

Static postural control in children with developmental dyslexia

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Abstract

The present investigation tries to better understand potential association and causal relationship between phonological and postural impairment due to developmental dyslexia. The study included 50 boys with developmental dyslexia and selected on the basis of their overall reading difficulties, and 42 control boys. Body sway during a quite standing posture eye open and eye closed on a force platform were tested in the two groups of subjects that were between 10 and 13 years of age. Analysis of classical parameters quantifying the centre of pressure (CP) displacements along antero-posterior and lateral axes showed a significant difference between the two groups. Dyslexic children showed on average greater instability, with greater length, variability and mean power frequency of CP displacements with or without vision. Our results demonstrate that postural parameters may discriminate between children with dyslexia and age-equivalent controls.

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Classically, developmental dyslexia is considered as resulting from specific impairment of phonological representations. Dyslexic children have learning disorder despite the absence of exogenous factors as lack of educational access for instance. It is usually diagnosed when reading achievement is low and has been supposed to be a neurological deficiency from a genetic origin [11,13,23]. However, recent studies have in fact emphasized that children with dyslexia may have more general deficits in the auditory, visual and motor system [22,26,30], based upon evidence of reduced sensitivity to external stimuli and a decrease in the speed of information processing [33]. Interestingly, reliable deficits in certain motor skill have been described like for instance postural unbalance, the efficiency of which relies on a multimodal integration of all available sensory input [18,19]. A clear association between postural control and dyslexia was however inconclusive. Thus, abnormal postural disorders were found in all dyslexics [8,9,20], while other studies found postural disorder in some dyslexics

[27,28] and one study did not find any disorder [24]. Besides such discrepancies, one can mention very different tests and procedures used in these studies. For instance, postural stability where the subject was perturbed by calibrated pushes delivered to the back of the subject and then evaluated using a subjective scale [8,9,27]. In another study significant differences in head acceleration were found between control and dyslexic children during quiet standing eye open but not eye closed [31]. The two studies that quantified body sway with a force platform in static or dynamic conditions, did not show differences between dyslexics and control adults [24,27]. The present investigation tries to better understand potential association and causal relationship between reading impairment and postural deficiency by using the classical tools of stabilometry [2,14].

Our experimental protocol was administered in accordance with the guidelines of the Declaration of Helsinki. Fifty native French speakers males suffering from phonological or visual-lexical or mixed developmental dyslexia were recruited for the study (mean age = 11.5 ± 1.8 years; from 9.2 to 14.8 year old). The inclusion criteria were (1) childhood history of at least 2 years of school retardation for literacy impairment, and (2) documented diagnosis and past speech therapy. The dyslexics were also given two tests of written language abilities to assess the persistence of impairment in word reading and spelling abilities. All

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of them have been examined in the Ophthalmology Department of the University Hospital in Dijon by trained neuropsychologists. Forty-two native French speakers age- and sex-matched controls (mean age 12.4 ± 1.7 years) were recruited in schools of the area. The exclusion criteria for these participants were the same that dyslexics must display to be included in the study. To verify the lack of any reading or spelling impairment, the controls were administered with the two same tests than dyslexics.

Neuropsychological assessment consisted first in subjects administration with a classical regular and irregular isolated printed word reading task in which there were 24 words of each condition to read aloud. Then, they were administered with a computerized spelling decision task in which they had to detect 40 correctly written words mixed with 40 pseudowords (e.g., “axident” instead of ‘accident’). Moreover, the computerized task offers to record reaction times (RT) in the objective to show a significant difference between the groups. Actually, dyslexics are reported to be slower than non dyslexics in this kind of task. The results showed significant differences between the groups concerning the two tasks. In the classical reading task, dyslexics performed poorer than controls as expected (Dyslexics: mean number of errors = 26 ± 15.4 , Controls: mean number of errors = 4.2 ± 3.7 , $U = 7.5$, $z = -6.1$, $p < 0.0001$). For the computerized spelling decision task, the dyslexics showed worse results than controls in the level of performance (Dyslexics: mean number of errors = 26 ± 8.3 , Controls: mean number of errors = 11.6 ± 3.6 , $U = 34.5$, $z = -5$, $p < 0.0001$) as in the reaction time (Dyslexics: mean RT = 2433 ± 759 ms, Controls: mean RT = 1597 ± 455 ms, $U = 91$, $z = -3.8$, $p < 0.0005$).

