Developmental Dyslexia: The Role of the Cerebellum[1]

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Children with dyslexia suffer from unexpected problems in reading, writing and spelling. The dominant causal hypothesis has been that the deficits arise from some impairment in phonological processing ability. Initial studies revealed that children with dyslexia suffered severe deficits in skills including not only phonological skill but also picture naming speed, bead threading and balance. Given the growing evidence that the cerebellum is directly involved in acquiring 'language dexterity', all the above deficits are directly consistent with cerebellar impairment. Further studies established that the panel of children with dyslexia showed severe deficits on clinical and theoretical tests of cerebellar function, among the largest obtained in our research programme. The cerebellar impairment hypothesis provides a causal explanation for developental dyslexia, subsuming the phonological deficit account within a broader framework.

Keywords: dyslexia; reading disability; automatisation; cerebellum

INTRODUCTION: DEVELOPMENTAL DYSLEXIA

Specific developmental dyslexia is normally identified by unexpected problems in learning to read for children of average or above-average intelligence—'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' (from the definition by the World Federation of Neurology, 1968, p. 26). A typical estimate of the prevalence of dyslexia in Western school populations is 3%–4% (Badian, 1984; Jorm *et al.*, 1986; Miles, 1991), though considerably higher estimates have also been suggested. Roughly four times as many boys as girls are diagnosed.[2] Dyslexia is genetic in origin (e.g. Smith *et al.*, 1983; DeFries and Alarcón,

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[2]Some US investigators (e.g. Shaywitz *et al.*, 1990) have suggested that the gender imbalance may be a selection artefact and that the gender ratio is even. However, Miles, Haslum and Wheeler (1998) suggest that, on the contrary, the equal ratio derives from the use of the criterion 'poor reading in relation to intelligence' by the US researchers, rather than a more clinical definition including spelling and items from the Bangor Dyslexia Test (Miles, 1982, 1997).

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1996), and so, even though many dyslexic adults have substantially overcome their reading problems, their underlying dyslexia remains and may be identified by sensitive tests (Fawcett and Nicolson, 1995a, 1998).

Substantial progress has been made in the methods for overcoming the reading difficulties of children with dyslexia, but the success of reading remediation depends critically upon how early the support can be provided, with by far the best results being obtained if extra support is given as the child is learning to read, rather than after one or two years of reading failure (Strag, 1972; Bradley, 1988; Lundberg, Frost and Peterson, 1988; see also Clay, 1993). Unfortunately, the underlying cause has proved frustratingly elusive, to the extent that there is now widespread concern over methods of diagnosis even at 8 years, together with confusion over appropriate methods of theory development (see Lyon *et al.*, 1993, for a series of articles on these issues).

In this paper we present the findings of our 10-year research programme aimed at a fuller understanding of the underlying causes of dyslexia. The initial research was based on the skill learning framework and discovered symptoms consistent with faulty skill automatisation even in the gross motor skill of balance. The second research phase was deliberately exploratory, aimed at delineating the pattern, severity and incidence of difficulties shown by children with dyslexia across the spectrum of skills. These studies indicated severe and persistent problems on phonological skill, processing speed, balance and motor skill. The third phase attempted to identify possible cause(s) of the range of deficits, focusing on the possibility of cerebellar impairment. The cerebellar impairment hypothesis provides a potential parsimonious explanation of the range of problems and makes a number of specific predictions. Empirical work both with our existing panel of subjects and with further dyslexic subjects confirmed these predictions, thereby providing strong evidence that cerebellar impairment may well be one of the underlying causes of dyslexia.

The learning framework and the cerebellar impairment hypothesis run counter to the mainstream of dyslexia research, and it is therefore important to explain not only the rationale for our own work but also the rationale for alternative conceptualizations, attempting to explain that the cerebellar impairment hypothesis is not only compatible with existing approaches, but that it actually provides a coherent, explanatory framework capable of unifying what are currently diverse approaches. We start with a brief survey of current theoretical approaches to the understanding of dyslexia, focusing on the influential phonological deficit hypothesis and the more recent temporal processing deficit. This survey leads into the set of issues that our research programme was designed to address. We then outline the findings of our own research programme, leading to the establishment of the cerebellar impairment hypothesis. Finally we return to the key issues identified initially, arguing that the cerebellar impairment hypothesis presents a coherent explanatory framework for the underlying cause of dyslexia.

Dyslexia: the Range of Difficulties

One of the fascinations of dyslexia for researchers is that, whatever one's interest in human behaviour and performance, children with dyslexia will obligingly show interesting abnormalities in precisely that behaviour. Deficits have now been established in motor skill, in visual processing, in rapid processing and in memory, together with tantalizing links to neuroanatomical irregularities. The body of

knowledge on dyslexia is very extensive and growing rapidly. Neurological and genetic background factors are thoroughly discussed in edited works by Galaburda (1989) and Duane and Gray (1990). General cognitive accounts are provided in Seymour (1986), Snowling (1987) and Miles (1993). A set of reviews of skill (phonological, visual and motor, together with speed of processing) is provided in Fawcett and Nicolson (1994). Frith (1997) provides a valuable recent review of the area, suggesting that there are three major current 'causal' explanations of dyslexia, namely phonological deficit, rapid temporal processing deficit and cerebellar impairment.

