

Dyslexia: the role of the cerebellum

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Abstract

Introduction: In this review article we outline the thinking and evidence behind our hypothesis that the problems suffered by dyslexic people may be attributable to cerebellar deficit.

Method: Firstly, we provide an overview of recent evidence that proposes a central role for the cerebellum in cognitive skills, in particular those scaffolded by spoken language, in addition to its well-recognised role in motor skills. Secondly, we outline evidence from our laboratory that cerebellar function is abnormal in dyslexia.

Results: We consider two specific lines of evidence: behavioral, and converging evidence from neuroimaging, which demonstrate significant differences between the dyslexic and control groups. We also apply the same battery of behavioural tests to a group of children who are non-discrepant poor readers.

Discussion: Finally, we provide an ontogenetic causal chain for the development of dyslexia in terms of cerebellar deficit from birth, considering the implications of this framework for the key questions in dyslexia research.

Keywords: dyslexia, cerebellum, phonology, causal-chain

Introduction

Developmental dyslexia is the most prevalent and the most researched of the developmental disorders. The bulk of research in the past decade has investigated two alternative approaches, the phonological deficit hypotheses and the magnocellular deficit hypotheses. However, despite extensive research, these approaches have failed to converge to an agreed theoretical framework. In our laboratory, we have tried to discover the underlying cause(s) of dyslexia adopting a learning perspective. We argued that, unlike language, reading is not a human skill which has evolved, and therefore we need to understand the learning processes in order to find out why dyslexic children fail to learn to read. Automatisation is not a conscious process – by dint of practice under reasonably consistent conditions most humans just ‘pick up’ skills. Automatisation therefore gave an intuitively satisfying account not only of the reading problems but also of the phonological difficulties (because phonological awareness is a skill that is picked up initially just by listening to one’s own language). What was not clear was WHY dyslexic children have problems in skill automatisation, and for this we looked for an explanation at the brain level. We proposed the cerebellar deficit hypothesis (CDH) – that cerebellar abnormality was a cause of the difficulties suffered by dyslexic children. In this article we review our fifteen year research programme, and the evidence for the CDH.

We have argued that one of the intriguing aspects of dyslexia research is that, whatever one’s interest as a researcher – reading, phonology, writing, spelling, education, memory, speed, creativity, hearing, vision, balance, learning, skill, genetics, brain structure or brain function – dyslexic children will show interesting and unusual differences in that domain. Given the need for specialization in science, many researchers have gone on to undertake incisive and insightful studies in their specific domain of expertise. This explains why, on the one hand, there is an unrivalled wealth of research on dyslexia, and why, on the other hand, the research area fails to cumulate, to build towards a ‘grand’ theory of dyslexia. In an analogy much loved by psychologists, it is like the Hindu fable of the four blind men attempting to describe an elephant. One touches the trunk, another the leg, another the tail, another the side, leading to descriptions of ‘a pipe’, ‘a tree’, ‘a house’ and ‘a rope’ respectively. If one wants to describe the whole elephant, one needs a range of perspectives. Let us start the tour of the elephant by identifying some potent causes of confusion in the area.

In previous papers, Nicolson has outlined the different motivations of different researchers within the Dyslexia Ecosystem (Nicolson,2002), which lead to a major source of confusion in dyslexia research. Many applied theoreticians are naturally concerned with educational attainment, and in particular literacy. Consequently, they analyze the different components of reading, investigate the differential effects of various interventions, and often stress (correctly) the need for support for any child who is a risk of reading failure, whether or not they are dyslexic. By contrast ‘pure’ theorists are interested primarily in the underlying cause(s) of dyslexia (rather than literacy per se) and so they undertake theoretically-motivated tests, often in domains not directly related to literacy. In most areas of science the distinction between cause, symptoms and treatment is clearcut – in medicine for instance, the causes, symptoms and treatment of, say, malaria are quite different. Indeed, several diseases may have similar symptoms. Influenza and meningitis may lead to symptoms of fever, aching and nausea similar to those of malaria, but of course the underlying causes (and treatments) are quite different. In dyslexia, this distinction is much less clearcut but it is therefore particularly important to maintain the distinctions between cause, symptom and treatment

