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Cognitive demands impair postural control in developmental dyslexia: A negative effect that can be compensated

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ABSTRACT

Children with developmental dyslexia exhibit delayed reading abilities and various sensori-motor deficits. The way these various symptoms interact remain poorly understood. The objective of this study was twofold. First, we aimed to investigate whether postural control was impaired in dyslexic children when cognitive demands are increased. Second, we checked whether this effect could be reduced significantly by a treatment aiming to recalibrate ocular proprioception. Twelve dyslexic and fifteen treated dyslexic children (>3 months of treatment) were compared with twelve non-dyslexic children in two conditions (mean age: 11.6 ± 2.1, 12.5 ± 1.5 and 10.6 ± 1.7 years respectively). In a first condition they maintained balance while fixating a point in front of them. In the second condition the postural task was combined with a silently reading one. Balance was assessed by means of a force plate. Results demonstrated that the mean velocity (i.e. the total length) of the center of pressure (CoP) displacement was increased in the reading task only for the dyslexic group. Interestingly, for the treated children, an inverse tendency was observed: the mean velocity (i.e. the total length) and the surface of the 90% confidence ellipse of the CoP displacement decreased for 13/15 patients and for 12/15 patients respectively, while performing the reading task. Values remained similar to those observed for the control children. Altogether, these results strongly suggest that cognitive demands can impair postural control in developmental dyslexia but this interaction could be normalized. These results sustain the hypothesis of a cerebellar origin for dyslexia.

Children with developmental dyslexia have a normal level of intelligence but typically exhibit delayed reading abilities and various deficits in the sensory and motor domains [11,23,25]. Classically, the aetiology of dyslexia suggests an alteration of the phonological loop [23]. However, the many symptoms observed in dyslexia have also conducted to suspect a cerebellar origin [15]. Dyslexic children show cerebellar signs, such as motor coordination impairment, reach and gaze overshoot or unbalance [11]. According to this hypothesis, motor and balance impairments were also investigated, with conflicting results. No effect of dyslexia on the balance control were found or only for some cases [18,23,27], suggesting that impairments are not uniquely due to dyslexia but co-occurred with other developmental disorders [24]. By contrast, other studies have emphasized a link between dyslexia and balance impairments [10,2,3,9]. All above-mentioned results were obtained with battery of motor balance tests. These results were reinforced in two recent studies where balance control was assessed using a force plate allowing a more precise evaluation of the center of pressure displacement [19,6].

Using a global approach, some clinical studies have reinforced the possibility of a link between posture deficits and dyslexia [20,21]. Based on some clinical signs (e.g. tonic asymmetry, pseudo-vertigo, etc.) initially depicted by Da Cunha [1], these studies showed abnormalities of their posture for 60 dyslexic children and suggested the possibility to improve postural signs and reading abilities. As dyslexic children have unstable binocular movements [26], a proposed treatment is based on the controversial assumption that clinical signs in dyslexia are due, fully or partially, to an erroneous ocular proprioception of oblique muscles. The fact that ocular proprioception can influence the visual localization sustains this hypothesis [4,5,25]. The idea is to recalibrate ocular proprioception by means of oblique prisms with slight optical deviations in order to compensate for vertical and torsional heterophoria characterizing dyslexic children [22]. To measure heterophoria the principle is to dissociate vision of both eyes while they fixate the same spot of light (Maddox test). For one eye the spot of light becomes a line, by interposing a panel with special optical characteristics whereas the other still perceives the real spot of light. Vertical discrepancies between relative positions of the line and of the light determine the

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positioning of prisms. Their magnitude is function of asymmetries in the tonus of neck muscles and ranges between 2 and 3 dioptries (i.e. 1–1.5° of deviation). As heterophoria have also consequences on postural control [8], complementary modes of rehabilitation are researched: instruction to maintain some specific postures when reading, making specific respiratory trainings and wearing proprioceptive soles to recalibrate the general posture [21].

