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Authors

Nicolson, Roderick I.
Fawcett, Angela J.

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Toward the origins of dyslexia

Roderick I. Nicolson and Angela J. Fawcett
Department of Psychology
University of Sheffield
Sheffield S10 2TN, England
email: R.NICOLSON@UK.AC.SHEFFIELD.PRIMEA

Abstract

A series of experiments will be reported comparing the performance of groups of 11 year old and 15 year old children with dyslexia with groups of normal children matched for chronological age (CA) and reading age (RA) respectively. Experiments testing gross motor skill demonstrated that both groups of children with dyslexia showed significant deficits in balance when required to undertake a further task at the same time, whereas the control groups were not affected. It was concluded that the control children balanced automatically whereas the children with dyslexia did not. Further experiments indicated that working memory performance was not disordered, in that deficits in memory span were paralleled by deficits in speed of articulation. Tests of information processing speed led to an interesting dissociation. Simple reaction performance was indistinguishable from that of the CA controls. By contrast, on the simplest possible choice reaction, both groups of children with dyslexia were slowed to the level of their RA controls. It was concluded that the locus of the speed deficit lay within the decision-making process. Further experiments demonstrating deficits in sensory thresholds and abnormal evoked potentials will also be reported. We conclude that an automatisation deficit is consistent with most of the known problems of children with dyslexia.

Introduction

Developmental dyslexia is conventionally defined as "a disorder in children who, despite conventional classroom experience, fail to attain the language skills of reading, writing and spelling commensurate with their intellectual abilities" (World Federation of Neurology, 1968). A typical estimate of the prevalence of dyslexia in Western school populations is 5% (Badian, 1984; Jorm et al., 1986), with roughly four times as many boys as girls being

diagnosed. It has been assumed that the problems of children with dyslexia derive from impairment of some skill or cognitive component largely specific to the reading process, and the consensus view (e.g., Stanovich, 1988) is still that the deficits are attributable to some disorder of phonological processing.

Interestingly, however, dyslexia has caught the imagination of researchers from several disciplines, and there is now a wealth of inter-disciplinary information about dyslexia (though inconsistencies of diagnosis bedevil meta-studies of the literature). Frustratingly, different perspectives on dyslexia have led to quite different hypotheses. Neuroanatomical studies by Galaburda and his colleagues have identified both "a uniform absence of left-right asymmetry in the language area and focal dysgenesis referrable to midgestation ... possibly having widespread cytoarchitectonic and connectional repercussions. ... Both types of changes in the male brains are associated with increased numbers of neurons and connections and qualitatively different patterns of cellular architecture and connections" (Galaburda, Rosen & Sherman, 1989, p383). Genetic studies (e.g., Smith et al., 1983) have led to the conclusion that there is a strong genetic component. Studies mapping gross electrical activity in the brain have also uncovered anomalies in processing (e.g., Duffy et al., 1980; Hynd et al., 1990).

At present, there is little or no link between the genetic, the anatomical, the neurological and the cognitive approaches to dyslexia. The 'Holy Grail' of dyslexia research is surely to establish such a link. Not only would such a link between brain and mind prove a breakthrough in dyslexia research, it seems likely that the insights gained would provide a rich source of ideas for modelling normal cognition, bequeathing a research agenda stretching into the next century. This quest has proved the inspiration for our past five years' research, and in this paper we wish to summarise the early results, report recent findings, and outline future research directions.

Our broad research strategy was first to map out the full range of cognitive problems shown by children with dyslexia, and next to attempt to find the lowest common denominator of these problems by attempting to design simpler and simpler tests until

we eventually arrived at a situation where test A showed no deficit whereas test B, which involved addition of a minimal further component to A, revealed a deficit.

Overall Design of the Studies

It is clearly valuable to identify whether children with dyslexia perform significantly worse than their age-matched controls, but one of the key discriminants between theories is a test of performance of children with dyslexia against reading age controls, since a significant impairment compared with reading age controls is indicative of developmental disorder rather than just a developmental lag (cf. Bryant & Goswami, 1986). Since the specific nature of dyslexic children's deficits may also change with age, it is important to examine the effects of age separately. These considerations suggest an experiment with at least six groups of subjects: two groups of children with dyslexia of different mean ages; two groups of normal children matched to the children with dyslexia on chronological age; and two groups of normal children matched to the children with dyslexia on reading age.

