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The relationship between language-processing and visual-processing deficits in developmental dyslexia

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Abstract

Some research on developmental dyslexia focuses on linguistic abnormalities such as poor reading of nonwords or poor reading of exception words. Other research focuses on visual abnormalities such as poor performance on psychophysical tasks believed to assess the functioning of the magnocellular and parvocellular layers of the lateral geniculate nucleus (LGN). Little is known about what the relationships are between these two types of abnormalities. We measured nonword reading, exception word reading, and performance with Ternus apparent movement displays (the perception of which is believed to depend upon the magnocellular and parvocellular pathways) in dyslexic children and children without reading difficulties. Our results indicate that performance on the Ternus task is related to nonword reading ability but not to exception word reading ability. We offer two alternative interpretations of these findings. According to the first of these, nonword reading requires a serial left-to-right allocation of covert attention across the letter string being read and the neural systems involved in this attentional process also play a part in responses to the Ternus display. According to the second, poor nonword reading and abnormal Ternus performance are not directly related: perinatal/neurodevelopmental insult has affected the LGN (influencing Ternus performance) and the adjacent medial geniculate nucleus (MGN; affecting phonological ability) and the MGN abnormalities may be more functionally related to poor nonword reading.

Keywords: Dyslexia; Magnocellular; Dual route; Learning to read

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1. Introduction

According to the dual route theory of reading (Coltheart, 1978; Coltheart et al., 1993), there are two routes which can be used in order to read aloud: the nonlexical and the lexical routes. Nonwords and irregular words are used to examine these respective routes. Correct reading of nonwords (e.g. borp) requires proper use/knowledge of rules specifying grapheme-phoneme relationships in order to sound the nonword out. However, if such rules were used to read irregular words (e.g. yacht) they would be read incorrectly (regularised). Thus correct reading aloud of irregular words requires the ability to access their word-specific lexical representations. Skilled readers are proficient at both of these methods of processing letter strings. Therefore, if a child is to become a skilled reader, the child must acquire both of these subskills.[1]

A number of authors have suggested that this analysis of what is involved in learning to read implies that in some children with developmental dyslexia it will be the lexical procedure that is being acquired abnormally slowly, whilst in some others it will be the nonlexical procedure; in still other children both difficulties may be present.

A difficulty in acquiring the lexical procedure is known as developmental surface dyslexia and is indicated by abnormally poor performance on exception-word reading; numerous studies have documented this form of developmental dyslexia (Holmes, 1973, 1978; Coltheart et al., 1983; Job et al., 1984; Temple, 1984; Goulandris and Snowling, 1991; Hanley et al., 1992; Broom and Doctor, 1995a; Castles and Coltheart, 1993, 1996; Manis et al., 1996; Temple, 1997).

A difficulty in acquiring the nonlexical procedure is known as developmental phonological dyslexia and is indicated by abnormally poor performance on nonword reading; this form of developmental dyslexia has also been documented in numerous studies (Temple and Marshall, 1983; Temple, 1984, 1997; Campbell and Butterworth, 1985; Snowling et al., 1986; Snowling and Hulme, 1989; Castles and Coltheart, 1993; Manis et al., 1996; Broom and Doctor, 1995b).

1.1. Visual processing deficits in developmental dyslexia

The patterns of reading difficulty described above are characterised in linguistic terms, i.e. they are seen as arising because of difficulties in acquiring specific components of the language-processing system. Hence they contrast with patterns of reading difficulty the etiologies of which are considered to be visual rather than linguistic in origin.

In the 1980s, Lovegrove and others began to approach the issue of a visual deficit in developmentally dyslexic children from the standpoint of visible persistence theory (Breitmeyer and Ganz, 1976; Coltheart, 1980; Slaghuis and Lovegrove, 1984). For example, Slaghuis and Lovegrove (1984) suggested that, as dyslexics'

[1]This particular dual-route analysis of learning to read and developmental dyslexia is not of course the only one possible; see Manis et al. (1996) for an alternative approach.

eyes saccade across a page, visible persistence of previous fixations mask currently-fixated text, making it difficult to identify letters or words.

In Lovegrove's experiments, to test for visible persistence differences between good and poor readers, a sine or square wave grating was presented alternating with a blank interstimulus interval (ISI). Each trial consisted of a grating-ISI cycle repeated 10 times, and subjects reported whether they observed a blank interval between the gratings or not. Failure to report a blank interval was considered to indicate that the visible persistence of the first stimulus was still present at the onset of the second. This line of research over the past fifteen years has shown that children with poor reading have longer-lasting visible persistences at low spatial frequencies compared to good readers (Martin and Lovegrove, 1984, 1988; Slaghuis and Lovegrove, 1984, 1985, 1986ab; Slaghuis and Davidson, 1993). This is inferred from the finding that poor readers have more difficulty than good readers in detecting the blank ISI at low spatial frequencies. In addition to visible persistence being longer for low spatial frequencies in reading disabled children, it has also been reported as shorter for higher spatial frequencies in such children.

Such effects have been related to the distinction between two different visual pathways, the magnocellular and the parvocellular. Research into primate and human visual systems suggests there are two parallel and separate yet interacting pathways of the visual system which begin in the retina and end in the cortex. These two pathways are referred to as the magnocellular/transient and parvocellular/sustained pathways. The magno system responds rapidly to low spatial and high temporal frequencies, whereas the parvo system responds more slowly to high spatial and low temporal frequencies - hence the terms transient and sustained for magno and parvo systems respectively. The parvo system also distinguishes colours even when hues are of equal brightness, whereas the magno system cannot make such distinctions. Slaghuis et al. (1996) have referred to the parvo system as the determinant of what a visual stimulus is and the magno of where a visual stimulus is. Although neuroanatomically these systems appear independent, e.g. they are in different layers of the lateral geniculate nucleus (LGN) and the parvo path extends to the temporal cortex whereas the magno path is biased toward the parietal cortex, they also appear to interact, e.g. they meet in the path to the temporal cortex (Merigan and Maunsell, 1993).

In 1976, Breitmeyer and Ganz offered a theoretical interpretation of data obtained from primate/human visual studies which suggested a means by which these two pathways interact. The theory is referred to as visible persistence theory. Visible persistence is the amount of time for which a visual stimulus continues to be seen after it has been terminated. Breitmeyer and Ganz suggested that after a saccade has been made from one fixation point to another, magno/transient channels inhibit parvo/sustained channels so that the previously fixated stimulus does not remain visible and mask the next stimulus which is fixated. Such masking would obviously interfere with reading. Lovegrove and colleagues therefore proposed that the visual deficit in developmental dyslexia took the form of inadequate inhibition by the transient system.