Phonological Deficit

Early pioneers in dyslexia research such as Morgan, Hinshelwood and Orton believed that visual problems underlay the apparent 'word blindness', but it is now acknowledged that problems of language must be substantially involved. One of the major achievements of dyslexia research in the past decade was the demonstration that many of the reading-related deficits are attributable to some disorder of phonological processing (Vellutino, 1979; Bradley and Bryant, 1983; Snowling *et al*., 1986; Stanovich, 1988)—see Shankweiler *et al*. (1995) for a review. Initial evidence for a phonological deficit derived from Bradley and Bryant (1978), who discovered that children with dyslexia were significantly worse at a rhyme judgement task than younger children who had reached the same level in their reading. A further study on the effects of training in rhyming on pre-school children's subsequent reading performance (Bradley and Bryant, 1983) investigated the comparative effectiveness of training in rhyming and alliteration skills ('sound categorization') versus training in semantic categorization for pre-readers with deficits in rhyming skills. The group who were trained in rhyming made significantly more progress in reading, but equivalent progress in mathematics, providing evidence for a causal link between early phonological skills and acquisition of reading. A series of further studies have confirmed and extended Bradley and Bryant's results both for normal readers (e.g. Hatcher, Hulme and Ellis, 1994) and for children with dyslexia (e.g. Rack, 1985; Lundberg, Frost and Petersen, 1988; Olson, Wise and Rack, 1989; Ackerman, Dykman and Gardner, 1990). This converging evidence led to the emergence of the phonological deficit hypothesis as the consensus theoretical belief of most psychology researchers, namely that children with dyslexia suffer from an early impairment in their phonological awareness, and that this impairment prevents them from acquiring the word decoding and blending skills necessary for normal acquisition of the skill of reading. Indeed, this is reflected in a recent redefinition of dyslexia, in which a key aspect reads as follows: *a specific language based disorder of constitutional origin, characterized by difficulties in single word decoding, usually reflecting insufficient phonological processing abilities* (Orton Society, 1995).

Rapid Temporal Processing Deficit

Other dyslexia research has established problems more generally in rapid performance. Denckla and Rudel (1976) discovered problems in 'rapid automatized naming' of successive stimuli (including non-linguistic stimuli such as colours); Tallal (1985)

identified impairments in recalling the order of rapidly presented temporal events; and Wolf (1991), reporting a five-year longitudinal study, established that early deficits in naming speed for letters and numbers predicted later deficits in reading, with a direct relationship between the speed deficit and the severity of the reading impairment. Further indications of reduced speed of temporal processing, even for tasks without phonological components, were provided by Nicolson and Fawcett (1994a), who demonstrated reduced speed of lexical access and even of selective choice reaction to one of two pure tones. Interestingly, however, they established also that simple reaction to that tone was normal, suggesting that it was the need to make a decision that caused the reduced speed. Abnormalities have also been found in the auditory magnocellular system (Galaburda, Menard and Rosen, 1994).

Anomalies have also been identified in rapid visual processing. For instance, Lovegrove and his colleagues (e.g. Lovegrove, Garzia and Nicholson, 1990) demonstrated that children with dyslexia had impaired sensitivity for detecting flicker. Furthermore, this deficit has now been linked to neuroanatomical abnormalities in the magnocellular pathway linking the eye to the visual cortex via the lateral geniculate nucleus (Livingstone *et al.*, 1991). Visual deficits have also been identified by Stein and his colleagues (e.g. Stein, 1989).

The apparent generality across modalities of the deficits in temporal processing speed has led several researchers (Llinás, 1993; Tallal, Miller and Fitch, 1993; Stein, 1994; Stein and Walsh, 1997) to argue that a temporal processing deficit might be the cause of difficulties in dyslexia. This is an interesting hypothesis which does provide a link between known neuroanatomical irregularities through cognitive skills to reading. It can also account for specific phonological difficulties in terms of reduced ability to discriminate between consonants with similar acoustic spectra. On the other hand, it is not clear why such a problem could cause the established difficulties in terms of detecting rhyme or ability to segment words into phonemes. Furthermore, Mody, Studdert-Kennedy and Brady (1997) provide evidence that the temporal processing difficulties apply only to the speech domain rather than to all auditory processing.

Cerebellar Impairment

The third major causal explanation is our own cerebellar deficit theory. Rather than summarizing the evidence at this stage, we shall attempt to explain how the hypothesis evolved from our previous research. This forms the bulk of the remainder of this paper.

Other Evidence

It would be incomplete to conclude this review of the range of behavioural and anatomical differences between control children and those with dyslexia without providing pointers to recent functional imaging studies that provide further fascinating but inconclusive evidence of differences in brain activation patterns when subjects with and without dyslexia are undertaking experimental tasks. Evidence derives from both language-based tasks (e.g. Paulesu *et al.*, 1996) and visual tasks (e.g. Eden *et al.*, 1996). However, in view of the relatively small number of subjects involved, and the difficulty of definitive interpretation of functional imaging studies currently, we hesitate to attempt to interpret these studies within a theoretical framework.

It is clear, therefore, that researchers from different backgrounds have identified a range of apparently independent problems in dyslexia. One of the greatest challenges for theoretical research in dyslexia is to find an explanatory framework sufficiently general to accommodate the diversity of the deficits in dyslexia while sufficiently specific to generate testable predictions, to support better diagnostic procedures and to inform remediation methods. This challenge formed the motivation for our research programme.

THE SHEFFIELD DYSLEXIA RESEARCH PROGRAMME

Although by the late 1980s dyslexia had been investigated from a large range of theoretical perspectives, no researchers appeared to have adopted a learning framework, systematically assessing the various components of learning ability in children with dyslexia. This was something of a puzzle, given that the key difficulty is that of learning to read (and that dyslexia is known as 'learning disability' in the US and 'specific learning difficulties' in the UK). Consequently, in our initial research we adopted a learning, or skill acquisition, perspective.