Three separate issues are of interest in the statistical analyses for each experiment. First, whether there are any between-group differences at all. This involves a design which treats all the six groups within one factor, irrespective of age and presence/absence of dyslexia. We refer to this as the 'Overall Analysis'. A lack of a significant effect here would suggest that the variable under investigation was unaffected by either age or dyslexia. Second, it is important to identify whether children with dyslexia perform worse than their age-matched controls. This design has the two level factor age and the two level factor presence/absence of dyslexia. We refer to this as 'CA & Dyslexia'. A main effect of age would indicate a developmental trend in the variable in question, while a main effect of dyslexia would suggest a reliable difference between dyslexic and control subjects of equivalent age. Such a difference may, however, be attributable either to a fundamental difference, or to a developmental lag. Deciding between these requires a third analysis, one involving a comparison with reading age controls; this also has two factors, namely a two level factor reading age, and a two level factor presence/absence of dyslexia. We refer to this as 'RA & Dyslexia'. A negative effect of dyslexia on this analysis would indicate that dyslexic subjects are performing more poorly than younger children of equivalent reading age, and would argue against a developmental lag interpretation.

Note that the latter two analyses are based on only four of the experimental groups. Fortunately, as described in the next section we were able to select two groups of children with dyslexia who were sufficiently similar in IQ to allow a single control group to be used both as RA control for the older children with dyslexia and CA control for the younger children with dyslexia, thus leading to a total of only five groups.

The subject panel

Five groups of subjects participated in the initial studies. The groups were: 12 children with dyslexia around 15 years old; 11 children with dyslexia around 11 years old; a group of 12 normal children matched to the older children with dyslexia for age and full IQ; a group of 11 normal children of similar IQ to the two dyslexic groups, matched for chronological age with the younger children with dyslexia and for reading age with the older children with dyslexia; and a fifth group of 10 normal children around 8 years old matched for reading age and full IQ with the younger children with dyslexia. All the children with dyslexia had been diagnosed as dyslexic between the ages of 7 and 10, based on discrepancies of at least 18 months between chronological and reading age. Their IQ levels fell in the normal to superior range on the Wechsler Intelligence Scale for Children (Wechsler, 1976) and they had no known neurological deficit or primary emotional difficulty. For several children with dyslexia IQ and/or reading age deficit had changed since diagnosis, and a criterion of at least one year deficit in reading age compared with chronological age at the time of the experiment was adopted (this led to the exclusion of 5 children from a larger original pool). The children with dyslexia were recruited via the local dyslexia associations, and the normal controls were recruited from local schools. Recently we have recruited a group of 8 year old children with dyslexia and a group of 6 year old RA controls, and hope to report the combined results for all seven groups. The following analyses, however, are based only on our original five groups.

Study 1. Working Memory and Dyslexia

We first report briefly a set of experiments exploring the relationship between phonological processing, working memory and articulation rate. Recent research has demonstrated that in addition to their phonological deficits, children with dyslexia suffer impairments in working memory performance (Jorm, 1983; Snowling et al., 1986; Gathercole & Baddeley, 1990). In principle either a phonological deficit or a working memory deficit could underlie both sets of symptoms. We undertook a series of experiments with the above five groups of children designed to

Figure 1. Memory Span as a function of Articulation Rate
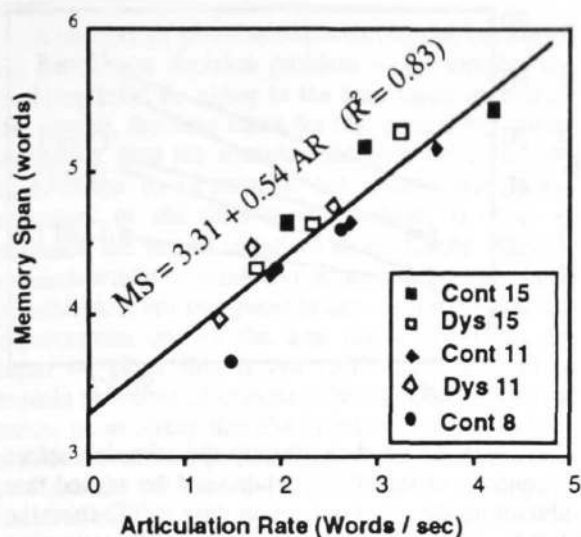
tease out which factors underlay the deficits (Nicolson, Fawcett & Baddeley, 1992).