Another technique for studying the magno and parvo channels, the measurement of contrast sensitivity (the minimum amount of contrast needed to detect a grating pattern), has also been popular. This technique yields contrast sensitivity functions (plots of contrast sensitivity against spatial frequency). For example, Lovegrove et al. (1980) presented good and poor readers with sine wave gratings for 40–1000 ms with 1 s blank ISIs and a total presentation of 10 s for each cycle. They found that poor readers had significantly lower contrast sensitivity at low spatial frequencies than good readers. When the displays were flickered (which is considered to further excite the magno system thereby increasing inhibition of the parvo), good readers showed an increase in contrast sensitivity compared to poor readers and this difference increased with an increase in spatial frequency. This was interpreted as a consequence of the disruption of the magno channels in poor readers, which resulted in the flickering being unsuccessful at exciting the magno channels further; the magno channels in good readers were normal and so the flickering did further excite magno paths, thereby increasing inhibition of parvo systems, allowing for greater contrast sensitivity.

In more recent years, a third technique for exploring the magno and parvo systems of dyslexics has been adopted: the Ternus test of visual motion detection.

1.2. The Ternus test of visual motion detection

When researchers set out to examine properties of visual motion detection as expressions of magno and parvo routes, they used a particular technique developed by Ternus (1938) which allows illusions of movement to be created. Three horizontally aligned equidistant squares are briefly presented (frame 1), then re-presented moved to the right by one imaginary equidistant square and flashed them on the screen again (frame 2). Several alternating presentations of frames 1 and 2 are given. When the interstimulus interval between the presentation of frames 1 and 2 was approximately 50 ms or more, the three squares appear to move backwards and forwards as a group. If, however, the interstimulus interval from frame 1 to 2 is less than 50 ms, the three squares did not appear to move to the right as a group; instead, the first square appeared to disappear from position one and reappear in a fourth position (after the third square). The former illusion can be referred to as apparent group movement and the latter as apparent element movement.

These group and element movement illusions are considered to depend upon parvo and magno routes respectively (Braddick, 1980; Breitmeyer and Ritter, 1986a,b; Petersik and Pantle, 1989). Breitmeyer and Ritter argue, for example, that during the successive presentations of the Ternus display images of the second and third squares persist from frame 1 to frame 2 when ISI is short enough. The integration of the persisting image from frame 1 with the contents of frame 2 leads to the perception that the second and third squares are stationary. That rules out the group movement percept; hence element movement is seen. As ISI increase, the visible persistence of frame 1 is progressively less likely to be present at the onset of frame 2, and so the group movement percept is progressively less likely to be ruled out.

From this standpoint, element movement depends upon the parvo system, since that system maintains the persisting images of central squares which rule out the group movement percept. Group movement depends upon the magno system, since that system is responsible for terminating visible persistence via inhibition of the parvo system.

Researchers have been in agreement concerning the usefulness of the Ternus test to examine magno and parvo systems or visible persistence for many years; however, the test only began to be used for studying dyslexia in the late 1980s (Patterson et al., 1988; Winters et al., 1989).

Recently, Slaghuis et al. (1996) administered the Ternus test to good and poor readers to determine whether (1) the Ternus test of apparent visual motion detection can be used as a measure of visible persistence, and (2) dyslexics actually do differ from controls on apparent visual motion detection. Slaghuis et al., like Breitmeyer and Ritter (1986a,b), maintained that the group movement illusion depends upon the magno system inhibiting the parvo system; if so, and if poor readers have abnormally weak inhibition of parvo by magno, they should show less group movement than good readers; and that is what they found (Experiment 1).

Slaghuis et al. also added a flicker masking component to the Ternus Paradigm, which led to an increase in group movement detection for good readers whilst poor reader performance remained the same (Slaghuis and Lovegrove, 1984; Martin and Lovegrove, 1988). As discussed above, this was interpreted as suggesting that poor readers' magno channels were disrupted so the flickering was not successful at exciting the magno channels further, whereas good readers' magno channels were normal so the flickering did further excite magno paths thereby increasing inhibition of parvo paths and so increasing the occurrence of the group movement percept.

Experiment 2 of Slaghuis et al. was identical to their Experiment 1 except that the dyslexics and controls were adults ($n = 9$ in each group) and the condition with uniform field flicker was not run. Once again, the dyslexics reported significantly less group movement than good reader controls.

1.3. How are the language-processing deficits related to the visual-processing deficits in developmental dyslexia?

Slaghuis et al. (1996) also measured nonword reading. In both experiments, the dyslexics were far worse than the normals (in fact there was no overlap between the two sets of scores).

What relationship does this finding have to the finding that the dyslexic children reported significantly less group movement in the Ternus display than did the normal children? It is not obvious why there should be any relationship between the functioning of the magno system and the ability to read single nonwords aloud.

Slaghuis et al. investigated this by determining the correlations between mean number of judgements of group movement and nonword reading accuracy; in their Experiment 1, these were +0.29 for the normal children and –0.37 for the dyslexics. These correlations were not statistically significant – perhaps, as Slaghuis et al. note,

because of the small sample sizes. They then report the results of a discriminant analysis using group movement and nonword accuracy to classify children as dyslexic or not, and report that this classification was 100% correct – but of course, that had to be so, since there was no overlap between the nonword reading scores of the two groups.

Similarly, the correlation between mean number of judgements of group movement and nonword reading accuracy in their Experiment 2 were –0.10 for the normal adults and +0.20 for the dyslexics, neither value being statistically insignificant.

Here, then, there was no evidence of any relationship between the visual deficit and the language-processing (i.e. nonword reading) deficit. Hence, although the poor readers were different from the good readers on both the Ternus test and nonword reading, the insignificance of these correlations suggest that these two differences between good and poor readers may arise for different reasons, which is inconsistent with the view that poor readers are poor at reading because of too-weak inhibition of the parvo system by the magno. If that view were correct, then the weaker this inhibition, the poorer reading would be, and the lower group-movement reports would be, i.e. group movement and reading would be significantly positively correlated.

1.4. Present study

Our study is a replication and extension of the study reported by Slaghuis et al. Firstly, in order to overcome the limitations of the correlational analyses caused by the small sample sizes in the Slaghuis et al. experiments, we used a larger sample of dyslexics and of controls (approximately 40 per group).

Secondly we addressed a much more serious limitation of that study, namely that it ignored the now very substantial body of research supporting the idea that there are subtypes of dyslexic children. This body of research makes it very unlikely that there will be any single defect, be it visual or linguistic, present in all dyslexic individuals. Two prominent linguistic defects, as discussed earlier, are poor exception word reading and poor nonword reading. If one is interested in the relationship between linguistic and visual deficits, it is therefore essential to measure both exception word reading and nonword reading and to relate each of these to the visual deficit as indexed by the Ternus technique.