Body sway was recorded from a quite standing posture with their arms relaxed comfortably at their sides, the two feet apart (2 cm between the two heels and the feet axes forming a 30° angle) on a force platform (TechnoConcept® France) with the eyes open or closed. Two postural conditions (bipodal and monopodal) were also tested. Centre of pressure (CP) displacements were recorded at 40 Hz during 25.6 and 12.8 s in bipodal and monopodal posture, respectively. The following parameters were computed: total length, standard deviation along lateral and antero-posterior axes, and mean power frequency (MPF) which was calculated as follows: $MPF = (F_1P_1 + F_2P_2 + \dots + F_nP_n) / (P_1 + P_2 + \dots + P_n)$, where F is the frequency and P the related spectrum [3].

In order to determine if dyslexic children did or did not show abnormal performance, we adopt a criterion of deviance. A common established procedure consists to set a threshold at n standard deviation of the mean of the control group ($n = 1.65$ S.D. as in Ramus et al. [28]). Performance of control subjects that exceeded this value was rejected (three subjects among the 42 tested). Thus, control mean and S.D. were recalculated and dyslexics who were outside ± 1.65 S.D. were identified. This procedure was applied for each calculated postural variables. The basic experimental design was a 2-factors (Groups \times Visions) within subject design with two replications per cell. Both factors have two levels: Dyslexic (D) versus Control subject (C) for the Group factor, eye open (EO) versus eye closed (EC) for the Vision factor. Due to the inability of some subjects to perform unipodal task during 12.5 s, we only quanti-

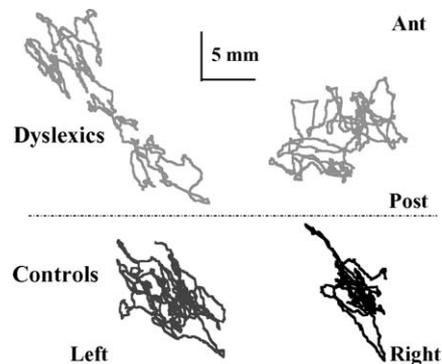


Fig. 1. Representative example of the centre of pressure displacements in the horizontal plane of two dyslexic (grey traces, upper part, subject one on the left and subject two on the right) and two control (black traces, lower part) children, in eyes open condition and during 25 s (Ant, anterior; Post, posterior).

fied the number of successful trials. Thus, each experiment was constituted by 8 trials (2 visions \times 2 postures \times 2 trials). The level of statistical significance selected was $p \leq 0.05$.

Figs. 1 and 2 illustrate qualitatively and quantitatively the postural performance of the two groups of subjects. A global inspection of the different parameters indicates greater instability in eyes closed compared to eyes open and in the dyslexics compared to the control subjects. For each group, we found a significant effect of the lack of visual input that degraded postural stability on each of the calculated variables (length, $p < 0.001$; S.D.x, $p < 0.001$; S.D.y, $p < 0.001$; MPFx, $p < 0.001$; MPFy, $p < 0.001$).

Control subjects presented greater stability compared to dyslexics for all parameters ($p < 0.01$) except the standard devi-