In recent papers, we have noted our hope that several subtypes of dyslexia might be identified over the next 5 years, each based on a different brain region, but each leading to core phonological difficulties. These may be linked to further and more distinctive symptoms (visual, auditory, motor, speed difficulties etc) in line with current theories of dyslexia. Considerable advances have been made in examining potential overlaps, which we return to in the later part of this article. Work in progress is revealing overlaps between specific types of dyslexia and other disorders, including ADHD, specific language impairment, dyspraxia, and generalised learning disability. We would hope that the identification of specific underlying causes might then lead to the specification of the most appropriate intervention strategies for a particular child, in addition to alleviating the reading symptoms. Above all, if a wider range of precursors can be identified, we should be able to provide proactive support before children fail, to cut into the cycle of failure for all children with special educational needs. This remains the applied challenge for pure theorists.

We have now made a case for the need for pure theoretical research in order to identify the underlying cause(s) of dyslexia, but what would we need for a causal theory, and specifically a causal theory of dyslexia? Typically scientific explanation move from descriptive to explanatory theories, which are based on a good description of the symptoms, and specifi-

cation of the neurological underpin respectively. Similarly, Morton & Frith (1995) distinguish three levels of explanation – biological, cognitive and behavioural, with the biological level the deepest level of explanation. In our view, an adequate framework for dyslexia must address the following key questions: What is the underlying cause of dyslexia?; Why does it appear to be specific to reading?; Why do weaknesses appear to be limited to reading?; and finally, given the wide range of difficulties outlined above, why are there so many high achieving people with dyslexia?

Before addressing these issues, it is important to consider the expanding role of the cerebellum, which until recently has been largely overlooked.

The Cerebellum

The cerebellum is a very densely packed and deeply folded subcortical brain structure situated at the back of the brain, sometimes known as the ‘hind-brain’ (Holmes, 1939). In humans, it accounts for 10-15% of brain weight, 40% of brain surface area, and 50% of the brain’s neurons.

Damage to different parts of the cerebellum can lead to different symptoms, including disturbances in posture and balance, limb rigidity and dyscoordination or decomposition of movement (that is, previously coordinated sequences of movements, such as picking up a cup, may break down into a series of separate movements). However, one of the features of cerebellar damage is the great plasticity of the system. Typically normal or close to normal performance is attained again within a few months of the initial damage (Holmes, 1922).

The proposed involvement of the cerebellum in cognitive skills led to considerable controversy in the field, in that the cerebellum had traditionally been considered as a motor area (Eccles, Ito & Szentagothai, 1967; Holmes, 1917; Holmes, 1939; Stein & Glickstein, 1992), and it is also claimed to be involved in the automatising of motor skill and in adaptive learning control via the cerebellar structures (Ito, 1984; Ito, 1990; Jenkins, Brooks, Nixon, Frackowiak & Passingham, 1994; Krupa, Thompson & Thompson, 1993). However, as Leiner, Leiner & 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 et al., 1989; Leiner, Leiner & Dow, 1991;

Leiner, Leiner & Dow, 1993) concluded that the cerebellum is therefore central for the acquisition of 'language dexterity'. In effect, then, they proposed that the cerebellum is critically involved in the automatization of any skill, whether motor or cognitive. There remains controversy over the role of the cerebellum in cognitive skills not involving speech or 'inner speech' (Ackermann, Wildgruber, Daum & Grodd, 1998; Glickstein, 1993), but there is now overwhelming evidence of the importance of the cerebellum in language (Ackermann & Hertrich, 2000; Fabbro, Moretti & Bava, 2000; Silveri & Misciagna, 2000), speech perception (Mathiak, Hertrich, Grodd and Ackermann, 2002) including a recent demonstration of specific cerebellar involvement in reading (Fulbright et al., 1999). It has now even been demonstrated that patients with cerebellar damage show deficits in attention and working memory (Malm et al. 1998) and dyslexic type symptoms in reading (Moretti et al., 2002).