Even if the correlations between nonword reading and group movement in the Slaghuis et al. studies had been significant, the authors would not have been entitled to conclude that this relationship was a causal one. We know that exception word reading and nonword reading accuracy are highly correlated in children (Coltheart and Leahy, 1996). Hence, if the true association were between exception word reading and group movement, there would still have been a correlation between nonword reading and group movement, a correlation which would disappear when the contribution of the exception word reading variable was partialled out. But Slaghuis et al. could not have performed such a partial correlation analysis, because they did not measure exception-word reading. We did, and so we could.

2. Method

2.1. Subjects

Forty-three poor and forty-four good readers from primary schools throughout Sydney, Australia, participated. Thirty-seven of the poor readers were enrolled in the Multilit Reading Remediation program at Macquarie University (an intensive three-term program to improve reading skills) and the remaining six poor readers were receiving special attention within their schools for reading difficulties. Good readers were obtained via an ad placed in a Sydney newspaper requesting participation in a study on reading/learning in exchange for psychometric/language reports. None of the children had histories of hearing problems with the exception of one good and one poor reader who both had increased hearing thresholds in high frequency ranges.

2.2. Materials

The Ternus display consisted of three bright squares on a dark background. Each side of the squares in the display subtended a visual angle of 1.2 deg. The distance between the nearest edges of adjacent squares was 1.2 deg. Viewing distance was held constant at 95 cm with a chin rest. The luminance of the squares was 52.30 cd/m^2 and the luminance of the background was 11.67 cd/m^2.

These displays were presented using a Pentium 150 PC, 16MB RAM, and programs written using custom software libraries provided as part of the VRG package in a 32-bit DOS environment. The PC houses the VRG hardware, which includes a Cambridge Research Systems 2/3 Visual Stimulus Generator, and a Texas Instruments TIGA interface card. These drive a Mitsubishi HL7955SFKL monitor. The screen refreshes every 8 1/3 ms, providing a frame rate of 120 Hz.

2.3. Procedure (Ternus data)

Apparent movement was measured at 12 interstimulus intervals: 8.3, 16.6, 24.9, 33.2, 41.5, 49.8, 58.1, 66.4, 74.7, 83.0, 91.3 and 99.6 ms (Slaghuis et al. (1996) used nine ISIs ranging from 11 to 99 ms in 11-ms steps). As with Slaghuis et al., the duration of individual display frames was 55 ms and there were 20 trials at each interstimulus interval (a trial consists of eight successive frame 1–2 presentations). With 20 trials at twelve ISIs (as opposed to the nine ISIs in the Slaghuis et al. (1996) study) there were a total of 240 trials (as opposed to 180 in the Slaghuis et al. study). The order of ISIs was randomised. Subjects were trained to press the left button on a large computer mouse for element movement, and the right button for group movement. The children were told to fixate on a small cross in the middle of the screen and not to move their eyes from the cross when the squares were flashing above or below it (despite saying this, a few children commented that it was too hard to stare at the cross all of the time or that they forgot to stare at the cross the whole time).

This experiment was carried out with 74 of the sample of 87 good and bad readers described above (35 good readers and 39 bad readers were tested).

The following was read to each child:

> Sometimes you will see three squares moving back and forth like this (pointed three consecutive fingers near the screen and moved them back and forth to demonstrate all three moving horizontally). When you see three moving back and forth like this, you press this (put their finger on the right button) button. Other times you will see the first square jumping to the end of the line and back again, like this (placed the index and little finger up toward the screen with the two inside fingers down and alternated moving the index finger down and then the little finger down). When you see one square jumping back and forth, you press this button (placed their finger on the left button). Do you understand? (If the child said 'no', it was explained again). I'll show you what I mean. Let's do some practice ones first.

The lights were turned out and all children were given practice until they mastered the task. If a child appeared to be having difficulty with the task during the practice, the experimenter asked the child to say aloud whether they saw one jumping or three moving as they pressed the button. This was to ensure that the difficulty did not lie in confusing the button presses with different illusions. If a child said 'three moving' while pressing the left button (one jumping button), for example, and did this consistently for approximately five or more trials, while also saying 'one jumping' while pressing the right button (three moving button) the experimenter would then say: 'Now remember, this (point to left) button is for one jumping and this (point to right) button is for three moving, right?' The child would then continue until the task was sufficiently mastered.

2.4. Procedure (reading data)

Eighty of the 87 children (40 good readers and 40 bad readers) were asked to read aloud, without time pressure, 30 regular words, 30 irregular words, and 30 non-words, derived from Castles (1993) and listed in Appendix I of Coltheart and Leahy (1996). Each word was presented on a flashcard and flashcards were presented in a different random order for each child.

3. Results

Age and reading data for good and poor readers are given in Table 1.

The groups differed significantly in age: $t(74) = 2.03$, $P < 0.05$. We did not consider that this difference posed a problem because it was very small, and because the poor readers were slightly older than good readers; hence, significantly better performance of good readers over bad readers could not be attributed to a confounding of reading ability with age. Bad readers were significantly worse than good readers on all three of the word types: regular words $t(78) = 10.09$,

Table 1
Age, and regular/irregular/nonword data of good and poor readers[a]

	Poor readers			Good readers		
Variable	Mean ± SD	Min.	Max.	Mean ± SD	Min.	Max.
Age	10.20 ± 1.21	7	12	9.57 ± 1.50	7	12
Regular	20.15 ± 5.50	7	29	29.30 ± 1.62	22	30
Irregular	13.37 ± 5.09	4	23	25.53 ± 3.00	19	30
Nonwords	14.67 ± 5.33	1	24	28.38 ± 2.26	20	30

[a]Poor readers: $n = 40$; good readers: $n = 40$ (age data available on 76 of these children). Regular word reading: 19 poor readers had regular word scores at or above the good reader floor of 22. Irregular word reading: six poor readers had irregular word scores at or above the good reader floor of 19. Nonword reading: nine poor readers had nonword scores at or above the good reader floor of 20.

$P < 0.001$, irregular words $t(78) = 13.00$, $P < 0.001$, and nonwords $t(78) = 14.96$, $P < 0.001$.

3.1. The Ternus effect in good versus poor readers

In order to determine if there was a significant difference between good and poor readers' response choices across all ISIs, a two-factor repeated analysis of variance was conducted with group the between factor and ISI the within/repeated factor. There was an insignificant group effect ($F(1,76) = 2.17$, $P > 0.05$), a significant IS effect ($F(11,836) = 239.28$, $P < 0.001$) and a significant interaction ($F(11,836) = 13.133$, $P < 0.001$). The average group movement choice at each ISI for the two reader groups is shown in Fig. 1.

