

Neuropsychophysiological Approach to Specific Developmental Learning Disabilities

G. A. Chiarenza

Introduction

Learning to read and write is one of the aspects of scholastic success on which the expectations of the family, the teacher, and the child itself weigh most heavily. The child's ability to satisfy these expectations is frequently a crucial factor in the child's current and future adaptation to life.

Various hypotheses have been advanced to explain reading and writing learning difficulties based on function models and knowledge of the central nervous system. Thus, from time to time, the key to interpretation was thought to have been found in an alteration of a given cognitive process. Further, since the dysfunction manifests itself at school age, educational specialists have seen the problem in terms of their own area of competence and highlighted sometimes its psychological, sometimes its neurological or pedagogic aspects. This variety of interpretation has not helped a fluid or immediately fruitful exchange of knowledge because of the different jargons involved, so an organic and overall formulation of the problem has only been arrived at with difficulty.

The definitions of dyslexia proposed up to now have been either (a) predominantly clinical, concentrating on a profile of reading and writing errors and associated disturbances, such as difficulties in visual-spatial abilities, temporal analysis of rhythm, motor coordination, and mixed cerebral dominance, or (b) limited to describing the disturbance as the alteration of specific cognitive processes at certain stages of information processing. Furthermore, since the children examined were subjected to a variety of tests, many of the results are not comparable owing to the use of both different definitions and methodologies.

A further difficulty lies in the fact that reading and writing learning difficulties appear when the child first goes to school, without premonitory specific symptoms, without obvious neurological or personality disturbance, and when there has already been development of quite complex linguistic ability.

It is known that learning reading and writing requires the separate and integrated processing of auditory and visual information. A hypothesis developed in this respect based on neuropsychological research by Luria (1973)

within the word or sentence. These processes need not be mutually exclusive and can occur simultaneously. The inability to read could be due to incorrect use of the simultaneous or sequential strategy or a defective comprehension process (Bateman 1968; Ingram et al. 1970; Bannatyne 1966; Kinsbourne and Warrington 1966; Myklebust 1965). A similar approach was used by E. Boder (1973). Boder also maintains that reading and spelling are closely connected interdependent functions and that the diagnosis of dyslexia can be seen by looking at the reading and writing performance only as a whole and not just at individual errors. *Reading* requires visual perception and discrimination, visual memory sequencing, and directional orientational processes (Benton 1962; Birch 1962); it also requires the integration of different sensory modalities and the translation of visual symbols into meaningful auditory equivalents (Ingram 1963; Birch and Belmont 1964; Rabinovitch 1968). *Speech* requires the conversion of sounds into their visual symbol equivalents and depends on the auditory perception and discrimination processes, on auditory memory sequencing, and on recall (Wepman 1962; Bannatyne 1966; Bakker 1970). *Writing* requires fine motor and visual-motor coordination and tactile-kinesthetic memory (Bannatyne 1966; Johnson and Myklebust 1967). Perception emerges from this list of functions involved in the process of reading and writing as the basis of learning reading and writing. This function must be seen as a complex of numerous integrated higher-order functions.

A useful psychophysiological definition of perception is that which sees it as the extraction of information from the environment (Gibson 1969). This implies that perception in an "active" process of search, selection, and organization of stimuli from the nearby environment which are related to precise intentions and tasks. Furthermore, perception includes a variety of processes other than those mentioned above: expectation, anticipation, attention, motivation, formulation, and verification of hypotheses in relation to the requirements of the task. The processes involved in the search and selection necessarily include motor components and the sequential organization of perceptual-motor patterns in relation to the task required (Birch and Lefford 1963; Luria 1973).

There is no test or school exercise which checks a single function: perception, language, personality, etc. On the contrary, the behavior which we see is the product of a complex set of interacting systems, none of which acts alone. These sensory systems interact in their turn with other systems, e.g., motor, linguistic, motivational, mnemonic, and programming. These are all in relation to the specific requirements of the task set in a socially determined context.

Naturally, the development and efficiency of a system are not determined only by the interaction of the subject and the environment, but also by the influence of one system on the other hierarchically organized. If this point of view is accepted, there are no school tests which involve only one of the processes in such a way as to study them individually and separately.

When the child is unable to reproduce a figure accurately it is thought to be due to distorted visual perception: If the figure is drawn by the child with an angle different from the model, the child is said to be unaware of spatial position or to be

incapable of perceiving it accurately. If the relationship between two figures is not respected, it is said that the child is unable to perceive spatial relations between objects.

These interpretations are not able to explain which function, perceptive or motor-perceptive, is really being compromised. For example, in order to copy the drawing it is necessary that the visual process analysis and synthesis, which interact together, are coordinated by the kinesthetic functions described above. Copying is a motor activity guided by sight; it requires that kinesthetic information from the movement and posture of the trunk, head, arm, hand, etc. is continually and dynamically related to movement. Further, the child must organize these determined motor patterns and control the appropriate muscle tension related to the necessary succession of movements.

The inability to copy accurately could be based on defective function of the visual system, the kinesthetic system, or the motor system, or on the lack of integration between the visual-kinesthetic complex and the motor system. A further possibility is that these systems may be efficient while the systems for programming or verification of the performance may not be adequate.

This has led to the hypothesis that deficiencies in perceptive function are at the basis of reading and writing learning difficulties (Belmont 1980). This single-factor approach has led to a wide collection of plausible explanations of this disorder. Included are theories proposing: a deficiency in visual perception (Lyle and Goyen 1968, 1975; de Hirsch et al. 1966; Jansky and de Hirsch 1972; Silver and Hagen 1971; Rourke 1976; Satz et al. 1974), a dysfunction in cross-modal integration (Birch and Belmont 1964, 1965), difficulty in memory recall (Senf 1969; Senf and Feshback 1970; Senf and Freundl 1971), a difficulty in temporal order recall (Bakker 1967, 1972; Groenendaal and Bakker 1971), a disorder in cerebral dominance characterized by abnormalities in the degree of lateralization (Hynd et al. 1979), a delay in the maturation of lateralization and differentiation of motor, somatosensory, and linguistic processes (Satz and Sparrow 1970; Satz et al. 1971), and bilateral representation of spatial processing, normally thought of as a function of the right hemisphere, which interferes with the linguistic functions of the left hemisphere (Witelson 1976). The conclusions of most of these studies seem to indicate that dyslexia is not due to a deficit in or specific retardation of development but is the result of various interacting factors and that some higher levels of integration common to both multimodal and single-modal information are deficient in many dyslexic and dysgraphic children.

