Impaired Performance of Children with Dyslexia on a Range of Cerebellar Tasks

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It is now thought that the cerebellum is involved in the acquisition of "language dexterity" in addition to its established role in motor skill acquisition and execution. Mild cerebellar impairment, therefore, provides a possible explanation of a range of problems shown by children with dyslexia. The authors have established suggestive evidence in support of this hypothesis in tests of balance and of time estimation. In a further test of the hypothesis, a battery of clinical tests for cerebellar impairment, including tests of muscle tone and of coordination, was administered to matched groups of children with dyslexia and control children aged 10, 14, and 18 years (55 subjects in all). The children with dyslexia showed highly significant impairments on all the cerebellar tests, and significant impairment compared even with reading age controls on 11 of the 14 tasks. Deficits on the majority of tests were among the largest found in our research program. The findings, therefore, provide further intriguing evidence of cerebellar impairment in dyslexia. We speculate that the well-established phonological deficits in dyslexia may arise initially from inefficient articulatory control attributable to cerebellar impairment.

Developmental dyslexia is traditionally defined as *a disorder in children who, despite conventional classroom experience, fail to attain the language skills of reading, writing and spelling commensurate with their intellectual abilities* (World Federation of Neurology

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1968). A recent redefinition as *a specific language based disorder of constitutional origin, characterized by difficulties in single word decoding, usually reflecting insufficient phonological processing abilities* (The Orton Dyslexia Society 1994) reflects a major achievement of dyslexia research—the identification and analysis of a phonological deficit (Bradley and Bryant 1983; Shankweiler et al. 1995; Stanovich 1988; Vellutino 1979) which became the consensus view of many dyslexia researchers. Background factors including its high population incidence, the high financial stakes involved in remedying the reading difficulties, and the high media profile for dyslexia research, have resulted in continuous publicity for dyslexia research and have inspired a wide range of research studies aimed at better understanding, diagnosis, or remediation of dyslexia.

Interestingly, it now appears that the phonological deficit hypothesis, though undoubtedly an accurate analysis of a major source of difficulties, is by no means a complete explanation of the range of difficulties encountered by children with dyslexia (Nicolson and Fawcett 1994a, 1995). At the time that the phonological deficit hypothesis was being established, there was already strong evidence that a range of "soft neurological signs" were implicated in the motor skill deficits associated with dyslexia. Denckla (1985) summarizes the early research, "Dr. Rudel and I have come to the conclusion that the most parsimonious explanation [of coordination difficulties] is as follows: the part of the 'motor analyzer' that is dependent on the left hemisphere and has been found to be important for timed, sequential movements is deficient in the first decade of life in this group of children whom we call dyslexic." A major factor in the full acceptance of the phonological account was that Denckla subsequently changed her view, arguing that soft neurological signs are attributable to ADHD rather than dyslexia, and therefore that the high incidence of soft neurological signs in groups of dyslexic children arises from the comorbidity of ADHD with dyslexia (Denckla et al. 1985). However, since that time cognitive research from different perspectives has now identified a range of non-phonological deficits, including problems in visual processing (Lovegrove et al. 1990; Stein 1989), reduced information processing speed (Denckla and Rudel 1976; Wolf 1991; Nicolson and Fawcett 1994b), and impairments in "automatic" balance (Nicolson and Fawcett 1990; Yap and van der Leij 1994). Further evidence of subtle deficits in motor skill has also been established. Many practitioners have reported motor skill difficulties, especially in early life (Augur 1985). A large scale cohort

study of 12,905 children at age 10 years reported in Haslum (1989) identified one-leg balance ability, walking backwards ability, match-sorting speed, and performance on a graphaesthetic test (reporting what shape was traced on the skin when blindfolded) as most highly correlated with dyslexia out of a large battery of tests. Studies of fine motor skills (Rudel 1985) identified deficits in toe tapping and in successive opposition of fingers and thumbs, and a series of studies by Wolff and his colleagues (e.g., 1990) has shown persistent problems in tapping rhythm for children with dyslexia, specifically when the hands were required to tap asynchronously. Fawcett and Nicolson (1995a) have also identified a range of problems in motor skill.

This pattern of widespread difficulties on skills is consistent with the automatization deficit hypothesis (Nicolson and Fawcett 1990) that children with dyslexia will suffer problems in fluency for any skill that should become automatic through extensive practice. However, the hypothesis is perhaps best seen as a parsimonious description of the pattern of difficulties, rather than as a causal explanation, since the authors were not able to specify what was causing the supposed difficulty in skill automatization.

Problems in execution of motor skill point to the cerebellum, which has traditionally been considered as a motor area (Holmes 1917, 1939; Eccles et al. 1967; Stein and Glickstein 1992). There is also extensive evidence that the cerebellum is centrally involved in the acquisition of motor skill, by way of its rich connections to motor cortex, to the skeleto-muscular system, and to sensory cortex, with an influential model of its role (Marr 1969; Albus 1971; Ito 1984, 1990) being that, following a motor movement, the cerebellum receives signals that indicate mismatch between plan and execution by way of the climbing fibers from the inferior olive, and these error signals allow the cerebellum to tune the motor plan timing and execution. A cerebellar inactivation study in rabbits (Krupa, Thompson, and Thompson 1993) has provided direct evidence that the cerebellum is centrally involved in initial skill acquisition. A clear demonstration of the role of the cerebellum in human motor skill acquisition was provided by a recent PET study (Jenkins et al. 1994) that revealed cerebellar activation associated both with new learning and with automatic sequential movement, but most extensively in new learning—". . . the cerebellum is involved in the process by which motor tasks become automatic" (p. 3775). However, despite the early work of Levinson (e.g., Frank and Levinson 1973; Levinson 1988), and despite the es-

tablished evidence for difficulties in the "motor analyzer" (Denckla 1985) the cerebellum has been discounted as a causal factor in dyslexia owing to its supposed lack of involvement in language.