Co-occurrences of postural deficits and dyslexia do not prove their interdependency. Using balance tests, Nicolson and Fawcett [10] observed that balance is adversely affected by any secondary task which serves to distract attention. They suggested that dyslexic children need to invest more conscious resources for monitoring balance. With the same objective, we used a force plate to demonstrate that the displacement of the center of pressure could be more impaired for dyslexic children when cognitive demands were required. To test this hypothesis, we designed an experiment where control and dyslexic children had to remain as stable as possible on a force plate while either fixating a point or exploring and finding some words written in a panel in front of them. If a link exists between postural control and reading/cognitive abilities, dyslexic children would be less stable in the reading/cognitive task. In addition, to assess effects of the "prismatic" treatment, a group of treated dyslexic children was also tested. It has been previously shown that wearing prism can improve reading performances [16,17,20,21], however, it is unknown whether prisms can modify postural control when an additional cognitive task is performed.

Three groups of children were tested within the guidelines of the declaration of Helsinski. The groups were respectively composed of 12 non-treated dyslexic (mean age = 11.6 ± 2.1 years, range = [8;16]) 15 treated dyslexic (mean age = 12.5 ± 1.5 years, range = [10;15] with mean duration for the treatment = 14.5 ± 11.8 months, range = [3.5;45]) and 12 control (mean age = 10.6 ± 1.7 years, range = [8;15]) children. In France, the diagnosis of dyslexia is given by a speech therapist. The inclusion criteria were at least 24 months of school retardation for literacy impairment with a normal IQ, documented diagnosis and past speech therapy. Importantly, this diagnosis is insufficient to determine whether children are suffering from a 'pure' dyslexia and in many others countries especially in UK and USA they would be diagnosed as co-morbid Specific Language Impairment (SLI) and Dyslexia. This is important in the context of claims by Rochelle and Talcott [24] that the balance difficulties tend to be associated with co-morbid but not 'pure' dyslexia. Due to ethical considerations, some speech therapists refused to communicate IQ but confirmed that IQ for were normal. For the dyslexics, we collected 9/12 values (mean IQ = 112.1 ± 15.8, range = [85;140]) and 8/15 values for the treated dyslexics (mean IQ = 110.8 ± 6.4, range = [102;123]). These two groups were also given one test of language abilities to assess impairments in word reading and spelling abilities (leximetric global validated test "de l'Alouette"). This test made it possible to estimate, for each child, a reading age, based on the time required to read a 265 words text, the number of errors and their level of severity [7]. Reading discrepancies (real age–reading age) were 41.1 ± 13.7 months (range = [27,69]) for the group of dyslexic and 41.5 ± 14.5 months (range = [21;65]) for the group of treated dyslexic.

The center of pressure (CoP) displacement was recorded from a quiet standing posture on a force plate (TechnoConcept®, France) using a sampling frequency of 40 Hz and for a duration of 30 s. Arms were relaxed on each side of the body. The two feet were apart (2 cm between the two heels and the feet axes forming a 30° angle). Children were instructed to remain as stable as possible in two conditions. In a first condition, they had to fixate a point located in front of their eyes at a distance of 40 cm (control). In a second condition, children had to look at a black panel printed on a A4 sheet of paper, of 50 equal rectangular cells (10 rows × 5 columns) also located and centered at 40 cm from the eyes. Each cell was containing a word defining a particular color written in a different color specified by the word itself (e.g. "red" written in green, etc.). This table was 23.2 cm long and 8.5 cm high. Colors were chosen and assigned in a random order for each subject. Four different names of colors ("blue", "red", "green" and "yellow") were written in the table in different colors. The number of times each color were present in the table was different. This procedure was defined to increase the attentional demand while standardizing oculomotor strategies. This was attained by focusing on one word after the other, in relation with a semantic goal in the visual exploration of the panel. The task was longer enough not to be completed in the 30 s of data recording. Children were neither given a time constraint nor any information about the duration of the task, to prevent any effects of stress onto recordings of the CoP displacement. They were instructed to freely explore and find quietly two given words in the small 50 cells panel in front of them. All children did find the correct numbers for a first given word but not for the second, thus suggesting that they were performing the task properly without any stress. We did not record the performance on this task. The two conditions (fixation and reading) were performed in a random order and counterbalanced for the Dyslexic and Control group. For the Treated Dyslexic group (n = 15), one subject more, performed the fixation condition first, then the reading condition. For this latter group, they performed the task without the equipment required for the treatment, namely prisms and soles, since a few minutes (range = [2;5] min).