As we have seen, all these results indicate that higher central processes are involved in dyslexia. In reality all the above-listed studies examined only some aspects of the complex perception function, losing sight of the overall picture and attributing the explanation of dyslexia to disorders of some of the higher processes. Moreover there have been physiological studies such as that by Duffy et al. (1980) which, by recording cerebral electrical activity of normal and dyslexic children, have shown differences in the EEG spectra both at rest and during tests to activate the right (presentation of music and geometrical figures) and left hemispheres (reading exercises) alone or together (visual-verbal association).

Differences appear in the following areas: the frontomedial (supplementary motor area), the left frontal anterolateral (Broca's area), the left medial temporal (auditory association area), and the posterolateral parietal quadrant (Wernicke's area; parietal associative and visual associative areas). These studies show that numerous cerebral areas participate in the process of reading and that some of the brain areas are different in dyslexic and normal children, but they do not indicate which specific process is altered during reading.

To answer some of these questions, studies were conducted with sensory and cognitive evoked brain potentials (ERPs) [see Rosenthal et al. (1982) for a review of the topic]. Differences were seen in the amplitude and latency of time intervals of certain peaks in the evoked potentials. In particular, an increase in the latency of certain waves was generally interpreted as indicating a greater slowness of children in analyzing sensory information while a decrease in amplitude was interpreted as reduced "neural capacity."

Apart from some attempts to correlate dyslexic subgroups with some specific alterations in the ERPs (Rosenthal et al. 1982), it can be said that the majority of these studies suffer from the same limitations as the psychological studies in that they analyze a single aspect of the perception function complex, ignoring the fundamental notion that the perception function is principally an active process of extracting information from the environment. Especially when we face such complex functions we must be equipped with a method which can give an overall view and is at the same time able to analyze the different parts which make up the process. The method which we chose to study children with specific reading learning difficulties was to have them perform an integrated complex "task" (a motor-perceptive exercise) and to simultaneously record the performance, the electromyographic activity, and the brain electrical activity. The brain electrical activity accompanying the performance of this task is defined as movement-related brain macropotentials (MRBMs). The motor task consisted in starting the sweep of an oscilloscope trace by pushing a button with the thumb of the left hand and stopping it within 40–60 ms by pushing a button with the thumb of the right hand (Papakostopoulos 1978). The short time interval involved forced the subject to preprogram the task before it was carried out. The completion of such a task requires good bimanual coordination and the execution of ballistic movements. As visual feedback was provided in real time, the subject could adjust his strategy accordingly.

If we assume that reading is a complex and skillful process and consists of a set of modular subroutines serially and hierarchically organized, of which writing is the harmonic and integrated expression of a series of ballistic movements preprogrammed and correctable only after they have been executed and evaluated (kinesthetic feedback), a method which incorporates the study of motor performance, electromyographic activity, and MRBMs during the execution of the motor-perception ability task can supply useful information on those systems and subsystems which regulate and organize the motor-perceptive functions.

Recently Papakostopoulos (1978), in a critical review of the data from adults, proposed a taxonomy of such electrical phenomena. From observation of the

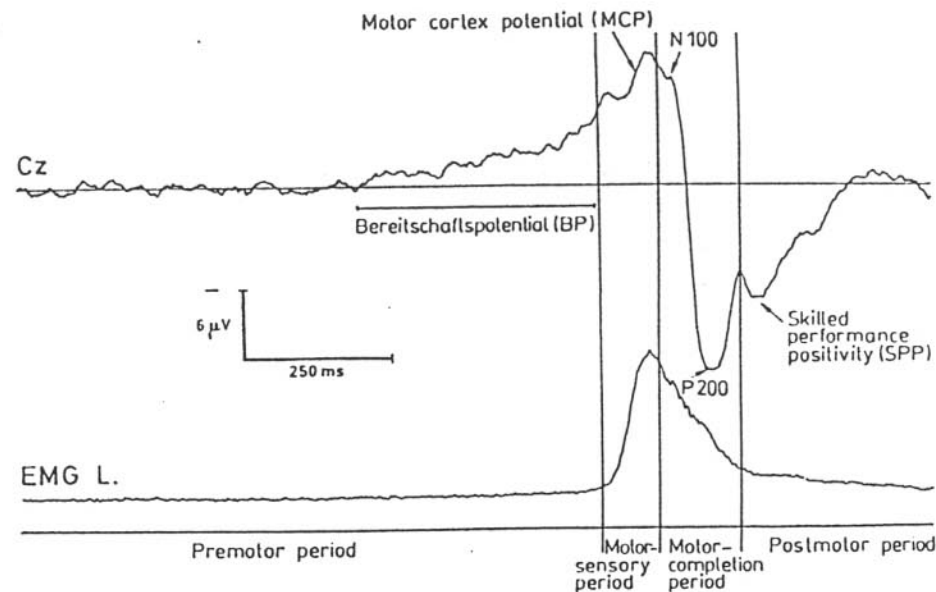


Fig. 1. Movement related brain macropotentials

myographic and brain electrical activity four periods can be distinguished: a premotor period, a motor-sensory period, a motor completion period, and a postmotor period (Fig. 1).