Recent evidence, however, suggests that the cerebellum may indeed be involved significantly in language development. As Leiner et al. (1989) note, the human cerebellum (in particular, the lateral cerebellar hemispheres and ventrolateral cerebellar dentate nucleus) has evolved enormously, becoming linked not only with the frontal motor areas, but also some areas further forward in the frontal cortex, including Broca's language area. Leiner et al. (1989, 1993) conclude that the cerebellum is therefore critical for the acquisition of "language dexterity." In effect, then, they propose that the cerebellum is critically involved in the automatization of any skill, whether motor or cognitive. Recent tomographic and magnetic resonance studies (Decety et al. 1990; Akshoomoff et al. 1992; Paulesu et al. 1993) and recent studies of cerebellar patients (Fiez et al. 1992; Silveri et al. 1994) have provided further support for the involvement of the cerebellum in cognitive activities. The involvement of the cerebellum in cognition is currently one of the "hottest" areas of cognitive neuroscience research, with a plethora of possible roles posited, including timing (Ivry and Keele 1989; Thach 1996); attentional shifting (Courchesne et al. 1994); and even sensory acquisition and discrimination (Gao et al. 1996).

The close match of the pattern of difficulties likely to be shown following mild early cerebellar damage with that independently established for dyslexia, make mild cerebellar impairment a prime candidate for the underlying cause of dyslexia. Ivry and Keele (1989) established that patients with acute cerebellar damage had specific deficits in time estimation but not loudness estimation. In a stringent test of the dyslexic cerebellar impairment hypothesis, Nicolson et al. (1995) replicated the study using matched children with dyslexia and control children. The predicted dissociation was obtained, with the children with dyslexia showing a significant deficit on temporal estimation (even when compared with reading age controls) but no deficit whatsoever on loudness estimation. The results, therefore, provided strong support for the cerebellar impairment hypothesis, especially since they were not predicted by any other current hypothesis for the cause of dyslexia.

However, the precise role of the cerebellum in cognitive skill is still not fully established (see Barinaga 1996 for an accessible overview of some of the issues). Moreover, the time estimation

task is by no means a direct index of cerebellar function, and it is hard to envisage that difficulties in estimating the duration of one-second tones would lead to reading difficulties! If there is indeed a cerebellar impairment in dyslexia, then children with dyslexia should show marked deficits on the traditional signs of cerebellar dysfunction. Clinical evidence of the range of deficits evident following gross damage to the cerebellum, has been described in detail in classic texts by Holmes (1917, 1939) and Dow and Moruzzi (1958). Classic symptoms of cerebellar dysfunction are dystonia (problems with muscle tone) and ataxia (disturbance in posture, gait, or movements of the extremities). Given that other theories of dyslexia do not predict difficulty on these tasks, presentation of the standard clinical tests for cerebellar dysfunction provides an opportunity for rigorous testing of the cerebellar impairment hypothesis for dyslexia.

EMPIRICAL STUDIES

The focus of the research reported here involved replication of the clinical cerebellar tests described in Dow and Moruzzi (1958), using matched groups of children with dyslexia and control children.

DESIGN

Although it is valuable to identify whether children with dyslexia perform significantly worse than their same-age controls, one of the key discriminants between theories is a test of performance of children with dyslexia against reading age controls, since a significant impairment compared with reading age controls is indicative of developmental disorder rather than just a developmental lag (Bryant and Goswami 1986). Since the specific nature of children with dyslexia's deficits may also change with age, it is important to examine the effects of age separately. These considerations suggest an experiment with at least six groups of subjects: two groups of children with dyslexia of different mean ages; two groups of normally achieving children matched to the children with dyslexia on chronological age; and two groups of normally achieving children matched to the children with dyslexia on reading age. This is a design we have used for some years (see Nicolson and Fawcett 1994b), and as in previous studies we were able to use a group of controls both as chronological controls for one group of children with dyslexia and reading age controls for an older group of children with dyslexia.

SUBJECTS

Subjects with dyslexia satisfied both of the two standard exclusionary criteria for dyslexia, namely (1) normal or above normal IQ (operationalized as IQ of 90 or more on the full scale WISC-R [Wechsler 1976]), without known primary emotional, behavioral, or socioeconomic problems, whose reading age (RA) was at least 18 months behind their chronological age (CA), and (2) significantly lower actual reading age than that predicted on the basis of their IQ (operationalized as a discrepancy of at least 1.5 standard deviations between actual and predicted reading age). None of the subjects showed evidence of ADHD as measured on the DSM-IIIR scales (American Psychiatric Association 1987)[1]. Three groups of children with dyslexia participated, together with three groups of normally achieving children matched for age and IQ. The children had been in our research panel for some years, and at the time of testing had mean ages of 18, 14, and 10 years. This gave six groups with around 10 children in each group, D18, D14, and D10; and C18, C14, and C10 for the three age groups of children with dyslexia and control children respectively. This three-age-group design allows performance to be compared with children of the same age (D18 vs. C18, D14 vs. C14; D10 vs. C10), children of around the same reading age (D18 vs. C14; D14 vs. C10) and children of around half the age (D18 vs. C10). Further psychometric details are given in table I.

The children with dyslexia were initially located through the local Dyslexia Institute or the local branch of the British Dyslexia Association. In view of the potential danger of implicit selection bias, it is important to note that, other than checking that the children met our criteria for "dyslexia pure," and that they were willing to undertake testing on a long-term basis, no other screening or selection whatsoever was undertaken. It should be noted, though, that the subjects had already participated in a range of experiments, and we had established that the subjects with dyslexia showed difficulties in phonological skill, motor skill, balance, and temporal estimation (Fawcett and Nicolson 1995a, 1995b; Nicolson and Fawcett 1994a; Nicolson, Fawcett, and Dean 1995). Subjects were paid around $5 per hour and participated with fully informed consent.