It is evident that a learning perspective should be of use in analyses of reading problems. Consider the conclusions of a recent influential overview and analysis of the teaching of reading: *. . . Laboratory research indicates that the most critical factor beneath fluent word reading is the ability to recognise letters, spelling patterns, and whole words effortlessly, automatically and visually. The central goal of all reading instruction—comprehension—depends critically on this ability* (Adams, 1990, p. 54). The reason that theorists have not seriously considered learning as a viable framework is that it fails to explain the apparent specificity of the deficits in dyslexia (Stanovich, 1988). If they have a general problem in learning, why do children with dyslexia not show problems in *all* skills, cognitive and motor? In our approach to this difficulty we were encouraged first by the observation that, whatever skill theorists had examined carefully, a deficit had been observed in children with dyslexia. Furthermore, careful observation of children with dyslexia suggests that, although they appear to be behaving normally, they show unusual lapses of concentration and get tired more quickly than normal when performing a skill (Augur, 1985). In the words of the parent of one of our subject panel, life for a child with dyslexia might be like living in a foreign country, where it is possible to get by adequately, but only at the expense of continual concentration and effort. One of the key concepts in skill acquisition is automatisation—the process by which skilled performance becomes smoother and smoother, requiring less and less effort, following extensive practice (Fitts and Posner, 1967; Shiffrin and Schneider, 1977; Anderson, 1982). This belief in a learning deficit led us (Nicolson and Fawcett, 1990) to formulate and test two linked hypotheses: first, the 'dyslexic automatisation deficit' hypothesis, that children with dyslexia have unusual difficulty in automatizing any skill, whether motor or cognitive; and second, the 'conscious compensation' hypothesis, namely that children with dyslexia are normally able to overcome their automatisation deficit by means of consciously compensating for it, that is, by trying harder and/or by using strategies to minimize or mask the deficit.

Phase 1: Automatization, Motor Skill and Dyslexia

The automatisation deficit hypothesis predicts difficulties in acquisition of any learned skill (including reading, spelling and phonological skill). There was therefore little

point in testing it in the phonological domain, since all theories predict deficit. Consequently, in a rigorous test of the hypothesis, we investigated a range of fine and gross motor skills, on the basis that these have absolutely no linguistic involvement, and so any deficits found would be hard to explain via the phonological deficit hypothesis. Details of the range of studies are provided in Fawcett (1990). The clearest results derive from the gross motor skill of balance (Nicolson and Fawcett, 1990), since this is one of the most practised of all skills and thus the most likely to be completely automatized. The subjects were 23 children with dyslexia around 13 years old (defined in terms of a standard discrepancy/exclusionary criterion used in all the studies—namely children of normal or above normal IQ (operationalized as IQ of 90 or more on the WISC-R (Wechsler, 1976)), without known primary emotional, behavioural or socioeconomic problems, whose reading age was at least 18 months behind their chronological age) and eight normally achieving children, with groups matched overall for age and IQ. Performance was monitored for three tasks: standing on both feet (one foot directly in front of the other); standing on one foot; and walking. The balance tasks were performed under two conditions: single task balance, in which the subjects had merely to balance; and dual task balance, in which they had to balance while undertaking a further secondary task. Two secondary tasks were used: either counting or performing a choice reaction task. Each secondary task was initially 'calibrated' so as to be of equivalent difficulty for each subject, by adjusting the task difficulty (for counting) or by providing extended training (for choice reactions) so that, under 'just counting' or 'just choice reaction' conditions, all subjects fell into the same performance band.

The results were exactly as predicted by the automatisation deficit/conscious compensation hypothesis. Under single task balance conditions there was no difference in balance between the groups. Under dual task conditions the children with dyslexia showed a highly significant impairment in balance (indicating lack of automaticity for balance) whereas the control children showed no deficit (indicating balance automaticity). Even more convincing, in addition to the significant differences at the group level, the pattern of performance also applied to almost all the individuals, with 22 out of the 23 children with dyslexia showing a decrement under dual task conditions whereas most of the controls actually improved (owing, no doubt, to the effect of practice). More recently we have extended these findings, obtaining qualitatively similar results with younger children with dyslexia and also with blindfold balance replacing the dual task balance (Fawcett and Nicolson, 1992). A Dutch study (Yap and van der Leij, 1994) has also replicated the general findings of balance deficit, though it should be noted that the balance task to be used needs to be adapted to the age of the subjects.

These results provided strong support for the automatisation deficit hypothesis, in that it alone of the major theories predicted the obtained pattern of results. Even so, in view of the small number of subjects participating and the lack of information about other deficits of the children with dyslexia, it would be foolhardy to attempt to generalize too much from these isolated studies. This led to a decision to collect much richer data, in phase 2 of the research.

Phase 2: Primitive Skills and Dyslexia

Of the three main cognitive theories for dyslexia (phonological deficit, temporal processing deficit and automatisation deficit), the first two were arguably a little too

specific to be credible explanations of the range of problems suffered by children with dyslexia, whereas the third was perhaps too general to account for the precise pattern of difficulties shown. It is difficult to distinguish these theories in the domain of reading, since all three predict that there will be phonological difficulties and that these wil in turn lead to reading problems. Furthermore, it is possible that there are different subtypes of dyslexia (Boder, 1973; Seymour, 1987) (though see Thomson, 1999) and that in fact the different approaches tend to sample different populations of children with dyslexia. The only way that the generality of deficit can be addressed is by administering a wide range of tests to the same groups of children with dyslexia, and by assessing the incidence of deficit on each task individually for each child as well as performing a between-group analysis. This allows both the severity and the incidence to be assessed independently. Consequently we attempted to characterize performance on the entire range of skill (even skills which were thought to be unaffected by dyslexia). In this way we hoped to be able to build up an overall picture of the pattern of difficulties associated with dyslexia, constructing a corpus of data which would be useful to theorists of all persuasions. Naturally we hoped also that creation of this corpus would hasten progress towards the major theoretical goal of dyslexia research, identification of the underlying cause or causes.

Subjects

The standard discrepancy/exclusionary criterion described above was adopted. Three groups of children with dyslexia, mean ages 16, 12 and 8 years,[3] were recruited, together with three groups of normally achieving children matched for age and IQ. This gave six groups, D16, D12 and D8, and C16, C12 and C8, for the three age groups of children with dyslexia and controls respectively. This three-age-group design allows performance to be compared with children of the same age (D16 vs. C16; D12 vs. C12; D8 vs. C8), children of around the same reading age (D16 vs. C12; D12 vs. C8) and children of around half the age (D16 vs. C8).