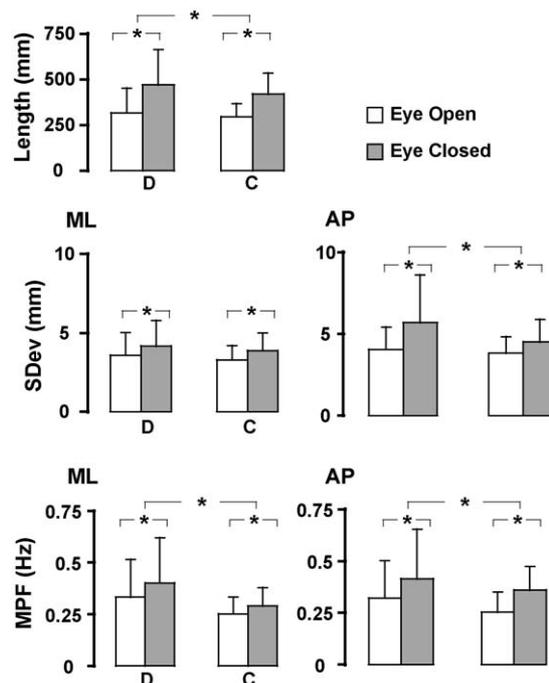


Fig. 2. Mean postural performance of the dyslexics (D) and control (C) subjects in the two visual conditions. From top to bottom, histograms of each calculated stabilometric variables: length, standard deviation (S.D.) and mean power frequency (MPF) of the centre of pressure displacements along medio-lateral (ML) and antero-posterior (AP) axes.

Table 1
Postural performances of the two groups during the unipodal stance in eye open (EO) and eye closed (EC) conditions

	Right foot		Left foot	
	EO (%)	EC (%)	EO (%)	EC (%)
Controls ($n = 39$)	100	75	98	65
Dyslexics ($n = 50$)	90	20	94	40

The percentage indicates the proportion of subjects able to perform the task during 12.5 s on the right foot or left foot.

ation along the lateral axis (length, $p < 0.01$; S.D.y, $p < 0.001$; MPF_x, $p < 0.001$; MPF_y, $p < 0.04$). An interaction was found for the length of the CP displacements between the visual conditions and the groups ($p < 0.03$). Table 1 shows the postural performances of the two groups expressed as a percentage of subjects able to achieve the task during 12.5 s on the right foot or left foot during the unipodal stance with and without vision. Roughly, all control subjects were able to perform the task eye open during 12.5 s on the right foot while in EC condition 25% of them fell down before the end of the recording session. The performance followed the same trend on the left foot; however it was worse compared to the right foot and a greater percentage of falls occurred before the end of the recording period especially in EC condition (35%). In contrast, an important percentage of dyslexic children were unable to perform the task in eye closed condition in both on the right (80%) or the left foot (60%), indicating poorer balance control. Fig. 3 gives the distribution of dyslexic children for which calculated postural variable values for the two visual conditions were outside the criterion of deviance. For all postural variables tested but the Mpf along lateral axis, more than half of the total number of dyslexics presented deviant performances whatever the visual conditions. When considering all postural variables and the two visual conditions, one value at least was found deviant in the 50 dyslexics tested.

In order to reveal potential relationship between cognitive and sensory motor aspects of dyslexia, we calculated the correlation between the three neuropsychological variables (errors during the classical reading task, the computerized spelling decision and the reaction time) and the four postural variables recorded eye open and eye closed in bipodal condition. No significant correlation emerged from this analysis.

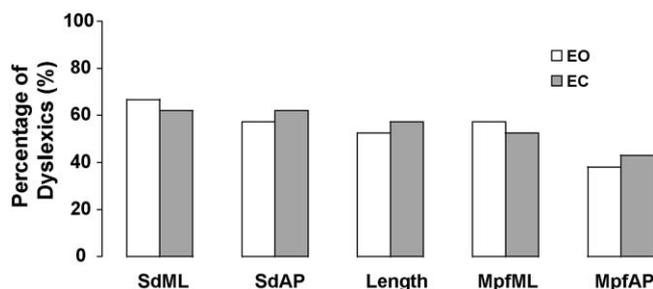


Fig. 3. Distribution of dyslexic children with respect to the deviant postural variables for the two visual conditions: standard deviation and mean power frequency along medio-lateral (SdML and MpfML) and antero-posterior (SdAP and MpfAP) axes, and length of the centre of pressure displacements in the horizontal plane.

