Rate and Timing Precision of Motor Coordination in Developmental Dyslexia

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Learning to read builds on the speech processes of the child at many levels (Denckla, 1979; Mann, 1986), and language impairment is one of the most common behavioral correlates of developmental dyslexia (Doehring, Trites, Patel, & Fiedorowicz, 1981; Jorm, 1979; Vellutino, 1978). Current dyslexia research therefore has emphasized the linguistic analysis of language deficits and has brought converging evidence to the effect that phonological processing deficits, for example, play a critical role in reading impairment (Mann, 1986; Wagner & Torgensen, 1987).

However, neuropsychological investigations have identified various biological and neurological correlates of developmental dyslexia including abnormal patterns of hemispheric specialization and genetic variations (Denckla, 1979; Hiscock & Kinsbourne, 1982; Smith, Goldgar, Pennington, Kimberling, & Lubs, 1986), the functional significance of which cannot be clarified by a linguistic analysis alone (Ellis, 1985; Tallal, Stark, & Mellits, 1985). An alternative research strategy has therefore emerged that recognizes the importance of language disabilities as proximal causes, but focuses on underlying "physiologically plausible" processes (Hammond, 1982) that may account both for the nonlinguistic, noncognitive behavioral correlates and the language impairment in developmental dyslexia.

Lashley's classic hypothesis that speech and skilled movements share neural mechanisms of timing precision and serial order control (Lashley, 1937, 1951; Lenneberg, 1967) has motivated a series of neuroanatomical and physiological investigations indicating that brain mechanisms for the temporal organization of language processes overlap extensively with those for skilled manual action (Bellman & Walter, 1984; Kimura & Archibald, 1974; MacNeilage, 1970; Ojemann, 1984). Related studies by others have demonstrated striking similarities in the dynamical properties of speech production and motor coordination (Kelso & Tuller, 1984). More specifically, it has been proposed that from an evolutionary and ontogenetic perspective, bimanual coordination may be the most direct functional precursors of speech and language (MacNeilage, Studdert-Kennedy, & Lindblom, 1984).

Although no direct causal relation between the development of language and movement can be inferred from such findings, the two domains appear to be related functionally by their common dependence on processes of temporal resolution (Shaffer, 1982). Therefore, one might expect that temporal resolution deficits that manifested clinically in the language impairment of dyslexic subjects will also be expressed in their time-dependent nonlinguistic behavior. In fact, dyslexic children have significantly greater difficulty than do normal readers in maintaining the correct tempo, prosody, and rhythm in language, reading, writing, and other skilled manual actions (Corkin, 1974; Denckla, 1979; Hanes, 1986), and in decoding the temporal sequence of auditory and visual stimuli (Bakker, 1972; Tallal, 1980; Zurif & Carson, 1970).

Such evidence gives partial support to the generic hypothesis that impaired temporal resolution is a domain-general deficit in some subtypes of dyslexia and to the corollary hypothesis that such temporal resolution deficits are related to left-hemisphere dysfunctions (Hammond, 1982; Tzeng & Wang, 1984). However, dichotic listening and tachistoscopic hemifield studies have brought only marginal support for the hypothesis of left-hemispheric dysfunctions or abnormal patterns of hemispheric specialization in dyslexia (Pirozzolo, Rayner, & Hynd, 1983), and there is no persuasive evidence that domain-general "pattern generators" or pacemaker clocks of the brain or mind control the timing of all psychologically complex human behavior, regardless of the material being sequenced (Ellis, 1985; Michon, 1967). Instead, the sequencing deficits of dyslexic subjects are selective rather than universal (Bakker, 1972; Vellutino, 1978; Zurif & Carson, 1970). Furthermore, the variables of temporal resolution presumably implicated in dyslexic subjects are rarely undefined (Denckla, 1979). An adequate test of the hypothesis would require a decomposition of the global concept of sequencing into specific temporal variables (e.g., rate, timing precision, serial order) and a specification of the boundaries within which the temporal resolution deficits are manifested.

Our previous studies (Klicpera, Wolff, & Drake, 1981; Wolff, Cohen, & Drake, 1984) have indicated that 11–13-year-old dyslexic students had great difficulty coordinating asymmetric finger responses of the left and right hands in a unified action, although they performed as well as normal readers on unimanual tasks or when the two hands responded in unison. However, even normal readers from 11 to 13 years had difficulty performing the asymmetric bimanual task, whereas normal adults do not. Thus, the results did not clarify whether impaired bimanual performance reflects a relative central nervous system immaturity or a developmentally stable deficit. Because performance was only tested at one (fast) metronome speed, the results also did not clarify whether the poor bimanual performance of dyslexic subjects was due to a general slowness of motor responding or whether it reflected a specific impairment of interlimb coordination and therefore perhaps of inter-hemispheric communication.

For the present study, we examined subjects on the same bimanual coordination tasks, but tested them at three different metronome speeds. Furthermore, we examined dyslexic adults as well as older adolescents to test whether the coordination deficit is compensated during the later growth years, and we balanced the adolescent samples for sex to test the clinical impression that dyslexic women, although generally underrepresented in the general population, are on the average more severely affected than dyslexic men (Satz & Zaide, 1983).