[1]The DSM-IIIR assessment to ADHD involves 14 simple yes/no questions, with a "yes" on at least 8 being the minimal criterion for diagnosis of weak ADHD. None of the dyslexic or control children showed evidence of ADHD (for the children with dyslexia the range was 0-6, with mean 1.2, and for the controls the range was 0-5 with mean 0.7). The difference between groups was negligible ($F < 1$).

TABLE I. PSYCHOMETRIC DATA FOR THE SIX GROUPS OF SUBJECTS.

The groups of children with dyslexia are labelled D18, D14 and D10, with the suffix indicating the mean age. Similarly the groups of normally achieving children are labelled C18, C14 and C10. The mean value for each group is presented first, with the range of values bracketed below.

	D10	C10	D14	C14	D18	C18
N	12	8	9	11	8	7
IQ (WISC-R)	111.6 [96-133]	114.1 [101-124]	110.8 [105-128]	110.9 [96-129]	102.9 [88-126]	106.0 [92-130]
Chronological Age	10.7 [10.2-11.0]	10.9 [10.2-11.5]	14.4 [12.8-15:9]	14.6 [13.4-15.2]	18.6 [17.4-19.6]	18.9 [18.5-19.3]
Reading Age	8.1 [7-9.9]	11.2 [11.0-11.9]	10.9 [8.0-12.9]	14.2 [13.3-14.8]	13.0 [11.2- 14.6]	17+† [16.0-17+]

†17+ represents ceiling on the Wechsler Objective Reading Dimensions test of single word reading used. All C18 subjects bar 1 were reading at this level. The WORD test lacks sensitivity near ceiling, with only 1 point difference between the 16.0 and 17.0 levels.

EXPERIMENTAL TASKS

The tests in the Dow and Moruzzi (1958) battery may be divided into three types: first, the ability to maintain posture and muscle tone while standing and in response to active displacement of station; second, a series of tests for hypotonia of the upper limbs in both a standing and sitting position, in response to active or passive displacement of the limbs; and finally, a series of tests of the ability to initiate and maintain a complex voluntary movement.

The tests included;

(1) *Maintenance of posture*

- Balance time—The length of time during which subjects could stand, blindfolded, with feet together and their arms outstretched forward before the first wobble.
- Postural stability—Subjects were asked to stand upright, looking straight ahead, arms by their sides, and were then blindfolded by the experimenter. The experimenter then stood behind the child, and explained that she was going to push him/her gently in the back, and that he/she should try to stand still. The experimenter then pushed gently in the small of the child's back with her index finger at a 2 Kg pressure (the experimenter "calibrated" herself prior to the session by practicing pushing at 2 Kg on a kit-

chen scales). Pressure was applied for 1.5 seconds and then released. The degree of sway was assessed and recorded for each trial (on a scale of 0 for good performance, 1 for a small movement and 2 for stepping forward or overbalancing). The test was performed six times, three times with the children's arms at their side, followed by three pushes with their arms straight out in front, which is slightly more difficult. Children with signs of cerebellar deficit would be predicted to generate a high score. The maximum score for this test was 12. Scoring was checked by video taping a sample of children and getting independent ratings by a trained observer who was unaware of the subjects' group, with an inter-rates reliability of 0.94.

(2) *Hypotonia (reduced muscle tone)*

- Static tremor—Subjects was blindfolded and held a pen at arm's length. Subjects held the pen for one minute with either hand. Scoring was tedious and involved measuring the total distance traveled by the felt tip pen, and then subtracting the drift—the straight line distance between the start and end points.
- Arm displacement—Subjects were blindfolded and asked to stand with their feet together, with their arms held out in front of their body. The experimenter tapped each hand gently in turn, for a series of three taps to each hand. Subjects were scored for the amount of movement in the limb, on a three point scale from 0 to 2, generating a maximum score of 12 for the 6 taps.
- Weight time—Subjects were again blindfolded and asked to stand with their feet together, with their arms held out to each side of their body, holding the neck of a filled bottle. They were asked to hold their arms rigid, actively resisting the weight of the bottles. Their performance was timed and the score noted was the time (seconds) until the arms fell by at least 20.
- Hand declination—Subjects were asked to sit down, with elbows resting on the arms of the chair and forearms held up vertically. The experimenter rolled up the child's sleeves and removed any watch, bracelet, etc., and then took hold of each forearm

and held it vertically so that the child's hands were about level with his/her shoulders. The subject was asked to let his/her hands "flop" as much as possible, like a puppet or a rag doll. The experimenter shook both hands slightly to make sure that they were limp. Then a protractor was used to measure the angle between the forearm and the top of the hand—the angle through which the hand drooped. The Dow and Moruzzi test is for a difference in angle between the hands, indicating abnormality in one of the cerebellar hemispheres.

- Arm shake—Subjects were asked to sit down, with their elbows resting on the chair arm and hands dangling loosely, as in the previous task. The experimenter grasped each hand at the wrist and shook it lightly from side to side. Degree of movement was assessed on a scale from 1 (little movement) to 3 (large, floppy movement). Mean score for both hands therefore ranged between 1 and 3.
- Muscle tone—Subjects again adopted the same position, and this time the experimenter told them that she was going to push gently against the child's muscles, and the child's task would be to try and resist. The experimenter pushed against the resistance of the right and left arm, and finally both arms together. Each response was scored for the ability to resist the experimenter's push, on a scale from 0 to 2, generating a maximum score of 6.
- Braking distance—Subjects again sat on a chair with their arms bent and with their elbows resting on the chair arm. The subject and experimenter then grasped opposite ends of a pencil, and the experimenter pulled at a constant force, then suddenly let go of the pencil. The score noted was the distance in inches travelled back by the subject's elbow before the backward movement was arrested. Data reported are the average for both arms.