Simple t-tests at each ISI show significant differences between the groups at ISIs 1, 2, 3, 7, 8, 9, 10, 11 and 12: 1 (8.3 ms) $t(76) = 4.07$, $P < 0.0001$; 2 (16.6 ms) $t(76) = 3.65$, $P < 0.001$; 3 (24.9 ms) $t(76) = 2.78$, $P < 0.01$; 7 (58.1 ms) $t(76) = 3.14$, $P < 0.01$; 8 (66.4 ms) $t(76) = 4.57$, $P < 0.001$; 9 (74.7 ms) $t(76) = 3.69$, $P < 0.001$; 10 (83.0ms) $t(76) = -2.91$, $P < 0.01$; 11 (91.3 ms) $t(76) = 3.42$, $P = 0.001$; and 12 (99.6 ms) $t(76) = 5.07$, $P < 0.001$. Thus, at short ISIs, the dyslexics show significantly more group movement than the controls, whilst at long ISIs the dyslexics show significantly less group movement than the controls.

Slaghuis et al. (1996) had found a significant between group effect in both of their experiments, which was not found here. They also, as we did, found a significant groups by ISI interaction in both experiments. In their experiment with young subjects, dyslexics showed less group movement then controls only at intermediate ISIs (33, 44 and 55 ms). However, this analysis is collapsed across the flicker and nonflicker condition. Flicker increased group movement in the controls but not the dyslexics. Hence if Slaghuis et al had analysed just their nonflicker condition, which is comparable to our condition whose data are reported in Fig. 1, the amount of group movement at low ISIs would have been reduced for controls but not for dyslexics. Hence it is possible that in their nonflicker condition, dyslexics may have shown more group movement than controls at short ISIs, as we found.

In their experiment with adult subjects, which only used the nonflicker condition as we did, a different pattern emerged: dyslexics showed less group movement then controls at all ISIs from 44 ms upwards (at the longest ISI, 99 ms, the effect is reported as nonsignificant, but it is as large as the effect at 88 ms; the failure to reach significance may have occurred because of a ceiling effect, since almost all of the controls were reporting 100% group movement). This agrees with our results at long ISIs. However, there is no suggestion in the data from their Experiment 2 that the dyslexic subjects showed more group movement at short ISIs than the controls, which was the case in our experiment and may have been the case in their Experiment 1.

Hence although it is clear from all three experiments that dyslexics differ from normals in their response to the Ternus task, it is not clear exactly what the pattern of the interaction between reader group and ISI actually is.

As is evident from Fig. 1, the plots of ISI against proportion of group movement for both good and poor readers are approximately ogival (cumulative-normal) in shape. Hence these plots will be approximately linearised if the proportions are converted to *Z*-scores, and that will allow the plots to be summarised by three values, a slope, an intercept, and a squared correlation (representing the strength of the relationship between ISI and percentage of group movement reports). So we converted each subject's proportion of group movement at each ISI to *Z*-scores. Analyses showed significant differences between the groups on the intercept $t(76) = 3.64$, $P < 0.001$, slope $t(76) = 4.93$, $P < 0.001$ and the amount of variance explained by the line (R^2), $t(76) = 2.71$, $P < 0.01$. The means of each of these measures for each group are shown in Table 2.

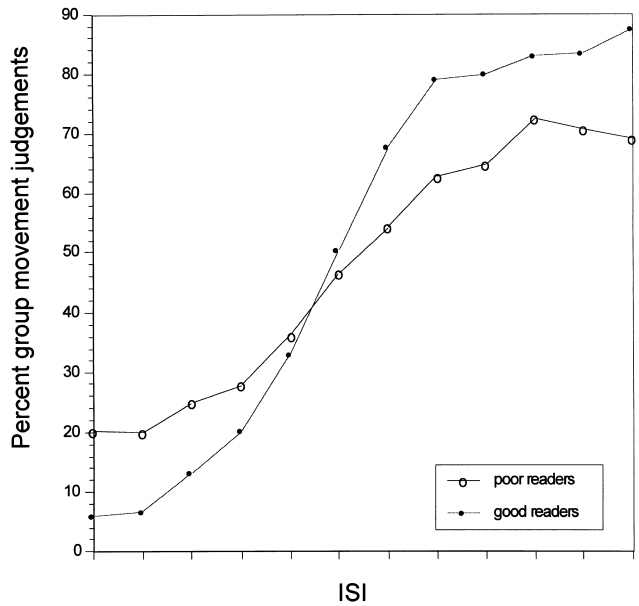

Fig. 1. Mean group movement as a function of ISI for the good and bad reader groups.

Table 2
Ternus indices for good, poor and all readers[a]

	RSQ	Slope	Intercept
Good readers	0.801 ± 0.112	–0.034 ± 0.008	1.786 ± 0.533
Poor readers	0.685 ± 0.238	–0.021 ± 0.014	1.251 ± 0.741
All readers	0.742 ± 0.195	–0.027 ± 0.013	1.512 ± 0.698

[a]Data are the mean ± SD.

3.2. Phonological versus surface dyslexics

Having established that our dyslexic children as a group showed a different pattern on the Ternus test than did the normal readers, we turn now to considerations of heterogeneity and subtyping. Did all of our dyslexics children display abnormal Ternus behaviour, or only some of them? And if only some, what distinguishes the dyslexic children who are abnormal on the Ternus test from the dyslexic children who are not?

One approach to investigating this issue is to try to pick out from the dyslexics a subset of children who are pure phonological dyslexics (abnormal nonword reading with normal exception-word reading) and a second subset who are pure surface dyslexics (normal nonword reading with abnormal exception-word reading), and to compare their Ternus profiles.

We did this as follows. For all 80 children, we subtracted the number of nonwords correctly read from the number of irregular words correctly read. Positive values of this number indicate a superiority of the lexical route over the nonlexical route, and so extremely high positive values signal phonological dyslexia. Negative values of this number indicate a superiority of the nonlexical route over the lexical route, and so extremely high negative values signal surface dyslexia.

Having ranked all 80 children according to this (irregular word–nonword) index, we used this method rather strictly. We counted as pure phonological dyslexics only those children whose index was larger than the largest positive index yielded by any normal reader. That is, the phonological dyslexics' indices were not only large, but completely outside the normal range; in other words, they had a larger disadvantage for nonwords relative to irregular words than any child in the normal reader group. There were 10 such children (25% of the poor reader sample).

We counted as pure surface dyslexics only those children whose index was smaller than the smallest negative index yielded by any normal reader. That is, the surface dyslexics' indices were not only low, but completely outside the normal range; in other words, they had a larger disadvantage for irregular words relative to nonwords than any child in the normal reader group. There were three such children (7.5% of the poor reader sample).