Method

Subjects

Dyslexic students

The adolescent sample included 25 male and 25 female dyslexic students from 13 to 18 years of age (5 boys and 5 girls in each 12-month age group) enrolled in a residential school for reading-impaired students of normal intelligence. Students at the school are tested every year for psychometric intelligence, language performance, and academic achievement by trained personnel. Subjects were selected on the basis of evaluations that had been performed within the preceding 12 months. To be included in the study, subjects had to (a) read at least two grades below expected level for age on the Gray Oral Reading Test (Wiederholt & Bryant, 1986) but have at least average psychometric intelligence as measured by the full scale Wechsler Adult Intelligence Scale–Revised (FS WAIS–R; Wechsler 1981), (b) be free of clinically significant neurological, organic, or uncorrected sensory deficits, (c) have been exposed to an adequate learning environment before enrolling in the residential school, and (d) come from a middle- to upper middle-class family. All of the students in the final sample scored at or above 95 on the FS WAIS–R and were reading at least 2½ grades below expectancy for age on the Gray Oral Reading Test. Some were still reading at a second-grade level (age equivalent = 7 years), but the majority read at a third–sixth-grade level (age equivalent = 8–11 years). By the Hollingshead-Redlich (1958) scale of father's occupation, their mean socioeconomic status was 2.4. Hand preference was examined by a gestural modification of the Oldfield Handedness inventory (Oldfield, 1971), and all subjects were classified as primarily right-handed, left-handed, or ambidextrous.

Adult dyslexic subjects were recruited from a residential college for young dyslexic adults. Subjects were included in the study if they had a history of persistent reading disabilities and were still reading below a ninth grade level on the Gray Oral Reading Test, but had a FS WAIS–R of at least 90. The final sample was composed of 41 men who had an IQ of at least 95 (FS WAIS–R); 7 students were above 24 years of age (25–32 years), and the other 34 students were between 18–23 years. Some were reading and spelling at a third-grade level, whereas most were reading at a fourth–seventh-grade level and spelling at a fifth–sixth-grade level. The number of dyslexic female adults meeting the selection criteria was not sufficient for inclusion in the analysis of results.

Pathological control subjects

The specificity of bimanual coordination deficits was tested by examining a control group of 50 learning disabled adolescent students without current or past reading difficulties. However, it was difficult to define a homogeneous pathological control group; many learning disabled students without evidence of reading disorders by standardized tests nevertheless either exhibit concomitant reading and language difficulties or else suffer from clinically significant neurological disabilities (Rourke, 1988). Instead, we therefore selected learning disabled students without neurological deficits, who could be case-matched with dyslexic subjects for sex, chronological age (±3 months), psychometric intelligence (±5 points, FS WAIS–R), and socioeconomic status (M = 2.3) ; who were reading at or above grade level and had no history of reading or spelling difficulties; and who had been identified by special educators as having either organizational problems in written work, poor study skills, or isolated difficulties in mathematics.

Normal control subjects

Adolescents were recruited from two affluent middle-class suburban schools whose student bodies had the same demographic characteristics as did the samples of dyslexic and pathological control students. School policies on confidentiality denied us access to student records, and we were asked not to administer individual psychometric intelligence tests. School guidance counselors were therefore asked to identify students with full scale IQs in the 95–110 range, who were reading within one grade level for age, and who had no history of reading difficulties. From this pool, we selected a final sample who could be matched with dyslexic students by sex, chronological age (±3 months), and socioeconomic status (2.4).

Adults

A total of 41 adult students without any individual or family history of reading difficulties, who could be matched with dyslexic adults by sex and chronological age (±12 months), were recruited from local colleges and universities. The sample included 34 undergraduates and 7 graduate students. No formal IQ tests were administered because of the students' time constraints.

All available psychometric data and achievement scores are summarized in Table 1; achievement scores are reported as grades below expected level for age (adolescents) or as years below a 12-grade ceiling (adults).

Table 1
Subject Characteristics

Variable	Subjects					
	Adolescent dyslexic		Pathological control		Adult dyslexic	
	M	*SD*	*M*	*SD*	*M*	*SD*
n	50		50		41	
Age (in years)	15.1	2.3	15.2	2.4	22.6	4.7
Full scale IQ	104.7	8.5	107.3	11.8	107.7	4.8
Verbal IQ	101.9	10.5	107.7	12.3	103.8	8.8
Performance IQ	107.4	11.5	105.1	13.3	110.9	13.5
Academic achievement						
Gray Oral Reading Test	−4.7	2.7	−0.2	0.2	−6.6	2.8
Comprehension	−3.0	2.5	−0.8	2.4	−2.3	1.6
Spelling	−3.8	1.8	−1.1	1.3	−7.4	3.1
Arithmetic	−2.0	2.6	−1.8	2.0	−1.8	2.4

Note. Academic achievement scores are reported as grades below expected level for age (adolescents) or below a 12th-grade ceiling (adults).

Procedure

Apparatus and measurement

Motor responses were recorded from two 8-cm × 10-cm brass plates mounted on a bakelite surface and placed on the table at a comfortable height in front of the seated subject. Finger contact with the plates generated voltage signals of fixed duration (50 ms), which were interfaced with two separate channels of a microprocessor (MAC-SYM 2, Analog Devices Co.) for data storage and off-line analysis. Subjects moved the index fingers of both hands vertically at the metacarpo-phalangeal joint while supporting the hand on the table. Practice trials were given before each set of test trials to familiarize subjects with the task. Subjects were also informed that on a signal from the examiner the metronome would be turned off in the middle of the trial, but they should continue tapping in the same pattern until told to stop. After listening to the beat of an electronic metronome for 15 s without tapping their fingers, they synchronized their motor responses to the metronome beat for 20 s (synchronization condition) and then continued tapping after the metronome was turned off (continuation condition) until told to stop. A total of 60 responses were collected by the computer during the continuation condition.