The premotor period is characterized by the presence of basic tonic muscular activity and the presence on the scalp of a phasic negative potential lasting 800–1200 ms. This potential is called the Bereitschaftspotential (BP) (Kornhuber and Deecke 1965; Vaughan et al. 1968). It is absent during passive movements. It has a low (5–7 μ V) amplitude during simple tasks and a higher amplitude during more complex tasks (Papakostopoulos 1978). It is recorded prevalently in the frontal and central regions. It is absent in children younger than 6 years and the amplitude increases progressively with age, reaching adult values at adolescence (Chiarenza et al. 1983). The BP is believed to reflect the process of organization and selection of the strategy needed to carry out the task.

The sensory-motor period lasts about 200 ms and begins at the onset of phasic electromyographic activity. It coincides with the appearance on the cortex of the motor cortex potential (MCP), a negative potential which follows the negative slope of the BP. The MCP is absent during passive movements, present in simple voluntary motor actions, and increases in amplitude during complex motor actions (Papakostopoulos 1978). It is recorded prevalently from the precentral and central regions and is absent at the parietal regions. The MCP has been proposed as an index of sensory information from the muscle, skin, and tendon receptors (Papakostopoulos et al. 1975).

completion period is characterized by the ending of the electromyographic phasic activity and by the presence of a negative cortical potential defined as N100 and a positive potential defined as P200 (Vaughan et al. 1968). P200 is considered to be the response normally evoked by the oscilloscope trace and is partially suppressed in the central and precentral areas during movement. It has a latency of 100 ms and is an index of visual perception processes. P200 is a positive potential following N100 with a latency of about 200 ms from the beginning of the light stimulus. This potential is present during passive and active movements, both simple and complex. This potential is thought to be one of the components of the late somatosensory potentials (Chiarenza et al. 1983).

The postmotor period is marked by electromyographic tonic activity similar to that in the premotor period, by the appearance in the cortex of a positive potential with a latency of about 450 ms, denominated "skilled performance positivity" (SPP) (Papakostopoulos 1978, 1980), and by a slow negative potential labeled "post-action negativity" (PAN) with a latency of about 600 ms (Chiarenza et al. 1983, 1984). The SPP is recorded mainly in the parietal regions and appears towards the 9th year in the frontocentral region. The SPP is present only when the subject can evaluate the result of his performance. This potential is independent of the motor act and of the presence of any exteroceptive stimulation (Papakostopoulos 1980). SPP coincides with the subject's awareness of success or failure in the performance. The PAN is recorded mainly in the frontocentral regions. This potential decreases in amplitude with age and disappears by about the 10th year. It appears to be related to analysis and evaluation strategies different from those generating the SPP (Chiarenza et al. 1983, 1984).

Method and Material

The subject sat in an armchair in front of a Tektronix 5111 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 excursion of the button was 5 mm. The task consisted in starting a sweep of the oscilloscope trace with the left thumb and stopping it in a predetermined part of the oscilloscope by pushing the other button with the right thumb. The speed of the trace was 10 ms/cm. The predetermined area corresponded to a time interval between 40 and 60 ms.

The time interval is measured and defined as "performance time." The distance from the target was also measured and defined as "performance shift." The number of performances reaching the target was measured and defined as "target performance." The number of performances shorter than 40 ms and longer than 60 ms was also measured.

After a verbal explanation of the task and before the placement of the electrodes, the subjects were allowed a short familiarization period and were asked to avoid eye movement or blinking during the execution of the task and to

keep an interval of 7–20 s between any two attempts. The subjects were also asked to remain relaxed and to avoid muscular preparatory movements before pressing.

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 k Ω . The time constant and high frequency were 4.5 s and 700 Hz for the EEG and 0.03 s and 700 Hz for the EMG respectively.

The EEGs and EMGs were recorded on magnetic tape for off-line analysis. The analysis begins with the acquisition of a $\pm 20 \mu\text{V}$ signal on each channel. During analysis, the arrival of the trigger signal, an electronic pulse generated by the left-hand button, starts the acquisition for each channel of 1600 points at a frequency of 500 Hz for 3.2 s. Of these points, 1100 precede the trigger and 500 follow it.

The first 500 points were averaged to give a baseline from which the amplitude of the potentials was measured. For every subject four blocks of 25 trials selected from those free of muscular artifacts, blinking, or eye movements were averaged and analyzed. The mean amplitude before movement, the peak amplitude during movement, and the rise time of the rectified electromyographic activity for the right and left forearms were measured. Further, the beginning of BP rise, called "BP onset," the total area of BP, and the mean amplitude of BP over the 200 ms prior to the beginning of movement were measured in the premotor period. In the sensory-motor period, the mean amplitude of the MCP, referred to the mean amplitude of the BP for 200 ms after the movement, and the latency of the MCP peak with respect to the beginning of the electromyographic phasic activity of the MCP peak were measured. In the motor completion period, the amplitudes of N100 and P200 from the baseline and with respect to the absolute amplitude of the MCP and their latency from the trigger were measured.

During the postmotor period, the mean amplitudes of SPP and PAN were taken as average values from the baseline over 200 ms centered around the main positive peak (SPP) and negative peak (PAN) in the latency band between 350 and 850 ms. SPP and PAN latencies were also measured from the trigger pulse.

There were 13 subjects aged 10 years, of whom nine were normal and four were dyslexic. All came from the same area of Milan and from the same school. All the children showed adequate visual acuity and normal hearing threshold.

To be considered as children with specific learning disabilities (LDs), they had to have an IQ of above 85 on both the Cattell and the WISC nonverbal performance test and to exceed the 5% tolerance limits on the reading and writing test in the Italian adaptation of the Metropolitan Achievement Test (Faglioni et al. 1970). Further, in order to obtain a better clinical and psychological evaluation, apart from the teacher's report and the medical history supplied by the parents, the following psychological tests were done: the Lincoln Oseretsky Motor

scale (Zucchi et al. 1959), the Bender Visual Motor Gestalt Test (1964), the Stamback test (Stamback 1965), the Goodenough test (Gesell and Matruđa 1974), and the Laterality test (Harris 1968).

