Motor-Perceptual Function in Children with Developmental Reading Disorders: Neuropsychophysiological Analysis

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The aim of this paper is to compare the motor performances and the movement-related macropotentials of 8 male children aged 10 years with developmental reading disorders with those of 9 nondisabled male children of the same age. The task employed consisted of initiating the sweep of an oscilloscope with a self-paced movement and terminating it within 50 ± 10 msec. The children with reading disorders were slower, less accurate, and achieved a smaller number of target performances. Their brain macropotentials associated with motor programming, processing of sensory information, and evaluation of the results were altered in amplitude and latency. In the children with reading disorders, the systems involved in planning strategies and processing of sensory information are inadequate and those involved in the correction of errors are less efficient. Therefore, reading disorders could express defective integration and dysfunction of numerous processes occurring at different levels and times.

A human performance may be considered as a response to a specific sensory stimulus or the execution of a project organized by the brain in the absence of external stimuli. The latter assumes that an individual carries out schemes organized in advance, which may be updated through environmental feedback that may be external (exteroceptive: visual or auditory information), internal (proprioceptive: kinesthetic information), or through knowledge of results (Bernstein, 1967; Bruner, 1970; Schmidt, 1975).

In recent years, it has been shown that certain types of psychomotor activity are correlated with a consistent pattern of brain electrical activity. In particular, when a subject is engaged in a skilled motor perceptual task in order to achieve a preset goal and receives real time information about the quality of his or her performance, a characteristic sequence of brain macropotentials can be recorded from the scalp both in adults (Papakostopoulos, 1978a) and in children (Chiarenza, Papakostopoulos, Giordana, & Guareschi-Cazzullo, 1983). Such a task requires advanced programming according to suitable motor strategies, their updating through sensory kinesthetic and visual feedback, and evaluation of results in order to improve motor performance.

The brain electrical activity accompanying the performance of this task is defined as movement-related brain macropotentials (MRBMs). Observation of the myographic and brain electrical activity allows us to distinguish four periods: a premotor period, a motor-sensory period, a motor completion period, and a postmotor period (Papakostopoulos, 1978b). Figure 1 shows a detailed diagram of the sequence of the electrical brain events accompanying the execution of this task.

The premotor period is characterized by the basic tonic muscular activity and the presence on the scalp of a phasic negative potential lasting 800 to 1200 msec: the Bereitschafts-potential (BP) (Kornhuber & Deecke, 1965), or readiness potential (Vaughan, Costa, & Ritter, 1968), that is absent during passive movements. Its amplitude increases progressively with age, being absent in children under 6 years of age and reaching adult values at adolescence (Chiarenza, 1986a), and is proportional to the complexity of the task both in adults (Papakostopoulos, 1978b) and in children (Chiarenza, Papakostopoulos, Guareschi-Cazzullo, & Giordana, 1980). It is mainly recorded in the frontal and central regions. The BP is believed to reflect the processes of organization and selection of the strategy needed to carry out the task and has been proposed as an electrophysiological index of cerebral efficiency during the premotor period.

The sensory-motor period begins at the onset of phasic electromyographic activity and lasts about 200 msec. It is during this period that behavior becomes manifest. It coincides with the appearance on the scalp of the motor cortex potential (MCP) and N100. MCP is a negative potential that follows the BP. It is absent during passive movements, present in simple voluntary motor actions, and increases in amplitude during ballistic and sustained motor actions (Grunewald, Grunewald-Zuberbier, Homberg, & Netz, 1979; Papakostopoulos, 1978a). Scalp and cortical recordings have shown that MCP is mainly present in the precentral and central regions and is absent from the parietal regions (Papakostopoulos, 1980; Papakostopoulos & Crow, 1984). The MCP is proposed as an index of response-generated reafferent activity from the muscle, skin, and tendon receptors (Papakostopoulos, Cooper, & Crow, 1975). It is present in both children and adults, its amplitude decreasing with senescence (Papakostopoulos & Banerji, 1980). N100 is a negative potential with a latency of 100 msec that follows the MCP and represents the response evoked by the appearance of the oscilloscope trace and is normally inhibited in the frontal and postcentral areas during movement.

The motor completion period is characterized by the ending of the electromyographic phasic activity and the presence of a positive potential defined as P200 that follows N100 with a latency of about 200 msec from the beginning of the light stimulus (Vaughan et al., 1968). This potential is present during passive and active movements, both simple and complex, and is believed to be one of the components of the reafferent somatosensory potentials on the basis of developmental course (Chiarenza et al., 1983).

The postmotor period is marked by the electromyographic tonic activity similar to the premotor period and by the appearance on the scalp of a positive potential with a latency of about 450 msec, called Skilled Performance Positivity (SPP) (Papakostopoulos, 1978a, 1980), and by a slow negative potential

defined as Post-Action Negativity (PAN), with a latency of about 600 msec (Chiarenza et al., 1983). The SPP has a higher amplitude in the parietal regions and appears toward the ninth year in the fronto-central regions (Chiarenza, 1986b). Scalp and cortical recordings have shown that SPP is present only when the subject can evaluate the result of his or her performance (Papakostopoulos, 1980; Papakostopoulos, Stamler, & Newton, 1986). This potential is independent of the motor action and the presence of any exteroceptive stimulation; it coincides with the subject's awareness of success or failure in the performance (Chiarenza, 1986b; Papakostopoulos, 1980).

Post-Action Negativity has a specific spatial distribution, mainly recorded in the fronto-central regions, and decreases in amplitude with age, disappearing around the 10th year (Chiarenza et al., 1983). Like the SPP, PAN is independent of the motor act and seems to be related to analysis and evaluation strategies different from those generating SPP (Chiarenza et al., 1984). The presence of these positive and negative potentials following the performance of a skilled action has been confirmed by other authors, each time a voluntary goal-directed motor task is employed (Elbert, Lutzenberger, Rockstroch, & Birbaumer, 1986; Foit, Grozinger, & Kornhuber, 1982; Grunewald et al., 1979; Knapp, Schmid, Ganglberger, & Haider, 1980; Netz, Homberg, Grunewald-Zuberbier, & Grunewald, 1984; Taylor, 1978; Weinberg, 1980).

It is clear from these experiments that a self-paced, goal-directed motor performance consists of various motor, sensory, and cognitive subsets located in different sites of the brain, that they probably operate in parallel, and that they may vary independently of one another. As Bernstein (1967) maintained, these subsets should be considered as "integral formations"; separation of them is only apparent.

