reactive balance performance was measured using the standardized motor control test on a computerized dynamic posturography (CDP) machine (Smart Equitest, NeuroCom International Inc., Clackamas, OR, USA) [14]. The motor control test (MCT) assesses a participant's ability to recover from an unexpected platform perturbation. Before the test, each participant was instructed to stand with their bare feet placed shoulder width apart, eyes open and arms by the side of the body on the dual forceplates of the CDP machine. Next, the platform was translated posteriorly or anteriorly at 3 amplitudes (in inches)—small (0.5 x height/72), medium (1.25 x height/72) and large (2.25 x height/72)—scaled to the height of the participant. Each platform translation was completed in <1 second, and each testing condition comprised 3 trials. The CDP machine automatically calculated the latency score (in ms), defined as the time between the onset of the platform translation and the force response in each lower limb registered by the dual forceplates. A latency score was then obtained for each lower limb per condition, with a higher score indicating a prolonged reactive postural response [14]. The latency scores of the dominant lower limb during the medium-amplitude anterior and posterior platform translations were selected for analysis because they best reflect the reactive balance response of the children participants. The composite latency score (i.e., the average of all condition-

specific latency scores during medium- and large-amplitude platform translations) [14] was also used in the analysis.

Lower limb postural muscle responses to the MCT support surface perturbation were measured using surface electromyography (EMG) (Biometrics, Newport, UK). An accelerometer (ACL300, Biometrics) was attached to the movable platform on the afore-mentioned CDP machine to register the initiation of translation. Postural muscle activities (i.e., the medial hamstrings and gastrocnemius for backward platform translation, and the rectus femoris and tibialis anterior for forward platform translation [3,4]) were monitored before and after the platform movement. It is because physiologically, a sudden backward platform translation would trigger reflexive contractions of the hamstrings and gastrocnemius, and a sudden forward platform translation would trigger reflexive contractions of the rectus femoris and tibialis anterior, allowing the participant to maintain postural stability [3,4]. Circular Ag/AgCl bipolar surface EMG active electrodes (diameter=1 cm, between electrode distance=2 cm) were placed longitudinally at the center of each muscle belly and a reference electrode was fixed on the ipsilateral lateral malleolus. The skin at the electrode placement sites was prepared by cleansing with alcohol swabs, and hair was shaved whenever necessary to reduce skin impedance [15]. The EMG signals were sampled at 1000 Hz and amplified by a gain factor of 1000. Other parameters included a bandwidth of 20–460 Hz, an input impedance of $>10^{15}$ Ω and a common mode rejection ratio of >96 dB [16].

All electrodes were connected to a DataLOG (Biometrics) that was securely attached to the participant's waist to reduce artifacts. The DataLOG employed both a high-pass filter (20 Hz) and a low-pass filter for frequencies >450 Hz and stored EMG data for offline analysis. Signals from the EMG electrodes and the accelerometer were post-processed using the Biometrics EMG analysis software. The accelerometer signal onset was defined as the point at which the signal amplitude differed from the resting value by 0.20 m/s^2, whereas the postural muscle response onset was defined as an EMG value 2 standard deviations from the mean resting EMG value with a duration of >25 ms [17]. The muscle onset latency, defined as the time interval (in ms) between the onset of the accelerometer signal and the first discernible EMG activity in each muscle, was then extracted [17]. The average muscle onset latencies of 3 medium-amplitude anterior and posterior platform translation trials were calculated and used for analysis.

The attention level during MCT was measured concurrently using a Mindwave Mobile electroencephalographic (EEG) headset recording device (NeuroSky Inc., San Jose, CA, USA). This instrument is valid and accurate for measuring the attention levels of children with DCD [18]. The active electrode of the headset was placed on the left forehead (position Fp1 [19]), and a reference electrode was clipped to the left earlobe. EEG activity in the prefrontal cortex was recorded 3 seconds before, during and 3 seconds after the MCT platform perturbation. EEG signals were sampled at 512 Hz, filtered by a band-pass filter (0.5–30 Hz) and subjected to a notch filter for noise at 50 Hz. Other known noise frequencies were also excluded using proprietary algorithms [20]. Data obtained using the headset were transmitted via Bluetooth to the NeuroView data acquisition software (NeuroSky Inc.) installed on a laptop. The software then transformed raw prefrontal cortex EEG signals into an attention index using a Fast Fourier Transform and preconfigured proportions of EEG alpha (8–12

Hz), beta (12–30 Hz), theta (4–7 Hz) and delta (0.1–3 Hz) activities. This attention index, for which possible values ranged from 0 to 100, was generated during each second of EEG recording and used to classify the attention level as very low (0–20), low (21–40), average (41–60), moderate (61–80) or high (81–100) [20]. The attention levels before the platform perturbation and throughout and after the perturbation process in each platform translation direction were averaged and used for the analysis.