Results

The neurological examination of the normal and LD children showed no classic signs of major or minor neurological damage. However, there were some consistent neurological signs in the group of LD children, even though they could not be grouped together as a well-defined clinical picture. The most frequent signs were disidiadochokinesia, motor clumsiness in fine manipulative activity, and synkinetic movements in the contralateral hand during the diadochokinesia test and finger opposition test. The quality of gross and fine movements was not optimal in terms of speed, adequacy, and fluidity. From the school reports of the LD children, the reading and writing difficulty was the problem most frequently noted, while signs of hyperactivity, impulsiveness, or attention disorders were rated as light. Comparison of the results of the psychological tests showed significant differences only on the WISC verbal tests (LDs = 100.25; normals = 134.44; $P < 0.05$) and the Oseretsky test, in which the LD subjects showed a lower developmental age than the normal subjects (LDs = 134.7; normals = 165.6; $P < 0.05$).

All the children completed their assigned motor-perceptive tasks, although with significant differences in terms of motor performance and MRBMs. The mean performance time was 99.44 ms for the children who had learning difficulties and 62.93 ms for the control group. The percentage of performances that could be defined as target performances was 14.25% and 26% respectively. Further, in LD subjects 66% of performances took longer than 60 ms compared with 47% in the normal children. The former were also less accurate, with a performance shift of 50 ms compared with 19.2 ms for the normal subjects. All these results were statistically significant ($P < 0.01$). Observing the performance during each of the four blocks of tests, it was apparent that the exercise produced a marked improvement in the LD children. In fact, their performance time fell from 112.03 ms in the first block to 62.61 ms in the fourth, while it fell from 67.5 ms to 60.47 ms among the normal children.

The accuracy of the LD children also showed the same improvement. The performance shift fell from 58.0 ms in the first block to 19.2 ms in the fourth, while it fell from 22.3 ms to 17.0 ms among the normal children. A comparison of the two groups of children shows significant differences ($P < 0.01$) in the first three blocks but not in the fourth for both performance time and performance shift.

The electromyographic activity of the two groups differed neither in amplitude of the electromyogram before and during the movement nor in the rise

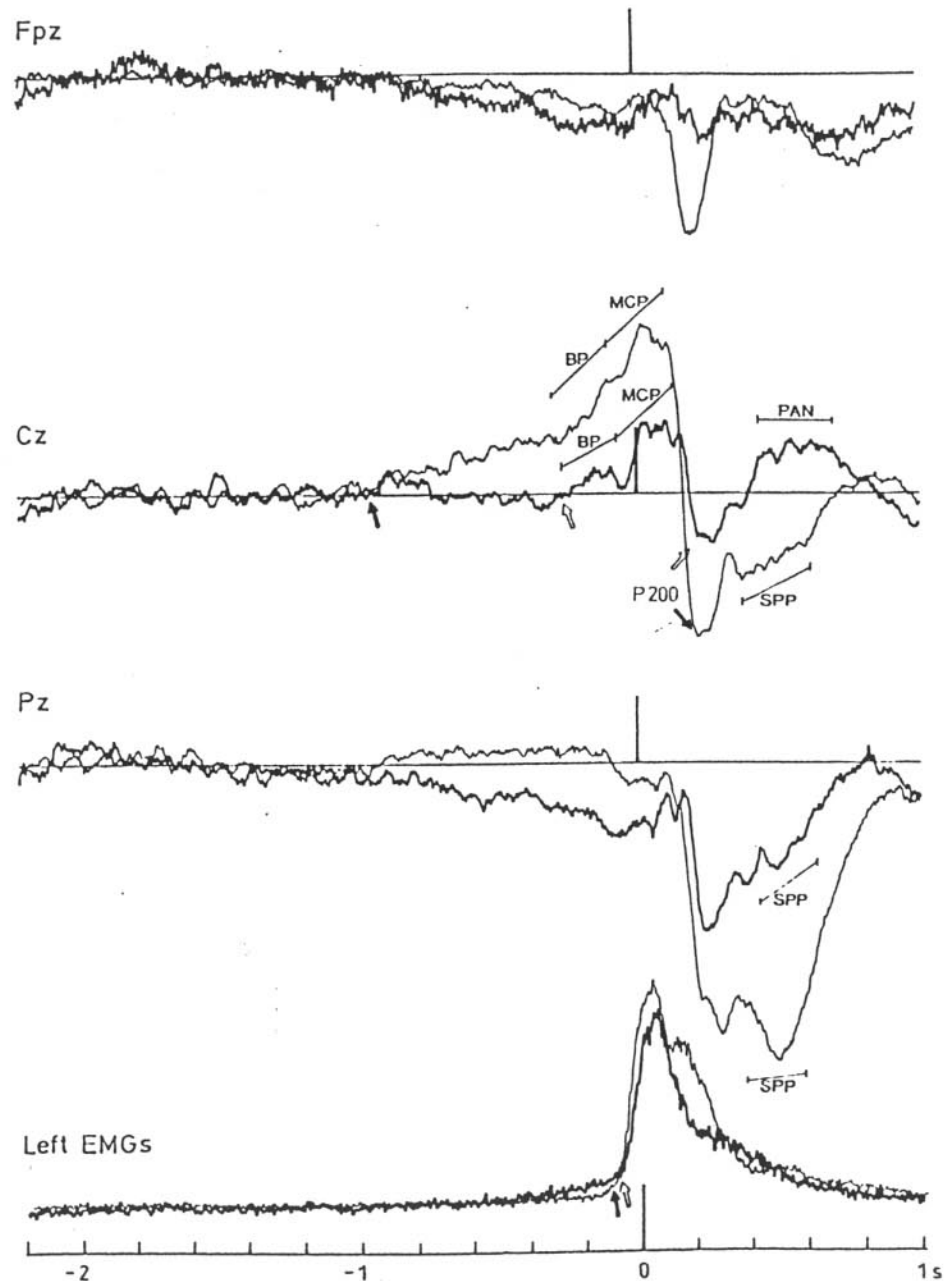


Fig. 2. Grand averages of MRBMs in normal (light lines) and dyslexic children (heavy lines). The divisions indicate the intervals of 200 ms over which the potentials shown above were measured. The arrows left from zero (black for the normal children and white for the dyslexic children) show the

differences were found in MRBMs during all four motor periods. The BP area in the dyslexic children in the premotor period was significantly reduced in the frontal central, precentral, and parietal regions (Fz, Cz, Pz, LPC: $P < 0.01$; RPC: $P < 0.05$). The BP amplitude 200 ms before the movement was reduced in Cz ($P < 0.05$) and Pz ($P < 0.01$). The BP onset was significantly delayed in the right and left precentral regions (RPC: $P < 0.05$; LPC: $P < 0.01$).