Basic parameters of the CoP displacement were analyzed: surface of the 90% confidence ellipse, mean velocity (i.e. total length), and standard deviations along the medio-lateral and anterio-posterior axes. The 90% confidence ellipse represents the spatial distribution of the CoP(x,y) positions and excludes extreme data.

There are between-group discrepancies in age (the non-treated dyslexic group was older than the control goup, $F(2,36) = 3.447$, $p = 0.04$). Consequently, all dependant variables were submitted to 3 × 2 ANCOVAs with group (Dyslexic, Control and Treated Dyslexic) and condition (Fixation and Reading) as factors with repeated measures on both factors, and age as covariate, in order to remove the variance for which it accounts. Post hoc comparisons were performed using the Fisher least significant differences test (LSD).

Fig. 1 illustrates mean values ± standard deviations for the mean velocity of the CoP displacement, for each group, in the fixation and reading conditions. Results of the ANCOVA showed a highly significant effect of group ($F(2,71) = 5.71$, $p = 0.005$) and no effect of age, condition or group × condition interaction ($F(1,71) = 2.72$, $p = 0.1$; $F(1,71) = 0.02$, $p = 0.02$ and $F(2,71) = 2.31$, $p = 0.11$ respectively). ANCOVA may have reduced the statistical number of degrees of freedom involved by the inclusion of the regression in the analysis without showing any effect of age. Some part of the difference

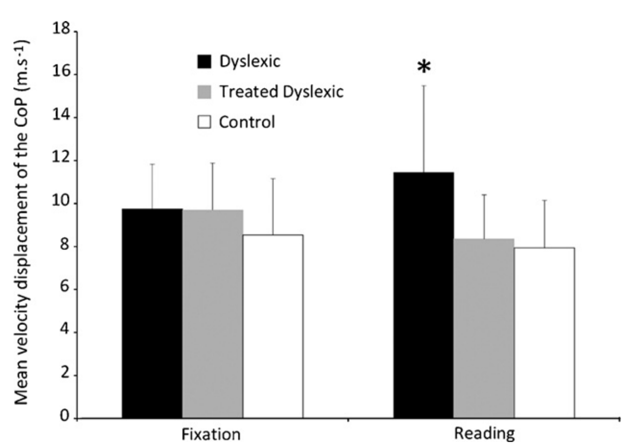

Fig. 1. Mean values ± standard deviations for the mean velocity displacement of the CoP, for each group in the fixation and reading conditions.