Skill tests

Next a variety of tests were designed which were intended to tap performance on 'primitive' cognitive and motor skills, that is, basic skills which form the building blocks for more complex skills in each domain. In addition to psychometric tests, four generic types of test were used, namely tests of phonological skill, working memory, information processing speed and motor skill. Detailed presentations of results and procedures for the different types of skill are given in Fawcett and Nicolson (1995a,b,c), and overviews are presented in Nicolson and Fawcett (1994b, 1995).

Results

There were significant differences between children with dyslexia and their chronological age controls on all but two of the 23 measures presented. Only on the simple reaction time measures did children with dyslexia consistently perform at normal standards at all three age levels. The impairments compared with chronological age controls were therefore extensive, but some impairment is to be expected given the secondary consequences of reading difficulties in terms of reduced exposure to print.

[3]The children with dyslexia were located via the local Dyslexia Institute or the local branch of the British Dyslexia Association. Other than checking that the children met our criteria for dyslexia and that they were willing to undertake testing on a long-term basis, no screening or selection whatsoever was undertaken. Subjects were paid around $5 per hour, and participated with fully informed consent.

Of critical theoretical significance is the analysis compared with reading age controls, since such an impairment cannot be caused entirely by reading difficulties and therefore may indicate some more fundamental problem (Bryant and Goswami, 1986). Tests on which significant impairments compared with reading age controls were found were as follows: spelling ($p<0.01$); segmentation ($p<0.0001$); word flash (minimum presentation time needed to perceive and read a simple word) ($p<0.05$); picture naming speed ($p<0.05$); bead threading ($p<0.01$); balance one foot blindfold ($p<0.0001$); and dual task balance (balance when also doing a choice reaction task) ($p<0.01$). Significantly better performance than reading age controls was found only for simple reaction time ($p<0.0001$). No other tests revealed significant differences.

It was clear, therefore, that the between-group analyses indicated significant deficits, even compared with reading age controls, in several skill domains. Further analyses were required to investigate two central issues: the relative severity of the deficits on the various tasks; and the relative individual incidence of deficit for the tasks. This was undertaken by first normalizing the data for each test for each group relative to those of the corresponding control group.[4] This procedure led to an age-appropriate 'effect size' in standard deviation units (analogous to a *z*-score) for each test for each child (e.g. Cohen, 1969). The sign was adjusted such that a negative effect size indicated below-normal performance. 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 expected performance for that age). 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 two standard deviations below. Representative effect sizes (averaged across age levels) and incidence (the proportion of at-risk children) for the groups with dyslexia were as follows: spelling age (-5.24, 97%); reading age (-3.64, 100%); segmentation (-3.58, 85%); balance one foot blindfold (-3.51, 82%); articulation rate (-2.45, 56%); picture naming speed (-2.28, 69%); pegboard time (-1.56, 46%); lexical decision time (-1.51, 36%); rhyme (-1.09, 46%); and memory span (-0.51, 31%). These results confirm that most of the children with dyslexia were impaired on a wide range of tasks. Naturally they were all impaired on reading, since this was a requirement for inclusion in the dyslexia groups; and the even greater impairment on spelling than reading is as expected (Thomson, 1984). The other tasks for which the mean effect size was -2 or worse are balance (dual task, one foot blindfold and one foot non-blindfold), segmentation, letter naming speed and articulation rate, with an incidence rate of around 80% on most of these skills.

Inspection of the individual data revealed that, of the 33 children with dyslexia who attempted both one foot blindfold balance and dual task balance, only three were not scored at risk on either task. Even these three showed a deficit in two feet blindfold balance. Balance difficulties, especially when blindfolded, appear therefore to be associated with all our sample of children with dyslexia. Furthermore, of the 32 children with dyslexia who completed at least two of the three tasks (dual task balance, segmentation, picture naming speed), 29 (90%) had positive scores on at least two. Only one child with dyslexia was not at risk on any of the three, and even he was at risk on rhyme and two foot blindfold balance. By contrast, only two out of the 32

[4]For example, for the D16 group the data for blindfold balance for each subject were normalized by obtaining the difference of that subject's blindfold balance score from the mean blindfold balance score for group C16, and then dividing this difference by the standard deviation of the C16 group for blindfold balance. Groups D16 and C16 were normalized relative to C16, groups D12 and C12 were normalized relative to C12, and groups C8 and D8 were normalized relative to C8.

controls completing at least two of these tasks showed a deficit on two or more tasks (Nicolson and Fawcett, 1995).

In short, the children with dyslexia showed deficits across the spectrum of primitive skills, and there was no evidence for subtypes of dyslexia, at least within the sample investigated. The qualitative aspects of performance were therefore consistent with automatisation deficit. However, despite this success, we feel that the automatisation deficit hypothesis is probably better seen as a descriptive theory—an economical characterization of the symptoms—rather than an explanation of the underlying cause. A causal explanation should account for the precise pattern of results obtained and should identify the mechanism(s) underlying these symptoms. The search for an underlying cause was the focus of the third phase of our research programme.

Phase 3: Dyslexia and the Cerebellum

The results from phase 2 indicate that any complete theory of dyslexia should be able to account for problems in phonological skill, in motor skill, in automatisation and in information processing speed. Phonological skill and blindfold balance not only show a marked deficit at age 8 years, but also appear resistant to maturational improvement, to the extent that the oldest children with dyslexia had barely reached the performance level of the youngest controls. Clearly, if there were some mechanism underlying the deficits in balance, automatisation, motor skill and phonological skill, that mechanism would be a prime candidate for the underlying cause of dyslexia.