In summary, the performance of the older children with dyslexia across the range of tasks was slightly but not significantly worse than their CA controls and indistinguishable from that of their RA controls. By contrast, the performance of the younger children with dyslexia was significantly worse than that of their CA controls on phonological discrimination, articulation rate, and nonword repetition, and was significantly worse even that of their RA controls on repetition of longer nonwords. On tests of memory span, all five groups showed the normal phonological similarity effect and the normal word length effect. When memory span was regressed as a function of articulation rate, there was no evidence of impaired slope or intercept for the children with dyslexia (see figure 1).

We concluded that the 11 year old children with dyslexia show residual problems on phonological processing, especially for tasks involving unfamiliar stimuli, but that by their mid-teens children with dyslexia have largely overcome these problems. The major remaining problem for the children with dyslexia appeared to be a continuing lack of fluency in articulation, a factor which is sufficient to account for the slight deficits on memory span. Furthermore, we argued that the articulation rate deficit provides a parsimonious explanation of the range of deficits shown by the younger children with dyslexia. We concluded that neither impaired phonological skills nor impaired working memory is sufficient in itself to explain the deficits, but that some deeper explanation must be sought.

Study 2. Motor Balance and Dyslexia

This set of experiments was designed to identify whether children with dyslexia showed any deficits in a skill as far removed as possible from reading, namely gross motor balance. Clearly any balance deficits would suggest that the phonological deficit hypothesis was insufficiently broad to account for the range of problems suffered by children with dyslexia. An initial study is reported in Nicolson & Fawcett (1990), with subsequent studies using the current subject groups reported in Fawcett & Nicolson (1991). In brief, we established that children with dyslexia balanced just as well as their controls under 'just balance' conditions, but that the children with dyslexia showed a significant balance deficit (ie. they wobbled more) when required to balance while carrying out a further task (even as simple a task as a selective choice reaction, in which they had to say 'Yes' or press a button on hearing a low tone, but to make no response if a high tone was presented).

We interpreted these results as evidence that dyslexic children's balance was not fully automated, unlike that of their controls, and consequently formulated our 'Dyslexic Automatisation Deficit' (DAD) hypothesis, which states that children with dyslexia suffer from extreme difficulties in fully automatising skills (whether cognitive or motor). In addition to DAD we formulated the 'Conscious Compensation' hypothesis which states that in normal circumstances children with dyslexia are able to mask their lack of automatisation by concentrating harder on the task (conscious compensation), and thus that the deficits will show up primarily under adverse conditions (such as in dual tasks; when high speed is required, as in reading; or in general when the child is stressed or tired). The DAD hypothesis has an inherent plausibility, and accounts for a range of findings about children with dyslexia such as quicker tiring, greater distractibility, problems in shoe lace tying, and the like (see Augur, 1985). However, it was by no means the only possible interpretation of the results, with one key issue being whether the dual task deficits were actually attributable to some general problem with attention sharing rather than a problem specific to balance.

We decided to investigate this issue further by running a further experiment in which we compared the balance performance of our subjects under normal conditions and when they were blindfolded. The latter condition is not a dual task condition but the subjects are prevented from using the normal visual cues to assist with balance. Consequently an attention-sharing deficit theory predicts equivalent performance to that of the controls, whereas DAD (and any motor skill deficit hypothesis) predicts impaired performance

Figure 2. Blindfold Balance

Figure 2. Blindfold Balance
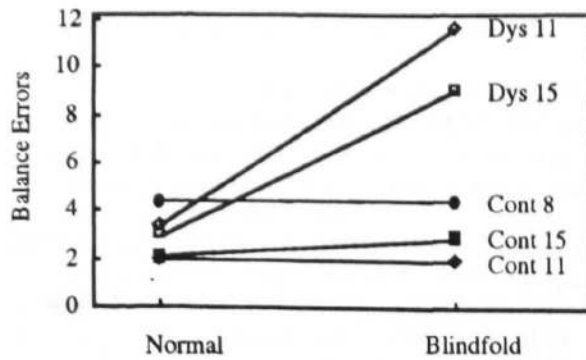
relative to the controls when blindfolded. The results were clearcut, following exactly the same pattern as we obtained in our earlier dual task balance experiments (see Fig. 2).