(3) *Complex movements*

- Past pointing—A bull's-eye target printed onto acetate was fastened to a wall at the eye level of the subject, who was then shown how to point repeatedly to the bull's-eye, using a marker pen so that a record of performance was maintained. After a practice, the experimenter put a blindfold on the child,

and asked him or her to perform 10 trials. The experimenter ensured that the child maintained a constant position in relation to the wall, and withdrew his or her hand after each trial. A score was generated for each annulus of the target, ranging from 0 for falling outside the target to 10 for the bull's-eye. The score was cumulated, with a maximum of 100 points.

- Finger-to-finger pointing—Subjects were blindfolded and were instructed to bring their index fingers rapidly together to try to touch in front of their body. After a short practice, 10 trials were conducted, with a paper target fixed to the index finger of the nonpreferred hand. Marks given for the accuracy of pointing, with a maximum of 10 for accurate performance hitting the finger. Maximum marks for this test were 100. Not all subjects undertook this test.
- Adiadochokinesis—This involved the supination and pronation of both hands on to the knees. Following an initial practice, this test was paced by a computer generated tone, with four speeds (5, 10, 20, and 30 movements per minute). A score from 0 to 5 was generated, based on the child's ability to keep pace with the speed of the tap, while maintaining a consistent pattern of performance. A score of 4 indicates that the child has successfully completed the first three levels, and attempted the fastest level. A score of 5 indicates completion of all four levels of the task, with successful performance at all speeds.
- Toe tap speed—After an initial practice subjects were asked to tap their foot as fast as they could on a tin lid. The sounds were recorded on an Apple Macintosh computer, and the speed of tapping assessed accurately using standard waveform analysis software. The score was the time taken to execute 10 taps.
- Finger and thumb—Subjects placed the index finger and thumb of one hand onto the index finger and thumb of the other hand. Keeping the top thumb and finger together, they were shown how to separate the lower finger and thumb, and turn one hand clockwise and the other counterclockwise, so that the finger and thumb touched again. This sequence of movements was repeated and practiced until the

subject was able to complete the movement fluently 5 times. The children were then told to perform the successive opposition ten times, as fast as possible. The score noted was the time taken.

Each child was tested individually in a quiet room by the first author. Reassurance and feedback was given throughout, but no comparative comments were made on the quality of each child's performance. Testing was completed in two sessions, each taking around one hour overall. The tests were based directly upon those described by Dow and Moruzzi. These tests are clinically based and are somewhat dependent on clinical judgment. Consequently, considerable care was taken to adapt the tests for experimental use, and wherever possible, equipment was designed to facilitate fully objective procedures for each test. The only tests defying full objectivity were muscle tone, braking distance, arm displacement, and limb shake. On the other hand, the remaining tests were explicitly designed for objective interpretation. Toe tap was entirely computer-based, and finger to finger opposition and adiadochokinesis were also computer-aided. Balance time, weight time, and hand declination are entirely objective. Tremor, finger to finger, and past pointing were recorded on card, and scored by students blind to the subjects' group.

RESULTS

QUANTITATIVE GROUP DATA

The mean and standard deviations for the battery of tests are collated in table II. Two factor analyses of variance were undertaken individually on the data for each test, with the factors being chronological age (10, 14, and 18 years) and dyslexia (children with dyslexia vs. control). The results of the inferential tests are collated in table III. It may be seen that the performance of the children with dyslexia was significantly worse than that of the chronological age controls on all of the 14 tasks.

A further set of analyses of variance was undertaken comparing performance with that of reading age controls. In this case the factors were reading age (10 vs. 14) and dyslexia (children with dyslexia vs. control). The results are collated in table IV. It may be seen that the performance of the children with dyslexia was significantly worse on 11 out of the 14 tests, with near-significant impairment on tremor and muscle tone, and with equivalent performance only on finger-to-finger pointing.

Table II. Summary of Mean Scores for each Group on each Test (standard deviations are given in parentheses). Times are given in seconds.

	D10	C10	D14	C14	D18	C18
Balance time (s)	8.49 (4.43)	18.1 (8.33)	14.8 (11.0)	46.1 (15.0)	12.01 (5.67)	44.6 (17.4)
Postural stability	9.00 (3.13)	1.13 (2.23)	7.50 (2.12)	1.09 (2.02)	7.50 (3.93)	2.79 (3.05)
Static tremor (cm)	5.62 (3.91)	0.58 (0.57)	2.23 (1.98)	0.25 (0.50)	0.25 (0.25)	0.13 (0.19)
Arm displacement	3.83 (0.58)	0.50 (0.93)	3.11 (1.05)	0.36 (0.81)	3.00 (1.07)	0.29 (0.76)
Weight time (s)	12.3 (6.91)	25.1 (10.2)	22.0 (15.0)	40.4 (12.6)	29.7 (12.0)	68.7 (36.1)
Hand declination angle difference (°)	9.17 (4.69)	0.63 (1.77)	14.4 (11.3)	2.27 (4.10)	8.75 (3.54)	2.14 (3.93)
Limb shake (1-3)	2.42 (0.56)	1.13 (0.35)	2.33 (0.58)	1.18 (0.40)	1.88 (0.83)	1.14 (0.38)
Muscle tone (0-6)	3.92 (2.71)	0.38 (0.74)	1.78 (1.99)	0.82 (1.40)	1.50 (3.27)	0.00 (0.10)
Braking distance (ins)	3.89 (1.83)	1.44 (1.64)	3.33 (1.64)	1.23 (0.81)	1.56 (0.68)	1.04 (0.64)
Past pointing (max. 100)	51.3 (25.9)	62.9 (15.6)	43.2 (19.6)	60.2 (13.2)	53.5 (11.84)	64.0 (12.4)
Finger to finger pointing (max. 100)	80.2 (8.02)	90.4 (9.4)	88.8 (2.95)	88.4 (4.28)	86.2 (8.6)	92.7 (4.89)
Adiadochokinesis (0-5)	1.42 (1.00)	3.13 (0.99)	2.11 (1.69)	4.27 (0.47)	2.13 (2.03)	4.14 (0.69)
Toe tapping (s)	2.82 (0.71)	2.04 (0.34)	2.58 (0.62)	1.76 (0.26)	2.67 (1.13)	1.63 (0.22)
Finger to thumb (s)	17.6 (12.3)	9.27 (2.96)	20.1 (17.4)	6.46 (1.41)	15.9 (9.47)	7.02 (1.16)