The deficit in balance (especially blindfold balance) suggests some disorder of the vestibular system or the cerebellum. Levinson (e.g. Frank and Levinson, 1973; Levinson, 1990), on the basis of studies of nystagmus and optokinetic fixation in children with dyslexia, has for some time argued for mild cerebellar dysfunction as a causal factor in dyslexia.[5] Deficits in motor skill and automatisation also point strongly to the cerebellum, which has traditionally been considered as a motor area (Holmes, 1917, 1939; Eccles, Ito and Szentagothai, 1967; Stein and Glickstein, 1992), and it is also claimed to be involved in the automatisation of motor skill and in adaptive learning control via the cerebellar structures (Ito, 1984, 1990). A cerebellar inactivation study in rabbits (Krupa, Thompson and Thompson, 1993) has provided further evidence that the cerebellum is particularly involved in initial skill acquisition. A clear demonstration of the role of the cerebellum in 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).

Nonetheless, despite the early work of Levinson, the cerebellum has largely been discounted in the dyslexia literature owing to its supposed lack of involvement in linguistic and cognitive skill. However, as Leiner, Leiner and Dow (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, Leiner and Dow (1989, 1993) concluded that the cerebellum is therefore central for the acquisition of 'language dexterity'. In effect,

[5]It is important to note that Levinson's findings are largely based on individual case studies, and the generality of his findings has been seriously questioned (e.g. Silver, 1987).

then, they proposed that the cerebellum is critically involved in the automatisation of any skill, whether motor or cognitive. Recent tomographic and magnetic resonance studies (Decety *et al.*, 1990; Roland *et al.*, 1990; Akshoomoff and Courchesne, 1992; Paulesu, Frith and Frackowiak, 1993) and recent studies of cerebellar patients (Fiez *et al.*, 1992; Silveri, Leggio and Molinari, 1994) have provided further support for the involvement of the cerebellum in cognitive activities. Recent reviews of these and related findings are provided in Thach (1996).

The results of our studies in phase 2 suggested that the most severe deficits for children with dyslexia were in terms of phonological skill, naming speed, motor skill and balance. This is precisely the pattern that would be predicted in the light of current understanding of the role of the cerebellum in skill acquisition and execution. Clearly, therefore, mild cerebellar impairment was a prime candidate for the underlying cause of dyslexia. Investigation of cerebellar functioning in dyslexia formed the focus of the third phase of our programme.

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. While space precludes a full discussion of this, 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.

Clinical cerebellar symptoms in children with dyslexia

If there is indeed a cerebellar dysfunction in dyslexia, then children with dyslexia should show marked impairment 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). Traditional symptoms of cerebellar dysfunction are dystonia (problems with muscle tone) and ataxia (disturbance in posture, gait or movements of the extremities). Apart from our own work on balance and Levinson's (1990) controversial findings, there was no evidence in the literature that children with dyslexia do suffer from this type of problem. Consequently, in another stringent test of the cerebellar impairment hypothesis, we replicated the clinical cerebellar tests described in Dow and Moruzzi (1958), using groups of children with dyslexia and matched controls. A fuller report is provided in Fawcett, Nicolson and Dean (1996).

Subjects. The design was the six-group design used in phase 2, and subject selection for children with dyslexia used the same discrepancy/exclusionary criterion as in previous phases. Subjects with dyslexia satisfied both of the two standard exclusionary criteria for dyslexia, namely (i) normal or above normal IQ (operationalized as IQ of 90 or more on the full-scale WISC-R (Wechsler, 1976)), without known primary emotional behavioural or socioeconomic problems, whose reading age (RA) was at least 18

months behind their chronological age (CA), and (ii) 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).[6] 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, D18, D14 and D10, and C18, C14 and C10, for the three age groups of children with dyslexia and matched controls respectively.

Experimental tasks. The tests in the Dow and Moruzzi (1958) battery may be divided into three types: first, two tasks assessing the ability to maintain posture and muscle tone while standing and in response to active displacement of station; second, a series of seven 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 five tests of the ability to initiate and maintain a complex voluntary movement.

Results. 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 (dyslexia vs. control). 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 (dyslexia vs. control). The performance of the children with dyslexia was significantly worse on 11 out of the 14 tests, with the performance decrement on tremor and muscle tone close to significance, and equivalent performance only on finger to finger pointing.

Effect size analyses were again used. 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. All but one task (finger to finger) produced an overall effect size for the groups with dyslexia of -1 or worse (at least one standard deviation worse than the controls). Deficits more severe than reading age (-2.26, 100%) were for finger and thumb opposition (-7.08, 79%); tremor (-4.44, 80%); arm displacement (-3.59, 100%); toe tap (-3.55, 82%); limb shake (-3.17, 83%); diadochokinesis—speed of alternating tapping on table with palm and back of hand (-3.22, 69%); postural stability—movement when pushed gently in the back (-2.86, 97%); and muscle tone (-2.42, 52%). The performance of the 10-year-old children with dyslexia was markedly poorer than for the older dyslexic children on several tests of muscle tone, with effect size of -4 and worse.

Discussion. It is clear that the predictions of the cerebellar impairment hypothesis were confirmed, with impairments (compared with chronological age controls) on all the cerebellar tests, and impairments compared with reading age controls on the majority.

[6]The DSM-IIIR assessment for ADHD involves 14 simple yes/no questions, with a 'yes' on at least eight 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 ($F<1$).

Moreover, the performance of the 18-year-old children with dyslexia was consistently worse than control children 8 years their junior on many tasks. The effect size analyses indicated for many tasks (postural stability, tremor, arm displacement, limb shake, muscle tone, adiadochokinesis, toe tap and finger/thumb opposition) that the magnitude of the impairment was greater than that for reading. Furthermore, the individual analyses indicated that for eight of the 14 tasks over 80% of the children with dyslexia were 'at risk'.