The present study shows significant postural instability in dyslexics compared to control subjects during bipodal and unipodal standing postures. These results are in accordance with other investigations that revealed postural unbalance in 50% or more of participants [12,32]. However, these studies demonstrated a proportion of dyslexic participants without balancing impairment, while all children of this sample presented at least one deviant postural variable among these usually studied in patients with balance disorder [1,6,29].

The current possibility to fully associate developmental dyslexia with postural disorders might be due to the greatest sensitivity provided by a conventional body sway analysis, not yet systematically adopted to investigate this pathology. Thus, previous studies were performed during unexpected postural disturbance [24] or a manual push exerted by the experimenter on the subject, and used clinical assessment, scoring [8,9,32] or head accelerations measurements [31].

In both groups we found a significant effect of eye closed condition on bipodal stability, as previously shown in healthy children [10]. Lack of visual input had similar effect on postural stability in dyslexic children as it did in healthy controls. Further, when subjects stood on one foot, lack of visual input affected more dyslexic than controls children. These results indicate that when equilibrium constraints increase (as in bipodal posture), dyslexic children become more dependent of visual input. Interestingly, visual deficiency has been reported in these children [7] as a consequence of abnormalities of the magnocellular component of the visual system. Reduce visual contrast sensitivity, visual transient processing defects, visual attention and spatial localization deficits in addition to poorer eye movement control were reported too [4,16,17]. Therefore, one can expect less effect of lack of visual input on equilibrium. Indeed, because of poor visual control in dyslexics other inputs (e.g., tactile, proprioceptive or vestibular) could be more fully utilized in maintaining balance to compensate visual deficiency. The present result does not confirm such hypothesis in the case of dyslexic children. One interpretation would be that sensory compensation is in progress but not yet stabilized in children belonging to the range of age tested here. This possibility could be verified by analyzing adult dyslexic postural performances in EO and EC conditions in order to document adaptation processes and the role of the other sensory inputs in dyslexic postural control. Alternatively, a visual dependency of adult dyslexics would minimize the role of compensatory processes along maturation and would open up the way to innovative rehabilitation methods applied during this period [25].

Dyslexics are notoriously clumsy and uncoordinated, their writing is appalling, and they show cerebellar signs, such as reach and gaze overshoot or unbalance [22]. Impairment of oculomotor proprioception has also been described in dyslexics [5,25]. Greater instability found here agrees a priori with a cerebellar hypothesis of dyslexia [22]. Indeed, because cerebellum is largely involved in equilibrium function [6] in addition to the acquisition and automation of elementary articulatory and auditory skills, it seems reasonable to assume that this structure plays a key role in dyslexics' postural deficiency. Otherwise, there is some evidence suggesting that magnocellular tempo-

ral processing deficits in dyslexics are not confined to vision and audition, but extend to other systems, such as vestibular and proprioception, as well. Deficit in sensory processing of group II spindle afferent fibers from foot and calf muscle, which provide an appropriate input to CNS for detecting body oscillations around the ankle [12], could also explain dyslexic postural instability. Further, the shear force acting on sole of the foot and associated tactile input that is another important factor for maintaining CM acceleration at zero [15] may also be altered.

Nonetheless, we hypothesize that such sensory deficiencies do not concern short-latency reflexes through feed back regulation, which seems not sufficient to ensure equilibrium, but a higher level of the CNS that must integrate all available sensory inputs to construct a global postural estimation and predict body oscillations [21]. Even if the maintenance of a quiet standing posture appears strongly automated, central mechanisms like spatial body orientation contribute to postural control [21]. The multiple sensory processing impairments would have altered such postural body schema. Because of abnormalities of magnocellular stream and consecutively in processing all sensory systems [33], postural body schema might be damaged during early development.

In conclusion, the present stabilometric measurements that provide greater sensitivity to quantify body oscillations compared to qualitative score, allowed to show that all dyslexic children tested here were affected by postural instability.