Analysis of the Ternus group movement scores of these two extreme groups revealed an insignificant group effect ($F(1,11) = 0.014$, $P > 0.05$), a significant ISI effect ($F(11,121) = 24.36$, $P < 0.001$) and a significant interaction

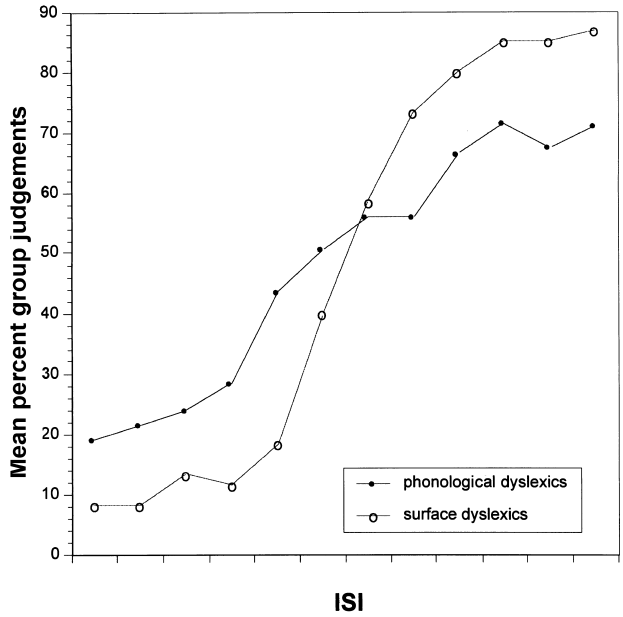

Fig. 2. Mean group movement as a function of ISI for the phonological-dyslexic and surface-dyslexic subgroups.

(F (11,121) = 2.05, $P < 0.05$). These scores are plotted in Fig. 2. It is evident that the surface dyslexics produced less group movement than the phonological dyslexics at short ISIs, and more group movement than the phonological dyslexics at long ISIs.

Next, the phonological dyslexics were compared to the good readers. A two factor ANOVA across all 12 ISIs showed an insignificant group effect (F(1,46) = 0.98, $P > 0.05$) and both significant ISI (F(11,506) = 119.97, $P < 0.001$) and interaction (F(11,506) = 8.14, $P < 0.001$) effects. The data are plotted in Fig. 3.

Next, the surface dyslexics were compared to the normal readers. A two factor ANOVA across all 12 ISIs showed that surface dyslexics and good readers do not differ on the Ternus. The group (F(1,39) = 0.35, $P > 0.05$) and interaction (F (11,429) = 0.61, $P > 0.05$) effects were both insignificant and the ISI effect was significant (F(11,429) = 79.04, $P < 0.001$). The data are plotted in Fig. 4. To further substantiate these findings, we slightly relaxed the surface dyslexia criterion (very stringent to begin with) to increase the sample size ($n = 7$) and ran this analysis again. Doing so, lead to precisely the same findings with very insignificant group (F (1,43) = 0.91, $P > 0.05$) and interaction (F(11,332) = 2.040, $P > 0.05$) effects and a significant ISI effect (F(11,332) = 126.92, $P < 0.001$).

The evidence for heterogeneity is clear. Only some developmental dyslexics behave abnormally with the Ternus display – the developmental phonological dyslexics. Developmental surface dyslexia is a different form of dyslexia and one in which there is no Ternus abnormality.

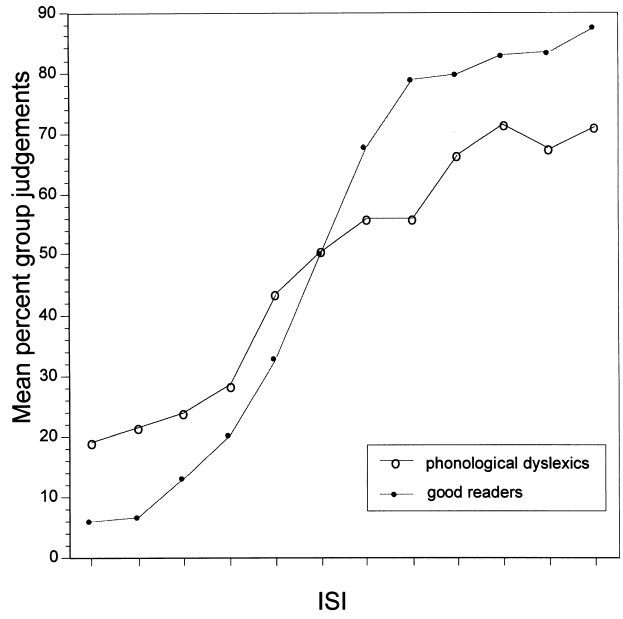

Fig. 3. Mean group movement judgements for the phonological dyslexic group and normal readers.

A second approach to investigating the issue of heterogeneity is to study the correlations between the linguistic and visual variables. Firstly, Table 3 shows the intercorrelations between the three reading measures for each of the two reader groups. Only children with both reading data and Ternus data are included here.

Table 4 shows the intercorrelations between the three reading measures and the total number of group-movement judgements, for each of the two reader groups.

Total group judgement was not correlated with performance on any of the word types. As we noted above, in the data of Slaghuis et al. (1996) there was no significant correlation between nonword reading accuracy and mean group judgement. They attributed the lack of significance to the small sample size in their experiment; however, the larger sample size in the present study did not lead to significant correlations. We consider instead that the failure to find correlations here occurs because mean group-movement judgement is a less appropriate way of characterising the differences in Ternus performance between good and bad readers than the slope, intercept and R^2 measures that come from Z transformation of the group movement judgements, since our two reader groups did not differ on mean group-movement judgements, but did differ on the scores derived from the Z transformations.

The correlations between each of the three Ternus indices and each of the three reading tests are shown in Table 5.

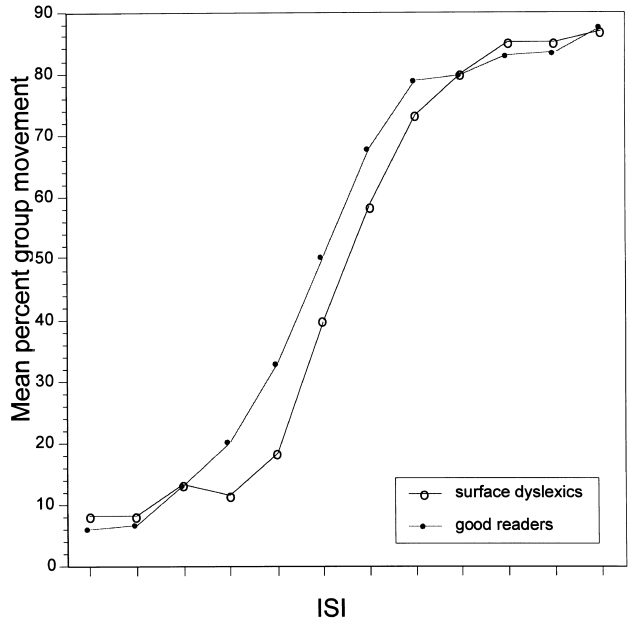

Fig. 4. Mean group movement judgements for the surface dyslexic subgroup and good readers.