Information about demographic factors, medical histories and exercise habits were obtained by interviewing the participants and their parents. The physical activity level (in metabolic equivalent [MET] hours per week) was calculated with reference to the Compendium of Energy Expenditures for Youth [21]. The body mass index (BMI) was calculated from the body weight and height. In addition, the motor performance of each participant was assessed using the MABC. Parents were invited to complete the DCD questionnaire, 2007 version [11] and a Child Behavioral Checklist [12].

2.3. Statistical analyses

Sample size calculation was performed using G*Power 3.1.0 (Universitat Kiel, Germany) and was based on a statistical power of 0.8 and a 2-tailed alpha level of 0.05. With reference to our previous studies [4,22], a conservative medium effect size of 0.4 was assumed for this study. Therefore, a minimum of 100 participants per group was required.

All data were analyzed using the SPSS Statistics 20.0 software package (IBM Corp., Armonk, NY, USA). Descriptive statistics was used to describe all the variables. Data normality was checked using histograms and/or Shapiro–Wilk tests. Continuous and categorical demographic variables were compared between the 2 groups using the independent t-test and chi-square test, respectively. Next, the following 3 sets of outcome variables were compared between the 2 groups using a multivariate analysis of covariance (MANCOVA): (1) MCT backward and forward platform translation latency scores, (2) corresponding EMG lower limb muscle onset latencies during MCT, and (3) EEG-derived attention scores before, during and after MCT platform perturbation. In addition, the MCT composite latency score was compared between the two groups using the independent t-test. To address the potential confounding effect of comorbid ASD on postural control, the MANCOVA were repeated after separating data collected from children with DCD and ASD (DCD + ASD), children with DCD and without ASD (DCD – ASD), and children with typically development (controls). The MCT composite latency score was compared among the three groups using the one-way analysis of covariance (ANCOVA). The indicated effect sizes for between-group comparisons were calculated using the partial eta-squared (MANCOVA) or Cohen's d (independent t-test) test. By convention, partial eta-square values of 0.01, 0.06 and 0.14 and Cohen's d values of 0.2, 0.5 and 0.8 indicate small, medium and large effect sizes, respectively. A significance level of 0.05 (two-tailed) was adopted for all statistical tests.

3. Results

Between March 2015 and March 2016, a total of 275 children were screened and 220 of them were considered eligible to participate in the study. Of these, 120 children were classified as DCD and 100 were classified as typically developing. Fifty-five children were excluded because they had ADHD or attained a T score of ≥70 on the CBC. Detailed characteristics of the participants are presented in Table 1.

Since significant differences in age and BMI were observed between the two groups, these demographic variables were treated as covariates in the multivariate analyses.

The MCT results revealed that children with DCD had a longer latency score in the backward platform translation condition (14.83 ms, 95% confidence interval [CI]: 1.08 to 28.59, p=0.048) but a shorter latency score in the forward platform translation condition (-12.26ms, 95% CI: -24.30 to -0.22, p=0.024) when compared to typically developing children overall. The composite latency scores were similar between the two groups (Table 2). Subgroup analysis revealed that only children with DCD and without ASD had a longer latency score in the backward platform translation condition when compared to typically developing children (p=0.048). For the forward platform translation condition, children with DCD and ASD demonstrated a shorter latency score (p=0.004). The composite latency score was higher in the DCD – ASD group when compared with the DCD + ASD group (p=0.036) (Table 3). The corresponding lower limb muscle onset latencies (Fig. 1) during the two MCT testing conditions were similar between the DCD group and control group (Table 2); and between the three groups (Table 3).

Regarding the EEG-derived attention scores, children with DCD exhibited lower attention scores when compared with typically developing children both during and after a backward platform translation (-3.24 points, 95% CI: -5.88 to -0.60, p=0.042) and a forward platform translation (-4.39 points, 95 CI: -7.21 to -1.58, p=0.031) overall (Table 2). Further analyses showed that only children with DCD and ASD had a lower attention score when compared with typically developing children during and after a forward platform translation (p=0.015) (Table 3). The attention scores of the two/ three groups were similar before the backward/ forward platform translations (Tables 2 and 3).