Psychophysiological studies of developmental reading disorders have mainly used externally paced experiments as stimulus-response paradigms, thus favoring the sequential aspects of information processing. However, this experimental model forces a separation between the various sensory-cognitive and motor processes involved in the execution of a performance. It has been observed that, according to the type of paradigm employed, some of the Event-Related Potentials (ERPs) N1, P2, P300, Contingent Negative Variations (CNV), are altered in latency and amplitude in children with developmental dyslexia (see Rosenthal, Boder, & Callaway, 1982, for an extensive review of the topic). It has been suggested that the various ERP components may reflect different stages of information processing; this has justified the identification of an altered specific cognitive process as the cause of reading and writing difficulties.

Conversely, from a psychological point

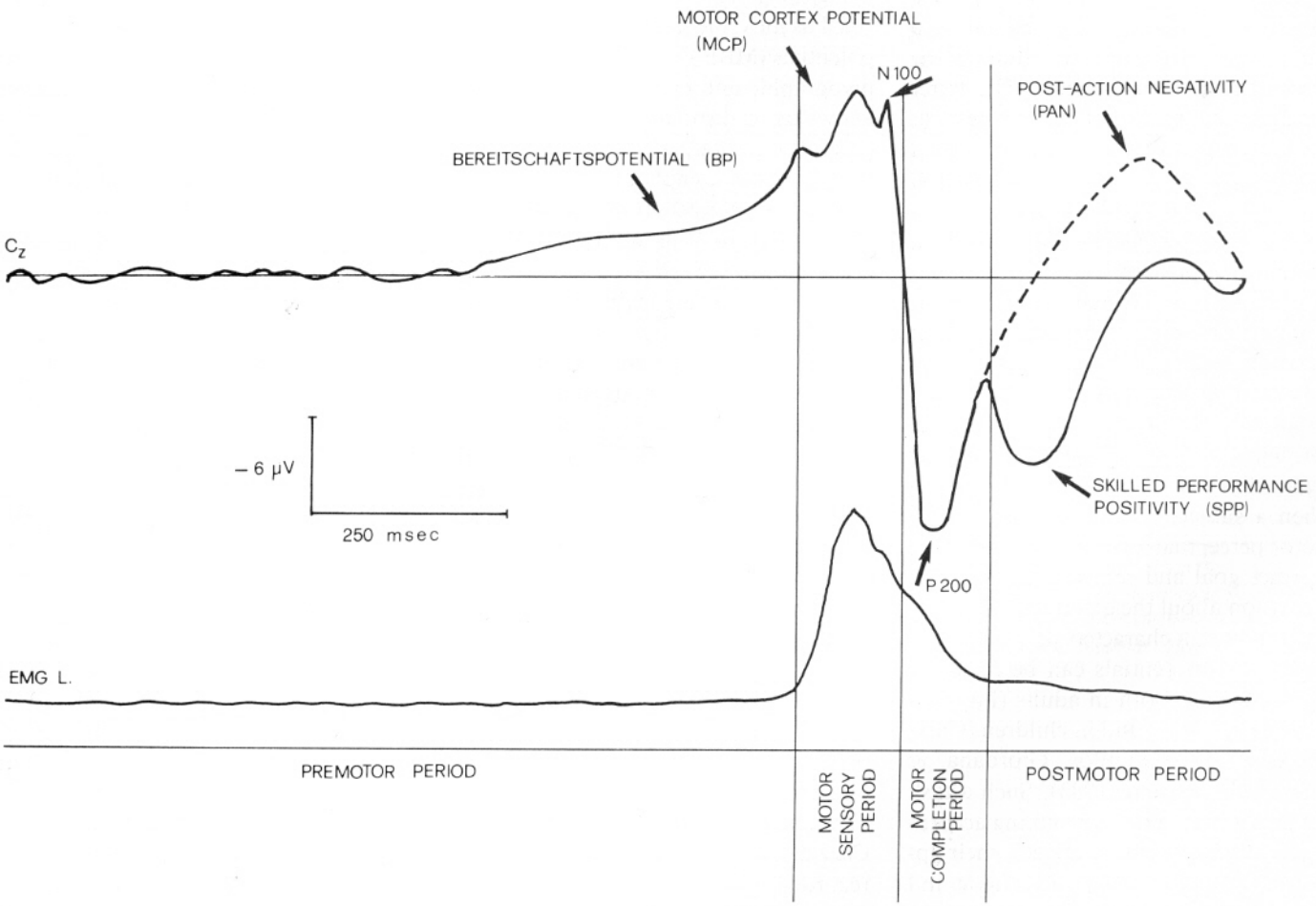

Figure 1. Schematic diagram of movement-related brain macropotential related to the skilled motor perceptual task.

of view, the hypothesis that developmental dyslexia is not attributable to a single disturbed process and does not form a unitary syndrome is receiving increasingly greater support (see Ellis, 1985, for an updated review of the topic). The interest in and use of self-paced and goal-directed paradigms for this type of disturbance are not widespread in spite of a few neurophysiological studies of EEG and evoked potentials suggesting that dyslexia is the result of various brain area dysfunctions (Duffy, Denckla, Bartels, & Sandini, 1980), and in spite of a few attempts to find possible electrophysiological correlates in subgroups of developmental dyslexia according to Boder's (1973) classification (Rosenthal et al., 1982).

It is well known that reading and writing involve a complex and skilled process consisting of a set of modular subroutines serially and hierarchically organized, and that children with developmental reading disorders lack control of perceptual and motor behaviors (Belmont, 1980). Therefore, a method that incorporates the study of motor performance, electromyographic activity, and MRBMs during the execution of a skilled motor-perceptual task appears to be particularly suitable for supplying useful information on those systems and subsystems that regulate and organize reading and writing functions.

The aim of the present paper is to compare the performances and the MRBMs of a group of children with developmental reading disorders with those of nondisabled children.

METHOD

Subjects

Forty male children coming from the same area of Milan have been tested; 9, aged 10 years, 4 months ($SD = \pm 3$ months), had been preclassified by the teachers as nondisabled, and 31, aged 10 years, 3 months ($SD = \pm 3$ months), had been sent for diagnostic testing concerning difficulties in reading. In order to ascertain whether the children were nondisabled (N) or met the criteria of the DSM III-R definition of developmental reading disorders (RD) (American Psychiatric Association, 1987), an extensive clinical and psychological test battery was administered to all children. The criteria were fulfilled by obtaining from the school the results of the medical school behavioral standard screening test for visual and auditory sensory acuity. In addition, the following were administered to all children: a full neurological examination (Towen, 1979); the Harris test for lateral dominance (Harris, 1968); the Wechsler Intelligence Scale for Children–Revised (Wechsler, 1976); the Culture Fair test, Scale 2, Form A (Cattell, 1951); a reading test consisting of the following items: meaningful words recognition, meaningless words recognition, and a spelling test (Italian adaptation of the Metropolitan Achievement Test) (Faglioni, Gatti, Paganoni, & Robutti, 1969, 1970). A detailed developmental and medical history was obtained from the parents together with information about the social and economic status of the family.