In the sensory-motor period the latency of MCP with respect to the EMG onset was greater, though not significantly so, in the frontal, central, and precentral regions of the dyslexic children. The amplitude of the MCP with respect to the BP amplitude was not different in the two groups.

In the motor completion period the latency of N100 was greater in the dyslexic children in the frontal, precentral, and central regions (Fz, Cz, RPC, LPC: $P < 0.05$) and increased further in the prefrontal and parietal regions (FPz, Pz: $P < 0.01$). There were no significant differences between the two groups in the latencies of P200 except in Fpz ($P < 0.05$). The amplitude of P200 with respect to the absolute amplitude of MCP was reduced in the LD children in all the brain areas (Fpz, Fz, Cz, Pz, RPC, LPC: $P < 0.05$). In the postmotor period, SPP was present in all brain areas of the normal subjects while in LD subjects it was only present in Pz and with a significantly reduced amplitude ($P < 0.05$). The latency of SPP in Pz was greater in children with learning difficulties than in normal children (LDs = 531.6 ms; normals = 495.2 ms). On the other hand, PAN was present in the frontal, central and precentral areas.

Discussion

The above results show that there are psychological and neurophysiological differences between normal children and children with learning difficulties. The most consistent differences are at the level of quality of movement—speed, fluidity, and adequacy—as shown by the neurological tests and the Oseretsky psychomotor examination. Children with learning difficulties showed lower than actual age in both motor development and in some tests of the neurological examination. These signs have been found previously, but their significance is still a topic of discussion since some are a matter of development and are present to a small extent in the normal population. Their presence could be interpreted as a sign of insufficient coordination and temporal control in motor sequencing.

Part of the preparation for movement is the activation of a central clock which controls the time sequencing of the motor action through afferent and efferent systems (Hirsch and Sterrich 1964; Rosenbaum and Patashnik 1980). The improvement of the performance of this clock depends on a greater synaptic efficiency of the central nervous system which in turn depends on age (Craik 1947) and on the presence of internal and external feedback on the accuracy of the performance.

Our subjects with learning difficulties showed themselves to be slower and less accurate and to have totalled a lower number of target performances in carrying out the motor-perceptual task. Further, a different trend in performance improvement was seen between the two groups of children during the experiment. The normal children achieved a better motor performance than the LD children and reached their peak in the second block, which they thereafter maintained. The children with learning difficulties, however, although they began with a much worse motor performance, improved steadily over the whole period of the experiment, reaching the level of the normal children only in the last 25 trials.

To these observations must be added the behavior observed during the performance of the task on closed circuit television and during the selection of the tests without artefacts. Compared with the normal children, those with learning difficulties had greater difficulty in controlling irrelevant and inappropriate movement, such as blinking immediately before or after the performance of the task or gross movement of the body or lower limbs. If all the results of the neurological tests, the Oseretsky test, and the performance of the motor-perception task are combined, it could be said that the setting of the central clock proceeds with difficulty. Further, these data seem to suggest that the control of the temporal sequencing of movements, based on the processing of internal and external feedback, does not occur in an appropriate way because, as we shall see later, MCP, N100, and SPP are altered in different ways in the children with learning difficulties.

It is known that children with learning difficulties have difficulty in executing simple motor tasks (Lewis et al. 1970; Pyfer and Carlson 1972; Bruininks and Bruininks 1977) and tests of bimanual coordination (Klicpera et al. 1981). Particularly insufficient motor performance has been observed in subjects with commissurotomy of the corpus callosum and it has been proposed that integrity of the callosum commissurae is essential to the performance of bimanual tasks (Kreuter et al. 1972; Preilowski 1972; Zaidel and Sperry 1977). It is not yet possible to ascertain to what extent these data agree with our observations. It is useful to point out that the myelination of the corpus callosum is completed approximately at the age of 10 years (Yakovlev and Lecours 1967).

In parallel with the motor performances, the MRBMs showed significant differences in normal and LD children.

The BP is a feature of the premotor period, when the ideomotor elements of the movement are being organized. In the LD children BP was reduced in amplitude in the parietal, central, and precentral areas and furthermore began only about 100 ms before the movement. It has recently been proposed that the BP could result from two components: the first begins 1.2 s before the movement and lasts for about 450/600 ms, and is followed by the second, characterized by a steep negative fall lasting 300/500 ms (Shibasaki et al. 1980). From the study of the development of the BP it was hypothesized that the first component might be linked to processes related to the representation of the action, while the second is the most operative part of the process and therefore the most automatic.

za, in preparation). This first component was absent in the LD children the second was considerably reduced in amplitude.

The sensory-motor period is characterized by the MCP. The MCP reflects processing of the kinesthetic reafferent information in the precentral and frontal areas related to the movement carried out. The MCP was of normal amplitude in LD children but the peak of this potential had a greater latency than in normal children. Experiments with animals (Dubrovsky and Garcia-Rill 1971) and observations of patients with posterior column lesions have shown that total or partial deafferentation impedes the temporal control of a motor sequence. The increase in latency of the MCP may be interpreted as a difficulty of these children in processing the kinesthetic feedback.