The three conditions of interlimb coordination (see also Wolff, Cohen, & Drake, 1984) were (a) tapping in unison so that each finger responded symmetrically and synchronously to each beat of the metronome, (b) tapping in rhythmic alternation so that each finger responded symmetrically but asynchronously to every other beat of the metronome, and (c) tapping asymmetrically and asynchronously so that the (right or left) leading finger had to respond to each beat of the metronome while the nonleading finger responded to every other beat of the metronome or at half the rate of the leading finger.

Each condition was tested at metronome speeds of 92, 120, and 184 beats per minute (BPM); for purposes of data analysis, the fastest speed was chosen to be a multiple of the slowest speed. Metronome entraining speed and finger responding rate were the same in the unison condition, but had to be distinguished for bimanual alternations when each finger responded at one half the metronome speed, and for the asymmetric tapping condition when one finger responded at the metronome speed while the other responded at one half that speed.

Data analysis

The comparisons were based on three main outcome variables. The first was the standard deviation of interresponse intervals (IRI), computed separately for the left and right fingers for each trial in each subject. This variable served as a measure of tapping variability. Because the measure varied inversely with finger-responding rate or metronome speed, it was not used to test the effects of responding rate on timing precision. Rate effects were tested by computing tapping errors, defined as the number of IRI in each trial that exceeded

the standard deviation of IRI for that trial by 2 SD or more, so that the measure was not systematically affected by finger responding rate. Tapping errors were informative only in asymmetric trials.

As an additional clue to mechanisms of impaired bimanual coordination, we computed tapping ratios for each trial, defined as the proportion of responses by the leading finger divided by responses of the nonleading finger. The theoretically correct ratio was always 2.0; to control for measurement errors, only ratios outside the range < 1.8−>2.2 were scored as deviant. Ratios less than 2.0 indicated that the nonleading finger had overresponded relative to the leading finger, whereas ratios greater than 2.0 indicated that the leading finger had overresponded relative to the nonleading finger.

Most comparisons were based on a repeated measures analysis of variance (ANOVA) design, with the Geisser–Greenhouse correction for degrees of freedom to control for chance effects (Geisser & Greenhouse, 1958) and post hoc Scheffe' tests to decompose multifactorial variables and interaction effects. Data on adults and adolescents were analyzed separately because the adolescent samples included male and female as well as pathological control students, whereas the adult samples included only normal and dyslexic men.

The analysis of tapping variability was performed in two major steps. In the first set of analyses, we compared performance across responding rate and bimanual condition, and in the second set, across condition tapping variability while controlling for finger responding rate. At slow responding rates, this was accomplished by comparing unison trials at 92 BPM (Condition 1 in Figure 1), alternation trials at 184 BPM (Condition 2 in Figure 1), and performance of the nonleading finger in asymmetric trials at 184 BPM (Condition 3 of Figure 1). At fast responding rates, this was accomplished by comparing unison trials at 184 BPM and performance of the leading finger (asymmetric trials) at 184 BPM (Conditions 4 and 5 of Figure 1). Similar analyses at the same finger responding rate across conditions at 120 BPM were based on a comparison of alternation trials and the nonleading finger (slow responding rate) or on unison trials and the leading finger (fast responding rate).

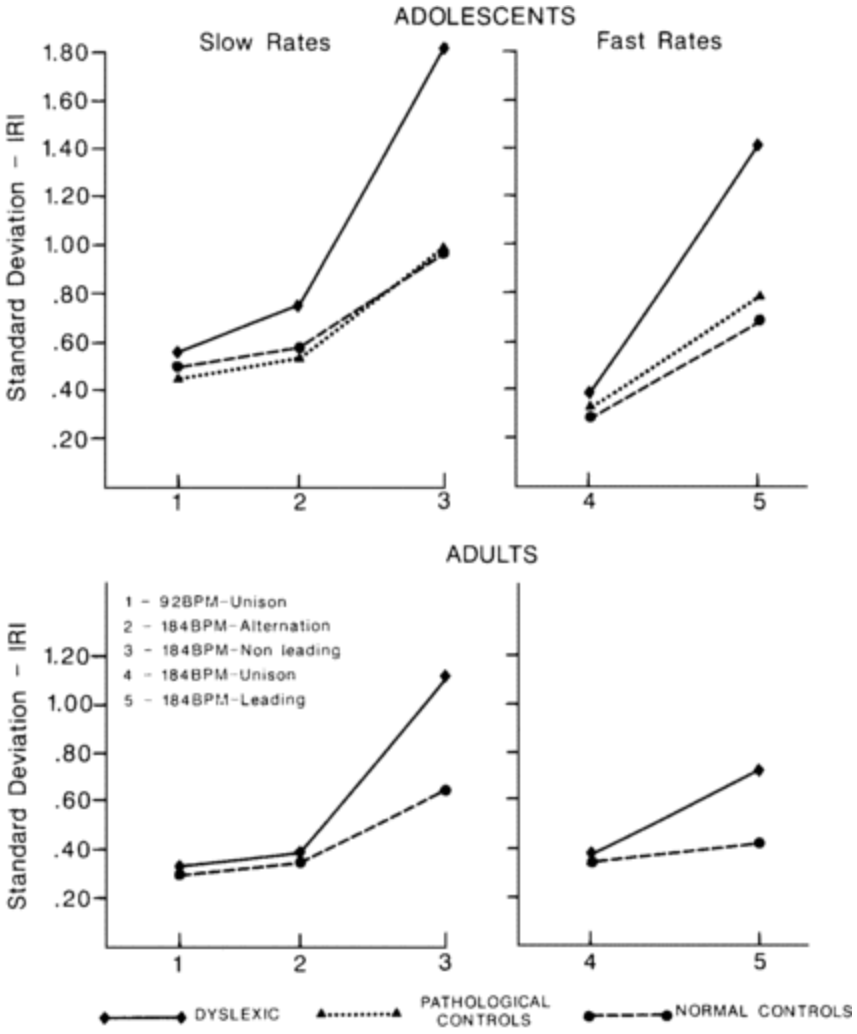

Figure 1. Variability of interresponse intervals (IRI) across bimanual conditions, controlled for finger responding rate.