In order to be included in the group with developmental reading disorders, each child had to have an IQ of above 85 on the Culture Fair test, Form 2A, and on the WISC-R performance tests. The evaluation of the child's reading abilities was based on the results of the Culture Fair test and the reading and spelling tests and comparing these results with the chronological age of the child at the time of the testing. The value obtained from the interaction of these three variables had to exceed the tolerance limits fixed at 95% at least in one of the items of the reading test and in the spelling test. In addition, the children had to have had adequate schooling and shown difficulties in reading and writing from the early school years. The parents were literate and belonged to a middle socioeconomic class. Furthermore, in order to obtain a better clinical and psychological evaluation, the following psychological tests were administered: Lincoln Oseretzki Motor Development scale (Zucchi, Giugagnino, & Stella, 1959), Bender Visual Motor Gestalt test (Koppitz, 1964), Stamback test (Stamback, 1965), and Draw-a-Man test (Gesell & Amatruda, 1974).

The 9 children prelabeled as nondisabled were confirmed by the clinical and psychological evaluations. Out of the 31 children preclassified as children with developmental reading disorders, 23 children were excluded because the reading difficulties were dependent on other causes, such as cerebral palsy ($n = 3$), epilepsy ($n = 4$), mental retardation ($n = 12$), or evident behavioral or emotional problems ($n = 4$).

Procedure

The Skilled Motor-Perceptual Task (SMPT). The subject sat in an armchair in front of an oscilloscope at a distance of 70 cm in a lighted and electrically shielded room. The subject held a joystick-type push button in each hand. The travel of the button was 5 mm. The task consisted of starting a sweep of the oscilloscope trace with the left thumb and of stopping it in a predetermined area of the oscilloscope by pushing the other button with the right thumb; the sweep velocity was 1 mm/msec; the target area corresponded to a time interval of between 40 and 60 msec.

After a verbal explanation of the task, to ensure that all children understood the verbal instructions, the experimenter carried out a few trials and the children had to indicate the performance results; subsequently, so that all children may start from the same training level, they were allowed a short practice period. The recording procedure was initiated only after all children were able to stop the oscilloscope sweep at least twice in the 40- to 60-msec interval. This practice was also necessary to enable the children to become familiar with controlling eye movements or blinking during the execution of the task and to keep an interval of 7 to 20 seconds between any two attempts. The subjects were also asked to remain relaxed during the task and to avoid muscular preparatory movements before pressing.

EEG Recording. Silver chloride electrodes were fixed to the scalp with collodion in the prefrontal (Fpz), frontal (Fz), central (Cz), right precentral (RPC), left precentral (LPC), and parietal (Pz) regions. Each electrode was referred bilaterally to the mastoids. The surface electromyogram was recorded from the flexor muscles on the right and left forearms. The impedance of the electrodes was less than 3 KOhm. The time constant and high frequency were 4.5 sec and 700

Hz for the EEG and 0.03 sec and 700 Hz for the EMG, respectively.

The EEGs and EMGs were stored on FM magnetic tape for off-line analysis. The analysis started with sampling for each channel, a square wave of $\pm 25\mu V$ for calibration purposes. During the off-line analysis, on reception of the trigger pulse obtained from an electric pulse generated at the press of the left-hand button, 3.2 seconds for each channel were sampled at a rate of 500 Hz. Of these, 2.2 seconds preceded the trigger pulse and 1 second immediately followed it. An average of the first second was then taken to establish the baseline from which the amplitudes of the various potentials were measured. All values were normalized to the calibration detected and stored on disk.

Data Analyses

Performances. The time interval between the two presses was measured and defined as "performance time." The distance from the target area was also measured and defined as the "performance shift." The number of performances reaching the target was measured and defined "target performance." The number of performances shorter than 40 msec and longer than 60 msec was also counted and defined "wrong performance."

Movement-Related Brain Macropotentials. For every subject, four blocks of 25 sequential trials free of muscular artifacts, blinking, or eye movements were averaged and measured. In addition, to ensure that any differences in MRBMs between the two groups were not due to a jitter effect related to performance variability, or to an increased number of wrong performances in the group of children with developmental reading disorders, each performance during SMPT was allocated according to performance time in one of nine time intervals (interval 1: 0 to 20 msec; interval 2: 21 to 39 msec; interval 3: 40 to 60 msec; interval 4: 61 to 80 msec; interval 5: 81 to 100 msec; interval 6: 101 to 125 msec; interval 7: 126 to 150 msec; interval 8: 151 to 200 msec; interval 9: >200 msec), and the MRBMs were averaged consequently.

EMG and MRBM Measurements. The mean amplitude of the EMG prior to movement, the peak amplitude during movement, and the EMG rise time of the rectified surface left and right electromyograms were calculated after locating the EMG onset and the EMG peak. The MRBMs were measured as follows: The area of BP was measured from the BP onset to the point corresponding to that of the EMG onset. The mean amplitudes of BP and motor cortex potential were computed for 200-msec periods, the BP immediately preceding the left-EMG onset, and the MCP immediately following it. The MCP value was measured as the difference between the BP and MCP values measured from the baseline. The latency of the MCP peak was measured with respect to the EMG onset. The amplitudes of N100 and P200 were measured from the baseline, and their latencies were calculated from the left-hand trigger. The mean amplitudes of Skilled Performance Positivity and Post-Action Negativity were taken as average values from the baseline over 200 msec centered around the main positive (SPP) and also negative (PAN) peaks in the latency band between 350 and 850 msec, respectively. SPP and PAN latencies were measured from the trigger pulse. The two groups were compared using confidence limits at 95% ($p<.05$) and 99% ($p<.01$).

RESULTS

The neurological examination of the nondisabled children and children with developmental reading disorders showed no classic signs of major or minor neurological damage. However, there were some consistent neurological signs in the group of children with reading disorders, even though they could not be grouped together in a well-defined clinical picture. The most frequent signs were dysdiadochokinesis, motor clumsiness in fine manipulative activity, and synkinetic movements in the contro-lateral hand during the diadochokinesis test and finger opposition test. The quality of gross and fine movement was not optimal in terms of speed, adequacy, and fluidity. Comparison of the results of the psychological tests showed significant differences only for the WISC-R verbal tests (RD = 100.25; N = 134.44; $p<.05$) and for the Oseretzki test, in which the subjects with developmental reading disorders showed a lower developmental age than the nondisabled subjects (RD = 134.7; N = 165.6; $p<.05$).