Dyslexia and the Cerebellum

Let us now return to dyslexia, bearing in mind the increasing overlap between what is known about cerebellar deficits and the symptoms of dyslexia.

The Sheffield Dyslexia Research Programme

In our approach to dyslexia, the Sheffield group have been unusual amongst dyslexia researchers in adopting a learning and skills perspective. The distinctive strength of the automatization hypothesis was that it was also consistent with the outcome of a series of studies in the early 1990s, in which we investigated a range of skills outside the literacy domain, and found that our panel of dyslexic children showed severe deficits in a range of skills. These included balance (Fawcett & Nicolson, 1992; Nicolson & Fawcett, 1990); - see also Yap & van der Leij (1994); motor skill (Fawcett & Nicolson, 1995b) - see also Daum et al. (1993), rapid processing (Fawcett & Nicolson, 1994; Nicolson & Fawcett, 1994). Furthermore, taking all the data together (Nicolson & Fawcett, 1995a; Nicolson & Fawcett, 1995b), the majority of (individual) dyslexic children showed problems 'across the board', rather than with different children showing different profiles, as would be expected if there were a range of sub-types (Boder, 1973; Castles & Holmes, 1996). The automatization deficit therefore provided an excellent account of the range of symptoms of dyslexia, but it did not specify an

underlying neurological structure. In subsequent research we subsumed this ‘cognitive level’ hypothesis within the ‘neurological level’ hypothesis of cerebellar deficit, as outlined below.

The Cerebellar Deficit Hypothesis

As noted earlier, deficits in motor skill and automatised point clearly to the cerebellum. However, early findings by Levinson (Frank & Levinson, 1973; Levinson, 1988) arguing for mild cerebellar impairment were largely discounted owing to shortcomings in research methodology (Silver, 1987), allied to the belief that the cerebellum was not involved in language-related skills. Furthermore, the hypothesis falls foul of the ‘assumption of specificity’. If there are indeed problems in the cerebellum, why are the major symptoms specific to the reading domain?.

Method

Subjects

In our attempts to address these issues, we worked with our panel of ‘pure’ dyslexic children with IQ over 90, and reading age at least 18 months behind their chronological age, with no sign of ADHD, and no significant emotional or behavioural problems, and a control group from a similar social background, matched for age and IQ. Subjects with dyslexia satisfied both of the two standard exclusionary criteria for dyslexia. Three age groups of children with dyslexia participated, together with three groups of normally-achieving children. It should be noted, 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 Nicholson, 1995a; 1995b; Nicholson and Fawcett, 1994a; Nicholson, Fawcett and Dean, 1995). Subjects were paid around 5 Euros per hour and participated with fully informed consent.

Clinical tests of cerebellar function

Traditional symptoms of cerebellar impairment are dystonia (problems with muscle tone) and ataxia (disturbance in posture, gait, or limb movements). If there is a cerebellar im-