Table III. Inferential statistics for Chronological Age and Dyslexia Groups included were D10, C10; D14, C14; and D18, C18.

Task	Dyslexia	Age	Interaction
Balance time	$F(1,50) = 67.6, p < .0001$	$F(2,50) = 14.0, p < .0001$	$F(2,50) = 6.4, p < .01$
Postural stability	$F(1,50) = 71.2, p < .0001$	$F(2,50) = 0.7$, NS	$F(2,50) = 0.9$, NS
Static tremor	$F(1,44) = 15.0, p < .001$	$F(2,44) = 7.6, p < .01$	$F(2,44) = 5.3, p < .01$
Arm displacement	$F(1,50) = 152, p < .0001$	$F(2,50) = 2.2$, NS	$F(2,50) = 0.9$, NS
Weight time	$F(1,50) = 28.2, p < .0001$	$F(2,50) = 14.3, p < .0001$	$F(2,50) = 3.2, p < .05$
Hand declination	$F(1,50) = 39.7, p < .0001$	$F(2,50) = 1.9$, NS	$F(2,50) = 0.5$, NS
Limb shake	$F(1,50) = 63.4, p < .0001$	$F(2,50) = 1.6$, NS	$F(2,50) = 1.6$, NS
Muscle tone	$F(1,50) = 14.7, p < .001$	$F(2,50) = 2.8$, NS	$F(2,50) = 2.7$, NS
Braking distance	$F(1,48) = 26.5, p < .0001$	$F(2,48) = 7.0, p < .01$	$F(2,48) = 4.0, p < .05$
Past pointing	$F(1,50) = 5.6, p < .05$	$F(2,50) = 1.1$, NS	$F(2,50) = 0.3$, NS
Finger to finger	$F(1,39) = 9.0, p < .01$	$F(2,39) = 2.7$, NS	$F(2,39) = 3.0$, NS
Adiadochokinesis	$F(1,50) = 31.2, p < .0001$	$F(2,50) = 3.6, p < .05$	$F(2,50) = 0.2$, NS
Toe tapping	$F(1,48) = 28.1, p < .0001$	$F(2,48) = 1.3$, NS	$F(2,48) = 0.2$, NS
Finger to thumb	$F(1,50) = 13.7, p < .001$	$F(2,50) = 0.3$, NS	$F(2,50) = 0.5$, NS

Table iv. Inferential statistics for Reading Age and Dyslexia Groups included were D14, C10; and D18, C14.

Task	Dyslexia	Reading Age	Interaction
Balance time	$F(1,32) = 28.0, p < .0001$	$F(1,32) = 11.0, p < .01$	$F(1,32) = 19.1, p < .0001$
Postural stability	$F(1,32) = 87.1, p < .0001$	$F(1,32) = 0.9$, NS	$F(1,32) = 1.0$, NS
Static tremor	$F(1,28) = 3.4$, NS	$F(1,28) = 7.3, p < .01$	$F(1,28) = 3.9$, NS
Arm displacement	$F(1,32) = 66.2, p < .0001$	$F(1,32) = 0.1$, NS	$F(1,32) = 0.1$, NS
Weight time	$F(1,32) = 5.1, p < .05$	$F(1,32) = 5.6, p < .05$	$F(1,32) = 2.3$, NS
Hand declination	$F(1,32) = 27.1, p < .0001$	$F(1,32) = 0.5$, NS	$F(1,32) = 2.2$, NS
Limb shake	$F(1,32) = 29.2, p < .0001$	$F(1,32) = 1.4$, NS	$F(1,32) = 2.2$, NS
Muscle tone	$F(1,32) = 3.4$, NS	$F(1,32) = 0.2$, NS	$F(1,32) = 0.4$, NS
Braking distance	$F(1,32) = 7.3, p < .05$	$F(1,32) = 6.1, p < .05$	$F(1,32) = 3.8$, NS
Past pointing	$F(1,32) = 4.6, p < .05$	$F(1,32) = 1.0$, NS	$F(1,32) = 2.1$, NS
Finger to finger	$F(1,22) = 0.4$, NS	$F(1,22) = 0.7$, NS	$F(1,22) = 0.0$, NS
Adiadochokinesis	$F(1,32) = 11.6, p < .01$	$F(1,32) = 1.6$ NS	$F(1,32) = 1.5$, NS
Toe tapping	$F(1,32) = 14.6, p < .001$	$F(1,32) = 0.1$, NS	$F(1,32) = 1.2$, NS
Finger to thumb	$F(1,32) = 9.2, p < .01$	$F(1,32) = 1.1$, NS	$F(1,32) = 0.1$, NS

EFFECT SIZE ANALYSES

Effect size analyses (e.g., Cohen 1969) were used in order to facilitate comparison between the tests. Data for each test for each group were first normalized relative to the data for the corre-

sponding control group. For example, for the D18 group, the data for postural stability for each subject were normalized by obtaining the difference of that subject's postural stability score from the mean postural stability score for group C18, and then dividing this difference by the standard deviation of the C18 group for postural stability. Groups D18 and C18 were normalized relative to C18, groups D14 and C14 were normalized relative to C14, and groups C10 and D10 were normalized relative to C10. The sign was adjusted so that a negative effect size indicated below-normal performance. This procedure led to an age-appropriate "effect size" in standard deviation units (analogous to a z-score) for each test for each child. Comparison of effect size magnitudes between tasks gives an index of which tasks prove the most problematic for the children with dyslexia, though it should be noted that the small numbers involved per group limit the precision of the analyses.