The strength of the findings is highlighted by several features. First, as noted above, the effect size analyses indicate that for most tasks the magnitude of the impairment was greater than that for reading. Second, all the children with dyslexia in this study showed clear deficits on the cerebellar tasks—when the 55 participants were ranked in terms of percentage impairment on the set of tests, there was no overlap between the children with dyslexia and the controls, with the children with dyslexia ranked 1–29 and the controls 30–55! Third, deficits were apparent for almost all the cerebellar tasks, suggesting that impairments probably apply across the entire cerebellum, rather than being specific to a single region—28 of the 29 children with dyslexia were impaired (effect size of -1 or worse) on at least eight of the 14 tests, and there were strong effects for all three categories of test, with all 29 children with dyslexia impaired on arm displacement, 28 impaired on postural stability and 23 impaired on finger/thumb opposition. Finally, the incidence rates for impairments in the children with dyslexia were actually higher than those cited for cerebellar patients (Dow and Moruzzi, 1958). This may well reflect localization of damage in the cerebellar patients, with many having spared functionality in several cerebellar regions, but even so it is an extraordinary finding given the failure of earlier dyslexia research to identify these problems.

It may be seen, therefore, that the hypothesis of impairment of the cerebellum or its neural tracts provides a natural explanation of the three major deficits (balance, phonological skill and temporal estimation) obtained in this series of studies. Given the current view that the cerebellum is particularly active in initial skill acquisition (Ito, 1990; Krupa, Thompson and Thompson, 1993; Jenkins *et al*., 1994), the hypothesis also accounts naturally for our finding (Fawcett and Nicolson, 1994b) that children with dyslexia have problems *initially* in blending two skills, but that subsequent skill acquisition is not abnormal save for difficulties in error elimination.

Cerebellar function in further groups of children with dyslexia

It should be noted, however, that even though the data reported above provided 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 controls would lead to lower estimates of effect size and incidence rate. We investigated this issue in parallel research (Fawcett and Nicolson, 1999), using further samples of 126 children with dyslexia and control children. The subjects were split into four age groups (8–9 years, 10–11 years, 12–13 years and 14–16 years), with roughly equal numbers of controls and children with dyslexia in each group. Children with dyslexia were taken from dyslexia units at private schools, and controls were taken from the same school where possible. No selection was made on the children with dyslexia other than that they fulfilled the standard discrepancy/exclusionary criterion used in the other studies. The children with dyslexia were not matched for IQ with the controls, and in fact the control children had a higher full-scale IQ overall. The control children were also reading significantly above their

age level, leading to extreme effect sizes for reading and spelling for the groups with dyslexia.

A selection of experimental tasks was administered to the children, including both cerebellar tasks and other tasks known to be sensitive to dyslexia. In all the tests of cerebellar function, together with segmentation and nonsense word repetition, the performance of the groups with dyslexia was significantly worse than that of their chronological age groups. Only picture naming speed was not significantly worse. The effect size analyses also provide a similar picture to the panel study,[7] though (as one would expect for the larger and more heterogeneous set of control children) the overall effect sizes were lower. Spelling had the most extreme effect size (− 4.26, 91%), with limb shake (− 2.62, 86%) and postural stability (− 2.88, 78%) being comparable with reading (− 3.56, 92%). Segmentation was somewhat less strong[8] (− 1.76, 56%), which in turn was more marked than nonsense word repetition (− 1.45, 63%). In line with the earlier study, comparing children with dyslexia and controls, some of the most notable results were the exceptionally poor performance of all four groups with dyslexia on postural stability and limb shake. It is interesting to note that the balance impairment as revealed by postural stability (reaction to a push in the back) was considerably more marked than that shown by one foot balance—the number of wobbles without external disturbance (− 0.51, 23%).

In order to check that these results did not simply derive from the higher IQ of the control children, a second analysis was undertaken on a subgroup of the dyslexics and controls matched for IQ. The same pattern of results obtained suggesting that the cerebellar impairments were not simply a function of low IQ (see Fawcett and Nicolson, 1999, for details).

It seems that we can conclude, as in the previous study, that despite strong tendencies for evidence of acute cerebellar impairment to dissipate over time with the overlay of learning and compensatory skills, the majority of the 13- and 15-year-old children with dyslexia tested continue to show deficits on both simple and complex tests. It would appear, therefore, that the evidence of cerebellar impairment obtained in the previous study does generalize well to further groups of children with dyslexia.

FROM CEREBELLAR IMPAIRMENT TO READING, SPELLING AND WRITING: A HYPOTHETICAL CAUSAL CHAIN

In summary of the results of phases 1–3 of our research programme, the initial studies confirmed the fruitfulness of a learning framework in dyslexia research, revealing unexpectedly general symptoms consistent with incomplete automatisation of skill. The second phase of research confirmed and extended these findings with further groups of children with dyslexia and controls and further tasks, indicating that the children with dyslexia suffered significant deficits compared with controls of the same

[7]Balance performance is somewhat anomalous here, in that, despite the significant effect of dyslexia, the overall effect size of the discrepancy is low. Analysis of the individual results indicated that this anomaly was attributable to high variability in the control groups, with the standard deviation almost equal to the mean. Consequently effect sizes are reduced across the board. This greater variability suggests that balance may not be a useful task for screening purposes.

[8]It is important to note that training in phonological awareness and in grapheme–phoneme translation was a central component of the teaching methodology of the school for the children with dyslexia. We interpret the relatively mild deficit on phonological skills as a tribute to the quality of teaching.

reading age in primitive skills from phonological skill through picture naming speed to motor skill and balance. The pattern of deficits in phase 2 was strongly suggestive of cerebellar impairment and in phase 3 cerebellar function was assessed in a number of ways. In each study, children with dyslexia showed precisely the pattern of difficulties predicted from cerebellar impairments, with perhaps the most direct evidence being the clear classic clinical signs of cerebellar dysfunction. The issue that we wish to address in this final section is the extent to which cerebellar impairment may be considered a truly causal explanation.