pairment, dyslexic children should also show these traditional signs of cerebellar dysfunction (see Holmes, 1917, 1939; and Dow and Moruzzi, 1958). Consequently, we replicated the tests described in Dow and Moruzzi (1958), using groups of children with dyslexia and matched controls aged 18, 14 and 10 years (see Fawcett, Nicolson and Dean, 1996). Tasks fell into three types; posture and muscle tone; hypotonia of the upper limbs; and complex voluntary movement, a total of 14 tasks in all. The performance of the children with dyslexia was significantly worse than that of the chronological age controls on all 14 tasks, and significantly worse than reading age controls on 11 out of the 14 tests. Using age-appropriate 'effect size' in standard deviation units (analogous to a z-score) for each test for each child (e.g., Cohen, 1969), we estimated the severity and incidence of deficit on each task. Children were deemed 'at risk' if their performance fell one standard deviation or more below that expected for their age, and deficits of this size or greater were found on all but one of the tasks, with several of the cerebellar tasks showing deficits larger than reading. These results were not confined to the dyslexic children in our panel, but also found with a further sample of 126 children drawn from private schools specialising in dyslexia. The sample included dyslexic and control children, aged 8-16, divided into four age groups. We administered both a range of cerebellar tasks and other tasks sensitive to dyslexia. In all the cerebellar tests, together with segmentation and nonsense word repetition, the performance of the dyslexic children was significantly worse than controls. The effect size analyses are also similar to the panel study. In line with the earlier study, comparing dyslexic children 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.

Cerebellar function in slow learners

Much of the recent research from the USA has suggested that there is no point in differentiating between children with low IQ and children with dyslexia, because they both show problems with phonological skills. This proposition has strong implications for dyslexia research, and has aroused considerable controversy in the field, with US researchers such as Stanovich (1988) and the Shaywitzes advocating that groups of poor readers should be collapsed, and UK researchers such as Nicolson (1999) and Snowling et al (2003) advocating that the distinction between the groups be maintained. Albeit for different reasons, both Nicolson and Snowling independently have advocated that concentration on phonological

skills alone is too narrow, with Nicholson advocating testing broader skills, and Snowling advocating wider testing of language and reading related skills. In order to test the hypothesis that it is worthwhile to maintain the distinction between dyslexic and slow learning children in research, a comprehensive test battery, including phonological, speed, motor and cerebellar tasks, was administered to the entire cohort of two schools for children with learning disabilities. Testing was undertaken 'blind' without accessing the psychometric data on the children. Children were then allocated to a 'discrepancy' group on the basis of their IQ, with the majority (n=29) classed as 'non-discrepant' (IQ<90) and a smaller set (n=7), with IQ at least 90, classed as 'discrepant' (with dyslexia). Both groups showed significant deficits relative to age-matched controls on almost all the tests. On phonological, speed and motor tasks the non-discrepant group were at least as severely impaired as the discrepant group. By contrast, on the cerebellar tests of postural stability and muscle tone the non-discrepant group performed significantly better than the children with dyslexia, and close to the level of the controls. The findings indicate that cerebellar tests may prove a valuable method of differentiating between poor readers with and without IQ discrepancy.

The study outlined here was intended to establish whether poor readers with IQ discrepancy (children with dyslexia) can be distinguished from poor readers with no discrepancy (ND-PR), using a range of tests of skills known to be impaired in children with dyslexia. A dissociation was established between the groups with dyslexia and those with ND-PR. In this study, the cerebellar tests were split into 'static' and 'dynamic' linked to dystonia and ataxia respectively. Basically the static tests involved stability and muscle tone in response to perturbation by the experimenter, whereas the dynamic tests involved self generated speed of performance of simple and complex movements. The children with ND-PR performed at near-normal levels on static cerebellar tests and were significantly better than children with dyslexia on these tests. By contrast, children with ND-PR showed problems equivalent to, or significantly greater than, children with dyslexia on dynamic speeded tests, on phonological and verbal memory tests, and on speed of processing tests. The findings provide evidence of the generality of phonological and speed deficits in both ND-PR and dyslexia, compared with the specificity of static cerebellar tests of muscle tone and stability deficits in dyslexia.