Figure 1. Variability of interresponse intervals (IRI) across bimanual conditions, controlled for finger responding rate

The analyses of tapping errors and tapping ratios were more straight-forward because they involved only asymmetric trials and required no control for the inverse correlation between finger responding rate and IRI variability.

Results

Tapping Variability

Symmetric trials (unison and alternation condition)

For adolescents, there were no main or interaction effects by sex or hand (left, right) on any outcome measure and no main or interaction effects by age across the years from 13 to 18. Variables of sex, age, and hand were therefore combined in all subsequent analyses.

The preliminary ANOVA for reading groups across metronome speed and condition revealed main effects by group, $F(2, 147) = 6.7$, $p < .001$. Dyslexic adolescents tapped with greater variability than did normal or pathological controls students, and there were no significant differences between control groups (see Table 2). There were also main effects by speed, $F(2, 147) = 19.3$, $p < .001$, and condition, $F(1, 148) = 43.6$, $p < .001$, but these reflected primarily the inverse correlation between responding rate and IRI variability. The

informative finding was a Group × Condition interaction, $F(2, 145) = 4.6$, $p < .05$. Dyslexic students differed from control subjects in the alternation but not the unison condition.

Table 2
Mean Variability of Interresponse Intervals as a Function of Condition and Metronome Speed for Adolescents

Condition/ speed	Subjects					
	Dyslexic		Pathological control		Normal control	
	M	*SD*	*M*	*SD*	*M*	*SD*
Unison						
92	0.56	0.63	0.48	0.39	0.50	0.56
120	0.37	0.23	0.32	0.31	0.33	0.25
184	0.39	0.40	0.36	0.31	0.29	0.20
Alternation						
92	1.17	1.16	0.96	0.63	0.95	0.66
120	0.91	1.32	0.68	0.60	0.71	0.68
184	0.76	0.96	0.53	0.44	0.56	0.30
Asymmetric Leading						
92	0.98	0.81	0.52	0.43	0.45	0.27
120	0.81	0.87	0.43	0.39	0.40	0.30
184	1.41	0.79	0.69	0.52	0.79	0.59
Nonleading						
92	1.55	1.16	0.92	0.76	0.85	0.63
120	1.52	1.29	0.63	0.48	0.67	0.64
184	1.83	1.32	1.03	1.02	0.98	0.75

Note. Metronome speed is in beats per minute.

The definitive analysis of condition effects at a constant finger responding rate (unison trials at 92 BPM and alternation trials at 184 BPM) again revealed main effects by group, $F(2, 147) = 7.9$, $p < .001$, and by condition, $F(1, 145) = 18.7$, $p < .001$. Dyslexic adolescents tapped with greater variability than did normal or pathological control students, who did not differ; and all subjects tapped with greater variability in the alternation than in the unison condition. The informative finding was a Group × Condition interaction, $F(2, 145) = 4.7$, $p < .05$. Post hoc analyses indicated that dyslexic subjects performed with greater variability than either control group in the alternation condition, but not in the unison condition.

For adults, preliminary analysis of tapping variability across metronome speed and condition (see Table 3) revealed the expected main effect by speed, $F(2, 79) = 8.3$, $p < .01$, but no main or interaction effects by group or condition. When finger responding rate was controlled, there was a main effect by condition, $F(1, 80) = 5.3$, $p < .05$, but no main effect by group, and no Group × Condition interactions. In contrast to adolescents, dyslexic adults performed both the alternation and unison trials with the same timing precision as did the age-matched control students.

Table 3
Mean Variability of Interresponse Intervals as a Function of Condition and Metronome Speed for Adults

Condition/ speed	Subjects			
	Dyslexic		Normal control	
	M	*SD*	*M*	*SD*
Unison				
92	0.36	0.24	0.31	0.16
120	0.26	0.09	0.25	0.12
184	0.39	0.32	0.36	0.41
Alternation				
92	0.69	0.56	0.60	0.19
120	0.35	0.22	0.40	0.12
184	0.39	0.29	0.36	0.47
Asymmetric				
Leading				
92	0.63	0.48	0.43	0.23
120	0.41	0.43	0.32	0.20
184	0.72	0.56	0.43	0.39
Nonleading				
92	1.05	0.90	0.61	0.39
120	0.79	0.64	0.48	0.39
184	1.11	0.79	0.63	0.58

Note. Metronome speed is in beats per minute.

Asymmetric trials

For adolescents, the analysis across metronome speed and condition (right-hand leading, left-hand leading) revealed main effects by group, $F(2, 147) = 7.1$, $p < .01$, and speed, $F(2, 147) = 7.1$, $p < .01$. Dyslexic students performed with greater variability than did either control group; there were no differences between normal and pathological control students (see Table 2). Despite the inverse correlation between finger responding rate and tapping variability, subjects tapped with greater variability at 184 than at 120 or 92 BPM, with no differences between 120 and 92 BPM (see Table 2). There were no main or interaction effects by asymmetric condition (left-hand leading, right-hand leading).