Nicolson (1996) develops the argument that much dyslexia research has been unnecessarily confrontational owing to a failure to distinguish clearly between cause, symptom and treatment for dyslexia. Even though it now appears that cerebellar-type problems are important symptoms of dyslexia (and therefore valuable for diagnosis), there is no evidence that remediation of the cerebellar symptoms will have any effect on the primary educational difficulties relating to literacy. Furthermore, there is no compelling reason as yet to give cerebellar symptoms a privileged position as manifestations of the 'true' underlying cause. It may well be that cerebellar symptoms, literacy difficulties and phonological difficulties are all covariates of some as yet unidentified underlying cause. Consequently, it is incumbent upon us to attempt to outline some method by which cerebellar impairment might indeed lead to the range of symptoms shown. The following account is, we think, plausible but certainly speculative.

Our initial discussion of the cerebellum described its known role in execution and automatisation of motor skill. Now consider the possible role of the cerebellum in the proceduralization of cognitive skills. Leiner, Leiner and Dow (1989) put the case clearly in their 'cognitive cerebellum' hypothesis, that the neocerebellum is centrally involved in cognitive skill acquisition: *The mammalian cerebellum seems able to improve the skilled performance of any cerebral area to which it is linked by 2-way neural connections . . . the 2-way connections linking the cerebellum to Broca's area make it possible for the cerebellum to improve language dexterity, which combines motor and mental skills* (p. 1007). It should be noted that Glickstein (1993) has rightly cautioned that there is insufficient hard evidence that the cerebellum is involved in execution of all cognitive skills. An important point here is that, whether or not the final procedural code is stored in the cerebellum, the cerebellum can 'scaffold' the development of a cognitive skill by using its in-built timing and error analysis machinery (Ito, 1990). An analogous process may occur for the acquisition of declarative knowledge, with the hippocampus acting as a sort of scratch pad and trainer for the modification of semantic memory in the neocortex (McClelland, McNaughton and O'Reilly, 1995).

For our purposes the critical issue is the involvement of the cerebellum in language skills. Here the case is an interesting one. There is no doubt that the cerebellum is a key structure in the development of articulatory skill—articulation is of course a motor skill, a skill requiring exquisite timing and fluency. Infants spend around 18 months learning to articulate their first word, and it is arguably this additional requirement that has caused the huge development of the neocerebellum in humans (Passingham, 1975). It is significant in this context that in phase 2 of our research programme we established that there was a highly significant deficit in articulation time for the children with dyslexia, with the worst deficits (mean effect size -3.4—the most severe mean impairment for this group) for the youngest children. It is accepted that articulatory skills play a key role in language development and, in particular, the development of phonemic awareness (Locke, 1983). From here, therefore, the

cerebellar impairment hypothesis subsumes the mechanisms proposed by advocates of the phonological deficit hypothesis.

Interestingly, however, the cerebellar impairment hypothesis also accounts naturally for the difficulties in spelling and handwriting, which have always proved somewhat troublesome to a pure phonological deficit hypothesis. Poor handwriting quality follows naturally from the expectation of reduced motor skill. Poor spelling is attributable not only to reduced ability to acquire implicit knowledge of orthographic regularities, but also to a reduced ability to automatize knowledge of spelling patterns. It may be that this twin deficit route accounts for the more severe difficulties with spelling than with reading (Thomson, 1984). The account is developed at greater lengths below.

Requirements for a Causal Explanation of Dyslexia

An early *a priori* analysis of the requirements for any 'process deficit' theory of dyslexia was provided by Morrison and Manis (1983). These authors suggested that any viable theory must address four issues: why does the deficit affect primarily the task of reading; why do children with dyslexia perform adequately on other tasks; what is the mechanism by which the deficit results in the reading problems; and what is the direction of causality?

We show below that the cerebellar impairment hypothesis fulfils all four requirements. We also believe that it is a truly explanatory theory. In a recent analysis, Seidenberg (1993, p. 231) argues that one important requirement for an explanatory theory is that it should *explain phenomena in terms of independently motivated principles*. This distinguishes *ad hoc* descriptive theories, such as Mendeleyev's original pattern-based hypothesis for the atomic table, from truly explanatory theories, such as the subsequent atomic weights explanation of the pattern. A further important criterion introduced by Seidenberg (1993, p. 233) is that *an explanatory theory shows how phenomena previously thought to be unrelated actually derive from a common underlying source*.

In terms of the four Morrison/Manis criteria the account is as follows: (i) deficits appear specific to reading-related processes, first because reading is a complex task requiring good automatisation and blending of its component skills, and second because reading-related processes are of the most significance educationally and thus have been most carefully studied; (ii) although in many domains performance will appear normal (owing to conscious compensation), more careful study may well reveal subtle deficits in fluency; (iii) it is the cerebellar impairments that cause the reading problems, rather than *vice versa*. Finally, as discussed in the next paragraph, the cerebellar impairment hypothesis provides a complete mechanism for the causal chain (iv), as outlined in Figure 1, starting from the known function of the neural substrate (the cerebellum) and moving through the intervening cognitive processes (primarily phonemic awareness, but also other factors such as automatisation deficits for acquired letter knowledge), explaining the full range of reading-related deficits suffered by children with dyslexia (reading, writing and spelling), together with other symptoms not directly related to reading. The above explanation satisfies Seidenberg's first criterion for explanatory theories, namely explanation in terms of independently motivated principles. It also meets his second criterion extremely well, accommodating a range of apparently disparate deficits within a unitary framework.