The processing of the visual information, as represented by N100, the brain response evoked by the appearance of the light trace on the oscilloscope, was delayed in children with learning difficulties. It was delayed in the frontal central and precentral regions and even more so in the prefrontal and parietal regions. The P200 is thought to be one of the late components of the somatosensory potentials (Chiarenza et al. 1983); the reduction in amplitude of P200 seen in the various brain areas of the LD children but not in the normals could indicate a defect in the integration of the reafferent kinesthetic information.

The SPP was present only in the parietal regions in LD children and with reduced amplitude. There was increased latency compared with normal children. This may suggest difficulty for these children in the awareness and evaluation of their own performance. The PAN was present in the frontal, central, and precentral regions. This potential is recorded more often in children below the age of 10 years (Chiarenza et al. 1983). If, instead of the sequential averages, we look only at the averages of the MRBMs related to the target performance (Fig. 3), we see that there is SPP even in the LD children, in the frontocentral and precentral areas, even though the amplitude is considerably reduced compared with normal children. So the presence of PAN in the LD children could be due to a different strategy 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.

Conclusions

Perception is an active process which requires the participation of various subsystems. The present data seem to indicate that during a motor-perceptual act, the different processes involved in the various brain areas are defective in children with learning difficulties. In particular, it can be stated that systems involved in the planning and programming of effective strategies are inadequate, and that, furthermore, those involved in verification and correction of errors are less efficient. These systems may be altered in themselves or may reflect deficiencies in those subsystems concerned with kinesthetic and visual process-

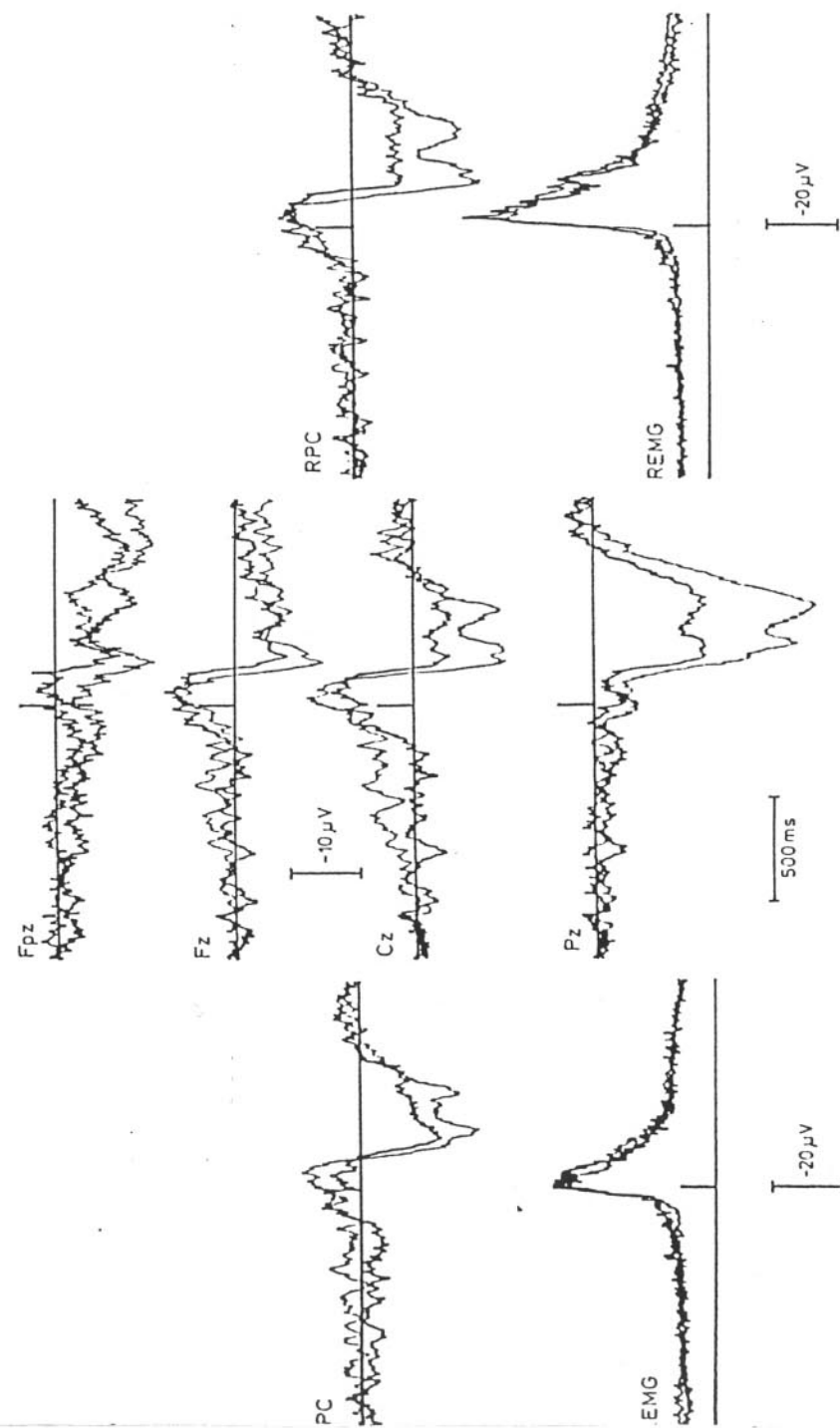


Fig. 3. MRBMs related to target performance of normal (light lines) and dyslexic children (heavy lines). Note the appearance of the SPP in the frontal, central, and precentral areas of the dyslexic children, even though the amplitude is lower than that of the normal children when they hit the target.

or they could be potentially adequate but not fully developed. Therefore, it can be said that dyslexia results from the defective integration and dysfunction of numerous processes which occur on different levels and at different times (Chiarenza et al. 1986).

Summary

Perception can be regarded as an active process of extracting information from the environment. It includes a variety of processes and is essential in learning to read and to write. Until now psychological and neurophysiological research has mainly examined isolated processes of perceptual function. The author suggests a method for evaluating the perceptual motor function by recording the MRBMs during the execution of a motor perceptual task.