In conclusion, from a theoretical viewpoint, this study suggests that there are differences between the phenotypes of children with dyslexia and children with more generalized learning difficulties. Although we may well expect some overlap between the two groups,

these results suggest that the majority of children with dyslexia suffer from a mild 'cerebellar' abnormality in static tests, whereas the majority of children with ND-PR do not. Naturally enough, these results need to be replicated with further groups of children with dyslexia and groups with ND-PR. The dissociation between cerebellar tests and phonological tests for these groups provides further strong support for the cerebellar deficit hypothesis (Nicolson, Fawcett & Dean, 1995). Furthermore, regardless of the specific interpretation made, the dissociation obtained in the present study between children with ND-PR and children with dyslexia demonstrates that there are indeed theoretically valid reasons for distinguishing between poor readers with discrepancy and those without.

Direct tests of cerebellar anatomy and function

Space precludes a full account of the neuroanatomy of the cerebellum (Finch et al, 2002), the PET studies of brain activation in motor learning (Nicolson et al, 1999), and the eye-blink conditioning study (Nicolson et al, 2002). For a review of this work see the Trends in Neuroscience debate, in Nicolson et al, 2001. Most strikingly, from the PET study, the dyslexic adults showed only 10% the level of increased blood flow found in the controls in cerebellar cortex and vermis when performing the tasks. These results are highly significant and would not be predicted by any other theory of dyslexia. They provide direct evidence that the behavioural signs of cerebellar abnormality do indeed reflect underlying abnormalities in cerebellar activation.

Discussion

Let us now consider the implications of a cerebellar deficit for the understanding of dyslexia, for how dyslexia develops, and for future work in the area.

Let us start by summarising the evidence to date. Our behavioural studies showed that a common symptom of performance in dyslexic children is that it is less well automatised, not only for literacy but also for all the other tasks studied. The well-established role of the cerebellum in skill learning and automatised tasks made it a good candidate for investigation, particularly when coupled with evidence from cognitive neuroscience on the central role of the cerebellum in language-related cognitive tasks. We demonstrated not only that our panel of

dyslexic children showed clinical symptoms of cerebellar abnormality, but also that these symptoms characterised a much larger group of dyslexic children.

We went on to demonstrate that it is worthwhile to maintain the distinction between children with dyslexia and children with non-dyslexic reading difficulties, that is children with more generalised difficulties. The same pattern of impairments was found in both groups for phonology, speed and dynamic cerebellar tests. However, the dyslexic group showed significantly greater deficits in static cerebellar tasks, involving postural stability and muscle tone. Interestingly enough, many research groups adopt a broader classification of dyslexia than the one used in Sheffield, including children with an IQ of 80 or above, by contrast with the cutoff at 90 adopted in the research here. This means that interpretation of the results may be clouded by the presence of non-discrepant poor readers within the dyslexic group. This suggests that the findings of any research project may be significantly different depending on the parameters adopted for group classification.