Figure 1 outlines the hypothetical ontogenetic causal chain linking cerebellar problems, phonological difficulties and eventual reading problems. If an infant has a

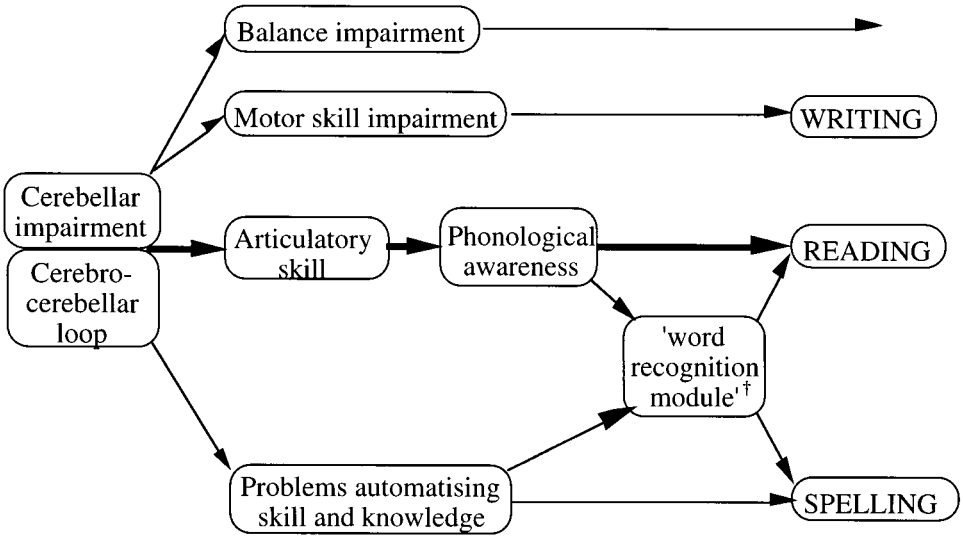

Figure 1. Proposed causal chain for the cerebellum and reading.

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 and co-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, p. 189) 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 co-articulated gestures become grouped into the representations of phonemes. Even after speech and walking emerge, one might expect that the skills would be less fluent, less 'dextrous', in infants with cerebellar impairment. If articulation is less fluent than normal, then it takes up more conscious resources, leaving fewer resources 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, rhyme and the phonemic structure of laguage—in short, one would expect early deficits in phonological awareness (see Snowling and Hulme, 1994, for a related account).

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 which, like articulation,

requires precise timing and co-ordination of diverse muscle groups. It may be that one reason that spelling, the third criterial skill, appears particularly resistant to remediation (Thomson, 1984) is that it requires the simultaneous use both of phonological skill and of motor output. Perhaps one of the major reasons for the success of computer-based support for reading and spelling (e.g. Nicolson and Fawcett, 1994d; Wise and Olson, 1995) is that it relieves the student of the motor writing task, leaving capacity free to focus on the spelling or reading itself.

Dyslexia and High Achievement

One of the key theoretical concerns of dyslexia researchers has been the discrepancy between the low reading performance and good intellectual functioning of children with dyslexia. Indeed, there is evidence that adults with dyslexia may be among the most creative and successful of their generation (West, 1991). How can this be explained in the light of cerebellar impairment which apparently causes significant difficulties with acquisition of skills and with linguistic skill? We believe that the resolution of this paradox lies in the problematic but undoubtedly real distinction between declarative and procedural knowledge; between explicit and implicit knowledge; and between explicit and implicit learning (e.g. Squire, Knowlton and Musen, 1993). The cerebellar impairment hypothesis suggests that children with dyslexia will have difficulties specifically with the procedural, implicit learning mediated by the cerebellum. There is no reason to expect difficulties in explicit learning and reasoning, which are mediated via the hippocampus and frontal lobes (McClelland, McNaughton and O'Reilly, 1995). Reasoning ability is not dependent upon fluency. In Sternberg's (1988) triarchic theory of intelligence, at the top level he identifies three types of thinking: analytic, creative and practical. None of these depends directly upon skill or fluency. Indeed, fluency may well be the enemy of creativity—trying to solve new sorts of problems that require thinking about the problem and its elements in a different way—in that fluency is in essence the ability to repeat previous actions or thoughts more and more quickly.

Many children and adults with dyslexia will unfortunately never overcome the associated difficulties—neither the primary difficulties in terms of skill deficits, nor the secondary ones in terms of motivation and reduced access to print. Those who do succeed, however, will have done so by developing to abnormal lengths their explicit knowledge and creative skills, maybe in much the same way that a partially sighted person develops abnormally good auditory skills. Furthermore, adults with dyslexia often have a burning desire to achieve, brought on in no small part by the adverse conditions of their childhood.

Conclusions

In summary, recent research on primitive skills (Nicolson and Fawcett, 1994b, 1995) has shown that children with dyslexia have deficits in phonological skill, naming speed, motor skill and balance. These deficits may be characterized as problems in skill automatisation that are normally masked by the process of conscious compensation (Nicolson and Fawcett, 1990). However, to describe the symptoms as a 'general automatisation deficit' explains neither the cause nor the specific pattern of difficulties. Its involvement in the above skills and in skill automatisation 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 in clinical tests of co-ordination and muscle tone. It provides a natural and principled explanation (in terms of impaired articulatory fluency) for the existence of phonological deficits; it explains difficulties in handwriting in terms of limitations on motor skill; and it explains difficulties in reading and spelling in terms of reduced phonological awareness and difficulties in automatizing the underlying sub-skills, together with difficulties in error elimination.

Even so, the evidence cited here is still indirect. It would be premature to assign difficulties to the cerebellum alone owing to its rich interconnections with cerebral cortex and thalamic nuclei. Studies of neuroanatomical structure in the cerebellum and cerebellar circuits (Galaburda, Rosen and Sherman, 1989), together with brain imaging work focused on cerebellar activation, might lead to fuller 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 more complete understanding of the role of the cerebellum in normal skill acquisition. Nonetheless, the elegance and parsimony with which the cerebellar impairment hypothesis accounts for the range of known problems (and previously unsuspected problems) in dyslexia lead us to believe that cerebellar impairments must be centrally involved.

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