The results obtained in a group of dyslexic children point to the conclusion that the subsystems for programming and verifying strategies are less efficient in these children; furthermore, those subsystems related to evaluation of kinesthetic and visual information are deficient.

It is hypothesized that dyslexia and dysgraphia are the results of a defective integration of the subsystems mentioned above.

References

- Aaron PG, Bakker C (1982) The neuropsychology of dyslexia in college students. In: Malatesha RN, Hartlage LC (eds) *Neuropsychology and cognition*, vol 1. Sijthoff and Noordhoff, Alphen aan den Rijn, pp 128-146
- Bakker DJ (1967) Temporal order, meaningfulness, and reading ability. *Percept Mot Skills* 24:1027-1030
- Bakker DJ (1970) Temporal order perception and reading retardation. In: Bakker DJ, Satz P (eds) *Specific reading disability: advances in theory and method*. Rotterdam University Press, Rotterdam, pp 81-92
- Bakker DJ (1972) Temporal order in disturbed reading. Rotterdam University Press, Rotterdam
- Bannatyne AD (1966) The etiology of dyslexia and the color phonics system. In: Money J (ed) *The disabled reader: education of the dyslexic child*. Johns Hopkins University Press, Baltimore, pp 97-107
- Bateman BC (1968) Interpretation of the 1961 Illinois test of psycholinguistic abilities. Special Child Publications, Seattle
- Belmont L (1980) Perceptual organization and minimal brain dysfunctions. In: Rie HE, Rie ED (eds) *Handbook of minimal brain dysfunctions. A critical view*. Wiley, New York, pp 253-271
- Benton AL (1962) Dyslexia in relation to form perception and directional sense. In: Money J (ed) *Reading disability: progress and research needs in dyslexia*. Johns Hopkins University Press, Baltimore, pp 25-32
- Birch HG (1962) Dyslexia and the maturation of visual function. In: Money J (ed) *Reading disability: progress and research needs in dyslexia*. Johns Hopkins University Press, Baltimore, pp 1-47
- Birch HG, Belmont L (1964) Auditory-visual integration in normal and retarded readers. *Am J Orthopsychiatry* 34:852-861
- Birch HG, Belmont L (1965) Auditory-visual integration, intelligence, and reading ability in school children. *Percept Mot Skills* 20:295-305

- Birch HG, Lefford A (1963) Intersensory development in children. *Monogr Soc Res Child Dev* 28, Serial no 89
- Boder E (1973) Developmental dyslexia: a diagnostic approach based on three atypical reading patterns. *Dev Med Child Neurol* 15:663-687
- Bruininks VI, Bruininks RH (1977) Motor proficiency of learning disabled and non-disabled students. *Percept Mot Skills* 44:1131-1138
- Chiarenza GA, Papakostopoulos D, Giordana F, Guareschi Cazzullo A (1983) Movement related brain macropotentials during skilled performances. A developmental study. *Electroencephalogr Clin Neurophysiol* 56:373-383
- Chiarenza GA, Tengattini MB, Grioni A, Ganguzza D, Vasile G, Massenti A, Albizzati A, Papakostopoulos D, Guareschi Cazzullo A (1984) Long latency negative potentials durante un compito percettivo motorio. Caratteristiche evolutive in bambini normali. *Riv Ital EEG Neurofisiol Clin* 7:538-541
- Chiarenza GA, Papakostopoulos D, Grioni A, Tengattini MB, Mascellani P, Guareschi Cazzullo A (1986) Movement related brain macropotentials during a motor perceptual task in dyslexic and dysgraphic children. In: McCallum WC, Zappoli R, Denoth F (eds) *Cerebral psychophysiology studies in event related potentials (EEG Suppl 38)*. Elsevier, Amsterdam, pp 489-491
- Craik KJW (1947) Theory of the human operator in control systems. *Br J Psychol* 38:56-61
- de Hirsch K, Jansky J, Langford W (1966) Predicting reading failure. Harper and Row, New York
- Duffy FH, Denckla MB, Bartles PH, Sandini G (1980) Dyslexia: regional differences in brain electrical activity by topographic mapping. *Ann Neurol* 7:412-420
- Dubrovsy B, Garcia-Rill E (1971) Role of dorsal columns in sequential motor acts requiring precise forelimb projection. *Exp Brain Res* 18:165-177
- Faglioni P, Gatti B, Paganoni AM, Robutti A (1970) Test di valutazione del linguaggio scritto. Manuale di istruzioni. Edizioni Organizzazioni Speciali, Firenze
- Gesell A, Amatruda C (1974) *Developmental diagnosis*, 2nd edn. Hoeber, New York
- Gibson EJ (1969) *Principles of perceptual learning and development*. Appleton-Century-Crofts, New York
- Groenendaal HA, Bakker DJ (1971) The part played by mediation processes in the retention of temporal sequences by two reading groups. *Hum Dev* 14:62-70
- Hynd GW, Obrzut JE, Weed W, Hynd CR (1979) Development of cerebral dominance: dichotic listening asymmetry in normal and learning disabled children. *J Exp Child Psychol* 28:445-454
- Harris AJ (1968) Harris test of lateral dominance, 3rd edn. Psychological Corporation, New York
- Hirsch IJ, Sterrick CE (1964) Perceived order in different sense modalities. *J Exp Psychol* 67:103-112
- Ingram TTS (1963) Delayed development of speech reference to dyslexia. *Proc R Soc Med* 56:199-205
- Ingram TTS, Mason AW, Blackburn I (1970) A retrospective study of 82 children with reading disability. *Dev Med Child Neurol* 12:271-281
- Jansky J, de Hirsch K (1972) Preventing reading failure—prediction, diagnosis and intervention. Harper and Row, New York
- Johnson DJ, Myklebust HR (1967) *Learning disabilities: educational principles and practices*. Grune and Stratton, New York
- Kinsbourne M, Warrington E (1966) Developmental factors in reading and writing backwardness. In: Money J (ed) *The disabled reader: education of the dyslexic child*. Johns Hopkins University Press, Baltimore, pp 77-83
- Klicpera C, Wolff PH, Drake C (1981) Bimanual co-ordination in adolescent boys with reading retardation. *Dev Med Child Neurol* 23:617-625
- Koppitz EM (1964) The Bender Gestalt test for young children. Grune and Stratton, New York
- Kornhuber HH, Deecke L (1965) Hirnpotentialänderungen bei Willkürbewegungen und passiven Bewegungen des Menschen: Bereitschaftspotential und reafferente Potentiale. *Pflügers Arch* 284:1-17
- Kreuter C, Kinsbourne M, Trevarthen C (1972) Are disconnected cerebral hemispheres independent channels? A preliminary study of the effects of unilateral loading on bilateral finger tapping. *Neuropsychologia* 10:453-461
- Lewis FD, Bell DB, Anderson RP (1970) Relationship of motor proficiency and reading retardation. *Percept Mot Skills* 31:395-401
- Lyle JG, Goyen J (1968) Visual recognition, developmental lag and strephosymbolia in reading retardation. *J Abnorm Psychol* 73:25-29
- Lyle JG, Goyen J (1975) Effects of speed of exposure and difficulty of discrimination on visual recognition of retarded readers. *J Abnorm Psychol* 8:613-616