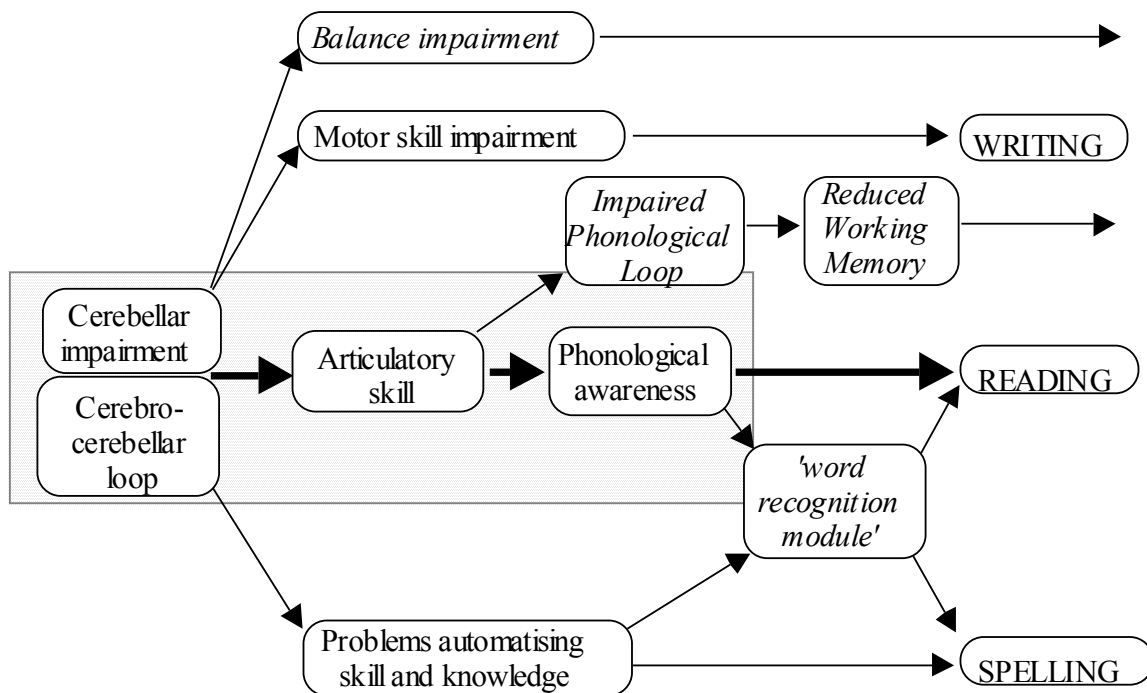
Our PET study of motor sequence learning showed that there were indeed abnormalities in cerebellar activation in automatic processing and in new learning, for subjects in our panel. Given that the dyslexic subjects whom we had scanned also showed classic clinical signs of cerebellar deficit, this demonstration that the dyslexic group really did have abnormal use of the cerebellum, in turn lends greater strength to our previous findings that around 80% of dyslexic children show clinical signs of cerebellar abnormality.

Furthermore, in the research reported here, we are beginning to unpick the locus of the problems within the cerebellum.. The dissociation between static and dynamic cerebellar tests for these ND-PR groups may indicate that the abnormalities for the children with dyslexia lie within the lateral parts of the posterior lobe of the cerebellum, in that lesions in this area are often associated (Holmes, 1922) with dysmetria (inaccurate limb movement) and hypotonia (low muscle tone). These findings are particularly interesting in view of the recent PET findings of abnormal activation patterns in the ipsilateral posterior lobe of the cerebellum of adults with dyslexia both when executing a previously overlearned motor sequence task, and when learning a new motor sequence (Nicholson et al., 1999).

Toward a causal explanation

We are now able to start filling in the blanks in an ontogenetic causal chain. Note the impact of these difficulties in learning on working memory, which is clearly impaired in dyslexia.

Figure 1 Dyslexia: an ontogenetic causal chain



In Figure 1 (from Nicolson & Fawcett, 1999) the hypothetical ontogenetic causal chain between cerebellar problems, phonological difficulties and eventual reading problems is outlined, accounting for the three criterial difficulties, writing, reading and spelling. Dyslexic children frequently show poor quality handwriting, which has been hard to explain under other theories, but can be handled naturally by the CDH as a motor skill requiring precise timing and co-ordination of the muscles. Although literacy difficulties arise from several routes, the central route is highlighted as the most important. 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. These problems may not seem too serious, unless we appreciate that our most complex motor skill is articulation, and consequently, the infant might be slower to start babbling, (see e.g., Ejiri and Mastaka, 2001

for evidence relating babbling to motor control), and, later, talking (cf. Bates & Dick, 2002). Once speech and walking develop, skills may be less fluent, less ‘dextrous’, in infants with cerebellar impairment. If articulation is less fluent than normal, it takes up more conscious resources, leaving fewer resources to process sensory feedback. Processing the auditory, phonemic structure of spoken words may be less complete, leading to loss of awareness of onset, rime, and the phonemic structure of language – (see Snowling & Hulme, 1994). Cerebellar impairment would therefore be predicted to cause the ‘phonological core deficit’ that has proved such a fruitful explanatory framework for dyslexia. Based on this framework, standard explanations of reading deficits apply, coupled with problems in learning and automatisisation, which lead to impaired fluency and speed of reading, or the double deficit hypothesis (Wolf & Bowers, 1999). One of the keys to fluent reading is the ability to articulate sub-vocally, and the cerebellum is known to be activated in internal speech (Thach, 1996). The third criterial skill, Spelling may be the most resistant to remediation based on a combination of over-effortful reading, poor phonological awareness, and difficulties in automatising skills and eliminating errors, as well as the simultaneous use of both phonological and motor skills (Thomson, 1984).