4. Discussion

Reactive balance performance

This study presents the novel finding that the reactive balance performance of children with DCD (with and without comorbid ASD) is direction-specific. When compared with their typically developing peers, children with DCD reacted more slowly in response to a backward platform perturbation but more rapidly in response to a forward platform perturbation. The presence of ASD could be a confounding factor that shortened the postural response time in children with DCD. Children with DCD alone had longer response latency than typically developing children. It may be because children with DCD have altered structures and activation patterns in various brain regions and neuronal networks. Specifically, corticocerebellar dysfunction in children with DCD contributes to the deficits in motor control and timing [23]. This may explain why force responses in the legs of children with DCD during the MCT backward platform translation condition were delayed (i.e., reacted slower), as detected by the force platform.

When the platform translated forward, the COG of the participant was displaced backward. Although the timing of movement control could be delayed in children with DCD, they had a decreased maximum excursion of LOS in the backward direction [24]. They needed to respond more rapidly to maintain the COG within the base of support (BOS) to prevent falling in the backward direction. Therefore, the force platform detected an earlier force response in the legs (i.e., faster reaction) in the

legs of children with DCD (with and without comorbid ASD) during the MCT forward platform translation condition. Subgroup analysis further revealed that only children with both DCD and ASD reacted faster to a forward platform translation compared to typically developing children. It may be related to their anticipation of the platform perturbation or other neurological dysfunctions associated with ASD [25].

For the overall reactive balance performance, as children with DCD (with and without comorbid ASD) exhibited faster reactions in one movement direction but slower reactions in another, the MCT composite latency scores, which comprise the average values of all direction-specific latency scores, of children with DCD were no different from those of typically developing children. However, in the subgroup analysis, results revealed that children with both DCD and ASD reacted faster than children with DCD alone. Further study is needed to examine the reactive balance control and the underlying mechanisms in children with DCD and ASD.

Neuromuscular responses to unpredictable balance perturbations

Although the reactive balance performances differed between children with DCD and their typically developing peers, we observed no significant differences in the lower limb muscle onset latencies in response to both the forward and backward platform translations between the two/ three groups. Our findings exactly agreed with those of Williams and Castro [5], who reported that the average onset latency of postural muscle activation in response to a sudden, unexpected platform translation was similar between children with and without DCD. However, our previous study demonstrated that if the unexpected perturbation was executed at the trunk level (instead of a platform perturbation), children with DCD demonstrated longer hamstring and gastrocnemius neuromuscular reaction times than did typically developing children [4]. Therefore, we postulated that children with DCD might respond in a timely manner to a soleus/gastrocnemius stretch (induced by a platform perturbation), but not a hamstring stretch (induced by a trunk perturbation). Certainly, further study is needed to specifically examine the postural stretch reflexes in children with DCD.

Cognitive responses to unpredictable balance perturbations

To the best of our knowledge, this is the first study to investigate the attention levels of children with DCD during a reactive balance task. The results revealed that before the unexpected platform perturbation (at baseline), both children with DCD and their typically developing peers exhibited a similar attention levels. However, both during and after the MCT backward/forward platform perturbations (i.e., during the reactive balance task), the attention levels of children with DCD, especially those children with both DCD and ASD, were much lower than those of typically developing children. These findings were similar to those reported by Fong et al. [9], who demonstrated that children with DCD had significantly lower attention levels during functional tasks and that this phenomenon was associated with poor motor performance. Although the exact underlying neurophysiological mechanisms remain unclear, this difference might may be related to the lower attention capacity [26,27] or under-activation of brain areas responsible for motor tasks in children with DCD [23,28,29]. Further multi-channel EEG device-based investigations involving various reactive balance tasks are therefore needed.

This study had several limitations of note. First, this was a cross-sectional study, and causal relationships between the attention level, neuromuscular performance and reactive balance performance could not be established. Second, the EEG-derived attention index could not differentiate the different types of attentional processing (e.g., focused attention and selective attention) or the different brain areas responsible for attentional processing [30]. Future studies might implement a multi-channel EEG device and analysis of EEG frequency bands or an event-related design with which to study the neural mechanisms during a reactive balance task. Finally, although the sample size was large, the sample was not homogenous and the presence of comorbidities (e.g., ASD) may have confounded the results.