All the children understood the verbal explanations of the task and, after a short training period, completed their assigned motor-perceptual task, although with significant differences in terms of motor performance and MRBMs. There was no statistical difference in the rate of rejected trials between children with developmental reading disorder (0.572) and the control group (0.521). There was no statistical difference when the performance times of rejected and accepted trials of each subject were compared.

The mean performance time was 99.44 msec for the children with reading disorders and 62.93 msec for the control group. The percentage of target performance was 14.52% and 26%, respectively. Furthermore, the subjects with reading disorders had a percentage of performances in the last four time-intervals greater than the nondisabled children (see Figure 2). They were also less accurate, with a performance shift of 50 msec, compared with 19.2 msec for the nondisabled subjects. All these results were statistically significant ($p<.01$). Observing the performance during each of the four sequential blocks of tests, it was apparent that the exercise produced a marked improvement in the children with reading disorders. In fact, their performance time dropped from 112.03 msec in the first block to 62.61 msec in the fourth, while it dropped from 67.5 msec to 60.47 msec for the nondisabled children (see Table 1). The accuracy of the children with reading disorders showed the same improvement. The performance shift dropped from 58.0 msec in the first block to 19.2 msec in the fourth, while it dropped from 22.3 msec to 17.0 msec for the nondisabled children (see Table 1). A comparison of the two groups of children shows significant differences ($p<.01$) in the first three blocks but not in the fourth, for both performance time and performance shift.

The electromyographic activity of the two groups was not different either in amplitude before and during the movement, or in the rise time of either left or right forearm muscles. Consistent differences were found in MRBMs during

all four motor periods (see Figure 3 and Table 2). In the premotor period, the BP area of the children with developmental reading disorders was significantly reduced in the central, precentral, and parietal regions (Cz,Pz,LPC: $p<.05$; RPC: $p<.01$); the BP amplitude was reduced in Cz ($p<.01$) and Pz ($p<.05$). The BP onset was significantly delayed in the right and left precentral regions (RPC: $p<.01$; LPC: $p<.05$).

In the sensory-motor period the amplitude and the latency of MCP with respect to the EMG onset was not different in the two groups of children. The latency of N100 was significantly greater in the children with dyslexia in the prefrontal and parietal regions (FPz,Pz: $p<.05$) and increased further in the frontal central and precentral regions (Fz,Cz,RPC, LPC: $p<.01$).

In the motor completion period, there were no significant differences between the two groups in the latencies of P200 except in Fpz ($p<.01$). The amplitude of P200 was significantly reduced in the children with reading disorders in all the brain areas (Fpz,Fz,Cz,Pz,RPC,LPC: $p<.01$).

In the postmotor period, SPP was present in all brain areas of the nondisabled subjects; in children with reading disorders, SPP was absent or reduced in the frontal, central, and precentral regions (Fz,RPC,LPC: $p<.05$; Cz: $p<.01$); in the parietal region it was present but with significantly reduced amplitude ($p<.01$). In children with reading disorders, the latency of SPP was greater in all the brain areas recorded than in the nondisabled children. This difference did not reach statistical significant except at Cz ($p<.05$). PAN was more frequent and had a higher amplitude in the children with reading disorders than in the nondisabled children; it was mainly recorded in the frontal, central, and precentral areas.

When the MRBMs related to the target performance of the nondisabled children were compared with those of children with reading disorders, significant differences were observed only in the postmotor period (see Figure 4). Only during the target performance was SPP present in all the brain areas of children with reading disorders, although with a reduced amplitude. This reduction was

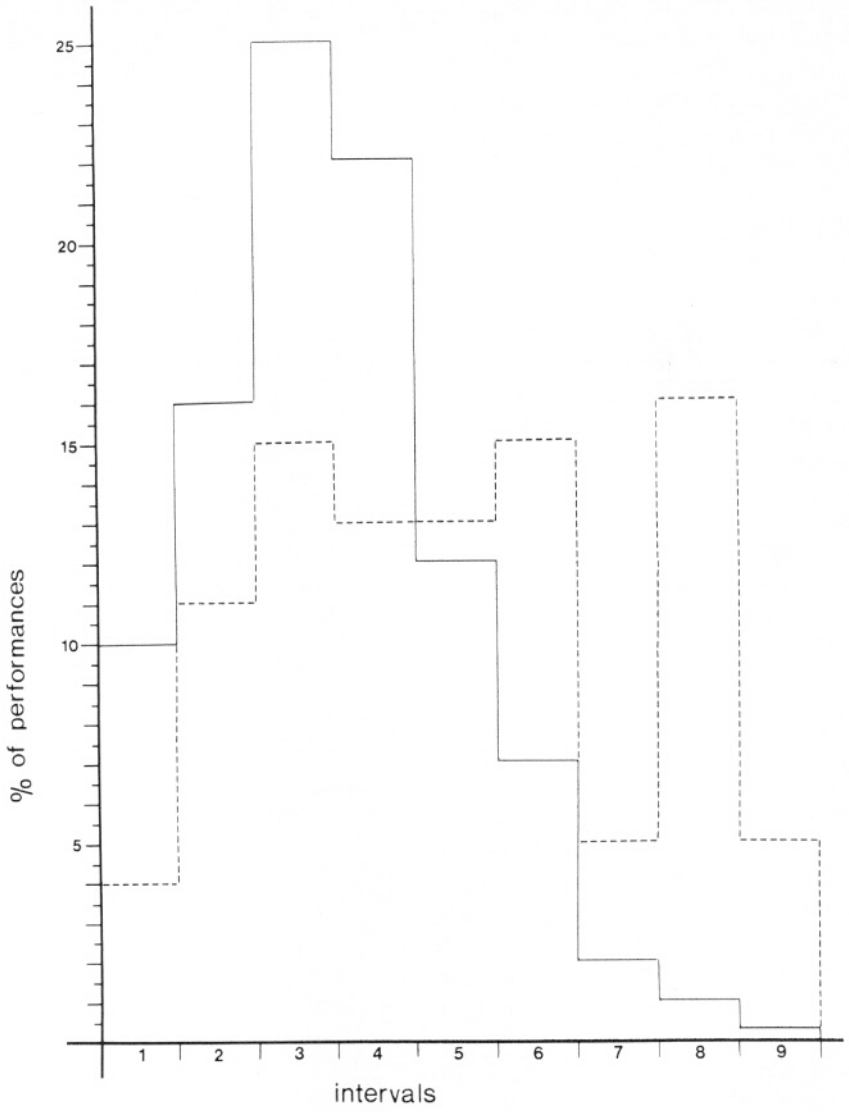

Figure 2. Percentage of performance time of nondisabled children (unbroken line) and children with developmental reading disorders (broken line) in the nine time intervals. See text for definition of intervals.