As demonstrated in Table 5, the intercept and slope indices were significantly correlated with performance on all three word types, and the R^2 index was significantly correlated with regular word and nonword reading ($P < 0.04$ in all cases); the correlation with irregular word reading was marginally significant.

We have found that performance of the lexical route, as measured by accuracy of exception-word reading, is significantly correlated with all three Ternus measures, and also that performance of the nonlexical route, as measured by accuracy of nonword reading, is also significantly correlated with all three Ternus measures.

However, this does not mean that there is a genuine relationship between Ternus behaviour and both of the routes, because there is a significant correlation between exception-word reading accuracy and nonword reading accuracy, so that, if just one of the routes is genuinely associated with Ternus behaviour, scores measuring the performance of the other route will also show a significant (but misleading) correlation with Ternus scores. The way to deal with this is to calculate partial correlations.

Table 3
Intercorrelations of reading measures

	Irreg. and Reg.	Irreg. and Non	Reg. and Non
Good readers ($n = 34$)	0.579	0.572	0.765
Poor readers ($n = 37$)	0.725	0.418	0.786
All readers ($n = 71$)	0.873	0.837	0.906

Table 4
Correlations between reading accuracy and total group judgements (t.g.j) for each item type for good, poor and all readers

	Regular and t.g.j.	Irregular and t.g.j.	Nonwords and t.g.j.
Good readers	+0.025	+0.145	–0.077
Poor readers	+0.044	–0.038	–0.001
All readers	+0.097	+0.096	+0.076

The partial correlations between exception-word reading and Ternus indices with nonword reading partialled out are shown for each reader group in Table 6. None of the partial correlations with irregular-word reading approached significance. In contrast, the partial correlations between nonword reading and Ternus indices with exception-word reading partialled out (also shown in Table 6) were significant for intercept ($P < 0.01$) and for slope ($P < 0.02$), and the R^2 partial correlation approached significance ($P < 0.10$, two-tailed).

The outcome is clear. Performance on the Ternus test is unrelated to the lexical route, but is related to the nonlexical route: the worse a child is at nonword reading, the higher the intercept and the flatter the slope of the plot of Z-transformed group judgement proportions against ISI.

That agrees with the conclusion we reached in our analyses of the two subtypes (that pure phonological dyslexics show abnormal performance on the Ternus tests, but pure surface dyslexics do not).

4. Discussion

4.1. Evidence for subtypes of developmental dyslexia

As we noted in Section 1, it is now widely accepted that there is a surface-dyslexic subtype of developmental dyslexia (Holmes, 1973, 1978; Coltheart et al., 1983; Job et al., 1984; Temple, 1984, 1997; Goulandris and Snowling, 1991; Hanley et al., 1992; Castles and Coltheart, 1993, 1996; Broom and Doctor, 1995a; Manis et al., 1996) and also a phonological-dyslexic subtype of developmental dyslexia (Temple and Marshall, 1983; Temple, 1984, 1997; Campbell and Butterworth, 1985; Snowling et al., 1986; Snowling and Hulme, 1989; Castles and Coltheart, 1993; Broom and

Table 5
Correlations between regular/irregular/nonword reading accuracy and Ternus indices (intercept, slope and R^2

	R^2	Slope	Intercept
Reg.	0.328	0.488	–0.379
Irreg.	0.244	0.469	–0.335
Non	0.309	0.526	–0.447

Table 6
Partial correlations between irregular word reading accuracy and Ternus indices (intercept, slope and R^2) with nonword reading accuracy partialled out, and between nonword reading and Ternus indices (intercept, slope and R^2) with irregular word reading accuracy partialled out

	R^2	Intercept	Slope
Irregular word	–0.028	0.080	0.062
Nonword	0.197	–0.323	0.276

Doctor, 1995b; Manis et al., 1996). Our results provide yet more evidence for this distinction, since we have shown that whether dyslexics show abnormal performance on the Ternus test depends upon whether they are phonological dyslexics or surface dyslexics.

Our results also agree with those of three recent studies which sought to study neural correlates of developmental dyslexia (Borsting et al., 1996; McPherson et al., 1996; Spinelli et al., 1997).

Borsting et al. (1996) studied functioning of the magnocellular system in 17 dyslexic adults and nine adults who were normal readers. No standardised assessment of the reading abilities of the 26 subjects was carried out, but all the subjects classified as dyslexic reported having had difficulties with reading and spelling, and none of the subjects classified as normal readers did. Magnocellular functioning was determined by measuring contrast sensitivity at six spatial frequencies from 0.5 to 12.0 c/deg, drifting at two temporal frequencies (1 and 10 Hz). Since the magnocellular system is important at low spatial frequencies and high temporal frequencies, a deficit of that system in the dyslexic group would be indicated if the dyslexics had lower contrast sensitivity than the normals at low spatial frequencies (especially at high temporal frequencies), with no difference in contrast sensitivity between the groups at high spatial frequencies.

What is very unusual about this study is that it sought to subtype the dyslexic subjects. The subtypes sought were those of Boder (1973), who identified ‘three basic subtypes: dyseidesia, dysphonesia, and dysphoneidesia. Dyseidesia is a deficit in the ability to perceive whole words as visual gestalts and match these words with auditory gestalts. Phonetically regular words (e.g. stop, did, devoted) present no problems for correct decoding but phonetically irregular words (e.g. laugh, does, foreign) may not be decoded correctly..... Dysphonesia is a deficit in word analysis synthesis skills. Dysphonetics have difficulty using grapheme-phoneme relationships when encountering unfamiliar words..... A third type of dyslexia, dysphoneidesia, is a combination of the two types of deficits in eidetic and phonetic coding skills’ (Borsting et al., 1996p. 1048). Two subtypes of dyslexics were selected: nine dyseidetics and eight dysphoneidetics. Subtyping was carried out using the Adult Dyslexia Test.

At 1 Hz, contrast sensitivity did not differ between the three groups. At 10 Hz, however, there was a difference. Here, the dyseidetics did not differ from the normals, but the dysphoneidetics had lower contrast sensitivity than the dyseidetics or the normals, at spatial frequencies of 0.5, 1.0 and 2.0 c/deg; there were no

differences, however, at higher spatial frequencies. As Borsting et al. (1996) (p. 1051) concluded, 'the results are consistent with the notion that adult dysphoneidetic subjects have a deficit in the magnocellular pathway, whereas dyseidetics do not'.