5. Conclusions

Children with DCD (with and without comorbid ASD) reacted more slowly in response to a backward platform perturbation but more rapidly in response to a forward platform perturbation when compared with typically developing children. However, the corresponding lower limb EMG muscle onset latencies did not differ between the two groups. Concurrent EEG recording revealed that children with DCD were less attentive both during and after forward and backward platform translations. Our results imply that balance training for children with DCD might require additional emphasis on sudden posterior-to-anterior balance perturbations, as well as on inattentiveness.

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Conflict of interest

The authors declare that they have no conflicts of interest.

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Tables

Table 1. Characteristics of the participants.

	DCD group (n = 120)	Control group (n = 100)	p value
Age (years)	7.38 ± 1.25	6.73 ± 1.12	<0.001*
Sex (male/female, n)	99/21	79/21	0.511
Body weight (kg)	26.10 ± 7.21	23.31 ± 5.87	0.002*
Height (cm)	124.16 ± 8.94	120.39 ± 8.54	0.002*
Body mass index (kg/m^2)	16.66 ± 2.72	15.87 ± 2.26	0.020*
Physical activity level (metabolic equivalent hours per week)	10.03 ± 9.80	12.78 ± 12.22	0.065
Movement Assessment Battery for Children total impairment score	18.23 ± 8.99	4.70 ± 2.55	<0.001**
Movement Assessment Battery for Children balance subscore	3.75 ± 4.17	0.28 ± 0.59	<0.001**
DCD questionnaire 2007 total score	36.58 ± 9.65	43.46 ± 10.10	<0.001*
Child Behavioral Checklist attention problem score	61.48 ± 10.32	61.07 ± 8.56	0.752
Child Behavioral Checklist attention problem percentile	83.76 ± 16.90	83.29 ± 14.97	0.831
Comorbidities (n and %)			
Autism spectrum disorder	58 (48.3%)	---	
Dyslexia	11 (9.2%)	---	

Means ± standard deviations are presented unless otherwise specified.
Abbreviation: DCD = developmental coordination disorder.
* Indicates p <0.05.

Table 2. Comparison of outcome measurements between children with DCD and typically developing children.

Outcome measure	DCD group (n = 120)	Control group (n = 100)	Mean difference between groups (DCD children – Typically developing children) (95% confidence interval)	p value	Effect size
Motor control test latency scores (ms)					
Backward platform translation	118.67 ± 45.37	103.84 ± 54.86	14.83 (1.26, 28.41)	0.048*	$\eta_p^2 = 0.019$
Forward platform translation	138.85 ± 51.60	151.11 ± 34.28	-12.26 (-24.30, -0.22)	0.024*	$\eta_p^2 = 0.024$
Composite	82.69 ± 68.40	88.69 ± 68.22	-5.99 (-24.54, 12.54)	0.524	d = 0.088
EMG muscle onset latencies during motor control test (ms)					
Backward platform translation					
Hamstrings	123.04 ± 57.09	123.59 ± 58.77	-0.55 (-16.67, 15.56)	0.549	$\eta_p^2 = 0.002$
Gastrocnemius	103.82 ± 37.88	102.01 ± 47.37	1.81 (-10.10, 13.71)	0.925	$\eta_p^2 < 0.001$
Forward platform translation					
Rectus femoris	151.29 ± 44.12	148.42 ± 39.25	2.87 (-8.87, 14.61)	0.741	$\eta_p^2 < 0.001$
Tibialis anterior	131.66 ± 35.55	130.22 ± 36.34	1.44 (-8.64, 11.52)	0.758	$\eta_p^2 < 0.001$
EEG-derived attention score during motor control test					
Before backward platform translation	50.87 ± 18.88	56.92 ± 17.98	-6.05 (-11.40, 0.70)	0.062	$\eta_p^2 = 0.019$
During and after backward platform translation	50.55 ± 9.16	53.79 ± 9.07	-3.24 (-5.88, -0.60)	0.042*	$\eta_p^2 = 0.023$
Before forward platform translation	46.37 ± 15.23	49.46 ± 14.71	-3.09 (-7.40, 1.23)	0.153	$\eta_p^2 = 0.011$
During and after forward platform translation	47.33 ± 9.77	51.72 ± 9.77	-4.39 (-7.21, -1.58)	0.018*	$\eta_p^2 = 0.030$

Means ± standard deviations are presented unless otherwise specified.

Abbreviations: DCD = developmental coordination disorder; EEG = electroencephalography; EMG = electromyography; d = Cohen's d; η_p^2 = partial eta-squared.
* Indicates p <0.05.
** Indicates p <0.025 (Bonferroni adjusted).