TABLE 1
Means and Standard Deviations (*SD*) of Performance of Nondisabled Children (N) and Children with Developmental Reading Disorders (RD)

		Performance time		% target performance		Performance shift	
		N	RD	*N*	RD	*N*	RD
First block	$\bar{X}$	67.5	112.0	22.2	17.0	22.3	58.0
	SD	37.8	59.6	41.6	37.7	26.5	52.6
Second block	$\bar{X}$	64.3	105.4	23.1	8.0	21.2	56.0
	SD	53.3	69.5	42.2	27.2	45.6	59.4
Third block	$\bar{X}$	59.4	107.3	31.5	15.9	16.4	58.2
	SD	45.3	97.3	46.5	36.8	38.2	89.9
Fourth block	$\bar{X}$	60.4	62.6	27.1	25.3	17.0	19.2
	SD	33.2	34.0	44.5	43.8	22.2	21.6
Total	$\bar{X}$	62.9	99.4	26.0	15.8	19.2	50.0
	SD	43.1	72.5	43.8	36.6	34.4	64.0

significant only in Pz ($p < .05$). SPP latency was significantly longer in the group of children with developmental reading disorders in the central and right and left precentral regions (Cz: $p < .05$; RPC: $p < .05$; LPC: $p < .01$) (see Table 3).

DISCUSSION

The above results show that there are psychological and neurophysiological differences between nondisabled children and children with developmental reading disorders. The most consistent differences are at the level of quality of movement: speed, fluidity, and adequacy as shown by the neurological tests and the Oseretzki psychomotor test. These children showed lower than actual age in both motor development and in some tests of the neurological examination. These signs could be interpreted as insufficient coordination and temporal control in motor sequencing.

It has been suggested that part of the preparation for movement entails the activation of a central clock that controls the time sequencing of the motor action through afferent and efferent systems (Hirsch & Sherrick, 1961; Rosenbaum & Patashnik, 1980). The improvement of the performance of this clock depends on a greater synaptic efficiency of the central nervous system, which depends on age (Craik, 1947) and on the presence of an internal and external feedback on the accuracy of the performance.

Our subjects with reading disorders appeared to be slower, less accurate, and to have achieved a smaller number of target performances in carrying out this motor-perceptual task. Furthermore, the nondisabled children achieved a better motor performance and reached their peak in the second block, which they thereafter maintained. However, although they began with a much worse motor performance, the children with reading disorders improved steadily over the whole period of the experiment, reaching the levels of the nondisabled children only in the last 25 trials.

In addition, compared with the nondisabled children, those with reading disorders had greater difficulty in controlling irrelevant and inappropriate movements, such as blinking immediately before or after the performance of the task, or gross movements of the body or lower limbs. These differences cannot be attributed to misunderstanding the verbal instructions, or a different training level or lack of commitment in performing the task, as all the children carried out the task correctly, respecting the bimanual motor sequence, and had equal opportunity to practice. If the results of the neurological tests, the Oseretzki test, and performance on the motor-perceptual task are combined, it can be said that the setting of the central clock proceeds with difficulty and its control, based on the processing of internal and external feedback, does not occur correctly, as we shall see later.

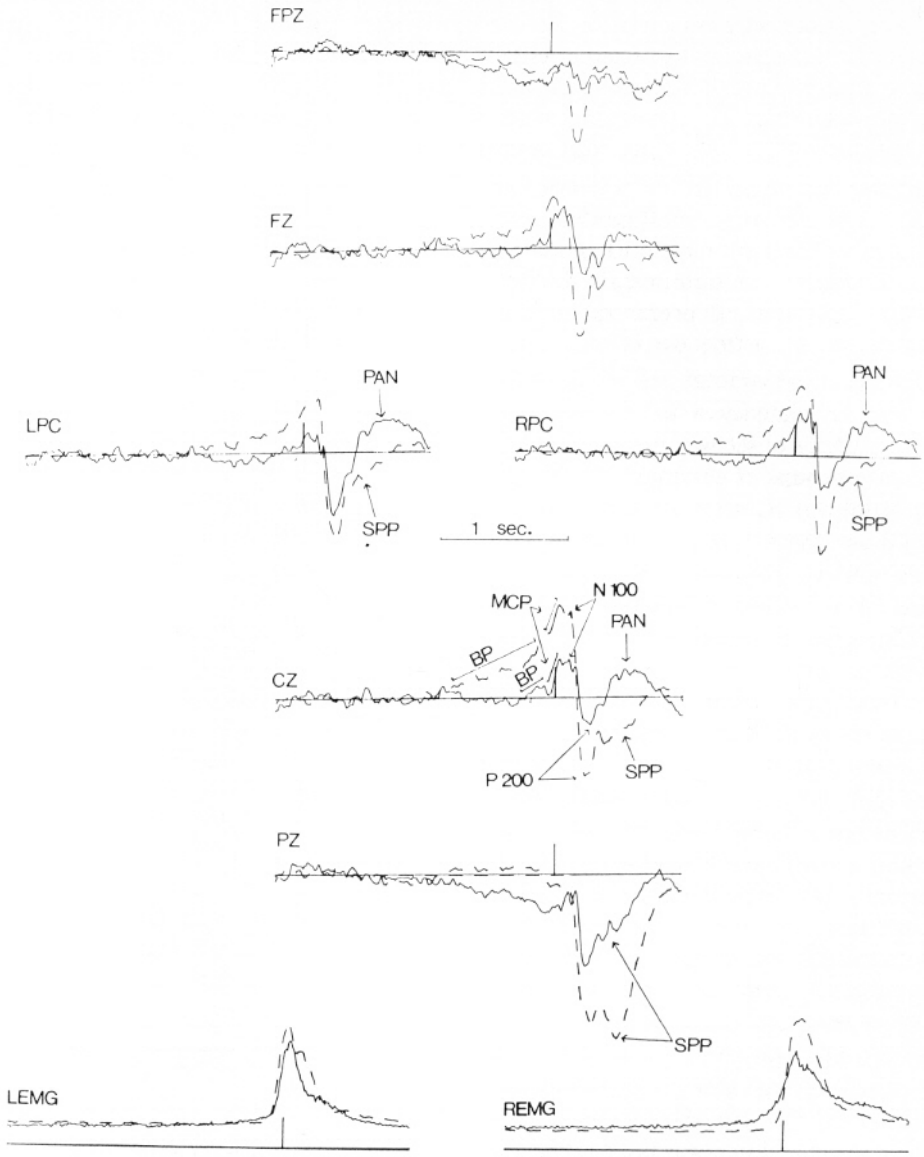

Figure 3. Grand average of rectified EMGs and MRBMs in nondisabled children (broken line) and children with developmental reading disorders (unbroken line). In this and the following figures, the vertical bar, in each trace, indicates the instance of the computer trigger and a calibration signal of 5 microvolt. Negativity is upwards. For the abbreviations of the potentials see the text.