The finding of an association between the lack of balance recorded during quiet standing posture and developmental dyslexia differs from several previous investigations that did not succeeded in demonstrating a clear correlation between reading impairment and postural disorders. This difference could be due to the inclusion of a large number of subjects in our study and also due to the relative homogeneity in the age of the subjects. Therefore, postural evaluation could represent a simple novel tool to evaluate this pathology during its progression as well as to better understand its underlying mechanisms. In fact because numerous daily life activities depend on an efficient body equilibrium, one can suspect several other motor disorders, still to be discovered in dyslexic children. For instance further investigations combining 3D kinematic analysis and electromyography with the present one could reveal significant alteration of the locomotor pattern.

Finally the idea that dyslexia has multiple causes and behavioural symptoms is now broadly accepted. Because equilibrium is a multimodal function and results in a subtle integration of several sensory inputs, it is not surprising to find a strong correlation between dyslexia and postural unbalance. In this context poor balance would represent the common manifestation of a neurodevelopmental syndrome that results from several different impairments.

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References

- [1] R.W. Baloh, K.M. Jacobson, K. Beykirch, V. Honrubia, Static and dynamic posturography in patients with vestibular and cerebellar lesions, *Arch. Neurol.* 55 (1998) 649–654.
- [2] G. Bizzo, N. Guillet, A. Patat, P.M. Gagey, Specifications for building a vertical force platform designed for clinical stabilometry, *Med. Biol. Eng. Comput.* 23 (1985) 474–476.
- [3] J.J. Collins, C.J. De Luca, Open-loop and closed-loop control of posture: a random-walk analysis of center-of-pressure trajectories, *Exp. Brain Res.* 95 (1993) 308–318.
- [4] P. Cornelissen, A. Richardson, A. Mason, S. Fowler, J. Stein, Contrast sensitivity and coherent motion detection measured at photopic luminance levels in dyslexics and controls, *Vision Res.* 35 (1995) 1483–1494.
- [5] M.H. Da Cunha, O.A. Da Silva, Les syndrome de déficience posturale, *J. Fr. Ophthalmol.* 9 (1986) 747–755.
- [6] H.C. Diener, J. Dichgans, M. Bacher, B. Gompf, Quantification of postural sway in normals and patients with cerebellar diseases, *Electroencephalogr. Clin. Neurophysiol.* 57 (1984) 134–142.
- [7] G.F. Eden, J.W. VanMeter, J.M. Rumsey, J.M. Maisog, R.P. Woods, T.A. Zeffiro, Abnormal processing of visual motion in dyslexia revealed by functional brain imaging, *Nature* 382 (1996) 66–69.
- [8] A.J. Fawcett, R.I. Nicolson, Performance of dyslexic children on cerebellar and cognitive tests, *J. Mot. Behav.* 31 (1999) 68–78.
- [9] A.J. Fawcett, R.I. Nicolson, P. Dean, Impaired performance of children with dyslexia on a range of cerebellar tasks, *Ann. Dyslex.* 46 (1996) 259–283.
- [10] M. Ferdjallah, G.F. Harris, P. Smith, J.J. Wertsch, Analysis of postural control synergies during quiet standing in healthy children and children with cerebral palsy, *Clin. Biomech. (Bristol Avon)* 17 (2002) 203–210.
- [11] E.L. Grigorenko, The first candidate gene for dyslexia: turning the page of a new chapter of research, *Proc. Natl. Acad. Sci. U.S.A.* 100 (2003) 11190–11192.
- [12] V.S. Gurfinkel, P. Ivanenko Yu, S. Levik Yu, I.A. Babakova, Kinesthetic reference for human orthograde posture, *Neuroscience* 68 (1995) 229–243.
- [13] M. Habib, The neurological basis of developmental dyslexia: an overview and working hypothesis, *Brain* 123 (Pt 12) (2000) 2373–2399.
- [14] T. Kapteyn, W. Bles, C. Nijokiktjen, L. Kodde, C. Massen, J. Mol, Standardization in platform stabilometry being apart of posturography, *Agressologie* 24 (1989) 321–326.
- [15] A. Kavounoudias, R. Roll, J.P. Roll, Foot sole and ankle muscle inputs contribute jointly to human erect posture regulation, *J. Physiol.* 532 (2001) 869–878.
- [16] M.S. Livingstone, G.D. Rosen, F.W. Drislane, A.M. Galaburda, Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia, *Proc. Natl. Acad. Sci. U.S.A.* 88 (1991) 7943–7947.
- [17] W.J. Lovegrove, A. Bowling, D. Badcock, M. Blackwood, Specific reading disability: differences in contrast sensitivity as a function of spatial frequency, *Science* 210 (1980) 439–440.
- [18] C. Maurer, T. Mergner, B. Bolha, F. Hlavacka, Vestibular, visual, and somatosensory contributions to human control of upright stance, *Neurosci. Lett.* 281 (2000) 99–102.
- [19] T. Mergner, C. Maurer, R.J. Peterka, A multisensory posture control model of human upright stance, *Prog. Brain Res.* 142 (2003) 189–201.
- [20] R. Moe-Nilssen, J.L. Helbostad, J.B. Talcott, F.E. Toennesen, Balance and gait in children with dyslexia, *Exp. Brain Res.* 150 (2003) 237–244.
- [21] P.G. Morasso, M. Schieppati, Can muscle stiffness alone stabilize upright standing? *J. Neurophysiol.* 82 (1999) 1622–1626.
- [22] R.I. Nicolson, A.J. Fawcett, E.L. Berry, I.H. Jenkins, P. Dean, D.J. Brooks, Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults, *Lancet* 353 (1999) 1662–1667.
- [23] E. Paulesu, J.F. Demonet, F. Fazio, E. McCrory, V. Chanoine, N. Brunswick, S.F. Cappa, G. Cossu, M. Habib, C.D. Frith, U. Frith, Dyslexia: cultural diversity and biological unity, *Science* 291 (2001) 2165–2167.