This brings us back to the key questions in dyslexia research. We have already addressed the mechanism and direction of causality, and suggest that difficulties appear to be specific to reading and spelling because they involve a combination of phonological skills, fluency, automatisisation, and multi-tasking – a combination of all the skills that dyslexic children find difficult. Why does performance appear to be normal in other skills? Because literacy is of such critical educational importance, it is examined minutely, where other skills are largely overlooked. Moreover many skills are unimpaired or even overcompensating, because skills can be acquired without much cerebellar involvement, they simply demand more conscious ‘frontal’ involvement – precisely the pattern shown in our sequence learning task. Lack of automaticity is only a real problem if rapid processing or multi-tasking is required, because most skills including ‘intellectual’ skills require frontal involvement – thinking rather than rote learning. This brings us back to an explanation for the discrepancy between the low reading performance and good intellectual functioning of children with dyslexia. There is suggestive 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? Reasoning ability is not dependent upon 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 without conscious thought.

Interpretations in terms of alternative hypotheses

We have suggested that the CDH framework naturally subsumes the phonological deficit and that the double deficit hypothesis (Wolf & Bowers, 1999) may also be accounted for in a similar manner, given that the double deficit hypothesis is a cognitive level description. However, the key question remains, why do children become faster as they mature? It seems likely that this reflects improved efficiency of the central processing mechanisms in which the cerebellum will be centrally involved

Stein (e.g., Stein & Walsh, 1997) has argued that cerebellar impairment might be attributable to faulty input via impaired magnocellular pathways. It seems clear that there is a sub-type of dyslexia with magnocellular impairment and Tallal (Tallal, Miller & Fitch, 1993) has suggested that there may be a pan-sensory impairment, including motor output as well as visual and auditory input. Stein notes that there are magnocells in the cerebellum and in the motor output systems, which make it difficult to distinguish these theories from the CDH. However, if one limits the magnocellular deficit hypotheses to the sensory input stage it is not clear why dyslexic children have problems in detecting rhymes, which do not involve rapid processing. From our own work, there is no obvious magnocellular explanation for normal speed of simple reactions, with the same response slowed when a choice needs to be made (Nicolson & Fawcett, 1994); no explanation for difficulties in time estimation, lowered muscle tone or no abnormal cerebellar activation in the motor sequence learning task. Future research may reveal a ‘magnocellular’ sub-type, a ‘cerebellar’ sub-type, and various ‘mixed’ sub-types.

Wider Research on the cerebellum and dyslexia

Since the CDH was first introduced, the role of the cerebellum in reading and cognitive processes has moved from controversy to orthodoxy, with researchers in cognitive neuroscience trying to locate the areas of the cerebellum involved in language. Recent findings

include activation of the cerebellum in non-motor mental operations (Hanakawa et al, 2002) or even generating antonyms, in the absence of mental movement (Gebhart et al, 2002). One meta-analysis (Turkeltaub et al., 2002) concluded that in reading single words aloud the cerebellum was reliably activated. However, in a recent metanalysis of 35 neuroimaging studies of the dual route to reading, it was found that although regions such as the cerebellum have been reliably found activated, they are rarely discussed (Jobard, Crivello and Tzourio-Mazoyer, 2003 in press). Most excitingly, perhaps, for the cerebellar deficit hypothesis, Bower and Parsons (2003) have used their investigations of touch to derive a new hypothesis for the role of the cerebellum in sensory processing.