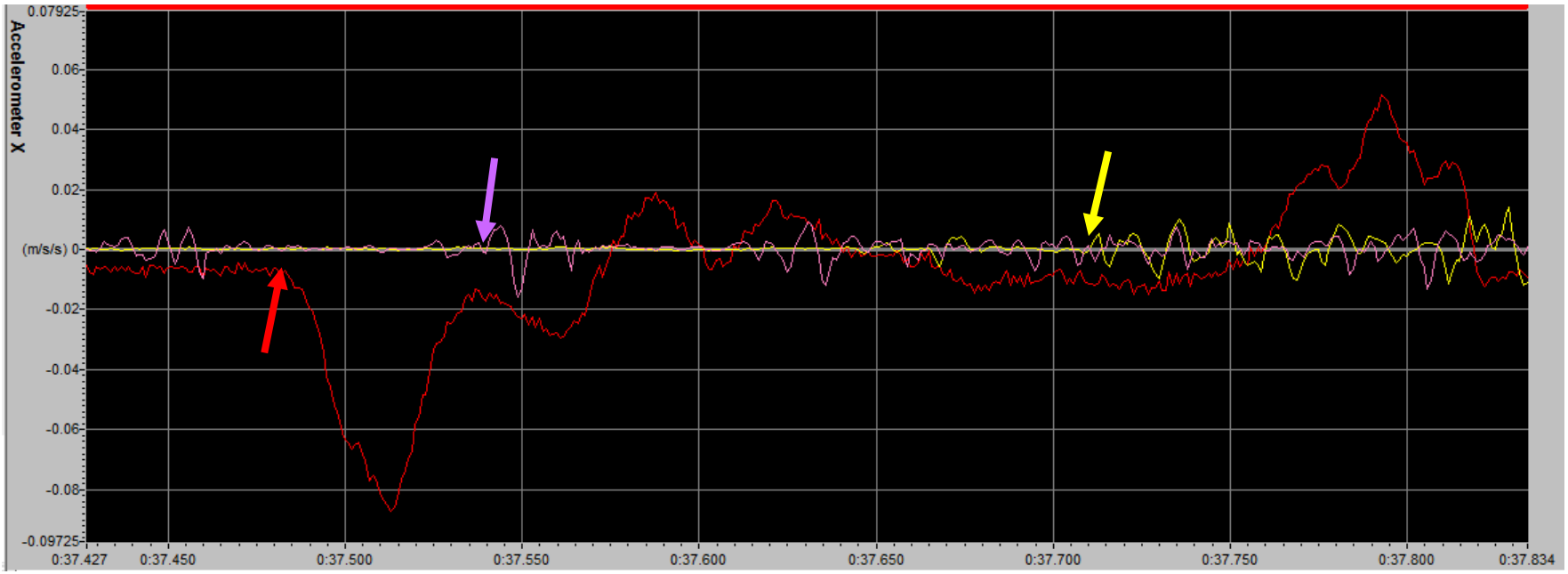
Table 3. Comparison of outcome measurements between children with DCD and ASD, children with DCD and without ASD, and typically developing children.

Outcome measure	DCD + ASD group (n = 58)	DCD – ASD group (n = 62)	Control group (n = 100)	p value	Effect size (η_P^2)
Motor control test latency scores (ms)					
Backward platform translation	112.16 ± 55.87	124.03 ± 33.99[a]	103.84 ± 54.86	0.054*	0.028
Forward platform translation	127.65 ± 62.85[a]	148.06 ± 38.19	151.11 ± 34.28	0.005*	0.051
Composite	64.90 ± 69.91[b]	97.32 ± 64.04	88.69 ± 68.22	0.028*	0.034
EMG muscle onset latencies during motor control test (ms)					
Backward platform translation	129.53 ± 68.37	118.38 ± 47.49	123.59 ± 58.77	0.585	0.005
Hamstrings					
Gastrocnemius	105.16 ± 47.45	102.85 ± 29.58	102.01 ± 47.37	0.979	<0.001
Forward platform translation	151.19 ± 45.11	151.37 ± 43.78	148.42 ± 39.25	0.996	<0.001
Rectus femoris					
Tibialis anterior	134.53 ± 40.50	129.60 ± 31.73	130.22 ± 36.34	0.680	0.004
EEG-derived attention score during motor control test					
Before backward platform translation	49.74 ± 20.55	51.64 ± 17.78	56.92 ± 17.98	0.148	0.021
During and after backward platform translation	50.50 ± 11.24	50.59 ± 7.51	53.79 ± 9.07	0.118	0.023
Before forward platform translation	46.17 ± 15.25	46.52 ± 15.35	49.46 ± 14.71	0.361	0.011
During and after forward platform translation	45.91 ± 10.23[a]	48.33 ± 9.39	51.72 ± 9.77	0.039*	0.035