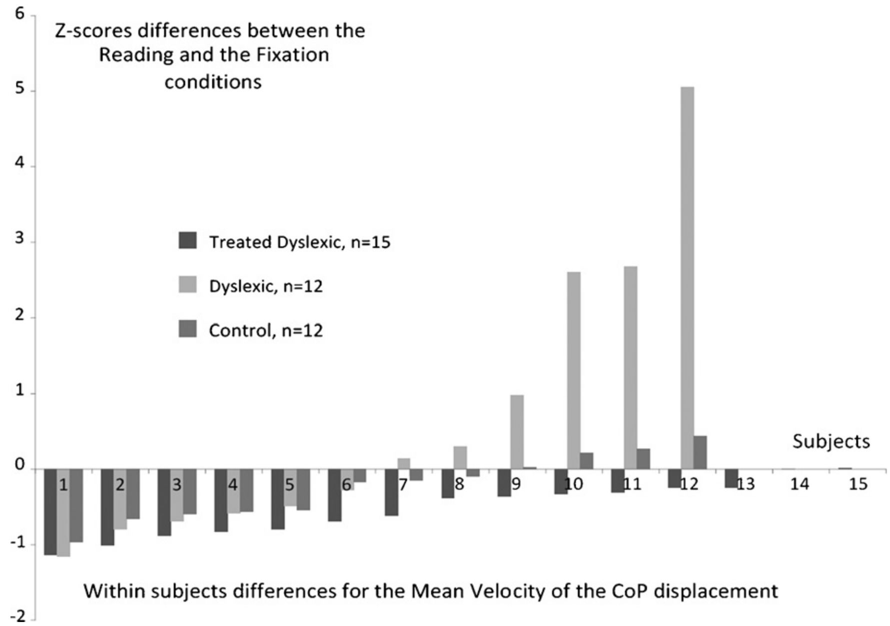

Fig. 2. Within subjects differences for the mean velocity of the CoP displacement between the Reading minus the Fixation condition for each subject of each group. *Z* scores are standardized relative to the mean and standard deviation of the control performance in the fixation condition to determine individual effect sizes. For the sake of clarity data are plot in an increasing order.

between groups in balance ability may be attributable to this age difference and justify an ANCOVA. However, this argument is predicated on the unstated assumption that balance gets worse with age. To rather privilege the use of an ANOVA and legitimate to discount the ANCOVA (i.e. exclude any effect of age in our results) we have reasoned on the following arguments. First, there was no direct evidence of statistical differences between the dyslexic and the control groups as given by the post hoc analyses ($p = 0.19$), even if the dyslexic group was older than the control group. Post hoc analyses only revealed significant differences between the treated dyslexic and the control groups ($p = 0.01$). Second, a correlation analysis performed with age and mean velocity of the CoP displacement for the control group confirmed that balance effectively improved with age in both conditions ($r = -0.59$, $p < 0.05$, mean velocity (fixation) = 18.126 − 0.9056 × age and $r = -0.45$, $p > 0.05$, mean velocity (reading) = 14.141 − 0.5862 × age). Consequently, the age differences would actually if anything bias the results against the findings.

Results of the ANOVA showed a significant effect of group ($F(2,36) = 3.36$, $p = 0.046$), none effect of condition ($F(1,36) = 0.04$, $p = 0.85$) and a significant group × condition interaction ($F(2,36) = 4.06$, $p = 0.026$). A decomposition of this interaction revealed that the mean velocity (i.e. the total length) of the CoP displacement was superior for the dyslexic children when they performed the reading task in comparison to the control and treated dyslexic group for the fixation and reading conditions. A one-way ANCOVA performed only for the reading condition and for the three groups also confirmed this result. It revealed an effect of group ($F(2,35) = 5.5$, $p = 0.008$) and none effect of age ($F(1,35) = 1.47$, $p = 0.23$). Post hoc analyses showed that for the reading condition, the mean velocity of the CoP displacement significantly increased only for the dyslexic group in comparison to the two others ($p = 0.009$ and $p = 0.006$ for the treated dyslexic and the control group respectively). Finally, reading discrepancies of dyslexic children were not correlated with mean velocities of the CoP displacement in the reading task ($r = 0.04$).

Table 1 sums up results for the other parameters, namely: surface of the 90% confidence ellipse and standard deviations along the medio-lateral (*x*-std) and antero-posterior (*y*-std) axes. Results of the ANCOVA for the surface of the 90% confidence ellipse demonstrated a significant effect of age and group but none effect of condition or group × condition interaction. Post hoc analyses revealed that the surface of the 90% confidence ellipse was superior for the dyslexic group in comparison to the control group but not in comparison to the treated dyslexic group ($p = 0.02$ and $p = 0.09$ respectively). Results for *x*-std and *y*-std were never significant for the group, condition or group × condition interactions. These two parameters are sensible to extreme data and to abnormal excursions of the CoP within the base of support. The fact that they were similar for all conditions reveals that all groups performed the pos-

Table 1
Results for the other parameters, namely: surface of the 90% confidence ellipse and standard deviations along the medio-lateral (x-std) and antero-posterior (y-std axes).