All but one task (finger to finger) produced an overall effect size for the groups with dyslexia of –1 or worse (at least 1 *SD* worse than the controls). The largest deficits (3 *SD* or worse away from the controls) were obtained for tremor, arm displacement, hand declination, adiadochokinesis, toe tap, and finger/thumb opposition. The effect sizes for the remaining tasks were predominantly between 2 and 3 *SD* away from control performance, similar to the effect size found for reading deficit for these children (–2.26). The 10-year-old children with dyslexia had markedly reduced muscle tone, resulting in effect sizes of –4 and worse on several tests.

QUALITATIVE OBSERVATIONS

The quantitative data above demonstrate that the children with dyslexia suffered marked difficulties on the tests, but give little feel for quite how the difficulties were manifested. In some cases, performance of the children with dyslexia appeared qualitatively different from that of the controls, and it may be valuable to note these informal observations.

Comments from the subjects on standing with arms extended holding a bottle in each hand (weight time) indicated that the majority found this task physically tiring. However, only the groups with dyslexia consistently complained of pain, or developed wobbliness and tremor. Informal questioning of some of the older subjects with dyslexia on the postural stability task established that they found it very difficult to resist the pressure in their backs, despite their efforts to maintain their posture. Interestingly enough, many of the children with dys-

lexia resisted so actively that they tended to sway backwards, while others attempted to arch their back away from the finger to maintain their balance.

On the arm displacement task, for several children with dyslexia the hand fell progressively further down with each tap. By contrast, the control groups showed normal resilience in response to this perturbation. On the static tremor task, performance was variable for the controls, but all the children with dyslexia showed difficulty with this task, with a few of the 10-year-old children with dyslexia showing such severe tremor that it became difficult to measure accurately. In the muscle tone test, many of the 10-year-old group with dyslexia were unable to produce much tangible resistance, and their muscles felt soft and spongy to the experimenter's touch.

In the past pointing task, the children with dyslexia tended to drift away from the bull's-eye, and showed a strong tendency to drift progressively further downwards, in some cases moving totally off the target (an A4 sheet) to mark the wall. This was rarely seen in even the youngest controls. In both finger and thumb training and adiadochokinesis, it took longer for the children with dyslexia to reach criterial performance level to start testing. In the adiadochokinesis test, in their efforts to keep pace with the tone, many children with dyslexia failed to turn their hands over, lost the rhythm, or lagged behind throughout. The movements of finger and thumb in the group with dyslexia appeared both clumsy and labored in comparison with the smooth performance of even the youngest controls.

INDIVIDUAL ANALYSES

An important question relating to the issue of subtypes (Boder 1973) is whether the above group difficulties also apply for most of the children with dyslexia, or whether the effects are caused by subgroups in the children with dyslexia. The qualitative observations above suggest that most of the children with dyslexia showed difficulties on most of the tasks, but it is preferable to investigate this issue quantitatively, using effect size analyses to derive a rough at-risk measure for each child task. A child was deemed to be at risk on a given task if his or her effect size on that task was –1 or worse (that is, at least one standard deviation below the normal performance for that age). This is a standard statistical procedure (though the reliability of the estimates is again reduced by the small numbers involved). If data are normally distributed, one would expect 15% of the population to be at least one standard deviation below the

mean, and 2% to be at least 2 standard deviations below. The incidence (the proportion of at-risk children) for each group and each task is given in table V (note here that the control groups are included). Overall incidence for the children with dyslexia and controls is presented in the right hand two columns. The right hand column indicates that overall, 5 out of 26 control children, that is, 19% of them, were at risk for balance time, as opposed to (penultimate column) 25 out of the 29 children with dyslexia (86%).

DISCUSSION

Before comparing performance of groups with dyslexia and control groups, it is worth discussing briefly the effects of the other independent variable—age. There were highly significant effects of chronological age ($p < .0001$) for balance time and weight time (table III). There were also significant effects of age for static tremor, braking distance, adiadochokinesis, and to some extent, muscle tone. The mean performance of older and younger controls was roughly equivalent on postural stability, hand tap, hand declination, limb shake, past pointing, finger to finger, toe tapping speed, and finger/thumb opposition.

Turning now to an analysis of the comparative performance of the children with dyslexia and control children, it is clear that predictions of the cerebellar impairment hypothesis have been supported, with deficits (compared with chronological age controls) on all the cerebellar tests, and deficits compared with reading age controls on the majority. Moreover, the performance of the 18-year-old children with dyslexia was consistently worse than children 8 years their junior on many tasks. The only tasks on which the performance of the oldest children with dyslexia was better than the youngest controls were reading age and weight time, and their performance was roughly equivalent to the youngest controls on braking distance.

The strength of the findings is highlighted by several further features. First, the effect size analyses indicate that for many tasks the magnitude of the impairment was greater than that for reading. Second, all the dyslexic children in this study showed clear deficits on the cerebellar tasks—when the 55 participants were ranked in terms of mean effect size across the 14 tasks, there was no overlap between the groups, with the dyslexic children ranked 1–29 (range –1.04 to –6.90) and the controls ranked 30–55 (range +0.68 to –0.96). Third, deficits were appar-

TABLE V. INCIDENCE RATES FOR THE EXPERIMENTAL TASKS.