The dyseidetics in this study presumably must have had normal nonword reading (otherwise they would have been classified as dysphoneidetics) though no data relevant to this point are reported by Borsting et al. (1996). If this is so, then their results of agree with ours: a deficit of the magnocellular pathway is not seen in all developmental dyslexics, but only in those who are impaired at nonword reading.

McPherson et al. (1996) measured event-related potentials (ERPs) of the brain whilst developmentally dyslexic adolescent subjects judged whether pairs of pictures did or did not have rhyming names. The 14 subjects all had a history of reading difficulty. They were assessed on the WRAT-R, Gray Oral Reading, Woodcock Reading Mastery and Decoding Skills tests, but the results of these assessments are not reported, except for the latter, a nonword reading test, on which the group averaged 19.3/60 correct; a group of normal readers averaged 44/60 correct.

The subjects were divided into two equal groups of seven on the basis of a median split on their scores on the Woodcock word reading tests. Comparisons of ERP data between these two groups showed no group differences.

However, when the subjects were divided into two equal groups of seven on the basis of a median split on their scores on the nonword reading test, a variety of differences between the two groups did emerge. For example, compared to the worse nonword readers, the better nonword readers showed a larger difference between ERPs to rhyming versus nonrhyming pictures (for left-hemisphere but not right-hemisphere recording sites). The neuroanatomical or cognitive implications of the various interactions between ERP data and nonword reading ability are unclear. Nevertheless it seems reasonable to conclude that 'Demonstrating the N400 … effect to be stronger in one group of reading disabled subjects, compared to another, supports the position that specific subtypes of reading disability exist' (McPherson et al., 1996 p. 3).

In particular, the finding that reading ability as assessed by the Woodcock word reading test was not related to any ERP indices but nonlexical reading ability was so related may be interpreted as follows. Most of the words of the Woodcock test are regular words. So subjects can attain a high score on that test either by having good lexical reading abilities or by having good nonlexical reading abilities. Hence the high scorers on this test will be a heterogeneous group, which makes it unlikely that they will yield some characteristic ERP signatures. It would have been a different matter if the word reading test had used only irregular words. A median split on irregular word reading might have identified more homogeneous groups (better vs. worse users of the lexical route for reading), just as the nonword test median split identified better vs. worse users of the lexical route.

Spinelli et al. (1997) identified ten Italian children with developmental surface dyslexia, and assessed contrast sensitivity thresholds for reversal gratings, which were within normal limits for most subjects; they concluded (p. 1807) that 'this

indicates that developmental surface dyslexia is not associated with a deficit in the transient system'.

4.2. Why have some studies failed to find a magno deficit in poor readers?

Although the majority of investigations of this issue have found evidence for a magno deficit in poor readers, at least six such studies have not obtained such evidence: Smith et al. (1986), Victor et al. (1993), Gross-Glenn et al. (1995), Hayduk et al. (1996), Hogben et al. (1995) and Walther-Muller (1995). Hogben (1996) has suggested that these studies may have been sampling a different population of poor readers, perhaps because of differences in method of subject recruitment (mass-testing of children in schools versus recruitment from reading clinics, for example) or differences in selection criteria – in particular 'selecting for poor phonological coding (which the Neale Accuracy scale seems to do to large degree, at least in our own samples) leads to the inclusion of a great enough proportion of dyslexics with a magno deficiency to detect a between-group difference, whereas other selection criteria dilute the proportion of such dyslexics in the sample' (Hogben, 1996p. 174).

Our results confirm Hogben's suggestion. Since ability to read by the lexical procedure is not directly related to the presence or absence of a magno deficit, diagnostic reading tests which emphasise this aspect of reading (for example, reading-aloud tests with irregular words) will be much less likely to pick out poor readers with a magno deficit than diagnostic tests involving nonword reading, such as the Woodcock Word Attack subtest.

This can be directly demonstrated in our data. We took the 40 bad readers for whom we had measurements of reading of regular, irregular and nonword reading, divided them into two groups by a median split on nonword reading, and analysed the differences in Ternus performance between the two groups. The group worse on nonword reading had a significantly lower R^2 (0.759 vs. 0.580; $F(1,34) = 4.216$, $P < 0.05$), and a marginally significantly lower slope (0.0261 vs. 0.0167; $F(1,34) = 3.211$, $P = 0.0821$), though no difference in intercept. When we divided the bad readers into two groups on the basis of a median split on irregular word reading, there were no traces of any differences between the two groups on any of the Ternus indices.

4.3. What is responsible for the relationship between nonword reading and response to the Ternus display?

We have found that there is an association between the ability to read via the nonlexical route and Ternus-related visual processes, whereas the latter processes are not associated with the ability to read via the lexical route. Why is this the case?

One very simple approach that could be taken here is to argue that both the Ternus task and nonword reading are related in some way to temporal information-processing, and that dyslexics have a generalised impairment of temporal information processing. However, as Stein (1993) (p. 83) points out, this argument commits

one to the position that 'there would need to be, in normals, a generalised system, transcending sensory and motor boundaries' which was responsible for all forms of temporal information processing. But there is no evidence that such a general modality-independent temporal processing system exists in the brain; hence this approach to the question appears unprofitable.

We will instead offer two possible explanations, both somewhat speculative. According to the first speculation, the association between nonword reading and performance on the Ternus test occurs because nonword reading requires a sequential left-to-right allocation of attention that might engage the magno and parvo LGN pathways in just the way that these pathways would be engaged by a sequential left-to-right series of eye movements. According to the second speculation, the association between nonword reading and performance on the Ternus test is a coincidence due to anatomical adjacency of two functionally unrelated neural systems.

Before we can approach the first of these speculations, it is necessary for us to be more specific about how we see the lexical and nonlexical reading routes as operating. Our view is that lexical reading is a parallel process across letters, whereas nonlexical reading is a serial left-to-right process across letters. Specifically, the lexical route receives input from all the letters in a letter string simultaneously: all the letters in a word simultaneously contribute activation to the orthographic lexical entry for that word. In contrast, the nonlexical route processes the letters in a letter-string left-to-right, one at a time.

Evidence for this view comes from the 'whammy effect' discovered by Rastle and Coltheart (1998). They studied nonword reading both in a dual-route computational model of reading aloud, the DRC model (Coltheart et al., 1993, 1996; Coltheart and Rastle, 1994; Coltheart et al., 1999; Rastle and Coltheart, 1999a,b) and in human adult normal readers. This model mimicked adult performance, suggesting that input from the letter identification system to the nonlexical route is letter by letter, serially left to right, and that that it takes longer to name nonwords with one or more phonemes represented by multiple letters (SOACH) than nonwords with one-to-one mappings of letters to phonemes (STELD).