	Dyslexic	Treated dyslexic	Control	Effects
90% CE mean ± std (mm²)				
Fixation	140 ± 69	141 ± 68	103 ± 48	**Age: $F(1,71) = 5.06$, $p = 0.028$***; **group: $F(2,71) = 3.7$, $p = 0.03$***; condition:
Reading	183 ± 154	102 ± 61	105 ± 78	$F(1,71) = 0.017$, $p = 0.898$; group × condition: $F(2,71) = 1.5$, $p = 0.233$
x-Std mean ± std (mm)				
Fixation	2.7 ± 0.75	2.7 ± 0.85	2.3 ± 0.97	Age: $F(1,71) = 1.2$, $p = 0.28$; group: $F(2,71) = 0.32$, $p = 0.73$; condition:
Reading	2.7 ± 0.65	2.3 ± 0.84	2.7 ± 1.38	$F(1,71) \sim 0$, $p = 0.98$; group × condition: $F(2,71) = 1$, $p = 0.36$
y-Std mean ± std (mm)				
Fixation	3.6 ± 1.13	3.7 ± 0.96	3.3 ± 1.06	**Age: $F(1,71) = 9.4$, $p = 0.003$***; group: $F(2,71) = 2.7$, $p = 0.076$; condition:
Reading	4.7 ± 3.4	3.1 ± 1.37	3.3 ± 1.54	$F(1,71) = 0.092$, $p = 0.762$; group × condition: $F(2,71) = 1.8$, $p = 0.17$

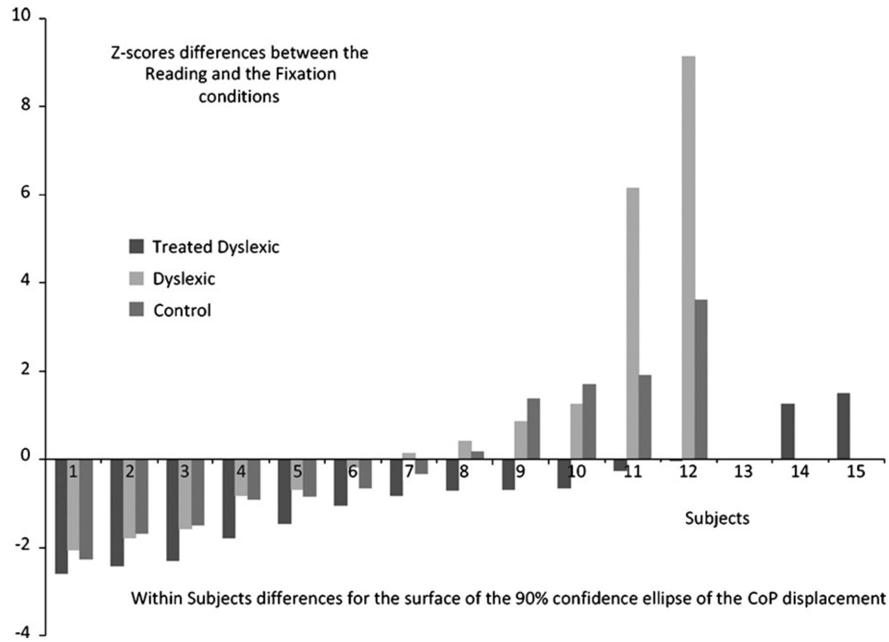

Fig. 3. Within subject differences for the surface of the 90% confidence ellipse of the CoP displacement between the Reading minus the Fixation condition for each subject of each group. *Z* scores are standardized relative to the mean and standard deviation of the control performance in the fixation condition to determine individual effect sizes. For the sake of clarity data are plot in an increasing order. Note that the order of subjects is not the same in Figs. 2 and 3.

tural task similarly without any movements influencing the CoP displacement. To sum-up, the reading task only altered the CoP displacement for the dyslexic group. In addition, values obtained for the treated dyslexic group were similar to those obtained for the control group, despite children were not equipped with prisms and soles during the test.