Incidence	D10	C10	D14	C14	D18	C18	Dys. total (%)	Cont. total (%)
Balance time	8/12	1/8	9/9	2/11	8/8	2/7	25/29 (86)	5/26 (19)
Postural stability	12/12	1/8	9/9	1/11	7/8	2/7	28/29 (97)	4/26 (15)
Tremor	11/11	1/8	7/9	1/11	2/5	1/6	20/25 (80)	3/25 (12)
Arm displacement	12/12	2/8	9/9	2/11	8/8	1/7	29/29 (100)	5/26 (19)
Weight time	7/12	2/8	6/9	2/11	6/8	1/7	19/29 (66)	5/26 (19)
Hand declination	11/12	1/8	8/9	2/11	6/7	1/7	25/28 (89)	4/26 (15)
Limb shake	12/12	1/8	7/9	2/11	5/8	1/7	24/29 (83)	4/26 (15)
Muscle tone	9/12	1/8	3/9	3/11	3/8	0/7	15/29 (52)	4/26 (15)
Braking distance	9/12	1/8	8/9	1/11	5/8	1/7	22/29 (76)	3/26 (12)
Past pointing	4/12	1/8	6/9	2/11	4/8	1/7	14/29 (48)	4/26 (15)
Finger to finger	3/11	1/7	1/5	1/7	5/7	2/7	9/23 (40)	4/21 (19)
Adiadochokinesis	10/12	3/8	6/9	0/11	4/8	1/7	20/29 (69)	4/26 (15)
Toe tap	7/10	1/8	8/9	1/11	7/8	1/7	22/27 (82)	3/26 (12)
Finger & thumb	7/12	1/8	9/9	3/11	7/8	2/7	23/29 (79)	6/26 (23)

ent for almost all the cerebellar tasks—every dyslexic child but one was impaired (effect size of -1 or worse) on at least 8 of the 14 tests, and there were strong effects for all three categories of tests, with all 29 dyslexic children impaired on arm displacement, 28 on postural stability, and 23 on finger/thumb opposition. The mean incidence rate for impairments across all 14 tasks for the dyslexic children was 74%, as opposed to 16% for the controls.

THEORETICAL INTERPRETATION

The severity and generality of the deficits on the cerebellar tests, both across tests and across subjects with dyslexia, provide further intriguing evidence for the cerebellar impairment hypothesis. These are very striking findings, and not readily explained under any other theory of dyslexia.

It is important, however, to note the limitations of these findings. First, though the data reported here provide strong evidence of cerebellar impairment in the groups of children with dyslexia tested, it is possible that research with different samples of children with dyslexia and control children would lead to lower estimates of effect size and incidence rate. Second, the evidence cited here is still indirect and non-specific. Studies of neuroanatomical structure in the cerebellum and cerebellar circuits (cf., Galaburda, Rosen and Sherman 1989), together with brain imaging work focused on cerebellar activation, might lead to better understanding of the deficits obtained on cerebellar tasks. To a large extent, the task of identifying the precise cause(s) of cerebellar impairments in dyslexia awaits the development of a fuller understanding of the role of the cerebellum in normal skill acquisition. We believe that this will prove to be a synergistic interdependence, with the availability of a population of subjects with dyslexia, who exhibit symptoms of cerebellar impairment, providing comparative data to inform the development of better models of normal cerebellar function.

The results obtained here lead to an important paradox that must be addressed. Developmental dyslexia is one of the most heavily studied developmental disabilities. Given the severity and generality of cerebellar deficit in dyslexia, why is it that other research teams have not focused on this apparently obvious problem? As noted earlier, Levinson has for many years argued for cerebellar/vestibular problems, but his subjects were not representative of the general population of children with dyslexia. Denckla (e.g., 1985) and Rudel (1985) also identified motor problems and "soft neurological signs" for representative

groups with dyslexia, but Denckla (1985) then decided that these arose from comorbidity with ADD (Denckla et al. 1985). Denckla and her colleagues compared performance of "pure dyslexia" children (with ADD screened out) and dyslexic children unscreened for ADD. They found the latter to be slower on several rapid repetitive or alternating movements. Unfortunately, in this study, the authors did not use a non-dyslexic control group, so one cannot tell whether or not the pure dyslexia group was also significantly slower than normal. Furthermore, the authors do not report how well the groups were matched for age, so, given that they found strong age effects (as one would expect), it is not clear how much of the difference might be attributable to age differences. The only subsequent study we have found addressing this issue was by Share et al. (1986). These authors found no difference between pure dyslexia and dyslexia plus ADHD on motor skills. Since children with ADHD were excluded from our panel of children with dyslexia, our findings support Denckla's earlier conclusion (with Rudel) that children with dyslexia (whether or not they also suffer from ADHD) show a range of soft neurological signs.

It may be that these research issues were not exhaustively analyzed because of an understandable belief that motor (or cerebellar) deficits were of little theoretical significance because they could not cause the reading difficulties, and were, if anything, a distraction to the main applied question of how to overcome the reading problems. The recent evidence of the role of the cerebellum in motor skill and in language skill (Leiner et al. 1993; Thach 1996) makes it timely to reconsider these issues.

CEREBELLAR DIFFICULTIES: CAUSE OR COVARIATE OF READING PROBLEMS?