Word reading is primarily influenced by the lexical route, with some influence from the nonlexical route, whereas nonword reading is primarily influenced by the nonlexical route. If the lexical route operates in parallel, it will be uninfluenced by letter length; if the lexical route operates serially letter-by-letter, it will be influenced by letter length. Given all of these theoretical proposals, the prediction is that naming latency will be much less influenced by letter length when the stimulus is a word than when it is a nonword. This is so both for human subjects (Weekes, 1997) and for the DRC model.

Thus the lexical reading procedure operates in parallel, whilst the nonlexical route operates sequentially. Now, if the letter-by-letter operation of the nonlexical route actually involved moving the eyes from one letter to the next, it would be obvious why the magno pathway would be important for nonword reading but not important for irregular word reading, and why defective inhibition of parvo pathways by magno pathways would affect nonword reading but not irregular word reading. Of course, eye movements from one letter to the next are not needed for nonword

reading.; but some sort of attentional shift from one letter to the next letter to the left must be involved, if the nonlexical route operates as we are claiming it does. Could the magno and parvo pathways operate in this attentional domain in the way they operate when the eye, rather than just attention, moves? The idea here is that magno responding is triggered not just by physical onset and offset of a stimulus, but also by attentional onset and offset, and when attention switches from position N to position (N + 1) in the letter string, might this require the parvo response to the letter in position N be inhibited by the magno pathways? If that were so, then a defect of the magno pathways would selectively impair nonword (nonlexical) reading, relative to irregular-word (lexical) reading.

These ideas are related to proposals considered by Corbetta and Shulman (1998). Those authors were not specifically concerned with magnocellular and parvocellular processing, but they were specifically concerned with 'the existence of a set of psychological and neural processes for covertly (without changes in eye position) directing attention to locations' (Corbetta and Shulman, 1998, p. 1356), and our speculation is that just such processes are involved in nonword reading since nonword reading requires successive allocation of attention to successive positions in the letter string being read. The same idea has also been proposed by Ladavas et al. (1997) on the basis of investigations of the form of acquired dyslexia known as neglect dyslexia. Ladavas et al. explicitly adopted a dual route model of reading to account for their data, particularly the finding that reading aloud is much worse for nonword reading is much worse than nonwords than for words in neglect dyslexia: their explanation (p. 1083) was that 'the lexical routes lead to an attentional focus that encompasses the single perceptual unit, whereas the phonological route leads to a bias of the attentional focus to the rightmost position of multiple perceptual units within the display'.

Our alternative speculation as to why nonword reading and Ternus performance might be associated depends upon the fact that, just as the LGN contains magno and parvo cells which process visual information, so the medial geniculate nucleus (MGN) contains large and small cells that process auditory information. The traditional distinction for the MGN is between medial and ventral divisions; the medial is also known as the magnocellular division, and although the ventral division is not generally referred to as parvocellular, it clearly is by contrast.

Stein (1996) has proposed that the MGN magno cells are involved in responding to rapid temporal changes in auditory stimuli. As far we know, no one has ever compared the two divisions of the MGN in terms of processing of rapidly time-varying signals. However, the ventral division is the major source of projections to middle layers of the primary cortical field, and has shorter latencies than the medial division, a reasonable basis for the supposition that it might play a more important role in such temporal processing. If so, speech perception would crucially depend upon these cells, a claim consistent with the results of Kraus et al. (1994), who found that in separate regions of guinea-pig MGN there was 'a hierarchy of processing of the spectrotemporal changes which characterise formant transitions' in response to synthetic speech stimuli.

Galaburda and Livingstone (1993) analysed both LGN and MGN cells in the

brains of five dyslexics who came to autopsy. For the left MGN, the dyslexics showed a relative paucity of large cells when compared to normal controls. For both left and right LGN, cells in the magnocellular layer were smaller in dyslexics than in normals, whereas the sizes of cells in the parvocellular layer did not differ between the groups.

It can thus be argued that, had these dyslexics been given the appropriate tests, they might have shown abnormal behaviour on the Ternus test (because of their LGN magno anomalies) and also might have shown abnormalities of phonological aspects of speech perception (because of their MGN magno anomalies). Assuming that these MGN anomalies were present from birth, these individuals would have had lifelong phonological difficulties, and it is highly likely that such difficulties would have made it difficult to learn a system of grapheme-phoneme rules, i.e. difficult to acquire the nonlexical reading procedure. Hence there is some reason to believe that these individuals would have been bad nonword readers as well as showing Ternus abnormalities.

The data here are fragmentary, and the theorising speculative, but the general story could be as follows. The large LGN cells and the large MGN cells are completely different cell populations, with completely different functions. However, since large cells are particularly vulnerable to perinatal insult, both populations are developmentally vulnerable, and so both will be damaged from birth in some individuals. Such individuals will show Ternus abnormalities and bad nonword reading, but not because these two tasks have any processing mechanisms in common; this association of defects will be an accident of brain anatomy.

Anyone who is poor at nonword reading will find it difficult to learn to read, since the ability to sound out printed words that are outside one's sight vocabulary but inside one's auditory vocabulary is crucial for learning to read (Harris and Coltheart, 1986; Share and Stanovich, 1995). Thus even if these individuals did not have Ternus abnormalities, it would be expected that they would be developmentally dyslexic. Therefore on this view the abnormality of the visual magno pathway may not have a causal connection to the developmental dyslexia.

The first of our two speculative explanations of the relationship between Ternus performance and nonword reading ability is perhaps the more interesting; but it needs to be considered in conjunction with results reported by Burr et al. (1982) and Burr et al. (1994). They studied the detectability of horizontal gratings presented during horizontal saccades or with the eyes stationary. Relative to the stationary condition, grating sensitivity was selectively impaired for low frequency gratings, but not for isoluminant gratings. Since the magnocellular system is critical for the perception of low frequency gratings, but insensitive to isoluminant gratings, these results indicate that the magnocellular system is inactive during eye movements. That would be a sensible way for the visual system to behave; if it were not so, we would be highly distracted by the perception of image motion during eye movements. Now, if it is the case that that the magnocellular system is inactive during eye movements, then, as Hogben (1997) points out, there can be no transient-on-sustained inhibition during eye movements. Hence such inhibition could play no role in reading. If so, weakness of this magnocellular inhibitory

system could not harm reading, as was originally suggested by Lovegrove et al. (1980).

However, Hogben's critique is not relevant to the idea that nonlexical reading with the eyes stationary depends upon covert attentional shifting from letter to letter, nor to our speculation that the neural processes responsible for such shifting are also involved in the Ternus illusion.

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