The critical finding was a Group × Speed interaction, $F(2, 145) = 13.7$, $p < .001$. Dyslexic adolescents performed with greater variability than both control groups at 184 and 120 BPM, but not at 92 BPM. Post hoc analyses indicated that at 184 BPM the effect was significant both in the leading finger (responding 184 times per minute), $F(2, 145) = 6.7$, $p < .01$, and the nonleading finger (responding 92 times per minute), $F(2, 145) = 16.9$, $p < .001$. The fact that reading groups did not differ at a metronome speed of 92 BPM when the leading finger was also responding 92 times per minute suggests that the primary source of difficulty for dyslexic subjects was the rate at which they had to coordinate the timed responses of the two fingers, rather than an inability to move either finger fast enough.

For adults, a similar analysis revealed main effects by group, $F(1, 80) = 10.0$, $p < .001$, and speed, $F(2, 79) = 6.6$, $p < .01$. Dyslexic subjects performed with greater variability than did control subjects, and all of the subjects tapped with greater variability at 92 and 184 BPM than at 120 BPM; differences between 184 and 92 BPM were not significant (see Table 3). There were no Group × Rate interactions.

Comparison of Symmetric and Asymmetric Trials

The comparison across symmetric and asymmetric conditions with finger responding rate controlled yielded similar results. For adolescents, at the slow responding rates (unison trials at 92 BPM, alternation trials at 184 BPM, and nonleading hand of asymmetric trials at 184 BPM), there were main effects by group, $F(2, 147) = 4.7$, $p < .01$, and conditions, $F(2, 147) = 42.9$, $p < .001$. Dyslexic students performed with greater variability than did normal or pathological control students; all of the subjects performed with greater variability in the asymmetric than in the symmetric (unison or alternation) conditions. The informative finding was again the Group × Condition interaction, $F(2, 145) = 13.2$, $p < .001$. Dyslexic students performed with greater variability

than did control students in both the asymmetric and alternation conditions but not in the unison condition (see Figure 1).

At fast responding rates (unison trials at 184 BPM, leading finger of asymmetric trials at 184 BPM) there were also main effects by group, $F(2, 147) = 9.7$, $p < .001$, and by condition, $F(1, 148) = 96.9$, $p < .001$. Dyslexic students performed with greater variability than did pathological or normal control subjects, and all of the subjects performed with greater variability of the leading finger than with either finger in unison trials. The informative finding was a Group × Condition interaction, $F(2, 145) = 5.8$, $p < .01$; dyslexic students differed from control subjects in the asymmetric but not the unison condition.

For adults, at slow responding rates there were the expected main effects by group, $F(1, 80) = 10.4$, $p < .001$, and condition, $F(2, 79) = 7.8$, $p < .001$. Dyslexic adults tapped with greater variability than did control subjects; all of the subjects performed with greater variability in asymmetric than in either the alternation or unison trials, which in adults did not differ. The informative finding was a Group × Condition interaction, $F(2, 79) = 6.3$, $p < .01$. Dyslexic adults differed from control subjects in the asymmetric, but neither in the alternation nor in the unison condition.

At fast rates, there were expected main effects by group, $F(1, 80) = 10.3$, $p < .01$, and condition, $F(1, 80) = 35.7$, $p < .001$, as well as a Group × Condition interaction, $F(1, 79) = 19.0$, $p < .001$. Dyslexic subjects differed from control subjects in asymmetric but not in unison trials (see Figure 1).

Similar comparisons for slow responding rates (alternation, nonleading hand of asymmetric trials) at a metronome speed of 120 BPM (all dyslexic and normal control students combined) revealed main effects by group, $F(1, 180) = 17.3$, $p < .001$, and condition, $F(1, 180) = 49.7$, $p < .001$, as well as Group × Condition interactions, $F(1, 179) = 26.9$, $p < .001$. Dyslexic subjects performed with greater variability than did control subjects; and all subjects performed with greater variability in asymmetric than in alternation trials. However, dyslexic subjects differed from control subjects only in the asymmetric condition. At fast responding rates (unison trials, leading hand of asymmetric trials at 120 BPM), there were expected effects by group, $F(1, 180) = 19.7$, $p < .001$, and condition, $F(1, 180) = 8.7$, $p < .001$, and a Group × Condition interaction, $F(1, 179) = 23.8$, $p < .001$. Dyslexic adults performed with greater variability than did control adults; all subjects tapped with greater variability in asymmetric than in unison trials; and dyslexic subjects differed from control subjects only in asymmetric trials.

Tapping Errors

Adolescents

A repeated measures ANOVA with metronome speed, condition (leading/nonleading hand), and hand (left, right) as repeated measures, revealed main effects by group, $F(2, 147) = 7.3$, $p < .001$, and by speed, $F(2, 147) = 21.0$, $p < .001$. Dyslexic students made more errors than did normal or pathological control subjects, who did not differ. Subjects made more errors at 184 than at 120 or 92 BPM, with no differences between 120 and 92 BPM (see Table 4).

Table 4
Tapping Errors of Subjects on Asymmetric Trials

Hand/ speed	Subjects: Dyslexic *M*	Dyslexic *SD*	Pathological control *M*	Pathological control *SD*	Normal control *M*	Normal control *SD*
Adolescents						
Leading						
92	3.3	0.92	2.2	0.95	2.1	0.63
120	3.5	1.16	2.0	0.88	2.1	0.50
184	7.3	2.68	2.7	1.29	2.5	0.97
Nonleading						
92	3.7	1.83	1.9	0.59	1.9	0.92
120	2.9	0.77	1.7	1.10	1.5	0.56
184	6.3	2.54	3.6	1.35	3.4	1.00
Adults						
Leading						
92	3.9	1.33			1.4	0.68
120	3.2	0.60			1.2	0.42
184	5.6	1.34			1.1	0.60
Nonleading						
92	3.0	0.87			1.9	0.88
120	2.0	0.75			0.7	0.30
184	4.7	2.52			2.1	0.84

Note. Speed is measured in beats per minute.