It may be seen that the children with dyslexia balanced as well as their controls in the normal balance condition, but, unlike the controls, the dyslexic children's balance was impaired in the blindfold balance condition. Statistical analyses of the blindfold balance deficit (the increase in errors when blindfolded) showed that both groups of children with dyslexia were significantly more impaired than all three control groups (p<.01).

Study 3. Speed of Processing and Dyslexia.

In an effort to trace the dyslexic deficit back to its source, we decided to investigate speed of information processing, using a variety of reaction time tasks in the hope that at some point we would find a cut off where tasks of lesser complexity would show no deficit, whereas more complex tasks would result in a deficit. We tried a variety of simpler and simpler tasks — 'Coding', lexical access, choice reaction, and finally simple reaction to a tone in an attempt to find normal performance. Deficits were found all the way down to 2-choice reactions, and so we present the data for the two simplest conceivable tasks, a simple reaction and a selective choice reaction task (SCRT). The study is presented in detail in Nicolson & Fawcett (1992) and so a summary should suffice here.

In both tasks, subjects sat with a single button in their preferred hand, and their task was to press it as quickly as possible whenever they heard a low tone. In the simple reaction task, no other tone was ever presented, but in the SCRT task, there was an equal probability of a high tone being presented. The subject had to ignore the high tone, thereby make a selective choice reaction to the low tone. These tasks were introduced by Donders well over a century ago. His rationale was that the only difference between the

Figure 3. Simple Reactions and Selective Choice Reactions

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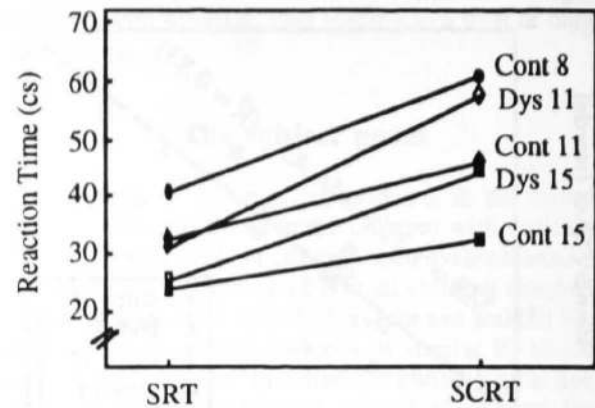
tasks was the need to classify the stimulus before responding in the SCRT trials, and he argued that subtracting the simple reaction time (SRT) from the SCRT time gave an estimate of 'stimulus classification' time. The experiment was computer-controlled, and 100 stimuli were presented at an average rate of 1 per 2 seconds. The results are shown in figure 3, plotted on the same graph to facilitate comparison.

It is clear that, although the subjects with dyslexia performed at the same level as their same-age controls on the simple reactions, they were slowed down more by the need to make an SCRT. Analysis of the simple reactions indicated that both groups of older subjects were significantly faster than all three groups of younger subjects.

Analysis of the SRT's in terms of age and dyslexia indicated a highly significant effect of age (p<.0001), whereas there was no effect whatsoever of dyslexia. Analysis of the SCRTs indicated a significant effect of both age and dyslexia at the .01 level. Pairwise comparisons indicated that performance of the older controls was significantly faster than for the other four groups.

A third analysis of variance was conducted on the SRT's, omitting the oldest controls and comparing the two dyslexic groups with their reading age controls. The main effects of both age and dyslexia were highly significant at the .001 level. Analysis of the SCRTs indicated a significant effect of age (p<.01) but no effect of dyslexia.

To summarise the RT results: for the simple reaction task both groups of dyslexic subjects performed at the appropriate level for their age, and significantly faster than their RA controls. However, for the SCRT condition the dyslexic subjects slipped back, to the extent that they were significantly slower than their CA controls and equivalent to their RA controls. The same pattern of results applied for both mean and median latency. Furthermore, the dyslexic children were, if anything, less accurate than their

age-matched controls, showing that the SCRT results cannot be attributed to some speed-accuracy trade-off effect.