- AR (1973) *The working brain*. Penguin, London
- Yklebust HR (1965) Development and disorders of written language: picture story language test. Grune and Stratton, New York
- Papakostopoulos D (1978) Electrical activity of the brain associated with skilled performance. In: Otto DA (ed) *Multidisciplinary perspectives in event-related brain potential research*. US Environmental Protection Agency, Washington DC, pp 134-137
- Papakostopoulos D (1980) A no stimulus no response event-related potential of the human cortex. *Electroencephalogr Clin Neurophysiol* 48:622-638
- Papakostopoulos D, Cooper R, Crow HJ (1975) Inhibition of cortical evoked potentials and sensation by self-initiated movement in man. *Nature* 258:321-324
- Preilowski BFB (1972) Possible contribution of the anterior forebrain commissures to bilateral motor coordination. *Neuropsychologia* 10:267-277
- Pyfer III, Carlson BR (1972) Characteristic motor development of children with learning disabilities. *Percept Mot Skills* 35:291-296
- Rabinovitch RD (1968) Reading problems in children: definitions and classification. In: Keeney AH, Keeney VT (eds) *Dyslexia: diagnosis and treatment of reading disorders*. Mosby, St Louis, pp 28-48
- Rosenbaum DA, Patashnik O (1980) A mental clock setting process revealed by reaction times. In: Stelmach GF, Requin J (eds) *Tutorials in motor behavior*. North-Holland, Amsterdam, pp 1-14
- Rosenthal JH, Boder E, Callaway E (1982) Typology of developmental dyslexia: Evidence for its construct validity. In: Malatesha RN, Aaron PG (eds) *Reading disorders, varieties and treatment*. Academic, New York, pp 93-120
- Rourke BD (1976) Issues in the neuropsychological assessment of children with learning disabilities. *Can Psychol Rev* 17:89-102
- Satz P, Sparrow S (1970) Specific developmental dyslexia: a theoretical reformation. In: Bakker DJ, Satz P (eds) *Specific reading disability: advances in theory and method*. University of Rotterdam Press, Rotterdam, pp 17-40
- Satz P, Rardin D, Ross J (1971) An evaluation of a theory of specific developmental dyslexia. *Child Dev* 42:2009-2021
- Satz P, Friel J, Rudegeair F (1974) Differential changes in the acquisition of developmental skills in children who later became dyslexic. In: Stein DG, Rosen JJ, Butters N (eds) *Plasticity and recovery of function in the central nervous system*. Academic, New York, pp 88-98
- Senf GM (1969) Development of immediate memory for bisensory stimuli in normal children, and children with learning disabilities. *Dev Psychol* 6:28-32
- Senf GM, Feshback S (1970) Development of bisensory memory in culturally deprived dyslexic and normal readers. *J Ed Psychol* 61:461-470
- Senf GM, Freundl PC, Silver A, Hagen R (1971) Memory and attention factors in specific learning disabilities. *J Learn Dis* 4:94-106
- Shibasaki H, Barrett G, Halliday E, Halliday AM (1980) Components of the movement-related cortical potential and their scalp topography. *Electroencephalogr Clin Neurophysiol* 49:213-226
- Stamback M (1965) Epreuves de niveau et de style moteurs. *Actualités pédagogiques et psychologiques*. Fasc 2:5-65
- Vaughan GH, Costa LD, Ritter W (1968) Topography of the human motor potential. *Electroencephalogr Clin Neurophysiol* 25:1-10
- Wepman JM (1962) Dyslexia: its relationship to language acquisition and concept formation. In: Money J (ed) *Reading disability. Progress and research needs in dyslexia*. Johns Hopkins University Press, Baltimore, pp 48-62
- Witelson SF (1976) Abnormal right hemisphere specialization in developmental dyslexia. In: Knights RM, Bakker DJ (eds) *Neuropsychology of learning disorders: theoretical approaches*. University Park Press, Baltimore, pp 233-256
- Yakovlev PI, Lecours AR (1967) Myelogenetic cycles of regional maturation in the brain. In: Minkowski A (ed) *Regional development of the brain in early life*. Blackwell, Oxford, pp 3-70
- Zaidel D, Sperry RW (1977) Some long-term motor coordination effects of cerebral commissurotomy in man. *Neuropsychologia* 15:193-204
- Zucchi M, Giuganino BM, Stella L (1959) Adattamento italiano della scala di sviluppo motorio di Oseretsky. *Boll Psicol Sociol Appl* 31-36