It is known that children with reading disorders have difficulty in executing simple motor tasks (Bruininks & Bruininks, 1977; Lewis, Bell, & Anderson, 1970; Pyfer & Carlson, 1972) and tests of bimanual coordination (Klicpera, Wolff, & Drake, 1981). It has been suggested that they may suffer from a deficit in the temporal organization of motor inhibitory commands when they are required to switch rapidly back and forth across different articulation patterns or timing motor commands, precisely to suppress unintentional repetitions (Wolff, Cohen, & Drake, 1984). Insufficient motor performance, in particular, has been observed in subjects with commissurotomy of the corpus callosum, and it has been suggested that integrity of the

callosum commisura is essential to the performance of bimanual tasks (Kreuter, Kinsbourne, & Trevarthen, 1972; Preilowski, 1972; Zaidel & Sperry, 1977). It is noteworthy that the myelination of the corpus callosum is completed at approximately the age of 10 (Yakovlev & Lecours, 1967).

In parallel with the motor performances, the MRBMs showed significant differences between nondisabled children and children with developmental reading disorders. The BP is a feature of the premotor period, when the ideokinetic elements of the movement are being organized. In the children with reading disorders, BP was reduced in the parietal, central, and precentral areas, and furthermore, began only about 100 msec before the movement. It has recently been suggested that the BP could consist of two components: The first begins 1.2 seconds before the movement, lasts for about 450 to 600 msec, and is linked to processes related to representation of the action, while the second is characterized by a steep negative ramp lasting 300 to 500 msec (Shibasaki, Barrett, Halliday, & Halliday, 1980); it is the most effectual part of the process and therefore the most automatic (Chiarenza, 1989). This first component was absent in the children with reading disorders, while the second one was considerably reduced in amplitude.

The sensory-motor period is characterized by the motor cortex potential and N100. MCP reflects processing of the kinesthetic reafferent information in the precentral and frontal areas related to the movement carried out. Its absence from postcentral and parietal regions has been interpreted as a possible phenomenon of somatosensory activity suppression on these areas during movement (Cohen & Starr, 1987; Papakostopoulos et al., 1975; Rushton, Rothwell, & Cragg, 1981). The latency and amplitude of the MCP were normal in the two groups of children. N100, one of the components of the visual response evoked by the appearance of the light trace on the oscilloscope, was delayed in children with reading disorders, in the prefrontal and parietal regions, and was even more delayed in the frontal central and precentral regions. A reduction in the amplitude of the potentials evoked on prefrontal and postcentral regions has been observed when stimuli are generated by the subject with a brisk movement instead of being administered by the experimenter. This phenomenon of centrifugal inhibition upon sensory information is present with all types of sensory stimuli, somatosensory (Cohen & Starr, 1987; Grunewald, Grunewald-Zuberbier, Schumaker, Mewald, & Noth, 1984; Hazemann, Audin, & Lille, 1975; Papakostopoulos & Crow, 1984; Rushton et al., 1981), visual (Papakostopoulos, 1980; Volkman, 1962), and auditory (Hazemann et al., 1975; Tapia, Cohen, & Starr, 1987), and should be considered as one of the brain phenomena that take place during a self-paced movement. In our subjects, no statistical comparison between the two groups was possible as the measurement of N100 amplitude is dependent on BP amplitude. However, the increased latency of this peak might support the hypothesis that this phenomenon takes place in children with developmental reading disorders with different delays in the various brain areas.

The P200 is a positive potential largely distributed on the scalp, present during both skilled and unskilled tasks, which coincides in time with the visual evoked potential of the oscilloscope trace. However, the fact that P200 changes with age (Chiarenza et al., 1983), exists even in the absence of external stimuli (Vaughan et al., 1968), and increases in amplitude with force (Wilke & Lansing, 1973), supports the hypothesis that P200 is possibly