The informative finding was a Group × Speed interaction, $F(2, 145) = 2.8$, $p < .05$. Dyslexic subjects made more errors than control students, but the effect was significant only at 184 BPM (see Figure 2). A post hoc analysis of asymmetric trials at 184 BPM indicated that dyslexic subjects differed from control subjects in both the leading finger (M dyslexics = 7.3, M both groups = 3.1) and the nonleading finger (M dyslexics = 6.3, M controls = 3.5). Because the nonleading finger was expected to respond at only one half the rate of the leading finger, these findings again suggest that the primary difficulty of dyslexic subjects was their inability to coordinate asymmetric timing responses of the two fingers at a fast rate.

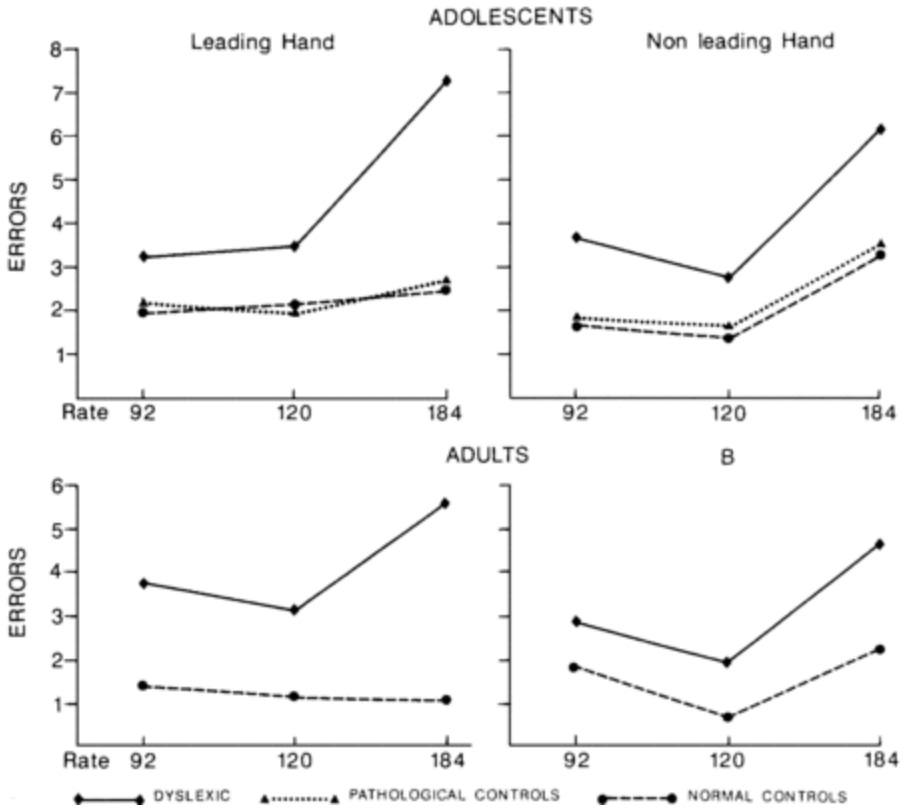

Figure 2. Tapping errors during asymmetric bimanual performance as a function of metronome speed

The measure of tapping errors also revealed a main effect by hand, $F(1, 148) = 5.5$, $p < .05$, and a Speed × Hand interaction, $F(2, 145) = 5.7$, $p < .01$. The left hand made slightly more errors than the right, but the laterality effect was significant only at 184 BPM, and there were no Group × Hand interactions.

A parallel analysis in adults revealed main effects by group, $F(1, 80) = 9.7$, $p < .002$, and speed, $F(2, 79) = 4.6$, $p < .02$, as well as by Group × Rate, $F(2, 79) = 8.3$, $p < .01$, and Group × Condition interactions, $F(1, 80) = 5.6$, $p < .05$. Dyslexic adults made more tapping errors than did control adults (see Table 4). Subjects made more errors at 184 BPM and 92 BPM than at 120 BPM, with no differences between 184 and 92 BPM. However, the differences between reading groups were significant only at 184 BPM. A post hoc analyses at 184 BPM indicated that in dyslexic adults, tapping errors differed from those of control subjects in the leading finger (M dyslexic = 4.3, M normal = 1.2) but not in the nonleading finger (M dyslexic = 3.2, M normal = 1.7). There were no Group × Condition × Rate interactions.

Tapping Ratios

Tapping ratios were introduced to determine whether the impaired interlimb coordination in asymmetric trials was due to overresponding of the leading or nonleading finger. As a first step, we computed the number of subjects in each reading group who performed within or outside the tolerance range of tapping ratios (< 1.8–>2.2), and we classified subjects into subgroups who exceeded the range in at least one of the six asymmetric trials and those who performed all six asymmetric trials within the tolerance range. In all, 28 dyslexic adolescent (56%), 2 normal control (4%), and 3 pathological control (6%) subjects failed to meet the criterion, $\chi 2$ (2, N = 150) = 48.5, $p < .001$. Similarly, 19 dyslexic adult (46%) and 2 normal control (5%) subjects tapped in deviant ratios, $\chi 2$ (1, N = 82) = 23.6, $p < .001$. Thus, one half of the dyslexic and very few control subjects failed to maintain the correct ratio between leading and nonleading finger. Furthermore, more than 90% of the deviant ratios were below the tolerance range of 1.8–2.2, and most of the abnormal ratios occurred at metronome speeds of 184 BPM (74%), whereas very few occurred at a metronome speed of 92 BPM (3%). In other words, most of the abnormal ratios were the result of a tendency by the nonleading finger

to overrespond relative to the leading finger; this tendency was expressed at fast, but not at slow, responding rates.