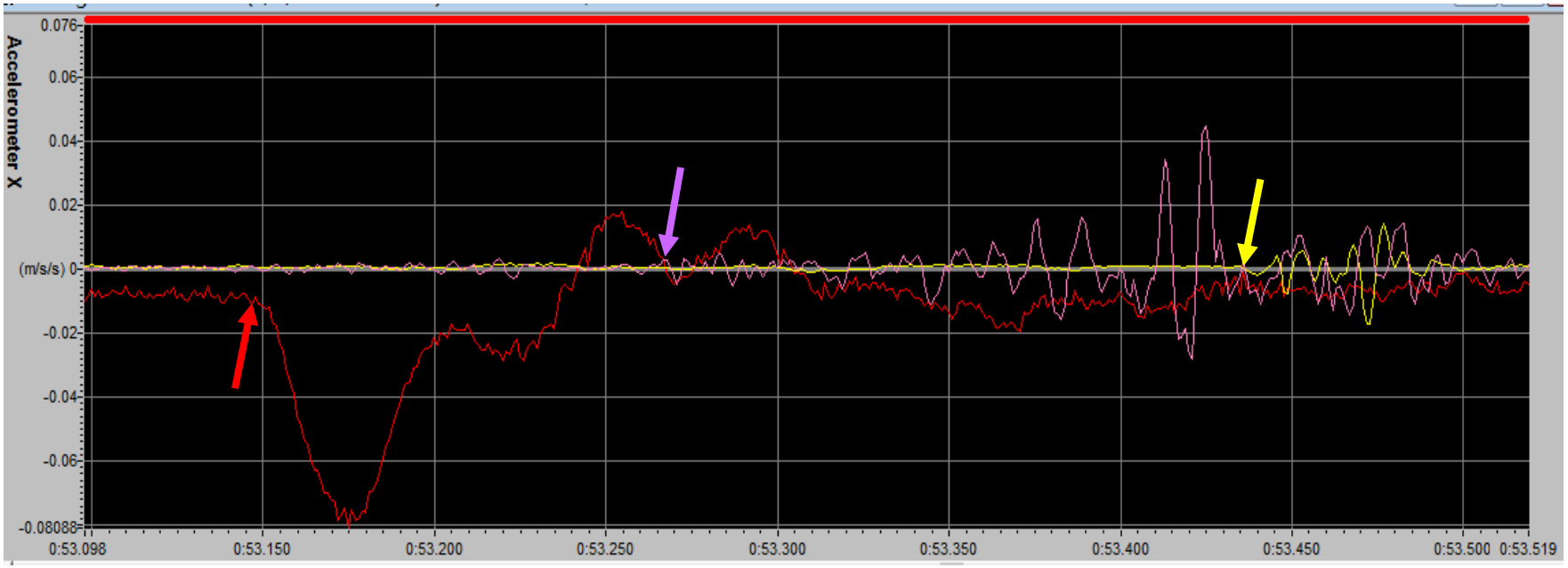
Means ± standard deviations are presented unless otherwise specified.

Abbreviations: DCD = developmental coordination disorder; ASD = autism spectrum disorder; EEG = electroencephalography; EMG = electromyography; η_P^2 = partial eta-squared.
* Indicates $p \leq 0.05$.
[a] Indicates $p < 0.05$ when compared with the control group.
[b] Indicates $p < 0.05$ when compared with the DCD – ASD group.

Figure



(A) A boy with developmental coordination disorder



(B) A boy with typical development

Fig. 1. Comparison of muscle activation patterns in a boy with developmental coordination disorder (A) and a boy with typical development (B), illustrating muscle activation patterns in response to a backward platform translation (forward body sway) during the motor control test. Shown is the hamstrings (yellow curve) and gastrocnemius (pink curve) EMG responses and the accelerometer signal (red curve) with time on the x-axis. The hamstrings and gastrocnemius muscle onset latencies [i.e. the time interval between the onset of the accelerometer signal (red arrow) and the first discernible EMG activity in hamstrings (yellow arrow) or gastrocnemius (pink arrow)] are similar between the two children.