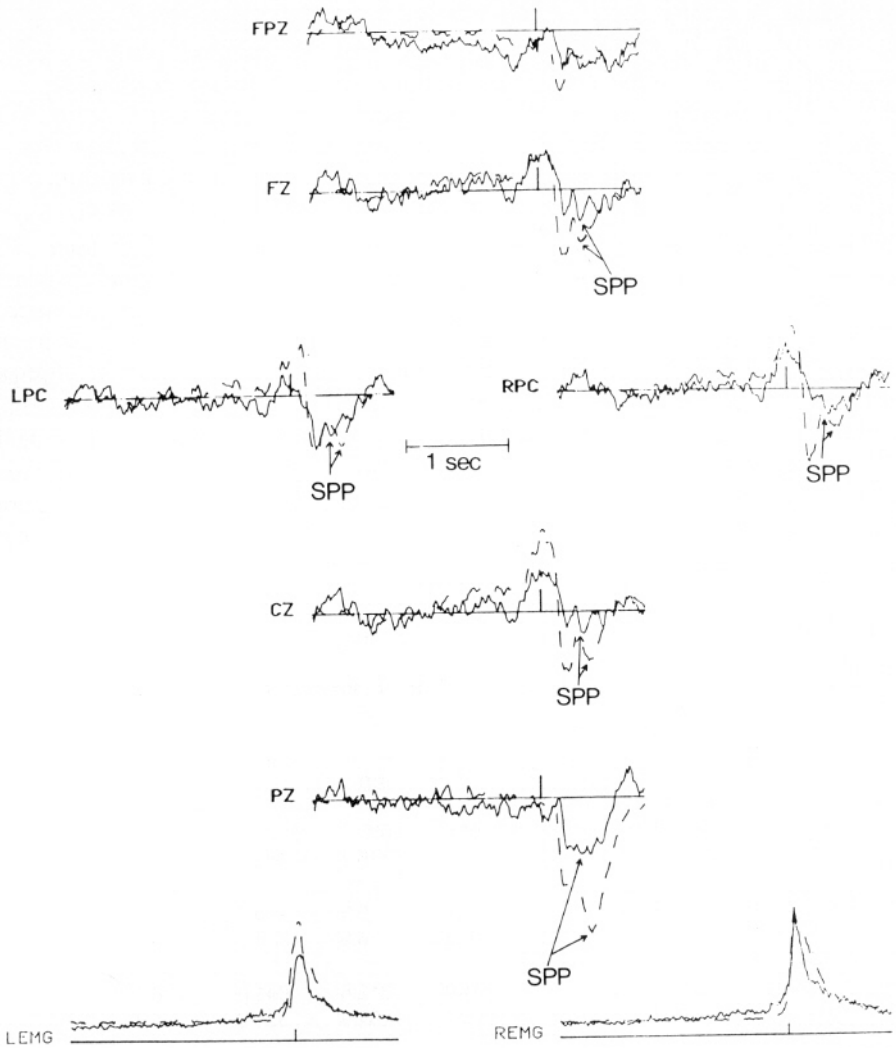

Figure 4. Grand average of rectified EMGs and MRBMs related to target performance of nondisabled children (broken line) and children with developmental reading disorders (unbroken line). Note the appearance of the SPP in the frontal, central, and precentral areas of the children with developmental reading disorders, though the amplitude is lower compared to that of the nondisabled children when they hit the target.

a reafferent somatosensory potential related to movement. The reduction in amplitude of P200 seen in the various brain areas of the children with reading disorders, also during the same performance interval (target performance) (see Figure 4), but not in the nondisabled children, could indicate a defect in the integration of reafferent kinesthetic information.

The SPP was absent in the frontocentral and precentral regions and present in the parietal regions, with reduced amplitude in children with reading disorders. There was an increased SPP latency compared with the nondisabled children. The averages of the MRBMs related to the target performance (see Figure 4) show that there is SPP in the children with reading disorders, in the fronto-central and precentral areas, even though the amplitude is considerably reduced and the latency significantly increased compared with the nondisabled children. In our group of children with reading disorders, the increased SPP latency may suggest difficulty in awareness and evaluation of their own performance. Similar results have been obtained with the same SMPT in younger children with learning disabilities (Chiarenza, Papakostopoulos, Guareschi-Cazzullo, Giordana, & Giammari-Aldè, 1982) and with externally paced paradigms to elicit P300 (Holcomb, Ackerman, Roscoe, & Dykman, 1985).

The averages of the wrong performances (interval 2: 21–39 msec) show that PAN was present in the frontal, central, and precentral regions (see Figure 5). Long latency negative potentials with similar scalp distribution have been described in children during externally paced experiments and have been associated with the importance, interest, or surprise of the stimulus (Courchesne, Elmsian, & Yeung-Courchesne, 1987). It is highly likely that children with developmental reading disorders perceive their unsuccessful performance as an unexpected, although likely, event. The presence of PAN in the children with reading disorders during wrong performances could be related to a different strategy

TABLE 2
Means and Standard Deviations (SD) of MRBMs of Nondisabled Children (N) and Children with Developmental Reading Disorders (RD)

		FPZ		FZ		CZ		PZ		RPC		LPC	
		$\bar{X}$	*SD*	$\bar{X}$	*SD*	$\bar{X}$	*SD*	$\bar{X}$	*SD*	$\bar{X}$	*SD*	$\bar{X}$	*SD*
Ar.BP	N	941.3	1261.1	−1054.6	1661.6	−1771.8	1641.0	−568.9	1872.8	−1342.4	1333.5	−954.5	1345.3
	RD	1475.1	1416.7	93.0	776.1	−187.7*	1011.8	1174.9*	1065.8	−19.6**	813.5	−9.1*	687.3
A.BP	N	3.1	4.8	−4.4	6.9	−9.2	7.5	−0.6	7.6	−6.5	6.5	−5.0	6.5
	RD	4.5	5.2	−0.6	5.9	−1.5**	5.0	4.6*	4.4	−1.5	5.9	−0.7	4.2
O.BP	N	575.1	233.4	564.0	312.6	641.0	278.5	545.3	306.6	573.9	322.4	540.1	299.1
	RD	556.5	372.7	420.2	284.0	391.8	215.4	498.0	307.4	299.6**	150.6	297.8*	154.0
L.MCP	N	151.3	26.8	130.0	24.1	129.4	24.9	139.6	40.0	124.9	22.6	129.3	27.0
	RD	105.0	55.1	107.0	34.4	107.8	31.4	93.0	22.1	104.8	29.2	105.7	30.2
A.MCP	N	0.4	2.7	−1.6	4.6	−2.2	7.1	3.8	5.2	−0.8	6.7	−1.2	6.3
	RD	−1.1	2.7	−3.0	4.6	−1.5	4.2	1.1	3.4	−3.2	5.2	−0.6	6.4
L.N100	N	121.7	20.2	119.4	19.6	118.4	17.8	127.6	25.1	117.5	18.8	116.9	16.9
	RD	146.5*	19.9	151.8**	30.2	150.9**	21.8	152.8*	25.1	151.7**	19.4	153.3**	24.8
L.P200	N	210.4	23.5	223.0	18.8	226.5	26.7	232.5	27.3	219.2	27.0	227.0	28.3
	RD	244.7**	22.5	235.7	26.9	231.4	27.2	239.7	18.4	221.2	20.5	225.2	15.7
A.P200	N	12.6	4.6	22.1	6.9	26.0	7.6	22.0	10.5	25.3	7.2	23.3	6.4
	RD	4.8**	5.1	11.9**	5.2	9.4**	5.9	10.0**	6.6	11.6**	6.6	12.6**	5.4
L.SPP	N	537.0	99.2	539.4	99.8	501.7	65.3	495.2	45.7	516.7	81.9	510.7	79.8
	RD	568.4	59.8	569.3	59.4	572.4*	58.9	531.6	60.0	571.8	65.1	546.8	53.2
A.SPP	N	6.9	7.4	5.8	9.5	7.6	12.9	25.2	10.8	5.0	9.7	6.0	9.9
	RD	5.3	4.9	−1.0*	4.9	−2.9**	4.5	8.1**	7.9	−3.5*	5.3	−3.9*	6.2
L.PAN	N	—	—	769.3	116.2	802.2	131.8	—	—	824.2	49.0	753.7	163.1
	RD	—	—	748.0	172.5	700.2	92.8	—	—	669.0	131.0	672.0	107.7
A.PAN	N	—	—	8.3	6.1	−7.1	4.1	—	—	−11.4	3.9	−11.1	5.9
	RD	—	—	−5.6	0.3	−4.7	6.5	—	—	−14.7	—	−16.4	5.5

*$p < .05$; **$p < .01$; Ar = area; A = amplitude; O = onset; L = latency; BP = Bereitschaftspotential; MCP = motor cortex potentials; SPP = skilled performance positivity; PAN = postaction negativity; FPZ = middle prefrontal; FZ = middle frontal; CZ = vertex; PZ = middle parietal; RPC = right precentral; LPC = left precentral.