- [24] A. Poblano, K. Ishiwara, M. de Lourdes Arias, F. Garcia-Pedroza, H. Marin, M. Trujillo, Motor control alteration in posturography in learning-disabled children, *Arch. Med. Res.* 33 (2002) 485–488.
- [25] P. Quercia, A. Seigneuric, S. Chariot, P. Vernet, T. Pozzo, A. Bron, C. Creuzot-Garcher, F. Robichon, Ocular proprioception and developmental dyslexia. Sixty clinical observations, *J. Fr. Ophtalmol.* 28 (2005) 713–723.
- [26] F. Ramus, Developmental dyslexia: specific phonological deficit or general sensorimotor dysfunction? *Curr. Opin. Neurobiol.* 13 (2003) 212–218.
- [27] F. Ramus, E. Pidgeon, U. Frith, The relationship between motor control and phonology in dyslexic children, *J. Child Psychol. Psychiatry* 44 (2003) 712–722.
- [28] F. Ramus, S. Rosen, S.C. Dakin, B.L. Day, J.M. Castellote, S. White, U. Frith, Theories of developmental dyslexia: insights from a multiple case study of dyslexic adults, *Brain* 126 (2003) 841–865.
- [29] O. Sasaki, P.M. Gagey, A.M. Ouaknine, J. Martinerie, M. Le Van Quyen, M. Toupet, A. L'Heritier, Nonlinear analysis of orthostatic posture in patients with vertigo or balance disorders, *Neurosci. Res.* 41 (2001) 185–192.
- [30] J. Stein, The magnocellular theory of developmental dyslexia, *Dyslexia* 7 (2001) 12–36.
- [31] C.J. Stoodley, J.B. Talcott, E.L. Carter, C. Witton, J.F. Stein, Selective deficits of vibrotactile sensitivity in dyslexic readers, *Neurosci. Lett.* 295 (2000) 13–16.
- [32] H. Wimmer, H. Mayringer, T. Raberger, Reading and dual-task balancing: evidence against the automatization deficit explanation of developmental dyslexia, *J. Learn Disabil.* 32 (1999) 473–478.
- [33] B.A. Wright, R.W. Bowen, S.G. Zecker, Nonlinguistic perceptual deficits associated with reading and language disorders, *Curr. Opin. Neurobiol.* 10 (2000) 482–486.