Prevalence of Impaired Interlimb Coordination

As a more quantitative test of the distribution of interlimb-coordination deficits among dyslexic subjects, we divided the samples into subgroups that did and did not exceed the mean frequency of tapping errors of normal control subjects in asymmetric trials at 184 BPM by 1 SD or more. Table 5 indicates that about one half of all of the dyslexic adolescents and adults, but very few pathological or normal control subjects, exceeded the threshold.

Table 5
Distribution of Tapping Errors Exceeding Threshold Criterion

	Subjects		
Hand/*SD*	Dyslexic	Pathological	Normal
Adolescents			
Leading			
>1	27	4	5
<1	23	46	45
		$\chi^2(2, N = 150) = 37.1$*	
Nonleading			
>1	26	6	4
<1	24	44	46
		$\chi^2(2, N = 150) = 32.5$*	
Adults			
Leading			
>1	17	—	2
<1	24	—	39
		$\chi^2(1, N = 82) = 15.4$*	
Nonleading			
>1	19	—	2
<1	22	—	39
		$\chi^2(1, N = 82) = 18.5$*	

* $p < .001$.

A comparison of subgroups of dyslexics with and without interlimb-coordination deficits (as defined earlier) on the various standardized measures of academic achievement and on WAIS–R Verbal–Performance discrepancies revealed no significant differences.

Left-handedness is sometimes taken as an outward manifestation of abnormal variations in hemispheric specialization in dyslexic subjects (Geschwind & Galaburda, 1985). Although performance asymmetries in this study were marginal and there were no Group × Hand interactions, differences in hand-use preference might have accounted for some of the group differences in bimanual coordination. The incidence of non right-handedness was indeed greater among dyslexic subjects (adolescents 21%, adults 22%) than among normally reading control subjects (adolescents 16%, adults 11%), but in this sample, the differences were not significant (by a chi-square analysis); and within dyslexic samples, hand-use preference was not correlated with any measure of interlimb coordination.

Discussion

The main findings of the study were as follows:

1. Timing precision for bimanual coordination differed significantly between dyslexic adolescents or adults and agematched control subjects.
2. The motor deficits were evident only on tasks requiring the integration of asynchronous responses between the fingers (bimanual alternation or asymmetric 2:1 patterns).
3. The motor deficit was rate dependent and more pronounced in asymmetric than in alternation trials.
4. Dyslexic women did not differ significantly from dyslexic men.

5. Only about one half of the dyslexic subjects demonstrated the bimanual coordination deficits, and within this subsample, impaired bimanual coordination was not correlated with reading impairment or IQ Verbal–Performance discrepancies.

We conclude that bimanual coordination tasks requiring the rapid and continuous integration of asynchronous or asynchronous and asymmetric finger movements identify a nonlinguistic, noncognitive, neuropsychological deficit in a substantial subgroup of dyslexic adolescents and adults. The motor deficit may be an outward manifestation of a developmentally stable and physiologically plausible deficit that impinges on the timing control of speech and language processes in a subgroup of dyslexic subjects. However, the specific pathways by which such impaired temporal resolution contributes to reading impairment remain to be investigated.

Three limitations of the study should be made explicit. Neither young dyslexic nor reading-age-matched normal control children were examined, and it is possible that the bimanual coordination deficits of older dyslexic students were a consequence, rather than a cause, of chronic reading failure. However, our earlier studies indicated that even 8–11-year-old reading-impaired children differ from adequate readers when they perform the bimanual alternation tasks, whereas they perform like average readers on unimanual tapping tasks; none of the young elementary-school children were able to perform the asymmetric bimanual tasks that were most informative in the present study (Badian & Wolff, 1977). Subsequent studies of 11–13-year-old students indicated that a substantial proportion of normal readers as well as dyslexic subjects still had difficulty performing the asymmetric bimanual tasks (Klicpera, Wolff, & Drake, 1981). Therefore, it is unlikely that a reading-age-matched control group (i.e., 10–11-year-old normal readers) would have been unable to perform the asymmetric bimanual tasks.

No individual standardized test scores were obtained on normal adolescent control students, although the control sample was selected to match dyslexic subjects. The possibility therefore cannot be excluded that differences in psychometric intelligence might have contributed to group differences in motor performance. However, pathological control subjects were case matched with dyslexic subjects for psychometric intelligence, and they differed significantly from the latter in bimanual coordination, but did not differ from normal control subjects, with whom they were group matched. Furthermore, the distribution of IQ scores in dyslexic subjects from 95 to 135 were not correlated with variations of interlimb coordination. In adults, the potential confounding effect of IQ on motor performance can, however, not be excluded.

Finally, tapping performance was only analyzed for those segments of a trial after the metronome was turned off (continuation condition). Cognitive defects in the ability to construct an internal representation of the required tempo might therefore have accounted for poor motor performance in dyslexic subjects. In principle, however, such a cognitive defect should have interfered with bimanual conditions at slow as well as at fast rates, but dyslexic subjects differed from control subjects only at fast rates and only in the asynchronous conditions. Our previous normative studies (Wolff & Hurwitz, 1976) had shown that all subjects perform with greater precision when the metronome is on than when it is off, but that reading group differences are as significant in the synchronization as in the continuation condition. On the other hand, if one assumes that such cognitive deficits pertain to a difficulty in representing the sequence of movements for asymmetric trials, it is debatable whether such difficulties have a basis in cognition or motor coordination because the serial order of many complex patterns of motor coordination is probably not controlled by cognitive maps but by the inherent dynamics that coordinate large numbers of individual movements into low dimensional ensembles (Bernstein, 1967).