TABLE 3
Means and Standard Deviations (*SD*) of Skilled Performance Positivity (SPP) of Nondisabled Children (N) and Children with Reading Disorders (RD) During Target Performances

		FZ		CZ		PZ		RPC		LPC	
		$\bar{X}$	*SD*	$\bar{X}$	*SD*	$\bar{X}$	*SD*	$\bar{X}$	*SD*	$\bar{X}$	*SD*
SPP latency	N	473.7	57.2	454.0	49.1	479.3	36.5	456.5	44.8	567.0	40.0
	RD	563.0	14.4	567.0*	42.4	502.0	62.2	519.0*	70.0	551.0**	12.7
SPP amplitude	N	14.1	11.4	14.2	6.3	31.5	8.2	13.1	11.9	14.3	12.8
	RD	3.23	1.39	2.4	4.04	11.6*	7.5	4.2	2.2	4.24	2.2

*$p < .05$; **$p < .01$; FZ = middle frontal; CZ = vertex; PZ = middle parietal; RPC = right precentral; LPC = left precentral.

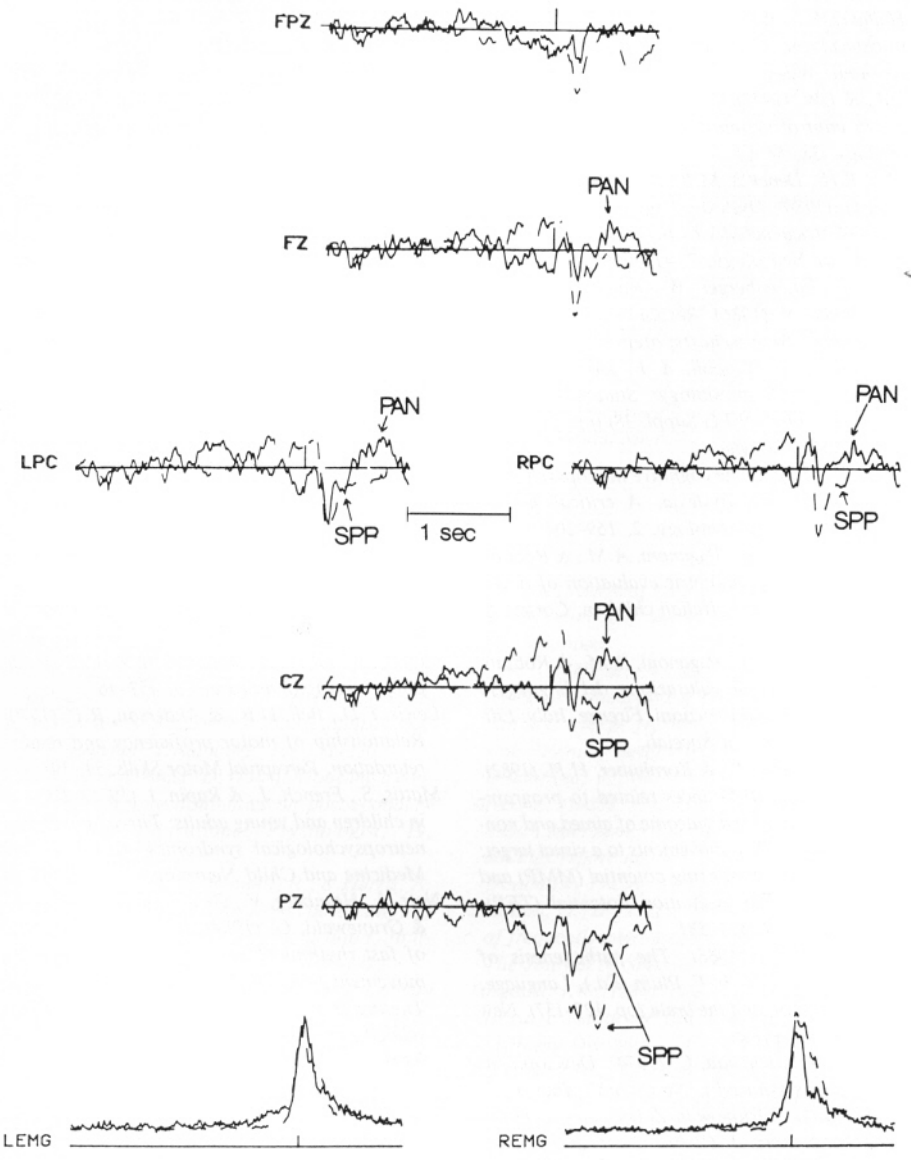

Figure 5. Grand average of rectified EMGs and MRBMs related to performance time in interval 2 (21 to 39 msec) of nondisabled children (broken line) and children with reading disorders (unbroken line). Note the presence of PAN in the frontal, central, and precentral regions in the children with reading disorder when they miss the target and the presence of SPP in the nondisabled children.

activated during the processing when the target was missed. These children seemed to give significance to the target performance only, without recognizing the failed performance, as the presence of SPP during wrong performance in the nondisabled children suggests.

The present data seem to indicate that during a motor-perceptual act, the systems involved in the planning and programming of effective strategies are inadequate, and that, furthermore, those involved in the verification and correction of errors are less efficient. These systems may be altered as such, may reflect deficiencies in those subsystems concerned with sensory information processing, or they may be potentially adequate but not fully developed. Therefore, it can be said that reading disorders result from the defective integration and dysfunction of numerous processes that occur at different levels and times. Our conclusions seem to be confirmed by the neuropathological observations of Galaburda's autopsy cases (Galaburda, 1988) and speculations by Hynd and Semrud-Clikeman (1989) proposing that important cortical-subcortical frontal-inhibitory systems may be implicated in reading disability. From this perspective, given the basic character of the processes involved in the execution of this motor-perceptual task, it would be interesting to test groups of children belonging to different subtypes of developmental reading disorders as classified by Mattis, French, and Rapin (1975) or Boder (1973) and then compare them with groups of children with other types of learning disorders.

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