Strikingly, we also observed for 13/15 of the treated children a marked decrease of the mean velocity of the CoP displacement in the reading task. For the two remaining children the values were closed to 0. This result is illustrated in Fig. 2 and was highly significant (paired *t*-test, $t(14) = 5.64$ $p < <0.001$). This decrease was not proportional to the duration of the treatment ($r = -0.15$).

This result was also paralleled by a diminution of the surface of the 90% confidence ellipse (paired *t*-test, $t(14) = 2.54$ $p = 0.023$) for this group (see Fig. 3). This diminution was also present for 12/15 of the treated children and was not correlated to the decrease of the mean velocity displacement of the CoP in the reading task with the treatment ($r = 0.45$).

The treatment seems to increase the postural stability when children are confronted to the reading task. This is valid with a certain persistence (range = [2;5]min), since children were not equipped during the experiment.

The objective of the study was twofold. First, we tested whether the balance control of dyslexic children is impaired when they have to find simple words written in a panel located in front of them. Second, we tried to assess the effect of at least 3 months of a "prismatic" treatment onto balance control for another group of dyslexic children.

Results clearly demonstrated that the reading task impaired balance for the dyslexic children independently of their reading discrepancies. By contrast, the treatment seems to improve balance in the reading compared to the fixation task. It seems possible to recalibrate the relationship between cognitive demands and postural control in dyslexic children. Our results strongly suggest that cognitive demands and balance control are linked and interact in developmental dyslexia. This is in accordance with initial results of Nicolson and Fawcett observed for more global motor test [10].

The reading task modifies the behavior at various levels: oculomotor strategies, vergence, and attention can influence balance control for dyslexic children. Stein et al. [26] have argued for many years that unstable binocular vergence was a key issue for dyslexia. Kapoula and Bucci [6] had shown that postural stability of dyslexic children was improved when eyes are moving, as alternated fixation of eyes to a near and far point improved their balance control. They also demonstrated that balance control in dyslexic children became impaired when vergence decreased and when the fixation point was localized further, at 1.5 m in front of the subjects. They concluded that eyes movement could improve balance control for this population. These results suggest that oculomotor strategies and vergence modifications could have a marginal effect in our results. Consequently, we suspect more attentional or cognitive causes to explain impairments of balance control in the reading task, since our task do not disambiguate the influences of cognitive and attentional constraints.

In any cases, our results are rather in agreement with the theory of a cerebellar origin of the developmental dyslexia in which sensori-motor and cognitive deficits interact [11–15]. According to Nicolson and Fawcett [14] suggestions many symptoms could be idiosyncratic, namely not present in all dyslexic patients with the same clinical signs. It stresses the importance of a clinical, differential and global approach of dyslexia.

The results also demonstrated an effect of the treatment: quasi-systematic decreases were observed for the mean velocity and for the surface of the 90% confidence ellipse of the CoP displacement, when performing the reading task. This effect was persistent since the treated children were not equipped with prisms and soles when performing the tests. This result suggests that it is possible to improve balances abilities by means of prisms when an additional cognitive task is performed.

Pestalozzi [16,17] found a good influence of prisms onto dyslexia in 71% of patients ($n = 370$). He mentioned that "[. . .] prismatic corrections may save energy [i.e. less attentional and cognitive resources are used] as the patients have no longer to compensate their heterophoria themselves. Thus they dispose on more energy e.g. for understanding the text they are reading".

In conclusion, cognitive and probably attentional demands impair the control of the CoP displacement in developmental dyslexia but this effect could be reduced significantly by means of a "prismatic" treatment. More extensive researches are needed to investigate these phenomena and explore the possible causality between the many sensori-motor and cognitive impairments that characterize the developmental dyslexia.

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