The above analyses show that the present sample of children with dyslexia show evidence of dystonia and dyscoordination considered diagnostic of cerebellar damage. However, consider the findings from the important theoretical perspective of the underlying cause of reading difficulties. An objector might reasonably say "Assuming that there is a cerebellar impairment associated with dyslexia, surely you are not saying that this is the cause of the reading problems—I can't see that training our children to balance will really help them with reading or spelling. Rather than causes of the reading problem, cerebellar difficulties are just covariates (symptoms with no direct relationship to reading)."

Our answer to this challenge goes as follows. First, it is important to distinguish between cause, symptom, and remediation. The fact that cerebellar difficulties are a useful symptom does not mean necessarily that they are a valuable method of remediation (nor does it mean that they are the underlying cause). The appropriate remediation depends upon the behavior to be remedied. If the problem is reading, then the appropriate remediation is reading support targeted on the specific difficulties shown. If the problem is handwriting control, then the appropriate support is in terms of motor skill, and so on. We would certainly confirm the importance of phonological support as a central component of a structured program of reading remediation.

The issue of cause versus covariate is an interesting one, to which we do not claim to have the definitive answer. There are, however, good reasons to think that cerebellar problems might well be an important factor underlying the phonological difficulties shown, and hence might provide a causal explanation of the reading difficulties. If an infant has a cerebellar impairment, this will first show up as a mild motor difficulty the infant may be slower to sit up and to walk, and may have greater problems with fine muscular control. Arguably, our most complex motor skill, and that needing the finest control over muscular sequencing, is, in fact, that of articulation. Consequently, one would expect that the infant might be slower to start babbling, and later, talking. Indeed, there is emerging evidence that the early articulatory and manual skills develop in step (Ramsay 1984). Locke (1993) speculates that the co-occurrence of these motor and speech milestones might be attributable to the initial development of the left hemisphere cortical control over the precisely timed muscular movements needed for reaching and speech. In particular, that the left hemisphere assumes control of speech-like activity, and that babbling represents the functional convergence of motor control and sensory feedback systems. Evidence for this view derives from Fowler (1991) who found that very young children first perceive words as a loose bundle of articulated gestures, and in time, the coarticulated gestures become grouped into the representations of phonemes. As Studdert Kennedy (1991, p.10) observes: ". . . language is not an object, or even a skill, that lies outside the child and somehow has to be acquired or internalized. Rather it is a mode of action into which the child grows because the mode is implicit in the human developmental system." Even after speech and walking emerge, one might expect that the skills would be less fluent, less "dex-

trous." If articulation is less fluent than normal, then it takes up more conscious resources, leaving fewer resurces to process the ensuing sensory feedback. In particular, the processing of the auditory, phonemic structure of the words spoken may be less complete. There may, therefore, not be a natural sensitivity to onset, rime, and the phonemic structure of language—in short, one would expect early deficits in phonological awareness (see Snowling and Hume 1994, for a related account).

Note that this argument has links with the established "motor theory" of speech perception first suggested by Liberman ". . . speech is perceived by reference to articulation—that is, the articulatory movements and their sensory effects mediate between the acoustic stimulus and the event we call perception" (1957, p. 122), and subsequently developed by the speech researchers at the Haskins Laboratory. However, it is a much weaker form of the theory, requiring only that use of speech output allows a child to learn to better control articulation, and thereby to better understand speech structure, in much the same way that, much later, practice at driving until the various component skills become fluent, will better allow the driver to notice road signs, etc. In a similar fashion, if one's handwriting is slow and effortful, this presumably will leave less resources available for noticing the shape of the letters drawn, thus militating against the automatic learning of letter shapes. Multisensory support, where a child explicitly traces the shape of a letter with the finger, is an established method of overcoming this failure.

Cerebellar impairment would therefore be predicted to cause the "phonological core deficit" (Stanovich 1988) that has proved such a fruitful explanatory framework for many aspects of dyslexia. Furthermore, the cerebellar impairment hypothesis provides a natural causal explanation of the execrable quality of handwriting frequently shown by children with dyslexia (a characteristic that is but poorly addressed by most existing theories of dyslexia). Handwriting, of course, is a motor skill that, like articulation, requires exquisite timing and coordination of diverse muscle groups. It may be that one reason that spelling, the third criterial skill, appears particularly resistant to remediation (Thomson 1990) is that it requires the simultaneous use of phonological skill and of motor ouput. Perhaps one of the major reasons for the success of computer-based support for reading and spelling (e.g., Nicolson and Fawcett 1994c; Wise and Olson 1995) is that it relieves the student of the motor writing task, leaving capacity free to focus on spelling or reading itself.

CONCLUSIONS

In conclusion, recent research on primitive skills (Nicolson and Fawcett 1994b, 1995) has shown that children with dyslexia have deficits in phonological skill, speed of processing, and motor skill. These deficits are well-characterized as problems in skill automatization, which are normally masked by the process of conscious compensation (Nicolson and Fawcett 1990). However, to describe the symptoms as a "general automatization deficit" explains neither the cause nor the pattern of difficulties. Its involvement in the above skills and in skill automatization suggested the cerebellum as a natural focus for further dyslexia research. The cerebellar impairment hypothesis provides a principled account of the qualitative aspects of the data; it provides a reasonable account of the precise quantitative nature of the effects; and it has predicted hitherto unsuspected deficits in temporal estimation and clinical tests of coordination and muscle tone. Furthermore, cerebellar impairment provides a natural causal explanation of phonological difficulties and of reading, writing and spelling problems that are the criterial measures for dyslexia. Therefore, if similar difficulties are shown by further groups of children with dyslexia, cerebellar impairment may provide a coherent and integrative framework for understanding dyslexia. In conclusion, although it would be premature to assign the difficulties of children with dyslexia to the cerebellum alone owing to its rich interconnections with cerebral cortex and the basal ganglia, the severity of the classic cerebellar signs suggests strongly that the cerebellum is one of the key structures involved.

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