Our results neither support nor disconfirm the hypothesis that temporal resolution deficits in dyslexia are the outward manifestations of a left-hemisphere dysfunction. Temporal resolution deficits in the motor action of brain-damaged adults are associated far more often with localized left- than right-hemisphere lesions (Hecaen, 1962; Kimura & Archibald, 1974), but the site of a brain lesion rarely identifies the neurological structure that controls the impaired function (Caplan, 1981; Jackson, 1878). Furthermore, the timing control of motor action has input from neural processes that are widely distributed throughout the primary and supplementary motor

area, cerebellum, extrapyramidal system, and midbrain structures (Anderson, 1981; Edelman & Mountcastle, 1978; Llinas & Simpson, 1981) and can probably not be localized to the left hemisphere alone, if it can in fact be localized in any specific neural structure (Kelso & Tuller, 1984).

An alternative model of developmental dyslexia is based on the premise that many complex psychological functions, including the translation of graphic symbols into a phonemic code, apparently depend on component processes from both cerebral hemispheres and that at least some subtypes of dyslexia may be due to impaired interhemispheric communication (Gazzaniga, 1973; Gladstone & Best, 1985). Partial support for the hypothesis comes from brain imaging and electrophysiological coherence studies indicating that many language-based functions activate topographically discrete regions in both cerebral hemispheres (Larsen, Shinkoj, & Lassen, 1978) and from corollary clinical evidence that electrophysiological and metabolic activity and interhemispheric coherence differ significantly between dyslexic and control children (Duffy, Denckla, Bartels, Sandin, & Kiessling, 1980; Leisman & Ashkenazi, 1980). However, on the basis of such neurophysiological data, it is difficult to specify the functional deficits in dyslexia.

Behavioral evidence that might support this hypothesis is both sparse and contradictory. For example, discrete trial reaction-time paradigms have failed to demonstrate any differences between dyslexic children and average readers (Broman, Rudel, Helfgott, & Krieger, 1985; Vellutino, Scanlon, & Bentley, 1983). Yet, an adequate test of the hypothesis may depend on measuring the continuous and rapid exchange of information between the hemispheres (Jeeves, Silver, & Milne, 1988). For example, the study of adult neurological patients with complete surgical callosectomy has demonstrated that they are unable to learn any novel bimanual motor tasks requiring the continuous coordination of asynchronous or asymmetrical movements between the right and left hands (Kreuter, Kinsbourne, & Trevarthen, 1972; Preilowski, 1972; Zaidel & Sperry, 1977) and that the callosectomized patient's inability to learn such tasks is due in large part to the subject's tendency to move the two hands synchronously and symmetrically when the task requires the two hands to move asymmetrically or asynchronously (see also Tuller & Kelso, 1989). Young normal children exhibit the same tendency on similar bimanual tasks, but unlike callosectomized patients they do learn after extensive training (Fagard, 1987; Fagard, Morioka, & Wolff, 1985; Jeeves et al., 1988).

Young normal children also exhibit unintended mirror movements when they are asked to tap simple unimanual finger patterns (Wolff & Hurwitz, 1976) or to perform simple motor maneuvers (Connolly & Stratton, 1968; Wolff, Gunnoe, & Cohen, 1983). The frequency and amplitude of such mirror movements diminish predictably with chronological age (Wolff et al., 1983), but electromyographic studies have indicated that the bilateral activation of homotopic muscles when only one side is needed for the intended act remains a stable characteristic of the motor system throughout adult life whether or not it is expressed as observable movements (Cernacek, 1961; Green, 1967; Hopf, Schlegel, & Lowiztsch, 1974). The failure to suppress unintended mirror movements may therefore directly interfere with the performance of bimanual tasks that require asynchronous or asymmetric responses from the two sides of the body.

Cross-callosal motor pathways are generally assumed to mediate such bilateral activation, but it remains a matter of controversy whether the corpus callosum mediates excitatory or inhibitory impulses or both (Dennis, 1976; Woods & Teuber, 1978). Either an excess of bilateral excitations or the failure to suppress unintended mirror movements should in principle interfere with bimanual performance in tasks that require the rapid and continuous integration of timed responses between the two sides of the body. Whether similar deficiencies of interhemispheric communication also contribute to the reading impairment and language impairment of dyslexic subjects cannot be deduced from the study of bimanual coordination alone.

The exact functions of the corpus callosum and its role in the organization of human behavior remain a matter of conjecture. Adults with complete callosectomies show remarkably few behavioral handicaps (Selnes, 1974) other than the inability to learn novel bimanual skills. For example, surgical callosectomy has little effect on the reading fluency of adults, and many high functioning children with agenesis of the corpus callosum but no other associated brain anomalies learn to read. On the basis of such clinical evidence, one might conclude that

impaired efficiency of interhemispheric communication is not of etiologic significance in developmental dyslexia.

However, the effect of total callosectomy in adults, or the absence of the corpus callosum throughout development, on the organization of behavior probably differs qualitatively from the effect of a grossly intact corpus callosum that transmits either (a) degraded information efficiently between the hemispheres, (b) intact information either too slowly or to the "wrong address" on the contralateral side (Bianki & Schramm, 1985), or (c) redundant or conflicting information (e.g., failure of suppression or inhibition; Dennis, 1976). Thus, variations in the dynamics of interhemispheric interaction, rather than neuroanatomically specified structural deficits, may be the factor that accounts for the temporal resolution deficits of dyslexic subjects during interlimb coordination, as well as for deficits in other time-dependent functions distributed across both hemispheres that are directly involved in learning to read.

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