A number of possible explanations can be offered for the slower decision problem — presumably the problem must lie either in the time taken to analyse the stimuli, the time taken for the 'central executive' to 'notice' that the stimulus has been classified, the time taken to determine the appropriate motor response, or the time taken to 'load' it ready to despatch the neural impulses to the finger. Further research would be needed to distinguish between these possibilities, but our guess is that the problem is not a perceptual one — the time taken to classify the input — since this is not sufficiently general to explain the range of deficits suffered. Consequently it seems most likely that the problem lies somewhere within the central processing system. We return to this issue in the overall discussion of the findings in the three studies.

Discussion

Simplifying greatly, there are three major groups of theory for the origins of dyslexia: the phonological deficit hypothesis, the visual deficit hypothesis, and the automatisation deficit hypothesis (henceforth DPD, DVD and DAD respectively). The most recent formulation of the DVD hypothesis is that there is a deficit in the transient visual system, caused by neuroanatomical abnormalities in the magnocellular pathways to the lateral geniculate nucleus (Livingstone et al., 1991). The DVD hypothesis is unable to account for the established phonological deficits or for the balance deficits reported here, and so can only be a partial explanation. Phonological processing is of course a skill built up via continual practice throughout childhood. It would therefore be expected under DAD that phonological deficits would arise, and indeed, the predictions of DAD appear to be indistinguishable from those of DPD in the area of phonological processing, except that DAD also predicts reduced speed of articulation. One might interpret, therefore, the DPD hypothesis as a specialisation of DAD to the phonological domain. It may, however, be significant that although automaticity for phonological skills develops in a similar fashion to visual automaticity, at least in the early stages it tends to be less resistant to interference from multi-channel input (Mullinex, Sawusch & Garrison, 1992). The two theories differ in that DAD predicts deficits outside the phonological domain (indeed, for any skilled activity where automatisation deficits cannot be masked by conscious compensation). This was the reason why we selected balance as a task to investigate in the first place — it

was a sharp test which discriminated between DPD and DAD.

Unlike DPD, the DAD hypothesis provides a natural explanation of both the dual task balance deficits and the blindfold balance deficits. It is also able to explain the working memory deficit naturally in terms of the reduced (less fluent) articulation rate. The remaining issue from the studies reported here is whether DAD is able to give a principled account of the reason for the dichotomy between SRTs and SCRTs. Here the argument is less convincing. Why, for instance, is there no deficit in SRTs, but a deficit in SCRT and other more complex choice reactions? Presumably the reason lies in the need to make a decision in the latter cases, rather than merely execute a pre-determined response. The slowed cognitive decision is quite consistent with the framework of automatisation — for the same amount of practice the decision is less automatic — but unfortunately for DAD, it can offer no principled reason why automatisation should be difficult only for decision-based tasks. Once one is attempting to explore central processes, the DAD hypothesis offers too coarse grain an analysis.

One speculation, given the involvement of the cerebellum not only in motor skill but also in automatisation (Ito, 1984) and in the development of cognitive skill and language (Leiner, Leiner & Dow, 1989), is that some cerebellar abnormality is mediating the range of deficits shown. Given the complexity of the brain circuitry involved, however, it seems likely that abnormalities in almost any component of the cerebro-cerebellar loop might lead to qualitatively similar symptoms.

Conclusions

To summarise our research findings, we established that a phonological deficit alone was not capable of accounting for the range of deficits found. There was strong evidence of an automatisation deficit on motor balance, and the automatisation deficit hypothesis appeared capable of explaining not only the balance deficits, but also the phonological deficit and the established problems of working memory. However, the automatisation deficits are best seen also as a symptom of some deeper underlying cause. The lack of a deficit for simple reactions, taken together with the appearance of a deficit in even the simplest choice reactions, suggest that the most likely cause is some problem within the central brain processes. One speculation is that these deficits derive from abnormalities within the cerebro-cerebellar neural pathways.

One must be cautious in generalising from the results from two small groups of children with dyslexia, but, if our findings are replicated on a wide

range of children with dyslexia, we believe that this analysis sets new and exciting agenda for dyslexia research for the next decade. The research agenda are clear: inter-disciplinary collaboration towards a clearly identified target that promises to disperse some of the mists which presently shroud the relationship between mind, brain and behaviour.

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