Recent research into dyslexia from other groups has investigated both the cerebellar deficit and the sensory processing deficit. Stein's group compared the metabolism of the cerebellum in dyslexics with controls' using magnetic resonance spectroscopy (MRS). They found a lower ratio in the cerebellum of the dyslexics compared with the controls, particularly on the right hand side (Rae et al. 1998). More recently, a further study from Rae et al, (2002), used imaging to show cerebellar symmetry in dyslexics but not controls. Rae et al argued that the relationship of cerebellar asymmetry to phonological decoding ability and handedness, together with their previous finding of altered metabolite ratios in the cerebellum of dyslexics, suggests that there are alterations in the neurological organisation of the cerebellum which relate to phonological decoding skills, in addition to motor skills and handedness.

Evidence has been published from other countries, such as Norway, on the incidence of balance and gait deficits linked to the cerebellum in children with dyslexia (Moe-Nilssen et al, 2003) using an accelerometer to measure posture more accurately. Similarly, a series of studies have been run using the polhemus, a device for measuring the position of limbs in 3D space, with preliminary results indicating significant differences between dyslexics and controls. Studies of implicit learning in Italy have identified deficits in dyslexic children, which have been linked to the cerebellum (Vicari et al, 2003). Shifting attention has been examined most notably by Moores et al, (2003) from the Sheffield lab, but also by Facoetti et al, 2003), In terms of activation of the brain, imaging revealed significantly smaller right anterior lobes in dyslexic subjects, correlated with deficits in reading, spelling and language

associated with dyslexia. Further, individuals with dyslexia could be distinguished from controls based on the volume of the right anterior lobe of the cerebellum (Eckert et al, 2002). Furthermore, there have been a series of studies of eye blink conditioning, which is known to be mediated by the cerebellum, showing impairments in dyslexia, (Coffin et al, 2003). All these studies provide strong support for the cerebellar deficit hypothesis of dyslexia.

Nevertheless, there have been studies of the cerebellar deficit, which have reached less positive conclusions, including the work of Wimmer (Wimmer et al, 1999), which concludes that ADHD children are the most impaired on cerebellar skills. However, it is likely that German speaking dyslexics are different from English dyslexics, because they show only rate not accuracy deficits. Further work is in progress with children with ADHD, but currently there is a dearth of published studies examining children with different diagnoses on the same battery of tests. Studies by Ramus and colleagues have identified significant differences between dyslexic and control children on balance and other cerebellar tasks, although no significant differences were found on tests of time estimation (Ramus et al, 2003a and b).

Conclusions

Both a strength and a limitation of the Sheffield research is that we have worked with ‘pure’ dyslexic children, deliberately excluding borderline dyslexic children, or comorbid ADHD/dyslexia children. We included all dyslexic children who met our criteria (and were willing to participate), thus avoiding any selection bias. However, this meant that our groups were small and the results may not generalise completely to further groups. It is clearly a priority to establish the prevalence of cerebellar symptoms in larger populations of dyslexic children (and comorbid groups).

It is gratifying to note that this research is now in progress, with the Sheffield group working with children with generalised learning difficulties, ADHD and dyspraxia, as well as dyslexia, and other research groups examining broader aspects of performance in children with a spectrum of difficulties.

In conclusion, the cerebellar deficit hypothesis is a biological-level hypothesis that is well described at the cognitive level as an automatization deficit hypothesis. The two hypotheses between them have provided a true causal explanation of the varied findings in dyslexia research. No doubt further research will reveal that the story is not yet complete, but meanwhile the CDH generates a number of new and interesting avenues for dyslexia research. One exciting direction for further research is the dissociation between the findings for dyslexic and non-discrepant poor readers on static and dynamic cerebellar tests, which may begin to resolve one of the more controversial aspects of dyslexia research.

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